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36th TURKISH CARDIOLOGY CONGRESS

WITH INTERNATIONAL PARTICIPATION

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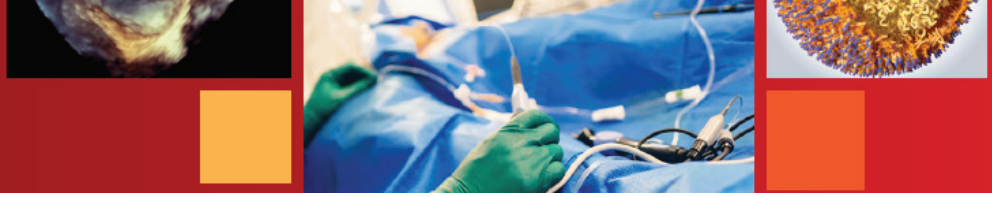

TURKISH SOCIETY OF CARDIOLOGY

36th TURKISH CARDIOLOGY CONGRESS

WITH INTERNATIONAL PARTICIPATION

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TURKISH SOCIETY OF CARDIOLOGY

36th TURKISH CARDIOLOGY CONGRESS

WITH INTERNATIONAL PARTICIPATION

Dear Colleagues,

First of all, we are very sorry for the COVID-19 pandemic, which has affected all countries of the world in terms of health, education and especially the economy. We strongly hope that the wounds of this difficult period will be healed quickly and the social and economic effects will be over with the least damage.

As you know, "36th Turkish Cardiology Congress with International Participation" could not be held on the planned dates due to the Covid-19 pandemic. We aim to hold the National Congress on "TSC2020DIGITAL" platform between "3-6 December 2020".

Our goal has always been to make our Cardiology Congress the leading convention in our region. This year, our colleagues from the European countries, from the United States, Russia, Turkic World and other countries will also participate in our congress. This will make us strong to share knowledge as the preceding years

We will update and discuss our latest information about cardiovascular diseases through our "Symposia", "Pro-Con" and "How-to" sessions. We are pleased to announce that the "TSC Young" program was organized and successfully ran by our young colleagues. It will take place this year in the 3rd main hall. Furthermore, this year we will be discussing the topics that intersect clinical and basic sciences in a detailed manner in our "Focused Sessions" and "Interactive Courses" in "TSC2020DIGITAL" webinars.

In each session, we will have distinguished speakers from both Turkey and across the globe who have a great power in their respective fields. We believe that you will have special interest to our joint sessions with ESC, ACC, Russian Society of Cardiology, EAS, Turkic World Cardiology Association, EACVI, ACCA, EHRA, EAPCI, and HFA.

It's a pleasure to welcome all of you to our "36th Turkish Cardiology Congress with International Participation" on "3rd – 6th of December 2020" to share the knowledge.

Yours Sincerely,

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

Prof. Vedat Aytekin, M.D.
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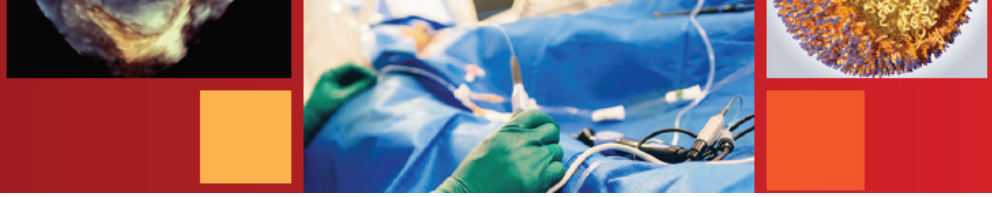

TURKISH SOCIETY OF CARDIOLOGY

36th TURKISH CARDIOLOGY CONGRESS

WITH INTERNATIONAL PARTICIPATION

CONTENTS

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-001

Genetic variants associated with long term atrial tachyarrhythmia recurrence after catheter ablation for atrial fibrillation in Turkish patients

Taner Ulus,[1] Muhammet Dural,[1] Pelin Meşe,[1] Furkan Yetmiş,[1] Kadir Uğur Mert,[1] Bülent Görenek,[1] Oğuz Çilingir,[2] Ebru Erzurumluoğlu,[2] Serap Aslan,[2] Sevilhan Artan,[2] Özlem Aykaç,[3] Ertuğrul Çolak,[4] Hikmet Yorgun,[5] Uğur Canpolat,[5] Kudret Aytemir[6]

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Background and Aim: Catheter ablation is an effective treatment option in restoring and maintaining sinus rhythm in patients with symptomatic paroxysmal or persistent atrial fibrillation (AF). Genome-wide association studies have demonstrated that single nucleotide polymorphisms (SNPs) can predict long-term atrial tachyarrhythmia (ATa) recurrence after catheter ablation for AF in different societies. However, there is paucity of data on this subject in Turkish patients. We aimed to investigate if SNPs in the PITX2, ZFHX3, EPHX2, CAV1, TBX5, TGF-1 and SCN10A genes predicted long-term ATa recurrence after catheter ablation for AF in Turkish patients.

Methods: One hundred twenty-eight patients who underwent catheter ablation for pulmonary vein Isoiation (PVI) using second-generation cryoballoon at Eskişehir Osmangazi University and Hacettepe University Hospitals were enrolled. Any ATa episode lasting at least 30 s was defined as recurrence. Early recurrence was defined as a recurrence within a 3-month blanking period. The patients were followed-up with physical examination and 24-h Holter recording at outpatient clinics at 3th, 6th,12th months and at every one year thereafter. If the patients experienced symptoms related to ATa recurrence or procedural complications, they were evaluated earlier. Effects of genotypes were analyzed under dominant (wild type vs. heterozygous and homozygous variant), additive (wild type vs. heterozygous variant vs. homozygous variant), and recessive (homozygous variant vs. heterozygous variant and wild type) models.

Results: Patients were followed-up for 30.50 (21.25-41.00) months. Early recurrence was observed in 12 patients (9.3%) and long term recurrence after blanking period developed in 46 patients (35.1%). Hypertension was more frequent (p=0.031), early recurrence was higher (p=0.004), AF duration was longer (p<0.001) and LA diameter was higher (p=0.009) in patients with long term recurrence than those without recurrence. Clinical and procedural characteristics of the patients are presented in Table 1. Relationships between 11 SNPs and ATa recurrence after blanking period in univariate analysis are presented in Table 2. In the additive model, rs3853445 variant in the PITX2 gene was significantly associated with long term recurrence (p=0.014). In the dominant and additive models, the rs751141 variant in the EPHX2 gene (p=0.032 and p= 0.016) and rs3807989 variant in the CAV1 gene (p=0.044 and p=0.038) were significantly associated with long term recurrence. Multivariate analysis showed that rs3807989 variant in the CAV1 gene (p=0.043) in the additive model and early recurrence (p=0.002) predicted long term ATa recurrence after catheter ablation (Table 3).

Conclusions: One genetic variant in the CAV1 gene predicts long term recurrence after catheter ablation for AF. This finding may help to define individuals most likely to benefit from PVI. In addition, early recurrence was predictive for long term recurrence after catheter ablation.

Table 1. Patient characteristics in patients with and without ATa recurrence after blanking period

	Recurrence (-) (n=82)	Recurrence (+) (n=46)	p
Age (years)	58.00 (48.00-62.25)	60.00 (52.75-64.25)	0.323
Sex (Male)	40 (48.8)	19 (41.3)	0.416
BMI (kg/m2)	27.52 (25.71-29.01)	28.03 (26.40-32.22)	0.058
Hypertension (n,%)	39 (47.6)	31 (67.4)	0.031
Diabetes mellitus (n,%)	15 (18.3)	10 (21.7)	0.637
Coronary artery disease (n,%)	11 (13.4)	4 (8.7)	0.426
HF with reduced LVEF (n,%)	5 (6.1)	3 (6.5)	1.000
CHA2DS2-VASc score	1.50 (1.00-2.25)	2.00 (1.00-3.00)	0.338
AF duration (months)	24.00 (18.00-28.00)	30.00 (24.00-36.25)	<0.001
Persistent AF (n,%)	18 (22.0)	14 (30.4)	0.288
Hemoglobin (g/dl)	13.47 ± 1.44	13.67 ± 1.65	0.475
eGFR (mL/min/1.73m2)	90.22 ± 19.43	87.01 ± 18.56	0.364
LA diameter (mm)	38.00 (36.00-40.00)	42.00 (36.75-45.00)	0.009
LVEF (%)	60.00 (60.00-65.00)	61.50 (60.00-65.00)	0.362
Common trunk PV (n,%)	17 (20.7)	16 (34.8)	0.081
Procedure time (min)	75.00 (60.00-90.00)	65.50 (54.00-86.25)	0.123
Fluoroscopy time (min)	14.00 (8.00-19.25)	12.50 (3.00-17.00)	0.110
Early recurrence (n,%)	4 (4.9)	8 (17.4)	0.027
Follow-up (months)	27.00 (19.75-40.25)	33.00 (23.75-43.25)	0.108

AF: Atrial fibrillation, ATa: Atrial tachyarrhythmia, BMI: Body mass index, eGFR: Estimated glomerular filtration rate, HF: Heart failure, LA: Left atrium, LVEF: Left ventricular ejection fraction, PV: Pulmonary vein.

Table 2. Genotype distribution of studied SNPs among subjects subdivided according to ATa recurrence after blanking period

SNP	Recurrence (-) * (%)	Recurrence (+) * (%)	Dominant model		Additive model		Recessive model	
			OR, 95% CI	p	OR, 95% CI	p	OR, 95% CI	p
rs2200733	47.6/ 36.6/ 15.9	34.8/ 43.5/ 21.7	1.65 (0.90-3.03)	0.104	1.63 (0.84-3.16)	0.141	1.27 (0.63-2.57)	0.492
rs10033464	19.5/ 72.0/ 8.5	21.7/ 69.6/ 8.7	0.96 (0.47-1.94)	0.917	0.95 (0.46-1.94)	0.896	1.13 (0.40-3.15)	0.816
rs6838973	40.2/ 50.0/ 9.8	30.4/ 58.7/ 10.9	1.35 (0.27-2.53)	0.349	1.36 (0.71-2.60)	0.342	0.98 (0.38-2.48)	0.965
rs3853445	36.6/ 34.1/ 29.3	21.7/ 60.9/ 17.4	1.88 (0.93-3.80)	0.076	2.47 (1.19-5.09)	0.014	0.57 (0.26-1.23)	0.154
rs17570669	40.2/ 32.9/ 26.8	34.8/ 34.8/ 30.4	1.31 (0.71-2.40)	0.380	1.30 (0.65-2.60)	0.459	1.14 (0.61-2.14)	0.671
rs2106261	24.4/ 72.0/ 3.7	19.6/ 71.7/ 8.7	1.38 (0.66-2.86)	0.384	2.83 (0.86-9.28)	0.085	2.04 (0.73-5.71)	0.173
rs751141	79.3/ 18.3/ 2.4	60.9/ 39.1/ 0	1.91 (1.05-3.46)	0.032	2.07 (1.14-3.75)	0.016	Not analyzed	
rs3807989	24.4/ 56.1/ 19.5	8.7/ 71.7/ 19.6	2.87 (1.03-8.01)	0.044	3.00 (1.06-8.48)	0.038	1.01 (0.48-2.09)	0.980
rs10507248	18.3/ 53.7/ 28.0	23.9/ 52.2/ 23.9	0.78 (0.39-1.54)	0.487	0.80 (0.39-1.63)	0.543	0.87 (0.44-1.73)	0.708
rs1800469	28.0/ 52.4/ 19.5	32.6/ 50.0/ 17.4	1.09 (0.59-2.03)	0.768	0.92 (0.48-1.77)	0.821	0.91 (0.42-1.96)	0.826
rs6795970	51.2/ 17.1/ 31.7	39.1/ 30.4/ 30.4	1.41 (0.78-2.55)	0.253	1.78 (0.88-3.59)	0.104	0.93 (0.50-1.76)	0.844

ATa: Atrial tachyarrhythmia; MAF: Minor allele frequency; SNP: Single nucleotide polymorphism.: Wild type/ polymorphic heterozygous allele/ polymorphic homozygous allele.*

Table 3. Parameters predicting long term ATa recurrence after catheter ablation

	Univariate		Multivariate	
	OR (95% CI)	p	OR (95% CI)	p
Hypertension	1.93 (1.04-3.58)	0.036	0.81 (0.36-1.78)	0.601
Persistent AF	1.35 (0.72-2.54)	0.338	2.23 (0.96-5.14)	0.059
AF duration (months)	1.02 (1.01-1.03)	0.001	1.00 (0.99-1.02)	0.435
LA diameter (mm)	1.10 (1.03-1.17)	0.002	1.07 (0.98-1.17)	0.101
Early recurrence (n,%)	3.69 (1.71-7.98)	0.001	8.06 (2.12-30.55)	0.002
rs3853445_C	2.47 (1.19-5.09)	0.014	1.76 (0.64-4.79)	0.267
rs751141_A	2.07 (1.14-3.75)	0.016	1.24 (0.53-2.87)	0.616
rs3807989_G	3.00 (1.06-8.48)	0.038	4.50 (1.04-19.31)	0.043

AF: Atrial fibrillation, ATa: Atrial tachyarrhythmia, CI: Confidence interval, LA: Left atrium, OR: Odds ratio, SNP: Single nucleotide polymorphism.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-002

Effect of metabolic syndrome on prevalence of early repolarization pattern in Turkish population

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Background and Aim: Early repolarization pattern (ER) in ECG has recently been associated with vulnerability to malignant arrhythmias. Little is known about the association of metabolic factors with ER. We sought to determine the effects of metabolic syndrome (MS) parameters on ER prevalence in Turkish adult population.

Methods: ECGs were obtained 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 seven geographical regions of Turkey. After the exclusion of subjects with complete bundle branch blocks or established coronary artery disease; 3422 subjects ([mean±SD]age, 51±11, [range] 35-100 years) were enrolled (female n [overall%]:1966 [57.5%]). ER was defined as J-point elevation ≥0.1 mV in ≥2 leads in either the inferior [ERI] or lateral [ERL] leads or both [ERIL] with QRS notching. MS was defined according to the NCEP-

ATP III criteria. ECGs were interpreted manually by two experienced cardiologists.
Results: Prevalence of ER was 4.3% in general population (ERI, ERL, ERIL; 2.9%, 0.8%, 0.6% respectively). ER prevalence was significantly higher in men (5.4% vs 3.5% p=0.004) and subjects with ER were younger than without ER (median age±SD; 47±10 vs 50±12 p=0.01). Mean HDL level was significantly lower in ER group (±SEM 41.5±0.9 vs 44.2±0.2 p=0.006). The percentage of subjects who fulfill MS HDL criteria was significantly higher compared with ER (-) while other criteria didn't significantly relate with ER (+) (Table 1). In logistic regression analyses, HDL criteria was an independent predictor of the presence of ER after adjusting for age and gender (OR: 1.86 95% CI:1.20-2.90 p=0.006).
Conclusions: The significant impact of HDL on ER may provide an impetus for further research on metabolic parameters regulating cardiac ion channel regulation mechanisms, especially revealing the mechanisms of the previously demonstrated suppressive effects of hypercholesterolemia on inwardly rectifying potassium (Kir) channel functions.

Table 1.

MS Criteria	ER (+) (%)	ER (-) (%)	p
HDL	67.5	59.7	0.032
Glucose	36.8	34	0.268
HT	50	53.3	0.427
TG	59.7	54.1	0.181
Waist Circumference	42.8	49.9	0.054
MS Diagnosis	49.3	44.7	0.273

%: Percentage of the subjects fulfilling the associated MS criteria within each group.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-003

Genetic variants associated with atrial fibrillation in Turkish patients

Taner Ulus,[1] Muhammet Dural,[1] Pelin Meşe,[1] Furkan Yetmiş,[1] Kadir Uğur Mert,[1] Bülent Görenek,[1] Oğuz Çilingir,[2] Ebru Erzurumluoğlu,[2] Serap Aslan,[2] Sevilhan Artan,[2] Özlem Aykaç,[3] Ertuğrul Çolak,[4] Hikmet Yorgun,[5] Uğur Canpolat,[5] Kudret Aytemir[6]

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Background and Aim: Atrial fibrillation (AF) is the most common sustained arrhythmia in clinical practice. AF can also be encountered in individuals who do not have classical risk factors. Individuals who have AF in one of their parents have been shown to develop AF three-fold more often. These findings suggest that genetic factors may play an important role in the pathogenesis of AF. Genome-wide association studies have revealed that single nucleotide polymorphisms (SNPs) are associated with AF in different societies. However, there is paucity of data regarding genetic predictors related to AF in Turkish patients. In this study, we aimed to investigate if eleven SNPs in the PITX2, ZFHX3, EPHX2, CAV1, TBX5, TGF-1 and SCN10A genes were related to AF.
Methods: A total of 245 consecutive patients with non-valvular AF (44.9% male, mean age: 60.2±13.2 years, 65.3% paroxysmal AF) and 50 age- and sex-matched controls were included. Clinical features and genetic variants were compared between the groups. We analyzed 11 SNPs by using Snapshot technique to identify the associated SNPs with Turkish AF patients. Effects of genotypes were analyzed under dominant (wild type vs. heterozygous and homozygous variant), additive (wild type vs. heterozygous variant vs. homozygous variant), and recessive (homozygous variant vs. heterozygous variant and wild type) models. Logistic regression analysis was used to determine the relationship of genotypes with AF in three different models.
Results: Baseline characteristics of the study population are presented in Table 1. In the dominant and additive models, rs10033464, rs6838973, rs3853445 and rs17570669 variants in the PITX2 gene, rs2106261 variant in the ZFHX3 gene, rs751141 variant in the EPHX2 gene, and rs3807989 variant in the CAV1 gene were significantly associated with AF. In the recessive model, rs17570669 variant in the PITX2 gene was significantly associated with AF. Relationships between 11 SNPs and AF in univariate analysis are presented in Table 2. Multivariate analysis demonstrated that four variants at the PITX2 gene were significantly associated with AF (rs10033464_T: OR 3.29, 95%CI: 1.38-7.82, p=0.007; rs6838973_T: OR 3.06, 95% CI 1.36-6.87, p=0.007; rs3853445_C: OR 2.84, 95%CI: 1.27-6.36, p=0.011; rs17570669_T: OR 4.03, 95% CI: 1.71-9.51, p=0.001) in the dominant model. In addition, LA diameter was significantly associated with AF (OR: 1.16, 95%CI: 1.06-1.27, p=0.001) (Table 3).
Conclusions: There are significant associations between four SNPs in the PITX2 gene and AF (rs10033464, rs6838973, rs3853445 and rs17570669) in Turkish patients. These findings can be used to identify individuals where measures such as blood pressure control and weight control will be applied more tightly to reduce the frequency of AF development.

Table 1. Baseline characteristics of the study population

	Controls (n=50)	AF (n=245)	p value
Age (years)	58.50 (52.75-67.00)	62.00 (53.00-70.00)	0.250
Sex (male) (n,%)	20 (40.0)	110 (44.9)	0.525
BMI (kg/m2)	27.21 (24.87-29.75)	27.60 (25.71-30.47)	0.472
Hypertension (n,%)	22 (44.0)	144 (58.8)	0.055
Diabetes mellitus (n,%)	7 (14.0)	56 (22.9)	0.164
Stable coronary artery disease (n,%)	5 (10.0)	38 (15.5)	0.314
Current smoking (n,%)	5 (10.0)	27 (11.0)	0.833
Alcohol intake (n,%)	2 (4.0)	6 (2.4)	0.627
Hemoglobin (g/dl)	14.01 ± 1.39	13.59 ± 1.60	0.087
eGFR (mL/min/1.73m2)	92.46 ± 24.00	84.37 ± 23.53	0.028
LA dimeter (mm)	36.00 (34.00-38.00)	40.00 (37.00-44.00)	<0.001
LVEF (%)	64.00 (60.00-65.00)	60.00 (60.00-65.00)	<0.001

AF: Atrial fibrillation, BMI: Body mass index, eGFR: Estimated glomerular filtration rate, LA: Left atrium, LVEF: Left ventricular ejection fraction.

Table 2. Relationship between 11 SNPs and AF in univariate analysis

SNP	Reference allele	AF related allele	Dominant model		Additive model		Recessive model	
			OR (95% CI), p	p	OR (95% CI)	p	OR (95% CI)	p
rs2200733	C	T	0.67 (0.36-1.23)	0.197	0.75 (0.38-1.46)	0.402	0.54 (0.22-1.29)	0.170
rs10033464	C	T	3.78 (2.00-7.17)	<0.001	3.03 (1.59-5.77)	0.001	Not analyzed	
rs6838973	C	T	2.76 (1.46-5.23)	0.002	3.29 (1.64-6.57)	0.001	0.64 (0.20-2.06)	0.460
rs3853445	T	C	4.88 (2.58-9.24)	<0.001	4.87 (2.38-9.93)	<0.001	2.17 (0.93-5.07)	0.073
rs17570669	A	T	6.25 (3.25-12.05)	<0.001	13.37 (4.50-39.69)	<0.001	7.15 (2.49-20.53)	<0.001
rs2106261	C	T	2.26 (1.13-4.53)	0.021	2.82 (1.35-5.86)	0.006	0.49 (0.22-1.06)	0.073
rs751141	G	A	2.59 (1.20-5.59)	0.015	2.21 (1.02-4.80)	0.043	Not analyzed	
rs3807989	A	G	3.41 (1.82-6.39)	<0.001	2.69 (1.43-5.07)	0.002	Not analyzed	
rs10507248	G	T	1.25 (0.66-2.36)	0.492	3.07 (0.85-11.09)	0.086	2.96 (0.87-10.00)	0.080
rs1800469	G	A	1.40 (0.69-2.84)	0.344	0.65 (0.31-1.34)	0.246	Not analyzed	
rs6795970	A	G	0.81 (0.44-1.50)	0.518	1.53 (0.60-3.93)	0.369	1.27 (0.53-3.01)	0.587

AF: Atrial fibrillation, CI: Confidence interval, OR: Odds ratio, SNP: Single nucleotide polymorphism.

Table 3. Multivariate analysis of clinical features and SNPs associated with AF

Risk factor	OR (95% CI)	p
Hypertension	0.91 (0.39-2.13)	0.831
eGFR (mL/min/1.73m2)	0.99 (0.97-1.01)	0.696
LA dimeter (mm)	1.16 (1.06-1.27)	0.001
LVEF (%)	0.89 (0.79-1.00)	0.061
rs10033464_T	3.29 (1.38-7.82)	0.007
rs6838973_T	3.06 (1.36-6.87)	0.007
rs3853445_C	2.84 (1.27-6.36)	0.011
rs17570669_T	4.03 (1.71-9.51)	0.001
rs2106261_T	0.71 (0.28-1.79)	0.479
rs751141_A	1.79 (0.69-4.63)	0.224
rs3807989_G	2.03 (0.92-4.46)	0.076

AF: Atrial fibrillation, eGFR: Estimated glomerular filtration rate, LA: Left atrium, LVEF: Left ventricular ejection fraction, SNP: Single nucleotide polymorphism.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-004

P wave duration / P wave voltage ratio plays a strong role for prediction of atrial fibrillation: A new player in the game

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Background and Aim: Atrial fibrillation (AF) is the most common sustained arrhythmia in clinical practice. Identification of patients at risk for developing AF and the opportunity for early targeted intervention might have a significant impact on morbidity and mortality. Prolonged P wave duration and decreased P wave voltage have been shown to be independent predictors of AF. The present study aimed to investigate the role of P wave duration / P wave voltage to predict new-onset AF.
Methods: We screened a total of 640 consecutive patients who admitted to cardiology outpatient clinic with a complaint of palpitation between 2012 and 2014. 24-h holter monitoring, echocardiography, electrocardi-

ography (ECG) recordings were reviewed to identify new-onset AF. Patients were assigned in two groups based on presence (n=150) and absence (n=490) of new-onset AF. Previous ECGs with sinus rhytm were analyzed. P wave duration was measured in inferior leads and P wave voltage was measured in lead one. P wave duration / P wave voltage was also calculated for each patients.

Results: One hundread and fifty subjects (23.4%) had new-onset AF among 640 patients. P wave duration (123.27±12.87 vs 119.33±17.39 ms, p=0.024) and P wave duration / P wave voltage (1284.70±508.03 vs. 924.14±462.06 ms/mv, p<0.001) were higher and P wave voltage (0.12±0.04 vs. 0.13±0.04 mv, p<0.001) was significantly lower in new-onset AF group as compared to non-AF's. P wave duration / P wave voltage had 83.3% sensitivity and 62% spesificity in a receiver operating characteristic curve (AUC 0.728, 95% CI 0.687-0.769; p<0.001). Their negative and positive predictive values were 78.7% and 68.6%, respectively.

In a univariate regression analysis; age, left atrial diameter, left atrial volume index, P wave duration, P wave voltage and P wave duration / P wave voltage were significantly associated with the development of new atrial fibrillation. Moreover, left atrial volume index (OR 6.856, 95% CI 4.265-11.021, p<0.001) and P wave duration / P wave voltage (OR 1.002, 95% CI 1.000-1.003, p=0.008) were found to be significant independent predictors of new-onset AF in a multivariate analysis, after adjusting for other risk parameters.

Conclusions: P wave duration / P wave voltage ratio is a practical, easy to use, cheap and reliable electrocardiographic parameter, which can play a major role for both in predicting and elucidating a mechanism of new-onset AF.

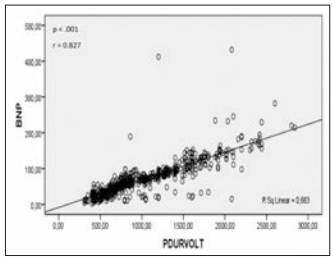

Figure 1. Correlation between P wave duration / P wave voltage and BNP.

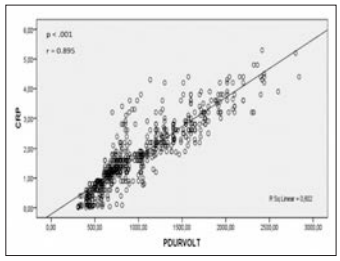

Figure 2. Correlation between P wave duration / P wave voltage and CRP

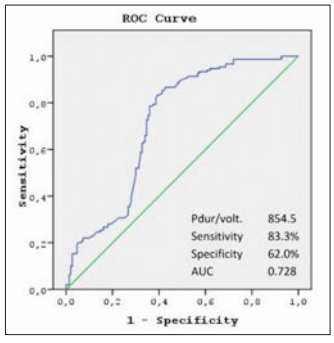

Figure 3. ROC Curve of P wave duration / P wave voltage. *P wave duration / P wave voltage cutoff of 854.5 predicts new-onset atrial fibrillation, with a sensitivity of 83.3% and a specificity of 62.0%. ROC receiver operating characteristic, AUC area under the curve.*

Table 1. Demographical characteristics

	NEW-ONSET ATRIAL FIBRILLATION (n=150)	NON-ATRIAL FIBRILLATION (n=490)	p Value
Age (years)	68.36 ± 7.97	66.98 ± 6.76	0.149
Male Sex	54 (36.0%)	201 (41.0%)	0.272
Diabetes mellitus	50 (33.3%)	196 (40.0%)	0.142
Smoking	67 (44.7%)	147 (30.0%)	0.001
Hypertension	122 (81.3%)	373 (76.1%)	0.182
Hyperlipidemia	15 (10.0%)	32 (6.5%)	0.154
Prior TIA or stroke	9 (6.0%)	20 (4.1%)	0.323
Prior diagnosis of CHF	14 (9.3%)	32 (6.5%)	0.245
Known coronary artery disease	65 (43.3%)	199 (40.6%)	0.554
Peripheral vascular disease	6 (4.0%)	59 (12.0%)	0.004
COPD	34 (22.7%)	126 (25.7%)	0.451
CHA2DS2VASc score	3.453 ± 1.329	3.273 ± 1.432	0.181

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-005

Relationship between left atrial structure and functions and semaphorin4D in patients with paroxysmal atrial fibrillation who had recurrent atrial arrhythmias following catheter ablation procedure

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Background and Aim: Atrial fibrillation (AF) is one of the most commonly seen arrhythmia in worldwide. It, independent from types and duration, may lead to mechanical, structural and electrophysiological atrial remodeling, which results in diastolic or systolic heart failure. A relation between increased left atrial diameter and presence and maintenance of AF was reported in clinical studies. Semaphorin 4D (Sema4D), which is an integral membrane glycoprotein, participated in the pathophysiology of myocardial infarction, heart failure, atrial fibrillation and inflammatory diseases. The aim of this study was to investigate a relationship between left atrial structure and functions and semaphorin4D in patients with paroxysmal AF who had recurrent atrial arrhythmias following catheter ablation (CA) procedure.

Methods: A total of 161 consecutive patients, admitted to outpatient cardiology clinics between January 2017 and 2019 with a diagnosis of paroxysmal AF, were prospectively enrolled in this study. A 101 patients had undergone index circumferential pulmonary vein (PV) radiofrequency ablation for refractory symptomatic paroxysmal AF. Moreover, 60 patients with paroxysmal AF, who had not undergone ablation procedure, and 60 healthy control subjects were included in this study. All participants underwent 2-D transthoracic and transesophageal echocardiographic examinations. Patients were followed-up for 3 months and 1 year respectively from the index CA procedure in terms of recurrence. Serum sema4D concentrations were measured by using sandwich enzyme-labeled immunosorbent assay.

Results: Twenty patients had recurrent atrial arrhythmias after one year from the procedure. Left atrial diameter, left atrial area and left atrial volume index were reported to be significantly raised in the recurrent group as compared to the non-recurrent subjects (p<0.01, p=0.037 and p<0.001 respectively). Sema4D level was importantly higher in the recurrent group than the non-recurrent subjects (p<0.001). A significant positive correlation between sema4D and left atrial volume index was also found in statistical analysis (r=0.51, p<0.013). In multivariate regression analysis, left atrial diameter [odds ratio (OR)=1.12, 95% CI 1.03–1.22; p=0.006) and sema4D (OR=1.93, 95% CI 1.56–2.38; p<0.001) were demonstrated to be significant independent risk factors for recurrence in PAF.

Conclusions: Left atrial volume index, which was positively correlated with sema4D, may help to detect individuals with recurrent atrial events after CA procedure in long term period in PAF. Moreover, it may be used as a practical echocardiographic parameter for risk stratification and follow-up of PAF patients, who undergo CA procedure.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-006

Features of patients with premature ventricular complex ablation: Single center case series

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Background and Aim: In patients who do not respond to medical treatment with idiopathic ventricular extra beat (VEB), catheter ablation is performed using the electroanatomic mapping (3D EAM) system for ablation. The aim of this study is to evaluate the acute and long-term success of patients and the procedural features and complication results associated with VEB localization in patients who underwent catheter ablation in our center.

Methods: 217 patients who underwent activation mapping and ablation using 3D EAM for VEB were included in the study. Patients were followed up for acute procedure success, periprocedural complications, and six-month long-term recurrence. In addition, these parameters, VEBs were evaluated in three groups as right ventricular outflow tract (RVOT), coronary cusp and rare localized origin, and clinical outcomes and interventional variables related to the success of the VEB's location were compared.

Results: In our study, the mean age of the patients was 43±12.1 and the female gender ratio was 37.8% (Table 1). When catheter ablated VEB foci were evaluated, it was seen that 81 (37.3%) were from RVOT and 56 (25.8%) were from coronary cusp. In addition, 6 (2.8%) are aortomitral continuity, 22 (10.1%) are left ventricular summit / epicardial, 17 (7.8%) are parahisian, and total 80 (36.8%) are rare localized VEBs. Acute procedure success was 92.6% and long-term procedure success was 83% in all cases (Table 2). When the patients in our study were analyzed according to their PVC locations and procedure successes, those with rare localization compared to those with RVOT and coronary cusp origin (66 (87.5%), 79 (96.3%), 53 (94.6%) p=0.03) and long-term success. (58 (72.5%), 73 (90.1%), 49 (87.5%) p<0.05, respectively)(Table-3). Long-term transaction success was lower.

Conclusions: Frequent PVCs can be treated with electroanatomic mapping and radiofrequency ablation with high success rate and low complication rate. Patients with RVOT and coronary cusp-derived PVC had a high acute and long-term success rate, while success rates were lower in rare localized PVCs from epicardial / summit, papillary muscle, parahisian and tricuspid-mitral anulus.

Table 1. Demographic features of patients

	All patients n=217
Age	43 ± 12,1
Female Gender,n (%)	82 (37.8)
Tobacco, n (%)	60 (27.6)
Hypertension, n (%)	45(20.7)
Hyperlipidemia, n (%)	45 (20.7)
Diyabetes Mellitus, n (%)	31 (14.3)
LVEF (%)	45.9 ± 8,2

Table 3. Procedural features and complications

	All patients n=217
Ventricular extra beat distribution, n (%)	
Right ventricular outflow tract VEA	81(37.3)
Coronary cuspis VEA	56(25.8)
Rare localized VEA	80 (36.8)
Aorto-mitral continuity	6 (2.8)
Parahisian	17 (7.8)
Left ventricular summit/ Epicardial	22 (10.1)
Papillary muscle	10 (4.6)
Tricuspis annulus	7 (3.2)
Mitral annulus	8 (3.7)
Fascicles	4 (1.8)
Multiple focus	6 (2.8)
Acute procedure success, n (%)	201 (92.6)
Long-term procedure success, n (%)	180 (83)
Procedure Time, minute	81 ± 31
Ablation Time, second	466± 117
Fluoroscopy Time, minute	19 ± 8.7
Tamponade, n (%)	2 (0.9)
Vascular access hematoma, n (%)	9 (4.1)
AV Complete block, n (%)	1(0.4)

Table 3. Clinical results and interventional variables related to the success of the procedure according to the locations of ventricular extra beats

Variables	RVOT (n=81)	Coronary cuspis (n=56)	Rare Localization (n=80)	P value
Acute procedure success	79 (96.3 %) b	53 (94,6%) b	66 (87.5 %) a	0,03
Long-term procedure success	73 (90.1) b	49 (87.5 %) b	58 (72.5 %) a	<0,05
Procedure Time (minute)	71± 13,4 b	64 ± 11,9 b	101 ± 32,7 a	<0,05
Fluoroscopy Time (minute)	15,8 ± 12,7 b	16,7 ± 11,7 b	27,2 ±7,9 a	<0,05
Total ablation Time (second)	407 ± 76 b	391 ± 48 b	812 ± 96 a	<0,05
Total complication rate	3.7 %	3.6 %	5 %	0,18

P <0,05 considered statistically significant. a,b the same letters show no significant difference between groups based on Bonferroni multiple comparison tests.

Table 1. Baseline demographic characteristics of study population

Patient characteristics	Value
Gender, M, n (%)	48 (44)
Age, year, mean ± SD	57.3 ± 14.4
Hypertension, n (%)	49 (45)
DM, n (%)	32 (29.4)
CAD, n (%)	24 (22)
HFrEF or HFpEF, n (%)	10 (9.2)
COPD, n (%)	22 (20.2)
Cancer or taking chemoprophylaxis, n (%)	2 (1.8)
Tisdale risk score, n (%)	
• low (< 7)	93 (85.3)
• moderate (7-10)	12 (11)
• high (≥11)	4 (3.6)
ACEI or ARB, n (%)	31 (28.4)
CCB, n (%)	22 (20.2)
Diuretics, n (%)	27 (24.8)
Ivabradine, n (%)	0 (0)
Ranolazine, n (%)	1 (0.9)
Amiodarone, n (%)	1 (0.9)
Propafenone, n (%)	0 (0)
Favipiravir, n (%)	31 (28.4)
Oseltamivir. n (%)	68 (62.4)
SSRI, n (%)	7 (6.4)
Tocilizumab, n (%)	2 (1.8)

Table 2. Baseline laboratory findings of study population

Parameters	Variables
Hemoglobin, g/dL, mean ± SD	13.07 ± 1.85
Serum creatinine, mg/dL, mean ± SD	0.93 ± 0.38
BUN, mg/dL, mean ± SD	16.82 ± 12.18
eGFR, ml/min, mean ± SD	79.77 ± 24.34
Serum potassium, mmol/L, mean ± SD	4.07 ± 0.50
Serum calcium, mg/dL, mean ± SD	8.95 ± 0.70
Serum magnesium, mg/dL, mean ± SD	1.99 ± 0.23
Serum natrium, mmol/L, mean ± SD	137.12 ± 3.03
CRP, mg/dL, median (IQR)	31.10 (10.31 - 76.09)
Ferritin, mg/dL, median (IQR)	213.39 (68.43 - 417.59)
ESR, mm/h, median (IQR)	28 (18-46)
Procalcitonin, median (IQR)	0.21 (0.09 – 0.35)
Serum albumin, median (IQR)	3.90 (3.53 – 4.10)

BUN- blood urine nitrogen; CRP- C reactive protein; ESR- erythrocyte sedimentation rate; IQR- interquartile range; SD- standard deviation.

ACEI- angiotensin converting enzyme inhibitory; ARB- angiotensin receptor blocker; CAD- coronary artery disease; CCB- calcium channel blocker; COPD- chronic obstructive pulmonary disease; DM- diabetes mellitus; HFpEF- heart failure with preserved ejection fraction; HFrEF- heart failure with reduced ejection fraction; SSRI- selective serotonin receptor inhibitor.

Table 3. Changings in electrocardiographic findings during treatment course

	Baseline ECG	On-treatment first ECG	On-treatment second ECG
Heart rate, bpm, mean ± SD	86 ± 14	77 ± 12	76 ± 12
RR duration, ms, mean ± SD	739.06 ± 128.84	801.59 ± 140.49	816.06 ± 161.21
PR interval, ms, mean ± SD	158.47 ± 25.10	156.35 ± 26.00	155.53 ± 26.77
QRS duration, ms, mean ± SD	94.00 ± 20.55	97.88 ± 21.73	99.18 ± 20.99
QT interval, ms, mean ± SD	370.09 ± 37.15	389.68 ± 42.92	397.88 ± 55.66
QTc interval, ms, mean ± SD			
• by Bazett	435.28 ± 32.78	459.68 ± 38.40	441.91 ± 38.71
• by Fridericia	415.67 ± 28.51	442.30 ± 40.42	426.33 ± 41.19
• by Framingham Heart Study	412.07 ± 25.65	440.97 ± 39.11	426.21 ± 39.68
LBBB, n (%)	4 (3.7)	4 (3.7)	4 (3.7)
RBBB, n (%)	3 (2.8)	3 (2.8)	3 (2.8)
NIVCD, n (%)	4 (3.7)	4 (3.7)	4 (3.7)

ECG- electrocardiogram; LBBB- left bundle branch block; NIVCD - Nonspecific intraventricular conduction delay; QTc- corrected QT; RBBB- right bundle branch block; SD- standard deviation.

Table 4. Comparison of electrocardiographic findings during treatment course

Parameters	Δ1. on-treatment ECG vs. baseline ECG	Δ2. on-treatment vs. Δ1. on-treatment ECG	Δ2. on-treatment ECG vs. baseline ECG
Heart rate, bpm, mean ± SEM	10 ± 1 P<0.001	1 ± 1 P=0.4	10 ± 1 P<0.001
RR duration, ms, mean ± SD	62.53 ± 9.42 P<0.001	14.47 ± 14.17 P=0.29	77 ± 24.95 P=0.009
PR interval, ms, mean ± SD	-2.12 ± 18.90 P=0.65	-0.82 ± 9.79 P=0.73	-2.94 ± 19.93 P=0.55
QRS duration, ms, mean ± SEM	3.88 ± 8.37 P=0.074	1.29 ± 8.51 P=0.54	5.18 ± 8.94 P=0.03
QTc interval, ms, mean ± SEM	24.40 ± 2.99 P<0.001	-17.76 ± 3.94 P<0001	-17.76 ± 3.94 P=0.5
• by Bazett			
• by Frederica	26.64 ± 3.12 P<0.001	-15.96 ± 3.94 P=0.001	-15.96 ± 3.94 P=0.19
• by FHS	28.90 ± 2.97 P<0.001	-14.76 ± 3.65 P=0.001	-14.76 ± 3.65 P=0.16

SD- standard mean; SEM- standard error of mean.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-007

The effect of 5-day course of Hydroxychloroquine and Azithromycin combination on QT interval in non-intensive care unit in patients with coronavirus disease 2019

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Background and Aim: The combination of Hydroxychloroquine (HCQ) and azithromycin showed effectiveness as a treatment for COVID-19 and is being used widely all around the world. Despite that those drugs are known to cause prolonged QT interval individually there is no study assessing the impact of this combination on electrocardiography (ECG). This study aimed to assess the impact of a 5-day course of HCQ and azithromycin combination on ECG in non-ICU COVID19(+) patients

Methods: In this retrospective observational study, We enrolled 109 COVID19(+) patients who required non-ICU hospitalization. All patients received 5-day protocol of HCQ and azithromycin combination. On-treatment ECGs were repeated 3-6h after the second HCQ loading dose and 48-72h after the first dose of the combination. ECGs were assessed in terms of rhythm, PR interval, QRS duration, QT and QTc intervals. Baseline and on-treatment ECG findings were compared. Demographic characteristics, laboratory results were recorded. Daily phone call-visit or bed-side visit were performed by attending physician.

Results: Of the 109 patients included in the study, the mean age was 57.3±14.4 years and 48 (44%) were male. Mean baseline PR interval was 158.47±25.10 ms, QRS duration was 94.00±20.55 ms, QTc interval was 435.28±32.78 ms, 415.67±28.51, 412.07±25.65 according to Bazett's, Fridericia's and Framingham Heart Study formulas respectively. ΔPR was -2.94±19.93 ms (p=0.55), ΔQRS duration was 5.18±8.94 ms (p=0.03). ΔQTc interval was 6.64±9.60 ms (p=0.5), 10.67±9.9 ms (p=0.19), 14.14±9.68 ms (p=0.16) according to Bazett's, Fridericia's and Framingham Heart Study formulas respectively. There were no statistically significant differences between QTc intervals. No ventricular tachycardia, ventricular fibrillation or significant conduction delay was seen during follow-up. There was no death or worsening heart function.

Conclusions: The 5-day course of HCQ- AZM combination did not lead to clinically significant QT prolongation and other conduction delays compared to baseline ECG in non-ICU COVID19(+) patients.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-008

Serum ionized calcium levels are more closely related to the admission QTc İnterval than total calcium levels in patients hospitalized with Covid-19

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Background and Aim: Coronavirus Disease-2019 (COVID-19), declared as a pandemic by the World Health Organization, affects many organs and tissues, especially the lungs. Recent studies have shown that electrolyte abnormalities such as hypocalcemia are common in patients with COVID-19 and associated with hospitalization rates and disease severity. QTc prolongation can be seen in these patients, depending on the effect of the disease, age, gender and comorbidities as well as the drugs used in treatment. Hypocalcemia has been shown in previous studies to prolong the QTc interval. We aimed to investigate the relationship between the admission QTc interval and ionized calcium (IC), corrected total calcium (CTC) levels in patients hospitalized with COVID-19.

Methods: The records of patients hospitalized in our hospital with the diagnosis of COVID-19 between 15 April and 15 May 2020 were screened retrospectively. QTc was measured from the admission ECG. Serum IC data were obtained from blood gas tests and CTC levels were calculated by correcting the calcium levels with albumin at the emergency department laboratory results. The relationship between QTc interval and IC was assessed with multivariable linear regression analysis by adjusting with demographic and clinical predictors (age, gender, heart failure, potassium level, Systemic inflammatory response syndrome (SIRS), myocardial injury, Beta-blocker use).

Results: A total of 132 patients with real-time PCR positivity were included in the study. The mean age of the patients was 50±19 and 62 (47%) were female. Demographic and clinical characteristics were given in table 1. Hypocalcemia (IC<1.15 mmol/l) was observed in 70 (53%) patients. A weak negative correlation was observed between QTc interval and IC, and correlation was not observed with CTC (respectively r=-0.354, r=-0.068) (Figure 1). There was a significant relationship between QTc and IC (β=-2.238, 95% CI-119.974--7.331, p=0.027), age (β=-2,006, 95% CI 0.003-0.486, p=0.047), gender (β=-2.268, 95% CI 1.117-17.247, p=0.025) and SIRS (β=-2.233, 95% CI 1.157-19.276, p=0.027) with regression analysis.

Conclusions: The QT interval reflects ventricular electrical activity. Studies have shown that QTc prolongation was associated with arrhythmias and high mortality rates. It is known that hypocalcemia may prolong the QTc interval. In our study, a significant relationship was observed between QTc and IC, but not with CTC. Drugs such as hydroxychloroquine and azithromycin used in treatment during hospitalization in these patient groups can prolong the QTc interval and QTc prolongation may cause discontinuation of treatment. Considering the significant relationship between QTc and IC, using IC for calcium monitoring and calcium replacement in patients with hypocalcemia may prevent arrhythmias and discontinuation of therapy. In conclusion, hypocalcemia is common in patients with COVID-19, and there was a significant relationship between the admission QTc interval and IC and no association with CTC.

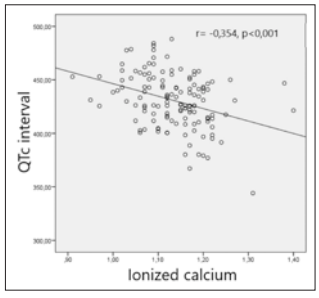

Figure 1. Correlation between QTc interval and Ionized calcium.

Table 1. Demographic and clinical characteristics of patients (n=132)

Age (year)	50±19
Gender (female)	62 (%47)
Hypertension	27 (%20,5)
Smoking	35 (%26,5)
Congestive heart failure	2 (%1,6)
Coronary artery disease	9 (%6,8)
Chronic obstructive pulmonary diease	9 (%6,8)
Myocardial injury	4 (%3,2)
≥2 SIRS criteria	40 (%30)
Radiographic finding of pneumonia	115 (%87)
Lenght of stay hospital (day)	7 (6-9)
Temperature (°C)	37,3 (36,8-3,78)
Systolic blood pressure (mmHg)	110 (100-120)
Diastolic blood pressure (mmHg)	70 (65-75)
White blood cell (10^3/uL)	6,31 (4,73-8,03)
Neutrophil (10^3/uL)	4,31 (2,86-5,63)
Lymphosit (10^3/uL)	1,37 (0,96-1,97)
Hemoglobin g/dL	13,9 (12,8-14,8)
C-reactif protein mg/L	28 (2-74)
D-dimer (ng/mL)	185 (120-316)
Kreatinin (mg/dl)	0,79 (0,69-0,97)
Potassium (mmol/L)	3,98 (3,72-4,28)
Albumin (g/L)	39 (36-43)
Total calcium (mg/dL)	8,50 (8,10-8,90)
Corrected calcium (mg/dL)	8,57 (8,32-8,82)
Ionized calcium(mmol/l)	1,13 (1,08-1,18)
Heart rate (beat/min)	83 (74-91)
QRS duration (ms)	92 (84-100)
QT interval (ms)	355 (334-375)
QTc interval (ms)	431 (414-450)

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-09

A new risk score in the evaluation of left atrial thrombogenicity in atrial fibrillation: The PALSE score

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Background and Aim: Atrial fibrillation (AF) is the most common cardiac arrhythmia encountered in clinical practice, and its thromboembolic complications can cause significant morbidity and mortality. Therefore, the evaluation of thromboembolic risk and the administration of anticoagulant therapy based on this risk are crucial. Together with left atrial thrombus, a vast majority of literature considered moderate to severe SEC as a component of left atrial thrombogenicity (LAT). The current AF guidelines recommend CHA2DS2VASc score in stroke prevention; however, this clinical score gives inadequate information in the setting of LAT evaluation. On the grounds, we aimed to assess the predictors of LAT in patients who were performed TEE with the diagnosis of paroxysmal AF undergoing electrical cardioversion or catheter ablation and compose an effectual risk model for detecting LAT.

Methods: We included a total of 434 patients with non-valvular paroxysmal AF who underwent transoesophageal echocardiography (TEE) prior to cardioversion or catheter ablation.

Results: LAT (+) group was older, and levels of urea, creatinine, total protein, CRP were higher than LAT (-) group. LVEDD, LA diameter, and sPAP levels were higher in LAT (+) group, whereas LVEF was lower than the LAT (-) group. Adjusting with other parameters, age (Odds Ratio (OR): 1.044), total protein (OR:4.234, LVEF (OR: 0.954), LA diameter (OR: 1.080) and sPAP (OR: 1.087) were determined to be independent predictors of the presence of LAT. In the ROC curve analysis, the cut-off values of these parameters were determined and we composed of a risk model abbreviated as PALSE score. To predict LAT, The AUC of PALSE score was 0.833 (95% Confidence Interval: 0.774-0.891, p<0.001). Additionally, PALSE score significantly predicted LA thrombus (AUC: 0.780, 95% Confidence Interval: 0.685-0.876, p<0.001) and moderate - high grade SEC (AUC: 0.847, 95%Confidence Interval: 0.786-0.908, p<0.001). Patients with PALSE score <1 had neither thrombus nor SEC.

Conclusions: In our study, we determined that total protein level, LA diameter, sPAP, age, as well as LVEF were independent predictors of LAT in paroxysmal AF patients undergoing cardioversion or catheter ablation. We also specified the optimal cut-off values of these parameters and composed a risk score, namely PALSE. PALSE Score seemed to predict the presence of LAT accurately. Besides, its predictive ability pertained in either SEC and thrombus, solely. PALSE score Patients with PALSE score <1 had neither thrombus nor SEC in TEE. On the other side, CHA2DS2VASc Score did not predict LAT and demonstrated an unsatisfying utility in this manner.

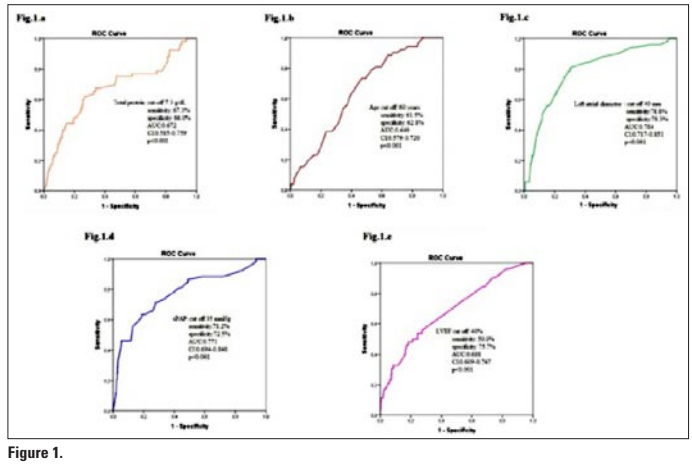

Figure 1.

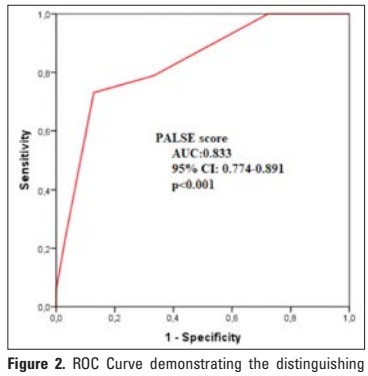

Figure 2. ROC Curve demonstrating the distinguishing ability of PALSE score for left atrial thrombogenity.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-010

Comparision of right ventricular septal and apical pacing: Single center experience

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Background and Aim: Based on current cardiac pacing therapy guideline, the ventricular lead is placed in right ventricular apical (RVA) position in the treatment of atrioventricular block or sinus node disease. This approach successfully imrpoves the life expectancy and quality of life (QoL). On the other hand, it has negative impact on hemodynamic and clinical effects of spontaneous left bundle branch block, new data have emerged showing negative effects of the left bundle branch block-like activation determined by RVA pacing. The aim of our study is To evaluate the safety and efficacy of the permanent high interventricular septal pacing, as alternative to RVA pacing.
Methods: In our study, we retrospectively evaluated 62 patients implanted with a single (37 pts) or dual chamber (25 pts) pacemaker (PM) with ventricular screw-in lead placed at the right ventricular high septal parahisian site (SEPTAL pacing) and 50 patients implanted with a single (26 pts) or dual chamber (24 pts) pacemaker (PM) with ventricular screw-in lead placed at RV apeks. Patients with permanent pacemaker and low percentage of pacing (< 20%) were excluded from the study. All patients had a narrow spontaneous QRS (97 ± 19 ms). We evaluated New York Heart Association (NYHA) class, quality of life (QoL), 6-min walking test (6MWT) and transthoracic echocrdiographic (TEE) measurements.
Results: There was no significant diffrence between apical and septal pacing groups in demographic and clinical characteristics. Pacing parameters were stable during follow up (14 mo/patient). In SEPTAL pacing group we observed an improvement in NYHA class (2.6±1.0 vs. 1.6±0.7, p<0.001), QoL score (26±15 vs. 17±11) and 6MWT (362±85 vs. 449±91, p<0.001). In TEE measurements, there was no significant difference between 2 groups, However, Left atrial volume index (33.0±9.2 vs. 30.3±10.0, p<0.001) was lower in apical pacing group compared with septal pacing group.
Conclusions: RV permanent high septal pacing might be safe and effective in a long term follow up evaluation; it could be a good alternative to the conventional RVA pacing in order to avoid its deleterious effects.

Table 1. Demographic, clinical and laboratory characteristics of the patients

	Right venticular apical pacing pre-implanationgroup at baseline (n:62)	Right venticular apical pacing group at the end-of the follow up (n:62)	Parahisian pacing pre-implantation group at baseline (n:50)	Parahisian pacing group at the end of follow up (n:50)	P value
Age (years)	59.5+16.8		58.8+18.0		0.75
Gender (female) (n%)	26		23		0.63
Body mass index (kg/m2)	27.7+5.6		28.3+5.9		0.55
Smokers (n%)	0.55		21		0.29
NYHA class	2.5+/-0.9	2.2+/-0.8	2.6+/-1.0	1.6+/-0.7	<0.001
6MWT distance (m)	397+/-81	356+/-115	362+/-85	449+/-91	<0.001
QoL score	29+/-14	27+/-16	26+/-15	17+/-11	<0.001
LA volume index ml/m2	31.3+/-10.2	35.3+/-10.9	33+/-9.2	30.3+/-10	<0.001
Left venticular end-diastolic diameter (mm)	49.3+/-10.9	49.0+/-12.2	47.3+/-9.3	46.3+/-9.9	0.16
LV ejection fraction (%)	63.9+/-17.2	62.9+/-15	62.5+/-17.8	63+/-14.8	0.21

LA: Left atrium; LV: Left ventricle; NYHA: New York Heart Association; 6MWT: 6-minute walking test.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-011

Number of parity prolongs QTc interval

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Background and Aim: Pregnancy causes significant changes on cardiovascular system. Hormonal changes, increased plasma volume and sympathetic tone increases the tendency for arrhythmic episodes. Also a series of electrocardiographic changes occur along with the pregnancy. Qtc interval progresses and reaches its maximum level in the third trimester. The reason of QT interval change can be explained by hormonal changes, sympathetic alterations, etc. Gender is also an important factor for QT interval. In female rabbit models, QT interval is longer than male and this difference disappears with the oophorectomization of the rabbit. But estradiol therapy reverses the changes. Also several studies showed that parity number may cause structural changes at heart. In this study, we aimed to determine the effect of the parity number on electrocardiographic parameters.
Methods: 205 women, above 18-years of age, without a history of structural heart diseases, arrhythmia, coronary artery disease were included. ECG measurements were performed manually by two cardiologists. Nulliparous (NP) was defined as women with no delivery history, women with one delivery was defined as primiparous (PP), women with 2 to 5 deliveries were defined as multiparous (MP), women who gave birth 5 to 9 times were defined as grand multiparous (GMP) and women with more than 9 deliveries were defined as great grand multiparous (GGMP).
Results: The mean age of study population was 60.4±10.3. NP constituted 4.9% (n=10), PM constituted 7.8% (n=16), MP constituted 35.6% (n=73), GMP constituted 22.4% (n=46) and GGMP constituted 29.3% (n=60) of the study population. Electrocardiograms of all parity groups were compared in terms of QT interval, QTc interval, Tp-Te interval, Tp-Te/QT ratio, Tp-Te/QTc ratio, and heart rate. There was statistical difference on QT interval (p=0.002) and QTc interval (p=0.000) among all groups. There was no statistical difference on Tp-Te interval, Tp-Te/QT ratio, Tp-Te/QTc ratio and heart rate. Pearson correlation analysis showed that number of parity (p=0.000, r=0.303) and age (p=0.000, r=0.243) are positively correlated with QTc and hypertension (p=0.000, r=-0.231) has negative correlation with QTc. There was statistically significant difference in terms of QT and QTc interval among NP and GGMP, PP and GGMP, MP and GGMP and GMP and GGMP (p<0.05). There was statistically significant difference among MP versus GGMP and GMP versus GGMP in terms of Tp-Te interval (p<0.05). The comparison of the other parameters was not significantly different (p>0.05). Regression models showed that number of parity and GGMP have explanatory power on QTc interval (p<0.05).
Conclusions: In our study QTc interval prolongs as the number of parity increases. This result most probably caused by increased exposure to sex hormones. These hormones may cause irreversible changes among the structure of the heart and effects the cardiac repolarization mechanism of the heart resulting increase in QTc interval.

Table 1. Electrocardiographic parameters stratified by parity category

	Nulliparous No delivery n=10	Primiparous 1 delivery n=16	Multiparous 1 < to 5 deliveries n=73	Grand multiparous 5 < to 9 deliveries n=46	Great grand multiparous 9 < deliveries n=60	p
QT interval, ms	368.8±27.5	379.2±19.2	380.8±24.8	385.1±34.8	399.4±34.3	0.002
QTc Interval, ms	415.9±25.4	418.9±19.5	431.1±25.1	430.3±25.3	443.3±22.8	0.000
Tp-Te interval, ms	91.4±13.5	95.2±14.8	94.0±13.1	92.9±11.4	99.3±16.3	0.108
Tp-Te/QT ratio	0.25±0.03	0.25±0.04	0.25±0.03	0.24±0.03	0.25±0.04	0.943
Tp-Te/QTc ratio	0.22±0.03	0.22±0.03	0.22±0.03	0.22±0.02	0.22±0.04	0.511
Heart rate, bpm (±SD)	77.7±13.7	73.9±9.4	78.0±12.0	76.8±14.0	75.5±12.4	0.698

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-012

Risk factors for atrial fibrillation recurrence in patients undergoing ablation

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Background and Aim: Atrial fibrillation is the most common type of arrhythmia encountered in clinical practice with an estimated prevalence of 1.5 to 2% in developed countries. Atrial fibrillation (AF) ablation is a treatment modality with a low rate of complications in specialized centers that is particularly preferred in patients without structural cardiac disease. The objective of the current study was to investigate the predictors of recurrence in patients with paroxysmal atrial fibrillation undergoing cryoballoon ablation.
Methods: This study was conducted with the participation of 68 patients who underwent cryoballoon ablation at Uludağ University Faculty of Medicine, Cardiology Department between October 2013 and March 2016. Patients' medical records were retrospectively evaluated in electronic setting. Patients were followed for a mean duration of 22 months (range: 8-37 months) with outpatient visits and via telephone calls.
Results: A total of 68 patients undergoing cryoballoon ablation were included in the study. Mean age of the patients was 57.3±12 years, and 32% were male. Concomitant conditions included coronary artery disease (CAD) in 25 patients (36.8%), diabetes mellitus (DM) in 9 (13.2%), hypertension (HT) in 46 (67.6%), and history of cerebrovascular event (CVE) in 3 (4.4%). Left atrium size, left atrial appendage (LAA) flow rate, early AF episode within the first three months, pulmonary anomaly, number of antiarrhythmic drugs and a history of cardioversion were identified as predictors of AF recurrence.
Conclusions: According to the results, post-procedure AF recurrence was found to be associated with early AF development within the first three months, use of multiple antiarrhythmic drugs before the procedure, history of cardioversion, increased left ventricular mass, increased left atrial diameter, reduced flow rate in the left atrial appendage, and pulmonary vein anomaly.

Table 1. Demographics and clinical characteristics of study subjects

	The study group (n=68)
Age (years)	57.3±12
Gender (male)	32 (47.1%)
BMI (kg/m2)	29 (21-39)
BSA (m2)	1.90±0.18
Diabetes mellitus	9 (13.2%)
Hypertension	46 (67.6%)
Hyperlipidemia	6 (8.8%)
Coronary artery disease	25 (36.8%)
Cerebrovascular event	3 (4.4%)
CHA2DS2-VASc score	2.2±1.39
Mean duration of follow up (months)	22 (8-37)
Pulmonary vein anomaly	11 (16.2%)
Number of antiarrhythmic drugs	1.94±0.7
Left ventricular ejection fraction	62±5.7
Left atrial diameter	39.5±6.0
Medical Treatment	
Beta-blocker	47 (69.1%)
Calcium channel blocker	18 (26.5%)
Digoxin	4 (5.8%)
Amiodarone	26 (38.2%)
ACEI/ARB	36 (52.9%)
Sotalol	14 (20.6%)
Propafenone	28 (41.2%)
Warfarin	6 (8.8%)
ASA	37 (54.4%)

BMI: Body mass index, BSA: Body surface area,CHA2DS2-VASc: (heart failure, hypertension, age, diabetes, history of stroke, vascular disease, female gender), ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, ASA: Acetylsalicylic acid.

Table 2. Demographics and clinical characteristics of groups with or without AF recurrence

	No AF recurrence	AF recurrence	P-value
Age	57.5±12.3	61.5±11.5	0.158
Gender (male)	21 (40.4%)	11 (68.8%)	0.084
BSA (m2)	1.90±0.18	1.98±0.17	0.183
BMI (kg/m²)	28.9 (21.5-39.1)	29.2 (25.5-39.8)	0.241
Duration of atrial fibrillation	27.5 (6-100)	34.5 (12-96)	0.397
Diabetes mellitus	6 (11.5%)	3 (18.8%)	0.430
Hypertension	35 (67.3%)	11 (68.8%)	0.914
Hyperlipidemia	5 (9.6%)	1 (6.3%)	1.000
Coronary artery disease	16 (30.8%)	9 (56.3%)	0.65
Cerebrovascular event	2 (3.8%)	1 (6.3%)	0.559
COPD	5 (9.6%)	1 (6.3%)	1.000
Smoking	11 (21%)	7 (43%)	0.105
CHA2DS2-VASc score	2.01±1.32	2.81±1.51	0.077
Pre-procedure AF rhythm	4 (7.7%)	0	0.556
AF episode within the first three months	6 (11.5%)	6 (37.5%)	0.027
Use of amiodarone	18 (34.8%)	8 (50%)	0.268
Total number of antiarrhythmic drugs	1.78±0.7	2.43±0.5	0.002
History of cardioversion	0 (0-3)	1 (0-4)	<0.001

AF: Atrial fibrillation, BMI: body mass index, BSA: body surface area, COPD: Chronic obstructive pulmonary disease, CHA2DS2-VASc: (congestive heart failure, hypertension, age ≥ 75 years, diabetes mellitus, history of stroke, vascular disease, 65-74 years of age, female gender).

Table 3. Distribution of echocardiographic findings across the groups

	No AF recurrence	AF recurrence	P-value
LV mass (g)	181±37.9	195±51	0.028
LV mass index (g/m2)	97±17.7	104±22.3	0.075
LA/BSA (cm/m2	21.4±3	22.1±2.6	0.515
LVEDD (mm)	46±3.5	46±4.3	0.660
Left atrium (mm)	38±5.3	44±6.6	0.003
Septum thickness (mm)	11±1.7	12±1.47	0.008
Posterior wall thickness (mm)	11±0.9	12±1.3	0.007
Ejection fraction (%)	62±3.96	64±9.2	0.306
Mitral annular calcification	6 (11.5%)	1 (6.3%)	1.000
sPAB (mmHg)	25 (19-45)	31 (20-39)	0.111
Left atrial appendage flow rate (cm/sec)	38 (24-62)	28 (22-55)	<0.001

LV: Left ventricle, LVEDD: Left ventricular end diastolic diameter, LA: Left atrium, BSA: Body surface area, sPAB: systemic pulmonary artery pressure.

Table 4. Pulmonary vein anatomy and procedural characteristics in patients undergoing cryoablation

	No AF recurrence	AF recurrence	P-value
Duration of fluoroscopy (min)	26.5±6.2	25±5.8	0.304
Total cooling time (sec)	1920 (960-3200)	1910 (960-3200)	0.836
Pulmonary vein anomaly	5 (9.6%)	6 (37.5%)	0.016
Upper PV freezing temperature, left -°C	48±6.9	50±7.0	0.454
Lower PV freezing temperature, left -°C	45±7.1	48±7.3	0.319
Upper PV freezing temperature, right -°C	48.±6.6	49±6.6	0.922
Lower PV freezing temperature, right -°C	41±5.6	47±6.3	0.001

PV: Pulmonary vein.

Table 5. Distribution of laboratory findings across the groups

	No AF recurrence	AF recurrence	P-value
Creatinine (mg/dL)	0.73±0.24	0.70±0.1	0.919
GFR (mL/min)	109.5±33	113.5±25	0.603
Uric acid (mg/dL)	4.8±1.36	4.2±1.56	0.76
CRP (mg/dL)	0.45±1.71	0.52±0.5	0.939
ESR (mm/h)	10 (2-68)	8 (3-62)	0.467
Leukocyte count (103/ µL)	8±3.16	6.8±3.7	0.452
Hemoglobin (g/dL)	13.2±1.61	13.6±1.52	0.670
LDL cholesterol (mg/dL)	115±43	116±27	0.836
HDL cholesterol (mg/dL)	43±13	36±6.9	0.44
Triglycerides (mg/dL)	115 (52-454)	141 (66-400)	0.517
TSH (µLU/mg)	0.7±1.09	1.56±1.06	0.228

GFR: Glomerular filtration rate, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate, LDL: Low-density lipoprotein, HDL: High-density lipoprotein TSH: Thyroid stimulating hormone.

Table 6. Clinical risk factors associated with late AF recurrence

Risk factor	OR (95% CI)	P-value
AF recurrence within the first three months	39.3 (2.31-671.35)	0.011
Total number of antiarrhythmic drugs with failure	8.33 (1.42-48.95)	0.019

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-013

Correlation between apnea-hypopnea index and Tp-Te interval, Tp-Te/QT, Tp-Te/QTc ratios in obstructive sleep apnea

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Background and Aim: Obstructive sleep apnea (OSA) is a highly prevalent sleep disorder associated with important cardiovascular complications including ventricular arrhythmias. Tp-Te interval, Tp-Te/QT and Tp-Te/QTc ratios are repolarization indices representing ventricular arrhythmogenic potential. These parameters are associated with ventricular arrhythmias and sudden cardiac death. The aim of this study is to investigate the correlation between apnea-hypopnea index and Tp-Te interval, Tp-Te/QT, Tp-Te/QTc ratios in OSA.
Methods: We screened a total of 280 patients who underwent overnight polysomnography (PSG) between the years 2012-2017 at our institution. Patients were assigned into four groups based on severity of apnea-hypopnea index: 70 with apnea-hypopnea index (AHI) < 5 (control group), 70 with 5≤ AHI <15, 70 with 15 ≤ AHI <30, 70 with AHI ≥30. Tp-Te interval, Tp-Te/QT and Tp-Te/QTc ratios were measured.
Results: Compared to control group electrocardiographic repolarization parameters were significantly prolonged in other groups (Tp-Te interval: 68.308±6.757, 71.761±6.300, 79.132±5.544 and 85.105±6.414 ms, p<0.001; Tp-Te/QT ratio: 167.53±12.658, 181.73±13.033, 202.24±9.954 and 219.36±13.451, p<0.001; Tp-Te/QTc ratio: 151.09±16.565, 167.64±16.636, 193.67±14.418, 225.49±16.955, p<0.001). There was a significant trend toward higher Tp-Te levels, Tp-Te/QT and Tp-Te/QTc ratios across higher AHI categories. In a univariate regression analysis, smoking status, Tp-Te interval and Tp-Te/QTc ratio were significantly associated with the severity of AHI in OSA. Tp-Te interval (OR 0.913, 95% CI 0.860-0.969, p=0.003) and Tp-Te/QTc ratio (OR 0.923, 95% CI 0.899-0.948, p<0.001) were found to be significant independent predictors of severity of AHI in a multivariate analysis, after adjusting for other risk parameters.
Conclusions: Our study showed that Tp-Te interval, Tp-Te/QT and Tp-Te/QTc ratios were prolonged in patients with OSA. There was significant correlation between apnea-hypopnea index and these parameters.

Table 1. Electrocardiographic findings of the study population

	Control	AHI < 15	AHI 15-29	AHI ≥ 30	p Value
Heart rate (beats/min)	78.46 ± 7.28	78.29 ± 7.75	78.61 ± 6.95	78.39 ± 7.26	0.995
Tp-Te (ms)	68.308 ± 6.757	71.761 ± 6.300	79.132 ± 5.544	85.105 ± 6.414	<0.001
Tp-Te/QT	167.53 ± 12.658	181.73 ± 13.033	202.24 ± 9.954	219.36 ± 13.451	<0.001
Tp-Te/QTc	151.09 ± 16.565	167.64 ± 16.636	193.67 ± 14.418	225.49 ± 16.955	<0.001

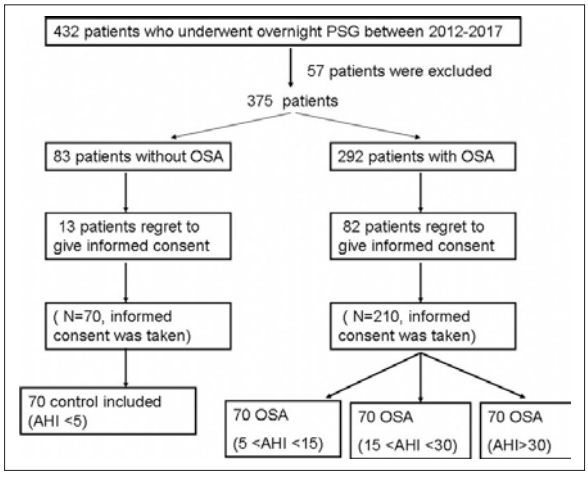

Figure 1. Flow chart of search strategy.

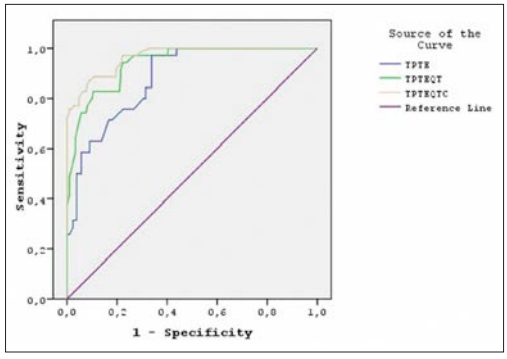

Figure 2. Receiver operating characteristic (ROC) curve comparison of Tp-Te interval, Tp-Te/QT and Tp-Te/QTc ratio for OSA severity prediction.

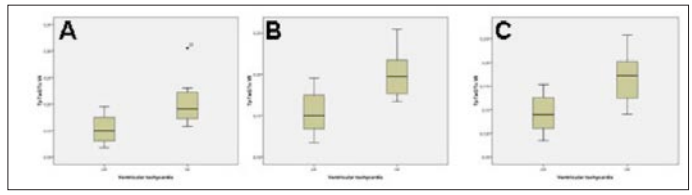

Figure 1. Box-plot graph showing TpTe/QTc V4 (A), TpTe/QTc V5 (B), TpTe/QTc V6 (C) ratio between ventricular tachycardia and non-ventricular tachycardia group.

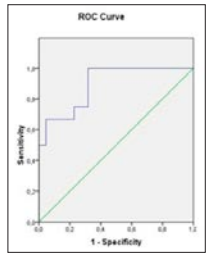

Figure 2. Receiver operating characteristic curve representing the cut-off point of TpTe/QTc V5 ratio in prediction of ventricular tachycardia detected by ICD.

Table 1. Comparison of the ECG parameters between two groups

Parameters	VT group (n=12)	non-VT group (n=22)	p value
HR (beat/min)	67.41 ± 13.35	75.77 ± 14.00	0.976
V4 Tp-Te interval (ms)	91.25 ± 16.25	69.54 ± 12.80	0.001
V5 Tp-Te interval (ms)	91.66 ± 13.20	70.45 ± 11.74	<0.001
V6 Tp-Te interval (ms)	83.75 ± 10.89	66.63 ± 9.35	<0.001
QRS duration (ms)	121.08 ± 40.54	119.95 ± 24.98	0.931
QTc interval (ms)	458.16 ± 43.87	457.27 ± 33.23	0.217
V4 Tp-Te/QTc ratio	20.06 ± 4.08	15.21 ± 2.61	0.002
V5 Tp-Te/QTc ratio	20.09 ± 2.83	15.41 ± 2.36	<0.001
V6 Tp-Te/QTc ratio	18.39 ± 2.61	14.58 ± 1.81	<0.001
Abnormal QRS-T angle (%)	11 (91.6)	11 (50)	0.024

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-014

Association between Tp-Te interval, Tp-Te/QT ratio and incidence of ventricular arrhythmia detected by implantable cardioverter defibrillator

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Background and Aim: Ventricular arrhythmia is the most common reason of sudden cardiac death in patients with heart failure reduced ejection fraction (HFrEF). Transmural dispersion of heterogenous electrical activity is demonstrated as a responsible mechanism of arrhythmogenesis. Tp-Te interval and Tp-Te/QT ratio is used for the assessment of transmural dispersion. Also, in recent years, QRS-T angle which is defined as the difference between ventricular depolarization and repolarization.have gained importance for the prediction of cardiac poor prognosis. We aimed to evaluate the association between Tp-Te interval, Tp-Te/QT ratio, QRS-T angle and ventricular arrhythmia risk in patients with implantable cardioverter defibrillator (ICD).

Methods: Thirty-four patients (mean age was 69.26±11.81) undergoing ICD implantation to prevent sudden cardiac death with HFrEF were included to study, prospectively. Tp-Te interval, Tp-Te/QTc and QRS-T angle were analyzed from 12 lead electrocardiography at the same time with ICD checking. Patients were divided into two group as; ventricular tachycardia (VT) or non-ventricular tachycardia group.

Results: 35.5% (12) of the patients had non-sustained VT. Tp-Te interval in V4, V5 and V6 leads were significantly higher in the VT group (91.25±16.25 vs 69.54±12.80 ms, p=0.001; 91.66±13.20 vs 70.45±11.74 ms, p<0.001; 83.75±10.89 vs 66.63±9.35 ms, p<0.001; respectively). Tp-Te/QTc ratio in lead V4, V5, V6 was higher in VT group (20.06±4.08 vs 15.21±2.61, p=0.002; 20.09±2.83 vs 15.41±2.36, p<0.001; 18.39±2.61 vs 14.58±1.81, p<0.001; respectively). As compared QRS-T angle between two group, VT group had abnormally widened QRS-T angle than non-VT group (p=0.024). Among demographic features and ECG findings, Tp-Te/QTc ratio in lead V5 was the only independent predictor of VT in multivariate logistic regression analysis (OR=3.161, 95% CI=4.240-2.357, p=0.007). Receiver operating characteristic (ROC) curve was used to explore the relationship between TpTe/QTc ratio in lead V5 and VT. The area under the curve (AUC) was 0.894. P<0.0001. Using a cut-off level of ≥17.5, Tp-Te/QTc in V5 was associated with prediction of VT in patients with ICD with a sensitivity of 77% and specificity of 73%.

Conclusions: Prolonged Tp-Te interval and Tp-Te/QTc ratio can be used as a indicator of VT incidence detected by ICD. Tp-Te/QTc in V5 was the only independent predictor of VT detected by ICD. Also, widened QRS-T angle is associated with incidence of VT in patients with ICD.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-016

Assessment of the relationship between semaphorin4D level and recurrence after catheter ablation in paroxysmal atrial fibrillation

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Background and Aim: Atrial fibrillation (AF) is one of the most commonly encountered arrhythmia in clinical practice. Recently, important roles of both inflammation and oxidative stress in pathophysiology of the AF recurrence after catheter ablation (CA) procedure have been reported in clinical studies. Semaphorin4D (Sema4D), a novel integral membrane glycoprotein, plays a role in atherosclerosis, angiogenesis and chronic inflammation. Elevated levels of sema4D were presented in myocardial infarction, heart failure and AF. The aim of this study was to investigate the relation between sema4D and recurrence after CA in paroxysmal AF. Moreover, we aimed to demonstrate an association between sema4D and inflammatory markers in this setting. Finally, significant independent risk parameters for developing of recurrent atrial events after CA in long term period in PAF were investigated in the present study.

Methods: A total of 161 consecutive patients, who admitted to outpatient cardiology clinics of high volume training and research hospital between January 2017 and 2019 with complaints of palpitations, dizziness or syncope and diagnosed with a paroxysmal AF, were prospectively enrolled in this study. A hundread and one of them had undergone index circumferential pulmonary vein (PV) radiofrequency ablation for refractory symptomatic paroxysmal AF. Moreover, 60 patients with paroxysmal AF, who had not undergone ablation procedure, and 60 healthy control subjects were included in the current study. Serum levels of sema4D were measured using the enzyme-labeled immunosorbent assay method. Study participants were followed-up for 3 months and 1 year since CA in terms of recurrence respectively.

Results: While there were 20 patients in the recurrence group, 81 patients had no recurrence after one year from the procedure. Sema4D levels were significantly higher in the PAF group than in the controls (p<0.001). Furthermore, it was importantly increased in the non-ablation group compared to the ablation group (p=0.02). Sema4D levels were significantly elevated in the recurrent group compared to the non-recurrent PAF patients (p<0.001). Sema4D was importantly positively correlated with high sensitive C-reactive protein (r=0.38), p<0.011). In a multivariate analysis, sema4D [odds ratio (OR)=1.93, 95% CI 1.56–2.38; p<0.001] was found to be significant independent risk parameter for recurrence in paroxysmal AF.

Conclusions: We demonstrated a significant association between serum sema4D levels and recurrent atrial events in long term follow-up of patients with AF, who undergone CA procedure. Sema4D is a novel biomarker that may help to identify individuals with recurrent atrial arrhythmia after index CA procedure in long term period in PAF, who are potentially at risk of low quality of life, heart failure, atrial and ventricular arrhythmia and stroke. In addition, sema4D may be used as a significant independent marker for risk stratification and follow-up of PAF patients, who undergo CA treatment.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-018

The effects of Hydroxychloroquine on ECG repolarization parameters of adults being treated for COVID-19 and its relation with clinical poor outcomes: A multicenter clinical cohort study

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Background and Aim: Experimental Hydroxychloroquine (HCQ)/Azithromycin (AZT) combination treatment is a widely accepted experimental treatment for COVID-19 and concerns stated about the potential lethal ventricular arrhythmias (VA). Corrected QT, Tpeak-Tend interval (Tp-e) and QT dispersion have been accepted as novel markers for the assessment of myocardial repolarization and VA. We aimed to evaluate the effects of HCQ±AZT treatment on ECG repolarization parameters among patients treated for COVID-19 and their association with the with poor prognos.

Methods: All consecutive adult patients diagnosed with COVID-19 and hospitalized for treatment with HK-K±AZT in participating centers were evaluated. Exclusion criteria: structural heart disease, Class I/III antiarrhythmic use, complete-bundle-branch-block, high-grade-AV-block, non-sinus rhythms and acute coronary syndrome in follow-up. Bazett qtc corrected tpte… "Poor clinical outcome (PCO)" is defined as a combined definition for any of the following clinical features as in hospital death/>7 days of hospitalization/endotracheal entubation and/or ICU stay.

Results: Of 312 cases, 296 patients (153 females, 56±21 years) were included for analysis. 136 patients also received AZT in addition to HCQ (46% of population, male%:female% 48.5:44 p=0.44). Mean follow up time was 8±5 days (Min-Max 1-35 days). In hospital death was observed in 14 patients (4.7%, 78±17 years) and all were due to multi-organ failure in intensive care unit. PCO occurred in 88 patients (29.7%, mean±SD 64±20 years which was significantly older, p<0.001). Female mortality rate=5.2% while male=4.2% non significant trend for females p=0.7. No lethal VA or any dysrhythmic death was observed in the follow up. QT/QTc intervals and QTdisp were significantly prolonged at the end of the treatment protocol with HCQ±AZT (mean±SD ms change from baseline to the end of the protocol in both sexes = QTc 422±30 to 431±32, p<0.001, QT dispersion-C median ± SEM ms 26±1.4 to 27±1.5 p=). 7.4% (17 cases) >50 ms Delta QTc and. TpTe, TpTe-c, QTd, QTdc and TpTe/QT parameters did not significantly prolong throughout the protocol. However, delta QTc was found to be correlated with and delta QTc >50 ms significantly predicts PCO [(OR 3.8 (95% CI 1.2-12) (p=0.02)]. Presence of prolonged long QT features on ECG at the end of the protocol (p=0.04) and QTdc >50 ms (p=0.04) were significantly associated with PCO.

Conclusions: HCQ/AZT treatment prolongs QTc interval while seemingly exerting no profound effects on surface ECG repolarization parameters. This might be hypothesized as one of the reasons of observed low dysrhythmic events in our cohort of COVID-19 patients. More homogenous transmural repolarization prolongation without evident dispersion of repolarization on human myocardium obsrerved in our cohort with the HCQ use might be protective against the expected deleterious effects of ordinary QT prolonging drugs.

Table 1. Multivariate logistic regression analysis on predictors of poor clinical outcome patients with COVID-19 (+)

Parameters	Odds Ratio	95% CI	p
STEP1			
Age	1,026	1,008 - 1,045	0,005
Gender	1,099	0,589 - 2,051	0,766
HT	0,826	0,387 - 1,761	0,620
Long QT	1,999	0,980 - 4,078	0,057
delta_QTc > 50 ms	3,903	1,243 - 12,249	0,020
STEP2			
Age	1,026	1,008 - 1,045	0,004
HT	0,833	0,391 - 1,772	0,635
Long QT	1,961	0,973 - 3,951	0,060
delta_QTc > 50 ms	3,873	1,235 - 12,149	0,02
STEP3			
Age	1,024	1,009 - 1,039	0,002
Long QT	1,952	0,969 - 3,932	0,061
delta_QTc > 50 ms	3,870	1,233 - 12,149	0,020

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-019

Do sporting activities and using protein supplements change the frontal QRS-T angle?

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Background and Aim: In this study, we investigated whether the frontal QRS-T angle was different between the athletes and normal healthy people.

Methods: The study included 122 healthy athletes (the mean age was 29.7±7.7 years, of them, were 73.8% male) and a control group consisted of 60 healthy people (the mean age was 29.8±7.8 years, of them, were 26% male). Then, the athletes were divided into two groups as who used protein supplements (PS) and those who did not. In the 12-lead ECG, heart rate (HR), P, QRS, QT, corrected QT (QTc) duration, QT and corrected QT dispersion (QTD, QTcD), the sum of V1 or V2S amplitude and V5 or V6R amplitude (V1/2S+V5/6R), frontal QRS-T angle were calculated.

Results: There was no significant difference between the athletes and control groups regarding age, gender, smoking, body mass index, systolic blood pressure (SBP) and diastolic blood pressure (DBP), echocardiographic features, P, PR duration, P, QRS, T axis, QTD and QTcD (p>0.05).HR and QTc were significantly lower (p<0.05) and QRS, QT duration was longer in athletes group (p<0.001). The V1/2S+V5/6R and frontal QRS-T angle values were higher in the athlete's group (p<0.001). There was no significant difference between PS users and non PS users regarding demographic characteristics, duration of sports years, SBP and DBP (p>0.05). However, male gender was dominant in the PS users group (p=0.018). The P axis, PR and QRS duration were longer in the PS users group (p<0.05). It was found that the T axis was negatively correlated (r=-0.431, p<0.001) but the QRS axis was positively correlated (r =0.395, p<0.001) with frontal QRS-T angle.

Conclusions: The frontal QRS-T angle, was found to be wider in athletes compared to normal healthy participants. However, there was no significant difference between who used PS and those who didn't.

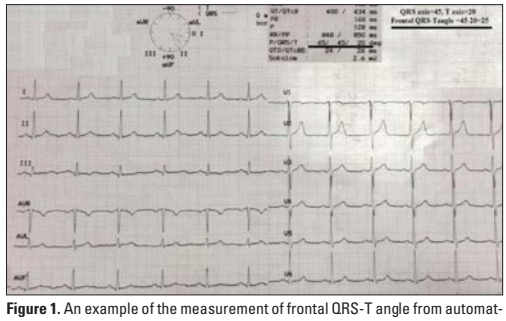

Figure 1. An example of the measurement of frontal QRS-T angle from automatic report of 12-lead surface electrocardiography.

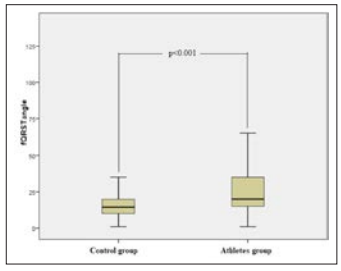

Figure 2. Comparison of frontal QRS-T angle values between control and athletes group.

Table 1. Clinical characteristic of the athletes and control group patients

Variables	Control group (n=60)	Athletes group (n=122)	p-value
Age (years)	30.7 ± 7.8	29.7 ± 7.7	0.398
Sex (men), n (%)	42 (70.0%)	90 (73.8%)	0.086
LVEF (%)	59.2 ± 1.6	59.1 ± 1.3	0.357
HR (bpm)	79.2 ± 10.8	69.1 ± 10.4	<0.001
QRS duration (ms)	92.8 ± 9.3	97.5 ± 9.6	0.002
QT interval (ms)	370.5 ± 27.6	387.9 ± 30.1	<0.001
QTc duration (ms)	424.0 ± 21.0	414.2 ± 22.1	0.007
V1/2S+V5/6R (mV)	18.5 ± 5.08	22.7 ± 5.7	<0.001
Frontal QRS-T angle (x0)	15.4 ± 9.7	26.3 ± 21.4	<0.001

bpm: beats per minute, HR: heart rate, x0: Degree, LVEF: Left ventricular ejection fraction, V1/2S+V5/6R:The sum of V1or 2S+V5 or V6 R.

Table 2. Clinical characteristic of protein supplements-using and non-using athletes

Variables	Non-protein using group (n=78)	Protein using group (n=44)	p-value
Age (years)	29.6 ± 7.5	30.8 ± 8.0	0.219
Sex (men) n (%)	52 (57.8)	38 (42.2)	0.018
LVEF (%)	59.2±1.3	59.5±1.1	0.428
HR (bpm)	69.1 ± 10.7	59.5±1.1	0.990
QRS duration (ms)	96.0 ± 9.5	100.2 ± 9.4	0.019
P axis (x0)	62.1 ± 18.9	75.4 46.2	0.990
PR interval (ms)	145.7 ± 21.1	154.0 ± 23.5	0.047
V1/2S+V5/6R (mV)	22.3 ± 5.9	23.4 ± 5.3	0.298
Frontal QRS-T angle (x0)	23.9 ± 18.5	30.6 ± 25.3	0.099

bpm: beats per minute, HR: heart rate, x0: Degree, LVEF: Left ventricular ejection fraction, V1/2S+V5/6R:The sum of V1or 2S+V5 or V6 R.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-021

Selvester score may be the predictor of ICD therapies in patients with dilated cardiomyopathy

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Background and Aim: Life-threatening ventricular arrhythmias, including sustained ventricular tachycardia (VT) and ventricular fibrillation (VF), are common in patients with systolic heart failure (HF) and dilated cardiomyopathy and may lead to sudden cardiac death (SCD). Primary prevention of SCD refers to medical or interventional therapy undertaken to prevent SCD in patients who have not experienced symptomatic life-threatening sustained VT/VF or sudden cardiac arrest (SCA) but who are felt to be at an increased risk for such an event. The primary prevention of SCD in patients with HF and cardiomyopathy with reduced ejection fraction, either due to coronary heart disease or a dilated nonischemic etiology, will be reviewed here with emphasis on the role of implantable cardioverter-defibrillators (ICDs). However, the benefit of ICD in patients with dilated cardiomyopathy is still an issue under discussion. Twelve-lead electrocardiogram (ECG) is a standard cardiac examination, and is low cost, noninvasive, reproducible, rapid, and usable anywhere. Abnormal findings on ECG such as fragmented QRS or bundle branch block and prolonged QRS duration were reported as prognostic predictors in heart failure patients. In the 1980s, Selvester et al. developed a unique QRS scoring system composed of 32 points, in which each point was allocated 3% of the left ventricular (LV) mass. MRI studies have shown that, ventricular scar tissue size and Selvester score show excellent correlation Studies examining the relationship between ventricular scar tissue and ICD shock with MRI are promising. However, development of a simple, low cost, and noninvasive method for risk stratification isurgently required to reduce healthcare costs, inappropriate ICD shock and to reduce theburden of HF for patients and medical staff ICD are still controversial in patients with dilated cardiopathy.In the light of this information, we aimed to investigate the potential relationship between Selvester score and ICD therapies.

Methods: The study included 48 patients who had undergone ICD implantation with a diagnosis of dilated cardiomyopathy and who had undergone routine 6-month ICD control in outpatient clinic controls between December 2018 and October 2019. Selvester score and other data were compared between patients who received ICD therapy (shock and ATP) and those who did not (Inappropriate therapies were not evaluated).

Results: Selvester score (p<0.001) was higher in ICD therapy group. Mean ejection fraction was lower in ICD therapy group (p=0.019). Positive correlation found between ICD shock therapy and selvester score (p=0.002, r=0.843).

Conclusions: In our study, it was found that high Selvester score may be a predictor for ICD therapies in patients with dilated cardiomyopathy. Indications for ICD are still controversial in patients with dilated cardiopathy. As a cheap and non-invasive method, the Selvester score can help us make decisions in these patients.

Lead	RBBB		LAFB		LAFB+RBBB		LVH		No Confounders		LBBB	
	Criteria	Pts	Criteria	Pts	Criteria	Pts	Criteria	Pts	Criteria	Pts	Criteria	Pts
I	Q≥30 ms	1	Q≥30 ms	1	Q≥30 ms	1	Q≥30 ms	1	Q≥30 ms	1	anyQ	1
	R/Q R≤0.2 mV	1	R/Q R≤0.2 mV	1	R/Q R≤0.2 mV	1	R/Q R≤0.2 mV	1	R/Q R≤0.2 mV	1	R/Q ≤1 R/S ≤1 R/Q ≤1.5 R/S ≤1.5	2 1
II	Q≥40 ms Q≥30 ms	2 1	Q≥40 ms Q≥30 ms	2 1	Q≥40 ms Q≥30 ms	2 1	Q≥40 ms Q≥30 ms	2 1	Q≥40 ms Q≥30 ms	2 1	Q≥40 ms Q≥30 ms R/Q≤0.5 R/S≤ 0.5	2 1
AVL	Q ≥ 30 ms	1	Q ≥40 ms	1	Q ≥40 ms	1	Q ≥40 ms	1	Q ≥30 ms	1	Q≥50 ms Q≥40 ms	2 1
	R/Q ≤1	1	R/Q ≤1	1	R/Q ≤1	1	R/Q ≤1	1	R/Q ≤1	1	R/S ≤0.5 R/Q ≤0.5 R/S ≤1 R/Q ≤1	2 1
AVF	Q≥50 ms Q≥40 ms Q≥30 ms	3 2 1	Q≥50 ms Q≥40 ms Q≥30 ms	3 2 1	Q≥50 ms Q≥40 ms Q≥30 ms	3 2 1	Q≥60 ms Q≥50 ms Q≥40 ms	3 2 1	Q≥50 ms Q≥40 ms Q≥30 ms	3 2 1	Q≥50 ms Q≥40 ms	2 1
	R/Q≤1 R/Q≤2	2 1	R/Q≤1 R/Q≤2	2 1	R/Q≤1 R/Q≤2	2 1	R/Q≤1 R/Q≤2	2 1	R/Q≤1 R/Q≤2	2 1	R/Q ≤0.5 R/S ≤0.5	1
V1 Ant.	Q≥50 ms Any Q InitRs≥0ms	2 1	Any QR	1	Any Q	1	Any QR (or any Q. if*)	1	Any Q	1	Nchnit40 R ≥0.3mV R ≥30ms R ≥0.2mV R ≥20ms	1 2 1
V1 Post**	InitR ≥60ms InitR ≥ 1 mV InitR ≥50ms InitR ≥1.0mV	2 1	R/S >1 InitR ≥60ms R ≥1mV R ≥40ms R ≥0.7mV	1 2 1	InitR ≥60ms InitR ≥ 1 Mv InitR ≥50ms InitR ≥1.0mV	2 1	R/S ≥1 R ≥50ms R ≥1mV R ≥40ms R ≥0.7mV	1 2 1	R/S ≥1 R ≥50ms R ≥1mV R ≥40ms R ≥0.7mV	1 2 1	S/S' ≥3.0 S/S' ≥1.5 S/S' ≥1.25	3 2 1
			Q≥0.2&S≤0.2mV	1			Q≥0.2&S≤0.2mv	1	Q≥0.2&S≤0.2mv	1		
V2 Ant.	Q≥50ms any Q Rs≥0ms Rs≥0.1mV	2 1	Q≥50ms any Q Rs≥0ms Rs≥0.1mV	2 1	any QR (or any Q. 1 if*) Nchinit40	1 1	anyQ Rs≥0ms Rs≥0.1mV	1			Nchinit40 R ≥0.4mV R ≥30ms R ≥0.3mV R ≥20ms	1 2 1
V2 Post.**	InitR ≥70ms InitR ≥ 2.5mV InitR ≥50ms InitR ≥2.0mV	2 1	R/S ≥1.5 R ≥60ms R ≥2mV R ≥50ms R ≥1.5 m	1 2 1	InitR ≥70ms InitR ≥ 2.5mV InitR ≥50ms InitR ≥2.0mV	2 1	R/S ≥1.5 R ≥60ms R ≥2mV R ≥50ms R ≥1.5mV	1 2 1	R/S ≥1.5 R ≥60ms R ≥2mV R ≥50ms Rq1.5mV	1 2 1	S/S' ≥2.5 S/S' ≥2.0 S/S' ≥1.5	3 2 1
			Q≥0.3&S≤0.3mV	1			Q≥0.3&S≤0.3mV	1	Q≥0.3&S≤0.3Mv	1		
V3	Q≥30ms Rs≥0ms Q≥20ms Rs≥0ms	2 1	Q≥30ms Rs≥0ms Q≥20ms Rs≥0ms	2 1	Q≥30ms Rs≥0ms Q≥20ms Rs≥0ms	2 1	QRs(Q≥30ms) Nchinit40 anyQR (or anyQ if*)	2 1	Q≥30ms Rs≥0ms Q≥20ms Rs≥0ms	2 1		
V4	Q≥20ms R/Q ≤0.5 R/S ≤0.5 R/Q ≤1 R/S ≤1 R ≤0.5mV Ntchinit40	1 2 1	Q≥20ms R/Q ≤0.5 R/S ≤0.5 R/Q ≤1 R/S ≤1 R ≤0.5mV Ntchinit40	1 2 1	Q≥20ms R/Q ≤0.5 R/S ≤0.5 R/Q ≤1 R/S ≤1 R ≤0.5mV Ntchinit40	1 2 1	Q≥20ms R/Q ≤0.5 R/S ≤0.5 R/Q ≤1 R/S ≤1 R ≤0.5mV Nchinit40	1 2 1	Q≥20ms R/Q ≤0.5 R/S ≤0.5 R/Q ≤1 R/S ≤1 R ≤0.5mV Nchinit40	1 2 1		
V5	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤2 R/S ≤2 R ≤0.6mV Ntchinit40	1 2 1	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤2 R/S ≤1.5 R ≤0.6mV Ntchinit40	1 2 1	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤2 R/S ≤1.5 R ≤0.6mV Ntchinit40	1 2 1	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤2 R/S ≤2 R ≤0.6mV Nchinit40	1 2 1	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤2 R/S ≤2 R ≤0.6mV Nchinit40	1 2 1	anyQ R/R' ≥2 R/R' ≥1 R/S ≤2 Rs0.6Mv	1 2 1 1
V6	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤3 R/S ≤3 R ≤0.6mV Nchinit40	1 2 1	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤3 R/S ≤2 R ≤0.6mV Nchinit40	1 2 1	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤3 R/S ≤2 R ≤0.6mV Nchinit40	1 2 1	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤3 R/S ≤3 R ≤0.6mV Nchinit40	1 2 1	Q≥30ms R/Q ≤1 R/S ≤1 R/Q ≤3 R/S ≤3 R ≤0.6mV Nchinit40	1 2 1	Q≥20ms R/R' ≥2 R/R' ≥1 R/S ≤2 Rs0.6Mv	1 2 1 1
Total	Points		Points		Points		Points		Points		Points	

Figure 1. Selvester score chart.

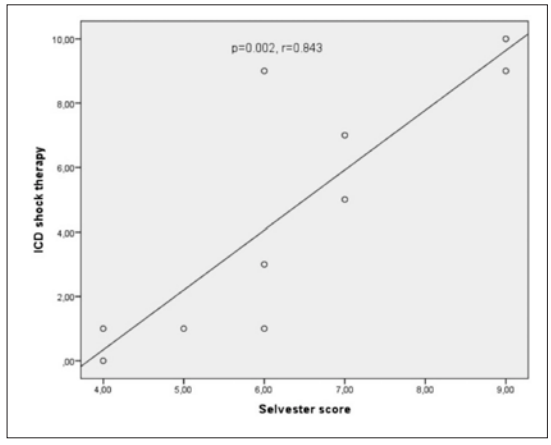

Figure 2. Correlation Between selvester score and ICD shock therapies.

Table 1. Demographic, echocardiographic and drug use characteristics of patients

Variables	ICD Therapy (+) (n=10)	ICD Therapy (-) (n=38)	p value
Age,years	65.3 ± 8.6	61.2 ± 10.7	0.251
Female, n (%)	1 (10.0)	8 (21.1)	0.426
Hypertension, n (%)	3 (30.0)	10 (26.3)	0.859
Diabetes Mellitus, n (%)	2 (20.0)	9 (23.7)	0.805
Ejection Fraction, (%)	26.3 ± 4.6	29.4 ± 7.2	0.019
Mean NYHA Score	2.00 ± 0.9	1.82 ± 0.8	0.522
Usage of Beta Blockers, n (%)	10 (100.0)	34 (89.5)	0.284
Usage of ACEi/ARB, n (%)	7 (70.0)	30 (78.9)	0.549
Usage of Sacubitril-Valsartan, n (%)	1 (10.0)	2 (5.3)	0.582
Usage of 0.251eralocorticoid antagonist, n (%)	6 (60.0)	24 (63.2)	0.854
Usage of Diuretic, n (%)	5 (50.0)	23 (60.5)	0.548
Usage of Digoxin, n (%)	2 (20.0)	10 (26.3)	0.682
Usage of Ivabradine, n (%)	1 (10.0)	4 (10.5)	0.961
Usage of Amiodarone, n (%)	1 (10.0)	2 (5.3)	0.582
Usage of Mexiletine, n (%)	0 (0.0)	0 (0.0)	0.403
Usage of other antiarrhythmics, n (%)	0 (0.0)	0 (0.0)	

Data are given as mean ± SD, n or median (interquartile range). NYHA, New York Heart Association Classification score; ACEi/ARB, angiotensin converting enzyme inhibitors and angiotensin receptor blockers.

Table 2. ECG and ICD parameters of the patients

Variables	ICD Therapy (+) (n=10)	ICD Therapy (-) (n=38)	p value
Heart Rate, bpm	72.5 ± 8.9	73.5 ± 13.3	0.815
QRS duration, ms	65.3 ± 8.6	61.2 ± 10.7	0.251
CRT device, n (%)	2 (20.0)	6 (15.8)	0.751
VT 1 zone, ms	340 ± 20	335 ± 60	0.826
VT 2 zone, ms	305 ± 20	303 ± 42	0.831
VF zone, ms	292 ± 9	293 ± 16	0.807
Monitored Non-sustained VT episodes	10.6 ± 2	1.6 ± 1	<0.001
ATP therapies	1		
Shock	3		
Shock After ATP	2		
Shock and ATP in different episodes	4		
Selvester Score	6.3 ± 1.8	4.1 ± 2.4	< 0.001
Sinus rhythm	6 (60.0%)	20 (52.6%)	0.677
Left bundle branch block	2 (20.0)	6 (15.8)	0.751
Left anterior fascicular block	1 (10.0%)	3 (7.9%)	0.709
Left posterior fascicular block	0 (0.0%)	2 (5.3%)	0.459
Right bundle branch block	1 (10.0%)	6 (15.8)	0.644
Right bundle branch block + Left anterior fascicular block	0 (0.0%)	1 (2.6%)	0.604

Data are given as mean ± SD, n or median (interquartile range). CRT, Cardiac Resynchronization Therapy; VT, ventricular tachycardia; VF, ventricular fibrillation; ATP, antitachycardia pacing.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-022

Impaired repolarization parameters may predict fatal ventricular arrhythmias in patients with hypertrophic cardiomyopathy

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Background and Aim: Hypertrophic cardiomyopathy (HCM) as a common genetic heart disease characterized by ventricular hypertrophy and myocardial fibrosis is significantly associated with a higher risk of fatal ventricular arrhythmias (VAs). Frontal QRST angle (fQRSTa) is a marker of ventricular repolarization abnormalities. In addition, Tp–e interval, Tp–e/QTc ratio are described as ventricular repolarization parameters, and they are found related to arrhythmias. In this study, we aimed to investigate the predictive value of repolarization parameters for fatal VAs in patients with HCM.

Methods: A total of 127 HCM patients (mean age: 47.9±12.6 years; male:79) were enrolled in this retrospective study. All patients underwent two-dimensional and M-mode transthoracic echocardiography. Moreover, electrocardiograms within 3 months prior to the VA documentation were assessed. The primary outcome was the occurrence of fatal VAs including sustained ventricular tachycardia and ventricular fibrillation.

Results: There were documented sustained VAs in 37 (29.1%) patients during a mean follow-up time of 70.1±22.6 months. The prevalence of sustained VAs was significantly higher in patients with fQRSTa ≥140 degrees (67.4 vs. 7.4 %; p<0.001). In addition, prevalence of sustained VAs was significantly higher in patients with Tpe/QTc ratio ≥0.19 (61.5 vs. 6.7%; p<0.001). High Tpe/QTc ratio (hazard ratio: 1.564; 95% confidence interval: 1.086-4.796; p=0.032) and high fQRSTa (hazard ratio: 1.864; 95% confidence interval: 1.106-8.745; p=0.002) were found to be independent predictors of sustained VAs in HCM patients.

Conclusions: Wider fQRSTa, prolonged Tp–e interval, and increased Tp–e/QTc ratio were independently associated with fatal VAs in HCM patients. In addition to traditional risk factors, simple ECG parameters can provide clinicians with valuable information when evaluating SCD risk in the HCM population. However, these parameters should be confirmed by larger-scale studies.

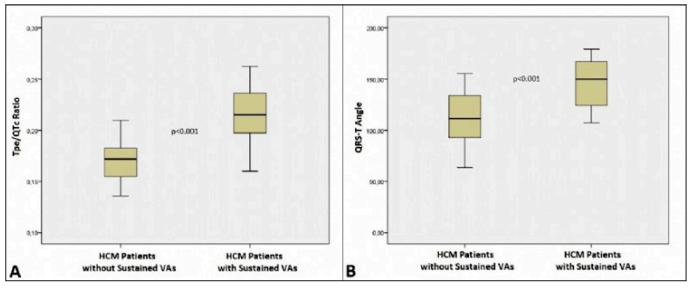
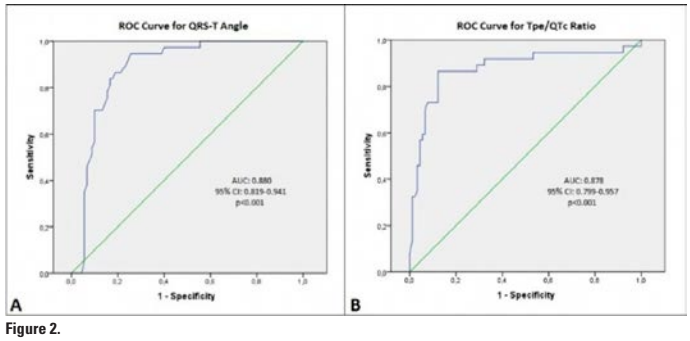


Figure 1.



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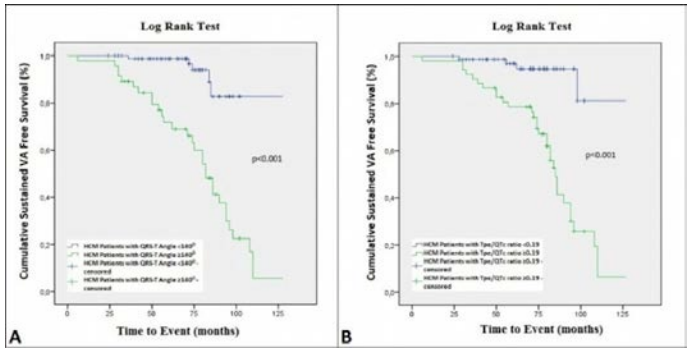

Figure 3.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-023

A retrospective analysis of cardiac device infections, risk factors, diagnosis, treatment and follow-up results

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Background and Aim: Permanent pacemaker infections are rare but serious complications of permanent pacemaker implantation. Current guidelines recommend the removal of all system components for patients with infected cardiac devices. In the present study, we aimed to contribute to the literature regarding cardiac device infections and the management.

Methods: This retrospective study included a total of 1705 patients to whom permanent pacemaker had been implanted in the cardiology department of Ondokuz Mayis University, Faculty of Medicine between January 2005 and June 2014. All clinical and laboratory data of the patients were collected by investigating the patient files and the database.

Results: Permanent pacemaker infection rate was 3%. Cardiac device infection rate was higher in patients with heart failure as the indication for implantation (p=0.001) and the ones cardiac resyncronization therapy (CRT) had been applied (p=0.015). Battery replacement was not found to be a risk factor for device infections in our study. The most common clinic was battery pocket infection. The most common organisms were Staphylococci in blood and wound cultures. The patients whom permanent pacemaker system had been completely removed showed significantly lower recurrence rates (p=0.001). The use of temporary pacemaker didn't increase recurrent infections, but temporary pacemaker had been used in a small number of patients.

Conclusions: In the present study, we demonstrated that patients with heart failure as the indication for pacemaker implantation and the ones CRT had been applied had higher rates of cardiac device infections. In case of cardiac device infection, total extraction which can be performed percutaneously, combined with an appropriate duration of antibiotics may prevent recurrent infections.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-024

The effect of different therapies on myocardial repolarization parameters and P-wave dispersion in chronic kidney disease

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Background and Aim: Chronic kidney disease (CKD) patients are higher risk for cardiac arrhythmias. The risk of arrhythmia may change with different treatment modalities. ECG is a simple and useful method to evaluate risk. We aimed to compare the effects of different treatment strategies on myocardial repolarization parameters (Tp-e, QT, QTc intervals, Tp-e/QT, Tp-e/QTc ratios) and P-wave dispersion (PWD) in patients with CKD.

Methods: Three groups were created from the patients aged between 18-65 years, as Group-1 including CKD patients receiving HD 3 times a week, Group-2 consisting of pre-dialysis CKD patients and Group-3 including CKD patients who underwent a successful transplantation. All patients' basic demographic data, risk factors, echocardiographic parameters recorded. The risk of cardiac arrhythmia was evaluated by calculating the electrocardiographic repolarization parameters and P wave dispersion were analysed.

Results: The PR, QT, QTc intervals were significantly shorter in the transplantation group compared to the other groups (p=0.020, p<0.001, p=0.035; respectively). While Tp-e interval, Tp-e/QT, Tp-e/QTc ratios were significantly higher in the pre-dialysis group compared to the other groups (p<0.001, p<0.001, p=0.001; respectively), no important variation was observed between the haemodialysis and transplantation groups (p>0.05). PWD was significantly increased in the pre-dialysis group compared to other two groups (p<0.001), while no important variation was found between the haemodialysis and transplantation groups (Table 1, Figure 1).

Conclusions: We found that Tp-e interval, Tp-e/QT, Tp-e/QTc ratios, PWD were significantly higher in pre-dialysis CKD group, but PR, QT, QTc intervals were significantly shorter in transplantation group compare to other groups. The prognostic significance and predictive to the arrhythmic events these parameters in CKD patients require further evaluation with long-term follow-up and large-scale prospective studies.

Table 1.

Variable	Hemodialysis group (n=40)	Pre-dialysis CKD group (n=40)	Transplantation group (n=40)	P value
Heart Rate, bpm	75 (50 - 116)	77 (50 - 117)	77 (50 - 109)	0.281
PR (msn)	160 (100 - 200) b	159 (112 - 205) b	145.5 (104 - 205) a	0.020
QRS (msn)	84 (65 - 112)	84.5 (68 - 110)	84.5 (68 - 106)	0.970
QT (msn)	384 (300 - 450) b	368 (304 - 440) b	358 (258 - 482) a	<0.001
QTc (msn)	421 (375 - 524) b	423.5 (362-514) b	413.5 (338 - 536) a	0.035
Tp-e interval (msn)	85 (60 - 120) a	100 (60 - 120) b	82 (60 - 120) a	<0.001
Tp-e/QT ratio	0.23 (0.14 – 0.33) a	0.26 (0.20 – 0.32) b	0.24 (0.15 – 0.34) a	<0.001
Tp-e/QTc ratio	0.21 (0.13 – 0.29) a	0.23 (0.18 – 0.29) b	0.21 (0.14 – 0.35) a	0.001
Pmax. (msn)	120 (80 - 140) ab	122 (90 - 165) b	120 (80 - 145) a	0.009
Pmin. (msn)	70 (45 - 95) a	60 (45 - 85) b	65 (43 - 80) ab	0.001
PWD (msn)	50 (20 - 75) a	65 (40 - 108) b	50 (30 - 80) a	<0.001

Myocardial repolarization parameters and P wave dispersion of CKD patients according to the hemodialysis, pre-dialysis and transplantation groups. a-b: In pairwise comparisons between groups, there is no difference between groups with the same character.

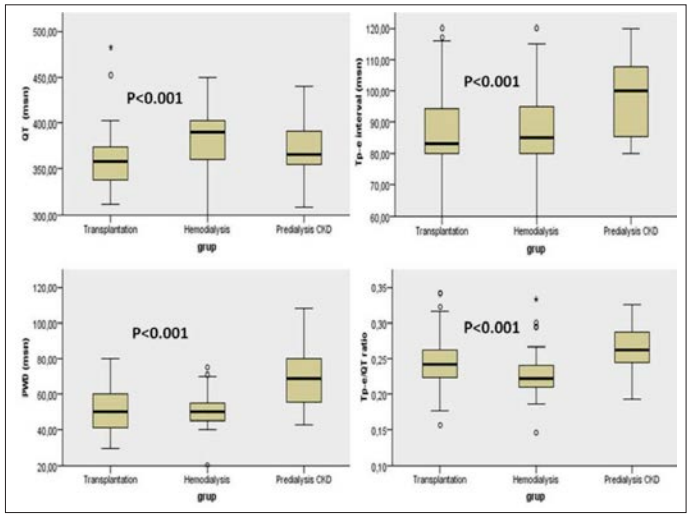

Figure 1. Imaging of distribution the repolarization parameters and P wave dispersion according to the hemodialysis, pre-dialysis and transplantation groups.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-025

Single Center single operator experience with cardiac device infection: Importance of periprocedural precautions

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Background and Aim: While technological developments in the field of interventional cardiology increase the life expectancy of cardiology patients, the number of patients who have permanent pacemakers and implantable cardioverter defibrillators is increasing day by day due to the needs of surviving patients. The increase in the number of patients with cardiac devices increases the complications related to the procedure. Device-related infections head these complications due to their high mortality and morbidity rates. The frequency of device-related infections varies from center to center, between 1% and 12.5%. The rates of cardiac device-related infections and the effect of periprocedural protection measures on device-related infections between November 2019 and May 2019 in our center were investigated.
Methods: Patients who had undergone implantation of permanent pacemakers and implantable cardioverter defibrillators between November 2019 and May 2019 were retrospectively examined, and the presence of device-related infections was retrospectively scanned in their 1- and 3-month follow-ups.
Results: A total of 103 patients were included in the study The patients were ≥75 (n=34), 65-75 (n=37), and <65 (n=32) years of age, while 31% of the patients were admitted from the emergency service. Pacemakers was implanted in 47, implantable cardioverter defibrillators in 42 and a 3-chamber implantable cardioverter defibrillators in 14 patients. Seventy-six patients received the first procedure, 21 patients underwent battery replacement, and 6 patients lead implantation and battery replacement. Aesthetic stitches were applied in 93.2%, and matrix stitches in the remaining patients. Subcutaneous stitches of all patients were closed with continuous sutures. A battery pocket was opened under the muscle in 4 and under the pectoral fascia in other patients. All patients were discharged with a pressure dressing and non-steroidal anti-inflammatory and narcotic drugs were used for pain control. Hematoma was observed in the battery pocket of four patients and a battery pocket infection was seen in one patient.
Conclusions: Battery infection rate is low in procedures performed in our hospital. We think that this situation is due to working under antibiotic prophylaxis in accordance with periprocedural measures and in collaboration with the department of infectious diseases. We think that working with a single team increases the effectiveness of the implementation of periprocedural measures.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-027

Assesment of total atrial conduction time in patients with atrioventricular nodal reentrant tachycardia and control subjects

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Background and Aim: Most forms of atrioventricular nodal reentrant tachycardia (AVNRT) are created by reentry between 2 (or more) atrial connections to the AV node. Atrial vulnerability and functional remodeling that leads to reentrant circuits in AVNRT is largely unknown. PA-TDI interval assessed by tissue Doppler imaging is a useful technique to determine total atrial conduction time (TACT) which reflects atrial remodeling and arrhythmic substrate. In this prospective, case – control study, we aimed to assess TACT in patients with AVNRT and control subjects.
Methods: Study population consisted of 62 consecutive patients (age 44±12 years; 74% women) undergoing electrophysiological study and ablation for symptomatic, drug-resistant AVNRT and 42 age and sex matched control subjects. All patients and control subjects underwent tissue Doppler imaging for assesment of TACT.
Results: Left atrial volume index and left atrial phasic functions were similar in patients with AVNRT and control subjects. PA-TDI interval was longer in patients with AVNRT compared to control subjects (121±13 ms vs 105±11 ms; p<0.001). PA-TDI interval was significantly correlated with P-wave duration on the surface ECG (p=0.02).
Conclusions: TACT is prolonged in patients with AVNRT. Further studies are needed to determine the underlying factor(s) such as AVNRT that leads to prolonged atrial conduction time.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-028

Tpe interval predicts good neurological recovery in ischemic stroke patients treated with fibrinolytic therapy

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Background and Aim: Ischemic stroke is a significant cause of cardiovascular morbidity, and mortality and prognosis is generally worse without clot directed reperfusion therapy. T wave peak-to-end (Tpe) interval is a marker of ventricular repolarization and also found to be related to cerebrovascular disorders. In this study, we aimed to search the association between Tpe interval at admission and neurological recovery success in ischemic stroke patients managed with fibrinolytic therapy.
Methods: Ischemic stroke patients were searched from the hospital computer database, and Tpe intervals at admission electrocardiography (ECG) recordings were measured. We searched the association between NIH Stroke Scores (NIHSS) and Tpe intervals and Tpe/QTc ratios. Modified Rankin Scores (mRS) at discharge were calculated, and successful functional recovery was defined for the patients with mRS between 0-2. Patients with a rhythm of atrial fibrillation at admission, with hemorrhagic transformation after fibrinolytic therapy or without an interpretable ECG recording were excluded from statistical evaluation.
Results: Fifty-five patients were involved in the study population. The mean age was 64.7±11.9 years, and the male gender rate was 61.8% (34 patients). Mean NIHSS at admission was 10.1±5.3, and there was a moderate, positive correlation between admission NIHSS and Tpe interval (r=0.325, p=0.015). Twenty-four patients (43.6%) were evaluated as mRS 0-2 and defined as to reach a good functional recovery. In ROC curve analysis, a cut-off value of 89.0 ms for admission Tpe interval predicted successful reperfusion with fibrinolytic therapy with a sensitivity of 70.8% and a specificity of 54.8% (AUC=0.698; 95% CI [0.559-0.837]; p=0.012) (Figure-1).
Conclusions: Shorter Tpe interval on surface ECG at admission can predict a higher reperfusion success and a better neurological recovery in ischemic stroke patients treated with fibrinolytic therapy.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-029

Cardiac electrophysiological procedures during the Covid-19 pandemic: A single center experience

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Background and Aim: The aim of this study is to report our experience on electrophysiological (EP) procedures during the Coronavirus disease of 2019 (Covid-19) pandemic and compare the results with findings from a similar timeframe from the previous year.
Methods: Patients who underwent an EP procedure during the Covid-19 pandemic in our hospital between dates March 20, 2020 and May 1, 2020 constituted the study group. Patients who underwent an EP procedure between dates March 20, 2019 and May 1, 2019 constituted the control group. Baseline characteristics and the indication for EP procedure between the groups were compared.
Results: Compared to previous year, there was a 61.5% reduction in the total number of EP procedures during the Covid-19 pandemic. Compared to control group, the number of VT ablation was higher in the study group (22.7% vs. 5.3%, p=0.001). The number of AF ablation (16.7% vs. 2.3%, p=0.015) and PVC ablation (9.6% vs. 0%, p=0.033) were higher in the control group. The number of PM implantation was higher during the Covid-19 pandemic compared to the control group (13.6% vs 2.6%, p=0.007). Although the number of ICD implantation was similar between the groups (15.9% vs 14.9%, p=0.875), more patients in the study group had ICD implantation for secondary prevention (11.4% vs 4.5%). There was no CRT implantation performed in Covid-19 pandemic period.
Conclusions: The number of EP procedures is decreased and there are important differences in the indications of EP procedures during the Covid-19 pandemic compared to routine practice.

Table 1. The comparison of electrophysiological procedures between the groups

	Covid-19 Pandemic Period (n=44)	Control Group (n=114)	p value
Diagnostic EPS, n(%)	3 (6.8%)	16(14.0%)	0.211
Supraventricular Tachycardia Ablation, n(%)	11(25%)	30(26.3%)	0.866
Ventricular Tachycardia Ablation, n(%)	10 (22.7%)	6(5.3%)	**0.001**
Ventricular Extrasystole Ablation, n(%)	0(0%)	11(9.6%)	**0.033**
Atrial Fibrillation Ablation, n(%)	1(2.3%)	19(16.7%)	**0.015**
Pacemaker Implantation, n(%)	6(13.6%)	3(2.6%)	**0.007**
ICD Implantation, n(%)	7(15.9%)	17(14.9%)	0.875
CRT Implantation, n(%)	0(0%)	9(7.9%)	0.055
Pacemaker or ICD Generator Replacement, n(%)	6(13.6%)	12(10.5%)	0.581

* Chi-square Test, Fishes's Exact Test *p<0.05 statistically significant. Categorical variables are reported n(%).
Abbreviations: CRT; Cardiac Resynchronization Therapy, EPS; Electrophysiological Study, ICD; Implantable Cardioverter Defibrillator

Table 2. The comparison regarding triage of electrophysiological procedures between

	Covid-19 Pandemic Period (n=44)	Control Group (n=114)	p value
URGENT PROCEDURES	**17 (38.6%)**	**10(8.7%)**	**<0.001**
VT Ablation for Electrical Storm	7(15.9%)	3(2.6%)	**0.002**
PM Implantation for Complete AV Block	3(6.8%)	2(1.8%)	0.132
ICD Implantation for Secondary Prevention	5(11.4%)	2(1.8%)	**0.009**
PM or ICD Generator Replacement in PM Dependent Patient	2(4.5%)	3(2.6%)	0.619
SEMI-URGENT PROCEDURES	**24 (54.5%)**	**18 (15.8%)**	**<0.001**
VT Ablation other than Electrical Storm	3(6.8%)	3(2.6%)	0.349
SVT Ablation in Highly Symptomatic Patients and had Repeated Emergency Service Admissions	11(25%)	0(0%)	**<0.001**
AF Ablation in a Patient with Rapid Ventricular Rate During AF Causing Hemodynamic Compromise	1(2.3%)	0(0%)	0.278
PM Implantation for SSS + Syncope	3(6.8%)	0(0%)	**0.021**
ICD Implantation for Primary Prevention	2(4.5%)	6 (5.3%)	0.853
PM or ICD Generator Replacement in non-PM Dependent Patient	4(9.1%)	9(7.9%)	0.806
ELECTIVE PROCEDURES	**3(6.8%)**	**86 (75.4%)**	**<0.001**
SVT Ablation	0(0%)	30(26.3%)	**<0.001**
Atrial Fibrillation Ablation	0(0%)	19(16.7%)	**0.004**
PVC Ablation	0(0%)	11(9.6%)	**0.033**
Diagnostic EPS	3 (6.8%)	16(14%)	0.211
PM Implantation for SSS	0(0%)	1(0.9%)	1.000
CRT Implantation	0(0%)	9(7.9%)	0.055

* Chi-square Test, Fisher's Exact Test *p<0.05 statistically significant. Categorical variables are reported n(%).
Abbreviations: AF; Atrial Fibrillation, AV; Atrioventricular, CRT; Cardiac Resynchronization Therapy, EPS; Electrophysiological Study, ICD; Implantable Cardioverter Defibrillator, PM; Pacemaker, PVC, Premature Ventricular Contraction, SSS; Sick Sinus Syndrome, SVT; Supraventricular Tachycardia VT; Ventricular Tachycardia

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-030

The effect of disease severity on cardiac autonomic functions in COVID-19

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Background and Aim: The new coronavirus (SARS-COV-2) pandemic has rapidly spread to many countries and caused morbidity and mortality all over the world. Central nervous system (CNS) can be affected in this disease because of the coronavirus capability to invade brainsteam. Olfactorial neurons blame to be entry for neuroinvasion and the spread the infection to the brainsteam via transneural route. Medulla oblangata contains cardiac, respiratory and vasomotor centers. A quarter of admitted COVID-19 complain from anosmia (loss sense of smell) and which may be indicator of CNS involvement. Heart rate variability (HRV) has long been associated to be a surrogate markers of cardiac autonomic tone. Inflammatory bowel disease (IBD) has effects on neural cardiovascular control mechanisms the effect of IBD disease status on cardiovascular autonomic functions by measuring (HRV) parameters with 24-hour holter electrocardiogram (ECG) recording. The aim of this study was to evaluate the heart rate variability (HRV) in a patient with coronavirus disease 2019 (COVID-19) as a marker disease severity and CNS involvement.
Methods: A prospective analytical case control study was conducted in Health Science University, Tepecik Education and Research Hospital İzmir, Turkey. We enrolled 50 patients with COVID-19 and 54 matched control subjects in the study. All participants underwent a 24-hour Holter recording to assess HRV parameters. Firstly, we design two groups as patient and controle group. Than the study population was separated into 3 groups of symptomatic disease, asymptomatic disease and control group to analyse the effect of disease severity on the HRV parameters.
Results: Age and gender distribution were well matched between groups (38.5±8.46 vs 39.9±15.3; p=0.95). Disease duration was 10.46±4.67 and %26.5 of patient complained from lost sense of smell and taste. %50 of the COVID-19 patient had symptomatic disease on admission. Control patient have higher BMI (26.2±3.34 vs. 28.1±4.0; p=0.02) than COVID patients. Plasma CRP (2.88±4.2 vs. 0.63±1.12 p=0.003) levels were found significantly high in patient with COVID-19. No difference was determined between the COVID-19 and control groups in respect of any HRV parameters (Table 2). Significant differences were found both between the symptomatic patients and the control group and between symptomatic and asymptomatic patients in terms of the HRV measurements of SDANN5, RMSSD, CCVLF, LF/HF (Table 3).
Conclusions: The results of this study demonstrated for the first time that the disease severity and symptom status in COVID-19 patients is associated with cardiac autonomic abnormalities compared to both a control groups and asymptomatic COVID-19 patients. Patients who have had symptomatic COVID-19 infection appear to be at risk for arrhythmia, particularly patient with sign of CNS involvement. Further research is needed to clarify long term effect of COVID-19 infection on HRV.

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

Variables	COVID-19 (n:50)	Control (n:52)	P
Age, yrs	38.5 ± 8.46	39.9 ± 15.3	0.45
Male/Female (n)	23/27	24/28	0.98
Smoking (%)	26	13.5	0.19
Hypertension (%)	12.2	17.3	0.25
Hyperlidemia (%)	10	5.8	0.89
Diabetes Mellitus(%)	6	9.6	0.12
BMI, kg/m2	26.2±3.34	28.1 ± 4.0	0.02
BSA (m^2)	1.85 ± 0.22	1.9±0.18	0.4
ESR (mm/h)	14 ± 6.6	12.4 ± 4.6	0.46
C-reactive protein (mg/L)	2.88 ± 4.2	0.6 ± 1.17	0.003
Hgb, g/dL	14.04 ± 1.6	13.0 ± 1.6	0.37
PLT, mL/mm3	299.4 ± 93.5	258.04 ± 58.05	0.59
NL	2.16 ± 1.24	2.07 ± 1.03	0.75
Creatinine, mg/dL	0.86 ± 0.2	0.83 ± 0.15	0.52
Number of COVID medications, %			
1 medication (HCQ)	60		
2 medications	16		
?3 medications	24		
Severity of Disease (%)			
Uncomplicated	64		
Mild Pneumonia	28		
Moderate-Severe Pneumonia	8		
Asymptomatic / Symptomatic disease, %	50 / 50		
Disease duration, days	10.46 ± 4.67		
Lost of sense (taste/smell) (%)	26.5		

BMI: body mass index, BSA: Body surface area, ESR: erythrocyte sedimentation rate, HCQ: Hydroxychloroquine, Hgb: hemoglobin, PLT: platelets, WBC; white blood cells.

Table 2. Differences in heart rate variability measures between the study population

Variables	Control (n:52)	COVID-19 (n:50)	P
Time Domain Measures			
SDNN (ms)	136.9 ± 36.6	132.3 ± 34	0.51
SDANN5 (ms)	123.9 ± 36.0	115.3 ± 36.6	0.23
RMSSD (ms)	12.7 (23.2 - 36)	18 (22 – 40)	0.37
SDNN Index(ms)	19.5 (65.5-85)	25(63-88)	0.58
pNN50 (%)	8.08 (2.38 - 10.4)	9.54 (2.87- 12.4)	0.75
Frequency Domain Measures			
CCVLF	0.02 (0.03- 0.05)	0.02 (0.03-0.05)	0.43
CCVHF	0.02 (0.03- 0.05)	0.02 (0.03 – 0.045)	0.74
LF/HF	1.24 ±0.33	1.27 ±0.3	0.54
Basic parameters			
Mean HR	78.2 ± 8.2	81.06 ± 7.8	0.08
Max HR	140 ± 17.4	147.8 ± 18.1	0.03
Min HR	52.6 ± 6.9	52.2 ± 6.7	0.78
QTc	434.8±20.9	439.7±19.1	0.24

The values are presented as median (interquartile range) CCVLF (Coefficient of Component Variance for LF) , CCVHF (Coefficient of Component Variance for HF) , SDNN: Standard deviation of all NN intervals, SDANN5: Standard deviation of the mean values of the NN intervals in the 5-minute intervals within the defined period, RMSSD: root mean of squared successive differences, pNN50: Percentage of the NN intervals within the defined period that differ from the previous NN interval by more than 50 mS.HR: Heart rate,

Table 3. Comparison of heart rate variability measures according to severity of disease

Variables	COVID-19 symptomatic patient (n:25)	COVID-19 asymptomatic patient (n:25)	Control (n: 51)	p
Time Domain Measures				
SDNN (ms)	120.0 ± 25.6 [a]	143.2 ± 37.3	136.9 ± 36.6	0.057
SDANN5 (ms)	99.4 ± 30.4 [a,b]	129.3 ± 36.3	123.9 ± 36.0	0.007
RMSSD (ms)	11 (21 - 32) [a,b]	27(24 -51)	12.7 (23.2 - 36)	0.02
SDNN Index (ms)	32 (51 - 83) [a]	22.5 (74.5 -97)	19.5 (65.5-85)	0.02
pNN50 (%)	6.82 (2.43 – 9.25) [a]	12.1 (2.93 - 15.0)	8.08 (2.38 - 10.4)	0.5
Frequency Domain Measures				
CCVLF (ms^2)	0.02 (0.02-0.04) [a,b]	0.02 (0.04 - 0.06)	0.02 (0.03- 0.05)	0.001
CCVHF (ms^2)	0.01 (0.03 -0.04) [a]	0.02 (0.03 – 0.05)	0.02 (0.03- 0.05)	0.09
LF /HF	0.88 ±0.24 [a,b]	1.22 ±0.25	1.24 ±0.33	0.001
Mean HR	79.8 ± 5.6	82.1 ± 9.4	78.2 ± 8.2	0.13
Max HR	147.1 ± 18.0	148.5 ± 18.5	140 ± 17.4	0.08
Min HR	51.4 ± 4.1	52.9 ± 8.3	52.6 ± 6.9	0.73
QTc	439.2 ±19.5	440.2 ±19.06	434.8±20.9	0,5

The values are presented as median (interquartile range), HR: Heart rate,
a was significant between symptomatic disease and asymptomatic groups
b was significant between symptomatic disease and control groups
significance p<0.05

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-031

Blood urea nitrogen and gamma glutamyl transferase levels are independently associated with prolongation of his-ventricular interval

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Background and Aim: His-ventricle (HV) interval is one of the basic measurements of the electrophysiological study, reflecting conduction time through the His-Purkinje system. Fibrosis and fatty infiltration play a role in the pathogenesis of HV interval prolongation. Recent studies claimed that fibrosis in the conduction system could be triggered by oxidative stress. Blood urea nitrogen (BUN) and gamma-glutamyl transferase (GGT) are non-specific markers of oxidative stress and can be practically evaluated in the clinic. In this study, we investigated whether HV interval prolongation is associated with BUN and GGT levels.
Methods: The study population included 94 consecutive patients (58 women) who underwent clinically indicated electrophysiology study (EPS) with the diagnosis of supraventricular tachycardia in the drug-free state. Measurements of BUN and GGT levels were made with routine laboratory tests before the procedure. The EPS protocol was similar in all patients. Quadripolar electrode catheters were inserted via the femoral vein and positioned in the coronary sinus, the upper right atrium, the right ventricular apex, and His-bundle. Atrium-His, HV intervals, and basal cycle length were measured.
Results: The mean age of the patients was 47.9±15.8 years. In univariate logistic regression analyses age (p=0.002), male gender (p=0.033), hypertension (p=0.010), hyperlipidemia (p=0.029), coronary artery disease (p=0.017), diabetes (p=0.029), body-mass index (p=0.002), BUN (p<0.001), eGFR (p=0.001), and GGT (p=0.001) were associated with HV interval prolongation. In multivariate logistic regression analysis BUN (OR: 0.358,

95% CI: 0.085-0.489, p=0.006) and GGT (OR: 0.347, 95% CI: 0.051-0.322, p=0.008) were independently associated with prolongation of HV interval.
Conclusions: Blood urea nitrogen and GGT may have an independent association with HV interval prolongation. This relationship, which should be confirmed by extensive studies, can be a practical aid in the follow-up of patients with infra-His conduction defects.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-032

Electrocardiographic changes in intensive care unit patients during their last hours of life: Can we use these parameters as prognostic indicators?

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Background and Aim: QRS amplitude attenuation and prolonged QRS duration has been associated with increased mortality in various clinical conditions including critical care patients and general population. Relative bradycardia has been found to be associated with lower mortality in patients with septic shock, but there are no studies in literature evaluating the electrocardiographic (ECG) changes and changes in heart rate (HR) just before death. Our aim of this study is to calculate the gradual changes in these parameters in the last hours of life from II derivation telemetry records.
Methods: We included 30 patients who died in intensive care unit irrespective of their diagnosis during admission and follow up. HR, QRS amplitude and QRS duration were analysed from the telemetry recordings obtained from the last 10 hours of their life.
Results: QRS duration prolongs and heart rate decreases during the last 10 hours of life and the changes in these parameters were more prominent in the last hours. QRS duration increased at rate of 5.43 ms per hour (p<0.001) and heart rate decreased at rate of 2.68/min each hour (p<0.001). QRS amplitude attenuation were more subtle (decreased by 0.23 mV per hour, p=0.02) compared to QRS duration and heart rate.
Conclusions: During last 10 hours of life, there was widening of QRS complex, attenuation of QRS voltage and decrease in heart rate. Automated softwares could present these findings in graphics and can be used as a prognostic indicators to recognize a dying patient. This information could be used in certain acute reversible critical conditions such as fulminant myocarditis, anaphylactic shock, trauma patients as a sign of poor prognosis or on decision making regarding end-of-life in irreversible illness such as terminal cancer patients.

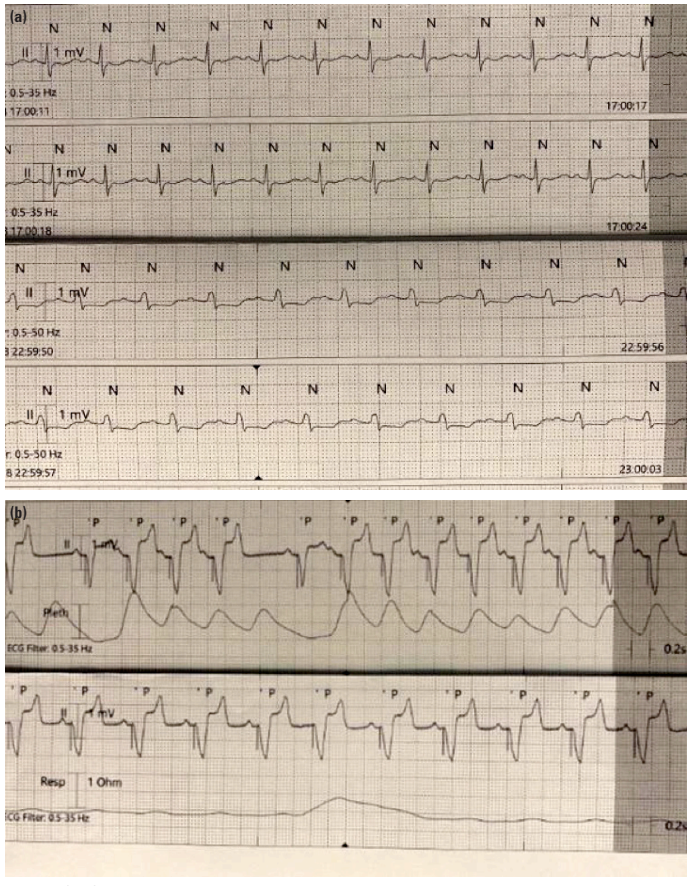

Figure 1. (a, b) ECG samples of two different patients during their last hours of life. In Picture 2 pacemaker ECG was presented to demonstrate changes in QRS voltage and QRS duration despite the same pacing site and same pacing voltage.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-033

Macular cell thickness is associated with ventricular repolarization abnormality in patients with non-diabetic hypertension

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Background and Aim: Endothelial dysfunction, inflammatory, and atherosclerotic effects of arterial hypertension on the arterial system are the main trigger factors of the side effects on all other systems. Retinal fundus oculi are one of the most commonly affected organ. T-peak to T-end interval (tpe) and tpe / QT ratio have been shown to be predictive of ventricular arrhythmias. In this study, we investigated the relationship between eye examination findings and tpe / QT ratios.
Methods: Patients with newly diagnosed with hypertension who were admitted to our clinic between January 2019 and May 2019 were included in the study. The population consisted of 70 individuals. Patients were referred to the ophthalmology clinic for a retinal fundus examination. The tpe / QT ratio of these patients was calculated on the electrocardiogram and compared to the eye examination measurements.
Results: The mean macular thickness was negatively and significantly correlated with the tpe / QT ratio (p=0.05). A significant negative correlation remained in the regression analysis (p=0.011). There was no significant difference in terms of basic features.
Conclusions: The mean macular thickness was associated with the tpe / QT ratio in patients with newly diagnosed hypertension. Hypertension is a systemic disease that affects the entire arterial system of the body. Eye fundus examination provides the detection of the development of retinopathy. It may also give a clue about the development of arrhythmia in hypertensive patients. Patient groups with low macular thickness can be identified as high-risk patients for arrhythmogenic events and can be kept under closer observation.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-034

The clinical importance of the combined use of P wave dispersion and troponin I for predicting atrial fibrillation recurrence in patients with paroxysmal atrial fibrillation

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Background and Aim: P-wave dispersion (PWD) and cardiac troponin were seperately shown to be associated with atrial fibrillation paroxysmal atrial fibrillation (PAF) recurrence. To our knowledge, there is no study to eveluating the clinical importance of the combined use of PWD and cardiac troponin I levels in patients with PAF. The aim of our study was to investigate the clinical importance of the combined use of PWD and cardiac troponin I level for predicting AF recurrence in patients presenting to the emergency department with PAF.
Methods: 65 patients with paroxysmal atrial fibrillation (PAF) were included in the study. Patients were categorized three groups according to the baseline troponin and PWD cut-off values. Group 1 was defined as troponin <0.11ng/dl and PWD <44.5 ms; group II was defined as troponin <0.11ng/dl and PWD ≥44.5 ms, or troponin ≥0.11ng/dl and PWD and PWD <44.5 ms; group III was defined as both troponin ≥0.11ng/dl and PWD ≥44.5 ms.
Results: Patients were divided into two groups as recurrent and non-recurrent AF. Twenty-one (32.3%) patients had AF recurrence within 6 months. Table 1 presents the distribution of some descriptive and clinical features among AF recurrent and non-recurrent groups. There was no statistically significant difference between two groups in terms of clinical features (Table 1). Laboratory parameters of two groups are presented in Table 2. There was no statistically significant difference between two groups in terms of laboratory parameters. The PWD and baseline troponin values of patients are shown in Table 3. The PWD (p<0.001) and baseline troponin (p=0.004) values were significantly higher in patients with AF recurrence than in non-recurrent patients. In addition, the frequency of troponin positivity in patients with AF recurrence was significantly higher than those without AF recurrence. ROC curve analysis was performed to determine the cut-off values of troponin and PWD for predicting AF recurrence. Troponin ≥0.11 ng / mL predicted AF recurrence with a specificity of 61.9% and sensitiviy of 72.7% (Figure 1). Also, PWD ≥44.5 ms predicted AF recurrence with a specificity of 72.7% and sensitiviy of 71.4% (Figure 2). Twenty two (33.9%) patients had both troponin ≥0.11 ng / mL and PWD ≥44.5 ms (Group 3), 13 (20%) had troponin ≥0.11 ng/mL or PWD ≥44.5 ms (group II) and 30 (46.2%) patients had both troponin <0.11 ng/mL and PWD <44.5 ms (group I). Group III patients had significantly higher AF recurrence than group I (59.1% vs 16.7%, p=0.001) and group II patients (59.1% vs 23.1%, p=0.039) (Figure 3). Multivariate logistic regression analysis showed that group II (odds ratio [OR]: 1.025, 95% confidence Interval (CI): 0.189-5.553, p=0.977) and group III (OR: 7.236, 95% CI: 1.879-27.861, p=0.004) were independent predictors of AF recurrence (Table 4).
Conclusions: The combined use of PWD and basal troponin results in higher clinical importance compared with the use of PWD or troponin alone in predicting AF recurrence.

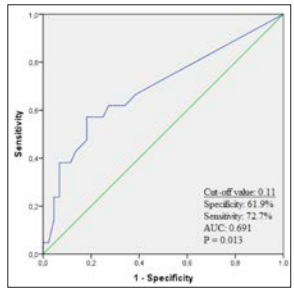

Figure 1. ROC of troponin level and AF recurrence.

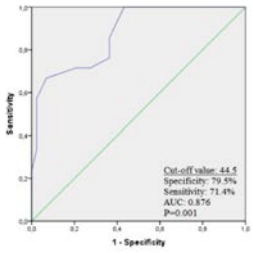

Figure 2. ROC of PWD and AF recurrence.

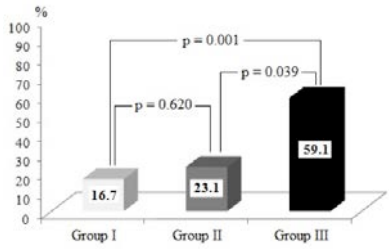

Figure 3. Comparison of AF recurrence rates the three groups.

Table 1. Distribution of some descriptive and clinical features among AF recurrent and non-recurrent groups

	Recurrent AF group (n=21)	Non-recurrent AF group (n=44)	P Values
Age (years),	47.1 ± 5.0	46.1 ± 5.8	0.568
Male gender, (%)	12 (57.1)	26 (59.1)	0.882
BMI, (kg/m^2),	28.1 ± 5.0	26.4 ± 4.4	0.163
SBP (mmHg),	127.1 ± 10.1	122.3 ± 12.9	0.182
DBP (mmHg),	77.1 ± 9.0	76.8 ± 7.2	0.690
Heart rate, bpm	64.5 ± 7.6	64.0 ± 8.3	0.657
Ejection fraction, (%)	56.2 ± 3.4	57.9 ± 3.8	0.511
Left atrium, (mm)	33.4 ± 2.5	32.8 ± 3.3	0.849

BMI: Body mass index; SBP; systolic blood pressure, DBP; diatolic blood pressure,

Table 2. Baselinelaboratorycharacteristicsbetween AF recurrentandnon-recurrentgroups

	Recurrent AF group (n=21)	Non-recurrent AF group (n=44)	P Values
Glucose (mg/dL)	91.9 ± 5.5	89.3 ± 6.5	0.113
Total Cholesterol (mg/dL)	168.8 ± 70.9)	181.1 ± 49.5	0.421
LDL (mg/dL)	113.1 ± 41.5	125.7 ± 41.2	0.254
HDL (mg/dL)	47.1 ± 18.8	44.8 ± 15.3	0.752
Üre (mg/dL)	39.7 ± 19.0	34.3 ± 13.3	0.523
Kreatinin (mg/dL)	1.02 ± 0.32	0.88 ± 0.22	0.163
ESR (nm/h)	18.0 ± 12.9	21.2 ± 17.8	0.822
CRP (mg/L)	5.4 ± 5.6	4.9 ± 7.6	0.066
Na (mmol/L)	136.3 ± 2.9	137.6 ± 3.1	0.148
K (mmol/L)	4.0 ± 0.7	4.1 ± 0.5	0.436
Hemoglobin (g/dL)	13.8 ± 1.7	14.0 ± 1.8	0.630
WBC (10^3/mm^3)	11.5 ± 3.2	11.4 ± 3.4	0.828
Platelet (10^3/mm^3)	246.0 ± 63.2	243.6 ± 67.	0.817

LDL; low dencity lypoprotein; HDL; high-density lipoprotein, ESR; Erythrocyte sedimentation rate, CRP; C reactive protein, WBC; White blood count,

Table 3. Distribution of PWD and troponin values between AF recurrent and non-recurrent groups

	Recurrent AF group (n=21)	Non-recurrent AF group (n=44)	P Values
PWD (ms)	51.9±14.2 (40-90)	35.8±9.0 (16-50)	**<0.001**[a]
Troponin level ()	0.16 ± 0.16 (0-0.6)	0.07±0.11 (0-0.5)	**0.009**[a]
Troponin categorization			**0.034**[b]
Positive (%)	14 (66.7)	17 (38.6)	
Negative (%)	7 (33.3)	27 (61.4)	

[a]Mann-Whitney U Test; [b]Ki-Kare Test
PWD: P-wave dispersion, AF

Table 4. Multivariate logistic regression analysis representing the independent predictors of recurrence of PAF

Variables	OR (95% CI)	P Values
Glucose	1.106(0.990-1.235)	0.076
Group I[a]	-	-
Group II	1.025 (0.189-5.553)	0.977
Group III	7.236 (1.879-27.861)	**0.004**

Entered variables: Age; C-reactive protein; white blood count; Erythrocyte sedimentation rate; Glucose; platelet; Gender; Groups; OR - odds ratio; CI - confidence interval.

a Reference group.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-035

Right ventricular apical and septal pacing: Long term impact on ventricular functions

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Background and Aim: In patients requiring permanent pacemaker, in order to protect left and right ventricular functions the optimal pacing site has yet to be determined. Conflicting results exist about septal and apical pacing sites. Our purpose was to evaluate the long term effects of right ventricular apical and septal pacing on left and right ventricular functions.
Methods: We scanned 378 patients from 2007 to 2012, who received a permanent pacemaker for the treatment of symptomatic bradyarrythmia. As exclusion criteria we identified the patients who did not have an echocardiography before the procedure, patients who died and those who rejected our invitation. 54 women and 66 men were eligible for our study. To determine the patients' New York Heart Association Class (NYHA) we questioned and did the physical examination. Lead position confirmed by fluoroscopy in two planes, and electrocardiograms were obtained. Finally, we compared the pre-procedural echocardiographic data with our up-to-date findings.
Results: In sixteen patients lead placement was at septal site and in one hundred and four patients apical site. Median follow up duration was 9 years. The mean ejection fraction before the implantation was 58.86±4.08 in the apical, and 56.37±8.8 in the septal group (p<0.05). The long term follow up showed that these values have been reduced, 56.66±8.38 for the apical group and 51.33±13.94 for the septal group, respectively (p<0.05). Placing the right ventricular lead in both septal and apical site resulted in reduced tricuspid annular plane systolic excursion (from mean 2.25 to 2.18, (p<0.05)), and in increased systolic pulmonary artery pressure (from 35.46±9.93 to 39.84±11.21 (p<0.05)). There were no differences regarding the mitral and tricuspid insufficiencies, and diastolic functions before the implantation and long term follow up. These findings were independent of neither the etiology of implanting the pacemaker nor the underlying diseases.
Conclusions: These two selective ventricular pacing sites caused a reduction in both left and right ventricular functions. Despite the ejection fraction declines, most of these patients have a good quality of life, without symptoms and signs of heart failure. But certainly, there is emerging need for more randomized trials in order to describe the optimal RV pacing site. The main purpose must be preserving better ventricular functions in patients requiring permanent ventricular pacing.

Table 1. Echocardiographic findings

Septal and apical group (n=120)	Pre-implantation	Long term results
TAPSE	1,1-3,2(mean.2,25)	1,0-3,6(mean.2,18) (p<0,05)
sPAP	35,46±9,93	39,84±11,21 (p<0,05)

TAPSE=tricuspid annular plane systolic excursion, sPAP=systolic pulmonary artery pressure

Echocardiographic findings

Groups	Septal (n=16)	Apical (n=104)
LVEF(pre-implantation)	58,86±4,08	56,37±8,8 (p<0,05)
LVEF(long term results)	56,66±8,38	51,33±13,94 (p<0,05)

LVEF: Left ventricular ejection fraction.

Epidemiology

OP-037

The opinions of cardiologists on carrying out scientific study and scientific writing and the problems they encounter

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Background and Aim: Conducting a scientific study and writing a scientific manuscript is a demanding and difficult process. The goal of this study was to investigate the opinions of cardiologists in Turkey on scientific study and scientific writing and the difficulties they encounter.
Methods: In this cross-sectional study, a 24-item questionnaire was distributed to cardiology residents, specialists, and academics in Turkey by e-mail, or WhatsApp message via https://docs.google.com/forms. We performed a pre-test among 10 cardiologists to determine the errors and weak sides of the questionnaire. The questionnaire included both closed- and open-ended questions.
Results: Of 188 cardiologists who responded the questionnaire 160 (85.1%) were male. The mean age was 37.53±5.60 years. Fourteen (7.4%) participants were resident, 120 (63.8%) were specialist and 54 (28.8%) had an academic degree. Of all participants 174 (92.6%) currently conduct or desire to conduct a scientific study. The leading aim of scientific study was promoting academic degree (n=133, 70.7%). Thirty (16.0%) participants responded that they could not conduct scientific study due to heavy working conditions, 20 (10.6%) participants responded that lack of motivation was the reason not to conduct research. Finding an original hypothesis and designing the study (n=86, 45.7%), writing the manuscript (n=34, 18.1%) finding an appropriate journal to submit (n=23, 12.2%), collecting the data (n=18, 9.6%), obtaining an ethics committee approval (n=15, 8.0%), performing the statistical analysis (n=12, 6.4%) were the most difficult stages of a scientific research responded by participants (Figure 1). The discussion part of a manuscript was accepted as the most diffucult part to write by 114 (60.4%) participants. The support of cardiology societies to scientific research were found insufficient by 133 (70.5%) participants. A course about 'writing a scientific manuscript' was the mostly demanded educational issue (n=94, 50.0%) (Figure 2).

Conclusions: Most of the cardiologists conduct or desire to conduct a scientific study. The leading aim is promoting academically. Finding an original hypothesis and designing the study is the commonest difficulty. A course about 'writing a scientific manuscript' is the mostly required educational issue.

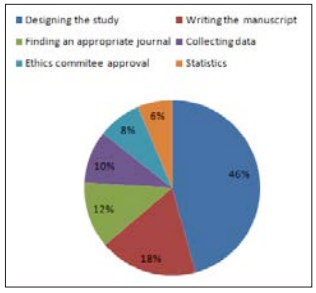

Figure 1. The most difficult stage of conducting a scientific study according to participants.

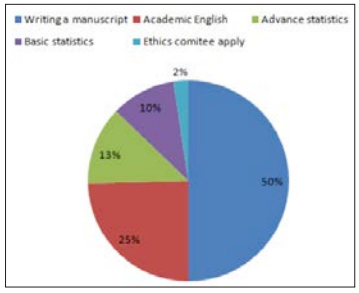

Figure 2. Educational courses demanded by the participants.

Epidemiology

OP-038

Evaluation of the demographic characteristics of patients who had an ischemic stroke while using non-vitamin K antagonist oral anticoagulant

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Background and Aim: Approximately 87% of stroke patients are of ischemic and 13% are of haemorrhagic origin. Atrial fibrillation(AF) is the most common type of arrhythmia in clinical practice. The prognosis for AF-related strokes is known to be worse than that of other aetiologies. For this reason, there is a need to start prophylactic treatment before a major stroke or a severe disability develops in patients with AF. Previous studies have shown that the incidence of ischemic stroke in patients who receive non-vitamin K antagonist oral anticoagulants(NOAC) is approximately 1-2%. We aimed to evaluate the demographic characteristics of patients who had an ischemic stroke while using non-vitamin K antagonist oral anticoagulants.
Methods: Patients who had an ischemic stroke while using NOACs between January 2015 and January 2020 were included in the study. Patients and their relatives were called by phone and their file records were used to obtain their age, gender, comorbidities, NOAC use, routine biochemical and haematological parameters and transthoracic echocardiography information.The CHA2DS2-VASc score was used to calculate the risk of ischemic stroke in patients with non-valvular AF. This scheme is scored by two points each for a history of stroke/transient ischemic attack (TIA) and age >75, and 1 point each for age 65–74, history of hypertension, diabetes, heart failure, vascular disease (myocardial infarction, complex aortic plaque and past revascularization, and peripheral arterial disease (PAD), including angiographic PAD findings, PAD-related amputation) and female gender.
Results: The study included 73 patients diagnosed with non-valvular atrial fibrillation (AF) who had a stroke while using NOACs. Of these patients, 23 (31.5%) were male and 50 (68.5%) were female. The mean age of the patients was 74.6±10 years. The mean CHA2DS2-VASc score was 5.6±1.5. Of the patients, 34 (46.6%) had recurrent strokes. All patients had vascular disease. Sixty-seven (91.8%) patients had hypertension. Twenty-one patients who were receiving NOACs were found to be receiving the NOACs at doses not recommended by the current (Table 1). The mean age was higher and the CHA2DS2-VASc score was lower in patients who did not receive the appropriate dose compared to those who did receive the appropriate dose. The history of ischemic stroke was lower in patients who did not receive the appropriate dose compared to those who did receive the appropriate dose (Table 2).
Conclusions: The high CHA2DS2-VASc score of patients who had a stroke despite treatment with NOACs, the presence of vascular disease in all of them, the presence of hypertension in the majority and a history of ischemic stroke in almost half of these patients all suggest that NOAC treatment may be insufficient in patients with the high risk factors of vascular disease, hypertension and a history of ischemic stroke. In addition, the reasons why about one third of patients use NOACs at an insufficient dose needs to be investigated.

Table 1. Demographic characteristics of patients who had an ischemic stroke while using -vitamin K antagonist oral anticoagulants

Parameter	n, (%)
Ages (years) *	74.6±10
Gender (female), n (%)	50, (68.5)
Hypertansion, n (%)	67, (91.8)
Coronary Artery Disease, n (%)	34, (46.6)
Diabetes Mellitus, n (%)	24, (32.9)
Heart Failure, n (%)	22, (30.1)
Carotid Artery Disease, n (%)	68, (93.2)
Stroke, n (%)	34, (46.6)
Vascular Disease, n (%)	73, (100)
Chronic Kidney Disease, n (%)	8, (11.0)
CHA2DS2VASc score *	5.6±1.5
Appropriate dose, n (%)	52, (71.2)

**Avarage±SD, CHA2DS2VASc: Congestive Heart failure, hypertension, Age ≥75 (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65–74, and Sex (female).*

Table 2. Comparison of demographic characteristics between patients with appropriate dose and inappropriate dose

	Appropriate dose	Inappropriate dose	p
Age, years *	73.2±11.1	78.1±5.3	NS
Gender, female, n	35	15	NS
Hypertension, n	49	18	NS
Diabetes Mellitus, n	18	6	NS
Heart Failure, n	15	7	NS
CHA2DS2VASc *	5.8±1.6	5.6±1.7	NS
Stroke, n	29	5	0.013
Vascular Disease, n	52	21	NS

**Avarage±SD, CHA2DS2VASc: Congestive Heart failure, hypertension, Age ≥75 (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65–74, and Sex (female), NS: Not significant (p>0.05).*

Epidemiology

OP-039

Effects of admission body mass index, waist circumference and waist to height ratio on very long term mortality in patients with STEMI

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Background and Aim: Previous studies demonstrated that there is a U shaped association between body mass index (BMI) and mortality in patients with coronary artery disease(CAD). However it is suggested that anthropometric indices reflecting body fat distribution can predict the obesity related risk better than BMI. Waist circumference and waist to height ratio are being used to detect visceral adiposity and obesity. In this study we aimed to investigate the effect of these anthropometric indices on very long term mortality in patients who survived after the first ST elevational myocardial infarction (STEMI).
Methods: Patients who were diagnosed with STEMI between 2004-2006 years in three different tertiary centers were included into this retrospective study. Their all-cause mortality information was extracted by hospital database search, phone visits and electronic death notification system search. The predictors of mortality were tested with multivariate Cox regression analysis including variables like BMI, waist circumference and waist to height ratio.
Results: Two hundred and thirty nine STEMI patients were included into the retrospective analysis. Median follow up duration was 177 months. Seventy nine deaths occurred during the follow up. Metabolic syndrome frequency and female gender frequency was higher in patients who died compared to patients who were alive. Patients who died were significantly older at the admission, and their hemoglobin levels were significantly lower than alive patients. While mean height was lower, mean weight and waist to height ratio was higher in patient who died compared to patients who were alive. The reperfusion strategy, infarct size markers and ejection fraction were similar between the groups; but presence of a depressed left ventricular systolic function (EF<40%) was significantly higher in patients who were dead compared to patient who were alive. Cox regression analysis revealed that older age, increased waist to height ratio and presence of depressed left ventricular systolic functions were the independent predictor of very long term mortality.
Conclusions: Waist to height ratio can be used as an independent predictor of very long term mortality in first STEMI survivors.

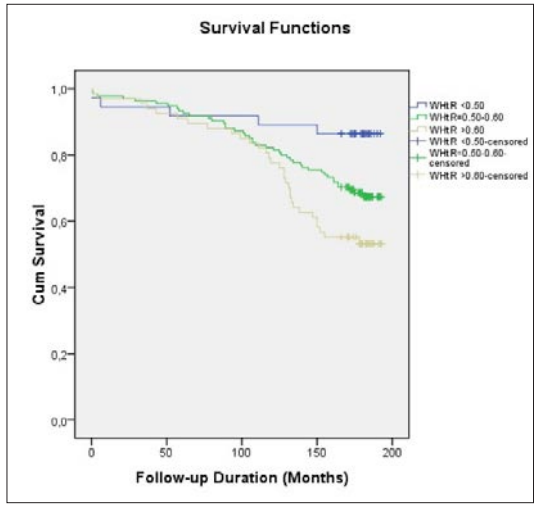

Figure 1. Kaplan Meier survival curves according to waist to height ratio groups

Table 1. Cox regression analysis for the predictors of very long term mortality

	Exp(B)	95,0% CI for Exp(B) Lower	Upper	p
Gender (Female)	1,013	,464	2,211	,974
Age	1,052	1,027	1,077	,000
Hypertension	,839	,487	1,446	,527
Metabolic Syndrome	1,541	,879	2,699	,131
Hemoglobin	,941	,801	1,106	,463
Ejection Fraction <40%	1,812	1,051	3,123	,032
Body Mass Index	,953	,865	1,051	,338
Waist to Height Ratio	295,829	7,360	11890,244	,003
Waist Circumference	,985	,943	1,028	,486

Table 2. Prognostic and quality of care parameters that may related with the survival between the study groups. Group 1: Alive patients, Group 2: Dead Patients

	Group 1 (160)	Group 2 (79)	p
Reperfusion Strategy			
Primary PCI	52(32.7)	21(26.6)	0.463
Thrombolysis	71 (44.7)	35(44.3)	
Others	36(22.6)	23(29.1)	
Door to needle time	35(30-50)	40(30-60)	0.642
Door to angiography time	35(30-60)	30(20-45)	0.265
Peak CKMB	201(113-327)	220(135-379)	0.165
Ejection Fraction	50±8	47±11	0.109
Wall Motion Score Index	1.56±0.31	1.66±0.41	0.052
Ejection Fraction <40%	21(13.1)	20(25.3)	0.019

Table 3. Demographic features and laboratory parameters of the study groups, Group 1: Alive patients, Group 2: Dead Patients

	Group 1 (160)	Group 2 (79)	P
Age, years	54±10	61±10	<0.001
Gender (Female), n (%)	16(10.0)	16(20.3)	0.029
Hypertension. n (%)	41(25.6)	28(35.4)	0.115
Diabetes, n (%)	22(13.8)	15(19.0)	0.292
Smoking, n (%)	110(68.8)	56(70.9)	0.736
Metabolic Syndrome, n (%)	59 (36.9)	40(50.6)	0.042
Height, cm	170±8	166±8	<0.001
Weight, kg	78±12	75±12	0.031
Waist Circumference, cm	94±9	96±10	0.209
Waist to Height Ratio	0.55±0.07	0.59±0.07	0.001
Body Mass Index, kg/m^2	27.01±3.81	27.10±3.65	0.867
MI Localization, n(%)			
Anterior	90(56.2)	40(50.6)	0.412
Non-Anterior	70(43.8)	39(49.4)	
Hemoglobin	14.8±1.4	14.2±1.9	0.009
Creatinine	1.04±0.21	1.07±0.29	0.318
Total cholesterol	195±44	188±41	0.235
LDL	126±39	120±37	0.288
HDL	41±10	41±10	0.960
Triglyceride	115(80-185)	115(75-160)	0.203

Epidemiology

OP-040

Evaluation of supraventricular arrhythmias among women undergoing controlled ovarian stimulation before in vitro fertilization

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Background and Aim: Controlled ovarian stimulation (COS) is the second stage of in vitro fertilization (IVF) to harvest as many matured eggs as possible to maximize the chance of fertilization. Estrogen affects every system including both vascular and electrical system of heart. The effects of estrogen on electrical system was studied in animal models and also in human beings and this studies demonstrated increased level of estrogen was associated with prolonged QT interval. There is a strong correlation between the supraventricular arrhythmia episodes and plasma concentrations of estrogen. However, the main mechanism of the effects are not known so far. The aim of our study to determine the frequency of supraventricular arrhythmia episodes in patients underwent ovarian stimulation with high doses of estradiol.
Methods: This study included 102 patients administered to infertility clinic and underwent ovarian stimulation treatment with estradiol according to assisted reproductive technology protocols. Patients were questioned if they had a supraventricular arrhythmia episode before treatment and also followed up along the treatment in terms of any arrhythmia episode. Whole patients had basal electrocardiography (ECG) record and ECG results were evaluated by cardiologists. All the palpitation episodes were recorded and patients were invited to ECG.
Results: The mean age of 102 patients was 30.6±8.2 and 5.8% (n=6) patients had palpitations during the treatment. The ECG recordings of 1.9% (n=2) patients during palpitation symptom can not be captured and 2 patients were suffered from sinus tachycardia (110 bpm and 122 bpm). 1.9% (n=2) patients had supraventricular tachycardia (SVT and AVNRT) episode, SVT last spontaneously and AVNRT ended up with IV adenosine treatment in emergency service also repeated once more in the same week. This patient declared that she had never palpitation attack before estradiol treatment.
Conclusions: Slight prolongation of QT interval with supraphysiological estradiol levels were reported in small study populations but there was not any study point out any supraventricular arrhythmia increase in ovarian stimulation treatment. Epidemiological studies notified the incidence of paroxysmal supraventricular tachycardia 30-60 per 100,000 in women. Also a few study demonstrated increase in the incidences arrhythmias in pregnant women especially during the last trimester which accounted to increase of plasma hormone concentrations. We demonstrated an increase of supraventricular arrhythmia frequencies under high dose estradiol treatment in small study population may retrieve more clinical sense in case of enhanced population. It is better to be aware of arrhythmias before administering ovarian stimulation, however supraventricular tachycardias not cause a real life threatening risk, ruin out patients comfort.

