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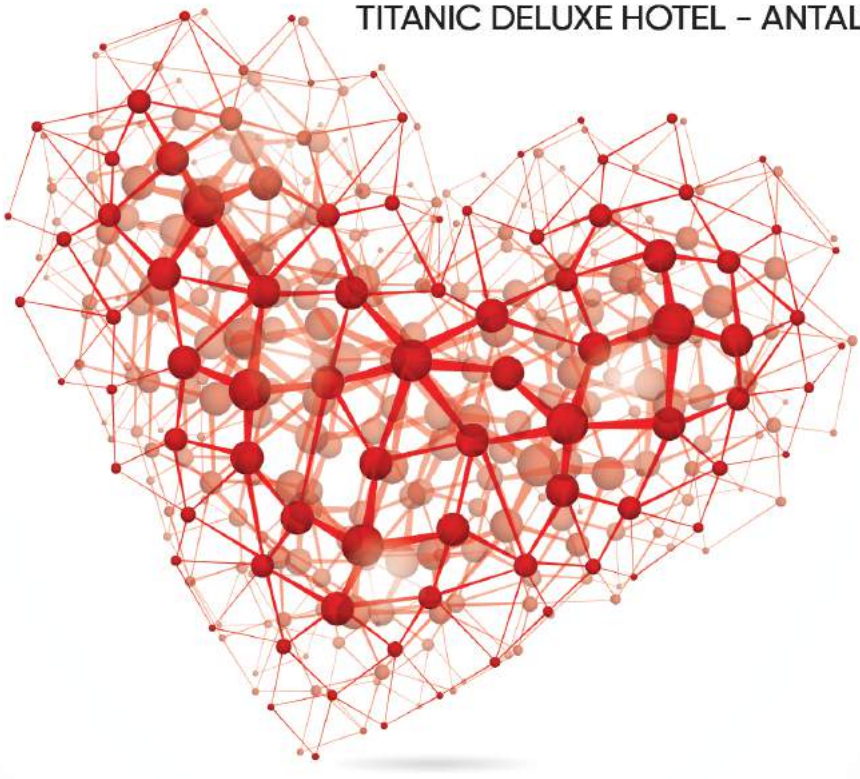

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THE ANATOLIAN JOURNAL OF CARDIOLOGY

THE EXECUTIVE BOARD OF TURKISH SOCIETY OF CARDIOLOGY

Dear Colleagues,

In addition to organizing various training programs and events throughout the year, the Turkish Society of Cardiology plans to hold the National Cardiology Congress in November, as always, at a level worthy of its 59th year.

The Society is planning to present the rich content of our congress, which is one of the leading scientific congresses at national and international level with both the number of participants and high quality scientific content, with a wide range of satisfying scientific programs that will appeal to all our participants.

Our goal of making our congress the leading meeting of the region in the field of Cardiology continues. We will update and discuss our latest knowledge on cardiovascular diseases through "Symposiums", "Contrasting View" and "How To" sessions. We expanded our "Cardiology in Daily Practice" sessions under the title of "Young Cardiologists Sessions" to cover the entire cardiology practice.

We will improve our skills as well as our knowledge with the certified "Interactive Courses", the number of which we have increased due to the intense interest in the past years. In each of our sessions, there are valuable speakers and panelists from Turkey and the world who are prominent scientists of their areas. We believe that our joint sessions with ESC, ACC, Turkish World Society of Cardiology, EACVI, EHRA and EAPCI will be watched carefully.

As always, our congress will be credited by the TSD.

We will be pleased to see you among us at our congress.

Looking forward to meeting with you all within the 38th Turkish Cardiology Congress on 10-13 November 2022, to share our knowledge,

With our best wishes and best regards,

Prof. Dr. Vedat Aytekin
President of TSC

Prof. Dr. Muzaffer Değertekin
President – Elect
Chairman of the Scientific Committee

THE ANATOLIAN JOURNAL OF CARDIOLOGY

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38th NATIONAL CARDIOLOGY CONGRESS

ORAL PRESENTATIONS

THE ANATOLIAN JOURNAL OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Coronary artery disease / Acute coronary syndrome

OP-001

Development and internal validation of diagnostic prediction model for myocardial infarction with non-obstructive coronary arteries (MINOCA)

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Background and Aim: Myocardial infarction (MI) with non-obstructive coronary arteries (MINOCA) is characterized by clinical evidence of MI with normal or near-normal coronary arteries on coronary angiography (CA). Cardiologists should consider MINOCA a working diagnosis, and be ready to start diagnostic processes such as left ventricular angiography (LVA), optical coherence tomography (OCT), intravascular ultrasound (IVUS), and coronary provocation tests during diagnostic CA. Therefore, predicting the patients who haa d possible diagnosis of MINOCA before diagnostic CA is precious for getting ready to provide these test. Prevalence and clinical profile of patients with MINOCA in Turkey (MINOCA-TR) Study, investigated demographic and clinical characteristics of these patients in Turkish healthcare settings. The aim of this study was to development a prediction model for MINOCA based on the MINOCA-TR Study database.

Methods: The association between candidate predictors and outcome was assessed by fitting penalized maximum likelihood estimation logistic regression. The association between candidate predictors and outcomes was quantified by the adjusted odds ratio with a 95% confidence interval. To capture non-linear association, age was modelled by using restricted cubic spline transformations (3 knots). The relative importance of each predictor in the model was estimated by their partial X2 values and graphically displayed. A nomogram was built for estimating the probability of MINOCA as a graphical representation of the multivariable risk prediction models. Then the nomograms were used to construct a risk calculator.

Results: 1626 patients were enrolled to the MINOCA-TR Study. MINOCA prevalence was 6.7% (n=109). In the full model, 14 predictors derived from data were included in the multivariable models. Simple models were constructed with a back-ward step-down variable selection (10 predictors, alpha=0.25). Table 1 summarizes adjusted odds ratios for individual predictors in full and simple models. The (younger) age, (female) sex, (admission patterns other than) STEMI and (absence of history of) previous antiplatelet use were found to be strong predictors for MINOCA based on their partial X2 (Figure 1, Panel A). Overall discriminative ability of the model was excellent (AUC: 0.875, 95% CI 0.838–0.904). The R2 of model was 0.350. The bootstrap resampling was performed to assess internal validations. There was negligible model optimism (bootstrapped AUC 0.859 and R2: 0.318). Calibration plot were given in Figure 1, Panel B. There was a good agreement between predicted and observed frequency of MINOCA. We demonstrated our nomogram in Figure 1, Panel C.

Conclusions: We developed and internally validated model that accurately predicts the presence of MINOCA. The model may have a potential to ease management of patients with ACS via obtaining personalized risk predictions after external validation with prospectively designed epidemiological studies.

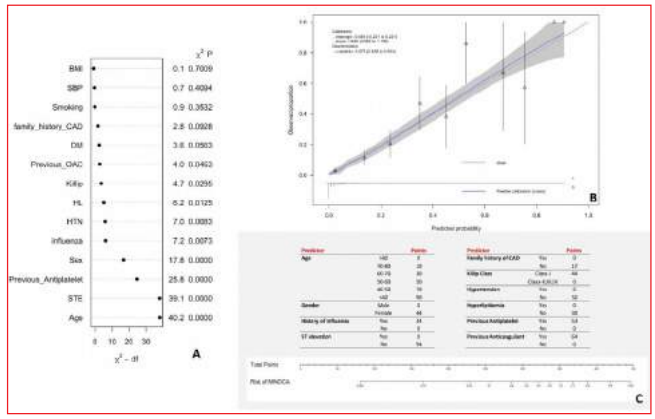

Figure 1. Panel A, strong predictors for MINOCA based on their partial X2. Panel B, Calibration plot. Panel C, The nomograms representing a graphical tool for estimating the probabilities of MINOCA based on the coefficients from a simple multivariable regression model. In the nomograms, the categories of each factor are assigned a score (points), then all scores are summed up to obtain the total points, which relate to the predicted outcomes.

	Full model Adjusted OR, 95% CI	Simple Model Adjusted OR, 95% CI
Age, years (from 53 to 71 year)	0.47 (0.33 – 0.67)	0.49 (0.35 – 0.69)
Sex, female	3.01 (1.81 – 5.03)	3.19 (1.96 – 5.19)
BMI, (from 24.6 to 29.8 kg/m²)	1.05 (0.82 – 1.35)	-
SBP, (from 110 to 143 mmHg)	0.86 (0.60 – 1.23)	-
Smoking, yes	0.77 (0.45 – 1.34)	-
Influenza history, yes	2.23 (1.24 – 4.02)	2.38 (1.32 – 4.29)
STEMI, yes	0.08 (0.03 – 0.17)	0.07 (0.03 – 0.17)
Family history CAD, yes	0.64 (0.37 – 1.08)	0.62 (0.36 – 1.05)
HTN, yes	0.50 (0.30 – 0.84)	0.44 (0.27 – 0.73)
DM, yes	0.56 (0.31 – 1.01)	-
HL, yes	0.50 (0.29 – 0.86)	0.47 (0.27 – 0.82)
Killip II-IV, yes	0.30 (0.10 – 0.89)	0.26 (0.09 – 0.79)
Previous Antiplatelet use, yes	4.14 (2.39 – 7.17)	4.30 (2.50 – 7.39)
Previous OAC, yes	4.20(1.02 – 17.3)	4.66 (1.14 – 18.9)

Coronary artery disease / Acute coronary syndrome

OP-003

Long-term observational study of the isolated osteal diagonal stenosis in patients with chronic coronary syndrome

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Background and Aim: Isolated ostial diagonal stenoses are very rare lesions in which percutaneous intervention could cause significant vessel compromise, and the long-term results have been reported in a few studies. This study sought the characteristics and long-term follow-up of the patients with isolated osteal diagonal stenosis regarding PCI and presence of angina.

Methods: This was an observational retrospective study conducted between January 2014 and December 2020. A total of 9769 patients who underwent coronary angiography were analyzed, and eighty-seven patients had isolated diagonal stenosis. The patients were evaluated according to treatment modality and angina severity in long-term pattern.

Results: Median follow-up time was thirty-six months. A total of fifty-four (83.1%) patients were followed up with only medical treatment, and eleven (16.9%) patients underwent revascularization in addition to medical treatment. The degree of stenosis of the diagonal artery was significantly higher in the PCI group than medical group (p=0.002) and the patients with wider reference diameter of diagonal artery were complaint of more angina (p=0.007). There was no significant difference between the two groups in terms of recurrent angiography and long-term angina severity.

Conclusions: PCI was mainly performed to diagonal arteries with a higher degree of stenosis; however, angina severity did not differ in the long-term between the only medical group and the medical plus PCI group. Furthermore, the patients with ongoing angina had a larger diameter of the diagonal artery regardless of the type of treatment.

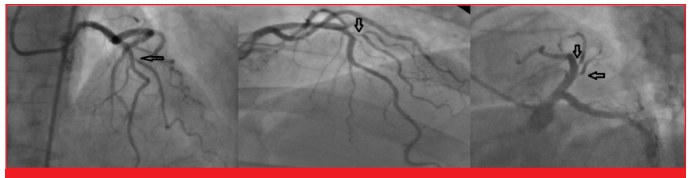

Figure 1. Angiography

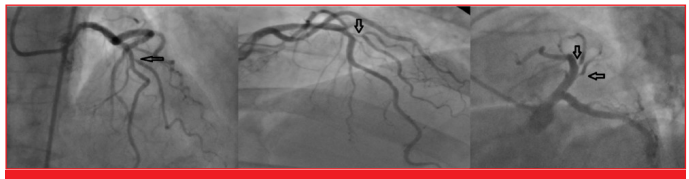

Figure 2. Osteal stenosis PCI

Table 1. Comparison of the demographic and clinical characteristics of the patients according to treatment groups

Variable	Medical Group (n=54)	PCI Group (n=11)	p value
Age, years (mean ± SD)	61 ± 11.5	65 ± 7.6	0.611
Male gender, n (%)	36 (66.7)	6 (54.5)	0.443
Body mass index, kg/m^2 (mean ± SD)	26.7 ± 11.4	29.8 ± 3.7	0.387
Canada angina classification, n (%)			**0.002**
I	40 (74.0)	3 (27.2)	
II	11 (20.3)	5 (45.4)	
III	3 (5.5)	3 (27.2)	
IV	0	0	
Smoking, pack/year, (mean± SD)	28.9 ± 13.2	30.3 ± 12.9	0.818
Smoking, n (%)			0.918
Current	8 (14.8)	2 (18.2)	
Quit	23 (42.6)	4 (36.4)	
Never	23 (42.6)	5 (45.5)	
Hypertension, n (%)	31 (57.4)	6 (54.5)	0.861
Diabetes mellitus, n (%)	23 (42.6)	4 (36.4)	0.702
Hyperlipidemia, n (%)	22 (40.7)	4 (36.4)	0.787
Chronic renal failure, n (%)	4 (7.4)	0	-
Family history, n (%)	18 (33.3)	4 (36.4)	0.846
Peripheral artery disease, n (%)	11 (20.4)	0	-
Positive Exercise ECG	20 / 27 (74.1%)	5/7 (71.4%)	0.887
Ischemia in myocardial perfusion scintigraphy, n (%)	17/24 (70.8%)	4/6 (66.7%)	0.842
FFR value < 0.80	0/2	-	-

*SD, standard deviation; FFR, fractional flow reverse

Table 2. Index angiographic findings of the patients according to treatment groups

Variable	Medical Group (n=54)	PCI Group (n=11)	p value
Diagonal artery diameter, mm (median, IQR)	2.25 (0.50)	2.25 (0.25)	0.082
Percent stenosis, % (median, IQR)	60 (45)	80 (20)	**0.002**
Diagonal artery branches, n (%)			0.654
First branch	32 (59)	7 (63)	
Second branch	16 (30)	3 (29)	
Both branches	6 (11)	1 (9)	

*IQR, interquartile range

Table 3. Comparison of patients' follow-up data and medical treatment among the groups

Variable	Medical (n=54)	PCI (n=11)	p value
Acetylsalicylic acid, n (%)	41 (75.9)	8 (72.7)	0.822
P2Y12 receptor inhibitors, n (%)	7 (13.0)	3 (27.3)	0.231
Anticoagulants, n (%)	4 (7.4)	2 (18.2)	0.260
ACEI /ARB, n (%)	30 (55.6)	10 (90.9)	**0.028**
Beta blocker, n (%)	26 (48.1)	10 (90.9)	**0.009**
Lipid lowering drugs, n (%)	25 (46.3)	7 (63.6)	0.294
Nitrate, n (%)	5 (9.3)	2 (18.2)	0.384
Ivabradine, n (%)	0	0	
Ranolazine, n (%)	9 (16.7)	1 (9.1)	0.526
Trimetazidine, n (%)	9 (16.7)	2 (18.2)	0.903
Calcium channel blockers, n (%)	6 (11.1)	1 (9.1)	0.844
Follow-up time, months (median, IQR)	33.5 (36)	40 (28)	0.134
Recurrent angiography, n (%)	19 (35.2)	5 (45.5)	0.520
Anjina class (CCS), n (%)			0.106
0	33 (61.1)	3 (27.3)	
I	17 (31.5)	7 (63.6)	
II	4 (7.4)	1 (9.1)	
III-IV	0	0	

*CCS, Angina according to the Canadian Heart Association classification; ACEIs, angiotensin converting enzyme inhibitors; ARB, angiotensin receptor blockers; SD, standard deviation: IQR, interquartile range.

Table 4. Assessment of the patients according to angina presence during follow-up

Variable	Patients with angina (n=29)	Patients without angina (n=36)	p value
Age, year (mean ± SD)	61 ± 9.4	61.8 ± 12.2	0.864
BMI, kg/m^2	27 ± 9.2	27.4 ± 11.6	0.858
Male gender, n (%)	19 (65.5)	23 (63.9)	0.891
Follow up, months (mean ± SD)	33.8 ± 22.7	33.9 ± 24.9	0.858
Hypertension, n (%)	14 (48.3)	23 (63.9)	0.206
Diabetes Mellitus, n (%)	9 (31)	18 (50)	0.123
Hyperlipidemia, n (%)	12 (41.4)	14 (38.9)	0.839
Smoking, n (%)			0.879
Current	4 (13.8)	6 (16.7)	
Quit	13 (44.8)	14 (38.9)	
Never	12 (41.4)	16 (46.7)	
Family history, n (%)	11 (37.9)	11 (30.6)	0.532
Peripheral artery disease, n (%)	4 (13.8)	7 (19.4)	0.546
Chronic kidney disease, n (%)	1 (3.4)	3 (8.3)	0.415
Medical treatment, n (%)			
Acetylsalicylic acid	21 (72.4)	28 (77.8)	0.618
P2Y12 inhibitor	5 (17.2)	5 (13.9)	0.710
Anticoagulants	4 (13.8)	2 (5.6)	0.254
Lipid lowering drugs	16 (55.2)	16 (44.4)	0.390
ACEI / ARB	17 (58.6)	23 (63.9)	0.664
Beta blocker	19 (65.5)	17 (47.2)	0.140
Nitrate	2 (6.9)	5 (13.9)	0.366
Ivabradine	0	0	-
Ranolazine	4 (13.8)	6 (16.7)	0.750
Trimetazidine	4 (13.8)	7 (19.4)	0.546
Calcium channel blocker	3 (10.3)	4 (11.1)	0.921
Recatheterization, n (%)	12 (41.4)	12 (33.3)	0.504
Diagonal diameter, (median, IQR)	2.25 (0.25)	2.00 (0.50)	**0.007**
Percentage of diagonal stenosis, (median, IQR)	70 (20)	70 (25)	0.462

*ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; SD, standard deviation; IQR, interquartile range.

Coronary artery disease / Acute coronary syndrome

OP-004

Effects of sodium-glucose co-transporter 2 inhibition on renal functions following an acute coronary syndrome in patients with type 2 diabetes

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Background and Aim: Sodium-glucose cotransporter 2 inhibitors (SGLT-2i) are a unique class of oral glucose-lowering agents that reduce glucose reabsorption in the renal proximal tubes. Although SGLT-2i treatment leads to a reduction of 10–20% in the number of myocardial infarction (MI) in diabetic patients, clinical data regarding to usage after acute coronary syndrome (ACS) is sparse. Experimental data highlights a reduction in infarct size and consecutively less remodeling and development of HF after MI. To date, no published human studies have investigated the effect of SGLT-2i on renal function in the early post-ACS period.

Methods: This prospective observational study was conducted at an tertiary hospital cardiology unit during 01.2021-05.2022. Consecutive patients admitted for an ACS with a history of T2D were identified during hospital admission. Exclusion criteria included age < 20 years or > 80 years, history of diabetic ketoacidosis, type 1 diabetes, estimated glomerular filtration rate (eGFR) < 15 ml/min/1.73m^2, pregnancy, and inability to attend follow-up. Eligible patients were divided into three groups: patients who were on SGLT-2i treatment prior to ACS (group 1), patients who has just started SGLT-2i therapy in hospital (group 2), and patients who were not initiated on a SGLT-2i were used as a comparison group (group 3). The primary end point was acute kidney injury (AKI) development which is usually defined as a rise in serum creatinine of ≥ 0.5 mg/dL or a 25% increase from baseline assessed within 48 hours after coronary angiography. The secondary outcomes were differences in change between groups in renal function tests (creatinine, eGFR) during three months follow-up.

Results: The baseline characteristics of the 108 participants included in this study are summarized in Table 1. The mean duration of T2D at study entry was 10.6 ± 7.4 years; glycated hemoglobin was 8.3 ± 1.9% and 19 (17.6%) patients were newly diagnosed. Most of the patients (93 patients, 86.1%) were presented with MI (53, 49.1% NSTEMI; 40, 37.0% STEMI) and primary PCI were performed (94 patients, 87.0%). Baseline characteristics on admission were similar between the groups. There were no significant differences between groups for baseline medications (Table 2). The primary end point, AKI was encountered in 8 of 108 patients (7.4%): 1 (10%) in group 1, 3 (6.1%) in group 2 and 4 (4.8%) in group 3; p=0.61 (Table 3). The creatinine elevation persisted in only 2 patients during follow-up. There was no significant difference between the treatment groups at any interval. No patient required hemodialysis in either group.

Conclusions: To our knowledge, the present observational study is the first to examine the potential effect of SGLT-2i on renal functions in the early post-ACS period. In patients with ACS and T2D, addition of SGLT-2i to standard ACS medical therapy was not associated with AKI development or renal impairment at follow-up when compared with patients who were not initiated on a SGLT-2i.

Table 1. Baseline characteristics according to SGLT-2i treatment

Characteristic	Previous SGLT-2i users (n=10)	Newly started Empagliflozin (n=31)	Newly started Dapagliflozin (n=18)	SGLT2i Naïve (n=49)	P
Age, years	66.5 ± 7.1	63.1 ± 9.4	58.8 ± 8.9	62.4 ± 9.5	0.29
Male gender, n (%)	4 (40)	21 (67.7)	11 (61.1)	35 (71.4)	0.27
BMI (kg/m^2)	29.2 ± 3.9	29.7 ± 3.6	28.9 ± 4.1	29.1 ± 3.8	0.56
Diabetes duration	10.0 ± 5.1	10.4 ± 8.0	6.0 ± 5.5	12.1 ± 7.8	0.37
Diabetes newly diagnosed	0	7 (22.6)	5 (27.8)	7 (14.3)	0.23
Admission diagnosis UAP NSTEMI STEMI	 2 (20) 5 (50) 3 (30)	 2 (6.5) 17 (54.8) 12 (38.7)	 2 (11.1) 9 (50.0) 7 (38.9)	 9 (18.4) 22 (44.9) 18 (36.7)	0.82
Treatment Medical PCI CABG	 1 (10.0) 9 (90.0) 0	 0 30 (96.8) 1 (3.2)	 1 (5.6) 16 (88.9) 1 (5.6)	 7 (14.3) 39 (79.6) 3 (6.1)	0.38
Known Hypertension	9 (90.0)	24 (77.4)	11 (61.1)	38 (79.2)	0.31
Known dyslipidemia	5 (50)	11 (36.7)	8 (47.1)	21 (42.9)	0.85
Ever smoker	5 (50%)	20 (64.5%)	9 (50%)	25 (51%)	0.89
Prior PCI or CABG	4 (40) / 2 (20)	9(29.0)/3(9.7)	9 (50)/0	14 (28.6) / 8 (16.3)	NS
Documented CKD	0	4 (13.3)	2 (11.2)	10 (20.4)	0.36
HbA1c (%)	8.3 ± 1.6	8.5 ± 1.5	9.3 ± 2.1	7.9 ± 1.9	0.08
NT-proBNP (ng/L)	250.0 ± 311.0	266.3 ± 341.7	300.6 ± 367.9	312.9 ± 354.1	0.95
Total cholesterol (mg/dL)	214.5 ± 60.0	206.3 ± 53.9	206.3 ± 83.7	202.9 ± 59.2	0.99
HDL-C (mg/dL)	42.1 ± 8.5	40.7 ± 10.3	39.1 ± 8.1	41.4 ± 10.8	0.85
LDL-C (mg/dL)	109.5 ± 21.1	136.2 ± 41.2	118.4 ± 34.5	120.5 ± 37.7	0.14
Triglycerides (mg/dL)	186.6 ± 75.5	187.5 ± 124.4	178.2 ± 76.6	180.0 ± 90.7	0.99
Haemoglobin (g/L)	13.1 ± 3.1	13.7 ± 2.3	13.8 ± 1.9	13.8 ± 1.8	0.83

Data are presented as count (percentage), n (%).

Table 2. Medications on admission and at discharge according to SGLT-2i use

Medications	Previous SGLT-2i users (n=10) Admission	Previous SGLT-2i users (n=10) Discharge	Newly started Empagliflozin (n=31) Admission	Newly started Empagliflozin (n=31) Discharge	Newly started Dapagliflozin (n=18) Admission	Newly started Dapagliflozin (n=18) Discharge	SGLT-2i Naïve (n=49) Admission	SGLT-2i Naïve (n=49) Discharge
ACEi or ARB	6 (60)	8 (80)	17 (54.8)	30 (96.8)	9 (50)	16 (88.9)	25 (51)	43 (87.8)
Beta-blocker	7 (70)	10 (100)	12 (38.7)	30 (96.8)	8 (44.4)	16 (88.9)	20 (40.8)	40 (83.3)
MRA	1 (10)	1 (10)	0	3 (9.7)	3 (16.7)	5 (27.8)	5 (10.2)	7 (14.3)
CCB	5 (50)	6 (60)	9 (29)	9 (29)	3 (16.7)	2 (11.1)	15 (30.6)	15 (30.6)
Loop diuretic	3 (30)	2 (20)	0	3 (9.7)	1 (5.6)	3 (16.7)	2 (4.1)	7 (14.3)
Thiazide diuretic	3 (30)	3 (30)	10 (32.3)	10 (32.3)	3 (16.7)	3 (16.7)	17 (34.7)	24 (49.0)
Statin	5 (50)	9 (90)	6 (19.4)	29 (93.5)	9 (50)	16 (88.9)	12 (24.5)	47 (95.9)
Metformin	8 (80)	9 (90)	17 (54.8)	24 (77.4)	8 (44.4)	15(83.3)	31 (63.3)	36 (73.5)
Sulfonylurea	8 (80)	6 (60)	8 (25.8)	4 (12.9)	3 (16.7)	3 (16.7)	7 (14.3)	9 (18.4)
DPP4 inhibitor	7 (70)	8 (80)	5 (16.1)	10 (32.3)	7 (38.9)	12 (66.7)	17 (34.7)	23 (46.9)
Thiazolidinediones	0	0	2 (6.5)	2 (6.5)	1 (5.6)	1 (5.6)	2 (4.1)	2 (4.1)
AGI	2 (20)	2 (20)	2 (6.5)	2 (6.5)	2 (11.1)	0	1 (2)	0
Insulin	1 (10)	5 (50)	5 (16.1)	10 (32.3)	5 (27.8)	8 (44.4)	10 (20.4)	15 (30.6)
GLP-1 RA	0	0	0	0	1 (5.6)	0	0	0

Data are presented as count (percentage), n (%). No significant differences between groups in medications at admission No significant differences in the proportion of new prescriptions during admission between groups

A, Admission, D, Discharge, SGLT-2i, sodium-glucose cotransporter 2 inhibitor, ACEi, angiotensin converting enzyme inhibitor, ARB, angiotensin receptor blocker, MRA, mineralocorticoid receptor antagonist, CCB, Calcium-channel blocker, DPP4, dipeptidyl peptidase 4, AGI, α-glucosidase inhibitors, GLP-1 RA, glucagon like peptide-1 receptor agonist.

