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34th TURKISH CARDIOLOGY CONGRESS

WITH INTERNATIONAL PARTICIPATION

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Dear Colleagues,

In addition to its various training events and activities through the year, Turkish Society of Cardiology has planned to hold this year's National Cardiology Congress in October at a high level to meet the expectations on its 54th anniversary.

The congress, as a leading scientific event both at the national and international level with a remarkable number of participants and high quality scientific content, is being designed to appeal to all participants in a satisfactory way and broad range of its scientific program and diversified flavors of social events.

As well as our colleagues as members of European Society of Cardiology, there will be again our colleagues from different continents as participants to this year's congress. We expect to have a higher number of participation to this year's congress in which the number of participation was nearly 3000 last year. We have been working hard to prepare the best program for you. During our "Symposia", "Debates" and "How to Do" sessions, we will be updating our latest knowledge on cardiovascular diseases. We have extended "Cardiology in Daily Practice" sessions which attracted real attention last year, as "Practical Cardiology" to cover all the practices in Cardiology. We will advance our skills besides our knowledge thanks to "Interactive Video Courses" which we have increased the number in line with the high attention paid during the recent years and a certificate is provided to the participants of the courses.

Every session will take place with participation of esteemed speakers and discussants from Turkey and from around the globe. We strongly believe that our joint sessions with the ESC, ACC, Turkic World Cardiology Association, EHRA and EAPCI will be closely followed by the attendees.

The congress having strengthened more than ever with its international flavor will again be credited by the Turkish Medical Association and EBAC.

We will be more than happy to have you with us during our congress.

With hopes and best regards to meet you and share our information on 05 – 08 October 2017 for the occasion of our 33th Turkish Cardiology Congress with International Participation.

Respectfully,

Prof. Mustafa Kemal Erol, M.D.
President of TSC

Prof. Vedat Aytakin, M.D.
President Elect of TSC



34th TURKISH CARDIOLOGY CONGRESS

WITH INTERNATIONAL PARTICIPATION

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The abstracts are being reprinted without Journal editorial review. The opinions expressed in this supplement are those of the panelists and are not attributable to the sponsor or the publisher, editor, or editorial board of the Anatolian Journal of Cardiology. Clinical judgment must guide each physician in weighing the benefits of treatment against the risk of toxicity. References made in the articles may indicate uses of drugs at dosages, for periods of time, and in combinations not included in the current prescribing information.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-001

The impact of QT interval duration on mortality in a large cohort of community based adult population with a long term follow up

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Background and Aim: Prolonged rate corrected QT interval (QTc) was found to be associated with malignant ventricular arrhythmias/cardiovascular (CV) mortality. However, the impact of QTc on mortality yielded controversial results on community based cohorts. The aim of our study is elucidate the QTc distribution and impact on mortality in Turkish adults.

Methods: We evaluated resting ECGs/data obtained from the HAPPY (Heart Failure Prevalence and Predictors in Turkey) study involving 4650 subjects ≥35 years. Mortalities were detected by registry search. Subjects with missing ECG, antiarrhythmic use, bundle branch blocks, non sinus rhythms and ventricular hypertrophies were excluded. ECGs were interpreted by two cardiologists for baseline parameters/ QTc analysis (Bazett's) [LongQTc >440 ms (male)/>460 ms (female)]. We categorized QTc intervals into seven percentile groups.

Results: 1870 subjects ((mean±SD) years, 51.3±11) were enrolled (female: 55.3%). In 90 months of follow up, 121 deaths occurred [All cause mortality: 6.5%, CV mortality n=79 (4.2%)]. Prevalence of long QTc was 2.8% [n=59 (82% female), 58.3±13 years) and was associated with significantly higher mortality in survival analysis (All cause mortality: 13.6%, CV mortality: 10.2%, p=0.02) (HR:2.2; 95%CI 1,14.5; p=0.03) (Figure 1a). We acquired a U shaped mortality pattern and higher mortality rates on extremes of QTc spectrum (<5th and >95th percentile) (Table1, Figure 1b) (p<0.001), evident in males with QTc >95th (HR:3.3; 95%CI 1.66.8; p=0.01) and females with QTc <5th (HR: 3.8; 95%CI 1.213.9; p=0.02) when compared with 40th 60th percentile QTc population within the same gender even adjusted for age.

Conclusions: Extreme QTc values, even in the normal reference range, might have an impact on mortality with a dose response relationship. Gender differences might be a consequence of the hormonal effects on cardiac ion channels.

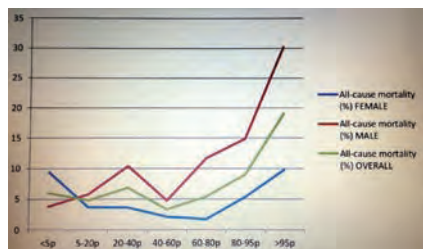


Figure 1. Relationships between the QTc interval and cardiovascular mortality in the general Turkish population.

Table 1. Relationships between the QTc interval and cardiovascular mortality in the general Turkish population

QTc percentile	Female	Female	Female	Male	Male	Overall	Overall	Overall	Overall	
QTc ms	All-cause mortality (%)	CV mortality (%)	QTc ms	All-cause mortality (%)	CV mortality (%)	QTc ms	All-cause mortality (%)	CV mortality (%)	n	
<5th	<383	9,4	9,4	<377	3,8	1,9	<379	6,0	4,8	84
5-20th	383-393	3,7	3,7	377-389	5,8	3,6	379-391	4,8	3,7	272
20-40th	394-403	3,6	2,6	390-399	10,4	5,2	392-401	6,8	3,8	370
40-60th	404-411	2,1	1,6	400-407	4,7	2,3	402-409	3,3	1,9	360
60-80th	412-422	1,7	1,2	408-418	11,7	9	410-420	5,4	4,1	387
80-95th	423-439	5,3	3,7	419-440	14,9	7,9	421-440	9,0	5,3	301
>95th	>439	9,8	7,8	>440	30,2	18,6	>440	19,1	12,8	94
Overall (mean)	408	3,7	2,9	405	9,9	5,9	407	6,5	4,2	1868

with low LVEF. However, there are no studies investigating the prospective value of TWA in patients with young ST-elevated myocardial infarction (STEMI) patients with preserved LVEF. The aim of this study is to determine the capacity of TWA to stratify risk for cardiovascular mortality whether in patients with low arrhythmic risk, such as young MI patients with preserved EF.

Methods: We prospectively recruited 108 consecutive post-MI patients (age 39.5±4.1 years) with preserved cardiac function treated with successful single vessel primary percutaneous coronary intervention. All patients received standard therapy for during hospitalization and after discharge. Maximum TWA was performed using the Modified Moving Average method from continuous electrocardiographic recordings and interpreted blinded. Abnormal TWA was determined as the maximal voltage was >64 µV at heart rate, 125 beats per minute as mentioned in previous studies. The patients followed up for 5 years and the primary outcome was defined as overall cardiac mortality and fatal arrhythmic events.

Results: TWA was positive in 21 patients (19.4%). When STEMI patients were divided into 2 groups according to the TWA positivity, there was no difference in drug use, traditional risk factors, syntax score, and LVEF. During follow-up of 5 years, 7 patients (6.5%) reached the endpoint. After adjustment for known confounding factors in a logistic regression model, Abnormal exercise TWA in post-MI patients was associated with primary outcome [OR=10.78 (95% CI=1.94-59.89) p=0.007].

Conclusions: Time-domain TWA analysis powerfully predicts cardiac mortality in young STEMI undergoing primary percutaneous intervention with preserved LVEF. TWA may serve as a therapeutic target for low-risk MI patients such as young ones and deserves further exploration.

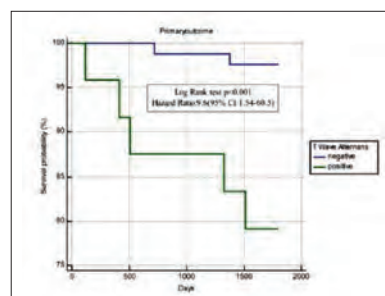


Figure 1. Five year cardiovascular mortality ratios of each group, (Kaplan-Meier Analysis).

Table 1. Baseline characteristics and drug use according to T wave alternans positivity

	Positive TWA n=21	Negatif TWA n=87	P value
Age	38.7±4.0	39.4±4.8	0.172
Sex(male) n(%)	22(91.7)	75(89.3)	0.737
Hypertension, n(%)	11(45.8)	39(46.4)	0.959
Diabetes Mellitus, n(%)	4(16.7)	14(16.7)	1.000
Smoking, n(%)	19(79.2)	64(76.2)	0.760
Body Mass Index	30.0±3.9	29.0±4.7	0.303
Antiplatelet use, n(%)	23(95.8)	80(95.2)	0.903
Beta bloker use, n(%)	21(87.5)	66(78.6)	0.330
ACE inhibitor/ARB use, n(%)	16(66.7)	51(60.7)	0.596
Statin use, n(%)	18(75.0)	61(72.6)	0.816
Left ventricular ejection fraction	55.1±4.7	55.8±4.6	0.509
C-reactive protein(mg/dl)	3.94±2.52	5.71±10.2	0.406

Table 2. Logistic regression analysis for potential predictors of 5-year cardiovascular mortality

	Univariate analysis	P value	Multivariate analysis	P value
Age, years	1.033(0.855-1.248)	0.735		
Male, yes	1.517(0.165-13.901)	0.713		
Beta bloker use, yes	0.589(0.104-3.726)	0.532		
Diabetes Mellitus, yes	0.824(0.093-7.287)	0.861		
Hypertension, yes	1.635(0.344-7.767)	0.536		
Statin use, yes	0.247(0.052-1.107)	0.079	0.256(0.042-1.542)	0.137
C reactive protein	0.985(0.881-1.102)	0.796		
Left ventricular ejection fraction	0.859(0.721-1.023)	0.088	0.875(0.734-1.044)	0.138
T wave alternans positivity, yes	10.789(1.944-59.896)	0.007	10.789(1.944-59.896)	0.007

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-002

T-Wave alternans predicts cardiac mortality in young myocardial infarction patients with preserved cardiac function

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Background and Aim: Secondary prevention of cardiac death in young myocardial infarction (MI) patients with preserved left ventricular ejection fraction (LVEF) is a matter of hot debate due to the fact that the population of these patients is highly heterogeneous in terms of the fatal arrhythmic risk. The association of T-wave alternans (TWA) based on the modified moving average method with the appearance of cardiac mortality and sudden cardiac death has been assessed in different heart diseases especially in patients

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-003

Risk of recurrent stroke in cryptogenic stroke patients with nonsustained episodes of atrial fibrillation

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Background and Aim: Nonsustained episodes of atrial fibrillation (NS-AF) are considered a risk factor for future development of paroxysmal or persistent AF however information about their effect on the risk of recurrent stroke is limited in cryptogenic stroke (CS) patients. We studied the risks of recurrent stroke associated within NS-AF.

Methods: We reviewed 46 patients (age, 22 to 88 years) with cryptogenic stroke from our registry. Supraventricular

tricular runs with >3 beats, lasting <30 s with absolutely irregular RR interval and no distinct p-waves in 24-hour Holter monitoring were considered as nonsustained AF. Patients with PFO were excluded from our study. All patients received aspirin (100 mg per day) for secondary prevention.

Results: Of the 46 patients, 6 (13%) experienced recurrent stroke (mean follow-up for 6.7 months). Baseline characteristics, comorbidities, and echocardiography results of the groups are detailed in Table 1. Patients with recurrent stroke had more prevalent NS-AF (66.7% vs. 17.5%, $p=0.02$) and lower left ventricular ejection fraction (60.0 ± 3.10 vs. 62.6 ± 3.0 , $p=0.04$). The recurrent stroke was significantly higher among patients with NS-AF in comparison to SR (sinus ritm) during follow-up (Long-rank=7.46, $p=0.006$) (Figure 1). After univariable analysis in multivariable analysis, the presence of NSAF (n=11) was a significant independent predictor of an increased risk of recurrent stroke (hazard ratio for the comparison with SR (n=35), 8.64; 95 percent confidence interval, 0.91 to 81.33) (Table 2).

Conclusions: Our study shows higher recurrent stroke in patients with NS-AF in comparison to SR.

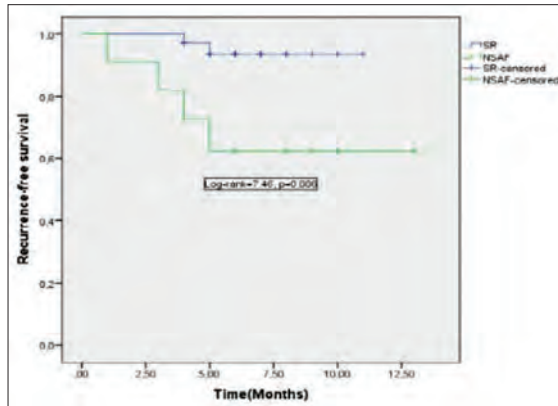


Figure 1. The recurrent stroke was significantly higher among patients with NS-AF (n=11) in comparison to SR (n=35) during follow-up (Long-rank=7.46, $p=0.006$).

Table 1. Baseline patient characteristics

	No Recurrent Stroke (n=40)	Recurrent Stroke(n=6)	p
Value			
Age, yrs	54.0±16.9	59.8±17.1	0.43
Male	21 (%52.5)	2 (%33.8)	0.66
Smoking	7 (%17.5)	1 (%16.7)	0.96
Diabetes mellitus	9 (%22.5)	1 (%16.7)	0.74
Hypertension	19 (%47.5)	4 (%66.7)	0.66
Hypercholesterolemia	9 (%22.5)	1 (%16.7)	0.75
Ischemic heart disease	7 (%17.5)	0 (%0)	0.57
NS-AF	7 (%17.5)	4 (%66.7)	0.02
LVEF(%)	62.6±3.0	60.0±3.10	0.04
LA(mm)	34.8±4.3	36.6±3.8	0.32
Following Time(month)	6.5±2.2	7.8±3.7	0.45

Values are mean±SD or n(%).
LVEF left ventricular ejection fraction, LA left atrium, NS-AF Nonsustained episodes of Atrial Fibrillation

Table 2. Predictors of recurrent stroke in cryptogenic stroke (CS)

Recurrent events	Multivariable HR (95% CI)	p Value
NSAF	8.64 (0.91-81.33)	0.05
LVEF	0.80 (0.56-1.16)	0.25
LA	1.03(0.73-1.34)	0.96

LVEF left ventricular ejection fraction, LA left atrium, NS-AF Nonsustained episodes of Atrial Fibrillation

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-004

Elevated LV mass and LV mass index sign on the athlete's ECG:
Athletes' hearts are prone to ventricular arrhythmia

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Background and Aim: Intense exercise elevates all heart chambers' dimensions, left ventricular mass (LV mass), and left ventricular mass index (LV mass index). The relationship between increased ventricular arrhythmias and sudden cardiac death with LV dilatation and elevated LV mass has been previously demonstrated. We investigated whether sports-related LV dilatation and elevated LV mass and LV mass index cause an increase in ventricular repolarization heterogeneity.

Methods: This prospective observational study recruited 565 participants. There were 226 (female: 28) ath-

letes and 339 (female: 45) healthy controls between 17 and 42 years of age. They were evaluated using 12-lead electrocardiography and transthoracic echocardiography. Electrocardiograms were obtained at a rate of 50 mm/s and an amplitude of 10 mV, including at least 3 QRS complexes for each derivation. They were taken with 12 standard deviations. Transmural dispersion of repolarization indexes (TDR) (Tp-Te interval, Tp-Te/QT ratio and Tp-Te/QTc ratio, Tp-Te(d)) were measured from precordial derivations. Measurements were taken with a program which was generated with MATLAB codes.

Results: Tp-Te interval, Tp-Te/QT ratio, Tp-Te/QTc ratio, Tp-Te(d), PW (posterior wall thickness), IVS (inter-ventricular septal thickness), LVEDD (left ventricular end-diastolic diameter), LV mass (left ventricular mass), and LV mass index (left ventricular mass index) for the athlete group were significantly higher than for the control group (Table 1). Correlation analyses revealed that TDR indexes significantly correlated with PW, IVS, LVEDD, LV mass, and LV mass index (Table 2, Figure 1).

Conclusions: LV mass and LV mass index increase in well-trained athletes, and this increase leads to an increase in TDR indexes. The increased frequency of ventricular arrhythmia and sudden cardiac death may be explained with increasing ventricular repolarization heterogeneity in these individuals.

Table 1. Inter-group comparison of demographical and laboratory data

	Athlete Group n= 226	Control Group n= 339	p
Age (Year)	25 (21-30)	25 (20-33)	0.63
Gender (Male/Female)	198/28	294/45	0.76
Tp-Te (ms)	80.0 (72.0-85.0)	65.0 (62.0-68.0)	<0.0001
QTmax(ms)	381.0 (369.0-402.0)	356.0 (347.0-365.0)	<0.0001
QTc (ms)	401.29±17.47	393.34 ±23.23	<0.0001#
Tp-Te/QT ratio	0.20 (0.19-0.21)	0.18 (0.17-0.19)	<0.0001
Tp-Te/QTc ratio	0.20 (0.18-0.21)	0.16 (0.15-0.17)	<0.0001
Tp-Te(d) (ms)	20 (15-26)	10 (10-16)	<0.0001
HR	65 (57.75-72)	74 (70-80)	<0.0001
PW (mm)	10.0 (9.0-11.0)	8.0 (7.0-8.0)	<0.0001
IVS (mm)	11.0 (10.0-12.0)	8.0 (7.0-8.0)	<0.0001
LVEDD (mm)	54.0 (48.0-58.0)	42.0 (40.0-44.0)	<0.0001
LVESD (mm)	39.0 (34.75-43.0)	27.0 (25.0-29.0)	<0.0001
EF %	54.0 (50.0-58.0)	66.13 (63.26-69.41)	<0.0001
FS %	28.0 (26.0-30.0)	36.1 (34.1-38.6)	<0.0001
RWT %	37.5 (34.5-40.0)	37.0 (34-40)	0.83
LVEDV (ml)	141.31 (107.52-166.56)	78.58 (70.0-87.69)	<0.0001
LVEDVI (ml/m ²)	75.43 (62.78-87.81)	42 (37.68-46.72)	<0.0001
Length(meter)	1.78 (1.75-1.82)	1.74 (1.70-1.76)	<0.0001
Weight(kilogram)	70.78 ±9.02	75.65 ±9.78	<0.0001#
BMI (kg/m ²)	21.97 (21.22-22.87)	25.42 (23.18-27.11)	<0.0001
BSA (m ²)	1.87 ±0.16	1.90 ±0.14	0.02#
LV mass (g)	220.59 (158.82-280.42)	100.60 (89.37-113.63)	<0.0001
LV mass index (g/m ²)	118.74 (87.94-148.26)	53.22 (47.54-58.88)	<0.0001
SBP (mmHg)	110.0 (100.0-120.0)	115.0 (110.0-120.0)	<0.0001
DBP (mmHg)	70.0 (65.0-70.0)	70.0 (70.0-80.0)	<0.0001

BMI: Body Mass Index, BSA: Body Surface Area, HR:Heart rate, IVS: Interventricular septum, LV: Left ventricle, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, PW: Posterior wall, EF: Ejection fraction, FS: Fractional shortening, RWT: Relative wall thickness, LVEDV: Left ventricular end-diastolic volume, LVEDVI: Left ventricular end-diastolic volume index, QTmax: QT maximum, ms: millisecond, mm: millimeter, QTc: QT corrected, SBP: Systolic blood pressure, DBP: Diastolic blood pressure. #Normality of the distribution was evaluated by the Kolmogorov-Smirnov test and the Mann-Whitney U test applied to compare for continuous variables except from QTc, weight and body surface area (BSA)

Table 2. Pearson correlation analysis between ventricular repolarisation parameters and echocardiographic parameters

	Tp-Te (ms)	QTmax(ms)	QTc (ms)	TP-Te/QT ratio	TP-Te/QTc ratio	Tp-Te(d)(ms)
	r p	r p	r p	r p	r p	r p
PW (mm)	0.874, <0.0001	0.799, <0.0001	0.152, <0.0001	0.741, <0.0001	0.808, <0.0001	0.809, <0.0001
IVS (mm)	0.883, <0.0001	0.803, <0.0001	0.173, <0.0001	0.755, <0.0001	0.808, <0.0001	0.792, <0.0001
LVEDD (mm)	0.883, <0.0001	0.771, <0.0001	0.106, <0.050	0.776, <0.0001	0.835, <0.0001	0.779, <0.0001
LVESD (mm)	0.872, <0.0001	0.762, <0.0001	0.119, <0.020	0.768, <0.0001	0.820, <0.0001	0.758, <0.0001
LVEDV(ml)	0.880, <0.0001	0.768, <0.0001	0.095, <0.023	0.771, <0.0001	0.836, <0.0001	0.786, <0.0001
LVEDVI(ml/m ²)	0.872, <0.0001	0.765, <0.0001	0.114, <0.007	0.760, <0.0001	0.820, <0.0001	0.769, <0.0001
RWT%	0.237, <0.0001	0.268, <0.0001	0.122, <0.004	0.164, <0.0001	0.186, <0.0001	0.271, <0.0001
LV mass (g)	0.926, <0.0001	0.821, <0.0001	0.117, <0.010	0.798, <0.0001	0.872, <0.0001	0.844, <0.0001
LV mass index (g/m ²)	0.923, <0.0001	0.823, <0.0001	0.132, <0.004	0.791, <0.0001	0.863, <0.0001	0.835, <0.0001

IVS: Interventricular septal thickness, LV mass: Left ventricular mass, LVEDD: Left ventricular end-diastolic diameter, PW: Posterior wall thickness.

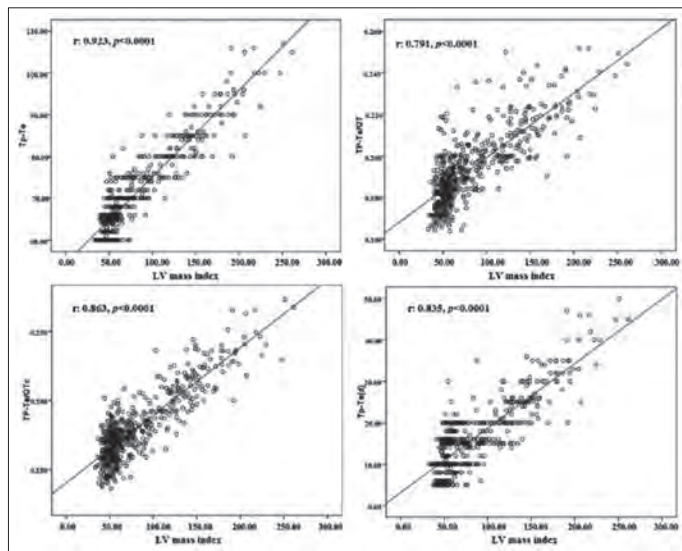


Figure 1. The correlations between TP-Te, TP-Te/QT, TP-Te/QTc, TP-Te(d) and LV mass index.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-006

Atrial electromechanical delay is a useful parameter to predict atrial fibrillation in hemodialysis patients

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Background and Aim: Prevalence of atrial fibrillation is higher in hemodialysis patients as compared to general population. Atrial electromechanical delay is known as a significant predictor of atrial fibrillation. In this study, we aimed to reveal the relationship between atrial electromechanical delay and attacks of atrial fibrillation.

Methods: The study included 77 hemodialysis patients over 18 years of age giving written consent to participate in the study. The patients were divided into 2 groups based on the results of 24-hour ECG holter as the ones having attacks of atrial fibrillation and the others without any attack of atrial fibrillation. Standard echocardiographic measurements were taken from all patients. Additionally, atrial conduction times were measured by tissue Doppler technique and atrial electromechanical delays were calculated.

Results: Intra- and interatrial electromechanical conduction times were found as significantly lengthened in the group of patients with attacks of atrial fibrillation (p=0.03 and p<0.001 respectively). The optimal cut-off time for interatrial electromechanical delay to predict atrial fibrillation was >21 msec with a specificity of 79.3% and a sensitivity of 73.7% (area under the curve 0.820; 95% confidence interval [CI], 0.716-0.898). In the multivariate logistic regression model, interatrial electromechanical delay (odds ratio=1.230; 95% CI, 1.104-1.370; p<0.001), and hypertension (odds ratio=4.525; 95% CI, 1.042-19.651; p=0.044), were also associated with atrial fibrillation after adjustment for variables found to be statistically significant in univariate analysis and correlated with interatrial electromechanical delay.

Conclusions: Interatrial conduction delay is independently related with the attacks of atrial fibrillation detected on holter ECG records in hemodialysis patients.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-005

Epidemiology and management of atrial fibrillation in Turkey in comparison to other countries

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Background and Aim: Over the recent years, large-scale observational studies on atrial fibrillation (AF) have been conducted in Turkey. The purpose of this study is to compare the Turkish data with data obtained through observational studies in Europe and United States.

Methods: Data from two large-scale observational AF studies conducted in Turkey, AFTER and RAMSES, were compared with data from GARFIELD, ORBIT-AF, and EORP-AF studies, which were conducted in Europe and United States.

Results: AF patients in Turkey are younger than those from developed countries (69.5±10.5 vs. 71.2±11.2, p=0.03). While males constitute the majority of the AF patients in Europe and United States, majority of the AF patients in Turkey are females. No significant differences are evident in the frequency of comorbid diseases such as diabetes mellitus, heart failure, history of stroke, or hypertension (Table). Similarities are also evident in terms of CHADS2 and CHA2DS2VASc scores. Time in Therapeutic Range (TTR) rates of patients on warfarin therapy were lower in Turkey as compared with other populations (49% vs. 54%, p<0.01). The proportion of patients who do not receive oral anticoagulants, despite having indications, is higher in Turkey than in developed populations.

Conclusions: Turkey differs from other countries in terms of epidemiology and treatment of AF. There is a need for developing guidelines specific for Turkish population.

Table 1. Comparison of baseline characteristics in patients with nonvalvular af enrolled in large-scale observational studies

	GLORIA-AF (n=10675)	GARFIELD (n=10614)	ORBIT-AF (n=9484)	EORP-AF (n=3049)	AFTER (N=1745)	RAMSES (n=6273)
Age±SD, year	-	70.2±11.2	-	68.8	69.1±11.2	69.7±10.7
Age, median (IQR), year	71	-	75	-	-	70
Male	5813	6034	-	-	761	2769
Previous stroke or TIA	999 (9.4)	1528 (14.4)	(15)	(13.1)	266 (15.2)	845 (13.5)
Heart failure	2530 (23.7)	2229 (21.0)	(33)	(47.5)	537 (30.7)	1386 (22.0)
Diabetes mellitus	2454 (23.0)	2330 (22.0)	(29)	(20.6)	416 (23.8)	1389 (22.0)
Hypertension	7993 (74.9)	8249 (77.8)	(83)	(70.9)	1274 (73.0)	4305 (69.0)
CHADS2	1.9±1.1	1.9±1.2	2.3±1.3	1.9±1.3	-	1.8±1.7
CHA2DS2VASc	3.2±1.5	3.2±1.6	-	3.2±1.8	3.5±1.7	3.3±1.6
OAC	VKA:3449 D:3439 R:1282 A:369	VKA:5925 NOAC:475	-	-	-	VKA:2173 D:1148 R:942 A:250
OAC (%)	VKA:32 D:32 R:12 A:3	VKA:56 NOAC:4	VKA:72 D:4.8	VKA:71 D:7 R:2	VKA:40 D:18 R:18 A:4	VKA:34 D:18 R:18 A:4
No OAC (%)	2136(20%)	4214(40%)	(23%)	(20%)	(60%)	1716(27%)

NOAC: New Oral Anticoagulan; D: Dabigatran; R: Rivaroxaban; A: Apixaban.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-007

Is the Tpeak-to-Tend interval assessment a reliable method for estimating the risk of sudden cardiac death and ventricular arrhythmic events in hypertrophic cardiomyopathy patients?

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Background and Aim: Sudden cardiac death (SCD) is the most devastating complication of hypertrophic cardiomyopathy (HCM). Tpeak-Tend interval (Tpe) found to be associated with poor prognosis in various cardiac disorders. The aim of the study was to assess the relationship between the TPe, corrected TPe (TPec) duration and the predicted five-year risk of SCD score (HCM Risk-SCD), some ventricular arrhythmic events among patients with HCM.

Methods: This study included 118 consecutive patients with HCM and a control group of 63 normal subjects. Measured Tpe interval was corrected for heart rate using Bazett (TPec) formula. Presence of fragmented QRS (fQRS), QRS, QT and QTc duration were evaluated on an electrocardiography (ECG). Presence of micro T wave alternans (MTWA) evaluated on ambulatory ECG and serum galectin-3 levels and some echocardiographic (ECHO) parameters were evaluated in participants and control group. The HCM Risk-SCD score (%) calculated in patients with HCM for each patient.

Results: In the statistical comparison of the patient and control group TPe, TPec interval, the presence of fQRS and MTWA, QRS, QT, QTc duration, serum galectin-3 levels, and some clinical, ECHO, and ambulatory ECG findings were all statistically higher in HCM group than in control group (all p<0.05). A receiver operating characteristics curve analysis identified TPe and TPec>78.5 ms as an effective cut-off point in both TPe and TPec for HCM (Figure 1). Patients were divided into two groups according to their Tpe and TPec intervals above or below 78.5ms. There were statistically significant differences in some clinical, ECHO, ECG and ambulatory ECG findings, the HCM-Risk SCD score (%), presence of fQRS and MTWA, serum galectin-3 levels between TPe>78.5ms (n=48), TPec>78.5ms (n=48) and TPe<78.5ms (n=70), TPec<78.5ms (n=70) (all p<0.001). Statistically significant correlation was observed between TPe, TPec intervals and the percentages of some clinical, ECHO, ECG, ambulatory ECG findings, the HCM-Risk SCD score, the presence of fQRS, MTWA, serum galectin-3 level, the requirement for cardiopulmonary resuscitation(%) and implantable cardioverter defibrillator implantation (%). Both univariate and multivariate analysis distinguished TPe and TPec as independent predictors of high risk for the HCM-Risk SCD. TPe and TPec intervals of more than 78.5 ms yielded a with sensitivity, specificity, positive predictive value and negative predictive value of 76%, 77%, 76%, 78% and 81%, 78%, 80%, 78%, respectively.

Conclusions: Both TPe and TPec were identified as independent predictors for high risk of the HCM-Risk SCD. Evaluation these simple ECG intervals may provide incremental predictive value to traditional risk factors and can potentially enrich SCD risk stratification. In addition, TPec measurement has been shown to be more effective in determining HCM SCD risk score than TPe measurement.

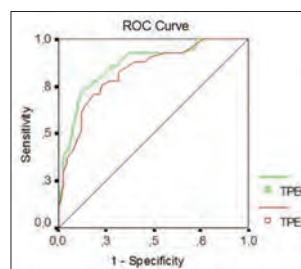


Figure 1. In a ROC curve analysis, TPe and TPec>78.5 was identified as an effective cut-off point in HCM Risk-SCD for HCM.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-008

Ventricular repolarization markers as new electrocardiographic criteria using sinus beat to differentiate epicardial from endocardial origin of ventricular tachycardia in patients with structural heart disease

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Background and Aim: Electrocardiography (ECG) may help differentiate epicardial and endocardial ventricular tachycardia (VT) in patients with structural heart disease. However QT dispersion in these two groups has not been studied previously. This retrospective study compared QT duration and dispersion in patients who had successful epicardial and endocardial VT ablation between January 2009 and May 2014 performed at VA Medical center, Minneapolis, USA.

Methods: A total of 32 patients with structural heart disease who underwent successful VT ablation between January 2009 and May 2014 were included in the study. Fourteen patients who had successful epicardial VT ablation were defined as epicardial VT group (EPI) whereas 18 patients who were successfully ablated endocardially were grouped as endocardial VT group (ENDO). QT dispersion (QTdisp) was defined as the difference between the longest (QTmax) and shortest (QTmin) QT interval on 12 lead ECG.

Results: Mean age was 67±9 years and mean ejection fraction was 40±16% in EPI group while 61±12 years and 42±14% for ENDO group. Baseline 12 lead electrocardiography (ECG) analysis showed that QTmax and QTdisp measurements were higher in the EPI group compared to ENDO group (479±34 vs 449±20, p=0.008 and 59±14 vs 35±8, p=0.001, respectively). The best combined sensitivity and specificity that separated patients with Epicardial vs Endocardial focus was at QTdisp of 45msec, which resulted in a combined sensitivity 94% and specificity 86% with an area under the ROC curve of 0.95.

Conclusions: The patients in EPI group have higher QTdisp compared to patients in ENDO group. QTdisp may be additional criterion to differentiate epicardial vs endocardial origin of VT in patients with structural heart disease.

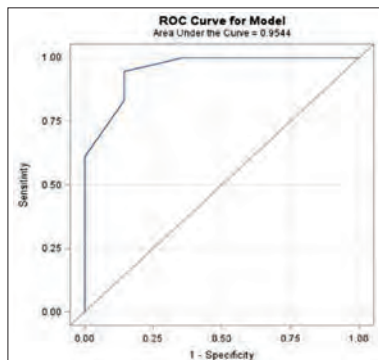


Figure 1. The best combined sensitivity and specificity that separated patients with epicardial focus vs patients with endocardial focus was a cutoff QT dispersion of 45msec, which resulted in a combined sensitivity/specificity of 94%/86% with an area under the ROC curve of 0.95.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-009

New left bundle branch block after transcatheter aortic valve replacement:
Prognosis, single center data

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Background and Aim: Whether or not a permanent pacemaker (PPM) implantation is indicated in patients who developed persistent new onset left bundle branch block (LBBB) after transcatheter aortic valve replacement (TAVR) is not clear. The primary objective of this study was to determine PPM utilization and mortality in this group.

Methods: In this retrospective study, 47 out of 190 patients who underwent TAVR between March 2012 and June 2015 and developed a persistent new LBBB post-procedure were studied. Seven patients were deemed to require a PPM due to either symptomatic bradycardia or concern over new LBBB. Mortality outcome was assessed in all at 6 months.

Results: No patients died in the PPM group at 6 months. In PPM group (n=7), 5 patients required <1% ventricular pacing. The 2 patients who required ventricular pacing had atrial fibrillation with need of high dose beta-blocker for rate control. In patients with LBBB but without PPM (n=40), 2 died in the hospital of cardiogenic shock and aortic dissection and 7 died in 6 months due to exacerbation of heart failure (n=5), sepsis (n=1) and stroke (n=1) (Figure 1). Diabetes mellitus (p=0.023), chronic kidney disease (p=0.003) and persistent LBBB at one month (p=0.037) were associated with mortality at 6 months. None of the deaths was related to atrioventricular block or sudden cardiac death and only one of the 40 patients later required a PPM implant due to high degree AVB.

Conclusions: Our findings suggest that early PPM implantation is not indicated for post-TAVR LBBB without complete or high degree AV block. Further, in most patients who receive PPM for early bradycardia, the conduction disturbance appears to improve during the first 6 months of follow-up.

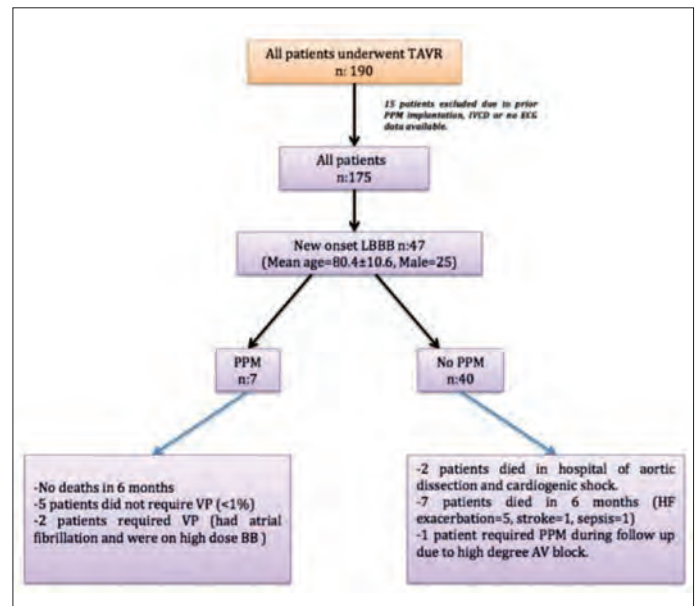


Figure 1. Study design and outcomes. TAVR: Transcatheter aortic valve replacement; PPM: Permanent pacemaker; IVCD: Intraventricular conduction delay; ECG: Electrocardiogram; LBBB: Left bundle branch block; VP: Ventricular pacing; BB: Beta blocker; HF: Heart failure; AV: Atrioventricular.

Table 1. Baseline characteristics

	No new LBBB (n= 104)	New LBBB (n= 47)	P value
Age (years±SD)	79±9	80±11	0.63
Male Gender % (n)	46.2 (48)	53.2 (25)	0.42
Diabetes mellitus % (n)	27.9 (29)	36.2 (17)	0.31
Hypertension % (n)	95.2 (99)	80.9 (38)	0.005
Hyperlipidemia % (n)	81.7 (85)	76.6 (36)	0.46
CAD % (n)	72.1 (75)	83.0 (39)	0.15
CABG % (n)	20.2 (21)	21.3 (10)	0.88
PCI % (n)	38.5 (40)	44.7 (21)	0.47
CKD % (n)	17.3 (18)	31.9 (15)	0.04
Liver disease % (n)	6.8 (7)	6.4 (3)	0.92
COPD % (n)	29.1 (30)	27.7 (13)	0.85
Pulmonary HTN % (n)	27.2 (28)	23.4 (11)	0.62
AV nodal blocking agents % (n)	76.0 (79)	72.3 (34)	0.63
Type of valve % (n)			
1- Edwards Sapien Valve	83.6 (87)	66.0 (31)	0.001
2- Medtronic Core Valve	16.4 (17)	23.4 (11)	-
3- Lotus	0 (0)	10.6 (5)	-
Balloon Size (mm±SD)	25.7±2.4	26.3±2.6	0.15
Aortic annulus diameter (mm±SD)	23.8±2.3	23.8±2.3	0.92

Table 2. Baseline electrocardiographic and echocardiographic parameters

	No new LBBB (n= 104)	New LBBB (n= 47)	P value
Sinus rhythm % (n)	74.0 (77)	85.1 (40)	0.13
Right bundle branch block % (n)	10.6 (11)	8.5 (4)	0.69
Left anterior fascicle block % (n)	10.6 (11)	14.9 (7)	0.45
Left posterior fascicle block % (n)	1.6 (2)	6.4 (3)	0.17
Low degree AV block % (n)	4.8 (5)	10.6 (5)	0.18
Atrial arrhythmia* % (n)	26.0 (27)	14.9 (7)	0.13
Ejection fraction (mean±SD, %)	56.4±10.8	52.5±11.1	0.047
Mean aortic valve gradient (Mean±SD, mmHg)	45.7±14.1	40.6±11.5	0.022

Table 3. Outcomes

	No new LBBB (n= 104)	New LBBB (n= 47)	P value
Mortality in 1 year % (n)	9.6 (10)	14.9 (7)	0.34
Ejection fraction in 1 year (mean±SD, %)	57.6±8.3	51.8±11.2	0.002
PPM implant during index hospitalization % (n)	0 (0)	14.9 (7)	<0.001
PPM implant after discharge in 1 year % (n)	3.8 (4)	2.13 (1)	0.58
Length of stay (mean±SD, days)	7.3±7.3	5.9±2.7	0.09
Post-op atrial fibrillation % (n)	16.3 (17)	8.5 (4)	0.20

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-010

Electrocardiographic properties in patients with synthetic cannabinoid intoxication

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Background and Aim: Synthetic cannabinoids (SC) are part of a group of drugs called new psychoactive substances. People who have used synthetic cannabinoids and have been taken to emergency rooms have shown severe effects including: rapid heart rate, vomiting, violent behavior, suicidal thoughts. Synthetic cannabinoids can also raise blood pressure and cause reduced blood supply to the heart, as well as kidney damage and seizures. In this study we aimed to investigate the effects of synthetic cannabinoid drugs (BONZAI-by in name Turkey) to 12 derivation electrocardiographic (ECG) parameters in patients admitted to the emergency department with SC intoxication.

Methods: Eighty-one SC drug addict admitted to the emergency department with the signs and symptoms of intoxication (group 1; 76 M, mean age 32.5±11.3 years) and thirty control subjects (Group 2; 25 M, mean age 43.7 ± 12.0 years) were included to the study. A detailed medical history, physical examination, heart rate, systolic and diastolic blood pressures were recorded. 12 lead ECGs were obtained simultaneously using a recorder set at a 50 mm/s paper speed and a voltage calibration of 1 mV/cm. ECGs were interpreted using standard criteria. Biochemical and haematological parameters were measured at the admission. ECG parameters were measured using a digital caliper (sensitivity: 1/100 mm) by magnifying lens. Maximum, minimum P wave, P peak time, PR and maximum, minimum QT durations were calculated. P and QT dispersion were calculated. Corrected QT was calculated according to Bazett's formula. P wave amplitude, intrinsicoid deflection time, index of cardio-electrophysiological balance (iCEB: QT/QRS) were also calculated.

Results: Age was significantly lower in group 1 compared to group 2 (Table 1). Heart rate was significantly higher in group 1 compared to group 2 (Table 1). Glucose and White blood cells were significantly higher in group 1 compared to group 2 (Table 1). P wave dispersion, P wave amplitude were significantly higher in group 1 compared to group 2 (Table 1). QTd and cQT were significantly higher in group 1 compared to group 2 (Table 1). iCEB was significantly higher in group 1 compared to group 2 (Table 1). PR interval, intrinsicoid deflection time and P wave peak time were similar between the groups. Correlation analysis revealed, significant positive correlation between WBC levels and QT dispersion, corrected QT, iCEB (r=0.74; p<0.001, r=0.45; p=0.019, r=0.44; p=0.005 respectively). There were positive correlation between glucose levels and P dispersion and QT dispersion (r=0.43; p=0.025; r=0.42; p=0.02, respectively).

Conclusions: Subjects with SC (BONZAI) intoxication are more prone to both atrial and ventricular arrhythmias. These synthetic drugs have significant effect to surface electrocardiographic parameters. The index of cardio-electrophysiological balance which has been demonstrated utility in predicting cardiac arrhythmias after administration of drugs are significantly higher in patients with SC intoxication.

Table 1. Demographic, laboratory and electrocardiographic characteristic of the groups

	Group 1 (n=81)	Group 2 (n=30)	P
Age (years)	32.5±11.3	43.7 ± 12.0	0.02
Gender (M)	76	25	0.09
Systolic blood pressure (mm Hg)	116.5±23.8	125.0±11.6	0.11
Diastolic blood pressure (mm Hg)	67.9±12.5	75.6±8.2	0.09
Heart rate (beat/min)	96.2±21.5	76.1±6.5	0.01
Glucose (mg/dL)	152.5±68.7	93.6±5.3	0.018
WBC (x103/L)	13.50 ±6.11	6.23±2.76	0.001
Sodium (mmol/l)	134.9±18.7	140.2±2.4	0.42
Potassium (mmol/L)	4.3±3.1	4.4±3.2	0.83
P wave dispersion (ms)	24.1±13.1	16.6±9.8	0.02
P wave peak time (ms)	32.3±11.8	33.4±11.9	0.75
QT dispersion (ms)	52.0±29.8	20.6±10.3	<0.001
cQT (ms)	458±45	431±29	0.01
QRS (ms)	93.5±17.6	86.5±13.7	0.12
Intrinsicoid deflection time (ms)	31.4±11.3	29.5±9.4	0.50
iCEB	5.40±0.75	4.90±0.58	0.04

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-011

T peak to T end interval changes before and after left ventricular assist device implantation

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Background and Aim: The interval between the T-wave's peak and end (Tpe) and Tpe/QTc (QT interval cor-

rected for heart rate) ratios are well known markers of ventricular transmural dispersion of repolarization. Prolongation of Tpe has been shown to correlate with the inducibility of ventricular tachycardias at electrophysiology studies and was also associated with sudden cardiac death in some retrospective observational studies. Left ventricular assist devices (LVADs) improve the functional status and survival in patients with advanced heart failure and now they are being implanted with increasing frequency. However, ventricular arrhythmias are still important under LVAD support. The aim of this study is to evaluate the effects of LVAD on the heart's electrical system, especially the Tpe interval and Tpe/QTc ratio.

Methods: Forty-eight patients with LVAD were included in this retrospective study. ECG's prior to implantation and 3 or 6 months after implantation were evaluated. QRS duration, RR, QT and Tpe intervals were measured using semi automated on screen software.

Results: All patients had continuous flow pumps and mean age was 51.8±10.3, 85.4% were male and 52.1% had ischemic cardiomyopathy. After cf-LVAD implantation QRS durations, QTc intervals were significantly prolonged (p=0.003, p=0.043 values respectively). There was a mean 8.7±38ms decrease in Tpe interval, however this change was not statistically significant (p=0.119). Tpe/QTc ratio change, which is proposed as a more accurate way of measuring transmural dispersion of repolarization, was statistically significant (p=0.016) (Table 1, Figure 1 and 2).

Conclusions: Implantation of LVAD appears to have a positive effect on transmural dispersion of repolarization of the failing myocardium, which might reduce the risk of ventricular arrhythmias in post-implantation 3rd to 6th months.

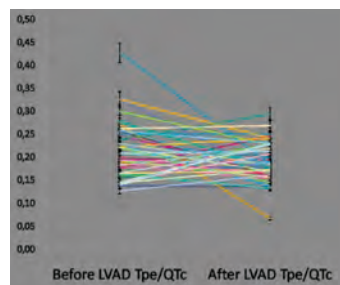


Figure 1. Tpe/QTc ratio change after LVAD implantation.

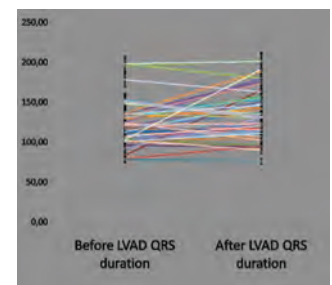


Figure 2. QRS duration change after LVAD implantation.

Table 1. Baseline characteristics and comparisons of the decreased and increased Tpe/QTc groups

	All Patients	Decreased Tpe/QTc	Increased Tpe/QTc	p
Age	51.8±10.3	51.7±10.6	52±11.3	0.753
Male gender	85.4%	74.1%	100%	0.026*
Ischemic CM	52.1%	48.1%	56.3%	0.607
HT	52.1%	55.6%	50%	0.724
DM	31.3%	29.6%	25%	1
BMI(kg/m2)	26.4±3.9	26.5±4.5	26.5±3.4	0.965
Internacs profile ≥3	68.7%	77.8%	68.8%	0.719
Bridge to Tx	93.8%	92.6%	93.8%	1
LVEF(%)	21.1±3	20.8±2.6	21.1±3.7	0.802
Spap(mmHg)	53.1±15	53.9±11.8	56.8±18.5	0.870

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-012

The impact of testosterone levels on early repolarization patterns observed in healthy Turkish males

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Background and Aim: Early repolarization pattern (ER) on surface ECG, has recently been associated with an increased risk of sudden cardiac death. Scarce data is present about the evident male dominance in ER and the effect of gonadal hormones on cardiac ion channel functions. Our aim was to evaluate the relationship with testosterone levels and the presence of ER in healthy Turkish-males.

Methods: 180 healthy male volunteers between ≥18 to ≤50 years old without any known cardiac/systemic disorders were evaluated. ECG, blood biochemistry and total testosterone levels were obtained together with thorough physical examination. Subjects with complete-bundle-branch-block, non-sinus-rhythms and any abnormality on cardiac examination were excluded from the study. ER on ECG was defined as J-point elevation of ≥0.1 mV in ≥2 leads in the inferior (II, III, aVF) (Inferior ER), lateral (DI, aVL, V4-6) (Lateral ER) or both (Inferolateral ER).

Results: 172 subjects (mean age 34.9±7.9 years) were included in our analyses. 45 ER (26%) were detected. 22 were lateral (49%), 13 inferior (29%) and 10 were (22%) inferolateral. ER+ subjects were demonstrating significantly lower basal heart rates (73.9±11 bpm vs 68.4±10.3 bpm, p=0.001) and longer PR intervals (153.9±20.3 ms vs 163.3±21.6 ms p=0.01). ER+ subjects had significantly higher testosterone levels compared with the ones without (485.5±128.3 ng/dl vs 559.3±167.7 p<0.001). In the subgroup analyses, Inferior/Infero-

lateral ER was significantly associated with higher testosterone levels compared with the ER- population, while testosterone levels of subjects with lateral ER was not significantly higher. Electrolytes and blood chemistry values were non-significant between ER+ and - subjects.

Conclusions: Testosterone might be associated with the male predominance observed in the ER patterns. More malignant Inferior/Inferolateral ER seems to be mainly associated with the high testosterone levels in Turkish male population. This finding might be attributed to the previously demonstrated effects of testosterone on ion channel functions, especially Ito (K) channels.

Table 1. Mean testosterone levels in each ER subgroup and their statistical significance

Patterns	Testosterone level (ng/dl, ±SD)	p value
ER + (n=45)	559.3±167.7	<0.001
ER - (n=131)	485.5±128.3	
Lateral ER (n=22)	520.6±145.5	
Inferior ER (n=13)	593.4±201.6	0.007
Inferolateral ER (n=10)	600.2±163.3	
Total (n=176)	505.3±143.0	

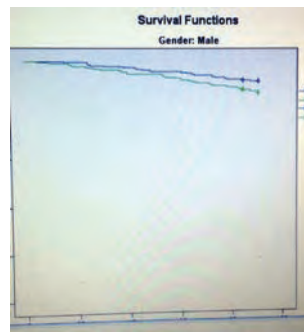


Figure 1. Kaplan-Meier survival analysis of male subjects with MetS for overall mortality. Log-rank p=0.005.

Epidemiology

OP-013

Gender specific effects of metabolic syndrome on mortality in a large cohort of Turkish middle aged adult population with long term follow up

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Background and Aim: Physicians have long known that certain conditions increase a person's risk of developing atherosclerotic cardiovascular disease (CVD) and certain CVD risks tend to cluster together. Metabolic Syndrome (MetS) is referred to as the group of CVD risk factors that result from insulin resistance and include hyperglycemia, dyslipidemia, hypertension, and central adiposity. Even if there are robust data about MetS' importance in predicting poor overall and CVD outcomes on population based studies, little is known about the gender and age specific value of MetS.

Methods: Data and survival information were obtained as a secondary analysis from the HAPPY (Heart Failure Prevalence and Predictors in Turkey) study including randomly selected 4650 subjects ≥35 years with laboratory and clinical data from all geographical regions of Turkey. According to the most recent NCEP ATP III definition, MetS was deemed positive if three or more of the following five criteria are met: waist circumference over 102 cm (men) or 88cm (women), blood pressure over 130/85 mmHg, fasting triglyceride (TG) level over 150 mg/dl, fasting high-density lipoprotein (HDL) cholesterol level less than 40 mg/dl (men) or 50 mg/dl (women) and fasting blood sugar over 100 mg/dl. All cause and cardiovascular (CV) mortality data were taken from the national registry records and telephone interviews.

Results: After the exclusion of subjects with incomplete clinical data and no survival information, 1862 subjects (mean±SD)years, 51.3±11 were enrolled (female n=1029, 55.3%). In 90-months-of-follow-up, 113 deaths occurred [All-cause-mortality: 6%, CV-mortality n=70 (4%)]. Prevalence of MetS in general population was 51.1% (n=952) (female n=594 %62.4). The contributing parameters of MetS criteria were significantly different between males and females (Table-1). MetS was significantly predicting overall and CV mortality in males however it failed to predict mortality in females [Hazard Ratios in males with MetS for overall mortality: 1.6 (CI=1.1-2.5 p=0.036) females NS. CV mortality HR: 1.7 (CI=1.1-3.1 p=0.046) females NS] (Figure 1). The predictive value of MetS was more evident in males between the age of >55 and <65 years old [HR 5.1 CI=1.5-17.9 p=0.011 for overall mortality and HR: 10.5 (CI 1.3-83.5 p=0.025) for CV mortality] and also NS in males with MetS over 65 years old for both overall and CV mortality.

Conclusions: MetS criteria failed to predict poor CV and overall outcomes in female adult population. It might be a consequence of different contribution of MetS scoring parameters for females. It might also be inferred from the findings that we might need different and additive parameters in order to risk stratify specific gender and age groups.

Table 1. Baseline characteristics of the study population by gender in healthy and metabolic syndrome

Parameters / %	Female		p	Male		p	n
	Healthy	MetS		Healthy	MetS		
n	435	594	<0.001	475	358	<0.001	1862
Age (years)	49±11	53±11	<0.001	52±12	54±11	0.005	
BMI (kg/m ²)	27±4	31±5	<0.001	26±3	26±3	<0.001	
Systolic blood pressure (mmHg)	126±17	142±20	<0.001	128±17	26±142±20	<0.001	
Diastolic blood pressure (mmHg)	81±10	90±12	<0.001	81±10	90±12	<0.001	
Cigarette smoking (%)	(14.9%)	(14.5%)	0.882	(47.4%)	(40.8%)	0.012	
BNP	136	113	0.221	132	157	0.426	
GFR	83±16	79±16	<0.001	85±16	81±16	<0.001	
Diabetes Mellitus n(%)	(9.9%)	(29.5%)	<0.001	(8.2%)	(30.6%)	<0.001	
Hypertension n(%)	(33.9%)	(66.2%)	<0.001	(36.1%)	(67.3%)	<0.001	
CAD n(%)	(1.9%)	(4.1%)	0.004	(4.6%)	(4.6%)	0.126	
CV Mortality n(%)	9 (2.1%)	16 (2.7%)	0.521	20 (4.2%)	26 (7.3%)	0.040	71
Total Mortality n(%)	14 (0.75%)	19 (1%)	0.986	37 (1.9%)	43 (2.3%)	0.041	113

MetS: Metabolic syndrome BMI: Body mass index. Data are shown as mean±SD or percentage values. Overall p values were calculated using an analysis of covariance test. Probability values of <0.05 were considered significant

Epidemiology

OP-014

Cardiopulmonary resuscitation in clinical practice among cardiologists

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Background and Aim: Cardiopulmonary Resuscitation (CPR) is an emergency lifesaving procedure after cardiopulmonary arrest and immediate CPR can double or triple chances of survival. For a successful resuscitation, a trained medical team and a strong collaboration between them is essential. In this study, we aimed to investigate the implementation of guidelines recommendations in real life practice among cardiologists.

Methods: A total of 80 cardiologists from 10 different medical centers (4 university hospitals, 6 education and research hospital) were included in the study. Participants were evaluated with 25 open-ended and multiple choice questions according to the European Resuscitation Council Guidelines for Resuscitation published in 2015.

Results: The total percentage of multiple choice questions answered correctly was 56%. Early defibrillation rate (within 3-5 minutes) after cardiopulmonary arrest was 65%. The routine use of peri-arrest transthoracic echocardiography to identify reversible causes of cardiac arrest was 71%. The majority of the participants (90%) declared that they use a supraglottic airway for ventilation instead of endotracheal intubation. Interestingly, in the absence of a reversible cause, the decision about withholding CPR was longer than the guideline recommendation (40 minutes vs. 20 minutes). In contrast to current guideline, the routine use of sodium bicarbonate during CPR was relatively higher (30%). None of the participants were informed about the use of waveform capnography to confirm and continually monitor tracheal tube placement, quality of CPR and to provide an early indication of return of spontaneous circulation. Of these participants, 65 (81%) stated that they did not receive any education programme about CPR in the last 1 year.

Conclusions: CPR performed by well-trained medical staff has a critical role on survival rate after cardiopulmonary arrest. In order to increase the success rate of CPR in clinical practice, clinicians should follow and update the current guidelines about CPR. Additionally, theoretical and practical educational programmes should be held in institutions regularly.

Epidemiology

OP-015

A descriptive analysis of abstracts presented at the Turkish National Cardiology Congresses between 2011 and 2015

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Background and Aim: The aim of this study was to investigate the scientific publication performance of the abstracts presented at the annual Turkish National Cardiology Congress (TNCC) between 2011 and 2015 and to analyze the variables associated with publication.

Methods: The accepted abstracts of five congresses (2011-2015) were screened using the title and names of all authors in English via PubMed and Google Scholar databases. The parameters recorded included presentation type, publication rate, time to publication, affiliated institution, journal name and average impact factor, and average citation number per year for each publication.

Results: A total of 2897 abstracts (966 oral presentations and 1931 poster presentations) were accepted in five meetings and 23.4% (n=680) of these were published in national or international peer-reviewed journals (Figure 1). Of the published articles, 32.6% (n=222) were oral presentations and 67.4% (n=458) were poster presentations. The mean time to publication of oral and poster presentations were similar [9 (0-58) vs. 8 (0-62) months, p = 0.150]. According to the type of institution, university hospitals had the highest ratio of publication (58.6%) (p<0.001). All publications were published in 148 journals from 37 different countries. The average citation number of publications was significantly higher than the average impact factor of the journals [1.4 (0-30.1) vs. 1.29 (0.11-19.8), p<0.001]. Of all publications published in 148 different international peer-reviewed journals from 37 countries, 33.2% were published in five journals [the Anatolian Journal of Cardiology, Archives of the Turkish Society of Cardiology, Angiology, Kardiologia Polska (Polish Heart Journal), Cardiology Journal].

Conclusions: Compared with other national-based literature in other medical fields, the overall publication rate was found to be similar while the time to publication was shorter. The significant difference between citation number and impact factor may be interpreted as positive indicator in terms of high level scientific value for cardiology publications presented in the TNCC. The encouragement of researchers and elimination of preclusive factors in terms of publication may improve the publication rates of the TNCC.

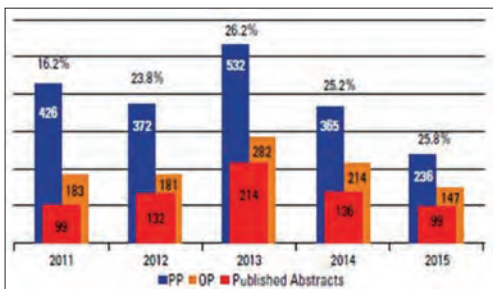
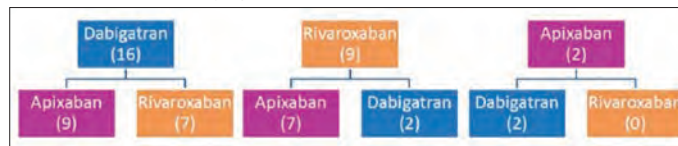


Figure 1. Percentages of abstracts published per year. OP: Oral presentation; PP: Poster presentation.

Table 3. Total bleedings was found higher in rivaroxaban arm (p=0.036)

	Dabigatran	Rivaroxaban	Apixaban	Total
Number of all patients	148	141	60	349
Bleeding rates n (%)	15 (%10.1)	28 (%19.9)	6 (%10)	49 (%14)

Table 4. Switch between NOAC groups



Epidemiology

OP-016

Real life data of NOACs: detection of inappropriate usage, patient adherence and clinicians behaviors in specific conditions

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Background and Aim: Vitamin-K antagonists(VKA) were gold standard therapy for patient with AF who need oral anticoagulant drugs for decades. Non-Vitamin-K Oral Anticoagulants (NOAC) had been compared against VKA in clinical trials and showed lower bleeding endpoints by the time reducing %10 of ischemic events in meta-analyses. Although dose reduction of NOAC is described in patients with kidney disease, real time data shows patients use more frequently lower doses independent of GFR and this is called 'inappropriate dosage' of NOAC's. In our study we aimed to research patients using NOAC for demographical characteristics, bleeding rates, ischemic endpoints, 'inappropriate dosage', drug adherence and drug changes between NOAC groups.

Methods: By searching hospital information system we found 484 patient using NOAC. We investigated their medical history and called them to complete case data report form by phone.

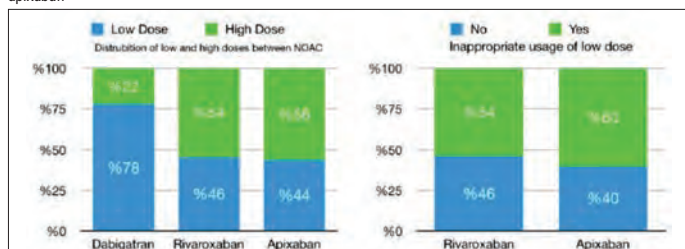
Results: Distribution of drugs was 200 (41.3%) in dabigatran arm, 202 (41.7%) in rivaroxaban arm and 82 (16.9%) in apixaban arm. Mean age, GFR (Cockcroft-Gault), CHA2DS2-VASc and HAS-BLED score established same between the NOAC groups. Mean duration of drug use was significantly lower in the apixaban group (p=0.001) (Table 1). 156 (78%) patients in dabigatran arm were using 110 mg of dabigatran. As dabigatran 110 mg is effective for ischemic endpoints comparing with warfarin, we didn't describe inappropriate dosage of dabigatran. In rivaroxaban arm 92 (45.5%) patients were using 15 mg of rivaroxaban. Inappropriate use of low dose rivaroxaban found 54.5% (n=47) in the 15 mg rivaroxaban group (n=87). In apixaban arm 36 (43.9%) patients were using 2.5 mg of apixaban. Inappropriate use of low dose of apixaban was %60 (n=21) in the low dose of apixaban group (n=35) (Table 2). There was no difference between rivaroxaban and apixaban arm for inappropriate use of low dose (p=0.346). Major bleeding rates was similar between groups, whereas clinically relevant non-major bleedings and total bleedings was higher in rivaroxaban arm (p=0.007 for non-major bleedings and p=0.015 for total bleeding) (Table 3). Ischemic endpoints were same between groups. There was no difference between groups for drug adherence. However, if the patients were grouped according to whether they have history of ischemic stroke or no previous stroke, the adherence was higher in patients with history of ischemic stroke (p=0.02). We also evaluated the rate and direction of change among NOACs. In the follow up it was observed that NOAC molecule used was replaced by another NOAC molecule in 27 patients. While the most abandoned molecule was dabigatran (p=0.004) and the most directed molecule was apixaban (p=0.013) (Table 4).

Conclusions: We found that the inappropriate use of low dose rivaroxaban and apixaban is high. Non-major bleedings were higher in rivaroxaban group. Patients with history of stroke were more adherent to NOAC therapy. In patients who change the NOAC molecule for some reason, the physicians preferred apixaban among other NOACs more often.

Table 1. Mean age, GFR (Cockcroft-Gault), CHA2DS2-VASc score and HAS-BLED score established same between the NOAC groups. Mean duration of drug use was significantly lower in the apixaban group

Drug name	NOAC usage time (months)	Patients age (years)	HAS-BLED score	GFR (Cockcroft-Gault)	CHA2DS2-VASc
Dabigatran	27,81	75,08	2,3	71,19	3,79
Rivaroxaban	25,24	75,04	2,15	69,08	3,68
Apixaban	18,70	75,06	2,24	69,24	4,05

Table 2. Distribution of doses in NOAC groups and inappropriate low dose usage data of rivaroxaban and apixaban



Epidemiology

OP-017

Evaluation of obesity, nutrition behavior and physical activity level of elementary students in Istanbul

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Background and Aim: Health related negative attitudes and behaviors causing cardiovascular disease begin to develop during childhood. The school environment has a significant position to provide adequate, balanced diet and regular exercise habits. This study was conducted to evaluate obesity, nutrition behavior and physical activity level of elementary students in Istanbul.

Methods: A school was selected from each of the districts of Istanbul that research was conducted in 39 primary schools. The permission was received from parents of students'. 5620 fourth grade elementary school students were enrolled in research. Data were collected using a questionnaire. After the anthropometric measurements of the students (height, weight) were computed. Body mass indexes (BMI) based on an age and sex specific were determined.

Results: The mean age of students 9.5 (± 0.64 range 8-14). 51.1% of students who participated in the research were female. 15.5% of the female students were overweight and 14.1% of them were obese. 15.1% of the male students were overweight and 17.3% of them were obese. In total, 3% of the students were underweight, 66.1% were in normal weight, 15.3% were overweight and 15.7% were obese. 68.4% of the students sometimes skip their meals; 36.4% of the students did not have regular breakfast, 50.9% of them sometimes eat foods such as chips, French fries potatoes. 51.9% of students play an active team games and 30.5% of them make walking less than half an hour a day. The mean durations of time spent on watching TV and computer were 1.6 hours (weekday), 1.9 hours (weekend) and 1.0 hours (weekday), 1.2 hours (weekend) respectively. It was found that the body mass index of students increases as durations of time spent on watching TV (r=0.064, p<0.05) and computer (r=0.037, p<0.05) is increasing. In addition, the BMI was differentiated by gender (p=0.004), male students had a higher BMI compared to female students, BMI was higher in those who skipped main meals (p=0.001) and those who did not eat regular breakfast (p=0.001).

Conclusions: Obesity, a serious health problem and the main risk factor for cardiovascular diseases, that has been found considerably high percentages in children. For this reason, to maintenance of heart health may be recommended that take part programs saving healthy eating habits and increasing the activity level in of the school curriculum.

Interventional cardiology / Carotid and peripheral vascular

OP-018

Management of large vessel occlusions: Safety of endovascular stroke procedures after Iv-tPA treatment

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Background and Aim: Following the positive results from recent trials on endovascular therapy (EVT), bridging therapy (intravenous alteplase plus EVT) is increasingly being used for the treatment of acute ischemic stroke. We investigated the efficacy and safety of combined intravenous (IV) recombinant tissue plasminogen activator (rtPA) and simultaneous endovascular therapy (EVT) for acute large vessel occlusion.

Methods: Between April 2016 and May 2018, Ninety-seven acute stroke patients treated endovascularly in our hospital were evaluated retrospectively. Patients data regarding age, sex, and medical history, as well as thrombus location by Computed tomography angiography(CTA), postprocedural reperfusion status, and clinical outcomes were reviewed.

Results: Thirty one patients (31%) received IV tPA before thrombectomy and sixty six patients did not qualify for IV tPA (68%). Successful recanalization (thrombolysis in cerebral infarction 2b/3) was achieved in 78% (52/66) patients of thrombectomy patients without preprocedural IV tPA and 77% (24/31) of patients who received it (p=0.06). MRS of 2 or less at 90 days was 72% (48/66) in patients with no preprocedural IV tPA and 66% (22/33) in the combined therapy group (p<0.05). Symptomatic hemorrhage occurred in 15.1% (5/33) of patients with preprocedural IV tPA; and 16.6% (11/66) in the patients without preprocedural Iv tPA (p=0.08).

Conclusions: There was no difference in outcome in patients with large-vessel occlusion stroke treated with direct EVT compared with those treated with bridging thrombolysis. Endovascular therapy can achieve good clinical outcomes in patients with acute large-artery occlusion ineligible for IV thrombolysis.

Interventional cardiology / Carotid and peripheral vascular

OP-019

Emergent revascularization procedures of cerebral large vessel artery tandem occlusions

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Background and Aim: Tandem occlusions involving both the extracranial internal carotid artery (ICA) and an intracranial artery typically respond poorly to intravenous (IV) tissue plasminogen activator (t-PA). We retrospectively review our experience with proximal ICA stenting or only balloon angioplasty and stent-assisted thrombectomy of the distal artery.

Methods: The data included patients that underwent carotid balloon angioplasty without carotid stenting and mechanical thrombectomy between 2016–2018. Radiographic, clinical, and procedural data were drawn from case notes, imaging records and discharge reports. Clinical outcomes were evaluated using the National Institutes of Health Stroke Scale (NIHSS) and the modified Rankin scale (mRs).

Results: Sixteen patients, with a mean age of 65.2 years and a mean admission NIHSS of 15 underwent this procedure and were included. Each presented with an occlusion of the proximal ICA, with additional occlusions of the ICA terminus (n=4), middle cerebral artery (n=12). Recanalisation of all identified occlusions was achieved in all patients, with a Thrombolysis in Cerebral Infarction (TICI) score >2b achieved in each case. Mean time from onset of stroke symptoms to recanalisation was 282 min; mean time from first angiography to recanalisation was 70 min. Thirteen patients underwent stenting one week later. Two patients were medically followed because the residual stenosis was 50% after balloon angioplasty. Intracranial haemorrhages occurred in two patients, with no increase in NIHSS. There were no mortalities. Mean NIHSS at discharge was 4.9, and mRs at 90 days was <2 in all patients.

Conclusions: Treatment of tandem extracranial ICA and intracranial occlusions in the setting of acute ischaemic stroke with extracranial carotid artery balloon angioplasty followed by adjunctive intracranial mechanical thrombectomy is both safe and effective, but further evaluation of this treatment modality is necessary.

Interventional cardiology / Carotid and peripheral vascular

OP-020

Clinical outcomes of extended endovascular recanalization of 16 consecutive Buerger's disease patients

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Background and Aim: We aimed to present the clinical outcomes of 16 consecutive Buerger's disease patients underwent extended endovascular recanalization.

Methods: A total of 16 consecutive patients with confirmed diagnosis of Buerger's disease that percutaneously treated in our center between February 2014 and March 2018 were included to the study. All the patients had a history of smoking while one of them quit 1 year ago. Two of the patients had hypertension and three of them were obese (BMI ≥ 30 kg/m²). After physical examination and complementary diagnostic tests, performance of extended angioplasty for occluded arteries was intended to restore direct blood flow through at least one of the blow-the-knee arteries.

Results: A successful extended endovascular treatment was performed in 20 of 22 limbs, achieving a technical success of 91%. All the patients were successfully discharged without mortality and any complication. During a mean follow-up of 21.43 months (standard error: ± 7.08) reintervention was needed in those 20 successfully treated limbs and one minor amputation needed in one of the failed limbs. Limb salvage rate was 100%. A statistically significant difference was observed in Rutherford classification, ankle brachial index, direct blood flow to foot, presence of ulcer and rest pain before and after the intervention.

Conclusions: We showed successful extended endovascular recanalization of Buerger's disease patients with a high technical success rate and sustained clinical improvement. Extended endovascular recanalization could be a therapeutic option in Buerger's disease patients since they are not good candidates for surgery.

Table 1. Demographic characteristics of patients

Demographic and risk factors	Patients (n=16)
Age, (yr)	44.23 \pm 4.28
Male, n (%)	16 (100)
Hypertension, n (%)	2 (12.5)
Diabetes Mellitus, n (%)	0 (0)
Hypertension, n (%)	0 (0)
Coronary Artery Disease, n (%)	0 (0)
Obesity (BMI ≥ 30 kg/m ²), n (%)	3 (18.8)
Chronic kidney disease, n (%)	0 (0)
Smoking	16 (100)
Current, n (%)	14 (87.5)
Former, n (%)	2 (12.5)
Previous PTA attempt, n (%)	1 (6.3)
Carotid stenosis, n (%)	19 (96.4)
Type I	4 (20.0)
Type II	8 (40.0)
Type III	4 (20.0)
Type IV	3 (15.0)
Rutherford classification, n (%)	16 (100)
Class 0-2 (mild to moderate classification)	0 (0)
Class 3 (severe classification)	0 (0)
Class 4 (ischemic rest pain)	1 (6.3)
Class 5 (minor limb loss)	16 (100)
Class 6 (major limb loss)	0 (0)
Target arteries, n (%)	Number=46
Superficial femoral artery	24 (3)
Popliteal artery	16 (3)
Anterior tibial artery	12 (3)
Posterior tibial artery	14 (3)
Femoral artery	12 (3)
Tibioperoneal trunk	12 (3)
Distal popliteal artery	24 (3)
Femoral artery	12 (3)
Type of technique used for PTA, n (%)	16 (100)
Fluorocath	16 (100)
Subcutaneous Loop	2 (12.5)
Parallel wire	14 (87.5)
Anchor wire	14 (87.5)
Kissing	2 (12.5)
Reverse CART	2 (12.5)
Direct catheter	4 (25.0)
Secondary stent implantation, n (%)	0 (0)
Failed to restore direct blood flow, n (%)	2 (12.5)
Technical success rate, n (%)	20 (100)
Complication associated to intervention (stroke, CVA), n (%)	0 (0)
Access site complications, n (%)	0 (0)
Number of amputations (minor, n (%) major, n (%)	1 (6.3) 0 (0)
Limb salvage rate, n (%)	20 (100)
Death during follow up, n (%)	0 (0)
Reintervention, n (%)	14 (87.5)
Duration of follow-up (mean \pm SD), months	21.43 \pm 7.08

BMI: body mass index, PTA: percutaneous transluminal angioplasty, CART: controlled angioplasty and retrograde tracking, CVA: cerebral vascular accident

Table 2. Clinical outcomes of patients

	Pre Intervention	1 st month	3 rd month	6 th month	1 year	P
Rutherford Classification	4.81 \pm 0.50	3.51 \pm 0.73	3.40 \pm 0.79	3.36 \pm 0.78	3.27 \pm 0.76	<0.05
Ankle Brachial Index (ABI)	4.04 \pm 1.21	9.18 \pm 2.34	8.86 \pm 2.33	8.50 \pm 2.01	8.40 \pm 1.96	<0.05
Triphasic Blood flow, Revascularization (%)	0 (0)	20/22 (91)	20/22 (91)	20/22 (91)	20/22 (91)	<0.05
Ulcer, Revascularization (%)	17/22 (77.3)	15/22 (68.2)	7/22 (31.9)	2/22 (9.1)	2/22 (9.1)	<0.05
Rest Pain, Revascularization (%)	22/22 (100)	2/22 (9)	1/22 (4.5)	1/22 (4.5)	1/22 (4.5)	<0.05
Smoking, Revascularization (%)	15/16 (93.7)	6/16 (37.5)	6/16 (37.5)	6/16 (37.5)	6/16 (37.5)	<0.05

Interventional cardiology / Carotid and peripheral vascular

OP-021

Hybrid approach in thrombotic critical hand ischemia

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Background and Aim: Critical hand ischemia represents a complex medical problem for even the experienced clinician. The rarity of hand ischemia, the small caliber of the distal vasculature, and the limited surgical options available for treatment all compound the difficulty of intervention. Percutaneous transluminal angioplasty (PTA) is now considered a first-line therapy for above-the-elbow artery disease while there are few data regarding the treatment of below-the-elbow (BTE) arteries. The aim of this study is to review the current literature on BTE vessel and early outcomes and comparison of three different treatment options; 1) standard endovascular 2) standard endovascular angioplasty and 3) endovascular angioplasty approach including catheter-based thrombolysis plus iloprost.

Methods: A prospective review of 14 patients treated with standard embolectomy (group-1), 11 patients treated with angioplasty (group-2) and 12 patients treated with endovascular angioplasty approach including catheter-based thrombolysis plus iloprost (group-3) was performed. Procedural success, procedural durations, patency estimates, complications, amputation rates and permanent disability were compared and analyzed.

Results: When the standard embolectomy (group-1) and standard endovascular angioplasty group (group-2), was compared with endovascular angioplasty approach including catheter-based thrombolysis plus iloprost group(group-3). Amputation rates and permanent disability were similar in group 1 and group 2 and higher than group 3 (p<0.001). But procedure duration and bleeding was significantly increased in group-3 (p<0.001).

Conclusions: Endovascular angioplasty including catheter-based thrombolysis plus iloprost treatment of below-the-elbow arteries is effective than the standard embolectomy or standard endovascular angioplasty in patients with critical hand ischemia, with an elevated immediate technical success and satisfactory clinical results.

Interventional cardiology / Carotid and peripheral vascular

OP-022

Comparison of automated oscillometric device with standard Doppler method to measure ankle brachial index in peripheral arterial disease

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Background and Aim: Ankle Brachial Index (ABI) is a widely used method for diagnosis and prediction of peripheral arterial and cardiovascular diseases. The objective of our study is to compare the Doppler method of ABI measurement with more practical automated oscillometric method of abi measurement.

Methods: Between January 2016 and September 2017, 107 patients (214 lower extremities) who applied to our outpatient clinic for peripheral arterial disease were evaluated by both oscillometric and doppler ABI measurements. In oscillometric measurement of ABI calcification of the lower extremities were excluded from the analysis as an oscillometric code error; thus only data from 200 lower extremities were analyzed.

Results: The mean age of the patients was 62.2±11.31, 59.8% were diabetic, 63.6% were hypertensive and 24.3% were female patients. Compared to gold standard Doppler method of ABI measurement in the diagnosis of peripheral arterial disease, the sensitivity and specificity of oscillometric ABI method was 78.33% and 85% respectively, with positive predictive value of 88.67 and negative predictive value of 72.33. The positive likelihood ratio was 5.2 while the negative likelihood ratio was 0.25. A significantly good correlation was observed between the both methods [Pearson correlation coefficient: 0.740 (p=0.000)]. The mean oscillometric and Doppler ABI measurement were determined as 0.879±0.210 and 0.878±0.223 respectively. With Bland-Altman method the mean difference between the Oscillometric and Doppler ABI methods was 0.0013±0.3. By ROC analysis; the area under the curve was 0.883 [p=0.000, 95% CI: 0.835-0.930], specificity was 0.74 and sensitivity was 0.92, and the cutoff value for the oscillometric ABI was 0.875.

Conclusions: It was observed that the oscillometric ABI method was highly correlated with the gold standard Doppler ABI method and statistically a significant measure of sensitivity and specificity was observed in diagnosis of the peripheral arterial diseases. The need for skilled medical staff and technical difficulties and time consuming nature of the doppler method causes significant limitations in its use, in contrast, oscillometric measurement of the ABI allows for more practical, quicker and precise results, emergence of these oscillometric devices can be a good alternative to doppler ABI method.

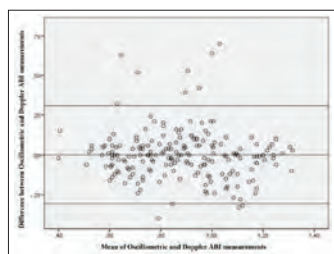


Figure 1. Bland and Altman graphic of Oscillometric and Doppler ABI.

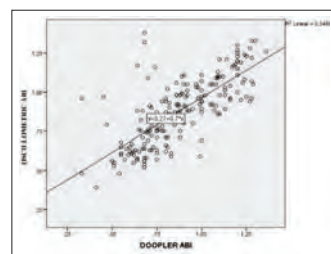


Figure 2. Oscillometric and Doppler ABI correlation graphic.

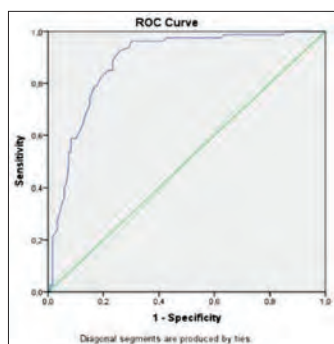


Figure 3. ROC curve for Oscillometric ABI.

Table 1. Oscilometric ABI sensitivity, specificity, positive predictive value (PPV) ve negative predictive value (NPV)

	Doppler ABI ABI<0,9	Doppler ABI ABI>0,9	
Oscilometric ABI	ABI<0,9	94	12
Oscilometric ABI	ABI>0,9	26	68
			PPV: %88,67
			NPV: %72,34
			Sensitivity: %78,33
			Specificity: %85
			+LHR: 5,2
			-LHR: 0,25

Interventional cardiology / Carotid and peripheral vascular

OP-023

Feasibility of the left distal radial artery Access site in terms of complications and radiation exposure: A comparison evaluation with the right radial artery and the right femoral artery in coronary angiography and interventions

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Background and Aim: Transradial approach is shown to be more beneficial than the transfemoral approach in terms of vascular complications. Almost all of these studies are made with the right radial artery. Our aim of this study was to compare the new Access site; the left distal artery with the right radial artery and the right femoral artery approach in terms of complications and radiation exposure.

Methods: Sixty one (61) Patients eligible for coronary angiography and or intervention were included in our study from May 2017 to June 2018. They were divided into three (3) groups according to the Access artery site. Different operators with different experiences performing the procedure were left blind of this study and freely decided the access artery site. The sheath size was 6 french for both the radial and femoral approach. Patients in which the left distal radial artery was chosen as the access site had their left arm gently bent into their right groin with slight adduction and comfortable position of the hand. The operator stood at the right side of the patient where he could make the arterial puncture and continue with coronary interventions. All patients with a transradial approach had a cocktail of weight adjusted heparine, nitrate and serum physiologic to prevent radial artery occlusion. Demographic features, complications and radiation exposure were recorded during the hospital stay. To compare complications and radiation exposure we made multiple comparisons between the three groups by using Bonferroni and Dunnett T3 test with the SPSS 25 program and a significant value was defined as <0.05.

Results: Mean age was 59.4 and 78.6 % of them was male. Right radial artery was used in 19 patients. Twenty (20) patients had left distal artery used as the access site. In the other 22 patients right femoral artery was used. The most common cardiovascular risk factors were Diabetes and Hypertension with 36% and 77% respectively. Almost one third of patients had coronary artery disease with previous intervention. Coronary intervention was contemplated in 40 patients where the left distal radial artery was the most common access site (15 patients). Only one (1) radial spasm and hematoma occurred in the right radial Access site. Femoral hematoma was encountered in only one patient without in that manual compression for a short time lead to recovering of the hematoma. Fluoroscopy time, total air kerma and total dose area product was found similar between the three groups without any significant difference. Only the left distal radial artery Access site showed a trend towards radiation exposure (total dose area product; p=0.65). Patients were discharged in a mean time of 3 days.

Conclusions: Although a small population we found that the newly prescribed Access site; the left distal radial artery is associated with no higher fluoroscopy time and radiation exposure in comparison with the right radial artery and the right femoral artery.



Figure 1. Operator on the right side of the patient performing coronary intervention with left distal radial access.



Figure 2. The introduction of 6 French hydrophilic radial sheath into the left distal radial artery in the anatomical snuffbox.

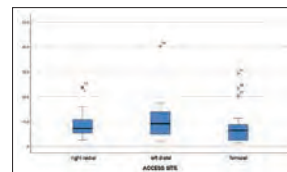


Figure 3. Fluoroscopy time between the three groups.

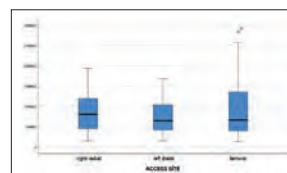


Figure 4. Radiation exposure (total air kerma) between the three groups.

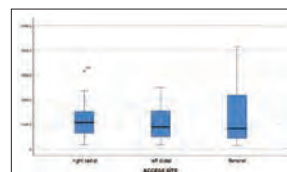


Figure 5. Radiation exposure (total dose area product) between the three groups.

Table 1. Demographic features of study population

Age	20-85 (59.4±11.5)
Male	48 (78.6%)
Female	23 (21.4%)
LVEF	30-60 (47.8±8.32)
Hospital stay (days)	0-13 (3.05±2.75)
DM	22 (36%)
HT	47 (77%)
Smoking	9 (14.7%)
CAD	14 (22.9%)

Table 2. Procedural features during coronary angiography and or intervention n=61 (%)

Anterior STEMI	8 (13.1%)
Inferior STEMI	8 (13.1%)
Non-STEMI	6 (9.8%)
UAP	2 (3.2%)
PCI (total)	40 (65.5%)
LAD intervention (total)	20 (32.7%)
CX intervention (total)	6 (9.8%)
RCA intervention (total)	14 (22.9%)

Table 3. Radiation exposure between the three groups

	Right radial artery	Left distal radial artery	Right femoral artery
Fluoroscopy time (minutes)	9.24±6.31	10.46±8.36	7.98±7.29
Radiation exposure (Total air kerma: mGy)	8571.6±5014.27	7573.8±4419.6	9466.3±7806.6
Radiation exposure (Total dose area product: µGy·m2)	1164.4±780.4	1055.9±717.01	1267.9±1117.2

Table 3. Complications and radiation exposure

Radial spasm	1
Radial occlusion	0
Radial hematoma	1
Femoral hematoma	1
Fluoroscopy time (minutes)	1.1-40.1 (9.18±7.33)
Radiation exposure (Total air kerma: mGy)	1314.6-28337.4 (8567.1±5990)
Radiation exposure (Total dose area product: µGy·m2)	138.1-4142 (1166.18±888.99)

Interventional cardiology / Carotid and peripheral vascular

OP-024

Diagnosis and management of subclavian artery occlusive disease:
A single center's experience

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Background and Aim: The incidence of subclavian artery stenosis (SAS) ranges between 2% and 4% in the general population. The frequency of SAS increases in patients with peripheral artery disease. Although SAS is usually asymptomatic, vertebrobasilar insufficiency and upper limb ischemia symptoms may occur due to retrograde blood flow in the ipsilateral vertebral artery (subclavian steal syndrome). Also, SAS is an important cause of ischemic heart disease and angina pectoris in patients with internal mammary artery (IMA) grafts due to the same pathophysiological pathway. Only the patients suffering from these symptoms should be treated. Endovascular revascularization has become the treatment of choice for SAS in recent years with the evolution of devices such as low-profile balloon expandable stents, guiding catheters and guidewires. The literature from our country mainly consists of case reports. There are only few studies evaluating the underlying risk factors, clinical characteristics, angiographic findings and technical details of the endovascular procedure in patients with SAS. The aim of this study was to evaluate the patients with SAS and determine the risk factors, diagnostic tools, lesion characteristics and procedural success rates of these patients treated at our tertiary referral center.

Methods: In the present retrospective study, we searched our hospital's database for subclavian stenosis between the years of 2015 and 2018 and the data of 32 consecutive symptomatic patients treated with endovascular therapy were reviewed.

Results: A total number of 32 patients (59.4% men) were included in the study. Mean age was 63.2±8.9 years (range: 43-79). Dyslipidemia (62.5%) was the most prevalent cardiovascular risk factor in our SAS patients. There was 14 (43.8%) patients with a history of significant coronary artery disease and 5 (15.6%) patients with previous carotid stenting. The frequency of multivessel disease in coronary artery disease patients were 92.8% (13 of 14 patients). Upper extremity claudication (59.4%) was the most frequent symptom in our SAS patients. Anterograde approach from common femoral artery was performed in 27 (84.4%) patients. Procedural success was achieved in 29 of 32 patients (90.6%). Total occlusion of the subclavian artery was observed in 7 of 32 patients (21.9%) and only 4 of these 7 patients were successfully revascularized. Access site complication (1 of 32) and upper extremity embolism (1 of 32) were detected as procedure related complications. There was no procedure related death, rupture and dissection involving thoracic aorta.

Conclusions: Endovascular management of SAS is an effective treatment strategy with high procedural success and low complication rates. Total occlusion of the subclavian artery presents a particular challenge and procedural success of total occlusion is heavily dependent upon operator's experience.

Table 1. Demographics and clinical characteristics of patients with subclavian artery stenosis

Variable	n/32
Age, years	63.2 ± 8.9
Male, n(%)	19 (59.4)
Hypertension, n(%)	19 (59.4)
Diabetes Mellitus, n(%)	10 (31.3)
Dyslipidemia, n(%)	20 (62.5)
Smoking, n(%)	16 (50)
Renal disease, n(%)	0 (0)
Previous PCI, n(%)	8 (25)
Previous CABG, n(%)	10 (31.3)
Previous CAD, n(%)	14 (43.8)
Previous carotid stenting, n(%)	5 (15.6)
Lower extremity arterial disease, n(%)	1 (3.1)
CVA, n(%)	2 (6.3)
TIA, n(%)	2 (6.3)
Myocardial infarction, n(%)	8 (25)
CHF, n(%)	3 (9.4)
Multivessel disease, n(%)	13 (40.6)
Ejection fraction, %	55.3 ± 11.3
Indications n(%)	n:32
Upper extremity symptoms	19 (59.4)
Vertebrobasilar insufficiency symptoms	15 (46.9)
Coronary ischemia symptoms	5 (15.6)
Before the use of the left internal mammary artery in bypass grafting	2 (6.3)
Diagnostic tools	n:32
Doppler USG	19 (59.4)
CT angiography	18 (56.3)
Conventional angiography	5 (15.6)

Table 2. Angiographic features and procedural details of patients with subclavian artery stenosis

Variable	n:32
Anterograde CFA access, n(%)	27 (84.4)
Dual access, n(%)	2 (6.3)
Guiding catheter based system, n(%)	23 (71.9)
Procedural success, n(%)	29 (90.6)
Predilatation, n(%)	17 (53.1)
Balloon expandable stent, n(%)	27 (84.4)
Percentage of stenosis, %	87.9 ± 11.1
Total occlusion, n(%)	7 (21.9)
Balloon size, mm	5.4 ± 1.1
Stent size, mm	7.6 ± 0.7
Stent length, mm	27.7 ± 7.3
Lesion length, mm	20.7 ± 7.8
Stenosis at the origin of subclavian artery, n(%)	7 (21.9)
Stenosis between origin and vertebral artery, n(%)	32 (100)
Complications	n:32
Upper extremity embolism, n(%)	1 (3.1)
Cerebral embolism, n(%)	0 (0)
Access site complication	1 (3.1)
Transfusion	2 (6.3)

PCI:percutaneous coronary intervention, CABG: coronary artery bypass graft, CAD: coronary artery disease, CVA: cerebrovascular accident, TIA: transient ischemic attack, CHF: congestive heart failure; CFA: common femoral artery.

Interventional cardiology / Carotid and peripheral vascular

OP-025

Neurological complications following carotid artery stenting
and their relationship with risk factorsEmrah Bayam,¹ Ender Ozgun Cakmak²¹Department of Cardiology, S.B. Ümraniye Training and Research Hospital, Istanbul²Department of Cardiology, Kartal Koşuyolu Yüksek İhtisas Training and Research Hospital, Istanbul

Background and Aim: Complexity of coronary artery disease (CAD) and lesion characteristics are predictors of periprocedural complications and long-term mortality. The Syntax score is a universal angiographic scoring system that is derived completely from the coronary anatomy and lesion characteristics. There have been a few previous study investigating the association between Syntax score and carotid artery stenting complications. We aimed to examine the correlation between Syntax score and carotid artery stenting neurologic complications.

Methods: We retrospectively included 402 patients admitted to Kartal Koşuyolu Heart Training and Research Hospital from January 2012 to January 2016 who underwent coronary angiography, carotid angiography and carotid artery stenting. Demographic and clinical features of the patients (age, gender, diabetes mellitus, hypertension, hyperlipidemia, peripheral arterial disease, chronic kidney disease, history of smoking) were determined by retrospective chart review. Patients with dual antiplatelet therapy is not available (active bleeding, recent intracranial hemorrhage, advanced Alzheimer's disease), Takayasu arteritis and fibromuscular dysplasia were excluded from the study. Carotid artery stenosis was assessed according to the North American Symptomatic Carotid Endarterectomy Study (NASCET) criteria. Total occluded carotid artery lesions were not intervened. Patients were compared in terms of sex, age, coronary artery bypass graft (CABG), diabetes mellitus, hyperlipidemia, hypertension, peripheral arterial disease, coronary artery disease and Syntax scores (low <22, middle 22-32, high >32).

Results: 32 of 402 patients undergoing carotid artery stenting had neurological complications (7.9%). Neurological complications were: stroke (24 patients 5.9%), transient ischemic attack (7 patients 1.7%), convulsions (2 patients 0.5%). Four (4) patients were exitus (1%). In our study, high syntax score (p=0.029) was found in patients with neurological complications. The cigarette risk factor in high syntax score was significant (p=0.33); diabetes mellitus was found to be a significant risk factor for moderate and high syntax scores (p=0.015). Gender, age, hyperlipidemia, hypertension, CABG, peripheral artery disease, coronary artery disease were not found to be significant.

Conclusions: In conclusion, the Syntax score is well associated with the severity of coronary artery disease. Syntax score may be predictive for carotid artery stenting neurologic complications. The most important limitation of our study is retrospective and single-centered.

Interventional cardiology / Carotid and peripheral vascular

OP-026

The relationship among neutrophil to lymphocyte ratio, stroke territory
and 3-months mortality in patients with acute ischemic strokeOzcan Kocaturk,¹ Feyzullah Besli,² Fatih Gungoren,² Mehtap Kocaturk,¹ Zulkif Tanirverdi¹¹Department of Neurology, Harran University Faculty of Medicine, Şanlıurfa²Department of Cardiology, Harran University Faculty of Medicine, Şanlıurfa

Background and Aim: Stroke therapy options have focused on limiting the infarct volume. Neutrophil to lymphocyte ratio (NLR) can be valuable to detect the patients that required intensive treatment at early stage by predicting infarct volume. The aim of this study is to evaluate the relationship between NLR and infarct volume according to the stroke territory, and to determine the prognostic value of NLR for predicting 3-months mortality in acute ischemic stroke (AIS) patients.

Methods: A total of 107 patients with AIS were enrolled and followed up 3-months in terms of mortality. Study population was divided into two groups according to the stroke territory: anterior circulating stroke (ACS, n=84) and posterior circulating stroke (PCS, n=23). PCS was defined as ischemia in the vascular territory of basilar, vertebral, or posterior cerebral arteries. ACS was defined as ischemia in the vascular territory internal carotid, middle, or anterior cerebral arteries. All patients underwent diffusion magnetic resonance imaging. Laboratory parameters were analyzed after a 12-hour overnight fasting.

Results: A total of 107 patients were enrolled in this study and followed up 3-month in terms of mortality. The mean age of the patients were 67 (55-74) years. 84 (78.5%) had ACS, whereas 23 patients (21.5%) had PCS. There was no significant difference between ACS and PCS groups regarding baseline characteristics, and co-morbid diseases. Also, laboratory parameters including NLR and CRP were similar between ACS and PCS groups. In correlation analysis, infarct volume was positively correlated with CRP (r=0.350, p=0.001) and NLR (r=0.482, p<0.001) in ACS group, but there was any correlation between these parameters in PCS group. Multivariate logistic regression analysis demonstrated that NLR was the only independent predictor of 3-months mortality. ROC curve analysis was used to detect the sensitivity and specificity of the NLR for predicting 3-months mortality (Table 1). NLR ≥ 4.7 predicted 3-months mortality with a specificity of 76.5% and sensitivity of 63.6% (Figure 1).

Conclusions: In conclusion, NLR is significantly correlated with ACS infarct volume, whereas not correlated with PCS infarct volume. In addition, it is an independent predictor of 3-months mortality in AIS patients. Therefore, it may guide the physician deciding the necessity of endovascular intervention particularly in ACS patients.

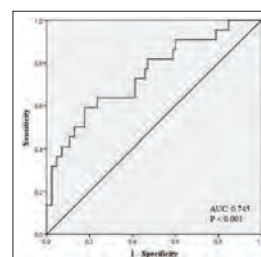
**Figure 1.** ROC curve of neutrophil to lymphocyte ratio ≥4.7 for predicting 3-months mortality.

Table 1. Univariate and multivariate logistic regression analysis showing the independent predictors of 3-months mortality

	Univariate			Multivariate		
	OR	95% CI	P	OR	95% CI	P
Age	1.049	1.008-1.092	0.019	1.015	0.971-1.061	0.515
Male gender	1.874	0.723-4.855	0.196			
Hypertension	1.528	0.591-3.950	0.382			
Diabetes Mellitus	0.729	0.272-1.955	0.530			
Heart failure	0.366	0.057-2.338	0.288			
Atrial fibrillation	6.144	1.810-20.858	0.004	2.426	0.574-10.261	0.228
NIHSS	1.082	0.990-1.183	0.082	1.025	0.927-1.134	0.628
Stroke territory	1.295	0.391-4.291	0.672			
Therapy strategy	0.913	0.297-2.813	0.875			
Infarct volume	0.994	0.980-1.008	0.388			
CRP	2.345	1.248-4.408	0.008	2.046	0.991-4.221	0.053
NLR	1.245	1.090-1.421	0.001	1.186	1.032-1.363	0.016

OR: Odds ratio; CI: Confidence interval; NIHSS: The National Institutes of Health Stroke Scale; CRP: C-reactive protein; HLR: Neutrophil to lymphocyte ratio.

Interventional cardiology / Carotid and peripheral vascular

OP-027

Investigation of risk factors with long-term follow-up and restenosis rate after stent implantation in patients with iliac artery stenosis

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Background and Aim: We aimed to investigate the stent patency rates of patients with symptomatic iliac artery stenosis or occlusion and revascularized with stent implantation in our clinic and to define the demographic, anatomical and procedural characteristics of the patients to assess the possible risks of stenosis in patients with restenosis.

Methods: We included 58 patients with iliac artery stenosis and critic limb ischemia or patients who underwent stent implantation due to claudication that decreased the quality of life despite the medical treatment between January 2013 and January 2016 years at Kocaeli University Cardiology Clinic. Patients with history of endovascular intervention in the target artery, surgical revascularization history, acute thrombus, and aneurysm in target vasculature were excluded from the study. Loss of stent patency was defined as 2,4-fold increase in flow velocity in Doppler ultrasonography, demonstration of occlusion in contrast-enhanced angiography or >50% stenosis of vessel lumen. Demographic characteristics, lesion characteristics, procedural procedures and the amount of restenosis developed in follow-up and factors affecting restenosis were investigated retrospectively.

Results: When the stent patency rates were compared according to the clinical and procedural characteristics of the patients; restenosis was not detected in 26 (89.6%) patients with common iliac artery stent implantation, in 8 (66.6%) patients with lesions involving main iliac and external iliac artery and in 15 (100%) patients with external iliac artery stenting (p=0.034). In addition, restenosis was not observed in 6 (54.5%) of the patients with multiple stenting and 43 (95.5%) of those who had single stent (p=0.002). There was no statistically significant difference in restenosis in terms of other parameters.

Conclusions: It has been shown that endovascular procedures can be safely used with high success rates as a strategy of revascularization in iliac artery stenosis, with or without a totally occluded lesion in each TASC group lesion.

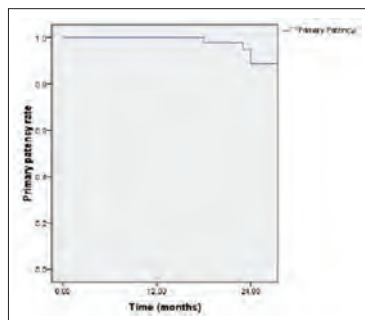


Figure 1. Primary patency at 24 months.

Table 1. Demographic data

Age (mean± SD)	60,31±8,74
Male gender (n, %)	49 (%84,5)
Smoking (n, %)	45 (%77,6)
Coronary artery disease (n, %)	33 (%56,9)
Diabetes Mellitus (n, %)	20 (%34,5)
Ankle-brachial index (mean± SD)	0.58 (0.52-0.64)
Hyperlipidemia (n, %)	37 (%63,8)
Rutherford class (n, %)	
Class 2	8 (%13,8)
Class 3	38 (%65,5)
Class 4	6 (%10,3)
Class 5	5 (%8,6)
Class 6	1 (%1,7)

Table 2. Lesion characteristics

Target arteries involved	
Common iliac artery (n, %)	30 (%51,7)
Common and external iliac artery	12 (%20,7)
External iliac artery	16 (%27,6)
Occlusion (n, %)	24 (%41,4)
Lesion length in mm	45,0 (30,0-80,0)
Reference target lumen in mm	7,0 (7,0-8,0)
Average degree of stenosis (n, %)	90,0 (80,0-100,0)
Calcification (n, %)	
None	37 (%63,8)
Mild	13 (%22,4)
Moderate	3 (%5,2)
Severe	5 (%8,6)
TASC Classification (n, %)	
A	18 (%31,0)
B	7 (%12,1)
C	13 (%22,4)
D	20 (%34,5)

Table 3. Primay patency rates according to clinical characteristics

	Primary Patency Rates	p value
Target artery involvement (n, %)		0,034
Common iliac artery	26 (%89,6)	
Common and external iliac artery	8 (%66,6)	
External iliac artery	15 (%100)	
Stent type (n, %)		0,610
Balloon expandable stent	9 (%88,8)	
Self expandable stent	40 (%81,8)	
TASC Classification (n, %)		0,170
A	17 (%94,4)	
B	6 (%100)	
C	12 (%92,3)	
D	14 (%73,6)	
Calcification (n, %)		0,152
None	33 (%91,6)	
Mild	9 (%69,2)	
Moderate	3 (%100)	
Severe	4 (%100)	
Occlusion (n, %)		0,110
Yes	18 (%78,2)	
No	31 (%93,9)	
Number of stents		0,002
Single	43 (%95,5)	
Multiple	6 (%54,5)	

Cardiac imaging / Echocardiography

OP-028

An investigation of right ventricular dysfunction with novel echocardiographic parameters in patients with mitral stenosis

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Background and Aim: Although right ventricular (RV) function is affected early in mitral stenosis (MS), there are not enough studies showing this situation. In this study, we aimed to investigate the correlation between conventional echocardiographic parameters and tricuspid annular plane systolic excursion (TAPSE) which is a sensitive indicator of RV dysfunction.

Methods: We enrolled 59 consecutive patients with MS in this study. Two groups were generated according to TAPSE value 16. (Group 1: TAPSE <16, Group 2: TAPSE >16). And the association between RV dysfunction and echocardiographic parameters were investigated.

Results: The mean age of the study population was 42.2±8. And 74.6% of patients were female. In univariate analysis; maximal mitral valve gradient, mean mitral valve gradient, systolic pulmonary arterial pressure, RV strain and RV strain rate were associated with RV dysfunction. In multivariate analysis both strain parameters were found to be independent predictors of RV dysfunction. In Kaplan Maier survival analysis, we showed that patient with lower RV strain had more rehospitalization rate in one year follow up period.

Conclusions: RV dysfunction is common in patients with MS and this is associated with mortality and morbidity and evaluation of RV strain and strain rate for early detection of RV dysfunction may be an appropriate approach in mitral stenosis.

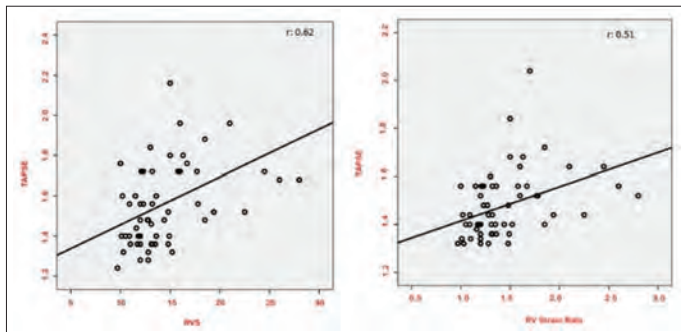


Figure 1. The correlation plots of tricuspid annular plane systolic excursion (TAPSE) and right ventricular(RV) strain (A)/ strain rate (B).

Cardiac imaging / Echocardiography

OP-030

Presystolic wave is associated with subclinical left ventricular dysfunction assessed by myocardial performance index in Type 2 diabetes mellitus

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Background and Aim: Myocardial performance index (MPI), demonstrates both systolic and diastolic functions of the left ventricle. Presystolic wave (PSW) is frequently detected on Doppler examination of the left ventricular outflow tract and possible mechanism of PSW is impaired LV compliance and left ventricular stiffness (Figure 1). In this study, we aimed to investigate the relationship between PSW and MPI in type 2 diabetic patients.

Methods: A total of 129 type 2 diabetic patients were included in this study. Patients were divided into two groups according to the presence of PSW on doppler echocardiography. There were 90 patients (38 male, mean age 57.77±10.91) in the PSW-positive group and 39 patients (13 male; mean age: 55.31±11.29) in the PSW-negative group.

Results: MPI was higher in PSW- positive group (0.63±0.17vs 0.52±0.13, p<0.001). In addition, subclinical left ventricle dysfunction (LVD) was higher in the PSW- positive group (p=0.029)(Table1). Pearson correlation analysis showed that PSW velocity correlated with MPI (r=0.286, p=0.006). Univariate analysis showed that the independent predictor of abnormal MPI was PSW (95% CI: 0.183-0.922, p=0.031).

Conclusions: Presence of the PSW on doppler examination was associated with subclinical LV dysfunction in patients with DM type 2. This easy-to-perform echocardiographic parameter may predict subclinical LVD among patients with type 2 DM.

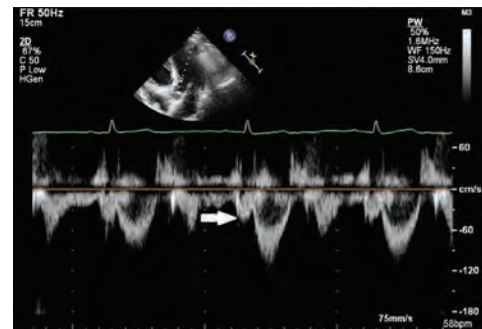


Figure 1. Arrow shows the PSW. PSW, presystolic wave.

Table 1.

	PSW - negative (n = 39)	PSW - positive (n = 90)	p
Age (years)	55.31 ± 11.29	57.77 ± 10.91	0.190
Sex, male	13	38	0.343
Hypertension, n	16	47	0.257
Current smokers, n	3	10	0.541
Family CAD, n	3	17	0.102
Dislipidemia, n	7	13	0.632
BMI (kg/m ²)	30.42 ± 4.97	31.29 ± 5.80	0.423
LVEF, %	64.18 ± 3.14	64.14 ± 2.38	0.858
MPI	0.52 ± 0.13	0.63 ± 0.17	<0.001
Subclinical LV dysfunction	23	70	0.029

BMI, Body mass index; LVEF, left ventricle ejection fraction; MPI, myocard performance index

Cardiac imaging / Echocardiography

OP-031

Evaluation of atrial and ventricular functions with echocardiography in embolic stroke of undetermined source

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Background and Aim: Left atrial (LA) cardiopathy may lead to stroke. Myocardial strain analysis may offer insights into LA and left ventricular (LV) pathophysiology. The aim of this study was to analyze LA function and LV strain in patients with embolic stroke of undetermined source (ESUS).

Methods: This prospective study included 35 patients with ESUS (61±10 years old) and 37 age and sex-matched controls (60±10 years old). All the participants underwent brain computed tomography (CT), conventional and diffusion-weighted magnetic resonance imaging (fMRI), CT or MR angiography, 12 lead electrocardiography (ECG), transthoracic echocardiographic examination and Holter ECG monitoring. Individuals with major-risk cardioembolic sources were excluded. Left atrial volume and function were determined

Cardiac imaging / Echocardiography

OP-029

Acute effects of ultrafiltration on arterial stiffness index β and elastic modulus in chronic hemodialysis patients

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Background and Aim: Hemodialysis sessions make abnormalities in mechanical properties of the arterial vessel by rapid volume changes, inflammation and calcification. Cardiovascular diseases are common in chronic renal failure patients and accelerated with hemodialysis. Arterial stiffness strongly predicts cardiovascular complications also vascular disorders. With the extension of the vascular disorder the distensibility and strain of the aorta lessens. Previous studies were demonstrated the relationship between severity of atherosclerosis and parameters such as aortic strain, aortic distensibility, elastic modulus and aortic stiffness index. We aim to evaluate the alterations of these parameters after a single ultrafiltration session.

Methods: Totally 30 patients who have been already undergoing conventional hemodialysis for 3 days a week and 4 hours long for the last 6 months. Prior to hemodialysis all patients underwent echocardiography and left ventricular wall motions, chambers, valves and pulmonary arterial pressures were evaluated. Moderate and more severe valve disorders were excluded. Each patient's mechanical vascular properties at ascending aorta were studied non-invasively by echocardiography device. The systolic and diastolic diameters to the ascending aorta were measured with M-mode echocardiography 3 cm above the aortic valve. Aortic strain, elastic modulus, aortic stiffness index and arterial distensibility parameters were calculated with specific formulas.

Results: 30 patients (17 male, 13 female) with a mean age 61.4±17.1 were attended this study. 10 of them had already coronary artery disease, 12 of them had hypertension, 9 of them had diabetes mellitus. The mean left ventricular ejection fraction 56±8.6%, 6 patients had systolic dysfunction. An average of 2.5±0.89 liters fluid was withdrawn during hemodialysis sessions. Mean systolic and diastolic blood pressures before and after the sessions were 148.6±19.3, 76.1±12.2 and 120.8±26.6, 73.3±13.2, respectively. Aortic both systolic and diastolic diameters were significantly decrease after hemodialysis session. There was a statistically significant change in aortic strain, elastic modulus, arterial stiffness index and arterial distensibility, demonstrated at Table 1.

Conclusions: Hemodialysis treatment may worsen aortic mechanical properties and the changes can be noticed in acute terms after sessions. Sympathetic system activation induced by hemodialysis cause a tonic restraint accompanied by change in aortic strain, arterial stiffness and elastic modulus. Renin system activation after dialysis may precipitate acute deterioration of arterial distensibility. Arterial stiffness is influenced by hydration status, temperature and serum ionized calcium levels. Although patients undergo hemodialysis show worse mechanical properties, ultrafiltration during hemodialysis significantly improves the aortic strain and arterial distensibility which is inversely related to the elastic modulus and arterial stiffness index.

Table 1. Hemodynamic values before and after hemodialysis, statistical significance of p value was <0.05

	Pre-Hemodialysis	Post-Hemodialysis	p value
Aort systolic diameter (mm)	28.5 ± 3.57	26 ± 2.68	< 0.001
Aort diastolic diameter (mm)	32 ± 3.53	29.5 ± 2.25	< 0.001
Aortic strain (%)	0.11 ± 0.06	0.17 ± 0.09	0.009
Elastic modulus (P)	560 ± 72.3	333 ± 52.4	< 0.001
Arterial stiffness index (β)	2.7 ± 0.4	1.17 ± 0.32	0.018
Arterial distensibility (cm2 dyn ⁻¹ 110 ⁷ 3)	3.90 ± 1.60	4.05 ± 1.48	0.022

by echocardiography. Left atrial and ventricular strains were measured longitudinally by speckle-tracking method. CHA2DS2-VASc, The National Institutes of Health Stroke Scale (NIHSS), Modified Rankin Scale (mRS) scores were calculated for each patient. The results were analyzed by Independent samples t-test and Mann-Whitney U test.

Results: Major cardiovascular risk factors were similar between the 2 groups. The mean CHA2DS2-VASc score was 2.6±1.2, NIHSS was 3.9±3.0 and mRS was 1.3±0.8. Atrial electromechanical coupling (PA) and delay (EMD), LA emptying fraction (EF) and volumes were similar between the 2 groups. There was a trend that atrial PA, EMD were longer and LA EF was lower than controls (56.7±9.8% vs. 61.2±8.6%, p=0.137). LA global longitudinal strain was lower than controls (25.2±7.2 vs. 29.7±8.8, p=0.019). LV global longitudinal strain was lower than controls (-14.7±4.2% vs -16.4±3.9%, p=0.031). There was no correlation between LA, LV strains and the scores (CHA2DS2-VASc, NIHSS, mRS).

Conclusions: In this study, the patients had lower LA and LV longitudinal global strains than controls. In contrast, LA volume index did not differ between the 2 groups. LA and LV strains could be useful in the determination of which patients should be closely followed up in order that more aggressive cardiovascular risk factor modification and surveillance for asymptomatic atrial fibrillation (AF) may be applied.

Table 1. Clinical and laboratory characteristics of subjects

	Patients (n=35)		p
	Mean ± Std. Dev.	Controls (n=37) Mean ± Std. Dev.	
Age, years	61 ± 10	60 ± 10	0,964
LA global longitudinal strain, %	25,2 ± 7,2	29,7 ± 8,8	0,019*
LV global longitudinal strain, %	-14,7 ± 4,2	-16,4 ± 3,9	0,031*
LA emptying fraction, %	56,7 ± 9,8	61,2 ± 8,6	0,137
Maximum LA volume index, ml/m ²	24,2 ± 6,5	25,2 ± 5,8	0,489
Minimum LA volume index, ml/m ²	10,7 ± 4,4	9,8 ± 3,7	0,414
PA lateral, ms	43,9 ± 14,3	38,6 ± 14,8	0,178
PA septal, ms	33,0 ± 13,0	28,1 ± 11,9	0,096
PA tricuspid, ms	24,0 ± 11,4	21,1 ± 8,4	0,222
Right intra-atrial EMD, ms	8,8 ± 7,3	7,0 ± 5,6	0,344
Inter-atrial EMD, ms	19,9 ± 9,1	17,5 ± 10,5	0,351

Cardiac imaging / Echocardiography

OP-032

The usefulness of resting four dimensional speckle tracking for identifying coronary artery stenosis

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Background and Aim: Patients with suspected coronary artery disease (CAD) are commonly evaluated by non-invasive tests in order to identify those that must be referred to coronary angiography. Four-dimensional speckle tracking echocardiography (4DSTE) is a novel and sensitive technique for quantitative evaluation of myocardial deformation. The aim of this study was to investigate the usefulness of resting 4D-STE for identifying critical coronary artery stenosis.

Methods: This is a prospective single center study. One hundred fifty suspected coronary artery disease (CAD) patients with biventricular regional wall motion abnormality (RWMA) observed visually from traditional echocardiography were divided into two groups according to coronary artery angiography (CAG). The parameters from 4D-STE were compared between different groups and then the diagnostic value of global longitudinal strains indicating different graded coronary artery stenosis was analyzed by the receiver operating characteristic curve (ROC).

Results: Fifty-three patients enrolled in critical stenosis group (group 1) (stenosis rate (SR) >75%), and 97 patients in non-critical stenosis group (group 2) (SR<75%). There were not any significant difference on age, gender, cardiac risk factors, two dimensional ejection fraction (EF) in, and three dimensional EF between two groups. There were significant difference in left ventricle (LV) 4D global longitudinal strains (GLS) between two groups (LGS group1: 4D LGS -14.6±3.1%; group 2: -19.9±4.5%, p<0.001). ROC analyses showed that the area under the curve as LV 4D GLS was 0.841 and less than -14.13% allowed a sensitivity of 91% and a specificity of 81.4%, for evaluating critical coronary artery stenosis without the need for any stress factors.

Conclusions: This study showed that resting 4D-STE had the potential to improve the value of echocardiography in the detection of the CAD in patients without RWMA and it maybe use as a diagnostic tool before CAG.

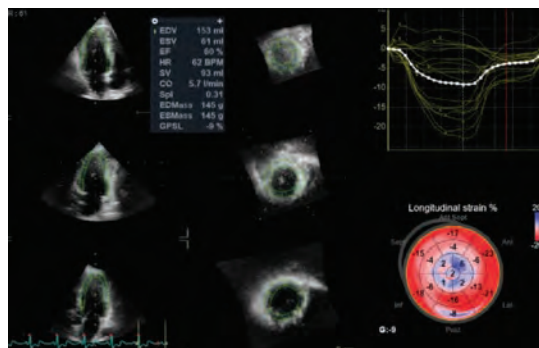


Figure 1. An image of 4DSTE in the patient with severe coronary artery stenosis rate classified by CAG. Despite calculated 3D ef was found normal (60%), GLS was -9% that indicated the corresponded myocardial segment deformation decreased.

Cardiac imaging / Echocardiography

OP-033

Early Detection of strain/strain rate and time to strain/strain rate abnormalities for left atrial mechanical function in hypertensive patients

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Background and Aim: To evaluate left atrial (LA) deformation parameters strain (S)/strain rate (SR) and time to peak S/SR obtained by two-dimensional speckle tracking echocardiography (2D-STE) in patients with hypertension for three LA mechanical phases and to compare them with the same indices in control subjects.

Methods: Eighty four subjects (mean age 57.358±12.45 years, 46% male) over the age of 18 were included in the study. Subjects were divided into two groups: hypertensive group consisted of 55 patients (mean age 58.86±11.61 years, 32 females) with hypertension, and control group comprised 29 healthy subjects (mean age 54.84±13.54 years, 16 females). All patients had normal LA poster-anterior diameter, LA and left ventricular ejection fractions (LVEF >50%) in 2-Dimensional echocardiography (2-DE). The peak S/SR values (PS/PSR), the time to peak S/SR (TPS/TPSR) were measured using the 12-segment model for the left atrium during contractile (CP) reservoir (RP) and conduit period (COP) of the LA cycle (Figure 1, 2).

Results: Comparison of LA longitudinal S an SR values between two groups is shown in Table 1. For two periods (RP and COP), all of the PS and PSR values were significantly lower in hypertensive patients with preserved LAEF and LVEF compared to those in the controls, except for the CP. Similarly, hypertensive patients had significantly higher TPS and TPSR than those in the controls for the RP and COP in the LA wall, except for the CP (Table 2).

Conclusions: The study showed that LA myocardial longitudinal function was impaired in hypertensive patients during reservoir, conduit and contractile phases. Decreased PS and PSR values in all of the three LA periods would be a signal of atrial mechanical function due to hypertension in these patients. In addition, we suggested that a prolonged deformation period might be an additional factor to the deterioration of segmental LA myocardial functions in HT patients. The PS/PSR and TPS/TPSR of LA could be quantified in HT patients with an objective manner by 2-DSTE and their analysis was a sensitive tool to detect LA myocardial dysfunction in patients with hypertension.

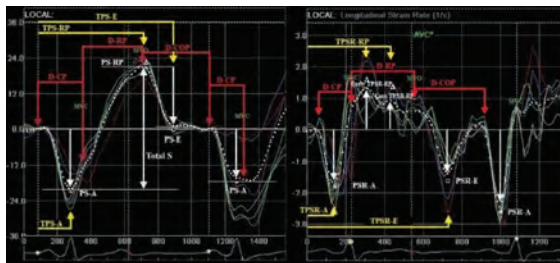


Figure 1. Pattern and measurements of the LA strain, strain rate, time to peak strain and time to peak strain rate. D-COP, the duration conduit period; D-CP, the duration contractile period; D-RP, the duration reservoir period; LA, left atrium; MVC, mitral valve closure; MVO, mitral valve opening; PS-A, the peak strain-A view; PS-E, the peak strain-E view; PS-RP, the peak left atrial strain; TPR-A, the time to peak strain-A view; TPR-RP, the time to peak to strain reservoir period; TPR-E, the time to peak strain-E view; TSR-A, the peak to atrial strain rate-A view; TSR-E, the peak to strain rate-E view; TSR-RP, the peak to strain rate.

Table 1. Global LA longitudinal peak strain values in hypertensive group compared with control group with normal tension for the CP. Difference between PS-A and PS-E, Difference between PS-RP and PS-A, RP and COP of left atria

		A4CV		A2CV	
		Ct	HT	Ct	HT
CP	PS-A	Ct	-10.74 ± 6.40	-10.98 ± 7.16	
		HT	-11.63 ± 6.20	-11.02 ± 7.90	
		p	0.522	0.980	
Difference between PS-A and PS-E		Ct	11.01 ± 7.04	11.43 ± 8.25	
		HT	14.32 ± 7.04	13.01 ± 8.54	
		p	0.036	0.404	
Difference between PS-RP and PS-A		Ct	12.6 ± 9.33	9.28 ± 10.53	
		HT	2.55 ± 9.48	1.19 ± 10.25	
		p	<0.001	<0.001	
Total S	PS-RP	Ct	34 ± 9.32	31.39 ± 8.88	
		HT	25.63 ± 9.68	23.16 ± 10.65	
		p	<0.01	0.001	
RP	PS-RP	Ct	20.18 ± 7.66	15.94 ± 6.84	
		HT	11.25 ± 5.81	9.86 ± 7.22	
		p	<0.001	<0.001	
COP	PS-E	Ct	0.28 ± 2.95	0.44 ± 3.08	
		HT	2.69 ± 2.79	2.09 ± 2.88	
		p	<0.001	0.014	
Difference of PS-RP to PS-E		Ct	24.01 ± 8.09	19.5 ± 6.91	
		HT	10.72 ± 4.75	10.34 ± 6	
		p	<0.001	<0.001	
LV PS	PS-A	Ct	-21.3 ± 2.72	-21.2 ± 4.17	
		HT	-18.38 ± 8.88	-19.76 ± 3.88	
		p	0.088	0.125	

COP, conduit period; CP, contractile period; LA, left atria; LV, left ventricle peak strain; p, p value; PS-A, peak strain A wave; PS-E, peak strain E wave; PS-RP, peak strain reservoir period; RP, reservoir period; S, Strain.

Table 2. Global LA longitudinal peak strain rate values in hypertensive group compared with control group with normal tension for the CP, RP and COP of left atria

CP	A2CV	Ct	A4CV	A2CV	
			HT	-1.67 ± 0.9	-1.72 ± 0.91
			HT	-1.62 ± 0.8	-1.62 ± 0.78
			p	0.808	0.575
RP	Early PSR-RP	Ct	HT	1.57 ± 0.53	1.51 ± 0.46
			HT	1.22 ± 0.45	1.13 ± 0.47
			p	0.020	0.010
Late PSR-RP	Ct	HT	1.26 ± 0.45	0.97 ± 0.41	
		HT	0.86 ± 0.37	0.77 ± 0.44	
		p	<0.001	0.036	
COP	PSR-E	Ct	HT	-1.84 ± 0.88	-1.70 ± 0.88
			HT	-0.95 ± 0.54	-0.88 ± 0.59
			p	<0.001	<0.001

PSR-A, peak strain rate A wave; PSR-E, peak strain rate E wave; PSR-RP, peak strain rate reservoir period (early and late); p, p value.

Table 3. Global LA longitudinal time to peak strain values and durations for the CP, RP and COP of left atrial in hypertensive group compared with control group

CP	TPS-A	Ct	A4CV	A2CV	
			HT	100.33 ± 19.83	104.93 ± 21.06
			HT	135.18 ± 22.62	138.03 ± 24.78
			p	<0.001	<0.001
D-CP	Ct	HT	158.75 ± 23.18	159.12 ± 25.19	
		HT	210.96 ± 38.39	211.41 ± 36.73	
		p	<0.001	<0.001	
COP	TPS-RP	Ct	HT	498.87 ± 47.99	497.51 ± 58.65
			HT	563.29 ± 67.58	571.94 ± 67.50
			p	<0.001	<0.001
D-RP	Ct	HT	340.12 ± 36.28	329.81 ± 40.16	
		HT	351.79 ± 67.20	365.46 ± 73.16	
		p	0.361	0.012	
COP	TPS-E	Ct	HT	658.09 ± 56.47	651.75 ± 74.31
			HT	719.38 ± 88.91	721.78 ± 93.70
			p	0.001	<0.001
D-COP	Ct	HT	273.93 ± 90.15	257.84 ± 105.59	
		HT	251.12 ± 96.17	249.53 ± 98.89	
		p	0.273	0.712	

D-COP, duration of conduit period; D-CP, duration of contractile period; D-RP, duration of reservoir period; TPS-A, time to peak strain A wave; TPS-E, time to peak strain E wave; TPS-RP, time to peak strain reservoir period; p, p value.

Table 4. Global LA longitudinal strain rate values and durations for the CP, RP and COP of the left atrial in hypertensive group compared with control group with normal tension

CP	TPSR-A	Ct	Values		
			HT	94.06 ± 34.85	79.18 ± 23.32
		HT	100.50 ± 37.64	104.90 ± 23.91	
		p	0.426	<0.001	
RP	D-CP	Ct	HT	175.33 ± 30.97	175.27 ± 51.47
			HT	197.14 ± 43.47	211.98 ± 39.86
			p	0.014	<0.001
Early TPSR-RP	Ct	HT	263.21 ± 46.16	258.21 ± 48.37	
		HT	310.18 ± 57.93	312.05 ± 50.41	
		p	<0.001	<0.001	
Late TPSR-RP	Ct	HT	369.48 ± 42.78	377.48 ± 63.99	
		HT	417.01 ± 72.38	429.51 ± 66.28	
		p	0.001	<0.001	
D-RP	Ct	HT	329.81 ± 40.16	330.45 ± 58.59	
		HT	365.46 ± 73.16	358.74 ± 63.60	
		p	0.012	0.041	
COP	TPSR-E	Ct	HT	606.93 ± 58.92	604.96 ± 65.90
			HT	670.38 ± 79.49	674.32 ± 67.97
			p	<0.001	<0.001
D-COP	Ct	HT	259.03 ± 92.94	250.18 ± 102.20	
		HT	253.57 ± 77.72	247.87 ± 81.99	
		p	0.769	0.908	

TPSR-A, time to peak strain rate A wave; TPSR-E, time to strain rate E wave; TPSR-RP, time to peak strain rate reservoir period (early and late); p, p value.

Interventional cardiology / Coronary

OP-034

The accuracy of contrast-FFR for evaluation of intermediate coronary artery stenosis

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Background and Aim: Fractional flow reserve is gold standard method to evaluate coronary lesions that functionally significant to be revascularization. Adenosine is used in FFR measurements to induce hyperemia and minimum microvascular resistance but side effects of adenosine limits its usage. Contrast mediated submaksimal hyperemia can effect the Pd/Pa ratio. The aim of this study was to compare contrast mediated Pd/Pa ratio (cFFR) to FFR.

Methods: 91 patients with intermediate coronary lesions scheduled for FFR measurement. Heparin (5000 IU) was administered at the beginning of the procedure followed by an intracoronary bolus of nitroglycerine (0.1 mg). Aortic pressure (Pa) was measured via a 6 Fr guiding catheter. Intracoronary distal pressure (Pd) was measured simultaneously using a 0.014 pressure-sensor guidewire. Contrast-FFR were taken throughout reactive hyperaemia induced by manually a standard 10 ml intracoronary bolus of the contrast agent, Iohexol (Omnipaque 350 mg I/ml). The measurements were repeated after administration of i.v. adenosine 140 µg/kg/min in three minutes to obtain FFR. Correlation assessed between two measurements.

Results: Baseline characteristics of patients were given in table 1. There was strong correlation between cFFR and FFR (r=0.919, p<0.001). ROC curve analysis showed cFFR <0.815 in predicting FFR <0.80 (AUC: 0.965 [95% CI]; sensitivity 0.93 specificity 0.84) (Figure 1).

Conclusions: We found that the cut-off value of cFFR is <0.815 for assessment of coronary stenosis severity. Contrast-FFR can be used to evaluate the intermediate coronary stenosis severity. Especially if contrast FFR is positive there is no need to perform standard FFR.

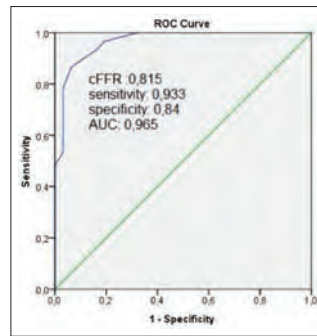


Figure 1. ROC curve of contrast FFR.

Table 1. Baseline characteristics of patients (n=91)

Age	59 ± 9,9
Male	69 (%75)
DM	28 (%30,8)
HT	37 (%40,8)
Smoking	32 (%35,1)
Dyslipidemia	38 (%41,7)
PCI	30 (%33)
Previous MI	24 (%26,3)
Medications	
ASA	89 (%97,8)
P2Y12 inh	51 (%56)
Beta-Blockers	77 (%84,6)
RAS inh	68 (%74)
CaCB	16 (%17,5)
Statins	67 (%73,66)
Coronary lesions	
LAD	74 (%81,3)
Cx	14 (%15,4)
RCA	3 (%3,3)

Interventional cardiology / Coronary

OP-035

In vitro analysis of the effect of contrast agents on the antiagregan effect of P2Y12 inhibitors

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Background and Aim: There is insufficient data on whether the antiagregan effect of P2Y12 inhibitors used during coronary interventions are affected by the contrast agents. The aim of this study was to evaluate the in vitro effect of different contrast agents (iohexol, iodoxanol) on antiagregant activity of P2Y12 inhibitors (clopidogrel, ticagrelor, prasugrel).

Methods: Thirty patients (performed percutaneous coronary intervention and treated with P2Y12 inhibitor for minimum 10 days) and 5 healthy volunteers were included in the study. The study patients were divided into 4 groups: patients in the first group using clopidogrel (10 patients), patients in the second group using ticagrelor (10 patients), patients in the third group using prasugrel (10 patients) and patients in the fourth group was control (5 patients). Iohexol (low osmolar nonionic) and iodoxanol (iso osmolar nonionic) was used in this trial. Clopidogrel, ticagrelor and prasugrel anti-agregan properties were evaluated using the Verify-Now method. The antiagregant activity was measured by calculating the PRU (P2Y12 Reaction Units) values. 3 tubes of blood (3.2% sodium citrate tubes) were withdrawn from the patients and control group 2 hours after the last dose of the drug (clopidogrel, prasugrel, ticagrelor). There were 2ml of blood in the first tube and 9ml of blood in the other 2 tubes. All of the blood tubes was kept on the dialing device for 30 min. After 30 min, 1ml iohexol was add the second tube and 1ml iodoxanol was add the third tube. PRU values were checked from the control tube (first tube) and the contrast material tubes 5 min after the contrast material was added. PRU values were calculated again 30 min after the 2nd and 3rd tubes (include contrast agent).

Results: The patients age were older than the control group (Age (year); Clopidogrel:54.3±11, Ticagrelor:52.8±9.9, Prasugrel:50.8±9.3, Control:39.4±9.4), and the EF was lower than the control group (Clopi-

grel:40.5±8.9, Ticagrelor:41.5±8.5, Prasugrel:46±9.6, Control:62.0±3.1) (p<0.05). PRU values were significantly lower in clopidogrel, ticagrelor and prasugrel groups than control group (p<0.05) (Figure 1). PRU values of ticagrelor and prasugrel groups were significantly lower than clopidogrel group (p<0.001) and ticagrelor and prasugrel PRU values were similar (p>0.05) (Figure 1). Ilohexol and idoxanol were lead to a significant decrease in PRU values in the control group and the clopidogrel group, but not in the ticagrelor and prasugrel groups (Figure 1). The antiaggregant activity of Ilohexol and idoxanol were found similar at 5th and 30th minutes (Figure 2).

Conclusions: Ilohexol and Idoxanol were have in vitro antiaggregant effects and both of them antiaggregant effects were similar. Ilohexol and Idoxanol were increase the clopidogrel antiaggregant activity in vitro but did not significantly alter the prasugrel and ticagrelor antiaggregant activity. The antiaggregant activity of ticagrelor and prasugrel was significantly higher than that of clopidogrel.

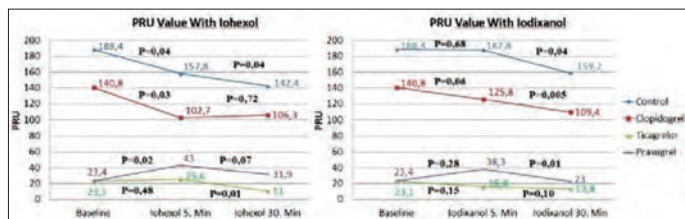


Figure 1. PRU (P2Y12 Reaction Units).

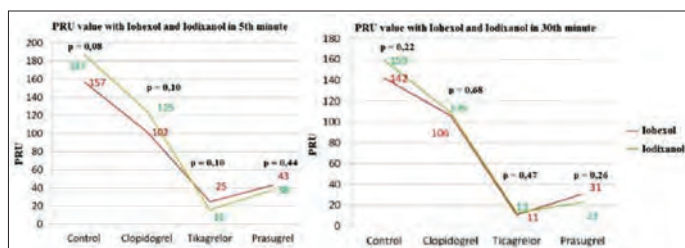


Figure 2. PRU (P2Y12 Reaction Units).

Table 1. Baseline characteristics and laboratory parameters of the study population

	Min-Max	Median	Mean,±s.d./n-%
Age,years	34.0 -96.0	62.4	60.5 ± 10.8
Creatinine,mg/dl	0.6 -1.5	1.0	1.0 ± 0.2
Radial diameter,mm	1.4 -4.4	2.3	2.35 ± 0.5
Sex			
Male			56 70%
Female			24 30%
Arm			
Right			60 75%
Left			20 25%
HT			
No			33 41%
Yes			47 59%
DM			
No			56 70%
Yes			24 30%
HL			
No			48 60%
Yes			32 40%
Smoking			
No			45 56%
Yes			35 24%
BB			
No			27 34%
Yes			53 66%
CCB			
No			64 80%
Yes			16 20%
ASA			
No			19 24%
Yes			61 76%
P2Y12			
No			69 86%
Yes			11 14%
CAD			
No			44 55%
Yes			36 45%
Puncture success			
No			12 15%
Yes			68 85%

ASA, acetyl salicylic acid; BB, beta blocker ; CAD, coronary artery disease;CCB, calcium channel blocker; DM, diabetes mellitus; HT, hypertension; HL, hyperlipidemia; P2Y12, P2Y12 inhibitors

Table 2. Baseline characteristics and laboratory parameters of the study patients according to the intermittent contralateral arm ischemia

	Arm ischemia (-)		Arm ischemia (+)		p
Age, years	61.2 (53-65)		64.5 (58-67)		0.089
Creatinine,mg/dl	0.9 (0.84-1.12)		1.02 (0.90-1.24)		0.069
Radial diameter,mm	2.21 (2.0-2.70)		2.40 (2.09-2.68)		0.301
Sex, n(%)	Male	28 70%	28 70%		1.000
	Female	12 30%	12 30%		
Smoking,n(%)	No	23 57%	22 55%		1.000
	Yes	17 43%	18 45%		
HT	No	20 50%	13 32%		0.173
	Yes	20 50%	27 68%		
HL	No	21 52%	27 67%		0.254
	Yes	19 48%	13 33%		
DM	No	32 80%	24 60%		0.087
	Yes	8 20%	16 40%		
CAD	No	24 60%	20 50%		0.500
	Yes	16 40%	20 50%		
BB	No	14 35%	13 32%		1.000
	Yes	26 65%	27 68%		
CCB	No	33 82%	31 77%		0.781
	Yes	7 18%	9 23%		
ASA	No	13 32%	6 15%		0.114
	Yes	27 68%	34 85%		
P2Y12	No	35 87%	34 85%		1.000
	Yes	5 13%	6 15%		
Arm	Right	33 83%	27 68%		0.196
	Sol	7 17%	13 32%		
Puncture success	Yes	30 75%	38 95%		0.025
	No	10 25%	2 5%		

ASA, acetyl salicylic acid; BB, beta blocker ; CAD, coronary artery disease;CCB, calcium channel blocker; DM, diabetes mellitus; HT, hypertension; HL, hyperlipidemia; P2Y12, P2Y12 inhibitors

Table 3. Multivariable logistic regression analysis of associations between radial puncture success and variables in the study patients

Variables	Odds ratio	95% CI	P value
Age	0.970	0.901-1.044	0.411
Creatinine	7.467	0.206-28.346	0.270
Diabetes mellitus	0.535	0.120-2.378	0.411
Intermittent arm ischemia	8.261	1.427-47.823	0.018

Interventional cardiology / Coronary

OP-036

Periprocedural intermittent contralateral arm ischemia facilitates radial artery puncture

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Background and Aim: Transradial access (TRA) is widely used for both diagnostic and interventional cardiac procedures. Intermittent arm ischemia before percutaneous coronary intervention induces remote ischemic preconditioning and attenuates myocardial injury in patients with myocardial infarction. Several studies have shown that intermittent arm ischemia increases coronary flow and is related to autonomic nerve system. In this study we sought to investigate whether periprocedural intermittent arm ischemia facilitates radial artery puncture.

Methods: Patients (n=80) undergoing planned coronary interventions using TRA were prospectively randomized to intermittent contralateral arm ischemia (n=40) group and control (40) group. Radial artery diameters were measured by ultrasonography. The study endpoint aimed to determine whether the intermittent arm ischemia would improve radial artery access, number of punctures and successful cannulation.

Results: Baseline demographic and clinical characteristics were similar in two groups. There were also no significant differences regarding radial artery diameters before intervention between groups (2.21 mm [2.0-2.70] for intermittent contralateral arm ischemia group and 2.40 mm [2.09-2.68] for controls, p=0.301). However, the radial artery puncture success ratio was higher in the intermittent contralateral arm ischemia group as compared to the control group (p=0.025). In multivariable logistic regression analysis intermittent contralateral arm ischemia was found to be significantly associated with radial artery puncture success (HR: 8.261, 95% CI: 1.427-47.823, p=0.018).

Conclusions: Periprocedural intermittent contralateral arm ischemia facilitates radial artery puncture in patients undergoing transradial cardiac catheterization.

Interventional cardiology / Coronary

OP-037

The role of thiol levels in predicting contrast-induced nephropathy in patients with ST-segment elevation myocardial infarction who underwent primary percutaneous coronary intervention

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Background and Aim: Contrast-induced nephropathy (CIN) is a common complication of that may arise from intravascular contrast media administration. Recent studies have reported the thiol-disulfide ratio as a novel oxidative stress marker. We therefore investigated the role of thiol levels in predicting CIN in patients with ST-segment elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention (PCI).

Methods: A total of 302 patients were enrolled in the study. CIN was defined as an increase in serum creatinine concentration ≥ 0.5 mg/dL compared with admission value, or a $>25\%$ relative rise during the first 48-72 hours after the procedure. Patients were divided into CIN (+) and CIN (-) groups and thiol levels were evaluated.

Results: CIN occurred in 44 (15%) patients. Native thiol (274.8 ± 84.7 $\mu\text{mol/L}$ vs 220.8 ± 97.1 $\mu\text{mol/L}$, $p=0.001$) and total thiol (305.4 ± 89.7 $\mu\text{mol/L}$ vs 260.1 ± 102.1 $\mu\text{mol/L}$, $p=0.009$) levels were higher in patients within the non-CIN group. Disulfide (15.8 ± 6.6 $\mu\text{mol/L}$ vs. 19.6 ± 8.4 $\mu\text{mol/L}$, $p=0.002$) levels, and mean disulfide / total thiol ratios (8.4 ± 3.7 vs. 5.9 ± 3.1 , $p=0.001$) were higher in patients with CIN(+) group. In univariate analysis; initial native thiol, total thiol, disulfide levels and disulfide/total thiol ratio were found to have prognostic significance in the development of CIN. In the multivariate regression analysis, only disulfide/total thiol ratio (OR=1.190; 95% CI, 1.090-1.300; $p=0.001$) was significantly and independently associated with CIN. Finally, ROC analysis was performed to determine the cut-off value of disulfide/total thiol ratio to predict CIN. The cut-off value of disulfide/total thiol ratio on admission to predict CIN in patients with STEMI who underwent primary PCI was 7, with a sensitivity of 68.2% and a specificity of 79.8% (AUC=0.740 (0.655-0.825), $p<0.001$).

Conclusions: Our results suggest that thiol/disulfide homeostasis could be a good biochemical risk marker for CIN in STEMI patients who underwent primary PCI. Considering its clinical significance, the thiol/disulfide homeostasis may help identify high-risk candidates of CIN in AMI.

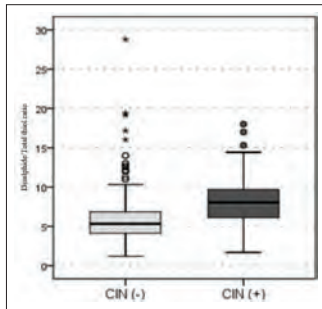


Figure 1. Relationship between disulfide/total thiol ratio levels with contrast induced nephropathy in patients with acute myocardial infarction.

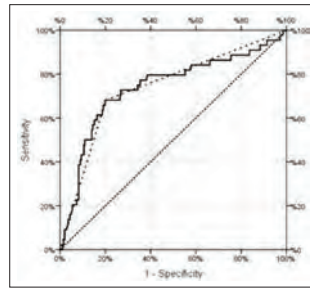


Figure 2. Receiver operator characteristic (ROC) curve analysis. ROC curve analysis showed that at a cutoff of 7, the value of disulfide/total thiol ratio exhibited 68.2% sensitivity and 79.8% specificity for predicting contrast induced nephropathy.

Table 1. Baseline clinical, demographic and laboratory characteristics of the study population

	Min-Max	Median	Mean \pm sd, n (%)
Age	33.0-81.0	59.0	66.2 \pm 12.7
Sex	Male Female	172 130	57% 43%
DM	No Yes	196 106	65% 35%
HT	No Yes	171 131	57% 43%
CAD	No Yes	213 87	71% 29%
CABG	No Yes	270 32	89% 11%
TIA/Stroke	No Yes	250 22	83% 7%
AMI localization	Anterior Non-anterior	174 126	58% 42%
Access site	Femoral Radial	184 118	61% 39%
Smoking	No Yes	122 180	40% 60%
Glucose (mg/dL)	68-333	124.5	130.3 \pm 84.3
Serum creatinine (mg/dL)	0.63-1.8	1.03	1.122 \pm 0.677
Hemoglobin (g/L)	9.6-16.5	13.9	13.7 \pm 1.6
WBC count (x1000/mm ³)	5.8-23.7	12.3	11.5 \pm 4.1
Platelet count (x1000/mm ³)	110-826	251	240.5 \pm 87.3
Total cholesterol (mg/dL)	77-587	191	188.5 \pm 43.6
LDL (mg/dL)	51-345	126	117.4 \pm 38.6
HDL (mg/dL)	16-70	37.8	39.7 \pm 10.1
Triglycerides (mg/dL)	35-617	116	147 \pm 92
LVEF (%)	20-84	51	48.1 \pm 11
Contrast medium volume (mL)	90-400	220	210.1 \pm 50.2

AMI, acute myocardial infarction; CABG, coronary artery bypass graft; CAD, coronary artery disease; DM, diabetes mellitus; HDL, high-density lipoprotein; HT, hypertension; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; TIA, transient ischemic attack; WBC, white blood cell.

Table 2. Baseline clinical, demographic and laboratory characteristics of the patients with and without contrast-induced nephropathy

	CIN (-)		CIN (+)		p	
	Meantstd, n/n-%	Median	Meantstd, n/n-%	Median		
Age	60.1 \pm 12.4	59.0	60.9 \pm 14.1	58.5	0.724 ^{ns}	
Sex	Male Female	155 103	60% 40%	27 17	61% 39%	0.810 ^{ns}
DM	No Yes	170 88	66% 34%	26 18	59% 41%	0.382 ^{ns}
HT	No Yes	144 114	56% 44%	27 17	61% 39%	0.492 ^{ns}
Smoking	No Yes	104 154	40% 60%	18 26	41% 59%	0.675 ^{ns}
CAD	No Yes	185 73	72% 28%	30 14	68% 32%	0.633 ^{ns}
CABG	No Yes	232 26	90% 10%	38 6	86% 14%	0.478 ^{ns}
TIA/Stroke	No Yes	237 21	92% 8%	43 1	98% 2%	0.166 ^{ns}
AMI localization	Anterior Non-anterior	148 110	57% 43%	28 16	64% 36%	0.435 ^{ns}
Access site	Femoral Radial	138 100	61% 39%	26 18	59% 41%	0.311 ^{ns}
Glucose (mg/dL)	146 \pm 76	124	160 \pm 86	125	0.424 ^{ns}	
Serum creatinine (mg/dL)	1.06 \pm 0.32	1.01	1.13 \pm 0.28	1.16	0.036 ^{ns}	
Hemoglobin (g/L)	13.8 \pm 1.8	13.8	13.7 \pm 1.6	13.5	0.594 ^{ns}	
WBC count (x1000/mm ³)	11.4 \pm 3.8	11.1	12.0 \pm 4.7	11.3	0.422 ^{ns}	
Platelet count (x1000/mm ³)	239 \pm 78	231	247 \pm 74	243	0.367 ^{ns}	
Total cholesterol (mg/dL)	187 \pm 45	190	199 \pm 49	199	0.095 ^{ns}	
LDL (mg/dL)	116 \pm 38	111	126 \pm 37	122	0.082 ^{ns}	
HDL (mg/dL)	39 \pm 11	37	40 \pm 10	39	0.383 ^{ns}	
Triglycerides (mg/dL)	142 \pm 89	132	154 \pm 95	148	0.082 ^{ns}	
LVEF (%)	48 \pm 10	50	46 \pm 12	47	0.455 ^{ns}	
Contrast medium volume (mL)	210 \pm 50	212	217 \pm 60	223	0.105 ^{ns}	

AMI, acute myocardial infarction; CABG, coronary artery bypass graft; CAD, coronary artery disease; DM, diabetes mellitus; HDL, high-density lipoprotein; HT, hypertension; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; TIA, transient ischemic attack; WBC, white blood cell.
^{ns}Mann-Whitney U Test. ^{ns}Chi Square (2) Test

Table 3. The level of native thiol, total thiol, disulfide, and disulfide/total thiol ratio between the patients with and without contrast-induced nephropathy (CIN)

	CIN (-)		CIN (+)		p
	Meantstd	Median	Meantstd	Median	
Total thiol, ($\mu\text{mol/L}$)	305.4 \pm 89.7	302.8	260.1 \pm 102.1	263.3	0.009 ^{ns}
Native thiol, ($\mu\text{mol/L}$)	274.8 \pm 84.7	282.5	220.8 \pm 97.1	229.1	0.001 ^{ns}
Disulfide, ($\mu\text{mol/L}$)	15.8 \pm 6.6	15.2	19.6 \pm 8.4	18.4	0.002 ^{ns}
Disulfide/Total thiol $\times 100$	5.9 \pm 3.1	5.3	8.4 \pm 3.7	8	0.001 ^{ns}

Table 4. Univariate and multivariate logistic regression analysis of contrast-induced nephropathy (CIN)

	Univariate			Multivariate		
	OR	95% CI	p	OR	95% CI	p
Native thiol	0.993	0.989-0.997	<0.001			
Total thiol	0.995	0.991-0.998	0.003			
Disulfide	1.077	1.030-1.126	0.001			
Disulfide/Total thiol ratio	1.199	1.098-1.310	<0.001	1.190	1.090-1.300	0.001

Interventional cardiology / Coronary

OP-038

The combined use of fragmented QRS and QRS distortion has higher prognostic value than using one of them alone

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Background and Aim: 12 lead admission ECG has a crucial role in the diagnosis and early risk stratification in patients with acute ST segment elevation myocardial infarction (STEMI). QRS distortion (QRSd) reflects

the severity of myocardial ischemia and has been found to be associated with poor prognosis in acute STEMI. Fragmented QRS (fQRS) has also been reported to be associated with poor clinical outcomes in acute STEMI patients. Although both ECG parameters were separately demonstrated to be associated with increased poor prognostic events, there is no study investigating the prognostic importance of the combined use of these two parameters. The aim of our study was to evaluate the prognostic significance of the combined use of fQRS and QRSd in patients with acute STEMI undergoing primary percutaneous coronary intervention (pPCI).

Methods: A total of 454 patients with acute STEMI treated with pPCI were included in this study. fQRS was defined as an additional R wave, notching of the R wave, notching of the downstroke or upstroke of the S wave, or more than one R' without a typical bundle branch block in at least two contiguous leads. QRSd was defined as the presence of the following in ≥2 adjacent leads: (1) emergence of the J point at ≥50% of the R wave amplitude in leads with a qR configuration or (2) disappearance of S wave in leads with an rS configuration. Patients were categorized into three groups according to the presence of fQRS and QRSd on ECG. Group I was defined as fQRS (-) and QRS distortion (-), group 2 was defined as fQRS (+) and QRS distortion (-), or fQRS (-) and QRS distortion (+), and group 3 was defined as both fQRS (+) and QRS distortion (+). An ECG example of a patient who had both fQRS and QRSd is showed in Fig. 1.

Results: Basal demographic characteristics, duration of chest pain on admission, MI localization, and length of hospitalization were similar among the three groups (Table 1). However, the patients in group III had a lower LVEF, higher maximum CK-MB levels, a lower ratio of STR, and a higher rate of three-vessel disease compared with the patients in groups I and II. Similarly, the patients in group II also had a lower LVEF, higher maximum CK-MB levels, a lower ratio of STR, and a higher rate of three-vessel disease compared with the patients in group I (Fig. 2). More importantly, in-hospital mortality rate of group III was the highest, and roughly 10-fold and 2-fold higher compared with groups I and II, respectively (Fig. 3). Multivariate analysis showed that age (OR: 1.04, 95% CI: 1.01-1.08, p=0.016), three-vessel disease (OR: 2.69, 95% CI: 1.26-5.73, p=0.010), group II (OR: 4.63, 95% CI: 1.46-14.73, p=0.009), and group III (OR: 8.84, 95% CI: 2.73-28.62, p<0.001) were independent predictors of in-hospital mortality (Table 2).

Conclusions: The combined use of QRSd and fQRS provides additional prognostic value compared with the presence of QRS distortion or fQRS alone for early risk stratification in patients with STEMI treated with pPCI.

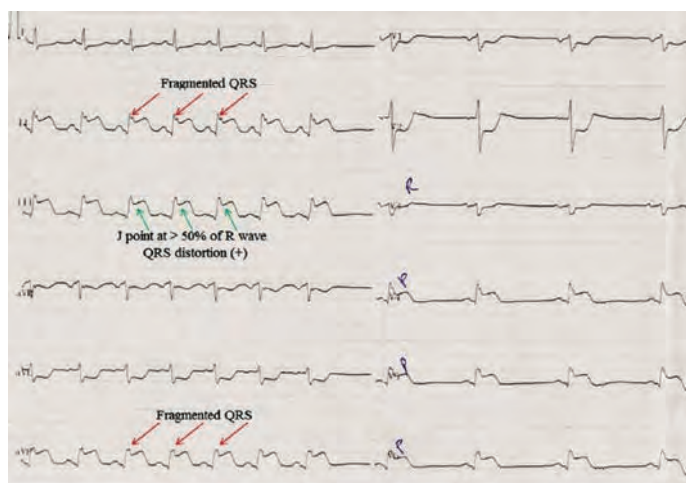


Figure 1. An ECG example of a patient who had both fQRS and QRS distortion.

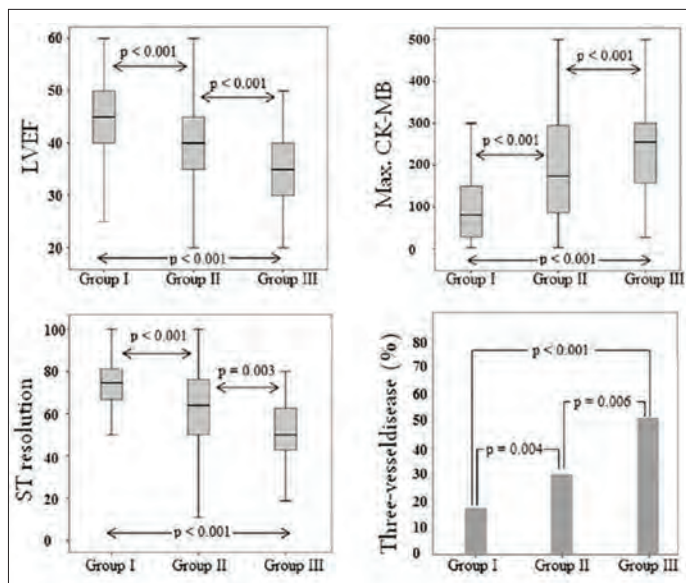


Figure 2. Left ventricular ejection fraction, maximum CK-MB, ST segment resolution, and the frequency of three-vessel disease according to three groups based on QRS distortion and fQRS.

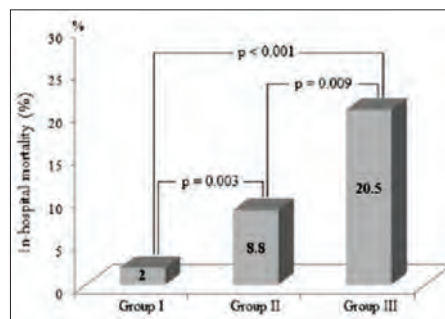


Figure 3. In-hospital mortality rates according to three groups based on QRS distortion and fQRS.

Table 1. Comparisons of three groups in term of baseline characteristics

Variables	Group I (n = 201)	Group II (n = 170)	Group III (n = 83)	p Value
Age (years)	61.0 ± 11.5	60.4 ± 11.7	63.7 ± 12.6	0.108
Gender, male (%)	155 (77.1)	133 (78.2)	61 (73.5)	0.699
Hypertension (%)	83 (41.3)	81 (47.6)	40 (48.2)	0.379
Diabetes mellitus (%)	39 (19.4)	34 (20.0)	25 (30.1)	0.111
Smoking (%)	123 (61.2)	113 (66.5)	48 (57.8)	0.357
SBP (mm Hg)	127.5 ± 23.4	128.9 ± 27.0	127.7 ± 23.8	0.854
DBP (mm Hg)	75.7 ± 15.4	77.9 ± 18.3	76.6 ± 16.6	0.459
Duration of chest pain on admission (min.)	162.1 ± 173.5	191.9 ± 250.1	168.0 ± 168.0	0.359
LVEF (%)	45.4 ± 7.0	40.6 ± 7.8	34.9 ± 6.2	<0.001
Creatinine (mg/dl)	0.9 ± 0.3	0.9 ± 0.2	1.0 ± 0.4	0.099
GFR (ml·min ⁻¹ ·73m ²)	95.2 ± 28.6	95.7 ± 26.1	89.7 ± 35.5	0.273
LDL-cholesterol (mg/dl)	122.8 ± 31.2	118.0 ± 33.7	122.8 ± 31.4	0.311
HDL-cholesterol (mg/dl)	37.2 ± 8.9	38.2 ± 9.9	37.4 ± 8.5	0.685
Maximum CK-MB (ng/ml.)	99.9 ± 81.2	179.6 ± 111.9	213.9 ± 98.9	<0.001
Maximum troponin (ng/ml.)	25.8 ± 16.1	41.6 ± 22.2	57.9 ± 23.8	<0.001
Duration of hospitalizations (days)	4.0 ± 0.4	3.8 ± 0.8	3.8 ± 1.0	0.066
STR ratio (%)	73.6 ± 18.2	61.7 ± 23.6	52.9 ± 13.3	<0.001
MI localization				0.821
Anterior	95 (47.3)	80 (47.1)	36 (43.4)	
Non-anterior	106 (52.7)	90 (52.9)	47 (56.6)	
Number of vessels with significant stenosis	1.6 ± 0.8	1.8 ± 0.9	2.2 ± 0.9	<0.001
Three-vessel disease (%)	16 (7.9)	52 (30.6)	43 (51.8)	<0.001
Proximal lesion (%)	73 (36.3)	88 (51.8)	67 (80.7)	<0.001
In-hospital mortality (%)	4 (2)	15 (8.8)	17 (20.5)	<0.001

Table 2. Independent predictors of in-hospital mortality

	Odds ratio	95% CI	p Value
Age	1.04	1.01-1.08	0.016
Three-vessel disease	2.69	1.26-5.73	0.010
Group I			
Group II	4.63	1.46-14.73	0.009
Group III	8.84	2.73-28.62	<0.001

Interventional cardiology / Coronary

OP-039

Development of thyroid dysfunction after intervention of coronary chronic total occlusion

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Background and Aim: We reported a retrospective study to determine the influence of extremely supraphysiological levels of iodide administered during percutaneous coronary interventions (PCIs) of chronic total occlusions (CTOs) on thyroid function when compared to PCI of non-complex coronary lesions, which has never been investigated before.

Methods: A total of 615 patients, 205 of whom underwent elective PCI of CTO lesions, group I and 410 of whom underwent elective non-complex (non-CTO, non-bifurcation, non-calcified) PCI, group II were enrolled in the study. Patients using antithyroid drugs or patients with manifest hyperthyroidism (low TSH and high FT4 and FT3) were excluded from the study.

Results: The flow-chart of our study was demonstrated in Figure 1. Ten of 186 (5.4%) euthyroid patients of group I developed subclinical hyperthyroidism, whereas 19 of 379 (5%) euthyroid patients of group II developed subclinical hyperthyroidism. There was no statistical difference among group I and II regarding developing subclinical hyperthyroidism, (p=0.854). However, 7 of 14 (50%) subclinical hyperthyroid patients of group I developed overt hyperthyroidism, whereas 3 of 25 (12%) subclinical hyperthyroid patients of group II developed overt hyperthyroidism, (p=0.019). In our trial, 29 of 565 (5.1%) euthyroid patients developed subclinical hyperthyroidism, whereas 10 of 39 (25.6%) patients with subclinical hyperthyroidism developed overt thyroid dysfunction (p<0.001).

Conclusions: There are not many studies investigating the influence of ICM first to coronary angiography on thyroid function. However, to our knowledge, this retrospective study is the first to evaluate the effect of extremely supraphysiological levels of iodide administered during PCI of coronary CTO. Moreover, in this study, not only euthyroid but also subclinical hyperthyroid patients were enrolled, which is different from the literature. Our study demonstrated that PCI of coronary CTO lesions did not increase the risk of developing subclinical hyperthyroidism compared to PCI of non-complex coronary lesions in euthyroid patients (%5.4 vs %5.1 p=0.854). However, PCI of coronary CTO lesions increased the risk of developing overt hyperthyroidism with subclinical hyperthyroidism at baseline compared to PCI of non-complex coronary lesions (%50 vs %12, p=0.019). Moreover, our study revealed an increased risk of developing further thyroid dysfunction in patients with subclinical hyperthyroidism at baseline, compared to euthyroid patients (%25.6 vs %5.1 p<0.001).

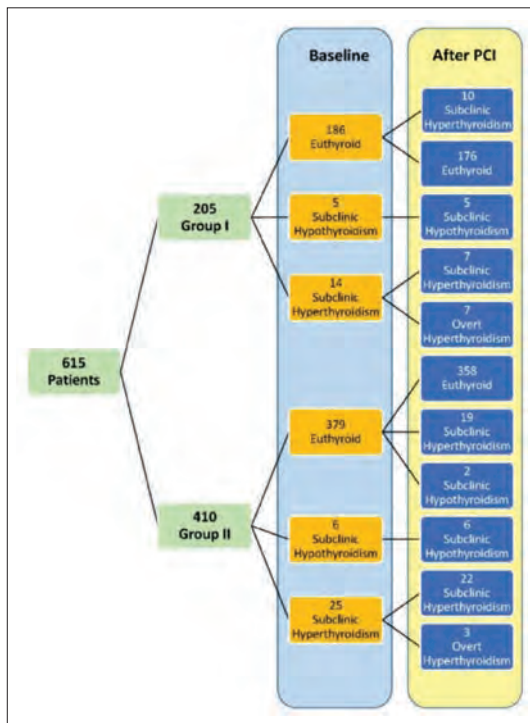


Figure 1. The flow-chart of the study.

Table 1. Baseline characteristics

Variables	Group I (n: 205)	Group II (n:410)	p
Age, years (mean)	60,9	62,7	0,042
Male, n, %	173, (84,4)	320, (78)	0,063
Diabetes Mellitus, n, %	83, (40,5)	163, (39,8)	0,861
Hypertension, n, %	101, (49,3)	191, (46,6)	0,530
Hyperlipidemia, n, %	106, (51,7)	200, (48,8)	0,494
Active Smoking, n, %	68, (33,2)	140, (34,1)	0,809
Ex-Smoker, n, %	77, (37,6)	144, (35,1)	0,552
History of MI	117, (57,1)	219, (53,4)	0,390
History of PCI	125, (61)	232, (56,6)	0,298
History of CVA	19, (9,3)	33, (8)	0,608
History of PAD	22, (10,7)	32, (7,8)	0,227
History of CHF	42, (20,5)	76, (18,5)	0,562

Epidemiology

OP-040

Clinical characteristics of Turkish HIV patients referred for cardiovascular evaluation: A pilot study

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Background and Aim: HIV infection is known to be associated with increased cardiovascular morbidity and mortality due to increased prevalence of cardiovascular (CV) risk factors, effects of anti-retroviral therapies including dyslipidemia, and the HIV virus itself. The success of highly active anti-retroviral treatments (ART) have translated into increased longevity of HIV patients making them prone to long-term effects of CV disorders making primary and secondary cardiovascular prevention of utmost importance in this patient group. Data regarding CV characteristics of Turkish patients treated for HIV is limited. We aimed to document clinical characteristics of HIV patients treated in a large tertiary infectious disease center who were referred for CV evaluation in a 3 year time span.

Methods: Data including demographics, history of prior CV disease, time since HIV diagnosis, CV risk factors including lipid parameters, diabetes mellitus, hypertension, smoking status, family history and any relevant positive findings in physical examination, as well as in CV tests carried out in an as needed basis were gathered from patient files retrospectively. In patients without history of any atherosclerotic CV disease,

risk scores were calculated to guide further therapy. CV recommendations including need for therapy for dyslipidemias were also noted.

Results: A total of 84 HIV patients were referred for CV evaluation. Demographic and clinical characteristics are summarized in Table 1. Clinically relevant CV disease was detected in 9 patients (10.7%). Atherosclerotic CV disease was present in 6 (7.1%) patients (2 coronary by-pass, 2 PCI, 3 medically managed stable angina). One patient had aortic valvular disease and paroxysmal SVT was detected in another patient. Reason for referral was evaluation for primary prevention due to abnormal lipid profile in 46 (54.8%) patients, CV symptoms in 32 (38.1%) patients, switching to abacavir therapy in 5 (6%) patients and prior history of CV disease in 1 patient. Among patients without CV disease 25 (34.7%) were found to have high CV risk using 2013 ACC/AHA Atherosclerotic Cardiovascular Disease (ASCVD) risk score. Statin therapy was recommended in 39 (40.5%) patients whereas fibrates were prescribed in 5 (6%) patients.

Conclusions: Evaluation of HIV patients referred to cardiology resulted in documentation of numerous individuals at high risk of CV disease and triggered change in treatment in considerable number of patients. Risk profile of the evaluated patients suggests that systematic CV evaluation of Turkish HIV patients may be warranted.

Table 1. Clinical characteristics of HIV patients referred for cardiovascular evaluation

Age (years)	49.3 +/- 12.6
Male sex (n,%)	65 (77.4)
HIV duration in months	36 (12-84)
Hypertension (n,%)	16 (19)
Diabetes (n,%)	5 (6)
Smoking History (n,%)	43 (51,2)
Family history of CVD (n,%)	25 (29,8)
Total cholesterol (mg/dl)	224 +/- 42
LDL cholesterol (mg/dl)	153 +/- 39
HDL cholesterol (mg/dl)	47 +/- 15
Triglycerides (mg/dl)	211 +/- 140

CVD: cardiovascular disease. Values are expressed as mean +/- standard deviation or median and interquartile range.

Epidemiology

OP-041

Is it really necessary to say whether octogenarians to stay in the coronary intensive care unit?

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Background and Aim: The elderly population increases in worldwide and these population needs more hospitalization than the others. Additional complications develop in this group of patients frequently and the length of hospital stay is significantly prolonged. Advanced age is an independent risk factor for mortality in coronary intensive care unit (CICU) admissions. Interventions, as compared to younger patients during CICU stay are less likely to undergo even after adjustment of severity of disease. In this study, possibility of protecting elderly patients away from coronary care units have been investigated.

Methods: Single center, retrospective and observational study analyzed 420 patients, whose age over 80 years were hospitalized in coronary intensive care unit in our institution. Interventions that can be done only in ICU not performed in wards have been noted and also CICU complications were analyzed. Primary outcomes were mortality and development of hemodynamic instability in CICU follow ups. Secondary outcomes were invasive procedures (invasive blood pressure monitoring, external or permanent pacing, cardioversion, defibrillation, coronary angiography etc.) that can be performed in CICU and detection of arrhythmia on monitor follow ups.