Epidemiology

OP-041

Clinical outcomes and in-hospital mortality of patients with definite infective endocarditis: a single center experience

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Background and Aim: Infective endocarditis (IE) is among the most fatal infectious diseases with rapidly evolving clinical characteristics since the introduction of various intracardiac devices and interventions. Herein, we aimed to summarize clinical and laboratory characteristics, and outcomes of patients diagnosed with definite IE and to identify the predictors associated with poor outcome.
Methods: In this retrospective analysis of patients with TEE proven vegetation, those fulfilling the modified Duke criteria for definite IE were identified. The clinical and laboratory characteristics and outcomes in relation to mortality were recorded
Results: Of 50 patients with a vegetation and definite IE, (mean age 57±15.9 years, 34% female) 72% was native-valve endocarditis of which only 32% had a predisposing valve disease such as rheumatic valve. The rate of prosthetic-valve IE was 10% and that of device-related IE was 18% (Table 1). The most common causative organisms were S.aureus (16%), Coagulase-negative Staphylococci(16%), Enterococci(14%) and Viridans streptococci (12%). In-hospital mortality was 24% and was associated with concomitant diabetes, coronary artery disease, higher baseline creatinine, anemia, occurrence of cranial complications and absence of surgery during the index of hospitalization (Table 2). Independent predictors of mortality were concomitant CAD and absence of surgery (Table 3).
Conclusions: Our small cohort of definite IE patients showed that contemporary characteristics of IE has evolved with higher rates of device-related IE and smaller number of viridans streptococci as the causative organism. In addition to established prognostic factors, timing of surgery has become a major concern suggesting need of early surgery in selective patients.

Table 1. Demographic and clinical characteristics of the overall study population and study groups with regard to mortality

	Overall study population	Without mortality(n:38)	With mortality (n:12)	p value
Age, years	57±15,9	57±17	60±12,3	0,265
Female gender, n(%)	17(34)	14(36,8)	3(25%)	0,510
Type of IE				
Native valve	36(72)	28(73,7)	8(66,7)	0,536
Mitral	15	14(50)	3(37)	
Aortic	11	9(32)	4(50)	
Mitral+aortic	2	2(7)	0(0)	
Tricuspid	3	3(10)	1(12,5)	
Prosthetic valve	5(10)	4(10,5)	1(8,3)	1
Mitral	2(4)	3(7,9)	1(8,3)	0,920
Aortic	3(6)	2(5,3)	1(8,3)	
Device-related	9(18)	6(15,8)	3(25)	0,660
Pace	1(2)	1(2,6)		0,301
icd	1(2)	0(0)	1(8,3)	
crt	7(14)	5(13,2)	2(16,7)	
Risk Factors				
Diabetes mellitus	26(52)	16(42,1)	10(83,3)	0,031
Hypertension	39(78)	30(78,9)	9(75)	1
Previous stroke	5(10)	5(13,2)	0	0,319
CAD	16(32)	9(23,7)	5(41,7)	0,036
CKD	17(34)	11(28,9)	6(50)	0,294
Dialysis	4(8)	2(5,3)	2(16,7)	0,240
Catheter	5(10)	3(7,9)	2(16,7)	0,582
Autoimmunity	3(6)	3(7,9)	0(0)	1
Cancer	5(10)	5(13,2)	0(0)	0,319
Immune supression	7(14)	6(15,8)	1(8,3)	1
Heart failure	14(28)	9(23,7)	5(41,7)	0,285
Thyroid disease	7(14)	7(18,4)	0(0)	0,174
COLD	4(8)	2(5,3)	2(16,7)	0,240
Predisposing native valve condition	16(32)	12(31,6)	4(33,3)	1
Rheumatic valve	8(16)	5(13,2)	3(25)	0,600
Mitral valve disease	3(6)	2(5,3)	1(8,3)	0,801
Obstructive HCM	1(2)	1(2,6)	0(0)	
Bicuspid aortic valve	1(2)	1(2,6)	0(0)	

**Indicates subgroups with significant difference.*

Table 2. Laboratory findings and clinical outcomes of patients

	Overall study population	Without mortality (m:38)	With mortality (n:12)	P value
Laboratory Findings				
WBC	9,55(3,2-37,7)	9,4(3,2-37,7)	13,1(5,8-23,7)	0,265
NE	7,1(2,38-30,1)	7,1(2,4-30,1)	7,9(3,4-18,5)	0,229
LYM	1,5(0,4-4,1)	1,4(0,5-4,1)	1,8(0,4-4,1)	0,345
Hb	10,7(5,5-16,5)	11,4(5,5-16,5)	9,6(6,4-13,6)	0,04
PLT	228(33-483)	218(35-446)	261(33-483)	0,525
CRP	76(1-559)	67,1(1-559)	134(4,1-387)	0,140
ESR	(53(3-163)	53(4-131)	85(20-163)	0,325
Cr	1,2(0,5-9,9)	0,88(0,47-6,4)	1,76(0,79-9,9)	0,002
Bilirubin	0,5(0,2-5,4)	0,52(0,16-5,4)	0,69(0,22-3,2)	0,676
RF	14±8,9	13(0-38,5)	17,5(15,9-19)	0,366
SII	1024(126-13632)	961(126,5-5033)	1508(438-13637)	0,469
Blood culture				
Culture negative	14(28)	10(26,3)	4(33,3)	0,718
Staphylococcus aureus	8(16	5(13,2)	3(25)	0,396
Viridans Streptococci	6(12)	6(15,8)	0(0)	
Enterobacteriacea	3(6)	3(7,9)	0(0)	
Enterococci	7(14)	6(15,8)	1(8,3)	
Brucella spp	2(4)	1(2,6)	1(8,3)	
Gemella	1(2)	1(2,6)	0(0)	
KNS	8(16)	6(15,8)	2(16,7)	
Candida	1(2)	0(0)	1(8,3)	
Embolic event				
Peripheral	2(4)	2(5,2)	0	0,001
Spleen	4(8)	3(7,8)	1(8,3)	
Cranial	8(16)	2(5,2)*	6(50)*	
Absence of surgery	21(42)	10(26,3)	11(91,6)	<0,001

Table 3. Univariate and multivariate analyses showing the predictors of in hospital mortality

	Univariate logistic regression				Multivariate logistic regression			
	OR	95% Confidence interval		P value	OR	95% Confidence interval		P value
DM	6,8	3,014	15,68	0,022	3,9	0,794	19,29	0,094
CAD	4,5	1,147	17,749	0,031	7,6	1,397	41,429	0,019
Hemoglobin	0,7	0,534	1,033	0,077				
Creatinine	1,65	0,985	2,784	0,057				
Cerebral emboli	11,66	2,276	59,798	0,003	3,0	0,640	14,448	0,162
Lack of surgery	28,6	3,25	251,277	0,002	48,2	6,8	341,352	<0,001

Epidemiology

OP-042

Covid-19 and acute myocarditis: Current literature review and diagnostic challenges

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Background and Aim: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a new variant form of coronavirus that is responsible of coronavirus disease of 2019 (Covid-19). Although the virus mainly infects the lung epithelial cells causing respiratory signs and symptoms, there has been an upsurge of cases who presented with Covid-19-induced acute myocarditis. Currently, several mechanisms have been proposed to explain the underlying pathophysiology of Covid-19-related acute myocarditis. It has been suggested that a direct viral contact through an angiotensin-converting enzyme 2 (ACE-2) signaling pathways might have a role in the myocardial injury. In addition, cytokine release syndrome has been proposed to be the main pathophysiology of Covid-19-induced acute fulminant myocarditis. In this case-based review, we aimed to describe the clinical characteristics, imaging findings, and in-hospital course of acute myocarditis as well as the limitations in regard to myocarditis diagnosis.

Methods: We performed a review of the literature of all patients who were reported to have Covid-19-induced acute myocarditis using the databases of PubMed, Embase, and the Cochrane. All databases have been searched on June 2020 for the following search inputs: ''covid-19, acute myocarditis' and 'covid-19, acute myopericarditis'. In total, 16 case reports have found to be related with Covid-19-induced acute myocarditis. Despite the fact that neither endomyocardial biopsy (EMB) nor cardiac magnetic rezones imaging (CMR) have been performed in several reports, the cases reported under the title of acute myocarditis have been included in order to provide the current circumstance in terms of acute myocarditis in Covid-19 era.

Results: We observed that ECG findings in most of the Covid-19 patients were non-specific, including diffuse ST-segment elevation, non-specific intraventricular conduction delay, sinus tachycardia, and inverted T waves in anterior leads. Echocardiographic findings of Covid-19 acute myocarditis patients ranged from the preserved left ventricular ejection fraction (LVEF) without segmental abnormalities to reduced LVEF with global hypokinesia. Interestingly, a few patients with Covid-19-induced acute fulminant myocarditis were steroid responsive and had an amelioration with glucocorticoid and immunoglobulin therapy.

Conclusions: Despite the Covid-19 pandemic worldwide, a limited number of cases has been shared in the current literature. There are a lot of difficulties for the differential diagnosis of acute myocarditis in the context of Covid-19 and the information about Covid-19- related acute myocarditis remains unclear. Also, there is no consensus about the diagnostic and treatment algorithms in patients with Covid-19 acute myocarditis. Hence, further studies and case reports on Covid-19-associated acute myocarditis are needed to clarify an appropriate approach to these patients.

Table 1. Baseline characteristics of all Covid-19 acute myocarditis patients

	Age, sex	Risk factors	Presenting symptoms	Admission findings	ECG findings	Lab findings
Case 1	53, F	-	Fatigue, chest pain and dyspnea	Fever: 36.6 oC HR:100 beats SBP: 90 mm Hg DBP:50 mm Hg Spo2:98	Minimal diffuse STsegment elevation (more preominent in the inferior and lateral leads), and an STsegment depression with T-wave inversion in lead V1 and aVR	WBC: 8.0×103/μL Lymphocyte: 1.4×103/μL CRP: 25 mg/dl D-dimer: 500 U/F Troponin: 300 ng/L BNP: -
Case 2	21,F	-	Coughing, sputum, diarrhea, and shortness of breath	-	Nonspecific intraventricular conduction delay and multiple peemature ventricular complexes	WBC: - Lymphocyte: - CRP: - D-dimer: Troponin: 1.26 ng/L BNP: 1929 pg/mL
Case 3	63,M		Shortness of breath and chest tihgtness after activity	Fever: 39.3 oC HR: - SBP: - DBP: - Spo2: 91	Sinus tachycardia and no ST-segment elevation	WBC: - Lymphocyte: - CRP: - D-dimer: - Troponin: 11.37 g/L BNP: 22.600 pg/mL
Case 4	69,M	Hypertension	Cough, fever, dyspnea	Fever: 39.1 oC HR: - SBP: - DBP: - Spo2: 91	Inverted T waves in anterior leads	WBC: 15.400 g/L Lymphocyte: 141.9×10□ per L CRP: - D-dimer: - Troponin: 9002 ng/L
Case 5	17,M	Obesity, asthma,spondylolysis	fever and neck pain, diarrhea	Fever 103 °F HR: 150 bpm SBP: 79 mm Hg DBP: 66 mm Hg Spo2: 91	Sinus tachycardia and T-wave inversion particularly in the inferior leads	BNP: 22.600 pg/mL WBC: 15.4 g/dL Lymphocyte:0.9×103 /μL CRP: 167 mg/L D-dimer: 1218 ng/mL Troponin: 2.97 ng/ml BNP: 2124 pg/mL
Case 6	31,M	-	Dyspnea exertion and low-grade fever	Fever: 37.8 oC HR: 70 bpm SBP: 110 mm Hg DBP: 70 mm Hg Spo2: 98	Normal findings	WBC: - Lymphocyte: - CRP: 105 mg/L D-dimer: - Troponin: 2.97 ng/ml BNP: -
Case 7	57,M	Hypertension	Shortness of breath, fevers, cough, myalgia, decreased appetite, nausea, and diarrhea	Fever: - HR: - SBP: - DBP: - Spo2: -	Sinus tachycardia without ST/T wave changes	WBC: - Lymphocyte: Lymphopenia CRP: Elevated D-dimer: - Troponin: Rapid rise BNP: Rapid rise
Case 8	20,M	-	Febrile sensation and chest pain	Fever: 39 oC HR: 111bpm SBP: 149 mm Hg DBP: 63 mm Hg Spo2: 97	-	WBC: 6.74 × 109 per L Lymphocyte: Lymphopenia CRP: 0.0812 g/L D-dimer: - Troponin: 0.572 ng/mL BNP: 127 ng/L
Case 9	33,M	Hypertension	Chest pain, fever and muscle ache.	Fever: 37.3 oC HR: 121bpm SBP: 115mm Hg DBP: 79 mm Hg Spo2: -	Ventricular tachycardia.	WBC: - Lymphocyte: - CRP: - D-dimer: - Troponin: - BNP: -
Case 10	78,M	-	Chest pain and shortness of breath.	Fever: 37.3 oC HR: 150 bpm SBP: 115 mm Hg DBP: 79 mm Hg Spo2: -	Atrial fibrillation with 150 beat/minute and concave ST elevation except for aVR lead	WBC: Leukocytosis Lymphocyte: Lymphopenia CRP: 94.6 mg/L D-dimer: - Troponin: 998.1 ng/L BNP: 127 ng/L
Case 11	64,M	Hypertension, hyperlipidemia	Dyspnea	Fever: - HR: 80 bpm SBP: 85 mm Hg DBP: 50 mm Hg Spo2: 70	Non-specific T wave changes	WBC: Leukocytosis Lymphocyte: Lymphopenia CRP: 94.6 mg/L D-dimer: - Troponin: 0.17 ng/ml. BNP: -
Case 12	71,F	Multiple myeloma	Fever, cough and dyspnea	Fever: - HR: 125 bpm SBP: 70 mm Hg DBP: 41 mm Hg Spo2: 70	1 mm ST elevation in leads V2–V6 with associated Q waves in leads V4–V6	WBC: - Lymphocyte: - CRP: - D-dimer: - Troponin: 1.6 ng/ml BNP: -
Case 13	45,F Pregnant (39 weeks)	Obesity, gestational diabetic	Contractions and emesis	Fever: 99.6 oF HR: 120 bpm SBP: 183 mm Hg DBP: 114 mm Hg Spo2: 96	Nonspecific T-wave abnormalities	Lymphocyte: - CRP: - D-dimer: - Troponin: 0.046 ng/mL BNP: 114 pg/mL
Case 14	Case 26, F Pregnant (33 weeks)	Obesity, polycystic ovary syndrome,	Shortness of breath, dyspnea,	Fever: 99.6 oF HR: 130 bpm SBP: 110 mm Hg DBP: 70 mm Hg Spo2: 95	Supraventricular tachycardia	WBC: - Lymphocyte: - CRP: 7.68 mg/dL D-dimer: - Troponin: 0.046 ng/mL, BNP: <10 pg/mL,
Case 15	64,M	Pulmonary sarcoidosis and epilepsy	Chest pain and dyspnea.	Fever: 39.3°C HR: - SBP: - DBP: - SpO2: -	Unremarkable	WBC: 18.7 gr/L Lymphocyte: - CRP: - D-dimer: 1210 ng/ml Troponin: 1843 ng/L BNP: -
Case 16	59,F	Hypertension, cervical degenerative arthropathy, chronic lumbar radiculopathy, erythema nodosum, migraine	Anginal chest pain in the absence of respiratory symptoms.	Fever: 39.3°C HR: - SBP: 75 mm Hg DBP: 53 mm Hg Spo2: 96	Concave ST-segment elevation and PR-segment depression, as well as low voltages	WBC: 14.17x 109/L Lymphocyte: 2.59x109/L CRP: - D-dimer: 23.242 ng/mL Troponin: Elevated BNP: 4421 ng/L

F; female, M; male, HR; heart rate, SBP; systolic blood pressure, DBP; diastolic blood pressure, WBC; white blood cell, CRP; c-reactive protein, BNP; brain natriuretic peptide.

Table 2. Imaging findings as well as in-hospital treatment and course of all Covid-19 acute myocarditis patients

	Echocardiographic 9 findings	CMR imaging findings	EMB findings	In-hospital treatment	In-hospital course
Case 1	Diffuse hypokinesia, with an estimated left ventricle ejection fraction of 40%	Diffuse biventricular hypokinesia, especially in the apical segments, and severe LV dysfunction	-	- Hydroxychloroquine (200 mg twice daily), lopinavir/ritonavir (2 tablets of 200/50 mg twice daily), and intravenous methylprednisolone (1 mg/kg daily for), 50 mg of kanrenone, 25 to 50 mg of furosemide, and 2.5 mg of bisoprolol	Cardiogenic shock, clinical follow-up
Case 2	Severe left ventricular systolic dysfunction	A diffuse high signal intensity in the left ventricle myocardium on T2 short inversion recovery image	-	-	Discharged
Case 3	An enlarged left ventricle (61 mm), diffuse myocardial dyskinesia along with a low left ventricular ejection fraction of 32%	-	-	High-flow oxygen, lopinavir– ritonavir, interferon α-1b, methylprednisolone, immunoglobulin, piperacillin– tazobactam, and continuous renal replacement therapy	Cardiogenic shock, exitus
Case 4	Mild left ventricle hypertrophy, the left ventricular ejection fraction and wall motion were within normal limits	Subepicardial late gadolinium enhancement of the apex and inferolateral wall— suggestive of myocarditis	-	- Hydrocortisone,aspirin, fundaparinux	Acute respiratory distress syndrome, discharged
Case 5	Left ventricular ejection fraction qualitatively noted to be mildly depressed without obvious intracardiac clots or pericardial effusion	Normal size left ventricle (LV) with mildly decreased systolic function (40%) and normal right ventricular (RV) size with mildly diminished systolic function (RVEF of 39%). There was an area of mid-wall late gadolinium enhancement at the inferior LV–RV junction coresponding to an area of increased T2 signal, as well as an area of hypokinesia	-	Hydroxychloroquine, piperacillin/tazobactam, enoxaparin	Discharged
Case 6	Mild left ventricular dysfunction	Normal left ventricular size with mildly reduced ejection fraction of 50%. T2-weighted sequence with its post-analysis showed edema/inflammation in the mid inferoseptal and inferior wall. Late gadolinium enhancement showed subepicardial fibrosis in the mid inferior wall	-	- Bisoprolol and lisinopril	Discharged
Case 7	Moderate diffuse hypokinesia with relative apical sparing and a left ventricular ejection fraction of 35-40%	Diffuse bi-ventricular and bi-atrial edema with a small area of late gadolinium enhancement	-	Hydroxychloroquine, azithromycin, ceftriaxone,methylprednisolone, colchicine	Discharged
Case 8	-	Short tau inversion recovery (STIR) sequence revealed a subepicardial high signal intensity in the mid posterolateral wall of the left ventricle which suggests myocardial wall edema	-	Hydroxychloroquine, azithromycin, ceftriaxone, tigecycline, favipiravir, colchicine	Discharged
Case 9	-	The signal of T2 weighed image in the apical region of the left ventricle was increased, which indicated the possibility of myocardial cell edema. Left ventricular systolic function was slightly decreased	-	-	-
Case 10	Not being able to perform echocardiography	Not being able to perform magnetic resonance imaging	-	Furosemide, beta-blocker and angiotensin converting enzyme inhibitor was added to his Covid-19 specific therapy.	-
Case 11	Normal left ventricle ejection fraction of 70–75% with no regional wall motion abnormalities	-	-	Aspirin 81 mg, clopidogrel 75 mg,heparin, azithromycin, hydroxychloroquine, meropenem, tocilizumab, norepinephrine, phenylephrine, vasopressin, atracurium, propofol, fentanyl	Intubated
Case 12	Normal left ventricle ejection fraction of 65–70% with no regional wall motion abnormalities	-	-	- Aspirin 81 mg, clopidogrel 75 mg, heparin, azithromycin, cefepime, vancomycin, tocilizumab, norepinephrine, phenylephrine, midazolam and fentanyl	Intubated
Case 13	Moderately reduced left ventricular ejection fraction of 40% with global hypokinesia	-	-	IV methylprednisolone, hydroxychloroquine, tocilizumab	Intubated, (a primary cesarean)
Case 14	Moderately reduced reduced left ventricular ejection fraction of 40-45% with global hypokinesia	-	-	Metoprolol	Cesarean
Case 15	Moderately reduced reduced left ventricular ejection fraction of 47%.	T2-mapping sequences showed myocardial edema and subepicardial late gadolinium enhancement in the anterior interventricular septum, in the inferior and inferolateral walls	-	Piperacillin/tazobactam	Discharged
Case 16	Preserved left ventricular ejection fraction without segmental abnormalities, and moderate pericardial effusion with no clear signs of hemodynamic deterioration	-	-	Immunoglobulins (80 mg/d) methylprednisolone (500 mg/d), antiviral treatment consisting of interferon-B (0.25 mg/48 h) and ritonavir/lopinavir (400 mg/100 mg/12 h)	Cardiogenic shock

CMR; cardiac magnetic resonance, EMB; endomyocardial biopsy.

Epidemiology

OP-043

Comparison of risk scoring models used in preoperative evaluation of patients underwent noncardiac surgery

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Background and Aim: A number of scoring systems are used to assess the overall mortality risk in non-cardiac surgery patients. Preoperative cardiovascular assessment tests and some biomarkers have shown to be effective in predicting postoperative complications and mortality. In this study, we aimed to compare three highly recommended preoperative risk stratification models and the predictive value of preoperative baseline clinical parameters for risk assessment.

Methods: Retrospectively, 1242 noncardiac surgery patients who had pre-operative cardiology evaluation were included in our study. All co-morbidities, biochemical parameters, echocardiographic and electrocardiographic findings were evaluated. In addition, ASA, Lee Index, Gupta scores of all patients were calculated and their results were compared with each other.

Results: 671 (50.6%) of the patients were male and the mean age was 63.6±11. All-cause mortality was observed in 47 (3.8%) patients.The main characteristics of patients were shown in table 1. Preoperative body mass index (BMI) and hemoglobin (Hb) values were significantly lower and mean age, C-Reaktive protein (CRP), sistolik pulmonary artery pressure (sPAB) values were significantly higher in patients with all-cause death. The incidence of atrial fibrillation (AF), hypertension (HT), chronic kidney disease (CKD), cerebrovascular disease (CVD), previous myocardial infarction (MI), chronic obstructive pulmonary disease (COPD), Diabetes Mellitus (DM) with using insulin were significantly higher in patients with all-cause death. Additionally, Lee index, Gupta and ASA scores were significantly higher in this group (Table 2). BMI (1), sPAB (2), CRP (3), and ASA (4) score were detected as independent predictors of all-cause mortality (OR1:0,9, p 1: 0.03, CI1:0.84-0.99, OR2: 1.0, p2 0.002, CI2: 1.0-1.1, OR3:1.0 p3:0.001, CI3:1.0-1.1, OR4:2.1, p4:0.005, CI4:1.2-3.7, respectively). Cardiac morbidities (MI, death, arrhythmia, heart failure) were observed in 28 (2.2 %) patients. Lee index (1), LVEF (2), sPAB (3) and COPD (4)were detected as independent predictors of these endpoints (OR1:1.5, p 1: 0.02, CI1:1.0-2.3, OR2: 0.9, p2 0.003, CI2: 0.8-0.9, OR3:1.0 p3:0.001, CI3:1.0-1.1, OR4:6.2, p4:0.005, CI4:1.7-22.5, respectively).

Conclusions: As a result of this study high ASA score, CRP, sPAB and low BMI are independent predictors of all-cause mortality. In addition high Lee Index, sPAB, low EF and coexistence of COPD are independent predictors of cardiac morbidity and mortality. ASA score is more effective to predict all-cause mortality and Lee index is more predictive for cardiac mortality and morbidity.

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Table : Main Characteristics of Patients with all cause Mortality and Survivors

	Patients with all cause mortality n:47	Survivors n: 1189	p
Age (year)	69.8 ±15.2	63.4±16.2	0.008
Men (n%)	28(59.6%)	596(50.1%)	0.20
BMI	26.1±4.9	28.1±5.9	NS
HT (n%)	38(80.9%)	781 (65.7%)	0.03
HL (n%)	15(31.9 %)	316(26.7%)	0.42
Smoker (n%)	15 (4.8%)	308(26%)	0.36
CAD (n%)	14 (29.8%)	246 (20.7%)	0.13
COPD (n%)	8 (17.4%)	90 (7.6%)	0.017
PASP (mmhg)	35.5±13.4	29.5±9.4	<0.001
LVEF (n%)	56.4±8.8	58.3±6.4	0.56
DM (n%)	16 (34%)	341 (28.7%)	0.42
DM + insulin usage (n%)	11 (9.9%)	118 (23.4%)	0.003
Previous Miyocard infarction (n%)	13 (27.7%)	130 (11%)	<0.001
Previous CABG (n%)	9 (19.1 %)	94 (7.9%)	0.006
CVA (n%)	6 (12.8%)	59(5%)	0.019
PAD (n%)	9 (19.1%)	61 (5.1%)	<0.001
CKD (n%)	12 (25.5 %)	148 (12.4%)	0.009
HF (n%)	3 (6.4%)	66(5.6%)	0.80
AF (n%)	11 (23.4 %)	148 (12.5%)	0.028
HB	10.7±2.0	12.2±2.1	<0.001
WBC	15.9±4.7	8.1±3.8	<0.001
CRP	67.8±8.0	28.7±5.6	<0.001
Creatinine (mg/dl)	1.2±1.4	1.3±1.7	0.90
ASA Score	3.0±0.9	2.2±0.7	<0.001
Guoss Score	2.5±5.0	0.5±1.2	<0.001
LEE Index	3.8±4.1	1.7±2.6	<0.001

NS: HT: Hypertension, HL: Hyperlipidemia, LVEF: Left ventricular ejection fraction, PASP: Pulmonary artery systolic, CAD: Coronary artery disease, DM: Diabetes Mellitus, HF: Heart failure, COPD: Chronic obstructive pulmonary disease, CVH: Cerebrovascular disease, CKD: Chronic Kidney Disease.

Epidemiology

OP-044

Association between medication adherence and outcomes in atrial fibrillation patients treated with oral anticoagulants: An observational follow up study

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Background and Aim: Medication adherence which is a dynamic process, should be closely monitored as it affects the success of oral anticoagulants. The aim of the study is to evaluate the association between medication adherence and short term outcomes and to explore the factors associated with non adherence in non valvular atrial fibrillation patients receiving oral anticoagulants.
Methods: This observational follow up study was conducted at Eskisehir State Hospital in Turkey. The patients who were diagnosed with non valvular atrial fibrillation and who were taking oral anticoagulants were recruited from outpatients between November 2018 and April 2019. Patients who were included in the study, re-interviewed 6 months after the initial meeting. Data was collected through face-to-face interview method using a questionnaire form. Chi square test, Mann Whitney U analysis and multiple logistic regression analysis were used in the study. Statistical significance level was accepted as p<0.05.
Results: Four hundred and seventy eight patients were enrolled and 302 (63.2%) of them were female. The average age was 71.1±8.9 years. Two hundred seventy seven (57.9%) patients were found to be adherent to oral anticoagulant treatment. In the study, oral anticoagulant treatment adherence was found to be less in non-smokers, those with no history of other chronic diseases, those with poor subjective health perception, those who take double dose after forgetting to take it on time and those who did not take their medication every day at the same time but no association was found with health literacy level and memory, orientation and concentration status. In the second interview 392 (82.0%) patients were reached. In the end of the follow up period, no difference was found in terms of bleeding and thromboembolic complications between adherent and non adherent patients. (RR (95% CI), 1.381 (0.689-2.769), 0.884 (0.284-2.752), respectively).
Conclusions: In this study almost one in two patients were found to be adherent to oral anticoagulant treatment. Further studies are needed to identify factors responsible for non adherence and evaluate the impact on outcomes.

Interventional cardiology / Cover and structural heart diseases

OP-045

Impact of different degrees of oversizing on procedural and clinical outcomes after transcatheter aortic valve implantation using sapien XT valve

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Background and Aim: Paravalvular aortic regurgitation (PAR) is a common and serious complication after transcatheter aortic valve implantation (TAVI). The aim of this study was to examine the impact of different degrees of oversizing on the incidence and severity of PAR for the Edwards SAPIEN XT device and to evaluate whether the oversizing could provide useful information about valve selection.
Methods: This study retrospectively analyzed 121 patients (80.2±6.7 years) with severe symptomatic aortic stenosis who underwent TAVI using SAPIEN XT from June 2013 to February 2020. All patients underwent preprocedural multidetector computed tomography (MDCT). Patients were classified into three groups according to the degree of pre-procedural MDCT area oversizing: below 10%, 10% to 20%, and above %20. Oversizing was calculated using the following formula: % oversizing = (prosthesis nominal area / MDCT derived annular area - 1) * 100. In order to calculate the cut off value of oversizing, receiver operator characteristic (ROC) analysis was performed and the value with the highest sensitivity and specificity value was considered as cut off. Multivariate analysis was performed to determine the predictors of mild or greater PAR. Procedural data and clinical outcomes were evaluated by The Valve Academic Research Consortium-2 definitions.
Results: There were no differences in procedural and clinical events associated with different degrees of oversizing. Overall, mild PAR was present in 39.7% of patients (58 of 121) and moderate PAR was present in 11.6% of patients (14 of 121). No severe PAR was observed. Increasing oversizing ratios are associated with lower rates of mild or greater paravalvular aortic regurgitation: oversizing ≤10%, 68.1%; oversizing 10% to 20%, 50.0%; and oversizing >20%, 30.6%; p=0.003. On ROC analysis, an MDCT area oversizing a percentage value of 13.6% was the optimal threshold value to predict mild or greater PAR (AUC: 0.665 95% confidence interval (CI), 0.568 to 0.762; p=0.002). On multivariate analysis, a lower percentage of oversizing was an independent predictor of mild or greater PAR (odds ratio 2.645; 95% CI, 1.230–5.687; p=0.013).
Conclusions: The degree of area-based oversizing by MDCT is inversely related to the frequency and extent of PAR. Assessment of the oversizing prior to TAVI might serve as an additional tool to guide appropriate device selection to reduce the risk of PAR.

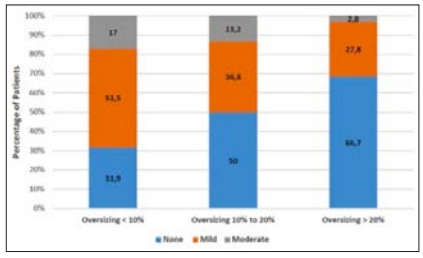

Figure 1. Paravalvular regurgitation assessment after TAVI.

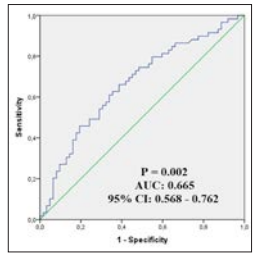

Figure 2. ROC analysis of oversizing for prediction of paravalvular aortic regurgitation.

Table 1. Baseline characteristics of study population

Oversizing	All N = 121	< 10% N = 47	10% - 20% N = 38	> 20% N = 36	P value
Parameters					
Age, years	77.9 ± 7.4	76.7 ± 8.6	79.6 ± 6.4	77.7 ± 6.4	0.195
Sex; male	44 (36.4%)	23 (48.9%)	11 (28.9%)	10 (27.8%)	0.720
STS risk score	6.3 ± 2.8	6.2 ± 3.1	6.0 ± 2.5	6.8 ± 2.7	0.458
Coronary artery disease	68 (56.2%)	24 (51.1%)	23 (60.5%)	21 (58.3%)	0.651
COPD	73 (60.3%)	23 (48.9%)	27 (71.1%)	23 (63.9%)	0.102
Diabetes mellitus	50 (41.3%)	15 (31.9%)	18 (47.4%)	17 (47.2%)	0.246
Renal failure	38 (31.4%)	15 (31.9%)	14 (36.8%)	9 (25%)	0.545
Hypertension	83 (68.6%)	32 (68.1%)	27 (71.1%)	24 (66.7%)	0.917
Prior CABG	22 (18.2%)	14 (29.8%)	4 (10.5%)	4 (11.1%)	0.310
Atrial fibrillation	22 (18.2%)	4 (8.5%)	8 (21.1%)	10 (27.8%)	0.670
Echocardiographic data					
LVEF [%]	55.8 ± 10.8	52.4 ± 13.3	57.1 ± 7.7	55.8 ± 9.5	0.122
Aortic valve area [cm²]	0.73 ± 0.15	0.72 ± 0.16	0.73 ± 0.14	0.76 ± 0.15	0.449
Maximum aortic transvalvular velocity [m/s]	4.3 ± 0.5	4.2 ± 0.6	4.5 ± 0.4	4.3 ± 0.3	0.145
Maximum aortic transvalvular gradient [mm Hg]	79.3 ± 18.8	74.2 ± 17.2	84.4 ± 17.8	80.7 ± 20.4	0.540
Mean aortic transvalvular gradient [mm Hg]	49.3 ± 12.4	47.2 ± 11.3	51.8 ± 12.1	49.3 ± 14.0	0.260

Table 2. MDCT dimensions for all patients and implanted valve size

Oversizing	All N = 121	< 10% N = 47	10% - 20% N = 38	> 20% N = 36	P value
MDCT parameters					
Device-annular sizing ratio, %	15.2 ± 13.8	2.5 ± 6.7	15.4 ± 2.7	31.6 ± 9.6	< 0.001
Annulus area, (mm^2)	484.6 ± 105.8	542.6 ± 119.9	475.0 ± 76.3	419.2 ± 65.7	< 0.001
Annulus perimeter, (mm)	79.0 ± 8.3	83.2 ± 9.0	78.3 ± 6.2	74.1 ± 6.4	< 0.001
Minimum annulus size, (mm)	21.5 ± 2.8	22.9 ± 2.9	21.3 ± 2.5	19.9 ± 2.1	< 0.001
Maximum annulus size, (mm)	26.7 ± 2.9	27.9 ± 3.0	26.5 ± 2.4	25.2 ± 2.5	< 0.001
Mean annulus size, (mm)	24.1 ± 2.7	25.4 ± 2.8	23.9 ± 2.2	22.6 ± 2.0	< 0.001
Eccentricity ratio	0.19 ± 0.07	0.18 ± 0.06	0.19 ± 0.07	0.20 ± 0.08	0.270
Calcification in basal septum	24 (19.8%)	13 (27.7%)	6 (15.8%)	5 (13.9%)	0.223
Height right coronary ostium, (mm)	14.0 ± 2.3	13.6 ± 2.6	14.2 ± 1.8	14.2 ± 2.6	0.477
Height left coronary ostium, (mm)	12.8 ± 2.3	13.0 ± 2.5	13.1 ± 2.1	12.2 ± 2.3	0.268
Bioprosthesis diameter, (mm)	26.3 ± 2.3	26.3 ± 2.5	26.3 ± 2.0	26.4 ± 2.2	0.982
Bioprosthesis area, (mm^2)	550.3 ± 94.8	551.6 ± 104.1	547.1 ± 85.0	552.0 ± 93.9	0.969
Implantation depth, (mm)	6.4 ± 1.7	6.5 ± 1.9	6.4 ± 1.8	6.5 ± 1.6	0.940

Table 3. Relationship between annular sizing ratio and procedural and clinical outcomes

Procedural and clinical Outcomes	All N = 121	< 10% N = 47	10% - 20% N = 38	> 20% N = 36	P value
Tamponade	4 (3.3%)	1 (2.1%)	1 (2.6%)	2 (5.7%)	0.642
Annular rupture	1 (0.8%)	0 (0.0%)	0 (0.0%)	1 (2.9%)	0.294
Conversion to open surgery	1 (0.8%)	0 (0.0%)	0 (0.0%)	1 (2.9%)	0.294
Coronary obstruction	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	-
TAV-in-TAV deployment	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	-
All cause 30-day mortality	10 (8.3%)	2 (4.3%)	5 (13.2%)	3 (8.3%)	0.333
Stroke	4 (3.3%)	2 (4.3%)	0 (0.0%)	2 (5.5%)	0.458
Acute kidney injury	29 (24.4%)	11 (23.4%)	9 (25%)	9 (25%)	0.981
Permanent pacemaker	20 (16.5%)	6 (12.8%)	8 (21.1%)	6 (16.7%)	0.593

Table 4. Predictors of paravalvular aortic regurgitation on univariate and multivariate analysis

Parameters	Univariate analysis		Multivariate analysis	
	OR (95% CI)	P value	OR (95% CI)	P value
Implantation depth	0.993 (0.814 – 1.212)	0.948		
Calcification in basal septum	0.451 (0.177 – 1.152)	0.96		
Oversizing (%)	3.087 (1.469 – 6.489)	0.003	2.645 (1.230 – 5.687)	0.13
Eccentricity ratio	0.009 (0.000 – 1.376)	0.066		

Interventional cardiology / Cover and structural heart diseases

OP-046

The prognostic value of CHA2DS2-VASc score in patients undergoing transcatheter aortic valve implantation

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Background and Aim: Transcatheter aortic valve implantation (TAVI) is the accepted treatment option in patients with severe aortic stenosis who are under intermediate to high surgical risk. Thus, new prognostic markers for patients undergoing TAVI are still of great interest. In this study, our aim is to investigate the impact of CHA2DS2-VASc score on long term mortality in patients undergoing TAVI.

Methods: A total of 253 patients with severe symptomatic aortic stenosis undergoing TAVI were included. Baseline demographic and clinical variables were recorded as well as CHA2DS2-VASc score and long term prognosis. Then, patients were divided into two groups as patients without mortality (178 patients, group 1) and patients with mortality (75 patients, group 2).

Results: The CHA2DS2-VASc score was higher in patients with mortality [4 (3-4); 4 (3-5), p=0.012]. The incidence of congestive heart failure [24 (13.5%); 20 (26.7%), p=0.012] and diabetes mellitus [57 (32.0); 37 (49.3), p=0.009] were higher in group 2. In multivariate logistic regression analysis, STS score [OR:1.105; 95% CI:1.031-1.185; p=0.005], ejection fraction [OR: 0.972; 95%CI: 0.948-0.996; p=0.022], mean aortic gradient [OR: 1.030; 95%CI: 1.004-1.057; p=0.024] and CHA2DS2-VASc score [OR: 1.490; 95%CI: 1.113-1.995; p=0.007] were independent predictors of mortality. ROC curve analysis demonstrated CHA2DS2-VASc score had a good predictive value for mortality with a cut-off value of 4.5 (AUC: 0.595, 95%CI: 0.517-0.674, p=0.017). Additionally, Kaplan-Meier survival analysis revealed that long term mortality was higher in patients with increased CHA2DS2-VASc score (Log Rank p=0.010).

Conclusions: The CHA2DS2-VASc score had a good prognostic value in patients undergoing TAVI.

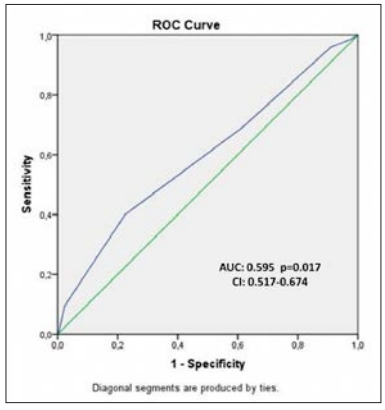

Figure 1. Receiver-operating characteristic curves indicating the discriminative ability of the CHA2DS2-VASc score.

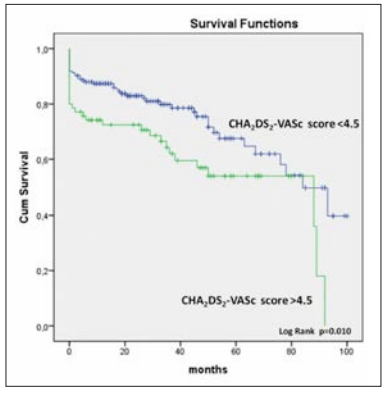

Figure 2. Kaplan-Meier survival curves for low and high CHA2DS2-VASc groups.

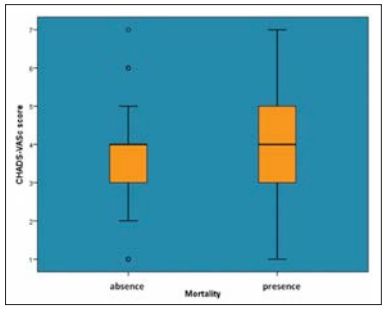

Figure 3. Comparison of CHA2DS2-VASc scores of patients with and without mortality.

Table 1. Baseline demographic and clinical variables of study population

	Patients without mortality (n=178)	Patients with mortality (n=75)	p
Age (years)	78±8	80±8	0.095
Gender (female), % (n)	113 (63.5)	43 (57.3)	0.358
Coronary artery disease, % (n)	112 (62.9)	45 (60.0)	0.662
Smoking, % (n)	48 (27.0)	18 (24.0)	0.624
Body mass index (kg/m2)	27.0 (23.6-29.3)	27.6 (24.1-29.7)	0.325
COPD, % (n)	101 (56.7)	49 (65.3)	0.204
Dyslipidemia, % (n)	47 (26.4)	27 (36.0)	0.125
Atrial fibrillation, % (n)	28 (15.7)	21 (28.0)	0.024
Creatinine (mg/dl)	0.97 (0.77-1.2)	1.1 (0.8-1.3)	0.037
Hemoglobine (g/dl))	11.49±1.68	11.24±1.75	0.284
Leukocytes × 103/mm3	7.8 (6.8-9.1)	7.7 (6.4-9.2)	0.689
Thrombocyte × 103/mm3	232 (192-292)	224 (199-268)	0.825
Balloon expandable valve, % (n)	126 (70.8)	53 (70.7)	0.719
Self expandable valve, % (n)	38 (21.3)	14 (18.7)	0.719
Mechanically expandable valve, % (n)	14 (7.9)	8 (10.7)	0.719
The society of thoracic surgeons (STS) score	9.8 (8.4-11.8)	10.5 (8.4-15.17)	0.011
CHA2DS2-VASc score	4 (3-4)	4 (3-5)	0.012
Ejection fraction (%)	60 (50-60)	60 (40-60)	0.212
Aortic valve area (cm2)	0.75 (0.60-0.84)	0.69 (0.60-0.80)	0.008
Maximum gradient (mmHg)	77.5 (69-90)	83 (73-92)	0.097
Mean gradient (mmHg)	45.5 (42-56)	53 (43-61)	0.016
Postoperative discharging time (days)	7 (5-9)	6 (3-10)	0.074
Cardiac pacemaker implantation, % (n)	30 (16.9)	6 (8.0)	0.100
In-hospital mortality, % (n)	0	29 (38.7)	<0.001
Follow-up time (months)	29 (14-50)	5 (0-37)	<0.001

Table 2. Comparison of variables into the CHA2DS2-VASc score

	Patients without mortality (n=178)	Patients with mortality (n=75)	p
Congestive heart failure/LV dysfunction, n (%)	24 (13.5)	20 (26.7)	0.012
Hypertension, n (%)	126 (70.8)	55 (73.3)	0.682
Age ≥75 years, n (%)	127 (71.3)	59 (78.7)	0.294
Diabetes mellitus, n (%)	57 (32.0)	37 (49.3)	0.009
Stroke/TIA, n (%)	1 (0.6)	1 (1.3)	0.506
Vascular disease, n (%)	50 (28.1)	25 (33.3)	0.494
Age 65-74 years, n (%)	43 (24.2)	11 (14.7)	0.130
Sex (female), n (%)	113 (63.5)	43 (57.3)	0.358

Table 3. Multivariate logistic regression analysis giving information about independent predictors of mortality in patients undergoing transcatheter aortic valve implantation

		Multivariate analysis	
	Odds ratio	95% C.I. (Lower-Upper)	p
The society of thoracic surgeons (STS) score	1.105	1.031-1.185	0.005
Ejection fraction	0.972	0.948-0.996	0.022
Mean gradient	1.030	1.004-1.057	0.024
CHA2DS2-VASc score	1.490	1.113-1.995	0.007

Interventional cardiology / Cover and structural heart diseases

OP-048

Feasibility of transcatheter valve implantation in neurological disorders

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Background and Aim: Aortic stenosis (AS) is the most common valve disease for which aortic-valve replacement (AVR) is the only effective therapy, and its prevalence is increasing due to the aging population. Also, with increasing age, neurological disorders (ND) accompany these patients. Transcatheter aortic valve implantation (TAVI) has emerged as an alternative to intermediate or high-risk surgery in patients with comorbid conditions. Also important risk factors that are concomitant in ND such as Parkinson's disease, dementia-Alzheimer's disease, cognitive impairment, intracranial tumor, and epilepsy which is not included in the traditional risk scoring system. There is still no consensus on the treatment of ND with severe symptomatic aortic stenosis (AS). The purpose of this study to evaluate the feasibility and safety of TAVI in patients with ND.
Methods: Five hundred and fifty six consecutive symptomatic severe AS patients who underwent TAVI,of whom 26 patients had a ND, were included in this retrospective study.Follow-up was performed post-procedural, after 30 days, 6 months, and annually.
Results: Twenty six (4.7%) had any neurological disorders. ND patients were predominantly Parkinson's disease (12) patients and the rest were dementia (8), epilepsy (4), Myasthenia Gravis (1), and meningioma (1) (Figure 1). The average age was 79.4±5.7 years in ND group and there was no statistical difference between ND and No ND groups. In addition, 65.4% of ND patients were male, and there was a statistically difference with the no ND group. The admission NYHA class of patients in the ND group was significantly better than in the No ND group. Although the patients in the ND group were considered as high risk by the cardiac team, the STS score of the ND group was significantly lower than that of the No ND group due to neurological disorders are not included in scoring systems. There were no differences in baseline echocardiographic and procedural features and in hospital complications between the groups. Stroke, major bleeding, and pericardial effusion were no seen in the ND group. While no significant difference was observed between the groups in terms of postTAVI paravalvular leak (PVL), postTAVI mean gradient was significantly lower in the ND group. Post TAVI echocardiographic evaluation was performed and it revealed that all of the implanted valves had successful results with no moderate or severe paravalvular in the ND group leak at discharge. In 30-day follow-up, patients have an improvement in functional capacity at both groups with no moderate or severe PVL in the ND group. Mortality during follow-up was similar between the two groups, and there was no statistically significant difference in the Cox regression model in Kaplan Meier survival rates (Figure 2).
Conclusions: In our knowledge, this is the first study in the literature demonstrates that patients with neurological disorders who underwent TAVI had similar procedural success and numerically lower complication rates compared with patients without ND.

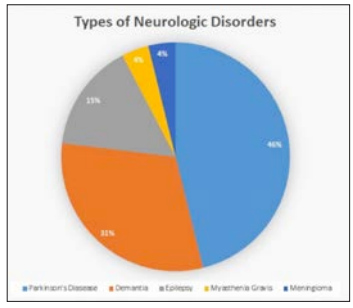

Figure 1. Types of the neurologic disorders.

Table 1. Baseline clinical and echocardiographic parameters

Parameters	All patients n=556	Neurological disorder n=26	No Neurological disorder n=530	p value
Age (years)	77.6±7.9	79.4±5.7	77.5±8.0	0.238
Female n (%)	305 (54.9)	9 (34.6)	296 (55.8)	**0.034**
BMI (kg/m²)	26.9±6.1	25.9±3.7	27.8±6.2	0.279
NYHA n (%)				
- 2	144 (26.1)	5 (19.2)	139 (26.4)	
- 3	313 (56.7)	21 (80.8)	292 (55.5)	**0.047**
- 4	83 (15.0)	-	83 (15.8)	
- Pulmonary edema	12 (2.2)	-	12 (2.3)	
DM n (%)	164 (29.7)	6 (23.1)	158 (30.0)	0.448
HT n (%)	458 (83.0)	21 (80.8)	437 (83.1)	0.760
Previous CABG n (%)	130 (23.6)	4 (15.4)	126 (24.0)	0.313
Moderate to severe COPD n (%)	234 (42.4)	8 (30.8)	226 (43.0)	0.268
AF n (%)	132 (24.0)	8 (30.8)	124 (23.7)	0.408
Previous Stroke n (%)	33 (6.0)	-	33 (6.3)	0.188
STS score n (%)	6.0±3.3	4.1±1.8	6.1±3.4	**0.044**
EuroSCORE II n (%)	9.0±5.7	5.9±3.7	9.1±5.8	0.050
CAD				
- Normal	175 (31.8)	12 (46.2)	163 (31.1)	
- Non-obstructive	241 (43.8)	10 (38.5)	231 (44.1)	0.241
- Obstructive	134 (24.4)	4 (15.4)	130 (24.8)	
Echocardiographic Parameters				
LVEF (%)	51.7±14.0	54.1±10.7	51.6±14.1	0.368
Aortic max gradient (mmHg)	82.0±23.0	75.0±18.0	82.4±23.2	0.111
Aortic mean gradient (mm Hg)	50.5±15.1	45.6±9.8	50.7±15.3	0.092
AVA (cm²)	0.67±0.16	0.71±0.14	0.67±0.16	0.235
sPAP (mmHg)	44.0±16.9	44.6±17.6	44.0±16.9	0.872

Table 2. Procedural characteristics and clinical outcomes

Parameters	All patients n=556	Neurological disorder n=26	No Neurological disorder n=530	p value
Access site n (%)				
- Trans-femoral	536 (96.4)	25 (96.1)	511 (96.4)	0.930
- Trans-axillary	20 (3.6)	1 (3.9)	19 (3.6)	
Closure Method n (%)				
- Prostar	179 (34.2)	5 (20.8)	174 (34.9)	
- Proglide	332 (63.5)	19 (79.2)	313 (62.7)	0.238
- Cut-Down	12 (2.3)	-	12 (2.4)	
Valve type n (%)				
- SAPIEN XT	480 (86.4)	22 (84.6)	458 (86.4)	
- Edwards SAPIEN 3	46 (8.2)	4 (15.4)	42 (7.9)	0.793
- LOTUS	24 (4.4)	-	24 (4.5)	
- ACURATE neo	6 (1.1)	-	6 (1.2)	
Device Success (%)	530 (96.2)	26 (100.0)	504 (96.0)	0.298
Procedural Outcomes				
Pace Maker n (%)	40 (7.3)	3 (11.5)	37 (7.1)	0.677
Stroke n (%)	4 (0.7)	-	4 (0.8)	0.655
Pericardial effusion n (%)	10 (1.9)	-	10 (1.9)	0.777
Major Bleeding n (%)	4 (0.7)	-	4 (0.8)	0.920
Major vascular complication n (%)	30 (5.9)	1 (3.8)	32 (6.4)	0.725
Discharge time (day)	4.5±2.3	4.4±2.1	4.5±2.3	0.800
In-hospital mortality n (%)	22 (4.0)	1 (3.8)	21 (4.0)	0.969
PostTAVI LVEF (%)	54.1±12.1	55.2±10.3	54.1±12.8	0.673
PostTAVI Aortic mean gradient (mm Hg)	10.5±3.9	8.7±3.1	10.6±3.9	**0.028**
PostTAVI PVL n (%)				
- Mild	94 (17.4)	3 (12.5)	91 (18.1)	0.610
- Moderate	5 (1.0)	-	5 (1.0)	
30-Day mortality n (%)	11 (2.2)	1 (4.2)	10 (2.1)	0.509
30-Day NYHA n (%)				
- 1	139 (41.6)	8 (53.3)	131 (41.1)	
- 2	171 (51.2)	7 (46.7)	164 (51.4)	0.425
- 3	24 (7.2)	-	24 (7.5)	
30-Day LVEF (%)	55.2±11.4	53.9±9.8	55.2±11.5	0.655
30-Day Aortic mean gradient (mm Hg)	11.0±4.4	8.9±3.6	11.1±4.4	0.067
30-Day PVL (%)				
- Mild	52 (17.2)	2 (13.3)	50 (17.4)	0.594
- Moderate	6 (2.0)	-	6 (2.1)	
Total Mortality n (%)	158 (28.7)	7 (26.9)	151 (28.8)	0.544

Interventional cardiology / Cover and structural heart diseases

OP-49

Comparing prostar versus proglide closure devices in transfemoral transcatheter aortic valve implantation

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Background and Aim: In the transcatheter aortic valve implantation (TAVI) procedure, transfemoral access was found to be better in terms of morbidity and mortality when compared to other alternative approaches. Despite the technological developments, the materials used in the process are still large compared to routine, which is associated with a significant risk of vascular access complications. Vascular closure devices (VCDs) are used to decrease such complications after transfemoral TAVI. In this study we aimed to to compare the efŞcacy of a Prostar XL-based vascular closure strategy vs. a Perclose ProGlide-based vascular closure strategy. In this study, we aimed to compare the effectiveness of a Prostar vascular closure approach vs. a ProGlide vascular closure approach.

Methods: We retrospectively consecutive severe symptomatic AS patients, except surgical cut-down approaches, undergoing transfemoral TAVI at our hospital. Baseline characteristics, laboratory and echocardiographic data, procedural data, and outcome data were retrospectively collected. Follow-up was performed post-procedural, after 30 days and annually.

Results: A total of 511 patients with an age of 77.6±7.8 years were included in the study, of whom 172 patients were in the Prostar group and 339 patients in the ProGlide group. Baseline clinic features differed between the two groups, and the Prostar group had higher NYHA and STS scores, higher chronic obstructive pulmonary disease ratio and lower AVA. The femoral artery diameters in the MSCT were statistically higher in the Prostar group (right; 7.9±1.0 vs 7.1±1.1 p=<0.001; left: 7.8±1.0 vs 7.3±1.1 p=0.005). SAPIEN XT valve use was significantly higher in the Prostar group, and Edwards SAPIEN 3 and Lotus valve use in the ProGlide group. In the Proglide group, significantly larger valve size was used and less predilatation was performed. VCD failure occurred statistical less frequently in the ProGlide group (3.9% vs. 1.2%, p=0.010). There were no significant differences between the major vascular complications, major bleeding, permanent pacemaker rate, and device success between the two groups. While there was an improvement in the functional capacity of both groups, this was statistically better in the ProGlide group. Although in-hospital and first-year mortality were similar between the two groups, cumulative mortality was fewer in ProGlide group. However Kaplan–Meier analysis of survival curves was significantly different in these groups, adjusted Cox regression analysis after the differences in basal features of survival curves overall survival probability was not significantly different in those patients (p=0.654; HR=1.296, 95% CI 0.601-2.794)

Conclusions: This study demonstrates that the rates of VCD failure is higher in Prostar VCD. However, major vascular complications were comparable between the two vascular closure strategies, ProGlide-based vascular closure strategy was associated with lower rates of cumulative mortality.

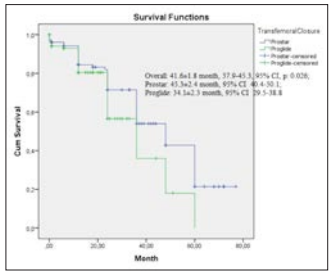

Figure 1. Kaplan–Meier analysis of survival curves in patients with all Prostar and Proglide groups. Overall survival probability was significantly different in those patients (Overall: 41.6±1.8 month, 37.9-45.3, 95% CI, p=0.026; Prostar: 45.3±2.4 month, 95% CI 40.4-50.1; Proglide: 34.1±2.3 month, 95% CI 29.5-38.8).

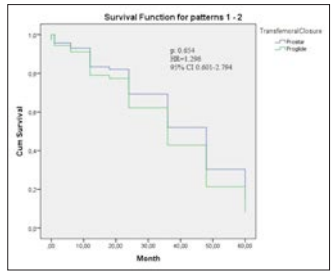

Figure 2. Adjusted Cox regression analysis after the differences in basal features of survival curves in patients with Prostar and Proglide groups. Overall survival probability was not significantly different in those patients (p=0.654; HR=1.296, 95% CI 0.601-2.794).

Table 1. Baseline features of the patients

Parametes	All patients n=511	Prostar n=172	Proglide n=339	p value
Age (years)	77.6±7.8	78.1±7.4	77.4±8.0	0.355
Female n (%)	279 (54.6)	107 (59.8)	172 (51.8)	0.084
BMI (kg/m²)	27.5±6.1	27.6±7.1	27.5±5.2	0.845
NYHA n (%)				
- 2	138 (27.0)	23 (12.8)	115 (34.6)	
- 3	286 (56.0)	108 (60.3)	178 (53.6)	**<0.001**
- 4	77 (15.1)	42 (23.5)	35 (10.5)	
- Pulmonary edema	10 (2.0)	6 (3.4)	4 (1.2)	
DM n (%)	150 (29.4)	48 (26.8)	102 (30.7)	0.355
HT n (%)	418 (81.8)	141 (78.8)	277 (83.4)	0.192
HL n (%)	250 (48.9)	82 (45.8)	168 (50.6)	0.301
Previous CABG n (%)	118 (23.1)	39 (21.8)	79 (23.8)	0.607
Severe COPD n (%)	68 (13.3)	33 (18.4)	35 (10.5)	**0.013**
AF n (%)	123 (24.1)	42 (23.5)	81 (24.4)	0.408
Previous Stroke n (%)	28 (5.5)	14 (7.8)	14 (4.2)	0.088
STS score (%)	6.0±3.3	6.5±3.6	5.4±2.9	**0.004**
Logistic EuroSCORE (%)	22.1±14.1	23.1±14.2	19.2±13.6	0.102
CAD				
- Normal	167 (32.7)	71 (39.7)	96 (28.9)	
- Non-obstructive	220 (43.1)	70 (39.1)	150 (45.2)	**0.046**
- Obstructive	124 (24.3)	38 (21.2)	86 (25.9)	
Echocardiographic and MSCT Features				
LVEF (%)	51.8±14.0	52.2±14.9	51.6±13.5	0.634
Aortic max gradient (mmHg)	82.3±23.2	84.5±20.3	81.1±24.6	0.112
Aortic mean gradient (mm Hg)	50.6±15.1	51.8±13.4	50.7±16.0	0.213
AVA (cm²)	0.67±0.16	0.64±0.15	0.68±0.16	**0.007**
sPAP (mmHg)	44.2±16.8	45.8±16.8	43.4±16.8	0.133
TTE mean aortic annulus (mm)	21.5±0.20	21.2±0.17	21.6±0.22	0.054
MSCT mean annulus (mm)	24.7±2.4	24.6±2.6	24.7±2.3	0.783
MSCT mean annulus area (cm2)	483.2±96.9	481.8±108.6	483.7±92.9	0.874
MSCT Annulus perimeter (mm)	77.5±7.6	77.3±8.4	77.6±7.3	0.783
MSCT Mean right CFA size (mm)	7.5±1.1	7.9±1.0	7.1±1.1	**<0.001**
MSCT Mean left CFA size (mm)	7.6±1.1	7.8±1.0	7.3±1.1	**0.005**

BMI: Body Mass Index; NYHA: New York Heart Association; DM: Diabetes Mellitus; HT: Hypertension; CABG: Coronary artery Bypass Grafting, COPD: Chronic Obstructive Pulmonary Disease, AF: Atrial Fibrillation; STS: Society of Thoracic Surgeons; CAD: Coronary Artery Disease, LVEF: Left Ventricular Ejection Fraction, LA: Left Atrium; AVA: Aortic Valve Area, sPAP: systolic Pulmonary Artery Pressure, CFA: common femoral artery

Table 2. Procedural characteristics and clinical outcomes

Parameters	All patients n=511	Prostar n=172	Proglide n=339	p value
Valve type n (%)				
- SAPIEN XT	441 (86.5)	167 (93.3)	274 (82.8)	
- Edwards SAPIEN 3	41 (8.0)	8 (4.5)	33 (10.0)	**0.019**
- LOTUS	22 (4.3)	3 (1.7)	19 (5.7)	
- ACURATE neo	6 (1.2)	1 (0.6)	5 (1.5)	
Valve size mm n (%)				
- 20	2 (0.4)	-	2 (0.6)	
- 23	212 (41.6)	93 (52.0)	119 (36.0)	
- 25	14 (2.7)	1 (0.6)	13 (3.9)	**0.001**
- 26	210 (41.2)	70 (39.1)	140 (42.3)	
- 27	5 (1.0)	1 (0.6)	4 (1.2)	
- 29	67 (13.1)	14 (7.8)	53 (16.0)	
Predilatation n (%)	364 (71.4)	150 (83.8)	214 (64.7)	**<0.001**
Device Success (%)	496 (97.2)	174 (97.0)	322 (97.1)	0.889
Procedural Outcomes				
Closure Device Failure n (%)	11 (2.2)	7 (3.9)	4 (1.2)	**0.010**
Pace Maker n (%)	39 (7.6)	10 (5.6)	29 (8.7)	0.201
Stroke n (%)	2 (0.4)	1 (0.6)	1 (0.3)	0.657
Pericardial effusion n (%)	6 (1.2)	2 (1.1)	4 (1.2)	0.053
Major Bleeding n (%)	4 (0.8)	2 (1.1)	2 (0.6)	0.426
Major vascular complication n (%)	30 (5.9)	14 (8.1)	16 (5.0)	0.437
Discharge time (day)	4.4±2.2	4.7±2.5	4.3±2.1	0.120
In-hospital mortality n (%)	17 (3.3)	5 (2.8)	12 (3.6)	0.621
PostTAVI LVEF (%)	54.1±12.8	55.3±12.7	53.5±12.8	0.149
PostTAVI Aortic mean gradient (mm Hg)	10.5±3.9	10.1±3.6	10.7±4.0	0.106
PostTAVI PVL n (%)				
- Mild	83 (16.9)	53 (30.3)	30 (9.5)	**<0.001**
- Moderate	5 (1.0)	3 (1.7)	2 (0.6)	
30-Day mortality n (%)	10 (2.2)	3 (1.8)	7 (2.4)	0.509
30-Day NYHA n (%)				
- 1	133 (42.4)	42 (34.7)	91 (47.2)	
- 2	158 (50.3)	65 (53.7)	93 (48.2)	**0.018**
- 3	23 (7.3)	14 (11.6)	9 (4.7)	
30-Day LVEF (%)	55.3±11.4	56.7±10.8	54.5±11.7	0.149
30-Day Aortic mean gradient (mm Hg)	10.9±4.4	10.9±5.2	10.9±3.8	0.106
30-Day PVL (%)				
- Mild	49 (17.0)	26 (24.3)	23 (12.6)	**0.005**
- Moderate	6 (2.1)	4 (3.7)	2 (1.1)	
1st Year Mortality n (%)	48 (12.3)	15 (10.3)	33 (13.5)	0.364
Cumulative Mortality n (%)	138 (27.0)	63 (35.2)	75 (22.6)	**0.002**

AF: Atrial Fibrillation, LBBB: Left Bundle Branch Block, LVEF: Left Ventricular Ejection Fraction, AVA: Aortic Valve Area, EOA: Effective Orifis Area, PVL: Paravalvular Leakage

Interventional cardiology / Cover and structural heart diseases

OP-050

Predictive role of C-reactive protein/albumin ratio on mortality in patients undergoing transcatheter aortic valve implantation

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Background and Aim: The ratio of serum C-reactive protein (CRP) to albumin (CAR) has been investigated in determining the prognosis of diseases such as cancer and critical illness. The aim of our study was to evaluate the prognostic value of CAR in patients with severe aortic valve stenosis undergoing transcatheter aortic valve implantation (TAVI).

Methods: One hundred fourteen patients with severe degenerative aortic stenosis who underwent successful TAVI procedure were enrolled. Patients with evident infection were excluded. The participants were divided into two groups, as 82 patients without mortality and 32 patients with mortality.

Results: CAR was higher in patients with mortality than in patients without mortality (2.38 [1.56-7.67] vs. 1.05 [0.46-3.26] p<0.001) during follow up. The median value of follow up time for all-cause mortality was 19 (6-32) months. CRP, creatinine, CAR were significantly higher and albumin was lower in mortality group. In multivariate logistic regression analysis CAR (OR: 1.231, 95% CI: 1.076-1.407, p=0.002) and creatinine (OR: 2.685, 95% CI: 1.076-6.707, p=0.034) were independent predictors of mortality. ROC-curve analysis demonstrated that, CAR had a good predictive marker for mortality with a cut-off value of 1.158 (AUC:0.751 95% CI:0.661-0.841, p<0.001). Furthermore, Kaplan- Meier survival analysis revealed that long-term mortality was higher in patients with high CAR value (Log Rank p<0.001).

Conclusions: CAR is a simple and good prognostic marker that might help for long-term risk stratification of patients undergoing TAVI.

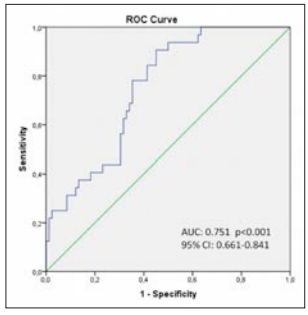

Figure 1. The receiver-operating characteristic curve of CRP to albumin ratio (CAR) for predicting mortality in patients undergone TAVI. Cut-off value: 1.158 (sensitivity: 90.6%, specificity: 54.9%) AUC: area under the curve.

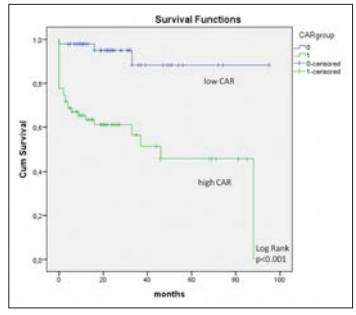

Figure 2. Kaplan-Meier survival curve for low and high CAR groups.

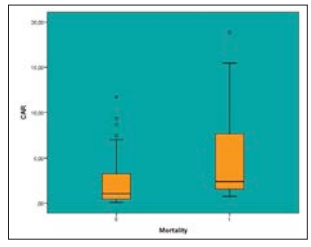

Figure 3. Box-plot of CAR values comparing mortality – and +.

Table 1.

	Patients without mortality (n=82)	Patients with mortality (n=32)	p
Age (years)	78±7	77±9	0.355
Gender (female), % (n)	56 (68.3)	16 (50.0)	0.109
Coronary artery disease, % (n)	50 (61.0)	21 (65.6)	0.645
Smoking, % (n)	23 (28.0)	5 (15.6)	0.253
COPD, % (n)	44 (53.7)	22 (68.8)	0.143
Dyslipidemia, % (n)	24 (32.0)	12 (44.4)	0.246
Hypertension, n (%)	51 (62.2)	20 (62.5)	0.976
Diabetes mellitus, n (%)	30 (36.6)	15 (46.9)	0.313
Stroke/TIA, n (%)	2 (2.4)	0 (0)	0.516
Atrial fibrillation, % (n)	12 (14.6)	7 (21.9)	0.351
Creatinine (mg/dl)	1 (0.80-1.20)	1.20 (0.97-1.51)	0.008
Hemoglobine (g/dl)	11.25±1.41	10.95±1.77	0.348
Leukocytes × 103/mm3	7.84 (6.80-9.10)	8.0 (5.50-9.12)	0.324
Thrombocyte × 103/mm3	237 (199-291)	229 (205-287)	0.940
CRP	4.02 (1.78-10.80)	9.36 (5,35-21.99)	<0.001
Albumin	3.76±0.45	3.46±0.52	0.003
CRP to albumin ratio (CAR)	1.05(0.46-3.26)	2.38 (1.56-7.67)	<0.001
low CAR (<1.158)	44 (53.7)	3 (9.4)	<0.001
high CAR (>1.158)	38 (46.3)	29 (90.6)	<0.001
The society of thoracic surgeons (STS) score	9.0 (7.20-11.00)	10.0 (8.20-12.80)	0.111
Ejection fraction (%)	60 (48-60)	59 (40-60)	0.392
Aortic valve area (cm2)	0.70 (0.60-0.80)	0.70 (0.59-0.82)	0.615
Maximum gradient (mmHg)	78.5 (68-90)	83.0 (71.5-93.5)	0.214
Mean gradient (mmHg)	45.5 (42.0-56.0)	51.0 (43.0-59.5)	0.233
Postoperative discharging time (days)	6 (5-9)	5 (1-9)	0.072
Cardiac pacemaker implantation, % (n)	18 (22.0)	3 (9.4)	0.198

Baseline demographic and clinical variables of study patients.

Table 2.

	Univariate analysis			Multivariate analysis		
	Odds ratio	95% C.I. (Lower-Upper	p	Odds ratio	95% C.I. (Lower-Upper)	p
CAR	1.249	1.097-1.421	0.001	1.231	1.076-1.407	0.002
Creatinine	3.057	1.293-7.225	0.011	2.685	1.076-6.707	0.034

Significant predictors of long-term mortality in multivariable regression analysis.

Interventional cardiology / Cover and structural heart diseases

OP-051

Evaluation of ATRIA, CHA2DS2VASc, the intermountain risk scores, echocardiographic and laboratory parameters to determine the long-term mortality of transcatheter aortic valve implantation procedure

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Background and Aim: As a result of increasing spectrum and frequency of use of transcatheter aortic valve implantation (TAVI), the question of which patients have high mortality has become important. We aimed to determinate the prediction of the long-term mortality of TAVI patients using different risk scores (ATRIA, CHA2DS2VASc, The Intermountain risk scores (IMRS)), echocardiographic and laboratory parameters.
Methods: In this retrospective study, a total of 136 patients, who underwent TAVI procedure in our clinic, between 2010 and 2019 were collected. Three patients were excluded from the study due to lack of data during hospitalization and the follow-up period. The value of three risk scores, echocardiographic and laboratory parameters was assessed in respect of mortality determination in the overall patient population.
Results: Mortality did not develop during follow-up in 73 patients (Group 1) and total mortality was seen in 60 patients (45.1%) (Group 2). No difference was determined between the groups in respect of the baseline demographic, clinical and laboratory parameters. Female gender predominance was detected with 35 females (58.3%) in Group 1 and 44 females (60.3%) in Group 2 (p=0.820). The mean age of patients was determined as 78.90±7.95 years in Group 1 and 78.38±7.14 years in Group 2 (p=0.694). Survival was calculated as mean 1433±124 days(median, 1279 days) (Table 1). Univariate analyses demonstrated that aortic peak gradient and IMRS were risk factors for long-term mortality. Only moderate IMRS (Hazard ratio (HR): 7.533, 95% confidence Interval (CI):1.025-55.338, p=0.047) and high IMRS (HR:9.175 95% CI 1.243-67.704, p=0.030) independently predicted long-term mortality (Table 2). As the main result the IMRS predictive risk score was seen to be acceptable in the determination of long-term mortality of TAVI patients unlike the ATRIA, and CHA2DS2VASc which were not determined to be predictive. No statistical significance was seen between the groups in respect of any other laboratory and echocardiography parameters (Figure 1).
Conclusions: The main benefits of these conventional risk scores are to detect the patient mortality and disability, and to help the physician define these problems and thereby select appropriate patients for the procedure. However, it is unclear whether these conventional risk scores are effective in the prediction of early and late mortality in patients undergoing TAVI procedure. From the evaluation of all the risk scores, laboratory and echocardiograpic parameters, the long-term mortality (>30 days) following the TAVI procedure, can be said to be higher in patients with a moderate and high IMRS. As the data of this study, the IMRS can provide new insights in the prediction of patient outcomes following TAVI and it may be useful to identify patients who will benefit in the long term from the TAVI procedure. As a limitation, larger-scale evaluations are needed to confirm the findings of this study which included a limited number of patients from a single centre.

Table 1.

Variable	Survivors (Group 1)	Non-survivors (Group 2)	p-value
Age(years)	78.38±7.1	78.90±7.9	0. 694
Female Gender(n,%)	35 (58.3%)	44 (60.3%)	0.820
Coronary Artery Disease(n,%)	64 (87.7%)	51(85%)	0.348
Diabetes Mellutus(n,%)	20 (27%)	21(35%)	0.423
Arterial Hypertension(n,%)	66 (90.3%)	50(83%)	0.759
Atrial fibrillation(n,%)	16 (21.9%)	16(26.7%)	0.327
Glomerular filtration rate (mL/dk/1,73m2)	69.50±21.8	68.54±25.6	0. 859
Contrast-induced nephropathy(n,%)	10(13.7%)	9(15%)	0.831
Aortic peak valvular gradient (mmHg)	88.78±22.72	79.17±18.8	0.010
Pre-procedural Left Ventricular Ejection Fraction (%)	55.0±9.13	56.2±8.96	0.448
Systolic Pulmonary Artery Pressure (mmHg)	43.07±14.09	42.63±13.79	0.862

Basal clinic and laboratory parameters of patients.

Table 2.

Variable		Survivors (Group 1)	Non-survivors (Group 2)	p-value
The Intermountain Risk Score(n,%)	High	26 (36%)	27 (45%)	0.006
	Moderate	33 (45%)	32 (53%)	
	Low	14 (19%)	1 (2%)	
ATRIA Risk Score(n,%)	Mean Score	6.97±1.66	7.10±1.79	0.671
	High (≥6 points)	61 (84%)	52 (87%)	0.808
CHADS2VASc2 Risk Score	Mean Score	4.49±1.02	4.50±1.19	0.972
	Score ≥ 2 points	71 (97%)	59 (98%)	1
Mean Decrease in Platelet Count(x109/L)		114.29±53.6	111.95±86.1	0.850

Risk score and pletelet change of the two groups. Only The Intermountain Risk Score had a predictive value for motality on follow-up.

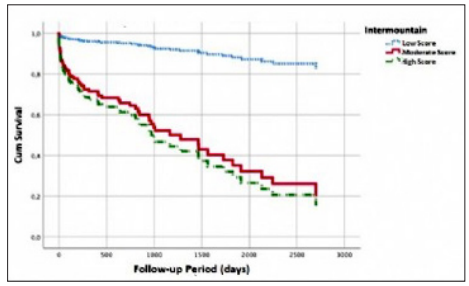

Figure 1. Survival function for the Intermountain Risk Score (IMRS), IMRS is associated with high risk of Transcatheter Aortic Valve Implantation Procedure mortality.

Interventional cardiology / Cover and structural heart diseases

OP-052

EuroSCORE II and STS score as a predictor of acute kidney injury following transcatheter aortic valve replacement: two birds with one stone?

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Background and Aim: Acute kidney injury (AKI) is a significant predictor of mortality in patients who underwent transcatheter aortic valve replacement (TAVR). Early identification and management of AKI can mitigate further complications and improve survival. The incidence and predictors of acute kidney injury in patients with TAVR were evaluated in different studies. Our aim is to evaluate EuroSCORE II and STS score in terms of predicting AKI following.
Methods: One hundred and five patients who underwent TAVR procedure due to severe aortic stenosis in our clinic were retrospectively screened. Demographic, laboratory, echocardiographic and procedural data were collected retrospectively. AKI was defined according to the valve academic research consortium-2 (VARC-2).
Results: Sixty-five (61.9%) patients out of 105 were females with a mean age of 77 (4.88) years. AKI developed in 31.4% of all patients who underwent TAVR. The mean (SD) STS score was (8.03) 2.30 while mean (SD) EuroSCORE II was 10.93 (7.53). Mean (SD) STS score and EuroSCORE II were 14.35 (7.66) and 22.34 (6.07) in patients who developed AKI respectively and 5.26 (2.41) and 4.88 (3.07) respectively in those who did not develop AKI. A EuroSCORE cut-off value of 6.16 exhibited a 91% sensitivity and 68% specificity while STS score cut-off value of 6.30 showed 84% sensitivity and 75% specificity in predicting AKI following TAVR.
Conclusions: STS score and EuroSCORE II which were developed for the prediction of postoperative mortality may be the predictors of AKI in patients following TAVR.

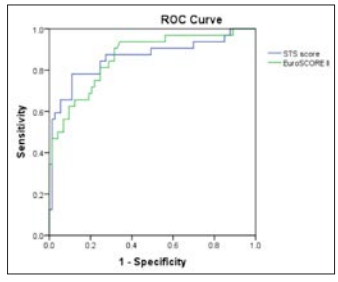

Figure 1. ROC characteristic showing the sensitivity and specificity of STS and EuroSCORE for predicting of acute kidney injury following.

Table 1. Demographics of study population

Variables	Overall population	No Acute Kidney Injury (n=72)	With Acute Kidney Injury(n=33)	p value
Age	77.00±4.88	77.48±4.65	75.91±5.28	0.13
Female, n (%)	65 (61.9)	46 (63)	19 (59.4)	0.73
Hypertension, n (%)	75 (71.4)	49 (67.1)	26 (81.3)	0.17
Diabetes mellitus, n (%)	39 (37.1)	24 (32.9)	15 (46.9)	0.14
Smoking, n (%)	16 (15.2)	11 (15.1)	5 (15.6)	0.94
CAD, n (%)	62 (59.0)	39 (53.4)	23 (71.9)	0.08
Previous CABG, n (%)	24 (22.9)	13(17.8)	11(34.4)	0.06
COPD, n (%)	37 (35.2)	27(37)	10(31.3)	0.57
BMI	25.78±3.48	26.23±3.63	24.71±2.98	0.23
STS score	8.03±2.3	5.26±2.41	14.35±7.66	<0.001
EuroSCORE II	10.93±7.53	4.88±3.07	22.34±6.07	<0.001
Contrast volume, (ml)	206±30	202±25	215±36	0.07

BMI- body mass index, CABG- coronary artery bypass grafting, CAD- coronary artery disease, COPD- chronic obstructive pulmonary disease.

Table 2. Baseline laboratory, electrocardiographic, echocardiographic and postprocedural features

Variables	Overall population	No acute kidney Injury	With Acute kidney Injury	p value
Serum Creatinine (mg/dL)	0.82±0.24	0.97±0.34	1.33±1.17	0.02
Hematocrit (%)	33.23±4.97	34.29±5.06	34.76±4.81	0.67
WBC, (x 103 /uL), median (IQR)	6.63(5.46-8.30)	6.56(5.45-8.35)	6.75(6.08-8.28)	0.84
Platelet, (x103 /μL)	201.08±63.72	217.53±69.04	224.27±78.42	0.67
Left Ventricular EF, %	51.16±10.96	53.30±9.01	46.28±13.39	0.007
AVA, cm2	0.74±0.14	0.74±1.13	0.66±0.16	0.21
Mean Gradient, mmHg	48.95±12.77	49.02±13.33	48.69±10.89	0.94
Max Gradient, mmHg	80.16±18.06	79.86±17.53	81.31±20.66	0.89
AR, moderate-to severe, (%)	13.7	13.7	15.6	0.04
MR, moderate-to-severe, (%)	6.8	3.8	14.3	0.01
MS, (%)	4.8	1.4	12.5	0.01
SPAP, mmHg	44.87±13.22	44.30±13.47	36.82±12.95	0.65
Atrial fibrillation, %	22.9	24.2	18.8	0.51
LBBB, %	12.5	8.2	9.4	0.55
RBBB, %	8.6	8.2	6.2	0.67
Serum creatinine, (md/dL)	1.11±0.43	1.00±0.34	2.45±2.07	<0.001
Hematocrit, (%)	28,45±4.94	29.04±5.14	27.12±4.37	0.29
RBC transfusion, n (%)	53 (50.5)	33(45.2)	20(62.5)	0.10
Prosthetic valve size, mm	25.73±2.49	25.18±2.11	25.19±2.09	0.98
AR, moderate -to-severe, (%)	6.7	6.9	6.1	0.77

AR- aortic regurgitation, AVA- aortic valve area, LBBB-left bundle branch block, MR- mitral regurgitation, MS- mitral stenosis, RBBB- right bundle branch block, RBC- red blood cell, SPAP- systolic pulmonary artery pressure, WBC- white blood cell

Table 3. Predictors of acute kidney injury: Univariate and Multivariate analysis

Parameters	Univariate analysis	Multivariate analysis
Female gender	0.86 (0.37- 2.01) p= 0.724	
Age	0.93(0.85-1.02) p=0.13	
Hypertension	0.47(0.17-1.30) p=0.15	
DM	0.56(0.24-1.30) p=0.17	
Prior CABG	0.41(0.16-1.06) p=0.067	
Contrast exposure in last 5 days	2.82(0.83-9.58) p=0.96	
LVEF	0.94(0.91-0.98) p=0.005	0.98(0.91-1.05) p=0.54
Baseline serum creatinine	1.7(0.99-7.5) p=0.051	1.04(0.30-3.59) p=0.95
Contrast volume	1.01(1.00-1.03) p=0.07	1.01(0.99-1.03) p=0.35
STS score	1.62(1.32-1.98) p<0.001	1.30(1.04-1.62) p=0.02
EuroSCORE	1.29(1.16-1.43) p<0.001	1.26(1.10-1.44) p=0.001

CABG- coronary artery bypass grafting, DM-diabetes mellitus, LVEF- left ventricle ejection fraction.

Methods: This study cohort was divided into two groups: dual PP versus one AS with one PP (AS + PP) used for common femoral artery haemostasis. Dual PP was used for 14F-20F sheaths, whereas AS+PP combination was used for 14-19 F sheaths. The baseline, procedural characteristics and all outcomes (defined according toVARC-2 criteria) were collected and retrospectively compared.
Results: Overall, a total of 185 consecutive patients (51% male, mean age 79 years) with dual PP (n=139) patients vs. one AS with one PP (n=46) were evaluated. Baseline characteristics of both patient groups were similar. There were no statistically significant differences between the dual PP vs. PP +AS groups with regard to the in-hospital Valve Academic Research Consortium-2 (VARC-2) primary endpoints of major vascular complications (7.9% vs. 2.2%, p=0.29), minor vascular complications (21.7% vs. 12.2%, p=0.11), major bleeding (10.1% vs. 8.7%, p=0.79), and minor bleeding (11.5% vs. 21.7%, p=0.08). Patients in AS + PP group had lower rate of arterial stricture (18.7% vs 6.5%, p=0.049), however hematomas tended to occur more frequently in AS+PP group (3.6% vs 10.6%, p=0.059).
Conclusions: Combination of one PP and one 8Fr AS for femoral access haemostasis in patients undergoing TAVR is feasible and safe, in regards of major and minor vascular complications, nor major and minor bleeding with relatively increased rates of hematoma.

Interventional cardiology / Cover and structural heart diseases

OP-053

Hybrid use of ProGlide and Angio-Seal for femoral access haemostasis for transfemoral transcatheter aortic valve replacement

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Background and Aim: Two-device technique using two Perclose devices has been well established for transcatheter transfemoral transcatheter aortic valve replacement (TAVR). Given the lack of data combining one Perclose with one 8F Angioseal, we sought to evaluate whether combination of one Angio-Seal (AS) and one Perclose ProGlide (PP) could achieve haemostasis compared to two PP in patients undergoing TAVR procedure.

Interventional cardiology / Cover and structural heart diseases

OP-054

Increased intima media thickness of the ascending aorta may predict neurological complications associated with TAVI

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Background and Aim: TAVI is increasingly performed with great success in patients with aortic stenosis. However, neurological complications associated with this procedure are still important due to its morbidity and mortality risks. The purpose of this study was to investigate the importance of the features of the aortic valve and ascending aorta to predict the neurological complications associated with TAVI.
Methods: The patients for whom the heart team decided to perform TAVI were included in the study. All patients underwent TEE preoperatively. In order to assess possible neurological complications, cerebral diffusion-weighted MRI was performed pre- and post-operatively. The diameter of the patients' aortic root and ascending aorta, aortic valve scores, the intimate media thickness of the ascending aorta from the long axis of the aorta (AA-IMT) were measured from their TEE records. The value of these parameters assessed through TEE in predicting new lesions after TAVI on MRI scans was investigated.
Results: 108 patients who had severe aortic stenosis and for whom TAVI was decided were included in the study. All patients underwent TAVI performed with Edwards Sapien balloon expandable valve. 31 patients were found to develop a new lesion (MR+) detected on diffusion-weighted MRI after TAVI, while 76 patients did not have any new lesions (MR-). Only two of the MR+ patients were clinically found to have neurological signs, whereas the other patients were considered as having silent infarcts. No significant difference was found between the MR+ and MR- patients as regards age, sex, presence of comorbid coronary artery diseases and left ventricular ejection fraction. In the MR+ group, the incidence of DM was found to be higher (48.4% vs 23.7%). The groups did not have any significant differences in their aortic valve features and scores. However, AA-IMT was found to be higher in the MR+ group (1.8 mm [1.6-2.3] vs 1.4 mm [1.2-1.8] interquartile range). The univariate logistic regression analysis conducted to detect new lesions revealed that AA-IMT and DM led to a significantly increased risk while the multivariate analysis showed that such increased risk existed only for AA-IMT. Assessment with a receiver operating characteristic curve demonstrated that IMT >1.52 mm had a sensitivity of 92% and specificity of 56% in predicting the presence of new lesions on MRI scans.
Conclusions: The features of the ascending are more important than the demographic characteristics of patients and features of the native valve in predicting new lesions on diffusion-weighted MRI scans and thus neurological events after TAVI. To this end, AA-IMT measurement may be used as an easily accessible and simple technique.

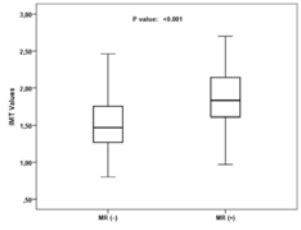

Figure 1.

Interventional cardiology / Cover and structural heart diseases

OP-055

The relationship between contrast nephropathy after transcatheter aortic valve implantation and contrast volume / glomerular filtration rate ratio

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Background and Aim: Acute kidney injury (AKI) after transcatheter aortic valve implantation (TAVI) has been reported to increase mortality and morbidity. The aim of our study was to evaluate the relationship between the rate of contrast volume / glomerular filtration rate (CV / GFR) and the rate of AKI in patients undergoing TAVI.

Methods: This study is a prospective study. Among the 117 patients who underwent TAVI for symptomatic severe aortic stenosis (AS) in our center between January 2017–December 2019, 106 patients were included in the study. The criteria recommended in the second consensus report of valve academic research consortium (VARC) for the diagnosis of AKI are; The serum creatinine level was increased more than 0.3 mg / dL or ≥ 50% increase from the baseline value within 72 hours following the procedure. The baseline, perioperative and postoperative clinical features, echocardiographic and laboratory findings of patients with AKI (AKI (+)) and AKI (AKI (-)) were evaluated by univariate and multivariate analysis. In univariate analysis, all variables those were significant for AKI (p<0.05) were evaluated in multivariate logistic regression analysis model.
Results: 106 patients (71 males, 35 females, mean age 78.45 years) were included in the study. 23 of patients (21.7%) developed AKI. The mean age of patients with AKI (81.95±6.94) was statistically higher. There was no significant difference between the groups in terms of demographic characteristics. Mean GFR was lower in patients with AKI [AKI (+): 49.05±13.46, AKI (-): 67.18±20.05 (p<0.0001)] Creatinine levels were higher in AKI (+) group than AKI (-) group. [1.18±0.34 vs. 1±0.33 (p=0.02), respectively]. The ejection fraction (EF) was significantly lower in the AKI (+) group than in the AKI (-) group [49.56±5.82 vs. 59.06±10.06 (p<0.0001)]. The mean gradient was higher in AKI (+) group. No significant difference was found between the groups in the procedural data and other postoperative complications. The mean CV/GFR value was higher in the AKI (+) group than in the AKI (-) group [3.48±1.50 vs. 2.18±1.08 (p<0.0001)]. Postoperative erythrocyte suspension transfusion was higher in AKI (+) group. A CV/GFR >2.3 predicts acute renal injury after TAVI with a sensitivity of 82.6% and specificity of 71.1%.
Conclusions: Advanced age, low EF and low CV / GFR values are independent predictors of AKI in patients undergoing TAVI.

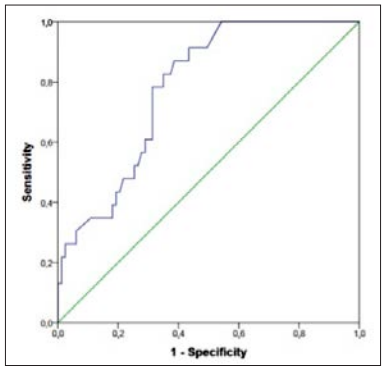

Figure 1. The cut-off value of > 2.3 for CV / GFR ratio yielded 82.6% sensitivity and 71.1% specificity in predicting AKI after TAVI.

Table 1. Baseline demographic and clinical characteristics of the patients

	AKI (+) (n=23)	AKI (-) (n =83)	P
Age	81.95 ± 6.94	77.48 ± 7.47	0.01
Gender (m/f)	16 (69.6%) / 7 (30.4%)	55 (66.3%) / 28 (33.7%)	NS
Diabetes Mellitus (y/n)	7 (30.4%) / 16 (69.6%)	31 (37.3%) / 52 (62.7%)	NS
Hypertension (y/n)	11 (47.8%) / 12 (52.2%)	52 (62.7%) / 12 (52.2%)	NS
History of Coronary Artery Disease (y/n)	16 (69.6%) / 7(30.4%)	57 (68.7%) / 26(31.3%)	NS
History of Peripheral artery Disease (y/n)	10 (43.5%) / 13 (56.5%)	23 (27.7%) / 60 (72.3%)	NS
History of Pulmonary Hypertension (y/n)	10 (43.5%) / 13 (56.5%)	26 (31.3%) / 57 (68.7%)	NS
Chronic Obstructive Pulmonary Disease (y/n)	14 (60.9%) / 9 (30.4%)	36 (43.4%) / 47 (56.6%)	NS
History of Coronary Artery By-pass Surgery (y/n)	1 (4.3%) / 22 (95.7%9	23 (27.7%) / 60 (72.3%)	**0.01**
History of Cerebrovascular Event (y/n)	0 (0) / 23 (100%)	1 (1.2%) / 82 (98.8%)	NS
Creatinine	1.18 ± 0.34	1 ± 0.33	**0.02**
Glomerular Filtration Rate (GFR)	49.05 ± 13.46	67.18 ± 20.05	**<0.0001**
Hemoglobin	10.68 ± 1.77	11.26 ± 1.72	NS
Platelet	214.86 ± 70.08	244.03 ± 79.93	NS
White Blood Count	7.27 ± 1.9	8.11 ± 5.57	NS
STS Score	9.57 ± 3.92	8.28 ± 3.29	NS
Ejection Fraction	49.56 ± 5.82	59.06 ± 10.06	**<0.0001**
Aortic Valve Mean Gradient	49.56 ± 12.03	48.30 ± 9.94	NS
Aortic Valve Area	0.72 ± 0.15	0.75 ± 0.12	NS

Table 2. Procedural data of the patients

	All Patients	AKI (+)	AKI (–)	P
Balloon expandable	46 (43.4%9	10 (43.5%)	36 (43.4%)	NS
Self expandable	60 (56.6%)	13 (56.5%)	47 (56.6%)	NS
Valve size	27.2 ± 2.09	27.56 ± 1.72	27.10 ± 2.18	NS
Contrast Volume	148.5 ± 75.11	155.65 ± 55.62	146.53 ± 79.85	NS
CV/GFR	2.46 ± 1.29	3.48 ± 1.50	2.18 ± 1.08	<0.0001
Major Vascular Complication (y/n)	9 (8.5%) / 97 (91.5%)	5 (21.7%) / 18 (78.3%)	4 (4.8%) / 79 (95.2%)	0.02
Minor Vascular Complication (y/n)	23 (21.7%) / 83 (78.3%)	4 (17.4%) / 19 (82.6%)	19 (22.9%) / 64 (77.1%)	NS
Cerebrovascular Event (y/n)	6 (5.7%) / 100 (94.3%)	1 (4.3%) / 22 (95.7%)	5 (6%) / 78 (94%)	NS
Post-procedural Blood Transfusion (y/n)	26 (24.5%) / 80 (75.5%)	10 (43.4%) / 14 (66.6%)	17 (20.5%) / 66 (79.5%)	0.02
Coronary Occlusion (y/n)	0 (0) / 106 (100%)	0 (0%) / 23 (100%)	0 (0%) / 83 (100%)	NS
Permanent Pacemaker (y/n)	18 (17%) / 88 (83%)	5 (21.7%) / 18 (78.3%)	13 (15.7%) / 70 (84.3%)	NS
Post-procedural Aortic Insufficiency (y/n)	47 (44.3%) / 58 (54.7%)	11 (47.8%) / 12 (52.2%)	36 (43.4%) / 47 (56.6%)	NS

Table 3. Multivariate analyses of the findings

Variables	Odds Ratio	P	95%CI
Increased CV/GFR	2.1	0.002	1.31–3.64
Age	1.09	0.03	1.08–1.18
Lower EF	0.9	0.0001	0.8–0.9

Interventional cardiology / Cover and structural heart diseases

OP-056

Impact of postdischarge care fragmentation on clinical outcomes and survival following transcatheter aortic valve replacement

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Background and Aim: The study aimed to evaluate the prognostic impact of postdischarge care fragmentation in patients undergoing transcatheter aortic valve replacement (TAVR).
Methods: A total of 266 patients undergoing TAVR due to severe aortic stenosis were included in this retrospective cohort study. Patients were assigned into two groups based on presence (n=104) and absence (n=162) of postdischarge care fragmentation. Fragmented care was defined as at least one readmission to a site other than the implanting TAVR center within 90 days. Prognostic impact of care fragmentation on clinical outcomes and predictors of long term mortality were investigated.
Results: Increased major vascular complication (16.3 vs 8.0%, p=0.037), permenant pacemaker implantation (14.4 vs 6.2%, p=0.025) and acute kidney injury (22.1 vs 14.2%, p<0.001) were reported in fragmented care group. Although early mortality (6.7 vs 4.3%, p=0.152) was similar between groups, there was significant difference in 5-year mortality (66.3 vs 45.7%, p<0.001). In a univariate regression analysis fragmented care, age, chronic obstructive pulmonary disease, glomerular filtration rate and pulmonary artey systolic pressure were significantly associated with five-year mortality. Fragmented care (HR 2.615, 95% CI 1.522-4.493; p<0.001), age (HR 1.042, 95% CI 1.005-1.079; p=0.024) and chronic obstructive pulmonary disease (HR 2.134, 95% CI 1.138-4.002; p=0.018) were found to be significant independent predictors of five-year mortality in a multivariate analysis, after adjusting for other risk.
Conclusions: Fragmented care has a significant prognostic impact on clinical outcomes and survival.

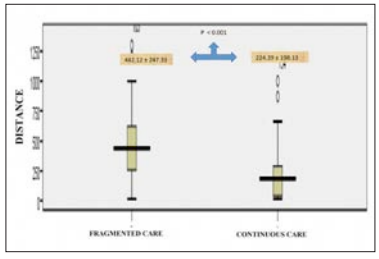

Figure 1. Box plot of the distance from patients home to index hospital in fragmented care vs continuous care.

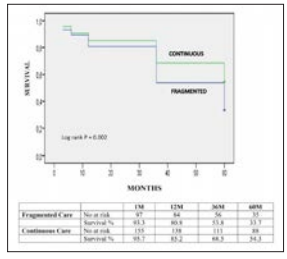

Figure 2. Kaplan Meier survival curves for patients with fragmented care vs continuous care.

Table 1. Clinical outcomes

	FRAGMENTED CARE (n = 104)	CONTINUOUS CARE (n = 162)	p value
Device Succes (VARC-2)	104 (100%)	158 (97.5%)	0.158
30 Days			
All-cause death	7 (6.7%)	7 (4.3%)	0.152
Cardiovascular death	5 (4.8%)	6 (3.7%)	0.148
Major vascular complication	17 (16.3%)	13 (8.0%)	0.037
All stroke	6 (5.8%)	7 (4.3%)	0.594
Life-threatening bleeding	2 (1.9%)	3 (1.85%)	0.821
Permenant pacemaker implantation	15 (14.4%)	10 (6.2%)	0.025
Valve-related dysfunction requiring repeat procedure	0 (0%)	2 (1.2%)	0.158
Acute kidney injury	23 (22.1%)	23 (14.2%)	< 0.001
Early safety (VARC-2)	69 (66.3%)	133 (82.1%)	0.003

Interventional cardiology / Cover and structural heart diseases

OP-057

Single center experience in transcatheter aortic valve implantation for 7 years

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Background and Aim: Many patients have limited life expectancy or comorbid conditions that may limit the benefits of transcatheter aortic valve implantation (TAVI) and little is known about long-term durability of transcatheter heart valves.The purpose of this study was to investigate the long-term outcome after TAVI.
Methods: A total of 82 patients following succesful TAVI with balloon-expandable or self- expandable heart valve between 2012-2019 October were eveluated retrospectively. Preoperative demographic data, postoperative clinic results and long-term mortality of patients were analyzed.
Results: Mean age of patients was 76.9±10.3 and 35.3% were female. Mean logistic Euroscore was 15.1±6.5 (Table 1). TAVI was performed with transfemoral route in all patients except in one (transapical). In 17.6% (n=15) of cases we used self expandable heart valve. Mortality rate was 36% in average follow up time of 22.6±20.5 months (1-72 months). In-hospital mortality rate was 14.1% (n=12). The operation was accepted as unsuccesful due to aortic perforation in one patient, left ventricular perforation in one patient and valve embolization to the left ventricle in another one. These three patients died during emergency operation. The permanent pacemaker implantation was required in two patients in the early period (1< month) and three patients in the late period (1> month). The overall neurologic event rate was 6.8% in five patients (n=73). Among all re-hospitalizations (n=29) (39.7%) heart failure (n=8) (30.6%) was the most frequently reported. Median lifetime was 16 months (n=31) (95% CI 4,23-27,7) (Figure 1). Valve thrombosis or late valve embolization were not reported.
Conclusions: Our study demostrated favorably long-term outcomes after TAVI. No patients demostrated severe prosthetic regurgitation or stenosis. Comorbidities notably permanent atrial fibrillation (95% CI 0.09-0,61, p=0.003) and chronic heart failure (95% CI 0.95-2.66, p=0.02); Euroscore (95% CI 1.04-1.19, p=0.001) and STS score (95% CI 1.04-1.20, p=0.001) were the predictors of the long-term mortality.

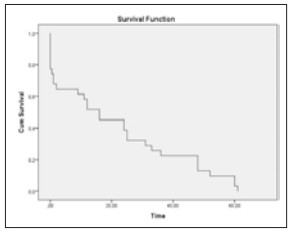

Figure 1. Kaplan Meier Curve.

Table 1. Baseline demographic and clinic characteristics of the study population

	n	%	Mean±SD
Age(years)	85		76.9±10.3
Average follow up(months)	73		22.6±20.5
Gender(Female)	30	35.3	
Euroscore	85		15.1±6.5
STS score	85		15.0±6.7
Creatinine	85		1.2±0.8
Chronic renal failure	26	30.6	
Hypertension	40	47.1	
Diabetes Mellitus	23	27.1	
Previous stroke	12	14.1	
Permanent atrial fibrillation	22	25.9	
Previous Percutaneous coronary intervention	12	14.1	
Previous Coronary Bypass Surgery	8	9.4	
Chronic Obstructive Pulmonary Disease	27	31.8	
Peripheral arterial disease	19	22.4	

Interventional cardiology / Carotid and peripheral vascular

OP-058

Mid and long term effect of acute kidney injury in patients undergoing endovascular abdominal aortic aneurysm repair

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Background and Aim: Acute kidney injury (AKI) is an important complication that increases mortality, morbidity, hospital stay and cost after cardiac invasive procedures. The incidence after endovascular aneurysm repair (EVAR) should remain uncertain in the medium and long term. Our aim is to investigate the relationship between AKI and all-cause mortality related to EVAR.
Methods: Between October 2010 and August 2019, we retrospectively analyzed 144 consecutive patients who underwent EVAR with infrarenal abdominal aortic aneurysm at our institution. AKI was defined as meeting the AKIN (Acute Kidney Injury Neywork) group criteria with the appearance of creatinine changes within the first 48 hours after the procedure. The patients were divided into two groups according to AKI development.
Results: AKI was detected in 27 (18.8%) of the patients. The AKI (+) group had higher body mass index (p=0.008) and aneurysm diameter (p=0.004). In addition, it was observed that the procedure time (p=0.001) and fluoroscopy time (p=0.002) were higher in this group. Although the length of hospital stay was longer in the AKI (+) group, it did not reach the statistical significance level (p=0.076). Two-year mortality was detected in 16 patients (11.1%) and five-year mortality in 39 patients (27.1%) after EVAR. Kaplan-Meier curves show that the incidence of two years of death in patients who develop AKI is significantly higher (Log-rang test, p=0.005), but this difference could not be carried over to the fifth year (Log-rang test, p=0.172).
Conclusions: When we look at the studies comparing EVAR with open surgical repair in terms of long-term results, we see that EVAR lost its success gained in the short term. On the other hand, it has been demonstrated by current studies that patients with aneurysm repair had worse survival data than the normal population with age and gender matching. Both results led to the need to find new markers to improve risk classification in this patient group. Few studies have evaluated the development of AKI after EVAR and to our knowledge only one study has been questioned about the prognostic aspect of AKI for patients undergoing EVAR. Saratzis et al found the development of AKI in 149 EVAR patients followed for an average of 33 months associated with the development of medium-term mortality. While the two-year results of our study confirm this relationship, when we look at the five-year results, we see that this relationship decreases. In conclusion, as well as AKI is a serious complication in patients undergoing EVAR, strict follow-up is essential for negative results that may develop especially in the early and mid-term.

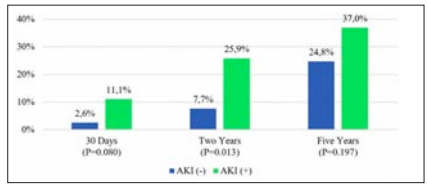

Figure 1. Mortality rates by period for those with and without AKI.

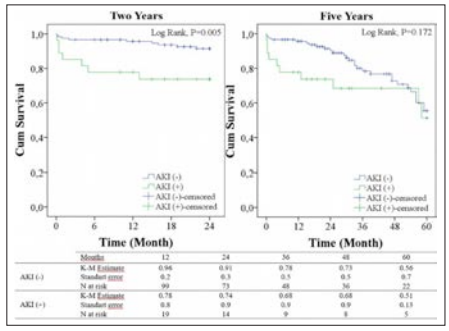

Figure 2. Kaplan–Meier curves for all-cause death of two and five years stratified by AKI.

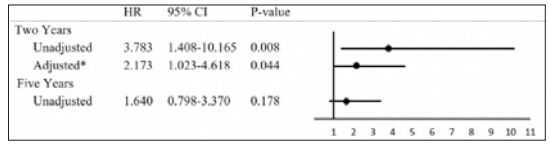

Figure 3. Univariable and multivariable Cox proportional hazard model of all-cause mortality. ** Adjusted for significant parameters in univariate analysis (chronic obstructive pulmonary disease, left ventricular ejection fraction and glucose). HR, hazard ratio; CI, confidence interval.*

Table 1. Baseline characteristics for those with and without AKI

	All Patients (N=144)	AKI (-) (n=117)	AKI (+) (n=27)	P-value
Age, years	69.3±8.2	69.4±8.1	69.1±9.0	0.884
Male, n (%)	132 (91.7%)	108 (92.3%)	24 (88.9%)	0.562
Body mass index, kg/m2	25.9±3.4	25.6±3.0	27.5±4.4	0.008
Current smoking, n (%)	45 (31.3%)	39 (33.3%)	6 (22.2%)	0.529
Diabetes mellitus, n (%)	34 (23.6%)	29 (24.8%)	5 (18.5%)	0.489
Hypertension, n (%)	87 (60.4%)	71 (60.7%)	16 (60.4%)	0.891
Coronary artery disease, n (%)	68 (47.2%)	56 (47.9%)	12 (44.4%)	0.748
Congestive heart failure, n (%)	22 (15.3%)	18 (15.4%)	4 (14.8%)	1.0
Hyperlipidemia, n (%)	46 (31.9%)	37 (31.6%)	9 (33.3%)	0.864
Cancer history, n (%)v	14 (9.7%)	10 (8.5%)	4 (14.8%)	0.299
Cerebrovascular disease, n (%)	7 (4.9%)	7 (6.0%)	0 (0.0%)	0.348
Atrial fibrillation, n (%)	15 (10.4%)	11 (9.4%)	4 (14.8%)	0.483
Peripheral artery disease, n (%)	11 (7.6%)	7 (6.0%)	4 (14.8%)	0.219
Chronic obstructive pulmonary disease, n (%)	24 (16.7%)	20 (17.1%)	4 (14.8%)	1.0
Chronic kidney disease, n (%)	22 (15.3%)	17 (14.5%)	5 (18.5%)	0.564
LV ejection fraction, %	55.3±9.4	55.4±9.5	55.0±9.2	0.844
Hemoglobin, g/dL	12.74±1.84	12.74±1.81	12.72±1.98	0.955
White Blood Cells, 10^3/uL	7.91±2.37	7.95±2.39	7.73±2.31	0.674
Platelet, 10^3/uL	226±80	225±81	229±75	0.819
Glucose, mg/dL	101 (91-114)	102 (92-116)	99 (87-106)	0.090
Creatinine, mg/dL	1.0 (0.8-1.2)	1.0 (0.8-1.1)	1.1 (0.9-1.5)	0.087
AAA diameter, mm	66.5±12.0	65.1±11.0	72.4±14.2	0.004
Symptomatic, n (%)	32 (22.2%)	27 (23.1%)	5 (18.5%)	0.608
Type of endograft Medtronic Endurant, n (%) TriVascular Ovation, n (%) Cook Zenith, n (%) Medtronic Talent, n (%) Vascutec Anaconda, n (%) Gore Exluder, n (%)	110 (76.4%) 14 (9.7%) 9 (6.0%) 7 (4.9%) 3 (2.1%) 1 (0.7%)	87 (74.4%) 13 (11.1%) 7 (6.9%) 6 (5.1%) 3 (2.6%) 1 (0.9%)	23 (85.2%) 1 (3.7%) 2 (4.3%) 1 (3.7%) 0 (0.0%) 0 (0.0%)	0.752
Contrast medium, mL	100 (70-150)	90 (70-140)	135 (68-179)	0.196
Operation time, min	147 (108 - 181)	138 (101-171)	186 (127-225)	0.001
Fluoroscopy time, min	28 (19-35)	26 (18-34)	35 (26-55)	0.002
ICU stay, days	2 (1-3)	2 (1-3)	2 (1-5)	0.133
Hospital stay, days	5 (4-7)	5 (4-7)	6 (4-11)	0.076

Data are presented as percentage, mean ± standard deviation or median (interquartile range). AKI, acute kidney injury; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft; LV, Left ventricular; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; AAA, abdominal aortic aneurysm; ICU, intensive care unit.

Interventional cardiology / Carotid and peripheral vascular

OP-059

The predictive value of CHADS2 score for subclinical cerebral ischemia after carotid artery stenting (from the prevent-cas trial)

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Background and Aim: Carotid artery stenting (CAS) is being increasingly used as an alternative revascularization procedure to carotid endarterectomy; however, subclinical ischemic brain lesions after CAS remain as a matter of concern. Hence, we aimed to assess the clinical utility of the CHADS2 score in predicting subclinical ischemic events in CAS.

Methods: We prospectively evaluated pre- and post-procedure diffusion-weighted imaging (DWI) examinations of 107 patients who underwent CAS in our center for carotid artery revascularization. The presence of new hyperintense lesion on DWI without any neurological findings was investigated. Patients were classified into two groups DWI (+) and DWI (-) groups. Patients risk status according to CHADS2 score was interpreted as high-risk (≥3 points) and low to intermediate risk (0-2 points).

Results: Among 107 patients, 28 patients (26.1%) had subclinical cerebral embolism. The DWI (+) group had a significantly higher number of patients with high-risk according to CHADS2 score (15/25 patients, 60%) compared to the DWI (-) group (13/82 patients, 15.9%) (p<0.001).

Conclusions: CHADS2 score may be a suitable for predicting the risk of subclinical embolism during the CAS procedure. Hence, this scoring system may have a clinical effect on the application of the best treatment options in patients with carotid artery stenosis.

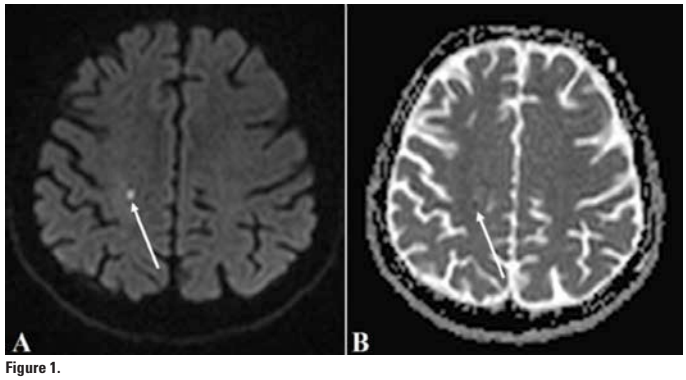

Figure 1.

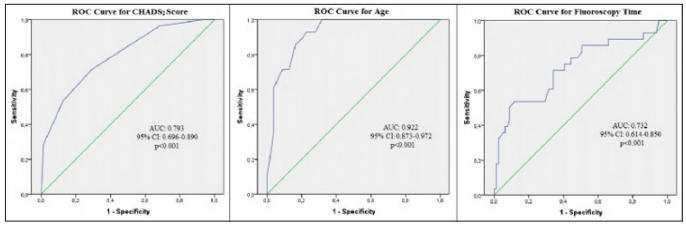

Figure 2.

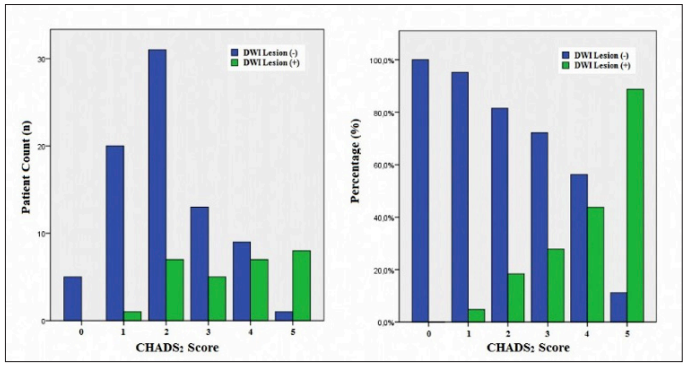

Figure 3.

Table 1. Comparison of the demographic clinical and angiographic charecteristics between patients with and without silent DWI lesion

Parameters	All Patients (N=107)	DWI Lesion (+) (n=28)	DWI Lesion (-) (n=79)	p value
Age, years	70.4±6.6	77.3±4.8	68.1±5.8	<0.001
Male, n (%)	77 (72)	23 (82.1)	54 (68.4)	0.163
Coronary artery disease, n (%)	55 (51.4)	15 (53.6)	40 (50.6)	0.789
$CHADS_2$ parameters				
Congestive heart failure, n (%)	5 (4.7)	2 (7.1)	3 (3.8)	0.392
Hypertension, n (%)	78 (72.9)	22 (78.6)	56 (70.9)	0.432
Age >75 years, n (%)	30 (28)	20 (71.4)	10 (12.7)	<0.001
Diabetes mellitus, n (%)	32 (29.9)	11 (39.3)	21 (26.6)	0.207
History of stroke, n (%)	52 (48.6)	19 (67.9)	33 (41.8)	0.018
$CHADS_2$ Score	2.4±1.3	3.5±1.3	2.1±1.1	<0.001
$CHADS_2$ Score ≥4, n (%)	25 (23.4)	15 (53.6)	10 (12.7)	<0.001
$CHADS_2$ Score ≥2, n (%)	81 (75.7)	27 (96.4)	54 (68.4)	0.003
Aortic arch type				
Type I, n (%)	78 (72.9)	12 (42.9)	66 (83.5)	
Type II, n (%)	13 (12.1)	6 (21.4)	7 (8.9)	
Type III, n (%)	12 (11.2)	9 (32.1)	3 (3.8)	<0.001
Type IV, n (%)	4 (3.7)	1 (3.6)	3 (3.8)	
Preprocedural Symptoms, n (%)	54 (50.5)	17 (60.7)	37 (46.8)	0.207
Contralateral stenosis, n (%)	29 (27.1)	7 (25)	22 (27.8)	0.771
Stenosis degree, %	85.7±8.8	83.6±7.4	86.4±9.1	0.139
Lesion length, mm	17.9±6.2	19.6±8.1	17.3±5.4	0.155
Fluoroscopy time, min	15.2±8.6	21.2±10.6	13.1±6.7	0.001
Predilatation, n (%)	23 (21.5)	5 (17.9)	18 (22.8)	0.585
Postdilatation, n (%)	70 (65.4)	15 (53.6)	55 (69.6)	0.125

Table 2. Regression analysis of potential predictor factors for silent embolism

Variables	Univariate analysis		Multivariable analysis	
	OR (95% CI)	p value	OR (95% CI)	p value
Age >75 years	1.566 (1.302-1.884)	<0.001	1.553 (1.245-1.939)	0.001
$CHADS_2$ Score ≥4	7.962 (2.942-21.549)	<0.001	5.584 (1.516-20.566)	0.010
Type III aortic arch	8.250 (2.959-24.663)	0.007	4.581 (1.341-17.295)	0.020
Long fluoroscopy time	1.115 (1.053-1.181)	<0.001	1.101 (1.037-1.169)	0.002
History of stroke	2.943 (1.184-7.314)	0.020	1.172 (0.331-4.157)	0.806

OR: Odds Ratio; CI: Confidence Interval.

Table 3. Comparison of the demographic clinical and angiographic charecteristics between patients with low and high CHADS2 score

Parameters	$CHADS_2$ score<3 (n=82)	$CHADS_2$ score≥3 (n=25)	p value
Age, years	69.0±6.2	75.1±5.8	<0.001
Male, n (%)	54 (65.9)	23 (92)	0.011
Coronary artery disease, n (%)	40 (48.8)	15 (60)	0.326
$CHADS_2$ parameters			
Congestive heart failure, n (%)	1 (1.2)	4 (16)	0.002
Hypertension, n (%)	55 (67.1)	23 (92)	0.056
Age >75 years, n (%)	16 (19.5)	14 (56)	<0.001
Diabetes mellitus, n (%)	17 (20.7)	15 (60)	<0.001
History of stroke, n (%)	29 (35.4)	23 (92)	<0.001
$CHADS_2$ Score	1.8±0.8	4.3±0.5	<0.001
Aortic arch type			
Type I, n (%)	65 (79.3)	13 (52)	
Type II, n (%)	10 (12.2)	3 (12)	
Type III, n (%)	5 (6.1)	7 (28)	0.008
Type IV, n (%)	2 (2.4)	2 (8)	
Preprocedural Symptoms, n (%)	31 (37.8)	23 (92)	<0.001
Contralateral stenosis, n (%)	25 (30.5)	4 (16)	0.154
Stenosis degree, %	85.3±9.2	86.9±9.2	0.423
Lesion length, mm	17.3±5.3	19.9±8.4	0.152
Fluoroscopy time, min	14.3±8.2	18.2±9.5	0.048
Predilatation, n (%)	17 (20.7)	6 (24)	0.728
Postdilatation, n (%)	55 (67.1)	15 (60)	0.515
DWI lesion, n (%)	13 (15.9)	15 (60)	<0.001

DWI: Diffusion-weighted imaging.

Interventional cardiology / Carotid and peripheral vascular

OP-061

The comparison of the treatment of arteriovenous hemodialysis fistulas with percutaneous transluminal angioplasty with plain balloon vs. drug-coated balloon

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Background and Aim: Treatment of the arteriovenous fistulas with percutaneous plain balloon angioplasty (PTA) and drug-coated balloon angioplasty (DCB) are safe and effective at short term follow up. The current study aims to investigate the comparison of the clinical outcomes in hospital and 12 months after the treatment of arteriovenous hemodialysis fistulas with percutaneous plain balloon angioplasty vs. drug-coated balloon angioplasty.

Methods: Forty-six patients who had hemodialysis fistula flow insufficiency, divided into two groups randomly. In this study, twenty-three patients (10 men; mean age 63.51±6.43 years), who underwent successful recanalization of brachial arteriovenous fistulae stenosis, were recruited for plain PTA group (15 brachio-cephalic, 8 ulno-basilic distal AVF) and twenty- three patients (12 men; mean age 65.51±9.21 years), who underwent successful recanalization of brachial arteriovenous fistulae stenosis with DCB angioplasty were recruited (18 brachio-cephalic, 5 ulno-basilic distal AVF) for DCB group from July 2016 to January 2018. For the PTA interventions, after achieving hemodynamic success (<30% residual stenosis) procedure was stopped. The follow-up intervals were in-hospital, 3, 6 and 12 months. Clinical endpoints analyzed, included the composite of all-cause death, hemodialysis insufficiency due to restenosis and acute thrombosis of fistula.

Results: Five consecutive patients were (all-cause) death (21.73 %) in plain PTA group and four consecutive patients were (all-cause) death (17.39%) in DCB group (p>0.05). Fistula dysfunction recurred in three patients (13.04%) in PTA group and fistula dysfunction recurred in two patients (8.69%) in DCB group (p>0.05). PTA was repeated in five patients (21.73%) in plain PTA group, PTA was repeated in two patients (8.69%) in DCB group (p=0.01) during the follow-up period. And one patient referred to redo surgery (4,76 %) at a median FU time of 340 days (p=0.01) in plain PTA group. No thrombosis was observed in both groups. One-year primary patency rates were 85.72%±3.24 in plain PTA group and 87.62±3.12 in DCB group (p>0.05). Under no access-induced distal ischemia occurred during follow-up.

Conclusions: Treatment of arteriovenous fistulas with PTA was as safe and effective in both plain balloon and DCB in the treatment of hemodialysis fistulae with acceptable restenosis rates at mid-term follow-up results. But in plain PTA group repeated PTA necessity will be significantly higher.

Interventional cardiology / Carotid and peripheral vascular

OP-062

The relationship between SYNTAX score and rest/post-exercise Ankle-Brachial index in patients with acute coronary syndrome

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Background and Aim: The aim of this study was to determine the relationship between the complexity of coronary artery disease (CAD), determined by the SYNTAX score, and the Ankle-Brachial Index (ABI) rest and post-exercise.

Methods: Patients who were followed up and treated in our center with the diagnosis of Acute Coronary Syndrome (ACS) between October 2018 and February 2019 were evaluated within the scope of the study. Study patients divided into two groups: SYNTAX Score >22 and ≤22. The correlation between the ABI measurements and the SYNTAX score were evaluated by pearson analysis. Logistic regression analysis was performed to determine the independent predictors to predict the complexity of coronary artery disease (CAD). The ROC curve was used to determine the ABI measurement cut off value.

Results: The mean age of 118 patients included in our study was 57.50±11.19 years and 26 (22%) patients were female. The SYNTAX score was >22 in 32 (27.11%) patients. In the group with SYNTAX Score >22, lower resting ABI (p<0.001) and post-exercise ABI (p
<0.001) were observed, whereas the higher SYNTAX II PCI (p=0.005) score was found. As a result of the ROC analysis: rest ABI cut off value was detected as 0,935 with a sensitivity of 75% and a specificity of 75% to predict SYNTAX score >22 [p<0.001, AUC (95% CI)=0.786 (0.697-0.875)] and post-exercise ABI cut off value was detected as 0.945% 80 with a sensitivity of 80% and a specificity of 81% to predict SYNTAX score >22 [p<0.001, AUC (95% CI)=0.836 (0.761-0.912)]. Diabetes mellitus [p=0.041, OR (95% CI)=1.901 (0.691-5.233)], resting ABI [p<0.001, OR (95% CI)=<0.001 (<0.001-0.025)] and post-exercise ABI [p<0.001, OR (95% CI)=<0.001 (<0.001-0.006)] were found to be independent predictors for the complexity of CAD.

Conclusions: The complexity of CAD in patients presenting to the hospital with the diagnosis of acute coronary syndrome can be determined by ABI measurements that can be easily applied on the bed head. We think that in this patient population ABI can be easily applied and can guide clinicians in determining the risk classification and treatment methods.

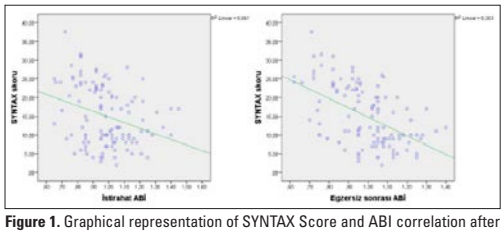

Figure 1. Graphical representation of SYNTAX Score and ABI correlation after Rest and Exercise.

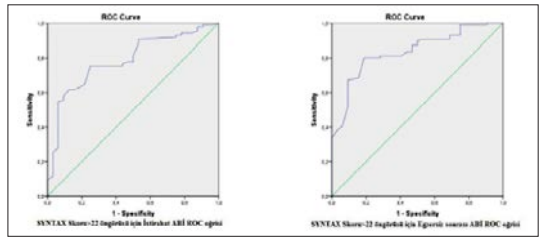

Figure 2. ROC curves of ABI for Rest and Exercise for SYNTAX Score> 22.

Table 1. Demographic characteristics and pre-procedure laboratory tests of all study patients and SYNTAX score groups

	Total (N=118)	SYNTAX Score≤22 (n=86)	SYNTAX Score>22 (n=32)	P value
Age, years	57.50±11.19	56.85±11.39	59.25±10.60	0.302
Male gender, n (%)	92 (78.0)	66 (76.7)	26 (81.3)	0.600
Smoking, n (%)	69 (58.5)	46 (53.5)	23 (71.9)	0.072
DM, n (%)	37 (31.4)	23 (26.7)	14 (43.8)	0.077
HT, n (%)	56 (47.5)	39 (45.3)	17 (53.1)	0.452
HL, n (%)	33 (28.0)	24 (27.9)	9 (28.1)	0.981
CAD, n (%)	32 (27.1)	19 (22.1)	13 (40.6)	0.044
COPD, n (%)	5 (4.2)	4 (4.7)	1 (3.1)	1.0
SBP	131.3±20.9	128.9±21.9	137.5±16.6	0.048
DBP	77.8±14.3	77.2±15.8	79.4±9.5	0.467
Heart rate, beats / min	76.0±12.7	74.8±12.5	79.2±12.9	0.097
Creatinine, mg / dL	0.89±0.22	0.90±0.22	0.85±0.21	0.224
GFR, ml / min / 1,73 m2	88.2±22.1	86.7±22.7	92.4±20.1	0.217
Hemoglobin, g / dL	13.51±1.93	13.55±1.89	13.38±2.05	0.672
Platelet, 10 ^ 3 / uL	261.5±80.0	258.5±76.5	269.4±89.5	0.514
MPV, fL	10.56±1.17	10.61±1.25	10.43±0.89	0.453
RDW,%	13.32±1.41	13.26±1.37	13.49±1.52	0.424
Neutrophil, 10 ^ 3 / uL	5.41±1.68	5.55±1.50	5.04±2.09	0.147
Lymphocyte, 10 ^ 3 / uL	2.64±1.04	2.67±0.92	2.54±1.31	0.552

SYNTAX: Synergy Between PCI With TAXUS and Cardiac Surgery, DM: Diabetes Mellitus, HT: Hypertension, HL: Hyperlipidemia, CAD: Coronary Artery Disease, COPD: Chronic Obstructive Pulmonary Disease, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, GFR: Glomerular Filtation Rate.

Table 2. Ankle-Brachial Index and risk score measurements of all study patients and SYNTAX score groups

	Total (N=118)	SYNTAX Score≤22 (n=86)	SYNTAX Score>22 (n=32)	P value
Rest ABI	0,98±0.17	1.02±0,16	0,87±0.12	<0.001
Post-exercise ABI	0.99±0.16	1.04±0.15	0.85±0.12	<0.001
TIMI score	3.73±1.26	3.72±1.29	3.75±1.19	0.902
GRACE score	98.77±24.16	97.61±23.57	101.84±25.80	0.401
KILLIP class	1.09±0.43	1.08±0.38	1.13±0.55	0.629
SYNTAX II PCI	23.2 (18.4-31.4)	22.0 (17.1-29.4)	28.4 (22.3-35.1)	0.005
SYNTAX II CABG	19.3 (13.8-25.8)	18.5 (12.0-26.3)	21.7 (14.9-25.1)	0.484

ABI: Ankle-Brakial Index, SYNTAX: Synergy Between PCI With TAXUS and Cardiac Surgery, GRACE: Global Registry of Acute Coronary Events, TIMI: Thrombolysis in Myocardial Infarction.

Table 3. Correlation of ABI with other variables after resting and exercise

	Rest ABI r	Rest ABI p	Exercise ABI r	Exercise ABI p
Age	-0.071	0.445	-0.136	0.143
Systolic Blood Pressure	-0.439	<0.001	-0.421	<0.001
Diastolic Blood Pressure	-0.202	0.028	-0.146	0.114
Heart rate	0.065	0.485	-0.053	0.571
creatinine	-0.001	0.994	0.056	0.550
GFR	0.008	0.932	0.032	0.731
Hemoglobin	-0.058	0.529	0.020	0.827
platelets	0.100	0.282	0.069	0.458
MPV	-0.053	0.572	-0.104	0.262
neutrophils	-0.063	0.498	-0.007	0.937
lymphocytes	0.051	0.586	0.066	0.478
TIMI score	-0.109	0.241	-0.118	0.207
GRACE score	-0.039	0.673	-0.111	0.234
KILLIP class	-0.054	0.563	-0.082	0.377
SYNTAX II PCI	-0.222	0.016	-0.322	<0.001
SYNTAX II CABG	-0.150	0.107	-.0.123	0.185
SYNTAX Score	-0.312	0.001	-0.513	<0.001

ABI: Ankle-Brakial İndex, SYNTAX: Synergy Between PCI With TAXUS and Cardiac Surgery, GRACE: Global Registry of Acute Coronary Events,TIMI: Thrombolysis in Myocardial Infarction

Table 4. ABI ROC analysis results after rest and exercise

	Area Under the Curve	95% Confidence Interval	p value	Estimation value	specificity	sensitivity
Rest ABI	0,786	0,697- 0,875	<0,001	0,935	%75	%75
Post-exercise ABI	0,836	0,761- 0,912	<0,001	0,945	%81	%80

ABI: Ankle-Brakial İndex.

Table 5. Univariate analysis results of all variables for SYNTAX Score >22

	Univariate Analysis OR (CI %95)	Univariate Analysis P
Age	1.020 (0.983-1.058)	0.300
Male sex	1.313 (0.474-3.638)	0.600
Cigaret	2.222 (0.922-5.354)	0.075
DM	2.130 (0.914-4.965)	0.080
HT	1.366 (0.605-3.082)	0.453
HL	1.011 (0.410-2.494)	0.981
CAD	2.413 (1.011-5.760)	0.047
COPD	0.661 (0.071-6.149)	0.716
SBP	1.020 (1.000-1.040)	0.052
DBP	1.010 (0.983-1.039)	0.465
Heart rate	1.027 (0.995-1.059)	0.102
creatinine	0.269 (0.032-2.245)	0.225
GFR	1.012 (0.993-1.033)	0.217
Rest ABI	0.001 (<0.001-0.022)	<0.001
Post-exercise ABI	<0.001 (<0.001-0.004)	<0.001
TIMI score	1.021 (0.738-1.411)	0.901
GRACE score	1.007 (0.991-1.024)	0.398
KILLIP class	1.237 (0.521-2.935)	0.630

ABI: Ankle-Brachial Index, SYNTAX: Synergy Between PCI With TAXUS and Cardiac Surgery, GRACE: Global Registry of Acute Coronary Events, TIMI: Thrombolysis in Myocardial Infarction, DM: Diabetes Mellitus, HT: Hypertension, HL: Hyperlipidemia, CAD: Coronary Arterial Disease, COPD: Chronic Obstructive Pulmonary Disease, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, GFR: Glomerular Filtation Rate.

Table 6. SYNTAX Score> 22 independent predictors found by multivariate regression analysis

	Rest ABI OR (95% CI)	Rest ABI p	Exercise ABI OR (95% CI)	Exercise ABI p
DM				
Cigaret	1.080 (0.052-24.815)	0.214	2.184 (0.739-6.455)	0.158
DM	1.901 (0.691-5.233)	0.041	1.951 (0.697-5.464)	0.203
CAD	2.687 (0.929-7.772)	0.068	2.298 (0.745-7.091)	0.148
SBP	1.004 (0.979-1.029)	0.765	1.001 (0.976-1.027)	0.929
Rest ABI	<0.001 (<0.001-0.025)	<0.001		
Post-exercise ABI				<0.001
			<0.001 (<0.001-0.006)	

ABI: Ankle-Brachial Index, DM: Diabetes Mellitus, CAD: Coronary Artery Disease, SBP: Systolic Blood Pressure

Interventional cardiology / Carotid and peripheral vascular

OP-063

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, vertebrobasiler 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 baloon 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 2019 and the data of 44 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 prevelant 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
Hypertension, n(%)	19 (59.4)
Diabetes Mellitus, n(%)	19 (59.4)
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)

Demographics and clinical characteristics of patients with subclavian artery stenosis PCI;percutaneous coronary intervention, CABG; coronary artery bypass graft, CAD; coronary artery disease, CVA; cerebrovascular accident, TIA; transient ischemic attack, CHF; congestive heart failure

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

Variable	n:32
Anterograde CFA (CFA; common femoral artery), n(%)	27 (84.4)
Dual access, n(%)	2 (6.3)
Guiding catheteter based system, n(%)	23 (71.9)
Procedural success, n(%)	29 (90.6)
Predilatation, n(%)	17 (53.1)
Baloon expandable stent, n(%)	27 (84.4)
Percentage of stenosis, %	87.9 ± 11.1
Total occlusion, n(%)	7 (21.9)
Baloon 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)
Serebral embolism, n(%)	0 (0)
Access site complication	1 (3.1)
Transfusion	2 (6.3)

Interventional cardiology / Carotid and peripheral vascular

OP-064

Relationship between monocyte / HDL ratio and carotid artery disease in patients with acute ischemic cerebrovascular disease

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Background and Aim: Macrophages play a major role in the pathogenesis of atherosclerotic plaque formation. It has been shown that the circulating monocyte count is predictive of new plaque development There is an established relation between Monocyte/HDL Ratio (MHR) and adverse outcomes in patients with cerebrovascular disease (CVD) and cardiovascular diseases. Recently, small studies have shown that MHR is independently associated with increased mortality in patients ischemic stroke. In this study, we aimed to determine whether higher MHR is related to the presence of carotid artery disease in patients with Acute Ischemic Cerebrovascular Disease.

Methods: A total of 209 patients diagnosed with acute ischemic stroke were retrospectively analyzed in this study. Acute ischemic cerebrovascular disease diagnosed patients are classified as group with and without carotid artery disease (group 1 and group 2). Doppler ultrasonography, Computed tomography and MRI were used for etiological research. MHR was calculated using data obtained from the complete blood. Patients were compared in terms of demographic features and hematological parameters. Systematic acute/chronic inflammatory/autoimmune or infectious diseases, chronic connective tissue diseases, hematological disorders, heart failure, acute coronary syndrome within the past three months and prior acute ischemic cerebrovascular disease were excluded

Results: The groups were composed of 62 with carotid artery disease and 147 patients without carotid artery disease. Median age was 75 (68-82) years. There was no significant difference between age, sex, total cholesterol level, triglyceride and smoking among the groups. There was a significant difference in both groups in terms of MHR, HDL, HbA1c, hypertension, chronic obstructive pulmonary disease, history of peripheral artery disease and dyslipidemia. In multivariate analysis, MHR independently associated with presence of carotid artery disease. In this study, a MHR level of >18.1, measured upon admission, had 82% sensitivity and 69% specificity in demonstrating the presence of carotid artery disease in patients with acute ischemic stroke (AUC:0.823 %95 CI 0.768-0.877 p<0.001).

Conclusions: In this study, we showed that MHR, a novel biomarker, was associated with presence of carotid artery disease in patients with CVD and higher MHR may predicted carotid artery disease in patients CVD.

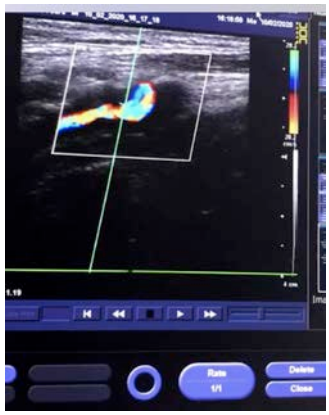

Figure 1. Doppler ultrasonography of carotid artery.

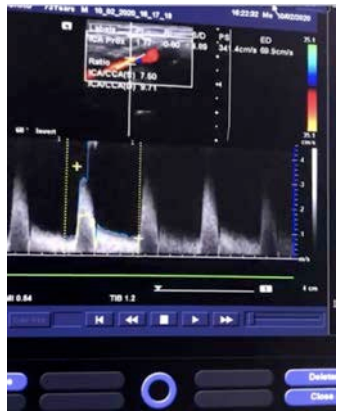

Figure 2. Stenosis evaluation of the carotid artery.

Table 1. Demographic features of the population study according to gender (No:38)

Features	Male No 26 (%)	Female No 12(%)	P vlaue
Age	72.23±10.4	69±8.9	0.35
Height	168.2±5.02	160.5±4.42	<0.001
Weight	79.54±4.32	75.75±5.59	0.028
DM	13	8	0.337
HT	23	10	0.643
CAD	17	4	0.065
SVO	9	4	1.00
LVEF	46.53±13.4	47.08±13.4	0.751
Sinus rhythm	26 (100)	12 (100)	-
SYSTOLİC	129.08±7.96	127.5±8.66	0.867
ASYMTOMATİC	7 (26.9)	5 (41.6)	0.245
TIA	13 (50)	7 (58.3)	0.065
STROKE	3 (11.5)	0 (0)	0.152

Table 2. Procedural features of carotid stent procedure

Feature	Male N 26 (%)	Female N 12 (%)	P value
Left ICA stenosis	12 (46)	5 (41.7)	0.73
Right ICA stenosis	13 (50)	12 (100)	0.73
Catheter 8 French	26 (100)	12 (100)	-
Distal embolic protection device	26 (100)	12 (100)	-
Predilataiton	6 (23.1)	2 (16.7)	1.00
Postdilataiton	10 (38)	4(33.3)	0.654
Open cell stent	26 (100)	12 (100)	-
Successful stent implantation	26 (100)	12 (100)	-
Hypotension	8 (30.7)	1 (8.3)	0.034
Bradycardia	4 (15.4)	1 (8.3)	0.065

Table 3. Time fashion changes of carotid doppler ultrasonography data pre and post carotid artery stenting (No:38)

	Basal		Post-stent procedure 1 month		1 year		
Velocity	Male	Female	Male	Female	Male	Female	P (Time;Time-gender Interaction; only gender)
Left CCA PSV	72.6±27.45	81.17±17.42	79.88±30.47	92.67±27.88	56.4±40.36	93±85.36	0.294 0.176 0.087
Left CCA EDV	20.24±14.25	19.5±6.8	18.52±6.79	23.17±13.2	13.8±9.36	25.5±34.27	0.924 0.154 0.174
Left ICA PSV	187.48±92.68	194.67±98.33	96.56±43.74	97.58±27.74	70.04±58.22	86.58±58.62	<0.001 0.777 0.627
Left ICA EDV	39.2±16.14	35.33±12.04	24.76±11.42	22.5±8.78	18.04±13.3	16.42±10.94	<0.001 0.740 0.421
Right CCA PSV	79.08±24.04	76.67±13.2	75.19±20.87	74.83±14.61	57.08±35.4	66.25±35.74	0.009 0.529 0.752
Right CCA EDV	18.73±6.71	21.5±4.92	19.08±10.28	18.17±4.72	13.12±8.44	16.08±9.54	0.018 0.539 0.350
Right ICA PSV	180.58±91.45	182.92±76.97	84.65±37.27	108.42±46.5	74.69±48.39	97.58±64.69	<0.001 0.816 0.227
Right ICA EDV	35.23±17.46	38.08±10.88	27.77±20.22	26.83±7.86	19.04±12.92	24.08±14.15	0.001 0.727 0.478

Interventional cardiology / Carotid and peripheral vascular

OP-065

Duplex ultrasound surveillance after carotid artery stent procedure: A time fashion assessment of duplex velocity data

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Background and Aim: Carotid artery percutaneous angioplasty has been reported as an alternative to carotid endarterectomy. Carotid artery duplex ultrasonography is the primary tool for surveillance after stenting procedure. However, the course of sonographic velocities after caortid stenting angioplasty is unclear. We aimed to accurately assess the change of sonographic velocities after successful carotid artery stent procedure.

Methods: Thirty-eight patients undergoing a successful carotid artery stent procedure between January and June 2019 were enrolled in the study. A retrospective evaluation of the time fashion change of duplex carotid sonographic velocities was performed. All doppler spectra were obtained by an experienced sonographer in the noninvasive vascular laboratory of our clinic. Accuson X300 (Siemens) was used for duplex evaluation of the carotid arteries. All doppler spectra were obtained using a doppler sample volume of 1 to 1.5 mL and a doppler angle of 60* or less. The ultrasound evaluation was performed on admission, one month after the stent procedure and 1 year after during follow up. All the doppler recordings were done by the same sonographer. All these recordings were evaluated offline one year after. Patients demographic data together with clinical events during hospitalization and follow-up were recorded.All the data were analyzed in the SPSS 25 program where continuous and categorical variables were shown as mean ± standard deviation and frequency with proportion respectively. For time change assessment of variables, variance analysis was performed. Statistical significance level was accepted as P level <0.05 for all hypotheses.

Results: There were 26 male and 12 female patients who had underwent a carotid stent angioplasty procedure. The average age was similar in Both genders (72.23±10.4 vs 69±8.9 p=0.35). All patients were homogenous in terms of comorbidities (Table 1). Almost half of patients (both male and female) presented with a transient ischemic attack (male: 11 patients; female 7 patients). Only 3 male patients presented with minor stroke not necessitating intracranial intervention. The others were asymtomatic. Male patients had more profound atheterosclerosis in both left and right internal carotid artery (ICA) (left: 12 vs 5; right 13 vs 7 p=0.73). All had a distal embolic protection device (Spider FX) and open cell tapered slelf expandable stent (Protege Ev 3) was successfully implanted (Table 2). The sonographic peak systolic velocities of the stented ICA were significantly decreased and enter the normal range early after the stent procedure and this continoued by one year after (Table 3). This significant change occurred in both genders without any difference between.

Conclusions: Duplex peak systolic velocities of the stented artery decrease turning to normal ranges early after the stent procedure. This decrease continoues at 1 year follow up. Change of this course should prompt for investigation of possible restenosis.

Interventional cardiology / Carotid and peripheral vascular

OP-066

Predictors of major adverse cardiovascular and cerebrovascular events in carotid artery stenosis patients after stent implantation

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Background and Aim: We hypothesized that long-term morbidity and mortality can be identified by the presence of comorbidities in patients with carotid artery stenosis. In our study we aimed to investigate the relationship between clinical characteristics, laboratory findings and long-term prognosis in carotid artery stenosis patients after stent implantation

Methods: We prospectively enrolled 212 patients whom underwent successfully carotid artery stenting (CAS) in Kartal Kosuyolu Training and Research Hospital. CAS was performed in symptomatic patients with >60% stenosis and in asymptomatic patients with >80% stenosis of extra-cranial carotid artery. The degree of stenosis was determined by carotid duplex ultrasound imaging and confirmed by carotid angiography according current guidelines. Symptoms were defined by an ipsilateral cerebral or ocular minor or major ischemic event within the past 6 months.

Results: Mean age of study population was 67.4±7.9 years and 158 patients (74.5%) were male. In the follow-up period (mean 3 years) 18 patients had MI, 18 patients had major stroke, 23 patients had transient ischemic attack. Twenty-one patients (9.9%) died from cerebral-cardiovascular causes. All major adverse cardiac and cerebrovascular events (MACCE) was found in 64 patients (30.2%). Multivariate analysis revealed that age (HR: 1.09, 95% CI: 1.02-1.17, p=0.05), heart failure (HR: 3.78, 95% CI: 1.48-9.62, p=0.005), creatinine (HR: 3.54, 95% CI: 1.16-10.82, p=0.026) and NLR (Neutophil/ lymphocyte rario) (HR:2.88, 95% CI: 1.90-4.36, p<0.0001) were independent predictors of the MACCE.

Conclusions: Although, the short-term risk of patients undergoing CAS dominated by lesion-related factors, pre-existing comorbidities may be even more important for the long-term event. Age, heart failure, creatinine and NLR were found as the most important risk factors of MACCE.

Table 1. Predictors of Major Cardiovascular-Cerebrovascular Adverse (MACCE)s Using Logistic Regression Analyses

Variables	Univariate HR	% 95 CI	p value	Multivariate HR	% 95 CI	p value
Age	1.12	1.07-1.18	<0.001	1.09	1.02-1.17	0.05
Creatinine	1.74	4.70-29.30	<0.001	3.54	1.16-10.82	0.026
Hs-CRP	1.32	1.05-1.66	0.014	0.96	0.70-1.30	0,792
NLR	2.93	2.07-4.16	<0.001	2.88	1.90-4.36	<0,001
Heart Failure	4.17	2.13-8.18	<0.001	3.78	1.48-9.62	0,005
Inadequate glycemic control	3.80	1.88-7.68	<0.001	2.37	0.86-6.61	0.094
Prior coronary stent	1.85	0.99-3.46	0.052			
Contrlateral carotid stent	2.86	1.14-7.13	0.024	2.75	0.67-11.17	0.157

Interventional cardiology / Carotid and peripheral vascular

OP-067

Decreased psoas muscle area may predict acute kidney injury after lower extremity peripheral arterial endovascular interventions

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Background and Aim: Endovascular interventions are of great importance in the treatment of PAD with increasing process success. Acute kidney injury (AKI) after endovascular intervention is one of the important causes of morbidity and mortality. AKI, which develops after the procedure, is observed more frequently, especially in the presence of concomitant diabetes mellitus, chronic kidney disease (CKD), and frailty. The psoas muscle area (PMA) is a simple and validated measurement for frailty. Previous studies showed that the PMA, an indicator of central sarcopenia, is a useful measurement for assessing the frailty of patients with PAD, and further decreased PMA has been shown to predict poor patient outcomes, prolonged length of hospital stays and mortality in PAD patients. Based on these studies, we aimed to investigate the relationship between the psoas muscle area measured by CT and acute kidney injury in patients who underwent endovascular intervention due to peripheral artery disease, assuming that the psoas muscle area, which is an indicator of frailty, can be a useful marker in predicting patients who may develop acute kidney injury after the procedure.

Methods: We retrospectively reviewed the consecutive patients who underwent angiography for lower extremity PAD and objective evidence of arterial occlusive disease based on angiography. Of these patients, those who had an abdominal CT scan for various indications within six months before the procedure, or up to 1 month after the procedure, were included in the study. AKI was defined, according to the KDIGO criteria (Kidney Disease: Improving Global Outcomes (KDIGO), as a creatinine increase of more than 0.3 mg/dL or a 20% reduction in eGFR during the hospital stay.

Results: 209 patients were included in the study. The patients were divided into two groups according to the KDIGO criteria (Kidney Disease: Improving Global Outcomes (KDIGO), as 167 patients without AKI and 42 patients with AKI. The AKI group were older age, had a higher percentage of hypertension history, a higher percentage of CKD history, lower glomerular filtration rate levels, lower high-density lipoprotein levels, lower hemoglobin levels, lower left ventricular ejection fraction (LVEF), more complex lesions according to TASC-II classification and lower PMA levels compared to the non-AKI group (p<0.05 for all parameters). Based on the results of the multivariate logistic regression test, advanced age, CKD history, LVEF, length of the lesion and psoas muscle area were determined as independent predictive parameters for AKI in PAD.

Conclusions: AKI is the most important and most frequent renal complication after endovascular peripheral arterial treatment, contributing to increasing rates of morbidity and mortality. Knowing the high risk of acute kidney injury groups may play an important role in improving renal outcomes in patients with PAD. In this context, PMA may help identify a higher risk group for AKI in PAD patients who underwent endovascular interventions.

Interventional cardiology / Carotid and peripheral vascular

OP-068

Factors affecting wound healing in peripheral artery patients under knee percutaneous intervention

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Background and Aim: The aim of our study was to investigate the factors affecting wound healing in patients who developed AEPAH because of risk factors and subsequently underwent endovascular intervention

Methods: The study was performed with 200 patients in Kocaeli University Cardiology Department between January 2015 and August 2019 because of stenotic and / or occlusive lesions in the infrapopliteal arteries endovascular revascularization with conventional / drug-eluting balloon dilatation. Patients records were retrospectively reviewed. Burger's disease patients were excluded. Therefore, eight patients with Burger's disease were excluded from the study and 192 patients were included in the study. Angiography and angioplasty images of patients who underwent PACS archive system were re-evaluated by stenosis and / or occlusion of the vessels, which vessels were affected, how many vessels were affected, which vessels were treated and the success of angioplasty procedure was recorded. Wound healing was evaluated by accessing the polyclinic control records from the nucleus archive system. Wound assessment was performed after the procedure by telephone interview with patients with incomplete information. Wound healing was evaluated at least 3 months after peripheral intervention. Patients were divided into the two groups according to wound healing after the procedure. Patients who underwent early amputation were included in the non-wound healing group.

Results: When the demographic and procedure characteristics of the patients were compared in terms of wound healing; in the group without wound healing, male gender (52.2% vs 70% p=0.01), chronic kidney disease (16.3% versus 37% p=0.01), smoking exposure (32.6% versus 50% p=0.01), absence of tibialis anterior flow after the procedure was statistically higher. Multivariate regression analysis was performed for p<0.01 univariate regression analysis. In multivariate logistic regression analysis, presence of cronic kidney disease, to be in the Rutherford class 6 and the absence of tibialis anterior flow after the procedure were found as negative independent predictors of wound healing. There was no statistically significant difference in wound healing in other parameters.

Conclusions: When the factors affecting wound healing after peripheral endovascular intervention were examined, we found that in patients with chronic kidney disease, clinical classification of Rutherford 6 before the procedure, and lack of flow in the tibialis anterior after PTA procedure were the negative predictive factors for wound healing.

Table 1. Target lesion and procedure features

	Yara iyileşmesi olan	Yara iyileşmesi olmayan	P Değeri
Lezyonun yeri			
Popliteal, n (%)	49 (53,3)	56 (56)	0,70
Tibialis anterior, n (%)	80 (87)	91 (91)	0,37
Tibialis posterior, n (%)	84 (91,3)	89 (89)	0,59
Peroneal, n (%)	68 (73,9)	80 (80)	0,31
Distal, n (%)	78 (84,8)	86 (86)	0,81
İnfrapopliteal tüm bölgede total oklüzyon	38 (41,3)	40 (40)	0,85
Trombüs aspirayonu, n (%)	5 (5,4)	4 (4)	0,63
TASC C, n (%)	7 (7,6)	3 (3)	0,15
TASC D, n (%)	85 (92,4)	97 (97)	0,15
Endovasküler işlem tipi			
Aterektomi+anjiyoplasti			
Popliteal, n (%)	23 (25)	34 (34)	0,17
Tibialis anterior, n (%)	14 (15,2)	26 (26)	0,06
Tibialis posterior, n (%)	9 (9,8)	23 (23)	0,01
Peroneal, n (%)	19(20,7)	13(13)	0,15
Distal, n (%)	3 (3,3)	3(3)	0,06
Anjiyoplasti			
Popliteal, n (%)	54 (58,7)	68(68)	0,76
Tibialis anterior, n (%)	55 (59,8)	58 (58)	0,80
Tibialis posterior, n (%)	38 (41,3)	51 (51)	0,17
Peroneal, n (%)	48 (52,2)	41 (41)	0,12
Distal, n (%)	17 (18,5)	12 (12)	0,21

Table 2. Multivariate analysis table of factors affecting wound healing after endovascular intervention

	OR	CI 95%	P
Erkek			
HL			
DM			
HT			
KAH			
KBH	0,36	0,13-0,61	0,02
SİGARA MARUZİYETİ			
Rutherford 5			
Rutherford 6	0,32	0,14-0,75	0,009
Anjiyoplasti			
Popliteal			
Anterior			
Posterior			
Peroneal			
Distal			
Aterektomi+anjiyoplasti			
Popliteal			
Anterior			
Posterior			
Peroneal			
Distal			
İşlem sonrası akım			
olmaması			
Popliteal			
Anterior	0,33	0,16-0,66	0,02
Posterior			
Peroneal			
Distal			
TROMBUS ASPİRASYONU			
ATEREKTOMİ			

Interventional cardiology / Coronary

OP-069

Comparison of clopidogrel and ticagrelor in the development of left ventricular apical thrombus in patients with acute anterior myocardial infarction survivors

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Background and Aim: Ticagrelor has been acknowledged as an orally active antagonist of P2Y12-adenosine diphosphate (ADP) receptor, so it has the advantage of a more rapid onset of action and can more completely inhibit the platelet aggregation response to ADP than clopidogrel. In the PLATO study, ticagrelor was superior to clopidogrel in reducing cardiovascular events among patients with acute coronary syndrome (ACS),however, contradictory results are shown in later studies. We investigated whether ticagrelor and clopidogrel treatment have superiority over each other in the development of left ventricular thrombus (LVT) in patients with acute anterior myocardial infarction (AAMI) survivors.

Methods: A total of 955 patients who were admitted to our hospital with acute anterior ST-segment elevation myocardial infarction (STEMI) and who underwent primary percutaneous coronary intervention (pPCI) were prospectively evaluated. Each patient received standard pharmacologic treatment according to current treatment guidelines; We use an initial intravenous unfractionated heparin (UFH) bolus of 70-100 IU/kg up to a maximum of 10,000 IU and dual antiplatelet therapy (DAPT) consisting of aspirin (a loading dose of 300 mg followed by 100 mg/day) and either clopidogrel (a loading dose of 600 mg followed by 75 mg/day) or ticagrelor (a loading dose of 180 mg followed by 90 mg/day). All patients underwent screening transthoracic echocardiography (TTE) within 24h of admission. Repeat TTE was performed before discharge (within 3-5 days). Besides, since most thrombus cases were reported within the first month, patients were re-examined on the 30th day after discharge. If the presence of a diagnosis of LVT, triple anticoagulation therapy administered according to current guidelines which included aspirin, clopidogrel, and warfarin (with target international normalized ratio (INR) of 2-2.5) for 1 month. During months 2 and 3,warfarin and clopidogrel were continued. The study protocol was approved by the local ethical committee and informed consent was obtained from each participant before enrolment.

Results: 256 (26.8%) and 699 (73.2%) patients were taking ticagrelor and clopidogrel, respectively. During the first month after the index event, LVT was observed in 112 (11.7%) patients. Among those 112 patients, 77 of them (68.8%) were taking clopidogrel and 35 of them (31.3%) were taking ticagrelor. No significant difference in the rates of LVT was found between groups before discharge and on the 30th day of control. Before discharge, LVT had occurred 3.5% of patients receiving ticagrelor as compared with 3.4% of those receiving clopidogrel (p=0.951). In the period from discharge to first-month control, LVT had occurred 10.2% of patients receiving ticagrelor as compared with 7.6% of those receiving clopidogrel (p=0.201).

Conclusions: In this prospective study of patients with AAMI who underwent pPCI, the use of ticagrelor was not associated with a statistically significant reduction in LVT development versus clopidogrel.

Table 1. Characteristics of study populations

Demographics	All patients (n=955)	Clopidogrel (n=699)	Ticagrelor (n=256)	p
Age (years)	60 (55-63)	60 (55-64)	60 (53-63)	0.084
Male gender, n (%)	736 (77.1%)	537 (76.8%)	199 (77.7%)	0.767
Diabetes mellitus, n (%)	282 (29.5%)	211 (30.2%)	46 (36.5%)	0.462
Smoking, n (%)	339 (35.5%)	248 (35.5%)	91 (35.5%)	0.985
Hypertension*, n (%)	366 (38.3%)	275 (39.3%)	91 (35.5%)	0.285
Dyslipidemia#, n (%)	731 (76.5%)	521 (74.5%)	210 (82%)	0.017
Family history of CAD, n (%)	264 (27.6%)	205 (29.3%)	59 (23%)	0.055
Hemoglobin (mg/dl)	13.5 (12.6-14.4)	13.5 (12.6-14.3)	13.6 (12.6-14.5)	0.368
Hematocrit (%)	38 (35-41)	38 (35-41)	38 (36-41)	0.298
Neutrophil (103/µl)	7.2 (5.08-9.31)	7.15 (5.09-9.30)	7.41 (5.05-10)	0.234
Serum creatinine (mg/dl)	0.99 (0.74-1.23)	0.99 (0.73-1.23)	0.98 (0.76-1.22)	0.805
Total protein (g/l)	73.4 (68.6-78.1)	73.3 (68.6-77.9)	73.6 (68.5-78.9)	0.569
Albumin (g/dl)	4.0 (3.7-4.3)	4.0 (3.7-4.2)	4.0 (3.7-4.3)	0.832
Total cholesterol (mg/dl)	199 (165-222)	199 (166-222)	198 (164-227)	0.995
LDL-C (mg/dl)	139 (101-165)	139 (101-163)	140 (100-165)	0.440
HDL-C (mg/dl)	39 (36-42)	39 (36-42)	39 (36-42)	0.666
CRP (mg/dl)	0.52 (0.34-0.67)	0.50 (0.34-0.68)	0.54 (0.36-0.67)	0.466
Peak CKMB (IU/l)	229 (200-254)	227 (201-252)	229 (196-260)	0.611
Angiographic characteristics				
Culprit Proximal LAD, n (%)	307 (32.1%)	229 (32.8%)	78 (30.5%)	0.502
Stent length, mm	24 (20-28)	24 (20-28)	24 (20-28)	0.967
Number of diseased arteries >1, n (%)	290 (30.4%)	208 (29.8%)	82 (32%)	0.498
Post procedural TIMI flow <3, n (%)	141 (14.8%)	93 (13.3%)	48 (18.8%)	0.036
No-reflow, n (%)	77 (8.1%)	52 (7.4%)	25 (9.8%)	0.242
GP IIb/IIIa inhibitors treatment, n (%)	148 (15.5%)	105 (15%)	43 (16.8%)	0.502
Echocardiography				
LVEF on admission (%)	38 (35-41)	38 (33-43)	38 (33-44)	0.942
LVA, n (%)	38 (4%)	26 (3.7%)	12 (4.7%)	0.498
LVT, n (%)	112 (11.7%)	77 (11%)	35 (13.7%)	0.258
- Before hospital discharge	33 (3.5%)	24 (3.4%)	9 (3.5%)	0.951
- 30th day control	79 (8.3)	53 (7.6%)	26 (10.2%)	0.201

CAD: Coronary artery disease; CAR: C-reactive protein to albumin ratio; CKMB: Creatine kinase myocardial band; CRP: C-reactive protein; GP: Glycoprotein; HDL-C: High-density lipoprotein cholesterol; LAD: Left anterior descending; LDL-C: Low-density lipoprotein cholesterol; LVA: left ventricular aneurysm; LVEF: Left ventricular ejection fraction; LVT: left ventricular thrombus; TIMI: Trombolysis in myocardial infarction; WBC: White blood cell. Data are presented as mean ± standard deviation for normally distributed continuous data, as median and inter-quartile ranges for skew-distributed continuous data and percentage (%) for categorical variables.

Interventional cardiology / Coronary

OP-070

Comparison of two different bioresorbable vascular scaffolds: Real world use of everolimus- versus novolimus- eluting BVS

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Background and Aim: Majority of clinical trials have compared everolimus-eluting bioresorbable scaffold (eBVS) and metallic stents showing elevated risk of adverse outcomes for eBVS (Absorb, Abbott Vascular, Santa Clara, CA, USA). It remains unclear as to whether, novolimus-eluting bioresorbable vascular scaffold (nBVS) could be better than eBVS. This study sought to evaluate the long-term clinical outcomes between eBVS and nBVS.

Methods: A total of 140 patients with nBVS (n=202) and 98 patients with eBVS (n=135) were found to be eligible for matching. Of these, 98 patients treated with 135 eBVS were matched 1:1 with 98 patients treated with 136 nBVS. The primary outcome was the 3-year rate of major adverse cardiovascular events (MACE), defined as the composite of cardiac death, target vessel myocardial infarction (TV-MI), and target-lesion revascularization (TLR).

Results: Baseline characteristics, clinical presentation, and lesion characteristics were comparable in both groups. The proportions of patients with stable angina pectoris (83% vs. 80%), acute coronary syndrome (17% vs. 20%) were comparable in the two matched groups. There were no differences in terms of procedural success between the two groups (98% vs. 99% in eBVS vs. nBVS groups, respectively). In nBVS group, one patient suffered side branch occlusion, whereas in eBVS there was one scaffold rupture during post-dilatation and second patient had TIMI 1 flow after overlapping of two scaffolds. Mean follow-up time for patients implanted with eBRS was 52.9±13.9 months and 36.4±8 months for nBVS treated patients (p<0.001). The 3-year MACE rate was higher in eBVS group (17.3% vs. 6.1%; p log-rank=0.02). The occurrence of TLR

(16.3% vs. 5.1%; p log-rank=0.02) and TV-MI (8.2% vs. 0%; p log-rank=0.004) was also higher in eBVS group except for cardiac deaths (1% vs.2%; p log-rank=0.98, eBVS vs. nBVS, respectively). Of note,definite device thrombosis rate was markedly increased in the eBVS group (5.1% vs. 0%; p log-rank=0.03).
Conclusions: The present study is the first to report mid-term comparative analysis of nBVS and eBVS outcomes based on propensity score matching. Three -year event risk was found to be lower for nBVS compared with eBVS.

Interventional cardiology / Coronary

OP-071

ECG criteria for the prediction of infarct-related artery and impact of coronary dominance on ECG in patients with inferior ST-elevation myocardial infarction

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Background and Aim: Previous studies have reported that the distribution of ST elevation (STE) may predict the infarct related artery (IRA). However, the impact of coronary dominance on ECG is not clearly demonstrated. Our primary aim was to investigate the impact of coronary dominance on surface ECG. Our secondary aim was to investigate the ECG predictors of IRA according to the 4th definition of myocardial infarction guidelines.
Methods: We retrospectively included patients with inferior STEMI. Previously defined ECG predictors of IRA were tested; higher STE in DIII>DII or presence of ST depression in aVL and/or DI presumes right coronary artery (RCA); STE in DII>DIII presumes left circumflex artery (LCX).
Results: Our study encompasses 192 patients. The culprit artery was RCA in 151 patients and LCX in 41 patients. The sensitivity and specificity of STE DIII>DII for prediction of RCA were 84% and 35% respectively. In comparison,the sensitivity of STE DII>DIII for prediction of LCX was lower, whereas specificity was considerably higher (48% and 65%, respectively). However, the best sensitivity was observed with a STE DIII>DII, which strongly predicts the coronary dominance with a sensitivity of 97% and a specificity of 27%. If the IRA is RCA the magnitude of ST-elevation was significantly higher when the reference point was QRS onset (p=0.021) compared to the prior guideline recommendations. However, this significance was not observed in patients with LCX occlusion (p=0.212).
Conclusions: In this study, we have demonstrated that ECG predictors of IRA are significantly related to coronary dominance. Moreover, new ECG criteria increased the diagnostic ability of ECG when RCA is the culprit artery but did not differ when the LCX is the IRA.