Table 3. Renal functions at admission, discharge and follow-up according to SGLT-2i treatment

Characteristic	Previous SGLT-2i users (n=10)	Newly started Empagliflozin (n=31)	Newly started Dapagliflozin (n=18)	SGLT2i Naïve (n=49)	P
Creatinine Admission Discharge Follow-up	0.86 ± 0.13 0.87 ± 0.20 0.82 ± 0.12	0.95 ± 0.30 0.98 ± 0.33 0.98 ± 0.29	0.98 ± 0.34 0.95 ± 0.28 1.14 ± 0.67	1.11 ± 0.58 1.14 ± 0.65 1.21 ± 0.76	0.82
eGFR Admission Follow-up	80.5 ± 13.7 83.0 ± 9.1	80.5 ± 22.4 77.3 ± 23.0	80.5 ± 24.5 77.9 ± 27.9	76.5 ± 26.7 70.5 ± 26.2	0.52
AKI	1 (10)	3 (9.7)	0	4 (8.2)	0.61

Data are presented as count (percentage), n (%). AKI, Acute kidney injury

Coronary artery disease / Acute coronary syndrome

OP-005

The effects of Benfotiamin in experimental acute myocardial infarction

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[2]*Department of Cardiology, Fırat University Faculty of Medicine, Elazığ*

Background and Aim: Myocardial Infarction (MI) is a group of disease that causes the most deaths in cardiovascular diseases. Isoproterenol (ISO) is a -adrenergic agent, which is commonly used for induction of experimental acute myocardial infarction model. Benfotiamine is a fat-soluble form of vitamin B1 and is known to have antioxidant effects. In this study, we aimed to investigate the effects of Benfotiamine, which has antioxidant properties on heart tissues of rats with ISO-induced acute myocardial infarction.

Methods: Twenty-four 8-week-old male Wistar rats were used in this study. Animals were divided into 4 different groups (6 animals /group). The control group (Group I) received no treatment during the 14 days. The Benfotiamine group (Group II) received oral Benfotiamine at a dose of 70 mg/kg/day. The MI group (Group III) and MI + Benfotiamine group (Group IV) received intraperitoneal isoproterenol at a dose of 150 mg/kg 2 times with 24-hour interval. MI + Benfotiamine group was given oral 70 mg/kg/day Benfotiamine, while MI group received no treatment.

Results: At the end of the study, all rats were decapitated, and heart tissues were removed under anesthesia. Heart tissues were examined immunohistochemically by Haematoxylin & Eosin, Masson's trichrome, Bax and Caspase-3 and TUNEL staining. Immunohistochemical staining revealed a marked increase in Bax and Caspase-3 immunoreactivity in MI group compared to control and Benfotiamine groups and decrease was observed in MI + Benfotiamine group. TUNEL staining revealed increased apoptotic cells in MI group compared to the control and Benfotiamine groups and a decrease was observed in MI + Benfotiamine group.

Conclusions: In conclusion, we observed protective effects of Benfotiamine against cellular changes in experimental acute myocardial infarction and so Benfotiamine may be a useful agent to challenge MI associated complications.

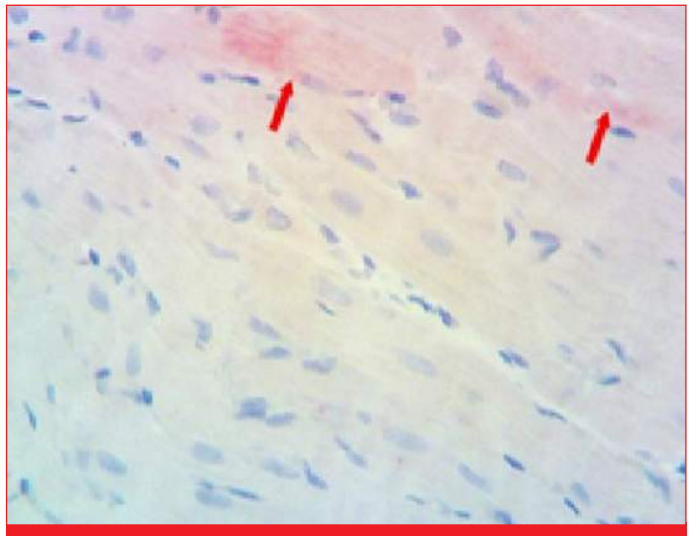

Figure 1. +1 Caspase-3 immunoreactivity in heart tissue of Benfotiamine group.

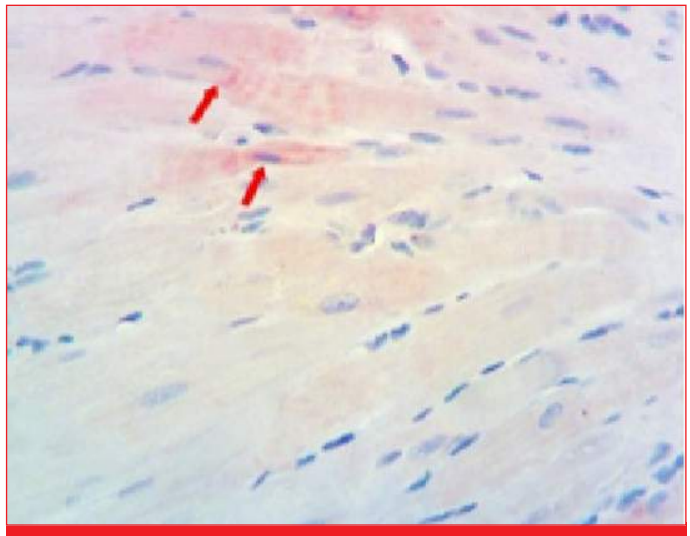

Figure 2. +1 Caspase-3 immunoreactivity in heart tissue of control group.

Figure 3. +1 TUNEL positivity in heart tissue of MI + Benfotiamine group.

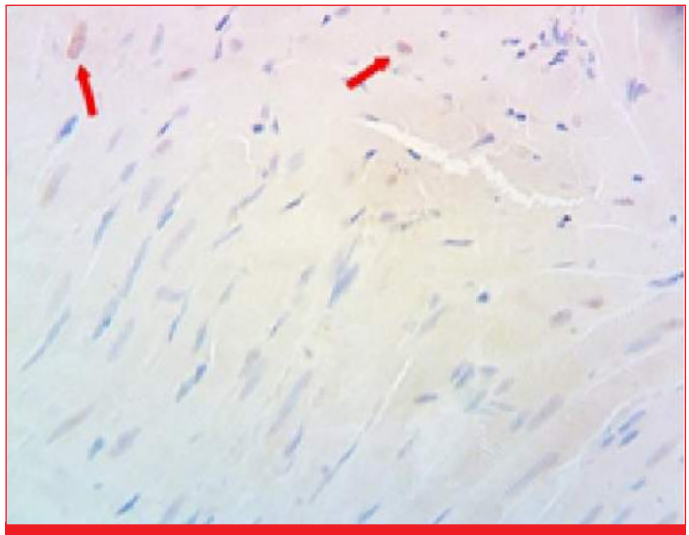

Figure 4. +1 TUNEL positivity in heart tissue of the Benfotiamine group.

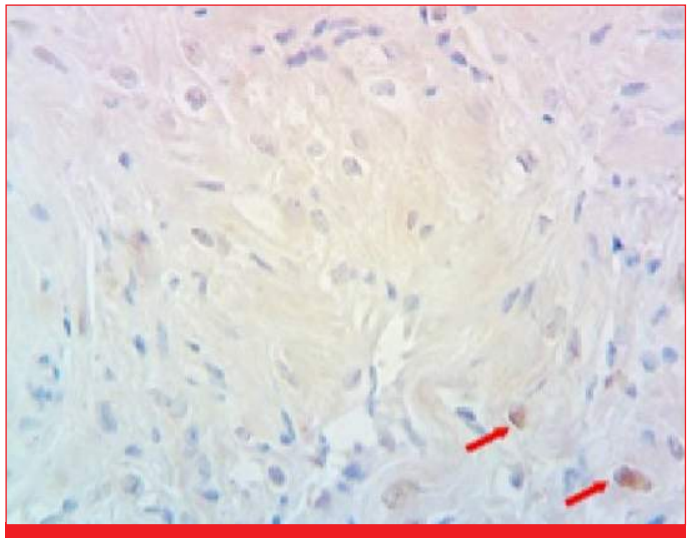

Figure 5. +1 TUNEL positivity in heart tissue of the control group.

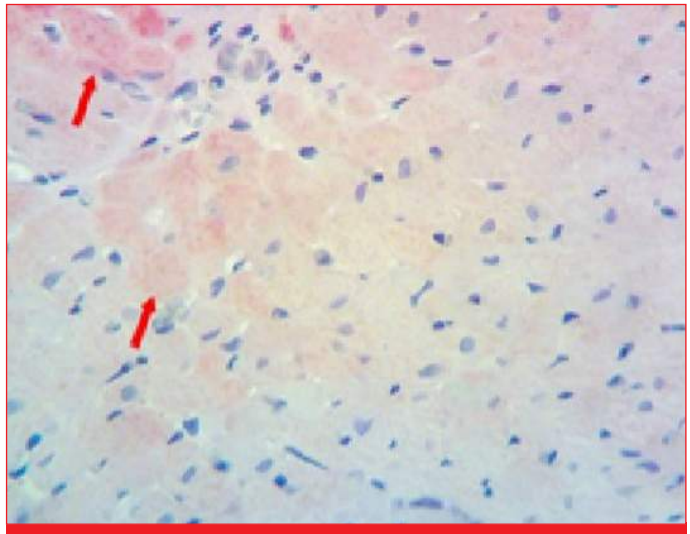

Figure 6. +2 Caspase-3 immunoreactivity in heart tissue of MI +Benfotiamine group.

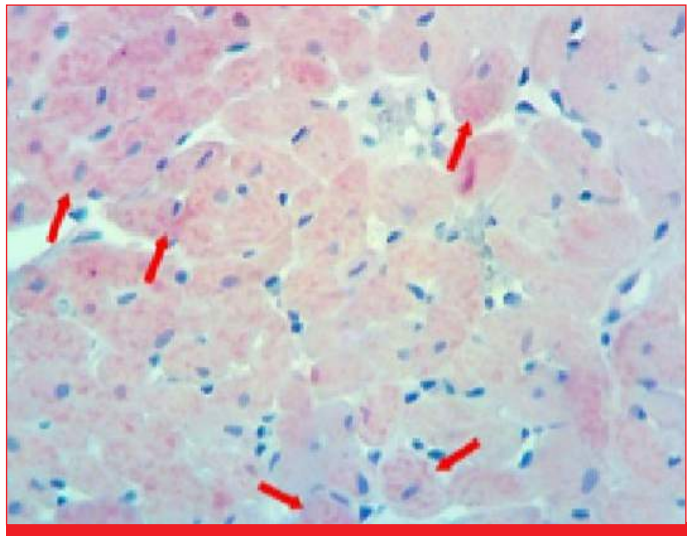

Figure 7. +3 Caspase-3 immunoreactivity in heart tissue of MI group.

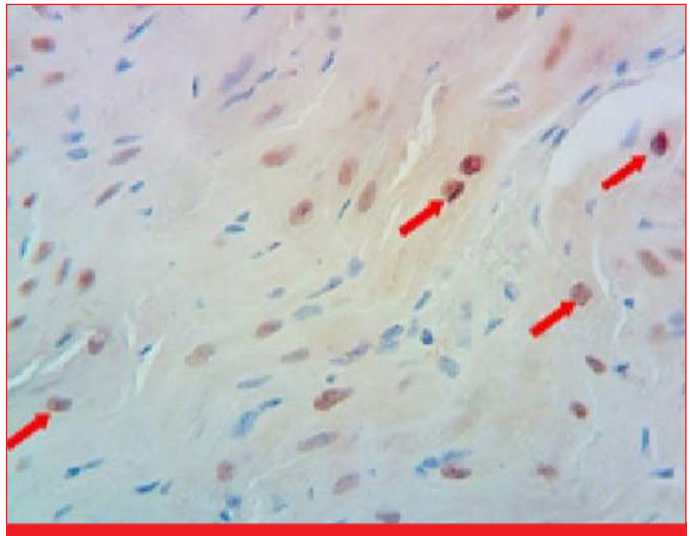

Figure 8. +3 TUNEL positivity in heart tissue of MI group

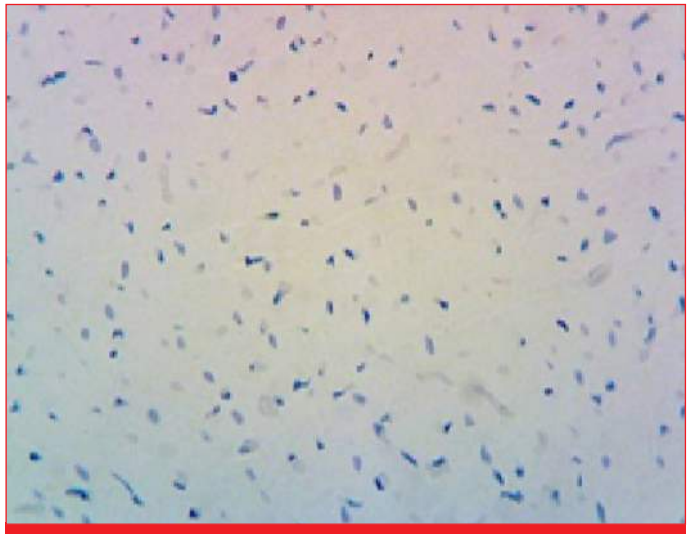

Figure 9. TUNEL negative control

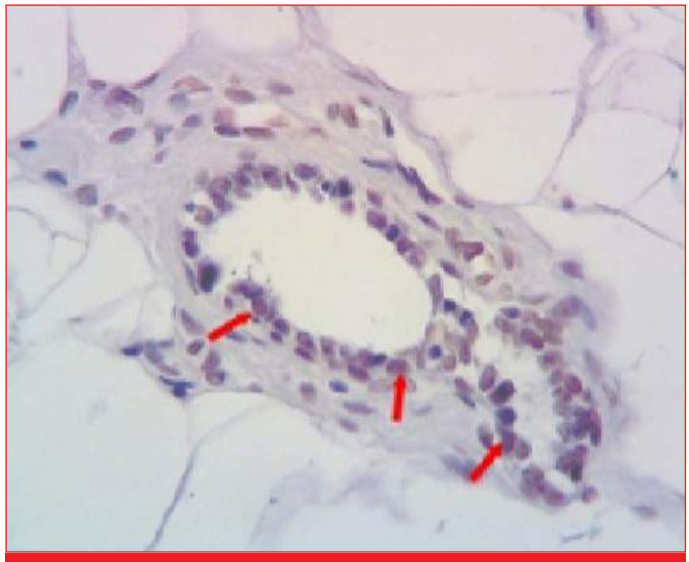

Figure 10. TUNEL positive control. Breast tissue.

Table 1. The degree of immunohistochemical staining extent

Degree	Meaning
0	No
+1	Mild
+2	Moderate
+3	Severe

Coronary artery disease / Acute coronary syndrome

OP-006

Door to balloon time of non-STEMI may be reconsidered according to systemic immune-inflammation index

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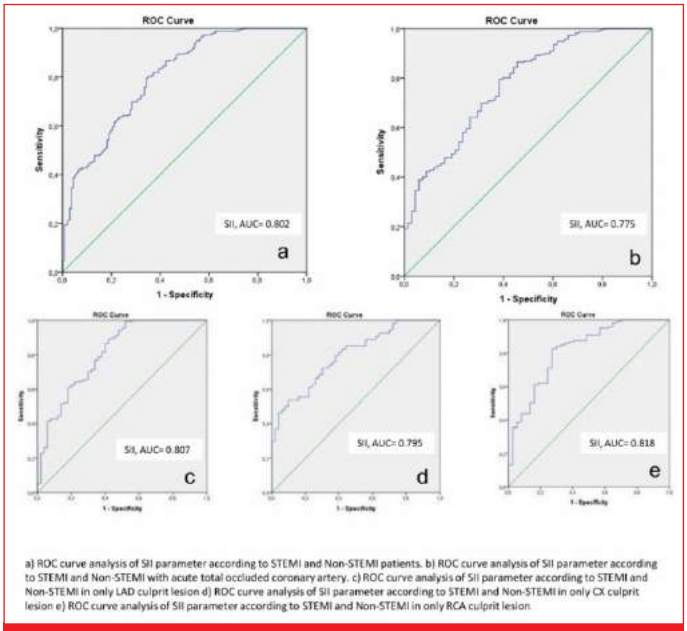
Background and Aim: Early diagnosis and treatment is very important in acute coronary syndromes (ACS). ST segment elevation myocardial infarction (STEMI) patients are recommended to intervene in 120 minutes after first medical contact however, non-STEMI patients are recommended to intervene in 24 hours. Previous studies showed that not all non-STEMI patients should be considered and treated in the same way. Systemic immune-inflammation index (SII) that is easy accessible, fast resultant and cost-effective parameter was studied in this study to reconsider the optimal intervention time for non-STEMI.

Methods: 469 patients diagnosed with ACS were included to the study. STEMI and non-STEMI patients were compared according to SII. In addition, non-STEMI with acute total occluded coronary arteries (ATOCA) and non-ATOCA patients were also compared. Univariate and binary logistic regression analysis were performed to find out which parameters have significant effect on the discrimination of MI types. All patients were followed-up for a specific time for survival analysis. The Kaplan–Meier curve for survival analysis was plotted to assess the prognosis between subgroups, divided according to the ROC curve cut-off points with the log-rank test.

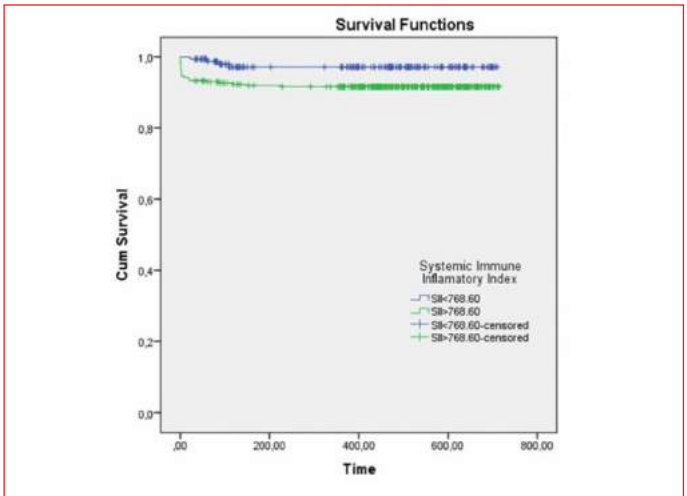
Results: The mean age was 61.43 ± 11.52 and 348 (74.2%) were male. 332 (70.8%) patients were diagnosed as STEMI. STEMI patients had higher SII value than non-STEMI ($p<0.001$). Non-STEMI patients with the SII value higher than 768.6 x 109 may be assumed as STEMI ($p<0.001$). In addition, STEMI and non-STEMI with ATOCA patients were compared with ROC analysis according to SII and interestingly, cut-off point was 768.60 x 109/L (sensitivity= 79.5% and specifity= 65.7%), similar to the previous one. Univariate analysis and binary logistic regression showed that only SII and hypertension had statistically impact on differentiation of STEMI and non-STEMI. Mean follow-up time was 436.94 ± 208.78 days and 30 (6.4%) patients were died as a result of any cardiovascular reason. ROC curve analysis showed that, SII value of 1105.23 x 109/L is the cut-off point (sensitivity= 83.3% and specifity= 57.9%) for discrimination of cardiovascular survivals and those who died from cardiovascular causes with statistically significance ($p<0.001$, AUC= 0.741).

Conclusions: This study was performed to find out which Non-STEMI patients should be treated percutanously immediately after first medical contact according to SII. It was found that, SII value of higher than 768.60 x 109/L is related with STEMI. In addition, subgroup of Non-STEMI with ATOCA were compared with STEMI patients and same cut-off value (768.60 x 109/L) was found according to ROC curve analysis. In conclusion, non-STEMI patients with SII value higher than 768.6 x 109 may be considered as STEMI and treated in 120 minutes after first medical contact. In addition, SII was found a cardiovascular mortality predictor after myocardial infarction and this may be used for identifying high-risk patients after PCI treatment.



a) ROC curve analysis of SII parameter according to STEMI and Non-STEMI patients. b) ROC curve analysis of SII parameter according to STEMI and Non-STEMI with acute total occluded coronary artery. c) ROC curve analysis of SII parameter according to STEMI and Non-STEMI in only LAD culprit lesion d) ROC curve analysis of SII parameter according to STEMI and Non-STEMI in only CX culprit lesion e) ROC curve analysis of SII parameter according to STEMI and Non-STEMI in only RCA culprit lesion

Figure 1. ROC curve analysis of SII parameter according to STEMI and Non-STEMI patients. b) ROC curve analysis of SII parameter according to STEMI and Non-STEMI with acute total occluded coronary artery. c) ROC curve analysis of SII parameter according to STEMI and Non-STEMI in only LAD culprit lesion d) ROC curve analysis of SII parameter according to STEMI and Non-STEMI in only CX culprit lesion e) ROC curve analysis of SII parameter according to STEMI and Non-STEMI in only RCA culprit lesion



Kaplan-Meier survival curves of long-term mortality according to SII cut-off value (Log Rank: p=0.018, Breslow: p=0.015 and Tarone-Ware:p=0.016

Figure 2. Kaplan-Meier survival curves of long-termmortality according to SII cut-off value (Log Rank: p=0.018, Breslow: p=0.015 and Tarone-Ware: p=0.016)

Table 1. Comparison of baseline demographic data and blood parameters according to myocardial infarction types.

Variable	STEMI	Non-STEMI	P
Age, year	61.92 ± 11.95	60.22 ± 10.35	0.123
Male, n (%)	249 (53.1%)	230 (21.1%)	0.563
Smoking (Exist), n (%)	171 (36.5%)	74 (15.8%)	0.684
Hypertension (Exist), n (%)	232 (49.5%)	81 (17.3%)	0.031
Diabetes Mellitus (Exist), n (%)	159 (33.9%)	62 (13.2%)	0.613
Coronary Heart Disease (Exist), n (%)	55 (11.7%)	22 (4.7%)	0.893
SII parameter	1691.35 ± 1529.29	743.58 ± 530.84	<0.001
Alive or Exitus (Exitus), n (%)	26 (5.5%)	4 (0.9%)	0.06
Hemoglobin (gr/dl)	14.29 ± 1.92	14.29 ± 1.85	0.984
Platelet (10^9/L)	259.22 ± 72.13	233.18 ± 63.67	<0.001
WBC count (109/L)	13.47 ± 8.47	10.46 ± 3.48	<0.001
HDL (mg/dl)	41.07 ± 12.85	40.65 ± 9.30	0.735
LDL (mg/dl)	119.76 ± 37.92	124.20 ± 38.69	0.266
Triglyceride (mg/dl)	145.90 ± 115.74	157.88 ± 89.96	0.292
Cholesterol (mg/dl)	189.24 ± 47.99	195.25 ± 46.72	0.226

Table 2. Comparison of STEMI and non-STEMI patients according to SII values.

Variable	STEMI	Non-STEMI	P
For all patients (n=469)	n=332	n=137	
SII parameter	1691.35 ± 1529.29	743.58 ± 530.84	<0.001
SII according to LAD culprit lesion (n=207)	1779.97 ± 1642.65	778.02 ± 674.28	<0.001
SII according to CX culprit lesion (n=104)	1752.21 ± 1303.71	746.77 ± 429.03	<0.001
SII according to RCA culprit lesion (n=158)	1549.20 ± 1469.76	692.75 ± 436.89	<0.001
For acute total occluded patients (n=400)	n=332	n=68	
SII parameter	1691.35 ± 1529.28	791.83 ± 465.07	<0.001
SII according to LAD culprit lesion (n=177)	1779.97 ± 1642.65	700.76 ± 579.70	<0.001
SII according to CX culprit lesion (n=86)	1752.21 ± 1303.71	849.41 ± 436.16	<0.001
SII according to RCA culprit lesion (n=137)	1549.20 ± 1469.76	790.49 ± 359.34	<0.001

Table 3. Univariate analysis of myocardial infarction subtypes according to clinical parameters

Depended variable (n=435)			
Source	Type III Sum of Squares	F	Sig.
SII parameter	8.577	45.257	0.000
Age	0.001	0.003	0.953
Gender	6.162	0.000	0.995
Smoking Status	0.051	0.272	0.603
Hypertension	1.394	7.358	0.007
Diabetes Mellitus	0.003	0.017	0.895
Coronary Artery Disease	0.209	1.100	0.295
Number of Diseased Vessel	0.600	3.164	0.076
Hemoglobin	0.179	0.943	0.332
HDL	0.063	0.331	0.566
LDL	0.018	0.093	0.761
Triglyceride	0.263	1.390	0.239
HDL/LDL ratio	0.263	1.389	0.239
Trig/HDL ratio	0.147	0.777	0.379

Table 4. Binary logistic regression of myocardial infarction subtypes according to clinical parameters

	B	S.E.	Wald	df	Sig.	Exp. (B)
Age	0.001	0.012	0.005	1	0.944	1.001
Gender	-0.006	0.330	0.000	1	0.984	0.994
Smoking status	0.210	0.265	0.627	1	0.428	1.234
Hypertension	0.739	0.266	7.699	1	0.006	2.094
Diabetes mellitus	0.209	0.253	0.680	1	0.410	1.232
Coronary artery disease	-0.212	0.327	0.418	1	0.518	0.809
Hemoglobin	-0.091	0.083	1.202	1	0.273	0.913
HDL	-0.014	0.019	0.500	1	0.479	0.986
LDL	-0.007	0.014	0.268	1	0.605	0.993
Cholesterol	0.012	0.014	0.669	1	0.413	1.012
Triglyceride	-0.001	0.003	0.238	1	0.626	0.999
SII parameter	-0.002	0.000	51.500	1	0.000	0.998
Constant	1.676	1.689	0.985	1	0.321	5.344

Cardiac imaging / Echocardiography

OP-007

Assessment of bi-atrial mechanical functions in patients with isolated atrial septal aneurysm

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Background and Aim: Atrial septal aneurysm (ASA) is a congenital abnormality associated with an increased risk of systemic embolism. Atrial mechanical dysfunction may represent an alternative mechanism for embolic events in these patients. The aim of the study is to evaluate left (LA) and right atrial (RA) functions with two dimensional speckle tracking echocardiography (2D STE) in patients with isolated ASA.