Results: The mean age of the patients were 84.12±4.62. 120 of the patients were hospitalized by acute coronary syndrome, 58 of them by pneumonia, 212 of them by decompensated heart failure and 30 of them by other diagnosis. The rate of patients had primary non-cardiac diagnosis at discharge was 17.6% (n=74). 19 patients were required cardiopulmonary resuscitation and 16 of them were exitus. 5 patients had shockable fatal arrhythmias on monitor follow-ups and underwent defibrillation and/or cardioversion. 19% (n=80) of the patients had invasive blood pressure monitoring, although only 8 of them had hemodynamic deterioration. 6 of the patients required external or permanent pacing. 12.8% of the patients have mild to severe delirium symptoms during admission. 9.76% patients had fewer and 87.8% of them treated with antibiotics. With the advanced age, the incidence of delirium and infections were incrementally increased.

Conclusions: Elderly patients have poorer outcome with prolonged CICU stays and very frequently have to deal with delirium and septicemia caused by hospital infections. Patients with primary cardiac pathology have benefit from CICU stay however, many of the patients have primary non-cardiac diagnosis at discharge. Patients in these age groups have worse outcomes and staying in CICU does not matter on mortality. Elderly patients can be followed up in wards until any hemodynamic and electrical instability, active chest pain or severe dyspnea. In this manner, physicians should establish a balance between patients' and his/her relatives' desire and the medical condition of the patient to decide admission on CICU.

Interventional cardiology / Cover and structural heart diseases

OP-042

Incidence and predictors of permanent pacemaker after transcatheter aortic valve implantation (TAVI) with Edwards Sapien XT device in patients with severe aortic stenosis due to bicuspid aortic valve

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Background and Aim: Transcatheter aortic valve implantation (TAVI) is being increasingly performed in patients with bicuspid aortic valve (BAV) stenosis. Cardiac conduction disturbances requiring permanent pacemaker (PPM) are a frequent complication of TAVI. However, there is a lack of data on PPM predictors after TAVI in patients with BAV stenosis. The aim of this study was to assess the incidence and the predictors of postoperative PPM requirement in BAV patients undergoing TAVI with Edwards Sapien XT. **Methods:** Sixty two patients who were diagnosed as BAV stenosis by multislice computed tomography (MSCT) and transthoracic echocardiography (TTE) from 416 patients who underwent TAVI with Edwards Sapien XT device due to symptomatic severe aortic stenosis were included retrospectively in Ankara Yıldırım Beyazıt University, Department of Cardiology between November 2012 and January 2018. Preprocedural clinical, anatomic, electrocardiography (ECG), MSCT measurements and procedural features of the patients were evaluated by univariate analysis for predictors of PPM implantation. All variables that are significant for PPM implantation in univariate analysis (p<0.05) were entered into a multivariate logistic regression analysis model. **Results:** Post-TAVI PPM implantation was performed in 8 patients, with an incidence of 12.9%. All patients who underwent PPM implantation were found to have bicuspid type 1 right coronary cusp-left coronary cusp (L-R) fusion. In univariate analysis, it was observed that the large diameter of the aortic annulus measured by TTE before treatment had a tendency to implant the PPM but did not reach statistical significance (p=0.07). For univariate analysis of post-TAVI PPM implantation; preprocedural right bundle branch block (RBBB) (p<0.0001), shortness of membranous septum in MSCT (p<0.0001), long aortic annulus-left main coronary artery (LMCA) distance (p=0.04) were determined statistically significant. When these variables were tested in multivariate logistic regression analysis, the preprocedural RBBB (p<0.0001) and the short length of the membranous septum (p=0.03) were determined statistically significant. In multivariate logistic regression analysis, a statistically significant trend was observed in the increase of aortic annulus-LMCA distance (p=0.08). **Conclusions:** The presence of RBBB and the short length of membranous septum before the procedure were found to be independent risk factors for post-TAVI PPM implantation in patients with BAV treated with Edwards Sapien XT. The type 1 (L-R) patients in the bicuspid patient group were may be at risk for PPM implantation after TAVI. Large annulus diameter and increased annulus-LMCA distance might be risk factors for post-TAVI PPM implantation in patients with BAV stenosis. Further prospective randomized studies with available number of patients size are required to evaluate predictors of PPM after TAVI in BAV.

Interventional cardiology / Cover and structural heart diseases

OP-043

Improvement in mitral regurgitation after mitraclip: value of three-dimensional transesophageal echocardiographic assessment of mitral geometry

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Background and Aim: The effects of edge-to-edge percutaneous mitral valve repair on the shape and size of the mitral annulus and its relation to mitral regurgitation (MR) have not been characterized. This study evaluates the acute changes in mitral annular shape and dimensions, and their effect on MR severity, in patients with functional and degenerative MR following MitraClip®. **Methods:** Patients that underwent percutaneous edge-to-edge mitral repair between January 2013 and May 2016 at our institution were retrospectively reviewed. Exclusions: patients with inadequate images, prior mitral valve repair, and rapid atrial fibrillation. Digitally stored TEE 3D images acquired intraprocedurally prior and immediately after implantation of MitraClip® were analyzed by using software to model the mitral valve apparatus. **Results:** Of seventy-eight patients that underwent MitraClip® procedure between January 2013 and May 2016, 60 were eligible. Mean age was 78.3±11 years. Severe MR (4+) was present in 37 patients (62%), moderately severe MR (3+) in 23. All patients achieved MR reduction to ≤2+. End systolic and end diastolic measurements of 3D annular circumference, bicommissural diameter and anteroposterior diameter had a significant size reduction after MitraClip®. There was a significant correlation between reduction in MR severity and reduction in the bicommissural annular dimension (p=0.003). **Conclusions:** In patients with functional or degenerative MR, the MitraClip® affects leaflet coaptation and modifies the annulus with secondary effects. A reduction of the bicommissural dimension is associated with MR severity reduction.

Table 1. Echocardiographic parameters of all patients in terms of anatomical changes

n=60	Pre-MitraClip® (mean)	Post-MitraClip® (mean)	Δ mean	p value
3D annular circumference (mm)-end systole	130 ± 17.4	126.4 ± 17.5	-3.583	< 0.0001
3D annular circumference (mm)-end diastole	133.02 ± 17.6	128.27 ± 17.11	-4.75	< 0.0001
Bicommissural diameter (mm)-end systole	41 ± 5.5	39.6 ± 3.6	-1.482	< 0.0001
Bicommissural diameter (mm)-end diastole	41.6 ± 5.7	40.2 ± 5.3	-1.417	< 0.0001
Anteroposterior diameter (mm)-end systole	35.2 ± 4.8	30.4 ± 4.9	-2.807	< 0.0001
Anteroposterior diameter (mm)-end diastole	34.7 ± 4.5	31.5 ± 5	-3.188	< 0.0001

Interventional cardiology / Cover and structural heart diseases

OP-044

The relation between lead aVR findings and mortality in patients with transcatheter aortic valve implantation

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Background and Aim: Our aim was to determine the relation between the lead aVR findings in before and after procedure surface ECGs and mortality in transcatheter aortic valve implantation (TAVI) patients. **Methods:** Pre and post-procedural (after 24 hours) 12 lead surface ECGs of all patients were recorded (Figure 1a, 1b). QRS duration and axis, P wave duration, PR interval, QT and QTc durations, the existence of LBBB were recorded. Negative and positive numeric dates were recorded according to the below and above location of ST segment in lead aVR (STaVR). T wave polarity (TPaVR) was measured depending on the PR segment in lead aVR so that negative values below segment (<0), positive numeric values above segment (0≥) and through total vector magnitude for biphasic values. Absolute value of TPaVR and STaVR were calculated. Then, a ratio was obtained from the division of larger absolute value by smaller one (the ratio: |larger value|/|smaller value|). Diameters of aortic annulus in mid-systolic phase, ascending aorta sinotubular junction, sinus valsalva, ascending aorta and distance between aortic annulus and left main coronary artery (LMCA) in mid-diastolic phase were measured with TOE before TAVI (Figure 2). **Results:** The patients were divided into two groups as living and deceased. The living group had 36 patients (mean age 76.3±8.6 years, mean follow-up period 15.0±11.4 months) and the deceased group had 18 patients (mean age 73.7±7.0 years, mean follow-up period 14.7±8.7 months). There were no statistical differences in the demographic variables between two groups (Table 1). TPaVR after TAVI (p=0.037) and the ratio were significantly higher in the deceased group (p=0.016, Table 2). The deceased group had significantly shorter aortic annulus-LMCA distance (p=0.019) and all other echocardiographic measurements were similar (Table 3). Coronary angiography findings were similar in both groups (Table 4). Aortic annulus-LMCA distance (OR:0.552, 95% CI:0.311-0.980, p=0.042) and the ratio (OR:1.670, 95% CI:1.209-2.306, p=0.002) were determined as independent predictors for mortality in the binominal logistic regression analysis (Table 5). **Conclusions:** If there is an ST segment and T wave changes in patients with TAVI, they should be closely monitored.

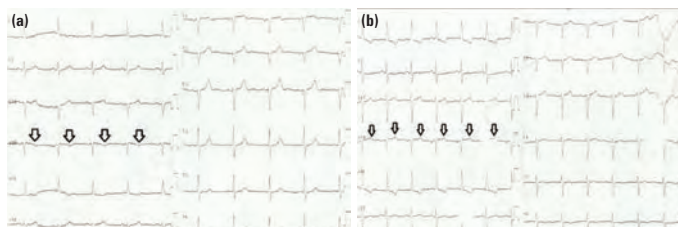


Figure 1. (a, b) Demonstrated of negative T wave on surface ECG before transcatheter aortic valve implantation (arrows).



Figure 2. Demonstrated of distance between aortic annulus and left main coronary artery with transesophageal echocardiography.

Table 1. Comparison of patients demographic findings

	Living (n=36)	Deceased (n=18)	p
Age (years)	76.3 ± 8.6	73.7 ± 7.0	0.275
Male gender, n, (%)	15 (41.7)	5 (27.8)	0.319
Systolic blood pressure (mmHg)	117.5±9.8	119.0±8.5	0.591
Diastolic blood pressure (mmHg)	71.3±8.4	72.5±7.8	0.6
Pulse (beat/minute)	70.8 ±12.9	73.1 ±20.2	0.622
BMI (kg/m2)	28.6±5.8	28.6±1.8	0.986
Smoking, n (%)	8 (22.2)	6 (33.3)	0.380
DM, n (%)	10 (27.8)	6 (33.3)	0.673
HT, n (%)	24 (66.7)	13 (72.2)	0.679
HPL, n (%)	10 (27.8)	4 (22.2)	0.661
Stroke, n (%)	2 (5.6)	0	0.547
CAD, n (%)	18 (10.5)	0 (12.8)	0.595
COPD, n (%)	7 (19.4)	6 (33.3)	0.26
STS-PROM score, n	10.4±1.8	10.8±2.0	0.479

BMI: body mass index, CAD: coronary artery disease, COPD: chronic obstructive pulmonary disease DM: diabetes mellitus, HT: hypertension, HPL: hyperlipidemia, STS-PROM: Society of Thoracic Surgeons probability of mortality.

Table 2. Comparison of patients electrocardiographic findings

	Living n=36	Deceased n=18	p
QRS before TAVI (msn)	98.1±21.2	91.6±17.2	0.315
QRS after TAVI (msn)	119.7±26.5	105.4±23.4	0.167
P duration before TAVI (ms)	108.2±27.3	108.3±19.7	0.991
P duration after TAVI (ms)	102.7±11.5	102.0±10.4	0.931
PR interval before TAVI (ms)	165.1±25.1	161.7±24.7	0.630
PR interval after TAVI (ms)	179.7±34.3	173.0±20.1	0.590
QT interval before TAVI (ms)	417.6±35.9	394.7±41.4	0.081
QT interval after TAVI (ms)	424.4±52.8	448.8±45.0	0.434
QTc interval before TAVI (ms)	443.6±32.9	423.2±30.1	0.057
QTc interval after TAVI (ms)	459.7±30.3	445.7±35.8	0.23
QRS axis before TAVI (°)	30.1±43.4	3.8±31.4	0.224
QRS axis after TAVI (°)	-7.8±44.2	-27±22.3	0.478
LBBB before TAVI, n (%)	5 (13.9)	2 (11.1)	0.775
LBBB after TAVI, n (%)	6 (44.4)	5 (27.8)	0.236
Fragmentation, n (%)	10 (27.8)	2 (11.1)	0.298
Positive TPavR before TAVI, n (%)	8 (22.2)	6 (33.3)	0.380
Positive TPavR after TAVI, n (%)	7 (20)	9 (50)	0.024
TPavR before TAVI (mV)	-0.6±1.7	-0.5±1.5	0.828
TPavR after TAVI (mV)	-0.6±1.9	0.5±2.0	0.037
STaVR before TAVI (mm)	1.0±0.8	1.1±1.1	0.749
STaVR after TAVI (mm)	1.0±0.6	0.9±0.7	0.561
Ratio before TAVI, n	1.8±0.9	2.8±2.3	0.102
Ratio after TAVI, n	2.6±1.9	5.4±3.9	0.016

TAVI: transcatheter aortic valve implantation, TPavR: T wave polarity in lead aVR, LBBB: left bundle branch block, STaVR: ST deviation in lead aVR *Ratio: The absolute value of bigger one (STaVR or TPavR)/ The absolute value of smaller one (STaVR or TPavR).

Table 3. Comparison of patients echocardiographic findings

	Living n=36	Deceased n=18	p
EF (%)	40.1±4.6	41.1±4.8	0.492
LaD (mm)	42.7±4.7	41.3±4.7	0.681
Gradient before TAVI (mmhg)	57.8±12.1	56.1±10.1	0.593
Gradient after TAVI (mmhg)	11.9±2.8	12.8±2.8	0.271
AVA (cm ²)	0.8±0.1	0.7±0.2	0.219
Aortic annulus (cm)	2.2±0.2	2.1±0.8	0.782
Sinus valsalva (cm)	3.1±0.2	2.9±1.5	0.210
Asendan aorta (cm)	3.2±0.3	3.2±0.6	0.955
Paravalvular leak after, n (%)	1 (2.8)	1 (5.6)	0.560
Prosthetic valve size (mm)	25.8±2.6	26.1±2.1	0.714
Distance between aortic annulus and LMCA (mm)	10.8±1.6	9.9±0.3	0.019
PAPs (mmhg)	42.0±8.1	40.0±13.2	0.782

EF: ejection fraction, LaD: left atrial diameter, TAVI: transcatheter aortic valve implantation, AVA: aortic valve area, PAPs: systolic pulmonary artery pressure, LMCA: left main coronary artery.

Table 5. Independent predictors for mortality in TAVI patients

	Odds ratio	95% Confidence Interval	p
BUN	1.026	0.982-1.071	0.247
Albumin	0.539	0.122-2.389	0.416
Distance between aortic annulus and LMCA	0.552	0.311-0.980	0.042
Ratio after TAVI	1.692	1.211-2.363	0.002

BUN: blood urea nitrogen, TAVI: transcatheter aortic valve implantation, LMCA: left main coronary artery *Ratio: The absolute value of bigger one (STaVR or TPavR)/ The absolute value of smaller one (STaVR or TPavR).

Table 4. Comparisons of patients angiographic findings

	Living n=36	Deceased n=18	p
LMCA, n (%)	4 (11.1)	2 (11.1)	1
LAD, n (%)	7 (19.4)	4 (22.2)	0.811
CX, n (%)	7 (19.4)	3 (16.7)	0.804
RCA, n (%)	4 (11.1)	2 (11.1)	1
SS, n	3.8±7.6	3.6±7.4	0.880

CX: circumflex artery, LAD: left anterior descending artery, LMCA: left main coronary artery, RCA: right coronary artery, SS: syntax score.

Results: The mean age of study populations was 32.7±10.2 years old (range between 14-66 years old). There were 33 women (58.9%) and 23 men (41.1%) (n=56). The total correction operation age was mean 6.3±4.85 years old (range between 6 months and 30 years old). The mean follow up ranges between 9 and 45 years. 34 of 56 patients (61%) were in NYHA functional class I, the rest 22 of 56 patients in NYHA functional class II (39%). None of the patients were in NYHA III and IV. According to 24 hour ambulatory holter recordings, 8 patients (14.3%) had supra-ventricular tachycardia (SVT), 7 patients (12.5%) had non-sustained ventricular tachycardia (VT). After total correction operation, one patient was implanted ICD due to sustained VT. There were 53 patients with normal sinus rhythm, 2 patients with atrial fibrillation and 1 patient with pacemaker rhythm. The mean NT-proBNP level was ranged between 17-623 pg/ml. Mean TAPSE was 1.7±0.2 mm. Mean tricuspid annulus peak S' velocity was 10.3±2.4 mm. Pulmonary regurgitation was severe in 4 patients (7.1%), moderate in 9 (16.1%) patients and mild in 43 patients (76.8%). There was no statically significant correlation between RVEF and severity of PR. There was only correlation between TAPSE and BNP (r=0.38, p=0.04). There was no difference between patients with normal RV function and with RV dysfunction in the meaning of QRS duration and mean BNP level.

Conclusions: After total correction of ToF, patients should be followed up for progression PR, presence of RV dysfunction, RVOT obstruction and arrhythmias. Re-operation may be necessary within years. BNP is also useful follow up parameters to diagnose RV dysfunction earlier.

Table 1. Association between BNP and echocardiographic RV systolic functional parameters

Parameters	Correlation coeffic.	BNP
FAC (%; mean ± SD)		0,11
	P value	0,47
TAPSE (cm; mean ± SD)	Correlation coeffic.	0,32
	P value	0,04
S' (cm/sn; mean ± SD)	Correlation coeffic.	0,01
	P value	0,92

Interventional cardiology / Coronary

OP-046

Feasibility of left distal radial artery access for percutaneous coronary interventions

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Background and Aim: Left distal radial artery access site has emerged as a new technique for coronary angiography procedures. By introducing the radial sheath into the left fossa radialis or the so called 'Anatomic snuffbox' we aimed to assess the feasibility of this new access site for coronary interventions.

Methods: The left distal radial artery was used as radial access site for 46 patients admitted for coronary interventions to our clinic from June 2017 to April 2018. All the patients had a prominent pulse in their left forearm and distal radial artery. Each patient's left arm was gently bent into his right groin with slight adduction and comfortable position of the hand. The operator stood at the right side of the patient where he could make the arterial puncture and continue with coronary interventions. All the patients had a cocktail of weight adjusted heparine, nitrate and serum physiologic to prevent radial artery occlusion. Demographic features and complications were recorded during the hospital stay.

Results: Mean age was 61.4 and 87% of them was male. Puncture time to left distal radial artery was less than one minute. We used Judkins catheters for all the procedures with 6 French dimension. The most predisposing cardiac risk factors were hypertension and smoking with 80.4% and 26.1% respectively. Half of patients had chronic coronary artery disease with previous interventions. Acute coronary syndrome was diagnosed in 15 patients (36.6%) of which 11 of them had primary angioplasty intervention. Elective interventions were performed in 31 patients (67.4%). All the interventions were successfully contemplated without any serious complication. Left anterior descending coronary artery was the most common artery requiring intervention. Two patients with left main coronary artery disease were successfully stented without any complication. Multivessel intervention at the same time was performed in 7 patients (15.3%). Spasm was seen in only one patient that was resolved with intraarterial nitrate. There were no radial occlusion, nor hematoma or bleeding events. The radial sheath was removed at the termination of the procedure with hemostasis provided by manual compression. Patients were discharged in a mean time of 2.2 days.

Conclusions: Left distal radial artery seems to be a feasible access site for coronary interventions. A learning curve is required for the operator to perform a successful intervention.



Figure 1. The introduction of 6 French hydrophilic radial sheath into the left distal radial artery in the anatomical snuffbox.



Figure 2. Operator on the right side of the patient performing coronary intervention with left distal radial access.

Congenital heart disease

OP-045

Long term follow-up results of adult operated ToF patients

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Background and Aim: We aimed to review the long term clinical and echocardiographical data, presence of arrhythmias, assessment of ventricular function of patients with operated ToF.

Methods: We retrospectively reviewed the medical records of operated ToF patients that was followed up at adult congenital heart disease outpatient clinic of our university hospital between January 2015 and June 2017.



Figure 3. Successful inferior STEMI primary intervention of the right coronary artery.

Table 1. Demographic features of study population

Age	61.4±12.2 (40-87)
Length (cm)	170.9±6.5 (153-185)
Weight (kg)	80±10.7 (53-110)
LVEF (%)	0.51±0.7 (0.22-0.60)
Hospital stay (days)	2.2 ± 1.9 (0-10)
Male	40 (87%)
Female	6 (23%)
DM	17 (37%)
HT	37 (80.4%)
AF	2 (4,3%)
Smoking	12 (26.1%)
Chronic CAD	25 (54.3%)
CABG	3 (6.5%)
Peripheral artery disease	1 (2.2%)
Mechanic prosthetic heart valve	1 (2.2%)

Table 3. Complications of the procedure n=46 (%)

Radial occlusion	0
Radial spasm	1 (2.1%)
Hematoma	0
Numbness	0
Left arm neurologic deficit	0

Methods: 48 patients with intermediate coronary lesions was scheduled for FFR and FLAME ratio. Heparin (5000 IU) was administered at the beginning of the procedure followed by an intracoronary bolus of nitroglycerine (0.1 mg). Aortic pressure (Pa) was measured via a 6 Fr guiding catheter. Intracoronary distal pressure (Pd) was measured simultaneously using a 0.014 pressure-sensor guidewire. FLAME was calculated via length of related coronary artery, passage time of contrast, cross sectional area. (FLASH flow (ml=ml)=[(Distance (cm)/Time (s)) * mean cross sectional area (cm²)] * 60). First basal FLAME and basal FFR measured. After administration of i.v. adenosine 140 µg/kg/min in three minutes FFR was measured and FLAME was calculated in the same angiographic view of basal. FLAME ratio was obtained by dividing basal FLAME to hyperemic state FLAME. Correlation of FLAME ratio and FFR was assessed.

Results: Baseline characteristics of patients were in table 1. There is a significantly correlation between FLAME ratio and FFR. (r=0.765 p<0.001). ROC curve analysis showed, FLAME ratio <0.839 in predicting FFR <0.80; AUC:0.899, sensitivity 0.84 specificity 0.97.

Conclusions: FLAME ratio is a simple method in predicting intermediate coronary lesions severity. We found that the cut-off value for FLAME ratio is <0.839 for assessment of coronary stenosis severity. If FFR measurements facility can't available, FLAME ratio can be used for evaluation for coronary stenosis severity.

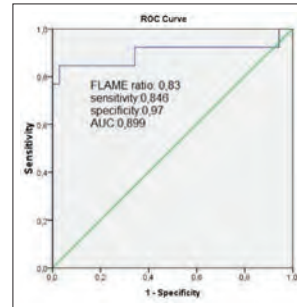


Figure 1. ROC curve of FLAME ratio.

Table 1. Baseline characteristics of patients (n=48)

Age	57,75±10,42
DM	22 (%45,8)
HT	27 (%56,2)
Smoking	13 (%27)
Dyslipidemia	20 (%41,6)
Prior MI	6 (%12,5)
PCI	16 (%33,3)
Hereditary	7 (%14,5)
Medications	
ASA	48 (%100)
P2Y12 inhibitors	23 (%48)
Beta-blockers	22 (%45,8)
RAS antagonists	19 (%39,5)
CaCBs	4 (%8,3)
Statins	21 (%43,7)

Table 2. Procedural features during percutaneous coronary intervention n=46 (%)

Radial sheath (6 French)	46 (100%)
Judkins catheters (6 French)	46 (100%)
Acute coronary syndrome	15 (36.6%)
Anterior STEMI	5 (10.9%)
Inferior STEMI	6 (13%)
Non-STEMI	4 (8.7%)
Elective PCI	31 (67.4%)
Left anterior descending artery intervention	19 (47.9%)
Left circumflex artery intervention	9 (19.7%)
Right coronary artery intervention	16 (30.4%)
Left main coronary artery intervention	2 (4.4%)
Multivessel intervention	7 (15.3)
DES	39 (84.9%)
BMS	7 (15.3%)
Baloon+ Stent	25 (54.3%)
Stent only	19 (41.3%)
Artery puncture time (minutes)	0.85±0.69 (0.5-3.0)
Fluoroscopy time (minutes)	15.5±11.5 (4-63.4)
Radiation exposure (Total air kerma: mGy)	13464±9865 (2347-58325)
Radiation exposure (Total doze area product: µGy·m2)	19287±1493 (309-7610)

Interventional cardiology / Coronary

OP-047

A novel predictor of intermediate coronary artery stenosis severity: Fluoroscopy assisted measuring of coronary volume (FLAME) Ratio

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Background and Aim: Fluoroscopy assisted scoring of myocardial hypoperfusion (FLASH) method was used to estimate coronary blood flow in primary PCI patients. That was a simple and reliable method to show coronary blood flow. The distal flow volume of coronary arteries will be low in severe coronary artery stenosis. In hyperemic state; the blood flow volume increasing in stenotic coronary arteries are lower than normal coronary arteries. In this study we measured the ratio of basal coronary blood volume and hyperemia mediated coronary blood volume with FLASH method for evaluating the coronary stenosis severity. The accuracy of fluoroscopy assisted measuring of coronary volume (FLAME) ratio was evaluated with standard FFR.

Interventional cardiology / Coronary

OP-048

Evaluation of radial artery endothelial functions in transradial coronary angiography

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Background and Aim: Transradial as a new method for coronary angiography and interventions has recently increased its popularity. But endothelial dysfunction may occur after transradial coronary angiography. We aimed to investigate the endothelial functions and the most reliable intervention through flow mediated vasodilatation test in three different radial access sites: right forearm radial artery, left forearm radial artery and left distal radial artery.

Methods: Transradial access was used in 70 patients scheduled for coronary angiography and intervention from September 6-th 2017 and March 6-th 2018, prospectively. They were divided into 3 groups: group 1 left forearm radial (17 patients), group 2 left distal radial (27 patients) and group 3 right forearm radial artery (26 patients) access. Flow mediated endothelial functions were measured 3 times; on admission, 24 hour after and 2 months after the procedure. The test was performed with the patient supine, relaxed for 5 minutes. GE Healthcare Vivid E9 4D Cardiovascular ultrasound system instrument, 11L-D, 4.5-12 MHz linear probe was used for radial artery diameter measurement. Arm cuff inflation till 220 mmHg lasted for 5 minutes. After deflation radial artery diameter and their percentage change was recorded in the 1.st, 2nd and 3rd minute. All the data was evaluated by the IBM SPSS Statistics 21.0 programme. Kruskal Wallis test and Anova test was performed for comparison of variables.

Results: Mean age was 58.8 (±12.3) with male predominance 48 (68%), 5) and hypertension 54 (77.1%). Diabetes 28 (40%) were the most common risk factors. Radial artery diameters and percent change reached the maximum level in the first minute of flow-through dilation test. In the first 24 hours endothelial function is preserved in left distal intervention group compared to right intervention group (p<0.001) and left intervention group (p=0.043). This preservation continued at 2 months after the procedure. Radial artery thrombosis 6 (8.6%), ecchymosis 4 (5.7%) and radial artery occlusion 2 (2.9%) were seen in patients but there was no statistical difference between the 3 groups. Only one complication (ecchymosis) was seen in the left distal radial artery access site. There was no statistically significant difference between the 3 groups at the time of fluoroscopy and stent implantation.

Conclusions: The radial artery endothelial function is impaired in the first 24 hours but endothelial function is protected by the attempts made in the distal left access compared to the attempts made by the method right-left konvansiyonel. After long-term endothelial dysfunction is reduced. Left distal radial access is a reliable method of angiography intervention with less risk of arterial vasospasm, occlusion, major bleeding, hematoma. Left distal radial coronary angiography is a method of CAG procedure in which patients are highly satisfied and can recommend to their relatives because the treatment comfort is high and the rate of causing pain that disturb daily activity is low.



Figure 1. Coronary angiography via radial artery intervention.



Figure 2. Flow mediated dilatation test via by GE Healthcare Vivid E9 4D Cardiovascular ultrasound system instrument, 11L-D, 4.5-12 MHz probe and blood pressure cuff.

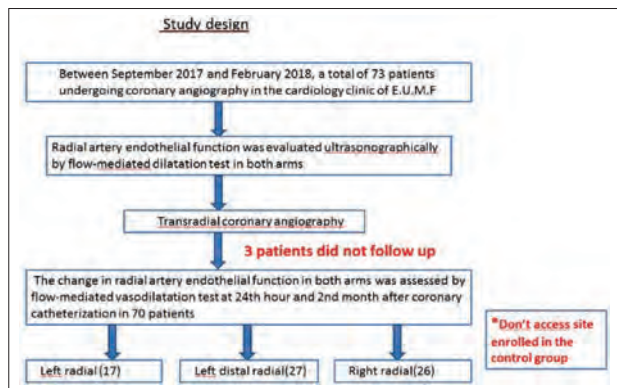


Figure 3. Study design.



Figure 4. Transradial intervention equipments.

Table 1. Demographic data

	Left radial	Left distal radial	Right radial	Total	P value
Age	58,5 (±14,56)	57,6 (±12,5)	60,3 (±10,8)	58,8 (±12,3)	0,720
Sex(Male)	15(88,2)	17(63)	16(61,5)	48(68,5)	0,133
Height	171,1 (±5,6)	169,8 (±8,5)	167,5 (±8,2)	169,3 (±7,8)	0,311
Weight	78,4 (±11,5)	83,8 (±16,3)	79,3 (±13,4)	80,8 (±14,2)	0,375
BMI	26,7 (±3,9)	27,9 (±7,1)	28,1 (±3,9)	27,7 (±5,3)	0,487
Pulse	76,7 (±11,8)	79 (±23,1)	75,8 (±13,0)	77,2 (±17,3)	0,963
SBP	133,2 (±12,5)	139,6 (±19,6)	147,0 (±22,9)	140,8 (±20,0)	0,081
DBP	75,6 (±12,6)	83,3 (±14,9)	79,0 (±12,5)	79,8 (±13,6)	0,181
NHYA I	12(70,6)	15(55,6)	16(61,5)	43(61,4)	0,608
Smoking	6(35,3)	9(33,3)	6(23,1)	21 (30,0)	0,840

Demographic data N (±Standart deviation) n (%).

Table 2. Comorbidities according to access site

	Left radial N(%)	Left distal radial N(%)	Right radial N(%)	Total N(%)	P value
Hypertension	11(67,4)	21(77,8)	22(84,6)	54(77,1)	0,313
Diabetes mellitus	7(41,2)	11(40,7)	10(38,5)	28(40)	0,979
Coronary artery disease	6(35,3)	12(44,4)	9(34,6)	27(38,6)	0,725
Hyperlipidemia	7(41,2)	13(48,1)	6(23,1)	26(37,1)	0,155
Anemia	1(5,9)	2(7,4)	3(11,5)	6(8,6)	0,781
Peripheral artery disease	2(11,8)	2(7,4)	2(7,7)	6(8,6)	0,863
Heart failure	2(11,8)	3(11,1)	0(0)	5(7,1)	0,564
Chronic renal failure	2(11,8)	1(3,7)	2(7,7)	5(7,1)	0,594
Chronic obstructive lung disease	2(11,8)	0(0)	3(11,5)	5(7,1)	0,184
Heart valve disease	2(11,8)	2(7,4)	0(0)	4(5,7)	0,238
Thyroid disease	1(5,9)	0(0)	3(11,5)	4(5,7)	0,195
Atrial fibrillation	0(0)	1(3,7)	1(3,8)	2(2,9)	0,718
Stroke/TIA	1(5,9)	0(0)	0(0)	1(1,4)	0,206

Table 3. Echocardiographic findings

	Left radial	Left distal radial	Right radial	Total	P value
LVEF	51,7 (±11,8)	53,7 (±10,7)	55,2 (±6,6)	53,6 (±9,4)	0,440
LVEDD	4,9 (±0,5)	4,8 (±0,4)	4,7 (±0,6)	4,8 (±0,5)	0,294
LA	4,0 (±0,8)	3,9 (±0,5)	3,7 (±0,5)	3,8 (±0,6)	0,158
Moderate- severe MR	5(29,4)	3(11,1)	3(11,5)	11(15,7)	0,478
Moderate- severe AR	1(5,8)	2(7,4)	4(15,3)	7(10)	0,155
Moderate- severe TR	4(23,5)	6(22,2)	4(15,3)	14(20)	0,234
LVDD	10(58,8)	16(59,3)	22(84,6)	48(68,5)	0,085
SPAP	26,1 (±18,8)	22,3 (±11,9)	24,3 (±10,4)	24,0 (±13,3)	0,694
IVS	1,09 (±0,12)	1,08 (±0,14)	1,15 (±0,18)	1,11 (±0,15)	0,582
RV diameter	2,64 (±0,34)	2,63 (±0,34)	2,59 (±0,30)	2,62 (±0,32)	0,872

Echocardiographic findings according to the location of the procedure N(± standart deviation) N(%) N: number of patients MR:Mitral regurgitation AR: Aort regurgitation TR: Triküspid regurgitation.

Table 4. Control group

	Baseline	30.sn	1.min	2.min	3.min
Diameter change	0,2499 (±0,028)	0,2586 (±0,032)	0,2846 (±0,031)	0,2774 (±0,031)	0,2649 (±0,028)
Percentage change		3,4296 (±4,551)	13,966 (±3,844)	11,095 (±3,318)	5,9927 (±3,797)

Control group radial artery diameter change by flow mediated dilatation test Parameters are defined by centimetre-cm and percentage-%(±standart deviation).

Table 5. Radial artery diameter and FMD change at the time before and after intervention

	Left radial artery diameter (Percentage change)	Left distal radial artery diameter (Percentage change)	Right radial artery diameter (Percentage change)	P value
Before angiography	0,30 ±0,02 (13,2 ±2,2)	0,27 ±0,03 (13,66 ±2,16)	0,27±0,02 (12,30 ±3,53)	0,952
After 24.hours	0,32 ±0,02 (6,06 ±3,15)	0,31 ±0,02 (8,59 ±3,39)	0,30 ±0,02 (3,71 ±2,31)	Left distal-left 0,043 Left distal Right 0,001
After 2. month	0,30 ±0,03 (11,09 ±2,58)	0,28±0,02 (12,10 ±4,24)	0,29 ±0,02 (10,21 ±2,34)	0,079

Radial artery diameter and FMD change at the time before and after intervention Parameter is defined by centimetre (cm).

Table 6. Complication according to access site

	Left radial(17) N(%)	Left distal radial(27) N(%)	Right radial(26) N(%)	Total(17) N(%)	P değeri
Radial thrombosed areas	2(11,8)	0(0)	3(11,5)	6(8,6)	0,184
Ecchymosis	1(5,9)	1(3,7)	2(7,7)	4(5,7)	0,822
Occlusion	1(5,9)	0(0)	1(3,8)	2(2,9)	0,485
Hematoma	0(0)	0(0)	1(3,8)	1(1,4)	0,480
Radial spasm	0(0)	0(0)	1(3,8)	1(1,4)	0,424
Pseudoaneurysm	0(0)	0(0)	1(3,8)	1(1,4)	0,424

Numbers in paranthesis are presented in percentage.

Table 7. Patient satisfaction according to access site

	Yes N(%)	No N(%)	I do not know N(%)	P value
Are you satisfied with the transradial coronary angiography procedure?	Left radial group 13(76,5) Left distal radial group 26(96,3) Right radial group 15(57,7) Total 54(77,1)	Left radial group 1(5,9) Left distal radial group 0(0) Right radial group 1(3,8) Total 2(2,9)	Left radial group 3(17,6) Left distal radial group 1(3,7) Right radial group 10(38,5) Total 14(20)	0,017
Would you recommend transradial intervention if your relatives have angiography?	Left radial group 14(82,4) Left distal radial group 26(96,3) Right radial group 16(61,5) Total 56(80)	Left radial group 2(11,7) Left distal radial group 0(0) Right radial group 3(11,5) Total 5(7,1)	Left radial group 1(20) Left distal radial group 0(0) Right radial group 7(26,9) Total 9(12,9)	0,021
Do you have a pain that disturbs the daily activity caused by transradial coronary angiography?	Left radial group 3(17,6) Left distal radial group 1(3,7) Right radial group 6(23,1) Total 9(12,8)	Left radial group 14(82,4) Left distal radial group 26(96,3) Right radial group 20(76,9) Total 61(87,2)		0,034

Interventional cardiology / Coronary

OP-049

Optical coherence tomography-verified longer balloon inflation time may provide better stent apposition and optimal index parameters

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Background and Aim: The study aim is to determine the optimal balloon inflation time required for optimal stent expansion and apposition.

Methods: We prospectively enrolled 38 patients with stable angina pectoris in whom single significant de novo coronary artery stenosis. All patients received Xience pro (Abbott Vascular) stent implanted with nominal pressure (12 atm) and stent balloon inflation of 10 s. Afterwards, stent balloon was reintroduced fully covering the implanted stent and re-inflated at the same pressure for additional 20s. Lastly, stent balloon was again re-introduced fully covering the implanted stent and re-inflated at the same pressure for additional 30 s this time. OCT images were acquired immediately by using the same catheter following each balloon inflation (first, second and third).

Results: Number of malapposed struts (213.1±128.8, 74.9±65.9 and 5.9±11.8, p<0.001) and percent malapposed struts (11.0% [8.8-14.1], 3.5% [0.71-6.7] and 0.12% [0.0-0.33], p<0.001) were significantly reduced with longer durations of balloon inflation. Tissue protrusion (27 (71.1), 24 (63.2) and 7 (18.4), p<0.001) decreased and lumen eccentricity index (0.84±0.13, 0.85±0.13 and 0.92±0.06, p<0.001) increased significantly with longer durations.

Conclusions: 60 s (10+20+30 s) of balloon inflation had beneficial effects on stent expansion and apposition.

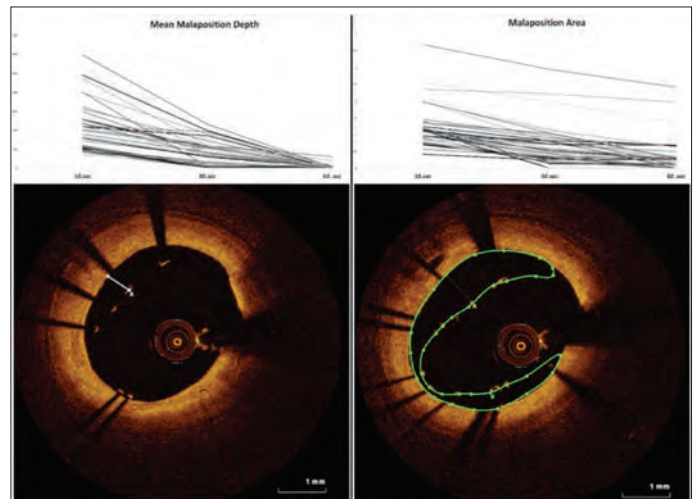


Figure 1.

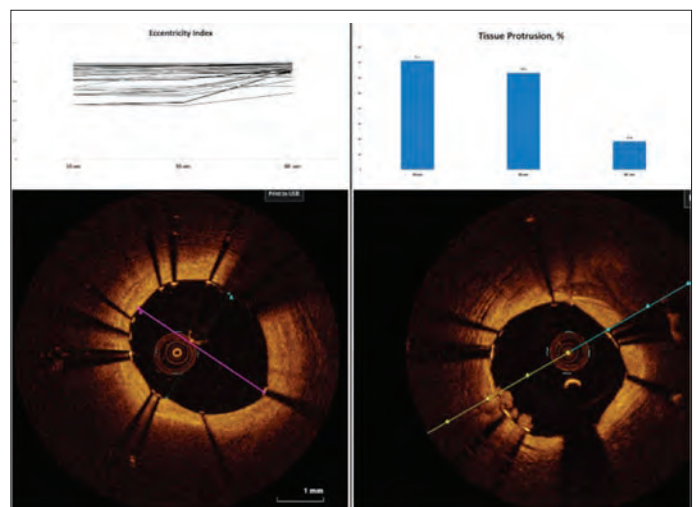


Figure 2.

Interventional cardiology / Coronary

OP-050

Electrocardiographic R wave peak independently predict in-hospital mortality in patients with acute ST elevated myocardial infarction

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Background and Aim: R wave peak time (RWPT) (is also called as ventricular activation time or intrinsicoid deflection) is defined as the time from the onset of QRS complex to the peak of R wave. It represents the propagation of the electrical activity from the endocardium to the epicardium. Studies have shown that RWPT is prolonged in the presence of myocardial ischemia due to conduction delay in Purkinje fibers and myocytes. However, to our knowledge, no study investigated the association between RWPT and in-hospital mortality in patients with acute ST elevated myocardial infarction (STEMI). The aim of this study is to evaluate the relationship between RWPT and in-hospital mortality in acute STEMI patients.

Methods: A total of 212 acute STEMI patients undergoing coronary angiography and treated with percutaneous coronary intervention were included in this retrospective study. 12-lead surface ECG was taken from all patients. PR interval, QRS duration, QT interval, QTc interval and RWPT were recorded from this 12-lead ECG. The longest measurement in ECG lead was used for each variable.

Results: In-hospital mortality was developed in 14 (6.6%) patients in our study population. Baseline clinical and characteristic variables were similar between in-hospital mortality (+) and in-hospital mortality (-) groups. Similarly, PR interval (p=0.801), QRS duration (p=0.786), QT (p=0.419) and QTc interval (p=0.710) were similar between two groups. However, RWPT was significantly longer in patients who developed in-hospital mortality (+) group compared to in-hospital mortality (-) group (p=0.028) (Table 1). Multivariate logistic regression analysis showed that RWPT was the only independent predictor of in-hospital mortality (Odds Ratio: 1.04, 95% Confidence Interval: 1.00-1.08, p=0.046).

Conclusions: We found that baseline ECG can provide important information for prognosis of acute STEMI patients. This is the first study that demonstrating the association between RWPT and increased in-hospital mortality in acute STEMI patients. According to our findings, it can be concluded that longer RWPT is independently associated with in-hospital mortality in patients with acute STEMI. Further studies with larger participant are required to better explain the prognostic role of RWPT in STEMI.

Table 1. Comparison of electrocardiographic variables between two groups

Variables	In-hospital mortality (+)	In-hospital mortality (-)	P
PR interval	160.7 ± 51.0	163.58 ± 36.617	0.801
QRS duration	87.8 ± 20.8	89.28 ± 19.799	0.786
QT interval	353.3 ± 41.8	363.57 ± 46.191	0.419
QTc interval	411.2 ± 37.4	406.80 ± 43.153	0.710
R wave peak time	40.8 ± 15.9	33.7 ± 11.3	0.028

Interventional cardiology / Coronary

OP-051

Contrast induced nephropathy is associated with residual syntax score in without ST segment elevation and with prevent ejection fraction patients

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Background and Aim: Although residual residual syntax (rSS) score after incomplete coronary revascularization in non-ST-elevation myocardial infarction (NSTEMI) patients is associated with many cardiovascular mortality and morbidity, it is unclear in relation to contrast induced nephropathy (CIN). Our aim in this study was to investigate the relationship between CIN and rSS developed in patients with preserved fractionated (EF) NSTEMI.

Methods: We included 252 patients (mean age 61.9±12.3) with non-CIN and 54 patients with CIN (mean age 68.4±13.1) who had undergone coronary intervention with a diagnosis of emergency. We previously excluded patients with coronary artery disease history, patients with chronic renal and hepatic disease, patients with oral feeding impairment who did not undergo coronary angiography. We recorded routine blood tests and echocardiograms of patients. From the angiography laboratory, we recorded the amount of contrast medium used. Finally, we calculated the syntax score before and after percutaneous coronary intervention (www.syntax.com).

Results: When the demographic data were compared, the mean age of the CIN patient group was significantly higher (p=0.001) and the other findings were similar (Table 1). When the laboratory findings were compared, haemoglobin and lymphocytes were higher in the non-CIN group (p<0.001 for each), creatinine (p<0.001), uric acid (p=0.001), Hs-CRP (p=0.015), Hs-TnT (p=0.03) and CK-MB (p=0.013) were higher in CIN group, other findings were similar (Table 2). The EF was significantly higher in the non-CIN group (p=0.034) and the rSS was significantly higher in the CIN group (p=0.002) and the other findings were similar (Table 3) when comparing echocardiographic and angiographic data. In the binomial logistic regression analysis, age (p=0.031, OR: 1.031, 95% CI: 1.003-1.059) and rSS (p=0.04, OR: 1.036, 95% CI: 1.002-1.071) were independent predictor for CIN. In ROC analyses, when cut-off value of rSS was taken 3.5, CIN was determined with 79% sensitivity and 65% specificity (Figure 1).

Conclusions: Residual syntax score is an important predictor of CIN development. Care should be taken in the development of CIN in incomplete coronary revascularizations.

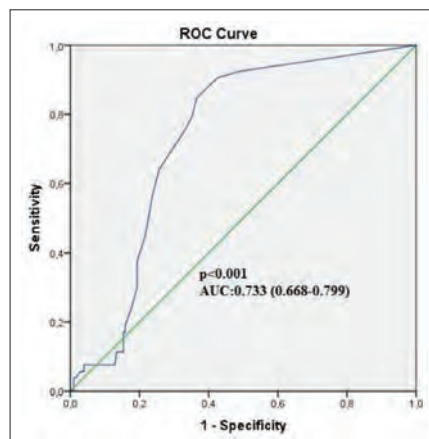


Figure 1. ROC analyse to determine predictive value of residual syntax score for contrast induced nephropathy.

Table 1. Comparison of Patients demographic findings

	CIN (-) n=252	CIN (+) N=54	p
Age (years)	61.9 ± 12.3	68.4 ± 13.1	0.001
Male gender, n, %	139 (55.2)	25 (46.3)	0.236
SBP (mmHg)	137.1 ± 7.0	137.1 ± 6.7	0.982
DBP (mmHg)	73.6 ± 11.3	73.7 ± 7.9	0.954
BMI (kg/m ²)	26.2 ± 3.1	26.4 ± 2.3	0.746
Smoking, n (%)	111 (44.0)	20 (37.0)	0.345
DM, n (%)	126 (52.1)	30 (55.6)	0.642
HT, n (%)	140 (55.6)	24 (44.4)	0.137
HPL, n (%)	128 (50.8)	23 (42.6)	0.274

CIN: Contrast induced nephropathy, DM: Diabetes mellitus, DBP: Diastolic blood pressure, HT: Hypertension, HPL: Hyperlipidemia, SBP: Systolic blood pressure.

Table 2. Comparison of laboratory findings

	Patients n=254	Patients n=52	p
WBC (uL)	10.2 ± 10.1	10.4 ± 4.0	0.866
Hb (mg/dl)	13.6 ± 2.3	11.8 ± 1.8	<0.001
Lymphocyte (%)	24.3 ± 9.6	18.8 ± 8.5	<0.001
Neutrophil (%)	66.9 ± 29.2	71.9 ± 10.6	0.251
BUN (mg/dL)	32.2 ± 13.2	53.8 ± 21.4	<0.001
Cr (mg/dL)	0.8 ± 0.5	1.4 ± 0.8	<0.001
Uric acid (mg/dl)	5.3 ± 1.6	6.4 ± 2.1	0.001
Na (mmol/L)	138.5 ± 8.8	133.4 ± 24.4	0.140
K (mmol/L)	4.5 ± 3.0	4.6 ± 0.6	0.804
Total cholesterole (mg/dL)	184.5 ± 48.2	168.9 ± 54.5	0.061
LDL (mg/dL)	110.6 ± 44.2	105.3 ± 42.5	0.439
HDL (mg/dL)	41.5 ± 10.9	41.3 ± 16.4	0.908
Triglyceride (mg/dL)	174.6 ± 132.8	142.9 ± 85.0	0.127
Hs-CRP (mg/L)	2.5 ± 2.7	4.1 ± 4.1	0.015
Hs-TnT (ng/ml) (IQR)*	36.5 (247)	91.1 (1098)	0.03
CK-MB (ng/ml) (IQR)*	2.7 (5.9)	5.0 (23.2)	0.014

Table 3. Comparison of patients echocardiographic and angiographic findings

	CIN (-) n=252	CIN (+) n=54	P
Ejection fraction	55.4 ± 5.4	53.6 ± 5.4	0.034
SS (n)	12.3 ± 9.7	13.4 ± 7.3	0.340
rSS (n)	5.2 ± 8.6	9.2 ± 7.7	0.002
Contrast volume (ml)	146.1 ± 12.3	146.8 ± 8.5	0.736

SS: Syntax Score, rSS: Residual syntax score.

Cardiac imaging / Echocardiography

OP-052

Evaluation of the predictive capabilities of left ventricular wall motion pattern and other strain characteristics in left ventricular noncompaction patients' first degree relatives for early diagnosis of cardiomyopathy

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Background and Aim: None of the diagnostic indices present in noncompaction cardiomyopathy involve left ventricular dysfunction. Left ventricular function and hemodynamics can be normal in these patients. For demonstrating regional deformation Tissue Doppler imaging studies, two and three dimensional 'speckle-tracking' and 'strain' echocardiography studies are available. The aim of this study is to evaluate 'Left ventricular noncompaction' patients and first degree relatives of these patients with respect to ventricular motion pattern and other strain characteristics, and demonstrate the predictive capabilities of these features for early diagnosis of cardiomyopathy.

Methods: This cross-sectional, case-control study included 32 noncompaction cardiomyopathy patients, 30 first-degree relatives and 31 healthy volunteers. All patients were evaluated for baseline echocardiography, strain measurements, and ventricular wall motion pattern. Student t test, chi square test and ce fisher tests were used for statistical analysis. In all comparisons, p <0.05 was considered significant.

Results: There was no difference between the case and control groups in terms of age, weight and body surface area. There was a statistically significant decrease in EF, FS, E / E', GLS, GLSr, GCS, GCSr, GRS and GRSr values from the control group to the patient relatives and the patient group, respectively (patient relatives and control EF p=0.023, for all other groups p<0.01). There was a significant correlation between EF and strain values in all groups (p<0.001). When the rotation values were examined, the decrease was observed from the control group to the patient relatives and the patient group, respectively, but significant difference was observed between the patient and the other groups and not between the patient relatives and control groups. In the case group, 'Rigid Body Rotation (RBR)' movement pattern was observed in 17 patients and the present pattern was observed in 9 patients in the patient relatives group. EF, GLS, GLSr and basal rotation values were significantly lower and GRS and GRSr values were higher in the group with RBR after comparison of patients with and without RBR pattern.

Conclusions: It may be considered that the evaluation of the strain characteristics of all three study groups, observations of significant differences and findings in terms of RBR motion pattern contribute to reveal the genotype - phenotype relation of disease and to suggest that these features are predictive of early diagnosis of cardiomyopathy.

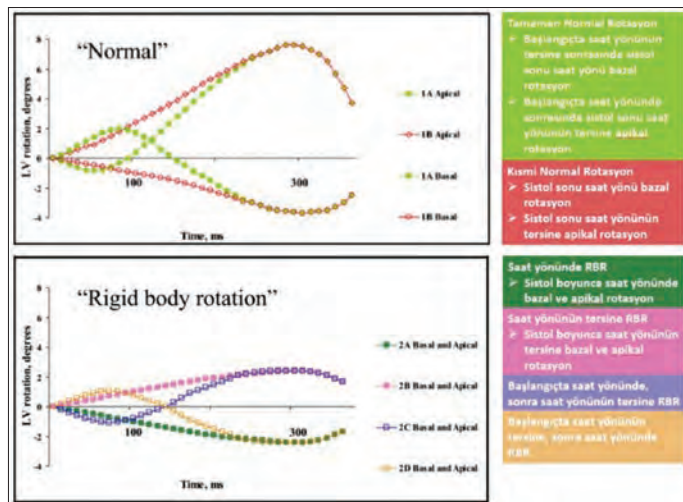


Figure 1. Ventricular motion patterns. The graph shows normal and RBR compatible wall motion patterns.

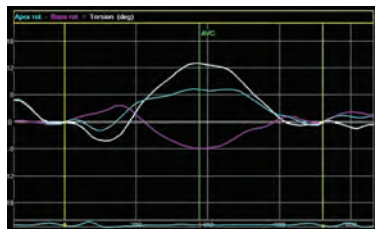


Figure 2. Normal Motion - Rotation Pattern. Graph created by device of normal motion pattern.

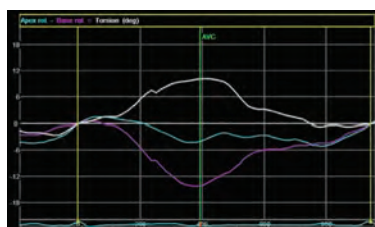


Figure 3. 'Rigid Body Rotation (RBR)' Movement Pattern. Graph created by device of RBR motion pattern.

Table 1. Evaluation of study groups in terms of demographic data, vital findings and ECG characteristics

Değişkenler	OLGU (N=32)		HASTA YAKINI (N=30)		KONTROL GRUBU (N=31)		p *	p **	p ***
	Ort ± Std	Min-Max	Ort ± Std	Min-Max	Ort ± Std	Min-Max			
Yaş (yıl)	36,22±13,52	18-65	40,43±15,06	18-65	36,10±9,36	21-56	0,055	0,440	0,092
Boy (cm)	168,21±8,17	159-182	165,27±6,69	152-180	174,77±9,99	152-195	0,075	0,001	0,001
Kilo (kg)	69,22±15,51	46-115	74,27±14,08	42-103	70,90±11,86	41-94	0,092	0,314	0,159
VKİ (kg/m ²)	24,23±4,60	16,91-34,71	27,24±5,99	15,81-41,78	23,13±2,91	16,97-31,43	0,015	0,019	0,002
VYA (m ²)	1,79±0,22	1,38-2,41	1,84±0,18	1,37-2,15	1,85±0,19	1,31-2,24	0,178	0,128	0,391
Sistol. Basıncı (mmHg)	118,16±22,49	73-168	120,23±14,99	90-146	116,84±11,93	84-138	0,032	0,041	0,019
Diast. Basıncı (mmHg)	70,13±14,30	50-107	70,10±11,34	43-90	71,16±8,55	54-85	0,007	0,363	0,341
Kalp Hızı (dk)	73,25±14,31	53-121	73,50±11,46	49-91	74,65±10,32	59-108	0,076	0,124	0,128
EKG: QRS Sıklığı (min)	103,06±28,09	70-200	90,47±11,76	72-124	86,06±10,19	70-114	0,137	0,258	0,279

p * Ölçü Grubu ile Hasta Yakını Grubu'nun karşılaştırılması
p ** Ölçü Grubu ile Kontrol Grubu'nun karşılaştırılması
p *** Hasta Yakını Grubu ile Kontrol Grubu'nun karşılaştırılması

Table 2. Evaluation of study groups in terms of heart rhythm, functional class and application complaints

Değişkenler		OLGU (N=32)		HASTA YAKINI (N=30)		KONTROL GRUBU (N=31)		p *	p **	p ***
		n	%	n	%	n	%			
Ritim	NSR	30	93,8	30	100,0	31	100,0	0,214	0,332	-
	AF	0	0	0	0,0	0	0,0			
	PACE Ritmi	2	6,3	0	0,0	0	0,0			
Dal Bloğu	LBBB	6	18,7	0	0,0	0	0,0	0,035	0,001	-
NHYA Sınıfı	1	16	50,0	30	100,0	31	100,0			
	2	13	40,6	0	0,0	0	0,0			
	3	3	9,4	0	0,0	0	0,0			
Çarpıntı	Var	9	28,1	5	16,7	4	12,9	0,325	0,458	0,632
	Yok	23	71,9	25	83,3	27	87,1			
Göğüs Ağrısı	Var	6	18,8	2	6,7	2	6,5	0,256	0,625	0,289
	Yok	26	81,3	28	93,3	29	93,5			
Nefes Darlığı	Var	11	34,4	3	10,0	0	0,0	0,374	0,449	-
	Yok	21	65,6	27	90,0	31	100,0			
Senkop	Var	1	3,1	0	0,0	0	0,0	0,822	0,632	-
	Yok	31	96,9	30	100,0	31	100,0			
Ortopne	Var	4	12,5	0	0,0	0	0,0	0,121	0,121	-
	Yok	28	87,5	30	100,0	31	100,0			
Asemptomatik		12	37,5	20	66,6	25	80			

p * Ölçü Grubu ile Hasta Yakını Grubu'nun karşılaştırılması
p ** Ölçü Grubu ile Kontrol Grubu'nun karşılaştırılması
p *** Hasta Yakını Grubu ile Kontrol Grubu'nun karşılaştırılması

Table 3. Basic Echocardiographic Data (Basic measurements)

Değişkenler	OLGU (N=32)		HASTA YAKINI (N=30)		KONTROL GRUBU (N=31)		p *	p **	p ***
	Ort ± Std	Ort ± Std	Ort ± Std	Ort ± Std	Ort ± Std	Ort ± Std			
Aort Kapak Açıklığı (mm)	1,86±0,27	1,85±0,23	1,99±0,21	0,442	0,018	0,007			
Aort Kök (mm)	2,13±0,29	2,08±0,23	2,19±0,20	0,725	0,235	0,028			
İkinci Yataca (mm)	2,73±0,27	2,76±0,34	2,57±0,33	0,330	0,012	0,374			
Aortdan Aorta (mm)	3,10±0,34	3,07±0,41	3,02±0,31	0,311	0,170	0,263			
Sol Atriyum(LA) (mm)	3,88±0,72	3,29±0,45	3,20±0,33	0,001	0,019	0,001			
SVSDC (mm)	5,53±0,95	4,61±0,61	4,53±0,42	0,001	0,168	0,308			
SVSSC (mm)	4,37±1,15	3,05±0,60	2,88±0,43	0,001	0,001	0,106			
İVS (mm)	1,02±0,15	0,87±0,12	0,87±0,09	0,002	0,001	0,035			
Şeş Ventrikülü(RV) (mm)	2,48±0,47	2,25±0,27	2,38±0,21	0,021	0,001	0,022			
FK (%)	0,22±0,10	0,34±0,07	0,36±0,08	0,002	0,001	0,001			
E	82,50±28,14	87,47±26,1	83,52±16,08	0,236	0,001	0,708			
E/A	64,81±23,20	68,67±19,01	67,03±16,16	0,138	0,001	0,247			
E/A	1,45±0,77	1,36±0,50	1,52±0,34	0,295	0,430	0,689			
Dassalaksyon Zamanı (ms)	150,47±24,13	152,61±18,14	148,51±16,55	0,347	0,063	0,411			
Sistol S (cm/s)	7,13±2,66	9,17±1,76	9,48±1,69	0,003	0,313	0,474			
Sistol E (cm/s)	8,78±4,86	10,47±3,68	13,94±2,76	0,064	0,258	0,098			
Sistol A (cm/s)	6,66±2,43	9,91±2,84	9,77±1,98	0,001	0,002	0,003			
Lateral S (cm/s)	7,69±2,71	10,31±2,62	10,68±2,71	0,058	0,011	0,066			
Lateral E (cm/s)	10,03±5,30	13,73±4,37	17,10±4,17	0,002	0,232	0,365			
Lateral A (cm/s)	6,78±3,36	10,47±2,82	10,03±2,70	0,001	0,101	0,270			
E/E'	11,04±7,41	7,51±1,83	5,54±1,26	0,001	0,001	0,001			

p * Ölçü Grubu ile Hasta Yakını Grubu'nun karşılaştırılması
p ** Ölçü Grubu ile Kontrol Grubu'nun karşılaştırılması
p *** Hasta Yakını Grubu ile Kontrol Grubu'nun karşılaştırılması

Table 4. Basic Echocardiographic Data [LVEF and Right Ventricular EF (RVEF) values

Değişkenler	OLGU (N=32)		HASTA YAKINI (N=30)		KONTROL GRUBU (N=31)		p *	p **	p ***
	Ort ± Std	Ort ± Std	Ort ± Std	Ort ± Std	Ort ± Std	Ort ± Std			
TAPSE (mm)	21,4±5,1	25,3±4,6	26,1±2,8	0,012	0,002	0,185			
RVSM (cm/s)	11,8±2,7	13,9±2,7	14,3±1,9	0,001	0,001	0,297			
TRV (m/s)	2,4±0,5	1,9±0,3	1,5±0,2	0,004	0,001	0,236			
SPAP (mmHg)	29,6±13,4	22,3±3,9	16,7±2,1	0,001	0,001	0,037			
SVDSH (ml)	160,7±54,0	119,4±27,6	109,4±24,4	0,001	0,001	0,044			
SVSSH (ml)	94,9±49,7	47,4±21,7	38,8±11,8	0,001	0,001	0,011			
EF(Simpson) (%)	43,2±16,0	60,9±7,2	64,2±5,7	0,001	0,001	0,023			

p * Ölçü Grubu ile Hasta Yakını Grubu'nun karşılaştırılması
p ** Ölçü Grubu ile Kontrol Grubu'nun karşılaştırılması
p *** Hasta Yakını Grubu ile Kontrol Grubu'nun karşılaştırılması

Table 5. Strain echocardiography data

Değişkenler	OLGU (N=32)		HASTA YAKINI (N=30)		KONTROL GRUBU (N=31)		p*	p**	p***
	Ort ± Std	Min-Max	Ort ± Std	Min-Max	Ort ± Std	Min-Max			
GLS (%) (-)	11,6±5,5	3,7-23,7	18,6±3,6	10,2-22,4	20,7±19,7	14,6-24,4	<0,001	<0,001	0,002
GLSr (s ⁻¹) (-)	0,8±0,3	0,3-1,4	1,1±0,1	0,6-1,2	1,16±1,3	0,9-1,2	<0,001	<0,001	0,009
GCS (%) (-)	11,9±4,5	5,7-20,7	17,1±3,1	8,9-22,7	18,2±15,5	13,1-23,8	<0,001	<0,001	0,003
GCSr (s ⁻¹) (-)	1,1±0,2	0,6-1,3	1,2±0,1	0,8-1,3	1,3±1,2	1,2-1,4	<0,001	<0,001	0,001
GRS (%) (+)	23,8±12,1	6,9-57,2	37,1±6,2	22,7-46,8	39,8±39,2	26,9-51,4	<0,001	<0,001	0,002
GRSr (s ⁻¹) (+)	1,4±0,2	0,8-1,8	1,7±0,1	1,4-1,8	1,8±1,8	1,5-2,1	<0,001	<0,001	0,001

p* : Olgu Grubu ile Hasta Yakini Grubu/Grup Karşılaştırılması
p** : Olgu Grubu ile Kontrol Grubu/Grup Karşılaştırılması
p*** : Hasta Yakini Grubu ile Kontrol Grubu/Grup Karşılaştırılması

Table 6. Evaluation of working groups in terms of rotation, twist and RBR motion pattern

Değişkenler		OLGU (N=32)		HASTA YAKINI (N=30)		KONTROL GRUBU (N=31)		p*	p**	p***
		n	%	n	%	n	%			
Apikal	EKSI	11	34,4	5	16,7	0	0,0	0,003	0,056	0,049
	ARTI	21	65,6	25	83,3	31	100,0			
Bazal	EKSI	23	71,9	26	86,7	31	100,0	0,002	0,077	0,147
	ARTI	9	28,1	4	13,3	0	0,0			
Net Twist Derecesi	EKSI	6	18,8	2	6,7	-	-	0,004	-	-
	ARTI	26	81,3	28	93,3	-	-			
RBR	YOK	15	46,9	21	70,0	31	100,0	0,001	0,001	0,007
	VAR	17	53,1	9	30,0	0	0,0			
RBR Yönü (ikiisi de Aynı Yönde Olanlar)	EKSI	10	-	5	-	-	-	0,003	-	-
	ARTI	7	-	4	-	-	-			

p* : Olgu Grubu ile Hasta Yakini Grubu/Grup Karşılaştırılması
p** : Olgu Grubu ile Kontrol Grubu/Grup Karşılaştırılması
p*** : Hasta Yakini Grubu ile Kontrol Grubu/Grup Karşılaştırılması

Değişkenler		OLGU (N=32)		HASTA YAKINI (N=30)		KONTROL GRUBU (N=31)		p*	p**	p***
		Ort ± Std	Ort ± Std	Ort ± Std	Ort ± Std					
Apikal Rotasyon Derecesi		1,19±3,4	4,18±3,53	5,92±2,58		0,002	0,001	0,078		
Bazal Rotasyon Derecesi		(-),2,72±4,33	(-),4,37±3,23	(-),4,53±1,35		0,354	0,241	0,510		
Net Twist Derecesi		3,59±4,79	7,15±5,95	10,45±2,41		0,011	0,001	-		

p* : Olgu Grubu ile Hasta Yakini Grubu/Grup Karşılaştırılması
p** : Olgu Grubu ile Kontrol Grubu/Grup Karşılaştırılması
p*** : Hasta Yakini Grubu ile Kontrol Grubu/Grup Karşılaştırılması

Table 7. Comparison of groups with and without RBR movement patterns

Değişkenler	OLGU (N=32)		p	HASTA YAKINI (N=30)		p
	RBR (YOK)	RBR (VAR)		RBR (YOK)	RBR (VAR)	
Yaş (yıl)	33,93±11,59	38,23±15,08	0,124	39,29±14,77	43,11±16,28	0,378
Cinsiyet (Kadın)	8 (%25)	4 (%12,5)	0,258	14 (%46,66)	2 (%6,66)	0,474
Vkl (kg/m ²)	24,52±4,99	24,16±4,38	0,031	27,12±6,31	27,85±5,50	0,052
Sist. Basınc (mmHg)	117,0±25,48	119,17±20,23	0,547	121,90±15,52	116,66±13,81	0,748
Diast. Basınc (mmHg)	70,93±15,70	69,41±13,38	0,489	71,24±12,34	67,44±8,63	0,786
Kalp Hızı (/dk)	71,53±13,31	74,76±15,38	0,823	74,50±12,46	78,32±11,54	0,524
SVDSH (ml)	5,52±0,78	5,52±1,09	0,365	4,51±0,54	4,78±0,75	0,564
SVSSH (ml)	4,31±0,94	4,42±1,33	0,227	2,92±0,25	3,33±1,01	0,423
EF(Simpson) (%)	44,80±16,08	41,82±16,28	0,002	62,10±3,86	58,11±11,73	0,033
E/A	1,54±0,85	1,37±0,71	0,059	1,36±0,49	1,36±0,57	0,289
E/E'	11,98±9,09	10,21±5,71	0,118	7,14±1,81	8,34±1,64	0,078
GLS (%) (-)	12,77±6,18	10,61±4,96	0,003	18,82±2,68	16,62±3,51	0,002
GLSr (s ⁻¹) (-)	0,87±0,36	0,79±0,31	0,045	1,12±0,09	1,01±0,23	0,011
GCS (%) (-)	12,39±4,57	11,62±4,59	0,086	17,05±2,38	17,31±4,54	0,215
GCSr (s ⁻¹) (-)	1,12±0,17	1,01±0,24	0,055	1,28±0,03	1,23±0,14	0,215
GRS (%) (+)	22,74±11,58	24,76±12,89	0,036	37,88±5,68	35,25±7,36	0,041
GRSr (s ⁻¹) (+)	1,34±0,25	1,43±0,18	0,016	1,75±0,08	1,66±0,12	0,022
Apikal Rotasyon Derecesi	2,36±3,01	(-),0,18±2,57	0,021	4,01±3,25	4,06±3,28	0,056
Bazal Rotasyon Derecesi	(-),3,33±4,02	(-),0,98±5,03	0,002	(-),4,68±3,41	(-),4,06±3,14	0,003
Twist Açısı	5,69±3,01	1,73±2,13	0,231	4,86±5,06	6,04±4,12	0,063

the subclinical cardiovascular right ventricular dysfunction in asthma patients, area under the curve (AUC) for RVfree was 0.623 (95% CI: 0.537-0.710, p=0.006) with a cut-off value below -24.65, and sensitivity was detected to be 0.663 and specificity to be 0.624. Area under the curve (AUC) for RVGLS 0.629 (95% CI: 0.543-0.715, p=0.004) with a cut-off value below -20.85, and sensitivity was detected to be 0.687 and specificity to be 0.620.

Conclusions: Our study demonstrated that while newly-diagnosed, adult, mild-stage asthma patients have normal parameters in standard echocardiography, they have reduced right ventricular functions by STE.

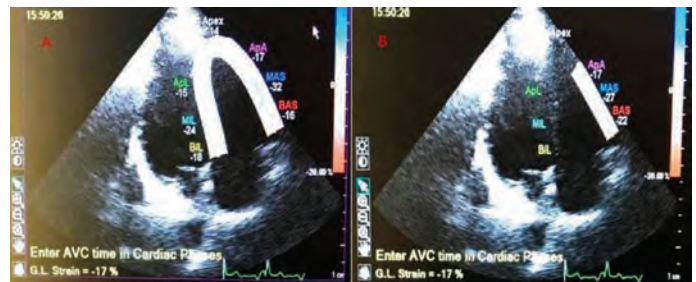


Figure 1. (a) Right ventricular global longitudinal strain analysis (RVGLS), (b) right ventricular free wall strain analysis (RVfree).

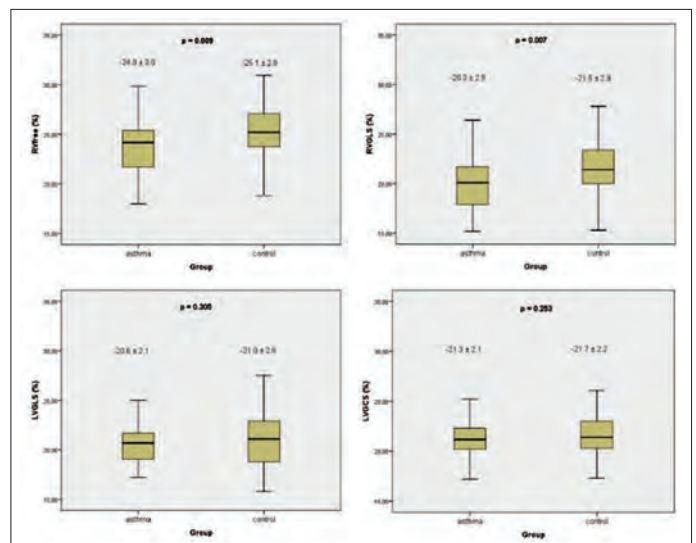


Figure 2. Box plots of the left and right ventricular global longitudinal strain (LVGLS, RVGLS), left ventricular global circumferential strain (LVGCS) and right ventricular free wall longitudinal strain (RVfree) measurements.

Cardiac imaging / Echocardiography

OP-054

Effects of melatonin on the phosphorylation of vascular endothelial growth factor-A (VEGF-A) in coronary vessels of non-diabetic and diabetic rats

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Background and Aim: In the physiological cardiac hypertrophy, an increase and in the pathological cardiac hypertrophy a decrease of the coronary capillaries indicated an association between myocardial hypertrophy and myocardial angiogenesis. Lower nocturnal melatonin secretion was prospectively and independently had a relation with raised risk for developing DM and hypertension (HT), which are very important risk factors for myocardial infarction. However, in a DM setting, the mechanism of the effects of melatonin on cardiac hypertrophy is unknown. It is known that the migration of endothelial cells is an essential step for angiogenesis, and the endothelial cell migration is regulated by phosphorylation of VEGFR2 and VEGF-A. Therefore, the present study aims to investigate the effects on melatonin on antioxidant enzymes and on VEGF-A expression in coronary vessels of the non-diabetic and diabetic heart by quantitative immunohistochemistry using total and phospho-specific antibodies against VEGF-A.

Methods: 40 male Wistar rats were enrolled in the study. Rats were randomly allocated into the following four groups; group 1 was control group, group 2 was DM group (iatrogenically DM-generated group), group 3 was melatonin treatment group (only melatonin given group), and group 4: melatonin treatment plus DM group (melatonin given group after iatrogenically DM generation). Melatonin was injected intraperitoneally at a dose of 50 mg/kg/day for 56 days to group 3 and group 4. We investigated expression and phosphorylation of the VEGF-A in coronary vessels of all groups. Laboratory analysis and transthoracic echocardiography were performed.

Cardiac imaging / Echocardiography

OP-055

Assesment of biventricular function with speckle tracking echocardiography in newly-diagnosed adult-onset asthma

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Background and Aim: There is limited number of studies on the effect of asthma disease on cardiac functions. This study aimed to assess the cardiac functions of newly-diagnosed adult asthma patients using speckle tracking echocardiography (STE) method.

Methods: Our study included 83 newly-diagnosed, non-smoking asthma patients with the age between 18 and 65, and 83 control subjects with comparable age and sex distribution. Two-dimensional, M-mode and tissue Doppler examinations were performed. STE analysis was obtained using QLAB software. Complete blood count and high-sensitive C-reactive protein (hsCRP) levels were measured.

Results: There was no difference between two groups for standard echocardiography and Doppler parameters. While tricuspid annular plane systolic excursion (TAPSE) was observed to be lower in asthmatics (24.9±2.0 vs. 25.5±2.1, p=0.043), and right ventricular myocardial performance index (RV MPI) was observed to be higher (0.36±0.07 vs 0.32±0.06, p<0.001). There was no significant difference between the groups for left ventricular STE parameters. Measurements of right ventricular global longitudinal strain (RVGLS) and right ventricular free wall strain (RVfree) were observed to be lower in the asthma group (-20.3±2.9 vs -21.5±2.9, p=0.007; -24.0±3.0 vs 25.1±2.9, p=0.009, respectively) (Figure 2). In the ROC analysis performed to determine

Results: In comparison to the group 1, DM induced a decrease in p-VEGF-A in coronary vessels of group 2. The lower constitutive phosphorylation of VEGF-A in the group 2 was also increased in coronary vessels after melatonin treatment (p<0.05). Diabetic rats developed myocardial hypertrophy with preserved cardiac function (p<0.05). The serum antioxidant enzymes activities; SOD, CAT, and GPX were significantly lower in DM group (group 2) than in non-DM groups. Injection of melatonin (group 4) significantly decreased the levels of MDA compared with that in the DM group (group 2). On the contrary, antioxidant enzyme activities were found to be significantly increased as a result of melatonin administration in group 3 and group 4 (p<0.05).
Conclusions: Melatonin may be required for the physiological coronary angiogenesis and may regulate phosphorylation of VEGF-A under physiological conditions. The phosphorylation of VEGF-A in wild-type diabetic rats was significantly decreased and with the melatonin treatment, the phosphorylation was increased in diabetic rats. This was indicating a preventive treatment of melatonin in the phosphorylation of VEGF-A in coronary vessels in the case of experimental type I DM. The melatonin-dependent phosphorylation of VEGF-A may be involved in the physiological as well as in the pathological cardiac hypertrophy.

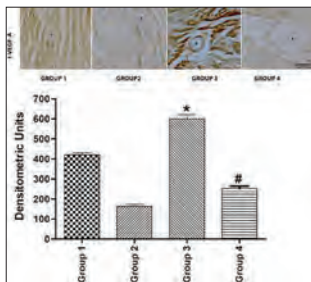


Figure 1. Expression and densitometric analysis of total form of VEGF-A (t-VEGF-A) in coronary vessels of rats. The endogenously t-VEGF-A was detected in the coronary vessels of the control (group 1) (A), DM (group 2) (B), melatonin (group 3) (C) and melatonin+DM (group 4) (D). Data were presented as mean±SD; n=10 for each group. “*” indicates statistical significance vs. control (group 1), “#” indicates statistical significance DM (group 2), p<0.05. Bar: 50 µm.

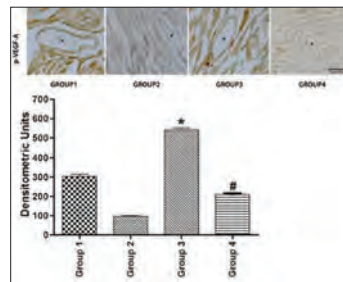


Figure 2. Localization and densitometric analysis of phosphorylated form of VEGF-A (p-VEGF-A) in coronary vessels of rats. The p-VEGFR2 was detected in the coronary vessels of the control (group 1) (A), DM (group 2) (B), melatonin (group 3) (C) and melatonin+DM (group 4) (D). Data were presented as mean±SD; n=10 for each group. “*” indicates statistical significance vs. control (Group 1), “#” indicates statistical significance diabetes mellitus (Group 2), p<0.05. Bar: 50 µm.

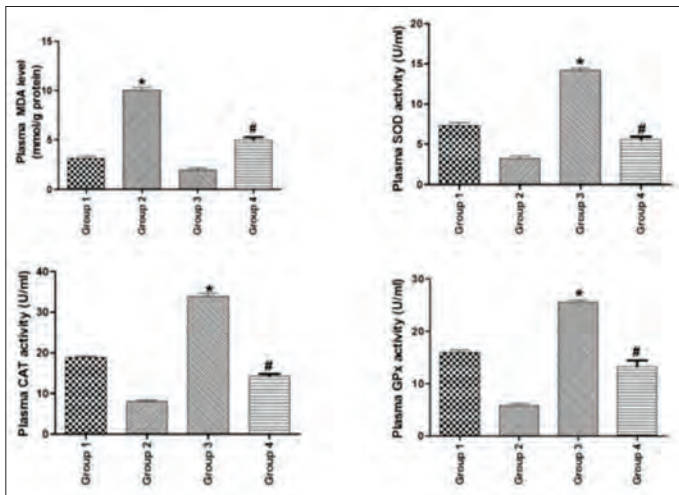


Figure 3. Antioxidant enzymes (GPX, CAT, and SOD) activities and malondialdehyde (MDA) levels Diagram showing GPX, CAT, and SOD enzymes activities and MDA levels in plasma samples of animal groups. “*” indicates significant difference between group 1 and group 2; “#” indicates significant difference between group 3 and group 4. Note that GPX, CAT, and SOD activities and MDA levels were significantly elevated after melatonin administration in both experimental paradigms.

Table 1. Definition of animal groups and applied drugs

Groups	Injection	Dose	Period	n
Group 1 (Control)	Ethanol	10 mg/kg	For 56 days	10
Group 2 (DM)	Streptozotocin	130 mg/kg	First day	10
Group 3 (Melatonin)	Melatonin	50 mg/kg/day	For 56 days	10
Group 4 (Melatonin+DM)	Melatonin+ Streptozotocin	50 mg/kg/day + 130 mg/kg	For 56 days + First day	10

Table 2. Blood glucose levels of group 2 and 4

Groups	3rd day post-injection	14th day Post-injection	28th day Post-injection	42th day Post-injection	56th day Post-injection
Group 2	348±13	389±22	409±27	413±30	413±17*
Group 4	363±14	387±19	413±25	416± 29	418±16*

Blood glucose levels of the DM (group 2) and Melatonin+DM (group 4) groups in day post injection. * Indicates a p value <0.5 compared with 3rd day injection.

Table 3. Systolic and diastolic echocardiographic findings in of the groups

		Group 1 (n=10)	Group 2 (n=10)	Group 3 (n=10)	Group 4 (n=10)
Systolic	Septum (mm)	1.53 ± 0.02	1.91 ± 0.06 *	1.55 ± 0.08	1.44 ± 0.02
	LVESD (mm)	1.7 ± 0.076	1.33 ± 0.18 *	1.73 ± 0.14	1.62 ± 0.11
	LVPW (mm)	1.15 ± 0.30	1.69 ± 0.03 *	1.14 ± 0.07	1.33 ± 0.04
Diastolic	Septum (mm)	0.70± 0.02	1.08 ± 0.02 *	0.74 ± 0.03	0.86 ± 0.04
	LVEDD (mm)	3.77±0.09	3.16±0.14*	3.64±0.14	3.59±0.12
	LVPW (mm)	0.71±0.05	1.18±0.02*	0.80±0.04	0.84±0.05
	LVM (mg)	95±11	127±10*	107±90	106±11
	FS (%)	0.55±0.02	0.56±0.06	0.54±0.02	0.53±0.01
Heart rate (bpm)		482±22	472±22	473±16	480±23

The myocardial dimensions determined by two-dimensional echocardiography included the thickness of the septum and the left ventricular posterior wall (LVPW), left ventricular end- diastolic diameters (LVEDD), and left ventricular end systolic diameters (LVESD). Data are shown as mean±SD. FS: fractional shortening; LVM: left ventricular mass; bpm: beats per min. * Indicates a p value <0.05 compared with control group.

Cardiac imaging / Echocardiography

OP-055

Impact of 3D transesophageal echocardiography in predicting structural abnormalities that underlie ischemic mitral regurgitation

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Background and Aim: Ischemic mitral regurgitation (IMR) is a complex multifactorial disease that involves left ventricular geometry, the mitral annulus, and the valvular/subvalvular apparatus. IMR affects patients' prognosis, doubling mortality following myocardial infarction and heart failure. The purpose of this study was to validate the role of 3D transesophageal echocardiography (3D TEE) in assessing anatomical predictors and mechanism behind IMR.

Methods: A total of 54 IMR patients underwent 2D transthoracic echo (2D TTE) and 3D TEE to define mitral valve structure, LV remodeling and severity of IMR. IMR was evaluated by the PISA method; EROA, regurgitation fraction (RF) and regurgitation volume (RV), measured with both 2D TTE and 3D TEE. Multiple views were obtained with TTE for calculating sphericity index (SI) and apical conicity index (CI). Mitral valve and papillary muscle anatomy evaluated, including anterolateral (AL) or posteromedial (PM) tethering length (TL), coaptation length (CL) and the strain of papillary muscle were measured.

Results: Majority of the patients had previous anterior MI (57%). In the whole group comprises both anterior and non-anterior MI's, functional severity as assessed by RF-3DTEE showed strong correlations with anatomical parameters (SI diastole [r=0.477, p=0.001], SI systole [r=0.395, p=0.006] and PM TL [r=0.366, p=0.011]). EROA-3DTEE (r=0.588, p=0.008) and RV-3DTEE (r=0.602, p=0.006) controlled by EDV-3DTEE showed significant correlations with PM TL in non-anterior MI patients. EROA-TTE and RV-TTE assessed by TTE were not correlated with any of the anatomical parameters after controlling of EDV-2D. 3DTEE derived parameters [EROA: r=0.503, p<0.001], (RV: r=0.631, p<0.001) and (RF: r=0.567, p<0.001)] were much more strongly associated with LA volume compared to TTE-derived parameters. Pulmonary artery pressure (PAP) was strongly associated with 3DTEE-derived EROA (r=0.571, p<0.001), RV (r=0.455, p=0.001) and RF (r=0.501, p<0.001). SI-diastole was also more strongly correlated with 3DTEE-derived parameters of MR [EROA: r=0.378, p=0.005], (RV: r=0.477, p<0.001) and (RF: r=0.477, p=0.001)] than TTE-derived parameters.

Conclusions: 3D TEE is probably the most powerful and convincing imaging method for understanding the complicated multifactorial morphology and for evaluating geometry, dynamics and severity of ischemic mitral valve regurgitation. These study suggest the superiority of 3D-TEE over TTE in predicting structural abnormalities that underlie MR.

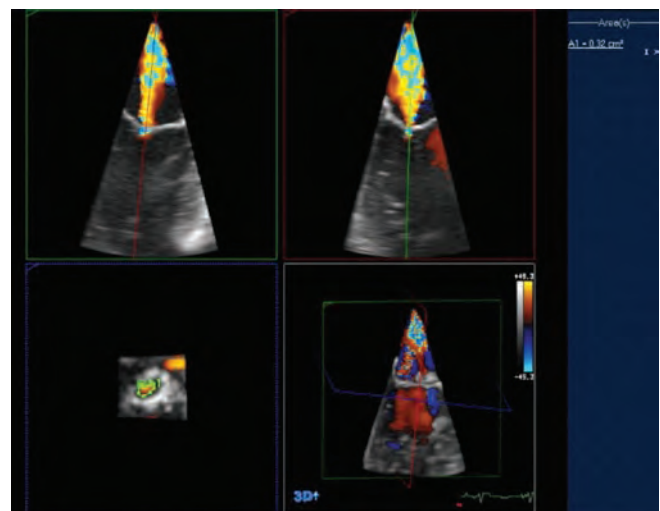


Figure 4. 3D TEE EROA.

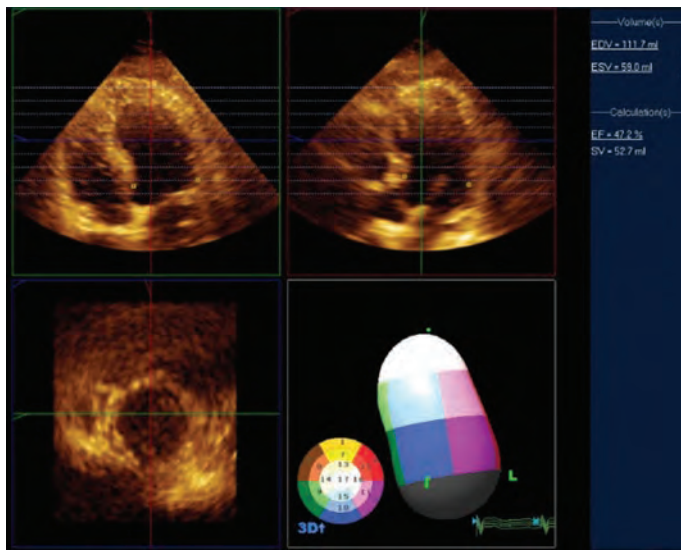


Figure 2. 3D TTE EF.

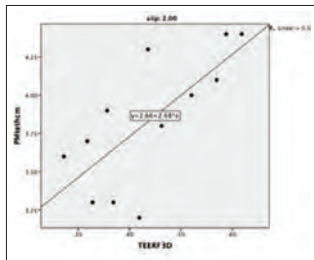


Figure 3. 3DTEE-derived RF and PM tethering.

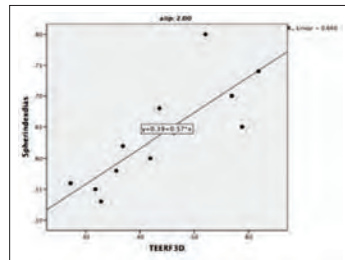


Figure 4. 3DTEE-derived RF and Sphericity index diastole.

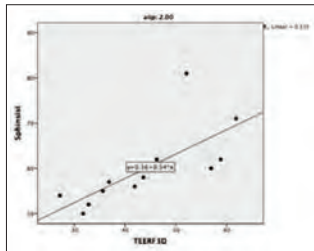


Figure 5. 3DTEE-derived RF and Sphericity index systole.

Table 2. Patient characteristics

Number of the patient (n)	54
Age (mean)	62.72 ± 10.50
Sex (male, %)	38 (70%)
DM (%)	22(40%)
HT (%)	36(66%)
Smoking (%)	27 (50%)
MI localization (Ant.%)	31 (57%)
Time passed from index MI (year, mean)	4.87 ± 4.07
Multivessel disease (%)	32 (59%)

Table 1. Echocardiographic characteristics

Number of the patient (n)	54
EF (mean, SD, %)	0.40 ± 0.11
AI strain	-12.79 ± 5.97
PM strain	-11.90 ± 5.91
Thrombus (%)	9 (16.7)
Aneurysm (%)	14 (25.9)
Tethering area (mean, cm)	1.93 ± 0.42
Anterolateral tethering length (mean, cm)	3.95 ± 0.44
Posteromedial tethering length (mean, cm)	3.93 ± 0.36
Coaptation height (mean, cm)	0.96 ± 0.26
Sphericity index diastole (mean, cm)	0.63 ± 0.08
Sphericity index systole (mean)	0.56 ± 0.08
Conicity index diastole (mean)	0.51 ± 0.09
Conicity index systole (mean)	0.53 ± 0.10

Cardiac imaging / Echocardiography

OP-056

The predictive value of novel echocardiographic method aortic-flow propagation velocity on target organ damage in newly diagnosed hypertensive patients

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Background and Aim: Classical manifestations of hypertensive end-organ damage include atherosclerotic changes in the vasculature, coronary artery disease (CAD), left ventricular hypertrophy (LVH), heart failure, vascular and hemorrhagic stroke, retinopathy, proteinuria, and renal failure. Subclinical target organ damage (TOD) is a crucial intermediary that links risk factors to cardiovascular events and death. Subclinical signs of TOD include LVH, higher carotid intima-media thickness (CIMT) or plaque, renal impairment, including micro-albuminuria and a slight increase in serum creatinine. Recently, aortic-flow propagation velocity (APV) in the descending aorta measured by color M-Mode has been used in the assessment of arterial stiffness besides conventional TOD parameters. There is no study in the literature demonstrating the association of APV with subclinical TOD, and its predictive value on TOD. In the present study, we aimed to investigate the relationship between this novel APV method and LVH, and proteinuria in newly diagnosed hypertension patients. **Methods:** 149 newly diagnosed HT patients were included in the study. Urine samples and blood tests were obtained from each patient for diagnosis of proteinuria. All patients underwent echocardiographic examination. All patients' APV measurements, carotid intima media thicknesses (CIMT), and ankle brachial indexes (ABI) were measured and recorded. APV was measured from suprasternal window, at supine position, color M-mode Doppler recordings were obtained with the cursor parallel to the main flow of direction in the descending aorta. Color Doppler Nyquist limit was adapted to 30-50 cm/s and switched to M-mode with a recorder sweep rate of 200 mm/s, then an M-mode spatiotemporal velocity map with the shape of a flame was displayed (Figure 1). **Results:** The LVH (+) group consisted of 47 patients and the LVH (-) group consisted of 102 patients. The proteinuria (+) group consisted of 32 patients and the proteinuria (-) group consisted of 117 patients. Average CIMT was significantly higher in both proteinuria (+) and LVH (+) groups compared with the (-) groups. ABI and APV were significantly lower in both proteinuria (+) and LVH (+) groups compared with the (-) groups. APV was negatively correlated with LVH, proteinuria and CIMT, and positively correlated with ABI. In the multivariate binary logistic regression analysis, APV was the only significant independent predictor of proteinuria. Additionally, APV and ABI were found to be independent predictors of LVH or/and proteinuria. **Conclusions:** Transthoracic echocardiographic determination of color M-mode propagation velocity of the descending aorta is a simple practical method. It has a powerful predictive value for identifying the patients with higher risk for TOD. It might provide clinical information to identify patients prone to the development of TOD and cardiovascular risk in newly diagnosed HT.

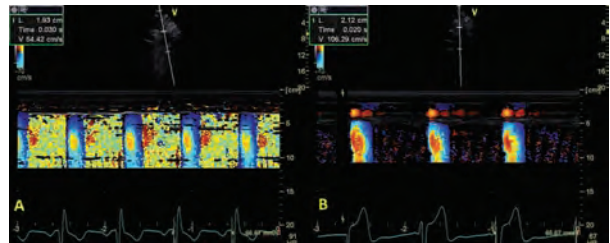


Figure 1. The measurement of aortic-flow propagation velocity of the descending aorta in a patient with target organ damage (a) and in a patient without target organ damage (b).

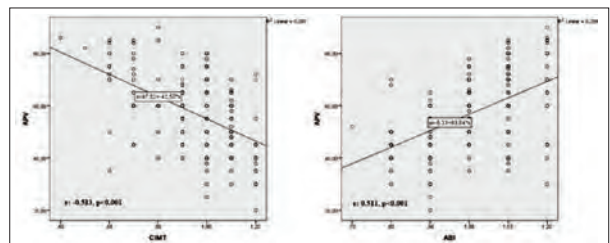


Figure 2. Correlation analysis of APV-CIMT, APV-ABI were performed by Pearson correlation analysis test and presented by using Scatter dot analysis; APV: aortic-flow propagation velocity; CIMT: carotid intima media thickness; ABI: ankle brachial index.

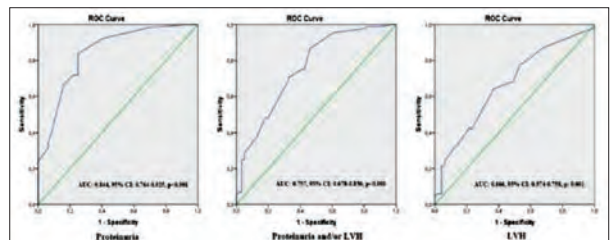


Figure 3. Receiver operating characteristic (ROC) curve analysis to identify target organ damages. The cut-off level of APV was set at 46.5 cm/sec; APV: aortic-flow propagation velocity; LVH: left ventricular hypertrophy; AUC: area under curve; 95% CI: confidence intervals.

Table 1. Demographic, clinical and echocardiographic findings

Variables	Proteinuria+ (n:32)	Proteinuria- (n:117)	p	LVH+ (n:47)	LVH- (n:102)	p
Age (years)	67.8±10.1	59.4±13.7	0.002	64.3±13.8	59.8±13.1	0.540
Male n (%)	16(50)	37(31.6)	0.055	20(42.6)	33(32.4)	0.230
BMI (kg/m ²)	28.2±3.7	29.1±3.6	0.437	28.4±3.2	29.3±4.1	0.454
Heart rate (beats/min)	82.2±15.9	75.2±10.5	0.005	76.3±11.3	76.8±12.6	0.830
SBP (mmHg)	165.0(160.0-173.7)	160.0(150.0-170.0)	0.181	165.0(160.0-170.0)	160.0(150.0-170.0)	0.147
DBP (mmHg)	101.0(95.0-110.0)	100.0(95.0-110.0)	0.983	101.0(100.0-110.0)	100.3(95.0-110.0)	0.052
Hb (g/dL)	13.2±2.5	13.9±1.8	0.730	13.5±2.4	13.8±1.8	0.459
HTC (%)	40.3±7.4	41.3±6.4	0.349	41.1±7.4	40.1±6.2	0.935
PLT (103/μL)	247.2±51.9	242.8±60.3	0.772	252.1±53.3	240.0±63.6	0.384
FBG (mg/dL)	106.0(97.3-149.3)	100.3(91.0-122.5)	0.163	98.0(91.0-129.0)	102.1(91.0-131.2)	0.592
Creatinine (mg/dL)	0.96(0.77-1.5)	0.79(0.68-0.92)	0.005	0.90(0.77-1.48)	0.76(0.68-0.9)	0.001
LDL (mg/dL)	127.1±38.8	120.0±42.9	0.393	119.9±46.5	122.8±39.9	0.569
Triglyceride (mg/dL)	150.5(93.5-197.5)	152.0(109.0-199.5)	0.888	150.0(111.0-182.0)	151.5 (100.5-199.6)	0.896
Na (mEq/L)	139.5±5.8	138.5±4.5	0.307	139.0±5.3	138.6±4.6	0.679
LVEF (%)	54.1±10.5	59.2±7.4	0.002	57.2±7.8	58.6±8.6	0.341
AoD (cm)	2.9±0.2	2.9±0.3	0.384	2.91±0.2	2.83±0.3	0.131
LA (cm)	4.2±0.3	3.82±0.5	0.002	4.2±0.5	3.78±0.5	<0.001
IVS (cm)	1.3±0.2	1.2±0.1	0.002	1.4±0.1	1.1±0.1	<0.001
PwD (cm)	1.2±0.2	1.1±0.1	0.01	1.3±0.1	1.1±0.1	<0.001
LVEDD (cm)	4.9±0.7	4.8±0.4	0.210	4.8±0.5	4.79±0.5	0.997
LVESD (cm)	3.4±0.8	3.3±0.5	0.147	3.13±0.6	3.43±0.6	0.070
LVMI (g/m ²)	135.9±24.1	120.2±24.1	0.001	150.3±21.7	111.2±14.4	<0.001
Overall average CIMT (mm)	1.06(1.0-1.2)	0.9(0.8-1.1)	0.01	1.02(0.9-1.2)	0.9(0.8-1.0)	0.013
ABI	0.93(0.82-1.07)	1.03(1.0-1.1)	0.01	0.95(0.9-1.0)	1.04(1.0-1.1)	0.012
AVP (cm/sec)	42.9±12.4	61.4±13.4	0.002	51.8±13.7	60.1±14.9	<0.001

Abbreviations: BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; Hb: hemoglobin; HTC: hematocrit; PLT: platelet counts; FBG: fasting blood glucose; LDL: low density lipoprotein; LVEF: left ventricular ejection fraction; AoD: aorta diameter; LA: left atrial diameter; IVS: inter-ventricular septum; PwD: posterior wall diameter; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; LVMI: left ventricular mass index; CIMT: carotid intima media thickness; ABI: ankle-brachial index; APV: aortic-flow propagation velocity. Values represent means± standard deviation, median (interquartile range), or number (percentage).

Table 2. Pearson Correlation (r) between APV and other parameters

Variables	r	p
LVH	-0.258	<0.001
Proteinuria	-0.494	<0.001
CIMT	-0.513	<0.001
ABI	0.511	<0.001

Abbreviations: APV: aortic-flow propagation velocity; LVH: left ventricular hypertrophy; CIMT: carotid intima media thickness; ABI: ankle-brachial index.

Table 3. The predictors of LVH and proteinuria in binary logistic regression analysis

Variables	Unadjusted OR (95 % CI)	p	Adjusted OR (95 % CI)	p
LVH				
Age	1.02(0.999-1.057)	0.057	1.00(0.964-1.038)	0.979
DBP	1.04(1.003-1.074)	0.034	1.04(0.993-1.085)	0.102
LA	3.78(1.881-7.596)	<0.001	4.91(1.928-12.525)	0.001
LVESD	0.42(0.219-0.814)	0.010	0.19(0.075-0.481)	<0.001
CIMT	4.39(0.475-9.710)	<0.001	1.87(0.490-7.116)	0.735
ABI	0.01(0.001-0.026)	<0.001	0.02(0.001-2.889)	0.117
APV	0.96(0.938-0.986)	0.002	0.98(0.950-1.028)	0.558
Proteinuria				
Proteinuria	0.26(0.113-0.579)	0.001	0.64(0.196-2.108)	0.465
Age	1.06(1.021-1.099)	0.002	1.01(0.967-1.064)	0.556
Heart rate	1.04(1.012-1.078)	0.007	1.02(0.983-1.071)	0.234
Hb	0.83(0.687-1.018)	0.075	1.07(0.802-1.428)	0.646
Creatinine	1.29(0.997-1.685)	0.053	1.05(0.978-2.239)	0.064
LVEF	0.93(0.895-0.978)	0.003	0.98(0.913-1.048)	0.529
LA	3.90(1.814-8.217)	<0.001	1.02(0.303-3.459)	0.969
LVEDD	2.75(1.137-6.695)	0.025	1.41(0.384-5.107)	0.610
CIMT	3.26(1.598-6.698)	<0.001	0.32(0.030-36.116)	0.640
ABI	0.01(0.001-0.025)	<0.001	0.12(0.103-5.039)	0.489
APV	0.89(0.853-0.931)	<0.001	0.90(0.853-0.955)	<0.001
LVH	0.25(0.113-0.579)	0.001	0.49(0.100-2.466)	0.392
LVH and/or Proteinuria				
Age	1.04(1.016-1.075)	0.002	1.01(0.978-1.046)	0.510
Heart rate	1.03(0.998-1.055)	0.06	1.01(0.979-1.051)	0.423
Creatinine	1.26(0.960-1.676)	0.095	1.17(0.880-1.580)	0.270
LVEF	0.95(0.920-0.999)	0.044	1.01(0.957-1.056)	0.828
LA	3.94(1.966-7.910)	<0.001	2.17(0.962-4.902)	0.062
CIMT	5.34(0.667-42.773)	<0.001	0.33(0.014-8.110)	0.498
ABI	0.01(0.001-0.010)	<0.001	0.01(0.001-0.628)	0.031
APV	0.93(0.905-0.957)	<0.001	0.95(0.918-0.984)	0.004

Abbreviations: LVH: left ventricular hypertrophy; DBP: diastolic blood pressure; FBG: fasting blood glucose; LDL: low density lipoprotein; Hb: hemoglobin; LVEF: left ventricular ejection fraction; AoD: aorta diameter; LA: left atrial diameter; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; CIMT: carotid intima media thickness; ABI: ankle-brachial index; APV: aortic-flow propagation velocity; OR: odds ratio; 95% CI: confidence intervals.

Cardiac imaging / Echocardiography

OP-057

Left ventricular non-compaction (LVNC): A Rare Cardiomyopathy. Evaluation of large series in literature and treatment approaches

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Background and Aim: Left ventricular non-compaction (LVNC) is a rare congenital cardiomyopathy, especially with LV dysfunction, characterized by prominent trabeculations and deep recesses that communicate with the ventricular cavity. LVNC can remain asymptomatic in many years. The most common clinical situations are symptoms heart failure(HF), systemic embolism, and ventricular arrhythmia. The frequency of the diagnosis of LVNC has been increased but still little is known among physicians. The purpose of this article is to improve the current knowledge of this rare cardiomyopathy.

Methods: Over 1000 both articles and case reports were reviewed in the PubMed database by searching the keywords “ventricular noncompaction”, “left ventricular noncompaction”, “ventricular noncompaction cardiomyopathy”, “left ventricular noncompaction cardiomyopathy” and “isolated left ventricular noncompaction”. 812 patients’ data from 14 articles were included.

Results: The age of diagnosis was between 1 week to 93 years. Mean age was 45.5 years. 480 patients(63,5%) were male and 276 patients (36,5%) were female. The most common clinical condition was HF. Ventricular arrhythmias, chest pain, cerebrovascular events,syncope and systemic emboli are other clinical conditions. Thromboembolic events are more common in elderly patients. The most common ECG findings are non-specific ST/T wave abnormalities and LBBB. Also ventricular tachycardias were reported. Preexcitation syndrome was more frequently in young patients. Thromboembolic events are common in patients with atrial fibrillation(AF). 15 patients have undergone heart transplantation. Mortality occurred in 142 patients (22,9%), which was due to: HF in 50 patients; sudden cardiac death in 24 patients; other causes included cerebrovascular events, pulmonary embolism, undefined causes in 53 patients.

Conclusions: LVNC is a rare congenital cardiomyopathy can cause LV dysfunction. Hospitalization for HF was the leading cause of morbidity. LVNC patients with HF should be treated with optimal medical treatment. ICDs with or without CRT should be considered for progressive HF. Heart transplantation is another treatment option for progressive HF. Anticoagulant therapy should be used primarily for AF, LVEF ≤30% or thromboembolism. Predictors of mortality in LVNC patients are HF, ventricular arrhythmias, LV systolic dysfunction and AF. For all first-degree relatives, family screening by echocardiography is recommended. Early diagnosis and treatment may prevent sudden cardiac deaths (with ICDs).

Table 1. Supplement table

Author (Year)	Country	n	Age (mean)	Male (%)	Female (%)	Mean age at diagnosis (years)	Median age at diagnosis (years)	Range (years)	Family history (%)	Genetic testing (%)	Diagnosis criteria (%)	ECG abnormalities (%)	Arrhythmias (%)	Symptoms (%)	Complications (%)	Mortality (%)	Survival (%)	Transcatheter treatment (%)	Heart transplantation (%)
Chen et al (2017)	China	512	45.5	63.5	36.5	1.7-93	3.5	0-93	15	10	75	30	25	30	15	22.9	77.1	25	22.9
Kim et al (2017)	Korea	112	45.5	63.5	36.5	1.7-93	3.5	0-93	15	10	75	30	25	30	15	22.9	77.1	25	22.9
...

Lipid / Preventive cardiology

OP-058

Possible cardiovascular effects of resistance training and whey protein supplementation

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Background and Aim: The usage of whey protein is already widespread; as resistance trainers and even some in-patients with malnutrition use it as dietary supplement. There is reliable evidence against other supplements such as creatinine preperates and anabolic steroids, evidence is lacking against whey protein. The aim of the study is to identify the cardiovascular adverse effects of whey protein usage.

Methods: 48 young male albino Wistar rats are divided into 6 (n=8): Normal (20%) Protein(NP)+Sedentary [NPS]; NP+Resistance training [NPRT]; High (45%) Protein (HP)+Sedentary [HPS]; HP+Resistance Training [HPRT]; Abuse (70% Protein) (A)+Sedentary [AS]; and Abuse+Resistance Training [ART] groups. The rats were fed ad libitum. Training was maintained by resistance protocol in a motorised treadmill. On days 1,30, 60 and 90, blood samples were collected from each animal. The blood analysis included: AST, ALT, LDL, Total Cholesterol, Triglycerides, urea and creatinine. On day 90 Rats were sacrificed. Carotid, femoral, aortic artery samples were taken and fixed with 10% Formalin and stained by H&E.

Results: On the biochemical analysis, AST and ALT levels were elevated in high and abuse protein groups compared to others (p<0.05), with no difference compared to exercise. Urea levels were increased in groups that exercise and used protein supplements. Total cholesterol was elevated in groups with protein supplementation, and elevated cholesterol levels were decreased with exercise slightly. LDL levels were elevated in groups HPRT, ART, HPS, AS, however the increase was lower in groups HPRT and ART (p<0.05). Histo-pathological examination showed no sign of atherosclerosis on H&E stained arteries in any group. However cartilage-like aggregation found within the ascending aorta on rats with high protein intake.

Conclusions: Whey protein use adversely affects the lipid profile and liver function tests. The short term effect on liver functions increases the chances of atherosclerosis. Since the increase on LDL cholesterol and triglyceride levels are shown to be risk factors on atherosclerosis. Although the detrimental effects of whey protein on lipid profile accelerates atherosclerotic plaque formation, this experimental study was too short to these changes on vessels and Wistar rats are not the ideal experimental animal for an atherosclerosis development. There are studies that support the opinion that high protein diets increase risk for heart failure through, and whey protein intake naturally carries the same risk, along with the added adverse effects to liver functions. People use whey protein with little to no worry, since there simply isn't any proven risks to using whey protein. Thus there is no advice regarding to whey protein intake except general high protein intake guidelines. We advise that there should be regulations regarding to the availability of whey protein supplements and require regular screening in individuals who use whey protein supplements and warning labels on whey protein prepares.

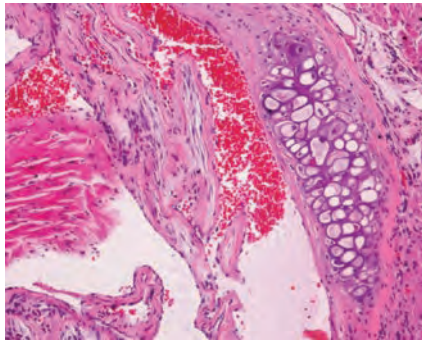


Figure 1. Ascending Aortic Artery Stained with H&E. Cartilage-Like aggregation on ascending aortic artery from HP-RT group.

Table 1. Results day 1 and day 30

Parameter	Day 1							Day 30							
	LDL	HDL	ALT	AST	Chol	Urea	CreaC	LDL	HDL	ALT	AST	Chol	Urea	CreaC	
NPRT	Mean	12.85	36.475	81.875	217.875	57.125	43.625	0.4988	9.0875	34.0875	79.125	121.25	50.375	45.5	0.5038
	Std. Devia	3.07482	4.39002	7.12014	58.56361	3.60307	3.92505	0.01553	2.20612	3.36853	9.35638	14.4593	5.75541	3.38062	0.02334
HPRT	Mean	7.25	27.2375	83.75	188.25	41.125	86.5	0.48	7.4857	32.3143	55.7142	156.1429	46	88.1429	0.4514
	Std. Devia	1.29505	4.05108	5.17683	18.90389	5.56616	7.85584	0.02007	1.22124	5.27176	15.62812	20.20209	6.32456	4.45079	0.01676
ART	Mean	6.925	27.025	54.875	179.25	41	100.875	0.4338	6.7125	31.4625	64.375	144.25	44.75	97.325	0.435
	Std. Devia	2.79527	5.77154	15.8345	31.81581	5.28901	16.59505	0.022	2.39788	6.595	43.21685	46.0892	11.7794	7.41499	0.02
AS	Mean	7.4625	31.05	80.625	180.3	46.375	91.875	0.4263	9.05	42.0375	48.125	148.125	57.125	85	0.4763
	Std. Devia	1.12498	5.12836	18.47887	30.01428	7.463	9.01487	0.01847	2.04101	6.78376	20.08153	25.43584	9.01487	16.55372	0.01847
HPS	Mean	7.6375	30.2375	86.625	181.625	44.625	79.875	0.4088	10.6714	54.1143	48.1429	120.2857	70.5714	85	0.4929
	Std. Devia	2.35561	4.94771	47.55129	36.07832	8.31414	8.35699	0.03091	3.30777	15.15931	11.18368	11.42679	20.0238	6.9282	0.03251
NPS	Mean	9.0125	39.2625	87.25	188.25	56.625	40	0.4588	10.2875	49.7125	63.25	94.625	66.875	49.5	0.5181
	Std. Devia	1.06975	3.23328	6.84801	9.99641	4.17261	1.51180	0.02416	1.78321	4.37279	20.09797	18.49276	3.90989	4.42261	0.02329

Table 2. Results day 60 and day 90

Parameter	Day 60							Day 90							
	LDL	HDL	ALT	AST	Chol	Urea	CreaC	LDL	HDL	ALT	AST	Chol	Urea	CreaC	
NPRT	Mean	9.8875	42.8125	70.875	111.875	58.25	57.375	0.5663	7.6	32.6875	64.875	104.375	46.125	36.875	0.57
	Std. Devia	3.83662	5.54701	22.02231	25.09375	8.24188	7.34725	0.02556	1.06617	3.50242	7.03943	13.97894	4.45413	2.3566	0.02339
HPRT	Mean	6.15	36.425	49.25	143.125	49.875	71.25	0.51	4.4875	26.2625	54.625	124.5	38.25	57.5	0.5325
	Std. Devia	2.82033	5.83261	9.57383	14.13645	7.79403	6.71353	0.02028	5.10306	6.70734	5.55069	15.60409	10.18051	1.92725	0.05497
ART	Mean	3.2875	34.45	77	159.625	46	82.625	0.4888	7.5125	23.625	50	141.125	53.875	84.625	0.5825
	Std. Devia	3.63807	6.47349	35.108	64.00630	11.28843	8.94327	0.01227	3.37827	5.66007	9.10259	22.74352	9.7018	8.51784	0.06475
AS	Mean	6.325	32.275	45.125	140.5	47.5	94.25	0.4588	10.8875	37.2375	45.75	159	53.125	55.5	0.5638
	Std. Devia	1.06601	5.45992	8.45893	20	7.30949	6.31891	0.02597	3.79551	5.79703	12.83689	15.96211	9.49342	7.91021	0.01685
HPS	Mean	8.8125	39.7125	76.25	137.125	57.375	59.75	0.4638	12.475	34.1	46.875	179.75	49.25	43.625	0.5575
	Std. Devia	2.96284	8.81201	16.44859	37.37822	11.09962	12.89241	0.0342	4.94072	9.95074	7.21952	27.05418	14.48801	6.93056	0.0327
NPS	Mean	10.1625	41.1125	74	129	62.75	42.375	0.5061	19	48.4875	59.75	161.25	69	48.25	0.5913
	Std. Devia	2.06463	5.00783	10.18402	12.6004	7.53561	1.99553	0.03204	3.56731	4.04349	45.95883	47.63477	7.11136	8.03119	0.03227

Lipid / Preventive cardiology

OP-059

Evaluation of lipid management strategies in high cardiovascular risk patients: An analysis of EPHEUS study

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Background and Aim: Statin treatment is the cornerstone of hyperlipidemia management. Although the beneficial effect of statins has been shown in variety of studies, underutilization is still a major obstacle. Patient

with atherosclerotic cardiovascular disease (ASCVD) should receive statin treatment unless contraindicated however real world data shows “there is a long way to go”. We aimed to investigate statin treatment strategies in patients with ASCVD namely secondary prevention (SP) group, or high risk for ASCVD namely primary prevention (PP) group in a real world setting.

Methods: Evaluation of Perceptions, Knowledge and Compliance with the Guidelines for Secondary Prevention in Real Life Practice: A survey on the Under-treatment of Hypercholesterolemia (EPHEUS) was planned as a national, multi-center survey to evaluate lipid management in Turkey. Basal demographic characteristics, lipid parameters and risk factors for ASCVD were questioned by a survey. Lipid parameters were collected and target LDL value was calculated according to every single patient's risk level. Both PP and SP groups were evaluated whether they were on statin or not and on target LDL or not. All the information was collected in a single visit.

Results: Of the 1868 patients 1158 (61.9) were on statin treatment. Physicians prescribed statin treatment to SP patients more than PP patients 1036 (69.9%), 122 (31.6%) respectively (p<0.001). Statin therapy did not significantly change the mean LDL value of PP group (136 ± 49, 140 ± 41 respectively (p=0.423). However the mean LDL value was significantly lower in SP patients who were on statin compared to SP patients who were not on statin 101±41, 128±39 respectively (p<0.001). Of the statin prescribed patients 253 (21.8%) were on target LDL while 55 (7.7%) patients who were not on statin were on target LDL (p<0.001). If LDL target value set to 100 mg/dl, 605 (52.2%) of statin treated patients and 151 (21.3%) of non statin treated patients would be on target (p<0.001). Low to intermediate dose statin therapy was the most common preferred medication in both groups 94 (80.3%) vs 647 (63.2%) for PP and SP respectively (p<0.001). However high dose statin therapy was more common in SP patients compared to PP patients 377 (36.8%), 23 (19.7%) respectively (p<0.001) (Table). Statin dose intensity had no effect on reaching the goal of treatment, 154 (21.1%) of low to intermediate and 97 (24.4%) of high dose statin patients were on target LDLs (p=0.194).

Conclusions: We showed statin therapy was underutilized in Turkey in a large multicenter study. Statins were beneficial in reaching target LDL, however most of the patients were not on guideline recommended LDL despite statin therapy. Of note statin dose intensity had no effect on reaching the goal of therapy. Our study clearly showed there is an unmet need for better implementation of guidelines in lipid management.

Table 1. Lipid management in primary and secondary prevention patients

	Overall (n=1868)	PP (n=386)	SP (n=1482)	p
Statin	1158 (61.9)	122 (31.6)	1036 (69.9)	<0.001
Intermediate dose statin	741 (64.9)	94 (80.3)	647 (63.2)	<0.001
High dose statin	400 (35.1)	23 (19.7)	377 (36.8)	<0.001
On target LDL	308 (16.5)	41 (10.6)	267 (18.0)	<0.001
LDL 100	756 (40.5)	74 (19.2)	682 (46.0)	<0.001

Lipid / Preventive cardiology

OP-060

Evaluation of perceptions, knowledge and compliance with the guidelines for secondary prevention in real life practice: A survey on the Under-treatment of Hypercholesterolemia (EPHEUS Study)

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Background and Aim: Few studies have directly assessed the suboptimal management of dyslipidemia in Turkey. This study was conducted to assess patients' understanding and perception of high cholesterol and physicians' knowledge and awareness of issues surrounding cholesterol risk management.

Methods: EPHEUS (Evaluation of Perceptions, Knowledge and Compliance with the Guidelines for Secondary Prevention in Real Life Practice: A survey on the Under-treatment of hypercholesterolemia trial) is an observational and multicenter study (NCT02608645). Consecutive patients admitted to the cardiology clinics who were at least 18 years of age and who are in the secondary prevention group (diabetes mellitus, coronary heart disease, peripheral artery disease, atherosclerotic cerebrovascular disease) and who are in the high-risk primary prevention group (type 2 diabetes mellitus with no prior known coronary heart disease) were included.

Results: A total of 1868 consecutive adult patients (61.83±10.93 years, 1154 [61.8%] men) were enrolled. Of the 1868 patients 386 (20.7%) had no cardiovascular diseases diagnoses (PP group), whereas 1482 (79.3%) had one or more atherosclerotic vascular diseases (SP group). Of the 386 patients who were in high-risk PP group; 286 (74.1%) had very high risk and 100 (25.9%) had high risk for cardiovascular disease. The percentage of patients who were at the LDL-C recommended level was 10.8% for those on primary prevention, 18% for those on secondary prevention (p<0.001). However when physicians were questioned regarding LDL targets, 102 (27%) of PP and 508 (34.5%) of SP patients were identified to be on target (p=0.006). The correlation was moderate between physicians perception and patients on target LDL (r=0.570, p<0.001). Statin treatment was initiated most frequently by cardiologists (1232, 66.2%), followed by internal medicine (444, 23.8%) and family medicine (68, 3.6%) specialists. Total of 602 patients (32.2%) had discontinued statin treatment on at least one occasion in the past. Negative information about statin treatment disseminated by media programs (176, 9.4%) was the most common reason for treatment discontinuation, followed by physician recommendations (118, 6.3%), and problems related to drug access (108, 5.8%). Treatment discontinuation was higher in the PP group (132, 61.7%) compared to SP (471, 36.5%) patients (p<0.001).

Conclusions: EPHEUS study is the largest study in Turkey evaluating the adherence to dyslipidemia guidelines in high risk primary prevention and secondary prevention patients. Perceptions, knowledge, and compliance with the guidelines for primary and secondary prevention in real-life practice have increased, but it is not enough. Patients and physicians need to be more aware of both statin therapy and hyperlipidemia as well as the control of media programs, may increase the therapeutic target.

Lipid / Preventive cardiology

OP-061

Which algorithm should be preferred in Turkish society?
SCORE for high risk country vs AHA risk calculator system

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Background and Aim: Cardiovascular events continue to be the leading cause of mortality and morbidity worldwide. In the prevention of these events, the most effective treatment is lipid lowering drugs. Statins have the strongest evidence among these drugs. Statin therapy is recommended by calculating the risk of cardiovascular events in primary protection. However it is known that algorithms that try to predict the risk of cardiovascular events are inadequate. There is no unique risk algorithm for Turkish society. The SCORE-Turkey algorithm is recalibrated for Turkish society. However, in our analysis we have shown that this is insufficient. We showed that SCORE for high risk countries is better than SCORE-Turkey. However SCORE for high risk countries was not enough. In this study, we compared SCORE for high risk countries and AHA risk calculator system.

Methods: A total of 359 patients who experienced their first episode of acute coronary syndrome were included in the study. Currently, almost all patients with acute coronary syndrome (ACS) are prescribed statins at the time of discharge. These patients are considered to be at very high risk for cardiovascular events. If these patients could come to the outpatient clinic just before they had ACS, what would be the frequency of initiating statin therapy? Furthermore, what would be the cardiovascular risk groups of these patients according to the SCORE algorithms? The risk calculation was determined using age, sex, smoking status, blood pressure, total cholesterol, and high density lipoprotein (HDL-C) levels with HeartScore algorithms and AHA risk calculator. Statin treatment indications were evaluated according to the ESC guidelines (2016) and AHA guideline (2013).

Results: General characteristics of the patients are given in Table 1. There were significant differences in the statin indication ratios which were calculated by HeartScore/High-risk or AHA risk calculator (respectively 39.3%, 69.9%, p<0.001). When patients were divided into 3 groups according to their age (<50 years, 51-60 years, ≥61 years), the AHA risk calculator was more successful in all three groups. Especially in the group <50 years, the difference was very clear (AHA 51.1% HeartScore/High-Risk 6.4% p<0.001) (Table 2). Both calculation systems have a lower success rate in female sex. However, this inadequacy was more pronounced in the SCORE algorithm (p<0.001) (Table 3).

Conclusions: AHA risk calculator is much better than SCORE for high risk country. This difference is more evident, especially at younger ages. Although AHA was better in the younger age group, 49% of the patients were not given an indication. For this reason, it is obvious that an algorithm unique to the Turkish society needs to be developed. Currently AHA risk calculator is the best method for risk calculations for Turkish society.

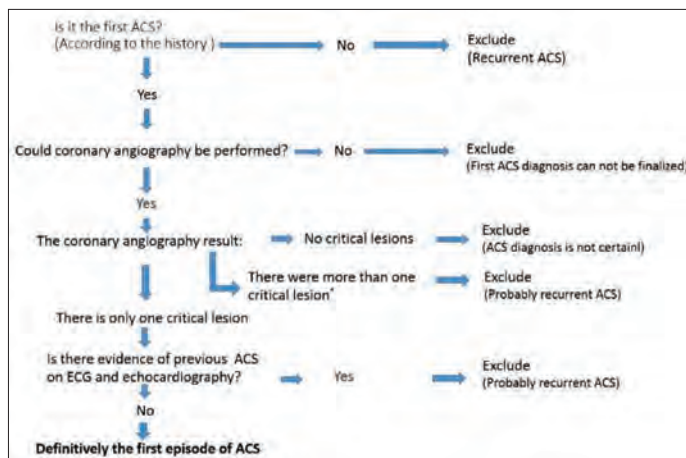


Figure 1. Basic design of the study; how do we determine the first episode of ACS? *More than one critical lesion: If there was another critical lesion (complete or near-complete vascular occlusion) apart from culprit lesion related with current clinical condition, the patient was diagnosed as "suspect recurrent ACS Acute coronary syndrome.

Table 1. General characteristics

	Overall	Statin treatment indication with AHA		P	Statin treatment indication with ESC		P
		YES	NO		YES	NO	
Patients (n)	359	249	110		141	218	<0.001
Male gender (% n)	81.1 (291)	74.2 (216)	25.8 (75)	<0.001	45.0 (131)	55.0 (160)	<0.001
Smoking (% n)	67.7 (243)	71.2 (173)	28.8 (70)	0.273	38.3 (93)	61.7 (150)	0.573
HT (% n)	29.8 (107)	80.4 (88)	19.6 (21)	0.003	42.1 (45)	57.9 (62)	0.482
Age, (years, mean±SD)	54.4 ± 11.9	57.6±11.9	46.9±7.9	<0.001	61.6±11.4	49.7±11.4	<0.001
LDL-C (mg/dl, mean±SD)	138.3 ± 40.2	145.9±41.1	125.6±35.2	<0.001	142.9±30.8	135.3±45.1	0.61
Total-C (mg/dl, mean±SD)	208.2 ± 48.1	214.8±49	193.3±42.3	<0.001	211.3±3	206.2±53.5	0.285
HDL-C (mg/dl, mean±SD)	41.9 ± 9.5	41.5±9.2	43.0±9.9	0.170	41.8±7.9	42.6±10.3	0.855
SBP (mean ±SD)		142.0±29.4	130±20.9	<0.001	139.1±25.3	124.8±22.1	<0.001
DBP (mean ±SD)	81.0 ± 15.7	82.9±15.6	76.7±14.9	0.001	82.4±17.2	80.0±14.5	0.157

AHA: American Heart Association, DBP: Diastolic blood pressure, ESC: European Society of Cardiology, HDL-C: High density lipoprotein cholesterol, HT: Hypertension LDL-C: Low density lipoprotein cholesterol, SBP: Systolic blood pressure.

Table 2. The comparison of the HeartScore/High-risk and AHA algorithms

Statin indications		HeartScore/High-risk		AHA	P
		n (%)	n (%)		
Statin indications	all patients	n (%) 141 (39,3)	251 (69,9)	<0,001	
	< 50 years	n (%) 9 (6,4)	72 (51,1)	<0,001	
	51-60 years	n (%) 63 (51,2)	88 (71,5)	<0,001	
	≥ 61 years	n (%) 69 (72,6)	91 (95,8)	<0,001	
CV risk for 10 years		5,25±4,85	13,2±9,36		

AHA: American heart association, CV: Cardiovascular.

Table 3. The cooperation of statin indications ratio with AHA risk calculator and SCORE in both gender

Gender	AHA	SCORE	P value
Female (% n)	48,5 (33)	14,7 (10)	<0,001
Male (% n)	74,2 (216)	45 (131)	<0,001
P value	<0,001	<0,001	

AHA: American Heart Association, SCORE: Systematic Coronary Risk Evaluation.

Lipid / Preventive cardiology

OP-062

SCORE-Turkey vs SCORE for high risk country. Which Algorithm is More Suitable for Turkish society? A cross-sectional analysis of patients presenting with the first episode of acute coronary syndrome

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Background and Aim: This study was a comparison of the SCORE for high risk countries and SCORE-Turkey risk algorithms, aimed at assessing the adequacy of the statin therapy protocol issued by the European Society of Cardiology (ESC) for primary prevention.

Methods: A total of 323 patients who experienced their first episode of acute coronary syndrome were included in the study. The risk calculation was determined using age, sex, smoking status, blood pressure, total cholesterol, and high density lipoprotein (HDL-C) levels with HeartScore algorithms. Statin treatment indications were evaluated according to the ESC guidelines (2016).

Results: There were significant differences in the statin indication ratios calculated using the SCORE for high-risk country or SCORE-Turkey charts (respectively 38.7%, 35.0%, p=0.02). However, with both charts, recommendations were inadequate to commence statin therapy according to the protocol issued by the ESC guidelines. There was also a significant difference between two systems in the 10 years cardiovascular (CV) risk estimation (5.09±4.67 with SCORE for High-risk country, 4.8±4.67 with SCORE for Turkey, p<0.001). The main difference between two systems was related to HDL-C levels. The HeartScore/High-risk chart was more successful if the patient's HDL-C level was low. As HDL-C levels increased, HeartScore/Turk was better (p<0.001). Our study population consisted of patients in a "very high-risk" group for CV events. While only 10.8% of patients aged 50-59 were in a "very high-risk" group, there were no patients aged <50 in this group, according to the SCORE for High-risk country chart.

Conclusions: The SCORE for high risk countries is better than the SCORE-Turkey chart for Turkish society. It is only appropriate to use the HeartScore/Turk algorithm in a group of patients with high HDL-C levels. The HeartScore risk algorithm is inadequate to describe the "very high-risk" group. Therefore, the ESC recommendation is inadequate to commence statin therapy, especially in young patients. The HeartScore algorithms need to be recalibrated according to risk levels and a new algorithm needs to be developed for Turkish society.

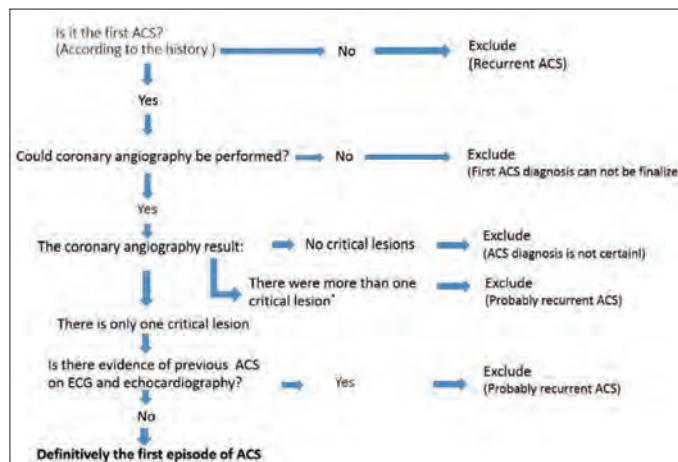


Figure 1. Basic design of the study; how do we determine the first episode of ACS? *More than one critical lesion: If there was another critical lesion (complete or near-complete vascular occlusion) apart from culprit lesion related with current clinical condition, the patient was diagnosed as "suspect recurrent ACS Acute coronary syndrome.

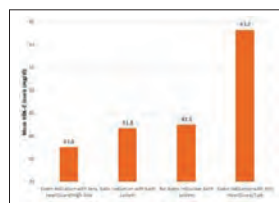


Figure 2. The comparison of the mean HDL-C values in the different groups which were divided according to statin therapy indications status.

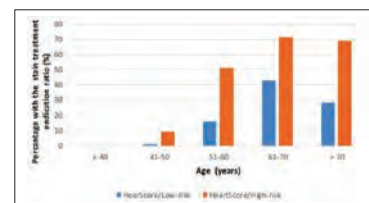


Figure 3. Comparison of ESC Statin Therapy indication ratios according to HeartScore/High-risk/Low-risk algorithms.

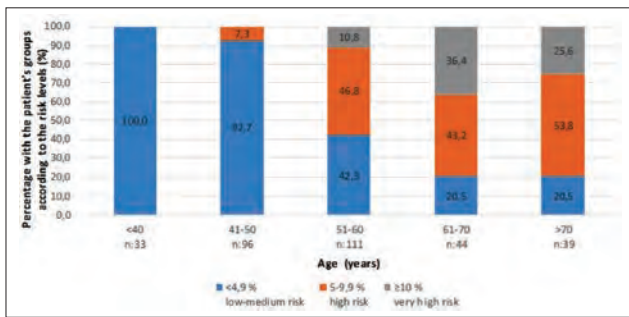


Figure 4. Distribution cardiovascular risk ratios by age groups according to risk levels.

Table 1. Exclusion criteria

- Diabetes Mellitus
- Chronic renal insufficiency
- Thrombocytosis or thrombocytopenia
- History of bleeding disorder
- Predisposition to thrombosis
- Current statin users
- Current aspirin users
- History of previous coronary revascularization
- Age < 18 years
- Inability to obtain inadequate anamnesis
- Inability to perform coronary angiography for any reason

Table 2. Sample of cholesterol recording into the HeartScore system

LDL-C values (mg/dL)			HDL-C values (mg/dL)		
Measured value	Values that can be entered into HeartScore system	Entered values	Measured value	Values that can be entered into HeartScore system	Entered values
220	220	220	42	42	42
221		220	43		42
222		224	44		42
223		224	45		46
224	224	224	46	46	46

Table 3. Intervention strategies as a function of total cardiovascular risk and low-density lipoprotein cholesterol level (2016 ESC/EAS Guidelines for the Management of Dyslipidias)

Total CV risk (SCORE) %	LDL-C levels				
	<70 mg/dL <1.8 mmol/L	70 to <100 mg/dL 1.8 to <2.6 mmol/L	100 to <135 mg/dL 2.6 to <4.0 mmol/L	135 to <190 mg/dL 4.0 to <4.9 mmol/L	≥190 mg/dL ≥4.9 mmol/L
<1	No lipid intervention	No lipid intervention	No lipid intervention	No lipid intervention	Lifestyle intervention, consider drug if uncontrolled
Class ^a /Level ^b	IC	IC	IC	IC	IaA
≥1 to <5	No lipid intervention	No lipid intervention	Lifestyle intervention, consider drug if uncontrolled	Lifestyle intervention, consider drug if uncontrolled	Lifestyle intervention, consider drug if uncontrolled
Class ^a /Level ^b	IC	IC	IaA	IaA	IaA
≥5 to <10, or high-risk	No lipid intervention	Lifestyle intervention, consider drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
Class ^a /Level ^b	IaA	IaA	IaA	IaA	IaA
≥10 or very high-risk	Lifestyle intervention, consider drug	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
Class ^a /Level ^b	IaA	IaA	IaA	IaA	IaA

Table 4. General characteristics

Total patients (n)	323
Male gender (%n)	80,5 (260)
Smoking (%n)	68,1 (220)
HT (% n)	31,6 (102)
Age; (years, mean±SD)	54,4±12,1
LDL-C (mg/dl, mean±SD)	138±40
Total-C (mg/dl, mean±SD)	207,6±46,7
HDL-C (mg/dl, mean±SD)	42,1±9,5
SBP (mean±SD)	133,3±24,3

HDL-C: High density lipoprotein cholesterol, HT: Hypertension
LDL-C: Low density lipoprotein cholesterol, SBP: Systolic blood pressure.

Table 5. The comparison of the HeartScore/High-risk and HeartScore/Turk algorithms

	HeartScore/High-risk	HeartScore/Turk	p
Statin indications n (%)	125 (38.7)	110 (35.0)	0,012
CV risk for 10 years (mean±SD)	5.09±4.67	4.8±4.67	<0,001

Cardiac imaging / Echocardiography

OP-063

Right atrial and ventricular functions assessed by speckle tracking echocardiography in nonmassive acute pulmonary embolism

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Background and Aim: Right ventricular functions are impaired due to acute right ventricular pressure overload in patients with massive and submassive acute pulmonary embolism. Right ventricular size and functions are presumed to be normal in patients with nonmassive pulmonary embolism. The aim of this study was to assess right atrial and ventricular parameters in patients with nonmassive acute pulmonary embolism.

Methods: Twenty-nine patients with acute nonmassive pulmonary embolism and 29 normal subjects were consecutively included. The right atrial, right and left ventricular functions were evaluated by both conventional and speckle-tracking echocardiography.

Results: The echocardiographic parameters of the pulmonary embolism patients and controls are listed in Table 1. Although conventional parameters except tricuspid annular plane systolic excursion (TAPSE) were similar, the right ventricular global longitudinal strain was significantly lower compared to those of controls. The pulmonary embolism patients had also reduced right atrial reservoir and conduit strain, but the differences were not statistically significant. The left ventricular global longitudinal strain of the pulmonary embolism patients was also significantly lower while the left ventricular ejection fraction was similar between patients and controls.

Conclusions: Although right ventricular size and functions are presumed to be normal in patients with nonmassive pulmonary embolism, right atrial and ventricular functions may be impaired and speckle tracking echocardiography may be useful in determining the subclinical right and left ventricular dysfunction in those patients.

Table 1.

	Pulmonary Embolism (n= 29)	Controls (n= 29)	p
Age	58.7 ±14.7	37.1 ± 6.3	<0.001
Male sex (n-%)	20 (69.0%)	14 (48.3%)	0.110
Right ventricle (mm)	34.4 ± 4.7	32.8 ± 5.1	0.242
Right ventricular fractional area change (%)	40.7 ± 9.0	44.1 ± 5.6	0.146
Tricuspid annular S velocity (cm/s)	12.9 ± 1.8	14.5 ± 1.4	0.919
TAPSE (mm)	20.1 ± 4.3	23.8 ± 2.8	0.003
Systolic pulmonary arterial pressure (mmHg)	30.0 ± 13.5	21.4 ± 7.4	0.005
Right ventricular global longitudinal strain (%)	-17.6 ± 3.0	-19.0 ± 2.5	0.048
Right atrial reservoir function (%)	31.7 ± 12.9	36.0 ± 10.3	0.156
Right atrial conduit function (%)	16.6 ± 9.1	16.8 ± 6.0	0.639
Left ventricular ejection fraction (%)	52.6 ± 6.0	54.3 ± 3.2	0.241
Left ventricular global longitudinal strain (%)	-17.3 ± 2.1	-19.8 ± 2.2	<0.001

TAPSE: Tricuspid annular plane systolic excursion.

Cardiac imaging / Echocardiography

OP-064

Right ventricle mechanical dispersion in patients with systemic sclerosis

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Background and Aim: Systemic sclerosis (SSc) is a connective tissue disorder associated with fibrosis of the skin and many internal organs. Most common cardiac involvements are pulmonary hypertension, diastolic dysfunction and pericarditis associated with poor prognosis. Cardiac arrhythmias are common in SSc patients due to myocardial fibrosis development. Mechanical dispersion is an evolving echocardiographic parameter measuring heterogeneity of regional contraction. The aim of this study is to compare the right ventricle mechanical dispersion (RVSD6) in patients with SSc with or without pulmonary hypertension to healthy controls.

Methods: Fifty patients with SSc (mean age:49.6±10.1 years, 45 female) and 25 age and sex matched controls (mean age:44.4±11.7 years, 18 female) were consecutively enrolled into the study. All study subjects underwent conventional two dimensional (2D) echocardiography as well as speckle tracking echocardiography. Right ventricle mechanical dispersion is measured by the standard deviation of time to peak strain of 6 segments (Fig 1). Right ventricle global and free wall strain values were also measured.

Results: In SSc group, ten (10) patients had systolic pulmonary pressure ≥40 mm Hg. Conventional and speckle tracking echocardiographic parameters of the SSc patients and controls are listed in Table 1. The SSc patients had significantly lower global right ventricle strain values (-18.0±4.3% vs -20.4±2.5%, p=0.013) and had significantly higher right ventricle mechanical dispersion compared to controls (57.6±25.4 msec vs 38.3±20.0 msec, p<0.001). Right ventricle mechanical dispersion prolongs with disease severity determined by systolic pulmonary artery pressure (r=0.3, p=0.018). The presence of fragmented QRS in ECG is the independent predictor of RVSD6 (β=0.241 CI: 2.72-27.31, p=0.017).

Conclusions: Our study is first to investigate right ventricle mechanical dispersion in systemic sclerosis patients. RVSD6 increases in the early phases of the disease before the development of pulmonary hypertension in this special population. The clinical significance of this novel echocardiographic parameter needs further studies.

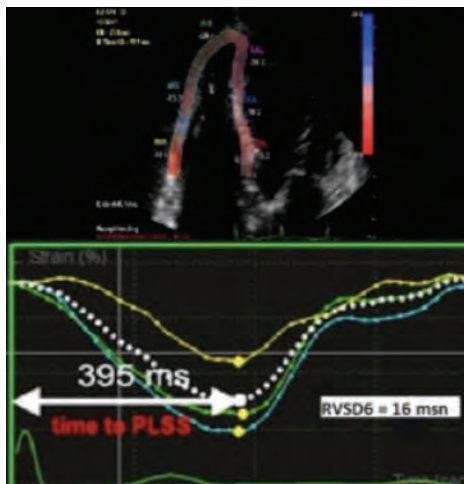


Figure 1. Right ventricle mechanical dispersion measured by 2D speckle tracking echocardiography. PLSS: peak longitudinal systolic strain, RVSD6: right ventricle six segment mechanical dispersion.

Table 1. Comparison of conventional and speckle tracking echocardiography parameters

	SSc (n=50)	Control (n=25)	p
FAC (%)	45±11	53±8	0.001
TAPSE (mm)	22.4±4.1	24.6±3.9	0.032
RVs' (cm/sec)	13.0±2.6	13.1±2.4	0.964
sPAP (mmHg)	35.6±16.1	20.6±6.7	<0.001
RV-GLS (%)	-18.0±4.3	-20.4±2.5	0.013
RV-FWS (%)	-22.3±6.4	-24.6±5.9	0.166
RVSD6 (msec)	57.6±25.4	38.3±20.0	0.001

SSc: systemic sclerosis, FAC: fractional area change TAPSE: Tricuspid annular plane systolic excursion, RVs': Right ventricle annular systolic velocity, sPAP: systolic pulmonary artery pressure, RV-GLS: right ventricle global longitudinal strain RV-FWS: right ventricle free wall strain RVSD6: right ventricle mechanical dispersion.

Cardiac imaging / Echocardiography

OP-065

Is apixaban an effective therapy for the left atrial appendage thrombus and dense spontaneous echo contrast in patients undergoing atrial fibrillation catheter ablation?

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Background and Aim: We have designed a new study for patients who have had thrombus or dense spontaneous echo contrast (SEK) in atrial appendage that planned CA. Because of contraindication of the appendage thrombus and SEK, before ablation we use Apixaban as the first anticoagulant therapy or we change previous anticoagulant with Apixaban and compared to Apixaban using patients with effective dose VKA users. In this study, we aimed to investigate the efficacy of Apixaban for treatment of left atrial appendage thrombus and/or dense SEK in patients who will be performed CA.

Methods: Apixaban Twice Daily Oral Direct Factor Xa Inhibition and Vitamin K are compared, Antagonism for treating the left atrial appendage thrombus or dense SEK in patient that will perform CA was a cross sectional trial which was done on 47 patients from both Apixaban group and 50 patients from VKA group. National regulatory authorities and ethics committees at participating centers approved the protocol. We received patients with nonvalvular atrial fibrillation, as documented on electrocardiography, All patients were provided with a written informed consent. Exclusion criteria were valvular AF, Severe chronic kidney disease (GFR<30). Any contraindications for using VKA and NOAC, a pregnant woman or a breastfeeding woman. Inclusion criteria were >18 age, who had at least one of the CHADs stroke risk factors and atrial fibrillation with a clinical indication for catheter ablation.

Results: A CA planning patient, firstly, assessed with transoesophageal echocardiography (TEE) to exclude any contraindication as left atrial appendage thrombus or dense SEK, when contraindicated TEK or thrombus were founded, these patients were designated to receive fixed-dose Apixaban (at a dose 5 mg twice daily) or adjusted-dose warfarin (target international normalized ratio [INR], 2.0 to 3.0) After 3 months, we performed the TEE again for assessing the benefit of therapy. The study population consisted of 97 patients (25 women; mean age of 67±8 in warfarin group and 24 women; mean age of 65±9 in Apixaban group). When apixaban is compared with Coumadin according to the treatment of both dense sek and thrombus, we obtained a data which showed us that apixaban was more effective (p<0.001).

Conclusions: In this trial, we compared two oral anticoagulant drugs which are in class 1 indicated in non-

valvular AF in current guidelines in order to assess efficacy of Apixaban left atrial appendage thrombus and/or dense SEK treatment which is important for CA. There are no studies for assessing the effectiveness of Apixaban in left atrial appendage thrombus treatment. We designed the first effectiveness study with Apixaban compared with VKA for treating the thrombus and dense SEK and also, we found that Apixaban is more effective than VKA which is a traditional therapy for intracardiac thrombus. We planned a single center, non randomised study. Nonetheless, it should be supported with multi-centered randomized studies.

Cardiac imaging / Echocardiography

OP-066

Melatonin protects against streptozotocin-induced diabetic cardiomyopathy through the mammalian target of rapamycin (mTOR) signaling pathway

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Background and Aim: Several studies demonstrated that the overexpression of mammalian target of rapamycin (mTOR) signaling protein was associated with in cardiac hypertrophy. However, the effect of mTOR on the heart in hyperglycemic condition is still controversial. We aimed to investigate the expression of mTOR and antioxidant enzyme activity in cardiac hypertrophy in streptozotocin-induced diabetic rats and effects of the melatonin on the diabetic cardiomyopathy.

Methods: 40 male Wistar rats were enrolled in the study. Rats were divided into four groups: group 1 (control), group 2 (ethanol vehicle), group 3 (DM), and group 4 (melatonin+DM). Streptozotocin was injected intraperitoneally to the group 3 and 4 to induce experimental type 1 DM. Melatonin was injected intraperitoneally at a dose of 50 mg/kg/day for 56 days to group 4. We investigated expression of mTOR levels in heart muscle fibers of all groups. Laboratory analysis and transthoracic echocardiography were performed.

Results: Melatonin caused an increase in the superoxide dismutase, catalase, and glutathione peroxidase activities, which are reduced due to hyperglycemia. mTOR expression levels were significantly higher in the group 3 (DM) compared with controls, whereas melatonin treatment significantly decreased the levels of mTOR expression in the group 4 (melatonin+DM). Diabetic rats developed myocardial hypertrophy with preserved cardiac function.

Conclusions: In conclusion, our data proposed that the cardio-protective effect of melatonin is caused by inhibiting mTOR expression may prevent cardiac hypertrophy. Demonstrating the underlying mechanism behind mTOR-mediated pathophysiological features in the heart will be important in improving influential therapies against diabetic cardiomyopathy in terms of melatonin.

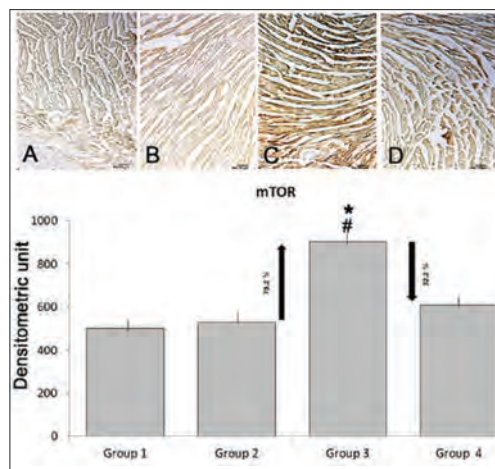


Figure 1. Expression and densitometry analysis of the mammalian target of Rapamycin (mTOR) in heart muscle fibers shown by immunohistochemistry. The staining intensities of the expression of the mTOR levels of the control (group 1) (A), vehicle (group 2) (B), diabetes mellitus (group 3) (C), and melatonin+DM (group 4) (D). Data were presented as mean±standard deviation; n=10 for each group. “***” indicates statistical significance between DM group (group 3) and control (group 1) “#” indicates statistical significance between DM group (group 3) and melatonin+DM group (group 4), p<0.05. Bar: 50 µm.

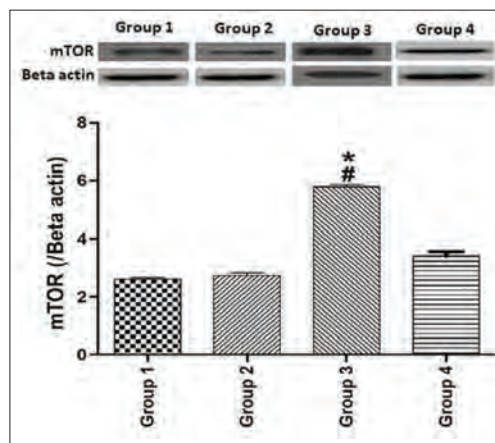


Figure 2. The mammalian target of Rapamycin (mTOR) levels in heart muscle fibers shown by western blot. The mTOR levels of the control (group 1), vehicle (group 2), diabetes mellitus (group 3), and melatonin+DM (group 4). Beta actin was used for internal control. Data were presented as mean±standard deviation; n=10 for each group. “*” indicates statistical significance between DM group (group 3) and control (group 1) “#” indicates statistical significance between DM group (group 3) and melatonin+DM group (group 4), p<0.05. Bar: 50 µm.

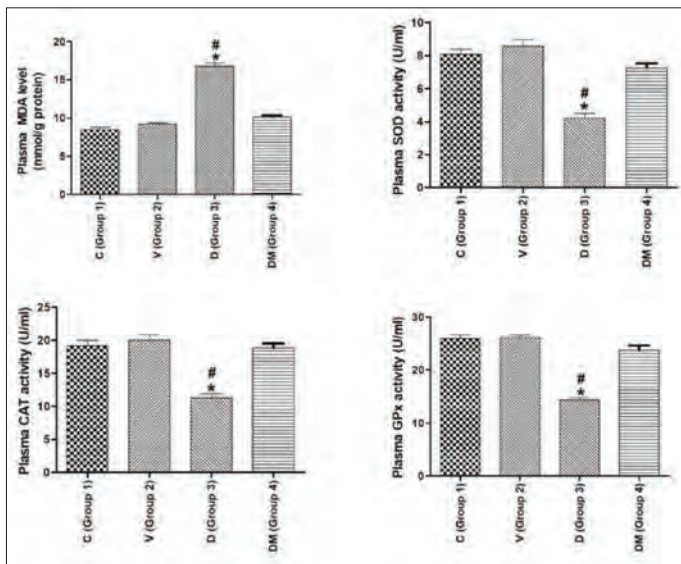


Figure 3. Malondialdehyde (MDA) levels and antioxidant enzymes (GPX, CAT, and SOD) activities between groups. Diagram showing GPX, CAT, and SOD enzymes activities and MDA levels in plasma samples of animal groups. “**” indicates statistical significance between DM group (group 3) and control (group 1) “#” indicates statistical significance between DM group (group 3) and melatonin+DM group (group 4), p<0.05. Bar: 50 μm.

Table 1. Definition of animal groups and applied drugs

Groups	Injected drug	Dose	Period	n
Group 1 (Control)	None	None	None	10
Group 2 (Vehicle)	%10 Ethanol	50 cc/kg/day	First day	10
Group 3 (DM)	Streptozotocin	130 mg/kg	For 56 days	10
Group 4 (Melatonin + DM)	Melatonin + Streptozotocin	50 mg/kg/day + 130 mg/kg	First day + For 56 days	10

Abbreviations: DM: diabetes mellitus.

Table 2. Blood glucose levels of DM (Group 3) and Melatonin+DM (Group 4) groups post streptozotocin injection

	3rd day post injection	14th day post injection	28th day post injection	42th day post injection	56th day post injection
Group 3	354±18	367±14	413±14	417±17	419±12*
Group 4	347±12	363±21	402±17	403±24	404±13*

Abbreviations: DM: diabetes mellitus Data are presented as mean±standard deviation * indicates a p value <0.05 compared with 3rd day injection

Table 3. Systolic and diastolic echocardiographic measurements of the groups

	Variables	Group 1 n = 10	Group 2 n = 10	Group 3 n = 10	Group 4 n = 10
Systolic	Septum (mm)	1.43±0.03	1.45±0.06	1.97±0.03*	1.39±0.13
	LVEDD (mm)	1.5±0.06	1.63±0.3	1.43±0.15*	1.57±0.10
	LVPW (mm)	1.21±0.41	1.24±0.08	1.79±0.02*	1.33±0.04
Diastolic	Septum (mm)	0.68±0.4	0.71±0.01	1.05±0.05*	0.79±0.05
	LVEDD (mm)	2.97±0.3	3.01±0.24	3.96±0.12*	3.02±0.01
	LVPW (mm)	0.82±0.03	0.80±0.04	1.17±0.06*	0.79±0.02
	LVM (mg)	98±10	106±7	137±18*	104±11
	FS (%)	54±1	44±3	49±3	51±7
	Heart rate (bpm/min)	479±12	475±12	476±19	474±19

Abbreviations: LVEDD: left ventricular end systolic diameter; LVPW: left ventricular posterior wall diameter; LVEDD: left ventricular end diastolic diameter; LVM: left ventricular mass; FS: fractional shortening Data are presented as mean±standard deviation * indicates a p value <0.05 compared with control group.

Cardiac imaging / Echocardiography

OP-067

Inferior vena cava assessment can predict contrast induced nephropathy in patients undergoing cardiac catheterization: A single center prospective study

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Background and Aim: Contrast-induced nephropathy (CIN) following cardiac catheterization remains a considerable clinic challenge. Volume status is very important in the development of CIN. It can be assessed noninvasively by measuring inferior vena cava (IVC) diameters. The aim of this study was to assess whether IVC can be used for prediction of CIN in patient undergoing cardiac catheterization.

Methods: A total of 269 patients undergoing cardiac catheterization were prospectively enrolled in this study. IVC inspiratory (IVCi) and expiratory (IVCe) diameters were measured by transthoracic echocardiography. Caval index was calculated as the percentage decrease in the IVC diameter during respiration. An illustration of the measurement of caval index is showed in figure 1. CIN was defined as a ≥0.5 mg/dL and/or a ≥25% increase in serum creatinine within 72 hours post-procedure.

Results: A total of 269 patients were enrolled in this prospective study. CIN developed in 46 (17.1%) patients after cardiac catheterization. There was no significant difference between CIN (-) and CIN (+) groups with regard to baseline clinical characteristics and laboratory parameters. However, patients with CIN was significantly older (64.8±9.8 vs. 58.2±12.4, p<0.001), had a higher frequency of PCI (50% vs. 17.9%, p<0.001) and higher used contrast volume [145 (90-217) vs. 70 (60-100), p<0.001] compared to patients without CIN. In addition baseline echocardiographic parameters, and IVCe and IVCi diameters were similar between CIN (-) and CIN (+) groups (Table 1). However, caval index was significantly higher in CIN (+) group than in CIN (-) group [47% (40-64) vs. 35% (26-50), p<0.001]. A caval index ≥41% was found to predict the development of CIN with a specificity of 69% and sensitivity of 72% (Figure 2). Multivariate logistic regression analysis showed that age (OR: 1.038, 95% CI: 1.000-1.078, p=0.048), contrast volume (OR: 1.012, 95% CI: 1.007-1.017, p<0.001) and caval index (OR: 3.367, 95% CI: 1.574-7.203, p=0.002) were the independent predictor of the CIN development (Table 2).

Conclusions: Bedside evaluation of the IVC with transthoracic echocardiography provides important information for the clinician. Caval index is a simple, noninvasive and easily obtainable parameter from transthoracic echocardiography. It can be used as a beneficial tool to predict CIN development in patients undergoing cardiac catheterization.

Table 1. Comparisons of the echocardiographic features in patients with and without contrast induced nephropathy

Variable	CIN (-) patients (n = 223)	CIN (+) patients (n = 46)	P
LVEDD (mm)	48.4 ± 4.0	50.2 ± 3.9	0.165
LVESD (mm)	33 ± 4.1	35 ± 4.1	0.110
IVST (mm)	11.4 ± 1.4	11 ± 1.4	0.078
PWT (mm)	10.7 ± 1.3	10.4 ± 1.3	0.248
LVEF (%)	58 ± 6.7	56 ± 6.7	0.185
LA diameter	37.2 ± 3.3	38.3 ± 3.6	0.137
E velocity (m/sn)	0.78 ± 0.17	0.76 ± 0.11	0.391
A velocity (m/sn)	0.69 ± 0.17	0.66 ± 0.22	0.246
RA diameter (mm)	32 ± 3.4	33 ± 3.5	0.194
RV diameter (mm)	32 ± 3.9	34 ± 3.7	0.117
IVCe diameter (mm)	16 (13-19)	16.5 (11.7-20)	0.909
IVCi diameter (mm)	10 (6-13)	9.0 (4.7-12)	0.104
Caval index (%)	35 (26-50)	47 (40-64)	<0.001

LVEDD, Left ventricular end-diastolic diameter; LVESD, Left ventricular end-systolic diameter; IVST, Interventricular septum thickness; PWT, Posterior wall thickness; LVEF, Left ventricular ejection fraction; LA, Left atrium; E, Transmural diastolic E-wave velocity; A, Transmural diastolic A-wave velocity; RA, Right atrium; RV, Right ventricle; IVCe, maximum inferior vena cava diameter in expiration; IVCi, minimum inferior vena cava diameter in inspiration; mm, millimeter; m, meter; sn, second

Table 2. Univariate and multivariate logistic regression analysis showing the independent predictors of contrast induced nephropathy

Variable	Univariate			Multivariate		
	OR	95% CI	P	OR	95% CI	P
Age	1.051	1.020-1.083	0.001	1.038	1.000-1.078	0.048
Hypertension	0.734	0.388-1.388	0.342			
Diabetes Mellitus	0.937	0.456-1.928	0.860			
WBC	0.878	0.756-1.021	0.091	0.910	0.769-1.077	0.274
LVEF	0.920	0.810-1.110	0.145			
Contrast volume	1.014	1.009-1.019	<0.001	1.012	1.007-1.017	<0.001
PCI	4.575	2.337-8.955	<0.001	1.644	0.480-5.681	0.431
IVCe	0.987	0.926-1.053	0.699			
IVCi	0.945	0.882-1.012	0.105			
Caval index (≥ 0.41)	5.548	2.752-11.188	<0.001	3.367	1.574-7.203	0.002

OR, odds ratio; CI, confidence interval; BMI, Body mass index; WBC, White blood cell count; LVEF, Left ventricular ejection fraction; PCI, Percutaneous coronary intervention; IVCe, maximum inferior vena cava diameter in expiration; IVCi, minimum inferior vena cava diameter in inspiration.

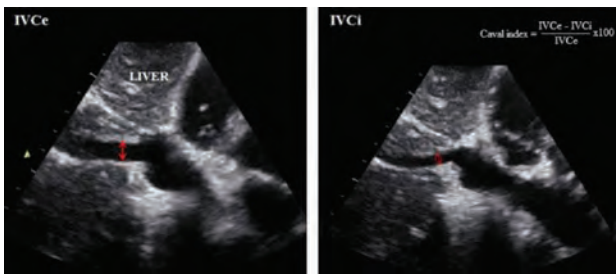


Figure 1. Echocardiographic measurement of inspiratory inferior vena cava (IVCI) and expiratory inferior vena cava (IVCe) diameters, and calculation of caval index.

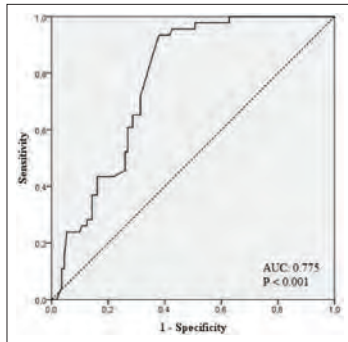


Figure 2. ROC curve analysis of caval index for predicting CIN development.

Cardiac imaging / Echocardiography

OP-068

Effect of glycemc control on right ventricle functions in type 2 diabetes mellitus patients free of clinical cardiovascular disease

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Background and Aim: Diabetes mellitus (DM), is one of the most common chronic endocrine disorders that occurs up to %9 of adults. %90 percent of all diabetics are diagnosed with type 2 DM1. It is well known that, DM closely associated with various vascular diseases and successful glycemc control prevents micro and macrovascular complications. Close relation between DM and left ventricle function is already known. Furthermore, in previous studies it is shown that improved glycemc control is associated with regression of left ventricular mass. Although there are data about the relation between glycemc control and left ventricle function, there is hardly any data about the relation between the right ventricular function. In this study we analysed the relationship between glycemc control and right ventricle function in type 2 DM patients free of clinical cardiovascular diseases.

Methods: In this cross sectional study, patients were randomly selected from the cardiology outpatient clinic. After exclusion criteria 53 patients formed DM group, age and sex matched 51 patients formed control group. All patients' demographic data were recorded. Routine biochemical tests and echocardiographic examinations were performed.

Results: Right atrium (RA) and right ventricle (RV) diameters were significantly higher in DM group (3.36±0.32 vs 3.13±0.34 p=0.015; 2.80±0.32 vs 2.56±0.22 p=0.005 respectively). Myocardial velocity during contraction measured at the lateral tricuspid annulus (RV/IVV) and myocardial acceleration during Isovolumetric contraction measured at the lateral tricuspid annulus (RV/IVA) were significantly lower in the DM group (14.4±3.17 vs 16.04±4.13 p=0.019; 3.25±0.75 vs 3.95±1.25 p=0.015). Furthermore there was an intermediate negative correlation between RV/IVV and HBA1C (r=-0.406; p=0.036). simple linear regression analysis showed that HBA1C level was an independent risk factor for RV/IVV (β=-0.406; p=0.036).

Conclusions: In this study, it is shown that RA, RV diameter were significantly higher, RV/IVV and RV/IVA were significantly lower in diabetes mellitus patients free of clinical cardiovascular disease. Furthermore there was a significant negative correlation between RV/IVV and HBA1C levels and glycemc control, evaluated by the HBA1C level, was an independent risk factor for RV/IVV. This is the first study analysing the effect of glycemc control on right ventricle functions.

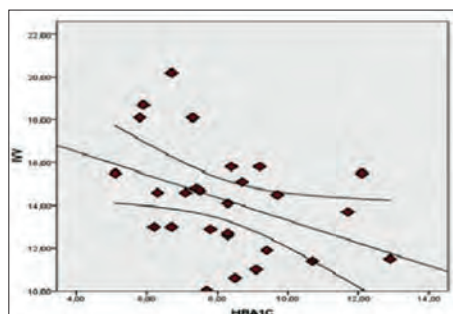


Figure 1. There is significantly negative correlation between RV/IVV and HBA1C (r=-0.406; p=0.036).

Table 1. Demographic and biochemical characteristics of DM and control groups

	DM group	Control group	p value
Age (year)	54,47±9,8	50,64±9,1	0,079
Male/female (number)	35/18	36/15	0,086
Fasting blood glucose (mg/dl)	183,89±8,12	97,33±6,16	<0,0001
LDL (mg/dl)	121,43±38,21	156±47,12	0,008
HDL (mg/dl)	41,14±11,12	51,83±11,17	0,003
Triglyceride (mg/dl)	211,00±78,24	167,83±59,96	0,32
Total cholesterol (mg/dl)	202,43±42,19	242,22±51,79	0,007
Blood urea nitrogen (mg/dl)	30,20±14,01	25,64±10,56	0,24
Creatinine (mg/dl)	0,83±0,20	0,79±0,13	0,51
Sodium (mEq/L)	139,26±2,72	140±1,79	0,22
Potassium (mEq/L)	4,09±0,40	4,15±0,43	0,64

Table 2. Comparison of echocardiographic parameters of DM and control groups

	DM group	Control group	p value
Right ventricle diameter (cm)	2,80±0,32	2,56±0,22	0,005
Right atrium diameter (cm)	3,36±0,32	3,13±0,32	0,015
TAPSE (mm)	33,92±5,83	35,12±5,08	0,44
Acceleration time (sec)	0,043±0,009	0,042±0,012	0,87
IVV (cm/sec)	14,44±3,17	16,04±4,30	0,019
IVA (m/sec ²)	3,25±0,75	3,95±1,24	0,015
Left ventricle EF (%)	61,50±3,13	63,20±2,50	0,068

Heart failure

OP-070

The association of whole blood viscosity with hemodynamic parameters and prognosis in patients with heart failure and reduced ejection fraction: The thicker lives not only better but also longer

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Background and Aim: In heart failure with reduced ejection fraction (HFrEF), determination of prognosis and volume status is critical to improve survival. Although numerous measures are suggested, standardized metrics are still lacking. Whole blood viscosity (WBV), which can be estimated from hematocrit and plasma total protein values, may utilize volum status and prognostic assessment. We aimed to investigate the usefulness of WBV to address volume status using well-known clinical and invasive measures, as well as its prognostic importance in patients with HFrEF who were candidates for heart transplantation.

Methods: We included consecutive 542 patients with HFrEF (mean age 46, 81.4% male) who were performed right heart catheterizations between January 2007 and January 2017. The mean follow-up period of the cohort was 13 months (0-114 months). WBV values for both high (WBV(h)) and low shear rates (WBV(l)) were calculated from the formula of De Simone et al. Composite endpoint (CEP) was defined as having any of these outcomes: mechanical circulatory support, transplantation or all-cause mortality. Two matched sub-cohorts were generated to derive and validate findings.

Results: The patients in the lowest WBV quartile were in advanced NYHA classes, had more congestion and RV dysfunction and also required more inotropes than in the patients in the higher quartile. The incidence of CEP and all-cause mortality were higher in lowest WBV quartile. WBV parameters had a strong negative correlation with plasma volume status, moderate negative correlation with congestion, BNP and right atrium pressure and a weak positive correlation with ejection fraction (EF), mean aortic pressure and cardiac index. In multivariate Cox regression analysis, adjusted with other parameters, WBV parameters were found to be independent predictors of CEP and all-cause mortality. Independent of NYHA classes and cardiac index, every one cP increases of WBV(h) and WBV(l) were associated with 17% and 1% reductions of CEP. WBV parameters demonstrated the best area under the curve in predicting one-month CEP. Lowest quartiles for WBV(h) (<15.64 cP) and WBV(l) (<27.41 cP) was associated with 4.2-fold and 3.3-fold increased odds for one-month CEP and also 3.2-fold and 2.7-fold increased odds for long-term CEP respectively. In the Kaplan-Meier analysis, patients with low WBV quartiles were found to have significantly more CEP (both one-month and long-term) compared with the other groups.

Conclusions: WBV seems to be a novel marker not only quantifying volume status but also determining prognosis in HFrEF. The evidence is that WBV was correlated with clinical and invasive measures of congestion. We also found that the lowest WBV quartiles suffered more CEP than others. In addition, lowest WBV quartiles showed further increased CEP in the short follow-up intervals than in long-term. Being an easily-accessible and costless prognosticator, it may be an emerging tool to individualize HFrEF management.