Table 1. Sensitivity and Specificity analysis of ECG predictors

Predictor	Presumed IRA	Sensitivity	Specificity	PPV	NPV	C- statistics
STE DIII>DII	RCA	%84	%35	%76	%46	0.59 (0.50-0.69)
STE DII>DIII	Cx	%48	%64	%56	%66	0.48 (0.39-0.58)
STE DIII>DII	RCA Coronary dominance	%97	%27	%77	%78	0.62 (0.52-0.71)

IRA: infarct related artery, PPV: positive predictive value, NPV: negative predictive value, STE: ST segment elevation, RCA: right coronary artery, Cx: circumflex artery.

Interventional cardiology / Coronary

OP-072

The compatibility of SYNTAX score calculations, assessed by invasive coronary angiography and computed tomographic angiography

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Background and Aim: Syntax score has become one of the routine methods in terms of evaluating the complexity of coronary artery disease in the realm of interventional cardiology practice. According to European and American guidelines reperfusion strategies are determined in respect to syntax score. There are few studies on the calculation of syntax score with computed tomographic coronary angiography (CT angiography). Moreover, the consistency between the scores calculated with CT angiography and conventional coronary angiography invasive coronary angiography are not well established. In this study, we aimed to assess the correlation of syntax scores calculated with PCA in the syntax score calculations between invasive coronary angiography and CT angiography.
Methods: Those patients who underwent both CT angiography and invasive coronary angiography between May 2018 and May 2020 were enrolled to our study. Cardiac CT angiography was performed with a 640-slice computed tomography. The CT is equipped with a gantry rotation of 0.275 seconds, covering 16 cm area in a single rotation. Which is able to evaluate slices of 0.5 mm thickness. During the injection the target heart rate was equal to 85 beats/min or lower than 85 beats/min. Beta blocker agents were administered for those patients with higher heart rates before the procedure.
Both invasive coronary angiography and CT angiography syntax scores were calculated by following the algorithms on wwww.syntaxscore.com website. The relationship between invasive coronary angiography and CT angiography, which was calculated by syntax scores, was primarily analyzed by loess algeration and spearman correlation tests. Bland-Altman analysis was performed to assess the agreement between two methods in terms of calculating syntax score.
Results: One hundred thirty-nine patients with coronary artery disease detected by both CT and invasive coronary angiography were included to the study. The mean of syntax score measured according to invasive angiography was 17.9, the median value was 18 (9.5-24), whereas mean syntax score measured by evaluation of the CT angiography images was 17.2, the median was 16 (11-23). Significant correlation was demonstrated between syntax scores both calculated according to CT and invasive coronary angiography images (spearman correlation 0.78, p<0.001). According to bland-altman analysis, the mean bias was 0.7 (95% CI; -0.38,1.73), and 95% compliance limits were -11, 13.
Conclusions: Syntax score measured by CT angiography was lower than the syntax score measured by invasive coronary angiography. In addition, moderate level of bias was demonstrated between the syntax scores measured by two different methods. Further studies with long term follow up periods are needed in terms of demonstrating the prognostic value of CT technique in regard to assessment of syntax score with CT angiography.

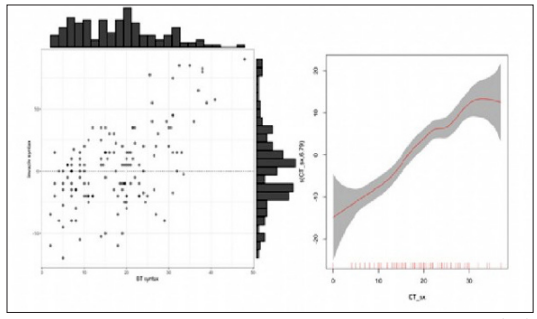

Figure 1. The agreement between CT syntax score and invasive syntax score (left), and the partial effect plot of invasive syntax and CT syntax (right).

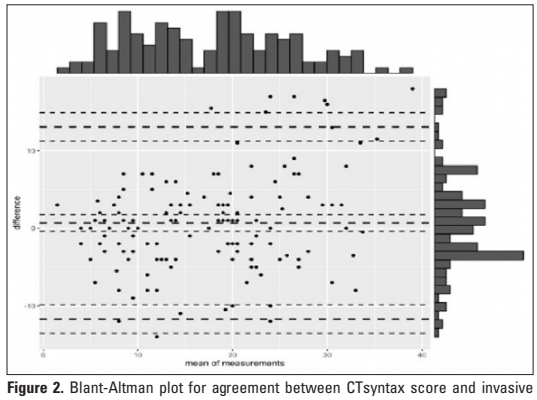

Figure 2. Blant-Altman plot for agreement between CTsyntax score and invasive syntax score.

Interventional cardiology / Coronary

OP-073

The psychological impact of COVID-19 pandemic on cardiologist and their ST segment-elevation myocardial infarction treatment strategies

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Background and Aim: In Worldwide, Primary percutaneous coronary intervention (PPCI) is the first line recommended therapy for Acute ST-segment–elevation myocardial infarction (STEMI) for many years. During this pandemic period, changes on STEMI treatment protocol has been published. Recommendations were based on each regional system's PCI Center, STEMI referral hospitals and EMS (emergency medical system). Another important point is the new type coronavirus epidemic that shook the world poses a serious threat to cardiologists who perform interventional procedures. Little is known about the psychological effects of this type of disease outbreak on health care workers. This document addresses the evaluation of invasive cardiologists' STEMI treatment strategies with the Impact of Event Scale (IES) during COVID-19 pandemic in Turkey.
Methods: This online, questionnaire study conducted via e-mail or WhatsApp message to 1000 cardiologist who works in PCI capable centers in Turkey. Participation was voluntary and participation to the study was expected to be between 10-15%. 136 cardiologists agreed to participate in the study by completing the questionnaire. Self-administered, anonymous questionnaires results were evaluated by a blind researcher. In the study, a 5-point Likert type scale 18 stress-related questions were applied to invasive cardiologists and their STEMI treatment changes during the pandemic were identified by the answers.
Results: It's a small sample size study but the power of the study was 98.53%. This study showed that Turkish cardiologists changed their approach to culprit lesions during PPCI in STEMI patients during pandemic based upon to psychological impact of COVİD-19 pneumonia stress-related factors. Performing PPCI in STEMI patients during pandemic has statistically significant effects on total IES, F1 (Daily moods), F2 (Willingness and ability to work) and F3 (Anxiety about infection) scores. The cardiologists with higher F3 scores preferred more simple and shorter techniques during PPCI in the study. The participants with higher total IES scores preferred interfering only the responsible coronary artery during PPCI in patients with multivessel disease as the participants with higher F1 scores did in the study. Also, these participants with higher total IES and F1 scores showed statistically significant change in treatment protocol of STEMI patients

with multi-vessel disease as rather preferring CABG procedure instead of PCI compared with routine days.
Conclusions: This is the unique study evaluating cardiologists STEMI treatment strategies with the IES during COVID-19 pandemic. This study showed that doctors stress-related factors must be added to account while talking in treatment strategies and publishing recommendation reports during the pandemic besides countries health system, EMS, referral hospitals conditions.

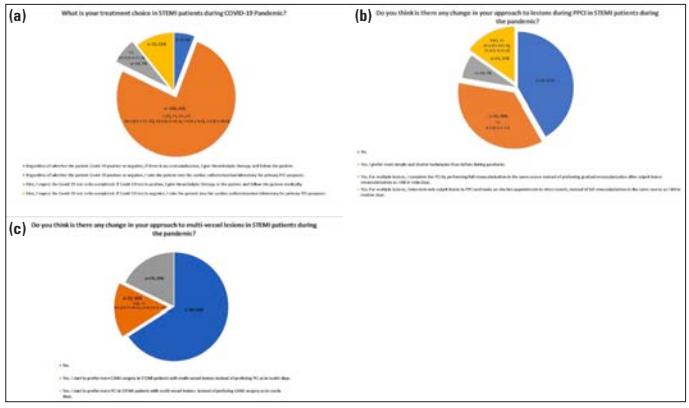

Figure 1. (a-c) Choosing mor than one answer was available.

Table 1. Factor analysis of the 18 stress-related questions

	Factors					
Questions	**F1**	**F2**	**F3**	**F4**	**F5**	h^2
Factor 1. Daily moods ..... (Cronbach α =0.897)						
Q10 Physical exhaustion	**0.87**	0.17	0.09	0.14	-0.09	0.17
Q11 Mental exhaustion	**0.83**	0.13	0.09	0.21	0.02	0.24
Q12 Insomnia	**0.81**	0.02	0.03	0.02	-0.06	0.35
Q15 Burden of increase quantity of work	**0.79**	0.09	0.10	0.07	0.09	0.34
Q16 Burnout from changes in daily functioning	**0.78**	0.11	0.10	0.10	0.13	0.34
Q14 Feeling of being isolated	**0.72**	0.24	0.04	0.12	0.09	0.39
Q13 Elevated mood	**0.56**	-0.02	0.14	0.02	-0.10	0.65
Factor 2. Willingness and ability to work ..... (Cronbach α =0.844)						
Q8 Hesitation to work	0.40	**0.76**	0.26	0.06	-0.02	0.20
Q9 Feeling of having no choice but to work due to obligation	0.45	**0.73**	0.09	0.09	0.04	0.25
Q7 Anxiety about compensation	0.29	**0.70**	0.20	0.08	-0.16	0.35
Q6 Lack of material in protection from infection	0.30	**0.58**	0.14	0.46	-0.18	0.31
Factor 3. Anxiety about infection ..... (Cronbach α =0.822)						
Q2 Anxiety about infecting family	0.17	0.16	**0.85**	0.10	0.01	0.21
Q1 Anxiety about being infected	0.18	0.09	**0.83**	0.22	0.05	0.21
Q3 Anxiety of being infected during commuting	0.27	0.26	**0.71**	0.16	0.02	0.34
Factor 4. Lack of medical information ..... (Cronbach α =0.913)						
Q4 Lack of knowledge about infectivity and virulence	0.23	0.10	0.17	**0.89**	0.03	0.11
Q5 Lack of knowledge about prevention and protection from infection	0.31	0.07	0.16	**0.88**	-0.06	0.09
Factor 5. Feeling of being protected ..... (Cronbach α =0.781)						
Q17 Feeling of being protected by national and local governments	0.04	-0.12	0.11	0.06	**0.90**	0.16
Q18 Feeling of being protected by hospital	0.01	0.01	-0.05	-0.11	**0.89**	0.19
Eigenvalue	6.99	1.96	1.78	1.33	1.02	
Variance Explained (%)					72.07	
Between Factor Corelation						
F2	$-6{,}49E^{-16}$					
F3	$-1{,}96E^{-15}$	$1{,}73E^{-16}$				
F4	$-8{,}31E^{-16}$	$-2{,}39E^{-16}$	$9{,}56E^{-16}$			
F5	$-2{,}01E^{-15}$	$9{,}75E^{-16}$	$2{,}95E^{-16}$	$1{,}26E^{-15}$		

• **Bold**, factor loading ≥ 0.50

Interventional cardiology / Coronary

OP-074

Assessment of silent cerebral infarcts in chronic total occlusion patients with percutaneous coronary intervention

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Background and Aim: Neuron-specific enolase is a cytoplasmic enzyme and sensitive neuronal ischemia marker found in nerve cells. Elevation of Neuron Specific Enolase (NSE) in the absence of any clinically apparent stroke or transient ischemic attack, so-called silent cerebral infarcts (SCIs). SCI may be associated with neurologic deficits. In this study, we aimed to evaluate the incidence of Silent Cerebral infarcts, defined as elevated Neuron Specific Enzyme after coronary chronic total occlusion (CTO) intervention and elective coronary stenting, and procedural factors affecting Silent Cerebral infarcts.
Methods: Study population consisted of 2 groups of patients. Group 1 included consecutive patients with elective coronary chronic total occlusion stenting; group 2 consisted of patients who underwent elective coronary stenting. NSE blood levels were measured before and 12-18 hours after the procedure. Elevation of >20 ng/ml was considered as SCI. Exclusion criteria were baseline NSE elevation, acute coronary syndromes or cardiac surgery within 4 weeks, planned use of glycoprotein IIb/IIIa receptor inhibitors, patients with recent cerebrovascular accident, intracranial hemorrhage, and head trauma, central nervous system tumor, degenerative central nervous system disorders, neuroendocrine tumors.
Results: After pre-evaluation, 120 patients met the study criteria. and 12 of them were excluded for following reasons: 1 patient had myocardial infarction from another coronary artery within 24 hours, 1 patient had acute stent thrombosis, 1 patient had ventricular fibrillation, 1 patient had a stroke during the intervention, 1 patient had transient ischemic attack after PCI, 2 patients underwent unplanned left main coronary artery (LMCA) stenting, 1 patient had hypotension requiring inotropic agent, 4 patient had elevated baseline NSE. Finally, 108 patients were included in the study. fifty-five of 108 study patients (50.9%) had SCI after the procedure. The rate of silent brain infarction was 59.7% in the CTO group and 39.1% in the elective coronary stenting group. Patients with SCI were more likely to have Diabetes Mellitus, hyperlipidemia, higher HbA1c, total stent length, procedural time. Multivariate logistic regression analysis demonstrated CTO procedure (odds ratio [OR] 3.129; 95% confidence interval [CI] 1.246 to 7.858; p<0.015), and presence Diabetes Mellitus (odds ratio [OR] 2.93; 95% confidence interval [CI] 1.185 to 7.291; p<0.020) as independent predictors of SCI.
Conclusions: Increased catheter manipulations, procedure time, and the number of equipment used may lead to an increase in the frequency of silent brain damage in complex procedures such as CTO. It can occur even in patients with elective coronary interventions. This may lead the decreased cerebral functions in the long term. CTO and similar complicated procedures, especially in diabetic patients should be careful about SCI.

Interventional cardiology / Coronary

OP-075

The association between Interleukin-1 Beta single nucleotide gene polymorphisms and acute coronary syndromes

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Background and Aim: Inflammation plays a major role in the pathogenesis of atherosclerosis and acute coronary syndromes. We evaluated the presence of interleukin 1(IL-1) gene polymorphisms in patients presenting with an acute coronary syndrome (ACS).
Methods: A total of 267 patients were enrolled (114 patients presenting with ACS and 153 patients with stable angina pectoris) and underwent coronary angiography. 79 patients in stable angina pectoris (SAP) group with either normal epicardial coronary arteries or mild degree coronary stenosis (<40%) were defined as the control group. PCR analysis was used to determine IL-1 RN genotype. PCR and restriction enzyme digestion method was used to evaluate IL-1 β single nucleotide gene polymorphisms (SNP -511 and +3953).
Results: IL-1 RN polymorphism genotype distribution was not significantly different between ACS - control (p=0.317) and SAP-control groups (p=0.565). IL-1RN allele frequencies did not differ between ACS-control (p=0.126) and SAP-control groups (p=0.411). IL-1β SNP (-511) genotype distribution were statistically similar between ACS-control (p=0.135) and SAP-control groups (p=0.784). Although IL-1β SNP (-511) allele frequency was not different between SAP and control groups (p=0.541), there was significant difference for IL-1β SNP (-511) allele frequency between ACS and control subjects (p=0.039). In addition, IL-1β SNP (+3953) allele frequency was also different for ACS-control (p=0.033) and ACS-SAP groups (p=0.045).
Conclusions: IL-1β single nucleotide gene polymorphisms (IL-1β SNP-511 and IL-1β SNP +3953) are found to be increased in patients presenting with ACS.

Interventional cardiology / Coronary

OP-076

Can the SYNTAX score predict depression in patients with coronary artery disease?

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Background and Aim: Our aim in this study was to investigate between the SYNTAX score (SS) which we evaluated the severity of coronary artery disease and Patient Health Questionnaire-9 (PHQ-9) score which we used for diagnosis and measure of depression.
Methods: Totally 386 patients who were diagnosed acute coronary syndrome (ACS, n=142) and chronic coronary syndrome (CCS, n=244) were included in this study. After coronary angiography, the SS was calculated by Website software (http://www.SYNTAXscore.com) and the PHQ-9 score completed by patients themselves.
Results: There was no different between in two groups regarding mean age, male gender, body mass index, systolic and diastolic blood pressure (p>0.05). But, the SS (5.5±9.0 vs 13.4±11.8, p<0.0001). PHQ-9 score (5.3±4.7 vs 7.3±5.2, p=0.011), heart rate (80.8±13.2 vs 90.1±17.5), creatinine (0.8±0.1 vs 0.9±0.3, p=0.031), aspartate transaminase (24.7±11.6 vs 54.6±82.9, p=0.003) and white blood cells levels (7.6±2.0 vs 9.7±3.1, p<0.001) were lower in CCS when compared to ACS (p>0.05).Only serum LDL level was higher in CCS group (125.2±42.1 vs 111.7±34.0, p=0.026).Correlation analyses showed a strong association between depression score and SS (r=0.267, p<0.001), age (r=0.180, p=0.014) and heart rate (r=0.215, p=0.005). In the regression analysis only the SS [OR:2.7, 95%CI (0.027-0.167), p=0.007] and heart rate [OR:2.44, 95%CI (0.011-0.107), p=0.016] significantly associated with increased the risk of depression.
Conclusions: We found that the SS and the PHQ-9 score were higher in patients with ACS and there was a positive correlation between PHQ-9 score and SS, age, heart rate. Only the SYNTAX score and heart rate were an independent risk factor for depression.

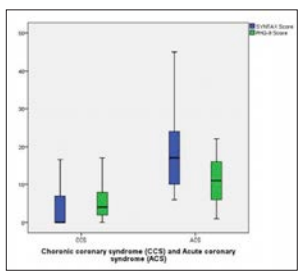

Figure 1. There was a significant difference between PHQ-9 Score and SYNTAX Score between ACS and CCS groups.

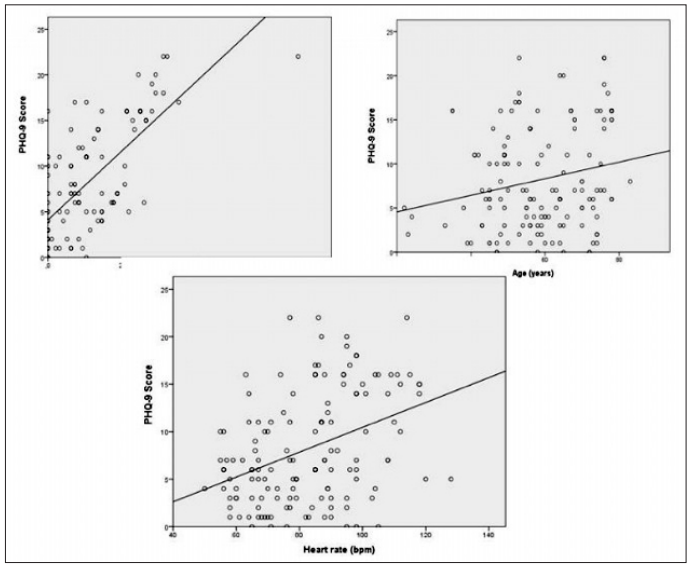

Figure 2. Diagrams showing the association between PHQ-9 score and SYNTAX Score, age, heart rate.

Table 1. The comparison of demographic features, medical history, SYNTAX score and PHQ-9 score of patients with CAD

Variables	Chronic Coronary Syndrome (n=244)	Acute Coronary Syndrome (n=142)	p value
Age (years)	59.3 ± 11.2	61.5 ± 13.1	0.257
Gender (male %)	197 (80.7)	107 (75.3)	0.406
BMI (kg/m2)	30.1 ± 5.8	28.9 ± 5.4	0.185
Heart rate (bpm)	80.8 ± 13.2	90.1 ± 17.5	0.022
Hypertension (%)	117 (47.9)	72 (50.0)	0.638
Diabetes Mellitus (%)	63 (25.8)	46 (33.3)	0.315
Creatinine (mg/dl)	0.8 ± 0.1	0.9 ± 0.3	0.031
AST (U/L)	24.7 ± 11.6	54.6 ± 82.9	0.003
WBC (x103/uL)	7.6 ± 2.0	9.7 ± 3.1	<0.001
LDL-C (mg/dL)	125.2 ± 29.0	111.7 ± 34.0	0.026
Vitamin D (ng/mL)	19.8 ± 23.0	18.1 ± 12.0	0.567
Vitamin B12 (pg/mL)	419.7 ± 197.4	361.3 ± 193.2	0.915
SYNTAX Score	5.5 ± 9.0	13.4 ± 11.8	<0.001
PHQ-9 Score	5.3 ± 4.7	7.3 ± 5.2	0.011

AST: Aspartate Aminotransferase;BMI: Body mass index; LDL:Low-density lipoprotein cholesterol, n: number of patients, WBC:,White blood cell

Table 2. The association between PHQ-9 Score and SYNTAX score, heart rate, age and BMI

Variables	r value	p value
SYNTAX score	0.267	0.000
Heart rate (bpm)	o.215	0.005
Age (year)	0.018	0.014
BMI (kg/m2)	-0.002	0.978

BMI: Body mass index.

Table 3. The regression analyses between PHQ score and SYNTAX score, heart rate, age

Variables	OR	%95 CI	p value
SYNTAX score	2.723	0.027 - 0.167	0.007
Heart rate (bpm)	2.444	0.011 - 0.107	0.016
Age (year)	1.350	0.020 - 0.108	0.179

Interventional cardiology / Coronary

OP-077

The effect of conventional and distal radial access techniques on radial artery structure and vascular functions

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Background and Aim: The transradial (TRA) approach has gained increasing popularity in the world of invasive cardiology in recent years, as it offers many advantages to the patient and operator compared to the transfemoral (TFR) intervention. Yet, transradial intervention also has complications such as radial artery (RA) spasm, RA stenosis, RA occlusion.Also in the previous studies,decreases in RA diameter and functions have been detected after TRA intervention. Radial arteries need to be protected for coronary artery bypass graft (CABG) surgery, hemodialysis fistula preparation and repeated percutaneous coronary interventions. For reasons aforementioned, distal transradial (DTRA) intervention appears as a promising venture. The purpose of this study is to investigate and compare RA diameters, intimal thickness values and vascular functions in patients with conventional RA and distal RA(DRA) cannulation before and after the procedure.
Methods: A total of 40 patients who underwent coronary angiography (CAG) between April 2019 and September 2019 were included in the study. Conventional TRA(TRA) technique was used for 20 patients and DTRA access technique for the other 20. Patients were taken and randomized to the one of the two methods respectively and consecutively. RA flow rates, diameters, intimal thickness values, flow-mediated dilation responses (FMD) were recorded before and after the procedure, using the first day and the first month RA ultrasonography (USG) and Doppler parameters. The success of both procedures and their effects on complications were investigated.
Results: It was found that the number of punctures were significantly higher in the procedures performed with the distal transradial route compared to conventional TRA. When the radial artery intimal thickness values were compared, basal and 1st day intimal thickness values were similar; but in the 1st month, the intimal thickness in the CTRA group was found to be statistically significantly higher than in the DTRA group.In the study, the radial artery occlusion (RAO) was detected in 3 (15%) patients in the CTRA intervention group in the 1st month, and in none of the patients in the DTRA group. However, there was no statistically significant difference due to the insufficient number of patients.
Conclusions: It can be said that DTRA intervention is better in terms of radial artery health. Based on our findings and the studies showing that the patency of the radial artery grafts decreased after catheterization, it can be said that interventional cardiologists should be careful in accessing the radial artery during coronary catheterization in patients who may be suitable for radial artery grafts when a CABG surgery is indicated. This study showed that RA intima thickness increase, RAO ratio, and FMD-evaluated endothelial dysfunction were higher in the CTRA group compared to the DTRA group. DTRA technique should be preferred to CTRA technique in patients who may need intervention with bypass graft, hemodialysis fistula and RA access again in the future.

Interventional cardiology / Coronary

OP-078

The relationship between thrombus burden and left ventricular thrombus in patients with acute anterior ST segment elevation myocardial infarction

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Background and Aim: Thrombus formation in the left ventricle (LVT) causes complications such as systemic embolism and cerebral infarction after acute anterior myocardial infarction. In our study, we aimed to investigate the relationship between thrombus burden in IRA and LVT formation in acute anterior STEMI.
Methods: Patients who underwent p-PCI due to acute anterior STEMI between January 2016 and January 2018, and who underwent echocardiographic controls 1 month later, were included in the study. The patients were divided into two groups as those with low thrombus burden (1-3 degree) and high thrombus burden (4-5 degree) in the infarct-related artery (IRA).
Results: The average age of 312 patients included in the study was 55±12. 70% (217) had male gender and 34% had HT. There were 32 patients with LV thrombus after 1 month of control. There was no statistically significant difference in IRA between patients with high thrombus burden (n=243) and patients with low thrombus burden (n=69) in terms of LVT formation (p=0.42) In the univariate and multivariete logistic regression analysis, a significant relation was found between cigarette use (odds ratio [OR]:3.8, 95% confidence interval [CI]: 2.3-5.9, p<0.001), male gender (OR: 0.14, 95% CI: 0.03-0.55, p=0.005), hyperlipidemia (OR: 8.3, 95% CI: 2.19-31.6, p=0.002), total ischemia time (OR: 0.51, 95% CI: 0.28-0, 91, p=0.023) and low EF (OR: 0.064, 95% CI: 0.55-0.74, p<0.001).
Conclusions: There was no significant relationship between thrombus burden in the IRA and LVT in patients with acute anterior STEMI.

Interventional cardiology / Coronary

OP-079

The predictive value of hemoglobin to creatinine ratio for contrast-induced nephropathy in elective percutaneous coronary interventions

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Background and Aim: To investigate the predictive value of Hemoglobin to Creatinine ratio for Contrast-Induced Nephropathy (CIN) development in patients with elective percutaneous coronary intervention (PCI).
Methods: 500 patients who underwent elective PCI in our clinic were evaluated prospectively in terms of CIN. Hemoglobin to Creatinine ratio calculated = baseline hemoglobin / baseline serum creatinine value. If serum creatinine values were ≥2 mg/dl or patients with hemodialysis were excluded from the study. GFR was calculated with Cockcroft-Gault formula; [(140-age) x body weight (kg)] / [72 x serum creatinine] (if women x 0.85). The definition of CIN includes absolute (≥0.5mg/dl) or relative increase (≥25%) in serum creatinine at 48-72 h after exposure to a contrast agent compared to baseline serum creatinine values.
Results: CIN was detected in 13.8% (69 patients) of 500 patients. The patients with and without CIN are compared and showed that; age, ejection fraction (EF), GFR, Contrast amount used, creatinine, hemoglobine and hemoglobin to creatinine ratio were significantly different between the two groups (Table 1). In multivariate lineer regression analysis, hemoglobin to creatinine ratio (Beta: -0.227, t:-1.682, p=0.03) and EF (Beta: -0.161, t:-3.807, p<0.001), contrast amount used (Beta: 0.231, t: 5.541, p<0.001) were found to be significant predictors for the development of CIN (table 2). The risk of CIN was calculated as: 1.408 – (0.227 x hemoglobin to creatinine ratio) – (0.161 x EF) + (0.231 x contrast amount used). In ROC analysis with hemoglobin to creatinine ratio, EF and contrast amount used variables; AUC=0.730 (0.66-0.79) for hemoglobin to creatinine ratio, p<0.001, AUC = 0.694 (0.62-0.76) for EF, p<0.001 and AUC=0.731 (0.67-0.78) for contrast amount used (Figure 1, 2).

Conclusions: Hemoglobin to creatinine ratio, EF and contrast amount used were independent predictors for CIN development in patients with elective PCI.

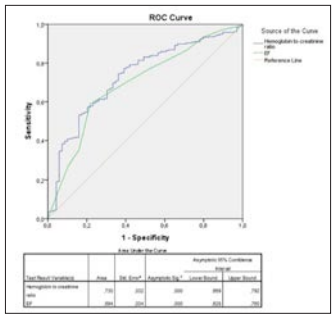

Figure 1. ROC of EF and Hemoglobin to creatinine ratio.

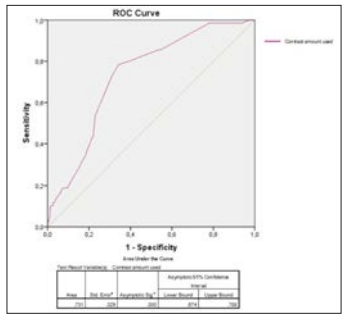

Figure 2. ROC of Contrast amount of used.

Table 1. Characteristics of patients with and without CIN

Variable	CIN (+) 69 patients	CIN (-) 431 patients	P value
Age (year)	67.09±10.20	60.22±11.06	<0.001
Gender, n (%) Female Male	20 (29.0) 49 (71.0)	108 (25.1) 323 (74.9)	0.29
BMI (kg/m2)	27.86±4.28	28.43±4.20	0.29
Hypertension n (%)	40 (58.0)	206 (47.8)	0.11
Diabetes mellitus n (%)	27 (39.1)	140 (32.5)	0.27
LV EF (%)	41.88±10.25	48.65±9.73	<0.001
Metformin,n (%)	19 (27.5)	117 (27.3)	0.96
ACEI n (%)	43 (62.3)	307 (71.2)	0.13
Statin n (%)	68 (98.6)	407 (94.4)	0.14
Hemoglobin (gr/dl)	12.82±1.74	13.22±1.61	0.03
Glucose (mg/dl)	162.49±67.88	152.66±81.60	0.34
Creatinine (mg/dl)	0.99±0.17	0.85±0.21	<0.001
GFR (ml/min)	79.19±28.03	105.47±33.20	<0.001
Contrast amount (ml)	190.43±105.77	130.94±66.64	<0.001
Hemoglobin to creatinine ratio	13.32±3.41	16.21±3.89	<0.001

BMI: Body mass index, LV EF: Left ventricular ejection fraction, ACEI: Angiotensin-converting enzyme inhibitor, GFR: Glomerular filtration rate.

Table 2. Multivariate linear regression analysis

Variable	Standartize Coefficient Beta	t Value	P value
Age	0.053	0.814	0.41
LV EF	-0.161	-3.807	<0.001
GFR	-0.082	-1.006	0.31
Contrast amount	0.231	5.541	<0.001
Creatinine	-0.058	-0.493	0.622
Hemoglobin	0.083	1.127	0.260
Hemoglobin to creatinine ratio	-0.227	-1.682	0.03

LV EF: Left ventricular ejection fraction, GFR: Glomerular filtration rate.

Interventional cardiology / Coronary

OP-080

Rational guidewire use in the coronary chronic total occlusion interventions

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Background and Aim: Procedures for the chronic total occlusion (CTO) is a still clinical challenge with relatively lower success rate. Recent advance in the biotechnology and introduction of CTO dedicated guidewires increased the procedural success rate of CTO interventions. Herein, we aimed to reveal the clinical and angiographic predictors of the initial choice guidewire crossability and rational guidewire usage in the CTO interventions.
Methods: A total of 177 patients with indication of coronary CTO procedure were included to study. Use of 1-3 guidewires and crossing of the CTO lesion with initial choice guidewires was defined as rational guidewires usage. The CTO lesions was classified according to J-CTO score and Eurocto score for evaluating the difficulty of the procedures. Then statistical analysis was performed to assess the guidewire choise, crossability and contributors of rational guidewire usage.
Results: The mean J-CTO score was 1.42±1.16 and the mean Eurocto score was 1.44±1.18. Success rate of the procedures was 90.4%. Initial choice guidewires was crossed the lesion in the 44.1% of the cases in which 1-3 guidewires was used (82.1%). Crossability of polymeric and moderate tip stiff guidewires are higher (82.1% and 64.1%) and Pilot series was the most successfull brand (36.2%). Logistic regression analysis confirmed that J-CTO score, procedural technique, guidewire type and the tip stiffness was the major predictors of rational guidewire usage.
Conclusions: Our analysis showed that, use of polymeric and moderate tip stiff guidewires, particulary Pilot brand, is associated with rational guidewire usage in the easy and intermediate difficulty CTO cases.

Interventional cardiology / Coronary

OP-081

Usefulness of triglyceride-glucose index for the prediction of coronary artery disease in patients undergoing elective coronary angiography

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Background and Aim: Coronary artery disease (CAD) is the leading cause of morbidity and mortality of cardiovascular diseases all over the world. In a recent study, the triglyceride-glucose index (TyG index) has been shown to be a reliable alternative marker of insulin resistance in addition to be an independent predictor of poor cardiovascular outcomes. However, the predictive value of TyG index in predicting CAD for patients undergoing elective coronary angiography (CAG) is unknown. In this study, we aimed to determine the predictive role of the TyG index for the presence of CAD in patients undergoing elective CAG.
Methods: In total, 184 patients who underwent elective CAG due to either the presence of ischemic findings in the imaging tests or moderate or high-risk cardiovascular stress test were included in this retrospective study. The study cohort was divided into two groups; patients with CAD and those without CAD. The SYNTAX score and TyG index were calculated for all patients. The TyG index was calculated using the following equation; Ln [fasting TG (mg/dl) × fasting glucose (mg/ dl)/2].
Results: The incidence of CAD was 60.8% (n=112 patients). The patients with CAD were older, diabetic, and hypertensive. Respectively, the TyG index and the SYNTAX score were significantly higher in patients with CAD than those without (4.92±0.31 vs. 4.78±0.32, p<0.001 and 16.41±9.88 vs. 1.46±2.63, p<0.001, respectively). Multivariate logistic regression analyses revealed that TyG index was an independent predictor of CAD (OR: 6.611, 95%CI: 2.232–19.584; p<0.001). A receiver operating characteristic curves analysis yielded that the optimal cut-off value of TyG index score for CAD was 4.79 with sensitivity 63.4% and specificity 61.1% (AUC: 0.642, 95%CI: 0.559-0.726, p<0.001).
Conclusions: Based on the findings of the study, we were able to show that the TyG index may be an independent predictor of CAD in patients undergoing elective CAG. This simple and easily achieved risk score may be used in predicting the risk of CAD before elective CAG as additional to the standard cardiovascular tests.

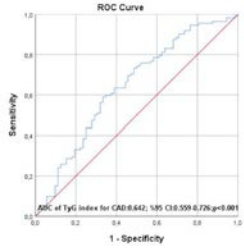

Figure 1.

Table 1. Baseline characteristic of all patients

	Patients without CAD, (n:72)	Patients with CAD, (n:112)	P value
Age, years	58.7 ± 11.6	64.9 ± 10.9	<0.001
Body mass index, kg/m2	28.6 ± 5.5	29.8 ± 5.0	0.261
Male, gender, n (%)	46 (63.9)	68 (60.7)	0.782
Systolic blood pressure, mmHg	133.1 ± 21.5	135.4 ± 20.9	0.857
Diastolic blood pressure, mmHg	74.2 ± 12.0	74.5 ± 13.1	0.828
Heart rate, beats per minute	72.9 ± 19.9	78.5 ± 16.4	0.002
History			
Hypertension, n (%)	34 (47.2)	74 (66.1)	0.011
Diabetes mellitus, n (%)	16 (22.2)	51 (45.5)	0.001
Hyperlipidemia, n (%)	22 (30.6)	38 (33.9)	0.633
Current smoking status, n (%)	37 (51.4)	50 (44.6)	0.371
Admission laboratory variables			
Admission glucose, mg/dL	118.1 ± 52.2	138.5 ± 57.1	0.001
Admission BUN, ng/mL	35.8 ± 14.5	41.1 ± 19.7	0.083
Creatinine, mg/dL	0.84 ± 0.25	1.02 ± 0.77	0.020
White blood cell count, cells/μL	8.7 ± 2.6	8.1 ± 3.5	0.403
Hemoglobin, g/dL	13.2 ± 1.7	12.4 ± 2.1	0.009
Platelet count, cells/μL	254.5 ± 80.9	247.3 ± 108.5	0.523
ALT, U/L	23.7 ± 21.1	32.5 ± 79.7	0.002
AST, U/L	31.0 ± 45.5	46.7 ± 143.8	0.690
Total cholesterol, mg/dL	196.1 ± 45.4	189.9 ± 53.7	0.616
Triglyceride, mg/dL	144.7 ± 81.6	167.7 ± 93.1	0.058
HDL-cholesterol, mg/dL	43.1 ± 11.2	40.4 ± 10.8	0.187
LDL-cholesterol, mg/dL	125.7 ± 40.0	117.9 ± 43.2	0.380
SYNTAX Score	1.46 ± 2.63	16.41 ± 9.88	<0.001
TyG index	4.78 ± 0.32	4.92 ± 0.31	<0.001

Continuous variables are presented as mean ± SD, nominal variables presented as frequency (%). BUN indicates blood urea nitrogen; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; HDL indicates high density lipoprotein; LDL indicates low density lipoprotein, TyG index indicates triglyceride and glucose index.

Interventional cardiology / Coronary

OP-082

Prognostic nutritional index predicts contrast-associated acute kidney injury in patients with ST-segment elevation myocardial infarction

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Background and Aim: Contrast-associated acute kidney injury (CA-AKI) previously named as contrast-induced nephropathy is associated with worse prognosis in patients with acute ST-elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (PCI). Prognostic nutritional index (PNI) is a simple index comprised of serum albumin levels and lymphocyte count to reflect the immunonutritional-inflammatory status. Recently, clinical researches have shown associations between PNI and clinical outcomes in several cardiovascular diseases.The aim of the study was to assess the possible utilization of PNI for development of CA-AKI after primary PCI.
Methods: We retrospectively included 836 patients (mean age of 58±12, 76% men) with STEMI treated with primary PCI. The PNI was calculated as 10 x serum albumin (g/dL) + 0.005 x total lymphocyte count (per mm^3). The patients were divided into two groups according to whether CA-AKI developed or not.
Results: The overall incidence of CA-AKI was 9.4%. Compared to patients without CA-AKI, patients with CA-AKI had significantly lower PNI values (35.2±4.9 vs 40.7±3.7; p<0.001). In a multivariate logistic regression model, PNI, as well as age and creatinine, was independently associated with CI-AKI (odds ratio 0.797, 95% confidence interval 0.709-0.895; p<0.001). In receiver operating characteristics analysis, the optimal cutoff value of PNI to predict CA-AKI was 38, with 82% sensitivity and 70% specificity (area under the curve 0.836, p<0.001).
Conclusions: In conclusion, the PNI was significantly associated with the development of CA-AKI in patients with acute STEMI. Assessing PNI may be useful for risk stratification of STEMI patients.

Interventional cardiology / Coronary

OP-083

Evaluation of late clinical follow-up and outcomes of patients who presented with NSTEMI and treated with bioresorbable scaffolds (BRS)

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Background and Aim: Bioresorbable stents (BRS) are used in the last stage of stent technology and have bad results than drug-eluting stents (DES). There are many publications on these stents that have been studied in patients with SAP and whose results are encouraging. We aimed to evaluate the late (4 years) clinical outcomes of BRS patients who were admitted with non-ST-segment elevation myocardial infarction (NSTEMI), a type of ACS in our study.
Methods: The study included 39 patients with a total of 53 coronary lesions who were treated with BRS in Istanbul Medipol University Faculty of Medicine between June 2015-April 2016. 4 (Four) years endpoints were considered as device failure, procedural success, stent thrombosis, and major cardiac events (MACE).
Results: The transactions took place with 98,1% of device success and 98,1% of transaction success. Target lesion revascularization (TLR) was performed in 4 (four) patient in 4 (four) years without death in any patient or detection of stent thrombosis. A total MACE of 1.9 was detected. During the procedure and during admission, complications developed in 2 (two) patients.
Conclusions: High procedural success, low complication rate and low rate of major cardiac events; BRSs can be safely chosen if they are operated by experienced operators in NSTEMI patients. To obtain better results, it is necessary to develop BRSs that have good radial strength, thinner strut thickness and shorter dissolution time.

Interventional cardiology / Coronary

OP-085

Fragmented QRS at hospital presentation is independently associated with the development of no-reflow in patients with first anterior ST-elevation myocardial infarction

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Background and Aim: No-reflow which is the absence of sufficient myocardial perfusion after stent implantation is associated with increased mortality in patients with ST-elevation myocardial infarction (STEMI). There is no definitive treatment although some preventive approaches. Therefore, it is important to predict the patients at risk of no-reflow. Fragmented QRS (fQRS) is the presence of different RSR' patterns without bundle branch on electrocardiography. Both fQRS and no-reflow share similar pathophysiological mechanisms. Thus, we thought that the presence of fQRS on admission to hospital may reflect an increased risk for no-reflow. The aim of the study was to assess the possible relationship between fQRS on admission and the development of no-reflow.
Methods: The study included patients with first anterior STEMI who underwent primary percutaneous intervention. fQRS was evaluated at the time of hospital admission. No-reflow was diagnosed by thrombolysis in myocardial infarction flow grade (<3) after stent implantation. A multivariable model was created to determine factors independently associated with no-reflow.
Results: The study included 259 patients, comprising 56 (20.3%) with no-reflow and 203 (73.6%) without no-reflow. fQRS was more frequent in patients with no-reflow than in those without (71.4% vs 42.4%, p<0.001). In the multivariable model, fQRS (OR:3.731, 95% CI: 1.912-7.279, p>0.001) and male gender (OR:2.351, 95% CI:1.025-5.391, p=0.043) were independently associated with the development of no-reflow.
Conclusions: The presence of fQRS on admission is independently associated with no-reflow in these patients. It can be used a simple and non-invasive tool to predict the patients at risk for no-reflow.

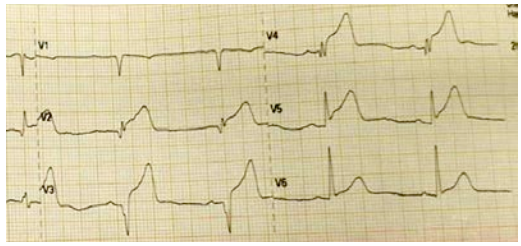

Figure 1. There were fragmented QRS between derivations from V2 to V4 on electrocardiography obtained at the time of admission to hospital.

Interventional cardiology / Coronary

OP-086

The clinical significance left ventricular end-diastolic pressure in patients with ST elevation myocardial infarction

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Background and Aim: We hypothesize that increased Left ventricular end-diastolic pressure (LVEDP) is associated with adverse outcomes following ST-elevation myocardial infarction (STEMI). Our principal aim was to demonstrate the factors associated with increased LVEDP. We also search for predictive value of increased LVEDP for adverse cardiac events. LVEDP is an indicator of both systolic and diastolic functions of left ventricle.
Methods: We prospectively included 155 patients treated with primary percutaneous intervention (pPCI). LVEDP was measured prior to pPCI. Patients' baseline characteristics, comorbidities and clinical variables were recorded. Normal LVEDP was accepted as <18 mmHg.
Results: Patients were divided into two groups; group 1 included patients with normal LVEDP (41 patients) and rest of the study population placed into group 2 (114 patients). There was no significant difference between groups with respect to age (p=0.233) and gender (p=0.382). Comorbidities in terms of coronary artery disease (p=0.927), diabetes mellitus (0.839), cerebrovascular accident (p=0.707) and hypertension (p=0.504) was also similar between groups. Ischemia duration was significantly longer in group 2 (p<0.001). Anterior location of STEMI (30% vs 54%, p=0.007) and total occlusion of infarct-related artery (46% vs 74%, p=0.001) are significantly more prevalent in group 2 patients. Pro-BNP and peak troponin levels were also higher in group 2 (p<0.001 for pro-BNP and p=0.002 for troponin). Left ventricular ejection fraction was also lower in group 2 (48.7±6.2 vs 46.2±8.1, p=0.042). Multivariate analyses revealed that, ischemia duration, anterior location, total occlusion of IRA and pro-BNP independently associated with increased LVEDP. New-onset heart failure was significantly more prevalent in group 2 (p=0.001), whereas although 30-day mortality was observed numerically higher in group 2, there was no significant difference between groups (p=0.448).
Conclusions: We have demonstrated that increased LVEDP is significantly associated with markers of higher myocardial injury. Moreover, increased LVEDP predicts new-onset heart failure and has a potential to be used as a predictor of adverse cardiovascular outcomes.

Interventional cardiology / Coronary

OP-087

The effect of successful percutaneous coronary intervention on ventricular repolarization in patients with chronic total occlusion

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Background and Aim: The QRST angle (QRSTa) is a novel marker of myocardial repolarization heterogeneity which is related with adverse cardiovascular events. Our aim in this study is to investigate the effect of successful percutaneous coronary intervention (PCI) for chronic total occlusion (CTO) on frontal QRSTa.
Methods: A total of 132 patients undergoing PCI for CTO were included in this study. Of these 132 patients, successful PCI of CTO segment was performed in 84 patients (group 2) while 48 failed CTO was observed (group 1). Baseline demographic and clinical variables were evaluated as well as a 12-lead surface ECG of all subjects that had been recorded before performing coronary angiography and 1-month and 6-month later after the index procedure for group 2 patients.
Results: QRSTa values significantly decreased during follow-up visits compared to baseline value [92.5 (63.25-110.75); 85.0 (59.0-101.0); 80.0 (53.0-99.0), p<0.001]. 1. month QRSTa [92.5 (63.25-110.75); 85.0 (59.0-101.0), p=0.002] and 6. month QRSTa [92.5 (63.25-110.75); 80.0 (53.0-99.0), p<0.001] were lower than baseline value while 6. month value [85.0 (59.0-101.0); 80.0 (53.0-99.0), p=0.002] was lower compared to 1. month value. Additionally, decreasing of QRSTa was observed regardless of target vessel or Rentrop classification [98 (63.5-118.5); 95 (67.5-115.25); 85 (49.25-121.75), p=0.010 for LAD], [84 (40-103.5); 76 (33-94); 77 (30.5-93.5), p<0.001 for CXA], [90 (64-111); 86 (59-105); 81 (53-102), p<0.001 for RCA], [74 (38.5-97); 72 (31.5-85.5); 71 (28.5-85.5), p=0.003 for Rentrop 1], [89 (76.25-98.75); 87 (64.25-91); 80 (66.25-86.5), p=0.011 for Rentrop 2], [99 (65.5-118.5); 88 (63-115); 84 (61.5-105), p<0.001 for Rentrop 3].
Conclusions: Successful percutaneous revascularization of CTO was effective on ventricular repolarization. QRSTa significantly decreased after the successful PCI of CTO at 6-month follow-up.

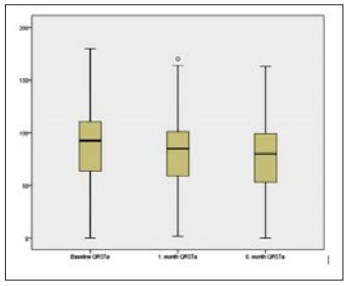

Figure 1.

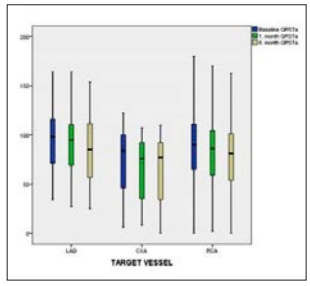

Figure 2.

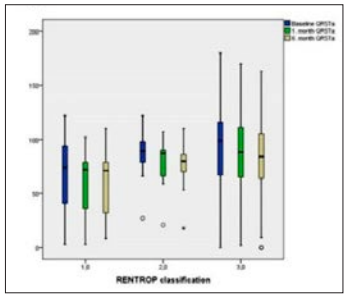

Figure 3.

Interventional cardiology / Coronary

OP-088

Coronary reflux sign: The practical marker for prediction for left ventricular filling pressure in patients undergoing coronary angiography

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Background and Aim: In angiographic view, dye flow features is important for clinicians. The segmental back flow (milking phenomenon), and slow flow pattern have been known as typical angiographic appearance. Similarly, we have declared new coronary imagine 'reflux sign' that dye fluctuation reflux in the left main coronary artery in coronary angiography.
Methods: The prospective study included 2400 patients admitted angiography laboratory between February 2018 and March 2019. In coronary angiography, we saw the contrast agent's fluctuation in left main coronary artery and defined this appearance as coronary reflux sign. Of the patients, 128 patients had coronary reflux sign in left main coronary vessel imaging. The control group (n=100) had no coronary reflux sign. The both groups were examined in terms of left ventricle catheterization findings.
Results: The age of both of groups were similar. The patients with reflux sign composed of severe aortic valvular disorder, severe mitral insufficiency, untreated hypertension, and hypertrophic cardiomyopathy. The patients with coronary reflux sign had higher left ventricular end-diastolic pressure than control group (22.37±1.36 mmHg, 14.40±2.42 mmHg, respectively, p<0.05).
Conclusions: The coronary reflux sign is significantly associated with high left ventricular end-diastolic pressure. This imagine may be practical marker for clinicians.

Interventional cardiology / Coronary

OP-089

The effect of heart rate on fractıonal flow reserve

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Background and Aim: Coronary artery disease is the most important cause of morbidity and mortality around the world. Coronary angiography is the gold standard diagnostic method for the diagnosis of coronary artery disease. However, it is not possible to determine whether the lesion causes ischemia in patients with moderate stenosis by coronary angiography. Therefore, Fractional Flow Reserve (FFR) method has been developed to evaluate these lesions functionally. FFR values are also affected by some hemodynamic parameters such as heart rate and blood pressure. In this study, we aimed to investigate the effect of heart rate on FFR.
Methods: Thirty-eight patients with moderate stenosis with FFR indication were included in the study. Primarily basal FFR measurement, then, FFR measurements were recorded by making 300 mcg and 500 mcg adenosine to ensure hyperemia. Then, atrial pacing was performed and the heart rate was 100/min, 120/min, 140/min, 160/min, 180/min FFR measurements were taken and recorded. The latest heart rate was set to 140/min with pace, simultaneous 300 mcg and 500 mcg adenosine were obtained and FFR measurements was recorded. All measurements taken compared to each other.
Results: FFR values decreased with increased heart rate. However, a significant decrease was observed when the heart rate increased above 140 / min (p<0.001). The highest decrease was observed when heart rate 140/min + adenosine was administered. As a result of comparison of the demographic characteristics of patients and whether or not nitrate was performed before the procedure, a statistically significant difference was found when heart rate was 140/min and 180/min compared to those without diabetes
Conclusions: As a result, it was observed that FFR measurement in patients with moderate lesions was affected by the change in heart rate. As the heart rate increased, FFR values decreased. Significant decrease in FFR was observed when heart rate increased to 140/min and above, and maximum decrease was observed when heart rate 140/min and simultaneous adenosine was administered.Although the number of previous studies on this subject was limited, the results were consistent with the results of other previous studies.

Interventional cardiology / Coronary

OP-090

Acute coronary syndrome with stent restenosis accompanied by large side branch osteal lesion: Which bifurcation stenting strategy should be preferred?

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Background and Aim: Coronary Bifurcation Lesions (CBLs) are complicated from the point of difficulties at making decision between contemporary techniques and peri-postprecedural results as stent thrombosis. Mini crust stenting and culotte stenting are most frequently used intervention techniques at narrower bifurcation angle cases. At this stage, native diameter difference among the target vessels could be determinative to make decision. We want to present this case that we have performed successful culotte stenting technique to stent restenosis
Results: A 54-years-old man presented to our clinic as Non-ST-Segment Elevation Myocard Infarction. Medical history was significant for Coronary Artery Disease, Type 2 Diabetes Mellitus, Hypertension and Hyperlipidemia. On coronary angiogram there was sequential severe (99%) stenosis on middle segment and begining of the distal segment of Circumflex artery(Cx) and 90% blockage on osteal segment of Obtuse Marginalis 2 (OM2) branch (Figure 1). Two floppy guidewires were placed in Cx and OM2.Percutaneous transluminal Coronary Angioplasty (PTCA) were performed at either branches (Figure 2). Although we thought that there would be a confined three stent layer region on middle segment of Cx, Culotte stenting was found more suitable than mini-crush stenting by us. Because there wouldn't be a multiple stent strate on osteal segment of large seen OM2. Therefore, a 2.75 * 30-mm Resolute Integrity- Drug Eluting Stent (DES) (Medtronic Inc., Minnesota, USA) was implanted to cover lesions from Cx to OM2 (Figure 3). Cx was rewarded and PTCA was performed in this region. Then a 3.5 * 48 mm Xience Pro-DES (Abbott laboratories, Abbott Park, IL) was implanted (Figure 4). OM2 rewired; The procedure was terminated with final kissing baloon dilatation and final proximal optimization technique (POT) (Figure 5 and Figure 6).
Conclusions: At CBLs, operator could have difficulty in deciding,in the present of well –developed side branch lesions originated from the stented area of major vessel. In this case, we have chosen culotte stenting technique rather than mini-crush stenting cause of being more suitable for rewiring and final kissing baloon dilatation.

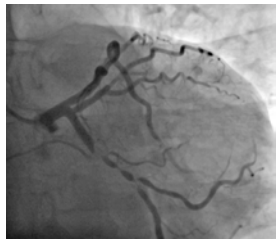

Figure 1. Sequential severe (99%) stenosis on middle segment and begining of the distal segment of Cx, 90% blockage at OM2 ostium.

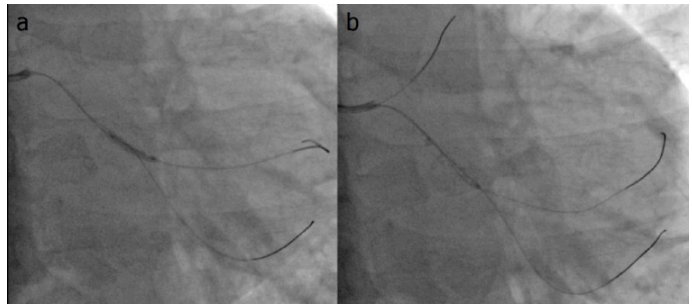

Figure 2. (a) First PTCA on OM2 lesion **(b)** First PTCA on Cx lesions.

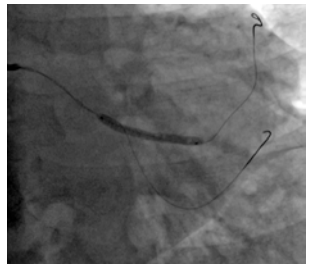

Figure 3. DES implantation on OM2 lesion.

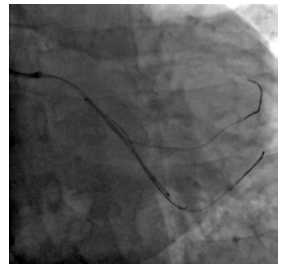

Figure 4. DES implantation on Cx lesions.

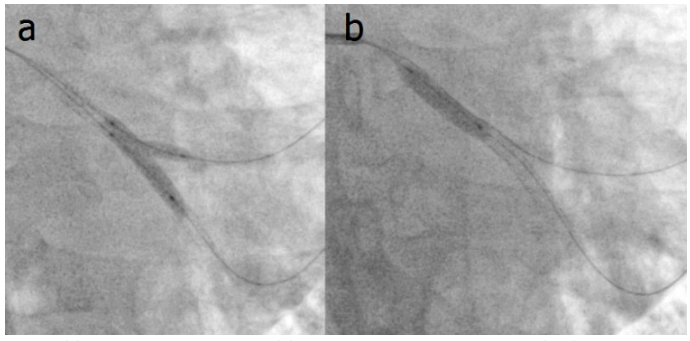

Figure 5. (a) Final kissing baloon dilatation **(b)** Final Proximal Optimization Technique (POT).

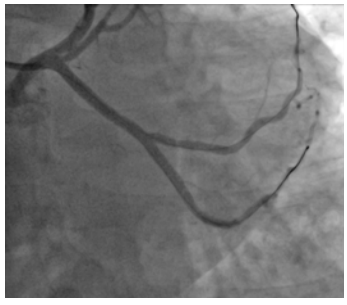

Figure 6. Post-procedural angiographic appearance of Cx and OM2.

Interventional cardiology / Coronary

OP-091

Serum irisin levels and coronary collateral circulation

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Background and Aim: Among the most important determinants of development of coronary collateral circulation (CCC) are presence of a severe coronary stenosis or total chronic total occlusion (CTO), diabetes mellitus (DM), some inflammatory agents and growth factors. The patients with DM have lower irisin levels, and the effect of irisin on the pathophysiological process of atherosclerosis has recently been shown. We investigated whether there is a possible connection between irisin and CCC in patients with at least one epicardial coronary stenosis of 90% or more

Methods: We included Rentrop 0-1 into poor CCC group (N=45) and Rentrop 2-3 into good CCC group (n=41), and measured serum irisin levels.

Results: Serum irisin levels did not differ (17585 [882-37741] pg / ml and (17504 [813-47683] pg / ml, p=0.772). Serum irisin levels were negatively correlated with fasting blood glucose (r=-0.209; p=0.046) and triglyceride levels (r=-0.282; p=0.009) but had no correlation with C reactive protein and SYNTAX score (p>0.05 for all). Serum irisin levels were lower in patients with diabetes (= 41) (14485 [813-29398] pg/ml) than those without diabetes (n=45; 19724 [865-47683] pg/ml (p=0.002). Regression analysis, the determinant of serum irisin level was found to be the diabetes (R^2=35.6, p=0.001).

Conclusions: Although its level is decreased in patients with diabetes, serum irisin levels have no role in the pathophysiology of collateral development and lesion complexity.

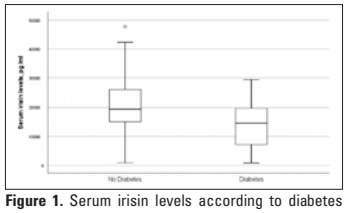

Figure 1. Serum irisin levels according to diabetes mellitus or non-diabetics in patients with coronary artery disease.

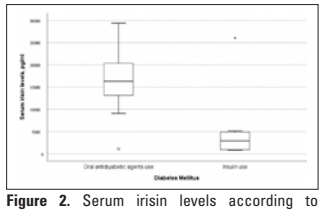

Figure 2. Serum irisin levels according to medication in patients with coronary artery disease and diabetes mellitus.

Table 1. Comparison of demographic, anthropological and biochemical properties of the good and bad collateral groups

Variables	Good collateral group n=41	Poor collateral group n=45	P value
Age, year	62.3±5.6	59±5.3	0.104
Female/Male	8/33	6/39	0.512†
Body mass index, kg / m2	27.8±5.1	27.7±4.3	0.982†
Diabetes mellitus, n (%)	19(46.3)	22(48.9)	0.813
Serum irisin, pg/ml	17585(882-37741)	17504(813-47683)	0.772
Hs-CRP, mg /dl	5.6(0.2-9.3)	4.9(0.1-9.8)	0.905
Creatinine, mg / dl	0.84(0.61-1.32)	0.91(0.7-1.29)	0.218

Interventional cardiology / Coronary

OP-092

Can plasma TWEAK level predict coronary slow flow in patients with mild to moderate chronic kidney disease?

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Background and Aim: The tumor necrosis factor-like weak inducer of apoptosis (TWEAK) is one of the inflammatory mediators contributing to the atherosclerotic process. This prospective study was designed to clarify any relationship between coronary slow flow (CSF) and TWEAK levels.

Methods: This prospective study included 93 consecutive patients undergoing invasive coronary angiography (ICA) for any reason except for acute coronary syndromes from May 2019 to March 2020 (Table 1). A total of 93 patients were divided into two groups with regard to having CSF (n=35) or no-CSF (n=58).

Results: Patients with CSF had higher TWEAK levels than those without CSF (695.2± 225.2 vs 465.8±157.6, p<0.001) (Fig 1). As the number of coronary arteries with slow flow increased, the TWEAK level was increased statistically significant (r=0.635/ p<0.001). The TWEAK level of 516 pg/mL was found for the prediction of CSF in ROC analysis (Fig 2).

Conclusions: Our study has shown that plasma TWEAK level is an independent predictor for the CSF in patients with mild to moderate CKD (Table 2).

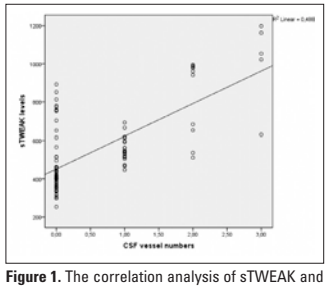

Figure 1. The correlation analysis of sTWEAK and the number of coronary arteries with slow flow.

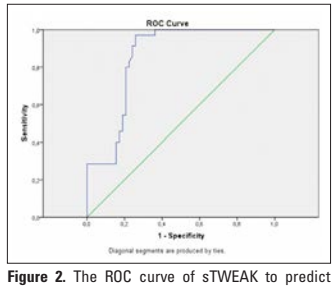

Figure 2. The ROC curve of sTWEAK to predict coronary slow flow.

Table 1. Clinical and laboratory characteristics of patients with grade 2-3 kidney disease divided into 2 groups: CSF and no-CSF

	CSF(+) (n=35)	CSF(-) (n=58)	p
Age, years	64.5 ± 8.6	60.6 ± 7.3	**0.023**
Gender (male%)	16 (%45)	23 (%39)	0.566
BMI, kg/m2	27.8 ± 4.1	29.4± 4.0	0.075
HTN n(%)	16(%45)	14(%24)	**0.031**
DM n(%)	15(%42)	13(%22)	**0.037**
Smoking n(%)	13(%37)	14(%24)	0.181
sBP, mmHg	134.6±21.9	130.7±25.8	0.459
dBP, mmHg	74.6±11.2	72.2±13.0	0.364
LVEF,%	64.6±3.6	65.7±3.9	0.181
Hemoglobin, g/dl	13.22±1.14	13.98±1.52	0.770
Hematocrit, %	41.80±5.55	42.06±5.22	0.681
Creatinine, mg/dl	0.8±0.1	0.8±0.1	0.655
GFR, ml/dk/1.73 m2	78.5±7.7	76.4±8.4	0.250
Sodium, mmol/L	139.6±1.6	139.5±2.3	0.829
Potassium, mmol/L	4.3±0.3	4.3±0.4	0.730
Calcium, mmol/L	11.9±1.6	9.3±0.4	0.223
Phosphate, mmol/L	3.5±0.4	3.5±0.5	0.699
AST, U/L	19.4±7.3	21.2±8.6	0.297
ALT, U/L	21.0±8.2	21.0±7.7	0.993
CRP , mg/dl	20(20-30)	20(20-40)	0.762
Albumine, g/dl	4.2±0.2	4.3±0.3	0.746
TC, mg/dl	197.2±40.2	190.2±47.0	0.460
LDL, mg/dl	116.9±40.3	111.1±39.8	0.508
HDL, mg/dl	46.2±12.4	49.7±11.5	0.184
Non-HDL, mg/dl	150.7±41.1	137.7±44.7	0.166
TG, mg/dl	146.0±61.4	150.1±72.3	0.800
sTWEAK , pg/ml	695.2± 225.2	465.8±157. 6	**<0.001**
PTH, pg/ml	55.3±20.7	48.2±19.0	0.100

Abbreviations: AST, aspartate aminotransferase; ALT, alanine aminotransferase; BMI, body mass index; CRP, C-reactive protein; dBP, diastolic blood pressure; DM, diabetes mellitus; ; GFR, glomerul filtration rate; HDL, high-density lipoprotein; HTN, hypertension; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; PTH, parathyroid hormone; sBP, systolic blood pressure; TC, total cholesterol; TG, triglyceride.

*Continuous variables are presented as mean (SD); nominal variables presented as frequency (%).

Table 2. Univariate and multivariate cox regression analyses for predicting coronary slow flow

Variable	Univariate			Multivariate		
	OR	95%CI	P	OR	95%CI	P
Age	1.068	1.008-1.132	**0.027**	1.084	1.011-1.164	**0.024**
Gender	0.780	0.334-1.822	0.566			
BMI	0.909	0.817-1.011	0.078	0.923	0.798-1.067	0.279
HTN	1.255	1.071-1.465	**0.033**	1.079	1.008-1.156	**0.028**
DM	2.596	1.044-6.453	**0.040**	1.409	0.359-5.531	0.623
Smoking	1.857	0.746-4.623	0.183			
LVEF	0.926	0.826-1.037	0.181			
sTWEAK (>516 pg/mL)	12.333	3.399-33.548	**<0.001**	8.687	2.985-19.241	**<0.001**

Abbreviations: BMI, body mass index; DM, Diabetes Mellitus; HTN, hypertension;; LVEF, left ventricular ejection fraction.

*All clinically relevant parameters were included in the model.

Interventional cardiology / Coronary

OP-093

Comparison of the effects of bare metal stents and drug eluting stents on C-reactive protein levels

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Background and Aim: It's suggested that drug eluting stents (DES) may have systemic anti inflammatory properties and this can play a role in decreased restenosis rates. We aimed to compare bare metal stents (BMS) and DES on their effects on C-reactive protein (CRP) levels, a good marker of systemic inflammation. We also aimed to investigate the relation between the inflammation levels and myonecrosis and adverse cardiac events.

Methods: The patients undergoing elective stent implantation were grouped according to the stent type as BMS (n=70) and DES (n=42). Basal and 24th hour postprocedural CRP and CKMB levels were measured and the difference (Δ- delta) was compared between the groups. The patients were followed up for adverse cardiac events (non-fatal myocardial infarction, death and target vessel revascularisation) for one year. Basal and 24th hour postprocedural CRP and CKMB levels were measured and the difference (Δ) was compared between the groups. The patients were followed up for adverse cardiac events for one year.

Results: Mean age was 62±11 and 75% were males. The patients with diabetes, were significantly higher in BMS group when the basal characteristics of the stent groups were compared. There was significant CRP rise in both groups at 24th hour (Figure 1), but the ΔCRP level was 2.1 (0.5–6.2) mg/L in BMS and 2.3 (0.2–5.2) mg/L in DES group, the difference was not statistically significant (p=0.703). When we assessed the variables contributing to ΔCRP in BMS and DES groups seperately, only the stent length variable was significantly correlated with ΔCRP in DES group (p=0.016), whereas in BMS group stent length was not significantly related with ΔCRP (p=0.341) (Figure 2). ΔCKMB levels and adverse cardiac event rates were similar between the two groups (p=0.897 and p=0.785). There was no correlation between ΔCRP and ΔCKMB levels in both groups (r=-0.090 and p=0.459 for BMS, r=0.158 and p=0.318 for DES group). Similarly, the effect of ΔCRP on the incidence of adverse cardiac events was not significant (p=0.349 for BMS group and p=0.135 for DES group).

Conclusions: In our study searching for the relationship between stent type and systemic inflammatory response in 112 electively stented patients, we found similar amount of post procedure CRP increase in both BMS and DES groups. Also, periprocedural myonecrosis and adverse cardiac event rates were similar in both groups. In assessment of our results, we could relate the non-significant relationship of the level of inflammation and adverse cardiac events with the high degree of statin use, in about 90% of the patients.

As a result, we concluded that there was no difference at the level of systemic inflammation between BMS and DES groups, in consistency with most of the published trials in this topic. At similar levels of systemic inflammation, the local anti-inflammatory properties of DES can play a role at decreased restenosis rates.

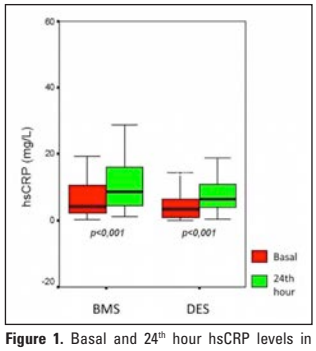

Figure 1. Basal and 24th hour hsCRP levels in BMS and DES groups.

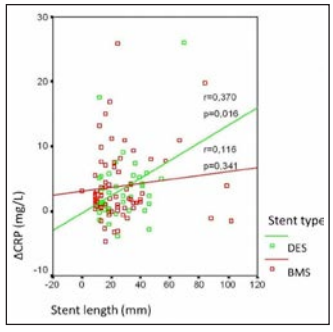

Figure 2. Correlation of stent length and ΔCRP in bare metal stent and drug eluting stent groups. (BMS = bare metal stent, DES = drug eluting stent).

Interventional cardiology / Coronary

OP-094

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 implantedwere evaluated in the study. Baseline characteristics, coronary risk factors and coronary angiography data assesed 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 familiy history rates were significantly different. More patients in the BMS group undervent PCI for STEMI (p=0.04). At 1-year follow up, there were less MACE rates int the DES group (p=0.011). There were no diference 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 superiorty with less MACE rates at 1-year follow up and safety with longterm lower mortalitiy.

Interventional cardiology / Coronary

OP-095

Is tirofiban used effectively in patients with acute coronary syndrome in daily practice?

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Background and Aim: To investigate the efficacy of intracoronary tirofiban during acute coronary syndrome (ACS) and then infusion for 18 hours following intracoronary bolus dose

Methods: A 52-year-old female patient was admitted to the our emergency service because of chest pain for the last 2 days. On electrocardiography (ECG) record, 1 mm ST segment depression was revealed in V5-V6 derivations. Her physical examination was unremarkable. Left ventricular ejection fraction (LVEF) was %60 on bedside transthoracic echocardiography (TTE). There was a history of stent implantation for LAD and RCA 3 months ago. The patient with high troponin values was hospitalized with the diagnosis of Non-ST-segment elevation myocardial infarction (NSTEMI). Diagnostic coronary angiography (CAG) was performed and thrombus image was observed causing a significant stenosis in Cx ostium (Figure 1). It was decided to perform tirofiban infusion for 18 hours following an intracoronary bolus dose in addition to subcutaneous low-molecular-weight heparin treatment and to perform control coronary angiography. In control CAG, the thrombus image was disappeared completely and the patient was discharged with dual antiplatelet therapy (DAPT).

Results: Acute coronary syndromes (ACS) is the leading cause of death in the world and it seems that aging of the population and the increased incidence of chronic diseases such as diabetes and obesity will increase the burden of atherosclerotic cronary artery disease (CAD) in the future. In most patients, coronary plaque rupture or erosion are the initiating events of ACS which expose the underlying endothelial surface to formed elements of circulating blood, leading to activation of platelets, thrombin generation and thrombus formation. The glycoprotein IIb/IIIa receptor plays a key role in platelet aggregation once it has been activated by specific ligands. The development of glycoprotein IIb/IIIa inhibitors as the most potent inhibitors of platelet aggregation has revolutionized the management of acute coronary syndromes. Tirofiban is one of three parenteral glycoprotein IIb/IIIa inhibitors in clinical use, and many trials have demonstrated its clinical efficacy and low rate of adverse effects in patients with non-ST-segment elevation acute coronary syndrome. According to the current guidelines, the administration of glycoprotein IIb/IIIa inhibitors concomitant with DAPT during invasive procedures should be restricted and may only be considered in specific 'bail-out' situations including high intraprocedural thrombus burden, slow flow, or no-flow with closure of the stented coronary vessel.

Conclusions: In this case report, we showed that administration of tirofiban for patients diagnosed with NSTEMI may have a role to reduce the burden of thrombus and also may prevent the unnecessary complex coronary interventions.

Hypertension

OP-096

Comparison of risk scoring systems for the development of long-term cardiovascular events in patients with essential hypertension

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Background and Aim: The prevalence of hypertension is 31.8%. 17 million people worldwide each year, and approximately 200,000 people living in Turkey is losing its reasons linked to heart and vascular disease. The aim of our study was to evaluate the efficacy of SCORE (Systemic Coronary Risk Evaluation) and PCRAE (Pooled Cohort Risk Assesment Equation) risk scoring systems in predicting long-term cardiovascular events in patients with essential hypertension

Methods: It will be determined whether cardiovascular events develop in patients diagnosed with essential hypertension in our center between 01/January/2009 - 01/January/2018. Long-term cardiovascular event risk rates will be calculated with SCORE and PCRAE systems based on the data of the patients on the date of diagnosis of hypertension. Patients will be divided into two groups in terms of the development of cardiovascular events, whether there is a significant difference between the two groups in terms of the risk rates calculated by SCORE and PCRAE risk score systems, and their value in predicting the event. By using ROC analysis, risk rates predicting the patients who are actually seen in the event will be found and it will be tried to determine which system is more successful in predicting cardiovascular events according to the size of the areas under the curve.

Results: The mean age of the 788 patients with essential hypertension was 53.65±9.08 and 426 (54.1%) female, and the mean follow-up period was 5.9±1.3 years. Our endpoint, major adverse cardiovascular event and cerebrovascular event (MACCE) was detected in 173 (22.0%) patients. In MACCE (+) patients, while these scores were significantly higher compared to patients who did not develop events (p<0.001), the prediction of MACCE viability of SCORE and PCRAE risk scoring systems was significant for both (p<0.001) in favor of PCRAE. The predictive value of the PCRAE risk scoring system in all parameters that constitute our study endpoint (ROC analysis AUC: 0.624 versus 0.724 for coronary artery disase (CAD), 0.762 versus 0.714 for stroke, 0.757 versus 0.757 for transient ischemic attack (TIA) and 0.632 for MACCE) It was found to be superior to the SCORE risk system. The predictive values for SCORE and PCRAE risk scoring systems for prediction of MACCE development did not make a significant difference in predicting cardiovascular (CV) death (p=0.184, p=0.082, respectively); The predictions of coroner artery disease (CAD), stroke, transient ischemic attack (TIA) and MACCE were significantly predictive (p<0.001).

Conclusions: The PCRAE risk scoring system is superior to the SCORE risk system in predicting cardiovascular and cerebrovascular events that are expected to occur in long-term follow-up in patients with essential primary hypertension.

Table 1. Demographic characteristics, laboratory findings and 24-hour blood pressure holter results of essential hypertension patients according to the presence of MACCE

N=788	MACCE (-) (n=615)	MACCE (+) (n=173)	P value
Age (years)	52.51±8.78	57.69±9.00	<0.001
Gender (male), n (%)	271 (44.1)	91 (52.6)	0.047
Body mass index, (kg / m²)	30.55±4.99	30.55±4.60	0.998
Smoking, n (%)	80 (21.1)	32 (28.3)	0.109
Alcohol, n (%)	19 (5.0)	9 (8.0)	0.237
Diabetes mellitus, n (%)	94 (16.0)	59 (34.3)	<0.001
Hyperlipidemia, n (%)	287 (48.7)	99 (60.7)	0.007
Blood pressure medication, n (%)	290 (75.7)	94 (81.0)	0.234
ACEi or ARB, n (%)	259 (67.6)	79 (68.1)	0.923
Calcium channel blocker, n (%)	146 (38.2)	62 (53.4)	0.004
Beta blocker, n (%)	111 (29.1)	56 (48.3)	<0.001
Diuretic, n (%)	186 (48.7)	67 (57.8)	0.087
Acetylsalicylic acid, n (%)	54 (14.1)	42 (36.2)	<0.001
Statin, n (%)	30 (7.9)	29 (25.0)	<0.001
Hemoglobin, g / dL	13.80±1.63	13.89±1.70	0.538
Leukocyte, 10 ^ 3 / uL	7.59±1.87	8.00±2.06	0.017
Platelet, 10 ^ 3 / uL	267.6±73.0	263.2±66.5	0.483
Creatine, mg / dL	0.80±0.20	0.86±0.23	<0.001
Glucose, mg / dL	98 (91-109)	107 (95-138)	<0.001
LDL-C, mg / dL	129.9±36.6	131.6±35.8	0.609
HDL-C, mg / dL	47.8±13.6	44.1±10.6	0.002
Total cholesterol, mg / dL	203.7±41.8	208.1±41.0	0.251
Triglyceride, mg / dL	133 (98-189)	159 (119-245)	<0.001
C-reactive protein, mg / L	2.8 (1.4-6.0)	3.7 (1.9-6.1)	0.141
Uric acid, mg / dL LVEF,%	5.34±1.40	5.78±1.39	0.002
LVEDd, mm	63.68±4.12	61.99±4.09	<0.001
LVESd, mm	47.19±4.24	47.63±4.77	0.275
IVS, mm	28.13±4.04	30.03±4.06	0.016
PWD, mm	11.09±1.82	11.99±2.41	<0.001
Heart rate, beats / min	75.45±13.40	75.30±14.23	0.919
24 hours SBP, mmHg	142.7±17.4	149.9±16.6	<0.001
24 hours DBP, mmHg	88.8±11.9	90.9±12.1	0.045
Daytime SBP, mmHg	144.8±17.5	151.5±16.9	<0.001
Daytime DBP, mmHg	91.1±12.2	92.8±12.5	0.112
Night SBP, mmHg	135.7±19.4	143.9±18.5	<0.001
Night DBP, mmHg	81.7±12.6	84.2±12.7	0.024
Non-dipper HT, n (%)	394 (67.7)	117 (71.8)	0.321
Opposite dipper HT, n (%)	94 (16.2)	43 (26.4)	0.003
Resistant HT, n (%)	87 (14.2)	48 (28.1)	<0.001
Isolated systolic HT, n (%)	34 (5.6)	18 (10.8)	0.019

MACCE: Major adverse cardiovascular and cerebrovascular event, ACEi: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, LVEF: Left ventricular ejection fraction, LVEDd: Left ventricular end diastolic diameter, LVESd: Left ventricular end systolic diameter, IVS: Interventricular septum, PWD: Diastolic posterior wall thickness, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, HT: Hypertension.

Table 2. Comparison of the groups according to the risk ratios obtained from the ESC and ACC / AHA risk scoring systems

N=788	MACCE (-) (n=615)	MACCE (+) (n=173)	P value
ESC, %	2.0 (1.0-4.0)	4.0 (2.0-7.0)	<0.001
ACC/AHA, %	5.8 (2.7-11.2)	13.3 (7.3-24.3)	<0.001

MACCE: Major adverse cardiovascular and cerebrovascular event, ESC: European society of cardiology, ACC / AHA: American college of cardiology / American heart association.

Table 3. Multivariate regression analysis with ESC and ACC / AHA models to predict long-term MACCE in patients with essential hypertension

	ESC OR (CI %95)	ESC P	ACC/AHA OR (CI %95)	AHA/ACC P
Age	1.094 (1.062-1.127)	<0.001	1.089 (1.051-1.128)	<0.001
Gender (male)	1.703 (1.020-2.845)	0.042	1.558 (0.884-2.747)	0.125
Cigaret	2.385 (1.314-4.327)	0.004	2.053 (1.098-3.836)	0.024
24 hours SBP	1.017 (1.003-1.031)	0.018	1.015 (0.989-1.043)	0.260
Total cholesterol	1.004 (0.998-1.010)	0.171	1.008 (1.001-1.014)	0.018
24 hours DBP			0.995 (0.956-1.036)	0.817
HDL-C			0.968 (0.943-0.994)	0.015
Diabetes mellitus			1.616 (0.892-2.928)	0.113
Antihypertensive drugs			1.171 (0.540-2.540)	0.689

ESC: European society of cardiology, ACC / AHA: American college of cardiology / American heart association, OR: odds ratio, CI: confidence interval, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, HDL-C: High density lipoprotein cholesterol.

Table 4. Values obtained by multivariate regression analysis using ESC and ACC / AHA models

	-2 Log likelihood	Cox & Snell R2	Nagelkerke R2	Model P value
ESC	393.310	0.115	0.177	<0.001
ACC/AHA	367.156	0.136	0.205	<0.001

ESC: European society of cardiology, ACC / AHA: American college of cardiology / American heart association.

Table 5. Areas under the curve obtained with ESC and ACC / AHA risk ratios for the detection of endpoints

	ESC AUC (CI %95)	ESC P	ACC/AHA AUC (CI %95)	ACC/AHA P
Coronary artery disease	0.688 (0.640-0.736)	<0.001	0.724 (0.680-0.769)	<0.001
Stroke	0.662 (0.571-0.752)	0.002	0.714 (627-0.800)	<0.001
Transient ischemic attack	0.690 (0.526-0.853)	0.015	0.757 (0.640-0.875)	0.001
MACCE	0.689 (0.644-0.735)	<0.001	0.732 (0.691-0.773)	<0.001

ESC: European society of cardiology, ACC / AHA: American college of cardiology / American heart association, OR: odds ratio, CI: confidence interval, MACCE: Major adverse cardiovascular and cerebrovascular event.

Table 6. Diagnostic powers of risk scoring systems according to the best estimation values

	Sensitivity	Specificity	PPV	NPV	Accuracy
ESC (>3.50)	%58.7	%72.5	%62.6	%86.3	%69.5
ACC/AHA (>9.45)	%69.4	%68.0	%62.1	%88.7	%68.3

PPV: positive predictive value, NPD: negative predictive value, ESC: European society of cardiology, ACC / AHA: American college of cardiology / American heart association.

Table 7. Correlation of risk scores with other variables

	ESC r	ESC p	ACC/AHA r	ACC/AHA p
Age	0.708	<0.001	0.669	<0.001
Body mass index	-0.064	0.156	-0.009	0.835
LVEF	-0.186	<0.001	-0.215	<0.001
LVEDd	0.098	0.010	0.055	0.151
LVESd	0.103	0.007	0.082	0.032
IVS	0.282	<0.001	0.329	<0.001
PWD	0.298	<0.001	0.330	<0.001
Heart rate	-0.095	0.045	-0.020	0.665
24 hours SBP	0.332	<0.001	0.368	<0.001
24 hours DBP	0.156	<0.001	0.159	<0.001
Daytime SBP	0.318	<0.001	0.353	<0.001
Daytime DBP	0.133	<0.001	0.141	<0.001
Night SBP	0.343	<0.001	0.381	<0.001
Night DBP	0.208	<0.001	0.204	<0.001
Hemoglobin	0.268	<0.001	0.212	<0.001
Leukocyte	0.022	0.543	0.116	0.002
platelets	-0.147	<0.001	-0.084	0.022
Creatine	0.329	<0.001	0.281	<0.001
glucose	0.180	<0.001	0.307	<0.001
LDL-C	0.092	0.014	0.124	<0.001
HDL-C	-0.077	0.037	-0.271	<0.001
Total cholesterol	0.078	0.042	0.110	0.004
triglycerides	0.132	<0.001	0.276	<0.001
C-reactive protein	0.054	0.279	0.141	0.005
Uric acid	0.270	<0.001	0.292	<0.001
ESC			0.810	<0.001
ACC / AHA	0.810	<0.001		

ESC: European society of cardiology, ACC / AHA: American college of cardiology / American heart association, LVEF: Left ventricular ejection fraction, LVEDd: Left ventricular end diastolic diameter, LVESd: Left ventricular end systolic diameter, IVS: Interventricular septum, PWD: Diastolic posterior wall thickness, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol.

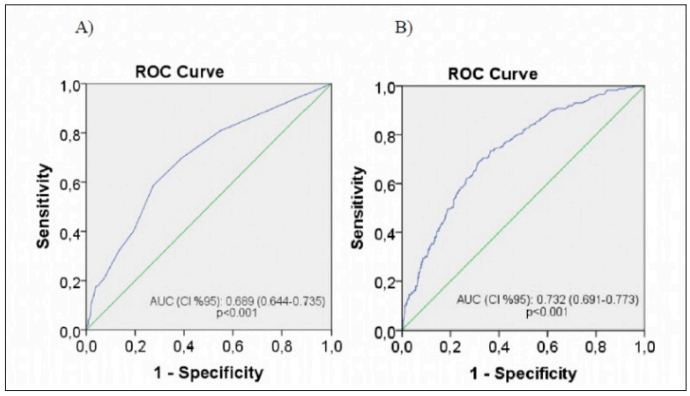

Figure 1. ROC curves for risk ratios obtained with ESC **(a)** and ACC / AHA **(b)** risk scoring systems for the detection of the presence of MACCE.

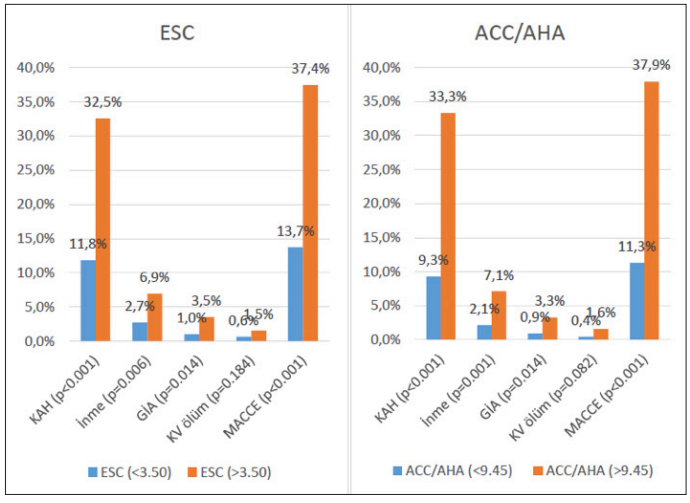

Figure 2. Comparison of risk scoring systems in terms of patients' endpoints according to the best estimation values.

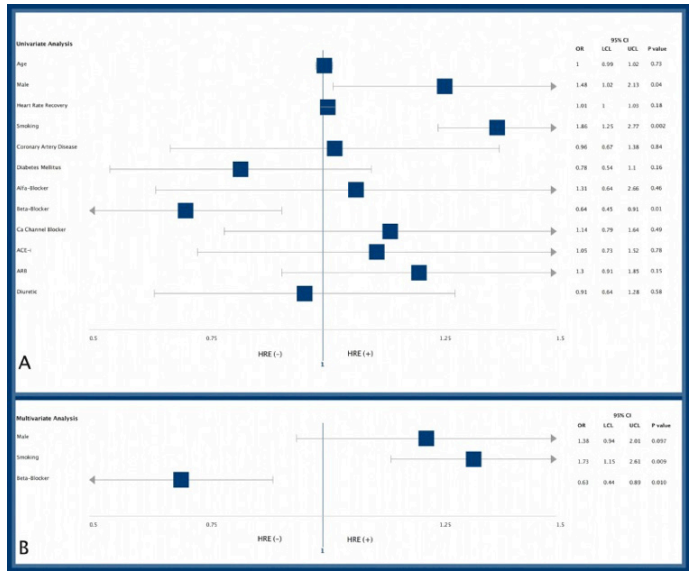

Figure 1. Predicted probability of Hypertensive Response to Exercise (HRE) in univariate and multivariate logistic regression analysis. HRE: Hypertensive response to exercise.

Table 1. The demographics and medications among HRE in patients with HT who underwent exercise stress testing

Speciality	Family Medicine / General practitioner	Internal medicine	Cardiology	p value
Confidence level of ACE-i/ARBs	5.0 ±3	8.0± 4	8.0 ±4	0,02a
General confidence	9.0 ± 5	9.0±2	9.0±6	0,31
Repeating ACE/ARB	8.0±4	8.0±4	9.0±3	0,009a,b
Starting New ACE/ARB				
Changing prescribing pattern in different clinical conditions	2.0±4	2.0±2	1.0±2	0,09
General	1.0±3	2.0±2	2.0±3	0,8
Hypertension	1.5±3	1.0±2	1.0±0	0,005a,b
Heart failure	1.5±2	1.0±1	1.0±1	0,2
Coronary artery disease	2.0±4	2.0±2	1.0±2	0,1
Multipl comorbidities				

HRE: Hypertensive response to exercise.

Hypertension

OP-097

The blunting effect of antihypertensives on exaggerated blood pressure response to exercise

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Background and Aim: An elevation in blood pressure (BP) during exercise is the normal physiological response, however the abnormally exaggerated rise in BP, in terms of hypertensive response to exercise (HRE), is encountered as a prognostic factor for end-organ damage and mortality. HRE is more common in patients with hypertension (HT). There is a lack of data on the effect of antihypertensive medication on the HRE. In this study, we evaluated patients who underwent exercise stress testing to reveal the effect of antihypertensive medications on the HRE.

Methods: A cohort of 992 patients with HT undergoing treadmill exercise test (TET) were screened and data was evaluated whether an HRE was developed. An HRE has been defined as an exceeding a systolic BP >210 mmHg in males and >190 mmHg in females or a difference between peak and baseline systolic BP at least 50 mmHg in females and 60 mmHg in males throughout the TET. Analysis were performed to evaluate the association between HRE and antihypertensives.

Results: HRE was observed in 15% (n=149) of patients with HT. An HRE was observed significantly more in males (57.3% vs 66.4%; p=0.038), smokers (17.4% vs 28.2%; p=0.003). There was not any significant association between medications and an HRE development, except beta-blockers. Also, an HRE was significantly lower by beta-blocker monotherapy rather than the other monotherapies in patients with HT. In multivariate analysis smoking status (odds ratio: 1.728, 95% CI: 1.146–2.011, p=0.009) and being under beta-blocker treatment (odds ratio: 0.628, 95% CI: 0.441–0.893, p=0.010) were found to be the independent predictors of an HRE.

Conclusions: Beta-blocker based treatments, whether mono- or combination therapy in patients with HT, may be a significantly good preventive strategy for an HRE. Besides the beneficial effect of beta blockers in preventing HRE, they may increase exercise tolerance, reduce morbidity and mortality in hypertensive patients.

Hypertension

OP-098

Relationship between the presence of fragmented QRS and microalbuminuria in newly diagnosed hypertension patients

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Background and Aim: The fragmented QRS complex (fQRS) is closely related to hypertension(HT). Many studies have reported presence of fQRS was associated with increased incidence of poor outcomes in HT patients. Although there were clinical studies investigating the association of fQRS with target organ damage in patients with HT, the relationship between microalbuminuria (MAU), which is the first marker of development of target organ damage in kidney, and fQRS has never been studied in HT patients. This study aims to investigate the relationship between the presence of fQRS and MAU in newly diagnosed HT patients.

Methods: We included 246 newly diagnosed HT patients (age: 53.4±9.6 years, 62.5% male) who applied to the outpatient clinic in a consecutive study. fQRS was defined as additional R' wave or notching/splitting of S wave in two contiguous ECG leads. Patients with HT were divided into two groups according to the presence of fQRS: absence of fQRS in any lead, and presence of fQRS in two or more contiguous leads. Spot urine sample was collected for the assessment of MA. Then, appropriate statistical tests were carried out.

Results: The basal characteristics of both groups were similar. In this study, incidence of fQRS was 32.1% (n=80) in hypertensive patients. The presence of MAU was significantly higher in the fQRS (+) group (p<0.001). The presence of fQRS (OR: 3.567, 95%CI: 1.934 to 5.267; p<0.001) and left ventricular mass index (OR: 1.49, 95% CI: 1.27-1.98; p=0.003) were found as independent predictors of MAU. Additionally, there was a positive correlation between left ventricular mass index and the presence of fQRS (r=0.481, p<0.001).

Conclusions: In conclusion, presence of fQRS complex on standard 12-lead ECG predicts MAU in new diagnosly hypertansion patients. We think that further renal evaluation will be appropriate in fQRS (+) HT patients. So, large scale and prospective studies are neededed to confirm those findings.

Hypertension

OP-099

Relation between fragmented QRS complex and arterial stfness in asymptomatic subjects

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Background and Aim: Stiffness of large arteries has been related to cardiovascular mortality. Cardio-ankle vascular index (CAVI) is a novel marker of arterial stiffness. Herein, we aimed to study the relationship between fragmented QRS (fQRS) in electrocardiogram and arterial stiffness.
Methods: Patients admitted to the cardiology outpatient clinic with and without fQRS were consecutively enrolled. The fQRS complexes were looked for in the 12-lead electrocardiogram. Arterial stiffness was assessed by cardioankle vascular index (CAVI). It was measured using a VaSera VS-1000 CAVI instrument.
Results: CAVI of the patients with fQRS was significantly higher (8.625 (7.9-9.2) versus 6.65 (6.7-8.4) p=0.000). In a univariate analysis it was revealed that there existed a significant correlation between increased CAVI and fQRS, age, and epicedial fat thıckness. Multiple binary logistic regression analysis revealed that age [95% confidence interval (CI): 1.068-1.214, p=0.000] and fQRS [95% (CI): 1.766-23.117, p=0.005] were the independent determinants of increased arterial stifness.
Conclusions: fQRS in electrocardiogram may provide a significant predictive value for arterial stiffness in asymptomatic subjects.

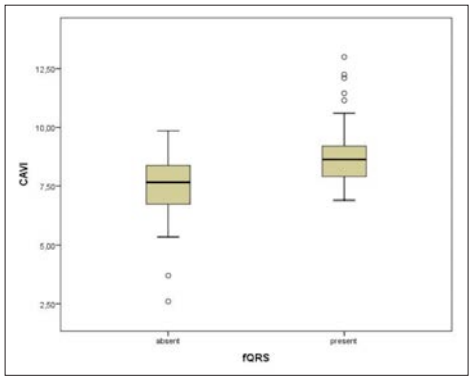

Figure 1. CAVI values in patients of fQRS- absent and fQRS - positive subjects. CAVI: Cardio-ankle vascular index.

Table 1. Clinical and laboratory charactheristics of subjects

Variables	fQRS (-) N=72	fQRS (+) N=72	P
Age, years	49.5(44-58.5)	55(49-62.75)	0.004
Male gender, n(%)	34 (47.2)	35 (48.6)	1
Hypertension, n(%)	29(40.3)	33(45.8)	0.61
Diabet, n (%)	6 (7.6)	3 (3.8)	0.32
Dyslipidemia, n(%)	3 (4.2)	8(11.1)	0.20
Smoking, n(%)	6(8.3)	13(18.1)	0.13
LDL(mg/dl)	134(109.5-148.5)	129(113-152)	0.92
HDL(mg/dl)	48.7±11.4	46.8±11.4	0.34
Triglyceride (mg/dl)	144(90-185)	139(94-233)	0.33
Cholesterol (mg/dl)	204(180-229)	208(186-233)	0.61
CAVI	6.65(6.7-8.4)	8.625(7.9-9.2)	0.000
BMI	24.8(21.3-31.1)	26.9(21.4-33.8)	0.14
EFT	4(2-5)	5(3-7)	0.001
Cardiovascular medication			
ACE inhibitors, ARB, n(%)	15 (20.8)	16 (22.2)	1.0
Calcium channel blokers, n(%)	11 (15.3)	10(13.9)	1.0
Beta Bloker, n (%)	6(8.3)	2 (2.8)	0.27

ACE, angiotensin converting enzyme; ARB, angiotensin reseptor blocker; BMI, body mass index; EFT, epicardial fat thickness; CAVI,Cardio-ankle vascular index

Table 2. Conditional logistic regression analysis showing independent factors associated with anormal CAVI (CAVI>9)

Variables*	CAVI < 9	CAV≥ 9	OR (95 % CI)	P
Age	50(45-55)	63.5(58.5-72.25)	1.139 (1,068-1.214)	0.000
fQRS, n (%)	43(39.8)	29(80.6)	6.38 (1.766-23.117)	0.005

**The covariates included Age,presence of hypertension, presence of Fqrs, supramedian epicardial fat thickness.*

Hypertension

OP-100

Impact of a new hypothesis regarding angiotensin-converting enzyme 2 (ACE2) receptor on hypertension treatment during COVID-19 pandemic: Physician's perspective

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Background and Aim: A debate is raging about ACE inhibitors (ACE-i)/ angiotensin receptor blockers (ARBs) and SARS-CoV-2 due to the concern that these agents may upregulate ACE2 expression. This study was designed to evaluate thoughts and applications of physicians related to hypertension treatment with ACE-i or ARBs during the COVID-19 pandemic.
Methods: This study was a cross-sectional survey which included cardiologists, internists, and family physicians that are responsible for the treatment of hypertension or prescribing antihypertensive drugs. The study was designed in six different metropolitan (Izmir, Ankara, Kocaeli, Manisa, Balıkesir, Konya) located in central and western Turkey, between 1 May-19 May 2020. The hospitals or family health centers were randomly selected in the above-mentioned cities where were intensified for COVID-19 patients. The survey consisted of 26 questions about current hypertension treatment behavior with ACE-i and ARBs during the COVID-19 pandemic.
Results: A total of 460 physicians were approached, and 220 (47.8%) physicians give permission to participate in the study; 142 (65%) were male, and 13 % above 50 years. Half of the physicians (50.4%) were working in tertiary health centers (training and research hospitals University hospitals). Of the physicians, 40% were working in the specialty of cardiology, 32% in family medicine /general practitioners, and 28% in internal medicine. Most of the clinicians (79%) have not changed their antihypertensive medication prescribing pattern; only 8.5% of clinicians changed ACE-i/ ARBs medicine of patients during the COVID-19 pandemic. The median (±interquartile range) score indicating general confidence of clinicians to ACE-i/ARBs therapy was 8±4 (range of 1-10). In multiple comparison analyses; the general confidence level of ACE-i/ARBs, confidence level when starting a new ACEi/ARBs and changing behavior in heart failure patients were significantly different with regard to the specialties (p; 0.02, 0.009, 0.005 respectively)
Conclusions: The present survey was the first study that provides a snapshot showing the prescribing practice of Turkish clinicians on pandemic episode. This study revealed that the majority of physicians was not affected by the existing concerns and continues to prescribing ACE-i/ARBs treatments confidentially. However, there was a statistically difference in confidence level in different clinical situations among different physician specialties. Given the probability of emerging new evidence on this manner, additional studies should be conducted to clarify the prescribing habitual changes of physicians on antihypertensive medications in the course of COVID-19 disease.

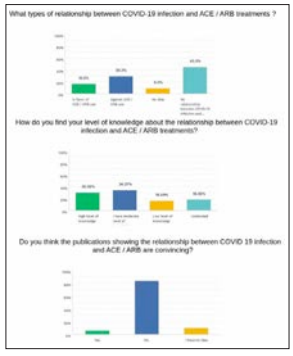

Figure 1. Answers of the clinicians to level of knowledge questions about the relationship between ACE/ARB treatment and Covid-19.

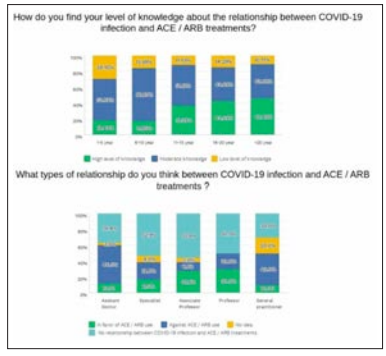

Figure 2. Effect of the experience on level of knowledge of the physicians.

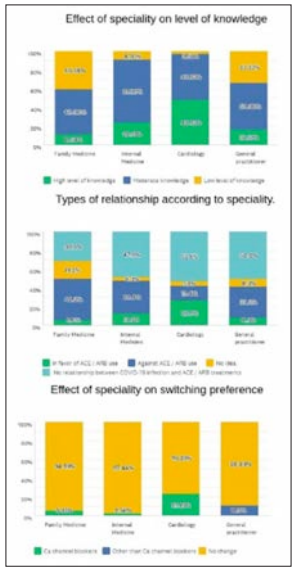

Figure 3. Effects of physicians' specialties on level of knowledge and treatment preferences.

Table 1. Characteristics of the physicians participating in the survey

	n	%
Gender (Male/Female)	142/78	64.5%/35.5%
Age		
20-29	39	17.7%
30-39	100	45.4%
40-49	53	24.1%
≥50	28	12.8%
Specialty		
Family medicine /General practitioner	71	32.3%
Internal medicine	61	27.7%
Cardiology	88	40%
Seniority		
Assistant /general practitioner	83	37.7%
Specialist	97	44.1%
Assoc Prof./Prof.	40	18.2%
Duration of practice		
1-10 years	104	47.3
11-20 years	77	35
>20 years	39	17.7
Hospital		
Primary care hospital	56	25.5%
Secondary care hospital	53	24.1%
Tertiary hospital	111	50.4%

Table 2. Effect of specialty on confidence level of ACE-i/ARBs and changing behavior of prescribing pattern in different clinical conditions

Speciality	Family Medicine / General practitioner	Internal medicine	Cardiology	p value
Confidence level of ACE-i/ARBs	5.0 ±3	8.0± 4	8.0 ±4	0,02a
General confidence	9.0 ± 5	9.0±2	9.0±6	0,31
Repeating ACE/ARB	8.0±4	8.0±4	9.0±3	0,009a,b
Starting New ACE/ARB				
Changing prescribing pattern in different clinical conditions				
General	2.0±4	2.0±2	1.0±2	0,09
Hypertension	1.0±3	2.0±2	2.0±3	0,8
Heart failure	1.5±3	1.0±2	1.0±0	0,005a,b
Coronary artery disease	1.5±2	1.0±1	1.0±1	0,2
Multipl comorbidities	2.0±4	2.0±2	1.0±2	0,1

The data are expressed as median ± interquartile range a: Between Family Medicine / General practitioner and Cardiology doctors p<0.05 b: Between Internal medicine and Cardiology doctors p<0.05.

Hypertension

OP-101

Evaluation of the relationship between short-term blood pressure variability and diastolic dysfunction

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Background and Aim: Diastolic dysfunction may be completely asymptomatic, as well as showing signs and symptoms of decompensated heart failure. Studies have shown that the increase in short and long term fluctuations in blood pressure is associated with poor prognosis for target organ damage. Our aim in this study is to investigate the effect of short-term blood pressure variability (BPV) on the development of diastolic dysfunction (DD).

Methods: The study group consisted of 79 patients aged 18 to 70 years who were decided to undergo coronary angiography with any indication and who were treated by using radial approach in the Cardiology Department of Erciyes University Faculty of Medicine from October 2019 to March 2020. Patients with coronary artery disease, low ejection fraction, moderate to severe valve disease and pronounced arrhythmia were excluded. After performing transthoracic echocardiography on the patients involved, the diastolic parameters were examined in detail and they were divided into two groups as 45 patients with DD and 34 patients without DD. 2-minute intra-arterial blood pressure recordings from both the central and periphery were taken from the patients, and then 24-hour ambulatory blood pressure was monitored. In this way, beat-to-beat and 24-hour BPV were examined. Arithmetic real variability (ARV) and standard deviation (SD) values were calculated individiually for systole and diastole from the measurements obtained to evaluate BPV. Statistical analyses were performmed by using SPSS.

Results: The average age of the group without DD was 46.91±9.09 and the average age of the group with DD was 59.98±7.34 (p<0.001). The incidence of DD in female gender was significantly higher than male gender (p=0.001). The frequency of HT was significantly higher in the DD group compared to the non-DD group (p=0.001). Intra-arterial and ambulatory BPV parameters were significantly higher in patients with DD (p<0.001). It was determined that the height in the BPV parameters correlated with the decrease in the E/A ratio and the increase in the average E/e´ ratio. In the multivariate analysis, it was observed that especially the central systolic beat-to-beat and 24-hour systolic BPV values were significantly higher in the DD developing group (p=0.02 and p=0.017 respectively). In the subgroup analysis of hypertensive and normotensive patients, the effect of short-term elevation of BPV on DD development in both groups was statistically significant.

Conclusions: It was shown that the elevation in short-term BPV values, especially from central beat-to-beat and ambulatory systolic BPV parameters, may cause DD development in both hypertensive and normotensive patients.

Table 1. Multivariant analyses of central BPV

Variables	Unadjusted OR	95% CI	P value	Adjusted OR	95% CI	P value
Age	1,309	1,019-1,483	<0,001	1,143	0,999-1,309	0,052
BMI	1,062	0,964-1,170	0,226			
HT	0,207	0,079-0,542	0,001	0,915	0,089-9,396	0,94
DM	0,596	0,208-1,706	0,335			
Smoking	2,348	0,833-6,618	0,106			
Sex	0,198	0,075-0,525	0,001	1,856	0,069-5,160	0,713
HGB	0,668	0,490-0,911	0,011	0,579	0,247-1,354	0,207
WBC	0,370	0,356-0,577	0,911			
sBPV central ARV	1,170	1,096-1,250	<0,001	1,099	1,015-1,190	0,02
dBPV central ARV	1,154	1,085-1,226	<0,001	1,108	1,001-1,227	0,049

s: systolic, d: diastolic, ARV: arithmetic real variability, BMI: body mass index, HT: hypertension, DM: diabetes mellitus, HGB: hemoglobin, WBC: white blood cell

Table 2. Multivariant analyses of ambulatory BPV

Variables	Unadjusted OR	95% CI	P value	Adjusted OR	95% CI	P value
Age	1,309	1,019-1,483	<0,001	1,225	1,080-1,389	0,002
BMI	1,062	0,964-1,170	0,226			
HT	0,207	0,079-0,542	0,001	1,048	0,197-5,582	0,956
DM	0,596	0,208-1,706	0,335			
Smoking	2,348	0,833-6,618	0,106			
Sex	0,198	0,075-0,525	0,001	1,533	0,135-17,454	0,731
HGB	0,668	0,490-0,911	0,011	0,634	0,309-1,302	0,215
WBC	0,370	0,356-0,577	0,911			
sBPV amb ARV	2,467	1,680-3,623	<0,001	1,008	1,001-1,015	0,017
dBPV amb ARV	2,004	1,407-2,855	<0,001	1,003	0,995-1,011	0,439

s: systolic, d: diastolic, ARV: arithmetic real variability, BMI: body mass index, HT: hypertension, DM: diabetes mellitus, HGB: hemoglobin, WBC: white blood cell

Hypertension

OP-102

The relationship between mean platelet volume and reverse dipping blood pressure pattern in patients with essential hypertension

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Background and Aim: An association between increased mean platelet volume (MPV) and fatal and nonfatal cardiovascular events is well established. Recent studies suggest that nondipper blood pressure pattern is closely related to increased MPV. However little information has been revealed about the relationship between reverse dipper hypertension (RDHT) and MPV. Our aim was to investigate the relation between MPV and reverse-dipping blood pressure pattern in essential hypertension.

Methods: Ambulatory blood pressure monitoring (ABPM) records of a total of 317 patients were retrospectively evaluated between December 2019 and May 2020. Patients were categotized into 3 groups according to their ABPM values as RDHT (n=63), non-dipper hypertension (NDHT) (n=95), and dipper hypertension (DHT) groups (n=159). MPV and biochemical analyses were recorded from hospital database. The value of MPV was determined by automated hematology analyzer.

Results: The MPV levels were significantly higher in RDHT than in the NDHT and DHT groups and were higher in the NDHT than DHT group (9.1±0.4 fl, 8.8±0.5 fl, and 8.6±0.6 fl, respectively, for all p<0.05). There was a positive correlation between MPV and average 24-hour systolic blood pressure (r=0.383; p=0.001), average 24-hour diastolic blood pressure (r=0.205; p=0.001), average daytime systolic blood pressure (r=0.195; p=0.001) and average nighttime systolic blood pressure (r=0.391; p=0.001) and average nighttime diastolic blood pressure (r=0.335; p=0.001) In multivariate logistic regression analysis, MPV (OR 1.761, 95% CI 1.329 to 2.334, p=0.001) and age (OR 1.065, 95% CI 1.019 to 1.113, p=0.001) were detected as predictors for reverse dipper BP pattern. ROC curve analysis of MPV for prediction of reverse dipper hypertension showed that at the cut-off value of >9.1 fl, sensitivity and specificity of MPV 60% and, 69% respectively (AUC=0.696±0.035, 95% CI: 0.627–0.764).

Conclusions: Our data shows that increased MPV might be associated with the reverse dipper BP.

Hypertension

OP-103

The relationship between beta blocker types and exaggerated blood pressure response to exercise

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Background and Aim: If blood pressure (BP) is not offset during exercise, a sharp rise in systolic BP may be a result, in terms of exaggerated BP response to exercise (eBPR). There is paucity of data on whether different β-blockers show a significant effect on the management of eBPR. In this study, we aimed to evaluate the effect of cardio-selective β-blockers comparatively on the development of eBPR.
Methods: In total, 2755 individuals undergoing a treadmill exercise test in our retrospective cohort were included in the analysis. eBPR has been defined as a systolic BP exceeding the 90th percentile (BP >210 mmHg in males and >190 mmHg in females) or a difference between the peak and baseline systolic BP of at least 50 mmHg in females and 60 mmHg in males throughout the TET. To reveal the effects of β-blocker treatment and the type of β-blocker on eBPR, results were comparatively evaluated.
Results: eBPR was seen in 243 (14.1%) patients who did not take beta-blockers and 116 (10.9%) patients who received beta-blockers (p=0.007). There was no statistically significant association between the type of β-blocker (Metoprolol, bisoprolol and nebivolol) and eBPR development (respectively; 84/11.4%, 12/9.8%, 19/9.7%; p=0.742).
Conclusions: The eBPR is literally about endothelial dysfunction and arterial stiffness; it needs to be controlled by β-blockers to decrease mortality and morbidity, yet it is not a treatment target. The favourable effects of avoiding eBPR development were similar by either metoprolol or bisoprolol or nebivolol.

Table 1. The demographics, stress test parameters and medications among the type of β-blocker who underwent exercise stress testing

	Metoprolol n=740	Bisoprolol n=133	Nebivolol n=195	P value
Stress Test Parameters				
Target HR (bpm)	159.44±11.0	158.89±11.6	159.29±11.2	0.811
Max SBP (mmHg)	155.0±21.9	153.61±20.7	155.69±22.2	0.482
Max DBP (mmHg)	83.20±34.7	82.99±7.12	83.22±9.5	0.055
eBPR	84/11.4	12/9.8	19/9.7	0.742
METs	9.01±6.1	9.07±4.8	9.1±2.9	0.293
Demographics				
Age (years)	56.42±10.2	56.26±10.5	55.02±9.8	0.161
Male, n/%	527/71.2	85/63.9	106/54.9	<0.001
HT, n/%	325/43.9	58/43.6	115/59.0	0.001
DM, n/%	257/34.7	48/36.1	79/40.5	0.326
CHD, n/%	457/61.8	72/54.1	58/29.7	<0.001
Heart Failure, n/%	101/13.6	10/7.5	4/2.1	<0.001
Smoking, n/%	143/19.3	23/17.3	25/12.8	0.106
Medications				
Diuretics, n/%	315/42.6	45/33.8	87/44.6	0.118
Doksazosine,n/%	27/3.6	6/4.5	8/4.1	0.414
Propofenon, n/%	1/0.1	1/0.8	1/0.5	0.370
Amiodarone, n/%	19/2.6	5/3.8	1/0.5	0.123
Ca channel blocker, n/%	127/17.2	20/15.0	33/16.4	0.641
ACE-I, n/%	277/37.4	43/32.3	58/29.7	0.100
ARB, n/%	168/22.7	31/23.3	58/29.7	0.120

Hypertension

OP-104

Smoking status was associated with blood pressure increase during exercise in patients with hypertension

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Background and Aim: Activation in sympathetic nerve activity during exercise results increase in cardiac output to meet the needs of active muscularity with more oxygenated blood. The increase in cardiac output is more dominant when compared with the reduction of vascular resistance. Diastolic blood pressure (BP) does not change or decreases slightly, while systolic BP is expected to increase with increasing exercise intensity on the treadmill exercise test (TET). The cardiovascular risk of a patient with hypertension (HT) cannot be entirely affirmed by measuring the BP at rest while sitting in a chair. In this study, we aimed to reveal the explanatory variables to predict the increase in systolic BP in patients with HT.
Methods: In total, 1055 consecutive patients with HT undergoing a treadmill exercise test (TET) in two years were screened. Because of lack of data 62 were excluded and of the remaining 993 individuals were included in the analysis. A difference between the peak and baseline systolic BP throughout the TET was calculated. To reveal the effects explanatory variables to predict the increase in systolic BP multiple linear regression analysis were performed.
Results: Of the 993 patients with HT 190 (19.2%) were current smoker. Current smokers were leaner (body mass index: 29.4±4.5 vs 28.5±4.8; p=0.002) and males were more prone to smoke (53.7% vs 80.0%; <0.001). Increase in BP was higher in smokers (20.1±1.69 vs 25.1±1.86; p<0.001). Multiple linear regression analysis to predict BP increase including body mass index, age, gender, diabetes mellitus and smoking status was demonstrated in table 1. There was no collinearity problem and BP increase were only associated with smoking status (p=0.004).
Conclusions: Smoking status was an independent predictor of BP increase during exercise in patients with HT. Activation in sympathetic nerve system is predominant pathophysiological mechanism of an abnormally BP response to exercise. When diabetes, age, sex, body mass index and smoking status; that are thought to affect sympathetic nerve activity; were evaluated, BP increase during exercise was only associated with smoking status in patients with HT. Smoking cessation strategies should be key-stone for the management of hypertensives.

Table 1. Multiple linear regression analysis to predict BP increase during exercise

	Odds ratio	Lower (95% C.I.)	Upper (95% C.I.)	p value
Body mass index	0.117	-0.130	0.365	0.351
Age	0.044	-0.069	0.157	0.443
Male gender	2.09	-0.200	4.379	0.074
Diabetes Mellitus	-1.371	-3.573	0.830	0.222
Current Smoker	4.273	1.408	7.138	0.004

Hypertension

OP-105

The relationship between the desired blood pressure level and anxiety in hypertensive patients with COVID-19

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Background and Aim: The aim of the study is to evaluate the relationship between anxiety and hypertension control in patients with COVID-19 diagnosed with hypertension.
Methods: Eighty patients with hypertension who were admitted to our clinic with the diagnosis of COVID-19 were evaluated. Routine blood pressure follow-up of inpatients was performed twice a day. Blood pressure medications used by the patients were ordered. Demographic and laboratory features were recorded. The anxiety of the patients was evaluated with the Beck Anxiety Inventory (BAI).
Results: During follow-up, 56 patients (group 1) had their blood pressure under control, while 24 patients (group 2) had uncontrolled blood pressure. When groups with and without blood pressure are compared; the beck anxiety index of the group whose blood pressure was not under control was found to be significantly higher (79% vs 55%, p=0.03). There was no difference in terms of other parameters (Table 1).
Conclusions: Increased anxiety in hypertensive COVID-19 patients has been found to cause uncontrolled hypertension. The demographic and biochemical characteristics of the patients did not make a difference in terms of blood pressure control.

Table 1. Demographic, clinical and socioeconomic characteristics of the study patients

	Overall patients n= 80	Regular Blood pressure n= 56	Irregular Blood pressure n= 24	P value
Age, (years)	64±16	64±17	64±14	0.96
Female, n (%)	50 (63)	33 (59)	17 (71)	0.45
History of CAD, n (%)	15 (19)	9 (16)	6 (25)	0.53
Diabetes mellitus, n (%)	34 (43)	23 (41)	11 (46)	0.69
ACEI/ARB, n (%)	46 (57)	30 (54)	16 (67)	0.33
Smoking, n (%)	14 (18)	11 (20)	3 (12)	0.65
COPD, n (%)	5 (6)	3 (5)	2 (8)	0.63
Married, n (%)	67 (84)	45 (80)	22 (92)	0.32
Number of child	4±3	4±3	5±3	0.20
Education level, n (%) Primary Secondary High school University	27 (34) 42 (53) 7 (9) 4 (5)	17 (30) 30 (54) 6 (11) 3 (5)	10 (42) 12 (50) 1 (4) 1 (4)	0.67
Income level, n (%) * Low Low-intermediate İntermediate-high	42 (53) 30 (37) 8 (10)	28 (50) 22 (39) 6 (11)	14 (58) 8 (33) 2 (8)	0.79
Anxiety, n	13.7±10.9	12.8±10.9	15.8±10.9	0.26
BAI ≥ 10, n (%)	50 (63)	31 (55)	19 (79)	0.03

*ACEI: Angiotensin converting enzyme inhibitor; ARB:angiotensin receptor blocker; BAI: Beck anxiety inventory; CAD: Coronary artery disease; COPD: Chronic obstructive pulmonary disease * Income level (Turkish Lira) <2000, low; 2000-4000, low-intermediate; 4000-10000, intermediate-high; (1 Turkish Lira = 0.13 Euro and 0.15 dollar).*

Hypertension

OP-106

Association of vitamin-D deficiency with arterial stiffness in newly diagnosed hypertension

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Background and Aim: Vitamin D has beneficial effects on vascular endothelial function, blood pressure and arterial stiffness. Arterial stiffness increases in early stage hypertensive patients (HTP) and it is a strong predictor of cardiovascular morbidity and mortality. The purpose of this study was to assess the association between serum 25-hydroxyvitamin D (25-OH D) leves and arterial stiffness in patients with newly diagnosed hypertension.
Methods: Our study included 100 newly diagnosed HTP (63 male, 37 female and mean age:51.7 ± 10 years) without cardiovasculer disease, malignancy, chronic kidney disease and diabetes mellitus. Patients were divided into two groups: vitamin D deficiency group (<20 ng/ml) and normal vitamin D group (≥20 ng/ml). 24-hour, daytime and night-time ambulatory blood pressure (BP) readings were recorded. Mobil-O-Graph® ARC solver algorithm was used to evaluate arterial stiffness parameters of pulse wave velocity (PWV), augmentation index normalized with 75/min heart rate (Alx@75).
Results: Patients with vitamin D deficency had higher values of Alx@75 and PWV values (20.9±9 vs.16.8±6.9, p=0.018; 8.37±1.16 vs. 6.9±0.9, p=0.001, respectively) despite similar 24-hour ambulatory BP monitoring in both groups. Level of serum calcium was significantly higher in vitamin D deficiency group (9.5±0.23 vs. 9.3±0.12, p=0.007). Night-time systolic BP was higher in vitamin D deficiency group (133±14 mmHg vs. 126±17 mmHg; p=0.03) and also, vitamin D deficiency group had non-dipping systolic BP pattern compared to normal Vitamin D group.
Conclusions: Vitamin D deficiency is associated with increased arterial stiffness in newly diagnosed HTP in terms of increased PWV and Alx@75 values.

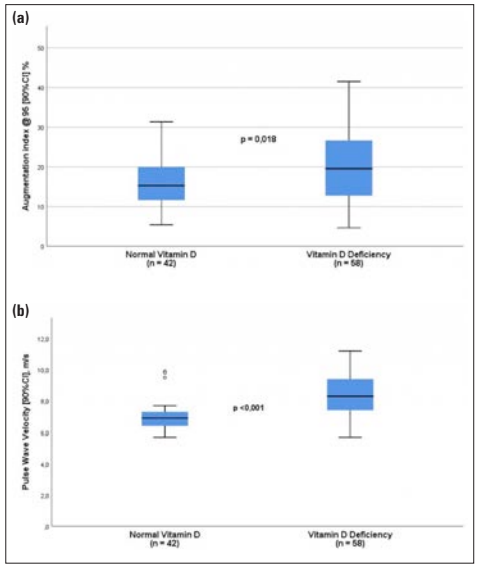

Figure 1. (a) Augmentation Index between normal Vitamin D and Vitamin D deficient patients. **(b)** Pulse wave velocity between normal Vitamin D and Vitamin D deficient patients

Table 1. Comparison of arterial stiffness and blood pressure parameters of groups

Characteristic	Total (n=100)	Vitamin D Deficiency (n=58)	Normal Vitamin D (n=42)	p
Pulse Wave Velocity [90%Cl] m/s	7.7 ±1.2	8.37±1.16	6.9±0.9	0.001
Augmentation index @ 75 [90%Cl] %	19.2±8	20.9±9	16.8±6.9	0.018
Augmentation Pressure (mmHg)	9.9 ±5.3	10.4±4.9	8.7±4.2	0.09
Cardiac output (l/min)	5.2±0.6	5.2±0.7	5.1±0.4	0.1
Cardiac index (l/min*1/m²)	2.7 ±0.4	2.77±0.4	2.76±0.3	0.8
Mean Arterial Pressure (mmHg)	110± 10	110.45±10	109.43±11	0.1
Pulse pressure (mmHg)	50.9±10	50.2±10.6	51.8±10.2	0.4
24th average SBP (mmHg)	141.3±14	142±15	139±12	0.3
24th average DBP (mmHg)	87.4±11.5	88±9	85±13	0.2
Day-time average SBP (mmHg)	144.4±14	145±16	143±12	0.4
Day-time average DBP (mmHg)	90.9 ±12	91±10	89±14	0.5
Night-time average SBP (mmHg)	130.4 ±16	133±14	126±17	0.03
Night-time average DBP (mmHg)	78.2 ±12	80±11	75±14	0.07
Total vascular resistance (s*mmHg/ml)	1.17±0.15	1.19 ±0.1	1.15±0.12	0.6
Reflection Magnitude (%)	67.5±8.3	67.4±7.9	67.7±8.9	0.3

Table 2. Correlations Between 25 Hydroxyvitamin D values and Arterial Stiffness parameters

Variable	25 Hydroxyvitamin D value r	25 Hydroxyvitamin D value p
Pulse Wave Velocity [90%Cl] (m/s)	-0.599**	<0.001
Augmentation index @ 95 [90%Cl] %	-0.341**	0.001
Augmentation Pressure (mmHg)	-0.325**	0.001
Cardiac output (l/min)	-0.148	0.1
Cardiac index (l/min*1/m²)	-0.05	0.6
Mean Arterial Pressure (mmHg)	-0.100	0.3
Pulse pressure (mmHg)	0.04	0.6
Office SBP (mmHg)	-0.066	0.5
Office DBP (mmHg)	-0.104	0.3
Total vascular resistance, s*mmHg/ml	-0.060	0.5
Reflection Magnitude, %	0.068	0.5
Heart rate (beats per minute)	-0.156	0.12

Hypertension

OP-107

The analysis of the relationship between low vitamin D levels and cardiovascular diseases: hypertension, cardiac hypertrophy, atrial fibrillation, stroke

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Background and Aim: Even though there are many treatments and therapies for the control of hypertension; hypertensive organ damage such as, cardiac hypertrophy, atrial fibrillation and ischemic stroke is still very common worldwide. Thus in this study we have investigated the relationship between the potential risk factor 'low vitamin D levels' and; stroke types, atrial fibrillation, hypertension, left ventricular hypertrophy (LVH).
Methods: A total of 340 atrial fibrillation, hypertension, LVH, stroke clinical cases between the years of 2018-2019 were studied in a single tertiary hospital. Vitamin D levels of atrial fibrillation, hypertension, LVH, stroke patients were compared with 85 healthy controls. Stroke subtypes were grouped as cryptogenic (unknown etiology) stroke, major artery disease and cardioembolic/ischemic stroke. The vitamin D levels were grouped as; deficient (<20 ng/ml), insufficient (20-30 ng/ml) and normal (>30 ng/ml). Data collected from included patients were; Left Ventricular Thickness, Electrocardiography, Congenital and Chronic Diseases, Blood Pressure, Gender, Age, Echocardiography. Exclusion criteria were; Hemorrhagic stroke, Rheumatic Valve Disease, Prosthetic Valve, Vasculitis, Liver failure, Chronic Renal Failure, Uncontrolled Thyroid Dysfunction, Malignant Diseases, taking vitamin D supplements.
Results: Comparison of vitamin D levels between normotensives and hypertensive patients show a significant tendency of hypertensive patients having lower vitamin D levels (Cramer's V=0.212, p=0.034) (Figure 1). Compared to hypertensive men, hypertensive women have lower vitamin D levels (p=0.023, Cramer`s V=0.325). Low vitamin D levels were found significantly much more in left ventricular hypertrophy patients when compared to healthy controls (p>0.01, Cramer's V=0.402) (Figure 2). Vitamin D deficiency and insufficiency was significantly higher in female atrial fibrillation patients (p=0.043, Cramer`s V=0.298). The analysis of stroke subtypes shows that ischemic/cardioembolic stroke patients had vitamin D levels significantly lower than other stroke subtypes (p=0.013, Cramer`s V=0.319). Most of the women (82.6%) were postmenopausal.
Conclusions: To our knowledge this is the first study conducted in our country where vitamin D levels were evaluated in stroke sub types and also, this type of study is very limited in the world literature. It has been seen that cardioembolic and ischemic stroke patients have higher vitamin D3 insufficiency. When compared to men, in women vitamin D deficiency and insufficiency were seen much higher in cardiovascular diseases. One of the most important findings of this study is that gender and postmenopausal states may affect the results of these kinds of studies. Thus, in the future studies of this topic, these kinds of parameters should be studied in detail.

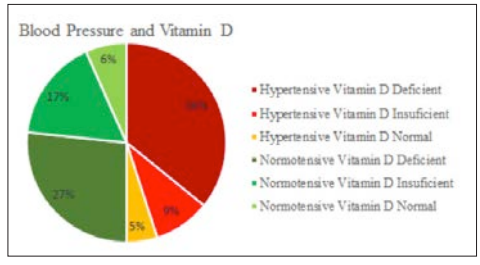

Figure 1.

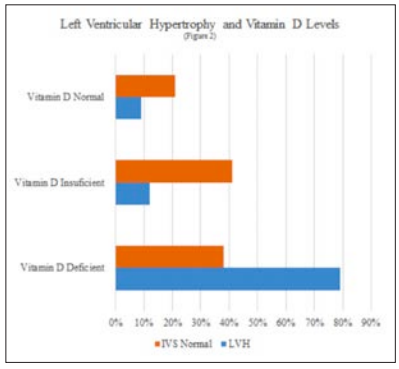

Figure 2.

Hypertension

OP-108

Effects of Antihypertensive drug classes on central aortic pressure

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Background and Aim: Central aortic pressure is the more important indicator of cardiovascular risks when compared with peripheral blood pressure (BP). We aimed to evaluate the effects of beta-blockers and other antihypertensive drugs on central aortic pressure which was measured by invasive direct method.
Methods: We included 201 patients using antihypertensive treatment from the patients referred for diagnostic coronary angiographic evaluation. Brachial BP was measured synchronously with the central aortic BP at the beginning of coronary angiographic examination before the contrast agent injections to the coronary arteries.
Results: Systolic BP and pulse pressure (PP) were significantly higher in central measurements (150±26 vs 148±20.8, p=0.006; 72±18.5 vs 66±15.1, p<0.001; respectively), diastolic BP was significantly higher in peripheral measurements (81±11.4 vs 78±15, p<0.001) however, mean BP was similar between central and peripheral measurements (104±13.4 vs 102±17.3, p=0.141). There was no statistically significant difference in central and peripheral BP measurements regarding systolic BP, diastolic BP, PP, mean BP between beta blockers group and non-beta blocker group. Also, there was no statistically significant difference with augmentation pressure and augmentation index between two groups (20.2±12.3 vs 18.8±11.2, p=0.56; 27.7±15.1 vs 25.4±13.3, p=0.39; respectively).
Conclusions: In this study we evaluated the effects of different antihypertensive drugs on central BP with an invasive method and showed that there was no difference between drug classes.

Table 1. Comparison of central and peripheral blood pressure between beta-blockers and non-beta blockers

	BB group (n=67)	Non-BB group (n=134)	p value
Brachial BP			
SBP (mmHg)	146.4±24.7	149.2±18.5	0.42
DBP (mmHg)	82.5±13.3	81.5±10.4	0.60
MBP (mmHg)	103.8±16	104.1±12	0.89
PP (mmHg)	63.9±17.1	67.7±13.9	0.11
Central BP			
SBP (mmHg)	147.5±31.3	152.7±22.8	0.23
DBP (mmHg)	76.3±16.6	80.2±14	0.07
MBP (mmHg)	100±20	104.4±15.6	0.12
PP (mmHg)	71.2±22.2	72.4±16.4	0.70
Augmentation pressure	20.2±12.3	18.8±11.2	0.56
Augmentation index	27.7±15.1	25.4±13.3	0.39

Hypertension

OP-109

The change of blood pressure and heart rate recovery index values according to body mass index in healthy individuals

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Background and Aim: In the Treadmill exercise test (TET), an unexpected increase in blood pressure and development of hypotension are associated with cardiovascular morbidity and mortality. The aim of our study is to compare systolic blood pressure and heart rate recovery index (HRR-i) values of healthy individuals with body mass index (BMI) values below and above 30 kg/m² in the TET.
Methods: The study was conducted between March 2017 and April 2020 at the Sincan State Hospital cardiology outpatient clinic. 154 healthy patients (Group 1) who came to the cardiology outpatient clinic with atypical angina, entered TET, whose test did not indicate coronary artery disease, and hypertension was not diagnosed and BMI values were below 30 kg/m², and 113 healthy participants (Group 2), BMI values were over 30 kg/m², included in the study. HRR-i and blood pressure recovery index (BPR-i) values were calculated and compared from the data in TET.
Results: The first, second and third-minute HRR-i values of group 1 were significantly higher than the HRR-i value of group 2 (Respectively, 56.92±5.20 vs 48.34±4.56, p<0.0001 and 63.20±3.18 vs 58.36±4.34, p<0.0001 and 77.40±4.47 vs 71.38±6.43, p<0.0001). The first, second and third minute systolic BPR-i values of group 1 were significantly higher than the systolic BPR-i value of group 2 (Respectively, 26.74±4.26 vs 21.45±3.68, p<0.0001 and 35.96±3.84 vs 31.24±5.41, p<0.0001 and 46.48±5.73 vs 40.62±6.33, p<0.0001).
Conclusions: HRR-i and systolic BPR-i values of group 1 were significantly higher than the values of group 2. These values show that autonomic functions are significantly delayed in group 2 compared to group 1.

Hypertension

OP-110

Effects of antihypertensive drugs on myocardial deformation parameters by 2D specle tracking echocardiography

Belma Yaman

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Background and Aim: Hypertension (HT) is the most important reason of left ventricle (LV) diastolic dysfunction. Antihypertensive drug classes have different effects on blood pressure and target organ damages. In this study we aimed to compare the effects of different antihypertensive drugs on myocardial deformation parameters by speckle tracking echocardiography.
Methods: 29 patients who use angiotensin converting enzyme (ACE) inhibitor/angiotensin receptor blocker (ARB) or calcium channel blocker for HT prospectively included to study. Patients were divided into two group; group 1 (ACE inhibitor/ARB), group 2 (calcium channel blocker). In addition to standard 2D echocardiographic measurements, left ventricle global longitidunal strain (LvGLS), right ventricle global longitidunal strain (RvGLS) and left atrium strain (LAS) were analyzed. LAS is defined according to three LA cycles; LAS during reservoir phase (LASr), LAS during conduit phase (LAScd), LAS during contraction phase (LASct).
Results: 62.1% (18) of the participants were taking group 1 antihypertensive. Conventional echocardiographic parameters including left/right ventricular diameter, ejection fraction, left atrial diameter and volume were similar between two group. E/a ratio was significantly higher in group 2 (p=0.02). As compared myocardial deformation parameters regarding Lv GLS, Rv GLS and LAS, there was no statistically significant difference between ACE inhibitor/ARB and calcium channel blockers (for Lv GLS -18.1±2.3% vs. -19.2±2.4%, p=0.230; for Rv GLS -19.3±5.5% vs. -21.1±4.6%, p=0.358).
Conclusions: ACE inhibitors/ARB and calcium channel blockers have similar effects on myocardial deformation parameters regarding LV strain, RV strain and LA strain which are the early indicator of systolic and diastolic dysfunction.

Table 1. Comparison of basal demographic features and echocardiographic parameters between two group

	Group 1 (ACEinh/ARB) (n=18)	Group 2 (CCB) (n=11)	p value
Age (year)	54.3 ± 7.2	51 ± 0.9	NS
Gender male (%)	9 (50)	8 (72.7)	NS
Septum thickness	10.8 ± 0.9	10.6 ± 1	NS
Peak E velocity (cm/s)	65.3 ± 11.7	72.3 ± 16.8	NS
Peak A velocity (cm/s)	78.8 ± 21.5	63.4 ± 14.6	0.03
E/A ratio	0.8 ± 0.2	1.1 ± 0.3	0.02
Lv GLS %	-18.1 ± 2.3	-19.2 ± 2.4	NS
Rv GLS %	-19.3 ± 5.5	-21.1 ± 4.6	NS
LASr %	30.2 ± 7.9	29.7 ± 10.2	NS
LAScd %	12.3 ± 6.4	14.3 ± 7.7	NS
LASct %	17.7 ± 4.7	15.4 ± 4.9	NS

Cardiovascular surgery

OP-111

The relationship between incomplete surgical obliteration of the left atrial appendage and thromboembolic events after mitral valve surgery (from the ISOLATE registry)

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Background and Aim: Left atrial appendage (LAA) is a common site of thrombus formation especially in patients with atrial fibrillation (AF). Complete surgical LAA closure (cSLC) is the surgical aim, however incomplete surgical LAA closure (iSLC) is not rare. In this study, we aimed to evaluate the risk of thromboembolic complications (TEC) in AF patients with iSLC after mitral valve surgery.
Methods: A total of 101 AF patients (mean age: 61.8±11.8 years; male:32), who underwent surgical suture ligation during mitral valve surgery were enrolled in this retrospective study. All patients underwent transthoracic

and transesophageal echocardiography (TEE) at least 3 months after surgery. The primary outcome was the occurrence of TEC including any ischemic stroke, transient ischemic attack, coronary or peripheral embolism.
Results: TEE examination revealed cSLC in 66 (65.3%) and iSLC in 35 patients (34.6%). A total of 12 TECs (11.9%) occurred during a mean follow-up time of 41.1±15.6 months. TECs were found to be significantly higher in the iSLC group (25.7% vs 4.5%, p=0.002). The prevalence of iSLC was significantly higher in patients with TEC (75 vs. 29.2 %, p=0.002). High CHA2DS2-VASc Score and iSLC were found to be independent predictors of TEC. Long term TEC free survival was found to be significantly decreased in patients with iSLC.
Conclusions: The presence of iSLC was associated with a significantly increased risk of TEC in AF patients after mitral valve surgery. Routine intraoperative and postoperative screening for iSLC by TEE and long-term strict anticoagulation therapy are recommended in these patients.

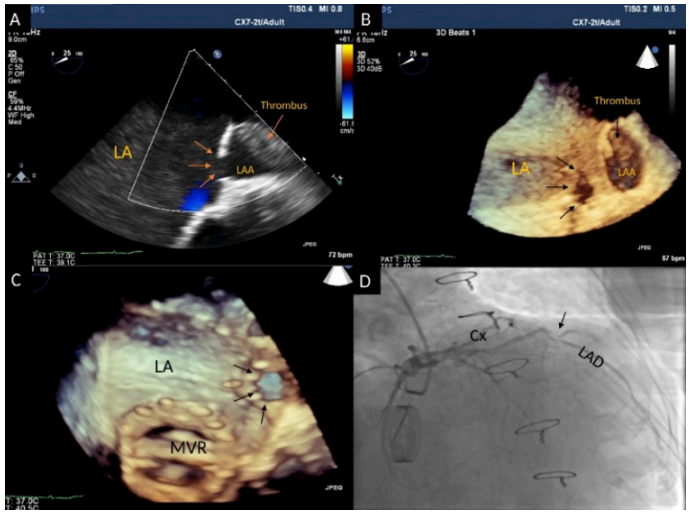

Figure 1.

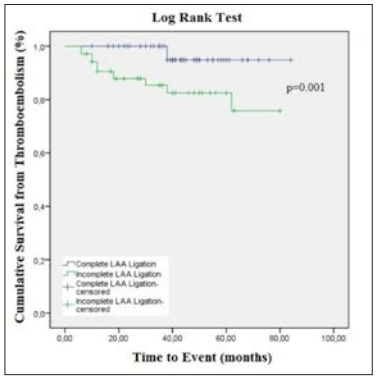

Figure 2.

Cardiovascular surgery

OP-112

Usefulness of aortic knob width and calcification on chest radiography to predict saphenous vein graft patency after coronary artery bypass grafting

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Background and Aim: Saphenous vein graft (SVG) occlusion is an independent predictor of adverse outcomes such as cardiovascular death and myocardial infarction. Aortic knob width (AKW) and aortic knob aortic knob calcification (AKC), which can be detected by chest X-ray, have been associated with cardiac events. The main aim of this study was to investigate the relation between AKW, AKC, and saphenous vein graft patency after coronary artery by-pass grafting surgery (CABG).
Methods: We analyzed 144 patients who had undergone isolated CABG surgery and presented for computed tomography (CT) angiography evaluation for graft patency 40.32±26.34 months after CABG surgery. AKC and AKW were evaluated with pre-operative chest X-ray.
Results: Presence of AKC (21 [21.8%] vs. 24 [50%], p<0.0001) and AKW (33.36±4.05 vs. 38.41±4.93, p<0.0001) were significantly higher in the group of occluded saphenous grafts compared with the group of patent grafts (p <0.05 for each comparison). Multiple logistic regression analysis showed that AKW (odds ratio [OR] 1.4, 95% confidence interval [CI] 1.25 to 1.6, p=0.001) and AKC (OR 6, 95% CI 2.1 to 16.7, p=0.001) were independently associated with saphenous graft occlusion. ROC analyses showed positive correlation for AKW and the presence of AKC in predicting saphenous graft occlusion (for AKW: Area under the curve (AUC) 0.86, p<0.0001; 95% CI=0.81-0.92, and for AKC: AUC 0.62, p=0.01; 95% CI=0.52-0.72).
Conclusions: The results suggest that pre-operative evaluation of AKW and AKC with a simple routine chest X-ray, might give more information about the future outcome of the upcoming CABG surgery.

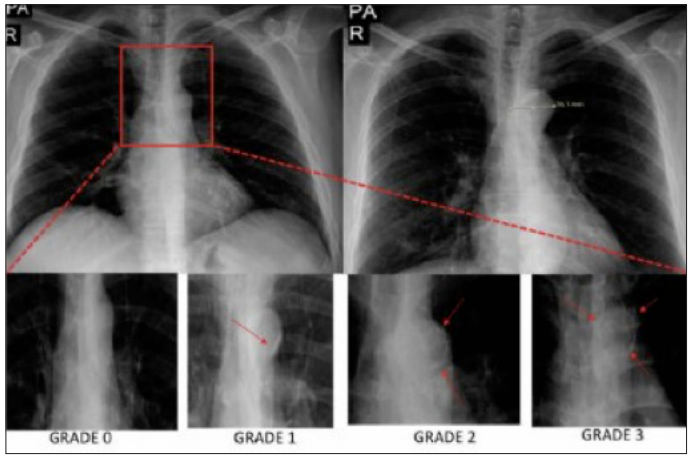

Figure 1. Postero-anterior chest radiography view of aortic knob width assessment and grading of aortic knob calcification.

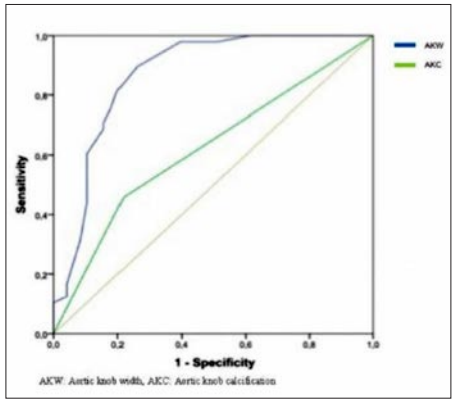

Figure 2. ROC analyses of AKW (blue line) and the presence of AKC (green line) in predicting saphenous graft occlusion (for AKW: area under the curve (AUC) 0.86; p<0.0001, 95% CI=0.81-0.92, and for the presence of AKC: AUC 0.62, p=0.01; 95% CI=0.52-0.72).

Table 1. Pre-procedural demographic, clinical, laboratory and radiological characteristics of patients

Patients Clinical Features	Study cohort (n=144)	Patients with patent grafts (n=96)	Patients with occluded saphenous grafts (n=48)	P value
Age (years)	57.81 ± 9.97	56.92± 9.58	59.58 ± 10.58	NS
Male Gender, n (%)	97 (67.4%)	66 (68.8%)	31 (64.6%)	NS
Weight (kg)	79.7 ± 13.13	80.85 ± 13.13	77.58 ± 12.98	NS
Height (cm)	167.33 ± 8.44	167.12 ± 8.9	167.75 ± 7.39	NS
BMI , (kg/m²)	28.55 ± 4.75	29.1 ± 4.73	27.62 ± 4.61	NS
Hypertension, n (%)	116 (80.6%)	74 (77.1%)	42 (87.5%9	NS
Diabetes Mellitus, n (%)	67 (46.5)	44 (45.8%)	23 (47.9%)	NS
Dyslipidemia, n (%)	97 (67.4%)	65 (67.7%)	32 (66.7%)	NS
Current smoker, n (%)	60 (41.7%)	17 (35.5%)	43 (44.8%)	NS
Peripheral artery disease, n (%)	23 (16%)	14 (14.6%)	9 (18.8%)	NS
Cerebrovascular events, n (%)	11 (7.6%)	8 (8.3%)	3 (6.3%)	NS
Previous MI, n (%)	53 (36.8%)	40 (41.7%)	13 (27.1%)	NS
Previous coronary stent, n (%)	40 (27.8%)	12 (25%)	28 (29.2%)	NS
Carotid artery disease, n (%)	22 (15.3%)	15 (15.6%)	7 (14.6%)	NS
COPD, n (%)	41 (28.5%)	19 (19.8%)	12 (25%)	NS
Ejection fraction (%)	54.72 ± 9.05	54.38 ± 9.77	55.39 ± 2.74	NS
Total cholesterol (mg/dL)	195.69 ± 48.93	188.99 ± 48.55	209.8 ± 48.4	**0.02**
HDL-c (mg/dL)	40.24 ± 11.03	40.26 ± 12.29	20.21 ± 8.07	NS
LDL-c (mg/dL)	124.6 ± 41.48	118.28 ± 41.79	137.23 ± 38.43	**0.009**
Triglyceride(mg/dL)	163 (117)	161 (95)	167 (90)	NS
Serum creatinine (mg/dL)	0.85 ± 0.5	0.87 ± 0.18	0.82 ± 0.19	NS
Hemoglobin (g/dl)	13.73 ± 1.74	13.87 ± 1.71	13.46 ± 1.77	NS

BMI, body mass index; MI, myocardial infarction; COPD, chronic obstructive pulmonary disease; NYHA, New York Heart Association; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol;

Table 2. Pre-procedural data of chest X-ray evaluations of the patients

Patients Clinical Features	Study cohort (n=144)	Patients with patent grafts (n=96)	Patients with occluded saphenous grafts (n=48)	P value
Presence of Aortic Knob Calcium, n (%)	46 (31.9%)	21 (21.8%)	24 (50%)	**<0.0001**
Aortic Knob Calcium Grade, n (%)				**<0.0001**
0	98 (68.1)	76 (79.2%)	24 (27%)	
1	34 (23.6%)	14 (14.6%)	13 (35.4%)	
2	12 (8.3%)	6 (6.3%)	6 (12.5%)	
3	6 (4.2%)	1 (1.6%)	5 (10.4%)	
Aortic Knob Width (mm)	35.41 ± 4.39	33.36 ± 4.05	38.41 ± 4.93	**<0.0001**
Interval from operation to angiogram (months)	40.32 ± 26.34	37.25 ± 24.92	46.20 ± 28.3	**0.04**

Table 3. Procedural and post-procedural characteristics of the patients

Variables	Study cohort (n=144)	Patients with patent grafts (n=96)	Patients with occluded grafts (n=48)	P value
CPB time (min)	87 ± 22	84.69 ± 21.72	93.66 ± 22.5	**0.01**
Aortic cross clamp time (min)	47.81± 15.3	42.07 ± 14.63	47.81 ± 15.39	**0.03**
LITA use	132	93 (96.9%)	44 (91.6%)	NS
RITA use	2 (1.4%)	0 (%0)	2 (4.2%)	NS
Sequential grafting	16 (11.1%)	10 (9.6%)	6 (12.5%)	NS
Off pump revascularization	7 (4.8%)	3 (4.1%)	4 (8.3%)	NS
Intensive care unit Stay >48h	22 (15.3%)	12 (12.5%)	10 (20.8%)	NS
Neurologic complications	3 (2.1%)	3 (3.1%)	0	NS
Post-operative atrial fibrillation	22 (15.3%)	15 (15.6%)	7 (14.6%)	NS
Deep sternal wound infection	2 (1.3%)	1 (1%)	1 (2.1%)	NS
Any postoperative use of antibiotics	43 (29.9%)	28 (29.2%)	15 (31.3%)	NS
Acute kidney injury	35 (24.3%)	23 (24%9	12 (25%)	NS
Reintervention for hemorrhage or sternal dehiscence	8 (5.6%)	4 (4.2%)	4 (8.3%)	NS

CPB, cardiopulmonary bypass; LITA, left internal thoracic artery; RITA, right internal thoracic artery

Table 4. Logistic regression analysis of pre-procedural and procedural selected variables in predicting saphenous graft occlusion

Variables	Odds Ratio	95% CI	P value
Increased Aortic Knob Width	1.4	1.25-1.6	**0.001**
Presence of Aortic Knob Calcium	6	2.1-16.7	**0.001**
Higher LDL	0.9	0.9-1.1	NS
Higher Total cholesterol	1	0.9-1.1	NS
Longer Aortic cross clamp time	0.3		NS
Interval from operation to angiogram	1.3	0.8-1.8	NS

Cardiovascular surgery

OP-113

The relationship of incomplete surgical closure of the left atrial appendage and thromboembolic events

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Background and Aim: Surgical left atrial appendage (LAA) exclusion, particularly with suture ligation, can often yield incomplete LAA closure, which may inturn be associated with increased thromboembolic risk. Incomplete surgical LAA closure can be further defined as incompletely surgically ligated LAA (iSLL). In this study, we aimed to evaluate the risk of thromboembolic complications (TEC) in AF patients who had iSLL or complete LAA closure (cSLL), after surgical suture ligation was performed in conjunction with heart valve or AF surgery.

Methods: We retrospectively evaluated a total of 101 patients, who were operated between February 2008 and October 2019 were included in this multicentric study (mostly from Koşuyolu Kartal Heart Training and Research Hospital and Mehmet Akif Ersoy Chest and Cardiovascular Surgery Training and Research Hospital). All patients underwent transesophageal echocardiography (TEE). Surgical ligation of the LAA was clearly identified by the lack of any anatomical structure between the mitral valve base and the upper left pulmonary artery. The definition of incomplete ligation was made according to the following criteria: 1) a jet showing the distinction between LAA and LA body with colored Doppler flow, 2) no structural continuity with 2-dimensional (2D) TEE. In addition, ISLL was evaluated in detail with real-time 3D TEE. The data on thromboembolic complication (TEC) and mortality were recruited from medical records and telephone interviews

Results: TEE examination revealed cSLL in 66 (65.3%) and iSLL in 35 patients (34.6%). The mean follow-up time was 41.1±15.6 months. During this time, a total of 12 TECs (11.9%) occured. TECs were found to be significantly higher in the iSLL group compared to cSLL groups (25.7% vs 4.5%, p=0.002). However, there was no significant difference between the two groups in terms of all-cause mortality (6.1% vs 8.6%, p=0.63). These findings (postoperative parameters) were similar between iSLL patients with and without TECs. All-cause mortality was significantly higher in the group with TECs (25% vs 4.5% p=0.009).

Conclusions: In AF patients undergoing surgical suture ligation of LAA, the presence of iSLL was associated with a significantly stronger risk of TEC than cSLL. Therefore, routine postoperative screening for iSLL, long-term strict anticoagulation therapy and follow-up are strongly encouraged in this high-risk group of patients. Lastly, new surgical techniques and devices should be developed for surgical LAA ligation in parallel with technological developments.

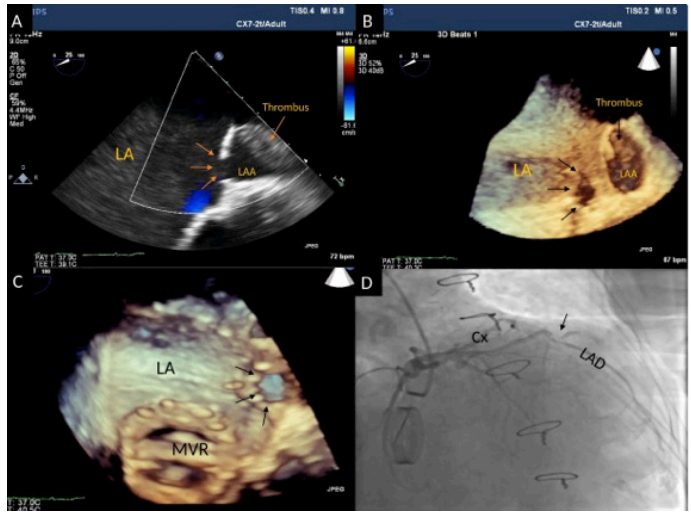

Figure 1.

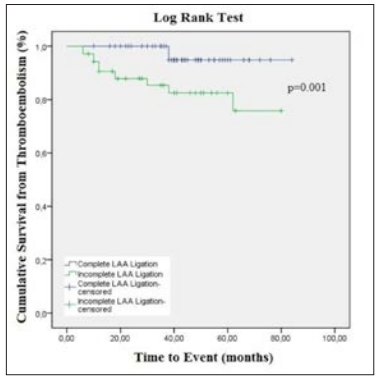

Figure 2.

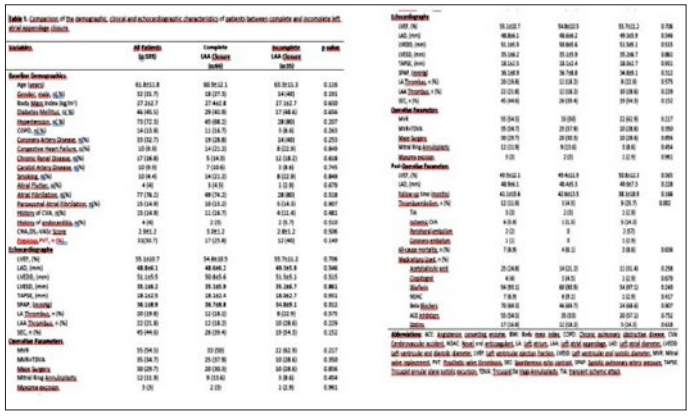

Table 2. Comparison of the demographic, clinical and echocardiographic characteristics between patients with and without thromboembolic complications

Variables	Patients with TECs (n:12)	Patients without TECs (n:89)	p value
Baseline Demographics			
Age (years)	67.4±10.8	61.1±11.7	0.077
Gender, male, n (%)	4 (33.3)	28 (31.5)	0.896
Body Mass Index (kg/m²)	26.9±1.7	27.3±2.8	0.698
Diabetes Mellitus, n (%)	9 (75)	37 (41.6)	0.029
Hypertension, n (%)	9 (75)	64 (71.9)	0.822
COPD, n (%)	3 (25)	11 (12.4)	0.234
Coronary Artery Disease, n (%)	3 (25)	30 (33.7)	0.546
Congestive Heart Failure, n (%)	5 (41.7)	17 (19.1)	0.075
Chronic Renal Disease, n (%)	1 (8.3)	16 (18)	0.402
Carotid Artery Disease, n (%)	1 (8.3)	9 (10.1)	0.846
Smoker, n (%)	1 (8.3)	21 (23.6)	0.229
Atrial Flutter, n (%)	1 (8.3)	3 (3.4)	0.408
Atrial Fibrillation, n (%)	10 (83.3)	67 (75.3)	0.538
Paroxysmal Atrial Fibrillation, n (%)	1 (8.3)	14 (15.7)	0.499
History of CVA, n (%)	2 (16.7)	13 (14.6)	0.851
History of endocarditis, n (%)	2 (16.7)	2 (2.2)	0.016
CHA2DS2-VASc Score	3.82±1.08	2.78±1.15	0.005
Previous PVT, n (%)	5 (41.7)	26 (29.2)	0.380
Echocardiography			
LVEF, (%)	52.8±12.7	55.4±10.5	0.423
LAD, (mm)	51.1±8.5	48.6±5.7	0.192
LVEDD, (mm)	52.2±6.1	50.9±5.4	0.455
LVESD, (mm)	36.6±8.1	34.9±5.9	0.373
TAPSE, (mm)	18.2±3.2	18.1±2.4	0.892
SPAP, (mmHg)	32.4±4.2	36.6±9.3	0.132
LA Thrombus, n (%)	2 (16.7)	18 (20.2)	0.772
LAA Thrombus, n (%)	3 (25)	19 (21.3)	0.774
SEC, n (%)	8 (66.7)	37 (41.6)	0.101
Operative Parameters			
MVR	9 (75)	46 (51.7)	0.128
MVR+TDVA	2 (16.7)	33 (37.1)	0.163
Maze Surgery	3 (25)	27 (30.3)	0.704
Mitral Ring Annuloplasty	2 (16.7)	10 (11.2)	0.585
Myxoma excision	1 (8.3)	2 (2.2)	0.244
Post-Operative Parameters			
LVEF, (%)	49.9±12.1	49.6±12.9	0.923
LAD, (mm)	48.6±5.6	51.8±8.9	0.256
LAA Closure, n (%)			
Complete	3 (25)	63 (70.8)	0.002
Incomplete	9 (75)	26 (29.2)	
Follow-up time (months)	42.8±19.5	40.8±15.2	0.673
All-cause mortality, n (%)	3 (25)	4 (4.5)	0.009

Table 3. Multivariate regression analysis showing independent predictors of thromboembolism.

Parameters	OR	95% CI	p value
Age	1.165	0.980-1.384	0.083
Diabetes mellitus	3.098	0.572-16.767	0.189
Congestive heart failure	7.465	1.157-48.180	0.035
History of endocarditis	12.345	0.603-252.917	0.103
CHA2DS2-VASc Score	1.114	0.423-2.929	0.828
Incomplete LAA closure	9.006	1.592-50.952	0.013

Abbreviations: CI: Confidence Interval; LAA: Left atrial appendage, OR: Odds ratio

Cardiovascular surgery

OP-115

The cerebrovascular accident event in postoperative patients undergoing coronary artery bypass surgery may have higher incidence in smokers and high number of grafts?

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Background and Aim: The present study aimed to investigate the predictors especially graft number and smoking for postoperative stroke in the patients that have undergone on- or off-pump coronary artery bypass graft (CABG).
Methods: The present study comprises a total of 623 patients that have undergone CABG surgery in our clinic. Initially, isolated CABG surgery with full revascularization was performed in all study participants. The left internal mammary artery (LIMA) was used in all of the cases. Great saphenous vein was preferred than radial artery as a conduit. Proximal anastomoses to the aorta in both On-pump and Off-pump techniques were performed by side clamps. The patients were discharged from the hospital between postoperative Day 6 and Day 11 and were monitored for stroke over postoperative 30 days.
Results: The patients were monitored for CVA until the postoperative Day 30. There were a total of six patients with cerebrovascular accident; two patients had ischemic optic neuritis and two patients had left hemiplegia and two patients had right hemiplegia.
Conclusions: The results of the present study can be summarized under two items: the first, the risk of postoperative stroke increases as the number of grafts increases in CABG cases that underwent complete revascularization. Second, being a smoker until hospital admission enhances the risk of postoperative stroke.

Table 1. Characteristics of the patients according to stroke

	No stroke Patients n=613	Stroke Patients n=10
Age (±SD) (year)	62.9±10.1	65.6± 6.7
Gender (Male/Female)	400 (65.3%) / 213 (34.7%)	6 (60%) / 4 (40%)
Smoking	248 (40.5%)	9 (90%)
COPD	121(19.7%)	3 (30%)
Hypertension	497 (81.1%)	10 (100%)
PAD	32 (5.2%)	0
Preoperative thrombocyst count	257.9±85.5	253.5 ± 48.4
Preoperative stroke story	42 (6.9%)	1 (10%)
Diabet oral a/d parenteral a/d	181 (29.5%) 113 (18.4%)	4 (40 %) 1(10 %)
Right carotid artery stenosis<%50 %50< stenosis≤%70 % 70≤ stenosis<%100 stenosis=%100	200 (32.6%) 29 (4.7%) 3 (0.5%) 3 (0.5%)	3 (30%) 0 1 (10%) 0
Left carotid artery stenosis<%50 %50< stenosis≤%70 % 70≤ stenosis<%100 stenosis=%100	196 (32%) 31 (5.1%) 5 (0.8%) 8 (1.3%)	3(30%) 0 1 (10%) 0
Weigth(kg)	78.3±13.6	74.4±13.9
BMI	29.5 ± 5.1	28.3±4.7
Preoperative Ejection Fraction (EF)	54.3± 9.2	56.2±13.9
30%≤EF<50% (Male/Female)	107 (26.7%) / 48 (22.5%)	2 (33.3%) / 2 (50%)
Numbers of grafting	3.2±0.9	4.6 ±0.5
Type of CABG Surgery (On-pump / Off-pump)	394 (64.3%) / 219 (35.7)	5 (50%) / 5 (50%)
(On-Pump CABG) CPB time X Clamp time(min.)	107.4±40.7 51.9±16.1	110.8±1 53.8±4.3

BMI: Body Mass Index, COPD: Chronic obstructive pulmonary disease, PAD: Peripheral artery disease.

Valvular heart diseases

OP-116

A retrospective single-center study/Transcatheter aortic valve implantation versus surgical aortic valve replacement in patients with severe aortic stenosis and intermediate surgical risk

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Background and Aim: The aim of this study was to evaluate the clinical experience of our center on Transcatheter Aortic Valve Implantation (TAVI) and to compare the early and late clinical outcomes of patients with severe Aortic Stenosis and intermediate risk group who underwent TAVI or Surgical Aortic Valve Replacement (SAVR).
Methods: The Our retrospective study included a total of 128 patients over 60 years of age with Severe Aortic Stenosis who were admitted to Cardiology and Cardiovascular Surgery outpatient clinic of Atatürk University Faculty of Medicine between 2012 and 2018 and underwent TAVI or SAVR. The study population was in the middle-risk surgical group according to the surgical risk scores which are proposed in the current guidelines with degenerative etiology of an aortic valve with tricuspid structure. The number of intermediate risk surgical patient was 67 in the TAVI group and 61 in the SAVR group.The demographic data, echocardiography,laboratory results and other clinical information of the patients were obtained from the hospital database (Table 1).
Results: In Our study, the mean age was 76.4±8.79 years in the TAVI group and 70.72±5.08 years in the the SAVR group. The mean follow-up period was 29.55±19.76 months in the TAVI group and 31.28±23.77 months in the SAVR group. There was no statistically significant difference between early and late mortality, survival rates and survival time in the both groups (Figure 1). While the need for postoperative transfusion was significantly higher in the SAVR group, the frequency of acute and life threatening Ischemic and Hemorrhagic

Stroke, Acute Renal Failure was similar in both groups. Early and late period due to Heart Failure, Coronary Artery Disease, Permanent Pacemaker Implantation and miyocardial Infarction hospital re-admissions were similar in both groups. In the group with ventricular and supraventricular tachycardias, infective endocarditis and pericardial effusion, which we determined as other cardiovascular causes, were statistically more common in the SAVR group (p=0<0.05); paravalvular leakage rate was more common in the TAVI group.
Conclusions: In this study, we found that TAVI was noninferior to SAVR in the surgical intermediate risk group. As shown in the recent studies and in our study, these classical surgical risk scores show that the calculated risk of early in-hospital mortality in patients undergoing TAVI is more than rates obtained in the clinic studies. Therefore, new scoring systems are needed. Although age plays an important role in mortality scores, it gains significance with the patients' frailty status and additional comorbidities.

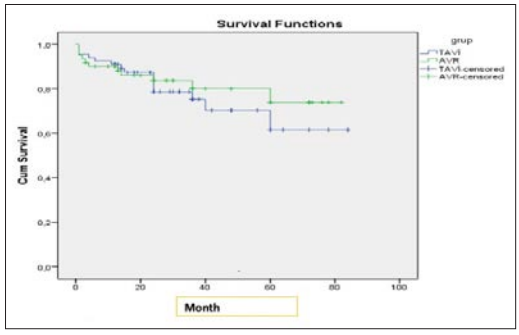

Figure 1. Life curve of TAVI and SAVR group patients.

Table 2. Comparison of the electrocardiographic parameters of the groups

Variables	MAK (+) (n =100)	MAK (-) (n =100)	p value
Heart Rate (per minute)	79 ± 18.5	70.5 ± 15.9	0.351
QRS duration (ms)	99 ± 15.9	92 ± 12.9	0.278
PR interval (ms)	155 ± 18.4	158 ± 19.3	0.643
P wave (ms)	109 ± 14.9	89 ± 11.3	<0.001
QT interval (ms)	384 ± 20.5	394 ± 33.4	0.198
QTc interval (ms)	481 ± 26.4	426 ± 15.4	<0.001
Tp-e interval, (ms)	90 ± 4.4	81 ± 5.1	<0.001
Tp-e/QT ratio	0.23 ± 0.03	0.20 ± 0.02	<0.001
Tp-e/QTc ratio	0.19 ± 0.02	0.17 ± 0.02	<0.001
f(QRS)□T (°)	69.2 ± 36.5	50.3 ± 37.5	<0.001

QTc - corrected QT interval; MAK, mitral annular calcification; f(QRS) T; frontal QRS-T angle.