Methods: Fifty-four patients with ASA (mean age 50.3 ± 12.48, male 37%) and 48 healthy individuals with similar age and gender (mean age 48.3 ± 10.84, male 39.6%) were enrolled in the study. Patients with ischemic heart disease, heart failure, moderate to severe heart valve disease, congenital heart disease other than ASA, history of cerebrovascular event (CVE), rhythm other than sinus rhythm and conduction disorders on electrocardiography (ECG), poor echogenicity and patients presented with complaints of palpitation were excluded. For the assessment of atrial mechanical functions, left and right atrial peak longitudinal strain (PLS), peak contraction strain (PCS) and conduit strain (CS) measurements reflecting atrial reservoir, contraction and conduit functions, respectively, were performed with 2D STE in addition to conventional evaluation with transthoracic echocardiography.

Results: Cardiovascular risk factors, baseline clinical characteristics and conventional echocardiographic measurements were similar in both groups. Left atrial PLS and PCS were found to be significantly lower in ASA group than the controls (37.52 ± 2.89 vs. 40.16 ± 2.68, p<0.001; 17.29 ± 2.5 vs. 19.18 ± 2.23 p<0.001, respectively). Similarly RA PLS and RA PCS were observed significantly lower in patients with ASA (36.97 ± 2.19 vs. 39.77 ± 2.36, p<0.001; 16.78 ± 2.10 vs. 18.54 ± 2.43, p<0.001, respectively).

Conclusions: The findings we obtained in our study showed that bi-atrial functions decreased in isolated ASA patients. This may be suggested as a different possible cause of increased arterial embolism other than atrial arrhythmias and PFO in this patient group. Supporting these results with larger studies may change the treatment and follow-up approaches in patients with isolated ASA.

Table 1. Demographic and clinical features of the study group

Parameter	ASA patients (n=54)	Control group (n=48)	p value
Age	50.3±12.48	48.3±10.84	0.402
Male, % (n)	37 (20)	39.6 (19)	0.792
SBP (mmHg)	115.83± 13.34	117.5±14.98	0.554
DBP (mmHg)	73.14±7.09	74.06±8.79	0.563
Heart rate (beat/m)	74.66±8.94	71.97±79.32	0.141
BSA (m^2)	1.80±0.22	1.82±0.20	0.585
Hypertension, % (n)	16.7 (9)	29.4 (14)	0.132
Hyperlipidemia, % (n)	3.7 (2)	10.4 (5)	0.181
Diabetes mellitus, % (n)	3.7 (2)	8.3 (4)	0.321
Smoking, % (n)	7.4 (4)	8.3 (4)	0.862
ACEI /ARB, % (n)	7.4 (4)	14.6 (7)	0.244
Beta blockers, % (n)	5.6 (3)	8.3 (4)	0.580
CCB, % (n)	3.7 (2)	8.3 (4)	0.321
Statin % (n)	1.9 (1)	8.3 (4)	0.130

SBP, systolic blood pressure; DBP, diastolic blood pressure; BSA, body surface area; ACEI, angiotensin converting enzyme

Table 2. Comparison of echocardiographic measurements of the groups

Parameter	ASA patients (n=54)	Control group (n=48)	p value
IVS (cm)	1.03± 0.66	0.91±0.11	0.250
PW (cm)	0.95±0.36	0.9±0.11	0.317
LVEDD (cm)	4.52±0.33	4.56±0.37	0.663
LVESD (cm)	2.91±0.27	2.92±0.25	0.790
LV EF (%)	60.88±1.38	60.81±1.49	0.789
LAD (cm)	3.55±0.21	3.60±0.18	0.229
RAD (cm)	3.39±0.23	3.45±0.19	0.168
RVD (cm)	3.24±0.23	3.27±0.22	0.541
E wave velocity (cm/s)	**73.53 ±17.65**	**79.56 ±14.62**	**0.036**
A wave velocity (cm/s)	73.13±20.14	71.06±15.13	0.526
E/A ratio	1.14±0.31	1.12±0.23	0.752
DT (msn)	182.41±36.78	182.29±24.04	0.985
IVRT (msn)	93.72±15.07	92.14±10.18	0.435
E' wave velocity (cm/s)	**12.92±3.06**	14.31±3.21	**0.033**
E/E' ratio	6.11±2.22	5.70±1.06	0.257
LAV	38.63±11.16	39.75±9.27	0.586
LAVI	21.24±4.87	21.72±4.46	0.603
LA PLS	**37.52±2.89**	**40.16±2.68**	**<0.001**
LA PCS	**17.29±2.51**	**19.18±2.23**	**<0.001**
LA CS	20.29±2.99	20.98±3.95	0.106
RA PLS	**36.97±2.19**	**39.77±2.36**	**<0.001**
RA PCS	**16.78±2.10**	**18.54±2.43**	**<0.001**
RA CS	20.18±2.59	21.23±3.50	0.080

IVS, interventricular septal thickness; PW, posterior wall thickness ; LVEDD, Left ventricular end diastolic diameter; LVESI Left ventricular end systolic diameter; LV EF, Left ventricular ejection fraction; LAD, left atrial end systolic diameter; RAI right atrial end systolic diameter; RVD, right ventricular end diastolic diameter; DT, deceleration time; IVRT, isovolumetr relaxation time; LAV, left atrial volume, LAVI, left atrial volume index; PLS, peak longitudinal strain; PCS, peak contractic strain; CS, conduit strain.

Cardiac imaging / Echocardiography

OP-008

The right ventricle strain value is highly associated with hospitalization in patients with heart failure with preserved ejection fraction

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Background and Aim: Heart failure with preserved ejection fraction (HFpEF) is the leading cause of the hospital admission due to heart failure. Therefore, several studies have investigated the development of heart failure and the prevention of heart failure in patients with diastolic dysfunction. Although studies primarily concentrates upon the left ventricle and left atrial functions, there have been no studies to evaluate right ventricle function on the effect of heart failure development in these patients group. In this study, we sought to investigate the echocardiographic parameters measuring right ventricle functions including right ventricle free wall longitudinal strain could predict hospital admission with heart failure in patients with HFpEF.

Methods: A total of 69 patients with HFpEF that undergoing transthoracic echocardiography were reviewed. Fifty-nine of those who had feasible views for RV strain measurements were included to our study. All patients were asymptomatic for heart failure at the time of echocardiography. All study populations were observed prospectively. Hospital admission for heart failure was determined as the end point. All patients were divided into two groups according to having end point. 31 patients admitted to hospital with heart failure symptom in the first year of the study.

Results: The mean age of the study population was 63.1 ± 9.5 years. No statistical difference was determined between the groups' demographic and laboratory characteristics. Echocardiographic parameters were compared between groups. The left atrial reservoir strain (LASr) and right ventricle free wall longitudinal strain (RV FWLS) were impaired in the end point developing group. In addition there was no differences in parameters measuring right ventricle function including TAPSE, Tricuspid lateral annulus s velocity and TR jet velocity. In multivariate analysis, RV FWLS was an independent predictor of hospitalization for heart failure in patients with HFpEF. A cut-off value of RV FWLS was found as -15.5% using ROC analysis.

Conclusions: This study demonstrated that RV FWLS was the independent predictor of hospitalization for heart failure in patients with diastolic dysfunction. Larger, prospective studies are necessary to determine the impact of RV FWLS on predicting heart failure in patients with diastolic dysfunction.

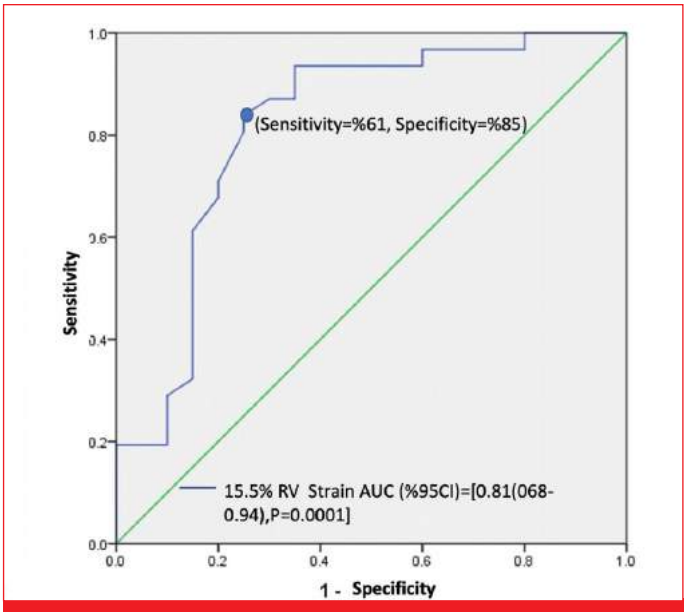

Figure 1. ROC curve of RV FWLS. -15.5% cut point of RV FWLS using ROC analysis

Table 1. Echocardiographic parameters were divided by end point. Echocardiographic parameters were compared between two groups

Parameters	End point developer	End point non-developer	P
EF%	57.9 ± 4.1	57.1 ± 2.5	0.45
TR velocity	2.67 ± 0.68	2.8 ± 0.63	0.64
TAPSE mm	17.6 ± 0.34	19.4 ± 0.23	0.23
Lateral annulus s velocity cm/sn	11.2 ± 0.21	10.9 ± 0.35	0.53
LAVi	34.2 ± 5.3	32.9 ± 7.06	0.13
E/e'	17.4 ± 0.78	18.1 ± 0.12	0.18
LASr (Left atrial strain)	25.1 ± 8.2	32.0 ± 6.3	0.002
RV free wall strain	-15.3 ± 2.5	-20.0 ± 4.6	<0.0001

Table 2. Univariate and multivariate analysis of parameters. Parameters with p<0.2 were added to the univariate analysis In univariate analysis parameters with <0.2 were added multivariate analysis

Parameters	Univariate analysis Odds Ratio (95% CI) P value	Multivariate analysis Odds Ratio (95% CI) P value
LASr	0.88 (0.80-0.96) 0.006	1.04 (0.96-1.12) 0.32
Creatinine	1.56 (0.81-3.02) 0.20	
ALT	1.01 (0.99-1.04) 0.22	
E/e'	1.09 (0.91-1.31) 0.32	
LAVi	0.93 (0.84-1.02) 0.14	0.9 (0.79-1.04) 0.20
RV free wall strain	1.4 (1.1-1.8) 0.001	1.5 (1.18-1.91) 0.001

Cardiac imaging / Echocardiography

OP-009

The relationship between the global left ventricle functions and long-term survival in non-ischemic dilated cardiomyopathy patients: A retrospective cohort study

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Background and Aim: Idiopathic dilated cardiomyopathy (DCM), is one of the leading causes of heart failure (HF) with low ejection fraction (EF). The Tei index, also known as a myocardial performance index (MPI), is a reliable marker reflecting both systolic and diastolic functions of the left ventricle (LV) and has prognostic value in patients with DCM. In the present study, we aimed to investigate the relationship between the MPI and long-term survival in non-ischemic DCM patients.

Methods: The present study was included 98 patients with non-ischemic DCM. The patients were divided into two groups according to survival. During the follow-up period, 66 patients (41 females and 25 males) died and 32 patients (23 females and 9 males) were alive. The mean survival time of all patients was 59 (3-144) months. The patients underwent coronary angiography to exclude ischemic etiology and echocardiography to evaluate global LV functions (systolic and diastolic).

Results: At the last follow-up, the number of patients who died was 66 (67%). In deceased patients, while LVEF (p<0.01) was significantly lower, LV end-systole volume (p<0.01), LV end-diastolic volume (p<0.01) were significantly higher. Moreover, tissue Doppler imaging (TDI)-MPI (0.64 ± 0.08 vs. 0.71 ± 0.12, respectively; p=0.01) was prominantly higher in patients who died. LV end-systolic volume and LVEF were independent prognostic factors and predicted worse long-term survival in DCM patients. More importantly, the patients with LVEF ≥ 32.7% and MPI ≤0.76 had significantly longer survival (Figure 1).

Conclusions: The present study showed that TDI-MPI was significantly associated with mortality and the patients with both low LVEF (≤32.7%) and high MPI (≥0.76) values had a shorter life expectancy. As a result, we suggest that the Tei index may be a useful echocardiographic marker to predict long-term survival along with LVEF.

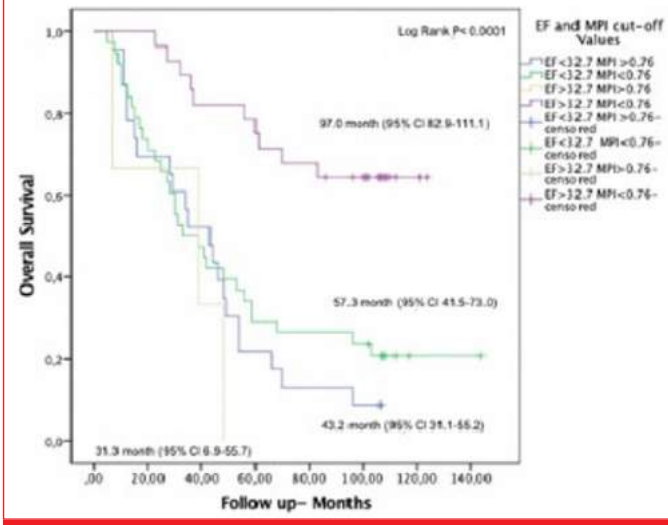

Figure 1. Kaplan-Meier median overall survival curves reflect the differences in survival rates relative to the cut-off LV EF and MPI values in DCMP patients.

Cardiac imaging / Echocardiography

OP-010

The pulmonary annular motion velocity with using tissue Doppler imaging could predict the severity of the cirrhosis

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Background and Aim: Chronic liver disease has high incidence and prevalence worldwide. An association has been found between liver disease and the cardiovascular system. Liver function deterioration is associated with structural and functional cardiac abnormalities irrespective of its etiology. It has been defined as cirrhotic cardiomyopathy (CCM). There are previous reports that biventricular functions are affected by cirrhosis and that some echocardiographic predictors may be associated with the severity of cirrhosis. Pulmonary annular motion velocity is an important systolic function indicator of RVOT. So far, we have not come across a study investigating RVOT functions in cirrhotic patients in the literature. In this study, we investigated the relationship between PAMVUT values, which indicate RVOT systole function, and the severity of the disease in patients with cirrhosis.

Methods: This study enrolled 74 consecutive hepatic cirrhotic patients referred from the departments of gastroenterology and internal medicine to the echo lab of the Cardiology Department of Bolu Abant İzzet Baysal University between 01/01/2018 and 01/01/2022. The demographic, laboratouary panel, electrocardiographic, echocardiographic and ultrasound data were collected. Then the patients were divided into three separate group based on their cirrhosis severity which defined with Child-Pough classsification. Data were collected, revised, tabulated, and statistically analyzed. Continuous data are expressed as mean standard deviation, and categorical variables as the frequency and percentage for each group. The χ^2 test was used for categorical variables, to compare between different groups. One-way ANOVA was used for normally distributed quantitative variables, to compare between more than two groups, with Tukey's multiple comparison post hoc tests to identify individual group differences. The Kruskal-Wallis test was used for non-normally distributed quantitative variables, to compare between more than two studied groups. The result was considered significant when p value was less than 0.05 and highly significant when less than 0.01.

Results: The demographic characteristics and laboratory data for the patients based on their Child Pugh Classes. In terms of clinical qualities, neither group was significantly different. We found no significant changes between groups in the several conventional echocardiographic measures that measured the diameter and function of the right and left ventricules. However, a statistical association was found between Child Pugh Classes and FAC, PAMVUT, St and combined S. PAMVUT levels were significantly high in Class A compared Class C. Combined S levels were significantly high in Class A (mild) compared Class B (moderate) and Class C (severe).

Conclusions: In this study, where we showed that the power of CSV values obtained by considering RVOT functions to predict the severity of cirrhosis is more important than the rate of St.

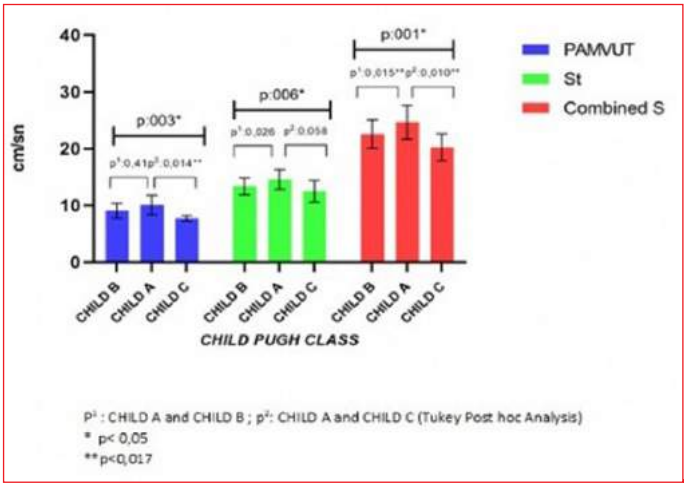

Figure 1.

Table 1. Clinical characteristics of all patients with cirrhosis

	n =74, %
Cause of liver cirrhosis, %	
Hepatitis B	28 (37,8)
Hepatitis C	16 (21,6)
Alchol	5 (6,7)
Wilson's	4 (5,4)
Autoimmune liver disease	2 (2,7)
Primary biliary cirrhosis	3 (4,1)
Cryptogenic	7 (9,4)
Hepatic steatosis	2 (2,7)
Hemochromatosis	2 (2,7)
Budd Chiari Syndrome	1 (1,3)
Portal vein thrombosis	1 (1,3)
Others	3 (4,1)
Complications of cirrhosis, %	
Ascites	28 (37,8)
Splenomegaly	56 (75,6)
Varices	51 (68,9)
Variceal bleeding	17 (22,9)
Liver cancer	4 (5,4)
MELD-Na Score	**11,18 ± 4,17**
Child-Pugh Class, %	
A	49 (66,2)
B	21 (28,3)
C	4 (5,4)

Table 2. Clinical charcteristics and laborotory findings of patients

	Child-Pugh Class A (n=49)	Child-Pugh Class B(n=21)	Child-Pugh Class C(n=4)	P value
Male[a],	29 (%59,1)	9 (%42,8)	2 (%50,0)	,448
Age[b], years	54,16 ± 14,26	58,47 ± 13,00	45,25 ± 13,67	,185
Hypertension[a]	12 (%24,4)	9 (%42,8)	1 (%25,0)	,298
Diabetes[a]	19 (%38,7)	11 (%52,3)	1 (%25,0)	,446
CKD[a]	2 (%4,1)	2 (%9,5)	0 (%0)	,579
Bilirubin[c], mg/dl	,93 (,31 – 2,45)	1,62 (,48 – 17,38)	6,05 (3,06 – 9,31)	,001
Prothrombin time[b], INR	1,24 ± ,26	1,50 ± ,66	1,64 ± ,40	,023
Platelet count cells[b], uL	130,12± 79,90	113,25±43,45	119,32± 40,34	,647
Albumin[b], g/L	4,16 ± ,51	3,17 ± ,81	2,75 ± ,77	,001
ALT[c], U/L	24,5 (6 – 148)	29,5 (9 – 71)	33,5 (15 – 72)	,252
AST[c], U/L	27 (12 – 187)	46 (17 – 107)	84 (23 – 241)	,002
ALP[c]	85 (32 – 268)	113 (51 – 290)	154 (109 -286)	,006
GGT[c]	28,5 (9 – 294)	70 (12 – 185)	62,5 (57 – 308)	,196
AFP[c]	2,74 (,73 – 134,5)	3,25 (1,27 – 322)	4,46 (2,35 – 1534)	,437
Blood urea nitrogen[c], mg/dL	28 (13 – 92)	39 (16 – 152)	31 (24 – 58)	,072
Creatinine[c], mg/dL	,78 (,45 – 2,63)	,85 (,58 – 1,73)	,805 (,56 – 1,02)	,517
Sodium[b], mmol/L	138,85 ± 2,87	136,66 ± 4,54	138,08 ± 1,82	,026
Potassium[b], mmol/L	4,29 ± ,33	4,43 ± ,33	3,85 ± ,44	,008

CKD : Chronic Kidney Disease, INR: international normalized ratio, ALT: Alanine aminotransferase, AST: aspartate aminotransferase, ALP: alkaline phosphatase, GGT: gamma glutamyl transferase, AFP: alpha-fetoprotein,

[a]χ2 – chi-square

[b] one way ANOVA

[c]Kruskal-Wallis

Table 3. Echocardiography findings of patients usingANOVA

	Child-Pugh Class A (n=49)	Child-Pugh Class B(n=21)	Child-Pugh Class C(n=4)	P value
LVEF, %	61,67 ± 5,09	60,9048 ± 4,76345	61,2500 ± 2,50000	,834
LA,cm	3,50 ± ,28	3,5952 ± ,39809	3,7250 ± ,22174	,282
IVS, cm	1,03 ± ,14	1,0952 ± ,13220	1,0500 ± ,12910	,214
LVEDD, cm	4,52 ± ,33	4,4333 ± ,28519	4,8250 ± ,43493	,091
LVESD, cm	2,71 ± ,48	2,6143 ± ,53971	3,0250 ± ,42720	,318
PW, cm	1,16 ± 0,10	1,0905 ± ,10911	1,0500 ± ,10000	,912
E, mm/sn	83,04 ± 13,47	84,80 ± 22,79	86,50 ± 10,24	,867
A, mm/sn	79,93 ± 19,44	80,09 ± 16,89	72,50 ± 12,60	,735
E', mm/sn	11,80 ± 2,27	11,09 ± 1,49	11,40 ± 2,57	,437
A', mm/sn	13,14 ± 2,38	13,13 ± 1,66	11,65 ± 1,44	,413
TAPSE, mm	24,58 ± 2,35	23,40 ± 2,52	23,25 ± 1,89	,129
FAC, %	39,43 ± 5,55	34,71 ± 4,99	31,75 ± 2,50	,001
sPAP, mm Hg	23,73 ± 4,03	25,00 ± 5,68	27,00 ± 6,27	,288
PAMVUT,cm/sn	10,10 ± 1,69	9,09 ± 1,30	7,75 ± ,50	,003
St, cm/sn	14,58 ± 1,77	13,39 ± 1,50	12,50 ± 1,91	,006
Combined S, cm/sn	24,67 ± 2,95	22,57 ± 2,50	20,25 ± 2,36	,001

LVEF: Left Ventriculer Ejeksion Fraction, LA: left Atrium, IVS: Interventriculer Septum, LVEDD:Left Ventriculer End-Diastolic Diameter, LVESD: Left Ventriculer End-Systolic Diameter, PW: Posterior Wall, E: Early Ventricular Filling Velocity, A: Late Ventricular Filling Velocity, E': Ventricular Tissue Doppler Early Diastolic Velocity, A': Ventricular Tissue Doppler Late Diastolic Velocity, TAPSE: Tricuspid Annular Plane Systolic Excursion, FAC: Fractional Area Change, sPAP: Systolic Pulmonary Artery Presssure, PAMVUT: Pulmonary Annulus Motion Velocity, St: Tricuspid Annulus Systolic Velocity

Table 4. Correlation of Combined S with right ventricular echocardiographic measurements

Combined S		
Variables	r	p
TAPSE, cm	0,350	0,002
FAC, %	0,458	<0,001
St, cm/sn	0,884	<0,001
PAMVUT, cm/sn	0,855	<0,001

TAPSE: Tricuspid Annular Plane Systolic Excursion, FAC: Fractional Area Change, St: Tricuspid Annulus Systolic Velocity, PAMVUT: Pulmonary Annulus Motion Velocity

Pulmonary hypertension / Pulmonary vascular diseases

OP-011

Levels of oxidant and antioxidant status in a model of pulmonary hypertension secondary to lung fibrosis

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Background and Aim: Pulmonary fibrosis (PF) is a progressive fatal disease. Unfortunately, there is no effective treatment yet. Bleomycin is commonly administered intratracheally to rats as an experimental model of pulmonary fibrosis, which is characterized by the development of secondary PH. In this study, we aimed to investigate the effect of the lung fibrosis model we created with bleomycin experimentally on the oxidant and antioxidant system.

Methods: The bleomycin lung model (BLM) rat model of lung fibrosis was used. Twenty male Wistar albino rats were used. After sacrifice, lung histopathologic evaluations of PF were performed using the Ashcroft scoring method. Biochemical Analysis total oxidant status (TOS), total antioxidant status (TAS), thiol-disulfide, lipid hydroperoxide (LOOH) levels, from the venous blood samples taken were determined as described by Erel (2004, 2005, 2014). Data are expressed as mean ± standard deviation of the mean. Statistical analysis was made by one–way ANOVA test. Correlation was calculated using Pearson's test. Scores and values p<0.05 were accepted as statistically significant.

Results: The fibrosis scores were significantly enhanced by BLM stimulation. The TAS and Thiol levels were decreased by BLM stimulation. The TOS and LOOH levels were significantly enhanced by BLM stimulation. The correlation between TOS, oxidative stress index (OSi), LOOH levels and fibrosis scores were analyzed and statistically significant correlation was found between TOS, LOOH, OSI levels and fibrosis scores (p=0.001, r=0.783, p=0.001, r=0.624, p=001, r=0.655) (Tables 1-3).

Conclusions: PF is a chronic, progressive and fatal disease resulting from the complex interaction of genetic and environmental factors that affect on many basic cellular process. Oxidative stress threatens genetic integrity and contributes to the development of PF. Avoiding damage products due to oxidative stress and drawing a protective treatment protocol may be a crucial strategy for maintaining healthy cardio-pulmonary function and protecting the organism from cardio-pulmonary diseases such as PF. Determination of antioxidant enzyme regulations in PF and progressive pulmonary vascular diseases may be helpful in determining new diagnostic and therapeutic approaches in this chronic, fatal and poor prognosis disease of the lung.

Table 1. Levels of the oxidative and anti-oxidative status in groups

Groups (n)	Control (10)	BLM (10)	P
TOS±SD	215.77 ± 31.63	330.97 ± 28.31	<0.001
OSI±SD	4.57 ± 1.3	7.59 ± 1.4	<0.001
LOOH±SD	109.59 ± 12.59	142.49 ± 13.65	0.004
TAS±SD	4.86 ± 0.769	4.45 ± 0.65	0.373
TIHOL±SD	2.17 ± 0.204	1.95 ± 0.241	0.004

Table 2. Comparing grades of pulmonary fibrosis in groups

	Control (10)	BLM (10)	P
Grades of pulmonary fibrosis ± SD	0.700 ± 0.483[a]	4.800 ± 1.032[b]	<0.001

Table 3. Correlation analysis fibrsosis scores and oxidative-anti-oxidative parameters

	Fibrosis Scores	
	r	P
TOS	0.783	<0.001
OSI	0.655	<0.001
LOOH	0.624	<0.001

Pulmonary hypertension / Pulmonary vascular diseases

OP-012

Pulmonary hypertension classification based on machine learning using standart chest X-Ray: ATA Artificial Intelligence Study-1

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Background and Aim: An accurate diagnosis of pulmonary hypertension (PH) is crucial to ensure that patients receive timely treatment. One of the used imaging models to detect pulmonary hypertension is the X-ray. Therefore, a new automated PH type classification model has been presented to depict the separation ability of deep learning for PH types

Methods: We retrospectively enrolled 6642 images of patients with PH and the control group. A new X-ray image dataset was collected from a multicenter in this work. A transfer learning-based image classification model has been presented in classifying PH types. Our proposed model was applied to the collected dataset, and this dataset contains six categories (five PH and a non-PH).