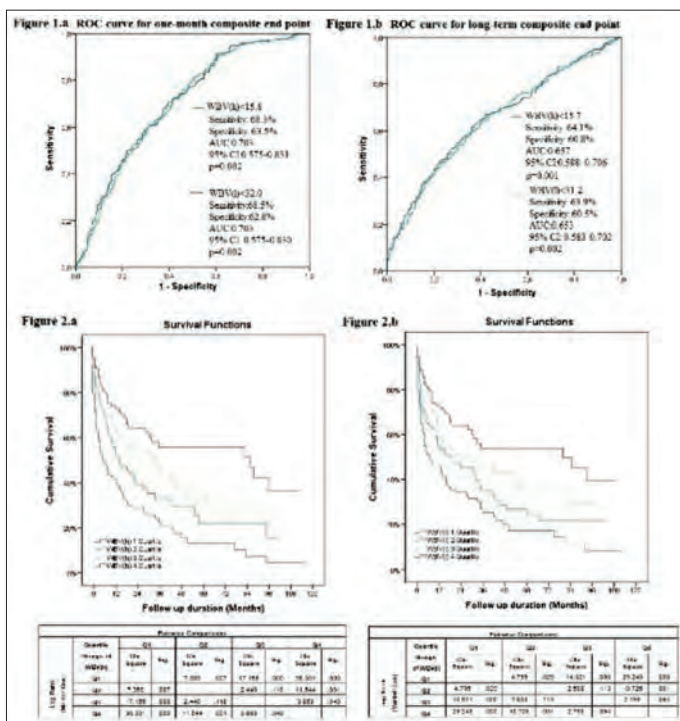


Figure 1 and 2.

Heart failure

OP-071

Effect of ischemic versus nonischemic cardiomyopathy on invasive hemodynamic findings in heart transplant candidates

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Background and Aim: Results of studies evaluating the effect of heart failure (HF) etiology (ischemic or non-ischemic) on invasive hemodynamic measurements are inconsistent because of heterogeneities of studies and relatively small number of patients in those studies. We aimed to investigate how does etiology of HF affect the invasive hemodynamic findings about right ventricular function, including novel parameters such as pulmonary artery pulsatility index (PAPI), pulmonary arterial capacitance (PAC) and pulmonary arterial elastance (PAE).

Methods: A total of 215 patients with end-stage heart failure undergoing evaluation for heart transplantation were stratified into two groups; namely, those with ischemic cardiomyopathy (ICMP) and nonischemic cardiomyopathy (NICMP) and all of the patients underwent right heart catheterization.

Results: One hundred and one patients had ICM and 114 patients had NICM. Patients with left ventricle ejection fraction (LVEF) $\leq 25\%$, NYHA class III or IV and INTERMACS IV and VII levels were included to study. There was no difference in terms of LVEF, duration of HF, NYHA and INTERMACS grades between ICMP and NICMP ($p>0.05$). The patients with ICMP had higher pulmonary artery systolic and mean pressures, pulmonary vascular resistance (PVR) compared to NICMP [59.0 (42.0-73.0) vs. 46.0 (37.0-59.0), $p<0.001$; 35.0 (27.0-46.0) vs. 31.0 (23.0-39.0), $p=0.002$ and 3.5 (1.8-6.6) vs. 2.4 (1.0-4.4), $p=0.007$; respectively]. The cardiac output, cardiac index, stroke volume, right atrial pressure, pulmonary wedge pressure and pulmonary artery diastolic pressure were similar between group [3.3 \pm 0.81 vs. 3.4 \pm 0.96, 1.7 \pm 0.45 vs. 1.8 \pm 0.5, 41.6 \pm 12.9 vs. 41.4 \pm 15.1, 10.0 (5.0-15.0) vs. 11.0 (6.0-16.0), 23.7 \pm 7.8 vs. 22.0 \pm 7.3 and 24.0 (16.0-30.0) vs. 31.0 (23.0-39.0); $p>0.05$ in all]. The right ventricular stroke work index (RVSWI) and PAPI were higher and PAC was lower in patients with ICMP compared to NICM [6.5 (4.8-8.2) vs. 5.4 (3.7-7.7), $p=0.007$; 3.4 (2.2-5.2) vs. 2.5 (1.7-4.0) $p=0.004$ and 1.2 (0.8-1.8) vs. 1.5 (1.0-2.2), $p=0.002$; respectively], the PAE was similar among to groups [1.3 (0.92-2.0) vs. 1.2 (0.81-1.6), $p=0.638$].

Conclusions: The RVSWI and PAPI values measuring right ventricular function were better in patients with ischemic cardiomyopathy compared to nonischemic cardiomyopathy, while afterload of RV were higher in ICMP.

Heart failure

OP-072

Reported bendopnea is associated with worse survival in chronic heart failure

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Background and Aim: Heart failure is a complex syndrome with several dimensions. TREAT-HF network has created a questionnaire recently in order to investigate to several aspects of chronic HF outpatients including

bendopnea, which is defined as a shortness of breath while bending over.

Methods: Turkish Research Team-HF (TREAT-HF) is a network which undertakes multicentric observational studies in HF among HF centers. Herein, data including stable 601 heart failure with reduced ejection fraction (HFREF) patients out of eight HF centers were presented. Patients with acute HF or up-titration or switch of therapy or changing symptoms were not included. In the questionnaire, bendopnea was first defined to the patients and then asked whether they experienced bendopnea in daily life, at least once daily, or not, and 325 reported "yes", 189 reported "sometimes" and 87 reported "no" (total n=601 patients). These patients were followed up for all-cause mortality.

Results: There were 601 (417/184 male/female) responded patients with a mean age of 63.7 \pm 13.2 years and with a mean left ventricular ejection fraction (EF) of 31 \pm 8% and with mean HF history of 3.4 \pm 1.6 years. Coronary artery disease was present in 46.5%, hypertension was present in 34.2%, diabetes mellitus was present in 27.6% and COPD was present in 18.1% of all patients. 45.6% of all patients had NYHA Class III-IV symptoms, 54.4% had NYHA Class II symptoms. Patients were followed up for 9 \pm 4 months up to 17 months by preparation of this abstract. HFREF patients reported to have "yes" bendopnea versus "sometimes" bendopnea and "no" bendopnea had EF of 30 \pm 8% vs. 33 \pm 8% vs 31 \pm 8% respectively (post-hoc, yes vs sometimes $p=0.001$, yes vs no $p=0.909$, sometimes vs no $p=0.072$). Patients who reported "yes" had 30.3%, those who reported to have "sometimes" had 16.7% and those who reported to have "no" had 5.6% all-cause mortality on follow up ($p=0.005$). Kaplan Meier analysis was provided with regard to these three groups, and it yielded significant and graded divergence of survival curves with the best prognosis in "no" group, "sometimes" in between, and the worst in the "yes" group (Fig. 1, $p=0.019$).

Conclusions: Reported bendopnea is associated with worse survival in chronic HFREF patients.

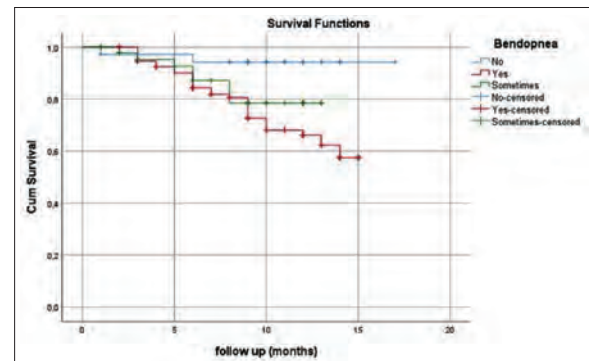


Figure 1. Kaplan Meier Curve for long term mortality.

Heart failure

OP-073

Comparison of heart failure patients with preserved and mid-range ejection fraction: An analysis of APOLLON (A comprehensive, observational registry of heart failure with mid-range and preserved ejection fraction) study

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Background and Aim: To determine and compare the demographic characteristics, clinical profile and management of patients with heart failure with mid-range ejection fraction (HFmrEF) and heart failure with preserved ejection fraction (HFpEF) in a Turkish cohort.

Methods: The APOLLON trial (A comprehensive, Observational registry of heart failure with mid-range and preserved ejection fraction) is an observational and multicenter conducted in Turkey. Consecutive patients admitted to the cardiology clinics who were at least 18 years of age and had HFmrEF or HFpEF were included (NCT03026114).

Results: The study population included 1065 (mean age of 67.1 \pm 10.6 years, 54% women) patients from 12 sites in Turkey. Among participants, 246 (23.1%) had HFmrEF and 819 (76.9%) had HFpEF. The univariate analysis of the associated factors with HFmrEF and HFpEF are shown in Figure 1. Compared to patients with HFpEF; those with HFmrEF were more likely to be male (57.7 vs 42.2%; $p<0.001$), had higher N-terminal pro-B-type natriuretic peptide levels (853 vs 528 pg/ml, $p<0.001$), were more likely to have ECG abnormalities (72.4 vs 53.5%, $p<0.001$) and hospitalization history for heart failure in the last year (28 vs 18.6%, $p=0.002$). HFmrEF patients were more likely to use β -blockers (69.9 vs 55.2%, $p<0.001$), aldosterone receptor antagonists (24 vs 14.7%, $p=0.001$), statins (37 vs 23%, $p<0.001$), loop diuretics (39.8 vs 30.5%, $p=0.006$), and antiaggregant drugs (60.6 vs 40.3%, $p<0.001$) compared to patients with HFpEF. After multivariable adjustment; presence of coronary artery disease (adjusted odds ratio [OR] 3.222, 95% confidence interval [CI] 2.136-4.861, $p<0.001$), mitral regurgitation (OR 3.188, 95% CI 2.021-5.029, $p<0.001$), absence of atrial fibrillation (OR 2.045, 95% CI 1.180-3.545, $p=0.011$), alcohol use (OR 2.287, 95% CI 1.080-4.843, $p=0.031$), having higher NT-proBNP levels (OR 2.681, 95% CI 1.730-4.157, $p<0.001$) and having abnormalities on ECG (OR 2.451, 95% CI 1.659-3.621, $p<0.001$) were associated with HFmrEF (Figure 2). Although ischemic heart disease was the most common cause of heart failure in patients with HFmrEF (63.4%), atrial fibrillation was the most frequent cause in HFpEF (31.3%) (Table 1).

Conclusions: The results of APOLLON study support that the basic characteristics of HFmrEF and the etiology of heart failure in these patients are significantly different from HFpEF. This registry also showed that the patients with HFmrEF and HFpEF were younger but undertreated in Turkey compared to patients in western countries.

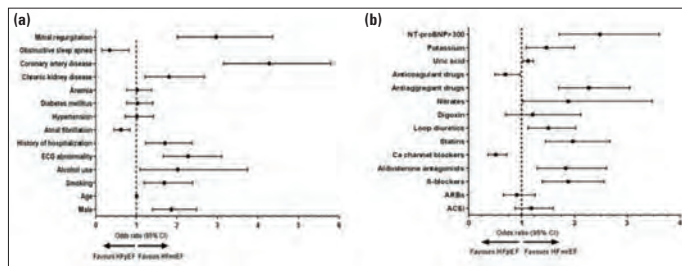


Figure 1. Forest plots of unadjusted odds ratios for heart failure with preserved ejection fraction vs. heart failure with mid-range ejection fraction. Baseline characteristics and comorbid features (a), medications and laboratory data (b). ACEI, angiotensin-converting enzyme inhibitor; ARBs, angiotensin receptor blockers; HFmrEF, heart failure with mid-range ejection fraction; HFpEF, heart failure with preserved left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

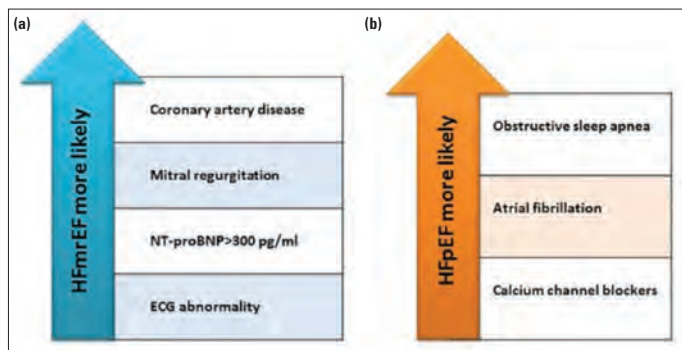


Figure 2. Associated factors with heart failure with mid-range ejection fraction (a) and heart failure with preserved ejection fraction (b). HFmrEF, heart failure with mid-range ejection fraction; HFpEF, heart failure with preserved left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

Table 1. Etiology of heart failure

	Overall (n=1065)	HFmrEF (n=246)	HFpEF (n=819)	p value
Ischemic	330 (31.0)	156 (63.4)	174 (21.2)	<0.001
Atrial fibrillation	295 (27.7)	39 (15.9)	256 (31.3)	<0.001
Hypertension	268 (25.2)	22 (8.9)	246 (30.0)	<0.001
Valve disease	120 (11.3)	15 (6.1)	105 (12.8)	<0.001
Other	52 (4.9)	14 (5.7)	38 (4.6)	<0.001

Data are presented as number (%). HFmrEF, heart failure with mid-range ejection fraction; HFpEF, heart failure with preserved ejection fraction.

Heart valve diseases

OP-074

Transcatheter Aortic Valve Implantation (TAVI) experience of a single center with different types of bioprosthetic valves

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Background and Aim: Since its first description in 2002 by Cribier et al., transcatheter aortic valve implantation (TAVI) has undergone a rapid evolution. Although the most used TAVI devices are CoreValve (CV) (Medtronic Inc., Minneapolis, MN), and Edwards Sapien XT (EST) (Edwards Lifesciences Irvine, CA) in worldwide, novel TAVI devices have also developed. In this study, we aimed to present seven years of experience with TAVI by the same heart team in a single center. To the best of our knowledge this is one of the largest TAVI registries including four types of bioprosthetic valves in our country.

Methods: A total of 194 patients with severe symptomatic aortic stenosis (AS) and undergoing TAVI between June 09, 2012 and May 29, 2018 were investigated in this study. 141 (72.7%) patients treated with MCV, 34 (17.5%) treated with ESV, 3 (1.5%) treated with Direct flow medical (DFM) (Direct Flow Medical, Santa Rosa, CA, USA), and 16 (8.2%) treated with Portico Valve (St.Jude Medical Inc., St. Paul, MI) by the same heart team. **Results:** Mean age of the patients was 78±8 with a frequency of 59.8% female. Procedural success was 100% (194/194). The mean Logistic Euroscore was 31±15; STS score was 7±5; and Euroscore II was 10±8. Mean aortic valve area (AVA) was 0.6±0.2 cm², with aortic valve gradients of 76±22 mm Hg / 46±15 mm Hg. Femoral route was used in all patients; 99 (51%) with surgical cut down and 95 (49%) with percutaneous closure system. Post-TAVI, AVA increased from 0.6±0.2 to 1.8±0.3, and Ejection Fraction was increased from

52±15 to 55±13. Four patients (2%) had cardiac tamponade after TAVI and 3 of them treated with emergent pericardiocentesis. Seven patients (3.6%) required the second valve implantation; 4 with MCV and 3 with Portico. Permanent pacemaker was needed in 25 (12.9%) patients; 18 (12.8%) with MCV, 4 (11.8%) with ESV and 3 (18.8%) with Portico. 18 (9.3%) of the patients had paravalvular leak (≥2 degree) after TAVI. Stroke occurred in only 2 (1%) patient; one of them had a second MCV bioprotosis due to malposition of the first bioprotosis. Ventricular septal defect was developed in only one patient; it was developed after ESV implantation and treated successfully with percutaneous closure. There was no mortality during the procedure, whereas in-hospital mortality rate was 7 (3.6%). Total mortality rate of the total population was 59 (30.4%).

Conclusions: TAVI is a safe treatment modality alternative to surgery with a low complication rate and high procedural success. In our registry, we presented our experience of TAVI with four different bioprosthetic valves. Our results are comparable with the current TAVI registries.

Heart valve diseases

OP-075

Progression of aortic stenosis and associated heavy metal in blood

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Background and Aim: Aortic stenosis (AD) is the most common cardiopulmonary disease, and with the increase in elderly population, the frequency is increasing in developed countries. Degenerative / calcific aortic stenosis is a very common disease in developed societies, 21-26% in people over 65 years old and 48% in people over 85 years old. The rate of progression of aortic stenosis and the factors affecting it are known; it is important that the rate of progress can be slowed down by treatment approaches for these factors, if there are controllable risk factors that affect the rate of progression, and which patients should be assessed on a frequent basis. We considered the effects of heavy metals on the aortic valve progression, taking into consideration the increasing environmental pollution conditions.

Methods: A total of 55 patients (33 patients with mild AD and 22 patients with moderate AD) were included in the study, who had mild to moderate AD with a calcified aortic valve over 50 years of age who were diagnosed with aortic stenosis between 2011 and 2014. Patients' blood heavy metal levels and progression status (mean 3-year follow-up) were studied.

Results: There were two groups of patients showing progressive and non-progressive follow-up. Twenty-six patients in the progressive group and 29 in the non-progressive group were identified. Baseline characteristics were similar between the two groups. A significant difference was found between zinc, chromium, magnesium and selenium in blood heavy metal levels (p<0.001), there was no correlation with copper (p=0.1) (Figure 1). Progression in the group with mild aortic stenosis was seen more (19 vs 7).

Conclusions: Some findings have been obtained in our study that may help to understand the relationship between serum trace element levels and calcific AD severity and progression. Changes in the levels of tracer elements may cause oxidative stress and endothelial dysfunction leading to the development of calcific AD. Trace element levels may be indicative of rapid progression in patients with calcific AD.

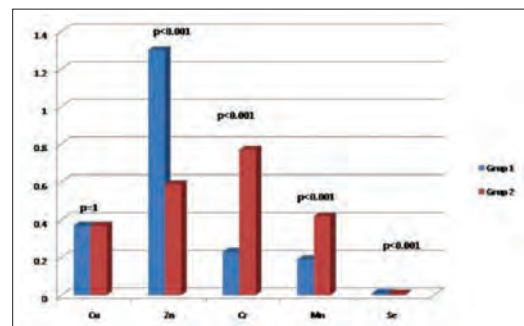


Figure 1. Serum trace element levels of patients.

Heart valve diseases

OP-076

Long-term effects of anticoagulation and over-anticoagulation with warfarin on renal function in patients with mechanical prosthetic valves

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Background and Aim: Warfarin-related nephropathy (WRN) is a complication of warfarin over-anticoagulation that is associated with acute and/or chronic renal dysfunction and increased mortality. Long-term effects of warfarin on renal function is not adequately studied in patients with a mechanical prosthetic valve (MPV). Our aim was to study the time-dependents effects of over-anticoagulation on renal function in patients with a MPV.

Methods: A total of 193 patients that underwent MPV implantation and followed-up in study institution were found eligible for analysis. Time above therapeutic INR range (TATR) was calculated by dividing the number of INR measurements above target in a year to the total number of INR measurements within a year. Patients were divided into quartiles according to average TATR at 60 months.

Results: At 60 months, more patients within the 4th quartile had more than ≥20% reduction in eGFR (25.0%, p=0.04) and chronic kidney disease (CKD) (33.0%, p=0.07) compared to patients within the 1st quartile. High

TATR remained as a significant determinant for reduction in eGFR (OR:7.50, 95% CI:1.55-36.32) and CKD (OR:5.15, 95% CI:1.26-20.62) after adjusting for other variables. Longitudinal analysis has revealed that the change in eGFR was related with the duration of warfarin use ($p<0.001$) and the interaction between the duration of warfarin use and TATR ($p=0.03$). Similar findings were observed in patients without CKD at baseline, but not in those with CKD before index operation.

Conclusions: Anticoagulation over targeted INR values is associated with a steeper decline in eGFR and an increased frequency of CKD in patients with a MPV.

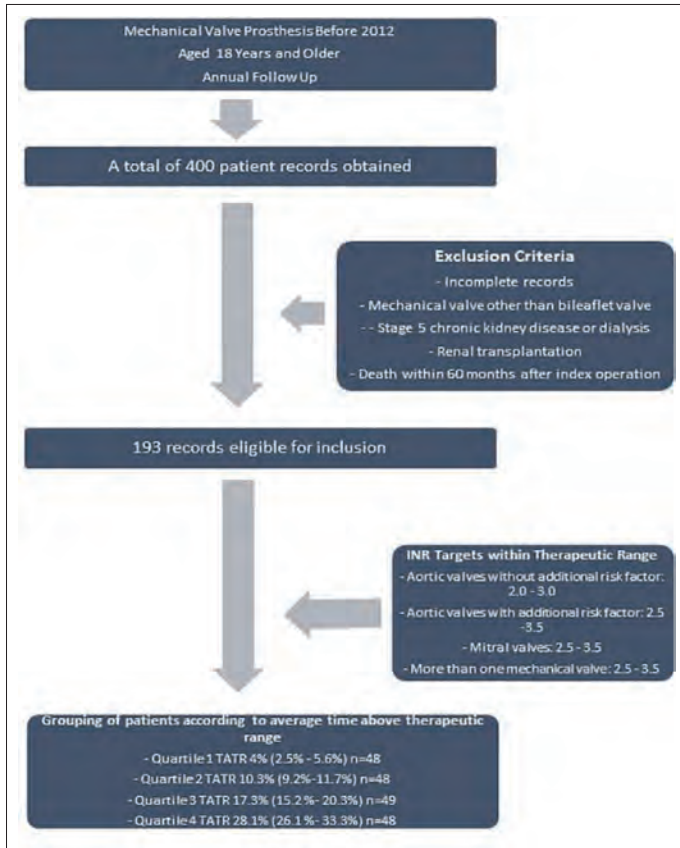


Figure 1. Flow chart.

Table 1. Baseline demographic, clinical and laboratory data for study patients

PARAMETER	1st Quartile (n=48)	2nd Quartile (n=48)	3rd Quartile (n=49)	4th Quartile (n=48)	p
Age	51 (45.0 - 63.5)	56.5 (45.0 - 64.0)	53.0 (44.0 - 62.0)	55.5 (47.0 - 63.0)	0.69
Gender (Female%)	60.4	62.5	71.4	58.3	0.56
Isolated MVR (%)	64.6	68.8	63.3	41.7	0.03
Isolated AVR (%)	22.9	14.6	18.4	43.8	0.004
MVR and AVR (%)	10.4	16.7	18.4	14.6	0.72
Additional TVR (%)	6.3	2.1	4.1	0	0.33
Hypertension (%)	37.5	35.4	40.8	41.7	0.91
Diabetes (%)	20.8	22.9	28.6	31.3	0.62
Hyperlipidemia (%)	35.4	33.3	46.9	33.3	0.45
Smoking (%)	12.5	8.3	10.2	14.6	0.79
Heart Failure (%)	12.5	22.9	12.2	20.8	0.37
LVSD (%)	6.3	18.8	8.2	16.7	0.17
CAD (%)	14.6	16.7	14.3	20.8	0.81
AF (%)	37.5	39.6	40.8	47.9	0.75
CKD (%)	12.5	10.4	18.4	12.5	0.69
TATR (%)	4 (2.5 - 5.6)	10.3 (9.2 - 11.7)	17.3 (15.2 - 20.3)	28.1 (26.1 - 33.3)	<0.001
Basal Creatinine (mg/dl)	0.88 (0.65 - 1.03)	0.80 (0.70 - 0.91)	0.80 (0.70 - 1.00)	0.84 (0.70 - 1.00)	0.58
Basal eGFR (ml/min/1.73 m2)	90.5 (63.8 - 104.5)	90.0 (74.5 - 111.0)	86.0 (69.0 - 102.5)	91.0 (66.3 - 106.8)	0.88

The patients were subgrouped into four quartiles according to 60 months' mean time above therapeutic range values.

Heart valve diseases

OP-077

Role of gut microbiota in pathogenesis of calcific aortic valve stenosis

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Background and Aim: Calcific aortic valve disease (CAVD) is the most prevalent valvular heart disease. Gut microbiota has recently shown to be associated with atherosclerosis and several degenerative diseases. In this study, we aimed to investigate the role of gut microbiota in CAVD pathogenesis.

Methods: We recruited eligible subjects with calcific aortic stenosis (CAS) (n=60), aortic sclerosis (ASC) (n=49) and age and gender-matched control subjects (n=48). Coronary artery, aortic valve and mitral annular calcium score quantifications were performed. Plasma levels of gut microbiota metabolites, namely choline, betaine and trimethylamine N-oxide (TMAO) were measured. Histopathological examinations were performed in aortic valves excised during aortic valve surgery due to either severe CAS or any other non-stenotic disease of the aortic valve.

Results: Prevalence of traditional CV risk factors, co-morbidities or medications did not differ among groups. Patients with moderate-severe CAS had significantly higher plasma levels of choline, a marker of gut microbiota metabolism, when compared to both ASC (p=0.006) and control (p<0.001) groups. Plasma betaine/choline ratio, which is another marker of gut microbiota metabolism, was significantly lower in patients with moderate-severe CAS compared to both ASC (p=0.009) and control (p=0.002) groups. Plasma TMAO levels did not statistically differ between groups (p=0.509). Plasma choline levels were significantly correlated with determinants of CAS severity, namely aortic peak flow velocity (p<0.001) and aortic valve calcium score (p<0.001). Plasma choline levels were also independently associated with aortic peak flow velocity (p=0.009). There was a significant negative correlation between betaine/choline ratio and aortic peak flow velocity (p<0.001). In histopathological examinations, plasma choline levels were significantly elevated in patients who had aortic valves with denser lymphocyte infiltration (p<0.001), more severe tissue remodeling (p=0.002) and calcification (p=0.002), neovascularization (p=0.011), and osteoid metaplasia (p=0.004). These valves also demonstrated significantly higher CD11c+ dendritic cell and plasma cell infiltration, reflecting the chronic inflammatory nature of the diseases.

Conclusions: Our study hereby demonstrates a significant association between gut microbiota metabolites and CAVD presence and severity evaluated with echocardiography, computed tomography and histopathological examinations for the first time in the literature.

Cardiac imaging / Echocardiography

OP-078

Assessment of myocardial dysfunction using speckle tracking echocardiography in patients with ankylosing spondylitis and nonradiographic axial spondyloarthritis

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Background and Aim: Axial spondyloarthritis (axSpA) is a chronic inflammatory disease that mainly affects axial skeleton. Although some differences like sex and objective signs of inflammation were described between ankylosing spondylitis and non-radiographic (nr-) axSpA patients, overall disease burden was found to be similar in these subgroups of axSpA. The association of chronic inflammation with cardiac dysfunction was well documented in many inflammatory rheumatic diseases. However it was not assessed in the subgroups of axSpA. Advanced two-dimensional (2D) speckle tracking echocardiographic analysis is more sensitive and accurate method of early detection of myocardial dysfunction than the conventional 2D transthoracic echocardiography (TTE). Therefore the aim of this study was to evaluate the left ventricular function by using speckle tracking echocardiography in patients with both r- and nr-axSpA.

Methods: This cross-sectional case control study included 72 AS and 38 nr-axSpA patients and 56 age matched healthy control subjects. All patients underwent detailed echocardiographic examination including M-mode, pulsed-wave Doppler imaging, pulsed-wave tissue Doppler imaging and 2D speckle tracking.

Results: Although AS, nr-axSpA and control groups had similar ejection fraction (59±5.2, 60±4.6, 60±4.6 respectively), global longitudinal strain (GLS) (20.5±3.3, 21.1±3.5, 22.3±2.4 p=0.004) was different between groups respectively. Post-hoc tests showed that GLS was similar between nr-axSpA and control groups however GLS was significantly low in AS patients. In univariate analysis GLS was correlated with age (r=-0.253, p=0.032) and peripheral arthritis (r=-0.233, p=0.035). However in regression analysis peripheral arthritis (Odds ratio: -2.28 [95% Confidence interval: (-4.49) - (-0.67)], p=0.009) was found as the only independent predictor of GLS.

Conclusions: Left ventricular function had impaired in AS patients and impaired ventricular function might also be the other differentiating factor between radiographic and nr-axSpA patients.

Cardiac imaging / Echocardiography

OP-079

Impact of ischemic preconditioning on ischemic mitral regurgitation in patients with acute coronary syndrome

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Background and Aim: Twenty percent of patients presenting with acute coronary syndrome (ACS) develop pronounced mitral regurgitation (MR) in 3 months. This MR develops because of mitral valve annular dilatation due to left ventricular (LV) remodeling and/or tethering of mitral valve chorda and is called ischemic/functional MR. Previously performed clinical trials have shown that, in patients with angina and ischemic preconditioning prior to ACS have less LV remodeling and preserved LV function. The aim of this study based on this hypothesis is; to determine the development and severity of ischemic MR in patients presenting with ACS, at admission and 3 months later with and without prior angina pectoris and ischemic preconditioning.

Methods: Forty five (45) patients presenting with ST elevation ACS and revascularization with percutaneous coronary intervention were enrolled in this study. The presence of angina within 72 hours before ACS were questioned with a detailed history. Patients were then divided into two groups as patients with and without angina pectoris. Transthoracic echocardiography was performed in all patients in the first 24 hours, 72 hours and 3 months after ACS development. Ischemic MR was evaluated by the PISA method, PISA radius (RAD), effective regurgitant orifice area (EROA) and regurgitant volume (RV) and ejection fraction (EF %) were calculated; troponin T (TnT), CK-MB and pro-brain natriuretic peptid (pro-BNP) levels were compared between two groups.

Results: MR parameters in the first 24 hours and 72 hours were similar in patients with and without angina. When the 24 hour and 3rd. month MR parameters were compared in patients with and without angina; MR, RAD, EROA, RV levels were significantly decreased and EF (%) was significantly increased in patients with angina (p<0.001 for all). The two groups were also compared at the 3rd. month and was found that MR RAD, ERO and RV were significantly decreased and EF was significantly increased (p=0.012; p=0.007; p=0.011; and, p=0.04 respectively). According to the biochemical results, pro-BNP and troponin T levels were similar at the end of 3rd. month, but CK-MB decline in patients with angina at 3rd. month was significant (p=0.03).

Conclusions: In patients with ST elevation ACS treated with standard percutaneous revascularization procedures, those who describe angina within 72 hours prior to ACS, have significantly decreased ischemic MR and significantly increased EF compared to those without angina, at three months follow-up.

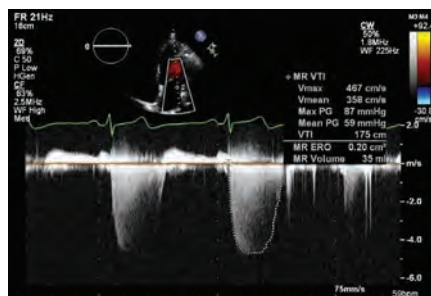


Figure 1. Calculating EROA (effective regurgitant orifice area) and RV (regurgitant volume) with VTI (velocity time integral).

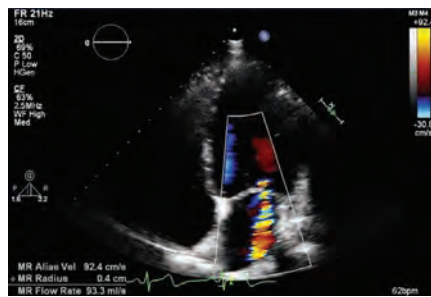


Figure 2. Mitral regurgitation with color doppler echocardiography.

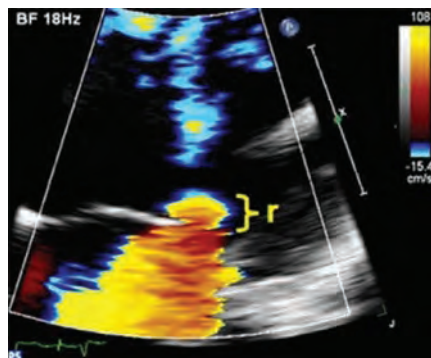


Figure 3. PISA radius (r), apical 4-chamber view.

Table 1. Comparison of PISA RAD, PISA EROA, PISA RV and LV EF (%) in patients with and without angina pectoris, first 24 hours, 72 hours and 3 months after ACS development

	Angina pectoris (+)				Angina pectoris (-)			
	PISA RAD (cm)	PISA EROA (cm ²)	PISA RV (ml)	LV EF (%)	PISA RAD (cm)	PISA EROA (cm ²)	PISA RV (ml)	LV EF (%)
1.day	0,19±0,14	0,08±0,07	8,05±9,54	48,85±9,09	0,13±0,14	0,06±0,07	6,37±8,87	55,25±8,26
3.day	0,19±0,15	0,08±0,09	8,27±10,76	49,50±9,45	0,17±0,15	0,08±0,08	9,11±11,20	56,12±9,44
3.month	0,07±0,08	0,02±0,03	2,35±4,11	56,19±7,01	0,23±0,23	0,10±0,09	11,95±11,85	53,47±8,62

Table 2. Comparison of PISA RAD, PISA EROA, PISA RV and LV EF (%) in patients with angina pectoris, first 24 hours and 3 months after ACS development

Angina pectoris(+)	PISA RAD (cm)	PISA EROA (cm ²)	PISA RV (ml)	LV EF (%)
1.day	0,19±0,14	0,08±0,07	8,50±9,54	48,85±9,09
3.month	0,07±0,08	0,02±0,03	2,35±4,11	56,19±7,01
p value	<0,001	<0,001	<0,001	<0,001

Table 3. Comparison of PISA RAD, PISA EROA, PISA RV and LV EF (%) in patients without angina pectoris, first 24 hours and 3 months after ACS development

Angina pectoris(-)	PISA RAD (cm)	PISA EROA (cm ²)	PISA RV (ml)	LV EF (%)
1.day	0,13±0,14	0,06±0,07	6,37±8,87	55,25±8,26
3.month	0,23±0,23	0,10±0,09	11,95±11,85	53,47±8,62
p value	0,064	0,064	0,030	0,300

Heart failure

OP-080

Predictors of mortality in patients admitted to hospital with acute heart failure: tertiary center results

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Background and Aim: Acute heart failure (AHF) is an important hospitalization reason for chronic heart failure patients. Every AHF hospitalization is known to be related with rehospitalization and increased mortality risk. Aim of this study was to assess the predictors of in-hospital mortality in AHF patients admitted to tertiary center.

Methods: The data of 4,829 patients who were admitted to intensive cardiac care unit (ICCU) between Sep 2014 and Jan 2016 was analyzed retrospectively. The data was retrieved from the ICCU electronic database of our clinic. 384 patients had primary diagnosis of AHF. Their mean age was 65±15 years and 67% were male (258 patients). Demographic, clinical, laboratory characteristics of the study population were analyzed.

Results: Mortality rate was 30% among these patients during hospitalization. There were no differences between demographic characteristic and previous cardiac events. However chronic renal failure was significantly higher in mortality (+) group. Creatinine levels were higher in these patients and they were hypotensive (Table 1). Univariate and multivariate regression analysis were performed to determine predictors of mortality in these patients. Systolic blood pressure lower than 90 mmHg (OR: 13.072, 95%CI:7.211-23.698, p<0.001), addition of acute renal failure during hospitalization (OR:2.119, 95%CI:1.193-3.765, p=0.010), previous stroke (OR:3.248, 95%CI:1.074-9.822, p=0.037) and chronic obstructive pulmonary disease (OR:2.589, 95%CI: 1.046-6.407, p=0.04) were assessed as predictors.

Conclusions: We determined most important parameter to predict in-hospital mortality in patients admitted with AHF as hypotension during hospitalization. In daily clinical practice hypotension in a AHF patient should be a warning sign for clinicians to be more aggressive for treatment.

Table 1.

Characteristics	Mortality (+)	Mortality (-)	p value
Age (years)	66±16	64±14	0.374
Male/Female (n, %)	74(28%)/43 (34%)	184 (71%)/83 (65%)	0.29
Creatinine (mg/dl)	2.02±1.32	1.60±1.36	0.005
Hemoglobin (mg/dl)	11.4±2.3	11.9±2.1	0.414
Hypertension	56 (47%)	146 (54%)	0.224
Diabetes Mellitus	40 (34%)	105 (39%)	0.362
Smoking	11 (9%)	33 (12%)	0.488
Previous myocardial infarction	49 (41%)	89 (33%)	0.307
Previous stroke	9 (7.7%)	11 (4.1%)	0.210
Previous revascularization	40 (34%)	97 (36%)	0.72
Chronic obstructive pulmonary disease	14 (12%)	18 (6%)	0.108
Chronic renal failure	35 (29.9%)	53 (19.9%)	0.035
Acute renal failure	50 (42.7%)	52 (19.5%)	<0.001
Normotension on admittance	23 (19%)	157 (58%)	
Hypotension on admittance	86 (73%)	45 (16%)	<0.001
Hypertension on admittance	8 (6%)	65 (24%)	

Heart failure

OP-081

Decreased epicardial fat thickness is associated with all-cause mortality in patients with heart failure with reduced ejection fraction

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Background and Aim: Heart failure with reduced ejection fraction (HFrEF) constitutes a mortal condition in which prognostification may be necessitated. Epicardial fat tissue (EFT) is a part of visceral fat depot which has a direct relationship among many cardiovascular conditions. Reduced EFT has been shown in patients with heart failure, however, its effects on prognosis has not been addressed yet. In our study, we investigated the effect of EFT thickness on all-cause mortality in patients with HFrEF.

Methods: 100 patients with HFrEF and 25 control cases were included. Patients risk factors, heart failure and device status were noted. Telephone calls and health ministry records were used for determining all-cause mortality. EFT was measured in systole from the anterior right ventricular free wall as previously described.

Results: During median follow-up of 51 months (range:0 to 54 months), half of the patients (49 patients) did not survive. Non-survivors were older (59.8±12.5 vs. 52.7±13.6), had lower mean arterial pressure (77.2±12.7 vs. 89.3±18.7), had prominently ischemic cardiomyopathy, were mostly in advanced functional classes and had three times likely congestive heart failure than survivors. Diabetes mellitus was 2 times prevalent and antiplatelet and beta blocker usage was more pronounced in non-survivor group. Also in non-survivor group, ejection fraction was 40% lower and systolic pulmonary arterial pressure (sPAB) was 50% higher. Non-survivors' troponin, uric acid, and C-reactive protein levels were higher whereas glomerular filtration rate, serum sodium, hemoglobin and albumin levels were lower than survivors. EFT was 70% higher in survivors than in non-survivors (4.2±2.1 vs. 2.5±1.3, p<0.001). In multivariate analysis, along with mean arterial pressure (HR:0.952, p<0.001), sPAB (1.032, p=0.003) and GFR (0.978, p=0.001), 1 mm increase in EFT was associated with 28% decreased mortality (HR:0.721, p=0.006). In ROC analysis, EFT discriminated all-cause mortality with an accuracy of 73%. An EFT cut-off value of 2.5 mm had 63.3% sensitivity and 72.5% specificity. Patients with an EFT ≥2.5 mm had a survival benefit of 15 months than patients with EFT <2.5 mm (log-rank<0.001).

Conclusions: Decreased EFT was associated with increased all-cause mortality in patients HFrEF. Measurement of this costless and readily available parameter may be beneficial in HFrEF prognostification.

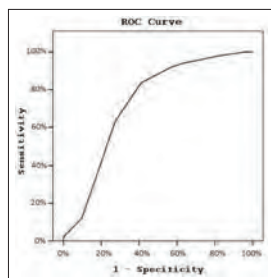


Figure 1.

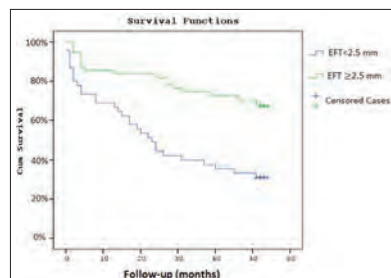


Figure 2.

Heart failure

OP-082

Relationship between plasma levels of PIIINP (N-Terminal Propeptide Procollagen Type III) and myocardial fibrosis with cardiac MRI in heart failure

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Background and Aim: Myocardial fibrosis, defined as a key component of heart failure and since then various studies showed a strong connection between fibrosis and progression of heart failure. Cardiac magnetic resonance emerged as a crucial noninvasive imaging technique because of its high accuracy and high fidelity in detection of fibrosis. PIIINP (N-Terminal Propeptide Procollagen Type III) is a propeptide while occurs synthesis cardiac type III collagen. The noninvasive assessment of fibrosis is advantageous in early prediction of possible adverse outcomes and creates an opportunity to utilize new therapeutic approaches that target fibrosis in heart failure. Our aim is with this study, correlation detection of myocardial fibrosis with cardiac MRI and plasma PIIINP concentration in heart failure patients.

Methods: The study included 65 patients, of whom indicated for several causes for cardiac MRI (cardiac masses, cardiac thrombus, searching viability, estimating EF, suspecting ARVD etc.) and older than ≥18 years, EF ≤40 patients at 01/2017-02/2018. At first, these patients were divided into 2 groups according to study group with EF ≤40 (n=45 age:56±8.67) and control group (n=20 age:37.55±9.96) with normal cardiac MRI. Then the study group were divided into 2 groups again according to detection (n=25) and nondetection (n=20) of myocardial fibrosis with cardiac MRI and after that compared of their plasma PIIINP concentration.

Results: N-Terminal Propeptide of Type III Procollagen were increased in plasma whom detection of cardiac fibrosis with cardiac MRI compared with other patients groups (p=0.002; p<0.01). Also statistical analysis revealed significant correlation (p<0.05) between presence of %LGE and plasma PIIINP quantity (r=0.494; p=0.012; p<0.05). Incidence of coronary artery disease increased in heart failure and myocardial fibrosis group besides that determined using ACE inh. decreasing quantity of myocardial fibrosis (p=0.034; p=0.010; p<0.05). Using ARB, MRA and statin determined non-effective quantity of myocardial fibrosis (p>0.05). Low GFR level determined heart failure and myocardial fibrosis group (p<0.01). Low EF and high LVEDV and LVEDS determined myocardial fibrosis group (p<0.01).

Conclusions: The main findings of our study were N-Terminal Propeptide of Type III Procollagen (PIIINP) were predictors of heart failure in patients for predicting left ventricular fibrosis. The use of PIIINP in clinical practice will identify patients with heart failure with structural and functional changes in the myocardium in the early stages of the disease.

Heart failure

OP-083

Heart failure with preserved and mid-range ejection fraction in octogenarians: Results from APOLLON (A comprehensive, observational registry of heart failure with mid-range and preserved ejection fraction) study

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Background and Aim: To compare real-world characteristics and management of individuals aged 80 and older with heart failure and mid-range ejection fraction (HFmrEF) and heart failure with preserved ejection fraction (HFpEF) derived from a large cohort of survey and to compare them with those younger than 80 from the same survey.

Methods: The APOLLON trial (A Comprehensive, Observational registry of heart failure with mid-range and preserved ejection fraction) is an observational, multicenter, and non-interventional study conducted in Turkey (NCT03026114). Consecutive patients admitted to the cardiology clinic aged ≥18 years at the time of enrollment with symptoms/signs of heart failure, a left ventricular ejection fraction (LVEF) ≥40%, elevated levels of natriuretic peptides and evidence of other cardiac functional or structural alterations were included. Exclusion criteria were; patients with a LVEF <40%; patients with significant chronic pulmonary disease; patients with primary hemodynamically significant uncorrected valvular heart disease; patients with any history of surgically corrected heart valve diseases (e.g., mechanical or bioprosthetic heart valves); patients with myocardial infarction, stroke, or coronary artery bypass graft surgery in the past 90 days; percutaneous coronary intervention or pacemaker implantation in the past 30 days; heart transplant recipients; known infiltrative or hypertrophic obstructive cardiomyopathy or known pericardial constriction; patients with congenital heart diseases or cor pulmonale; and pregnant.

Results: Twelve cardiology units enrolled 1065 (mean age of 67.1±10.6 years, 54% women) patients. Participants aged 80 and older (n=123, 11.5%) were more likely to be female (66.7% vs 52.5%, p=0.003), had a higher prevalence of atrial fibrillation (49.6 vs 34%, p=0.001), anemia (46.3 vs 33.4%, p=0.005), and cerebrovascular accident (13 vs 5.7%, p=0.002) than those who were younger than 80. N-terminal pro B-type natriuretic peptide levels [1037 (541–1899) pg/ml vs 550 (258–1014) pg/ml, p<0.001] and blood urea nitrogen [21 (16–27) vs 17 (13–22), p<0.001] were higher in those aged 80 and older than in those younger than 80 (Figure 1). Octogenarians did not significantly differ from younger patients in the prevalence of HFmrEF (24.4 vs 22.9%) and HFpEF (75.6 vs 77.1%).

Conclusions: APOLLON study revealed that nearly 12% of individuals with HFmrEF and HFpEF in this real-world sample were aged 80 and older. Participants aged 80 and older were more likely to be female and have more comorbidities than those who were younger than 80. One-fourth of the octogenarians with signs and symptoms of heart failure, LVEF $\geq 40\%$ and increased natriuretic peptide levels had HFmrEF.

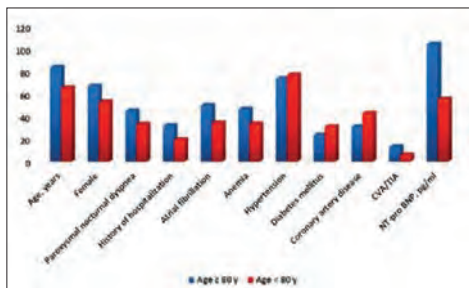


Figure 1. Clinical profile and comorbid features in patients aged 80 and older vs younger than 80. NT-pro BNP, N-terminal pro-B-type natriuretic peptide.

Heart failure

OP-084

Effect of severe functional mitral regurgitation on novel hemodynamic findings in heart transplant candidates

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Background and Aim: A severe functional mitral regurgitation had additive factor for left ventricular filling pressure and pulmonary arterial pressures. We aimed to investigate the relationship between severe functional mitral regurgitation (FMR) and left ventricle stroke work index (LVSWI), left ventricular cardiac power index (LVCPi), right ventricle stroke work index (RVSWI), pulmonary artery pulsatility index (PAPi), pulmonary arterial capacitance (PAC) and pulmonary arterial elastance (PAE) in patients who were heart transplant candidates.

Methods: A total of 238 patients with left ventricle ejection fraction (LVEF) $\leq 25\%$, NYHA class III-V and INTERMACS IV -VII were included to study and they were divided into two groups; namely, those with severe and non-severe FMR. The severe FMR was defined as effective regurgitation area $\geq 20\text{ mm}^2$ and regurgitation volume $\geq 30\text{ ml}$.

Results: Seventy six patients had severe and 162 patients had non-severe FMR. There was no difference in terms of LVEF, NYHA and INTERMACS grades and heart failure duration among to groups ($p > 0.05$). The CO, CI, SV, SVI, LVSWI and LVCPi were lower in severe FMR compared to non-severe FMR [3.1 \pm 0.7 vs. 3.5 \pm 0.89, $p = 0.005$; 1.6 \pm 0.3 vs. 1.8 \pm 0.5, $p = 0.021$; 35.0 \pm 9.1 vs. 44.7 \pm 15.8, $p = 0.001$; 19.0 \pm 4.5 vs. 23.4 \pm 8.9, $p = 0.003$; 13.1 (10.8-18.5) vs. 17.1 (12.4-24.9), $p < 0.001$ and 1482 (1185.1-1830.7) vs. 1760.0 (1324.0-2396.0), $p = 0.004$]. The systemic vascular resistance (SVR) was similar among to groups (22.0 \pm 8.2 vs. 21.5 \pm 7.9, $p = 0.656$). The pulmonary arterial systolic pressure, pulmonary wedge pressure were higher in patients with severe FMR than those with non-severe FMR (58.3 \pm 16.0 vs. 49.3 \pm 18.6, $p = 0.001$; 38.4 \pm 11.5 vs. 30.7 \pm 11.0, $p < 0.001$ and 25.9 \pm 7.0 vs. 21.5 \pm 7.4, $p < 0.001$). The RVSWI and PAPI were similar among to groups (6.5 \pm 2.9 vs. 6.2 vs. 3.1, $p = 0.416$; 3.2 \pm 2.0 vs. 3.7 \pm 2.5, $p = 0.232$). The patients with severe FMR had higher PVR and PAE value and lower PAC than patients with non-severe FMR [4.0 (2.3-6.8) vs. 2.6 (1.2-4.3), $p = 0.001$; 1.5 (1.1-2.3) vs. 1.1 (0.79-1.68), $p < 0.001$ and 1.1 (0.78-1.74) vs. 1.5 (1.0-2.3), $p = 0.001$].

Conclusions: The patients with severe FMR had lower left ventricular cardiac performance assessed by CI, LVSWI, and LVCPi without increase in afterload (SVR), while they had higher pulmonary afterload assessed by PVR, PAC, PAE without increase in right ventricular work defined by RVSWI and PAPI.

Heart failure

OP-085

Could plasma osmolality change in heart failure be a predictor of thromboembolism?

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Background and Aim: Heart failure (HF) is a complex clinical condition with a poor prognosis and high cardiovascular mortality. The plasma osmolality is a useful parameter measuring the fluid and electrolyte balance of the body in HF patients (1). Mean platelet volume (MPV), platelet distribution width (PDW), red blood cell distribution width (RDW), mean platelet volume to platelet count ratio (MPV/PLT), neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) have been shown to be associated with platelet hyperreactivity, thromboembolism and so many cardiovascular diseases (2). The aim of our study was to evaluate a possible relationship between these parameters and plasma osmolality in HF patients. To date, in the literature, there have been no studies showing the relationship between them.

Methods: The study included 112 consecutive patients with systolic heart failure with an ejection fraction (EF) of $\leq 35\%$. Plasma osmolality was calculated as $(2 \times \text{Na}) + (\text{BUN}/2.8) + (\text{Glucose}/18)$. The patients were classified into three groups based on admission osmolality. The first group ($n = 30$) was defined as hypo osmolar ($< 285\text{ mOsm/kg}$), second group ($n = 59$) was defined as normo-osmolar (285 and 295 mOsm/kg) and third group ($n = 23$) was defined as hyperosmolar ($> 295\text{ mOsm/kg}$). On admission, demographic data, medications and all laboratory measures were recorded for each patient. Patients with acute myocardial ischemia, chronic in-

flammatory and infection disorders, cancer and pregnancy were excluded from the study.

Results: The mean age of the patients was 66.4 ± 10.3 (range 40-85; 85 males, 27 females). The median level of EF was 30%. The mean plasma osmolality was $289.48 \pm 7.03\text{ mOsm/kg}$. Baseline characteristics, laboratory findings and medications used are shown in Table 1, 2 and 3A. This study showed that hyperosmolar group had higher MPV (9.5 ± 0.89 ; 9.3 ± 0.86 ; $9.1 \pm 0.82\text{ fL}$, $p = 0.167$, respectively), PDW (17.46 ± 2.28 ; $15.92 \pm 2.18\%$, $p = 0.065$, respectively) compared to normo and hypoosmolar group (Table 3B). Hyperosmolar group had significantly higher RDW [17.50 (13.40-25.3); 15.6 (12.2-26.4); 15.55 (12.7-20.3), $p = 0.003$, respectively] and NLR [3.29 (1.21-18.31); 2.19 (0.85-9.84); 2.06 (1.23-7.04), $p = 0.001$, respectively] compared to normo and hypoosmolar group (Table 3B). Moreover, in correlation analysis, plasma osmolality was found to be significantly positively correlated with PDW ($r = 0.189$, $p = 0.045$), RDW ($r = 0.325$, $p < 0.001$) and NLR ($r = 0.229$, $p = 0.015$) (Table 4) (Fig 1).

Conclusions: Our study results showed that MPV, PDW, RDW and NLR were associated with plasma osmolality in HF and as the main finding, hyperosmolar group had higher MPV, PDW, RDW, PLR and NLR than other groups (Table 3B). The change in plasma osmolality may be predictive for thromboembolism in HF. Thus, the evaluation of plasma osmolality in HF may be important for prognosis and mortality. To the best of our knowledge, our study is the first to show the relationship between plasma osmolality and thromboembolism in HF by these parameters.

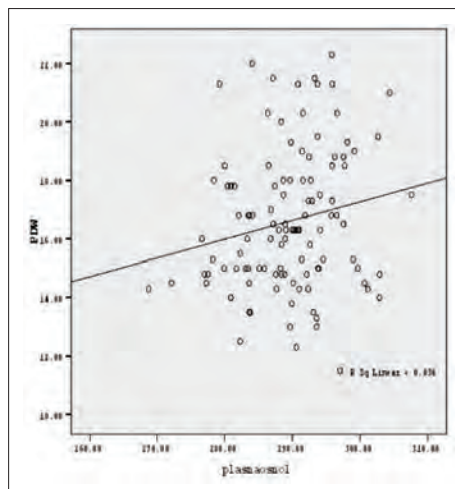


Figure 1.

Table 1. Baseline characteristics of study patients

Table 1:	Group 1 (Hypoosmolar) (N: 30)	Group 2 (Normoosmolar) (N: 59)	Group 3 (Hyperosmolar) (N: 23)	P value
Mean age (years)	64.3 \pm 11.2	66.1 \pm 10.7	69.6 \pm 8.2	0.18
Male/Female	23/7	45/14	17/6	0.969
Hypertension	12 (% 41)	39 (%66)	14 (% 61)	0.084
Diabetes mellitus	8 (% 28)	19 (% 32)	13 (%57)	0.065
Coronary artery disease	21 (%72)	44 (%75)	17 (%74)	0.977
ejection fraction (%)	30 (22 – 35)	30 (20 – 35)	30 (20 – 35)	0.915

Table 2. Medication of study patients

Table 2: Medication	Group 1 (Hypoosmolar) (N: 30)	Group 2 (Normoosmolar) (N: 59)	Group 3 (Hyperosmolar) (N: 23)	P value
ACE inhibitor/ARB*	19 (% 65.5)	39 (%66)	15 (% 65)	0.997
Beta-blockers	27 (% 93)	51 (% 86)	21 (%91)	0.598
Diuretics	18 (% 62)	38 (%64)	19 (%82)	0.218
Mineralocorticoid receptor antagonist	18 (%62)	34 (%58)	11 (% 48)	0.577

Table 3a. Laboratory findings of patients

Table 3A:	Group 1 (Hypoosmolar) (N: 30)	Group 2 (Normoosmolar) (N: 59)	Group 3 (Hyperosmolar) (N: 23)	P value
Plasma osmolality (mOsm/kg)	280,56 ± 3.6	290,31 ± 2.4	299 ± 3.2	< 0.001*
Fasting glucose(mg/dl)	93.5 (77- 183)	106 (79 – 307)	124 (83- 325)	< 0.001*
BUN (mg/dl)	15.17 (7.47-32.22)	18.21 (11.68-51.37)	25.68 (12.61-61.64)	< 0.001*
Creatinine (mg/dl)	1.02 (0.73-1.35)	1.07 (0.65-1.75)	1.33 (0.79-2.85)	< 0.001*
Sodium (mEq/L)	134,7 ± 2,3	138,3 ± 1,9	139,9 ± 2,9	< 0.001*
Hemoglobin (gr/dl)	13,3 ± 1,35	13,6 ± 1,97	12 ± 1,39	0.001*
Brain natriuretic peptide (NT-ProBNP) (pg/ml)	1950 (202- 19600)	1145 (136-26700)	3580 (170-35000)	0.03*

Table 3b.

Table 3B:	Group 1 (Hypoosmolar) (N: 30)	Group 2 (Normoosmolar) (N: 59)	Group 3 (Hyperosmolar) (N: 23)	P value
Mean platelet volume (MPV)(fL)	9.1 ± 0.82	9.3 ± 0.86	9.5 ± 0.89	0.167
Platelet (PLT) Distribution Width (PDW) (%)	15.92 ± 2.18	16.59 ± 2.28	17.46 ± 2.69	0.065
Red Cell Distribution Width (RDW) (%)	15.55 (12.7-20.3)	15.6 (12.2 – 26.4)	17.50 (13.40-25.3)	0.003*
MPV/PLT ratio	0.041(0.026-0.095)	0.038(0.018-0.081)	0.046(0.026-0.104)	0.091
Neutrophil to lymphocyte ratio (NLR)	2.06 (1.23 – 7. 04)	2.19 (0.85 -9.84)	3,29 (1.21 – 18.31)	0.001*
Platelet to lymphocyte ratio (PLR)	128.2 (67.72-250)	116 (47.15-308.49)	139.4(66.21-461.54)	0.115

Table 4. Correlation analysis between plasma osmolality and laboratory findings

TABLE 4	P value	r value
Mean platelet volume (MPV)	P= 0.121	r= 0.148
Platelet Distribution Width (PDW)	P= 0.045*	r=0.189*
Red Cell Distribution Width (RDW)	P= 0.000*	r= 0.325*
MPV/PLT ratio	P= 0.248	r=0.110
Neutrophil to lymphocyte ratio (NLR)	P= 0.015*	r= 0.229*
Platelet to lymphocyte ratio (PLR)	p= 0.616	r= 0.048
Brain natriuretic peptide (NT-ProBNP)	p= 0.069	r= 0.173

Coronary artery disease / Acute coronary syndrome

OP-086

Association of angiotensin II type 1 A1166C gene polymorphisms with coronary artery disease in thrace region of Turkey

Gokay Taylan, Yuksek Aksoy, Mustafa Adem Yilmaztepe, Nasir Sivri, Flora Ozkalayci, Ahmet Kenan Yalta

Department of Cardiology, Trakya University Faculty of Medicine, Edirne

Background and Aim: Even though risk factors for coronary artery disease (CAD) are well established, there still exists a significant gap to fully understand the pathology of atherosclerotic heart disease evolving without conventional risk factors. Therefore, genetic factors are considered to play a significant role in this setting. In the literature, there exists a plenitude of studies on the relation of genetic background with atherosclerosis. Accordingly, the current study was devised to assess the relation between Angiotensin 2 type 1 receptor A1166C gene polymorphism and CAD.

Methods: Patients with documented CAD (n=121) were compared with control group with normal coronary arteries (n=121). Diagnosis of CAD was made through coronary angiogram (CAG). Median age was 59±12 with an equal gender distribution. Through amplification of DNA with PCR technique, we made a comparison between the two groups with regard to Angiotensin 2 type 1 receptor A1166C gene polymorphism.

Results: We demonstrated that AA and CC genotypes were more frequent, yet AC genotype was less frequent among patients with CAD as compared with control group (95% CI, p=0.001). AT1RA1166C gene polymorphism along with CC and C alleles were found to be associated with CAD (OR 0.93, 95% CI 0.891-0.979, p=0.004). Furthermore, HT and low levels of serum HDL also had a significant association with AT1RA1166C gene polymorphism (95% CI, p=0.03 ve p<0.01).

Conclusions: Current study suggests AT1RA1166C gene polymorphism, CC genotype and C allele as potential risk factors for atherosclerotic CAD. Patients harboring these genetic variants should be under close supervision for future development of CAD.

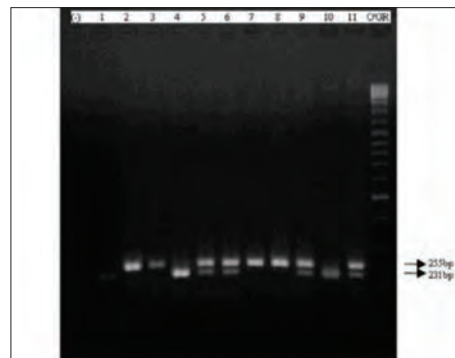


Figure 1. 3% Agarose gel image stained with EtBr showing AT1RA1166C polymorphism. AA (255 bp; 2, 3, 7, ve 8), AC (255 bp, 231 bp, and 24 bp (do not appear); 5, 6, 9, and 11) ve CC (231 bp; 1, 4 and 10), O'GR; 100 bp DNA marker (O'GeneRuler 100bp DNA Ladder-Fermentas Life Sciences).

Table 1. Hardy-Weinberg equilibrium in the study groups

Hardy-Weinberg equilibrium and Pearson Ki-square test in the study groups					
Groups	Genotypes	Observed	Expected	Ki-square	p
CAD	AA	71	67,5	0,821	0,365
	AC+CC	50	53,5		
CONTROL	AA	64	67,5	0,821	0,365
	AC+CC	57	53,5		

CAD: Coronary artery disease.

Table 2. Demographic characteristics of control and patient groups for AT1RA1166C gene polymorphism

Patient and control groups information	Control groups (n=121)	Patient groups (n=121)	P
Age	54±11	64±12	0,07
Gender			
Female	77(%63.6)	44(%36.3)	0,07
Male	44(%36.3)	77(%63.6)	
BMI	27.7±0.7	27.6±0.7	0,3
HT	60(%49.5)	95(%78)	<0,01
DM	26(%21.4)	37(%30.5)	<0,01
Smoking	47(%38.8)	59(%48.7)	0.01
Family history	16(%13.2)	35(%28.9)	<0,01
Total cholesterol	184±40	176±50	0.03
Triglyceride	146±84	153±101	0.97
HDL	49±13	41±12	0.65
LDL	117±36	113±44	0.05

BMI: Body Mass Index, HT: Hypertension, DM: Diabetes Mellitus, HDL: High-density lipoprotein, LDL: Low-Density Lipoprotein, n: Patient number.

ADD-1

T.C. TRAKYA ÜNİVERSİTESİ TIP FAKÜLTESİ DEKANLIĞI
GİRİŞİMSEL OLMAYAN KLİNİK ARAŞTIRMALAR ETİK KURULU Başkanlığı

T.C. TRAKYA ÜNİVERSİTESİ TIP FAKÜLTESİ DEKANLIĞI
GİRİŞİMSEL OLMAYAN KLİNİK ARAŞTIRMALAR ETİK KURULU Başkanlığı

ARASTIRMA BAŞVURUSU ÖZETİ

PROTOKOL ADI: T01F-G08AK-B03111
PROTOKOL ADI: Biyogenetik Koroner Arter Hastalıklarının Genetik ve Klinik Özelliklerinin Belirlenmesi
ARASTIRMA BAŞVURUSU ÖZETİ: Biyogenetik Koroner Arter Hastalıklarının Genetik ve Klinik Özelliklerinin Belirlenmesi
ARASTIRMA BAŞVURUSU ÖZETİ: Biyogenetik Koroner Arter Hastalıklarının Genetik ve Klinik Özelliklerinin Belirlenmesi

KARAR BİLGİLERİ: Karar No: 12/18
Tarih: 09.08.2018
Yeterli: Evet

ETİK KURULU BAŞKANLIĞI: Başkan: Doç. Dr. Mustafa Umut Somuncu

Ünvan/Ad Soyad	Ünvan/Dak	Kurumu	Onaylı	Bilgi(*)	Katılım(**)	İmza
Prof. Dr. Tuba Vatansever Özdemir	Genetik Bilgiyi ve Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. Fatma Kadir Yıldırım	Tıp Tarihi ve Etik	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Prof. Dr. C. Hakan Karadağ	Tıbbi Farmakoloji	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. F. Nispet Turan	Biyoistatistik	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Tokdemir	Tıbbi Genetik	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Ümit	İç Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. S. Hakan Vardar	Patoloji	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. S. Hakan Yıldırım	İç Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Yılmaz	Halk Sağlığı	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Prof. Dr. K. Hakan Yıldırım	Klinik Hastalıkları ve Diş Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Yıldırım	Halk Sağlığı ve Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Prof. Dr. H. Hakan Yıldırım	Klinik, Genetik ve Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Yıldırım	Genel Cerrahi	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Yıldırım	T.C. Cerrahi ve Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Yıldırım	T.C. Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>

Prof. Dr. Mustafa Umut Somuncu
Başkan

Figure 1. Ethics committee approval.

ADD-2

T.C. TRAKYA ÜNİVERSİTESİ TIP FAKÜLTESİ DEKANLIĞI
GİRİŞİMSEL OLMAYAN KLİNİK ARAŞTIRMALAR ETİK KURULU Başkanlığı

ARASTIRMA BAŞVURUSU ÖZETİ

PROTOKOL ADI: T01F-G08AK-B03111
PROTOKOL ADI: Biyogenetik Koroner Arter Hastalıklarının Genetik ve Klinik Özelliklerinin Belirlenmesi
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ARASTIRMA BAŞVURUSU ÖZETİ: Biyogenetik Koroner Arter Hastalıklarının Genetik ve Klinik Özelliklerinin Belirlenmesi

KARAR BİLGİLERİ: Karar No: 13/18
Tarih: 09.08.2018
Yeterli: Evet

ETİK KURULU BAŞKANLIĞI: Başkan: Doç. Dr. Mustafa Umut Somuncu

Ünvan/Ad Soyad	Ünvan/Dak	Kurumu	Onaylı	Bilgi(*)	Katılım(**)	İmza
Prof. Dr. Tuba Vatansever Özdemir	Genetik Bilgiyi ve Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
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Doç. Dr. S. Hakan Yıldırım	İç Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Yılmaz	Halk Sağlığı	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Prof. Dr. K. Hakan Yıldırım	Klinik Hastalıkları ve Diş Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
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Doç. Dr. H. Hakan Yıldırım	Genel Cerrahi	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Yıldırım	T.C. Cerrahi ve Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>
Doç. Dr. H. Hakan Yıldırım	T.C. Hastalıkları	T01F/G08AK/B03111	E	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<i>[Signature]</i>

Prof. Dr. Mustafa Umut Somuncu
Başkan

Figure 2. Ethics committee approval.

Table 3. Distribution of AT1R A1166C genotypes between patients and control groups

AT1R A1166C Genotypes	GROUPS		Total	P
	Patient	Control		
AA	71(58.7)	64(52.9)	135(55.8)	0,001
AC	42(34.7)	57(47.1)	99(40.9)	
CC	8(6.6)	0(0.0)	8(3.3)	
Total	121(100)	121(100)	242(100)	

AA: Adenine-Adenine, AC: Adenine- Cytosine, CC:Cytosine-Cytosine.

Table 4. Relationship between AT1R A1166C genotypes and coronary artery disease

Genotype	Odds ratio(OR)	P
AA göre AC+CC	1,26(0,761-2,10)	0,365
CC göre AA+AC	0,93 (0,891-0,979)	0,004
AC göre AA+CC	0,60 (0,356-1,001)	0,05

AA: Adenine-Adenine, AC: Adenine- Cytosine, CC:Cytosine-Cytosine.

Table 5. Relationship between AT1R A1166C genotypes and coronary artery disease severity

CAD severity	AT1R A1166C Gene Polymorphisms			Total	P
	AA	AC	CC		
Single vessel	31	13	4	48	0,62
Two vessel	11	10	1	22	
Three vessel	29	19	3	51	
Total	71	42	8	121	

AA: Adenine-Adenine, AC: Adenine-Cytosine, CC:Cytosine-Cytosine, CAD: Coronary artery disease, AT1R:Angiotensin II type 1 receptor

Table 6. Correlation between coronary artery disease risk factors and AT1R A1166C gene polymorphisms

Risk factors	Odds Ratio(OR) and Confidence interval(CI)	P
Age*		
Male	1,89 (0,66-5,4)	0,23
Female	1 (0,47-2,1)	0,99
HT	1,59(1,44-1,75)	0,03
DM	1,05(0,20-5,38)	0,94
Smoking	1,31(0,30-5,61)	0,71
Family history	1,9 (0,22-15,82)	0,54
BMI**	0,7 (0,13-3,62)	0,69
Total Cholesterol ***	1,3 (0,65-1,94)	0,67
Triglyceride ****	1,14 (0,59-2,18)	0,70
HDL*****	2,21 (1,26-3,88)	<0,01
LDL*****	1,21 (0,70-2,1)	0,5

BMI: Body Mass Index, HT: Hypertension, DM: Diabetes Mellitus, HDL:High-density lipoprotein, LDL: Low-Density Lipoprotein Risk factors for CAD as stated in the national cholesterol education program (NCEP ATP III); *The age risk factor for males was taken as over 45 years old. For females it was taken as over 55 years old. ** BMI was taken above 25 as a risk factor. *** Total cholesterol was taken as a risk value above 200 mg/dl. **** Triglyceride risk factor was taken above 200 mg/dl. ***** Low HDL was taken as a risk factor below 40mg / dl. ***** LDL height was taken as a risk factor above 130 mg/dl.

Coronary artery disease / Acute coronary syndrome

OP-087

The Prognostic value of elevated matrix metalloproteinases-9 in patients undergoing primary percutaneous coronary intervention for ST-elevation myocardial infarction: A two year prospective study

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Background and Aim: The increase in matrix metalloproteinases (MMP)-9 in ST-elevation myocardial infarction (STEMI) is well established, however the existing data about admission MMP-9 values as prognostic marker after STEMI are limited and have been conflicting. In this study, we aim to assess the clinical significance of MMP-9 in predicting 2-year adverse cardiovascular events in patients who underwent primary percutaneous coronary intervention (PCI) after STEMI.

Methods: In this prospective study, we included 249 patients with STEMI undergoing PCI. The high-MMP-9 (n=125, age=56.9±12.7) group and the low MMP group (n=124, age: 55.2±11.2) was formed using the value of 12.92 ng/ml as a threshold from receiver operator characteristic analysis. In-hospital, 1-year and 2-year follow-ups were evaluated for both groups.

Results: Higher 1-year cardiovascular mortality and 1 year-advanced heart failure were remarked in the high MMP-9 group (16.9 vs. 6.4%, respectively, $p=0.010$; 18.5 vs. 8%, respectively, $p=0.001$). When the follow-up period was extended to 2 years, it was determined that the difference between the groups was statistically more serious for same parameters (21.0 vs. 7.2%, respectively, $p=0.002$; 25.0 vs. 10.4%, respectively, $p<0.001$). In a Cox multivariate analysis, a high admission MMP-9 value (≥ 12.92 ng/ml) was found to be an independent predictor of 2-year cardiovascular mortality (hazard ratio: 3.5, 95% confidence interval: 1.344-9.456, $p=0.011$).

Conclusions: These results suggest that patients high MMP-9 levels are an essential predictor cardiovascular mortality and advanced heart failure in 1 and 2-year follow-up period in STEMI patients undergoing primary PCI.

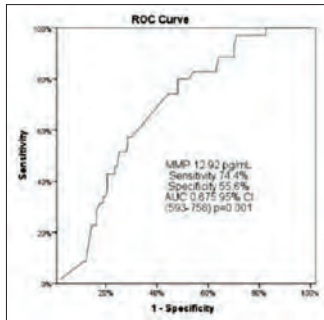


Figure 1. Receiver operating characteristics curve showing the distinguishing ability of Matrix Metalloproteinases-9 level for 2-year mortality. MMP, matrix metalloproteinases; AUC, area under the ROC curve; CI, confidence interval; ROC, receiver operating characteristics.

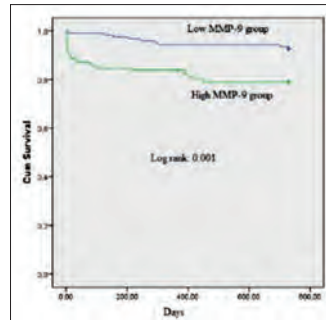


Figure 2. Kaplan-Meier analysis showing 2-year mortality ratio according to MMP-9 level. Based on the ROC curve, 12.92 ng / mL values were taken as the basis, and patients with above this value were evaluated as high MMP group and those below this group as low MMP group. MMP, Matrix metalloproteinases; cum, cumulative.

Table 1.

Variable	MMP \geq 12.92 (ng/mL) (n=124)	MMP<12.92 (ng/mL) (n=125)	P
Primary outcomes 1-year*	46 (37.1)	26 (20.8)	0.005
Secondary outcomes 1-year†			
Cardiac mortality	21 (16.9)	8 (6.4)	0.010
Non-fatal MI	14 (11.3)	9 (7.2)	0.283
TVR	23 (18.5)	15 (12.0)	0.163
Stroke	4 (3.2)	3 (2.4)	0.722
Advanced heart failure	23 (18.5)	10 (8)	0.015
Primary outcomes 2-year*	59 (47.6)	33 (26.4)	0.015
Secondary outcomes 2-year†			
Cardiac mortality	26 (21.0)	9 (7.2)	0.002
Non-fatal MI	18 (14.5)	10 (8.1)	0.113
TVR	29 (23.4)	18 (14.4)	0.077
Stroke	5 (4.0)	3 (2.4)	0.500
Advanced heart failure	31 (25.0)	13 (10.4)	<0.001

One year and two year outcomes of all study population, n (%)# Abbreviations: MMP, matrix metalloproteinases; TVR, target vessel revascularization; IABP, intra aortic balloon pump; MI, myocardial infarction * Primary clinical outcomes were composed of cardiovascular(CV) mortality, non-fatal reinfarction, target vessel revascularization(TVR), advanced heart failure, stroke † Secondary clinical outcomes were CV mortality, non-fatal reinfarction, TVR, stroke, and advanced heart failure separately ‡ All data in the table consists of percentages

Table 2.

Variables	Univariate analysis HR (CI 95%)	p	Multivariate analysis HR (CI 95%)	p
Age, years	1.088 (1.057-1.121)	<0.001	1.080 (1.040-1.122)	<0.001
Male, yes	0.496 (0.243-1.012)	0.054	1.046 (0.461-2.437)	0.915
DM, yes	1.447 (0.709-2.954)	0.311		
HT, yes	1.581 (0.813-3.074)	0.177		
HL, yes	1.028 (0.399-2.651)	0.954		
MI history, yes	1.193 (0.495-2.874)	0.694		
Killip >1, yes	4.177 (2.044-8.537)	<0.001	1.077 (0.350-3.317)	0.897
GFR	0.977 (0.966-0.989)	<0.001	0.996 (0.978-1.013)	0.622
Troponin	1.029 (1.010-1.049)	0.003	0.998 (0.973-1.024)	0.887
VT/VF, yes	4.473 (1.854-10.791)	0.001	0.939 (0.350-2.522)	0.900
LVEF	0.900 (0.871-0.931)	<0.001	0.870 (0.821-0.923)	<0.001
MMP-9 class¶	3.255 (1.525-6.947)	0.002	3.565 (1.344-9.456)	0.011

Multivariate Cox Regression Analysis for Potential Predictors of 2-Year Cardiovascular Mortality Abbreviations: CI, confidence interval; DM, Diabetes mellitus; HT, hypertension; HL, hyperlipidemia; MI, myocardial infarction, GFR, glomerular filtration rate; VT, ventricular tachycardia; VF, ventricular fibrillation; MMP, matrix metalloproteinases; LVEF left ventricular ejection fraction; GFR, glomerular filtration rate; HR, hazard ratio; ¶ The classification was made based on the cut-off value of 12.92 ng / ml obtained in ROC analysis.

Coronary artery disease / Acute coronary syndrome

OP-088

The importance of two candidate long non coding RNA expression levels on acute myocardial infarction

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Background and Aim: Acute myocardial infarction (AMI) is a major cause of mortality and morbidity in the world and in our country, mainly due to atherosclerosis. In the literature, it has been determined that several long-coding RNAs (lncRNA) are associated with cardiovascular diseases. lncRNAs are more than 200 base pairs in length. Recently, lncRNAs have important roles in regulating gene expression. In our study aim to determine the expression levels of two candidate lncRNAs in acute myocardial infarction patients and to evaluate them as new biomarkers that can be used in the diagnosis and prognosis of the disease in the acute phase.

Methods: Of the 228 subjects included in the study, 130 had acute myocardial infarction (AMI, age: 55.4±10.9 years, male; 83.1%), the group without myocardial infarction constitutes 66 had angina pectoris without MI (age: 59.2±9.4 years, male; 75.6%). The control group consisted of 32 individuals with heart valve surgery and Gensini and SYNTAX scores below 8 and 8 (age: 56.1±10.5, male %50). Total RNA was isolated in circulating peripheral blood leukocytes from all individual and cDNA synthesis was performed. The expression levels of lncRNAs BAT5 and IL21R-AS1 were analysed by the delta delta Ct method, identified by the SYBR-Green quantitative PCR. Expression levels of lncRNAs were statistically compared between the groups with SPSS-14 software.

Results: Clinical and demographic parameters of the groups are given in Table 1. There was a statistically significant difference in the expression levels of BAT5 and IL21R-AS1 lncRNAs among the research groups ($p<0.05$, Table 2). In AMI patients compared to the control group, BAT5 levels were 1.5 times higher and IL21R-AS1 levels were 3.8 times lower. Expression levels of BAT5 and IL21R-AS1 in leukocytes were found to change significantly in patients with AMI ($p=0.017$ and $p=0.008$ respectively) (Table 2).

Conclusions: In previously study was shown that increased levels of BAT5 and IL21R-AS1 expression as biomarker candidates in patients with coronary artery disease ($n=20$), however the cellular effects of these lncRNAs are unknown yet. For the first time in this study, it was shown in a larger group of research that acute myocardial infarction is associated with expression levels of both lncRNAs. This study was supported by Scientific Research Projects Coordination Unit of Istanbul University (Project number: TYL-2017-27636).

Table 1. Clinical and demographic parameters of the groups

Parameters	AMI (n=130)	Non-MI (n=66)	Control (n=32)	P value
Gender (M/F), n	108/22	50/16	16/16	0.0001
Age, year	55.4±10.9	59.2±9.4	56.1±10.5	0.055
BMI, kg/m ²	27.5±3.3	27.8±4.1	26.6±5.0	0.466
Gensini score	49.5±30.5	54.5±30.2	3.1±3.5	0.0001
SNYNTAX score	17.1±8.4	21.9±8.9	2.4±3.3	0.0001
Troponin, ng/mL	2.6±2.9	0.1±0.2	0.2±1.1	0.0001
DBP, mmHg	101.4±31.9	108.9±24.5	89.6±20.8	0.096
SBP, mmHg	98.6±26.9	107.8±20.8	105.7±40.4	0.096
Triglyceride, mg/dL	163.7±88.5	185.7±105.8	167.0±103.6	0.370
T-Cholesterol, mg/dL	183.6±45.1	196.7±48.4	194.7±44.3	0.269
HDL, mg/dL	36.9±8.9	41.9±12.7	44.1±12.3	0.002
LDL, mg/dL	123.9±40.4	129.4±43.1	124.6±37.8	0.704
CRP, mg/L	15.9±26.6	8.8±15.2	9.2±13.8	0.132
HbA1c, %	6.8±2.0	6.7±1.7	6.5±1.7	0.740
Glucose, mg/dL	128.9±41.5	114.9±43.6	117.1±40.9	0.157
PT	5.3±12.8	9.8±4.4	9.5±5.2	0.006
INR	1.1±0.3	1.3±2.2	1.0±0.1	0.647

Table 2. The expression levels of lncRNAs among groups

lncRNAs	Groups (n)	Mean±SD	P* Value	Mean±SD	P** Value
	Control (n=32)	1.4±1.8		1.8±1.8	
BAT5	Non-MI (n=66)	2.0±1.9	0.021		0.017
	AMI (n=130)	2.1±3.6		2.1±3.6	
	Control (n=18)	9.1±30.2		6.0± 17.6	
IL21R-AS1	Non-MI (n=45)	4.8± 8.9	0.030		0.008
	AMI (n=85)	2.4± 6.7		2.4± 6.7	

SD: Standard deviation, * Kruskal-Wallis Test, ** Mann-Whitney U test

Coronary artery disease / Acute coronary syndrome

OP-089

Micro RNA 199 a is impaired in patients with coronary artery disease and associated with increased levels of SIRT 1 protein and major adverse cardiovascular events

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Background and Aim: Very few studies have reported the role of micro RNAs (miRs) in risk and survey prediction of cardiovascular diseases. The cardioprotective protein SIRT1 with its anti-oxidant, anti-inflammatory and anti-apoptotic activities is compensatory increased in sera of patients with coronary artery disease (CAD). But less is known about the association of SIRT 1 and its regulating micro RNAs (miRs) on tissue level in patients with CAD and their effect on disease survey.

Methods: Sixty-three patients undergoing coronary artery bypass graft (CABG) surgery and 34 control patients undergoing heart valve surgery were recruited and biopsies were obtained from the right atrial appendage during cannulation. The expression of the SIRT 1-specific micro RNAs miR-199a and miR-195 was quantified by Real Time PCR. SIRT 1 protein was detected by Western Blot analysis. Major adverse cardiac and cerebrovascular events (MACCEs), including death, myocardial infarction (MI), re-vascularization, re-hospitalization for heart failure and stroke were analyzed at a median follow up (FU) period of 3.2 years.

Results: The relative expression of miR-199a in patients with CAD was significantly decreased compared to the control group (1.03±0.62 vs 1.92±0.90, p=0.001), whereas miR-195 was increased in patients with CAD (0.78±0.41 vs 0.006±0.003, p<0.001). SIRT 1 protein was significantly induced in tissue probes of patients with CAD (p<0.001). The MACCE rate inversely correlated with the amount of miR-199a, indicating that lower levels of miR-199a were associated with a higher risk of adverse cardiovascular events.

Conclusions: Altered expression of miR-199a in human atrial tissue was found to be mainly responsible for SIRT 1 upregulation in patients with CAD and was associated with an increased MACCE rate at follow up.

Coronary artery disease / Acute coronary syndrome

OP-090

The relationship between the severity of the coronary artery disease and soluble TWEAK/MCP-1 levels

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Background and Aim: Patients diagnosed with chronic kidney disease (CKD) have a greater rate of cardiovascular mortality compared with the general population. The soluble form of TNF-like weak inducer of apoptosis (TWEAK) and monocyte chemoattractant protein-1 (MCP-1) play important roles in cellular proliferation, migration and apoptosis. The current study aimed to investigate whether soluble TWEAK (sTWEAK) and MCP-1 levels were associated with the severity of coronary artery disease (CAD) in CKD patients.

Methods: This prospective study included 97 patients diagnosed with CKD stages 2–3, which were calculated by the simplified version of the Modification of Diet in Renal Disease formula, were included in the study (Table 1). Plasma sTWEAK and MCP-1 concentrations were determined using commercially available ELISA kits (Table 2). All patients underwent conventional coronary angiography and severity of the CAD were assessed by Gensini scoring.

Results: Correlation analysis of sTWEAK and Gensini scores showed significant association (p<0.01, r2=0.287) (Figure 1). Also significant correlation has been found in MCP-1 levels and Gensini scores (p<0.01, r2=0.414) (Figure 2). When patients were divided into two groups with a limit of 17 according to their Gensini score, sTWEAK levels indicated a statistically significant difference (p<0.01).

Conclusions: Our findings support a relationship between sTWEAK and MCP-1 levels and severity of the CAD in CKD stages 2–3 patients.

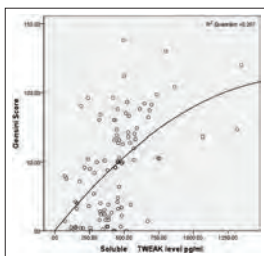


Figure 1.

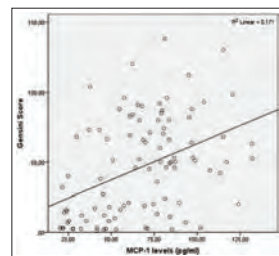


Figure 2.

Table 1. Demographic and clinical characteristics of the entire study population

	60–89	30–59	p-Value
eGFR mL/dk/1.73 m2	60.82	68.38	0.08
Age (years)	64.82(41–78)	68.38 (49–82)	0.08
Sex (M/F)	44/7	37/2	0.65
Body mass index (kg/m ²)	29.35	26.47	0.54
History of CVD	3	4	0.66
CKD etiology			
Diabetes mellitus	19	20	0.72
Hypertension	23	8	0.021
Glomerulonephritis	6	9	0.15
Unknown	3	2	0.53
Currently smoking	16	4	0.017

Table 2. Biochemical evaluation of the study population according to CKD stages

	All patients	CKD stage 2	CKD stage 3	p-Value
eGFR (mL/min)	64.18 ± 16.5	71.9 ± 8.86	50.11 ± 7	<0.01
SBP (mmHg)	133.76 ± 5.1	134.21 ± 5.13	133.84 ± 4.79	0.857
DBP (mmHg)	84.79 ± 6.41	86.07 ± 6.58	83.2 ± 5.9	0.31
Albumin (g/dL)	4.12 ± 0.42	3.9 ± 0.72	4.2 ± 0.54	0.43
Total cholesterol (mg/dL)	188.38 ± 48.88	193.68 ± 50.26	178.05 ± 45.56	0.111
Triglyceride (mg/dL)	157.8 ± 78.14	171.29 ± 90.05	135.32 ± 55.71	0.29
Low density lipoprotein (mg/dL)	116.29 ± 43.54	118.41 ± 45.01	113.23 ± 41.81	0.597
High density lipoprotein (mg/dL)	40.03 ± 11.27	41.64 ± 12.4	37.6 ± 8.8	0.112
Calcium (mg/dL)	8.52 ± 0.63	9.09 ± 0.43	8.73 ± 0.32	0.09
Phosphate (mg/dL)	5.35 ± 1.82	4.64 ± 0.83	4.84 ± 0.69	0.102
Hemoglobin (g/dL)	14.09 ± 1.75	14.55 ± 1.73	13.6 ± 1.7	<0.01
iPTH (pg/mL)	132 ± 76	72 ± 28	162 ± 53	0.086
CRP (mg/L)	6.25 ± 11.8	3.8 ± 4.5	7.99 ± 11.38	0.062
MCP-1 (pg/mL)	67.41 ± 28.11	62.56 ± 31.17	72.9 ± 22.3	0.107
sTWEAK (pg/mL)	439.29 ± 223.18	453.72 ± 240.19	435.71 ± 215.39	0.542
Gensini score	46.14 ± 35.4	41.94 ± 36.8	53.75 ± 32.4	0.047

eGFR: estimated glomerular filtration rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, iPTH: intact parathyroid hormone, CRP: c-reactive protein, sTWEAK: soluble TNF-like weak inducer of apoptosis, MCP-1: monocyte chemoattractant protein-1.

Coronary artery disease / Acute coronary syndrome

OP-091

The additive predictive role of blood urea nitrogen to the TIMI risk index in patients with ST-segment elevation myocardial infarction

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Background and Aim: The prognostic impact of Thrombolysis in Myocardial Infarction (TIMI) risk index (TRI) was reported in patients with coronary artery disease. Blood urea nitrogen (BUN) is another important prognostic factor and was associated with increased risk of mortality in patients with ST-segment elevation myocardial infarction (STEMI). In the current study, we evaluated the additional prognostic value of BUN to the TRI in patients with STEMI.

Methods: We evaluated the in-hospital mortality, cardiogenic shock and major adverse cardiac events (MACE) prognostic impact of modified TRI (mTRI) in 5018 patients with STEMI. The TRI is calculated using the following equation; TRI= (HR x [age/10]²)/systolic BP. The mTRI is calculated using the following equation; mTRI= (TRI x blood urea nitrogen)/10.

Results: During hospitalization, the incidence of all-cause death, cardiogenic shock and MACE were significantly higher in patients with higher mTRI. Similarly, the adjusted risks for all-cause death, cardiogenic shock and MACE were significantly higher in patients with higher mTRI. (OR: 3.3, 95% CI: 2.5 to 3.8, p<0.001; OR:2.8, 95% CI: 2.0 to 3.9, p<0.001, OR: 2.6, 95% CI: 2.1 to 3.2, p<0.001 respectively).

Conclusions: This investigation demonstrated that mTRI, calculated based on age, systolic blood pressure, heart rate and BUN, is an independent prognostic factor for survival of patients with STEMI. We introduce a new assessment tool by multiplying TRI with BUN/10. Via this calculation, the predictive value of TRI has been augmented. This risk index could be used as a practical tool for risk stratification patients with STEMI.

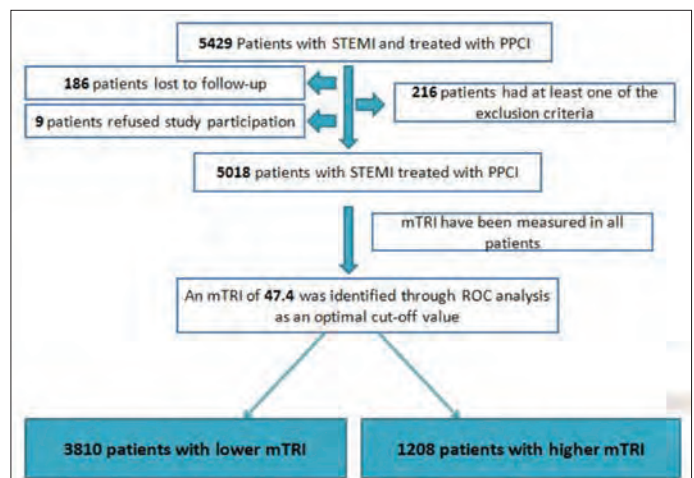


Figure 1. Study flow chart. STEMI indicates ST-segment elevation myocardial infarction; PPCI, primary percutaneous coronary intervention; mTRI, modified Thrombolysis in Myocardial Infarction risk index; ROC, receiver operating characteristic.

Table 1. Crude and adjusted in-hospital outcomes stratified by mTRI level

	mTRI <47.4 (n=3810) No. of events	mTRI ≥47.4 (n=1208) No. of events	Crude OR (95% CI)	P value	Adjusted OR (95% CI)	P value
All-cause death	127 (3.3)	188 (15.6)	5.3 (4.2 – 6.7)	<0.001	3.3 (2.5 – 3.8)	<0.001
Cardiogenic shock	94 (2.5)	101 (8.4)	3.6 (2.7 – 4.8)	<0.001	2.8 (2.0 – 3.9)	<0.001
Major adverse cardiac events	231 (6.1)	223 (18.5)	3.5 (2.8 – 4.2)	<0.001	2.6 (2.1 – 3.2)	<0.001

Abbreviations: mTRI, modified Thrombolysis in Myocardial Infarction Risk Index; OR, odds ratio. Includes sex, first measurement during hospitalization of the following laboratory values (admission glomerular filtration rate calculated by CKD-EPI, white blood cell count, hematoctrit, platelet count); admission and peak creatine kinase-MB; Killip class and left ventricular ejection fraction; acute kidney injury during hospitalization; chest pain and door-to-balloon period; culprit vessel; comorbidities (diabetes, chronic kidney disease, hypertension) and medications during hospitalization

Heart failure

OP-092

Increased inflammatory state can be a predictive marker for acute kidney injury in acute decompensated heart failure patients with preserved ejection fraction

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Background and Aim: Acute kidney injury (AKI) is an independent risk factor for mortality in acute decompensated heart failure (ADHF). The purpose of this study is to determine the predictors and prognostic impact of AKI in ADHF patients with preserved ejection fraction.

Methods: Fifty-three patients with ADHF with preserved ejection fraction (29 male; mean age 79±10 years) who were hospitalized at our intensive cardiac care unit (ICCU) during January 2016 – April 2018 were retrospectively investigated. All patients received prespecified standardized decongestive heart failure therapy as a part of "ICCU - Acute Heart Failure Treatment Protocol". AKI is defined as an increase in serum creatinine ≥0.3 mg/dL during the hospitalization.

Results: AKI developed in 37.7% of patients with ADHF with preserved ejection fraction. The baseline demographic characteristics, co-morbidities, medical therapies and laboratory analyses were similar between patients with and without AKI, except for higher N-terminal pro-brain natriuretic peptide (NT-proBNP) levels in the AKI group (13879±12534 vs 7422±8924; p=0.03) (Table 1-2). Baseline C-reactive protein (CRP) levels were increased in both groups on admission. CRP increase during hospitalization period (CRPΔ: maximal CRP – baseline CRP), but not CRP value on admission, was found to be an independent risk factor for AKI [odds ratio (OR): 1.07, 95% confidence interval (CI): 1.01–1.14, p=0.01] despite clinically diagnosed infection was not different between the groups. AKI was associated with increased length of hospital stay (9.9±10.2 vs 5.4±2.8; p=0.02) and also increased risk of in-hospital mortality (Table 2).

Conclusions: Increased inflammatory state (i.e. CRP levels) may be a marker of uncontrolled incidious infection which is associated with AKI leading to longer hospital stay and higher in-hospital mortality in ADHF patients with preserved ejection fraction.

Table 1. Baseline characteristics of patients according to the presence of acute kidney injury (AKI)

Variable	Overall n=53	AKI (-) n=33 (62.3%)	AKI (+) n=20 (37.7%)	P-value
Age (years)	79.9 ± 10.8	80.5 ± 8.8	78.9 ± 13.6	0.59
Sex (male)	29 (54.7)	16 (48.5)	13 (65)	0.27
LVEF (%)	55.3 ± 4.2	55 ± 3.7	55.8 ± 5	0.51
Coronary artery disease (n, %)	26 (49.1)	13 (39.4)	13 (65)	0.09
Hypertension (n, %)	46 (86.8)	29 (87.9)	17 (85)	0.92
Diabetes mellitus (n, %)	14 (26.4)	9 (27.3)	5 (25)	0.96
Atrial fibrillation (n, %)	33 (62.3)	23 (69.7)	10 (50)	0.24
Chronic kidney disease (n, %)	31 (58.5)	17 (51.5)	14 (70)	0.25
COPD	20 (37.7)	15 (45.5)	5 (25)	0.15
Anemia (n, %)	31 (58.5)	17 (51.5)	14 (70)	0.24
Cerebrovascular disease (n, %)	5 (9.4)	4 (12.1)	1 (5)	0.63
Peripheral artery disease (n, %)	9 (17)	6 (18.2)	3 (15)	0.89
ACEI / ARB (n, %)	26 (49.1)	14 (42.4)	12 (60)	0.26
Beta – blockers (n, %)	37 (69.8)	22 (66.7)	15 (75)	0.75
Spirolactone (n, %)	12 (22.6)	8 (24.2)	4 (20)	0.78
Furosemide (n, %)	39 (73.6)	24 (72.7)	15 (75)	0.91
Statins (n, %)	12 (22.6)	6 (18.2)	6 (30)	0.33

Table 2. Laboratory, intervention and outcome data of 53 patients diagnosed with acute decompensated heart failure preserved ejection fraction according to the presence of acute kidney injury

Variable	Overall n=53	AKI (-) n=33 (62.3%)	AKI (+) n=20 (37.7%)	P-value
BUN (baseline) (mg/dl)	47.6 ± 21.6	39.5 ± 20.4	48.2 ± 22.9	0.15
BUN (maximum) (mg/dl)	54.2 ± 28.3	42.8 ± 24.4	72.6 ± 24.8	<0.001
Creatinine (baseline) (mg/dl)	1.87 ± 1.2	1.64 ± 0.9	2.24 ± 1.6	0.08
Creatinine (maximum) (mg/dl)	2.29 ± 1.5	1.66 ± 0.9	3.32 ± 1.9	<0.001
Creatinine (delta) (mg/dl)	0.45 ± 0.7	0.07 ± 0.1	1.08 ± 1	<0.001
GFR (baseline) (ml/min/1.73 m ²)	45.3 ± 26.2	48.9 ± 27.3	40 ± 24	0.26
GFR (minimum) (ml/min/1.73 m ²)	39.2 ± 24.8	48.4 ± 25.1	23 ± 13.7	<0.001
GFR (delta) (%)	21.3 ± 24.6	10.7 ± 22.1	38.8 ± 17.8	<0.001
Hemoglobin (g/dL)	11.2 ± 2	11.4 ± 2	10.9 ± 1.9	0.39
White blood cell (K/μL)	9.36 ± 3.73	8.92 ± 3.66	10.08 ± 3.84	0.27
Albumin (g/dL)	3.5 ± 0.7	3.6 ± 0.4	3.3 ± 1	0.28
NT-proBNP (baseline) (pg/ml)	9905 ± 10816	7422 ± 8924	13879 ± 12534	0.03
CRP (baseline) (mg/L)	39.3 ± 40.9	36.5 ± 41.1	43.7 ± 41.2	0.54
CRP (maximum) (mg/L)	67 ± 66.8	48.2 ± 49.8	96.1 ± 79.7	0.01
CRP (delta) (mg/L)	27 ± 55.6	10.6 ± 17	52.4 ± 81.1	0.008
NIMV / IMV (n, %)	19 (35.8)	9 (27.3)	10 (50)	0.14
Toracemesis (n, %)	13 (24.5)	7 (21.2)	6 (30)	0.52
Infection (n, %)	24 (45.3)	13 (39.4)	11 (55)	0.39
HD / UF (n, %)	7 (13.2)	1 (3)	6 (30)	0.009
Length of stay in-hospital (days)	7.1 ± 6.9	5.4 ± 2.8	9.9 ± 10.2	0.02
Exitus (n, %)	4 (7.5)	-	4 (20)	0.01

BUN: blood urea nitrogen, GFR: glomerular filtration rate, NT-proBNP: N-terminal pro-brain natriuretic peptide, CRP: C-reactive protein, NIMV: non-invasive mechanical ventilation, IMV: invasive mechanical ventilation, HD: hemodialysis, UF: ultrafiltration

Heart failure

OP-093

Changes in plasma neprilysin levels after left ventricular assist device implantation and association with short-term outcomes

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Background and Aim: Neprilysin plasma levels tend to increase with neurohormonal activation seen in heart failure inducing degradation of natriuretic peptides which in turn increases fibrosis and decreases diuresis. After left ventricular assist device (LVAD) implantation neurohormonal activation mostly declines. However neprilysin plasma level changes after LVAD implantation is not studied before and also there is lack of data on association with pre-LVAD elevated neprilysin levels and short term outcomes after implantation. We aimed to study changes in neprilysin plasma levels after LVAD implantation and association of short term adverse events.

Methods: Patient whom had LVAD implantation procedure between January 2017 and October 2017 prospectively included in the study. Plasma Neprilysin levels measured before and 3 months after operation. Patients monitored for adverse events including mortality, stroke, pump thrombosis, gastrointestinal bleeding and right ventricular failure.

Results: Totally 47 patients (40 were male, average age was 54±11 years, BMI was 25.70±3.89 kg/m²) were included the study. 42.6% (20) of the patients had ischemic etiology. Neprilysin levels significantly decreased from 175.28 pg/ml to 75.30 pg/ml 3 months after LVAD implantation (p=0.007) Seven patients who had died after LVAD implantation has elevated neprilysin levels but weren't the statistically significant cause of limited mortality event (1798.51 pg/ml, 371.59 pg/ml p=0.134). Neprilysin levels were unrelated with age, gender, body mass index, renal function and etiology of heart failure. A cut-off value of 431 pg/ml was found to be predictive of adverse events within 3 months after the implantation with 71% sensitivity and 77% specificity (EAA: 0.682; 95% CI:0.387-0.977; p=0.128). -Patients with LVAD complication in first three months had liminal higher neprilysin levels and statistically significance (p=0.05).

Conclusions: Although neurohormonal system activation decreases after LVAD implantation, it never returns to normal. Those patients who had complications within 3 months after the operation still had high levels of neurohormonal activation. For this reason, it is recommended to continue optimal medical therapy in patients who still have high levels of neprilysin after LVAD implantation.

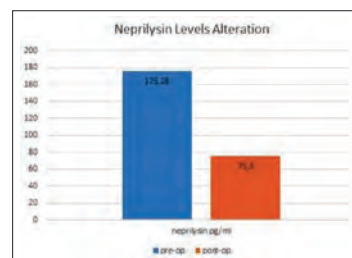


Figure 1. Neprilysin Levels Alteration.

Heart failure

OP-094

Combined post-capillary and pre-capillary pulmonary hypertension in heart transplant candidates with ischemic versus nonischemic etiology

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Background and Aim: A pulmonary hypertension (PH) is relevant complication of left ventricular heart failure. We aimed to investigate the effect of heart failure etiology (ischemic or non-ischemic) on the presence of isolated postcapillary PH (Ipc-PH) or combined pre- and postcapillary PH (Cpc-PH) or no-PH.

Methods: A total of 210 patients with end-stage heart failure undergoing evaluation for heart transplantation were stratified into two groups; namely, those with ischemic cardiomyopathy (ICMP) and nonischemic cardiomyopathy (NICMP). The patients with left ventricle ejection fraction (LVEF) \leq 25%, NYHA class III or IV and INTERMACS III and VII levels were included to study.

Results: Ninety seven patients had ICMP and 113 patients had NICMP. There was no difference in terms of left ventricular ejection fraction, NYHA, INTERMACS grades, presence of severe mitral regurgitation, left ventricular diastolic dysfunction and duration of heart failure between groups. The systolic pulmonary pressures, PAPm and PVR were higher in ICMP group compared to NICMP group [57 \pm 18 vs. 47.2 \pm 15.1, p<0.001; 35.8 \pm 11.5 vs. 30.6 \pm 10.7 p=0.001 and 3.4 (1.7-6.5) vs. 2.4 (1.0-4.4); p=0.007]. Although more Cpc-PH was found in ICMP it didn't reached statistically significance (55.6% vs. 40.7%, p=0.07). The patients with ICMP and NICMP had similar rate of Ipc-PH and no-PH (25.7% vs. 33.6%, p=0.216; and 19.5% vs. 28.3%, p=0.150; respectively). The patients with ICMP had a similar rate of PVR \geq 3 but significantly increased rate of PVR \geq 5 WU compared to NICMP (56.7% vs. 44.2%, p=0.05 and 32.9% vs. 17.6%, p=0.008). In subgroup analysis of patients who don't require inotropic treatment (170 patients), patients with ICMP had higher rate of Cpc-PH compared to NICMP (55.8% vs. 33.7%, p=0.043).

Conclusions: The ischemic versus nonischemic cardiomyopathy didn't affect the rate of Ipc-PH, Cpc-PH and No-PH. However, more Cpc-PH was determined in patients with ICM who didn't require inotropic treatment.

Heart failure

OP-095

Lead aVR is a predictor for mortality in heart failure with preserved ejection fraction patients

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Background and Aim: Our aim was to investigate the relationship between lead aVR and mortality in heart failure with preserved ejection fraction patients (HFpEF). **METHODS:** The absolute numerical values of T wave polarity (TPaVR) and ST deviation in lead aVR were recorded from the 12 lead surface electrocardiography (ECG). A ratio was obtained from the division of bigger absolute value by lesser absolute value. This proportional parameter was named as third ratio.

Methods: Twelve lead surface ECGs of all patients were recorded. Electrocardiograms had 25 mm/sec speed and 1 mv/10 mm standard calibration. These ECGs were assessed by two independent cardiologists. QRS duration and axis, fragmentation in QRS complex, P wave duration, PR interval, QT and QTc durations, the existence of positive TPaVR, ST deviation in lead aVR and quantitative TPaVR values were recorded. TPaVR and lead aVR ST deviation's absolute numerical values were recorded and a ratio was obtained from the division of bigger absolute value by lesser absolute value. We recorded EF, left ventricular end diastolic and end systolic diameters (LVDD, LVDS), left atrium diameter (LaD) from echocardiographic data. Pulsed wave E velocity, A velocity, S velocity, e' velocity, a' velocity, E/e' values were measured with tissue Doppler method and systolic pulmonary artery pressures (PAPs) were recorded.

Results: The patients were divided into two groups, living and deceased. The living group consisted of 171 patients (mean age 68.9 \pm 11.8 years, mean follow-up period 37.5 \pm 9.6 months) and the deceased group had 78 patients (mean age 75.1 \pm 11.3 years, mean follow-up period 39.9 \pm 9.2 months). In the demographic comparison, the deceased group had significantly higher mean age (p<0.001, Table 1). Both groups had similar laboratory and drug treatment findings (Table 2 and 3). LaD (p=0.021), QRS duration (p=0.044), patient count with positive T wave in lead aVR (p<0.001), ST segment deviation in lead aVR (p=0.001), TPaVR (p=0.002) ve ratio (<0.001) were significantly higher in the deceased group (Table 4). Age (OR:1.106, 95% CI:1.057-1.157, p<0.001), ST segment elevation in aVR (OR:2.156, 95% CI:1.482-3.138, p<0.001), TPaVR (OR:1.595, 95% CI:1.230-2.067, p<0.001) and ratio (OR:3.294, 95% CI: 2.306-4.073, p<0.001) were determined as independent predictors for mortality in the binominal logistic regression analysis (Table 5).

Conclusions: Lead aVR in surface ECG closely associated with mortality in patients with HFpEF.

Table 1. Comparison of patients demographic findings

	Living n=171	Deceased n=78	p
Age (years)	68.9 \pm 11.8	75.1 \pm 11.3	<0.001
Male gender, n(%)	57 (33.3)	28 (35.9)	0.692
Systolic blood pressure (mmHg)	115.6 \pm 16.1	105.9 \pm 23.2	0.04
Diastolic blood pressure (mmHg)	75.5 \pm 12.7	70.9 \pm 12.9	0.086
Pulse (beat/minute)	82.7 \pm 18.7	87.1 \pm 18.4	0.097
BMI (kg/m ²)	30.4 \pm 6.7	30.2 \pm 6.2	0.845
Smoking, n (%)	35 (20.5)	19(24.4)	0.490
DM, n (%)	60(35.1)	27 (34.6)	0.942
HT, n (%)	34 (39.9)	16 (20.5)	0.908
HPL, n (%)	3 (1.8)	0 (0)	0.554
Stroke, n (%)	6 (3.5)	5 (6.4)	0.301
CAD, n (%)	18 (10.5)	10 (12.8)	0.595
NYHA 3-4, n (%)	46 (26.9)	39 (50.0)	<0.001

BMI:body mass index, CAD:coronary artery disease, COPD:chronical obstructive pulmonary disease DM:diabetes mellitus, HT:hypertension, HPL:hyperlipidemia, NYHA:New York Heart Association.

Table 2. Comparison of patients medications

	Living n=171	Deceased n=78	p
ACE (n, %)	12 (7.0)	10 (12.8)	0.135
ARB (n, %)	12 (7.0)	5 (6.4)	0.860
B blocker (n, %)	45 (26.3)	19 (24.4)	0.743
Furosemid (n, %)	169(98.8)	77 (98.7)	0.940
Spironolactone (n, %)	53 (31.0)	20 (25.6)	0.389
Anticoagulant (n, %)	9 (5.9)	3 (3.8)	0.628
Digoxin (n, %)	6 (3.5)	3 (3.8)	0.291
ASA (n, %)	33(19.3)	18 (23.1)	0.493

ACE:angiotensin converting enzym, ARB:angiotensin receptor blocker, ASA:acetylsalicylic acid.

Table 3. Comparison of patients laboratory findings

	Living n=191	Deceased n=22	p
Glucose (mg/dl)	123.2 \pm 82.5	123.7 \pm 67.8	0.961
WBC (uL)	9.9 \pm 4.1	11.1 \pm 4.8	0.056
Hb (mg/dl)	12.2 \pm 1.6	11.8 \pm 1.5	0.065
BUN (mg/dL)	51.2 \pm 21.2	51.0 \pm 20.9	0.937
Cr (mg/dL)	1.3 \pm 0.7	1.4 \pm 0.5	0.215
Na (mmol/L)	135.7 \pm 4.5	136.9 \pm 5.2	0.073
K (mmol/L)	4.2 \pm 0.5	4.2 \pm 0.7	0.71
Gfr (mL/min/m2)	62.6 \pm 20.4	58.1 \pm 13.9	0.340
Uric acid (mg/dL)	7.3 \pm 2.4	7.4 \pm 2.7	0.827
Total protein (g/dL)	6.4 \pm 1.2	6.3 \pm 1.1	0.368
Albumin (g/dL)	3.6 \pm 0.6	3.5 \pm 1.0	0.378
Calcium (mg/dL)	8.8 \pm 0.6	8.7 \pm 0.8	0.306
Phospor (mg/dL)	4.5 \pm 0.6	4.9 \pm 3.5	0.817
Hs-CRP (mg/L)	2.6 \pm 2.7	3.3 \pm 3.1	0.109
NT-proBNP (pg/mL)	4033 \pm 6937	6325 \pm 9686	0.101
Hs-TrfT (pg/L)	0.4 \pm 1.4	1.5 \pm 7.9	0.247

WBC:white blood cells, Hb:hemoglobin, BUN:blood urea nitrogen, Cr:creatinin,Hs-CRP:high sensitive C reactive protein, NT-proBNP: N-terminal brain natueric peptide.

Table 4. Comparison of patients echocardiographic and electrocardiographic findings

	Living n=171	Deceased n=78	p
EF (%)	55.2 \pm 5.3	54.7 \pm 4.8	0.375
LAD (mm)	44.6 \pm 4.1	45.9 \pm 3.7	0.021
E velocity (cm/s)	89.3 \pm 23.9	91.2 \pm 20.5	0.344
A velocity (cm/s)	60.7 \pm 21.0	61.8 \pm 21.4	0.692
S velocity (cm/s)	7.2 \pm 1.9	7.3 \pm 1.9	0.804
e' velocity (cm/s)	7.1 \pm 1.9	7.0 \pm 2.0	0.792
a' velocity (cm/s)	4.1 \pm 1.7	3.9 \pm 1.7	0.217
E/e'	13.4 \pm 5.1	13.9 \pm 4.7	0.415
PAPs (mmHg)	33.2 \pm 8.6	32.2 \pm 7.9	0.379
QRS (msn)	87.9 \pm 18.4	93.8 \pm 25.9	0.044
QRS axis (°)	20 \pm 45.9	60.1 \pm 86.5	0.066
Fragmentation, n (%)	30 (17.5)	18 (20.5)	0.576
P duration (ms)	91.2 \pm 7.4	87.9 \pm 6.3	0.013
PR interval (ms)	160.7 \pm 20.2	159.8 \pm 30.5	0.846
QT (ms)	386.2 \pm 52.7	383.2 \pm 60.0	0.699
QTc (ms)	440.4 \pm 37.8	447.5 \pm 47.5	0.219
Positive TPaVR, n (%)	39 (22.8)	36 (46.2)	<0.001
ST deviation in lead aVR (mm)	0.5 \pm 1.0	1.1 \pm 1.1	0.001
TPaVR (mV)	-0.6 \pm 1.3	0.1 \pm 1.5	0.002
Ratio (n)	2.1 \pm 1.2	4.3 \pm 0.4	<0.001

EF:ejection fraction, LAD:left atrium diameter, PAPs:systolic pulmonary artery pressure, TPaVR:T wave polarity in lead aVR.

Table 5. Independent predictors for mortality in patient with Heart failure preserved EF

	Odds ratio	95% Confidence Interval	p
Age	1.106	1.057-1.157	<0.001
NYHA (for each unit increase)	0.513	0.276-954	0.035
QRS duration	0.997	0.977-1.017	0.748
LAD	1.088	0.992-1.193	0.075
ST deviation in lead aVR (mm)	2.349	1.498-3.684	<0.001
TPaVR (mV)	1.612	1.183-2.196	0.002
Ratio	5.156	3.141-8.465	<0.001

LAD:left atrium diameter, TPaVR: T wave polarity in lead aVR, HFpEF:heart failure preserved ejection fraction,NYHA:New York Heart Association.

Heart failure

OP-096

Acute decompensated heart failure: A risk factor for silent cerebral ischemia?

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Background and Aim: Magnetic resonance imaging studies demonstrated that substantial number of patients with chronic heart failure have silent cerebral infarcts (SCI), defined as evidence of cerebral damage in the absence of clinically apparent stroke or transient ischemic attack. Serum neuron specific enolase (NSE) is a sensitive neuronal ischemia marker, which increases 2 hours after the neuronal damage. Our aim is to evaluate the role of acute decompensation on SCI in patients with chronic reduced ejection fraction heart failure (HFpEF).

Methods: We consecutively recruited 75 patients with HFpEF, who were admitted with acute decompensation. Blood samples for NSE were collected on the day of admission. Patients were treated according to the recent ESC heart failure management guidelines. After reaching the compensated state, second blood samples for NSE were collected. Elevation of NSE >12 ng/ml was considered as SCI.

Results: Mean age of the study population was 72.4 \pm 9.4 and 52% of them were male. On admission, 29% (22/75) of the patients with acute decompensation had NSE elevation. All patients were compensated after \pm 2 days of treatment and only 4% (3/75) of them had elevated NSE levels after compensation (p<0.0001) (Figure 1). Multivariate predictors of NSE elevation during acute decompensation were; being a current smoker (OR:23.281 (2.250-240.842), p=0.008), lower oxygen saturation at admission (OR: 1.264 (1.536-1.041), p=0.02), presence of spontaneous echo contrast in echocardiography (OR: 17.626 (1.905-163.076), p=0.01) and the presence of apical aneurysm (OR: 9.853 (1.514-64.130), p=0.02).

Conclusions: Our data show that, almost one third of patients with acute decompensation had NSE elevation. This may help to explain the increased prevalence of SCI in patients with chronic heart failure.

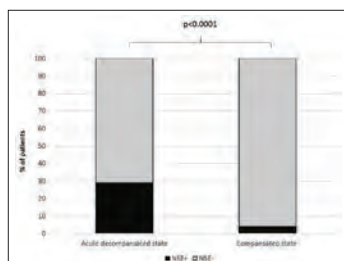


Figure 1. Comparison of the NSE positivity of the 75 patients according to the compensation state of the heart failure.

Heart failure

OP-097

Lowered mid-regional pro-adrenomedullin in response to levosimendan therapy is associated with improved survival in patients with acutely decompensated heart failure

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Background and Aim: Levosimendan treatment for acute decompensated heart failure failed to show survival benefit over placebo or dobutamine. Novel biomarkers such as MR-proADM can help us to identify some subgroup of patients that have survival benefit with levosimendan treatment.

Methods: A total of 61 patients with acutely decompensated heart failure with New York Heart Association (NYHA) class III or IV symptoms were randomized to receive either levosimendan or dobutamine 2:1 in an open-label fashion. Before and 5 days after the initiation of infusions, functional class was assessed, N-terminal prohormone of B-type natriuretic peptide (NT-proBNP), mid-regional pro-adrenomedullin (MR-proADM) levels and left ventricular ejection fraction (LVEF) were measured. After discharge patients were followed-up for 24 months for all-cause mortality.

Results: Levosimendan infusion did not cause survival benefit over dobutamine both for short and long-term follow up. Mortality was 19.7% at 90 days and 62.3% at 24 months. HF etiology, NYHA and MR-proADM, but not NT-proBNP, was an independent predictor of 24 month mortality (p=0.002, p=0.010, p<0.001 respectively). When the effects of changes in laboratory values on 24-month mortality were evaluated, LVEF and MR-proADM ratio were found as an independent predictor of mortality (p=0.006, p=0.012 respectively). Besides, MR-proADM responders due to levosimendan therapy was associated with 7.4 times improved mortality compared to non-responders.

Conclusions: Before initiation of infusion value of MR-proADM and change with treatment is a better biomarker than NT-proBNP for identification of patients at high risk of death in advanced chronic heart failure. Also, levosimendan induced a decrease in MR-proADM is associated with improved survival.

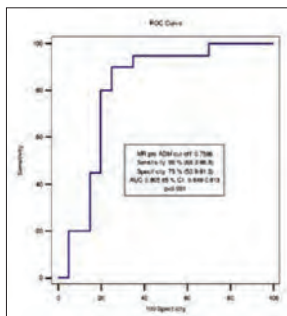


Figure 1. Receiver operating characteristics curve showing the distinguishing ability of MR pro-ADM ratio for 2-year mortality for levosimendan therapy. MR pro-ADM ratio was found by the ratio of the value of 5th day after treatment to the value of first day before treatment.

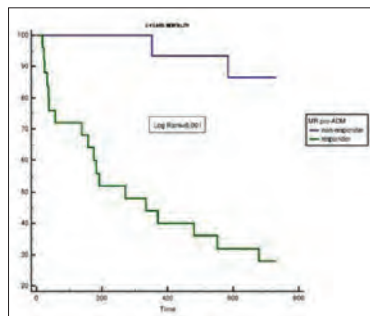


Figure 2. 2-year mortality ratio according to MR pro-ADM response in levosimendan therapy group. Based on the cut-off value of 0.75 in the ROC curve, patients with a MR-proADM value reduction of more than 25% were evaluated as responder.

Table 1.

Variables	Levosimendan before	Levosimendan after	Dobutamine before	Dobutamine after	F	P
SBP (mmHg)	116.5±22.9	110.1±17.6	115.4±23	110.4±14.9	2.58	0.11
DBP (mmHg)	68.0±12.3	65.5±12.6	65.2±12	65.1±11.3	0.571	0.45
Heart Rate (beats/min)	77(66-83)	77(67-83)	75(66-82)	77(69-82)	0.58	0.45
Creatinine (mg/dl)	1.2(1.0-1.4)	1.3(1.0-1.5)	1.3(1.1-1.8)	1.5(1.1-1.8)	0.06	0.81
NYHA class	3 (3-4)	2 (2-3)	3 (3-3)	3 (3-4)	31.95	<0.001
WBC(x1000/mm ³)	8.3±2.3	7.7±1.9	8.6±2.7	8.3±3.4	2.25	0.14
Sodium (mEq/L)	135.6±3.7	131.7±5.0	136.2±5.2	133.4±5	27.51	<0.001
LVEF(%)	24.5±5.4	30.1±4.4	26.1±5.5	27.4±5.2	32.286	<0.001
NT-proBNP(pg/ml)	7099 (2754-12100)	3484 (2020-5647)	4130 (2168-9060)	3830 (1768-18653)	10.881	0.002
MR-proADM (nmol/L)	5.5(3.4-7.3)	4.2(2.4-6.0)	3.6(2.5-7.3)	3.5(2.5-6.5)	12.041	0.001

Clinical and laboratory parameters before and after treatment in the levosimendan and dobutamine groups

Table 2.

	90 day			24 month		
	univariate	multivariate		univariate	multivariate	
	HR	CI(%95)	P	HR	CI(%95)	P
First model						
Etiology, ischemic	2.926	.379-22.738	0.303			
Therapy type	1.293	.410-4.074	0.664	1.485	.759-2.945	0.257
SBP(mmHg)	0.994	.969-1.020	0.661	.988	.973-1.004	0.141
DBP(mmHg)	1.021	.971-1.075	0.416	1.011	.982-1.042	0.445
Hr (beats/min)	.989	.947-1.034	0.638	.989	.964-1.014	0.394
Age	1.035	.975-1.110	0.259	1.022	.982-1.057	0.006
Kreatinine(mg/dl)	2.955	1.029-8.483	0.044	2.955	1.029-8.483	0.044
Sodium(mEq/L)	1.002	.872-1.152	0.976	.932	.834-1.006	0.105
MRproADM (nmol/L)	1.117	.986-1.265	0.083	-	-	-
NT-proBNP(pg/ml)	1.000	1.000-1.000	0.38	-	-	-
LVEF(%)	.915	.821-1.020	0.110	.972	.914-1.033	0.361
NYHA class	.597	.131-2.727	0.506	.435	.168-1.126	0.086
Second Model*						
MR-proADM ratio†	3.659	.657-20.382	0.139	-	-	-
NT-pro BNP ratio†	1.154	.589-2.260	0.677	-	-	-
LVEF ratio†	14.397	1.391-149.054	0.25	17.869	1.186-175.836	0.013
				7.325	1.533-35.009	0.013
				11.869	2.001-70.392	0.006

Univariate and multivariate analysis for predictors of mortality at 90 Day and at 24 months †In the first model, the values at the time of admission to the hospital were included in the analysis. *In the second model, mr-proadm, nt bnp, and hvaf rates are substituted for mr-pro-adm nt-bnp and hvaf basal values. Ratio was calculated by dividing the values of the 5th day after the treatment into hospital admission values.

Coronary artery disease / Acute coronary syndrome

OP-098

Left main coronary intervention via radial approach: A single center experience

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Background and Aim: Left main disease is considered as a distinct group in management of coronary artery disease due to its heterogeneity, complexity and also during percutaneous intervention it poses high procedural risks. In this study we wanted share our single center experience in percutaneous intervention of left main disease with radial approach.

Methods: From January 2009 to May 2018, 69 consecutive patients who underwent percutaneous left main coronary intervention with radial approach in Yeditepe University Hospital were included in our study. Demographical, clinical and procedural data were obtained from hospital records and patient files. Procedural data included total treated lesion number, total implanted stent number, number of stent per lesion, total implanted stent length, total treated lesion length, minimum and maximum stent diameters and stent lengths, median stent diameter and stent length, median lesion diameter and lesion length. Interventions other than left main (whether angioplasty or stent implantation) at the same setting were excluded. Indication for intervention was defined as stable coronary heart disease or acute coronary syndrome which included unstable angina pectoris, non ST elevated myocardial infarction or ST segment elevated myocardial infarction. Procedural data were compared between cardiovascular risk factor and indication groups using independent samples T-test or one way ANOVA.

Results: Mean age was 65.32±11.39 (max: 92 min:31), 58 patients (84.1%) were male and 22 patients (31.9%) were diabetic. 43 patients (62.9%) had a history of coronary heart disease. Demographical and clinical features were shown in Table 1. Majority of cases (81%) were performed via 6 French sheath. In 19 procedures (27.5%) stent implantation was performed both in left anterior descending and circumflex arteries. Mean implanted stent number was 1.54±0.584 (max:3 min:1), mean treated lesion number was 1.59±0.69. Mean number of implanted stent per lesion was 1.02±0.33. Mean stent length and diameter per procedure was 21.59±6.89 and 3.27±0.43 respectively. All procedural data was depicted in Table 2 No significant difference

regarding procedural data was found across gender, cardiovascular risk factor and indication groups.

Conclusions: Left main coronary intervention via radial approach is feasible and safe. Follow up data will be presented in the corresponding meeting.

Table 1.

Mean age (±SD)	65.32±11.39
Male gender- n(%)	58 (84.1)
Diabetes n(%)	22 (31.9)
History of CAD n(%)	43 (62.9)
Stable CAD n(%)	45 (65.2)

Demographical and clinical data of study population.

Table 2.

No of implanted stents	1.54±0.58
No of treated lesions	1.59±0.69
No of stents per lesion	1.02±0.33
Maximum stent length (mm)	23.22±6.93
Minimum stent length (mm)	19.96±7.61
Maximum stent diameter (mm)	3.46±0.51
Minimum stent diameter (mm)	3.08±0.49
Mean stent diameter (mm)	3.27±0.43
Mean stent length (mm)	21.59±6.89
Total stent length (mm)	32.71±13.57
Total lesion length (mm)	30.28±12.82
Mean lesion diameter (mm)	3.12±0.44
Mean lesion length (mm)	20.12±6.46

Procedural data (per procedure) (mean±SD).

Coronary artery disease / Acute coronary syndrome

OP-099

Premature myocardial infarction: Between genetic susceptibility and life style induced risk factors

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Background and Aim: Premature myocardial infarction (PMI) will probably rise, as lifestyle is changing characterized by work stress, smoking and overeating. Thus identifying genetic susceptibility, which modifies the cardiovascular disease (CVD) phenotype, might be helpful, to initiate comprehensive prevention very early in young people at risk. SIRT1 single nucleotide polymorphisms (SNPs) have been frequently found in older patients, who were screened positive for coronary artery disease (CAD) or suffering from myocardial infarction (MI), indicating that variations of this gene influence the CVD phenotype. The aim of this study was to investigate the association between SIRT1 SNPs, SIRT1 and eNOS (endothelial nitric oxide synthase) protein expression as well as common cardiovascular risk factors and major adverse cardiac events (MACEs) in young patients suffering from premature ST-elevation myocardial infarction (STEMI).

Methods: Genotyping of the three SNPs (rs7895833 A>G in the promoter region, rs7069102 C>G in intron 4 and rs2273773 C>T in exon 5) in SIRT1 gene was performed in 108 consecutive patients (87.0% were men with a mean age of 40.74±3.82 years) suffering from STEMI at the age of ≤45 and 91 control subjects. Protein levels were detected by ELISA.

Results: SIRT1 protein levels were enhanced and eNOS levels were decreased in MI patients regardless of the underlying gene variant. Besides SIRT1 protein expression was correlating with age in patients, but not in controls, suggesting that SIRT1 induction is disease- and age- related. The risk for PMI was increased by 1.96 times in carriers of the CC or CG genotypes of rs7069102 C>G. This was more evident in patients with age less than 40 years. Recurrent MI was observed in 12 cases and all patients had their first coronary event under the age of 40. SIRT1 protein levels were enhanced compared to the patients without an adverse event, while eNOS levels were similar. Furthermore, a negative correlation was detected between recurrent MI and Left Ventricular Ejection Fraction (LVEF) as well as between SIRT1 values and LVEF.

Conclusions: SIRT1 SNPs are associated with premature MI, affecting SIRT1 protein expression and subsequently the patients' clinical profile and disease survey.

Coronary artery disease / Acute coronary syndrome

OP-100

The value of heat shock protein (HSP) 60 on in-hospital and short-term prognosis in patients with acute ST segment elevation myocardial infarction

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Background and Aim: HSP60 has previously been shown to be an independent risk factor for congestive heart failure. Recently, increased HSP60 values have been shown to be associated with increased mortality in patients hospitalized with acute heart failure. Our aim was to determine whether HSP 60 was an independent predictor of in-hospital or short-term mortality in patients with acute ST segment elevation myocardial

infarction (STEMI) and to assess whether it could be used as an early predictor of in-hospital heart failure or cardiogenic shock development in the same population.

Methods: Patients over 18 years of age presenting within 12 hours after the onset of symptoms with ASTEMI. Ten cc blood was drawn into a gel-filled tube at admission which were centrifuged at 3000 rpm for 5 minutes. The supernatant was immediately aliquoted and stored at -80 °C. Several clinical conditions, such as clinical heart failure, development of cardiogenic shock and death during hospitalization were recorded together with the data pertaining to the length of hospital stay, MI localization, revascularization strategy and risk factors associated with coronary heart disease. Patients were called 1 month after discharge to find out if anyone had died.

Results: The ages of all 221 patients ranged between 25 and 89 years. Mean age was found to be 59.8±14.6. Of all the patients, 176 (80) were male and 45 (20%) were female. The mean age of the male and the female patients were 57.69 and 68.57, respectively (Table 1). Respective incidence of hypertension, diabetes mellitus, hyperlipidemia and history of smoking were 40.5%, 18.6%, 13.2% and 70.9%. Of all patients, 85% was admitted with Killip class 1. GpIIb/IIIa antagonistic agent (tirofiban) was used in 33.2% of the patients. (Table 2) HSP 60 values were significantly higher in patients who died during hospitalization (p<0.05) or within 1 month after discharge (p<0.05) (Table 3). These results were independent from other factors associated with mortality following ASTEMI. However, no significant association was found between HSP60 values and in-hospital development of cardiogenic shock (p>0.05) or heart failure (p>0.05). ROC curve analysis was utilized in an attempt to specify a potential cut-off point of HSP60 to be used in the determination of mortality, which yielded 7.325 value as cut-off point (Figure 1).

Conclusions: Our study showed that HSP60 possess a prognostic significance in the prediction of mortality in patients presenting with acute STMI within 12 hours of onset symptom. HSP60 was found to be independently associated with increased mortality in patients admitted for ASTEMI and therefore may be used as a promising predictor of mortality. However, the fact that our study recruited a relatively small group of patients may hinder true interpretation of our findings. Hence, future studies including larger groups of patients are needed to confirm them.

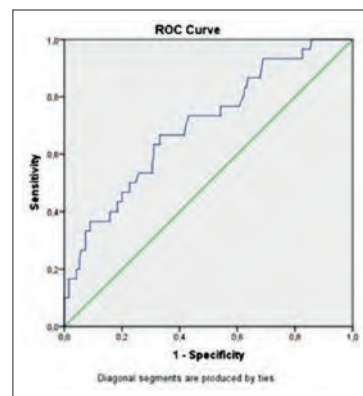


Figure 1. ROC Curve in the determination of Mortality.

Table 1. Clinical and demographic characteristics of the patients

Mean Age	59.8 ± 14,6
Hypertension	%40,5
DM	%18,6
Hyperlipidemia	%13,2
Smoking	%70,9
Killip class 1	%85
GpIIb/IIIa antagonist use	%33,2

Table 2. Relationship between mortality and HSP60 values

	n	p
In-Hospital Mortality	21	0,004
One-Month Mortality	9	0,020

Table 3. Clinical and demographic characteristics of the patients

	WOMEN	MEN
n	45(%20)	176(%80)
Mean Age	68.57	57.69

Coronary artery disease / Acute coronary syndrome

OP-101

ST segment and T wave changes in lead aVR is related to left ventricle thrombus and grade 3-4 spontaneous echo contrast in acute anterior myocardial infarction patients

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Background and Aim: Left ventricular thrombus (LVT) is accepted as a major complication after AAMI. Lead aVR gives unique and useful informations alone in surface electrocardiogram (ECG). Our aim was to investigate the relation between changes in lead aVR and LVT or grade 3-4 spontaneous echo contrast (SEC) in patients with AAMI.

Methods: We retrospectively examined and enrolled 144 AAMI patients. Admission and 48th hour ECGs were recorded. ST segment elevations in V1,V2,V5,V6 were recorded from admission ECGs. QRS duration, P wave duration, PR interval, QT and QTc durations, T polarity in lead aVR (TPaVR) and ST deviation in lead aVR (STaVR) were measured from 48th hour admission ECG (figure 1a and 1b). Absolute value of TPaVR and STaVR were calculated. A ratio was obtained from division of larger absolute value by smaller one. Syntax score (SS), clinical SS (cSS), and residual SS (rSS) were calculated. Echocardiography was performed in all patients.

Results: There were 22 patients (mean age: 58.1±11.0 years) with LV thrombus and grade 3-4 SEC and this patients named as group 1. 15 patients had LV thrombus (10.4%) and 7 patients had grade 3-4 SEC. There were 122 patients (mean age: 57.1±12.1 years) who did not have any LV thrombus or grade 3-4 SEC and this

patients named as group 2. In the comparison of demographic findings, smoking frequency was significantly higher in the group 2 (p=0.043). Other demographic variables were similar (Table 1). Creatinine (p=0.008) and hs-CRP (p=0.022) levels were significantly higher in the group 1. WBC levels (p=0.032) were significantly higher in the group 2 (Table 2). Electrocardiography and echocardiography findings comparison was showed that V2 ST segment elevation (p=0.029), TPaVR (p<0.001), and ratio (p<0.001) were significantly higher in the group 1. Ejection fraction (p<0.001) was significantly lower in the group 1 (Table 3). Syntax score (p=0.012) and cSS (p=0.023) were significantly higher in the group 1. Residual SS (0.007) was significantly higher in group 2 (Table 4). Binominal logistic regression analysis was performed with the significant variables. Ratio (OR:1.613, 95% CI:1.239-2.101, p<0.001) and EF (OR:0.824, 95% CI:0.746-0.910, p<0.001) were detected as independent predictors for the presence of grade 3-4 SEC or LV thrombus.

Conclusions: Ischemic changes in lead aVR should be closely monitored about the LVT formation and grade 3-4 SEC presence in AAMI patients. If there is a need, an early echocardiographic evaluation ought to be performed.

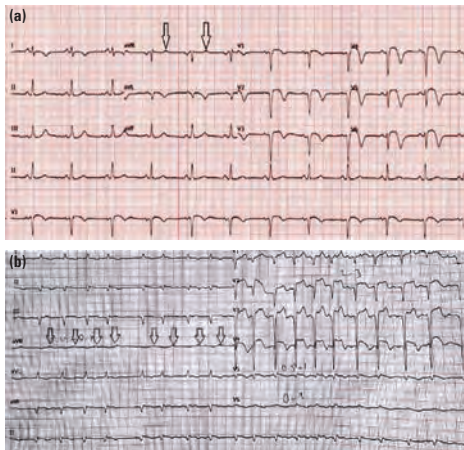


Figure 1. (a) Positive T wave in lead aVR on surface ECG in patient with left ventricular thrombus (arrows). (b) Positive T wave in lead aVR on surface ECG in patient with grade 3-4 SEC (arrows).

Table 1. Comparison of patients demographic findings

	Group 1 (n=22)	Group 2 (n=122)	p
Age (years)	58.1 ± 11.0	57.1 ± 12.1	0.733
Male gender, n(%)	18 (81.8)	106 (86.9)	0.527
Systolic blood pressure (mmHg)	121.4 ± 14.1	119.6 ± 19.0	0.724
Diastolic blood pressure (mmHg)	77.1 ± 10.6	77.4 ± 10.9	0.127
BMI (kg/m ²)	29.2 ± 4.6	27.8 ± 3.5	0.374
Smoking, n (%)	6 (27.3)	62 (50.8)	0.042
DM, n (%)	8 (36.4)	40 (32.8)	0.743
HT, n (%)	6 (27.3)	36 (29.5)	0.832
HPL, n (%)	0 (0)	12 (9.8)	0.124

DM:diabetes mellitus, HT:hypertension, HPL:hyperlipidemia.

Table 2. Comparison of patients laboratory findings

	Group 1 (n=22)	Group 2 (n=122)	p
Glucose (mg/dl)	182.1 ± 76.5	190.1 ± 116.0	0.733
WBC (uL)	10.4 ± 2.4	12.1 ± 3.4	0.032
Hb (%)	13.3 ± 2.4	13.8 ± 1.6	0.322
BUN (mg/dL)	50.2 ± 35.1	35.8 ± 16.7	0.086
Cr (mg/dL)	1.1 ± 0.3	0.9 ± 0.2	0.008
Na (mmol/L)	135.5 ± 2.7	136.4 ± 3.1	0.214
K (mmol/L)	4.4 ± 0.6	4.2 ± 0.5	0.123
Total cholesterol (mg/dL)	186.7 ± 41.8	183.3 ± 39.5	0.735
LDL (mg/dL)	123.31 ± 34.5	119.8 ± 31.5	0.684
HDL (mg/dL)	38.1 ± 8.7	38.5 ± 7.6	0.864
Triglyceride (mg/dL)	135.3 ± 65.9	138.2 ± 60.1	0.847
Hs-CRP (mg/L)	7.1 ± 8.1	2.7 ± 4.5	0.022
Uric acid (mg/dL)	5.1 ± 1.8	5.6 ± 1.4	0.219
NT-proBNP (pg/mL)	10598 ± 12899	3043 ± 4216	0.013
Hs-TnT (pg/l)	32.4 ± 59.5	21.4 ± 27.4	0.407

BUN: blood urea nitrogen, Cr:creatinin, HDL:high density lipoprotein, Hs-CRP:high sensitive C reactive protein,Hs-TnT: high sensitive troponin T, Hb:hemoglobin, LDL:low density lipoprotein TSH:thyroid stimulation hormone, NT-proBNP: N-terminal brain natuoretic peptide WBC:white blood cells

Table 3. Comparisons of electrocardiographic and echocardiographic findings

	Group 1 (n=22)	Group 2 (n=122)	p
TTE performed time, n (days)	3.9±1.3	3.7±1.4	0.514
EF (%)	37.0 ± 8.3	43.8 ± 7.5	<0.001
LVDD (mm)	39.1±18.8	45.6±7.3	0.313
LVDS (mm)	29.7±17.0	34.8±4.6	0.429
QRS duration (ms)	103.8±10.1	98.4±13.5	0.731
P wave duration(ms)	76.7±17.5	74.6±19.1	0.08
PR interval (ms)	151.3±24.2	143.0±15.1	0.132
QT interval (ms)	378.7±48.8	374.3±43.3	0.667
QTc interval (ms)	438.4±26.6	435.7±37.0	0.744
V1 ST segment elevation (mm)	1.1±0.6	1.1±0.7	0.892
V2 ST segment elevation (mm)	3.3±1.0	2.8±1.8	0.029
V5 ST segment elevation (mm)	2.0±0.9	1.7±1.5	0.253
V6 ST segment elevation (mm)	1.3±0.9	1.0±1.1	0.26
STaVR (mm)	0.5±0.3	0.6±0.4	0.08
TPaVR (mV)	0.6±1.3	-0.9±1.7	<0.001
*Ratio	6.2±3.6	3.3±2.5	<0.001

EF:Ejection fraction, LVDD:Left ventricular diastolic diameter, LVDS: Left ventricular systolic diameter, TTE:Trans thoracic echocardiography, TPaVR:T wave polarity in lead aVR, STaVR: ST segment deviation in lead aVR *Ratio: The absolute value of bigger one (STaVR or TPaVR)/ The absolute value of smaller one (STaVR or TPaVR).

Table 4. Comparisons of patients angiographic findings

	Group 1 (n=22)	Group 2 (n=122)	p
SS, n	19.1±5.5	15.5±7.4	0.012
cSS, n	38.1±17.3	28.8±17.3	0.023
rSS, n	1.5±1.6	3.5±6.8	0.007
Door-balloon time (hours)	32.2±5.2	31.4±10.6	0.835
Contrast volume (ml)	167.7±57.7	143.1±49.4	0.068
Abciximab or tirofiban infusion, n (%)	5 (22.7)	23 (18.9)	0.673

SS:Syntax score, cSS:Clinical syntax score, rSS:Residual syntax score.

Coronary artery disease / Acute coronary syndrome

OP-102

The relationship between CRP/albumin ratio and late left ventricular thrombus after first acute anterior ST elevation myocardial infarction

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Background and Aim: The aim of this study is to investigate relationship between CRP/Albumin ratio (CAR) and late left ventricular (LV) thrombus formation in patients with first acute anterior ST elevation myocardial infarction (STEMI) who have undergone primary percutaneous coronary intervention (p-PCI).

Methods: This single-center retrospective study included 1,272 patients with first acute anterior STEMI who have undergone p-PCI. These patients were enrolled after discharge and were followed up for 4 weeks from January 2012 to January 2018. 896 patients who met the inclusion criterias were divided into two groups based on presence and absence of LV thrombus. All patients underwent an echocardiogram before discharge from the hospital, as well as a month later.

Results: The study population comprised 896 STEMI patients with a mean age of 61.6±11.7 years; 71.7% (642) of the subjects were men. Left ventricular thrombus was detected in 76 (8.5%) patients. The subjects were divided into two groups according to presence of LV thrombus; as patients with LV thrombus (76), and those without LV thrombus (820). Increased age, diabetes mellitus, door-to-balloon time, time from symptom onset to PCI, neutrophil count,CRP and decreased LVEFpredischarge/postdischarge, hemoglobin, albumin level, were observed in patients with LV thrombus as compared to those without LV thrombus (Table 1). The mean CAR of the study population was 0.27 (0.09-0.50). CAR was increased in patients with LV thrombus than those without LV thrombus [0.32 (0.1-0.53) vs 0.24 (0.09-0.45); p<0.001]. To identify the independent predictors of LV thrombus, multivariate logistic regression analyses with a stepwise backward model were performed using the variables in the univariate analyses including age, sex, door-to-balloon time, time from symptom onset to PCI, diabetes mellitus, LVEFpredischarge, hemoglobin, neutrophil, albumin and CAR. CAR (OR:1.53, 95% confidence interval [CI]:1.39-1.64, p<0.001), neutrophil (OR:1.63, 95% CI:1.24-1.75, p<0.001), LVEFpre (OR: 0.83, 95% CI 0.76-0.88, p <0.001) were found to be independent predictors of LV thrombus (Table 2). The optimal cut-off values of CAR for predicting LV thrombus > 0.367 with a sensitivity of 49% and a specificity of 81% (area under the curve[AUC]: 0.624; 95%CI: 0.564-0.651).

Conclusions: Our study demonstrated that CAR calculated from the admission blood samples could be a useful parameter for predicting LV thrombus in patients with anterior STEMI after the discharge.

Table 1. Demographic, clinical, angiographic, echocardiographic characteristics and laboratory parameters of patients with and without LV thrombus

Age (year)	61.4±11.9	63.7±7.2	0.039
Male[n (%)]	594 (72.4)	48 (63.2)	0.08
Hypertension [n (%)]	226 (27.6)	23 (30.3)	0.61
Diabetes mellitus [n (%)]	146 (17.8)	21 (27.6)	0.03
Current smoking [n (%)]	332 (40.5)	30 (39.5)	0.86
Family history of CAD [n (%)]	179 (21.8)	14 (18.4)	0.49
History of hyperlipidemia[n (%)]	130 (25.1)	12 (24.0)	0.68
Obesity [n (%)]	99 (12.1)	13 (17.1)	0.20
Previous PCI [n (%)]	88 (10.7)	8 (10.5)	0.95
Time from symptoms onset to PCI (hour)	4.36±1.51	5.23±1.77	0.014
Door to balloon time (minute)	42.9±6.7	44.5±5.6	0.005
Postprocedural TIMI flow			0.40
Unsuccessful (TIMI 0-1)[n (%)]	73 (8.9)	9 (11.8)	
Successful (TIMI 2-3) [n (%)]	747 (91.1)	67 (88.2)	
LV EF pre-discharge (%)	36.1±4.7	32.1±3.7	<0.001
LV EF post-discharge (%)	41.4±5.1	37.2±3.9	<0.001
Heart rate [/min]	102±18	98±14	0.36
Hemoglobin [g/dl]	12.3±1.7	13.7±1.5	0.01
White blood cell count [103/μl]	12.4±3.4	11.2±3.0	0.74
Platelet count [103/μl]	267±74	271±58	0.26
Neutrophil count [103/μl]	10.5±3.4	9.2±2.8	<0.001
Lymphocyte count [103/μl]	1.7±1.5	1.7±1.2	0.82
Fasting blood glucose [mg/dl]	148.2±66.3	144.5±61.5	0.24
C-reactive protein [mg/l]	9.3 (4.9-16.1)	7.2 (4.1-14.2)	<0.001
Albumin [g/dl]	3.6 (3.5-4.0)	3.8 (3.5-4.1)	<0.001
CAR	0.32(0.1-0.53)	0.24(0.09-0.45)	<0.001

Data are expressed as mean ± SD for normally distributed data or count (percentage) for categorical variables; CAD, Coronary artery disease; DES, Drug-eluting stent; EF, Ejection fraction; LV, Left ventricle; PCI, Percutaneous coronary intervention; TIMI, Thrombolysis In Myocardial Infarction

Table 2. Univariate and multivariate logistic regression analysis for LV thrombus

Variable	Univariate			Multivariate		
	Unadjusted OR	95% CI	p value	Adjusted OR	95% CI	p value
Age (year)	1.02	0.99-1.04	0.09			
Male sex	0.84	0.47-1.54	0.48			
Diabetes Mellitus	1.74	1.06-3.12	0.07			
Obesity	1.42	0.72-2.53	0.32			
LV EF pre-discharge (%)	0.84	0.72-0.89	<0.001	0.83	0.76-0.88	<0.001
Door to balloon time (minute)	1.02	1.01-1.08	0.048			
Time from symptoms onset to PCI (hour)	1.28	1.03-1.68	<0.001			
Albumin	1.43	1.17-1.53	0.05			
CAR	1.82	1.65-1.96	<0.001	1.53	1.39-1.64	<0.001
Hemoglobin	1.35	1.14-1.38	0.24			
Neutrophil	1.72	1.56-1.84	<0.001	1.63	1.54-1.75	<0.001

CI=confidence interval; OR=odds ratio; CAR, C-reactive protein to albumin ratio; EF, Ejection fraction; LV, Left ventricle.

Coronary artery disease / Acute coronary syndrome

OP-103

Predictive value of CHA2DS2-VASc and CHA2DS2-VASc-HS scores for developing contrast induced nephropathy in patients with ST-elevation myocardial infarction

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Background and Aim: We aimed to investigate the predictive value of the CHA2DS2-VASc and CHA2DS2-VASc-HS scores in the development of contrast-induced nephropathy (CIN).

Methods: Total of 360 patients who had been diagnosed with ST elevation myocardial infarction and who had undergone primary coronary angioplasty or thrombolytic therapy were included in the study. The pa-

tients were divided into two groups according to the CHA2DS2-VASc score, i.e.: low risk (0 or 1 point), high risk (≥2 points). The groups were followed with regard to CIN development.

Results: The median CHA2DS2-VASc and CHA2DS2-VASc-HS score was significantly higher in the CIN (+) group compared to the CIN (-) group (2.8±1.8 vs 1.8±1.4 and 3.2±1.3 vs 2.6±1.3, p<0.001 and p=0.001). The rate of CIN was 3.32-fold higher (OR 2.88, 95% CI 1.60-5.20, p<0.001) in the high-risk group (CHA2DS2-VASc ≥2) compared to the low-risk group (CHA2DS2-VASc = 0 or 1). Older age, diabetes, left ventricle ejection fraction, CHA2DS2-VASc score, CHA2DS2-VASc-HS score reached statistical significance in univariable logistic regression analysis. (Age (OR 1.03, 95% CI 1.01-1.05, p<0.001), female gender (OR 1.83, 95% CI 1.04-3.2, p=0.03), CHA2DS2-VASc score (OR 1.43, 95% CI 1.20-1.71, p<0.001), CHA2DS2-VASc-HS score (OR 1.34, 95% CI 1.11-1.30, p<0.001), diabetes mellitus (OR 1.53, 95% CI 0.88-2.67, p<0.001), left ventricle ejection fraction Age (OR 1.03, 95% CI 1.01-1.05, p<0.001), female gender (OR 1.83, 95% CI 1.04-3.2, p<0.001), CHA2DS2-VASc score (OR 1.43, 95% CI 1.20-1.71, p<0.001), CHA2DS2-VASc-HS score (OR 1.34, 95% CI 1.11-1.30, p<0.001), diabetes mellitus (OR 1.53, 95% CI 0.88-2.27, p<0.001). When we put these variables into multivariable logistic regression analysis older age, left ventricle ejection fraction, CHA2DS2-VASc score and CHA2DS2-VASc-HS score were found to be independent predictors of CIN. According to ROC analysis, a CHA2DS2-VASc score of at least 1,5 was predictive of CIN with 78% sensitivity and 45% specificity (area under curve =0.66, p<0.001, 95% CI (0.59-0.78) and a CHA2DS2-VASc-HS score of at 2,5 was predictive of CIN with 70% sensitivity and 50% specificity (area under curve =0.62, p<0.001, 95% CI (0.55-0.69).

Conclusions: The CHA2DS2-VASc and CHA2DS2-VASc-HS score is an independent and strong predictor of CIN development in patients with acute STEMI.

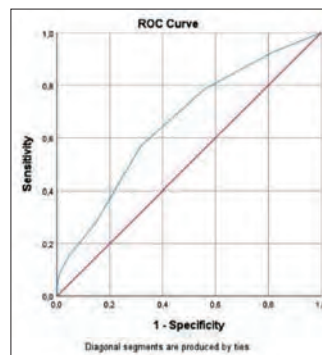


Figure 1. Receiver operating characteristic curve for CHA2DS2VASC score for predicting CIN. CHA2DS2-VASc indicates congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, previous stroke, vascular disease, age 65 to 74 years, female.

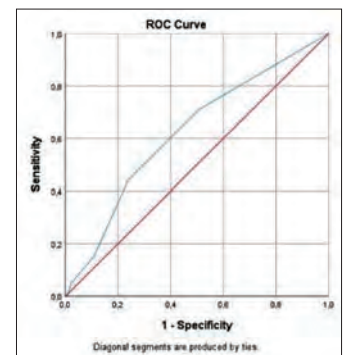


Figure 2. Receiver operating characteristic curve for CHA2DS2VASC score for predicting CIN. CHA2DS2-VASc-HS indicates congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, previous stroke, vascular disease, age 65 to 74 years, female.

Table 1. Baseline parameters

	CIN (-)	CIN (+)	P value
Chads-vasc score	1.8±1.4	2.8±1.8	<0.001
Chads-vasc-HS score	2.6±1.3	3.2±1.3	0.001
LV ejection fraction	45 ±9.6	41±10.5	0.003
Age (years)	61±13	67±11	<0.001

CIN: contrast induced nephropathy.

Table 2. Baseline demographic parameters

	CIN(-)	CIN(+)	P value
Chads-vasc group (high score)	55	78	0.003
Diabetes Mellitus	23	31	0.08
Hypertension	47	55	0.13
Hyperlipidemia	19	21	0.42
Smoking	53	31	<0.001
Female gender	20	31	0.025

CIN: contrast induced nephropathy.

Table 3. Predictors of contrast induced nephropathy in univariable logistic regression analysis

	OR (95% CI)	P value
Chadsvasc group	2.88 (1.60-5.20)	<0.001
Chadsvasc score	1.43 (1.20-1.71)	<0.001
Chadsvaschs score	1.340 (1,11-1.30)	<0.001
Diabetes Mellitus	1.53 (0.88- 2.67)	<0.001
Hypertension	1.37 (0.83-2.27)	0.216
Hyperlipidemia	1.11 (0.60- 2.0)	0.734
Smoking	0.39 (0.23-0.67)	<0.001
LV ejection fraction	0.96 (0.93-0.98)	<0.001
Age (years)	1,03 (1.01- 1.05)	<0.001
Female gender	1.83 (1.04-3.2)	0.034

Table 4. Predictors of contrast induced nephropathy in multivariable logistic regression analysis

	OR (95% CI)	P value
Chadsvasc score	1,3 (0.99-7.7)	0,05
LV ejection fraction	0.96 (0.94-0.99)	0.01
Age (years)	1.0 (1-105)	0.05
Female gender	0.94 (0.44-2.0)	0.8

Lipid / Preventive cardiology

Lipid / Preventive cardiology

OP-104

Arrhythmia tendency in elite athletes: predictive value of novel inflammatory and atherogenic lipid indices

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Background and Aim: Sudden death and arrhythmias in athletes may be devastating and necessitate screening and predicting measures. In this context, well-established and as well as novel indices are necessary to promote athlete health. Index of cardioelectrophysiologic balance (iCEB) is a recently defined non-invasive marker of ventricular arrhythmias serving as cardiac wavelength surrogate easily calculated from surface ECG by dividing QT to QRS duration. An increased iCEB indicates Torsades des pointes risk, where a decreased iCEB may be related with non-Torsades VT/VF, Figure 1. In addition to atherosclerosis, deranged plasma lipids and inflammation may cause alterations cell membrane properties, ion channel functions and membrane fluidity which in turn may have role in arrhythmias. Thus, this study aims to assess the possible relation between novel atherogenic lipid and inflammatory indices like Atherogenic index of plasma (AIP), Castell risk index 1 and 2 (CRI), Atherogenic Coefficient (AC), Monocyte/HDL ratio (MHR), Neutrophil/Lymphocyte ratio (NLR) and iCEB in elite athletes.

Methods: Data of 88 elite male national league football and basketball players were evaluated. Participant were divided into 3 groups according to iCEB tertiles. Mid iCEB group was taken as control group since there is no established normal value for iCEB, high iCEB group was attributed as high torsades tendency where low iCEB group as non-Torsades VT/VF tendency. Statistical analyses were carried out between these groups. Biochemical tests were taken after 12 hr fasting. Atherogenic indices were calculated as follows: AIP= log Triglyceride/HDL, CRI-1=Total Cholesterol/HDL, CRI-2=LDL/HDL, AC=non-HDL cholesterol/HDL cholesterol, MHR=Monocyte count/HDL, NLR=neutrophile count/lymphocyte count.

Results: Age was similar (p=0.414) and conventional lipid parameters including LDL (p=0.171), HDL (p=0.576) and Triglycerides (p=0.357) did not differ significantly, Table 1. Additionally, novel atherogenic lipid indices were similar among tertiles (AIP; p=0.691, CRI-1; p=0.657, CRI-2; p=0.464 and AC; p=0.652). MHR did not differ significantly among tertile (p=0.321), however NLR was statistically different among tertiles (p=0.022). In post-hoc analysis, this difference was due to the difference between low iCEB and high iCEB groups (p=0.022). In correlation analysis, NLR and MHR significantly correlated with iCEB (NLR; r=-0.310, p=0.005 and MHR; r=-0.251, p=0.025). In multivariate linear regression analysis, NLR was still significantly associated with iCEB (β=-0.249; p=0.034) Figure 2.

Conclusions: Among many novel indices, only NLR was significantly possible associated with arrhythmias in elite athletes. These results may be interpreted as increased inflammation status may be related with non-torsades VT/VF rather than Torsades in elite athletes. Negative results related with lipid parameters may be attributed to the regular training exercise promoting lipid values hence not negatively affecting cellular electricity of myocardium.

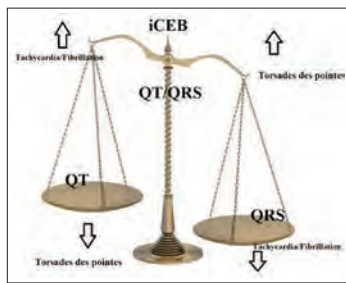


Figure 1. Schematic overview of index of cardioelectrophysiological balance.

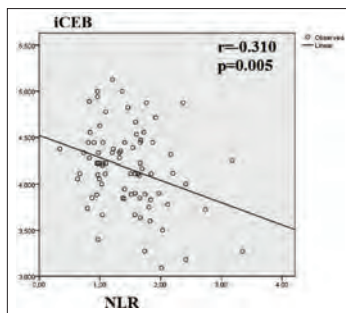


Figure 2. Correlation between neutrophil lymphocyte ratio and index of cardioelectrophysiological balance.

Table 1. Atherogenic ve inflammatory indices according to iCEB tertiles

	Low iCEB tertile (n=29)	Mid iCEB tertile (n=29)	High iCEB tertile (n=30)	P	p (post hoc)
Age	25.7±4.4	24.7±5.3	26.3±4.7	.414	-
Atherogenic index of plasma	.1987±.27	.1391±.23	.1749±.28	.691	-
Castelli Risk index	2.33±.64	2.16±.81	2.42±.97	.657	-
Atherogenic coefficient	2.52±.91	2.36±.92	2.59±1.10	.652	-
Monocyte HDL ratio	.0125±.006	.0113±.004	.0106±.004	.321	-
Neutrophil Lymphocyte ratio	1.7±.6	1.4±.4	1.3±.4	.024	(low vs high).022

OP-105

Comparing the cardiovascular risk scores by mortality predictability

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Background and Aim: Cardiovascular causes are leading cause of mortality in all over the world. There are several risk scores that predicts future cardiovascular events and their predictability success can be variable in different populations. We aimed to evaluate and compare the mortality predictability of three different cardiovascular risk scores in Turkish patients.

Methods: All the patients who had been performed coronary computed tomography (CTA) between January 2007 and January 2010 in our university hospitals were enrolled to the study. Datas including mortality and needed parameters for score estimation were collected from electronic database.

Results: Totally 1427 patients were enrolled to the study. Mean follow-up time was 78 (±20) months. Increased Framingham risk score, SCORE and SCORE Turkey values were associated with increased 1 year, 5 year and 10 year mortality rates (Table 1, Table 2 and Table 3). Correlation analysis showed that "SCORE Turkey" is the best score predicting mortality in Turkish population.

Conclusions: Although all of the scores predicts mortality well, Score Turkey is better than other scores for Turkish population. These results shows that cardiovascular risk scores should be customized for each nations and races.

Table 1. Framingham risk score and survival

FRS	1 year survival	5 year survival	10 year survival
Low risk	99,3%	95,3%	93,2%
Moderate risk	99,2%	88,8%	83,6%
High risk	97,3%	83,9%	79,6%

Table 2. SCORE and survival

SCORE	1 year survival (%)	5 year survival (%)	10 year survival (%)
Very low risk	100	98,9	98,9
Low risk	99,4	93,1	89,5
Moderate risk	97,8	89,7	82
High risk	98,4	89,6	79

Table 3. SCORE Turkey and survival

SCORE Turkey	1 year survival (%)	5 year survival (%)	10 year survival (%)
Very low risk	100	98	96,9
Low risk	99,6	99,6	86,8
Moderate risk	99,3	92,8	88,7
High risk	96,9	82,9	74,7

Lipid / Preventive cardiology

OP-106

Sodium-glucose cotransporter 2 inhibitors and cardiovascular outcomes: A comprehensive meta-analysis of FDA mandated trials

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Background and Aim: The effect of antihyperglycemic agents on cardiovascular outcomes is of utmost clinical importance. Sodium-glucose cotransporter 2 (SGLT-2) inhibitors have been shown to improve certain cardiovascular outcomes in individual clinical trials. The aim of this study is to systematically examine the effect of SGLT-2 inhibitors on cardiovascular outcomes across dedicated cardiovascular outcome trials using meta-analytic methods

Methods: Data was extracted from publications of randomized placebo-controlled cardiovascular outcome trials of SGLT-2 inhibitors mandated by the Food and Drug Administration for the assessment of their cardiovascular safety. Hazard Ratios (HR) were extracted for each cardiovascular outcome in each trial. Heterogeneity was assessed using I² values. Fixed effect models were used to obtain meta-analytic HR's, unless there was evidence of heterogeneity, where random effects models were used.

Results: A total of 3 randomized trials (1 testing 2 different doses of empagliflozin and 2 testing canagliflozin) enrolling a total of 17,162 patients (10,482 randomized to an SGLT-2 inhibitor, 6680 to placebo) were included. SGLT-

2 inhibitors reduced cardiovascular mortality and total mortality, with evidence of some heterogeneity across the trials ($I^2=54.7\%$, $HR=0.73$ [95% CI: 0.60-0.90], $p=0.003$ for cardiovascular mortality; $I^2=37.7\%$, $HR=0.77$ [95% CI: 0.69-0.86], $p<0.00001$ for total mortality). SGLT-2 inhibitors homogeneously reduced heart failure hospitalizations, as well as non-fatal myocardial infarctions and had no effect of non-fatal stroke ($I^2=0\%$, $HR=0.66$ [95% CI: 0.56-0.78], $p<0.00001$ for heart failure hospitalizations; $I^2=0\%$, $HR=0.86$ [95% CI: 0.75-0.99], $p=0.035$ for non-fatal myocardial infarctions). Meta-regression analyses suggested a greater prevalence of coronary artery disease at baseline is associated with more pronounced effects on cardiovascular ($p=0.01$) and total mortality ($p=0.04$). **Conclusions:** The examined SGLT-2 inhibitors homogeneously reduce the risk of heart failure hospitalizations and non-fatal myocardial infarctions. The reductions on cardiovascular and total mortality were more pronounced in the empagliflozin trial. While the exact reason for this is not clear, the current analysis suggests that a greater prevalence of coronary disease at baseline may be associated with a greater benefit in mortality with SGLT-2 inhibition.

Lipid / Preventive cardiology

OP-107

The effects of genetic and environmental factors to atorvastatin therapy in coronary artery disease patients

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Background and Aim: Coronary artery disease (CAD) and its complications are the major causes of death in the world. Although statins have been used to lower lipid levels in CAD patients, this goal can not be attained in 1/3 of the patients. Variability in response to statin therapy results from genetic and environmental factors which may have role of the hepatic regulation of plasma cholesterol. Therefore the objective of this study was to investigate whether common variations in HMG-CoA Reductase (HMGCR) and Apolipoprotein E (ApoE) genes involved in lipid and statin metabolism modify the effect of statins on serum lipid and lipoprotein concentrations also to investigate the effects of environmental factors to lipid-lowering therapy. **Methods:** A hundred CAD patients were enrolled into the study. All patients were started to atorvastatin therapy (40mg/day) and overnight fasting blood sample was taken at baseline and at 2 months after starting therapy for measuring plasma lipid and lipoprotein concentrations. The patients were divided into two groups according to their LDL-c levels; 50 patients whose LDL-c levels were not sufficiently reduced (>100 mg/dL) and 50 patients whose LDL-c levels were sufficiently reduced (<100 mg/dL) were assigned as non-responders and responders respectively. The information regarding the risk factors such as smoking, alcohol consumption etc. were also obtained. DNA was isolated from peripheral blood. The presence of rs17244841 ve rs17238540 mutations in HMGCR and ε2, ε3 and ε4 variants of ApoE were determined by using real time polymerase chain reaction (RT-PCR). Results were evaluated statistically. **Results:** rs17244841 (8%) and rs17238540 (10%) mutations in HMGCR were mostly found in responders and ε4 variant (22%) of ApoE was mostly found in non-responders. It was also found that presence of HMGCR mutations causes a significant reduction in total cholesterol and LDL-c levels. Also presence of ε2 variant of ApoE causes a statistically significant increase in triglyceride levels. Additionally statistically significant relations were detected between genotypes and some biochemical parameters. **Conclusions:** Basic research is required to explain the mechanisms governing the association of HMGCR and ApoE genotypes and response to hypolipidemic medication, whereas clinical studies with larger numbers of patients will answer the question of whether it is meaningful to incorporate HMGCR and ApoE polymorphisms in the individualization of hypolipidemic treatment in CAD patients.

Lipid / Preventive cardiology

OP-108

The relationship between nesfatin-1 levels and SYNTAX score in patients with non-ST segment elevation myocardial infarction

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Background and Aim: Nesfatin-1 is a novel anorectic neuropeptide with potent metabolic regulatory effects. Nesfatin-1 regulates blood pressure, heart rate, cardiomyocyte metabolism and permeability. SYNTAX score, which is an angiographic scoring system, defines grade and complexity of coronary artery disease (CAD). We aimed to evaluate the relationship between Nesfatin-1 level and severity of CAD according to the SYNTAX score in patients with non-ST segment elevation myocardial infarction (NSTEMI). **Methods:** A total of 109 subjects enrolled into the study. Eighty patients who underwent coronary angiography (CA) with the diagnosis of NSTEMI and 29 patients with normal coronary artery detected in CA were included in the study. NSTEMI patients were divided into 2 groups: low SYNTAX score (<32) (45 patients) and high SYNTAX score (≥32) (35 patients). **Results:** NSTEMI patients with a high SYNTAX score (score ≥32) had lower serum nesfatin-1 levels 62 pg/ml (39-98) compared to NSTEMI patients with a low SYNTAX score (score <32) 138 pg/ml (65-286) and control group 392 pg/ml (178-1320). Also, there was a negative correlation between serum nesfatin-1 level and SYNTAX score ($r=-0.594$, $p<0.001$). Lower serum level of nesfatin-1 (OR=0.116; 95% CI: 0.138-0.094; $p<0.001$) was detected as independent predictor for high SYNTAX score in NSTEMI patients after multiple linear regression analysis. **Conclusions:** Serum nesfatin-1 level was lower in the high SYNTAX group than low SYNTAX group in patients with NSTEMI. Nesfatin-1 could have a role in the pathogenesis of atherosclerotic burden in patients with NSTEMI.

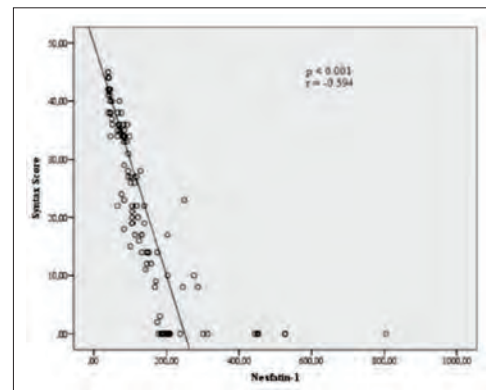


Figure 1. The correlation between Nesfatin-1 level and SYNTAX score.