Valvular heart diseases

OP-117

The effects of mitral annular calcification on electrocardiogram parameters

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Background and Aim: Mitral annular calcification (MAK) has been found to be associated with mortality indicators such as reduced heart rate variability, impaired P wave dispersion and atrial electromechanical delay. Taking into consideration that MAK may have potential arrhythmogenic effects, we aimed to investigate the effects of MAK on cardiac electrophysiology using classical ECG parameters in addition to new arrhythmogenic determinants such as f(QRS-T) angle and Tp-e/QT.
Methods: Total 200 consecutive patients [n=100 MAK (+) ve n=100 MAK (-)] were included study. All collected data and ECG parametreds were compared between groups.
Results: P wave was significantly longer in the MAK (+) group than the control group (p<0.001). Corrected QT interval was significantly longer in the MAK (+) group than the control group (p<0.001). Tp-e interval was significantly longer in the MAK (+) group than the control group (p<0.001). Tp-e / QT interval and Tp-e / corrected QT interval ratio were significantly longer in the MAK (+) group than the control group (p<0.001). The frontal QRS-T angle was significantly higher in the MAK (+) group (p<0.001).
Conclusions: We assessed the effects of MAK on ECG in our study and it has been found that for the first time in the literature, MAK has negative effects on cardiac repolarization parameters.

Table 1. Comparison of the basic demographic features of the groups with laboratory parameters

Variables	MAK (+) (n =100)	MAK (-) (n =100)	p value
Age	71.6 ± 8.5	69.5 ± 9.9	0.107
Male Gender, n(%)	44	49	0.478
Hypertension, n(%)	32	42	0.143
Diabetes mellitus, n(%)	31	30	0.878
Smoking, n(%)	20	17	0.538
History of coronary artery disease, n(%)	16	12	0.415
Hemoglobin, g/dl	13.9 ± 2.1	13.6 ± 1.8	0.469
Platelet,10□/l	290 ± 72.7	269 ± 48.8	0.132
White Blood Cell,10□/l	7.8 ± 2.7	7.4 ± 1.7	0.157
Neutrophil,10□/l	5.7 ± 1.4	4.3 ± 1.2	<0.001
Lymphocyte,10□/l	1.3 ± 0.5	2.3 ± 0.7	<0.001
Monocyte,10□/l	0.6 ± 0.2	0.5 ± 0.2	0.379
Serum creatinine, mg/dL	1.1 ± 0.3	1.0 ± 0.3	0.663
Total cholesterol, mmol/l	4.6 ± 1.1	4.6 ± 0.6	0.786
HDL cholesterol, mmol/l	1.1 ± 0.3	1.2 ± 0.2	0.149
LDL cholesterol, mmol/l	2.9 ± 0.8	2.7 ± 0.5	0.094
Triglyceride, mmol/l	1.6 ± 1.0	1.6 ± 0.7	0.724
Left ventricular ejection fraction (%)	62.3 ± 6.0	62.4 ± 5.8	0.256

HDL, high density lipoprotein; LDL, high density lipoprotein; MAK, mitral annular calcification. Data are given as mean ± standard deviation or percentage [n (%)].

Valvular heart diseases

OP-118

Degenerated bioprosthetic mitral valve thrombosis in pregnant woman: A case report

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Background and Aim: Despite the disadvantage of rapid development of structural valve degeneration in women of childbearing age, bioprosthetic heart valves are encouraged due to the low risk of thromboembolic complications and no need of anticoagulation (1). Bioprosthetic valves are less thrombogenic and do not require anticoagulation, however they have high rates of structural degeneration over time. Degenerated prosthetic valves in pregnancy impacts both mother and fetus, as valvular dysfunction can cause hemodynamic and thromboembolic complications in pregnancy (1). The current guidelines advise follow-up, medical treatment and indications for intervention are similar to native valve dysfunction in pregnancy (2).
Methods: A 33 weeks pregnant, gravida four, para three, 29-year-old patient with bioprosthetic mitral valves presented to emergency clinic with cardiac arrest. Emergent delivery was performed with C/S. Cardiologist and cardiovascular surgeon evaluated the patient after successful CPR and evaluated as acute bioprosthetic valve thrombosis.
Results: Urgent surgical intervention was warranted due to patient unstabil haemodynamic situation despite heparine infussion and inotropic support. Eventually, a successful mitral valve replacement was performed.
Conclusions: However, bioprosthetic valves in pregnancy remain still controversial, and the expected event rates for valve-related complications have not been clearly postulated. Here, we report a case of pregnant woman with thrombotic bioprosthetic mitral valve.

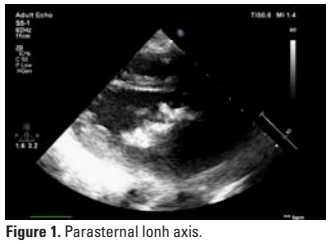

Figure 1. Parasternal lonh axis.

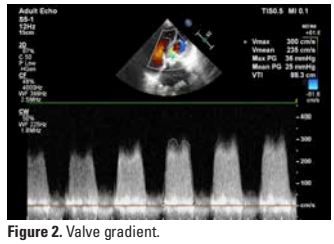

Figure 2. Valve gradient.

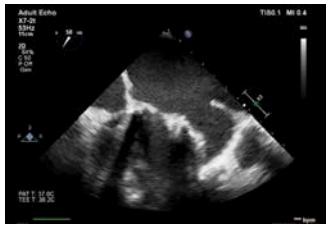

Figure 3. Tee view.

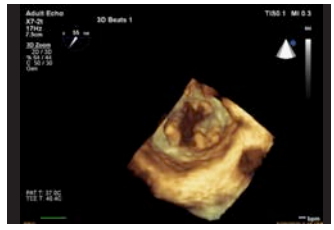

Figure 4. Tee 3d view.

Heart failure

OP-119

Empagliflozin; significantly attenuates necrosis and prevent left ventricle systolic functions in doxorubicin induced cardiomyopathy via non-antioxidant pathways: Echocardiographic, histologic and biochemical findings

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Background and Aim: Empagliflozin (EMPA) is a novel SGLT-2 inhibitor has been shown to have positive effects on cardiovascular outcomes. The mechanism of these positive effects seen in clinical outcomes has not been fully explained yet; but these effects were mainly attributed to increasing ketone bodies and reducing reactive oxygen species (ROS). The effects of EMPAon the cardiotoxicity of doxorubicin (DOX), which is known to increase ROS to cause heart failure, has not been shown yet.
We aim to evaluate protective effects of EMPA against DOX cardiotoxicity.
Methods: 38 Sprague Dawley rats were randomized. The control group (n=10) received SF (1ml) via orogastric gavage (OG) and intraperitoneally (i.p.) for14 days. The EMPA group (n=10) received EMPA (10 mg/kg) via OG and SF(i.p.) for 14 days. The DOX group (n=9)was given cumulatively 18 mg/kg body weight/6 days DOX(i.p.) and SF (1 ml) via OG for 14 days. The DOX+EMPA group (n=9) was received DOX and EMPA at the same dose. On the 15th day, the rats were anesthetized and echocardiographic and ECG examinations were done. Then blood samples were taken directly from heart to evaluate biochemical parameteres and heart tissues were excised to evaluate histopathological findings.
Results: In DOX group; LVDED (p<0.05) and LVSED (p<0.01),QTc interval (p<0.001), ratio of karyolisys and karyorheksis (p<0.001), infiltrative cell proliferation (p<0.001) were found to be significantly higher than the control group; EF, FS and normal cell morphology and were lower than the control group (p<0.001). In the DOX+EMPA group; LVEDD (p<0.05) and LVESD (p<0.01) values, QTc interval (p<0.001), Karyolisys and karyorheksis ratios (p<0.001) and infiltrative cell proliferation were significantly lower (p<0.01); normal cell morphology and EF was significantly higher compared to the DOX group (p<0.001) (Figure 1–3; Table 1). Biochemical analysis (including MDA, SOD, BNP, NO levels) were similar between four groups.
Conclusions: Our results showed that DOX caused left ventricle dilatation, deterioration in left ventricular function, QTc prolongation and myocyte cell necrosis. EMPA significantly ameliorated DOX induced left ventricle dilatation, QT prolongation; protected left ventricle sistolic function, infiltrative cell proliferation and necrosis. To our knowledge; this is the first study presenting protective effect of EMPA against DOX-induced cardiotoxicity. The data obtained in the current study suggest that the protective effect of EMPA is resulted from its regulating effects on the intracellular calcium metabolism (SERCA and Ryd) and from the increase in mitochondrial PGC-1 alpha levels, rather than the EMPA- indued mechanisims such as the decrease in preload and afterload due to natriuresis or the antioxidant effect provided by the elevated levels of beta-hydroxybutyrate and PAMPK, as preiously proposed. Due to our data EMPA can be recommended to patients with diabetes mellitus receiving DOX treatment in daily clinical practice.

Table 1.

	CONTROL (n=10)	EMPA (n=10)	DOX (n=10)	DOX+EMPA(n=9)
LVEDD mm	56 (10.25)	60 (9)	71 (9) a	55(8) b
LVESD mm	29 (6,25)	27 (9,5)	53 (9,5) c	28 (5,5) d
EF (%)	74,9 ± 4,9	78,4 ± 5,5	35,2 ± 10,5 e	74,5 ± 5,5 f
FS (%)	50,5 ± 6,2	50,3 ± 3,4	13,7 ± 6,2 e	45,4 ± 7 f
QTc interval (ms)	171,9 ± 31,4	180,9 ± 27,5	265,8 ± 34,3 e	182,4 ± 33,9 f
Normal Myocytes ratio (number / 100 myocyte)	0,954± 0,039	0,987± 0,008	0,452± 0,023 e	0,809±,026 f
Karyolysis ratio (number / 100 myocyte)	0,015± 0,012	0,003 ± 0,004	0,255 ±0,028 e	0,061 ± 0,008 f
Karyorrhexis ratio (number / 100 myocyte)	0,030 ± 0,027	0,012 ±0,008	0,293 ±0,025 e	0,130±0,021 f
Infiltrative Cell Quantity (number / per field)	3,1 ± 1,4	2,6 ± 1,1	11,2 ± 1,4 e	7,9 ± 2,9 d

a: DOX vs Control P<0.05; b: DOX +EMPA vs DOX p<0.05; c: DOX vs Control p<0.01 d: DOX +EMPA vs DOX p<0.01. e: DOX vs Control p<0.001; f: DOX +EMPA vs DOX p<0.001 Data are given as median (IQR) or mean ± standard deviation.

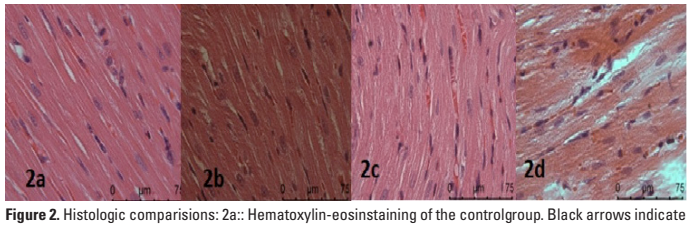

Figure 2. Histologic comparisions: 2a:: Hematoxylin-eosinstaining of the controlgroup. Black arrows indicate discus intercalaris, yellow arrows indicate normal cardiomyocyte nuclei 2b: Hematoxylin-eosinstaining of the EMPA group. Theyellowarrowsindicatethe normal cardiomyocyte nuclei and the black arrow shows the cardiomyocyte nucleus with karyorrhexis (63x magnification). 2c: Hematoxylin-eosinstaining of DOXO group. The yellow arrows indicate normal cardiomyocyte nuclei, the black arrow shows the cardiomyocyte nucleus with pyknosis, and the blue arrow heads indicate the cardiomyocyte nuclei with karyolysis. d: Hematoxylin-eosinstaining of DOX+EMPA group. The yellow arrows indicate the normal cardiomyocyte nuclei, the black arrow shows the cardiomyocyte nuclei with karyolysis and white arrow indicates pyknosis

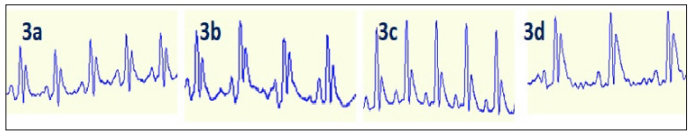

Figure 3. Electrocardiographic comparisions: **(a)** Control group **(b)** EMPA group **(c)** DOX group, QTc was prolonged **(d)** DOX+ EMPA group.

Heart failure

OP-120

Role of systemic immune-inflammation index to determine the rejection in heart transplant patients

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Background and Aim: End-stage heart failure refractory to medical treatment is associated with very high rates of morbidity and mortality. Currently, heart transplantation remains the gold-standard therapy for patients with end stage heart failure. At the same time, it is known the induction of inflammation play an important role during transplantation process. Evaluation of hematological parameters in peripheral blood including neutrophil, lymphocyte and platelet counts, which are indicators of inflammatory process, have important predictive value for the rejection process. The systemic immune-inflammation index (SII) was developed based on platelet counts and NLR (SII, platelet count * neutrophil / lymphocyte ratio), and simultaneously takes into account the inflammatory and immune status of patients. We thought that SII levels can be a useful, inexpensive, reproducible, and noninvasive tool for monitoring rejection process. Therefore, we sought to determine the relationship between the inflammation and immune-based score (SII) and rejection in patients with heart transplantation. To the best of our knowledge, there are no previous research in literature regarding the prognostic value of SII in heart transplant patients.
Methods: We retrospectively analyzed 80 heart transplant patients and 672 endomyocardial biopsies at the Department of Cardiology, University of Başkent. Systemic immune-inflammation index (SII) was calculated as total platelets count (P) * neutrophil-to-lymphocyte ratio (N/L) (SII = P * N/L ratio). All biopsy specimens were graded for the presence of rejection in according to ISHLT standards by an experienced pathologist. According to the 2010 ISHLT report, the cardiac allograft vasculopathy (CAV) was classified by a retrospective review of all coronary angiographic studies.
Results: Humoral rejection, cellular rejection and CAV was detected in %6.7,%22.3 and %15.2 of the 80 patients, respectively. According to the ROC curve and Youden index, the optimal cutoff value of SII predicted was 848.37, the sensitivity of this point was 65.33, the specificity was 54.21, and the AUC was 0.602. The cut-off value for high SII (≥ 848.3) was used for our study cohort. In this retrospective cohort study, we found that SII levels were significantly associated with the cellular rejection and CAV. Heart transplant patients with cellular rejection had significantly higher levels of SII (p 0.002). Analysis of the relationship between SII and the cellular rejection showed significantly elevated level in rejected patients. In addition, we found statically significant relationship between SII level and CAV in this study (p 0.029).
Conclusions: Numerous studies have been done about hematological markers and host immune reaction. In conclusion, we can use SII level as an auxiliary parameter to determine a rejection and cardiac allograft vasculopathy, which is important for heart transplant patients.

Heart failure

OP-121

Impact of invasively detected cardiac power index on survival in patients with advanced chronic heart failure

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Background and Aim: Advanced chronic heart failure (HF) is associated with high mortality and morbidity rates. Hydraulic function of the heart is identified by cardiac power. Ability of cardiac pumping can be represented by cardiac power output (CPO). CPO is a result of mean arterial pressure and cardiac output (CO). Resting CPO is found to be associated with mortality in acute HF, especially in cardiogenic shock. Cardiac power index (CPI) is CPO indexed to body surface area (BSA). The current study aimed to investigate the effect of resting cardiac power index on the survival in advanced chronic HF patients.
Methods: The study group enrolled 56 patients with advanced chronic HF. All patients had left ventricle ejection fraction <30% and were class III and IV in terms of functional capacity. The patients who underwent right and left catheterization due to advanced chronic HF between January 2017 and March 2020 were included. Left ventricular assist device (LVAD) implantation and heart transplantation operation were exclusion criteria for the study. The patients were followed from catheterization time until June 2020. Follow-up duration was defined as the period from catheterization to either cardiac mortality or June 2020 (if no mortality develops). Cardiac power output [W] was calculated as follows: CPO = CO x mean arterial pressure × K (K=0.0022, conversion factor). Cardiac power index (CPI) was calculated by dividing CPO with body surface area (BSA) CPI [W/m^2] = CPO [W] / (BSA [m^2]).
Results: 19 patients develop cardiac mortality from 56 patients (33.9%) over a period of 16 months (median). CPI was associated with cardiac mortality [0.32 (0.26-0.38) vs 0.42 (0.31 vs 0.52), p=0.003]. In terms of ROC analysis, cut-off level of CPI for cardiac mortality was 0.41 [89.5% sensitivity and 56.8% specificity, AUC: 0.745, p=0.003, 95% CI (0.616-0.875)] (Figure 1). According to this cut-off level, the study group was stratified into two groups. No significant difference was found in baseline clinical and laboratory features (Table 1). Of catheterization findings, reduced CPI (<0.41W/m^2) was also related with high right atrial pressure (p:0.016)

and pulmonary vascular resistance (p=0.012), apart from cardiac output and index (Table 2). A Kaplan-Meier survival analysis indicated that long term survival was found to be significantly reduced in patients with CPI <0.41 (Log Rank p=0.003) (Figure 2).

Conclusions: Resting cardiac power index is related with cardiac mortality in patients with advanced chronic heart failure. CPI below 0.41 level is associated with worse survival rates.

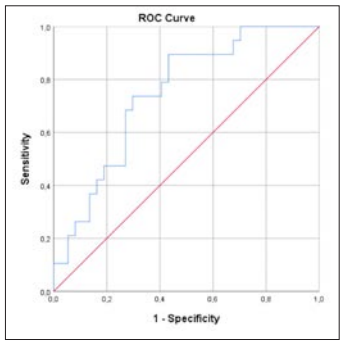

Figure 1. ROC analysis of CPI and cardiac mortality.

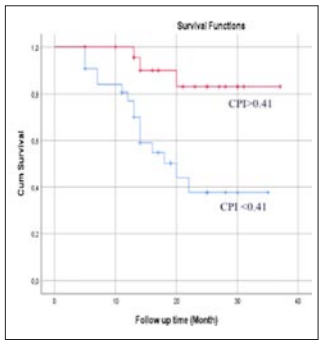

Figure 2. Kaplan-Meier survival curves for low and high CPI (according to cut-off level of 0.41).

Table 1. Baseline clinical, laboratory features of patients

	CPI < 0.41	CPI ≥ 0.41	P value
Age, (years)	53.5 (45.5-59.75)	53.5 (41-61)	0.810
Gender (male), n, %	26 (81.3)	21 (87.5)	0.718
Hypertension, n, %	20 (62.5)	12 (50.0)	0.350
Diabetes Mellitus, n, %	11 (34.4)	4 (25.0)	0.450
Chronic Renal Failure, n, %	8 (25.0)	9 (37.5)	0.314
Cardiomyopathy (ischemic), n, %	15(46.9)	14 (58.3)	0.396
WBC, µl	8.9±2.6	7.8±1.9	0.084
Hemoglobin, g/dL	13.6 (12.1-14.4)	13.4 (11.7-15.1)	0.703
Platelet, 103/ µl	246.8±96.5	221.8±86.4	0.321
MPV, fL	10.7 (10.2-11.4)	10.8 (10.0-11.4)	0.960
RCDW, %	42.1 (40.4-48.4)	41.2 (38.3-46.3)	0.451
CRP, mg/dL	10.4 (3.6-26.5)	7.1 (2.1-16.2)	0.240
Urea, mg/dL	20.1 (16.5-29.0)	20.5 (16.4-32.3)	0.766
Creatinine, mg/dL	1.0 (0.8-1.2)	1.1 (0.9-4.1)	0.177
ALT, U/L	25.5(20.2-39.5)	24.0(12.0-50.0)	0.384
Sodium, mEq/L	138.5 (136-141)	139.5(137.0-141.7)	0.444
Potassium, mEq/L	4.4±0.6	4.4±0.5	0.827
LDL-C, mg/dL	99.8±40.2	97±33.7	0.785
HDL-C, mg/dL	34.7±9.6	39.6±11.7	0.090
Triglyceride, mg/dL	131 (88.5-160.5)	133 (86.5-155.2)	0.954
LV-EF, %	21.3±3.5	23.1±3.8	0.089
TAPSE, mm	15.8(14.7-17.8)	16.5 (14.5-18.9)	0.613

Table 2. Catheterization findings of the patients

	CPI < 0.41	CPI ≥0.41	P value
Cardiac output, L/min	3.3±0.6	4.3±0.8	<0.001
Cardiac index, L/min/m2	1.7 (1.5-1.9)	2.2 (1.9-2.5)	<0.001
Stroke volume, mL	41.1±11.0	52.4±11.7	<0.001
Stroke volume index, mL/m2	21.1±5.0	28.1±6.7	< 0.001
Mean aortic pressure, mmHg	80 (74-88.8)	106 (95.2-124.5)	<0.001
Mean pulmonary artery pressure, mmHg	36.3±11.0	33.9±10.9	0.417
Right atrial pressure, mmHg	12 (8.25-16)	10 (7.25-11.0)	0.016
PCWP, mmHg	24.5±6.1	23.2±7.2	0.476
PVR, Wood Units	3.5(2-5)	2 (1.3-4)	0.012

Heart failure

OP-122

A novel scoring system for the assessment of optimal versus suboptimal adherence to the guideline-directed medical therapy in patients with heart failure and reduced ejection fraction: RBM score

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Background and Aim: Heart failure (HF) guidelines recommend the use of guideline-directed medical therapies (GDMT) including renin–angiotensin system (RAS) inhibitors, beta-blockers, mineralocorticoid antagonists (MRAs) in patients with chronic heart failure and reduced ejection fraction (HFrEF) to reduce mortality and hospitalization rates. Despite the evidence-based recommendations to use of GDMT at maximally tolerated target doses, many studies suggest that HFrEF patients are rarely receive GDMT at the target doses. The aim of the present study is to determine demographic and clinical differences between patients with optimal versus suboptimal adherence to the current HF guidelines.

Methods: Adherence to guideline-directed medical and device Therapy in heArt failure with reduced ejection fraction: ATA study is a prospective, multicenter, observational study conducted in 24 centres from seven geographical regions of Turkey from January 2019 to June 2019. For the assessment of optimal versus suboptimal adherence to the HF guidelines, we designed a novel RBM scoring system (RAS inhibitors + Beta-blockers + MRAs) on the basis of the use of target doses of GDMT (Figure). A high RBM score (≥6) –in other words, use of RAS inhibitors, beta-blockers, and MRAs at least 50% of target doses– is indicative of the optimal adherence to the HF guidelines.

Results: The study included 1,462 outpatients (male: 70.1%, mean age: 67±11 years, mean LVEF: 30±6%) with chronic HFrEF. RAS inhibitors, beta-blockers, and MRAs were prescribed in 78.2%, 90.2%, and 55.4% of the patients, respectively. The proportions of HF patients receiving target doses of GDMT were 24.6% for RAS inhibitors, 9.9% for beta-blockers, and 10.5% for MRAs. The proportions of patients according to the novel RBM score are presented in Figure. Among study population, only 22.9% of HFrEF patients had optimal adherence (RBM score ≥6) to the recommendations of current HF guidelines. Patients with suboptimal adherence (RBM score <6) to the HF guidelines were older and had low level of education and low household income. Patients with optimal adherence had more co-morbidities, including hypertension, diabetes mellitus, peripheral arterial disease, depression and smoking. Patients with optimal adherence were more likely to be on treatment with ivabradin, statins, and diuretics.

Conclusions: Although majority of HFrEF patients in ATA study receive RAS blockers and beta-blockers –but not MRAs–, most eligible patients do not receive target doses of GDMT. Among study population, only 22.9% of HFrEF patients had optimal adherence to the HF guidelines. Suboptimal adherence to the HF guideline was associated with older age, low level of education, and low level of household income. In our opinion, a novel RBM score can be use for individual assessment of adherence to the GDMT of HFrEF patients. This novel scoring system should be tested in larger cohorts to predict mortality and morbidity in patients with HFrEF.

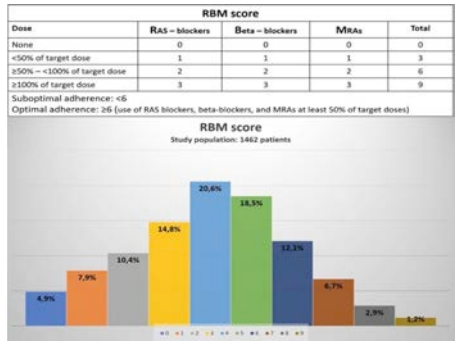

Figure 1.

Heart failure

OP-123

3-year results of a new established heart transplant center

Oğuz Konukoğlu, Özgür Yıldırım

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Background and Aim: Heart transplantation is the gold standard treatment of heart failure. There is no treatment option that can compete with heart transplantation in the end stage heart failure in terms of both expected life expectancy and quality of life. The limited donor pool is the most important obstacle of this treatment. In particular, a serious organization and coordination network should be established in order to form a recipient pool and to be included in the donor system. In this article, we wanted to share the results of our transplant team within 3 years.
Methods: Patients who underwent heart transplantation between March 2017 and February 2020 were included in the study.
Results: 18 (6 women, 12 male) patients were undergone heart transplantation. Of these patients, 11 were ischemic, 5 were dilated, 2 were hypertrophic cardiomyopathy. 7 patients (38.8%) had left ventricular long-term mechanical support (LVAD), and one patient had short-term extracorporeal membrane oxygenator (ECMO) support. There were 2 recipients in the marginal donor/recipient group. In terms of status of emergency, 7 patients were 1a, 10 were 1b, 1 was class 2. In 10 of the 13 patients who lived, acute resection attacks were seen within the first 1 year. There was no mortality due to the reduction attack. When the marginal group was removed, operative mortality was 18.7% (3 patients). All patients in the mortality group were patients under mechanical support. No mortality seen in our follow up group yet.
Conclusions: Heart transplantation is the best option of treatment for the end stage cardiac failure patients. Although mechanical circulatory support is a promising option for the patients who do not have enough time to wait for a donor, first line treatment with transplantation seems as a better option for the smaller lucky group.

Heart failure

OP-124

Prognostic performance of copeptin among patients with acute decompensated heart failure

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Background and Aim: In heart failure (HF), various biomarkers have been established for prognosis. However, little is known about the relevance of copeptin measurements to HF. This study aimed to explore the prognostic value of copeptin for predicting cardiovascular (CV) death or HF-related re-hospitalization in patients with acute decompensated HF.
Methods: We prospectively enrolled 155 consecutive patients with acute signs and symptoms of HF. Plasma copeptin and N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels were measured at admission. Patients were monitored for 90 days regarding the composite endpoint of CV death or acute HF-related re-hospitalization.
Results: Of the 155 patients enrolled, 40 reached the endpoint, and 115 were in a stable condition during follow-up. Patients who reached an adverse endpoint showed higher NT-proBNP and copeptin levels compared to patients in stable condition. Receiver operating characteristic curve analysis revealed that the area under curve of copeptin 0.844 (95% CI, 0.753–0.935) was superior to that of NT-proBNP 0.809 (95% CI, 0.729–0.890) for the prediction of adverse events within 90 days. Meanwhile, compared to the group with lower copeptin levels (<34 pmol/L), patients with higher copeptin levels (≥34 pmol/L) were at a 10.672-times higher risk of CV death or acute HF-related re-hospitalization. Multivariate Cox proportional hazards regression analysis revealed that increased copeptin level was a significantly independent predictor of adverse events (risk ratio, 1.051; 95% CI, 1.020–1.083; p<0.001).
Conclusions: Copeptin was found to be a strong, novel marker for predicting CV death or HF-related re-hospitalization in patients with acute decompensated HF.

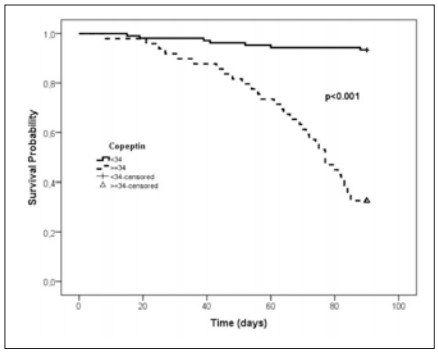

Figure 1. Kaplan–Meier plots showing the proportion of the 155 patients with severe acute decompensated heart failure who were stratified into two groups according to plasma copeptin cut-off point at baseline (straight line, <34 pmol/L, n=106; dashed line, ≥34 pmol/L, n=49; log rank test for trend, p<0.001).

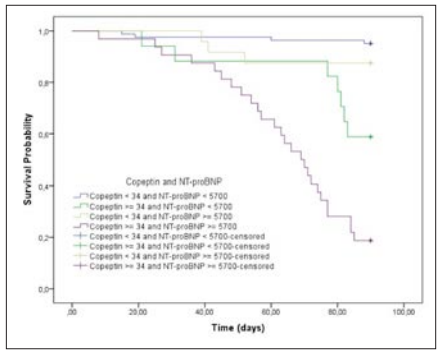

Figure 2. Kaplan–Meier plots showing the proportion of the 155 patients with severe acute decompensated heart failure who were stratified into four groups according to plasma NT-proBNP and copeptin cut-off point at baseline (blue line, <5700 pg/mL for NT-proBNP and 34 pmol/L for copeptin; green line, 5700 pg/mL for NT-proBNP and ≥34 pmol/L for copeptin; yellow line, ≥5700 pg/mL for NT-proBNP and 34 pmol/L for copeptin; purple line, ≥5700 pg/mL for NT-proBNP and ≥34 pmol/L for copeptin). NT proBNP, N terminal pro B type natriuretic peptide.

Heart failure

OP-125

Evaluation of the importance of microtubules and microtubule inhibition through blood β-tubulin 1 levels in patients with heart failure with reduced ejection fraction

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Background and Aim: α- and β-tubulin (TUBA and TUBB) proteins form obligate heterodimers that, through the nucleating activity of γ-tubulin (TUBG), polymerize into microtubules. β-Tubulin is a microtubule that binds with α-Tubulin to form tubulin heterodimer which is a structural protein of cardiomyocytes.Colchicine inhibits tubulin polymerization.The aim of our study is to determine the beta-tubulin level, which is one of the cardiomyocyte structure proteins in serum and to investigate the relationship with heart failure, and also to investigate the importance of microtubulins and microtubule inhibition via β -tubulin-1 levels, which are the building blocks of microtubulins of cytoskeleton filaments in stage 4 heart failure with reduced ejection fraction.
Methods: Fifty patients who diagnosed with Heart Failure with reduced Ejection Fraction (HFrEF) and 30 healthy subjects as a control group were included in our study. Also, we investigated serum tubulin-1 levels according to etiological classifications (ischemic/non-ischemic subgroups). Besides, the subgroup with 13 non-ischemic HFrEF patients using low dose colchicine (0.5-1mg) as a microtubule inhibitor for at least three months was also included in the study. Blood samples have been centrifuged and β- tubulin plasma concentrations were measured by the Elisa method with Human β- tubulin-1 Chain Elisa kit (Bioassay Technology Laboratory).
Results: We found higher levels of β-Tubulin 1 in HFrEF patients from the healthy control group. But statistical analysis between the two groups showed no significant difference (p=0.6). Also, in subgroup analysis, β- tubulin levels had increased in ischemic HFrEF patients according to the non-ischemic subgroup (p=0.26). Besides, in the subgroup analysis of non-ischemic HFrEF patients used colchicine (n=13) was detected decreased the levels of β- tubulin (p=0.29) and NT*proBNP (p=0.69). But, in for at least three months low dose colchicine used patient subgroup had better EF (p=0.009) and smaller diastolic left ventricular diameters (p=0.002) respectively (Table 1). When the relationship between ejection fraction and β-tubulin levels of the HFrEF group in the study was examined, we found that patients with lower ejection fraction had higher β- tubulin levels. (p=0.018, R^2: 0.086). When HFrEF groups with ischemic and non-ischemic etiology were compared, renal functions were similar (p=0.88). There was also no significant correlation between creatinine and eGFR levels and β- tubulin (p=0.482).
Conclusions: Our study gives the impression that the adverse remodeling of the left ventricle can be reversed by low dose colchicine.Thus, actin microfilaments are more effective in the environment, and due to the inhibition of microtubules, they bind more to myosin heads (myotropic effect). Therefore, microtubule inhibitor drugs such as colchicine should be investigated for long time treatment options in heart failure.

Table 1. Parameters A, B, C

Parameters A	Patient(N=50)	Control(N=30)	P -Value
NT-proBNP(pg/dl)	2230.4±207.7	53.6±21.5	0.01
Beta-Tubulin(pg/dl)	97.4±174.8	81.3±172.7	0.60
Parameters B	Ischemic (N: 25)	Non-ischemic (N: 25)	P -Value
NT-proBNP(pg/dl)	2647±2182	1813±1924	0.01
Beta-Tubulin(pg/dl)	108±222	53±95	0.26
Parameters C	Colchicine + (N:13	Colchicine – (N:37)	P -Value
NT-proBNP(pg/dl)	2029.9±2344	2300±2000	0.69
BETA-Tubulin(pg/dl)	37±81	97±194	0.29
EF(%)	32.6±6.4	27.5±3.2	0.009
LVD (mm)	58.1±8.2	66.1±9.0	0.002
TAPSE(mm)	18.4±3.0	19.8±2.7	0.33

A): Average of EF values, NT-ProBNP and Beta-tubulin levels of Patients with Heart Failure (N: 50) and Control Group (N: 30) Participating in the Study(mean ± SD). B) NT-proBNP and Beta-tubulin values of patients with heart failure according to ischemic (N: 25) and non-ischemic (N: 25) etiology included in the study(mean ± SD). C)Differences in NT-ProBNP and Beta-tubulin levels and echocardiographic parameters of for at least three months colchicine treated and non-colchicine patients in the HFrEF patient group (N: 50) included in the study (mean ± SD).

Heart failure

OP-126

Sacubitril/valsartan treatment may be associated with improved cardiac repolarization parameters

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Background and Aim: Patients with low ejection fraction heart failure (HF) have a high risk of sudden cardiac death (SCD). The main cause of SCD in these patients is ventricular arrhythmias. Comparing patients using sacubitril/valsartan with enalapril compared to PARADIGM-HF study, the rate of hospitalization and death due to HF was significantly lower. In addition, SCD and fatal cardiac arrhythmias were found to be less. In a study by Diego et al., They found a decrease in the incidence of paroximal atrial tachycardia and atrial fibrillation, in patients receiving sacubitril/valsartan. In a retrospective study, switching from angiotensin-converting enzyme inhibitors or angiotensin receptor blockers to sacubitril/valsartan in patients with Implantable cardioverter-defibrillator implantation caused a decrease in ventricular arrhythmias with appropriate treatment. Delayed ventricular repolarization is associated with ventricular arrhythmias. Ventricular repolarization can be determined on electrocardiography (ECG) using QT interval, QT dispersion and T-wave measurements. Recent studies have revealed that the Tp-e interval, the interval between the peak and the end of the T wave, is specified as an index of total dispersion of repolarization. Prolonged Tp-e interval may predict ventricular arrhythmias and mortality. Therefore, Tp-e/QT ratio was suggested to be a better marker of ventricular depolarization. Frontal plane QRS-T [f(QRS-T)] angle, which is defined as the angle between the directions of ventricular depolarization (QRS axis) and repolarization (T axis), is a novel marker of ventricular repolarization heterogeneity. It can be calculated from surface ECG by subtracting the QRS axis from the T axis. Prognostic value of this easily available parameter has been shown in different populations. Impaired repolarization is known to be associated with sudden cardiac deaths. One of the mechanisms responsible for Sacubitril/valsartan treatment being associated with a decrease in SCD may be its healing effects on impaired repolarization. In the light of this information, we aimed to investigate the potential relationship between Sacubitril/valsartan treatment and cardiac repolarization parameters.
Methods: The study included 26 patients who started Sacubitril/valsartan therapy due to HF. The ECG was evaluated before the treatment and on the 90th day of the treatment. ECG repolarization parameters were compared before and after treatment.
Results: A statistically significant increase has been detected in, Tp-e/QT ratio (p=0.026), Tp-e/corrected QT ratio (p=0.017), Tp-e interval (p=0.004) and QRS- T angle (p<0.001).
Conclusions: In our study, Sacubitril/valsartan treatment was found to be associated with an improvement in cardiac repolarization parameters. One of the mechanisms of sacubitril / valsartan treatment to prevent SCD may be that it causes improvement in cardiac repolarization. However, our hypothesis should be supported by larger and more comprehensive studies.

Table 1. Demographic and echocardiographic features of the study group

Parameters	(n=26)
Age, years	52.98 ± 5.07
Female, n (%)	9 (34.6%)
BMI, kg/m2	27.6 ± 2.3
Ischemic Etiology, n(%)	19 (73.1%)
Hypertension, n (%)	10 (38.5%)
Diabetes Mellitus, n (%)	9 (34.6%)
Dyslipidemia, n (%)	6 (23.1%)
Smoking, n (%)	10 (38.5%)
Ejection fraction, (%)	27.88 ± 4.26
NYHA Class	1.73 ± 0.60

Data are given as mean ± SD, n or median (interquartile range). BMI, Body mass index; NYHA Class, New York Heart Association Classification.

Table 2. Comparison of ECG parameters before Sacubitril/valsartan treatment and 90. day of the treatment

Parameters	Before theraphy	90. day of theraphy	p value
QT interval, ms	346.1 ± 26.4	357.6 ± 32.0	0.088
QTc interval, ms	388.7 ± 36.0	390.5 ± 30.40	0.865
Tp-e interval, ms	83.5 ± 12.1	77.2 ± 10.1	0.004
Tp-e/QT ratio	0.24 ± 0.04	0.22 ± 0.04	0.026
Tp-e/QTc ratio	0.22 ± 0.04	0.20 ± 0.03	0.017
f(QRS/T) (°)	61.19 ± 29.6	86.33 ± 31.6	<0.001

Data are given as mean ± standard deviation. QTc, Corrected QT interval; f(QRS/T), Frontal plane QRS-T angle.

Heart failure

OP-127

Prognostic value of right ventricular stroke work index in ambulatory patients with advanced heart failure

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Background and Aim: Right myocardial contractility plays a pivotal role in patients with advanced heart failure (HF). Right ventricular stroke work index (RVSWI) is considered as a hemodynamic measure of right ventricular contractility. The present work aims both to evaluate the prognostic value of the RVSWI at rest in outpatients with advanced HF and to discuss the misinterpretation of the measurement unit for RVSWI.
Methods: Between September 2010 and July 2013, 172 outpatients with advanced HF admitted to the tertiary centre for optimization of drug therapy and or to be evaluated for ventricular assist device (VAD) therapy and or to be evaluated for heart transplantation (HTx) were enrolled in this study. At baseline, we performed right heart catheterization for each patient. Then we assessed the hemodynamic parameters with longitudinal follow-up for adverse outcomes such as cardiac mortality, VAD therapy, and HTx.
Results: During a median follow-up period of 52 months, we observed 50 cardiac deaths, 12 VAD implantations and 10 HTx. A threshold for RVSWI at rest of 7.35 centijoule/m^2 (cJ/m^2) was ascertained (Area 0.75, %95 confidence interval 0.67-0.82, p=0.001). Decreased RVSWI at rest (<7.35 cJ/m^2) was correlated with increased adverse events. The prognostic value of RVSWI at rest remained significant after adjustment for age, gender, pulmonary vascular resistance and right atrial pressure (hazard ratio 0.80, 95% confidence interval 0.70-0.91, p=0.001).
Conclusions: Lower RVSWI at rest is an independent predictor of adverse outcomes. Hence, it could be used for individual risk stratification in patients with advanced HF. On the other hand, this study aims to eliminate the misinterpretation of the measurement unit for RVSWI in all future scientific papers.

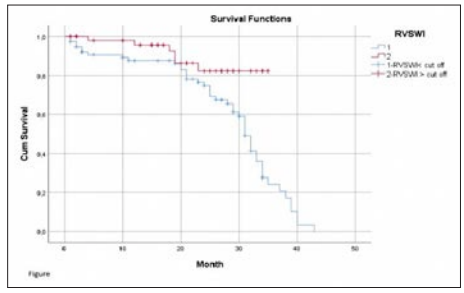

Figure 1. Kaplan-Meier estimates for low and high right ventricular stroke work index (RVSWI) at rest levels.

Table 1. Baseline characteristics

Variable	RVSWI ≤ 7.35 cj/m2 (n = 76)	RVSWI > 7.35 cj/m2 (n = 53)	p-value
Age (years)	58.2 ± 11.5	60.7 ± 11.6	0.21
Men, n (%)	55 (72.4%)	40 (75.5%)	0.69
Hypertensiyon, n (%)	30 (39.5 %)	16 (30.2 %)	0.27
Diabetes mellitus, n (%)	30 (39.5%)	16 (30.2%)	0.27
COPD, n (%)	13(17.1%)	4 (7.5%)	0.11
CRT, n (%)	33 (43.4%)	18 (34.0 %)	0.28
CKD, n (%)	20 (26.3%)	9 (17.3%)	0.23
CVH, n (%)	6 (7.9%)	2 (3.8%)	0.34
Hyperlipidemia, n (%)	30 (39.5%)	16 (34.8%)	0.27
LVEF (%)	28.3 ± 5.4	28.8 ± 6.6	0.63
mPAP (mm Hg)	32.9 ± 12.5	34.3 ± 10.9	0.51
PCWP (mm Hg)	22.0 ± 8.8	21.5 ± 8.4	0.72
PVR (dyn/sn/cm5)	273.6 ± 225.2	281.6 ± 193.3	0.83
SVR (dyn/sn/cm5)	1759.5 ± 552.0	1570.7 ± 591.9	0.066
CO (l/dk)	3.8 ± 1.1	4.0 ± 0.9	0.20
CI (l/dk/m2)	2.0 ± 0.5	2.1 ± 0.4	0.50

Heart failure

OP-128

Interleukin - 26 plays a cardioprotective role in cardiac repair after ST - segment elevation myocardial infarction

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Background and Aim: Recently discovered Interleukin (IL)-26, a sub-family of IL-10; is produced by macrophages, natural killer cells, T helper (Th)-1 and Th17 cells, and has been shown to play a role in the etiopathogenesis of autoimmune diseases such as colitis, rheumatoid arthritis and psoriasis. This study is the first pilot study investigating the relationship between post - STEMI cardiac repair and IL-26.
Methods: 41 patients who were diagnosed with STEMI above 50 years of age and underwent primary percutaneous coronary intervention in the left anterior descending coronary artery and/or left circumflex coronary artery were included in the study. Cardiac functions were evaluated by magnetic resonance imaging at the 2nd week and 6th month of post – MI period. Osteocalcin levels were measured with enzyme-linked immunosorbent assay at post - MI 1st day, 2nd week and 6th week. The percent change in follow-up times was shown by Δ. The change (Δ) was calculated post MI 6th week-1st day for IL-26, and Δ left ventricular end diastolic volume (LVEDV) and left ventricular end systolic volume (LVESV) = post MI 6th months-6th week.
Results: The mean age of the research population was 57.8±5.9 years and 87.8% (n=36) were males. In post-MI 6th month, 17 patients were identified with adverse remodeling (AR) and 24 patients with reverse remodeling (RR). The median IL-26 level was higher in the AR group compared to the RR group at post - MI day 1 (80.2 vs 51.1; p<0.001), at post - MI week 2 (48.1 vs 52.8; p=0.578) and week 6 (37.1 vs 54.1; p=0.266), no significant difference was detected. While IL-26 level decreased significantly in post-MI 2nd week in AR patients, IL-26 level was similar in post-MI 1st day and others follow-up in RR patients. There was a negative correlation between the changes in post-MI ΔIL-26 levels and ΔLV EDV and ΔLV ESV (respectively; r=-0.549, p<0.001; r=-0.484; p<0.001).
Conclusions: IL-26 expression, an anti - inflammatory cytokine, remains permanently high in RR patients after MI. We can say that this is related to the cardioprotective effect of IL-26 in cardiac repair in post-MI.

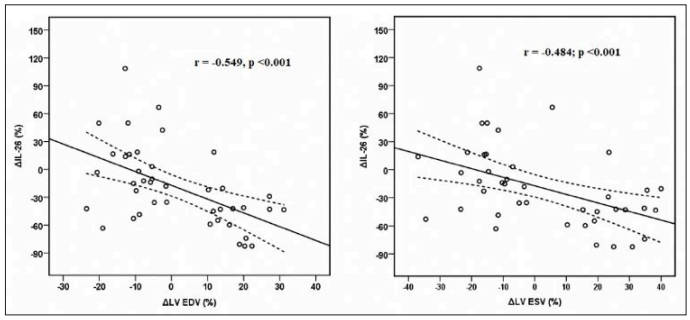


Figure 1. Correlation between ΔIL-26 and ΔLV EDV and ΔLV ESV. LV, left ventricle; EDV, end-diastolic volume; ESV, end-systolic volüme; IL, interleukin; Δ, the percentage change in the follow-up period.

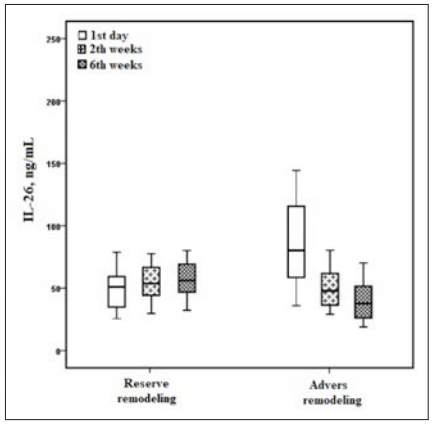


Figure 2. Distribution of Interleukin - 26 levels according to follow-up in remodeling groups. While IL-26 level decreased significantly in post-MI 2nd week in AR patients, IL-26 level was similar in post-MI 1st day and others follow-up in RR patients.

Table 1. Cardiac magnetic resonance imaging (CMR) findings and Interleukin - 26 levels

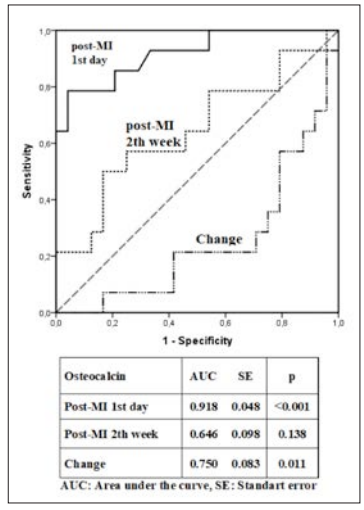
Variables	Reverse remodeling n = 24	Advers remodeling n = 17	p
CMR findings			
2th week			
LVEF, %	45.3±9.8	48.6±11.4	0.328
LV EDV, mL	169.3±37.1	153.9±24.6	0.144
LV ESV, mL	90(62; 146)	85(55; 122)	0.284
Cardiac output, L/min	4.4±0.9	4.8±1.2	0.193
Cardiac index, L/min/m2	2.5±0.4	2.6±0.4	0.287
6th week			
LVEF, %	50.3±8.5	44.2±11.4	0.048*
LV EDV, mL	151.8±34.1	181.4±29.0	0.006*
LV ESV, mL	78(58; 122)	105(69; 154)	0.045*
Cardiac output, L/min	4.7±0.9	4.6±1.5	0.650
Cardiac index, L/min/m2	2.7±0.4	2.6±0.5	0.310
IL-26, ng/mL			
1st day	51.1(34.8; 59.7)	80.2(57.5; 118.5)	<0.001*
2th week	52.8(33.4; 52.3)	48.1(26.8; 52.6)	0.578
6th week	54.1(34.3; 57.7)	37.1(25.5; 51.7)	0.266

*Numerical variables were shown as mean ± standard deviation or median (IQR:25-75). * p<0.05 shows statistical significance. Abbreviations: LV, left ventricle; EF, ejection fraction; EDV, end-diastolic volume; ESV, end-systolic volüme; IL, interleukin.*

resonance imaging at the 2nd and 6th months of post – MI period. Osteocalcin levels were measured by enzyme-linked immunosorbent assay in post - MI 1st day and 2nd week.

Results: The mean age of the research population was 53.5 ± 5.3 years and 89.5% (n=34) were males. The rate of those who developed AR in post-MI 6th month was 36.8% (n=14). The median osteocalcin levels were found to be higher in post - MI 1st day (1458.1 vs 2946.7; p<0.001) compared to those who did not develop AR, while it did not differ significantly in post - MI 2nd week (3200.3 vs 3729.5; p=0.144). While osteocalcin expression did not change significantly in the 2nd week compared to baseline in those who developed AR, it was determined that it increased significantly in those who did not develop AR. There was a negative correlation between changes in post - MI osteocalcin levels (post MI week 2-day 1) and left ventricular end diastolic volume and left ventricular end systolic volume changes (respectively; r=-0.446, p = 0.005; r=-0.476; p=0.003). The diagnostic performance of osteocalcin expression on post-MI 1st day in predicting AR was found to be high (AUC±SE=0.918±0.05; p<0.001).

Conclusions: We can say that osteocalcin expression after post-MI may play a role in the pathogenesis of AR, and high osteocalcin expression in early stage is associated with worse cardiac repair.



Osteocalcin	AUC	SE	p
Post-MI 1st day	0.918	0.048	<0.001
Post-MI 2th week	0.646	0.098	0.138
Change	0.750	0.083	0.011

AUC: Area under the curve, SE: Standart error

Figure 1. Diagnostic performance assessment of osteocalcin levels in predicting adverse cardiac remodeling according to the follow-up.

Table 1. Cardiac magnetic resonance imaging findings and Osteocalcin levels

Variables	Non-adverse remodeling n = 24	Adverse remodeling n = 14	p
CMR findings			
2th week	46.7±10.2	46.8±11.5	0.983
LVEF, %	162.5±35.2	162.2±27.8	0.982
LV EDV, mL	81(60.5;126)	103(56;141)	0.424
LV ESV, mL	4.2±0.7	4.4±1.5	0.391
Cardiac output, L/min	2.5±0.4	2.6±0.5	0.311
Cardiac index, L/min/m2			
6th week	51.9±8.8	42.5±11.8	0.008*
LVEF, %	141.4±30.1	191.3±32.5	<0.001*
LV EDV, mL	69.5(53;102)	132(70;186)	<0.001*
LV ESV, mL	4.7±0.7	4.5±1.4	0.661
Cardiac output, L/min	2.6±0.5	2.5±0.4	0.231
Cardiac index, L/min/m2			
Osteocalcin, ng/mL	1458.1(1251.1;1847.7)	2946.7(2700.5;3210.1)	<0.001*
1st day	3200.4(2523.3;3652.5)	3729.5(2978.4;4724.6)	0.144
2th week			

*Numerical variables were shown as mean ± standard deviation or median (IQR:25-75). *p<0.05 shows statistical significance. Abbreviations: LV, left ventricle; EF, ejection fraction; EDV, end-diastolic volume; ESV, end-systolic volume.*

Heart failure

OP-129

Potential role for osteocalcin in the development of adverse cardiac remodelling in non-geriatric STEMI patients: First pilot research

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Background and Aim: Acute myocardial infarction (MI), followed by negative progression of histopathological changes, leads to the development of adverse remodeling (AR) by causing structural and functional impairments in the myocardial region. This event plays an important role in the pathogenesis of heart failure. Osteocalcin, synthesized by osteoblasts, has been shown to be an important predictor of atherosclerosis, vascular calcification, stroke and cardiac diseases, as well as bone tissue development. In this study, the relationship between the development of AR after MI and osteocalcin was investigated. It is the first pilot study in the literature.

Methods: 38 non-geriatric patients who were diagnosed with STEMI over 18 years of age and who underwent primary percutaneous coronary intervention in the left anterior descending coronary artery and/or left circumflex coronary artery were included in the study. Cardiac functions were evaluated by magnetic

Heart failure

OP-130

Changing the end of the story; can everything return to normal in a patient with heart failure due to uremic cardiomyopathy?

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Background and Aim: Chronic kidney disease (CKD) is a global public health problem that shortens lifespan primarily by increasing the risk of cardiovascular diseases. Although the dominant phenotype in these patients is consist of left ventricular hypertrophy, myocardial fibrosis and related diastolic dysfunction; heart failure with reduced ejection fraction prevails in a small group. In addition, due to the high risk of mortality in these patients with heart failure; the kidney transplantation operation, which is the only treatment of ESRD, is often not encouraged. From this point we planned a prospective study (between January 2018 and December 2019) to follow patients who have heart failure (without any primary heart diseases) and being prepared for transplantation with end-stage renal failure. The aim of this study was to investigate whether myocardial functions were changed after renal transplantation.

Methods: For this purpose we have seen 968 patients as a renal transplantation candidate between February 2018 and September 2019. 314 patients were excluded from the study due to their previous heart surgery, previous coronary stent implantation and heart valve disease. Among the remaining patients 20 renal transplant candidates with end stage heart failure (ef<45%) without primary heart disease) and 22 patients with normal left ventricular systolic function were chosen to form study groups. Detailed echocardiography, magnetic resonance imaging of the heart, right and left heart catheterization, coronary angiography and endomyocardial biopsy was performed for further examination of cardiac tissue prior to transplantation as is the case in routine practice, for those with markedly impaired left ventricular systolic function. Echocardiography was repeated on the first, third and 6th months following transplantation and compared with the pretransplant values.
Results: In the follow-up of all three groups for 6 months, there was no difference in terms of change in Glomerular Filtration rates over time (p>0.05); In the heart failure group, the ejection fraction global longitudinal left ventricular strain (GLS) values increased over time and reached normal values. There was no significant change in ejection fraction and GLS over time in the control group with normal ejection fraction (p=0.001 for Month 1, p<0.001 for Month 3, p<0.001 for Month 6). Additionally; EF and GLS increased with a continuous linear fashion and reached normal EF levels in recipients with reduced EF heart failure (p<0.05).
Conclusions: Since myocardial systolic functions and myocardial deformation parameters improved significantly after 6 months of follow-up in all 20 patients who were at high risk at the beginning; we can say that uremic cardiomyopathy can be reversible when treated with kidney transplantation, although it is not yet being prevented in the course of kidney failure.

Heart failure

OP-131

Is there a relationship between diastolic global longitudinal strain rate and mortality, rehospitalization and length of hospital stay in patients with heart failure with reduced ejection fraction?

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Background and Aim: Heart failure is still a major cause of mortality in the world, and its incidence continues to increase recently. The increase in mortality rates directed clinicians to different searches in order to predict the prognosis of the patients and therefore it was aimed to detect them at the microvascular level before the disease becomes more evident. Therefore, tissue doppler parameters, which have gained popularity in recent years, are promising. Echocardiographically, early changes in the wall tension of the left ventricle have contributed greatly to the prognosis of heart failure patients and especially to mortality prediction. In this study, we aimed to investigate the relationship of diastolic global longitudinal strain rate with mortality, rehospitalization and hospitalization time in patients with heart failure with reduced ejection fraction.
Methods: The clinical, laboratory and echocardiographic parameters of 116 patients with EF ≤40% in New York Heart Association class 3 and 4 symptoms who our hospitalized in the cardiology clinics were evaluated within the first 24 hours. Fifty eight people without heart failure as control group were included in the study. Echocardiographic measurements, tissue doppler parameters and strain rate were measured. N-terminal pro-brain natriuretic peptide was measured in addition to standard biochemical and hematological parameters.
Results: There was no significant difference between the patient and control groups in terms of clinical and demographic features (Table 1). Echocardiographic parameters and tissue doppler parameters of left ventricular size, performance and functions were more significant in the patient group arm (Table 2). E strain rate and E / E'SR were statistically significant in patients with one month mortality (p=0.009) (Table 3). The relationship of these parameters with in-hospital mortality and rehospitalization is weak and not statistically significant (p>0.05).
Conclusions: It is a superior parameter compared to other tissue doppler parameters to show prognosis and mortality in E strain rate and E / E'SR in patients with heart failure. E / E'SR is a left ventricular diastolic function indicator superior to other tissue doppler parameters.

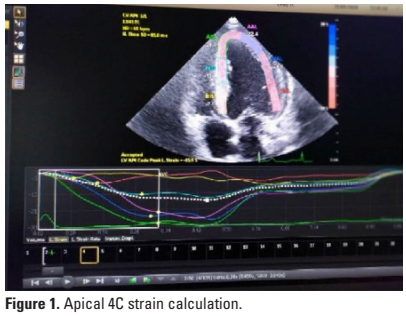

Figure 1. Apical 4C strain calculation.

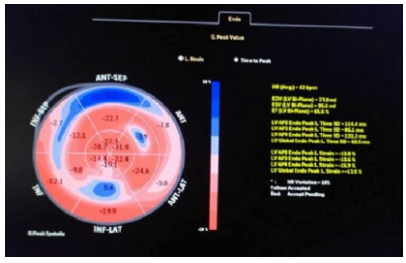

Figure 2. GLS calculation.

Table 1. Demographic, clinical and laboratory features of the patient and control groups

Demographic features	Patient group (116) Mean±SD/Median (IQR)	Control group (58) Mean±SD/Median (IQR)	P value
Age (year)	67.7±12.8	68.1±11.2	0.81
Sex Male Female	 71 (%61.2) 45 (%38.8)	 36 (%62.1) 22 (%37.9)	0.524
BMI (kg/m2)	28.7±6.3	29.0±5.2	0.75
Heart rate (bpm)	84.0±22.0	80,0±11.0	0.178
Systolic BP (mmHg)	122.0±18.8	125.4±13.6	0.216
Diastolic BP (mmHg)	73.09±12.4	74.5±7.9	0.416
CAD, n (%) None Medical treatment PCI CABG	 41(%35.3) 15(%12.9) 39(%33.6) 21(%18,1)	 35(%60.3) 5(%8.6) 11(%19) 7(%12.1)	0.019
Hypertension, n (%)	84 (%72.4)	47 (%81)	0.145
Smoker, n (%)	40(%34.5)	15(%25.9)	0.321
Diabetes mellitus, n (%)	52 (%44.8)	34(%58.6)	0.06
Family history, n (%)	32(%27.6)	8 (%13.8)	0.03
Hyperlipidemia, n (%)	49 (%42.2)	23 (%39.7)	0.43
Alcohol, n (%)	19 (%16.4)	1 (%1.7)	0.002
CVA, n (%)	16 (%13.8)	6 (%10.3)	0.35
CKD, n (%)	53 (%45.7)	21 (%36.2)	0.151
COPD, n (%)	23 (%19.8)	10 (%17.2)	0.424
AF, n (%)	34 (%29.3)	10 (%25.3)	0.059

Table 2. Echocardiographic features of the patient and control groups

Echocardiographic features	Patient group (116) Mean±SD/Median (IQR	Control group (58) Mean±SD/Median (IQR)	P value
EDD (cm)	56.8±7.7	45.3±4.5	0.001
ESD (cm)	44.3±8.7	27.8±4.0	0.001
LA (cm)	43.5±7.0	34.8±6.4	0.001
PAPs (mmHg)	35(21)	30(9)	0.001
Right ventricular S	9.7±2.6	12.7±3.1	0.001
Right ventricular E	9.4±3.2	11.4±3.5	0.001
Lateral S	6.2±2.0	9.1±2.0	0.001
Lateral E	7.3±2.0	9.8±3.0	0.001
Septal S	5.3±1.2	8.0±1.8	0.001
Septal E	5.3±1.5	7.9±2.1	0.001
EDV (ml)	182.8 (90)	42,4 (32)	0.001
ESV (ml)	122.5 (60)	17.3 (14)	0.001
EF (%)	30.6 (11)	57.1 (13)	0.001
Mitral E	70.2±18.5	63.7±18.5	0.001
Mitral A	60.7±23.0	78.0±16.9	0.001
Apical 4C strain	-7.3±2.6	-11.4±3.6	0.001
Apical 3C strain	-6.7±2.9	-9.3 ±3.2	0.001
GLS	-7.2±2.46	-10.5±2.6	0.001
E strain rate	0.34 (0.4)	1.0 (0.5)	0.001
A strain rate	0,3 (0)	1.1 (1)	0.001
E/E' SR	188,8 (323	54.1 (21)	0.001

Table 3. The relationship of demographic, laboratory and echocardiographic parameters with one-month mortality and survival in the patient group

	Mortality (16) Mean±SD/Median (IQR)	Survival (100) Mean±SD/Median (IQR)	P value
Heart rate (bpm)	94.9±18.9	82.1±22.0	0.030
Beta blocker, n (%)	8 (%50)	75 (%75)	0.043
ACEİ, n (%)	5 (%31.2)	59 (%59)	0.036
EDD (cm)	52.7±8.7	57.4±7.3	0.021
LA (cm)	40.4±8.2	44.0±6.6	0.049
ProBNP (ng/L)	13800 (30747)	4860 (9174)	0.002
E strain rate	0.1 (0.3)	0.36 (0.3)	0.004
E/ E'SR	600 (679)	184.4 (318)	0.009

Heart failure

OP-132

Frequency of vitamin D insufficiency and deficiency in heart failure patients-single center registry in Turkey

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Background and Aim: Vitamin D is probably a cardioprotective and immunomodulator on the cardiovascular system with its pleiotropic functions. Although low vitamin D concentrations have been reported to be associated with negative outcomes in selected populations with heart failure (HF), the cause-effect relationship between the two remains uncertain. The aim of this study is to evaluate the frequency of vitamin D deficiency and insufficiency in patients with HF in the cardiology outpatient clinic and to determine the factors associated with vitamin D concentrations.
Methods: This is a single-center observation study conducted in a tertiary hospital in Turkey. Patients who were followed up in the outpatient clinic with the diagnosis of HF were included in the study. The classification of patients according to vitamin D levels was made as vitamin D deficiency 25 (OH) D <20 ng / mL, insufficiency 21-29 ng / mL and sufficiency at least 30 ng / ml according to Endocrine Society Vitamin D Guidelines.
Results: 622 patients whose vitamin D levels were monitored in the heart failure outpatient clinic were included in the study. While the median ejection fraction of the patients were 30% (25-30), 74.9% were NYHA I-II and 25.1% were NYHA III-IV patients. Vitamin D deficiency was found in 374 patients (60.1%) and vitamin D insufficiency in 170 patients (27.3%), while vitamin D was sufficient in 78 patients (12.6%). Vitamin D deficiency was observed in 64.3% of women and 58.6% of men, while vitamin D insufficiency was observed in 20.8% of women and 29.7% of men and no significant difference was found between them (p=0.073). When vitamin D deficient, vitamin D insufficient, and vitamin D normal patients were compared, there was a significant difference between the heart rate, albumin level, calcium level, hemoglobin level, serum iron level, transferrin saturation and parathormone level. The relationship between vitamin D deficiency and baseline characteristics is shown in Table 1.
Conclusions: These data showed us that vitamin D deficiency and insufficiency are very common in HF patients. This study also showed its association with factors known to be associated with poor prognosis, such as increased heart rate, anemia, parathormone elevation, and iron deficiency. We think that replacing vitamin D deficiency and correcting the related factors may improve prognosis.

Table 1. Baseline characteristics according to status of vitamin D deficiency

	Vitamin D deficiency (n:374, 60,1%)	Vitamin D insufficiency (n:170, 27,3%)	Vitamin D sufficiency (n:78, 12,5%)	P-value
Vitamin D level (ng/mL)	13,24 (9,87-16,45)	23,95 (21,90-26,99)	35,05 (32,35-43,60)	<0,001
Female	108 (28,9%)	35 (20,6%)	25 (32,1%)	0,073
Male	266 (71,1%)	135 (79,4%)	53 (67,9%)	
Age	64 (53,5-72,5)	64 (55-72)	65 (58,5-77,2)	0,136
Heart rate(beat/min)	78 (68- 87)	73 (63-86)	75 (66-91)	0,039
LVEF (%)	30 (20-35)	30 (25-35)	30 (25-35)	0,916
NYHA I-II	269 (72.5%)	140 (81%)	57 (73,1%)	0,103
NYHA III-IV	105 (27.5%)	30 (19%)	21 (26,9%)	
Creatinine(mg/dL)	1,1 (0,94-1,38)	1,1 (0,93-1,33)	1,14 (0,96-1,40)	0,488
eGFR (mL/min/1.73m²)	65 (48,35-80,84)	66 (52,58-85,50)	60,17 (49,80-80,40)	0,157
Albumin (g/dL)	4,1 (3,8-4,4)	4,3 (4-4,6)	4,2 (3,9-4,4)	0,001
Calcium (mg/dL)	9,3 (9-9,7)	9,5 (9,2-9,9)	9,5 (9-9,82)	0,009
Hemoglobin (g/dL)	12,9 (11,55-14,30)	13,5 (12,3-14,9)	13,6 (11,5-14,7)	0,006
Serum iron (µg/dL)	63 (40-88)	78 (53-105)	67 (40-96)	<0,001
Ferritin (µg/L)	63 (32-111,25)	62,5 (34-109)	51,5 (24,75-100,25)	0,140
Transferrin (mg/dL)	272,5 (235-311,5)	273 (235-307)	285,5 (253,25-320)	0,180
Transferrin saturation (%)	17,75 (11,62-25,99)	22,69 (14,23-30,37)	17,92 (9,79-29,94)	0,002
Parathyroid hormone (ng/L)	71,5 (46,25-102,75)	54 (40-81,5)	49 (35-84)	<0,001
NT-proBNP (ng/L)	1932,5 (722,02-4901)	1440 (377,90-3414,5)	1858,5 (738,2-5058,2)	0,109
CRP (mg/dL)	5 (2-12,6)	4 (2-9)	4 (2-12)	0,119

Vitamin D, 25(OH)D, 25-hydroxy-vitamin D; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; eGFR, estimated glomerular filtration rate; NT-proBNP, N-terminal pro-brain natriuretic peptide; CRP, C-reactive protein. Normally distributed data are presented as mean+SD. Non-normally distributed data are presented as median (interquartile range).

Heart failure

OP-133

Gender-related clinical and management differences in patients with chronic heart failure with reduced ejection fraction

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Background and Aim: Gender-related differences have been described in the clinical characteristics and management of patients with chronic heart failure with reduced ejection fraction (HFrEF). However, published data is conflictive in this regard.
Methods: We investigated differences in clinical and management variables between male and female patients from the ATA study, a prospective, multicenter, observational study that included 1462 outpatients with chronic HFrEF between January and June 2019.
Results: Study population was predominantly male (70.1%). In comparison to men, women with chronic HFrEF were older (66±11 years vs. 69±12 years, p<0.001), suffered more hospitalizations, and presented more frequently with NYHA class III or IV symptoms. Ischemic heart disease was more frequent in men, whereas anemia, thyroid disease, and depression were more frequent in women. No difference was seen between genders in the use rate of renin–angiotensin system inhibitors, beta-blockers, mineralocorticoid receptor antagonists, or ivabradine, or in the proportion of patients achieving target doses of these drugs. Regarding device therapies, men were more often treated with an implantable cardioverter-defibrillator and women received more cardiac resynchronization therapy.
Conclusions: In summary, although management seemed to be equivalent between genders, women tended to present with more symptoms, require hospitalization more frequently, and have different comorbidities than men. These results highlight the importance of gender-related differences in HFrEF and call for further research to clarify the causes of these disparities. Gender-specific recommendations should be included in future guidelines in HFrEF.

Heart failure

OP-134

Impact of sacubitril – Valsartan on sleep quality in patients with congestive heart failure

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Background and Aim: Sacubitril – Valsartan is a new agent approved for the treatment of congestive heart failure patients with reduced ejection fraction especially in symptomatic patients with essential treatment. Previous studies declared a negative correlation between sleep quality and severity of heart failure. Impaired quality of night sleep is associated with poor prognostic outcomes in congestive heart failure.
Methods: 117 patients with congestive heart failure on angiotensin receptor neprilysin inhibitor (ARNI), angiotensin converting enzyme inhibitors (ACE-I) or angiotensin receptor blocker (ARB) treatment were eligible for this study and 109 patients accepted survey questionnaire. Sleep quality was measured by the Pittsburgh Sleep Quality Index (PSQI). Patients were asked 24 questionnaires including subjective sleep quality, sleep latency, sleep time, conventional sleep efficacy, sleep disorder, sleep drug use and daytime dysfunction.
Results: This study included 49 patients on ARNI and 60 patients on ACE-I or ARB treatment. The mean age of patients were 67.5±7.40 vs 64.3±8.20; p=0.620, respectively. Left ventricular ejection fraction was lower in ARNI group than ACE-I and ARB group but not statistically significant (35.5%±4.8 vs 38.6%±3.9; p value: 0.234). PSQI score in ARNI group was 6.75±4.1 and in ACE-I and ARB group was 6.30±3.8 (p value 0.320). 34.6% (n=17) of patients in the ARNI group and 40% (n=24) of the patients in the ACE-I and ARB group complained about poor sleep quality at night (p=0.055).

Conclusions: Patients suffered from nocturnal dyspnea and frequent breathing shortness attacks usually awakens patient and causes poor quality of night sleep. According to subjective evaluations of patients with congestive heart failure, sleep quality was better monitored in ARNI group than others, and the difference may have been due to better control of congestion symptoms in ARNI users. Insomnia and impaired quality of night sleep negatively impact quality of life. Clinical trials should systematically document the role of neprilysin inhibition on sleep physiology.

Heart failure

OP-135

Evaluation of the role of autophagy and microtubules inhibition through blood beclin-1 levels in patients with heart failure with reduced ejection fraction

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Background and Aim: In this study, we aimed to compare the serum beclin-1 levels which is one of markers and moderators of autophagic activity in the serum of patients with heart failure with reduced ejection fraction (HFrEF), and those healthy subjects. Besides, we will be investigated serum beclin-1 levels according to etiological classifications (ischemic/non-ischemic subgroups). Also, the subgroup of patients using colchicine as a microtubule inhibitor was followed-up due to HFrEF included.
Methods: This study included between January 2018 and December 2019 in Istanbul University-Cerrahpaşa, Cardiology Institute, and 50 patients with HFrEF (25 with ischemic etiology, 25 with non-ischemic etiology) and 30 healthy subjects. Serum beclin-1 levels were determined by using the ELISA method by the ELISA Kit. Although serum beclin-1 levels of all HFrEF group compared to the control group.
Results: Although serum beclin-1 levels of all HFrEF group compared to the control group did not reach statistical significance, increased serum beclin-1 levels were found (p=0.64). However, NT-proBNP levels were found significantly higher (p=0.01). Serum beclin-1 levels correlated with ejection fraction in 50 patient with HFrEF (p=0.018, R^2=0.088). In the non-ischemic etiology subgroup with HFrEF especially had higher serum beclin-1 levels (p=0.01). There was also no significant correlation between creatinine and eGFR levels and autophagic activity (p=0.482). Also, we found lower levels of NT-proBNP that did not reach statistical significance and higher beclin-1 levels to reach statistical significance (p=0.015) in colchicine- using patient group.
Conclusions: Beclin-1 levels especially increased in the HFrEF with non-ischemic etiology group. Low dose colchicine regulates autophagy and vesicle trafficking in HFrEF.

Table 1. Parameters A, B, C

Parameters A,B,C			
Parameters of A Group	All Patients (N: 50)	Controls (N: 30)	P-value*
EF (%)	31.3±6.2	60	0.001
NT-proBNP (pg/dl)	2230.4±2079,7	53.6±21,5	0,01
Beclin-1 (ng/ml)	6.1±10.4	2.7±6.3	0.64
Parameters of B Group	İschemic HFrEF subgroup(N:25)	Non-ischemic HFrEF subgroup (N: 25)	P-value
Age	64.4±10,2	53.9±13.3	0.004
Gender (male %) Diabetes Mellitus (%)	23 (%92) 14 (%56)	20 (80%) 8 (32%)	0.2 0.08
Hypertension (%)	19 (76%)	10 (40%)	0.01
Hyperlipidemia (%)	19 (76%)	6 (24%)	0,0001
Cigaret (%)	18 (72%)	6 (24%)	0.001
Alcohol (%)	1 (4%)	2 (8%)	0.5
NT-proBNP (pg/dl	2647±2182	1813±1924	0.01
Beclin-1 (ng/ml)	2.07±4.7	12.7±16.1	0.01
Parameters of C Group	Colchicine + HFrEF subgroup (N: 13)	Colchicine – HFrEF subgroup (N:37)	P Value
EF (%)	27.5±3.2	32.6±6.4	0.009
LVD (mm)	59.7±8.9	69±7.6	0.002
LA (mm) RVD (mm)	46.8±9.4 25.0±1.8	46.0±8.8 25.5±3.3	0.59 0.54
TAPSE (mm)	18.4±3.0	19.8±2.7	0.33
NT-proBNP (pg/dl)	2029.9±2344	2300±2000	0,69
Beclin-1 (ng/ml)	12.44±10	3.4±8.4	0.015

*Table 3 Parameters A. Average of Echocardiographic EF values, NT-ProBNP and Beclin-1 Levels of Patients with Heart Failure (N: 50) and Control Group (N: 30) participating in the study (mean ± SD). Parameters B. Differences in NT-proBNP and Beclin-1 values of patients with heart failure according to ischemic (n=25) and non-ischemic (n=25) etiology included in the study (mean ± SD). Parameters C. Differences in NT-ProBNP and Beclin-1 Levels and echocardiographic parameters of colchicine used(N:13) and non-colchicine used patients in the HFrEF patient group (n=37) included in the study (mean ± SD). *Continuous variables are presented as mean ± SD and dichotomous variables as percentages. A two-tailed t-test was used for comparison of means, and x2-test for percentages.*

Heart failure

OP-137

The association of hyponatremia with atrial fibrillation in patients with heart failure

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Background and Aim: Coexistence of hyponatremia and atrial fibrillation (AF) increases morbidity and mortality in patients with heart failure (HF). However, it is not established whether hyponatremia is related to AF or not. Our study aim was to seek the possible association of hyponatremia with AF in patients with HF and reduced ejection fraction (HFrEF).
Methods: The study included 280 outpatients with HFrEF. Based on the sodium concentrations ≤135 mEq/L or higher, the patients were classified into hyponatremia (n=66) and normonatremia (n=214).
Results: AF was detected in 124 (44.3%) patients. AF rate was higher [n=39 (59.1%)] in patients with hyponatremia compared to those with hyponatremia [n=85 (39.7%)], (p=0.020). In logistic regression analysis, hyponatremia was established as the predictor of AF (odds ratio [OR]=3.416, 95% confidence interval [CI]= 1.243-4.016, p=0.001), as well as other well-known predictors of AF. AF was higher rate in outpatients with HFrEF and hyponatremia.
Conclusions: There is an association between hyponatremia and AF in patients with HFrEF.

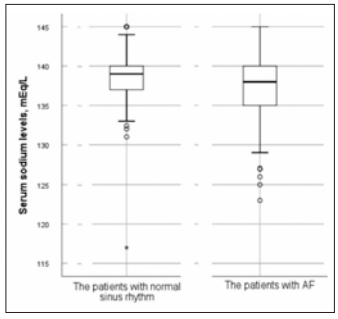

Figure 1. Shows sodium concentrations in patients with heart failure according to normal sinus rhythm and atrial fibrillation.

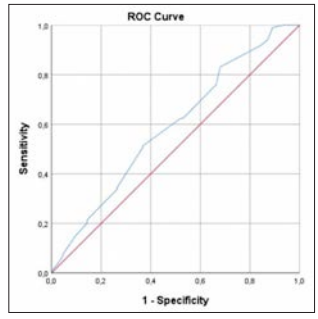

Figure 2. ROC curve shows hyponatremia' predictive value for AF.

Table 1. Clinical characteristics, laboratory and medications

Variables	All Patients n=280	Hyponatremia Group n=66	Normonatremia Group n=214	P
Age, years	67.6±10.5	67±11	68±10	0.820
Female, n (%)	78(27.8)	19(28.8)	59(27.6)	0.847
Hypertension, n (%)	185(66.1)	41(62.1)	144(67.3)	0.438
Coronary artery disease, n(%)	195(69.6)	44(66.7)	151(70.6)	0.548
Atrial fibrillation (AF) (%)	124(44.3)	39(59.1)	85(39.7)	0.020
NT-proBNP, pg/mL	6021(104-35000)	7300(104-35000)	8000(136-35000)	0.199
Osmolality (mOsm/kg)	291±9	283±9	294±7	<0.001

Heart failure

OP-138

Complementary and alternative medicine methods used by Turkish patients with heart failure and their effect on quality of life

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Background and Aim: The aim of this study is to investigate the attitudes of patients with heart failure who use complementary and alternative medicine towards complementary and alternative medicine use and the effects of these methods on their quality of life.
Methods: This cross-sectional and descriptive study was conducted with 648 heart failure patients who applied to the cardiology outpatient clinic of a public hospital in Istanbul. The data were collected using the Information Form, the Holistic Complementary and Alternative Medicine Questionnaire and the Minnesota Living with Heart Failure Questionnaire. Kruskal-Wallis-H Test, Mann Whitney U Test, Fisher Exact Test, Pearson Correlation Test and Pearson Chi-Square Test were used to analyze the data.
Results: 87% of the patients used complementary and alternative medicine, the most commonly used mind-body therapy was prayer-worship (55.6%), and the most commonly used phytotherapeutic was omega-3

(38%). The mean Minnesota Living with Heart Failure Questionnaire score was 75.5473±17.21 and the mean Holistic Complementary and Alternative Medicine Questionnaire score was 25.60±11.32. A statistically significant, high level, negative correlation was found between these scales. (r=-0.657, p<.01)
Conclusions: It was determined that complementary and alternative medicine methods were widely used in patients who applied to cardiology outpatient clinics and the quality of life of the participants was at a good level. It is essential for healthcare professionals to routinely ask patients about their use of complementary and alternative medicine, as cardiac drugs can interact negatively with herbal medicines due to the narrow therapeutic range.

Heart failure

OP-140

The relationship between plasma vitamin B12 and the severity of the disease in patients with advanced heart failure

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Background and Aim: The liver plays an important role in storing nutritional products such as vitamin B12 and converting them into active forms for the body. As a result of hepatocyte dysfunction due to hepatic congestion and hypoxia caused by HF, serum B12 vitamin levels increase. In this retrospective cohort study, it was aimed to compare serum B12 vitamin levels in advanced and non-advanced HF patient groups, to correlate serum B12 levels with the parameters of HF, the presence of right HF and 1-year hospitalization / mortality.
Methods: Records of 235 consecutive patients with HFrEF who applied to our cardiology clinic between January 2017 and March 2019 with a diagnosis of heart failure were analyzed retrospectively. Data regarding demographics, clinical signs, and therapeutic and conventional echocardiographic measurements were recorded as well as routine laboratory parameters including hematinic indices. Diagnostic decision-making features of serum B12 levels in predicting right HF and advanced HF were performed by Receiver Operating Characteristics (ROC) curve analysis. In addition, the correlation of serum B12 levels with clinical, biochemical and echocardiographic parameters was evaluated.
Results: The mean follow-up was 12 months. B12 level of the patients had positive correlation with serum NT-proBNP, AST, LDH, GGT, direct and indirect bilirubin levels, and negative correlation with TAPSE and albumin levels. To predict advanced HF, when ROC analysis was performed to find the most accurate B12 level, 370 pg/dL threshold level of B12 was found to have appropriate sensitivity (70.3%) and specificity (60%) (AUC: 686, %95 C-IN 0.613-0.758, p<0.001). Likewise, when ROC analysis was performed to find the most accurate B12 level in right HF prediction, 414 pg /dL threshold level of B12 was found to have appropriate sensitivity (72.5%) and specificity (67.8%) (AUC: 751, %95 C-IN 0.688-0.814, p<0.001). In the multivariate logistic regression analysis, where death was selected as the dependent variable and variables with p<0.1 were included in the univariate analysis, serum B12 vitamin levels were found to be an independent predictor of 1-year mortality.
Conclusions: This study shows that serum B12 levels increase in HFrEF patients due to increased congestion caused by progress of the disease stage and the right HF. Serum B12 vitamin levels can provide information regarding prognosis as an independent predictorof mortality in Heart failure with reduced ejection fraction.

Table 1. Comparing advanced and non-advanced HF patients

	Non-Advanced HF n= 70	Advanced HF n= 165	p
Age, years	52 (21-77)	52 (19-70)	0.606
Female, n (%)	14 (20%)	22 (13.3%)	0.194
Hypertension, n (%)	32 (45.7%)	78 (47.3%)	0.827
Diabetes, n (%)	21 (30%)	25 (33%)	0.617
Atrial fibrillation, n (%)	11 (16%)	42 (26%)	0.102
ICD/CRT, n (%)	49 (70%)	150 (90%)	<0.001
Creatinine, mg/dl	0.8 (0.4-2.4)	1.0 (0.49-3.78)	<0.001
GFR, ml/dk/1.73 m2	96 (22-145)	80 (12-192)	<0.001
Uric acid, mg/dl	6.4±2.0	7.2±4.4	0.007
Sodium, mEq/L	140 (110-147)	138 (123-147)	<0.001
AST, U/L	24 (5-551)	24 (9-2167)	0.031
GGT, U/L	27 (10-632)	57 (12-589)	<0.001
Direct biluribine, mg/dl	0.21 (0.09-2.27)	0.4 (0.07-15.7)	<0.001
NT-proBNP, ng/L	598 (33-25000)	3236 (82-35000)	<0.001
Beta-blokers, n (%)	68 (97,1%)	162 (98,2%)	0.636
ACEinh/ARB/ARNI	69 (98,6%)	159 (96,4%)	0.677
Digoxin, n (%)	9 (12,9%)	27 (16,4%)	0,495
MRA, n (%)	58 (82,9%)	141 (85,5%)	0.613
Furosemide, n (%)	36 (51,4%)	131 (79,4%)	<0.001
Ivabradine, n (%)	5 (7,1%)	13 (7,9%)	0.846
Hemoglobine, gr/dl	14,4 (9,8-15,1)	13,7 (8,3-17,7)	0,017
WBCx10^3/uL	7,7±1,8	8,8±3,0	0.008
vit B12	326 (170-2015)	542 (148-2000)	<0.001
Right HF, n (%)	15 (21,4%)	105 (63,6%)	<0.001
1-year mortality, n(%)	2 (2,9%)	21 (12,7%)	0.002
1-year hospitalization n, (%)	0 (0-4)	2 (0-5)	<0.001

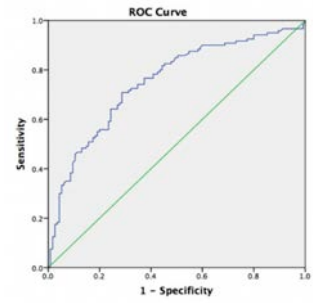

Figure 1. ROC curve of vitamin B12 predicting right heart failure.

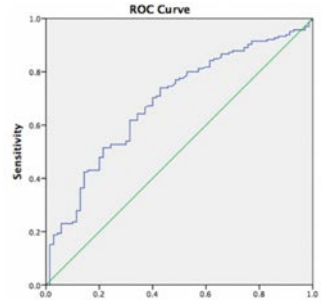

Figure 2. ROC curve of vitamin B12 predicting advanced heart failure.

Cardiac imaging / Echocardiography

OP-141

Evaluation of biventricular function in patients with COVID-19 using speckle tracking echocardiography

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Background and Aim: A new infectious outbreak sustained by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is now spreading all around the world. The aim of this study was to evaluate the prognostic value of left ventricular global longitudinal strain (LV-GLS) and right ventricular longitudinal strain (RV-LS) in patients with coronavirus disease 2019 (COVID-19).
Methods: In this prospective, single-center study, data were gathered from patients treated for COVID-19 between April 15 and April 30, 2020. Two-dimensional echocardiography (2-DE) and speckle tracking echocardiography (STE) images were obtained for all patients. Patients were divided into three groups: those with severe COVID-19 infection, those with non-severe COVID-19 infection, and those without COVID-19 infection (the control group). Data regarding clinical characteristics and laboratory findings were obtained from electronic medical records. The primary endpoint was in-hospital mortality.
Results: A total of 100 patients hospitalized for COVID-19 were included in this study. The mean age of the severe group (n=44) was 59.1±12.9, 40% of whom were male. The mean age of the non-severe group (n=56) was 53.7±15.1, 58% of whom were male. Of these patients, 22 died in the hospital. In patients in the severe group, LV-GLS and RV-LS were decreased compared to patients in the non-severe and control groups (LV-GLS: −14.5±1.8 vs. −16.7±1.3 vs. −19.4±1.6, respectively [p<0.001]; RV-LS: −17.2±2.3 vs. −20.5±3.2 vs. −27.3±3.1, respectively [p<0.001]). The presence of cardiac injury, D-dimer, arterial oxygen saturation (SaO2), LV-GLS (OR:1.63, 95% confidence interval [CI] 1.08–2.47; p=0.010) and RV-LS (OR:1.55, 95% CI 1.07–2.25; p=0.019) were identified as independent predictors of mortality via multivariate analysis.
Conclusions: LV-GLS and RV-LS are independent predictors of in-hospital mortality in patients with COVID-19.

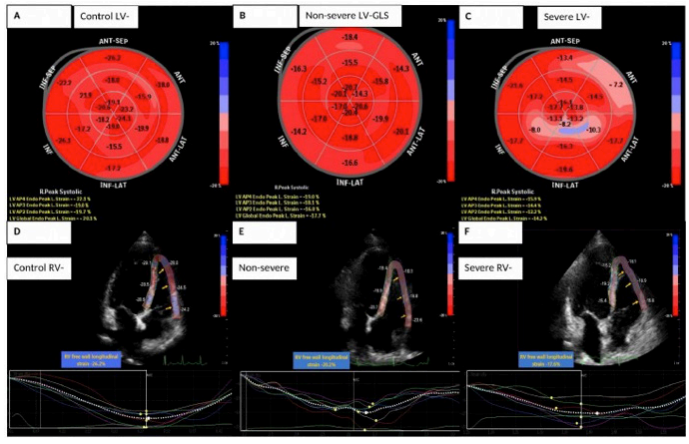

Figure 1. Bull's eye images of right ventricular longitudinal strain (RV-LS) values of control, non severe and severe patients.

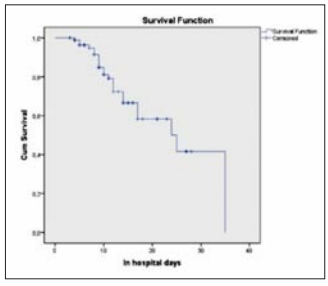

Figure 2. Kaplan-Meier survival curves for mortality during the time from admission.

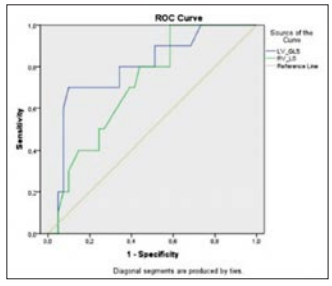

Figure 3. ROC curve analysis showing the specificity and sensitivity of the LV-GLS and RV-LS in predicting death.

Table 1. Demographic and clinical characteristics of patients severe and non-severe group

Variables	Total patients (n=145)	Control(n=45)	Non-severe (n = 56)	Severe (n = 44)	p
Clinical characteristics					
Age (years)	55.6±14.4	54.6±14.7	53.7 ± 15.1	59.1 ± 12.9	0.152
Male, n(%)	73(50%)	22(48%)	33(58%)	18(40%)	0.196
BMI (kg/m2)	23.4±3.4	23.7±3.4	23.0 ± 2.9	23.5 ±4.1	0.578
HR, beats/min	86.9±18.0	83.6±15.6	85.3±16.0	92.2±21.6	0.055
RR, times/min	23.3±4.1	21.8±2.3 *	21.3±2.3 *	27.5±4.4 **	<0.001
SAP, mmHg	123.0±15.8	125.6±13.6	121.1±14.5	122.7±19.3	0.363
DAP, mmHg	76.9±8.3	77.0±7.9	76.8±8.3	77.0±8.7	0.986
Smoker, n (%)	44(30%)	13(28%)	21(37%)	10(22%)	0.271
Pneumonia on CT , n(%)	75(51%)	-	31(55%)	44(100%)	<0.001
Chronic medical illness					
HT, n(%)	43(29%)	11(24%)	15(26%)	17(38%)	0.285
DM, n(%)	25(17%)	7(15%)	8(14%)	10(22%)	0.506
HLD, n(%)	20(13%)	8(17%)	5(8%)	7(15%)	0.390
Laboratory findings					
Haemoglobin(g/dl)	12.5±2.0	12.9±1.6 *	13.2 ± 1.2*	11.1 ± 2.6 **	<0.001
WBC (10^3 /µl)	6.6(5.1-11.4)	6.1(5.1-10.8)	6.1(5.1-10.8)	9.1(5.2-13.7)	0.251
Creatinine (mg/dl)	0.8(0.7-1.0)	0.8(0.7-1.0)	0.8(0.7-1.0)	0.8(0.7-1.2)	0.968
Sodium (mmol/L)	136.6±3.3	136.9±3.1	136.8 ± 2.8	135.9 ± 4.0	0.254
Potassium (mmol/L)	4.1±0.5	4.0±0.6	4.1 ± 0.5	4.0 ± 0.5	0.456
Glucose (mg/dL)	122.7±30.4	114.4 ±18.6*	116.4±28.9*	139.3±35.7**	<0.001
CRP (mg/dL)	43(15-81)	-	29(9-61)	81(46-132)	<0.001
hs-TnI (NR<14pg/ml)	10(10-17)	-	10(10-11)	16(10-59)	<0.001
D-dimer (ng/mL)	690(327-1020)	-	420(192-690)	1140(707-1700)	<0.001
CK-MB (ng/mL)	5.0(1.1-11.0)	-	5.2(1.1-9.1)	5.0(1.2-17.0)	0.756
SaO2	93.3±4.7	96.3±1.3*	95.7±2.3*	87.3±3.6**	<0.001
Treatments					
Antiviral therapy, n (%)	84(58%)	-	48(85%)	36(81%)	0.598
Antibiotic therapy, n (%)	94(65%)	-	52(92%)	42(95%)	0.587
Oxygen therapy, n (%)	20(14%)	-	20(35%)	0(0%)	<0.001
High-flow oxygen, n (%)	26(18%)	-	0(0%)	26(59%)	<0.001
NIMV, n (%)	20(13%)	-	0(0%)	20(45%)	<0.001
ICU admission, n (%)	33(22%)	-	0(0%)	33(75%)	<0.001
Complications					
Acute heart injury, n (%)	36(24%)	-	5(9%)	31(70%)	<0.001
Acute kidney injury, n (%)	31(21%)	-	3(5%)	28(63%)	<0.001
Prognosis					
Hospital stay (days)	9(5-13)	-	6(4-10)	13(9-20)	<0.001
Discharge, n (%)	60(41%)	-	46(82%)	14(31%)	<0.001
Death, n(%)	22(15%)	-	0(0%)	22(50%)	<0.001

*P<0.05 Between control group and non-severe group,, *P<0.05 between control group and severe group, *P<0.05 between non-severe group and severe group

Abbreviations: BMI, body mass index; HR, heart rate; RR, respiratory rate; SAP, systolic arterial pressure; DAP, diastolic arterial pressure; HT, hypertension; DM, diabetes mellitus; HLD, hyperlipidemia; WBC, white blood cell, CRP, C-reactive protein; hs-TnI, high sensitive-Troponin I; NR, normal range; CK, creatinine kinase; SaO2, arterial oxygen saturation; NIMV, non invasive mechanical ventilation; ICU, intensive care unit.

Table 2. Comparison of conventional echocardiographic and two-dimensional speckle tracking global longitudinal strain parameters of patients

Variables	Total patients (n=145)	Control(n=45)	Non-severe (n = 56)	Severe (n = 44)	p
Left heart findings					
LVEF (%)	60.3±4.6	60.8±3.7	59.9 ± 4.9	58.1 ± 4.6	0.117
LV-GLS (%)	-16.8±2.5	-19.4±1.6* *	-16.7±1.3* *	-14.5±1.8**	<0.001
SV (ml)	66.7±18.6	68.8±11.8	65.6±13.4	65.9±28	0.660
CO(L/min)	5.9±2.1	6.4±1.7	5.6±1.6	5.7±2.7	0.101
LVEDD (mm)	46.2±4.4	46.6±4.4	46.2±4.1	45.8±4.9	0.669
LVESD (mm)	30.1±3.9	29.9±3.4	30 ± 3.5	30.6 ±4.9	0.684
LV mass (g)	166.9± 19.8	165.7±19.1	167.1±18.8	168.1 ±21.7	0.531
WMSI	1±0.2	1±0.2	1±0.1	1±0.2	0.614
LA (mm)	30.0±4.6	33.3±4.4*	34.5±3.3*	37.3±5.4**	<0.001
E/A ratio	1±0.3	1±0.4	1±0.3	0.9±0.3	0.551
E/e' ratio	9.2±2.5	8.7±2.7	9.1±2.1	9.9±2.9	0.114
Right heart findings					
RV-FAC (%)	45.2±5.3	46.4±5.4	45.1±4.8	44.1±5.6	0.127
RV-LS (%)	-21.6±5	-27.3±3.1* *	-20.5±3.2* *	-17.2±2.3**	<0.001
TAPSE (mm)	21.8±3.3	22.4±3.3	22.1±3.3	21±3.3	0.146
sPAP, mmHg	31±8.3	28.6±5.3 *	28.7±6.3 *	36.5±10.4 **	<0.001
RV (mm)	31.8±4.5	29.9±2.8 *	31.7±4.2	33.7±5.6 *	<0.001
RA (mm)	32.9±4.4	31.7±2.9	33.2±4.5	33.9 ± 5.3	0.063
TDI S', cm/s	15.2±3.1	15.5±3.2	15±3.1	15.1±3	0.721
PA, mm	21.4±2.6	20.9±2.8	21.3±2.6	22.1±2.1	0.080

*P<0.05 Between control group and non-severe group,, *P<0.05 between control group and severe group, *P<0.05 between non-severe group and severe group

Abbreviations: LVEF, left ventricular ejection fraction; LV-GLS, left ventricular global longitudinal strain; SV, stroke volume; CO, cardiac output; LVEDD, left ventricular end diastolic diameter; LVESV, left ventricular end systolic diameter; WMSI, wall motion score index; LA, left atrial; RV-FAC, right ventricular fractional area change; RV-LS, right ventricular longitudinal strain ; TAPSE, tricuspid annular plane systolic excursion; sPAP, systolic pulmonary artery pressure; RV, right ventricular; RA, right atrial; TDI S', tissue Doppler imaging systolic wave S' velocity; PA, pulmonary artery.

Table 3. Correlation of strain findings with prognostic laboratory parameters

	Spearman	RV-LS	Age	hs-TnI	D-dimer	CRP	Hgb	sPAP	SaO2	RR	HR
LV-GLS	r	0.794	0.065	0.633	0.577	0.175	0.062	0.355	-0.549	0.396	0.206
	p	<0.001	0.437	<0.001	<0.001	0.168	0.460	<0.001	<0.001	<0.001	0.013
RV-LS	r		0.108	0.608	0.620	0.158	0.111	0.385	-0.608	0.492	0.123
	p		0.197	<0.001	<0.001	0.351	0.184	<0.001	<0.001	<0.001	0.141

Abbreviations: LV-GLS, left ventricular global longitudinal strain; RV-LS, right ventricular longitudinal strain; hs-TnI, high-sensitive troponin I; CRP, C-reactive protein; Hgb, haemoglobin; sPAP, systolic pulmonary artery pressure; SaO2, arterial oxygen saturation; RR, respiratory rate; HR, heart rate.

Table 4. Multivariate Logistic Regression analysis on the risk factors associated with mortality in patients with COVID-19

Variable	OR	95% CI	P	Variable	OR	95% CI	P
Age	0.984	0.930-1.042	0.588	Age	0.986	0.928-1.046	0.637
Gender	2.942	0.723-11.970	0.132	Gender	3.049	0.721-12.899	0.130
Cardiac injury	5.125	1.206-21.783	**0.027**	Cardiac injury	1.417	1.125-1.709	**0.031**
D-dimer	1.001	0.999-1.003	0.792	D-dimer	4.250	1.312-21.418	**0.021**
SaO2	0.842	0.724-0.979	**0.025**	SaO2	0.830	0.717-0.961	**0.012**
*LV-GLS	1.635	1.080-2.474	**0.010**	*RV-GLS	1.557	1.075-2.256	**0.019**
*LV-GLS > -15.20%	8.342	2.779-79.351	**<0.001**	*RV-GLS > -18.45%	6.229	1.512-25.670	**0.011**

*LV-GLS and RV-GLS were analyzed in logistic regression separately as linear and categorical variables

Abbreviations: SaO2, arterial oxygen saturation; LV-GLS, left ventricular global longitudinal strain; RV-LS, right ventricular longitudinal strain.

Cardiac imaging / Echocardiography

OP-142

The simple right ventricle contraction pressure Index-A novel method for the echocardiographic assessment of right ventricle dysfunction in acute pulmonary embolism

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Background and Aim: Acute pulmonary embolism (APE) is a disease that has shown an increasing trend in recent years. The simple right ventricular contraction pressure index (sRVCPI) is a new parameter for estimating the right ventricular stroke work index (RVSWI) with echocardiographic parameters. In this research, we intend to represent the association between the sRVCPI, the pulmonary embolism severity index (PESI) and mortality in APE.

Methods: In the presented study, a sum of 116 patients who were diagnosed with APE using pulmonary computed tomography angiography or ventilation/perfusion scintigraphy were involved. Patients were divided into 2 groups based on the simplified PESI (sPESI): sPESI <1 (n=64) and sPESI ≥1 (n=52). Echocardiographic parameters, including the sRVCPI, were measured.

Results: There was a positive correlation with the mortality rate and the sRVCPI; the mortality was higher in patients with a higher sRVCPI (<0.001). In receiver operating characteristic (ROC) curve analysis using a cut-off level of 312.8 mmHg·mm, the sRVCPI prognosticated mortality with a sensitivity of 86.8% and specificity of 69.5% (ROC area under curve: 0.712; 95% CI: 0.597-0.882; p<0.001). In the sPESI ≥1 group, the sRVCPI was lower than the sPESI<1 group (364.3±31.9 vs 511.6±26.1; p<0.001). Between the sRVCPI and sPESI score, there was an inverse correlation (-0.784; p<0.001).

Conclusions: The sRVCPI is well-correlated with the sPESI score and is linked with mortality in patients with APE. This easily-measurable parameter may be applied to predict short-term mortality in APE patients.

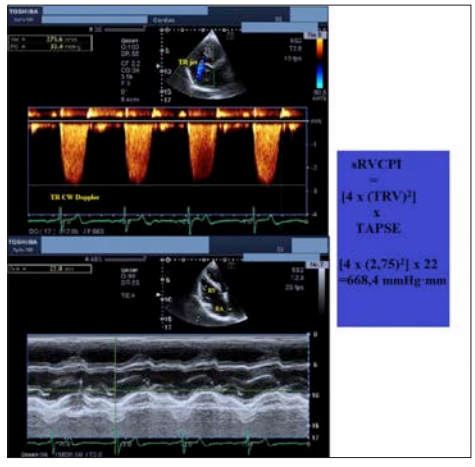

Figure 1. Calculation of the simple Right Ventricle Contraction Pressure Index. CW; continious wave, RA; right atrium, RV; right ventricle, sRVCPI; the simple Right Ventricle Contraction Pressure Index, TAPSE; tricuspid annular plane systolic excursion, TR; tricuspid regurgitation, TRV; tricuspid regurgitation velocity.

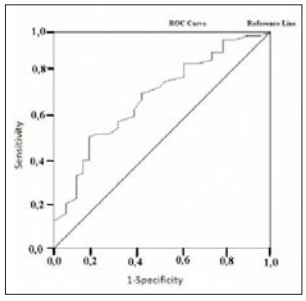

Figure 2. Receiver operating characteristic curve analysis.

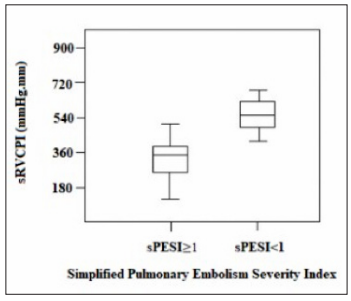

Figure 3. Box plot graph showing that the RVCPI was lower in the sPESI ≥1 group than in the patients with sPESI <1.

Cardiac imaging / Echocardiography

OP-143

CMR-derived infarct characteristics and outcome of patients with transient ST-segment elevation myocardial infarction compared to ST-segment and non-ST-segment elevation myocardial infarctions

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Background and Aim: Up to one out of four patients with signs of ST-segment elevation myocardial infarction (STEMI) express complete normalization of ST elevation before primary revascularization procedure. This condition is commonly referred to as 'transient ST-segment elevation myocardial infarction' (TSTEMI) and recent data suggests that this group of patients may have favorable outcome compared to STEMI patients. However, it is currently unknown how these patients compare to both STEMI and non-ST-segment elevation myocardial infarction (NSTEMI) patients with respect to infarct size characteristics and outcome. This study aims to explore cardiovascular magnetic resonance (CMR) derived scar tissue and 1-year outcome in patients with TSTEMI by comparison to STEMI and NSTEMI.

Methods: Patients with STEMI were enrolled from two prospective studies (n=170); the patients with TSTEMI were recruited from the TRANSIENT trial (n=141); the patients with NSTEMI were prospectively and consecutively enrolled at Amsterdam UMC (n=57) and Maastricht UMC (n=51). All patients underwent CMR examination 2-8 days after the index event. Cine imaging was done for volume and function assessment. Late gadolinium enhancement imaging was performed to identify infarct size (in grams) and the presence of microvascular obstruction (MVO). All CMR images were processed in a single core laboratory (Amsterdam UMC). Finally, clinical outcome data after 1 year were collected.

Results: The TSTEMI group demonstrated the lowest end-systolic left ventricular volume and highest left ventricular ejection fraction across the groups (overall p<0.001). Although there was a remarkably lower infarct size in TSTEMI patients compared to STEMI (1.41g [0.00–3.91] vs 13.48g [5.31–26.81], p<0.001), there was only a trend towards lower infarct size compared to NSTEMI patients (1.41g [0.00–3.91] vs 2.13g [0.00–8.64], p=0.06). Whilst MVO was observed less frequently in TSTEMI compared to STEMI patients (5 (4%) vs 53 (31%), p<0.001), no significant difference was seen between TSTEMI and NSTEMI patients (5 (4%) vs 5 (5%), p=0.72). Multivariable linear regression analysis identified infarct type, smoking, peak troponin-T and pre-PCI TIMI flow as predictors for infarct size (p=0.03, p=0.03, p<0.001 and p<0.001, respectively). One-year mortality rate was low and comparable in all 3 MI types (TSTEMI 3 (2.2%), NSTEMI 3 (3.1%), STEMI 4 (2.4%), log-rank test p=0.91).

Conclusions: In comparison to NSTEMI and STEMI, TSTEMI yielded favorable cardiac left ventricular function and scar mass. However, this did not lead to benefit in short term (1-year) outcome; further studies are needed with longer follow-up.

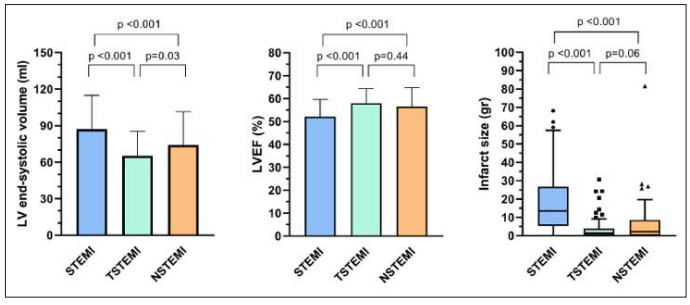

Figure 1. Kardiyovasküler MR bulguları.

Cardiac imaging / Echocardiography

OP-144

The effects of endobronchial coil therapy on right ventricular functions

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Background and Aim: Chronic obstructive pulmonary disease (COPD) is a major global health issue that causes significant morbidity and mortality. Persistent airflow reduction is the hallmark of the disease, and frequently accompanied by hyperinflation in advanced cases.Hyperinflation is related to dyspnea, a decrease in quality of life, frequent exacerbations and even mortality.Lung volume reduction surgery (LVRS) can be used in select patients who are severely limited in their daily activities to improve their lung functions and quality life. However, this surgery is associated with significant morbidity and mortality. Recently, bronchoscopic techniques were developed as an alternative to LVRS to achieve results with lesser complications. Consequently, a number of studies have shown that endobronchial coil therapy (EBCT) that reduce end-expiratory lung volume may be a potential therapeutic option in advanced cases of emphysema and hyperinflation (Figure 1). However, there is paucity of data regarding the effects of these therapies on the heart functions. The aim of this study is to evaluate the right ventricular functions before and after the procedure in patients who underwent EBCT.

Methods: Patients who were between 18 and 80 years of age and scheduled for EBCT with GOLD 3-4 were enrolled in the study. Comprehensive echocardiographic evaluation focused on right ventricular functions before and after a month of the procedure was planned for patients scheduled for EBCT. Right heart functions were evaluated using MPI, TAS, TAPSE. Right atrium area and maximum velocity of tricuspid regurgitation were also noted.

Results: A total of 23 patients were enrolled in the study (Figure 2). 21 patients underwent bilateral intervention, while only 2 patients received unilateral treatment. There were statistically significant differences in tricuspid lateral annular systolic velocity (TAS) and myocardial performance index (MPI) values were better than the pre-operative values (TAS, 11.6 (9–15) vs 13.2 (9.80–17.0), p=0.001; MPI, 0.49±0.15 vs 0.39±0.11, p<0.001 pre and post-operative, respectively). Peak tricuspid regurgitant jet velocity (2.52±0.6, 2.38±0.6, p=0.02) and systolic pulmonary artery pressure values were lower in the post-operative period (41.15±5.94 vs 36.83±8.01 p=0.019).Trans-tricuspid E velocity was found to be significantly higher and right atrium (RA) area was smaller in the postoperative period (E, 43±10.88 vs 46.61±9.95, p=0.013; RA Area, 17.3±3.73 vs 15.26±3.4, p<0.001; pre and post-operative, respectively) (Figure 3).

Conclusions: Endobronchial therapies are increasingly being used to reduce end-expiratory lung volume in advanced cases of emphysema and hyperinflation. Considering close relationship between heart and lung, these therapies potentially affect the heart. In this study, we showed improvements in echocardiographic parameters of RV function and PA pressure. The effects of these therapies on heart should be evaluated in larger future studies.

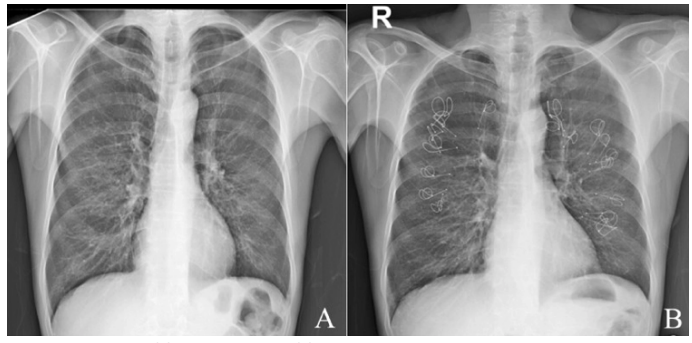

Figure 1. Preoperative **(a)** and postoperative **(b)** chest X-Ray images for a patient who underwent bilateral EBCT procedure.

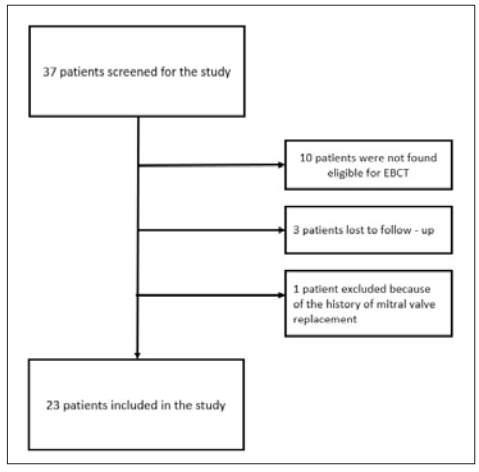

Figure 2. Flow chart of the patient enrolment in the study.

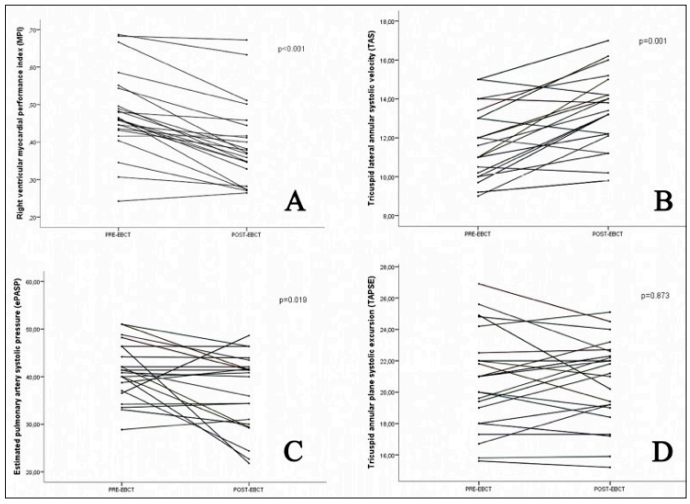

Figure 3. Echocardiographic parameters before and after the procedure in paired samples. **(a)** Right ventricle myocardial performance index, **(b)** Doppler–derived tricuspid lateral annular systolic velocity, **(c)** Estimated pulmonary artery systolic pressure, **(d)** Tricuspid annular plane systolic excursion.

Cardiac imaging / Echocardiography

OP-145

Evaluation of myocardial functions with strain and strain rates echocardiography measurements in atrial septal defect patients with percutaneous closure

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Background and Aim: In this study we aimed to evaluate myocardial functions of patients with atrial septal defect (ASD) by strain and strain rate echocardiography. Because, evaluation of myocardial functions is very important for management of responsive to treatment in congenital heart disease. However, due to complex structure and physiology it is difficult to evaluate right ventricle (RV). Recently, strain and strain rate echocardiography have been introduced as an encouraging method to evaluate myocardial functions.
Methods: 31 patients with ASD and 31 control subjects with similar chracteristics were included in the study. All patients' defect were closed with 'ASD Occluder' device. In control patients, strain and strain rate echocardiography measurements were conducted additional to conventional echocardiographic parameters. Patients with ASD data were divided into three groups; pre-process, post-process first and sixth month. The data obtained were compared with the control group and also within themselves.
Results: RV dimensions and pulmonary arterial pressure were significantly higher in patients with ASD compared to the control group (4.4±0.8 cm vs. 3.3±0.5 cm, p<0.001), after ASD closure, they significantly decreased (4.4±0.8 cm vs. 3.6±0.5 cm, p<0.001). Left ventricle (LV) end-diastolic diameter (4.1±0.3 cm vs. 4.6±0.6 cm, p<0.001) and LV end-systolic diameter (2.5±0.2 cm vs. 2.9±0.3 cm, p<0.001) significantly increased after the procedure. Tricuspid annular plane sistolic excursion (TAPSE) significantly decreased in the 6th postoperative month (27±8 mm vs. 22±4 mm, p<0.001). The baseline and post closure characteristics by conventional echocardiographic measurements were given in Table 1-2. LV apical, mid, basal strain and strain rates; septal apical, mid, basal, strain and strain rates; left atrium strain and strain rate in terms of; There were no significant changes in control group and ASD group (before and after ASD closure). RV and right atrial (RA) strain and strain rate echocardiographic findings of the patients before and after closure of ASD were compared with the control group (Table 3). There were statistically significant changes in RV mid, basal and RA strain and strain rates and strain values approached normal values after closure of ASD (Table 4).
Conclusions: In ASD patients, RV dysfunction occurs over time due to volume overload. In our study, we demonstrated that RV functions were impaired in patients with ASD and improvement in RV functions after percutaneous ASD closure was demonstrated by echocardiography which is a new echocardiographic method. It was concluded that strain and strain rate echocardiography could be used for response to treatment and follow up for ASD patients. We believe that new studies including higher number of patients with moderate and severe pulmonary hypertension will further emphasize the importance of strain and strain rate echocardiography to assess RV functions.

Table 1. Comparison of conventional echocardiography parameters between control and ASD groups

	Control Group (n=31)	ASD Group (n=31) Baseline	ASD Group (n=31) 6.Mounths	p Values Control/ Basline	p Values Control/ 6.Months
End Diastolic Diameter Of Left Ventricle (cm)	4,5±0,4	4,1±0,3	4,6±0,6	0,008	0,123
End Sistolic Diameter of Left Ventricle (cm)	2,8±0,2	2,5±0,2	3,0±0,3	0,012	0,065
Left Ventricular Ejection Fraction (%)	64±8	68±10	62±6	<0,001	0,075
End Diastolic Diameter of Right Ventricle (cm)	3,3±0,5	4,4±0,8	3,6±0,5	<0,001	0,068
Diastolic Diameter of Right Ventricle / Diastolic Diameter of Left Ventricle	0,80±0,03	1,1±0,1	0,80±0,04	<0,001	0,489
Pulmonary Arterial Pressure (mm/Hg)	18±4	29± 9	18±5	<0,001	0,639
Tricuspid Annular Plane Sistolic Excursion (TAPSE) (mm)	22±5	27±8	22±4	<0,001	0,854
Left Ventricular Myocardial Performance Index (Tei Index)	0,38±0,08	0,35±0,04	0,37±0,06	0,003	0,080
Right Ventricular Myocardial Performance Index (Tei Index)	0,30±0,04	0,38±0,06	0,32±0,05	<0,001	0,092
The Pulmonary To Systemic Flow Shunt Ratio (Qp/Qs)	1,0±0,1	2,1±0,9	1,10±0,02	<0,001	0,128

Table 2. Comparison of conventional echocardiography parameters in ASD group

	ASD group (n:31) Baseline	ASD group (n:31) 1.month	ASD group (n:31) 6.month	p Values Baseline/ 1. month	p Values Baseline/ 6.month
End Diastolic Diameter of Left Ventricle (cm)	4,1±0,3	4,4±0,5	4,6±0,6	<0,001	<0,001
End Sistolic Diameter of Left Ventricle (cm)	2,5±0,2	2,9±0,3	3,0±0,3	<0,001	<0,001
Left Ventricular Ejection Fraction (%)	68±10	63±8	62±6	<0,001	<0,001
End Diastolic Diameter of Right Ventricle (cm)	4,4±0,8	4,0±0,6	4,0±0,6	<0,001	<0,001
Diastolic Diameter of Right Ventricle / Diastolic Diameter of Left Ventricle	1,1±0,1	0,90±0,09	0,80±0,04	<0,001	<0,001
Pulmonary Arterial Pressure (mm/Hg)	29±9	22±7	18±5	<0,001	<0,001
Tricuspid Annular Plane Sistolic Excursion (TAPSE) (mm)	27±8	23±6	22±4	<0,001	<0,001
Left Ventricular Myocardial Performance Index (Tei Index)	0,35±0,04	0,36±0,05	0,37±0,06	<0,001	<0,001
Right Ventricular Myocardial Performance Index (Tei İndex)	0,38±0,06	0,33±0,05	0,33±0,05	<0,001	<0,001
The Pulmonary To Systemic Flow Shunt Ratio (Qp/Qs)	2,1±0,9	1,40±0,18	1,10±0,02	<0,001	<0,001

Table 3. Comparison of Strain Echocardiography Parameters and strain rates between control and ASD Groups

	Control Group (n:31)	ASD Group (n:31) baseline	ASD Group (n:31) 6.month	p Values Control/ Baseline	p Values Control/ 6.month
Right Ventricle Apical Strain (%)	-25,2±1,6	-25,5±0,9	-25,4±0,8	0,312	0,507
Right Ventricle Mid Strain (%)	-27,2±1,6	-32,8±1,1	-30,1±2,5	<0,001	<0,001
Rigt Ventricle Basal Strain (%)	-29,4±1,3	-34,7±1,2	-31,4±2,7	<0,001	<0,001
Right Atrial Strain (%)	37,4±1,6	43,6±0,9	40,0±2,5	<0,001	<0,001
Right Ventricle Apical Strain Rate (1/s)	-2,01±0,19	-2,01±0,26	-1,91±0,22	0,312	0,507
Right Ventricle Mid Strain Rate (1/s)	-2,10±0,36	-2,90±0,38	-2,52±0,38	<0,001	<0,001
Right ventricle basal strain rate (1/s)	-2,20±0,28	-3,09±0,23	-2,63±0,42	<0,001	0,002
Right atrial strain rate (1/s)	2,60±0,47	3,47±0,24	3,00±0,47	<0,001	<0,001

Table 4. Comparison of strain echocardiography parameters and strain rates in ASD group

	ASD Group (n:31) Baseline	ASD Group (n:31) 1.mounth	ASD Group (n:31) 6.month	p Values Baseline/ 1.month	p Values Baseline/ 6.month
Right Ventricle Apical Strain (%)	-25,5±0,9	-25,5±0,9	-25,4±0,8	0,579	0,354
Right Ventricle Mid Strain (%)	-32,8±0,1	-30,6±2,1	-30,1±2,5	<0,001	<0,001
Rigt Ventricle Basal Strain (%)	-34,7±1,2	-31,6±2,1	-31,4±2,7	<0,001	<0,001
Right Atrial Strain (%)	43,6±0,9	40,8±2,0	40,0±2,5	<0,001	<0,001
Right Ventricle Apical Strain Rate (1/s)	-2,01±0,26	-1,99±0,20	-1,91±0,22	0,312	0,507
Right Ventricle Mid Strain Rate (1/s)	-2,90±0,38	-2,61±0,29	-2,10±0,38	<0,001	<0,001
Right Ventricle Basal Strain Rate (1/s)	-2,90±0,38	-2,62±0,36	-2,63±0,42	<0,001	<0,001
Right Atrial Strain Rate (1/s)	3,47±0,24	3,15±0,35	3,00±0,47	<0,001	<0,001

Cardiac imaging / Echocardiography

OP-146

Evaluation of changes in the hearth of pregnant woman by speckle tracking echocardiography

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Background and Aim: The aim of our study is to evaluate functional and structural changes in left and right ventricles in healthy pregnant women by transthoracic echocardiography. Left ventricular systolic, diastolic and right ventricular diastolic function were measured. Left ventricular global longitudinal, global radial, global circumferential strain and right ventricular global longitudinal strain were evaluated by speckle tracking echocardiography.

Methods: The study was started with 50 healthy pregnant women. Five pregnant women were excluded from follow-up and the study was completed with 45 healthy pregnant women. Patients were evalueted in the first, second, third trimesters and postpartum 10th week of pregnancy. Basic demographic and hemodynamic data were obtained and two-dimensional echocardiography (TTE) was performed. Left ventricular systolic and diastolic parameters with left ventricular global longitudinal strain (LV-GLS), left ventricular global radial strain (LV-GRS), left ventricular global circumferential strain (LV-GCS) and right ventricular global longitudinal strain (RV- GLS) have been evaluated. Fig 1. LV GLS measerument was demonstrated in Fig 1.

Results: The mean age of pregnant women at the beginning of the study is 27.4. There was no any significant variation in blood pressure during pregnancy (p=0.67). Clinical and hemodynamic features of pregnant women shown in Table 1. There was no significant difference in left ventricular EF, E / E', TAPSE values during pregnancy and postpartum period (p=0.33, p=0.90 and p=0.87, respectively). There was a significant increase in left ventricular wall thickness during the pregnancy (IVS; p=<0.01, LVPW; p=<0.01). There was an increase in left ventricular systolic and diastolic diameters and endsystolic and endiastolic volumes during pregnancy (p=0.03, p=<0.01, p=0.02, p=<0.01, respectively). The morphological and functional changes in the left and right ventricles were demonstrated in Table 2. While LV-GLS and LV-GCS decreased significantly throughout pregnancy, they increased towards baseline values in postpartum period (LV-GLS for the first trimester - 22.3±2.8%, for the second trimester - 21.4±2.1%, for the third trimester - 19.3%±2.1, postpartum - 21.9±1.8%, p<0.01, LV-GCS for the first trimester - 20.3±1.8%, for the second trimester - 19.1±2.2%, for the third trimester - 18.1±2.6%, postpartum - 20.1±2.1%, p<0.01). There was no significant difference in LV-GRS and RV-GLS values during and after pregnancy (p=0.23, p=0.18, respectively). Left and right ventricle strain evaluation during pregnancy shown in Table 3.

Conclusions: In this study, it was determined that there was an increase in left ventricular volumes and wall thicknesses during pregnancy, and there was no significant difference in left ventricular EF and TAPSE. LV-GLS and LV-GCS, the indicators of left ventricular function, were significantly decreased. There was no significant difference in LV-GRS and RV-GLS.

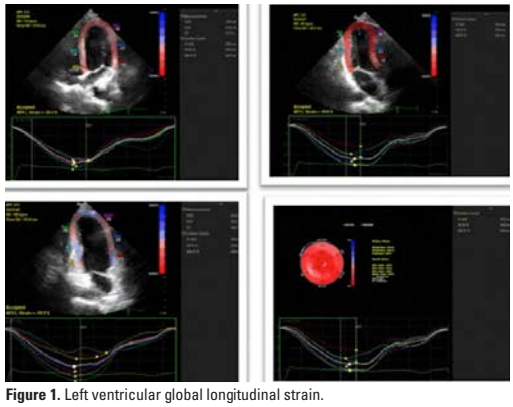

Figure 1. Left ventricular global longitudinal strain.

Table 1. Clinical and hemodynamic features of pregnant women SBP systolic blood pressure, DBP diastolic blood pressure, MBP mean blood pressure, *p<0.05 vs. Trimester 1; Ω p<0.05 vs. Trimester 3, ¥ p<0.05 vs. Postpartum

	Trimester 1 (13 ± 2)wk	Trimester 2 wk (24 ± 2)wk	Trimester 3 (36 ± 2)wk	Postpartum (10 ± 2.4)wk
n=	45	45	45	45
Weight (kg)	63.3 ± 8.5	67.6 ±9.6 *Ω¥	72.7 ± 7.5 *	64.5± 12.5 Ω
Heart rate (bpm)	79.6 ±10.4	82.1 ± 15.2	92.1 ± 9.0 *	82.3 ± 12.2 Ω
SBP (mmHg)	107.1 ±6.7	105.9 ± 10.5	108.1 ± 8.3	107.2 ± 9.4
DBP (mmHg)	65.3 ± 7.2	64.8 ± 7.7	66.2 ± 6.9	66.6 ± 6.0
MBP (mmHg)	79,2.5 ± 6.9	78.5 ± 8.3	80.1 ± 7.5	80.1 ± 7.8

Table 2. The morphological and functional changes in the left and right ventricle in pregnant women LVEDd indicates left ventricular end-diastolic dimension, LVEDs left ventricular end-systolic dimension, IVS interventricular wall thickness, PWD posterior wall LVEDV indicates left ventricular end-diastolic volume, LVESV left ventricular end-systolic volume, EF ejection fraction, E peak early diastole transmitral wave velocity, DT deceleration time *p<0.05 vs. Trimester 1; β p<0.05 vs. Trimester 2; Ω p<0.05 vs. Trimester 3, ¥p<0.05 vs. Postpartum

	Trimester 1 (13 ± 2) wk	Trimester 2 wk (24 ± 2)wk	Trimester 3 (36 ± 2)wk	Postpartum (10 ± 2.4)wk VV
n=	45	45	45	45
IVS (cm)	64.2 ± 8.8	69.7±9.6	73.6 ± 5.0* β	65.9 ± 9.6* β
PWD (cm)	78.6 ±14.4	83.6 ± 16.2	88.4 ± 10.2* β	82.3 ± 10.6* β
LVEDd (mm)	45.8 ± 2.0	47.8 ± 3.1	48.89 ± 2.6*	45.3 ± 2.4
LVEDs (mm)	27.4 ± 2.2	28.6 ± 2.6	30.5 ± 2.9*	27.9 ± 2.1
LVEDV(ml)	76.0 ± 7.8	78.5 ± 7.2	86.5 ± 7.5 *	84.2 ± 6.1
LVESV(ml)	33.3 ±6.5	35.8 ± 5.6	40.7 ±8.3*	33.4± 9.1
EF (%)	65.9 ± 4.4	65.4 ±5.4	66.1 ± 4.3	6 6.8± 3.8
E/A	1.3 ± 0.4	1.2 ± 0.3	1.01 ± 0.2 * β	1.21 ± 0.3*Ω
E/E'	5.7 ± 0.9	5.8 ± 1.1	5.8 ± 1.3	5.7 ± 1.8
DT	160± 5	212 ± 4	255 ± 3 * β	222 ± 7*Ω
TAPSE (cm)	23.4± 2.2	23.1± 2.6	23.7± 1.8	23.9 ± 3.1

Table 2. Left and right ventricle strain evolution during pregnancy LV- GLS: Left ventricular global longitudinal strain; LV-CS: Left ventricular circumferantial strain, LV- RS: Left ventricular radial strain, RV- GLS: Right ventricular global longitudinal strain; *p<0.05 vs. Trimester 1; β p<0.05 vs. Trimester 2; Ω p<0.05 vs. Trimester 3, ¥p<0.05 vs. Postpartum

	Trimester 1 (13 ± 2) wk	Trimester 2 wk (24 ± 2)wk	Trimester 3 (36 ± 2)wk	Postpartum (10 ± 2.4)wk
n=	45	45	45	45
LV-GLS (%)	-22.3 ± 2.8	- 21.4 ± 2.1	-19.3% ± 2.1*β	-21.9 ± 1.8*
LV-GCS (%)	- 20.3 ± 1.8	- 19.1 ± 2.2	- 18.1 ± 2.6*β	- 20.1 ± 2. Ω
LV-GRS (%)	45.4 ± 12.4	44.7 ± 14.2	42.5 ± 11.2	46.2 ± 10.2
RV-GLS (%)	-21.2± 2.4	-20.9± 2.9	-20.1± 2.5	-20.9 ± 2.7

Cardiac imaging / Echocardiography

OP-147

Clinical impact of coronary slow flow on the heart: A detailed CMR study

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Background and Aim: Although coronary slow flow (CSF) is seen in 2% of patients undergoing coronary angiography, its clinical significance and impact on ventricular function remains controversial. Cardiac MRI (CMR) is gold standard for evaluate ventricular function and volumes. We aimed assess the impact of CSF on ventricular function by Cardiac MRI and CMR based deformation imaging.

Methods: 12 subjects with CSF and 10 subjects with normal flow and normal cardiac function were compared by CMR and CMR strain.

Results: LV and RV functions and volumes were similar. There was no difference between CMR strains in both groups. Furthermore, there was no correlation between age and heart function in patient with CSF.

Conclusions: CSF has no or limited impact on the cardiac functions. Further long-term prospective studies should be carried out to establish the impact and significance of CSF in patients with CSF.

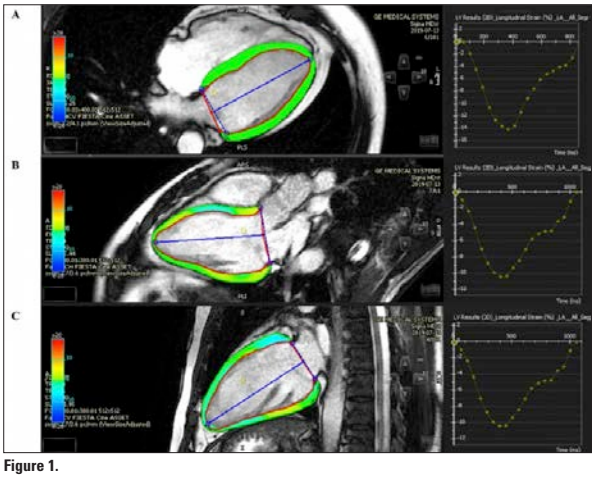

Figure 1.

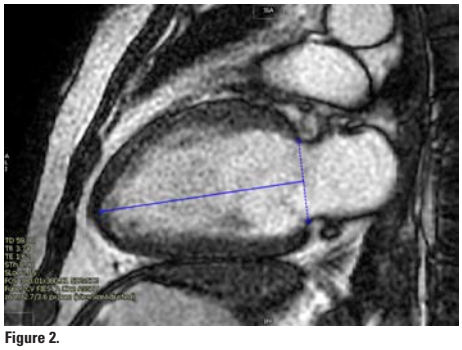

Figure 2.

Cardiac MRI strain parameters			
Variables	CSF group n=12	Control group n=10	p value
Global longitudinal strain	-12.63 ± 2.06	-13.14 ± 2.65	0.639
4 CH longitudinal strain	-12.78 ± 2.53	-13.21 ± 3.20	0.731
3 CH longitudinal strain	-12.43 ± 3.33	-12.73 ± 2.33	0.820
2 CH longitudinal strain	-11.80 ± 1.70	-13.64 ± 3.64	0.177
Global Radial Strain	19.40 ± 4.21	19.92 ± 5.88	0.825

Cardiac imaging / Echocardiography

OP-148

Left atrial dimension to left ventricle ejection fraction ratio predicted MACE more than left ventricle ejection fraction in patients with acute coronary syndrome treated with percutaneous coronary intervention

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Background and Aim: It is substantial to determine who are in high-risk following primary percutaneous coronary intervention (P-PCI) in patients with acute coronary syndrome (ACS). Left ventricle ejection fraction (LVEF) and left atrial diameter (LAD) are the most important parameter obtained from transthoracic echocardiography (TTE) for risk stratification. We evaluated the value of LAD to LVEF rate (LADEFr) for the prediction of major adverse cardiovascular events (MACE) in patients with ACS who underwent P-PCI.
Methods: A total of 262 patients admitted to the emergency department and diagnosed with acute coronary syndrome (ACS) were included in this study. All patients underwent TTE examination before discharge. The composite primary endpoint of the study was all-cause mortality and new onset heart failure (HF) during 2 years follow up.
Results: A total of 262 patients were included in the study. The mean age was 62.1±11.5 years and 39 (18.3%) were female. MACE was defined as in-and-out hospital all-cause mortality and new-onset HF and occurred in 73 (28%) patients during a mean of 2 years. In the backward multivariate Cox regression analysis, age [OR=1.037, 95%CI: 1.016-1.058, p<0.001], Killip class [OR=2.097, 95%CI: 1.009-4.361, p=0.047], creatinine [OR=2.200, 95%CI: 1.271-3.807, p=0.005], LADEFr [OR=1.027, 95%CI: 1.018-1.036, p<0.001] were independent predictor of MACE during 2 years follow up.
Conclusions: In patients who were performed P-PCI for the treatment for ACS age, Killip class, creatinine, and LADEFr were independent predictors of MACE during 2 years follow up.

Cardiac imaging / Echocardiography

OP-149

Evaluation of left ventricular functions in individuals with iron deficiency anemia by conventional echocardiography and strain imaging method

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Background and Aim: Iron deficiency anemia is a common public health problem. Iron deficiency anemia affects many systems, its effects especially on the cardiovascular system are very important. It is also an important cause of morbidity and mortality in the heart failure. Although anemia is common in patients with heart failure, there is no data on whether left ventricular dysfunction develops in anemic patients. In our study, we aimed to evaluate left ventricular functions by conventional echocardiographic parameters and the presence of subclinical left ventricular dysfunction with two-dimensional (2D) and three-dimensional (3D) strain imaging in individuals with mild to moderate iron deficiency anemia without known cardiac disease.
Methods: A total of 72 anemia patients, 3 (4.2%) males and 69 (95.8%) females aged between 21-62 (mean age 37.8), were included in the study. 38 healthy volunteers were included in the control group (Table 1). The patients were divided into two groups as moderate (7-9 g / dL) and mild (≥9-12 g/dL) severity anemia according to Hemoglobin (Hb) values and were named as Group A and Group B, respectively.The Hb value of the control group was ≥12 g/dL and it was named as Group C. Group A consisted of 19 patients (26.4%) with an average Hb value of 7.6 g/dL. Group B consists of 53 (73.6%) patients and the average Hb value was measured as 11.3 g/dL. The average Hb value of Group C was calculated as 13.8 g/dL.
Results: When the groups were compared in terms of conventional echocardiographic parameters, there was no significant difference. Among diastolic parameters, E/A ratio and lateral A' were lower in the anemic group (p=0.045, p=0.001) (Table 2, 3). As a result of strain evaluation, no significant difference was found in 2D and 3D Global Longitudinal Strain (GLS) values (p=0.241 for 2D GLS, GLS p=0.423 for 3D). Three-dimensional Ejection Fraction (EF) value was lower in the anemic patient group (58.5% versus 60.89%, p=0.038) (Table 4, 5). A significant correlation was shown between ferritin and 2D GLS and 2D EF (r=0.307, p<0.01; r=-0.301, p<0.05) (Table 6).
Conclusions: Mild to moderate iron deficiency anemia does not lead to a significant change in left ventricular diastolic and systolic functions and left ventricular global longitudinal strain values.

Table 1. Demographic characteristics of the study population

	Patient (n=72)	Control (n=38)
Age	37,82 ± 9,98	43,34 ± 8,26
Sex (female/male)	69/3	36/2
IDA time (ay)	18,9±23,1	-
Heart rate (atım/dk)	85 ± 10	79 ± 9
BMI (kg/m2)	24,2 ± 2,5	25,6 ± 2,9
SBP (mmHg)	113 ± 9	121 ± 7
DBP (mmHg)	79 ± 5	83 ± 8

Abbreviations: IDA, Iron Deficiency Anemia; BMI, Body Mass Index; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure

Table 2. Echocardiographic parameters of the study population

	Patient (n= 72)	Control (n=38)	p
General echocardiographic measurements			
Aortic diameter (cm)	2,83 ± 0,83	2,93 ± 0,25	**0.035**
Left atrium diameter (cm)	3,39 ± 0,39	3,5 ± 0,37	0.059
LVDd (cm)	5,16 ± 0,25	4,56 ± 0,38	0.960
LVDd (cm)	3,30 ± 0,56	2,84 ± 0,43	0.297
IVST (cm)	1,00 ± 0,10	0,92 ± 0,28	0.129
PW (cm)	0,90 ± 0,14	0,93 ± 0,11	0.359
PAP (mm Hg)	24,05 ± 3,70	24,96 ± 5,88	0.106
TAPSE (mm)	25,13 ± 4,15	24,52 ± 2,87	0.361
LVMI (g/m²)	84,0 ± 5,20	79,3± 6,30	0.255
Diastolic parameters			
Mitral E (cm/sn)	84,86 ± 18,55	79,78 ± 13,16	0.186
Mitral A (cm/sn)	63,98 ± 15,44	67,06 ± 18,64	0.163
E/A	1.38 ± 0.38	1.60 ± 2.32	**0.045**
DT (msn)	194,74 ± 37,98	202,35 ± 48,71	0.534

Abbreviations: LVDd, Left Ventricular End-diastolic Diameter; LVDs, Left Ventricular end-systolic Diameter; IVST, Interventricular Septum Thickness; PW, Posterior Wall Thickness; PAP, Pulmonary Artery Pressure; TAPSE, Tricuspid Annular Plane Systolic Excursion; LVMI, Left Ventricular Mass Index; DT, Deceleration Time

Table 3. Echocardiographic parameters of the study population

	Patient (n= 72)	Control (n=38)	p
Tissue Doppler parameters			
Lateral E'(cm/sn)	14,14 ± 3,98	13,85 ± 3,58	0.667
Lateral A'(cm/sn)	8,98 ± 2,00	11,32 ± 3,60	**0.001**
Lateral S (cm/sn)	9,79 ± 2,66	10,00 ± 2,63	0.539
Septal E' (cm/sn)	11,73 ± 2,43	11,38 ± 2,56	0.608
Septal A' (cm/sn)	9,00 ± 2,10	9,82 ± 2,71	0.185
Ejection Fraction and Global Longitudinal Strain parameters			
2D EF (%)	64,73 ± 5,28	65,62 ± 4,82	0.360
3D EF (%)	58,52 ± 6,25	60,89 ± 5,06	**0.038**
2D GLS (%)	-21,08 ± 3,63	-20,15 ± 3,19	0.241
3D GLS (%)	-15,29 ± 5,16	-14,38 ± 4,83	0.423

Abbreviations: 2D EF, Ejection Fraction Evaluated by Two-dimensional Echocardiography, 3D EF, Ejection Fraction Evaluated by Three-dimensional Echocardiography; 2D GLS, Global Longitudinal Strain Evaluated by Two-Dimensional Echocardiography; 3D GLS, Global Longitudinal Strain Evaluated by Three-dimensional Echocardiography

Table 4. Echocardiographic parameters of anemia groups and control group

	Group A	Group B	Group C	p
General echocardiographic measurements				
Aortic diameter (cm)	2,79 ± 0,22	2,85 ± 0,25	2,93 ± 0,27	0.110
Left atrium diameter (cm)	3,36 ± 0,52	3,44 ± 0,46	3,49 ± 0,34	0.435
LVDd (cm)	4,63 ± 0,38	5,37 ± 0,24	4,56 ± 0,38	0.696
LVDd (cm)	2,96 ± 0,37	3,43 ± 0,23	2,86 ±0,43	0.355
IVST (cm)	1,00 ± 0,00	1,00 ± 0,00	0,92 ±0,28	0.315
PW (cm)	0,92 ± 0,06	0,89 ± 0,16	0,94 ±0,11	0.393
PAP (mm Hg)	23,93±4,46	24,66±3,76	24,41±5,71	0.526
TAPSE (mm)	25,78±4,12	24,73±4,21	24,64±2,83	0.753
LVMI (g/m^2)	82 ± 3,10	86 ± 1,10	80 ± 4,30	0.452
Diastolic parameters				
Mitral E (cm/sn)	90,78 ± 19,03	82,41 ± 18,36	80,08 ± 12,86	0.216
Mitral A (cm/sn)	63,67 ± 16,75	65,00 ± 16,04	65,86 ± 17,67	0.600
E/A	1.49 ± 0.37	1.33 ± 0.40	1.61 ± 2.29	0.139
DT (msn)	184,00 ± 42,83	200,68 ± 36,13	199,69 ± 47,96	0.336

Abbreviations: LVDd, Left Ventricular End-diastolic Diameter; LVDs, Left Ventricular end-systolic Diameter; IVST, Interventricular Septum Thickness; PW, Posterior Wall Thickness; PAP, Pulmonary Artery Pressure; TAPSE, Tricuspid Annular Plane Systolic Excursion; LVMI, Left Ventricular Mass Index; DT, Deceleration Time

Table 5. Echocardiographic parameters of anemia groups and control group

	Group A	Group B	Group C	p
Tissue Doppler parameters				
Lateral E'(cm/sn)	15,35 ± 5,57	13,44 ± 3,14	14,17 ± 3,56	0.644
Lateral A'(cm/sn)	8,88 ± 2,34	9,22 ± 2,39	11,00 ± 3,33	**0.015**
Lateral S (cm/sn)	10,76 ± 3,77	9,38 ± 2,00	10,06 ± 2,65	0.453
Septal E' (cm/sn)	12,31 ± 2,02	11,51 ± 2,59	11,40 ± 2,52	0.432
Septal A' (cm/sn)	8,69 ± 2,44	9,02 ± 2,05	9,91 ± 2,59	0.140
Ejection Fraction and Global Longitudinal Strain parameters				
2D EF (%)	64,25 ± 4,93	64,84 ± 5,42	65,73 ± 4,85	0.495
3D EF (%)	57,78 ± 6,50	58,93 ± 6,29	60,63 ± 5,04	0.164
2D GLS (%)	-19,85 ± 2,95	-21,61 ± 3,75	-20,20 ± 3,28	0.119
3D GLS (%)	-14,39 ± 4,97	-15,81 ± 5,31	-14,33 ± 4,74	0.427

Abbreviations: 2D EF, Ejection Fraction Evaluated by Two-dimensional Echocardiography, 3D EF, Ejection Fraction Evaluated by Three-dimensional Echocardiography; 2D GLS, Global Longitudinal Strain Evaluated by Two-Dimensional Echocardiography; 3D GLS, Global Longitudinal Strain Evaluated by Three-dimensional Echocardiography

Table 6. Correlation of hemoglobin and ferritin parameters with ejection fraction and global longitudinal strain parameters

	Hemoglobin	Ferritin
2D EF (%)	0.137	0.307**
3D EF (%)	0.086	0.255*
2D GLS (%)	-0.170	-0.301*
3D GLS (%)	0.088	-0.064

*p<0.05, **p<0.01

r =0.000-0.300 weak relationship
0.301-0.600 medium strength relationship
0.601-0.800 strong relationship
0.801-1.00 very strong relationship

Abbreviations: 2D EF, Ejection Fraction Evaluated by Two-dimensional Echocardiography, 3D EF, Ejection Fraction Evaluated by Three-dimensional Echocardiography; 2D GLS, Global Longitudinal Strain Evaluated by Two-Dimensional Echocardiography; 3D GLS, Global Longitudinal Strain Evaluated by Three-dimensional Echocardiography

Cardiac imaging / Echocardiography

OP-150

Cardiac CT angiography and high-sensitivity troponins for evaluation of acute chest pain, a single center experience

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Background and Aim: Accurate diagnosis of acute chest pain (ACP) remains one of the most challenging problems in the emergency department (ED). While the proportion of patients that present with myocardial infarction (MI) is relatively low, a missed diagnosis can be life threatening. Electrocardiography (ECG) and high-sensitivity troponin (Hs Trp) assays have been increasingly incorporated as a rapid and efficient diagnostic test in the triage of ACP due to their higher sensitivity and negative predictive value of myocardial infarction. Sometimes Hs Trp and ECG may not be diagnostic in acute chest pain. Additional diagnostic tests may be needed for a definitive diagnosis. Coronary computed tomography angiography (CCTA) has developed as a strong diagnostic tool in the triage of ACP over the past decade, with several trials showing that it can reliably identify patients at low risk of major adverse cardiovascular events, shorten the length of stay in the ED. We aim to show effectiveness of using CCTA for evaluation intermediate-risk patients with suspected ACS, non-conclusive troponins and ECG results.

Methods: 138 patients with normal echocardiography and ECG findings who admitted to the emergency department with acute chest pain were included in the study. CCTA, HsTrp T and other biochemistry tests were done quickly. Patients with myocarditis, pericarditis, severe troponin elevation (>2 folds) and ECG changes during follow up were excluded from the study.

Results: Mean chest pain duration of the patients (83 men 60.1%; and 56.6±15.3 years) was 3.0±2.6 hours. The baseline clinical characteristics of patients are shown in Table I. HsTrp T values were determined above the upper limit (≥14 ng/dl) in 52 (37.7%) patients. 33 (23.9%) patients who underwent CCTA, had significant coronary artery stenosis and 105 of patients had normal coronary arteries. 21 (63.6%) troponin positive patients and 12 (36.4%) troponin negative patients had significant coronary artery stenosis in CCTA. Additionally, 31 of (29.5%) CCTA negative patients ' Hs Tr T results were positive and 21 (63.6%) of CCTA positive patients' Hs Tr T results were positive. CCTA positive patients were hospitalized and underwent conventional coronary angiography. Coronary angiography revealed significant stenosis in 27 (81.8%) patients, 20 of the patients were underwent percutaneus coronary intervention and 7 of the patients were underwent coronary bypass grefting. Our results showed that CCTA had 100 % sensitivity and 94.5% specificity for accurate diagnosis of acute chest pain in our ED (Table 2, 3).

Conclusions: CCTA seems to be very useful for diagnosis in patients presenting to the emergency department with acute chest pain, especially may be most valuable for intermediate-risk patients with non-conclusive troponins and ECG results. These results will require further investigation and new prospective trials.

Table 1. Main Characteristics of Patients

	N:138
Age (year)	59.6± 15.3
Men (n%)	83 (60.1%)
HF (n%)	6(4.3%)
HT (n%)	63(45.7 %)
HL (n%)	32 (23.2 %)
DM (n%)	23 (16.7 %)
Smoker (n%)	55 (39.9%)
Previous Miyocardial Infarction(n%)	8(5.8 %)
Previous CABG(n%)	7 (5.1%)
Family History (n%)	12(8.7%)
CVD (n%)	3(2.2%)
AF(n%)	10 (7.3%)
Creatinine (mg/dl)	0.8±0.1
EF(n%)	58.5±4.4
Troponin peak (ng/dl)	19.91 4.8
CRP (mg/l)	10.5 ±2.1
WBC	8.1±2.6
HGB	13.2±1.7
PLT	253±80.1
Significant coronary artery stenosis in CCTA(n%)	33(23.9%)
Medical Treatment after Coronary Angiography	6(18.1%)
Percutaneus coronary intervention after Coronary Angiography (n%)	20(60.6%)
CABG after Coronary Angiography (n%)	7(21.2%)

HT: Hypertension, DM: Diabetes Mellitus, HL: Hyperlipidemia, CVD: Cerebrovascular disease, AF: Atrial fibrillation, EF : Ejection fraction, HF: Heart failure, CCTA: Coronary computed tomography angiography

Table 2.

	Troponin positive patients n: 52	Troponin negative patients n:86	p
Significant coronary artery stenosis in CCTA	21 (63.6%)	12(36.4%)	<0.001
Significant coronary artery stenosis in Coronary Angiography(n%)	16 (30.8%)	11 (12.8%)	0.010

Table 3.

	Significant coronary artery stenosis in CCTA N:33	Normal coronary artery in CCTA N:105	p
Troponin positive patients	21 (63.6%)	31(29.5%)	<0.001
Significant coronary artery stenosis in Coronary Angiography(n%)	27 (81.8%)	105 (100%)	<0.001

Cardiac imaging / Echocardiography

OP-151

Evaluation of left atrial volume and function in patients receiving peritoneal dialysis using real-time three dimensional echocardiography

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Background and Aim: Left atrial (LA) volume has defined as an independent predictor of mortality in several conditions. Therefore, numerous studies have been conducted in order to reveal LA changes in dialysis patients. However, these studies have been performed with two dimensional echocardiography. This study aimed to evaluate the LA volume and functions in patients receiving peritoneal dialysis (PD) by using real-time three-dimensional echocardiography (RT3DE).

Methods: Twenty-five patients with a diagnosis of ESRD and 25 age and sex-matched controls enrolled in the study. Due to all patients with ESRD had HT, the control group selected in patients with HT. Subjects were excluded from the study if they were known to have heart failure, any evidence of CVD, valvular heart disease, cardiomyopathy, arrhythmias, reduced echogenicity, and malignancy. Those with DM were also excluded from the study to avoid a confounding effect on LA volume and functions. Transthoracic echocardiography was performed in order to evaluate LA phasic volumes and functions.

Results: Baseline characteristics of study population listed in Table1. The Mitral E/e', Left ventricular mass index (LVMI), and deceleration time of the PD group was significantly higher than those of the controls. In contrast, septal E and lateral E wave of controls were significantly higher than those of PD (Table 2). Left atrial phasic volumes (maximal volume index, minimal volume index, pre-atrial contraction volume index) were significantly higher in patients receiving PD (Table 3) (Figure 1). Left atrial phasic volumes were correlated with LVMI and age. Residual renal function was correlated with LA phasic volumes, LVMI, and mitral E/e'(Figure 2).

Conclusions: The current study demonstrated that significant alterations in LA phasic volume using RT3DE in uremic patients receiving PD compared to hypertensive subjects. It seems that the mechanisms of LA dysfunction in PD patients beyond to effects of HT. Further large scale studies are needed to elucidate mechanisms of LA dysfunction in these patients. Real-time three-dimensional echocardiography could identify the diagnosis of LA enlargement and dysfunction more accurately than two-dimensional echocardiography. Structural abnormalities in the heart intensify as RRF decreases in uremic patients receiving PD.

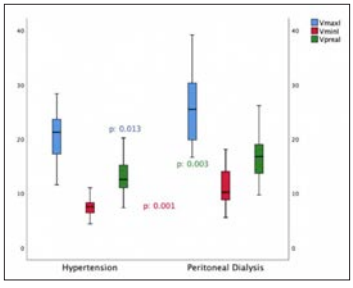

Figure 1. Box plot for Vmax, Vmin, Vprea. Vmax: Maximum left atrial volume; Vmin: Minimum left atrial volume; Vpre A: Before left atrial contraction volume.

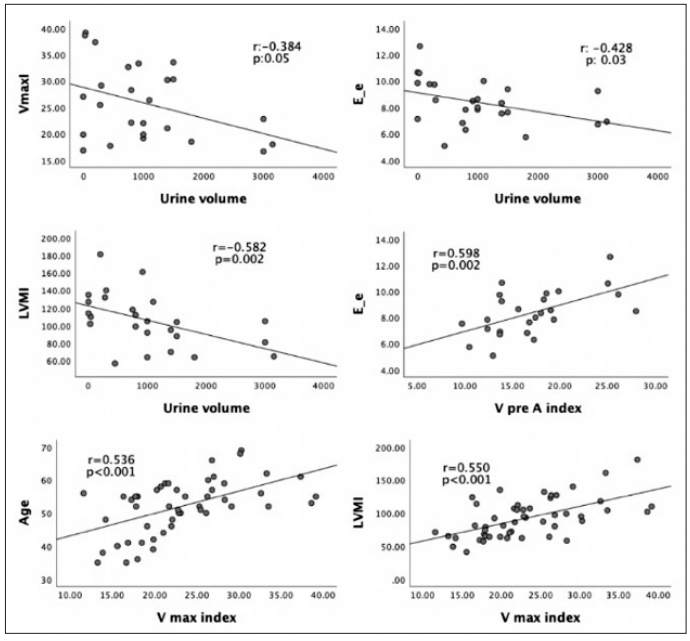

Figure 2. Correlation plot between Urine volume and Vmax index (A); Urine volume and mitral E/e' (B); Urine volume and LVMI (C) in patients receiving peritoneal dialysis. Correlation plot between Vpre A index and mitral E/e'(D); Vmax index and age (E); Vmax index and LVMI (F) in all study population. Vmax: Maximum left atrial volume, Vmin: Minimum left atrial volume, VpreA: Before left atrial contraction volume. LVMI: Left ventricle mass index.

Table 1. Baseline demographic and biochemical characteristics of study population

Variables	Peritoneal Dialysis with Hypertension (n:25)	Hypertension (n:25)	P value
Age, years	52 (18)	55 (5)	0.547
Sex, male (%)	13 (52%)	12 (52%)	1.0
BMI, kg/m²	24.8 (3.8)	25.7 (3.3)	0.45
BUN, mg/dl	53 (28)	16 (9)	**<0.001**
Creatinine, mg/dl	8.6 (5)	0.7 (0.2)	**<0.001**
Albumin, g/dl	3.9 (0.6)	4.4 (0.7)	**<0.001**
Total cholesterol, mg/dl	215 (43)	198 (54)	0.250
HDL cholesterol, mg/dl	46 (12)	52 (20)	0.061
LDL cholesterol, mg/dl	136 (49)	123 (50)	0.068
Triglyceride, mg/dl	146 (122)	107 (137)	**0.023**
Hemoglobin, g/dl	10.8 (1.1)	14.4 (1.4)	**<0.001**
Leucocyte, 10^3 ul	8.1 (2.9)	5.4 (1.9)	**<0.001**
CRP, mg/L	6.2 (23)	3 (0.1)	0.15
Duration of PD, months	52 (53)		
Type of PD, CAPD, n (%)	21 (86%)		
Parathormone levels, pg/mL	450 (337)		
Cause of ESRD			
Hypertension, n (%)	9 (36%)		
Glomerulonephritis, n (%)	5 (20%)		
Polycystic kidney, n (%)	4 (16%)		
VUR, n (%)	1 (4%)		
Unknown, n (%)	6 (24%)		

BMI: Body mass index; **BUN**: Blood urea nitrogen; HDL: High density lipoprotein; **LDL**: Low density lipoprotein; **CRP**: C reactive protein; **PD** : Peritoneal dialysis; **CAPD:** Continuous ambulatory peritoneal dialysis; **ESRD:** End stage renal disease **VUR:** Vesico-ureteral reflux. Data presented as median (interquartile range)

Table 2. Conventional echocardiographic characteristics of the study population

Variables	Peritoneal Dialysis with hypertension (n:25)	Hypertension (n:25)	P value
LV end diastolic diameter (mm)	48 (6)	46 (5)	0.110
LV end systolic diameter (mm)	31 (7)	28(4)	0.064
LV ejection fraction	64 (5)	63 (4)	0.304
Left atrial diameter (mm)	37 (10)	34 (6)	0.185
Interventricular septum thickness (mm)	11 (8)	9 (4)	**0.005**
Posterior wall thickness (mm)	10 (3)	8 (2)	**0.017**
Mitral E (m/s)	0.6 (0.30)	0.65(0.16)	0.148
Mitral A (m/s)	0.78 (0.28)	0.7 (0.15)	0.680
Deceleration time	201(110)	191(65)	**0.04**
Septal e'	7 (2.4)	8.2 (2.9)	**0.002**
Septal a'	9.5 (2.7)	10.8 (2.1)	**0.027**
Lateral e'	9 (4.1)	10.3 (3.4)	**0.004**
Lateral a'	10.3 (2.5)	12.2 (3.3)	**0.017**
Mitral E/A ratio	0.86 (0.73)	0.82 (0.43)	0.327
E/e' ratio	8.3 (2.7)	7.3 (2.4)	**0.049**
LV mass index (g/m²)	105 (42)	74 (32)	**0.01**

LV: Left ventricle; Data presented as median (interquartile range).

Table 3. Comparison of three dimensional left atrial volume and function between PD patients and controls

Variables	Peritoneal Dialysis with hypertension (n:25)	Hypertension (n:25)	P value
V max index (ml/m²)	25.5 (12)	21.3 (7.7)	**0.013**
V min index (ml/m²)	10.3 (5.4)	7.6 (3)	**0.001**
V pre A index (ml/m²)	16.8 (5.5)	12.6 (4.6)	**0.003**
Total Emptying fraction (%)	53.8 (19.3)	60 (11.5)	0.190
Active Emptying fraction (%)	30.6 (26.6)	38.6 (13.9)	0.237
Passive Emptying fraction (%)	31.6 (15.6)	36.3 (12)	0.273
Expansion index	116.5 (114)	150 (71)	0.197
Total stroke volume index (ml/m²)	15.2 (12)	12.3(5.7)	0.184
Active stroke volume index (ml/m²)	4.6 (4.5)	4.7 (2.3)	0.662
Passive stroke volume index (ml/m²)	8.5 (7.1)	8.1 (3.7)	0.308

V max: Left atrial maximum volume; **V min:** Left atrial minimum volume **Vpre A:** Pre-atrial contraction volume. Data presented as median (interquartile range).

Cardiac imaging / Echocardiography

OP-154

Effect of empaglifozin in treatment of diabetes to the diastolic fonctions

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Background and Aim: Emphaglifosin is used in the treatment of diabetes in the sodium glucose co-transport-2 protein inhibitor group and is an agent that blocks renal glucose reabsorption. Empaglifosin has been reported to reduce cardiovascular mortality and hospitalizations due to heart failure. The aim of this study was to evaluate the effect of empaglifosine treatment on diastolic function in patients with diabetes mellitus (DM) receiving empaglifosine treatment.

Methods: The study included clinical patients who had diabetes and had not cardiac history. 78 patients with empaglifosine added to the treatment by endocrinology were included as study group, and 80 patients without empaglifosine added as control group. The echocardiography was performed to the patients at the first visit and 6 months after the 2nd visit. Echocardiographic measurements were conducted and mitral E velocity, lateral e' velocity, septal 'e velocity, E/e' ratio, left atrial volume index, tricuspit regurjitant velocity were calculated.

Results: There were no significant differences between the groups with respect to basal clinical and laboratory characteristics exclude patients weight, body mass index and end diastolic left ventricul diameter. According to the sixth month results of our study; we found a significant difference in favor of empaglifosine in the lateral e' velocity (p<0.0001), septal e' velocity (p<0.0001) and E/e' ratio (p<0.0001) parameters which indirectly showed lower left ventricular filling pressure when compared with the control group.

Conclusions: During the follow-up at 6 months, significant improvements were observed in diastolic dysfunction with DM receiving empaglifosine treatment. This outcome may indicate that empaglifosine treatment may correct diastolic dysfunction. Randomize clinical trials studies are needed to reveal possible mechanisms of action.

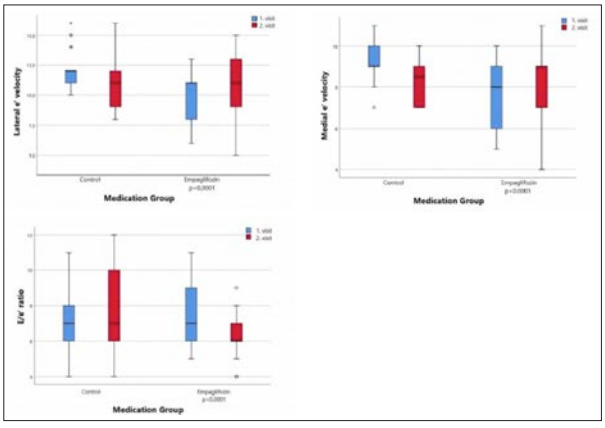

Figure 1. Lateral E, Medial E, E/e' velosities.

Table 1. Comparison of empaglifozin and control groups first and six mount later visits results

	Control			Empaglifosin			
	First visit	6Th mount later	In group p	First visit	6Th mount later	In group p	Intergroup p
LV End Diastolic diameter	48,63 ± 3,58	48,02 ± 3,17	0.0001	48,6 ± 3,03	47,63 ± 3,23	0.0001	0,070
LV ned sistolik diameter	29,96 ± 2,33	29,04 ± 1,89	0.0001	31,91 ± 2,72	30,51 ± 2,32	0.0001	0,194
interventrikuler septum diameter	10,25 ± 0,69	10,21 ± 0,68	0,1573	10,47 ± 0,78	10,28 ± 0,67	0,0020	0,014
Posterior Wall diameter	9,96 ± 0,54	9,91 ± 0,51	0,083	10,18 ± 0,5	9,89 ± 0,59	0.0001	0,002
LVEF	61,88 ± 2,44	61,88 ± 2,44	1,000	60,79 ± 2,27	60,96 ± 2,2	0,157	0,159
E Velosity	73,98 ± 11,55	68,96 ± 13,29	0.0001	63,05 ± 10,9	59,47 ± 10,44	0.0001	0,159
A Velosity	85,38 ± 11,11	84,2 ± 11,58	0,4354	85,91 ± 15,74	85,46 ± 13,26	0,4780	0,159
Laterral e' velosity	11,9 ± 1,47	10,82 ± 2,19	0.0001	9,72 ± 2,06	10,89 ± 2,21	0.0001	0.0001
Medila e' velosity	9,32 ± 0,97	8,23 ± 1,18	0.0001	7,56 ± 1,72	8,21 ± 1,54	0.0001	0.0001
E/e' ratio	7,02 ± 1,17	7,61 ± 2,23	0.003	7,61 ± 1,63	6,42 ± 1,19	0.0001	0.0001
LAVI	28,41 ± 10,88	27,63 ± 11	0,1246	28,81 ± 7,27	28,7 ± 7,73	0,1290	0,986
TRV	2,13 ± 0,19	2,1 ± 0,15	0,0614	2,2 ± 0,18	2,19 ± 0,15	0,2720	0,862
SPAB	22,36 ± 3,06	22,27 ± 2,12	0,7116	23,49 ± 3,08	23,96 ± 2,88	0,9580	0,944

Cardiac imaging / Echocardiography

OP-155

Evaluation of endothelial function patient infected with SARS-CoV-2 virus

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Background and Aim: Vascular endothelium is an active organ with paracrine, autocrine, and endocrine functions that is vital for regulation of vascular tone and the maintenance of vascular homoeostasis. Endothelial dysfunction is the primary factor of microvascular dysfunction characterized by vasoconstriction and subsequent organ ischemia, inflammation associated with tissue edema, and pro-coagulant state. SARS-CoV-2 infects the host using the angiotensin converting enzyme 2 (ACE2) receptor, which is expressed in several organs, including the lung, heart, kidney, and intestine. ACE2 receptors are also expressed by endothelial cells. Endothelial cell infection and endotheliitis were currently demonstrated histologically in COVID-19 patients. Whether vascular derangements in COVID-19 are due to endothelial cell involvement by the virus is currently unknown.In this study we investigated early-mid term effect of SARS-CoV-2 viruse on endothelial functions in patient with COVID-19 infection.