Results: The presented deep feature engineering (computer vision) model attained 86.14% accuracy on this dataset. According to the extracted ROC curve, the average area under the curve rate has been calculated at 0.945

Conclusions: A new deep learning model has been presented to classify PH types in this research. The given model attained 86.14% classification accuracy and 94.50% UAR value. Furthermore, our proposed model can separate PH and non-PH X-ray images easily.

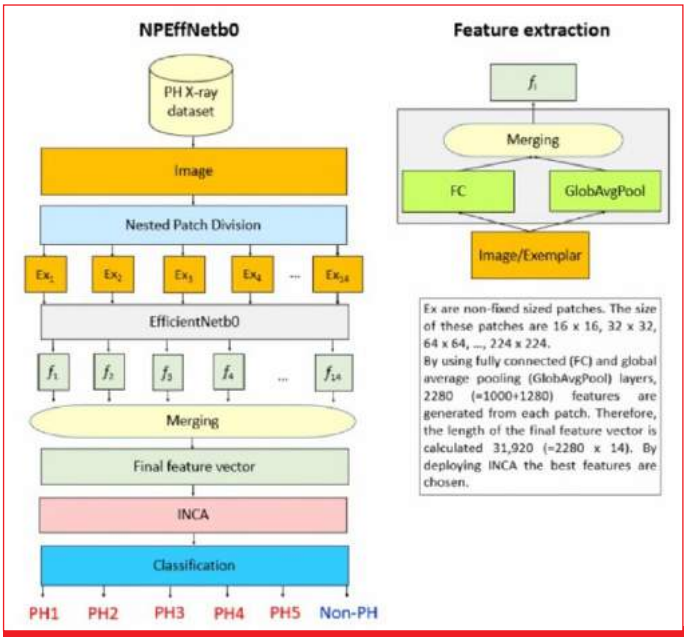

Figure 1. Graph of the presented NPEffNetb0 classification model

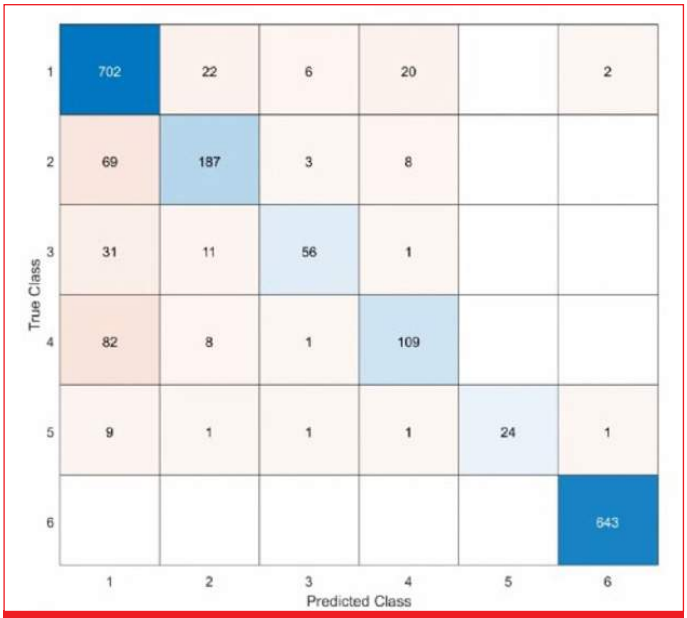

Figure 2. Confusion matrix calculated of the proposed EffNetb0 on the collected X-ray dataset for PH detection

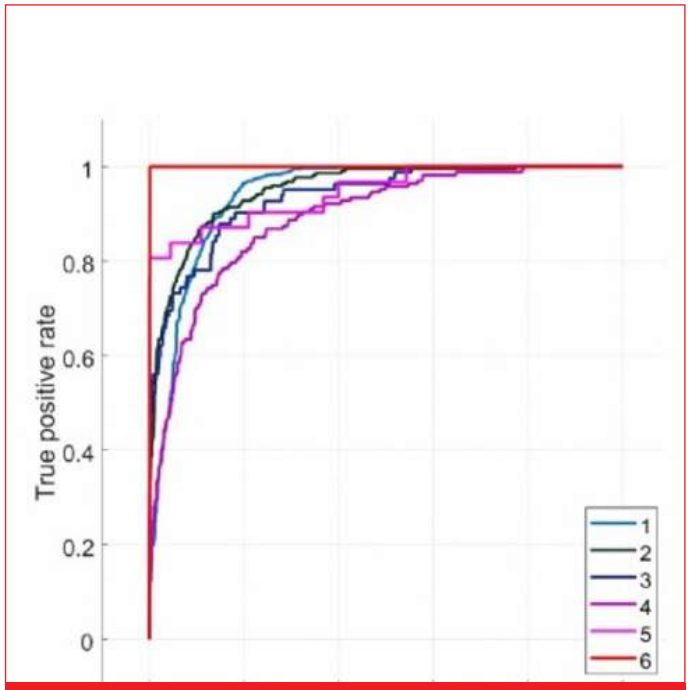

Figure 4. ROC curve of the presented model

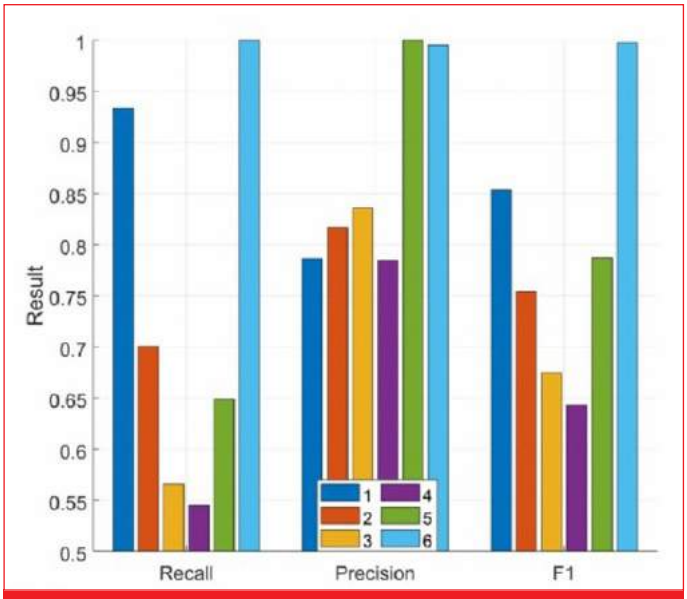

Figure 3. Class-wise performance metrics of the proposed EffNetb0 feature extraction-based model

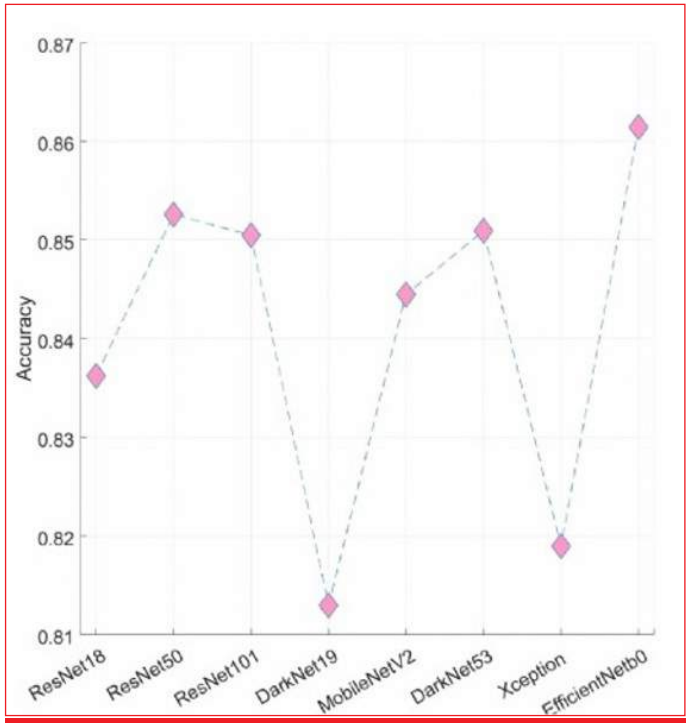

Figure 5. Classification performances (accuracy) of eight pre-trained CNNs

Table 1. Attributes of the X-Ray image dataset collected

No	Category	Number of Image
1	Pulmonary arterial hypertension	2563
2	Pulmonary hypertension due to left heart disease	891
3	Pulmonary hypertension due to lung disease and/or hypoxia	330
4	Chronic thromboembolic pulmonary hypertension	670
5	Pulmonary hypertension with unclear and/or multi-factorial mechanism and	65
6	Non-PH	2145

Table 2. Overall classification performances.

Performance metric	Results
AP	86.97
UAR	73.22
F1	78.50
Accuracy	86.14

Pulmonary hypertension / Pulmonary vascular diseases

OP-013

Investigation of serum endocan level in patients with pulmonary embolism

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Background and Aim: Pulmonary embolism (PE) occurs as a result of occlusion of the pulmonary artery bed with a clot and it is a serious life-threatening pathology. Pulmonary endothelial dysfunction seen in the vascular bed is an important factor in the pathogenesis of PE.

Endothelial cell specific molecule (endocan) is a soluble dermatan sulfate proteoglycan secreted from the lung and kidney vascular endothelium. Endocan has an important role in endothelium-dependent pathological processes. The aim of this study is to evaluate serum endocan in patients with acute PTE and to asses the relationship between serum endocan levels and disease severity.

Methods: 25 patients with acute PTE and 20 healthy controls were included in the study. Controls and cases of pulmonary embolism were similar in terms of age and gender distribution. All PTE patients were diagnosed Computed Tomograph (CT) with contrast. According to Wells clinical predictive scoring, patients were divided into 3 groups as high-risk, intermediate-risk, and low-risk. Endocan levels between groups were compared. Of 25 patients, 4 were in the low probability risk group, 8 were in the medium risk group, and 13 were in the high probability risk group.

Results: Endocan level (561.43 ± 358.31 pg\ml) in the pulmonary embolism group was statistically significantly higher than the control group (148.20 ± 133.53 pg\ml) (p <0.001). Among the pulmonary embolism risk groups, the endocan levels of the group with high probability were statistically higher than the other two groups (p <0.001). Serum endocan is 1050.12 ± 493.49 pg/ml in the high-risk group, 623.48 ± 545 pg/ml in the medium-risk group, and 218.41 ± 178.43 pg/ml in the low-risk group.

Conclusions: Serum endocan levels are higher in PTE patients and the elevation is associated with the severity of the disease. In addition, endocan can be an indicator that can be used in the diagnosis of PTE and in the estimation of disease severity.

Table 1. Comparison of demographic characteristics and biochemical values of groups

	PTE	Healthy group	P value
Age	49.45 ± 14.42	47.65 ± 12.28	0.59
Gender	13\12	11\9	0.64
BMI	28.7	27.9	0.72
Troponin	0.248 ± 0.183	0.02 ± 0.0034	0.001
CRP	1.2 ± 0.62	0.48±0.35	0.01
en ENDOCAN	(561.43 ± 358.31 pg\ml)	148.20 ± 133.53 pg\ml	0.01

Table 2. Comparison of groups in terms of endocan and troponin levels

	High Risk Group	Intermediate Risk Group	Low Risk Group
Endocan	1050.12 ± 493.49 pg\ml	623.48 ± 545 pg\ml	218.41 ± 178.43 pg\ml
Troponin	423.1 ± 323.2 pg\ml	335± 128 ± 128.6 pg\ml	216 ± 166.8 pg\ml

Coronary artery disease / Acute coronary syndrome

OP-014

Relationship between serum cystatin C level and spontaneous recanalization in ST-segment elevation myocardial infarction

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Background and Aim: Cystatin C is associated with increased coronary atherosclerotic burden and indirectly no-reflow phenomenon. We sought to investigate the association of serum cystatin c levels at admission with spontaneous recanalization (SRC) of the infarct-related artery (IRA) on coronary angiography (CAG) of ST-segment elevation myocardial infarction (STEMI) patients undergoing primary percutaneous coronary intervention (pPCI).

Methods: 144 consecutive STEMI patients who underwent pPCI between September 2018 and November 2018 were

enrolled in the study. Those with a glomerular filtration rate <60 ml/min were excluded from the study. Laboratory parameters and demographic characteristics were recorded. Serum cystatin C levels were also obtained from each patient at admission. Participants were classified according to the presence or absence of SRC on CAG, SRC (+) and SRC (+).

Results: Forty-eight (33.3%) had SRC. There was no difference in terms of age and gender between SRC (+) and SRC (-). The frequency of diabetes mellitus (33.3% vs. 15.5%, p=0.015) and serum cystatin C levels (0.99 ± 0.27 vs. 0.89 ± 0.27, p=0.022) were higher in patients with SRC (Figure 1) than in those without; whereas high-sensitivity cardiac troponin I level at admission [5.7 (0.9-26.4) vs. 26.4 (5.1-146.2), p<0.001] was higher in the latter. Lipid profile, C-reactive protein, and complete blood counts were comparable between the groups (Table 1). On multivariate logistic regression analysis; high-sensitivity cardiac troponin I [odds ratio (OR)=0.993 (0.986-1.000), 95% confidence interval (CI), p=0.036] and serum cystatin C [OR=0.184 (0.038-0.893), 95% CI, p=0.036)] were independently associated with SRC of the IRA for STEMI patients undergoing pPCI (Table 2).

Conclusions: Cystatin C is still under investigation for its impact on predicting new onset/worsening coronary artery disease or cardiovascular mortality, and its contribution to risk assessment, rather than its effect in demonstrating kidney function, which was its first use in clinical practice. Compatible with the existing literature, we demonstrated an independent and inverse relationship between spontaneous recanalization of the IRA, which is associated with mortality among STEMI patients, and serum cystatin C level at admission.

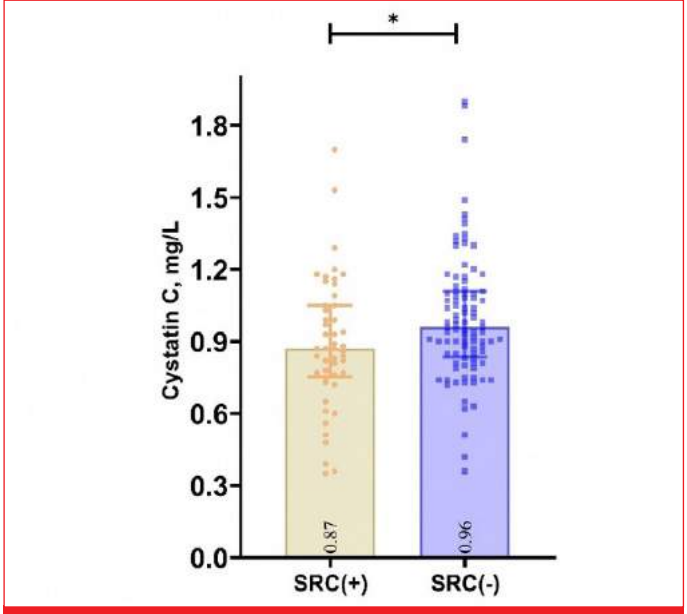

Figure 1. Serum cystatin C levels of the study population separated by the presence or absence of SRC of the IRA. Note that shown with median values. *P=0.022 (SRC, spontaneous recanalization; IRA, infarct-related artery)

Table 1. Demographic characteristics of the study population

	All (n=144)	SRC (+) (n=48)	SRC (-) (n=96)	P
Age, years	60.7 ± 10.4	61.4 ± 9.8	60.2 ± 10.7	0.51
Gender (female), n (%)	27 (18.8)	11 (22.9)	16 (16.7)	0.37
BMI, kg/m^2	26.8 ± 3.9	26.7 ± 4.4	26.8 ± 3.7	0.84
Diabetes mellitus, n (%)	31 (21.5)	16 (33.3)	15 (15.6)	0.015
Hypertension, n (%)	43 (29.9)	18 (37.5)	25 (26.0)	0.16
Current smoker, n (%)	100 (69.4)	35 (72.9)	65 (67.7)	0.52
Hyperlipidemia, n (%)	27 (18.8)	10 (20.8)	17 (17.7)	0.65
History of family, n (%)	18 (12.5)	4 (8.3)	14 (14.6)	0.22
Stroke/TIA, n (%)	1 (0.7)	1 (1.0)	0 (0)	0.29
Ejection fraction, %	42.8 ± 7.6	42.5 ± 8.1	42.9 ± 7.4	0.74
Hemoglobin, g/dL	13.7 ± 1.8	13.6 ± 1.9	13.7 ± 1.7	0.68
e-GFR, ml/min	106.0 (90.0-126.0)	111.5 (92.8-124.8)	105.5 (88.4-128.8)	0.70
LDL, mg/dL	133 ± 35	139 ± 34	130 ± 35	0.22
HDL, mg/dL	39 ± 8	41 ± 8	39 ± 8	0.10
Triglycerides, mg/dL	114 (71-204)	107 (70-188)	118 (70-198)	0.96
CRP, mg/L	1.8 (0.7-3.9)	2.5 (0.9-4.5)	1.6 (0.6-3.1)	0.11
High sensitive Troponin I at admission, ng/L	22.0 (2.7-84.1)	5.7 (0.9-26.4)	26.4 (5.1-146.2)	< 0.001
Neutrophil count, 10^3/µL	9.3 (7.2-12.4)	8.9 (7.2-11.2)	9.5 (7.2-12.4)	0.52
Lymphocyte count, 10^3/µL	1.9 (1.4-2.4)	1.6 (1.2-2.5)	1.9 (1.5-2.4)	0.24
Platelet count, 10^3/µL	251 ± 64	246 ± 66	254 ± 64	0.49
Serum cystatin C at admission, mg/L	0.97 ± 0.28	0.99 ± 0.27	0.89 ± 0.27	0.022

Data are presented as numbers and percentages (%), mean (standard deviation) or median (interquartile range). P-value was calculated using the Independent Samples t-test or the 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. Abbreviations: BMI, body mass index; TIA, transient ischemic attack; e-GFR, estimated glomerular filtration rate; LDL, low-density lipoprotein; HDL, high-density lipoprotein; CRP, c-reactive protein; SRC, spontaneous recanalization.

Table 2. Univariate and multivariate analysis for predicting spontaneous recanalization of STEMI patients undergoing primary percutaneous intervention

	Univariate Analysis		Multivariate Analysis	
	OR (95 % CI)	P	OR (95 % CI)	P
Age, years	1.011 (0.978-1.045)	0.51		
LDL, mg/dL	1.007 (0.996-1.018)	0.22		
HDL, mg/dL	1.038 (0.992-1.085)	0.10		
Cystatin C, mg/L	0.193 (0.046-0.818)	0.026	0.184 (0.038-0.893)	0.036
CRP, mg/L	1.094 (0.991-1.209)	0.076		
Lymphocyte count, 10^3/µL	0.915 (0.619-1.351)	0.65		
Gender (female)	1.486 (0.628-3.516)	0.37		
History of family	0.532 (0.165-1.716)	0.29		
Diabetes mellitus	2.700 (1.196-6.098)	0.017		
Hypertension	1.704 (0.812-3.575)	0.16		
Hs-Troponin I, ng/L	0.992 (0.986-0.998)	0.01	0.993 (0.986-1.000)	0.036

P-value <0.05 was considered significant. -2 log likelihood, 130,809; Model chi-square, 21,954; Nagelkerke R2, 0.232, P < 0.001. Abbreviations: LDL, low-density lipoprotein; HDL, high-density lipoprotein; CRP, C reactive protein; Hs-Troponin I, high sensitive troponin I; OR, odds ratio; CI, confidence interval

Coronary artery disease / Acute coronary syndrome

OP-015

Impact of different types of no-reflow phenomenon on clinical outcomes in patients with myocardial infarction with persistant ST-segment elevation

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Background and Aim: Achieving tissue perfusion is the primary goal of treatment in patients with ST-segment elevation myocardial infarction(STEMI). Although primary percutaneous coronary intervention (pPCI) provides recanalization of infarct related artery, tissue perfusion can not be re-establish in considerable amount of patients with STEMI which is called no-reflow phenomenon (NR). Failure in tissue perfusion diagnoses either absence/slow flow in IRA (TIMI flow < 3) or reduced staining of myocardium (myocardial blush grade <2). In this study, we aimed to investigate the clinical outcomes in patients with different types of NR.

Methods: We retrospectively included patients who were treated with pPCI. NR phenomenon was defined as angiographic NR when final distal flow was below TIMI III (group 1) and tissue level NR when myocardial blush grade was less than grade II (group 2). Patients without NR was constituted group 3. Patients' characteristics, clinical variables and cardiac outcomes were recorded.

Results: We included 604 patients; group 1 consist of 119 patients, group 2 228 patients and group 3 250 patients. Patients with NR was significantly older (p< 0.001). Hypertension, diabetes mellitus and chronic renal failure were more prevalent among patients with NR (p= 0.002, p= 0< 0.001 and p= 0.047 respectively). Ischemia duration and door-to-balloon time were also longor in NR groups(p= 0. 012 and 0.017 respectively). There was no significant difference between groups in terms of angiographic parameters such as infarct-related artery (p= 0.822), multivessel disease (p= 0.423) presence of LMCA disease (0.526) or non-IRA chronic total occlusion (0.281). 30-day cardiac mortality was highest in group 1 patients (p < 0.001) and significantly higher than group 2 (14.3% vs. 5.7%). Heart failure during follow-up was also highest among group 1 patients (61.3% vs. 53.5%, and 33.6% respectively, p < 0.001).

Conclusions: Our results demonstrated that NR is a strong indicator of adverse cardiac outcomes. Furthermore, compared to tissue level NR, angiographic NR is associated with worse outcomes. Hence, discrimination of NR phenomenon is important for risk stratification during follow-up.

Coronary artery disease / Acute coronary syndrome

OP-016

Relationship between cystatin C and syntax score in patients with acute coronary syndrome

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Background and Aim: Syntax (SYNergy between PCI with TAXUS and Cardiac Surgery) Score (SS) is an angiographic scoring system for determining the severity of coronary artery disease (CAD).Cystatin C (Cys-C) has been proposed as a useful biomarker of early impaired kidney function and predictor of mortality risk. As far as we know, there is no study in the literature examining the relationship between Syntax Score and Cystatin C. In this study, we aimed to investigate the relationship between Cys-C level and SS in patients with Non ST-Elevation Myocardial Infarction (NSTEMI) and ST-Elevation Myocardial Infarction (STEMI).

Methods: The study included 85 patients with NSTEMI and STEMI undergoing coronary angiography. Patients were divided into two groups according to the SS values: group 1 defined as patients with SS <22, while group 2 was defined as patients with SS ≥22. Two groups were compared in terms of baseline characteristics and serum Cys-C level.

Results: A total of 85 patients were included in the study. Demographic and clinical characteristics of the patients are given in Table 1. There was no significant difference between the two groups in terms of baseline demographic characteristics. However, Cystatin C was significantly higher in patients in group 2 compared with patients in group 1 (p= 0.007). In the correlation analysis, there was a positive correlation between Syntax score and Cystatin C (r = 0.385, p = 0.006) and smoking status (r = 0.218, p = 0.044). Multivariate logistic regression analysis was performed to identify independent predictors of high SS.Cystatin C was found to independently predict high SS in multivariate logistic regression analysis Table 2. The best cut-off value of Cystatin C in predicting high SS was determined by ROC analysis (Figure 1). When ROC curve analysis was performed, the optimal cut-off value of Cystatin C to predict severe coronary artery disease, Cystatin C ≥1.53 was predictive for severe coronary artery disease with 71% sensitivity and 69% specificity (area under the curve: 0.718, 95% confidence interval [CI]: 0.622-0.844, p<0.001).

Conclusions: Cystatin C level is independently strongly associated with high SS in patients with STEMI and NSTEMI. In summary, our results suggested that Cys-C is a promising clinical biomarker could be used to evaluate the severity of coronary artery lesions and this provides complementary information to the established risk determinants.

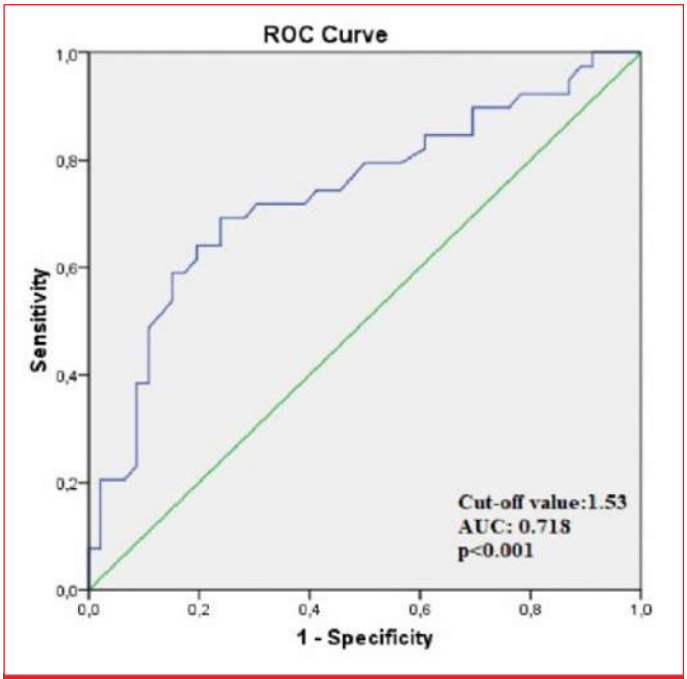

Figure 1. ROC curve analysis of cystatin C predicting high syntax score

Table 1. Demographic and characteristic features of study population

Variables	Group 1 (n = 46)	Group 2 (n=39)	P
Age	55 ± 13	60 ± 13	0.037
Gender, male (%)	39 (84)	32 (82)	0.73
Diabetes mellitus, (%)	7 (15)	8 (20)	0.52
Hypertension, (%)	15 (32)	22 (56)	0.02
Hyperlipidemia, (%)	8 (17)	8 (20)	0.71
Smoking, (%)	36 (78)	19 (48)	0.005
SBP, mm Hg	113 ± 17	113 ± 19	0.85
DBP,mm Hg	69 ± 10	71 ± 13	0.38
Hemoglobin, (mg/dL)	14 ± 1.7	13 ± 1.5	0.34
Pulse	73 ± 15	76 ± 17	0.42
WBC, (10^9)/L)	13.1±4.4	11.6±3.2	0.09
PLT, x 10^3/μL	260 ± 75	241 ± 83	0.25
Hematocrit, (%)	41 ± 4.7	40 ± 4.6	0.09
CRP, mg/L	19.6 ± 19.6	24.9 ± 24.5	0.27
Sediment, (mm/hour)	16.0 ± 15.4	24.7 ± 22.2	0.03
BUN, (mg/dL)	16.9 ± 4.3	19.3 ± 10.1	0.15
Creatinin, (mg/dL)	0.8 ± 0.2	0.9 ± 0.2	0.03
LDL, (mg/dL)	125 ± 50	110 ± 31	0.13
HDL, (mg/dL)	43 ± 11	42 ± 9	0.49
Total cholesterol, (mg/dL)	198 ± 49	178 ± 43	0.08
Triglyceride, (mg/dL)	144 ± 89	144 ± 62	0.98
Cystatin C, (mg/L)	1.4 ± 0.39	1.6 ± 0.46	0.007

Group 1 (Syntax score <22, mild coronary artery disease);Group 2 (syntax score ≥22, severe coronary artery disease); SBP, Systolic blood pressure; DBP, Diastolic blood pressure;WBC, White blood cell count; PLT, Platelet count; BUN, Blood urea nitrogen; LDL, Low density lipoprotein; HDL, High density lipoprotein

Table 2. Multivariate linear regression analysis showing independent predictor of severe coronary artery disease

	Unstandardized coefficients	Unstandardized coefficients	Standardized coefficients	Standardized coefficients	
Variables	**B**	**SE**	**β**	**t**	**P**
Hyperlipidemia	0.171	3.011	0.007	0.057	0.955
Systolic blood pressure	0.005	0.061	0.009	0.077	0.939
Hypertension	0.371	2.486	0.020	0.149	0.882
Diabetes mellitus	0.411	2.708	0.017	0.152	0.880
Age	0.101	0.090	0.147	1.117	0.268
Smoker	4.542	2.254	0.229	2.015	0.048
Cystatin C	7.577	2.640	0.396	2.870	0.005

Coronary artery disease / Acute coronary syndrome

OP-017

Which is more important for development coronary collateral circulation in chronic coronary syndromes; inflammation or oxidative stress?