Table 1. Baseline clinical and angiographic characteristics of the study population

Variables	NSTEMI SYNTAX score ≥32 (n=35)	NSTEMI SYNTAX score <32 (n=45)	NCA (n=29)	p value*	p valueα	p valueβ	p valueγ
Age, years	62.94±9.49	62.29±8.25	58.90±10.64	0.187			
BMI, kg/m ²	30.03±5.67	30.09±4.99	29.90±6.13	0.989			
Waist circumference, cm	90.28±7.21	88.02±6.67	87.45±5.42	0.168			
Systolic blood pressure, mm Hg	136.48±7.35	135.66±10.87	136.79±5.75	0.843			
Diastolic blood pressure, mm Hg	84.65±6.36	84.60±9.53	85.00±4.51	0.973			
Female, n(%)	12 (34.3)	11 (22.4)	8 (27.6)	0.622			
Hypertipidemia, n(%)	17 (48.6)	21 (46.7)	6 (20.7)	0.041			
Diabetes Mellitus, n(%)	13 (37.1)	10 (22.2)	7 (24.1)	0.298			
Smoking, n(%)	17 (48.6)	28 (62.2)	8 (27.6)	0.014	<0.001	<0.001	<0.001
Previous MI, n (%)	5 (14.3)	11 (24.4)	0 (0)	0.015	<0.001		
Multi-vessel disease, n (%)	32 (91.4)	30 (66.7)	0 (0)	<0.001	<0.001		
Chronic total occlusion, n (%)	13 (37.1)	7 (15.6)	0 (0)	<0.001	<0.001		
Stent implantation, n (%)	15 (42.8)	29 (64.4)	0 (0)	<0.001	<0.001		
Decision for CABG, n (%)	15 (42.8)	5 (11.1)	0 (0)	<0.001	<0.001		
Collateral vessel, n (%)	10 (28.6)	4 (8.9)	0 (0)	<0.001	<0.001		
SYNTAX score	37.77±3.66	17.88±7.13	0±0	<0.001	<0.001		
LVEF, %	53.92±10.86	51.82±8.89	58.04±6.85	0.035	0.619	0.24	0.026
Drug usage, n (%)							
β blockers	8 (22.9)	7 (15.6)	2 (6.9)	0.193			
ACEi/ARB	7 (20.0)	14 (31.1)	5 (17.2)	0.318			
Calcium Channel Blocker	6 (17.1)	7 (15.6)	5 (17.2)	0.975			
Spirolactone/ Diuretics	7 (20.0)	7 (15.6)	4 (13.8)	0.784			
Statins	13 (37.1)	10 (22.2)	7 (24.1)	0.298			
Antidepressant	5 (14.3)	7 (15.6)	5 (17.2)	0.949			
ASA	7 (20.0)	7 (15.6)	1 (3.4)	0.145			
Klopidoğrel/ Ticagrelor/ Prasugrel	2 (5.7)	2 (4.4)	0 (0.0)	0.273			
Warfarin/ NOAC	2 (5.7)	2 (4.4)	0 (0.0)	0.273			
İnsulin	3 (8.6)	2 (4.4)	0 (0.0)	0.154			
Oral Antidiabetic	13 (37.1)	10 (22.2)	7 (24.1)	0.298			

Data are given as mean ± SD, n (%) or median (lower-upper limit). BMI, body mass index; CABG, coronary artery bypass grafting; LVEF, left ventricular ejection fraction; NCA, normal coronary artery; NSTEMI, Non ST-segment elevation myocardial infarction; MI, myocardial infarction; NOAC, Novel oral anticoagulants; ASA, Acetylsalicylic acid; ACEi, angiotensin converting enzyme inhibitors; ARB, angiotensin II receptor blockers. # parameters without normally distributed (One-Way ANOVA test was used to show the differences between the three groups for continuous numeric parameters with normal distribution. Tukey's test was used for the post-hoc analysis. Kruskal-Wallis test was used for the differences between the three groups in parameters without normally distributed, if there was statistical significance the Mann-Whitney U test was used for nonpaired groups. Differences between three groups for the categorical variables were analyzed using the chi-square test.) * p value between all groups α, p value between NSTEMI SYNTAX score ≥32 and NSTEMI SYNTAX score <32 groups β, p value between NSTEMI SYNTAX score ≥32 and NCA groups γ, p value between NSTEMI SYNTAX score <32 groups and NCA groups groups

Table 2. Biochemical and hematological measurements of the study patients

Variables	NSTEMI SYNTAX score ≥32 (n=35)	NSTEMI SYNTAX score <32 (n=45)	NCA (n=29)	p value*	p value ^α	p value ^β	p value ^γ
Glucose, mg/dL	151.92±72.33	131.08±47.76	133.75±45.02	0.234			
Creatinine, mg/dL	0.99±0.26	1.12±0.78	0.91±0.113	0.234			
Total cholesterol, mg/dL	199.71±49.03	188.44±55.27	176.20±45.28	0.237			
LDL-C, mg/dL	118.96±42.78	112.18±45.09	98.75±37.77	0.215			
HDL-C, mg/dL	47.65±12.56	46.18±10.11	58.54±16.91	0.001	0.876	0.006	0.001
Triglyceride, mg/dL	174.84±113.24	150.76±91.30	147.13±79.68	0.479			
Hemoglobin, g/dL	13.79±1.63	13.82±1.76	13.37±1.28	0.494			
WBC, 10 ³ /mm ³	9.20±2.15	8.81±2.12	7.52±1.59	0.003	0.668	0.003	0.024
Platelet, 10 ³ /mm ³	238.91±70.66	248.83±74.45	249.46±66.39	0.787			
HbA1c (%)	6.58±1.04	6.69±1.24	7.05±1.62	0.365			
hs-CRP, mg/L	4.02±0.81	3.32±1.23	1.58±0.59	<0.001	0.005	<0.001	<0.001
Adropin, pg/mL	2357.30±821.58	3077.00±912.86	3688.00±956.65	<0.001	0.003	<0.001	0.016
Peak CK-MB, U/L	46 (15-229)	38 (0-131)		0.488			
Peak troponin-T, ng/mL	4.51 (0-34.7)	5.27 (0.09-31.00)		0.884			
Nesfatin-1, pg/ml	62 (39-98)	138 (65-286)	392 (178-1320)	<0.001	<0.001	<0.001	<0.001

Data are given as mean ± SD, n (%) or median (lower-upper limit). CK-MB, creatine kinase-myocardial band; hs-CRP - high sensitivity C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; WBC, white blood cell. # parameters without normally distributed (One-Way ANOVA test was used to show the differences between the three groups in continuous numeric parameters with normal distribution. Tukey's test was used for the post-hoc analysis. Kruskal-Wallis test was used for the differences between the three groups in parameters without normally distributed, if there was statistical significance the Mann-Whitney U test was used for nonpaired groups. Differences between three groups for the categorical variables were analyzed using the chi-square test.) * p value between all groups; α p value between NSTEMI SYNTAX score ≥32 and NSTEMI SYNTAX score <32 groups; β p value between NSTEMI SYNTAX score ≥32 and NCA groups; γ p value between NSTEMI SYNTAX score <32 groups and NCA groups.

Table 3. Pearson analysis of nesfatin-1 with other parameters

Variables	r value	p value
Age	0.005	0.959
BMI	-0.054	0.591
SYNTAX score	-0.594	<0.001
LDL-C	-0.154	0.143
HDL-C	0.175	0.096
Triglyceride	-0.013	0.904
WBC	-0.167	0.101
Peak CK-MB	-0.284	0.013
Peak troponin-T	-0.192	0.094
hs-CRP	-0.147	0.394
Adropin	0.102	0.245

CK-MB, creatine kinase-myocardial band; hs-CRP - high sensitivity C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; WBC, white blood cell; BMI, body mass index.

Table 4. Multivariate logistic regression analysis showing the predictors for the SYNTAX ≥32 score

Variables	Univariable Beta (95% CI)	p value	Multivariable Beta (95% CI)	p value
Smoking	1.744 (0.712-4.272)	0.224		
Nesfatin-1	0.047 (0.059-0.034)	<0.001	0.116 (0.138-0.094)	<0.001
Peak CK-MB	0.111 (0.019-0.203)	0.018	0.067 (0.052-0.083)	0.098
Dyslipidemia	0.926 (0.383-2.244)	0.866		
hs-CRP	1.874 (1.179-2.979)	0.008	1.154 (0.753-1.556)	0.139
Adropin	0.999 (0.998-1.000)	0.002	1.003 (1.002-1.004)	0.046
WBC	1.091 (0.883-1.349)	0.012	0.485 (0.527-1.497)	0.245
HDL-C	1.012 (0.971-1.055)	0.571		

CK-MB, creatine kinase-myocardial band; hs-CRP - high sensitivity C-reactive protein; HDL, high-density lipoprotein; WBC, white blood cell.

Results: There was no statistical difference between the arrhythmogenic P wave dispersion at baseline and after one year of treatment. However Tp-e, and Tp-e/QT values were significantly found to decrease after 1 year of colchicine treatment (p=0.022 and p=0.014, respectively).

Conclusions: Although colchicine treatment may have no effect on the atrial arrhythmia risk in FMF patients, it might have a favorable effect on ventricular repolarization indices and might be protective against ventricular arrhythmias and sudden death in patients with FMF.

Table 1. Baseline clinical and laboratory characteristics in patients with familial Mediterranean fever

	Patients (n=28)
Female gender, n (%)	20 (72)
Age (years)	31.4±8.2
BMI (kg/m ²)	26.4±3.4
Smoking, n (%)	7 (25)
Comorbidities*, n (%)	3 (10.7)
Frequency of attacks (years)	10.6±7
Duration of attacks (days)	3.3±1.5
Main FMF Symptoms	
Peritonitis, n (%)	21 (75)
Fever, n (%)	2 (7.1)
Musculoskeletal symptoms, n (%)	5 (17.9)
Creatinine (mg/dl)	0.6±0.2
ALT (U/L)	21.7±10.4
AST (U/L)	20.9±10.9
Albumine (g/dL)	4.6±0.3
CRP (mg/L)	19.2±32
ESR (mm/h)	23.8±16.6
Protein/creatinin in spot urine	1±11.2
WBC (x10 ³ /mL)	7.2±1.7
Hemoglobin (g/dL)	12.6±1.8
Platelet (x10 ³ /mL)	266.4±79.2
Documented tachycardia, n (%)	6 (21.4)

Values are presented as means ± standard deviations and medians with interquartile range in parentheses. BMI: body mass index; ALT: alanin aminotransferase; AST: aspartate aminotransferase; CRP: c-reactive protein; ESR: eritrosit sedimentation rate; WBC: white blood cell. *Hypertension, hypothyroidism, hyperthyroidism, cardiovascular diseases, coronary artery disease, cerebrovascular disease, chronic renal disease, chronic obstructive pulmonary disease, diabetes mellitus.

Table 2. Electrocardiographic findings and disease parameters of the patients at baseline and 1 year after colchicine treatment

	Baseline (n=28)	After colchicine treatment (n=28)	P value
Tp-e interval (ms)	69.93±14.68	66.21±14.35	0.022
Tp-e/QT	0.21±0.04	0.19±0.03	0.014
Tp-e/QTc	0.17±0.37	0.16±0.03	0.507
QTd (ms)	53.11±10.33	47.07±18.7	0.133
Pd (ms)	31.64±11.67	32.93±10.33	0.437
Pd ≥ 40 ms, n (%)	7 (25)	10 (35.7)	0.207
CRP (mg/L)	19.2±32	9.5±17.1	0.059
ESR (mm/h)	23.8±16.6	16.8±14.9	0.006
Frequency of attacks (years)	10.6±7	0.8±1.2	<0.001
Colchicine dose(mg)*		1.2±0.3	

Values are presented as means ± standard deviations. Tp-e: transmural dispersion of repolarization interval; QT: QT interval; QTc: corrected QT interval; QTd: QT dispersion interval; Pd: P wave dispersion. CRP: c-reactive protein; ESR: eritrosit sedimentation rate. *Average colchicine dose in the first year.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-109

May colchicine be protective against ventricular arrhythmias in patients with familial Mediterranean fever?

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Background and Aim: Familial Mediterranean fever (FMF) is an autoinflammatory disease. Colchicine is used to prevent attacks and amyloidosis in patients with FMF and may be also protective against cardiac arrhythmia in patients without FMF. Therefore, in this study we proposed to investigate whether colchicine decrease cardiac arrhythmia risk at 1 year after treatment in patients with FMF.

Methods: Twenty eight new diagnosed FMF patients (20 women, mean age 31.4± 8.2) who fulfilled modified Tel Hashomer criteria were recruited to study. After baseline assessments, colchicine treatment was started. ECG, laboratory parameters, demographic and disease related parameters of the patients were obtained at the first visit and one year after treatment. We evaluated the risk of atrial arrhythmia with P dispersion and risk of ventricular arrhythmia with Tp-e, Tp-e/QT, Tp-e/QTc. P dispersion ≥40 ms was accepted as arrhythmogenic anomaly for atrial arrhythmias.

Coronary artery disease / Acute coronary syndrome

OP-110

Relation between de-ritris ratio and myocardial performance index in patients with a first acute anterior myocardial infarction

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Background and Aim: Aminotransferase is a well-known marker for liver injury and is composed of alanine aminotransferase (ALT) and aspartate aminotransferase (AST). ALT is only located in the liver, but AST is located in both the liver and myocardial tissue. Cardiac disease leads to hypoxic hepatitis, which results in a rise in serum transaminase activity caused by anoxic necrosis of the centrilobular liver cells [10, 11]. The elevation of aminotransferase in cardiac disease is characterized by a greater increase in AST compared with

ALT (due to the localization of these two enzymes). De Ritis described the ratio between the serum levels of aspartate transaminase (AST) and alanine transaminase (ALT). An increased aspartate aminotransferase-to-alanine aminotransferase ratio (AAR) has been widely used as a marker of advanced hepatic fibrosis. Increased AAR was also shown to be significantly associated with the risk of developing cardiovascular (CV) disease. Myocardial performance index (MPI) is an easily measured index for the assessment of global heart function, combining both systolic and diastolic components. MPI has been shown to be independent of HR, blood pressure, loading conditions and the geometry of the ventricles and can be used to evaluate the function of both the RV and the LV. The aim of this study was to assess the relationship between the AAR and LV MPI in patients with a first anterior acute myocardial infarction.

Methods: The subjects were 141 patients (114 men, 27 women, 59±13 years) with a first anterior acute myocardial infarction. Demographic, clinical, and laboratory data were collected for all patients. Peripheral venous blood samples were taken from patients at the admission to cardiology care unit. Biochemical parameters were measured. Cardiac evaluation with echocardiography was performed within 24 hours of admission to cardiology care unit. LV MPI by TD) was obtained from the apical four-chamber view by placing the sample volume at the lateral mitral annulus (Figure 1). Based on the myocardial performance index (MPI), they were classified into Group 1 (LV MPI <0.45) and Group 2 (LV MPI ≥ 0.45). Echocardiograms were used to determine LV MPI, in the 24 hours after the onset of AMI.

Results: There was no significant difference in the baseline characteristics of patients (Table 1). De-Ritis ratio was 1.787±1.218 in patients with Group 1 and 2.563±1.975 in patients with Group 2. De-Ritis ratio was significantly lower in patients Group 1 than Group 2 (p=0.004).

Conclusions: We suggested that a significant association between the De-Ritis ratio and LV MPI in patients with a first anterior AMI.

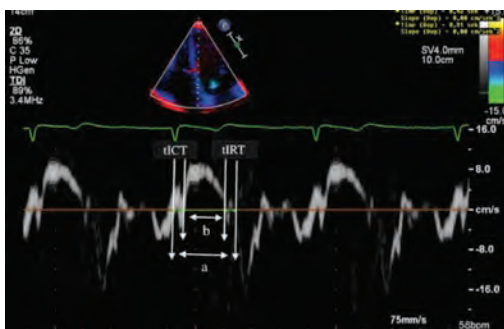


Figure 1. Myocardial performance index measured by tissue Doppler imaging. Time intervals by tissue Doppler imaging derived from septal annulus. tICT, tissue isovolumic contraction time; tIRT, tissue isovolumic relaxation time; a = isovolumic contraction time + isovolumic relaxation time + ejection time; b = ejection time. MPI = (a-b)/b.

Table 1. Clinical characteristics of patients

Variables	Grup 1 (LVMPI <0.45) n=93	Grup 2 (LVMPI ≥0.45) n=48	p Value
Age (Year)	59±14	58±12	NS
Gender (F/M)	12/81	15/33	NS
Hypertension	22	16	NS
Diabetes Mellitus	7	5	NS
Smoking	60	29	NS
Hyperlipidemia	27	11	NS

Coronary artery disease / Acute coronary syndrome

OP-111

Relation between angiotensin converting enzyme gene polymorphisms and Musekna Index (modified prognostic index) in patients with a first acute anterior myocardial infarction

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Background and Aim: Carriers of the D-allele of ACE insertion/deletion (I/D) polymorphism display elevated serum and cardiac ACE activity ACE DD-genotype was associated with augmented neurohumoral activation and cardiac dilatation. Patients with acute anterior myocardial infarction who have ACE DD genotype are high risk of heart failure and death. Prediction tools are particularly helpful in this context in guiding medical decision-making. Antonini L et al found that prognostic index was predictor of death and heart failure in patients with ischemic cardiomyopathy implanted with an ICD. The prognostic index (PI) was built according to the formula: 120 - age + mean 24 h systolic blood pressure - (creatinine * 10). However, mean 24 h systolic blood pressure calculation is not clinically easy. Therefore, we propose a new modified prognostic index (Musekna Index). Musekna Index was calculated as "120 - age + mean arterial pressure - (creatinine * 10)". We aimed to investigate relation between Angiotensin Converting Enzyme gene polymorphisms and Musekna Index in patients with a first acute anterior myocardial infarction.

Methods: Overall 140 patients with a first acute anterior myocardial infarction (MI) were included in this cross-sectional study. DNA was isolated from peripheral leukocytes. The ID status was determined by polymerase chain reaction by a laboratory staff member who was unaware of the clinical details. Based on the

polymorphism of the ACE gene, they were classified into 2 groups: Deletion/Deletion (DD) genotype (Group 1, n=57), Insertion/Deletion (ID), Insertion/Insertion (II) genotypes (Group 2, n=83) (Figure 1). Musekna Index (Modified Prognostic Index) was calculated as "120 - age + mean arterial pressure - (creatinine * 10)" at admission to coronary care unit. Echocardiographic examinations were performed using the recommendations of the American Echocardiography Committee. One-way analysis of variance (ANOVA) and Chi-square analyses were used to compare differences among subjects with different genotypes. The study was approved by the local Ethics Committee, and each patient gave a written consent.

Results: There were no significant differences among clinical parameters of patients (Table 1). Musekna Index was significantly lower in patients who have ACE DD genotypes than in patients who have ACE ID/II genotype (132.5±16.7 and, 143.9±19.2, p=0.0025).

Conclusions: Our results suggested that, ACE Gene I/D polymorphism D allele may affect Musekna Index in patients with a first acute AMI.

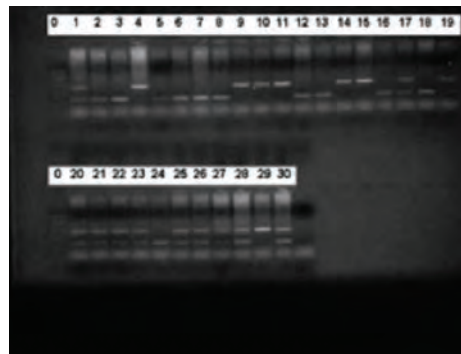


Figure 1. Gel electrophoresis of the ACE ID polymorphism. 0: a DNA size marker (100bp), 1:ID, 2:DD, 3:DD, 4:II, 5:DD, 6:DD, 7:DD, 8:DD, 9:II, 10:II, 11:II, 12:D-D, 13:DD, 14:I:II, 15:II, 16:DD, 17:ID, 18:DD, 19:ID, 20:ID, 21:ID, 22:ID, 23:ID, 24:D-D, 25:ID, 26:ID, 27:ID, 28:ID, 29:II, 30:ID.

Table 1. Clinical characteristics of patients according to ACE I/D Genotype

Parameters	ACE DD (n=57)	ACE ID /II (n=83)	p value
Age, years	58±11	59±13	NS
Gender, F/M	8/49	18/65	NS
BMI, kg/m2	22±3	23±3	NS
Hypertension, n(%)	20(35%)	16(19%)	NS
Diabetes Mellitus, n(%)	6 (10 %)	6 (7 %)	NS
Current Smoking, n(%)	34 (59 %)	50 (60 %)	NS
Hypercholesterolemia, n(%)	12 (21 %)	26 (31 %)	NS
MI localisation, n(%)			
1) Anteroseptal	8 (14 %)	13 (16 %)	NS
2) Anterior	17 (30 %)	21 (25 %)	
3) Large Anterior	30 (52 %)	46 (55 %)	
4) Anterolateral	2 (4 %)	3 (4 %)	

Coronary artery disease / Acute coronary syndrome

OP-112

A simple formula discriminating subtle anterior MI from normal variant ST-segment elevation

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Background and Aim: Benign variant ST-segment elevation (BV-STE) is present in anterior chest leads in most individuals and may cause diagnostic confusion in patients presenting with chest pain. Recently, two regression formulas were proposed for differentiation of BV-STE from anterior ST-elevation myocardial infarction on the electrocardiogram, computation of which are heavily dependent. We hypothesized that a simpler visual-assessment-based formula will be non-inferior to these formulas.

Methods: Consecutive cases of proven LAD occlusion were reviewed, and those that were obvious ST elevation myocardial infarction were excluded. First 200 consecutive patients with non-cardiac chest pain were also enrolled as a control group. Relevant electrocardiographic parameters were measured. A simple formula using R amplitude in lead V4, ST-segment elevation in lead V3, uncorrected QT interval and QRS amplitude in lead V2 was calculated.

Results: There were 138 anterior MI and 196 BV-STE cases. Our simple formula was superior to the three- and non-inferior to the four-variable formulas. This new practical formula had an excellent area-under curve (AUC) of 0.963 (95% CI, 0.946 to 0.980, p<0.001). It also had a sensitivity, specificity and diagnostic accuracy of 86.9%, 92.3% and 90.1%, respectively.

Conclusions: A simple visual-assessment-based formula can reliably differentiate ST-segment elevation myocardial infarction from BV-STE.

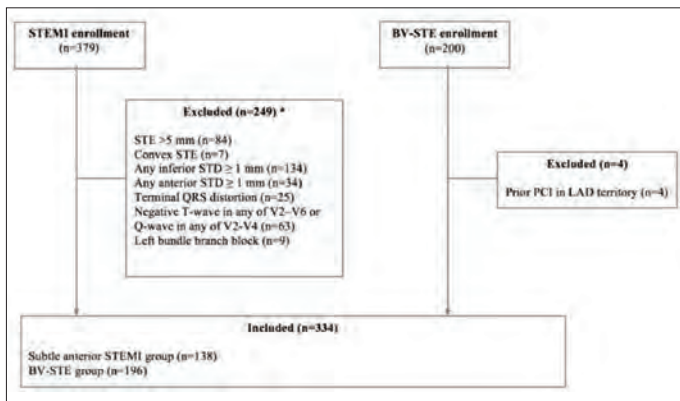


Figure 1. Patient selection flowchart.

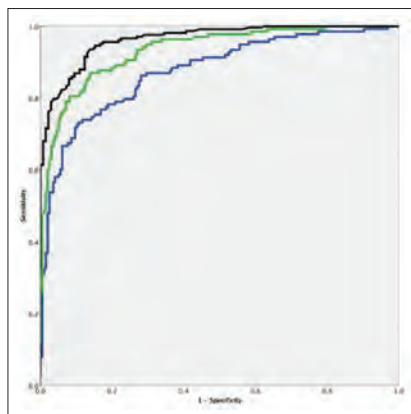


Figure 2. ROC curves.

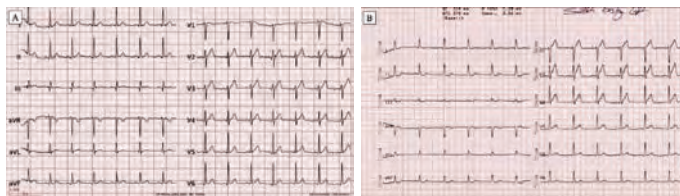


Figure 3. (a, b) Two ECGs look similar at first glance. A, ECG of a patient with chest pain ruled out for myocardial infarction. Computerized corrected QT was 402 milliseconds. The four-variable formula misdiagnosed it as STEMI: $(0.052 \times \text{Bazett-corrected QT}) - (0.151 \times \text{QRS amplitude in V2}) - (0.268 \times \text{R-wave amplitude in V4}) + (1.062 \times \text{STE60 in V3}) = 20,904 - 2.114 - 2.412 + 2.124 = 18.5$ (above 18.2). However, our simple formula classified it as BV-STEMI: $(\text{R-wave amplitude in lead V4} + \text{QRS amplitude in V2}) - (\text{QT interval in millimeters} + \text{STE60 in V3}) = (9 + 14) - (8.5 + 2) = 12.5$ (higher than 12). It was also correctly classified by 3-variable formula: $(1.196 \times \text{STE60 in V3}) + (0.059 \times \text{Bazett-corrected QT}) - (0.326 \times \text{RA in V4}) = 1.794 + 23.718 - 2.771 = 22.7$. Value smaller than 23.4 correctly predicts BV-STEMI. B, ECG of a patient with occlusion of left anterior descending coronary artery after first diagonal and septal artery take-off. This ECG represents another limitation of the previous formulas, namely the dependence on computerized corrected QT interval. In this example, ECG machine mistakenly measured QT interval as 334 milliseconds and calculated computerized corrected-QT as 383 milliseconds. The four-variable formula misdiagnosed it as BV-STEMI: $(0.052 \times \text{Bazett-corrected QT}) - (0.151 \times \text{QRS amplitude in V2}) - (0.268 \times \text{R-wave amplitude in V4}) + (1.062 \times \text{STE60 in V3}) = 19,5 - 1.661 - 3.216 + 3.186 = 17.8$ (below 18.2). However, our simple formula classified it as STEMI: $(\text{R-wave amplitude in lead V4} + \text{QRS amplitude in V2}) - (\text{QT interval in millimeters} + \text{STE60 in V3}) = (12 + 11) - (8.5 + 3) = 11,5$ (lower than 12). It was also incorrectly classified by 3-variable formula: $(1.196 \times \text{STE60 in V3}) + (0.059 \times \text{Bazett-corrected QT}) - (0.326 \times \text{RA in V4}) = 3.588 + 22.125 - 3.912 = 21.8$. Value smaller than 23.4 predicts BV-STEMI.

Coronary artery disease / Acute coronary syndrome

OP-113

Inflammation in epikardial adipose tissue is associated with coronary artery disease

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Background and Aim: Inflammatory activity originating from the epikardial adipose tissue (EAT) may have a role in coronary artery disease (CAD) pathogenesis. The relationship between inflammation in the (EAT), detected by pathological investigation, and (CAD) was investigated in this study.

Methods: Epicardial fat tissue samples were taken intraoperatively around the left atrium and around the coronary artery in 23 patients (CAD group) who underwent isolated coronary artery bypass graft surgery and 15 patients (control group) who underwent isolated valve surgery. Specimens were stained and the presence and amount of inflammatory cell infiltrates were examined. More than 50 inflammatory cell counts in the pathological examination were accepted as significant inflammation.

Results: The median value for the ages of 38 patients in the study was 55 (46-64) and 81% were male. Median white blood cell (7.45 vs 7.13 p=0.842) and median CRP (0.4 vs 0.5 p=0.755) values were similar in both groups. In the CAD group, periarterial inflammation was more frequent (43% vs 6.6% p=0.014) than the control group. Periarial inflammation was also found more frequently in the CAD group (34% vs 6.6% p=0.046).

Conclusions: In this study we showed that both periarterial and periarial fat tissue inflammation is more frequent in patients with coronary artery disease than in valvular disease patients. This result suggests that inflammation in EAT plays a role in the pathogenesis of atherosclerosis.

Coronary artery disease / Acute coronary syndrome

OP-114

Relationship between epicardial fat tissue thickness and coronary thrombus burden in patients with ST elevation myocardial infarction

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Background and Aim: Thrombus burden is an important factor affecting the success of the procedure especially in patients with percutaneous coronary intervention. Therefore, determining factors predicting the intracoronary thrombus burden has great importance in predicting adverse cardiovascular events as well as determining the most appropriate treatment strategy to prevent percutaneous coronary intervention failure. The aim of this study was to evaluate the relationship between EAT thickness and thrombus burden in the patients with STEMI who undergo primary PCI (pPCI).

Methods: Our study includes prospectively evaluated 156 patients. Patients who were referred to the Koşuyolu Research and Education hospital with STEMI between 2016 and 2017 were enrolled to our study. Thrombus burden was scored in five grades: 0 (no thrombus), 1 (possible thrombus), 2 (definite thrombus <0.5 reference vessel diameter), 3 (definite thrombus 0.5-2 reference vessel diameters), 4 (definite thrombus >2 reference vessel diameters), and 5 (complete vessel occlusion) as previously described. The patients were grouped into 2 categories of low thrombus burden (grades 0-3) and high thrombus burden (grades 4 and 5). Epicardial adipose thickness, identified as an echo-free space between the myocardium and visceral pericardium, was measured perpendicularly, on the free wall of the right ventricle from both parasternal long-axis and short-axis views at end diastole in three cardiac cycles.

Results: A total of 156 patients were included in this study (51 in the low thrombus burden group and 105 in the high thrombus burden group). There were no differences in the two groups for LVEF, smoker, family history of CAD, DM, HT, and hypercholesterolemia and for values of total cholesterol, triglyceride, GFR, LDL-C and HDL-C. The predictors of high thrombus burden were studied by multivariate logistic regression analysis. The EAT thickness (odds ratio: 2.53, 95% CI: 1.76-3.67; p<.001) was found as an independent predictor of high thrombus burden.

Conclusions: Present study we demonstrated that EAT is an independent predictors of coronary thrombus burden with STEMI who underwent pPCI.

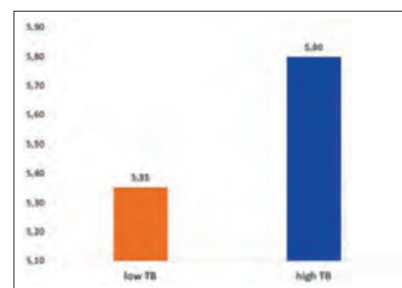


Figure 1. The EAT value between low vs high thrombus burden.

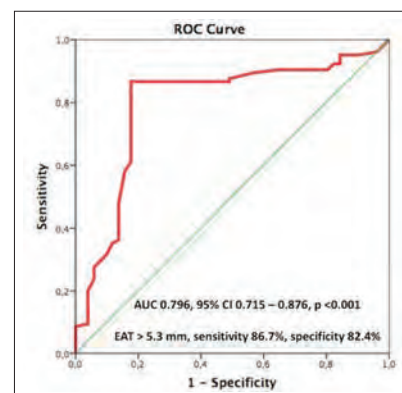


Figure 2. The ROC curve analysis in prediction of high thrombus burden for EAT values.

Table 1. Baseline clinical and angiographic characteristics between patients with low vs high thrombus burden

	Low n=51	High n=105	P value
Age, years	53.7±11.2	56.3±10.6	0.730
Sex, male % (n)	32.8 (42)	67.2 (86)	0.657
Dislipidemia %	21.0±(37.5)	33.0±(30.30)	0.350
Familial History CAD, %	12 (23)	32 (30.50)	0.367
Waist circumference, cm	95.37±11.22	99.30±11.28	0.046
BMI, kg/m ²	27.67±3.23	27.99±3.9	0.525
Killip	12.0 (23.5)	27.0 (25.7)	0.768
Fasting glucose level mg/dl	130.73±42.26	142.29±48.13	0.038
WBC, x10 ⁹	11339±1997	12656±3199	0.001
HDL cholesterol, mg/dl	39.5±9.87	38.5±9.36	0.454
LDL cholesterol, mg/dl	135.2±29.2	130.58±34.6	0.334
TG, mg/dl	131.0 (103-184)	136.0 (85-195)	0.962
GFR	85.6±26.1	94.4±34.6	0.951
Fasting CKMB ng/ml	34.0 (24-55)	44.0 (25-57)	0.193
Fasting Troponin ng/ml	4.40 (2.0-8.0)	5.0 (3-7)	0.398
LVEF, %	47.9±6.87	46.9±7.09	0.242
EAT thickness, mm	5.35±1.58	5.80±1.45	0.008
Gate-balloon, minute	31.06±7.20	31.88±7.43	0.506
Pain-balloon, minute	140.0 (90-189)	152.0 (97-249)	0.192
Culprit lesion location, %			0.698
LAD	23.00±45.10	48.00±45.70	
CX	16.00±31.40	25.00±23.80	
RCMA	12.00±27.60	31.00±29.50	
LMCA	0.00±0.00	1.00±0.90	
Trofiban (+) / %	29±46.80	82±71.30	0.900
Trofiban (-) / %	24.0±38.70	32.0±27.80	0.900
Predilatation (+)	43.0±68.30	93.0±80.90	0.860
Predilatasyon (-)	15.0±23.80	20.0±17.40	0.860
Stent length, mm	21.81±8.60	22.63±7.78	0.487
Stent diameter, mm	3.37±0.52	3.37±0.47	0.808

Table 2. Logistic regression analysis in prediction of patients with high thrombus burden

	Univariate OR, 95% CI	P value	Multivariate OR, 95% CI	P value
WBC	1.00 (1.00-1.00)	0.013	1.00 (1.00-1.00)	0.134
EAT	2.53 (1.76-3.67)	<0.001	2.32 (1.60-3.36)	<0.001

Coronary artery disease / Acute coronary syndrome

OP-115

Genetic risk factors for coronary artery disease that are specific to Turkish population

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Background and Aim: Genetic risk factors that cause coronary artery disease (CAD) show variations in different population. Our aim is to establish a genetic risk panel that potentially causes atherosclerotic CAD. In this study, 14 single nucleotide polymorphisms (SNP) of several candidate genes have been investigated and associated with CAD.

Methods: 525 patients with angina or acute myocardial infarction who underwent coronary angiography were divided into 2 groups: normal coronary arteries (coronary lesion with ≤30% stenosis) and critical disease (≥1 coronary lesion with ≥50% stenosis). DNA was extracted from peripheral blood leucocytes using inorganic method. The angiographic severity and the extent of atherosclerotic coronary artery disease were assessed using Gensini and SYNTAX scores. In this study, 14 SNP selected from APOE, LDLR, APOA5, APOB, APOC1, APOD, MIF, ESR1, CLU, CYP19A1, CH25H, SORL1 and LPA genes were genotyped using high throughput system, LC-480, and the results were statistically evaluated.

Results: Variations of APOE, APOA5, APOC1, ESR1, CYP19A1, CLU and CH25H genes were found associated with serum lipid levels in CAD (p<0.05). It was found that SNPs in APOA5, APOD, APOC1, LDLR and APOE genes are associated with type 2 diabetes and obesity (p<0.05). Moreover, it was determined that APOE genes ε2 allele carriage and rare alleles of polymorphism in CLU decreased the risk for diabetes and CAD (p<0.05). In contrast, rare allele carriage of ESR1 polymorphism was found significantly associated with the increased risk of CAD (p<0.05). In addition, APOC1, CLU, APOE, LDLR and MIF gene polymorphisms were demonstrated to affect serum glucose and HbA1c levels (p<0.05). In the present study CAD severity was found significantly associated with selected CLU and CYP19A1 gene polymorphisms. The association of rare alleles of selected genes with clinical parameters is influenced in a gender specific manner.

Conclusions: In this study, genetic risk factors for the development of CAD have been defined and associated with disease that is specific to Turkish population.

Pediatric cardiology

OP-116

Conditions requiring percutaneous interventional procedures before and after cavopulmonary circulation surgery

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Background and Aim: For patients with single ventricle complex congenital heart diseases primary surgery option is Fontan or Glenn cavopulmonary anastomosis that maintain systemic venous return with passive flow through the lungs. Interventional catheterization is an effective method for the treatment of residual defects and complications of cavopulmonary anastomoses.

Methods: In this study patients who underwent interventional catheterization for cavopulmonary anastomoses were retrospectively analyzed.

Results: In our hospital between 2008 and 2016 years, Fontan or Glenn surgery was performed between age of 2 and 38 years and patients were weighing between 6 and 85 kg. Ninety-two cases required cardiac catheterization, 42 cases required diagnostic and 47 patients required interventional catheterization. Forty-seven patients were enrolled in this study. Interventional process were needed in 33 patients with Glenn, 12 patients with Fontan and 2 patient with Kawasima. In 34 patients aorto-pulmonary collateral (APCA) were embolized with coil or vascular plug, in 6 patients veno-venous collateral embolized by vascular plug. Stent implantation were performed in 15 cases; 4 patients had vena cava superior and pulmonary artery anastomosis, 5 had pulmonary artery branch stenosis, 6 had vena cava inferior and extracardiac tube anastomosis. Hemodynamic improvement was observed in all cases, significant increase in oxygen saturations, and congestive findings were dramatically improved. In 3 patients, closure of the fenestration (ASD occluder in 2 cases, coated stent in 1 case) was performed. Balloon valvuloplasty was effectively performed to a patient with pulmonary atresia and tricuspid valv anomaly who underwent bioprosthetic valve replacement for tricuspid valve stenosis.

Conclusions: Angiography and haemodynamic study is necessary before or after the surgical procedures for cavopulmonary circulation, or when any hemodynamic problem occur. Then interventional treatment may be needed. Patients who underwent Glenn surgery frequently required embolization, patients with Fontan in frequently required stent implantation for stenosis of pulmonary arteries and anastomosis. The combination of surgical and interventional catheterization is an indispensable method in the treatment of early and late complications.



Figure 1. Extracardiac tube anastomosis stenosis.

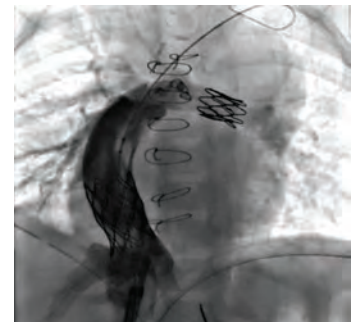


Figure 2. Implanted stent for extracardiac tube anastomosis stenosis.

Pediatric cardiology

OP-117

When catheterization and angiography are required following arterial switch operation?

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Background and Aim: After arterial switch operation (ASO) in surgical treatment of transposition of the great arteries (TGA), long-term survival and quality are higher because left ventricular systemic function is preserved. One of the most critical stages of the operation is translocation of coronary arteries and acute myocardial infarction is reported in 1-2% in cases of late mortality. In addition, problems such as supra-valvular neopulmonary stenosis, neo-aortic root enlargement or neo-aortic valve insufficiency may be life threatening in this patient group.

Methods: In this study patients who underwent ASO and clinical, echocardiographic, electrocardiographic monitoring and cardiac catheterization findings (left ventricular systolic functions, right ventricular and neopulmonary arterial pressures, neo-aortic root diameters, coronary artery problems) were reviewed retrospectively.

Results: Patients (n=30) who underwent ASO and evaluated by cardiac catheterization between 2008 and 2018 were studied. The follow-up period of the patients after surgery was 9.9±6.3 years. Diagnostic catheterization was performed in 22 patients and interventional catheterization was required in 8 patients due to neopulmonary or neo-aortic stenosis. Neopulmonary stenosis was observed in 16 patients, neo-aortic stenosis in 2 patients and neo-aortic insufficiency in 6 patients. Four patients underwent balloon pulmonary angioplasty, 3 patients underwent balloon pulmonary angioplasty and stenting. Five patients underwent to surgery. Sinus valsalva dilatation was revealed in thirteen patients. Coronary angiography evaluation was normal in 18 patients, right and left coronary arteries originating from single root or orifice were found in 7 patients and stenosis in proximal of left coronary artery was revealed in 4 patients. LIMA-LAD bypass was

performed in two patients, coronary patch plasty was performed in one patient and operation was planned in one patient.

Conclusions: Because ASO provides anatomical complete corection, it is the first choice in TGA treatment. Coronary reimplantation complications are due to morphologic factors such as coronary artery course types and intramural coronary pathology. Depending on neo-aortic dilatations, coronary problems may also occur due to withdrawal, torsion or bending in coronary arteries. Ischemia, myocardial perfusion impairment can be detected clinically, with echocardiographic examination or scintigraphy. Coronary arteries form according to the growth and metabolic rates of children and coronary problems may not be seen. Surgical coronary morphology and postoperative and subsequent cardiac and vascular development and residual problems such as pulmonary stenosis lead to coronary imaging. So, coronary artery imaging can be recommended for all children who underwent ASO before sporting age as a term of preventive cardiology.

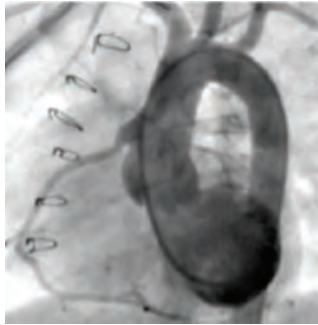


Figure 1. Coronary angiography with stenosis in left coronary artery orifice at 17 months after arterial switch operation.

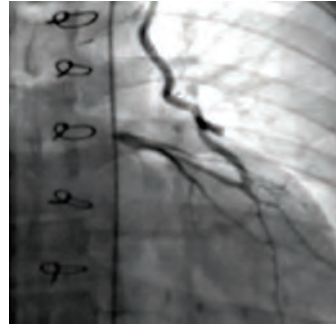


Figure 2. LIMA-LAD bypass for LAD stenosis.

Other

OP-118

The value of high-sensitive troponin in systemic sclerosis patients

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Background and Aim: Cardiac involvement in systemic sclerosis (SSc) carries a poor prognosis. Few studies have examined the role of high sensitive cardiac troponin levels in SSc patients. The aim of this study was to explore the relation between echocardiographic abnormalities and cardiac biomarkers in patients with SSc. **Methods:** Fifty patients with SSc (mean age: 49.6±10.1 years, 45 female) and 25 age and sex matched controls (mean age: 44.4±11.7 years, 18 female) were consecutively enrolled into the study. All patients underwent 2D conventional and speckle-tracking echocardiography. Right ventricular (RV) size, fractional area change (FAC), tricuspid annular plane systolic excursion (TAPSE), tricuspid annular systolic velocity (RVs), as well as biventricular global myocardial strain were measured. Plasma HS-cTnT and NT-proBNP concentrations were measured using the electrochemiluminescence method and sandwich immunoassay, respectively. **Results:** HS-cTnT levels were significantly elevated in SSc patients compared to controls (5.6±3.8 ng/L vs 9.2±5.9 ng/L, p=0.012). Twenty percent of SSc patients had systolic pulmonary artery pressure ≥40 mm Hg. SSc patients with pulmonary hypertension (PHT) had the highest HS-cTnT levels 13.1±6.8 ng/L compared to PHT (-) SSc group 8.4±5.1 ng/L (p=0.030). The NT-proBNP levels were also statistically higher in SSc patients (265.9±647.2 pg/ml vs 49.7±46.5 pg/ml, p=0.024). The univariate correlation analysis of HS-cTnT levels with echocardiographic parameters are listed in Table 1. Among all significant variables, left ventricular global strain is the only independent predictor of increased HS-cTnT levels in systemic sclerosis patients (β = -0.52 CI: -0.97 - -0.062, p=0.027). **Conclusions:** HS-cTnT increases in patients with systemic sclerosis even before the development of pulmonary hypertension. The only independent predictor of increased HS-cTnT level is decreased left ventricular longitudinal strain. Patients with increased HS-cTnT levels need to be closely monitored for subtle ventricular dysfunction.

Table 1. Univariate correlation analysis of plasma HS-cTnT and echocardiographic parameters

	r	p
LV-Global Longitudinal Strain	-0.42	<0.001
RV-Global Longitudinal Strain	-0.27	0.025
LA-Peak Longitudinal Strain	-0.39	<0.001
RA- Peak Longitudinal Strain	-0.36	0.001
RV Fractional Area Change	-0.33	0.002
TAPSE	-0.18	0.096
Systolic Pulmonary Artery Pressure	0.48	<0.001

LV: left ventricle, RV: right ventricle, LA: left atrium, RA: right atrium, TAPSE: tricuspid annular plane systolic excursion

Other

OP-119

Heart sound recognition with deep neural networks

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Background and Aim: This project aims to use machine learning algorithms for recognition and interpretation of heart sounds with high accuracy. Heart sounds are the mechanical waves generated by the natural physiologic movements of heart parts and the resultant blood flow and circulation through it. Unique and distinct mechanical waves provide significantly important auditory data regarding the condition of the heart in cardiac auscultation. S1 (first heart sound) and S2 (second heart sound) are described as two normal heart sounds for healthy adults. These mechanical waves of heart is produced by the closing of atrioventricular valves and semilunar valves. Beside these, there are a variety of heart sounds such as heart murmurs, adventitious sounds, and gallop rhythms S3 and S4. Interpretation and recognition of heart sounds requires expertise and experience for physicians. Human failure in recognition and interpretation process can be tolerated with further examinations. Cardiac auscultation is fundamental method used in medicine for understanding heart condition regarding auditory data of heart. Artificial intelligence algorithms holds great promise in medicine for detecting abnormalities in heart sounds.

Methods: Heart sounds are recorded from auscultation areas of heart: Aortic area, Pulmonic area, Tricuspid area, Mitral Area (Apex). All recorded sounds are obtained from voluntary patients of Bezmialem Vakif University Cardiology Outpatient Clinic by the Littmann electronic stethoscope in the supine position (lying back down). All heart sound records are exported as microsoft wav. format. 212 heart sounds are interpreted by two cardiology specialist, skilled with grand expertise and experience, labeled records as normal and abnormal. And then, they proceed the 212 heart sound records from 53 patients as 44 abnormal and 168 normal heart sound records. Heart sound signals are cropped to obtain 5 second segments. After that, MFCC (Mel Frequency Cepstral Coefficients) features are extracted from heart sound signals. Deep convolutional neural networks are used for classification of MFCC heat map. The Challenge training set consists of five databases (A through E) containing a total of 3,126 heart sound recordings, lasting from 5 seconds to just over 120 seconds from PhysioNet/CinC Challenge 2016 used for training with 92% accuracy results.

Results: Our clinical data test records resulted 81% accuracy. This study aimed to provide a more efficient and accurate process of heart sound recognition by the computer based diagnosis. This project proposes an efficient and effective method for extracting features for heart sound recognition process.

Conclusions: According to recent studies, cardiologists have approximately 70% accuracy of interpretation of heart sounds from auscultation data. Artificial intelligence has future in medicine for diagnosis and education. In this study, artificial intelligence proved to be very successful for use in medicine.

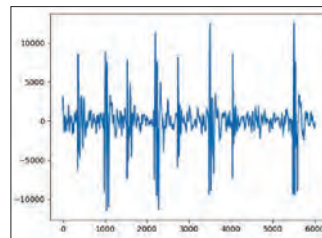


Figure 1. Normal heart sound signal.

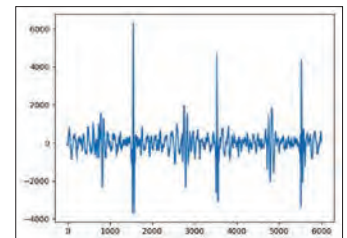


Figure 2. Abnormal heart sound signal.

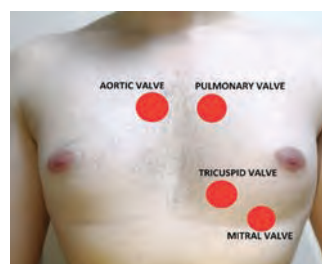


Figure 3. Heart sound auscultation areas: Aortic area, Pulmonic area, Tricuspid area, Mitral Area (Apex).



Figure 4. Ethics committee approved heart sound recording from voluntary patient with electronic stethoscope.

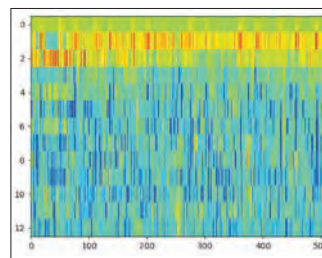


Figure 5. Normal heart sound signal MFCC heat map visualization.

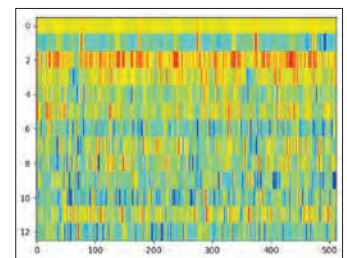


Figure 6. Abnormal heart sound signal MFCC heat map visualization.

Table 1. PhysioNet/CinC Challenge 2016 heart sound record data set training results

HYPER PARAMETERS	VALUE
Learning rate	1e-4
Optimizer	Adam Optimization
Weight decay	1e-5
Dropout	0.1
Batching Size	No batching
Number of epochs	600

Other

OP-120

The acute impact of hemodialysis on high-sensitive troponin t level

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Background and Aim: Circulating troponin levels are higher in patients with end-stage renal disease, even in the absence of acute coronary syndrome. In these patients, underlying cardiac problems that frequently lead to troponin elevation are also common. However, the relationship between the renal dysfunction and the circulating troponin levels is still not fully understood. Moreover, the effect of dialysis on troponin levels has not been elucidated well. Circulating troponin levels have been found to be elevated but stable in this population. However, there are not many studies examining the acute effect of dialysis on troponin levels and the existing studies show varying results. We planned to investigate the relationship between the changes in troponin levels and the volume depletion in patients with end-stage renal disease.

Methods: Patients between 18 and 85 years of age, receiving HD for at least 6 months were included. High-sensitive troponin T (hs-Tn T) levels were studied in blood samples taken at the beginning and at the end of dialysis. Hs-TnT levels were measured with a high sensitive troponin assay with lower detection and upper reference (99th percentile) limits of 3 and 14 ng/L, respectively. The changes in circulating hs-Tn T levels before and after hemodialysis were examined. Correlation between volume depletion and change in hs-Tn T levels were also calculated.

Results: 70 patients (50.7±16.9 years of age, 27 women) were included in study. The mean volume of ultrafiltration was 3260±990 ml. We observed a significant increase in circulating hs-cTn T levels and a prominent decrease in left ventricular global longitudinal strain (GLS) after HD (52.4±40.2 ng/l vs. 66.8±48.5 ng/l, p<0.001 and 20.1±3.6% vs. 16.8±3.8% p<0.001, respectively). Relative change in hs-cTnT and ultrafiltrated volume showed a significant correlation (R=0.74, p=0.001)

Conclusions: Hemodialysis can cause a significant increase in hsTn T. This can jeopardize the accuracy of clinical diagnoses based on the hsTn T. It is therefore important to consider effect of hemodialysis to prevent misdiagnosis.

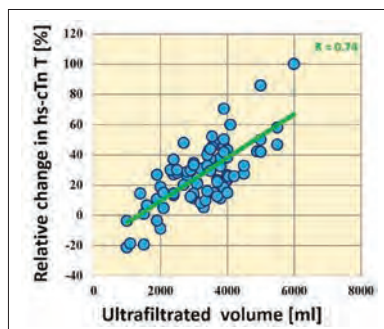


Figure 1. Comparison between relative change of high-sensitive cardiac troponin T and the ultrafiltrated volume. The correlation coefficient (R) is indicated. P<0.001. (Hs-cTn T: high-sensitive cardiac troponin T).

Other

OP-121

Full publication rates of the abstracts presented during the 30th Turkish Cardiology Congress with international participation

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Background and Aim: Many cardiology specialists related to cardiology worldwide attend to annual cardiology congresses in an attempt to improve their medical understandings and to catch up pace with the contemporary scientific data. In the present study, we aimed to determine the fate of the abstract presentations submitted in the 30th Turkish Cardiology Congress (30.TCC) held in 2014. Ours is the first study performed in Turkey, inquiring the fate of the abstracts presented in a Turkish Congress.

Methods: A total of 538 abstracts deemed eligible to be presented at the 30.TCC by the congress abstract evaluation committee were gathered from the published supplements in the Anatolian Journal of Cardiology. Full-text publications were searched through PubMed Medline, Scopus and Google Scholar, commencing from October 2014 to January 2018. We searched the abstracts on the basis of the methodology by Scherer et al. Once the data of the encountered full-text publication was ascertained to be thoroughly concordant with that of the previously presented abstract, then the full-text publication was included in the study. Upon gathering the information intended, we determined the publication rate the relevant abstracts, journal names, publication dates, publication categories and impact factors of the relevant journals, time-course of the publications.

Results: The study was identified that 273 (50%) of all 538 abstracts evaluated were published in full-text in peer-reviewed journals. 538 abstracts presented in the congress, 365 (67%) were poster presentation and 173 (33%) were oral presentations. The number of the abstract presentations, their presentation modalities, number and percent of subsequent full-text publications on the basis of subcategories were presented in Table 1. Median time to publication was 10.3 months. 239 (87.5%) of all 273 full-text publications were published in SCI and SCI-E indexed journals, while the remaining 34 were published in non-SCI-E indexed journals. Median impact factor of the journals was 1,39, with the highest impact factor to be 19,89 and the lowest impact factor to be 0,17.

Conclusions: This study found that one half of all the presented abstracts, either orally or as a poster, was subsequently published in full-text in peer-reviewed journals. We hope that our study may serve as a small catalyst for a further promotion in the rate of subsequent full publication in internationally indexed databases of future abstracts at future Turkish Cardiology Congresses.

Table 1. Publication rates of the abstracts presented in 30th Turkish Cardiology Congress with International Participation. The numbers and the percentages were given according to their cardiologic subcategories

	Oral Abstract Presentation (number)	Poster Abstract Presentation (number)	Total Number	Number of Full-text Publication (number)	Percentage(%)
Coronary Heart Diseases	41	73	114	59	51
Echocardiography	16	44	60	32	53
General Cardiology	15	40	55	33	60
Interventional Cardiology	10	26	36	11	30
Valvular Heart Disease	17	24	41	20	48
Arrhythmia	10	29	39	18	46
Non-invasive Arrhythmia	4	21	25	18	72
Hypertension	8	18	26	12	46
Heart Failure	13	18	31	20	64
Electrophysiology-Ablation	3	13	16	8	50
Cardiac Imaging	4	14	18	10	55
Others	-	8	8	3	37
Coronary Cases	-	8	8	3	37
Lipid	5	6	11	4	36
Pulmonary Hypertension	8	5	13	6	46
Pacemaker	1	4	5	4	80
Epidemiology	9	6	15	5	33
Pulmonary Vascular	1	1	2	0	0
Congenital Heart Disease	-	4	4	1	25
Peripheral Vascular	5	1	6	4	66
Cardiovascular surgery	1	-	1	0	0
Congestive Heart Failure	1	1	2	1	50
Pediatric Cardiology	-	1	1	1	1
Withdrawn	1	-	-	-	-
TOTAL	173	365	538	273	50

Coronary artery disease / Acute coronary syndrome

OP-122

The BUN, Age, contrast volume as predictors for the development of Contrast induced nephropathy in acute coronary syndrome patients who underwent percutaneous coronary intervention

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Background and Aim: Contrast-induced nephropathy (CIN) is a common and potential severe complication in patient with acute coronary syndromes (ACS) who were undergoing percutaneous coronary intervention

(PCI). Moreover it is significantly associated with increased mortality in these patients. The incidence of CIN ranges from 2% to 30% because of different study populations, different clinical settings and CIN definitions. Thus, identifying patients at risk CIN easily and accurately would allow the administration of prophylactic interventions to those at high risk. Blood urea nitrogen (BUN) is one of markers of kidney function. BUN may serve as a comprehensive marker reflecting impaired cardiology function and neurohormonal activation. Also BUN reflects both GFR and neurohormonal activations, it may serve as a marker of CIN compared with creatinine. It has been shown that contrast volume was associated with the development of CIN. Advanced age is one of risk factors of CIN. Our aim was to determine whether BUNxAgexContrast volume (BACV) is able to predict CI-AKI in patients in these patients.

Methods: A total of 1058 acute coronary syndromes patient were included in this retrospective study. BUNxAgexContrast volume was calculated as age, BUN multiplied by contrast volume. CIN was defined as a rise in serum creatinine of 0.3 mg/dL or a 25% increase from baseline value within 48-72 hours after contrast exposure. The patients were grouped as CIN or no CIN. The natural logarithmic transformations of BACV (Log BACV) was performed because of its extreme positive large value.

Results: A total of 81 patients developed CIN (7.7%). The patients in CIN group were significantly older (70.7±11 vs 60.7±12, p<0.001). BUN level was higher in patients with CIN than those without CIN (22.2±6.4 vs 17.9±8.6, p<0.001). Contrast volume was higher in CIN group compared with no CIN group (186±72 cc vs 160±55cc, p<0.001). Also, the patients with CIN was higher levels of Log BACV (5.4±0.2 vs 5.2±0.3, p<0.001). Multivariate analysis showed that history of stroke, left ventricular ejection fraction, killip class, and Log BACV (OR: 8.156 [1.907-34.885], p=0.005) were independent predictors of CIN. Areas under the ROC curve (95% CI) of Log BACV was 0.771 (0.726-0.816).

Conclusions: BACV is easily applicable with a useful predictive value in identify high-risk patients for CIN development in ACS patients treated with PCI.

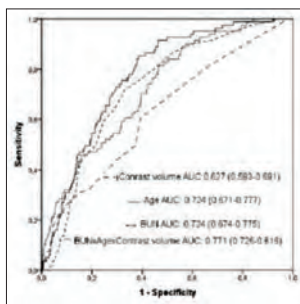


Figure 1. ROC.

Table 1. Univariate and multivariate regression analysis for CIN

Variables	OR (95% CI)	P-value	OR (95% CI)	P-value
Age* (year)	1.075 (1.053-1.098)	< 0.001		
Killip class ≥ 2	5.649 (3.213-9.933)	< 0.001	2.479 (1.017-6.044)	0.046
BUN* (mg/dl)	1.043 (1.022-1.064)	< 0.001		
Hemoglobin levels (mg/dl)	0.812 (0.729-0.905)	< 0.001		
Admission creatinine levels (mg/dl)	1.341 (1.084-1.659)	0.007		
LVEF (%)	0.938 (0.917-0.960)	< 0.001	0.935 (0.906-0.965)	< 0.001
Furosemide usage	4.267 (2.508-7.261)	< 0.001		
Statin usage	0.620 (0.356-1.079)	0.091		
Hypotension/positive inotrop	1.581 (1.374-1.819)	< 0.001		
Multi-vessel disease	2.080 (1.315-3.289)	0.002		
IABP	1.436 (1.228-1.681)	< 0.001		
Stroke/TIA	4.051 (1.905-8.614)	< 0.001	3.880 (1.558-9.658)	0.004
Hypertension	1.542 (0.958-2.481)	0.074		
Contrast volume* (cc)	1.005 (1.002-1.008)	< 0.001		
Male gender	0.475 (0.299-0.756)	< 0.001		
BACV	1.236 (1.177-1.298)	< 0.001	8.156 (1.907-34.885)	0.005

Table 1. Baseline clinical, demographic and echocardiographic characteristics of study population

	AF (-) N:684	AF (+) N:36	P value
Age	56.7±10.9	66.83±14.9	<0.01
Male (%)	85.7 %	69.4 %	<0.01
BMI(kg/m2)	27.78±5.49	27.82±5.82	0.98
Active Smoking %	59.2 %	30.6 %	<0.01
Prior CVA %	1.3 %	5.6 %	0.04
DM %	28.5 %	30.6 %	0.79
HT %	34.8 %	55.6 %	0.01
Anterior MI %	44.3 %	38.9 %	0.55
Ejection fraction (%)	47.8±9.3	42.6±13.9	<0.01
Left Atrial Diameter(cm)	3.76±0.5	4.30±0.5	<0.01
Ventricular Septum Thickness(cm)	1.11±0.3	1.37±0.9	<0.01
CHADS-VASC score	2.27±1.2	3.58±1.6	<0.01

CVA: cerebrovascular accident, DM: diabetes mellitus, HT: Hypertension, MI: myocardial infarction, BMI: body mass index.

Table 2. Primary endpoints long-term follow-up

	AF (-) N:684	AF (+) N:36	P value
Death %	7.2 %	10 %	<0.01
MI %	12.9 %	13.9 %	0.85
CVA %	0.4 %	2.8 %	0.06
Primary end points % (death, MI, CVA)	18.4 %	38.9 %	< 0.01

MI: myocardial infarction, CVA: cerebrovascular accident.

Table 3. Multivariable predictors of new-onset AF

	OR	%95 CI	p value
Male	0.86	0.28-2.60	0.78
Hypertension	1.56	0.52-4.75	0.43
Age	1.02	0.97-1.07	0.38
Ejection fraction (%)	1.01	0.96-1.06	0.79
Left Atrial Diameter	4.36	1.97-9.66	< 0.01
Ventricular Septum Thickness	1.92	0.73-1.95	0.49
CHADS-VASC score	1.24	0.69-2.23	0.46

Coronary artery disease / Acute coronary syndrome

OP-123

Frequency of new-onset atrial fibrillation in ST segment elevated acute coronary syndrome: In the primary PCI era

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Background and Aim: Atrial fibrillation (AF) in ST segment elevation myocardial infarction (STEMI) is one of the rhythm disorder that can occur frequently. The new-onset AF during STEMI leads to significant deterioration in prognosis compared to patients without AF. In the thrombolytic era, the development frequency of AF in the course of STEMI was determined as 7-21%. We aimed to investigate the frequency of new-onset AF during STEMI who undergoing primary percutaneous coronary intervention.

Methods: 720 patients with STEMI who undergoing primary percutaneous coronary intervention were enrolled. Patients were categorized into two groups according to whether new-onset atrial fibrillation developed during hospitalization. Primary endpoints were defined as all-cause mortality, MI and cerebrovascular event (CVE) at 1 year follow-up.

Results: Results are displayed in Table 1, 2 and 3. The incidence of new-onset AF in STEMI patients with undergoing PCI was 5%. 52.7% of developed AF was returned to normal sinus rhythm after revascularization.
Conclusions: In our study, the incidence of new-onset AF in STEMI patients with undergoing primary PCI was found to be lower than thrombolytic era. This situation may be associated with effect of the treatment of new drugs and rapid revascularization of primary PCI.

Coronary artery disease / Acute coronary syndrome

OP-124

The effect of coronary artery disease categories on ventricular repolarization reflecting by Tp-e interval, Tp-e/QT and Tp-e/QTc ratios

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Background and Aim: Coronary artery disease (CAD) can cause electrical heterogeneity on ventricular myocardium and ventricular arrhythmia due to myocardial ischemia linked to ventricular repolarization abnormalities. In this study, our aim is to investigate the impact of increased level of CAD spectrum on ventricular repolarization via Tp-e interval, Tp-e/QT and Tp-e/QTc ratios.

Methods: 127 patients with normal coronary artery (group 1), 129 patients with stable CAD (group 2) and 121 patients with acute coronary syndrome (group 3) were enrolled in this study. Tp-e interval, Tp-e/QT and Tp-e/QTc ratios were evaluated on surface electrocardiography (ECG) for each patient.

Results: Tp-e interval [66 (50-83), 71 (59-82) and 76 (64-86); group 1, 2 and 3 respectively, p<0.001], Tp-e/QT (0,170,02, 0,180,01 and 0,190,01; group 1, 2 and 3 respectively, p<0.001) and Tp-e/QTc (0,150,02, 0,160,02 and 0,170,02; group 1, 2 and 3 respectively, p<0.001) ratios were found to be associated with increased level of CAD spectrum. Compared to group 2, group 3 had prolonged Tp-e interval and increased Tp-e/QT and Tp-e/QTc ratios, while group 2 had increased parameters than group 1.

Conclusions: Myocardial ischemia presence is one the most important reasons of abnormal ventricular

repolariation which is reflected on surface ECG by Tp-e interval, Tp-e/QT and Tp-e/QTc ratios. Although it is well known that increased level of these parameters are associated with ventricular repolarization abnormalities and ventricular arrhythmias, the relationship between CAD spectrum and repolarization markers has not been investigated yet. Unsurprisingly, in our study, prolonged Tp-e interval and increased Tp-e/QT and Tp-e/QTc ratios were detected in the presence of CAD and also in patients with unstable coronary atherosclerosis. These are valuable indicators of the association between increased level of CAD spectrum and damaged ventricular repolarization because of the myocardial injury. In conclusion, progressive coronary atherosclerosis and unstable disease make ventricular repolarization critical and it seems to be correlated with repolarization markers such as Tp-e interval, Tp-e/QT and Tp-e/QTc ratios on surface ECG.

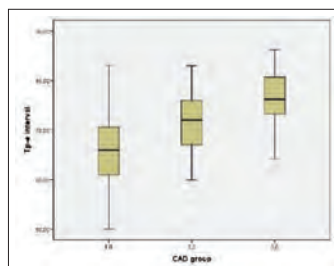


Figure 1. Comparisons of Tp-e interval between CAD groups. CAD: coronary artery disease.

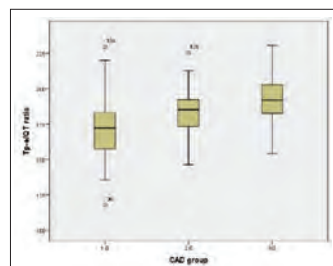


Figure 2. Comparisons of Tp-e/QT ratio between CAD groups. CAD: coronary artery disease.

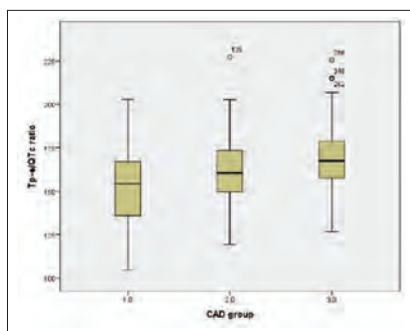


Figure 3. Comparisons of Tp-e/QTc ratio between CAD groups. CAD: coronary artery disease.

Table 1. Comparison of the effects of coronary artery disease spectrum on electrocardiographic variables

	Control group (n=127)	Stable CAD (n=129)	ACS (n=120)	P
Heart rate (beat/min)	79 (50-138)	79 (57-97)	80 (58-110)	0,374
QRS duration (ms)	84 +- 8,8	89 +- 11,1	87 +- 9,1	<0,001
QT interval (ms)	381 (357-438)	390 (362-444)	396 (369-424)	<0,001
QTc interval (ms)	432 (352-591)	446 (365-553)	450 (373-556)	<0,001
Tp-e interval (ms)	66 (50-83)	71 (59-82)	76 (64-86)	<0,001
Tp-e/QT ratio	0,17 +- 0,02	0,18 +- 0,01	0,19 +- 0,01	<0,001
Tp-e/QTc ratio	0,15 +- 0,02	0,16 +- 0,02	0,17 +- 0,02	<0,001

CAD: coronary artery disease, ACS: acute coronary syndrome.

Table 2. Comparison of electrocardiographic parameters between CAD groups

	Group 1-2	Group 1-3	Group 2-3
QRS duration (ms)	<0,001*	0,023*	0,309
QT interval (ms)	0,001*	<0,001*	0,047*
QTc interval (ms)	0,048*	<0,001*	0,355
Tp-e interval (ms)	<0,001*	<0,001*	<0,001*
Tp-e/QT ratio	<0,001*	<0,001*	<0,001*
Tp-e/QTc ratio	0,002*	<0,001*	0,005*

CAD: coronary artery disease.

Coronary artery disease / Acute coronary syndrome

OP-125

Short term outcomes of non-guideline-concordant treatment in patients with multivessel coronary artery disease

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Background and Aim: In this study we aimed to find the prevalence and 30-day mortality of patients with multivessel coronary artery disease who did not receive guideline-concordant revascularization following index coronary angiography.

Methods: All consecutive patients who underwent coronary angiography due to stable angina pectoris or non-ST-elevation acute coronary syndrome and finally diagnosed to have multivessel coronary artery disease at our hospital between August 2017 and February 2018 prospectively included in this study. Multivessel coronary artery disease was defined as follows: Isolated or non-isolated unprotected left main coronary artery disease (stenosis ≥50%), 70% or greater stenosis in left anterior descending artery and 70% or greater stenosis in at least one other major epicardial vessel. Standalone medical treatment, percutaneous coronary intervention (pci), coronary artery bypass grafting (cabg) or hybrid revascularization are recommended treatment methods for appropriate patients and conditions by The European Society of Cardiology and The European Association for Cardio-Thoracic Surgery Guidelines on Myocardial Revascularization. Primary end point was 30-day all-cause mortality. A p<0.05 was considered to be statistically significant.

Results: Overall 140 patients (96 male, 68.6%) included study. Of these, 65 (46.4%) received non-guideline-concordant (group 1) whereas 75 (53.6%) received guideline-concordant treatment (group 2). Sociodemographic and clinical characteristics did not differ statistically between two groups (Table 1). LVEF was lower (p=0.017) whereas SYNTAX I score (p<0.001) and SYNTAX II score (p<0.001) were higher in the group 1. The reasons of receiving non-guideline-concordant treatment were patient's preference and/or cardiologist's decision pci rather than cabg (42, 64.6%), refusal of any revascularization method by patient (14, 21.5%), operation refusal by surgery team due to advanced patient age or low left ventricular ejection fraction (5, 7.7%) unavailability of surgery team (1, 1.5%), developing ischemia/myocardial infarction during waiting period (3, 4.6%). Thirty-day mortality tend to be lower in the group 1, however this was not statistically significant (p=0.188).

Conclusions: Prevalence of non-guideline-concordant revascularization is high. Although short term mortality was not statistically different between two groups, long term follow-up may reveal a significant difference.

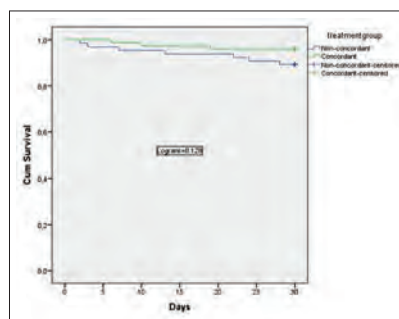


Figure 1. Kaplan-Meier curves for 30-days survival of two groups.

Table 1. Sociodemographic and clinical characteristics of the patient groups

	Group 1 (Non-concordant, n=65)	Group 2 (Concordant, n=75)	P
Age (years) ± SD	65 ± 11	62 ± 8.5	0,113
Sex, male, n(%)	24 (36.9)	21 (28.0)	0,260
Economic status			
Low, %	25 (38.5)	34 (45.3)	0,644
Intermediate, %	27 (41.5)	27 (36.0)	
High, %	13 (20.0)	14 (18.6)	
Living area			
Urban	43 (66.2)	44 (58.7)	0,628
Rural	22 (33.8)	31 (41.3)	
Education Level, n (%)			
Illiterate	39 (60.0)	49 (65.3)	0,509
Primary school	21 (32.3)	18 (24.0)	
High school and University	5 (7.7)	8 (10.7)	
Diabetes mellitus, n(%)	30 (46.2)	28 (37.3)	0,291
Peripheral Arterial Disease, n(%)	6 (9.2)	4 (5.3)	0,372
Chronic Kidney Disease, n(%)	3 (4.6)	2 (2.7)	0,535
Smoking, n(%)	19 (29.2)	26 (34.7)	0,492
Family History of Coronary Artery Disease, n(%)	11 (16.9)	10 (13.3)	0,533
Systolic Blood Pressure, mmHg	139 ± 21	138 ± 22	0,920
Diastolic Blood Pressure, mmHg	84 ± 13	83 ± 12	0,613
LVEF, %	48 ± 8	52 ± 7	0,017
Number of Diseased Vessels	2.9 ± 0.5	2.9 ± 0.6	0,879
SYNTAX I Score	22.1 ± 6.3	17.7 ± 6.4	<0,001
SYNTAX II SCORE for PCI	37.4 ± 9.3	30.9 ± 7.9	<0,001
SYNTAX II SCORE for CABG	25.3 ± 9	25.8 ± 7.9	0,303
Mortality, n (%)	7 (10.8)	3 (4.0)	0,188

SD: Standart deviation LVEF: Left ventricular ejection fraction SYNTAX: The Synergy between percutaneous coronary intervention with Taxus and cardiac surgery.

Coronary artery disease / Acute coronary syndrome

OP-126

Predictors of new-onset atrial fibrillation in elderly patients with acute coronary syndrome undergoing percutaneous coronary intervention

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Background and Aim: The development of atrial fibrillation (AF) during the course of acute coronary syndrome (ACS) is related to poor prognosis. Possible predictors of new-onset AF (NOAF) have not been adequately investigated in elderly patients with ACS undergoing percutaneous coronary intervention (PCI). We aimed to identify the factors associated with NOAF in such patients.

Methods: A total of 308 elderly patients with ACS undergoing PCI were enrolled in the study. Patients were divided into two groups: Without NOAF [254 patients, 64.6% men, age: 73.5 (69.0-79.0) years] and with NOAF [54 patients, 70.4% men, age: 75.0 (68.7-81.2) years]. Clinical, angiographic, and laboratory features including neutrophil to lymphocyte ratio (NLR), monocyte to high-density lipoprotein ratio (MHR), and mean platelet volume (MPV) were compared between the groups. Venous blood samples were drawn from antecubital veins immediately after the electrogram recordings. The NLR, MHR, and MPV were obtained using the same blood samples obtained before PCI. The patients were followed-up prospectively for six months.

Results: The percentages of prior myocardial infarction (MI), Killip III/ IV and triple vessel disease were higher, and left ventricular ejection fraction was lower in the patients with NOAF compared to the others (p=0.020, <0.001, 0.006, and 0.006, respectively) (Table 1). Estimated glomerular filtration rate was lower (p=0.009), peak troponin-T, preprocedural NLR, MHR, and MPV were higher in patients with NOAF (p=0.004, 0.030, <0.001 and 0.014, respectively). Rates of contrast induced nephropathy and six-month overall mortality were higher in the patients with NOAF compared to the others (Table 1). In binary logistic regression analysis, prior MI and preprocedural MHR were independent predictors of NOAF (p= 0.003 and <0.001, respectively) (Table 2). The receiver operating characteristic curve analysis showed that the best cut-off point of MHR was 15.87 to identify patients with NOAF (Sensitivity: 75.9%, specificity: 65.0%, p<0.001) (Figure).

Conclusions: NOAF is associated with an increased overall mortality in elderly patients with ACS undergoing PCI. Prior MI and preprocedural MHR are independent predictors of NOAF in such patients. They may help to early identify elderly patients with ACS having the highest risk of NOAF to prevent poor outcomes.

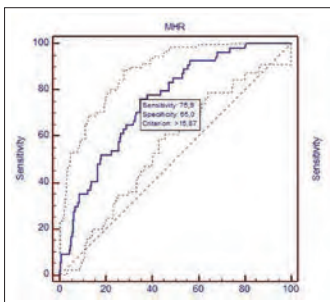


Figure 1. Receiver operating characteristic curve analysis suggested that the optimal MHR level cut-off point for patients with NOAF was 15.87 with a sensitivity and specificity of 75.9% and 65.0%, respectively (Area under curve: 0.750; 95% confidence interval: 0.698–0.798). MHR: Monocyte-to-high-density lipoprotein ratio, NOAF: new-onset atrial fibrillation.

Table 1. Baseline clinic and laboratory features, and clinical endpoints of the groups

	NOAF (-) (n=254)	NOAF (+) (n=54)	P value
Age (years)	73.5 (69.0-79.0)	75.0 (68.7-81.2)	0.414
Sex (male) (n,%)	164 (64.6)	38 (70.4)	0.415
Diabetes mellitus (n,%)	101 (39.8)	16 (29.6)	0.164
Hypertension (n,%)	142 (55.9)	28 (51.9)	0.586
Prior MI (n,%)	15 (5.9)	11 (20.4)	0.002
STEMI (n,%)	164 (64.6)	40 (74.1)	0.180
Killip III/ IV (n,%)	18 (7.1)	13 (24.1)	<0.001
LV EF (%)	45.0 (33.0-55.0)	36.5 (27.0-48.5)	0.006
LA diameter (mm)	40.3 ± 5.3	40.7 ± 5.1	0.575
MR (grade >II/IV)	36 (14.2)	9 (16.7)	0.638
Triple vessel disease (n,%)	90 (35.4)	30 (55.6)	0.006
eGFR (mL-1 min-1 1.73 m-2)	69.7 ± 21.6	61.3 ± 20.4	0.009
Peak troponin-T (ng/mL)	1.7 (0.4-5.8)	3.9 (1.6-7.7)	0.004
NLR	3.2 (2.0-6.0)	4.5 (2.6-7.2)	0.030
MHR	12.9 (9.9-18.5)	19.4 (15.7-26.5)	<0.001
MPV (fl)	8.6 (7.8-9.5)	9.1 (8.3-9.9)	0.014
CIN (n,%)	42 (16.5)	16 (29.6)	0.025
Six-month overall mortality (n,%)	34 (13.4)	14 (25.9)	0.021

CIN, contrast induced nephropathy; eGFR, estimated glomerular filtration rate; LA, left atrium; LV EF, left ventricular ejection; MHR: monocyte to high-density lipoprotein ratio; MI: myocardial infarction; MPV, mean platelet volume; MR, mitral regurgitation; NLR: neutrophil to lymphocyte ratio; NOAF, new-onset atrial fibrillation; STEMI: ST-segment elevation myocardial infarction.

Table 2. Independent predictors of new-onset atrial fibrillation.

	OR	95%CI	P value
Prior MI	4.509	1.679-12.106	0.003
Killip III/IV	2.008	0.769-5.243	0.155
Triple vessel disease	1.813	0.900-3.655	0.096
CIN	1.115	0.500-2.488	0.791
eGFR	0.994	0.977-1.011	0.503
Peak troponin-T	1.071	0.964-1.189	0.200
NLR	1.048	0.983-1.117	0.150
MHR	1.102	1.054-1.152	<0.001
MPV	1.120	0.861-1.457	0.399

CI: confidence interval; CIN, contrast induced nephropathy; eGFR, estimated glomerular filtration rate; MHR: monocyte to high-density lipoprotein ratio; MI, myocardial infarction; MPV, mean platelet volume; NLR: neutrophil to lymphocyte ratio, OR: odds ratio.

Coronary artery disease / Acute coronary syndrome

OP-127

The predictive value of GRACE risk score on the left ventricular ejection fraction after acute anterior ST-segment myocardial infarction

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Background and Aim: Even though rapid and complete reperfusion in acute myocardial infarction (AMI), inadequate recovery of left ventricular (LV) function may result in a decrease in LV ejection fraction (LVEF). To identify patients with high risk of heart failure after AMI by calculating GRACE risk score would have important impact on mortality and morbidity. In this study, we aimed to investigate the predictive value of GRACE score in LVEF after acute anterior segment AMI.

Methods: We retrospectively analyzed 710 patients presented with AMI and 290 patients were included after exclusion criteria. Patients were divided into two groups according to LVEF and a value <50% was defined as depressed EF group (group 1), a value ≥50% was defined as preserved EF group (group 2). The GRACE risk scores of all patients were calculated.

Results: 132 patients were included in the group 1, 158 patients were included in the group 2. In-hospital death GRACE risk score (165.5±28.3 vs. 136.6±26.7, <0.001) and in-hospital death/MI GRACE risk score (228.0 [198.0-252.0] vs. 203.2 [183.5-216.2], <0.001) were higher in group 1. A significant negative correlation was found between risk scores and LVEF (rho: -0.518, p<0.001 and rho: -0.440, p<0.001, respectively). In multivariate regression analysis, in-hospital death risk score (OR: 1.10; 95% CI: 1.05-1.15; p<0.001), and in-hospital death/MI risk score (OR: 0.96; 95% CI: 0.92-1.00; p=0.032) were found to be independently predictors of depressed LVEF.

Conclusions: GRACE risk score has a clinically important role predicting depressed LVEF in acute anterior segment AMI patients treated with primary PCI.

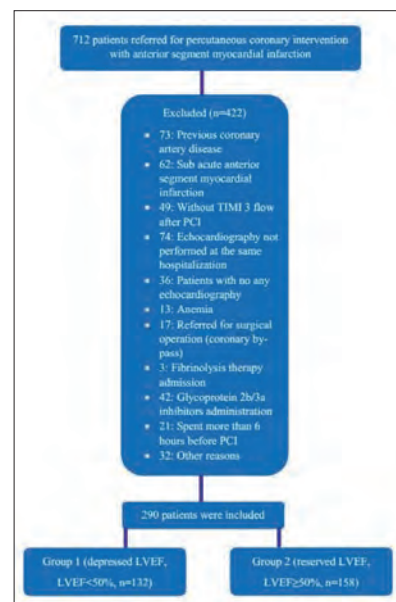


Figure 1. Flow diagram of patient selection.

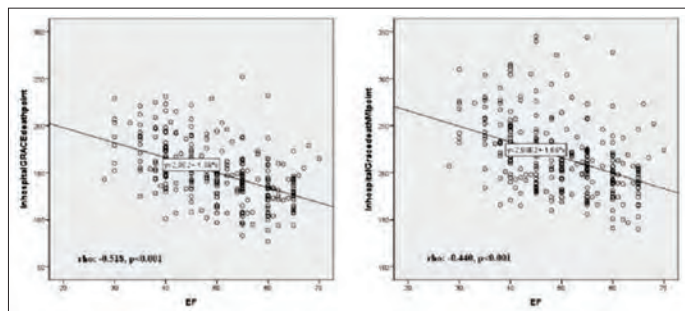


Figure 2. Correlation analysis of in-hospital death score and depressed EF (A), of in-hospital death/MI score and depressed EF (B) were performed by spearman rank correlation analysis test and presented by using scatter dot analysis.

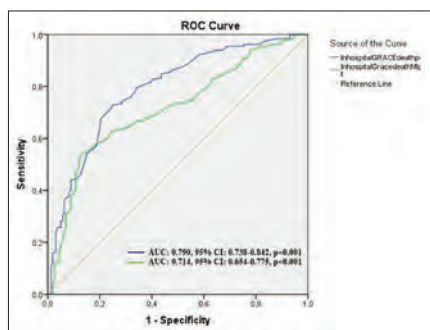


Figure 3. Receiver operating characteristic (ROC) curve analysis to identify depressed EF. The cut-off level of in-hospital death risk score was set at 143, the cut-off level of in-hospital death/MI risk score was set at 196.

Table 1. Basal characteristics and demographics features in the study groups

Variables	EF< 50 (n: 132)	EF≥ 50 (n: 158)	p value
Age (years)	60.2±13.8	55.6±10.9	0.002
Male n (%)	104 (78.8)	121 (76.6)	0.674
BMI (kg/m2)	27.2 (24.3-29.4)	26.9 (24.2-28.8)	0.733
DM n (%)	51 (38.6)	48 (30.4)	0.171
HT n (%)	75 (56.8)	68 (43.0)	0.025
HPL n (%)	47 (35.6)	51 (32.3)	0.618
Current smoker n (%)	55 (41.7)	74 (46.8)	0.407
Glucose (mg/dL)	169.7 (114.0-179.0)	155.7 (113.8-170.0)	0.436
Creatinine (mg/dL)	0.91 (0.8-1.0)	0.9 (0.8-1.0)	0.297
HB (g/dL)	14.9 (13.7-15.9)	14.8 (13.9-16.0)	0.949
HTC (%)	44.2±4.7	43.5±4.1	0.342
Neutrophil (x109/L)	9.78 (7.3-11.0)	7.79 (5.9-9.1)	0.021
Lymphocyte (x109/L)	2.34 (1.5-3.1)	3.0 (1.7-3.7)	0.002
Platelet (x109/L)	239.1 (190.5-285.0)	253.5 (213.5-312.0)	0.020
RDW (%)	13.7 (12.8-14.3)	13.5 (12.8-14.0)	0.159
MPV (fl)	8.3±1.2	8.0±0.9	0.061

Variables are presented as n (%), mean (SD), or median (range) Abbreviations: EF: ejection fraction; BMI: body mass index; DM: diabetes mellitus; HT: hypertension; HPL: hyperlipidemia; HB: hemoglobin; HTC: hematocrit; RDW: red cell distribution width; MPV: mean platelet volume.

Table 2. Echocardiography and coronary angiography findings

variables	EF< 50 (n: 132)	EF≥ 50 (n: 158)	p value
LVEF (%)	41.2 (38.0-45.0)	57.5 (54.8-61.0)	<0.001
LVEDD (mm)	51.1 (47.3-54.0)	47.3 (45.0-50.0)	<0.001
LVESD (mm)	39.1 (35.0-42.0)	31.4 (29.0-34.0)	<0.001
LAD (mm)	39.4±4.6	36.9±3.3	<0.001
Lesion localization n (%)			
Proximal	97 (73.5)	100 (63.3)	0.258
Medial	34 (25.8)	55 (34.8)	0.218
Distal	1 (0.8)	2 (1.3)	0.108
Pre-PCI TIMI flow n (%)			
TIMI 0	85 (64.4)	66 (41.8)	0.002
TIMI 1	20 (15.2)	41 (25.9)	0.002
TIMI 2	25 (18.9)	45 (28.5)	<0.001
TIMI 3	2 (1.5)	6 (3.8)	<0.001
In-hospital death score	165.5±28.3	136.6±26.7	<0.001
In-hospital death/MI score	228.0 (198.0-252.0)	203.2 (183.5-216.2)	<0.001

Variables are presented as n (%), mean (SD), or median (range) LVEF: left ventricular ejection fraction; LVEDD: left ventricular end diastolic diameter; LVESD: left ventricular end systolic diameter; LAD: left atrial diameter; PCI: percutaneous coronary intervention; TIMI: thrombolysis in myocardial infarction score; in-hospital death score: in-hospital 6 months mortality risk; in-hospital death/MI score: in-hospital 1 year mortality and recurrent myocardial infarction risk.

Table 3. Spearman Rank Correlation (R) between LVEF and in-hospital death, in-hospital death/MI

variables	R	p
LVEF and in-hospital death score	-0.518	<0.001
LVEF and in-hospital death/MI score	-0.440	<0.001

Abbreviations: LVEF: left ventricular ejection fraction; in-hospital death score: in-hospital 6 months mortality risk; in-hospital death/MI score: in-hospital 1-year mortality and recurrent myocardial infarction risk

Table 4. The predictors of low EF in binary logistic regression analysis

Variables	Unadjusted OR (95 % CI)	p	Adjusted OR (95 % CI)	p
Age	1.03 (1.01-1.05)	0.002	0.94 (0.89-0.99)	0.015
HT	1.74 (1.9-2.78)	0.02	1.70 (0.72-3.99)	0.224
KILLIP score	2.76 (1.83-4.18)	<0.001	1.30 (0.45-3.78)	0.633
Pre-PCI TIMI flow	0.63 (0.48-0.82)	<0.001	0.79 (0.50-1.25)	0.313
In-hospital death score	1.02 (1.02-1.03)	<0.001	1.10 (1.05-1.15)	<0.001
In-hospital death/MI score	1.02 (1.02-1.03)	<0.001	0.96 (0.92-1.00)	0.032
HTC	1.06 (0.99-1.11)	0.053	1.12 (1.07-1.24)	0.023
MPV	1.23 (0.99-1.53)	0.059	1.09 (0.75-1.57)	0.663
Neutrophil	1.00 (1.00-1.00)	0.002	1.00 (1.00-1.00)	0.486
Lymphocyte	1.00 (1.00-1.00)	<0.001	1.00 (0.99-1.01)	0.001
LVEDD	1.15 (1.09-1.22)	<0.001	0.74 (0.63-0.88)	<0.001
LVESD	1.45 (1.33-1.58)	<0.001	1.87 (1.55-2.24)	<0.001
LAD	1.18 (1.10-1.25)	<0.001	1.00 (0.90-1.12)	0.94

Abbreviations: EF: ejection fraction; OR: odds ratio; CI: confidence interval; HT: hypertension; PCI: percutaneous coronary intervention; TIMI: thrombolysis in myocardial infarction score; HTC: hematocrit; MPV: mean platelet volume; LVEDD: left ventricular end diastolic diameter; LVESD: left ventricular end systolic diameter; LAD: left atrial diameter; in-hospital death score: in-hospital 6 months mortality risk; in-hospital death/MI score: in-hospital 1 year mortality and recurrent myocardial infarction risk.

Pulmonary hypertension / Pulmonary vascular diseases

OP-128

Impaired six-minute walk test performance in patients with Systemic Sarcoidosis: Is there any effect of subclinical cardiac involvement?

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Background and Aim: Systemic Sarcoidosis (SS) is a multisystem and granulomatous disease known with impaired functional capacity associated with pulmonary and cardiac involvement or pulmonary hypertension (PH). The affecting factors of functional capacity in patients with SS have not been systematically assessed. We aimed to evaluate the factors that affect functional capacity in patients with SS who do not clinically manifest cardiac involvement.

Methods: 56 (18% male) extracardiac biopsy proven SS patients with a mean age 52.5±10.7 years, and 26 age and gender similar controls (mean age 48.8±7.1 years, 23% male) were included consecutively. The patients' functional capacities were assessed with six-minute walk test (6-MWT). Affecting factors of functional capacity were determined by pulmonary function tests (%FEV1 and %FVC values) and two dimensional (2D) standard transthoracic and speckle tracking echocardiogram (STE) parameters. Linear regression analyses were then performed to assess the independent correlates 6 minutes walk distance in the total patient population.

Results: Patients with SS had low 6-MWT performance and higher NYHA classes and NT-proBNP levels. There were no significant differences between the controls and SS patients in %FEV1 and %FVC values. Biventricular global strain levels (LV GLS, LVGCS and RV GLS) and atrial reservoir and conduit function values were lower and systolic pulmonary artery pressure (SPAP) was significantly higher in patients with SS. Only age (beta coefficient -0.20, 95% CI for B -3.5 - 0.079, p=0.05) and SPAP (beta coefficient -0.28, 95% CI for B -4.7 - 0.45, p=0.05) were found as independent predictors of poor 6-MWT performance.

Conclusions: Although biventricular global strain levels were lower in the patients with sarcoidosis, only age and SPAP elevations were independent predictors of the functional capacity.

Table 1. Demographic characteristics, cardiovascular risk factors and laboratory findings of the patients with sarcoidosis and controls

	Patients with sarcoidosis n 56	Controls n 26	P value
Age (years)	52.5 10.7	48.8 7,1	0.114
Gender male (n) (%)	10 (17.9)	6 (23.1)	0.565
BMI (kg/m ²)	30.2 5.2	28.2 4.4	0.095
Hypertension (n) (%)	11 (19.6)	2 (7.7)	0.209
Diabetes (n) (%)	12 (19.6)	2 (7.7)	0.206
Smokers (n) (%)	6 (10.7)	7 (26.9)	0.101
NT-proBNP (pg/ml)	80.1 (223) 531.6 1490.6	27.3 (49.8) 35.1 34.1	0.001
Uric acid (mg/dl)	5.3 1.6	4.2 1.2	0.003
Creatinine (mg/dl)	0.75 0.3	0.7 0.1	0.317
Hemoglobin (gr/dl)	13.2 1.7	13.3 1.2	0.681
FEV 1 (%)	91.1 20.2	98.3 8.9	0.08
FVC (%)	94.1 21.1	98.9 11.7	0.28
FEV1/FVC ratio	0.97 0.1	1.0 0.1	0.157
6 MWD (m)	425 88.6	501.9 48.9	0.001
BT sat (%)	96.8 3.3	98.6 0.6	0.009
AT sat (%)	96,6 4.5	98,8 0.5	0.018
O2 demand (n) (%)	3 (5.4)	-	0.548

6 MWD: six minutes walk distance; AT sat: After test oxygen saturation; BT sat: Before test oxygen saturation; FEV 1: Forced expiratory volume in 1 second; FVC: Forced vital capacity; NT-proBNP: N terminal probrain natriuretic peptide O2: Oxygen.

Table 2. Conventional transthoracic echocardiographic findings of the patients with sarcoidosis and controls

	Patients with sarcoidosis n 56	Controls n 26	P value
LVEDD (mm)	45.0 4.7	44.5 3.9	0.59
LVESD (mm)	28.9 5.8	27.6 3.9	0.31
LV EF (%)	64.5 8.8	66.5 5.5	0.28
RV basal diameter (mm)	30.2 3.7	29.3 4.0	0.32
RV/LV ratio	0.72 0.7	0.73 0.7	0.66
LA diameter (mm)	33,7 3,4	32,6 3,4	0.16
LAA (cm ²)	15,2 3,5	14,6 2,6	0.48
RAA (cm ²)	12,9 2,7	12,5 2,4	0.48
Septum (mm)	10,0 1,4	10,2 1	0.54
PW (mm)	9,5 1,1	9,7 1,0	0.48
LV mass (gr)	149,4 36,2	147,7 28,9	0.83
RV wall thickness (mm)	4,9 0,9	4,3 0,4	0.007
Transmitral E velocity (cm/sec)	0,8 0,2	0,8 0,1	0.59
Transmitral A velocity (cm/sec)	0,8 0,2	0,7 0,1	0.18
Transmitral E-wave DT (msec)	199,3 40,9	178,9 24,9	0.022
Transmitral E/A ratio	1,0 0,3	1,2 0,3	0.049
Mitral lateral E' (cm/sec)	11,2 3,7	13,0 3,5	0.03
Mitral lateral A' (cm/sec)	10,8 2,4	11,0 3,0	0.747
Mitral lateral S' (cm/sec)	10,7 2,9	10,6 2,2	0.925
E/E' ratio (cm/sec)	7,5 2,9	6,4 1,6	0.079
Tricuspid lateral S' (cm/sec)	13,0 2,4	13,9 2,2	0.09
TAPSE (cm)	22,9 4	25,5 3,4	0.005
RV FAC (%)	45,4 8,9	56,6 7,9	0.001
SPAP (mm Hg)	25,9 9,5	18,8 5,8	0.001

DT: Deceleration time; FAC: Fractional area change; LA: Left atrium; LAA: Left atrium area; LV: Left ventricular; LVEDD: LV end-diastolic diameter; LVESD: LV end-systolic diameter; PW: Posterior wall; RAA: Right atrium area; RV: Right ventricular; SPAP: Systolic pulmonary artery pressure; TAPSE: Tricuspid annular plane systolic excursion.

Table 3. Comparison of two-dimensional speckle tracking echocardiography parameters between patients with sarcoidosis and controls

	Patients with sarcoidosis n 56	Controls n 26	P value
LV GLS (- %)	16.7 4.1	22.8 3.2	0.001
LV GCS (- %)	19.1 5.7	18.1 4.4	0.001
RV GLS (- %)	17.0 5.2	23.4 3.2	0.001
LA reservoir function (%)	27.7 11.0	41.1 9.8	0.001
LA conduit function (%)	14.2 7.2	20.6 6.0	0.001
RA reservoir function (%)	27.4 10.2	40.5 8.4	0.001
RA conduit function (%)	13.7 6.5	20.3 5.2	0.001

LA: Left atrium; LV GLS: Left ventricular global longitudinal strain; LV GCS: LV global circumferential strain; RA: Right atrium; RV: Right ventricular; RV GLS: RV global longitudinal strain.

Pulmonary hypertension / Pulmonary vascular diseases

OP-129

Right ventricle outflow tract fractional shortening: A practical parameter for the assessment of right ventricular systolic function

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Background and Aim: The contractile function of the right ventricle (RV) is difficult to measure due to its complex anatomy. It has three distinctive parts such as inlet, trabeculated apical and right ventricle outflow tract (RVOT). The contribution of RVOT contraction to overall right ventricle systolic performance is not fully evaluated. The aim of the study is to compare RVOT contraction in systemic sclerosis (SSc) patients and healthy controls.

Methods: Fifty patients with SSc (mean age: 49.6±10.1 years, 45 female) and 25 age and sex matched controls (mean age: 44.4±11.7 years, 18 female) were consecutively enrolled into the study. All patients underwent 2D conventional and speckle-tracking echocardiography. RV size, fractional area change (FAC), tricuspid annular plane systolic excursion (TAPSE), tricuspid annular systolic velocity (RVs'), as well as RV global myocardial strain were measured. Measurement of RVOT fractional shortening (FS) is illustrated in Figure 1. M-mode recordings of RVOT both at systole and diastole were measured at the level of aortic level with the leading edge methodology.

Results: Ten patients in SSc group had pulmonary artery pressure (sPAP) ≥40 mmHg. RVOT-FS is significantly elevated in SSc patients with or without pulmonary hypertension compared to controls (Table 1). The RVOT-FS in controls, PHT(-) and PHT(+) SSc patients were as follows: 68.2%±15.6, 55.9%±17.9, 41.8%±14.8) RVOT-FS is significantly correlated with sPAP (r=-0.4 p<0.001), TAPSE (r=0.2 p=0.026) and with RV global longitudinal strain (r=0.2 p=0.049).

Conclusions: Not only longitudinal contraction but also right ventricle outflow tract contraction is impaired in patients with systemic sclerosis. RVOT-FS is inversely correlated with systolic pulmonary hypertension. This easy to measure echocardiographic parameter can be used as an additional tool for diagnosis of RV systolic dysfunction.

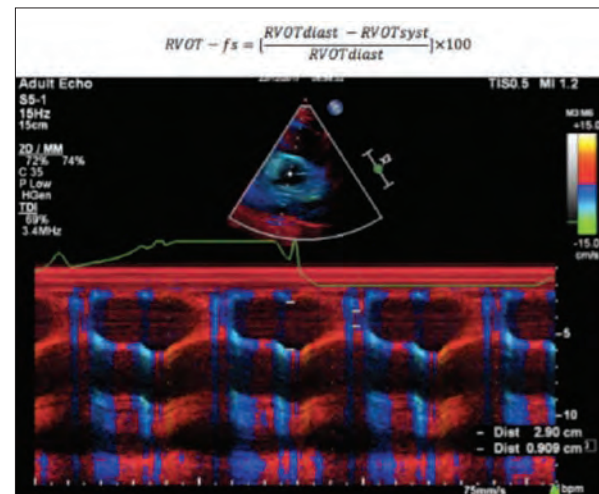


Figure 1. 1- Measurement of RVOT-FS. RVOT-FS: right ventricle outflow tract fractional shortening.

Table 1. Comparison of RVOT-FS with other 2D- echocardiographic parameters to assess right ventricle systolic function in systemic sclerosis patients

			Mean±SD	Mean±SD	p
sPAP mmHg	Control	PHT(-) SSc	20.6±6.7	29.9±6.2	<0.001
	Control	PHT(+) SSc	20.6±6.7	61.6±21.7	<0.001
	PHT(-) SSc	PHT(+) SSc	29.9±6.2	61.6±21.7	<0.001
RVs' cm/sec	Control	PHT(-) SSc	13.1±2.4	12.9±2.3	0.709
	Control	PHT(+) SSc	13.1±2.4	12.0±3.3	0.296
	PHT(-) SSc	PHT(+) SSc	12.9±2.3	12.0±3.3	0.383
TAPSE mm	Control	PHT(-) SSc	24.6±3.9	22.6±3.9	0.029
	Control	PHT(+) SSc	24.6±3.9	21.8±4.2	0.052
	PHT(-) SSc	PHT(+) SSc	22.6±3.9	21.8±4.2	0.547
FAC %	Control	PHT(-) SSc	53.5±8.5	46.7±9.7	0.001
	Control	PHT(+) SSc	53.5±8.5	38.8±14.5	0.003
	PHT(-) SSc	PHT(+) SSc	46.7±9.7	38.8±14.5	0.073
RVOT-FS %	Control	PHT(-) SSc	68.2±15.6	55.9±17.9	0.001
	Control	PHT(+) SSc	68.2±15.6	41.8±14.8	<0.001
	PHT(-) SSc	PHT(+) SSc	55.9±17.9	41.8±14.8	0.007

FAC: fractional area change; TAPSE: tricuspid annular plane systolic excursion; RVs': tricuspid annular systolic velocity; sPAP: systolic pulmonary artery pressure; RVOT-FS: right ventricle outflow tract fractional shortening

Pulmonary hypertension / Pulmonary vascular diseases

OP-130

Association of disease duration and pulmonary hypertension in patients with myeloproliferative neoplasms

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Background and Aim: Pulmonary hypertension (PH) can complicate the course of myeloproliferative neoplasms (MPN). The prevalence of PH may be underestimated. Echocardiography is a useful noninvasive screening test for PH in at-risk populations. We aimed to investigate the presence of PH and clinical characteristics of patients with MPN.

Methods: This study included 197 patients with MPN (mean age, 59±14 years old; women, 53%; mean disease duration, 3.4±2.8 years). Clinical and laboratory characteristics were obtained. All the participants underwent a comprehensive transthoracic echocardiographic (TTE) examination which included peak tricuspid regurgitation velocity, RV/LV basal diameter ratio, LV eccentricity index, RV outflow Doppler acceleration time, early diastolic pulmonary regurgitation velocity, PA diameter, inferior vena cava diameter with inspiratory response, and RA area. PH was defined as SPAP >40 mm Hg. Four patients with symptoms related to PH underwent a right-heart catheterization (RHC) and PH was confirmed.

Results: SPAP was >40 mm Hg in 7 patients on TTE and 4 additional patients had PH confirmed by RHC. JAK2 mutation was positive in 60% of the patients. Patients with myelofibrosis had PH more often than patients with other MPN (p<0.001). Disease duration was associated with the development of PH (OR: 1.26, p<0.001). JAK2 mutation was not associated with PH.

Conclusions: Our results suggest that patients with myelofibrosis could be more likely to develop PH than other MPN patients. Disease duration could predict the development of PH in MPN patients.

Pulmonary hypertension / Pulmonary vascular diseases

OP-131

Comparison of cardiac output measurement methods for mortality prediction in pulmonary hypertension

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Background and Aim: There isn't any study comparing different CO measurement methods for mortality prediction in PH. The aim of this study is to compare the estimated Fick (eFick), Thermodilution (TD) and impedance cardiography (ICG) methods with regard to mortality prediction in PH patients.

Methods: Study included the retrospective analysis of all patients who had undergone right heart catheterization (RHC) for PH from 2008 to 2015. Only patients who have CO measurement with at least two different methods were included. NICCOMO device with arterial compliance modulation technique for ICG- CO measurements was used. CO was indexed to body surface area and cardiac index (CI) was used for all mortality analyses. Kaplan-Meier survival analyses and Cox-proportional hazard regression models were used to compare prediction of all-cause mortality of the 3 different CO methods.

Results: 121 patients had 134 RHC including CO measurements by at least 2 different methods. Median (IQR) follow up time was 45 (44) months and 56.2% of the patients died during follow up. There were good correlations between eFick and TD (n=111, p<0.001 r=0.626) and also TD vs. ICG (n=47, p<0.001 r=0.622). eFick and ICG was moderately correlated (n=70, p<0.001, r=0.469). However, limits of agreement results were high in Bland-Altman analysis. Three different methods were used in 47 RHC and low eFick CI (<2.2/l/min/m²) was the only significant mortality predictor compared to other 2 methods (Log rank p values for low eFick: 0.003, HR: 3.671, low TD CI: 0.2, low ICG CI:0.302). Also 111 patients had simultaneous CO measurements with eFick and TD. Low eFick CI was still significant mortality predictor (For low eFick p=0.003 HR:2.388 and for low TD CI p=0.056). Patients were also categorized in four groups based on agreement between eFick CI and TD CI results. (Concordant normal, concordant low and 2 discordant groups: low eFick CI and normal TD CI or normal eFick CI and low TD CI). Mortality was significantly higher in groups including patients with low eFick CI. However there was no statistical difference between concordant normal CI group and normal eFick CI and low TD CI groups (p=0.329) (Figure 1).

Conclusions: Although, eFick, TD and ICG methods had statistically significant correlations they showed modest agreement in Bland-Altman analyses. eFick CI predicts mortality more accurately than other methods in PH patients.

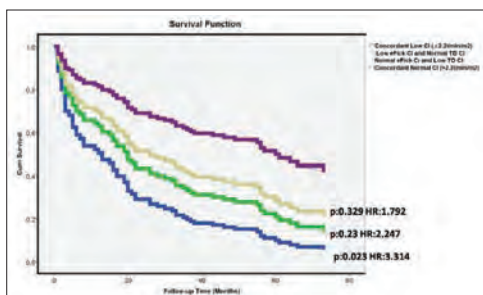


Figure 1. Mortality analyses for groups based on agreement between eFick CI and TD CI results. (Concordant normal, concordant low and 2 discordant groups: low eFick CI and normal TD CI or normal eFick CI and low TD CI).

Pulmonary hypertension / Pulmonary vascular diseases

OP-132

Relationship between CHA2DS2-VASc score and right ventricular dysfunction in patients with acute pulmonary thromboembolism

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Background and Aim: The CHA2DS2-VASc score is used to evaluate the risk for thromboembolic events and stroke, and to manage treatment in patients with atrial fibrillation. In this study, the association between the right ventricle dysfunction (RVD) and CHA2DS2-VASc scores was investigated in patients with acute pulmonary thromboembolism (PTE).

Methods: The patients presenting with acute PTE to the emergency department during the dates between January 2015 and June 2017 were included in the study. All radiological, laboratory, clinical, and demographic data of the patients were obtained from the hospital electronic information system. The patients have been assigned to 3 subgroups as massive PTE, submassive PTE, and nonmassive PTE. The CHA2DS2-VASc scores were calculated for all of the patients and the scores have been classified into 3 groups as the scores between 0 and 1, the scores of 2, and the scores of 3 and over. The independent predictors of the RVD were investigated by the univariate and multivariate regression analyses.

Results: A total of 545 patients were included in the study. Of these patients, 251 of them were males and 294 of them were females. The CHA2DS2-VASc scores were found to be higher in the massive and submassive PTE groups compared to those of the nonmassive PTE group (p=0.002). The independent predictors of the RVD were determined to be the CHA2DS2-VASc scores (p=0.034), the systolic pulmonary artery pressure (p<0.001), the presence of acute deep vein thrombosis (p=0.007), high sPESI (p<0.001), D-dimer (p<0.006), and the mean platelet volume (p<0.001). The CHA2DS2-VASc scores predicted the RVD with 70% sensitivity and with 50% specificity as determined by the receiver operating characteristics analysis.

Conclusions: The CHA2DS2-VASc score is an independent predictor of the RVD in patients with acute PTE.

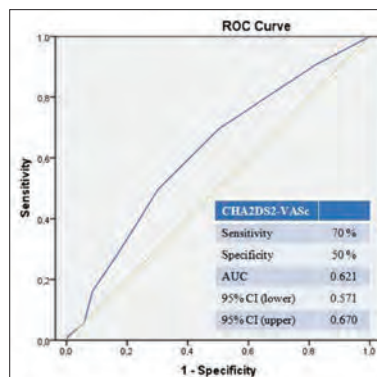


Figure 1.

Cardiovascular surgery

OP-133

Syntax and clinic syntax score as predictors of stroke after isolated coronary artery surgery

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Background and Aim: Stroke is a major cause of mortality and morbidity after coronary surgery, there are many studies to predict stroke after coronary surgery, but there has not ever been investigated correlation between syntax score and clinic syntax score and stroke after isolated coronary artery bypass surgery (CABG).

Methods: From October 2011 to December 2013, of 1850 patients undergoing isolated CABG, 249 patients included in study after eliminating patients having exclusion criteria such as emergent coronary surgery, valvular diseases and congenital heart diseases. Stroke was defined as any neurological deficit lasting 24 hours. Syntax score and adding clinical parameters (age, lvef, gfr) to anatomical clinic syntax score (CSS) were calculated for each patient using the last coronary angiograms before cabg.

Results: In the present study, in postoperative stroke (PoS)(+) group, average age (66.71±7.78) was higher significantly than PoS(-) group (60.82±6.81) (p=0.002). As a parallel with age, total cholesterol (p=0.048), glukoz (p=0.022), uric acid (p=0.032), creatinin (p=0.022), neutrophil (p=0.06), circumflex-saphenous grafting (p=0.01), CSS (p=0.003) were found statistically higher than PoS(-) group. In addition, left ventricular ejection fraction (LVEF) (p=0.019) and glomerular filtration rate (GFR) (p=0.013) were detected lower statistically than PoS(-) group. The predictors evaluated statistically significant analysed with logistic regression to demonstrate combined effects. Therefore, average age (p=0.017), glukoz higness (p=0.08), existence of cx-saphenous grafting (p=0.011) and CSS (p=0.026) were found contributed factors.

Conclusions: The area under the receiver operating curves (ROC) were found 0.648 (0.585-0.707) for syntax score and 0.725 (0.665-0.780) for CSS, respectively. Taken the cut-off value for syntax score >25.5; cencivity 85.71 specificity 38.72, positive predictive value (PPV) 17.7 and negative predictive value (NPV) 97.8 and likelihood ratio 1.40 were found for syntax score. Moreover, criterion value for CSS if taken >8; cencivity 78.57 specificity 68.94, PPV 23.1 NPV 98.2 and LR(+) value 2.53 were found for CSS. This study showed CSS may use for predicting stroke as a valuable parameter and precautions were taken before surgery such as giving antiagregan or antikoagulan drugs.

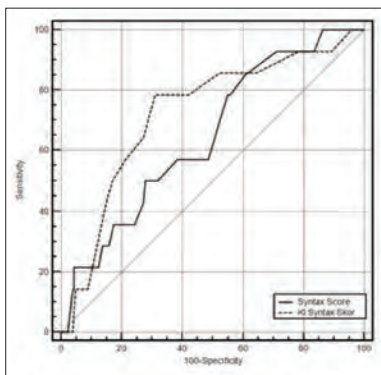


Figure 1. ROC analysis.

Table 1. Basic parameters of patients

	Postop Stroke (-) n:235	Postop Stroke (+) n:14	P
Age, years	60,82±6,81	66,71±7,78	0,002
Gender			
Female, n (%)	56 23,83%	2 14,29%	
Male, n (%)	179 76,17%	12 85,71%	0,412
Diabetes mellitus, n (%)	99 42,13%	8 57,14%	0,270
Hypertension, n (%)	125 53,19%	10 71,43%	0,183
Smoking, n	96 40,85%	8 57,14%	0,230
LVEF, %	52,66±9,38	46,57±9,72	0,019
Total cholesterol, mg/dl	199,91±52,24	227,21±48,41	0,048
HDL, mg/dl	38,91±11,83	40,21±8,4	0,684
LDL, mg/dl	130,06±42,62	145±42,52	0,204
Triglycerids, mg/dl	198,24±142,27	188,71±79,81	0,804
Glucose, mg/dl	136,87±56,94	174,02±84,08	0,022
HbA1c, (%)	7,12±3,67	7,82±2,56	0,482
Uric acid, mg/dl	5,26±1,71	5,96±1,88	0,032
Creatinin, mg/dl	1,31±2,02	2,69±4,16	0,022
GFR ml/min	92,27±29,63	72,07±25,82	0,013
Leucocyte K/µl	8,92±2,69	7,84±2,14	0,144
Hemoglobin g/dl	15,02±23,17	14,22±1,19	0,898
Platelets, 10 ³ /mm ³	247,97±69,09	244,57±60,75	0,858
Neutrophil K/µl	5,32±1,37	6,36±1,25	0,006
Lenfocyte %	2,14±0,63	2,03±0,31	0,525
NLR %	2,78±1,37	3,15±0,60	0,058
Previous MI, n (%)	105 44,68%	5 35,71%	0,512
Previous PCI, n (%)	87 37,02%	4 28,57%	0,524
LAD Lima, n	230 97,87%	13 92,86%	0,235
LAD Safenous, n	15 6,38%	1 7,14%	0,910
DL Safenous, n	115 48,94%	6 42,86%	0,658
IM Safenous, n	23 9,79%	3 21,43%	0,166
CX Safenous, n	48 20,43%	7 50,00%	0,01
CXOM Safenous, n	146 62,13%	8 57,14%	0,709
RCA Safenous, n	121 51,49%	8 57,14%	0,681
RCA-PDA saphenous, n	53 22,55%	3 21,43%	0,922
RCA-PL saphenous, n	7 2,98%	1 7,14%	0,391
Graft per patient, n	3,22±0,81	3,57±0,51	0,108
Syntax Score	28,63±5,92	31,54±6,11	0,076
CI Syntax Score	6,91±4,22	10,36±3,99	0,003
Follow-up in ICU, days	1,12±0,65	1,5±1,16	0,245
Follow-up in hospital, days	8,69±4,21	12,5±7,59	0,085

Table 2. Multivariate Regression Analysis for the Investigation of Independent parameters for predictor of PoS

	B	S.E.	p	OR	OR % 95 GA	
					Low	High
Age	0,12	0,05	0,017	1,13	1,02	1,25
LVEF	-0,00	0,05	0,960	1,00	0,90	1,10
Total cholesterol	0,01	0,01	0,065	1,01	1,00	1,02
Glucose	0,01	0,00	0,008	1,01	1,00	1,02
GFR	-0,00	0,01	0,966	1,00	0,97	1,03
NLR	-0,14	0,35	0,682	0,87	0,44	1,71
CX Safenous	1,60	0,63	0,011	0,20	0,06	0,70
Syntax Score	0,04	0,05	0,443	1,04	0,94	1,16
CI Syntax Score	0,17	0,08	0,026	1,19	1,02	1,39

Table 3.

	AUC	SE	95% CI
Syntax Score	0,648	0,082	0,585 - 0,707
KI Syntax Skor	0,725	0,079	0,665 - 0,780

Table 4.

	Criterion	Sensitivity	Specificity	PPV	NPV	+LR
Syntax Score	>25,5	85,71	38,72	17,7	97,8	1,40
KI Syntax Skor	>8	78,57	68,94	23,1	98,2	2,53

Other

OP-134

Assessment of postoperative changes in frontal QRS-T angle in renal transplant patients

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Background and Aim: Cardiovascular diseases; is the main cause of mortality in patients undergoing hemodialysis or peritoneal dialysis and renal transplantation (tx) is the treatment which is most likely to increase survival. The frontal QRS-T angle (QRS - T a) is defined as the difference between the frontal QRS axis (QRS a) and T wave axis (T a), in other words it defines the angle between the ventricular depolarization and repolarization vectors. So, QRS-T a can reflect the presence of electrical heterogeneity and structural cardiac anomalies; it has also been shown to play a role in predicting total mortality and sudden cardiac death in the general population. It is known that total cardiovascular mortality decreases after renal tx. However, it is unclear how the QRS-T angle changes in patients with renal tx.

Methods: The data of 234 patients with renal tx were reviewed retrospectively. Patients with known heart disease or who did not have regular controls, had no ECG in their controls, were younger than 18 years of age, developed a rejection level requiring hemodialysis again were excluded from the study. The remained 76 patients with renal tx were enrolled to the research. The demographic features, comorbidities, echocardiographic examination, biochemical and complete blood count tests were recorded. The axis measurements, taking by electrocardiography machine automatically, were evaluated before the tx and at one year follow up visit.

Results: Total of 76 patients, 16 (21.1%) were cadaveric tx; 24 (31.6%) were female; 49 (64.5%) had hypertension; 5 (6.6%) had hyperlipidemia; 4 (5.3%) had diabetes mellitus; 32 (42.1%) were smoker or ex-smoker. Mean age of the patients was 35.36±13.58. There was a statistically significant decline in QRS-T angle value between preoperative and postoperative 12th month [26 (10-50.75; IQR=40.75) vs. 21.5 (9.25-36.75; IQR=27.5), p=0.041]. Table 1 summarizes comparisons of QRSA, Ta and QRS-T a before and after tx. Figure 1 shows comparisons of QRS-Ta values before and after tx.

Conclusions: This is the first study to examine how the renal tx changes the QRS-T a, which is considered to be an important predictor of cardiovascular risk. We revealed that QRS-T a decreased after tx, so we can speculate that cardiovascular risk reduces as a result of renal tx. But large prospective trials with longer follow-up period are needed to investigate the correlation between the QRS-T a and cardiovascular outcome in patients undergoing renal tx.

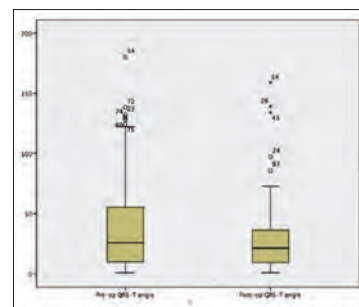


Figure 1. Comparison of QRS-T angle value before and after transplantation.

Table 1.

	Before Transplantation	After Transplantation	p value
QRS axis	43 (20.25-61.75; IQR=41.5)	36 (20.25-55.75; IQR=35.50)	0.039
T axis	59 (36-76; IQR=40)	43.5 (33.25-60.75; IQR=27.5)	0.024
QRS-T angle	26 (10-57.75; IQR=47.75)	21.5 (9.25-36.75; IQR=27.5)	0.041

The alteration of QRS axis, T axis and QRS-T angle after renal transplantation.

Other

OP-135

Secondary prevention in rural patients with coronary heart disease living in rural and urban areas

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Background and Aim: The aim of the present study was to determine the differences between urban and rural patients with coronary heart disease (CHD) with respect to secondary prevention.

Methods: This cross-sectional study included all consecutive patients diagnosed with CHD in two different cardiology clinics between January 2016 and January 2017. The demographic characteristics and clinical history of the patients and routine laboratory values within the last three months were recorded at routine controls. Patients were divided into two groups according to their residence; urban (n=1752) and rural (n=456).

Results: The mean age of rural patients were significantly higher than that of urban patients (64.3±9.6 vs. 62.8±9.5 years; p=0.002). A mean of 4.1±2.1 years passed after the first (index) coronary event. We found that 22.2% of patients continued smoking, and the rate of quitting was significantly higher in urban group (20.5% vs. 11.2%; p<0.001). Hypertension (64.3% vs. 56.7%), diabetes mellitus (45.6% vs. 39.2%), cerebrovascular events (9.2% vs. 3.8%), and chronic obstructive pulmonary disease (11.4% vs. 5.5%) were all significantly higher in rural patients (p<0.05 for all). In rural patients, 34.2% were obese, and obesity was significantly higher in this group compared to the urban group (46.4% vs. 31.2%, p<0.001). The number of patients performing regular exercise was significantly lower in rural patients (34.4% vs. 23.9%; p<0.001). Overall, 88.9% of patients received antiplatelet agents; 62.1%, statins; 73.1%, beta-blockers; and 34.2%, ACEI/ARBs. The rate of receiving medication was significantly higher in urban patients than in rural patients (p<0.05 in all cases).

Conclusions: The level of secondary prevention among patients with CHD requires further improvement. Moreover, the level of secondary prevention in the rural population is worse than that in the urban population.

Table 1.

Parameters	All patients (n=2208)	Rural (n=456)	Urban (n=1752)	P
Age, year, median (IQR)	64 (32)	65 (58-70)	64 (33)	0.441
Female, n (%)	795 (35.5)	162 (35.5)	633 (36.1)	0.811
Age at the first diagnosis of CHD, n (%)				
<50 year	466 (21.1)	119 (26.1)	347 (19.8)	0.020
50-59 year	730 (33.1)	141 (30.9)	589 (33.6)	
60-69 year	807 (36.5)	162 (35.5)	645 (36.8)	
≥70 year	205 (9.3)	34 (7.5)	171 (9.8)	
CAAG, n (%)	702 (31.8)	158 (34.6)	544 (31.2)	0.546
PTCA/stent/stenosis%50, n (%)	1473 (67.2)	306 (66.9)	1167 (67.4)	
Heart failure, n (%)	531 (24.0)	134 (29.0)	417 (23.8)	0.594
Smoking, n (%)				
Current smoker	489 (22.2)	108 (23.7)	381 (21.7)	<0.001
Quit smoking	411 (18.6)	51 (11.2)	360 (20.5)	
Never smoked	1508 (59.2)	297 (65.1)	1011 (57.7)	
Hypertension, n (%)	1287 (58.3)	298 (64.9)	989 (56.7)	0.004
Hyperlipidemia, n (%)	1044 (47.3)	213 (46.7)	831 (47.4)	0.784
Diabetes mellitus, n (%)	894 (40.5)	208 (45.6)	686 (39.2)	0.012
Cerebrovascular disease, n (%)	108 (4.9)	42 (9.2)	66 (3.8)	<0.001
Chronic kidney disease, n (%)	144 (6.5)	24 (5.3)	120 (6.8)	0.222
Chronic obstructive pulmonary disease, n (%)	149 (6.7)	52 (11.4)	97 (5.5)	<0.001
Education level, n (%)				
illiterate	1005 (45.5)	213 (46.7)	792 (45.2)	<0.001
Primary school	915 (41.4)	216 (47.4)	699 (39.9)	
High school	204 (9.2)	21 (4.6)	183 (10.4)	
University	84 (3.8)	6 (1.3)	78 (4.5)	

Table-1 Comparison of baseline characteristics of patients.

CAAG: Coronary artery bypass graft, CHD: Coronary heart disease, IQR: Interquartile Range, PTCA: Percutaneous transluminal coronary angioplasty

Table 2.

Parameters	All patients (n=2208)	Rural (n=456)	Urban (n=1752)	P
Waist circumference (cm), mean ±SD (n=1579)	101 ± 10.7	102.4 ± 10.3	100.4 ± 10.8	0.002
Waist circumference (Men≥102cm, Women≥88cm), n (%)	968 (61.3)	226 (68.5)	742 (59.4)	0.002
BMI (kg/m ²), mean ±SD (n=2040)	29.2 ± 10.7	31.1 ± 9.9	27.9 ± 13.7	<0.001
Overweight, (BMI≥23kg/m ²), n (%)	1863 (81.5)	383 (94.6)	1280 (78.3)	<0.001
Obesity, (BMI≥30kg/m ²), n (%)	698 (34.2)	188 (46.4)	510 (31.2)	<0.001
Systolic blood pressure (mmHg) median (IQR)	130 (20)	130 (20)	130 (19)	0.131
Diastolic blood pressure (mmHg) median (IQR)	74 (10)	70 (10)	75 (10)	0.523
Systolic blood pressure / Diastolic blood pressure ≥140/90 (mmHg), n (%)	818 (37.0)	168 (36.8)	650 (37.1)	0.241
Total cholesterol (mg/dl) median(IQR) (n=2133)	190 (63)	192 (65)	187 (65)	0.007
LDL-C (mg/dl) median (IQR) (n=2133)	104 (46)	107 (45)	99 (48)	0.001
LDL-C ≥70 mg/dl, n (%)	1854 (86.9)	381 (85.2)	1473 (87.4)	0.235
HDL-C (mg/dl) median (IQR) (n=1842)	40 (13)	39 (13)	40 (13)	0.163
Triglyceride (mg/dl) median (IQR) (n=1886)	177 (128)	180 (136)	176 (128)	0.318
Fasting Blood Glucose (mg/dl), median (IQR) (n=1731)	121 (76)	118 (81)	122 (72)	0.849
Fasting Blood Glucose ≥126mg/dl, n (%)	807 (46.6)	162 (46.2)	645 (46.7)	0.844

Table-2 Comparison of anthropometric and laboratory parameters of patients

BMI: Body mass index, HDL-C: High density lipoprotein cholesterol, IQR: Interquartile Range, LDL-C: Low density lipoprotein cholesterol

Table 3.

Drug treatment	All patients (n=2208)	Rural (n=456)	Urban (n=1752)	P
Antiplatelet, n (%)	1962 (88.9)	393 (86.2)	1569 (89.6)	0.042
Statin, n (%)	1371 (62.1)	217 (47.6)	1154 (65.9)	<0.001
Beta blocker, n (%)	1613 (73.1)	334 (73.2)	1279 (73.0)	0.917
ACEI/ARB, n (%)	756 (34.2)	123 (27.0)	633 (36.1)	<0.001
CCB, n (%)	226 (10.2)	39 (8.6)	187 (10.7)	0.088
Diuretic, n (%)	201 (9.1)	24 (5.3)	177 (10.1)	0.001
PPI, n (%)	477 (21.6)	66 (14.5)	411 (23.5)	<0.001
Antiplatelet-Statin, n (%)	1217 (55.1)	177 (38.8)	1040 (59.4)	<0.001
Antiplatelet-Statin-Beta-blocker combination, n (%)	1170 (53.0)	177 (38.8)	993 (56.7)	<0.001
Antiplatelet-Statin-Beta-blocker-ACEI/ARB combination, n (%)	400 (18.1)	48 (10.5)	352 (20.1)	<0.001

Table-3. Pharmacological treatment of study population.

ACEI: Angiotensin Converting Enzyme, ARB: Angiotensin Receptor Blocker, CCB: calcium channel blocker, PPI: Proton Pump Inhibitor, ACEI: Angiotensin Converting Enzyme, ARB: Angiotensin Receptor Blocker, CCB: calcium channel blocker, PPI: Proton Pump Inhibitor

Other

OP-136

Comparison of radiation-induced DNA damage between conventional and computed tomography coronary angiography

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Background and Aim: Evaluation of coronary artery anatomy is essential for the diagnosis of coronary artery disease (CAD). Computed tomography angiography (CCTA) has been widely used for this purpose instead of conventional coronary angiography (CCA). Ionizing radiation is present in both methods. In this study, we aimed to compare radiation-induced DNA damage in both CCTA and CCA.

Methods: Adult patients in whom CAD is suspected and planned to have CCTA and CCA are included in the study. Chromosomal aberration rates are examined by conducting chromosomal aberration test before and after CCTA and CCA scans. Patients having CCTA scans are classified according to flash mode use. Chromosomal aberration rates are compared among Flash mode on CCTA, Flash mode off CCTA and CCA groups.

Results: Patient population consisted of 31 women and (40.8%) and 45 men (49.2%) and three groups have similar distributions in terms of sex (p=0.460). Flash mode off CCTA group has lower average age compared to CCA group (p=0.016). The lowest heart rate is observed in flash mode on CCTA scanned group (p<0.001). Other basal characteristics are similar for all groups. Effective radiation dose showed a significant difference among groups by being highest for flash mode off CCTA and least for Flash mode on CCTA groups (Figure 1, p<0.05). The change in total chromosomal aberration rates also showed significant difference among groups by being least for Flash mode on CCTA group (Figure 2, p<0.05).

Conclusions: The level of effective radiation and DNA damage due to radiation is lowest for Flash mode on CCTA and this particular imaging method does not increase rate of chromosomal aberration. While the use frequency of CCTA method is increasing, flash mode should be preferred if possible, since patients will be exposed to less radiation levels than genotoxic effects of radiation may be decreased.

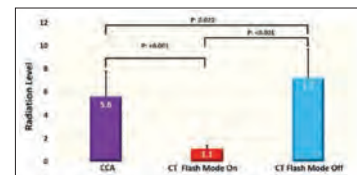


Figure 1.

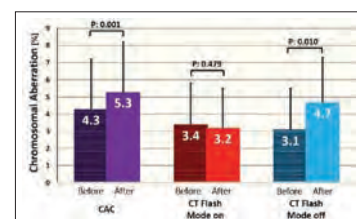


Figure 2.

Other

OP-137

The characteristics of the stroke patients with elevated troponin I

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Background and Aim: Stroke patients may have ST segment/T wave changes and elevated troponin values although they are not complicated with acute coronary syndrome. Neurogenic myocardial injury is a newly described concept in stroke patients and is a result of dysregulation of autonomic nervous system and may cause stress induced cardiomyopathy. The aim of the study was to explore the frequency of the troponin elevation in stroke patients and the characteristics of these patients.

Methods: One hundred and eleven consecutive patients (111 patients; mean age 66±15; 66 male) presenting with acute ischemic stroke documented by cranial imaging were included in the study. All patients were carefully evaluated to determine their Framingham Heart Study Risk Score (FRS). Blood samples were taken to assess troponin I levels, which were accepted as elevated when >0.04 ng/L. ST segment depression and T wave inversion were noted from electrocardiograms. All patients underwent transthoracic echocardiography to determine left ventricular systolic functions. Endothelial function and carotid artery intima-media thickness (CIMT) were assessed by brachial artery flow mediated dilatation (FMD) and carotid artery ultrasonography, respectively. Patients were followed for six months for detection of major cardiovascular events including myocardial infarction, recurrent stroke or cardiovascular death.

Results: Twenty-five patients (22.5%) had elevated troponin I levels. The characteristics of ischemic stroke patients according to the troponin levels are listed in Table 1. The prevalence of coexisting coronary artery disease and ST segment depression/T wave inversion were significantly higher in patients with high troponin levels while left ventricular ejection fraction was significantly lower. During six month of follow-up, 23 patients had experienced major cardiovascular events including myocardial infarction, recurrent stroke or cardiovascular death. The prevalence of cardiovascular events was higher in the elevated troponin group although the difference was not statistically significant.

Conclusions: Neurogenic stunned myocardium may be related to increase in troponin levels, ST segment/T wave abnormalities, wall motion abnormalities and decrease in LVEF in patients with acute stroke. Our study points out the presence of left ventricular systolic function abnormality in stroke patients with higher troponin levels and suggests close follow of these patients for presence of neurogenic stunned myocardium.

Table 1.

	Stroke patients with elevated Troponin I (n=25)	Stroke patients with normal Troponin I (n=86)	p
Age (years)	66 ± 15	65 ± 14	0.751
Male sex (n - %)	14 (56%)	52 (60.5%)	0.689
Coexisting coronary artery disease (n - %)	12 (48%)	21 (24.4%)	0.023
Framingham Risk Score (%)	20.6 ± 10.1	22.9 ± 8.8	0.448
ST segment depression/T wave inversion (n - %)	19 (76%)	40 (46.5)	0.009
NT-proBNP (pg/mL)	1324 ± 1362	963 ± 2327	0.061
hs-CRP (mg/L)	27.0 ± 36.2	24.1 ± 34.8	0.408
Carotid artery intima media thickness (mm)	0.87 ± 0.16	0.88 ± 0.20	0.927
Flow mediated dilation (%)	6.6 ± 6.8	8.5 ± 2.6	0.843
Left ventricular ejection fraction (%)	54 ± 14	64 ± 11	0.034
Cardiovascular events (n - %)	8 (32%)	15 (17.4%)	0.114

NT-proBNP: N terminal pro-brain natriuretic peptide; hs-CRP: High sensitive C reactive protein;

Other

OP-138

The effect of time between coronary angiography and surgery on the development of postoperative acute kidney injury in patients with isolated diabetes mellitus who underwent isolated coronary artery bypass graft surgery

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Background and Aim: Diabetes mellitus (DM) in which coronary artery disease is common is an important risk factor for both contrast induced nephropathy (CIN) and postoperative acute kidney injury (AKI). We aimed to investigate how the time interval between coronary angiography and coronary bypass graft surgery (CABG) affects the postoperative AKI incidence in DM patients.

Methods: In this retrospective study 412 DM patients who underwent CABG operation between 2013 and 2016 were included. The demographic properties, comorbidities, medical and surgical history, coronary angiography and operational data and laboratory parameters of the patients were scanned from our institution data base and recorded. AKI was evaluated according to RIFLE criterias.

Results: The number of the patients with postoperative AKI was 108 (25.6%). 15% of them was Stage R, 5.5% of them was stage I and 5.1% of them was stage F. The median of the time between the coronary angiography and the CABG operation was not statistically different between groups [11.5 (7-16) days in AKI group and 12 (7-17) days in the group without AKI] (p=0.871). We classified patients into three groups according to

time interval (0-3 days, 3-7 days and >7 days); also the risk of AKI was not statistically significant in all three time intervals. We perform multivariate analysis; the previous myocardial infarction [OR and 95% CI, 5.192 (2.176-12.38), p<0.001] and the increase in the postoperative first day creatinin levels [OR and 95% CI, 4.102 (1.278-13.17), p=0.018] were found independent predictors of the development of AKI.

Conclusions: In patients with diabetes mellitus, CABG surgery can be performed without any delay after angiography, without an increase in postoperative risk of AKI but especially in those who have previous myocardial infarction postoperative first day creatinine level should be noted.

Hypertension

OP-139

Short-term prognostic effect of blood pressure variability in patients with acute ST-elevation myocardial infarction

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Background and Aim: There have been a number of reports that daytime, night-time, and 24-hour blood pressure variability (BPV) were associated with cardiovascular outcomes. The aim of this study was to evaluate the one month prognostic value of blood pressure(BP) variability estimated with SD and coefficient of variation (CV) of daytime and night time systolic and diastolic BP in patients with acute ST elevation myocardial infarction (STEMI).

Methods: In this prospective study, 116 consecutive patients with their first STEMI were recruited and finally 92 patients (74 male, 55±11 years) were included. The patients were followed up for the development of cardiac events (all-cause mortality, recurrent myocardial infarction, recurrence of angina pectoris) at 1 months after AMI. Accordingly, patients were assigned into two groups: group 1 (n=25); patients with cardiac event and group 2 (n=67); patients without cardiac event. Ambulatory BP was recorded using the fully automatic monitor to take a measurement throughout the 24 hours. The coefficient of variation (CV) of BP (expressed as percent of SD/mean BP) was also calculated for every time period.

Results: During follow-up a total of twenty-five patients developed a cardiac event (Group 1), whereas remaining 67 patients did not develop any cardiac events (Group 2). Cardiac events (n=25) included 4 fatal or nonfatal acute myocardial infarction, 15 cases of recurrent angina pectoris and 4 cases of cardiac death. Mean age was significantly higher in group 1 compared to group 2 (61 vs 55, p=0.011). The rate of hypertension history was higher in group 1 (40% vs 20%, p=0.042). Night-time systolic both SD and CV values were significantly higher in group 1 compared to group 2 (p<0.0001, p=0.001, respectively) (Table 1). No difference were shown between two group according to diastolic BP values. Although the hypertension story was significantly higher in group 1, the 24-h mean systolic and diastolic blood pressures levels were similar between the two groups. To determine the cut-off values of blood pressure variability for predicting cardiovascular event, ROC analyses were performed. The cut-off values of 9.15 mmHg with 69% sensitivity and 67% specificity (area under the curve: 0.737, 95% CI: 0.618-0.857) for night-time systolic SD (Figure 2) and cut-off values of 7.8% with 69% sensitivity and 68% specificity (area under the curve: 0.718, 95% CI: 0.580-0.812) for night-time systolic CV, were found to be highly sensitive and specific for predicting early term cardiovascular event after AMI.

Conclusions: In our study, we found that higher night-time ambulatory systolic BPV indices including SD and CV values in patients with STEMI were significantly associated with cardiac events in the one month follow-up. Risk stratification of the patients with AMI is important in clinical decision regarding subsequent treatment. Our study results showed that the increased in-hospital systolic BPV may helpful in risk stratification of STEMI patients.

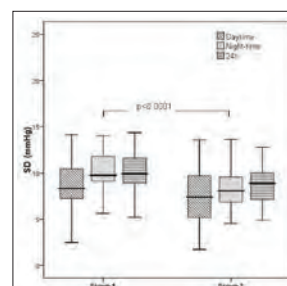


Figure 1. Box-plot of systolic SD values in both group.

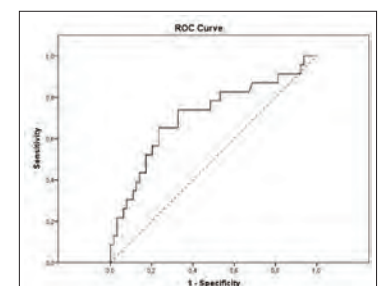


Figure 2. The receiver operating characteristic (ROC) curve for the night-time systolic SD and CV to predict cardiovascular event.

Table 1.

Systolic Blood pressure (mmHg)	Group 1 (n=25)	Group 2 (n=67)	P value
24-h mean	115,4 ±13,3	112,0±11,2	0.220
Night-time mean	114,1±12,3	111,3±12,0	0.331
Daytime mean	116,8±17,2	112,7±11,9	0.305
24-h SD	9,3(8.0-11.3)	8,8(6.8-10.0)	0.030
Night-time SD	10,4± 2,9	8,2±1,9	<0.0001
Daytime SD	9,6(9.0-11.7)	7,9(6.7-9.5)	0.08
24-h CV (%)	8.2(7.3-10.6)	7.7(6.1-8.6)	0.052
Night-time CV(%)	9,2±2,7	7,4±1,9	0.001
Daytime CV(%)	9,0(7.1-11.1)	7,1(6.2-8.2)	0.207

Comparison of ambulatory systolic BPV values in both group.

Hypertension

OP-140

Frontal QRS-T angle as a marker of left ventricular hypertrophy in patients with essential hypertension

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Background and Aim: Previous studies showed that myocardial repolarization markers prolonged in hypertensive patients with left ventricular hypertrophy (LVH) compared to patients with non-LVH. Frontal QRS-T angle, angle between the QRS and T wave axes, is novel marker of myocardial repolarization. The aim of our study is to investigate the relationship between frontal QRS-T angle and LVH in hypertensive patients.

Methods: A total 187 hypertensive patients were included our study. Frontal QRS-T angle was obtained from the automatic reports of the ECG machine. An illustration of the measurement of the frontal QRS-T angle is showed in Figure 1. LVH was defined as left ventricular mass index (LVMI) >115 g/m² in men and 95 g/m² in women.

Results: Patients with LVH had significantly longer QT dispersion (p=0.028), corrected QT dispersion (p=0.010), Tp-e interval (p=0.045) and wider frontal QRS-T angle (p<0.001) compared to patients with non-LVH (Table 1). In correlation analysis, LVMI was positively correlated with QT dispersion (r=0.150, p=0.041), corrected QT dispersion (r=0.167, p=0.022), Tp-e interval (r=0.160, p=0.046) and frontal QRS-T angle (r=0.360, p<0.001). By a multivariate analysis, frontal QRS-T (OR: 1.04, 95% CI: 1.02-1.06, p<0.001) angle was found to be the only independent predictor of LVH. ROC curve analysis showed that the best cut-off value of frontal QRS-T angle for predicting LVH was ≥28 (Figure 2). This cut-off value predicted LVH with a sensitivity of 70.5% and a specificity of 54.5%. When patients were divided into two group according to this cut-off value, it was found that patients with frontal QRS-T angle ≥28 had significantly higher LVMI and had higher frequency of the presence of LVH (Figure 3).

Conclusions: Frontal QRS-T angle is a simple, inexpensive and easily obtainable parameter from surface electrocardiography. It can be used as a simple marker of LVH in hypertensive subjects. To our knowledge, this is the first study demonstrating the relationship between the frontal QRS-T angle and the presence of LVH.



Figure 1. An illustration of the measurement of the frontal QRS-T angle.

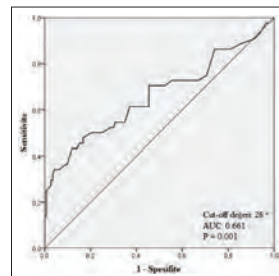


Figure 2. ROC curve of frontal QRS-T angle for predicting presence of LVH.

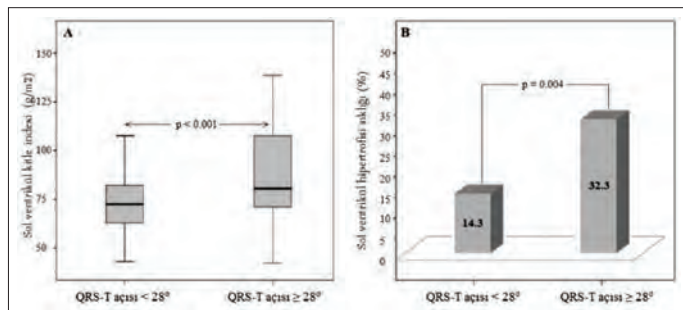


Figure 3. Comparison of patients with frontal QRS-T angle ≥28 and <28.

Table 1. Comparison of electrocardiographic and echocardiographic variables of the population

	LVH (+) (n = 44)	LVH (-) (n = 143)	P
Heart rate	78.2 ± 12.2	74.7 ± 9.1	0.047
QRS duration (msn)	95.7 ± 6.9	95.2 ± 6.6	0.650
QTd (msn)	21.0 ± 4.3	19.2 ± 6.0	0.028
cQTd (msn)	23.8 ± 4.9	21.3 ± 7.5	0.010
Tp-e interval (msn)	88.9 ± 8.7	85.8 ± 8.8	0.045
Tp-e/QT ratio	0.24 ± 0.4	0.24 ± 0.3	0.508
Tp-e/QTc ratio	0.21 ± 0.3	0.22 ± 0.3	0.410
Frontal QRST angle (°)	43.5 ± 30.0	26.6 ± 15.4	<0.001
LVEF (%)	62.4 ± 2.5	62.5 ± 2.9	0.843
LVMI (g/m ²)	116.9 ± 10.8	69.8 ± 10.8	<0.001

Hypertension

OP-141

Is there a relation between non-dipping hypertension and postural orthostatic tachycardia syndrome?

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Background and Aim: Postural Orthostatic Tachycardia Syndrome (POTS) is a disorder which negatively affects patient's life quality, frequently overlooked, and hard to diagnose. Non-dipping blood pressure is defined as the nocturnal average blood pressure (BP) drop is less than 10% when compared to daytime values. This profile increases the cardiovascular event risk in both normotensive and hypertensive patients. There are some studies showing the relation between POTS and non-dipping profile in normotensive patients. But, data is limited about this relation in hypertensive patients. Our aim was to investigate that whether there was a relation between non-dipping HT and POTS or not in our study.

Methods: A total of 271 patients with hypertension (HT) were enrolled in our study. Tilt table test, 24-hour ambulatory BP measurement (ABPM), echocardiography, and laboratory tests were performed for all patients. POTS was defined by all of the following: 1) symptoms that occur with standing; and 2) an increase in heart rate of ≥30 bpm during a positional change from supine to standing bpm; and 3) the absence of orthostatic hypotension.

Results: There were 39 (14.3%) patients with POTS. There were no differences about demographic variables between groups (Table 1). Drug therapies were similar in both groups (Table 2). Laboratory and echocardiography parameters were also similar (Table 3 and 4). In the 24-hour ABPM results comparison, daytime mean SBP (p=0.002), daytime mean DBP (p=0.008), nighttime mean SBP (p=0.015), nighttime mean DBP (p=0.008), 24-hour mean SBP (p=0.002), 24-hour mean DBP (p=0.023), and frequency of non-dipping HT (p=0.038) were significantly higher in patients with POTS. Daytime mean SBP (OR: 1.047, 95% CI: 1.008-1.086, p=0.016) and non-dipping (OR: 3.152, 95% CI: 1.396-7.118, p=0.006) were determined as independent predictors for POTS in binominal logistic regression analysis (Table 6).

Conclusions: POTS is more frequent in non-dipping HT patients. Non-dipping HT situation can be easily determined by ABPM. Clinicians might be aware of POTS if their HT patients complaining about palpitations, lightheadedness, and fatigue in the upright position.

Table 1. Comparison of patients' demographic findings

	POTS (-) n=232	POTS (+) n=39	P
Age (years)	53.4 ± 10.3	52.1 ± 12.6	0.49
Gender (male)	152 (65.5)	27 (69.2)	0.650
Office systolic BP (mmHg)	151.6 ± 19.8	156.9 ± 19.4	0.12
Office diastolic BP (mmHg)	90.6 ± 11.0	94.1 ± 11.9	0.069
Heart rate (beat/min)	82.1 ± 8.4	80.7 ± 8.6	0.313
Weight (kg)	84.1 ± 15.6	85.2 ± 15.5	0.688
Height (cm)	164.5 ± 8.5	165.6 ± 9.1	0.449
BMI (kg/m ²)	24.4 ± 2.3	24.5 ± 2.1	0.859
Smoking, n (%)	48 (20.7)	10 (25.6)	0.485
Diabetes mellitus, n (%)	38 (16.4)	7 (17.9)	0.807
Hypercholesterolemia, n (%)	50 (21.6)	7 (17.9)	0.609

Table 2. Comparison of patients' medications

	POTS (-) n=232	POTS (+) n=39	p
ACE (n, %)	60 (25.9)	11 (28.2)	0.758
ARB (n, %)	39 (16.8)	2 (5.1)	0.06
β blocker (n, %)	91 (39.2)	17 (43.6)	0.606
Ca channel blocker	72 (31.0)	16 (41)	0.218
α blocker (n, %)	7 (3.0)	0 (0)	0.598
Diuretic (n, %)	86 (37.1)	10 (25.6)	0.167

ACE: Angiotensin converting enzyme, ARB: Angiotensin receptor blocker

Table 3. Comparison of patients' laboratory findings

	POTS (-) n=232	POTS (+) n=39	P
Glucose (mg/dL)	111.1 ± 36.4	116.2 ± 44.1	0.437
BUN (mg/dL)	27.8 ± 8.8	32.6 ± 20.8	0.264
Creatinine (mg/dL)	0.77 ± 0.21	0.99 ± 1.05	0.214
Alanine transferase (u/L)	28.6 ± 11.5	27.2 ± 11.7	0.536
Uric acid (mg/dL)	1.8 ± 0.9	1.7 ± 0.9	0.848
Total Cholesterol (mg/dL)	207.9 ± 40.5	205.6 ± 40.8	0.741
LDL Cholesterol (mg/dL)	136.3 ± 34.7	135.3 ± 35.7	0.867
HDL Cholesterol (mg/dL)	46.9 ± 11.1	47.6 ± 10.1	0.703
Triglyceride (mg/dL)	180.1 ± 96.1	177.0 ± 89.7	0.848
Hemoglobin (g/dL)	13.8 ± 1.6	13.5 ± 1.5	0.265
BNP (ng/mL)	95.4 ± 148.8	75.6 ± 106.7	0.536
Hs-CRP (mg/L)	0.40 ± 0.51	0.35 ± 0.21	0.603

Table 4. Comparison of patients' ambulatory blood pressure findings

	POTS (-) n=232	POTS (+) n=39	p
Daytime mean SBP (mmHg)	131.2 ± 14.1	138.9 ± 16.0	0.002
Daytime mean DBP (mmHg)	75.9 ± 11.6	81.3 ± 12.7	0.008
Nighttime mean SBP (mmHg)	124.0 ± 17.2	131.4 ± 17.2	0.015
Nighttime mean DBP (mmHg)	75.9 ± 11.6	81.3 ± 12.7	0.008
24-hour mean SBP (mmHg)	129.6 ± 14.1	137.3 ± 15.9	0.002
24-hour mean DBP (mmHg)	81.6 ± 11.0	86.1 ± 11.9	0.023
Augmentation index	28.9 ± 11.7	28.7 ± 11.5	0.937
Cardiac output	4.7 ± 0.7	4.9 ± 0.8	0.263
Pww	8.1 ± 1.7	8.3 ± 1.6	0.465
Dipper (n,%)	113 (48.7)	13 (33.3)	0.075
Non-dipper (n,%)	107 (46.1)	25 (64.1)	0.038
Extreme dipper (n,%)	12 (5.2)	1(2.6)	0.700

SBP: Systolic blood pressure, DBP: Diastolic blood pressure

Table 5. Independent predictors for POTS

	OR	95 % CI	p
Daytime mean SBP (mmHg)	1.047	1.008 – 1.086	0.016
Daytime mean DBP (mmHg)	1.056	1.000 – 1.116	0.05
Nighttime mean SBP (mmHg)	0.975	0.925 – 1.028	0.355
Nighttime mean DBP (mmHg)	1.014	0.973 - 1.056	0.512
24-hour mean SBP (mmHg)	1.033	0.797 – 1.339	0.806
24-hour mean DBP (mmHg)	1.097	0.875 – 1.375	0.424
Non-dipper	3.152	1.396 – 7.118	0.006

SBP: Systolic blood pressure, DBP: Diastolic blood pressure

Hypertension

OP-142

Early organ damage and neutrophil gelatinase related lipocalin (NGAL) levels in primary hypertensive patients

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Background and Aim: End organ damage caused by hypertension can be early detected with various approaches. Early diagnosis and treatment may prevent end organ damage. The aim of this study was to evaluate the relation among neutrophil gelatinase-related lipocalin (NGAL) - a new biomarker for subclinical myocardial dysfunction and retinopathy-, global longitudinal strain (GLS) and early organ damage in hypertensive patients.

Methods: Forty-six hypertensive patients and 21 healthy volunteers were included in the study. All patients had arterial blood pressure measurements and ECG was obtained. Echocardiographic examination was also performed to all subjects. Left ventricular morphology and geometry, systolic and diastolic functions were analyzed as suggested in the American Society of Echocardiography guidelines. GLS was calculated as the average of longitudinal strain measurements from 4-, 2-, and 3- chamber views. NGAL was measured from serum with ELISA method.

Results: GLS was found to be significantly decreased in the hypertensive group (compared to healthy subjects (-18.2±4.8% vs. -23.7±3.2, p<0.001). GLS measurements also showed significant differences among subjects when grouped according to diastolic dysfunction stages (ANOVA p<0.001). The NGAL level was 144.1±16.2 ng/ml in patients without diastolic dysfunction, 159.8±17.8 ng/ml in the stage 1 diastolic dysfunction group and 163.7±18.6 in the stage 2 diastolic dysfunction group (ANOVA p=0.001). There was a significant correlation between NGAL levels and GLS (r=0.753, p<0.001).

Conclusions: NGAL levels had a strong correlation with GLS measurement and showed that it could be used as a new marker to detect early organ damage in hipertensif patient.

Hypertension

OP-143

Evaluation of atrial electromechanical functions in dipper and non-dipper hypertension patients using left atrial strain P Wave Dispersion, and P terminal force

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Background and Aim: Non-dippers are known to carry a high risk of cardiovascular morbidity and mortality. The aim of this study was to investigate the effects of dipper and non-dipper status of hypertension on left atrial (LA) systolic and diastolic functions using two-dimensional speckle tracking echocardiography (2D-STE), P wave dispersion (PWD), and P terminal force (PTF) in hypertensive patients.

Methods: A total of 72 patients and 39 healthy individuals were included in the study. The patients were classified as non-dippers if their daytime ambulatory systolic and diastolic blood pressure did not decrease by at least 10% during the night. Atrial electromechanical delay times, LA volume indexes, LA strain values were obtained by 2D-STE with automated software and compared between the groups. PWD and PTF data were calculated on the electrocardiography.

Results: Inter-atrial (dippers: 25.5±3.9, non-dippers: 32.2±7.4, p<0.001), left-atrial (dippers: 14.9±3.7, non-dippers: 18.2±6.0, p=0.016) and right atrial (dippers: 10.5±2.1, non-dippers: 14.2±5.2, p<0.001) electromechanical delay times were significantly longer in non-dippers. LA strain S (dippers: 34.2 (29.7-38.7), non-dippers: 27.7 (22.7-32.2), p<0.001), LA strain E (dippers: 18.2 (16.6-20.1), non-dippers: 14.4 (11.6-16.8), p<0.001), and LA strain A (dippers: 15.8 (13.5-17.9), non-dippers: 12.7 (9.9-14.5), p<0.001) were significantly lower in non-dippers. Non-dippers also had an increased values of maximum P wave duration [dippers: 0.117 (0.10-0.12), non-dippers: 0.126 (0.12-0.14), p<0.001], PWD [dippers: 0.062 (0.06-0.07), non-dippers: 0.069 (0.06-0.08), p=0.004], and PTF [dippers: 0.055±0.02, non-dippers: 0.066±0.02, p=0.02]. We found a significant negative correlation between PTF and LA S-A, LA S-E, and LA S-S (r=-0.465, p<0.001; r=-0.475, p<0.001; r=-0.507, p<0.001, respectively). Additionally, there was a positive correlation between PTF and atrial electromechanical conduction times (inter-atrial, intra-LA and intra-RA) (r=0.549, p<0.001; r=0.423, p<0.001; r=0.505, p<0.001, respectively), and left atrial volume indexes (LAVip, LAVmax, and LAVmin) (r=0.414, p<0.001; r=0.246, p=0.009; r=0.333, p<0.001, respectively).

Conclusions: Non-dipping pattern in hypertensive patients had a worse cardiac remodelling and impaired mechanical LA function compared with dipping pattern. The PWD and PTF findings support these changes. PTF was significantly correlated with other atrial deformation parameters.

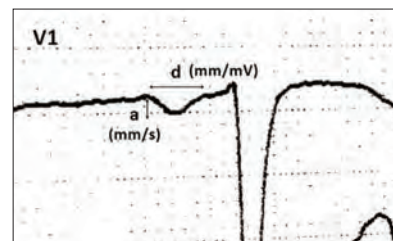


Figure 1. A 49 years old patient's ECG sample. P wave terminal force calculation was shown in V1.

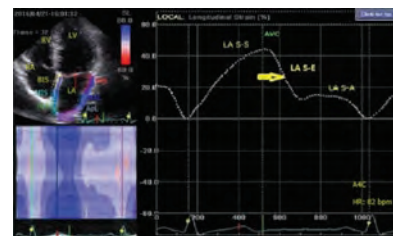


Figure 2. Two-dimensional left atrial strain parameters acquired from apical four chamber view from a 57 years old patient in dipper HT group. LA S-S: Left atrial strain S, LA S-E: Left atrial strain E, LA S-A: Left atrial strain A.

Table 1.

Variables	Control (n=39)	DHT (n=30)	NDHT (n=42)	p value
Age (years)	44.2±6.9*	55.3±10.6	55.3±10.6	0.812
Male n (%)	25 (22.5)	17 (15.3)	21 (18.9)	0.441
BMI (kg/m2)	24.9±3.5*	29.4±4.1	29.34.4	0.997
Smoking n (%)	16 (42.1)	12 (31.6)	10 (26.3)	0.195
HT duration (year)	-	3.5 (1.8-5.0)	4.9 (3.0-7.0)	0.102
ACEI/ARB n (%)	-	6 (66.7)	3 (33.3)	0.151
CCB n (%)	-	3 (33.3)	6 (66.7)	0.726
DIURETICS n (%)	-	1 (100)	0 (0)	0.417
ACEI/ARB+CCB n (%)	-	4 (28.6)	10 (71.4)	0.268
ACEI/ARB+Diuretics n (%)	-	9 (52.9)	8 (47.1)	0.281
ACEI/ARB+BB+ Diuretics n (%)	-	3 (50)	3 (50)	0.688
ACEI/ARB+CCB+Diuretics n (%)	-	2 (66.7)	1 (33.3)	0.567
All Drug Groups n (%)	-	2 (28.6)	5 (71.4)	0.692

Clinical characteristics and distribution of the using drugs of control and patient groups. Abbreviations: BMI: body mass index, HT: hypertension, ACEI/ARB: Angiotensin converting enzyme inhibitor / angiotensin receptor blocker, CCB: calcium channel blocker, BB: beta blocker, AB: alpha-1 blocker. * Between patient and control p<0.05

Table 1.

variables	DHT (n=30)	NDHT (n=42)	p value
Daytime SBP (mmHg)	131.2±14.3	135.4±16.5	0.493
Daytime DBP (mmHg)	85.1±12	84±12.5	0.881
Daytime MAP (mmHg)	105.7±12.8	105.7±13.4	0.999
Daytime PP (mmHg)	45.7±7.8	48.1±10.27	0.364
Night SKB (mmHg)	116.8±13.1	130.2±15.4	0.008+
Night DBP (mmHg)	73.2±10.1	80±15.5	0.02+
Night MAP (mmHg)	93.2±11.4	104.3±12.3	<0.005+
Night PP (mmHg)	43.1±7.5	47.9±10.1	0.022+
24 hours SBP (mmHg)	126.3±13.6	131.4±15.5	0.178
24 hours DBP (mmHg)	80.9±11.7	83.6±11.4	0.445
24 hours MAP (mmHg)	100.9±12.2	105.4±12.4	0.147
24 hours PP (mmHg)	44.9±7.4	47.7±10.3	0.252

ABPM findings Abbreviations: SBP: systolic blood pressure, DBP: diastolic blood pressure, MAP: mean arterial pressure, PP: pulse pressure + Between DHT and NDHT group p<0.05.

Table 3.

Variables	Control (n=39)	DHT (n=30)	NDHT (n=42)	p value
LA (mm)	33.0±3.3*	36.1±4.2	37.1±3.9	0.549
LVEDD (mm)	45.7±4.4	46.0±5.0	48.2±4.2	0.889
LVESD (mm)	27.5±3.7	27.4±4.5	30.3±3.8	0.988
LVEF Simpson (%)	70.9±5.4	68.4±6.2	71.6±6.4	0.068
IVS (mm)	9.7±1.1*	12.1±1.1	12.5±1.5	0.603
PW (mm)	10.0 (8.0-10.1)*	12.0 (11.7-13.0)	12.5 (11.0-13.0)	0.820
SV (mL)	68.8±15.3	72.8±19.6	72.0±16.7	0.682
FS %	40.3±4.9	38.6±5.2	41.2±5.8	0.110
LVMi (gr/m ²)	101.1±19.6*	141.3±22.8	157.7±26.6	0.019+
Mitral E (m/s)	0.79±0.14	0.72±0.10	0.72±0.17	0.985
Mitral A (m/s)	0.62±0.11*	0.88±0.20	0.86±0.15	0.820
Mitral E/A	1.33 (1.05-1.50)*	0.90 (0.79-1.07)	0.82 (0.72-0.93)	0.008+
Mitral E/Em	5.7±1.3*	7.8±1.9	9.2±2.5	0.040+
LATEm (m/s)	0.16±0.04*	0.10±0.03	0.09±0.02	0.890
LATAm (m/s)	0.11 (0.08-0.12)*	0.13 (0.10-0.16)	0.13 (0.09-0.14)	0.699
LATS (m/s)	0.12±0.03*	0.10±0.02	0.09±0.02	0.644
SEPEm (m/s)	0.11±0.02*	0.07±0.01	0.07±0.02	0.984
SEPA m (m/s)	0.09±0.02	0.10±0.02	0.10±0.02	0.999
SEPS (m/s)	0.10 (0.08-0.11)	0.09 (0.08-0.10)	0.09 (0.07-0.10)	0.932
DT (ms)	179.8±30.0*	202.1±44.2	229.9±41.8	0.028+
MPI (%)	0.522±0.071*	0.665±0.141	0.622±0.107	0.210

Conventional 2D echocardiographic, pulse and tissue Doppler findings of control and patient groups. Abbreviations: LA: left atrium, LVEDD: left ventricular end-diastolic diameter, LVESD: left ventricular end systolic diameter, LVEF: left ventricular ejection fraction, IVS: interventricular septum, PW: posterior wall, SV: Stroke volume, LVMi: left ventricular mass index, FS: fractional shortening, mitral E: early diastolic mitral flow velocities, mitral A: late diastolic mitral flow velocities, LATEm: mitral lateral e, LATAm: mitral lateral a, LATS: mitral lateral s, SEPEm: mitral septal e, SEPA m: mitral septal a, SEPS: mitral septal s, DT: mitral deceleration time, MPI: myocardial performance index * Between patient and control p<0.05, + Between DHT and NDHT group p<0.05

Table 4.

Variables	Control (n=39)	DHT (n=30)	NDHT (n=42)	p value
Inter-atrial conduction time (ms)	15.9±6.9*	25.5±3.9	32.2±7.4	<0.001+
Left atrial conduction time (ms)	9.2±5.6*	14.9±3.7	18.2±6.0	0.016+
Right atrial conduction time (ms)	6.7±2.3*	10.5±2.1	14.2±5.2	<0.001+
LAVImax (cm ³ /mL)	17.9±4.2*	20.3±5.3	21.6±6.3	0.663
LAVImin (cm ³ /mL)	6.5±2.1*	9.3±4.0	10.6±4.3	0.453
LAVIp (cm ³ /mL)	11.8 (9.1-12.4)*	13.5 (12.0-20.0)	17.5 (12.0-20.1)	0.181
LA S-S	43.3 (38.4-57.9)*	34.2 (29.7-38.7)	27.7 (22.7-32.2)	<0.001+
LA S-E	23.1 (18.9-30.5)*	18.2 (16.6-20.1)	14.4 (11.6-16.8)	<0.001+
LA S-A	21.8 (17.5-28.1)*	15.8 (13.5-17.9)	12.7 (9.9-14.5)	<0.001+

Atrial electromechanical delay, LA volume indexes and LA strain values of control and patient groups. Abbreviations: LAVImax: Left atrium maximum volume index, LAVImin: Left atrium minimum volume index, LAVIp: left atrium presystolic volume index, LA S-S: left atrial strain S, LA S-E: left atrial strain E, LA S-A: left atrial strain A *Between patient and control group p<0.05, + Between DHT and NDHT group p<0.05.

Table 5.

Variables	Control (n=39)	DHT (n=30)	NDHT (n=42)	p value
Pmax (s)	0.105 (0.10-0.11)*	0.117 (0.10-0.12)	0.126 (0.12-0.14)	<0.001+
Pmin (s)	0.056 (0.05-0.07)	0.055 (0.05-0.06)	0.057 (0.05-0.06)	0.756
PWD (s)	0.045 (0.04-0.05)*	0.062 (0.06-0.07)	0.069 (0.06-0.08)	0.004+
PTF (mm*s)	0.033±0.02*	0.055±0.02	0.066±0.02	0.02+

P wave dispersion and PTF values of control and patient groups. Abbreviations: Pmax: the longest P wave duration, Pmin: the shortest P wave duration, PWD: P wave dispersion, PTF: P terminal force * Between patient and control group p<0.05, + Between DHT and NDHT group p<0.05.

Table 6.

Variables	r	p	%95 CI
LA S-E	-0.475	<0.001	0.328-0.610
LA S-S	-0.507	<0.001	0.373-0.625
LA S-A	-0.465	<0.001	0.341-0.590
LAVIp	0.414	<0.001	0.275-0.541
LAVImin	0.333	<0.001	0.201-0.472
LAVImax	0.246	0.009	0.075-0.407
Inter-atrial conduction time	0.549	<0.001	0.397-0.673
Intra-right conduction time	0.505	<0.001	0.347-0.637
Intra-left conduction time	0.423	<0.001	0.250-0.570

Pearson correlation (r) between PTF and other LA dysfunction parameters. Abbreviations: PTF: P terminal force; LA: left atrium; LA S-E: left atrial strain E; LA S-S: left atrial strain S; LA S-A: left atrial strain A; LAVIp: presystolic left atrial volume index; LAVImin: minimum left atrial volume index; LAVImax: maximum left atrial volume index CI: confidence interval

Hypertension

OP-144

Using lens opacities to predict salt sensitivity hypertension

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Background and Aim: Salt-sensitive hypertension (SSH) accounts for about the half of all HT cases. Na⁺ / K⁺-ATPase activity is impaired in patients with SSH. A definite diagnosis of salt sensitivity is difficult, expensive, and associated with low patient compliance. Impaired Na⁺ /K⁺-ATPase activity in the lens epithelium results in cortical opacities in the peripheral equatorial region of the lens. The present study is the first to investigate the potential of using lens opacity to predict SSH.

Methods: Patients who were measuring their blood pressure at home were taken to study. At first, patients were given low Na diet (50 mmol(3 gr)/day) for the duration of one week and then patients were switched to high Na diet (250 mmol(15 gr)/day) for another one week. Salt sensitivity hypertension(SSH) was diagnosed when the difference in mean arterial blood pressure values obtained during high and low Na diets is ≥%10. On the other hand, salt resistance hypertension (Non-SSH, SRH) was diagnosed when the difference is less than 10%. The study included 163 SSH and 142 Non-SSH, SRH patients (305 HT patients total) and 124 control patients without HT, aged 40–80 years. The HT and control groups were subdivided into age groups of <50, 50–59, 60–69, and ≥70 years. Patients with apparent cataracts, diabetes mellitus, smoking, hypo/hypercalcemia, hyperparathyroidism, eye trauma, excessive exposure to sunlight, excessive uptake of heat or radiation, history of using corticosteroids or digoxin, coronary artery disease, cardiac failure, renal failure, hyperlipidemia, or atrial fibrillation were excluded from the study. Estimation of Daily Salt Intake (EDSI) was estimated by measuring the Na and creatinine contents of second morning urine samples, according to the Kawasaki formula. The presence of peripheral cortical lens opacity was biomicroscopically examined by two researchers using the diffuse, direct, Scheimpflug and retroillumination from the fundus methods.

Results: The incidence of opacity was higher in the SSH than in the Non-SSH group for patients with HT for >6 years, HT stage 2 or 3, an EDSI < 10 g or 10–14 g, and retinal grade 1 or 2. The incidence of opacity was similar between the SSH and Non-SSH groups for patients with HT for 1–5 years, stage 1 HT, an EDSI ≥15 g, retinal grade 3, and LVH.