Methods: We included 51 COVID-19 patient (27 symptomatic, 24 asymptomatic) and 54 healthy controle in this study. Endothelial function was assessed by measuring endothelial-dependent flow-mediated vasodilatation (FMD %) and nitroglycerin-mediated dilatation (NMD %) in the brachial artery. We enrolled COVID-19 patients in the study after three negative PCR test and median duration from PCR negativity was 40.4±13.4 days.

Results: Age and gender distribution were well matched between groups (38.2±8.4 vs 38.1±11.4; p=0.95). Although controle patient have higher BMI than COVID patients, BSA was similar in both groups. Whilst hyperlipidemia and smoking habitus were similar between the groups, there were more hypertensive patient in controle group. Plasma CRP (2.19±1.83 vs. 0.63±1.17 p=0.001), Total cholesterol (206.9±38.8 vs. 181.1±49.2 p=0.005) and LDL cholesterol (127.9±37.7 vs. 109.2±40.9, p=0.015) levels were found significantly high in patient with COVID-19 infection. In patients who have had COVID-19 infection, FMD% was significantly impaired compared to patients with controle (8.5±3.27 vs.10.4±2.77, p=0.002), however no significant difference was observed in NMD% (11.7±2.36 vs. 12.0±2.52, p=0.55).

Conclusions: In this study we found that endothelial function assessed by endothelium-dependent vasodilation was significantly impaired in patients who have had COVID-19 infection before average 40 days ago. We determined that abnormalities in arterial function may persist for at least 6 weeks after COVID-19 infection. These could help to explain in part the earlier reported increase in cardiovascular risk during the first weeks after COVID-19 incfection. In the current situation because of little known about long term residual adverse effect of COVID-19 on arterial endothelial function and cardiovascular system, more comprehensive and long-term studies are needed in this area.

Table 1. Demographic characteristics and echocardiographic parameters of the COVID-19 and the controle groups

	COVID-19 n=51	Controle n=54	p
	38.2± 8.4		
Age (year)		38.1 ± 11.4	0.95
Gender (F/M) (%)	26 / 25 (50.9% / 49.1%)	27 / 27 (50.0 % / 50.0%)	
BSA (m2)	1.86 ± 0.2	1.9 ± 0.2	0.6
BMI (kg/m2)	26.1 ± 3.3	28.1 ± 3.8	0.016
Smoking (%)	15 (29.4%)	12 (22.2%)	0.17
Hypertension (%)	6 (11.8%)	21 (38.8%)	0.001
Hyperlipidemia (%)	5 (9.8%)	3 (5.5%	0.6
Duration from last PCR negativity (days))	40.4±13.4		
SBP(mm/hg)	114.9±19.8	119.2±10.0	0.31
	74.7±10.3		
DBP(mm/hg)		74.1±9.9	0.8
Heart rate (beat/min)	73.7±10.3	76.2±8.6	0.28
Total cholesterol (mg /dl)	206.9 ± 38.8	181.1 ± 49.2	0.005
LDL-C (mg/dl)	127.9 ± 37.7	109.2± 40.9	0.015
HDL-C (mg/dl)	53.0 ± 11.6	49.0 ± 11.3	0.09
Triglyceride (mg/dl)	122.5 ± 65.3	119.1 ± 81.9	0.82
Creatinine (mg/dl)	0.87±0.2	0.82±0.15	0.3
CRP	2.19±1.83	0.63±1.17	0.001

The data are expressed as mean ± SD for parametric tests, BSA: body surface area; BMI: body mass index; DBP: Diastolic blood pressure; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; SBP: Systolic Blood pressure.

Table 2. Comparison of study groups according to flow-mediated dilatation and nitroglycerin mediated dilation results

	COVID-19 n=51	Controle n=54	p
Baseline vessel diameter (mm)	3.48 ± 0.7	3.27 ± 0.68	0.17
Max vessel diameter (mm)	3.76 ± 0.86	3.6 ± 0.7	0.29
Baseline flow (mL / min)	87.01 ± 22.5	78.9 ±16.4	0.04
Hypremic flow (mL / min)	146.4 ± 37.7	123.2 ± 33.2	0.001
FMD (%)	8.5 ± 3.27	10.4 ± 2.77	0.002
NMD (%	11.7 ± 2.36	12.0 ± 2.52	0.55

The data are expressed as mean ± SD for parametric tests FMD: flow-mediated vasodilation; NMD: nitroglycerin mediated vasodilation.

Cardiac imaging / Echocardiography

OP-156

Evaluation of left and right ventricular dysfunction in patients with congenital muscular disorders using two-dimensional speckle tracking echocardiography

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Background and Aim: Progressive cardiac dysfunction is one of the leading causes of death in patients with congenital muscular disorders (CMD). Cardiac function is measured and followed up by conventional transthorasic echocardiography (TTE) in these patients. Speckle tracking echocardiography (STE) is used for early identification of cardiac dysfunction in some disorders. We aimed to determine whether two-dimensional STE can be used to identify cardiac dysfunction earlier in patients with CMD.

Methods: Twenty seven CMD patients (19 with congenital myopathy and 8 with muscular dystrophy) (40.8±13.8 years, 19 men, 70.4%) with normal cardiac function and 30 healthy control patients (46.3±13.1 years, 19 men, 63.3%) were included. All patients were examined with TTE, tissue Doppler imaging (TDI), and STE. Standard echocardiographic measurements of left ventricular (LV) and right ventricular (RV) systolic and diastolic function were obtained. Speckle tracking analyses were performed by acquiring apical four-, three-, and two-chamber views with the highest possible frame rates.

Results: TDI showed that CMD patients had worse diastolic parameters than controls (e': 9.5±2.7 cm/s vs. 13.2±3.7, p<0.001 and E/e' ratio: 9.1±2.9 vs. 6.45±1.5, p<0.001). A significant decrease in LV and RV global longitudinal systolic strain (GLS) and GLS rate levels were found in patients with CMD (LV GLS: – 14.4±3.5 vs. – 19.0±0.8, p<0.001, LV GLS rate: 0.56±0.37 vs. 0.73±0.12, p=0.019), (RV GLS: -14.0±7.2 vs. -17.9±1.7, p=0.007, RV GLS rate: 0.38±0.21 vs. 0.65±0.18, p<0.001). Additionally, LV GLS and RV GLS were positively corralated with e' (r1: 0. 423, p1: 0.001 and r2: 0.382, p2: 0.003, respectively.) and were negatively correlated with E/e' (r1: -0.408, p1: 0.002 and r2:-0.387, p2: 0.003, respectively).

Conclusions: In adult patients with CMD who have global normal systolic function, systolic and diastolic dysfunction parameters can be detected earlier by STE. The early detection of ventricular dysfunction may warn the physician earlier for the management of the patient during the follow-up period.

Table 1. Main Characteristics of Patients Patients with congenital muscular disorders and Controls

	Patients with congenital muscular disorders (CMD). n: 27	Controls n: 30	p
Age (year)	40.8 ±13.8	46.3±13.1	NS
Men (n%)	19 (70.4%)	19(63.3%)	NS
BSA (m2)	1.81±0.1	1.92±0.2	NS
HT (n%)	3(11.1%)	4 (13.3%)	NS
HL(n%)	1 (3,7 %)	5(16.7%)	NS
Smoker (n%)	3 (11.1%)	3(10.0%)	NS
E/a ratio	1.2± 0.3	1.1 ± 0.2	NS
TAPSE (mm)	25.07±1.2	25.2±1.9	NS
PASP (mmhg)	23.5±4.8	25.5±3.5	NS
LVEF(n%)	60.1±1.2	60.07±1.1	NS
S' wave (cm/sec)	14.4± 0.8	14.4± 1.6	NS
FAC (%)	57.7± 3.3	57.1± 5.8	NS
LVEDD (cm)	4.9± 0.5	4.7± 0.5	0.009
LVESD (cm)	2.8± 0.4	3.1± 0.3	0.009
LA(cm)	5.5± 0.5	5.6± 0.1	NS
RV(cm)	3.2± 0.2	3.3± 0.2	NS
RA(cm)	3.3± 0.3	3.5± 0.1	<0.001
e' (cm/s)	9.5 ± 2.7	13.2 ± 3.7	< 0.001
E/e'	9.1 ± 2.9	6.45 ± 1.5	< 0.001
LV GLS	− 14.4 ± 3.5	− 19.0± 0.8	< 0.001
LV GLS rate	0.56± 0.37	0.65± 0.18	0.019
RV GLS	-14.0 ± 7.2	-17.9 ±1.7	0.007
RV GLS rate	0.38± 0.21	0.65± 0.18	< 0.001

NS: Non significant, HT: Hypertension, HL: Hyperlipidemia, LVEF: Left ventricular ejection fraction., LVEDD: Left ventricular end diastolic diameter, LVESD: Left ventricular end systolic diameter, LA: Left atrium , RA: Right atrium, RV: Right Ventricul, PASP: Pulmonary artery systolic pressure , TAPSE: Tricuspid annular plane systolic excursion, FAC: *Fractional Area Change*, GLS: *Global strain rate*.

Cardiac imaging / Echocardiography

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Evaluation of pulmonary artery stiffness in chronic renal failure

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Background and Aim: Pulmonary hypertension (PH) is one of the complications of chronic renal failure patients. Many of the current screening modalities are dependent on detecting a rise in pulmonary arterial pressure (PAP). However, high capacitance of the pulmonary circulation implies that early microcirculation loss is not accompanied by a change in resting PAP. Therefore, we aimed to demonstrate early changes in pulmonary vascular disease in chronic renal failure patients with a new echocardiographic parameter, called as pulmonary arterial stiffness (PAS).

Methods: Sixty chronic renal failure patients and 60 age- and sex-matched healthy control subjects were enrolled in this study. PAS was calculated echocardiographically by using maximal frequency shift and acceleration time of the pulmonary artery flow trace.

Results: PAS was significantly increased in the chronic renal failure group compared to the control group (19.5±2.4 vs. 11.4±1.3, p<0.001).There was a significant negative correlation between PAS and eGFR level (r=-0.470, p<0.001).

Conclusions: Our results suggest that chronic renal failure affects pulmonary vascular bed starting early onset of disease and this can be demonstrated by an easy-to-measure echocardiographic parameter.

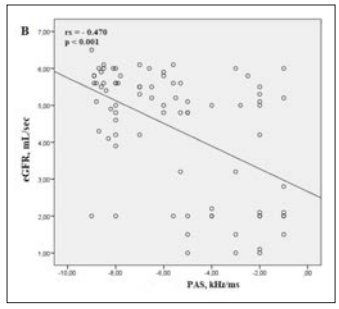

Figure 1.

Table 1. Demographic procedural and clinical data for the study group

Variables	CRF group (n=60)	Control group (n=60)	p-value
Age, years	69.1 ± 10.9	71.7± 11.1	0.070
Gender, male n,(%)	67 (54)	68 (58.6)	0.471
Hypertension n,(%)	92 (74.1)	95 (81.8)	0.102
Diabetes Mellitus n,(%)	44 (35.4)	46 (39.7)	0.073
Hyperlipidemia n,(%)	46 (37.1)	42 (36.2)	0.880
Coronary Artery Disease n,(%)	40 (32.2)	47 (31.9)	0.090
Smoking n,(%)	49 (39.5)	61 (52.6)	0.042
Hemoglobin, g/dL	13.3 ± 1.2	13.9 ± 1.0	0.122
Hematocrit, %	40.0 ± 3.6	42.3 ± 3.5	0.101
White Blood Cell, $10^3/\mu l$	10.2 ± 2.8	10.3 ± 2.9	0.690
Platelet, $10^3/\mu l$	241 ± 73	244 ± 71	0.570
Total Protein, g/L	68.8 ± 2.5	73.4 ± 2.7	0.085
Albumin, g/dL	3.6 ± 0.3	3.9 ± 0.4	0.071
Glucose, mg/dl	105 ± 17	107 ± 14	0.120
Total cholesterol, mg/dL	172 ± 35	169 ± 37	0.770
LDL cholesterol, mg/dl	115.9 ± 23	92.8 ± 24	**0.005**
HDL cholesterol, mg/dl	39 ± 10	36 ± 11	0.880
Triglycerides, mg/dl	118 ± 51	123 ± 56	0.470
Antidiabetic n,(%)	44 (35.4)	46 (39.6)	0.262
Statin n,(%)	50 (40.3)	54 (46.5)	0.183
ACEi/ARB, n (%)	75 (60.4)	78 (67.2)	0.691
CCB n,(%)	23 (18.5)	20 (17.2)	0.380
Beta blocker n,(%)	50 (81)	47 (78)	0.320

LDL, Low-density lipoprotein; HDL, High-density lipoprotein; ACE-i/ARB, Angiotensin converting enzyme inhibitor/angiotensin receptor blocker; CCB, Calcium channel blocker;

Table 2. Ecocardiographic data for the study group

Variables	CRF group (n=60)	Control group (n=60)	p-value
LVEF, (%)	52.8 ± 2.8	54.3 ± 2.4	0.126
TAPSE, mm	16.8 ± 3.5	20.8 ± 2.8	**0.045**
mPAP, mmHg	30.2 ± 3.9	20.5 ± 4.3	**0.004**
PAS, KhZ/ms	19.5 ± 2.4	11.4 ± 1.3	**<0.001**

LVEF: Left ventricular ejection fraction; TAPSE: Tricuspid annular plane systolic excursion; mPAP: mean Pulmonary arterial pressure; PAS: Pulmonary arterial stifness.

Cardiac imaging / Echocardiography

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Association between epicardial adipose tissue thickness and left ventricular diastolic functions

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Background and Aim: Epicardial adipose tissue (EAT) has been found to be associated with the diastolic dysfunction in recent years, but this relationship has not been fully elucidated. Echocardiography is a non-in-

vasive, simple, cost effective and accessible approach to assess EAT thickness, which can be performed easily. The aim of this study was to evaluate the effectiveness of EAT on prediction of diastolic dysfunction.
Methods: A total of 138 patients without any cardiovascular, inflammatory, autoimmune and cancer disease, were enrolled. Subjects were divided into two groups, those with and without diastolic disfunctions. Conventional echocardiography parameters and tissue Doppler imaging (TDI) were performed to evaluate left ventricular functions. EAT thickness on the free wall of the right ventricle in parasternal long-axis view were measured using transthoracic echocardiography.
Results: The patients with diastolic dysfunction were older (57.7±9.8 vs 44.1± 8.5, p<0.001), more likely female (53.1% vs 44.8%), and had more comorbidites including diabetes, hypertension and had increased BMI.The patient characteristics of both groups are showed in table 1.EAT thickness showed a significant positive correlation with age (r=0.479, p<0.001), BMI (r=0.538, p<0.001), diabetes mellitus (r=0.353, p<0.001) and hypertension (r=0.380, p<0.001). There was no correlation between epicardial fat thickness and HDL-C,LDL-C and TG level. Also, EAT was associated with increased left ventricular mass (r=0.399, p<0.001) and reduced diastolic function by lower early diastolic myocardial velocity (e')(r=-0.595, p<0.001), early transmitral inflow velocity (E) (r=-0.399, p<0.001), E/A (r=-0.505, p<0.001) and higher E/e' ratio (r=0.316, p<0.001). Multivariate analysis showed that age (OR, 0.376, 95% CI, 0.009-0.024), hypertension (OR, 0.194; 95% CI, 0.043-0.375), diabetes (OR, 0.284; 95% CI,0.011-0.420), BMI (OR, 0.201; 95% CI, 0.000-0.036) and LVM (OR, 0.181; 95% CI, 0.000-0.004) were independent factors affecting diastolic dysfunction. Also thick EAT was predictor of diastolic dysfunction (OR, 0.225, 95%CI 0.191-1.270) after adjustment for covariates (Table 2). The area under the curve on receiver operating characteristic (ROC) analysis of EAT thickness for predicting diastolic dysfunction was 4.9 with a sensitivity of 75% and specificity of 73% (ROC area 0.820, p<0.001, 95% CI, 0.746-0.893) (Figure 1).
Conclusions: The measurement of echocardiographic EAT thickness seems to be an acceptable method which can be used as an easily, cost affectively, and non-invasively. Increased EAT thickness is independently assotiated with diastolic dysfunction. Adding EAT measurement on top of classic echocardiographic diastolic dysfunction findings may provide further evidence in predicting diastolic dysfunction in daily clinical practice. But large, more definitive studies are needed to confirm these findings.

Table 1. Clinical and echocardiographic characteristics of the study population

Variable	Diastolic dysfunction group, (n=64)	Non-Diastolic dysfunction group (n=58)	p
Age, mean±SD	57.7±9.8	44.1±8.5	<0.001
Female, n (%)	34(53)	26(44.8)	0.364
Diabetes mellitus, n(%)	10(15.6)	0	0.001
Hypertension,n(%)	34(53.1)	4(6.9)	<0.001
Smoking,n(%)	17(26.6)	23(39.7)	0.126
EAT (mm), mean±SD	5.98±1.52	4.32±1.03	<0.001
BMI (kg/m2), mean±SD	30.1±4.7	25.6±3.5	<0.001
CRP mean±SD	3.2±3.8	1.5±3.3	0,019
LDL (mg/dl), mean±SD	148±42	147±36	0.856
HDL (mg/dl), mean±SD	48±12	49±13	0.829
TG (mg/dl), mean±SD	154±77	161±127	0.740
EF, mean±SD	61±4	63±6	0.006
E, mean±SD	59.6±12	74.8±12	<0.001
A, mean±SD	77.7±15	65±10	<0.001
e'(cm/s), mean±SD	7.06±1.2	9.8±1.7	<0.001
a'(cm/s), Mean±SD	9.86±2	9.9±1.9	0.959
E/e', mean±SD	9.1±3	7.8±1.5	0.009
LVEDD (mm), mean±SD	42.4±4.3	40.4±6.1	0.044
IVST (mm), mean±SD	11.1±1.7	9.8±1.7	<0.001
PWT (mm), mean±SD	10.8±1.1	9.8±1.1	<0.001
LVM (g), mean±SD	182.5±40.6	152.8±37.7	<0.001

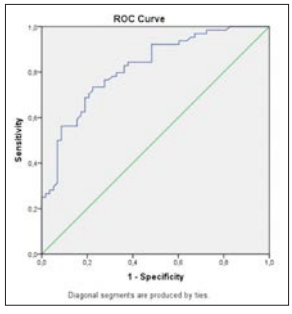

Figure 1. ROC curve (Receiver operating characteristic curve) illustrating the accuracy of epicardial adipose tissue thickness for diastolic dysfunction.

Table 2. Correlation of clinical quantitative variables with diastolic dysfunction and predictors of diastolic dysfunction determined by multi-linear regression analysis

	p	r	OR	CI
EAT	<0.001	0.534	0.225	0.191-1.270
Age	<0.001	0.594	0.376	0.009-0.024
Hypertension	<0.001	0.499	0.194	0.043-0.375
BMI	<0.001	0.477	0.201	0.000-0.036
Diabetes mellitus	0.001	0.284	0.118	0.011-0.422
E/A	<0.001	-0.760	-0.573	-1.372-(-0.769)
E/e'	0.009	0.239	0.278	0.031-0.083
LVM	0.002	0.290	0.181	0.000-0.004

Cardiac imaging / Echocardiography

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Assessment of subtle left ventricular dysfunction by two dimensional strain echocardiography of HIV-1 positive individuals receiving highly active antiretroviral therapy

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Background and Aim: People living with human immunodeficiency virus (PLHIV) has improved long life expectancy by means of new highly active antiretroviral therapy (HAART). Both HIV and HAART may induce myocardial dysfunction by fibrosis, apoptosis and steatosis that caused by increased levels of pro-inflammatory cytokines. PLHIV, who are receiving HAART, are more prone to develop myocardial impairment from subtle left ventricular systolic or diastolic dysfunction to severe dilated cardiomyopathy. Two dimensional longitudinal strain echocardiography has emerged accurate and reproducible assessment of myocardial wall motion and deformation. We compare the echocardiographic findings of PLHIV dolutegravir (DTG) plus abacavir (ABC)/lamivudine (3TC) and dolutregravir (DTG) plus tenofovir (TDF)/emtrisitabin (FTC) treatment with healthy control group. The aim of the study is to evaluate cardiac structure and functions by current echocardiographic techniques of PLHIV treated by DTG/ABC/3TC and DTG/TDF/FTC also compare among themselves.
Methods: We enrolled 106 PLHIV with a mean age of 34.9±8.8 and 56 demographically matched healthy volunteers with a mean age of 33.2±7.3. All of the PLHIV were on HAART (55 of them on DTG/ABC/3TC, 51 of them on DTG/TDF/FTC). Left ventricular systolic ejection fraction, global longitudinal strain and diastolic function parameters were assessed by 2 dimensional strain echocardiography and conventional echocardiography according to current American Society of Echocardiography guidelines. CD4 T-cell counts and HIV-ribonucleic acid (HIV-RNA) values of PLHIV were measured and therapy durations were recorded.
Results: All the patients had normal ejection fraction (>%55) and sinus rhythm at echocardiographic evaluation. The mean CD4 T-cell count and HIV- RNA load were similar in both HIV positive groups. Interventricular septum and posterior wall thickness were increased both in two HIV positive groups. The mean global longitudinal strain was lower both in two HIV positive groups rather than control group but this study did not demonstrate any statistically significant ventricular strain difference between the groups of DTG/ABC/3TC and DTG/TDF/FTC (Table 1).
Conclusions: PLHIV established on different antiretroviral combinations have decreased global strain despite conventional echocardiographic parameters within normal range. Global strain is more sensitive for detecting subtle left ventricular systolic dysfunction, not apparently impaired myocardial function. It is controversial, whether the development of ventricular strain impairment in PLHIV are derived form HAART toxicity or ongoing immune activation and systemic inflammation as a natural pathogenesis of HIV infection.

Table 1. Characteristics and echocardiographic parameters of study population

Table 1	HIV (-) (n: 56)	DTG/ABC/3TC (n: 55)	DTG/TDF/FTC (n: 51)	p value
Age (years)	33.2 ± 7.3	35.1 ± 8.1	34.3 ± 9.3	0.736
Male (n)	76.7 % (43)	81.8 % (n: 45)	86.2 % (n: 44)	0.542
Duration of HAART (years)	-	6.5 ± 2.2	5.8 ± 1.6	0.320
CD4 counts (ceels/uL)	-	538.6 ± 208	551.2 ± 220	0.640
Viral loads (< 50 copies/ml) n (%)		63.6% (35)	76.4% (n: 39)	0.238
GLS (%)	-21.2 ± 1.8	-18.1 ± 2.4	-18.9 ± 2.2	0.044
Tapse (mm)	25 ± 2.1	23.5 ± 1.7	23.6 ± 1.9	0.833
LVEF (%)	65.2 ± 4.3	63.8 ± 5.3	64.4 ± 2.8	0.365
IVS (mm)	9.7 ± 0.8	10.5 ± 1.1	10.4 ± 1.2	0.041
PW (mm)	8.0 ± 0.6	8.4 ± 1.0	8.8 ± 1.1	0.030

NS, nonsignificant; HAART, highly active antiretroviral therapy; GLS, global longitudinal strain; CS, circumferential strain; RS, radial strain; TAPSE, tricuspid annular plane systolic excursion; LVEF, left ventricular ejection fraction; RV GLS, right ventricular global longitudinal strain; IVS, interventricular septum; PW posterior wall.

Cardiac imaging / Echocardiography

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The role of layer specific strain echocardiography in the diagnosis of severe coronary artery disease

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Background and Aim: Several imaging techniques are used for diagnostic and risk assessment in patients with suspected coronary artery disease (CAD) such as echocardiography, myocardial perfusion scintigraphy (MPS), stress imaging tests. Transthoracic echocardiography (TTE) is one of these techniques which allows the evaluation of the left ventricular systolic and diastolic functions. However, non-critical lesions are often observed in elective coronary angiography (CAG) applied patients despite these tests. The calculation of global longitudinal strain (GLS), via 2D speckle tracking method, is a more accurate and reliable technique in contrast to 2D-TTE for the evaluation of left ventricular functions. Layer-specific strain analyses are used for the assessment of myocardial segments on an individual basis, and it provides a quantitative measurement of regional functions. This study aimed determination of the relationship between resting longitudinal

strain analysis and severe coronary lesions in patients with suspected stable coronary artery disease and guidance of patient selection before coronary angiography.

Methods: A total of 242 patients with suspected stable CAD who are planned to be performed selective CAG and are suitable for inclusion criteria were included in this study. Patients were divided into two main groups as those with and without severe CAD. After CAG, patients who have 70% or more stenosis of any vessel or multiple vessels were included in the severe CAD group. In the study, 48.3% (117) of the patients were taken part in the group with severe CAD, and the rest of the patients 42.7% (125) were constituted in the group without severe CAD. Layer-specific GLS were compared between the groups as mid-myocardial, endocardial and epicardial layer by using 2D speckle tracking method.

Results: This study revealed that the GLS values of all layers were significantly lower in the patient group with severe CAD compared with control group (Table 1). ROC curves were constructed to evaluate diagnostic performance of GLS values and the area under the curve was 81-82% in three layers (Figure 1). When the Syntax scores of the patients were calculated according to the multiple comparison tests, it was determined that GLS values were significantly low in 22-32 points group (Table 2). Moreover, GLS values of MPS false-positive and true-positive patients were compared, and it was found that GLS values were significantly lower in the true-positive group than false positive group (Table 3).

Conclusions: In our study, strain values were lower and significant in all layers in patient with suspected coronary artery disease and without wall motion disorder in TTE. LSS assessment is useful for the detection of severe CAD in terms of patient selection. The technique of speckle tracking echocardiography is open to research and needs to be developed. In this way, it provides advanced patient management in diagnosis, treatment, and follow-up, and it gives us a new perspective to the physiology of the heart.

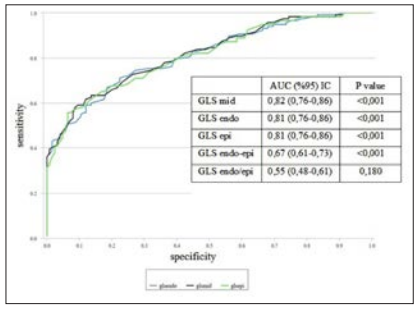

Figure 1. ROC curves demonstrate value of layer specific GLS for the diagnosis of CAD.

Table 1. Conventional echocardiographic parameters, Global-Regional longitudinal strain values and some clinical characteristics of patients

	Non-Significant CAD (n=125)	Significant CAD (n=117)	P value
Echocardiographic Parameters and 2D Global longitudinal strain (GLS) parameters			
LV EF, (%)	65,78±4,43	1,28 ± 0,057	0,156
LV Mass index, (gr/m2)	93,01±17,09	98,12±20,44	0,036
E/A ratio	1,01±0,29	1,05±0,95	0,681
GLS mid-myocardial, %	-21,68 ± 2,27	-18,25 ± 2,92	<0,001
GLS endocardium, %	-24,58 ± 2,57	-20,78 ± 3,31	<0,001
GLS epicardium, %	-19,18 ± 2,05	-16,07 ± 2,72	<0,001
GLS endo-epi, %	5,40 ± 1,07	4,71 ± 1,11	<0,001
GLS endo/epi ratio	1,28 ± 0,057	1,30 ± 0,076	0,10
2D Regional longitudinal strain (RLS) parameters	Non-Significant CAD (n=125)	Significant CAD (n=117)	P value
----LAD----			
RLS mid-myocardium	-21,90 ± 2,88	-18,98 ± 3,89	<0,001
RLS endocardium	-26,01 ± 3,48	-23,02 ± 4,44	<0,001
RLS epicardium	-18,85 ± 2,58	-16,19 ± 3,29	<0,001
----Cx----			
RLS mid-myocardium	-20,75 ± 4,74	-17,56 ± 3,27	<0,001
RLS endocardium	-22,91 ± 3,32	-19,81 ± 3,58	<0,001
RLS epicardium	-18,55 ± 3,19	-18,55 ± 3,19	<0,001
----RCA----			
RLS mid-myocardium	-21,65 ± 3,18	-18,92 ± 3,55	<0,001
RLS endocardium	-23,67 ± 3,43	-20,66 ± 3,93	<0,001
RLS epicardium	-20,01 ± 3,00	-17,90 ± 5,23	<0,001
	Non-Significant CAD (n=125)	Significant CAD (n=117)	P value
Age	56,65±9,57	61,75±9,45	<0,001
Male, n (%)	48 (%38,4)	78 (%66,7)	<0,001
DM, n (%)	26 (%20,80)	50 (%42,7)	<0,001
HL, n (%)	44 (%35,20)	71 (%60,70)	<0,001
HT, n (%)	88 (%70,40)	93 (79,50)	0,104
Smoker, n (%)	58 (%46,40)	70 (%59,80)	0,036

CAD: Coronary artery disease, EF: Ejection fraction, E: Pulsed wave trans-mitral early diastolic velocity, GLS: Global longitudinal strain, LV: Left ventricle, DM: Diabetes Mellitus, HL: Hyperlipidaemia, HT: Hypertension, RLS: Regional longitudinal strain, Cx: Circumflex artery, LAD: Left anterior descending artery, LMCA: Left main coronary artery, RCA Right coronary Artery.

Table 2. The relationship between Syntax scoring and GLS

	Syntax<22 (1. Group) (n=94)	Syntax 22-32 (2. Group) (n= 19)	Syntax>32 (3. Group) (n=4)	P value
GLS mid-myocardium	-18,80 (16,63-20,28)	-15,80 (14,15-18,50)	-21,10 (19,13-21,25)	0,019
GLS endocardium	-21,45 (18,93-22,98)	-18,50 (15,75-21,25)	-23,60 (22,55-24,00)	0,009
GLS epicardium	-16,30 (14,80-18,20)	-14,10 (12,30-16,40)	-18,35 (16,70-19,08)	0,034
GLS endo-epi	4,70 (4,00-5,65)	4,20 (3,45-4,75)	5,35 (4,52-6,35)	0,023
GLS endo/epi	1,29 (1,24-1,34)	1,28 (1,25-1,32)	1,34 (1,24-1,43)	0,486

GLS: Global longitudinal strain.

Table 3. GLS values in MPS true positive and false positive patients

	MPS true positive (n=59)	MPS false positive (n= 51)	p value
GLS mid-myocardium	-18,42 ± 2,81	-21,66 ± 2,23	<0,001
GLS endocardium	-20,97 ± 3,18	-24,51 ± 2,60	<0,001
GLS epicardium	-16,16 ± 2,67	-19,17 ± 1,93	<0,001
GLS endo-epi	4,81 ±1,12	5,34 ± 1,13	0,015
GLS endo/epi	1,30 ± 0,08	1,28 ± 0,05	0,09

GLS: Global longitudinal strain, MPS: Myocardial perfusion scintigraphy.

Cardiac imaging / Echocardiography

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Evaluation of ischemia with speckle tracking echocardiography and MPS in non obstructive coronary artery disease patients

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Background and Aim: 2D STE can detect ischemia at obstructive (≥%50) coronary artery disease(CAD) with high diagnostic sensitivity and specificity. The aim of the study was to evaluate the efficiency of 2D speckle tracking echocardiography in the diagnosis of non-obsructive CAD (<50% stenosis).

Methods: Forty patient with ischemi on myocardial perfusion sintigrapy (MPS) and non-obsructive CAD seen with coronary angiography were included in the study. Strain analysis was performed by 2D speckle tracking echocardiography in all patients. Longitudinal strain was measured for 17 left ventricule myocardial segments. LV segments were grouped as ischemic and non-ischemic segments according to myocard perfusion sintigraphy results and compared.

Results: The mean longitudinal strain values of the segments with ischemia were -20.24±6.00% and of the segments without ischemia were -20.11±6.46% and no statistically significant difference was observed (p>0.05). Subgroup analysis according to gender, hypertension, diabetes mellitus, hyperlipidemia and hypertriglyceridemia showed no difference in longitudinal strain between the ischemic and non-ischemic segments.

Conclusions: Left ventricular 2D longitudinal strain analysis with speckle tracking is not sufficient for the diagnosis of non-obstructive CAD. Again in this patient group, STE can not detect ischemia which could be detected by MPS.

Cardiac imaging / Echocardiography

OP-164

Mitral annular calcification is rrelated with increased level of fibrinogen in patients with acute coronary syndrome

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Background and Aim: Despite it was shown strong relationship between mitral annular calcification (MAC) and atherosclerotic vascular diseases, predictive value of the MAC in the hemostatic process is not fully understood. In this study, we investigated the relationship between the presence of MAC and blood fibrinogen level in patients with acute coronary syndrome (ACS).

Methods: A total of 360 patients admitted to the emergency department and diagnosed with ACS were included in this study. Plasma fibrinogen levels were measured. The patients were grouped according to whether they have MAC or not. Serum fibrinogen levels were compared between MAC groups.

Results: Advanced age (OR: 1.107; 95%CI: 1.063-1.152, p<0.001), Ratio of early diastolic filling velocity-to-mitral annulus velocity (E/e') (OR:1.127; 95%CI: 1.029-1.235; p<0.001), Serum fibrinogen level (OR:1.005; 95%CI: 1.001-1.009; p=0.032) were independent predictors for MAC (+) independent of other variables.

Conclusions: The higher fibrinogen level in patients with MAC + ACS suggests that MAC has an increased hemostatic contribution to the atherosclerosis in ACS.

Cardiac imaging / Echocardiography

OP-165

Epicardial fat volume predicts clinical severity of COVID-19

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Background and Aim: The inflammatory response plays a critical role in coronavirus disease 2019 (COVID-19) and inflammatory cytokine storm increases the severity of COVID-19. Epicardial adipose tissue serves as a source of inflammatory cytokines and mediators. This study aimed to investigate the association between epicardial fat volume (EFV), inflammatory biomarkers and clinical severity of COVID-19.
Methods: This retrospective study included 101 patients hospitalized with COVID-19 between March 11 and April 21, 2020. Laboratory findings, treatment and complications were recorded. The serum inflammatory biomarkers including C-reactive protein (CRP), interleukin-6 (IL-6), procalcitonin (PCT) and ferritin levels were measured. Computed tomographic images were analyzed and semi-automated measurements for EFV were obtained. The primary composite endpoint was admission to intensive care unit (ICU) or death.
Results: The primary composite endpoint occurred in 25.1% (n=26) of patients (mean age 64.8±14.8 years, 14 male). A total of 10 patients died (mean age 71.9±14.3, 6 male). EFV (115.1±44.0 cm^3 vs 94.3±45.5 cm^3, respectively, p=0.037), CRP, PCT, ferritin and IL-6 levels were significantly higher in ICU patients. Moreover, a positive correlation between EFV and CRP (r=0.494, p<0.001), PCT (r=.287, p=0.005), ferritin (r=0.265, p=0.01) and IL-6 (r=0.311, p=0.005) was determined. At receiver operating characteristic analysis, patients with EFV >102 cm^3 were more likely to have severe complications.
Conclusions: Epicardial fat volume and the serum levels of CRP, IL-6, PCT and ferritin can effectively assess disease severity and predict outcome in patients with COVID-19.

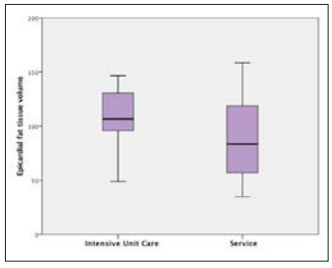

Figure 1. Comparison of epicardial fat volume in patients admitted to ICU and service.

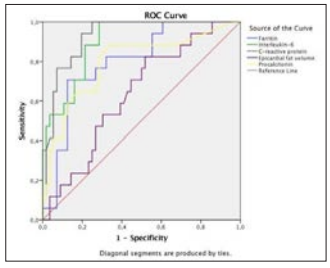

Figure 2. Receiver operating characteristic curve showing sensitivity and specificity of EFV, CRP, IL-6, PCT and ferritin in predicting the severity of COVID-19.

Table 1. Comparison of the laboratory features of the patients with COVID-19 who were admitted to the service and intensive care unit

Variables	Service	Intensive Care Unit	p value
Glucose, mg/dL	110 ± 32	129 ± 39	0.035*
Urea, mg/dL	30 (24-37)	37 (30-54)	0.006*
Creatinine, mg/dL	0.88 (0.75-1.04)	1.01 (0.82-1.15)	0.101
AST, U/L	25 (16-34)	42 (23-67)	<0.001*
ALT, U/L	27 (19-38)	28 (20-52)	0.273
Total protein, g/L	7.0 (6.5-7.2)	6.3 (5.8-6.8)	<0.001*
Albumin, g/L	4.3 ± 0.4	3.9 ± 0.5	<0.001*
Troponin I, ng/L	3.0 (0-6.0)	10.0 (6.0-22.5)	<0.001*
D-dimer, mg/L	0.50 (0.29-0.82)	0.50 (0.29-0.82)	<0.001*
CRP, g/L	12.4 (3.1-37.6)	133 (59.8-189.0)	<0.001*
IL-6, pg/mL	8.8 (4.0-24.1)	45.4 (27.2-89.0)	<0.001*
Procalcitonin, µg/L	0.05 (0.03-0.10)	0.14 (0.07-0.89)	0.001*
Ferritin, µg/L	172 (60-296)	432 (208-646)	<0.001*
Epicardial fat volume, cm3	94.3 ± 45.5	115.1 ± 44.0	0.037*

Parameters are mean ± standard deviation or median (interquartile range). ALT, alanine transaminase; AST, aspartate transaminase; CRP, C-reactive protein; IL-6, interleukin-6; LMR, lymphocyte to monocyte ratio; NLR, neutrophil to lymphocyte ratio; PLR, platelet to lymphocyte ratio; WBC, white blood cell.

Table 2. The receiver operating characteristic curves for severity in COVID-19 patients

Variables	Cut-off	AUC (95 % CI)	Sensitivity %	Specificity %	PPV %	NPV %	PLR	NLR
CRP	46.0	0.904 (0.843-0.965)	82.6	81.1	57.5	93.7	4.37	0.21
IL-6	25.65	0.857 (0.745-0.969)	83.3	80.3	55.5	94.3	4.22	0.21
Procalcitonin	0.075	0.739 (0.606-0.872)	73.9	67.1	41.4	89.09	2.24	0.39
Ferritin	202.5	0.740 (0.630-0.851)	80.0	60.9	42.5	89.3	2.04	0.33
EFV	102.1	0.654 (0.542-0.767)	69.2	60.0	37.5	84.9	1.73	0.51

AUC, Area under the curve; CRP, C-reactive protein; EFV, epicardial fat volume; IL-6, interleukin-6; NLR, negative likelihood ratio; NPV, negative predictive value; PLR, positive likelihood Ratio; PPV, positive predictive value.

Cardiac imaging / Echocardiography

OP-167

Impact of inflammation on left ventricular mass index in end stage renal disease patients

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Background and Aim: Cardiovascular diseases are the most common cause of mortality in patients on hemodialysis. Left ventricular mass is independent risk factor for cardiovascular death among end-stage renal disease patients (ESRD) undergoing hemodialysis. The aim of this study was to investigate the relationship between inflammatory markers, and left ventricular hypertrophy (LVH) in patients with ESRD.
Methods: Fifty-three patients with ESRD treated with maintenance HD in Boyabat State Hospital were consecutively enrolled between December 2019 and March 2020. Assessments of cardiac structure and function were performed by echocardiography according to American Society of Echocardiography guidelines. Echocardiographic parameters obtained by cardiologist after hemodialysis. Two-dimensionally guided M-mode echocardiograms (Philips HD11 XE ultrasound system) of the left ventricle were obtained from patients in the left decubitus position. The left ventricular mass was calculated according to the formula of Devereux and Reicheck and this was indexed for body surface area to obtain the left ventricular mass index (LVMI).
Results: A total of 53 patients with 61.09 ± 12.12 [22-86] years of age were included. Study population consisted of 32(60.4%) males. In the general population of the study, 11.3% of participants were current smokers, 37.7% were ex-smokers and 50.7% were non-smokers. The predominant etiologies of ESRD were hypertension (34%), idiopathic (26.4%) and diabetes mellitus (22.6%). The median duration of HD treatment was 48 months [0.5-288]. The mean LV mass index value was 104.66 ± 36.6 and the mean LVEF value was 59.41± 3.66. Left ventricular mass index was positive correlated with serum CRP (r=0.283, p=0.040) and proBNP levels (r=0.607, p<0.001). There was not a correlation between left ventricular mass index and lipid parameters.
Conclusions: Hypertension, hypervolemia, anemia, and age have been identified as major risk factor of LVH in ESRD patients. Left ventricular hypertrophy also can be caused by inappropriate activation of the renin-angiotensin-aldosterone system, oxidative stress, and inflammation. The C-reactive protein is a marker of systemic inflammation that has been associated with an increased risk of incident myocardial infarction and stroke. Inflammation has also been hypothesized to play a role in the development left ventricular hypertrophy.

Cardiac imaging / Echocardiography

OP-168

Evaluation of inflammation markers in mitral valve prolapse

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Background and Aim: Mitral valve prolapse (MVP) is the most common cause of mitral regurgitation in developed countries and affects approximately 2.4% of the population. Recently, there has been increased interest in MVP. It especially has serious complications including endocarditis, arrhythmias, and death. As time has progressed, the prevalence of MVP has varied in the general population. This is partially because of different definitions of MVP in recent guidelines. The current imaging definition of MVP is billowing of any portion of mitral leaflets ≥2 mm above the annular plane. The role of inflammation in the pathogenesis of MVP is still not clear. We aimed to search the predictive role of inflammatory markers such as monocyte-to-HDL ratio (MHR), lymphocyte-to-monocyte ratio (LMR), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-neutrophil ratio (PLR) on MVP patients.
Methods: In this cross-sectional study, we included 461 patients with MVP and 459 normal echocardiographic patients, matched with gender and age. İnflammatory markers and all variables were compared between the two groups.
Results: There were no statistically significant differences in age, sex, or body mass index between the two groups. MVP group had significantly more serum TC and LDL-C than the control group, whereas HDL-C and TG levels were lower (p<0.05). Furthermore, neutrophil count (5521±2.3, 4708±1.5 p<0.001), NLR (4.19±7.40,

2.13±1.25 p<0.001) MHR (15.8±0.01, 14.4±0.01 p=0.003), PLR (150.36±119.14, 117.66±57.24 p<0.001) and C-reactive protein (0.71±0.50, 0.67±0.33 p<0.001) were significantly higher in the MVP group than the control group, respectively. In logistic regression analysis; NLR [OR: 1,058 (1.047-1.072); p<0.001], LMR [OR: 1.560 (1.211-2.522); p=0.027], and PLR [OR: 1.015 (1.012-1.019); p=0.003] were found to be independent predictors for MVP presence.

Conclusions: The main goal of the current study was to determine the association between MVP and new inflammatory markers. From the results of our study, we found that MHR, NLR and PLR were significantly higher in MVP patients compared to the control group. To the best of our knowledge, these findings are the first data for these markers in MVP and represent compelling results. These parameters may be used as a simple, low-cost, reproducible tool to detect inflammation and oxidation level in MVP patients. Nonetheless, we need further prospective, randomized, large-scale studies involving other inflammatory biomarkers.

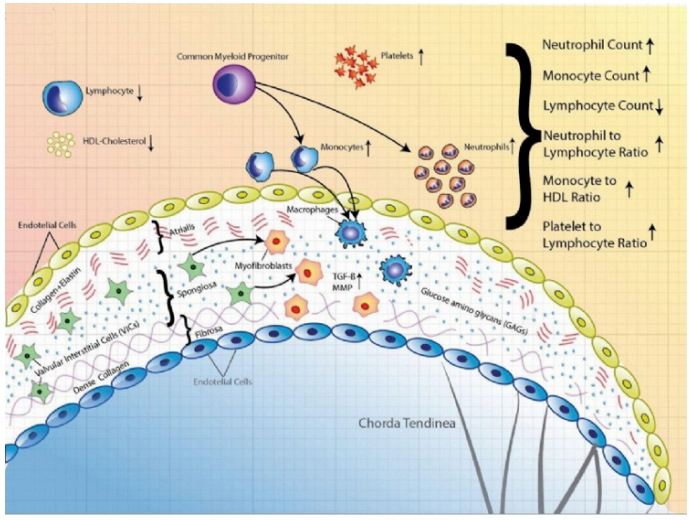

Figure 1. According to our results pathophysiology and cellular changes of mitral valve prolapse.

Table 1. Baseline demographic and echocardiographic measurements of study groups

Parameters	MVP (n=461)	Control (n=459)	P-Value
Age, years	33.75(±9,97)	33.73(±12.10)	0.980
Male, n (%)	162 (32.5%)	161 (32.3%)	0.945
Hypertension, n (%)	39 (4.2%)	44 (4.5%)	0.567
Hyperlipidaemia, n (%)	31 (3.4%)	32 (3.5%)	0.897
LVEF (%)	62.39±5.27%	61.74±6.65%	0.100
LVDD, mm	45.78±4.69	45.81±4.37	0.916
LVSD, mm	29.92±0.23	29.58±0.18	0.251
LA, mm	35.21±5.69	34.37±5.42	0.021*
Degree of mitral regurgitation 0/1/2/3/4, n	169/186/75/16/15	432/16/11/0/0	<0.001*
Fasting blood glucose, mg/dl	90.9 (81-100)	92.5 (83-103)	0.124#
Neutrophil count, µl	4960 (3900-6780)	4200 (3800-5600)	<0.001*#
Lymphocyte count, µl	2073±0.8	2385±0.7	0.01*
Monocyte count, µl	600 (500-800)	510 (400-700)	<0.001*#
Platelet, 10^3/mm^3	249 (186-283)	231 (215-289)	<0.001*#
HDL-cholesterol, mg/dl	41 (37-45)	43 (38-48)	<0.001*#
LDL-cholesterol, mg/dl	115.6±33.1	106.7±29.4	0.016

** p-value <0.05 was considered statistically significant.*

Table 2. Comparing inflamatuar markers between MVP and control groups

MHR	14.9 (11.9-18.6)	12.2 (9.4-17.3)	0.003*#
LMR	3.75 (2.75-5.09)	4.06 (3.12-4.83)	0.016*#
NLR	2.488 (1.72-4.51)	1.857 (1.49-2.38)	<0.001*#
PLR	122.4 (85-171)	104.4 (85-130)	<0.001*#
C-reactive protein, mg/dl	0.71±0.50	0.67±0.33	<0.001*

**p-value <0.05 was considered statistically significant, #: Mann Whitney U test, MHR: Monocyte to HDL ratio, NLR: Neutrophil to Lymphocyte ratio, LMR: Lymphocyte to Monocyte to ratio, PLR: Platelet to Lymphocyte ratio.*

Cardiac imaging / Echocardiography

OP-169

Early atherosclerotic markers detected by echocardiography in patients who underwent renal transplantation at least five years before admission

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Background and Aim: Atherosclerosis is a leading cause of all cause mortality.Chronic renal disease is one of the most important risk factors of ats.The impact of renal tx on the ats is not well defined yet. Renal transplantation is expected to reduce inflammation in arterial wall in such pts; this may take a long period.
Methods: We aimed to investigate early atherosclerosis indicators by 2D echocardiography in patiens who underwent renal transplantation for at least 5 yrs before evaluation. 53 pts (33 male, 20 female) were enrolled. 79.2% (n=42) of the group was underwent living kidney transplant and 20.8% was underwent cadaveric kidney transplant,respectively. pts with history of coronary disease, ischemic stroke,peripheral arterial disease and smoking were excluded. Patient basal characteristic were recorded.
Results: Patients with cadaveric kidney transplantation had tend to have higher carotid intima media thickness and end-diastolic epicardial fat tissue thickness. Left ventricle mass and mass index were also higher in cadaveric renal transplant group.CIMT was highly correlated with end-systolic epicardial fat tissue thickness. (p=0.002) Left ventricle mass index is correlated with end-systolic fat tissue thickness (p=0.007). The pre-atherosclerotic markers are statistically higher in cadaveric renal transplant patients. Cadaveric transplant group had longer period of chronic renal disease and received longer duration of dialysis treatment; in addition had higher number of comorbidities.
Conclusions: Thus, cadaveric renal tx pts should be closely monitored in terms of atherosclerosis despite being asymptomatic. Risk factors should be restricted in such patient group.

Cardiovascular nursing / Technician

OP-170

Determination of frailty in elderly individuals with heart failure

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Background and Aim: This research was carried out to determine the frailty of elderly individuals with heart failure.
Methods: The research was carried out in descriptive type. 151 patients aged 65 and over, who were hospitalized in the cardiology services of an education and research hospital in Istanbul, were diagnosed with heart failure. In the study, patient diagnostic form, Edmonton Frail Scale, Mini Nutritional Assessment (MNA) Questionnaire- Long Version and Health Perception Scale were used as data collection tools. The statistics of the study were done with Windows IBM SPSS 21.0 package program. Statistical analyzes were used for number, percentage, arithmetic mean, t-test, Mann-Whitney U test, Pearson and Spearman's correlation analysis. The results were evaluated in the 95% confidence interval and the significance level was p<0.05.
Results: It was found that 58.3% of the individuals participating in the study were male, 69.5% were married, 86.8% were primary school graduates, mean age was 71.6±6.38, and body mass index averages were 28.07±4.78. According to the Edmonton Frail Scale mean scores (9.63±2.99), individuals were found to be at the middle frail level. It was found that the mean scores of frailty were significantly higher for women, single people, those without social security, non-smokers and those who felt sad for two weeks. According to MNA mean scores (19.25±4.38), individuals were found to be at risk in terms of malnutrition. A statistically significant difference was found between patients' risk of malnutrition and their frailty. It was determined that as the risk of malnutrition increased, the severity of frailty also increased. It was determined that the total point average of the health perception scale (33.56±7.16) was at a medium level. There was no significant relationship between individuals' perception of health and malnutrition.
Conclusions: It was determined that the frailty level of elderly individuals with heart failure was at a medium level, they were at risk for malnutrition, and their health perception was at a moderate level. It was found that female gender, single, without social security, no smoking, feeling sad for two weeks and those at risk in terms of nutritional status increases the frailty of individuals.

Congenital heart disease

OP-171

Evaluation of atrial electromechanical delay in patients undergoing transcatheter atrial septal defect closure

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Background and Aim: Percutaneous closure is the treatment of choice when feasible morphology of atrial septum is present. Atrial electromechanical delay (AEMD) was prolonged in patients with atrial septal defect. Higher electromechanical delay was associated with paroxysmal supraventricular tachycardia in ASD patients. We aim to compare pre and post closure AEMD in secundum type atrial septal defect patients.
Methods: After exclusion criteria 34 patients who underwent percutaneous atrial septal defect closure were prospectively enrolled in this study. Echocardiographic evaluation of atrial conduction times and clinical assessment were performed before intervention and at 6 months follow up. To assess atrial electromechanical coupling (PA), the time intervals from the onset of P wave on ECG to the beginning of late diastolic wave (A') at the septal (PA septal) and lateral (PA lateral) mitral annulus and lateral tricuspid annulus (PA tricus-

pid) were measured on Tissue Doppler Echocardiography. The differences between PA septal-PA lateral, PA septal-PA tricuspid, and PA lateral-PA tricuspid were defined as left intra-atrial, right intra-atrial, and interatrial EMD, respectively.
Results: Percutaneous closure was performed successfully in all patients. Echocardiographic parameters are shown in Table 1. All patients had decreased right heart dimensions and improved functional capacity. Tricuspid Annular Plane Systolic Excursion (TAPSE) was increased after closure. Atrial conduction times were decreased at 6 months follow up. Pre-procedure and follow up measurements were 12.8±4.7 msn vs 11.5±3.3 msn (p 0.07) for left-intra EMD; 10.6±4.7 msn vs 8.9±3.4 msn (p=0.02) for right-intra EMD; 22.4±7.6 msn vs 20.6±5.7 (p=0.04) for interatrial EMD respectively.
Conclusions: Transcatheter closure of ASD was associated with decreased intra and interatrial conduction times so shortened atrial conduction times may have positive effect on atrial arrthymia burden in ASD closure patients.

Table 1. Echocardiographic parameters

	Pre procedure (n=34)	Follow up (n=34)	p
RV End Diastolic (mm)	46.6±4.7	38.4±3.6	0.01
RA-Mediolateral Diameter (mm)	43.6±4.4	37.7±4.5	0.01
RA-Apicobasal Diameter (mm)	53.4±5.4	44.7±5.1	0.01
TAPSE (mm)	25.5±3.5	29.2±2.6	0.01
EMD Lateral (msn)	77.9±9.1	65.5±8.9	0.01
EMD Septal (msn)	65.1±9.1	53.9±8.6	0.01
EMD Tricuspid (msn)	54.7±9.5	44.9±8.5	0.01
Left Intra-atrial EMD(msn)	12.8±4.7	11.5±3.3	0.07
Right Intra-atrial EMD(msn)	10.6±4.7	8.9±3.4	0.02
Interatrial EMD (msn)	22.4±7.6	20.6±5.7	0.04

Epworth Uykululuk Ölçeği: Gündüz uyku halini göstermekte kullanılan bir testtir. Toplam 8 sorudan oluşur. Her soru hastanın kendisi tarafından 0-3 puan verilecek şekilde doldurulur. Bu ankette hastanın aşırı yorgun olmadığı sıradan bir günde, belli durumlarda uykuya dalma olasılığı sorgulanır. Tüm sorularda puanlama yöntemi aynı olup, uykuya dalma olasılığı hiç yoksa 0, uykuya dalması düşük olasılıklı ise 1, orta olasılıklı ise 2 ve yüksek olasılıklı ise 3 puan alır. Toplam puan 10 ve üzerinde ise gündüz aşırı uyku halinin varlığına işaret eder

Sorular

Aşırı yorgun olmadığınız bir günde aşağıdaki durumlarda uykuya dalma olasılığınız nedir?

1. Oturur durumda gazete veya kitap okurken
2. Televizyon seyrederken
3. Pasif olarak toplum içinde otururken (tiyatro, toplantı,...)
4. Aralıksız 1 saatlik araç yolculuğu yaparken
5. Öğleden sonra uzanınca
6. Alkolsüz bir öğle yemeğinden sonra otururken
7. Birisi ile konuşurken
8. Araç kullanırken birkaç dakika trafik durduğu

Figure 2. Epworth Sleepiness Scale.

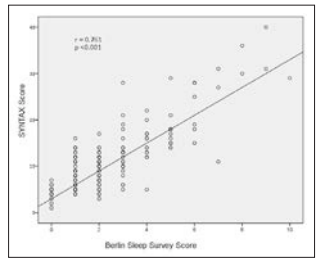

Figure 3. Berlin Sleep Questionnaire and SYNTAX Score correlation analysis.

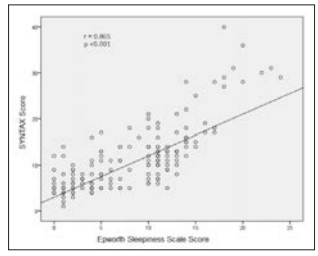

Figure 4. Epworth Sleepiness Scale and SYNTAX Score correlation analysis.

Congenital heart disease

OP-172

Acute coronary syndrome and obstructive sleep apnea syndrome

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Background and Aim: Obstructive sleep apnea syndrome (OSAS) is closely associated with cardiovascular diseases. Evaluation of simplified OSAS diagnostic methodologies is still new and has not been deeply investigated in the field of cardiology. In this study, we aimed to evaluate the relationship between OSAS and the severity of coronary artery disease in our patients with acute coronary syndrome (ACS), using the SYNTAX score, the Berlin Sleep Questionnaire (BUA) and the Epworth Sleepiness Scale (EUÖ).
Methods: 200 patients with acute coronary syndrome who underwent coronary angiography were enrolled in the study. SYNTAX scores of the patients were calculated. Patients were administered BUA and EUÖ before discharge. The scales were compared between the two groups and with SYNTAX scores of patients.
Results: In NSTEMI patients, the rate of high-risk patients in the BUA and ACL was statistically significant compared to the STEMI group (p<0.001, p=0.023). The total score of the BUA and the total score in the EUÖ was statistically significant in NSTEMI patients compared to the STEMI group (p<0.001). Pearson correlation analysis revealed a significant positive correlation between the SYNTAX score and the total score in the BUA and between the SYNTAX score and the EUÖ (r=0.865, p<0.001 ve r=0.761, p<0.001)
Conclusions: In this study, the relationship between OSAS and ACS was evaluated. The results of the BUA and EUÖ scale were higher than those of other countries and a positive correlation was found between SYNTAX score and OSAS risk.

Figure 1. Berlin Survey Questions and Evaluation.

Table 1. Comparison of the basic demographic features of the groups with laboratory parameters

Variables	NSTEMI (n=80)	STEMI (n=80)	p value
Age	60.1 ± 4.5	59.5 ± 4.4	0.733
BMI, kg/m2	29.2 ± 12.0	28.5 ± 12.0	0.355
Waist circumference, cm	90.31 ± 8.63	87.13 ± 5.24	0.401
Neck circumference, cm	37.36 ± 3.08	35.91 ± 3.20	0.658
Male gender, n(%)	50 (62.5)	54 (67.5)	0.507
Diabetes mellitus, n(%)	31 (38.8)	22 (27.5)	0.131
Hypertension, n(%)	37 (46.3)	27 (33.8)	0.107
Hypelipidemia, n(%)	30 (37.5)	25 (31.3)	0.405
Smoking, n(%)	34 (42.5)	38 (47.5)	0.525
Family history of heart disease	34 (42.5)	24 (30.0)	0.100
EUÖ total score	10.9 ± 5.3	6.2 ± 4.6	<0.001
EUÖ high risk patient rate	38 (47.5)	24 (33.8)	0.023
BUA total score	3.1 ± 1.6	1.8 ± 1.3	<0.001
BUA high risk patient rate	37 (46.3)	22 (27.5)	<0.001
SYNTAX score	14.7 ± 7.8	7.7 ± 4.2	<0.001
Ejection Fraction	52 ± 6.8	43 ± 10.8	<0.001

NSTEMI, non-ST segment elevation myocardial infarction; STEMI, ST segment elevation myocardial infarction; BUA, Berlin Sleep Questionnaire; EUÖ, Epworth Sleepiness Scale; BMI, body mass index. Data are given as mean ± standard deviation or percentage [n (%)].

Congenital heart disease

OP-173

Impact of shunt ratio on ventricular repolarization in patients with ASD

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Background and Aim: Ventricular arrhythmias are not rare in patients with atrial septal defect (ASD). Deteriorations in cardiac hemodynamics may lead to enlargement and fibrosis in heart. Studies indicate that prolongation of the interval between the peak and end of the T wave (Tpeak to Tend, Tp-e) on the 12-lead ECG, is a marker of ventricular arrhythmogenesis. The aim of this study was to assess if there is an impact of shunt ratio on ventricular repolarization in patients with ASD by using Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio.
Methods: Patient records of Samsun Training and Research Hospital were retrospectively analyzed. Electrocardiograms of 133 patients, who were diagnosed as ASD between January 2016 and December 2019 were obtained and scanned. ECG intervals were measured. Shunt ratios, right ventricle diameters and volumes were also acquired.
Results: Both groups' baseline characteristics were similar. Right ventricle dimensions and systolic pulmonary artery pressure were higher in large ASD group. Furthermore, ASD patients with Qp/Qs ratio ≥2 had significantly higher ECG measurements than controls, Tp-e: 103.0±22.1vs 76.2±10.2; Tp-e/QT: 0,25 vs 0,21; Tp-e/QTc:0,22 vs, 17; for all p<0,001). Of all ECG parameters; Tp-e (r=0.631, p<0.001), Tp-e/QT (r=0.531, p<0.001) and Tp-e/QTc (r=0.614, p<0.001) had moderate correlation with shunt ratio.
Conclusions: T wave peak to end interval is a measure of transmural dispersion of repolarization and accepted as a surrogate for increased ventricular arrhythmogenesis risk. Our findings show that ASD patients whose shunt ratio are ≥2 show increased risk for arrhythmias.

Table 1. Echocardiographic and electrocardiographic parameters between both groups

Parameters	Shunt ratio ≥2 (n=68)	Shunt ratio <2 (n=65)	p value
Mean shunt ratio	2.2±0.1	1.3±0.2	<0.001
RV1 (mm)	35.5±2.4	32.4±1.7	<0.001
RV2 (mm)	28.0±2.2	24.7±1.4	<0.001
RV3 (mm)	66.5± 2.8	64.0± 1.9	<0.001
RVEDV (ml)	185.4±13.9	147.62±7.0	<0.001
RVESV (ml)	88.46±7.5	51.8±5.4	<0.001
SPAP (mmHg)	35.5±7.6	19.3± 5.2	<0.001
TAPSE (mm)	11.2± 2.9	16.2± 1.6	<0.001
LVEF (%)	60.9±3.7	61.0±4.0	0.859
QT (msec)	409.9±62.7	361.4±35.7	<0.001
QTc (msec)	459.7±44.0	428.2±43.1	<0.001
Tp-e (msec)	103.0±22.1	76.2±10.2	<0.001
Tp-e/QT ratio	0,25±0.0	0.21±0.0	<0.001
Tp-e/QTc ratio	0.22±0.0	0.17±0.0	<0.001

LVEF: left ventricle ejection fraction, ml: milliliter, mm: millimeter, msec: millisecond, QTc: corrected QT, RV1: right ventricle end-diastolic annulus line diameter, RV2: right ventricle end-diastolic mid-line diameter, RV3: right ventricleend-diastolic base-to-apex distance, RVEDV: right ventricle end diastolic volume, RVESV: right ventricle end systoliv volume, SPAP: systolic pulmonary artery pressure, TAPSE: tricuspid annuler plane systolic excursion, Tp-e: T wave peak to end interval. Data are presented as means ± SD.

Table 2. Correlations between electrocardiographic parameters and shunt ratio

Variables	Shunt ratio	
	r	p
QT(msec)	0.457	<0.001
QTc(msec)	0.375	<0.001
Tpe(msec)	0.631	<0.001
Tpe/QT	0.531	<0.001
Tpe/QTc	0.614	<0.001

Tp-e: T wave peak to end interval, msec: millisecond, QTc: corrected QT

Table 3. Correlations between ehocardiographic parameters and shunt ratio

Variables	Shunt ratio	
	r	p
RV1	0.553	<0.001
RV2	0.569	<0.001
RV3	0.393	<0.001
RVEDV	0.783	<0.001
RVESV	0.833	<0.001
LVEF	0.008	0.926
SPAP	0.677	<0.001
TAPSE	-0.638	<0.001

LVEF: left ventricle ejection fraction, ml: milliliter, mm: millimeter, msec: millisecond, RV1: right ventricle end-diastolic annulus line diameter, RV2: right ventricle end-diastolic mid-line diameter, RV3: right ventricleend-diastolic base-to-apex distance, RVEDV: right ventricle end diastolic volume, RVESV: right ventricle end systoliv volume, SPAP: systolic pulmonary artery pressure, TAPSE: tricuspid annuler plane systolic excursion.

Coronary artery disease / Acute coronary syndrome

OP-176

Prevalence, clinical and angiographic characteristic, and outcomes of coronary artery embolism-associated acute myocardial infarctions

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Background and Aim: The updated universal definition of myocardial infarction (MI) has classified the coronary embolism (CE) associated MI as Type 2, which falls into this category along with atherosclerosis, coronary arterial spasm, anemia, arrhythmias, and hyper/hypotension. The exact prevalence of CE is not known, but previous autopsy studies reported that up to 13% of MI cases had coronary artery embolic infarcts. This study aimed to determine the prevalence, clinical and angiographic characteristics, and outcomes of CE-associated MIs.

Methods: A total of 19393 angiography reports were retrospectively evaluated, and 4655 patients with acute MI (2363 STEMI and 2292 NSTEMI) were identified. Diagnosis of CE was done using the diagnostic criteria of National Cerebral and Cardiovascular Center that proposed by Shibata et al. The demographic, clinical, treatment, and long-term follow-up data were analyzed.

Results: Mean age was 70.1±13.6 years, and 70% (n=21) were females. The total number of CE events in 30 patients was 32, which corresponds to a prevalence of 0.7% for CE in acute MI cases. The prevalence in all STEMI and NSTEMI cases were 0.8% and 0.5%, respectively. At admission, 96.7% of cases had chest pain, and 43.4% had dyspnea. Most frequent cardiovascular risk factors in the medical history of patients were hypertension in 16 (53.3%), hyperlipidemia in 7 (23.3%), and diabetes mellitus in 6 patients (20%). Six patients had history of extra-coronary embolism, 3 (10%) in brain, and 3 (10%) in lower extremities. Prosthetic valve thrombus was present in 3 (10%), and rheumatic valve disease in 3 cases (10%). Most common underlying disease was atrial fibrillation in 26 patients (86.7%), which distributed as 18 (60%) permanent, 6 (20%) paroxysmal, 1 (3.3%) persistent and 1 (3.3%) new-onset atrial fibrillation, of which 22 (77.3%) had non-valvular and 4 (13.3%) had valvular atrial fibrillation. Angiography revealed distal coronary occlusion in 20 patients (66.7%). Affected coronaries were RCA in 12 (40%), LAD in 14 (46.7%), and Cx in 6 cases (20%). During coronary interventions, glycoprotein IIb/IIIa inhibitors (9 patients; 30%) were used more than the stent (4 cases, 13.3%). Thrombo-aspiration alone was sufficient to restore a TIMI flow grade ≥2 in 3 patients (10%) (Table 1). The in-hospital mortality was 3.3% (n=1), and during the median follow-up of 46.5 months 10 patients died, but no recurrent myocardial infarction or thromboembolism was developed. The overall mortality was found to be 33.3% (Figure 1).

Conclusions: The prevalence of CE was lower than the previous reports in the literature. Most common underlying cause was atrial fibrillation. Administration of a combination of appropriate modalities including angioplasty and/or stent, thrombectomy, thrombolytics, and Gp-IIb/IIIa can achieve favorable outcomes in the management of these patients. Nevertheless, the mortality is still high in this subcategory of cases.

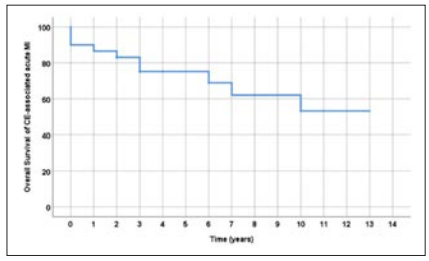

Figure 1. Kaplan-Meier survival curve of CE-associated acute MI patients.

Table 1. Demographic, clinical, angiographic, and treatment characteristics of patients

	n (%) / Mean±SD
Age, years	70±13.7
Sex, female	21 (70)
Body mass index (kg/m2)	27.2±4.3
Chief complaint	
Chest pain	29 (96.7)
Dyspnea	13 (43.3)
Weakness	1 (3.3)
Cardiovascular risk factors	
Hypertension	16 (53.3)
Dyslipidemia	7 (23.3)
Diabetes mellitus	6 (20)
Smoker	4 (13.3)
Angiography	
Left anterior descending	14 (46.7)
Left circumflex	6 (20)
Right coronary artery	11 (36.7)
Posterolateral branch	5 (16.7)
Posterior descending artery	1 (3.3)
Treatment	
Heparin	30 (100)
Glycoprotein IIb/IIIa blocker	10 (33.3)
Manual thrombectomy	7 (23.3)
Reperfusion strategies used	
Antithrombotic drugs alone	9 (30)
Thrombolytic treatment	7 (23.3)
Primary angioplasty	5 (16.7)
Stent implantation	4 (13.3)
Manual thrombectomy without angioplasty	3 (10)
Guidewire without angioplasty or manual thrombectomy	2 (6.7)

Table 1. Continues

LVEF (%)	47.7±7.8
TIMI flow after reperfusion	
0-I	5 (16.7)
II	6 (20)
III	19 (63.3)
Atrial fibrillation	26 (86.7)
Sinus rhythm	4 (13.3)
Non-valvuler	22 (73.3)
Permanent	14 (46.7)
Paroxismal	6 (20)
persistant	1 (3.3)
new-onset	1 (3.3)
Valvuler	4 (13.3)
Permanent	4 (13.3)
Prosthetic valve trombi	3 (10)
Rheumatic valve disease	4 (13.3)
Mitral stenosis	4 (13.3)
Hypertrophic cardiomyopathy	1 (3.3)
Cancer	1 (3.3)
RCA	12 (40)
RCA distal	6 (20)
Posterior descending	1 (3.3)
Posterolateral from RCA	5 (16.7)
LAD	14 (46.7)
LAD mid	2 (6.7)
LAD apical	11 (36.7)
First diagonal	1 (3.3)
Cx	6 (20)
Distal circumflex	4 (13.3)
Left posterolateral	2 (6.7)

Coronary artery disease / Acute coronary syndrome

OP-177

Myocardial infarction with non-obstructive coronary arteries (MINOCA) in single center experience: Clinical features, prognosis

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Background and Aim: Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a working diagnosis with different etiology and characterized by clinical evidence of myocardial infarction with normal or near-normal coronary arteries on angiography. The awareness of clinicians in the MINOCA group has been increasing in recent years.The aim of this study is to analyze the clinical profile of MINOCA patients compared to those with myocardial infarction with obstructive lesions and evaluate MINOCA patients prognosis

Methods: A total of 1421 consecutive patients with acute myocardial infarction admitted to the Dokuz Eylul University hospital between January 2016 and March 2019 were retrospectively screened. Patients with prior history of coronary artery disease and those with procedure-related acute myocardial infarction were

excluded. Patients were classified into two groups: MINOCA, comprising patients with no significant lesions on angiography, and MI-CAD, comprising patients with lesions of the coronary artery tree. We used the 2016 ESC Working Group position paper on MINOCA. A total 130 patients with MINOCA and 210 patients with MI-CAD were included in the study. Clinical, demographic, laboratory, echocardiography, angiographic and prognostic features were studied at both groups. Duration of median follow-up was of 22.5 months.
Results: The prevalence of MINOCA was 9.1% in our tertiary center. Patients with MINOCA had more frequently women (especially premenopausal women) and had younger age (Table 1). NSTEMI was more frequent than STEMI in MINOCA group. Compared with patients with MI-CAD, the prevalence of traditional CAD risk factors was lower in MINOCA patients. Interestingly, patients with MINOCA were more likely to have a history of upper-respiratory-tract infections (URIs) and antidepressant drugs use compared to MI-CAD at the admission. The blood cholesterol and WBC level were significantly lower in the MINOCA group. Left ventricular ejection fraction was higher in MINOCA group. In-hospital mortality was significantly lower in patients with MINOCA than MI-CAD (0% vs 5,7%). Mortality of MINOCA patients at follow up was 7%, and non-mortality adverse events were 9.7%. Duration of hospitalization was found to be shorter in MINOCA group's.
Conclusions: Patients with MINOCA constitute a population that differs from classical MI profile. Compared with MI-CAD, MINOCA was accompanied by fewer traditional risk factors of CAD. Remarkably premenopausal status, history of URI and antidepressant use were found independent predictors for MINOCA in this study. This finding might be hypothesis generating.

Table 1. Univariate and multivariate parameter analysis of MINOCA vs MI-CAD groups

	Univariate Analysis			Multivariate Analysis	
	MINOCA	MI-CAD	P value	Odds Ratio	IC %95
Gender(Female)	44,2%	24,2%	<0,001	2,37	1,4-3,75
Premenapausal Women	24,6%	9,6%	<0,001	3,06	1.1-9,2
NSTEMİ	79,8%	44,3%	<0,001	4,98	2,9-8,3
Hyperlipidemia	27,8%	55.5%	<0,001	0,33	0,23-0,57
Diabetes	16,2%	%34,3	0,001	0,26	0,44-0,75
Anti depressant Drug Use	20.9%	9.1%	0.003	2.63	1.3-5.52
History of Upper Tract İnfection	16.7%	3.3%	<0,001	5.51	2.3-13.55
LDL (mg/dl)	111.88±44.82	127±38.5	<0,001		
LVEF%	54.9 ± 9.9	47.41 ± 9.5	<0,001		
In hospital mortality	0%	5,7%	0,05		

Coronary artery disease / Acute coronary syndrome

OP-180

The relationship between treatment strategy and mortality in elderly patients with non-ST-segment elevation myocardial infarction

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Background and Aim: It is known that non-ST-segment elevation myocardial infarction (NSTEMI) is one of the leading causes of hospitalization and mortality in elderly patients. Although the guidelines have recommended that elderly patients with NSTEMI should undergo an examination for invasive revascularization, the number of elderly patients undergoing invasive treatment in clinical practice is relatively low. Therefore, the fact that older patients represent a higher risk subgroup and receive less optimal treatment than younger patients increased the importance of examining the effect of treatment strategies in elderly NSTEMI patients. The present study aimed to examine the effect of PCI treatment on one-year mortality in NSTEMI patients over the age of 65.
Methods: The study which was an observational, clinical, and prospective The study was first planned as a pragmatic clinical study embedded in routine clinical practice at the hospital. The sample of the study consisted of 324 patients with NSTEMI aged 65 years or older who underwent coronary angiography and treated with either a conservative strategy or PCI. Demographic data, electrocardiography, echocardiography results, the history of the medical disease, and laboratory testing values were carried out according to the routine practice. For all patients was calculated with the GRACE, SYNTAX, and Gensini risk score. Participants included in the study were followed for ≥14 months after NSTEMI.
Results: 208 patients (64.19%) were treated with PCI and 116 patients (35.81%) of the participant with conservative methods. The mean age of the participants was 75.41±6.65 years. There was a statistically significant difference between the conservative strategy and PCI group with respect to age, gender, history of coronary artery disease, stroke, and PCI (p<0.05, Table 1) A statistically significant difference was found between these two groups: hemoglobin, using ASA, clopidogrel, warfarin, statin at discharge, and GRACE scores (p<0.05, Table 2) The independent predictors of one-year mortality were revealed the treatment strategy, Killip class ≥2, LVEF, and renal failure with an adjusted hazard ratio of 1.965 (95% CI:1.037 to 3.720), 2.392 (95% CI:1.268 to 4.511), 2.637 (95% CI:1.373 to 5.065), and 3.471 (95% CI:1.853 to 6.502) respectively (Table 3). The Kaplan Meier analysis revealed signiŞcant differences for all-cause mortality for the treatment strategy at one-year (Figure 1).
Conclusions: The effects of PCI and the conservative treatment strategies on mortality rate in NSTEMI patients over the age of 65 were compared in this study. The most important result of the study was that PCI was found to be effective in year-month all-cause mortality. In addition, it was noted that renal failure, LVEF, and Killip class had an effect on mortality. Considering the results of our study, we believe that the use of invasive management in elderly patients with NSTEMI will reduce the mortality rate.

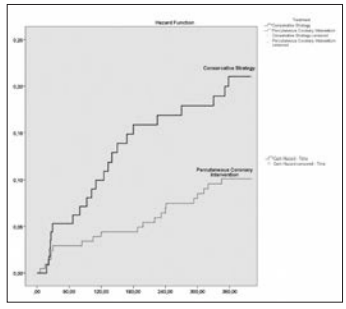

Figure 1. The cumulative risk of overall death in patients stratified according to conservative strategy and percutaneous coronary intervention.

Table 1. Demographic and baseline characteristics of the study population stratified according to conservative strategy and percutaneous coronary intervention

Variable	Conservative Strategy (n=116)	Percutaneous Coronary Intervention (N=208)	t/X2 Value	P Value
Age, years (mean±SD)	76.69±7.10	74.70±6.29	2.613	0.009**
Males, n(%)	57 (30.8)	128 (69.2)	4.675	0.031*
History, n(%)				
Diabetes	34 (29.3)	58 (27.9)	0.074	0.785
Dyslipidemia	35 (30.2)	81 (38.9)	2.492	0.114
Hypertension	71 (61.2)	143 (68.8)	1.890	0.169
Coronary artery disease	36 (31.0)	97 (46.6)	7.486	0.006**
Peripheral artery disease	4 (3.4)	12 (5.8)	0.855	0.355
Chronic renal failure	33 (28.4)	52 (25.0)	0.458	0.499
COPD	15 (12.9)	18 (8.7)	1.489	0.222
Stroke	8 (6.9)	2 (1.0)	8.770	0.003**
PCI	8 (6.9)	46 (22.1)	12.419	<0.001**
Current Smoking, n(%)	23 (19.8)	20 (14.4)	1.590	0.207

COPD: chronic obstructive pulmonary disease.

Table 2. Clinical characteristics and mortality of the study population stratified according to conservative strategy and percutaneous coronary intervention

Variable	Conservative Strategy (n=116)	Percutaneous Coronary Intervention (N=208)	t/X2 Value	P Value
Hemoglobin (mg/dl)	12.76±2.25	13.27±1.89	-2.036	0.043*
ST segment deviation (yes)	36 (31.0)	53 (25.5)	1.153	0.283
Discharge Medications n(%)				
ASA	85 (73.3)	194 (93.3)	24.890	<0.001**
Clopidogrel	70 (60.3)	199 (95.7)	65.947	<0.001**
Warfarin	19 (16.4)	17 (8.2)	5.078	0.024*
NOACs	12 (16.4)	16 (10.5)	1.629	0.202
RAS Blockers	74 (63.8)	143 (68.8)	0.827	0.363
Beta-Blockers	101 (87.1)	184 (88.5)	0.136	0.712
Statin	66 (56.9)	160 (76.9)	14.156	<0.001**
Killip class ≥2, n(%)	32 (27.6)	36 (17.3)	4.744	0.029*
SYNTAX, (mean±SD)	8.86±10.11	10.35±6.74	-1.345	0.180
GRACE Score, (mean±SD)	143.46±25.46	130.96±18.71	3.689	<0.001**
Ejection fraction (%), (mean±SD)	49.32±9.95	51.39±9.27	-1.873	0.062
Estimated GFR, (mean±SD)	57.55±19.20	59.96±10.14	-1.050	0.295
Admission Troponin, (mean±SD)	1.79±3.81	2.61±6.56	-1.376	0.170
1-Year mortality, n (%)	22 (19.0)	20 (9.6)	5.770	0.016*

ASA: acetylsalicylic acid, GFR: Glomerular filtration rate (Using the Cockroft-Gault formula); NOACs: novel oral anticoagulants, RAS Blockers: Renin-angiotensin system blockers.

Table 3. Predictors of 1-year mortality: univariable and multivariable Cox Regression Analysis

Predictor	Univariable (Hazard Ratio (95% CI); P Value)	Multivariable (Hazard Ratio (95% CI); P Value)
Treatment (Conservative Strategy)	2.094 (1.143-3.837); p=0.017	1.965(1.037-3.720); p=0.038
Admission Troponin (1-point increase)	1.035 (1.000-1.071); p=0.049	1.033(0.996-1.073); p=0.083
Killip class ≥2	3.440(1.873-6.318); p<0.001	2.392(1.268-4.511); p=0.007
Ejection fraction (%)<42	4.044(2.202-7.429); p<0.001	2.637(1.373-5.065); p=0.004
Renal Failure	4.051(2.206-7.442); p<0.001	3.471(1.853-6.502); p<0.001

Coronary artery disease / Acute coronary syndrome

OP-181

Prognostic value of pericardial effusion in STEMI patients treated with a primary percutaneous coronary or a pharmacoinvasive intervention: (From an experience of a tertiary center)

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Background and Aim: Pericardial effusion (PE) is commonly seen a complication after ST-segment elevation myocardial infarction (STEMI). The presence of PE in STEMI patients has been associated with mortality. However, most previous studies were performed in patients with treated with fibrinolysis. There are limited data impact of PE on long-term mortality in STEMI patients. In this retrospective analysis, we aimed to investigate the long-term mortality of patients with PE who were treated with a pharmacoinvasive or a primary percutaneous intervention (PCI) strategy in an experience of a tertiary center.

Methods: 860 patients with STEMI were enrolled in this study. The patients were divided into 2 groups; patients with PE (n=51) or patients without PE (n=809). The primary end point of the study was the occurrence of all-cause mortality. The secondary end points included recurrent MI, target vessel revascularization, heart failure admission, major bleeding, and stroke at follow-up.

Results: Median follow-up was 6.7 (IQR 4.5 to 8.1) years. Pericardial effusion was seen in 51 patients (5.9%). There was no difference between patients who were treated with a pharmacoinvasive and those underwent primary PCI (p=0.514). Long-term mortality rate was higher in patients with PE compared with those without PE (41% vs 27%, p=0.025). However, presence of PE was not associated with long-term mortality in multivariate analysis (HR: 1.262 95%CI: 0.776-2.054, p=0.348).

Conclusions: The presence of PE was not associated with long-term mortality in acute STEMI treated with a primary percutaneous coronary intervention or a a pharmacoinvasive intervention was not independently associated with long-term mortality.

Table 1. Clinical and labratory findings

Variables	Patients without PE (n =809)	Patients with PE (n = 51)	p-value
Age, years	60.3 ± 1	62.7 ± 12.5	0.179
Men n (%)	615 (76)	38 (75)	0.875
Smoking n (%)	328 (41)	19 (38)	0.727
Hypertension n (%)	331(41)	22 (44)	0.662
Diabetes mellitus n (%)	183(23)	13 (26)	0.577
History of CAD n (%)	139 (17)	11 (22)	0.381
Dyslipidemia n (%)	152 (19)	8 (16)	0.626
Stroke/TIA n (%)	25 (3)	1 (2)	0.663
Killip class ≥ 2	61 (8)	10 (20)	0.002
Multivessel disease n (%)	288 (36)	23 (45)	0.177
Infarct related artery n (%)			0.917
LAD	370 (46)	27 (53)	
CX	102 (13)	6 (12)	
RCA	306 (38)	17 (33)	
Other	31 (4)	1 (2	
Laboratory findings			
Hgb (mg/dl)	13.9 ± 1.9	13.5 ± 2.1	0.239
WBC	12.0 ± 4.3	13.2 ± 4.6	0.065
e GFR	86.9 ± 27.4	85.9 ± 29.1	0.800
LVEF (%)	45.2 ± 9.6	40.4 ± 9.2	< 0.001
Discharge treatment			
Beta-blocker n (%)	690 (85)	43 (86)	0.875
ACE-I/ARB n (%)	679 (85)	36 (72)	0.021
Statin n (%)	674 (85)	42 (84)	0.930
Treatment			0.514
Rescue PCI after fibrinolysis n (%)	46 (6)	2 (4)	
Scheduled PCI after fibrinolysis n(%)	194 (24)	9 (18)	
Primary PCI n (%)	570 (70)	39 (78)	
Long -term mortality n (%)	216 (27)	21 (41)	0.025
In-hospital mortality n (%)	23 (3)	5 (10)	0.006
Myocardial re-infarction n (%)	100 (12)	4 (8)	0.360
Target-vessel revascularization n (%)	109 (14)	6 (12)	0.769
Heart failure admission n(%)	44 (5)	5 (10)	0.176
Stroke at follow-up n (%)	32 (4)	0 (0)	0.152

Coronary artery disease / Acute coronary syndrome

OP-182

Increased fructose consumption may be associated with slow coronary flow

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Background and Aim: Slow coronary flow (SCF) is an important coronary angiographic phenomenon characterized by delayed progression of angiographic contrast medium in the coronary arteries in the absence of obstructive coronary artery disease (CAD). The pathophysiological mechanisms underlying primary SCF have not been clearly understood so far. Potential underlying mechanisms such as microvascular dysfunction, endothelial dysfunction, vasomotor dysfunction, small vessel disease, atherosclerosis, inflammation, oxidative stress, and increased platelet aggregation have been evaluated High fructose intake can lead to the atherosclerotic process by increasing the number of low-density lipoprotein (LDL) particles, reducing the amount of particles with atherogenic effects, increasing the expression of adhesion molecules in endothelial cells, and triggering thrombosis pathophysiology. Also, there are many studies showing that increased Fructose consumption is associated with oxidative stress and inflammation, which play a role in the pathophysiology of SCF. In light of these findings, this study aims to evaluate the relationship between fructose consumption and SCF.

Methods: In this study, 496 patients who underwent coronary angiography due to clinical suspicion demonstrated by exercise stress test or myocardial perfusion scintigraphy or myocardial ischemia between December 2018 and April 2019 at our Hospital were evaluated. Two groups were formed. Forty-five patients with normal coronary artery anatomy and SCF were selected as the patient group (SCF group), and 50 patients with normal coronary flow patterns (NCF) were accepted as the control group. In order to determine the fructose consumption and nutritional status of the patients, the dietician questioned the food consumption records of the patients for three days (two weekdays and one weekend). Regarding the patients' nutrient consumption status, their daily intake of energy, macronutrients, and fructose was calculated in the Beslenme Bilgi Sistemleri 7.1 (BEBIS - Nutrition Information Systems) program and the results were evaluated.

Results: Serum CRP levels (p=0.024), white blood cell count (p=0.038) and smoking rate (p=0.012) were higher in SCF group. Total energy (p=0.029), carbohydrate (p=0.047) and fructose consumption (p<0.001) were higher in SCF group. We performed univariate and multiple logistic regression analysis for major clinical factors and predictors of SCF as depicted in Table 1-2-3. The model we determined with logistic regression analysis was found to be significant (p<0.001). Multivariable logistic regression analyses demonstrated that higher, fructose consumption and smoking were independently associated with SCF.

Conclusions: In our study fructose consumption was higher in SCF group. High fructose consumption has the potential to play a role in SCF pathophysiology.

Table 1. Baseline characteristics of the study groups

Parameters	Patients with NCF (n=50)	Patients with SCF (n=45)	p value
Age, years	57.0 ± 10.6	55.1 ± 9.4	0.354
BMI, kg/m2	27.0 ± 4.3	28.2± 5.0	0.218
Female, n (%)	21 (42.0)	17 (37.8)	0.675
Diabetes Mellitus, n (%)	9 (18.0)	8 (17.8)	0.977
Hypertension, n (%)	18 (36.0)	14 (31.1)	0.615
Dyslipidemia, n (%)	13 (26.0)	17 (37.8)	0.218
Family history, n (%)	4 (8.0)	9 (20.0)	0.089
Smoking, n (%)	16 (32.0)	26 (57.8)	0.012

Data are given as mean ± SD, n or median (interquartile range). BMI, Body mass index; NCF, normal coronary flow; SCF, slow coronary flow. Categorical variables were compared using Pearson's chi square test, continuity correction chi square, or Fisher's exact test, as appropriate, and an independent samples t-test and the Mann-Whitney U test were used to compare continuous variables.

Table 2. Comparisons of laboratory findings, TIMI frame counts

Parameters	Patients with NCF (n=50)	Patients with SCF (n=45)	p value
Glucose, mg/dl	116.4 ± 47.6	124.0 ± 59.1	0.509
Creatinine, mg/dl	0.97 ± 0.2	1.01 ± 0.3	0.487
UricAcid, mg/dl	5.5 ± 2.1	5.8 ± 1.4	0.413
WBC count, 10^3/mm^3	9.6 ± 2.3	10.9 ± 2.8	0.038
Hemoglobin, g/dL	13.2 ± 1.7	13.8 ± 1.7	0.175
Platelet count, 10^3/mm^3	221.0 ± 57.4	231.2 ± 64.8	0.311
Total cholesterol, mg/dL	193.0 ± 86.6	187.2 ± 84.4	0.740
Triglyceride, mg/dL	160.1 ± 78.9	176.5 ± 88.9	0.679
LDL-cholesterol, mg/dL	116.6 ± 64.7	112.7 ± 59.9	0.790
HDL-cholesterol, mg/dL	45.5 ± 28.0	45.7 ± 26.1	0.967
Hs-CRP, mg/L	3.6 ± 2.3	5.1 ± 3.7	0.024
LVEF, %	58.7 ± 5.2	57.1 ± 4.5	0.100
TFC-LAD	35.9 ± 10.1	15.8 ± 4.9	< 0.001
TFC-Cx	28.9 ± 6.3	15.7 ± 6.1	< 0.001
TFC-RCA	29.6 ± 4.5	14.8 ± 5.1	< 0.001
FC-mean	31.5 ± 7.0	15.4 ± 5.4	< 0.001

Data are given as mean ± SD, n or median (interquartile range). HDL, high density lipoprotein; Hs-CRP, high-sensitivity C-reactive protein; LDL, low-density lipoprotein; LVEF, left ventricle ejection fraction; NCF, normal coronary flow; SCF, slow coronary flow; TFC,TIMI frame count; WBC, white blood cells. Independent samples t-test and the Mann-Whitney U test were used to compare continuous variables.

Table 3. Comparisons daily diet energy, macro nutrients and fructose consumption

Parameters	Patients with NCF (n=50)	Patients with SCF (n=45)	p value
Energy (kcal)	2472.9 ± 571.9	2780.5 ± 678.2	0.029
CHO (g)	245.5 ± 90.1	285.1 ± 101.2	0.047
CHO (TE%)	41.4 ± 8.4	41.7 ± 8.9	0.839
Protein (g)	84.4 ± 23.4	90.8 ± 29.2	0.235
Protein (TE%)	14.1 ± 3.0	13.4 ± 2.9	0.251
Lipid (g)	122.6 ± 35.7	137.3 ± 38.8	0.060
Lipid (TE%)	44.5 ± 7.7	44.5 ± 8.4	0.973
Fiber (g)	26.5 ± 8.1	27.4 ± 8.9	0.632
Fructose (g)	34.2 ± 14.8	48.9 ± 16.9	<0.001
Fructose (TE%)	5.5 ± 2.0	6.9 ± 1.5	<0.001

Data are given as mean ± SD, n or median (interquartile range). CHO, carbohydrate; TE, total energy. Independent samples t-test and the Mann-Whitney U test were used to compare continuous variables.

Table 4. Multivariate logistic regression analysis to predictig the slow coronary flow

	Univariable OR (95% CI)	p value	Multivariable OR (95% CI)	p value
Smoking	2.908 (1.257-6.725)	0.013	3.086 (1.140-8.353)	0.027
WBC count	1.190 (1.007-1.406)	0.041	1.189 (0.967-1.462)	0.101
Hs-CRP	1.140 (1.001-1.298)	0.048	1.180 (1.020-1.365)	0.069
Fructose consumption	1.058 (1.028-1.090)	<0.001	1.087 (1.037-1.140)	<0.001
Total energy consumption	1.003 (0.998-1.008)	0.023	0.999 (0.997-1.000)	0.188
Total carbohydrate consumption	1.004 (1.000-1.009)	0.059	1.004 (0.993-1.009)	0.129

CI, confidence interval; Hs-CRP, high-sensitivity C-reactive protein; WBC, white blood cells; OR, Odds ratio.

Coronary artery disease / Acute coronary syndrome

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Comparisons of microbiota-generated metabolites in patients with young and elderly acute coronary syndrome

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Background and Aim: Acute coronary syndrome (ACS) is a leading cause of death worldwide. There is great interest in defining the risk factors and under lying mechanisms of ACS among young people. The microbiota and its metabolites have recently become a popular research topic, yet there is still no study that investigated microbiota-generated metabolites as a possible risk factor in young patients with ACS. In this study, we aimed to investigate the relationship between microbiota-generated metabolites and ACS in young people.
Methods: This study included 44 young patients with ACS (<50 years of age), 39 elderly patients with ACS,44 patients with normal coronary arteries. Inflammatory parameters and serum trimethylamine N-oxide (TMAO) and choline levels were measured in all patients.
Results: Over-all, 44 young patients with ACS, 39 elderly patients with ACS, and 44 patients with normal coronary arteries were included in this study. Baseline characteristics and laboratory parameters of study population are presented in Table 1-2. There was no significant difference among the groups in terms of baseline characteristics except age (p<0.001). HDL (p=0.001), triglycerides (p=0.003), ESR (p=0.001), and hsCRP (p<0.001) were significantly different among the three groups. Microbiota parameters of study groups are shown in Table 3. TMAO and choline levels were significantly different among the three groups. To determine the clinical importance of these parameters in young patients with ACS, comparison of the three study groups was made. It was found that young patients with ACS had significantly higher levels of TMAO and choline compared to both the control and elderly ACS groups and elderly patients with ACS had significantly higher levels of TMAO than the control group. When all participants were included in correlation analysis (control group and ACS patients), TMAO was positively correlated with hsCRP(r=0.311, p<0.001), ESR (r=0.324, p=0.006), and BMI (r=0.200, p=0.024), while choline was positively correlated with hsCRP (r=0.222, p=0.012) and BMI (r=0.235, p=0.008). When only ACS patients were included in the analysis (young and elderly ACS patients), choline was negatively correlated with age (r=–0.243, p=0.027). Linear regression analysis was performed to determine the statistically significant prognostic factors (significant predictors) of TMAO. Two regression models were involved. The first model included young ACS and control groups, while the second model included young ACS and elderly ACS groups. In the first model, we found that young ACS (ß=0.399, p=0.004) and smoking ACS (ß=0.211, p=0.046) were significantly associated with TMAO level. In the second model, young ACS was significantly associated with TMAO level (ß=0.230, p=0.035) (Table 4).
Conclusions: The microbiota and its metabolites have recently become a popular research topic. TMAO is the most harmful dead-end metabolite of the microbiota. In this study, we found that young ACS was significantly associated with increased TMAO level.

Table 1. Comparison of baseline characteristics among groups

Variables	Control group (n=44)	Young ACS (n=44)	Elder ACS (n=39)	P	Control vs. Young	Control vs. Elder	Young vs. Elder
Age, years	46 (41 - 55)	48 (44-50)	65 (61-70)	<0.001	0.827	<0.001	<0.001
Male Gender (%)	23 (52)	30 (68)	28 (72)	0.137	0.130	0.070	0.722
BMI (kg/m²)	26.5 ± 3.2	27.6 ± 2.3	27.0 ± 2.4	0.135	0.062	0.377	0.253
DM (%)	3 (7)	4 (9)	4 (10)	0.851	0.695	0.576	0.858
HT (%)	6 (14)	11 (25)	5 (13)	0.880	0.179	0.388	0.37
Smoking (%)	8 (18)	6(14)	6 (15)	0.841	0.562	0.736	0.822
SBP (mm Hg)	126 (120-138)	120 (110-130)	124 (120-130)	0.310	0.059	0.643	0.150
DBP (mm Hg)	75 (70-83)	70 (65-80)	73 (70-80)	0.305	0.239	0.743	0.153
LVEF (%)	62 (58-65)	55(58-60)	55 (50-57)	0.058	0.056	0.080	0.062
Type of ACS (%)				0.556			
USAP		10 (23)	12 (31)				
NSTEMI	-	11 (25)	12 (31)				
STEMI		23 (52)	15 (39)				
Number of vessel with critical stenosis (%)				0.253			
1-vessel disease		18 (41)	10 (26)				
2-vessel disease		17 (39)	16 (41)				
3-vessel disease		9 (20)	13 (33)				
Final therapy (%)	-						
Medical therapy		0 (0)	4 (10.3)	0.029			
PCI		38 (86)	27 (62.2)	0.404			
CABG		6 (14)	8 (20.5)	0.742			

ACS - acute coronary syndrome, BMI- body mass index, SBP- systolic blood pressure, DBP- diastolic blood pressure, DM- Diabetes mellitus, HT- Hypertension, EF- ejection fraction, NSTEMI- non-ST segment elevation myocardial infarction, STEMI- ST elevated myocardial infarction, USAP- unstable angina pectoris, CAD- coronary artery disease, PCI- percutaneous coronary intervention, CABG- Coronary Artery Bypass Grafting

Table 2. Comparison of laboratory parameters among groups

Variables	Control group (n=44)	Young ACS (n=44)	Elder ACS (n=39)	P	Control vs. Young	Control vs. Elder	Young vs. Elder
WBC ($10^3/\mu L$)	10 (9-11)	11 (10-13)	10 (8-13)	0.780	0.040	0.844	0.135
Hemoglobin (g/dL)	12.5 ± 1.5	13.3 ±1.5	13.2 ±2.1	0.129	0.152	0.275	0.442
Platelets ($10^3/\mu L$)	264 (209-294)	228 (196-290)	234 (209-290)	0.185	0.056	0.315	0.578
Glucose (mg/dL)	88 (83-95)	91 (85-96)	85 (83-93)	0.132	0.162	0.598	0.062
Urea (mg/dL)	31.2 ± 10.5	29.7 ± 10.0	30.5 ± 10.3	0.184	0.494	0.752	0.728
Creatinine (mg/dL)	0.8± 0.20	0.9± 0.19	0.9 ± 0.20	0.280	0.132	0.120	0.908
Sodium (mEq/L)	138.6 ± 2.3	138.2± 4.6	139.5± 4.1	0.274	0.738	0.078	0.288
Potassium (mEq/L)	4.2 ± 0.4	4.2 ± 0.5	4.3± 0.4	0.463	0.521	0.192	0.553
AST (U/L)	19 (16-25)	19 (14-29)	19 (15-25)	0.658	0.235	0.603	0.210
ALT (U/L)	16 (13-20)	25 (19-47)	19 (14-28)	0.110	0.430	0.204	0.070
Total-C (mg/dL)	178.5 ± 26.9	162.6 ± 39.8	168.7 ± 53.2	0.184	0.030	0.285	0.554
LDL-C (mg/dL)	102 (90-122)	107 (72 -115)	103(75 -128)	0.819	0.499	0.767	0.834
HDL-C (mg/dL)	46 (39-56)	37 (32-42)	34 (29-45)	0.001	0.001	0.002	0.982
TG (mg/dL)	114 (76-150)	167(121-284)	169(107-227)	0.003	0.008	0.092	0.688
hs-CRP (mg/dL)	0.58(0.20 - 3.20)	6.70(2.70 - 14.4)	2.44 (1.60-7.40)	<0.001	<0.001	0.509	0.019
ESR (mm/h)	6 (3-13)	18 (9-26)	23 (10-42)	0.001	0.130	<0.001	0.135

ACS- acute coronary syndrome, ESR- Erythrocyte sedimentation rate, hsCRP- High sensitive C reactive protein, AST-Aspartate aminotransferase, ALT- Alanine aminotransferase, Total-C- Total cholesterol, HDL- High density lipoprotein, LDL- Low density lipoprotein, TG- Triglyceride, WBC- white blood count

Table 3. Comparison of microbiota parameters among groups

Variables	Control group (n=44)	Young ACS (n=44)	Elder ACS (n=39)	P	Control vs. Young	Control vs. Elder	Young vs. Elder
Choline (ng/ml)	0.45 (0.43-0.61)	0.62 (0.52-4.10)	0.54 (0.51-0.59)	0.001	<0.001	0.008	0.013
TMAO (µg/ml)	10 (8.60-13.31)	57 (13-123)	21(10-95)	0.001	<0.001	<0.001	0.041

TMAO- Trymetilamine-N oxide, ACS- acute coronary syndrome

Table 4. Multivariate linear regression analyses showing significant predictor of the TMAO

	Unstandardized coefficients		Standardized coefficients		
MODEL 1 (Young ACS + Control group)					
	B	**SE**	**β**	**t**	**p**
Age	0.641	0.877	0.080	0.732	0.467
Gender	19.616	14.392	0.153	1.363	0.177
BMI	4.688	2.427	0.212	1.931	0.057
HT	7.526	17.229	0.047	0.437	0.664
DM	14.469	24.692	0.062	0.586	0.560
Smoking	36.184	17.811	0.211	2.032	0.046
SBP	0.462	0.518	0.093	0.891	0.376
Creatinine	43.358	36.584	0.139	1.185	0.240
TC	0.148	0.220	0.082	0.674	0.502
LDL	0.188	0.242	0.094	0.776	0.440
HDL	0.036	0.470	0.009	.077	0.939
Triglyceride	0.013	0.055	0.028	0.242	0.809
hsCRP	0.260	0.550	0.054	0.473	0.637
Young ACS	50.046	16.748	0.399	2.988	**0.004**
MODEL 2 (Young ACS + Elder ACS)					
Age	2.309	1.870	0.387	1.235	0.221
Gender	4.627	15.389	0.035	0.301	0.765
BMI	0.664	3.329	0.026	0.200	0.842
HT	3.896	19.549	0.024	0.199	0.843
DM	10.991	22.386	0.053	0.491	0.625
Smoking	1.832	21.310	0.011	0.086	0.932
SBP	0.476	0.530	0.100	0.899	0.371
Creatinine	13.381	36.058	0.041	0.371	0.712
TC	0.362	0.210	0.272	1.727	0.088
LDL	0.383	0.221	0.274	1.736	0.087
Triglyceride	0.033	0.046	0.083	0.718	0.475
hsCRP	0.180	0.546	0.074	0.346	0.536
Young ACS	28.275	13.139	0.230	2.152	0.035

Coronary artery disease / Acute coronary syndrome

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Evaluation of the clinical value of heart rate variability in myocardial bridge

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Background and Aim: The muscle bridge (MB) seen in the coronary arteries is mostly benign. However, MB has been reported to be associated with adverse cardiac events in some studies. Although these adverse events appear to be caused primarily by coronary ischemia, it is thought that myocardial electrical conduction may also have negative effects. Also, rarely, sudden cardiac death is an important cause of mortality in

MB. The heart rate variability (HRV) used to assess problems in the cardiac autonomic nervous system predicts arrhythmic events. In this study, to determine the clinical value of HRV assessment in MB was aimed.
Methods: This study included 48 patients (33 males, mean age 58.4±12.2 years) who were diagnosed with MB (without other any coronary artery disease) and 48 patients with normal coronary artery (35 male, mean age 56.2±11.5 years). 24-hour Holter recordings were obtained, and HRV parameters were recorded from both groups. In the HRV analysis, the standard parameters obtained from the time-domain analysis of HRV including SDNN [Standard deviation (SD) of all NN intervals], SDANN (SD of the averages of NN intervals in all 5-minute segments of the entire recording), RMSSD (square root of the mean of the sum of the squares of differences between adjacent RR intervals), and PNN50 (the proportion of differences in successive NN intervals greater than 50 msn) were used.
Results: There were no significant differences between MB group and control group in terms of clinical and baseline demographic characteristics (Table 1). SDNN (93.59±12.81 vs 127.7±35.90 msn, p<0.001), SDANN (84.53±20.16 vs 145.16±32.07 msn, p<0.001), RMSSD (28 vs 50 msn, p=0.013), and PNN50 (15.4 vs 33%, p=0.021) were significantly lower in patients with MB compared to the control group (Table 2). In angiographic evaluation, myocardial bridging according to segment involvement is shown in table 3 (Number of patients with mid- left anterior descending coronary artery lumen systolic compression percentage >50% are 7). Also negative linear correlation was observed between the angiographic assessed degree of narrowing and HRV parameters [r=-0.538, p<0.001 for degree of narrowing and SDNN; r=-0.504, p=0.001 for degree of narrowing and SDANN; r=-0.398, p=0.029 for degree of narrowing and RMSSD; r=-0.515, p=0.001 for degree of narrowing and PNN50] (Table 4).
Conclusions: This study revealed that MB was significantly associated with impaired cardiac autonomic functions when evaluated with HRV parameters and an increased risk of sudden cardiac death in MB. In addition, these results suggest that HRV can be used for risk stratification in MB.

Table 1. Baseline characteristics of the study groups

Variables	MB group (n=48)	Control group (48)	P value
Age, years	58.4±12.2	56.2±11.5	0.428
Female, (n%)	15 (31.2)	10 (20.8)	0.204
Systolic blood pressure (mmHg)	112±11	114±12	0.356
Diastolic blood pressure (mmHg)	71±12	67±9	0.278
Pulse (beats / min)	72±10	74±12	0.305
Body mass index, kg/m2	29.2±4.9	28.1±4.2	0.289
Diabetes, (n%)	8 (16.6	7 (14.5)	0.674
Hypertension, (n%)	11 (22.9)	12 (25)	0.554
Dyslipidemia, (n%)	4 (8.3)	6 (12.5)	0.178
Smoker, (n%)	33	22	0.112
Glukose, mg/dL	116±44	124±49	0.498
Total cholesterol, mg/dL	193±84.4	188±86.4	0.652
Triglyceride, mg/dL	159.9±78.7	175.4±89.2	0.642
Low dencity cholesterol, mg/dL	115.9±63.9	112.1±58.6	0.692
Creatinine, mg/dL	0.9±0.2	1.0±0.3	0.408
Sodium, mml/L	138.6±2.1	139.1±3.1	0.562
Potassium, mmol/L	4.3±0.5	4.2±0.4	0.112
Hemoglobin, g/L	13.2±2.3	13.6±1.9	0.401
Left ventricular ejection fraction (%)	58.5±5.4	57.4±4.4	0.206
Left ventricular end diastolic dimension (mm)	44.7±5.3	45.6±7.4	0.248
Left ventricular end systolic dimension (mm)	29.4±6.4	30.4±5.9	0.192
Interventricular septum thickness (mm)	11.1±3.2	11.3±3.4	0.604
Left atrium dimension (mm)	37.2±5.3	38.2±6.4	0.551
Myocardial bridge lenght (mm)	18.6±3.4		

MB: myocardial bridge.

Table 2. Comparison of HRV parameters between groups

	MB group (n=48)	Control group (n=48)	P value
SDNN (msn)	93.59±12.81	127.7±35.90	<0.001
SDANN (msn)	84.53±20.16	145.16±32.07	<0.001
RMSSD (msn)	28(12/48)	50(36/86)	0.013
PNN50 (%)	15.4(5/25)	33(22/55)	0.021

SDNN: standard deviations of all NN intervals, SDANN: standard deviation of the averages of NN intervals in all 5-minute segments of the entire recording, RMSSD: the square root of the mean of the sum of the squares of differences between adjacent NN intervals, PNN50: the number of pairs of adjacent NN intervals differing by more than 50 msn divided by the total number of all NN intervals,HRV: heart rate variability, MB: myocardial bridge.

Table 3. Myocardial bridging according to segment involvement in angiographic evaluation

Coronary artery involvement	Group A	Group B
Mid- left anterior descending artery	15	7
Distal-left anterior descending artery	12	8
Circumflex artery	3	1
Right coronary artery	1	-
Left anterior descending artery and circumflex artery	1	-

Group A, the percentage of systolic compression of LAD coronary artery lumen <50% Group B, the percentage of systolic compression of LAD coronary artery lumen ≥50%.

Table 4. Correlation between degree of narrowing and HRV parameters

Variables	Coefficient	P value
SDNN	-0.538	<0.001
SDANN	-0.504	0.001
RMSSD	-0.398	0.029
PNN50	-0.515	0.001

SDNN: standard deviations of all NN intervals, SDANN: standard deviation of the averages of NN intervals in all 5-minute segments of the entire recording, RMSSD: the square root of the mean of the sum of the squares of differences between adjacent NN intervals, PNN50: the number of pairs of adjacent NN intervals differing by more than 50 msn divided by the total number of all NN intervals, HRV: heart rate variability.

Coronary artery disease / Acute coronary syndrome

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Effects of inspiratory muscle training on cardiac functions, exercise capacity and functional capacity in patients with stable angina: Preliminary results

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Background and Aim: Stable angina is a life restricting disease. However, there has not been shown the superiority of revascularization strategies when compared to optical medical therapy in prevention of cardiac mortality. Pulmonary rehabilitation is not only used for weaning from supportive devices in patients at intensive care units but also with chronic pulmonary diseases and heart failure patients in outpatients clinics. Inspiratory muscle training (IMT) applies a load to the diaphragm and increases functional capacity, inspiratory muscle strength and quality of life. So, we aimed to evaluate the effectiveness inspiratory muscle training in patients with stable angina without any respiratory complaints.
Methods: Patients with atherosclerotic stable angina (Canada 2-3) were splitted into the 2 groups, randomly. Twelve patients(age 55.58±4.75 years) in training group received IMT at 30% of MIP and 12 patients in control group (age 61.16±8.44 years) received minimum load of the device. IMT was applied to all patients by POWERbreathe Classic Medium Resistance (Fitness) (PowerBreathe, IMT Technologies Ltd., Birmingham, England) during 30 minutes every day for 8 weeks. Additionally walking program was applied to all patients. for 30 minutes-3 days in a week during 8 weeks Cardiac functions with echocardiography, exercise capacity with treadmill exercise test and functional capacity with 6-minute walking test (6-MWT) were evaluated both at the begining and at the end of the training.
Results: Clinical and demographic characteristics were similar both in groups (p>0.05,table 1).Echocardiographic findings were also similar between groups (p>0.05). 6-MWT distance was increased after training both within groups; in training group result was significant [-38.14 95% CI [(-57.0)1-(-19.27)m] (p<0.05), however in control group [-15.14 95% CI (-32.93-(2,85)m] (p=0.09) it was borderline (Table 2). METs result was significant (pre-training 8.04±2.47 METs vs post-training 10.16±1.87 METs) within the training group (p<0.05), however, not in control group (pre-therapy 8.04±3.31 vs. post-therapy 7.84±2.97 METs) (p>0.05). Differences of METs result was significant (2.14±2.52 vs. -0.01±1,46 METs) between the groups (p<0.05) while differences of 6-minute walking test (38.14±29.69 m vs. 15.04±25.01 m) was borderline different between the groups (p =0.06). Finally angina severity was decreased (p<0.05) in training group [pre-therapy 2(2-3) vs post-therapy 1.5(1-2)] as Canada classification. However, there was not observed any effect of treatment on angina severity in control group. Angina severity was also statistifically significant between groups p<0.05, Table 2).
Conclusions: METs and functional capacity of stable angina patients were increased after IMT treatment. Since stable angina lowers the quality of life, maximal IMT may be performed in whom stricken from stable angina. However, tretment should be performed in maximal inspiratory pressure.

Table 1. Demographic and clinical characteristics of the groups

Variables	Training Group Mean±SD Median(IQR)	Control Group Mean±SD median(IQR)	p
Age (years)	55.58±4.75	61.16±8.44	0.06
Female / Male n (%)	2(%16.7) / 10(%83.3.9)	0(%0) / 12(%100)	0.14
Body mass index (kg/m2)	29.59±5.72	30.22±5.00	0.78
Pack x years-smoking (n)	26.5(8-47.5)	43.50(26.25-60)	0.22
Smoking n (%) Current Ex Non-smoker	 33.3 50.0 16.7	 25 58.3 16.7	 0.89
Diabetes (%)	33.3	16,7	0.35
Hypertension (%)	33,3	41.7	0.68
Diabetes +Hypertension (%)	41.7	25	0.39
Dyslipidemia (%)	33.3	16.7	0.35
Alcohol Use (%) Never Current Ex	 58.3 33.3 8.3	 66.7 33.3 0	 0.56
Exercise habit (%) Yes No	 33.3 66.7	 41.7 58.3	 0.50
Canada Class (II-III)	2(2-3)	2(2-2)	0.14

Table 2. Comparision of functional capacity, exercise capacity and left ventricular ejection fraction between and within the groups

	Training Group Pretest (n:12) X±SD /median(IQR)	Training Group Post test(n:12) X±SD/ median(IQR)	Training Group Within the group p	Control Group Pretest (n:12) X±SD/ median(IQR)	Control Group Post test (n:12) X±SD/ median(IQR)	Control Group Within the group p	Δ Between the groups p
6MWT(m)	503.41±47.14	541.55±56.98	0.01*	481.70±70.84	496.74±66.68	0.09	0.06
LVEF (%)	61.66±7.69	61.31±7.77	0.89	58.50±6.87	60.92±7.29	0.68	0.63
METs	8.04±2.47	10.16±1.87	0.02*	8.04±3.01	7.84±2.97	0.98	0.03*
Canada Class(II-III)	2(2-3)	1.5(1-2)	0.02*	2(2-2)	2(2-2)	0,99	0.01*

*Δ: pre-training, post-training test 6MWT:Six minute walking test, LVEF: Left ventricular ejection fraction *:p<0.05.*

Coronary artery disease / Acute coronary syndrome

OP-187

Association between the prevalence of coronary artery disease in acute coronary syndrome patients as assessed by Syntax Score and the sleep disorder as measured by Pittsburgh Sleep Quality Index (PSQI)

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Background and Aim: Ischemic heart disease (IHD) has the highest mortality rate globally. Acute Coronary Syndrome (ACS) is the acute manifestation of IHD. It is known that sleep disorder may cause additional coronary situations in ACS patients through several mechanisms such as increased sympathetic activity. Pittsburgh Sleep Quality Index (PSQI), efficiency determining sleep disorder of which has been scientifically proven, is in the form of a questionnaire consisted of 11 questions, which can be easily applied to the patient in polyclinic environment. The aim of this study is to show the relation between the prevalence of Coronary Artery Disease determined via Syntax Score with the sleep disorder and thus make it possible to implement a risk classification in terms of unfavorable cardiac events.

Methods: The study has been conducted retrospectively. 424 ACS patients were chosen among those who applied to Ankara City Hospital as of February 2019 and satisfy the criteria for inclusion through coronary angiography. They were applied the PSQI questionnaire either face to face at the polyclinic or via phone. The patients were divided into two groups according to their syntax scores as "≤22" and ">22." Their PSQI scores were evaluated in order to reveal any difference between these two groups.

Results: Median (min-max) age of the patients in our study was 60 (28-93) and a positive correlation was detected between the age of the patients and their PSQI global score (p<0.01). 79% of the patients were male and no meaningful difference between male and female patients was detected (p>0.05). While PSQI global score median (min-max) of 294 patients with Syntax Score ≤22 was 4 (0-19), PSQI global score median (min-max) of 130 patients with Syntax Score >22 was found 6,5 (1-19), and a meaningful difference is present between these two groups in terms of global score (p<0.05). Syntax Score average for patients with a global score of 6 and over, which is accepted as the limit for a possible sleep disorder or low sleep quality was considerably higher than those patients with a global score below 6 (p<0,05). Sleep latencies, total sleep durations and sleep efficiencies revealed a significant difference between the two groups of Syntax Score ≤22 and >22 (p<0.05). Also other independent predictors for patients of Syntax Score >22 are detected as follows: PSQI global score (<0.001), Hypertension (=0.049) and left ventricular ejection fraction (=0.0090).

Conclusions: In conclusion, PSQI global score was found significantly higher in ACS patients with a Syntax Score >22 when compared to those with a Syntax Score ≤22. An association between global score and the Syntax Score was detected. The role of sleep disorders in the development of additional unfavorable cardiovascular events, and the association of Syntax Score with low sleep quality show that treatment of sleep disorders may help prevent morbidity and mortality rates in ACS patients.

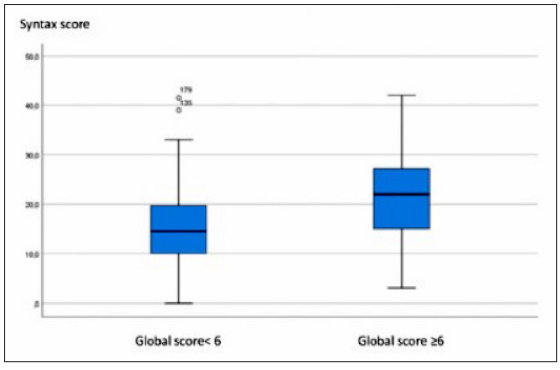

Figure 1. Syntax score comparison of Global score <6 and Global score ≥6 Groups.

Table 1. Syntax Score groups baseline characteristic

	Total (N=424)	SYNTAX Score≤22 (n=294)	SYNTAX Score>22 (n=130)	p value
Age,year	60 (28-93)	59,69±11,85	61,9±12,98	0,130
Male, sex, n (%)	333 (78,5)	227 (77,2)	106 (81,5)	0,232
Smoking, n (%)	147 (34,7)	110 (37,4)	37 (28,5)	0,028*
BMI (kg/m2)	27,5 (17,9-41,6)	27,5 (17,9-41,5)	27,5 (20,8-41,6)	0,322
Hypertension, n (%)	173 (40,8)	127 (43,2)	46 (35,4)	0,055
Diabetes, n (%)	102 (24,1)	62 (21,1)	40 (30,8)	0,024*
Ejection fraction %	47,71±8,45	48,91±8,23	44,98±8,32	0,000*
Diuretic, n (%)	57 (13,4)	37 (12,6)	20 (15,4)	0,477
Hemoglobin, g/dl	14,4 (5,2-18,3)	14,4 (5,2-18,3)	14,3 (7,1-17,9)	0,335
Platelets,x10^3/uL	242 (10-1125)	247 (10-1125)	235 (108-683)	0,425
Whitebloodcell, x10^3/uL	10,7 (4,6-23,4)	10,7 (4,8-22,5)	10,7 (4,6-23,4)	0,880
Creatinine,mg/dl	0,94±0,31	0,93±0,31	0,97±0,31	0,159
Gfr,ml/dk/1.73 m2	87,89±23,41	89,2±22,24	84,93±25,69	0,110
Uric acit,mg/dl	5,6 (0-93)	5,6 (0-93)	5,6 (2,9-17)	0,816
Crp,mg/L	4 (0-255,2)	4 (0-255,2)	4,3 (0-121)	0,223
Total cholesterol,mg/dl	176 (81-579)	176,89±38,25	177 (85-579)	0,110
HDL,mg/dl	39 (11-259)	39 (11-259)	38 (17-259)	0,801
LDL,mg/dl	112,67±35,43	110,5±34,09	117,57±37,94	0,121
Triglycerides,mg/dl	119 (28-1420)	123 (28-521)	115,5 (31-1420)	0,719
Fasting glucose,mg/dl	105 (51-486)	102 (51-355)	109 (69-486)	0,061
First troponin,ng/ml	0,4 (0-91,4)	0,4 (0-91,4)	0,3 (0-82,4)	0,783
Peak troponin,ng/ml	3,7 (0-195)	3,8 (0-103)	3,7 (0-195)	0,555
Ckmb,U/L	35,4 (0,1-652)	31,9 (0,7-445,9)	46,2 (0,1-652)	0,007*

LDL:Low dansity lipoprotein,HDL:High dansity lipoprotein,Syntax:*The* Synergy Between *Percutaneous Coronary Intervention With* Taxus and Cardiac Surgery, BMI:Body mass index

P<0,05 shows statistical significance

Table 2. Global score comparison of Syntax score ≤22 and >22 groups

	Syntax score ≤ 22 (n=294)	Syntax score > 22 (n=130)	P value
Global score	4 (0-19)	6,5 (1-19)	< 0,05

P<0,05 shows statistical significance

Table 3. Syntax score >22 predictors

	Univariate Analysis		Multivariate Analysis	
	OR (CI %95)	p	OR (CI %95)	p
Global Score	1.209 (1.128 1.296)	**<0.001**	1.172 (1.083 1.269)	**<0.001**
BMI	0.970 (0.919 1.024)	0.273		
ACS type (STEMI)	1.353 (0.837 2.187)	0.426		
Sex (Male)	1.304 (0.775 2.193)	0.318		
Age	1.015 (0.998 1.032)	**0.087**	1.004 (0.978 1.031)	0.758
Hypertension	1.397 (0.911 2.142)	0.125	1.777 (1.002 3.149)	**0.049**
Diabetes	0.601 (0.377 0.958)	**0.032**	0.832 (0.404 1.712)	0.617
Stroke	0.436 (0.087 2.192)	0.314		
Atrial fibrillation	1.107 (0.212 5.783)	0.904		
Ejection fraction	0.945 (0.921 0.970)	**<0.001**	0.957 (0.927 0.989)	**0.009**
Smoking	1.503 (0.960 2.352)	**0.075**	1.623 (0.917 2.874)	0.097
Diuretic	1.263 (0.701 2.274)	0.437		
Hemoglobin	0.911 (0.815 1.019)	0.102	0.956 (0.830 1.102)	0.535
Platelets	1.000 (0.997 1.002)	0.840		
White blood cell	1.016 (0.953 1.084)	0.620		
Creatinine	1.539 (0.800 2.959)	0.196	1.085 (0.401 2.937)	0.873
Gfr	0.992 (0.983 1.001)	**0.084**	0.992 (0.975 1.010)	0.397
Uric acid	0.987 (0.931 1.045)	0.647		
Crp	1.008 (0.998 1.019)	0.131	1.001 (0.989 1.012)	0.927
Total cholesterol	1.005 (1.000 1.010)	**0.040**	1.003 (0.995 1.012)	0.468
HDL	1.002 (0.992 1.013)	0.662		
LDL	1.006 (1.000 1.012)	**0.059**	1.004 (0.993 1.015)	0.464
Triglycerides	1.001 (0.999 1.002)	0.578		
Fasting glucose	1.005 (1.001 1.009)	**0.007**	1.004 (0.998 1.011)	0.160
First troponin	0.998 (0.979 1.018)	0.869		
Peak troponin	0.993 (0.982 1.003)	0.181	0.997 (0.985 1.010)	0.666
Ckmb	1.004 (1.001 1.006)	**0.002**	1.002 (0.999 1.006)	0.181

Coronary artery disease / Acute coronary syndrome

OP-188

The relation between CHA2DS2VASC score and reperfusion success with short-term mortality in patients with saphenous vein graft disease who underwent elective coronary angiography

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Background and Aim: Coronary artery disease (CAD) is the most significant cause of morbidity and mortality in developed countries. No-reflow can develop after stenting, even if the percutaneous coronary intervention (PCI) is conducted well. Patients with degenerated saphenous vein grafts (SVG) have a higher risk of developing no-reflow due to thrombus formation and/or distal emboli. No-reflow is associated with adverse cardiac events. In the literature, there are studies that the CHA2DS2-VASc score is a no-reflow predictor in patients with acute coronary syndrome. In our study, we aimed to assess the association between CHA2DS2-VASc score and no-reflow after the procedure and short-term mortality in patients with SVG who underwent elective PCI.

Methods: Our study was designed retrospectively. A total of 118 patients who underwent elective PCI to SVG and fulfilled the inclusion criteria were included in the study. Coronary angiographies of the patients included in the study were evaluated. The subjects were divided into two groups according to the no-reflow (patients with a TIMI score of 2 or less) and normal-flow (those with a TIMI score of 3). The CHA2DS2-VASc score of all patients was calculated.

Results: In total, 118 patients were involved in the study (age 66.4±9.2 years, female gender 25.4%). Patients were divided into two groups based on the no-reflow phenomenon (Table 1). Apart from the history of diabetes (p=0.032), demographic data, blood parameters, ejection fraction, total stent length and diameter, drug use, median CHA2DS2-VASc score, and adverse cardiac events did not differ between the groups. When the patients were classified as CHA2DS2-VASc >2 and CHA2DS2-VASc ≤2, the presence of no-reflow did not differ between the groups (Table1 and 2). In univariate logistic regression analysis, the presence of diabetes and stent length appeared to be associated with no-reflow, but not in multivariate analysis (Table 3). When the study patients were evaluated in terms of early adverse events, in-hospital death, 1-year death, TVR, and 1-year total adverse cardiac events (TVR and 1-year death); outcomes did not differ between the groups. The median CHA2DS2-VASc score was higher in patients who died at a 1-year follow-up [4.5 (2-6) vs. 3 (1-7), p=0.047].

Conclusions: No reflow reflects an important clinical condition that can cause heart failure, malignant arrhythmia, cardiogenic shock, and death. The correct determination of the risks for no-reflow before the procedure will enable precise intervention preference in elective patients and, on an individual basis, medical follow-up or native vessel revascularization will be preferred. In our study, we did not observe a significant relationship between no-reflow and CHA2DS2-VASc score. Larger studies are needed to reveal the indicators for the improvement of the post-intervention reperfusion status and the predictors of no-reflow in patients undergoing PCI to the SVG that stands out as a risky group even if performed in elective patients.

Table 1. Distribution of baseline features of the study patients according to the reperfusion status after the procedure

Variables	Normal flow	No-reflow	P-value
Age,year	66,4±9,9	64,0±6,8	0,951
Female gender, n (%)	27 (27,2%)	3 (15,7%)	0,394
Hypertension, n (%)	65 (65,6%)	13 (68,4%)	0,859
Diabetes, n(%)	36 (36%)	12 (63%)	0,032
Heart failure, n (%)	21 (21,2%)	6 (31,5%)	0,363
CVD, n (%)	4 (4,0%)	2(10,5%)	0,251
Cigaret, n(%)	20 (20,2%)	6 (31,5%)	0,343
LVEF, %	50 (20-65)	47 (30-55)	0,092
Bypass age, yıl	13,2±5,2	13,7±6,7	0,330
CHA2DS2-VASc score	3 (1-6)	4 (2-7)	0,504
CHA2DS2-VASc score >2, n (%)	71 (71,7%)	12 (63,1)	0,454
Saphenous vein graft with lesion, n (%)			0,189
RCA	45(45,4%)	12 (63,1)	
CX	37 (37,3%)	4 (21,0%)	
Diagonal	13 (13,1%)	2 (10,5%)	
LAD	4 (4,0%)	1 (5,2%)	
Stent length, mm	20 (9-70)	24 (12-60)	0,061
Stent diameter, mm	3,0 (2,25-4,5)	3,5 (2,5-4,0)	0,272
The use of tirofiban during PCI	5 (5,0%)	15 (78,9%)	<0,001
Urea, mg/dl	38 (16-108)	34 (19-67)	0,382
Creatinine, mg/dl	1,0 (0,5-2,0)	1,0 (0,7-1,2)	0,740
Fasting blood sugar, mg/dl	113 (71-318)	122 (97-232)	0,330
Total kolesterol, mg/dl	241,9±58,1	197,6±82,2	0,515
Total cholesterol, mg/dl	115,1±75,1	138,8±44,2	0,223
HDL-cholesterol, mg/dl	42 (50-74)	48 (36-71)	0,482
Triglycerides, mg/dl	145 (50-747)	174 (86-312)	0,287
Hemoglobin, g/dl	13,4±1,8	13,9±1,4	0,094
Platelets, x10^3/uL	224,3±63,2	217,6±59,0	0,809
White blood cell, x10^3/uL	8,5±6,6	9,3±2,4	0,681

LDL: Low dansity lipoprotein, HDL: High dansity lipoprotein, CVD: Cerebrovascular disease, PCI: Percutaneous coronary intervention, LAD: Left anterior descending artery, CX: Sirkumflex artery, RCA: Right coronary artery p<0.05 shows statistical significance.

Table 2. Comparison of reperfusion groups in terms of mortality and cardiac adverse events

Cardiac event, n (%)	Normal flow	No-reflow	p-value
In-hospital death	0(0%)	1 (5,2%)	0,164
All-cause death in 1-year follow-up	5 (5,0%)	1 (5,2%)	0,969
TVR in 1-year follow-up	7 (0,07%)	0 (0%)	0,597
Total adverse cardiac event at 1 year follow-up	12 (12,1%)	1(5,2%)	0,690

Adverse cardiac event description: TVR + all-cause death, TVR: target vessel revascularization p<0.05 shows statistical significance.

Table 3. No reflow predictors

	Univariate			Multivariate		
Variables	Odds ratio	95% Confidence Interval	p-value	Odds ratio	95% Confidence Interval	p-value
Age	1,002	0,950-1,056	0,951			
Male gender	0,500	0,135-1,853	0,300			
Diabetes	3,000	1,084-8,304	**0,034**	3,655	0,837-15,962	0,085
Hypertension	1,100	0,383-3,157	0,859			
Cigarette	2,000	0,633-6,318	**0,238**	1,937	0,442-8,481	0,380
Heart failure	1,810	0,607-5,396	0,287			
CVD	2,735	0,464-16,129	0,266			
CHA2DS2-VASc	1,243	0,856-1,807	0,253			
Use of clopidogrel	0,545	0,133-2,237	0,400			
Statin use	0,618	0,073-5,251	0,659			
Fasting blood sugar	1,001	0,992-1,011	0,796			
LDL	1,004	0,997-1,011	0,261			
Bypass age	1,043	0,958-1,136	0,328			
Stent length	1,041	1,001-1,083	**0,044**	1,032	0,910-1,017	0,171
Stent diameter	0,518	0,172-1,563	**0,243**	0,632	0,150-2,656	0,581

Coronary artery disease / Acute coronary syndrome

OP-189

Association of up-regulated miR-29b in epicardial adipose tissue with coronary atherosclerosis

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Background and Aim: Epicardial adipose tissue (EAT) is a particular form of visceral adipose tissue deposited around the heart and found in considerable quantities around subepicardial coronary arteries. There is no Şbrous fascial layer between EAT and its adjacent coronary arteries to impede diffusion of free fatty acids and adipokines. Because of the close anatomical relationship, ıt was thought that, EAT plays a role in the development of coronary atherosclerosis through direct endocrine and paracrine effects. MicroRNAs (miRNAs) regulate cardiovascular biology and disease, but the role of flow-sensitive microRNAs in atherosclerosis is still unclear. In this study, we aimed to determine the expression levels of miR-29b-3p gene in EAT and the association of this gene with the coronary atherosclerosis. We aimed to investigate the relationships between miR-29b-3p and coronary atherosclerosis in EAT.

Methods: EAT was obtained during coronary artery bypass graft (CABG) surgery from 41 CAD male patients and from12 non-CAD male patients presented for valve surgery. Trizol based RNA isolation was performed. Expression levels for miRNA gene were examined by real-time PCR analysis on LC480 using Taqman miRNA expression assays.

Results: The expression of miRNA-29b-3p were found to be up-regulated in CAD group (2.82±2.9) when compared to non-CAD group (1.09±1.4) (p=0.020). In the case of diabetes, which is an important risk factor of Coronary Atherosclerosis, a significant association was detected in the groups (p<0.05). However, these association was not determined after controlling for age and diabetes mellitus covariates.

Conclusions: Our findings indicated that increased miR-29b-3p expression levels in EAT are associated with coronary atherosclerois but this relationship was not determined after controlling age and diabetes mellitus. In conclusion, it may be speculated that miR-29b-3p expression levels affect the increase of susceptibility for coronary atherosclerosis, but this experiment should done with larger population to clarify this issue.

Coronary artery disease / Acute coronary syndrome

OP-190

Clinical features and treatment outcomes of patients with Takotsubo Cardiomyopathy: a single center experience

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Background and Aim: Takotsubo cardiomyopathy (TC) was first described in 1990 as a transient and prominent akinesia of the apical and middle segments of the left ventricle. Patients generally present following a severe emotional or physical stress, and the syndrome mimics an acute myocardial infarction. The electrocardiograph includes ST elevation, inversed T waves, and pathological Q waves, which strongly suggest a STEMI. Angiographic assessments generally fail to diagnose an obstructed coronary artery. The outcomes are generally good. Nevertheless, it may also cause considerable in-hospital mortality rates that can reach

up to 2%. Based on this background, we have retrospectively evaluated our experience on TC cases to identify the clinical characteristics and outcomes among Turkish patients.

Methods: The study was conducted as a retrospective chart-review. A total of 1782 patients (from a total of 5363 left heart catheterizations) who had urgent angiography due to suspected acute coronary syndrome (ACS) were screened for whom met the Mayo clinic diagnostic criteria for TC, and 24 patients with TC were included in the analyses.

Results: 92% of cases were females, and the median age was 60.5 years. Most frequent cardiovascular risk factor was hypertension (62.5%), and a physical or emotional stressor was present in 79.2% of the cases. Median symptom duration until admission was 4 hours. Prevailing presenting symptom was chest pain (95.8%). Median CK-MB was 28.1 U/L, and troponin was 1496.5 pg/mL at admission. Electrocardiographic assessments revealed that ST elevation was present in 41.7% and T wave inversion in 66.7% of the cases. The echocardiography revealed the median EF at admission was 35%, and ECG revealed that 58.3% of patients had normal angiography, and 41.7% had non-significant chances. 66.7% of patients had midventricular, and 54.2% had apical type TC (Table 1). Distribution of in-hospital and discharge medications were presented in Table 2, and patient outcomes in Table 3. Median hospitalization was 3 days. During hospitalization 12.5% had acute heart failure. In-hospital and long-term mortality rates were 4.2%, 8.3%, respectively. The changes in the clinical assessments of patients during follow-ups revealed that the left-ventricular EF (p<0.001) and T wave inversion (p<0.001) were significantly improved (Table 4).

Conclusions: Our results were generally in accordance with the literature data. Majority of the cases were presented following an emotional or physical stressor, and presenting symptoms and electrocardiographic assessments were clinically in accordance with the TC. Although the in-hospital and long-term mortality rates in this study seems higher than expected, the relatively small study population should be considered when interpreting the results. Likewise, no recurrence was recorded among our cases, which was expected to reach up to 10% in the first year of follow-up according to the literature data.

Table 1. Demographic and clinical characteristics of patients

	Patients (n=24)
	Median [min-max] / n (%)
Demographics	
Age (years)	60.5 [28-88]
Sex (female)	22 (91.7)
Cardiovascular risk factors	
Hypertension	15 (62.5)
Obesity (BMI≥30kg/m2)	6 (25)
Dyslipidemia	5 (20.8)
Diabetes Mellitus	5 (20.8)
Psychiatric history	1 (4.2)
Predisposing factors	
Physical or emotional stress	19 (79.2)
Emotional stress	17 (70.8)
Physical stress	4 (16.7)
Neurologic disorders	1 (4.2)
Presentation	
Symptom duration (hours)	4 [1-12]
Chest pain	23 (95.8)
General weaknes	13 (54.2)
Nausea-vomiting	7 (29.2)
Dyspnea	6 (25)
Heart palpitations	5 (20.8)
Syncope	2 (8.3)
Clinical examination	
Mean Heart rate (beats/min)	72 [51-118]
Mean Systolic blood pressure (mmHg)	120 [90-160]
Mean Diastolic blood pressure (mmHg)	70 [60-90]
Laboratory values, mean±SD	
C-Reactive protein (mg/dl)	15.7 [2.9-61.3]

Table 1. Continues

Hemoglobin (g/dl)	12.7 [9.1-15.9]
Creatinine (mg/dl)	0.8 [0.6-1.5]
HbA1c (%)	6.6 [6.1-7.1]
Cardiac biomarkers	
Creatinine kinase MB fraction (U/L)	28.1 [7.3-163]
Admidted troponin level (pg/Ml)	1496.5 [86.3-21005.7]
CHA2DS2-VASc score	2 [0-6]
ECG findings	
ST elevation	10 (41.7)
ST depression	5 (20.8)
Nonspecific changes	5 (20.8)
Minor ST ve T wave abnormalities	5 (20.8)
Atrial flutter/fibrillation	1 (4.2)
Twave inversion	16 (66.7)
Positive T waves in aVR	13 (54.2)
Negative T waves are deep, symmetrical and widespread	11 (45.8)
No negative T waves in V1	11 (45.8)
No negative T wave in V1 plus positive T wave in aVR	10 (41.7)
Progressive QT-interval prolongation	3 (12.5)
QT prolongation in acute phase	8 (33.3)
Echocardiographic characteristics	
Ejection fraction in acute phase LVEF (%)	35 [30-40]
Angiographic and ventriculographic characteristics	
Normal coronary angiography	14 (58.3)
Non-significant abnormalities	10 (41.7)
Takotsubo type	
Mid ventricular	16 (66.7)
Apical+Midventricular	13 (54.2)
Focal	1 (4.2)

Table 2. Distribution of in-hospital and discharge medications

	Patients (n=24)
	n (%)
Medication	
Pre-hospital	
ACE/ARB	5 (20.8)
In-hospital	
Aspirin	23 (95.8)
ACE/ARB	23 (95.8)
Beta bockers	22 (91.7)
Statine	22 (91.7)
Clopidogrel	21 (87.5)
LMWH	20 (83.3)
Nitrate	14 (58.3)
MRA (aldacton)	5 (20.8)
Oral anticoagulant	2 (8.3)
Discharge	
Beta bockers	22 (91.7)
Aspirin	22 (91.7)
Clopidogrel	20 (83.3)
Statine	19 (79.2)
ACE/ARB	19 (79.2)
Spronalactone	3 (12.5)
Nitrate	1 (4.2)

Table 3. Patient outcomes

	Patients (n=24)
	Median [min-max] / n (%)
Hospitalization (days)	3 [3-6]
In-hospital complications	
Acute heart failure	3 (12.5)
Asystole	1 (4.2)
Mortality	
In-hospital	1 (4.2)
Long-term (all-cause)	2 (8.3)

Table 4. Admission, discharge, and follow-up assessments

	Admission	Discharge	1st month	6th month	12th month	
	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	p
Heart rate (bpm)	76.5±20.4	67.4±8.4	65.5±6.8	65.9±4.2	64.8±3.7	0.581
LVEF (%)	35.5±6.4	50.9±6	62.2±2.4	63.1±2.6	63.2±2.4	<0.001
Twave inversion	16 (66.7)	18 (75)	1 (4.2)	1 (4.2)	-	<0.001
Neutrophil/Lymphocyte ratio	4.1±2.7	3.7±5.1	12.5±39.7	3.4±3.7	1.9±0.8	0.042
Platelet/Lymphocyte ratio	127.6±63.7	121.3±63.6	120.7±54.5	156.1±154.9	122±61.4	0.871
Lymphocyte/Monocyte ratio	3.5±1.8	3.3±2	4.3±2.6	4.3±1.7	4.7±2.4	0.017
MCV (Mean Corpuscular Volume)	86.3±6.0	83.6±14.3	86.3±6.7	78.4±22.1	96.3±38.8	0.23
MPV (mean platelet volume)	8.4±1.7	8.5±1.6	8.3±2.1	8.2±2.3	7.4±1.4	0.6

Coronary artery disease / Acute coronary syndrome

OP-191

Echocardiographic assessment of left ventricular filling pressure in patients with ST elevation myocardial infarction: An invasive validation study

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Background and Aim: Assessment of left ventricular filling pressure (LVFP) is of clinical importance in patients with ST elevation myocardial infarction (STEMI). Although several echocardiographic parameters are recommended for the assessment of LVFP, validation of these parameters in patients with STEMI is missing. We aimed to investigate the clinical utility of these parameters in acute settings.

Methods: We prospectively included consecutive patients with STEMI. LV pressure was measured following primary PCI and echocardiographic examination was performed within 24 hours after admission. Mean left atrial pressure (mLAP) was calculated both invasively using Yamamoto's formula and non-invasively using Naugeh's formula. mLAP was considered increased when it exceeded 18 mmHg.

Results: Patients were grouped according to mLAP; group 1 (41 patients, mLAP <18) and group 2 (114 patients, mLAP >18). There was no significant difference between groups in terms of comorbidities. Pro-BNP levels [83.0 (39.0-294) vs 380.5 (135-920), p<0.001] and peak level of Hs-Tnt [2.50 (1.62-5.14) vs 5.21 (2.80-9.70), p-value: 0.002] were significantly higher in group 2. Average E/e' ratio was significantly higher in group 2 (10.19±3.15 vs 12.04±4.83, p=0.046). Isovolumetric relaxation time was longer in group 2 (79.9±12.6 vs 90.4±17.2, p<0.001) and left atrial volume index(LAVI) was also significantly higher in group 2 (23.8±6.4 vs 28.5±8.3, p<0.001). Regression analyses revealed that septal, lateral and average E/e' ratio, tricuspid regurgitation velocity, LAVI and left ventricular volume are correlated with mLAP. Among group 2 patients only 14 Patients fulfilled the increased LVFP criteria suggested by current guidelines.

Conclusions: In conclusion, echocardiographic parameters indicating increased LVFP requires validation and may be modified in patients with STEMI. Moreover, current algorithms underestimate the actual number of patients with increased LVFP.

Table 1. Correlation of clinical variables with i-mLAP

Parameter	R	p-value
Peak troponinT	0.231	0.007
Mitral E velocity	0.087	0.310
Desceleration time	-0.163	0.057
Lateral e' velocity	-0.70	0.414
Lateral E/e'	0.194	0.023
Septal e' velocity	.050	0.564
Septal E/e'	0.178	0.037
Average E/e'	0.204	0.024
Echo-mLAP	0.187	0.038
TRV	0.162	0.050
IVRT	0.065	0.450
LVEDV	0.182	0.047
LVESV	0.191	0.040
LAVI	0.428	<0.001
LVEDP	0.940	<0.001
Pro-BNP	0.305	0.005

i-mLAP: invasively estimated mean left atrial pressures.

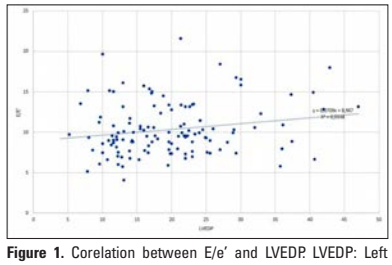

Figure 1. Corelation between E/e' and LVEDP. LVEDP: Left ventricular diastolic dysfunction.

Coronary artery disease / Acute coronary syndrome

OP-192

Interarm blood pressure differences and two-year mortality in acute coronary syndrome patients

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Background and Aim: In current guides, it is recommended to measure blood pressure in both arms at the first visit. Large differences, however, can have clinical and prognostic implications, which have been explored in various populations. In fact, interarm blood pressure difference (IABPD) has been associated with left ventricular hypertrophy, arterial stiffness, presence and severity of coronary artery disease, carotid intima media thickness, peripheral artery disease, and cardiovascular risk factors such as diabetes, hypertension and dyslipidemia. Moreover, increased IABPD was associated with increased cardio-vascular and/or all-cause mortality in several prior studies. This association was observed in various cohorts including patients with hypertension, diabetes mellitus, chronic kidney disease, and acute stroke. Previous aggregate-data meta-analyses also confirmed that higher IABPD may be related to a higher cardiovascular risk. In this study we planned to explore the association between the IABPD obtained with simultaneous measurements in both arms and the risk of mortality during a 2- year follow-up in patients with ACS.
Methods: In this multicenter - prospective cohort study, patients with acute coronary syndrome included in the study and followed for 2 years. Simultaneous blood pressure measurements were performed during initial admission. Systolic ≥10 mmHg and diastolic ≥5 mmHg absolute IABPD was defined as cut-off values in this study. The relationship of IABPD and all- cause mortality was assessed using Kaplan-Meier curves and Cox analysis.
Results: A total of 532 patients with acute coronary syndrome were included in the study. Mean age of the study participants was 60.1±12.6. Patients included in the study were followed for 23.2±7.2 months (median 25.3, min: 0, max: 28.7 months). Survival was assessed using Kaplan- Meier curves. Patients with systolic IABPD ≥10 mmHg and systolic IABPD <10 mmHg had an average survival time of 25.94±0.84 and 25.92±0.38 months (p=0.925), respectively (Figure 1b). Survival times of diastolic IABPD ≥5 mmHg and diastolic IABPD <5 mmHg were 26.44±0.62 and 25.71±0.41 (p=0.251) months, respectively (Figure 1a).
Conclusions: Routine blood pressure measurements in both arms are recommended by the guidelines. Blood pressure difference between arms can be easily obtained and has been shown to be associated with mortality in various patient populations, including acute stroke. In this study we did not find a significant association between IABPD and mortality in patients with ACS in 2- years follow-up. Future studies may be required for further evaluation of the prognostic importance of IABPD in patients with ACS or CAD.

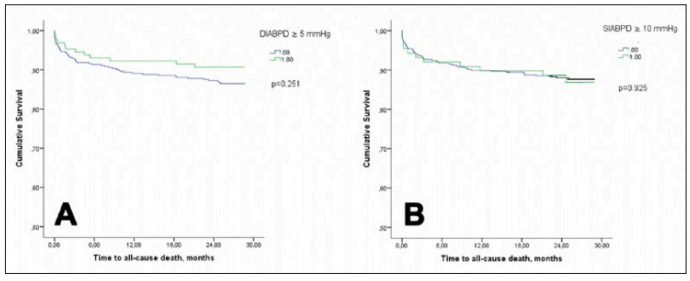

Figure 1. Kaplan-Meier survival curves for acute coronary syndrome patients according to interarm difference of diastolic **(a)** and systolic **(b)** blood pressure.

Coronary artery disease / Acute coronary syndrome

OP-193

High fructose consumption may have part in the pathophysiology of coronary artery ectasia

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Background and Aim: Ccoronary artery ectasia (CAE) is considered a variant of coronary atherosclerosis. In a study of Markis et al. an anatomical classification of CAE determined a two-year mortality rate of 15% as similar to 3-vessel coronary artery stenosis. Pathophysiology and clinical importance of CAE are not totally understood. CAE is a disease of coronary atherosclerosis variant. The pathophysiological underlying mechanisms of CAE have been already studied and however not so far understood well, including endothelial insufficiency, microvascular dysfunction, vasomotor dysfunction, increased platelet aggregation, connective tissue diseases, inflammation, and oxidative stress. Many epidemiological, clinical, and experimental studies have shown that fructose naturally found in fruit and known as fruit sugar has become the most popular sweetener in food industry and that increased fructose intake is associated with diseases such as obesity, type 2 diabetes, insulin resistance, poor glucose tolerance, hyperlipidemia, metabolic syndrome, gout, hyperuricemia, and cardiovascular diseases. In light of these findings, this study aims to determine the effects of the amount of fructose intake on isolated CAE.
Methods: The study group consists of the patients with stable angina pectoris who had coronary angiography (CAG) reports dated from December 2018 to April 2019. Based on these reports, sampling was made as follow: the patient or CAE group of 50 patients also with isolated CAE and the control group of 50 patients with normal coronary flow pattern (NCF). A comparative analysis was performed using the exact data of both groups including nutrient consumption. In order to determine the fructose consumption and nutritional status of the patients, the dietician questioned the food consumption records of the patients for three days (two weekdays and one weekend). Regarding the patients' nutrient consumption status, their daily intake of energy, macronutrients, and fructose was calculated in the Beslenme Bilgi Sistemleri 7.1 (BEBIS - Nutrition Information Systems) program and the results were evaluated.
Results: The patient group with higher high-sensitivity C-reactive protein levels (p=0.029), greater platelet count (p=0.015), and increased hypertension rate (p=0.012) were observed to have higher energy in total (p=0.008), carbohydrate (p=0.003), and fructose intake (p<0.001). Multivariable logistic regression analyses demonstrated that rising Hs-CRP levels (p=0.031), greater platelet count (p=0.017), higher fructose intake (p=0.029), and increased hypertension (p=0.032) were individually associated with CAE.
Conclusions: In the CAE group higher fructose consumption was observed and thus determined to potentially contribute to the CAE pathophysiology.

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

Parameters	Normal Coronary Artery (n=50)	Coronary Artery Ectasia (n=50)	p value
Age, years	57.0 ± 10.6	59.3 ± 11.2	0.308
BMI, kg/m2	27.0 ± 4.3	27.9 ± 4.3	0.205
Female, n (%)	21 (42.0)	16 (32.0)	0.300
Diabetes Mellitus, n (%)	9 (18.0)	15 (30.0)	0.160
Hypertension, n (%)	18 (36.0)	29 (58.0)	0.028
Dyslipidemia, n (%)	13 (26.0)	15 (30.0)	0.656
Family history, n (%)	4 (8.0)	8 (16.0)	0.218
Smoking, n (%)	16 (32.0)	22 (44.0)	0.216
Glucose, mg/dl	116.4 ± 47.6	123.5 ± 54.9	0.505
Creatinine, mg/dl	0.97 ± 0.20	1.07 ± 0.39	0.208
Uric Acid, mg/dl	5.5 ± 2.1	5.9 ± 2.2	0.825
WBC count, 10^3/mm^3	9.6 ± 2.3	10.2 ± 2.3	0.338
Hemoglobin, g/dL	13.2 ± 1.7	13.2 ± 1.7	0.113
Platelet count, 10^3/mm^3	221.0 ± 57.4	262.4 ± 60.9	0.015
Total cholesterol, mg/dL	193.0 ± 86.6	187.5 ± 83.1	0.848
Triglyceride, mg/dL	160.1 ± 78.9	176.48 ± 80.7	0.456
LDL-cholesterol, mg/dL	116.6 ± 64.7	112.7 ± 59.9	0.786
HDL-cholesterol, mg/dL	45.5 ± 28.0	45.7 ± 24.3	0.931
Hs-CRP, mg/L	3.6 ± 2.3	5.1 ± 2.3	0.029
LVEF, %	58.7 ± 5.2	56.0 ± 4.0	0.307
Diameter of ectasia (mm)	-	4.23 (3.70-5.21)	
Ectasia type			
Type I		9	
Type II		7	
Type III		5	
Type IV		29	

Data are given as mean ± SD, n or median (interquartile range). BMI, Body mass index; HDL, high density lipoprotein; Hs-CRP, high-sensitivity C-reactive protein; LDL, low-density lipoprotein; LVEF, left ventricle ejection fraction; WBC, white blood cells.

Table 2. Comparisons daily diet energy, macro nutrients and fructose consumption

Parameters	Normal Coronary Artery (n=50)	Coronary Artery Ectasia (n=50)	p value
Energy (kcal)	2472.9 ± 571.9	2807.2 ± 659.6	0.008
CHO (g)	245.5 ± 90.1	298.1 ± 106.9	0.003
CHO (TE%)	41.4 ± 8.4	42.0 ± 9.6	0.764
Protein (g)	84.4 ± 23.4	92.0 ± 28.3	0.147
Protein (TE%)	14.1 ± 3.0	13.5 ± 2.9	0.269
Lipid (g)	122.6 ± 35.7	137.7 ± 38.9	0.046
Lipid (TE%)	44.5 ± 7.7	44.3 ± 8.9	0.943
Fiber (g)	26.5 ± 8.1	27.5 ± 8.8	0.566
Fructose (g)	34.2 ± 14.8	46.4 ± 16.5	<0.001
Fructose (TE%)	5.5 ± 2.0	6.6 ± 1.7	0.005

Data are given as mean ± SD, n or median (interquartile range). CHO, carbohydrate; TE, total energy.

Table 3. Multivariate logistic regression analysis to predictig the slow coronary flow

	Univariable OR (95% CI)	p value	Multivariable OR (95% CI)	p value
Hypertension	2.455 (1.097- 5.494)	0.029	2.960 (1.100 -7.964)	0.032
Platelet count	1.011 (1.002-1.021)	0.020	1.014 (1.002-1.025)	0.017
Hs-CRP	1.136 (1.006-1.283)	0.040	1.169 (1.015-1.347)	0.031
Fructose consumption	1.051 (1.022-1.081)	<0.001	1.048 (1.005-1.092)	0.029
Total energy consumption	1.001 (1.000-1.002)	0.011	1.000 (0.999-1.002)	0.143
Total carbohydrate consumption	1.005 (1.000-1.009)	0.036	1.000 (0.993-1.007)	0.198

CI, confidence interval; Hs-CRP, high-sensitivity C-reactive protein; OR, Odds ratio.

Coronary artery disease / Acute coronary syndrome

OP-194

Determination of bleeding risk in patients with acute coronary syndrome undergoing percutaneous coronary intervention

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Background and Aim: PRECISE – DAPT score is the scoring system recommended by current guidelines to decide the duration and type of dual antiplatelet therapy of acute coronary syndromes (ACS) patients undergoing percutaneous coronary intervention (PCI). There is limited data showing the long-term bleeding risk of patients with acute coronary syndrome of PRECISE – DAPT score. In our study, we aimed to investigate the value of PRECISE – DAPT score in predicting bleeding in out-hospital long-term follow-up in patients with ACS who were undergoing PCI.
Methods: 1071 patients with ACS who underwent PCI were included in the study. PRECISE – DAPT score of patients was calculated and patients were divided into two groups as low (<25 points) and high (≥25) PRECISE – DAPT score. The bleeding seen in the follow-up was classified as major and minor bleeding.
Results: The median follow-up period is 7.3 (5.3–8.3) years. The mean PRECISE-DAPT score of the study population was 17 (12–25). Compared to men, women had a higher PRECISE-DAPT score [23 (16-31) vs 15 (11-22)]. The rate of patients with a PRECISE-DAPT score ≥25 was 44% in women and was 18.3% in men. In the long-term follow-up of patients, major bleeding was observed in 5% and minor bleeding in 6%. 3% of major bleeding was BARC type 3a, 1% was BARC type 3b, 1% was BARC type 3c - 5. There was no difference in the rate of bleeding in terms of sex during long-term follow-up. In the ROC curve analysis, AUC of PRECISE - DAPT score in predicting long-term bleeding was found to be 0.609 (0.515-0.703, p<0.05).
Conclusions: The PRECISE – DAPT score was not very strong in determining long-term bleeding in ACS patients underwent PCI. Although the women had higher PRECISE - DAPT score compared with men, there was no difference between groups in the rate of bleeding during long-term follow-up. This may be due to the use of less potent agent clopidogrel as part of dual antiplatelet therapy in our clinic.

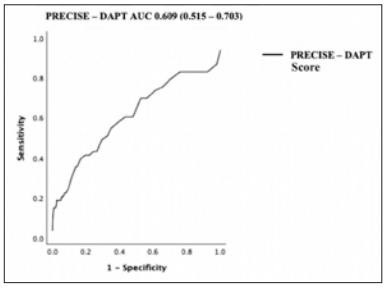

Figure 1. PRECISE - Receiver operating characteristic (ROC) curve for DAPT score.

Table 1. Demographic data of patients according to PRECISE - DAPT score

Variables	PRECISE – DAPT Score ≥ 25 n = 271	PRECISE – DAPT Score < 25 n = 800	p – value
Age (Year)	70.3 ± 10.7	59.6 ± 10.3	<0.001
Sex			<0.001
Female n (%)	129 (48)	164 (21)	
Male n (%)	142 (52)	636 (80)	
Diabetes Mellitus n (%)	101 (41)	167 (24)	<0.001
Hypertension n (%)	156 (63)	353 (49)	<0.001
Heart Failure n (%)	7 (3)	9 (2)	0.243
Coronary Artery Disease n (%)	77 (32)	175 (25)	0.025
Cerebrovascular Disease n (%)	22 (9)	19 (3)	<0.001
Dyslipidemia n (%)	47 (20)	166 (24)	0.178
Bleeding History n (%)	27 (10)	0 (0)	<0.001
Smoking n (%)	46 (19)	339 (49)	<0.001
Multi-vascular Disease n(%)	230 (85)	610 (76)	0.003
Diagnosis			0.037
STEMI n (%)	139 (51)	470 (59)	
NSTEMI n (%)	94 (35)	213 (27)	
UAP Sayı (%)	38 (14)	117 (15)	
Infarct Relevated Artery			0.909
LAD n (%)	115 (42)	356 (45)	
LCX n (%)	47 (17)	130 (74)	
RCA n (%)	89 (33)	251 (31)	
Others (Side branch, Saphenous graft, Left main) n (%)	20 (7)	63 (8)	
Stent type			0.397
Bare Metal Stent n (%)	212 (89)	511 (90)	
Drug Eluting Stents (%)	17 (7)	45 (8)	
Both n (%)	10 (4)	14 (3)	
Use of Thrombus Aspiration Device n (%)	37 (14)	111 (14)	0.927
Use of GP IIb/IIIa Inhibitors n (%)	70 (26)	227 (29)	0.419
Discharge Therapy			
Acetylsalicylic Acid n (%)	271 (100)	800 (100)	1
Clopidogrel n(%)	259 (96)	779 (97)	0.138
Ticagrelor – Prasugrel n (%)	12 (4)	21 (3)	0.138
ACEI – ARB n (%)	177 (71)	677 (89)	<0.001
Beta Blocker n (%)	36 (14)	251 (88)	<0.001
Statin n (%)	196 (79)	649 (85)	0.021
Spironolactone n (%)	17 (7)	47 (7)	0.837
Furosemide n (%)	42 (18)	54 (8)	<0.001
PPI n (%)	130 (60)	312 (60)	0.926

STEMI: ST elevation myocardial infarction, NSTEMI: Myocardial infarction without ST elevation, UAP: Unstable angina pectoris, LAD: Left anterior descending coronary artery, LCX: Left circumflex coronary artery, RCA: Right coronary artery, ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, PPI: Proton pump inhibitor.

Table 2. Laboratory data of patients according to PRECISE - DAPT score

Variables	PRECISE – DAPT Score ≥ 25 n = 271	PRECISE – DAPT Score < 25 n = 800	p – value
Hemoglobin (g/dL)	12.3 ± 2.2	14.1 ± 1.6	< 0.001
Anemia n (%)	120 (44.3)	80 (10)	< 0.001
Leukocyte (10^3/uL)	11.5 ± 4.3	11.3 ± 4	0.507
GFR (ml/dk)	60.5 ± 25.1	91.6 ± 20.3	< 0.001
GFR < 60 (ml/min) n (%)	140 (51.7)	32 (4.0)	< 0.001
Creatinin† (mg/dL)	1.14 (0.87 – 1.44)	0.86 (0.76 – 0.98)	< 0.001
Total cholesterol (mg/dL)	166 ± 45.8	181 ± 40	<0.001
Triglycerides † (mg/dL)	131 (98 – 181)	148 (105 – 199)	0.030
LDL (mg/dL)	109 ± 34	112 ± 33	< 0.001
HDL (mg/dL)	35.5 ± 9.5	35.5 ± 9	0.940
Glucose (mg/dL)	138 ± 73	124.5 ± 56	0.005
EF (%)	47 ± 11	49 ± 10	0.030
PRECISE – DAPT Score†	30 (27 – 38)	14 (11 – 19)	< 0.001

† – Since these parameters are not normally distributed, they are expressed as median interquartile range (25th and 75th percentiles). Abbreviations: GFR: Glomerular filtration rate. LDL: Low density lipoprotein. HDL: High density lipoprotein. EF: Ejection fraction.

Table 3. Long-term bleeding, mortality and major cardiac event rates of patients according to the PRECISE - DAPT score

Variables	PRECISE – DAPT Score ≥ 25 n = 271	PRECISE – DAPT Score < 25 n = 800	p – value
Bleeding			<0.001
Major Bleeding n (%)			
BARC Type 3a	13 (5)	21 (62)	
BARC Type 3b	5 (56)	4 (44)	
BARC Type 3c – 5	5 (2)	5 (0.6)	
Minor Bleeding n (%)			
BARC Type 1	5 (2)	4 (0.5)	
BARC Type 2	21 (8)	34 (62)	
Bleeding Types			0.014
Gingival bleeding n (%)	1 (0.4)	1 (0.1)	
Epistaxis n (%)	7 (3)	10 (1)	
Limb Hematoma n (%)	0 (0)	1 (0.1)	
GI Bleeding n (%)	21 (8)	31 (4)	
Hematuria n (%)	9 (3)	13 (2)	
Hemoptysis n (%)	3 (1)	3 (0.4)	
Cranial (Parenchymal) Bleeding n (%)	4 (2)	5 (0.6)	
Conjunctival Bleeding n (%)	1 (0.4)	1 (0.1)	
In-Ear Bleeding n (%)	1 (0.4)	0 (0)	
Retroperitoneal Bleeding Sayı (%)	0 (0)	1 (0.1)	
Subarachnoid Hemorrhage n (%)	1 (0)	0 (0)	
Vaginal Bleeding n (%)	1 (0.4)	2 (0.3)	
Recurrent Myocardial Infarction n (%)	32 (12)	98 (12)	0.847
Hospitalization with Heart Failure n (%)	17 (6)	26 (3)	0.028
Recurrent Cerebrovascular Incident n (%)	14 (5)	24 (3)	0.096
Recurrent Revascularization n (%)			
PCI n (%)	34 (13)	118 (15)	0.396
CABG n (%)	7 (3)	30 (4)	0.363
Total Mortality n (%)	108 (40)	109 (13)	<0.001

BARC: The bleeding academic research consortium, GI Bleeding: Gastrointestinal bleeding, PCI: Percutaneous coronary intervention, CABG: Coronary artery bypass graft.

Coronary artery disease / Acute coronary syndrome

OP-196

The relationship between thiol/disulfide homeostasis and pre-or post-interventional TIMI flow in patients who underwent coronary angiography with a diagnosis of ST segment elevation myocardial infarctus

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Background and Aim: The patency of the infarct-related artery (IRA) before and after percutaneous coronary intervention (PCI) is associated with better clinical outcome and mortality in ST-elevation myocardial infarction (STEMI). In this study, we evaluated the pre- and post-interventional relationships between IRA patency and thiol/disulfide homeostasis in patients who presented with STEMI and underwent PCI.
Methods: A total of 176 patients with STEMI who had applied to the hospital within 2 hours following pain development and underwent PCI were prospectively enrolled in this study. The pre- and post-intervention (PrI and PoI, respectively) IRA patency values were determined according to TIMI (Thrombolysis in Myocardial Infarction) flow grades. Pre- and post-interventional TIMI flow grades of 0, 1 and 2 were defined as non-patent IRA, while a TIMI flow grade of 3 was defined as patent IRA. The thiol/disulfide homeostasis was

determined from blood drawn at first application to our center.