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Background and Aim: Myocardial ischemia is of great importance in the development of coronary collateral vessels, which link coronary vessels to each other. However, the reason for the development of different coronary collateral circulation (CCC) networks among people with similar ischemic heart disease remains unclear. Inflammation and oxidative stress are two systems involved in CCC development. In this study, our ultimate aim was to investigate the relationship between inflammation and oxidative stress parameters [such as; thiol, disulfide, neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR), High-sensitivity C-reactive protein (hsCRP)] and CCC.

Methods: A total of 249 patients participated in the study. The patients consisted of those with chronic coronary syndrome (CCS) and total occlusion in at least 1 vessel. We graded CCC in the patients using the Rentrop method and obtained their thiol, disulfide, hsCRP,NLR, and PLR levels. We divided the patients into 2 groups by their CCC grades as poor and good.

Results: Baseline clinical and demographic parameters were similar between groups with good and poor CCC. We discovered more prevalent diabetes mellitus (DM), older age, and more elevated hsCRP levels in the group of patients with poor CCC (p=0.004, p=0.015, and p=0.012, respectively). In multivariate logistic regression analysis, we determined that DM, total thiol, and disulfide to be independent predictors of poor CCC (OR: 1.012, 95% CI: 1.008-1.017, p <0.001; OR: 1.022, 95% CI: 1.000-1.044, p=0.044; OR: 2.671, 95% CI: 1.238-5.761, p=0.012, respectively). The ROC analysis showed a cut-off value of 328.7 for native thiol to predict poor CCC with 78% sensitivity and 67.4% specificity. For disulfide, it revealed a cut-off value of 15.1 to predict poor CCC with 69.5% sensitivity and 57.9% specificity.

Conclusions: In our study, we found that the patients with CCS who developed poor CCC had lower levels of total thiol, native thiol, and disulfide compared to those with good CCC. The most striking finding of this study was that antioxidant cascade, rather than inflammation cascade, was an effective predictor of CCC formation in patients with CCS.

Table 1. Demographic characteristics of the study populations

	Coronary collateral circulation	Coronary collateral circulation	Coronary collateral circulation
	Poor (n=49)	**Good (n=200)**	**P**
Variables			
Age (years)	67 (60-74)	62.5 (55-70)	0.015
Men gender (n, %)	37 (75.5%)	144 (72%)	0,621
Diabetes Mellitus (n, %)	20 (40.8%)	42 (21%)	0.004
Hypertension (n, %)	27 (55.1%)	105 (52.5%)	0.744
Dyslipidemia (n, %)	17 (34.6%)	71 (35.5%)	0.916
Smoking (n, %)	25 (51%)	97 (48.5%)	0.957
BMI (kg/m^2)	27.4 ± 3.8	26.9 ± 2.9	0.639
Systolic blood pressure (mm Hg)	126.8 ± 14.6	125.9 ± 16.3	0.721
Diastolic blood pressure (mm Hg)	73.9 ± 18.5	74.6 ± 17.6	0.859
Heart rate	77.3 ± 8.8	75.6 ± 9.2	0.231
LVEF (%)	55.4 ± 7.6	56.1 ± 9.1	0.551
Previous medications, n (%)			
Aspirin	14 (28.6%)	55 (27.5%)	0.912
B-blocker	11 (22.4%)	46 (23%)	0.932
Angiotensin–aldosterone antagonists	9 (18.3%)	36 (18%)	0.923
Statin	11 (22.4%)	40 (2%)	0.813
Clopidogrel	3 (6.1%)	12 (6%)	0.894

BMI, body mass index, LVEF, left ventricular ejection fraction

Table 2. Laboratory findings of the study populations

	Coronary collateral circulation	Coronary collateral circulation	Coronary collateral circulation
	Poor	Good	P
Number of patients	(n=49)	(n=200)	
Glucose (mg/dl)	127.1 ± 85.2	109.2 ± 73.3	0.125
Creatinine (mg/dl)	0.91 ± 0.2	0.86 ± 0.2	0.153
AST (U/L)	30.3 ± 8.8	35.5 ± 9.6	0.346
ALT (U/L)	24.5 ± 11.2	27.2 ± 12.1	0.454
Total cholesterol (mg/dl)	189 (164-231)	186 (164-219)	0.701
High density lipoprotein cholesterol (mg/dl)	45 (35-53)	41 (35-47)	0.662
Low density lipoprotein cholesterol (mg/dl)	123 (87-145)	115 (93-137)	0.059
Triglyceride (mg/dl)	143 (106-204)	150 (105-215)	0.513
Hemoglobin (mg/dL)	14 (12.5-15)	14 (13-15)	0.816
Platelets (10^3/μL)	267 (232-323)	248 (209-291)	0.027
WBC (10^3/μL)	9.3 (7.9-11.2)	8.9 (7.2-11.3)	0.331
Hs-CRP	3.4 ± 2.9	2.6 ± 1.8	0.012
Neutrophil (10^3/μL)	6.5 (5.5-8.5)	5.4 (3.4-6.4)	<0.001
Lymphocyte (10^3/μL)	2 (1.5-3)	2 (2-3)	0.975
Neutrophil / Lymphocyte ratio (NLR)	3.2 (1.8-6.3)	2.7 (1.7-3.7)	0.008
Platelets / Lymphocyte ratio (PLR)	124 (97-186)	119 (94-116)	0.204
Total thiol	278 (236-387)	432.2 (356.4-534.8)	<0.001
Native thiol	258 (214-344)	384.6 (334.6-462.8)	<0.001
Disulfide	16.2 (10.3-23.7)	22.3 (13.6-33.6)	0.003

NLR; Neutrophil / Lymphocyte ratio, PLR; Platelets / Lymphocyte ratio, CRP: C reactive protein

Table 3. Angiographic data

	Coronary collateral circulation	Coronary collateral circulation	Coronary collateral circulation
	Poor (n=49)	Good (n=200)	P
Rentrop collateral grades:			
0	24		
1	25		
2		137	
3		63	
Position of chronic total occlusion:			
Left anterior descending coronary artery	23 (46.9%)	98 (49%)	0.765
Left circumflex coronary artery	11 (22.4%)	45 (22.5%)	0.952
Right coronary artery	15 (30.7%)	57 (28.5%)	0.736
Number of diseased coronary artery			
One-vessel disease	14 (28.6%)	59 (29.5%)	0.811
Two-vessel disease	18 (36.7%)	68 (34%)	0.797
Three-vessel disease	17 (34.7%)	73 (36.5%)	0.812
Syntax score	19.7 ± 4.5	20.3 ± 5.3	0.645

Table 4. Univariate and multivariate predictors of well-developed coronary collateral circulation in patients with stable coronary artery disease

	Univariate analysis	Univariate analysis	Univariate analysis	Multivariate analysis	Multivariate analysis	Multivariate analysis
	Odds Ratio	95% CI	P	Odds Ratio	95% CI	P
Diabetes Mellitus	2.594	1.336-5.038	0.005	2.671	1.238-5.761	0.012
Age	1.039	1.007-1.073	0.018			
Platelet	1.003	0.999-1.000	0.097			
Hs CRP	1.182	1.033-1.351	0.015			
NLR	1.081	1.002-1.166	0.045			
Total Thyol	1.011	1.007-1.015	<0.001	1.012	1.008-1.017	<0.001
Disulfide	0.961	0.935-0.989	0.006	1.022	1.000-1.044	0.044

NLR, Neutrophil / Lymphocyte ratio, CRP, C-reactive protein

Coronary artery disease / Acute coronary syndrome

OP-018

Evaluation of relationship between semaphorin4D level and coronary slow-flow phenomenon

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Background and Aim: Coronary slow flow (CSF), which is commonly seen angiographic and clinical phenomenon, has complications such as stable and unstable angina pectoris, acute coronary syndromes, hypertension and life-threatening arrhythmias. Semaphorin4D (Sema4D), a novel type I integral membrane glycoprotein, is widely expressed by endothelial cells, platelets, B and T lymphocytes, monocytes and neutrophils. It also plays a significant role in inflammation, oxidative stress, atherosclerosis and angiogenesis. Increased levels of the sema4D were reported in atrial fibrillation, acute coronary syndrome and heart failure.

Aim of the study was to investigate a relation between the sema4D and presence and extent of the CSF.

Methods: The present study included 79 CSF patients and 81 healthy subjects. Serum levels of the sema4D and high sensitive C-reactive protein (hs-CRP) were measured by using an enzyme-linked immunosorbent assay method. Coronary blood flow was quantified by thrombolysis in myocardial infarction (TIMI) frame count (TFC). Moreover, neutrophil to lymphocyte (NLR) ratio was calculated as dividing the absolute neutrophil and lymphocyte counts by the absolute lymphocyte counts. Analyses were done by using appropriate statistical methods.

Results: Neutrophil to lymphocyte ratio and hs-CRP were significantly higher in the CSF group than in the controls ($p < 0.001$). Moreover, sema4D levels were significantly elevated in the CSF group compared to the healthy subjects ($p < 0.001$) (Figure 1). It was importantly positively correlated with hs-CRP (r= 073), $p < 0.001$), mean TFC (r= 088), $p < 0.001$), and NLR (r = 0.37, $p < 0.001$). The area under the receiver operating characteristic curve was 0.990 (95% confidence interval, 0.980 - 1.00, $p < 0.001$) for the sema4D in the diagnosis of the CSF (Figure 2). If a cut-off value of 5.79 ng/ml was used, higher levels of sema4D could predict the presence of the CSF with 97.0% sensitivity and 95.0% specificity.

Conclusions: The present study showed a significant relationship between raised serum sema4D levels and the presence and extent of the CSF in patients with stable angina pectoris, who underwent coronary angiography procedure. Moreover, it was found to be associated with well-known inflammatory markers such as hs-CRP and NLR in this disease. Sema4D may play a crucial role in development and progression of atherosclerosis in coronary arteries, which can be presented as isolated CSF. To clarify this relation, further large scale multicenter clinical studies are needed. Sema4D is a novel biomarker that can be used to identify the presence and extent of the CSF in the setting of stable angina.

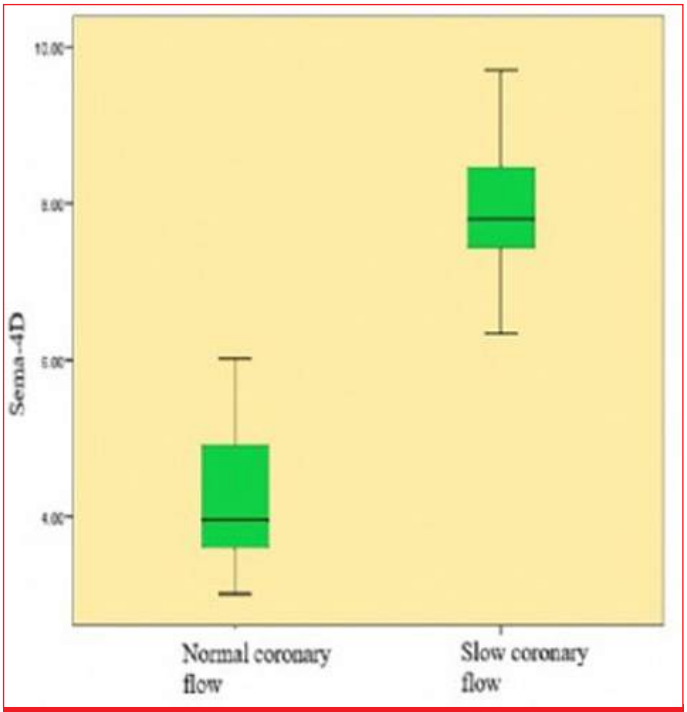

Figure 1. Sema4D levels in patients with the CSF and CNF

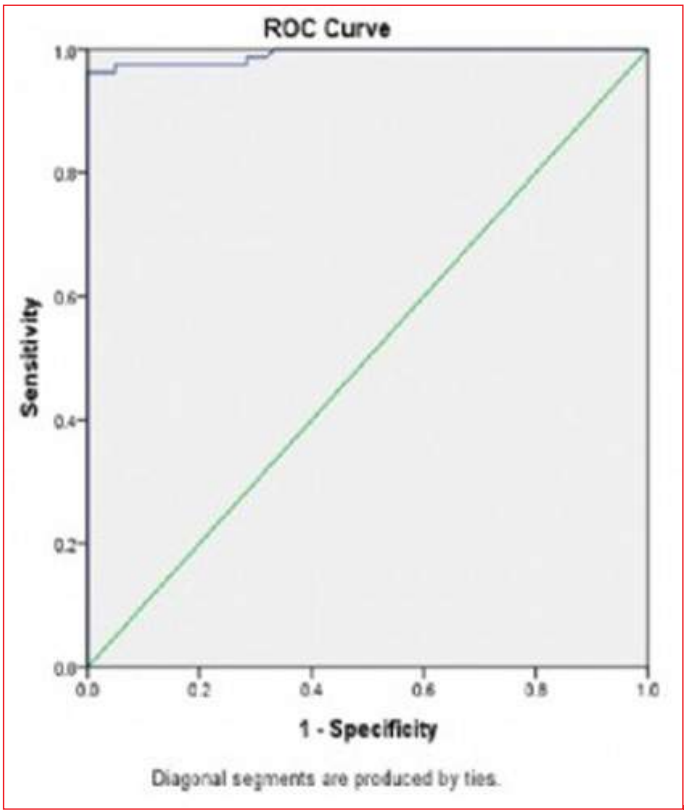

Figure 2. Receiver operating characteristic curve analysis for the discrimination of patients with CSF from CNF

Coronary artery disease / Acute coronary syndrome

OP-019

Coronary artery disease severity scores and lipid levels are associated with ANRIL polymorphisms in female patients

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Background and Aim: Despite recent advances in treatment, coronary artery disease (CAD) is still one of the most important causes of morbidity and mortality worldwide. It has been shown that non-coding RNAs play an important role in the atherosclerotic process. ANRIL is a long non-coding RNA (lncRNA) transcribed from the INK4 locus on chromosome 9p21 in humans. Polymorphisms at this locus have been associated with many cardiovascular diseases. In this study, we aim to investigate the association between ANRIL polymorphisms rs1333049C>G, rs564398T>C, and rs10757274A>G and the occurrence of CAD.

Methods: A total of 1285 subjects were grouped as CAD patients [≥50 stenosis, n=736, (mean age 63.3 ± 10.5 years, 34.4% female)], and non-CAD controls [≤30 stenosis, n=549, (mean age 57.5 ± 11.0 years, 55.5% female)]. Angiographic severity and extent of atherosclerotic CAD were evaluated using Gensini and SYNTAX scores. DNA isolation performed from peripheral blood samples and biochemical analyses were done. Quantitative real time polymerase chain reaction (qRT-PCR) was used for genotyping of rs133049, rs564398, and rs10757274.

Results: For ANRIL rs1333049, female CAD patients with CG genotype had higher Gensini scores (p=0.009) and SYNTAX scores (p=0.027) than the CC genotype. Moreover, female CAD patients with GG genotype had higher SYNTAX scores compared to patients with CC genotype (p=0.045). The rs1333049 CG genotype was associated with higher total cholesterol in female CAD patients as compared to the CC genotype (p=0.043). Furthermore, female patients with GG genotype exhibited an increased triglyceride level than in those with CC genotype (p=0.041). For ANRIL rs10757274 the female patients with GG genotypes had higher SYNTAX scores (p=0.043), total cholesterol levels (p=0.043) and triglyceride levels (p=0.012) compared with female subjects with the AA genotype. Besides, the presence of AG genotype as compared to AA genotype was associated with higher Gensini scores in the female CAD group (p=0.025). There were no associations between the selected polymorphisms and examined parameters in male subjects.

Conclusions: According to these results, ANRIL polymorphisms (rs1333049 and rs10757274) at 9p21 locus were found associated with CAD severity and lipid levels in female patients. These polymorphisms could be useful for the identification of patients with increased risk for severe CAD.

Coronary artery disease / Acute coronary syndrome

OP-020

Effects of mental health condition and sleep quality on angiographic progression in patients with chronic coronary artery disease

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Background and Aim: Previous studies focused on the relationship between chronic coronary artery disease (CAD) and mental health conditions and sleep quality. But the angiographic progression had not been established. The aim of this study was to evaluate the relationship between the angiographically progression of CAD with mental health conditions and sleep quality.

Methods: This is a prospective study of patients who had prior coronary angiography (CAG) because of chronic CAD. Study population had stable angina requiring repeat CAG. Between January 2020 and December 2021, 118 patients were suitable for the study. We applied Beck Anxiety Inventory (BAI), Pittsburgh Sleep Quality Index (PSQI) and STOP-Bang questionnaire. We also compared two groups in terms of CHA2DS2-VASc score. Coronary artery lesions were evaluated by QCA. Also, to confirm the progression of CAD, angiographic scores including Syntax, Gensini, Jeopardy and ΔSyntax, ΔGensini, ΔJeopardy were calculated from the prior and new CAG images. Non-progressors group (n=40) was determined as patients without CAD progression, meanwhile progressors group (n=78) was determined as patients with CAD progression.

Results: There was no statistically significant difference between the two groups in terms of cardiovascular risk factors and medications. But progressors had significantly higher CHA2DS2-VASc score (p=0.005) (Table 1). There were also no significant differences between the groups in laboratory measurements except for high density lipoprotein (HDL). HDL levels were significantly lower in the progressors (p=0.007) (Table 2). Compared to prior CAG, progressors had significant progression in all coronary vessel territories with significantly higher median Syntax, Gensini, and Jeopardy scores at novel CAG (p=0.001) (Table 3). Also, ΔSyntax, ΔGensini and ΔJeopardy scores were significantly higher in the progression group (p=0.001). Progressors had higher BAI score and more frequent patients with moderate-severe anxiety (p<0.05). They had higher PSQI score and poorer sleep quality with higher STOP BANG score and sleep apnea risk (p=0.001) (Table 4). They also had higher daily calorie intake and night eating habit with higher rate of fat-heavy diet but lower protein-heavy diet (p=0.001). As indicated in Table 5, after adjustment using binary logistic regression, low HDL levels (OR:1.59, p=0.023), poor sleep quality (OR:2.10, p=0.002), high apnea risk (OR:1.64, p=0.029), moderate or severe anxiety (OR: 2.32, p=0.023), fatty heavy diet (OR:1.71,

p=0.012), and high CHA2DS2-VASc score (OR:2.12, p=0.004) were independently associated with significant risk of angiographic CAD progression.

Conclusions: To our knowledge, this was the first study suggesting that angiographically proven chronic CAD progression was associated with anxious mental condition and poor sleep quality including high apnea risk. In addition, it was determined that fatty heavy diet, low HDL level and high CHA2DS2-VASc score had significant effect on progression.

Table 1. Baseline characteristics of the patients

Variables	All Patients (n=118)	Group 1 (n =40)	Group 2 (n = 78)	p value
Patient characteristics				
Age	60,56±8,69	60.85±8.57	59.71±9.24	0.51
Male	76(90)	65(26)	82.1(64)	0.07
BMI	29,73±5,22	29.82±4.98	29.05±5.04	0.43
Current smoker	23%	21%	25%	0.53
Alcohol abuse	6.3(14)	5.3(4)	6.8(10)	0.65
LVEF, %	53 ± 10	55 ± 8	52 ± 10	0.15
Previously known diseases				
Hypertension	65. 3 (77)	70(28)	62.8(49)	0.44
Diabetes Mellitus	41.5(49)	37.5 (15)	43.6 (34)	0.53
Hyperlipidemia	54.2 (64)	55(22)	53.8(42)	0.91
Prior history of CAD	99.2(117)	97.5(39)	100(78)	0.34
Prior history of stroke	5.9 (7)	7.5 (3)	5.1(4)	0.44
Family history of CAD	22(26)	25(10)	20.5(16)	0.58
Heart Failure	11(13)	12.5(5)	10.3(8)	0.71
Chronic kidney disease	11.9(14)	10(4)	12.8(10)	0.65
CHA2DS2-VASc score	2.72 ± 1.54	2.18 ± 0.95	3 ± 1.7	0.005
Vital signs at rest				
Systolic blood pressure, mmHg	127,08±18,81	128.85±17.58	125.17±19.54	0.32
DBP, mmHg	75,18± 10,99	76.7±11.86	73.97±10.87	0.21
Mean blood pressure	90,97±15,52	93.85±13.34	89.24±15.67	0.12
Heart rate, beats/min	78,02±11,75	77.98±10.84	77.62±12.10	0.88
Previous Medications				
Beta-blockers	67.8(80)	70(28)	66.7(52)	0.71
ACE inhibitors/ARB	55.9(66)	50(20)	59(46)	0.35
Acetyl salicylic acid	77.1(91)	67.5(27)	82.1(64)	0.08
Statins	49.2(58)	50(20)	48.7(38)	0.90
Diuretics	19.5(23)	20(8)	19.2(15)	0.92
Nitrates	45,6%	41,8%	47,9%	0.31
Calcium Channel Blockers	26,2%	23,6%	27,7%	0.45
Ranolazine	35,9%	39,1%	34,0%	0.38

Table 2. The laboratory findings of the patients.

Variables	All Patients (n= 118)	Group 1 (n= 40)	Group 2 (n= 78)	p value
White blood cell count, $\times10^9$/L	8.7 (7.4-10.4)	8.6 (7.2-9.9)	9.6 (7.2-11.4)	0.97
Hemoglobin, g/dL	13.65 ± 2.2	13.59 ± 2.1	13.77 ± 2.15	0.19
Platelet, $\times10^9$/L	246.96 ± 92.5	253.56 ± 76	244.92 ± 85.25	0.49
Neutrophil, $\times10^9$/L	5.1 (4.1-6.5)	5 (4.4-5.9)	5.5 (4.1-6.6)	0.26
Lymphocyte, %10^9/L	2.1 (1.7-3.0)	2.1(1.8-3.0)	2.3(1.8-3.3)	0.34
Neutrophil-to-lymphocyte ratio	2,3 (1.6-3.0)	2,1 (1.5-2.7)	2,2(1.6-2.8)	0.30
Glucose, mg/dL	144.2 ± 75.88	143.84 ± 67.32	144.4 ± 80.72	0.57
HbA1c, %	7,27 ± 1,71	7,51 ± 1,96	7,18 ± 1,61	0.35
Creatinine, mg/dL	0,92 (0.8-1.14)	0,90 (0.75-1.13)	0,92(0.8-1,17)	0.29
GFR, mL/min/1.73 m^2	82 (62-96)	88 (69-96)	78 (58-95)	0.11
AST, U/L	21 (15-31)	20 (15-34.5)	21 (14-34)	0.06
ALT, U/L	18 (12-28)	14 (11-23.5)	18 (11.75-26)	0.54
Albumin, g/dL	41.35 ± 6.12	40.79 ± 7.43	41.81 ± 4.87	0.15
Hs-cTnT, ng/L	7 (3,9-13.8)	7,79 (3,2-14,1)	8,45(6,18-15,71)	0.92
CKMB, ng/mL	2.0 (1.44-2.9)	1,76(1,27-2,7)	2,08(1,46-2,1)	0.21
High-density lipoprotein, mg/dL	42.58 ± 13.88	51.3 ± 16.73	41.71 ± 10.9	**0.007**
Low-density lipoprotein, mg/dL	99.41 ± 46.2	100.36 ± 36.12	97.7 ± 50.8	0.62
Triglyceride, mg/dL	146 (110-199)	183 (103-223.5)	135 (100-183.2)	0.28
Total Cholesterol, mg/dL	182.42 ± 44.5	174.35 ± 45.82	196 ± 50.1	0.08
C-reactive protein, mg/dL	3,5 (1,4-12)	2,2 (1.2-7)	3,8 (1.6-14)	0.28

Table 3. Angiographic features of the patients.