Conclusions: Lens opacity contributes to the diagnosis of SSH, with a sensitivity of 40.5%, specificity of 84.5%, and PPV of 75% at ages 40–80 years. Further large-scale studies would yield more sensitive results.

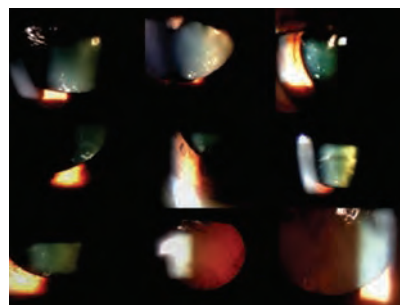


Figure 1. Examples of cortical lens opacity, small opacity, total (small+ large) opacity, large opacity, retroillumination view, Coexistence of initial cortical cataract and opacity (LOCS2, C1 class).

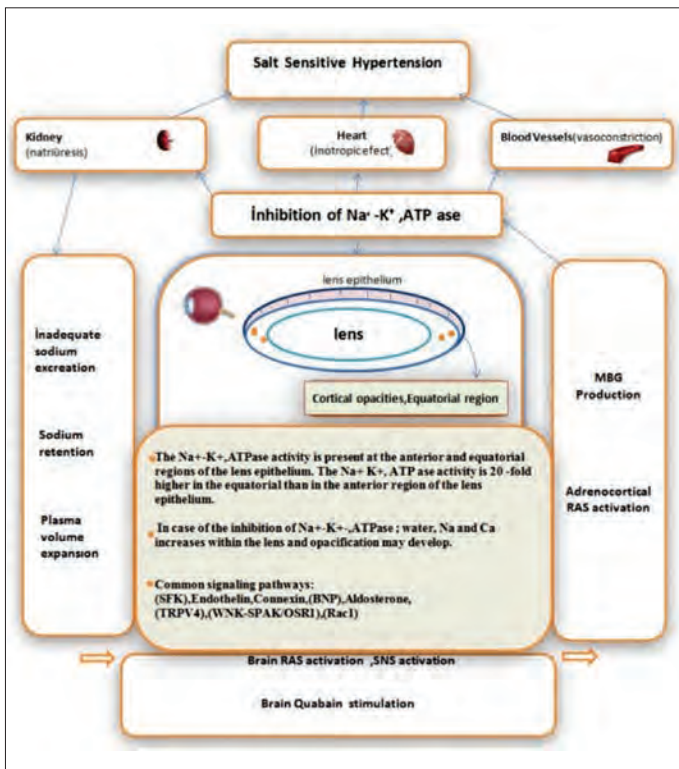


Figure 2. Potential mechanism of salt-sensitive hypertension and lens opacity. SNS:Sympathetic nervous system RAS:Renin Angiotensin System MBG: Marinobufagenin.

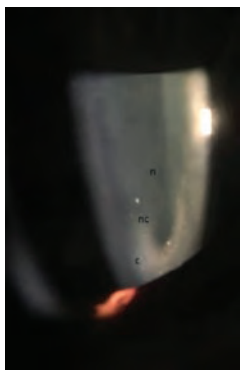


Figure 3. Examples of Scheimpflug image, cortical lens opacities. n: nucleus; c: cortex; nc: nucleocortical junction.

Table 1. Demographic characteristics of the cases by the control and hypertension groups

	Control group (n=124)	HT group (n=305)	p-value
Age (years)	59.9±9.6	59.1±10.3	0.430*
Age groups			0.438†
<50 years	19 (15.3%)	54 (17.7%)	
50-59 years	42 (33.9%)	109 (35.7%)	
60-69 years	42 (33.9%)	80 (26.2%)	
≥70 years	21 (16.9%)	62 (20.3%)	
Gender			0.994†
Male	65 (52.4%)	160 (52.5%)	
Female	59 (47.6%)	145 (47.5%)	
BMI(kg/m2)	28,33±2,64	28,5±3,13	0,569‡
BP(mmHg)	120±10/64±6,9	SS: 145± 15/85± 14	0,001‡
		SR: 139± 11/84± 10	0,001
		SSE: 182± 11 /105± 15	0,001

*Student's t test, †Pearson's Chi-square test, ‡Independent Samples Test BMI: Body Mass Index BP:Blood Pressure SS:Salt Sensitive SR:Salt Resistance SSE:Salt Sensitive Emergency Room

Table 2. Total lens opacity incidence by control and hypertension groups

	Control group (n=124)	HT group (n=305)	p-value
Overall			
<50 years	1/19 (5.3%)	5/54 (9.3%)	1.000*
50-59 years	4/42 (9.5%)	26/109 (23.9%)	0.080†
60-69 years	6/42 (14.3%)	28/80 (35.0%)	0.027†
≥70 years	4/21 (19.0%)	29/62 (46.8%)	0.047†
Total	15/124 (12.1%)	88/305 (28.9%)	<0.001‡

* Fisher's exact test, †Continuity corrected Chi-square test, ‡Pearson's Chi-square test.

Table 3. Total lens opacity development rates among hypertensive patients by salt-sensitive and salt-resistant groups and other clinical characteristics

	Salt resistant	Salt sensitive	p-value *	Overall
Duration of HT				
1-5 years	9/74 (12.2%)	19/76 (25.0%)	0.071†	28/150 (18.7%)
6-10 years	8/41 (19.5%)	25/49 (51.0%)	0.004†	33/90 (36.7%)
>10 years	5/27 (18.5%)	22/38 (57.9%)	0.004†	27/65 (41.5%)
p-value ‡	0.516§	<0.001§		<0.001§
HT stage				
I	2/42 (4.8%)	3/36 (8.3%)	0.657¶	5/78 (6.4%)
II	16/80 (20.0%)	37/95 (38.9%)	0.011†	53/175 (30.3%)
III	4/20 (20.0%)	26/32 (81.3%)	<0.001†	30/52 (57.7%)
p-value ‡	0.073§	<0.001§		<0.001§
Retinal grade				
I	16/108 (14.8%)	38/106 (35.8%)	<0.001§	54/214 (25.2%)
II	3/29 (10.3%)	26/53 (49.1%)	<0.001†	29/82 (35.4%)
III	3/5 (60.0%)	2/4 (50.0%)	1.000¶	5/9 (55.6%)
p-value ‡	0.017§	0.258§		0.045§
Salt intake				
<10 g	3/50 (6.0%)	19/52 (36.5%)	<0.001†	22/102 (21.6%)
10-14 g	18/84 (21.4%)	45/106 (42.5%)	0.002§	63/190 (33.2%)
≥15 g	1/8 (12.5%)	2/5 (40.0%)	0.510¶	3/13 (23.1%)
p-value ‡	0.056§	0.776§		0.102§
LVH				
Absent	18/120 (15.0%)	57/136 (41.9%)	<0.001§	75/256 (29.3%)
Present	4/22 (18.2%)	9/26 (34.6%)	0.342†	13/48 (27.1%)
p-value ‡	0.749¶	0.634†		0.891†
Total	22/142 (15.5%)	66/163 (40.5%)	<0.001§	88/305 (28.9%)

*The comparisons between salt resistance and salt sensitive groups, † The comparisons within salt resistance, salt sensitive groups and overall cases, ‡ Continuity corrected Chi-square test, § Pearson's Chi-square test, ¶ Fisher's exact test.

Table 4. The diagnostic performance of using the total lens opacity to predict salt-sensitive hypertension in hypertensive patients

Overall(Femal e+Male)	N	Sensitiv ity	C.I (% 95)	Specific ity	C.I (% 95)	PPV	C.I (% 95)	NPV	C.I (% 95)	Accura cy	C.I (% 95)	p- valu e	
		TP/(TP+ FN)		TN/(TN +FP)		TP/(TP +FP)		TN/(TN +FN)		(TP+T N)/N			
<50 years	54	4/27 (14.8%)	4,1 to 33	26/27 (96.3%)	99,9	81 (80.0%)	4/5 (80.0%)	28/99 (28.3%)	26/99 (26.3%)	30/54 (55.5%)	42 to 68	0.35 1†	
50-59 years	109	18/55 (32.7%)	20 to 46	46/54 (85.2%)	93,3	72 (69.2%)	18/26 (69.2%)	48/85 (56.5%)	210/217 (96.8%)	64/109 (58.7%)	49 to 67	0.04 9‡	
60-69 years	80	21/44 (47.7%)	32 to 63	29/36 (80.6%)	91,8	63 (79.3%)	21/28 (75.0%)	55/89 (61.8%)	110/177 (62.1%)	50/80 (62.6%)	51 to 73	0.01 6‡	
≥70 years	62	23/37 (62.2%)	44 to 77	19/25 (76.0%)	90,6	54 (79.3%)	23/29 (79.3%)	60/92 (65.2%)	19/33 (57.6%)	60,2 to 92,0	42/62 (67.7%)	56 to 79	0.00 7‡
Total	305	66/163 (40.5%)	32 to 48	120/142 (84.5%)	90,0	77 (75.0%)	66/88 (75.0%)	64/83 (77.1%)	120/217 (55.3%)	186/305 (60.9%)	55 to 66	<0.0 01¶	

N: Number of total cases, TP: true positive, FN: false negative, TN: true negative, FP: false positive, PPV: positive predictive value, NPV: negative predictive value, * Fisher's exact test, †Continuity corrected Chi-square test, ‡ Pearson's Chi-square test.

Cardiovascular surgery

OP-145

Feasibility of robotically assisted concomitant procedures during mitral valve operations

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Background and Aim: Related to concerns of the potential for prolonged duration of cardiopulmonary bypass times many surgical teams are not enthusiastic for concomitant procedures during robotic mitral valve

operations. However in many cases tricuspid valve intervention or ablation for atrial fibrillation is required during mitral valve operations. With the advances of surgical experience and technological support this concern is gradually diminishing. Here in this report, we present our clinical experience with the robot assisted surgery of the concomitant procedures during mitral valve interventions.

Methods: From March 2010 to February 2018, a total of 238 patients underwent robotic cardiac procedures in our center. Additional procedures (n=76) beside mitral valve intervention was performed in 34 of these patients (14.2%) with robotic assistance using the da Vinci Si, Xi HD surgical systems.

Results: Totally 76 robotic assisted concomitant procedures were performed during mitral valve repair (n=11) or replacement (n=23) in 34 patients. These procedures were cryoablation (n=28), TV repair (n=6), TV replacement (n=2), LAA ligation (n=30), ASD and PFO closure (n=8), LA thrombectomy (n=1) and paravalvular leak repair (n=1). Normal sinus rhythm was restored in 85% (24/28) of patients after cryoablation in early postoperative period. Two patients (5.8%) had permanent pacemaker. There was one mortality at early postoperative period (2.9%) and the reason was hemorrhage related to posterior left ventricular wall rupture. Any kind of blood product was not used in 82.4% of patients. Renal failure requiring dialysis was not needed in any patient.

Conclusions: As a conclusion, translating the outcomes of the present study to other centers may be difficult related to previous experience of our surgical group on minimal invasive and robotic mitral surgery. However, robotic assisted concomitant procedures beside mitral valve operations can be performed with low complication rates following the learning curve with the aid of technological improvements.

Other

OP-146

Coronary sinus diameter to inferior vena cava diameter ratio as a novel predictor of progression to cardiac tamponade

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Background and Aim: Pericardial tamponade constitutes one of the medical emergencies in which early suspicion is critical. Coronary sinus (CS) is an intrapericardial structure that can be compressed in cardiac tamponade in contrast to extrapericardial structures such as inferior vena cava (IVC). Computed tomography provides spatial information where intra- and extracardiac structures can easily be separated. Therefore, in this study, we assessed the utility of CS to IVC ratio to predict to the progression of cardiac tamponade in patients with pericardial effusion.

Methods: This study consisted of 66 patients with moderate to severe pericardial effusion. Diagnosis of tamponade was established by hemodynamic findings with the evidence of echocardiographic right ventricular compression.

Results: During the in-hospital follow-up, cardiac tamponade occurred in 23 patients. Patients with cardiac tamponade had 40% lower CS diameter (5.3±1.8 vs 8.8±2.6 mm p<0.001) and 35% lower CS/IVC ratio (20.7±5.5 vs 34.7±10.5% p<0.001) than patients without tamponade respectively. In multivariate analysis, the CS parameters independently predicted cardiac tamponade in two different models (Nagelkerke r square for CS was 53.7, and CS/IVC ratio was 72.1). One mm increase in CS and one percent increase in CS/IVC ratio were associated with 59%, and 39% decreased odds for progression to tamponade respectively (Both p values <0.001). A cut off 6.85 mm for CS differentiated tamponade with an accuracy of 95% (sensitivity 87.0% and specificity 86.0%).

Conclusions: Tomography derived CS parameters, and especially CS/IVC ratio predicts progression to cardiac tamponade in patients with moderate to severe pericardial effusion. Further studies are warranted to validate and replicate these results especially with radiation free procedures such as echocardiography.

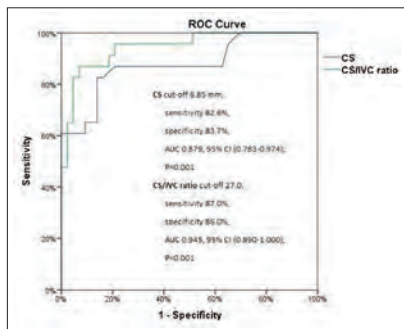


Figure 1.

Other

OP-147

Is there any difference among radial or femoral access regarding radiation parameters while performing coronary procedures?

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Background and Aim: Radial access is nowadays more frequently used during coronary interventions and recommended over femoral access in our contemporary cardiology guidelines. We hereby investigated whether there is a difference among radial and femoral access regarding the parameters of ionizing radiation as dose-area product (DAP), which demonstrates the stochastic risk (carcinogenic and genetic effects) of cancer, number of exposures (NoE), effective dose (ED), which demonstrates the deterministic effect as radiation-induced skin injury and cataract, and fluoroscopy time (FT).

Methods: 4215 patients underwent diagnostic or interventional coronary procedures (ICPs) were enrolled to the study excluding lesions of bifurcation and chronic total occlusion due to the fact that these lesions require femoral access owing to the need of larger size access sheath or bilateral access. ICPs were also classified as elective (stable angina) and acute coronary syndromes, demonstrated in Table 1.

Results: In patients undergoing diagnostic CAG, there was no significant difference regarding DAP, NoE, ED and FT among femoral and radial routes, whereas in patients with stable angina and ACS, PCI via radial access was significantly related to increased DAP, NoE, ED and FT, as shown in Table 2.

Conclusions: In spite of an increased experience of performing CAG via radial route, PCI of coronary lesions via radial route was still related to an enhanced risk of radiation in our trial. On the other hand, diagnostic CAG via radial access was not related to an increased risk of radiation, compared to femoral route. To conclude, we demonstrated that performing diagnostic CAG via radial route had similar results with femoral route regarding radiation exposure. Therefore, it may be deemed as a safe and preferred technique due to similar results with femoral route regarding radiation exposure. However, PCI of coronary lesions via radial route may increase the stochastic risk (demonstrated as DAP) and deterministic effects (demonstrated as ED) of radiation, compared to femoral route despite the fact that radial route is strongly recommended over femoral route in contemporary cardiology guidelines to decrease the access site complications. Therefore, radial coronary interventions should be done with the awareness of possible increased risk of radiation exposure.

Table 1.

	Diagnostic CAG (n, %)	PCI of Stable Angina (n, %)	PCI of ACS (n, %)
Femoral	856 (%41,4)	407 (%57,2)	989 (%69)
Radial	1213 (%58,6)	305 (%42,8)	445 (31)
Total	2069 (%100)	712 (%100)	1434 (%100)

ACS: Acute Coronary Syndrome, CAG: Coronary Angiography, n: Number, PCI: Percutaneous Coronary Intervention

Table 2.

	Diagnostic CAG	P value	PCI of Stable Angina	P value	PCI of ACS	P value
DAP	Femoral 798,7 (126,78-5907,71)	0,580	3006,31 (428,72-14546,2)	<0,001	3492,88 (563,07-20854,49)	0,006
	Radial 721,88 (129,54-4767,13)	0,580	3241,47 (625-12787,69)	<0,001	3686,68 (510,5-23311,97)	0,006
NoE	Femoral 114 (0-585)	0,260	257 (45-770)	<0,001	284 (17-2018)	0,139
	Radial 116 (21-571)	0,260	274 (31-837)	<0,001	287 (70-1610)	0,139
ED	Femoral 64,75 (8,84-768,60)	0,802	259,24 (39,77-1824,92)	<0,001	323,27 (38,96-3938,5)	0,001
	Radial 61,94 (7,49-789,56)	0,802	284,39 (37,3-1636,8)	<0,001	349,27 (33,18-4189,03)	0,001
FT	Femoral 1,9 (0,3-27,8)	0,156	7,3 (1,1-36,7)	<0,001	7,8 (0,5-48,9)	0,003
	Radial 1,6 (0,5-18,1)	0,156	7,9 (2,2-39,6)	<0,001	8,1 (2-54,1)	0,003

ACS: Acute Coronary Syndrome, CAG: Coronary Angiography, DAP: Dose-Area Product, ED: Entrance Dose, FT: Fluoroscopy Time, NoE: Number of Exposures, PCI: Percutaneous Coronary Intervention

Other

OP-148

The prognostic role of fragmented QRS complex in acute myocarditis

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Background and Aim: Although a fulminant course of the myocarditis is difficult to predict, it may lead to acute heart failure and death. Previous studies have demonstrated that reduced left ventricular systolic function and prolonged QRS duration can predict the fulminant course. This study aimed to identify whether fragmented QRS complex (fQRS) could also be predictive of fulminant disease in this population.

Methods: We retrospectively included 156 patients diagnosed with acute myocarditis. The fQRS was defined as the presence of ≥1 additional R wave (R') or notch on the R/S waves in ≥2 contiguous leads. They were divided into the fulminant group (n=18) and the non-fulminant group (n=138). Multivariate logistic regression analysis was used to identify the independent factors predictive of fulminant disease.

Results: Fragmented QRS developed in 11 (61%) in the fulminant group and only 10 patients (7%) in the non-fulminant group (p<0.001). Patients with fulminant myocarditis had a higher mortality rate than those with non-fulminant disease (44.6% vs. 0%, p<0.001). Multivariate analysis revealed that the presence of fQRS (p=0.019), longer Tpe/qt ratio (p=0.022) and clinical heart failure (<0.001) were significant predictors associated with a fulminant course of myocarditis.

Conclusions: The presence of fQRS complex, as a simple and feasible electrocardiographic marker, seems to be a novel predictor fulminant myocarditis. This simple parameter may be used in identifying patients at high risk for fulminancy and so early mechanical support could provide improved patient outcomes.

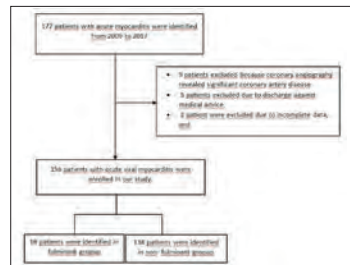


Figure 1. Study cohort.

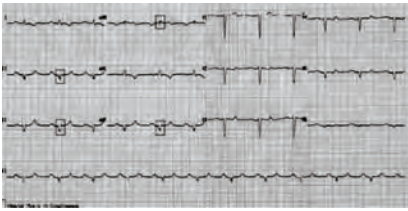


Figure 2. Examples of fQRSs in our patients with fulminant myocarditis.

Table 1. Demographical, laboratory, electrocardiographic, echocardiographic and clinical characteristics of study and control subjects

	Non-fulminant group (N = 138)	Fulminant group (N = 18)	p-value
Age (years)	28 ± (16.01)	34 ± (14.85)	0.144
Female Gender, n(%)	20 (14)	3 (16)	0.807
White Blood Cell(x10 ³ /μL)	8.8 (6.5-10.0)	8.8 (8.0-9.6)	0.267
CRP(mg/dL)	5.0 (2.0-12)	4.0 (1.4-9.0)	0.170
Creatinin (IU/L)	0.80 (0.60-0.90)	0.79 (0.90-2.08)	0.158
Glucose(mg/dL)	97 (88-100)	102 (96-106)	0.056
Troponin-I (ng/L)	0.46 (0.08-2.56)	0.49 (0.06-2.58)	0.601
HR (per minute)	70 (65-85)	79 (63-86)	0.120
QRS (ms)	92 (78-98)	101 (80-110)	0.647
PR (ms)	140 (130-160)	160 (158-170)	<0.001
QT (ms)	390 (370-402)	381 (352-401)	0.198
QTc (ms)	426 (401-447)	481 (448-503)	<0.001
Tp-e interval, ms	81 (76-84)	82 (74-101)	0.469
Tp-e/QT ratio	0.20 (0.18-0.22)	0.24 (0.20-0.26)	0.010
Tp-e/QTc ratio	0.18 (0.19-0.21)	0.17 (0.15-0.20)	0.045
Fragmented qrs n(%)	10 (7)	11 (61)	<0.001
IVS (mm)	0.8 (0.8-1.0)	0.9 (0.8-1.35)	0.267
LVPW (mm)	0.9 (0.7-1.0)	1.0 (0.8-1.5)	0.355
LA dimension (mm)	3.2 ± 0.7	3.5 ± 0.9	0.061
LVEDs (mm)	2.7 (2.4-3.5)	2.9 (1.7-3.2)	0.689
LVEDd (mm)	4.6 (4.0-5.0)	4.9 (4.5-6.5)	0.090
LVEF (%)	62 (55-67)	25 (23-30)	<0.001
Pericardial effusion n(%)	30 (21)	11 (61)	<0.001
Cardiac tamponade n(%)	8 (5)	5 (27)	0.002
Clinical heart failure n(%)	1 (0.7)	17 (94)	<0.001
ECMO- LVAD n(%)	0 (0)	16 (88)	<0.001
1 month mortality n(%)	0 (0)	8 (44.5)	<0.001

Data are given as mean ± SD or %. CRP, C-reactive protein; ECMO, Extracorporeal Membrane Oxygenation; HR, heart rate; IVS, intraventricular septum; LA, left atrium; LVEDd, end-diastolic left ventricular diameter; LVEDs, end-systolic left ventricular diameter; LVEF, left ventricular ejection fraction; LVPW, left ventricular posterior wall; LVAD, Left Ventricular Assist Device

Table 2. Univariate and multivariate logistic regression analysis showing the predictors for fulminancy

Variables	Univariable OR (95% CI)	p-value	Multivariable OR (95% CI)	p-value
Age	1.071 (1.032-1.113)	<0.001	1.044 (0.995-1.106)	0.144
Heart Rate	1.059 (0.914-1.195)	0.108	-	-
Tp-e/QT ratio	1.062 (1.034-1.091)	<0.001	1.024 (1.017-1.071)	0.022
Fragmented QRS	20.114 (6.398-63.239)	<0.001	6.825 (1.370-13.071)	0.019
Cardiac Tamponade	6.250 (1.783-21.911)	0.004	5.337 (0.711-40.072)	0.104
LVEF, (%)	1.150 (1.034-1.191)	0.035	1.013 (0.896-1.123)	0.234
Clinical Heart Failure	2329 (139-38966)	<0.001	999 (35-28013)	<0.001

Data are given as mean ± SD or %. CI, confidence interval; LVEF, left ventricular ejection fraction; OR, odds ratio.

Other

OP-149

Serum matrix metalloproteinases as biomarkers for myocardial fibrosis in patients with hypertrophic cardiomyopathy

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Background and Aim: Hypertrophic cardiomyopathy (HCM) is defined by the presence of unexplained left ventricular hypertrophy, myocyte disarray, and interstitial fibrosis. An increase in extracellular matrix produces interstitial fibrosis, by raised amounts of collagen type I/III. Regions of myocardial late gadolinium enhancement by cardiac magnetic resonance (CMR) represented increased myocardial collagen. Regarding

the role of matrix metalloproteinases (MMPs) in myocardial remodeling and subsequent fibrosis, the aim of our study was to explore the relation between MMP system and myocardial late gadolinium enhancement by CMR (as expression of image-documented fibrosis) in HCM.

Methods: To the research; 53 HCM patients and 17 control group have been admitted to our clinic who diagnosed by Transthoracic Echocardiography and Cardiac MRI between the dates January 2015 – March 2018. They have been divided into two groups as the patients who have fibrosis and who have not. Their plasma MMP-9 levels has been compared.

Results: The patients in HCM group were older than the patients in non-HCMP group, the incidence of hypertension, diabetes and history of coronary artery disease were higher in HCM group than non- HCM group. There were no significant differences in gender and LVEF when the study and control groups were compared. LV MASS, LVOT gradient, LVEDV, and LVESV were significantly higher in the HCM group than in the control group. Serum MMP-9 levels was significantly higher in the HCM group (1570.9±84 vs 1170.3±100, p<0.001). We divided the study population into 2 subgroups according to the presence of myocardial fibrosis. Patients with myocardial fibrosis have higher maximum wall thickness (p=0.017), and lower LVESV (P = 0.007). There were no differences between groups regarding age, sex, the incidence of hypertension, diabetes, history of coronary artery disease, LV MASS, LVOT gradient, and LVEDV. Serum MMP-9 levels was significantly higher in the fibrosis group compared with the without fibrosis group (1618±62 vs 1531±50, p<0.001). In HCM group, MMP-9 was positively correlated with quantity of LGE (r=0.649, p<0.001). Significant univariate predictors of myocardial fibrosis were history of coronary artery disease, LVMI, and MMP-9. Using multivariate logistic regression analysis, we found that LVMI (OR: 1.056, CI: 1.004-1.112, p=0.035) and MMP (OR: 1.031, CI: 1.013-1.049, p=0.003) were independent predictors of myocardial fibrosis. Results of multivariate logistic regression analysis are presented The ROC analysis yielded a cut-off value of 1580.5 for MMP-9 to predict myocardial fibrosis with 84% sensitivity and 83% specificity (AUC=0.884, 95% CI=0.787-0.981, p<0.001).

Conclusions: Matrix metalloproteinase 9 is independently associated with gadolinium enhancement on CMR in patients with hypertrophic cardiomyopathy, suggesting that the MMP system has an important role in cardiac remodeling and fibrosis in this condition.

Other

OP-150

Silent cerebral infarction in anticoagulated patients with non-valvular atrial fibrillation

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Background and Aim: Cerebral infarction in patients with atrial fibrillation (AF) may vary from being clinically silent to catastrophic. Elevation of neuron-specific enolase (NSE) in the absence of any clinically apparent stroke or transient ischemic attack (TIA), so-called silent cerebral infarction (SCI), may be associated with neurologic deficits, cognitive decline and even increased mortality. We aim to evaluate the prevalence of SCI in patients with non-valvular atrial fibrillation (AF) who are taking oral anticoagulants.

Methods: Blood samples were collected from 100 consecutive patients with non-valvular AF admitted to outpatient clinic. NSE levels of greater than 12 ng/ml was considered as SCI.

Results: Patients were mainly female with a mean age of 70 years. Forty-nine of them (49%) were taking warfarin. Mean INR level was 2.3±1.1. Fifty-one patients (51%) were on direct oral anticoagulant (DOAC) treatment (dabigatran (n=7), rivaroxaban (n=13) and apixaban (n=31)). Mean CHA2DS2-VASc score of the study population was 3.8±1.5. Forty-three patients (43%) were found to have NSE elevation. They were older and more likely to have history of chronic heart failure and previous stroke/TIA. Increased left atrial diameter, reduced glomerular filtration rate, and higher CHA2DS2-VASc score were other factors associated with SCI. Patients taking DOACs and patients who were taking aspirin on top of oral anticoagulant treatment were less likely to have SCI. Multivariate analysis demonstrated higher CHA2DS2-VASc score (OR: 2.6; 95% CI: 1.3-5.1; p=0.007) and use of warfarin (OR: 3.8; 95% CI: 1.2-11.9; p=0.02) as independent predictors of SCI.

Conclusions: Silent brain injury is highly prevalent among patients with non-valvular atrial fibrillation despite the use of oral anticoagulant therapy.

Table 1. Clinical characteristics of silent cerebral ischemia (+) and (-) patients

	Silent cerebral infarct (+) (n=43)	Silent cerebral infarct (-) (n=57)	P value
Age, mean ± SD, (years)	74±12	67±10	0.002
CHA2DS2-VASc	4.7±1.4	3.2±1.2	<0.001
HfrEF	24(55%)	20(46%)	0.04
Previous stroke/TIA	12(28%)	2(4%)	<0.001
Asetil salisilic acite	14(33%)	32(56%)	0.02
Warfarin	26(61%)	23(40%)	0.046
INR value	2.01±0.93	2.67±1.19	0.035
Direct-oral anticoagulants	17(40%)	34(60%)	0.046
LAD	5.3±0.9	4.9±0.6	0.04

HfrEF: Heart failure with reduced ejection fraction, LAD: Left atrial diameter, TIA: Transient ischemic attack

Hypertension

PP-001

Effect of situational anxiety and depression on 24-hour ambulatory blood pressure monitoring in patients with hypertension

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Background and Aim: Normally, there is a physiological decrease in blood pressure (BP) during sleep relative to the wakefulness; this is called as dipping pattern. Autonomic nervous system abnormalities, dietary factors, smoking, sedentary lifestyle, anxiety disorders all have been suggested as potential etiological factors of this impaired BP circadian rhythm. In this study, starting with the possibility of ambulatory blood pressure monitoring (ABPM) induced stress and anxiety in hypertensive patients, we intend to compare anxiety level in dipper and non-dipper hypertensive patients.

Methods: A total of 326 medically treated hypertensive patients were enrolled in the study. 162 patients were categorized as dipper hypertensives and the remaining were categorized as non-dipper hypertensives. All patients underwent 24-hour ABPM using non-dominant arm. All patients completed self-report questionnaires which include General Health Questionnaire (GHQ-12), State-trait anxiety inventory (STAI) form TX-I and STAI form TX-II before ABPM. STAI FORM TX-I questionnaire was repeated after ABPM.

Results: Clinical and demographic characteristics of the two groups were similar (Table 1, 2). There was no statistically significant difference in STAI FORM TX-II score between two groups. GHQ and STAI FORM TX-I score of the non-dipper group were statistically significantly higher than the dipper group (Table 3). Following ABPM, There was a statistically significant decrease in STAI FORM TX-I score in non-dipper group. Although, the scores on STAI FORM TX-I decreased in dipper group, this decrease did not reach statistical significance (Table 4). A positive correlation was found between age and STAI FORM TX-I score (r=0.531, p=0.0001). There were significant differences in GHQ-12 and STAI FORM TX-I scores among groups according to education level (F=6.175, p=0.003 and F=5.877, p=0.003, respectively). Post hoc analysis revealed that the significance was due to the differences between group 3 (university level) and group 1 and 2 (elementary school and high school level). STAI FORM TX-I scores of the patients differed significantly in terms of their socioeconomic status (F=5.9701, p=0.003). Patients with higher socioeconomic status had lower STAI FORM TX-I scores compared to the middle and poor socioeconomic status.

Conclusions: 24-hour ABPM could induce anxiety in elderly, poorer and less educated patients, providing information about the device and the process may help patients to overcome their anxiety.

Table 1. Clinical and demographic characteristics of the patients

	Non-dipper group (n=164)	Dipper group (n=162)	P
Sex			0.288
Female n,(%)	83 (50.6)	87(53.7)	
Male n,(%)	81 (49.4)	75 (46.3)	
Age	49.9±10.8	50.8±12.0	0.512
Height	1.6±0.09	1.6±0.09	0.725
Weight	76.5±12.1	77.7±12.4	0.362
BMI	26.9±3.3	27.2±3.6	0.496
Smoking n,(%)	41(25)	42(25.9)	0.474
Alcohol n,(%)	19(11.58)	22(13.58)	0.354
Educational level			0.867
Elementary n,(%)	90 (54.87)	92 (56.79)	
High school n,(%)	52 (31.70)	47 (29.01)	
University n,(%)	22 (13.41)	23 (14.19)	
Family type			0.507
Nuclear n,(%)	115 (70.12)	122 (75.30)	
Joint n,(%)	37 (22.56)	32 (19.75)	
Broken n,(%)	12 (7.31)	8 (4.93)	
Socioeconomic status			0.794
Poor n,(%)	25 (15.24)	29 (17.90)	
Middle n,(%)	105 (64.02)	99 (61.11)	
High n,(%)	34 (20.73)	34(20.98)	

Table 2. Biochemical parameters and the antihypertensive treatment of the two groups

	Non-dipper group (n=164)	Dipper group (n=162)	p
Glucose (mg/dl)	99.0±10.9	97.1±9.6	0.086
Total cholesterol (mg/dl)	187.9±32.4	189.1±30.5	0.740
LDL-C (mg/dl)	121.3±29.3	123.6±32.4	0.503
HDL-C (mg/dl)	44.0±10.4	42.6±9.8	0.227
TG (mg/dl)	148.4±71.2	145.4±71.5	0.706
Systolic blood pressure (DAY) (mmHg)	132.9±11.9	131.6±12.7	0.163
Diastolic blood pressure (DAY) (mmHg)	81.9±9.2	82.4±8.4	0.614
Systolic blood pressure (NIGHT) (mmHg)	127.1±11.5	112.7±26.8	0.000
Diastolic blood pressure (NIGHT) (mmHg)	79.1±6.3	68.2±8.8	0.000
Number of medications used by the patients			0.870
One, n (%)	23 (14.02)	26 (16.04)	
Two, n (%)	118 (71.95)	113 (69.75)	
More than two, n(%)	23 (14.02)	23 (14.19)	
ACE-I/ARB n (%)	81 (49.39)	76 (46.91)	0.368
B-BLOCKER n (%)	57 (34.75)	54 (33.33)	0.439
CCB n (%)	61 (37.19)	64 (39.50)	0.376
AB n (%)	36 (21.95)	30 (18.51)	0.263

Table 3. GSA, STAI FORM TX-I and STAI FORM TX-II scores of the two groups

	Non-dipper group (n=164)	Dipper group (n=162)	p
STAI FORM TX-I score	43.3±11.9	38.3±9.2	<0.0001
STAI FORM TX-II score	35.1±13.1	35.5±9.6	0.753
GSA-12 score	14.0±4.2	13.1±3.3	0.041
STAI FORM TX-I after ABPM	37.7±12.9	37.7±10.1	0.998

Table 4. STAI FORM TX-I before ABPM

	STAI FORM TX-I before ABPM	STAI FORM TX-I after ABPM	p
Non-dipper group	43.3±11.9	37.7±12.9	<0,001
Dipper group	38.3±9.2	37.7±10.1	0,234

Epidemiology

PP-002

Relationship between breast arterial calcifications and lipid profile, plasma atherogenic index, Castelli's risk index and atherogenic coefficient in premenopausal women

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Background and Aim: Several studies have found correlations between breast arterial calcifications (BAC) and hypertension, diabetes mellitus, cardiovascular disease and cardiovascular mortality. Similarly, BAC has predictive value for cerebral, carotid and peripheral artery disease. There are new cardiovascular risk predictors obtained by different combinations of lipid profile parameters. These are; atherogenic index of plasma (PAI); based on two important parameters TG and HDLc, both of which are independent risk factors for coronary artery disease (CAD). Castelli risk index-I (CRI-I); calculated as (TC / HDLc), Castelli risk index-II (CRI-II); as (LDLc/HDLc) is another fraction which involves independent risk factors for CAD, and atherogenic coefficient (AC) calculated as (TC/HDLc) / HDLc is yet another ratio relying on the significance of HDLc in predicting the risk of CAD. The aim of this study was to investigate the relationship between BAC and PAI, AC, CRI-I and CRI-II in premenopausal women.

Methods: The study included 60 premenopausal women aged over 40 years with BAC on mammograms and control group of 60 women without BAC. Serum glucose, TG, LDLc, HDLc, and TC, levels were measured. Lipid indices were calculated using the appropriate formula.

Results: The demographic characteristics of both groups including age, height, weight, body mass index (BMI), blood pressure were similar. Serum LDLc and non-HDLc levels of the patient group were significantly higher than the control group (p=0.007 and p=0.027, respectively), whereas serum HDLc level of the patient group was lower than the control group. PAI, AC, CRI-I and CRI-II were found to be increased significantly in the patient group as compared to their values in the control group (p=0.003, p=0.002, p=0.002 and p=0.003, respectively) (Table 1). BAC was positively correlated with non-HDLc and LDLc levels (r=0.202, p=0.027 and r=0.188, p=0.039, respectively) and negatively correlated with HDLc levels (r=-0.223, p=0.014). There was a significant positive correlation between BAC and PAI, AC, CRI-I and CRI-II (r=0.267 and p=0.003, r=0.282, and p=0.002, r=0.282 and p=0.002, r=0.273 and p=0.003, respectively) (Table 2).

Conclusions: BAC is a valuable tool for the prediction of deranged lipid profile. Our results indicate that BAC is potentially useful tool for the detection of dyslipidemia and early atherosclerosis in premenopausal women.

Table 1. Patient's clinical, lipid profile and ratios among study groups

	BAC positive (n=60)	BAC negative (n=60)	p
Age	45.1 ± 3.1	45.6 ± 3.6	0.3
BMI (kg/m2)	27.4 ± 1.4	26.2 ± 1.8	0.5
Systolic blood pressure (mm Hg)	127.1 ± 8.3	126.3 ± 9.3	0.7
Diastolic blood pressure (mm Hg)	72.5 ± 9.8	70.1 ± 7.3	0.6
Lipid profile (mg/dl)			
Total Cholesterol	207.7 ± 22.9	201.8 ± 18.4	0.135
High Density Lipoprotein Cholesterol	46.5 ± 7.2	49.6 ± 5.8	0.014
Low Density Lipoprotein Cholesterol	133.2 ± 23.2	122.8 ± 18.3	0.007
Triglyceride	150.8 ± 12.3	146.9 ± 12.2	0.093
Non HDLc (TC - HDLc)	161.1 ± 23.5	152.2 ± 18.8	0.027
Lipid ratios			
Atherogenic index of plasma (PAI)	0.51 ± 0.07	0.47 ± 0.06	0.003
Atherogenic coefficient (AC)	3.55 ± 0.83	3.12 ± 0.60	0.002
Castelli's risk index-I (CRI-I)	4.55 ± 0.83	4.12 ± 0.60	0.002
Castelli's risk index-II (CRI-II)	2.89 ± 0.75	2.52 ± 0.53	0.003

Table 2. Pearson's correlations between BAC and lipid profile and ratios

	r	p
Plasma atherogenic index (PAI)	0.267	0.003
Atherogenic coefficient (AC)	0.282	0.002
Castelli's risk index-I (CRI-I)	0.282	0.002
Castelli's risk index-II (CRI-II)	0.271	0.003
Non-HDLc	0.202	0.027
Total Cholesterol	0.137	0.135
High Density Lipoprotein Cholesterol	-0.223	0.014
Low Density Lipoprotein Cholesterol	0.188	0.039

Hypertension**PP-003**

The role of apelin in patients with resistant hypertension

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Background and Aim: Apelin is a recently discovered peptide which induces vasodilatory effects by acting as an antagonist to the renin-angiotensin-aldosterone system and enhancing nitric oxide release. Based on this, studies conducted in hypertensive patients consistently revealed lower plasma apelin levels even in their prehypertensive stages. However, the association between plasma apelin levels and resistant hypertension remains unknown. Therefore, in our study we investigated the relationship between plasma apelin levels and resistant hypertension.

Methods: A total of 84 patients, 57 of whom with resistant hypertension and 27 healthy controls were prospectively enrolled in the study. Patients with secondary hypertension, diabetes mellitus, heart failure, malignancy, chronic inflammatory diseases, severe renal or liver dysfunction were excluded. Plasma human apelin-12 levels were measured by ELISA method and all patients underwent echocardiographic examination.

Results: There was no statistical difference in plasma apelin levels of patients with resistant hypertension compared to controls (0.51 ± 0.40 ng/ml, 0.40 ± 0.15 ng/ml respectively, $p=0.08$). Resistant hypertensive patients had higher fasting plasma glucose levels (99.3 ± 14.7 mg/dl vs 89.6 ± 8.1 mg/dl, $p=0.002$).

Conclusions: There was no association between plasma apelin levels and resistant hypertension which represent a clinical pathology that incorporates multiple factors. More research is needed in this area.

Table 1. Baseline characteristics and the laboratory values of the study patients

	Resistant hypertension (n=57)	Control (n=27)	P value
Age	56±10	52±9	0,09
Gender (female)	33 (60%)	20 (74%)	0.15
Height (cm)	165±8	164±10	0.90
Weight (kg)	81±15	74±13	0.03
Body mass index (kg/m ²)	30±5	27±4	0,01
LVH	21 (37%)	1 (3%)	<0.01
Serum glucose (mg/dL)	99±14	89±8	<0.01
eGFR (ml/min)	123±45	129±40	0.55
Total cholesterol (mg/dl)	198±47	190±40	0.50
LDL-cholesterol (mg/dl)	124±39	114±37	0.29
HDL-cholesterol (mg/dl)	47±12	49±17	0.49
Triglyceride (mg/dl)	133±66	117±41	0.29
Plasma Apelin (ng/ml)	0.40±0.15	0.51±0.40	0.08

LVH: left ventricular hypertrophy, eGFR: estimated glomerular filtration rate, LDL: low density lipoprotein, HDL: high density lipoprotein

Hypertension**PP-004**

Clinical effect of non-dipper and dipper hypertension with acute coronary syndrome patients

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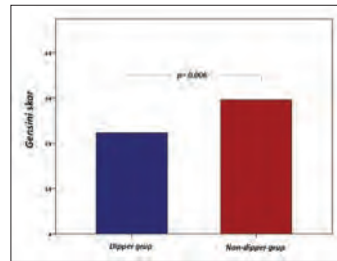
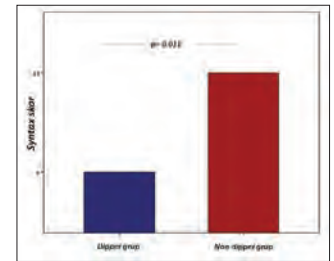
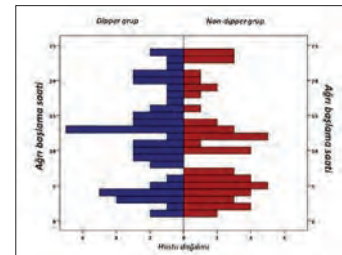
Background and Aim: Coronary artery disease is a health problem in developed countries and developing countries recently. Cardiovascular parameters such as blood pressure, heart rate and coronary tons

change during the day with circadian rhythm. Because blood pressure does not show %10-20 decrease during sleep, target organ damage risk of non-dipper hypertension is related with left ventricle hypertrophy, congestive heart failure, myocardial infarction, stroke and liver damage (albuminuria and end-stage renal failure). In this study, we analyzed the effect of non-dipper and dipper hypertension on prevalence of coronary artery disease, time of symptom onset, contrast nephropathy and in-hospital MACE in patients with acute coronary syndrome.

Methods: We included 107 patients who were diagnosed as acute coronary syndrome with coronary angiography and had angina pectoris lasting 12 hours at most and no history of CAG in this study. Patients were monitored for 24-hour ambulatory blood pressure. Patients were stratified as non-dipper and dipper according to decrease of blood pressure during night. We compared both groups for prevalence of coronary artery disease, time of symptom onset in-hospital MACE.

Results: We included 52 patients in non-dipper group and 55 patients in dipper group in this study. We didn't find statistically significant difference between basic characteristics of patients. When we compared both groups for Syntax and Gensini scores, Syntax score was 10.2 ± 4.8 in average in non-dipper group while it was 8.0 ± 4.2 in average in dipper group and found statistically significant ($p=0.011$). Gensini score was 29.5 ± 13.3 in average in non-dipper group and 22.2 ± 13.2 in dipper group and it was found statistically significant. ($p=0.006$) In terms of symptom onset hours, 32 patients admitted with night angina pectoris in non-dipper group (62%) while 19 patients admitted with night angina pectoris in dipper group (35%) and it was found statistically significant ($p=0.007$). In terms of in-hospital MACE ratios, we identified MACE in 6 patients in non-dipper group and 3 patients in dipper group. This difference was not statistically significant. ($p=0.223$).

Conclusions: In our study, one can conclude that non-dipper hypertension increases number of lesions, MI cases at night and MACE ratios in coronary artery disease by causing endothelium dysfunction and stimulating thrombocyte activation.

**Figure 1.** Evaluation of gensini risk score among patient groups.**Figure 2.** Evaluation of syntax risk score among patient groups.**Figure 3.** Patients' pain start time distribution.**Congenital heart disease****PP-005**

A different indication for closing atrial septal defect: Endothelial dysfunctions?

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Background and Aim: The development of pulmonary hypertension (PAH) secondary to atrial septal defect (ASD) impairment of endothelial structure so that increase in vasoconstriction mediators such as endothelin and decrease vasodilatory mediators. Histologic abnormalities of endothelial structure consist of endothelial dysfunction. This change in the blood vessels and increase the secretion of mediators of pulmonary vessels and its undergo systemic vessels changes flow mediated vasodilatation. Our aim in this study was to evaluate the endothelial dysfunction after ASD closure via flow mediated vasodilatation (FMD).

Methods: 43 patients with pre and one month after post treatment secundum-type ASD and 40 healthy volunteer were prospectively enrolled. ASD was treated with transcatheter closure procedure. FMD was measured to evaluate endothelial function prior to and one month after the closure procedure.

Results: FMD values were significantly higher in the patients with ASD than in the healthy subjects (11.2 ± 1.01 m/s vs. 12.7 ± 1.18 m/s, $p<0.001$). Systolic pulmonary arterial pressure, RVD and FMD values were significantly reduced at the follow-up one month after the procedure compared to baseline. And there was a significant negative correlation between systolic PAP values and FMD ($r=-0.347$; $p=0.013$) in pretreatment group.

Conclusions: It was observed that following closure of the ASD by trans-catheter route, the FMD values were significantly reduced in the right cardiac chambers and the systolic pulmonary arterial pressure was improved. This result has shown us that ASD closure may benefit from endothelial dysfunction.

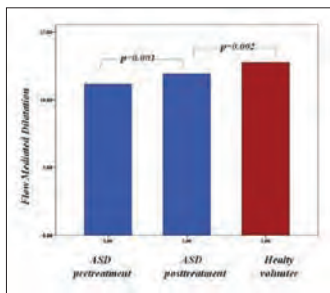


Figure 1. FMD distribution of groups.

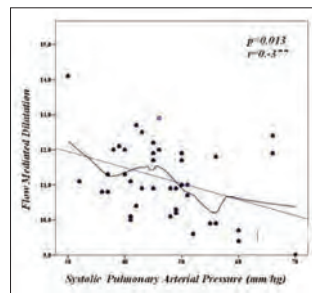


Figure 2. Relation to systolic pulmonary pressure FMD.

Hypertension

PP-006

Hypertensive retinopathy may confer better outcome prediction than estimated as it well correlates with aortic stiffness parameters

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Background and Aim: Investigation of end organ damage in hypertension maintains its importance since it structures the treatment regimen. Although some authors conclude that objectivity of this finding is questionable, hypertensive retinopathy is still mentioned in the state of art guidelines. Aortic stiffness is a well established predictor of cardiovascular morbidity and mortality in hypertensive patients. The aim of this study is to investigate the association between hypertensive retinopathy and aortic stiffness.

Methods: A total of 110 newly diagnosed hypertensive patients were recruited in the study. The patients were divided into Retinopathy Positive and Retinopathy Negative groups considering the presence of retinopathy. The Retinopathy Positive group was then subdivided into Grade 1 and Grade 2 Retinopathy groups. Aortic stiffness parameters were compared between groups.

Results: Comparison of clinical characteristics is given in Table 1. The patients in the Retinopathy Positive group had increased arterial stiffness as represented by the significantly higher values of pulse wave velocity (PWV) when compared to Retinopathy Negative Group. In optical coherence tomographic (OCT) evaluation, foveal thickness and macular volume were significantly higher and submacular chorioidal thickness was significantly lower in Retinopathy Positive group. None of the patients had Grade 3 retinopathy. The severity of arterial stiffness increased with increasing grade of hypertensive retinopathy (Table 2). The patients in the Retinopathy Negative group had significantly lower values of PWV when compared to Grade 1 Retinopathy and Grade 2 Retinopathy groups. Moreover binary analysis have revealed that the patients in the Grade 2 Retinopathy group had significantly higher values of PWV when compared to Grade 1 Retinopathy group. On the other hand while augmentation index (AIx) also increased with increasing grade of retinopathy, the difference could not reach a statistical significance. The correlation analysis have revealed that there was a weak but statistically significant positive correlation between AIx and foveal thickness as well as macular volume. The negative correlation between AIx and submacular chorioidal thickness was also statistically significant. Similar correlation pattern was also observed between PWV and foveal thickness, macular volume and submacular chorioidal thickness. Foveal thickness and macular volume increased with increasing values of PWV. The negative correlation between PWV and submacular chorioidal thickness was again statistically significant.

Conclusions: Although blood pressure values were similar in all patients, the subjects with higher grades of hypertensive retinopathy had higher values of aortic stiffness parameters. Moreover, beside retinal findings, presence of a good correlation between macular findings in OCT and aortic stiffness parameters strengthens the hypothesis that ophthalmologic findings can still be a surrogate marker of cardiovascular outcomes.

Table 1. Comparison of demographical and clinical characteristics in retinopathy positive and retinopathy negative groups

	Retinopathy Negative (n=9)	Retinopathy Positive (n=101)	p=
Age (years)	56.77±8.89	54.67±7.84	p=0.494
Foveal thickness (µm)	210.0 [200.0-234.0]	225.0 [198.0-241.0]	p=0.001
Submacular chorioidal thickness (µm)	247.11±5.60	225.43±10.96	p=0.000
Macular volume (µm)	8.0 [7.9-8.2]	8.5 [7.8-9.3]	p=0.000
Peripheral systolic blood pressure (mmHg)	141.00±18.01	145.55±19.93	p=0.510
Peripheral diastolic blood pressure (mmHg)	91.89±13.40	89.89±13.80	p=0.678
Central systolic blood pressure (mmHg)	128.78±13.74	135.57±19.40	p=0.307
Central diastolic blood pressure (mmHg)	93.78±13.29	91.61±13.69	p=0.650
Augmentation indeks	19.0 [7.0-40.0]	32.0 [3.0-59.0]	p=0.077
Pulse wave velocity	6.73±0.72	8.78±1.44	p=0.000
Heart rate (bpm)	77.0 [66.0-100.0]	71.0 [54.0-114.0]	p=0.082
Aortic root diameter (cm)	3.1 [2.9-3.6]	3.1 [2.4-3.9]	p=0.826
Ascending aorta diameter (cm)	3.0 [2.8-3.4]	3.2 [2.3-4.7]	p=0.103
Septum thickness (cm)	1.1 [1.0-1.4]	1.1 [0.8-1.6]	p=0.333
Posterior wall thickness (cm)	1.0 [0.9-1.4]	1.0 [0.7-1.5]	p=0.723
End diastolic diameter (cm)	4.88±0.41	4.64±0.46	p=0.146
End systolic diameter (cm)	2.9 [1.8-3.0]	2.9 [2.0-4.8]	p=0.443
Ejection fraction (%)	65.0 [60.0-68.0]	65.0 [50.0-71.0]	p=0.538
Aortic velocity (cm/s)	1.1 [1.0-1.5]	1.3 [0.8-2.0]	p=0.054
Pulmonary velocity (cm/s)	0.8 [0.7-1.1]	0.8 [0.5-1.4]	p=0.722

Table 2. Comparison of arterial stiffness parameters between Retinopathy Negative, Grade 1 Retinopathy and Grade 2 Retinopathy groups

	Retinopathy Negative (0) (n=9)	Retinopathy Grade 1 (1) (n=71)	Retinopathy Grade 2 (2) (n=30)	p=
Augmentation indeks	21.67±10.95	28.86±14.51	32.60±10.60	p=0.092
Pulse wave velocity	6.73±0.72	8.41±1.25	9.65±1.50	p=0.000 [0-1,2] [1-2]*

* [0-1,2] means significant difference when Group 0 is compared with Group 1 and Group 2 [1-2] means significant difference when Group 1 is compared with Group 2.

Table 3. The correlation between ophthalmologic measurements and aortic stiffness parameters

	Augmentation indeks r-p	Pulse Wave Velocity r-p
Foveal thickness	0.318-0.001	0.454-0.000
Submacular chorioidal thickness	-0.280-0.003	-0.576-0.000
Macular volume	0.315-0.001	0.433-0.000

Hypertension

PP-007

Is preeclampsia associated with increased elastic arteries stiffness?

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Background and Aim: An altered endothelial function (EF) could associate with preeclampsia (PE). But, first analyses are needed to approve this topic. Flow-mediated dilation (FMD) measurements offer significant information about EF. The aim of this present study is to evaluate, in healthy pregnant and pregnant with PE, central arterial parameters association with EF

Methods: Nonhypertensive (HP) and pregnant with preeclampsia (PE) included. Aortic blood pressure (BP), augmentation index adjusted to a heart rate of 75 bpm (AIx@75), aortic pulse wave velocity (PWVcf), and carotid stiffness were measured.

Results: Baseline peripheral SBP, DBP, and MBP levels were significantly higher than in PE (<0.001) Aortic BP and AIx@75 were increased in the sick group. PE demonstrated stricter elastic but not muscular arteries.

Conclusions: Aortic BP, wave reflections, and elastic arteries stiffness are increased in PE. We demonstrated endothelial dysfunctions in PE patients.

Table 1. Comparison of demographical, laboratory, and study parameters between two groups in the study population

	Healthy pregnant (n=20)	Preeclampsia (n=20)	P value
Age (years)	28.3 ± 6.1	32.5 ± 5.8	0.370
Gestational age (weeks)	33.6 ± 3.6	35.3 ± 2.1	0.500
Number of gestations (n)	1.7 ± 2.1	3.4 ± 4.5	0.280
Body mass index (kg/m2)	26.1 ± 3.6	37.1 ± 6.6	0.001
Creatinine (mg/dL)	0.65 ± 0.1	0.55 ± 0.1	0.600
24-hour proteinuria (g)	0.0 ± 0.1	0.48 ± 0.1	<0.001
Uric acid (mg/dL)	3.1 ± 1.1	6.6 ± 2.5	0.001
SGOT (U/L)	11.2 ± 3.2	13.5 ± 2.5	0.701
SGPT (U/L)	10.5 ± 4.2	10.8 ± 3.1	0.570
Hematocrit (%)	30.7 ± 3.7	32.7 ± 2.0	0.604
Heart rate (bpm)	78.2 ± 13.6	81.3 ± 12.0	0.832
Peripheral SBP (mmHg)	110.8 ± 8.2	147.5 ± 7.5	<0.001
Peripheral DBP (mmHg)	60.5 ± 9.5	85.6 ± 8.8	<0.001
Mean BP (mmHg)	75.6 ± 5.8	105.9 ± 7.3	<0.001
Peripheral pulse pressure (mmHg)	50.3 ± 18.6	58.0 ± 12.7	0.565
Central SBP (mmHg)	95.7 ± 6.7	130.3 ± 5.8	<0.001
Central DBP (mmHg)	60.5 ± 9.5	85.4 ± 10.0	<0.001
Central pulse pressure (mmHg)	35.2 ± 11.4	42.6 ± 10.8	0.035
Amplification ratio	1.3 ± 0.2	1.5 ± 0.2	<0.001
Augmentation index*	12.2 ± 12.0	20.3 ± 5.7	0.016
Pulse wave velocity (m/s)	4.6 ± 0.8	9.2 ± 1.2	<0.001

* Augmentation index adjusted to a heart rate of 75 bpm. SBP: systolic blood pressure; DBP: diastolic blood pressure

Hypertension

PP-008

Relation between arylesterase activity and pulse pressure index in patients with a acute ischemic stroke

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Background and Aim: Stroke is a multifactorial disease arising from genetic and environmental risk factors or their interaction. The underlying cause of the majority of ischemic strokes is an atherosclerotic plaque in the carotid arteries. Oxidative stress and blood pressure were found to be closely related with cardiovascular and cerebrovascular diseases. Paraoxonase-1 (PON1) has been considered as an anti-atherosclerosis factor, because it can reduce the oxidative modification of lipoproteins and macrophage foam cell formation occurring in the early phase of atherosclerosis. Therefore, it is easily inferred that the changes in serum PON1 concentrations or activity can affect vascular disease status. Arylesterase (ARE) is considered as the main protein indicator which is not affected by changes in PON1. Elevated pulse pressure (PP) may lead to an increased risk of cardiovascular and cerebrovascular morbidity and mortality. However, there are limitations for PP as an evaluation index. In order to overcome the defects of PP, there is a novel parameter, "pulse pressure/systolic pressure" called "pulse pressure index (PPI)" for assessment of cardiovascular outcomes. We investigated the association between Arylesterase (ARE) activities and pulse pressure index (PPI) in acute ischemic stroke patients.

Methods: We evaluated and compared the ARE activity and PP in 46 ischemic stroke patients and 32 control patients. They were classified into 2 groups: Ischemic stroke patients (Group 1, n=46), control patients (Group 2, n=32). Blood pressure measurements were performed in all patients within 10 minutes admitted to emergency care unit. The PP was calculated by subtraction of diastolic blood pressure (DBP) from systolic blood pressure (SBP). PPI was calculated " pulse pressure / systolic pressure". Independent sample t test and Chi-square analyses were used to compare differences between groups.

Results: Hypertension, age, diabetes mellitus, dyslipidemia significantly higher in ischemic stroke patients than control group (Table 1) (p<0.05). PPI was significantly higher in ischemic stroke patients than control group (0.471±0.0726 and 0.423±0.0485, p<0.05). ARE activity was significantly lower in ischemic stroke patients than in control group (521.88±72.49 and 577.99±87.51, p=0.013).

Conclusions: Our results suggested that, ARE activity and PPI are important risk factors in acute ischemic stroke patients.

Table 1. Clinical characteristics of patients

Variables	Group-1 (Ischemic Stroke) n=46	Group-2 (Control Group) n=32	p Value
Age (year)	68.57 ± 13.49	44.16 ± 18.67	0.003
Gender (F/M)	27 / 19	17 / 15	0.234
Hypertension	29 (65%)	6 (18%)	0.005
Diabetes Mellitus	14 (30%)	2 (6%)	0.007
Smoking	7 (16%)	3 (10%)	0.583
Dyslipidemia	11 (24%)	0 (0%)	0.004

Hypertension

PP-009

The relationship between hypertension knowledge level and treatment compliance in patients with diagnosis of hypertension and affecting factors

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Background and Aim: This study aimed to determine the relationship between hypertension knowledge level and treatment compliance in patients with diagnosis of hypertension and affecting factors.

Methods: Data were collected from 234 patients admitted to cardiology clinic of an university hospital, who were diagnosed with hypertension at least 6 months ago and who were taking antihypertensive drugs. A questionnaire form including socio-demographic and disease-related characteristics of patients, Hypertension Knowledge - Level Scale (HK-LS) and Hill-Bone Hypertension Treatment Compliance Scale (HBHTCS) were used to collect data. Statistical analysis was performed using with percentages, averages, Mann-Whitney U test, Kruskal Wallis test and Spearman correlation analysis in SPSS 20.0 program.

Results: The average age of the patients was 58.2±10.79 (years), 53% were male, 86.3% were married and 58.5% were in primary and lower education. Mean hypertension duration of patients was 9.13±6.67 years. Of the patients, 76.9% had a family history of hypertension, and 82.2% had an additional chronic disease. Of the patients 69.7% had followed blood pressure at home. It was determined that 66.2% of the patients were not informed about hypertension. Average score on HK-LS of patients was 14.24±6, and average score on HBHTCS was 8.15±5. HK-LS score of female patients was significantly higher than male patients (p=0.016) and HBHTCS score of male patients was significantly higher than female patients (p=0.004). HK-LS score was higher in patients who were informed about hypertension than not informed. HBHTCS score was lower in patients informed about hypertension than not informed (p=0.000). Patients with additional chronic disease were found to have low levels of compliance with hypertension treatment (p=0.034). There was a significant negative correlation between the HK-LS scores and HBHTCS scores of the patients (p=0.000).

Conclusions: In the study, it was found that the patient's knowledge level of hypertension was slightly higher than the average. Patients with low levels of knowledge were found to have low treatment compliance. The level of knowledge and treatment compliance of the patients who were informed about hypertension during the treatment period was high, and the compliance of patients with additional chronic diseases was low. It is important to improve treatment compliance for the success of treatment in chronic diseases such as hypertension. Patient education should be emphasized to improve compliance.

Epidemiology

PP-011

Risk factors for early coronary artery disease in the region of the lakes

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Background and Aim: Coronary Artery diseases (CAD) are often present in old populations and rare in young people. CAD incidence significantly increased recent years. Control of cardiovascular risk factors is of particular importance for the prevention of cardiovascular diseases. It is possible to stop progression or prevent these diseases by elimination or modification of modifiable risk factors in the light of treatment goals. The aim of this study was to analyze clinical risk factors of CAD in young patients.

Methods: Medical recordings of 20907 patients who underwent coronary angiography were screened for this study between January 2002 and March 2017. Patients older than 45 years old and severe valvular disease were excluded study. A total 862 patients included study. Major risk factors for atherosclerotic cardiovascular disease were identified. Angiographic characteristics and management were reviewed.

Results: Out of total 862 patients studied, 595 had coronary artery disease (CAD) (306 had ST segment elevation myocardial infarction, 182 had non-ST segment elevation ACS, 107 had stable angina pectoris) and 267 had normal coronary arteries (NCA). The young CAD group had a significantly higher proportion of males (94.1 vs. 58.8%; p<0.01), smokers (78 vs. 45%; p<0.01) than age-matched control patients with NCA (Table 1). In patients with CAD, the rate of obesity was higher in female group than males with CAD (41 vs. 14%; p<0.01) while the smoking rate was higher than female group with CAD (80 vs. 44%; p<0.01) (Table 2). When I investigated only men or women, the smoking rate was higher in CAD group (68 vs 80%; p=0.001 and 14 vs 24%; p=0.001) (Table 3). Univariable Logistic regression analysis indicated that male sex (OR=11, p<0.01), smoking (OR=4.37, p<0.01) are associated with CAD in young patients. Similarly, when we put these variables into multivariable logistic regression analysis male sex (OR=7.3, p<0.01) and smoking (OR=2.3, p<0.01) were found to be independent predictors of CAD (Table 4). When univariate analysis was done among genders, the rate of CAD was 1.9-fold higher in male gender and 4.5 -fold higher in female gender (OR=1.955, p<0.01 vs OR=4.5, p<0.01) (Table 5).

Conclusions: The early CAD at young ages can be largely explained by higher smoking and male gender of cardiovascular disease in the region of lakes. Primary prevention of smoking should be more aggressively promoted in young adults. Prevention of obesity should be promoted especially female gender.

Table 1. Baseline demographic parameters of premature coronary artery disease

	CAD(-)	CAD(+)	P value
LV ejection fraction (%)	58±10	50±11	>0.01
Age (years)	39±6.4	39±8.4	0.537
Diabetes Mellitus (%)	12	15	0.169
Hypertension (%)	21	18	0.218
Hyperlipidemia (%)	21	21	0.470
Smoking (%)	45	78	>0.01
Obesity (%)	22	16	0.018
Family history (%)	33	30	0.209
Male gender (%)	58	94	>0.01

CAD: coronary artery disease

Table 2. Baseline demographic parameters of patients with coronary artery disease

	CAD (+)	CAD (+)	p value
	Male (n=539)	Female (n=34)	
Age (years)	39 ±8.3	37±10	0.436
Diabetes Mellitus (%)	14	23	0.213
Hypertension (%)	17	35	0.085
Hyperlipidemia (%)	21	32	0.097
Smoking (%)	80	44	>0.01
Obesity (%)	14	41	>0.01
Family history (%)	31	26	0.366

CAD: coronary artery disease

Table 3. Atherosclerotic risk factors among genders

	Male Gender			Female Gender		
	CAD (-)	CAD (+)	P value	CAD (-)	CAD (+)	P value
Diabetes Mellitus (%)	9	14	0.04	17	24	0.254
Hypertension (%)	12	17	0.08	33	33	0.566
Smoking (%)	68	80	>0.01	14	24	>0.01
Obesity (%)	16	14	0.354	31	39	0.254
Family history (%)	26	30	0.145	44	27	0.07
Hyperlipidemia (%)	19	21	0.323	24	30	0.298

Table 4. Predictors of coronary artery disease in Univariate and Multivariate Logistic Regression Analysis

	Univariate		Multivariate	
	OR (95 % CI)	P value	OR (95 % CI)	P value
Smoking	4.373 (3.2-5.9)	>0.01	2.30 (1.6-3.2)	>0.01
Male gender	11 (7.3-16.8)	>0.01	7.3 (4.6-11.4)	>0.01
Obesity	1.484 (1.04-2.11)	>0.01		

Table 5. Univariate Regression Analysis of smoking among genders

	Univariate analysis			
	Male gender		Female gender	
	OR (95 % CI)	P value	OR (95 % CI)	P value
Smoking	1.955 (1.3-2.8)	>0.01	4.5 (1.9-10.6)	>0.01

Hypertension

PP-012

Frontal QRS-T angle may predict non-dipper status in patients with hypertension

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Background and Aim: Hypertension is a common disease throughout the world and an important risk factor for cardiovascular diseases. Blood pressure (BP) has a circadian variation, but some hypertensive patients do not demonstrate this circadian variation and their BP reduction is less than 10%; they have been termed as non-dippers. Studies showed that patients with non-dipper hypertension had a higher cardiovascular risk compared to patients with dipper hypertension. Frontal QRS-T angle, defined as the angle between the directions of ventricular depolarization (QRS wave) and repolarization (T wave), has been described as a novel marker of ventricular repolarization. To the best of our knowledge, there is no study investigating the association of frontal QRS-T angle with non-dipper hypertension. The aim of our study is to investigate the association between frontal QRS-T angle and non-dipper status in hypertensive patients without left ventricular hypertrophy (LVH).

Methods: A total of 122 hypertensive patients without LVH were included in this study. The diagnosis of non-dipper status were based on 24-h ambulatory blood pressure monitoring (ABPM) records. 12-lead ECG was performed on all patients in the supine position. Frontal QRS and T wave axes were available in the reports of the ECG machine. QRS and T axes were checked from these reports, and the frontal QRS-T angle was calculated as the absolute value of the difference between QRS and T wave axes (QRS axis-T axis).

Results: According to 24-h ABPM records, 66 (54.1%) patients had dipper hypertension, while 56 (45.9%) patients had non-dipper hypertension. When patients were divided into two groups according to 24-h ABPM, basal demographic characteristics and antihypertensive treatments were not significantly different between the two groups. However, QT dispersion (QTd) (22.5±5.9 vs 20.5±4.6, p=0.040) and frontal QRS-T angle (47.9°±29.7° vs 26.7°±19.6°, p<0.001) were significantly higher in non-dipper hypertensive patients than in dipper hypertensive patients. In correlation analysis, a weak positive correlation was found between frontal QRS-T angle and sleeping systolic BP (r=0.211, p=0.020), and diastolic BPs (r=0.199, p=0.028). However, there was no significant correlation between QT intervals and BPs. Multivariate logistic regression analysis showed that frontal QRS-T angle (odds ratio 1.037, 95% confidence interval 1.019-1.056, p<0.001) was the only independent predictor of non-dipper status.

Conclusions: Our study has demonstrated an important relationship between frontal QRS-T angle and non-dipper hypertension. We found that frontal QRS-T angle was independent predictor of non-dipper status in hypertensive patients even before the development of LVH. In addition; although they were weak, frontal QRS-T angle was positively correlated with sleep systolic and diastolic BPs. Therefore, an increased frontal QRS-T angle can be used as a simple ECG marker to detect non-dipper status in hypertensive patients without LVH.

Table 1. Electrocardiographic variables of dipper and non-dipper hypertensive patients

	Dipper	Non-dipper	P
	(n = 66)	(n = 56)	
Heart rate (beats/min.)	76.6 ± 10.0	74.2 ± 9.4	0.172
QRS duration (ms)	96.3 ± 6.5	94.6 ± 5.9	0.138
QT dispersion (ms)	20.5 ± 4.6	22.5 ± 5.9	0.040
Corrected QT dispersion (ms)	23.1 ± 5.6	25.0 ± 6.8	0.099
Frontal QRS-T angle (°)	26.7 ± 19.6	47.9 ± 29.7	<0.001

Data are expressed as mean ± SD.

Values in bold indicate statistical significance (p < 0.05).

Table 2. Correlation analysis among electrocardiographic parameters, ambulatory blood pressure recordings, and LVMI

Parameters	QTd		cQTd		QRS-T angle	
	r	p	r	p	r	p
Systolic BP (total)	-0.034	0.712	-0.039	0.673	0.064	0.483
Diastolic BP (total)	-0.088	0.333	-0.106	0.246	0.081	0.378
Systolic BP (awake)	-0.058	0.526	-0.057	0.530	0.054	0.557
Diastolic BP (awake)	-0.097	0.290	-0.113	0.217	0.008	0.928
Systolic BP (sleep)	0.071	0.438	0.058	0.526	0.211	0.020
Diastolic BP (sleep)	0.033	0.722	0.017	0.850	0.199	0.028
LVMI	-0.012	0.894	-0.020	0.824	0.008	0.930

Data are expressed as mean ± SD.

BP: blood pressure; cQTd: corrected QT dispersion; LVMI: left ventricular mass index; QTd: QT dispersion.

Values in bold indicate statistical significance (p < 0.05).

Table 3. Univariate and multivariate logistic regression analysis showing independent predictors of non-dipper status

	Univariate			Multivariate		
	OR	95% CI	P	OR	95% CI	P
Age	1.031	0.997-1.067	0.077	1.036	0.997-1.076	0.068
Sex, males	1.143	0.552-2.368	0.719			
BMI	0.997	0.929-1.070	0.935			
Smoking	1.172	0.541-2.540	0.688			
Heart rate	0.974	0.939-1.011	0.173			
QRS duration	0.957	0.903-1.014	0.139			
QTd	1.078	1.004-1.158	0.039	1.080	0.999-1.168	0.054
cQTd	1.051	0.990-1.115	0.102			
QRS-T angle	1.036	1.018-1.054	<0.001	1.037	1.019-1.056	<0.001
LVEF	0.936	0.829-1.058	0.289			
LVMI	1.007	0.980-1.034	0.629			

BMI: body mass index; CI: confidence interval; cQTd: corrected QT dispersion; LVEF: left ventricular ejection fraction; LVMI: left ventricular mass index; OR: odds ratio; QTd: QT dispersion.

Values in bold indicate statistical significance (p < 0.05).

Hypertension

PP-013

The effect of fixed dose triple antihypertensive combination to parameters of ambulatory blood pressure monitoring

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Background and Aim: The "nondipper" state is more important predictor of cardiovascular outcomes than daytime blood pressure values. Re-establishing of "dipping" physiology by taking of one drug at night time except diuretics; significantly reduces total cardiovascular events. The aim of our study was to investigate whether there is any difference in the effect of distinct usage time of fixed dose triple antihypertensive combination, which has a diuretic component, on night time blood pressure fluctuations.

Methods: 68 patients (63.1±10.7 years, 27 male) presenting with grade-II hypertension (defined as SBP >160 mmHg and/or DBP >100 mmHg) were consecutively included in the study. The study population was evaluated for the presence of hyperlipidemia, diabetes mellitus, stroke, renal dysfunction, coronary artery disease and smoking status. All patients underwent a 24-hour ambulatory blood pressure monitoring for evaluation of mean blood pressure values and dipper or non-dipper status. The patients having reverse dipping or extreme dipping were included in the nondipper group due to the small number of patients.

Results: Two study groups each with 34 patients; containing equal numbers of patients taking ACEI and ARB. The characteristics of patients are listed in Table 1. Groups were very similar in terms of demographic data. There was no difference on average, night-time and day-time systolic and diastolic blood pressure values between groups (Table 2). Although there were numerical differences on the dipping pattern in evening pill group, this couldn't reach statistical significance.

Conclusions: Our study suggests that taking of fixed dose triple antihypertensive drug combination in the morning or evening time has same effect on ambulatory blood pressure monitoring parameters including dipping pattern.

Table 1. The general characteristics of the patients according to fixed dose drug combination taking time

	Morning pill group (n= 34)	Evening pill group (n= 34)	p
Age (years)	62.4 ± 12.3	63.9 ± 9	0.950
Male sex (n – %)	15 (44.1%)	12 (35.2%)	0.457
Diabetes (n – %)	17 (50%)	27 (79.4%)	0.650
Hyperlipidemia (n – %)	10 (29.4%)	5 (14.7%)	0.162
Chronic kidney failure (n – %)	2 (5.88%)	1 (2.94%)	0.573
Coronary artery disease (n – %)	10 (29.4%)	4 (11.76%)	0.082
Cerebrovascular disease (n – %)	1 (2.94%)	2 (5.88%)	0.573
Body mass index (kg/m2)	28.7 ± 7.89	29.5 ± 6.85	0.404
Smoking (n – %)	10 (29.4%)	4 (11.76%)	0.082
ACEI	17(50%)	17(50%)	

Table 2. Ambulatory blood pressure monitoring parameters of the patients

	Morning pill group (n= 34)	Evening pill group (n= 34)	p
Average systolic blood pressure over 24 hours (mmHg)	122 ± 12	124 ± 16	0.207
Average diastolic blood pressure over 24 hours (mmHg)	74 ± 8	75 ± 10	0.121
Day-time average systolic blood pressure (mmHg)	122 ± 11	126 ± 15	0.180
Day-time average diastolic blood pressure (mmHg)	75 ± 8	77 ± 10	0.507
Night-time average systolic blood pressure (mmHg)	118 ± 14	117 ± 18	0.175
Night-time average diastolic blood pressure (mmHg)	70 ± 9	69 ± 12	0.492
Dipper status - sistol (n – %)	6 (%17.6)	10 (%29.4)	0.253
Dipper status- diastol (n – %)	10 (%29.4)	17 (%50)	0.083

Hypertension

PP-014

Is endocan a biochemical marker for asymptomatic target organ damage in hypertensive patients?

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Background and Aim: Hypertension (HT) is a serious public health problem that affects almost all organs in the body and causes notable morbidity and mortality. When HT and cardiovascular (CV) disease on two ends of a spectrum, asymptomatic target organ damage (AOD) may actually exhibit an intermediate stage between. It is thought that endothelial (ET) dysfunction might have a role in pathogenesis of HT and serve as a link between HT and AOD. Endocan (EDC), is a soluble proteoglycan, secreted by human vascular ET cells and endothelium-dependent pathological conditions like inflammation, cancer, infections and atherosclerosis stimulates its secretion. Evidence is accumulating that EDC may be a potential ET cell marker representing immuno-inflammatory activation of endothelium. Elevated levels of serum endocan have constantly been shown in hypertensive (HTS) patients with symptomatic CV or renal disease (RD). There is paucity of data on EDC levels in asymptomatic vascular states of HT. Therefore, we aimed to investigate EDC levels in HTS patients without symptomatic CV or RD. Moreover, we explored the relationship between EDC levels and the presence and intensity of AOD in HTS patients.

Methods: This open-label study was performed at Sakarya University Hospital Cardiology Clinic between June 2016 and October 2017. The study is in accordance with the Declaration of Helsinki and study protocol was ratified by the local Ethics Committee. Informed consent was obtained from all patients (PTS). PTS diagnosed as having systemic HT were prospectively enrolled to study according to inclusion (INC) and exclusion (EXC) criteria. INC were; presence of systemic HT in a subject >18 years of age. EXC were: PTS with secondary HT, diabetes mellitus, chronic RD of stage ≥3, symptomatic CV disease (i.e. documented coronary and/or peripheral arterial and cerebrovascular disease, cardiac failure, valvulopathies). PTS with suggestive symptoms or signs of CV disease were also excluded. 153 eligible PTS met the criteria and were consecutively enrolled to the study.

Results: Majority of PTS were women (95 female, 58 male) and mean age was 50±9 years. 25 PTS (16%) had Stage 1, 89 PTS (58%) had Stage 2 and 39 PTS (26%) had Stage 3 HT. 132 PTS were receiving an antiHTS medication (86%). Depending on presence or absence of a specific AOD, serum EDC levels didn't show any difference. Serum EDC level didn't significantly correlate with any of the variable such as pulse pressure, CIMT, CAVI, ABI, LVMI, Sokolow-Lyon index, Cornell voltage duration product and eGFR.

Conclusions: In our study, serum EDC levels didn't differ between asymptomatic HTS patients with or without AOD. Serum EDC levels didn't correlate with any of the AOD variables neither. We found that EDC is not a proxy biochemical marker for the presence of AOD in HTS patients and its usage for this purpose is futile. As per our knowledge, this is the first study to examine the relationship of EDC with any specific AOD in HTS patients without symptomatic CV and RD.

Table 1. Frequency of patients with different AOD markers

	Asymptomatic Target Organ Damage	N	%
1	Pulse pressure ≥60 mmHg	48	31.3
2	Electrocardiographic LVH (Sokolow-Lyon index >3.5 mV; RaVL >1.1 mV; Cornell voltage duration product >244 mV*ms) or	68	44.4
	Echocardiographic LVH [LVM index: men >115 g/m ² ; women >95 g/m ² (BSA)]		
		74	48.3
3	Carotid wall thickening (CIMT >0.9 mm) or plaque	38	24.8
4	Ankle-brachial index <0.9	5	3.2
5	Cardio-ankle vascular index (CAVI) >8	58	37.9
6	CKD with eGFR 30–60 mL/min/1.73 m ²	0	0

AOD, asymptomatic target organ damage; BSA, body surface area; CAVI, Cardio-ankle vascular index; CIMT, carotid intima-media thickness; CKD, chronic kidney disease; LVH, left ventricular hypertrophy; LVM, left ventricular mass.

Table 2. Frequency of patients according to asymptomatic target organ damage load

Asymptomatic Target Organ Damage Load	N	%
0	35	22.9
1	49	32
2	37	24.2
3	28	18.3
4	4	2.6
5	0	0
6	0	0
Total	153	100

Table 3.

Asymptomatic Target Organ Damage Parameters	Endocan level*		p value
	(+)	(-)	
Pulse Pressure > 60 mmHg	3.4/4.0/5.2	3.4/3.9/4.4	0.31
Electrocardiographic LVH (Sokolow-Lyon index >3.5 mV; RaVL >1.1 mV; Cornell voltage duration product >244 mV*ms) or Echocardiographic LVH [LVM index: men >115 g/m ² ; women >95 g/m ² (BSA)]	3.3/3.9/4.5	3.5/4.0/4.7	0.32
Carotid wall thickening (CIMT >0.9 mm) or plaque	3.3/3.9/4.6	3.4/3.9/4.7	0.89
Ankle-brachial index <0.9	2.9/3.9/4.4	3.4/3.9/4.7	0.50
Cardio-ankle vascular index (CAVI) >8	3.4/4.0/4.6	3.4/3.9/4.7	0.89
CKD with eGFR 30–60 mL/min/1.73 m ²	-	3.4/3.9/4.7	

*Skew-distributed continuous variables were expressed as median and interquartile ranges. BSA, body surface area; CAVI, Cardio-ankle vascular index; CIMT, carotid intima-media thickness; CKD, chronic kidney disease; LVH, left ventricular hypertrophy; LVM, left ventricular mass.

Pediatric cardiology

PP-015

Stenting procedures in children with congenital heart diseases: A single centre experience and results

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Background and Aim: Stenting procedures are commonly performed for the treatment of Congenital Heart Diseases (CHD). In this study, we retrospectively examined and evaluated the medical records of patients who underwent intravascular stent placement for the treatment of CHD.

Methods: Seventy-six patients who were followed up and underwent intravascular stent placement between January 2011 and January 2016 at the Pediatric Cardiology and Cardiovascular Surgery Departments of Acibadem University were included in this study. Medical records of these patients were analyzed retrospectively: patient demographics, diagnoses, surgical interventions performed, angiographic data, pre and post intervention blood pressures, pressure gradients, vascular diameters, hemodynamic parameters such as oxygen saturations, materials used during interventions, complication rates, echocardiography and ECG findings, follow-up observations.

Results: Seventeen of our patients had stent implantation for the treatment of pulmonary artery stenosis, 14 patients for coarctation of the aorta, one patient for supravalvular aortic stenosis, 12 patients for stenotic right ventricle pulmonary artery conduit, 12 for stenosis of cavopulmonary anastomosis, seven for patent ductus arteriosus, two for systemic venous stenosis and three patients for major aorticopulmonary collateral arteries. Seven patients had stenting of the Blalock-Taussig shunt and one patient had stenting of the interatrial septum. The major complications observed were the following: one patient needed surgical by-pass due to injury to the femoral artery after stenting for coarctation of the aorta, one patient needed a second stent due to restricted dissection occurring after stent dilatation, and one patient had stent retrieval due to migration during stenting for pulmonary conduit stenosis. Two of our patients died during a prolonged post-operative hospital stay due to factors independent of the operations; infection and heart failure. Two of our patients died during the post-discharge follow-up period. No mortality observed which was otherwise related to operation itself.

Conclusions: Intravascular stent placement procedures are an alternative to surgical procedures for the treatment of congenital heart diseases, and they can be performed safely as they are non-invasive and have both lower mortality and morbidity rates.

Epidemiology

PP-016

Evaluation of the relationship between CK-MB levels and non-cardiac disease

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Background and Aim: Malignant Creatine Kinase-MB (CKMB) syndrome is a condition in which the CKMB level measured in the plasma is equal to or greater than the Creatine Kinase (CK) level. Although autoimmune diseases have been associated with malignancies in various publications, the prevalence and clinical significance of this syndrome is unknown. In this study, we investigated the frequency of malignant CKMB and the clinical significance of this condition in patients presenting with chest pain.

Methods: The study was included in the Cardiology polyclinic of Atatürk University between 2012-2016, for patients with cardiac follow-up CK and CKMB who applied with chest pain during hospitalization for any reason.

Results: A total of 5380 patients with a CKMB greater than or equal to CK were included in the study. The mean age of the patients was 62, and 51% of the patients were male. Patients with a CKMB greater than or equal to CK have a higher rate of malignant disease.

Conclusions: The increase in CKMB is usually seen in the course of malignant diseases presence of malignant CKMB syndrome is associated with poor prognosis and the presence of this parameter should be investigated with advanced diagnostic methods in this patients group.

Family medicine

PP-017

Assessment of family medicine residents' knowledge and attitudes about cardiovascular disease risk factors in Ankara

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Background and Aim: Cardiovascular diseases are the most important cause of morbidity and mortality worldwide. A large proportion of cardiovascular diseases can be prevented by reducing behavioral factors such as smoking, unhealthy diet and inadequate physical activity. Unhealthy habits lead to metabolic and physiological changes such as hypertension, obesity, diabetes and dyslipidemia. Primary health care professionals have a great responsibility to identify risky individuals in the prevention of cardiovascular diseases and to lead early diagnosis and treatment, raise awareness in the society and provide healthy lifestyle habits. We aimed to evaluate the knowledge and attitudes of family medicine residents in Ankara on cardiovascular disease risk factors.

Methods: Our research is a descriptive cross-sectional type of research. A questionnaire, originally structured by researchers for the study is administered 195 family medicine residents.

Results: 86.1% of the physicians reported that they're always counselling their patients about medication. Also 66.7%, 65.6% and 63%, of the physicians were counselling about smoking, hypertension and diabetes. 51.8% of the participants were counselling about hyperlipidemia, 64.1% about body mass index, 71.8% about diet and 67.2% about exercise, if there is a risk. Physicians believe in the importance of lifestyle changes for the protection from cardiovascular diseases and inform their patients about this; but did not clearly state the detailed recommendation of specific instructions for safe exercise, determining the appropriate exercise program for the patient by specifying frequency and duration, choosing a specific date for smoking cessation. Physicians did not have enough knowledge about cardiovascular disease risk scoring methods. Of the doctors who participated in the study, 48.2% knew Framingham, 33% knew SCORE and 13.8% knew PROCAM more than 50% of their parameters. While residents and contracted general practitioners were similar to each other in knowing more than 50% of the Framingham risk scoring parameters (p=1), contracted general practitioners (48.8%) were found to be statistically significantly better than assistant physicians (29.2%) knowing more than half of the parameters in SCORE (p=0.025). Most of the physicians who participated in the study thought that their knowledge level about risk scoring methods was insufficient, but there was no significant difference between resident physicians (56.5%) and contracted general practitioners (58.8%) (p=0.438).

Conclusions: In conclusion, physicians have sufficient knowledge about cardiovascular disease risk factors, but do not have enough information for risk scoring methods including these factors in clinical practise.

Hypertension

PP-018

The place of serum cyclophilin-A level for evaluating subclinical target organ damage in hypertensive patients

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Background and Aim: Hypertension (HTN) is a reversible risk factor for cardiovascular diseases and chronic renal failure with high mortality and morbidity. The subclinical target organ damage (STOD) is considered to be a strong indicator of major cardiac events in the future. Cyclophilin-A (CyPA) is a chaperone protein found in the body. Chen-Shu Chang et al. first showed that plasma CyPA level was altered in patients with HTN respond to systolic and diastolic blood pressure changes. Hongyan Su et al. showed the correlation of serum CyPA level with left ventricular hypertrophy (LVH) grade in hypertensive patients. We aimed to investigate

the association of serum CyPA level with known STOD markers in patients with HTN.

Methods: Patients who were admitted to the Istanbul University Cardiology Institute Outpatient Clinics between September 2015 and September 2016, and diagnosed with HTN or were already on antihypertensive treatment and whose EF on echocardiographic (ECHO) examination was above 50% were included in the study. The STOD evaluation of the patients was performed by the detection of LVH using the Sokolov index (S.I.) in electrocardiography (ECG) and the Deveroux formula in ECHO, the detection of microalbuminuria in the urine and the measurement of aortic pulse wave velocity (aPWV). CyPA values was measured from venous serum samples by ELISA method. IBM SPSS statistics 21, Chicago, USA was used for statistical analyses. We compared serum CyPA values in patients with and without STOD.

Results: 125 patients with HTN were prospectively included in the this study. At least one type of STOD was detected on 56 (44.8%) of the patients and STOD was demonstrated in 69 patients (55.2%). The S.I. was above 35 mm by ECG in 9 (7.2%) of 125 patients. LVH was detected in ECHO in 27 patients (21.6%). Aortic PWV was over 10 m/s in 17 patients (13.6%). Microalbuminuria was detected in 16 patients (12.8%) and GFR was found to be between 30-60 mL/min/1.73 m² in 4 patients (3.2%). Serum CyPA levels [10.75 ng/ml (8.64-84.66) vs 11.02 ng/ml (8.54-89.37)] were found to be similar in patients with and without STOD (p=0.6). In our study there was no statistically significant difference in serum CyPA levels [10.65 ng/ml (8.69-84.66) vs 10.95 ng/ml (8.54-89.37)] between patients with and without LVH detected by ECHO (p=0.8). Serum CyPA levels were statistically similar between patients with aPWV >10 m/sec. and ≤10 m/sec. [10.80 ng/ml (8.99-84.66) vs 10.88 ng/ml (8.54-89.37)] (p=0.7). Serum CyPA levels were statistically similar [10.47 ng/ml (9.51-14.34) vs 10.88 ng/ml (8.54-89.37)] in patients with and without microalbuminuria (p=0.4).

Conclusions: In our study, there was no significant difference in serum CyPA levels in patients with HTN with and without STOD. Therefore, serum CyPA level was not found to be useful tool for detecting STOD in this group of hypertensive patients. Since there are very few studies in this area, more studies in larger patient groups are needed for conclusive evidence.

Table 1. Demographic, clinical and biochemical characteristics of patients participating in the study

Demographic features	Target organ damage (+) N=56	Target organ damage (-) N=69	p values
Age	59,5±10,7	54,1±7,8	0,002
Gender (M/F)	21/35	32/37	0,3
BMI	29,22±4,7	30,3±4,5	0,3
Systolic blood pressure	141,5±20,1	136,0±19,1	0,1
Diastolic blood pressure	87,5±11,4	87,4±13,0	0,9
Mean blood pressure	112,1±14,4	108,6±15,2	0,2
Smoking status	10/56	17/69	0,4
New diagnosis hypertension	4/56	3/69	0,5
Glomerular filtration rate	88,3±21,3	89,3±13,3	0,7
Drugs			
ACEI/ARB	45/56	54/69	0,7
CCB	17/56	23/69	0,7
BB	15/56	16/69	0,6
Diuretic	24/56	22/69	0,2
Other	1/56	2/69	0,7
Laboratory parameters			
Total cholesterol	210,1±37,6	212,1±42,0	0,7
LDL	147,2±35,8	151,03±41,0	0,6
HDL*	53,5 (12-106)	49,0 (26-97)	0,02
Triglyceride*	122,0(61-549)	115,0 (48-407)	0,7
Serum cyclophilin-A*	10,75 (8,6-84,6)	11,02 (8,54-89,37)	0,6
Serum creatinine	0,80±,18	0,77±0,15	0,3
Fasting glucose	95,1±8,8	95,30±9,0	0,9
HbA1c*	5,6(4,7-6,4)	5,6(4,3-6,4)	0,7
Pulse wave velocity	9,0±1,7	8,1±1,2	0,001
Left ventricular mass index	94,9±15,9	83,3±13,3	0,000

*Creatinine, fasting glucose, total cholesterol, LDL, HDL, triglyceride: mg/dL; Systolic blood pressure, diastolic blood pressure, mean blood pressure: mmHg; Serum cyclophilin-A: ng/ml; BMI: kg/m²; Left ventricular mass index: g/m²; pulse wave velocity: m/s; HbA1c: % HDL: High-density lipoprotein; LDL: Low-density lipoprotein; BMI: Body mass index; ACEI: Angiotensin-converting enzyme inhibitor; CCB: Calcium channel blockers; BB: Beta blocker *Mann Whitney u, p <0.05 was considered statistically significant.*

Family medicine

PP-020

Evaluation of the knowledge, attitudes and behaviors of patients with hypertension who referred to family medicine polyclinic

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Background and Aim: Early repolarization pattern (ERP) is an umbrella term that refers to ST-segment elevation, terminal QRS slur, and terminal QRS notch in an asymptomatic individual. The prognostic significance of ERP detected in electrocardiograms (ECG) of healthy individuals remains to be elucidated. Some researchers has suggested ERP to be a benign incidental finding, while others reported that ERP is associated with life-threatening ventricular arrhythmias. Recently, Tp-e interval, Tp-e/QT and Tp-e/QTc

ratio has emerged as novel noninvasive ECG markers of transmural distribution of ventricular repolarization. Prolonged Tp-e interval and increased Tp-e/QT, Tp-e/QTc ratios was found to be related to tendency of ventricular arrhythmias and sudden cardiac death (SCD). Our aim was to evaluate the transmural distribution of repolarization (TDR) in otherwise healthy and asymptomatic children and adolescents with ERP.

Methods: Thirty-three children with early repolarization pattern in inferior and/or lateral precordial leads on ECG and thirty-two children without any early repolarization were compared in terms of new indices of transmural distribution of repolarization, namely Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio. The Tp-e interval was measured from Tpeak (the highest point of the T wave) to Tend. Tend was defined as the intersection point of the tangent of the down slope of the T-wave and the isoelectric line. All Tp-e measurements were performed from precordial leads.

Results: The median age and weight was 13 (8–17) years and 60 (15–86) kg, respectively (Table 1). Age and weight were not different between the case and control groups. The median Tp-e interval, Tp-e/QT and Tp-e/QTc in children with ERP was 60 (44–72) msn, 0.18 (0.14–0.23) and 0.16 (0.11–0.19), respectively. The median Tp-e interval, Tp-e/QT and Tp-e/QTc in control group was 64 (48–76) msn, 0.19 (0.12–0.23) and 0.17 (0.12–0.22), respectively. There was no difference between children with and without early repolarization in terms of Tp-e interval and Tp-e/QT, Tp-e/QTc ratios. ($p > 0.05$ in all). ERP was found to be present only on lateral leads in 3 cases, only on inferior leads in 15, on both inferior and lateral leads in 15 cases.

Conclusions: To our knowledge, this study is the first to evaluate the novel indices of transmural distribution of repolarization in individuals with. We showed that TDR and thus the tendency of ventricular arrhythmias and SCD were not different between children and adolescents with and without ERP. Further studies are warranted to evaluate TDR in larger groups healthy individuals with different types of ERP.

Table 1. Demographic and electrocardiographic characteristics of the cases

	ERP Group (n:33)	Control Group (n:32)	P value
Age	13 (8–17)	14 (8–17)	0,37
Male sex (%)	23 (69,7)	19 (59,4)	0,38
Weight	60 (15–86)	59 (27–87)	0,98
Heart rate	83,5 (59–97)	77 (58–99)	0,27
Tp-e interval (msn)	60 (44–72)	64 (48–76)	0,21
Tp-e /QT	0.18 (0.14–0.23)	0.19 (0.12–0.23)	0,16
Tp-e/QTc	0,16 (0.11–0.19)	0.17 (0.12–0.22)	0,14

ERP: Early repolarization pattern, msn: miliseconds. Data are expressed as median (minimum- maximum) and number (percentage) where available.

Epidemiology

PP-021

Annual cost of acute myocardial infarction in patients with type 2 diabetes mellitus in Turkey

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Background and Aim: The prevalence of coronary artery disease (CAD) in diabetic patients is 25.6% and the incidence of acute myocardial infarction (AMI) is approximately 0.17% per year in Turkey. In this study, the aim was to calculate the direct annual medical cost of AMI due to T2DM from the national healthcare payer's (SSI) perspective.

Methods: According to the cost of illness methodology, direct annual costs were calculated for T2DM-induced AMI. Local epidemiological data and unit costs of individual health sources were used. Comorbidities, complications, and adverse effects from treatments were included in the AMI cost and weighted by frequency of incidence. Purchasing power parity was used as currency (SPPP1=1.37 TL; OECD2017).

Results: Coronary angiography was the method of scanning in 90% of cases in Turkey; 67% of the patients received a percutaneous coronary intervention (PCI) and 6% underwent coronary by-pass grafting (CABG). Thrombolytic treatment was given in 27% of cases. The mean PCI cost was \$2,587.0. The mean cost of CABG procedure was \$5,467.8. The AMI annual mean cost per person was \$6,044.6 and the annual monitoring cost for CAD for subsequent years was \$895.1. AMI disease cost consisted of 49.0% medical intervention costs, 22.2% hospital costs and 12.5% medication costs. The remainder was formed of 6.4% for comorbidities, 4.8% for follow-up, and 4.7% for complications. Medication constituted 85% of the cost for subsequent years.

Conclusions: In Turkey, 1,983,154 T2DM patients have CAD and 13,170 T2DM patients develop an AMI. The cost of an AMI was \$79,607,897 and the yearly cost of CAD was \$1,854,677,923 due to T2DM. T2DM-induced AMI carries an annual cost of \$1.8 billion, which represents a major economic burden.

Hypertension

PP-022

Coronary flow reserve, carotid intima media thickness and galanin relations with prehypertension

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Background and Aim: Evaluate the coronary microvascular function and presence of subclinical atherosclerosis by measuring coronary flow reserve (CFR) and carotid intima media thickness (CIMT) in prehypertensive people, measure the relationship between blood levels of galanin molecule and blood pressure levels to investigate whether there is a relationship.

Methods: The study was conducted prospectively with patients who applied to the cardiology polyclinic of İstanbul Medeniyet University Göztepe Training and Research Hospital between January 2018 and March 2018. According to clinical blood pressure values, 50 patients were classified as "prehypertensive" and 50 as "normotensive". Baseline demographic characteristics of all cases were recorded, blood samples were taken, serum galanin measurements were performed, carotid intima-media thicknesses were measured, detailed transthoracic echocardiography was performed, and coronary flow reserves were calculated.

Results: There was no significant difference in baseline demographic characteristics between prehypertensive and normotensive groups. In the prehypertensive group, the basal flow was significantly higher, peak flow and CFR (2.4 ± 0.2 , $p < 0.001$ versus 2.1 ± 0.2) were significantly lower. Among the groups, CIMT (0.51 ± 0.01 versus 0.54 ± 0.01 , $p = 0.251$) and galanin (7.0 ± 1.8 versus 6.4 ± 1.5 , $p = 0.097$) were not significantly different. There was no significant correlation between CFR, CIMT, and galanin.

Conclusions: There is a decrease in CFR in prehypertensives and this finding indicates that microvascular function is impaired compared to normotensive. The assessment of CFR in prehypertension was thought to be important for the assessment of cardiovascular risk and for the detection of the presence of early atherosclerosis. In prehypertensives lifestyle changes or medical treatment (based on risk category) that will begin before the blood pressure level progresses to the hypertensive level, can prevent poor outcomes and reduce cardiovascular mortality.

Epidemiology

PP-023

Annual cost of illness of ischemic stroke in type 2 diabetic patients in Turkey

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Background and Aim: In diabetic patients, stroke has a prevalence of 5.7% and an annual incidence of 0.23%. There are 7.7 million diabetic patients in Turkey and diabetes causes a stroke in approximately 18,000 of them each year. In this study, the aim was to calculate the direct annual cost of illness of ischemic stroke (IS) due to type-2 diabetes mellitus (T2DM) from the national healthcare payer's (Social Security Institution, SSI) perspective.

Methods: According to the cost of illness methodology, a direct annual cost of illness was calculated. Local epidemiological data were identified. The unit costs for each resource were calculated from the SSI's perspective using direct costs over a year. Purchasing power parity was used as currency (SPPP1=1.37 TRY; OECD2017).

Results: In the acute phase, 8% of the patients received a stent, 4.6% an endarterectomy, 3% thrombolytic therapy and 1.5% mechanical thrombectomy. The mean costs by procedure were \$4,401.9 for stent, \$5,552.3 for endarterectomy, \$2,958.2 for mechanical thrombectomy and \$1,916.3 for thrombolytic treatment. About 55% of patients were rehabilitated during the year. The mean cost of rehabilitation was \$6,960.9. The mean annual cost of IS per patient was \$7,455.3, and the annual cost of subsequent years was \$1,680.5. The cost of IS was made up of 50% for hospitalizations, 35.6% for medical interventions, 6.8% for medications and 7.5% for follow-up. Of monitoring costs, 58% was for rehabilitation and 30% for medications.

Conclusions: When we consider that 441,562 T2DM patients develop a stroke at some point, of which 173,818 within one year, the cost of T2DM-induced stroke was \$13,838,986 in the first year and \$742,037,124 in subsequent years. T2DM-induced stroke is an important financial burden associated with a health expense of \$875 million per year in Turkey.

Pediatric cardiology

PP-024

Malnutrition; is the heart weak, too?

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Background and Aim: Cardiopathologies such as hypotension, cardiac arrhythmia, heart failure and sudden death are observed in malnutrition. In this study, it was intended to investigate cardiac effects of malnutrition in children.

Methods: The study has been conducted prospectively with 47 malnutrition patients and 44 healthy controls.

Malnutrition cases have been classified as per Gomez classification as mild, moderate and severe malnutrition; and as per Waterlow classification as wasted, stunted, wasted and stunted malnutrition. Electrocardiographic and echocardiographic examination, 24-hour Holter monitoring and biochemical assessment were performed on all cases.

Results: In the malnutrition group, there were 20 (42.5%) males, ages were mean±SD 69.4±57 months, and in the control group there were 19 (43.1%) males, ages were 68.94±48 months, there were no differences between two groups. Left ventricular mass was lower in patient group compared to control group (p<0.05). Cardiac output was lower in severe malnutrition group (p<0.05). Left ventricular ejection fraction and fractional shortening were lower in patient group (p<0.05). Myocardial performance index was determined to be significantly higher in patient group (p<0.01). Deterioration in cardiac functions was associated with severity and duration of malnutrition. Corrected QT dispersion was significantly higher in malnutrition patients (p<0.01) and this increase was associated with duration and severity of malnutrition.

Conclusions: In our study, it was determined that children with malnutrition had atrophy and deterioration in heart contractions and conduction system, mainly in systolic function. We suggest that cardiac morbidity and mortality may be prevented with early detection and treatment of malnutrition in these patients.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-025

Comparison of heart rate variability of the nurses working in intensive care unit during working and resting time

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Background and Aim: Autonomic nervous system (ANS) has significant effects on the regulation of body's functions. ANS is composed of sympathetic and parasympathetic systems that balance each other's work. Among health care personnel, there is a deterioration of the sleep pattern due to night shifts. Sympathetic and parasympathetic systems are adversely affected by this disruption. Heart rate variability (HRV) is an easy and noninvasive method of demonstrating ANS function. In this study, we planned to measure the difference in HRV among intensive care nurses between work and rest days. Thus, we aimed to show the negative situations and the risks that night shifts create on the body.

Methods: 51 nurses working in intensive care unit were included in this study. Subjects was underwent 24 hours 3 channel ambulatory rhythm holter from 08:00-08:00 o'clock twice, once during 24 hours work and once during rest day. We chose 24 hours work day as study group and 24 hours rest day as control group. We gained 5 time domain parameter (Mean NN, SDNN, SDANN, rMSSD and pNN50) and 7 frequency domain parameter (TP, VLF, LF, HF, LFnu, HFnu and LF/HF ratio) from HRV analysis. Either of this parameters reflect the autonomic nerves system status. In this study the datas were analyzed with Statistical Package for Social Science (SPSS) 24.0 package program.

Results: SDNN, SDANN, pNN50, TP, VLF, rMSSD, HF and HFnu are indirect indicator of parasympathetic system and were statistical significant in favor of rest day. LFnu and LF/HF ratio are indirect indicator of sympatic system and were statistical significant in favor of work day. At daytime SDNN, SDANN, pNN50, rMSSD, TP, HF and HFnu were statistical significant in favor of rest day whereas LF, LFnu and LF/HF ratio were statistical significant in favor of work day. At night, rMSSD, pNN50 and HFnu were statistical significant in favor of rest day whereas LFnu and LF/HF ratio were statistical significant in favor of work day. When compared at 24-hour intervals, sympathetic dominance of shift nights started earlier than the resting nights.

Conclusions: In conclusion, sympathetic activation is increased during work days and lasts for 24 hours. Regardless of work or rest day, daytime sympathetic dominance is much more than nights. This was interpreted as a sign of circadian effect. It was also thought that the early onset of sympathetic activity on work days may be related to the earlier onset of cortisol release time compared with rest day.

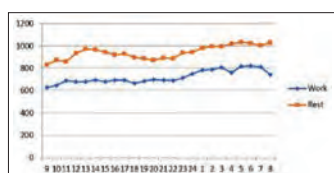


Figure 1. Comparison of work and rest day's mean NN.

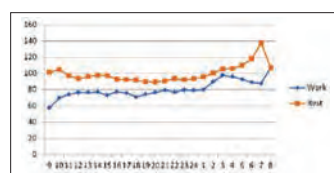


Figure 2. Comparison of work and rest day's mean SDNN.

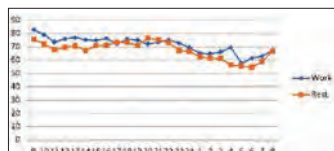


Figure 3. Comparison of work and rest day's mean LFnu.

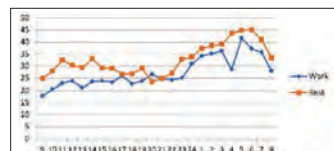


Figure 4. Comparison of work and rest day's mean HFnu.

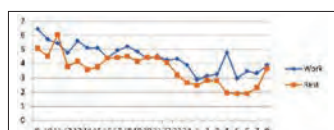


Figure 5. Comparison of work and rest day's mean LF/HF ratio.

Table 1. Defining characteristics of the study population

Characteristics	Mean±SD (Range)	Number %
Age, year	29,35±6,408 (21-44)	51 (100%)
Body mass index	24,79±4,08 (19,03-38,28)	51 (100%)
Working time in intensive care unit, year	39,12±32,89 (3-130)	51 (100%)
Gender		
Man		15 (29,4)
Woman		36 (70,6)
Smoking		
Yes		19 (37,3)
No		32 (62,7)
Marital status		
Single		25 (49)
Married		26 (51)

Table 2. Comparison of work and rest day's heart rate parameters

Heart Rate Parameters	Rest	Work	p	Test
Maximum heart rate, bpm Mean±SD	146,72±16,16	143,25±13,15	0,177	Paired Samples T test
Mean Heart Rate, bpm Mean±SD	80,64±9,59	85,1±8,29	0,00	Paired Samples T test
Minimum Heart Rate, bpm Mean±SD	50,47±6,86	52,63±6,63	0,002	Paired Samples T test
Mean NN (Total), msc Mean±SD	754,88±90,65	712,76±69,41	0,000	Paired Samples T test
Mean NN (Daytime), msc Median (IQR)	710 (152)	671 (94)	0,002	Wilcoxon Signed Ranks test
Mean NN (Night), msc Mean±SD	839,64±144,83	762,42±91,77	0,000	Paired Samples T test
Record Time, min Median(IQR)	1399 (90)	1385 (87)	0,153	Wilcoxon Signed Ranks test

Table 3. Comparison of rest and work day's HRV parameters

HRV parameters	Rest	Work	p	Test
SDNN, msc Median (IQR)	146 (57)	116 (44)	0,00	Wilcoxon Signed Ranks test
SDANN, msc Median (IQR)	130 (50)	104 (43)	0,00	Wilcoxon Signed Ranks test
rMSSD, msc Median (IQR)	36 (20)	31 (21)	0,00	Wilcoxon Signed Ranks test
pNN50, % Median (IQR)	11,72 (16,98)	8,47 (13,8)	0,002	Wilcoxon Signed Ranks test
TP, msc2 Median (IQR)	3000 (2788)	2757 (2618)	0,04	Wilcoxon Signed Ranks test
VLF, msc2 Median (IQR)	1471 (1063)	1276 (1042)	0,017	Wilcoxon Signed Ranks test
LF, msc2 Median (IQR)	960 (828)	873 (976)	0,963	Wilcoxon Signed Ranks test
HF, msc2 Median (IQR)	455 (617)	320 (549)	0,001	Wilcoxon Signed Ranks test
LFnu Ortalama±SD	64,44±13,82	70,06±12,35	0,000	Paired Samples T test
HFnu Ortalama±SD	35,59±13,79	29,93±12,35	0,000	Paired Samples T test
LF/HF Median (IQR)	1,89 (1,59)	2,51 (2,51)	0,000	Wilcoxon Signed Ranks test

Table 4. Comparison of rest and work day's HRV parameters at daytimes

HRV parameters	Rest	Work	p	Test
SDNN, msc Median (IQR)	101 (58,5)	79 (36)	0,001	Wilcoxon Signed Ranks test
SDANN, msc Median (IQR)	76 (55)	57 (32)	0,001	Wilcoxon Signed Ranks test
rMSSD, msc Median (IQR)	31 (26,5)	26 (21)	0,001	Wilcoxon Signed Ranks test
pNN50, % Median (IQR)	9,31 (18,68)	5,38 (12,31)	0,002	Wilcoxon Signed Ranks test
TP, msc2 Median (IQR)	2979 (2466,5)	2713 (2210)	0,014	Wilcoxon Signed Ranks test
VLF, msc2 Median (IQR)	1393 (1298,5)	1194 (1130)	0,1	Wilcoxon Signed Ranks test
LF, msc2 Median (IQR)	1046 (978)	941 (915)	0,257	Wilcoxon Signed Ranks test
HF, msc2 Median (IQR)	370 (907)	247 (386)	0,001	Wilcoxon Signed Ranks test
LFnu Median (IQR)	73,67 (20,75)	78,7 (14,39)	0,014	Wilcoxon Signed Ranks test
HFnu Median (IQR)	26,32 (20,37)	20,24 (13,89)	0,001	Wilcoxon Signed Ranks test
LF/HF Median (IQR)	3,73 (2,85)	4,64 (3,18)	0,035	Wilcoxon Signed Ranks test

Table 5. Comparison of rest and work day's HRV parameters at daytimes

HRV parameters	Rest	Work	p	Test
SDNN, msc Ortalama±SD	113,72±36,76	116,94±37,74	0,504	Paired Samples T test
SDANN, msc Ortalama±SD	84,76±32,69	95,04±36,64	0,053	Paired Samples T test
rMSSD, ms Median (IQR)	42,5 (30,5)	34,5 (30)	0,005	Wilcoxon Signed Ranks test
pNN50, % Median (IQR)	17,29 (26,14)	10,32 (20,58)	0,002	Wilcoxon Signed Ranks test
TP, msc2 Median (IQR)	3166,5 (3541)	2957,5 (3502)	0,308	Wilcoxon Signed Ranks test
VLF, msc2 Median (IQR)	1455,5 (1523,75)	1385,5 (1577)	0,233	Wilcoxon Signed Ranks test
LF, msc2 Median (IQR)	978 (1002,5)	881 (1174)	0,873	Wilcoxon Signed Ranks test
HF, msc2 Median (IQR)	556,5 (719)	449,5 (812)	0,215	Wilcoxon Signed Ranks test
LFnu Ortalama±SD	61,44±13,56	66,83±12,44	0,001	Paired Samples T test
HFnu Ortalama±SD	38,73±13,3	32,89±12,16	0,001	Paired Samples T test
LF/HF Median (IQR)	2,15 (1,97)	2,84 (2,82)	0,000	Wilcoxon Signed Ranks test

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD**PP-026**

Fragmented QRS complexes may predict ventricular arrhythmias in patients with mitral valve prolapsed

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Background and Aim: In mitral valve prolapse (MVP) patients arrhythmic complications are not rare and these arrhythmias are also associated with a risk of sudden death. Studies have shown that MVP may be associated with diffuse left ventricular myocardial fibrosis. Fragmented QRS (fQRS) has been demonstrated as a marker of myocardial fibrosis. fQRS is also a marker of malignant arrhythmias and mortality in various cardiovascular diseases.

Methods: A total of 85 patients with MVP were included in the study. The patients were divided into two groups; the first group consisted of 36 patients with fQRS on 12-lead electrocardiogram (ECG) and the other group consisted of 49 patients without fQRS. All patients were diagnosed with MVP by echocardiography and 24-hour holter ECG monitoring was performed. Then all holter records were analyzed by two independent cardiologists.

Results: There was no statistically significant difference in atrial arrhythmias between the two groups. The incidences of ventricular premature complex (VPC) and ventricular tachycardia (VT) were significantly higher in the fQRS (+) group. In multivariate analysis, the presence of fQRS (Odds ratio: 4.894, p=0.047) and VPC (Odds ratio: 1.008, p=0.012) were independent variables for predicting VT.

Conclusions: fQRS and VPC are independent predictors of malignant arrhythmias such as VT in patients with MVP.

Heart valve diseases**PP-027**

The relationship between serum apelin levels and the severity of calcific aortic stenosis

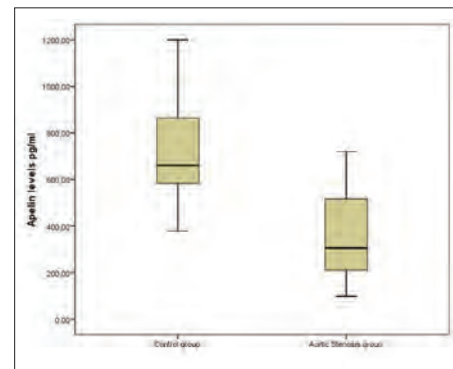
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Background and Aim: Apelin, an endogenous peptide, has recently gained attention due to its positive inotropic effects in heart failure pathophysiology. We investigated the relationship between serum apelin levels and the severity of calcific aortic stenosis (AS).

Methods: A total of 68 consecutive patients diagnosed with calcific AS and a control group of 32 subjects were included in the study. The subjects were divided into three groups as follows: the control group, the mild-moderate AS group, and the severe AS group. Blood samples were obtained from all of the subjects, which were used for biochemical comparisons of apelin 36 and high-sensitive C-reactive protein (hsCRP) levels.

Results: Plasma apelin 36 levels were significantly lower in the patients with severe AS [490 (247-1074) pg/ml] compared to both the mild-moderate AS [209 (97-453) pg/ml] and control [660 (378-1200) pg/ml] groups (p<0.001). Correlation analysis between the left ventricular mass index and apelin concentrations revealed a significant negative correlation between the two parameters (p<0.001, r=-0.478).

Conclusions: Our study demonstrated decreased apelin levels and increased hsCRP concentrations in patients with severe calcific AS. Our findings may help to clarify the exact pathophysiological role of apelin in cardiovascular diseases.

**Figure 1.** Serum apelin concentrations according to aortic stenosis and control group.**Table 1.** Plasma apelin and hsCRP concentrations in patients with AS and the control subjects

	Mild-moderate AS (n = 34)	Severe AS (n = 34)	Control group (n = 32)	p value
Apelin, ng/ml	490 (247-1074)	209 (97-453)	660 (378-1200)	< 0.001
hs-CRP, mg/L	0.40 (0.10-1.20)	0.75 (0.30-2.10)	0.20 (0.1-0.54)	< 0.001

Data are medians with range in parentheses. p values are from Kruskal-Wallis tests across the three groups. AS, aortic stenosis; hs-CRP, high-sensitive C-reactive protein.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD**PP-028**

Comparison of echocardiographic and electrocardiographic parameters of long term follow-up in patients with complete AV block who had implantation of either single or dual chamber pacemakers

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Background and Aim: In this study, at a median follow-up of 7.9 years (3-22), the patients who had implantation either single chamber (VDD) or dual chamber (DDD) pacemakers were compared according to the changes in left ventricular function, pacemaker-related complications, and mortality.

Methods: In between January 1985 and August 2014, a total of 806 patients, who presented with a diverse set of clinical situations and had implanted a single or double chamber pacemaker were retrospectively included in the present study. 446 (37.7%) patients with VDD, and 360 (62.3%) patients with DDD mode pacemaker were compared.

Results: Age, prior history of hypertension, diabetes mellitus, heart failure and coronary artery disease did not differ between the groups (p>0.05). When pre-implantation echocardiographic data was compared to the post-implantation values, in both groups, there was a significant decrease in ejection fraction (EF) (p<0.001) and increase in left ventricular end diastolic diameter (LVEDD) and left ventricular end systolic diameter (LVESD), left atrium (LA) size (p<0.001) and systolic pulmonary artery pressure (sPAP) (p<0.008). When the latest echocardiographic values were compared between VDD and DDD groups, decrease in ejection fraction, increase in LVEDD and LVESD values were higher than in VDD group (EF: 50.8±9.8 vs 48.0±10.4; p<0.009, LVEDD: 52.0±0.8 vs 54.2±0.6; p<0.004, LVESD: 37.3±0.8 vs 39.4±0.8; p<0.014, LA size: 43.4±0.5 vs 45.7±0.5; p<0.0001, respectively). But, sPAP values were similar in both groups (Systolic PAP: 38.0±11 vs 39.9±13.2; p=0.143). The patients who either underwent PCI and CABG before the PM implantation was significantly higher in the DDD-group (CABG 10.3% vs 20.2%, stent implantation 2.2% vs 10.2%; p<0.0001). When the rhythms in the last visit of the patients that could be reached were evaluated, we found that patients with VDD were mostly in sinus rhythm (92.7% vs 47.1%; p<0.001). Heart failure and pacemaker-related complications did not differ between the two groups (p>0.05).

Conclusions: Patients with VDD or DDD pacemakers have both a decline in LVEF and an increase in LV diameter during the longterm follow-up period. Mortality and complication rates were not different between the two groups. When compared DDD group, left ventricular dimensions and functions were better preserved than in VDD group. Therefore, while choosing VDD or DDD-PM selection, patients with AV block undergo individual evaluation in addition to sinus node function.

Table 1. Comparison of pre- and post-implantation echocardiographic data, complication rate, revascularization, current rhythm, between VDD and DDD-groups

	VDD (n=446)	DDD(360)	p value
Average follow-up, years	8.9±4.3	6.3±3.5	<0.0001
Sinus rhythm in last control, n%	82 (92.7)	74 (47.1)	<0.001
Atrial fibrillation in last control, n%	7 (7.3)	24 (15.2)	0.634
Complications, n%	26 (5.4)	16 (4.4)	0.602
Heart failure during the follow-up period, n %	15 (3.3)	16 (4.4)	<0.0001
Comparison of preimplantation echocardiographic parameters			
Ejection fraction (%)	54.4±9.6	50.3±3.5	<0.0001
LVEDD (mm)	51.0±3.5	53.2±0.6	<0.0001
LVESD (mm)	35.3±0.7	38.3±0.8	<0.0001
LA size (mm)	41.9±0.5	43.5±0.5	0.003
Systolic PAP (mmHg)	6.3±3.5	6.3±3.5	<0.0001
Comparison of last visit echocardiographic parameters			
Ejection fraction (%)	50.8±9.8	48.0±10.4	0.009
LVEDD (mm)	52.30±0.8	54.2±0.6	0.004
LVESD (mm)	37.3±0.8	39.4±0.8	0.014
LA size (mm)	43.4±0.5	45.7±0.5	<0.0001
Systolic PAP (mmHg)	38.0±11.0	39.9±13.2	0.143
Preimplantation revascularization, n%			
CABG	46 (10.3)	73 (20.2)	<0.0001
Stent implantation	10 (2.2)	37 (10.2)	<0.0001
All-cause mortality during the follow-up period, n%	102 (22.8)	76 (21.1)	0.271

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD**PP-029**

Relationship of novel parameters of ventricular repolarization with vitamin D level in apparently healthy humans

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Background and Aim: Vitamin D (VitD) is a vitamin which is affective on cardiovascular system. VitD deficiency has been related to increased cardiac and all-cause mortality even in healthy subjects. Preliminary evidence showed that such relatively novel electrocardiographic (ECG) parameters of ventricular repolarization (EPVR) as Tp-e interval, Tp-e/QT and Tp-e/QTc ratios may be related to increased cardiac arrhythmias and even sudden cardiac death (SCD). Little data is available about the effect of VitD deficiency EPVR. The purpose of this study was to evaluate the EPVR in apparently healthy humans with VitD deficiency.

Methods: 72 consecutive VitD deficient and 51 consecutive VitD non-deficient healthy humans who presented to our hospital's outpatient clinics were included in the study as two different groups. The relevant data were obtained through physical examination, electrocardiography and echocardiography. Tp-e interval, Tp-e/QT and Tp-e/QTc ratios were calculated from surface ECG and compared between the two groups using Mann-Whitney U test.

Results: Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio were all observed to be greater in vitamin D deficient group compared with the vitamin D non-deficient group, with robust statistical significance [68.1 ms (61.7-75.4) vs 58 ms (54-66.2); 0.197 (0.179-0.210) vs 0.164 (0.147-0.187); and, 0.172 (0.156-0.191) vs 0.150 (0.137-0.164); respectively; p<0.001].

Conclusions: Our study results reveal that such relatively novel ECG parameters of ventricular repolarization as Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio increase in vitamin D deficiency in apparently healthy humans, which may be related to cardiac arrhythmic complications and sudden cardiac death. Considering vitamin D deficiency gradually becoming a serious health problem worldwide, we hope that our results operate as an additional incentive to overcome this worldwide problem. However, further large-scale studies are needed to support our results.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD**PP-030**

Variability of heart rate or cerebrovascular events?

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Background and Aim: The autonomic system, with its complex central connections and widespread peripheral distribution, allows us continuation of our vitality and to adapt to ever-changing internal and external environmental conditions. Autonomic dysfunction defines disorders that occur in the autonomic nervous

system for various reasons and in the course of some diseases. While investigating cardiac autonomic dysfunction, 24-hour ambulatory Holter ECG recordings were made and the results were evaluated in computer environment. In this study, we aimed to investigate the effects of changes on the cardiac cycle in patients with acute cerebrovascular disease.

Methods: A total of 84 consecutive patients who were admitted with diagnosis of ischemic stroke (IS) (n=51) and transient ischemic attack (TIA) (n=33) were enrolled. A control group of 65 patients were also enrolled. The 24 h time-domain measures of SDNN, SDNN index, HF, LF, mean QT, mean QTc, long QT, long QTc were calculated. Patients with conduction abnormalities on ECG were excluded from the study.

Results: The mean age of the control group was 61±10.3, TIA group 65±9.8, IS group 71±9.3 years, respectively (p<0.000). A total of 79 patients were female. In patients who had IS and TIA had significantly higher hypertension (HT), diabetes mellitus (DM) and serum creatinine levels when compared to patients in control group (Control group HT: 21 (32.3%), DM: 10 (15.4%), serum creatinine level: 0.74±0.2 mg/dl, TIA group 15 (45.5%), 12 (36.4%), 0.76±0.1 mg/dl, IS group 37 (72.5%), 23 (45.1%) 0.97±0.4 mg/dl, respectively, (p<0.000). There were no differences regarding max HR (p=0.920), min HR (p=0.080), mean HR (p=0.060), between the 3 groups (p>0.05). Paroxysmal atrial fibrillation was higher in IS group (n=27, 52.9%, p<0.000). Number of atrial and ventricular early beats were more in the IS group when compared to TIA and control group (p<0.005, p<0.001, respectively). SDNN and SDNN index were lower in patients with IS (p<0.000, p<0.002, respectively). There was no difference between 3 group in terms of HF (p=0.7), but LF value was lower in IS and TIA group (p<0.05).

Conclusions: Heart rate variability, which is used as an early warning signal in determining mortality after myocardial infarction and diabetic neuropathy, may be a predictor of mortality or arrhythmia in patients with acute cerebrovascular disease.

Table 1. Demographic characteristics of patients and heart rate variability parameters

Variables	Control group (n=65)	TIA group (n=33)	IS group (n=51)	P value
Age (years)	61±10.3	65±9.8	71±9.3	0.000
Sex (men), n (%)	28(43.1)	18(54.5)	24(47.1)	0.411
Hypertension (mmHg)	21 (32.3)	15 (45.5)	37 (72.5)	0.000
Blood glucose (mg/dl)	106±21.0	141±21.0	148±66.0	0.000
Creatinine (mg/dl)	0.74±0.2	0.76±0.1	0.97±0.4	0.000
Hgb (gr/dl)	13.7±1.4	13.4±1.5	13.0±1.6	0.047
Diabetes mellitus, n (%)	10(15.4)	12(36.4)	23(45.1)	0.002
Paroxysmal atrial fibrillation n (%)	1(1.5)	12(36.4)	27(52.9)	0.000
Maksimum HR (bpm)	121±17.1	122±25.9	120±24.2	0.920
Minimum HR (bpm)	49±6.6	52±9.5	55±11.2	0.080
Mean HR (bpm)	71±7.9	72±10.8	76±13.4	0.060
Atrial early beat (n)	152±749	971±17031	2132±5163	0.005
Ventricles aerly beat (n)	109 ± 322	348±952	702±1178	0.001
SDNN (ms)	135±54.0	112±40.4	96±37.4	0.000
SDNN index (ms)	54±21.3	47±21.3	40±19.7	0.002
HF (ms ²)	261±401.7	234±380.3	206±344.1	0.742
LF (ms ²)	495±330.0	381±470.5	280±383.8	0.012
meanQT (ms)	359.2±21.2	359.4±22.7	350.1±29.6	0.094
Mean QTc (ms)	389.9±5.4	389.1±5.2	389.6±5.2	0.676
Long QT (ms)	435.1±32.7	433.6±42.1	417.6±39.6	0.033
Long QTc (ms)	441.6±24.8	443.7±21.9	436.8±18.0	0.330

bpm: beats per minute, HR: heart rate.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD**PP-031**

Evaluation of the needle-free transseptal puncture procedure in a tertiary center: Safe transseptal puncture experience

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Background and Aim: Today's modern cardiology has increased use of transseptal puncture (TSP) due to the need for increased left atrial access, despite to the past. The interatrial septum (IAS), because of its anatomic position, it is associated with possible mortal complications. We presented a safe TSP procedure that we performed as needle-free.

Methods: Our safe TSP procedure was routinely use a 6F pigtail catheter from the femoral artery to mark the aortic root. Müllen's sheath is preferred with a 0.032 needle-like shaped guide wire inside. After Müllen's sheath approached to superior vena cava(SVC), it withdrawn smoothly towards the patient's feet while observing the catheter movement, the first movement seen will be the catheter falling into the right atrium (RA) from the SVC and then there will be a second more subtle movement as the catheter falls from the thicker muscular intra-atrial septum. The position of sheath is corrected from LAO and ROA scopic view. Gently sheath pushes, after pressure check and opaque injection if TSP failed, this procedure repeats with needle instead of shape 0.032 guidewire. Retrospectively we collected 61 patients who underwent this procedure for 1 year. We compared complications and clinic features of them.

Results: The records kept by the operator for the year 2017 have been examined. The total number of TSP applications was 61 from these records. The mean age of the patients is 33.2±13.8 and female/male distribution is approximately equalas 31/30. Hypertension was observed in 6 patients (10%) as most seen comorbid disease in our cohort. In our patients, total needle-use TSP was 8 (13%), total needle-free safe TSP was 53 (87%). We have no complications as Pericardial effusion, Aortic root puncture, Atrial wall puncture, Stroke,

Transient ST elevation of inferior leads, Persistence of atrial septal defect, Death. In fact, this can not call as complication but in one case (1.5%) the sheath mistakenly push forward to pulmonary arterial trunk via tricuspid valve during AF ablation, the catheter was withdrawn and the TSP was repeated in his case.

Conclusions: Due to increased need to TSP, new generation equipments will be developed. The SafeSept Needle Free guidewire (SafeSept TM Pressure Products, CA, USA) is the one of these equipments. But in the countries like us, equipment supply can be difficult due to costs. Our procedure we shaped a 0.032 guide-wire like Brockenbrough needle. With this guidewire, we can perform a safe/needle-free TSP and profit from cost of needle.

Table 1. General features of the patients

Age	33.2 ± 13.8
Gender (Male/Female)	30 / 31
Hypertension, n (%)	6 (%10)
Coronary Artery Disease, n (%)	2 (%3)
Diabetes mellitus, n (%)	2 (%3)

Table 2. Procedural dispersion

Procedures	Number	Percent
Mitral Balloon Valvuloplasty	3	5%
-Needle Free	1	1,5%
-Needle-Use	2	3%
Electrophysiological Study	6	10%
-Needle Free	6	10%
-Needle-Use	0	0%
AF Ablation	52	85%
-Needle-Use	48	78%
-Needle-Use	6	10%
Total Needle-Free procedures	8	13%
Total Needle-Use Procedures	53	87%

TSP: Trans-septal puncture, AF: Atrial Fibrillation

Heart valve diseases

PP-032

A potential marker of mitral annular calcification: Monocyte count - to- HDL cholesterol ratio

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Background and Aim: To determine the association of monocyte count-to-high-density lipoprotein-cholesterol ratio (MHR), a recently emerged inflammatory marker, with mitral annular calcification (MAC).

Methods: A total of 1158 patients with MAC and 540 patients without MAC enrolled into the study. MHR and all data were compared between patients with and without MAC. We also performed univariate and multivariate analysis to possible risk factors.

Results: MHR was significantly lower in patients with MAC than control group (p<0.001). MHR (OR: 0.015, 95% CI: 0.001-0.152, p<0.001), mean platelet volume (OR: 0.614, 95% CI: 0.546-0.690, p<0.001), neutrophil-to-lymphocyte ratio (OR: 0.474, 95% CI: 0.322-0.698, p=0.026) and monocyte (OR: 0.032, 95% CI: 0.002-0.124, p=0.023) levels were found to be significantly and independently associated with the presence of MAC.

Conclusions: Higher MHR which indicates an enhanced inflammation and oxidative stress was significantly and independently associated with MAC.

Table 1. Baseline characteristics and laboratory parameters of the study groups

Parameters	MAC (+) group (n=1158)	Control group (n=540)	p value
Age, years	69.9±9.2	68.6±7.9	0.005
Male, n (%)	460(39)	215(39)	0.971
Hypertension, n (%)	448(39)	191(35)	0.189
Diabetes mellitus, n (%)	375(32)	140(27)	0.016
Smoking, n (%)	221(19)	96(18)	0.648
Coronary artery disease, n (%)	147(15)	75(14)	0.715
Hemoglobin, g/dl	13.3±1.5	13.4±1.3	0.279
Platelets, 10 ⁹ /l	248±72.7	244±41.8	0.137
Mean platelet volume, fL	9.1±1.1	8.4±1.5	<0.001
White blood cell, 10 ⁹ /l	7.8±2.0	7.0±1.7	<0.001
Neutrophils, 10 ⁹ /l	4.9±1.8	4.1±1.3	<0.001
Lymphocytes, 10 ⁹ /l	2.0±0.6	2.1±0.6	0.764
Monocytes, 10 ⁸ /l	0.6±0.2	0.5±0.2	<0.001
Neutrophil-to-lymphocyte ratio	2.7±1.2	2.1±0.9	<0.001
Monocyte -to-HDL ratio	0.53±0.18	0.42±0.26	<0.001
Serum creatinine, mg/dL	0.9±0.3	1.0±0.3	0.145
Total cholesterol, mmol/l	5.1±1.1	5.1±0.8	0.432
HDL cholesterol, mmol/l	1.2±0.3	1.3±0.3	<0.001
LDL cholesterol, mmol/l	3.0±0.9	3.1±0.7	0.330
Triglyceride, mmol/l	1.8±1.0	1.6±0.8	0.432
LVEF, %	62.0±6.3	62.4±5.5	0.204

HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; MAC, mitral annular calcification. Data are given as mean±standard deviation, or n (%).

Table 2. Univariate and multivariate logistic regression analyses showing the predictors for the presence of mitral annular calcification

Variables	Univariate analyses OR (95% CI)	p value	Multivariate analyses OR (95% CI)	p value
Age	0.984 (0.976 – 0.993)	0.008	0.988 (0.974 – 1.002)	0.087
Hypertension	1.153 (0.932 – 1.426)	0.189		
Diabetes mellitus	0.755 (0.601 – 0.950)	0.016	0.985 (0.743 – 1.305)	0.076
MPV	0.635 (0.576 – 0.701)	<0.001	0.614 (0.546 – 0.690)	<0.001
WBC	0.794 (0.748 – 0.842)	<0.001	1.123 (0.972 – 1.275)	0.068
Neutrophil	0.703 (0.651 – 0.759)	<0.001	0.810 (0.512 – 1.280)	0.061
Lymphocyte	1.025 (0.872 – 1.205)	0.763		
Monocyte	0.063 (0.033 – 0.121)	<0.001	0.032 (0.002 – 0.124)	0.023
NLR	0.856 (0.781 – 0.937)	<0.001	0.474 (0.322 – 0.698)	0.026
MHR	0.103 (0.059 – 0.180)	<0.001	0.015 (0.001 – 0.152)	<0.001
HDL cholesterol	1.674 (1.214 – 2.308)	<0.001	0.266 (0.097 – 0.726)	0.051

CI, confidence interval; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MPV, mean platelet volume; NLR, neutrophil to-lymphocyte ratio, OR: odds ratio; MHR, monocyte -to-HDL ratio; WBC, white blood cell; LVEF left ventricular ejection fraction.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-033

Relationship between a fragmented QRS and QT dispersion in patients with acute myocardial infarction

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Background and Aim: QT dispersion can be used to predict life-threatening ventricular arrhythmias. To our knowledge, the relationship between fragmented QRS (fQRS) and QT dispersion has not been studied. We evaluated the relationship between fragmented QRS (fQRS) and QT dispersion in patients with acute myocardial infarction (MI).

Methods: We studied 197 patients with acute myocardial infarction, and patients were classified into two groups: 85 patients with fQRS(+) group and 112 patients with fQRS(-) group. ECG were recorded at baseline, and fQRS contains different morphologies of QRS, and it includes an additional R wave (R') or notching in the nadir of the S wave damaged area in two contiguous leads in damaged area. ECG recordings were used for the evaluation of the QT dispersion, and the QT was defined as the interval between the QRS onset and the end of the T wave. The QT dispersion was calculated as the maximal QT interval minus the minimal QT interval in the leads.

Results: A total of 197 patients (mean age: 56.2±10 years) with acute MI were included, and the study included 45 female and 152 male patients. The patients with fQRS(+) had longer QT dispersion than patients with fQRS(-) at the baseline.

Conclusions: Our results showed that the presence of fQRS(+) had longer QT dispersion in patients with acute MI, and the patients can be followed for ventricular arrhythmia. The fragmented QRS can be used as an arrhythmia marker.

Table 1.

	fQRS(+)	fQRS(-)	P
Mean Age (year)	55,9±10	56,6±10	0,58
Glucose (mg/dl)	169±79	156±75	0,19
WBC Count, (×10 ⁹ /l)	15±4	12±3,6	<0,001
LDL (mg/dl)	121±30	119±33	0,56
LV EF (%)	42,7±9	48,3±8	<0,001
QTmax	41±24	40±29	0,39
QTmin	35±25	36±34	0,37
QT dispersion	59,5±20	50±24	0,004

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-034

Clinical characteristics and long-term follow-up results of patients who underwent tilt table test with vasovagal syncope pre-diagnosis

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Background and Aim: This study aimed to investigate the demographic and clinical characteristics and to compare the incidence of the events before application and after the treatment of patients who underwent tilt table test (TTT) with vasovagal syncope (VVS) pre-diagnosis.

Methods: This study included 74 patients who underwent TTT and whose follow-up was reached. TTT results, demographic and clinical data, recommended and/or applied treatments of patients were recorded.

The changes in symptoms were compared before application and after TTT follow-up period.

Results: The mean age of patients was 37 years and 52.7% were male. All patients had symptoms that accompany with the event. Palpitation (71.6%) was the most frequent accompanying symptom. TTT was positive in 40.5% of patients. Patients with TTT positive had low body mass indexes and high alcohol use rates. Of the patients with positive TTT, 76.7% didn't experience any event at treatment and follow-up. In TTT negative patients, the frequency of events was significantly reduced with lifestyle modification suggestions. **Conclusions:** In patients suspected of VVS, TTT is useful for diagnosis. Treatment with TTT positive patients in accordance with current guidelines reduces the frequency of events at high rates. In patients with suspected VVS but negative TTT, a lifestyle change should be proposed considering the false negative of the test.

Heart valve diseases

PP-035

Safety and efficacy of percutaneous balloon mitral valvotomy in severe mitral stenosis with moderate mitral regurgitation

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Background and Aim: Percutaneous balloon mitral valvotomy (PBMV) is generally considered as a contraindication in patients with mitral stenosis (MS) associated with moderate to severe mitral regurgitation (MR). We sought to compare the safety and efficacy of PBMV in patients with severe MS and with moderate MR with those with less than moderate or no MR.

Methods: One hundred and three RHD patients who underwent PBMV for rheumatic MS between May 2016 and June 2018 were included in our study patients of MS with mitral valve area 1.5 cm² were screened into two groups: Group I with moderate MR and Group II with less than moderate or no MR. Primary safety outcome was a composite of cardiovascular death and development of severe MR requirement for mitral valve replacement at 1 month of procedure.

Results: There were 94 patients in Group I and 9 in Group II. Both groups were comparable with respect to baseline characteristics, as shown in Table 1 Three patients in Group I developed severe MR, and 30 days emergency MVR. Perioperative complications developed one patients in Group 1 and group 2 this patient underwent emergency MVR, Table 2. The composite of primary events was not statistically different between the two groups p=0.373 Table 2 There was no statistical difference in EROA change between group 1 and group 2 after procedure. Wilkins Score was positively correlated with quantity of Mitral regurgitation (r=0.551, p<0.001), Figure 1.

Conclusions: In patients having severe MS associated with moderate MR, PBMV may be a safe option and provides sustained symptomatic benefit. PBMV is effective for treating rheumatic mitral stenosis in Turkish patients with moderate mitral regurgitation.

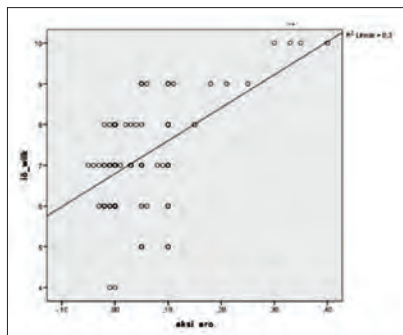


Figure 1. Wilkins Score correlation with quantity of Mitral regurgitation.

Table 1.

Parameter	Group I	Group II orta my	p
Age, years, mean (SD)	41,4 + 10,6	46,3+8,6	0,182
Women (%)	79 (%84)	7 %77.8	0.641
Pulmonary artery systolic pressure, mmHg, mean	51,39+16,8	57,4+18,2	0,308
Presence of atrial fibrillation (%)	10 %10,6	3 %33.3	0,085
Mean mitral gradients by echo, mean	16,45+5,51	15,78+5,72	0,729
2-D MVA, cm2, mean	0,98+0,23	1,06+0,23	0,341
WILKINS SCORE	7,2+1,2	6,9+1,7	0,457

Table 2.

	GROUP 1	GROUP 2	P
CV death within 30 days	0	0	1
MVR for severe MR within 30 days	3 %3.2	0	1
periprocedural complications	1 %1,8	1 %11,1	0,168
composite	4 %4.3	1 %11.1	0,373

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-036

A novel biomarker predicts sudden cardiac death in hypertrophic cardiomyopathy: Galectin-3

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Background and Aim: Hypertrophic cardiomyopathy (HCM) is a primary cardiac disease, characterized by left ventricular hypertrophy, myocyte hypertrophy and irregularities, and interstitial fibrosis in the absence of any cardiac or systemic diseases, which may lead to sudden cardiac death (SCD). Galectin-3 (gal-3) is a β-galactoside-binding lectin and it has been demonstrated to be associated with cardiac fibrosis and inflammation. In this study, we aimed to investigate the relationship between serum Galectin-3 levels and the criteria for 5-year sudden death risk (HCM Risk-SCD), which has been defined in the recent ESC guideline (2014), in patients with hypertrophic cardiomyopathy.

Methods: A total of 52 hypertrophic cardiomyopathy patients were enrolled in the study. Patients were questioned for sudden death risk predictors as outlined in the 2014 ESC guideline. A standardized clinical evaluation was carried out on the basis of previously described prognostic variables to be used to calculate the 5-year risk of sudden cardiac death. Statistical significance level was accepted as p<0.05 in all tests.

Results: Fifty two patients took part in the study and majority of them (n=34) were male. The mean age of the patients was 48±12.9 (min: 22, max: 65). Basic characteristics of patients were seen in Table 1. Serum Galectin-3 levels and HCM Risk-SCD score had statistically significant correlation (r=0.83, p<0.001). Age (r=-0.168, p=0.23), maximum left ventricular outflow gradient (r=0.04, p=0.78), maximal left ventricular wall thickness (r=0.26; p=0.07) and left atrial diameter (r=-0.21, p=0.88) didn't have any statistically significant correlations. (Table 2) Patients were grouped in three categories according to the HCM Risk-SCD scores. Patients in the low-risk group (<4%), group 1, had a mean serum level of Galectin-3 of 16.5±3.6 ng/ml, while patients with moderate risk (4-6%), group 2, had a mean serum level of Galectin-3 of 23.1±1.8 ng/ml group. High risk (>6%) group of patients, group 3, had a mean serum Galectin-3 levels of 28.5±3.9 ng/ml. Serum Galectin-3 levels were statistically different from each other according to risk groups (p<0.01) (Figure 1).

Conclusions: Galectin-3 may be a cheap and easily accessible parameter to predict arrhythmia risk. In addition, it can be used in the decision of anti-arrhythmic prophylaxis as a predictor of arrhythmia storm in ICD implanted patients who are not available for MR imaging.

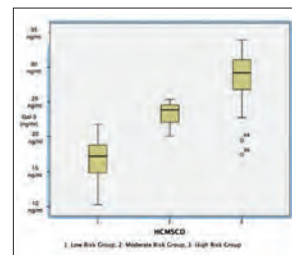


Figure 1. Patients' serum Galectin-3 levels according to risk classifications for sudden cardiac death.

Table 1. Basal demographic, clinical and echocardiographic parameters

Parameter	Minimum value	Maximum value	Mean±Standard deviation
Age (years)	22	65	48.3±12.9
Left atrial diameter	31	56	43.3±3.1
Maximal Left ventricular outflow tract gradient (mmHg)	2	130	26±31.6
Maximal left ventricular wall thickness	15	37	23.22±5.0
HCM Risk SCD score (%)	1.38	22.87	7.78±5.8

Sex	Male (n=38)	Female (n=14)
Cardiac arrest history	Yes (n=9)	No (n=43)
or Suitable ICD shock		
Syncope	Yes (n=19)	No (33)
Ventricular tachycardia	Yes (n=28)	No (24)
ICD implanted patients	Yes (n=25)	No (27)
History of sudden death in family	Yes (n=32)	No (20)

Table 2. Correlation of serum galectin-3 levels with HCM risk SCD score's and other parameters

	Correlation co-efficient	P value
Age	-0.168	0.23
Maximal Left ventricular outflow tract gradient	0.04	0.78
Maximal left ventricular wall thickness	0.26	0.05
Left atrial diameter	-0.21	0.88
HCM Risk SCD score	0.83	<0.001

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-037

Serum apelin and asymmetric dimethylarginine (ADMA) levels in patients with vasovagal syncope

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Background and Aim: Vasovagal syncope (VVS) has a high morbidity, with good prognosis, which is most often caused by syncope or disrupts the quality of life affected by more than one pathophysiological mechanism. Apelin is a newly defined adipokine. Asymmetric dimethylarginine (ADMA) is the major inhibitor of NO synthase. The role of apelin and ADMA in the pathophysiology of vasovagal syncope has not been previously investigated. In this study, we aimed to investigate the changes in Apelin and ADMA levels of patients who were diagnosed with VVS immediately before and after the tilt test.

Methods: 50 patients were included in the study whose over 18 years of age who were followed up with our outpatient cardiology clinic recurrent vasovagal syncope diagnosis. The control group consisted of 25 healthy volunteers with age and gender matched. Patients with structural heart disease, metabolic disease history, patient sinus syndrome, carotid hypersensitivity, intraventricular dysfunction, orthostatic hypotension, atrial fibrillation, renal dysfunction and vasoactive medications were excluded. Blood samples were taken before and immediately after the test to evaluate Apelin and ADMA levels in healthy volunteers with tilt table and VVS-diagnosed patients.

Results: The mean age of the patients with VVS was 24.70±3.86 while the mean age of healthy volunteers was 25.92±3.33. 33 were women (66%) of the 50 patients diagnosed with vasovagal syncope (VVS), and also 14 were women (53%) of the 26 healthy volunteers. There was no difference between Apelin vs ADMA values in VVS and control group before the tilt test (Apelin 267 (130-445) vs 215 (93-374), p 0.293; ADMA 939 (545-1343) vs 955 (503-1343), p 0.876). There was no statistically significant difference in both Apelin and ADMA values, when the measured values were compared prior to and following the tilt test in the control group (Apelin 215 (93-374) vs 215 (137-443); p 0.069, ADMA 955 (503-1343) vs 971 (539-1634); p 0.148). After Tilt test, in patients with VVS, Apelin values were higher than baseline and ADMA values were lower (Apelin 267 (130-445) vs 1529 (1152-2768); p<0.001, ADMA 939 (545-1343) vs 107 (66-198); p<0.001).

Conclusions: We reviewed the changing of apelin and ADMA levels prior to and following tilt table in patients with vasovagal syncope. It was detected that Apelin levels were increased while ADMA levels decreased after the test in the patient with VVS. The change in apelin and ADMA levels in patients with VVS may have a role in the appearance of syncope.

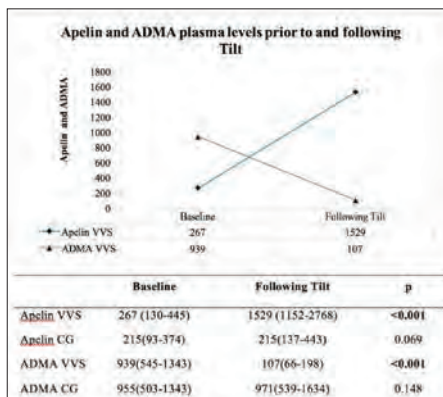


Figure 1. Apelin and ADMA levels measured prior to and following the tilt test.

Table 1. Comparison of demographic and clinical properties between VVS and control groups in the study population

	VVS(n=50)	Control(n=26)	p
Age (year)	24 ± 3.86	25.92 ± 3.33	0.175
Gender (women %)	33 (% 66)	14 (% 53)	0.301
LVEF	65.50± 5.19	67.53 ± 4.30	0.090
SBP (mmHg)	112.06± 11.63	115.00± 6.63	0.238
DBP (mmHg)	70.26 ± 8.58	73.26 ± 4.45	0.099
HR (min)	73.44± 3.59	74.76± 3.16	0.116
TTST	35 (22-38)		
VD Type	26(%52)		
Mixed Type	24(%48)		
TSS	4.26± 0.98		
SS (within 6 months)	2.34± 0.74		

SBP: Systolic blood pressure; DBP: Diastolic blood pressure; LVEF: Left ventricular ejection fraction; NPM: Number of pulse minutes; TSN: Total syncope number; SN: Syncope number; VD: Vazodpressor; TTST: Tilt test syncope time.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-038

Predictive value of CHA2DS2-VASc and CHA2DS2-VASc-HS scores for developing atrial fibrillation in patients with ST-elevation myocardial infarction

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Background and Aim: Atrial Fibrillation (AF) is the most common type of the supraventricular arrhythmia following ST-segment elevation myocardial infarction (STEMI), and its prevalence is even higher in elderly patients with heart failure and severe left ventricular impairment. The CHA2DS2-VASc risk score is a cheap and easy scoring system which is calculated by assigning 1 point for each; congestive heart failure (ejection fraction <40%), hypertension, age between 65 and 74 years, diabetes mellitus, vascular disease (myocardial infarction or peripheral arterial disease) and female sex, 2 points for; a history of stroke or transient ischemic attack (TIA) and age >75 years. The CHA2DS2-VASc risk score is used to predict the thromboembolism risk in non-valvular atrial fibrillation (NVAF) patients. We aimed to evaluate the predictive value of the CHA2DS2-VASc risk score in the development of AF in patients presenting with acute STEMI.

Methods: Total of 360 patients who had been diagnosed with STEMI and who had undergone primary coronary angioplasty or thrombolytic therapy were included in the study. The patients were divided into two groups according to the CHA2DS2-VASc score, i.e.: low risk (0 or 1 point), high risk (≥2 points). The groups were followed with regard to AF development.

Results: In the patients with AF, The median CHA2DS2-VASc and CHA2DS2-VASc-HS score was significantly higher compared to those without AF (2.6±1.5 vs 1.8±1.4 and 3.2±1.3 vs 2.3±1.4, p<0.001 and p=0.001, respectively). The rate of AF was 3.7-fold higher (OR 3.7, 95% CI 2.10-6.5, p<0.001) in the high-risk group (CHA2DS2-VASc ≥2) compared to the low-risk group (CHA2DS2-VASc = 0 or 1). Older age, hypertension, left ventricle ejection fraction, Left atrium diameter, female gender, CHA2DS2-VASc score, CHA2DS2-VASc-HS score reached statistical significance in univariable logistic regression analysis. (Age (OR 1.05, 95% CI 1.02-1.07, p<0.001), female gender (OR 1.99, 95% CI 1.15-3.43, p=0.03), CHA2DS2-VASc score (OR 1.5, 95% CI 1.30-1.78, p<0.001), CHA2DS2-VASc-HS score (OR 1.49, 95% CI 1.26-1.76, p<0.001), hypertension (OR 2.1, 95% CI 1.29-3.59, p=0.03), left ventricle ejection fraction (OR 1.05, 95% CI 1.03-1.11), p<0.001). When we put left atrial diameter, left ventricle ejection fraction and CHA2DS2-VASc score or CHA2DS2-VASc group or CHA2DS2-VASc-HS score into multivariable logistic regression analysis left ventricle ejection fraction, CHA2DS2-VASc score, CHA2DS2-VASc group and CHA2DS2-VASc-HS score were found to be independent predictors of AF. According to ROC analysis, a CHA2DS2-VASc score of at least 1,5 was predictive of CIN with 75% sensitivity and 55% specificity (area under curve =0.69, p<.001, 95% CI (0.62-0.75) and a CHA2DS2-VASc-HS score of at 2,5 was predictive of CIN with 75% sensitivity and 60% specificity (AUC=0.67, p<.001, 95% CI (0.61-0.74).

Conclusions: The CHA2DS2-VASc and CHA2DS2-VASc-HS score is an independent and strong predictor of AF development in patients with acute STEMI.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-039

SYNTAX and Clinic SYNTAX score as a predictor of atrial fibrillation after isolated coronary artery bypass

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Background and Aim: Atrial fibrillation (AF) is the most common arrhythmia after coronary artery bypass grafting (CABG). In our study we aimed to find out AF predictors especially SYNTAX score and Clinical SYNTAX score (CSS) after isolated CABG.

Methods: This study is designed retrospectively and included total 249 patient who has 1 vessel lesion at least between 2011-2013 underwent CABG. The exclusion criteria were a history of emergent CABG operation, severe valvular diseases went operation, congenital heart diseases. In this study, NCSS (Number Cruncher Statistical System) 2007 Statistical Software (Utah, USA) pocket programme was used.

Results: Retrospectively collected data showed in PoAF(+) groups DM frequency (p<0.001), uric acid (5.96 vs 5.21 p=0.032), neutrophil (6.11 vs 5.08 p<0.01), NLR average (3.48 vs 2.54 p<0.01) were found high statistically significant than PoAF(-) group. In PoAF(+) group leucocyte (1.94 vs 2.24 p<0.01) and EF average (50.06 vs 53.22 p=0.017) were found lower statistically significant than postop AF(-) group. In Postop AF(+) group LAD safenous (11.27% vs 4.49% p<0.01) and Cx safenous were found higher statistically significant than PoAF(-) group (p=0.049). In Postop AF(+) group Intensive Care Unit stay time(day) (p=0.045) and stay in hospital (day) were found higher statistically significant than PoAF(-) group (p=0.002). In PoAF(+) group, SYNTAX score average (30.69 vs 28.52 p<0.01) and CSS average were found higher statistically significant than PoAF(-) group (8.34 vs 6.49 p=0.001).

Conclusions: In our study, we found many predictors of PoAF, and independent predictors were found HbA1c, NLR and SYNTAX score highness. We could not found SYNTAX score and logistic CSS enough valuable of differential diagnosis of PoAF.

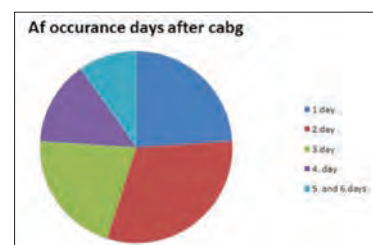


Figure 1. Atrial fibrillation occurrence days.

Table 1. Preoperative patient's basic characteristics

	Postop AF (-) n:178		Postop AF (+) n:71		p
Age	60,94±6,47		61,69±8,15		0,444
Sex	Female	36 20,22%	22 30,99%	0,07	
	Male	142 79,78%	49 69,01%		
DM	44 35,39%	63 61,97%			0,0001
HT	97 54,49%	38 53,52%			0,889
Smoking	71 39,89%	33 46,48%			0,341
EF	53,22±9,01	50,06±10,3			0,017
TCholesterol	199,94±54,03	205,24±47,97			0,472
HDL	39,28±12,15	38,24±10,35			0,528
LDL	129,85±43,57	133,54±40,5			0,540
TG	187,6±107,55	223,06±196,73			0,07
Glucose	128,61±47,02	165,25±77,37			0,0001
HbA1c	6,71±1,67	8,28±6,1			0,002
Uric acid	5,25±1,71	5,96±1,88			0,082
Creatinin	1,16±1,54	1,95±3,27			0,341
GFR	92,92±29,52	86,65±30,07			0,133
Leucocyte	6,76±2,56	9,1±2,93			0,366
Hemoglobin	15,53±26,55	13,55±1,53			0,536
Platelete	248,46±69,2	246,01±67,25			0,802
Neutrophil	5,08±1,32	6,11±2,27			0,0001
Lymphocyte	2,20±6,63	1,94±0,52			0,002
NLR	2,54±1,18	3,48±1,50			0,0001
Previous MI	78 43,82%	32 45,07%			0,858
Previous PCI	60 33,71%	31 43,66%			0,141
LAD Lima	174 97,75%	69 97,18%			0,791
LAD Saphenous	8 4,49%	8 11,27%			0,049
D1 Saphenous	89 50,00%	32 45,07%			0,482
IM Saphenous	19 10,67%	7 9,86%			0,849
CX Saphenous	44 24,72%	11 15,49%			0,113
CXOM Saphenous	107 60,11%	47 66,20%			0,372
RCA Saphenous	96 53,93%	33 46,48%			0,288
RCA PDA	39 21,91%	17 23,94%			0,729
RCA PI	5 2,81%	3 4,23%			0,567
Follow Time (mounth)	39,37±3,35	39,56±3,86			0,687
Graft number	3,25±0,81	3,2±0,79			0,622
SYNTAX score	28,52±5,62	30,69±6,28			0,008
Clinical SYNTAX Score	6,49±3,75	8,34±4,39			0,001
ICU Stay day	1,12±0,66	1,18±0,76			0,540
Stay in Hospital day	8,63±3,85	9,61±5,86			0,124

Table 2. Multivariate regression analysis for the investigation of independent correlates of predictor of PoAF

	B	S.E.	p	OR	OR %95 GA	
					Low	High
DM	0,01	0,50	0,983	1,01	0,38	2,70
HbA1c	0,34	0,12	0,004	1,41	1,11	1,77
Uric Acid	0,23	0,13	0,064	1,26	0,99	1,61
NLR	0,43	0,16	0,008	1,54	1,12	2,12
LAD Saphenous	-1,31	0,89	0,142	0,27	0,05	1,55
SYNTAX Score	0,07	0,03	0,047	1,07	1,00	1,14
Clinical SYNTAX Score	-0,05	0,06	0,384	0,95	0,85	1,07

Table 3.

	AUC	SE	95% CI
SYNTAX Score	0,586	0,041	0,522 - 0,648
Clinical SYNTAX Score	0,596	0,041	0,532 - 0,657

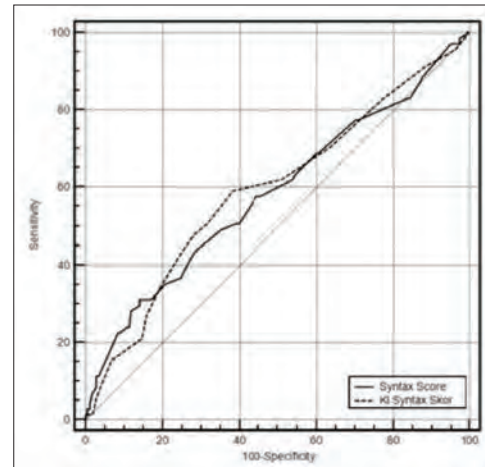


Figure 1. ROC analysis.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-040

Analysis of demographic, laboratory and 12 lead surface electrocardiographic parameters in patients with spontaneous ventricular tachycardia

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Background and Aim: The presence of ventricular tachycardia on ambulatory electrocardiographic (ECG) monitoring has been associated with an increased incidence of subsequent sustained tachycardia and sudden cardiac death in most patients. In this study; we aimed to investigate the 12 lead surface electrocardiographic and several laboratory properties in patients with spontaneous nonsustained/sustained ventricular tachycardia who have no structural heart disease.

Methods: Thirty subjects (group 1; 20 M, mean age 57.4±16.8) who has admitted to the cardiology polyclinic with complaint of palpitation, dizziness or syncope and 30 healthy subjects (group 2; 12 M, mean age 48.2±7.7) who have undergone 24- hour rhythm holter recordings were included to the study. Group 1 consisted from subjects who were detected nonsustained or sustained ventricular tachycardia in 24- hour rhythm holter recordings. Subjects with a history of long QT syndrome (personal or family) or other cardiac conduction disorder or other clinically significant cardiac disease or thyroid/renal dysfunction were excluded. All the subjects in group 1 has undergone transthoracic echocardiography, exercise stress test, myocardial perfusion scintigraphy and coronary angiography to exclude structural and coronary heart disease, if necessary. Fasting blood glucose, haemogram, lipid profile, renal and hepatic function tests were studied from venous blood samples taken at the time of admission. 12 lead ECGs were obtained simultaneously using a recorder set at a 50 mm/s paper speed and a voltage calibration of 1 mV/cm. ECGs were interpreted using standard criteria. ECG parameters were measured using a digital caliper (sensitivity: 1/100 mm) by magnifying lens. Maximum, minimum P wave, P peak time, PR and maximum, minimum QT durations were calculated. P, QT dispersion, and cQT dispersion were calculated. P wave amplitude, intrinsicoid deflection time, index of cardio-electrophysiological balance (iCEB: QT/QRS) were also calculated.

Results: The patients in group 1 have significantly higher age and glucose levels compared to group 2 (Table 1). Group 1 have significantly higher number of hypertensive and diabetic subjects. 2 lead surface ECG analysis revealed that significantly higher P wave dispersion, QT dispersion, cQT dispersion, P wave peak time, PR duration and QRS duration (Table 2). Intrinsicoid deflection time, P wave amplitude, P wave terminal force and iCEB were similar between the groups (Table 2). There were statistically significant correlation between glucose levels and QTd, cQTd and iCEB (r=0.38; p=0.16, r=0.37; p=0.21, r=0.35; p=0.03, respectively).

Conclusions: Patients with spontaneous nonsustained/sustained tachycardia and no structural/coronary heart disease, have prolonged 12 surface ECG parameters especially PWD, QTd, cQTd, Pwave peak time and QRS durations. These group of patients tend to have higher ages and glucose levels. Glucose levels may be associated with higher repolarization parameters.

Table 1. Demographic and laboratory characteristics of the groups

	Group 1 (n=30)	Group 2 (n=30)	P
Age (years)	57.4±16.8	48.2±7.7	0.03
Gender (M)	20	18	0.59
BMI (kg/m ²)	25.9±3.8	24.2±4.0	0.16
Glucose (mg/dL)	118.4±45.2	91.2±7.1	0.02
Urea (mg/dL)	36.8±14.7	31.6±13.4	0.21
Creatine (mg/dL)	0.85±0.27	0.72±0.23	0.09
WBC (x10 ³ /L)	7.87±2.50	7.83±1.67	0.94
Total cholesterol (mg/dL)	187.7±46.7	181.1±16.1	0.55
Triglyceride (mg/dL)	117.8±47.6	106.6±44.5	0.49
High density lipoprotein (mg/dL)	45.1±11.0	51.0±10.8	0.13
Low density lipoprotein (mg/dL)	121.0±36.8	108.3±24.4	0.22

Table 2. Comparison of electrocardiographic parameters of the groups

	Group 1 (n=30)	Group 2 (n=30)	P
P wave dispersion (ms)	16.4±4.9	12.8±4.2	0.012
P wave peak time (ms)	48.7±15.2	37.5±10.2	0.008
P wave amplitude (ms)	46.4±16.8	44.0±11.6	0.59
P wave peak time (ms)	48.7±15.2	37.5±10.7	0.008
P wave terminal force	30.0±12.1	32.5±11.7	0.49
PR duration (ms)	162.7±26.8	145.2±12.7	0.01
QT dispersion (ms)	27.6±16.6	16.7±7.4	0.003
σQTd (ms)	28.2±18.6	19.1±10.0	0.04
QRS (ms)	93.1±24.5	83.0±6.9	0.04
Intrinsicoid deflection time (ms)	35.4±10.7	29.7±10.1	0.08
iCEB	5.26±1.62	5.23±0.62	0.92

Heart valve diseases

PP-041

Immediate and long term effects of percutaneous mitral balloon valvuloplasty on atrial conduction velocities in patients with mitral stenosis

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Background and Aim: P-wave dispersion (PWD) is an electrocardiographic (ECG) marker of nonuniform and heterogeneous atrial conduction with ECG leads of different orientation. Previous investigations have shown that Pmax and PWD are increased in patients with rheumatic mitral stenosis (MS) and decreased after percutaneous mitral balloon valvuloplasty (PMBV). The aim of our study was to investigate the immediate and long term effects of successful PMBV on PWD in severe rheumatic MS patients with sinus rhythm (SR) and to analyse the restenosis, atrial fibrillation (AF) and redo balloon valvuloplasty rate.

Methods: We enrolled 41 consecutive MS patients with SR who underwent PMBV in our institution. A 12-lead ECG and transthoracic echocardiography were performed for each patient one day before and within 72 hours after the procedure. All patients were followed up by clinical visit at a mean of 5.57±1.46 (3-8) year after the index PMBV. Because having AF at 3rd year for four patients, we examined ECG at 3rd year for all patients and continued to follow up clinically and echocardiographically. Visits are grouped as 1 (prePMBV), 2 (1-3 days after PMBV), 3 (long term after PMBV) according to the time of PMBV. Composite endpoints included AF, restenosis, redoballoon valvuloplasty, mitral valve surgery.

Results: The mean patient age was 44.1±10.86 years and 95% (n=39) of the patients were female. Baseline characteristics were shown in Table 1. Pmax 1 and PWD 1 decreased 1-3 days after PMBV but the difference at long term was not significant (Figure 1, Table 1). MVA improved immediately after the procedure; but lately the mean MVA decreased significantly indicating the initiation of restenosis. Composite endpoints were associated with LA 1 (p=0.02), MVA 2 (p=0.019), mean gradient 2 (p=0.028), PWD 3 (p<0.001) and Pmax 3 (p<0.001). AF incidence was associated with PWD 2 (p=0.019) and PWD 3 (p=0.010). There was 14 composite endpoint on follow up and at multivariate analysis PWD 3 was identified as an independent predictor of the composite endpoint (p=0.048, hazard ratio=1.36, 95% confidence interval (CI): 1.002-1.867). By receiver operating characteristic (ROC) curve analysis, increased PWD was significantly associated with major adverse cardiac events (area under ROC curve [AUC]: 0.905, 95% confidence interval [CI]: 0.813-0.996).

Conclusions: This study has demonstrated that Pmax and PWD significantly decreased within 3 days after PMBV, furthermore, long term PWD was associated with AF and identified as an independent predictor of the composite endpoint. Patients without any decrease in Pmax and PWD long term after PMBV may potentially be at greater risk for atrial fibrillation and restenosis. By electrocardiographically P wave indices, immediate and long term effects of PMBV on relieving mitral valve obstruction can be reflected and also AF and restenosis can be predicted.

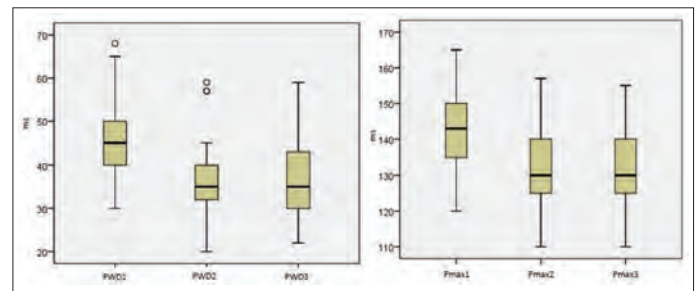


Figure 1. Box-plot representations for PWD and Pmax between three groups. PWD 1/Pmax 1:pre PMBV, PWD 2/Pmax 2:1-3 days after PMBV, PWD 3/Pmax 3:long term after PMBV.