Results: The IRA was patent in 71 (40.3%) pre-interventional and 153 (86%) post-interventional measurements. Female sex was significantly more frequent in those with non-patent IRA at PrI (p=0.048). The levels of ejection fraction (p=0.043), albumin (p=0.004), hemoglobin (p=0.004), native thiol (p<0.001), total thiol (p<0.001) and native/total thiol ratio (p=0.001) were found to be significantly higher in those with patent IRA at PrI compared to those with non-patent IRA at PrI; whereas age (p=0.011), fasting blood glucose (p=0.002), white blood cell count (p=0.016), red blood cell distribution width (p=0.044), disulfide/native thiol ratio (p<0.001) and disulfide/total thiol ratio (p<0.001) were significantly lower. When those with patent IRA at PoI and those with non-patent IRA and PoI were compared, we found that the levels of ejection fraction (p=0.013), albumin (p=0.003), native thiol (p<0.001), total thiol (p<0.001) and native thiol/total thiol ratio (p=0.023) were significantly higher in patients with patent IRA at PoI; whereas age (p=0.015), lactate dehydrogenase (p=0.012), disulfide (p=0.021), disulfide/native thiol ratio (p=0.001) and disulfide/total thiol ratio (p=0.003) were significantly lower. Multiple regression analysis revealed that native thiol levels were independently and significantly associated with IRA patency at PrI (OR: 1.08, 95% CI: 1.05-1.11, p<0.001) and PoI (OR: 1.02, 95% CI: 1.01-1.03, p<0.001).

Conclusions: The thiol/disulfide homeostasis and especially native thiol levels, as oxidative stress markers, may be utilized to predict pre- and post-interventional IRA patency in patients with STEMI.

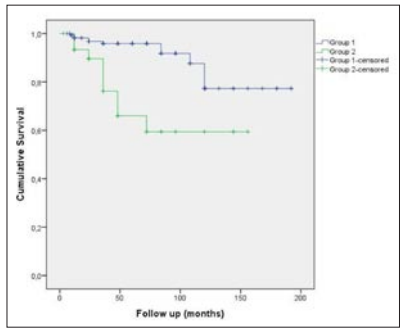

Figure 1. ROC curve for cardiac mortality.

Coronary artery disease / Acute coronary syndrome

OP-198

Predictors of type 4C myocadial infarction in previously diagnosed with in-stent restenosis

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Background and Aim: In-stent restenosis(ISR) in one the limiting factor of stent efficacy and drug-eluting stents were developed to lower the risk of ISR. However, ISR still compromises patients' outcomes. Fourth universal definition of myocardial infarction guidelines provided a new type of myocardial infarction as type 4C which state that MI due to stent restenosis. To the best of our knowledge, there is no demonstrated risk factor for the development of type 4C myocardial infarction. In this study, we aimed to investigate the predictors of type 4C myocardial infarction.

Methods: We retrospectively investigated patients with a diagnosis of ISR which is demonstrated with invasive coronary angiography. Patients' comorbidities, medications and stent properties in terms of drug-eluting stent (DES) and bare-metal stents (BMS), stent diameter and stent length were recorded. The time interval between stent implantation and diagnosis of ISR were also recorded. Follow-up time was calculated from the diagnosis of ISR to date. Patients in whom stent implantation or drug-eluting balloon angioplasty performed to treat ISR were excluded from study. Type 4C myocardial infarction was diagnosed as recommended by the 4th universal definition of myocardial infarction guidelines which state that myocardial infarction in patient with ISR without ant other reason which may be cause of MI.

Results: We included 189 patients with diagnosis of ISR. The mean time between stent implantation and diagnosis of ISR was 14 months. Mean follow-up time was 40±13 months. 79 patient underwent to revascularization and 43 revascularizations were performed for the treatment of ISR. Among 43 revascularization, 28 procedures were performed with the diagnosis of type 4C MI (group1). 15 patients who underwent to revascularization of ISR were in a stable clinical situation and aslo excluded from analysis. There was no significant difference between groups in terms of gender (p=0.338), age (p=0.479), diabetes mellitus (p=0.121) and previous myocardial infarction (p=0.195). Chronic renal failure was significantly higher in group 1 patients, there was no significant difference between groups with respect to medications. Cardiac mortality was significantly higher in patients with type 4C MI. There was no significant difference between groups in terms of stent types (BMS vs DES) (p=0.715). Multivariate analysis revealed that none of the risk factors is associated with type 4C myocardial infarction.

Conclusions: Our results indicated that none of the coronary artery disease risk factors is associated with type 4C myocardial infaction. Considering the difference between underlying pathophysiology of type 4C MI and type 1 MI, these results are not suprising. Futher studies for the definition of risk factors of type 4C myocardial infarction are necessary to define patient under risk.

Table 1. Logistic regression analysis

Variable	odds ratio	95% CI	p-value
Age	1.011	0.963 – 1.061	0.670
Gender	0.423	0.139 – 1.286	0.129
HT	1.122	0.186 – 6.772	0.900
DM	0.232	0.045 – 1.201	0.082
CVA	0.091	0.007 – 1.195	0.068
CKD	0.190	0.020 – 1.776	0.145
HL	0.336	0.060 – 1.871	0.213
Smoking	0.121	0.017 – 0.892	0.038*
Previous AMI	0.199	0.036 – 1.102	0.064
Family history	0.484	0.083 – 2.830	0.420
Stent type (DES)	2.129	0.792 – 5.726	0.134
Stent length	1.191	0.432 – 3.279	0.736
Stent diameter	0.704	0.270 – 1.835	0.473

Logistic regression analysis

Table 2. Logistic regression analysis

Variable	odds ratio	95% CI	p-value
Age	1.011	0.963 – 1.061	0.670
Gender	0.423	0.139 – 1.286	0.129
HT	1.122	0.186 – 6.772	0.900
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Logistic regression analysis

Coronary artery disease / Acute coronary syndrome

OP-199

The role of intestinal microbial metabolism in the development of coronary slow flow phenomenon

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Background and Aim: Coronary slow flow phenomenon may be responsible for processes that can lead to sudden death from recurrent chest pain. Diagnosis is made coronary angiographically and there is no known treatment method. Therefore, it is important to prevent the development of this phenomenon or find treatment modality. Intestinal microbial metabolism plays a major role in the balance of the whole organism. A problem in the functioning of this metabolism initiates inflammation systemically and causes the development of various diseases. It has been shown to be responsible for a variety of diseases, chronic kidney disease, diabetes mellitus, cardiovascular diseases. One hypothesis that was not previously included in the literature is that intestinal microbial metabolism plays a role in the development of coronary slow flow phenomenon.

Methods: In order to investigate the accuracy of this hypothesis, in this study, 40 patients with elective indications for coronary angiography were performed and a normal coronary artery was detected, and 40 patients with a coronary slow flow phenomenon were compared. Of these patients, those with impaired liver function, chronic inflammatory diseases, active infections and those using antibiotics were excluded.

Results: When the gender distribution of these patients is examined, 15 (37.5%) of the patients in the control group are male and 22 (55%) of the patients in the slow flow group are male (p=0.1). While 10 (25%) of the patients in the control group smoke, 16 (55%) patients in the slow flow group smoke (p=0.2). While the mean age was 57.2±10.2 in the control group, it was 53.1±13.7 in the slow flow group (p=0.1). Patients with hypertension were observed as 22 (55%) in the control group and 15 (37.5%) in the slow flow group (p=0.1). No statistically difference was found between the slow flow group 7 (17.5%) and the control group 6 (15.0%) for the frequency of diabetes (p=0.7). When the laboratory values were compared, no statistically significant difference was found between the two groups. When blood Trimetilamin N-oxid (TMAO) enzyme levels of both groups were compared, it was found that these enzyme levels were significantly higher in patients with coronary slow flow phenomenon (p=0.02). While the mean TMAO in the slow flow group was 62.03±18.89 ng/ml, the average of TMAO in the control group was 52.87±17.28 ng/ml.

Conclusions: Consequently, intestinal microbial metabolism may play a role in the development of coronary slow flow phenomenon. Normal functioning of intestinal microbial metabolism may prevent the development of coronary slow flow phenomenon. Multicenter and comprehensive studies should be carried out in order to approach and show clearer and definitive results in this field.

Coronary artery disease / Acute coronary syndrome

OP-200

Telomerase activity and hTERT gene expression in coronary artery disease

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Background and Aim: Telomeres are tandem repeats of specific DNA sequences located at the ends of chromosomes, which maintain genomic stability and integrity. Telomeres gradually shorten with each cell division, and therefore telomere length is considered as a marker of aging. In this study, we aimed to examine the telomerase concentration as a measure of telomerase activity (TA) and hTERT gene expression in patients with stable coronary artery disease (SCAD) and with acute coronary syndrome (ACS) and compare to subjects with controls.

Methods: The study included 211 patients (78 ACS and 71 SCAD patients) aged between 55 and 75 who underwent coronary angiography. The age range was kept limited in order to minimize the age effect on the telomere. Telomerase concentration was measured by ELISA and used to determine telomerase activity. hTERT gene expression was determined by Real-Time PCR.

Results: Mean age was 63.4±6.1 in the ACS group, 63.4±6.4 in the SCAD group, and 61.7±5.6 in the control group (p=0.200). Seventy three percent (57) of the ACS patients and 76% (54) of the SCAD patients were male (p=0.677). Forty percent (25) of the patients in the control group were male (p=0.001). Serum telomerase

enzyme concentration was lower in ACS (36.61±1.54) and SCAD (36.79±1.57) when compared to the control group (37.03±2.25); however, this difference did not reach statistical significance (p=0.890). hTERT gene expression acting in the telomerase enzyme synthesis was 2.7 -fold lower in ACS group (p=0.070) and 2.2-fold lower in the SCAD group (p=0.101) compared the control group (Table 1).
Conclusions: In the current study, telomerase activity or hTERT expression were similar in patients with ACS and SCAD, and controls.

Table 1. Telomerase enzyme concentration and hTERT gene expression results

	ACS (n=78)	SCAD (n=71)	CONTROL (n=62)	p-value
Telomerase enzyme concentration (ng / ml)	36.61 ± 1.54	36.79 ± 1.57	37.03 ± 2.25	0.890
Fold-regulation of hTERT gene compared to the controls	-2,7064	-	-	0.070
Fold-regulation of hTERT gene compared to the controls	-	-2,2896	-	0.101

Coronary artery disease / Acute coronary syndrome

OP-202

Relationship between magnesium / phosphate ratios and endothelial function in coronary artery disease, a prospective trial

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Background and Aim: The pathogenesis of atherosclerotic changes and endothelial dysfunction is complex and multifactorial. Magnesium and phosphate minerals, which are electrolytes in the blood, are important minerals in the pathophysiology of atherogenesis and endothelial dysfunction. It has been suggested that modification or reversal of endothelial dysfunction may provide significant therapeutic benefit in the treatment of coronary artery disease. In our study, we aimed to evaluate the relationship between magnesium / phosphate ratios and endothelial functions in patients with coronary artery disease.
Methods: 61 patients who had coronary artery disease documented by coronary angiography were included in the study. Endothelial functions were evaluated by flow-mediated vasodilation (FMD) test.
Results: The mean age of 61 patients included in the study was 61.2±10.1 years, and 72.1% of the patients were male and the mean body mass index was 27.8±5.4 kg/m². The average Mg / P ratio of the patients included in the study is 0.61 (±0.13), and the median value is 0.62 (minimum 0.20 - maximum 1.03). In the FMD test, the mean radial artery basal diameter was 25.21±2.8 mm, and the mean radial artery diameter after FMD was 28.33±3.2 mm. A weak correlation was observed between magnesium / phosphate ratios and the percentage change in artery diameter that showing endothelial functions (r=0.268, p=0.037).
Conclusions: In patients with coronary artery disease, low magnesium / phosphate ratios are an independent factor in endothelial dysfunction.

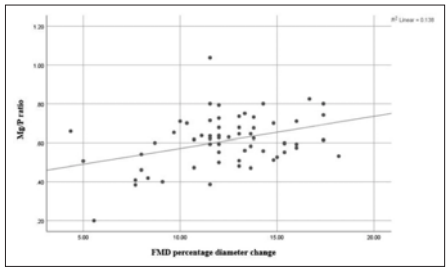

Figure 1. The relationship between Mg/P ratio and FMD percentage diameter change. A weak possitive correlation was observed between the magnesium / phosphate ratios and the percentage change in artery diameter that showing endothelial functions (r=0.268, p=0.037).

Table 1. Demographic and clinical data of patients

Demographic and clinical features	n= 61
Age, year	61.2±10.1
Male sex, n (%)	44 (72.1)
BMI, kg/m2	27.8±5.4
Systolic BP, mmHg	141.7±18.4
Heart rate, /min	77.5±18.1
NYHA class I, n (%)	35 (57.4)
Chest pain, n (%)	32 (52.5)
Dyspnea, n (%)	22 (36.1)
Smoking, n (%)	16 (26.2)

**BMI: body mass index; BP: blood pressure; NYHA: New York Heart Association.*

Table 2. Comorbid states and laboratory findings of the patients

Hypertension, n (%)	49 (80.3)
Diabetes, n (%)	25 (41.0)
Hyperlipidemia, n (%)	24 (39.3)
Anemia, n (%)	6 (9.8)
Urea, mg / dl	36.30 (± 18.33)
Hemoglobin, g / dl	13.43 (± 2.22)
LDL, mg / dl	100.81 (± 38.76)
Mg, mg / dl	1.99 (± 0.28)
P, mg / dl	3.36 (± 0.54)
Mg / P	0.61 (± 0.13)
LVEF, %	52.8 (± 9.7)
LA, cm	3.9 (± 0.7)

LDL: low density lipoprotein; Mg: magnesium, P: phosphate; LVEF: left ventricular ejection fraction; LA: left atrium.

Table 3. Flow-mediated vasodilation test

FMD test	Basal diameter	Diameter after FMD	Percentage change after FMD	P value
Mean (± standard deviation) mm	25.21 (±2.8)	28.33 (±3.2)	12.34 (±3.03)	<0.001

Coronary artery disease / Acute coronary syndrome

OP-203

Relationship between prognostic nutritional index and long term mortality in ST-elevation myocardial infarction patients undergoing emergent coronary artery bypass graft surgery

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Background and Aim: Correlation between malnutrition and prognosis in patients with coronary artery disease (CAD) has been reported. However prognostic significance of nutritional status in patients with ST elevation myocardial infarction (STEMI), stable CAD and elective coronary artery bypass graft (CABG) surgery were evaluated, to date the prognostic impact of poor nutritional status on STEMI patient who underwent emergent CABG was not investigated. Our aim is to evaluate the relation between nutritional status assessed by the prognostic nutritional index (PNI) and long term mortality in STEMI patients who underwent emergent CABG. To the best of our knowledge our study is the first one which evaluated PNI effect on this specific population.
Methods: 88 consecutive patients with STEMI, who did not qualify for primary PCI and required emergent CABG between 2010 and 2017 were included to our study. Study population was divided into two groups as survivors and non-survivors. The PNI was calculated as 10 × serum albumin (g/ dl) + 0.005 × total lymphocyte count (per mm3) for both groups, using the preoperative data. Optimal cut-off value was obtained by receiver operating characteristic (ROC) analysis. According to the cut-off value we investigated the relationship between PNI and the long term mortality.
Results: The mean age of the study population was 56.1±11.3. During median 92.8 (69.0-105.1) months follow up, 23 of 88 patients (26%) died. The mean PNI was 48.8±8.6. In mortality group PNI (44.8±7.0 vs 50.2±8.7, p=0.018) was significantly lower than survivor group. Cut-off value was 50.63, calculated by ROC analysis. Regression analysis showed the significant association between PNI and long term mortality (95% confidence interval: 0.943 (0.894-0.993) p=0.027. Mortality incidence was higher in lower PNI group (Figure 1). Also age, ejection fraction, glucose level, glomerular filtration rate, Killip classification, LAD - LIMA graft usage, LAD - saphenous graft usage were found to be associated with long term mortality.
Conclusions: The PNI was significantly associated with long-term mortality in patients with STEMI who underwent emergent CABG. PNI may improve the accuracy of risk assessment of STEMI patients undergoing emergent CABG.

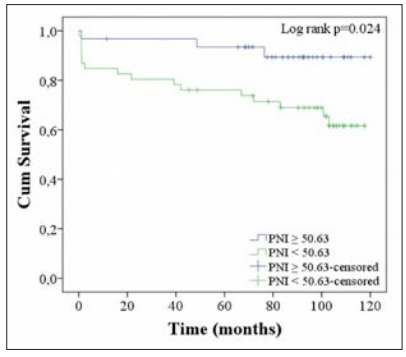

Figure 1. Kaplan Meier curve for long term mortality stratified by PNI.

Coronary artery disease / Acute coronary syndrome

OP-204

The relation of high levels of procalcitonin with lesion severity in non ST segment elevation myocardial infarction patients

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Background and Aim: Coronary angiography of non ST segment elevation myocardial infarction (NSTEMI) patients not always result with a critical stenosis limiting the coronary blood flow. High levels of inflammatory markers are shown to be related with progressive atherosclerosis, bad prognosis in acute coronary syndrome and increased number of coronary interventions. In the literature, procalcitonin was found to be in relation with high levels of inflammatory response and bad prognosis in acute coronary syndrome. However, its correlation with lesion severity has not yet been studied. We aimed to investigate the possible relation of levels of procalcitonin with the angiographic lesion severity.
Methods: We investigated the possible relation between admission procalcitonin levels with coronary lesion severity in 254 NSTEMI patients admitted to our clinic, retrospectively. Also, laboratory values at admission was studied by regression analysis for their independent effects on the severity of the lesion.

Results: 146 patients had severe coronary lesion (≥50%) and 108 patients non severe lesion. Mean age of the patients were 63 and 32% were female. Admission levels of CRP and procalcitonin were significantly high in severe lesion group (p=0.002 and p=0.02, respectively). Admission thrombocyte level was significantly low and mean platelet volume (MPV) was significantly high in the severe lesion group (p=0.02 and p=0.005, respectively). In the regression analysis, high procalcitonin levels, CRP and MPV values were found to be independent predictors of severe lesion (p=0.022, OR: 1.938 CI [1.098 -3.421], p=0.033, OR: 1.019 CI [1.002-1.036], p=0.030 OR: 1.342[1.029-1.752], respectively).
Conclusions: In our trial, we found that high levels of procalcitonin at admission was an independent predictor of severe coronary artery lesion at NSTEMI patients. High levels of procalcitonin may be used as a marker to define high levels of inflammation and high risk group of atherosclerotic patients.

Coronary artery disease / Acute coronary syndrome

OP-205

Effects of ATRIA score and CHAD2S2 VASc score on one-year mortality in patients with acute coronary syndrome

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Background and Aim: CHA2DS2-VASc and ATRIA scores are a valid,practical method for the risk classification of thromboembolic complications due to atrial fibrillation. In this study, it was aimed to evaluate the use of CHA2DS2-VASC and ATRIA scores in predicting one-year mortality in patients with acute coronary syndrome.
Methods: Between Jan 2018 and Jan 2019, 399 patients who were followed up with a diagnosis of acute coronary syndrome were included in the study. Baseline demographic characteristics, ATRIA score and CHA2DS2-VASC score were calculated. A year later, it was investigated whether the patients survived from the death notification system.The scores of the patients who died were compared with those of the patients who died.Kaplan-meier and cox regression analysis were performed for ATRIA score and CHAD2S2 VASc score.
Results: Out of a total of 399 patients, 52 (13%) patients died after one year. While the ATRIA score was 7.7±1.1 in patients who died, it was 2.81±2.37 in living patients (p<0.001). While CHAD2S2 VASc score was 3.9±0.9 in patients who died, it was 1.84±1.36 in living patients (p<0.001). The most significant value in cox regression analysis is ATRIA score. (Odds Ratio: 2.92, P value: <0.001) In the roc analysis, the sensitivity for ATRIA score is 96.08% and the specificity is 84.38%. The sensitivity for the CHAD2S2 VASc score is 98.08% and the specificity is 70.32%.
Conclusions: CHA2DS2-VASC and ATRIA scores are scoring systems that can be easily calculated. The specificity of the ATRIA score was higher than the CHAD2S2 VASc score. In patients with acute coronary syndrome, these scoring systems can be used to determine the one-year mortality.

Table 1. Demographic, clinical and laboratory features of the patients

	Not death (n=347)	death (n=52)	P value
Age, years	61.5±12.3	71.5±10	<0.001
Female, n (%)	70 (20.2)	20 (38.5)	0.007
Male, n (%)	277 (79.8)	32 (61.5)	0.007
Diabetes mellitus, n (%)	85 (24.1)	20 (38.5)	0.042
Hypertension, n (%)	163 (47)	36 (69.2)	0.003
Hyperlipidemia, n (%)	69 (19.9)	11 (21.2)	0.853
Smoking, n (%)	181(52.2)	11(21.2)	<0.001
Weight (kg)	74±15	68±14	0.565
Height (cm)	165±8	160±8	0.557
Peak CK MB	142±155	207±278	0.015
Peak Troponin	5.1±31.3	4.3±3.8	0.850
Ejection fraction	45.7±9.7	38±9.1	<0.001
LA diameter	36±4.5	38±4.7	0.012
Atria Score	2.81±2.37	7.7±1.1	<0.001
Mehran Score	5.0±3.4	11±3.1	<0.001
CHADS-VASc score	1.84±1.36	3.9±0.9	<0.001
Syntax score	13.6±8.1	16.3±8.1	0.027
Creatinine (mg/dL)	1.14±0.61	1.17±0.58	<0.001
Opak Miktarı	131±42	157±43	<0.001
CHADS-VASc score, n (%)	194 (55.9)	51 (98.1)	<0.001
NSTEMI, n (%)	121 (34.9)	20 (38.5)	0.642
STEMI, n (%)	226 (65.1)	32 (61.5)	0.642
β Blocker, n (%)	327 (94.2)	48 (92.3)	0.536
Calcium antagonist, n (%)	14 (4)	2 (3.8)	1.0
ACEI, n (%)	278 (80.1)	38 (73.1)	0.271
ARB, n (%)	24 (6.9)	6 (11.5)	0.256
Statin, n (%)	340 (98)	52 (100)	0.602
Aspirin, n (%)	346 (99.7)	52 (100)	1.0
Klopidogrel, n (%)	285 (82.1)	46 (88.5)	0.325

ACEI:Angiotensin-converting enzyme inhibitors,ARB:Angiotensin receptor blockers, LA: Left Atrium

Table 2. Independent markers for one-year mortality

					P VALUE	b	CI	
atrıason	1,072	0,126	72,150	1	0,000	2,920	2,280	3,739
ekoEF	-0,027	0,016	2,786	1	0,095	0,974	0,944	1,005
LAcapi	-0,066	0,032	4,297	1	0,038	0,936	0,880	0,996
peakCKMB	0,001	0,001	4,958	1	0,026	1,001	1,000	1,002
cins	-1,992	0,416	22,910	1	0,000	7,3286	3,242	16,56

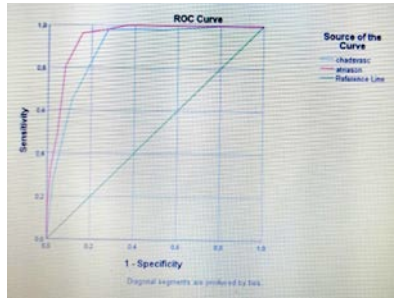

Figure 1. ATRIA score and CHAD2S2 VASc score roc analysis.

Coronary artery disease / Acute coronary syndrome

OP-206

Low superoxide dismutase and catalese is associated malondialdehyde and ischemia modified albumin in patients with non-ST elevated myocardial infarction

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Background and Aim: Acute coronary syndrome is a manifestation of cardiac ischemia and results in myocardial injury and necrosis in line with the duration of ischemia. Excessive production of Reactive Oxygen Species (ROS) is proposed to mediate ischemia-reperfusion injury. This study aimed to assess the IMA (ischemia modified albumin), MDA (malondialdehyde), SOD (superoxide dismutase), and catalase in patients with non-ST elevated myocardial infarction (NSTEMI).
Methods: The present study included 55 patients with NSTEMI and 55 healthy subjects prospectively. IMA, MDA, SOD, and catalase levels were measure from venous blood obtained from each patient within three h after the onset of symptoms. Angiography was performed within three days after the hospitalization. Significant coronary artery lesions were determined.
Results: IMA (3.14±0.06 vs. 1.49±0.03) and MDA (3.14±0.06 vs. 1.49±0.03) were higher, and SOD (1.10±0.03 vs. 2.31±0.02) and catalase (0.54±0.02) vs. 0.22±0.02) were lower in NSTEMI patients than control subjects. There was a significant correlation among IMA, MDA, SOD and catalase. Moreover, IMA values correlated positively with the multiple coronary lesions (r=-0.339 p=0.011; r=0.329 p=0.014). There was no significant correlation among the MDA, SOD, catalase and affected coronary vessel numbers.
Conclusions: Our data reveal that levels of MDA and IMA were increased, and SOD and catalase levels were decreased significantly in patients with NSTEMI.

Table 1. The demographic and clinical data of the study population

	NSTEMI (n=55)	Control (n=55	p
Age (years)	63.6±12.6	46.9±9.3	<0.001
Male/Female n	25.9±3.3	25.5±2.6	0.51
Hypertension n(%)	24(44)	15(27)	0.07
Troponin (ng/ml)	3.7(1.6-22.0)	0.016±0.005	<0.001
Hemoglobin (g/dL)	14.3±1.7 1	13.2±2.0	0.002
IMA(U/ml)	1.8±0.3	0.9±0.1	<0.001
MDA (μmol/L)	3.14±0.06	1.49±0.03	<0.001
SOD (U/ml)	3.14±0.06	2.31±0.02	<0.001
Catalase (U/ml)	0.22±0.02	0.54±0.02	<0.001

IMA (ischemia modified albumin), MDA (malondialdehyde), SOD (superoxide dismutase).

Coronary artery disease / Acute coronary syndrome

OP-207

Relationship between the prognostic nutritional index and clinical outcomes in Patients Aged 65 years with non-ST-segment elevation myocardial infarction

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Background and Aim: Acute coronary syndrome is an important cause of mortality worldwide and the frequency of coronary artery disease increases in elderly patients. The prognostic nutritional index that shows a relationship with clinical outcomes in patients with ST-segment elevation myocardial infarction or heart failure was developed to assess nutritional status based on serum albumin and total lymphocyte count. We aimed to investigate whether the prognostic nutritional index on admission was associated with mortality in older patients with Non-ST-segment elevation myocardial infarction.
Methods: The study population included 336 patients clinically diagnosed with NSTEMI at the aged ≥65 within undergoing PCI between February 2019 and February 2020. Patients were divided into two groups according to the primary endpoint.
Results: There were significant differences between the groups: PNI were significantly lower compared with those without a primary endpoint. Multivariable logistic regression analysis, PNI was an independent predictor of the primary endpoint and all-cause death in NSTEMI patients at the age of older 65.
Conclusions: This study showed that lower PNI level is an independent predictor of adverse cardiovascular outcomes and all-cause death in NSTEMI patients at the age of older 65.

Coronary artery disease / Acute coronary syndrome

OP-208

The relationship between myocardial injury and laboratory parameters and comorbid diseases in hospitalized COVID-19 patients

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Background and Aim: SARS-CoV2 enters human cells via the human angiotensin-converting enzyme 2 (ACE2) receptor, which is highly expressed by type 2 alveolar cells in the lung. Thus, lungs are the principal target and the most important cause of mortality in COVID-19 disease. Although myocardial cells scarcely express ACE 2 receptors under physiologic conditions, the situation changes in case of the presence of cardiovascular and renal diseases. The precise mechanism(s) by which SARS-CoV2 causes myocardial injury and chronic kidney disease (CKD) are yet to be elucidated.
The primary objective of this study was to investigate the effects of myocardial infarction with chronic kidney disease comorbidity on mortality and survival in hospitalized COVID-19 patients.
Methods: The present study was carried out according to international agreements (Declaration of Helsinki and World Medical Association). The study protocol was approved by our Hospital's Clinical Studies Ethics Committee. This study was a retrospective analysis of consecutive patients who were admitted either to the general hospital ward or to the intensive care unit of our hospital with the diagnosis of Covid19 and were followed up to death or discharge from the hospital before March 15, 2020. All patients with PCR-based COVID-19 diagnosis were enrolled. Patients who were below 18 years-old, who did not have PCR-based COVID-19 diagnosis, and who did not have serum troponin measurements were excluded from the study. Demographic characteristics, comorbid conditions, laboratory values, including hemogram parameters, serum creatinine, C-reactive protein, albumin, and high-sensitivity troponin I were extracted from the electronic database of the hospital and recorded by two investigators after cross-check.
Results: A total of 300 patients (135 male (55.0%), mean age 57.4±14.2 years), were included in this retrospective study. The percentages of patients who had hypertension, congestive heart failure, and chronic kidney disease were 45%, 5%, and 12%, respectively. The median serum troponin I value was 3.6 ng/mL (IQR, 1.5-14.8). Seventy-five patients (25.0%) had acute myocardial injury. Patients who developed myocardial injury were significantly older than patients without myocardial injury (mean ages 63±11.96 vs 55.3±14.2 years, respectively). In the myocardial injury group, coronary artery disease, hypertension, chronic kidney disease, and congestive heart failure were significantly more frequent compared with the patients without myocardial injury. Median NLR was also significantly higher in patients with myocardial injury compared to patients without myocardial injury. Only hypertension and CKD were significantly more common among the deceased compared with surviving patients.
Conclusions: Myocardial injury was not uncommon among hospitalized patients with COVID-19. One-fourth of all patients had myocardial injury. The presence of underlying chronic kidney disease and hypertension effect in-hospital mortality.

Table 1. Clinic-demographic characteristics and baseline laboratory values of the study subjects

Parameters	All Patients (n=300)
Age (years) (mean ± SD)	57.4±14.2
Sex (n (%))	
Female	135 (45.0%)
Male	165 (55.0%)
Comorbidities (n (%))	
Coronary artery disease	48 (16%)
Hypertension	135 (45%)
Diabetes mellitus	90 (30%)
Chronic kidney disease	36 (12%)
Congestive heart failure	15 (5%)
Laboratory parameters (median [IQR])	
Creatinine (mg/dL)	0.8 (0.6-1.03)
Hemoglobin (g/dL)	12.7 (11.4-13.8)
Neutrophil count (103/μL)	4.2 (2.84-6.04)
Lymphocyte count (103/μL)	1.18 (0.85-1.56)
White blood cell count (103/μL)	6.07 (4.56-7.8)
Platelet count (103/μL)	208.5 (168-263.5)
NLR	3.45 (2.06-5.78)
PLR	176.4 (124.4-244.3)
C-reactive protein (mg/L)	34.3 (13.2-99.2)
Troponin I (ng/mL)	3.6 (1.5-14.8)
Albumin (g/dL)	3.5 (3.1-3.9)

A total of 300 patients (135 male (55.0%), mean age 57.4±14.2 years), were included in this retrospective study. The percentages of patients who had hypertension, congestive heart failure, and chronic kidney disease were 45%, 5%, and 12%, respectively. The median serum troponin I value was 3.6 ng/mL (IQR, 1.5-14.8). Baseline clinic-demographic characteristics and admission laboratory values of the patients were shown in Table 1.

Table 2. Comparison of laboratory parameters of patients with and without myocardial injury

Parameters Age (year) (mean ± SD) Sex (n (%))	Patients with myocardial injury (n=75)	Patients without myocardial injury (n=225)	P
Female	63.0 ± 11.96	55.3 ±14.2	<0.001
Male			
Comorbidities (n (%))	24 (32.0%)	113 (50.5%)	0.009
Coronary artery disease	51 (68.0%)	112 (49.5%)	
Hypertension			<0.001
Diabetes mellitus	23 (30.0%)	27 (12.0%)	<0.001
Chronic kidney disease	48 (64.0%)	81 (36.0%)	0.677
Congestive heart failure	24 (32.0%)	63 (28.0%)	<0.001
Laboratory parameters	6 (8.0%)	54 (24%)	0.045
(median [IQR])	66 (88.0%)	9 (4.0%)	
Creatinine (mg/dL)			<0.001
Hemoglobin (g/dL)	0.8 (0.5-1.0)	0.76 (0.6-0.94)	0.001
Neutrophil count (103/μL)	12 (10-13)	12.9 (11.8-13.9)	<0.001
Lymphocyte count	5.7 (4.2-8.12)	3.96 (2.6-5.18)	0.001
(103/μL)	0.9 (0.6-1.5)	1.26 (0.9-1.57)	<0.001
White blood cell count	7.6 (5.67-9.6)	5.7 (4.38-7.26)	0.430
(103/μL)	207 (168-285)	209 (168-261)	<0.001
Platelet count (103/μL)	6.1 (3.3-10.8)	3.08 (1.86-5.1)	0.002
NLR	207.0 (132.5-371.4)	170 (122.7-231.5)	<0.001
PLR	124.1 (49-203)	24.9 (11.1-80.8)	<0.001
C-reactive protein (mg/L)	306 (72-852)	2.5 (1.3-5.5)	<0.001
Troponin I (ng/mL)	3.1 (2.8-3.4)	3.6 (3.26-3.9)	
Albumin (g/dL)			

Comparison of Laboratory Values between Patients with and without Myocardial Injury Seventy-five patients (25.0%) had acute myocardial injury. Patients who developed myocardial injury were significantly older than patients without myocardial injury (mean ages 63±11.96 vs 55.3±14.2 years, respectively). In the myocardial injury group, coronary artery disease, hypertension, chronic kidney disease, and congestive heart failure were significantly more frequent compared with the patients without myocardial injury. Median white blood cell and neutrophil counts were significantly higher, whereas lymphocyte count was significantly lower in patients with myocardial injury compared with the patients without myocardial injury. Median NLR was also significantly higher in patients with myocardial injury compared to patients without myocardial injury. Median serum troponin I levels were significantly higher in patients with myocardial injury compared with patients without myocardial injury (306 ng/mL (72-852) vs. 3.6 ng/mL (3.26-3.9), p<0.001).

Table 3. Comparison of clinical characteristics and laboratory values of deceased and survivor patients

Parameters Age (years) (mean ± SD) Sex (n (%))	Deceased Patients (n=50)	Surviving Patients (n=250)	p value
	63.0 ± 11.96	55.3 ±14.2	<0.001
Female			
Male	23 (46.0%)	130 (47.4%)	0.851
Comorbidities (n (%))	27 (54.0%)	144 (52.6%)	
Coronary artery disease			0.057
Hypertension	13 (26.0%)	30 (14.2 %)	<0.001
Diabetes mellitus	36 (72.0%)	104 (38.0%)	0.363
Chronic kidney disease	18 (36.0%)	81 (29.6%)	<0.001
Congestive heart failure	16 (32.0%)	22 (8.0)	0.497
Laboratory parameters (median [IQR])	4 (8.0%)	14 (5.1%)	
			0.018
Creatinine (mg/dL)	1.0 (0.8-1.55)	0.76 (0.6-0.92)	<0.001
Hemoglobin (g/dL)	12 (10-13)	12.9 (11.8-13.9)	<0.001
Neutrophil count (103/μL)	6.05 (4.2-8.6)	4.0 (2.7-5.27)	<0.001
Lymphocyte count (103/μL)	0.9 (0.63-1.6)	1.22 (0.9-1.54)	<0.001
White blood cell count (103/μL)	8.0 (6.1-10.8)	5.71 (4.4-7.29)	0.857
Platelet count (103/μL)	207 (168-285)	209 (168-261)	<0.001
NLR	6.1 (3.3-10.8)	3.08 (1.86-5.1)	0.002
PLR	207 (132.5-371.4)	170 (122.7-231.5)	<0.001
C-reactive protein (mg/L)	124.1 (49-203)	24.9 811.1-80.8)	<0.001
Troponin I (ng/mL)	306 (72-852)	2.5 (1.3-5.5)	<0.001
Albumin (g/dL)	3.1 (2.8-3.4)	3.6 (3.26-3.9)	

Comparison of Laboratory Values and Clinical Outcomes Between Deceased and Surviving Patients In total, 50 out of 300 patients (16.6%) died during the study period. The deceased patient group was significantly older compared with surviving patients (63.0 ± 11.96 vs. 55.3 ±14.2 years, respectively, p<0.001). Only hypertension and CKD were significantly more common among the deceased compared with surviving patients. There was no significant difference between the groups in terms of other comorbid conditions. The comparison of the laboratory parameters between the groups was depicted in table 3. Myocardial injury was present in 84% of the deceased group, whereas 12.8% of the surviving patients had myocardial injury (p<0.001). The median serum troponin I levels were 306 ng/mL (72-852) and 2.5 ng/mL (1.3-5.5) in the deceased and surviving patients, respectively (p<0.001).

Coronary artery disease / Acute coronary syndrome

OP-209

Impact of comorbidities on clinical parameters and length of stay in hospital among patients with acute coronary syndrome

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Background and Aim: Comorbidities often accompany acute coronary syndrome (ACS) and are associated with poor clinical outcomes. However, the representation rates of ACS patients with comorbidities in randomized controlled trials are interestingly low, and the relationship between comorbidities and clinical outcomes in patients with ACS is controversial. The aim of the present study is to evaluate the association between comorbidities and clinical properties in patients with ACS.

Methods: Among 407 adults, undergone coronary angiography (CAG) with ACS during 2016–2019, collected data on comorbidities, clinical specifities and length of stay in hospital (LOS). The comorbidities of the patients, such as peripheral artery disease, cerebral vascular disease, diabetes mellitus, chronic obstructive pulmonary disease, chronic renal failure and anemia were recorded. Patients were divided into two groups: with and without two or more comorbidities. Baseline characteristics and outcomes were compared between the two groups.

Results: The mean age of the patients was 65.99±32.31 and 114 (28,0%) was female. The number of patients had ≥2 comorbidities was 128 (31.4%). When two groups compared, left ventricular ejection fraction (LVEF) was lower in patients with two or more comorbidities (p<0.001) and the differences between left ventricular end-systolic diameter were significant (p=0.023). Also, the inflammation markers such as white blood cells (p=0.033) and neutrophil (p=0.007) count, the neutrophil-lymphocyte ratio was higher (p=0.030) in patients with ≥2 comorbidities but there was no significance between C-reactive protein levels. The rate of high troponin levels was higher and LOS was longer in patients with comorbidities (p=0.022). However, compared to have ≥2 comorbidities, having one or no comorbidity was not associated with SYNTAX score and deciding to treatment strategy.

Conclusions: One-third of patients who underwent coronary angiography with the diagnosis of ACS have two or more comorbidities. It would be beneficial to plan more comprehensive studies regarding prolonging hospitalization and to individualize the treatments according to the patients' comorbidities.

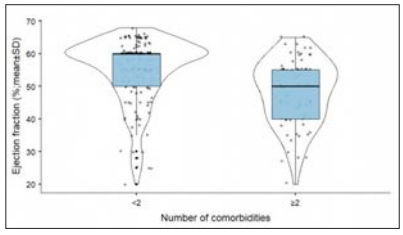

Figure 1. Graphical representation of the mean ejection fraction by comorbidity group.

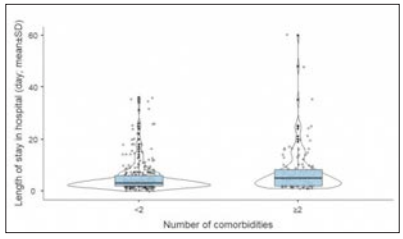

Figure 2. Graphical representation of the mean hospital stay by the comorbidity group.

Table 1. Sociodemographic and clinical characteristics of the participants

Variables	All patients (n=407)
Age (year; mean±SD)	65,99±32,31
Female (yes; n (%))	114 (%28,0)
Current smoking (yes; n (%))	164 (%40,3)
Medical history (yes; n (%))	
Hypertension	164 (%40,3)
Diabetes Mellitus	78 (%19,2)
Coronary artery disease	74 (%18,2)
Renal failure	24 (%5,9)
Chronic Pulmonary Disease	42 (%10,3)
Peripheral Artery Disease	24 (%5,9)
Cerebrovascular Disease	23 (%5,7)
Anemia	26 (%6,4)
Comorbidity (mean±SD)	1,15±1,28
Number of comorbidities (yes; n (%))	
≥2	128 (%31,4)
<2	279 (%68,6)

Table 2. Comparison of clinical characteristics and outcomes between the groups

Variables	All patients (n=407)	Number of comorbidities≥2 (n=128)	Number of comorbidities<2 (n=279)	Statistics
Systolic blood pressure (mm/Hg; mean±SD)	123,24±16,78	124,02±16,73	122,88±16,83	t=0,635, p=0,526
Diastolic blood pressure (mm/Hg; mean±SD)	76,89±10,10	76,23±9,80	77,20±10,24	t=-0,899, p=0,369
Heart rate (bpm; mean±SD)	77,19±12,39	77,53±13,38	77,03±11,93	t=0,364, p=0,704
Ejection fraction (%;mean±SD)	52,45±10,56	48,15±10,07	54,69±10,14	t=-4,564, p<0,001*
Left ventricular end-diastolic diameter (mm±SD)	46,27±9,52	47,15±9,86	45,81±9,33	t=1,013, p=0,312
Left ventricular end-systolic diameter (mm±SD)	31,54±8,81	33,37±9,41	30,58±8,35	t=2,284, p=0,023*
Interventricular septum (mm±SD)	10,86±3,32	11,18±3,28	10,70±3,34	t=1,046, p=0,297
SYNTAX score (mean±SD)	11,64±8,39	11,10±8,18	11,89±8,48	t=-0,885, p=0,377
Length of stay in hospital (day; mean±SD)	5,91±6,86	7,36±8,57	5,25±5,81	t=2,909, p=0,004**
Killip classification (mean±SD)	1,03±0,27	1,08±0,37	1,01±0,21	t=1,903, p=0,059
Rehospitalization (yes; n(%))	85 (29,4)	35 (36,8)	50 (25,8)	X2=3,763, p=0,052
Treatment strategy (yes; n(%))				X2=0,942, p=0,624
Conservative	186 (45,7)	63 (49,2)	123 (44,1)	
Percutaneous coronary intervention	171 (42,0)	121 (43,4)	50 (39,1)	
Coronary artery by-pass greft	50 (12,3)	35 (12,5)	15 (11,7)	
Left main coronary artery stenosis (yes; n(%))	11 (2,7)	5 (3,9)	6 (2,2)	X2=1,029, p=0,310
In-hospital clinical outcomes (yes; n(%))				
Heart Failure	1 (0,2)	0	1 (0,4)	X2=0,454, p=0,500
Pulmonary Edema	2 (0,5)	2 (1,6)	0	X2=4,458, p=0,035*
Serebrovascular disease	1 (0,2)	1 (0,8)	0	X2=2,214, p=0,137
ST-segment deviation	3 (0,8)	1 (0,8)	2 (0,7)	X2=0,008, p=0,930
Left Bundle Branch Block	19 (4,7)	10 (7,8)	9 (3,2)	X2=4,148, p=0,042*
Cardiac Arrest	2 (0,5)	0	2 (0,7)	X2=0,924, p=0,336
Cardiogenic Shock	0	0	0	
High Troponin Level	144 (35,4)	35 (27,3)	109 (39,1)	X2=5,276, p=0,022*

Table 3. Comparison of laboratory parameters between the groups

Variable	All patients (n=407)	Number of comorbidities≥2 (n=128)	Number of comorbidities<2 (n=279)	Statistics
Hemoglobin (g/dl)	14,60±2,39	14,42±3,22	14,68±1,89	t=-1,031, p=0,303
White blood cell (×109/L)	8,85±3,08	9,52±3,50	8,54±2,83	t=2,996, p=0,003**
Neutrophil (×109/L)	5,96±2,91	6,59±3,43	5,67±2,60	t=2,704, p=0,007**
Lymphocyte (×109/L)	2,07±0,93	2,16±1,00	2,04±0,89	t=1,202, p=0,230
Monocyte (×109/L)	0,65±0,47	0,66±0,31	0,64±0,52	t=0,325, p=0,745
Red Cell Distribution Width (fL)	14,33±1,86	14,48±2,37	14,26±1,58	t=-0,440, p=0,660
Platelet (×109/L)	237,70±71,32	248,13±69,45	232,90±71,77	t=2,007, p=0,045*
Neutrophil-Lymphocyte Ratio	3,71±3,81	4,32±5,14	3,43±2,97	t=2,183, p=0,030*
Platelet-Lymphocyte Ratio	137,64±87,25	144,65±106,34	134,76±96,78	t=-1,101, p=0,272
Troponin (µg/L)	3,07±10,47	4,49±17,06	2,46±5,60	t=1,233, p=0,220
Creatine kinase (µg/mL)	245,54±426,87	283,93±574,25	228,01±338,91	t=0,995, p=0,321
Creatine kinase-MB (µg/mL)	43,53±53,68	45,85±55,50	42,47±52,91	t=0,561, p=0,575
Creatinin (mg/dl)	1,46±6,92	1,26±1,44	1,56±8,31	t=0,400, p=0,690
Blood Urea Nitrogen (mg/dl)	21,43±11,83	22,41±17,55	20,97±17,90	t=-1,130, p=0,259
Erythrocyte Sedimentation Rate (mm/hour)	25,55±24,14	27,73±25,21	24,50±23,56	t=1,104, p=0,270
C-reactive protein (mg/dL)	13,58±26,76	17,24±33,76	11,81±22,53	t=1,236, p=0,219
Alanine Aminotransferase (U/L)	35,52±144,47	23,37±25,81	39,26±173,61	t=-0,756, p=0,450
Aspartate Aminotransferase (U/L)	47,50±230,79	38,78±50,19	51,51±276,92	t=-0,506, p=0,613
Gamma Glutamyl Transferase (U/L)	37,87±48,36	32,92±27,32	40,19±55,43	t=-1,661, p=0,098
Alkaline phosphatase (U/L)	86,66±38,00	86,28±31,67	86,84±40,57	t=-0,129, p=0,897
Lactate Dehydrogenase (U/L)	341,95±308,80	341,20±260,43	342,31±329,99	t=-0,032, p=0,975
Amylase (U/L)	73,41±42,23	77,70±49,63	71,43±38,30	t=1,164, p=0,246
Triglyceride (mg/dL)	174,07±103,69	172,11±101,45	174,98±104,90	t=-0,250, p=0,803
Total cholesterol (mg/dL)	191,60±48,78	187,23±54,21	193,63±46,02	t=-1,126, p=0,262
High-density lipoprotein cholesterol (mg/dL)	38,99±22,22	37,89±10,48	39,50±25,95	t=-0,657, p=0,512
Low-density lipoprotein cholesterol (mg/dL)	129,82±41,81	124,49±43,15	132,31±41,01	t=-1,701, p=0,090
Blood glucose (mg/dL)	139,15±110,25	146,31±73,68	135,86±123,48	t=0,884, p=0,377

Coronary artery disease / Acute coronary syndrome

OP-210

Prognostic power of neutrophil-to-lymphocyte ratio in young patients with acute coronary syndrome

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Background and Aim: Acute myocardial infarction induced acute inflammatory responses and leads myocardial injury. So it is characterized by increasing circulatuar inflamatuar and myocardial necrosis biomarkers. Although neutrophil-to-lymphocyte ratio(NLR) and cardiac troponin(cTn) has been proposed as prognostic biomarkers in ACS, the prognostic value of these in young patients has not been clearly defined. We sought to demonstrate using of NLR along with other clinical and laboratory markers to evaluate the possible prognostic role.

Methods: This is a retrospective single center study and consisted of 952 patients with acute coronary syndrome (ACS) under 45 years. The measurement of cTn and NLR was made according to a standard protocol at admission and repeated 3–6 h later to evaluate changes of concentrations during the first 24 h after and then daily. The NLR was calculated from the same blood sample with the highest hs-cTnI values. NLR was divided into two sub-groups based on an optimal cut off value predicting hospital all-cause mortality by receiving operator characteristic (ROC) curve analysis. Because the cTn and NLR can be affected by many non-cardiac diseases and conditions only young patients without comorbidity were included.

Results: The majority (54.8%) of the patients had STEMI followed by NSTEMI (26.7%) and USAP (18.5%). Male sex was dominant, smoking was the most common risk factor. The area under the curve on ROC analysis of NLR for predicting hospital mortality was 6.1 with a sensitivity of 82% and specificity of 78% (ROC area 0.827, p<0.001, 95%CI,0.741-0.913) (Figure 1). Increased NLR ratio was observed more frequently in STEMI (86.9% vs 44.4%, p<0.001) and associated with increased peak cTn levels (r=0.275, p<0.001), CRP(r=0.171, p=0.001) and showed significant reverse correlations with LVEF (r=-322, p<0.001). Patients with high NLR had significantly higher rate of hospital mortality (5.7% vs 0.6%, p<0.001), and hospital MACE (35.5% vs 8.5%, p<0.001) including cardiogenic shock, arrhythmia, stent thrombosis, heart failure, bleeding, infection or CIN. Also there was significant association with NLR and hemodynamic functions such as systolic blood pressure (SBB) (r=-0.189), diastolic blood pressure (DBP) (r=-0.118), pulse rate (r=0.218), symptom of heart failure (Killip ≥2) (r=0.486) and shock (r=0.213). was determined to be an independent predictor for hospital mortality (HR, 1.033; 95%CI 0.915-1.168) and MACE (HR, 1.044; 95%CI 1.010-1.068) in Cox regression analysis.

Conclusions: Patients with higher NLR levels tended to be had more STEMI, hospital mortality and MACE with higher hs-cTnT levels and it was independent predictor of hospital mortality and major adverse events for young AMI patients. We concluded that NLR was an inexpensive and readily available marker and NLR combination with clinical signs such as blod pressure, pulse rate, symptoms of heart failur, other laboratory marker including cTn, CRP levels and LVEF provide incremental prognostic information in patient with ACS.

Table 1. Baseline demographic characteristics, clinic presentations and laboratory outcomes of all study patients

Variables	NLR ≥ 6.1 (n=245)	NLR≤6.1 (n=707)	P
Male (%)	200(81.6)	595(84.2)	0.373
BMI (kg/m2)(%)	28±4	29±4	0.043
Smoking (%)	202(82.4)	583(82.5)	0.916
Diabetes mellitus (%)	55(22.6)	159(22.5)	0.924
Hypertension (%)	55(22.4)	227(32.1)	0.002
Dyslipidemia (%)	178(72.7)	556(78.6)	0.021
Previous CAD (%)	17(6.9)	72(10.2)	0.090
Family history (%)	54(22)	226(32)	0.019
Illegal drug use (%)	5(2)	21(3)	0.400
PAD (%)	14(1.6)	4(1)	0.178
Presentation (%)			
STEMI	215(87.8)	314(44.4)	<0.001
NSTEMI	23(9.4)	232(32.5)	<0.001
USAP	7(2.9)	163(23.1)	<0.001
SBP (mmHg)	113±22	125±24	<0.001
DBP (mmHg)	72±15	78±15	<0.001
Pulse rate	89±21	81±16	<0.001
Echocardiography (LVEF)	40±10	49±9	0.001
Hemoglobin (mg/dL)	14±1.2	14.2±1.5	0.227
Triglyceride (mg/dL)	187±207	247±226	<0.001
HDL-C (mg/dL)	40±10	39±9	0.010
LDL-C (mg/dL)	140±46	145±44	0.179
CRP (mg/L)	14.6±28.4	7.2±7.6	0.006
hs-Tn I	40502±37426	15248±22820	<0.001
Glucose (mg/dl)	165±105	140±75	0.001
Primary PCI	211(86)	303(42)	<0.001
Elective or emergent CABG	12(4.9)	74(10.5)	<0.001
Medical follow-up	13(5.3)	75(10.6)	<0.001
Minimal or non-occlusive CAD	18(7.3)	63(8.1)	0.223

Table 2. Clinical outcomes of patients with ACS during hospital stay

In hospital outcomes (%)	NLR ≥ 6.1 (n=245)	NLR < 6.1 (n=707)	P
In-hospital mortality, n (%)	14(5.7)	4(0.6)	0.001
Hospital MACE, n(%)	87(35.5)	60(8.5)	<0.001
Ventricular arrhythmia	45(18.4)	21(3)	<0.001
Atrioventricular block	7(2.8)	5(0.7)	<0.001
Cardiac rupture/tamponade	1(0.24)	0	
Bleeding complications	2(0.8)	6(0.8)	0.324
CIN	23(9.4)	7(1)	<0.001
Infection	14(5.6)	1(0.1)	0.012
Acute stent thrombosis	6(2.4)	7(1.1)	0.171
Subacute stent thrombosis	22(9)	23(3.3)	0.001
Killip ≥2	35(14.3)	15(2.1)	<0.001
Cardiogenic shock	8(3.3)	4(0.6)	0.001

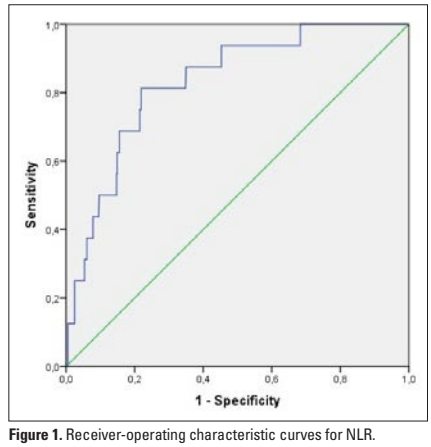

Figure 1. Receiver-operating characteristic curves for NLR.

Coronary artery disease / Acute coronary syndrome

OP-211

Can maintenance of a structured enhanced education and follow- up program be feasible in an economically challenged 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, effectiveness and maintenance 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: This longitudinal prospective study, took place between 2014 and 2020 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 other 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 172 participant were enrolled; 61% were women, mean age was 40±11.9 years, 41% were not working. Mean BMI was 27.8+5.07 kg/m^2. Overall, at baseline evaluation, 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 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. More importantly, study results showed that maintenance of this model was feasible to conduct.

Coronary artery disease / Acute coronary syndrome

OP-212

Association of myocardial bridge with coronary artery aneurysm and ectasia

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Background and Aim: Coronary artery ectasia (CAE) and coronary artery aneurysm (CAA) are uncommon coronary artery disease and CAE is generally defined dilatation of coronary artery to 1.5 times or more the size of adjacent normal segment of the coronary artery. CAA is defined as coronary dilatation that exceeds the diameter of normal adjacent segments more than 1.5-2 times. Although both CAE and CAA have many causes, the most common cause is known to be atherosclerosis. The myocardial bridge (MB) is a congenital coronary artery variant and is usually seen in the left anterior descending artery (LAD). Recent studies have shown that MB may be associated with more coronary artery disease, atherosclerosis and myocardial infarction. The recent meta-analyse and meta-regression by Sorin H et al; it has been reported that MB can cause important cardiovascular outcomes such as myocardial ischemia and major cardiac event. In this study, we aimed to investigate the association of CAE and CAA with MB.

Methods: Angiographic records of 6153 patients who underwent coronary angiography for any reason were retrospectively reviewed by interventional cardiologists. Patients with a previous history of coronary artery bypass operation and poor angiographic image quality were excluded from the study. Myocardial bridge was described as the case where a segment of the epicardial coronary artery is covered by myocardium.

Results: When both ectasia and aneurysm were evaluated together, their association with myocardial bridge was significantly higher ($p<0.05$), and when the association between CAE and CAA with presence of MB evaluated separately both of them were appeared significantly together with MB (Table 1). When the presence of MB in each coronary artery with CAA and CAE involvement was evaluated, CAA involvement was observed most commonly on the LAD artery, then on the RCA, and least on the Cx artery. And the association of aneurysm with MB was higher just in LAD ($p<0.05$) (Table 2). Furthermore, CAE involvement was noted most frequently on the RCA, then on the Cx, and least often on the LAD. The association of coronary artery ectasia with MB was significantly higher in all three of the LAD, CX and RCA ($p<0.05$) (Table 3).

Conclusions: It is known that CAA and CAE are closely related to atherosclerosis. Recent studies have shown that MB, which is a congenital coronary anomaly, may also be associated with atherosclerosis and may rarely cause serious cardiac events. The present study showed that MB, which can be seen alone or with aneurysm and ectasia, should be taken into account in the management of coronary artery disease.

Table 1. Association of myocardial bridge with coronary artery aneurysm and ectasia

Parameters	Total (6153)	Bridge (+) (n=91)	Bridge (-) (n=6062)	P
Ectasia and aneurysm(n,%)	377(6.1)	19(20.9)	358(5.9)	<0.05
Ectasia(n,%)	340(5.5)	16(17.6)	324(5.3)	<0.05
Aneurysm (n,%)	37(0.6)	3(3.3)	34(0.6)	<0.05

Table 2. Association of coronary artery aneurysm and myocardial bridge for each coronary artery

Aneurysm	Total (6153)	Bridge (+) (n=91)	Bridge (-) (n=6062)	P
LAD	12(0.2)	3(3.1)	9(0.1)	<0.05
CX	7(0.1)	0	7(0.1)	>0.05
RCA	10(0.2)	0	10(0.2)	>0.05
LAD-CX	1(0)	0	1(0)	>0.05
LAD-RCA	0	0	0	0
CX-RCA	0	0	0	0
LAD-CX-RCA	3(0)	0	3(0)	>0.05
LMCA	4(0.1)	0	4(0.1)	>0.05

Table 3. Association of coronary artery ectasia and myocardial bridge for each coronary artery

Ectasia	Total (6153)	Bridge (+) (n=91)	Bridge (-) (n=6062)	p
LAD	13(0.2)	3(3.3)	10(0.2)	<0.05
CX	63(1)	4(4.4)	59(1)	<0.05
RCA	151(2.5)	6(6.6)	145(2.4)	<0.05
LAD-CX	16(0.3)	1(1.1)	15(0.2)	>0.05
LAD-RCA	25(0.4)	0	25(0.4)	>0.05
CX-RCA	17(0.3)	1(1.1)	16(0.3)	>0.05
LAD-CX-RCA	52(0.9)	1(1.1)	51(0.8)	>0.05
LMCA	3(0)	0	3(0)	>0.05

Coronary artery disease / Acute coronary syndrome

OP-213

A Novel Nutritional Index Serves as A Useful Prognostic Indicator in patients with acute ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention

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Background and Aim: We aimed to investigate whether a novel nutritional index (TCBI) based on triglyceride (TG), total cholesterol (TC) and body weight (BW) was associated with mortality in patients with acute ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention.

Methods: In the study, between January 2016 and August 2017, 98 acute STEMI patients with an average age of 56.9±12.1 were enrolled. In the analysis, TCBI = (TG × TC × BW) / 1000 were calculated. The primary outcome of the study was general in-hospital mortality.

Results: The TCBI was significantly lower in the group with higher mortality than in the group with low mortality (1140.6±304.9 and 2716.8±2265.1 $p<0.001$) and a negative correlation was found between TCBI and mortality ($r=-0.293$, $p=0.003$). The TCBI remained an independent predictor for in-hospital mortality (OR, 0.99; 95% CI, 0.998 to 1.0; $p=0.03$).

Conclusions: The TCBI, was determined as an independent predictor for in-hospital mortality in STEMI patients. A new and simple nutritional index, TCBI, can be applied as a prognostic indicator in acute STEMI patients.

Table 1. Clinical, echocardiography and laboratory findings of groups

	mortality group (n:6)	no mortality group (n:92)	P
Age (years)	78.8 ± 12.1	55.5 ± 10.7	0.005
EF (%)	36.0 ± 8.2	45 ± 7.9	0.05
Heart rate (Beats / min)	82 ± 28	77 ± 17	0.5
SBP (mmHG)	132 ± 19	130 ± 26	0.9
DBP (mmHG)	79 ± 12	79 ± 17	0.9
Glucose (mg/dL)	125.6 ± 59.3	149.6 ± 69.8	0.4
Creatine (mg/dL)	1.5 ± 1.1	1.0 ± 0.2	0.01
Hemoglobin (g/dL)	11.1 ±2.0	14.9 ±1.7	< 0.0001
Triglycerides (mg/dL)	81.0 ± 28.3	174.2 ± 106.7	< 0.0001
Total cholesterol (mg/dL)	180.1 ± 36.8	190.7 ± 45.6	0.5
TCBI	1140.6 ± 304.9	2716.8 ± 2265.1	< 0.0001

EF:ejection fraction, SBP: systolic blood pressure, DBP:diastolic blood pressure, TCBI: nutritional index consisting of triglyceride, total cholesterol and body weight.

Coronary artery disease / Acute coronary syndrome

OP-214

The association between serum leptin and hs-CRP levels with cardiovascular disease in metabolic syndrome patients

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Background and Aim: Metabolic syndrome (MS) is a clustering of metabolic abnormalities such as obesity, insulin resistance, dyslipidemia which causes endothelial dysfunction and atherosclerosis and is detected independently associated with cardiovascular major adverse events. Previous studies suggest that high leptin and high sensitivity C-reactive protein (hs-CRP) levels in MS patients are independent indicators of cardiovascular risk. In our study, we aim to detect the association between plasma leptin and hs-CRP levels and cardiovascular disease (CVD) in MS patients.

Methods: Between December 2004 and January 2006, 84 patients who underwent first diagnostic angiography due to angina and/or ischemic electrocardiography changes and had three or more of 'The National Cholesterol Education Program Adult Treatment Panel III' MS criteria were enrolled in this study. Patients' demographic, biochemical and clinical characteristics and plasma leptin and hs-CRP levels were evaluated. A diagnosis of coronary artery disease (CAD) was made when left main CAD and/or 50% or more coronary stenosis in at least one major coronary artery were present.

Results: Patients were divided into two groups according to coronary angiography: Patients with CAD (Group 1, n=50) and patients without CAD (Group 2, n=34). Groups demographic and clinical characterixtics were similar. In Group 2 patients, female patients, waist circumference (WC), body mass index (BMI), abdominal obesity (AO) and WC/hip circumference (HC) ratio were higher than in Group 1.Serum leptin and hs-CRP levels were found higher than normal subjects but there was no statistically significant difference between serum leptin and hs-CRP levels in Group 1 and 2. Serum leptin levels was 20.41±2.18 ng/ml in Group 1 and 26.91±1.77 ng/ml in Group 2 patients ($p=0.081$). Serum hs-CRP levels are 4.88±3.22 mg/l in Group 1 patients and 3.04±2.91 mg/l in Group 2 patients ($p=0.06$). Serum leptin levels in female patients were higher than male patients ($p=0.012$ and $p=0.0001$, respectively).

Conclusions: In our study, serum leptin and hs-CRP levels were found higher than normal values in MS patients but there was no statistical significance between patients with or without CAD. In term of serum leptin levels, this could be explained several factors that; 1) all the patients had MS as a risk factor for hyperleptinemia, and 2) patients without CAD were predominantly female who have higher leptin levels than males and 3) had more abdominal obesity which are related to higher serum leptin levels. Hs-CRP levels were detected high in all MS patients. According to data, hs-CRP levels in patients with CAD were higher than patients without CAD but it didn't show statistical significance ($p=0.06$). To explain this it could be assumed that patients in Group 2 had higher BMI and WC, therefore more adipose tissue resulting in more inflammatory cytokins and higher hs-CRP levels. To get more conclusive results futher prospective and the higher patient number studies are needed.

Coronary artery disease / Acute coronary syndrome

OP-215

The management of acute coronary syndrome patients in COVID-19 pandemic

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Background and Aim: Acute coronary syndromes (ACS) are the diseases that requires emergent therapies and if not applicable most of these patients have high morbidity and mortality. Therefore, the management of these syndromes is very well-defined. However, the management of them during an infectious outbreak can be changed to reduce the contamination and thus to protect healthcare providers and other individuals. Nowadays, there is a Coronavirus (COVID-19) pandemic all over the world including Turkey and this pandemic affects lots of people, especially immunocompromised and elderly individuals. We aimed to report an algorithm about the management of ACS patients during COVID-19 pandemic.
Methods: According to this algorithm patients who admitted to the emergency department and diagnosed to have ACS divide into two groups within the scope of COVID-19 outbreak. At first group, patients with suspected or confirmed COVID-19 cases are included. At second group unsuspected cases that do not have the signs and symptoms of COVID-19 infection was included.If the patient has STEMI and includes in the first group, thrombolytic therapy (Actilyse 100 and 50 mg) is preferred at first. If the patient has NSTEMI and includes within the first group, the treatment decision is made according to the risk category of the patients.
Results: This algorithm was applied on a total of 47 patients who were hospitalized between 12 March 2020 to 31 March 2020 with the diagnosis of ACS. Among 47 ACS patients, 32 had STEMI (16 inferior, 14 anterior and 2 posterior MI, mean age: 52.8±19 years, male/female: 26/6, hypertension (HT) prevalance 53%, diabetes mellitus (DM) prevalance 18%) and 15 had NSTEMI (mean age: 63.0±16 years, male/female: 12/3, HT prevalance 66%, DM prevalance 26%). All STEMI was type I MI. 31 STEMI and 14 NSTEMI patients were included into group 2 patients and treated within our routine procedure protocol. All STEMI patients except one who was referred to cardiovascular surgery due to the LAD rupture were treated with PCI. On the other hand, 14 NSTEMI patients were treated with PCI and one patient was treated with medical therapy. 1 STEMI and 1 NSTEMI patients were included into group 1 because of the suspected COVID-19 infection.
Conclusions: The management of ACS patients during pandemics have to be well-planned and organised to protect both of health care workers and other individuals interested with the patients. Thrombolytic therapy is the first option for eligible STEMI patients. However, NSTEMI patients have to be categorised based on their risk and then the management strategy should be determined. CTA is also important for medium and low risk NSTEMI patients to decide the invasive therapy before discharge from the hospital. An isolation catheter room and isolation room in ICU with negative pressure is a requisite for follow-up of these patients and must be included into these management algorithm.

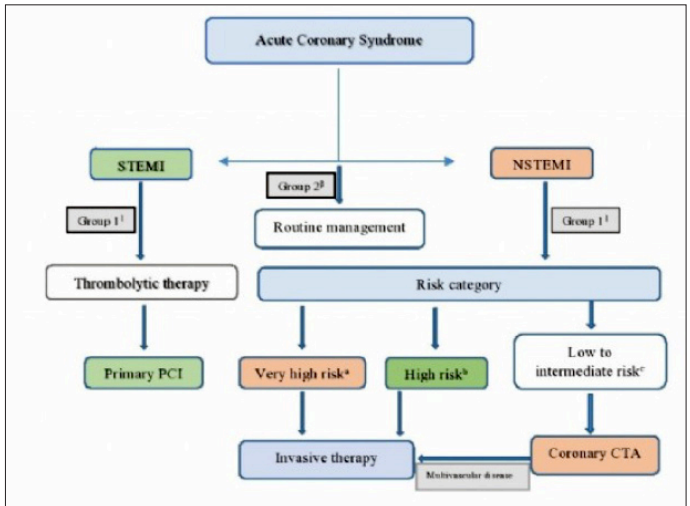

Figure. Algorithm of acute coronary syndrome patients in COVID-19 pandemic

Abbreviations ACS: acute coronary syndrome, CTA: computed tomography angiography, NSTEMI: non ST segment elevated myocardial infarction, PCI: percutaneous coronary intervention, STEMI: ST segment elevated myocardial infarction,

1 confirmed or suspected COVID-19 cases. B unsuspected COVID-19 cases

Only type 1 STEMI cases were included into this figure. Other STEMI types were excluded at emergency department and managed according to the current guidelines.

a patients with haemodynamic instability, recurrent or ongoing chest pain resistant to medical treatment, life threatening arrhythmia or cardiac arrest, developing mechanical complications or severe heart failure and showing repetitive ST-T wave changes including intermittent ST segment elevation.

b patients with an increase or decrease in troponin levels in accordance with an MI, dynamic ST-T wave change, GRACE risk score above 140.

c patients with diabetes mellitus, renal Insufficiency (eGFR <60), left ventricular ejection fraction < 40% or presence of congestive heart failure, early post-infarction angina,previous history of PCI or bypass surgery, GRACE risk score between 109-140.

Figure 1. Algorithm of acute coronary syndrome patients in COVID-19 pandemic.

Coronary artery disease / Acute coronary syndrome

OP-216

Routine immediate chest computed tomography of STEMI patients before pPCI as a rule-out strategy of COVID-19 infection; clinical outcomes and impact on reperfusion time

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Background and Aim: The coronavirus disease 2019 (COVID-19) pandemic poses an unprecedented challenge for the management of patients with ST-segment elevation myocardial infarction (STEMI).Fast and reliable COVID-19 diagnosis gains utmost importance for decision making. Our primary aim is to assess the feasibility of routine chest computed tomography (CT) of STEMI patients before primary percutaneous coronary intervention (pPCI) as a rule-out strategy of COVID-19. Our secondary aim was to investigate the impact of COVID-19 pandemic on STEMI patients.
Methods: All STEMI patients underwent immediate lung CT scan with the aim of ruling out COVID-19.Control group was consisted of matched STEMI patients treated prior to COVID-19 pandemic.
Results: 41 STEMI patients during pandemic were enrolled (group 1) and 80 STEMI patients from pre-pandemic era constituted (group 2) the control population. Ischemia duration was significantly higher in group 1[570 (264-2020 minutes vs 172 (108-316 minutes), p=<0.001]. Longer ischemia duration was driven by both prehospital delay and door-to-device time [80 minutes (47-120) vs 120 minutes (60-285), p=0.005]. Lung CT scan and interpretation of CT images was associated with a delay of 24 minutes (18-30 minutes). Initial troponin levels and pro-BNP levels were significantly higher in group 1 [1.00 ng/ml (0.625-2.35) vs 4.00 ng/ml (2.65-8.50), p=<0.001] and [respectively 297 (75-820) pg/ml vs 698 pg/ml (259-1757), p=0.004]. The admission Killip class in group 1 was higher (p=0.001). New-onset heart failure was significantly more prevalent in group 1 (34% vs 19.2%, p=0.003).
Conclusions: Our study demonstrated that immediate chest computed tomography of STEMI patients as a rule-out strategy of COVID-19 is fast,reliable and feasible. COVID-19 pandemic has serious impact on STEMI patients with regard to delays in medical attention and its clinical consequences.

Coronary artery disease / Acute coronary syndrome

OP-217

Is there any relationship between heart rate recovery index and asthma?

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Background and Aim: Asthma is a common chronic inflammatory airway disease that is prevalent worldwide. Both genetic and environmental factors act together, and there is widespread lung involvement, which is reversible spontaneously or with treatment. The impaired heart rate recovery index (HRRI) was diagnosed in patients with obstructive sleep apnea syndrome, silent myocardial ischemia, and heart failure with preserved ejection fraction. There is not sufficient number of studies investigating the relationship between asthma and HRRI. In this study, we aimed to determine the presence of impaired HRRI in patients with asthma, and to examine related factors, such as the severity of asthma, asthma control status.
Methods: A total of 70 people with asthma and healthy volunteers were enrolled. All datas were prospectively collected from individuals who applied to our hospital between July 2019 and August 2019 because of chest pain and/or dyspne. The determination of asthma was made according to Global Initiative for Asthma criteria. 35 five patients with asthma consequently were enrolled to study. All patients were questioned with the Asthma Control Test™(ACT) which was carried out to determine asthma control status. And 35 healthy volunteers, with similar characteristics to the patient group in terms of age, gender and education level were enrolled.
Results: The average age of asthma patients was 34.6±10.4 years, while the average age of control group was 33.2±8.3 years and there was no statistical difference. There was no statistically difference in demographic features and laboratory parameters. All individuals records of exercise stress tests were assessed for heart rate recovery index (HRRI) and other parameters. According to statistical analysis a meaningful difference was found in terms of Peak exercise heart rate, Exercise capacity (METs), Duration of exercise, HRRI1, (respectively p=0.003; <0.001; <0.001; <0.001) whereas was no statistical difference with regard to HRRI2 HRRI3, HRRI5. Furthermore, the HRRI2, HRRI3, HRRI5 were higher in control group than asthma group.
Conclusions: In this study, we were found that the HRRI1 which is assumed to be significant predictor of cardiovascular events and mortality was impaired in patients with asthma.

Table 1. Heart rate recovery indices of the groups

	Group 1	Group 2	P value
Peak exercise heart rate (beat/min)	159.9±18.0	171.7±14.3	0.003
Exercise capacity (METs)	8.9±2.0	11.9±1.1	<0.0001
Duration of exercise, sec	437.1±90.7	639.7±147.7	<0.0001
HRRI 1st min,beats/min	20.9±10.6	31.5±12.5	<0.0001
HRRI 2nd min,beats/min	45.8±13.8	49.9±13.9	0.21
HRRI 3rd min,beats/min	54.8±13.3	59.4±11.5	0.13
HRRI 5th min,beats/min	56.8±14.5	62.3±12.8	0.09

HRRI: Heart rate recovery index; METs: Metabolic equivalent units

Coronary artery disease / Acute coronary syndrome

OP-218

miR-130b and miR-18a correlated with stenosis and lipid levels in CAD patients

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Background and Aim: Although patients with coronary artery disease (CAD) have a high mortality rate, the pathogenesis of CAD is still poorly understood. During the past decade, microRNAs (miRNAs) have emerged as new, potential diagnostic biomarkers in several diseases, including CAD. The aim of this study was to investigate the expression profiles of miRNAs in patients with CAD and non-CAD controls.
Methods: The Agilent's microarray analyses were performed to compare the plasma miRNA profile of selected individuals with CAD (n=12, ≥90% luminal stenosis narrowing and ≥33 SYNTAX score) and non-CAD (n=12, ≤20 stenosis and ≤2 SYNTAX score). Target prediction tools were utilized to identify miRNA target genes involved in the atherosclerosis pathway. Ninety-three individuals with normal coronary arteries (≤30% stenosis) and critical disease (≥50% stenosis) were recruited. Expressions of selected miRNAs were analyzed in 40 non-CAD and 53 patients with CAD using real-time PCR.
Results: We identified 6 miRNAs were downregulated in CAD patients. For further testing, the expression levels of miR-18a-3p and miR-130b-5p which found differentially expressed were analyzed using qRT-PCR. Expression levels of miR-130b were found negatively correlated with SYNTAX score and stenosis in female CAD patients (p<0.05). In addition, miR-18a and miR130b were found positively correlated with plasma HDL and inversely correlated with fasting triglyceride levels (p<0.05). In multivariate linear regression analysis adjusted for CAD family history, age, sex, and lipid-lowering drug usage, miRNAs were found correlated with HDL levels and miR-130b was found inversely correlated with stenosis and SYNTAX score (p<0.05).
Conclusions: Our findings highlight a significantly different pattern of miRNA expression in CAD patients in microarray results. These miRNAs might serve as biomarkers of CAD development and progression and warrant further attention.

Coronary artery disease / Acute coronary syndrome

OP-219

The effect of inflammation parameters on obstructive coronary artery ectasia in NSTE-ACS patients

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Background and Aim: Coronary artery ectasia (CAE) is a coronary artery disease that can progress in a wide spectrum from asymptomatic cases to stable angina pectoris or acute coronary syndrome. Although its etiopathogenesis cannot be fully elucidated, increased systemic inflammatory response with atherosclerosis is accused. CAE has been shown to increase systemic inflammation compared to obstructive coronary artery disease and normal coronaries in stable angina pectoris, however the importance of systemic inflammation in CAE disease in the course non-ST elevated acute coronary syndromes (NSTE-ACS) are uncertain. In this study, we aimed to compare the platelet-lymphocyte ratio (PLR) and CRP in CAE patients that have obstructive type lesions with isolated CAE patients in NSTE-ACS.
Methods: A total of 190 CAE patients were included in the study. 95 of these patients were isolated CAE patients and 95 of them were CAE patients that had obstructive coronary lesion (obstructive CAE). PLR and systemic inflammatory parameters such as Neutrophil/lymphocyte ratio (NLR), CRP and sedimentation of the patients were compared between the two groups.
Results: There is no significant difference between baseline demographic data of the patients (Table 1). Although there was no significant difference in most of the basic hemogram and biochemical parameters, there was a significant difference between the groups in PLR and CRP (Table 2) CRP value was found to be significantly higher in obstructive CAE than isolated CAE, 4.0 (3.0-6.0) mg/L, 5.5 (3.0-9.0) mg/L (p=0.003), respectively. PLR value was found to be significantly higher in obstructive CAE than isolated CAE, 143±76, 120±45 (p=0.015), respectively. In univariate and multivariate regression analysis(Table3), CRP and PLR have been shown to be independent predictors (β+1.144, p<0.05; β+1.006, p<0.05, respectively). When patients with Markis type 1 and type 2 ectasia are identified as severe ectasia, and patients with Markis type 3 and type 4 ectasia are identified as mild ectasia, the difference in inflammation markers is given in Table 4. CRP and PLR levels were found to be statistically different between the groups (p<0.05). (Figure 1and Figure 2) ROC analysis was performed to predict lesion status in patients with CAE and if PLR was >145.21, sensitivity was detected as 56%, specificity as 76%, area under curve as 0.64 (Figure 3).
Conclusions: To best of our knowledge, this study is the first study to evaluate systemic inflammation in obstructive CAE patients with NSTE-ACS. We have shown that systemic inflammation is increased in the presence of obstructive coronary artery disease in CAE patients.

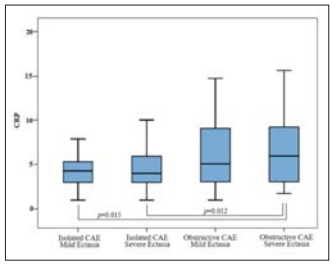

Figure 1. Comparison of CRP between groups.

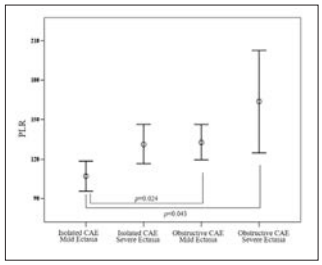

Figure 2. Comparison of PLR between groups.

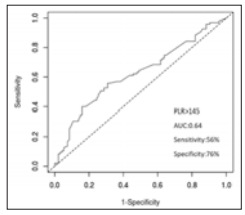

Figure 3. ROC analysis of PLR in distinguishing isolated CAE and obstructive CAE.

Table 1. Comparison of demographic and angiographic features of obstructive CAE and isolated CAE patients

variables	Isolated CAE (n=95)	Obstructive CAE (n=95)	p
Age, years	60.56±10.11	60.77±10.27	0.887
Gender (Female)	22(23.2)	13(13.7)	0.134
Smoking n_x(%)	36(37.9)	49(51.6)	0.058
DM n_x(%)	16(16.8)	19(20.0)	0.708
HL n_x(%)	25(26.3)	34(35.8)	0.158
USAP n_x(%)	34(35.8)	37(38.9)	0.653
MI n_x(%)	61(64.2)	58(61.1)	0.653
Ectasia			
LMCA n_x(%)	3(3.2)	2(2.1)	0.999
LAD n_x(%)	48(50.5)	32(33.7)	0.019
CX n_x(%)	43(45.3)	43(45.3)	0.999
RCA n_x(%)	57(60.0)	53(55.8)	0.557
Ektasia Severity			
Mild n_x(%)	48(50.5)	63(66.3)	0.027
Severe n_x(%)	47(49.5)	32(33.7)	
Obstructive Lesion			
LAD n_x(%)		71(74.7)	
Cx n_x(%)		41(43.2)	
RCA n_x(%)		51(53.7)	

DM: Diabetes mellitus, HT: Hypertension, HL: Hyperlipidemia, USAP: Unstable angina pectoris, LAD: Left anterior descending artery, MI: Myocardial infarction, CX: Left circumflex artery, RCA: Right coronary artery.

Table 2. Comparison of laboratory and echocardiographic parameters of obstructive CAE and isolated CAE patients

Variables	Isolated CAE (n=95)	Obstructive CAE (n=95)	p
RBC, (106/uL)	4.94±0.74	4.92±0.76	0.828
Neutrophil (103/uL)	5.4(4.0-8.0)	5.7(4.0-8.0)	0.722
Lymphocyte (103/uL)	2.0(2.0-3.0)	1.93(1.0-3.0)	0.999
Monocyte (103/uL)	0.5(0.0-1.0)	0.6(0.0-1.0)	0.151
Hb (g/dl)	14.7(13.0-16.0)	15.0(13.0-16.0)	0.605
Platelet (103/uL)	243.0(190.0-290.0)	254.0(209.0-306.0)	0.236
Glucose (mg/dl)	111.0(96.0-143.0)	113.0(99.0-137.0)	0.767
Triglyceride (mg/dl)	128.0(88.0-172.0)	141.0(101.0-179.0)	0.355
HDL, (mg/dl)	36.0(30.0-45.0)	35.0(29.0-40.0)	0.229
LDL (mg/dl)	112.0(81.0-144.0)	114.0(90.0-136.0)	0.589
Monocyte /HDL ratio	14.3(10.0-18.0)	16.7(12.0-21.0)	0.051
PLR	120.11±45.35	143.63±76.72	0.015
NLR	2.7(2.0-5.0)	3.0 (2.0-4.0)	0.874
Creatinin (mg/dl)	0.9(1.0-1.0)	1.0(1.0-1.0)	0.825
ALT (U/L)	23.0(15.0-31.0)	23.0(15.0-32.0)	0.816
AST (U/L)	25.0(19.0-37.0)	23.0(18.0-40.0)	0.560
Sedimentation (mm/h)	7.5(5.0-11.0)	7.0(5.0-12.0)	0.991
CRP (mg/L)	4.0(3.0-6.0)	5.5(3.0-9.0)	0.003
TSH (mIU/L)	1.1(1.0-2.0)	1.2(1.0-2.0)	0.518
EF, %	55.0(50.0-60.0)	55.0(50.0-57.0)	0.443

RBC: Red blood cell, Hb: Hemoglobin, HDL: High density lipoprotein, LDL: Low density lipoprotein, PLR: Platelet to Lymphocyte ratio, NLR: Neutrophil to Lymphocyte ratio, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, CRP: C reactive protein, TSH: Thyroid stimulating hormone, EF: Ejection fraction.

Table 3. Determination of risk factors affecting obstruction status in patients with ectasia

Variables	Univariate		Multivariate	
	Beta(%95 CI)	p	Beta(%95 CI)	p
Age	1.002(0.974-1.031)	0.886		
Gender (Male)	1.901(0.894-4.043)	0.095		
Platelet to lymphocyte ratio	1.006(1.001-1.011)	0.018	1.006(1.001-1.011)	0.045
CRP	1.066(1.009-1.126)	0.023	1.144(1.046-1.251)	0.003
DM	1.234(0.591-2.576)	0.575		
HT	0.881(0.499-1.557)	0.663		

CI: confidence interval, CRP: C reactive protein, DM: Diabetes mellitus, HT: Hypertension, PLR: Platelet to Lymphocyte ratio.

Table 4. Comparison of obstructive CAE, isolated CAE and ectasia severity

Variables	Isolated CAE Mild Ectasia (n=47)	Isolated CAE Severe Ectasia (n=48)	Obstructive CAE Mild Ectasia (n=63)	Obstructive CAE Severe Ectasia (n=32)	p
Sedimentation	7.0(4.0-11.0)	8.0(5.0-12.5)	7.0(5.0-12.0)	7.5(5.0-10.5)	0.866
CRP	4.3(3.0-5.3)a	4.0(3.0-6.0)a	5.1(3.0-9.1)ab	6.0(3.1-9.2)b	0.023
PLR	107.14±36.09a	131.39±49.76ab	132.88±51.28b	163.77±107.84b	0.003
NLR	2.4(1.7-5.0)	2.9(2.0-4.5)	3.0(2.3-4.0)	2.9(1.5-4.0)	0.649

CRP: C reactive protein, PLR: Platelet to Lymphocyte ratio, NLR: Neutrophil to Lymphocyte ratio a,b Similar letters in the same line indicate similarity between groups, different letters represent difference between groups.

Lipid / Preventive cardiology

OP-220

Empagliflozin significantly attenuates QTc prolongation in rats due to sotalol

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Background and Aim: Sotalol is a Class 3 antiarrhythmic drug and commonly used for various arrhythmia treatments in clinical use. Due to its potent potassium channel inhibition, it can prolong QT interval and lead to malignant arrhythmias. Empagliflozin is a selective SGLT-2 inhibitor used in the treatment of Type 2 diabetes andhas been shown to have positive effects on cardiovascular outcomes. Since the effect of empagliflozin on potassium channel activation is not yet known, there is no recommendation for the concomitant use of these drugs. In the present study, we aimed to evaluate possible protective effects of empagliflozin in sotalol induced QT prolongation.
Methods: Twenty-four male Wistar Alba rats (350–400 g) were randomized into four groups (1 control group and 3 experimental groups). The first (control) group (n=6) received only serum physiologic (1ml) via orogastric gavage (OG). The second (EMPA) group (n=6) received empagliflozin (10 mg/kg) via OG. The third (SOT) group (n=6) received sotalol (80 mg/kg) via OG. The fourth (EMPA+SOT) group (n=6) received empagliflozin (10 mg/kg) and sotalol (80 mg/kg) via OG. Under anesthesia; PR, QT intervals and heart rate (HR) were measured at baseline, first, second and third hours on lead II using electrocardiogram (ECG). QTc value was also calculated at the same time on lead II using ECG.
Results: In the SOT group; QT intervals, T wave durations and QTc values were found to be statistically longer than the control group, whereas HR was found to be lower than the control group (p<0.01). Empagliflozin significantly ameliorated sotalol induced QT and QTc prolongation. In the EMPA+SOT group; QT intervals, T wave durations and QTc values were significantly lower and HR was significantly higher compared to the SOT group (p<0.001, p<0.01, p<0.001, p<0.001 respectively) (Table, Figure).
Conclusions: In the present study, we detected that empagliflozin significantly ameliorates sotalol-induced QT prolongation. In addition to this, we have also shown that empagliflozin can be used safely with sotalol in clinical practice. With more clinical trials, the routine use of empagliflozin may be suggested to prevent QTc prolongation in diabetic patients receiving sotalol.

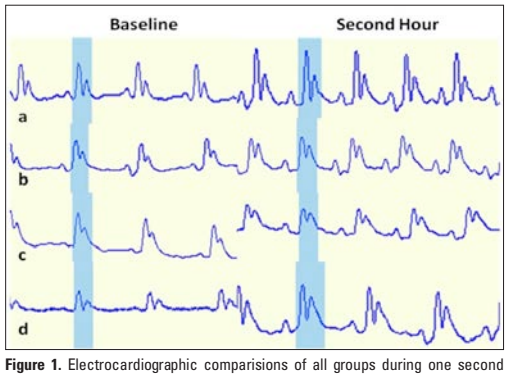

Figure 1. Electrocardiographic comparisions of all groups during one second duration in baseline and second hour a) Control b) EMPA c) EMPA+SOT d) SOT.

Table 1. PR, QT, QTc, T durations and heart rate (HR) for all groups in 2nd hour a: Control vs Sotalol; p<0.001, b: EMPA+SOT vs SOT; p<0.001, c: EMPA+SOT vs SOT; p<0.01

	Control	EMPA	SOT	EMPA+SOT	p
PR duration (ms)	54.8± 4.7	56.1± 5.5	59.6± 4.5	57.7± 3.2	0.36
QT duration (ms)	81.6 ± 5.9	73.9 ±8.1	123.5±7.7 a	83.7 ± 6.8 b	<0.001
HR (bpm)	332.0 ± 18.3	296.4±29.0	211.3±24.3 a	266.3±13.7 b	<0.001
QTc	191.54 ±10.5	163.9±18.5	231.6±22.1 a	176.1±11.6 b	<0.001
T duration (ms)	42.3±4.7	49.5±6.2	65.2±7.0 a	50.6±4.9 c	<0.001

Lipid / Preventive cardiology

OP-221

Atherogenic dyslipidemia index and its relationship with aortic valve sclerosis

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Background and Aim: Aortic valve sclerosis (AVS) is defined as calcified and thickened aortic leaflets without restriction of leaflet motion. AVS is associated with an increased risk of acute coronary syndrome, congestive heart failure, and cardiovascular mortality. Dyslipidemia is considered an independent risk factor for atherosclerotic heart disease (AHD). Although the relationship between atherogenic dyslipidemia and AHD is well known, there is not sufficient information about atherogenic dyslipidemia with AVS. Therefore, the relationship between AVS and atherogenic dyslipidemia was evaluated in the present study.
Methods: A total of 187 patients who were admitted for routine check-up and examined at outpatient clinics were enrolled into this cross-sectional study. The patient group included 92 patients with AVS. The control group consisted of 95 age- and gender-matched healthy subjects. Serum levels of total cholesterol (TC), triglyceride (TG), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), and atherogenic indices (atherogenic dyslipidemia index, non HDL-C, atherogenic coefficient, cardiac risk ratios 1 and 2) were analyzed.
Results: The mean age of the patients was 62.8±10.4 years, and 67.6% were male. The atherogenic dyslipidemia index, non HDL-C, atherogenic coefficient, and cardiac risk ratios 1 and 2 were significantly higher in the AVS (+) group than the control group (p<0.001; p=0.005; p=0.018; p=0.024; p=0.043, respectively). In multivariate logistic regression analysis; atherogenic dyslipidemia index (p<0.001, Odds ratio (OR) = 2.73, 95% Confidence interval (C.I.) = 1.44–7.56) was found to be independent predictor of AVS.
Conclusions: The present study suggest that increased atherogenic indices (atherogenic dyslipidemia index, non HDL-C, atherogenic coefficient, cardiac risk ratios 1 and 2) are associated in the pathophysiologic process of AVS. Atherogenic dyslipidemia may be used as a predictor for AVS. Further studies are needed to demonstrate the pathophysiology and clinical outcomes of AVS.

Table 1. Clinical characteristics

	Control group (n = 95)	AVS (+) group (n = 92)	P value
Age	62.5 ± 10.6	63.2 ± 10.1	0.482
Male gender, n (%)	65 (68.4)	62 (67.3)	0.733
BMI (kg/m2)	27.6 ± 4.4	28.8 ± 4.6	0.027
Heart rate (bpm)	75 ± 12	78 ± 10	0.209
Systolic BP (mmHg)	127.4 ± 9.7	129.8 ± 9.4	0.414
Diastolic BP (mmHg)	77.3 ± 8.6	76.9 ± 8.8	0.826
Hypertension, n (%)	53 (55.7)	55 (59.7)	0.041
Diabetes Mellitus, n (%)	10 (10.5)	11 (11.9)	0.374
Hyperlipidemia, n (%)	14 (14.7)	26 (28.2)	0.018
Smoking, n (%)	27 (28.4)	29 (31.5)	0.077

Table 2. Dyslipidemic profile of the study population

	Control group (n = 95)	AVS (+) group (n = 92)	p-Value
TC (mg/dl)	189.6 ± 42.7	233.6 ± 54.5	0.002
TG (mg/dl)	139.5 ± 91.6	255.3 ± 94.9	< 0.001
LDL-C (mg/dl)	88.3 ± 36.2	120.4 ± 48.4	0.001
HDL-C (mg/dl)	42.4 ± 6.2	36.6 ± 5.7	0.025
Atherogenic Dyslipidemia Index (TG/HDL-C)	3.31 ± 2.52	7.08 ± 4.21	< 0.001
Non HDL-C (TC - HDL-C) (mg/dl)	147.2 ± 36.5	197 ± 48.8	0.005
Atherogenic Coefficient (Non HDL-C/HDL-C)	3.47 ± 1.18	5.38 ± 2.23	0.018
Cardiac Risk Ratio 1 (TC/HDL-C)	4.47 ± 1.43	6.38 ± 2.55	0.024
Cardiac Risk Ratio 2 (LDL/HDL-C)	2.08 ± 1.05	3.28 ± 2.17	0.043

Table 3. The independent predictors of AVS

Variable	P	Odss Ratio (%95 C.I.)
Atherogenic dyslipidemia index	<0.001	2.38 (1.39 – 8.94)
TG	0.006	1.54 (1.27 – 3.45)
Hyperlipidemia	0.068	1.11 (1.07 – 1.88)
Non HDL-C	0.083	1.01 (0.79 – 1.53)
BMI	0.116	0.92 (0.71 – 1.28)
Hypertension	0.308	0.85 (0.66 – 1.04)

Lipid / Preventive cardiology

OP-222

The triglyceride glucose index, a predictor of insulin resistance, is associated with subclinical atherosclerosis

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Background and Aim: İnsulin resistance(IR) is among one of the most important risk factors that accelerate atherosclerosis. The goal of this study is to investigate the relationship between the TyG index and subclinical atherosclerosis which is measured using PWV, in a non-diabetic asymptomatic Turkish population.
Methods: Non-diabetic, healthy patients with no previous history of coronary heart disease were enrolled into this study (n=1095). Subclinical atherosclerosis was detected by measuring PWV. The TyG index was calculated using the following equation; log [fasting triglycerides (mg/dl) x fasting glucose (mg/dl)/2]. The study population was divided into 2 groups based on their TyG index.
Results: The high TyG index group had higher PWV, cPWV, LVMI, BMI, rates of HT, and were predominately male. Age, gender, BUN level, and TyG index were detected as independent risk factors of PWV in logistic regression analysis.
Conclusions: This study has shown a relationship between TyG index and subclinical atherosclerosis, even in patients without evident risk of atherosclerotic cardiovascular disease.

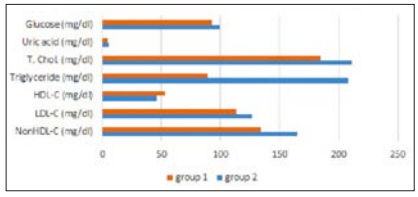

Figure 1. Comparison of metabolic parameters between groups.

Table 1. Baseline characteristics of study groups according to TyG index levels

Variables	Group 1 (TyG Index≤8,65)	Group 2 (TyG Index>8,65)	P Value
N(1095)	461	634	
Male, n (%)	%29,7	%33	<0,01
Age, years	49,6±15,5	52,3±12,9	<0,01
Pwv (m/s)	7,2±1,9	7,6±1,6	<0,01
cPwv n (%)	%31,0	%37,2	0,03
Glucose (mg/dl)	92,5±10,0	99,2±11,3	<0,01
UAE n (%)	%8,5	%12,5	0,04
Uric acid (mg/dl)	4,7±1,3	5,3±1,3	<0,01
T. Chol. (mg/dl)	184,8±38,1	210,7±41,8	<0,01
Triglyceride (mg/dl)	89,3±23,4	208,2±111,4	<0,01
HDL-C (mg/dl)	53,0±13,3	45,6±10,6	<0,01
LDL-C (mg/dl)	113,5±33,0	126,2±36,9	<0,01
NonHDL-C (mg/dl)	134,2±35,1	164,5±38,8	<0,01
TC/HDL-C	3,6±0,8	4,7±1,1	<0,01
TG/HDL-C	1,8±0,7	4,7±2,5	<0,01
LDL-C/HDL-C	2,2±0,8	2,7±0,9	<0,01
Non–HDL-C/HDL-C	2,6±0,9	3,7±1,0	<0,01
TyG Index	8,2±0,2	9,1±0,3	<0,01

Lipid / Preventive cardiology

OP-223

Serum endocan as a marker of endothelial dysfunction in subjects with metabolically healthy obesity

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Background and Aim: Metabolically healthy normal weight (MHN) and metabolically healthy obesity (MHO) phenotypes are previously known to be benign metabolic status. However, recent studies showed that the presence of MHO was associated with worse cardiovascular (CV) prognosis. However, the underlying mechanism is not precise. Endocan is known as an essential marker of endothelial dysfunction. Thus, we aimed to assess serum endocan levels between subjects with MHN vs. MHO.
Methods: In this cross-sectional study, a total of 40 subjects with MHN and 40 subjects with MHO who admitted to our outpatient clinic for check-up were enrolled. The MHO was defined as the body mass index (BMI) of ≥30 kg/m2 in the absence of metabolic syndrome parameters according to the Harmonized International Diabetes Federation criteria. Serum endocan and high sensitive C-reactive protein (hsCRP) levels were measured in all participants.
Results: Baseline characteristics except for BMI, waist circumference (WC), serum endocan levels, and hs-CRP were similar between study groups. However, BMI, WC, serum endocan, and hsCRP levels were significantly higher in subjects with MHO. Serum endocan levels showed significantly positive correlation with hsCRP (r=0.528, p<0.001), WC (r=0.370, p=0.001), and BMI (r=0.734, p<0.001). Serum endocan level of >0.479 ng/mL predicted the presence of MHO with sensitivity of 90% and specificity of 90% (AUC: 0.96, 95% CI: 0.92-1.00, p<0.001).
Conclusions: Serum endocan level, as a marker of endothelial dysfunction, was significantly higher in subjects with MHO. As the endothelial dysfunction is the initial insult in CV diseases, the positive correlation between serum endocan levels with body fat (BMI & WC) and pro-inflammatory marker (hsCRP) may explain the underlying mechanism of adverse CV outcomes in MHO status.

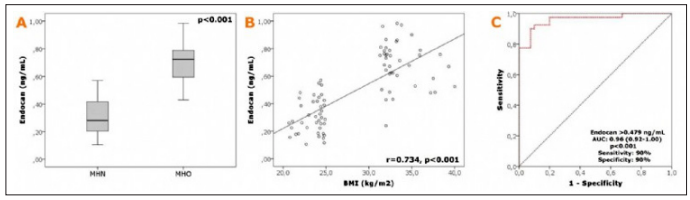

Figure 1. (a) Serum endocan levels were significantly higher in subjects with MHO compared to MHN **(b)** Correlation analysis showing the association of serum endocan levels with BMI **(c)** ROC analysis showing the predictive value of serum endocan for the presence of MHO.

Table 1. Baseline demographical, clinical, and laboratory characteristics of the study population (n=80)

Variables	MHN (n=40)	MHO (n=40)	p-value
Age, years	56.5±10.4	56.0±9.5	0.825
Male gender	21 (52.5%)	22 (55.0%)	1.000
BMI, kg/m2	23.5±1.3	33.6±2.4	<0.001
WC, cm	85.4±7.0	90.6±8.8	0.005
Hyperlipidemia	9 (22.5%)	10 (25%)	1.000
Diabetes mellitus	0 (0.0%)	0 (0.0%)	1.000
Hypertension	0 (0.0%)	0 (0.0%)	1.000
Smoking	8 (20.0%)	11 (27.5%)	0.600
SBP, mmHg	120.9±7.7	123.6±10.4	0.184
DBP, mmHg	74.8±5.6	76.5±6.6	0.231
Triglyceride, mg/dL	141.5±79.2	157.3±74.6	0.362
HDL-C, mg/dL	53.1±13.1	48.9±12.8	0.160
LDL-C, mg/dL	137.8±41.5	123.2±37.0	0.100
Fasting plasma glucose, mg/dL	88.2±9.9	86.7±10.1	0.496
Serum creatinine, mg/dL	0.82±0.32	0.78±0.24	0.484
Serum uric acid, mg/dL	5.23±1.30	5.67±1.44	0.149
Endocan, ng/mL	0.31±0.13	0.69±0.16	<0.001
hsCRP, mg/L	0.25±0.07	0.38±0.11	<0.001
Medications Statin	8 (20.0%)	9 (22.5%)	1.000

Lipid / Preventive cardiology

OP-224

Comparison of 2019 vs. 2016 ESC/EAS dyslipidemia guidelines for primary prevention of atherosclerotic cardiovascular disease

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Background and Aim: Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality. Primary cardiovascular prevention with statin therapy is the most important step for decreasing of CVD. However, which patient should be received on statin therapy is still controversial in primary prevention. Guidelines update and improve their suggestions on this issue. The ESC/EAS dyslipidemia guidelines was updated in August 2019. The purpose of this study is to compare statin treatment recommendations of ESC/EAS 2019 and 2016 guidelines.
Methods: 920 consecutive non-diabetic statin-naive patients who presented with first acute coronary syndrome (ACS) were included (Figure 1). We assumed they had undergone a health check the day before their MI and estimated the predicted risk. Their CVD risk was calculated HeartScore-High Risk charts both 2016 and 2019 version. Statin eligibility determined with 2019 and 2016 ESC dyslipidemia guidelines. "Lifestyle intervention and concomitant drug intervention", statin therapy recommendation was considered "concomitant" and "lifestyle intervention consider adding drug if uncontrolled" recommendation was considered "added" recommendation.
Results: Baseline characteristics are shown in Table 1. The positive predictive value of the 2019 ESC dyslipidemia guideline for concomitant statin therapy recommendation was 56.6% (n=521) (2016 guidelines n 437 47.5% p<0.001) (Figure 2).The positive predictive value of the 2019 guideline increased 9.9% in the concomitant recommendation and 3.8 % in the overall (concomitant + added) recommendation. As the patient age increases, the positive predictive value of the 2019 guidelines for concomitant treatment increases more (Figure 3). Raising the statin treatment age threshold in 2019 constitutes the main difference. When patients

in the common indication age range (40-65 years) are examined in both guidelines, the difference between the concomitant indications decreases (49% 2019, 45% 2016 guidelines, p<0.01). Concomitant statin treatment recommendation independent of risk level for LDL-C >190 mg/dl was found to provide concomitant indications in only 21 (2.3%) patients.The vast majority of patients are classified in the moderate risk group. This rate decreased markedly in the 2019 guideline update (31% 2019; 45% 2016 guidelines p<0.01). In the 2019 guideline update, the classification rates of patients in the very high risk group increased (28.3 % 2019 guidelines, 15.3 2016 guidelines p<0.01).

Conclusions: The 2019 dyslipidemia guidelines increased the positive predictive value of statin therapy eligibility. However, there is still a deficiency in comcomitant statin treatment recommendations, especially in individuals who are calculated as the moderate risk group.

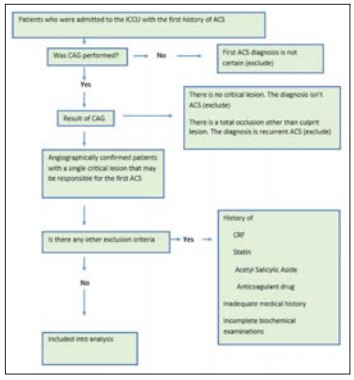

Figure 1. Flow scheme and inclusion, exclusion procedures.

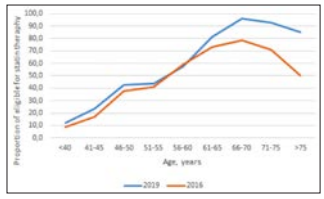

Figure 3. Concomitant Statin eligibility of the 2019 and 2016 European Society of Cardiology/ European Atherosclerosis Society guidelines stratified by 5-year age groups.

Positive predictive results according to 2019 ESC / EAS Dyslipidemias Guidelines

Total CV risk (SCORE)	n (%)	LDL – C levels (mg/dl) <55	55 to < 70	70 to <100	100 to <116	116 to < 190	≥190	Proportion eligible for statin therapy Concomitant	Added	Concomitant+Added
			n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
<1 %	32 (3.5)	0	1 (0.1)	9 (1.0)	7 (0.8)	14 (1.5)	1 (0.1)	1(0.1)	14 (1.5)	15 (1.6)
COR / LOE			I/C	I/C	I/C	IIa/A	IIa/A			
Mean age (years) 43,1 ± 8.1				37	41 ± 6,3		39			
≥1 to <5	342 (37.2)	2 (0.2)	13 (1.4)	51 (5.5)	52 (5.7)	204 (22.2)	26 (2.8)	26 (2.8)	256 (27.8)	276 (30.0)
COR / LOE			I/C	IIa/A	IIa/A	IIa/A	IIa/A			
Mean age (years) 50.8 ±1.3			47.5 ± 6,5	52.6 ± 12.3	48,7 ± 10.8	49,7 ± 10.8	45.2 ± 6.9			
≥5 to <10	286 (31.1)	0	3 (0.3)	36 (4.22)	32 (3.5)	160 (17.4)	55 (6.0)	247 (26.8)	36 (3.9)	283 (30.8)
COR / LOE			IIa/A	IIa/A	IIa/A	I/A	I/A			
Mean age (years) 61.5 ±11.5			71 ± 21.4	65.1 ± 9.3	61.5 ± 9.5	57.9 ± 7.4	53.4 ± 4.6			
≥ 10	260 (28.3)	2 (0.2)	5 (0.5)	58 (6.3)	39 (4.2)	174 (18.9)	32 (3.5)	233 (27.3)	5 (0.5)	258 (28.0)
COR / LOE			IIa/A	IIa/A	/A	/A	/A			
Mean age (years) 66.4 ± 8.9				66.0 ± 4.6	65,7 ± 7,03	63,3 ± 4,8	61,5 ± 7,4			
Total	920 (100)	4 (0.4)	22 (2.4)	122 (13.3)	131 (14.2)	555 (60.3)	86 (9.3)	521 (56.6)	311 (33.8)	832 (90.4)

Positive predictive results according to 2016 ESC / EAS Dyslipidemias Guidelines

Total CV risk (SCORE)	n (%)	LDL – C levels (mg/dl) < 70	70 to <100	100 to <155	155 to < 190	≥190	Proportion eligible for statin therapy Concomitant	Added	Concomitant+Added
		n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
<1 %	30 (3.3)	1 (0.1)	9 (1.0)	18 (2.0)	1 (0.1)	1 (0.1)	0	1 (0.11)	1 (0.1)
COR / LOE		I/C	I/C	I/C	I/C	IIa/A			
Mean age (years) 43,1 ± 8.1		1 (0.1)	37	41 ± 6,3	0	39			
≥1 to <5	415 (45.1)	17 (1.4)	71 (7.1)	232 (25.2)	75 (8.2)	20 (2.2)	0	327 (35.5)	327 (35.5)
COR / LOE		I/C	I/C	IIa/A	IIa/A	I/A			
Mean age (years) 50.8 ±1.3		47.5 ± 6,5	52.6 ± 12.3	48,7 ± 10.8	49,7 ± 10.8	45.2 ± 6.9			
≥5 to <10	334 (35.9)	6 (0.6)	30 (3.3)	157 (17.0)	109 (11.8)	32 (3.5)	298 (32.4)	30 (3.3)	328 (35.7)
COR / LOE		IIa/A	IIa/A	IIa/A	I/A	I/A			
Mean age (years) 61.5 ±11.5		71 ± 21.4	65.1 ± 9.3	61.5 ± 9.5	57.9 ± 7.4	53.4 ± 4.6			
≥ 10	141 (15.3)	2 (0.2)	37 (4.0)	69 (7.5)	43 (4.6)	12 (1.4)	139 (15.1)	2 (0.2)	141 (15.3)
COR / LOE		IIa/A	IIa/A	/A	/A	/A			
Mean age (years) 66.4 ± 8.9			66.0 ± 4.6	65,7 ± 7,03	63,3 ± 4,8	61,5 ± 7,4			
Total	901 (100)	26 (2.8)	122 (13.3)	507 (55.1)	179 (19.5)	86 (9.3)	437 (47.3)	360 (39.1)	797 (86.6)

Figure 2. Comparison statin eligibility by the 2016 and 2019 European Society of Cardiology/European Atherosclerosis Society (ESC/EAS) dyslipidaemia guidelines. COR: Class of recommendation, LDL-C: Low density lipoprotein cholesterol, LOE Level of evidence.

Table 1. Baseline characteristics

	ALL	2019 statin concomitant	2016 concomitant	Only 2019 concomitant
Individuals n %	920 (100)	521 (57)	437 (48)	84 (9)
Male gender (%)	83	90	90	85
Age (years)	56 (48-65)	61 (53-69)	62(54-69)	47 (41-50)
Systolic blood pressure (mmHg)	134 (124-140)	137 (128-144)	137 (128-145)	134 (128-140)
Diastolic blood pressure (mmHg)	80 (75-85)	81 (76-87)	81 (76-89)	81 (78-91)
Total Cholesterol(mg/dl)	204 (176-236)	216(189-252)	213 (187-245)	270(263-280)
LDL cholesterol (mg/dl)	132 (111-156)	145 (124-174)	142 (122-170)	200 (194-213)
HDL cholesterol (mg/dl)	42 (36-49)	43 (37-49)	43(37-49)	43(38-53)
Non HDL cholesterol (mg/dl)	159 (133-189)	172 (147-203)	169 (146-197)	227(223-242)
Triglyceride (mg/dl)	120 (84-182)	114(81-172)	118 (84-182)	134(74-194)
SCORE 10-year fatal ASCVD (%)	5 (2-7)	7 (5-10)	7 (5-10)	2(2-4)
Current smoke (%)	58	55	62	67
Age 40-65 years				
Individuals n %	648	320 (49)	291 (45)	29 (4)
Male gender (%)	87	93	94	84
Age (years)	53 (47- 59)	57 (52-61)	57(52-61)	48 (42-50)
Systolic blood pressure (mmHg)	134 (126-140)	137 (128-144)	137 (128-145)	134 (124-140)
Diastolic blood pressure (mmHg)	81 (75-85)	81(77-90)	81 (77-90)	81 (76-92)
Total Cholesterol(mg/dl)	206(179-240)	220 (193-261)	215 (191-251)	270 (257-287)
LDL cholesterol (mg/dl)	134 (111-163)	148 (127-185)	144 (125-174)	200 (194-212)
HDL cholesterol (mg/dl)	41(35-48)	41 (36-48)	41 (36-48)	43 (39-53)
Non HDL cholesterol (mg/dl)	161 (134-193)	175 (150-208)	171 (148-204)	226 (222-239)
Triglyceride (mg/dl)	126 (85-192)	129 (89-193)	126 (89-194)	132 (68-172)
SCORE 10-year fatal ASCVD (%)	4(2-7)	7 (5-9)	7 (5-9)	3 (2-4)
Current smoke (%)	61	73	74	64

HDL: High density lipoprotein LDL: Low density lipoprotein SCORE: Systematic Coronary Risk Estimation.

Lipid / Preventive cardiology

OP-225

Effects of traumatic stress on endothelial and cardiac autonomic functions

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Background and Aim: Traumatic stress is a psychobiological response to a noxious event which is of an extraordinary scope or which exceeds the individual's capacity of coping. This stressor event may have been experienced or witnessed by the individual. Recent evidence suggest that posttraumatic stress disorder (PTSD) and other trauma-related psychiatric disorders increase the risk of cardiovascular diseases. This study aimed to investigate the association of psychiatric symptoms related to traumatic stress with the endothelial function and cardiac autonomic functions.

Methods: One hundred thirty two adults were included into this study. The self-report PTSD Symptom Checklist-5 (PCL-5) questionnaire screening potentially traumatic life events and related symptoms was administered to all participants. All participants' carotid intima media thickness and flow mediated dilatation were measured for evaluating the endothelial function. Additionally, ewing test, heart rate, blood pressure and biochemical parameters were evaluated. Those who reported at least one stressor event of traumatic scope (n=48, 36%) were compared to those who did not (n=84, 64%).

Results: Carotid intima media thickness 61 mm (32-82) vs 41 mm (28-69), p<0.0005, systolic blood pressure 125 mmHg (109-130) vs 114 mmHg (95-130), p<0.0005, morning heart rate 81bpm (61-96) vs 77 bpm (50-100), p=0.042, serum C-reactive protein CRP, 1.40 mg/l (0.30-5.30) vs 0.30 mg/l (0.30-4.10), p=0.014 values were found significantly increased in the traumatic stressor group compared to non-stressor group. Flow mediated dilatation values 8% (4-16) vs 11% (4-23), p<0.0005 were lower in the traumatic stressor group than non-stressor group. Traumatic stressor group was positively correlated with carotid intima media thickness (r=0.56, p<0.0005), CRP (r=0.22, p=0.03), systolic blood pressure (r=0.41, p<0.0005). Traumatic stressor group was negatively correlated with flow-mediated dilatation (r=-0.35, p<0.0005). According to the logistic regression analysis, systolic blood pressure, carotid intima media thickness, flow mediated dilatation values were independently associated with traumatic stressor group. The Diagnostic and Statistical Manual of Mental Disorders (DSM-5) diagnosis of PTSD (n=19, 14%)) and PTSD symptom total scores (mean=22.3, SD=16.1) were, however, not associated with any of the assessed biological variables.

Conclusions: Having a stressful experience of traumatic scope was independently related with systolic blood pressure, carotid intima media thickness, flow mediated dilatation. Morning heart rate, serum CRP values were found significantly different between the participants with and without a history of traumatic stress. Traumatic experiences may have an important role in development of cardiovascular disease and risk factors.

Lipid / Preventive cardiology

OP-226

Mediterranean diet and DASH diet can be protective for ascending aortic aneurysm

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Background and Aim: Out of one hundred thousand people randomly selected, 5 or 10 may have ascending aortic aneurysms (AAA). Ascending aorta dilation may potentially occur when the extracellular matrix is deformed and destructed, and in long periods, the aortic wall is exposed to hemodynamic force, for instance, led by high blood pressure. In the pathophysiological aspect, although which mechanisms may cause AAAs has been now better understood by means of recent studies, there are limited biomarkers for risk assessment of aneurysms. Dietary Approaches to Stop Hypertension (DASH) diet is a dietary model that recommends moderate sodium intake and low intake of saturated fat, cholesterol and simple sugar, rich in fruits, vegetables, whole grains, and low-fat dairy products. It is reported to be sustainable throughout the life, since no food group or food is prohibited in the DASH diet and there are no difficulties in its administration. DASH diet; It is widely recommended for individuals with hypertension, and this diet aims to balance blood pressure. The DASH diet is thought to have potential benefits in terms of diabetes, cancer and heart disease as well as balancing blood pressure. Today, the typical Mediterranean diet (MD) is nutritionally safe and adequate, rich in biodiversity, eco-friendly, economically beneficial (supportive of local food production) and among sustainable diets with its socio-cultural characteristics. MD is characterized by high amounts of vegetable intake, fruits, hazelnut and unsaturated fatty acids, moderate consumption of dairy products (mainly cheese and yoghurt) and fish and low amounts of red meat and unprocessed products. According to many prospective studies, MD decreased the mortality caused by cardiovascular diseases and was associated with a decrease in the incidence of myocardial infarction and stroke. The benefits of both diet regimens on hypertension and cardiovascular diseases are known. In the light of this information, we aimed to investigate the relationship between DASH and MD with AAA.

Methods: 30 consecutive patients with AAA and 50 consecutive patients with normal ascending aortic diameter were recruited into the study by comprehensive transthoracic echocardiography. MD and DASH diet score questionnaires were performed to all patients. All data were compared between groups.

Results: Hs-CRP levels (p=0.015) hypertension rate (p=0.014) were higher in AAA group. Mean MD adherence score (p<0.001) and mean DASH diet score (p<0.001) was lower in AAA group. Multivariable logistic regression analyses demonstrated that MD adherence score (p=0.017), DASH diet score (p<0.001), Hs-CRP levels (p=0.045) and hypertension (p=0.038) were independently associated with AAA.

Conclusions: In our study, it was determined that MD and DASH diet could have potential positive effects on AAA. However, larger and more comprehensive studies are needed to prove our hypothesis.

DASH nutrient	DASH score target values (1 point)	Intermediate target values (0.5 points)
Sodium (mg/day)	<2300.0	2300–2650
Cholesterol (mg/day)	<149.1	149.1-224.7
Saturated fat (% of Kcal/day)	<6.0	6-11
Total fat (% of Kcal/day)	<27.0	27-32
Protein (% of Kcal/day)	>18.0	16.5-18.0
Calcium (mg/day)	1240.0	842.3–1240.0
Magnesium (mg/day)	>496.7	330.3–496.7
Potassium (mg/day)	>4673.3	3198.3–4673.3
Fiber (g/day)	>30	19.5-30.0

Notes: DASH score targets are based on a 2100 Kcal/day diet and the linear index model introduced by Lin et al. (1999). If the DASH nutrient intake does not meet either target, asit is scored as 0.

Figure 1. Nutrient targets for Dietary Approaches to Stop Hypertension (DASH) accordance.

Questions	Criteria for 1 point
1. Do you use olive oil as main culinary fat?	Yes
2. How much olive oil do you consume in a given day (including oil used for frying, salads, out-of-house meals, etc.)?	≥4 tbsp
3. How many vegetable servings do you consume per day? (1 serving : 200 g [consider side dishes as half a serving])	≥2 (≥1 portion raw or as a salad)
4. How many fruit units (including natural fruit juices) do you consume per day?	≥3
5. How many servings of red meat, hamburger, or meat products (ham, sausage, etc.) do you consume per day? (1 serving: 100–150 g)	<1
6. How many servings of butter, margarine, or cream do you consume per day? (1 serving: 12 g)	<1
7. How many sweet or carbonated beverages do you drink per day?	<1
8. How much wine do you drink per week?	≥7 glasses
9. How many servings of legumes do you consume per week? (1 serving : 150 g)	≥3
10. How many servings of fish or shellfish do you consume per week? (1 serving 100–150 g of fish or 4–5 units or 200 g of shellfish)	≥3
11. How many times per week do you consume commercial sweets or pastries (not homemade), such as cakes, cookies, biscuits, orcustard?	<3
12. How many servings of nuts (including peanuts) do you consume per week? (1 serving 30 g)	≥3
13. Do you preferentially consume chicken, turkey, or rabbit meat instead of veal, pork, hamburger, or sausage?	Yes
14. How many times per week do you consume vegetables, pasta, rice, or other dishes seasoned with sofrito (sauce made with tomatoand onion, leek, or garlic and simmered with olive oil)?	≥2

Figure 2. Validated 14-item Questionnaire of Mediterranean diet adherence.

Table 1. Clinical and demographic characteristics of the study population

Variables	Control group (n=50)	Case group (n=30)	p value
Age, years	60.28 ± 15.36	57.83 ±11.64	0.702
Female, n(%)	13 (26.0%)	7 (23.3%)	0.790
Body mass index, kg/m2	27.31 ± 3.45	28.19± 3.01	0.491
Diabetes Mellitus, n(%)	8 (16.0%)	5 (16.7%)	0.938
Hypertension, n(%)	34 (68.0%)	12 (40.0%)	0.014
Hyperlipidemia, n(%)	22 (44.0%)	12 (40.0%)	0.726
Smoking, n(%)	26 (52.0%)	18 (60.0%)	0.486
Coronary artery disease, n(%)	12 (24.0%)	6 (20.0%)	0.678
Peripheral vascular disease, n(%)	1 (2.0%)	0 (0.0%)	0.436
Atrial fibrillation, n(%)	12 (24.0%)	6 (20.0%)	0.678
Beta Blocker usage, n(%)	7 (14.0%)	14 (13.3%)	0.933

Data are given as mean ± SD, n or median (interquartile range).

Table 2. Echocardiographic characteristics of the study population

Variables	Control group (n=50)	Case group (n=30)	p value
LVEF (%)	58.9 ± 3.19	56.7 ± 8.2	0.103
ARVC (mm)	0.3 ± 0.1	1.2 ± 0.4	<0.001
Aortic annulus diameter (mm)	2.20 ± 0.11	2.41 ± 0.34	<0.001
Sinus valsalva diameter (mm)	3.37 ± 0.31	4.01 ± 0.89	<0.001
Ascending aorta diameter (mm)	3.21 ± 0.24	3.96 ± 0.79	<0.001
Arcus aorta diameter (mm)	2.41 ± 0.15	2.7 ± 0.56	<0.001
Bicuspid aortic valve, n(%)	1 (2.0%)	4 (13.3%)	<0.001

Data are given as mean ± SD, n or median (interquartile range). ARVC, vena contracta width of aortic regurgitation; LVEF, left ventricular ejection fraction.

Table 3. Blood and diet parameters of the study population

Variables	Control group (n=50)	Case group (n=30)	p value
Glucose, mg/dL	107.1 ± 30.6	104.2 ± 35.3	0.701
Creatinine, mg/dL	1.00 ± 0.16	1.04 ± 0.24	0.721
Uric Acid, mg/dl	5.60 ± 2.06	7.01 ± 1.82	0.429
Hemoglobin, g/dL	14.1 ± 1.6	14.0 ± 1.9	0.860
WBC,10^3/mm³	8.4 ± 2.1	7.9 ± 3.0	0.445
Neutrophil,10^3/mm³	5.4 ± 2.2	6.1 ± 3.3	0.579
Lymphocyte,10^3/mm³	2.0 ± 0.8	2.1 ± 0.8	0.593
Monocyte,10^3/mm³	0.6 ± 0.3	0.6 ± 0.3	0.882
Platelet, 10^3/mm³	221.4 ± 63.6	230.8 ± 63.0	0.527
Hs-CRP,mg/L	2.6 ± 1.5	4.7 ± 2.1	0.015
Total cholesterol, mg/dL	199.4 ± 40.7	189.5 ± 35.1	0.270
LDL-C, mg/dL	120.9 ± 38.4	108.8 ± 32.4	0.272
HDL-C, mg/dL	46.2 ± 9.4	43.1 ± 13.1	0.356
Triglyceride, mg/dL	158.0 ± 79.1	158.7 ± 87.9	0.460
Mediterranean Diet Score	6.01 ± 2.96	4.09 ± 1.53	<0.001
DASH diet score	3.12 ± 0.71	2.31 ± 0.38	<0.001

Data are given as mean ± SD, n or median (interquartile range). WBC, white blood cell; Hs-CRP, high-sensitivity C-reactive protein; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MHR, Monocyte – high-density lipoprotein ratio; DASH, Dietary Approaches to Stop Hypertension.

Table 4. Multivariate linear regression analysis showing the predictors for the Ascending aortic dilatation

Variables	Univariable Beta (95% CI)	p value	Multivariable Beta (95% CI)	p value
Hypertension	0.314 (0.122-0.804)	0.016	0.356 (0.134-0.944)	0.038
Hs-CRP,	0.801 (0.663-0.969)	0.023	0.817 (0.676-0.988)	0.045
Mediterranean Diet Score	0.401 (0.160-0.643)	0.005	0.480 (0.185-0.775)	0.017
DASH diet score	0.603 (0.351-0.855)	<0.001	0.701 (0.386-1.016)	<0.001

CI, confidence interval; OR, Odds ratio; Hs-CRP, high-sensitivity C-reactive protein; MHR, Monocyte – high-density lipoprotein ratio; DASH, Dietary Approaches to Stop Hypertension.

Lipid / Preventive cardiology

OP-227

Analysis of lipid profile in patients with coronary artery disease: Do we reach target levels during treatment?

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Background and Aim: Statins are the cornerstone of the treatment of dyslipidemia in patients with coronary artery disease (CAD). Despite presence of large number of studies proving their beneficial effects on mortality and clear definition of treatment targets in current guidelines, real-life achievement of specified targets is still uncertain. In this study, we aimed to determine the level of guideline adherence in choosing the convenient statin regimen; patient adherence to treatment and achievement of LDL targets in patients with CAD.
Methods: In this observational study, 450 patients (38.2% female) with angiographically confirmed diagnosis of CAD and no prior statin treatment were consecutively enrolled. The data on demographic characteristics, risk factors, lipid profile, presence of obstructive CAD and treatment regimen were recorded at the time of presentation; after which patients were re-evaluated in 8 (5-12) weeks. On the follow-up visit, control lipid parameters, drug adherence and possible adverse effects were recorded.
Results: Physicians' adherence to guidelines: Our study has shown that 51.9% of the patients underwent with appropriate intensity statin therapy, while 48% of patients were under-treated with lower-intensity statin than needed. Factors associated with undertreatment were DM, HT, clinical presentation, presence of obstructive CAD and baseline urea and LDL levels (all p<0.05). Independent predictors of physicians' adherence to guidelines were DM, acute coronary syndromes, presence of obstructive CAD, baseline urea and LDL levels (Tables 1 and 2). Patients' adherence to treatment: Patients with myocardial infarction as clinical presentation, those with obstructive CAD (72.2% vs 46.8%, p<0.001), those without side effect such as myalgia (37.2 vs 10.1%, p<0.001) and gastrointestinal side effects (14.9 vs 2.2%, p<0.001) were more adherent to antihyperlipidemic treatment (Table 3). Achievement of treatment targets: On follow-up visits, only 28% of the patients has reached target LDL levels. Table 4 summarizes the clinical and demographic variables with regard to achievement of target LDL. None of the patients with drug nonadherence could reach the target LDL. Presence of obstructive CAD (OR:1.99 (1.216-3.282), p=0.006), baseline LDL (OR: 0.99 (0.985-0.998), p=0.013) and myalgia (OR: 0.34 (0.161-0.71); p=0.005) were independent predictors of achievement of target LDL.
Conclusions: Although target treatment levels for high-risk patients are clearly explained in the guidelines, real-life achievement of treatment targets is still limited. This can be attributed to a variety of causes, but the most important factors are likely to be improper intensity dosage prescribed by physicians, patient nonadherence and personal variations.

Table 1. Clinical and demographic characteristics of patients with regard to Physicians' adherence to guidelines

		Treatment Adherent with Guidelines (n:229)	Treatment NON-Adherent with Guidelines(n:212)	P value
Age n (%)		62(35-85)	64 (30-86)	0,092
Female n (%)		80 (34,9%)	90 (42,4%)	0,105
Diabetes mellitus, n (%)		54 (23,6%)	84 (39,6%)	**<0,001**
Hypertension n (%)		124 (54,1%)	150 (70,7%)	**<0,001**
Family story n (%)		125 (54,6%)	105 (49,5%)	0,288
Smoke n (%)		106 (46,3%)	84 (39,6%)	0,158
Previous CABG n (%)		8 (3,5%)	6 (2,8%)	0,691
Previous PCI n (%)		35 (15,3%)	37 (17,4%)	0,538
Previous SVO n (%)		6 (2,6%)	10 (4,7%)	0,239
Clinic	SAP n (%)	**137 (59,8%)**	**171 (80,6%)**	**<0,001**
	USAP n (%)	24 (10,4%)	18 (8,4%)	
	NSTEMI n (%)	**40 (17,4%)**	**20 (9,4%)**	
	STEMI n (%)	**28 (12,2%)**	**3 (1,4%)**	
Obstructive KAH		172 (75,1%)	123 (58%)	**<0,001**
Basal laboratory values				
Urea (mg/dl)		32(13-81)	34(12-94)	**0,049**
Creatinine (mg/dl)		0,87(0,52-2,04)	0,9(0,48-1,96)	0,788
Glucose (mg/dl)		106(74-374)	108(76-406)	0,280
Total cholesterol (mg/dl)		209(125-402)	184,5(125-402)	**<0,001**
LDL (mg/dl)		131(41-317)	105(60-243)	**<0,001**
HDL (mg/dl)		42(19-99)	43(24-82)	0,304
Triglycerides (mg/dl)		164(68-886)	159(41-392)	0,505
AST (u/L)		21(11-106)	20(9-96)	0,204
ALT (u/L)		18(6-61)	18(6-94)	0,411
CK (u/L)		96(32-349)	92(18-430)	0,371
BMI (kg/m^2)		28,7(20,76-49,3)	29,3(18,7-49,9)	0,684

Table 2. Logistic regression analysis showing the predictors of physicians' adherence to guidelines

	Odds ratio (OR)	%95 Confidence interval		P value
		Lover	Upper	
Diabetes mellitus	0,504	0,306	0,830	**0,007**
Hypertension	0,795	0,498	1,271	0,338
NSTEMI	2,469	1,227	4,970	**0,011**
STEMI	11,177	2,856	43,735	**0,001**
Obstructive CAD	2,048	1,252	3,350	**0,004**
Baseline Urea	0,977	0,960	0,995	**0,010**
Baseline LDL	1,028	1,020	1,036	**<0,001**

Table 3. Patients adherence to treatment

		Patients without Drug adherence (n:94)	**Patients with drug adherence (n:346)**	**p value**
Age, n (%)		62 (38-81)	63 (30-86)	0,926
Female gender, n (%)		37 (39,4)	135 (37,9)	0,812
Diabetes mellitus, n (%)		25 (26,6)	117 (32,9)	0,264
Hypertension, n (%)		60 (63,8)	219 (61,5)	0,721
Family history, n (%)		43 (45,7)	192 (53,9)	0,165
Smoking, n (%)		37 (39,4)	157 (44,1)	0,483
previous CABG, n (%)		2 (2,1)	12 (3,4)	0,744
previous stent, n (%)		19 (20,2)	54 (15,2)	0,270
Previous stoke, n (%)		4 (4,3)	12 (3,4)	0,773
BMI		29,2(21,3-49,9)	28,9 (18,7-49,3)	0,945
Clinical presentation	SAP, n (%)	78 (83) *	238 (66,9) *	**0,02**
	USAP, n (%)	10 (10,6)	32 (9)	
	NSTEMI, n (%)	4 (4,3) *	56 (15,7) *	
	STEMI, n (%)	2 (2,1) *	30 (8,4) *	
Obstructive CAD, n (%)		44 (46,8	257(72,2)	**<0,001**
Baseline laboratory values				
Urea (mg/dl)		33 (15-81)	33 (12-82)	0,688
Creatinine (mg/dl)		0,83 (0,48-1,6)	0,9 (0,5-2,0)	0,408
Glucose (mg/dl)		106 (80-356)	109 (74-406)	0,375
Total cholesterol (mg/dl)		191 (134-444)	201(105-402)	0,170
LDL (mg/dl)		110(72-200)	119,5 (41-317)	0,061
HDL (mg/dl)		43(8-77)	42 (19-99)	0,172
Trigliseride (mg/dl)		159(52-908)	166(41-1908)	0,416
AST (u/L)		21 (10-87)	21(9-89)	0,515
ALT (u/L)		17 (8-81)	18(6-94)	0,352
CK (u/L)		96 (45-237)	93(18-430)	0,915
Treatment intensity				
Moderate		54 (57,4)	164(46,3)	0,063
High		40 (42,6)	190 (53,7)	
Physicians' adherence		41 (45,1)	188 (53,7)	0,158
Patients' drug adherence				
Side Effects	None, n (%)	41 (43,6) *	308 (86,5) *	**<0,001**
	Myalgia, n (%)	35 (37,2) *	36 (10,1) *	
	GIS disturbance, n (%)	14 (14,9) *	8 (2,2) *	
	Headache, n (%)	2 (2,1)	2 (0,6)	
	Alopecia, n	1 (1,1)	1 (0,3)	
	Allergic reactions, n	1(1,1)	-	

Table 4. Demographic and clinical characteristics of patients who did and did not achieve target LDL levels

		Patients who achieved Target LDL (n:122)	Patients who DID NOT achieve target LDL	P value
Age, n (%)		63 (36-85)	62 (30-86)	0,766
Female gender, n (%)		45 (36,8%)	123 (39,3%)	0,643
Diabetes mellitus, n (%)		40 (32,7%)	96(30,6%)	0,669
Hypertension, n (%)		70 (57,3%)	202 (64,5%)	0,166
Family history, n (%)		70 (57,3%)	157 (50,1%)	0,176
Smoking, n (%)		52 (42,6%)	135 (43,1%)	0,923
Previous CABG, n (%)		2 (1,6%)	12 (3,8%)	0,244
Previous stent, n (%)		19 (15,5%)	51 (16,2%)	0,854
Previous Stroke, n (%)		4 (3,2%)	12 (3,8%)	0,782
BMI		28,8 (21,3-40,2)	29 (18,7-49,9)	0,660
Clinical presentation	SAP, n (%)	82 (67,2%)	222 (70,9%)	0,103
	USAP, n (%)	7 (5,7%)	34 (10,8%)	
	NSTEMI, n (%)	21 (17,2%)	39 (12,4%)	
	STEMI, n (%)	12 (9,8%)	18 (5,7%)	
Control time, weeks		8 (6-12)	9 (5-12)	0,588
Obstructive CAD, n (%)		94 (77%)	198 (63,2%)	**0,006**
Baseline laboratory data				
Urea (mg/dl)		32 (14-94)	33 (12-91)	0,714
Creatinine (mg/dl)		0,89 (0,52-2,04)	0,90 (0,48-1,98)	0,610
Glucose (mg/dl)		109 (74-346)	106 (76-406)	0,609
Total cholesterol (mg/dl)		193 (124-285)	199 (105-402)	0,300
LDL (mg/dl)		114 (60-193)	120 (41-317)	0,710
HDL (mg/dl)		44 (23-82)	42 (19-99)	0,558
Triglyceride (mg/dl)		147 (51-646)	166 (41-795)	0,136
AST (u/L)		21 (11-106)	21 (9-95)	0,750
ALT (u/L)		17 (7-70)	18 (6-94)	0,628
CK (u/L)		92,5 (18-430)	95 (28-343)	0,861
Control laboratory data				
Total cholesterol (mg/dl)		117 (73-166)	166 (95-307)	**<0,001**
LDL (mg/dl)		47 (12-69)	92 (42-223)	**<0,001**
HDL (mg/dl)		43 (25-89)	43 (21-84)	0,755
Triglyceride (mg/dl)		111 (34-321)	140 (44-437)	**<0,001**
AST (u/L)		20 (11-271)	19 (9-87)	0,096
ALT (u/L)		20 (9-428)	18 (6-81)	0,294
CK (u/L)		85 (37-410)	93,5 (22-545)	0,606
Drug treatment intensity				
Moderate		52 (42,6%)	159 (50,7%)	0,125
High		70 (57,3%)	154 (49,2%)	
Physicians' guideline adherence		69 (56,5%)	155 (49,5%)	0,137
Patient adherence				
Drug nonadherence		0	87 (27,7%)	**<0,001**
Side effects	None, n (%)	108 (88,5%)	232 (74,1%)	0,073
	Myalgia, n (%)	**9 (7,3%)**	**59 (18,8%)**	
	GIS disturbance, n (%)	4 (3,2%)	16 (5,1%)	
	Headache, n (%)	1 (0,8%)	3 (0,9%)	
	Alopecia, n	1	0	
	Allergy, n	0	1	

Lipid / Preventive cardiology

OP-229

ApoE and CYPC19 mutations may play role in early-onset myocardial infarction

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Background and Aim: Myocardial infarction (MI) is a severe manifestation of coronary artery disease (CAD). It is the leading causes of death and disability worldwide. Whereas the majority of MIs occur in individuals bigger than 65 years old, 5-10% of new MIs occur in younger patients. Although many environmental risk factors contribute to its pathogenesis, genetic factors exert a substantial influence on disease risk, particularly for early-onset form of MI. ApoE and CYP2C19 variants may play role in this process. Therefore in this study, it was aimed to investigate the relation between early -onset MI with ApoE and CYP2C19 gene mutations.

Methods: Thirty diagnosed early-onset MI patients (<45 years old in males and <55 years old in females) and thirty healthy controls were enrolled to the study. After DNA was isolated from peripherial blood, e2, e3, e4 variants of ApoE; CYP2C19*2 and *3 variants of CYP2C19 gene were investigated with real-time polymerase chain reaction (RT-PCR). Results were evaluated statistically.

Results: At the end of the study, e2 variant was found statistically high in controls. (27%) whereas e4 variant was found high in patients (23%). Additionally CYP2C19*2 was detected statistically high in patients (50%). Also sigarette smoking, diabetes mellitus, hypertansion, total cholesterol, LDL, triglyceride, fasting blood glucose and body mass index (BMI) levels were found statistically high in patients. When the relation between risk factors and genotypes were investigated, there are some statistical relations found between hypertansion and CYP2C19*2; LDL, HDL, fasting blood glucose with e2; total cholesterol, LDL, HDL and triglyceride levels with e4; triglyceride, fasting blood glucose and BMI levels with CYP2C19*2.

Conclusions: It was thought that, e2 may have a protective role for early-onset MI. Also it was suggested that CYP2C19*2 may have strong association with early-onset of the disease. Additionally according to the increase of some blood parameters, it was thought that, e4 may effect the development of the disease. Therefore genetic screening of individuals for CYP2C19 and ApoE gene mutations may give some information about the development of MI in young adults.

Lipid / Preventive cardiology

OP-230

The relationship between Mediterranean diet and carotid artery disease

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Background and Aim: Mediterranean dietary nutrition is known to have protective roles in preventing cardiovascular events and atherosclerosis, but studies are limited. The aim of this study was to evaluate the relationship between carotid artery stenosis (KAD) and Mediterranean diet score.
Methods: The patients were divided into three groups of patients with KAD >60% (60 patients), KAD <60% (60 patients) and had no carotid atherosclerotic disease (60 patients). Afterwards, patients with KAD were divided into two subgroups, with calcific (67 patients) and non-calcific (53 patients) according to morphological characteristics of carotid atherosclerotic plaque. Diet quality was determined by scoring method (5, 6-9 and ≥10 points) 'Mediterranean Diet Adaptation Scale' and compared between groups.
Results: Mediterranean diet score was higher in patients without atherosclerotic disease than patients with KAD <60% (p<0.001). In addition, Mediterranean diet score was higher in patients with KAD <60 than those with KAD >60 (p<0.001). Mediterranean diet score was higher in patients with calcific atherosclerotic plaque (p<0.001).
Conclusions: The protective role of Mediterranean diet on the severity of carotid artery stenosis was clearly observed in our study. In addition, its potential inhibitory role on non-calcific carotid atherosclerosis, which is closely related to cerebrovascular diseases, has been demonstrated. This study, which is one of the limited studies examining the relationship between Mediterranean diet and KAD, may be useful in understanding the pathophysiology of carotid artery stenosis.

Table 1. Validated 14-item Questionnaire of Mediterranean diet adherence

Questions	
1. Do you use olive oil as main culinary fat?	Yes
2. How much olive oil do you consume in a given day (including oil used for frying, salads, out-of-house meals, etc.)?	≥4
3. How many vegetable servings do you consume per day? (1 serving: 200 g [consider side dishes as half a serving])	≥2 (≥1 portion raw or as a salad)
4. How many fruit units (including natural fruit juices) do you consume per day?	≥3
5. How many servings of red meat, hamburger, or meat products (ham, sausage, etc.) do you consume per day? (1 serving: 100–150 g)	<1
6. How many servings of butter, margarine, or cream do you consume per day? (1 serving: 12 g)	<1
7. How many sweet or carbonated beverages do you drink per day?	<1
8. How much wine do you drink per week?	≥7 glasses
9. How many servings of legumes do you consume per week? (1 serving: 150 g)	≥3
10. How many servings of fish or shellfish do you consume per week? (1 serving 100–150 g of fish or 4–5 units or 200 g of shellfish)	≥3
11. How many times per week do you consume commercial sweets or pastries (not homemade), such as cakes, cookies, biscuits, orcustard?	<3
12. How many servings of nuts (including peanuts) do you consume per week? (1 serving 30 g)	≥3
13. Do you preferentially consume chicken, turkey, or rabbit meat instead of veal, pork, hamburger, or sausage?	Yes
14. How many times per week do you consume vegetables, pasta, rice, or other dishes seasoned with sofrito (sauce made with tomatoand onion, leek, or garlic and simmered with olive oil)?	≥2

Table 2. Characteristics and laboratory parameters of the study groups

Variables	Control (N = 60)	Carotid artery stenosis <60% (N = 60),	Carotid artery stenosis ≥60% (N = 60)	p value*	p value α	p value β	p value γ
Age, years	55.98 ±9.07	57.43 ± 9.75	59.48 ±10.27	0.143			
Female gender, n (%)	27 (45.0%)	25 (41.7%)	27 (45.0)	0.809			
BMI, kg/m2	28.14 ± 2.80	28.26 ± 2.43	28.98 ±3.00	0.200			
Diabetes Mellitus, n (%)	51 (34.2)	47 (31.5)	51 (34.2)	0.536			
Hypertension, n (%)	15 (25.0)	21 (35.0)	24 (40.0)	0.207			
Hyperlipidemia, n (%)	19 (29.2)	24 (36.9)	22 (33.8)	0.633			
Smoking, n (%)	15 (37.1)	14 (22.2)	27 (24.1)	0.017	0.010	0.022	0.831
Coronary Artery Disease, n (%)	6 (10.0)	6 (10.0)	8 (13.3)	0.799			
Peripheral Arterial Disease, n (%)	3 (5.0)	4 (6.7)	8 (13.3)	0.217			
Carotid artery stenosis, (%)	0 ± 0.00	30.17 ± 12.73	69.33± 10.53	<0.001	<0.001	<0.001	<0.001
Calcific Plate, n (%)	0 (0.0)	34 (57.6)	33 (55.0)	<0.001	0.854	<0.001	<0.001
LVEF,%	58.0 ± 4.9	57.7 ± 5.9	58.4 ± 5.0	0.769			
Glucose, mg / dL	115.4 ± 44.1	114.2 ± 44.8	129.3 ± 52.3	0.153			
Creatinine, mg / dL	1.00 ± 0.16	1.05 ± 0.65	0.95 ± 0.21	0.403			
Uric Acid, mg / dl	5.78 ± 2.06	5.87 ± 2.06	5.98 ± 2.16	0.878			
White blood cell, 10^3 / mm^3	8.4 ± 2.1	9.0 ± 2.2	9.8 ± 2.4	0.004	0.068	0.001	0.120
Hemoglobin, g / dL	13.4 ± 1.7	13.7 ± 1.6	14.1 ± 1.6	0.088			
Platelet, 10^3 / mm^3	236.5 ± 62.4	238.1 ± 70.0	270.5 ± 71.6	0.012	0.018	0.009	0.894
CRP, mg / L	3.7 ± 3.7	4.0 ± 3.6	4.9 ± 4.6	0.207			
Total cholesterol, mg / dL	184.1 ± 79.6	190.4 ± 50.2	189.8 ± 46.7	0.812			
LDL-C, mg / dL	113.1 ± 57.3	114.3 ± 42.0	115.9 ± 38.2	0.947			
HDL-C, mg / dL	44.0 ± 24.2	45.3 ± 9.5	49.2 ± 13.1	0.206			
Triglyceride, mg / dL	168.1± 145.0	167.1 ± 106.0	139.1 ± 89.0	0.391			
Mediterranean Diet Score	7.75 ± 2.75	5.50 ± 2.66	4.33 ± 2.50	<0.001	<0.001	<0.001	<0.001

*Data are given as mean ± standard deviation or percentage [n (%)]. BMI, body mass index; CRP, C-reactive protein; HDL, high density lipoprotein; LDL, low density lipoprotein; LVEF, left ventricular ejection fraction. Carotid artery stenosis rate was calculated according to NASCET system. * p value between all groups α p value, between carotid artery stenosis ≥60% and carotid artery stenosis <60% groups, değeri p value is between kontrol60% between control groups and between control groups, değeri p value is between <60% between control groups and between control groups.*

Table 3. Multivariate logistic regression analysis showing the predictors for ≥60% carotid artery stenosis

Variables	Univariable Beta (95% CI)	p value	Multivariable Beta (95% CI)	p value
Diabetes mellitus	0.786 (0.337-1.832)	0.577		
Hypertension	1.556 (0.814- 2.972)	0.181		
Hyperlipidemia	1.037 (0.544-1.974)	0.913		
Smoking	2.567 (1.329-4.959)	0.005	1.483 (0.631-3.789)	0.428
Platelets	1.007 (1.002-1.011)	0.007	1.145 (0.993-1.252)	0.038
White Blood Cell.	0,814 (0.701-0.944)	0.004	0,904 (0701-1,215)	0.024
Mediterranean diet score	1.131 (1.048-1.214)	<0.001	1.248 (1.095-1.499)	<0.001

Table 4. Baseline characteristics and laboratory parameters of the patients according to plaque calcification

Variables	Calcified plaque group (n=67)	Non-calcified plaque group (n=53)	p value
Age,years	59.62 ±9.94	57.57 ±10.06	0.259
Female, n(%)	29 (43.3)	23 (43.4)	0.990
BMI, kg/m2	28.36±2.85	28.97±2.58	0.254
Diabetes mellitus, n(%)	16 (23.9)	6 (11.3)	0.077
Hypertension, n(%)	27 (40.3)	18 (34.0)	0.570
Hyperlipidemia, n(%)	24 (35.8)	22 (41.5)	0.524
Smoking, n(%)	20 (28.9)	21 (39.6)	0.262
Coronary artery disease, n(%)	9 (13.4)	5 (9.4)	0.498
Peripheral vascular disease, n(%)	6 (9.0)	6 (11.3)	0.668
Carotid artery stenosis, (%)	47.42 ± 22.70	53.04 ± 23.16	0.185
LVEF, %	58.0 ± 5.2	58.1± 5.8	0.841
Glucose, mg/dL	123.5 ± 55.1	119.6 ± 40.7	0.664
Creatinine, mg/dL	1.01 ± 0.26	0.99 ± 0.68	0.872
Uric acid, mg/dL	5.91 ± 2.10	5.93 ± 2.13	0.955
WBC,10^3/mm^3	8.5 ± 2.1	8.9 ± 2.7	0.274
Hemoglobin, g/dL	13.7 ± 1.5	14.0 ± 1.7	0.290
Platelets, 10^3/mm^3	255.1 ± 82.6	253.2 ± 65.6	0.890
CRP, mg/L	4.6 ± 3.5	4.3 ± 3.7	0.733
Total cholesterol, mg/dL	192.6 ± 43.5	187.0 ± 54.0	0.531
LDL cholesterol, mg/dL	114.8 ± 39.3	115.6 ± 41.20.913	0.531
HDL cholesterol, mg/dL	47.3 ± 10.7	47.3 ± 12.7	0.975
Triglyceride, mg/dL	147.2± 82.8	160.7± 117.0	0.464
Mediterranean diet score	5.22 ± 2.66	4.53 ± 2.72	<0.001

Data are given as mean ± SD, n (%) or median (lower-upper limit). BMI body mass index; CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; WBC, white blood cell. Carotid artery stenosis rate was calculated according to NASCET.

Lipid / Preventive cardiology

OP-231

Mediterranean diet effects on ventricular premature complexes

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Background and Aim: A style of diet that represents the typical nutritional habits of populations surrounding the Mediterranean Sea includes consumption of high rates of fruits, vegetables, monounsaturated fats, fish, whole-wheat grains, legumes and nuts, as well as low amounts of red meat. Such styles of healthy nutrition have an antiarrhythmic effect potential with their anti-inflammatory, antioxidant and cytoprotective effects. Previous studies have determined the protective effect of the Mediterranean diet from atrial fibrillation. While the antiarrhythmic effects associated with some components of the Mediterranean diet (fruits, walnuts and olive oil) have been determined, the number of studies that have examined the antiarrhythmic effects of the Mediterranean diet as a whole are still limited. Although ventricular premature beats are the most common form of arrhythmias in both patients with and without structural heart disease, ventricular premature beats may cause long-term left ventricular failure in patients with normal heart structure. Increased frequency of ventricular premature complexes (VPC) may be associated with mental and physical stress or lifestyle habits as well as with cardiovascular risk factors such as hypertension, dyslipidemia, diabetes mellitus, and coronary artery disease. Frequent VPC may cause tachycardiomyopathy, and therefore may lead to increased mortality and morbidity. This study aims to evaluate the relationship between frequent Ventricular Extrasystoles (VPC) disease and Mediterranean diet score.
Methods: This study was conducted on 50 patients with palpitations who were referred to the cardiology outpatient clinic and had more than 10000 VPC per day as a result of a 24-hour holter, and 50 patients who presented with palpitations but had less than 10,000 VPC in the holter. Diet quality was determined by scoring method (5, 6-9 and ≥10 points) 'Mediterranean Diet Adaptation Scale' and compared between groups.
Results: There was no difference between the two groups in terms of clinical and demographic characteristics. Mediterranean diet scores were found lower in patients with VPC's ≥10.000/day (p<0.001). There was a significant negative correlation found between number of VPC and Mediterranean diet score (p<0.001, r=-0.652).
Conclusions: The protective role of Mediterranean diet type nutrition on the frequency of VPC was clearly observed in our study. This study, which is one of the limited numbers of studies examining the relationship between Mediterranean diet and VPC, may be helpful in understanding the pathophysiology of VPC.

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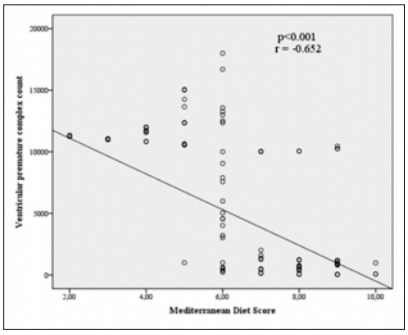

Figure 1. The correlation between Mediterranean diet score and ventricular premature complexes.

Table 1. Validated 14-item Questionnaire of Mediterranean diet adherence.

Questions	Criteria for 1 point
1. Do you use olive oil as main culinary fat?	Yes
2. How much olive oil do you consume in a given day (including oil used for frying, salads, out-of-house meals, etc.)?	≥4
3. How many vegetable servings do you consume per day? (1 serving: 200 g [consider side dishes as half a serving])	≥2 (≥1 portion raw or as a salad)
4. How many fruit units (including natural fruit juices) do you consume per day?	≥3
5. How many servings of red meat, hamburger, or meat products (ham, sausage, etc.) do you consume per day? (1 serving: 100–150 g)	<1
6. How many servings of butter, margarine, or cream do you consume per day? (1 serving: 12 g)	<1
7. How many sweet or carbonated beverages do you drink per day?	<1
8. How much wine do you drink per week?	≥7 glasses
9. How many servings of legumes do you consume per week? (1 serving: 150 g)	≥3
10. How many servings of fish or shellfish do you consume per week? (1 serving 100–150 g of fish or 4–5 units or 200 g of shellfish)	≥3
11. How many times per week do you consume commercial sweets or pastries (not homemade), such as cakes, cookies, biscuits, orcustard?	<3
12. How many servings of nuts (including peanuts) do you consume per week? (1 serving 30 g)	≥3
13. Do you preferentially consume chicken, turkey, or rabbit meat instead of veal, pork, hamburger, or sausage?	Yes
14. How many times per week do you consume vegetables, pasta, rice, or other dishes seasoned with sofrito (sauce made with tomatoand onion, leek, or garlic and simmered with olive oil)?	≥2

Table 2. Baseline general and clinical characteristics of the study population

Variables	VPC group (n=50)	Control group (n=50)	p value
Age, years	57.0 ± 10.6	55.1 ± 9.4	0.354
BMI, kg/m2	29.4 ± 3.1	27.7 ± 4.2	0.122
Waist circumference, cm	86.9 ± 7.3	85.1 ± 9.9	0.259
Waist to height ratio	0.54 ± 0.06	0.53 ± 0.09	0.722
Smoking, n (%)	22 (44.0)	15 (30.0)	0.147
Hypertension, n (%)	6 (12.0)	4 (8.0)	0.505
Hyperlipidemia, n (%)	12 (24.0)	10 (20.0)	0.629
Diabetes Mellitus, n (%)	6 (12.0)	5 (10.0)	0.749
Female, n (%)	30 (60.0)	33 (66.0)	0.534
Married, n (%)	16 (32.0)	20 (40.0)	0.403
Ejection Fraction, (%)	62.3 ± 4.1	61.5 ± 6.2	0.433
Education level, n (%)			0.807
Literate	10 (20.0)	12 (24.0)	
Middle School	22 (44.0)	38 (20.0)	
High School and above	18 (36.0)	19 (38.0)	
Physical activity			0.812
Sedentary (<600 METs-min/week)	30 (60.0)	32 (64.0)	
Inactive (600-3000 METs- min/week)	19 (38.0)	17 (34.0)	
Active (>3000 METs- min/week)	1 (2.0)	1 (2.0)	
Mediterranean diet score	5.5 ± 1.9	7.2 ± 1.7	<0.001

Data are given as mean ± standard deviation or number (%) [n (%)], BMI: Body mass index, METs: Metabolic Equivalent Minutes.

Lipid / Preventive cardiology

OP-232

Serum vitamin D deficiency is independently associated with mitral annular calcification in acute coronary syndrome patients

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Background and Aim: Vitamin D deficiency plays a role in many physiopathological processes, including coronary atherosclerosis. Recent studies reported that mitral annular calcification (MAC) might be a prognostic indicator of the acute coronary syndrome (ACS). The purpose of the study is to evaluate the relationship of vitamin D deficiency with MAC in patients with ACS.
Methods: The study consisted of 276 patients (54 women) diagnosed with ACS and underwent coronary angiography (CAG). Measurement of 25(OH)D and transthoracic echocardiography (TTE) were performed within 24 hours after hospitalization. Mitral annular calcification was defined as increased echo-density located at the junction of the atrioventricular groove and posterior mitral leaflet. We analyzed the relation between serum levels of 25(OH)D and echocardiographic parameters.
Results: The mean age of the patients was 62.5±11.4 years. In univariate logistic regression analyses female gender (p<0.001), MAC (p=0.006), left atrial diameter (p=0.026), and low-density lipoprotein (p=0.033) were associated with vitamin D deficiency. In multivariate logistic regression analysis MAC (OR: 2.533, 95% CI: 1.068-6.009, p=0.035) and female gender (OR: 0.137, 95% CI: 0.061-0.306, p<0.001) were independently associated with vitamin D deficiency.
Conclusions: In patients presented with ACS, vitamin D deficiency had an independent relationship with MAC. It may be more cost-effective to evaluate vitamin D deficiency in women with ACS, accompanied by MAC.

Lipid / Preventive cardiology

OP-233

Evaluation of sleep quality and nutritional status of patients with frequent ventricular premature complexes

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Background and Aim: Cardiac arrhythmias have serious effects on morbidity and mortality. Although ventricular premature beats are the most common form of arrhythmias in both patients with and without structural heart disease, ventricular premature beats may cause long-term left ventricular failure in patients with normal heart structure. Increased frequency of ventricular premature complexes (VPC) may be associated with mental and physical stress or lifestyle habits, as well as with cardiovascular risk factors such as hypertension, dyslipidemia, diabetes mellitus, and coronary artery disease. Sleep quality affects a wide range of health-related behaviors. Poor sleep quality may have indirect effects on nutrition/weight control, smoking, alcohol use, physical activity, and stress management, and may have direct effects on certain biological mechanisms such as insulin regulation and metabolic hormone deterioration, as well as changes in the cardiac and neuroendocrine system. Lifestyle change, pharmacotherapy or catheter ablation may play an essential role in the clinical evaluation of patients presenting with frequent VPC and management of VPC frequency in patients. This study aims to evaluate the relationship between frequent Ventricular Extrasystoles (VPC) and sleep quality and nutritional status.
Methods: This study was conducted on 50 patients with palpitations who were referred to the cardiology outpatient clinic and had more than 10,000 VPC per day as a result of a 24-hour holter, and 50 patients who presented with palpitations but had less than 10,000 VPC in the holter. Demographic characteristics, nutritional status, sleep quality and some anthropometric measurements of the patients were evaluated using a questionnaire. Food consumption record was taken to determine daily energy and macro nutrient intake of individuals. Pittsburgh Sleep Quality Index (PSQI) was used to evaluate sleep quality.
Results: PSQI scores indicating decreased sleep quality were found to be high in the VPC group (p<0.001). Energy consumption (p=0.004) and carbohydrate consumption (p<0.001) were significantly higher in the VPC group. Saturated fatty acid consumption was high (p<0.001) and polyunsaturated fatty acid consumption was low in the VPC group (p<0.001). There was significant positive correlation found between VPC count and PSQI scores (p<0.001, r=0.788).
Conclusions: It was clearly observed that the frequency of VPC decreases sleep quality and leads to imbalances in individuals' nutritional status. Therefore, in addition to the medical treatment of rhythm disorder, individuals should be given training to improve sleep quality and nutritional status by a multidisciplinary team (such as doctors, dietitians, psychologists).

Table 1. Baseline general and clinical characteristics of the study population

Variables	VPC group (n=50)	Control group (n=50)	p value
Age, years	57.0 ± 10.6	55.1 ± 9.4	0.354
BMI, kg/m2	29.4 ± 3.1	27.7 ± 4.2	0.122
Waist circumference, cm	86.9 ± 7.3	85.1 ± 9.9	0.259
Smoking, n (%)	22 (44.0)	15 (30.0)	0.147
Hypertension, n (%)	6 (12.0)	4 (8.0)	0.505
Hyperlipidemia, n (%)	12 (24.0)	10 (20.0)	0.629
Diabetes Mellitus, n (%)	6 (12.0)	5 (10.0)	0.749
Female, n (%)	30 (60.0)	33 (66.0)	0.534
Married, n (%)	16 (32.0)	20 (40.0)	0.403
Ejection Fraction, (%)	62.3 ± 4.1	61.5 ± 6.2	0.433
Education level, n (%)			0.807
Literate	10 (20.0)		
Middle School	22 (44.0)		
High School and above	18 (36.0)		
Physical activity			0.812
Sedentary (<600 METs-min/week)	30 (60.0)	12 (24.0)	
Inactive (600-3000 METs- min/week	19 (38.0)	38 (20.0)	
Active (>3000 METs- min/week)	1 (2.0)	19 (38.0)	
PSQI score			
0-5	17 (34.0)	33 (66.0)	<0.001
6-9	28 (56.0)	16 (32.0)	<0.001
>9	5 (10.0)	1 (2.0)	<0.001

Data are given as mean ± standard deviation or number (%) [n (%)], BMI: Body mass index, METs: Metabolic Equivalent Minutes, PSQI: Pittsburgh Sleep Quality Index.

Table 2. Daily energy and nutrient intake status of the participants

Variables	VPC group (n=50)	Control group (n=50)	0.004
Energy (kcal)	2513.7 ± 712.4	2067 ± 450.4	<0.001
Carbonhydrate (g)	270.4 ± 115.8	198.7 ± 48.2	0.083
Carbonhydrate (E%)	43.1 ± 9.1	39.6 ± 7.3	0.320
Protein(g)	79.9 ± 24.7	65.9± 21.7	0.408
Protein(g/kg)	1.1 ± 0.3	0.9 ± 0.3	0.480
Protein(E%)	13.2 ± 2.8	13.1 ± 2.9	0.776
Fat(g)	120.0 ± 34.6	109.7 ± 32.9	0.083
Fat(E%)	43.4 ± 8.6	47.1 ± 6.9	0.083
Saturated fatty asid	50.4 ± 15.2	40.3 ± 16.2	<0.001
Mono unsaturated fatty asid	46.8 ± 14.5	43.8 ± 15.5	0.125
Poly unsaturated fatty asid	22.7 ± 13.8	27.7 ± 12.8	<0.001
Fiber (g)	28.1 ± 13.4	21.8± 6.6	0.009

Data are given as mean ± standard deviation, E: total energy percent.

Lipid / Preventive cardiology

OP-234

Monocyte count to high-density lipoprotein cholesterol ratio may be a predictor in ascending aortic aneurysm

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Background and Aim: Out of one hundred thousand people randomly selected, 5 or 10 may have ascending aortic aneurysms (AAA). Etiologically, AAAs have remained idiopathic, and it is risky to diagnose this disease based on atherosclerosis, rather than descending and abdominal aortic aneurysms. Ascending aorta dilation may potentially occur when the extracellular matrix is deformed and destructed, and in long periods, the aortic wall is exposed to hemodynamic force, for instance, led by high blood pressure. In the pathophysiological aspect, although which mechanisms may cause AAAs has been now better understood by means of recent studies, there are limited biomarkers for risk assessment of aneurysms. Typically, in controlling the cholesterol attack from tissues and modulating inflammation and oxidative stress, normal HDL is an effective anti-inflammatory molecule and antioxidant. For human monocytes, its major protein component, apolipoprotein A-I (apo A-I), inhibits CD11b from activating. These monocytes help a variety of cytokines and molecules be produced and on the other hand, their interaction with platelets or endothelial cells during circulation allows proteases to destroy the extracellular matrix, and hence smooth muscle cells may differentiate (apoptosis), oxidative stress may progress, or neovascularization or calcification may occur. In cardiovascular science, there is an innovative precursor for chronic kidney patients in these days: the ratio of monocyte count to high-density lipoprotein cholesterol or MHR. It has been reported that MHR may slow coronary flow and cause stent thrombosis, stent thrombosis, coronary artery ectasia acute coronary syndrome and loss of aortic elasticity among hypertension patients. According to the literature review, the pathogenesis of degenerative AAA could be caused by monocytes or HDL-cholesterol and accordingly, its diagnosis and management should advance With this, the aim this of study is to determine whether the maximum diameter of ascending aorta observed in admission of each asymptomatic patients are associated with the MHR calculated.