Variables	All Patients (n= 118)	Group 1 (n= 40)	Group 2 (n= 78)	p value
Time between CAGs, months	26 (10 - 56)	31 (10 - 49)	24 (13.3 - 56)	0.91
Previous angiographic features				
Double-vessel disease, n (%)	16 (13.4)	4 (10)	12 (15.3)	0.33
Triple-vessel disease, n (%)	4 (3.3)	1 (2.5)	3 (3.8)	0.44
Syntax Score	0 (0 – 10.3)	1 (0 - 9)	0 (0 - 13)	0.98
Gensini Score	15 (2.5 – 49.6)	10.5 (0 - 52)	16 (5.2 - 48)	0.07
Jeopardy Score	0 (0 - 2)	0 (0 - 0)	0 (0 - 2)	0.09
LMCA, n (%)	7 (5.9)	2 (5)	5 (6.4)	0.38
LAD territory, n (%)	18 (15.2)	5 (12.5)	13 (16.6)	0.42
CX territory, n (%)	38 (32.2)	10 (25)	28 (35.9)	**0.03**
RCA territory, n (%)	21 (17.8)	5 (12.5)	16 (20.5)	0.12
Novel angiographic features				
Double-vessel disease, n (%)	8 (6.7)	2 (5)	6 (7.6)	0.14
Triple-vessel disease, n (%)	19 (16.1)	4 (10)	15 (19.2)	**0.04**
Syntax Score	3 (8 - 19)	0 (0 - 8.4)	14 (7 - 19.5)	**0.001**
Gensini Score	16 (5.3 - 48)	9.5 (0 – 34.5)	44 (23.3 – 74.3)	**0.001**
Jeopardy Score	2 (0 - 4)	0 (0 - 2)	4 (2 - 6)	**0.001**
LMCA, n (%)	12 (10.1)	2 (5)	19 (12.8)	**0.04**
LAD territory, n (%)	49 (41.5)	7 (17.5)	42 (53.8)	**0.001**
CX territory, n (%)	49 (41.5)	11 (27.5)	38 (48.7)	**0.001**
RCA territory, n (%)	53 (44.9)	11 (27.5)	42 (53.8)	**0.001**
Coronary progression				
ΔSyntax Score	3 (0 – 8.5)	0 (0 - 0)	8 (3 - 11)	**0.001**
ΔGensini Score	8 (0 - 25)	0 ([-1.5] - 2)	12 (5.3 - 30)	**0.001**
ΔJeopardy Score	3 (0 – 8.5)	0 (0 - 0)	4 (0 - 6)	**0.001**

Table 4. Mental health conditions, sleep characteristics and socioeconomics features of the patients.

Characteristics	All Patients (n= 118)	Group 1 (n= 40)	Group 2 (n= 78)	p value
Behavioural Characteristics				
Beck Depression Score	15.32 ± 9.03	13.73 ± 6.4	16.14 ± 10.05	0.12
Moderate-severe DeM, n (%)	38,7%	30,5%	42,5%	0,12
Beck Anxiety Score	14.64 ± 7.51	11.71 ± 5.15	16.02 ± 8,07	**0.02**
Moderate-severe AnM, n (%)	21 (17.7)	4 (10)	17 (21.8)	**0.03**
Sleep Characteristics				
Sleep Duration, mid-week, h	7.1 ± 1.3	7.3 ± 1.2	6.8 ± 1.2	**0.04**
Sleep Duration, weekend, h	7.3 ± 1.3	7.2 ± 1.4	7.4 ± 1.3	0.44
Pittsburgh Sleep Quality Index	7 ± 3.8	5.4 ± 3.1	7.9 ± 3.9	**0.001**
Poor sleep quality, n (%)	80 (67.8)	21 (52.5)	59 (74.3)	**0.001**
STOP-BANG score	4,3± 1,38	3,78±1,44	4,54± 1,29	**0.001**
High risk for sleep apnea, n (%)	50,0%	5,1%	70,9%	**0.001**
Dietary characteristics				
Average daily calorie intake, kcal	2132.8 ± 348.4	2029.8 ± 283.4	2248.3 ± 336.7	**<0.001**
Fat heavy diet, n (%)	30,1%	8,5%	40,2%	**0.001**
Carbohydrate heavy diet, n (%)	36,6%	37,3%	36,2%	0,90
Protein heavy diet, n (%)	32,3%	50,8%	23,6%	**0.001**
Having night eating habit, n (%)	44 (37.2)	10 (25)	34 (43.6)	**0.017**
Socioeconomic characteristics				
Married, n (%)	110 (93.2)	37 (92.5)	73 (93.6)	0.82
Being parent, n (%)	102 (86.4)	30 (75)	72 (92.3)	**0.001**
Low education level, n (%)	31 (26.3)	7 (17.5)	24 (30.7)	**0.001**
High education level, n (%)	41 (34.7)	13 (32.5)	28 (35.9)	0.77
Active worker, n (%)	41 (34.7)	10 (25)	31 (39.7)	**0.042**
Low level of income, n (%)	65 (55)	25 (62.7)	40 (52.5)	0.31
High level of income, n (%)	18(15.3)	2 (5)	16 (20.3)	**0.032**

Table 5.

Variables	Adjusted *P*	OR*	95% CI	
			Lower Bound	*Upper Bound*
Low HDL levels	0.023	1.59	1.07	2.34
Poor Sleep Quality	0,002	2.10	1.32	3.34
High apnea risk	0.029	1.64	1.05	2.54
M/S Anxiety	0.023	2.32	1.12	4.83
Fatty heavy diet	0.012	1.71	1.12	2.60
High CHADSVASc	0.004	2.12	1.26	3.56

*Adjusted for age, gender, cardiovascular risk factors, presence of moderate or severe depression, protein heavy diet, socioeconomic features, and treatments including statin and aspirin use.

Coronary artery disease / Acute coronary syndrome

OP-021

Effect of obesity paradox on mortality in patients with acute coronary syndrome: A comprehensive meta-analysis of the literature

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Background and Aim: Obesity prevalence has increased dramatically in worldwide and obesity is the major risk factor for cardiovascular diseases. However, some studies have shown that obesity might have a protective role in coronary artery disease, which is referred as an obesity paradox. It has been postulated that the obesity paradox is associated with a decreased risk of myocardial infarction (MI), and an improvement in the outcomes with a reduced risk of death due to MI. We aimed to investigate the role of the obesity paradox on mortality in patients with acute coronary syndrome (ACS) in this meta-analysis.

Methods: We searched PubMed, Google Scholar, and Cochrane libraries for studies that compared mortality of ACS patients according to their body mass index. After reviewing all eligible studies, this meta-analysis was conducted with remaining 53 studies. Heterogeneity was assessed with Higgin's I2 and Cochrane Q tests. Publication bias was tested with Egger's regression test and was visualized with a Funnel plot.

Results: This meta-analysis consisted of 53 studies with 515,095 patients. The overweight patients had lower 30-day (RR=0.69, 0.62-0.76, p<0.01, I2=65%) and long-term mortality (RR=0.73, 0.70-0.77, p<0.01, I2=47%) than patients with normal weight. The 30-day and long-term mortalities were lower in obese patients than in normal weight patients (RR=0.69, 0.62-0.76, p<0.01, I2=65%, RR=0.68, 0.62-0.75, p<0.01, I2=94%; respectively). Patients with low weight had higher mortality rates than patients with normal weight for 30-day and long-term (RR=1.74, 1.39-2.18, p<0.01, I2= 40%, RR= 2.00, 1.51-2.63, p<0.01, I2=96%; respectively) (Figure 1). Four studies (Nikolsky 2006, Nigam 2006, Kennedy 2005, and Angeras 2013) were detected as outliers and Kennedy 2005 was an influential study in comparison of long-term mortality between obese and normal weights. In sensitivity analysis, the pooled effect was still significant after removing these 4 studies with a lower heterogeneity (RR=0.66, 0.62-0.72, p<0.001; I2=58.7%). There might be publication bias in the pooled estimate of the long-term mortality between obesity and normal weight, which had a small study effect (Figure 2). To handle the bias, a bias-adjusted estimation was re-calculated using Duval&Tweedie Trim and Fill method. A bias-adjusted estimate was re-calculated by adding 6 studies for missing studies and the result did not change (RR=0.68, 0.63-0.74, p<0.001, I2=59.5). Five studies (Lazzeri 2012, Zeller 2008, Angeras 2013, Samanta 2018, and Li 2013) were detected as outliers, and studies Li 2013 and Angeras 2013 were influential studies for long-term mortality comparison between overweight and normal weight. The effect estimate did not change after removing these studies with a low heterogeneity (RR=0.72,0.70-0.73, p<0.001, I2=5%).

Conclusions: Increased BMI was linked to lower mortality risk for 30-day and long-term in patients with ACS in this meta-analysis, which might highlight the obesity paradox among these patients.

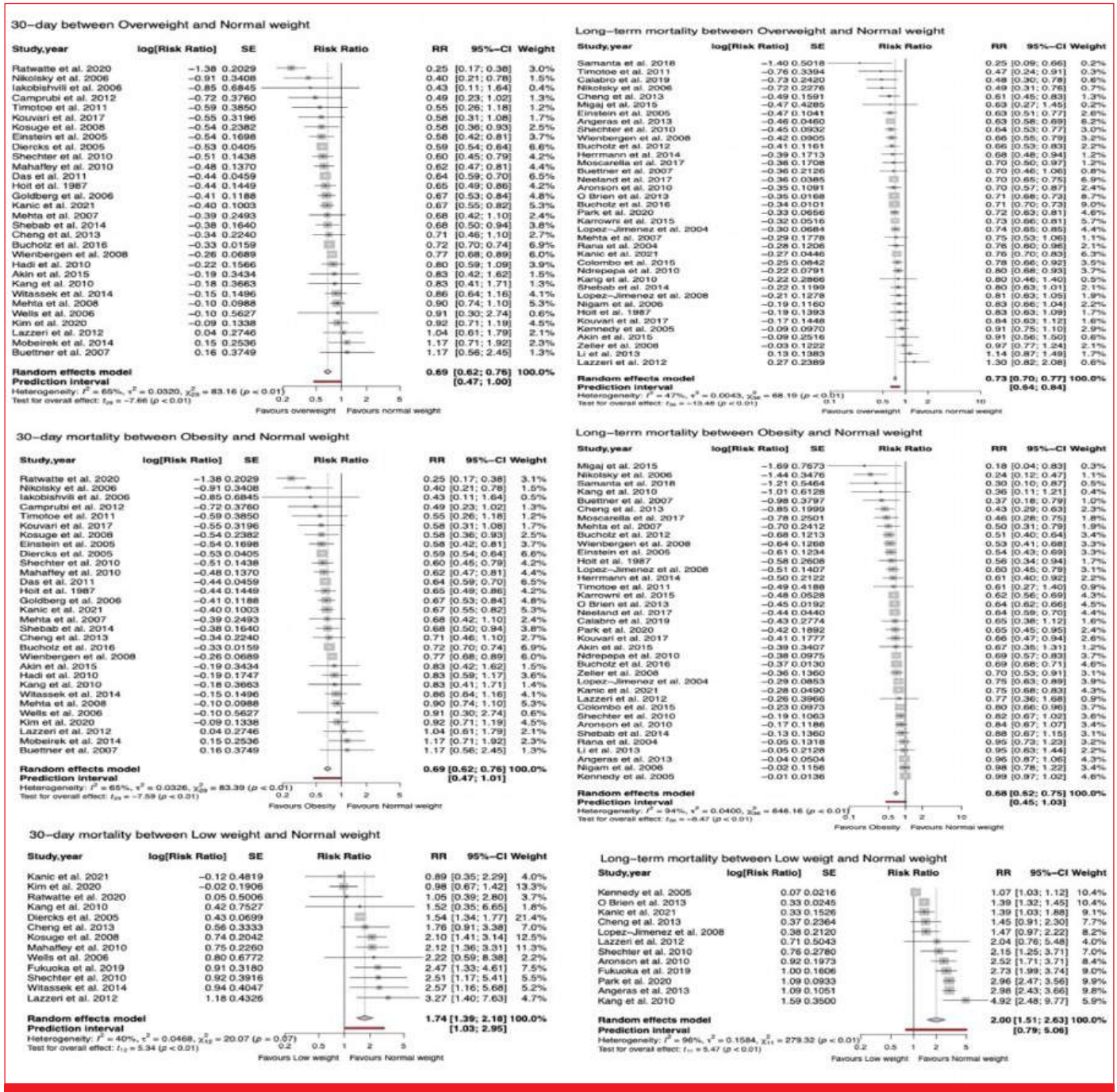
30–day between Overweight and Normal weight
Long–term mortality between Overweight and Normal weight
30–day mortality between Obesity and Normal weight
Long–term mortality between Obesity and Normal weight
30–day mortality between Low weight and Normal weight
Long–term mortality between Low weigt and Normal weight

Figure 1.

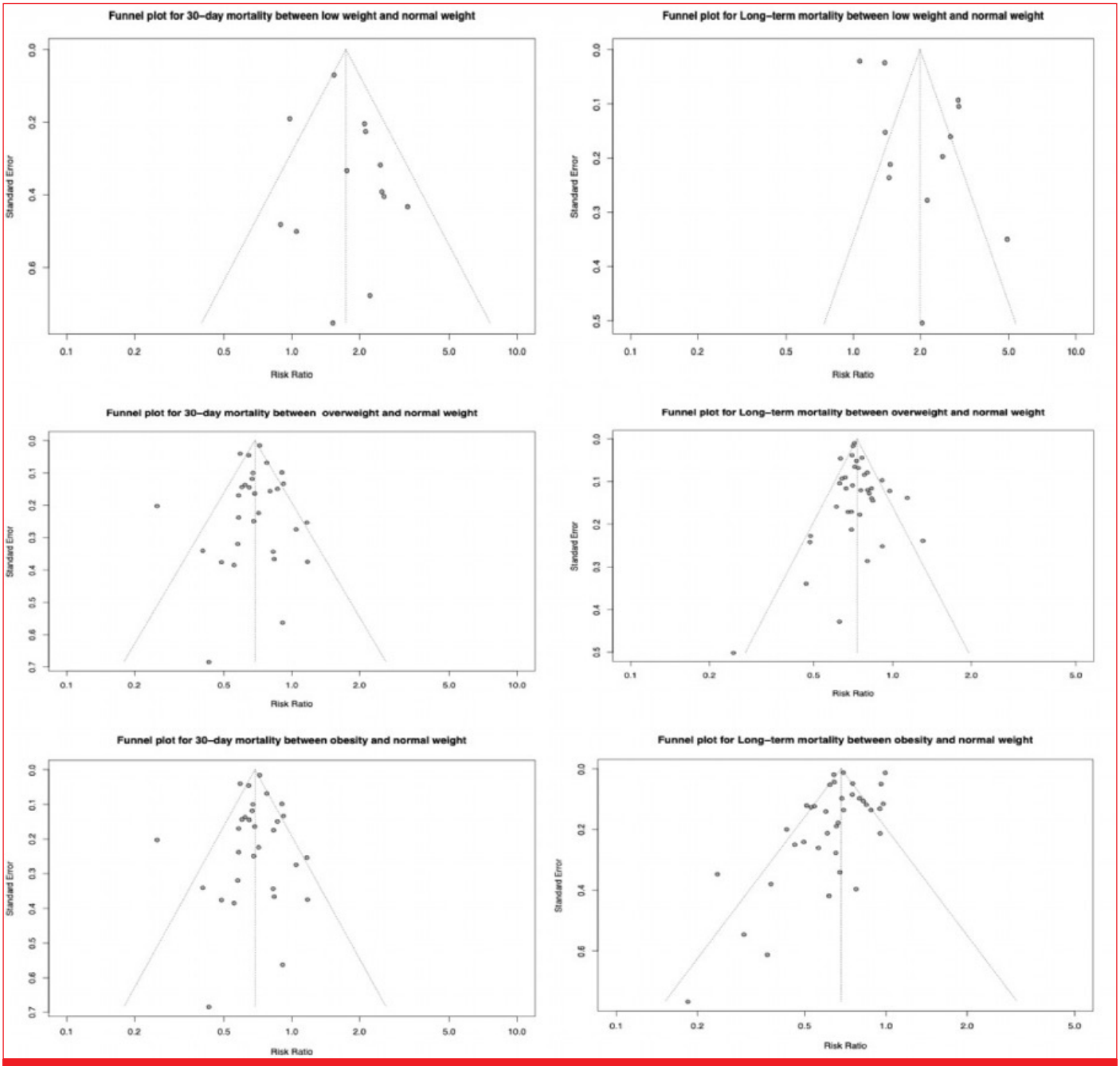

Figure 2.

Coronary artery disease / Acute coronary syndrome

OP-022

Copeptin levels predict left ventricular systolic function in STEMI patients A 2D speckle tracking echocardiography-based prospective observational study

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Background and Aim: In the present study, we aimed to investigate whether copeptin values on admission are related to left ventricle (LV) systolic function and its improvement at 6 months in ST-segment elevation myocardial infarction (STEMI) patients.

Methods: In this single-center, prospective observational study, we included 122 STEMI patients from January 2016 to November 2016. LV systolic functions in the form of global longitudinal strain (GLS) in addition to conventional echocardiography parameters were evaluated on admission and at 6-month. Serum copeptin levels were determined using an ultrasensitive immunofluorescence assay.

Results: The study population was divided into 2 groups according to median values of copeptin. GLS was significantly lower in patients with high copeptin levels compared to those with low copeptin levels at early stage and 6-month [-16% (16–16.5) vs. -15% (15–15.5), p<0.001 and -18% (18–19) vs. -16% (16–16.25), p<0.001, respectively]. Copeptin values were negatively correlated with an early and 6-month GLS (r=−0.459 at early stage and r=−0.662 at 6-month). In addition, we observed that copeptin values were negatively correlated with the improvement of GLS at 6-month follow-up (r=-0.458, P<.001 and r=-0.357, P=.005, respectively).

Conclusions: Serum copeptin levels in STEMI patients at the time of admission may predict early and 6-month LV systolic function assessed by two-dimensional GLS. To the best of our knowledge, this study is the first to specifically address the relationship between copeptin values and GLS in STEMI patients.

Table 1. Correlation between copeptin and echocardiographic parameters at baseline and 6-month.

Correlation between copeptin and echocardiographic parameters at baseline and 6-month.

	Baseline		6-month		Δ	
	r	P value	r	P value	r	P value
LV EF, %	−0.299	.019	−0.410	.001	−0.458	.001
LV GLS, %	−0.459	<.001	−0.662	.001	−0.357	.005

r=Spearman rho correlation coefficient; Δ=difference between baseline and 6-month
EF=ejection fraction, GLS=global longitudinal strain, LV=Left ventricle

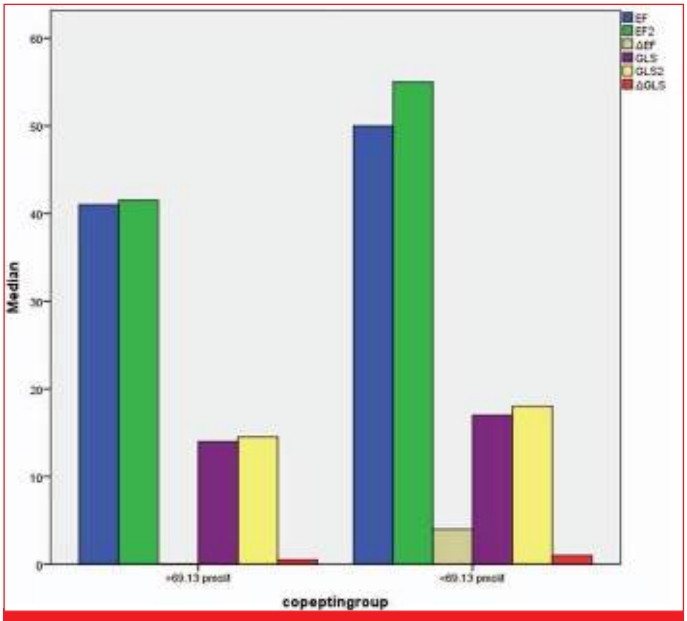

Figure 1. Echocardiography parameters according to median copeptin groups

Coronary artery disease / Acute coronary syndrome

OP-023

CHA2DS2VASc-HSF score predicts the coronary slow flow phenomenon in patients with non-obstructive coronary artery disease

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Background and Aim: In this study, we aimed to investigate the relationship between the CHA2DS2VASc-HSF score and coronary slow flow phenomenon (CSFP) in patients who underwent elective coronary angiography with suspected coronary artery disease.

Methods: This retrospective single-center study included 68 consecutive patients with CSFP who underwent coronary angiography between April 2021 and July 2022 and without obstructive coronary artery disease. Patients with CSFP were compared to 68 controls with age- and sex-matched normal coronary flow as evidenced by coronary angiography. Thrombolysis in Myocardial Infarction (TIMI) frame count (TFC) was used to measure coronary blood flow velocity. The CHADS2, CHA2DS2-VASc, and CHA2DS2VASc-HSF scores were calculated for all the patients.

Results: This research showed that the patient with CSFP has significantly higher CHA2DS2VASc-HSF score compared to normal flow, while the CHA2DS2 and CHA2DS2-VASc scores were similar (3.75 ± 1.27 vs. 2.85 ± 1.11, respectively; p < 0.001). The multivariate logistic regression analysis revealed that CHA2DS-2VASc-HSF score was an independent predictor of CSFP [OR:1.635, 95% confidence interval (CI) 1.053–2.540; p=0.029], and was positively correlated with the TFC in the CSFP group (r = 0.848, p < 0.001). The area under the receiver operating characteristic curve of CHA2DS2VASc-HSF for the diagnosis of CSFP was 0.70 (95% CI 0.61–0.78; p < 0.001). If a cut-off value of 3.5 was used, higher levels of CHA2DS2VASc-HSF score could predict the presence of CSFP with 56% sensitivity and 74% specificity.

Conclusions: Our study results suggest that CHA2DS-2VASc-HSF score is associated with CSFP and may be useful for predicting CSFP and its severity.

Table 1. CHADS2VASc-HSF score. *Definition of CHADS-2VASc-HSF score*

C	Congestive heart failure	1 point
H	Hypertension	1 point
A2	Age ≥ 75 years	2 point
D	Diabetes mellitus	1 point
S2	Previous stroke or TIA	2 point
V	Vascular disease	1 point
A	Age 65–74 years	1 point
Sc	Sex category (male gender)	1 point
H	Hyperlipidaemia	1 point
S	Smoking	1 point
F	Family history of CAD	1 point

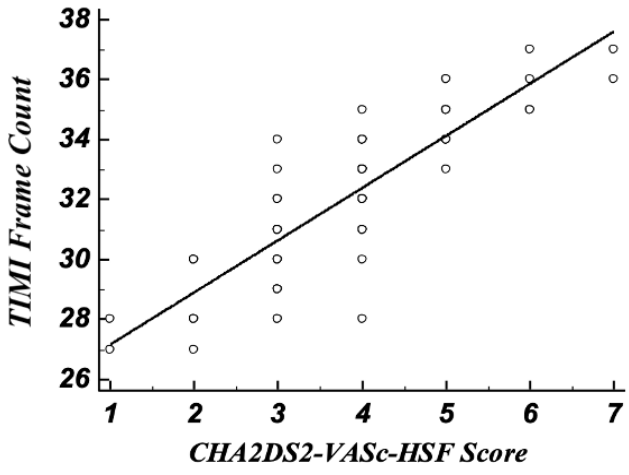

Figure 1. Correlation graph. Correlation graph of TIMI frame count and CHA2DS2VASc-HSF score

Table 2. Multivariate regression analysis. *Multivariate regression analysis to predict slow-flow phenomenon*

Variable	OR	95%CI	p-value
Age	0,999	0,955-1,045	0,964
Male Gender	0,928	0,381-2,262	0,869
Smoking	1,380	0,575-3,311	0,471
Hyperlipidemia	1,749	0,782-3,913	0,174
Family history	1,359	0,542-3,410	0,513
CHA_2DS_2VASc-HSF score	1,635	1,053-2,540	0,029*

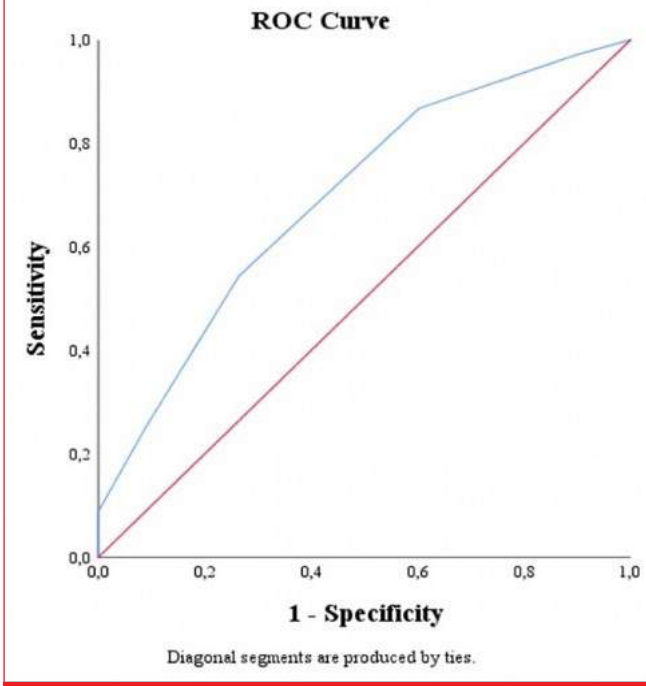

Figure 2. ROC curve graph. ***ROC curve graph of CHA2DS-2VASc-HSF score predicting slow flow phenomenon***

Coronary artery disease / Acute coronary syndrome

OP-024

Comparison of the effects of carvedilol and metoprolol treatments on the development of contrast-induced nephropathy after percutaneous coronary intervention in patients presenting with acute coronary syndrome

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Background and Aim: Carvedilol has been shown to inhibit inflammation, vasoconstriction and oxidative stress, which play important roles in the development and progression of contrast-induced nephropathy (CIN). However, to the best of our knowledge, no studies have investigated the potential effect of carvedilol on the prevalence of CIN after percutaneous coronary intervention (PCI) in the setting of acute coronary syndrome (ACS). Herein, this study aimed to determine whether carvedilol use is associated with the development of CIN.

Methods: Medical records of 319 patients (mean age, 59.19 ± 12.42 years; 77.7% male) with ACS who underwent urgent PCI at our institution between May 2019 and April 2022 were included prospectively. Overall, 100 and 219 patients were assigned to the carvedilol and metoprolol succinate groups, respectively. Serum creatinine levels were examined preoperatively and 24–48 h postoperatively and compared between groups. Data were grouped according to the occurrence of CIN, and univariate analysis was conducted to exclude suspected influencing factors that led to CIN occurrence. Logistic regression analysis was performed, after adjusting for confounding factors, to assess the association between carvedilol administration (independent variable) and CIN occurrence (dependent variable).

Results: CIN was detected in 46 (14.4%) patients. The prevalence of CIN was significantly lower in the carvedilol group (6.0%) than in the control group (18.3%; p=0.003). Results of the logistic regression analysis revealed that carvedilol [odds ratio (OR) 0.250, 95% confidence interval (CI) 0.092-0.677, p=0.006], amount of contrast agent (OR 1.004, 95% CI 1.000-1.008, p=0.031), and admission estimated glomerular filtration rate (OR 0.978, 95% CI 0.960-0.995, p=0.014) were independently associated with the development of CIN.

Conclusions: The use of carvedilol, in addition to hydration therapy and statin administration appear to be a promising method for the prevention of CIN in patients with ACS undergoing urgent PCI. Further multicenter studies are required in this regard.