Table 1. Baseline and after PMBV clinical, electrocardiographic and echocardiographic characteristics of patients

	pre-PMBV	1-3 days after PMBV	long term after PMBV	p
Age (years)	44,1±10,86	-	-	-
Echo score	8 (6-12)	-	-	-
Gender (F/M)	39/2	-	-	-
MVA	1,07±0,19	1,91±0,35	1,63±0,40	<0,001
Max grad	23±8,6	12±5,54	15,1±6,02	<0,001
Mean grad	14,51±6,22	5,76±3,04	7,29±3,92	<0,001
PAPs	45 (30-120)	35 (25-120)	35 (20-115)	<0,001
LA diameter (cm)	44 (34-69)	42 (33-54)	42 (33-50)	<0,001
LVEDD (cm)	28 (19-37)	29 (21-39)	30 (15-39)	0,142
LVEDD (cm)	46 (38-52)	47 (33-55)	48 (32-53)	0,058
LVEF (%)	65 (55-65)	65 (50-65)	60 (50-65)	0,131
P-max (ms)	143 (120-165)	130 (110-157)	130 (110-155)	<0,001
P-min (ms)	100 (75-115)	95 (75-115)	95 (80-111)	0,092
PWD (ms)	45 (30-68)	35 (20-59)	35 (22-59)	<0,001

Table 2. Univariate regression analysis for the occurrence of composite events included in restenosis, redoballoon, atrial fibrillation, mitral valve surgery

	composite endpoint - n:27	composite endpoint + n:14	p
Age	44±12	44±10	0,202
Echo scor	8 (6-12)	9 (6-10)	0,376
Pmax 1	141±11	146±13	0,202
Pmax 2	129,3±10	135,6±14,3	0,104
Pmax 3	126,4±9,8	142,4±7,7	<0,001
Pmin 1	96±10	99±11	0,284
Pmin 2	95±9	95±9	0,978
Pmin 3	93,4±8,8	97,7±6,7	0,195
PWD 1	45±7	46±10	0,578
PWD 2	35 (20-45)	40 (27-59)	0,081
PWD 3	33±7,2	46,1±7,8	<0,001
MVA 1	1±0	1±0	0,968
MVA 2	2 (2-3)	2 (2-2)	0,019
max grad 1	24 (12-46)	22 (8-34)	0,379
max grad 2	11 (5-24)	13 (7-32)	0,194
mean grad 1	15±7	14±5	0,788
mean grad 2	5 (2-13)	5 (4-18)	0,028
PAPs 1	45 (30-110)	53 (35-120)	0,406
PAPs 2	35 (25-62)	35 (30-120)	0,577
LA 1	44 (34-69)	47 (37-62)	0,020
LA 2	42±5	42±2	0,568

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-043

Link between epicardial fat thickness and time to conversion of recent-onset atrial fibrillation to sinus rhythm with amiodarone therapy

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Background and Aim: Recent-onset atrial fibrillation (AF) is a frequent cause for presentation to the emergency department. Clinical studies have demonstrated that epicardial fat (EPF) thickness is associated with the presence, severity, and recurrence of AF. There is no study investigates to predictors of the time to conversion of AF to sinus rhythm with amiodarone therapy. The aim of this study was to investigate predictors of time to conversion of AF to sinus rhythm in patients with recent-onset AF during cardiovascular emergency phase.

Methods: All 102 patients admitted to the emergency room with symptomatic recent-onset AF (lasting <48 h), were hemodynamically stable, were registered consecutively. Patients received an intravenous amiodarone doses of 150 mg over 10 minutes, with a subsequent infusion of 1mg/minute for six hours, then 0.5 mg/minute for 36 hours. Those who converted to sinus rhythm within the first 48 hours after the therapy were included. Finally 82 patients were assigned in our study. Time taken from the beginning of drug administration to conversion to sinus rhythm were recorded. EPF thickness was measured using 2-D echocardiographic parasternal long-axis views. Patients were divided into two group according to conversion duration; Group 1 had shorter conversion time (<12 h) and Group 2 had longer conversion time (>12 h).

Results: Median age was 62 years (interquartile range (IQR) 54-69 years). The mean initial heart rate was 138±21 bpm. Median admission time to hospital was 420 min (IQR 150-840 min) and time to conversion to sinus rhythm after the amiodarone therapy was 230 min (IQR 60-720 min). No significant differences were observed in the baseline characteristics of the two groups (Table 1). Mean EPF thickness were higher in group 2 compared to group 1 (4.6±1.25 mm vs. 5.2±1.4 mm, p=0.04). Also, a significant positive correlation was found between EPF thickness and conversion duration ($\rho = 0.307, p=0.005$) in all patients (Figure 1). Multivariate logistic regression analysis (including age, sex, diabetes mellitus, hypertension, LVEF, BMI, left atrial diameter, number of AF attack, duration of AF before the treatment, EPF thickness, C-reactive protein, troponin I) showed that EPF thickness (p=0.022, OR:2.9, 95% CI:1.1-7.6), admission troponin I level >0.01 (p=0.024, OR:27, 95% CI: 1.5-48.7), lower age (p=0.018, OR: 0.7, 95% CI: 0.6-0.9), female sex (p=0.039, OR: 139, 95% CI: 1.2-14.6) and low LVEF (P=0.039, OR: 0.6 95%CI: 0.4-0.9) were significantly associated with longer conversion time.

Conclusions: Our study is the first to evaluate to conversion duration by amiodarone therapy of recent-onset atrial fibrillation. We found that the EPF thickness were associated with time to conversion of sinus rhythm in patients with recent-onset AF EPF thickness may be used in prediction of the longer time to conversion. Longer conversion times extend the in-hospital stay length, which can affect medical care and costs.

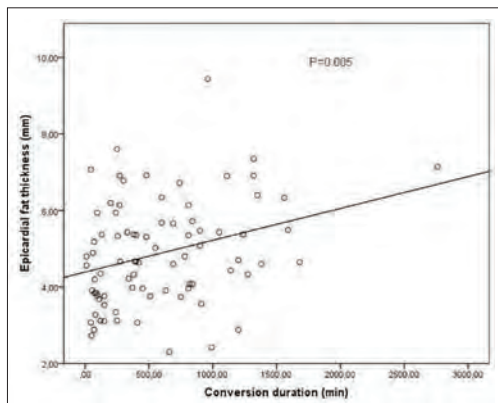


Figure 1. Correlation between EPF thickness and conversion duration ($\rho = 0.307, p=0.005$).

Table 1. Baseline and clinical variables

Variables	Group 1 (time to conversion <12 h)	Group 2 (time to conversion >12)	P value
Age (years)	61±10	57±12	0.113
Male sex (%)	25(46)	13(45)	0.898
CHA2DS2-VASc Score	2(1-3)	2(0.2-3)	0.327
Initial heart rate (bpm)	137±21	140±21	0.589
Hypertension (%)	32(59)	18(62)	0.803
Diabetes mellitus(%)	12(22)	5(17)	0.592
Coronary heart disease (%)	3(5)	0(0)	0.196
CRP level (mg/dl)	0.6±0.8	0.5±0.8	0.966
Troponin I level (ng/mL)	0.01±0.2	0.07±0.1	0.141
LVEF(%)	66±5	65±3	0.724
Left atrial diameter(mm)	35.2±4.8	36.0±6.5	0.529
BMI(kg/m2)	30.1(27.1-36.7)	29.6(27.3-33.8)	0.712
Epicardial fat thickness (mm)	4.6±1.2	5.2±1.4	0.040

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-044

A safe and rapid technique for pacemaker implantation: Roadmap guided subclavian vein puncture

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Background and Aim: Widely used method is blinded puncture of subclavian vein, but complication rate is high in this method. In this study, we aimed to demonstrate the effect of roadmap use during implantation of permanent pacemaker on the success rate, speed of puncture and complications.

Methods: The study was designed as a prospective randomized controlled study. Totally 125 devices were implanted to the patients included in the study, and 518 punctures were performed for implantation of these devices. 186 punctures were performed in roadmap group and 332 punctures were performed in conventional group. Two groups were compared with regard to clinical and demographic features, speed and success of puncture and complications.

Results: Baseline characteristics were similar between groups. Median duration of intervention for each puncture was 27 (15/46) seconds in roadmap group and 56 (30/100) seconds in conventional group. Number of attempts for a successful puncture was detected as 1 (1/2) in roadmap group and 2 (2/4) in conventional group. Arterial puncture incidence was 10.3% in roadmap group and 37% in conventional group. (p<0.001 for all) Considering complications, incidence of pneumothorax and intramuscular puncture was seen lower significantly (p=0.046 and p=0.006, respectively).

Conclusions: In conclusion, number of attempts for successful puncture, time needed for successful puncture, number of arterial puncture and complication rate was significantly lower in patients undergoing pacemaker implantation by roadmap technique. Based on these data, roadmap technique may take the place of conventional method of puncture.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-045

Impact of cardiac rehabilitation on ventricular repolarization indexes in patients with rheumatid arthritis

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Background and Aim: Abnormalities in ventricular repolarization (VR) parameters have been associated with sudden cardiac death (SCD) in patients with rheumatoid arthritis (RA). The benefits of cardiac rehabilitation (CR) in patients with RA are well recognized. We aimed to assess its impact on VR indexes in patients with RA.

Methods: This study included 45 patients with RA (36 female, age 58±5.5 years) and 50 age- and sex-matched otherwise healthy controls. Baseline electrocardiogram (ECG) recordings were used to compare VR parameters such as maximum and minimum QT intervals, and corrected, and dispersion (QTmax, QTmin, cQTmax, cQTmin, QTd, cQTd, respectively), JT and cJT intervals, Tp-e and cTp-e intervals, and Tp-e/QT and Tp-e/cQT ratios in patients with RA and healthy individuals. The effects of 6-week CR in patients with RA were also evaluated by comparing pre- and post-CR ECGs, exercise tolerance test (MET and VO2max) and RA characteristics (C-reactive protein (CRP), Disease Activity Score 28 (DAS28) and Health Assessment Questionnaire(HAQ)).

Results: In comparison with the healthy individuals, the patients with RA had significantly higher cQTmax and QTmin intervals, QTd, cQTd, Tp-e and cTp-e intervals, and Tp-e/QT and Tp-e/cQT ratios. At the end of CR, all VR indexes (p<0.05), except QTd, were significantly decreased as did the results for CRP, DAS28, and HAQ (all p<0.05), and MET and VO2max (p<0.05 for both) were significantly increased in patients with RA.

Conclusions: CR may provide an improvement in the majority of VR indexes which are related with ventricular arrhythmia and SCD in patients with RA. Changes in ETT parameters and RA characteristics may contribute to improvement of several VR indexes such as cQTd, cJT and Tp-e intervals at the end of CR.

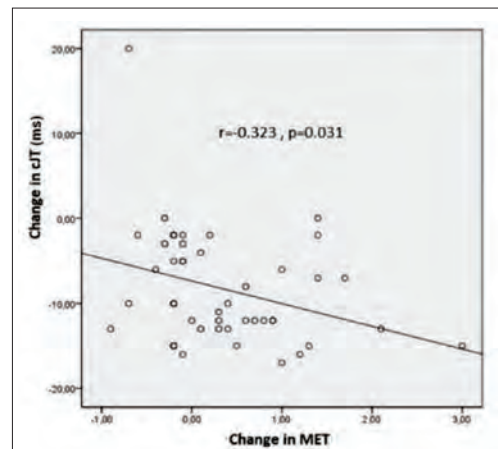


Figure 1. Significant negative correlation between changes in MET and cJT interval in the patients with RA receiving cardiac rehabilitation.

Table 1. Comparison of VR indexes between patients with RA and healthy controls, and the changes in VR indexes in patients with RA receiving CR

	Patients with RA (n= 45)		Patients with RA (n= 45)	Patients with RA (n= 45)	Patients with RA (n= 45)	Controls (n= 50)	P value (Between groups)
	Pre- CR	Post- CR					
QTmax (ms)	391.4±39	371.1±33.2	-20.3±37.2	-6.1	0.001	384.6±36.5	0.066
cQTmax (ms)	429.8±43.3	402.8±46.7	-27±38.5	-6.6	<0.001	405.1±39.5	0.018
QTmin (ms)	369.3±28.2	349.8±30	-19.5±28.1	-5.4	0.001	351.8±31	0.003
cQTmin (ms)	375.2±31.7	354±32.1	-21.1±21	-5.7	<0.001	370.7±37.5	0.537
QTd (ms)	43.1±36.6	38.2±37.6	-7.9±14.2	-15.6	0.055	33.7±38.5	0.015
cQTd (ms)	48.8±39.4	37.8±33.8	-11.5±23.5	-19.8	0.046	36.7±38.1	0.002
JT (ms)	278.1±44.7	267.6±46.6	-25.5±27.6	-8.1	0.003	270.8±48.6	0.038
cJT (ms)	298.2±45.6	279.8±47.6	-28.5±46.5	-9.3	<0.001	285.1±42.5	0.001
Tp-e (ms)	78.4±17.9	71.5±16.9	-7±18.9	-8.4	0.001	70.3±17.4	<0.001
cTp-e (ms)	87.1±17	79.8±16.1	-7.4±11.2	-8.7	<0.001	78.3±17.4	<0.001
Tp-e/QT	0.24±0.02	0.22±0.02	-0.02±0.01	-7.5	<0.001	0.22±0.03	<0.001
Tp-e/cQT	0.23±0.02	0.21±0.02	-0.01±0.01	-5.6	<0.001	0.21±0.03	<0.001

Values are presented as means ± standard deviations and medians with interquartile range in parentheses. RA: rheumatoid arthritis; CR: cardiac rehabilitation; QTmax: maximum QT interval; cQTmax: corrected maximum QT interval; QTmin: minimum QT interval; cQTmin: corrected minimum QT interval; QTd: QT dispersion interval; cQTd: corrected QT dispersion interval; JT: JT interval; cJT: corrected JT interval; Tp-e: transmural dispersion of repolarization interval; cTp-e: corrected transmural dispersion of repolarization interval.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-046

Microvolt T-wave alternans in young myocardial infarction patients with preserved cardiac function treated with single vessel primary percutaneous coronary intervention

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Background and Aim: The current study assessed microvolt T-wave alternans in young patients who had ST segment elevation myocardial infarction with preserved left ventricular function who underwent single vessel revascularization.

Methods: We enrolled 108 consecutive patients (age: 39.5±4.1) with ST segment elevation myocardial infarction (STEMI) undergoing percutaneous coronary intervention and 43 patients (age: 38.5±3.7) with normal coronary angiograms as a control group. The myocardial infarction patients were younger than 45 and had a preserved left ventricular ejection fraction. They were divided into three groups according to the culprit artery. The microvolt T-wave alternans (MTWA) values were calculated an average of 12 months after the primary percutaneous coronary intervention using the modified moving average method.

Results: MTWA positivity was significantly higher in STEMI group compared to the controls (p<0.001). It was also significantly higher in STEMI patients with left anterior descending artery lesions compared to patients with circumflex artery and right coronary artery lesions (p=0.013). Moreover, the culprit artery and neutrophil/lymphocyte ratio were independent predictors of MTWA positivity (p=0.043 and p=0.018, respectively). **Conclusions:** In STEMI patients of a young age, MTWA positivity was higher than in healthy individuals, especially when the responsible vessel fed a wider region and the disease was accompanied by increased chronic inflammation.

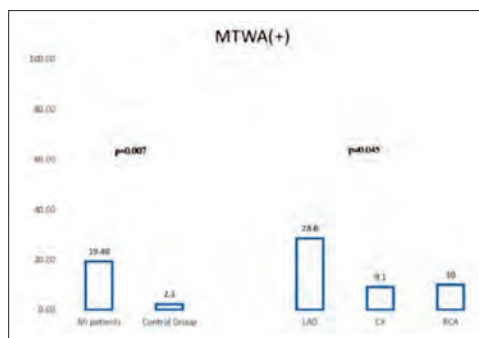


Figure 1. MTWA positivity in MI/control group and according to culprit artery in MI patients.

Table 1. Logistic Regression Analysis: Independent Predictors of MTWA Positivity in MI Patients

	Univariate analysis OR (CI 95%)	p value	Multivariate analysis OR (CI 95%)	p value
Age, years	0.936 (0.844-1.038)	0.210		
Sex(Male)	1.315 (0.609-2.840)	0.485		
Hypertension	2.747 (0.905-8.335)	0.104		
Hyperlipidemia	1.197 (0.427-3.335)	0.732		
Diabetes Mellitus	3.538 (0.426-29.32)	0.242		
Smoking	0.388 (0.124- 1.214)	0.104		
Culprit artery	2.760 (1.264-6.027)	0.011	3.325 (1.040-10.635)	0.043
LVEF	0.904 (0.834- 0.980)	0.014	0.982 (0.895-1.077)	0.699
Hematocrit	1.455 (0,556-3.802)	0.445		
WBC	1.166 (1.016-1.388)	0.108		
Creatinine	4.646 (0.331-65.247)	0.255		
N/L ratio	1.051 (0.999-1.105)	0.048		

LVEF: left ventricular ejection fraction; N/L ratio: neutrophil/lymphocyte ratio; WBC: white blood cell; CI: confidence interval; OR: odds ratio

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-048

Heart rate variability in patients with acute submassive pulmonary embolism

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Background and Aim: Sudden cardiac death is a major cause of mortality in patients with pulmonary hypertension. Heart rate variability is a validated method to evaluate cardiac autonomic system dysfunction and alterations in heart rate variability are used to predict arrhythmic events. The aim of this study was to evaluate heart rate variability in patients with submassive acute pulmonary embolism.

Methods: Twenty-five patients with confirmed submassive acute pulmonary embolism and 25 normal subjects were consecutively enrolled. All patients underwent 24-hour holter ECG recording for heart rate variability assessment by time- and frequency-domain analysis. The left and right ventricular functions of the patients and controls were evaluated by speckle tracking echocardiography.

Results: The characteristics and heart rate variability parameters of the patients and controls are listed in Table 1. Although there were not any significant differences in age, sex and ventricular functions of the patients, heart rate variability parameters were significantly lower in patients with acute pulmonary embolism compared to controls.

Conclusions: Although there were not apparent differences in ventricular functions, acute pulmonary embolism patients had reduced heart rate variability, which might be indicating a high risk for malignant arrhythmic events.

Table 1.

	Pulmonary Embolism (n= 25)	Controls (n= 25)	p
Age (years)	58.0 ±13.9	55.4 ± 16.1	0.152
Male sex (n-%)	13 (52.0%)	8 (32.0%)	0.668
LVEF (%)	53.1 ± 6.1	56.2 ± 3.8	0.257
LV GLS (%)	- 19.2 ± 2.5	- 20.1 ± 3.8	0.232
RV GLS (%)	- 18.4 ± 3.2	- 19.7 ± 2.8	0.127
RR (ms)	736.1 ± 105.6	834.0 ± 103.0	0.003
SDNN (ms)	98.3 ± 35.1	115.4 ± 31.4	0.017
SDANN (ms)	88.1 ± 36.8	108.4 ± 21.0	0.010
SDNNindex (ms)	38.9 ± 15.6	46.8 ± 18.0	0.079
pNN50 (%)	5.7 ± 11.8	9.9 ± 18.4	0.063
RMSSD (ms)	24.5 ± 16.1	32.2 ± 25.1	0.248

LVEF: Left ventricular ejection fraction, LV GLS: Left ventricular global longitudinal strain, RV GLS: Right ventricular global longitudinal strain, SDNN: Standard deviation of all normal R-R intervals, SDANN: Standard deviation of all the 5-minute R-R interval means, pNN50: Percentage of adjacent RR intervals >50 ms difference, RMSSD: Root of mean squared differences of successive R-R intervals

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PP-049

The relations of Brugada ECG pattern and fragmented QRS in patient with schizophrenia: A cross-sectional study in Turkish population

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Background and Aim: Schizophrenia is a psychiatric disease with high risk of fatal rhythm disorders and sudden cardiac death. A previous study reported that Brugada syndrome was highly prevalent (11.6%) in patients with schizophrenia. In this study we aimed to investigate the prevalence of Brugada syndrome and fragmented QRS in patients with schizophrenia in Turkish population.

Methods: Two hundred and fifty patients with schizophrenia who were followed up in the psychiatry clinic, and 400 age- and sex-matched non schizophrenia controls were included. Standard electrocardiography (ECGs) and high intercostal ECGs with V1 and V2 derivation above two intercostal intervals were taken. All ECGs were screened for ECG characteristics and Brugada syndrome by a cardiologist blind to patient diagnosis.

Results: In schizophrenia patients, Type 1 Brugada syndrome was not observed, Type 2 Brugada was identified in one subject and Type 3 Brugada was observed in two patients. In the control group, Type 2 Brugada pattern was identified in one subject and again one control had Type 3 Brugada pattern (p=0.320). The fragmented QRS (fQRS) incidence, QRS duration and corrected QT were observed to be higher in the schizophrenia group compared to the control group (p=0.001, p=0.003, p<0.001, respectively).

Conclusions: There is no increased prevalence of Brugada-ECG pattern in patients with schizophrenia in the Turkish population. Importantly, the prevalence of fQRS was significantly higher in patients with schizophrenia compared to the control group.

Table 1. Demographic, clinical, laboratory, renal and liver ultrasound findings of patients with and without diastolic dysfunction

Variable	Group I n=67	Group II n=72	P
Age (year)	55.2 ± 7.3	55.9 ± 6.9	0.556
Gender (female)	41	41	0.730
Hypertension, n (%)	23 (34%)	22 (31%)	0.718
Diabetes mellitus, n (%)	24 (36%)	23 (33%)	0.742
Current smoker, n (%)	15 (22%)	18 (25%)	0.436
Hyperlipidemia, n (%)	24 (36%)	18 (25%)	0.114
Obesity, n (%)	15 (22%)	19 (26%)	0.694
Systolic blood pressure (mmHg)	130 ± 11	128 ± 10.2	0.145
Diastolic blood pressure (mmHg)	86 ± 8	84 ± 9	0.152
Basal heart rate (pulse/minute)	78 ± 11	79 ± 11	0.774
Body mass index (kg/m ²)	28.0 ± 2.2	28.5 ± 2.4	0.184
Body mass index (kg/m ²)	28.0 ± 2.2	28.5 ± 2.4	0.184
Fasting plasma glucose (mg/dL)	112 ± 45	104 ± 28	0.464
Blood urea nitrogen (mg/dL)	29 ± 6.6	28 ± 10	0.242
Creatinine (mg/dL)	0.68 ± 0.15	0.67 ± 0.18	0.738
Total Cholesterol (mg/dL)	146 ± 44	139 ± 40	0.390
Low density lipoprotein (mg/dL)	128 ± 34	119 ± 35	0.128
High density lipoprotein (mg/dL)	45 ± 8.7	47 ± 8.3	0.386
Triglyceride (mg/dL)	176 ± 121	157 ± 102	0.408
Aspartate aminotransferase (u/L)	21.1 ± 7.6	20.6 ± 4.8	0.678
Alanine aminotransferase (u/L)	19.3 ± 10.2	18.6 ± 7.8	0.685
Caudal to cranial liver size (cm)	14.8 ± 2.4	14.5 ± 2.1	0.516
Liver echogenicity Grade 0-I-II-III, n	22-26-17-2	32-24-15-1	0.042
Liver stiffness (kPa)	6.21 ± 3.17	4.94 ± 2.88	0.015
Liver stiffness > 6 kPa, n (%)	35 (52%)	23 (33%)	0.019

Group I: Subjects with diastolic dysfunction, Group II: Patients without diastolic dysfunction

Cardiac imaging / Echocardiography

PP-050

The diastolic dysfunction that occurs in patients with cardiovascular risk factors is closely related to liver tissue stiffness: Echocardiography and elastography point quantification study

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Background and Aim: Heart and liver are vascular organs, that cardiovascular (CV) risk factors such as advanced age, diabetes mellitus, hypertension, hyperlipidemia and obesity negatively affect these organs in a similar way. The most important cause of left ventricular diastolic dysfunction (LV-DD) is ventricular interstitial fibrosis and remodeling. Similar to strain echocardiography, liver elastography studies provide information on tissue function and stiffness. The aim of this study was to evaluate the relationship between LV-DD and liver tissue stiffness (LSM) in patients with at least 1 CV risk factor.

Methods: Patients who were referred to our department for endocrinology and who had at least 1 CV risk factor were included in this study. Anamnesis and physical examination were performed for all subjects. Echocardiography evaluation was performed in the Department of Cardiology. LSM was calculated with elastography point quantification technique (ElastPQ) in conjunction with conventional liver ultrasonography (USG) for all patients. All patients included in the study were grouped as LV-DD and non-LV-DD according to echocardiography results.

Results: LV-DD was present in 69 (49, 6%) of the subjects studied. The demographic, clinical and laboratory parameters of patients with and without LV-DD are similar. When the liver USG findings are examined; USG revealed that liver size was similar between the two groups (Table). It was observed that the degree of liver echogenicity was higher in patients with LV-DD. When liver elastography was examined, it was found that patients with LV-DD had higher LSM. When the limit value for the LSM increase obtained from previous studies was taken as 6.0 kPa, it was found that LSM was more (in half) in patients with LV-DD.

Conclusions: LV-DD, which is associated with cardiovascular risk factors and it is closely related to the increased LSM in the same patient group. As a result of our study, heart and liver are similarly affected by cardiovascular diseases. While assessing the effects of systemic diseases on the heart, it was concluded that it would be advisable to think that it should be evaluated in other target organs in the same group of patients.

Cardiac imaging / Echocardiography

PP-051

Value of tissue Doppler imaging and peak acceleration time in prediction of paroxysmal atrial fibrillation in patients with paroxysmal atrial fibrillation

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Background and Aim: Atrial fibrillation is the most common arrhythmia in the population and its prevalence increases with age; and also is the most morbid and mortal arrhythmia. Usually the beginning of the persistent atrial fibrillation is recurrent episodes of the paroxysmal atrial fibrillation (PAF). Prediction of the paroxysmal atrial fibrillation can cause prevention of this arrhythmia and thus prevention of the adverse outcomes. We aimed to investigate tissue Doppler imaging (TDI) and peak acceleration time (pkAcc) parameters that can predict the paroxysmal atrial fibrillation in this study.

Methods: 20-73 years old (mean 47.5) 50 individuals that are diagnosed with PAF included the patient group, 50 individuals who have the similar baseline demographic characteristics with patient group and who have no persistent or PAF included the control group.(Table-1) Transthoracic echocardiographic (TTE) evaluation is applied all of the control and study groups. Tissue Doppler parameters and pkAcc is measured in TTE and statistical analyses is performed.

Results: In TTE evaluation, left atrium ejection fraction is lower in the patient group than the study group (50.6% vs. 59.2%, p<0.001) (Table 2). In TDI evaluation, the average of E/E' which was measured from the anterior, inferior, lateral and septal walls of the left ventricle; is found higher in the patient group compared to the control group (8.17 vs. 7.04; p=0.004) (Table 3). When two groups are compared in terms of pkAcc, it was found that patient group is higher, but this difference did not reach the statistical significance (1063 vs. 994, p=0.14).

Conclusions: TDI evaluation can play an important role in prediction of paroxysmal atrial fibrillation. The E/E' parameter used for predicting diastolic functions can also be used as a rapid, non-invasive and economical method for predicting paroxysmal atrial fibrillation in people with sinus rhythm. For this purpose, there is need for larger, randomized controlled studies in order to determine its use.

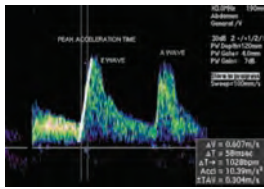


Figure 1. Measurement of pkAcc on Mitral E wave by PW-Doppler.

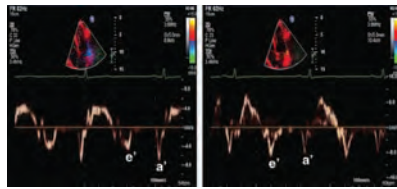


Figure 2. Mitral septal and lateral e' and a' waves from a patient's tissue-Doppler imaging (TDI).

Table 1. Baseline demographic characteristics of the groups

	Patient (n=50)	Control (n=50)	P value
Height (cm)	167	166	0,92
Weight (kg)	79,6	77,7	0,75
BMI	28,3	28,2	0,94
Age (years)	46,3	44,2	0,80
Female/male	23/27	27/23	0,64
Hypertension	17	14	0,42
Diabetes	9	8	0,79
Smoking	12	11	0,37
Beta-bloker	16	12	0,79
ACE-i+ARB	11	9	0,21
CCB	8	4	0,17
Urea(mg/dL)	26,5	24,9	0,08
Creatinine(mg/dL)	0,83	0,80	0,27
Sodium(mEq/L)	139,4	139,6	0,72
Potassium(mEq/L)	4,23	4,32	0,24
Total cholesterol(mg/dL)	190	187	0,70
Triglyceride(mg/dL)	163	150	0,35
HDL cholesterol(mg/dL)	38,7	41,9	0,06
LDL cholesterol(mg/dL)	118	114	0,53
WBC(x1000/uL)	8,33	8,28	0,91
Hemoglobin(g/dL)	14,0	13,5	0,06
TSH(uIU/mL)	1,81	2,09	0,11
FT3(uIU/mL)	2,68	2,67	0,88
FT4(uIU/mL)	1,41	1,49	0,22

BMI: body mass index, ACE-i: angiotensin-converting-enzyme inhibitor, ARB: angiotensin receptor blocker, CCB: calcium channel blocker, HDL: high-density lipoprotein, LDL: low density lipoprotein, WBC: White blood cell, TSH: thyroid-stimulating hormone, FT3: free triiodothyronine, FT4: free thyroxine

Table 2. Comparison of echocardiographic parameters in patient and control groups

	Patient group (n=50)	Control group (n=50)	P value
LVEDD (mm)	48,1	47,5	0,45
LVESD (mm)	30,6	30,4	0,76
IVS wall thickness (mm)	9,6	9,0	0,007
PW thickness (mm)	9,0	8,4	0,016
Left ventricle mass (gr)	179,9	163,5	0,06
Ejection fraction (%)	65,8	65,1	0,48
Fractional shortening (%)	36,3	35,8	0,48
Left atrial end-diastolic volume (mL)	41,8	36,4	0,003
Left atrial end-systolic volume (mL)	20,3	14,5	<0,001
Aortic diameter (cm)	2,88	2,73	0,01
LVOT diameter (cm)	2,26	2,17	0,026
LVOT ejection time (msn)	292	287	0,41
Stroke volume (mL)	85,3	77,7	0,051
Heart rate (/dk)	72,9	74,5	0,45
Cardiac output (L/dk)	6,18	5,76	0,12
E/A ratio	1,04	1,18	0,03
pkAcc	1063	994	0,14
E deceleration time (msn)	166,6	166,9	0,95

LVEDD: left ventricle end-diastolic diameter, LVESD: left ventricle end-systolic diameter, IVS: interventricular septum, PW: posterior wall, LVOT: left ventricular outflow tract, pkAcc: peak acceleration time

Table 3. Comparison of TDI parameters in patient and control groups

	Patient group (n=50)	Control group (n=50)	P value
E/E' ratio (anterior)	8,83	7,52	0,014
E/E' ratio (lateral)	6,98	6,50	0,18
E/E' ratio (septal)	8,68	7,77	0,02
E/E' ratio (inferior)	8,17	6,36	<0,001
E/E' ratio (tricuspid)	5,84	5,65	0,53
E/E' ratio (left ventricle average)	8,17	7,04	0,004

Cardiac imaging / Echocardiography

PP-052

Echocardiographic findings in patients with atrial septal aneurysm: A prospective case control study

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Background and Aim: Atrial septal aneurysm (ASA) is a localized pathology of interatrial septum with a prevalence of 1-2% in adult population. Although ASA has been supposed to be an incidental finding in echocardiographic examination, its structural and clinical associations have gained an increasing interest. In this study, we aimed to compare the clinical features and echocardiographic parameters between ASA patients and age- and gender- matched control group patients.

Methods: 410 patients with ASA were enrolled to the study, prospectively. After the exclusion of 33 patients, the remaining 377 patients comprised the study group. The control group consisted of 377 age and gender matched patients without ASA.

Results: Aortic valve regurgitation and mitral valve regurgitation were more often observed in patients with ASA and percentages of patients with ascending aortic aneurysm (AAA), patent foramen ovale (PFO) and atrial septal defect were higher in ASA patients compared to control group patients. Aortic root diameter was larger in ASA patients compared to control group patients (29.2±3.9, 28.6±3.1, p=0.05, respectively). Logistic regression analysis revealed that mitral valve regurgitation (OR:2.05, 95% CI:1.44-2.92, p<0.001), AAA (OR:2.69, 95% CI:0.97-7.47, p=0.05) and PFO (OR: 11.62, 95% CI:2.64-51.02, p=0.001) were positively and independently associated with presence of ASA.

Conclusions: We have demonstrated that ASA is significantly and positively associated with mitral regurgitation, PFO and AAA. This association highlights the possible role of connective tissue disorders as an underlying etiology in patients with ASA. Further clinical and pathological studies are warranted to elucidate the pathophysiological associates or contributors of ASA.

Pulmonary hypertension / Pulmonary vascular diseases

PP-053

Can fibrosis-4 index be a marker of severity in acute pulmonary embolism?

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Background and Aim: Acute pulmonary embolism (APE) is characterized by numerous clinical manifestations which are the result of a complex interplay between different organs. Identifying high-risk patients as soon as possible is crucial for diagnosis and therapy. The existence of right heart failure, haemodynamic decompensation and several indices such as pulmonary embolism severity index (PESI) are used for identifying patients at high risk. FIB-4 index is simple and noninvasive markers, were originally developed for predicting the degree of liver function deterioration in mainly chronic and also acute setting. In this study we aimed to investigate whether FIB-4 index can be used for identifying high risk patients in acute pulmonary embolism.

Methods: Seventy-seven patient (44 F, 33 M, mean age; 63.2±17.2) who admitted to the emergency service with the signs of APE were included to the study. A detailed medical history, physical examination, vital signs, situations predisposing to PE, PESI score were recorded (Table 1). PE were diagnosed according to computed tomography angiography. Biochemical and haematological parameters, high sensitive troponin, were measured at the admission. Transthoracic echocardiography was also performed. In addition to conventional parameters, ejection fraction, tricuspid annular plane systolic excursion was calculated. Patients were divided into two group according to existence of shock and hypotension. FIB-4 index was calculated as follows: age (years) x AST (IU/l) /platelet count (x109/l) x√ALT (IU/l).

Results: Demographic characteristics and situations predisposing to PE were similar between the groups. FIB-4 index was significantly higher in group 1 compared to group 2 (2.14±1.07vs1.53±0.91; p=0.18). FIB-4 index was significantly positively correlated with age, PESI score, high sensitive troponin levels, pulmonary artery systolic pressure (Table 2). FIB-4 index was significantly negatively correlated with diastolic systolic pressure and TPAPSE (Table 2). There was no correlation between FIB-4 with systolic blood pressure and ejection fraction (Table 2).

Conclusions: Patients with pulmonary embolism with shock or hypotension have higher FIB-4 index. PESI index, laboratory and echocardiographic markers which show the severity of APE are correlated with the index. In our opinion FIB-4 index which is a noninvasive marker of liver function deterioration might be used for classifying the high risk patients in APE.

Table 1. Characteristics of the groups

	Group 1 (n=20)	Group 2 (n=57)	P
Age (years)	69.1±14	61.2±17.6	0.075
Gender (F, n)	8	36	0.42
PESI score	130.6±27.4	88.6±30.2	<0.001
Systolic blood pressure (mm Hg)	83.2±8.8	131.0±18.6	<0.001
Diastolic blood pressure (mm Hg)	54.3±12.2	77.6±11.3	<0.001
Hypertension (n)	15	31	0.10
Diabetes mellitus (n)	8	18	0.49
Coronary artery disease (n)	5	6	0.11
Smoking (n)	8	16	0.32
Deep venous thrombosis (n)	6	15	0.75
Pregnancy (n)	0	1	0.55
History of operation (n)	5	17	0.68
Immobility (n)	9	21	0.52
History of stroke (n)	2	4	0.67
History of cancer (n)	5	15	0.90

Table 2. Correlation analysis between the FIB-4 index and PESI index, laboratory, echocardiographic parameters

	Age	PESI	SBP	DBP	hsTTP	D-dimer	EF	TAPSE	PABs
r	0.70	0.56	-0.15	-0.23	0.28	0.18	-0.15	-0.45	0.30
p	<0.001	<0.001	NS	0.05	0.015	NS	NS	0.015	0.01

Pulmonary hypertension / Pulmonary vascular diseases**PP-054**

Pulmonary hypertension spectrum: 16 years of experience of a single center

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Background and Aim: Pulmonary hypertension (PH) is multidisciplinary disorder that should be diagnosed and treated by specialized centers. The progress in the field of PH diagnosis also leads to new classifications of the disease. The aim of this study is to determine the etiological properties of PH to provide information about diagnosis.

Methods: The database of the right heart catheterization laboratory was retrospectively searched. All patients who were diagnosed with PH were included to registry. Patients were classified according to their etiologies. Classification of recent guidelines were applied to data. RHC and echocardiography findings were also recorded.

Results: 379 patients with PH (231 female, 53.2±14.74 yr) were included to PH registry. Of the 379 patients enrolled, 82 patients were classified as pulmonary arterial hypertension (PAH) – Group 1 PH (21.6%), 278 patients (74.4%) were classified as Grup 2 PH. The leading cause in PAH subgroups were congenital heart diseases. Valve diseases were the most common cause of post-capillary PH (See Table 1). There was statistically important difference in mean and systolic pulmonary artery pressures and left ventricular ejection fraction among groups, however no difference was observed for right atrial pressures and diastolic pulmonary artery pressures.

Conclusions: Our registry showed that the leading causes are congenital heart diseases and valve diseases for the PAH and post capillary PH respectively. It can also be conceived that patients with congenital heart disease were diagnosed lately as they developed PH. It is evident that PH registries must be performed to provide clinical information for management of PH.

Table 1. Distribution of the patients according to clinical classification of pulmonary hypertension

Clinical Classification	Number
Group 1: Pulmonary arterial hypertension	82
Group 1.1 Idiopathic	13
Group 1.4.1 Associated with connective tissue disease	9
Group 1.4.4 Associated with congenital heart disease	60
Group 2: Pulmonary hypertension due to left heart disease	278
Group 2.1 Left ventricular systolic dysfunction	67
Group 2.2 Left ventricular diastolic dysfunction	14
Group 2.3 Valvular disease obstruction and congenital cardiomyopathies	190
Group 2.4 Congenital/acquired left heart inflow/outflow tract obstruction and congenital cardiomyopathies	1
Group 2.5 Congenital/acquired pulmonary veins stenosis	6
Group 3: Pulmonary hypertension due to lung diseases and/or hypoxia	6
Group 3.1 Chronic obstructive pulmonary disease	3
Group 3.2 Interstitial lung disease	3
Group 4: Chronic thromboembolic pulmonary hypertension and other pulmonary artery obstructions	8
Group 4.1 Chronic thromboembolic pulmonary hypertension	6
Group 4.2 Other pulmonary artery obstructions	2
Group 5: Pulmonary hypertension with unclear and/or multifactorial mechanisms	5
Group 5.1 Haematological disorders: chronic haemolytic anaemia, myeloproliferative disorders, splenectomy	3
Group 5.2 Systemic disorders: sarcoidosis, pulmonary histiocytosis, lymphangioleiomyomatosis	2

Cardiac imaging / Echocardiography**PP-055**

Impact of volume overload on left ventricle function evaluated by speckle tracking echocardiography

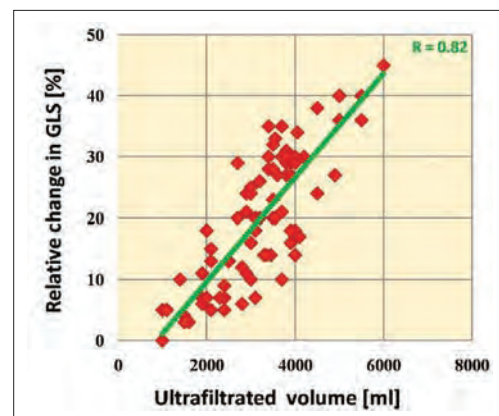
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Background and Aim: The aim of this study is to evaluate the impact of volume overload on main echocardiographic parameters used for assessment of left ventricle (LV) by examining end stage kidney patients before and after hemodialysis (HD).

Methods: Patients between 18 and 85 years of age, receiving HD therapy for at least 6 months were included. The echocardiographic images were obtained in 30 minutes before and after HD. 2D speckle tracking echocardiography was used for strain measurements. Global longitudinal strain (GLS) were calculated by obtaining average values from all apical views. Difference in echocardiographic parameters before and after HD, additionally correlation between ultrafiltrated volume and the relative change in echocardiographic parameters were calculated.

Results: 70 patients (50.7±16.9 years of age, 27 women) were included in study. The average ultrafiltrated volume was 3260±990 ml. The chamber sizes of LV and left atrium (LA) decreased after HD (LV end-diastolic diameter (cm) 4.4±0.7 vs. 4.0±0.8, LV end-diastolic volume (ml) 75.3±22.7 vs. 68.5±29.7, LA diameter (cm) 5.1±1.4 vs. 4.7±1.4, LA area (cm²) 29.9±10.6 vs. 19.9±10.3, all p<0.001). 2D speckle tracking echocardiographic analysis showed that; LV GLS decreased after HD whereas LV EF remained unchanged (LV GLS (%) 20.1±3.6 vs. 16.8±3.8, p<0.001, LV ejection fraction (%) 66.0±11.5 vs. 64.2±10.6, p=0.071). The relative change in LV GLS significantly correlated with ultrafiltrated volume (Figure 1).

Conclusions: A strong correlation was present between relative change of LV GLS and ultrafiltrated volume. Rapid volume depletion substantially affected myocardial contractility. The rate of ultrafiltration should be minimized to avoid possible negative consequences.

**Figure 1.** Comparison between relative change of global longitudinal strain measurements and the ultrafiltrated volume. The correlation coefficient (R) is indicated. P<0.001 (GLS: global longitudinal strain).**Cardiac imaging / Echocardiography****PP-056**

Aortic arch calcification depicted on chest radiography is strongly associated with metabolic syndrome in patients with obstructive sleep apnea

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Background and Aim: Obstructive sleep apnea (OSA) is associated with increased atherosclerosis and metabolic syndrome. Vascular calcification has a pivotal role in development of atherosclerosis. Data is insufficient about vascular calcification and metabolic syndrome (MS). The aim of the present study was to investigate relationship between metabolic syndrome and aortic arch calcification (AAC), as a prototyp of vascular calcification, in patients with OSA.

Methods: Patients who underwent an overnight polysomnography and diagnosed as OSA were enrolled to the study and were divided according to presence of metabolic syndrome. Two examiners who were unaware the result of the polysomnographic assessment reviewed the chest radiograms.

Results: 314 patients with OSA were included in the study (33% female, mean age 51.22±10.95). Of these, 44% had mild OSAS, 31% had moderate OSAS and 26% had severe OSA. AAC was detected in 56% (n=177). MS was detected in 59% (n=184). Prevalence of AAC was higher in patients with MS than without MS. In MS group; prevalence of hypertension, diabetes, hyperlipidemia and body mass index, waist circumference, neck circumference, systolic and diastolic blood pressure were found significantly higher than non-MS group. However; serum HDL level, LVEF and GFR measurements in the MS group were significantly lower than the non-MS group. In the multivariate logistic regression analysis body mass index (1.148 (95% CI: 1.089-1.210)), Apnea hypnea index (1.039 (95% CI: 1.016-1.062)) and AAC were found to be (4.986 (95% CI: 2.887-8.610)) a predictor for MS.

Conclusions: AAC on chest radiography is strongly associated with metabolic syndrome in patient with OSA. AAC may alert clinicians for development of metabolic syndrome in patients with OSA.

Table 1. Baseline characteristics of the study groups

	Metabolic Syndrome No (n=130)	Metabolic Syndrome Yes (n=184)	P value
Age (year)	50 ± 11	52 ± 11	0,193
Female gender (n, %)	43 (41)	62 (59)	0,909
Body mass index (kg/m ²)	30 ± 6	35 ± 6	0,001
Waist circumference (cm)	105 ± 15	116 ± 13	0,001
Neck circumference (cm)	39 ± 4	41 ± 4	0,001
Smoking (n, %)	35 (42)	48 (58)	0,869
Hypertension, n (%)	47 (31)	103 (69)	0,001
Diabetes mellitus; n (%)	23 (30)	55 (70)	0,014
Hyperlipidemi (n, %)	4 (18)	18 (82)	0,022
Systolic blood pressure, mm Hg	120 ± 14	128 ± 16	0,001
Diastolic blood pressure, mm Hg	73 ± 10	77 ± 10	0,001
Apnea Hypopnea Index (events/h)	5-50 (11)	5-88 (20)	0,001
Lowest SO ₂ , %	0-97 (80)	0-93 (78)	0,318
Time SO ₂ <90, min	0-96 (15)	0-96 (21)	0,078
Total sleep time, min	376 ± 53	377 ± 56	0,894
Obstructive sleep apnea, n (%)			
Mild	77 (56)	60 (44)	0,001
Moderate	36 (37)	61 (63)	0,001
Severe	17 (21)	63 (79)	0,001
Glomerular filtration rate (mL/min/1.73m ²)	99,04±11,36	95,47±10,91	0,005
Body surface area (m ²)	2,02±0,21	2,14±0,19	0,001

Continuous variables are normally distributed showed Mean ± standard deviation; continuous variables are not normally distributed showed minimum- maximum (median); categorical variables are presented as number (percentage.)

Table 2. Laboratory and echocardiographic findings

Left ventricular end-diastolic diameter (mm)	47,35±4,12	48,29±4,48	0,059
Left ventricular end-systolic diameter (mm)	29,15±4,57	30,80±4,83	0,002
Left atrial diameter (mm)	35,78±3,13	36,88±2,89	0,002
Interventricular septal thickness (mm)	11,17±1,31	11,63±1,38	0,003
Posterior wall thickness (mm)	10,38±0,92	10,78±1,00	0,001
E (cm/sn)	40-127 (70)	43-135 (70)	
A (cm/sn)	32-115 (75)	38-136 (76)	0,619
E/A	0,6-1,9 (0,9)	0,5-2,2 (0,9)	0,693
Glucose (mg/dL)	65-283 (95,5)	58-422 (104)	0,001
Urea (mg/dl)	12,7-96 (29,2)	13-80 (32)	0,048
Creatinine (mg/dL)	0,84±0,19	0,89±0,21	0,054
Total cholesterol (mg/dL)	193,54±38,70	199,79±40,15	0,169
Triglyceride (mg/dL)	40-447 (129,5)	63-511 (188)	0,001
High-density lipoprotein (mg/dL)	42,86±7,69	38,67±7,70	0,001
Low-density lipoprotein (mg/dL)	120,12±33,45	117,66±34,21	0,528
Gama-glutamyl transferaz, (U / L)	7-160 (25)	7-395 (25)	0,831
C-reactive protein, (mg/L)	0-22,7 (2,7)	0-21 (3,5)	0,046
Left ventricular mass index (gr/m ²)	92,73±20,20	95,18±20,10	0,289
Left ventricular ejection fraction (%)	63,17±7,18	60,24±8,00	0,001
Glomerular filtration rate (mL/min/1.73m ²)	99,04±11,36	95,47±10,91	0,005

Continuous variables are normally distributed showed Mean ± standard deviation; continuous variables are not normally distributed showed minimum- maximum (median); categorical variables are presented as number (percentage.)

Table 3. Logistic regression analysis for metabolic syndrome

	P value	OR	%95 CI	
			Lower	Upper
BMI	0,001	1,148	1,089	1,210
AAC (≥1)	0,001	4,986	2,887	8,610
AHI	0,001	1,039	1,016	1,062

CI indicates confidence interval, OR: Odds ratio, BMI: Body mass index, AAC: Aortic arch calcification, AHI: Apnea hypopnea index

Pulmonary hypertension / Pulmonary vascular diseases

PP-057

Determinants of quality of life in patients with pulmonary arterial hypertension

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Background and Aim: Pulmonary arterial hypertension (PAH) is a progressive disease that pervades all aspects of a patient's activities of daily living. However, despite the significant progress that has been made in the development of new therapies for PAH, the impact the disease has on patients' quality of life was less well understood. The effect of pulmonary arterial hypertension (PAH) symptoms on the patient's physical and emotional status can affect the quality of life. The aim of this study was to investigate the determinants of quality of life in patients with PAH.

Methods: Thirty-two patients with PAH were included in this study. The patients with PAH were categorized according to the New York Heart Association (NYHA) functional classification. The Hospital Anxiety and Depression Scale was used in the evaluation of anxiety-depression. Functional exercise capacity was measured by 6 Minute Walk Test. The quality of life was determined by the Nottingham Health Profiles scale.

Results: The median age of the patients were 58 (IQR, 38-66) years and, the median pulmonary arterial pressure was 70.0 (IQR, 54.0-93.0) mm Hg. There was a statistically significant correlation between quality of life and functional class, anxiety-depression level, and exercise capacity (p<0.05). Functional class and anxiety-depression level were explained about 81.5% of the variability of the quality of life.

Conclusions: Decline in quality of life was associated with an increased anxiety-depression level, decreased exercise capacity and worse functional class. Among these variables, functional class and anxiety-depression levels are important determinants of the quality of life. Associations of impaired quality of life with these specific variables can suggest potential areas for targeted intervention.

Pulmonary hypertension / Pulmonary vascular diseases

PP-058

The effect of noninvasive ventilation on left and right myocardial function in patients with obstructive sleep apnea syndrome: A speckle tracking echocardiographic based study

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Background and Aim: In patients with obstructive sleep apnea syndrome (OSAS), repetitive hypoxia due to sleep-induced apnea adversely affects the interaction between myocardial oxygen demand and supply, resulting in the development of subclinical cardiac dysfunction. The purpose of the study was to analyze the different involvement of left ventricle (LV) and right ventricle (RV) functions in patients with OSAS treated with noninvasive ventilation (NIV).

Methods: Conventional 2D echocardiography (2DE) and speckle tracking echocardiography (STE) were performed in 64 patients with OSAS undergoing NIV (M/F 48/16; mean age 66.5±10.3 years). LV and RV global longitudinal strain (GLS) was calculated by averaging local strain along the entire right and left ventricle, before and after 6 months of nocturnal NIV therapy.

Results: There was no statistically significant difference between before and after NIV therapy in 2DE measurements, except estimated SPAP (38±4.6 vs. 26±3.8). After NIV therapy RV GLS, LV GLS, and RV longitudinal systolic strain dyssynchrony index significantly decreased (Table 1).

Conclusions: STE is a useful tool for assessing left and right heart myocardial deformation in patients with OSAS and for monitoring the effect of NIV.

Table 1. Comparison of 2DE and STE measurements before and after NIV

	OSAS patients before NIV	OSAS patients after NIV	P value
LV ejection fraction (%)	64.4±6.7	62.7±5.9	0.48
RV FAC (%)	42.5±5.6	43.2±5.7	0.27
Mitral E/e'	7.5±3.4	6.8±3.2	0.39
Mitral E/A	0.8±0.2	0.9±0.1	0.35
SPAP (mmHg)	38.2±5.6	26.7±4.3	0.01
RV end-diastolic diameter (mm)	45.3±15.6	43.7±12.9	0.14
TAPSE(mm)	18.2±3.4	19.3±3.4	0.25
RVS (cm/s)	11.2±2.4	12.3±2.5	0.12
LV GLS (-%)	19.6±4.3	20.8±4.9	0.02
RV GLS (-%)	17.8±3.7	20.3±4.5	<0.001
RV PLSSD index	68.4±4.7	24.6±2.5	<0.001

NIV: non-invasive ventilation; GLS: global longitudinal strain; PLSSD: peak longitudinal systolic strain dyssynchrony.

Cardiac imaging / Echocardiography

PP-059

A novel predictor of chemotherapeutic cardiotoxicity in patients with non-hodgkin lymphoma

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Background and Aim: It is known that chemotherapeutic agents cause myocardial cell damage leading to left ventricular dysfunction and heart failure. Fragmented QRS is an indication of fibrosis developing as a result of myocardial cell damage. The aim of this study is to assess whether there is a relationship between the chemotherapeutic treatment and the development of the fragmented QRS complex in electrocardiography (ECG).

Methods: Among 130 patients diagnosed as having non-hodgkin lymphoma and receiving R-CHOP treatment regimen, the potential emergence of fragmented QRS on ECG as well as the changes in left ventricular ejection fraction (LVEF) (on TTE) in response to various chemotherapeutic regimens were sought.

Results: New development of fragmented QRS pattern was observed in 53 out of 130 (40.8%). These patients were found to have lower LVEF values along with higher number of chemotherapy courses and cumulative doses. In logistic regression analysis, age (Odds ratio (OR): 1.042; 95% Confidence interval (CI): 1.009-1.076); p=0.012) and number of courses (OR: 1.848 (95% CI: 1.409-2.423; p<0.001) were found to be the most important predictors of fragmented QRS development. In subjects with fragmented QRS pattern, there exists a significant difference between the initial and repeat LVEF values (p<0.001). Importantly emergence of fragmentation pattern occurred much earlier as compared with the drop in LVEF values (10.62±4.04 vs 15.24±7.49 months).

Conclusions: Development of fragmented QRS pattern in response to cancer therapy emerges as a new parameter potentially predicting chemotherapy-induced cardiotoxicity.

Cardiac imaging / Echocardiography

PP-060

The relationship between echocardiographic epicardial adipose tissue and P-wave dispersion and corrected QT interval

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Background and Aim: Epicardial adipose tissue (EAT) secretes various pro-inflammatory and atherogenic substances that have several effects on the heart. In this study, we aimed to evaluate the association between EAT thickness and both P-wave dispersion (Pd) and corrected QT (QTc) interval, as simple, non-invasive tools of pro-arrhythmia on surface electrocardiogram.

Methods: This retrospective observational study included 216 patients who had normal coronary arteries at coronary angiography. Each patient underwent 12-derivation electrocardiography to measure Pd and QTc interval and transthoracic echocardiography to measure EAT thickness. Patients were divided into two groups according to the median EAT value (EAT low group <5.35mm and EAT high group ≥5.35 mm).

Results: Body surface area (p=0.049), leucocyte count (p<0.001) and neutrophil count (p=0.033) were significantly increased in the EAT high group compared to the EAT low group. P-wave dispersion (p=0.001) was significantly increased in the EAT high group compared to the EAT low group. However, QTc interval (p=0.004) was significantly increased in the latter. Median left ventricular end-diastolic diameter (p=0.033), mean left ventricular end-systolic diameter (p=0.039) and mean left atrial diameter (p=0.012) were significantly increased in the EAT high group compared to the EAT low group. Multiple logistic regression analysis with backward method revealed that leucocyte count (OR=1.000, 95% CI: 1.000 to 1.000, p=0.001), Pd (OR=1.1026, 95% CI: 1.010 to 1.043, p=0.002), QTc interval (OR=0.988, 95% CI: 0.979 to 0.997, p=0.009) and left ventricular ejection fraction (OR=0.922, 95% CI: 0.859 to 0.989, p=0.023) were independently associated with high EAT thickness.

Conclusions: Echocardiographic end-diastolic EAT thickness on the free wall of the right ventricle is associated with Pd and QTc interval in patients with normal coronary arteries.

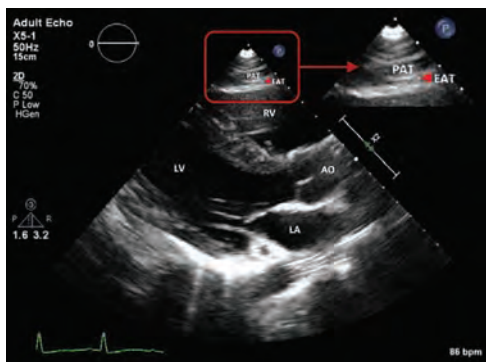


Figure 1. Echocardiography measurement of the end-diastolic epicardial adipose tissue thickness on the parasternal long-axis. EAT: Epicardial adipose tissue (Arrowhead (PAT: Pericardial adipose tissue, RV: Right ventricle; LV: Left ventricle; Ao: Aorta; LA: Left atrium.)

Cardiac imaging / Echocardiography

PP-061

Effect of vitamin D on myocardial deformation parameters in diabetic and non diabetic patients without significant coronary artery disease, a speckle tracking study

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Background and Aim: Vitamin D (VD) deficiency is a pandemic disease which appears in all stages of life. A growing number of studies call attention to the relationship between VD deficiency and cardiovascular disease. The aim of our study is to evaluate the effect of VD deficiency on subclinical myocardial function in the diabetic and non diabetic patients who have no significant coronary artery disease.

Methods: In our study we included 80 diabetic and 60 non-diabetic, a total of 140 patients in our clinic with symptoms of stable ischemic heart disease who underwent coronary angiography and had no significant coronary artery disease. In these patients VD levels were measured and patients who had VD levels below 20 ng/dl were defined as VD insufficiency group. In addition to conventional echocardiographic parameters, tissue Doppler echocardiography and 2D speckle tracking strain echocardiography were used for evaluating left ventricular functions.

Results: Average of VD levels in the diabetic group was 13,05 (interval 2.2-42.6) mg/dl, non-diabetic group was 16.4 (interval 1.6-48.8) and a total of 92 patients (65%) were inadequate. The baseline characteristics like age, gender were similar in the groups. In the diabetic group there were significant impairment of diastolic functions, increase of left ventricular myocardial performance index (MPI) and decrease of global longitudinal strain (GLS) and strain rate (SR) values. In the diabetic group, VD deficiency is significantly associated with impaired GLS (RR: 2.012, p<0.001). Also in the non-diabetic group, VD deficiency is associated with impaired GLS (RR: 1.591, p=0.049). There was no relationship between VD and diastolic function parameters in both group. Lastly another surprising results of our study were significant negative correlation between VD and GLS in the group with VD deficiency (r=-0.52623, p<0.001); but statistically significant positive correlation between VD and GLS in the group without VD deficiency (r= 0,286465, p=0,048).

Conclusions: There is a strong relationship between VD levels and GLS and SR. Both the low VD levels and the values above the physiological concentrations of VD may play a role in the pathogenesis of myocardial dysfunction in diabetic and non diabetic patients with normal coronary artery and preserved EF. VD deficiency is a pandemic disease less diagnosed by physicians although that can be treated easily and cheaply. VD deficiency should be treated but unnecessary replacement should be avoided for not cause VD toxicity.

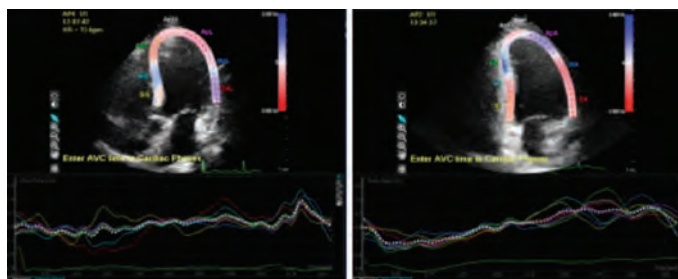


Figure 1. (a) decreased systolic strain rate in patients with VD deficiency, (b) normal systolic strain rate in patients without VD deficiency.

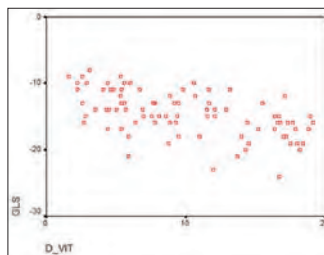


Figure 2. Correlation of vitamin D levels and LV global strain, VD deficiency group.

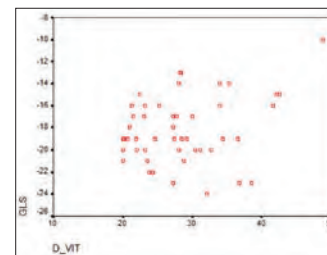


Figure 3. Correlation of vitamin D levels and LV global strain, without VD deficiency group.

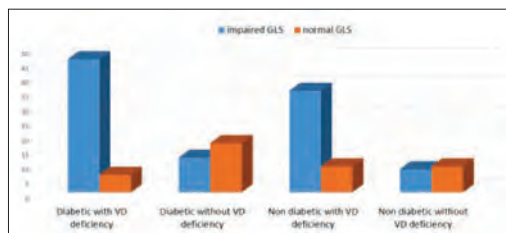


Figure 2. The effect of the VD deficiency on LV GLS, in diabetic and non-diabetic groups.

Pulmonary hypertension / Pulmonary vascular diseases

PP-062

Bendopnea and its clinical impact in pulmonary arterial hypertension outpatients

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Background and Aim: Bendopnea is a novel symptom recently reported, which is defined shortness of breath when bending forward in patients with heart failure (HF). It has been demonstrated that bendopnea is associated with advanced symptoms and worse clinical outcomes. The aim of this study was to assess presence of bendopnea and its clinical importance such as functional status, hemodynamic and echocardiographic characteristics in outpatient pulmonary arterial hypertension (PAH) patients.

Methods: We conducted a prospective observational study of 53 patients who admitted to our PAH clinic for routine controls. We determined presence of bendopnea and analyzed hemodynamic parameters, World Heart Organization (WHO) functional class, transcutaneous oxygen saturation, 6-minute walking distance (6-MWD), N-terminal pro-brain natriuretic peptide (NT-proBNP) and right ventricular (RV) function indicators in patients with and without bendopnea.

Results: Bendopnea was present 33.9% in PAH patients. The mean age was higher in patients with bendopnea than patients without bendopnea, but there was no difference between two groups ($p=0.201$). In patients with bendopnea was found lower 6-MWD and higher NT-proBNP levels ($p<0.001$). WHO functional class symptoms were worse in these patients ($p=0.010$). Mean right atrial pressure, pulmonary artery pressure, and pulmonary vascular resistance were found higher in patients with bendopnea. Patients with bendopnea had more dilated RVEDD and lower tricuspid annular plane systolic excursion values ($p<0.001$ and $p=0.001$, respectively).

Conclusions: Bendopnea is associated with worse functional capacity status, hemodynamic characteristics and RV functions in outpatient PAH patients. These data may reflect advanced symptoms and disease severity in PAH as well as HF.

Cardiac imaging / Echocardiography

PP-063

Relationship between two-dimensional speckle tracking echocardiographic parameters and infarct size in STEMI

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Background and Aim: We evaluated the relationship between single-photon emission computed tomography (SPECT) guided infarct size and echocardiographic left ventricular global torsion angle, apical rotation and basal rotation values after successful percutaneous coronary intervention (PCI) in patients with ST elevation myocardial infarction.

Methods: This prospective, randomized study consisted 102 patients (50 pts with anterior and 52 pts with inferior AMI) who underwent successful PCI. 50 age-matched subjects were included for controls. All patients had 2D echo and 2D-STI at 2 days, and SPECT MPI at 2 months after PCI. Left ventricular ejection fraction (LVEF), left ventricular end diastolic volume (LVEDV), end systolic volume (LVESV), wall motion score index (WMSI), average global longitudinal peak systolic strain (GLPSavg), global torsion angle (GTOR angle), apical rotation, and basal rotation were measured. The infarct size (left ventricular myocardium %) was calculated by SPECT myocardial imaging after 7 mCi Tc-99m MIBI injection.

Results: GLPSavg was lower in patients with Anterior MI (AntMI) compared to patients with Inferior MI (InfMI), and in patients with InfMI compared to the control group (11.10±2.5, 14.62±1.2, 18.50±1.1, respectively, P-values; Groups 1-2 = 0.000, Groups 1-3 = 0.000, Groups 2-3 = 0.000). Infarct size shows a significant correlation with GLPSavg values in AntMI and InfMI patients ($r=-0.669$, $p=0.000$ and $r=-0.722$ $p=0.000$, respectively). GTOR angle was lower in patients with AntMI compared to patients with InfMI and control group (11.38±3, 17.87±3, 18.73±3, respectively, P-values; Groups 1-2: 0.000, Groups 1-3: 0.000, Groups 2-3: 0.329). Apical rotation was lower in patients with AntMI compared to patients with InfMI and control group (6.51±2.4, 13.20±2.5, 14.3±2.1, respectively, P-values; Groups 1-2: 0.000, Groups 1-3: 0.000, Groups 2-3: 0.150). GTOR angle, apical rotation, and the progressive decrease in EF have been found to be associated with the increase in infarct size in AntMI ($r=-0.762$ $p=0.000$, $r=-0.815$ $p=0.000$, $r=-0.418$ $p=0.000$, respectively). GTOR angle and apical rotation showed no difference between InfMI and control group (17.87±3, 18.73±3 $p=0.329$ and 13.20±2.5, 14.3±2.1 $p=0.612$, respectively). There was no correlation between infarct size and apical rotation, GTOR angle in InfMI group. Basal rotation showed no difference between patients with AntMI, InfMI and control subjects (5.30±1.2, 4.94±1.8, 5.06±1.27, respectively, P-values; Groups 1-2: 0.998, Groups 1-3: 0.918, Groups 2-3: 0.935 respectively).

Conclusions: According to our study results, significant negative correlations were found between infarct size and GTOR angle, apical rotation after anterior MI, but not inferior MI. Influence of rotation parameters was related to infarct location. In early post-MI period: GTOR angle, apical rotation, and GLPSavg might be useful to estimate infarct size after AntMI, and GLPSavg might be useful to estimate infarct size after InfMI.

Table 1. Basic echocardiographic parameters, strain, rotation values and infarct size

	AntMI	InfMI	CONTROL	P value
GLPSavg (-)	11,10±2,5	14,62±1,2	18,50±1,1	Groups1-2:0,000 Groups1-3:0,000 Groups2-3:0,000
GTOR angle (°)	11,38±3	17,87±3	18,73±3	Groups1-2:0,000 Groups1-3:0,000 Groups2-3:0,329
Apical rotation angle (°)	6,51±2,4	13,20±2,5	14,3±2,1	Groups1-2:0,000 Groups1-3:0,000 Groups2-3:0,150
Basal rotation angle (°) (-)	5,30±1,2	4,94±1,8	5,06±1,27	Groups1-2:0,998 Groups1-3:0,918 Groups2-3:0,935
LVEDV (mL)	124,4±12,9	115,3±14,8	109,8±9	Groups1-2:0,010 Groups1-3:0,010 Groups2-3:0,100
LVESV (mL)	68,3±12	66,3±9,8	51,6±7	Groups1-2:0,612 Groups1-3:0,000 Groups2-3:0,000
EF (%)	42,3±7,8	50,6±8,6	59,2±4,2	Groups1-2:0,000 Groups1-3:0,000 Groups2-3:0,000
WMSI	1,48±,14	1,27±,1		Groups1-2:0,000
Infarct size (left ventricular mass %)	23,8±16,8	7,7±7,5		Groups1-2:0,000

AntMI=anterior myocardial infarction; InfMI=inferior myocardial infarction; GLPSavg=average global longitudinal strain; GTOR=global torsion.

Table 2. Correlation table

Infarct size in AntMI	r	P-value
WMSI	0,445	0,003
GLPSavg	-0,669	0,000
GTOR	-0,762	0,000
Apical rotation	-0,815	0,000
Basal rotation (-)	-0,244	0,240
EF (%)	-0,418	0,004

WMSI=wall-motion score index; GLPSavg=average global longitudinal strain; GTOR=global torsion; EF=ejection fraction.

Table 3. Correlation table

Infarct size in InfMI	r	P-value
WMSI	0,480	0,018
GLPSavg	-0,722	0,000
GTOR	-0,249	0,211
Apical rotation	-0,205	0,306
Basal rotation (-)	-0,104	0,605
EF (%)	-0,828	0,000

WMSI=wall-motion score index; GLPSavg=average global longitudinal strain; GTOR=global torsion; EF=ejection fraction.

Pulmonary hypertension / Pulmonary vascular diseases

PP-064

Evaluation of frontal QRS-T angle in patients with acute pulmonary thromboembolism

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Background and Aim: The frontal QRS-T angle (f-QRS-T) angle is defined as the difference between the frontal QRS axis and T wave axis. f-QRS-T angle can reflect the presence of cardiovascular events; it has also been shown to play a role in predicting total mortality and sudden cardiac death in the general population.

Methods: Occlusion of any branch of pulmonary artery vasculature, whether partial or complete, is defined as pulmonary artery thromboembolism (PTE). Although it is well known that acute mortality and morbidity are very high in this patient group, it is not clear whether the long-term cardiovascular risk is increased or not. The aim of this study was to investigate this risk. For this purpose, f-QRS-T angle that is predictor of cardiovascular events was evaluated in patients with acute PTE.

Results: In a retrospective study a total of 158 subjects (76 control, 82 patients with PTE) were examined. f-QRS-T angle calculated and compared between the groups. p values <0.05 were considered significant. RESULTS: There was no statistically significant difference between the groups in terms of baseline clinical characteristics ($p>0.05$). There was a statistically significant difference between the groups in terms of f-QRS-T angle values (control group f-QRS-T angle value median 25 (10.25-40.5; Interquartile range= 30.25), PTE group f-QRS-T angle value median 49 (26.75-95.25; Interquartile range=68.5), $p<0.001$).

Conclusions: According to the findings of our study, f-QRS-T angle value was elevated in patients with PTE compared to the control group. This result indicates that the risk of cardiovascular events in patients with PTE might be higher than in the normal population. This association can be explained by the observation that right ventricular (RV) pressure overload in acute PTE affects right-sided cardiac conduction and repolarization to a greater degree than does chronically elevated RV pressure. An animal study also suggested that experimental acute pulmonary hypertension can produce RV subendocardial ischemia, which may explain the impairment of right-sided cardiac conduction and repolarization.

Cardiac imaging / Echocardiography

PP-065

Is it really true that neutrophil lymphocyte ratio and C-reactive protein are associated with left atrial or left atrial appendage thrombus?

A transesophageal echocardiography study

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Background and Aim: There is no a clear study showing that neutrophil lymphocyte ratio (NLR) and C-reactive protein (CRP) are investigated only with left atrial (LA) or left atrial appendage thrombus (LAAT). The aim of this study is to investigate the association between NLR, CRP and LA or LAAT.

Methods: 461 non-valvular atrial fibrillation patients with their demographic, clinic and laboratory parameters were enrolled in the study. All patients were evaluated by transesophageal echocardiography (TEE) before direct current cardioversion (DCCV) and cryoablation therapy for thrombus in the LA and LAA. Dense echogenic masses sticking to LA or LAA wall, moving independently from the LAA wall was accepted as LA/LAA thrombus.

Results: LAT/LAAT (+) group consisted of 172 (37%) patients, LAT/LAAT (-) group consisted of 289 (63%) patients. Percentage of patients with CHA2DS2-VASc \geq 3 was significantly higher in LAT/LAAT (+) group compare with LAT/LAAT (-) group (89 [51.7%] vs 117 [40.5%], p<0.001). The CRP and NLR were higher in LAT/LAAT (+) group than in LAT/LAAT (-) group but these differences were not significant (1.45 [0.35-1.2] vs 1.22 [0.29-1.2], p=0.285 and 3.3 [1.73-3.5] vs 3.1 [1.75-3.8], p=0.506, respectively). Also, there was no any significant difference between high CRP value and LAT/LAAT (+) group by Chi-square test.

Conclusions: We advocate that in previous studies, patients with both LA/LAA SEC and LAT/LAAT were included. But, in our study, the patients with LA or LAA SEC were excluded from the analysis. In our study, accepting only patients with thrombus may be one of the reasons why the results differ from previous studies. In conclusion, our study will highlight the complex results of other studies on CRP, RDW, NLR with LAT/LAAT, and will show the necessity of further studies investigating the relationship between these parameters and non-valvular AF.

Table 1. Demographic characteristics and risk factors for left atrial thrombus/left atrial appendage thrombus (+) and (-) groups

Variables	LAT/LAAT (+) (n=172)	LAT/LAAT (-) (n=289)	P value
Age, years	62.7±12.1	59.5±12.6	0.479
Male (n)	87 (50.6%)	135 (46.7%)	0.574
Hypertension (n)	60 (34.9%)	109 (37.7%)	0.469
Diabetes mellitus (n)	35 (20.3%)	67 (23.2%)	0.589
Stroke (n)	30 (17.4%)	47 (16.3%)	0.469
Heart failure (n)	54 (31.4%)	83 (28.7%)	0.211
Vascular disease (n)	20 (11.6%)	35 (12.1%)	0.722
CHA2DS2-VASc score	3.22±1.3	2.47±1.1	0.08
CHA2DS2-VASc>3 (n)	89 (51.7%)	117 (40.5%)	<0.001

LAT: left atrial thrombus; LAAT: left atrial appendage thrombus; CHA2DS2-VASc: Congestive Heart Failure, Hypertension, Age, Diabetes Mellitus, Stroke, Vascular events, Sex.

Table 2. Comparison of laboratory parameters between left atrial thrombus/left atrial appendage thrombus (+) and (-) groups

Variables	LAT/LAAT (+) (n=172)	LAT/LAAT (-) (n=289)	P value
Hemoglobin, g/dL	14.45±1.62	13.97±1.89	0.421
Platelet, 103/ μ L	242.5±76.5	237.1±76.8	0.464
WBC, 103/ μ L	8298.3±2017.2	7976.2±2140.1	0.111
Neutrophil, 103/ μ L	5389.9±1856.4	5068.7±1839	0.077
Lymphocyte, 103/ μ L	2096.6±873.3	2060.1±999.8	0.699
RDW, %	14.9±1.7	14.6±1.8	0.065
MPV, %	8.8±1.2	8.6±1.2	0.295
CRP, mg/L	1.45 (0.35-1.2)	1.22 (0.29-1.2)	0.285
NLR	3.3 (1.73-3.5)	3.1 (1.75-3.8)	0.506

CRP: C-reactive protein; WBC: Wight blood cell; RDW: Red blood cell distribution width; NLR: neutrophil lymphocyte ratio; LAT: left atrial thrombus; LAAT: left atrial appendage thrombus

Table 3. Comparison of CRP groups between left atrial thrombus/left atrial appendage thrombus (+) and (-) groups

	LAT/LAAT (-)		LAT/LAAT (+)		Total	
	n	%	n	%	n	%
CRP Group 1	113	24.5	71	15.4	184	39.9
Group 2	176	38.2	101	21.9	277	60.1
Total	289	62.7	172	37.3	461	100

Group 1: patients with high CRP value (CRP value > 0.5 mg/dL); group 2: patients with normal CRP value (CRP value \leq 0.5 mg/dL); LAT: left atrial thrombus; LAAT: left atrial appendage thrombus $\chi^2=1.34$ p=0.648

Cardiac imaging / Echocardiography

PP-066

Aortic arch calcification detected by chest X ray is associated with early postoperative major cardiac events after elective orthopedic surgery

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Background and Aim: Cardiovascular complications after orthopedic surgery may be mortal. Therefore, predictors of early cardiovascular events after elective orthopedic surgery are required. The aim of this study is to investigate the relationship between aortic arch calcification and 30-day major adverse cardiac events following elective orthopedic surgery.

Methods: Patients who underwent elective orthopedic surgery are included to the study. Preoperative detailed anamnesis was taken. Echocardiography and standard chest x ray were performed. Patients were followed in terms of development of postoperative 30-days major cardiac event. Patients were divided into 2 groups according to postoperative cardiac event development. Aortic arch calcification was evaluated by 2 cardiologists blinded to patient information and was graded as 0 to 3 on chest x ray.

Results: A total of 714 patients were included in the study (Mean age 70±12, 465 (65%) women). Of these, 33 had cardiac events. Prevalence of aortic arch calcification \geq 2, coronary artery disease, hypertension and smoking were higher in the group with cardiac event than in the group without cardiac event. Moreover, LEE index was higher, left ventricular systole, diastole and left atrium diameter were larger and GFR was lower in cardiac event group. Multivariate regression analysis showed that smoking (OR 3.325, 95% CI 1.163to 9.505), presence of hypertension (OR 4.237, 95% CI 1.151 to 15.599) and aortic arch calcification \geq 2 (OR 22.565, 95% CI 7.598 to 67.020) are independent predictors of major cardiac event for postoperative 30-day-period after elective orthopedic surgery.

Conclusions: Grade 2 and greater aortic arch calcification may alert clinicians to develop a major cardiac event for 30 days after elective orthopedic surgery.

Table 1. Baseline characteristics of the study population

	Complication No	Complication Yes	P value
Female gender, n (%)	443 (65,1)	22 (66,7)	1,00
Aortic arch calcification \geq 1, n(%)	439 (64,5)	33 (100)	<0,001
Aortic arch calcification \geq 2, n(%)	149 (21,9)	29 (87,9)	<0,001
Aortic arch calcification \geq 3, n(%)	38 (5,6)	16 (48,5)	<0,001
Heart Failure, n(%)	41 (6,0)	3 (39,4)	0,449
Chronic Kidney Disease, n(%)	72 (10,6)	4 (12,1)	0,771
Coronary artery disease, n(%)	82 (12,0)	10 (30,3)	0,006
Diabetes mellitus, n(%)	208 (30,5)	13 (39,4)	0,335
Cerebrovascular disease, n(%)	40 (5,9)	2 (6,1)	1,00
Smoking n(%)	57(8,4)	7 (21,2)	0,022
Hyperlipidemia, n(%)	66 (9,7)	7 (21,2)	0,068
Hypertension, n(%)	473 (69,5)	30 (90,9)	0,006
Left ventricular hypertrophy, n(%)	353 (51,8)	20 (60,6)	0,375
LEE index, n(%)	0 (0-4)	1 (0-4)	0,014
Age, (year)	71 (18-96)	73 (49-94)	0,098
Left ventricular end diastolic diameter (mm)	47(40-68)	50(39-60)	0,001
Left ventricular end systolic diameter(mm)	30(19-56)	32(22-46)	0,032
Left atrium diameter (mm)	36(25-60)	37(30-53)	0,041
Glomerular filtration rate, ml/min \times 1.73 m ²	85,59(20,18-188,28)	80,38(43,82-100,87)	0,010
Total Cholesterol, (mg/dl)	181(87-383)	186,6(90-297)	0,915
Low-density lipoprotein,(mg/dl)	106(37-233)	112(34-191)	0,884
High density lipoprotein, (mg/dl)	44,3(15-78,6)	43(19-62)	0,242
Triglycerides,(mg/dl)	132(39-939)	136(81-337)	0,294
Left ventricular mass index (gr/m ²)	101,2 (58,3-201,0)	104,5(65,4-192,0)	0,252
Left ventricular mass (gr)	192,54 (109,7-397,2)	206,89 (136,2-320,7)	0,098
E/A	0,82 (0,46-2,58)	0,80 (0,47-1,4)	0,222
Body mass index (kg/m ²)	28,23(16,73-43,75)	28,73(19,53-36,98)	0,578
Creatinine, (mg/dl)	0,80 (0,32-6,53)	0,88 (0,5-3,6)	0,004
Glucose, (mg/dl)	118(56-531)	133(92-405)	0,165

Table 2. Risk factors associated with MACE in a multivariable logistic regression analysis

	P	OR	95% CI Lower	95% CI Upper
Aortic arch calcification \geq 2	<0,001	22,565	7,598	67,020
Coronary artery disease	0,125	2,835	0,748	10,743
Smoking	0,025	3,325	1,163	9,505
Hypertension	0,030	4,237	1,151	15,599
LEE Index	0,231	0,668	0,345	1,293
Left ventricular end diastolic diameter	0,061	1,182	0,992	1,408
Left ventricular end systolic diameter	0,188	0,908	0,787	1,048
Left atrial diameter	0,304	1,059	0,949	1,182
Creatinine	0,330	1,462	0,681	3,138
Glomerular filtration rate	0,919	1,002	0,971	1,033

OR: Odds ratio, CI: Confidence interval, MACE: Major adverse cardiovascular events.

Cardiac imaging / Echocardiography

PP-067

Epicardial adipose tissue of prospective definition in population (EPICARD STUDY)

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Background and Aim: Epicardial adipose tissue (EAT) is the fat compartment located inside the epicardial sac and in coronary artery surround region. EAT has been claimed co-existence with coronary atherosclerosis, atrial fibrillation. It has been known that the prevalence of atrial fibrillation and coronary atherosclerosis is related to age. Radiodensity of human tissues show differences of biological characteristics, such as fat degeneration. Due to amorphous shape of epicardial adipose tissue, radiodensity measurements are not standardized. Besides, the relationship between age and EAT radiodensity is not known very well. The aim of the present study is to quantify the relation between standardized individual EAT radiodensity in heart and a decade of the population.

Methods: A total of 147 patients of the Epicardial Adipose Tissue of Prospective Definition in Population (EPICARD) study underwent cardiac-CT angiography because of suspected coronary artery disease. EAT radiodensity (Hounsfield unit, HU) was measured in the three different epicardial regions (right atrioventricular groove, posterior interventricular groove, and anterior epicardial region). Due to amorphous shape of epicardial adipose tissue, these measurements were standardized with subxiphoid fat tissue and three epicardial fat radiodensity / subxiphoid fat radiodensity ratio value were obtained as constant ratio. While normal CT attenuation range for fat tissues is known in the interval -190<HU>-30, direction from negativity to positivity was accepted fat degeneration. Constant ratio was classified according to a period of ten years (decade).

Results: Three epicardial region (right atrioventricular groove, posterior interventricular groove, and anterior epicardial) radiodensity and individual subxiphoid fat radiodensity ratio were obtained. Every group ratio are shown table 1. We found that epicardial adipose tissue/ subxiphoid fat radiodensity ratio decreased with increasing age. After 3th decade, we observed that EAT/subxiphoid fat radiodensity ratio decrease sharply.

Conclusions: EAT radiodensity ratio changes with age. Increasing age is associated with less epicardial adipose tissue radiodensity ratio, which is accepted as fat degeneration. We realized sharply decreasing after third decade in ratio. The epicardial adipose tissue radiodensity/subxiphoid fat density can be used as a degeneration biomarker of cardiovascular risk.

Table 1. The radiodensity values of right atrioventricular groove EAT/subxiphoid fat, posterior or interventricular EAT/subxiphoid fat, and anterior EAT/subxiphoid fat

A decade (years)	Right atrioventricular groove	Posterior interventricular groove	Anterior
11-20	1.99±2.63	1.73±1.54	1.71±1.78
21-30	1.71±1.78	1.71±0.14	1.70±0.07
31-40	1.04±0.30	1.06±0.40	1.17±0.42
41-50	1.02±0.43	1.02±0.42	1.16±0.36
51-60	0.96±0.47	1.01±0.34	1.11±0.28
61-70	0.92±0.40	0.98±0.45	1.03±0.38
71-80	0.89±0.36	0.93±0.46	0.99±0.35

Cardiac imaging / Echocardiography

PP-068

Noncompaction cardiomyopathy and cardiac MR imaging in Ege University

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Background and Aim: Noncompaction cardiomyopathy (NCCM) is a myocardial disorder characterized by excessive and prominent trabeculations associated with deep recesses that communicate with the left ventricular (LV) cavity. The original definition of LVNC required the generation of echocardiography- and CMR-based quantitative indexes that measure ratios between noncompacted and compacted layers of the LV wall. Cardiac magnetic resonance imaging is increasingly utilized as a confirmatory imaging modality for evaluating suspected NCCM. LVNC can be regarded as an isolated entity or as one of the traits that may recur in cardiac and noncardiac diseases. We aimed to evaluate LVNC patients who referred our hospital and had advanced CMR imaging to indicate characteristics of these patients, detailed features of CMR reports and different groups of LVNC.

Methods: Our study is a retrospective case control study. We analyzed the CMR imagings between 2006 and 2018 years in our radiology department with the key word 'noncompaction'. Then we reached 64 reports which showed us related to noncompaction cardiomyopathy. We evaluated these reports in terms of diagnostic criteria, hypertrabeculated areas, EF values and different groups of LVNC and accompanying disease. After evaluation of these reports we searched for these patients morbidity, mortality and cardiac operations. We didn't evaluate patients for drugs using and other endpoints because of insufficient data.

Results: After evaluation of CMR reports of which people had advanced imaging for differential diagnosis of LVNC. In our study population 38(59%) of 64 patients were men. Mean age was 41 in between ages 18 - 73. Hypertrabeculation was prominently seen in the LV lateral wall followed by apex and inferior wall. Ratio of thickness of NC and C myocardial layers at the site of maximal WT was measured and averaged around 2,9 (2,2-4). 10 patients had suspicious diagnosis for NCCM, because 4 of them didn't meet the criteria and 6 of them had insufficient data. 58 patients had datas about left ventricular ejection values, mean value 35 (17-69). Considering accompanying disorders, we observed 5 patients had CAD, 2 patients had primary valve disorders, 1 patient had connective tissue disorder, 1 patient had distrophin gene mutation, 1 patient had norfibromatosis type 1, 3 patients had peripartum cardiomyopathy, 3 patients had congenital heart anomalies. 3 patients had biventricular NC and 1 patient had isolated right ventricular NC. Regarding to morbidity,

mortality and cardiac operations, 6 patients had CRT-D implantation, 5 patients had LVAD implantation and 3 patients had heart transplantation story. 4 of 64 patients were exitus.

Conclusions: Although NCCM was included in the 2006 WHO classification of primary cardiomyopathies, it remains subject to controversy because of a lack of consensus on its etiology, pathophysiology, diagnosis, and management. There is a requirement a consensus about diagnostic imaging modalities and accompanying conditions about NC.

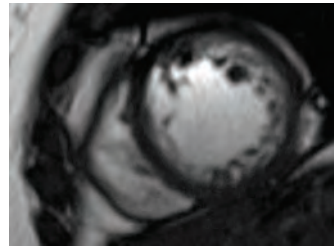


Figure 1. Short axis cardiac mr image of a LVNC. In the figure, hypertrabeculation was seen in short axis cardiac mr image of a LVNC patient.

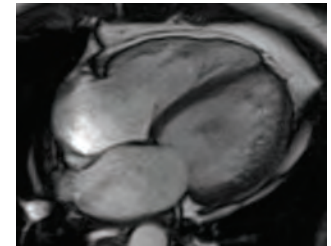


Figure 2. Diastolic four chamber cardiac mr image of a LVNC. In the figure, hypertrabeculation was seen in four chamber diastolic cardiac mr image of a LVNC patient.

Cardiac imaging / Echocardiography

PP-070

Evaluation of left atrial electromechanical delay and left atrial phasic functions in surgical early menopause patients

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Background and Aim: The present study evaluated the atrial electromechanical delay and left atrial mechanical functions in patients with surgical early menopause and compared with healthy individuals.

Methods: A total of 62 patients were added in the study; 33 patients with surgical early menopause and 29 age- and gender-equalled healthy individuals (control group). Echocardiographically, duration distances from the starting of P wave to the beginning of A wave from the lateral, septal mitral and lateral tricuspid annulus in tissue doppler imaging were measured. The distinctions among these durations showed the inter- and intra-atrial mechanical delays. LA volumes were evaluated using the biplane area-length technique, and LA mechanical function values were measured. Statistical evaluation was made using Student's t-test, chi-square test, and Pearson's test.

Results: The laboratory and basal clinical characteristics were similar in the two groups. Surgical early menopause patients displayed increased statically PA' septal and PA' tricuspid time as compared to the controls. However, PA' lateral time, inter-atrial, intra-LA, and right atrial EMD were statistically nonsignificant in surgical early menopause patients as compared to controls. Left atrium Vmax (49.6±14.1 vs. 42.9±16.1, p=0.004), Vmin (18.4±7.0 vs. 15.2±9.0, p=0.022), and VpreA (31.0±10.9 vs. 24.9±10.1, p=0.006) were importantly higher in the patients with surgical early menopause as compared to the controls. LA reservoir, conduit and pumping functions and total passive, active emptying volumes were similar in the two groups (p>0.05).

Conclusions: A statistically important impairment was detected in the patients who underwent surgical early menopause in terms of atrial electrical delay and electromechanical functions.

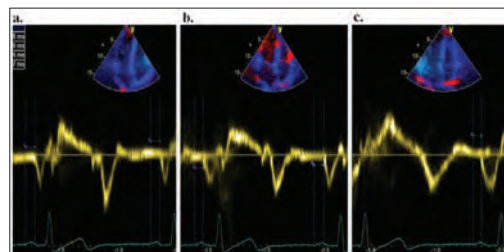


Figure 1. Atrial electromechanical coupling (PA'), the time period from the beginning of the P-wave on the superficial electrocardiogram to the starting of the late diastolic wave A' on tissue doppler echocardiography (Mitral lateral annulus A., Septal annulus B. and Tricuspid lateral annulus C.).

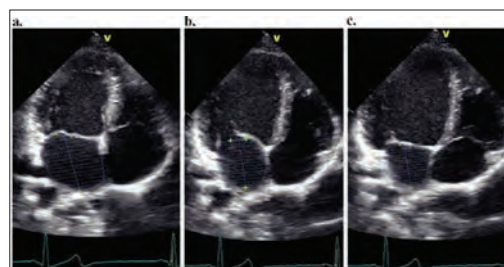


Figure 2. Maximum A. atrial precontraction B. and the minimum C. volume measurements of left atrium were made by transthoracic echocardiography on apical four-chamber window.

Table 1. Atrial electrical activity parameters between the surgical menopause and control groups

	Surgical Menopause (n=33) M±SD (min-max)	Control Group (n=29) M±SD (min-max)	P Value**
PA' lateral (ms)	69.6 ± 12.2 (48-101)	60.0 ± 17.3 (30-89)	0.051
PA' septal (ms)	62.8 ± 12.5 (39-88)	51.1 ± 14.7 (15-80)	0.003
PA' tricuspid (ms)	60.1 ± 11.8 (32-83)	47.7 ± 14.5 (20-80)	0.001
Interatrial-EMD (ms)	9.5 ± 11.6 (-9;39)	12.2 ± 12.0 (-15;38)	0.350
Intra-RA-EMD (ms)	2.6 ± 13.7 (-23;35)	3.0 ± 12.2 (-18;27)	0.928
Intra-LA-EMD (ms)	6.9 ± 16.2 (-33;42)	9.6 ± 14.9 (-15;41)	0.629

Table 2. Left atrial mechanical functions between the surgical menopause and control groups

	Surgical Menopause (n=33) M±SD (min-max)	Control Group (n=29) M±SD (min-max)	P Value**
LAVmax (mL)	49.6 ± 14.1 (17-90)	42.9 ± 16.1 (25-100)	0.004
LAVmin (mL)	18.4 ± 7.0 (5-40)	15.2 ± 9.0 (7-48)	0.022
LAVpreA (mL)	31.0 ± 10.9 (10-64)	24.9 ± 10.1 (13-61)	0.006
LA reservoir function %	63.0 ± 8.9 (36.1-80.5)	66.0 ± 9.4 (45.0-78.1)	0.090
LA conduit function %	37.6 ± 11.0 (11.1-58.2)	41.9 ± 10.1 (13.6-57.9)	0.069
LA pumping function %	40.1 ± 12.3 (14.-61.9)	41.2 ± 13.9 (12.0-63.2)	0.689
LA total emptying volume (mL)	31.2 ± 9.3 (12-51)	27.7 ± 8.4 (16-52)	0.060
LA passive emptying volume (mL)	18.6 ± 7.0 (4-32)	18.0 ± 8.0 (6-39)	0.481
LA active emptying volume (mL)	12.6 ± 6.0 (3-27)	9.7 ± 3.7 (3-19)	0.071

Cardiac imaging / Echocardiography

PP-071

Effectiveness of percutaneous coronary interventions in diabetic patients with dysfunctional myocardium

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Background and Aim: The aim of this study was to explore the effectiveness of percutaneous coronary interventions in diabetic patients with dysfunctional myocardium using cardiac MRI with late enhancement. **Methods:** 84 patients (pts) with regional WMA and LVEF <50% were recruited based on routine echocardiography. Other inclusion criteria were multivessel disease (SYNTAX score <32), CTO or subtotal stenosis of one or more coronary arteries; viable myocardium; heart failure (NYHA I-III). Pts were randomized in 2 groups with and without diabetes. The viability of myocardium and long-term results (18 months) of PCI were assessed by cardiac MRI. Cardiac MRI was performed to all pts prior to PCI. The follow-up echos were obtained in 1, 3, 6, 12 and 18 months after the revascularization to assess the recovery of WMA.

Results: Long-term results were observed in both groups. Major adverse cardiovascular events (MACE) were significantly more frequent in pts in which the distribution of fibrosis was 25% and more from the left ventricular myocardial mass. However, regarding the cumulative incidence rate in 18 months after the PCI there was no significant difference between two groups (14.3-12.5% p>0.05). The SYNTAX score was between 15 and 25. We haven't identified the correlation between severity of lesions of coronary arteries and the extent of LV myocardial fibrosis. The dynamics of recovery of myocardial function after stent implantation was significantly better in the group of pts without diabetes. Thus, in group I the average viable myocardial volume, in comparison with the data obtained at discharge from the hospital was 22.8 and 20.6%, while the increase in volume was 10.6%. In group II this indicator was 32.8 and 21.5%, while the volume increase was 32.1%, which is three times more than in group I (p<0.05). The additional analysis of the results of revasc Group I pts showed that of the functional recovery of viable, but dysfunctional myocardium, was most badly in pts with the LVEF <40%, the HgA1 level >7, as well as when time of revasc performing was more than 30 days after the onset of MI. In all studied groups, there was a significant increase in LVEF, compared with data obtained when the pt was discharged from the hospital.

Conclusions: The distribution of myocardial fibrosis in patients with MI doesn't depend on the severity of coronary arteries lesion assessed by Syntax score. The incidence of MACE arising in patients with dysfunctional myocardium and type 2 diabetes in the long-term period after revascularization is comparable to that in pts without diabetes. The dynamics of recovery of myocardial function in patients with diabetes is significantly worse, compared with patients without diabetes. The occurrence of MACE is significantly more frequent in pts with ischemic fibrosis volume of more than 25%. Performing myocardial revasc in pts with dysfunctional myocardium and type 2 diabetes, no later than 30 days after the onset of myocardial infarction, significantly improves PCI efficiency.

Cardiac imaging / Echocardiography

PP-072

Distribution of left ventricular ejection fraction according to age group

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Background and Aim: Heart failure is a clinical syndrome with functional and structural abnormalities. With increase of age, heart failure prevalence increases. In our daily practice, we noticed that if patient can arrive over 85 years of age, cardiac risk factors are lower and ejection fraction is better than younger patients. Therefore, in this study we retrospectively analysed the left ventricular ejection fraction in patients over 45 years old during one month period.

Methods: We retrospectively scanned echocardiography records of all patients over 45 years of age. 854 patients (51.1% men) records are analysed. We divided patients into 5 groups according to ages as 45-54 years group 1, 55-64 years group 2, 65-74 years group 3 and over 85 years group 5. Left ventricular ejection fraction is measured by using modified Simpson method during 2D echocardiography. Also, clinical risk factors were examined in all age groups.

Results: Left ventricular EF decreased from group 1 to group 4 however there was an increase in group 5 as after 85 years of age (Figure 1). The correlations between age groups and risk factors for coronary artery disease were given in Table 1.

Conclusions: In this study, we observed that during one month period of rutin echocardiographic examination over 45 years old, left ventricular EF decreased from group 1 to group 4 however there was an increase in group 5 after 85 years of age. Because, heart failure and coronary artery disease usually cause mortality before 85 years old. If a patient can arrive over 85 years, major cardiac problem possibility is low.

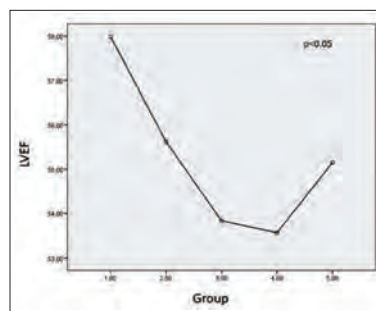


Figure 1. Relationship between LVEF and age of patients.

Table 1. The correlations between age groups and risk factors for coronary artery disease

	Group 1 n: 228	Group 2 n: 215	Group 3 n: 233	Group 4 n: 71	Group 5 n: 33
HT, n (%)	85(37.2)	116(63.4)*	144(61.8)*	27(38.0)	10(30.3)
DM, n (%)	56(24.5)	64(34.6)	71(30.5)	17(23.9)	7(21.1)
HL, n (%)	58(25.4)	69(32.4)	83(38.5)	15(23.1)	5(15.2)
KAH, n (%)	54(23.6)	83(47.3)*	117(50.2)*	18(25.4)	5(15.2)
Sigara, n (%)	52(22.8)	57(30.2)	9(3.9)	9(12.7)	4(12.1)

*p<0.05; group 5 vs. other group.

Pulmonary hypertension / Pulmonary vascular diseases

PP-073

The increasing prevalence of pre-capillary PH according to new PH definition described on 6th WSPH Congress

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Background and Aim: From the 1st WSPH (Geneva 1973), pulmonary hypertension has been defined arbitrarily as mean PAP > 25 mmHg measured by right heart catheterization (RHC) in the supine position at rest. This definition remained unchanged during subsequent WSPH. However, accumulating data in healthy individuals suggest that a normal mean PAP at rest is 14.0±3.3 mmHg. So, a mean PAP >20 mm Hg should be considered as above upper limit of normal value (mean value + 2 SD). However it is important to emphasize that a mean PAP >20 mm Hg is not defining a disease per se but is only an abnormal increase of pressure. In different conditions a mean PAP >20 mm Hg is associated with an increased risk of mortality, however it has never been demonstrated that decreasing PAP improves survival. This mild increase in PAP could be simply a marker of the underlying disease severity. On 6th WSPH congress (Nice 2018), it is suggested that pre-capillary PH could be defined as mean PAP >20 mm Hg, PAWP <15 mm Hg and PVR >3WU. The impact of the new definition of the number of pre-capillary PH patient identified would be low with preliminary data suggesting an increase <10%. In this study we aimed to investigate the impact of new definition of the number of pre-capillary PH.

Methods: We screened the RHC performed in our university hospital with various clinical indication between 2017 and 2018. The reports were screened from medical records retrospectively. Demographics, clinical indications, hemodynamic data were recorded. Both 2015 ESC/ERS and 6th WSPH congress PH definition

were used to define PH patients. Statistical analysis was performed using SPSS Statistics version 21.0 (IBM SPSS Statistics, IBM Corp., NY).

Results: Fifty-eight RHC was performed in our university in one year period. Most of the procedure was performed with a suspicion of PH. The rest of indications were valvular heart disease, left heart disease and lung disease. There were 40 female (69%) and 18 male (31%) patients. The mean age of study population was 53.3±16.6 years old. On RHC, the mean PAP was 36.4±16.4 mm Hg, mean PCWP was 12.6±3.9 mm Hg and mean PVR was 4.9±4.4 WU. While 43 of 58 patients (74.1%) were classified as pre-capillary PH according to 2015 ESC/ERS PH guideline, when 6th WSPH congress PH definition was used, 50 of 58 patients (86.2%) had pre-capillary PH. The impact of the new definition was 12.1%. Table 1 is showing demographic, hemodynamic and clinical characteristics of study population.

Conclusions: The impact of the new definition of the number of pre-capillary PH patient identified was predicted <10%, but our study revealed more prominent increase.

Table 1. Demographic, hemodynamic and clinical characteristics of study population

Parameter	
Age (years old)	53.3 ± 16.6
Female (N, %)	40 (69%)
Male (N, %)	18 (31%)
mean PAP (mmHg)	36.4 ± 16.4
PCWP (mmHg)	12.6 ± 3.9
PVR (WU)	4.9 ± 4.
Pre-capillary PH (2015 ESC/ERS definition)	43/58 (74.1%)
Pre-capillary PH 6 th WSPH definition	50/58 (86.2%)

Cardiac imaging / Echocardiography

PP-074

Does different slice thickness selection have an impact on the assessment of mitral valve structure?

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Background and Aim: Changing slice thickness during assessment of echocardiographic studies may impact results. The aim of this study is to detect proper slice thickness selection while using mitral valve navigator (MVN) and determine between inter and intra observer variability.

Methods: Patients that underwent percutaneous edge-to-edge mitral repair between January 2013 and May 2016 at a single institution were retrospectively reviewed. 3D mitral annular circumference, bicommissural diameter and anteroposterior diameter were measured using 3D transesophageal echocardiography images. Anatomical landmarks pointed as guided by the software model and editing applied to obtain the correct annular drawing. Measurements were taken both by slice thickness of 0 mm and 10 mm. Intra observer variability was assessed using repeated measurements performed by the same observer, whereas inter observer variability was evaluated by repeating the analysis by a second observer who was blinded and had equivocal experience in terms of software and 3D echocardiography. Intraclass correlation coefficient was determined for both the inter- and intra-observer variability for both slice thicknesses. Additionally, regression analyses were used to evaluate inter observer variability.

Results: With regard to intra observer variability, intraclass correlation coefficient (ICC) varied from 0.80 and 0.95 for slice thickness 0 mm. ICC varied between 0.86 and 0.96 for slice thickness 10 mm. When inter observer variability was evaluated, ICC was between 0.53 and 0.89 for slice thickness 0 mm. On the contrary, ICC was found to vary from 0.84 to 0.97 for slice thickness 10mm. Results indicated better inter observer correlation when slice thickness was set to 10 mm. Additionally, measurements of all anatomical structures were found to be increased when slice thickness chosen as 10 mm (p<0.001, for all).

Conclusions: Using different slice thickness alters the anatomic measurement results. Inter observer reliability was found to be better for slice thickness 10 mm when compared to 0 mm. Therefore, slice thickness should be set for 10 mm when using mitral valve navigator.

Table 1. Intra observer and inter observer variability of slice thicknesses 0 mm and 10 mm

	Intra observer variability (Range)	Inter observer variability (Range)
Slice thickness 0 mm	0.80 – 0.95	0.53 – 0.89
Slice thickness 10 mm	0.86 – 0.96	0.84 – 0.97

Cardiac imaging / Echocardiography

PP-075

Comparison between transthoracic and transesophageal echocardiographic parameters in the evaluation of right ventricular functions

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Background and Aim: Transthoracic echocardiography is the cornerstone method in evaluating right ventricular functions. Transesophageal echocardiography is a good alternative to TTE in patients with poor echogenicity or differential diagnosis of intracardiac mass. We aimed to investigate agreement between TTE and TEE in the assessment of RV functions.

Methods: Between January 2017 and December 2017, 47 patients were enrolled final analysis. TEE and TTE records of patients were compared with Bland-Altman analysis, and the agreement between two methods were evaluated.

Results: Bland-Altman analysis showed moderate agreement between TEE and TTE in terms of conventional doppler parameters and good agreement between TEE and TTE in terms of deformation parameters.

Conclusions: Assessment of right ventricular function may provide useful information in clinical decision making and adverse event prediction, and deformation parameters measured by TEE could be as useful and accurate as TTE and could be used in clinical decision making.

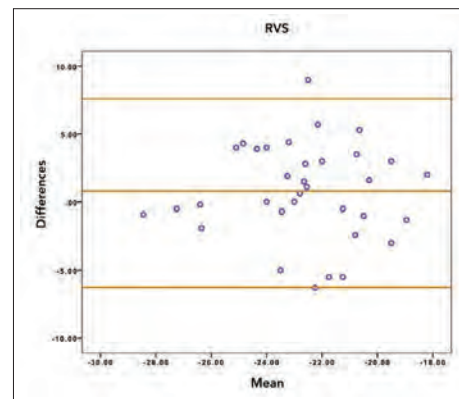


Figure 1. Bland-Altman analysis for Right ventricular strain.

Pulmonary hypertension / Pulmonary vascular diseases

PP-077

Right ventricular epicardial fat tissue thickness predicts right ventricular dysfunction in acute pulmonary thromboembolism

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Background and Aim: Pulmonary thromboembolism (PTE) manifests with an acute increase in right ventricular afterload leading hemodynamic compromise. With neurohormonal activation, right ventricular (RV) function determines the course of PTE. As a metabolically active organ, epicardial fat tissue (EFT) is a rich source of inflammatory cytokines and neurohumoral mediators which might be associated with right ventricular dysfunction (RVD). In this study, we aimed to evaluate the relationship between epicardial fat tissue thickness (EFT) and RVD in PTE patients.

Methods: In our retrospective cross-sectional study, we included consecutive 92 patients who were diagnosed with PTE, using computerized tomography pulmonary angiography. Patients' thromboembolic risk factors, laboratory parameters, echocardiographic and tomographic data were recorded. Axial images were used to measure the right ventricular and left ventricular diameter and right to left ventricular diameter ratio greater than 1.0 was regarded as RVD. PTE obstruction severity was calculated by Qanadli score. In axial 4 chamber view, the right and left atrioventricular groove and interventricular groove EFTs were identified. Maximal thicknesses of the measurements for these three regions were measured.

Results: RVD was observed in 40 patients. Patients with RVD were older (69.1±14.3 vs. 62.6±15.4) and mostly presented with hypotension (22.5% vs. 3.8%). Plasma glucose and troponin values were higher in patients with RVD. Systolic pulmonary artery pressure and tricuspid regurgitation severity were higher in patients with RVD. Qanadli score was 16% higher and mosaic attenuation and tomography detected pericardial effusion were more prevalent in patients with RVD. All of the epicardial fat tissue thicknesses were significantly greater in patients with RVD. Right ventricular groove EFT (RV EFT) produced most significant difference (18.6±4.7 vs. 14.7±3.3, p<0.001) between two groups. RV EFT had positive weak correlations with age (r=0.289), troponin (r=0.276) and Qanadli Score (r=0.269) and also has positive moderate correlation with right to left ventricular ratio (r=0.432). Adjusted with other parameters including pericardial effusion presence (odds ratio=7.7) and interventricular septum flattening (odds ratio=15.4), 1 mm increase in RV EFT was associated with 34.7% increased odds of RVD (p<0.001) (Nagelkerke R-squared=57.3%). In ROC analysis, a cut-off value 16.8 mm of RV EFT had a 70.0% sensitivity and 69.2% specificity for discrimination of RVD (AUC=0.750, 95% CI: 0.646-0.853). In a second model, RV EFT ≥16.8mm was associated with 5.2 times the odds of RVD presence (p<0.001, Nagelkerke R-squared 19.3%).

Conclusions: EFT, as a source of miscellaneous neurohumoral and inflammatory mediators, is associated with RVD in PTE patients. This noninvasive, simple tomographic measurement may guide us at management of patients with PTE.

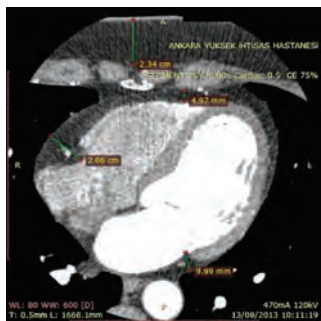


Figure 1.

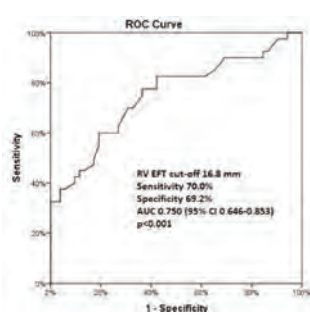


Figure 2.

Pulmonary hypertension / Pulmonary vascular diseases

PP-078

Successful treatment of late-onset pulmonary hypertension after atrial septal defect operation with macitentan: Single center experience

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Background and Aim: Macitentan significantly improves pulmonary hemodynamics and survival in patients with primary pulmonary hypertension (PPH). Its beneficial effect, however, may be blunted due to the adverse impacts such as anemia and peripheral edema. Pulmonary arterial hypertension (PAH) is a significant consequence of congenital heart disease (CHD). Its presence and severity are associated with increased morbidity and mortality. We tried to evaluate that the effectiveness of the macitentan in patients with late-onset pulmonary hypertension after atrial septal defect operation in our center.

Methods: The effect of a single dose of macitentan (10 mg) on pulmonary hemodynamics, functional capacity was examined in four patients with late-onset pulmonary hypertension after atrial septal defect operation.

Results: The macitentan significantly improved mean pulmonary artery pressure (MPAP), cardiac output (CO), TAPSE, RVS', 6-minute walking test and NT-proBNP levels compared with before treatment (Table 1).

Conclusions: Macitentan can be used in patients with late-onset pulmonary hypertension after shunt operation especially atrial septal defect.

Table 1. Haemodynamic values in patients with acquired pulmonary arterial hypertension at baseline and after treatment with macitentan

	1.Patient	2.Patient	3.Patient	4.Patient	P value
Age	41	71	68	36	
Gender	Female	Female	Female	Female	
Shunt Operation	ASD	ASD	ASD	ASD	
Operation Time(year)	12	19	11	7	
MPAP(mmHg)	28-15	30-20	25-15	30-20	<0.05
CO(L/min)	2.7-3.5	2.2-3.6	2.3-3	2.6-3.5	<0.05
PVR(Wood)	5-2	4-1	4-1	7-2	<0.05
Nt-ProBNP(pg/dl)	450-205	550-201	480-180	425-115	<0.05
TAPSE	11-17	13-16	13-17	11-18	<0.05
RVS'(cm/s)	6-9	7-11	7-13	8-12	<0.05
sPAB(mmHg)	50-30	55-27	52-25	60-30	<0.05
6-MWT(meters)	225-450	300-485	285-425	300-510	<0.05

Cardiac imaging / Echocardiography

PP-079

Right atrial strain as evaluated by 2D -speckle-tracking in systemic sclerosis patients

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Background and Aim: Cardiac involvement is a common clinical finding in systemic sclerosis (SSc) and is associated with poor prognosis. Collagen deposition in the myocardium leads to diastolic dysfunction with increased mortality. Right atrial dysfunction and remodeling can be an earlier sign of diastolic dysfunction. The aim of this study is to investigate the right atrial phasic functions assessed by two dimensional (2D) speckle-tracking echocardiography.

Methods: Fifty patients with SSc (mean age:49.6±10.1 years, 45 female) and 25 age and sex matched controls (mean age: 44.4±11.7 years, 18 female) were consecutively enrolled into the study. All study subjects underwent conventional two dimensional echocardiography and speckle tracking echocardiography. Echocardiographic examination was performed with a Philips EPIQ 7 Ultrasound System with 3.5 MHz probe. Both reservoir and conduit right atrial functions were measured (Fig 1).

Results: In SSc group, ten (10) patients had systolic pulmonary pressure ≥40 mm Hg. Conventional echocardiographic parameters of the SSc patients and controls are listed in Table 1. Tricuspid E/A was significantly lower in SSc patients compared to controls (1.14±0.32 vs 1.41±0.50, p=0.023) whereas Tricuspid E/e' did not differ significantly (4.8±0.14 vs 4.9±0.01, p=0.83). Right atrial peak longitudinal strain values in control group, SSc patients without pulmonary hypertension and SSc patients with PHT were as follows: % 37.8±9.9 vs % 36.1±11.1 vs % 26.6±11.1. Right atrial reservoir function decreased significantly in SSc patients with pulmonary hypertension. The conduit strain values did not differ significantly between the groups.

Conclusions: The main finding of this study is that right atrial dysfunction, especially reservoir function, is significantly impaired in systemic sclerosis patients with pulmonary hypertension. Pressure overload induces right atrial dysfunction which can be easily assessed by 2D speckle-tracking echocardiography before conventional echocardiographic parameters deteriorate.

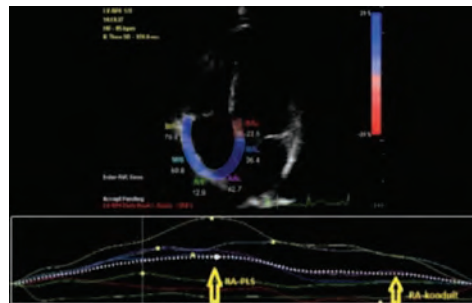


Figure 1. Right Atrial Strain Measurement by 2D Speckle-Tracking Method. PLS: Peak longitudinal strain.

Table 1. Conventional echocardiographic parameters of the study population

	SSc (n=50)	Control (n=25)	p
Tricuspid E (m/sec)	0.53± 0.13	0.59± 0.14	0.114
Tricuspid A (m/sec)	0.49± 0.16	0.44± 0.10	0.142
Tricuspid e' (cm/sec)	11.3± 2.7	12.1± 3.0	0.271
Tricuspid a' (cm/sec)	14.9± 3.4	15.3± 4.1	0.702
Tricuspid E/A	1.14± 0.32	1.41± 0.50	0.023
Tricuspid E/e'	4.8± 0.14	4.9± 0.01	0.830
Deceleration Time (m/sec)	117.2± 31.1	125.3± 30.2	0.288
Mitral E/e'	6.9± 1.96	5.8± 2.8	0.067

Cardiac imaging / Echocardiography

PP-080

The effect of smoking on left atrial volume and phasic functions in healthy subjects

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Background and Aim: Smoking is a risk factor for cardiovascular diseases. It is associated with activation of the autonomic nervous system, oxidative stress, inflammation and endothelial dysfunction and may result in significant alteration in the diastolic functions of both ventricles. The aim of this study was to evaluate the left atrial volume and phasic functions in apparently healthy smokers.

Methods: Forty healthy smokers and 20 healthy nonsmokers were consecutively included in the study. None of the subjects had additional cardiovascular risk factor other than smoking. The left atrial and ventricular functions were assessed by speckle tracking echocardiography.

Results: The echocardiographic parameters of the smokers and nonsmokers are listed in Table 1. The smokers had significantly larger left atrial volumes. The left atrial reservoir and conduit strain were significantly lower in healthy smokers compared to those of nonsmokers. Similarly, left ventricular global longitudinal strain was lower in smokers although the left ventricular ejection fraction was similar.

Conclusions: Smoking impairs left atrial and ventricular functions even in apparently healthy young people with no other additional cardiovascular risk factors. Speckle tracking echocardiography is useful in detecting subclinical left atrial and ventricular dysfunction in healthy smokers.

Table 1.

	Smokers (n= 40)	Nonsmokers (n= 20)	p
Age (years)	34.4 ± 7.8	26.5 ± 3.4	<0.001
Male sex (n-%)	28 (70%)	9 (45%)	0.060
LAVmax (mL)	32.3 ± 11.6	26.4 ± 7.2	0.044
LAVmin (mL)	10.7 ± 4.6	9.0 ± 3.0	0.215
LAVpre-A	18.2 ± 6.4	14.8 ± 4.5	0.040
LA total stroke volume (mL)	21.6 ± 9.1	17.4 ± 6.2	0.045
LA total emptying fraction (%)	65.9 ± 11.0	65.1 ± 9.8	0.461
LA passive stroke volume (mL)	14.1 ± 7.0	11.5 ± 4.9	0.170
LA passive emptying fraction (%)	42.5 ± 11.4	43.1 ± 10.8	0.938
LA active stroke volume (mL)	7.4 ± 3.1	5.9 ± 2.5	0.048
LA active emptying fraction (%)	41.6 ± 10.8	39.3 ± 10.8	0.410
LA expansion index	225.5 ± 118.3	215.2 ± 113.6	0.461
Left atrial reservoir function (%)	38.9 ± 10.0	46.8 ± 12.1	0.012
Left atrial conduit function (%)	18.3 ± 7.2	22.9 ± 8.5	0.029
Left ventricular ejection fraction (%)	54.9 ± 4.2	55.6 ± 3.7	0.308
Left ventricular global longitudinal strain (%)	-19.4 ± 2.3	-21.8 ± 1.9	<0.001

LA: left atrium, LAV: left atrial volume

Pulmonary hypertension / Pulmonary vascular diseases

PP-081

Prevalence of pulmonary hypertension in patients with Sarcoidosis: A single center experience

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Background and Aim: Sarcoidosis is a systemic granulomatous disease with unknown etiology. Pulmonary hypertension (PH) is recognized as a complication of advanced pulmonary sarcoidosis and is associated with increasing mortality. However, the exact prevalence of PH in patients with sarcoidosis is unclear. The aim of the study was to assess the prevalence and the predictors of PH in sarcoidosis.

Methods: 56 patients with biopsy proven sarcoidosis without known sarcoid-related or structural heart disease or alternative etiologies of PH were included. All patients were studied prospectively by transthoracic echocardiography to evaluate the level of probability of PH according to guidelines. In assumed intermediate to high level probability of PH, right heart catheterization (RHC) was performed. Clinical data, including functional capacity using New York Heart Association class, results of pulmonary function testing were recorded. To assess the functional capacity, six-minute walk distance (6 MWD) used for all patients.

Results: In 8 of the 56 patient (14.3%) had intermediate to high level echocardiographic probability for PH (Table 1). Two patients refused undergoing RHC. Of the six patients investigated with RHC, three showed combined pre and postcapillary PH. The prevalence of PH is found 5.4% (3/56). All three patients demographic, clinical, laboratory and echocardiographic parameters are presented on Table 2. Two patient with PH had radiologic stage IV. All three patients with PH had low diffusing capacity of the lung for carbon monoxide (DLCO) level (<65%) and low 6MWD (<350 m). Without significant systolic and diastolic dysfunction (EF >50%, E/e' <14), three patient with PH had high NT-proBNP level (>125 pg/ml) and markedly low right ventricular free wall strain (RV FWS) and left ventricular longitudinal strain (LV GLS).

Conclusions: The prevalence of PH was found 5.4% and all patients had postcapillary PH although had precapillary components. However known echocardiographic parameters using to assess probability of PH, NT-proBNP, DLCO, 6MWD, RV FWS and LV GLS can be useful predictors to detect sarcoidosis-related PH earlier.

Table 1. Echocardiographic probability of pulmonary hypertension of patients and controls

Echocardiographic probability of PH	Controls (n=26)	Patients with Sarcoidosis (n=56)
Low	26 (100%)	48 (85.7%)
Intermediate	-	5 (8.9%)
High	-	3 (5.4%)
mean PAP ≥ 25 mm Hg	-	3 (5.4%)

Mean PAP, mean pulmonary artery pressure measured by right heart catheterization; PH, pulmonary hypertension.

Table 2. Demographic, clinical, laboratory and echocardiographic parameters of PH patients

PH patients	1	2	3
Echocardiographic probability of PH	High	Intermediate	High
Age (year)	63	62	67
Gender	Female	Female	Female
Body mass index (kg/m ²)	36.2	41.6	28.4
Hypertension	+	-	-
Radiological stage	IV	I	IV
Duration of sarcoidosis (year)	4	10	8
Steroid usage	+	+	+
NHYA class	IV	II	IV
DLCO (%)	62	63	61
Desaturation during 6 MWD	+	-	+
NT-proBNP (pg/ml)	314	3551	2065
LV ejection fraction (%)	61	74	66
E/e' ratio	7.3	7.5	10
TAPSE	21	18.9	16
FAC (%)	32.6	29.6	23.8
RV ejection fraction (%)	30	27.9	40.7
RV free wall strain (%-)	13	9.8	10.3
RA reservoir function (%)	13	24	15.6
LV global longitudinal strain (%-)	17.7	13.2	13.4
mean PAP (mm Hg)	50	33	45
PVR (wood.U)	6	3	13
PCWP (mm Hg)	22	21	18
DPG (mm Hg)	10	0	13
CI (L/min./m ²)	1.9	1.7	2.03

6 MWD, six minutes walk distance; CI, cardiac index; DLCO, diffusing capacity of carbon monoxide; DPG, diastolic pulmonary gradient; FAC, fractional area changes; LV, left ventricle; NYHA, New York Heart Association; PAP, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RA, right atrium; RV, right ventricle; RV MPI, right ventricular myocardial performance index; TAPSE, tricuspid annular plane systolic excursion

Cardiac imaging / Echocardiography

PP-082

Epicardial adipose tissue thickness in urolithiasis patients

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Background and Aim: We aimed to assess the relationship between urinary stone disease which is accepted as a component of metabolic syndrome and epicardial adipose tissue (EAT) thickness.

Methods: Fifty-three patients with KS disease and 39 healthy subjects were enrolled in our study. Patients were excluded if they had inadequate view on echocardiography, a history of any kind of cardiovascular disease, active infection and history of uric acid, cystin or struvite stones. For this reasons 8 KS patients were excluded because of cardiovascular disease in 4, and insufficient echocardiographic view in 4. Echocardiograms were performed with a Vivid 7 (Vingmed electronic, GE, Horten, Norway) instrument according to standard techniques. The diagnosis of Kidney stone (KS) was established on the basis of the results of urinary ultrasound (Toshiba Aplio XG, Japan) using a 3.5 MHz transducer. Renal calcification was classified as a urinary stone if the calcification was located in the collecting system. Stone burden was also determined. All evaluations were performed by experienced radiologists, who did not have any information about metabolic status of the patients.

Results: Forty-five patients with KS disease and 39 healthy subjects were included in this study. Of the 84 patients included in the final analysis, 44% were men and 56% were women. Mean age was 50.52±10.4 years. Mean BMI was 25.5±3.4 kg/m². EAT thickness were higher (p<0.001) in KS disease patients than in healthy subjects. Multivariable analysis showed that increased EAT thickness was associated with family history of urolithiasis. EAT thickness was also significantly correlated with triglyceride levels (r=0.627, p<0.001).

Conclusions: In conclusion, the relationship between KS disease and EAT thickness was researched in this study and EAT thickness was found to be higher in KS disease patients than in controls. We suggest that urolithiasis should be considered as a component of metabolic syndrome and EAT thickness may be useful to detect early atherosclerosis in urolithiasis patients.



Figure 1.

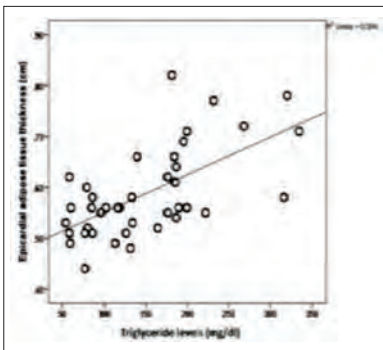


Figure 2.

Table 1. Results of multiple regression analysis of patients population

	β value	p value
age	0,327	0,008
family history	0,380	0,004
triglyceride	0,379	0,005

Table 1.

Parameter	
Age (years old)	59.5±13.7
Sex (%)	16 Female (55.2%) 13 Male (44.8%)
Median FU (month)	44 (1-113)
Initial NYHA FC III/IV (%)	75.8
Initial NYHA FC III/IV (%)	41.4
TTE	mean sPAP 66.1±26.7 mmHg TAPSE 1.6±0.4 mm
Initial 6MWD (meter)	321.4±119.9
Final 6MWD (meter)	356.1±132.8
RHC	Mean sPAP 79.4±22.9 mmHg Mean dPAP 35.1±13.2 mmHg Mean mPAP 50.9±16.1 mmHg Mean RA 15.1±5.7 mmHg Mean PCWP 14.3±5.6 mmHg Mean CO 4.8±1.5 L/per minute Mean PVR 8.6±5.9 WU
Initial NT-proBNP (pg/ml)	2468 (46.1 - 20.564)
Final NT-proBNP (pg/ml)	2382 (67.0 - 23.368)
Pulmonary endarterectomy	17/29 (58.6%)
Medical therapy	12/29 (41.4%)
10 year survival (%)	58.6

Characteristics of study group.

Pulmonary hypertension / Pulmonary vascular diseases

PP-083

The ten years outcome of CTEPH patients in a university hospital

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Background and Aim: Chronic thromboembolic pulmonary hypertension (CTEPH) is one of the leading cause of pulmonary hypertension (PH). We aimed to investigate the outcome of CTEPH patients were followed up at PH outpatient department of our institution.

Methods: We screened medical records of PH patients were followed up at our university hospital between 2009 and 2018. 29 patients with a diagnosis of CTEPH were included in the study.

Results: The study group consisted of 16 female (55.2%) and 13 male (44.8%). The mean age was 59.5±13.7 years old. Median follow up duration was 44 month (1-113 month). Most of the patients (22/29, 75.8%) were initially in NYHA FC III or IV. Mean systolic PAP was 66.1±26.7 mm Hg, mean TAPSE was 1.6±0.4 mm on TTE. Three patients had pericardial effusion. The mean initial 6 minute walking distance (6MWD) was 321.4±119.9 meters, and it increased at follow up to 356.1±132.8 meters. On RHC, The systolic, diastolic and mean PAP were 79.4±22.9 mm Hg, 35.1±13.2 and 50.9±16.1 mm Hg, respectively. The mean PVR was 8.6±5.9 WU, the mean PCWP was 14.3±5.6 mm Hg, mean RAP was 15.1±5.7 mm Hg and mean cardiac output was 4.8±1.5 L/per minute. On lab examination the median initial NT-proBNP was 2468 pg/ml (46.1-20.564 pg/ml), the median final NT-proBNP was 2382 pg/ml (67.0-23.368 pg/ml). All patients were taking oral anticoagulant therapy. Pulmonary endarterectomy was performed in seventeen of 29 patients (58.6%). Twelve patients were not operated. One patient did not accept the surgical treatment. The other one who had chronic thromboembolic disease (CTED) without PH was asymptomatic (NYHA FC I). The last patient who was diagnosed as CTEPH one month before has been evaluating for surgical operability and have not been operated yet. The other 9 patients have not been operated due to sub-segmenter disease and/or multiple comorbidities. None of them had balloon pulmonary angioplasty (BPA). Twelve patients who were not operated were taking PAH specific treatment. The survival rate at 10 years follow up was 58.6%. Twelve patients died during ten years follow up. Seven of 12 patients who were not operated died (58.3%) Out of 17 patients who were operated four died intra-operatively and only one who had residual PH after pulmonary endarterectomy died 45 months after surgery due to respiratory failure. After exclusion of patient who died during surgical procedure, survival improved with pulmonary endarterectomy. In univariate analysis age, higher systolic PAP and lower TAPSE on TTE, lower final 6MWD, higher final NT-proBNP, presence of final NYHA FC III or IV were associated with mortality. In multivariate analysis the association was still present for age, lower final 6MWD, higher final NT-proBNP and presence of final NYHA FC III or IV.

Conclusions: The prognosis of CTEPH is getting better with successful endarterectomy procedure. Age, lower functional capacity (NYHA FC III, IV and low 6MWD) and high NT-proBNP are the predictors of mortality.

Heart failure

PP-084

Investigation of relation between BNP and proadrenomedullin, copeptin and oxidative stress in the blood from patients who have heart failure with reduced ejection fraction

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Background and Aim: In this study, it was aimed to investigate the relationship between BNP and proadrenomedullin, copeptin and oxidant/antioxidant system markers in the patients diagnosed with reduced ejection fraction heart failure (REF-HF) and to determine whether these markers can be used in the diagnosis or follow-up of the disease.

Methods: For this aim, 72 patients older than 18 years old were included into the study. They were diagnosed with REF-HF for more than 1 year, followed at Ankara University cardiology clinics as inpatient or outpatient from September 2016 to May 2017. Suitable blood samples for BNP, proadrenomedullin, copeptin, erythrocyte and plasma oxidant/antioxidant system markers were taken from the patients on the same day. Plasma BNP levels were measured by chemiluminescence method, serum proadrenomedullin and copeptin levels were measured by ELISA method. Erythrocyte and plasma oxidant/antioxidant system markers were measured by using spectrophotometric methods.

Results: It was found that BNP showed insignificant positive weak correlation with proadrenomedullin and copeptin. On the other hand, we found that BNP was significantly positively correlated with erythrocyte superoxide dismutase (SOD) enzyme activity ($r=0.31$; $p=0.009$) and significantly negatively correlated with erythrocyte xanthine oxidase (XO) enzyme activity ($r=-0.25$; $p=0.03$). BNP was not significantly correlated with other oxidant/antioxidant system markers. However, there was highly significant positive correlation between proadrenomedullin and copeptin.

Conclusions: In conclusion, it is not suitable to use serum proadrenomedullin and copeptin measurements instead of plasma BNP in diagnosis or follow-up of patients with REF-HF. It may be suggested that BNP can play a protection role against oxidant injury by increasing erythrocyte SOD activity and decreasing erythrocyte XO activity in patients diagnosed with REF-HF. Additionally, it may be useful to combine erythrocyte SOD and XO enzyme activities together with plasma BNP in monitoring of REF-HF development and its complications.

Lipid / Preventive cardiology

PP-085

The effects of health warning labels on cigarette packages on patients who apply to cardiology clinics

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Background and Aim: Health warning labels (HWLs) on cigarette packages, which describe the health problems that may be caused by smoking, have been used in an effort to prevent smoking for many years. At first glance, HWLs on cigarette packages seem to be insignificant; however, a study conducted in Korea in 2017 showed the situation is quite the opposite. This study showed that HWLs reduce smoking in men by 4.79% and women by 0.66%. As a result of this effect, it is estimated that within ten years a reduction in the incidence of 85238 diabetes mellitus, 67948 chronic obstructive pulmonary diseases, 31526 ischemic heart diseases, 21036 lung cancers, and 3972 oral cancer cases is expected. There are no studies in Turkey that investigate how HWLs used abroad have an impact on the population, and especially on patients who applied to the cardiology outpatient clinic. In this study, we investigated the effect of HWLs on patients admitted to the cardiology outpatient clinic and compared the HWLs used in our country with those used abroad which we considered to be harsh and striking.

Methods: In this descriptive study, a questionnaire consisting of 45 questions was administered to 239 patients who applied to the cardiology outpatient clinic. 14 different visuals used in our country and 9 different visuals used in foreign countries. The questions were addressed through these posters.

Results: In our study, 28.9% of the participants who applied to cardiology outpatient clinic have never smoked, 36.0% of them still smoked and 35.1% of them were ex-smokers. The mean age at initiation of cigarette smoking for those who were still smokers or ex-smokers was 17.8±4.8 (median: 17, Min-Max: 5-35 years), and the mean of active smoking period was 23.6±16.1 (median: 20, Max: 5-62) years. 23.8% declared that there were images they were not familiar with among the HWLs used in Turkey. 57.7% of the survey participants stated that the HWLs on cigarette packages were effective and 90.8% of the participants stated that warnings in foreign countries were more effective than the ones in Turkey.

Conclusions: This study shows that even among patients who have applied to cardiology clinics, smoking is still common. When compared to the HWLs used in our country, the ones used abroad were found to be more effective by the majority of the participants, which shows that the use of harsher and more striking HWLs and their periodic renewal seem to be more effective.



Figure 1. Health warning labels in Turkey.

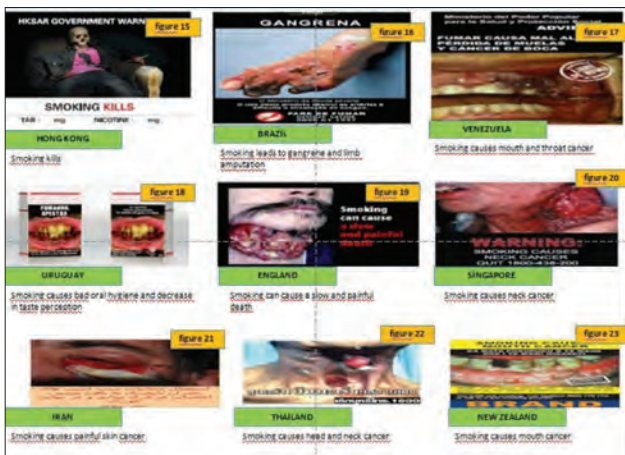


Figure 2. Health warning labels in foreign countries.

Table 1. The sociodemographic characteristics of the study group and distribution of smoking status, Çanakkale 2017

Sociodemographic Feature	
Gender	n %
Male	144 60,3
Female	95 39,7
Education Status	
Primary education and lower	109 45,6
High school and over	130 54,4
Marital Status	
Married	173 72,7
Single / Widowed / Divorced	66 27,3
Income Status	
Low	23 9,7
Average	160 66,9
High	56 23,4
Cigarette or any of tobacco product use	
Still smoking	86 36,0
Ex-smoker	84 35,1
Never smoked	69 28,9
Total	239 100,0

Table 2. Considerations about graphic warning labels, Çanakkale 2017

Variables	n	%
Do you think pictorial warnings on cigarette packs are effective to prevent people from smoking? (n=239)		
Yes	138	57,7
No	101	42,3
How do you think pictorial warnings should be? (n=157)		
On one side of the pack	26	16,6
On both sides of the pack	131	83,4
Do you think written warnings on cigarette packs are effective to prevent people from smoking? (n=237)		
Yes	119	50,2
No	118	49,8
How do you think written warnings should be? (n=148)		
On one side of the pack	36	24,3
On both sides of the pack	112	75,7
Which kind of warning is more important to prevent people from smoking: Pictorial or written warnings? (n=203)		
Pictorial warnings are more important	104	51,2
Written warnings are more important	17	8,4
Both are equally important	82	40,4
Evaluate the pictures used in foreign countries. According to those in Turkey, assess how they affect you. (n=239)		
They did not affect	21	8,8
Indifferent	1	0,4
They affected more	217	90,8

Lipid / Preventive cardiology

PP-086

Proportional serum lipid parameters in coronary slow flow phenomenon

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Background and Aim: The association between serum lipid parameters and coronary slow-flow (CSF) phenomenon has been searched previously. The aim of our study was to determine the association between proportional serum lipid parameters and CSF.

Methods: We enrolled 93 stable patients who had undergone coronary angiography and had near-normal coronary arteries with normal and slow coronary flow. Demographic variables, clinical features and laboratory data were recorded retrospectively from the local database. Coronary flow velocity was evaluated by thrombolysis in myocardial infarction (TIMI) frame count (TFC). CSF phenomenon was defined as a TFC greater than 27 frames.

Results: Fifty four patients were in CSF group and 39 patients were in control group. The mean age of the patients in the CSF group was significantly higher to those of the control group (55.4±9.5 vs. 50.5±9.8 years, p=0.019). Triglyceride (TG) levels was found higher in CSF group than control group (187.3±103.4 mg/dl, 125.3±63.8 mg/dl, p<0.001). Total cholesterol to high density lipoprotein cholesterol ratio (TC/HDL-c), TG/ HDL-c, low density lipoprotein cholesterol to HDL-c ratio (LDL-c/HDL-c) and non-HDL-c levels were significantly higher in CSF group (p=0.007, p=0.004, p=0.044, p=0.018, respectively). In multivariate logistic regression analysis age, smoking and the value of TG cut off obtain from ROC analysis were found statistically significant to predict of CSF. The value of TG cut off obtain from ROC analysis was found most associated with the presence of CSF (OR: 7.516, 95% CI: 2.323-24.323, p=0.001).

Conclusions: Higher TC/HDL-c, TG/HDL-c, LDL-c/HDL-c, TG and non-HDL-c levels were related with CFS phenomenon.

Table 1. Comparative analysis of laboratory parameters between two groups

Variables	CSF group (N=54)	Control group (N=39)	p
Urea (mg/dL)	32.2 ± 9	27.6 ± 8.7	0.016
Creatinine (mg/dL)	0.87 ± 0.19	0.80 ± 0.17	0.064
Glucose (mg/dL)	123.6 ± 53.3	105.6 ± 32.0	0.047
TC (mg/dL)	201.7 ± 44.3	184.2 ± 44.1	0.064
LDL (mg/dL)	122.1 ± 37.8	111.8 ± 35.2	0.185
HDL (mg/dL)	44.8 ± 10.5	47.5 ± 12.8	0.256
Triglyceride (mg/dL)	187.3 ± 103.4	125.3 ± 63.8	<0.001
TC/ HDL-c	4.65 ± 1.11	4.02 ± 1.05	0.007
TG/ HDL-c	4.43 ± 2.72	2.93 ± 1.96	0.004
LDL/ HDL-c	2.81 ± 0.89	2.44 ± 0.84	0.044
Non-HDL-c (mg/dL)	156.8 ± 40.5	136.6 ± 38.7	0.018

Cardiovascular surgery

PP-087

Is the CHA2DS2-VASc score predictor for in-hospital mortality after isolated coronary artery by pass grafting surgery

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Background and Aim: CHADS2 and CHA2DS2-VASc scores are proposed as a predictor for immediate and late stroke after CABG, there is no data evaluating the prognostic value of CHA2DS2-VASc score in patients undergoing isolated CABG surgery. When compared the mostly used traditional risk models such as Euroscore system and the Society of Thoracic Surgeons (STS) 2008 Cardiac Surgery Risk Model, it provides fast and simple method for physicians in risk evaluation that requires no calculator or computers. So, in our study we aimed to examine the prognostic value of CHA2DS2-VASc scores in individuals undergoing isolated CABG surgery.

Methods: Study population were retrospectively and consecutively analysed by using our database collected as a part of routine clinical practice. The primary endpoint of this study was MACE which is defined as composite of in hospital mortality, perioperative nonfatal myocardial infarction (MI) and stroke. Secondary endpoints were perioperative complications such as acute renal failure, acute atrial fibrillation, bleeding revision or mediastinitis. In hospital mortality was defined as death from all causes during per- and postoperative hospitalization. Since all patients underwent coronary bypass surgery, multiple coronary vessel disease at index hospitalization was not taken into account. After the CHA2DS2-VASc score was calculated, study population were divided into two groups as high (CHA2DS2-VASc score <2) and low (CHA2DS2-VASc score ≥2) score groups. Preoperative risk stratification was performed for all patients by using the EuroSCORE system using the downloaded version from euroscore.org and noted on a patient file. The severity of coronary stenosis was also quantitatively evaluated using the Syntax scoring system by 2 experienced interventional cardiologists who were blinded to study by using the downloaded version from www.syntaxscore.com.

Results: Clinical, laboratory and operative parameters were presented table 1 and table 2. High score group had significantly higher in hospital mortality and major adverse cardiac events (MACE) rates (p<0.001, p<0.001, respectively). High SYNTAX II CABG and CHA2DS2-VASc scores were predictor for MACE in univariate logistic regression analysis (Table 3). ROC curve analysis demonstrated that CHA2DS2-VASc greater than 2 had 52.6% sensitivity and 84.1% specificity to predict MACE (Table 4).

Conclusions: We demonstrated that high CHA2DS2-VASc score was predictor for MACE with 52.6% sensitivity and 84.1% specificity.

Table 1. Demographic, clinical and laboratory characteristics of groups

	CHA ₂ DS ₂ -VASc		p
	<2 n=238	≥2 n=226	
Sex (Female), n (%)	44 (18.5)	49 (19.9)	0.32
Age (years)	57 (52-63)	64 (55-67)	<0.001
Body mass index (kg/m ²)	27 (21.3-29.7)	26.1 (24.2-29.5)	0.19
Smoking, n (%)	99 (41.6)	82 (36.3)	0.24
DM, n (%)	19 (8)	143 (63.3)	<0.001
HT, n (%)	38 (16)	147 (65)	<0.001
COPD, n (%)	34 (14.3)	38 (16.8)	0.45
PAD, n (%)	14 (5.9)	43 (19)	0.014
CAD, n (%)	36 (15.1)	41 (18.1)	0.38
Stroke, n (%)	14 (5.9)	9 (4)	0.35
Ejection Fraction (%)	55 (50-60)	45 (40-60)	0.03
Total Cholesterol	240 (200-356)	226 (208-310)	0.47
LDL-C	116 (99.5-175)	116 (100-175)	0.94
HDL-C	45 (42-49)	45 (42-51.8)	0.48
CRP	5.5 (4-10)	5 (4-9)	0.70
EUROSCORE	3 (2-4)	3 (2-4)	0.53
SYNTAX score	19 (14-25.5)	19 (13-23.5)	0.40
SYNTAX II score (CABG)	23.8 (21.3-28)	25.7 (21.7-35)	0.001

DM, diabetes mellitus; HT, hypertension; COPD, chronic obstructive pulmonary disease; PAD, peripheral artery disease; CAD, coronary artery disease; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol

Table 2. Operative and postoperative parameters of groups

	CHA ₂ DS ₂ -VASc		p
	<2 n=238	≥2 n=226	
By-pass number	3 (2-3)	3 (2-3)	0.36
CPB Time (min)	92.5 (47-96)	51 (47-96)	0.16
Clamp Time (min)	50 (26-55)	48 (26-56)	0.58
Intra-aortic balloon pump, n (%)	3 (1.3)	14 (6.2)	0.005
Extubation Time (hours)	7 (5-9.5)	7 (5-10)	0.63
Bleeding Revision, n (%)	9 (3.8)	14 (6.2)	0.23
Hemorrhage (ml)	500 (350-600)	450 (350-600)	0.73
Sternal Dehiscence, n (%)	13 (5.5)	10 (4.4)	0.724
Wound Infection, n (%)	12 (5)	12 (5.3)	0.89
Mediastinitis, n (%)	0 (0)	12 (5.3)	<0.001
Acute renal failure,	1 (0.4)	14 (6.2)	<0.001
Acute atrial fibrillation, n (%)	27 (11.3)	25 (11.1)	0.92
Transient ischemic attack, n (%)	0 (0)	4 (1.9)	0.056
Stroke, n (%)	1 (0.4)	4 (1.8)	0.34
Post-operative MI, n (%)	0 (0)	7 (3.1)	0.006
In-hospital Mortality, n (%)	0 (0)	14 (6.2)	<0.001
MACE, n (%)	1 (0.4)	18 (8)	<0.001
Intensive care unite time (day)	2 (2-2)	2 (2-3)	0.91
Hospitalization time (day)	5 (5-6)	5 (5-6)	0.23

MACE, major adverse cardiac events

Table 3. Univariate analysis of major cardiac events

	OR	95% CI	p
SYNTAX I	1.003	0.977-1.029	0.83
SYNTAX II PCI	1.008	0.993-1.024	0.30
SYNTAX II CABG	1.068	1.017-1.122	<0.01
CHADSVASC	2.030	1.413-2.916	<0.01

Table 4. CHA2DS2-VASc greater than 2 had 52.6% sensitivity and 84.1% specificity to predict MACE [area under curve: 0.752, p<0.01, 95% CI (0.710-0.790)]

Criterion	Sensitivity	95% CI	Specificity
=0	100,00	82,4 - 100,0	0,00
=1	94,74	74,0 - 99,9	19,96
=2	84,21	60,4 - 96,6	55,38
=3	52,63	28,9 - 75,6	84,08
=4	15,79	3,4 - 39,6	95,96
=5	5,26	0,1 - 26,0	98,88
=6	0,00	0,0 - 17,6	99,78
=7	0,00	0,0 - 17,6	100,00

Lipid / Preventive cardiology

PP-088

Is elevated triglyceride high density lipoprotein cholesterol ratio a risk factor that causes acute coronary syndrome to appear earlier in life?

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Background and Aim: With an upward trend in changing to unhealthy lifestyle, acute myocardial infarction (AMI) in young adults became a growing public health problem. To date, there has been no detailed study that analysis the triglyceride to high density lipoprotein cholesterol ratio (Tg/HDL) of patients presenting with myocardial infarction (MI) at a young age. The purpose of this study was to assess the relation between Tg/HDL ratio and AMI in young adults.

Methods: A total of 621 patients (60.8% aged ≤50 years and 39.2% aged >50 years) who underwent coronary angiography (CAG) because of MI in our hospital were included in this study. Demographic characteristics, risk factor profile, laboratory test results, electrocardiographic and CAG findings were assessed in the selected groups.

Results: Total cholesterol, Tg levels, Tg/HDL ratio were higher in younger patients with MI while glucose and HDL levels were lower. No statistically significant difference was observed in LDL levels between two groups. In the matched population, using propensity score matching comparing young patients with AMI and older AMI patients, serum Tg levels [179 (145-231) vs 148 (101-197)] and Tg/HDL ratio [5.8 (4.1-9.1) vs 3.0 (1.8-4.6)] were significantly higher whereas dramatically lower HDL levels were observed (32.6±8.2 vs 41.7±8.8).

Conclusions: This study demonstrated that Tg/HDL ratio may be an important predictor for occurrence of acute coronary syndrome in young adults.

Lipid / Preventive cardiology

PP-089

The impact of a structured enhanced education and follow-up program in an underserved population

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Background and Aim: Prior studies suggest that primary prevention programs for coronary artery disease (CAD) may be effective in improving health-related behavioral outcomes. However, the implementation of these programs can be costly mainly due to staffing. Maintaining these programs with limited resources can be very challenging. Thus, the present study was designed to assess the feasibility and effectiveness of a structured, enhanced education and follow-up program for CAD prevention in an area where the diverse population and economy are major problems.

Methods: SANKO Trial took place between 2014 and 2018 and had 2 different education and training phases;

in the first phase, 2nd year Medical students underwent a one-year, specially designed, training program on primary prevention for CAD. In the second phase, a series of conferences on primary prevention for CAD were organized by the SANKO University and local municipalities for underserved populations. Participants were prospectively assigned to an intervention where pre and post conference knowledge were collected and assessed. Every intervention was conducted by specially trained 3rd year Medical students and an education booklet which was specifically designed for this study was given to the participants Every month thereafter, for 6 months, each participant was followed by phone. At the 6 month follow up, data was collected to assess the impact of enhanced education and follow-up program on behavioral outcomes.

Results: A total of 112 participants were enrolled; 83% were women, mean age was 41±13 years, only 27% had a graduate school degree; 59% were not working. Mean BMI was 28.7±5.8 kg/m². Overall knowledge on CAD risk factors, primary prevention measures, diet and daily exercise habits were very poor. After the enhanced education and follow-up program there was a significant improvement on the knowledge of CAD risk factors and primary prevention measures (p<0.001). More importantly, the follow-up program led the participants to implement those positive changes into their lives and maintain a healthy life style.

Conclusions: Our study results showed that a structured training program of medical students could be utilized to implement an enhanced education and follow-up program for primary prevention of CAD in an economically challenged, underserved population with successful outcomes. This model program is not only beneficial for public interest but also enhances active interaction of medical students with patients at a very early stage of their career.

Heart failure

PP-090

Frailty predicts early adverse outcomes after left ventricular assist device implantation

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Background and Aim: Frailty has been associated with morbidity and mortality in patients with heart failure and those undergoing cardiac surgery. Thus, assessment of frailty may help identify the patients that would likely experience adverse outcomes. We aimed to study the relationship between frailty and the cardiovascular outcomes in left ventricular assist device population.

Methods: All patients scheduled to undergo LVAD implantation between 2017 and 2018 were preoperatively assessed for frailty, cognitive function (by means of Mini-Cog) and depression (with Patient Health Questionnaire). Fried's frailty phenotype was used to evaluate frailty (frail ≥3/5). Patients were observed for adverse events for 3 months.

Results: A total of 52 patients (44 men; aged 52±8 years) were included. 29 patients (55%) were designated as frail. Frail patients had higher NT-proBNP values, higher NYHA class, and depression while frailty was associated with lower body mass index and body surface area. Frailty increased the risk of in-hospital mortality or prolonged length of stay with 21 (72%) frail patients meeting the primary endpoint compared to 9 (39%) non-frail patients (p=0.01). Of the secondary outcomes, only postoperative ventricular arrhythmias reached statistical significance with being more common among frail patients than non-frail ones (p=0.01).

Conclusions: Frailty is common among patients with heart failure and is associated with increased in-hospital mortality or prolonged length of stay after LVAD implantation.

Table 1. Baseline characteristics and outcomes

	Total (n=52)	Frail (n=29)	Non-frail (n=23)	p value
Baseline characteristics:				
NYHA class [Mdn, (IQR)]	3 (0)	3,34 (1)	3 (0)	0.006
Cognitive function score	3,9 ±1,3	3,7 ±1,5	4,1 ±1,1	0.2
PHQ-9 score [Mdn, (IQR)]	8 (6)	9 (6)	6 (8)	0.04
BMI	26,1 ±4	24,1 ±3,3	28,6 ±3,3	0.001
BSA	1,9 ±0,1	1,8 ±0,2	1,9 ±0,1	0.02
NT-proBNP [Mdn, (IQR)]	5091 (6024)	5755 (6173)	3225(5304)	0.01
Outcomes:				
In-hospital mortality or prolonged length of stay	39	21 (72%)	9 (39%)	0.01
Infection	19	12 (41%)	7 (30%)	0.8
Ventricular arrhythmias	11	10 (8%)	1 (21%)	0.01

Heart failure

PP-091

Assessment of nutrition habits according to healthy eating index of patients with chronic heart failure

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Background and Aim: Nutritional deficiency is a critical factor for the development and prognosis of heart failure. An optimal diet should be ensured and maintained for managing heart failure symptoms. This study aimed to determine the nutritional status of patients with chronic heart failure by assessing their nutrition habits with the healthy eating index.

Methods: A total of 100 patients with heart failure (44 females and 56 males, with a mean age of 66±11.38 years) admitted to the cardiology clinics of a university hospital in Istanbul between March 2012 and August 2014 were included in this study. The data were collected using an information form, a 24-h individual food consumption record form, a food variety form, a Healthy Eating Index, and a Food Consumption Preference Form. **Results:** In the study, the mean age of 44 female and 56 male patients was 66±11.38. According to the sex, there was no significant difference between the amount of energy received by the patients, the amount of fiber, protein, carbohydrate, omega 3 and 6, sodium, potassium, calcium, iron and vitamin A consumed (p>0.05), there was a significant difference between the amounts of C vitamins consumed (p<0.05). In terms of food consumption preferences, 55% of the patients stated that they always consumed the unsaturated fats, one of the foods recommended to be eaten less, and 38% stated that they never consumed them. The Healthy Eating Index total mean score was found to be 74.6±9.32 in the study. This study found that the daily total energy intake of the participants was inadequate. The diet quality of most individuals was found to be included in the "needs improvement" diet category. The Healthy Eating Index total mean score of females (77.5±8.98) was found to be significantly higher than that of males (72.4±9.04) (p<0.05). **Conclusions:** This study found that most of the participants generally did not prefer the foods that were not recommended to be consumed. The daily energy intake and carbohydrate and omega-6 consumption were inadequate. The omega-3 consumption was at high levels, the sodium consumption was at high levels, and the potassium consumption was inadequate. The results of Healthy Eating Index showed that the diet quality of females was higher than that of males, and most of the participants had the diet quality in the "needs improvement" category.

Heart failure

PP-092

Evaluation of osilometrically measured arterial stiffness parameters in normal weight obesity subjects

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Background and Aim: Normal weight obesity is associated with higher cardiovascular mortality and metabolic dysregulation. We hypothesized that whether normal weight obesity has a relationship with arterial stiffness parameters.

Methods: Thirtyfive normal weight obesity and 33 normal weight healthy subjects were enrolled in the study. Normal weight obesity group was composed of subjects who had normal body mass index (18.5–24.9 kg/m²) and had excess body fat ratio defined by the highest sex-specific tertiles of body fat ratio (>23.1% in men and >33.3% in women). Arterial stiffness parameters including pulse wave velocity (PWV) and augmentation index (Alx) are osilometrically measured. Body fat measurement was done by bioelectric impedance analysis (BIA) method.

Results: Osilometrically-measured arterial stiffness parameters were higher in normal weight obesity subjects than normal weight healthy subjects (p<0.001 for PWV, p=0.004 for Alx) (Figure 1). PWV and Alx were not correlated with waist circumference and waist-to-hip ratio (p>0.05). For cardiovascular hemodynamic parameters, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure, cardiac output, stroke volume, santral SBP, santral DBP were not different between two groups (p>0.05).

Conclusions: Normal weight obesity subjects have higher arterial stiffness parameters. In addition, we determined that normal body mass index is not enough for cardiovascular risk evaluation and body fat ratio by BIA is better for risk stratification.

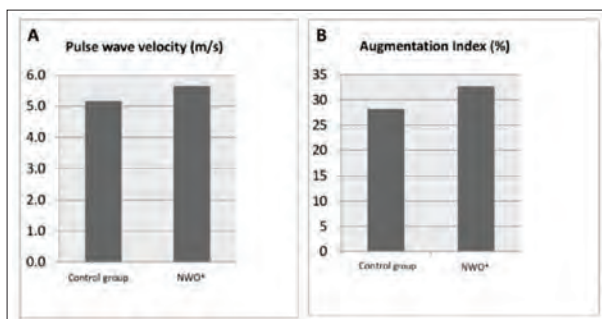


Figure 1. (a) Pulse wave velocity values in the study groups (p<0.001). (b) Augmentation index values in the study groups (p=0.004). NWO=normal weight obesity.

Lipid / Preventive cardiology

PP-093

The carotid intima media thickness in coal miners

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Background and Aim: The endothelial dysfunction causes many diseases which are associated with cardiovascular system by triggering atherosclerotic process. The atherosclerosis causes occurrence of a systemic disease by affecting large and medium arteries. Display of atherosclerotic changes in subclinical period may be important so as to reduce risk factors. Coal miners are exposed to high temperature, humidity, noise, vi-

bration and radiation. The chronic exposure to these physical conditions leads to arising some occupational diseases. In this study, it was investigated whether chronic exposure to these conditions has an effect on atherosclerotic process by assessing carotid intima media thickness.

Methods: The study was single centred and retrospective and consisting of total 100 patients between April 2016 and December 2017. There were 4 groups in the study. Twenty five of cases were coal miners (group 1), 25 of cases were coal miner retirements (group 2), 25 of cases were young controls (have never worked in coal mine and have never been exposed to coal dust (group 3) and 25 of cases were old control (have never worked in coal mine and have never been exposed to coal dust (group 4). The measuring of intima media thickness with ultrasonographic method is noninvasive, repeatable, easy, and costeffective. Carotid artery intima-media thickness (CIMT) are measured in longitudinal axis on one centimeter proximal posterior wall of bulbous in nonplaque areas. When the groups were classified regarding their ages, the age range of coal miners and healthy, young controls was determined 18-45 years old. The age range of healthy elderly control group and coal miner retirements group was determined 46-75 years old.

Results: The general characteristics of the participants were summarized in Table 1. When spirometry results were analysed; there were significant differences in point of forced vital capacity (FVC), forced expiratory volume in 1st second (FEV1), FEV1/FVC, peak expiratory flow (PEF), forced expiratory flow between 25% and 75% of vital capacity (FEF25-75) (respectively 0.000; 0.000; 0.014; 0.000; 0.000). The results were summarized in Table 2. When groups were compared in terms of hemogram parameters, there was a meaningful difference in mean cell hemoglobin concentration (MCHC) and mean cell volum (MCV) (respectively p values; 0.01 and 0.03; Table 3). There was no significant difference in point of EF, sPAB, TAPSE, left ventricle systolic and diastolic diameters. There was a difference among groups in point of CIMT, which was measured by doppler ultrasonography (p<0.000; Table 4, Graphic 1). In post-hoc analyse, there was no difference (p=0,994) between group 1 and 3; between group 2 and 4 there was a statistical difference (p=0.01).

Conclusions: In conclusion, it was found that increasing in carotid intima media thickness after chronic exposure to coal mine dust can make easy the early diagnosis of atherosclerosis. For this reason, profession questioning may be useful when risk factors for atherosclerosis are determined.

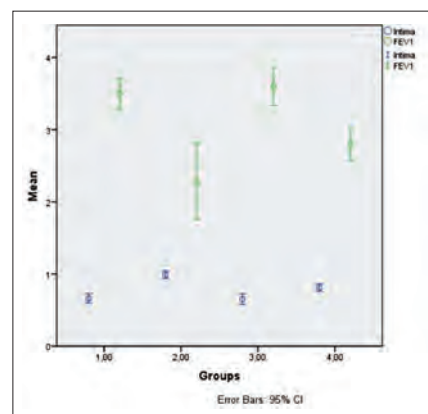


Figure 1. Mean values of intima media thickness and FEV1 in the groups.

Table 1. General characteristics of cases

	Coal miners (n=25) (Group 1)	Coal miners retirements (n=25) (Group 2)	Young control (n=25) (Group 3)	Elderly control (n=25) (Group 3)	p
Age(years)	39,76±5,33	60,43±7,16	36,36±3,24	57,2±8,62	0,000
Pulse (min)	85,84±13,74	79,8±14,81	80,72±78,75	80,20±16,92	0,531
Diastolic blood pressure (mmHg)	79,5±11,89	76,5±11,15	75,9±8,83	79,03±9,66	0,566
Systolic blood pressure(mmHg)	130,5±11,89	129,06±15,18	124,72±9,09	131,10±14,5	0,341
Working time(years)	11,03±5,92	19,62±3,87	-	-	-
Smoking status	12(%48)	10 (%40)	10 (%40)	12(48)	0,715**
Smoking(b/y)	11,38±10,07	18,2±17,85	15,26±7,16	27,24±17,1	0,68*
BMI	27,33±4,46	29,63±4,47	25,96±3,9	27,41±3,30	0,668

b/y= box/year; BMI= body mass index. p:One way ANOVA *Kruskal-Wallis test **Pearson chi square test

Table 2. Results of respiratory function test

	Group 1	Group 2	Group 3	Group 4	p
FVC (L)	4,12±0,67	2,95±1,03	4,31±0,74	3,31±0,79	0,000
FeV1(L)	3,49±0,5	2,28±0,97	3,59±0,59	2,80±0,61	0,000
FeV1/FVC	85,40±6,18	76,15±14,98	84,02±9,8	85,14±8,2	0,014
PEF (L/s)	7,47±1,7	5,74±2,28	6,14±2,08	5,89±1,89	0,000
FEF25-75 (L/s)	3,93±1,01	3±1,33	3,8±1,19	3,17±0,87	0,000

FEF25-75= the forced expiratory flow between 25% and 75% of vital capacity; FEV1= forced expiratory volume in 1st second; FVC= forced vital capacity; PEF= peak expiratory flow.

Table 3. The biochemical laboratory datas of cases

	Group 1	Group 2	Group 3	Group4	p
Total cholesterol (mg/dL)	195,07±34,52	193,62±21,86	181,18±34,69	199,93±40,42	0,293
LDL(mg/dL)	121,42±30,44	117,18±23,37	110,27±30,87	129,13±33,14	0,176
HDL(mg/dL)	41,42±9,86	47±12,14	44,72±7,21	44,58±8,57	0,287
Triglycerides (mg/dL)	170,30±118,3	139,5±80,26	131,09±42,26	130,86±45,92	0,228
Fasting blood glucose(mg/dL)	99,65±8,14	98,62±10,49	94,72±7,48	101,13±8,39	0,065
Hemoglobin(g/dL)	14,7±1,32	14,84±1,23	14,43±1,17	14,57±1,7	0,797
Hematocrit (%)	44,37±3,71	44,92±3,29	42,09±3,23	42,88±4,69	0,077
Platelet(109/L)	246,92±53,07	225,37±60,1	216,54±24,3	222,86±60,48	0,191
MCH (pg)	29,43±2,98	29,5±2,84	29,42±1,93	34,23±1,6	0,187
MCHC(g/dL)	32,91±1,6	32,81±1,55	34,33±1,29	33,7±1,97	0,01
MPV(fl)	9,68±0,93	9,96±1,52	9,45±1,03	9,19±1,47	0,191
MCV (fL)	87,1±5,42	89,47±7,25	85,69±3,04	85,52±4,71	0,03
N/L	2,71±2,98	2,29±1,9	2,37±0,84	1,99±0,58	0,384

HDL= high density lipoprotein; LDL= low density lipoprotein; MCH= mean cell hemoglobin; MCHC=mean cell hemoglobin consantration, MCV=mean cell volum; MPVs= mean platelet volum; N/L=neutrophil/lymphocyte ratio.

Table 4. The echocardiographic and carotid doppler ultrasonographic datas of cases

	Group 1	Group 2	Group 3	Group 4	p
EF(%)	63,07±3,18	59,68±4,26	63,63±2,27	61,55±4,45	0,059
sPAP(mmHg)	20,5±4,72	24,26±9,6	20,18±4,93	23,52±5,45	0,083
Pulmonary artery diameter(cm)	2,16±0,21	2,23±0,33	2,2±0,2	2,22±0,19	0,727
TAPSE (cm)	2,16±0,33	2,1±0,29	2,27±0,24	2,16±0,19	0,057
E/A	1,02±0,18	0,86±0,17	0,90±0,17	0,91±0,24	0,065
Intima media thickness(cm)	0,66±0,16	0,99±0,09	0,65±0,16	0,81±0,13	0,000

E/A= E wave/ A wave ratio; EF= ejection fraction; sPAP=systolic pulmonary artery pressure; TAPSE=tricuspid annular plane systolic excursion.

Heart failure

PP-094

Echocardiographic and laboratory predictors of heart failure in patients with preserved ejection fraction and left bundle branch block

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Background and Aim: The prevalence of left bundle brunch block (LBBB) is 0.16%-1.5% in general population. LBBB inversely affects the function of the left ventricle (LV), and LBBB is an independent risk factor for cardiac mortality. There limited data about the left ventricular and right ventricular (RV) functions in patient with LBBB and preserved left ventricular ejection fraction (LVEF). The purpose of this study was to investigate the effects of LBBB on systolic functions of left ventricle, functions and diameters of right ventricle in patients with preserved LVEF.

Methods: A total of 61 patients with LBBB and LVEF >55% who admitted to Adana Numune Training and Research Hospital Department of Cardiology between 2015-2017 were included to this study. Demographic and laboratory data of all patients were recorded. Twelve-lead electrocardiography (ECG) was recorded from all patients and ECGs were analysed for QRS duration. Echocardiographic examination was performed to all patients. Echocardiographic parameters of left ventricular systolic and diastolic functions, and right ventricular functions and diameters were recorded. Baseline and 6th month measurements of all these evaluations were recorded and compared.

Results: In the electrocardiographic, echocardiographic, and laboratory data comparison, the mean LVEF of patients was 61.0% and 58.8% (p=0.003), pro-BNP was 74 pg/ml and 90 pg/ml (p=0.028), right ventricle basal diameter was 3.2 mm and 3.32 mm (p=0.004), longitudinal diameter of right ventricle was 6.14 mm and 6.53 mm (p<0.001) and Tricuspid Annular Plane Systolic Excursion (TAPSE) was 22.6 mm and 22.9 mm (p=0.398) at the baseline and 6th month of the study, respectively. We determined that there was a negative correlation between LVEF and pro-BNP, QRS duration, LV lateral wall tissue Doppler e' velocity, and longitudinal diameter of right ventricle. Besides, pro-BNP was determined as an independent marker for the assessment of LVEF in the linear regression analysis (OR:-0.012, CI: [-0.019] - [-0.006], p<0.001).

Conclusions: LVEF decreased significantly after 6 months follow-up, and Pro-BNP was determined as an independent marker for the assessment of LVEF in patient with LBBB. In addition to that, basal and longitudinal diameters of right ventricle were significantly increased in the 6 months control.