Methods: 240 consecutive patients with AAA and 240 consecutive patients with normal ascending aortic diameter were recruited into the study by comprehensive transthoracic echocardiography. All data and MHR was compered between two groups.

Results: MHR levels were significantly higher in AAA group compared to normal ascending aortic diameter group (p<0.001). Higher levels of MHR was found significantly and independently associated with the AAA (p<0.001). Also there was significant positive correlation between the diameter of the ascending aorta and the MHR (p<0.017).

Conclusions: MHR as a marker of chronic low-grade inflammation may play a role in the pathogenesis of aneurysm of the ascending aorta.

Table 1. Clinical and demographic characteristics of the study population

Variables	Control group (n=240)	Case group (n=240)	p value
Age,years	58.81 ±14.82	57.45 ±15.04	0.312
Female, n(%)	76 (31.7%)	61 (25.4%)	0.130
Body mass index, kg/m2	28.95 ± 2.31	28.12 ± 2.78	0.534
Diabetes Mellitus, n(%)	42 (17.5%)	49 (20.4%)	0.415
Hypertension, n(%)	92 (38.3%)	119 (49.6%)	0.013
Hyperlipidemia, n(%)	53 (22.1%)	63 (26.3%)	0.286
Smoking, n(%)	91 (37.9%)	97 (40.4%)	0.575
Coronary artery disease, n(%)	32 (13.3%)	31 (12.9%)	0.892
Peripheral vascular disease, n(%)	24 (10.0%)	32 (13.3%)	0.255
Beta Blocker usage, n(%)	21 (8.8%)	32 (13.3%)	0.086

Table 2. Echocardiographic characteristics of the study population

Variables	Control group (n=240)	Case group (n=240)	p value
LVEF (%)	63.1 ± 2.1	60.8 ± 2.2	0.412
ARVC (mm)	2.4 ± 1.0	2.4 ± 1.0	<0.001
Aortic annulus diameter (mm)	2.15 ± 0.21	2.36 ± 0.30	0.036
Sinus valsalva diameter (mm)	3.42 ± 0.71	4.04 ± 0.77	<0.001
Ascending aorta diameter (mm)	4.51 ± 1.41	3.27 ± 0.24	<0.001
Arcus aorta diameter (mm)	2.49 ± 0.21	3.29 ± 0.71	<0.001
Bicuspid aortic valve, n(%)	7 (2.9%)	45 (18.8%)	<0.001

ARVC, vena contracta width of aortic regurgitation; LVEF, left ventricular ejection fraction.

Table 3. Blood parameters of the study population

Variables	Control group (n=240)	Case group (n=240)	p value
Glucose, mg/dL	112.6 ± 42.3	110.6 ± 35.9	0.507
Creatinine, mg/dL	1.06 ± 0.28	1.08 ± 0.31	0.676
Uric Acid, mg/dl	5.57 ± 2.42	6.58 ± 2.52	0.038
Hemoglobin, g/dL	13.7 ± 1.5	14.0 ± 1.7	0.290
WBC,10^3/mm^3	7.7 ± 2.3	8.0 ± 2.4	0.303
Neutrophil,10^3/mm^3	5.0 ± 2.0	5.2 ± 3.2	0.485
Lymphocyte,10^3/mm^3	1.9 ± 0.8	2.0 ± 1.0	0.195
Monocyte,10^3/mm^3	0.6 ± 0.2	0.7 ± 0.3	<0.001
Platelet, 10^3/mm^3	255.8 ± 78.6	259.2 ± 83.7	0.731
Hs-CRP, mg/L	4.6 ± 2.5	8.3 ± 3.7	<0.001
Total cholesterol, mg/dL	189.3 ± 43.0	184.3 ± 45.1	0.155
LDL-C, mg/dL	117.8 ± 33.6	112.3 ± 45.1	0.272
HDL-C, mg/dL	47.3 ± 10.7	47.3 ± 12.7	0.975
Triglyceride, mg/dL	143.1 ± 84.8	148.8 ± 73.0	0.460
MHR	0.013 ± 0.006	0.016 ± 0.006	<0.001

WBC, white blood cell; Hs-CRP, high-sensitivity C-reactive protein; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MHR, Monocyte – high-density lipoprotein ratio.

Table 4. Multivariate linear regression analysis showing the predictors for the Ascending aortic dilatation

Variables	Univariable Beta (95% CI)	p value	Multivariable Beta (95% CI)	p value
Hypertension	1.582 (1.101-2.274)	0.013	1.477 (0.998-2.187)	0.057
Uric Acid	1.041 (0.997-1.087)	0.070		
Monocyte count	7.092 (3.196-15.738)	0.030	4.483 (0.906-8.159)	0.066
Hs-CRP	1.040 (1.024-1.060)	<0.001	1.032 (1.013-1.052)	0.001
MHR	1.025 (1.011-1.039)	<0.001	1.013 (1.005-1.023)	0.001

Hs-CRP, high-sensitivity C-reactive protein; MHR, Monocyte – high-density lipoprotein ratio.

Lipid / Preventive cardiology

OP-236

The relationship between monocyte high-density lipoprotein ratio with adverse cardiac events among myocarditis patients

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Background and Aim: Myocarditis is an inflammatory disease of the myocardium. The present study aimed to evaluate MHR on the clinical endpoints in patients with myocarditis.
Methods: The study population included 156 patients clinically diagnosed with myocarditis. Patients were divided into two groups according to the primary endpoint.
Results: There were significant differences between the groups: MHR were significantly higher compared with those without a primary endpoint. In ROC analysis, an MHR cut-off of 14.6 had 84% sensitivity and 76% specificity for prediction of MACE. Multivariate Cox regression analysis, MHR was the only independent predictor of the primary endpoint.
Conclusions: This study showed that higher MHR level is an independent predictor of malignant arrhythmic event, new-onset heart failure and cardiovascular death in patients with myocarditis.

Pulmonary hypertension / Pulmonary vascular diseases

OP-238

Risk assessment in congenital heart disease associated pulmonary arterial hypertension

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Background and Aim: Prognosis of congenital heart disease associated pulmonary arterial hypertension (CHD-PAH) is much better than other subsets. However, patients with CHD-PAH in intermediate or high risk may need intensified therapies. We aimed to test an abbreviated version of the risk assessment strategy proposed by the PH guidelines and evaluate the effect of treatment strategy on risk status and survival in CHD-PAH.
Methods: We enrolled a mixed prevalent and incident cohort of patients with CHD-PAH from 6 PAH centers from January 2006 to June 2019 (n=121). Patients were treated with monotherapy or sequential combination therapy at the discretion of treating physician. An abbreviated version of the European risk stratification model comprising WHO functional class (FC), 6 minute walk distance (6 MWD), and N-terminal pro–brain natriuretic peptide (NT-proBNP) or BNP was used. Patients were divided into groups by the presence of low risk criteria. Risk was assessed at baseline and at follow-up. Kaplan-Meier (KM) survival was assessed in each risk group with all cause mortality as the end point. Log-rank test was used to compare estimates.
Results: The mean age was 33±16 years at diagnosis (70% women). The mean follow up was 93.37±55.52 months. 31 patients had died. 38% of patients were WHO FC I-II, 50% III, and 12% IV. This model effectively discriminated risk in our cohort. Patients achieving two or more low-risk criteria at follow-up had a significantly reduced risk of death compared with patients achieving no low risk criteria or only one low risk criterion. Figure 1 demonstrates KM survival in our cohort according to the presence of low risk criteria. Overall, 28.1%, 25.6%, 25.6%, and 20.7% of the patients had 0, 1, 2, and 3 low risk criteria, respectively, at follow-up. The estimated survival rates at 1, 3 and 5 years of patients meeting all three low-risk criteria at follow-up were 100%, 100% and 100%, respectively. The corresponding survival rates were 100%, 100% and 100% for patients meeting two low-risk criteria; 97%, 94% and 87% for patients meeting one low-risk criterion; and 94%, 74% and 65% for patients who had no low-risk criteria at follow-up (p=0.0001 by log-rank test; Figure 1). Distribution of risk groups between initial treatment strategies was similar. Initial treatment strategy had no effect on survival. 46.3% of patients met 2 or more low-risk criteria at follow-up. On multiple logistic regression analysis, the number of low risk criteria at follow-up being ≥2 had the most favorable impact on mortality (OR: 0.013, p=0.0001).
Conclusions: A non-invasive model comprising WHO FC, 6 MWD, and NT pro-BNP is more beneficial to predict risk on treatment than at the time of diagnosis. Patients who achieved two or more low risk criteria at follow-up have an excellent long term prognosis. This analysis also supports the value of goal-oriented treatment in CHD-PAH. Patients who have less than 2 low-risk criteria at follow-up may benefit from escalation of their treatment regimen.

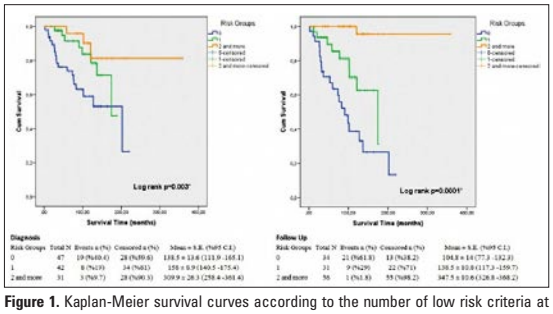

Figure 1. Kaplan-Meier survival curves according to the number of low risk criteria at diagnosis and follow-up.

Pulmonary hypertension / Pulmonary vascular diseases

OP-239

Assessment of the relationship between retinal microvascular perfusion and right-sided echocardiographic and hemodynamic measurements in patients with idiopathic pulmonary arterial hypertension

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Background and Aim: Idiopathic pulmonary arterial hypertension (IPAH) is a rare and progressive disease characterized by progressive neointimal proliferation and remodeling of the pulmonary arteries. These morphological changes in the pulmonary vasculature lead to elevation of the right-sided filling pressures and progressive dysfunction over time in the liver, gastrointestinal tract, kidney as well as the eye. The effects of right-sided pressure overload on retinal microcirculation have never been investigated in patients with IPAH. The aim of this study was to evaluate the retinal microvascular blood flow, determine the possible microvascular alterations of the retina associated with elevated right-sided intracardiac pressures in patients with idiopathic pulmonary arterial hypertension.
Methods: Twenty patients with a confirmed diagnosis of IPAH and 20 age- and sex-matched healthy control subjects were included in the study. Mean right atrial, mean pulmonary artery pressure (mPAP), systolic and diastolic pulmonary artery pressures, pulmonary capillary wedge pressure, pulmonary vascular resistance (PVR), and cardiac output were extracted from the most recent right heart catheterisation (RHC) data. Echocardiographic examination was performed within 24 hours of ophthalmological examination and right atrium area, peak tricuspid regurgitant velocity (TRV), pulmonary artery systolic pressure (sPAP) were obtained. For the right eyes of all participants, high-resolution scans of chorioretinal microvascular networks at different depths of the retina were captured via optical coherence tomography angiography (OCTA) (Optovue Inc., Fremont, California, USA) to assess the foveal avascular zone (FAZ) area; perfusion of the superŞcial and deep capillary plexus (SCP and DCP); and choriocapillaris (CCP) flow area.
Results: The perfusion of SCP and DCP; and CCP flow area were significantly lower in IPAH patients compared to healthy control subjects (p<0.05 for all). In IPAH group, PVR and mPAP were correlated significantly with the perfusion measurements at SCP and DCP (r=0.461, r=0.626 and r=0.625, r=0.730, respectively, p<0.05). PVR and mPAP were also correlated with flow area of CCP (r=0.456 and r=0.589, respectively, p<0.05). Moreover, sPAP and TRV were positively correlated with the perfusion measurements at SCP and DCP (r=0.600, r=0.662 and r=0.670, r=0.655, respectively, p<0.05).
Conclusions: The results of our study demonstrated that retinal microvascular perfusion is reduced in IPAH patients. The positive correlation of retinal perfusion at SCP and DCP with the right-sided echocardiographic and hemodynamic measurements may reveal that assessment of retinal microvascular perfusion could serve as a potential surrogate for monitoring pressure alterations in the pulmonary circulation of the patients with IPAH.

Table 1. Baseline characteristics of the patients with idiopathic PAH

		mean ± SD or n (%)
Demographics	Age (years)	49.40 ± 11.16
	Female n (%)	15 (75)
	Male n (%)	5 (25)
Diagnosis	idiopathic PAH n (%)	20 (100)
WHO FC	II n (%)	12 (60)
	III n (%)	7 (35)
	IV n (%)	1 (5)
RHC (rest)	mRAP (mmHg)	10.75 ± 4.93
	mPAP (mmHg)	49.75 ± 18.99
	sPAP (mmHg)	76.65 ± 26.23
	dPAP (mmHg)	32.65 ± 16.55
	PCWP (mmHg)	12.30 ± 1.59
	PVR (WU)	10.74 ± 9.21
	CO (l/min)	4.30 ± 1.28
Echocardiography	RA area	
	<18cm2 n (%)	2 (10)
	18-26cm2 n (%)	11 (55)
	>26cm2 n (%)	7 (35)
	sPAP (mmHg)	61.75 ± 14.53
	TRV (m/s)	3.52 ± 0.46
Laboratory analysis	BNP (pg/ml)	74.26 ± 81.27

BNP = Brain natriuretic peptide; CO = cardiac output; dPAP = diastolic pulmonary arterial pressure; mPAP = mean pulmonary arterial pressure; mRAP = mean right atrial pressure; PAH = Pulmonary arterial hypertension; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance; RA = right atrium; RHC = right heart catheterisation; SD = standard deviation; sPAP = systolic pulmonary arterial pressure; TRV= peak tricuspid regurgitant velocity; WHO FC = World Health Organization functional class.

Table 2. OCTA findings of IPAH patients and control group

	IPAH	Control	p-value
Age (years)	49.40 ± 11.17	49.85 ±10.94	0.898
Gender(Male/Female)	5/15	5/15	0.997
Spherical Equivalent (diopter)	-0.049 ±0.288	-0.049 ± 0.272	0.529
IOP (mmHg)	14.70 ± 2.30	14.52 ± 2.51	0.695
AL (mm)	22.6 (21.8–25.1)	22.8 (21.8–25.2)	0.597
FAZ area (mm2)	0.325 ± 0.176	0.286 ± 0.141	0.444
FAZ perimeter (mm)	2.216 ± 0.598	2.022 ± 0.518	0.280
Perfusion at SCP (%)	47.805 ± 4.471	51.390 ± 3.561	0.008*
Perfusion at DCP (%)	48.324 ± 6.210	51.890 ± 4.625	0.046*
CCP flow area (mm2)	2.096 ± 0.110	2.175 ±0.104	0.025*

*All data are presented as the mean±SD. Abbreviation: AL = axial length; CCP = choriocapillaris; D = diopter; DCP = deep capillary plexus; FAZ = foveal avascular zone; IOP = intraocular pressure; IPAH = idiopathic pulmonary arterial hypertension; OCTA = optical coherence tomography angiography; SD = standard deviation; SCP = superŞcial capillary plexus. *The value was statistically signiŞcant (p<0.05).*

Table 3. Correlation between right-sided echocardiographic and hemodynamic measurements with retinal microvascular perfusion parameters in IPAH patients

	SCP	DCP	CCP
mPAP	r =0.625, p =0.003	r =0.730, p <0.001	r =0.589, p =0.006
PVR	r =0.461, p =0.041	r =0.626, p =0.003	r =0.456, p =0.043
sPAP	r =0.600, p =0.005	r =0.662, p =0.001	r =0.493, p =0.027
TRV	r =0.670, p =0.001	r =0.655, p =0.002	r =0.517, p =0.020

CCP = choriocapillaris; DCP = deep capillary plexus; IPAH = idiopathic pulmonary arterial hypertension; mPAP = mean pulmonary arterial pressure as assessed by right heart catheterization (RHC); PVR = pulmonary vascular resistance as assessed by RHC; SCP = superficial capillary plexus; sPAP = systolic pulmonary arterial pressure as assessed by transthoracic echocardiography (TTE); TRV = peak tricuspid regurgitant velocity as assessed by TTE.

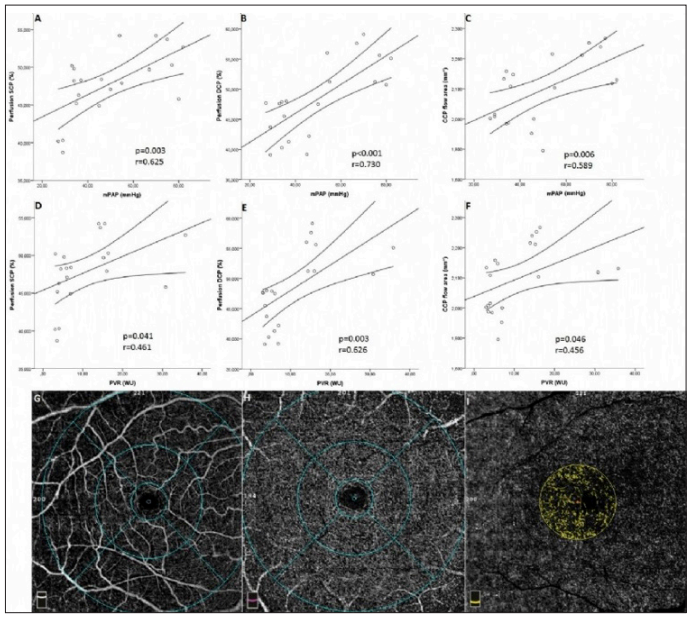

Figure 1. A-F Scatterplots illustrating the relationship between retinal microvascular perfusion parameters and right-sided hemodynamic measurements. OCTA scans demonstrating microvascular perfusion of the SCP (G), DCP (H); and CCP flow area (I).

Pulmonary hypertension / Pulmonary vascular diseases

OP-240

Balloon pulmonary angioplasty in patients with inoperable or recurrent/ residual chronic thromboembolic pulmonary hypertension: A single-center initial experience

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Background and Aim: Patients with inoperable chronic thromboembolic pulmonary hypertension (CTEPH) and residual or recurrent pulmonary hypertension (PH) after pulmonary endarterectomy(PEA) are often treated with PAH-specific therapy. However, despite medical therapy, most of these patients remain symptomatic. Balloon pulmonary angioplasty (BPA) is an emerging therapeutic intervention in patients with inoperable CTEPH and residual or recurrent PH after PEA. This study aims to report the initial experience of BPA in a tertiary referral center for CTEPH.

Methods: A total of 26 consecutive patients, who underwent 91 BPA interventions, were included in the study. All patients underwent a detailed workup, including functional class, 6 minutes walking test, right heart catheterization at baseline, and 4-6 weeks after the last BPA session.

Results: The mean age of patients was 51±17 years, and 18 (69.2%) were female. Fifteen of all patients deemed inoperable CTEPH and 11 of those had residual or recurrent CTEPH post PEA (Table 1). WHO functional class improved in 17 of 26 (65%) patients but remained unchanged in 9 (35%) patients (Figure 1). While 6-MWD increased from mean 315±129 to 411±140 m, serum NT pro-BNP levels reduced from median 456 (IQR:2517) to 189 (IQR:374) pg/ml. The number of patients required supplemental oxygen therapy at home decreased from 11 (42.3%) to 5 (19%) after BPA treatment. The mean PAP decreased from mean 47.5±13.4 to 38±10.9 mmHg, and the PVR decreased from mean 9.3±4.7 to 5.8±2.8 Wood units. While CI significantly increased from mean 2.4± 0.7 to 2.9±0.6 L/min/m², change in the RA pressure did not reach statistical significance (decreased from 9.8±4.2 to 9.2±4.6, p=0.46). Neither death nor invasive mechanical ventilation was occurred due to procedure-related causes (Table 2) (Figure 2). A total of 20 intervention-related complications occurred in 10 patients during 91 sessions (22% of all interventions, 38% of all study population) (Table 3).

Conclusions: BPA improves hemodynamics, 6-MWD, functional class, and reduce requiring supplemental oxygen therapy with an acceptable risk-benefit ratio in patients with inoperable CTEPH and residual or recurrent PH after PEA.

Table 1. Baseline characteristics of study population

Age, years	51 ±17
Gender, female	18 (69.2%)
Body mass index, m/kg²	27.7±6.8
History of VTE, n (%)	7 (26.9%)
Inoperable disease, n (%)	15 (57.7%)
Distal pre-dominancy of disease, n (%)	10 (38.5%)
Severe medical comorbidities, n (%)	5 (19.2%)
Previous PEA (Residual/recurrent), n (%)	11 (42.3%)
Underlying disease or hypercoagulable state, n (%)	11 (43.3%)
Splenectomy, n (%)	1 (3.8%)
Lupus, n (%)	2 (7.7%)
Isolated pulmonary vasculitis, n (%)	2 (7.7%)
Factor V Leiden homozygote, n (%)	2 (3.2%)
Behcet disease, n (%)	1 (3.2%)
History of Cancer, n (%)	3 (11.5%)
WHO functional class, n (%)	
I	0
II	7 (26.9%)
III	16 (61.5%)
IV	3 (11.5%)
Medications	
PAH-Targeted therapy	21 (80.8%)
Riociguat, n (%)	15 (57.7%)
Endotelin Receptor antagonists	6 (23.1%)
Phosphodiesterase 5 inhibitors	4 (15.4%)
Prostacyclin analogue	5 (19.2%)
Medications (none/single/double/triple), n	5/16/1/4
Anticoagulant drugs	
Warfarin, n(%)	10 (38.5%)
Direct oral anticoagulant, n(%)	16 (61.5%)

VTE: Venous thromboembolism **PEA:** Pulmonary endarterectomy **WHO:** World health organization. Data presented as mean± standard deviation and n (%).

Table 2. Changes in the hemodynamics and clinical data before and after BPA Treatment

Variable	Baseline (n: 26)	Final (n: 26)	P value
Hemodynamics			
Systolic PAP, mmHg	77.6±19.2	65±17.8	**<0.001**
Mean PAP, mmHg	47.5±13.4	38±10.9	**<0.001**
Diastolic PAP, mmHg	25.8±8.3	22.6±7.2	**0.013**
PCWP, mmHg	11.1±2.1	10.9±3.3	0.860
Right atrial pressure, mmHg	9.8±4.2	9.2±4.6	0.465
PVR, Woods Unit	9.3±4.7	5.8±2.8	**<0.001**
Pulmonary artery O_2 saturation (%)	60.1±12.9	65.6±7.7	**0.029**
CO, L/min	4.4±1.6	5.1±1	**0.039**
CI, L/min/m²	2.4±0.7	2.9±0.6	**0.008**
Clinical and laboratory			
SaO_2, %			
WHO ≥ 3, n(%)	19 (73.7%)	7 (26.3%)	**<0.001**
6 MWD, meters	315±129	411±140	**<0.001**
NT pro-BNP, pg/ml	456 (2517)	189 (374)	**0.001**
Supplemental oxygen, n(%)	11 (42.3%)	5 (19.2%)	**0.031**

BPA: Balloon pulmonary angioplasty **PAP:** Pulmonary artery pressure **PCWP:** Pulmonary capillary wedge pressure **PVR:** Pulmonary vascular resistance **CO:** Cardiac output **CI:** Cardiac index **WHO:** World health organization **6MWD:** Six minutes walking distance. Data presented as mean ± standard deviation and median (interquartile range).

Table 3. Periprocedural characteristics and complications related to BPA

Total Sessions	91 (100%)
Intervention per patients	3 (2-7)
Targeted vessel per intervention	4 (2-10)
Contrast agent, ml	254± 72
Fluoroscopy time, min	48.7±15.9
Hemoptysis due to vascular injury	9 (9.8%)
Mild	8 (8.8%)
Severe	1 (1.1%)
Reperfusion lung injury	3 (3.3%)
Mild	2 (2.1%)
Moderate	1 (1.1%)
Dissection	5 (5%)
Access site complication*	2 (2.2%)
Non-invasive mechanic ventilation	1 (1.1%)
Renal dysfunction	1 (1.1%)
Overall complications	20 (22%)
Mild	16 (17.6%)
Moderate to severe	4 (4.4%)
*One patient required blood transfusion due to retroperitoneal hematoma. Data presented as n (%) and median (minimum-maximum)	

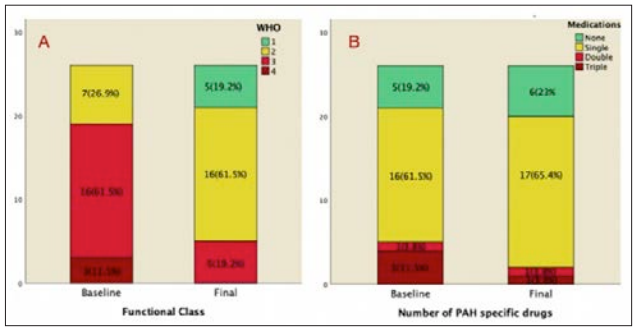

Figure 1. Improvement in WHO functional classes of the patients; baseline and after BPA (A). Reduction in targeted drugs; baseline and after BPA (B). WHO: World health organization, BPA: Balloon pulmonary angioplasty.

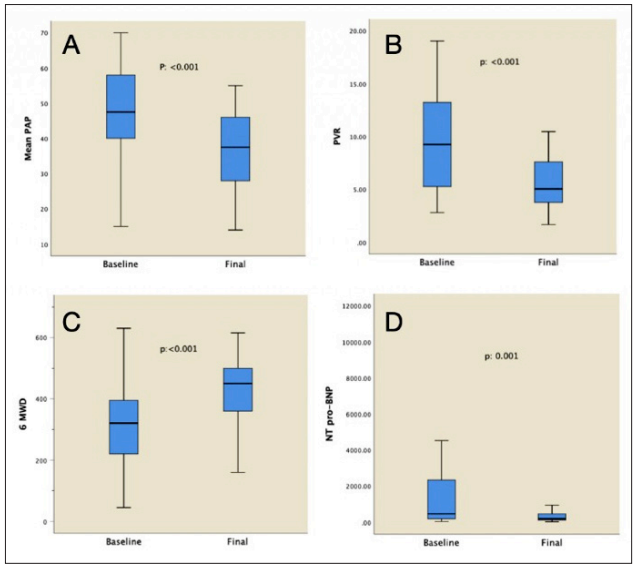

Figure 2. A box plot shows changes in mPAP (A), PVR (B), 6 MWD (C), and NT pro-BNP (D) with BPA treatment. mPAP: Mean pulmonary arterial pressure, PVR: Pulmonary vascular resistance, 6 MWD: 6 minutes walking distance, BPA: Balloon pulmonary angioplasty.

Pulmonary hypertension / Pulmonary vascular diseases

OP-241

Serum suPAR levels in patients with group 1 and group 4 pulmonary hypertension

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Background and Aim: Soluble urokinase type plasminogen activator receptor (suPAR) is a novel biomarker, mainly secreted from inflammatory and endothelial cells. suPAR reflects the chronic low-grade inflammation and associated with cancer, atherosclerosis and all-cause mortality. In this study we aimed to evaluate sUPAR levels in patients with group 1 and group 4 pulmonary hypertension (PH), a known inflammatory state.
Methods: We measured suPAR levels in 44 patients with PH; 36 patients with group 1 PH and 8 patients group 4 PH and 45 healthy controls. suPAR levels were measured using ELISA technique. Baseline clinical characteristics were assessed. Univariate and multivariate regression analyses were used to assess the association between the suPAR levels and the presence of PH.
Results: Mean age was 51.24±12.48 years in PH group and 51.05±18.18 years in control group (p=0.952). Systolic blood pressure (110 (100-130) vs. 120 (100-130) mmHg; p=0.041), heart rate (82.50 (70-95) vs. 70 (70-80) per minute; p=0.005), hemoglobin (12.65±2.33vs; 14.54±1.41 gm/dL; p<0.001), 6-MWD (372.50 (312.50-407.50) vs. 650 (622.50-680) meters; p<0.001) and NT- proBNP (442.00 (120.75-1516.75) vs; 37.60 (24.40-80.50) pg/mL; p<0.001), were significantly different in patients with PH when compared to control group. sUPAR levels significantly elevated in patients with PH when compared to controls (73.14 (62.77-167.13) vs. 65.52 (53.06-80.91) pg/mL; p=0.012). suPAR levels were similar between group 1 and group 4 PH (p=0.800). suPAR was found to be associated with the presence of PH on both the univariate and multivariate regression analysis (OR:1.007, p=0.047 and OR: 1.047, p=0.032 respectively). Multiple lineer regression analyses revealed that suPAR is significantly associated with only 6-minute walking distance.
Conclusions: suPAR levels are significantly elevated in patients with group 1 and group 4 PH. Large scale studies should further evaluate the role of suPAR as a prognostic biomarker and possible therapeutic target in patients with PH.

Pulmonary hypertension / Pulmonary vascular diseases

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The association of left atrial and ventricular strain and volumetric parameters between clinical, biohormonal and haemodynamic data in patients with pulmonary arterial hypertension

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Background and Aim: Pulmonary hypertension (ph) is a very rare and serious disease and effects not only right ventricle, but also left heart dynamics. Left ventricle and left atrium response to right ventricular overload on disease course is not clearly shown yet. Although cardiac magnetic resonance imaging has been the gold standard in evaluating cardiac function, it frequently requires sedation which can be avoided in patients with pulmonary hypertension. Thus, we aimed to evaluate 3D volumetric and 2D speckle tracking left ventricular and left atrial evaluation in patients with severe ph and its relation with clinical and biohormonal data and haemodynamic changes.
Methods: This is a cross-sectional study, approved by local ethics committee, all the participants provided written information consent. We enrolled 31 patients with different types of pulmonary hypertension. exclusion criteria were as follows, pulmonary hypertension due to left heart disease (type 2 pulmonary hypertension), patients with coronary artery disease, arterial hypertension,history of smoking, rhythm disturbances which can affect three-dimensional echocardiography image acquisition such as atrial fibrillation. Transthorasic echocardiography was performed in patients with severe pulmonary hypertension by using Philips IE33 equipment. Speckle tracking and 3D echocardiography evaluations were performed during a brief breath hold and with a stable ECG recording. 2D speckle tracking echocardiography was performed to derive left ventricle longitudinal strain and left atrial reservoir strain and strain rate, conduit strain rate and contraction strain rate. To simplify our strain examination, we only use longitudinal systolic strain, Right and Left heart catheterization were performed without any sedation. 7 F femoral sheaths were inserted to left and right femoral veins. Pig-tail catheter was used for both right and left heart pressure recordings. Swan-Ganz thermodilution catheter was also used instead of pig-tail catheher in order to avoid misinterpretations.
Results: 31 patients were enrolled to the study. 15 pts (48.4%) were male, 16 pts (51.6%) were female. PH subgroups were as follows: 14 pts with congenital heart disease and/or associated pulmonary arterial hypertension (45.2%), 12 pts with type 1 ph (38.7%), 4 patients with chronic thromboembolic pah (12.9%), 1 patient with group 5 ph (3.2%). Left atrial expansion index was found to be higher in patients with lower TAPSE and RV TDI St. (16 mm cut off for TAPSE, 10 mm for St) LAVI minimum and left ventricle end systolic volume was found to be lower in patients lower TAPSE. Total pulmonary gradient was correlated with 6 minutes walking test and BNP levels.
Conclusions: Patients with pulmonary hypertension except group 2 PH, have also decreased left atrial and left ventricular function. Patiens whose left end systolic left ventricular volume and LAVI-min are lower, hve tend to have higher BNP levels. Thus this may contribute to clinical worsening.

Pulmonary hypertension / Pulmonary vascular diseases

OP-243

A novel marker for predicting severity of acute pulmonary embolism: Systemic immune-inflammation index

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Background and Aim: Systemic pro-coagulatory and pro-inflammatory factors are the critical factors in acute pulmonary embolism (APE). Recently the systemic immune-inflammation index (SII) has emerged as a new inflammatory and prognostic marker. We aimed to determine whether there is a relationship between SII and the severity of the APE.
Methods: A total of 442 patients with APE, 202 women (45.7%) with an average age of 64±16, were included in this retrospective observational study.APE severity was classified as massive (high risk), submassive (intermediate risk), and nonmassive (low risk). On admission blood samples were collected for SII and other laboratory measurements. The SII was defined as platelet × neutrophil/lymphocyte counts.
Results: SII levels were higher in patients with massive APE and gradually increased from nonmassive to massive APE (p<0001). SII was also significantly higher in patients with in-hospital death. Multivariable analysis showed that SII was an independent predictor for massive APE (Odds ratio 1.005 (95% confidence interval 1.002 to 1.007), p<0.001), together with C-reactive protein and cardiac troponin. In receiver operating characteristic curve, optimal cutoff value of SII to predict a massive APE was 1161, with 91% sensitivity and 90% specificity (area under the curve: 0.957).
Conclusions: Our findings support an association between a higher SII level and a massive APE. As a simple biomarker SII is an independent predictor of more severe disease in patients with APE. SII is a more powerful tool than traditional inflammatory markers for predicting severity of disease in these patients.

Pulmonary hypertension / Pulmonary vascular diseases

OP-244

A novel prognostic marker in acute pulmonary embolism: Systolic aortic regurgitation

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Background and Aim: Aortic regurgitation usually occurs in diastole but systolic aortic regurgitation (SAR) is caused by the inability of ventricular contraction to overcome the aortic pressure in systole. The present study aimed to investigate the prevalence and SAR in acute piulmonary embolism.
Methods: We enrolled 208 consecutive patients (mean age of 71.3±9.5 years, 50% women) with acute piulmonary embolism and prospectively followed up for 1 year. Demographic, clinical, echocardiographic, and laboratory data were obtained at study entry. The primary endpoint of the study was cardiovascular mortality at one year.
Results: SAR was noted in 12 (5.8%) of the patients, and 20 patients (9.6%) died due to cardiovascular reasons at the end of the first year. The 1-year mortality rate was higher for patients with SAR (58.3%) compared to those without SAR (6.6%, p<0.001). After adjusting for important covariates, SAR remained independently associated with mortality (OR 3.657; 95% CI 1.674–8.567; p<0.001).
Conclusions: This is the first study to demonstrate that the presence of SAR is associated with adverse events in acute pulmonary embolism.

Pulmonary hypertension / Pulmonary vascular diseases

OP-245

Evaluation of thrombolytic treatment effect on frontal plane QRS-T angle in patients with acute pulmonary embolism (a novel marker of successful thrombolysis)

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Background and Aim: Acute pulmonary embolism (APE) causes right ventricular pressure overload, patients with hypotension and/or shock should be evaluated for thrombolytic therapy and hemodynamics often improves after thrombolytic therapy. Frontal plane QRS-T [f(QRS-T)] angle is between the directions QRS axis and T axis, was described as a novel marker of ventricular repolarization heterogeneity. With right ventricular pressure overload, axis of heart may be affected and thrombolytic treatment may have an effect on this situation. In this study, we aimed to investigate thrombolytic efficiency and effect on axis of heart by using f(QRS-T) angle.
Methods: 61 APE patients treated with thrombolytic and 71 APE patients without thrombolytics were included. 61 patients with right ventricle presure overload in imaging modalities (computed tomography and/or echocardiography) and hemodynamic insatbility were treated with thrombolytics. Clinical findings and ECGs at diagnosis were collected. Second ECGs (control ECGs) were accepted as first 24 hours after treatment in patient with thrombolysis, at least 72 hours after treatment in patient without thrombolysis.
Results: There was no significant differences were observed in gender, age, hypertension, diabetes and cardiovascular disease. Respiartory rate, heart rate, pulmonary artery systolic pressure were significantly higher; saturation O2, systolic and diastolic blood pressure were significantly lower In patients treated with thrombolytic (Table 1). f(QRS-T) angle was significantly higher in APE with right ventricular pressure overload and was significantly changed after thrombolytic (Table 2). After trombolytic treatment, f(QRS-T) angle decreased to the level of patients without right ventricular pressure overload (Table 3).
Conclusions: Right ventricular pressure overload in APE is basically described by right ventricular dilation and/or the ratio of right ventricle to left ventricle >0.9 in imaging modalities. Right ventricular pressure overload with hemodynamic instability is indication for thrombolysis. Right ventricular pressure overload in APE has an effect on f(QRS-T) angle and after thrombolytic treatment, the change of f(QRS-T) angle may be a marker of successful thrombolytics efficiency.

Table 1. Baseline characteristics of the study groups

	Thrombolytic group n=61	Non-thrombolytic group n=71	P value
Age	58.61±16.85	62.75±13.98	0.125
Gender			
Female	34 (55.7%)	40 (56.3%)	0.945
Male	27 (44.3%)	31 (43.7%)	
Hypertension	10 (16.4%)	12 (16.9)	0.994
Diabetes Mellitus	5 (8.2%)	7 (9.9%)	0.772
ASCVD	4 (6.6%)	6 (8.5%)	0.752
Temperature (°C)	36.34±0.65	36.33±0.29	0.902
Respiratory rate	26.3±11.93	22±2.93	**0.006**
Heart rate (b.p.m)	109.51±13.98	100.59±11.40	**<0.001**
Systolic Blood Pressure (mmHg)	105,77±14.64	111.55±11.26	**0.012**
Diastolic Blood Pressure (mmHg)	65.59±9.72	71.69±7.02	**<0.001**
$SatO_2$ (%)	83.54±7.01	86.59±4.73	**0.005**
Pulmonary Artery Systolic Pressure (mmHg)	55.03±13.99	39.17±17.11	**<0.001**

Table 2. Comparisons of ECG parameters

	Thrombolytic group n=61	Non-thrombolytic group n=71	P value
Heart $rate_1$ (b.p.m.)	111.95±12.82	100.75±15.56	**<0.001**
QTc_1 (ms)	402.38±49.45	406.66±17.12	0.495
F QRS-T^0_1	62.43±36.99	48.38±31.15	**0.019**
Heart $rate_2$ (b.p.m.)	94.72±12.73	93.45±11.68	0.551
QTc_2 (ms)	413.03±20.02	407.52±15.95	**0.081**
F QRS-T^0_2	48.87±26.75	45.99±30.96	0.571

Table 3. Differences between before thrombolytic treatment and after thrombolytic treatment

	Before Thrombolytic	After Thrombolytic	*P* value
Systolic Blood Pressure (mmHg)	98.11±11.97	113.51±11.49	**<0.001**
Diastolic Blood Pressure (mmHg)	61.20±8.73	71.10±6.95	**<0.001**
Heart rate (b.p.m)	111.95±12.82	94.72±12.73	**<0.001**
f(QRS-T) angle (°)	62.43±36.99	48.87±26.75	**0.022**

Pulmonary hypertension / Pulmonary vascular diseases

OP-246

The relationship between whole blood viscosity and acute pulmonary emboli

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Background and Aim: The aim of this study was to invastigate the potential reliationship between whole blood viscosity (WBV) and acute pulmonary emboli (APE).
Methods: In this study, 105 patients who applied to the cardiology department and emergency department diagnosed with acute pulmonary emboli. January 2016 and May 2020, as well as 105 healthy population as a control group were included in the study. The measurement of WBV was carried out at both high shear rate (HSR)(208/s) and low shear rate (LSR) (0.5/s) by previously validated formulae using hematocrit (HcT) and total protein in g/L. WBV at HSR (208/s) is: (0.12 ×HcT) + 0.17 (TP-2.07) and WBV at LSR (0.5/s) is: (1.89 × HcT) + 3.76 (TP-78.42). The whole blood viscosity of acute pulmonary emboli patients and of control group were compared at both HSR and LSR.
Results: Baseline clinical characteristics and laboratory parameters of groups were reported in Table 1. The prevalence of smoking, diabetes mellitus were higher in the peripheral group. Also, total protein, LDL, hematocrit levels were statistically higher in patients with peripheral emboli. Peripheral emboli patients had significantly higher WBV for LSR (53.1±9.6 vs 47.3±4.3; p<0.001) and HSR (16.4±0.6 vs 14.9±0.2; p<0.001) (Table 1). Univariate analyses identified the following variables which were significantly associated with acute pulmonary emboli patients: smoking, diabetes mellitus, LDL, HSR, LSR. In multivariate analysis, two different models were constituted for WBV levels at each shear rate. In models adjusted with smoking, LDL, WBV at LSR (OR: 3.377, 95% CI: 1.209–9.431, p=0.006) and WBV at HSR (OR: 1.070, 95% CI: 1.020–1.123, p=0.02) were shown as independent predictors (Table 2). In ROC analysis for prediction of acute pulmonary emboli, a cut-off value 16.98 for WBV at HSR had 72% sensitivity and 68% specificity (p<0.001, AUC=0.75) and a cut-off value 56.8 for WBV at HSR had 78% sensitivity and 58% specificity (p<0.001, AUC=0.71.5) (Figure 1).
Conclusions: In conclusion, whole blood viscosity is found out to be an important and independent risk factor in patients with acute pulmonary emboli.

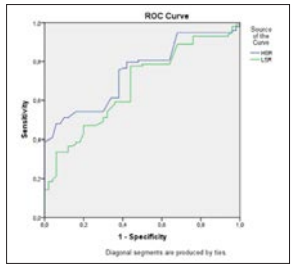

Figure 1.

Table 1. Baseline characteristics and laboratory findings of the study group

	Acute Pulmonary Emboli (n=105)	Controls (n=105)	p-value
Age, years (mean, SD)	72±9,8	70,7±2,7	0,32
Men, n (%)	53,1	44	0,29
HTN, n (%)	84	86	0,71
DM, n (%)	48	30	0,03
Coronary artery disease, n (%)	34	29	0.059
Smoking	46,9	26	0,015
BMI (kg/m2)	27,4±5	26,46±3,7	0.25
sPAB	48,1±10,1	26±7,7	0.002
LVEF (%)	56,7±4,4	56,4±5,1	0.702
Triglyceride (mg/dl)	148,7±68,5	141,3±84,2	0.56
Hemoglobin (g/dL)	14,5±1,1	14,2±0,5	0.04
Hematocrit (%)	43,78±2,9	42,64±1,46	0.01
Total protein (g/l)	73,41±2,63	72,3±1,72	0.008
Platelets (10^3)	259,8±90	249,5±64	0.47
Creatinine (mg/dL)	1,2±0,91	1,08±0,8	0.41
D-dimer	6,7±4,4	1,1±0,9	0.001
WBV at LSR, 208 s–1	53,1±9,6	47,3±4,3	<0.001
WBV at HSR, 0.5 s–1	16,4±0,6	14,9±0,2	<0.001

Table 2. The effects of variables on acute pulmonary emboli in univariate and multuvariate logistic regression analysis

Variables	Odds Ratio (OR)	95% CI	p-value
Univariate logistic regression analysis			
Age	1.022	(0.998– 1.047)	**0.030**
Hypertension	1.869	(0.879– 3.975)	**0.021**
Diabetes Mellitus	1.739	(1.011 – 2.992)	**0.011**
Coronary artery disease	1.347	(0.769 – 2.359)	0.292
Smoking	1.698	(1.017– 2.833)	**0.045**
sPAB, mmHg	1.008	(0.998– 1.019)	**0.021**
Triglycerides, mg/dl	1.002	(0.997 – 1006)	0.470
White Blood Cell (10^3 µl)	1.017	(0.934 – 1.108)	0.690
Platelet (10^3 µl)	1.001	(0.997– 1.004)	0.681
WBV at LSR ($0,5/s^{-1}$)	1.047	(1.022 – 1.074)	**<0.001**
WBV at HSR ($208/s^{-1}$)	2.547	(1.541 – 3.917)	**<0.001**
Multivariate logistic regression analysis			
MODEL-1			
sPAB, mmHg	1.010	(0.997– 1.023)	0.128
Age	0.531	(0.157 – 1.797)	0.309
Hypertension	1.939	(0.798– 4.712)	0.055
Diabetes Mellitus	1.254	(1.209 – 1.303)	0.078
Smoking	1.911	(1.108– 3.652)	0.052
WBV at LSR ($0,5/s^{-1}$)	2.121	(1.079 – 3.164)	**<0.001**
MODEL-2			
sPAB, mmHg	1.008	(0.997 – 1.020)	0.172
Age	1.023	(0.998 – 1.050)	0.079
Hypertension	1.914	(0.849 – 4.314)	0.067
Diabetes Mellitus	1.290	(1.112 – 1.562)	0.062
Smoking	1.914	(1.100 – 3.032)	0.058
WBV at HSR ($208/s^{-1}$)	2,737	(1.671 – 4.483)	**<0.001**

WBV at HSR: Whole blood viscosity at high shear rate, WBV at LSR: Whole blood viscosity at low shear rate. LDL, low-density lipoprotein. eGFR, estimated Glomerular filtration rate

Pulmonary hypertension / Pulmonary vascular diseases

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Prognostic significance of immature granulocyte index in acute pulmonary embolism

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Background and Aim: Acute pulmonary embolism(APE) is one of the leading causes of morbidity and mortality in the world. Inflammation has an important role in both the pathophysiology and prognosis of APE. Immature granulocytes index (Igx) known as an early marker of sepsis reflecting the inflammation. The goal of the present study was to evaluate the prognostic value of Igx, all parameters of complete blood count (CBC), and a simplified pulmonary embolism severity index (PESI) scoring system in patients with APE admitted to the emergency department.
Methods: A total of 285 patients who were hospitalized with the diagnosis of an acute PE were retrospectively enrolled in the study. All demographic and laboratory parameters of the patients were scanned through the electronic information management system of the hospital.
Results: Two hundred and eighty-five patients consisting of 121 males (42.5%) and 164 females (57.5%) were included in the study. The all-cause mortality during the hospital stays was 12.63 %. Igx, white blood cell (WBC), mean platelet volume(MPV), and PESI score were significantly higher in patients who died during the hospitalization [(3.2 vs. 0.7 p=0.01), (12.1 vs. 9.3 p=0.01), (10.4 vs. 10 p=0.01), and (178.8 vs.87.1 p=0.01) respectively] when compared to the patients that survived. On the contrary, platelet count were observed lower in non-survival group (237.4 vs. 279.8 p=0.01). There was no statistically significant difference in neutrophil-to-lymphocyte ratio (NLR) and platelet- to- lymphocyte ratio (PLR) between the two groups. According to the receiver operating characteristic curve analysis(ROC), the cut-off Value of Igx was 0.6 (AUC±SE= 0.769±0.048; p<0.001, Sensitivity=75, Specificity=71.3).
Conclusions: In conclusion, Igx and PESI score are useful markers to predict the prognosis of patients with APE. High Igx values may alert physicians earlier for possible poor prognosis.

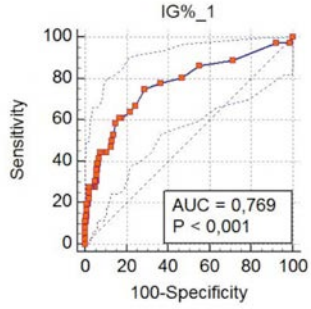

Figure 1. Receiver operating characteristic curves for the predictability of the Igx on hospital mortality.

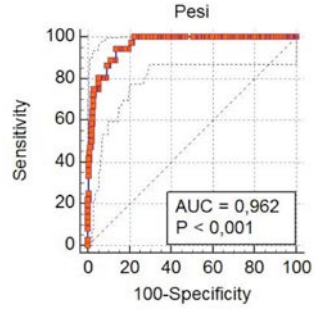

Figure 2. Receiver operating characteristic curves for the predictability of the PESI on hospital mortality.

Table 1. Comparison of laboratory parameters

Hematological parameters	Survivors (n = 249)	Non-survivors (n =36)	p value
Hemoglobin (g/Dl)	11,9±1,9	10,5±1,8	<0,01
WBC count (103/L)	9,3±4,3	12,1±7,6	<0,01
Neutrophil count (103/L)	6,7±4,0	8,3±6,3	0,25
Lymphocytes count (103/L)	1,7±0,9	2,9±6,1	0,74
Platelet count (103/L)	279,8±122,3	237,4±163,5	0,01
RDW (%)	46,2±7,3	53,2±10,5	<0,01
MPV (fL)	10,0±0,8	10,4±1,01	0,01
NLR	5,4±5,9	10,0±13,8	0,21
PLR	207,3±154,4	228,4±210,9	0,81
IG(%)	0,7±1,2	3,2±3,8	<0,01
Creatinine (mg/dL)	1,02±0,88	0,9±0,4	0,82
Hs-Troponin T (ng/mL)	159,4±765,2	129,8±259,5	0,01
BNP (pg/mL)	3342,4±5466,9	10356,7±7962,5	0,02
CRP	70,8±35,7	101,5±96,4	0,02

Pulmonary hypertension / Pulmonary vascular diseases

OP-248

Assessment of quality of life in patients with pulmonary arterial hypertension using WHOQOL-BREF questionnaire during COVID-19 pandemic

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Background and Aim: Increasing numbers of COVID-19 patients induced temporary redistribution and reorganization. Elective admittance for therapeutic purposes has been postponed for a very short period. We have transformed our daily practice with pulmonary arterial hypertension (PAH) population to telephone visits for prevention of infection. This document was designed to represent the PAH patients course, relation with COVID-19 and quality of life (QOL) during pandemic.
Methods: A cross-sectional study was conducted among 75 patients with PAH during COVID-19 pandemic. Demographic data including age, sex, duration of illness, duration on medical treatment were collected. QOL was assessed using the World Health Organization Quality of life-BREF(WHOQOL-BREF) questionnaire. Four domains (physical, psychological, social, environmental) of the WHOQOL-BREF were the primary end points. Each item is rated by a 5-point Likert scale. Higher score reflects a better QOL. Bivariate relationship between sociodemographic factors and QOL scores were analyzed using independent samples t-test. Multiple linear regression analysis was performed to determine independent predictors of QOL. Also second questionnaire was conducted for collecting the data during pandemic regarding the application to hospital due to PAH spesific/COVID related symptoms, hospital stay, COVID-19 PCR results if implemented, vaccine history.
Results: 75 respondents' distrubution has demonstrated as followed: 38.6% idiopathic pulmonary arterial hypertension; 1.33% drugs and toxins induced; 33.3% connective tissue disease associated; 6.6% PAH after the correction of the congenital defect without any residual shunt, 20% Eisenmenger patients. Mean age was 53.54±15.02.The majority of the patients were married (60%), retired due to disability (46.6%), and

educated (58.6%). Cronbach's alpha for all 26 questions of WHOQOL-BREF was acceptable (0.892). The following values of Cronbach's alpha for each domain: physical (0.926), psychological (0.871), social (0.782), environmental (0.820). Following QOL scores were recorded: physical (47.16±15.34), psychological (54.1±17.52), social (46.3±19.64), environmental domain (45.9±17.79). Younger age and being married was associated with a better QOL score in the social domain (p=0.012, p=0.005). Employed patients scored better in the environmental domain (p=0.027). Unemployed patients had significantly low scores in overall perception of health (p<0.05) as compared to other groups. Advanced functional class was found to be the only independent negative predictor of QOL in patients with PAH (p<0.05).
Conclusions: Patients with PAH had overall low QOL scores in all four domains during COVID-19 pandemic. None of the patients diagnosed as COVID-19. Considerable amount of patients (65%) administered self-isolation from the beginning of the COVID-19.But this self-isolation rendered the patients' mood to a more depressive and hopeless state. Advanced functional class was found to be the only independent negative predictor of QOL in patients with PAH.

Pulmonary hypertension / Pulmonary vascular diseases

OP-249

The correlation between suicidal ideation and self-esteem in pulmonary arterial hypertension patients

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Background and Aim: In pulmonary arterial hypertension (PAH) patients, despair and anxiety symptoms and depression are common due to the clinical symptoms of the disease and their negative impact on patient's quality of life, and the resulting socio-economic problems. These psychiatric symptoms that could accompany the primary disease could lead to a decrease in self-esteem and suicidal ideation in this group of patients. The present study aimed to investigate the frequency of suicidal ideation and its correlation with other clinical variables in PAH patients.
Methods: A patient group of 50 pulmonary hypertension patients, and a healthy control group of 50 individuals were included in the study. A socio-demographic and clinical data form was completed by both the patient and control groups and Beck Depression Inventory (BDI), Beck Hopelessness Scale (BHS), Rosenberg Self-Esteem Scale (RSE) and Suicide Probability Scale (SPS) were applied to the same groups. SPSS version 22 software was used in statistical analysis.
Results: It was determined that the mean right ventricular diameter of the patients was 22.54±3.32, mean systolic pulmonary pressure was 47.48±18.86, and mean pulmonary artery pressure was 33.32±19.69. BHS, BDI and SPS total scores were statistically significantly higher in the patient group when compared to the control group (p<0.001, p=0.001, p=0.026). RSE scale scores were also higher in the patient group compared to the control group (p=0.017). (A high score on the RSE indicates a low self-esteem level).
Conclusions: It is important to identify the PAH patients with intense feelings of hopelessness and depressive symptoms and to provide psychiatric treatment and conduct psychotherapeutic interventions to improve the self-esteem of these individuals.

Table 1. Patient and control group scale scores

	Patient n:50 mean±std.d	Control n:50 mean±std.d	p	t
Age	48.6±12.2	46.3±13.3	0.367	0.907
Beck Hopelessness Scale	6.8±4.08	3.92±2.8	0.000	4.116
Beck Depression Inventory	10.46±6.24	6.54±5.49	0.001	3.337
Rosenberg Self-Esteem Scale	0.83±0.72	0.55±0.38	0.017	2.435
Total Suicide Probability Scale	61.0±14.7	55.3±9.85	0.026	2.262

Other

OP-250

Evaluation of cardiology consultation quality and quantity requested from emergency departments in Turkey

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Background and Aim: Emergency cardiac consultation is a very big part of cardiology practice and there is not any trial evaluating this subject qualitatively and quantitatively with different perspectives yet. We wanted to investigate this process in terms of correlation between emergency physicians and cardiologists.
Methods: In this cross-sectional, observational and multicenter trial, we analysed the consultation data of 627 patients in 5 different state hospitals who were consulted to cardiology for different reasons. The main aims of the study was investigating the necessity and intensity of cardiac consultations and the correlation between emergency physicians and cardiologists in terms of diagnosis accuracy, electrocardiogram (ECG) interpretation and first medical management. Furthermore, finding the differences or similarities of consultation process in emergency medicine specialists (EMS) and practitioners.
Results: Within the first 60 days of attending cardiologists, all consultations were recorded and analysed. Totally 502 patients by practitioners and 125 patients by EMS were consulted to cardiology for any reason. The most common admission cause was chest pain (47.8%), and the most common cause of consultation was having no/weak idea about patient's clinical diagnosis (39.9%). A total of 620 patients (98.8%) were consulted within the first 5 hour and 45.6% of patients were within the first hour. The diagnosis consistency of 48% of consulted patients by EMS was excellent while it was 32.1% in practitioners (p=0.001). Good and excellent ECG interpretation of EMS was 72.8% and it was 50.7% in practitioners (p<0.001). Good and excellent first medical treatment were 46.4% in EMS while it was 38.4 in practitioners (p=0.05). Nearly half of consultations (48.8) were considered as definitely unnecessary or unnecessary by cardiologists. There was statistically significant correlation between the necessity of consultation and last decision (r=0.811, p<0.001).
Conclusions: There is a big variability in emergency cardiac consultations from the standpoint of consultation necessity, ECG interpretation, first medical management quality and accurate diagnosis. Adequate and high-quality medical school education and emergency residency training are the cornerstones of improving the quality of consultation and speeding up the process. They can also improve communication of emergency physicians and other departments.

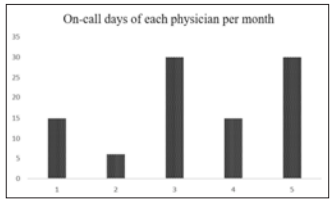

Figure 1. Frequency of consulting days per month for each cardiologist.

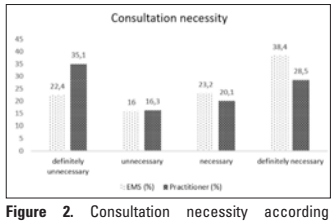

Figure 2. Consultation necessity according to consulting pyhsician (data are shown in percentages).

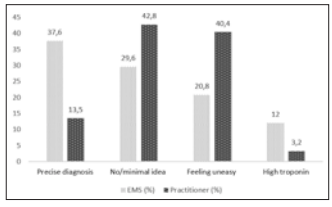

Figure 3. The causes of consultations according to physicians (data are shown in percentages).

Table 1. Baseline characteristics of patients and consultation process

Variable		n (%)
Consulting physician	EMS	125 (19,9)
	Practitioner	502 (80,1)
Gender	Male	335 (53,4)
Age	≥75	183 (29,2)
	<75	444 (70,8)
HT		365 (58,2)
Smoking		179 (28,5)
HL		32 (5,1)
DM		168 (26,8)
Prior Heart Disease		209 (33,3)
Chief Complaint	Chest pain	300 (47,8)
	Dyspnea	107 (17,1)
	Palpitation	83 (13,2)
	Syncope/presyncope	57 (9,1)
	Other	80 (12,8)
Consultation within	1 hour	286 (45,6)
	1-5 hour	334 (53,2)
	>5 hour	7 (1,2)
Time interval	8-16	288 (45,9)
	16-24	253 (40,4)
	24-08	86 (13,7)

Table 2. Consultation cause, accurate diagnosis, ECG interpretation, consultation necessity and last decision correlation between EMS and practitioners

		EMS n (%)	Practitioner n (%)	p value
Cause of consultation	Precise diagnosis	47 (37,6)	68 (13,5)	<0,001
	No/minimal idea	37 (29,6)	215 (42,8)	0,001
	Feeling uneasy	26 (20,8)	203 (40,4)	0,001
	High troponin level	15 (12)	16 (3,2)	0,003
Diagnosis consistency	Very bad	18 (14,4)	73 (14,5)	0,48
	Bad	26 (20,8)	160 (31,9)	0,004
	Moderate	7 (14,3)	42 (8,4)	0,12
	Good	14 (11,2)	66 (13,1)	0,72
	Excellent	60 (48)	161 (32,1)	0,001
ECG interpretation	Very bad	3 (2,4)	55 (11)	<0,001
	Bad	12 (9,6)	119 (23,8)	<0,001
	Moderate	19 (15,2)	72 (14,4)	0,59
	Good	18 (14,4)	94 (18,8)	0,88
	Excellent	73 (58,4)	159 (31,9)	<0,001
Consultation necessity	Definitely unnecessary	28 (22,4)	176 (35,1)	0,002
	Unnecessary	20 (16)	82 (16,3)	0,46
	Necessary	29 (23,2)	101 (20,1)	0,23
	Definitely necessary	48 (38,4)	143 (28,5)	0,019
Last decision	Discharge	44 (35,2)	295 (58,8)	<0,001
	Hospitalization	57 (45,6)	105 (20,9)	<0,001
	Transfer	24 (19,2)	102 (20,3)	0,6

Other

OP-251

Mortality in STEMI and NSTEMI patients at 1 year: Results of the nationwide TURKMI registry

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Background and Aim: Recent TURKMI registry demonstrated that treatment guidelines are largely implemented in patients with acute myocardial infarction (MI). We aimed to assess the risk of mortality at 1 year.
Methods: Fifty centres were selected from the EuroNUTS-Stat regions of Turkey using a sampling weight proportional to the census. All consecutive patients admitted to these centres between 1th-16th November 2018 with a diagnosis of acute MI were prospectively enrolled. Mortality rates at 1 year for STEMI and NSTEMI were compared using restricted mean survival time (RMST) ratio, adjusting for age, sex, and history of diabetes mellitus, hypertension, smoking, heart failure and coronary artery disease. Mortality rates for gender were compared using Kaplan-Meier method.
Results: Among 1930 patients admitted with MI (mean age 62±13 years, 26% female), 1195 (62%) had non-ST segment elevation MI (NSTEMI) and 735 (38%) had ST segment elevation MI (STEMI). During 1-year of follow-up, 174 patients died (cumulative risk of mortality 9.1%, 95% CI 7.9-19.5), of whom 62 had STEMI and 112 had NSTEMI, which corresponds to a cumulative risk of mortality 8.5% (95% CI 6.7-10.8) for STEMI and 9.5% (95% CI 7.9-11.3) for NSTEMI. Although the risk of mortality for STEMI was higher than NSTEMI at early period, the risk equalized nearly at 6 month, and became slightly higher but non-significant in NSTEMI (RMST ratio 0.988, 95% CI 0.963-1.013; p=0.325; Figure 1). Women with STEMI had significantly higher mortality compared to men (12.6% [95%CI 10.0-15.9) vs 7.8% [95% CI 6.6-9.4); log-rank p=0.001; Figure 2).
Conclusions: Risk of mortality was higher for STEMI at earlier stages but became slightly higher in NSTEMI. Women with STEMI has a very high risk of mortality compared to men.

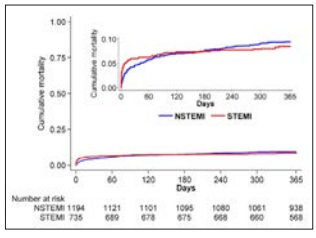

Figure 1.

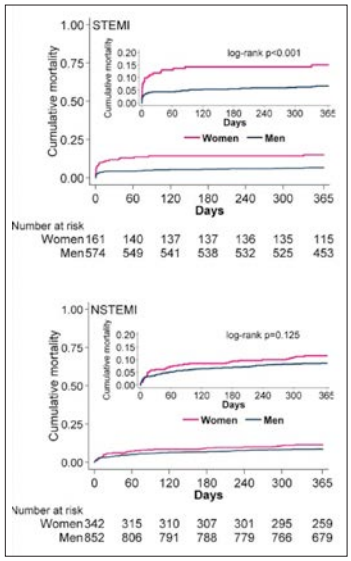

Figure 2.

Other

OP-252

Anti-platelet regimen, revascularization and in-hospital mortality rates in elderly patients with myocardial infarction- data from TURKMI Study

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Background and Aim: Despite improvements in early revascularization and medical therapy in myocardial infarction (MI), evidence-based medical treatment and revascularization remain inadequate in elderly MI patients compared to younger patients and these high-risk individuals are subjected to more conservative treatment strategies. The aim of this study was to present the course of MI including the revascularization procedures, anti-platelet regimen, and in-hospital mortality in elderly population in Turkey.
Methods: All data were collected from the registry, TURKMI Study which was designed as a national, multi-center, observational study. The population consisted of all consecutive patients presenting with acute MI in the participating hospitals (n=50) and divided into two groups according to their ages (<75 y vs. ≥75 y). All patients received standard therapy according to current guidelines.
Results: Of the 1930 patients enrolled 362 were ≥75 years of age. The proportion of patients undergoing coronary angiography and primary percutaneous interventions (PCI) were significantly lower in the elderly (96.4% vs.81.8%, p<0.001 and 42.6% vs.35.1%, p=0.01, respectively). In 5.6% patients in elderly group, no TIMI flow was obtained by coronary intervention, while TIMI 0 flow was observed only 2.2% in non-elderly group (p=0.009). Although bleeding was more common in elderly patients, it was not statistically significant (p=0.051). The proportions of cardiogenic shock, arrhythmias including sustained ventricular tachycardia, and ventricular fibrillation leading to cardiac arrest were higher in the elderly. In hospital mortality was also significantly higher in the elderly patients (9.1% vs.2.7%, p<0.001). When we compare the medications prescribed at discharge; clopidogrel was preferred in 71.2% of elderly patients, while almost half (45%) of non-elderly patients were prescribed ticagrelor at discharge from the hospital. Dual-antiplatelet therapy was prescribed in non-elderly patients with MI more than elderly patients (94.5% vs. 91.4%, p=0.032). Since atrial fibrillation was more frequent in elderly patients with high CHADS scores, anticoagulant therapy was initiated in more patients in this group. (7.7% vs.4.5%, p=0.013) Among all anticoagulants Apixaban was preferred most in elderly patients.
Conclusions: In our study, elderly patients with MI mostly underwent coronary angiograms contrary with the previous data. Although in hospital mortality rates were higher in elderly patients compare to non-elderly patients, in hospital mortality was also found much lower than the previous data. Clopidogrel remains the antiplatelet choice in most elderly patients. However in our study, the use of ticagrelor in non-elderly patients was almost close to clopidogrel in accordance with the recent data. Dual antiplatelet strategy was still preferred more frequently in non-elderly patients. These results could be interpreted as elderly patients are still subjected to more conservative treatment strategies.

Other

OP-254

Treatment delays and in-hospital outcomes in acute myocardial infarction during the Covid-19 Pandemic: A nationwide study

Mustafa Kemal Erol,[1] Meral Kayıkçıoğlu,[2] Mustafa Kılıçkap,[3] Arda Güler,[4] Abdullah Yıldırım,[5] Fatih Kahraman,[6] Veysi Can,[7] Sinan Inci,[8] Sadettin Selçuk Baysal,[9] Okan Er,[10] Utku Zeybey,[11] Çağrı Kafkas,[12] Çagrı Yayla,[13] Can Baba Arın,[14] Ibrahim Aktas,[15] Ahmet Arif Yalçın,[4] Ömer Genç, On The Behalf Turkmi 2 Study Group[5]

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Background and Aim: Delay on admission of myocardial infarction (MI) is an important prognostic factor. In this nationwide registry, we compared treatment delays and outcomes of patients with acute MI in Covid-19 pandemic period with a recent pre-pandemic registry conducted at the same centers.
Methods: A recent nationwide registry (TURKMI-1) enrolled consecutive patients with acute MI in November 1-15, 2018 in Turkey, and assessed time delay at each step from symptom-onset to treatment and outcomes. Fifty centers were selected using probability sampling. In the present study (TURKMI-2), the same information

was obtained from 48 of the same 50 centers during the pandemic between April 17 and May 2, 2020.
Results: In a 2-week period, 991 patients (51.1% NSTEMI, 48.9% STEMI) were admitted to the study centers within the 48-hour of symptom-onset. Compared with the TURKMI-1, admissions decreased by 31.2% in ST-segment elevation MI (STEMI), and 56.4% in non-STEMI (NSTEMI) (Figure 1). Median time from symptom-onset to hospital-arrival increased from 295 min to 419 min in NSTEMI, and from 150 min to 185 min in STEMI (p-values <0.001). Door-to-balloon time was similar in the two periods (37 vs. 40 min, p=0.448), however, total ischemic time increased significantly (195 min vs 245 min, p=0.001) mainly due to patient-related delay (figure 2,3). Percutaneous coronary intervention (PCI) was decreased especially in the NSTEMI group (60.3% vs 47.4% in NSTEMI, p<0.001; 94.8% vs 91.1% in STEMI, p=0.013). Major cardiac adverse events (MACE) defined as in-hospital death, heart failure, or cardiogenic shock were significantly higher in the pandemic period compared to non-pandemic (4.8% vs 8.9%; p<0.001). Age and sex adjusted risk of MACE was two times higher during pandemic (Odds ratio [95% confidence interval] was 1.96 [1.20-3.22] for NSTEMI, p=0.007; and 2.08 [1.38-3.13] for STEMI, p<0.001) (figure 4).
Conclusions: Besides the overall 47.1% reduction in acute MI admissions, there was a significant patient related treatment delay during the pandemic. Although PCI was performed in a timely fashion, increase in total ischemic time and decrease in PCI might be responsible for the increased risk of MACE in acute MI.

Other

OP-255

Mortality Predictors for severe coronavirus disease 2019 patients who suffered miyocardial injury: A single center experience

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Background and Aim: To investigate the clinical features of the severe coronavirus disease 2019 (COVID-19) patients with myocardial injury and predictors of adverse outcome in this population.
Methods: The 81 consecutive severe COVID-19 patients who suffered myocardial injury were admitted to this study, from 15 March to 30 April 2020. The demographic characteristics (age and gender), clinical data (comorbidities, laboratory findings, treatments, complications, and outcomes), laboratory findings and results of cardiac examinations (cardiac biomarkers) for participants during hospitalization were were collected by using electronic medical records. The study outcome was rate of in-hospital death, and the national death notification system and hospital records were used to obtain information on mortality.
Results: A total of 81 severe COVID-19 cases (mean age of 63±14 years) with myocardial injury (26%) constituted the study population. During the follow up period (median 13 days), in-hospital mortality was observed in 27 (33.3%) patients. Compared to the survivors, those who died were older (60±13 vs 70±14; p<0.01, respectively). In terms of the most common main comorbidities, hypertension (33.3% vs 59.3%; p=0.03), coronary artery disease (9.3% vs, 44.4%; p<0.01), heart failure (7.4% vs 37%; p<0.01, respectively), diabetes (20.4% vs 44.4%; p=0.02), and chronic renal failure (9.3% vs, 29.6%; p=0.02) were frequently observed in nonsurvivors than the others. Acute heart failure were also more frequent in nonsurvivors (p=0.03) and two of the nonsurvivors had malignant ventricular arrhythmia. Detailed demographic, clinical and laboratory characteristics of the population are summarized in Table 1 and Table 2. Multivariate cox regression analysis by using Backward LR method revealed that to be older age (HR: 1.049; p=0.04), coronary artery disease history (HR: 3.098; p=0.02), decreased eGFR value (HR: 0.984; p=0.04), increased admission CRP level (HR: 1.006; p<0.01) and higher peak Dimer level (HR: 1.567; p=0.04) were independent predictors for in-hospital mortality. The area under the ROC curves of hs-TnI, CK-MB, peak D-Dimer, and admission CRP for the in-hospital mortality were 0.81, 0.80, 0.72, and 0.79, respectively (all p<0.01). The single cut-off concentrations of hs-TnI, CK-MB, peak D-Dimer, and admission CRP were >309 pg/mL, > 15 ng/mL, >2.5 mg/L, and >60 mg/L, respectively. On Kaplan–Meier analysis, levels of the peak cTnI, peak CK-MB, baseline CRP and peak D-Dimer above aforementioned cut-offs were significantly associated with higher in-hospital mortality (Figure 2).
Conclusions: The risk of in-hospital mortality was significantly associated with older age, inflammatory response, and cardiovascular comorbidities and can be predicted by the biomarkers for the myocardial injury in severe COVID-19 patients.

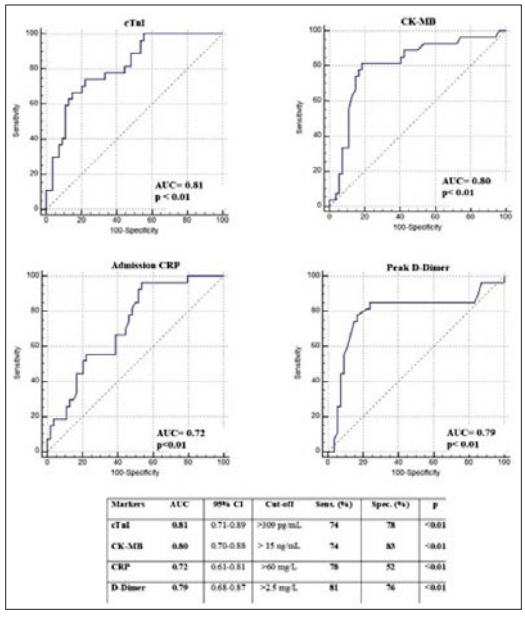

Figure 1. Receiver operating characteristic curve analysis of the biomarkers for determining the in-hospital mortality in severe COVID 19 patients with myocardial injury.

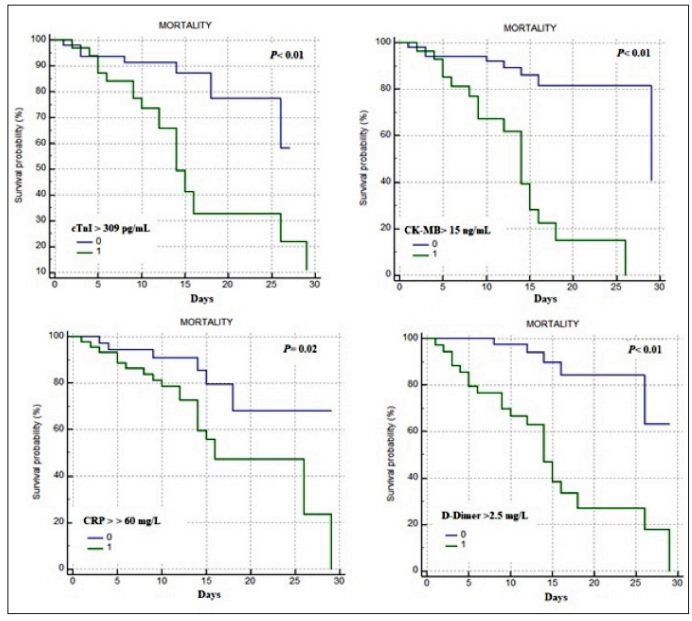

Figure 2. Kaplan–Meier plots of survival curves of low- (blue line) and high-risk (green line) patients who grouped according to cut-off values determined by youden index.

Table 1. Demographic and admission clinical parameters of the study cohort

Variables	All population (n= 81)	Survivors (n= 54)	Non-survivors (n= 27)	p
Male gender, n (%)	55 (67.9)	34 (63)	21 (77.8)	0.18
Age, year	63±14	60±13	70±14	<0.01
Body Mass Index (kg/m2)	25.9±2.9	26.1±3.1	25.7±2.7	0.53
Hypertension, n (%)	34 (42)	18 (33.3)	16 (59.3)	0.03
Coronary artery disease, n (%)	17 (21)	5 (9.3)	12 (44.4)	<0.01
Heart Failure, n (%)	14 (17.3)	4 (7.4)	10 (37)	<0.01
Atrial fibrillation, n (%)	8 (9.9)	3 (5.6)	5 (18.5)	0.07
Diabetes Mellitus, n (%)	23 (28.4)	11 (20.4)	12 (44.4)	0.02
CVA, n (%)	5 (6.2)	3 (5.6)	2 (7.4)	0.74
Current Smoking, n (%)	30 (37)	22 (40.7)	8 (29.6)	0.33
COPD, n (%)	16 (19.8)	8 (14.8)	8 (29.3)	0.11
Cancer, n (%)	10 (12.3)	4 (7.4)	6 (22.2)	0.06
Chronic renal disease, n (%)	13 (16)	5 (9.3)	8 (29.6)	0.02
ACEI/ARB use history, n (%)	32 (39.5)	21 (38.9)	11 (40.7)	0.87
SOFA score, n (%)	4.6±1.6	3.9± 1.1	6±1.7	<0.01
ICU need, n (%)	51 (63)	27 (50)	24 (88.9)	<0.01
Invasive mechanical ventilation, n (%)	34 (42)	15 (27.8)	19 (70.4)	<0.01
ARDS, n (%)	29 (35.8)	14 (25.9)	15 (55.6)	<0.01
Malign Ventricular Arrythmia, n (%)	2 (2.5)	0 (0)	2 (7.2)	0.04
Acute heart failure, n (%)	7 (8.6)	2 (3.7)	5 (18.5)	0.03
Acute kidney injury	9 (11.1)	4 (7.4)	5 (18.6)	0.13
Multiorgan dysfunction syndrome	3 (3.7)	0 (0)	3 (11.1)	0.01
Length of hospital stay in days, median, [IQR]	13 [8-17]	13 [9-17]	12 [5-15]	0.29

CVA, cerebrovascular accident (stroke or transient ischemic attack); COPD, chronic obstructive pulmonary disease; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; SOFA, sequential organ failure assessment; ICU, intensive care unit; ARDS, acute respiratory distress syndrom.

Table 2. Laboratory parameters of the study population

Variables	All population (n= 81)	Survivors (n= 54)	Non-survivors (n= 27)	p
FBG, mg/dL	149±53	138±44	171±64	<0.01
eGFR, mL/min/1.73m2, median, [IQR]	81 [64-100]	90 [65-100]	74 [33-95]	0.03
Neutrophil, 10^3/uL, median, [IQR]	6.3 [3.7 -8.2]	6.2 [3.0-7.7]	6.9 [4.8-8.9]	0.03
Lymphocyte, 10^3/uL, median, [IQR]	0.9 [0.7-1.4]	1.1 [0.8-1.5]	0.7 [0.6-1.0]	<0.01
Haemoglobin, g/L	12.4±2.3	12.6±1.9	11.9±2.8	0.22
Platelet, 10^3/uL, median, [IQR]	210 [153 - 290]	210 [159-289]	214 [152-338]	0.74
D-Dimer, mg/L, median, [IQR]	1.9 [1.1-3.2]	1.5 [1.1-2.45]	3.2 [2.8-3.5]	<0.01
CRP, mg/L, median, [IQR]	71 [30-179]	50 [18-124]	134 [63-234]	<0.01
Albumin, g/L	33±5.7	34±5.9	30±4.6	0.01
CK-MB, ng/mL, median, [IQR]	8.7 [3.5-21]	5.3 [2.4-11]	21 [15-37]	<0.01
hs-TnI, pg/mL, median, [IQR]	203 [104-800]	148 [48-295]	854 [209-1700]	<0.01

FBG, fasting blood glucose; eGFR, estimated glomerular filtration rate; CRP, C-reactive protein; CK-MB, creatine kinase myocardial band; hs-TnI, high sensitive troponin I.

Table 3. Factors that were found to be independently associated with in-hospital mortality in univariate and multivariate cox regression analysis model

Variables	Univariate OR (95% CI)	p	Multivariate* OR (95% CI)	p
Age	1.049 (1.016-1.083)	<0.01	1.049 (1.001-1.099)	0.04
CAD	4.490 (2.048-9.665)	<0.01	3.098 (1.206-7.957)	0.02
eGFR	0.980 (0.968-0.992)	<0.01	0.984 (0.969-1.000)	0.04
CRP	1.002 (1.000-1.004)	0.04	1.006 (1.002-1.010)	<0.01
D-Dimer	2.073 (1.428-3.011)	<0.01	1.567 (1.006-2.441)	0.04
Heart Failure	3.190 (1.433-7.101)	<0.01	-	-
SOFA score	1.539 (1.266 - 1.872)	<0.01	-	-
Lymphocyte	0.226 (0.07-0.722)	0.01	-	-

**The variables with a p-value of less than 0.1 in the univariate analysis were incorporated into the multivariate cox regression analysis by using Backward LR method. Abbreviations: CAD, coronary artery disease; eGFR, estimated glomerular filtration rate; SOFA, sequential organ failure assessment, CRP, C-reactive Protein.*

Other

OP-257

Relationship between mortality and troponin I levels in hospitalized patients with novel Coronavirus (COVID-19)

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Background and Aim: Coronaviruses are important pathogens of humans and animals that can cause diseases ranging from mildly to seriously and fatally respiratory, enteric, cardiovascular diseases. Recent studies emphasized that some of the patients had COVID 19 associated cardiac injury as an ejection fraction decline and troponin I elevation. In hospitalized patients, cardiac damage leading to cardiac death with a high troponin level were reported to be between 7.2-40%. The aim of this study was to evaluate the relationship between mortality and cardiac laboratory findings in patients who were diagnosed and hospitalized with COVID 19 infection caused by the novel coronavirus SARS-CoV-2.
Methods: A total of 105 patients who were hospitalized and diagnosed with COVID 19 infection between 20 March 2020 and 20 June 2020 were included in the study. Patients's medical history, clinical, laboratory, radiological, and treatment data were collected and analyzed.
Results: The baseline clinical features of the 2 groups (troponin I high and low group) are summarized in Table 1. There was shown a statistically significant differences between the 2 groups in Table 1 (all p<0.05). A statistically significant correlation was observed between the presence of age, saturation, CK-MB, PT, neutrophil count, lymphocyte count, NLR, hemoglobin, CRP, ferritin, D dimer, calcium, 25-hydroxy vitamin(OH) D3 (r and p values, respectively: 0.478, <0.001; -0.626, <0.001; 0.697, <0.001; 0.617, <0.001; 0.562, <0.001; -0.418, <0.001; 0.695, <0.001; -0.368, <0.001; 0.587, <0.001; 0.531, <0.001; 0.683, <0.001; -0.619, <0.001; -0.464, <0.001). We performed univariate and multivariate analyses to determine the independent factors associated with mortality. The troponin I, dyspnea, hypertention, hyperlipidemia, diabetes mellitus, and coronary artery disease were analyzed using the multivariate Cox regression model. In multivariate analyses, a significant association was noted between troponin I, dyspnea and the adjusted risk of mortality (odds ratio=1.002, 95% confidence interval=1.000-1.004; p=0.045; odds ratio=6.639, 95% confidence interval=1.039-42.445; p=0.046, respectively). In a receiver operating characteristic (ROC) curve analysis, troponin I value >7.8 pg/ml was identified as an effective cut-off point in mortality for patients with COVID 19 (area under curve = 0.832, 95% CI=0.654-1.009, p=0.001). A troponin I value of less than 7.8 pg/ml yielded a sensitivity of 78% and a specificityof 86% (Figure1). The Kaplan–Meier curve for mortality according to troponin I group (troponin I >7.8 pg/ml, troponin I ≤7.8 pg/ml) in the entire population of patients (p<0.001 by log-rank test).
Conclusions: Troponin value constituted an independent risk indicator for mortality when it was above the cut-off value of >7.8 pg/ml. This highlights the need to monitor more closely patients with troponin levels above this value. Aggressive treatment may be considered for patients at high risk of myocardial injury.

Table 1. Baseline and clinical characteristics of troponin I high and low group

Characteristics of patients	All patients [n-mean±SD or median(min-max)]	Troponin I high group(>7.8pg/ml) [n-mean±SD or median(min-max)]	Troponin I low group(<7.8pg/ml) [n-mean±SD or median(min-max)]	P values
Number of patients(n)	105	21(20%)	84(80%)	-
Male(n)	76	15(71.4%)	61(72.6%)	0.053
Female(n)	29	6(28.6%)	23(27.4%)	0.053
Age(years)	45(20-87)	60(26-87)	45(20-83)	0.005
Body mass index(kg/m2)	25(21-38)	27(21-32)	25(21-38)	0.008
Dyspnea	(+) 36 (-) 69	16(76.2%) 5(23.8%)	20(23.8%) 64(76.2)	<0.001
Saturation (%)	94(45-98)	85(45-96)	94(60-98)	<0.001
Hypertension	(+) 23 (-) 82	11(52.4%) 10(47.6%)	12(14.3%) 72(85.7%)	<0.001
Stay at the intensive care unit	(+) 15 (-) 90	10(47.6%) 11(52.4%)	5(6%) 79(94%)	<0.001
Mortality	(+) 9 (-) 96	7(33.3%) 14(66.7%)	2(2.4%) 82(97.6%)	<0.001
♣ Favipiravir	(+) 15 (-) 90	9(42.9%) 12(57.1%)	6(7.1%) 78(92.9%)	<0.001
♣ Plasma exchange	(+) 2 (-) 103	2(9.5%) 19(90.5%)	0 84(100%)	-
♣ ECMO	(+) 1 (-) 104	1(4.8%) 20(95.2%)	0 84(100%)	0.044
Blood urea nitrogen	30.09±21.66	(47.28±39.93)	(25.74±10.28)	<0.001
Creatinine	0.86(0-1)	0.98(0.63-3.74)	0.82(0.49-2)	0.005
Glomerular filtration rate	98(15-457)	88(15-124)	13.2(37-457)	0.004
CRP	47.5(0.01-319)	148(5.1-273)	13.2(0.01-319)	<0.001
Neutrophil count	4.1(1.4-83)	8(2.0-83)	3.56(1.4-19.7)	<0.001
Lymphocyte count	1.1(0.6-6.9)	0.81(0.1-6.9)	1.28(0.36-3.33)	0.007
Neutrophil-Lymphocyte ratio	3.09(0.89-31)	11.1(2.21-30)	13.2(0.89-31)	<0.001
Hemoglobin	13(8.5-41.7)	12(9-15.5)	13.2(8.5-41.7)	0.007
Lactate dehydrogenase	329.80±214.81	(498.71±362.77)	(287.07±129.25)	<0.001
Ferritin	354(3.61-3258)	727(48-2684)	291(3.6-3258)	0.008
D Dimer	583(0-8902)	2271(271-8802)	422(0-8902)	<0.001
Troponin(pg/ml)	2.6(0-1774.5)	15.4(8.6-1774.5)	1.9(0-7)	<0.001
CK-MB	15.03±20.00	(23.55±39.99)	(12.58±6.97)	0.046
PT	15.3±6.0	(18.1±10.6)	(14.3±2.0)	0.020
Calcium	8.5(7.3-10)	7.95(7.3-9.1)	8.6(7.7-10)	<0.001
25-hydroxy vitamin 25 (OH) D3	16.5(4.1-90)	8.15(4.1-47)	18(6-90)	<0.001

ALT: alanine aminotransferase, AST: aspartate aminotransferase, CRP: C- reactive protein, CK: Creatine kinase, CK MB: CreatineKinase MB, COPD: chronicobstructivepulmonarydisease, ECMO: Extracorporeal membrane oxygenation, ESR: Erythrocyte sedimentation rate, BMI: body mass index, DM:diabetes mellitus,HL: hyperlipidemia, HT:hypertension, INR: International Normalized Ratio, PT: Prothrombin time, PTT: partial thromboplastin time.

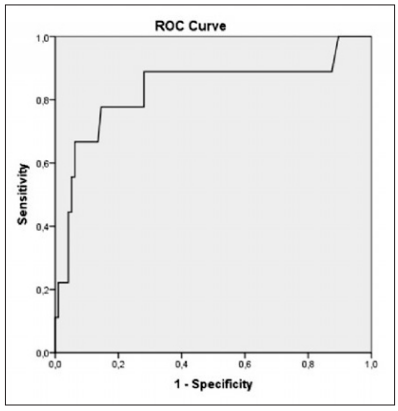

Figure 1. In a receiver operating characteristic (ROC) curve analysis, a troponin I >7.8 pg/ml was identified as an effective cut-off point in the COVID 19 patients (area under curve=0.832, 95% CI=0.654-1.009, p=0.001). A troponin I value of more than 7.8 pg/ml yielded a sensitivity of 78% and a specificity of 86%.

Other

OP-258

Clinical utility of high sensitive troponin-I measurement in COVID-19 infection: a predictor of major adverse events

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Background and Aim: It is important to classifying patients effectively to identify the highest-risk patients, who may require more intensive supervision and support in Covid-19 infection. The aim of this study was to investigate the relationship between in-hospital major adverse events (MAEs) and high-sensitive troponin I (hs-TnI) in hospitalized patients with Covid-19 infection.
Methods: A cohort of 123 patients with the diagnose of Covid-19, who admitted to Sakarya University Education and Research Hospital and hospitalized, were retrospectively analyzed. The demographic, clinical, admission laboratory findings, and treatment data were collected and analyzed. In-hospital MAEs, which were defined as acute pulmonary edema, need for invasive ventilation, and death, were determined as the primary outcome of the study. Risk factors of MAEs were analyzed using multivariable regression models.
Results: The primary outcome, MAEs, occurred in 56.2% in the elevated hs-TnI group and 3.3% in the normal hs-TnI group (p<.001). The rates of acute pulmonary edema (p=.001), the need for invasive ventilation (p<.001), and death (p<.001) were significantly higher in elevated hs-TnI group. Moreover, patients with elevated hs-TnI were more likely to require intensive care unit admission (p<.001), noninvasive oxygen support (p<.001), acute renal failure (p<.001), acute arrhythmia (p<.001) and treatment with inotropic agents (p<.001). The multivariate regression analysis indicated that hs-TnI independently predict MAEs in hospitalized patients with Covid-19 (p<.001). In the receiver operating characteristic (ROC) curve analysis, a cut-off hs-TnI value of 21.8 was determined to predict MAEs with 81.0% sensitivity and 79.0% specificity (ROC area:.856; 95% CI:.774 -.938; p<.001).
Conclusions: These results could provide early diagnosis and management of patients at risk, who should need additional monitoring and aggressive supportive care.

Table 1. Comparison of elevated hs-TnI and normal hs-TnI groups in terms of in-hospital outcomes

	Total (n=123)	Elevated hs-TnI (n=32)	Normal hs-TnI (n=91)	P-value
İntensive care unit, n (%)	36 (29.3)	29 (90.6)	7 (7.7)	<.001
Noninvasive oxygen support, n (%)	53 (43.1)	31 (96.9)	22 (24.2)	<.001
Invasive ventilation, n (%)	7 (5.7)	6 (18.8)	1 (1.1)	<.001
Acute pulmonary edema, n (%)	6 (4.9)	5 (15.6)	1 (1.1)	.001
Usage of inotropic agents, n (%)	11 (8.9)	8 (25.0)	3 (3.3)	<.001
Acute arrhythmia, n (%)	5 (4.1)	5 (15.6)	0 (0)	<.001
Death, n (%)	10 (8.1)	9 (28.1)	1 (1.1)	<.001
MAEs, n (%)	21 (17.1)	18 (56.2)	3 (3.3)	<.001

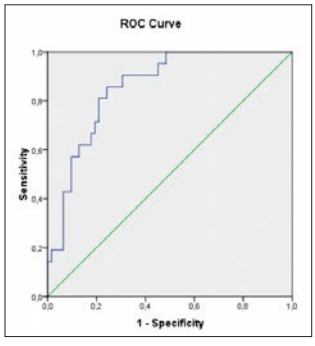

Figure 1. Receiver operator characteristic (ROC) curve analysis of high-sensitive troponin I (hs-TnI) for the prediction of major adverse events (MAEs) in hospitalized patients with Covid-19.

Other

OP-259

Is right ventricular strain pattern on ECG associated with mortality in patients with COVID-19?

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Background and Aim: In December 2019, a new coronavirus (SARS-CoV-2) is life-threatening in all countries of the world and causing serious morbidity and mortality. The aim of this study to delineate the prognostic value of right ventricular strain pattern on ECG in patients with COVID-19.
Methods: Hospitalized patients with COVID-19 without a cardiac history were included consecutively in the study from 1 May to 31 May, 2020. ECG was performed on hospital admission and was evaluated as blind. ECG evidence of RV strain pattern was defined as (1) presence of SIQ3T3; (2) presence of complete or incomplete RBBB; (3) T-wave inversions in the precordial leads (V1-V3). The main outcome measure was death during hospitalization. Right ventricular echocardiographic and electrocardiographic parameters were compared. Clinical characteristics, laboratory findings and outcomes data were obtained from electronic medical records.
Results: Hospitalized 107 patients with COVID-19 were included in this study. The patients were divided two main groups as non-survivor and survivor. The mean age of the patients with non-survivor group was 59.0±18.6, while the mean age of the patients survivor group was 50.2±10.7. ST depression (38% vs 13%) and RV strain pattern (45% vs 9%) were higher in patients with non-survivor group. Multivariate analyses revealed that ARDS (OR:22.25, 95%CI:6.54-74.84; p<0.001), presence of cardiac injury (OR:6.95, 95%CI:1.45-33.27; p=0.015), RV strain pattern (OR:14.41, 95%CI:2.84-73.07; p=0.001) and ST depression (OR:6.06, 95%CI:1.23-29.72; p=0.026) are independent predictors of mortality.
Conclusions: Right Ventricular Strain Pattern on ECG is an independent predictor of mortality in patients with COVID-19.

Table 1. Demographic and clinical characteristics of patients non-survivor and survivor

Characteristic	Non-surviver (n = 31)	Surviver (n = 76)	p
Age (years)	59.0 ± 18.6	50.2 ± 10.7	0.018
Male, n(%)	12 (38%)	40 (52%)	0.191
BMI (kg/m2)	28.1 ± 4.3	27.8 ± 5.3	0.689
HR	92.0±20.0	84.1±17.5	0.047
SBP	110.7±19.0	121.4±14.9	0.015
DBP	67.1±13.2	73.8±9.9	0.007
RR	25.6±8.7	22.2±3.0	0.041
Chronic medical illness			
HT, n(%)	9(29%)	28(36%)	0.441
DM, n(%)	7(22%)	11(14%)	0.309
HLD, n(%)	6(19%)	10(13%)	0.415
Smoker, n(%)	13(41%)	31(40%)	0.913
Laboratory findings on admission hospital			
Haemoglobin(g/dl)	12.0 ± 1.7	12.4 ± 2.1	0.338
WBC (10^3 /µl)	7(5-14)	5(4-10)	0.022
Creatinine (mg/dl)	0.8±0.1	0.8±0.1	0.600
Sodium (mmol/L)	137.4 ± 4.9	137.9 ± 4.5	0.592
Potassium (mmol/L)	4.1 ± 0.4	4.1 ± 0.5	0.949
Glucose (mg/dL)	125.7±35.5	121.7±36.0	0.601
CRP (mg/dL)	5.2(2.3-14.9)	2.4(0.4-7.8)	0.035
hsTroponin(pg/ml)(NR1<14pg/ml)	63(10-412)	10(10-16)	<0.001
D-dimer (ng/mL)	620(190-1050)	730(510-910)	0.371
sO2	91.2±6.3	95.3±3.9	0.002
pCO2	42.5±6.9	39.2±7.1	0.030
pH	7.4±0.0	7.4±0.0	0.301
Complications and clinical outcome			
Hospital length of stay, days	10(6-16)	6(3-15)	0.135
ICU, n(%)	27(87%)	17(22%)	<0.001
Cardiac injury, n(%)	21(67%)	20(27%)	<0.001
Acut renal injury, n(%)	8(25%)	17(22%)	0.703
ARDS, n(%)	20(64%)	8(10%)	<0.001
Discharged, n(%)	-	70(92%)	

Table 2. Comparison of right ventricular echocardiographic and electrocardiographic parameters in non-survivor and survivor groups

Variables	Non-survivor (n = 31)	Survivor (n = 76)	p
RV-FAC (%)	38.8±10.1	42.6±6.3	0.023
TAPSE (mm)	19.6±4.0	22.4±3.4	0.001
sPAP, mmHg	35.2±10.3	30.9±9.2	0.037
RV (mm)	35.5±6.6	32.3±4.9	0.019
RA (mm)	36.7±6.5	33.0 ± 5.0	0.007
TDI E', cm/s	10.5±2.7	11.1±4.0	0.448
TDI A', cm/s	16.8±5.0	15.5±5.0	0.243
TDI S', cm/s	13.8±3.5	15.0±2.8	0.079
PA, mm	20.2±2.1	21.1±2.3	0.080
RV-ECG-Strain, n(%)	14(45%)	7(9%)	<0.001
fQRS, n(%)	6(19%)	6(7%)	0.088
ST depression, n(%)	12(38%)	10(13%)	0.003

Table 3. Univariate and Multivariate Cox Regression Analysis on the Risk Factors Associated Mortality in Patients with COVID-19

Variable	Univariate			Multivariate		
	OR	95%CI	p	OR	95%CI	p
Age	1.048	1.015-1.082	**0.005**	1.039	0.972-1.111	0.262
Gender	1.759	0.751-4.122	0.194			
Hypertension	1.426	0.577-3.524	0.442			
Diabetes mellitus	0.580	0.202-1.670	0.313			
sO2	0.855	0.783-0.934	**0.001**	0.889	0.767-1.030	0.116
CRP	1.072	1.015-1.134	**0.013**	1.028	0.930-1.137	0.587
Cardiac injury	5.670	2.280-14.103	**<0.001**	6.955	1.454-33.275	**0.015**
ARDS	15.455	5.472-43.648	**<0.001**	22.254	6.541-74.842	**<0.001**
RV-FAC	0.940	0.890-0.993	**0.027**	0.938	0.856-1.028	0.171
TAPSE	0.821	0.729-0.926	**0.001**	0.955	0.776-1.176	0.664
RV-ECG-strain	8.118	2.838-23.223	**<0.001**	14.412	2.842-73.073	**0.001**
ST depression	4.168	1.561-11.131	**0.004**	6.061	1.236-29.720	**0.026**

Other

OP-260

Angiotensin 1-7 prevents diabetes induced left ventricular diastolic disfunction in rats

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Background and Aim: Renin-Angiotensin-Aldostrerone System (RAAS) plays critical role in pathophysiology of heart failure and therapies suppressing this pathways improves outcomes. Suppression of angiotensin II (Ang II) or its receptor activity has been shown to reverse remodeling in cardiomyopathic process including diabetic cardiomyopathy (DCM). Recently angiotensin 1-7 (Ang 1-7) and angiotensin 1-9, which are produced via ACE-2 enzyme pathway were shown as novel components of RAAS. Counter effects of Ang 1-7 opposing to Ang II caused increasing research focused on novel RAAS concept systemic and locally at tissue level. Hereby we investigated effects of Ang 1-7 on diabetic alterations on cardiac functions in a DCM model.
Methods: Experimental DCM model was instituted by application of a single dose of 50 mg/kg streptozocin as i.p. Rats were cared for 4 weeks to allow DCM development and 600 µg/kg Ang 1-7 was applied for 30 days to reveal effects of Ang 1-7. Tail-cuff method was utilized to measure systolic, diastolic and mean arterial blood pressures. Left ventricular functions were evaluated by pressure-volume loop studies.
Results: Experimental DCM model has resulted increase in systolic and mean arterial blood pressure, but diastolic blood pressure did not change significantly. Application of Ang 1-7 prevented these alterations due to diabetes, where in physiological conditions did not result hypotension. Diabetes decreased stroke work (SW) by decreasing stroke volume (SV) and cardiac output (CO). Main reason of this phenomena is of decreased end diastolic volume (EDV) in diabetic state. Moreover in diabetic state; pressure-volume area (PVA) and potential energy (PE) area are reduced and cardiac efficiency (CE) is decreased due to changes in myocardial contraction (+dP/dt) and relaxation (Tau) kinetics. By opposing or reducing diabetes related changes Ang 1-7 results normalization of these parameters to control levels.
Conclusions: Ang1-7 influence recovery of cardiac function by improving SW and contractility in DCMP model. Also, Ang1-7 preserves maintenance of ventriculo-arterial coupling by increasing ventricular compliance and decreasing arterial elastance. ACE-2 pathway may be considered as a potential therapeutic target, as our results suggests improvement in remodelling due to DCM by Ang 1-7.

Other

OP-262

Evaluation of Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio in patients with preserved ejection fraction before and after coronary artery bypass grafting

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Background and Aim: QT interval and T wave represent ventricular repolarization on surface electrocardiography (ECG). T-peak–T-end (Tp-e) interval, which shows the dispersion of repolarization, is defined as the interval between the peak and end of the T-wave. It is shown that prolonged Tp-e interval and Tp-e dispersion are related with ventricular arrhythmias and mortality in patients with coronary artery disease (CAD). However, as far as we have researched, there are limited studies on the assesment of Tp-e interval, Tp-e/QT ratio, and Tp-e/corrected QT interval (QTc) ratio in patients with CAD undergoing to coronary artery bypass grafting (CABG). The aim of this study is to evaluate Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio in patients with preserved ejection fraction (EF) before and after CABG.
Methods: 100 patients with stable CAD and preserved EF who underwent successful CABG were included in the study population. Ventricular repolarization related ECG markers that QT interval, QTc interval, Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio were recorded at before CABG and 1 month after CABG. ECG and echocardiography registries at before CABG and after CABG compared.
Results: Baseline characteristics of the study patients shown in Table 1. The EF values were similar at before and after CABG. QT interval and QTc interval values were statistically similar before and after CABG. Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio (for each p<0.001) were significantly different before and after CABG (Table 2).
Conclusions: In our study, we demonstrated that Tp-e interval, Tp-e/QT and Tp-e/QTc ratios were shortened in patients with CAD and preserved EF after successful CABG than before procedure. CAD causes myocardial ischemia. During myocardial ischemia, several metabolic and electrochemical changes occur in cardiac muscle affecting tissue oxygen levels, pH, intercellular and intracellular ion channel status, electrochemical gradients, and current. These changes have a complex influence on the duration of action potentials in the ischemic zone; thus, the Tp-e and QT intervals display changes that are modestly concordant. We hypothesized that these changes should reduced after successful revascularization. It is known that myocardial ischemia prolongs QT interval while re¬perfusion shortens it; however, there was no statistically significant relationship between QT intervals and reperfusion in our study. We observed that Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio reduced after successful CABG. This study demonstrated that after CABG shortening in Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio are associated with reperfusion success. Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio may serve as a prognostic predictor of adverse outcomes after successful CABG in stable CAD patients, and more studies should be carried to further evaluate its clinical value.

Table 1. Demographic, clinical and laboratory parameters of the patients

Variable	Before CABG n:100	After CABG n:100	p
Age (years)	59 ± 14.2	-	-
Gender (female), n (%)	48 (48)	-	-
Hypertension, n (%)	45 (45)	-	-
Diabetes mellitus, n (%)	31 (31)	-	-
Hyperlipidemia, n (%)	23 (23)	-	-
Current smoker, n (%)	49 (49)	-	-
Heart rate, pulse/minute	83.4 ± 11.9	79.6 ± 10.3	0.263
Systolic blood pressure, mmHg	133 ± 18	131 ± 19	0.742
Diastolic blood pressure, mmHg	82 ± 12	80 ± 11	0.633
Body mass index, kg/m2	28.2 ± 2.3	27.5 ± 1.7	0.165
Plasma glucose, mg/dl	127 ± 49.3	130 ± 46.1	0.531
Creatinin, mg/dl	0.75 ± 0.42	0.69 ± 0.23	0.138
Blood urea nitrogen, mg/dl	32.9 ± 16.6	29.5 ± 10.1	0.139
Total cholesterol, mg/dl	236 ± 58	217 ± 60	0.085
Low density lipoprotein, mg/dl	163 ± 41	151 ± 44	0.193
High density lipoprotein, mg/dl	39.3 ± 13.8	43.2 ± 8.6	0.134
Triglyceride, mg/dl	191 ± 98	180 ± 77	0.102
Troponin I, ng/l	7.1 ± 8.3	8.5 ± 6.8	0.798
Hemoglobin, g/dl	13.2 ± 1.8	12.8 ± 1.2	0.327
White blood cell, 10^3/mm^3	7.3 ± 1.9	7.5 ± 1.6	0.854
Platelet, 10^3/mm^3	290.4 ± 54.7	301 ± 64.1	0.362

Table 2. Electrocardiographic and ejection fraction parameters of the patients at before and after CABG

Variable	Before CABG n:100	After CABG n:100	p
Left ventricle ejection fraction, %	53.9 ± 5.3	55.1 ± 4.7	0.173
QT interval, ms	366.8 ± 27.1	362.7 ± 25.2	0.228
QTc interval, ms	401.6 ± 34.3	396.4 ± 31.9	0.140
Tp-e interval, ms	85.8 ± 13.4	78.1 ± 10.9	<0.001
Tp-e/QT ratio	0.22 ± 0.03	0.20 ± 0.04	<0.001
Tp-e/QTc ratio	0.21 ± 0.03	0.19 ± 0.04	<0.001

Other

OP-264

Arterial stiffness measured by cardio-ankle vascular index is greater in young women with polycystic ovarian syndrome

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Background and Aim: The association of metabolic abnormalities and polycystic ovarian syndrome (PCOS) has been documented, but few studies have focused on cardiovascular risk in these women. Arterial stiffness has increasingly been used as a surrogate marker for cardiovascular risk. The aim of study was to compare arterial stiffness by using cardio-ankle vascular index (CAVI) in PCOS women with controls, and to evaluate whether any clinical or laboratory variables had independent associations with it.
Methods: A group of 160 women, matched for age and body mass index were recruited. Diagnosis of PCOS was made according to Rotterdam criteria. Arterial stiffness using CAVI were evaluated in non-obese, young woman with and without PCOS. CAVI was measured using VaSera TM (VS-1500 system, Fukuda Denshi, Tokyo, Japan).The blood samples were analyzed in the central laboratory on the same day. Total tes-

tosterone, SHBG, DHEAS, TSH and prolactin concentrations were measured by electrochemiluminescent immunoassay with inter and intra-assay coefficient of variation (CV) of <5%. The serum levels of androstenedione and free testosterone were analyzed with radioactive immunoassay method.

Results: In the PCOS (n=80) group, 60 cases (75%) had findings of hyperandrogenism, 59 (73.8%) had ovulatory dysfunction, and 70 (87.5%) had ultrasonographic appearance of polycystic ovaries. Anti mullerian hormone, total testosterone, free androgen index, free testosterone, triglycerides, and LH/FSH ratio were found higher in PCOS women compared to controls (p<0.05). In contrast, sex hormone binding globulin levels were lower in women with PCOS (mean difference=9.53, p=0.007). Women with PCOS had significantly higher mean CAVI values when compared to subjects without PCOS (5.78±0.64 vs. 5.28±0.77, p<0.001). Androgen excess was found to be associated with increased arterial stiffness, independent of ovulatory dysfunction and polycystic ovaries.

Conclusions: Previous studies indicate PCOS can be associated with cardiometabolic abnormalities. Arterial stiffness has increasingly been used as a surrogate marker for cardiovascular risk. In this study, we found increased arterial stiffness using CAVI in a group of young women with PCOS compared to women without. Moreover, our results also suggest that androgen excess in young women is related with decreased vascular compliance and have the potential per se to increase the cardiovascular risk, which warrants further studies.

Other

OP-265

The predictors of in-hospital major adverse cardiac events in Takotsubo Syndrome

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Background and Aim: Takotsubo Syndrome (TS) also referred to as stress induced cardiomyopathy is a syndrome which mimics acute coronary syndrome in terms of its clinical features. The underlying mechanisms of TS are not known exactly. However many pathophysiological mechanisms are emphasized including multi-vessel epicardial spasm, coronary microvascular spasm, catecholamine induced myocardial stunning, inflammation and transient left ventricular outflow tract obstruction to identify TS. In this study we wanted to learn the predictors of in-hospital major adverse cardiac events (MACE) in patients with TS.

Methods: Retrospectively, 59 TS patients and 50 control grups matched for age, gender and comorbidity were included in the study. The predictor of in-hospital MACE (death, cardiogenic shock, sustained and non-sustained ventricular tachycardia, acute heart failure, thrombus, left ventricular outflow tract obstruction) was investigated in patients with TS.

Results: Univariate and Multivariate logistic regression analysis was performed to learn the predictors of in-hospital MACE in TS.In univariate logistic regression analysis WBC counts, Neutrophil counts,neutrophil lymphocyte ratio (NLR), high sensivity troponin I, and b-type natriuretic peptide (BNP) were significantly predictors of in-hospital MACE in TS (respectively; p=0.008, p=0.005, p=0.004, p=0.004 p=0.001). According to outcomes of multivariate logistic regression analysis BNP (OR: 1.003 95% CI:1.001-1.006; p=0.009) and NLR (OR:5.887 95% CI:1.033-33.558; p=0.046) were found to be independent predictors of in-hospital MACE in TS.

Conclusions: Both BNP and NLR can be independent predictors of in-hospital MACE and can help to recognize high risk patients in TS.

Table 1. Clinical characteristic data in Takotsubo Syndrome

DATA	TS group (n=59)	Control group (n=50)	P
Age	54±9.04	53±8.00	0.393
Gender (female) n (%)	48 (81.4)	37 (74)	0.356
BMI	26.72 ±3.08	26.33±4.27	0.577
Hypertension n (%)	28 (47.5)	16 (32)	0.101
Diabetes mellitus n (%)	14 (23.7)	7 (14)	0.199
CAD n (%)	25 (42.4)	13 (26)	0.074
Hyperlipidemia n (%)	25 (42.4)	14 (28)	0.119
Smoking n (%)	17 (28.8)	8 (16)	0.113
Clinical presentation			
Chest pain n (%)	37 (62.7)	---	
Dyspnea n (%)	11 (18.6)	---	
Tachycardia	6 (10.2)	---	
General Weakness	5 (8.5)	---	
ECG changes			
ST elevation n(%)	32 (54.2)		
Pathological Q wave n (%)	8 (13.6)		
T inversion n (%)	16 (27.1)		
Nonspecific changes n (%)	3 (5.1)		
Balloning pattern			
Apical n (%)	49 (83.1)		
Mid n (%)	6 (10.2)		
Basal n (%)	4 (6.8)		
Laboratory findings			
WBC (103/µl)	9.17±1.96	7.47±1.24	<0.001
Neutrophil (103/µl)	6.28±1.89	4.55±0.80	<0.001
Lymphocyte (103/ µl)	1.99±0.41	2.10±0.60	0.238
NLR	3.25±1.03	2.26±0.53	<0.001
hs-troponin I (ng/ml)	3 (1.8-4.5)	---	
BNP (pq/ml)	750 (500-1700)	35.5 (25-53.47)	<0.001

Table 2. Major adverse cardiac event in Takotsubo Syndrome

	TS group (n=59)
MACE n (%)	12 (20.3)
Death n (%)	4 (6.8)
Cardiogenic shock n (%)	6 (10.2)
VT-NSVT n (%)	7 (11.9)
Acute heart failure (%)	4 (6.8)
Trombus n (%)	2 (3.4)
Outflow tract obstraction	2 (3.4)

Table 3. The predictors of major advers cardiac events in TS

Variables	Univariate analysis			Multivariate analysis		
	OR	95% CI	P	OR	95 % CI	P
Age	1.035	0.960-1.116	0.365			
HT	0.745	0.207-2.687	0.653			
DM	1.850	0.461-7.418	0.385			
CAD	1.474	0.413-5.262	0.550			
HL	0.964	0.267-3.486	0.956			
Smoking	1.308	0.336-5.094	0.699			
WBC	1.667	1.140-2.438	0.008	6.042	0.490-74.451	0.160
Neutrophil	1.732	1.178-2.547	0.005	0.126	0.007-2.378	0.167
Lymphocyte	0.568	0.119-2.721	0.479			
NLR	3.062	1.416-6.620	0.004	5.887	1.033-33.558	0.046
Troponin I	2.011	1.255-3.223	0.004	1.470	0.834-2.591	0.183
BNP	1.003	1.001-1.004	0.001	1.003	1.001-1.006	0.009

Other

OP-266

The effect of sacubitril valsartan treatment in the early period of experimental autoimmune myocarditis model on mortality, morbidity, myocardial fibrosis parameters; animal study

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Background and Aim: The aim of this study is to see the effectiveness of early sacubitril-valsartan treatment in heart failure caused by myocarditis which is a common reason of non-ischemic dilated cardiomyopathy in young people by creating an experimental autoimmune myocarditis model.

Methods: In the study, three groups were created by using a total of 18 rats. There were six wistar rats in each group and determined as EAM, EAM + Treatment and Control. The EAM model was created by giving an equal amount of porcine cardiac myosin and Complete Freund's Adjuvant (CFA) to foot pad. No treatment was given to the EAM group. EAM + Treatment group received 20 mg/kg of sacubitril-valsartan twice daily from day 21 to day 42. No treatment was applied to the Control group. Histopathological, biochemical and PCR analysis were performed on the heart tissues taken after sacrification in 42nd day of study.

Results: No significant difference was observed between EAM and EAM + Treatment group in congestion, fibrosis and cellular changes (p>0.05). No significant difference was observed between EAM and EAM + Treatment group at TNF α, TGF β1 levels (p>0.05). At IL 6 levels, there was no significant difference between the three groups (p>0.05). In pro BNP levels, no significant difference was observed between EAM and EAM + Treatment group (p=0.052). In the results of analysis other than IL 6, a significant difference was observed in both EAM and the EAM + Treatment group compared with the Control group (p<0.05).

Conclusions: As a result of the examination analysis between the treatment group and the patient group, no significant difference between fibrosis, cytokine, BNP values was observed in the treatment group compaired with patient group although the values were lower in the treatment group. In the experimental models to be created in the future, it is thought that significant results can be obtained by giving high doses of drugs, using more animals or starting treatment earlier.

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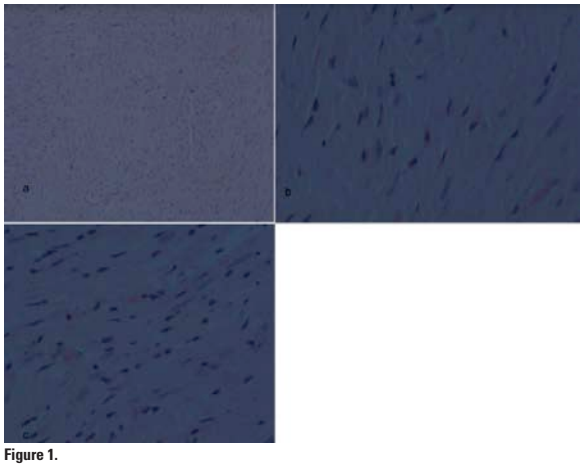

Figure 1.

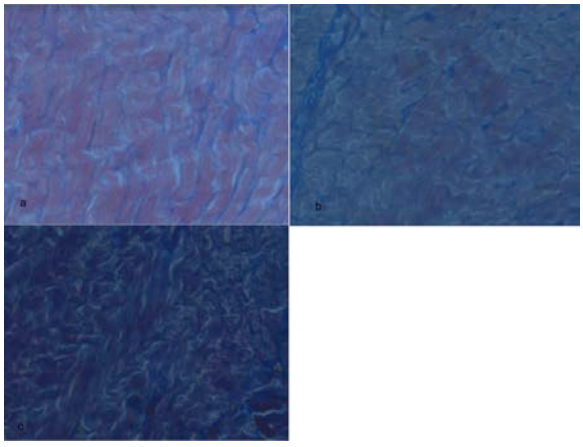

Figure 2.

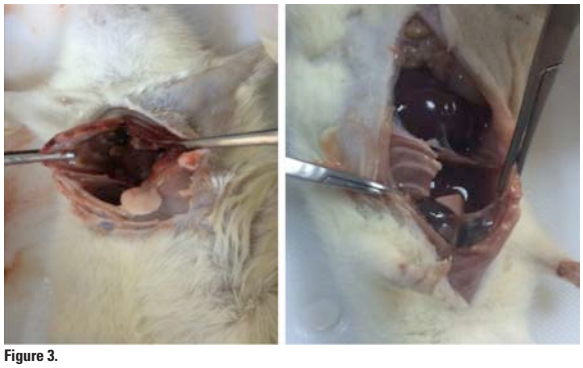

Figure 3.

Other

OP-267

The relation between arterial stiffness and optic neuropathy in patient with nonarteritic anterior ischemic optic neuropathy

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Background and Aim: Nonarteritic anterior ischemic optic neuropathy (NAION) is the most common form of ischemic optic neuropathy and the second most common optic neuropathy in western union. The exact mechanism of NAION has not been clearly understood yet. The hypoperfusion and ischemia are well-known underlying mecanısm of patients. Pervious studies demostrated that the circulatuar insufiency of the optic nerve head leading to hypoperfusion and ischemia. Increasing arterial stiffness is associated with hypoperfusion and ischemia of tissues such as brain and miyocard. The aim of this study was to determine the relationship between arterial stiffness and optic neuropathy using the central pulse-wave analysis method.
Methods: This prospective, single center study included 76 participants, comprising 34 patients diagnosed with nonarteritic ischemic optic neuropathy (Group 1) in hospital of Ondokuz Mayıs Universty and a control group of 42 healthy subjects (Group 2). The Mobil-O-Graph 24h PWA Monitor (I.E.M. GmbH, Stolberg, Germany) is a validated monitor for 24-h blood pressure monitoring and this device was used to determine the arterial stiffness and cardiovascular hemodynamic parameters. Augmentation index and pulse wave velocity (PWV) were obtained with the device.
Results: No statistically significant difference was determined between Group 1 and Group 2 in respect of gender and blood pressure (p>0.05). Group 1 was older than Group 2 p<0.05*. Pulse wave velocity was significantly higher in terms of arterial stiffness in Group 1 compared to the control group (8.8±1.6 vsn 6.1±0.8, p<0.001). No statistical variation was detected in terms of the augmentation index (24.6±12.5 vs 19.6±9.8, p=0.391). The Multivariate analysis indicated that Increased PWV was discerned as risk factors for NAION with odds ratios of 1.44 (95% confidence interval [CI], 1.6–10.7) and Increased PWV is related with 1.4 fold higher risc of optic neuropathy in patients with NAION.
Conclusions: Our results demostrated that arterial stiffness is associated with optic neuropathy in patients with NAION and this result that may help to understand of pathophysiology of NAION. The relationship was examined via oscillometric method, which is simple to perform and has been recommended in guidelines for the determination of cardiovascular event risk.

Table 1. Distribution of demographic descriptive characteristics,arterial stiffness and central hemodynamic parameters between NAION and control groups and Multivariate analysis

	NAION (n=34) mean±SD (min-max	Control group (n=42) mean±SD (min-max.)	P value
Age (year), mean±SD (min-max)	60,1±8.9 (43-75)	49,619±9,7 (39-68)	<0.05*
Gender(female)%	n:22 (64,7)	n:25 (59,5)	0.822*
Systolic BP (mmHg)	122.7±13.2 (89-149)	120.5±10.4 (100-142)	0.290*
Diastolic BP (mmHg)	81.0±11.4 (44-98)	78.0±9.7(59-94)	0.779*
BMI (kg/m2), mean±SD (min-max)	27,5±2.2 (23.0-33.0)	26,6±3.1 (19.9-33.1)	0.498*
Cigarette, n (%)	7 (20)	9 (21,4)	0.320#
Hypertension, n (%)	10 (29.4)	13(30,9)	0.280#
Diabetes Mellitus, n (%)	4(12,5)	5 (11,9)	0.361#
Hyperlipidemia, n (%)	10(31.1)	11(26.2)	0,960#
Augmentation Index (Alx)	24,6±12.5	19,6±9,8	0,391**
Pulse Wave Velocity (PWV)	8.8±1.6 (6.2-14)	6,1±0.8 (4.4-8.3)	0.001**
Multivariate analysis	OR	Lower CI/Upper CI	
Age (year), mean±SD (min-max)	0,095	0,955/1,266	0,185
Augmentation Index (Alx)	0,008	0,929/1,093	0,850
Pulse Wave Velocity (PWV)	1,440	1,662/10,715	0,002

**Student's T Test; **Mann-Whitney U Test, #Ki-Kare BP: Blood Pressure CI:Confidence Interval OR:Odds Ratio.*

Other

OP-268

Lens opacity classification for predicting atrial fibrillation duration

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Background and Aim: The incidence of atrial fibrillation (AF) increases with age, diabetes (DM), hypertension (HT), heart failure (HF), and cortical cataracts.The levels of sodium (Na) and glucose in the aqueous humor are closely correlated with those in plasma. Cortical cataract formation is largely due to changes in osmolarity.AF and cortical cataracts are both affected by changes in osmolarity. As the duration of AF increases, the frequency of complications increases. This study was performed to investigate the relationships between plasma osmolarity, cortical cataract stage, and AF duration.
Methods: The study population consisted of 200 heart failure (HF) patients without AF (non-AF control group) and 200 HF patients with AF (mean age in both groups, 70 years). AF duration and plasma osmolarity were recorded. The degree of cortical cataracts according to the lens opacity classification system (LOCS) was determined using biomicroscopy, and ejection fraction and left atrium volume index (LAVi; ml/m^2) were measured using echocardiography. The cases were divided into two separate groups in terms of AF duration according to the weighted means method (25^{th} percentile): ≤2.8 years (n=52, 26%) and >2.8 years (n=148, 74%).
Results: DM incidence was higher, HT duration was longer, and glucose and Na levels were higher in the AF group than in the non-AF control group (p<0.05). Significantly higher osmolarity [290.9 (286.6–296.0) vs. 283.5 (279.8–290.1) mOsm, respectively] and LAVi values [31.3 (26.5–34.3) vs. 2 1.5 (18.6–25.6) ml/m^2, respectively] were observed in the AF group compared to the non-AF control group (both, p<0.001). A greater proportion of AF vs. non-AF patients at each LOCS stage was also recorded [stage 1, 84 (42.0%) vs. 41 (20.5%); stage 2, 34 (17.0%) vs. 17 (8.5%); stage 3, 15 (7.5%) vs. 7 (3.5%); stage 4, 16 (8.0%) vs. 3 (1.5%), respectively] (all, p<0.001). Higher osmolarity was observed in patients with AF duration >2.8 years (n=148) compared to those with AF duration ≤2.8 years (n=52) [292.2 (287.3–296.9) vs. 289.4 (283.5–293.2) mOsm, respectively] (p<0.001). Significantly higher LAVi values [32.5 (30.6–35.6) vs. 23.6 (19.8–25.6) ml/m^2, respectively; p<0.001] were observed in patients with AF duration >2.8 years compared to those with AF duration ≤2.8 years. More patients with AF duration >2.8 years were classified as LOCS stage 1 [132 (89.2%) vs. 17 (32.7%), respectively; p<0.001], and these patients were also classified at higher LOCS stages [(1–2) vs. 0 (0–1), respectively; p<0.001]. In diagnostic performance tests, the threshold and area under the curve (AUC) values of parameters used to

predict AF duration >2.8 years were as follows: osmolarity >287.5 mOsm [AUC=0.711 (0.625–0.785)]; LAVi >28.45 ml/m² [AUC=0.958 (0.931–0.985)]; and LOCS stage ≥1 [AUC=0.854 (0.800–0.909)].
Conclusions: LOCS stage ≥1 exhibited good sensitivity [89.2% (82.8%–93.5%)] for predicting AF duration >2.8 years.

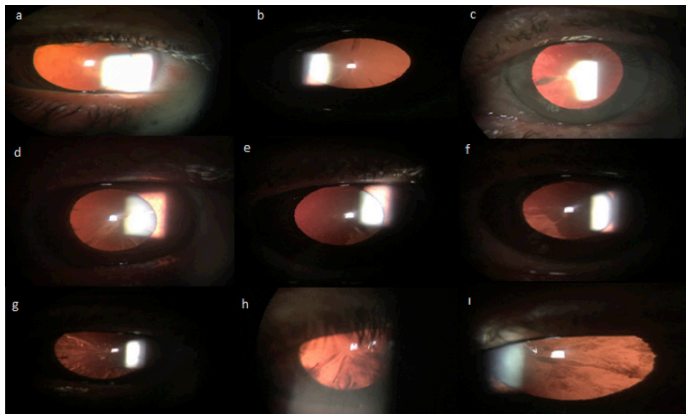

Figure 1. The Lens Opacity Classification System II (LOCS II). Classification according to locsII; a:locs 0 - b,c,d,e:locs1-f,g:locs 2 -h:locs 3-ı: locs 4.

Table 1. Demographical, clinical characteristics and laboratory measurements regarding for groups

	NonAF (n=200)	AF (n=200)	p-value
Age (years)	70 (68-72)	70 (69-71)	0.455†
Female	100 (50.0%)	100 (50.0%)	-
SBP (mmHg)	124 (112-130)	125 (115-135)	0.107†
DBP (mmHg)	71 (65-78)	72 (65-82)	0.357†
BMI (kg/m2)	26.1 (25.6-28.9)	26.5 (25.3-28.1)	0.054†
DM n(%)	65 (32.5%)	67 (33.5%)	0.832‡
Duration of DM(year)	9 (8-12)	11.5 (9-20)	<0.001†
Insulin usage n(%)	28 (43.1%)	32 (47.8%)	0.589‡
HT n(%)	139 (69.5%)	139 (69.5%)	-
Duration of HT(year)	8 (6-12)	11 (9-15)	<0.001†
HFn(%)	60 (30.0%)	66 (33.0%)	0.518‡
Duration of HF(year)	8 (6-10)	8 (6-12.5)	0.075†
EF (%)	57 (43.5-61)	55 (47-58)	0.012†
Osmolarity (mOsm/l)	283.5 (279.8-290.1)	290.9 (286.6-296.0)	<0.001†
Lavi (ml/m2)	21.5 (18.6-25.6)	31.3 (26.5-34.3)	<0.001†
Locs stage			<0.001†
0	132 (66.0%)	51 (25.5%)	
1	41 (20.5%)	84 (42.0%)	
2	17 (8.5%)	34 (17.0%)	
3	7 (3.5%)	15 (7.5%)	
4	3 (1.5%)	16 (8.0%)	
Locs positiveness n(%)	68 (34.0%)	149 (74.5%)	<0.001‡
CHADVASC score			0.167†
1	20 (10.0%)	0 (0.0%)	
2	54 (27.0%)	56 (28.0%)	
3	66 (33.0%)	83 (41.5%)	
4	28 (14.0%)	40 (20.0%)	
5	21 (10.5%)	14 (7.0%)	
6	11 (5.5%)	7 (3.5%)	

While, the descriptive statistics for continuous variables were shown as median (25th-75th) percentiles, otherwise number of cases and (%) were used for categorical data, † Mann Whitney U test, ‡ Pearson's Chi-square test.

Table 2. The results of ROC analyses

	Osmolarity(mOsm/l)	Lavi(ml/m2)	Locs stage
All cases			
AUC	0,711	0.958	0.854
95% CI	0,625-0,785	0.931-0.985	0.800-0.909
p-value	0,007	<0.001	<0.001
The best cut-off point	>287,5	>28.45	≥1
Sensitivity, % (95% CI)	50,2(45,2-55,8)	91.9 (86.0-95.5)	89.2 (82.8-93.5)
Specificity, % (95% CI)	84.6 (71.4-92.7)	90.4 (78.2-96.4)	67.3 (52.8-79.3)
PPV, % (95% CI)	78,4(64,5-83,4)	96.5 (91.5-98.7)	88.6 (82.1-93.0)
NPV, % (95% CI)	52,2(43,5-58,7)	79.7 (66.8-88.6)	68.6 (54.0-80.5)

Table 3. The results of multiple logistic regression analyses

All patients with AF	OR	95% CI for OR Lower	95% CI for OR Upper	Wald	p-value
Male factor	3.216	1.080	9,575	4.405	0.036
DM	2.954	0.577	15.123	1.690	0.194
HT stage	1.413	0.561	3.560	0.537	0.464
HF	0.600	0.125	2.877	0.408	0.523
Osmolarity >287,5mOsm/L	2,874	0,960	3,559	4.632	0.031
Lavi >28.45 ml/m2	87,819	29,574	260,772	64,950	<0.001
Locs stage ≥1	2.788	0.835	9.310	2.779	0.046

OR: Odds ratio, CI: Confidence interval.

Other

OP-269

Evaluation of ventricular repolarization parameters in patients admitted to emergency department with electrical injury

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Background and Aim: Prolongation of the peak-to-end interval of the T wave (Tp-e) has been reported to be associated with ventricular arrhythmias. Novel electrocardiogram (ECG) parameters of ventricular repolarization have called considerable attention recently. The aim of this study was to investigate ventricular repolarization in patients with electrical injury (EI) using the Tp-e, corrected Tp-e (Tp-ec) interval, and Tp-e/QT, Tp-e/QTc, Tp-ec/QT, and Tp-ec/QTc ratios.
Methods: A total of 36 patients diagnosed with electrical injury and 35 age- and sex-matched otherwise healthy controls were enrolled. Admission ECGs of the EI patients were compared with those of the healthy controls. QT and QTc intervals were measured and Tp-e interval and Tp-ec interval which was calculated by using the Bazett's formula (Tp-e/√RR) to increase its specificity, Tp-e/QT, Tp-ec/QT, Tp-e/QTc, Tp-ec/QTc ratios were then calculated from 12-lead surface ECG.
Results: QT, Tp-e, Tp-e/QT, Tp-e/QTc, Tp-ec/QT and Tp-ec/QTc were not significantly different between the control group and the EI group (p>0.05). However the mean QTc interval was significantly higher in the EI group compared to the control group (412.81±25.46 vs 396.31±26.47 msn; p=0.009). Furthermore, Tp-ec and Tp-ec/QT of the EI group with elevated troponin levels significantly differed from those of the EI patients with normal troponin levels (p=0.033, p=0.016 respectively).
Conclusions: This retrospective study showed that patients with EI tend to have a prolonged QTc interval. Additionally, Tp-ec and Tp-ec/QT, which repoetedly indicate the tendency to ventricular arrhythmias, were significantly higher in the EI patients with elevated troponin I levels than the EI patients with normal troponin levels, suggesting that patients with myocardial injury may be prone to ventricular arrhythmias.

Table 1. Comparison of general demographic, clinical, biochemical data between subjects with and without electrical injury

Variable	EI patients (n=36)	Healthy Control Group (n=35)	P value
Age (Years)	28 (18-64)	29 (20-61)	0.342
Sex (male)	24 (66.6%)	23 (65.7%)	0.434
HT	1 (2.8%)	1 (2.9%)	0.511
Smoking	11 (30.6%)	10 (28.6%)	0.531
DM	1 (2.8%)	0	0.507
Na (mEq/L)	138.3±2.4	138.5±1.4	0.616
K (mEq/L)	4.0 (3.6-5.3)	4.1 (3.6-4.6)	0.125
Mg (mg/dL)	2.03±0.26	2.04±0.18	0.930
Ca (mg/dL)	9.08±0.45	9.1±0.21	0.690
Hemoglobin (g/ dL)	14.75±2.22	14.9±1.3	0.796
WBC (/µl)	9.3(5.5-33.5)	7.4(4.6-11.0)	<0.05
Heart Rate (/min)	82.47±15.27	75.46±11.39	<0.05(0.032)
QT	356.42±30.72	352.23±24.44	0.527
QTc	412.81±25.46	396.31±26.47	<0.05 (0.009)
Tp-e	77.99±12.69	76.01±10.83	0.481
Tp-ec	91.05±16.80	84.91±11.85	0.079
Tp-e/QT	0.220±0.041	0.215±0.031	0.623
Tp-ec/QT	0.258±0.062	0.240±0.036	0.138
Tp-e/QTc	0.190±0.034	0.191±0.031	0.816

Table 2. Comparison of general demographic, clinical, biochemical data between electrical injury patients with and without troponin I elevation

Variable	Elevated Troponin I (n=10)	Normal Troponin I (n=26)	P value
Age (Years)	36.5	25	0.041
Sex (male)	9 (90%)	15 (57.6%)	0.069
HT	1(10%)	0	0.484
Smoking	4 (40%)	7 (26.9%)	0.353
DM	1 (%10)	0	0.722
Na (mg/dL)	138	138	0.794
K (mg/dL)	4	3.95	0.958
Mg (mg/dL)	1.93	2.07	0.109
Ca (mg/dL)	8.8	9.2	0.087
Hemoglobin (g/dL)	15.1	14.6	0.664
WBC (/μl)	12.565	7.345	0.001
Heart Rate (/min)	95.5	77	0.134
QT (msn)	355.5	359	0.241
QTc (msn)	418.5	415.5	0.337
Tp-e (msn)	83.53	78.45	0.126
Tp-ec (msn)	97	87.25	0.031
Tp-e/QT	0.231	0.215	0.087
Tp-ec/QT	0.281	0.244	0.015
Tp-e/QTc	0.196	0.193	0.337

Other

OP-270

The impact of the pandemic on the acute coronary syndrome profile: A retrospective comparative analysis

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Background and Aim: The coronavirus outbreak of 2019 (COVID-19) was announced as a pandemic by the World Health Organization (WHO) on March 11, 2020. Similar to many other countries, a national emergency was declared in our country. In order to reserve the healthcare capacity for the COVID-19 patients by preventing hospitals from overloading, the routine of healthcare systems had to be remodeled. In many centers worldwide, the number of ACS patients applying to emergency services and being followed in coronary intensive care units were reported to be reduced following the COVID-19 pandemic, along with increased time before treatment and increased in-hospital mortality rate. These aspects have not been thoroughly evaluated in our country so far.

Methods: In this retrospective study, the data of the ACS patients who applied to our clinic before (March-April 2019) and during (March-April 2020) the COVID-19 pandemic were evaluated in regards to the type of application to the clinic, symptom duration, door-to-balloon times, treatment approaches, length of hospitalization, in-hospital mortality and complication rates. The data were obtained from the patient files and the hospital's database.

Results: The number of ACS patients that applied to our clinic, which operated as a 'COVID-free' tertiary referral center, before (n=98) and during (n=98) the pandemic was the same. In these two periods, NSTEMI diagnosis was similar (n=53, 54% vs. n=52, 53%), however, a decrease in the number of patients diagnosed with USAP (n=11, 11% vs. n=5, 5%), and an increase in STEMI patients (n=34, 34% vs. n=41, 42%) was remarkable. We observed an increase in the interval between the onset of symptoms and hospital admission during the pandemic (median 88.3 hrs vs. 106.5 hrs, p=0.02). In the patients treated with PCI revascularization, there was no significant change in the door-to-balloon time (median 64 min vs. 76 min, p=0.96). While there was a numerical increase in the complication rate during PCI, this increase was not statistically significant (n=6, %6.1 vs. n=11, %11.2, p=0,20). In-hospital mortality was %1.0 (n=1) before the pandemic, and increased to 3.1% (n=3) during the pandemic (p=0.14).

Conclusions: During the COVID-19 pandemic, there was an increase in the interval between the onset of symptoms and hospital admission among ACS patients, which may either be attributed to patient or transfer issues. On the other hand, door-to-balloon times were similar. While there was an increase in patients with STEMI, there was a decrease in patients with USAP. Unlike the observations worldwide, the total number of ACS patients remained stable during the pandemic. We attributed this to the fact that our clinic operated as a 'COVID-free' tertiary referral center, and there was an increase in the number of patients referred from other clinics in this period. There were no significant changes in the complication and mortality rates.

Table 1. Characteristics of patients

	Before COVID-19		During COVID-19		*p*-value
	n	%	n	%	
ACS patients	98	100	98	100	
STEMI	34	34.7	41	41.8	
NSTEMI	53	54.1	52	53.1	
USAP	11	11.2	5	5.1	
Complications	6	6.1	11	11.2	0.20
Mortality	1	1.0	3	3.1	0.14
Symptom duration (median)	88.3 hours		106.5 hours		0.02
Door-to-balloon time (median)	64 minutes		76 minutes		0.96

ACS: acute coronary syndrome, STEMI: ST-elevated myocardial infarction, NSTEMI: non-ST-elevated myocardial infarction, USAP: unstable angina pectoris

Other

OP-272

Non-cardiac diseases accompanying to coronary intensive care unit follow-up processes that result in death

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Background and Aim: We conducted a retrospective analysis of mortality data in the coronary intensive care unit (CICU) of a tertiary referral hospital. Currently, there are limited data on accompanying non-cardiac disorders that may not directly cause to death but contribute to some aspects of the mortality process in CICU patients. This study aimed to define and to better understand comorbid conditions encountered during the management of CICU patients, who could not survive, in the hope of improving the standard of care of patients admitted to these units.

Methods: The patients' data who had died during the CICU follow-up period, were screened in a tertiary care center between January 2017 and January 2020. Patients who were under the age of 18 or had died within 24 hours after admission were excluded from the survey. Baseline clinical characteristics, laboratory parameters and concomitant non-cardiac disorders, which possibly worsened the health of the patients and had need to be treated urgently during the follow-up period were recorded.

Results: The study included 91 patients with a mean age of 75 [14] and 48.4% being male. The average length of hospital stay for non-survivors was 7 days [IQR- 9 days; range 2 to 42 days]. The baseline clinical characteristics are summarized in Table 1 and admission diagnoses of the study population are presented in Figure 1. Before the point of death all of the 91 patients, 47 (51.6%) had experienced cardiac arrest before, 45 (49.5%) had required acute hemodialysis, 65 (74%) were on mechanical ventilation, 71 (78%) were receiving inotropic or pressor therapy, 68 (74.7%) were under antibiotherapy, 18 (19.8%) had required defibrillation for atrial or ventricular arrhythmias, during the CICU follow-up period. In terms of laboratory evaluation at the time of death in CICU, 39 (42.9%) patients had anemia, 25 (25.5%) patients had thrombocytopenia and 55 (60.4%) patients had leukocytosis. Hyponatremia (36.3%) and hyperkalemia (36.3%) were common encountered electrolyte disturbances. In parallel with the high percentage of antibiotic use, 37(40.6%) patients had culture growth in blood, urine or sputum. Study patients had decreased pH and elevated creatinine and C reactive protein levels (Table 2).

Conclusions: The main goal of this study was not the assessment of mortality, but the prevalence of non cardiac disorders that commonly complicate the follow up course of the non-survived patients in CICU. According to our results, in the CICU, the three primary non-cardiac disease that need to be managed were respiratory insufficiency, renal failure and infectious diseases. This study confirms that in cardiovascular diseases, many cases are complicated by multiple organ failure, Therefore, medical staffs who work at CICU are required to have the ability to practice systemic intensive care and to be able to successfully manage mechanical ventilation, renal replacement therapy, and infection control.

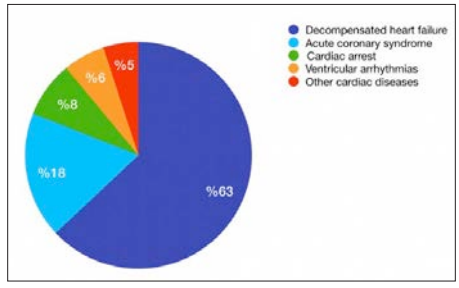

Figure 1. The percentage of admitting diagnoses of the study population.

Table 1. Baseline clinical characteristics before admission to coronary intensive care unit

Variables	n (%)
Hypertension	53 (58.2)
Diabetes Mellitus	44 (48,4)
Heart failure	
-Systolic heart failure	59 (64.8)
-Diastolic heart failure	11 (12.1)
-Right heart failure	4 (4.4)
Arrhythmias	
-Atrial arrhythmias	27 (29.7)
-Ventricular arrhythmias	3 (3.3)
-Bradyarrhythmias	2 (2.2)
Chronic obstructive pulmonary disease	13 (14.3)
Chronic renal disease	40 (44)
Dialysis	11 (12.1)
Coronary artery disease	53 (58.2)
Cerebrovascular accident	7 (7.7)
Pacemaker/intra cardiac defibrillator	17 (18.7)
Moderate or severe heart valve disease	45 (49.5)
Prosthetic heart valve	12 (13.2)
Pulmonary hypertension	39 (42.9)

Table 2. Laboratory results on the day of death. Variables with normal distribution are presented as mean±standard deviation, variables with non-normal distribution are presented as median [interquartile range]

Hemoglobin, g/dL	10.9 [2.6]
Leukocytes, x10^3/μL	12.35 [9.97]
Thrombocytes, x10^3/μL	214.18 ± 104.47
Creatinine, mg/dL	2.50 [1.90]
Sodium, mmol/L	137 [12]
Potassium, mmol/L	4.7 [1.50]
C-reactive protein, mg/L	127.68 ± 77.78
pH	7.33 ± 0.13
HCO3, mmol/L	19.53 ± 6.84

Other

OP-273

The Relationship between femoral T score and herat rate recovery in postmenopausal women

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Background and Aim: Post-exercise heart rate recovery index (HRRI) has been proposed as a measure of cardiac autonomic function and a delayed heart rate recovery in the immediate postexercise period has been linked to adverse cardiovascular prognosis. This study aimed to evaluate the relationship between bone mineral density using the femoral T score and heart rate recovery index parameters in postmenopausal women.

Methods: Between May-October 2019, A total of 135 postmenopausal women included in the study. After the routine history, physical examination and blood tests, patients underwent a routine electrocardiogram, transthoracic echocardiogram, and treadmill exercise test, and patients underwent femoral bone mineral densitometry. According to the results of femoral bone mineral densitometry, 45 patients with T score ≥-1 were named as group 1, 45 patients with T score up to -1 / -2.5 were named as group 2 and 45 patients with T score lower than -2.5 were named as group three. Heart rate recovery indexes were compared between three groups and their relationship with the femoral T score was evaluated.

Results: Heart rate recovery index values of group 1 were respectively 32.11±5.7, 46.91±8.64, and 64.59±11.32 beats/minute. Heart rate recovery index values of group 2 were respectively 23.99±7.17, 36.92±9.19, and 49.49±11.32 beats/minute. Heart rate recovery index values of group 3 were respectively 17.31±3.41, 27.98±4.23, and 35.58±3.55 beats/minute. There were statistically significant differences between the three groups in terms of heart recovery index values at the first-minute recovery phase of the treadmill exercise test (p value<0.01). There were statistically significant differences between the three groups in terms of heart recovery index values at the second-minute recovery phase of the treadmill exercise test (p value <0.01). There were statistically significant differences between the three groups in terms of heart recovery index values at the third-minute recovery phase of the treadmill exercise test (p value <0.01). There were statistically significant positive correlations between T score and heart rate recovery index value at first-minute recovery phases of treadmill exercise test in groups 1, 2, and 3 (respectively, p<0.01, p<0.01, and p<0.01).

Conclusions: In postmenopausal patients, the lower t score was associated with more delayed heart rate recovery after excersice and thus higher risk of adverse cardiovascular prognosis. There was a statistically significant relationship between Femoral T score and heart rate recovery index values.

Other

OP-274

A novel index for contrast-induced nephropathy prediction in patients with elective percutaneous coronary intervention

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Background and Aim: To investigate the predictive value of contrast volume to left ventricular ejection fraction ratio (CV / LV EF) for contrast-induced nephropathy (CIN) development in patients with elective percutaneous coronary intervention (PCI).

Methods: 882 patients who underwent elective PCI in our clinic were evaluated prospectively in terms of CIN. CV / LV EF ratio = the amount of contrast used in the procedure / LV EF. The ACEF score was calculated by using the following formula: age/LV EF + 1 (if serum creatinine >2.0 mg/dl). GFR was calculated with Cockcroft-Gault formula; [(140-age) x body weight (kg)] / [72 x serum creatinine] (if women x 0.85). The definition of CIN includes absolute (≥0.5 mg/dl) or relative increase (≥25%) in serum creatinine at 48-72 h after exposure to a contrast agent compared to baseline serum creatinine values. If serum creatinine values were ≥1.5 mg/dl, intraveneous hydration was performed with 0.9% sodium chloride (1 ml/kg/h) before the procedure.

Results: CIN was detected in 13.5% (119 patients) of 882 patients. The patients with and without CIN are compared and showed that; age, LV EF, GFR, Contrast amount used, ACEF score and CV / LV EF ratio were significantly different between the two groups (Table 1). In the multivariate linear regression analysis for CIN prediction; age, LV EF, contrast amount used, GFR value, ACEF score and CV / LV EF ratio were evaluated. In multivariate lineer regression analysis, age (Beta: 0.205, t: 3.408, p=0.001) and CV / LV EF ratio (Beta: 0.379, t: 2.769, p=0.006) were found to be significant predictors for the development of CIN (table 2). The risk of CIN was calculated as: 0.725 + 0.205 x age + 0.379 x CV / LV EF ratio. In ROC analysis with CV / LV EF ratio and age variables; AUC=0.686 (0.63-0.73) for CV / LV EF ratio, p<0.001, AUC=0.65 (0.59-0.70) for age, p<0.001 (Figure 1).

Conclusions: CV / LV EF ratio and age were independent predictors for CIN development in patients with elective PCI.

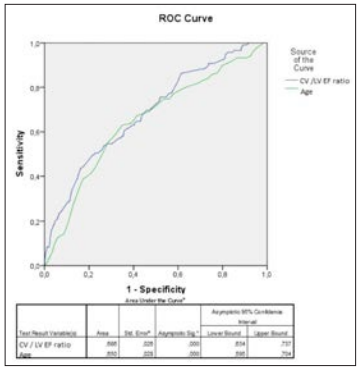

Figure 1. ROC of CV / LV EF ratio and age.

Table 1. Characteristics of patients with and without CIN

Variable	CIN (+) 119 patients	CIN (-) 763 patients	P value
Age (year)	65.22±10.75	59.77±11.04	<0.001
Gender, n (%) Female Male	38 (16.5) 81 (12.4)	192 (83.5) 571 (87.5)	0.18
BMI (kg/m2)	27.91±4.26	28.33±6.72	0.50
Hypertension n (%)	70 (58.8)	390 (51.1)	0.11
Diabetes mellitus n (%)	41 (34.5)	200 (26.2)	0.07
LV EF (%)	46.25±11.39	50.27±9.90	<0.001
Metformin	39 (27.7)	159 (20.8)	0.09
ACEI	61 (51.3)	451 (59.1)	0.11
Statin	87 (73.1)	508 (66.6)	0.16
Glukoz (mg/dl)	120.56±40.25	127.24±56.17	0.41
Ure (mg/dl)	34.50±13.47	33.02±15.35	0.52
Kreatinin (mg/dl)	0.89±0.24	0.90±0.28	0.69
GFR (ml/min)	91.48±35.14	99.86±31.91	<0.001
Contrast amount (ml)	190.08±111.45	138.58±73.04	0.009
ACEF score	1.52±0.57	1.26±0.47	<0.001
CV / LV EF ratio	4.49±3.19	2.90±1.74	<0.001

BMI: Body mass index, LV EF: Left ventricular ejection fraction, ACEI: Angiotensin-converting enzyme inhibitor, GFR: Glomerular filtration rate, CV: Contrast volume.

Table 2. Multivariate linear regression analysis

Variable	Standartize Coefficient Beta	t Value	P value
Age	0.205	3.408	0.001
LV EF	-0.028	-0.407	0.68
GFR	0.044	0.989	0.32
Contrast amount	-0.125	-1.027	0.304
CV/LV EF ratio	0.379	2.769	0.006
ACEF score	-0.099	-1.064	0.287

LV EF: Left ventricular ejection fraction, GFR: Glomerular filtration rate, CV: Contrast volume.

Other

OP-275

Aortic arch calcification is strongly associated with obstructive sleep apnea

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Background and Aim: Obstructive sleep apnea (OSA) is characterized by recurrent partial (hypopnea) or complete interruption (apnea) attacks in breathing caused by airway collapse in the pharyngeal region during sleep. OSA is a common clinical condition that causes an increase in cardiovascular morbidity and mortality. OSA and its cardiovascular results have been investigated in several studies. A lot of evidence confirms that OSA is involved in the etiology of many diseases such as hypertension, atrial fibrillation, coronary artery disease and heart failure. OSA is likely to show increased arterial stiffness and progressive systemic atherosclerosis. Chest radiography reveals atherosclerotic changes in the aorta. The aim of this study was to investigate the relationship between aortic arch calcification (AAC) on chest radiography and OSA.

Methods: 204 patients (age: 55±14 years; 78 men) who were diagnosed with OSA by performing night polysomnography were evaluated. On the other hand 200 (age: 48±15 years; 94 men) patients were selected to the group non OSA. AAC was evaluated with chest radiography and inter-observer agreement was analyzed by using kappa statistics. Univariate and multivariate logistic regression analysis was conducted to assess the association of AAC and OSA. P-value <0.05 was considered statistically significant.

Results: A total of 404 patients (median age: 51.5) were included. Patients included in the study; OSA (+) (n=204) and OSA (-) (n=200) were divided into two groups and compared. Overall, the prevalence of AAC was 207 (51.2%). OSA group had significantly higher prevalence of AAC (79% vs. 32.5%, p<0.0001) as compared to the normal group. Presence of AAC was a strong and independent predictor of OSA (OR 3.923, 95%CI 2.396 to 6.328) in multivariate analysis.

Conclusions: Presence of AAC on plain chest radiography is strongly and independently associated with the presence of OSA. Our findings suggest that a presence of ACC suggests atherosclerotic changes in the aorta and may be associated with OSA.

Table 1. Baseline characteristics of the study groups

	OSA (+) (n=204)	OSA (-) (n=200)	P value
Age (years)	55 ± 14	48 ± 15	< 0.001
Male, n (%)	78 (38.2)	94 (47)	0.064
BMI, kg/m2	29 ± 5	28 ± 4	0.062
Smoker, n (%)	65 (31.8)	52 (26)	0.110
Known CAD, n (%)	10 (4.9)	2 (1)	0.165
Hypertension, n (%)	104 (50.9)	83 (41.5)	0.051
Glucose (mg/dL)	106 ± 30	102 ± 29	0.086
Diabetes mellitus, n (%)	46 (22.5)	34 (17)	0.286
Total cholesterol (mg/dL)	189 ± 40.6	194 ± 40.4	0.198
Triglyceride (mg/dL)	191 ± 39	174 ± 81	0.019
Low-density lipoprotein (mg/dL)	116 ± 38	118 ± 37	0.902
High-density lipoprotein (mg/dL)	46 ± 12	45 ± 13	0.498
Creatinine (mg/dL)	0.82 ± 0.18	0.82 ± 0.27	0.880
Antihypertensive drug, n	47	35	0.049
Antidiabetic drug, n	2	1	0.988
AHI, /h	50.5 ± 14.4	2.1 ± 1.7	<0.001
Aurosal index, /h	48 ± 17.2	19.8 ± 9.6	<0.001
ODI (3%), /h	40.1 ± 20.2	0.8 ± 0.7	<0.001
Lowest SpO2, %	75 ± 9.7	90.5 ± 3.5	<0.001

AHI, apnea-hypopnea index; BMI, body mass index; CAD,coronary artery disease; ODI, oxygen desaturation index; SpO2, peripheral oxyen saturation.

Table 2. Aortic arch calcification grades in the study groups

Aortic arch calcification (n, %)	OSA (+) (n=204)	OSA (-) (n=200)	P value
Grade 0	62 (30.3)	135 (67.5)	<0.0001
Grade 1	86 (42.1)	50 (25)	<0.001
Grade 2	45 (22)	14 (7)	<0.001
Grade 3	10 (4.9)	1 (0.5)	<0.001

OSA, obstructive sleep apnea.

Table 3. Univariate analysis for obstructive sleep apnea

Variables	β	P value
Age	0.172	<0.001
Male	0.006	0.056
BMI	0.201	<0.001
Smoker	-0.186	0.103
Known CAD	0.068	0.024
Hypertension (%)	0.526	0.048
Diabetes mellitus (%)	0.438	0.301
Total cholesterol (mg/dL)	-0.012	0.235
Triglyceride (mg/dL)	-0.002	0.089
Antihypertensive drug	0.490	0.044
Antidiabetic drug	0.042	0.382
AHI	0.212	<0.001
Aruosal index	0.282	<0.001
ODI (%3)	0.224	<0.001
Lowest SpO2	-0.134	0.001

AHI, apnea-hypopnea index; BMI, body mass index; CAD,coronary artery disease; ODI, oxygen desaturation index; SpO2, periphral oxyen saturation.

Table 4. Correlations between OSA and AAC

Aortic arch calcification (%)	β	P value
Grade 0	Reference category	
Grade 1	1.384	<0.0001
Grade 2	1.846	<0.0001
Grade 3	2.698	0.006

ACC, aortic arch calcification; OSA, obstructive sleep apnea.

Table 5. Multivariate analysis for obstructive sleep apnea

Variables	β	OR	Lower	Upper
Age	0.014	1.014	0.978	1.034
Body mass index	0.035	1.036	0.987	1.089
Hypertension	0.060	1.062	0.658	1.690
Triglyceride	-0.002	0.985	0.989	0.996
Presence of aortic arch calcification	1.362	3.923	2.396	6.328
AHI	0.15	1.018	0.985	1.049
Male	0.15	1.008	0.964	1.027

AHI, apnea-hypopnea index.

Other

OP-276

Platelet count on admission is associated with the duration of hospital stay in patients with coronavirus disease (COVID-19)

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Background and Aim: In December 2019, an unknown pneumonia epidemic was detected in Wuhan, Hubei province, China. In February 2020 the World Health Organization referred it coronavirus disease 2019 (COVID-2019). Since then COVID-19 continues to spread rapidly worldwide. The duration of hospitalization of patients is a substantial matter regarding cost-effectiveness and the burden of the disease and the low capacity of health care systems in several countries, especially the developing countries. In the present study, we aimed to examine the factors affecting the duration of hospital stay.

Methods: 140 patients were enrolled in our retrospective cohort study. Patients divided into 2 groups as ≤9 days and >9 days duration of hospital stay. Diagnosis of COVID-19 was determined by detecting SARS-Cov-2 RNA with real-time PCR method according to the World Health Organization's temporary guideline. Indication for hospitalization was carried out in line with interim guidelines of the Republic of Turkey's coronavirus scientific advisory board.

Results: Both groups had similar baseline characteristics. Levels of high sensitive cardiac troponin I (1.9 (0.9-4.0) vs 4.7 (2.7-10.5), p<0.001), aspartate transaminase (23.0 (19.3-31.5) vs 29.0 (21.3-43.5), p=0.014), lactate dehydrogenase (229.5 (188.0-289.5) vs 260.0 (210.0-347.5.0), p=0.039) and c-reactive protein (1.6 (0.5-5.3) vs 3.6 (0.7-11.6), p=0.05) on admission were significantly higher in long-term hospitalization group. However, platelet count (194.0 (161.2-232.5) vs. 229.5 (197.5-281.5), p=0.001) and systolic blood pressure (111.5 (110.0-130.0) vs. 120.0 (110.0-131.5), p=0.045) on admission were lower in long-term hospitalization group. Forward stepwise logistic regression analysis showed that platelet count (OR: 0.994 (0.989-0.999), %95 CI, p=0.018) was an independent predictor of the duration of hospitalization in patients with COVID-19.

Conclusions: Platelet count on admission is significantly lower in long-term hospitalization patients with COVID-19 than in-short-term hospitalization patients.

Table 1. Comparison of baseline characteristics of inpatients with CoVID-19 according to hospitalization

Variable	Total (n=140)	Short-term hospitalization (n=84)	Long-term hospitalization (n=56)	p-value
Age (year)	49.2 ± 17.8	47.4 ± 17.1	51.9 ± 18.9	0.138
Gender, (male) n (%)	64 (45.7)	33 (39.3)	31 (55.4)	0.061
BMI (kg/m2)	27.3 (25.3-30.0)	27.4 (25.6-29.8)	26.8 (25.0-30.1)	0.602
Current smoker, n (%)	18 (12.9)	10 (11.9)	8 (14.3)	0.680
DM, n (%)	27 (19.3)	16 (19.0)	11 (19.6)	0.930
HT, n (%)	42 (30.0)	22 (26.2)	20 (35.7)	0.228
CAD, n (%)	16 (11.4)	8 (9.5)	8 (14.3)	0.386
HF, n (%)	3 (2.1)	1 (1.2)	2 (3.6)	0.564
COPD, n (%)	5 (3.6)	3 (3.6)	2 (3.6)	1.000
Stroke, n (%)	2 (1.4)	0 (0.0)	2 (3.6)	0.158
Hyperlipidemia	9 (6.4)	5 (6.0)	4 (7.1)	0.778

Data are presented as number (%), mean ± standard deviation or median (IQR) p-value was calculated using the Independent Samples t-test or Mann-whitney U test for continuous variables and the Chi-Square test or the Fisher's exact test for categorical variables as appropriate. p value <0.05 was considered significant. BMI: Body mass index, CAD: Coronary artery disease, COPD: Chronic obstructive pulmonary disease, DM: Diabetes mellitus, HF: Heart failure, HT: Hypertension.

Table 2. Comparison of laboratory characteristics of COVID-19 patients according to hospitalization

Variable	Total (n=140)	Short-term hospitalization (n=84)	Long-term hospitalization (n=56)	p-value
Hemoglobin (g/dL)	13.5 ± 1.8	13.5 ± 1.9	13.6 ± 1.5	0.138
Leukocyte count, x103/uL	5.9 (4.4-7.6)	5.8 (4.7-7.8)	5.8 (4.0-7.8)	0.359
Platelet count, x103/uL	222.0 (175.4-273.3)	229.5 (197.5-281.5)	194.0 (161.2-232.5)	0.001
Glucose (mg/dL)	104.0 (94.0-132.5)	105.0 (95.0-132.0)	101.5 (92.0-130.0)	0.683
GFR (mL/min per 1.73 m2)	97.7 ± 24.7	99.3 ± 26.7	95.9 ± 21.6	0.418
CRP (mg/L)	1.8 (0.6-7.2)	1.6 (0.5-5.3)	3.6 (0.7-11.6)	0.050
SBP (mmHg)	120.0 (110.0-132.0)	120.0 (110.0-131.5)	111.5 (110.0-130.0)	0.045
LDH (U/L)	243.0 (194.0-329.5)	229.5 (188.0-289.5)	260.0 (210.0-347.5.0)	0.039
ALT (U/L)	20.0 (15.0-28.0)	19.0 (15.0-26.8)	22.5 (15.3-26.8)	0.675
AST(U/L)	25.0 (20.0-36.0)	23.0 (19.3-31.5)	29.0 (21.3-43.5)	0.014
Hs-cTnI (ng/L)	3.0 (1.4-6.6)	1.9 (0.9-4.0)	4.7 (2.7-10.5)	<0.001
D-dimer (mcg/mL)	0.4 (0.2-3.9)	0.4 (0.2-3.8)	0.4 (0.2-1.1)	0.449
Lymphocyte count, x 103/uL	1.4 (0.8-2.0)	1.6 (1.1-2.0)	1.3 (0.8-2.0)	0.096

Data are presented as mean ± standard deviation or median (IQR). p-value was calculated using the Independent Samples t-test or the Mann-Whitney U-test. p value <0.05 was considered significant. ALT: Alanine transaminase, AST: Aspartate transaminase, CRP: C-reactive protein, GFR: Glomerular filtration rate, Hs-cTnI: High sensitivity cardiac troponin I, LDH: Lactate dehydrogenase, SBP: Systolic blood pressure.

Table 3. Risk factors associated with long-term hospitalization in COVID-19 patients

Variable	Univariate Analysis OR (95 % CI) p-value	Multivariate Analysis OR (95 % CI) p-value
Age (year)	1.015 (0.995-1.035) 0.139	-
Gender, (male) n (%)	1.916 (0.966-3.802) 0.069	-
HT, n (%)	1.566 (0.753-3.254) 0.230	-
SBP (mmHg)	0.985 (0.965-1.006) 0.170	-
Platelet count, x103/uL	0.970 (0.949-0.991) 0.015	0.994 (0.989-0.999) 0.018
CRP (mg/L)	1.007 (0.999-1.015) 0.101	-
LDH (U/L)	1.001 (0.999-1.004) 0.329	-
Hs-cTnI (ng/L)	1.002 (0.999-1.004) 0.315	-
AST (U/L)	1.009 (0.992-1.025) 0.309	-

p-value <0.05 was considered significant. HT: Hypertension, SBP: Systolic blood pressure, CRP: C-reactive protein, LDH: Lactate dehydrogenase, Hs-cTnI: High sensitivity cardiac troponin I, AST: Aspartate transaminase.

Other

OP-277

Ethical evaluation of the compliance of informed consent forms used in cardiology clinics with the templates of the Turkish Society of Cardiology

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Background and Aim: The informed consent (IC) process is a fundamental ethical step in health care, especially for invasive procedures. The ethical basis for IC depends on the respect for autonomy principle. Current literature and practical applications point to the existence of ethical problems in the IC procedures practiced in interventional cardiology departments. We aimed to evaluate the Turkish Society of Cardiology (TSC) ICF templates from an ethics point of view and to determine the compliance of ICFs used in three other institutions which are education and research, university, and private hospitals with these templates.
Methods: Templates of the TSC was evaluated in terms of the basic ethical criteria that should be included. In addition, the compliance of ICFs that received from the cardiology clinics of various hospitals with TSC templates was evaluated.
Results: Of the 185 ICFs received from hospitals, 106 (57.30%) were from nine university hospitals (U-ICF), 52 (28.11%) were from six state education and research hospitals (E&R-ICF) and 27 (14.60%) from six different private hospitals (P-ICF). Following criteria were 100% met in all TSC templates: Voluntariness/willingness, diagnosis of the patient, information about the diagnosed disease, the severity of the disease, description of the proposed action, risks of intervention, alternative treatments, designated space for signatures of the physician, designated space for signatures of the patient, addressing the legal guardian, a special arrangement for patients with reading difficulties/can't read, Explanation about the operator, training purpose, information that a copy of ICF will be given to the patient. But TSC templates were still incomplete regarding with expected benefits from treatment, duration of hospitalization, duration of the proposed intervention, the time needed to return to a normal life course information about the third party, prospects of the treatment, information for alternative treatments. Eventually, the discrepancy between diagnosis and explanations was detected in only one template (5.26%). The criteria included in the TSC template ICFs significantly higher than the ICFs used in hospitals were as follows: Information on the severity of the disease, quality of life after treatment, Information regarding a copy of ICF will be provided to the patient, Duration of the proposed treatment, Explanation about the diagnosis / pre-diagnosis of the patient, Adequate information about the disease, Information about alternative interventions, explanation of the operator, explanation of whether the procedure can be used for educational purposes.
Conclusions: Existing TSC forms should be increased in number to cover all interventions performed in cardiology as a pioneer and improved in accordance with the universal ethical standards to set a high-value healthcare system. This may improve ICF contents in an ethical manner and direct the institutions/healthcare providers in legitimate issues.