Heart failure

OP-025

Characteristics and long-term survival of patients with left ventricular non-compaction cardiomyopathy

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Background and Aim: Left ventricular non-compaction cardiomyopathy (LVNC) is a poorly understood entity resulting in heart failure. Whether it is a distinct form of cardiomyopathy or an anatomical phenotype is a subject of discussion. The current diagnosis is based on morphologic findings by comparing the compacted to non-compacted myocardium. The aim of the study was to compare demographic and prognostic variables of patients with dilated cardiomyopathy (DCM) and LVNC. Emphasis was given to cardiac magnetic resonance imaging (CMR) analysis. Data on survival was also assessed.

Methods: We retrospectively evaluated the characteristics and outcomes of 262 non-ischemic cardiomyopathy patients with LVNC and DCM phenotypes. Petersen's CMR criteria of non-compacted to the compacted myocardial ratio of 2.3 was used to diagnose LVNC. The primary endpoint was a composite end-point of major adverse cardiovascular events (MACE) comprising cardiovascular-related death, left ventricular assisted device (LVAD) implantation and heart transplantation surgery. A total of 262 patients with CMR data were included in the study (Figure 1).

Results: One hundred fifty-five patients who fulfilled CMR criteria were diagnosed as LVNC. CMR findings revealed that LVNC patients had higher left ventricular end-diastolic (137.2 ± 51.6, 116.8 ± 44.6, p=0.002) and systolic volume index (98.4 ± 49.5, 85.9 ± 42.7, p=0.049). However, associated cardiac hemodynamic variables like cardiac output (5.61 ± 2.03, 4.96 ± 1.83; p=0.010), stroke volume (73.9 ± 28.8, 65.1 ± 25.1, p=0.013), and cardiac index (2.85 ± 1.0, 2.37 ± 0.72; p<0.0001) were higher in LVNC patients (Table 3). Of all the 249 patients, 102 (40.9%) patients demonstrated LGE. According to Petersen criteria, the Kaplan-Meier survival outcome did not reveal significant differences (p=0.11, HR:1.53; 95% CI: [0.89-2.63]). The presence or pattern of LGE did not show significant importance for primary endpoint-free survival (Figure 2). When ROC analysis was applied for NC/C ratio to discriminate primary endpoint, a higher NC/C ratio of 2.57 was associated with adverse events (p=0.016, HR:1.90, 95% CI: [1.12-3.24]) (Graphical Abstract A).

Conclusions: This study evaluated patients diagnosed with CMR using Petersen criteria of 2.3 and compared the demographic and prognostic data to patients with DCM. There was no difference between the groups regarding prognosis and survival. However, our analysis revealed new criteria of 2.57. When we re-categorize the patients with this new criterion, demographic variables and survival analysis showed statistically significant differences. In 94.4% of LVNC patients, sub-epicardial LGE pattern was the main finding which could be a spesific predictor for prognosis. Different demographic data may indicate that there is a LVNC cardiomyopathy. It is probable that the criteria being used for the diagnosis is far from being perfect. We believe that apart from ejection fraction and LGE, the degree of trabeculation is essential for the diagnosis and prognosis of this perhaps distinct entity.

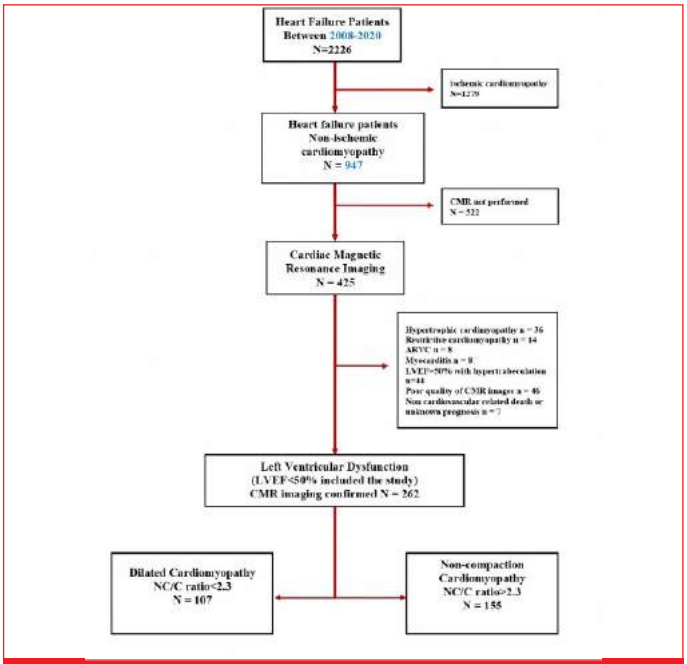


Figure 1. Flow chart of study patients/consort diagram

Patients with HF attributable to ischemic etiology, valvular heart disease or unacceptable unloading conditions, hypertrophic cardiomyopathy, restrictive cardiomyopathy and pediatric patients were excluded. Ischemic etiology was proved by coronary angiography, coronary computed tomography, positive myocardial perfusion scintigraphy for ischemia or CMR with transmural LGE. Stenosis of proximal left anterior descending coronary artery over 50% or two coronary segments with stenosis over 50% were agreed to have the possibility of ischemic HF etiology and were also excluded from the study. CMR, Cardiac magnetic resonance, ARVC, Arrhytmogenic right ventricular cardiomyopathy, C, compacted myocardium, NC, non-compacted myocardium, NC/C ratio, ratio of the noncompacted segment to compacted segment of the myocardium.

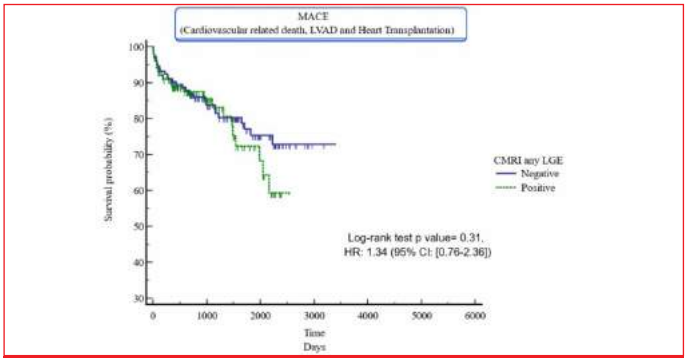
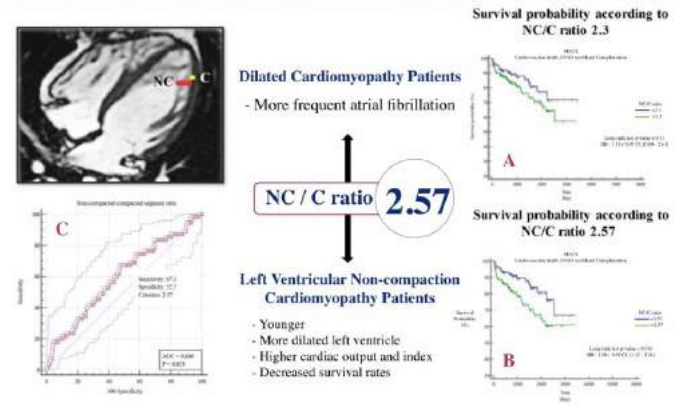


Figure 2. MACE free survival according to cardiac magnetic resonance imaging late gadolinium enhancement

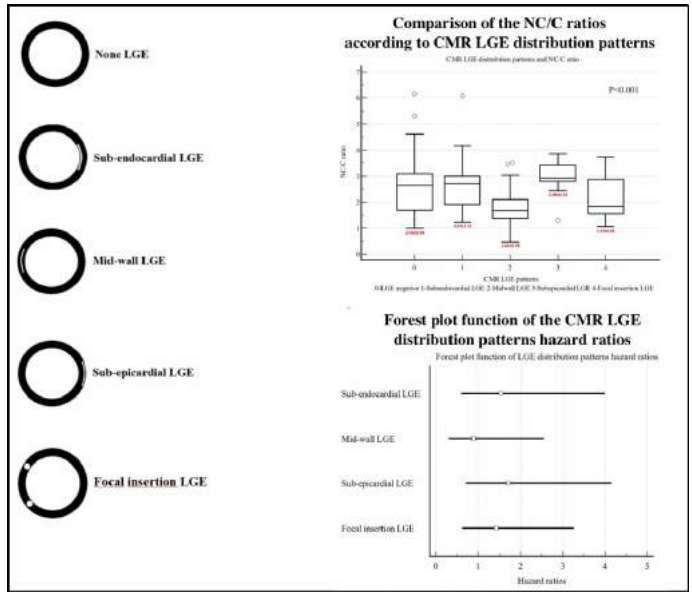
Kaplan Meier survival analysis estimates free of major cardiovascular events in the groups of patients left ventricular cardiac magnetic resonance imaging according to late gadolinium enhancement.

Graphical Abstract A: Schematic drafting of non-compacted to compacted myocardial length ratio and comprasion MACE free survival according to NC/C ratio 2.3 and 2.57



A- Kaplan-Meier survival analysis estimates free of major cardiovascular events in the groups of patients left ventricular non-compaction cardiomyopathy and dilated cardiomyopathy discriminated according to Petersen et al. CMR criteria. B- Kaplan-Meier survival analysis estimates free of major cardiovascular events in the groups of patients left ventricular non-compaction cardiomyopathy and dilated cardiomyopathy discriminated according to re-define NC/C ratio 2.57 CMR criteria. C- ROC curve analysis NC/C ratio for primary endpoints. Atrial fibrillation were more frequent in dilated cardiomyopathy patients and left ventricular non-compaction cardiomyopathy patients were younger, left ventricle more dilated, had better hemodynamic features and decreased survival according to NC/C ratio 2.57.

Graphical Abstract B: Schematic drafting of cardiac magnetic resonance late gadolinium enhancement distribution patterns



Mid-wall LGE pattern was primarily seen in the DCM patients, 24 of 31 (77%), and midwall LGE had an estimated hazard ratio of 0.88 [95% CI: (0.34-2.24)] compared to non-LGE patients. In contrast to mid-wall LGE distribution, a sub-epicardial LGE pattern was the main finding in 17 (94.4%) LVNC patients. Estimated and adjusted HR for patients with sub-epicardial LGE was 1.69 [95% CI: (0.60-4.80)], compared to non-LGE patients.

Table 1. Clinical characteristics of patients and comparison according to NC/C ratios

	Total	NC/C ratio < 2.3	NC/C ratio > 2.3	P-value	NC/C ratio < 2.57	NC/C ratio > 2.57	P-value
Age (years)	42.8 ± 14	45.2 ± 14.3	41.2 ± 13.7	0.026	44.8 ± 14.1	40.9 ± 13.8	0.017
Male (%)	168 (64.1%)	74 (69.1%)	94 (60.6%)	0.19	86 (68.3%)	82 (60.3%)	0.19
BMI (kg/m^2)	29.7±6.4	30.6±6.7	29.1±6.1	0.066	30.6±6.4	28.9±6.3	0.037
BSA (m^2)	2.0±0.26	2.04±0.28	1.97±0.24	0.037	2.04±0.28	1.96±0.24	0.014
Smoker n (%)	80 (31.5%)	39 (37.9%)	41 (27.2%)	0.075	45 (36.9%)	35 (26.5%)	0.080
Arterial hypertension n (%)	54 (20.9%)	19 (17.9%)	35 (23%)	0.32	21 (16.8%)	33 (24.8%)	0.12
Diabetes mellitus n (%)	44 (17.1%)	17 (16%)	27 (17.8%)	0.74	23 (18.4%)	21 (15.8%)	0.62
Dyslipidemia n (%)	18 (7.0%)	10 (9.4%)	8 (5.3%)	0.22	10 (8.0%)	8 (6.0%)	0.62
Stroke n (%)	14 (5.3%)	7 (6.6%)	7 (4.6%)	0.58	9 (7.2%)	5 (3.8%)	0.27
COPD n (%)	24 (9.3%)	10 (9.4%)	14 (9.2%)	0.95	11 (9%)	13 (9.9%)	0.80
Renal Disease n (%)	7 (2.7%)	2 (1.9%)	5 (3.3%)	0.70	2 (1.6%)	5 (3.8%)	0.29
AF n (%)	33 (12.6%)	19 (18.6%)	14 (10%)	0.060	23 (19.3%)	10 (8.1%)	0.014

Table 1. Clinical characteristics of patients and comparison according to NC/C ratios (Continued)

	Total	NC/C ratio < 2.3	NC/C ratio > 2.3	P-value	NC/C ratio < 2.57	NC/C ratio > 2.57	P-value
Heart rate (beat/min)	78 ± 15	79 ± 16	77 ± 15	0.56	78 ± 15	78 ± 15	0.88
Family History of heart failure n (%)	30 (11.5%)	8 (7.5%)	22 (14.2%)	0.11	9 (7.1%)	21 (15.4%)	0.037
ACEi or ARB n (%)	196 (76.3%)	85 (81%)	111 (73%)	0.17	96 (77.4%)	100 (75.2%)	0.76
Beta blocker n (%)	230 (89.1%)	98 (92.5%)	132 (86.8%)	0.22	113 (90.4%)	117 (88.0%)	0.5
Aldosterone antagonist n (%)	171 (65.3%)	73 (68.3%)	98 (63.2%)	0.43	84 (66.7%)	87 (64.0%)	0.69
Loop diuretic n (%)	147 (56.1%)	64 (59.0%)	83 (53.5%)	0.37	74 (58.7%)	73 (53.7%)	0.45
Anticoagulant n (%)	48 (18.3%)	25 (23.4%)	23 (14.8%)	0.10	29 (23.0%)	19 (14.0%)	0.078
Antiplatelet n (%)	83 (31.7%)	29 (27.1%)	54 (34.8%)	0.22	34 (27.0%)	49 (36.0%)	0.14
If-channel blocker n (%)	44 (16.8%)	17 (15.9%)	17 (17.4%)	0.86	18 (14.3%)	26 (19.1%)	0.32
ICD n (%)	60 (23.2%)	25 (23.6%)	35 (22.9%)	1.0	27 (21.6%)	33 (24.6%)	0.65

Values are mean ±SD or n (%) p value<0.05. Comparison of the patients according to non-compacted/compacted myocardial segment ratio. Firstly patients compared to Petersen criteria. NC/C ratio 2.3 and redefined NC/C ratio 2.57. ACEi, Angiotensinogen converting enzyme inhibitor; ARB, Angiotensin receptor blocker; AF, Atrial fibrillation; BMI, Body mass index; BSA, Body surface area; COPD, Chronic obstructive pulmonary disease and ICD, Implantable cardioverter-defibrillator.

Table 2. Echocardiographic parameters and comparison according to NC/C ratios

	Total	NC/C ratio < 2.3	NC/C ratio > 2.3	P-value	NC/C ratio < 2.57	NC/C ratio > 2.57	P-value
LVEDd (mm)	61.4 ± 9.3	60.7 ± 9.3	61.9 ± 9.2	0.36	60.1 ± 9.3	62.6 ± 9.1	0.045
LVESd (mm)	50.6 ± 11.7	49.8 ± 11.7	51.2 ± 11.8	0.37	49.3 ± 11.6	52 ± 11.7	0.08
LAd (mm)	43.5 ± 7.0	43.1 ± 6.1	43.8 ± 7.5	0.42	43 ± 6.7	44.1 ± 7.2	0.23
LVEF (%)	31.1 ± 11.3	31.7 ± 11.1	30.6 ± 11.4	0.45	32.5 ± 11.2	29.7 ± 11.2	0.06
Moderate or severe mitral regurgitation n (%)	94 (39.5%)	36 (35.6%)	58 (42.3%)	0.20	40 (34.2%)	54 (44.6%)	0.13
Moderate or severe aortic regurgitation n (%)	10 (4.2%)	3 (2.9%)	7 (5.1%)	0.66	3 (2.6%)	7 (5.8%)	0.43
Moderate or severe tricuspid regurgitation n (%)	49 (20.6%)	17 (16.8%)	32 (23.3%)	0.46	21 (17.9%)	28 (23.1%)	0.51
TAPSE (mm)	18.3±4.9	17.8±4.9	18.6±4.9	0.28	17.9±4.9	18.6±4.9	0.35
TAPSE<16 mm (RV dysfunction)	59 (29.1%)	30 (34.1%)	29 (25.3%)	0.21	34 (33.7%)	25 (24.5%)	0.16
RVsm (TDI) m/sec	11.1 ± 3.69	11.3 ± 4.4	10.9 ± 2.99	0.46	11.2 ± 4.23	10.9 ± 3.0	0.46

Values are mean ±SD or n (%) p value<0.05. Comparison of the patients according to non-compacted/compacted myocardial segment ratio. Firstly patients compared to Petersen criteria. NC/C ratio 2.3 and redefined NC/C ratio 2.57. LVEDd, Left ventricular end diastolic diameter; LVESd, Left ventricular end systolic diameter; LAd, Left atrial diameter; LVEF, Left ventricular systolic ejection fraction; TAPSE, Tricuspid annuler plane systolic excursion; RVsm, Right ventricular systolic motion tissue Doppler imaging

Table 3. Cardiac magnetic resonance imaging parameters and comparison according to NC/C ratios

	Total	NC/C ratio <2.3	NC/C ratio > 2.3	P-value	NC/C ratio < 2.57	NC/C ratio > 2.57	P-value
C length (mm)	5.3 ± 1.5	6.48 ± 1.62	4.59 ± 0.88	-	6.26 ± 1.62	4.55 ± 0.90	-
NC length (mm)	12.2 ± 3.3	9.59 ± 2.13	14.0 ± 2.67	-	9.92 ± 2.21	14.34 ± 2.67	-
NC/C ratio	2.47 ± 0.94	1.53 ± 0.35	3.12 ± 0.65	-	1.67 ± 0.46	3.21 ± 0.61	-
Average wall thickness (mm)	9.4 ± 2.0	9.7 ± 1.8	9.13 ± 2.1	0.19	9.7 ± 1.9	9.0 ± 2.1	0.019
Presence RV trabeculations n (%)	74 (28.7%)	16 (15.2%)	58 (37.9%)	<0.0001	18 (14.9%)	53 (40.2%)	<0.0001
LVEDd (mm)	63.2 ± 10.5	62.7 ± 10.0	63.6 ± 10.9	0.49	62.3 ± 9.8	64.1 ± 11.1	0.17
LVESd (mm)	54.1 ± 12.3	53.7 ± 11.3	54.4 ± 12.9	0.64	53.1 ± 11.3	55.0 ± 13.1	0.24
LAd (mm)	47.6 ± 9.3	46.4 ± 8.5	48.5 ± 9.8	0.064	46.6 ± 8.6	48.6 ± 9.9	0.14
LVEDV(i) (ml/m^2)	128.7 ± 49.8	116.8 ± 44.6	137.2 ± 51.6	0.002	116.2 ± 43.7	140.5 ± 53.2	<0.0001
LVESV(i) (ml/m^2)	93.2 ± 47.1	85.9 ± 42.7	98.4 ± 49.5	0.049	84.1 ± 42.0	101.8 ± 50.0	0.005
LV Mass(i) (g/m^2)	78.8 ± 25.6	79.5 ± 23.6	78.2 ± 27	0.71	77.9 ± 23.2	79.7 ± 27.7	0.66
LVEF (%)	29.5 ± 11.3	28.6 ± 10.3	30.1 ± 12.0	0.26	29.8 ± 10.7	29.2 ± 11.9	0.87
Cardiac Output (L/min)	5.34 ± 1.98	4.96 ± 1.83	5.61 ± 2.03	0.010	5.07 ± 1.76	5.60 ± 2.15	0.033
Stroke Volume (ml)	70.3 ± 27.7	65.1 ± 25.1	73.9 ± 28.8	0.013	66.8 ± 24.8	73.5 ± 30.0	0.053
Cardiac Index (L/min/m^2)	2.64 ± 0.92	2.37 ± 0.72	2.85 ± 1.0	<0.0001	2.44 ± 0.74	2.84 ± 1.03	0.001
PET (msec)	167.6 ± 67.4	161.3 ± 34.7	173 ± 86.2	0.25	166.7 ± 61.8	168.6 ± 74.0	0.84

Table 3. Cardiac magnetic resonance imaging parameters and comparison according to NC/C ratios (Continued)

	Total	NC/C ratio <2.3	NC/C ratio > 2.3	P-value	NC/C ratio < 2.57	NC/C ratio > 2.57	P-value
PFT (msec)	516.1 ± 154.5	535.9 ± 168.3	498.9 ± 140.2	0.14	535.3 ± 164.3	492.9 ± 139.5	0.12
LGE distribution n (%) = 102 (40.9%)							
Sub-endocardial LGEn (%)	21 (8.4%)	8 (7.8%)	13 (8.9%)	<0.0001	8 (6.7%)	13 (10.1%)	<0.0001
Mid-wall LGE n (%)	31 (12.4%)	24 (23.3%)	7 (4.8%)	<0.0001	26 (21.7%)	5 (3.9%)	<0.0001
Sub-epicardial LGE n (%)	19 (7.6%)	1 (1.0%)	18 (12.3%)	<0.0001	3 (2.5%)	16 (12.4%)	<0.0001
Focal insertion LGE n (%)	31 (12.4%)	16 (15.5%)	15 (10.3%)	<0.0001	18 (15.0%)	13 (10.1%)	<0.0001

249 of the 262 patients' CMR images was convenient for the LGE analysis. Of all the 249 patients, 102 (40.9%) patients demonstrated any pattern of LGE. The mean age of patients with LGE was 45.1±13, and by a majority, 75 (73.5%) were men. LGE distribution was classified as sub-endocardial in 21 (20.6%) patients, mid-wall in 31 (30.4%) patients, sub-epicardial in 19 (18.6%) patients, and focal insertion in 31 (30.4%) patients. Mid-wall LGE pattern was primarily seen in the DCM patients, 24 of 31 (77%), and midwall LGE had an estimated hazard ratio of 0.88 (95%CI: [0.34-2.24]) compared to non-LGE patients. In contrast to mid-wall LGE distribution, a sub-epicardial LGE pattern was the main finding in 17 (94.4%) LVNC patients. Estimated and adjusted HR for patients with subepicardial LGE was 1.69 (95%CI: [0.60-4.80]), compared to non-LGE patients. Values are mean ±SD or n (%) p value<0.05. Comparison of the patients according to non-compacted/compacted myocardial segment ratio. Firstly patients compared to Petersen criteria. NC/C ratio 2.3 and redefined NC/C ratio 2.57. C=compacted myocardium; NC= non-compacted myocardium; NC/C ratio= ratio of the non-compacted segment to compacted segment of the myocardium; LVEDd=Left ventricular end diastolic diameter; LVESd= Left ventricular end systolic diamater; LAd= Left atrial diameter; LVEF= Left ventricular ejection fraction; LVEDV(i)= Left ventricular end diastolic volume index; LVESV(i)= Left ventricular end systolic volume index; LV Mass(i)= Left ventricular mass index; PET= Peak ejection time; PFT= Peak filling time and LGE= Late gadolinium enhancement

Table 4. Endpoints and comparison according to NC/C ratios

	Total	NC/C ratio < 2.3	NC/C ratio > 2.3	P-value	NC/C ratio < 2.57	NC/C ratio > 2.57	P-value
Death n (%)	29 (11.1%)	11 (10.3%)	18 (11.6%)	0.84	12 (9.8%)	17 (12.5%)	0.55
LVAD n (%)	20 (7.6%)	6 (5.6%)	14 (9.0%)	0.35	6 (4.8%)	14 (10.3%)	0.1
Heart Transplantation n (%)	6 (2.3%)	0	6 (3.9%)	0.08	0	6 (4.5%)	0.03
Hospitalization n (%)	14 (5.3%)	6 (5.6%)	8 (5.2%)	1.0	7 (5.6%)	7 (5.1%)	1.0
Ventricular arrhythmia n (%)	10 (3.8%)	4 (3.7%)	6 (3.9%)	1.0	5 (4.0%)	5 (3.7%)	1.0
Cerebrovascular event n (%)	4 (1.5%)	2 (1.9%)	2 (1.3%)	1.0	3 (2.4%)	1 (0.7%)	0.35
Primary endpoint n (%)	55 (21%)	17 (15.9%)	38 (24.5%)	0.12	18 (14.3%)	37 (27.2%)	0.015
Follow uptime days (median-IQRs)	936 (422-1827)	933 (449-1667)	940 (430-1976)	0.95	946 (477-1687)	888 (397-1984)	0.60

Values are mean ±SD or n (%) p value<0.05. Comparison of the patients according to non-compacted/compacted myocardial segment ratio. Firstly patients compared to Petersen criteria. NC/C ratio criterion 2.3 and redefined NC/C criterion ratio 2.57. LVAD= Left ventricular assisted device; IQR= Interquartile range

Heart failure

OP-026

Hypokinetic non-dilated non-ischemic cardiomyopathy patient clinical characteristics, survival, and comparison of the dilated cardiomyopathy patients Ege University Cardiomyopathy Observational Research (EUCOR)-Hypokinetic non-Dilated Cardiomyopathy

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Background and Aim: Hypokinetic non-dilated non-ischemic cardiomyopathy is defined as a new phenotype of global systolic dysfunction with non-dilated ventricles. There is limited data on this non-dilated non-ischemic cardiomyopathy patient's characteristics and prognosis. This study aimed to evaluate the exhibit unknowns of the non-dilated non-ischemic cardiomyopathy patients.

Methods: Ege University Cardiomyopathy Observational Research Study data used for analysis. This retrospective study was designed between 2008-2021 and included patients with a left ventricle ejection fraction lower than 50%. Non-dilated patients are designated according to the American Society of Echocardiography cardiac chamber quantification recommendations. Left ventricle dilation is accepted for women with left ventricle end-diastolic dimensions over 52.2 mm and 58.4 mm for men. Ischemic, hypertrophic, and restrictive cardiomyopathy patients were excluded from the study. Primary end-points were composite of heart replacement therapies or all-cause mortality.

Results: 666 patients were included in the study, 142 patients were in the group of non-dilated non-ischemic cardiomyopathy, the mean age was 41.3 ± 13.9, and 64.8% were male. For comparison 524 dilated non-ischemic cardiomyopathy patients' data were analyzed simultaneously. Patients with hypokinetic non-dilated cardiomyopathy patients were younger, had more family history of heart failure, had lower usage of mineralocorticoid receptor antagonists, anticoagulants, and loop diuretics, and had lower intracardiac device implantation Table 1. In echocardiographic assessment, HnDCM had a lower left ventricular ejection fraction and a lower rate of moderate or severe mitral or tricuspid regurgitation. Table 2. Primary end-point free survival analyses revealed that HnDCM cardiomyopathy patients had better survival than DCM patients (HR:1,64; 95% CI: 1.25-2.14; p=0.0003). In cox regression, multivariate analyses of HnDCM patients with severe tricuspid regurgitation and the one-millimeter decrease of TAPSE were the worse predictors of primary end-points.