Table 1. Demographic variables of patients

	Patients n=61
Age (years)	60.6 ± 10.8
Gender (female/male)	21/40
Systolic blood pressure (mmHg)	137.6 ± 22.1
Diastolic blood pressure (mmHg)	78.5 ± 13.5
Pulse rate (beats/minute)	76.9 ± 11.5
Weight (kg)	77.5± 13.3
Height (cm)	164,7 ± 7.4
Body mass index (kg/m2)	28.6 ± 4.4
Smoking, n (%)	7 (%11.5)
Diabetes mellitus, n (%)	17 (%27.9)
Hypertension, n (%)	42 (%68.9)
Hyperlipidaemia, n (%)	12 (%19.7)
Family history of coronary artery disease, n (%)	7 (%11.5)
Coronary artery disease, n (%)	10 (%16.4)

Table 2. Patients' current medications

	Patients n=61
ACE inhibitor or ARB (n, %)	41 (%67.2)
Nitrates (n, %)	2 (%3.3)
Beta blockers (n, %)	63 (%54.1)
Furosemide (n, %)	22 (%36.1)
Aldosterone (n, %)	4 (%6.5)
Ivabradine (n, %)	1 (%1.6)
Ranolazine (n, %)	1 (%1.6)
Statins (n, %)	15 (%24.6)
Ca channel blocker (n, %)	18 (%29.5)
Acetyl salicylic acid (n, %)	25 (%40.1)
Metformin (n, %)	16 (%26.2)

Table 3. Comparison of baseline and 6th month's laboratory, ECG, and echocardiography data of patients

Laboratory, ECG and echocardiography data	Baseline	6 months	p
Pro-BNP (pg/ml)	74 (96.4)	90 (125.5)	0.028
Hs-Troponin I (pg/ml)	10.7±1.8	8.3±1.4	0.610
Hs-CRP(mg/dl)	0.4(0.65)	0.35(0.3)	0.845
GFR (ml/min)	93.3 ± 18.6	93.2 ± 20.2	0.955
Creatinine (mg/dl)	0.73 ± 0.16	0.74 ± 0.15	0.393
QRS (ms)	135.5 ± 13.1	135.7 ± 14.7	0.843
LVEF (%)	61.0 ± 6.2	58.8 ± 6.4	0.003
LA diameter (mm)	37.0 ± 4.3	37.7 ± 3.5	0.298
Mitral valve PW Doppler	Baseline	6 months	p
E velocity (cm/s)	71.8 ± 18.4	73.1 ± 19.1	0.419
A velocity (cm/s)	94.6 ± 24.2	97.4 ± 19.9	0.213
E/A ratio	0.81 ± 0.30	0.79 ± 0.29	0.25
LV lateral wall tissue Doppler	Baseline	6 months	p
e' velocity (cm/s)	7.8 ± 3.5	8.2 ± 3.7	0.351
a' velocity (cm/s)	12.9 ± 3.4	13.7 ± 3.6	0.060
E/e'	10.3 ± 3.6	9.9 ± 3.3	0.414
E'/a'	0.67 ± 0.45	0.68 ± 0.47	0.825
Right ventricle measurements	Baseline	6 months	p
Basal diameter (mm)	3.20 ± 0.35	3.32 ± 0.32	0.004
Longitudinal diameter (mm)	6.14 ± 0.62	6.54 ± 0.59	<0.001
TAPSE (mm)	22.6 ± 2.6	22.9 ± 2.3	0.398
PABs (mmhg)	31.1 ± 6.9	32.0 ± 7.6	0.353

Table 4. Correlation analysis between ejection fraction and 6th month's data

	r	p
Pro-BNP (pg/ml)	-0.387	0.002
QRS (ms)	-0.321	0.012
e' velocity (cm/s)	-0.273	0.033
Right ventricle longitudinal diameter (mm)	-0.295	0.021

Heart failure

PP-095

The rates of use of drugs that reduce mortality in patients with reduced ejection fraction in turkish population

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Background and Aim: Heart failure(HF) is associated with significant morbidity and mortality despite the use of medical therapies such as angiotensin-converting enzyme inhibitors, beta-adrenergic blockers, angiotensin receptor blockers, and mineralocorticoid receptor antagonists. Our purpose is to determine the number of patients receiving optimal medical treatment at first admission to heart failure polyclinic to our hospital.

Methods: We collected demographic, laboratory, drug history, physical examination and echocardiographic parameters data at first admission to our heart failure polyclinic (Table 1 and 2). We assessed 187 patients with reduced ejection fraction.

Results: In our trial, 51% of the patients were using ACE or ARB, 70% of the patients were using Beta-blocker, and 40% of the patients were using mineralocorticoid receptor antagonists (Table 3). The average ejection fraction was 29% in our trial. 92% of the patients were NYHA class II and above (Table 4).

Conclusions: In our country, heart failure patients with reduced ejection fraction don't receive enough treatment. There are many reasons for this situation. In Turkey, general cardiologists treat about 80-100 patients in a day. However, many centers have not heart failure polyclinic. For this reason, cardiologists can not leave enough time for chronic illness like heart failure. Heart failure polyclinics in our country should become widespread, and these patients should be directed to heart failure polyclinics.

Table 1. Usage rate of drugs in our trial

Drugs	Frequency(N:187)	Percent	Cumulative Percent
ACE/ARB	97	51	51
Spirolactone	75	40.1	40.1
Furosemide/Tiazide	97	51	51
Beta Bloker	134	71.4	71.4
Ivabradine	13	7	7
Acetylsalicylic Acid	108	57.8	57.8
Digoxin	32	17.1	17.1
Statin	29	15.5	15.5
ARNI	2	1.1	1.1

Heart failure

PP-096

The effect of spironolactone on atrial conduction in patients with heart failure with reduced ejection fraction

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Background and Aim: Atrial fibrillation (AF) is a common arrhythmia in patients with chronic heart failure (HF), and it is associated with worse prognosis. Although there is some evidence that mineralocorticoid receptor antagonists may reduce the risk of developing AF in such patients, the evidence in this regard is insufficient. Measurement of atrial conduction time on electrocardiography (ECG) is a commonly used method to evaluate the risk of developing AF. This study aims to evaluate the effect of spironolactone on atrial conduction in ECG in patients with HF with reduced ejection fraction (EF).

Methods: Twenty-five patients with compensated HF with reduced EF (<40%) and sinus rhythm who did not receive spironolactone therapy were included. Patients who had AF, decompensated HF findings, prosthetic heart valve or severe valve disease, HF in the course of acute coronary syndrome were excluded. One patient in the spironolactone group and one patient in the control group died due to cardiovascular causes and they could not complete the six-month follow-up period. Thus, 23 patients (spironolactone group: 12 patients, control group: 11 patients) were included and they were followed-up for six months. The basic clinical and laboratory characteristics of the patients were detected. At baseline and at the end of six-month follow-up, ECG and echocardiographic characteristics were compared within and between the groups.

Results: The groups were similar in terms of baseline clinical and laboratory features (Table 1). In the control group, electrocardiographic and echocardiographic measurements were similar at baseline and at the end of the six-month follow-up period (Table 2). After six-month follow-up, P maximum and P dispersion values significantly decreased compared to baseline in the spironolactone group (p=0.001 and <0.001, respectively) (Table 3). According to the two-way repeated measurement results, P maximum and P dispersion values significantly decreased in the spironolactone group compared to the control group at the end of the six-month follow-up period (p=0.011 and 0.002, respectively) (Figure 1 and 2).

Conclusions: In patients with compensated HF with reduced EF and sinus rhythm, spironolactone provides significant improvements in atrial conduction time, which presents an increased risk for AF.

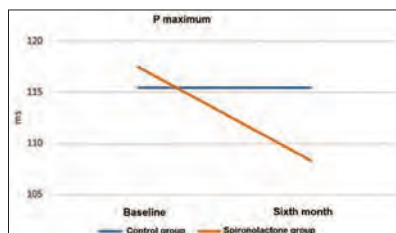


Figure 1. Changes in time at the P maximum value in the control and spironolactone groups (p=0.011).

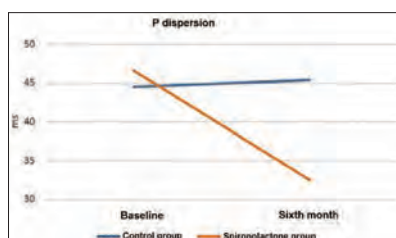


Figure 2. Changes in time at the P dispersion value in the control and spironolactone groups (p=0.002).

Table 1. Baseline clinic and laboratory features of the groups

	Control group (n=11)	Spirolactone group (n=12)	P value
Age (years)	61.00 (53.00 - 67.00)	60.00 (50.25 - 65.00)	0.783
Male sex (n,%)	9 (81.8)	8 (66.7)	0.640
Hypertension (n,%)	5 (%45.5)	7 (%58.3)	0.684
Diabetes mellitus (n,%)	3 (%27.3)	3 (%25.0)	1.000
Prior MI (n,%)	6 (%54.5)	4 (%33.3)	0.414
eGFR (mL/min/1.73 m2)	75.64 ± 41.47	95.75 ± 35.03	0.211
proBNP (pg/ml)	586.00 (407.00 - 979.00)	494.50 (196.75 - 763.50)	0.449
Heart rate (beats/min)	69.36 ± 13.38	75.33 ± 8.67	0.214
P maximum (ms)	115.45 ± 14.40	117.50 ± 15.45	0.746
P minimum (ms)	70.91 ± 17.00	70.83 ± 15.05	0.976
P dispersion (ms)	44.55 ± 9.34	46.67 ± 9.85	0.608
LV EF (%)	29.82 ± 6.01	29.83 ± 4.88	0.995
LV end-diastolic diameter (mm)	53.27 ± 5.04	56.75 ± 5.28	0.091
LV end-systolic diameter (mm)	38.27 ± 5.92	43.75 ± 5.55	0.032
LA diameter (mm)	38.00 (37.00 - 45.00)	40.00 (35.50 - 44.75)	0.695
Mitral E/A ratio	1.03 (0.58 - 2.28)	0.86 (0.74 - 1.27)	0.786

BNP, brain natriuretic peptide; eGFR, estimated glomerular filtration rate; LA, left atrium; LV, left ventricle; LV EF, left ventricular ejection fraction; MI, myocardial infarction.

Table 2. Comparison of electrocardiographic and echocardiographic features of baseline and sixth-month in the control group

	Baseline	Six-month	P value
P maximum (ms)	115.45 ± 14.40	115.45 ± 15.72	1.000
P minimum (ms)	70.91 ± 17.00	70.00 ± 18.97	0.786
P dispersion (ms)	44.55 ± 9.34	45.45 ± 9.34	0.770
LV EF (%)	29.82 ± 6.01	31.00 ± 5.97	0.520
LV end-diastolic diameter (mm)	53.27 ± 5.04	52.36 ± 5.33	0.562
LV end-systolic diameter (mm)	38.27 ± 5.92	40.45 ± 8.68	0.317
Mitral E/A ratio	1.25 ± 0.91	1.22 ± 0.84	0.881

LV, left ventricle; LV EF, left ventricular ejection fraction.

Table 3. Comparison of electrocardiographic and echocardiographic features of baseline and sixth-month in the spironolactone group

	Baseline	Six-month	P value
P maximum (ms)	117.50 ± 15.45	108.33 ± 18.01	0.001
P minimum (ms)	70.83 ± 15.05	73.33 ± 11.55	0.438
P dispersion (ms)	46.67 ± 9.85	32.50 ± 11.38	<0.001
LV EF (%)	29.83 ± 4.88	31.75 ± 5.36	0.281
LV end-diastolic diameter (mm)	56.75 ± 5.28	55.50 ± 4.06	0.408
LV end-systolic diameter (mm)	43.75 ± 5.55	45.25 ± 4.58	0.470
Mitral E/A ratio	1.12 ± 0.65	1.15 ± 0.61	0.850

LV, left ventricle; LV EF, left ventricular ejection fraction.

Lipid / Preventive cardiology

PP-097

Relationship between QT dispersion and biochemical parameters in Behçet's disease

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Background and Aim: Behçet's disease (BD) is a chronic recurring multisystemic vasculitic disease involving recurrent skin and ocular manifestations and effecting cardiovascular, neurologic, and intestinal systems. BD may cause sudden death due to aortic dissection, pulmonary embolism, cardiomyopathy and coronary artery disease secondary to BD vasculitis. The QT distance is defined as the time at the beginning of the QRS complex to the end of the T wave. Complex ventricular arrhythmia frequency is correlated with QTcd duration in patients with BD. In this study, we aimed to investigate the effects of biochemical parameters which may be related to BD on QT and QTc dispersion.

Methods: Thirty-five patients with BD and 47 age and sex-matched healthy individuals who applied dermatology and cardiology outpatient clinic between January 2014 to December 2015 were included. The QT

interval was manually measured using the tangent method from the beginning of the QRS complex to the end of the T wave from all 12 leads. The QTc interval was calculated in lead V5 using Bazett's formula (QTc = QT interval / \sqrt{RR} interval). QTdc was manually calculated by the difference between the longest and the shortest QTc intervals measured across the 12 leads. The fasting blood tests, hemogram parameters and echocardiographic data of participants were recorded. The study was approved by local ethics committee. **Results:** Demographics, clinical characteristics, echocardiographic and laboratory results of the participants were shown in Table 1 and Table 2. The values of the longest QT, the shortest QT, QT dispersion (QTd), corrected QTd in BD group and control group are 400.71 ms vs 374.67 ms, 360 ms vs 338.67 ms, 39.29 ms vs 36 ms and 46.21 ms vs 41.53 ms, respectively. The longest QT and the longest QTc values were significantly increased in BD group (p=0.004); however, the shortest QT value was longer in BD group even not reached statistically significance (p=0.054&0.018). The results were summed in Table 3. Albumin, mean platelet volume (MPV), sedimentation, the longest QT and QTc were compared with Spearman correlation. There was negative correlation between albumin and sedimentation with the longest QT; meanwhile, there was positive correlation between MPV and the longest QTc (Table 4). **Conclusions:** The longest QT-QTc, the shortest QT-QTc, QTd, QTcd were longer in BD group; but only the longest QT-QTc difference was significant. Cardiac arrhythmias may be seen more often in this patient group patients.

Table 1. General characteristics of participants

	Behçet's disease (n=35)	Controls (n=47)	p
Age (years)	36,23±10,1	39,02±3,8	0,354
Sex	F:20 M:15	F:22 M: 25	0,128
Smoking	12	17	0,251
Duration of disease (years)	9,15±5,18	-	-

F: female; M: male

Table 2. The biochemical parameters of participants

	Behçet's Disease Group	Control Group	p
Albumin (g/dL)	3,9±0,7	4,5±0,3	0,000
Total cholestrol(mg/dL)	189,3±25,1	197,2±36,1	0,249
LDL (mg/dL)	113,3±22,5	115,1±33,5	0,770
HDL (mg/dL)	53,3±14,1	50,9±15,4	0,460
Triglycerid (mg/dL)	130,1±36,8	157,7±86,7	0,054
MPV (fL)	8,2±0,5	9,9±1	0,000
CRP (mg/dL)	0,8±1,3	0,4±0,2	0,071
ESR (mm/h)	15,3±9,5	10±3,2	0,002
Fasting blood glucose (mg/dL)	94,94±7,12	92,23±7,35	0,099
İnsulin (uIU/mL)	8,52±3,32	10,65±8,91	0,182
HOMA-IR	1,29±0,5	1,32±0,5	0,749
EF(%)	60,3±3	60,72±5,07	0,526
LVDD(cm)	4,38±0,64	4,57±0,39	0,207
LVSD(cm)	3,39±0,0,46	3,57±0,37	0,961
LA(cm)	3,42±0,33	3,51±0,27	0,536

LDL: Low density lipoprotein, HDL: High density lipoprotein, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, MPV: Mean platelet volume, HOMA-IR: Homeostatic Model Assessment - InsulinResistance, EF: Ejection fraction, LVDD: Left ventricle end diastolic diameter, LVSD: Left ventricle end systolic diameter, LA: Left atrium

Table 3. QT values of groups

	Behçet's Disease Group	Control Group	p
Longest QT	400,71±22,34	374,67±21,996	0,004
Shortest QT	360±28,28	338,67±28,75	0,054
QTd	39,29±12,68	36±17,23	0,56
Longest QTc	445,57±20,59	428,07±16,68	0,018
Shortest QTc	399,07±20,35	386,53±19,27	0,1
QTcd	46,21±18,78	41,53±20,42	0,52

QTd: QT dispersion, QTc: Corrected QT, QTcd: Corrected QT dispersion

Table 4. Spearman Correlation Analysis

	Longest QT	Longest QTc	Sedimentation	MPV	Albumin
Longest QT	1	0,328	-0,376	0,399	-0,102
Longest QTc	0,328	1	-0,159	0,536	-0,023

QTc: Corrected QT, MPV: Mean platelet volum

Lipid / Preventive cardiology

PP-098

Changes in serum uric acid and gamma glutamyl transferase levels in morbid obese subjects after laparoscopic sleeve gastrectomy: A prospective study

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Background and Aim: Long-term results of pharmacological and non-pharmacological approaches against morbid obesity which is one of the important public health challenges, are not generally satisfactory. Bariatric surgery is an option when all these approaches fail to provide desired results. Laparoscopic sleeve gastrectomy (LSG) is one of the novel bariatric surgery procedure. Technical simplicity, favorable outcomes and low complication rates make this procedure widely preferred. Close association between cardiovascular disease (CVD) and elevation of GGT and uric acid levels have been previously suggested. We aimed to investigate the influence of significant weight loss following LSG on uric acid and gamma glutamyl transferase (GGT) levels in a prospective study design.

Methods: The medical data of 102 morbid obese subjects (29 male, 73 female) who underwent LSG between February 2015 and May 2017 were prospectively recorded. Patients were recruited for standard bariatric surgical indications according to obesity diagnosis and treatment guidelines; body mass index (BMI) >40 kg/m², BMI >35 kg/m² and additional co-morbidities associated with obesity such as type 2 Diabetes Mellitus. Delta (Δ) values were obtained by subtracting one-year values from the baseline values.

Results: Mean age of our study group was 41.71±11.92 years. Mean weight loss of our study population was 46.01±13.65 kg (36.8%) in one-year after the surgery. Mean BMI was decreased from 46.71±7.21 kg/m² to 29.53±4.63 kg/m² after LSG. Compared to the preoperative period, there was a significant decrease in both serum uric acid (5.53±1.04 mg/dl vs 4.64±0.94 mg/dl; p<0.001) and GGT (29.89±12.15 U/L vs 20.75±8.21 U/L, p<0.001) levels in the postoperative one-year follow up. It was shown that Δ VKI is significantly correlated with Δ uric acid (r=0.331, p=0.01) and Δ GGT (r=0.378, p<0.001).

Conclusions: Serum uric acid and GGT levels which are associated with CVD are decreased in morbid obese subjects who have lost sustained weight following LSG in a one-year follow up.

Cardiovascular surgery

PP-099

Can we control supraventricular tachycardia following congenital heart surgery

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Background and Aim: Dexmedetomidine can potentially have an adverse effect on the cardiovascular system secondary to its negative effects on chronotropic and dromotropic effects. However, it has been shown that these cardiac effects are currently being explored as a therapeutic option for the treatment of perioperative tachyarrhythmias in pediatric patients with congenital heart disease (CHD). Here, we have reported our experiences with dexmedetomidine in the treatment of junctional ectopic tachycardia (JET), which was developed following congenital heart surgery.

Methods: Our study included five patients admitted to the cardiovascular intensive care unit (CICU) after congenital heart surgery who took dexmedetomidine for both sedation/analgesia and for junctional ectopic tachycardia (JET), atrial ectopic tachycardia (AET), and reentry type supraventricular tachycardia (Re-SVT). Dexmedetomidine was used as a primary drug. Our primary end-point was occurrence of bradycardia, hypotension. In all patients arrhythmias occurred at the end of the operation or in the early postoperative period. Postoperatively, dexmedetomidine was administered at a dose of 0.05 to 0.5 µg/kg/hr infusion and continued 24 hours. We didn't use loading dose in order to prevent sudden bradycardia occurrence. Full monitoring made in the operation room was continued in CICU (pulse oximetry, ECG, invasive radial arterial blood pressure, cerebral Near infrared spectroscopy (NIRS), body temperature). Aortic cross clamp (ACC) time, total cardiopulmonary bypass (CPB) duration, mechanical ventilation duration and ICU stay was also recorded.

Results: There were five patients, 3 male and 2 female. The demographics and cardiac diagnosis were similar in the patients (Table 1). The median respiratory rate, median oxygen saturation, peripheral arterial blood gas values were similar and was within the normal ranges in all patients. There was two cases with tetralogy of Fallot (TOF), 2 cases with ventricular septal defect (VSD), 1 case with complete atrioventricular septal defect (CAVSD). We have started dexmedetomidine infusion in the CICU for these patients both for sedation/analgesia and for the treatment of tachyarrhythmias. The incidence of junctional ectopic tachycardia was significantly reduced in the patients receiving dexmedetomidine (Table 2). The mechanical ventilation duration, ICU stay, were also similar in patients (Table 2).

Conclusions: Dexmedetomidine has been reported to depress sinus node and atrioventricular nodal function in pediatric patients. This preliminary, observational report suggests that dexmedetomidine may have a potential therapeutic role in the acute phase of perioperative atrial and junctional tachyarrhythmias, which is one of the most serious and life threatening postoperative arrhythmias that is difficult to manage, for HR control without significant side effects. Nonetheless, larger prospective studies are needed to confirm these results.

Table 1. Demographic characteristics of the patients

	Age (month)	Weight (kg)	Cardiac diagnosis/Operation	Arrhythmia	MV duration/ICU stay
Patient I	8	6,7	VSD/VSD closure	Re-SVT	4/24
Patient II	5,5	5	TOF/Total correction	Re-SVT	6/28
Patient III	9	9,4	CAVSD/Total repair	JET	8/31
Patient IV	4,8	5,8	TOF/Total correction	Re-SVT	9/24
Patient V	6,5	6,1	VSD/VSD closure	JET	5/24

VSD:Ventricular Septal Defect; TOF:Tetralogy of Fallot; CAVSD:Complet Atrioventricular Septal Defect

Table 2. Heart rates of the patients during Dexmedetomidine infusion

	Initial	30th minutes	2nd hour	6th hour	12th hour	24th hour
Patient I	186	174	165	159	136	128
Patient II	184	178	162	145	130	125
Patient III	198	186	178	154	141	135
Patient IV	178	157	149	142	138	119
Patient V	204	187	161	131	124	119

Cardiovascular surgery

PP-100

The relationship between whole blood viscosity and deep vein thrombosis

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Lipid / Preventive cardiology

PP-101

Use of secondary preventive pharmacotherapy among middle eastern patients who survived a decade or more after coronary revascularization

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had PCI vs. CABG, men vs. women, or diabetics vs. non diabetics. Furthermore, no difference were observed in the use of these medications among those who survived 10 years vs. those who survived >20 years.

Conclusions: In this interim analysis, Middle East patients surviving at least a decade after coronary revascularization have a high rate of utilization of secondary cardiovascular medications. Full details will be presented at the conference.

Heart failure

PP-102

Disease modifying drugs in heart failure patients: A single center study

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Background and Aim: Heart failure is a growing health problem that is affecting about 1-2% of general population. The management of heart failure is complex and requires special, regular follow-up of the patient. There is also an unmet need for heart failure clinics in Turkey. We aimed to investigate utilization of disease modifying drugs for heart failure in outpatient clinics in Mugla.**Methods:** We planned an observational, prospective, single center study to evaluate prescription patterns of heart failure drugs in our hospital. Baseline characteristics were noted and medications for heart failure were inquired. If a patient was not on a disease modifying drug (ACE, ARB, BB, MRA, ivabradine, ARNI) the reason for not using the drug was noted.**Results:** A total of 52 (21% female) patients were enrolled to the study. The mean age was 67.31±10.88. Of the 52 patients 33 had NYHA class 2 symptoms and the most common symptoms were reduced exercise tolerance and fatigue, 57% and 64% respectively. The mean systolic and diastolic pressures were 125±23 and 75±10 respectively and the mean heart rate was 75±16. Of the study population 32 were not using an ACE inhibitor and 22 were not using an ACE inhibitor or ARB. Beta blockers were the most utilized drugs (43 on BB) whereas only one patient was on ARNI. Mineralocorticoid receptor antagonists and ivabradine were used by 19 and 13 patients respectively. The reasons for not being on a guideline recommended heart failure drug was summarized on Figure 1. The mean dosage of the drugs proportional to guideline recommended dosages were 22±33%, 11±27%, 33±26%, 16±23%, 16±29% for ACE, ARB, BB, MRA and ivabradine.**Conclusions:** Our study showed disease modifying drugs were underutilized in heart failure patients. Although there were plenty of evidence and clear guideline recommendations every two out of five patient was not on ACE or ARB and every one out of five patient was not on BB. This situation clearly highlights a regular follow-up of patients and education of physicians is the utmost important issue in the management of heart failure. There is also a need for special follow-up scheme to avoid neglecting of the heart failure patient by physician.**Figure 1.** Reasons for not being on disease modifying drugs.

Heart failure

PP-103

Sacubitril/valsartan treatment increases the left ventricular ejection fraction in patients with systolic heart failure: A bayesian analysis

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Background and Aim: Systolic heart failure is an important leading cause of mortality and morbidity despite the optimal medical treatment and device therapy. Sacubitril/ Valsartan is a first generation combination therapy, has recently been approved for treatment of systolic heart failure. No prior literature is available on the association between Sacubitril/valsartan therapy and the change in left ventricular (LV) contractile function.**Methods:** 52 heart failure patients with reduced ejection fraction (HFrEF) were enrolled in this study. The baseline demographic, clinical and echocardiographic characteristics of these patients were compared using Bayesian analysis method.**Results:** 52 heart failure patients with reduced ejection fraction (HFrEF) were included in final analysis (66.2±9.3 years, 69.2% male). Low initial dose was administered to 44.2% of patients and an intermediate initial dose was administered to 55.8% of them. There was an absolute increase in LVEF as 3.87 (95% HDI 1.53-6.20) and 5.89 (95% HDI 4.18-7.61) in low and intermediate initial dose populations, respectively (the

bayes factor was 0.681 and posterior probability was 92). The absolute increase in LVEF was 5.56 (95% HDI 3.49–7.63) in female population while 4.75 (95% HDI 2.91–6.58) in male population (the bayes factor was 0.334 and posterior probability was 72%).

Conclusions: We demonstrated that the use of Sacubitril/Valsartan is associated with increased left ventricular contractile function, regardless of sex and age, in patients with HFrEF. We also highlighted that the possible mechanism of this treatment might be cardiac remodeling.

Cardiovascular nursing / Technician

PP-104

Cancer patients in coronary care unit: A single, tertiary center experience

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Background and Aim: Although, advances in oncology prolonged life expectancy, cardiac side effects of anti-cancer drugs resulted in a new patient profile. We aimed to evaluate all cancer patients who applied to emergency departments with cardiac symptoms during coronary care unit (CCU) admission.

Methods: From May 2016 to May 2018, 120 patients (35 females) between 36 to 97 years of age, with anti-cancer treatment prior to admission who applied to our emergency department and/or referred from remote hospitals for invasive diagnosis and treatment were included. All patients with anti-cancer treatments were retrospectively evaluated for their baseline characteristics, laboratory values and clinical situations during their emergency department and CCU stay. No exclusion criteria were applied.

Results: As shown in table 1, diagnosis of lung cancer in 47 pts (39.1%), STEMI in 44 pts (36.6%), and atrial fibrillation with rapid ventricular response (AFVR) in 42 pts (35%) were noted. In hospital mortality was 15% (19 pts). The most frequently observed electrolyte disturbances were hyponatremia (51 pts, 42.5%) and hypomagnesemia (32 pts, 26.6%). According to their medical records, no patients with atrial fibrillation were on regular anticoagulation or anti-platelets prior to index hospital admission. Mean CCU stay was 6 days, in hospital mortality was 15.8% (19 pts). All pts had urinary catheters, 14 pts (11.6%) had colostomy bag, 17 pts (14.1%) required mechanically assisted ventilation, and 9 pts (7.5%) experienced decubitus ulcers.

Conclusions: A selection bias may have attributed to referral system is the main and the most important limitation of this study. According to current study results, majority of the patients were not on adequate medication for heart failure and atrial fibrillation. An interesting point is, in normal conditions, due to increased life expectancy with modern oncologic therapies, cardiac side effects of cancer therapy, common risk factors of cancer and heart diseases, advanced mean age of study patients, no application of any exclusion criteria, and two years of evaluation period; there should have been some patients with diagnosed hematologic, thyroid, cranial, male and female reproductive system, head-neck, pancreas, renal and skin malignancies. Absence of these reminds lack of adequate awareness, knowledge and experience for patient selection, modern treatment possibilities, referral to specialized centers and life expectancy in our geographic region.

Table 1. General Characteristics	Female	Male	Total
Gender	35 (29.1%)	85 (70.9%)	120 (100%)
Age	70.9 (49-91)	60.1 (36-97)	68.4 (36-97)
Hospital Readmission	2 (5.7%)	9 (10.6%)	11 (9.1%)
Cancer Type			
Breast Cancer	18 (51.4%)	1 (1.1%)	19 (15.8%)
Lung Cancer	8 (22.9%)	39 (45.9%)	47 (39.1%)
Colon Cancer	3 (8.5%)	11 (12.9%)	14 (11.6%)
Gastric Cancer	5 (14.3%)	10 (11.7%)	15 (12.5%)
Hepatocellular Cancer	1 (2.8%)	3 (3.5%)	4 (3.3%)
Prostate Cancer	NA	21 (24.7%)	20 (16.6%)
Diagnosis			
USAP	3 (8.5%)	9 (10.6%)	12 (10%)
STEMI	12 (34.3%)	32 (37.6%)	44 (36.6%)
NSTEMI	8 (22.9%)	21 (24.7%)	29 (24.1%)
Heart Failure	9 (25.7)	16 (18.9%)	25 (20.9%)
Atrial Fibrillation	12 (34.3%)	30 (35.3%)	42 (35%)
Pericardial Effusion	2 (5.7%)	5 (5.9%)	7 (5.8%)
VT, VF	9 (25.7)	15 (17.6%)	24 (20%)
Complete AV Block	3 (8.5%)	0 (0%)	3 (2.5%)
Electrolyte Disturbances			
Hypomagnesemia	7 (20%)	25 (29.4%)	32 (26.6%)
Hypokalemia	5 (14.3%)	2 (2.3%)	7 (5.8%)
Hyponatremia	15 (42.9%)	36 (42.3%)	51 (42.5%)
EPS			
Temporary PM	3 (8.5%)	2 (2.3%)	5 (4.1%)
PM Implantation	3 (8.5%)	0 (0%)	3 (2.5%)
AV Node Ablation	1 (2.8%)	0 (0%)	1 (0.8%)
Applied Procedures			
Urinary Catheters	35 (100%)	85 (100%)	120 (100%)
Dialysis (UF, HF, PD)	2 (5.7%)	3 (3.5%)	5 (4.1%)
Colostomy	3 (8.5%)	11 (12.9%)	14 (11.6%)
Endotracheal Intubation	8 (22.9%)	19 (22.3%)	27 (22.5%)
Decubitus Ulcers	6 (17.1%)	3 (3.5%)	9 (7.5%)
Mechanical Ventilation	9 (25.7)	8 (9.4%)	17 (14.1%)
Delirium	2 (5.7%)	3 (3.5%)	5 (4.1%)
DC Cardioversion	2 (5.7%)	3 (3.5%)	5 (4.1%)
In Hospital Mortality	5 (14.3%)	14 (16.4%)	19 (15.8%)

Heart failure

PP-105

Acute decompensated ischemic heart failure and GRACE risk score after non-ST elevation myocardial infarction

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Background and Aim: Myocardial infarction without ST-elevation is one of the emergencies that causes acute coronary syndrome. Non-STEMI is a cardiac pathology with vital significance due its high morbidity and mortality rates. The factors such as age, Killip class, heart rate, blood pressure irregularity, symptom-balloon time, size of myocardial necrosis, diabetes mellitus, hypertension, smoking, anemia and renal failure increase the complication rates in the patients with ACS. GRACE score is a scoring system which was started to be used for prognostic evaluation in the patients with ACS after the GRACE study in 2003. Its calculation is recommended in non-STEMI patients according to AHA and ESC guidelines. In this study, we aimed to evaluate the significance of GRACE score in predicting the development of acute decompensated ischemic heart failure after myocardial infarction without ST elevation.

Methods: The patients who were admitted with the diagnosis of non-STEMI between July 2016 and April 2017 were evaluated within the scope of this study. Of these patients, 182 patients (125 male, the mean age: 67.1±11.7) who had left ventricular ejection fraction (LV-EF) <50% and N-terminal proB-type natriuretic peptide (NT-proBNP) level >125 pg/mL were included in the study. During hospitalization period, the patients with Killip class (KC) ≥2 were included in the D-HF (n=50) and others were included in the C-HF groups (n=132). PCI procedure was performed on all patients within the first 24 hours. PCI was applied to the culprit and/or the other lesions on the same vessel when needed. Elective PCI was recommended for the lesions inside the coronary artery where the culprit lesion was located.

Results: The morbidity and mortality rates in the D-HF group were higher. Other than GS, LV-EF, age, creatinine, hemoglobin, NT-proBNP levels at admission and diabetes mellitus were determined as the predictors of D-HF. According to the ROC analysis, the patients with GS >177.5 were found to be at higher risk for D-HF development.

Conclusions: The development of D-HF after non-STEMI significantly increases the morbidity and mortality rates. Complication rates can be reduced by reducing the development of D-HF. For this purpose, high GS values in non-STEMI patients can be used as a predictor of D-HF development, together with the other known risk factors.

Interventional cardiology / Carotid and peripheral vascular

PP-106

Investigate on effectiveness of a successful revascularism in peripheral artery disease on aortic augmentation index

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Background and Aim: The aortic augmentation index (AIX), a marker of arterial stiffness, and peripheral arterial disease (PAD) are associated with an increased cardiovascular risk. Previous studies have shown that AIX@75 elevated in peripheral arterial diseases (PAH). In our study, We investigated the impact of successful percutaneous revascularization on AIX@75.

Methods: Fifty patients admitted to the cardiology department with a diagnosis of peripheral artery disease and recruited for revascularization were included in the study. Twenty patients were excluded from study for various reasons. Six patients were amputated during the first month of treatment. Atrial fibrillation developed in 2 patients. Kidney failure requiring dialysis developed in 1 patient after the procedure. 3 patients were re-revascularized. 1 patient died and oxygen therapy was applied to 7 patients. Our study was completed with 30 patients (25 males, 5 females). AIX@75 values of 30 patients were evaluated on the first day and first month of revascularization.

Results: According to AIX @ 75 baseline values (30.1±11.0), the change in the AIX@75 values after successful revascularization (24.8±10.3) on the first day was significant (t=2.46; p=0.022). Moreover, according to AIX@75 baseline values (30.1±11.0), the change in the AIX@75 values that was evaluated after a month from successful revascularization (27.1±9.2) was also significant (t=2.17; p=0.039).

Conclusions: Our study showed that successful revascularization of peripheral artery disease was significantly reduced the aortic augmentation index.

Interventional cardiology / Coronary

PP-107

The value of pre-percutaneous coronary intervention mean platelet volume to lymphocyte ratio in predicting in-stent restenosis

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Background and Aim: Although pharmacological therapies and interventional approaches have developed in the recent years, in-stent restenosis still continues as a problem. In the current study, our primary aim is to investigate the impact of pre-procedural mean platelet volume (MPV) to lymphocyte ratio (MLR) on risk of in-stent restenosis for the first time in literature.

Methods: Totally, 1411 patients were enrolled in this study (n=1045 patients in negative-in-stent restenosis group and n=365 patients in positive in-stent restenosis group).

Results: In multivariate Cox regression analyzes showed that the rate of diabetes mellitus, white blood cell, neutrophil to lymphocyte ratio (NLR), platelet to lymphocyte ratio (PLR), MPV to platelet ratio (MPR) and

MLR were significantly higher in positive in-stent restenosis group. On the other hand, stent diameter was significantly smaller in this group. Area under the curve was 0.774 (0.744-0.804; p<0.001) for MLR. Using a cutoff level of 4.90, MLR predicted presence of in-stent stenosis with a sensitivity of 72.6%, and specificity of 71.7%.

Conclusions: Our study results showed for the first time that increased MLR might predict in-stent restenosis better than other recent novel blood inflammatory parameters including NLR, PLR, and MPR in patients with stable angina pectoris.

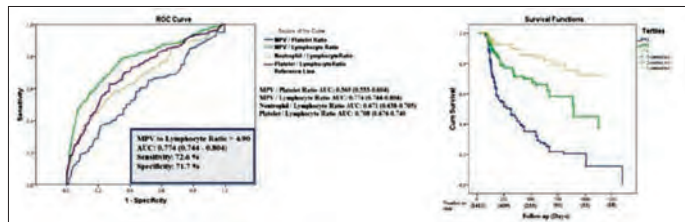


Figure 1. ROC and Kaplan-Meier Curves.

Interventional cardiology / Coronary

PP-108

Efficacy of different self-assessment anxiety scales for predicting radial artery spasm during coronary interventions

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Background and Aim: Radial artery spasm (RAS), is a major cause for procedural failure during coronary interventions performed via this route and constitutes a drawback for default usage of this method. Anxiety is a well-determined predictor of RAS, but efficacy of different measuring systems for anticipating RAS has never been compared. Here, we also aimed to assess the relationship between RAS and level of anxiety (LOA).

Methods: 123 consecutive patients scheduled for elective coronary angiography in a single default radial center were enrolled for the study. In addition to demographic features, patients were asked to fill three anxiety scales; Beck Anxiety Inventory (BAI), S-State-Trait Anxiety Inventory (s-STAI) and T-State-Trait Anxiety Inventory (t-STAI). In the cath lab, number of puncture attempts, total procedural time (TPT), largest catheter size were noted. If >2 catheters were required to complete imaging, it was additionally specified. RAS was accepted to exist, if 2 or more of predefined clinical features had been met.

Results: RAS was observed in 20 patients (16.3%). Patients were assembled in two distinct groups regarding to occurrence of RAS. In the RAS(+) group, frequency of female gender were significantly higher (% 36 vs 60, p=0.044), whereas smokers were less (% 68 vs 90, p=0.046). Remaining demographic features were comparable. With respect to operational data, TPT (mins, 23.4±10.1 vs 30.3±10; p=0.010) and procedures carried out with more than one puncture attempts (% 8 vs 15; p=0.041) were significantly higher in RAS(+) group. BAI (15.1±11.5 vs 22.3±13.6, p=0.013) and s-STAI (38.6±9.3 vs 43.4±10.6, p=0.041) scores of RAS(+) group were significantly higher. According to pre-defined cut-off values of all abovementioned scales, frequency of patients with considerable LOA between RAS(+) and RAS(-) groups were compared and only s-STAI based comparison reached statistical significance (% 45 vs 75, p=0.013). Continuous and categorical variables which had been revealed to be significantly different between groups, were involved in regression analyses. Eventually, TPT (HR:2.96, 95% CI=0.96-9.11; p=0.032) and having a s-STAI score over 40 (HR: 2.49, 95% CI=1.09-5.71; p=0.024) were designated as independent predictors of RAS.

Conclusions: Anxiety is a considerable risk factor for RAS occurrence but the testing method of LOA also matters. Regarding to our results s-STAI was the most accurate one for anticipating RAS and also established as an independent predictor.

Interventional cardiology / Coronary

PP-109

What predicts hemoglobin decline in patients undergoing elective coronary procedure

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Background and Aim: Hemoglobin (Hg) decline following elective coronary procedures is one of the most common complications of the coronary angiography. We investigated the predictors of the Hg decline in patients undergoing elective coronary procedures.

Methods: In this prospective analysis, we evaluated the predictors of hemoglobin decline on 273 patients undergoing elective coronary procedures. Patients were divided into three as no Hg decline (n=113), Hg decline <1 g/dl (n=110) and Hg decline ≥1 g/dl (n=50) groups according to post-procedure Hg decline. Independent predictors of Hg decline were evaluated.

Results: Male gender (OR 0.58, 95% CI 0.34-0.99, p=0.047), diastolic blood pressure after the procedure (OR 1.03 95% CI 1.01-1.06, p=0.009), coronary angiography as the reference procedure (OR 0.19, 95% CI 0.06-0.52, p=0.001), nitrate administration during the procedure (OR 0.33, 95% CI 0.13-0.80, p=0.015) and femoral vein puncture (OR 4.06, 95% CI 1.01-16.49, p=0.049) were detected to be independent predictors of Hg decline after elective coronary procedure.

Conclusions: Our data suggests the importance of male gender, diastolic blood pressure after the procedure, coronary angiography as the reference procedure, nitrate administration and femoral vein puncture were the predictors of Hg decline after the elective coronary procedures.

Table 1. Baseline characteristics, angiography, laboratory findings

	Hemoglobin change after the procedure			p
	No Hg decline (n=113)	Hg decline <1g/dl (n=110)	Hg decline ≥1g/dl (n=50)	
Age, y	62±13	63±14	63±12	0.835
Male gender	72 (63.7)	54 (49.1)	21 (44.0)	0.025
Body mass index	27.5±4.3	28.0±4.3	28.9±4.8	0.388
Hypertension	72 (63.7)	73 (66.4)	34 (68.0)	0.846
Diabetes Mellitus	27 (23.9)	26 (23.6)	16 (32.0)	0.480
Hyperlipidemia	37 (32.7)	27 (24.5)	14 (28.0)	0.397
Current smoker	39 (34.5)	34 (30.9)	28 (56.0)	0.007
Chronic Renal Failure	3 (2.7)	4 (3.6)	5 (10.0)	0.095
Peripheral artery disease	2 (1.8)	0 (0.0)	4 (8.0)	0.006
Hereditary	53 (46.9)	41 (37.3)	21 (42.0)	0.142
Chronic obstructive pulmonary disease	10 (8.8)	5 (4.5)	4 (8.0)	0.428
Congestive heart failure	4 (3.5)	4 (3.6)	2 (4.0)	0.821
Atrial fibrillation	4 (3.5)	3 (2.7)	3 (6.0)	0.336
Before the procedure				
Systolic blood pressure, mm Hg	142.7±26.7	139.2±20.7	138.4±16.8	0.319
Diastolic blood pressure, mm Hg	79.1±9.4	78.6±8.9	84.2±12.1	0.070
Heart rate, beats per minute	80.6±15.2	81.2±16.0	77.2±13.3	0.309
After the procedure				
Systolic blood pressure, mm Hg	130.6±18.9	137.4±17.9	138.4±16.8	0.005
Diastolic blood pressure, mm Hg	74.6±10.6	78.3±8.9	80.3±9.4	0.001
Heart rate, beats per minute	78.8±15.7	80.9±14.4	77.4±11.0	0.274
Procedure				
CAG	108 (95.6)	90 (81.8)	37 (74.0)	<0.001
PCI	4 (3.5)	13 (11.7)	9 (18.0)	0.002
CAG + PCI	1 (0.9)	1 (0.9)	4 (8.0)	0.008
Previous femoral procedure	45 (39.8)	34 (30.9)	15 (30.0)	0.287
Nitrate, intravenous administration	21 (18.6)	7 (6.4)	3 (6.0)	0.007
Previous procedure count	0.8±1.1	0.6±1.1	0.5±0.9	0.130
Femoral puncture count w/o vascular access	1.1±0.3	1.1±0.3	1.2±0.4	0.204
Femoral vein puncture	3 (2.7)	10 (9.1)	6 (12.0)	0.051
Femoral artery puncture w/o sheath insertion	1 (0.9)	5 (4.5)	5 (10.0)	0.023
Procedure duration, min: Sheath, f	21.8±12.5 6.0±0.2	23.7±13.7 6.0±0.2	23.9±14.1 6.1±0.3	0.471 0.254
Coronary Angiography				
LAD, >670	0 (0.0)	3 (2.7)	0 (0.0)	0.506
LAD, >670	43 (38.1)	47 (42.7)	13 (26.0)	0.129
LCx, >670	29 (25.1)	31 (28.2)	10 (20.0)	0.547
RCA, >670	25 (22.1)	26 (23.6)	13 (26.0)	0.863
Admission laboratory variables				
White blood cell, cells/μL	8.01±3.22	8.71±2.51	8.89±2.73	0.634
Hemoglobin before, g/dL	13.5±1.5	13.4±1.3	13.8±1.5	0.169
Hemoglobin after, g/dL	13.6±1.4	12.9±1.3	12.6±1.6	<0.001
Hemoglobin change, g/dL	-0.17±0.29	-0.47±0.29	-1.17±0.18	<0.001
Prothrombin, (mm*)	238.4±168.2	261.9±194.7	250.8±76.2	0.860
Urea nitrogen, (mg/dL)	0.22±0.11	0.23±0.16	0.20±0.07	0.671
Creatinine, (mg/dL)	1.05±0.27	1.08±0.32	1.14±0.37	0.480
BUN	20.6±12.81	21.8±11.33	22.9±11.40	0.747
ALT	27.8±14.1	29.6±15.5	28.2±13.4	0.694
AST	32.5±19.9	32.4±17.2	28.2±13.4	0.814
INR	1.07±0.14	1.08±0.14	1.07±0.14	0.931
Medication				
Acetylsalicylic acid	103 (91.2)	99 (90.0)	44 (88.0)	0.823
Clonidine	54 (49.4)	49 (44.5)	17 (34.0)	0.183
Statins	37 (32.7)	34 (31.8)	13 (26.0)	0.143
Beta-blocker	65 (57.5)	57 (51.8)	23 (46.0)	0.373

Table 2. Univariate and multivariate predictors of hemoglobin decline after coronary procedure

Univariate analysis	p value	Multivariate analysis	p value	OR(95% CI)
Gender,male	0.008	Gender,male	0.047	0.58 (0.34 - 0.99)
Systolic blood pressure,after the procedure	0.002			
Diastolic blood pressure, after the procedure	0.001	Diastolic blood pressure, after the procedure	0.009	1.03 (1.01 - 1.06)
Coronary Angiography	0.001	Coronary Angiography	0.001	0.19 (0.06 - 0.52)
Percutaneous coronary intervention	0.001			
Previous procedure count	0.060			
Nitrate administration	0.002	Nitrate administration	0.015	0.33 (0.13 - 0.80)
Femoral vein puncture	0.029	Femoral vein puncture	0.049	4.06 (1.01 - 16.49)
Femoral artery puncture without sheath insertion	0.057			

Interventional cardiology / Coronary

PP-110

Incidence of bifurcation coronary lesion as a culprit lesion in patients with acute myocardial infarction and short and long term outcomes of treatment strategy

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Background and Aim: Several studies of single and double stent techniques in patients with bifurcation lesions have been reported. However, studies of patients with acute coronary syndrome (ACS) and bifurcation lesions are still insufficient. In the present study, we assessed the short and long term outcomes of single and double stent techniques of bifurcation lesions in patients with ACS.

Methods: In our study, 2992 patients with ACS who underwent percutaneous coronary intervention (PCI) were enrolled retrospectively. Of 2992 patients, 385 patient with myocardial infarction(MI) had bifurcation lesions. The Synergy between PCI with TAXUS™ and Cardiac Surgery (SYNTAX) score, pre-PCI Thrombolysis in Myocardial Infarction (TIMI) flow, post-PCI TIMI flow, duration of procedure, angiographic features, post-PCI side branch loss, 1- and 12-month mortality rates were noted.

Results: 169 (43.9%) patient had ST segment elevation MI, whereas 216 (56.1%) patient had non-ST segment elevation MI. 355 (92.2%) patient underwent single stent and 30 (7.8%) patient underwent double stent technique. Side branch loss was found in 11.2% of patients of the single stent and in 3.3% of patients of the double stent group. Compared to single stent group, duration of angiography and procedure in double stent group were significantly longer. The rates of 1-month mortality in single and double stent group were 4.2% and 3.3%, respectively. On the other hand, 1- year mortality rates were 11.5% and 13.3%, respectively.

Conclusions: Side branch loss, short and long term mortality rates were similar in both single and double stent groups. However, related to this subject multicenter and large randomized controlled trials are needed.

Interventional cardiology / Cover and structural heart diseases

PP-111

Reduction in sympathetic activity in patients with aortic stenosis after transcatheter aortic valve implantation

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Background and Aim: Patients with aortic stenosis have increased sympathetic nervous system activity. Besides, the association between increased sympathetic activity and prolonged Tpeak-Tend (Tp-e) interval or Tp-e/QTc ratio has been demonstrated previously. This study aimed to evaluate if the procedure of transcatheter aortic valve implantation(TAVI) normalizes these parameters in patients with aortic stenosis.

Methods: Thirty-two patients with severe aortic stenosis were enrolled but 7 patients were excluded from analysis because of in-hospital mortality. In addition, 25 matched control subjects for the comparison were enrolled to the study. The severity of aortic stenosis was defined following clinical and echocardiographic examinations. Myocardial repolarization parameters (Tp-e interval, and Tp-e/QTc ratios) were measured using 12-lead electrocardiogram before and 30-days after TAVI procedure. First, the above-mentioned parameters were compared between patients with aortic stenosis and healthy control subjects. Second, these parameters were compared before and 30-days after TAVI in patients with aortic stenosis.

Results: The mean Tp-e interval (93.88±15.34) was significantly prolonged and Tp-e/QTc ratio (0.206±0.038) was significantly higher in patients with aortic stenosis compared with healthy control subjects(respectively; 79.87±5.82, 0.185±0.023). The mean transaortic gradient significantly decreased after TAVI. Compared with those before TAVI, Tp-e interval and Tp-e/QTc ratios (Figure-1) were decreased gradually. Myocardial repolarization parameters after TAVI were not significantly different when compared with control group. It was revealed that sympathetic activity which may cause malignant arrhythmias reduced by TAVI.

Conclusions: We revealed that Tp-e interval and Tp-e/QTc ratio increased in patients with severe aortic stenosis. Furthermore, TAVI had a favorable effect on sympathetic activity, namely, parameters associated with myocardial repolarization.

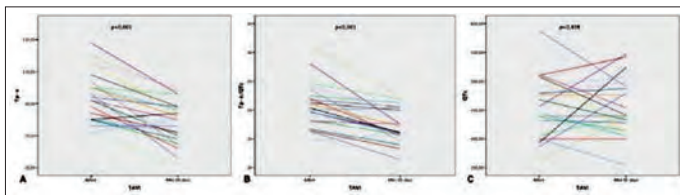


Figure 1. Changes in ECG parameters before and 30 days after transcatheter aortic valve implantation.

Interventional cardiology / Coronary

PP-112

Anomalous origination of coronary arteries from the opposite sinus: Data from a single center

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Background and Aim: Although left circumflex (LCx) artery originating from the right coronary sinus (RCS) or the proximal right coronary artery (RCA) were reported as the most common form of anomalous origination of a coronary artery from the opposite sinus (ACAOS), there are also a few studies that report RCA originating from the left coronary sinus(LCS) as the most frequent form. Methodological and racial differences may be the reason of this discrepancy. The aim of this study was to determine the most common type of ACAOS in a single center in Turkey.

Methods: We first searched our coronary angiogram database and then systematically reexamined a total of 5165 first coronary angiograms performed between 1999-2006 to disclose the most frequent type of ACAOS.

Results: The database search revealed 30 cases with ACAOS (11 RCA from the LCS, 13 LCx from the RCS or the RCA and 6 others). On the other hand, ACAOS was detected in 35 (16 RCA originating from the LCS, 13 LCx from the RCS or the RCA, and 6 others) out of 5165 coronary angiograms that were reevaluated. The most common form was RCA taking origin from LCS. 5 out of 16 cases with RCA originating from the LCS had gone unreported in the angiogram reporting database.

Conclusions: RCA originating from the LCS is the most common form of ACAOS in a single center from Turkey. Underreporting of this anomaly at the time of coronary angiography may have biased the true prevalence of this anomaly at least in some studies.

Interventional cardiology / Coronary

PP-113

Is there any link between vitamin D and coronary no-reflow phenomenon?

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Background and Aim: Coronary no-reflow phenomenon (CNP) is associated with an increased risk of major cardiovascular adverse events. Vitamin D is closely associated with hypertension, stroke, myocardial infarction, cardiovascular adverse events, and endothelial dysfunction. Considering endothelial dysfunction is one of the main responsible factors of CNP, we aim to evaluate the association between vitamin D and CNP.

Methods: The study group consisted of 109 patients. Taking into consideration the inclusion criteria, 60 patients with CNP and 49 patients without CNP were included in the study. CNP defined as TIMI grade <3 at the end of the procedure in the absence of any coronary dissection/spasm and/or less than 70% ST resolution at first hour ECG.

Results: Prevalence of CNP was found 55% in this study group. On univariate analysis age, balloon predilatation, stent diameter, serum creatinine, vitamin D, CRP level, initial TIMI flow <2, and reperfusion time >3 h were associated with CNP. On multivariate analysis reperfusion time >3 h, initial TIMI flow <2, and serum creatinine level were the independent predictors for CNP (OR 5.182; 95% CI: 3.159-8.327; p<0.001, OR 4.061; 95% CI: 2.729-6.327; p<0.001, OR 3.301; 95% CI: 1.937-4.623; p<0.001; respectively).

Conclusions: Prevalence of CNP was found 55% in this study group. On univariate analysis age, balloon predilatation, stent diameter, serum creatinine, vitamin D, CRP level, initial TIMI flow <2, and reperfusion time >3 h were associated with CNP. On multivariate analysis reperfusion time >3 h, initial TIMI flow <2, and serum creatinine level were the independent predictors for CNP (OR 5.182; 95% CI: 3.159-8.327; p<0.001, OR 4.061; 95% CI: 2.729-6.327; p<0.001, OR 3.301; 95% CI: 1.937-4.623; p<0.001; respectively).

Interventional cardiology / Cover and structural heart diseases

PP-114

Long-term survival analysis following transcatheter aortic valve implantation

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Background and Aim: Transcatheter aortic valve implantation (TAVI) has been used for over a decade as a less invasive option for those who cannot undergo surgical aortic valve replacement due to high risk of surgical complications. Since the most patients who underwent TAVI are aged 70 or older, survival analysis studies become important during follow-up period. In this study, we evaluated long-term survival analysis in patients, who underwent TAVI procedure in Cardiology Department of Bezmialem Vakif University Medical Faculty.

Methods: In this study, we retrospectively evaluated 275 consecutive patients who underwent TAVI in Cardiology Department of Bezmialem Vakif University Medical Faculty between 2012 to 2017. Baseline characteristics, postoperative results and as main outcome early and long-term mortality data was reviewed. The longest follow-up period was 6 years. Cox regression analysis for long-term mortality predictors and Kaplan-Meier survival analysis were performed.

Results: Mean age of the patients was 78.2±7.5 years and 174 were female (63.2%). Average logistic Euroscore was 20.9±4.2. Transfemoral access was used in all TAVI procedures and success rate was 98.5%. Postoperative 30-days mortality rate was 10.9%. During postoperative 24.9±20.6 months follow-up period (1-75 months), mortality rate was 41.0% with 113 patients. Survival rates for postoperative 1st, 3rd, 5th years were 83.2%, 63.8%, 42.3.2%, respectively (Figure 1). In a Cox proportional hazard model, left ventricular ejection fraction less than 35% (OR: 1.58, 95% CI 1.03-2.43, p=0.034), coincidence of moderate to severe mitral insufficiency (OR: 1.87, 95% CI 1.21-2.88, p=0.005) were significant independent predictors of long-term mortality.

Conclusions: We thought that favorable long-term survival rates suggested TAVI as a promising option for patients with symptomatic aortic stenosis. Following TAVI procedure survival rates were better in high risk patient with normal ejection fraction and pure aortic valve disease.

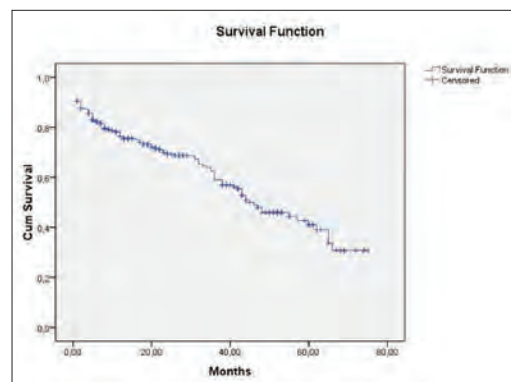


Figure 1. Kaplan-Meier survival analysis of TAVI mortality.

Interventional cardiology / Coronary

PP-115

Coronary artery diameters and distributions in Turkish population:
A large quantitative coronary analysis studyIbrahim Halil Kurt,¹ Yurdaer Donmez, Abdullah Yildirim, Omer Genc,
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Background and Aim: Major coronary artery (CA) diameters including especially the left main coronary artery (LMCA) are an important predictor for the success of revascularization therapies. In the international literature, there are some studies about angiographic findings of CA in different populations. But, there is limited information in the national literature. Our aim was to assess the LMCA and major CA diameters with Quantitative Coronary Analysis software, and to report their distributions in Turkish population.

Methods: In 2016, a total of 1139 patients who admitted to our polyclinic unit with complaints of chest pain were retrospectively included to our study. All patients had normal CA in the elective coronary angiography. Acute coronary syndrome and primary or elective percutaneous intervention performed patients were excluded. Angiographic views were evaluated and CA diameters were measured with Quantitative Coronary Analysis software. Anatomical findings of CA were recorded. The CA were measured from the best evaluated angiographic view.

Results: There were 528 woman (46.4%), and 611 man (53.6%) in our study group. The mean age was 57.3±11.4 years. There was no LMCA in 81 (7.1%) patients. A total of 183 patients had intermediate artery (16.1%). It was determined that 106 patients had (9.3%) rudimentary RCA. Fifty-six patients (4.9%) had rudimentary circumflex artery. Rudimentary LAD artery was present in 1 (0.1%) patient. Conus artery originated from the RCA in 957 (84.0%) patients and from the separate ostium in 182 (16.0%) patients. Dominant artery was the RCA in 693 (60.8%) patients and the circumflex artery in 314 (27.6%) patients. There was codominance in 132 (11.6%) patients. The mean proximal and distal diameters of LMCA were 4.86±1.07 mm and 4.64±1.03 mm in men, respectively. Women's mean proximal LMCA diameter was 4.74±0.93 mm and mean distal LMCA diameter was 4.44±0.85 mm. The mean length of LMCA was 7.72±3.68 mm. The mean circular diameter of LMCA was 17.3±7.27 mm². The mean diameters of LAD, circumflex, and right coronary arteries were 3.49±0.7 mm, 3.32±0.72 mm, 3.22±0.77 mm, respectively. The mean large diagonal artery and obtuse margin branch diameters were 1.92±0.75 mm and 1.82±0.82 mm, respectively.

Conclusions: As stated in the objective section, there is no detailed information in national literature. Nationwide, our study was the first in this subject. And, it has an importance as a knowledge in the selection of stent diameters for the interventional cardiologists.



Figure 1. (a) ECG example of single lead fQRS in a patient with anterior MI. (b) ECG example of ≥ 2 leads with fQRS in a patient with inferior MI.

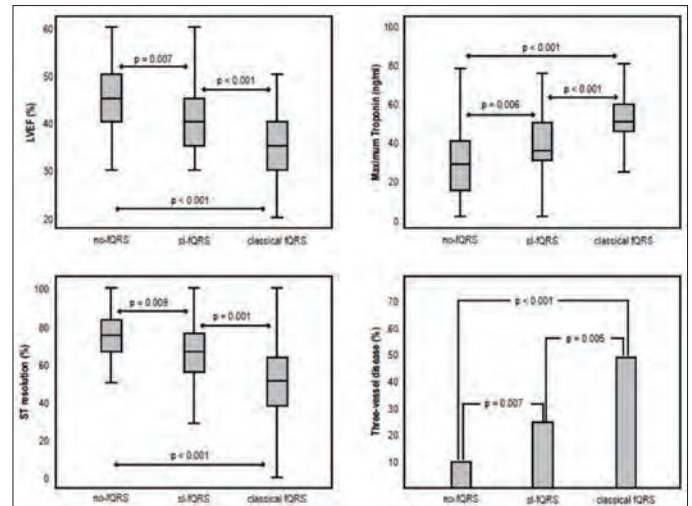


Figure 2. Comparisons among groups in terms of LVEF, maximum troponin, ST resolution, and the frequency of three-vessel disease. LVEF: left ventricular ejection fraction; fQRS: Fragmented QRS.

Interventional cardiology / Cover and structural heart diseases

PP-116

A novel concept for the definition of fragmented QRS:
Single-lead fragmented QRSZulkif Tanrıverdi,¹ Huseyin Dursun,² Tugce Colluoglu,³ Dayimi Kaya²¹Department of Cardiology, Harran University Faculty of Medicine, Şanlıurfa²Department of Cardiology, Dokuz Eylül University Faculty of Medicine, İzmir³Department of Cardiology, Karabük University Faculty of Medicine, Karabük

Background and Aim: Fragmented QRS (fQRS) on 12-lead electrocardiography (ECG) is classically defined by the presence of a single or multiple notches in the R or S wave, without a typical bundle branch block, in ≥ 2 contiguous leads corresponding to major coronary artery territories. The clinical significance of classical fQRS (in ≥ 2 contiguous leads) has been investigated in several studies, and it was found to be associated with increased mortality, myocardial scarring, cardiac arrhythmias, and adverse cardiac events. Because the concept of ≥ 2 derivations is mentioned for the classical definition of fQRS, the importance of the only one lead with fQRS (single-lead fQRS, sl-fQRS) is not known. The aim of our study was to investigate the clinical importance of sl-fQRS in patients with acute STEMI who had successful revascularization with primary percutaneous coronary intervention (pPCI).

Methods: A total of 330 patients with acute STEMI who had been successfully revascularized with pPCI were included in this study. The patient's electrocardiography was obtained in the first 48 hours, and the patients were divided into three groups according to the presence of fQRS on surface ECG: absence of fQRS in any lead (no-fQRS), its presence in a single lead (sl-fQRS) (Figure 1a), and its presence in two or more contiguous leads (classical fQRS) (Figure 1b).

Results: Patients with sl-fQRS had a lower LVEF (p=0.007), a lower ratio of STR (p=0.009), higher maximum troponin levels (p=0.019), and a higher rate of three-vessel disease (p = 0.007) than patients with no-fQRS. Similarly, patients with ≥ 2 leads with fQRS also had a lower LVEF, a lower ratio of STR, higher maximum troponin levels, and a higher rate of three-vessel disease than patients with sl-fQRS. Also, in-hospital mortality was significantly higher both in patients with sl-fQRS and in patients with ≥ 2 leads with fQRS compared to patients with no-fQRS. In ROC curve analysis, ≥ 1 leads with fQRS yielded a sensitivity of 75% and specificity of 57.4% for the prediction of in-hospital mortality. Multivariate analysis showed that sl-fQRS is an independent predictor of in-hospital mortality (OR: 3.989, 95% CI: 1.237-12.869, p=0.021).

Conclusions: The concept of at least two derivations is mentioned for the classical definition of fQRS, and only one lead with fQRS has not been accepted for the presence of fQRS. However, we showed for the first time that sl-fQRS is associated with greater extent of necrotic myocardium, increased in-hospital mortality and higher risk. Therefore, instead of the concept of at least two derivations, the presence of fQRS in only one lead and/or ≥ 1 leads with fQRS may also be enough when describing the patients under high cardiac risk. Further studies are needed to understand the importance of sl-fQRS.

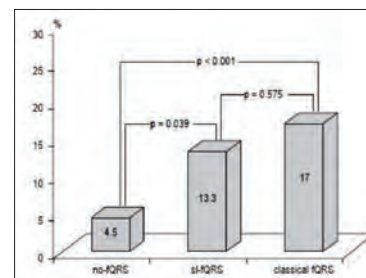


Figure 3. Comparisons among groups in terms of in-hospital mortality.

Interventional cardiology / Coronary

PP-117

Relation of killip classification to no-reflow phenomenon in patients
with acute ST-segment elevation myocardial infarction
undergoing primary coronary intervention

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Background and Aim: Primary goal in the management of acute ST segment elevation myocardial infarction (STEMI) is to open the culprit artery occlusion at an early stage. Primary percutaneous coronary intervention (PPCI) is the currently best available reperfusion therapy in patients with acute STEMI. Coronary no-reflow (NR) phenomenon is observed when cardiac tissue fails to perfuse normally despite opening of the occluded vessel and the absence of spasm, dissection, or distal macro-embolus. Multiple factors may contribute to development of no-reflow. These include distal embolization, ischemia-reperfusion injury resulting from oxygen free radical production, microvascular damage, myocardial necrosis and stunning, release of active tissue factor from the dissected plaque, and vasoconstriction secondary to alpha adrenergic tone, thromboxane A2 and serotonin released from platelets. The development of no-reflow is multifactorial, and the etiology is not fully understood. The association between killip classification and the no-reflow of the infarct-related artery (IRA) in patients with STEMI remains unknown. In this study, we investigated the relationship between killip classification and the no-reflow of IRA in patients with STEMI undergoing PPCI.

Methods: A total 646 patients with acute STEMI who underwent PPCI were enrolled. The patients were

divided into 2 groups according to thrombolysis in myocardial infarction (TIMI) flow grades after PPCI. Killip grades were determined on admission. Univariate and multivariate logistic regression analyses were conducted to assess association of killip grade and no-reflow.

Results: In the current study, 84 presented with no-reflow after PPCI, with an incidence of 13%. The patients in the no-reflow group had a significantly higher Killip grade (≥ 2 ; 35.7% vs 18.1%, $p < 0.001$). The multivariate logistic regression models revealed that killip grade ≥ 2 (OR=2.325, 95% CI:1.622-2.652; $p < 0.001$).

Conclusions: Our result suggest that killip grade showed a moderate diagnostic performance regarding the prediction of no reflow in patients with STEMI undergoing PPCI.

Table 1. Baseline characteristics and angiographic findings

	Normal-reflow (n=562)	No-reflow (n=84)	p value
Age (years)	62.5±12.5	66.8±12.5	<0.001
Male (%)	448 (79.7%)	68 (80.9%)	0.326
Hypertension (%)	226 (40.4%)	38 (45.2%)	<0.001
Diabetes Mellitus (%)	158 (28.1%)	27 (32.1%)	0.001
History of smoking (%)	130 (23.1%)	18 (21.4%)	0.285
Hyperlipidemia (%)	135 (24%)	20 (23.8%)	0.724
Family History of Coronary artery Disease (%)	52(9.2%)	8 (9.5%)	0.653
Previous myocardial infarction (%)	18 (3.2%)	3(3.6%)	0.105
Previous percutaneous coronary Intervention (%)	113 (20.1%)	18 (21.4%)	0.640
Previous coronary artery bypass grafting (%)	32 (5.7%)	12 (14.3%)	<0.001
Heart rate (beat/minute)	80±14	81±21	0.350
Time from symptoms onset to PCI (hours)	7.0±10.6	9.0±11.5	<0.001
Door to balloon time(minutes)	19.4±11.2	19.6±10.8	0.852
Left ventricular Ejection Fraction (%)	46.7±11.5	41.6±12.1	<0.001
Cardiac Function Killip Grade (%)			<0.001
	1 460 (82%)	54 (64.3%)	
	2 45 (8.0%)	10 (11.9%)	
	3 27 (4.8%)	9 (10.7%)	
	4 30 (5.2%)	11 (13.1%)	
Peak creatinin kinase-MB (U/L)	150.3±148.5	200.4±186.2	<0.001
Multivessel disease (%)	1365(41%)	279(59.2%)	<0.001
Preintervention TIMI-flow (%)			<0.001
	0 393 (70%)	64 (76.3%)	
	1 62 (11.0%)	12 (14.2%)	
	2 84 (14.9%)	5 (5.9%)	
	3 23 (4.1%)	3 (3.6%)	
Thrombus burden (%)			<0.001
low thrombus burden	270 (48%)	35 (41.6%)	
high thrombus burden	292 (52%)	49 (58.4%)	
Length of target lesion (mm)	14.5±12.0	20±8.3	<0.001
Reference vessel diameter (mm)	3.1±1.1	2.5±0.9	<0.001
Reperfusion Method (%)			<0.001
Balloon dilation	67 (11.8%)	13 (15.4%)	
Balloon predilatation+stent implantation	393 (70%)	64 (76.2%)	
Stent implantation	102 (18.2%)	7 (8.4%)	
Thrombus Aspirator (%)	33 (5.8%)	6 (7.1%)	0.120
Use of Tirofiban (%)	140(25.1%)	38 (45.2%)	<0.001

Data are presented as mean.

Table 2.

Variable	Univariate			Multivariable		
	Unadjusted OR	95% CI	p value	Adjusted OR	95%CI	p value
Age	1.079	1.042-1.098	<0.001	1.052	1.034-1.058	<0.001
Gender (male)	0.721	0.403-1.063	0.113			
Hypertension	1.581	1.283-1.942	<0.001			
Diabetes mellitus	1.454	1.054-1.807	0.003	1.625	1.009-2.556	0.03
Multivessel disease	1.854	1.492-2.177	<0.001	1.604	1.107-1.985	0.002
Reference vessel diameter (mm)	0.440	0.405-0.468	<0.001	0.404	0.329-0.542	<0.001
Length of target lesion (mm)	1.112	1.098-1.115	<0.001			
Time to balloon time (hours)	1.190	1.125-1.112	<0.001	1.080	1.021-1.045	0.002
Killip Grade (≥2)	2.799	2.325-2.793	<0.001	2.325	1.522-2.652	<0.001
Preintervention TIMI-flow	0.582	0.626-0.721	<0.001	0.525	0.3671-0.648	<0.001
Thrombus Burden	1.364	1.141-1.487	<0.001	1.450	1.181-1.708	0.003
Reperfusion method (balloon predilatation +stent implantation)	2.520	2.244-3.152	<0.001	1.764	1.123-2.503	0.03

OR:Odds ratio; CI:Confidence interval; TIMI:Thrombolysis in Myocardial Infarction

Interventional cardiology / Cover and structural heart diseases

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Effect of prognostic nutritional index on survival after transcatheter aortic valve implantation

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Background and Aim: Transcatheter aortic valve implantation (TAVI) is a good alternative to surgical aortic valve replacement in high-risk patients in severe aortic stenosis. Risk scores such as Society of Thoracic and Surgeons (STS) score and EuroSCORE include much comorbidity, but do not contain functional decline specific to elder people. Adding nutritional index to these risk scores could provide more information to predict mortality after TAVI. Our aim of the study is to determine the effect of prognostic nutritional index (PNI) on post-TAVI survival.

Methods: A total of 184 patients underwent TAVI were included. PNI was calculated using preoperative serum albumin value and total lymphocyte count in the peripheral blood. Study population was divided into

two groups according to cut-off value of PNI. We assessed predictive value of PNI on the 30-day mortality after TAVI.

Results: Cut-off value of PNI for 30-day survival determined with ROC analysis was 43.4 with 75% specificity and sensitivity. Patients with higher PNI were significantly higher incidence of survival compared to those with lower PNI (Chi Square value: 17.61, $p < 0.001$). The negative predictive value for PNI to predict 30-day survival was 96.9%. PNI and postoperative acute kidney injury were found to be independent risk factors for 30-day mortality after TAVI in multivariate analysis.

Conclusions: The higher level of PNI was found to be protective against mortality and related with the survival.

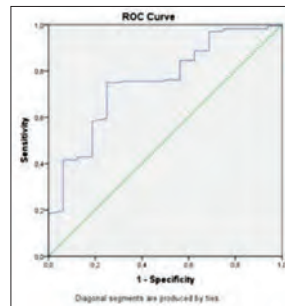


Figure 1. The ROC curve obtained for 30-day survival-predictive cut-off value of PNI.

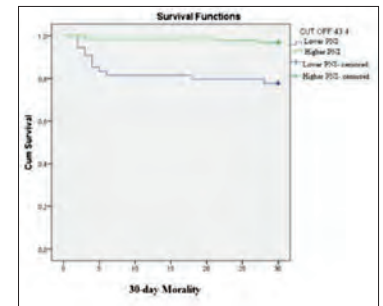


Figure 2. Graphical presentation of the 30 day survival rate of higher and lower PNI groups (Kaplan Meier survival analysis, Log Rank $p < 0.000$).

Table 1. Patient characteristics

Characteristics	All study participants (n=184)
Age—mean ± SD—years	79.4 ± 7.8
Female—no. (%)	113 (61.4)
EuroScore—mean ± SD	32,6 ± 15,3
STS Score—mean ± SD	12,0 ± 5,0
NYHA Class III—no. (%)	108 (58,7)
NYHA Class IV—no. (%)	65 (35,3)
Coronary Artery Disease—no. (%)	114 (62)
Chronic Obstructive Pulmonary Disease—no. (%)	112 (60,9)
Diabetes Mellitus—no. (%)	74 (40,2)
Chronic Kidney Disease—no. (%)	54 (29,3)
Hypertension—no. (%)	151 (82,1)
Coronary Artery Bypass Surgery—no. (%)	44 (23,9)
Pulmonary Hypertension—no. (%)	124 (67,4)
Peripheral Artery Disease—no. (%)	68 (37)
Cerebrovascular Disease—no. (%)	8 (4,3)
Atrial Fibrillation—no. (%)	30 (16,3)
Permanent Pacemaker—no. (%)	7 (3,8)

Table 2. TAVI procedural details

Variables	All study participants (n=184)
Access site	
Transfemoral—no. (%)	172 (93.5)
Transapikal—no. (%)	9 (4.9)
Transsubclavian—no. (%)	1 (0.5)
Transaortic—no. (%)	2 (1.1)
Valve system	
Core Valv—no. (%)	7 (3.8)
Lotus—no. (%)	26 (14.1)
Edwards Sapien—no. (%)	147 (79.9)
Portico—no. (%)	4 (2.2)
Valve Size	
23—no. (%)	60 (32.6)
25—no. (%)	15 (8.2)
26—no. (%)	69 (37.5)
27—no. (%)	6 (3.3)
29—no. (%)	34 (18.5)
Closure	
Prostar—no. (%)	118 (64.1)
Cutdown—no. (%)	50 (27.2)
Proglide—no. (%)	6 (3.3)
Opaque volume—ml	178.85 ± 26.86

Table 3. Post-TAVI complications

Complications	All study participants (n=184)
Acute Kidney Injury (AKI) — no. (%)	41 (22.3)
AKI stages:	
Stage 1 — no. (%)	22 (53.7)*
Stage 2 — no. (%)	11 (26.8)*
Hemodialysis — no. (%)	6 (3.3)
Permanent pacemaker — no. (%)	29 (15.8)
Blood transfusion — no. (%)	42 (22.8)

*Among the AKI patients

Table 4. Preoperative and postoperative echocardiographic parameters

Parameters	Preoperative	Postoperative
Aortic valve area — mean ± SD	0,730 ± 0,163	1,945 ± 0,334
Peak gradient — mean ± SD	82,24 ± 20,51	18,97 ± 7,827
Mean gradient — mean ± SD	50,97 ± 13,13	10,01 ± 4,435
Left ventricular EF — mean ± SD	53,68 ± 12,20	54,17 ± 10,64
Intermediate and severe mitral regurgitation — no. (%)	52 (28.3)	
≥ 2 + paravalvular aortic regurgitation — no. (%)		27(14.7)

Table 5. Comparison of the 30-day survival rate of the subgroups according to the PNI cut off value of 43.4

	Death	Survival	Total	
PNI < 43.4	12	42	54	PPV: %22.2
PNI > 43.4	4	126	130	NPV: %96.9
Total	16	168	184	
	Sensitivity: %75	Specificity: %75		
	+LHR: 3	-LHR: 0.33		

PPV: Positive Predictive Value, NPV: Negative Predictive Value, +LHR: Positive Likelihood Ratio, -LHR: Negative Likelihood Ratio.

Interventional cardiology / Coronary**PP-119**

Is the bolus intravenous administration of sodium bicarbonate immediately before contrast exposure more preventive strategy at contrast induced nephropathy in patient with also moderate-severe renal insufficiency

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Background and Aim: Contrast induced nephropathy (CIN) causes the third most frequently hospital acquired acute renal failure. Some randomized controlled multicenter trials have recently shown that among patients at high risk for renal complications who were undergoing angiographic procedure, there was no superiority between some protection strategies for the prevention of death, need for dialysis even from prevention of CIN. But some other studies showed the preventive effect of hydration and/or bicarbonate. So there are some controversies in this era. Also the effect of bolus administration of sodium bicarbonate is not well known. We aimed to compare the effect of a single-bolus iv administration of sodium bicarbonate immediately before contrast exposure in addition to hydration with sodium chloride alone to prevent CIN in patient with moderate and severe renal insufficiency.

Methods: 200 patients (70.5% men, mean age 72.03±10.78 years) with moderate to severe renal insufficiency who were undergoing angiographic procedure [coroner angiography, percutaneous coronary intervention, periferic angiography or transcatheter aortic valve implantation] were included in this study. Data were scanned retrospectively and were assigned to the following 2 groups. The sodium bicarbonate group (Group A, n=100) was defined as the patients who received standard hydration (SH) with sodium chloride plus a single bolus iv administration of sodium bicarbonate (20 meq) immediately before contrast exposure and Group B (n=100) defined as SH with sodium chloride alone. SH defined as the patients who received sodium chloride infusion 12 hours before the contrast exposure and continued to receive 12 hours after the procedure. Serum creatinine and the glomerular filtration rate (eGFR) were evaluated before as well as 24 and 48 hours after contrast media injection. The primary end point was development of CIN (defined as an increase >25% or >0.5 mg/dl in serum creatinine within 2 days after the procedure) and acute renal failure requiring dialysis because of the contrast exposure. The secondary end point was MACE (cardiovascular death, cerebrovascular event or myocardial infarction) or hospitalization with cardiovascular events for the long term follow-up.

Results: There were no significant differences between the 2 groups in clinical, procedural and biochemical characteristics. The incidence of CIN was significantly lower in Group A than in group B (9.0% vs 20.0%, p=0.026) but the incidence of dialysis because of the contrast exposure is not significant in 2 groups. There were no significant differences between 2 groups in secondary end points (39.0% vs 35.2%, p=0.58)

Conclusions: Our study suggests that single-bolus iv administration of sodium bicarbonate in addition to SH immediately before contrast exposure is more effectively to prevent CIN than SH alone in patients with moderate-severe renal insufficiency undergoing an angiographic procedure.

Interventional cardiology / Cover and structural heart diseases**PP-120**

Relation between post-TAVI blood pressure response, micro RNAs mir 1, 133a, 206, 202-3p and their implication on myocardial strain parameters

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Background and Aim: Recent studies have shown that blood pressure (BP) response after TAVI is strongly linked to post-TAVI outcome. However results are conflicting. Normally after successful TAVI we expect an improvement in myocardial deformation, which should be causally linked to blood pressure development. Improvement in myocardial global strain occur earlier than improvement of left ventricular ejection fraction, that remains unaltered directly after the procedure. Circulating microRNAs are emerging as a novel class of biomarkers. They can be secreted by cells within exosomal particles. Previous studies have demonstrated that the circulating levels of specific miRNAs are associated to LV remodelling and to changes in shear stress across the aortic valve. This study sought to investigate the blood pressure response after transcatheter aortic valve implantation and its association with micro RNAs 1, 133a, 206, 202-3p and myocardial strain parameters.

Methods: Sixty patients with symptomatic aortic valve stenosis will be included in the study, while fifteen patients have been already recruited. Blood pressure measurements are conducted by 24-hours BP monitoring before the procedure, before discharge and at 3 months follow up. Likewise, micro RNA levels in exosomes, obtained from peripheral blood samples, and echocardiographic myocardial strain parameters are measured by echocardiography before the procedure, 24h after the procedure, at discharge and at 3 months FU. Major adverse cardiovascular events (MACEs), including myocardial infarction, heart failure symptoms NYHA >2, hospitalisation for any cardiovascular reason and all cause death will be determined at 1 year FU.

Results: Blood pressure development post-TAVI was incongruent, with rise in 4 patients and a non-significant fall in 5 patients, with a median value around 130 mmHG (range 110-160). Global strain parameters improved significantly after the procedure at discharge, compared to pre-procedural determined parameters (p<0.05). Micro RNA results are in progress and will be obtained after 30 patients are included.

Conclusions: Strain echocardiography seems to be able to detect changes in LV systolic function occurring early after TAVI in severe AS. Ist relation to blood pressure changes and micro RNA levels is under progress.

Interventional cardiology / Coronary**PP-121**

Evaluation of spatial QRS-T angle in patients with coronary slow flow phenomenon

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Background and Aim: Spatial QRS-T angle (s-QRS-T) is defined as the difference between mean vectors of depolarization and repolarization. It has been found that associated with adverse cardiac events. Coronary slow flow phenomenon (CSFP) is defined as a microvascular disorder characterized by delayed distal vessel opacification in the absence of significant obstructive epicardial coronary disease. Although the relationship between atherosclerosis-adverse cardiac events is clearly known, the association between CSFP - adverse cardiac events has not been clearly determined. This study is planned to investigate whether s-QRS-T that is marker of adverse cardiac events, is increased in patients with CSFP.

Methods: In a retrospective study, a total of 162 participants [84 patients with CSFP and 78 controls (control subjects had a negative treadmill test and normal coronary computed tomography)] were enrolled into the study. s-QRS-T was measured and recorded in both groups. The statistical difference between the two groups was examined.

Results: There was a statistically significant difference between the groups in terms of s-QRS-T angle values [control group s-QRS-T angle value median 37 (32-52; Interquartile range=20), CSFP group s-QRS-T angle value median 80.2 (36.6-115.5; Interquartile range=78.9), p<0.001].

Conclusions: According to these results, s-QRS-T value was elevated in patients with CSFP than in normal population. Furthermore, these results may indirectly suggest that the risk of adverse cardiac events may be increased in patients with CSFP. s-QRS-T angle is a new marker of myocardial repolarization and associated with adverse cardiac events. According to our study CSFP may not be a totally benign condition, but the number of patients in this study was limited and more extensive studies are necessary.

Table 1.

	CSF (n=84)	Control (n=79)	p
Female, n (%)	20 (39.3)	24 (33.7)	0.345
Age	53±10.2	55.8±9.3	0.075
QRS-T Angle	80.2 (36.6-115.5; IQR=78.9)	37 (32-52; IQR=20)	<0.001

Interventional cardiology / Coronary

PP-122

Basophil levels in isolated coronary artery ectasia

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Background and Aim: The pathophysiology of isolated coronary artery ectasia (CAE) involves atherosclerosis, and inflammation. It has been known that basophils play a significant role in inflammation, atherosclerosis and endothelial dysfunction. We propose to evaluate the relationship between basophilia and isolated CAE.

Methods: All patients who underwent coronary angiography between January 2013 and April 2018 evaluated retrospectively. Of 10985 patients, 173 patients with isolated CAE (107 males) and 220 control subjects with normal coronary angiography (NCA) and atherosclerosis risk factors-matched subjects (119 males) were enrolled in this study. Baseline characteristics, hematological and biochemical test results were obtained from the hospital database.

Results: Patients with angiographic isolated CAE had significantly elevated WBC (Wight blood cell) count and basophil count when compared to the patients with NCA [7.87 (6.83-9.42)103/mm³ vs 7.48 (6.27-8.67) 103/mm³, p=0.01; 0.04 (0.03-0.05) 103/mm³ vs 0.03 (0.02-0.05)103/mm³, p=0.03, respectively] (Table 1). The ROC curve analysis demonstrated that the specificity of a basophil value >0.037 (measured prior to coronary angiography) in predicting isolated CAE was 57.3% and the sensitivity was 57.2% (area under the curve [AUC] 0.562, 95% CI 0.505, 0.618; p=0.03) (Figure 1).

Conclusions: Patients with isolated CAE have higher blood basophil count, and this may play an important role in the pathogenesis of isolated CAE.

Table 1. Inter-group comparison of demographical and laboratory data

	Isolated CAE (173)	NCA (220)	P value
Sex, n (male/ female)	107/66	119/101	0.12
Hypertension, n (%)	58/173 (33.5)	59/220 (26.8)	0.15
Hyperlipidemia, n (%)	71/173 (41.0)	82/220 (37.3)	0.44
Diabetes mellitus, n (%)	39/173 (22.5)	45/220 (20.5)	0.61
Smoking, n (%)	66/173 (38.2)	71/220 (32.3)	0.22
Age, (year)	56.0 (52.5-61.0)	55.0 (51.0-59.0)	0.06
Platelet, (103/mm ³)	260.0 (227.5-298.0)	255.0 (214.25-300.0)	0.19
Glucose, (mg/dl)	99.0 (90.0-110.50)	96.0 (89.0-108.0)	0.14
Triglycerides(mg/dL)	150.0 (108.50-218.0)	137.45 (115.0-155.0)	0.09
Low density lipoprotein cholesterol, (mg/dl)	115.0 (92.5-139.0)	112.0 (90.0-125.0)	0.05
Total cholesterol, (mg/dl)	190.0 (161.5-213.5)	188.20 (166.15-200.0)	0.28
HDL cholesterol, (mg/dL)	41.0 (35.8-50.0)	46.95 (40.0-56.0)	<0.0001
Basophil,(103/mm ³)	0.04 (0.03-0.05)	0.03 (0.02-0.05)	0.03
Hemoglobin,(g/dl)	14.40 (13.5-15.15)	14.0 (13.5-15.0)	0.08
Hematocrit, (%)	42.90 (40.35-45.60)	42.0 (40.5-45.0)	0.13
White blood cell,(103/mm ³)	7.87 (6.83-9.42)	7.48 (6.27-8.67)	0.01
Urea, (mg/dl)	30.0 (24.0-36.0)	29.85 (23.7- 35.0)	0.24
Creatinine, (mg/dl)	0.69 (0.51-0.80)	0.67 (0.57-0.78)	0.72
Sodium, (mmol/L)	140.0 (138.0-142.0)	140.0 (137.0-142.0)	0.29
Potassium, (meq/L)	4.35± 0.40	4.42± 0.51	0.15#
Calcium, (mg/dl)	9.24± 0.46	9.28± 0.48	0.38#

#Normality of the distribution was evaluated by the Kolmogorov-Smirnov test and the Mann-Whitney U test applied to compare for continuous variables except from potassium and calcium

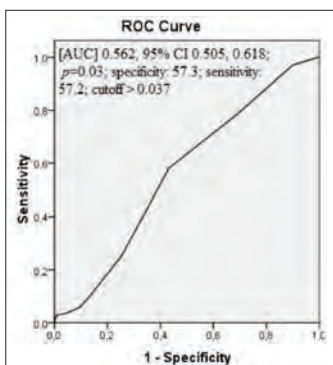


Figure 1. Receiver operator characteristic curve analysis against normal coronary anatomy (NCA) showing specificity and sensitivity of basophil counts in predicting isolated coronary artery ectasia (CAE). Receiver-operating characteristic (ROC) analysis between patients have isolated coronary artery ectasia (CAE) and subjects have normal coronary angiography (NCA). AUC; area under the curve, CI; Confidence interval.

Interventional cardiology / Coronary

PP-123

Acute hyperglycemia and contrast-induced nephropathy in patients with non ST elevation miyocardial infarction

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Background and Aim: Acute hyperglycemia and contrast-induced nephropathy (CIN) are frequently observed in Non ST-elevation myocardial infarction (NSTEMI) patients undergoing percutaneous coronary intervention (PCI), and both are associated with an increased mortality rate. We investigated the possible association between acute hyperglycemia and CIN in patients with NSTEMI undergoing PCI.

Methods: We prospectively enrolled 163 (85, 52% men) NSTEMI patients undergoing PCI. For each patient, plasma glucose levels were assessed at hospital admission. Acute hyperglycemia was defined as glucose levels >198 mg/dl (Group 1). Contrast-induced nephropathy was defined as an increase in serum creatinine 25% or 0.5 mg/dl from baseline in the first 48-72 hours.

Results: Overall, 34 (20.9%) patients had acute hyperglycemia; and 9 (26.5%) patients developed CIN. Patients with acute hyperglycemia had higher incidence of CIN than those without acute hyperglycemia (26.5% vs 9.3%, p=008) and in patients group 1; length of hospital stay, major bleeding, requirement of mechanical ventilation and dialysis were observed significantly higher than in patients group 2 (p<0.001, p=0.005, p=0.029, p=0.007, respectively) (Table 1).

Conclusions: In NSTEMI patients undergoing primary PCI, acute hyperglycemia is associated with an increased risk for CIN and in-hospital morbidity.

Table 1. Main characteristics of group 1 and 2

	Group 2	Group 1	p
Age (year)	56.9± 10.6	55.0± 11.1	NS
Men (n%)	64 (49.6%)	21(61.8%)	NS
Weight (kg)	75.8±6.0	78.3±3.6	0.003
HT (n%)	76(58.99%)	22 (64.7%)	NS
HL (n%)	47 (36.4%)	14 (41.2%)	NS
DM (n%)	38 (29.5%)	9 (26.5%)	NS
Smoker (n%)	38 (29.5%)	7(20.6%)	NS
Previous Myocardial Infarction(n%)	42(32.6%)	11 (32.4%)	NS
Previous CABG(n%)	33 (25.6%)	7(20.6%)	NS
Creatinine before PCI (mg/dl)	1.02±0.8	1.03±1	NS
EF(n%)	53.7±	53.2±	NS
eGFR (ml /min 1,73 ²)	88.2± 15	95.2± 17.2	0.022
Glucose at admission, (mg/dl)	90.4± 7.7	220.8± 18.4	<0.001
Contrast volume- ml	228.76± 17	227.35± 18.4	NS
Contrast nephropathy (n%)	9 (9.3%)	12 (26.5%)	0.08
Time to reperfusion h	4.0± 2.1	4.8± 2.2	NS
Troponin (ng/dl)	2.3± 0.9	2.2± 0.8	NS
Length of hospital stay (days)	4.4± 0.6	5.9± 2	<0,001
Major bleeding (n%)	2 (1.6%)	4 (11.8%)	0.005
Dialysis (n%)	1 (0.8%)	3 (8.8%)	0.007
Mechanical ventilation (n%)	2 (1.6%)	3 (8.8%)	0.029
In-hospital mortality(n%)	4 (3.1%)	3 (8.8%)	NS

Interventional cardiology / Coronary

PP-124

Analysis of early and long term mortality bare metal coronary stents versus drug elution stents in patients with large coronary vessel diameters

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Background and Aim: We aimed to compare early and long term outcomes of BMS and DES for patients with large coronary vessels (≥3.5 mm diameter).

Methods: Between 2007 and 2013, 241 patients that 3.5 mm and larger diameter stents implanted were evaluated in the study. Baseline characteristics, coronary risk factors and coronary angiography data assessed retrospectively. 1 year follow-up MACE data and 3 year survival data were collected.

Results: Mean age was 61.2±11.4 years. 17% of the patients were female. Patients with HT, DM, smoking, dyslipidemia and family history were 37.8%, 15.8%, 26.1%, 36.1% and 37.8% respectively. 72 (29.9%) patients were treated with DES and 169 (70.1%) patients with BMS. Between the DES and BMS groups age distribution and family history rates were significantly different. More patients in the BMS group underwent PCI for STEMI (p=0.04). At 1-year follow up, there were less MACE rates in the DES group (p=0.011). There were no difference between two groups at 1 year mortality analysis. Three-year cumulative mortality rates were higher in the BMS group (p=0.043).

Conclusions: In our study DES and BMS usage in large coronary artery patients were compared, DES usage has been proved superiority with less MACE rates at 1-year follow up and safety with longterm lower mortality.

Interventional cardiology / Coronary

PP-125

A challenging case: Acute left main coronary artery occlusion presented with anterior myocardial infarction

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Background and Aim: Acute left main coronary artery (LMCA) occlusion is a rare and life-threatening clinical condition. Although it has usually high mortality rate, due to the recent developments in interventional strategies, percutaneous coronary intervention therapies would be a lifesaving approach in this patient group. Here we present a case of acute LMCA occlusion presented with acute anterior myocardial infarction with hemodynamic instability and treated with complex interventional therapies.

Methods: A 54-year-old male patient was admitted to our emergency department with new onset chest pain radiating both shoulder and arm. On his physical examination, pulse rate was 100 beats per minute, respiratory rate 31 per minute, blood pressure 70/40 and SO₂ 89%. There were crackles in lung fields up to mid zones bilaterally. On his baseline ECG, there was an extensive ST elevation including anterior and lateral derivation with reciprocal ST depression in inferior derivation. The patient was diagnosed with anterior MI and transferred to catheter laboratory. Subsequently, the patient underwent coronary angiography via femoral access using Judkins technique. During coronary angiography, we detected an extremely rare situation that left main coronary artery (LMCA) occluded in distal edge without distal coronary flow.

Results: We immediately decided percutaneous coronary intervention due to patient's hemodynamic instability. The lesion passed with 0.014 inch floppy guidewire, both LAD and CX wired and then the lesion was pre-dilated with compliant balloon. After pre-dilatation, coronary angiography showed severe stenosis in the distal part of LMCA. Due to the lesions' characteristics we planned bifurcation technique. Using double kissing crush technique, LMCA, LAD and CX were stented and final angiography showed good result without residual stenosis. After left coronary angiography we performed right coronary angiography and demonstrated right coronary dominance. Angiography was finished and the patient transferred to coronary care unit and was discharged fourth day of admission without cardiac problem.

Conclusions: Coronary artery disease is the most common cause of mortality in developed countries and interventional therapies are widely used for diagnostic and therapeutic purposes. These therapies dramatically decreased mortality and morbidity rate of the coronary artery disease. Acute occlusion of LMCA is a challenging situation on coronary angiography and has a higher mortality rate. Therefore, early intervention and revascularization should be main target. We also think that, in these patients, right coronary artery dominance may be the most important mortality predictor during the coronary angiography like our patient.



Figure 1. Baseline ECG showed extensive ST elevation including anterior and lateral derivation with reciprocal ST depression in inferior derivation.

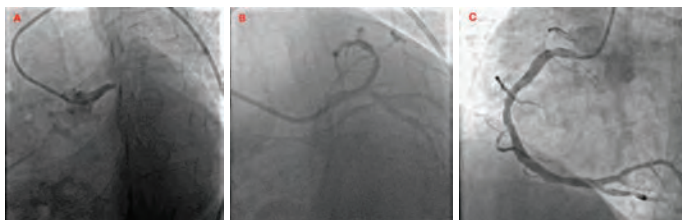


Figure 2. Total left main coronary artery (LMCA) occlusion (a) and after complex percutaneous intervention including LMCA, LAD and CX stent implantation, final angiography showed good result without residual stenosis (b) and right coronary dominance without severe coronary stenosis (c).

Interventional cardiology / Cover and structural heart diseases

PP-126

Single center experience in transcatheter aortic valve implantation for 7 years

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Background and Aim: Severe aortic stenosis (CAD) is the most common valvular heart disease in advanced age patients. Valvular surgery is a long-standing and well-known treatment option for CAD patients. The first successful transcatheter aortic valve implantation (TAVI) procedure was performed successfully by Cribber et al. in 2002. After that, it started to be widely applied all over the world.

Methods: Our study was designed retrospectively on patients who underwent TAVI procedure in Izmir Katip Çelebi University, Atatürk Training and Research Hospital between 2010-2017 October. Patients with severe and symptomatic AD were consulted in a council that includes cardiologist, cardiovascular surgeon, anesthesiologist. This council decided TAVI operation according to national and international guidelines as class 1 recommendations.

Results: We collected 66 patients underwent TAVI, between 2010-2017 October from hospital records. One-third of the patients were female and two-thirds were male. The mean age was 77.1±7.8 and hypertension was the most common comorbid disease with 82%. The mean baseline creatinine values were 0.94±0.43 mg/dL and the echocardiographic findings showing stenosis severity, baseline peak gradient was 79.9±22.4 mm Hg, basal mean gradient was 49.3±15.8 mm Hg, and annulus diameter was 23.5±5.2 mm, and the mean preoperative SPAB was 41.1±15.7 mm Hg. In our hospital, Edwards-Sapien XT was used as a balloon expandable valve in the first years and this corresponds to 30% of all patients. In the process, the use of self-expandable retractable valves increased as Core-valve 27%, Evolute-R 8%, Portico 35%. Mild paravalvular aortic insufficiency was observed in 23%, moderate paravalvular aortic insufficiency was observed in 8% and no paravalvular aortic insufficiency was not observed in 62% of patients. Early postoperative pacemaker requirement was 12%, late pacemaker requirement, known as after 6 months in the late phase, was 6%, contrast nephropathy was 20% of patients. The early death was observed in 12% of the patients, only one of these was caused of bleeding in intervention, the others were caused by pneumonia and acute renal failure. Late mortality was observed in 17% of the patients after 6 months of treatment and these ones were non-cardiac. The change in the degree of mitral regurgitation was not significant with p value: 0.324, but the sPAB value decreased significantly from 41.1±15.7 mm Hg to 33.7±11.2 mm Hg postoperatively with p value: 0.006.

Conclusions: In our clinic, this procedure is performed safely and with the complication rates stated in the literature. Procedures with lower complication rates is associated with patient's clinic and operator experience but there is always a risk of operation due to the nature of these patients due to severe comorbid conditions and advanced age. TAVI will continue to be a viable alternative for CAD in patients with moderate-to-high surgical risk, and perhaps will be the first independent choice for CAD in the future.

Table 1. General clinical features of TAVI treated patients

Age	77,1±7,8
Diabetes Mellitus	15 (%23)
Hypertension	54 (%82)
Coronary Arterial Disease	
Non-critical	32 (%48)
Operated CABG, medical	10 (%15)
Coronary Arterial Disease, PCI performed	10 (%15)
Severe Coronary Arterial Disease, medical	3 (%4)
Atrial Fibrillation	17 (%26)
Chronic Obstructive Pulmonary Disease	18 (%27)
Known Liver Disease	3 (%4)
Creatinin	0,94±0,43
Basal peak gradient	79,9±22,4
Basal average gradient	49,3±15,8
Annulus diameter	23,5±5,2

Table 2. GPpatients

Valve type	
Edwards Sapien	20(%30)
Core-valve	18 (%27)
Evolute-R	5(%8)
Portico	23 (%35)
Post-dilatation requirement	42 (%64)
Paravalvular aortic insufficiency	
No	41(%62)
Mild	15 (%23)
Moderate	5(%8)
Postprocedural early cardiac pacemaker requirement	8(%12)
Late phase cardiac pacemaker requirement	4(%6)
Postprocedural early death	8(%12)
Late phase death	11 (%17)
Contrast nephropathy	13 (%20)

Interventional cardiology / Coronary

PP-127

Hand dominance and coronary dominance: Is there a correlation

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Background and Aim: Coronary artery disease is present in anatomic risk factors such as dominance, ectasia, bifurcation angle, which are vascular geometric features of coronary arteries other than standard risk factors. The association of cerebral lateralization and hand dominance with some diseases has been described previously. The aim of this study is to investigate the relationship between hand dominance and coronary dominance determined by genetic factors.

Methods: Coronary angiograms of 226 patients who underwent coronary angiography between January 2017 and December 2017 were evaluated for coronary dominance. Patients were grouped as right, left and balanced dominance. At the same time, the "Oldfield Survey" was used to determine patients' hand preferences. Patients were divided into three groups as follows: left-hand, right-hand dominant and ambidextrous patients. The relationship between coronary dominance and hand dominance was investigated.

Results: Of the 226 patients who underwent angiography, 96 (42.5%) were female and 130 (57.5%) were male. 74.3% of the patients were hypertensive and 26.5% were diabetic. 170 patients (75.2%) had right hand, 14 patients had left hand (6.2%), and 42 patients (18.6%) had both hand dominance. Right coronary artery dominance was found in 160 patients (70.8%), left coronary dominance in 26 patients (11.5%) and balanced coronary dominance in 40 patients (17.7%). There is a statistically significant relationship between coronary artery dominance and cerebral functional dominance (p=0.00). There was no statistically significant relationship between hand dominance and SYNTAX score (p=0.945). There was no significant correlation between coronary dominance and SYNTAX score (p=0.408).

Conclusions: The coronary dominance right-left dominance rates and hand preference in the community are similar between right handedness and left handedness. Relations between anatomic lateralization and functional lateralization have been shown previously. Asymmetry between the thyroid lobes was also found to be related to hand preference. A statistically significant relationship was found between scoliotic convex pattern and dominant hand in adolocyte idiopathic scoliosis. In a cadaver study, there was a significant relationship between left coronary dominance and posterior cerebral circulation anomalies. In some parts of the Turkish society, social pressure is being applied to changing the hand preference of left-handed children and this individual then develops as both hands dominance. In our study, the proportion of left hand dominance is lower than that of other communities. In conclusion, there is a significant relationship between coronary artery dominance and cerebral functional dominance. However, cadaver studies are needed to compare cerebral and coronary dominance with advanced studies and anatomical lateralization with the wada test, which is an invasive test that shows us more clearly the dominant hemisphere.

Interventional cardiology / Carotid and peripheral vascular

PP-128

Is conventional method (palpation) or ultrasound guidance intervention the most appropriate method for common femoral artery cannulation in patients aged above 75 years?

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Background and Aim: Coronary and peripheral artery diseases are common among people aged >75 years. Angiography is the gold standard in the diagnosis and treatment of these diseases, and the femoral approach is usually preferred. Here we aimed to determine which intervention technique minimizes complications of the femoral approach.

Methods: Overall, 309 patients aged >75 years were included. They were divided into group 1 (ultrasound-guided intervention group) and group 2 (palpation-guided intervention group) and were compared in terms of clinical and demographic characteristics, laboratory findings, surgical complications, average intervention time, success rate at initial intervention, and accidental venous intervention rate.

Results: Clinical and demographic characteristics and laboratory findings were similar between the groups. Average intervention time (26 (21-25) vs 39 (25-61)min; p<0.001), average number of intervention attempts (1.10±0.35 vs. 1.58±1.05; p=0.012), average accidental venous intervention rate (3 (1.9%) vs. 16 (10%); p<0.001), and average visual analog scale score for pain (2 (1-5) vs. 7 (3-9); p<0.001) were significantly lower in group 1. Success rate at initial intervention was higher in group 1 [140 (89%) vs. 105 (69%); p<0.001]. Frequency of hematoma, early hematoma, and arteriovenous fistula were lower in group 1. There was no statistically significant difference between the groups in terms of major bleeding and pseudoaneurysm.

Conclusions: Ultrasound-guided femoral artery intervention requires less time and provides higher cannulation success rate at the initial attempt, with lesser pain and lower complication rates.

Interventional cardiology / Carotid and peripheral vascular

PP-129

Whole blood viscosity predicts all-cause mortality in patients with carotid artery disease

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Background and Aim: Carotid artery disease constitutes a major risk factor for stroke and mortality therefore prognostification and proper management is essential. Whole blood viscosity (WBV) plays a pivotal role in broad spectrum of cardiovascular diseases such as atherosclerosis. Beyond specific measurement techniques, WBV can be estimated from routine bloodwork parameters, namely hematocrit and serum total plasma proteins. Current literature demonstrated elevated WBV is a well-known risk factor for carotid atherosclerosis, however, prognostic role of WBV in carotid artery disease is lacking. In our study, we aim to demonstrate the role of estimated WBV for predicting all-cause mortality in patients who were referred to our clinic for invasive carotid angiography.

Methods: 146 patients with moderate to high extracranial internal carotid artery stenosis on doppler study who were referred for conventional selective carotid angiography were included in the study. Carotid stenosis were graded by the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria. Patients' risk factors, and clinical data were recorded. WBV at high shear rate (208 second⁻¹) was calculated with hematocrit (HCT) and plasma total protein (TP) by DeSimone's formula (WBV=(0.12xHCT)+0.17x(TP - 2.07)).

Results: Among 146 patients, 40 patients (27.4%) had symptomatic carotid stenosis (having stroke or transient ischemic attack) at admission. During median follow-up of 16 months (range: 0-65 months), 50 patients (34.2%), underwent carotid endarterectomy and 69 patients (47.3%) had non-carotid cardiac surgery. All-cause mortality was encountered in 15 patients (10.3%). NASCET grades were not different between survivors and non-survivors. Non-survivors were approximately 5 years older (73.7±8.4 vs. 68.7±8.5 years, p=0.046) and had one unit lower hemoglobin values (12.6±1.7 vs. 13.7±1.7 g/dL, p=0.021) than survivors. Non-survivors had one centipouse (cP) lower WBV than survivors (15.3±1.4 vs. 16.4±1.1 cP, p=0.002). In multivariate analysis, only WBV predicted all-cause mortality in patients with carotid artery stenosis. 1 cP increase in WBV was associated with 40% decrease in mortality (Hazard ratio (HR): 0.598, 95% confidence interval (CI) 0.393-0.908, p=0.016). In ROC analysis, a cut-off value of 15.9 cP for WBV discriminated non-survivors from non-survivors with a 71.4% sensitivity and 74.2% specificity (Area under the curve: 0.738, 95% CI: 0.587-0.888, p=0.004). WBV ≥15.9 cP was associated with 79% decreased mortality (HR:0.213, 95 CI: 0.071-0.636, p=0.006). Kaplan-Meier survival curves demonstrated 30% decreased survival in WBV <15.9 cP (63.8% vs. 94.6%, approximately 12 months survival benefit, log-rank p=0.002).

Conclusions: In our study, lower WBV was associated with increased all-cause mortality. As opposed to the context of higher WBV as a risk factor for carotid atherosclerosis, lower WBV may be a poor prognostic factor for patients with carotid artery disease.

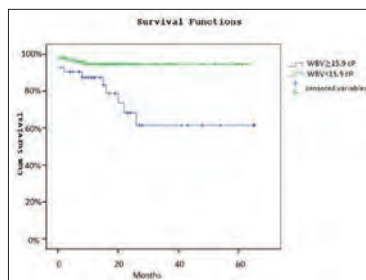


Figure 1. Kaplan-Meier analysis of WBV groups for all-cause mortality.

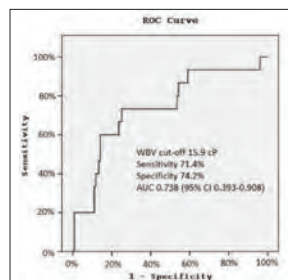


Figure 2. ROC analysis of WBV for all-cause mortality.

Interventional cardiology / Coronary

PP-130

Long term outcomes of hybrid percutaneous intervention with bioresorbable vascular scaffolds and drug eluting stents for complex coronary artery lesions

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Background and Aim: Bioresorbable vascular scaffolds (BVS) are considered as a new revolution in coronary intervention due to their potential advantages for long term follow up. However, current generation BVS have also some drawbacks that restrict the use for complex lesions. Using BVS and drug eluting stents (DES) together -implanting DES for BVS inappropriate segments -may be a feasible option to avoid the disadvantages of permanent foreign body and to reduce very late adverse events. In this context, we investigated the clinical outcomes following treatment with hybrid strategy with concomitant use of BVS and DES for complex lesions.

Methods: A single center retrospective cohort was performed enrolling 40 patients with complex lesions treated with hybrid approach from February 2015 up to April 2017. Lesion segments with a large plaque burden and/or severe calcification, aorto-ostial and bifurcation lesions that may be unfavorable for BVS, treated with DES. BVS and DES were implanted with minimal overlap of DES and BVS struts. The primary end-point was target lesion failure (TLF) which was a composite of cardiac death, target vessel myocardial infarction and target lesion revascularization (TLR). During follow-up, coronary angiography was performed when patients had ischemic symptoms.

Results: Mean age was 58 years, 87.5% were males, 30% diabetics. 92.5% of lesions were ACC/AHA type B2/C. Predilatation and postdilatation with non-compliant balloons were routinely performed for all lesions treated with BVS (62.5% novolimus, 37.5% everolimus). BVS length was 26.1 mm, maximum post-dilatation inflation pressure was 18 (18-20.5) atm. The median follow up period was 634.5 days. During follow up, 27.5% underwent coronary angiography. TLF was seen in 7.5%, this was consist of TLR. Cardiac death and target vessel myocardial infarction weren't observed within follow-up period. TLR occurred in proximal edges of DES. BVS and DES treated segments were patent.

Conclusions: The result of this study demonstrated that concomitant use of DES and BVS for treating complex lesions is a feasible and safe strategy with a 7.5% of TLF rate, when selected correct lesion and implemented standardized implantation technique for BVS.

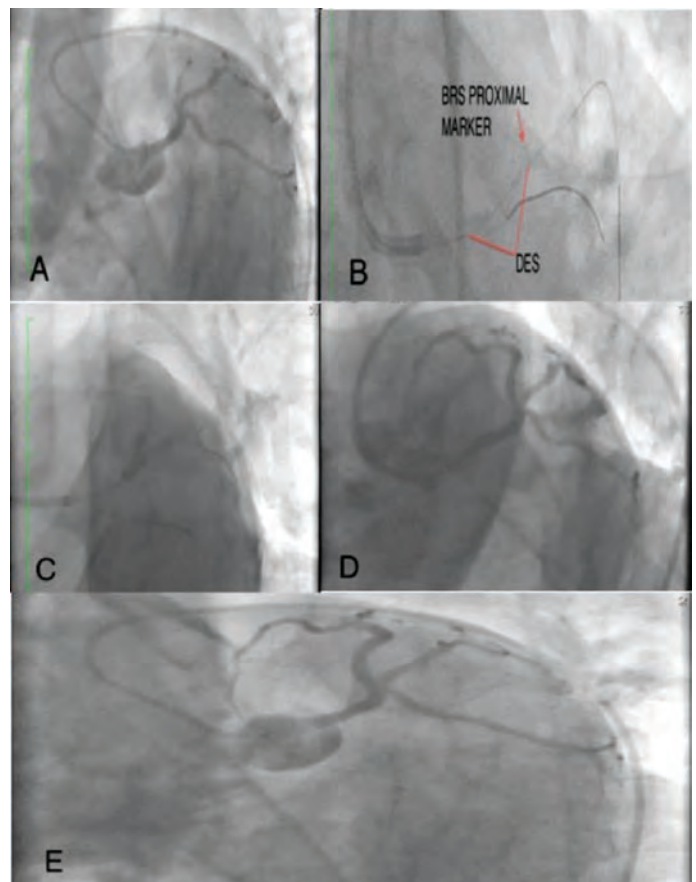


Figure 1. A case of hybrid drug eluting stent and bioresorbable vascular scaffold implantation. (a) Baseline angiogram showing LAD chronic total occlusion lesion. (b) Post PCI of LAD lesion using DES (3.5x15 mm, Xience Pro, Abbott) and BVS (3.0x28 mm, DESolve, Elixir). (c) Final kissing balloon inflation after side branch (high obtuse marginal artery) DES (2.75x18 mm, Xience Pro, Abbott) implantation with T stenting and small protrusion technique (TAP). (d) Final angiogram showing an excellent result. E) 24 month follow up angiogram demonstrating preserved patency of the stents. BRS: Bioresorbable scaffold.

Interventional cardiology / Carotid and peripheral vascular

PP-131

Determination of femoral calcium image subtraction using multiscale binary patterns: Preliminary results

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Background and Aim: Pre-processing algorithms based on Multiscale binary pattern (MBP) have proved to be effective for accurate calcification in various fields such as optical character recognition, calcium recognition and medical image analysis. The reader confidence and diagnostic accuracy of femoral angiography can be compromised by the presence of calcified plaques and stents causing blooming artifacts. Compared to conventional invasive femoral angiography (FAA), this may cause an overestimation of stenosis severity leading to false-positive results. In this study, we tested the feasibility of a new femoral calcium image subtraction algorithm in relation to reader confidence and diagnostic accuracy.

Methods: The records of patients referred for diagnostic femoral angiography between November 2016 and july 2018 at Cardiology Center were reviewed retrospectively. Selected for further review were those patients who had undergone a routine, contrast peripheral CT study at our hospital. The CT scans and conventional angiograms of 25 patients (50 vessels) were reviewed independently by two investigators who were blinded to the results of the angiogram when reviewing the CT scans and to the results of the CT scans when reviewing the angiogram. Differences in readings were resolved by consensus of two readers on joint review. Conventional angiography was performed using selective abdominal artery injections of omnipaque 300. Digital subtraction angiography was performed with AP projections of the left left iliac and femoral circulation. The degree of stenosis was measured with precision calipers by dividing the minimum femoral diameter by the diameter of the normal vessel distal to the stenosis. Angiographic findings in the femoral artery were characterized as normal, as showing minimal irregularity of the vessel wall, as showing less than 50% diameter stenosis, or as showing greater than 50% diameter stenosis (Figure 1, 2). Correlation between MBP and angiography was then performed.

Results: Patients ranged in age from 58 to 84 years (mean age, 65 years). Correlation of MBP calcification with angiographic findings were presented table 1. Patient with FAA (no stenosis) and MBP (<50% stenosis) is presented in Figure 1. Patient with FAA and MBP (>50% stenosis) is presented in Figure 2. Baseline characteristics of study patients is show in Table 2.

Conclusions: Our experience with femoral calcium image Multiscale binary pattern suggests that it is feasible and could lead to an improvement in reader confidence and diagnostic accuracy for identification of significant femoral artery disease.

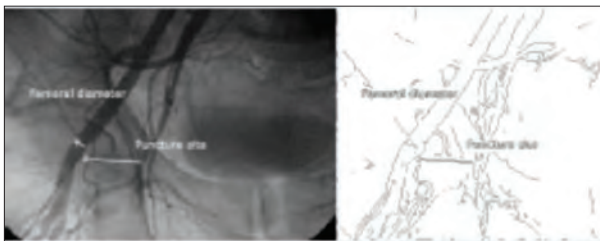


Figure 1. Patient with FCS (0% < stenosis < 50%) and MBP (no stenosis).

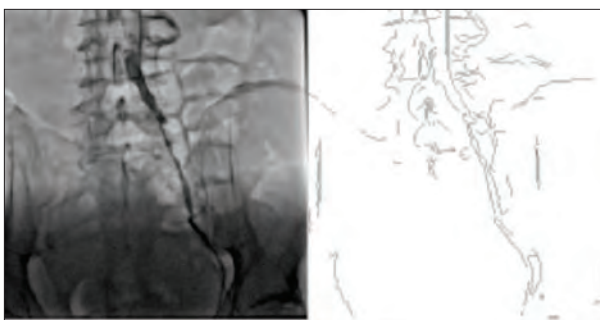


Figure 2. Patient with FCS (>50% stenosis) and MBP (>50% stenosis).

Table 1. Distribution of patients according to MBP findings and CCS

MBP Total(N=3576)	FCS	No of Patients (%)
No of Patients (%)	0	1080 (30.2%)
	>0	195 (5.2 %)
0 % <Stenosis< 50 %	0	1275 (34.6 %)
	>0	212 (6.1 %)
>50 % Stenosis		1063 (29.2 %)
	0	794 (25.2 %)
	>0	86 (2.4 %)

MBP, Multiscale Binary Patern FCS, Femoral Calcium Score

Table 2. Baseline characteristics of study patients

Variables	0% < Stenosis < 50 %	>50 % Stenosis	p value
Age (years)	64±11	68±12	<0.01
Gender (male)	163(65%)	1483 (68%)	<0.01
Hypertension	136(54%)	1647 (%74)	<0.01
Diabetes	62(25%)	779(35%)	<0.01
Current Smoking	42(17%)	536(24%)	<0.01
Dyslipidemia	150 (60%)	1421(65%)	<0.01
Total Cholesterol	242±41	244±43	<0.01
LDL Cholesterol	161±34	164±36	<0.01
HDL Cholesterol	47±16	45±15	<0.01

Interventional cardiology / Coronary

PP-132

Relationship between Tp-E, Tp-E/QT and mortality /morbidity in patients presenting with acute anterior myocardial infarction

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Background and Aim: Cardiovascular (CV) mortality is still leading cause of death in the world despite its decreasing rate in the last years. QT, QTc and QT dispersion are commonly used electrocardiographic measurements as an indicator of increased arrhythmic risk after myocardial infarction (MI). Beside these ones, new parameters like Tpeak-Tend (Tp-e) and Tpeak-T-end/QT ratio have been started to use and Tp-e and mortality relationship was shown. In this trial, Tp-e, Tp-e/QT and major adverse cardiac events (MACE) relationship was investigated in patients with acute anterior MI who were primary percutaneous coronary intervention (PCI) performed.

Methods: Three hundred ninety four patients with acute anterior MI who admitted Suleyman Demirel University Hospital Cardiology Department between 2012-2016 years were included in this retrospective trial. After excluding 248 patients according to exclusion criterias, 146 patients were registered to trial. Preprocedure, post procedure and 24th hour electrocardiographies (ECG) of all patients were analysed. Scanned ECGs were transferred to computer and Tp-e and QT intervals of chest derivations were measured via Bitrule programme. QTc was calculated by Bazett formula and parameter ratios with calculator. Hospital registries were investigated and first month and first year MACE were recorded by calling patients or patients' relatives.

Results: Mortality in 9 patients and morbidity in 8 patients were seen within 146 patients. Diabetes Mellitus (DM), hypertension (HT) and female gender were associated with MACE. Only antiarrhythmic drug use was found associated with MACE in terms of medical treatment type. Preprocedural, post procedural and 24th hour calculated Tp-e, Tp-e/QT ratio and Tp-e change in ECGs were not statistically associated with MACE.

Conclusions: This trial showed that Tp-e, Tp-e/QT values were not associated with MACE in patients presenting with ST elevation MI and being performed primary PCI. MACE was statistically higher in females and patients with DM and HT.

Table 1. Mean and standard deviations of ECG parameters of the patient group

	Mean±sd
Tpe1	79,46 ± 15,47
Tpe2	71,68 ± 14,26
Tpe3	70,84 ± 17,77
Tpe/ QT 1	0,22 ± 0,04
Tpe/ QT 2	0,19 ± 0,04
Tpe/ QT 3	0,19 ± 0,03

Tp-e1, Tp-e / QT1 pre-treatment values, Tpe2, Tp-e / QT2 post-treatment values, Tpe3, Tp-e / QT3 24. hour values

Table 2. Mean and standard deviations of ECG parameters in patients with morbidity

FEATURES (Mean±sd)	MORBIDITY		P
	Yes(n=9)	No(n=129)	
Tpe 1	78,11±17,89	79,41±15,62	0,796
Tpe 2	66,33±12,60	72,02±14,54	0,360
Tpe 3	67,88±16,32	71,10±17,79	0,595
Tpe/QT1	0,21±0,05	0,22±0,04	0,877
Tpe/QT2	0,18±0,04	0,19±0,04	0,193
Tpe/QT3	0,18±0,03	0,19±0,03	0,894

Tp-e1, Tp-e / QT1 pre-treatment values, Tpe2, Tp-e / QT2 post-treatment values, Tpe3, Tp-e/QT3 24. hour values

Table 3. Mean and standard deviations of ECG parameters in patients with developing mortality

FEATURES (Mean±sd)	MORTALITY		P
	No(n=138)	Yes(n=8)	
Tpe 1	79,32 ± 15,71	81,87 ± 10,85	0,389
Tpe 2	71,65 ± 14,45	72,25 ± 11,10	0,753
Tpe 3	70,89± 17,66	69,87 ± 20,83	0,648
Tpe/QT1	0,22± 0,04	0,23 ± 0,03	0,246
Tpe/QT2	0,19± 0,04	0,21 ± 0,03	0,389
Tpe/QT3	0,19± 0,03	0,19 ± 0,05	0,364

Tp-e1, Tp-e / QT1 pre-treatment values, Tpe2, Tp-e / QT2 post-treatment values, Tpe3, Tp-e/QT3 24. hour values

Table 4. Mean and standard deviations of ECG parameters in patients with MACE

	MACE	MACE	
FEATURES (Mean ±sd)	Yes (n=17)	No (n=129)	P
Tpe1	79,41± 15,62	79,88 ± 14,67	0,691
Tpe2	72,02 ± 14,54	69,11 ± 11,94	0,205
Tpe3	71,10 ± 17,79	68,82 ± 18,0	0,332
Tpe/Qt1	0,22 ± 0,04	0,22 ± 0,04	0,498
Tpe/Qt2	0,19 ± 0,04	0,19 ± 0,04	0,795
Tpe/Qt3	0,19 ± 0,03	0,19 ± 0,04	0,896

Tp-e1, Tp-e / QT1 pre-treatment values, Tp-e2, Tp-e / QT2 post-treatment values, Tp-e3, Tp-e/QT3 24. hour values

Interventional cardiology / Coronary

PP-133

A new and simple risk predictor of contrast induced nephropathy in patients undergoing primary percutaneous coronary intervention: TIMI Risk Index

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Background and Aim: The thrombolysis in Myocardial Infarction risk index (TRI) was developed to estimate prognosis at the initial contact of the healthcare provider in coronary artery disease patients without laboratory parameters. In this study we aimed to investigate the relationship of the baseline TRI and contrast-induced nephropathy (CIN) in patients with ST-elevation myocardial infarction (STEMI).

Methods: A total of 963 consecutive STEMI diagnosed patients who underwent primary percutaneous intervention were included in the study. TRI was calculated using formula of "Heart Rate X (age/10) / SBP" on admission. CIN was defined as an increase in serum creatinine concentration ≥25% 48 hours later over the baseline.

Results: Of a total of 963 patients, CIN was observed in 13.3% (n=128). TRI were significantly higher in the CIN (+) group compared with the CIN (-) group (32.9±18.8 vs 19.9±9.9, p<0.001). There was a stronger correlation between CIN and age, diastolic blood pressure, heart rate, Killip class, left ventricular ejection fraction, amount of contrast media and diabetes mellitus. Amount of contrast media (OR 1.010, 95% CI 1.007-1.012, p<0.001) and TRI (OR 1.047, 95% CI 1.020-1.075, p=0.01) were independent predictors of CIN. The best threshold TRI for predicting CIN was ≥25.8, with a 67.1% sensitivity and 80.4% specificity (area under the curve [AUC]: 0.740, 95% CI: 0.711-0.768, p<0.001).

Conclusions: TRI is an independently predictor of CIN and it may be used as a simple and reliable risk assessment of CIN in STEMI patients without the need for laboratory parameters.

Interventional cardiology / Cover and structural heart diseases

PP-134

Transcatheter aortic valve replacement in large annulus: Early results of single institution experience

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Background and Aim: New devices that accommodate into aortic ring have been invented, however outcomes of the interventions performed for large aortic rings are not completely clear-cut. The aim of this study is to investigate short-term outcomes of the patients with aortic stenosis having large aortic rings.

Methods: Forty-seven male (39.5%), and 72 (60.5%) female patients were enrolled in the study. Mean age of the patient population was 79±8 years. Twenty-one (17.6%) patients had a large aortic annulus, while EvolutR (n=8), and Sapien XT (n=13) valves were implanted in indicated number of patients. The patients were divided into two groups as those with large, or small aortic rings, and they were evaluated in terms of inpatient complications.

Results: Mean logistic Euroscore values of the patients with large and small annulus were calculated as 27.8±11.8, and 26.4±13.3, respectively p=0.408. Median operative time was 58 minutes. One patient with large, and five patients with small aortic rings required implantation of permanent pacemaker. (p=0.714). In patients with large aortic rings inpatient mortality was not detected, while five patients with small aortic rings exited (p=0.247). In both groups all the devices were implanted successfully (100%), two patients with small, and one patient with large annulus required implantation of a second valve because of valvular jump.

Conclusions: Transcatheter aortic valve implantation can be performed safely with similar success rates in critically ill advanced stage aortic stenosis patients with large aortic rings.

Interventional cardiology / Coronary

PP-135

Comparison of brachial flow mediated vasodilatation with subcutaneous nitrate injection for transradial artery access

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Background and Aim: The transradial artery access (TRA), which is considered to be more advantageous than the transfemoral artery access in many aspects, is now widely used in coronary interventions. It has been reported that subcutaneous nitrate injection (SNI) before radial artery puncture improved the success of TRA. The aim of this study is to compare the effects of brachial flow mediated vasodilatation (FMD) with SNI on the diameter of radial artery and on the success of transradial artery intervention.

Methods: A total of 90 cases were included in the study. Of these, 30 were injected with subcutaneous nitrate before TRA. Brachial flow mediated vasodilatation was performed in 30 patients and the remaining 30 patients were selected as the control group. In the control group, the diameter of the radial artery was measured in the normal state, after SNI and after FMD three times with doppler ultrasound. In patients who underwent coronary angiography the duration of radial artery puncture, numbers of radial artery puncture attempts and pain scales were recorded. In both the experimental and control groups, body mass index (BMI), age, smoking status, hypertension (HT), diabetes (DM) and hyperlipidemia (HL) were recorded.

Results: Significant differences were found between the radial artery diameter measurements (mm) in normal condition and after FMD and SNI in the control group patients. (basal; 2.49±.33, SGT; 2.57±.34 and FMD; 2.56±.35 p<0.05). The radial artery diameters of smoker and male cases were significantly higher than that of women in all of the measurements of Normal, SNI and FMD groups. Diameter values were not differentiated according to the age, BMI, presence of HT, DM and HL. There was no significant difference in pain scale, number of attempts and duration of the procedure between patients who underwent transradial intervention with SNI and FMD (p>.05). In the FMD group, the average of the women was significantly higher than the male patients in terms of the duration of the procedure and the level of pain. There was a 37% correlation between the age and the duration of the procedure, and the duration of the procedure was higher in the non-DM group. The level of pain was higher in patients who were smoking (p<.05).

Conclusions: While both SNI and FMD increase RA size before transradial intervention, there is no significant difference between the effects of SNI and FMD on TRA. Brachial flow mediated vasodilatation as well as SNI may be used to facilitate radial artery puncture in TRA.

Other

PP-136

The effect of thrombolytic therapy for intermediate-low and intermediate-high risk pulmonary embolism on in-hospital mortality

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Background and Aim: Pulmonary embolism (PE) is a cardiovascular disease associated with high rate of mortality and morbidity. While the thrombolytic therapy is the first choice in the treatment of massive PE, it is subject of debate in submassive PE. Our aim is to investigate patients who were diagnosed as PE before 2014 with intermediate risk that further classified into intermediate low or high and to examine the effects of thrombolytics on mortality. Patients' data and treatment modality were evaluated retrospectively.

Methods: The study included 227 patients who are admitted to our clinic between January 2009 and December 2013 and diagnosed as PE. Mortality risk stratification was done according to 2014 European Society of Cardiology guidelines on acute PE. The primary outcome was in-hospital death related to PE. Composite endpoint consisted of long-length-of-stay, in-hospital need of inotrope, in-hospital cardiogenic shock and in-hospital death related to hemorrhage.

Results: 79 (65.8%) of 120 patients in intermediate-low risk group and 78 (72.8%) of 107 patients in intermediate-high risk group received thrombolytic therapy. Correlation was not observed between thrombolytic therapy and mortality in intermediate-low risk PE (p>0.05). While the mortality rate of intermediate-high risk patients treated with thrombolytics was 5.1% (n=4), it was 24.1% (n=7) in non-thrombolytic group with significant difference between two groups (p<0.008). Correlation was not observed between thrombolytic therapy and composite endpoint in both groups.

Conclusions: In spite of there is no study exclusively relevant to mortality risk stratification of PE, the fact that the included patients in studies in which thrombolytic therapy for submassive PE was evaluated, had right ventricular (RV) insufficiency and/or dilatation and were cardiac-marker positive and also these patients' mortality rate was greater than the patients with normal RV function; made further classification of intermediate risk PE necessary. Thrombolytic therapy in submassive PE that includes hemodynamically stable patients with RV dysfunction, is still a matter of debate. Our study shows that thrombolytic therapy is not necessary in intermediate-low risk PE. However in-hospital mortality rate in intermediate-high risk PE is lower in patients given thrombolytic therapy. In addition to prove the benefit of thrombolytic therapy in intermediate-high risk PE, our study also shows that reclassification of intermediate risk PE is concordant with real life data.

Table 1. Patients' age, hemodynamic features and echocardiographic measurements and their distribution according to mortality risk and thrombolytic therapy status

		intermediate-low (n=120)		p (*p<0,05)	intermediate-high (n=107)		p (*p<0,05)
		tPA (-) (n=41)	tPA (+) (n=79)		tPA (-) (n=29)	tPA (+) (n=78)	
age	mean±Sd	65,71±17,4	60,85±15,65	0,123	70,14±15,16	65,37±15,14	0,151
heart rate	mean±Sd	98,05±20,48	107,06±17,08	0,012*	101,72±16,08	108,54±17,63	0,072
respiratory rate	mean±Sd	20,24±3,74	21,13±3,45	0,199	21,17±4,18	22,18±4,14	0,267
oxygen saturation	mean±Sd	94,17±2,59	92,32±5,77	0,049*	92,23±5,57	91,34±5,91	0,483
RV-diameter	mean±Sd	3,47±0,38	3,33±0,54	0,194	4,35±0,54	4,19±0,76	0,356
TAPSE	mean±Sd	18,88±2,9	17,26±3,66	0,036*	15,3±3,6	14,53±2,97	0,325
TPAP	mean±Sd	48,47±8,52	51,38±11,49	0,174	55,88±19,1	58,56±14,12	0,524

Table 2. Comparison of primary and composite endpoints according to thrombolytic therapy status in each mortality risk group

		intermediate low (n=120)		p	intermediate high (n=107)		p (**p<0,01)
		TPA (-) (n=41)	TPA (+) (n=79)		TPA (-) (n=29)	TPA (+) (n=78)	
mortality	n (%)	0 (0)	1 (1,3)	1,000	7 (24,1)	4 (5,1)	0,008**
in-hospital mortality related to hemorrhage	n (%)	0 (0)	0 (0)	-	0 (0)	1 (1,3)	1,000
in-hospital cardiogenic shock	n (%)	0 (0)	1 (1,3)	1,000	5 (17,2)	6 (7,7)	0,164
in-hospital need of inotrope	n (%)	1 (2,4)	1 (1,3)	1,000	5 (17,2)	7 (9)	0,300
long-length-of-stay	n (%)	23 (56,1)	53 (67,1)	0,236	14 (48,3)	48 (61,5)	0,217
composite end-point	n (%)	24 (58,5)	54 (69,2)	0,243	20 (69)	52 (67,5)	0,888

Other

PP-137

Correlation between red cell distribution width and peripheral vascular disease severity and complexity

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Background and Aim: A traditional hematological marker, red cell distribution width (RDW), is accepted as a novel marker of atherosclerotic vascular diseases. Correlation between RDW and carotid intima-media thickness was well-demonstrated. Elevated RDW levels was associated with future risk of stroke. Moreover, elevated RDW indicate presence and severity of atherosclerotic coronary artery disease. However, clinical importance of the RDW as a prognostic biomarker in the peripheral vascular disease (PVD) reported with a few report. Herein, we aimed to show the correlation between RDW and PVD severity and complexity in term of angiographic evaluation.

Methods: A total of subsequent 117 subsequent patients who underwent peripheral lower extremity angiography were evaluated retrospectively. Upon admission, RDW level was measured with automated complete blood count. Severity and complexity of the PVD was evaluated according to TASC-2 classification. TASC-2 A-B lesion was defined as simple PVD and TASC-2 C-D lesion was defined as pravelent and complex PVD. Then, both groups compared statistically according to clinical, laboratory and demographic features including RDW levels.

Results: The mean age was 58.5±11 with male predominance. On the 49.6% of the patients, TASC-2 C-D lesions were observed. Advanced age, male gender, body mass index were associated with TASC-2 groups. RDW levels was correlated with presence of PVD as well as TASC-2 grades (p=0.05). The upper quartiles (75th percentile) of the RDW levels was 14.1 and patients with RDW level ≥14.1 has more significant correlation with presence and severity of PVD (p=0.001). In the multivariate regression analysis, elevated RDW was found as independent predictor of presence of PVD and also TASC-2 C-D lesions (OR:2.26, with a 95% CI 0.051-0.774; p=0.02).

Conclusions: Red cell distribution width is cheap and widely reachable prognostic biomarker in the atherosclerotic vascular disease. Similar to atherosclerotic coronary disease, elevated RDW levels was associated with TASC-2 C-D lesions which indicated more prevalent and complex PVD.

Table 1. Distribution of clinical and demographic characteristics of the patients according to presence and severity of peripheral vascular disease

VARIABLES	NORMAL (n:33)	TASC-2 A-B (n:26)	TASC-2 C-D (n:58)	p
Age (years)	53,5±10,8	58,5±12,4	61,5±9,5	0,003
Sex (male)	54,5%	80,8%	91,4%	0,001
Diabetes mellitus	31,3%	28,0%	31,0%	0,95
Hypertension	48,5%	26,9%	29,3%	0,12
Dyslipidemia	34,4%	23,1%	24,1%	0,51
Current smoking	43,8%	38,5%	29,3%	0,36
Creatinine (mg/dl)	0,89±0,18	0,86±0,30	1,15±0,95	0,11
BMI (kg/m2)	25,9±2,4	28,1±4,7	28,5±4,3	0,009
RDW (%)	12,9±0,8	13,7±1,1	14,0±1,4	0,001
4th RDW quartile (≥14.1 %)	2,9%	34,6%	34,5%	0,002

Other

PP-138

The relationship between coronary arterial dominance and the QRS axis

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Background and Aim: Coronary artery dominance influences the amount and anatomic location of myocardium that is perfused by the left or right coronary circulation. However, the association between coronary arterial dominance and the QRS axis on 12-lead surface electrocardiography (ECG) was not investigated. The present study aims to evaluate the relationship between coronary arterial dominance and the QRS axis on ECG in patients without significant coronary artery and structural cardiac disease.

Methods: Overall, 133 patients, without significant CAD and who met the inclusion criteria, participated in this study. A standard surface 12-lead ECGs were performed in all study patients. QT interval, QTc interval, QRS duration, PR interval, P wave and QRS axis were determined. Based on the origin of the posterior descending coronary artery, coronary circulation was categorised into left, right, and balanced coronary dominance.

Results: There were 133 subjects with 56 right dominant (42%), 39 left dominant (29%) and 38 codominant (29%) pattern. QRS axis value was found to be significantly higher in the left dominant group when compared with the codominant and right dominant group (p<0.05). No significant difference was observed between the codominant and right dominant groups regarding QRS axis values. The axis of (+30)-(+90) ratio in the left dominant group was found to be significantly higher when compared with the codominant and right dominant group (p<0.05). No significant difference was observed between the codominant and right dominant groups regarding the axis of (+30)-(+90) ratio.

Conclusions: Our findings suggested that the QRS axis may be related to coronary artery dominance.

Table 1. Baseline characteristics of study patients

	Min-Max	Median	Mean.s.d./n-%
Age,(years)	23.0 - 76.0	55.0	52.1 ± 12.0
Sex	Female		69 51.9%
	Male		64 48.1%
Weight, (kg)	50.0 - 110.0	64.0	67.3 ± 12.4
Height,(m)	1.5 - 1.9	1.6	1.6 ± 0.1
BMI	20.3 - 29.8	25.2	25.7 ± 3.0
HT	No		68 51.1%
	Yes		65 48.9%
DM	No		100 75.2%
	Yes		33 24.8%
Smoking	No		97 72.9%
	Yes		36 27.1%
P axis	-30.0 - 132.0	47.0	47.6 ± 22.9
QRS axis	-25.0 - 85.0	22.0	28.2 ± 28.3
T axis	-40.0 - 90.0	35.0	36.1 ± 25.6
PR, (msn)	112.0 - 192.0	148.0	148.4 ± 13.5
QT,(msn)	350.0 - 450.0	390.0	396.3 ± 24.9
QRS axis	(-30) - (+30)		76 57.1%
	(+30) - (+90)		57 42.9%

BMI; body mass index, DM; diabetes mellitus, HT; hypertension, s.d; standard deviation

Table 2. Baseline demographics of the study population by coronary dominance

	Co-dominant (n:38)		Right dominant (n:56)		Left dominant (n:39)		p
	Mean, s.d./n-%	Median	Mean, s.d./n-%	Median	Mean, s.d./n-%	Median	
Age	52.1 ± 12.7	55.0	54.0 ± 9.9	55.5	49.4 ± 13.8	52.0	0.358 *
Sex	Female	20 52.6%	27 48.2%	22 56.4%			
	Male	18 47.4%	29 51.8%	17 43.6%			
Weight, kg	65.5 ± 12.6	63.3	67.2 ± 11.9	64.8	69.0 ± 12.9	64.0	0.366 *
Height, m	1.6 ± 0.1	1.6	1.6 ± 0.1	1.6	1.6 ± 0.1	1.6	0.347 *
BMI	25.5 ± 2.9	25.4	25.6 ± 3.2	25.1	26.1 ± 3.0	25.4	0.675 *
HT	No	22 57.9%	27 48.2%	19 48.7%			
	Yes	16 42.1%	29 51.8%	20 51.3%			
DM	No	30 78.9%	41 73.2%	29 74.4%			
	Yes	8 21.1%	15 26.8%	10 25.6%			
Smoking	No	26 68.4%	42 75.0%	29 74.4%			
	Yes	12 31.6%	14 25.0%	10 25.6%			

BMI; body mass index, DM; diabetes mellitus, HT; hypertension, kg; kilogram, m; meter, s.d; standard deviation, *, Kruskal-wallis (Mann-whitney u test), **, Chi-square test

Table 3. Baseline electrocardiographic findings and QRS axis of the study population by coronary dominance

	Co-dominant (n:38)		Right dominant (56)		Left dominant (n:39)		p
	Mean, s.d./n-%	Median	Mean, s.d./n-%	Median	Mean, s.d./n-%	Median	
P axis	42.4 ± 20.4	40.0	50.1 ± 26.3	50.0	48.9 ± 19.6	47.0	0.133 *
QRS axis	22.2 ± 27.2	15.5	24.9 ± 27.4	21.0	38.6 ± 28.6	41.0	0.019 *
T axis	37.0 ± 20.7	41.0	34.8 ± 26.4	34.0	37.0 ± 29.0	36.0	0.587 *
PR, msn	148.9 ± 13.0	149.0	148.3 ± 14.7	148.0	148.0 ± 12.6	148.0	0.943 *
QT, msn	398.9 ± 25.7	398.0	390.7 ± 23.2	390.0	401.8 ± 25.4	400.0	0.062 *
QRS axis	(-30)-(-90)	26 68.4%	35 62.5%	15 38.5%			
	(30)-(90)	12 31.6%	21 37.5%	24 61.5%			

*, Kruskal-wallis (Mann-whitney u test), s.d; standard deviation, **, Chi-square test

Other

PP-139

The affect of comorbid conditions to pulmonary embolism severity

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Background and Aim: Pulmonary embolism (PE) is a complex disease process with high rates of morbidity and mortality. Patients with PE usually have several comorbidities such as cancer, operation, stroke and etc. Charlson co-morbidity index (CCI) is a measure of co-morbidity burden providing a means of quantifying the prognostic impact of 22 co-morbid conditions on the basis of their number and prognostic impact. In this study we aimed to investigate the affect of comorbid conditions to vital signs at the time of application and pulmonary embolism severity index (PESI) in patients with PE.

Methods: Eighty-one patients who admitted to the emergency service with the signs of PE were included to the study. A detailed medical history, physical examination, situations predisposing to PE, PESI score, CCI score were recorded (Table 1). PE were diagnosed according to computed tomography angiography. CCI score was used for detecting the comorbid conditions. Biochemical and haematological parameters, high sensitive troponin, D-dimer levels were measured at the admission. Transthoracic echocardiography was also performed. Patients were divided into two group according to existence of shock and hypotension. [group 1-shock or hypotension (+); 22 pts, 10F, mean age 67.6±15.2 years; group 2-shock or hypotension (-); 59 pts, 38 F, mean age 60.8±18.0 years]. Patients also divided into three groups according to CCI scores (I-Low score; 1-2, II-medium score; 3-4, III-high score; >5).

Results: Demographic characteristics and situations predisposing to PE were similar between the groups. PESI score, systolic and diastolic blood pressures were significantly higher in group 1 compared to group 2 (128.5±29.1 vs 88.0±30.5, p<0.001; 83.5±8.4 vs 130.9±18.6 mm Hg, p<0.001; 54.3±11.7 vs 77.3±11.3 mm Hg, p<0.001 respectively). CCI score were also significantly higher in group 1 compared to group 2 (7.8±2.1 vs 6.1±3.1, p=0.005). CCI score was significantly correlated with age and PESI score (r=0.77, p<0.001). There were weak correlations between CCI and high sensitive troponin (r=0.23, p=0.05).

Conclusions: Patients with pulmonary embolism with shock or hypotension have higher CCI scores compared to patients with no hypotension. Higher CCI scores are associated with higher ages, PESI scores and cardiac injury markers. CCI scores might be used like a PESI score in risk stratification in patients with PE.

Table 1. Characteristic of the study population

Age (years)	62.7±17.5
Gender (F,n)	48
PESI score (n)	98.5±35.4
CCI score (n)	6.5±3.0
Hypertension (n)	47
Diabetes mellitus (n)	27
Coronary artery disease (n)	11
Smoking (n)	26
Deep venous thrombosis (n)	23
Pregnancy (n)	1
History of operation (n)	23
Immobility (n)	32
History of stroke (n)	6
History of cancer (n)	21

Other

PP-140

Cardiac effects of hyperbaric oxygen therapy: Is it safe?

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Background and Aim: Hyperbaric oxygen treatment involves intermittent inhalation of 100% oxygen under 2.5 absolute atmospheric pressure and is used to treat various clinical conditions, such as wound healing and carbon monoxide intoxication. However, the possible cardiac side effects related to prolonged and repetitive exposure to increased atmospheric pressures are controversial. Therefore, in this study, we investigated cardiac effects of hyperbaric oxygen therapy.

Methods: Twenty-five patients who had indications for hyperbaric oxygen therapy for various reasons were enrolled in this study prospectively. Blood samples were taken from each patient to measure serum high sensitive troponin T and B-type natriuretic peptide levels before starting hyperbaric oxygen therapy and after the 10th session. Also, each patient underwent echocardiographic examination both at the beginning and at the end of the study.

Results: Serum B-type natriuretic peptide levels were significantly higher after the 10th session (median: 77 pg/ml, IQR 75-25: 386-21 vs. 129 pg/ml, IQR 75-25: 784-51) p<0.01). However, there was no significant change in serum high sensitive troponin T values (median: 0.008 ng/ml, IQR 75-25: 0.016-0.005 vs 0.010 ng/ml IQR 75-25: 0.021-0.006 respectively, p=0.74). In addition, there was no significant change with respect to left ventricular ejection fraction, cardiac volumes and pulmonary artery pressures.

Conclusions: Hyperbaric oxygen therapy seems to be safe with respect to myocardial injury. On the other hand, consecutive sessions increase serum B-type natriuretic peptide levels which indicate higher filling pressure and volume overload. Therefore, patients with left ventricular dysfunction may have a tendency for decompensation during this treatment.

Table 1. Clinical characteristics of the study patients

	Patients n=25
Age	58±16
Sex(male)	16 (64%)
eGFR (ml/min)	71.6±20
BMI (kg/m2)	26.5±2.6
Diabetes Mellitus	11 (44%)
Hypertension	7 (28%)
HBOT indication (nonvascular)	18 (72%)
Coronary artery disease	2 (8%)

HBOT: hyperbaric oxygen therapy, eGFR: estimated glomerular filtration rate, BMI: body mass index.

Table 2. Laboratory values before and after the 10th HBOT session

	Pre-HBOT (n=25) Mean SD	Post-HBOT (n=25) Mean SD	P Value
LVEF (%)	59±7	60±6	0.20
LA (cm)	3.8±0.5	3.8±0.5	0.94
RVD (cm)	2.5±0.3	2.6±0.3	0.30
TAPSE (cm)	2.3±0.3	2.3±0.3	0.79
E/A	1.1±0.5	1.0±0.5	0.31
MPI	0.4±0.1	0.5±0.1	0.05
TDI S (cm/sec)	8.2±1.8	8.4±2.0	0.40
TDI E' (cm/sec)	10.3±2.5	10.6±3.1	0.44
TDI A' (cm/sec)	9.2±2.6	9.4±3.3	0.29
E/E'	7.9±2.7	8.1±2.5	0.75

HBOT: hyperbaric oxygen therapy, BNP: B-type natriuretic peptide, hsTnT: high-sensitive troponin T.

Table 3. Echocardiographic findings before and after the 10th HBOT session

	Pre-HBOT (n=25) Median (IQR 75-25)	Post-HBOT (n=25) Median (IQR 75-25)	P Value
BNP (pg/ml)	77 (386-21)	129 (784-51)	<0.01
hsTnT (ng/ml)	0.008 (0.016-0.005)	0.010 (0.021-0.006)	0.74
Creatinine (mg/dl)	0.8 (1.0-0.7)	0.9 (0.9-0.7)	0.80

HBOT: hyperbaric oxygen therapy, LVEF: left ventricular ejection fraction, RVD: right ventricle diameter, PABs: pulmonary artery systolic pressure, TAPSE: tricuspid annular plane systolic excursion, MPI: myocardial performance index, TDI: tissue Doppler imaging.

Other

PP-141

Impact of statin use on high sensitive troponin t levels with moderate exercise

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Background and Aim: High-sensitivity cardiac troponin (hsctn) levels can be also elevated in non-pathologic or non-cardiac events. An increase in cellular permeability might be a possible cause of this elevation. Statins, a widely used therapy for treatment and prevention of coronary artery disease, are known to increase the permeability of muscle cells, but their effects on cardiac muscle cells still are not fully elucidated. Therefore we aimed to evaluate the impact of statins on hsctn levels with moderate exercise.

Methods: We included twenty six statin users (55±9 y, 13 females) and thirty healthy controls (54±8 y, 13 females). None of the subjects had coronary artery disease, as proven by a coronary angiography. A fixed-protocol moderate level exercise on treadmill was performed by both groups. Venous blood was collected from all participants before and four hours after exercise to measure hsctn levels. Participants were also grouped according to their troponin T levels, as proposed in the recent ESC guidelines (0-1 hour algorithm) for acute coronary syndromes without persistent ST-segment elevation (NSTEMI).

Results: Statin users showed a significant increase in serum hsctn levels with moderate exercise (7.7±12.6 ng/l vs. 11.4±15.2 ng/l, p=0.004), whereas the control group only showed a modest increase without statisti-

cal significance (6.4±3.5 ng/l vs. 7.74±5.7 ng/l, p=0.664). Figure 1 presents increases in hscTn T levels for both groups. The number of subjects whose hscTnT levels exceeded rule-out limits for NSTEMI diagnosis (according to the 0-1 algorithm) after moderate exercise, differed significantly between groups: (statin users, 38.5% vs. control group, 10.0%, p=0.024). However, the number of subjects whose hscTnT levels exceed the 99th percentile upper reference limit with moderate exercise did not show any difference between groups (statin users, 7.7% vs. control group, 10.0%, p=0.999).

Conclusions: Statin therapy can cause significant increase in hscTnT levels after moderate exercise. This increase can jeopardize the correctness of clinical diagnoses which are based on newly implemented algorithms. The awareness of these adverse effects of statins, which are mainly used by patients with high risk of coronary events, can prevent misdiagnosis or unnecessary hospitalizations.

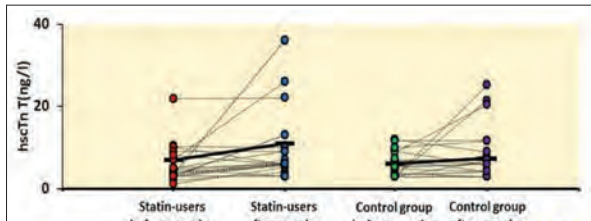


Figure 1. High-sensitive troponin T levels before and after exercise for statin users and control group. Significance is indicated on the graph.

Other

PP-142

Real time detection of S1 and S2 heart sounds

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Background and Aim: Phonocardiogram has recently become popular as a diagnostic tool, especially with the widespread use of electronic stethoscopes and rapid advances in artificial intelligence studies. Automatic detection of first heart sound (S1) and second heart sound (S2) is critical for diagnostic decision support systems that use heart sound as a means for decision making. Although these systems vary in both methodology and infrastructure, localisation of S1 and S2 in a cardiac cycle is the most critical step. A common detection approach in the literature is to locate peaks in the energy of the heart sound. The detected peaks are classified as S1 and S2 by analyzing all the peaks offline based on the assumption that systole is shorter than diastole. However, offline nature of these analysis are impractical since the output is needed during the auscultation not later. The subject of this study is to provide an algorithm for real-time detection of S1 and S2.

Methods: Study population consisted of three groups of patients: Group 1 included patients with normal cardiac auscultation findings. Group 2 consisted of patients with systolic murmurs, diastolic murmurs, physiological or paradoxical splitting. Group 3 consisted of pathological atrial and ventricular gallops. The proposed method starts with receiving raw audio signal from an e-stethoscope through the audio channel of the processing unit. The signal is first low-pass filtered then an envelope is constructed from the signal energy. The standard deviation of the envelope is employed as a threshold value for peak detection. Consecutive three peak values are utilized to estimate current and future locations of S1 and S2 and heart rate. These future estimates are used to optimize misinterpretations in S1 and S2 locations of the next heart cycle.

Results: The performance of the algorithm is tested on a study population of 25 patients. Group 1 included 24% (n=6) of the study group; Group 2 included 64% (n=16) of the patients and Group 3 included 12% (n=3) patients. The detection rate was 92%, 75% and 46% for Group 1, Group 2 and Group 3 patients successively.

Conclusions: In this study, the feasibility of real time detection of sS1 and S2 is shown. The method achieved 75 % success rate in Group 2 patients, although S1 and S2 sounds were barely visible in most of the cases. The fall in success rate in Group 3 patients is consistent with the findings in literature, since S and S4 are usually misinterpreted as S1 and S2 in severe gallop cases.



Figure 1. s1 and s2 detection example on phonocardiogram. Successful detection examples from each of the three groups.

Other

PP-143

Determination of malnutrition status of adult cardiology patients

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Background and Aim: Malnutrition refers to deficiencies, excesses or imbalances in a person's intake of energy and/or nutrients. Prevalence of malnutrition is about 15% in general population and 40% in the hospitalized patients. As well as causing serious problems in many organs, in disease states, it affects the prognosis negatively, increases complications, general morbidity and mortality rates. Thereby, early diagnosis of malnourished individuals and nutritional support is very important. In this study, we aimed to determine frequency of malnutrition by screening patients admitted to cardiology outpatient clinic and cardiology service.

Methods: Between October 2017 and April 2018, a total of 103 patients (55 outpatient clinic, 48 cardiology service) who applied to the cardiology department of Pamukkale University Hospital were included in the study. Demographic characteristics, anthropometric characteristics, body mass indexes of patients were recorded. Afterwards, "Mini Nutritional Assessment" (MNA) scale was used to determine the malnutritional status of patients. The ones whose MNA score is lower than 17 were considered as malnutrition group, MNA score between 17-24 were categorized at under risk and those with >25 were accepted as adequate nutrition group. Albumin, pre-albumin, vitamin D, vitamin B12 and transferrin were measured in the laboratory to determine the nutritional status of the patients.

Results: The average age of the total 103 patients was 57.5±17.5 and 51.5% of the study population was male. Of the patients, 37% were diabetic, 46% hypertensive, 34% hyperlipidemic and 58% had coronary artery disease. Mean albumin values of the patients were found as 4.2±0.5 gr / dl. The mean MNA value of the entire study group was 22.8±3.5, but 61% of patients' MNA value was below 24. Only 40 patients (39%) had sufficient nutritional status. When MNA values are compared between non-hospitalized and hospitalized patients, MNA values were found respectively as 23.3±3.5 and 22.3±3.5, (p=0.231). The albumin values (3.9±0.5 and 4.3±0.3, p < 0.001) and vitamin D values (10.7±5.8 and 17.8±11.1, p=0.010) were found significantly lower in the inpatient group compared to non-hospitalized patients.

Conclusions: In our study, 61% of patients were not at normal nutritional status. We strongly recommend physicians to evaluate their patients nutritional status and give nutritional support if needed.

Other

PP-144

Relation between paraoxonase-1 activity and pulse pressure index in patients with a acute ischemic stroke

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Background and Aim: Stroke is a multifactorial disease arising from genetic and environmental risk factors or their interaction. The underlying cause of the majority of ischemic strokes is an atherosclerotic plaque in the carotid arteries. Oxidative stress and blood pressure were found to be closely related with cardiovascular and cerebrovascular diseases. Oxidative modification of serum low density lipoproteins (LDL) in the arterial wall is considered to be an important early step in the development of atherosclerosis. Paraonase-1 (PON1) has been considered as an anti-atherosclerosis factor, because it can reduce the oxidative modification of lipoproteins and macrophage foam cell formation occurring in the early phase of atherosclerosis. Therefore, it is easily inferred that the changes in serum PON1 concentrations or activity can affect vascular disease status. Elevated pulse pressure (PP) may lead to an increased risk of cardiovascular and cerebrovascular morbidity and mortality. However, there are limitations for PP as an evaluation index. In order to overcome the defects of PP, there is a novel parameter, "pulse pressure/systolic pressure" called pulse pressure index (PPI) for assessment of cardiovascular outcomes. We investigated the association between paraoxonase-1 (PON1) activities and pulse pressure (PPI) in acute ischemic stroke patients.

Methods: We evaluated and compared the PON1 activity and PPI in 46 ischemic stroke patients and 32 control patients. They were classified into 2 groups: Ischemic stroke patients (Group 1, n=46), control patients (Group 2, n=32). Blood pressure measurements were performed in all patients within 10 minutes admitted to emergency care unit. The PP was calculated by subtraction of diastolic blood pressure (DBP) from systolic blood pressure (SBP). PPI was calculated "pulse pressure / systolic pressure". Independent sample t test and Chi-square analyses were used to compare differences between groups.

Results: Hypertension, age, diabetes mellitus, dyslipidemia significantly higher in ischemic stroke patients than control group (Table 1) (p<0.05). PPI was significantly higher in ischemic stroke patients than control group (0.469±0.0698 and 0.423±0.0485, p<0.05). PON-1 activity was significantly lower in ischemic stroke patients than in control group (417.76±79.72 and 432.34±92.05 p<0.05).

Conclusions: Our results suggested that, PON-1 activity and PPI are important risk factors in acute ischemic stroke patients.

Table 1. Clinical characteristics of patients

Variables	Group-1 (Ischemic Stroke) n=46	Group-2 (Control Group) n=32	p Value
Age (year)	68.57 ± 13.49	44.16 ± 18.67	0.003
Gender (F/M)	27 / 19	17 / 15	0.234
Hypertension	29 (65%)	6 (18%)	0.005
Diabetes Mellitus	14 (30%)	2 (6%)	0.007
Smoking	7 (16%)	3 (10%)	0.583
Dyslipidemia	11 (24%)	0 (0%)	0.004

Other

PP-145

Evaluation of serum digoxin measurements after educational intervention on healthcare workers in cardiology department: Preliminary results

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Background and Aim: In our prior retrospective study in which appropriateness of serum digoxin level measurement was evaluated, we found that 97% of the measurements were inappropriate regarding the blood sampling time (Oncu S, 2018). Therefore, in this study, we planned to investigate the effect of education on the appropriateness of serum digoxin concentration (SDC) measurement after training of the healthcare workers about digoxin therapeutic monitoring.

Methods: This study is an educational intervention study. During this study; the training was given to residents and nurses of Dokuz Eylul University Cardiology Department on December 2017; was reinforced with information meetings held once a month. SDC and other medical data of inpatients who were on digoxin treatment at the Department of Cardiology between January 1 and April 30 in 2018 were recorded prospectively. SDC measurements were evaluated according to the following 3 appropriateness criteria from the literature: right indications, appropriate blood sampling time: steady-state concentration reached (average 7 days), at least 6 hours after the last dose of drug. Results were evaluated with descriptive statistics, Mann Whitney-U and Chi-square analysis.

Results: Data of 58 patients (69.3±12.9 years; 62.1% women) were investigated. In 29.3% of the patients SDCs were not measured. In the remaining 70.7% of patients, 67 SDC measurements were recorded. In 19/67 (28.4%) of measurements, requests had no appropriate indication. In 28/67 (41.8%), samples were collected before reaching the steady-state concentration; while all samples were collected at the correct time after the last dose of drug. The inappropriate measurement rate was 41.8% when three of the appropriateness criteria were evaluated together whereas in our prior study conducted before educational intervention this rate was 97%. There was a significant relationship between increased patient age, duration of hospital stay, impairment of renal function tests, and inappropriate SDC measurement. Digoxin treatment was altered in 25.4% of patients after evaluation of SDC measurements; the most frequent alteration was discontinuation of treatment (88.2%).

Conclusions: Inappropriate SDC measurements were significantly reduced with training. However, concerns of physicians regarding the increased risk factors of the patients may still play a role in requesting inappropriate SDC measurement.

Table 1. Baseline characteristics and laboratory findings of the study population by appropriateness of measurements

	Appropriate Measurements	Inappropriate Measurements	P
Age (years)	70.7 ± 11.8	76.3 ± 13.3	0.030*
Female (n,%)	26 (66.7%)	15 (53.6%)	0.278**
BMI (kg/m2)	27.2 ± 5.4	26.0 ± 4.8	0.394*
Creatinine (mg/dL)	1.0 ± 0.4	1.3 ± 0.4	0.018*
GFR (CKD-EPI)	70.5 ± 25.2	54.5 ± 22.1	0.010*
BUN (mg/dL)	32.8 ± 39.6	32.4 ± 16.7	0.315*
Potassium (mmol/L)	4.3 ± 0.6	4.2 ± 0.6	0.894*
Digoxin Concentration (ng/mL)	1.1 ± 0.7	1.2 ± 0.7	0.995*
Number of Comorbidities (n)	4.2 ± 1.7	4.4 ± 1.3	0.444*
Hospitalization (day)	11.1 ± 8.9	15.3 ± 10.8	0.034*

*Mann Whitney-U **; Chi-square

Cardiovascular nursing / Technician

PP-146

Mental problems of caregivers of patients with heart failure

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Background and Aim: HF (heart failure) since there is a disease that requires lifelong follow-up, treatment and care, care givers occupy an important place in the lives of the patients. Although there are a large number of studies in the literature regarding care needs of patients with HF and many aspects of treatment, there is a small amount of data on psychiatric problems experienced by caregivers. The aim of the compilation planned in this direction is to draw attention to the caregivers of HF patients and to explain their psychological problems.

Methods: This research was prepared by searching the related researches published between 2010-2017.

Results: A survey of caregivers in among the causes of mental health problems seen in caregivers of patients with HF; low functional status, perception of heavy maintenance burden, lack of social support is located factors such as inadequate financial situation and low coping skills. Bahrami et al. In the qualitative researches that caregivers of HF patients examine the information needs and problems they experience, caregivers expressed lack of knowledge about disease management, physical and psychosocial burnout, and lack of social support. It has been reported that these negativities increase the burden of care and lead to the development of depression, sadness, fear and anxiety. In another study conducted on the subject, it was determined that 74.4% of the caregivers of the HF patients had psychological problems and the majority had anxiety and sleep disorders. In the same study, significant mental health problems were found in the

caregivers of KY patients under 40 years of age. In another study, it was determined that caregivers of HF patients had difficulty in giving care and that this affected their lives negatively. Again in the same research, the caregivers of single and young people have increased the incidence of mental problems. It has been reported that these negativities increase the burden of care and lead to the development of depression, sadness, fear and anxiety. In the same study, significant mental health problems were found in the caregivers of KY patients under 40 years of age.

Conclusions: In the light of this result, nurses should observe caregivers in terms of the symptoms of mental problems that may develop, should try to prevent mental problems before they develop, and plan nursing approaches to determine the factors that cause mental problems when they are observed and teach the coping methods.

Cardiovascular nursing / Technician

PP-147

Postoperative nursing care of norwood stage 1 procedure in patients with hypoplastic left heart syndrome

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Background and Aim: Hypoplastic left heart syndrome (HLHS) is a univentricular pathology, that is incompatible with life unless a multistage cardiac surgery or heart transplantation are performed. Norwood type operation is the procedure of choice. The first stage of the Norwood procedure constitutes aortic arch reconstruction using native main pulmonary artery, ductus ligation, atrial septectomy and construction of a modified Blalock-Taussig (MBT) shunt, or a RV-PA restrictive conduit named Sano modification. In this presentation we will share our experience of nursing care in postoperative stage 1 Norwood surgery patients.

Methods: Since May 2017, stage 1 Norwood surgery was performed in a total of 8 HLHS patients in our clinic. A MBT shunt was constructed in 3 patients, and Sano modification in the others. Surgery was performed in a mean day of 5, and at the mean weight of 3300 gr's. Patients were transferred to pediatric cardiovascular surgery intensive care unit with open sternum. Sternum could be closed at a mean of postoperative day four. In early postoperative period, patients were closely followed up by monitoring of diastolic blood pressure, urine output, blood gases and peripheral perfusion. To avoid pulmonary overflow we increased pulmonary vascular resistance (PVR). For this purpose, CO2 was kept between 45-55 and saturation was kept under 80% with respiratory maneuvers. To increase pulmonary flow, we used sildenafil and sometimes nitric oxide beside respiratory maneuvers to decrease PVR.

Results: 3 out of 8 patients died because of sepsis and multiorgan failure at postoperative days 22, 36 and 50. MBT shunt had been used in 2, and Sano in one of the deceased cases. 3 patients could be discharged from hospital at a mean of postoperative 18 days. Norwood second stage operation was performed in 2 of them. The last 2 cases are in postoperative 7. and 12. days, and in good cardiachemodynamic and neurologic condition.

Conclusions: In postoperative HLHS patients, aortopulmonary shunt, whether MBT or Sano, is the only source of pulmonary blood flow. So, increase in PVR decreases pulmonary flow and deteriorate patients condition. On the other hand any decrease in PVR increases blood flow to the lungs abruptly. Pulmonary overflow decreases coronary and peripheral perfusion. To our opinion, maintaining the balance between overflow and underflow is the most critical point in management of these patients. Complications of pulmonary overflow was less frequent, and so mortality was much less in our Sano patients. This is because RV-PA restrictive conduit is a more natural source of pulmonary flow than MBT shunts.

Other

PP-148

Effect of cardiac rehabilitation on exercise level in safe heart rate range

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Background and Aim: Cardiac rehabilitation (CR) is a treatment modality that has been proved to be useful and cost-effective in coronary artery disease and heart failure. It has been shown to decrease mortality and hospitalization. However, there is no evidence that shows CR increases maximal exercise capacity. Maximal exercise capacity is achieved at maximal heart rate, which is associated with high incidence of ischemia and arrhythmia. This level of heart rate is rarely needed in daily routine life. During CR sessions, the patients are promoted to exercise in heart rates lower than maximal heart rates, which are safer and more commonly used in daily routine life. In literature review, we couldn't find any study that compares the change in exercise capacity performed in this safe heart rate. In this study, we investigated the effect of exercise-based CR on exercise level performed in safer heart rates.

Methods: The study is designed retrospectively and patient analysis is still continuing. The exercise level in CR sessions are determined according to the symptoms and rate of perceived exertion. They are asked to exercise in 12-14/20. The study included all patients attended to CR regularly (>2/3 of sessions). The exercise level is quantified as MET (metabolic equivalent threshold). The maximum and mean heart rates, maximum and mean MET levels and MET/heart rate in the first session and last session are compared by Student's t test. Statistical significance was set at 0.05.