Other

OP-278

Effect of tripple antimicrobial theraphy on ECG parameters in patients with mild to moderate Covid-19 disease

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Background and Aim: The treatment of COVİD-19 with triple combination composed of hydroxychloroquine, antiviral and antibiotic on ECG parameters in mild to moderate symptomatic patients are not wholly understood. We aimed to explore the changes of ECG parameters after triple combination therapy for patients with mild to moderate symptomatic COVID-19.
Methods: This retrospective single-center case series analyzed 91 with mild to moderate symptomatic patients with COVID-19 at Gazi Mustafa Kemal State Hospital of Ankara City, Turkey, from April 1, 2020 to April 30, 2020. 43 patients were treated with hydroxychloroquine+oseltamivir+azithromycin (Group 1) and 48 patients were treated with hydroxychloroquine+oseltamivir+levofloxacin (Group 2).
Results: QTc, QRS duration, Tp-e, PR interval and P wave duration significantly increased after the treatment (p<0.001). Post-treatment CRP was significantly lower in group 1 (p=0.014). In 30% of patients had QT prolongation at admission, 4.3% of them had a QT duration >500 msc. QTc interval (p<0.001 vs. p<0.001), QRS duration (p=0.006 vs. p=0.014), Tp-e (p=0.036 vs. p<0.001), PR interval (p=0.002 vs. p=0.05) significantly prolonged in both groups. QTD significantly decreased in Group 1 (p<0.001). None of the patients experienced any overt ventricular arrhythmia.
Conclusions: To the best of our knowledge, this study is the first to investigate QT prolongation in a population treated with triple combination therapy. We found that there was a significant decrease of QTD after the treatment in patients who were taking triple theraphy with azithromycin.

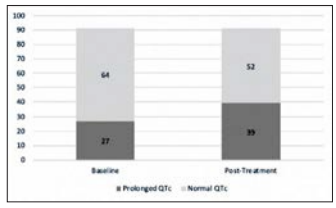

Figure 1. Baseline and post-treatment normal and prolonged QTc proportions.

Table 1. Clinical characteristics of patients with COVID-19

Age, years	41.1 ±15.4 (16-74)
Gender, Female, n, %	45 (49.5)
Diabetes Mellitus, n, %	9 (9.9)
Hypertension, n, %	11 (12.1)
CHD, n, %	3(3.3)
Any medication use,n, %	24 (26.2)
Antihypertensive	11 (12.1)
Oral Antidiabetics	9 (9.9)
Antiplatelets/Anticoagulants	5 (5.5)
Antiepileptic	3 (3.3)
Others	4 (4.4)
Creatinine, mg/dl	0.73±0.20 (0.38-1.56)
ALT, IU/L	28±25 (7-178)
AST, IU/L	26±15(10-97)
Sodium, mEq/L	136±2.8 (126-144)
Potassium, mEq/L	4.07±0.49(2.8-6.4)
WBC, x10^9/L	5.9±3.1 (0.11-25.6)
Haemoglobin, g/L	14.2±1.7 (9.2-18.6)
Platelet count, x10^9/L	200±57 (76-392)
Neutrophil count, x10^9/L	62.1±12.3 (6.8-87.6)
Lymphocyte count, x10^9/L	26.1±10.4 (5.5-58.7)
Moncyte count, x10^9/L	9.7±4.0 (0.5-30.5)
NLR	3.2±2.5 (0.15-15.93)

ALT. Alanine Aminotransferase; AST. Aspartate Aminotransferase; CHD. Coronary Heart Disease; NLR. Neutrophil to lymphocyte ratio; WBC. White blood cell.

Table 2. Clinical characteristics and ECG parameters of Group 1 and 2

	Group 1	Group 2	P
Age. years	35±14	46±15	0.001
Gender. Female. n. %	21(48.8)	24(50)	0.912
Hypertension. n. %	2(4.7)	9(18.8)	0.039
Diabetes Mellitus. n. %	5(11.6)	4(8.3)	0.730
CHD. n. %	0	3(6.2)	NA
Baseline Heart Rate. bpm. mean±SD	79±16	83±13	0.147
Baseline QTc. ms. mean±SD	446±30	434±40	0.139
Baseline QTD. ms. median(IQR)	20(15-28)	20(14-34)	0.593
Baseline QRS duration. ms. mean±SD	107±19	105±20	0.673
Baseline Tp-e. ms. mean±SD	102±16	86±18	<0.001
Baseline Tp-e Dispersion. ms. median(IQR)	12(8-18)	9(6-15)	0.095
Baseline Tp-e/QTc ratio. mean±SD	0.23±0.04	0.20±0.04	<0.001
Baseline PR interval. ms. median(IQR)	150(134-164)	148(129-176)	0.707
Baseline P wave duration. ms. mean±SD	113±17	106±19	0.105
Baseline P wave dispersion. ms. median(IQR)	9(5-15)	6(4-11)	0.101
PT Heart Rate. bpm. median(IQR)	72(66-78)	76(69-86)	0.045
PT QTc. ms. mean±SD	456±28	455±38	0.960
PT QTD. ms. median(IQR)	15(13-21)	20(13-27)	0.099
PT QRS duration. ms. median(IQR)	112(95-120)	106(98-126)	0.683
PT TP-e. ms. mean±SD	104±14	95±15	0.007
PT Tp-e dispersion. ms. median(IQR)	10(6-15)	10(6-14)	0.758
PT Tp-e/QTc ratio. mean±SD	0.23±0.03	0.21±0.03	0.005
PT PR interval. ms. mean±SD	160±24	163±35	0.582
PT P wave duration. ms. mean±SD	115±17	115±121	0.896
PT P wave dispersion. ms. median(IQR)	9(6-13)	8(6-13)	0.583
Delta QTc	12(4-18)	20.5(10.5-35)	0.008

Group 1: hydroxychloroquine + oseltamivir + azithromycin
Group 2: hydroxychloroquine + oseltamivir + levofloxacin
ALT. Alanine Aminotransferase; AST. Aspartate Aminotransferase; CHD. Coronary Heart Disease; CRP. C-reactive protein; IQR. interquartile range; NLR. Neutrophil to lymphocyte ratio; PLR. Platelet to lymphocyte ratio; PT. Post-Treatment; QTc. corrected QT interval; QTD. dispersion of QTc; SD. standard deviation; Tp-e. peak-to-end interval of the T wave; WBC. White blood cell.

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Table 3. Characteristics of ECG parameters in the patients before and after treatment

	Baseline		After Treatment		p
	Mean ± SD	Median(IQR)	Mean ± SD	Median(IQR)	
Heart Rate, bpm	81±14	82(70-91)	74±14	74(68-81)	<0.001
QTc, ms	440±35	442(418-462)	455±34	453(432-479)	<0.001
QTD, ms	24±15	20(15-30)	20±10	18(13-24)	0.004
QRS duration, ms	106±19	104(93-120)	112±23	112(96-124)	<0.001
Tp-e, ms	94±19	94(80-105)	99±15	98(88-111)	<0.001
Tp-e Dispersion, ms	12±7	10(7-16)	11±6	10(6-14)	0.195
Tp-e/QTc ratio	0.21±0.04	0.21(0.19-0.24)	0.22±0.03	0.22(0.20-0.24)	0.154
PR interval, ms	153±29	150(132-165)	161±30	162(139-176)	0.001
P wave duration, ms	110±18	112(96-122)	115±19	115(103-128)	0.001
P wave dispersion, ms	9±6	8(4-12)	10±5	8(6-13)	0.277

IQR, interquartile range; QTc, corrected QT interval; QTD, dispersion of QTc; SD, standard deviation; Tp-e, peak-to-end interval of the T wave.

Table 4. Characteristics of ECG parameters before and after treatment in the patients treated with ahd without Azithromycin

Variables	Group 1					Group 2				
	Baseline		Post Treatment			Baseline		Post Treatment		
	Mean±SD	Median (IQR)	Mean±SD	Median (IQR)	p	Mean±SD	Median (IQR)	Mean±SD	Median (IQR)	P
Heart Rate. bpm	79±16	77(69-90)	72±10	72(66-78)	<0.001	83±13	86(72-92)	76±17	76(69-86)	0.007
QTc. ms	446±30	447(425-464)	456±28	455(440-473)	<0.001	434±40	434(402-458)	455±38	453(426-489)	<0.001
QTD. ms	21±9	20(15-28)	18±9	15(13-21)	0.001	27±19	20(14-34)	21±11	20(13-27)	0.381
QRS duration	107±19	105(95-120)	110±21	112(95-120)	0.006	105±20	101(92-120)	114±26	106(98-126)	0.014
Tp-e. ms	102±16	100(89-110)	104±14	102(94-114)	0.036	86±18	86(76-98)	95±15	94(85-106)	<0.001
Tpe-e dispersion. ms	13±6	12(8-18)	11±6	10(6-15)	0.156	11±8	9(6-15)	10±6	10(6-14)	0.635
Tp-e/QTc ratio	0.23±0.04	0.23(0.20-0.25)	0.23±0.03	0.24(0.20-.025)	0.858	0.20±0.04	0.20(0.18-0.23)	0.21±0.03	0.21(0.19-0.23)	0.073
PR interval. ms	153±25	150(134-164)	160±24	162(139-172)	0.002	153±33	148(129-176)	163±35	160(139-183)	0.050
P wave duration. ms	113±17	114(97-124)	115±17	120(104-129)	0.075	106±19	106(93-119)	115±121	112(103-128)	0.007
P wave. dispersion. ms	10±7	9(5-15)	10±5	9(6-13)	0.535	8±6	6(4-11)	9±5	8(6-13)	0.055

Group 1: hydroxychloroquine + oseltamivir + azithromycin
Group 2: hydroxychloroquine + oseltamivir + levofloxacin
IQR. interquartile range; QTc. corrected QT interval; QTD. dispersion of QTc; SD. standard deviation; Tp-e. peak-to-end interval of the T wave.

Other

OP-279

Elevated troponin-T levels in patients with amyotrophic lateral sclerosis (ALS)

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Background and Aim: Cardiac troponin-T (TnT) is a specific marker of myocardial injury that is used when suspected acute coronary syndromes. However, TnT levels may rise due to non-cardiac conditions such as acute renal failure, pulmonary embolism, stroke, myocarditis, heart failure etc. In addition, some case reports and studies have shown ALS, the most common motor neuron disease, can raise TnT levels, but it's cause remains still unclear. In this study we aimed to compare the survival and clinical charateristics between troponin positive and negative ALS patients.

Methods: The diagnosis of ALS was established by a board certified neurologist who followed the applicable El Escorial World Federation of Neurology criteria. In accordance with the guidelines rise of Tnt levels above 99th percentile of normal reference population was referred abnormal. The patients with renal insufficiency, coronary artery disease (>%50 stenosis at least one coronary artery), malignancy, heart failure, artifical valve were excluded from the study.

Results: We identified 250 patients diagnosed with ALS, after exclusion there were 243 patients remained. In ALS cohort 206 patients (%84.8) had positive TnT. Myocardial perfusion scintigrapy was performed to 30 ALS patients with positive TnT. There was only one patient who require coronary angiograpy. After performing coronary angiography, significant coronary stenosis was observed and stenting was the preferred option to this patient. In addition, FEV1 and FVC values were found abnormally which is marked restrict lung expansion however we did not find any statistically difference between troponin positive and negative patients. When troponin negative and positive patients compared about mortality, any statistically difference did not found.

Conclusions: This study show that abnormal elevation of TnT were found in the vast majority of ALS patients. Although there is no statistically difference between troponin positive and negative patients in survival. In this study, we couldn' t explain why troponin levels were high. However, deterioration of respiratuar functions in Tnt positive patients shed light on that chronic myocardial ischemia may cause the elevation of troponin levels. But further investigations need to prove this mechanism.

Table 1. Patient characteristics

Positive Tnt	206 (%84.8)
Sex (female)	100 (%36.6)
Hypertension	36 (%13.2)
Diabetes Mellitus	39 (%14.3)

Tnt: Troponin T by high-sensitivity immuno-assay.

Table 2. Comparison of Tnt positive and negative ALS patients

	Tnt positive ALS patients (n=206)	Tnt negative ALS patients (n=37)	p value
Exitus	24 (%11.7)	4 (%10.8)	0,88
Hypertension	25 (%12.1)	7 (%18.9)	0,29
Diabetes Mellitus	33 (%16)	11 (%29.7)	0,06
FEV1 (% of predicted)	83.1 (±28.1)	80.4 (±23.8)	0,59
FVC (% of predicted)	78.9 (±29.5)	72.8 (±25.4)	0,37

FVC: Forced vital capacity, FEV1: Forced expiratory volume in 1 s, Tnt: troponin T by high-sensitivity immuno-assay.

Other

OP-280

Do women with endometriosis have increased arterial stiffness?

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Background and Aim: Endometriosis is a common gynecological disease, which is associated with systemic inflammation and atherogenic risk markers. Therefore, cardiovascular risk can be increased in women with endometriosis. Arterial stiffness is one of the surrogate markers used for cardiovascular events. In this study, we aim to evaluate arterial stiffness using cardio-ankle vascular index (CAVI) in women with endometriosis and compare with women without.

Methods: A group of 120 women, matched for age were recruited. Diagnosis of endometriosis was made on histopathological or magnetic resonance examination. Arterial stiffness using CAVI were evaluated in the study group with and without endometriosis. CAVI was measured using VaSera TM (VS-1500 system, Fukuda Denshi, Tokyo, Japan). All patients included in the study underwent comprehensive echocardiography. Left ventricular mass index and relative wall thickness were calculated from dimensions of cardiac chambers. Diastolic functions were evaluated for all patients.

Results: Women with endometriosis and control group showed similar left ventricular (LV) ejection fraction, LV mass index, relative wall thickness. On the other hand greater arterial stiffness was measured by CAVI in women with endometriosis compared to controls. Adjusted CAVI levels were also found to be higher in women with endometriosis.There was no difference in mean ankle-brachial index (ABI) among the two groups (Figure 1a). However, CAVI levels in the endometriosis group were significantly higher when compared to controls (p=0.001) (Figure 1b). Since age was documented to be independently correlated with CAVI, a covariance analysis was performed in which CAVI was adjusted for age. This analysis revealed that age adjusted CAVI levels were also found to be higher in women with endometriosis (p=0.001) (Figure 1c).Bivariate correlation between CAVI and LV mass index, EF, RWT, and E/E' was also performed. Only LV mass index was found to be significantly correlated with CAVI (p=0.01) (Figure 2). Since LV mass index was a significant independent predictor of CAVI, we further adjust CAVI for this confounder together with age. CAVI was higher in the endometriosis compared to controls, even after controlling for the effects of age and LV mass index (Figure 1d) (p=0.007).

Conclusions: Endometriosis is a common gynecological condition, that is associated with systemic inflammation and atherogenic risk factors. In this study, we found higher CAVI indicating increased arterial stiffness without significant alteration in echocardiographic parameters. This is suggestive of a subclinical state cardiovascular disease in women with endometriosis. Physicians should be aware of the possible increased cardiovascular risk in women with endometriosis.

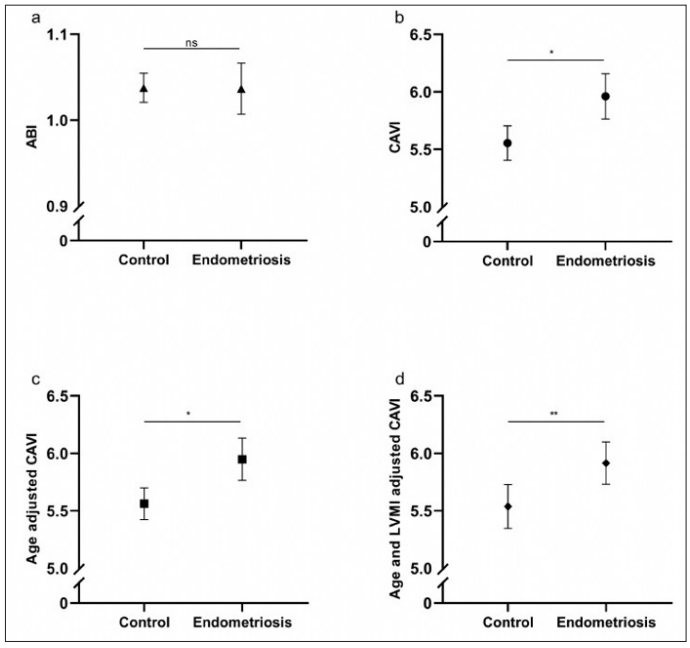

Figure 1. Comparison of arterial stiffness parameters between two groups. *p=0.001 vs. control **p=0.007 vs. control.

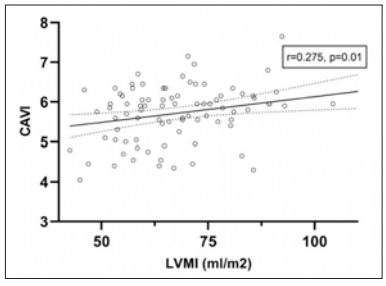

Figure 2. Correlation of left ventricular mass index with CAVI.

Other

OP-281

Right atrioventricular groove epicardial fat tissue thickness is associated with right ventricular dysfunction in patients with acute pulmonary embolism

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Background and Aim: As a part of the visceral adiposity, epicardial fat tissue (EFT) has unique inflammatory properties that has been linked to a broad spectrum of cardiovascular diseases. Inflammation has an intertwined relationship with thromboembolic diseases such as venous thromboembolism. Being in the grave end of the venous thromboembolism spectrum, acute pulmonary embolism (APE), has been already been investigated in studies addressing various markers of inflammation, however, its relationship with the EFT has not been adressed. In the context of APE, increased EFT may have detrimental effects on the right ventricle that may eventually conclude right ventricular dysfunction (RVD). EFT can easily be recognized on the images of the computerized tomographic studies of APE patients, and its thickness or volume, whichever applicable, can be measured. In this study, we aim to introduce the role of EFT, which is quantified as thickness, on the RVD in APE patients.

Methods: In this retrospective, cross-sectional study we included 92 low to moderate risk APE patients who were undergone pulmonary computerized tomography angiogram between April 2016 and January 2019. Patients' records were noted, simplified pulmonary embolism severity index (sPESI) scores were calculated, and computerized tomography pulmonary artery obstruction index (CTPAOI), which was initially introduced by Qanadli et al., was calculated. EFT thicknesses were measured from the regarding axial tomographic images representing the maximal thicknesses of the right and left atrioventricular and the anterior interventricular groove fat tissue. We categorized patients into two groups according to the RVD which was determined from right to left ventricular maximal diameter ratio of 0.9 that were measured from the regarding axial tomographic planes.

Results: Patients with RVD were 8 years older, presentation with syncope was higher than patients without RVD. Also, serum troponin levels were more elevated in patients with RVD. All of the EFT thicknesses were increased, and this increment was more pronounced in right atrioventricular groove EFT thickness (3 mm). In multivariate regression analysis only baseline serum troponin levels (OR: 1.006 per 0.001 ng/mL increase, p=0.050), and right atrioventricular groove EFT thickness (OR: 1.199, 95% Confidence Interval 1.024-1.404 per 1 mm increase, p=0.024). Right atriyoventricular groove EFT thickness had moderately positive correlations with increasing age (Pearson r=0.289) and, CTPAOI (r=0.269). In 71.7% cases (AUC 0.717, 95% CI of 0.612-0.823, p=0.001) a cut-off of 16.05 mm for right atrioventricular groove EFT thickness discriminated patients with RVD with a 69.1% sensitivity and, 65.7% spesificity.

Conclusions: Right atrioventricular groove EFT thickness seems a promising marker for RVD in patients with APE. This marker may find a valuable place in the APE management, especially when noninvasive tools such as transthoracic echocardiography are utilized for its measurement.

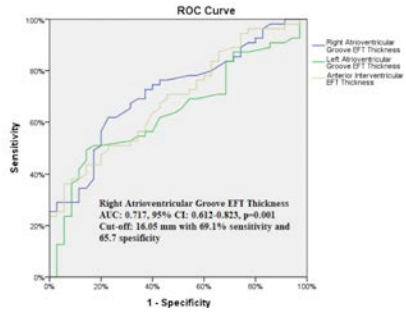

Figure 1.

Other

OP-282

Impact of ranolazine treatment on liver function tests in patients with coronary heart disease and non-alcoholic fatty liver disease

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Background and Aim: Non-alcoholic fatty liver disease (NAFLD) is the most common liver pathology and the most common cause of deterioration in liver function tests in the developed world. NAFLD's main significance lies in its association with atherosclerosis and a greater occurrence of cardiovascular disease. We set out to investigate the impact of ranolazine on liver function tests in patients with NAFLD and coronary artery disease (CAD), by measuring transaminase activities.

Methods: In this study, patients who established CAD and NAFLD were allocated to "on ranolazine" (n=40) or "not on ranolazine" (n=35) groups. Concentrations of serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were measured in all patients at baseline and at the end of study.

Results: The mean age was 59.2±9.4 years with 61 patients (81.3%) being male. After 6 months of ranolazine therapy, both ALT and AST levels were significantly lower in patients belonging to "on ranolazine" group compared with "not on ranolazine" patients (change from baseline for ALT, -10.97±1.70 IU/L, p<0.001 and change from baseline for AST, -5.22±1.93 IU/L, p=0.009, respectively). The decreases in ALT and AST are substantially higher in the ranolazine group.

Conclusions: The present study showed that treatment with ranolazine for 6 months led to a significant reduction in both serum aminotransferase levels in patients with stable CAD and NAFLD.

Other

OP-283

Preoperative cardiac risk factors associated with in-hospital mortality in elderly patients undergoing hip fracture surgery: A single center study

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Background and Aim: In elderly patients, hip fracture is associated with an increased morbidity and mortality. A standard approach to hip fracture usually involves a combination of surgery and rehabilitation. Currently, preoperative risk prediction indexes, such as the Goldman cardiac risk index and revised cardiac risk index, are not very specific to the elderly hip fracture patients. Therefore, it is important to find new parameters in the preoperative risk evaluation of such patients in addition to the above-mentioned risk indexes. In the present study, our aim was to determine the cardiac risk factors associated with the in-hospital mortality in the elderly patients (aged over 65 years) who required preoperative cardiology consultation for hip fracture surgery.

Methods: This retrospective, single-center study included the patients who were scheduled to hip fracture surgery at the Sultan Abdulhamid Han Training and Research Hospital. In all patients, an anesthesiologist performed a detailed preoperative evaluation and decided the need for the cardiac consultation. Patients underwent preoperative cardiac evaluation by a trained cardiologist using the algorithms proposed in the recent preoperative guidelines. The primary outcome of the study was the in-hospital all-cause mortality.

Results: A total of 277 elderly patients undergoing hip fracture surgery were enrolled in the study. The overall in-hospital mortality rate was 12.1% (n=30 cases). In a multivariate analysis, the independent predictors of in-hospital mortality were insulin dependency, cancer, urea, atrial fibrillation (AF) (OR: 3.906; 95% CI: 1.470–

10.381; p=0.006), and pulmonary artery systolic pressure (PASP) (OR: 1.057; 95% CI: 1.016–1.100; p=0.006). The receiver operating characteristic curve analysis revealed that the optimal value of PASP in predicting the in-hospital mortality was 35 mm Hg (area under the curve = 0.71; 95%CI: 0.60–0.81, p<0.001) with a sensitivity of 87.7% and a specificity of 59.5%.

Conclusions: The present research found that the preoperative cardiac risk factors, namely AF and PASP, might be associated with an increased in-hospital mortality in patients undergoing hip fracture surgery.

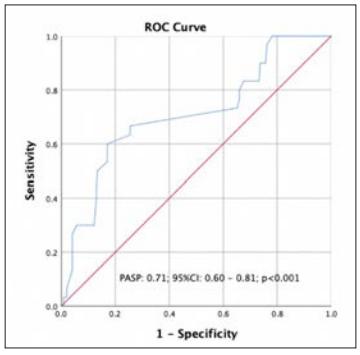

Figure 1. The optimal cut-off value of pulmonary artery systolic pressure in predicting in-hospital mortality of the elderly patients who required hip fracture surgery.

Table 1. Comparison of demographic clinical characteristics and echocardiography parameters all patients according to in-hospital mortality due to hip fracture

	Survivor, (+) n=247	Death, (+) n=30	p value
Age, y	81.7 ± 7.5	84.4 ± 6.2	0.075
Male gender, n (%)	66 (26.7)	8 (26.7)	0.995
Hypertension, n (%)	188 (76.1)	25 (83.3)	0.376
Diabetes mellitus, n (%)	69 (27.9)	11 (36.7)	0.319
Insulin dependency, n (%)	19 (7.7)	7 (23.3)	0.013
Hyperlipidemia, n (%)	17 (6.9)	3 (10.0)	0.464
COPD, n (%)	27 (10.9)	7 (23.3	0.071
Dementia, n (%)	65 (26.3)	9 (30.0)	0.670
Cancer, n (%)	37 (15.0)	12 (40.0)	0.002
Coronary artery disease, n (%)	39 (15.8)	2 (6.7)	0.276
CRF, n (%)	26 (10.5)	10 (33.3)	0.002
CVA, n (%)	26 (10.5)	3 (10.0)	1.000
Atrial fibrillation, n (%)	40 (16.2)	16 (53.3)	<0.001
Echocardiographic parameters			
Ejection fraction, %	60.0 ± 3.5	59.5 ± 4.5	0.695
LA anterior-posterior diameter,mm	38.2 ± 4.1	42.8 ± 7.3	0.001
LVEDD,mm	47.8 ± 3.7	48.3 ± 4.1	0.887
LVESD,mm	35.3 ± 3.6	36.1 ± 3.6	0.283
MR ≥+3, n (%)	42 (17.0)	7 (23.3)	0.406
TR ≥+3, n (%)	35 (14.2)	6 (20.0)	0.271
AR ≥+3, n (%)	17 (6.9)	4 (13.3)	0.180
PASP, mm Hg	29.4 ± 16.3	43.0 ± 9.4	<0.001

CVA, cerebrovascular accident; CRF, chronic renal failure; COPD, chronic obstructive pulmonary disease; AF, atrial fibrillation; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; MR, mitral regurgitation; TR, tricuspid regurgitation; AR, aortic regurgitation; AS, aortic stenosis; PASP, pulmonary artery systolic pressure.

Table 2. Multivariate analysis demonstrating independent predictors of in-hospital mortality

Multivariate analysis*	P value	OR (95% CI)
Insulin dependency	0.017	4.657 (1.312 – 16.528)
Cancer	<0.001	6.553 (2.375 – 18.080)
Urea	0.001	1.020 (1.009 – 1.032)
Atrial fibrillation	0.006	3.906 (1.470 – 10.381)
PASP	0.006	1.057 (1.016 – 1.100)

**Logistic regression analyses using the backward LR method were used for multivariate analysis of independent variables that were included if they were significantly different in univariate analyses (p<0.05). OR, odds ratio; CI, confidence interval.*

Other

OP-284

Serum magnesium level is associated with contrast induced nephropathy after primary percutaneous coronary intervention

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Background and Aim: Contrast induced nephropathy is an important complication of percutaneous coronary intervention since it is associated with increased morbidity and mortality. Previous studies showed the useful effects of magnesium on various processes including endothelial dependent vasodilatation and inflammation. Association between serum magnesium level and contrast induced nephropathy development after primary percutaneous coronary intervention with a diagnosis of ST-elevation myocardial infarction was evaluated in this study.

Methods: Data of 2426 consecutive patients who underwent primary percutaneous coronary intervention for ST-segment elevation myocardial infarction were analyzed. A serum creatinine level increase ≥0.5 mg/dl or ≥25% above baseline within 72 h after contrast administration were defined as contrast induced nephropathy. Independent predictors of contrast induced nephropathy were investigated with logistic regression analysis.

Results: Patients with contrast induced nephropathy had significantly lower magnesium level (2.111±0.183 vs. 1.926±0.208, p<0.001; Table 1, Table 2). In the study population, the cut-off value for Mg obtained by the receiver-operating characteristic curve analysis was less than 2.01mg/dl for the prediction of contrast induced nephropathy (area under the curve was 0.738; 95% CI, 0.716–0.760; p<0.001; sensitivity 72.1%; specificity 73.2%; Fig. 1). In multivariate logistic regression analysis magnesium (OR 0.092, 95% CI 0.039-0.218, p<0.001) was found as an independent predictor of contrast induced nephropathy. Age, male gender, admission Killip Class >1, white blood cell count and contrast volume were other independent predictors of contrast induced nephropathy (Table 3).

Conclusions: Magnesium may have a role to predict contrast induced nephropathy in patients undergoing primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. Further studies are required to elucidate the exact effect of magnesium on contrast induced nephropathy after percutaneous coronary intervention.

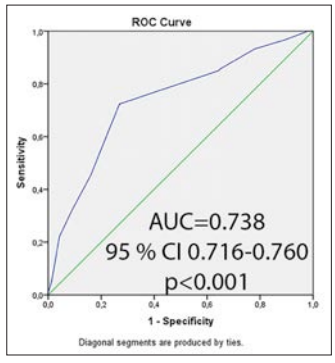

Figure 1. Receiver-operating characteristic curve analysis of magnesium for prediction of contrast-induced nephropathy.

Table 1. Baseline characteristics of study patients

Variables	CIN (-) (n=733)	CIN (+) (n=1693)	p value
Age	56.12 ± 11.53	59.49 ± 12.65	<0.001
Male gender	1314 (77.6)	607 (82.8)	0.010
Diabetes mellitus	426 (25.3)	221 (30.4)	0.010
Hypertension	680 (42.3)	325 (46.8)	0.051
Family history of CAD	275 (17.5)	124 (18.6)	0.511
Hyperlipidemia	624 (39.5)	237 (34.8)	0.034
Smoking	945 (60.5)	421 (63.2)	0.235
Previous CABG	59 (3.5)	15 (2.1)	0.080
Previous PCI	140 (8.3)	72 (9.9)	0.212
Killip class >1	71 (6.3)	88 (18.8)	<0.001
SBP < 100 mmHg	136 (13.7)	97 (22.8)	<0.001
HR > 100 min-1	57 (5.7)	73 (17.1)	<0.001
LVEF	48.23 (10.78)	44.64 (12.68)	<0.001
Contrast volume	233.53 ± 94.70	248.69 ± 106.31	<0.001
Tirofiban use	1086 (65.2)	449 (62.3)	0.167
Stent length	18.79 ± 6.48	20.19 ± 7.20	0.058
Stent diameter	3.152 ± 0.940	3.068 ± 0.321	0.042

Table 2. Laboratory findings of the patients

Variables	CIN (-) (N=1693)	CIN (+) (N=733)	P value
Admission creatinine	0.983 ± 0.336	1.012 ± 0.604	0.139
Peak creatinine	1.060 ± 0.308	1.478 ± 0.884	<0.001
Peak CK-MB	209.29 ± 169.27	253.84 ± 203.73	<0.001
Total cholesterol	189.72 ± 43.43	185.56 ± 42.55	0.055
LDL-C	117.58 ± 35.98	116.91 ± 34.87	0.721
HDL-C	41.21 ± 9.90	41.29 ± 10.10	0.865
Triglyceride	151.37 ± 111.63	143.99 ± 103.54	0.187
Glucose	155.16 ± 60.67	168.78 ± 87.34	<0.001
WBC	12.318 ± 3.833	13.139 ± 4.353	<0.001
Hemoglobin	13.58 ± 1.72	13.50 ±1.80	0.305
Mg	2.111 ± 0.183	1.926 ± 0.208	<0.001
K	4.096 ± 0.521	4.138 ± 0.712	0.106

Table 3. Independent Predictors of CIN in logistic regression analysis

Independent Predictors of CIN	Univariable OR (95% CI) p value	Multivariable OR (95% CI) p value
Age	1.024 (1.016-1.031) <0.001	1.036 (1.019-1.053) <0.001
Male gender	1.390 (1.111-1.737) 0.004	1.759 (1.083-2.858) 0.023
Diabetes	1.287 (1.062-1.560) 0.010	1.318 (0.816-2.131) 0.259
Hypertension	1.200 (1.003-1.435) 0.046	1.220 (0.833-1.788) 0.307
Killip class > 1	3.457 (2.476-4.828) <0.001	2.201 (1.005-4.822) 0.049
BP < 100 mmHg	1.851 (1.386-2.474) 0.001	1.231 (0.721-2.102) 0.446
HR > 100 min-1	3.435 (2.379-4.959) <0.001	1.404 (0.604-3.262) 0.430
LVEF	0.974 (0.963-0.984) <0.001	1.002 (0.983-1.020) 0.862
Admission anemia	1.108 (0.909-1.350) 0.311	1.308 (0.860-1.989) 0.209
Peak CK-MB	1.001 (1.001-1.002) <0.001	1.000 (0.999-1.001) 0.562
Admission creatinine	1.151 (0.952-1.392) 0.147	0.968 (0.608-1.540) 0.890
Glucose	1.002 (1.001-1.003) <0.001	1.001 (0.999-1.004) 0.282
Magnesium	0.102 (0.068-0.154) <0.001	0.092 (0.039-0.218) <0.001
WBC	1.050 (1.028-1.073) <0.001	1.071 (1.022-1.1249 0.004
Contrast volume	1.002 (1.001-1.002) 0.001	1.001 (1.001-1.002) 0.001

Other

OP-285

Advanced stage chronic obstructive pulmonary disease should be kept in mind in patients with high NT-proBNP levels

Armağan Acele

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Background and Aim: NT-proBNP level is closely related to many cardiovascular diseases and heart failure. It is a known fact that NT-proBNP level increases with right ventricular diastolic filling pressure increase in patients with chronic obstructive pulmonary disease (COPD). Our aim was to evaluate the relation between NT-proBNP level changes and COPD severity.

Methods: We included 140 patients who diagnosed as COPD according to criteria to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2017. Patients were separated as four groups: GOLD stage 1 (FEV1 >= 80%), GOLD stage 2 (50% < FEV1 < 80%), GOLD stage 3 (30% < FEV1 < 50%), GOLD stage 4 (FEV1 < 30%).

Results: GOLD stage I, II, III, and IV groups had 40, 26, 58, and 16 patients, respectively.

NT-proBNP, blood urea nitrogen, and aspartate aminotransferase levels increased significantly with the advancing of GOLD stage and stage 4 patients had the most increased levels (p<0.05 for all, Table 1). Other demographic properties were similar between groups.

The count of patients with NT-proBNP level >125 pg/mL was significantly increased with the advancing of GOLD stage. All patients in stage 4 had NT-proBNP level >125 pg/mL.

Conclusions: NT-proBNP level increases in COPD patients. Increased NT-proBNP is closely related to the severity of COPD. In patients with moderate or severe COPD, it should be kept in mind that NT-proBNP levels are significantly increased.

Table 1. Clinical, laboratory, and demographic variables in patients with different COPD stages

	Stage 1 n=40	Stage 2 n=26	Stage 3 n=58	Stage 4 n=16	p
Age (years)	63.2 ± 7.2	64.5 ± 6.9	63.9 ± 7.3	64.8 ± 4.51	0.430
Gender (male/female)	36/4	24/2	54/4	14/2	0.655
Systolic blood pressure (mmHg)	121 ± 7.7	120 ± 8.7	122 ± 9.9	122 ± 5.1	0.089
Diastolic blood pressure (mmHg)	78.2 ± 5.3	77.5 ± 8.0	79.9 ± 7.4	79.8± 4.6	0.071
Pulse (bpm)	74.9 ± 6.6	75.9 ± 5.9	73.9 ± 9.1	78.6 ± 13.1	0.223
BMI (kg/m2)	27.4 ± 2.9	29.7 ± 5.5	27.2 ± 6.7	25.1 ± 5.5	0.062
Hemoglobin (g/dL)	12.9 ± 1.55	12.7 ± 1.03	13.0 ± 1.55	12.5 ± 2.21	0.369
White blood cell (x103/μL)	9.2 ± 1.47	10.4 ± 2.75	10.3 ± 2.71	11.4 ± 3.0	0.123
AST (u/L)	24.6 ± 12.2	25.6 ± 20.4	36.6 ± 15.7	35.5 ± 18.7	0.019
ALT (u/L)	22.5 ± 12.4	24.8 ± 15.1	28.0 ± 18.6	25.0 ±12.6	0.456
BUN (mg/dL)	29.5 ± 9.0	31.9 ± 8.96	29.5 ± 7.2	43.6 ± 16.8	<0.001
Creatinine (mg/dL)	0.75 ± 0.19	0.72 ± 0.12	0.76 ± 0.20	0.85 ± 0.30	0.087
NT-proBNP (pg/mL)	102 ± 68	190 ± 147	215 ± 102	334 ± 178	<0.001
NT-proBNP > 125 pg/mL, n (%)	10 (%25)	10(%38)	29 (%50)	16 (%100)	<0.001

Other

OP-286

The effect of hydroxychloroquine and azithromycin on the QTc interval in patients with Covid-19

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Background and Aim: The exact treatment method of Covid-19 has not been found so far. However, some small-scale studies have shown the potential benefit of hydroxychloroquine / chloroquine (hydroxychloroquine treatment in our country) and azithromycin in Covid-19 treatment. It is known that these drugs (alone or combined use) may increase the risk of malignant arrhythmia such as torsades de pointes (polymorphic ventricular tachycardia) with prolonged QT interval. There is no data showing the effect of Favipiravir on cardiac arrhythmia. In this study, we aimed to investigate the effect of hydroxychloroquine and azithromycin on the QTc interval in patient with Covid-19.

Methods: Patients who hospitalized in our center between March and May 2020 for Covid-19 and treated with recommendations of Republic of Turkey Ministry of Health SARS-CoV-2 guide included in our study. The data of 142 consecutive patients who received hydroxychloroquine and / or azithromycin treatment were retrospectively analyzed and the QTc interval in ECG was calculated with the Bazzet formula. Patients who were already using hydroxychloroquine (i.e. because of autoimmune disease etc), hypersensitivity to hydroxychloroquine and / or azithromycin and patients who use drugs other than hydroxychloroquine and / or azithromycin that can prolong the QT interval were excluded. Patients with baseline QTc 500 milliseconds (msec) or longer were also excluded from the study. According to the protocol, ECG was performed all patients before treatment, and ECG controls were performed on the 1st, 3rd and 5th days of the treatment.

Results: The mean age of the study population was 46.9±17.3. A total of 142 patients (50.7% male and 49.3% female), received hydroxychloroquine therapy, 36 patients (25%) received hydroxychloroquine monotherapy, while 106 patients (75%) received hydroxychloroquine and azithromycin combination therapy (Table 1). Majority of the patients were in sinus rhythm (%95,1) with mean baseline heart rate of 81.9±14.7 beats per minute. Mean baseline QTc values of 142 patients were 417.3±24 msec, ranging between 356-486 msec. There were no significant differences between the baseline, 1st, 3rd and 5th day's QTc values of two groups. (p>0.05). When each groups were evaluated for QTc prolongation during the therapy period, it was observed that the baseline QTc interval was significantly prolonged with treatment in both the hydroxychloroquine group and the hydroxychloroquine + azithromycin group (Table 2) (p<0.05). There were no patients required discontinuation of these medications, no malign arrhythmia and no arrhythmogenic deaths due to QTc prolongation (Table 3).

Conclusions: Although treatment-related QTc prolongation is observed in our study population, no malignant arrhythmia was observed. Close monitoring of the treatment process by cardiologist and the predetermination of patients with long onset QTc distances are considered to be the most important factor in the safe management of the treatment.

Table 1.

TREATMENT	N = 142
Hydroxychloroquine	36 (%25)
Hydroxychloroquine + Azithromycin	106 (%75)

Table 2.

	QTc Measurements in Patients Treated with HCQ (N=36)	QTc Measurements in Patients Treated with HCQ/AZM (N=106)
Baseline QTc (msec)	418,4 ± 25,4	416,9 ± 23,7
Day 1 QTc (msec)	424,6 ± 26,0	422,2 ± 25,8
Day 3 QTc (msec)	427,5 ± 26,5	427,4 ± 25,0
Day 5 QTc (msec)	434,7 ± 26,4	435,8 ± 24,9

Table 3.

ECG Features	N = 142
Sinus Rhythm	135 (%95,1)
Atrial Fibrillation	7 (%4,9)
Baseline Heart Rate	81,9 ± 14,7
Hydroxychloroquine ± azithromycin discontinued due to QTc prolongation	0
Torsade de pointes	0
Arrhythmogenic death	0

Other

OP-287

The effects and reliability of the hydroxychloroquine-azithromycin combination on the cardiac conduction system in patients with Coronavirus disease 2019

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Background and Aim: The COVID-19 virus has influenced the whole world since late 2019 and has affected millions of people. The combination of hydroxychloroquine (HQ) and azithromycin (AZ) has entered the protocols worldwide to reduce virus replication and take advantage of its immunomodulatory effects.The frequency of QTc prolongation in combinational drug use, and its effect on the primary endpoint, as well as the predictive values of QTc prolongation are not clear.

Methods: The study was designed as a single-center, retrospective study. 135 patients who received hydroxychloroquine, azithromycin and oseltamivir for suspected/definitive COVID-19 with viral pneumonia were examined.

Results: The mean age was 55.6±19.1 years and 61 (45%) patients were female. According to the initial ECG values, the QTc1 value was found to be 422.44±35.72 ms, while the QTc2 value was 446.91±35.72 ms ($p<0.001$). The ECG evaluation after medication use indicated that the number of patients with a QTc value >500 ms was 9 (6.6%). The number of patients with prolongation in QTc values >60 ms was 11 (8.1%). The sum of frequency of prolongation in QTc was 16.2% in intensive care unit patients, when the frequency was 1.5% in low-risk patients in the inpatient unit. An elevation in troponin values >14 ng/L and a low GFR are predictors for QTc prolongation. None of these patients developed a malignant arrhythmia or sudden cardiac death.

Conclusions: Hydroxychloroquine and azithromycin combinations used in COVID-19 patients cause a prolongation in the QTc. The incidence of prolongation in QTc varies according to the comorbid characteristics and clinical status of the patients. Before starting hydroxychloroquine and azithromycin, the risk factors and clinical status of the patients should be well evaluated.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PB-01

Evaluation of demographic and clinical features of patients with pre-operatively long QT patients

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Background and Aim: Long QT syndrome (LQTS) is a rare syndrome that may present ventricular arrhythmia attacks and may be presented in the electrocardiogram (ECG) with prolonged QT interval and T wave abnormalities. Prevention of arrhythmic complications is of great importance in the anaesthesia induction and maintenance of these patients. However, in routine pre-operative evaluation, long QT patients can often be overlooked. In this study, we evaluated the frequency of LQTS, the demographic characteristics of patients, and the reasons for requesting cardiology consultations in pre-operative ECGs.

Methods: The pre-operative ECGs of patients scheduled for elective operation in our hospital between March 1, 2019 and March 1, 2020 were analysed retrospectively. Patients whose QTc duration was ≥480 ms in women and ≥460 ms in men were defined as LQTS. Pre-operative outpatient clinic data and consultation data of cardiology were evaluated using the protocol numbers of these patients. The demographic and clinical features of the patients were analysed.

Results: Among the ECGs taken in the pre-operative period, LQTS was detected in 45 patients, 13 women and 32 men. It was found that only n=25 (55.6%) patients were requested for cardiology consultation. When the reasons for the request for cardiology consultation were analysed, it was observed that the reason for consultation was the most frequent reason for organizing antiaggregant treatment and for examination of acquired heart diseases due to advanced age (n=7, 28% and n=6, 24%, respectively). It was observed that no patient was requested for a cardiology consultation due to LQTS.

Conclusions: In the analysis, it was found that the rate of be requested a cardiology consultation was relatively low in patients with LQTS, and consultation was requested from patients with elderly and low effort capacity, who were generally followed by the cardiology clinic, under anti-aggregate - anticoagulant treatment. Since LQTS is a situation that can be overlooked especially when attention is not paid, there is a need for increased attention.

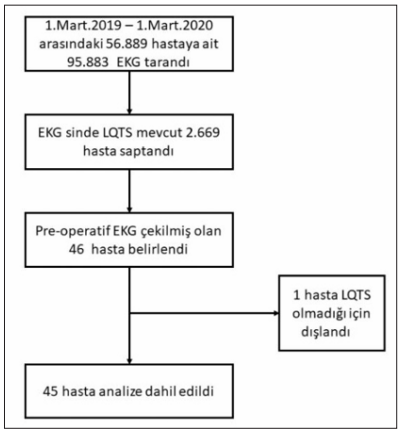

Figure 1. Patient enrollment.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PO-01

Distal esophageal spasm: An overlooked complication of catheter ablation of atrial fibrillation

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Catheter ablation of atrial fibrillation (AF) has recently become a mainstay in the management of symptomatic AF patients. However, this method is not free from complications. Thermal injury during radiofrequency or cryoenergy delivery can lead to esophageal damage. Although atrioesophageal fistula (AEF) is the most dreaded and life-threatening complication, distal esophageal spasm and Jackhammer esophagus can also be associated with similar symptoms in the post-ablation period. We report a case of 42 year-old-male patient with symptomatic paroxysmal atrial fibrillation. Catheter ablation with cryoballoon catheter was planned. Cryoballoon ablation procedure was performed using a 28-mm cryoballoon (Arctic FrontTM Medtronic Cryocath and Aortic Front Advance) with cryothermic energny delivery for 240 seconds per application for each pulmonary vein (PV). Exit and entrance block of all PVs were confirmed by pacing maneuvres. One month after the cryoablation procedure, the patient presented with dysphagia and chest pain. The initial work-up targeted elimination of severe complications such as acute coronary syndrome, AEF and PV stenosis. Electrocardiogram revealed sinus rhythm and no signs of ischemia. Troponin levels were within normal range. Computed tomography scanning with oral and intravenous contrast was performed and AEF and PV stenosis were ruled out. The patient was already on proton-pump inhibitor treatment but had difficulty in swallowing even liquids. An upper gastrointestinal endoscopy was planned and revealed antral gastritis with normal lower esophageal sphincter function. Implementation of a high-resolution esophageal manometry showed repetitive and high-amplitude contractions (1289 mmHg.s.cm) with decreased distal latency (4.4 seconds) in the distal esophageal segment (Figure 1). Therefore a diagnosis of distal esophageal spasm was established and the patient was treated with peppermint oil and calcium-channel blocker. Catheter ablation of atrial fibrillation (AF) has recently become a mainstay in the management of symptomatic AF patients. However, this method is not free from complications. Thermal injury during radiofrequency or cryoenergy delivery can lead to esophageal damage. Although atrioesophageal fistula (AEF) is the most dreaded and life-threatening complication, distal esophageal spasm and Jackhammer esophagus can also be associated with similar symptoms in the post-ablation period. Although overall complication rate is low, some uncommon complications may be overlooked in the post-ablation course. Following elimination of severe complications such as AEF and PV stenosis, one should always keep distal esophageal spasm and Jackhammer esophagus in mind during assessment of patients with chest pain and dysphagia.These unfamiliar antities to electophysiologists significantly differ from gastroesophageal reflux disease in terms of diagnosis and management.

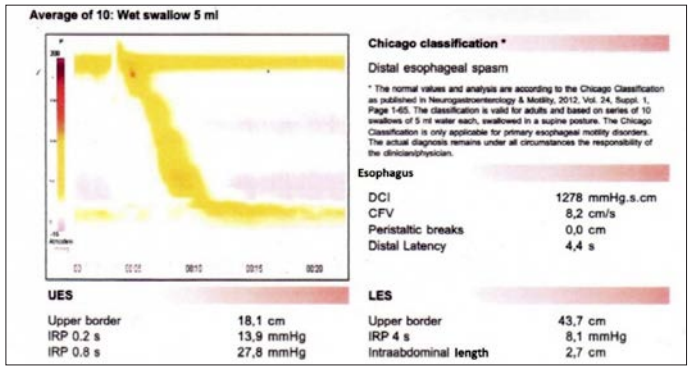

Figure 1. High-resolution manometry trace obtained 1 month after catheter ablation with cryoballoon. Distal contractile integral was measured as 1278 mmHg.s.cm consistent with distal esophageal spasm while values over 8000 mmHg.s.cm are consistent with Jackhammer Esophagus.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PO-02

Naproxen induced Kounis syndrome present with atrial fibrillation

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Allergic vasospastic angina (kounis syndrome) allergy is defined as acut coranary events associated with anaphylaxis or anaphylactoid reactions.. Although Kounis syndrome is defined by acute coronary syndrome, it has been observed that it may be associated with atrial fibrillation. In this case, we presented a case of kounis syndrome developing atrial fibrillation after the use of Naproxen. A 32-year-old male patient presented with the emergency department because of itching, rash and redness, cold sweating, shortness of breath, palpitations after taking naproxen medication due to headache complaints. He had no known systemic disease or prior drug use. Physical examination findings were as follows: cold, sweaty, blood pressure of 90/60 mm Hg, and pulse rate of 150 bpm and pulse was arrhythmic. Cardiac troponin-T(hs-cTnT) levels were <1.5 ng/l (normal range, 0.–0.19 ng/l), and mass ProBNP 284 pq/ml (0-125 pq/ml). After isotonic sodium chloride infusion, dexamethasone and phenyramine, all symptoms of the patient regressed except palpitations.Acute atrial fibrillation was detected on ECG (Figure 1). Echocardiographic findings were found within normal limits. Acute coronary syndrome was excluded due to the absence of chest pain, high cardiac enzyme levels, and no ischemic change in ECG. It was evaluated for medical cardioversion. After the amiadorone infusion was applied, normal sinus rhythm was achieved in the patient. Anaphylaxis induced atrial fibrillation was described for the first time in a report published nearly 50 years ago. The etiology of atrial fibrillation was connected a direct antigen antibody myocardial reaction, induced by either mediator released during anaphylaxis, pharmaceutical agents such as epinephrine used for treatment, anoxia, pre existing heart disease, or a combination of several factors. Three subtypes of Kounis syndrome have been described. Kounis syndrome Type 1 is the most common variant and is the group of patients with normal coronary arteries on angiographic imaging. Tip 2 is associated with atheromatous plaque, damage, and coronary vasospasm in the coronary arteries. Tip 3 is associated with stent thrombosis. Clinical symptoms of Kounis syndrome include chest pain, dyspnea, syncope, nausea, itching, and urticaria. Symptoms such as hypotension, sweating, pallor and bradycardia are accompanied. There are also electrocardiographic findings showing myocardial ischemia, arrhythmias and conduction disorders. Therefore, in patients with acute allergic reactions, the development of chest pain can be explained by the coronary arterial spasm mechanism provoked by histamine release, which creates the Kounis syndrome. Our case is Kounis syndrome developing atrial fibrillation due to anaphylaxis after drug use with its clinical history and electrocardiographic findings. Kounis syndrome should be kept in mind in cases of anaphylaxis accompanied by palpitations.

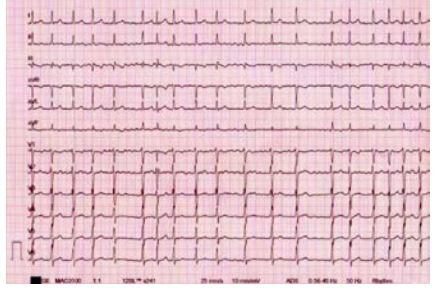

Figure 1. Atrial fibrillation.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

PO-03

Ictal asystole lasting for 27 seconds treated with pacemaker implantation

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Ictal asystole is defined as absence of cardiac electrical activity lasting for more than 4 seconds during seizure in patients with epilepsy. It is associated with morbidity and postulated to be related with sudden unexpected death in epilepsy (SUDEP).

Case Report: A 67-year-old right-handed male patient with a history of temporal-lobe epilepsy diagnosed 19 years ago suffered syncopal seizure episodes once in every 2-3 months so far. His first episode had been a collapse following a stressful event. His subsequent seizures were typically collapsing and no repetetive motion or myo-clonic jerks. The patient noted an aura of warmness and nervousness seconds before seizure. His last seizure happened in mosque after typical aura and over sweating followed with fatigue and confusion. He recovered his consciousness within 1 minute. Witnesses had described his eyes looking upside. He was on oral levetiracetam 500 mg for years. The patient also described exertional angina he noted in the last months. In order to better delineate his seizures and treatment options, the patient was admitted to the video EEG monitoring unit for 48 hours. In the first afternoon the patient went asleep and several minutes later an evolving electrographic seizure without any certain body movements was observed. The seizure originated from right posterior temporal region. Twelve seconds after electrographic alterations, gradual PR prolongation was followed with an asystole episode lasting for 27 seconds. During the asystole period, no body movements were observed but a slight initial head extension in lateral position. A few junctional beats were followed by sinus beats although epileptiform discharges continued for 5 seconds. The patient did not wake up after asystolic episode and kept sleeping for 30 minutes more. An ECG obtained after ictal asystole showed normal sinus rhythm. Echocardiography revealed normal ejection fraction and no structural abnormalities. The patient's medical condition and records were thoroughly discussed in a council. Taking the long duration of ictal asystole and frequency of his past episodes into account, the council decided for pacemaker implantation. Before the implantation procedure, the patient underwent coronary angiography for his angina and a critical LAD stenosis was treated with stent implantation. Several days after stent implantation, the patient received DDDR pacemaker implantation with MRI compatible features. The patient had no seizure recurrence during follow-up and no ventricular pacing was noted in his subsequent pacemaker interrogations.

Discussion: Cardiac rhythm disturbances during epileptic seizures are common and generally considered to be benign in nature. Ictal tachycardia is very frequent (70-90%), however, ictal bradycardia affects less 5% of patients with epilepsy. Ictal asystole is extremely rare (0.3-0.4%) and is not a benign condition. Documentation of ictal asystole can be lifesaving via pacemaker implantation.

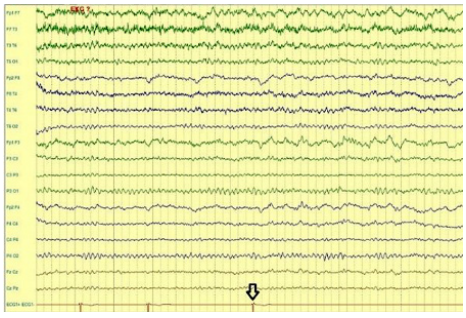

Figure 1.

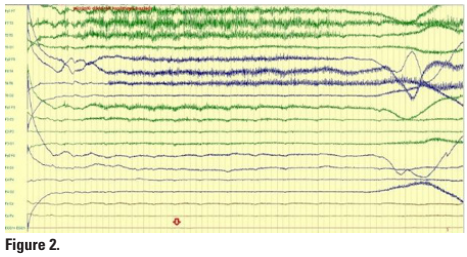

Figure 2.

Other

PB-02

The relationship of blood pressure with heart rate variability and heart rate turbulence in type 2 diabetes mellitus patients

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Background and Aim: Heart rate variability (HRV) evaluates heart rate regulation by looking at parasympathetic and sympathetic divisions of the autonomic nervous system to detect changes in the sinus rate. Another example of a method for assessment is heart rate turbulence (HRT), which evaluates the physiologic changes that occur in sinus rate following ventricular premature beats (VPBs) to identify baroreflex sensitivity. Numerous studies have indicated there to be a relationship between impaired HRT- HRV and mortality in cardiovascular diseases. Type 2 diabetes mellitus (T2DM) has become a significant global health care problem and its reported incidence is increasing at an alarming rate. When hypertension is combined with diabetes, the risk of CVD is even greater. Despite adequate glycemic and blood pressure control, diabetic hypertensives remain at increased cardiovascular risk compared to normotensive diabetic patients. İn this study, we aimed to evaluate the relationship between cardiac autonomic functions and blood pressure using 24-hour HRV and HRT analysis in diabetic patients.

Methods: The study was designed as a cross-sectionally study, and a total of 108 patients with T2DM, 50 normotensive and 58 hypertensives, were enrolled in the study. All patients underwent a twenty-four-hour course of Holter monitoring, after which all Holter recordings were examined and any artifacts the program indicated to be VPBs were rejected. Statistical analyses were conducted by using the SPSS software version 23. A P-value <0.05 was considered statistically significant.

Results: There was no significant difference between the patient and control groups in terms of their ages, gender, smoker, have a chronic disease and BMI values. All variable of patients were shown in Table 1. The HRV parameters of SDNN (103.7±2.5, 113.6±1.68, p=0.002), SDANN (95.1±2.3, 103.2±2.1, p=0.014) were significantly lower in the hypertensive T2DM group.

Conclusions: Disorders of autonomic balance developing in diabetic patients in the form of a marked decrease in parasympathetic tone and a relative increase in sympathetic tone are among the known features of diabetes. The development of cardiac autonomic neuropathy is a poor prognostic indicator in diabetic individuals, causing arrhythmia, silent infarction and sudden death. Considering our study results, a significant deterioration in HRV parameters is noticeable in asymptomatic hypertensive T2DM individuals. In our study, both HRV and HRT analysis were used together. We have found that HRV parameters, only SDNN and SDANN, are also corrupt. Patients suffering from both diabetes and hypertension are at the highest risk of reduced heart rate variability. Early assessment of the autonomic nerve function may be suggested in diabetic patients with hypertension. Although a healthy control group was not evaluated, the results suggest that systemic arterial hypertension further impairs HRV in diabetic patients.

Table 1. Baseline demographic and clinical characteristics of normotensive and hypertensive Type 2 DM patients

Variables	HT-T2DM+ (n:50) Mean ± SD	HT+T2DM+ (n:58) Mean ± SD	P value
Male n (%)	26 (%52)	25 (%49)	0.356*
Smoker n (%)	13 (%26)	14 (%24.1)	0.824*
Coronary artery disease	11 (%24.1)	16 (%24.1)	0.119
Chronic renal failure	6 (%12)	10 (%17.2)	0.445
Chronic obstructive lung disease	7 (%14)	8 (%13,8)	0.975
Age (years)	54.1±0.76	53.5±0.83	0.589
Body mass index (kg/m²)	27.8±0.19	28.3±0.28	0.734
Fasting glucose (mg/dl)	162.1±2.96	161.4±2.59	0.872
HbA1c (mg/dl)	7.84±0.81	7.89±0.12	0.788
Creatinine (mg/dl)	0.83±0.03	0.86±0.21	0.390
Total Cholesterol (mg / dl)	156.3±4.1	172.7±6.2	0.033
Triglyceride (mg / dl)	265.9±4.5	258.3±10.9	0.545
HDL Cholesterol (mg/dl)	45.1±0.71	42.2±0.66	0.006
LDL Cholesterol (mg/dl)	139.1±4.5	141.8±4.5	0.137
OAD n (%) OAD+İnsüline n (%) İnsüline n (%)	38 (%76) 8 (%16) 4 (%8)	46 (%79.3) 9 (%15.5) 3 (%5.2)	0.830
Hemoglobin (g/dl)	13.8±0.17	13.9±0.2	0.832
Neutrophile count (x103/uL)	5.52±0.22	5.63±0.23	0.729--
Platelet count (x103/uL)	259.7±8.4	262.4±9.8	0.921
Monocyte count (x103/uL)	0.61±0.03	0.68±0.04	0.893
Lymphocyte count (x103/uL)	2.09±0.09	2.15±0.09	0.657

OAD: oral anti-diabetic.

Table 2. Comparison of heart rate turbulence and heart rate variability values of study groups

Variables	HT-T2DM+ (n:50) Mean ± SD	HT+T2DM+ (n:58) Mean ± SD	P value
Heart rate, beats/min	78±1.07	76±0.85	0.658
SDNN, ms	113.6±1.68	103.7±2.5	0.002
SDNN index, ms	44.04±1.6	40.79±1.6	0.166
SDANN, ms	103.2±2.1	95.1±2.3	0.014
RMSSD, ms	26.8±0.94	24.2±1.1	0.072
pNN50, %	19±1.1	20.4±1.4	0.456
Triangular index	30.6±1.02	29.4±1.01	0.391
Turbulence onset, %	-1.15±0.13	-0.91±0.12	0.202
Turbulence slope, ms/RR	6.35±0.45	5.64±0.42	0.238

SDNN: standard deviation of all NN intervals, SDANN: standard deviation of the averages of NN intervals in all 5 min segments of the entire recording, RMSSD: the square root of the mean of the sum of the squares of differences between adjacent NN intervals, pNN50: the number of pairs of adjacent NN intervals differing by more than 50 ms divided by the total number of all NN intervals.p<0,05 statistical significance.

Other

PO-04

Terrible electrocardiogram artifact; Arterial pulse artifact

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Introduction: Abnormal electrocardiograms which is not depend on the electrical activity of the heart are called artifacts. Artifacts can cause ST segment deviations and it can be easily distinguished, since they often do not mimic a particular cardiac pathology. Common ECG artifacts are tremor, electrical alternans, and misplacement of electrodes. Rarely artifact can mimic a ischemic T wave and ST segment elevation and it can be considered as acute coronary syndrome. We present a rare case of arterial pulse ECG artifact.
Case Report: A 30-year-old male patient with no medical history admitted to the emergency room with a burning in the stomach, which has been relieved by eating. The patient was evaluated in terms of acute coronary syndrome. His cardiovascular system physical examination was normal. Arterial blood pressure was 135/85 mmHg, respiratory rate 13 / min and oxygen saturation was 95% in room air. The patient's first ECG was sinus rhythm with abnormal T waves in DII-DIII-AVF leads and biphasic T waves in precordial leads (Figure 1) but the control ECG was normal (Figure 2). Echocardiography was performed; left ventricular ejection fraction was 65% and there was no segmental wall motion defect. Because of no symptoms of active angina and angina equivalent and normal control ECG (Figure 3); the first ECG was evaluated as artifact. Cardiac enzyme follow-up remained within the normal range. Acute coronary syndrome was not considered.
Discussion: Different diagnosis for ST segment elevation includes acute myocardial infarction, early repolarization, left bundle branch block, acute pericarditis, and hyperkalemia. Özhan et al. presented a patient with "weird, large T waves" on only one ECG trace. They thought that this abnormalities might be related to left ventricular motion. Because abnormal T waves were detected in all leads In addition, artifacts were detected in each cardiac cycle. A fixed match was observed between the maximum amplitude of QRS and each affected lead. This indicates that the artifact is from the cardiac cycle. Aslanger stated that this weird electrocardiogram is an artifact resulting from an arterial pulse beat. While DI lead is normal, abnormal T waves in DII and DIII leads make us consider of the arterial pulse artifact of the left leg ECG artifacts can lead to misdiagnosis, unnecessary tests, and mismanagement of patients. We keep in mind that some ECG anormalities could be artifacts.

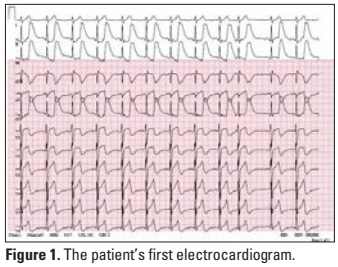

Figure 1. The patient's first electrocardiogram.

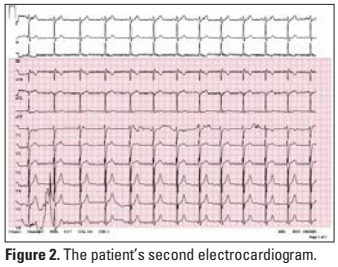

Figure 2. The patient's second electrocardiogram.

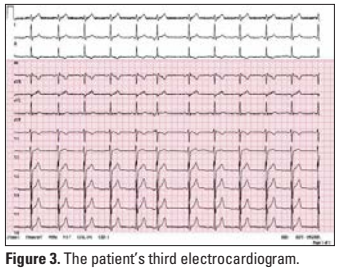

Figure 3. The patient's third electrocardiogram.

Other

PO-05

Brugada syndrome diagnosed with COVID-19 infection

Berat Uğuz, Selma Tiryakioğlu, Mustafa Adem Yılmaz, Selvi Coşar, İsmet Zengin

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The COVID-19 outbreak is a global public health problem, the disease has spread exponentianally since the end of December 2019, when the first cases of SARS-CoV-2 infection were detected in Wuhan, China. Fever is the most common presentation, seen in approximately 88% of cases, cough (68%), vomiting (5%), and diarrhea (3.8%) are the other common symptoms.In this case report we are presenting a 34-year-old male patient who was admitted to the emergency department with fever that had been ongoing for 3 days. Initial diagnostic work up including computerized tomography (CT) scan documented was compatible with viral pneumonia and the 12 lead electrocardiogram (ECG) showed a Brugada -type I pattern,ST segment elevation in the right pericordial leads with no resiprocal changes. The patient had no chest pain, or syncope and family history of sudden cardiac death. Cardiac enzymes were normal; echocardiographic examination demonstrated normal wall motions and there weren't any pericardial effusion. The patient was admitted with a diagnosis of COVID-19 pneumonia, which was affirmed with positive nasopharengeal swab test for COVID-19. Brugada syndrome is a rare disease manifested by ST-segment elevation in the right precordial leads. Fever may cause Brugada-like changes in the ECG. With reduce of the fever, the Brugada-like ECG changes had also disappeared.

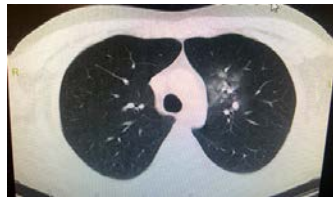

Figure 1. Computerized tomography image.

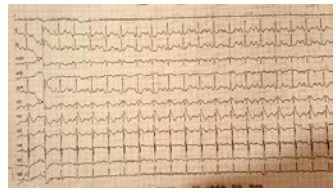

Figure 2. The patient's initial 12-lead electrocardiogram in the emergency department.

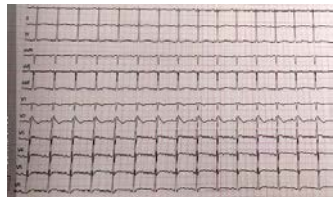

Figure 3. EKG on the second day.

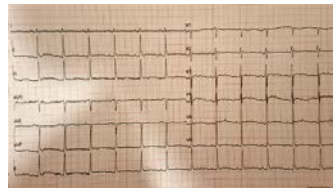

Figure 4. The patient's repeat 12-lead electrocardiogram with resolution of fever.

Other

PO-06

Cardiac and intracranial mass in a thrombophilia patient

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[2]Department of Cardiology, Denizli State Hospital, Denizli

Introduction: Hydatid cyst is a disease caused by Echinococcus granulosus larvae. It is most frequently located in the liver (50-70%) and lungs (20-30%). The spleen, kidney, central nervous system, bone, heart, muscle tissue are the primary locations. Cardiac involvement is rare, but may be mortal, especially if undiagnosed and complicated.
Case Report: A 26-year-old housewife without any additional disease, presented with complaints of weakness in the left arm and leg, and inability to speak. No bleeding was detected on cranial CT. As a result of the examinations, ischemic CVD was diagnosed. A 23x13 mm hyperechogenic mass was detected at the apex of the left ventricle which was considered in favor of thrombus. The presence of vasculitis and Behçet's disease was investigated without any relevant evidence.Protein S deficiency, factor 13 heterozygous mutation, MTHF heterozygous mutation and PAI heterozygous mutation were detected. Considering ischemic CVD and cardiac thrombus, the hematology department decided to prescribe life-long treatment with warfarin. One year later, the patient was admitted to the hospital with the complaint of not being able to speak again. In the brain MRI, it was determined that the lesion in the brain had a cystic appearance (Picture). Possible diagnosis was considered in favor of a hydatid cyst. Intracerebral cystectomy was performed by the neurosurgery department. After the pathology report, the diagnosis of hydatid cyst was definitely confirmed. On thoracic CT, cystic lesions compatible with hydatid cysts were detected at the level of the left ventricular apex and the anterior aspect of the right pulmonary upper lobe. The cyst in the left ventricle was removed by the CVS. Warfarin is not stopped during the follow-up period except preoperatively. He has been taking warfarin for four years and undergoing INR monitoring. All past reports of the patient were examined and it was determined that the first attack was missed hydatid cyst. Diagnoses of ischemic CVD and intracardiac thrombus were excluded in favor of hydatid cyst based on pathology reports. With the decision of the council, the patient's warfarin was stopped. The patient is in stable condition, and has been followed up uneventful for 5 months.
Discussion: Hydatid cyst is an important health problem in our country, especially in the southeastern and eastern regions. Primary cardiac echinococcosis is rare. Its frequency varies between 0.5 and 2% in patients with hydatid cysts. Patients are generally asymptomatic. However, the presence of serious complications that may result in sudden death requires urgent surgical treatment as soon as the diagnosis is made. Cysts may rupture into the intracardiac or intrapericardial area. As a result, anaphylactic shock, embolism, cardiac tamponade, acute pericarditis and chronic constrictive pericarditis may develop. Early recognition and treatment of cardiac echinococcosis is important due to the risk of cyst rupture. The diagnosis of hydatid cyst should be kept in mind especially in endemic areas and should not be omitted.
Conclusion: Cardiac echinococcosis should be considered at the forefront in the presence of a cardiac mass, especially in our endemic regions. In addition, detailed anamnesis of the patients should be taken and their history should be evaluated in detail.

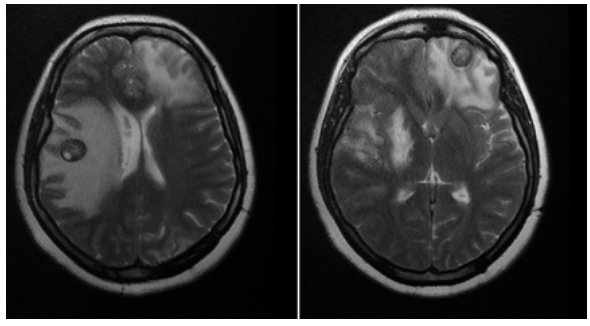

Figure 1. Intracranial cysts.

Interventional cardiology / Cover and structural heart diseases

PB-03

Prognostic impact of chronic obstructive pulmonary disease in octogenarians undergoing transcatheter aortic valve replacement

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Background and Aim: The study aimed to evaluate the prognostic impact of chronic obstructive pulmonary disease (COPD) in octogenarians undergoing transcatheter aortic valve replacement (TAVR).
Methods: A total of 157 octogenarians undergoing TAVR due to severe aortic stenosis were included in this retrospective cohort study. Patients were assigned into two groups based on presence (n=65) and absence (n=92) of chronic obstructive pulmonary disease. Prognostic impact of chronic obstructive pulmonary disease on clinical outcomes and predictors of long term mortality were investigated.
Results: Increased immediate procedural mortality (13.8 vs 4.3%, p=0.042), all cause of early mortality (23.1 vs 5.4%, p<0.001), cardiovascular mortality (15.4 vs 4.3%, p=0.017), major vascular complication (10.8 vs 3.3%, p=0.059) and stroke (9.2 vs 2.2%, p=0.048) were reported in COPD group. Although patients had a similar pre-TAVR NYHA-class (3.14±0.34 vs 3.07±0.32, p=0.18), improvement in post-TAVR NYHA-class (2.82±0.76 vs 2.29±0.74, p<0.05) was more pronounced in non-COPD group. In a univariate regression analysis age and COPD were significantly associated with five-year mortality. Age (HR 1.129, 95% CI 1.011-1.261, p=0.031) and COPD (HR 2.010, 95% CI 1.031-3.919, p=0.041) were found to be significant independent predictors of five-year mortality in a multivariate analysis, after adjusting for other risk.
Conclusions: COPD has a significant prognostic impact on early outcomes and five-year mortality in octogenarians.

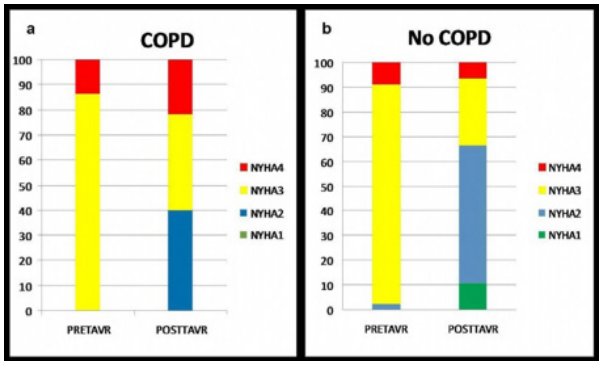

Figure 1. NYHA-class at baseline and 30-days postoperative in patients with **(a)** and without **(b)** COPD. COPD: chronic obstructive pulmonary disease; NYHA: New York Heart Association)

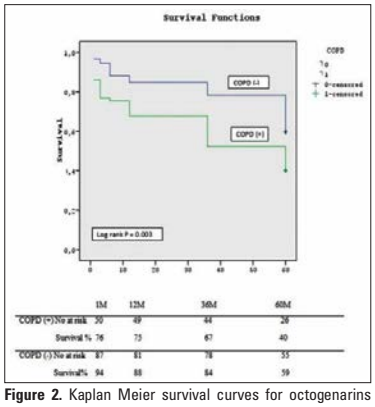

Figure 2. Kaplan Meier survival curves for octogenarins with COPD + vs COPD -.

Table 1. Baseline characteristics of the patients

	COPD (+) (n = 65)	No COPD (n = 92)	p value
Age (years)	85.05 ± 3.77	84.08 ± 2.60	0.201
Gender (male)	35 (53.8%)	49 (53.3%)	0.535
Hypertension	52 (80.0%)	74 (80.4%)	0.946
Coronary artery disease	34 (52.3%)	54 (58.7%)	0.710
Previous MI (<90 days)	6 (9.2%)	3 (3.3%)	0.164
Prior CABG	13 (20.0%)	13 (14.1%)	0.386
Stroke	3 (4.6%)	5 (5.4%)	0.067
Diabetes mellitus	15 (23.1%)	36 (39.1%)	<0.001
Dyslipidemia	4 (6.2%)	22 (23.9%)	<0.001
Smoking	45 (69.2%)	38 (41.3%)	0.002
Peripheral vascular disease	3 (4.6%)	6 (6.5%)	0.737
Logistic EuroSCORE	15.10 ± 5.48	13.89 ± 4.18	0.163

Interventional cardiology / Cover and structural heart diseases

PO-07

Methemoglobinemia due to the use of Prilocaine during the TAVI

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TAVI is becoming an increasingly popular treatment alternative for symptomatic severe aortic valve stenosis with pulmonary comorbidity. In cardiovascular interventions with local anesthesia, methemoglobinemia should be considered in hypoxia despite intubation in patients with low pulmonary capacity. Low methemoglobin level may lead to serious life-threatening hypoxia or death. TAVI is almost all conducted under conscious sedation and local anesthesia. However some patients with severe pulmonary pathologies are rarely need general anesthesia and intubation. We presented a COPD patient who developed methemoglobinemia due to the use of prilocaine as a local anesthetic in the TAVI procedure and was intubated due to sudden pulmonary depression.
Case Report: A 74-years-old woman with known COPD history admitted to clinic with dyspnea, orthopnea. She complained of exertional dyspnea for the last 3 months and was present with severe pulmonary edema. Her NYHA was class III and physical examination revealed 2/6 systolic murmur on aortic area and diffuse rhonchi and prolongation of the expiratory phase. ECG showed sinus rhythm. TTE showed that her LVEF 60%, severe aortic stenosis (AVA:0.59 cm^2) with moderate tricuspid and mitral regurgitation and pulmonary hypertension. Arterial blood sample with 4 lt/h nasal O_2 showed the pO_2 pressure of 59.9 mmHg and the pCO_2 pressure of 34.2 mmHg. The cardiac team decided to TAVI procedure due to low pulmonary capacity. Procedure started with applying local prilocaine hydrochloride 400 mg / 20 mL to both groins. Anesthesia team was intubated due to persistent hypoxia despite increasing O_2 level without any hemodynamic complications. A 26 mm CoreValve-EvolutR was implanted immediately. Transaortic gradient decreased from 77 mmHg to 7 mmHg. The procedure was completed. O_2 saturation by pulse oximetry (SpO_2) was 85% and didn't change after positive-pressure ventilation with a 100% fraction of inspired O_2. She was taken to the ICU, auscultation revealed equal breath sound and chest radiograph disclosed no evidence of pneumothorax. TTE ruled out any mechanical complication. She has central cyanosis and her arterial blood sample had a dark brown color appearance (Fig. 1a). Methemoglobinemia was suspected. Arterial blood sample showed methemoglobin fraction was 17%. She was given 500 mg vitamin C and 1U suspension of erythrocytes until methylene blue was provided. Although the control arterial blood sample methemoglobin fraction decreased by 14%, the patient's hypoxia persisted. Methylene blue (1 mg/kg/dose) was administered IV and her O_2 saturation increased dramatically. The urine color turned blue 30 minutes after IV administration of methylene blue (Fig 1b). Her cyanosis resolved, methemoglobin fraction regressed to 6% in a hour and O_2 saturation increased to 93%. Methemoglobinemia can cause life-threatening respiratory distress even in low levels in low pulmonary capacity as in this case report. It reduces mortality with rapid diagnosis and treatment.

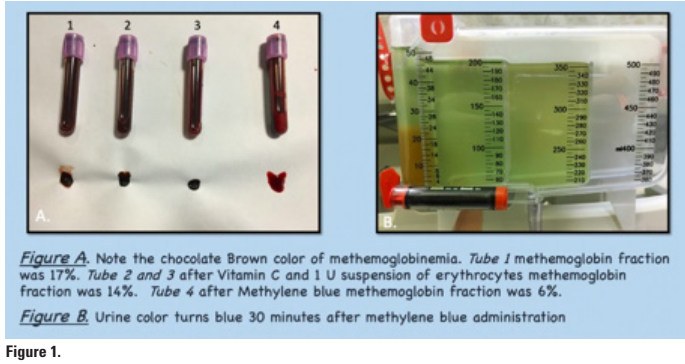

Figure A. Note the chocolate Brown color of methemoglobinemia. *Tube 1* methemoglobin fraction was 17%. *Tube 2 and 3* after Vitamin C and 1 U suspension of erythrocytes methemoglobin fraction was 14%. *Tube 4* after Methylene blue methemoglobin fraction was 6%.

Figure B. Urine color turns blue 30 minutes after methylene blue administration

Figure 1.

Interventional cardiology / Coronary

PO-08

Alveolar hemorrhage following abciximab infusion

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Alveolar hemorrhage following glycoprotein IIb/IIIa receptor antagonist administration is an extremely rare and catastrophic complication and few case is reported in the literature. We describe a case of acute myocardial infarction complicated by diffuse alveolar hemorrhage. A 57 year old man was admitted to our hospital with a diagnosis of anterior ST elevation myocardial infarction. A loading dose of aspirin (300 mg) and ticagrelor (180 mg) was given and emergency coronary angiography was performed. After a bolus dose of unfractioned heparin (60 units/kg) was given the occluded proximal left coronary artery was reopened by percutaneous coronary intervention with a drug-eluting stent in his proximal left anterior descending coronary artery resulting in thrombolysis in myocardial infarction (TIMI) grade 3 flow. Due to high thrombus burden, a boluse dose of Abciximab (0.75 mg/kg) was given and infusion via a vein pathway was scheduled for 12 hours. The patient complained respiratory distress with hemoptysis at the time of 3. hour of abciximab infusion. The haemoglobin level was found to be 11 g/dl and there was no thrombocytopenia. The chest X-ray was unremarkable (Figure 1). The abciximab infusion was then stopped and a chest computed tomography was performed with a sign of alveolar hemorrhage (Figure 2, arrows). The dual antiplatelet therapy (Aspirin/Ticagrelor) was continued. Patient's symptoms and hemoptysis dramatically improved after discontinuation of abciximab. Although we did not apply in this case, early bronchoscopy is a useful tool to confirm the diagnosis and to use a balloon tamponade or iced saline lavage. Acute respiratory distress with hemoptysis following administration of abciximab should alert physicians to the likelihood of alveolar hemorrhage. Although therapy remains supportive with discontinuation of all antiplatelet and anticoagulant agents in stented patients this approach may be harmful in terms of acute stent thrombosis.

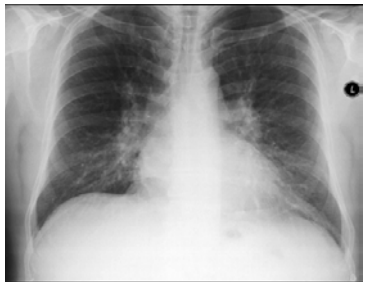

Figure 1. The chest X-ray.

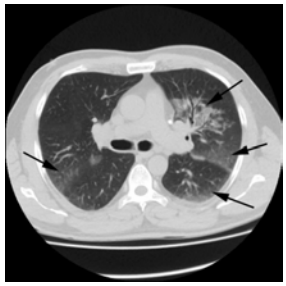

Figure 2. Computed tomography image showing alveolar hemorrhage (arrows).

Interventional cardiology / Coronary

PO-09

A case of coronary artery aneurysm after drug eluting stent implantation

Yavuzer Koza,[1] Oğuzhan Birdal,[1] Hakan Taş,[1] Ferhat Borulu,[2] Bilgehan Erkut[2]

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[2]Department of Cardiovascular Surgery, Atatürk Üniversitesi University Faculty of Medicine, Erzurum

Coronary artery aneurysms after coronary intervention are rare with an incidence of 0.3% to 6.0%. Most of them are pseudoaneurysms rather than true aneurysms. A 38-year-old man underwent percutaneous coronary intervention due to anterior myocardial infarction with an evorulimus eluting stent (Figure 1a, b). Ten months later, he suffered from a non ST elevation myocardial infarction. Coronary angiography showed an aneurysm extending to the left main coronary artery at the site of previous stent deployment (Figure 2a, b). Because aneurysm developed in a previously stented segment, we decided to surgery. He underwent on-pump coronary artery bypass surgery with a left internal mammarian artery graft to the proximal left anterior descending artery and a saphenous vein graft to the left circumflex artery. Aneurysm was proximally ligated and plicated (Figure 3). The postoperative course was uneventful and he was discharged 11 days after the operation. Coronary aneurysm formation after stenting can be associated with high pressure inflations, residual microdissection, deep arterial wall injury, coronary stent infection and hypersensitivity reactions to the drug or polymer. In our case, aneurysm could be arised from aggressive pre and postdilatation with the increased local drug delivery. Although interventional treatments such as stent grafts or coils have been proposed, a treatment strategy has not been established for this rare clinical entity. Treatment strategy should be tailored on the basis of the aneurysm size, expansion history, pathophysiology and symptoms of the patient.

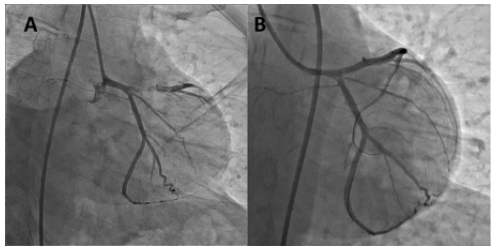

Figure 1. Angiographic image showing the proximal left anterior descending artery lesion with a high thrombus burden **(a)**, final result after stent deployment **(b)**.

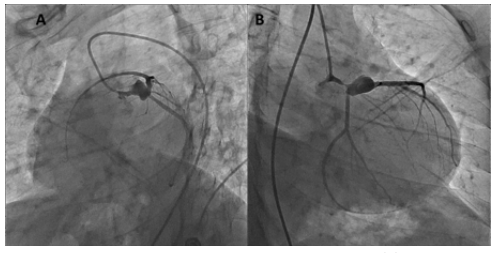

Figure 2. Angiographic image showing aneurysm on spider **(a)** and left coronary **(b)** views.

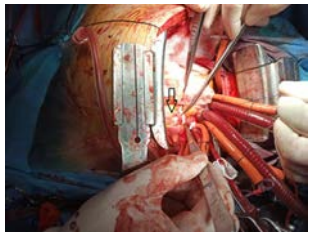

Figure 3. Intraoperative image of aneurysm (arrow).

Interventional cardiology / Coronary

PO-10

A bad surprise in the catheter lab: stent slippage

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[2]Department of Cardiology, Şırnak State Hospital, Şırnak
[3]Tekirdağ State Hospital, Tekirdağ

Introduction: A 55-year-old male patient, for whom the decision of surgical revascularization was made as a result of coronary angiography performed with a diagnosis of USAP, refused surgery, and the patient was informed about all the relevant risks and the decision of staged percutaneous revascularization was made. The procedure of removing the stent away from the coronary artery by inflating a small balloon distal to the stent that was slipped off from its balloon in the area of critical stenosis during Rca revascularization was explained to the patient.

Case Report: A 54-year-old male patient with known diabetes mellitus was admitted with the diagnosis of unstable angina pectroris. Echocardiography showed 55% EF with mild mitral and tricuspid insufficiency. Laboratory values were unremarkable. Physical examination findings were within normal limits (BP 120/80 mmHg; pulse rate: 70/min: body temperature: 36.5°C). Results of coronary angiography performed through right femoral artery using a 6 F sheath were as follows: LMCA: normal; LAD proximal 50-60% and 60-70% sequential stenosis, 70% in the mid segment; D2 ostial stenosis of 70-80% : diffuse plaque formation and 70% stenosis in S2 lateral branch; LCX: totally occluded after OM artery: antegradely filling RCA: 90% stenosis in mid segment (Figures 1,and 2). Decision: Surgical revascularization was planned based on the decision of the council (but the patient did not accept surgical revascularization). All risks were explained to the patient and the decision to perform PCI through LAD, CX and RCA with staged percutaneous revascularization was made. A Judkins right (Jr) guiding catheter was placed in RCA. The 90% stenosis in the mid segment of the RCA was passed with a floppy wire. The lesion could not be negotiated with a 3.0x16 mm DES. Therefore the decision to predilate the stenosis with a balloon was made. However, while the stent was withdrawn, it was observed that it slipped off from its balloon. The wire in the RCA was also retracted into the catheter Since we did not have a coronary snare, we tried to pass a floppy wire through the stent again. However, we could not pass the floppy wire through the stent, only it could be advanced under the strut. Then, the pt2 wire was passed through the stent. The 1.0x10 mm balloon was inflated distal to the stent and the slipped stent was placed inside the catheter (Figure 3). The catheter with the slipped stent at its tip was taken away from the femoral sheath (Figures 4, 5, and 6). Afterwards, the catheter system was changed and the stenosis was predilated with a 2.0 x 10 mm balloon. A 3.0x16 mm DES was implanted in the dilated area. Full patency was achieved (Figure 7). Since the procedure was prolonged, an elective PCİ was planned for LAD and LCX. With staged revascularization, PCI for LCX, and LAD was performed in two separate sessions. Full patency was achieved (Figures 8, and 9). The decision for medical follow-up was made.

Discussion: Stent slippage/migration and embolization is a rare complication that may pose an additional risk to the patient. Various methods have been described for percutaneous removal of the embolized stent. The choice of method to be applied depends on the clinical situation, operator's experience and available laboratory equipment. There is no need for treatment for stents embolized to the periphery / tips However various methods have been described to pull out the slipped stents as inflation of a small balloon distal to the stent, grasping the stent with a snare, or myocardial biopsy forceps, passing a second wire through the stent to withdraw the stent,and crushing the stent with another stent. Surgical option should always be kept in mind for patients in whom with percutaneous interbention methods failed.

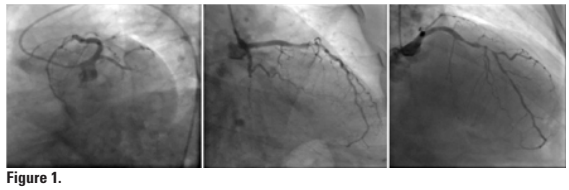


Figure 1.

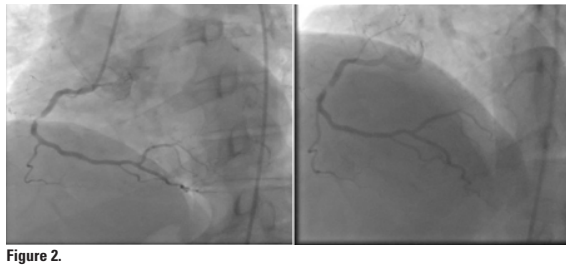


Figure 2.

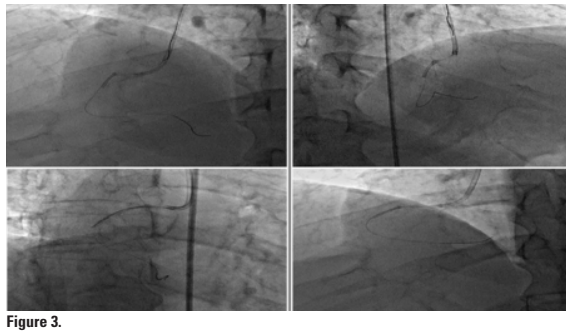


Figure 3.

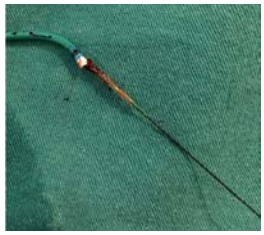


Figure 4.

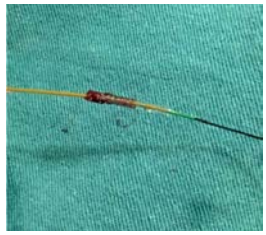


Figure 5.

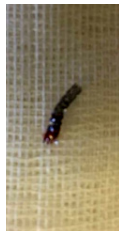


Figure 6.

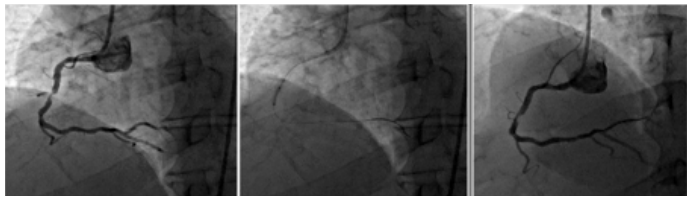


Figure 7.

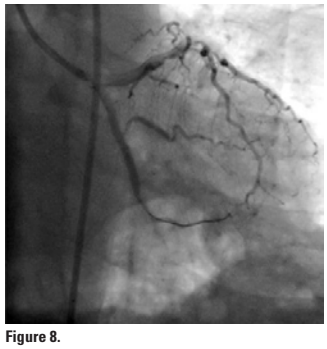
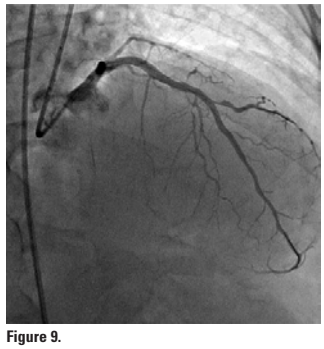


Figure 8.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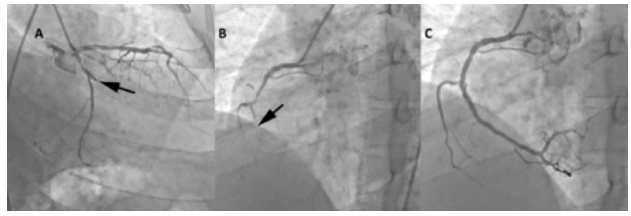

Figure 1. Angiographic image showing the simultaneous occlusion of the of the left circumflex artery (arrow) **(a)** and of the right coronary artery (arrow) **(b)**. The TIMI 3 flow in the right coronary artery **(c)**.

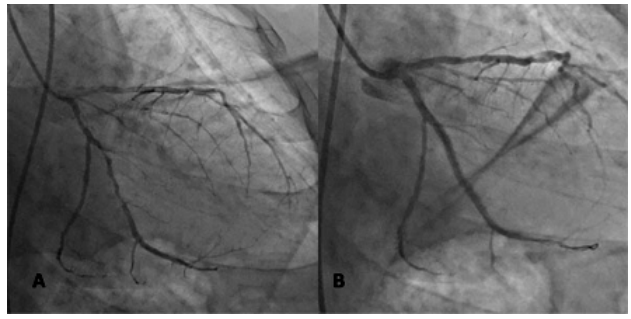

Figure 2. Angiographic view showing the left circumflex stenosis **(a)** and the final result after percutaneous intervention **(b)**.

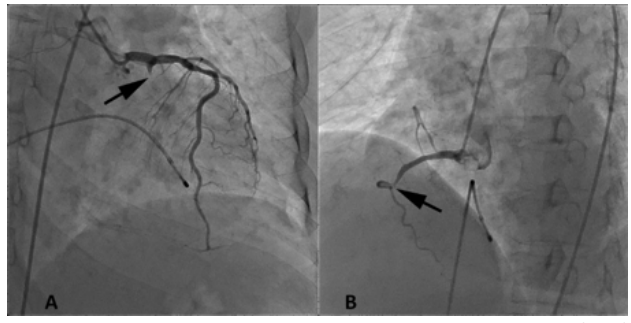

Figure 3. Angiograohic image showing simultaneous acute thrombosis of the left circumflex (arrow) **(a)** and of the left right coronary artery (arrow) **(b)**.

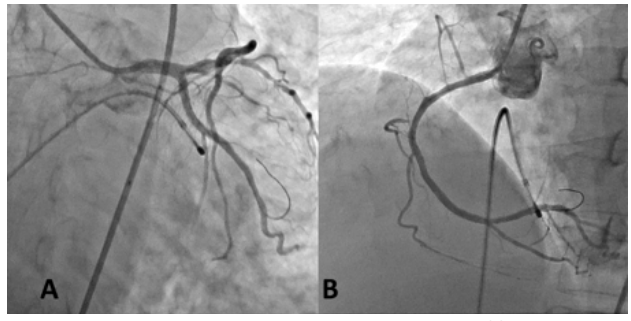

Figure 4. Angiographic image showing the final result of the left circumflex **(a)** and right coronary arteries **(b)** after percutaneous intervention.

Interventional cardiology / Coronary

PO-11

Myocardial infarction does not always present with single culprite lesion: Two cases of Simultaneous multi-vessel coronary thrombosis

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Myocardial infarction with ST segment elevation is a life-threating condition that can result with death causing myocyte necrosis, heart failure and cardiogenic shock. In its classical form, rupture of an atheromatous plaque leads to thrombosis of an epicardial coronary artery. Therefore, in the most of the patients the culprit lession is single. Multivessel coronary occlusion is a rare condition in patients with ST segment elevation myocardial infarction. The underlying pathology has not been clarified sufficiently. This is a life-threatening condition and there is no consensus about the management of these patients. Simultaneous thrombosis affecting more than one coronary artery has been reported to occur in about 4.8% of the cases at the time primary percutaneous coronary intervention (PCI). In this report, we describe two cases of ST elevation myocardial infarction with multivessel coronary occlusion.

Case 1: A 36 year-old man was admitted with the diagnosis of inferior ST elevation myocardial infarction. Coronary angiography showed simultaneous acute thrombosis of the right coronary artery (RCA) and of the left circumflex (Cx) artery (Fig. 1a, b). There was no reciprocal depression in V1-V3 leads. The Cx artery stenosis was accepted as chronic total occlusion and it was decided to perform PCI to the RCA. The occluded RCA was reopened by drug eluting stent implantation (Fig. 1c). The hospital course was uneventful and he was discharged on day 3. After 7 days, he presented to hospital with the chest and back pain. The electrocardiogram (ECG) showed isolated posterior myocardial infarction. Coronary angiography revealed a 95% stenosis in the left Cx artery with TIMI 3 flow. PCI was performed with successfully (Figure 2a, b).

Case 2: A 51 year-old woman with no cardiovascular disease referred to emergency department with typical chest pain.The ECG showed >1mm ST segment elevations in D2, D3 and aVF derivations, >2 mm ST segment depressions in V1-V3 derivations and third degree atrioventricular block. Coronary angiography showed simultaneous acute thrombosis of the RCA and of the left Cx artery (Figure 3a, b). The right coronary and the left Cx arteries were reopened by PCI (Figure 4a, b). PCI of the culprit lesion is a commonly accepted approach in acute myocardial infarction patients unless hemodynamic instability is involved or a sign of residual ischemia is found. Due to high mortality rate of simultaneous thrombosis early diagnosis and effective reperfusion of culprit lesions are mandatory.

Hypertension

PB-04

What is good for hypertensive patients: Presence or absence of presystolic wave

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Background and Aim: The absence of presystolic wave in the hypertensive patients with preserved left ventricular ejection fraction is an indicator of subclinical myocardial damage. The hypertensive patients who exhibit an absence of presystolic wave should be strictly followed up for cardiac complications.

Methods: The present study included 90 patients diagnosed with hypertension. The patients were divided into two groups on the basis of whether they exhibited the presence of PSW (n=64, age: 56±11 years) or its absence (n=26, age: 58±13 years), as revealed by the results of left ventricular outflow Doppler echocardi-

ography. The levels of Heart-type free fatty acid-binding protein were compared between the two groups

Results: In the 90 hypertensive patients included in the present study, the prevalence of presystolic wave was determined to be 71%. The Heart-type free fatty acid-binding protein levels in the non-PSW patients were observed to be statistically higher than those in the PSW patients [4.88 ng/mL (2.82–17.83) vs. 3.99 ng/mL (2.57–17.87), p=0.041]. Even though the ejection fractions and the left ventricular diameters were within the normal limits for the patients of both the groups, the value for the left ventricular end-diastolic diameter was observed to be statistically higher in the non-PSW group. No differences were observed in the mitral flow parameters between the two groups.

Conclusions: The present study demonstrated that the absence of presystolic wave in the hypertensive patients with preserved left ventricular ejection fraction is an indicator of subclinical myocardial damage. The hypertensive patients exhibiting the absence of the presystolic wave should be strictly followed up for cardiac complications.

Table 1. Clinical and demographic characteristics of patients

	Patients with PSW n=64	Patients without PSW n=26	p
Age (years)	56±11	58±13	0.443
Female Sex (n, %)	15(57%)	53(82%)	0.012
Heart Rate (beat/min)	75±12	72±13	0.271
Systolic Blood Pressure (mmHg)	130(120-150)	130(120-180)	0.398
Diastolic Blood Pressure (mmHg)	80(60-100)	80(70-95)	0.238
Hypertension Period (months)	60(1.2-480)	60(1.2-360)	0.731
Family History of Coranary Artery Disease (n, %)	21(32%)	5(19%)	0.198
Smoking (n, %)	8(12%)	2(%8)	0.511
Diabetes Mellitus (n, %)	19(29%)	4(15%)	0.159
Hyperlipidemia (n, %)	18(28%)	5(19%)	0.381
Body Mass Index (kg/m2)	33.2±5.6	31.1±5.8	0.129

Table 2. Drugs used by patients

	Patients with PSW n=64	Patients without PSW n=26	p
ACEI (n, %)	14(22%)	9(34%)	0.209
ARB (n, %)	21(32%)	7(27%)	0.584
Thiazide (n, %)	4(6%)	0(0%)	0.192
Beta Blocker (n, %)	14(22%)	3(11%)	0.256
CCB (n, %)	12(19%)	8(30%)	0.214
Alpha Blocker (n, %)	2(3%)	0(0%)	0.362
Spironolactone (n, %)	1(2%)	0(0%)	0.522
Statin (n, %)	4(6%)	0(0%)	0.192

Table 3. H-FABP and biochemical values

	Patients with PSW n=64	Patients without PSW n=26	p
H-FABP (ng/mL)	3.99(2.57-17.87)	4.88(2.82-17.83)	0.041
Glucose (mg/dL)	107.5(76-241)	101.5(76-393)	0.769
CRP (mg/dL)	0.35(0.0-8.5)	0.5(0.1-2.6)	0.444
UREA (mg/dL)	30.2±8.5	30.8±6.7	0.743
CREATINE (mg/dL)	0.74±0.12	0.75±0.18	0.743
eGFR (mL/min/1.73 m2)	94.5±12.1	95.4±17.1	0.776
LDL (mg/dL)	159±33	149±26	0.226
HDL (mg/dL)	48±10	48±11	0.912
Triglyceride (mg/dL)	164±56	162±83	0.891
NA (mmol/L)	139±2.1	139±2.6	0.812
K (mmol/L)	4.3±0.4	4.4±0.3	0.183

Table 4. Echocardiographic parameters

	Patients with PSW n=64	Patients without PSW n=26	p
EF (%)	65(55-68)	65(55-70)	0.895
LA (mm)	34.8±3.6	36.2±3.1	0.081
IVS (mm)	10(7-14)	10(8-14)	0.239
PW (mm)	10(7-14)	10(8-13)	0.282
LVEDD (mm)	46.6±4.5	49.3±3.5	0.008
LVESD (mm)	29.3±5.5	31.6±4.2	0.065
Mitral E (cm/sec)	65.9±17.4	66.5±13.3	0.0881
Mitral A (cm/sec)	76.6±18.6	79.3±20.7	0.547
E/A	0.78(0.48-2)	0.77(0.52-1.9)	0.940

Hypertension

PO-13

A rare cause of drug-induced gingival hypertrophy: Lercanidipine

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Introduction: Gingival tissue changes are frequently seen cases due to extensive use of some classified drugs among patients related to the different conditions. Being aware of this data is necessary for further changes in treatment strategies, and rescheduling the prescribed medicines provocated the gingival proliferation for patient's wellbeing. The gum hypertrophy is a well recognised side effect of dihydropyridine calcium channel blockers, with few reports following non-dihydropyridine calcium channel blockers.

Case Report: A 70 years old man, diagnosed with hypertension stage 1, has applied to our clinic with the main complaint of diffuse upper gingival tissue enlargement. He was consulted by the dentist before, and was referred to the Cardiology outpatient clinic, in order to go under the evaluation of the gingival hyperplasia with the suspicion of adverse effect of Lercanidipine use. The patient has complaints regarding to the bleeding and painful gingival tissue. During the oral examination gingival tissue was found to be quite massive, papillary, rubescent and predisposed to bleed. His gingival tissue was inflamated, largely lobulated, swelled and erythamatous on the general physical assessment (Figure 1). Normal periodontal treatment was suggested for the patient. Antiseptic oral rinse was administered after the scaling treatment and oral hygiene education was given by dentist, Then as a cause of this pathology the antihypertensive treatment tahat taken for 2 months, lercanidipin 20 miligram once a day, changed to an kandesartan. Following 1 month, it was observed that bleeding was reduced, rubescence and edema was recovered, hyperplasia was totally resolved as seen in Figure 2.

Discussion: Calcium channel blockers are drugs characterized by the prevention of cellular entrance of the Calcium ions which reduces oxygen consumption, inhibits contractions and indeed, causes the dilation of veins and arteries. It is the mechanism how calcium channel blockers regulates the blood pressure in hypertensive patients. The other mechansims of action are inhibition of the extracellular matrix proteins' synthesis (ex: collagen, fibronectin, vs), growth and proliferation of the smooth muscle cells and fibroblasts. Periferic vasodilation and over-accumulation of type 1 collagen is thepredicted underlying mechanism of the gingival hyperplasia. Gingival enlargement inducing drugs can have distinctive mechanism of action. In this process, fibroblasts of the underlying tissue are the major objective cells which are responsive for the connective tissue increase in gingivas. Another considerable predisposing factor for this undesirable effect is predicted to be the gingival inflammation. In conclusion, it is suggested that fibroblasts, drug metabolites and inflammatory mediators, originated from different structures, interacts between each other and eventuate in gingival hyperplasia.

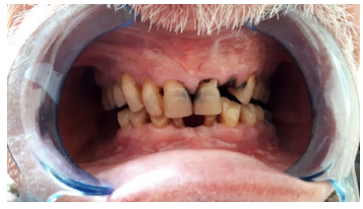

Figure 1. Gyngival Hypertrophia under lercanidipine treatment.

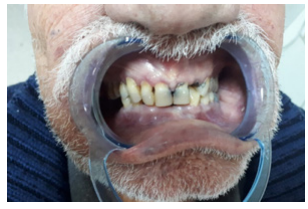

Figure 2. 1 month after cessation of lercanidipine, and treatment by dentist.

Cardiovascular surgery

PO-14

Large primary right ventricular sarcoma

Hatice Özdamar,[1] Tuba Ekin,[1] Önercan Çakmak,[1] Reha Toprak,[2] Kıvanç Gökhan Kaçar,[2] Şahin Karakılıç,[2] Dündar Özalp Karabay,[2] Nezihi Barış[1]

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Introduction: Primary sarcomas of the heart, although very rare, are the second most common type of primary cardiac neoplasm and account for most of the malignant primary cardiac tumors. Surgery remains to be the treatment of choice for cardiac sarcoma. In addition to histologic grade, the survival of the initial surgery is another important prognostic indicator for patients with high-grade cardiac sarcoma. Primary right ventricular sarcomas has a poor prognosis due to bulky and infiltrative late presentation, challenging resection and limited efficacy of systemic therapies.

Case Report: A-69-year old male presented in 10 days history of worsening episodic dyspnea, lower-extremity edema, and angina. Examination revealed diastolic murmur, jugular vein distention, and bilateral pitting pedal edema. There were no significant on electrocardiographic findings. Initial cardiac biomarkers were negative. D-dimer was positive and pulmonary computed tomography angiography ruled out pulmonary embolism, any mediastinal tumor or metastasis.CT observed an irregular mass in the Right Ventricle (RV) apex. Transthoracic echocardiography (TTE) demonstrated normal Left ventricular (LV) systolic function and mild diastolic limitation in the LV apex. Right side TTE 3.9 x 3.0 cm echogenic, organized mass extending from the right ventricular apex to right ventricular outflow tract (RVOT) mass continued with thin, highly mobile irregular structures extending from RVOT to RV cavity were observed. A large thrombus or cardiac tumor was considered as a preliminary diagnosis. The patient underwent urgent surgery due to hypotension bradycardia and shortness of breath. In operation, a 5.0 x 3.0 x 2.0 cm fragile tumor was found to begin from the RV apex and invade into the free wall. The mass was completely excised to reach normal myocardial tissue and ventriculoplasty was performed. Pathological examination showed the presence of primary cardiac sarcoma. After the operation, the patient was followed up in the intensive care unit. The post-operative bed-

side TTE showed right ventricular apical akinesia and hypokinesia of the RVOT. Acute renal failure requiring dialysis developed three days after the operation. The patient whose general condition worsened, died six days after the operation.

Discussion: Primary cardiac sarcomas are rare and very poor prognosis. Most useful diagnostic tools are TTE,CT, and CMR which defined cases location and resectability. Characterization of their pathoanatomy remains the gold standard for exact tumor diagnosis. Despite the use of resection surgery and or adjuvant therapies, the prognosis of some primary cardiac sarcomas is poor as seen in our case.

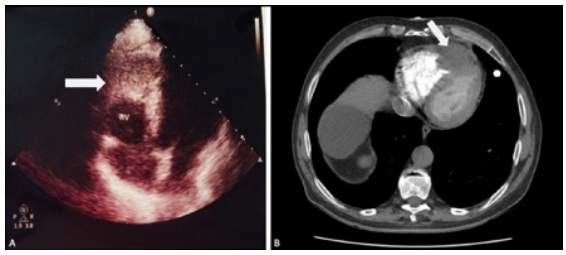

Figure 1. Imaging of primary cardiac sarcoma of RV **(a)**. Echocardiography showing a large echogenic mass adherent to right ventricular apex and free wall. **(b)** Pulmonary CTA showing mass in the RV invading the free wall and apex.

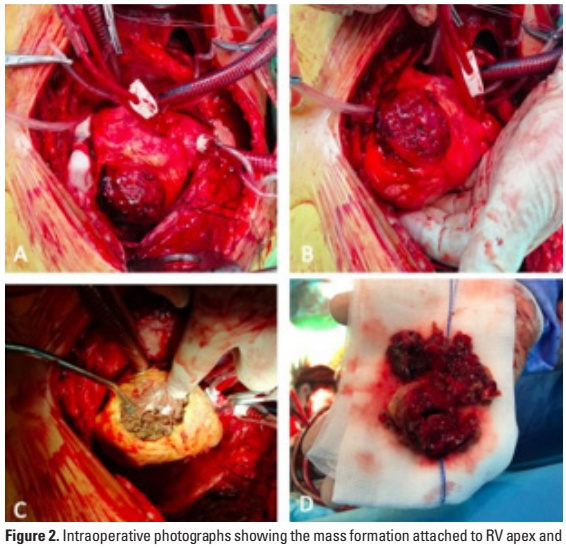

Figure 2. Intraoperative photographs showing the mass formation attached to RV apex and free wall. **(a-c)** Large, fragile mass begin from the RV apex and invade into the RV free wall. **(d)** Complete excision of large fragile mass.

Heart valve diseases

PO-15

Tricuspid valve infective endocarditis related to intravenous heroin (The first case was reported in Turkey)

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Summary: Although intravenous drug addiction (IVDA) is responsible for 10% of all İ.E cases, it is responsible for 1% in our country In this paper, we present the first case of IE involving the tricuspid valve (TV) due to IVDA in our country.

Introduction: Substance addiction and the use of intravenous narcotic drugs are serious social and health problems in both developed and developing countries. In parallel with this, physicians now encounter more drug-addicted patients and related health problems. While intravenous drug addiction (IVDA) is responsible for 10% of all infective endocarditis (IE) cases in developed countries, it is still quite rare in our country and responsible for 1% of all cases. The previously reported cases related to IVDA in our country were left-sided IE. Our case is the first case of the tricuspid valve (TV) IE due to IVDA in our country.

Case Report: A 23-year-old male patient was admitted to the emergency department with complaints of dyspnea, fever, and fatigue. Thorax tomography of the patient revealed multiple pulmonary septic embolism. To clarify the etiology, we applied transthoracic echocardiography which showed normal left ventricular and valvular functions but right chambers were dilated, right ventricular functions were depressed, there were severe tricuspid regurgitation and pulmonary hypertension (systolic pulmonary artery pressure:70 mmHg). Moreover, there was a mass with a 22x22 mm size on the tricuspid valve. His history revealed that he was addicted to intravenous heroin for 5 years and could not give up against several attempts. Intravenous meropenem and vancomycin treatments were started after cultures because of IE. Transesophageal echocardiography also showed a huge vegetative mass (24x28 mm) on the tricuspid valve and severe regurgitation (Figure 1). Because of recurrent septic pulmonary embolism, right heart failure, and the vegetative mass greater than 20 mm against intravenous antibiotics, surgery was planned for the patient. During the surgery, the huge mass with a 30x20 mm size involving septal and anterior leaflets of the valve was seen (Figure 2). The septal leaflet was completely destroyed. There were multiple endomyocardial vegetative focuses in the right ventricle. The tricuspid valve was replaced with a bioprosthetic valve (Labcor no: 33) and vegetative focuses were cleared in the right ventricle. The ventricle was irrigated with antibiotics and after the successful surgery the patient was taken to the intensive care unit. However, the patient died due to sepsis, right ventricle, and acute renal failure 5 days after the operation.

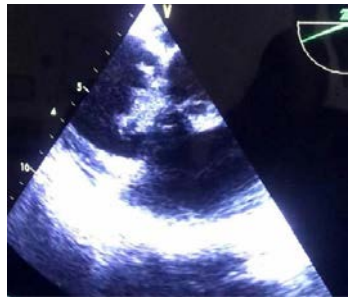

Figure 1. Vegetation TEE image on the tricuspid valve.

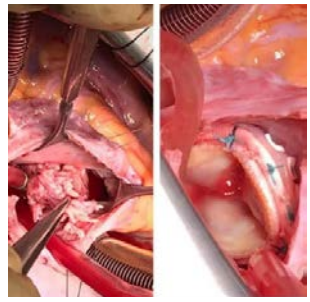

Figure 2. Tricuspid valve vegetation and TVR.

Heart failure

PB-06

Spironolactone may not be effective to reduce mortality in patients with atrial fibrillation in heart failure with reduced ejection fraction

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Background and Aim: HFrEF is associated with high morbidity and mortality. Beta blockers (BBs) and mineralocorticoid receptor antagonists are mainstay of the treatment. Aims of this study to determine the effects of BBs, ACE inhibitors (ACE-I), angiotensin II receptor blockers (ARB) and spironolactone on mortality in patients with sinus rhythm (SR) and in patients with atrial fibrillation (AF).

Methods: 423 males, 227 females (66±12 years old) with HFrEF were recruited into the study. 401 patients were on SR and 249 patients were AF.

Results: Left ventricular ejection fraction was 25.2±8.2% in patients with SR, 26.5±9.0% in patients with AF, p=0.070. Patients were followed average of 55 months. However, 177 (44%) patients with SR died and 91 (37%) patients with AF died, p=0.056 during follow up (FU). 40% of patients who were on beta blockers died, 55% of patients who were not on beta blocker treatment died in patients with SR during FU p=0.011. 32% of patients who were on BBs died and 46% patients who were not on BBs died in patients with AF, p=0.039. Although 38% of patients who were on Spironolactone treatment died in patients with SR, 50% of patients who were not on spironolactone died during follow up, p=0.017. While 41% of patients who were on spironolactone treatment died in patients with AF, 32% of patients who were not on spironolactone died during follow up p=0.117. Taking of ACE-I or ARB did not show any different effect on mortality in either patients with sinus rhythm or AF.

Conclusions: Although BBs reduced the mortality rate both in patients with SR and in patients with AF in HFrEF, spironolactone reduced mortality only in patients with SR.

Heart failure

PB-07

Warfarin use associated with recurrent hospitalization in chronic heart failure with systolic dysfunction

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Background and Aim: Chronic heart failure (CHF) is associated with a hypercoaguable state with increased incidence of left ventricular thrombi, ischemic strokes, and other thromboembolic events, even in the setting of sinus rhythm. Due to this, there is rationale for the use of systemic anticoagulation in these patients in an effort to reduce morbidity and mortality. Our aim is to determine the effects of warfarin use on recurrent multiple hospitalizations in patients with sinus rhythm in advanced CHF and left ventricular systolic dysfunction.

Methods: 268 male patients and 137 female patients (mean age, 65±12 years; mean ejection fraction, 25.5±9.5%) with advanced CHF and left ventricular systolic dysfunction were recruited into the study. All patients were on sinus rhythm.

Results: Sixty five (16%) patients were taken warfarin in this cohort. Patients were divided into 2 groups. Group 1: If patients were hospitalized less than 3 times and group 2: if patients were hospitalized ≥3 times during follow up period. Patients were followed average of 57 months. Patients in group 2 were younger (63±14 vs 66±11, p=0.020) and had reduced ejection fraction (23.7±9.8%, vs 26.2±9.3, p=0.016) and larger right ventricular dimension (27±5 mm vs 26±4, p=0.021) compared to the patients group 1. 26 patients (40%) with sinus rhythm who were on warfarin were hospitalized ≥ 3 times and 89 patients (26%) with sinus rhythm who

were not on the medication were hospitalized ≥3 during follow-up (p=0.022).
Conclusions: More patients who were on warfarin had ≥3 times hospitalizations than the patients who were not on the warfarin in patients with sinus rhythm in advanced CHF and left ventricular systolic dysfunction. Warfarin use was associated more recurrent hospitalizations in this cohort.

Heart failure

PB-08

Patients with peripheral artery disease have increased risk for cardiac mortality in patients with diabetes in heart failure with reduced ejection fraction

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Background and Aim: Heart failure is a prevalent clinical syndrome in the world population. Both heart failure and peripheral artery disease (PAD) share risk factors. Prognostic significance of PAD in patients with diabetes mellitus (DM) and heart failure with reduce ejection fraction (HFrEF) does not clear. Aims of this study 1: to determine the prevalence of patients with PAD in patients with DM and in patients without DM in HFrEF and 2: to compare the effect of PAD on cardiac mortality in patients with DM and in patients without DM in HFrEF.
Methods: Method and RESULTS: 250 (40%) patients with DM, 380 (60%) patients without DM with HFrEF were enrolled into the study. There were 399 (63%) males, 231 (37%) females in the group. Patients' mean age was 66±12 years old.
Results: There was no age difference between patients with DM and without DM p=0.343. While left ventricular ejection fraction was 25±9% in patients with DM, it was 25±10 % in patients without DM, p=0.481. 88 (14%) of patients had PAD in the group. Although 57 (23%) patients had PAD in patients with DM, 31 (8%) had PAD in patients without DM, p<0.001. Patients were followed average of 55 months. While 35 (61%) patients with PAD died, 86 (45%) patients without PAD died in patients with DM during follow up, p=0.025. 18 (58%) patients died with PAD, 149 (43%) patients died without PAD in patients without DM, p=0.098.
Conclusions: Cardiac mortality was higher in patients with PAD compared to patients without PAD in diabetic patients in this HFrEF cohort.

Heart failure

PO-16

A rare cause of heart failure and increased lactic acid level: Wet beriberi

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Beriberi is a rarely seen disease caused by a severe thiamine deficiency for more than three months. It is mostly seen in patients with severe alcoholism, malnutrition or gastric surgery. Wet beriberi, on the other hand, courses more often with cardiac symptoms in contrast to dry beriberi. Since beriberi caused by thiamine deficiency progresses with heart failure symptoms, it has no specific symptoms. For this reason, taking the past medical history carefully and awareness of the disease are the most important points in making a diagnosis in these patients. In this case, we aimed to present our diagnostic and therapeutic approach to a patient who had undergone gastric surgery and presented with high-output heart failure symptoms.

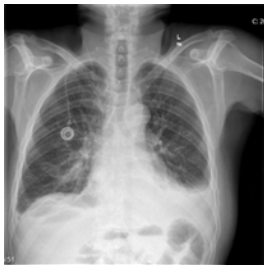

Figure 1. PA chest radiography at admission. Bilateral pleural effusion is seen. In addition, when the heart contour is evaluated, left atrium appears to be dilated.

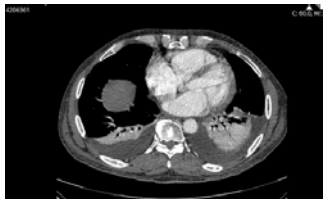

Figure 2. PA chest radiography at admission. Bilateral pleural effusion and compressive atelectasis are detected.

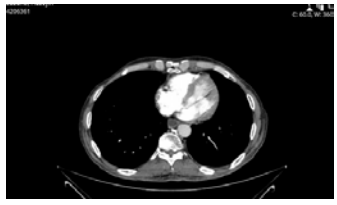

Figure 3. Control computed tomography image 1 month later.

Cardiac imaging / Echocardiography

PB-09

A new method for prediction of echocardiographic diastolic dysfunction: Electrocardiographic diastolic index

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Background and Aim: Diastolic dysfunction (DD) in transthoracic echocardiography (TTE), which is a poorly understood entity due to its limited treatment, has been frequently encountered in daily clinical practice of cardiology. An electrocardiographic (ECG) index to predict echocardiographic DD has not been elucidated yet. We aim to exhibit an electrocardiographic diastolic index (EDI) to predict TTE DD with a high sensitivity and specificity.
Methods: In this retrospective investigation, we tested the DD predictive value of EDI aVL R amplitude x (V1S amplitude+V5R amplitude) / D1 P amplitude] on 204 consecutive adult patients without known coronary artery disease. Patients were divided into tertiles according to their EDI starting from the lowest one. The power of the EDI was also compared with the subunits of its formula by a receiver operating curve (ROC) analysis.
Results: After adjustment for confounding baseline variables, EDI in tertile 3 was associated with 24.2-fold hazard ratio of DD (OR: 25.2, 95% CI 11.2–51.1, p<0.001). Spearman correlation analysis revealed moderate correlation between E/e' and EDI. A ROC analysis showed that the optimal cut-off value of the EDI to predict DD was 8.53 mV with 70% sensitivity and 70% specificity (AUC: 0.78; 95% CI: 0.71–0.84; p<0.001).
Conclusions: EDI, which is a cheap, feasible, and easily calculable formula, appears to have a considerable role to predict DD in adult patients.

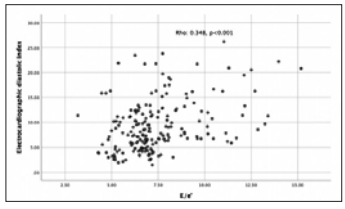

Figure 1. Scatterplot of the correlation between E/e' and electrocardiographic diastolic index. (Rho: 0.348, p<0.001).

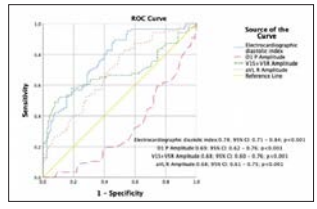

Figure 2. A ROC analysis showed that the optimal cut-off value of the EDI to predict DD was 8.53 mV with 70% sensitivity and 70% specificity (AUC: 0.78; 95% CI: 0.71–0.84; p<0.001), the optimal cut-off value of the PVL1 to predict DD was 0.102 mV with 67% sensitivity and 60% specificity (AUC: 0.69; 95% CI: 0.62–0.76; p<0.001), the optimal cut-off value of the aVL R wave amplitude to predict DD was 0.517 mV with 62% sensitivity and 61% specificity (AUC: 0.68; 95% CI: 0.61–0.75; p<0.001), the optimal cut-off value of the sum of V1S amplitude and V5R amplitude to predict DD was 1.85 mV with 65% sensitivity and 65% specificity (AUC: 0.68; 95% CI: 0.60–0.76; p<0.001).

Table 1. Echocardiographic and electrocardiographic findings of all patients

	T1, n=68	T2, n=68	T3, n=68	P value
Left ventricular end-systolic dimension, mm	28.2 ± 5.2	30.7 ± 5.7	32.8 ± 5.3	<0.001
Left ventricular end-diastolic dimension, mm	47.0 ± 3.2	49.2 ± 4.0	49.5 ± 4.0	<0.001
Interventricular septum thickness, cm	1.07 ± 0.16	1.16 ± 0.22	1.21 ± 0.18	<0.001
Posterior wall thickness, cm	0.97 ± 0.10	1.04 ± 0.16	1.09 ± 00.16	<0.001
Left ventricular ejection fraction, %	59.8 ± 2.6	59.6 ± 3.5	57.1 ± 3.7	<0.001
Left ventricular mass index, g/m2	95.5 ± 19.3	101.6 ± 29.6	112.7 ± 26.7	0.001
Left atrial volume, mL	56.4 ± 12.2	62.3 ± 16.4	72.0 ± 23.9	0.001
Left atrial volume index, mL/m2	30.3 ± 6.5	33.0 ± 8.2	37.5 ± 12.0	0.003
E, cm/s	79.7 ± 13.7	75.4 ±17.5	72.0 ± 23.9	0.011
A, cm/s	66.3 ± 16.9	75.4 ± 17.5	70.0 ± 23.2	0.014
e' lateral, cm/s	14.0 ± 2.7	11.2 ± 3.4	9.9 ± 4.0	<0.001
a' lateral, cm/s	11.3 ± 2.5	12.4 ± 2.7	13.1 ± 3.6	0.014
e' septal, cm/s	11.1 ± 2.6	9.4 ± 2.7	7.9 ± 3.0	<0.001
a' septal, cm/s	9.5 ± 1.9	10.6 ± 2.3	10.1 ± 3.1	0.030
E/e' average ratio	6.4 ± 1.2	7.5 ± 1.8	9.4 ± 4.6	<0.001
E/A ratio	1.27 ± 0.34	1.07 ± 0.39	1.05 ± 0.41	<0.001
Deceleration time, ms	188.1 ± 18.1	194.1 ± 23.8	205.0 ± 28.1	<0.001
Tricuspid regurtitation velocity	1.68 ± 0.5	2.16 ± 0.8	2.48 ± 0.8	<0.001
Electrocardiography parameters				
D1 P wave amplitude, mV	0.12 ± 0.03	0.11 ± 0.02	0.11 ± 0.02	0.002
aVL R amplitude, mV	0.29 ± 0.12	0.48 ± 0.12	0.87 ± 0.24	<0.001
V1S amplitude, mV	1.03 ± 0.12	1.11 ± 0.14	1.14 ± 0.14	<0.001
V5R amplitude, mV	0.78 ± 0.14	0.81 ± 0.21	0.94 ± 0.38	0.085
V1S amplitude + V5R amplitude, mV	1.81 ± 0.18	1.92 ± 0.27	2.08 ± 0.43	<0.001
Electrocardiographic Diastolic Index	4.38 ± 1.35	8.28 ± 1.27	18.35 ± 10.89	<0.001
Grade of left ventricle diastolic function				
Indeterminate diastolic function, n (%)	7 (10.3)	28 (41.2)	27 (39.7)	<0.001
Grade 1, n (%)	2 (2.9)	3 (4.4)	10 (14.7)	0.021
Grade 2, n (%)	0 (0.0)	0 (0.0)	9 (13.2)	<0.001

Table 2. Logistic regression models for any grade of left ventricle diastolic dysfunction by Electrocardiographic Diastolic Index tertile

	T1, n=68	T1, n=68	T1, n=68
Left ventricle diastolic dysfunction			
Number of patients	9	31	46
Case rate, %	13.2	45.6	67.6
Left ventricle diastolic dysfunction, OR (%95 CI)			
Model 1:unadjusted	1[Reference]	3.4 (1.6 – 7.0)	16.6 (7.2 – 33.9)
Model 2: adjusted for all covariatesa	1[Reference]	2.5 (1.4 – 4.8)	25.2 (11.2 - 51.1

Table 3. Spearman rank correlation between E/e' and Electrocardiographic Diastolic Index, aVL R amplitude, V1S+V5R amplitude, and D1 P amplitude

	R	P value
E/e' and Electrocardiographic Diastolic Index	0.348	<0.001
E/e' and aVL R amplitude	0.280	<0.001
E/e' and V1S+V5R amplitude	0.259	<0.001
E/e' and D1 P amplitude	-0.212	<0.001

Cardiac imaging / Echocardiography

PB-10

The role of presystolic A wave in the differentiation of non-obstructive hypertrophic cardiomyopathy and athlete's heart

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Background and Aim: Even though hypertrophic cardiomyopathy (HCM) can be distinguished from the athlete's heart with a difference in echocardiographic measurements, difficulties may be encountered in clinical practice from time to time. The aim of this study was to evaluate the use of presystolic A wave (PSAW) assessed by the Doppler echocardiography for differentiating between HCM and athlete's heart.
Methods: A total of 52 subjects were included in the present study, of which, 27 had HCM and 25 were athletes. The pulsed Doppler assessment of the left ventricular outflow tract was performed on the ventricular face from the immediate proximal of the aortic valve on the apical five-chamber view. Allpatients were assessed for the presence of PSAW,and the velocity of this wave was recordedin PSAW positive subjects.
Results: The frequency of PSAW was found to be higher in patients with HCM [n=12 (44%)] than in athletes [n=4 (16%)] (p=0.026). PSAW velocity measurements were observed to be higher in the HCM group; however, there was no statistical significance [53 ms (36–84)], [68 ms (35–193)], (p=0.362).
Conclusions: While there is a need for scaling-up similar studies, the current findings suggest that PSAW can be used as a distinguishing parameter for differentiating HCM and athlete's heart.

Table 1. Echocardiographic measurements

Variables	Control, n:25	HCM, n:27	p
IVS (mm)	11 (8–15)	19 (13–28)	<0.001
PW (mm)	9 (8–14)	14 (11–24)	<0.001
LVMI (g/m2)	84.7 (46.8–128.35)	168.1 (95.3–367.81)	<0.001
MITRAL E/A	1.5 (0.63–2.24)	0.93 (0.60–2.07)	0.019
PSAW	4 (16%)	12 (44%)	0.026
PSVEL (m/s)	53 (36–84)	68 (35–193)	0.362

IVS: Interventricular septum, PW: Posterior wall, LVDd: LVMI: Left ventricular mass index, PSAW: Presystolic a wave, PSVEL: Presystolic velocity

Cardiac imaging / Echocardiography

PO-17

An unusual echocardiographic finding in a patient with systolic dysfunction: Early diastolic mitral regurgitation with absence of E wave

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A 56-years-old male patient was referred to our cardiology department due to a recent cerebrovascular accident. The patient had a history of hypertension, diabetes mellitus, and non-ischemic systolic heart failure. The 12-lead electrocardiography showed a normal sinus rhythm, a normal PR (154 ms), a poor R wave progression, deep S-waves in precordial leads with decreased voltage in standard derivations and T-wave negativity on the leads V4-V6 (Figure 1a). Transthoracic echocardiography showed severe left ventricle (LV) systolic dysfunction with an ejection fraction of 30%. The patient underwent a transesophageal echocardiography (TEE) examination to exclude any thrombosis. The pulsed-wave Doppler TEE displayed mild mitral regurgitation (MR) along with systole extending to early diastole, lasting with the initiation of mitral A-wave and with absence of E wave (Figure 1b, c). Color M-mode at the mitral level revealed MR that continues after the end of systole, covering the early and diastasis phases of diastole (Figure 1d). There was a concomitant mild tricuspid regurgitation and trivial aortic regurgitation (AR). Early diastolic MR has been scarcely reported. The reported cases are those with moderate or advanced aortic insufficiency or hypertrophic cardiomyopathy. In literature review, we could not find any paper reporting early diastolic MR along with absence of the mitral E wave in a patient without significant aortic regurgitation. We think that the present case is unique with the following features: (a)There is no mitral E wave; (b)The aortic regurgitation is too mild to cause a diastolic MR; (c)There are no other clinical entities, such as hypertrophic cardiomyopathy, significant atrioventricular dissynchrony or first degree atrioventricular block, that causes diastolic MR.

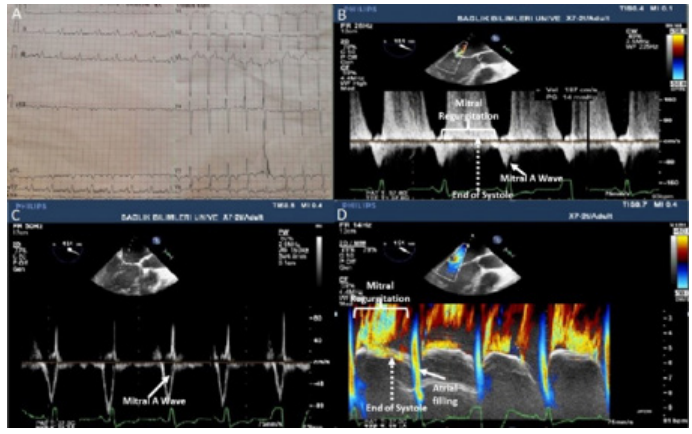

Figure 1. A-A 12-lead electrocardiography of the patient showing a normal PR (154 ms), deep S-waves in precordial leads with decreased voltage in standard derivations and T wave negativity on the leads V4-V6 B-C-Transesophageal echocardiographic examination showing the mitral regurgitation along with systole, extending to early diastole and lasting with the initiation of mitral A-wave and absence of E wave. D-Color M-mode transesophageal echocardiography examination showing early diastolic mitral regurgitation before the occurrence of the QRS complex.

Cardiac imaging / Echocardiography

PO-20

Bileaflet flail of mitral valve with myxomatous degeneration

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Mitral valve prolapse (MVP) is defined as anterior, posterior or bileaflet mitral valve (MV) billowing toward the left atrium by more than 2mm above the mitral annulus horns during Left Ventricle systole and affects about 5% of the general population. MVP following rupture of the chordae tendineae is a major cause of primary Mitral Regurgitation(MR) and is associated with several causes such as endocarditis, myxomatous degeneration, connective tissue abnormalities and Barlow's disease. Myxomatous MV has underlying pathological mechanisim which is the result of 'Fibroelastic Deficiency'. Although patients with ruptured and flail cords are mostly observed posterior leaflet involvement, less frequent anterior leaflet location can be observed. IIn this case, we reported a symptomatic patient who reported a serious MR jet due to anterior and posterior chorda rupture, which is rare in both transthoracic (TTE) and transesophageal echocardiography (TEE).

Case Report: A 63-year-old male admitted with complains of progressive dyspnea. According New York Heart Association (NYHA), the functional classification was from II to III. His blood pressure was 100/70 mmHg and his pulse rate 80 beats/min and regular rhythm. On physical examination he had no rale or rhoncus, grade 3/6 pansystolic ejection murmur at the apex with radiation to the axillae. His 12-lead-ECG showed sinus rythm with left ventricular hypertrophy and laboratory findings were normal. Two-dimension TTE demonstrated normal Left Ventricular systolic function, enlarged left ventricular and atrial diameters, severe MR caused by MVP with bileaflet chordea ruptured. TEE was performed and confirmed the myxomatous degeneration and Barlow syndrome of MVP of both anterior and posterior leaflets. In addition, Real time three-dimensional TEE there were flails of A2, P2 and P3 scallops of both anterior and posterior leaflets with severe MR (Figure 1). According to echocardiographical findings and symptoms, he was diagnosed as myxomatous degeneration MVP with flail posterior and also anterior leaflets. His coronary angiogram showed stable coronary plaques. He underwent MV replacement with a mechanical protheses.

Conclusions: Echocardiogram is the gold standard in grading of cordal rupture in leaflets and MR severity in MVP. TEE can accurately identify the underlying mechanism of MR, involved scallop segment of flail leaflet and for planning MV surgery. According to clinical studies, P2 is the most common site of localized prolapse and flail due to myxomatous degeneration and fibroelastic dysplasia(5) Most patients with bileaflet mitral valve prolapse caused by degenerative disease do not have significant anterior chordal pathology. In addition to the posterior leaflet, we want to present the accompanying kordal rupture phenomenon in the rarely seen front leaflet because we think it will contribute to the literature.

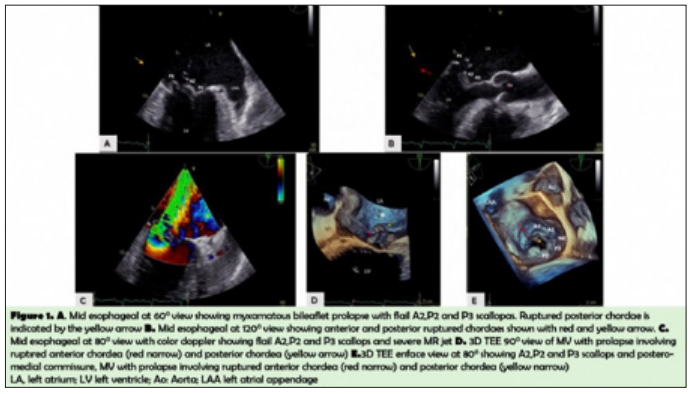

Figure 1. A. Mid esophageal at 60° view showing myxomatous bileaflet prolapse with flail A2,P2 and P3 scallops. Ruptured posterior chordae is indicated by the yellow arrow **B.** Mid esophageal at 120° view showing anterior and posterior ruptured chordaes shown with red and yellow arrow. **C.** Mid esophageal at 80° view with color doppler showing flail A2,P2 and P3 scallops and severe MR jet **D.** 3D TEE 90° view of MV with prolapse involving ruptured anterior chordea (red narrow) and posterior chordea (yellow arrow) **E.**3D TEE enface view at 80° showing A2,P2 and P3 scallops and postero-medical commisure, MV with prolapse involving ruptured anterior chordea (red narrow) and posterior chordea (yellow narrow)
LA, left atrium; LV left ventricle; Ao: Aorta; LAA left atrial appendage

Figure 1. Two and three-dimensional transesophageal echocardiography.

Coronary artery disease / Acute coronary syndrome

PB-11

Usefulness of whole blood viscosity estimated by de simeone's formula to predict left ventricular thrombus formation within one year following acute anterior myocardial infarction

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Background and Aim: Despite improvements in treatment of ST-segment myocardial infarction (STEMI), thrombus formation in the left ventricle is still a concerning complication which may lead to systemic thromboembolism such as stroke. Whole blood viscosity (WBV), the primary determinant of endothelial shear stress, has been associated with cardiovascular events and stroke. We evaluated the predictive value of estimated WBV in left ventricular thrombus development in patients with acute anterior myocardial infarction (AAMI) survivors.
Methods: This was a prospective, single-center study conducted between January 2016 and January 2019. A total of 780 patients who admitted to our tertiary center with acute anterior myocardial infarction (AAMI) and underwent primary percutaneous coronary intervention (pPCI) were enrolled consecutively. The peripheral blood samples were obtained at the emergency department of our hospital. Serial echocardiographic examinations were performed within 24h of admission, before hospital discharge and at 1, 3, 6 and 12 months following hospital discharge. To calculate WBV, we used the confirmed formulae due to calculating both low shear rate (LSR) (0.5 sec-1) and high shear rate (HSR) (208 sec-1) from HCT (%) and total plasma protein (TP) (g/l) concentration as suggested by De Simone et al.
Results: One hundred patients (12.8%) developed thrombus formation following within one year AAMI. Patients with left ventricular thrombus (LVT) had significantly higher WBV values. In multivariable logistic regression analysis, two different models were constituted for supramedian WBV levels at each shear rate. In models adjusted with LVEF on admission, LV aneurysm, diabetes mellitus, CRP levels, proximal LAD lesion location, number of diseased artery >1, GP Iib/IIIa inhibitors treatment, supramedian value of WBV at HSR (OR: 2.171; 95% CI: 1.313-3.590; p=0.003) and supramedian value of WBV at LSR (OR: 1.680; 95% CI: 1.025-2.753; p=0.040) were found as independent predictors of LVT development.
Conclusions: As an easily accessible parameter, WBV might be a useful predictor of LVT formation within one year following acute anterior myocardial infarction.

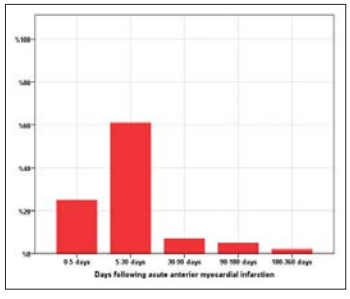

Figure 1. Diagnosing of LVT with days following acute anterior myocardial infarction.

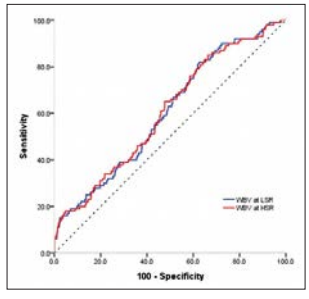

Figure 2. The ROC curve analysis of whole blood viscosity at low shear rate (blue line) and high shear rate (red line) to predict left ventricular thrombus (LVT). The optimal cut-off value 16.36 for WBV at HSR with 76% sensitivity and 42% specifity. (AUC= 0.660, 95% CI 0.549-0.664, p=0.001). The optimal cut-off value 44.65 for WBV at LSR with 77% sensitivity and 40% specifity. (AUC= 0.650, 95% CI 0.547-0.662, p=0.001). AUC: Area under curve; HSR: High shear rate; LSR: Low shear rate; ROC: Receiver operating characteristic; WBV: Whole blood viscosity.

Coronary artery disease / Acute coronary syndrome

PB-12

The relationship between telomerase activity and overall mortality in patients with coronary artery disease

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Background and Aim: Telomeres are DNA-protein complexes that cap the ends of linear chromosomes, protecting DNA from damage. When telomeres critically shorten, cells become susceptible to senescence and apoptosis. Telomerase adds telomeric repeats to terminal DNA, a critical process that helps stall genomic instability and apoptosis. In this study, we aimed to compare all- cause mortality according to the telomerase activity in patients with stable coronary artery disease (SCAD) and with acute coronary syndrome (ACS).
Methods: The study included 211 patients (78 ACS and 71 SCAD patients) aged between 55 and 75 who underwent coronary angiography and measured telomerase concentration by ELISA. Telomerase concentration was used to determine telomerase activity.
Results: Mean age was 63.4±6.1 in the ACS group, 63.4±6.4 in the SCAD group (p=0.200). Seventy three percent (57) of the ACS patients and 76% (54) of the SCAD patients were male (p=0.677). Patients were followed for a median of 32 months (minimum: 0.1, maximum: 46.8). During the follow-up, 11 (14.1%) patients died in the ACS group, whereas 6 patients died in the stable CAD group (p=0.275). In the stable coronary group serum telomerase concentration was numerically lower in patients who died during the follow- up than patients who survived, however, this was statistically insignificant (35.98±2.02 vs. 36.86±1.52 ng/ml; p=0.529) Likewise, in ACS patients, serum telomerase concentration were similar between the patients who died and survived (36.39±1.08 vs. 36.63±1.60 ng/ml, respectively, p=0.993). The association between telomerase activity and overall mortality rate was assessed by comparing the groups in tertiles. Neither in ACS group nor in SCAD group, there were differences in all – cause mortality between the highest, middle, or lowest telomerase concentration groups (p=0.998 in ACS group and p=0.775 in SCAD group). Similar results were observed when the groups were combined as CAD group (n=149, p=0.921, figure).
Conclusions: In the current study, telomerase activity was not associated with all- cause mortality in patients with ACS and SCAD, during the follow-up.

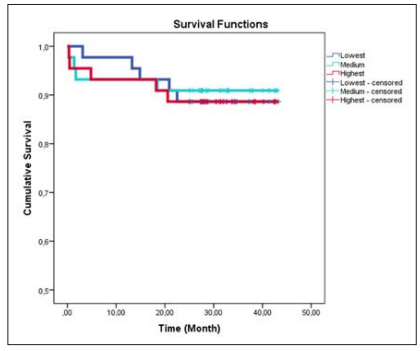

Figure 1. Kaplan-Meier survival curves according to the tertiles.

Coronary artery disease / Acute coronary syndrome

PB-13

Association between C-reactive protein to albumin ratio and left ventricular thrombus formation following acute anterior myocardial infarction

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Background and Aim: Left ventricular thrombus (LVT) is associated with inflammatory response in survivors with anterior ST-elevation myocardial infarction (STEMI). The C-reactive protein to albumin ratio (CAR) has been proposed as a marker of inflammation. However, there is a lack of data with respect to the role of CAR in LVT development. We investigated the relationship between CAR and LVT development in anterior STEMI patients treated percutaneously.
Methods: This was a prospective, single-center study conducted between January 2016 and June 2019. A total of 955 patients who were admitted to our tertiary center with acute anterior STEMI and who underwent primary percutaneous coronary intervention (pPCI) were enrolled consecutively. Patients with a previous history of coronary artery disease, advanced liver disease, renal failure, malignancy, hematological and inflammatory disease, hypo-and hyperthyroidism, active infection, severe valvular disease, treatment with fibrinolytic agents and presentation within >12 h of symptom onset were excluded. The baseline demographic and clinical characteristics of patients were obtained. STEMI diagnosis was defined according to the American College of Cardiology and European Society of Cardiology guidelines. Each patient received standard pharmacologic treatment according to current treatment guidelines. All patients underwent screening transthoracic echocardiography (TTE) within 24h of admission. Repeat TTE was performed before discharge (within 3-5 days). In addition, independently from the presence of thrombus, all patients underwent repeat TTE at 1, 3, and 6 months following hospital discharge.
Results: LVT was observed in 126 (13.2%) patients. The majority of LVT cases were seen within the first

30 days after anterior STEMI (88.9%) The CAR was significantly higher in patients with LVT (12.6 [8.6-16.1] vs 18.1 [11.5-23], p<0.001). In multivariable logistic regression analysis, lower left ventricular ejection fraction, presence of left ventricular aneurysm, neutrophil count, higher total protein level, higher peak CK-MB activity, proximal LAD artery lesion location, GPIIb/IIIa inhibitors use, number of diseased arteries >1 and higher CAR (odds ratio [OR]: 1.084; 95% confidence interval (CI): 1.054-1.115; p<0.001) were independent predictors of LVT.
Conclusions: The CAR may be a practical tool for predicting LVT development among survivors of anterior STEMI. Considering the association between higher CAR and the risk of LVT, closer follow-ups and more potent and/or extended use of anticoagulant therapy may have an additional benefit in patients with higher CAR following anterior STEMI.

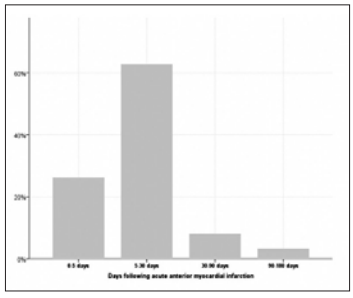

Figure 1. Detection of LVT with days following acute anterior myocardial infarction.

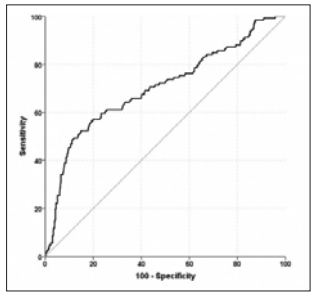

Figure 2. Receiver operating characteristic curve of C-reactive protein to albumin ratio (CAR) to predict left ventricular thrombus. The area under curve was 0.702 (95% CI, 0.658-0.757, p<0.001). The optimal cut-off level of CAR was 16 with 75% specificity and 61% sensitivity. CI: confidence interval.

Coronary artery disease / Acute coronary syndrome

PO-21

The plasticity of R wave amplitude due to transmural ischemia triggered by exercise stress test

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A 42-year-old smoker man with a complaint of chest pain for 6 months was admitted to the outpatient clinic for further evaluation and investigation. He described episodes of awakening by chest pain in the morning subsiding after less than 5 minutes. His father, who had had diabetes and smoked, had died of a sudden cardiac arrest at 46 years of age. The physical examination, routine laboratory tests, and electrocardiography results were completely normal. Transthoracic echocardiography revealed normal left ventricular systolic function with an ejection fraction of 64%. An exercise stress test was performed for ten minutes on the Bruce protocol and reached heart rate of 162 beats/min. The patient had a desire to stop because of a burning in the throat and retro-sternum. After the exercise stress test, the patient developed up to 3mm ST segment elevation concomitant with R wave amplitude enhancement and ST segment depression reciprocal to inferior and posterior epicardial ischemia. R wave amplitude was enhanced and already prominent while the patient was at the level of 4 met (Figure 1). The amplitude of R waves in leads II, III and aVF were tripled at the level of 10 met (Figure 1). ST segment elevation in leads II, III and aVF and reciprocal ST segment depression appeared in the recovery period (Figure 2). The considerable enhancement of R wave amplitude in leads II, III and aVF during exercise and decrease of the enhanced R wave amplitude in the recovery period were frankly presented in Figure 3. The patient had no symptoms of ischemia after the test and his ECG was completely normal. He was hospitalized for further evaluation and the follow-up cardiac enzymes were negative. The patient underwent a cardiac catheterization showing subtotal lesion in the proximal right coronary artery and there was milder disease in left anterior descending and circumflex artery (Figure 4a). The patient underwent successful percutaneous transluminal coronary arterioplasty and a drug eluting stent was successfully implanted to the culprit lesion (Figure 4b). He was discharged well on his 4th day and is currently asymptomatic followed at our cardiology department. R wave enhancement is one of the important markers of ischemia in patients with coronary artery disease. It is very difficult to detect all stages of transmural ischemia without myocardial infarction and R wave enhancement tracked by ST segment elevation in the absence of Q wave appearance is an infrequent event. Our case report illustrates to the nonspecific response of the myocardium to the triggered transmural ischemia.

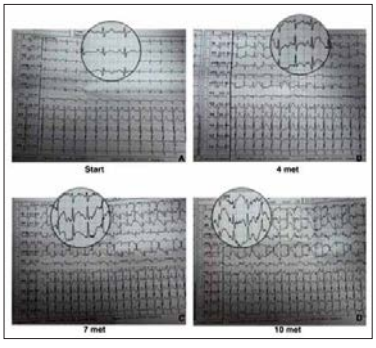

Figure 1. Figure 1-2 presents the R wave enhancement and concomitant ST segment elevation in leads II, III and aVF during the course of the exercise stress test.

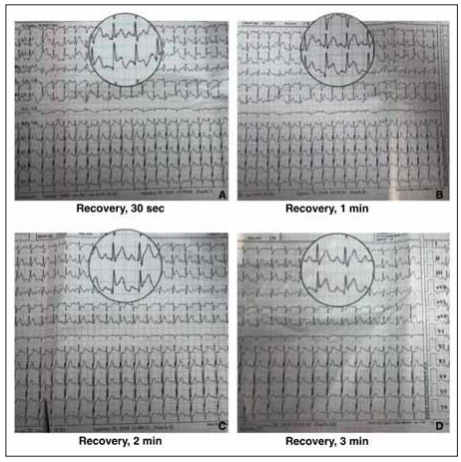

Figure 2. Figure 1-2 presents the R wave enhancement and concomitant ST segment elevation in leads II, III and aVF during the course of the exercise stress test.

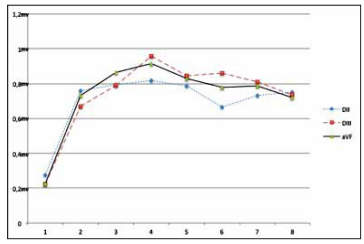

Figure 3. Figure 3 presents the R wave changes during the exercise stress test in leads II, III and aVF.

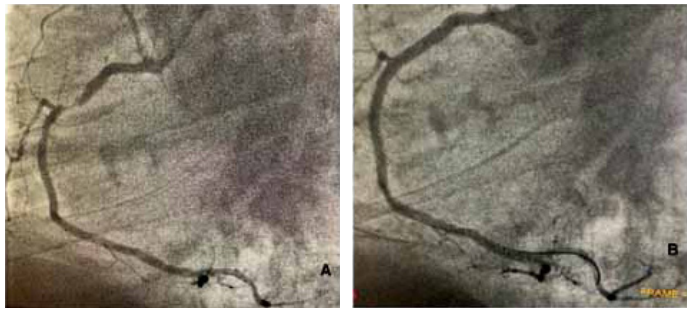

Figure 4. A shows the subtotal lesion in the proximal right coronary artery. Figure 4B shows the final view of the right coronary artery after stent implantation.

Coronary artery disease / Acute coronary syndrome

PO-22

Treatment of Giant morgagni hernia presented with acute coronary syndrome simultaneously with coronary Bypass surgery

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Introduction: Morgagni hernia (MH) is a congenital defect of the diaphragm that accounts for 3% of all diaphragmatic hernias. Most (90%) of them are located in the right hemithorax and are generally asymptomatic. Here, we present a rare case of MH with unstable angina-like symptoms, undergoing simultaneous surgical treatment with coronary artery bypass grafting, which is rarely reported in the literature.
Case Report: A 63-year-old female patient was admitted to the emergency department with complaints of chest pain and shortness of breath. The patient, whose ECG had not ischemic changes with normal cardiac troponin T values, was admitted to the coronary intensive care unit with the prediagnosis of unstable angina pectoris. Coronary angiography was performed in the patient whose chest pains did not respond to nitrate, and recurred. The council decision was made in the patient with 80% LAD osteal stenosis (Figure 1). Any valvular pathology was not observed in the echocardiography and left ventricular ejection fraction was found to be 60%. During the intensive care follow-up, chest radiography was performed on the patient due to ongoing chest pain and shortness of breath at rest. Bowel loops were observed in the right hemithorax on chest radiography (Figure 2). Thoracic and abdominal computed tomography (CT) was performed with the prediagnosis of diaphragmatic hernia detected in the patient, whose physical examination revealed no abnormality other than a marked decrease in respiratory sounds in the right hemithorax. In thorax CT, a 7 cm defect was detected in the right hemidiaphragm, causing herniation of the colon and omental fat tissue into the thorax (Figure 3). Since diaphragmatic herniation is also a surgical indication, it was decided to

perform coronary artery bypass graft surgery simultaneously. The right posterolateral diaphragmatic defect was repaired using a 5x10 cm poly-tetra-fluoro-ethylene fabric graft. Left internal mammary artery graft was applied to the LAD. On postoperative control radiography and CT, in the patient whose right lung was fully expanded (Figure 4), herniated organs have wre reduced to the abdomen, and the defect disappeared. This patient has been followed up for 3 years without any problem.

Discussion: The incidence of congenital diaphragmatic hernia is 0.02-0.05%. There are five types of diaphragmatic hernias: Bochdalek hernia, Morgagni hernia, diaphragm eventration, diaphragmatic herniation through the central tendon, and hiatus hernia. MH is usually asymptomatic, and patients are diagnosed either during routine radiological examination or when they present to the emergency department due to strangulation / volvulus of the organs within the hernia sac. The most common contents of the hernia sac are omentum and colon. Concomitant treatment with cardiac surgery is rarely described in the literature. When we look at the literature, Gopalakrishnan et al. described a large midline defect that caused hernia contents to enter into the pericardium during coronary revascularization, which was repaired with a prolene mesh before coronary revascularization. Surgical repair should be performed as soon as the diagnosis is confirmed to prevent serious complications in cases with congenital diaphragmatic hernia. Although rare, diaphragmatic hernias should be kept in mind in the differential diagnosis of chest pain.

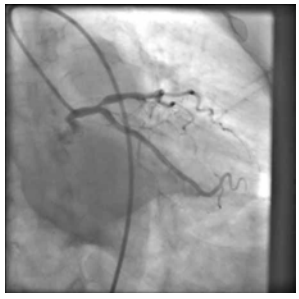

Figure 1. Coronary angiography: LAD osteal lesion.

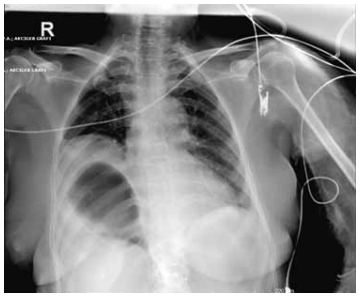

Figure 2. Preoperative chest radiography: Lung atelectasis and bowel loops due to diaphragmatic hernia in the right hemithorax.

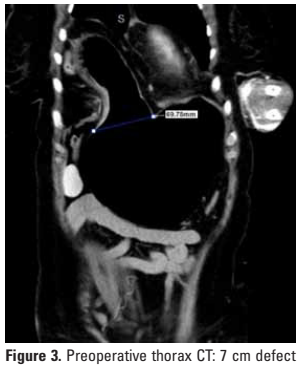

Figure 3. Preoperative thorax CT: 7 cm defect in the diaphragm.

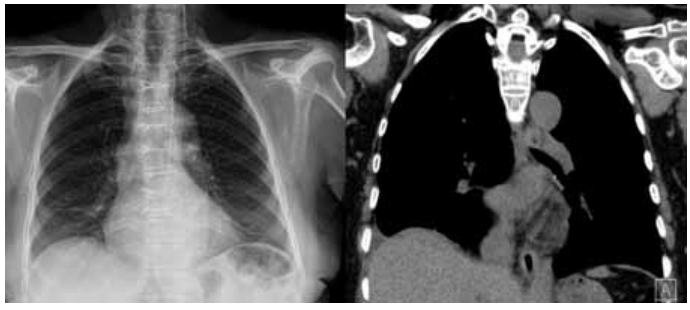

Figure 4. Postoperative chest radiography and thorax CT: Normally expanded right lung tissue and intact diaphragm.

Pulmonary hypertension / Pulmonary vascular diseases

PO-23

Advanced pulmonary hypertension secondary to dasatinib use due to CML

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Introduction: Pulmonary arterial hypertension (PAH) is a chronic and progressive cardio-pulmonary disease state in which the mean pulmonary artery pressure (mPAP) that is defined as precapillary pulmonary hypertension above 25 mmHg. Disruption of the mediator balance acting on the pulmonary arterial wall in the direction of proliferation and vasoconstriction leads to the development of clinic manifestations of PAH. Drug-related PAH is an example of this. In this case report, we aimed to present a patient with a history of pulmonary hypertension and recurrent pleural effusion due to the use of dasatinib, a tyrosine kinase inhibitor prescribed with the indication of chronic myeloid leukemia (CML) and follow-up method we used. We aimed to present our patient with a history of effusion and the method of follow-up.

Case Report: A 20-year-old male patient was admitted to the cardiology outpatient clinic because of progressively worsening shortness of breath persisting for 15 days. His general condition was good, and his vital signs were normal. In his physical examination, his heart sounds were rhythmic, S2 was loud and systolic murmur iwas heard on the tricuspid focus. Respiratory sounds were decreased bilaterally in the basilar regions of the lungs. He had been followed up with the diagnosis of CML for 7 years, so he was using dasatinib. Pleural effusion was detected in an external medical center and dasatinib was discontinued for a while and resumed after the effusion regressed. Right ventricular hypertrophy was present in the ECG of the patient. In the echocardiography performed, it was observed that EF was normal, right ventricular systolic functions were decreased, severe tricuspid regurgitation (TR) was present, and PAP calculated on the basis of maximum TR velocity was 105 mmHg. Increase in PAP persisted as detected in the control echocardiography of the patient whose volume load decreased with intravenous diuretic therapy. The patient, whose other tests for pulmonary hypertension etiologies were found to be normal, was evaluated as having dasatinib-associated pulmonary hypertension and the use of dasatinib was discontinued. The patient was discharged to continue follow-up on an ambulatory basis. In the control performed 3 weeks later, the patient's complaints were significantly alleviated. The severity of tricuspid insufficiency was reduced as revealed in echocardiography. His PAP was measured as 76 mmHg. Right ventricular systolic functions were increased. It was observed that PAP decreased to 65 mmHg after 3 months of control. The patient was followed up on an ambulatory basis clinically and echocardiographically in outpatient clinic of cardiology.

Discussion: Dasatinib is one of the second generation tyrosine kinase inhibitors that can be preferred in early chronic phase CML. Although the most common side effects are myelosuppression, nausea and vomiting, although it is rare, serious cardiac side effects such as significant fluid retention, pleural and pericardial effusion, dysrhythmia, pulmonary hypertension may develop. The first step in drug-associated pulmonary arterial hypertension is discontinuation of the drug. PAP may decrease and clinical condition of the patient may improve after discontinuation of the drug. Meanwhile, endothelin receptor blockers may be preferred to accelerate the recovery process. However, in some cases where dasatinib causes pulmonary hypertension, the clinical picture may continue irreversibly.

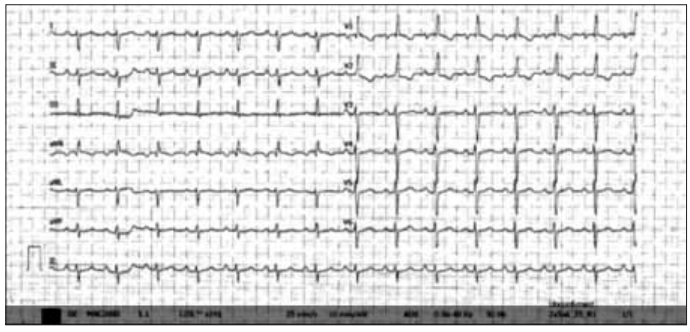

Figure 1. ECG; Right axis deviation and right ventricular hypertrophy.

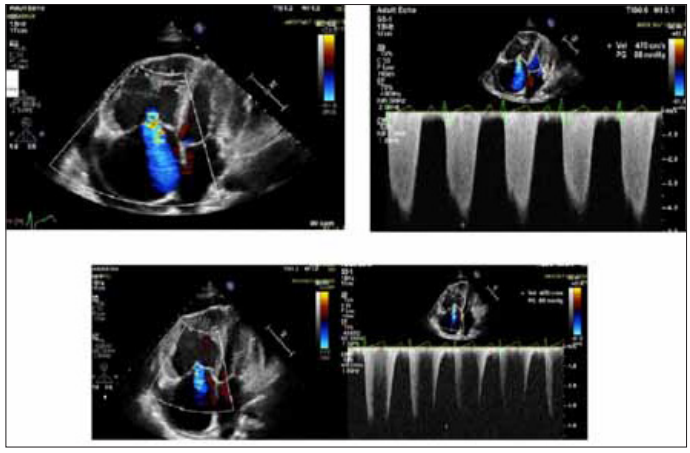

Figure 2. Comparative view of severe tricuspid insufficiency and PAP calculated over maximum TR velocity with the 2nd echocardiography performed after drug withdrawal and achievement of clinical euvolemia.

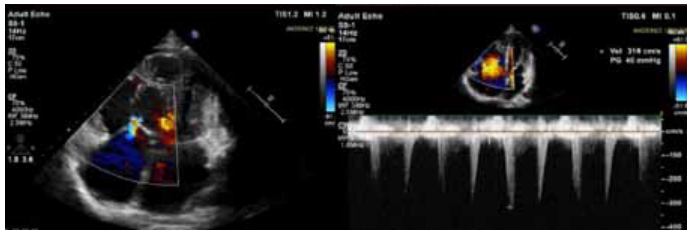

Figure 3. Mild tricuspid regurgitation and measurement of PAP as 65 mmHg in echocardiography performed at the 3rd month after discharge.