Results: Of 40 patients analyzed, 13 were female, mean age was 61±14 years. Average of maximum and mean HR are shown in Figure 1, MET levels in Figure 2 and MET/heart rate levels in Figure 3. Briefly, there were no differences in maximum heart rate levels. The other parameters were significantly increased. There were a decrease in maximum MET level in only 4 patients, mean MET level in only 2 patients and MET/heart rate in only one patient.

Conclusions: CR significantly increases exercise capacity performed at safe heart rate levels. The patients were able to exercise more intensively in heart rates that risk was low. All heart disease patients should be evaluated for suitability for CR and suitable ones should be referred to CR units.

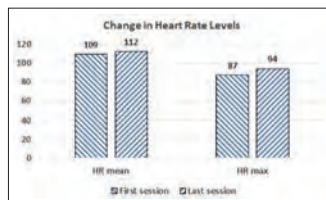


Figure 1.

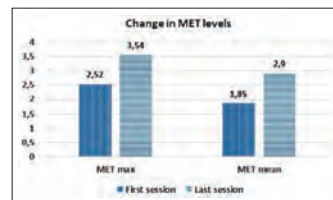


Figure 2.

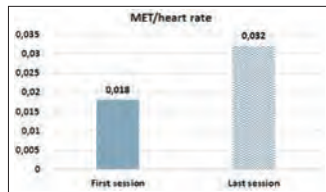


Figure 3.

Other

PP-149

The risk of thromboembolism may be associated with tumor size in patients with left atrial myxoma

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Background and Aim: Cardiac myxomas are the most common primary benign intracardiac tumors. Although myxomas are histologically benign, they are potentially dangerous due to potential risk of systemic and cerebral thromboembolism. In this study, we aimed to investigate the relationship between the tumor size and the risk of thromboembolism in patients with left atrial myxoma.

Methods: This single center retrospective study enrolled 93 patients [mean age: 52.9±15.3 years, female: 70 (75.3%)] with left atrial myxomas between 2014 and 2018. The patients were classified into embolic and non-embolic groups to investigate possible predictors of thromboembolism. Demographic parameters with electrocardiography, transthoracic and transesophageal echocardiography of all patients were recorded into a dataset.

Results: The study population was composed of 13 (14%) patients in embolic (11 cerebrovascular and 2 peripheral) and 80 (86%) patients in non-embolic group. There was no significant difference in terms of demographic parameters between the groups. The greatest tumor diameter assessed by transesophageal echocardiographic examination was significantly higher in the embolic group than non-embolic group (5.59±1.08 vs. 4.29±0.61; p=0.001). Possible predictors of thromboembolism such as age, left atrial diameter, left ventricular ejection fraction, tumor size, the presence of atrial fibrillation, hypertension and diabetes mellitus were entered into multivariate logistic regression analyses. By multivariate analysis, increased tumor size was identified as an independent predictor of thromboembolism. In ROC curve analyses, the greatest tumor diameter above 4.6 cm predicted thromboembolism with a sensitivity of 77% and a specificity of 73% (AUC: 0.858; 95% CI: 0.752 to 0.964; p<0.001).

Conclusions: Thromboembolism is a serious complication of left atrial myxomas. The risk of thromboembolism may be associated with increased tumor size. Early surgery should be scheduled in patient with large left atrial myxomas due to increased potential for thromboembolism.

Other

PP-150

The variant analysis in three exons of MYH7 gene related with hypertrophic cardiomyopathy

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Background and Aim: Although the frequency of pathogenic variants in the cardiac beta-myosin heavy chain 7 (MYH7) gene in the Turkish population is unknown, it is one of the primary candidate genes that cause hypertrophic cardiomyopathy (HCM). In this study, our aim was to determine a causing mutation in hotspot exons of MYH7 genes in Turkish patients with HCM.

Methods: The study included 95 unrelated patients with HCM (48.3±15.8 years, 40 female and 55 male, 15 familial and 80 sporadic cases). All participants were evaluated with a detailed history, physical examination, 12-lead electrocardiography and two-dimensional echocardiography. DNA was extracted from peripheral blood. We analyzed three exons of MYH7 gene in all patients using PCR-Sanger sequencing methods.

Results: From 95 patients with HCM, pathogenic variants were found in 5 patients in mutation screening of 18-20th exons in MYH7 gene and also one common polymorphism (rs3729818G>A) was genotyped. These missense mutations are Arg719Trp, Arg663His, Arg663Cys (in two cases) and Ile736Thr. These variants related with HCM were absent in exome database of 777 controls from Turkey. There was no significant dif-

ference between the patients with mutation (n=5, 32.6±22.8 years) and patients without mutation (49.4±15.04 years) for the clinical parameters except deceleration time (315.4±55.6 and 209.7±74.04ms, p=0.003, respectively). The rs3729818G>A polymorphism (global A-allele frequency=0.25 while A-allele frequency in this population=0.22) was not associated with any clinical parameters in patients with HCM.

Conclusions: In our study, the causal mutation rate in selected exons of MYH7 gene was 5.3%. In our population, this gene region might be a candidate for mutation screening in patients with HCM.

This study was supported by Turkish Society of Cardiology and Scientific Research Projects Coordination Unit of İstanbul University (Project numbers: 42173 and 53021).

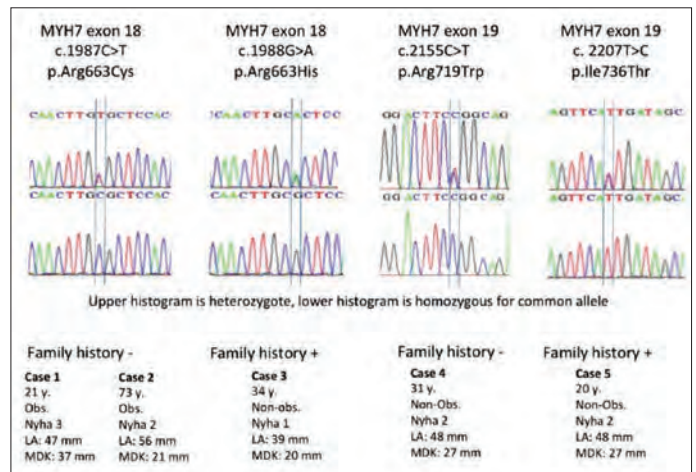


Figure 1. The identified clinical significance variants in five cases of HCM study population.

Other

PP-151

A new inflammatory marker: Elevated monocyte to HDL cholesterol ratio associated with smoking

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Background and Aim: The adverse effects of smoking in various pathologies are mediated by its effects on the inflammatory system. The monocyte to high-density lipoprotein cholesterol (HDL-C) ratio (MHR) has recently emerged as an indicator of inflammation. We aimed to investigate the relationship between MHR and cigarette smoking.

Methods: Three hundred and ninety seven consecutive participants who smoke and 515 healthy subjects with no history of smoking enrolled in the study. Complete blood count parameters and lipid profile were analyzed in all study participants. Smoking habits were calculated as pack.years and number of cigarettes smoked per day.

Results: MHR levels were significantly higher in smokers compared to non-smokers (respectively, 15.71 (12.02-20.00) and 11.17 (8.50-14.16), p<0.0001) (Table I). Pearson's correlation analysis revealed a weak but positive correlation between pack.year and MHR in the smokers group, and there was a moderate positive correlation between the number of cigarettes smoked daily and MHR in the group (Figure 1). In receiver operating characteristics (ROC) analyses, it was determined that a MHR value >13.00 measured in smoker participants at application had a predictive specificity of 66.6% and sensitivity of 70.0% for smoking (area under the curve [AUC] 0.729, 95% CI 0.696, 0.762; p<0.0001) (Figure 2).

Conclusions: Elevated MHR is associated with cigarette smoking and may be a useful indicator of a systemic inflammatory response in smokers. Smoker participants who have high MHR levels can easily be identified during routine complete blood count (CBC) analysis and could possibly benefit from preventive treatment.

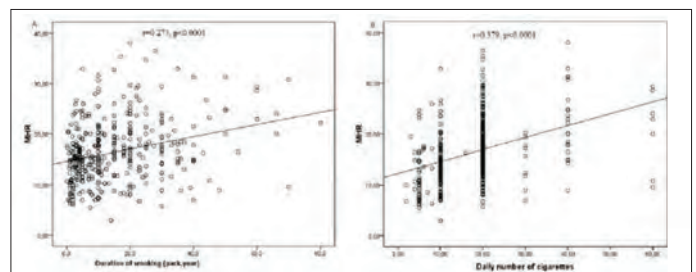


Figure 1. Correlations between MHR and duration of smoking (a) and number of cigarettes per day (b) in smokers.

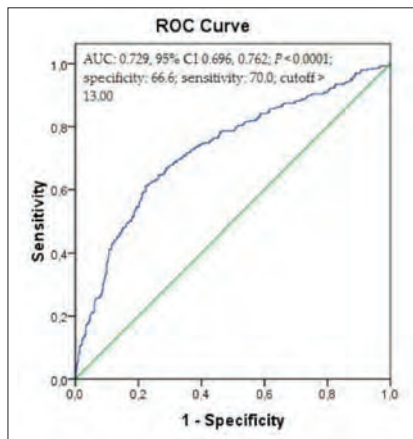


Figure 2. MHR ROC analysis between smokers and non-smokers.

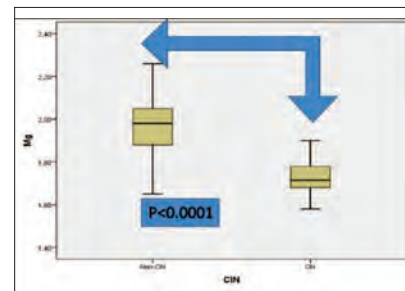


Figure 1. In comparison with patients without CIN, patients with CIN also had lower Mg levels (1.96± 0.15 vs. 1.73±0.09 p<0.0001).

Table 1. Inter-group comparison of demographical and laboratory data

	Smokers (397)	Non-smokers (515)	P value
Gender (Male/Female)	258/139	316/199	0.26
Age,(year)	37 (26.0-47.0)	34 (24.0-47.0)	0.10#
Hyperlipidemia, n (%)	78 (19.6)	82 (15.9)	0.14
Triglycerides, (mg/dL)	132 (94.75-186.0)	102 (72.0-147.0)	<0.0001#
Total cholesterol, (mg/dl)	177 (150.0-202.5)	172 (149.0-198.0)	0.33#
Low density lipoprotein cholesterol, (mg/dl)	104.0 (80.39-123.0)	99 (78.0-120.0)	0.07#
HDL cholesterol, (mg/dL)	41 (35-49)	50 (43-58)	<0.0001#
Monocytes, (×103/mm3)	0.65 (0.53-0.78)	0.56(0.46-0.67)	<0.0001#
MHR	15.71(12.02-20.00)	11.17(8.50-14.16)	<0.0001#
BMI (Body mass index)	25.40(24.28-27.35)	26.26(25.36-27.21)	<0.0001#
Platelet, (×103/mm3)	262 (228.0-301.0)	270 (230.0-313.0)	0.09#
White blood cell,(×103/mm3)	8.01±1.97	7.29±1.69	<0.0001
Glucose, (mg/dl)	94.73±18.66	94.25±15.91	0.68
Sodium, (mmol/L)	139.89±3.28	140.09±2.77	0.34
Potassium, (mmol/L)	4.27±0.41	4.25±0.47	0.50
Calcium, (mg/dL)	9.32±0.53	9.37±0.59	0.20
Urea, (mg/dl)	28.14±7.94	28.21±8.65	0.90
Creatinine, (mg/dl)	0.60±0.16	0.60±0.17	0.91
Hematocrit, (%)	43.88±3.21	41.88±2.98	<0.0001
Hemoglobin,(g/dl)	14.64±1.09	13.63±1.03	<0.0001

Normality of the distribution was evaluated by the Kolmogorov-Smirnov test and the Mann-Whitney U test applied to compare for continuous variables.

Other

PP-152

Magnesium levels and the risk of contrast induced nephropathy in patients undergoing coronary angiography or PCI

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Background and Aim: The aim of this study was to evaluate the role of hypomagnesemia(HypoMg) as a risk factor for the development of contrast induced nephropathy (CIN) after coronary angiography/percutaneous coronary intervention.

Methods: This is a single-center retrospective study conducted at a tertiary referral hospital. Between December 31, 2017, and February 28, 2018, a total of 149 patients who had undergone coronary angiography/intervention procedure were enrolled in this study. CIN was defined as an increase of >0.5 mg/dl or >25% in serum creatinine concentration over baseline within 48 h after administration. We categorized Mg levels as hypoMg, normoMg and hyperMg based on interquartile percentiles (Q1, Q3-Q1, Q3): less than 1.78, 1.78–2.01 and greater than 2.01 mg/dL. Logistic regression analysis was performed to obtain the odds ratio of CIN of various Mg levels using Mg with lowest CIN incidence (1.78–2.01 mg/dL) as the reference group.

Results: Of 149 patients enrolled, CIN occurred in 24 patients (16.1%). The lowest incidence of CIN was when serum Mg was within 1.78–2.01 and higher incidences of CIN associated with hypoMg (<1.78) (Figure 1). After adjusting for potential confounders, hypoMg was associated with an increased risk of developing CIN with odds ratios of 8.71 (95% CI 2.31-32.11) (Table 1).

Conclusions: HypoMg was associated with an increased risk for CIN.

Table 1. Odds ratios for the association between serum magnesium levels and CIN

Magnesium level (mg/dl)	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	Adjusted OR (95% CI)	p
<1.78	6.52(2.12-18.5)	<0.0001	8.71(2.31-32.11)	<0.0001
1.78-2.01	1(ref)		1(ref)	
2.01<	0.19(0.024-1.5)	0.22	0.31(0.028-3.41)	0.34

Note: Adjusted for age, sex, baseline GFR, history of CKD and coronary artery disease(Prior MI), ACS, anemia, diabetes mellitus, congestive heart failure, use of ACE/ARB and diuretic.

Other

PP-153

Are there any clinical and electrocardiographic predictors of heart rate reduction in relapsing- remitting multiple sclerosis patients treated with fingolimod?

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Background and Aim: Fingolimod, a sphingosine-1-phosphate (S1P) receptor agonist, is used for treatment of relapsing-remitting multiple sclerosis (RRMS). S1P receptors that fingolimod acts upon have also been shown to be expressed on atrial myocytes. This expression pattern has been linked with the drug's cardiovascular effects, such as bradycardia. We aimed to evaluate the clinical and electrocardiographic predictors of heart rate (HR) reduction in patients receiving first-dose fingolimod.

Methods: We retrospectively analyzed subjects diagnosed with RRMS who were allocated to fingolimod treatment. HR, systolic and diastolic blood pressure values and electrocardiography during the first dose of fingolimod were accessed.

Results: A total of 114 RRMS patients (65.8% female, 33.58±8.63 years) were included. After the initial dose of fingolimod, the heart rate decreased significantly at each hour (each p<0.001). Nadir heart rate was reached at 4 hours. The multivariate binary logistic regression analysis revealed that BMI (OR: 0.878, p=0.045), optic nerve involvement (OR: 3.205, p=0.018), baseline HR (OR: 1.079, p=0.002) and T-peak-T-end interval (OR: 1.046, p=0.030) were independent predictors of greater HR reduction. During 6-hour monitorization, none of the patients had relevant adverse reactions.

Conclusions: Our findings provide an insight on clinical and electrocardiographic predictors of HR reduction that occurs in RRMS patients receiving first dose of fingolimod.

Cardiovascular nursing / Technician

PP-154

The effect of media on heart health

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Background and Aim: Today, There are many reasons for the influence of media on health such as the rapid increase in the number of health relations programs on television and widespread use of computers and internet usage. The aim of study evaluates the effect of media on heart health.

Methods: This study was conducted as descriptive between May 2017 and June 2018 in Sivas as an activity World Hypertension Day. A hundred to eight people participated the study. As a data collection form, a questionnaire form was used to evaluate the effect of the media on heart health and a "motivation scale for monitoring health programs broadcast on television" developed by Koçak ve Bulduklı (2010). Data was analyzed using the Statistical Package for the Social Sciences (SPSS, Inc., Chicago, IL, USA) for Windows version 17.0. The descriptive statistics were presented using the arithmetic mean and standard deviation, minimum-maximum, frequency, percentage. Approval for the study was obtained from the ethical review boards at the authors' institution.

Results: Participants are constituted as 52.1% (n=98) male and 47.9% (n=90) female. The age of participants ranges from 18 to 78 years. The mean age was 42.21±16.01. Overall 95.2% of the participants have followed the media. The most frequently followed media tools are TV, Internet (Google etc.), Facebook. Most of the participants (n=142; 75.5%) stated that use media tools to access information about heart health. Information obtained from the media 57.4% of the participants stated that they restricted the salt. However few participant (n=65; 37.2%) stated that investigate the reliability of information.

Conclusions: It is important for health professionals to inform patients about the correct use of information in the media.

Other

PP-155

Long-term mortality among juxta-renal-aortic occlusion patients

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Background and Aim: Juxta-renal-aortic occlusion is frequently characterized by atherothrombotic occlusion. Atherosclerotic juxta-renal-aortic occlusion (JAO) frequently associated with mortality because of coronary artery disease (CAD) and stroke. Severity of peripheral arterial disease and predicted outcomes is effected by multiple factors. We aimed to search whether plasma d-dimer and fibrinogen levels on admission may predict survival, and long term prognosis.

Methods: In our study, 11345 patients which evaluated by peripheric angiography between 1998-2012 retrospectively examined and 7956 of them were diagnosed as peripheral arterial disease and 102 of them diagnosed as JAO (acute occlusion are excluded). Patients which have malignancy, recent pulmonary embolism, active infection, trauma or surgery history are also excluded. Classification of peripheral arterial disease has done by Fontaine calcification. Patients which have laboratory measurements at the time of diagnosis are included.

Results: JAO ratio between diagnosed patients by angiography in our center was %1.1. Mean age of JAO patients was 58±8.4 (38 to 87) with 87.2% male, 25.6% diabetes mellitus, 60.5% hypertension and 98.8% positive smoking history ratios. 26.7% of patients had critical limb ischemia (rest pain or ulcerated lesion or gangrene). 4.1% of patients were asymptomatic, 68.6% of them had intermittent claudication complaint. 41.2% of patients did not have CAD proven by angiography, one-vessel, two-vessels and three-vessels disease ratio were 18.8%, 22.4% and 17.6% consecutively. 91.9% of patients had invasive treatment by surgical or percutaneous method. Mean follow-up period was 81 months and 38 (44.2%) patients died during this period. Median survival time was 95 months. One-way analysis demonstrated low hemoglobin, high neutrophil and platelets, high d-dimer and fibrinogen levels and fontain class were associated. However, at multiple regression analysis, only d-dimer and fibrinogen levels were related with long-term mortality.

Conclusions: In the light of these data, which is widest and longest data in the literature, high fibrinogen and d-dimer levels in JAO patients foresights mortality and provides attention and option to more invasive intervention during follow-up and treatment process.

Other

PP-156

Evaluation of fetal safety of cardiovascular drug use in pregnancy with clinical pharmacology teratogenic risk analysis

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Background and Aim: Nowadays, the incidence of cardiovascular diseases is increasing. However, there is limited information on the use of cardiovascular drugs in pregnancy and in terms of fetal safety. In the treatment of cardiovascular diseases during pregnancy, it is important to select the drugs that best control the maternal disease as well as reduce the risks of both mother and the fetus. The purpose of this study is to assess the teratogenic safety of cardiovascular drugs in pregnancy.

Methods: Between 2014 and 2018, fifteen pregnant women who were admitted to the Clinical Pharmacology Teratology Information Service for teratogenic risk analysis due to cardiovascular drug use were included in the study. Teratogenic risk assessments were performed by questioning the demographic characteristics of the pregnant, additional risk factors, and the drugs they used. After giving birth, the families were contacted and information about infant development and health status was obtained.

Results: The diagnosis of pregnant women in the study population were hypertension, mitral valve replacement, arrhythmia and coronary artery disease. The drugs used by pregnant women were ramipril, trandolapril, warfarin, ticagrelor, aspirin, metoprolol, propranolol, bisoprolol, acetazolamide, hydrochlorothiazide, losartan, telmisartan, olmesartan and atorvastatin. Birth results were spontaneous abortion on the 10th week after the first 6 weeks of ramipril use, postnatal exitus on the 4th day due to congenital cardiac anomalies after the first 12 weeks of warfarin use, and intrauterine exitus on the 18th week after the first 6 weeks of telmisartan use.

Conclusions: Fetal/postnatal death after ramipril, telmisartan and warfarin were noted in this study. Drugs that act on the renin-angiotensin system may cause oligohydramnios due to decreased renal function in the fetus, lung hypoplasia, skeletal malformations and skull hypoplasia. When pregnancy is detected, these agents should be discontinued. Labetalol, nifedipine or methyldopa is recommended in the treatment of hypertension during pregnancy. On the other hand, warfarin used in thromboembolic conditions leads to coumarin embryopathy and nervous system anomalies during first trimester exposures. Except for mechanical heart valve pregnancies, which are at risk of high thromboembolism, their use in other pregnancies

Other

PP-157

Effect of heroin on right cardiac performance

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Background and Aim: Heroin addiction is one of the most destructive and expensive public health problems. Heroin, which is a central nervous system depressant (diacetylmorphine), is a semi-synthetic opiate. Mortality among heroin users varies between 1 and 3%, and the most effective treatment method of heroin addiction is opioid replacement therapy (1-2). Heroin is commonly used via smoking, snorting and iv injection. The common negative effects of heroin addiction are respiratory depression, which may lead to death, especially following intravenous injections. Additionally, heroin-related pulmonary edema has been reported in previous studies (3). However, not much is known about the cardiac effects of heroin addiction, which is an important public health problem of this extent. In particular, the iv use of the drug leads to an even more difficult evaluation, since the injection is generally performed together with other chemical substances named as adulterants (4). Therefore, we evaluated patients with heroin use via the smoking method, and aimed to investigate its effects on cardiac functions.

Methods: A total of 85 individuals including 45 patients using heroin via the smoking method as the study group and 40 healthy individuals with no drug addiction as the control group were included in the study. Patients using heroin via the injection method were excluded. Echocardiographic evaluation of those using heroin was performed and compared with those of the control group.

Results: The right ventricle and pulmonary artery diameters in the heroin group were found to be higher compared to the control group. The myocardial performance index (MPI) was higher and more abnormal in the heroin group (0.48±0.22 vs 0.39±0.11, p<0.05), whereas isovolumic acceleration (IVA) of the right ventricle was significantly lower in the heroin group (2.92±0.69 vs 3.4±0.68, p<0.01). No significant difference was observed between the groups with regard to the right ventricular ejection fraction (RVEF) (59.6±2.5 vs 60.6±2.3, p=0.08), tricuspid annular plain systolic excursion (TAPSE) (24.1±4.2 vs 24.5±2.4 p=0.7), tissue doppler imaging S wave (TDI-S) (13.7±2.1 vs 13.8±2.1, p=0.86) and right ventricular fractional area change (RV FAC) (42.7±8.3 vs 43.9±3.5, p=0.4). Multivariate and univariate regression analyses revealed independent correlation between the pulmonary artery diameter and RV IVA, and heroin addiction.

Conclusions: Heroin addiction, which is an important public health problem, negatively affects the right ventricular functions and more attention should be paid to the cardiac functions of these patients. Since the present knowledge on the effect of heroin use on cardiac functions is limited, this study is important for its contribution to the literature. However, further studies with larger sample size are needed for a consensus and clear results.

Other

PP-158

Impaired glucose tolerance is associated with severity of peripheral artery disease

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Background and Aim: Diabetes Mellitus is accepted as a risk factor for Peripheral Artery Disease (PAD). The correlation of Impaired Glucose Tolerance (IGT) with severity of PAD has not been investigated in literature.

Methods: 105 consecutive patients with normal fasting glucose levels who were diagnosed to have a preliminary diagnosis of PAD by noninvasive imaging methods were included in the study. 75 g Oral Glucose Tolerance Test (OGTT) was performed for diagnosis of IGT. The severity of PAD was defined according to TASC II (Trans-Atlantic Inter-Society Consensus document) criteria from conventional peripheral angiograms by a cardiologist who was blinded to OGTT results. TASC C-D patients constituted the severe PAD group and were compared to TASC A+B patients.

Results: Patients with severe PAD had higher frequencies of hyperlipidemia and smoking. Even though fasting glucose and HgA1c levels were similar between the two groups, 2-hours glucose levels of OGTT and frequency of IGT was higher in the severe PAD group. In Multivariate Logistic Regression Analysis, presence of IGT [odds ratio (OR)=3.296, 95% CI=(1.087-9.997), p=0.035], smoking (OR=3.395, 95% CI=(1.114-10.343), p=0.032) were independent correlates of severe PAD.

Conclusions: The presence of IGT is associated with more severe PAD and is important especially in the evaluation of the cardiovascular system.

Other

PP-159

The effect of exercise base cardiac rehabilitation on arterial stiffness in patients with coronary artery disease: By cardio-ankle vascular index

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Background and Aim: It was shown that exercise based cardiac rehabilitation reduced cardiovascular (CV) mortality in patients with coronary artery disease (CAD). Increased arterial stiffness is a marker of

CV disease and has been recognized as a key mediator of the CV events. We hypothesized that one of the mechanisms of exercise based CR for decreasing CV mortality and morbidity in patients with CAD may be related with improved arterial stiffness.

Methods: The study was composed of 97 patients with CAD and 33 patients without CAD. We analyzed CAVI values pre- and post- CR program. Patients with CAD were then divided into tertiles according to their SYNTAX score, defined as SYNTAX-low, SYNTAX-mid and SYNTAX-high. We also compare pre-post CAVI values of each SYNTAX groups.

Results: We showed that CAVI values are decreased with CR in CAD and control group patients (p<0.001 and p<0.001 respectively). The difference between post-CR and pre-CR CAVI values (CAVI-post-pre change %) is higher in CAD group than control group (15.4±7.8 vs 10.5±6.1 p<0.001). In addition, post-CR CAVI values are statistically lower than pre-CR CAVI values in SYNTAX-low, SYNTAX-mid and SYNTAX-high groups (p<0.001, p<0.001 and p<0.001 respectively).

Conclusions: This study showed that exercise based CR improves arterial stiffness in patients with CAD.

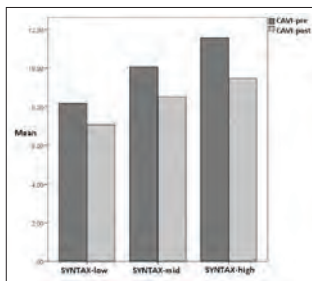


Figure 1. Pre- and post CR CAVI values in SYNTAX groups.

Table 1. Pre- and post-CR variables

Variables	CAD group(n:97)	Control group(n:33)	p value
CAVI-pre	10.10±1.86	7.92±1.11	<0.001
CAVI-post	8.47±1.37	7.07±1.04	<0.001
CAVI-post-pre change %	15.4±7.8	10.5±6.1	<0.001
P	<0.001	<0.001	

Other

PP-160

Right atrial volume and phasic functions in healthy smokers

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Background and Aim: Smoking is a risk factor for cardiovascular diseases. It may contribute to the development of atrial fibrosis via nicotine. The aim of this study was to evaluate the right atrial volume and phasic functions in apparently healthy smokers.

Methods: Forty healthy smokers and 20 healthy nonsmokers were consecutively included in the study. None of the subjects had additional cardiovascular risk factor other than smoking. The right atrial and ventricular functions were assessed by speckle tracking echocardiography.

Results: The echocardiographic parameters of the smokers and nonsmokers are listed in Table 1. The smokers had significantly larger right atrial volumes. The right atrial reservoir and conduit strain were lower in healthy smokers compared to those of nonsmokers although the difference was not statistically significant. Right ventricular global longitudinal strain was significantly lower in smokers.

Conclusions: Smoking impairs right atrial and ventricular functions even in apparently healthy young people with no other additional cardiovascular risk factors. Speckle tracking echocardiography is useful in detecting subclinical right atrial and ventricular dysfunction in healthy smokers.

Table 1. Pre- and post-CR variables

	Smokers (n= 40)	Nonsmokers (n= 20)	p
Age (years)	34.4 ± 7.8	26.5 ± 3.4	<0.001
Male sex (n-%)	28 (70%)	9 (45%)	0.060
RAVmax (mL)	42.4 ± 17.0	28.7 ± 9.2	0.001
RAVmin (mL)	17.0 ± 9.1	10.8 ± 3.8	0.002
RAVpre-A (mL)	26.4 ± 12.2	17.2 ± 5.4	0.001
RA total stroke volume (mL)	25.4 ± 11.2	17.9 ± 7.1	0.010
RA total emptying fraction (%)	60.3 ± 12.6	61.2 ± 10.6	0.857
RA passive stroke volume (mL)	16.0 ± 8.1	11.5 ± 5.4	0.033
RA passive emptying fraction (%)	38.2 ± 11.9	38.9 ± 10.5	0.707
RA active stroke volume (mL)	9.4 ± 4.6	6.4 ± 2.6	0.007
RA active emptying fraction (%)	36.7 ± 12.5	37.1 ± 9.8	0.695
RA expansion index	183.1 ± 114.8	178.4 ± 85.5	0.857
RA reservoir function (%)	35.7 ± 10.2	40.8 ± 13.1	0.207
RA conduit function (%)	16.0 ± 6.1	19.4 ± 7.6	0.106
Right ventricular global longitudinal strain (%)	-18.5 ± 2.2	-21.3 ± 1.5	<0.001

RA: right atrium, RAV: right atrial volume

Coronary artery disease / Acute coronary syndrome

PP-161

Is ischemia modified albumin indicative of good coronary collateral circulation

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Background and Aim: It is important to determine the severity of the disease in patients with stable coronary artery disease (CAD). We investigated the relationship between ischemia modified albumin (IMA) level and good coronary collateral circulation (CCC) in this study.

Methods: A total of 95 patients with coronary angiography with at least one main coronary artery obstruction were included in the study. Rentrop classification was used with coronary collateral circulation grading. 0 and 1 were defined as poor collateral, 2 and 3 were defined as good collateral. The IMA level of the patients was measured by Enzyme Linked Immune Sorbent Assay. The receiver-operating characteristic (ROC) curve was used to show the sensitivity and specificity of IMA levels and optimal cut off value for predicting good CCC.

Results: In multiple logistic regression analysis, the IMA level in the good collateral coronary circulation group was higher (p<0.045). Conversely, high-sensitivity C-reactive protein (HsCRP) level was also higher in the poor coronary collateral group (p<0.023). We found an IMA cut-off value (4.7 ng/ml) that showed good collateral level. This shows good coronary collateral circulation with 70.2% sensitivity and 60.3% specificity.

Conclusions: The level of IMA may be a simple and useful indicator of CCC.

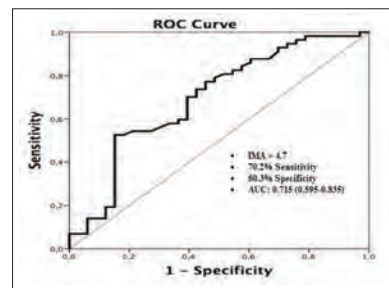


Figure 1.

Coronary artery disease / Acute coronary syndrome

PP-162

The association of nonalcoholic fatty liver disease with SYNTAX score in patients with st-segment elevation myocardial infarction

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Background and Aim: Nonalcoholic fatty liver disease (NAFLD) is the most common liver disease all around the world. Obesity, hyperlipidemia, diabetes mellitus and insulin resistance are mutual predisposing factors for coronary artery disease (CAD) and NAFLD. Coexistence of these numerous risk factors led to evaluate the association of NAFLD and CAD complexity, coronary artery calcification and endothelial dysfunction. Establishment of close relationship between NAFLD and STEMI may lead to screen and manage CAD aggressively in patients with NAFLD. We investigated the association of NAFLD and its grade with SYNTAX score in patients with ST-segment elevation myocardial infarction (STEMI).

Methods: The study group was consisted of 360 patients with STEMI. All patients were classified according to presence or absence of fatty liver and its severity using ultrasonography. Based on this classification, all patients were divided into 4 subgroups as Grade 0 (no fatty liver), Grade 1, Grade 2 and Grade 3.

Results: The patients with Grade 0 NAFLD had significantly lower 3 vessels stenosis, left main coronary artery (LMCA) stenosis and SYNTAX score compared to other subgroups. The mean SYNTAX scores for Grade 0, Grade 1, Grade 2 and Grade 3 NAFLD were 7, 14, 20 and 26 respectively. Additionally, the patients with Grade 3 NAFLD had significantly higher 3 vessels disease, LMCA stenosis and SYNTAX score compared to other subgroups. TIMI flow grades before and after intervention, intervention types and stent types were similar between the subgroups. As a consequence; Grade 0 subgroup had an especially lower severity of CAD and Grade 3 subgroup had an especially higher severity of CAD. Severity of CAD was similar between the Grade 1 and 2 subgroups (Fig. 1).

Conclusions: In patients with STEMI, the presence of NAFLD is associated with more severe and complex coronary artery disease. The current study support NAFLD screening in patients with STEMI.

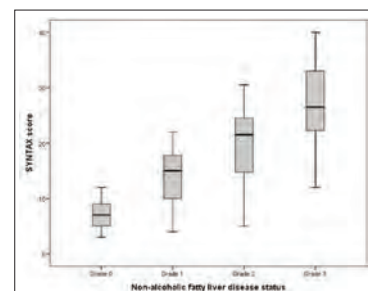


Figure 1. Box-plot illustrates the distribution of SYNTAX score stratified by non-alcoholic fatty liver disease status.

Table 1. Baseline characteristics, laboratory and angiographic analyses of the groups

	Grade 0 (n=169)	Grade 1 (n=84)	Grade 2 (n=71)	Grade 3 (n=36)	P
Baseline characteristics					
Age (years)	58 ± 13	59 ± 12	62 ± 12	63 ± 11	0.058
Male gender	104 (61.5%)	62 (73.8%)	47 (66.2%)	28 (77.8%)	0.114
Hypertension	78 (46.2%)	54 (64.3%)	44 (62.0%)	22 (61.1%)	0.017
Diabetes mellitus	40 (23.7%)	28 (33.3%)	27 (38.0%)	15 (41.7%)	0.045
Current smokers	49 (29.0%)	32 (38.1%)	39 (54.9%)	16 (44.4%)	0.002
Chronic renal failure	30 (17.8%)	16 (19.0%)	9 (12.7%)	9 (25.0%)	0.450
Hyperlipidemia	64 (37.9%)	46 (55.4%)	33 (46.5%)	23 (63.9%)	0.007
Left ventricular ejection fraction (%)	46 ± 16	44 ± 14	44 ± 13	40 ± 14	0.018
Systolic blood pressure (mmHg)	135 ± 25	131 ± 25	127 ± 29	132 ± 25	0.353
Laboratory analysis					
Fasting glucose (mg/dL)	115 ± 50	124 ± 55	131 ± 57	123 ± 41	0.026
Total cholesterol (mg/dL)	169 ± 62	174 ± 47	184 ± 41	188 ± 54	0.019
Triglyceride (mg/dL)	120 ± 58	165 ± 128	155 ± 84	160 ± 91	0.001
Serum creatinine (mg/dL)	0.89 ± 0.12	0.92 ± 0.14	0.96 ± 0.19	1.03 ± 0.24	0.112
White blood cell count (cells/ μ L)	8.2 ± 3.6	8.8 ± 3.2	8.9 ± 3.4	9.4 ± 3.2	0.929
Hematocrit (%)	35.9 ± 6.4	35.9 ± 5.7	38.6 ± 6.4	38.2 ± 5.2	0.072
Vessel stenosis (>50%)					
1 vessel	129 (76.3%)	24 (28.6%)	18 (25.4%)	4 (11.1%)	<0.001
2 vessels	25 (14.8%)	24 (28.6%)	15 (21.1%)	7 (19.4%)	0.077
3 vessels	15 (8.9%)	36 (42.9%)	38 (53.5%)	25 (69.4%)	<0.001
Left main coronary artery stenosis	1 (0.6%)	7 (8.3%)	16 (22.5%)	14 (38.9%)	<0.001
SYNTAX score	7 ± 2	14 ± 5	20 ± 9	26 ± 9	<0.001
Intervention type					
PTCA	23 (13.9%)	9 (11.0%)	9 (12.7%)	5 (14.7%)	0.919
PTCA and stent	78 (47.0%)	46 (56.1%)	26 (36.6%)	13 (38.2%)	0.080
Direct stent	20 (12.0%)	10 (12.2%)	12 (16.9%)	4 (11.8%)	0.758
Stent type					
Drug eluting stent	82 (49.4%)	48 (58.5%)	32 (45.1%)	15 (44.1%)	0.312
Bare metal stent	16 (9.6%)	8 (9.8%)	6 (8.5%)	2 (5.9%)	0.905

Coronary artery disease / Acute coronary syndrome**PP-163**

Effect of invasive strategy on long term mortality in elderly patients presented with acute coronary syndrome: A single center study

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Background and Aim: The elderly have the highest incidence of cardiovascular disease and frequently present with acute coronary syndrome (ACS). Due to multiple comorbidities and fragility at this age, treatment strategy of ACS at this population is not clear. In this study our aim was to evaluate the effect of invasive strategy on long term mortality in patients presented with ACS and ≥ 80 years old.

Methods: Patients who were admitted to our hospital with ACS were determined by using appropriate ICD codes in computerized hospital data system. After exclusion of patients below 80 years old, remaining 156 patients involved in final analyses. 94 of 156 patients (60.3) underwent coronary angiography and they constituted the "invasive strategy" group, whereas remaining 62 (39.7%) patients treated medically and they constituted "conservative strategy" group.

Results: Median follow-up duration of patients were 8.5 (0-61) months. Total mortality rates at the end of total follow-up period was 24 (25.5%) at invasive strategy group and 30 (48.4) at conservative strategy group ($p=0.006$). According to Cox regression analysis invasive strategy [OR:0.26, 95% CI (0.12-0.56), $p=0.001$], presentation with ST segment elevation myocardial infarction [OR:7.76, 95% CI (1.74-34.57), $p=0.002$], low ejection fraction below 40% [OR:3.11, 95% CI (1.43-6.76), $p=0.004$], heart rate [OR:0.98, 95% CI (0.96-0.99), $p=0.013$] and high GRACE risk score >150 [OR:7.76, 95% CI (1.74-34.57), $p=0.002$] were related to long term mortality.

Conclusions: Our results showed benefit of invasive strategy on mortality in elderly patients over ≥ 80 years old and presenting with ACS.

Coronary artery disease / Acute coronary syndrome**PP-164**

Fragmented QRS: A marker of microvascular dysfunction?

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Background and Aim: Fragmented QRS (fQRS) has been known to be a report of cardiac events but the relationship of fQRS on microvascular dysfunction in acute myocardial infarction has not been investigated sufficiently. We investigate the relationship between the presence of fQRS and microvascular dysfunction in patients with acute inferior myocardial infarction (MI) who underwent primary percutaneous coronary intervention (pPCI).

Methods: A total of 128 patients (mean age: 58 ± 9 years) with acute inferior MI were included. Fragmented QRS contains different morphologies of QRS, and it includes an additional R wave (R') or notching in the nadir of the S wave damaged area in two contiguous leads. ECG recordings were obtained for the evaluation of the fQRS, and ST segment resolution was evaluated at the first hour after the procedure. For the angiographic evaluation, the myocardial blush grade (MBG) was measured in infarct-related right coronary artery after the procedure. Patients were categorized into two groups of those with fQRS and without fQRS.

Results: In this study, 63 and 65 patients had an fQRS(+) or fQRS(-), respectively. Patients with fQRS(+) had lower ST segment resolution ($p=0.001$) and MBG ($p<0.001$) in the table. In addition, these patients have a lower ventricular ejection fraction ($p=0.02$). Correlation analyses showed a positive correlation between fQRS(+) and TIMI frame count at the post-procedure ($r=0.238$ and $p=0.01$).

Conclusions: In our study, microvascular dysfunction was more prominent in patients with fQRS(+). The fQRS is a simple and inexpensive parameter that can be used to predict the long term adverse events after a myocardial infarction.

Table 1.

	fQRS(+)	fQRS(-)	P
Age(years)	59±10	56±9	0,16
Myocardial blush grade	1,5±0,57	2,28±0,56	<0,001
ST segment resolution	45±22	59±23	0,001
TIMI frame count	26±11	20±9	0,01
Ejection fraction (%)	44±6	49±8	0,02

Coronary artery disease / Acute coronary syndrome**PP-165**

The comparison of prognosis between patients with inferior MI, non-complicated and complicated by right ventricular involvement after successful percutaneous coronary intervention

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Background and Aim: Right ventricular (RV) involvement portend a worse prognosis to uncomplicated inferior myocardial infarction (MI) with complications increasing in-hospital morbidity and mortality. Some studies have shown that reperfusion therapy, particularly by primary percutaneous coronary interventions (PCI), enhanced RV functional recovery in most patients. However there is limited data on the comparison between prognosis of inferior MI complicated by RV myocardial involvement and non-complicated inferior MI when successful primary PCI is provided in right coronary artery occlusions. In our study we aimed to evaluate the difference between the prognosis of these two groups after successful primary PCI.

Methods: Patients with inferior MI were divided into two groups as with and without RV myocardial involvement. We examined the incidence of death, duration of hospital stay, major and minor complications in patients after successful primary PCI. Right ventricular infarction diagnosis was identified with V4R >1 mV ECG criterion in right chest leads. Successful primary PCI criterion were accepted as relief in chest pain, 70% decreasing in the total ST segment elevation and obtaining TIMI III coronary flow. Transthoracic echocardiography was performed in the first 24 hours after PCI.

Results: A total of 46 patients with inferior MI were included to the study with pure inferior MI ($n=29$) and with RV involvement ($n=17$). General characteristics of the patients are given in Table 1. There were significant differences between the groups, in the TAPSE, right ventricular diameter and FAC measurements ($p<0.001$) (Figure 1-3, Table 2). No death and major complications were observed during the hospital stay in both groups. In terms of minor complications, one patient was found to have hypotension in the right ventricular infarction group and inotropic therapy was required ($p>0.05$) (Table 3). Atrial fibrillation was detected in one patient in the inferior MI group and statistically no difference was found. Although hospital stay was slightly longer in patients with right ventricular infarction group but it wasn't statistically significant ($p>0.05$). There was strong correlation between peak CK-MB levels and duration of coronary intensive care unit stay ($r=0.803$ $p<0.001$) and moderate correlation between peak CK-MB levels and duration of hospital stay ($r=0.668$ $p<0.01$) in the patients with inferior MI + RVMI (Table 4). But there was no correlation between peak CK-MB levels and duration of hospital stay in the patients with non-complicated inferior MI.

Conclusions: There was no difference in terms of major complication, minor complication, need for inotropic treatment, duration of coronary intensive care unit and hospital stay between patients with pure inferior MI and inferior MI complicated by RV involvement after successful primary PCI.

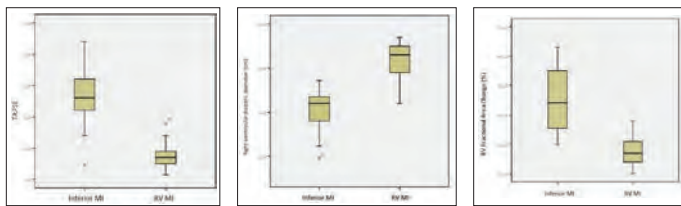


Figure 1. Tricuspid annular plane systolic excursion (TAPSE). MI: Myocardial Infarction, RV: Right ventricle.
Figure 2. Right ventricular diastolic diameter. MI: Myocardial infarction, RV: Right ventricle.
Figure 3. Right ventricle fractional area change. MI: Myocardial infarction, RV: Right ventricle.

Table 1. General characteristics

	Inferior MI (n:29)	Inferior + RV MI (n:17)	p value
Age (years)	57 (± 11)	55 (± 12)	> 0,05
Male gender (n (%))	24 (83 %)	15 (88 %)	> 0,05
HT (n, (%))	13 (45 %)	8 (47 %)	> 0,05
DM (n, (%))	12 (42 %)	8 (47 %)	> 0,05
FH (n, (%))	9 (31 %)	4 (23,5 %)	> 0,05
Smoke (n, (%))	15 (52 %)	8 (47,1)	> 0,05
DM (n, (%))	12 (42 %)	8 (47%)	>0,05
Pain- to Door time (m)	145 (± 146)	148 (± 118)	>0,05
Door to Baloon time (m)	22 (± 7)	25 (± 3,1)	>0,05
TIMI risk score	2,83 (± 1,00)	2,82 (± 0,95)	>0,05

DM: Diabetes Mellitus, FH: Family History, HT: Hypertension, m:Minute, MI: Myocardial Infarction, RV: Right Ventricular

Table 2. Echocardiographic findings

	Inferior MI	Inferior + RV MI	P value
LVEF (%)	44,16 (± 6,20)	(45,38 (± 5,19)	> 0,05
TAPSE	2,35 (± 0,46)	1,39 (± 0,21)	< 0,001
RVDD (cm)	3,06 (± 0,45)	1,17 (± 0,41)	<0,001
RV FAC (%)	44,79 (± 10,77)	28,15 (± 5,57)	<0,001

FAC: Fractional area change, LVEF:Left ventricular ejection fraction, MI:Myocardial Infarction, RVDD: Right ventricular diastolic diameter.

Table 3. Major and minor complications

MAJOR complications	Inferior MI	Inferior + RV MI	P value
Death	0	0	
Cardiogenic Shock	0	0	
VF	0	0	
3th AV block	0	0	
Other rhythm disorders that require PM	0	0	
MINOR complications	0	0	
Symptomatic others AV blocks	0	0	
Symptomatic sinus bradycardia	0	0	
Symptomatic hypotension	0	1	>0,05
Inotrop treatment	0	1	> 0,05
IABP	0	0	
AF	1	0	> 0,05

AF: Atrial Fibrillations, AV: Atrioventricular, IABP: Intraaortic baloon Pump, MI: Myocardial infarction, PM: Pace Maker, VF: Ventricular Fibrillation.

Table 4. Hospitalization times

	Inferior MI	Inferior + RV MI	P Value
Total HT (days)	5,69 (2,67)	7,00 (3,64)	> 0,05
Coronary ICU HT (days)	2,51 (0,94)	3,24 (2,31)	> 0,05

ICU: Intensive care unit, HT: Hospitalization times, RV MI: Right Ventricular myocardial infarction.

Coronary artery disease / Acute coronary syndrome

PP-166

Monocyte count to high density lipoprotein ratio as a novel parameter for early risk stratification in non-ST elevation acute coronary syndrome

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Background and Aim: A novel inflammation-based marker, the monocyte count to HDL-C ratio (MHR) has been recently reported as a new predictor and prognostic indicator of cardiovascular diseases. MHR has been found to be a predictor of stent thrombosis and in-hospital MACE as well as mortality in STEMI patients. Besides that, there is no specific data evaluating the association of MHR with the traditional risk score modalities in patients with NSTEMI-ACS. In this study, we aimed to investigate the usefulness of MHR in predicting coronary artery disease severity and relationship with the traditional risk scores in patients with NSTEMI-ACS. **Methods:** 443 NSTEMI ACS patients 311 patients records were retrospectively reviewed and analyzed by using our database. Based on hospital records, patient baseline clinical, demographic characteristics and laboratory parameters including complete blood cell count and blood evaluations for determining the blood glucose, creatinine, HDL-C and troponin I levels on admission were obtained. The TIMI, PURSUIT, and GRACE risk scores were calculated from the initial clinical history, electrocardiogram, and laboratory values collected on admission. The MHR was calculated as the ratio of monocyte count to HDL-C level multiplied by 100. The estimated glomerular filtration rate (eGFR) was calculated using the Cockcroft Gault equation. The anatomic and clinical severity of coronary stenosis identified as the syntax (SX), SXPCL and SX CABG was quantitatively evaluated by using the downloaded version from www.syntaxscore.com. **Results:** 337 NSTEMI ACS patients who underwent coronary angiography with or without percutaneous coronary intervention (PCI), were retrospectively enrolled in our study according to inclusion criteria. Of these, 232 (68.8%) were male and 215 (63.7%) were HT, 103 (30.6%) were DM and 112 (33.2%) were HL and 178 (52.8%) were smoking. According to the correlation analysis, MHR exhibited a significant positive correlation with the GRACE (p=0.02, r=0.162) and PURSUIT risk scores (p=0.006, r=0.190) but not with the TIMI risk score (p=0.827, r=0.15). Besides that, we did not demonstrate any relationship of MHR with the syntax (p=0.500, r=0.048), modified syntax PCI (p=0.113 r=0.111) and modified syntax CABG (p=0.311, r=0.342) which are quantitative indicators of coronary artery disease severity. **Conclusions:** MHR may be useful in early risk stratification for NSTEMI ACS patients, as an oxidative and inflammatory stress indices.

Coronary artery disease / Acute coronary syndrome

PP-167

Serum sirtuin 1, 3 and 6 levels in acute myocardial infarction patients

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Background and Aim: Sirtuins are involved in many cellular processes such as apoptosis, DNA repair, lipid/glucose metabolism. Recent experimental studies suggested that some sirtuin types may have some protective effects against endothelial dysfunction, atherosclerosis, cardiac hypertrophy and reperfusion injury. The data about the sirtuins in acute myocardial infarction (AMI) patients is scarce. This study investigated temporal changes of serum sirtuin 1, 3 and 6 levels in AMI patients, compared if there was any difference of serum sirtuin 1, 3 and 6 levels between AMI patients and control subjects and evaluated if there was any association between serum sirtuin 1, 3 and 6 levels with prognostic markers of AMI. **Methods:** Forty patients with AMI and 40 patients with normal coronary arteries were included. Left ventricle ejection fraction (LVEF), serum proBNP, CRP, sirtuin1, sirtuin 3 and sirtuin 6 levels were recorded. Peak troponin T levels, GRACE score and admission, first day and second day sirtuin levels were recorded in AMI patients. **Results:** Serum sirtuin 1,3 and 6 levels were similar in AMI patients and normal coronary patients. There was no temporal change in serum sirtuin 1,3 and 6 levels in AMI course. There was no correlation between sirtuins and proBNP, CRP, peak troponin and LVEF. Baseline sirtuin 1 and 6 levels were correlated with reperfusion duration. Baseline sirtuin 3 levels were negatively correlated with GRACE score. **Conclusions:** Serum sirtuin 1, 3 and 6 levels are similar in AMI patients and normal coronary patients. According to our results, there exist no evidence regarding the possible protective effects of sirtuin 1, 3 and 6 in AMI patients.

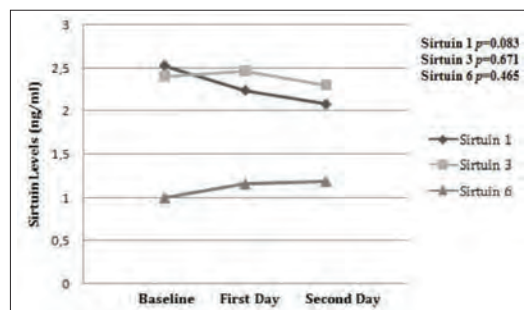


Figure 1. Temporal changes of serum sirtuin 1, 3 and 6 levels in acute myocardial infarction patients.

Table 1. Clinical and laboratory characteristics of the study patients

	Control patients (n=46)	AMI patients (n=46)	P Value
Age, y	68±9	67±14	0.481
Sex, Male, n(%)	33(71.7)	37(80.4)	<0.001
Hypertension, n(%)	20(43.5)	34(73.9)	0.175
Smoking, n(%)	8(17.2)	27(58.7)	<0.001
Diabetes Mellitus, n(%)	16(34.8)	11(23.9)	0.237
Family History for CAD, n(%)	4(8.7)	10(21.7)	0.077
Hyperlipidemia, n(%)	3(7.5)	5(12.8)	0.458
Fasting blood glucose, mmol/L	5.8(5.3-6.7)	5.9(5.4-6.2)	0.164
Creatinine, µmol/L	75±16	84±14	0.006
Total cholesterol, mmol/L	4.8±0.7	4.8±0.9	0.678
HDL, mmol/L	1.1±0.3	1.0±0.1	0.101
LDL, mmol/L	2.7±0.9	2.5±0.8	0.392
Triglyceride, mmol/L	4.3±2.2	3.9±2.1	0.305
Hemoglobin, g/dL	14.6±1.9	14.7±1.8	0.904
Platelet count, *10 ⁹	279±70	279±61	0.001
WBC, *10 ⁹	8.9±1.9	11.8±2.1	<0.001
Neutrophil count, *10 ⁹	5.3±1.4	8.8±2.1	<0.001
Lymphocyte count, *10 ⁹	2.6±0.9	2.0±0.3	0.001
Monocyte count, *10 ⁹	0.8±0.9	0.7±0.1	0.581
CRP, mg/dL	28.3(9.1-57.1)	85.7(35.1-130.9)	<0.001
SS score, n(%)	48.8(24.2-113.0)	500.9(212.0-1,039.0)	<0.001
SS score, n(%)			
SS score 1 basal	2.74(2.30-3.04)	2.53(2.06-3.21)	0.192
SS score 1 first day	NA	2.24(1.89-2.89)	
SS score 1 second day	NA	2.08(1.88-1.18)	
SS score 2 basal	2.62(2.16-3.34)	2.48(1.29-3.29)	0.204
SS score 2 first day	NA	2.46(1.27-2.97)	
SS score 2 second day	NA	2.38(1.36-1.55)	
SS score 3 basal	1.17(0.89-2.28)	1.00(0.79-1.27)	0.172
SS score 3 first day	NA	1.16(0.87-1.56)	
SS score 3 second day	NA	1.19(0.89-1.49)	
EF, %	64±7	47±8	<0.001
LVM, g/m ²	87.3(74.6)	94.4(76.7)	0.044

Coronary artery disease / Acute coronary syndrome

PP-168

Predictive values of inflammatory cell ratios for complexity of coronary artery disease in patients with acute coronary syndromes: A retrospective study

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Background and Aim: The aim of this study was to investigate the relationship between neutrophil to lymphocyte ratio (NLR) platelet to lymphocyte ratio (PLR), main platelet volume to lymphocyte ratio (MPVLR), main platelet volume to platelet ratio (MPVPR) and the complexity of coronary artery disease (CCAD) in patients with acute coronary syndromes (ACS) using the SYNTAX score (SS) algorithm.

Methods: A total of 599 patients with ACS undergoing coronary arteriography were enrolled and divided into three groups according to their SS: low syntax score group ≤22 (low-SSG); intermediate syntax score group ≥23 and ≤32 (in-SSG); and high syntax score group ≥33 (high-SSG). Routine complete blood count parameters were analyzed at hospital admission.

Results: Except for NLR and MPVPR values, significant difference was found between the three groups in terms of PLR and MPVLR values (p=0.007 and p=0.029) (Table 1). Correlation analysis showed that PLR and MPVLR were positive correlated with CCAD (r=0.095, p=0.018 and r=0.112, p=0.005, respectively) (Table 2). On multivariate logistic regression analysis, MPVLR was not an independent predictor of CCAD, whereas PLR was found to be a weak independent predictor of CCAD [odds ratio=1.003 (1.001-1.006), p=0.021] (Table 3). ROC analysis showed that PLR had low sensitivity (56.2%) and specificity (51.6%) for prediction of CCAD (Figure 1).

Conclusions: Our study showed that NLR, MPVPR, MPVLR were not independent predictors of CCAD in patients with ACS. PLR had such a weak relationship with CCAD that it cannot be used for prediction of CCAD in these patients.

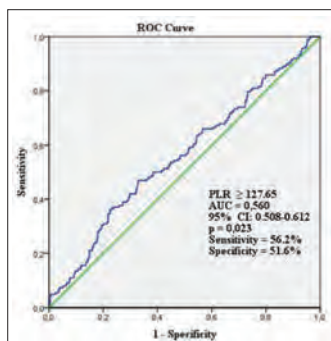


Figure 1. Receiver operating characteristics (ROC) curves for the platelet to lymphocyte ratio (PLR) value in the prediction of intermediate and high SYNTAX Score using SS = 22 cut-off value. AUC, area under the curve; CI, confidence interval.

Table 1. Complete blood count values and inflammatory cell ratios of the three groups

	Low-SS group (n=436)	In-SS group (n=127)	High-SS group (n=36)	p
WBC (K/uL)	8.97 ± 2.96	1.14 ± 2.5	8.46 ± 2.45	0.596
NC (K/uL)	6.07 ± 2.66	6.15 ± 2.84	5.77 ± 2.26	0.758
PLT (K/uL)	2.08 ± 1.17	1.96 ± 0.90	1.73 ± 0.87	0.139
MPV (fL)	9.97 ± 1.48	10.29 ± 1.54	9.86 ± 1.28	0.085
NLR	242.75 ± 70.37	255.87 ± 87.13	245.67 ± 82.67	0.218
PLR	142.87 ± 79.76	163.23 ± 111.55	4.63 ± 3.91	0.179
MPVLR	6.04 ± 3.40	6.70 ± 4.06	7.36 ± 3.89	0.029
MPVPR (%)	4.52 ± 1.84	4.44 ± 1.61	4.43 ± 1.45	0.881
CRP (mg/dL)	1.14 ± 2.5	1.22 ± 1.57	1.5 ± 2.26	<0.001

CRP, C-reactive proteins; LC, lymphocyte count; MPV, main platelet volume; MPVLR, main platelet volume to lymphocyte ratio; MPVPR, main platelet volume to platelet ratio; NC, neutrophil count; NLR, neutrophil to lymphocyte ratio; p, p value; PLR, platelet to lymphocyte ratio; PLT, platelet; WBC, white blood count.

Table 2. Correlation analysis between complete blood count values, inflammatory cell ratios and SYNTAX score of the patients

	r	p
WBC	-0.019	0.629
NC	0.007	0.860
LC	-0.056	0.159
PC	0.033	0.411
MPV	0.094	0.019
NLR	0.060	0.131
PLR	0.095	0.018
MPVLR	0.112	0.005
MPVPR	0.022	0.587
CRP	0.031	0.440

Table 3. Independent predictors of high SYNTAX score in acute coronary artery syndrome

	Univariate regression analysis		Multivariate regression analysis	
	β (95% CI)	p value	OR (95% CI)	p value
NLR	0.159 (-0.048-0.365)	0.131	-	-
PLR	0.009 (0.002-0.016)	0.018	1.003 (1.001-1.006)	0.021
MPVLR	0.267 (0.081-0.453)	0.005	1.070 (0.918-1.065)	0.761
MPVPR	10.492 (-27.391-48.375)	0.587	-	-

Coronary artery disease / Acute coronary syndrome

PP-169

Serum concentrations of PDGFRB and BDNF in patients with cardiac syndrome

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Background and Aim: Coronary microvascular dysfunction is reported to play a major role in the pathogenesis of cardiac syndrome X (CSX). The research about the biochemical pathways of coronary microvascular dysfunction still continues. Platelet-derived growth factor receptor B (PDGFRB) and brain-derived neurotrophic factor (BDNF) have been associated with cardiovascular diseases. We aim to investigate the role of these cardiac biomarkers in CSX.

Methods: Ninety-one patients median age 56 (40-79); 55 women with CSX and sixty-one individuals median age 52 (38-76); 32 women as control group were included into the study. Serum concentrations of PDGFRB, BDNF, uric acid and high sensitive C-reactive protein (hs-CRP) were measured on the blood samples.

Results: PDGFRB (2.82 ng/ml; IQR, 0.57e7.79 ng/ml vs 2.27 ng/ml; IQR, 0.41e7.16 ng/ml, p<0.0005) and BDNF (2.41 ng/ml; IQR, 0.97e7.97 ng/ml vs 1.92 ng/ml; IQR, 1.07e6.67 ng/ml, p=0.023) concentrations were significantly higher in patients with CSX compared to controls (Figure 1). Concentrations of PDGFRB correlated positively with low density lipoprotein (LDL) (r=0.18, p=0.02), and BDNF (r=0.47, p<0.01). Also, uric acid concentrations correlated positively with hs-CRP (r=0.17, p=0.03). Additionally, concentrations of PDGFRB, BDNF, hs-CRP, LDL, uric acid and with gender, hypertension and diabetes mellitus as potential confounders of coronary artery disease were performed stepwise logistic regression analysis. According to logistic regression analysis, uric acid, hs CRP, PDGFRB values were found out as the independent predictors of the CSX.

Conclusions: These findings suggest that there could be an association between CSX patients and high concentrations of PDGFRB and BDNF. Larger studies are needed to explore the mechanism underlying this relation.

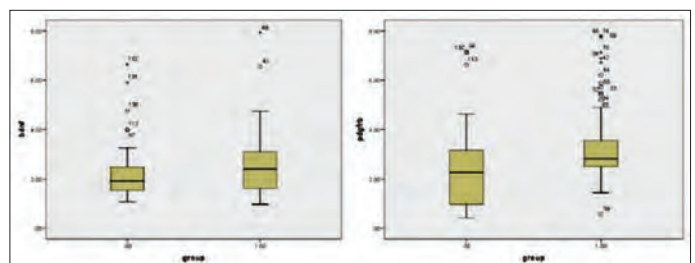


Figure 1. Serum BDNF and PDGFRB concentrations in the patients with control and CSX group.

Coronary artery disease / Acute coronary syndrome

PP-170

The no-reflow predictive role of aVR derivation in patients with acute anterior myocardial infarction who underwent primary percutaneous intervention

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Background and Aim: Acute transmural ischemia due to left anterior descending artery (LAD) occlusion changes R and Q waves in aVR due to depressed intramyocardial activation. We investigated the prognostic value of R and Q waves in aVR derivation in patients with first acute anterior myocardial infarction (AAMI) treated with primary percutaneous coronary intervention (PPCI).

Methods: We evaluated the no-reflow predictive value of R and Q waves in aVR derivation on 438 patients with first AAMI. Patients were divided into two as no-reflow (n=45) and control (n=393) groups according to post-PPCI no-reflow status.

Results: The patients in the no-reflow group had significantly higher frequency of Q wave in (-)aVR derivation on admission electrocardiography (ECG) compared with patients in the control group. (p<0.001). Multivariable analysis revealed higher rates of no-reflow for patients with chronic renal failure (OR 9.07, 95% CI 2.94–27.96, p<0.001), with higher onset-to-door time (OR 1.17 95% CI 1.06–1.30, p=0.002), with Killip≥3 (OR 3.45, 95% CI 1.09–12.33, p=0.048) and with lower ejection fraction (OR 0.92, 95% CI 0.87–0.98, p=0.022), with higher frequency of initial Q wave in (-)aVR derivation (OR 7.39 95% CI 3.65–24.16, p<0.001).

Conclusions: Q wave in (-)aVR in admission ECG in patients with first AAMI treated with PPCI may have a role as an independent predictive marker of no-reflow.

Table 1.

	Control, n=393	No-reflow, n=45	p value
Age,y	57 ± 12	63 ± 15	0.001
Male gender	338 (86.0)	36 (80.0)	0.280
Body mass index	27.9 ± 4.2	28.7 ± 5.0	0.782
Hypertension	159 (40.5)	23 (51.1)	0.170
Diabetes Mellitus	88 (22.4)	17 (37.8)	0.123
Hyperlipidemia	50 (12.7)	6 (13.3)	0.907
Current smoker	298 (75.8)	31 (68.9)	0.308
Chronic Renal Failure	34 (8.7)	12 (26.7)	<0.001
Onset-to-door time, hours	3.26 ± 2.96	4.91 ± 3.58	<0.001
Killip≥3	27 (6.9)	11 (24.4)	<0.001
At admission Systolic blood pressure, mm Hg	129.3 ± 28.0	125.2 ± 34.9	0.220
At admission Diastolic blood pressure, mm Hg	77.8 ± 17.8	72.0 ± 21.1	0.018
PTCA	319 (81.2)	41 (91.1)	0.099
Balloon diameter	1.79 ± 0.86	1.99 ± 0.78	0.308
Balloon length	12.3 ± 5.8	14.3 ± 5.2	0.011
Balloon pressure, atm	11.8 ± 5.5	13.1 ± 5.1	0.313
Glucose, (mg/dL)	167.0 ± 92.0	189.2 ± 76.7	0.006
Blood urea nitrogen, (mg/dL)	14.8 ± 9.2	20.8 ± 15.2	0.003
Creatinine, (mg/dL)	0.96 ± 1.01	0.90 ± 0.23	0.013
CRP, (mg/dL)	3.74 ± 4.91	5.63 ± 6.73	0.025
Troponin I, ng/mL	22.5 ± 20.8	15.3 ± 15.5	0.352
LVEF, %	40.2 ± 7.3	35.3 ± 7.2	<0.001
In-hospital mortality	32 (8.1)	10 (22.2)	0.002
ST elevation on admission, aVR	77 (19.6)	15 (33.3)	0.032
ST depression on admission, aVR	102 (26.0)	9 (20.0)	0.587
No ST segment change on admission, aVR	224 (57.0)	21 (46.7)	0.186
Initial Q wave presence, (-) aVR	57 (14.5)	30 (66.7)	<0.001
Q wave amplitude in (-)aVR, mV	0.02 ± 0.00	0.10 ± 0.01	<0.001
R wave amplitude in (-)aVR, mV	0.55 ± 0.01	0.60 ± 0.04	0.135

Table 2.

Multivariate analysis	P value	OR (95% CI)
Chronic renal failure	<0.001	9.07 (2.94 – 27.96)
Onset-to-door time	0.002	1.17 (1.06 – 1.30)
Killip≥3	0.048	3.45 (1.09 – 12.33)
Ejection fraction	0.022	0.92 (0.87 – 0.98)
Initial Q wave presence, (-) aVR	<0.001	7.39 (3.65 – 24.16)

Multivariate logistic regression analyses independent relationship between analyses between no-reflow and baseline, clinical, angiographic, laboratory and electrocardiography data. (Backward LR method) Variables were included if they were significantly different in univariate analyses (p<0.05). CI: Confidence interval.

Coronary artery disease / Acute coronary syndrome

PP-171

Predictors of in-hospital mortality in very elderly patients presented with acute coronary syndrome; A single center study

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Background and Aim: Acute coronary syndrome (ACS) becomes more frequent within elderly population, due to increased life expectancy. In this trial, our aim was to determine clinical and laboratory factors related to in-hospital mortality in patients over 80 years of age presented with ACS.

Methods: Total 171 patients over 80 years old and hospitalized due to the diagnosis of ACS were enrolled in this trial. Patients demographic, clinic features and laboratory data were screened from hospital data retrospectively.

Results: During follow-up 19 of 171 patients (11.1%) died. There were more patients with ST-segment elevation myocardial infarction (STEMI) presentation in the dying group [14 (73.7%) vs 31 (20.5%), p<0.001]. Patients who died during hospital follow-up had higher peak troponin [3.1 (7.2) ng/ml vs 0.3 (1.6) ng/ml, p<0.001] and CK-MB levels [96.7 (194) ng/ml vs 10.9 (36.2), p<0.001]. According to this; high GRACE risk score [OR:1.074, 95% CI (1.039-1.110), p<0.001], ejection fraction ≤40% [OR:8.113, 95% CI (1.101-59.773), p=0.040] and use of ACEI/ARB [OR:0.075, 95% CI (0.006-0.995), p=0.049] were significantly associated with in-hospital mortality in patients presented with ACS over 80 years.

Conclusions: Presentation with high GRACE risk score, not use of ACEI/ARB and low ejection fraction at admission were associated with in-hospital mortality in ACS patients over 80 years old.

Coronary artery disease / Acute coronary syndrome

PP-172

Another harmful effect of passive smoking: Aortic stiffness

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Background and Aim: Exposure to passive cigarette smoke is a serious environmental risk increasing the risk of heart diseases by 30%, and it is strongly associated with atherosclerosis, coronary artery disease, cardiovascular morbidity and mortality. In the present study, we evaluated the effects of acute exposure to passive cigarette smoke on blood gas and hemodynamic parameters, mainly on peripheral arterial stiffness, in healthy young men and women without active and passive smoking habits.

Methods: 20 male and 20 female participants without active smoking habit or passive exposure to cigarette smoking were included in the study. Transthoracic echocardiographic imaging was performed for all participants. Carotid-femoral pulse wave velocity (PWV) measurements, systolic and diastolic blood pressures, heart rates, venous blood carboxyhemoglobin and lactate concentrations and arterial pulse O2 saturations were obtained at baseline and just after passive cigarette-smoke exposure.

Results: All laboratory parameters were within normal limits. Heart rate, systolic and diastolic blood pressure, PWV, blood gas carboxyhemoglobin and lactate levels were significantly increased (p<0.001) in the total group, while a significant decrease was detected in arterial pulse O2 saturation (p<0.001) after exposure to passive smoking. There was a positive and significant association between post passive cigarette smoke exposure PWV and baseline uric acid, and between post-procedure carboxyhemoglobin and lactate levels. When the increase in PWV values was compared between male and female participants, a more profound increase was noted in PWV values of women compared to men following exposure to passive cigarette smoke.

Conclusions: Arterial stiffness, which is an important risk factor for cardiovascular diseases showing the disruption in endothelial function increases in acute exposure, similar to chronic exposure to passive cigarette smoke. This increase in arterial stiffness has been found to be significantly higher in women following acute exposure to passive cigarette smoke.

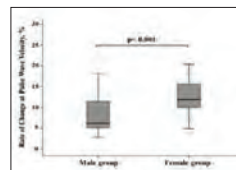


Figure 1. The association between gender and changes in PWV values following exposure to passive cigarette smoke.

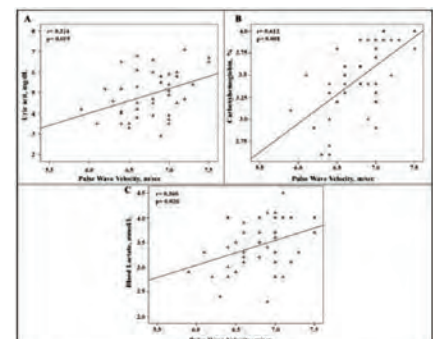


Figure 2. The association between PWV values and uric acid, carboxyhemoglobin and lactate levels following exposure to passive cigarette smoke.

Coronary artery disease / Acute coronary syndrome

PP-173

The relationship between serum rheumatoid factor level and no-reflow phenomenon in patients with acute ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention

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Background and Aim: This study aimed to evaluate the relationship between serum rheumatoid factor (RF) levels and no-reflow phenomenon in patients with acute ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI).

Methods: This single-center, cross-sectional study included a total of 318 consecutive patients who were diagnosed with STEMI and underwent primary PCI within 12 h of the onset of symptoms. Baseline serum RF levels of all patients were measured. The diagnosis of no-reflow phenomenon was defined as a flow of TIMI II or less without the presence of dissection, mechanical obstruction, significant residual stenosis or other plausible causes. The patients were divided into reflow group (n=283) and no-reflow group (n=46) regarding the angiographic features of thrombolysis in myocardial infarction (TIMI) flow of the infarct-related artery.

Results: No-reflow phenomenon was observed in 13.8% of the patients. Median RF level was significantly higher in no-reflow group than in reflow group (18.5 (7.0-27.6) vs. 8.0 (4.6-50.8), p<0.001). Forward conditional logistic regression analysis demonstrated that body mass index (OR=0.845, 95% CI: 0.765 to 0.933, p=0.001), diabetes mellitus (OR=5.257, 95% CI: 1.124 to 24.587, p=0.035), baseline RF level (OR=1.198, 95% CI: 1.108 to 1.295, p<0.001) and SYNTAX score I (OR=1.065, 95% CI: 1.025 to 1.107, p=0.001) were the independent predictors of no-reflow.

Conclusions: Baseline serum RF concentrations are independently associated with no-reflow phenomenon in patients undergoing primary PCI for acute STEMI.

Coronary artery disease / Acute coronary syndrome

PP-174

Impaired oscillometric arterial stiffness parameters in patients with coronary artery ectasia

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Background and Aim: We aimed to investigate the oscillometric measurements of elastic properties of the aorta in patients with isolated coronary artery ectasia (CAE).

Methods: This study included 137 patients (92 men and 45 women; mean age: 60.8±11.7 years) who underwent coronary angiography for the investigation of ischemic heart disease. The patients were divided into three groups; the first group consisted of 51 patients with CAE, the second group consisted of 36 patients with coronary artery disease (CAD), and the third group consisted of 50 patients with normal coronary arteries. Aortic stiffness (AS) measurements, including pulse wave velocity (PWV) and augmentation index (AIx), were measured by the oscillometric method.

Results: Mean PWV was significantly higher in the CAE group as compared with the CAD and control groups (9.1±2.3 vs. 8.2±1.3 and 8.0±1.6, p=0.008), whereas median AIx was significantly lower in the CAE group as compared with the CAD and control groups (10.0 (-3.0-63.0) vs. 15.5 (-2.0-57.0) and 21.5 (-1.0-45.0), p=0.010). Multinomial logistic regression analysis demonstrated that gender, hypertension, high-density lipoprotein cholesterol level, PWV and AIx were independently associated with CAE.

Conclusions: The oscillometric elastic properties of the aorta, including PWV and AIx, are impaired in patients with CAE.

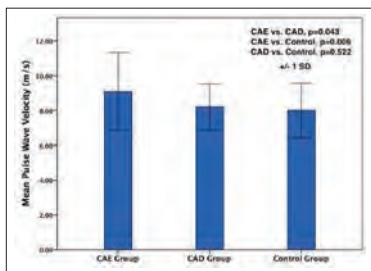


Figure 1. Comparison of pulse wave velocity levels of the patients. Mean pulse wave velocity was significantly higher in coronary artery ectasia group as compared with coronary artery disease and control groups. CAE: coronary artery ectasia, CAD: Coronary artery disease, SD: Standard deviation.

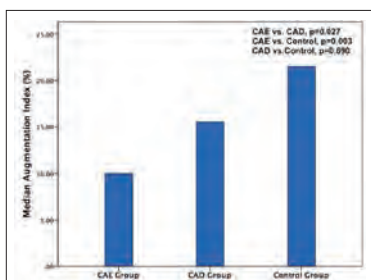


Figure 2. Comparison of augmentation index levels of the patients. Median augmentation index was significantly lower in coronary artery ectasia group as compared with coronary artery disease and control groups. CAE: coronary artery ectasia, CAD: Coronary artery disease.

Coronary artery disease / Acute coronary syndrome

PP-175

Increased renal cortical stiffness is associated with coronary artery disease severity in patients with acute coronary syndrome

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Background and Aim: Atherosclerosis is the main etiological factor in acute coronary syndromes (ACS). Kidneys have a lot of arterial vascular structure and they get affected by atherosclerosis like the coronary arteries. Renal shear wave elastography (SWE) is an ultrasonographic method which gives reliable information about renal parenchymal status (Figure 1a and 1b). In this study, we investigated the relationship between SWE findings and atherosclerosis severity.

Methods: Renal cortical stiffness (rCS) evaluated by SWE, renal resistive index (RRI), renal pulsatility index (RPI), and acceleration time (AT) were calculated. Mean Syntax score (SS) was calculated. Patients having SS lower than mean value were named as low risk (LR), equal or greater patients were named as high risk group (HR).

Results: Our study consisted of 132 patients. Low risk group (LR) included 76 patients (mean age 60.7±12.9 years), and HR had 56 patients (mean age 63.4±11.9 years). There was no significant difference in demographic variables between groups (Table 1). In the laboratory value comparison, creatinine (p=0.022) and hs-CRP (p=0.001) were significantly higher in HR. This group had also significantly lower GFR value (p=0.001), other variables were similar (Table 1). High risk group had significantly higher rCS value (p=0.006) in the echocardiographic and ultrasonographic evaluation (Table 2). Angiographic findings comparison revealed that HR had significantly higher frequency of 2 vessel, 3 vessel, SS, and cSS values, LR had significantly higher frequency of 1 vessel (p<0.001 for all, Table 2). Binomial logistic regression analysis was performed with statistically significant variables (p<0.05) of the univariate analysis. hs-CRP (OR:1.220, 95% CI:1.029-1.445, p=0.022), GFR (OR:0.967, 95% CI:0.943-0.992, p=0.009) and rCS (OR:1.316, 95% CI:1.075-1.611, p=0.008) were found independent predictors for HR. The cut-off value of rCS in the ROC analysis was 4.43 for the prediction of HR (sensitivity: 60.7%, specificity: 57.9%). The area under the curve was 0.642 (p=0.006) (Figure 2).

Conclusions: There is a close relation between CAD severity and rCS. Increased rCS values amplifies the possibility of having high risk CAD.

Table 1. Comparison of patients demographic and laboratory findings

	Low risk patients (n=76)	High risk patients (n=56)	p
Age (years)	60.7 ± 12.9	63.4 ± 11.9	0.194
Male gender, n (%)	59 (77.6)	46 (82.1)	0.525
Systolic blood pressure (mmHg)	126.2 ± 11.0	126.9 ± 12.5	0.723
Diastolic blood pressure (mmHg)	83.9 ± 8.6	85.2 ± 9.6	0.393
Heart rate (beat / minute)	77.5 ± 11.2	78.9 ± 10.1	0.445
Body mass index (kg / m2)	28.9 ± 2.4	29.5 ± 2.3	0.153
Smoking, n (%)	24 (31.6)	22 (39.3)	0.358
Diabetes, n (%)	15 (19.7)	19 (33.9)	0.065
Hypertension, n (%)	30 (39.5)	28 (50.0)	0.228
Hypertriglyceridemia, n (%)	12 (15.8)	16 (28.6)	0.076
White blood cell (µL)	10.7 ± 2.9	1.3 ± 3.8	0.333
Hemoglobin (mg/dL)	13.4 ± 1.7	13.0 ± 2.3	0.18
Glucose (mg/dl)	145.1 ± 67.1	159.4 ± 63.6	0.23
Blood urea nitrogen (mg/dL)	36.1 ± 9.9	39.4 ± 11.3	0.08
Creatinine (mg/dL)	0.85 ± 0.11	0.91 ± 0.16	0.022
Sodium (mmol/L)	137.3 ± 2.1	137.5 ± 2.0	0.62
Potassium (mmol/L)	4.3 ± 0.7	4.2 ± 0.7	0.68
Uric acid (mg/dL)	5.6 ± 1.1	5.2 ± 1.6	0.248
Total cholesterol (mg/dL)	188.8 ± 61.1	176.7 ± 21.1	0.263
LDL cholesterol (mg/dL)	113.7 ± 39.1	107.7 ± 29.5	0.473
HDL cholesterol (mg/dL)	39.7 ± 6.5	42.9 ± 6.9	0.032
Triglyceride (mg/dL)	156.4 ± 135.1	131.5 ± 61.3	0.33
Hs-TnT (ng/mL)	11.1 ± 19.4	108.7 ± 15.7	0.437
CK-MB (ng/mL)	11.5 ± 7.4	12.0 ± 9.3	0.741
Hs-CRP (mg/L)	1.3 ± 2.9	4.1 ± 4.8	0.001
CrCl (ml/dk)	111.3 ± 16.9	103.8 ± 18.2	0.016
GFR (ml/dk/m2)	68.9 ± 19.4	58.1 ± 17.7	0.001

LDL:low density lipoprotein,HDL:high density lipoprotein,CK-MB:creatinin kinase myocardial band, CrCl:Creatinine clearance, GFR: Glomerular filtration rate, Hs-CRP:High sensitive C reactive protein, Hs-TnT:High sensitive troponin T.

Table 2. Comparison of patients echocardiographic, ultrasound and angiographic findings

	Low risk patients n=76	High risk patients n=56	p
LV ejection fraction (%)	50.8 ± 5.6	48.9 ± 6.2	0.069
RRI (n)	0.73 ± 0.07	0.76 ± 0.08	0.115
RPI (n)	1.85 ± 0.64	1.98 ± 0.64	0.233
AT (ms)	96.4 ± 42.4	107.4 ± 45.3	0.151
rCS (kPa)	4.58 ± 2.09	5.91 ± 3.07	0.006
LMCA, n (%)	0 (0)	2 (3.6)	0.178
1 vessel, n (%)	72 (94.7)	11 (19.6)	<0.001
2 vessel, n (%)	4 (5.3)	25 (44.6)	<0.001
3 vessel, n (%)	0 (0)	20 (35.7)	<0.001
SS (n)	9.7 ± 2.1	15.7 ± 2.4	<0.001
cSS (n)	15.4 ± 4.5	24.3 ± 6.6	<0.001

AT:Acceleration time, LV:left ventricular, RRI:Renal resistive index, RPI:Renal pulsatility index, rCS: Renal cortical stiffness, LMCA:left main coronary artery, SS:Syntax score, cSS: Clinical syntax score.

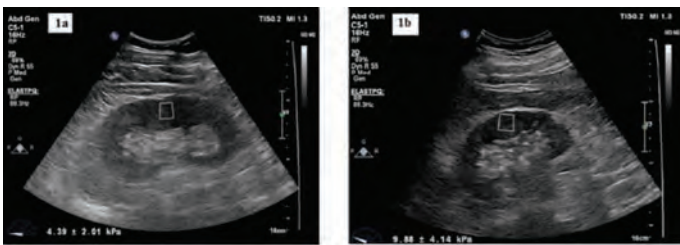


Figure 1. (a) Demonstrated of patient with normal renal shear wave elastography (cortical stiffness: 4.39 kPa). (b) Demonstrated of patient with increased renal shear wave elastography (cortical stiffness: 9.88 kPa).

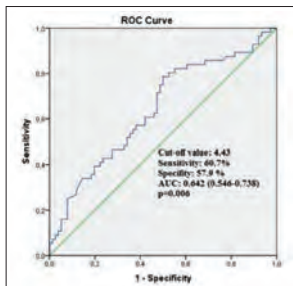


Figure 2. ROC curve analyses to determine predictive value of renal cortical stiffness for patients with severity coronary atherosclerosis.

Coronary artery disease / Acute coronary syndrome

PP-176

Monocyte to high-density lipoprotein ratio predicts contrast induced nephropathy in patients with acute coronary syndrome

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Background and Aim: Contrast-induced nephropathy (CIN) is associated with worse prognosis in patients with acute coronary syndrome (ACS). Early identification and intervention for patients with a high risk of CIN are very important to improve clinical outcomes. Inflammation plays an important role in the development of CIN in the setting of ACS. The monocyte to high-density lipoprotein (HDL) ratio (MHR) is a novel inflammatory marker. Bleeding is also associated with worse prognosis in such patients. We aimed to investigate whether the preprocedural MHR had a predictive role for CIN development in such patients. In addition, using the thrombolysis in myocardial infarction (TIMI) classification, we aimed to assess whether there was any relationship between bleeding and CIN.

Methods: A total of 647 patients (496 males; age: 63.3±12.7 years) with ACS who underwent percutaneous coronary intervention (PCI) were included in the study. Venous blood samples were drawn from antecubital veins immediately after the ECG recordings. The MHR was calculated by dividing the monocyte count to the HDL-C level using the same blood samples obtained before PCI. Patients were followed prospectively for 6 months for the development of overall mortality.

Results: Seventy patients (10.8%) had developed CIN. The patients with CIN were older, and they were more likely to have diabetes mellitus, new-onset atrial fibrillation and Killip class >2 on admission. The rate of triple vessel disease and contrast volume were higher in the patients with CIN. Left ventricle ejection fraction and estimated glomerular filtration rate (eGFR) were lower, and preprocedural MHR was higher in patients with CIN. TIMI bleeding and six-month overall mortality were higher in patients with CIN (Table 1). In binary logistic regression analysis, age, diabetes mellitus, contrast volume, eGFR, and preprocedural MHR were independent predictors for CIN (Table 2).

Conclusions: The development of CIN in the course of ACS is associated with increased mortality. Preprocedural MHR may be used as a simple marker of CIN. It may help early identification of patients with ACS at high risk of CIN and to take protective measures such as reducing the amount of contrast material.

Table 1. Baseline demographic, clinical, laboratory features of the patients with and without contrast-induced nephropathy

	CIN (-) (n=577)	CIN (+) (n=70)	P value
Age (years)	62.0 (54.0-72.0)	72.5 (66.0-79.0)	<0.001
Female sex (n,%)	132 (22.9)	19 (27.1)	0.426
Diabetes mellitus (n,%)	209 (36.2)	38 (54.3)	0.003
Known CAD (n,%)	181 (31.4)	28 (40.0)	0.145
Killip III/IV (n,%)	41 (7.1)	15 (21.4)	<0.001
New-onset AF (n,%)	56 (9.7)	17 (24.3)	<0.001
LV EF (%)	47.0 (36.0-56.0)	36.5 (27.7-50.0)	<0.001
Triple vessel disease (n,%)	166 (28.8)	36 (51.4)	<0.001
Contrast volume (ml)	170.0 (162.0-175.0)	175.0 (165.0-185.0)	<0.001
TIMI Bleeding (n,%)	25 (4.3)	8 (11.4)	0.019
eGFR (mL/min/1.73m ²)	79.7 (64.4-94.6)	61.1 (48.2-72.8)	<0.001
Preprocedural MHR	14.33 (10.33-19.12)	19.39 (14.99-25.00)	<0.001
Peak troponin-T (ng/mL)	2.34 (0.54-5.32)	3.32 (1.09-6.85)	0.108
Six-month overall mortality (n,%)	48 (8.3)	26 (37.1)	<0.001

AF, atrial fibrillation; CAD, coronary artery disease; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; MHR, monocyte to high-density lipoprotein ratio; TIMI, thrombolysis in myocardial infarction.

Table 2. Independent predictors of contrast-induced nephropathy

	OR	95% CI	P value
Age	1.062	1.031-1.093	<0.001
Diabetes mellitus	2.177	1.218-3.889	0.009
Triple vessel disease	1.634	0.915-2.917	0.097
TIMI bleeding	2.408	0.899-6.449	0.080
Contrast volume	1.034	1.013-1.056	<0.001
eGFR	0.982	0.969-0.996	0.010
Preprocedural MHR	1.085	1.051-1.121	<0.001

CI: Confidence interval; eGFR, estimated glomerular filtration rate; MHR, monocyte to high-density lipoprotein ratio; OR: Odds ratio; TIMI, thrombolysis in myocardial infarction.

Coronary artery disease / Acute coronary syndrome

PP-177

Contribution of QTc extension and QT dispersion in exercise ECG to diagnosis critical coronary artery disease

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Background and Aim: Coronary artery disease (CAD) is becoming a growing social problem day by day. Exercise electrocardiogram (ECG) is a relatively inexpensive non-invasive method often used in the diagnosis of CAD, but its sensitivity is relatively low compared to other tests. QT interval in superficial ECG is affected in many cardiovascular diseases. Our aim in this study was to investigate the contribution of QT and QT dispersion (QT disp) changes during exercise ECG in order to diagnose CAD.

Methods: We included 192 patients who had stable angina pectoris and applied cardiology polyclinic with positive ECG test. Coronary angiography was performed and demographic, laboratory data were recorded on all of these patients. QT, QTc (corrected QT) and QT dispersion (disp) values had been recorded before the exercise ECG started, the peak exercise and the second minute of resting. Coronary angiography images of all patients were examined, Syntax Score (SS) was calculated. Patients were divided into two groups: patients with SS 0, group 1 (no critical CAD), and patients with SS ≥1, group 2 (with critical CAD).

Results: There were 126 patients (age 53.8±9.6 years) in group 1 and 66 patients (age 57.5±9.1 years, 34.3%) in group 2 patients. No statistically significant difference was found between the demographic data of the groups (Table 1). Laboratory data were similar between the two groups (Table 2). When exercise ECG test data were compared, peak QT disp and resting QTc increased significantly in group 2 (p<0.001 for all). Heart rate recovery (p<0.001), target heart rate (p=0.012), base line systolic (p=0.005) and diastolic blood pressure (p<0.001) were significantly high in group 1 (table 3). In binominal logistic regression analysis, resting QTc (OR: 1.051, 95% CI: 1.031-1.071, p<0.001) and resting QT disp (OR: 1.117, 95% CI: 1.066-1.170, p<0.0001) were identified as independent markers for critical CAD (Table 4). In the ROC analysis with independent markers, when the recovery QTc cut-off value was taken as 404 ms or the QT disp cut-off value was taken as 37 ms, the sensitivity for determining CAD was 90% (Figure 1, 2).

Conclusions: QT and QT dispersion measurements in patients with exercise ECG may contribute to the diagnosis of critical CAD and have been found to increase the sensitivity of the test significantly.

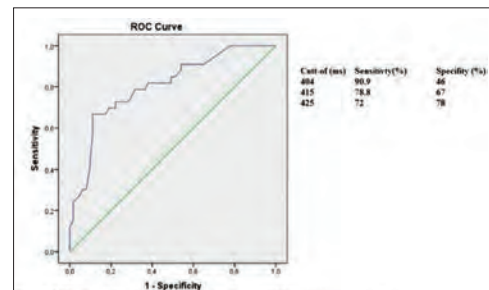


Figure 1. ROC analyses to determine sensitivity and specificity of recovery QTc for critical coronary artery disease.

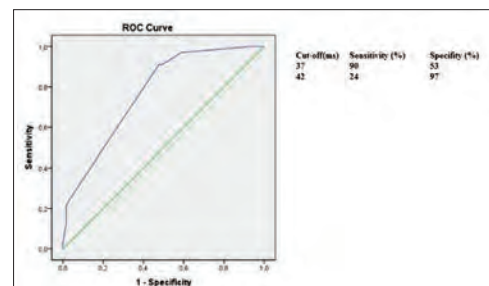


Figure 2. ROC analyses to determine sensitivity and specificity of QT dispersion for critical coronary artery disease.

Table 1. Comparison of demographic findings

	Group 1 n=126	Group 2 n=66	P
Age (years)	53.8±9.6	57.5±9.1	0.071
Male gender n (%)	74 (57.8)	40 (60.6)	0.859
Systolic blood pressure, (mmHg)	126.4±17.5	124.4±16.7	0.604
Diastolic blood pressure (mmHg)	82.5±8.4	83.4±7.9	0.607
BMI, (kg/m ²)	26.1±2.9	26.8±1.7	0.157
HT, n (%)	76 (60.3)	44 (66.7)	0.542
HPL, n (%)	8 (6.3)	8 (12.1)	0.331
DM, n (%)	54 (42.9)	34 (51.5)	0.419
Family history, n (%)	40 (31.7)	20 (30.3)	0.885
Smoking, n (%)	74 (58.7)	42 (63.6)	0.641

BMI: Body mass index, HT: hypertension, HPL: hiperlipidemia, DM: diabetes mellitus.

Table 2. Comparison of laboratory findings

	Group 1 n=126	Group 2 n=66	P
Glucose (mg/dl)	142.8 ± 72.5	166.3 ± 94.9	0.081
Urea (mg/dl)	30.1 ± 10.4	31.1 ± 8.5	0.564
Creatinine (mg/dl)	0.73 ± 0.14	0.74 ± 0.13	0.424
Sodium (mEq/L)	140.0 ± 2.5	139.4 ± 2.5	0.16
Potassium (mEq/L)	4.3 ± 0.3	4.3 ± 0.5	0.242
Uric acid (mg/dl)	5.4 ± 1.0	5.5 ± 0.9	0.418
Total cholesterol (mg/dl)	227.3 ± 61.4	232.4 ± 57.4	0.579
Triglycerides (mg/dl)	205.8 ± 112.3	224.0 ± 145.5	0.339
LDL cholesterol (mg/dl)	154.5 ± 46.1	151.3 ± 41.3	0.63
HDL cholesterol (mg/dl)	47.2 ± 7.5	46.8 ± 8.2	0.787
WBC (10 ³ /uL)	7.6 ± 2.0	8.1 ± 2.2	0.108
Hb (g/dl)	13.7 ± 1.6	13.5 ± 1.2	0.478
NLR	2.4 ± 1.1	2.3 ± 0.8	0.484

LDL: low-density lipoprotein, HDL: high-density lipoprotein, WBC: white blood cell, Hb: hemoglobin, NLR: neutrophil lymphocyte ratio

Table 3. Comparison of exercise ECG test findings

	Group 1 N=126	Group 2 N=66	P
Basal QTc	411.4±33.8	413.3±31.3	0.708
Peak QTc	421.6±30.9	429.6±37.9	0.117
Resting QTc	406.5±26.6	438.1±23.1	<0.001
Basal p disp.	23.6±14.9	23.9±13.3	0.866
Peak, p disp.	14.3±12.3	17.0±13.2	0.164
Resting p disp.	21.8±13.6	25.3±13.5	0.093
Basal QT disp.	34.4±13.6	35.3±10.7	0.657
Peak QT disp.	26.6±12.6	38.0±14.4	<0.001
Resting QT disp.	30.0±12.2	42.6±9.3	<0.001
Heart rate recovery	21.6±12.1	15.6±5.4	<0.001
Duration of exercise	5.9±2.4	6.1±2.5	0.725
Target heart rate	142.2±9.9	138.5±9.1	0.012
Max. heart rate	149.7±16.9	145.7±13.8	0.095
First min. heart rate	128.1±18.7	127.5±14.0	0.799
Basal systolic BP	133.8±14.4	127.5±15.5	0.005
Basal diastolic BP	82.6±5.9	78.0±9.5	<0.001

QTc: corrected QT, Disp: dispersion, Max: maximum, Min: minimum, BP: blood pressure

Table 4. Independent predictor for critical coronary artery disease

	Odds ratio	95% CI	p
Peak QT disp.	1.022	0.985-1.059	0.249
Heart rate recovery	0.955	0.896-1.017	0.15
Recovery QTc	1.051	1.031-1.071	<0.001
Recovery QT disp.	1.117	1.066-1.170	<0.001

LDL: low-density lipoprotein, HDL: high-density lipoprotein, WBC: white blood cell, Hb: hemoglobin, NLR: neutrophil lymphocyte ratio

Coronary artery disease / Acute coronary syndrome

PP-178

The relationship between QRS fragmentation and NT-proBNP levels in patients with acute coronary syndrome

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Background and Aim: Fragmented QRS (fQRS) is an important indicator of scar tissue and ventricular dysfunction. Increased levels of plasma natriuretic peptides have been found to be associated with infarct size and grade of left ventricular dysfunction. In the study, we aimed to evaluate the relationship between the presence of fQRS complexes and plasma level of N-terminal pro-brain natriuretic peptide (NT-proBNP) in patients with acute coronary syndrome (ACS).

Methods: This cross-sectional study included 511 patients with ST elevated myocardial infarction (STEMI) and 226 non-STEMI (NSTEMI). fQRS was defined as the presence of an additional R wave (R') or notching of the R wave or S wave, or the presence of R' >1 in two contiguous leads. NT-proBNP levels were obtained at 24 hours of admission to the hospital. Patients were divided into two groups according to the presence or absence of fQRS on electrocardiography (ECG). In addition, univariate and multivariate regression analyses were performed to determine the predictors of fQRS in each group.

Results: In the study population, 205 (40.1%) patients were found to have fQRS in their ECG. Plasma NT-proBNP level was significantly higher in fQRS (+) group (1555.8 [744.5-3874.0] vs 796.5 [279.0-1942.5] pg/mL, p<0.001). Plasma NT-proBNP (odds ratio: 2.0, p<0.001) and LVEF (odds ratio: 0.98, p=0.036) were found to be the independent predictors of fQRS formation in total population, plasma NT-proBNP level (odds ratio: 4.93, p<0.001) alone in NSTEMI patients, and LVEF (odds ratio: 0.95, p=0.002) alone in STEMI patients.

Conclusions: Our study demonstrates that fQRS (+) ACS patients have higher plasma NT-pro BNP levels. Additionally, in NSTEMI patients plasma NT-proBNP level and in STEMI patients LVEF are independent predictors of fQRS formation. The relationship between high NT-pro BNP level and fQRS in our study, has increased the importance of NT-pro BNP in patients with ACS.

Coronary artery disease / Acute coronary syndrome

PP-179

A rare cause of acute coronary syndromes in young adults: Myeloproliferative neoplasms: A case series

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Background and Aim: Acute coronary syndromes (ACSs) mostly occur in patients with traditional risk factors. Especially in young adults without major cardiovascular (CV) risk factors, one of the rare causes of ACSs is myeloproliferative neoplasms (MPNs).

Methods: We retrospectively collected data from 11 (9 men, 2 women, mean age 40.18±8.4) consecutive patients with the diagnosis of MPN presented with ACS. The data about the demographic characteristics of the study population, the type of MPN, clinical manifestations, the location of myocardial infarction (MI), coronary angiography (CAG) findings, CBC, other related findings, the treatment strategy before and after the diagnosis were analyzed.

Results: Six patients were diagnosed with PV, 4 patients with ET and one patient with PMF. JAK-2 mutation was found in nine patients. After presenting ACS, average time to diagnosis of MPN was 2.81 years. The mean age of the patients was 32.9±6 when they first suffered myocardial infarction. Six patients had no major CV risk factors. Ten patients had anterior MI, one patient had inferior MI. After the start of specific treatment for MPN, no recurrent thrombotic events were observed with a mean follow-up of 4±2.44 years.

Conclusions: In young adults presented with ACS, MPNs should be considered, especially in the absence of atherosclerotic coronary artery lesions. It is also important to pay attention to blood cell count abnormalities seen in intracoronary thrombotic events. Early diagnosis and treatment of MPNs is essential to prevent recurrence of thrombotic events and may reduce mortality and morbidity related to thrombotic complications.

Coronary artery disease / Acute coronary syndrome

PP-180

Myth or fact: Vitamin D and SYNTAX score

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Background and Aim: Growing evidence demonstrated that vitamin D deficiency is closely associated with hypertension, obesity, diabetes mellitus, and coronary artery disease. SYNTAX score (SS) is the angiographic scoring system and is commonly used to evaluate the severity and complexity of coronary artery disease. This study was conducted with the aim to evaluate the association between serum vitamin D level and SS.

Methods: The medical records of consecutive patients, who underwent coronary artery bypass graft surgery, were retrospectively reviewed. The study group consisted of 158 patients. Biochemical, clinical, echocardiographic parameters and SS were evaluated in all patients. The patients were divided into two groups according to SS (≥23: high, <23 low).

Results: High SYNTAX score (HSs) group is older and had higher body mass index, C-reactive protein (CRP), low-density lipoprotein, and fasting plasma glucose level. HSs group had lower high-density lipoprotein and vitamin D level. HSs group had a higher prevalence of diabetes mellitus (DM), hypertension (HT), hyperlipidemia (HL), and current smoking. On univariate analysis, age, HT, DM, HL, smoking, CRP, and serum vitamin D level were associated with higher SS. On multivariate analysis HT, DM, and HL were independent predictors for higher SS (OR 2.137; 95% CI: 1.468–2.935; p<0.001, OR 3.559; 95% CI: 2.763–5.927; p<0.001, OR 2.631; 95% CI: 1.529–3.438; p<0.001; respectively).

Conclusions: In our study, we have found that HT, DM, and HL were an independent predictor for higher SS. Serum vitD level was not found to be an independent predictor of higher SS.

Coronary artery disease / Acute coronary syndrome

PP-181

The co-existence of obstructive sleep apnea in patients with slow coronary flow using Berlin questionnaire study

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Background and Aim: Coronary slow-flow phenomenon (CSF) was defined as distal opacification of the coronary artery is delayed on angiography in the absence of significant coronary artery disease. Obstructive sleep apnea (OSA) is a prevalent condition commonly associated with obesity, hypertension, habitual snoring, and hypersomnolence. If untreated, OSA cause increased cardiovascular morbidity and mortality. Both OSA and coronary slow-flow phenomenon CSF have similar pathogenic mechanisms, such as chronic sympathetic activation, upregulation of inflammatory pathways, oxidative stress, and endothelial dysfunction. Two of the questionnaires (Berlin and STOP-Bang) were found to have a high sensitivity for OSA (proportion of patients with OSA who screen positive) and low specificity (proportion without OSA who screen negative). However, BQ is a brief and validated screening tool that identifies persons in the community who are at high risk for OSA. We examined the presence of OSA by using the Berlin questionnaire (BQ) in the patients diagnosed CSF after coronary angiography.

Methods: The prospective study included 4515 patients admitted angiography laboratory because of possible coronary artery disease in between April 2017 to May 2018. Of the patients, 316 patients were diagnosed CSF in coronary angiography. The patient with slow coronary flow were asked questions by using BQ. After then, 276 of the 316 patients were found high risk according to BQ.

Results: Of 4515 patients, 316 (6.9%) met the criteria for SCF. After BQ testing, 276 patients incorporated high-score BQ (Table 1). Study patients had 188 male (68%). The mean ages were 48.48±7.61 years. Body mass index (BMI, (kg/m²) in the 276 SCF patients were 31.30±3.68. Coronary TIMI frame count (CTFC) in SCF group were LAD 33.85±3.66, CX 33.71±4.56, and RCA 34.31±4.04. The BQ testing includes questions about snoring (category 1), daytime somnolence (category 2), and hypertension or BMI >30 kg/m² (category 3). Patients were scored as being at high-risk for OSA if they had a positive score on two or more categories. Of 276 high-risk patients, 155 (56.1%) had a positive score in category 1 of the BQ, 165 (59.7%) had a positive score in category 2, and 248 (89.8%) had a positive score in category 3.

Conclusions: CSF and OSA have similar pathogenic mechanisms and the patients with SCF pattern incorporated with OSA pathology. The evaluation in the patients with SCF should take account of the presence of OSA.

Table 1. Baseline characteristics of 276 CSF patients

Men/Total	188 (68%)		
Age (years)	48.48 ±7.61		
BMI (kg/m ²)	31.30 ±3.68		
Hypertension	118 (42.7%)		
CTFC scf group	LAD 33.85±3.66	CX 33.71±4.56	RCA 34.31±4.04
CTFC control group	LAD 23.83±1.80	CX 20.83±2.51	RCA 21.75±2.86 p<0.05

Coronary artery disease / Acute coronary syndrome

PP-182

Association of chromosome 9p21.3 region with increased risk of coronary artery disease in northern tanzanian patients

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Background and Aim: Coronary artery disease (CAD) is the main non-communicable conditions in sub-Saharan Africa (SSA) and now 9.2% of total death in the African region are caused by CAD. CAD is the leading cause of deaths in patients aged over 45 with prevalence being higher in the urban than the rural population. The association of genetics and environmental factors plays a major role in the pathogenesis of CAD. The genetic risk factors have not well studied. The 9p21.3 chromosomal region has been associated with CAD in many genome wide association studies (GWAS). Despite the importance of 9p21.3 region in CAD no genetic research has been conducted in SSA. This study aimed at conducting the first genetic study evaluating the polymorphisms in 9p21.3 region in Tanzanian CAD patients from different regions of Tanzania.

Methods: 90 patients from Northern region (age 63.10±11.25) and 45 patients from other regions (South, East, West and Central) (age 59.67±9.21) were included in the study. Biochemical measurements and genetic

studies were performed. DNA was obtained from peripheral blood with MagnaPure Compact. Genotyping was performed with the LightSNIP typing assay using QRT-PCR method.

Results: BMI, glucose, cholesterol, HDL, LDL and TG levels were significantly different in patients from Northern Tanzania compared with the other regions of Tanzania (p<0.05). The genotypic and allelic distributions of rs10757274A/G, rs10757278A/G and rs10811656C/T polymorphisms were significantly different in CAD patients from Northern Tanzania compared with the other regions (p<0.05, respectively). GG and AG genotypes of rs10757274, GG and AG genotypes of rs10757278 and TT and CT genotypes of rs10811656 were significantly associated with CAD in females from northern region (p<0.05).

Conclusions: rs10757274, rs10757278 and rs10811656 in 9p21.3 region might well be associated with the CAD risk in Northern Tanzania. Our findings also suggest a gender specific association of these polymorphisms with CAD in Northern patients. Further studies on larger samples are needed in order to assess the prevalence and major risk factors for development of CAD in SSA.

Coronary artery disease / Acute coronary syndrome

PP-183

Evaluation of the predictive value of echocardiographic aortic stiffness and distensibility for thromboembolism risk in patients with ST elevation myocardial infarction

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Background and Aim: The CHA2DS2-VASc (congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, previous stroke, vascular disease, age 65-74 years, female gender) score is used to estimate thromboembolic risk in atrial fibrillation (AF). Its relationship with aortic stiffness is unknown. The study aimed to evaluate the prognostic role and discriminative power of aortic stiffness in thromboembolic risk in patients with acute coronary syndrome.

Methods: A total 272 patient with ST Elevation myocardial infarction (116 females, 156 males, mean age 61±11.5 years) who admitted coronary care unit were divided into two groups according to CHA2DS2-VASc scores (a score of "0 and 1" was regarded as low risk, and "≥2" as high risk).

Results: 153 of 158 patients had high risk. Fasting glucose, peak CK-MB and creatinin levels were lower in low risk group than high risk group (Table 2). Aortic diameter, IVS, Left ventricle end systolic and diastolic diameters were higher in high risk group than low risk group (Table 3). Mean aortic strain and aortic distensibility were higher in low risk group than high risk group (14.1 ±4.0 vs. 9.1 ±3.3, p<0.001 and 7.0 ±3.3 vs. 3.5 ±2.4, p<0.001) However, aortic stiffness index and aortic elastic module were lower in low risk group than high risk group (3.5 ±1.4 vs. 6.3 ±2.9, p<0.001 and 351 ±185 vs. 746 ±377, p<0.001) (Table 4). While Aortic stiffness index and aortic elastic module were positively correlated with CHA2DS2-VASc scores, aortic strain and aortic distensibility were negatively correlated with CHA2DS2-VASc scores (Table 5). According to ROC analysis, value of 464 aortic elastic module value was found to be predictive of high risk of CHADS-VASc score with 78% sensitivity and 79% specificity (area under curve = 0.84, p<0.001, 95% CI=0.80-0.89) (Figure 1) (Table 6). According to multivariate logistic regression analysis, aortic elastic modul and peak CK-MB levels were independent predictors of high thromboembolic risk in terms of CHA2DS2-VASc score (OR 1.005, 95% CI 1.004-1.006 and OR 1.04, 95% CI 1.02-1.06), p<0.001) (Table 7).

Conclusions: Our findings suggest that aortic stiffness parameters measurement could provide additional information on assessing cardiovascular risk such as thromboembolic events, and individuals with increased EFT should receive more attention to reduce unfavorable cardiovascular risk factors and the development of future cardiovascular events. Abnormal vascular function may be a novel parameter for thromboembolic risk stratification in patients with ST elevation myocardial infarction. Aortic stiffness is significantly and independently associated with higher risk for cardiovascular morbidity and mortality in different diseases such as atherosclerosis, hypertension, and endstage renal disease.

Table 1. Demographic data of study population

Parameter	Low CHADS-VASc score (n=119)	High CHADS-VASc score (n=153)	p
Age, years	54.3± 8.2	67.5± 10.4	<0.001
Female gender, n (%)	14(11)	102(66)	<0.001
Hypertension, n (%)	22(18)	115(75)	<0.001
Diabetes mellitus, n (%)	13(10)	80(52)	<0.001
Hyperlipidemia, n (%)	20(16)	53(34)	.0001
Smoking, n (%)	75(63)	41(26)	<0.001
CAD n (%)	24(20)	73(47)	<0.001

Table 2. The clinical and echocardiographical parameters showing the significant correlation with CHADS-VASc score

Parameter	r	p
Age	0.675	<0.001
LA	0.436	<0.001
IVS	0.519	<0.001
LVEDD	0.357	<0.001
LVEDD	0.446	<0.001
EF	-0.453	<0.001
Aort diastole	0.621	<0.001
Fasting glucose, mg/dL	0.464	<0.001
Peak CK-MB	0.463	<0.001
LDH	0.491	<0.001
CHADS-VASc score	0.577	<0.001
Aortic strain (%)	-0.613	<0.001
Aortic distensibility (cm ² /dyn × 10 ⁻³)	-0.609	<0.001
aortic stiffness index	0.571	<0.001
aortic elastic modul	0.612	<0.001

Table 3. Predictors of CHADS-VASc risk classification in univariate and multivariate analysis

	OR	(95 % CI)	P value	OR	(95 % CI)	P value
Aortic strain (%)	0.706	0.64-0.77	<0.001			
Aortic distensibility (cm ² /dyn × 10 ⁻³)	0.630	0.55-0.71	<0.001			
aortic stiffness index	1.901	1.6-2.23	0.045			
aortic elastic modul	1.006	1.004-1.007	<0.001	1.005	1.004-1.006	<0.001
Peak CK-MB	1.05	1.04-1.07	<0.001	1.04	1.02-1.06	<0.001
Fasting glucose, mg/dL	1.02	1.01-1.03	<0.001			

Table 4. The laboratory findings of the study population

Parameter	Low CHADS-VASC score (n=119)	High CHADS-VASC score (n=153)	P
Hemoglobin, g/L	12.9±0.8	12.4±7	.481
Platelet count (x 103 /μL)	309± 24	320±244	.640
Fasting glucose, mg/dL	98± 32	168± 88	<0.001
HDL-cholesterol, mg/dL	41± 6	42± 5	.542
LDL-cholesterol, mg/dL	132± 92 132±19.961	132±19	.961
LDH	322±48 376±69 <0.001	376±69	<0.001
Peak CK-MB	100±15 119±21 <0.001	119±21	<0.001
Potassium	3,9±0,2	4,1±0,4	<0.001
Sodium	138±3	140±4	<0.001
AST	82±12	90±17	<0.001
ALT	44±8	38±10	<0.001
Creatinin	0.9±0.1	1.1±2.2	<0.001

Table 5. ROC analysis of aortic parameters

	AUC (95 % CI)	P value	Sensitivity (%)	Specificity (%)	P value
Aortic strain (%)	0.158 0.11-0.20	10,7	20	21	<0.001
Aortic distensibility(cm2/dyn × 10-3)	0.154 0.107-0.201	4,7	21	25	<0.001
Aortic stiffness index	0.816 0.76-0.86	4,4	75	75	0.045
Aortic elastic modulü	0.84 0.84-0.89	464	78	79	<0.001

Table 6. The echocardiographic findings of study population

Parameter	Low CHADS-VASC score (n=119)	High CHADS-VASC (n=153)	p
LVEF, %	48± 7.9 39± 11.3	39± 11.3	<0.001
Aort (end diastole)(mm)	27± 2.0	28± 1.9	<0.001
Aort (end systole) (mm)	33±27	30±1.8	0.301
LA (mm)	39± 31	47± 27	0.474
IVS (mm)	10± 0.8	11± 1.4	<0.001
LVPW (mm)	8± 0.6	9± 0.9	<0.001
LVESD (mm)	25± 2.0	26± 2.2	<0.001
LVESD (mm)	44± 3.9	48± 4.6	<0.001

Table 7. Aortic parameters of study population

Parameter	Low CHADS-VASC score (n=119)	High CHADS-VASC score (n=153)	P value
Aortic strain (%)	14.1±4.0	9.1± 3.3	<0.001
Aortic distensibility (cm2/dyn × 10-3)	7.0±3.3	3.5±2.4	<0.001
aortic stiffness index	3.5±1.4	6.3±2.9	<0.001
aortic elastic modulü	351±185	746±377	<0.001

Coronary artery disease / Acute coronary syndrome

PP-184

The association of platelet-to-lymphocyte ratio with acute stent thrombosis

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Background and Aim: Cardiovascular diseases are the leading causes of mortality in the world. Interventional methods used in the treatment of coronary artery disease have revolutionized the treatment of the disease. Balloon angioplasty and coronary stenting are two miraculous treatment methods of the disease. Acute stent thrombosis(ST) is a very serious and mortal complication of stent thrombosis. PLR, a novel inflammatory marker, has previously been shown to be associated with cardiac problems. In this study, we aimed to investigate the association of PLR with acute stent thrombosis.

Methods: 1300 patients (1263 ST- and 35 ST+) who underwent stent implantation between January 2013 and December 2013 in our hospital were included in the study. Demographic, clinical, angiographic and laboratory parameters of all participants were recorded.

Results: In the ST+ group hypertension, diabetes mellitus rates were higher, clopidogrel loading time was longer. The mean PLR value was significantly higher in the ST+ group as compared to ST- group (133.3±75.0 vs 110.1±47.0, p=0.005). In the multivariate analyses hypertension, diabetes mellitus, longer clopidogrel loading time and PLR was found to be independent predictors of acute stent thrombosis.

Conclusions: Our results demonstrated that PLR is an independent predictor of acute stent thrombosis.

Table 1. Multivariate logistic regression analysis to predicting the acute stent thrombosis

	Univariable OR (95% CI)	P value	Multivariable OR (95% CI)	P value
Diabetes Mellitus	2.954 (1.487-5.868)	0.002	4.045 (1.684-9.719)	0.002
Hypertension	5.062 (2.282-11.232)	<0.001	7.223 (2.725-19.150)	<0.001
Preprocedure LVEF	0.903 (0.875-0.932)	<0.001	0.969 (0.921-1.020)	0.224
Clopidogrel loading time	0.122 (0.037-0.401)	0.001	0.067 (0.019-0.237)	<0.001
Stent diameter	1.296 (0.659-2.550)	0.453	-	-
ACS or elective	0.292 (0.186-0.458)	<0.001	0.556 (0.299-1.033)	0.063
Stent length	0.992 (0.934-1.053)	0.784	-	-
PLR	1.008 (1.002-1.014)	0.005	1.008 (1.001-1.015)	0.018

Abbreviations: ACS, acute coronary syndrome; CI, confidence interval; LVEF, left ventricular ejection fraction; OR, odds ratio; PLR, platelet to lymphocyte ratio.

Coronary artery disease / Acute coronary syndrome

PP-185

May whole blood viscosity be a predictor of atherosclerosis?

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Background and Aim: Atherosclerosis is a prevalent cardiovascular disease characterized by inflammation and the plaque formation within arterial walls. Whole blood viscosity (WBV) is defined as intrinsic resistance of blood flow in vessels. It is shown that high WBV promotes endothelial shear stress, endothelial inflammation and vascular remodeling and leads atherosclerotic process. The aim of this study is to compare WBV levels in patients with microvascular angina (MVA), coronary artery disease (CAD) and normal population and to identify the relationship between WBV and inflammation markers in patients.

Methods: 573 patients were enrolled into the current study. The MVA group consisted of 189 subjects with anginal chest pain but a normal coronary angiography, the CAD group consisted of 203 subjects, and the control group consisted of 181 age and sex-matched individuals, without any sign of inducible ischaemia on myocardial perfusion scintigraphy or treadmill exercise test. WBV was calculated from hematocrit and plasma protein concentration at low shear rate (LSR) (0.5 s-1) and high shear rate (HSR) (208 s-1) by a validated equation (Table 1). **Results:** CAD and MVA patients had significantly higher WBV for LSR (64.2±33 and 55±26 vs 33.8±31; p<0.001) and HSR (17.4±1.5 and 16.8±1.2 vs 15.8±1.0; p<0.001) when compared to control group (Table 2). Correlation analysis revealed a significant relationship between the hsCRP and WBV for LSR (r=-0.556; p<0.001) and HSR (r=-0.562; p<0.001) in CAD group and LSR (r=0.475; p<0.001) and HSR (r=0.493; p<0.001) in MVA group.

Conclusions: Overall, these results demonstrate that participants with both CAD and MVA had higher WBV for both LSR and HSR than control group. Furthermore, hsCRP is reported to have positive correlation with WBV. The key strengths of this study to demonstrate a significant and independent association between simple evaluation of blood viscosity and the existence of endothelial inflammation and atherosclerotic process.

Table 1. Baseline characteristics and laboratory findings of the study groups

Variables	Control Group (n = 181)	MVA (n = 189)	CAD (n = 203)	p value
Clinical Parameters				
Age, years	59.7 ± 9.8	58.1 ± 7.7	59.3 ± 8.9	0.3
Gender/Male, n(%)	96 (53%)	93 (49.2%)	105 (52.5%)	0.55
Hypertension, n(%)	79 (43.6%)	82 (43.3%)	95 (46.3%)	0.21
Diabetes mellitus, n(%)	48 (26.5%)	66 (34.9%)	68 (33.4%)	0.43
Hyperlipidemia, n(%)	49 (27%)	88 (40.7%)	74 (34.3%)	0.09
Current smoker, n(%)	69 (38.1%)	72 (38.9%)	84 (41.3%)	0.67
Laboratory parameters				
Hemoglobin, g/dl	13.3 ± 1.5	13.2 ± 1.4	13.3 ± 1.6	0.60
HCT, %	39.9 ± 5.2	40 ± 4.2	44.3 ± 5	<0.001
WBC, ×103/μl	8.6 ± 2.6	6.5 ± 1.9	9.3 ± 3.9	<0.001
MPV, ×103/μl	9.1 ± 1.3	8.9 ± 0.8	9.4 ± 8.6	0.63
RDW, ×103/μl	14.7 ± 2.9	15.4 ± 1.6	14.7 ± 2.3	0.008
Glucose, mg/dl	95 ± 17	98 ± 25	94 ± 31	0.7
Urea, mg/dl	25.4 ± 1.3	25 ± 5.6	24.6 ± 7.2	0.33
Serum creatinine, mg/dl	0.9 ± 0.3	0.8 ± 0.5	0.9 ± 0.1	0.69
Total protein, g/l	67.2 ± 8.1	73 ± 6.2	73.4 ± 8.7	<0.001
Serum albumin, mg/dl	4.0 ± 0.7	4.2 ± 0.5	4.1 ± 0.9	0.27
Total cholesterol, mg/dl	176 ± 49	188 ± 43	171 ± 39	0.002
LDL cholesterol, mg/dl	109 ± 35	110 ± 41	101 ± 31	0.03
HDL cholesterol, mg/dl	42 ± 12	47 ± 15	43 ± 14	0.01
Triglycerides, mg/dl	147 ± 75	157 ± 93	145 ± 82	0.44
hsCRP, mg/dl	0.23 ± 0.07	0.43 ± 0.04	0.45 ± 0.04	<0.001
Sodium, mEq/l	140 ± 2.9	141 ± 3.7	140 ± 3.1	0.42
Potassium, mEq/l	4.3 ± 0.7	4.4 ± 0.4	4.4 ± 0.5	0.15

Table 2. Comparison of WBV parameters of the study groups

Variables	Conntrol Group	CSX	CAD	P value
WBV at HSR, 208 s-1	15.8 ± 1.4	16.8 ± 1.2	17.4 ± 1.5	<0.001
WBV at LSR, 0,5 s-1	33.8 ± 31	55 ± 26	64.2 ± 33	<0.001

Coronary artery disease / Acute coronary syndrome

PP-186

Evaluation of complete blood count parameters of younger and octogenarians patient with acute st elevated myocardial infarction

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Background and Aim: In our study, we compared the values of whole blood count (CBC) parameters, neutrophil lymphocyte ratio (NLR) and platelet lymphocyte ratio (PLR) parameters of patients over 80 years and under 40 years with acute myocardial infarction (AMI).

Methods: We performed a retrospective study between 2011 and 2016 by screening the files of AMI patients who underwent primary percutaneous coronary intervention. Patients under 40 years of age (Group 1) and over 80 years of age (Group 2) (259 patients in total) were included in the study.

Results: When the hematological parameters were examined, white blood cell (WBC), neutrophil, lymphocyte, thrombocyte, hemoglobin, hematocrit number were found to be significantly higher in Group 1 patients. When PLR and NLR were examined (105.8±67.6 & 149.2±94; p<0.001, 3.2±3.2 & 4.2±4.4, p<0.074 respectively), PLR was significantly higher in group 1. When the age was correlated with hematological and biochemical parameters, there was a significant negative correlation with WBC, neutrophil, lymphocyte and platelet, there was a positive correlation with PLR, NLR, red blood cell distribution width (RDW), Gensini score and creatinine.

Conclusions: In elderly patients over 80 years, the hematological response of cells to acute events such as AMI was reduced due to decreased bone marrow function.

Table 1. Demographic and clinical characteristics of patients

Variable	GROUP 1 (n=85)	GROUP 2 (n=174)	P value
Age, years	35.3 ± 2.9	83.5 ± 2.2	0.001
Gender, male/female	80/5	98/76	0.001
Urea	13 ± 3.5	23 ± 9	0.001
Creatinine	0.9 ± 0.18	1.2 ± 0.62	0.001
Glucose	135 ± 58	149 ± 78	0.264
AST	54 ± 91	41 ± 52	0.292
ALT	37 ± 29	22 ± 20	0.001
LDL	111 ± 44	101 ± 37	0.187
HDL	36 ± 9	46 ± 12	0.001
Triglyceride	197 ± 168	124 ± 64	0.001
Total Cholesterol	183 ± 50	170 ± 46	0.146
Troponin	0.53 ± 1.1	0.47 ± 1.3	0.695
CK-MB	43 ± 70	30 ± 47	0.088
GENSINI	44.5 ± 12.8	67.8 ± 13.4	0.001

Table 2. Hematological parameters

Variables	GROUP 1 (n=85)	GROUP 2 (n=174)	P Value
WBC	11252 ± 2973	8891 ± 3389	0.001
Hemoglobin	14.8 ± 1.3	12.3 ± 1.5	0.001
Hematocrit	42.3 ± 3.7	36.4 ± 4.1	0.001
Platelet	256388 ± 59351	229225 ± 74875	0.004
Neutrophil	7229 ± 3144	6064 ± 3180	0.006
Lymphocytes	2938 ± 1156	1870 ± 890	0.001
Monocytes	826 ± 287	763 ± 328	0.13
MPW	9.9 ± 0.9	10.1 ± 0.9	0.2
RDW	13.8 ± 1.3	14.4 ± 1.6	0.003
NLR	3.2 ± 3.2	4.2 ± 4.4	0.074
PLR	105.8 ± 67.6	149.2 ± 94	0.001

WBC: white blood cell count, MPW: mean platelet volume, RDW: red blood cell distribution width, NLR: Neutrophil / Lymphocytes ratio, PLR: Platelet / Lymphocytes ratio

Table 3. Correlation of age and other parameters

Age	r value	P Value
Creatinine	0.256	0.001
WBC	-0.313	0.001
Neutrophil	-0.159	0.01
Lymphocytes	-0.456	0.001
Platelet	-0.176	0.005
RDW	0.181	0.004
PLR	0.235	0.001
NLR	0.123	0.048
Gensini Score	0.642	0.001

WBC: white blood cell count, RDW: red blood cell distribution width, NLR: Neutrophil / Lymphocytes ratio, PLR: Platelet / Lymphocytes ratio

Coronary artery disease / Acute coronary syndrome

PP-187

Evaluation of association between common SNPs on 9p21.3 region and coronary artery disease in Tanzanian population

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Background and Aim: Coronary artery disease (CAD) is a multi-factorial and heterogenic disease, which develops from complex interactions between genetic and environmental factors. Based on genome-wide association studies (GWAS), a 58kb region on chromosome 9p21.3 has been confirmed strong association with CAD in different populations. But this association is not documented in Tanzanian populations. This study aimed at investigating the common SNPs at the chromosome 9p21.3 region in Tanzanian CAD patients and their associations with biochemical and demographical parameters.

Methods: 135 patients with CAD (age 62.01±10.65) and 140 non-CAD (age 58.21±12.62) patients were enrolled into the study. Further the biochemical analysis, and genomic DNA was isolated by MagnaPure Compact and the genotyping analysis was performed with LightSNIP typing assay using Quantitative Real-Time PCR. The results were examined using Melting Curve analysis program.

Results: The genotypic and allelic distributions of rs1333049, rs2383207, rs2383206, rs10757274, rs10757278, rs10757278 and rs10811656 were significantly different between the groups (p<0.005). Subgroup analysis of CAD patients revealed significant interaction of the risk genotypes of rs10757274 and rs10757278 with hypertension in conferring increased risk of CAD (p<0.05) but not in diabetic and obese subgroups. The genotype distribution of rs1333049 and rs10811656 polymorphisms were significantly different among patients with one, two, three stenotic vessels (p<0.05). Moreover, glucose, cholesterol, HDL, LDL levels were statistically significantly in rs10811656 CT and TT female carriers (p<0.05) and rs10811656 polymorphism was also significantly different among female patients with one, two, three stenotic vessels (p<0.05).

Conclusions: Our results suggest that chromosome 9p21.3 region might be associated with CAD in Tanzanian patients.

Coronary artery disease / Acute coronary syndrome

PP-188

Anxiety and depression can really be risky for cardiac syndrome X?

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Background and Aim: Cardiac syndrome X (CSX) is defined angina-like symptoms, abnormalities on stress testing performed with or without perfusion studies, and normal epicardial coronary arteries on coronary angiography. CSX has found to be an association with depression, anxiety and somatic concern suggested that an increased susceptibility induce coronary microvascular disturbances. The aim of the present study was to determine the Hospital Anxiety and Depression scores of patients with CSX and to compare with healthy controls.

Methods: Between January 2015 and December 2016, patients who admitted to the outpatient clinic with a complaint of chest pain were retrospectively examined. Two hundred ten subjects (110 patients with CSX, 100 controls) were enrolled in the study. The Turkish version of Hospital Anxiety and Depression Scale (HADS), which was translated and validated by Aydemir et al in 1997, was administered to the study population.

Results: The anxiety, depression and total scores in the patients with CSX were significantly higher than those that were observed in the control group (p<0.001, p=0.003, p=0.016, respectively). Among women, anxiety, depression, total scores and stressful life events were significantly higher in CSX group compared with the control group (p=0.006, p=0.015, p=0.001, p<0.001, respectively) (Table 1). Stressful life events and mean anxiety score were the only independent predictors of CSX in logistic regression analysis with comparable odds 2.34 (95% CI 1.093-5.038 p=0.029) and 1.10 (95% CI 1.039-1.177 p=0.002, respectively).

Conclusions: The results of current our research suggested that patients with CSX have a high prevalence of stress and psychiatric disturbances. Interventions targeted toward to improve quality of life and to give psychological support may have the potential benefits for especially most notably female and lower educated people.

Table 1. Baseline characteristics and findings of the study population

	CSX (n=110)	Control (n=100)	p-value
Age (mean ± SD)	52.56±10.8	52.41±9.77	0.91
Gender, female/male (n, %)	58 (52.7%)/52 (47.3%)	59 (59%)/41 (41%)	0.37
Recent Stressful life events	35 (31.8%)	17 (17%)	0.03
Anxiety score	11.25±5.48	8±5.35	<0.001
Depression score	6.55±3.99	5.58±3.76	0.003
Total score	18.80±8.03	13.58±7.62	0.016
HAD-A>10 (n, %)	62 (56.4%)	34 (34%)	0.003
HAD-D>7 (n, %)	52 (47.3%)	30 (30%)	0.019

Coronary artery disease / Acute coronary syndrome

PP-189

Concomitant ST-segment elevation in anterior wall myocardial infarction: Insights into left anterior descending artery anatomy

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Background and Aim: In a subgroup of patients with anterior wall myocardial infarction (MI), electrocardiogram (ECG) records concomitant inferior ST-segment elevation (STE) which is generally explained by a "wrap-around" left anterior descending (LAD) artery occlusion. However, recent evidence indicates that this may be due to distal LAD occlusion which may be irrelevant to LAD length. We investigated the relationship between inferior ST-T changes in anterior MI and the presence of a wrap-around LAD.

Methods: Consecutive patients with the diagnosis of anterior MI due to acute LAD occlusion were enrolled. All ECGs were measured manually by a cardiologist, who was blinded to the angiographic outcomes. The site of LAD occlusion was determined using multiple angiographic views. A wrap-around LAD was defined as an LAD artery from a post-reperfusion coronary angiogram that perfused at least one fourth of the inferior wall of the left ventricle in the right anterior oblique projection.

Results: A total of 379 anterior MI cases were enrolled, final study population consisted of 274 patients. Presence of a wrap-around LAD was more frequent in patients presenting with inferior STE compared with patients without inferior STE (62.1% vs. 31.1%, P=0.001), however this relationship was weak ($\phi=0.207$). STE was more frequent in distal occlusions (24.2% vs. 3.7%, P<0.001), which showed a stronger relationship ($\phi=0.312$). The polarity of T-wave in lead III did not give a clue about LAD anatomy.

Conclusions: Contrary to the popular belief, LAD anatomy cannot be accurately diagnosed from ECG.

Coronary artery disease / Acute coronary syndrome

PP-190

The role of MikroRNAs in premature cardiovascular disease

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Background and Aim: MikroRNAs (MiRNAs) freely circulate in plasma and have emerged as powerful regulators of cardiovascular diseases. However, the role of MiRNAs in the development of premature cardiovascular disease (CVD) still needs to be evaluated. In this study we aimed to compare the plasma levels of 17 MiRNAs in the serum of patients with premature CVD to that of age and sex-matched controls and older patients with CVD.

Methods: Thirty patients with a history of premature CVD (onset of disease <40 years old), 31 age and sex-matched healthy controls and 30 older patients with CVD (onset of disease >55 years old) were included in the study. Patients with diabetes mellitus or any other major systemic disease were excluded. Plasma levels of miR16, miR21, miR27b, miR92a, miR92b, miR122, miR125b, miR126, miR132, miR134, miR145, miR146a, miR146b, miR147b, miR150, miR155, miR370 were quantified by real time polymerase chain reactions in the study population.

Results: Mean age of the patients with premature CVD, the control group and older patients with CVD were 37.08±4.08, 34.81±4.37 and 64.9±7.1 years old, respectively. Eighty seven percent of each group were male. The clinical characteristics of the patients with CVD are given in Table 1. There was statistically significant difference in expression of miR27b, miR122, miR125b, miR126, miR132, miR134, miR145, miR146a, miR146b, miR147b, miR150, miR155 between the patients with premature CVD and age and sex-matched control group (Table 2). Plasma levels of miR27b, miR122, miR125b, miR146b, miR147b, miR150 and miR155 were more than 10 fold down-regulated in patients with premature CVD compared to healthy controls. On the other hand patients with early or late onset CVD had also statistically significant difference in expression of miR16, miR21, miR27b, miR92a, miR122, miR125b, miR146b, miR147b and miR155 (Table 3).

Conclusions: Circulating MiRNAs may become helpful and reliable biomarkers for premature onset of cardiovascular disease.

Table 1. Clinical characteristics of the patients with cardiovascular disease

	Patients with premature cardiovascular disease	Patients with late onset cardiovascular disease	p value
Active smoker (%)	47	27	<0.01
Hypertension (%)	27	47	<0.01
Hyperlipidemia (%)	9	17	0.03
Regular physical activity (%)	40	33	0.03
Acetylsalicylic acid therapy (%)	97	93	1.0
Clopidogrel therapy (%)	63	36	0.03
Beta blocker therapy (%)	87	67	0.12
ACEI/ARB therapy (%)	53	67	0.83
Statin therapy (%)	63	60	1.0

Table 2. Relative expression of MiRNAs in patients with premature CVD compared to healthy controls

	The expression of MiRNAs in patients with premature CVD compared to healthy controls	p value
Mir16	-1.22 fold	0.35
Mir21	1.52 fold	0.40
Mir27b	-16.85 fold	0.04
Mir92a	1.79 fold	0.18
Mir92b	1.58 fold	0.97
Mir122	-21.11 fold	0.04
Mir125b	-10.23 fold	0.04
Mir126	2.12 fold	0.04
Mir132	-2.84 fold	0.04
Mir134	-2.26 fold	0.04
Mir145	-6.31 fold	0.03
Mir146a	-3.46 fold	0.03
Mir146b	-13.04 fold	0.01
Mir147b	-25.39 fold	0.04
Mir150	-10.36 fold	0.03
Mir155	-21.19 fold	0.04
Mir370	-1.32 fold	0.17

CVD: Cardiovascular disease.

Table 3. The relative expression of MiRNAs in patients with premature CVD compared to older patients with CVD

	The expression of MiRNAs in patients with premature CVD compared to older patients with CVD	p value
Mir16	-2.27 fold	0.02
Mir21	-2.33 fold	0.04
Mir27b	-10.22 fold	0.01
Mir92a	6.32 fold	0.04
Mir92b	1.81 fold	0.81
Mir122	-4.19 fold	0.02
Mir125b	-22.06 fold	0.04
Mir126	1.71 fold	0.44
Mir132	-1.30 fold	0.41
Mir134	1.60 fold	0.16
Mir145	1.51 fold	0.16
Mir146a	1.31 fold	0.11
Mir146b	-12.72 fold	<0.01
Mir147b	-39.66 fold	<0.01
Mir150	-1.20 fold	0.71
Mir155	-5.10 fold	0.01
Mir370	-1.11 fold	0.95

CVD: Cardiovascular disease.

Coronary artery disease / Acute coronary syndrome

PP-191

Serum serglycin levels in acute myocardial infarction

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Background and Aim: Serglycin is a plasma glycoprotein and its role in the pathogenesis of atherosclerosis is being investigated. Despite being described as an important agent in fibrin stabilization and inflammation in studies with Serglycin, very little information is available about the clinical value. The purpose of this study is; to investigate a possible association between acute myocardial infarction and serglycin by comparing the serum serglycin level with those of patients with normal coronary arteries in the presence of acute myocardial infarction.

Methods: The study population consisted of 57 patients with ST elevation acute myocardial infarction (STEMI), 60 patients with acute myocardial infarction without ST elevation (NSTEMI) and 57 angiographically normal coronary arteries. Serglycin serum level was determined by ELISA method on admission. Patient characteristics, biochemical parameters and prognostic risk markers were recorded. Patients with known systemic disease, cancer diagnosis or active infection, creatinine level 1.6 mg / dL or higher were excluded from the study. **Results:** A total of 57 control groups, a total of 60 STEMI patients (41 males, 44 males) and a total of 57 NSTEMI patients (30 males with normal coronary arteries) were included in the study. The mean age of the control group (57±12), NSTEMI and STEMI group (64±15, 62±14, respectively) were statistically significantly lower than the mean age. When the AUC levels were evaluated, the mean AUC levels of the NSTEMI and STEMI groups (123,136 mg / dL, respectively) were statistically significantly higher than the control group. NSTEMI, STEMI and control groups were 49.1%, 66.7% and 28.1%, respectively. This proportional difference between the study groups is also statistically significant and this difference stems from the STEMI group and the control group. There was a statistically significant difference in the amount of troponin and CRP between NSTEMI and STEMI groups. There was a statistically significant difference between the STEMI

group average and the NSTEMI and control group averages in terms of Serglycin level. Although the NSTEMI group was higher than the serglycin level average control group, no statistically significant difference was found between them. Correlation analysis with troponin T and CRP also showed a statistically significant positive correlation. There was a low moderate positive correlation between Serglycin and troponin T (r value 0.301, p value 0.001) and a low correlation (r value 0.278, p value <0.001) between Serglycin and CRP. **Conclusions:** Due to the information we obtained as a result of our study, Serglycin levels in STEMI were statistically significantly higher than NSTEMI and control group, but there was no significant difference between NSTEMI and control groups. Correlation analysis with troponin T and CRP also showed a statistically significant positive correlation.

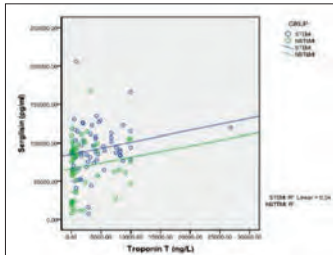


Figure 1.

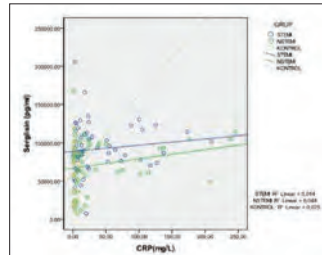


Figure 2.

Table 1.

	NSTEMI (n=57)	STEMI (n=60)	KONTROL (n=57)	p değeri
Hipertansiyon, %	40	43.9	35.1	0.631
Diyabetes Mellitus, %	31.6	33.3	22.8	0.412
KAH için A6, %	12.3	10	1.8	0.09
Sigara, %	49.1	66.7	28.1	<0.001

Table 2.

	NSTEMI (n=57)	STEMI (n=60)	KONTROL (n=57)	p değeri
AKS, mg/dl	123 (98-155)	136 (105.5-211)	103 (94-125)	<0.001
Üre, mg/dl	36 (28-49)	38 (27.5-44.5)	31.5 (27-36)	0.050
Kreatinin, mg/dl	1 (0.89-1.16)	0.97 (0.8-1.09)	0.83 (0.69-0.98)	<0.001
Total Kolesterol, mg/dl	167.3±42.38	170±43.39	183.98±38.87	0.088
LDL, mg/dl	100.63±35.1	99.5±35.63	107.23±36.49	0.481
HDL, mg/dl	38 (32-44)	40 (34-52)	42.5 (36-51)	0.041
TG, mg/dl	142 (89-185)	126.5 (86-179)	146.5 (92.5-195)	0.172
Hb, mg/dl	13.9 (12.5-14.9)	13.4 (11.4-14.8)	14.3 (12.5-15.9)	0.023
WBC, /mm ³	9.6 (8-13)	11.5 (9.5-14.35)	8 (6.8-9.6)	<0.001
Plt, /mm ³	229 (182-265)	235 (193-293)	256 (228-295)	0.007

Table 3.

	NSTEMI	STEMI	KONTROL	p değeri
Troponin T, ng/L	700 (188-2400)	3585 (1655-6740)	NA	<0.001
CRP, mg/L	9 (3-34)	16 (5.5-54.5)	3 (1-6)	<0.001
Serglisin, pg/ml	75690 (47255-93295)	89042 (75690-112368)	72693 (33526-92158)	<0.001

Table 4.

		Serglisin	CRP	Troponin T
Serglisin	Rho katsayısı	1.0	0.278	0.301
	p değeri		0.000	0.001
CRP	Rho katsayısı	0.278	1.0	0.378
	p değeri	0.000		0.000
Troponin T	Rho katsayısı	0.301	0.378	1.0
	p değeri	0.001	0.000	

Coronary artery disease / Acute coronary syndrome

PP-192

The relationship between serum KIM-1 levels and severity of coronary artery disease in patients with acute myocardial infarction

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Background and Aim: Kidney Injury Molecule-1 (KIM-1) is a transmembrane glycoprotein that has been described as an early biomarker for acute renal failure. It is known that the KIM-1 molecule is associated with chronic inflammation and atherosclerosis is also an inflammatory process. The aim of this study was to investigate whether there was a relationship between the KIM-1 molecule levels in the case group with acute myocardial infarction and the control group in addition to whether the KIM-1 level was correlated with age, gender, hypertension, diabetes mellitus, hyperlipidemia, smoking habits, Gensini and Syntax score, total cholesterol, HDL and LDL levels.

Methods: Patient characteristics, biochemical parameters and prognostic risk markers were recorded. The SYNTAX scores were assessed using the online SYNTAX Score Calculator version 2.11 and the Gensini score was measured by three cardiologists who did not know the clinical status of the individual by measuring coronary artery diameter and lumen stenosis. Patients who were treated with thrombolytics rescuer percutaneous coronary intervention, who has a known systemic disease (hyper or hypothyroidism, chronic liver disease, collagen tissue diseases), cancer diagnosis or active infection, high creatinine level of 1.6 mg / patients, who were treated with steroid or other non-steroidal anti-inflammatory drugs and patients who were missing KIM-1 data were excluded from the study.

Results: A total of 78 acute myocardial infarction patients and 53 control groups with normal coronary arteries were included in the study, including 34 female and 97 male. The mean age of the control group (57.72±9.68) and the mean age of the case group (60.92±11.66) were not found to be statistically significant. In our study, there was no statistically significant relationship between case group and control group (median values 55.82, 54.45, p>0.05 respectively) in terms of KIM-1 level. Similarly, there was no statistically significant correlation between KIM-1 level and Gensini score and Syntax score (-0.12, p>0.05 and -0.41, p>0.05, respectively) which are showing the severity and prevalence of coronary artery disease. There was a statistically significant weak correlation between age and KIM-1 level (correlation coefficient 0.225, p=0.01). There was statistically significant higher KIM-1 levels in hypertensive subjects (192.72, 127.96, p=0.010), but no correlation was found between KIM-1 level and gender or presence of diabetes mellitus. Similarly, there was no correlation between lipid parameters which are risk factors of coronary artery disease, creatinine values which is the renal function test and KIM-1 levels.

Conclusions: In our study, there was no statistically significant relationship between myocardial infarction and control group in terms of KIM-1 molecule level. Similarly, there was no statistically significant relationship between Gensini score and Syntax score which showed the severity and extent of coronary artery disease and KIM-1 level.

Coronary artery disease / Acute coronary syndrome

PP-193

Association of serum copeptin levels with infarct related artery patency in ST segment elevation myocardial infarction patients

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Background and Aim: Copeptin has been widely adopted as a predictor of adverse prognosis in many clinical conditions. Reduced antegrade coronary flow in an infarct-related artery (IRA) is associated with adverse clinical outcome in patients with ST-segment elevation myocardial infarction (STEMI). The aim of this study was to investigate whether copeptin level on admission was associated with the IRA patency in STEMI patients.

Methods: A total of 88 patients enrolled into the study and were divided into two groups according to TIMI flow grade in the IRA before primer percutaneous coronary intervention.

Results: White blood cell count (p=0.015), neutrophil (p=0.047), N-terminal pro-brain natriuretic peptid (NT-proBNP) (p<0.001), copeptin (p<0.001) and peak troponin I (p=0.001) were significantly higher in occluded IRA group with significantly lower serum sodium level (p<0.001). Adjusted (age and gender) univariable analysis showed that copeptin (OR=1.970; p=0.001), peak troponin I (1.055; p=0.005) and NTproBNP (OR=1.003; p=0.010) was risk factor of an occluded IRA. A copeptin cut-off value of >6.8 ng/mL was found to predict an occluded IRA with a sensitivity of 80% and specificity 100% (Area Under Curve: 0.917; p<0.001). A peak troponin I cut-off value of >33.2 ng/mL was found to predict an occluded IRA with a sensitivity of 80% and specificity 55.6% (Area Under Curve: 0.744; p<0.001). A NTproBNP cut-off value of >269 pg/mL was found to predict an occluded IRA with a sensitivity of 84.3% and specificity 55.6% (Area Under Curve: 0.755; p<0.001). Performance ranking of the copeptin, peak troponin I and NTproBNP that could predict occluded IRA was determined to be Copeptin > peak troponin I = NTproBNP.

Conclusions: In conclusion, our study results showed that there was a strong relationship between the levels of copeptin and the TIMI flow grade of IRA in the patients with STEMI who were admitted to the emergency department during the first three hours of chest pain.

Coronary artery disease / Acute coronary syndrome

PP-194

Serum copeptin level at admission predicts and well correlated with high SYNTAX score in patients with Non-ST segment elevated myocardial infarction

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Background and Aim: C-terminal pro-vasopressin (copeptin) is a relatively novel biomarker which can be used in diagnosis of acute coronary syndrome (ACS). We aimed to evaluate the relationship between the copeptin level at hospital admission of patients with Non-ST segment elevated myocardial infarction (NSTEMI) and SYNTAX score.

Methods: A total of 87 patients with NSTEMI were enrolled. Electrocardiography (ECG), complete blood count, serum biochemistry, copeptin, pro-BNP, troponin I and echocardiography were assessed in all patients. Coronary angiography results were systematically evaluated for each arterial segment along with the prevalence and severity of arterial disease based on the SYNTAX score system by two independent cardiologists. Patients were allocated as those with low SYNTAX score (≤ 22) and moderate-high SYNTAX score (> 22).

Results: Of the patients, 58 (66.7%) had a low SYNTAX score and 29 (33.3%) with a moderate-high SYNTAX score. Median serum copeptin (7.5 ng/ml vs 2.7 ng/ml; $p < 0.001$) and pro-BNP levels (236 ng/ml vs 161.5 ng/ml; $p < 0.001$) of the patients with moderate-high SYNTAX score were found to be higher than those with a low SYNTAX score. There was a positive high correlation between the copeptin levels and SYNTAX score ($r = 0.711$; $p < 0.001$). In the multivariate logistic regression model, copeptin level (OR=1.49; $p < 0.001$), peak troponin I (OR=1.08; $p = 0.003$) and proBNP level (OR=1.03; $p < 0.001$) were determined as independent predictors of moderate-high SYNTAX score.

Conclusions: During admission for NSTEMI, serum copeptin level is higher in patients with moderate-high SYNTAX score. There is a positive correlation with SYNTAX score and copeptin. Furthermore, serum copeptin level predicts the patients with moderate-high SYNTAX score.

Coronary artery disease / Acute coronary syndrome

PP-195

Diagnostic utility of chest pain characteristics in discriminating obstructive coronary artery disease: A 'Historic' Dilemma

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Background and Aim: Chest pain (CP) is the major presenting symptom of coronary artery disease (CAD) wherein history remains as a fundamental and challenging diagnostic step. Despite the clarity of 'typical' CP characteristics outlined in guidelines, interpretation and description of CP by patients are open to wide variations. Several studies failed to prove discriminatory value of these characteristics for prediction of significant CAD in acute CP but data on chronic CP is limited. Management decisions, however, rely primarily on the subjective interpretation of CP. The aim of this study was therefore to define potential components of CP that most closely associate to presence of obstructive CAD and determine the influence of gender on CP features.

Methods: Consecutive patients with suspected stable CAD (402 patients; 48% women; mean age 61.2±10.9 years) who were scheduled for a coronary angiography were prospectively enrolled. Patients with acute coronary syndromes or recent (within 6 months) revascularization were excluded. The data on demographic and pain characteristics were collected by a questionnaire completed prior to angiography. Biochemical parameters and the CAG results were recorded. Patients were categorized into 'CAD' and 'Normal' groups with respect to presence of CAD that causes luminal stenosis of $\geq 50\%$.

Results: Among 402 patients, 86 had chest discomfort or equivalent symptoms but denied 'CP'. Demographic and biochemical parameters with respect to presence of CAD for patients with and without CP are presented in the Table 1 and 2 respectively. Patients with CAD were more commonly older, male patients with lower BMI, prior revascularization and hence clopidogrel use; with higher creatinine and lower HDL levels than normal subjects. When main components of CP such as quality, location, duration, relationship to exertion and others were compared between CAD and normal groups; stabbing/sharp pain, CP related to cold or emotional stress and CP that radiates to back were significant factors against CAD; while absence of precipitating factors was related to CAD. (Table 3) More interestingly, none of the components of typical angina pectoris were significantly related to presence of CAD. When the determinants of CAD were evaluated by logistic regression analysis in terms of gender; prior revascularization (OR=22.7, $p = 0.021$), body mass index (OR=1.4, $p = 0.007$), clopidogrel use (OR=55.5, $p = 0.018$) and glucose (OR=1.02, $p = 0.046$) were significant predictors of CAD in women while age (OR=1.2, $p = 0.029$) was the single predictor of CAD in men.

Conclusions: This study demonstrated no association between classical features of typical CP and presence of CAD in patients with stable angina pectoris. On contrary, pain that radiates to back; that is exacerbated by cold or emotional stress was associated with absence of CAD. Various clinical factors influenced presence of CAD in different genders; yet none of the CP characteristics was independently associated with CAD in both genders.

Table 1. Baseline characteristics of patients with chest pain

Baseline Characteristics	Overall study population n=316	Obstructive CAD n=165	Normal n=151	p value	
Age	60.4±10.7	62.9±10.3	57.7±10.5	<0.001	
Female Gender, % (n)	50 (158)	35.8 (59)	65.6 (99)	<0.001	
Education, % (n)	primary	85.1 (269)	86.1 (142)	84.1 (127)	0.9
	high school	11.1 (35)	10.9 (18)	11.3 (17)	
	university	3.8 (12)	3 (5)	4.6 (7)	
Work status, % (n)	30.4 (96)	30.3 (50)	30.5 (46)	0.97	
BMI	29.2 (18.5-48.8)	28.2 (19.4-45.5)	31.1 (18.5-48.8)	0.004	
Hypertension, % (n)	54.7 (173)	55.2 (91)	54.3 (82)	0.88	
Diabetes mellitus, % (n)	30.7 (97)	31.5 (52)	29.8 (45)	0.74	
Hyperlipidemia, % (n)	31.6 (100)	30.9 (51)	32.5 (49)	0.76	
Current Smoker, % (n)	28.5 (90)	28.4 (41)	32.5 (49)	0.13	
Family history, % (n)	55.4 (175)	51.5 (85)	59.6 (90)	0.149	
Previous revascularization, % (n)		55.8 (92)	32.5 (49)	<0.001	
Medications, % (n)	ASA	50.3 (159)	54.5 (90)	45.7 (69)	0.11
	Clopidogrel	22.5 (71)	33.9 (56)	9.9 (15)	
	Beta blockers	51.3 (162)	50.9 (84)	51.7 (78)	
	RAAS blockers	41.5 (131)	45.5 (75)	37.1 (56)	
	CCB	16.5 (52)	14.5 (24)	18.5 (28)	
	Statin	26.3 (83)	30.3 (50)	21.9 (33)	
	OAD	21.5 (68)	21.2 (35)	21.9 (33)	
	Insulin	8.2 (26)	9.1 (15)	7.3 (11)	
	Nitrates	4.7 (15)	5.5 (9)	4 (6)	
	Glucose	112 (62-419)	113 (77-419)	110 (62-346)	
White blood cell, $\times 10^3$	7.2 (3.4-15.5)	7.3 (3.4-15.5)	7.2 (3.5-14.4)	0.50	
Hemoglobin, g/dL	13.4 (9.2-18.6)	13.5 (9.2-18.6)	13.3 (9.7-18)	0.50	
Platelet, $\times 10^3$	243 (108-547)	231 (114-475)	259 (108-547)	0.051	
Creatinine	0.85 (0.5-2.2)	0.87 (0.54-2.2)	0.81 (0.52-1.56)	0.02	
LDL, mg/dL	116 (25-269)	114 (25-269)	116 (28-247.5)	0.46	
HDL, mg/dL	44 (21-104.5)	42 (21-104.5)	46.1 (25-91.2)	0.013	
T. cholesterol, mg/dL	198 (96-356)	193 (110-359)	202 (96-353)	0.33	
Triglyceride, mg/dL	171 (48-743)	176 (48-722)	167 (54-743)	0.74	

Categorical variables are compared by chi square; continuous variables are compared by independent samples t test or Mann Whitney U as appropriate. * indicates p values calculated by continuity correction.

Table 2. Baseline characteristics of the patients without chest pain

Baseline Characteristics	Overall study population n=86	Obstructive CAD n=44	Normal n=42	p value	
Age	63.7±11.4	64.5±10.8	63±12.1	0.55	
Female Gender, % (n)	40.7 (35)	29.5 (13)	52.4 (22)	0.053*	
Education, % (n)	primary	73.3 (63)	77.3 (34)	69 (29)	0.44
	high school	10.5 (9)	11.4 (5)	9.5 (4)	
	university	16.3 (14)	11.4 (5)	21.4 (9)	
Work status, % (n)	26.7 (23)	20.5 (9)	33.3 (14)	0.26	
BMI	28.5 (18.3-44.4)	28.4 (18.3-44.4)	29.3 (20.9-42.5)	0.27	
Hypertension, % (n)	48.8 (42)	50 (22)	47.6 (20)	0.99	
Diabetes mellitus, % (n)	26.7 (23)	25 (11)	28.6 (12)	0.89	
Hyperlipidemia, % (n)	32.6 (28)	31.8 (14)	33.3 (14)	1	
Smoking status, % (n)	25.6 (22)	27.3 (12)	23.8 (10)	0.90	
Family history, % (n)	48.8 (42)	47.7 (21)	50 (21)	1	
Previous revascularization, % (n)		63.6 (28)	21.4 (9)	<0.001	
Medications, % (n)	ASA	50 (43)	65.9 (29)	33.3 (14)	0.05
	Clopidogrel	17.4 (15)	27.3 (12)	7 (3)	
	Beta blockers	41.9 (36)	47.7 (21)	35.7 (15)	
	RAAS blockers	47.7 (41)	59.1 (26)	35.7 (15)	
	CCB	15.1 (13)	20.5 (9)	9.5 (4)	
	Statin	27.9 (24)	34.1 (15)	21.4 (9)	
	OAD	19.8 (17)	20.5 (9)	19 (8)	
	Insulin	4.7 (4)	6.8 (3)	2.4 (1)	
	Nitrates	3.5 (3)	4.5 (2)	2.4 (1)	
	Glucose	111 (68-379)	114 (87-356)	110 (68-379)	
White blood cell, $\times 10^3$	7.5 (3.7-12.7)	7.3 (4.1-11.5)	7.6 (3.7-12.7)	0.19	
Hemoglobin, g/dL	13.2 (9-17.7)	13.3 (9-16.6)	13.2 (9.1-17.7)	0.61	
Platelet, $\times 10^3$	237.5 (121-457)	220 (141-432)	262 (121-457)	0.019	
Creatinine	0.9 (0.6-6.8)	0.88 (0.59-2.0)	0.91 (0.56-6.8)	0.56	
LDL, mg/dL	104 (27-357)	102 (27-176)	106 (40.8-358)	0.71	
HDL, mg/dL	44 (25-99)	43 (25-99)	45 (29-80)	0.38	
T. cholesterol, mg/dL	183 (98-439)	183 (101-275)	194 (98-439)	0.82	
Triglyceride, mg/dL	158 (14.2-527)	167.5 (14.2-527)	149 (60-518)	0.13	
Predominant symptom	Dyspnea	25.6 (22)	22.7 (10)	28.6 (12)	0.41
	pre/syncope	1.2 (1)	-	2.4 (1)	
	sweating	1.2 (1)	2.3 (1)	-	
	fatigue	25.6 (11)	9.1 (4)	16.7 (7)	
	chest discomfort	59.3 (51)	65.9 (29)	52.4 (22)	

Categorical variables are compared by continuity correction chi square; continuous variables are compared by independent samples t test or Mann Whitney U as appropriate. * indicates p values calculated by Fishers exact test.

Table 3. Characteristics of chest pain in patients with/out CAD

Characteristics of chest pain		Overall study population n=316	Obstructive CAD n=165	Normal n=151	p value
Quality, %(n)	squeezing/pressure	46.5(147)	47.3(78)	45.7(69)	0.77
	ache/burn	21.2(67)	21.8(36)	20.5(31)	0.78
	needling	21.2(67)	23.6(39)	18.5(28)	0.26
Localization, %(n)	stabbing/sharp	11.1(35)	7.3(12)	15.2(23)	0.03
	anterior thorax	94(297)	93.3(154)	94.7(143)	0.78
	epigastric	2(9)	2.4(4)	3.3(5)	0.74
Duration, %(n)	back	3.2(10)	4.2(7)	2(3)	0.34
	seconds	29.3(64)	20(33)	29.5(31)	0.80
	<5 min	34.2(108)	36.4(60)	31.8(48)	
	5-10 min	25(79)	23.5(39)	26.5(40)	
	10 min+	12(38)	13.3(22)	10.6(16)	
hours	6.3(20)	5.5(9)	7.3(11)		
Precipitating factors, %(n)	none identified	18.4(58)	23.6(39)	12.6(19)	0.011
	exercise	67.7(214)	64.2(106)	71.5(108)	0.167
	cold	31(98)	24.8(41)	37.7(57)	0.013
	emotional stress	57.6(182)	47.9(79)	68.2(103)	<0.001
Relieving factors, %(n)	none	15.8(50)	17.6(29)	13.9(21)	0.61
	rest	79.1(250)	77(127)	81.5(123)	
	nitrates	5(16)	5.5(9)	4.6(7)	
Radiation, %(n)	none	21.2(32)	26.1(43)	21.2(32)	0.4
	back	48.4(153)	42.4(70)	55.6(84)	0.014
	shoulders	26.6(84)	25.5(42)	27.8(42)	0.58
	arms	41.5(131)	38.8(64)	44.4(67)	0.26
	jaw	24.7(78)	23(38)	26.5(40)	0.43
Number of radiated places	1(0-4)	1(0-4)	1(0-4)	0.039	
Accompanying symptoms, %(n)	None	19(57)	20(33)	15.4(24)	0.34
	Dyspnea	49.1(155)	46.1(76)	52.3(79)	0.26
	nausea	19.9(63)	20(33)	19.9(30)	0.97
	pre/syncope	11.7(37)	9.1(15)	14.6(22)	0.18
	cold sweating	50.9(161)	49.1(81)	53(80)	0.49
fatigue	63.9(202)	63(104)	64.9(98)	0.72	
Scale		5(1-10)	5(2-10)	5(1-10)	0.9

Coronary artery disease / Acute coronary syndrome

PP-196

Evaluation of copeptin levels and the relationship between these values and subclinical atherosclerosis markers in sarcoidosis patients

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Background and Aim: Sarcoidosis is a multisystemic disease characterized by the formation of granulomatous inflammation with an unknown etiology. The aim of this study is to investigate the presence of subclinical atherosclerosis and endothelial dysfunction using carotid intima media thickness (CIMT) and flow-mediated dilatation (FMD) measurements in sarcoidosis patients and to evaluate the correlation of these measurements with the values of copeptin, based on the idea that chronic inflammatory diseases may cause subclinical atherosclerosis.

Methods: 87 patients (58 f, 29 m) with histopathological diagnosis of sarcoidosis and without conventional cardiovascular risk factors and 96 healthy volunteers (56 f, 40 m) with similar sociodemographic characteristics were included in this study. Measurements of CIMT and FMD were performed using B-mode ultrasound. Serum copeptin levels of all participants were measured.

Results: The values of the CIMT and Copeptin in sarcoidosis were significantly higher (p<0.001 for both variables) and FMD was significantly lower (p<0.05) than the control group. There was no significant correlation between Copeptin and CIMT and FMD values in the sarcoidosis patient group (p=0.243, p=0.817, respectively).

Conclusions: Demonstration of the presence of subclinical atherosclerosis and endothelial with measurements of CIMT and FMD in sarcoidosis patients suggests that patients should be investigated and followed closely for the development of coronary artery disease. In addition, more extensive studies are needed to investigate the pathophysiology of high copeptin levels in sarcoidosis patients and effects of these values on the prognosis of the disease.

Coronary artery disease / Acute coronary syndrome

PP-197

Impact of aortic pulse pressure on one-year outcomes after ST elevation myocardial infarction

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Background and Aim: Acute myocardial infarction (AMI) is one of the leading causes of death worldwide. Impaired cardiac function develops after ST-elevation myocardial infarction (STEMI). The changes in blood

pressure STEMI were demonstrated to be associated with increased morbidity and mortality. These changes affect the levels of systolic (SBP) and diastolic blood pressure (DBP). The difference between SBP and DBP is called pulse pressure (PP). Within the scope of this study, the prognostic value of PP which was measured intra-aortically with the aid of a catheter in one-year period STEMI was aimed to be evaluated.

Methods: A total of 261 patients whose blood pressure was measured with the aid of a catheter before primary percutaneous coronary intervention (PPCI) between May 2017 and May 2018 were included in the study. The patients in whom less than 12 hours passed after the onset of STEMI and who were taken to the angiography laboratory for PPCI were evaluated. Since hypotensive periods were more frequent in inferior STEMI, creating a homogeneous group was aimed. Therefore, only the patients with A-STEMI were included in the study. Electrocardiographic examinations were performed during the period when the patients were first admitted to the emergency department. Presence of ST elevation ≥ 0.2 m in at least two of the precordial leads, newly developed left bundle branch block (LBBB), presence of chest pain with typical onset and spread, Cardiac troponin-I (c-TnI) and creatinine kinase myocardial band (CK-MB) values greater than 99th percentile were considered as A-STEMI. Since our center has a team on call for 7/24, the patients were quickly taken to the angiography laboratory after being diagnosed with A-STEMI. The patients were divided into three groups according to their PP (Group-1; PP <35 mmHg, Group-2; 35 \leq PP \leq 50 mmHg, Group-3; PP >50 mmHg).

Results: The mean age of the patients was 61.4 \pm 12.1 years and 206 of them were male. The groups were similar in terms of age, BMI, DM and DBP. The ratio of female patients in Group-1 was higher and their SBP was lower than the other groups (p=0.002 vs p=0.022). The rates of MACE, mortality, cardiogenic shock and newly-developed AF were higher in Group-1. The predictive PP values was calculated to be 41.5 mmHg for MACE development and 40.5 mmHg for mortality. One-year survival was worse in Group-1 according to the Kaplan-Meier analysis (p<0.001).

Conclusions: According to the results of our study, it was determined that the level of PP which was measured intra-aortically after STEMI was associated with mortality and MACE development in one year period.

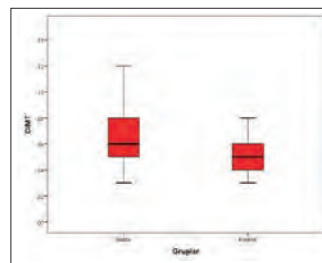


Figure 1. Distribution of CIMT levels according to the groups (p<0.001).

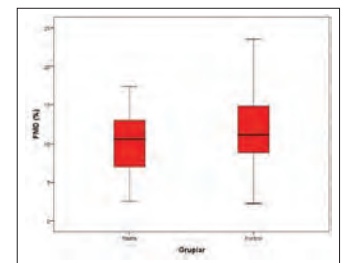


Figure 2. Distribution of CIMT levels according to the groups (p<0.001).

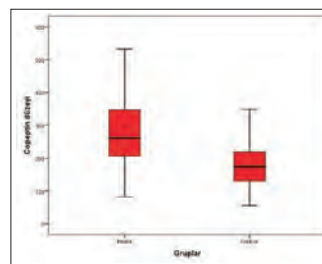


Figure 3. Distribution of copeptin levels according to groups (p<0.001).

Coronary artery disease / Acute coronary syndrome

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The relationship between high sensitive c-reactive protein and the lapse of time since the onset of the symptoms after acute myocardial infarction: An prospective-observational study

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Background and Aim: ST elevation myocardial infarction (STEMI) is one of the most important health problems that threaten the human life all over the world. It is important to know the elapsed symptom-to-door (StD) time between the emergence of ST-elevation myocardial infarction (STEMI) symptoms and admission to the hospital in terms of the selection of appropriate treatment and prognosis. In this study, we aimed to assess the relationship between serum C-reactive protein (CRP) and StD time after STEMI.

Methods: 136 of the patients admitted to our center with STEMI between August 2016-February 2018 (88 male, mean age, 62.7 \pm 12.8) were included in this prospective-observational cohort study. Blood samples were obtained from laboratory results of the first reference period. Patients were divided into four groups according to the duration of StD time [0-1. hour; group 1 (G1), 1-3. hour; group 2 (G2), 3-6. hour; group 3 (G3), 6-12. hour; group 4 (G4)]. Statistical analysis was performed via chi-square test, ANOVA test, Pearson's correlation analysis and receiver operator characteristic (ROC) analysis.

Results: As the time progressed, an increase in CRP levels was observed. The difference among the means of the G1-G3 (p=0.012), G1-G4 (p<0.001), G2-G4 (p<0.001) and G3-G4 (p<0.001) groups was found to be statistically significant. There was a good correlation between the StD time and CRP levels (r=0.716). ROC analysis of the predictive value of CRP for the third hour was determined as 4,8 mg/dL, respectively (AUC was 0.804; 95% C.I. was 0.755-0.826; 71.9% sensitivity, 72.1% specificity).

Conclusions: According to serum Hs-CRP levels after STEMI at hospital admission, StD time can be estimated.

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