DCM patient's worse survival predictors were loop diuretic usage, and every decrease in LVEF, nonetheless, MRA usage was protective for the primary end-point (*Tables 3-6)*.

Conclusions: This study questioned the HnDCM patient's characteristics and the survival uncertainties. This study's salient findings were that HnDCM patients had more family history. As opposed to DCM patients LVEF did not influence prognosis. HnDCM worse survival predictors revealed right ventricle function and tricuspid valve had a major impact on prognosis.

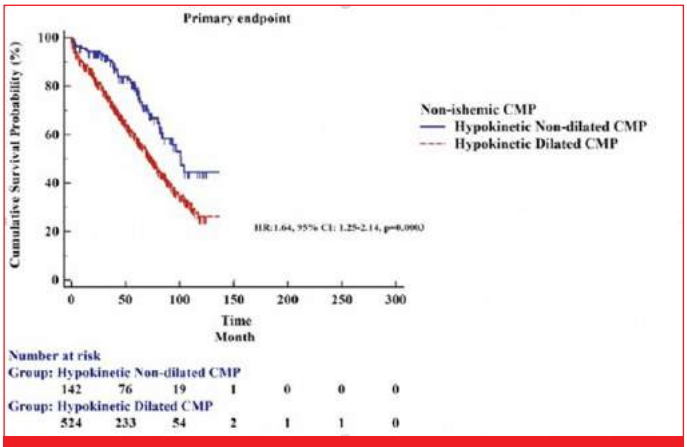

Figure 1. Time-to-Event Curves for all-cause of mortality, heart transplantation, and mechanical circulatory support

Table 1. Characteristics of non-dilated and dilated non-ischemic cardiomyopathy patient's

	Total (N:666)	HnDCM (N:142) (16.5%)	DCM (N:524) (61.0%)	P-value
Age years	62.6±12.1	61.3±13.9	62.9±12.0	0.018
Male %	348 (67.0%)	90 (63.8%)	357 (68.1%)	0.36
BSH kg/m²	28.2±5.7	26.6±6.0	28.6±5.1	0.013
BSA m²	1.88±0.26	1.81±0.19	1.90±0.24	0.001
Arterial hypertension n (%)	169 (32.6%)	30 (22.5%)	172 (32.8%)	1.0
Diabetes mellitus n (%)	176 (30.0%)	28 (19.7%)	188 (34.3%)	0.99
Dyslipidemia n (%)	58 (8.0%)	11 (7.8%)	47 (8.1%)	1.0
Smoking history n (%)	368 (80.2%)	66 (52.9%)	232 (63.8%)	0.034
Cerebrovascular event n (%)	5 (0.8%)	1 (0.1%)	4 (0.8%)	1.0
COPD n (%)	28 (4.3%)	8 (5.7%)	20 (3.9%)	0.40
Renal Disease n (%)	70 (10.0%)	3 (2.1%)	67 (13.3%)	0.39
AF n (%)	130 (19.5%)	30 (21.6%)	96 (19.0%)	0.025
Device				<0.001
Pacemaker n (%)	5 (0.8%)	2 (1.4%)	3 (0.6%)	-
ICD n (%)	232 (34.9%)	32 (22.5%)	200 (38.4%)	-
CRT-D n (%)	37 (5.6%)	1 (0.9%)	36 (7.1%)	-
Family History of heart failure n (%)	65 (9.8%)	11 (7.7%)	47 (9.0%)	0.30
ACEI or ARB n (%)	515 (77.6%)	109 (76.8%)	406 (77.3%)	0.82
Beta-blocker n (%)	519 (82.3%)	119 (83.8%)	400 (81.1%)	0.31
Aldosterone antagonist n (%)	305 (35.9%)	48 (41.0%)	417 (55.8%)	<0.001
Loop diuretic n (%)	468 (70.3%)	75 (52.8%)	393 (75%)	<0.001
Anticoagulant n (%)	167 (7.3%)	25 (17.6%)	132 (25.0%)	0.003
Antiplatelet n (%)	288 (43.2%)	57 (40.1%)	231 (44.1%)	0.69
If channel blocker n (%)	135 (20.2%)	21 (14.2%)	114 (21.8%)	0.23
Primary end-point n (%)	289 (43.4%)			
Follow-up time median IQR (days)	1390 (-)	1659 (-)	1347 (-)	-

** Values are mean ±SD or n (%) p value<0.05. LVEF=Left ventriculer ejection fraction; ICD= Implanted cardioverter-defibrillator; CRT-D= Cardiac resynchronization therapy-Defibrillator ACEi=Angiotensinogen was converting enzyme inhibitor; ARB=Angiotensin receptor blocker; AF= Atrial fibrillation; BMI=body mass index; BSA: body surface area; COPD= Chronic obstructive pulmonary disease*

Table 2. Echocardiographic parameters of the patients with non-dilated and dilated non-ischemic cardiomyopathy

	Total (N:532)	HnDCM (N:142) (16.5%)	DCM (N:524) (61.0%)	P-value
LVEDd mm	64.0±9.8	51.6±5.3	67.4±7.9	<0.001
LVESd mm	54.3±11.6	39.5±7.5	58.3±9.1	0.013
LAd mm	45.6±7.9	41.2±7.9	46.8±7.4	0.28
LVEF %	26.7±8.3	32.9±9.6	22.1±7.0	<0.0001
Mitral Regurgitation n (%)				<0.0001
Mild	282 (42.3%)	21 (57.0%)	201 (38.4%)	-
Moderate	230 (34.5%)	31 (21.8%)	199 (38.0%)	-
Severe	86 (12.9%)	4 (2.8%)	82 (15.6%)	-
Aort Regurgitation n (%)				0.47
Mild	109 (16.4%)	21 (14.8%)	88 (16.8%)	-
Moderate	15 (2.3%)	4 (2.8%)	11 (2.1%)	-
Severe	7 (1.1%)	0	7 (1.3%)	-
Tricuspid Regurgitation n (%)				0.002
Mild	330 (49.5%)	64 (45.1%)	266 (50.8%)	-
Moderate	130 (19.5%)	17 (12.0%)	113 (21.6%)	-
Severe	54 (8.1%)	15 (10.6%)	39 (7.4%)	-
TAPSE mm	17.6±5.5	17.5±5.5	17.6±5.4	0.42
RVsm (TDI) m/sec	10±3	10.2±2.9	10.8±3.3	0.90
TRV m/sec	2.87±1.01	2.73±1.0	2.91±1.0	0.71
SPAP mmHg	43.7±13.1	39.3±11.8	44.6±13.2	0.04

Values are mean ±SD or n (%) p value<0.05. LVEDd= Left ventricular end diastolic diameter; LVESd= Left ventricular end systolic diameter; LAd= Left atrial diameter; LVEF= Left ventricular systolic ejection fraction; TAPSE= Tricuspid annuler plane systolic excursion; RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure

Table 3. Cox univariate and multivariate analysis of clinical parameters for non-dilated non-ischemic cardiomyopathy patients

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	P-value	HR (95% CI)	P-value
Age years	0.986(0.964-1.009)	0.24	-	-
Gender	0.628 (0.314-1.258)	0.19	-	-
Arterial hypertension	0.611 (0.256-1.461)	0.61	-	-
Diabetes mellitus	0.458 (0.191-1.095)	0.079	-	-
Dyslipidemia	0.50 (0.120-2.076)	0.34	-	-
Smoking history	1.045 (0.553-1.974)	0.89	-	-
COPD	0.81 (0.196-3.374)	0.77	-	-
Renal Disease	-	-	-	-
Atrial fibrillation	1.356 (0.682-2.696)	0.38	-	-
Device		0.18	-	-
Pacemaker	2.974 (0.691-12.80)	0.14	-	-
ICD	1.827 (0.944-3.525)	0.074	-	-
CRT-D	0.876 (0.193-3.982)	0.86	-	-
Family History	1.388 (0.579-3.325)	0.46	-	-
ACEI or ARB	0.954 (0.465-1.961)	0.89	-	-
Beta-blocker	2.86 (0.393-20.8)	0.29	-	-
Aldosterone antagonist	1.973 (0.903-4.307)	0.08	-	-
Loop diuretic	2.019 (0.981-4.156)	0.057	-	-
Anticoagulant	0.933 (0.467-1.963)	0.80	-	-

Cox univariate and multivariate model, P value<0.05. HR= Hazard ratio; CI= Confidence interval ACEi=Angiotensinogen is converting enzyme inhibitor; ARB=Angiotensin receptor blocker; AF= Atrial fibrillation; COPD= Chronic obstructive pulmonary disease.

Table 4. Cox univariate and multivariate analysis of echocardiographic parameters for non-dilated non-ischemic cardiomyopathy patients

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	P-value	HR (95% CI)	P-value
LVEDd mm	1.035 (1.013-1.058)	0.002	-	-
LVESd mm	1.046 (1.024-1.068)	<0.001	-	-
LAd mm	1.054 (1.014-1.095)	0.008	-	-
LVEF %	0.966 (0.932-1.001)	0.06		
Mitral Regurgitation		0.031	-	-
Mild	1.142 (0.387-3.37)	0.81	-	-
Moderate	2.832 (0.939-8.547)	0.065	-	-
Severe	3.598 (0.653-19.76)	0.14	-	-
Aortic Regurgitation		0.41	-	-
Mild	1.699 (0.780-3.702)	0.18	-	-
Moderate	1.072 (0.146-7.891)	0.94	-	-
Severe	-	-	-	-
Tricuspid Regurgitation		<0.001		-
Mild	1.283 (0.960-3.704)	0.57	-	-
Moderate	1.735 (0.619-4.862)	0.29	-	-
Severe	9.008 (3.575-22.693)	<0.001	9.476 (2.701-33.249)	<0.0001
TAPSE mm	0.914 (0.85-0.982)	0.014	0.914 (0.836-0.999)	0.049
RVsm (TDI) m/sec	0.781 (0.668-0.918)	0.003	-	-
TRV m/sec	1.323 (0.858-2.038)	0.20	-	-
SPAP mmHg	1.017 (0.982-1.053)	0.34	-	-

Values are mean ±SD or n (%) p value<0.05. LVEDd= Left ventricular end diastolic diameter; LVESd= Left ventricular end systolic diameter; LAd= Left atrial diameter; LVEF= Left ventricular systolic ejection fraction; TAPSE= Tricuspid annuler plane systolic excursion; RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure

Table 5. Cox univariate and multivariate analysis of clinical parameters of dilated non-ischemic cardiomyopathy patients

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	P-value	HR (95% CI)	P-value
Age years	1.008 (0.998-1.019)	0.12	-	-
Gender (female)	0.825 (0.630-1.081)	0.16	-	-
Arterial hypertension	0.825 (0.61-1.117)	0.21	-	-
Diabetes mellitus	0.790 (0.577-1.081)	0.14	-	-
Dyslipidemia	1.095 (0.707-1.698)	0.68	-	-
Smoking history	1.379 (1.070-1.778)	0.013	-	-
COPD	1.714 (0.998-2.943)	0.051	-	-
Renal Disease	0.41 (0.131-1.281)	0.41	-	-
AF	1.114 (0.815-1.523)	0.49	-	-
Device		0.049	-	-
Pacemaker	1.556 (0.491-4.927)	0.45	-	-
ICD	1.440 (1.087-1.908)	0.011	-	-
CRT-D	1.475 (1.029-2.113)	0.034	-	-
Family History	0.923 (0.589-1.445)	0.72	-	-
ACEI or ARB	0.722 (0.537-0.970)	0.031	-	-
Beta blocker	0.412 (0.273-0.622)	<0.0001	-	-
Aldosterone antagonist	0.809 (0.582-1.124)	0.20	0.618 (0.405-0.943)	0.026
Loop diuretic	3.076 (2.049-4.619)	<0.0001	2.518 (1.472-4.308)	0.001
Anticoagulant	0.921 (0.700-1.211)	0.89	-	-
Antiplatelet	1.953 (0.272-14.0)	0.50	-	-
If channel blocker	0.787 (0.574-1.080)	0.13	-	-

Cox univariate and multivariate model, P value<0.05. HR= Hazard ratio; CI= Confidence interval ACEi=Angiotensinogen is converting enzyme inhibitor; ARB=Angiotensin receptor blocker; AF= Atrial fibrillation; COPD= Chronic obstructive pulmonary disease and; ICD= Implanted cardioverter-defibrillator

Table 6. Cox univariate and multivariate analysis of echocardiographic parameters dilated non-ischemic cardiomyopathy

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	P-value	HR (95% CI)	P-value
LVEDd mm	1.027 (1.006-1.048)	0.01	-	-
LVESd mm	1.034 (1.014-1.053)	0.001	-	-
LAd mm	1.052 (1.036-1.068)	<0.0001	-	-
LVEF %	0.918 (0.896-0.940)	<0.0001	0.927 (0.894-0.962)	<0.0001
Mitral Regurgitation		0.0001	-	-
Mild	0.77 (0.409-1.478)	0.44	-	-
Moderate	1.575 (0.918-2.704)	0.81	-	-
Severe	2.090 (1.177-3.709)	0.012	-	-
Aort Regurgitation		0.030	-	-
Mild	1.238 (0.899-1.704)	0.19	-	-
Moderate	1.052 (0.466-2.373)	0.90	-	-
Severe	3.561 (1.455-8.714)	0.005	-	-
Tricuspid Regurgitation		<0.0001	-	-
Mild	1.417 (0.958-2.095)	0.081	-	-
Moderate	2.854 (1.893-4.304)	<0.0001	-	-
Severe	3.222 (1.950-5.324)	<0.0001	-	-
TAPSE mm	0.933 (0.910-0.958)	<0.0001	-	-
RVsm (TDI) m/sec	0.844 (0.800-0.891)	<0.0001	-	-
TRV m/sec	0.990 (0.880-1.139)	0.99	-	-
SPAP mmHg	1.022 (1.011-1.033)	<0.0001	-	-

LVEDd= Left ventricular end diastolic diameter; LVESd= Left ventricular end systolic diameter; LAd= Left atrial diameter; LVEF= Left ventricular systolic ejection fraction; TAPSE= Tricuspid annuler plane systolic excursion; RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure; ICD= Implanted cardioverter-defibrillator

Heart failure

OP-027

Suboptimal guideline adherence is associated with higher MortalitY and hospitalization Rates in patieNts with heArt failure and reduced ejection fraction: The SMYRNA Study

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Background and Aim: Despite recommendations of heart failure guidelines, most patients with heart failure and reduced ejection fraction (HFrEF) do not receive guideline-directed medical therapy (GDMT). The aim of the present study was to assess the status of GDMT in patients with HFrEF and to evaluate its association with clinical outcomes in real-life setting.

Methods: The SMYRNA study is a multicenter, prospective, and observational study that included outpatients with chronic HFrEF. The status of GDMT at the time of first visit (prescription of renin–angiotensin system [RAS] inhibitors, beta-blockers, and mineralocorticoid receptor antagonists [MRAs]) and its association with all-cause mortality during follow-up period were investigated.

Results: Overall, 1,062 patients from 14 cardiology outpatient clinics were included. Mean age was 66 ± 11 years, 30.9% were female, and mean ejection fraction was 31 ± 6%. Among study population, 24%, 10.5%, and 44.9% were not prescribed RAS inhibitors, beta-blockers, and MRAs, respectively. The percentage of patients who could not be able to use one or more class of heart failure (HF) medications because of side effects, symptoms, and/or non-cardiac organ dysfunction was 22.4% (n= 238). Patients were divided into 3 groups: those treated with 3 class of HF medications (n= 491, 46.2%), 2 class of HF medications (n= 353, 33.2%), and ≤1 class of HF medica-

tions (n= 218, 20.6%). Median follow up was 24 months. Overall all-cause mortality and HHF rates were 16.7% and 32.3%, respectively. Patients receiving ≤1 class of HF medications had an increased risk of death (HR 1.47; 95% CI: 1.06–2.06) compared to those patients receiving 2 or 3 class of HF medications.

Conclusions: Patients with HFrEF who were received ≤1 class of HF medications seemed to have a greater risk of death and/or HHF compared with patients received 2 or 3 class of HF medications.

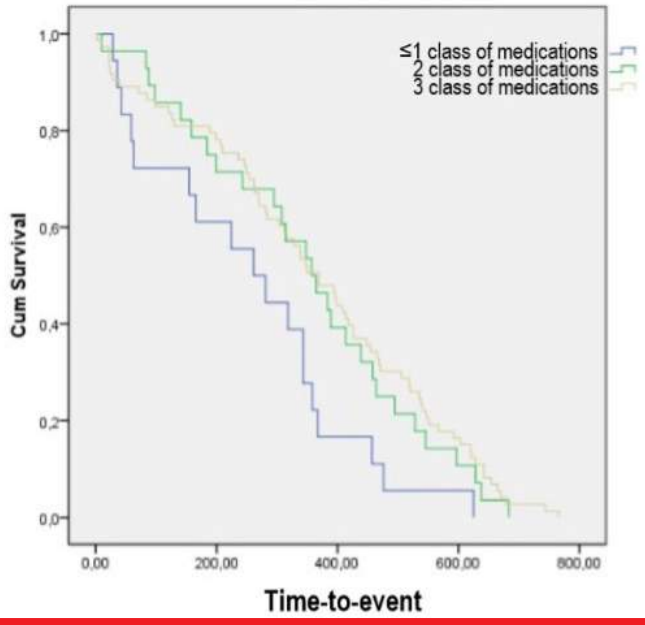

Figure 1. Kaplan-Meier survival analysis according to the number of heart failure medications

Heart failure

OP-028

EUCOR-DEVICE Trial; Non-ischemic cardiomyopathy patient's survival results of the intracardiac device implanted versus medical therapy (Ege University Cardiomyopathy Observational Research Device Trial)

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Background and Aim: It has been revealed the benefit of ICD implantation for primary prevention, particularly ischemic etiology, however, the benefit in those with non-ischemic etiology is unclear. The long-term results of SCD-HeFT and the DANISH ICD the study revealed the benefit of ICD, is questioned in these patients. This long-term retrospective study aimed to evaluate, the benefit of ICD, and CRT-D implantation in patients with non-ischemic systolic heart failure and the predictors of prognosis in each group.

Methods: 532 non-ischemic cardiomyopathy patients between 2008-2021 left ventricle ejection lower than 35% included in the study. Patients classified as medical treatment implanted cardiac device and cardiac resynchronization therapy with a defibrillator. Primary end-points were challenging and consisted of heart replacement therapy or the all-cause mortality. In this study, we questioned the implanted cardiac devices benefit on primary end-points.

Results: Patient's mean age was 42.6 ± 12 years, 30% were female and the mean LVEF was 23 ± 4.

CRT-D implanted patients were older, more using MRA and loop diuretic, and had lower LVEF. Medical treatment without, intracardiac implanted devices patients had less atrial fibrillation (Tables 1, 2).

The primary endpoints have occurred in 264 (49.6%) patients at a median follow-up of 3.8 years.

Kaplan Meier's survival analysis showed that intracardiac device patients had better survival in a short time though, loses the survival advantage in a long time medical treatment patients survival was better (Log-rank Mantel cox test p value=0.024) (Figure 1). Cox regression univariate analysis results revealed, (HR; 1.4; CI 95%; 1.07-1.84; p=0.01, HR; 1.4; CI 95%, 1.01-2.03; p=0.04; respectively), optimal medical therapy without intracardiac device implantation had better survival. Cox regression multivariate analysis demonstrated in the ICD group, that loop diuretic needed patients, lower LVEF, and the severe tricuspid regurgitation were worse prognosis predictors, and in CRT-D implanted patients, ACEI or ARB usage and RVsm (TDI) increase had a better prognosis (Tables 5-8).

Conclusions: In this study primary prophylaxis ICD in patients with non-ischemic cardiomyopathy LVEF ≤35% did not outperform medical treatment regardless of age, comorbidity, and functional capacity. The findings of support the DANISH study. Another important point is; CRT-D implantation is not superior to medical treatment in this study for a long time. Associated factors may be compliance with medical treatment, lower EF, and a lower number of patients in CRT-D patients. More studies are needed on this subject. In conclusion, ICD or CRT-D implantation for primary prophylaxis does not appear to be superior to medical therapy in patients with non-ischemic cardiomyopathy with LVEF ≤35%.

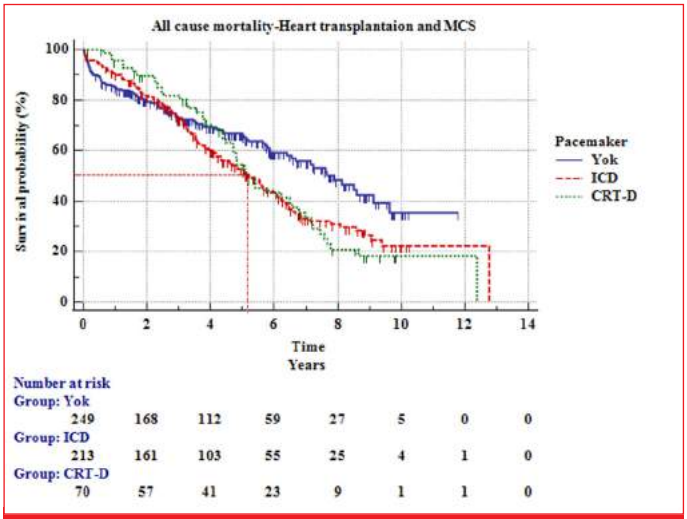

Figure 1. Time-to-event curves for all-cause mortality, heart transplantation, and mechanical circulatory support

Table 1. Characteristics of the patients with non-ischemic cardiomyopathy

	Total (N:532)	Device (-) (N:249) (46.8%)	ICD (N:213) (40.0%)	CRT-D (N:70) (13.1%)	P-value
Age years	42.6±12	42.6±13	41.5±12	46.3±11	0.02
Male %	374 (70%)	168 (67.5%)	160 (75.1%)	46 (65.7%)	0.13
BMI kg/m²	26±4.5	26.5±4.7	25.6±4.2	25.4±4.7	0.23
BSA m²	1.8±0.2	1.8±0.2	1.8±0.2	1.8±0.2	0.80
Heart Failure Etiology n (%)					0.17
Dilated Cardiomyopathy n (%)	436 (82%)	192 (77.1%)	183 (85.9%)	61 (87.1%)	
Noncompaction Cardiomyopathy n (%)	85 (16%)	49 (19.7%)	28 (13.1%)	8 (11.4%)	
Peripartum Cardiomyopathy n (%)	7 (1.3%)	3 (1.2%)	1 (0.5%)	-	
Arrhythmogenic Cardiomyopathy n (%)	4 (0.8%)	5 (2%)	1 (0.5%)	1 (1.4%)	
Arterial hypertension n (%)	117 (22%)	53 (21.3%)	44 (20.7%)	20 (29%)	0.32
Diabetes mellitus n (%)	110 (20.7%)	52 (20.9%)	40 (18.8%)	18 (26.1%)	0.42
Dyslipidemia n (%)	40 (7.5%)	16 (6.4 %)	19 (8.9%)	5 (7.2%)	0.59
Smoking history n (%)	225 (42.6%)	101 (40.9%)	100 (47.2%)	24 (34.8%)	0.14
Cerebrovascular event n (%)	33 (6.2%)	11 (4.5%)	19 (8.9%)	3 (4.3%)	0,11
COPD n (%)	23 (4.4%)	9 (3.7%)	12 (5.7%)	2 (2.9%)	0.40
Renal Disease n (%)	16 (3%)	12 (4.8%)	3 (1.4%)	1 (1.4%)	0.07
AF n (%)	107 (20.9%)	47 (19.5%)	42 (22.1%)	15 (22.1%)	<0.0001

Table 1. Characteristics of the patients with non-ischemic cardiomyopathy (Continued)

	Total (N:532)	Device (-) (N:249) (46.8%)	ICD (N:213) (40.0%)	CRT-D (N:70) (13.1%)	P-value
Family History of heart failure n (%)	51 (9.6%)	22 (8.8%)	25 (11.7%)	4 (5.7%)	0.28
ACEi or ARB n (%)	412 (77.4%)	189 (75.9%)	167 (78.4%)	56 (80%)	0.70
Beta-blocker n (%)	496 (93.2%)	230 (92.4%)	200 (93.9%)	66 (94.3%)	0.75
Aldosterone antagonist n (%)	430 (80.8%)	186 (74.7%)	183 (85.9%)	61 (87.1%)	0.003
Loop diuretic n (%)	402 (75.6%)	175 (70.3%)	169 (79.3%)	58 (82.9%)	0.02
Anticoagulant n (%)	155 (29.1%)	71 (28.5%)	61 (28.6%)	23 (32.9%)	0.76
Antiplatelet n (%)	239 (44.9%)	105 (42.2%)	99 (46.5%)	35 (50%)	0.58
If channel blocker n (%)	109 (20.5%)	51 (20.5%)	41 (19.2%)	17 (24.3%)	0.66
Follow-up time median IQR (days)	1399 (669-2230)	1251 (558-2152)	1404 (778-2227)	1724 (914-3217)	-
Outcomes n %	264(49.6%)	95 (38.2%)	122 (57.3%)	47 (67.1%)	0.02

* Values are mean ±SD or n (%) p value<0.05.
ICD= Implanted cardioverter-defibrillator; CRT-D= Cardiac resynchronization therapy-Defibrillator; BMI=body mass index; BSA: Body surface area; AF= Atrial fibrillation; COPD = Chronic obstructive pulmonary disease; ACEi=Angiotensinogen was converting enzyme inhibitor; ARB=Angiotensin receptor blocker

Table 2. Echocardiographic parameters of the patients with non-ischemic cardiomyopathy

	Total (N:532)	Device (-) (N:249) (46.8%)	ICD (N:213) (40.0%)	CRT-D (N:70) (13.1%)	P-value
LVEDd mm	66±9	64±8	66±8	70±10	<0.0001
LVESd mm	57±10	55±10	58±9	60±11	0.001
LAd mm	46±7	46±7	46±7	48±7	0.20
LVEF %	23±4	24±4	22±4	22±4	0.007
Mitral Regurgitation n (%)					0.15
Mild	215 (40.4%)	108 (43.4%)	81 (38%)	26 (37.1%)	
Moderate	195 (36.7%)	87 (34.9%)	74 (34.7%)	34 (48.6%)	
Severe	80 (15%)	36 (14.5%)	36 (16.9%)	8 (11.4%)	
Aort Regurgitation n (%)					0.41
Mild	90 (16.9%)	42 (16.9%)	33 (15.5%)	15 (21.4%)	
Moderate	13 (2.4%)	6 (2.4%)	4 (1.9%)	3 (4.3%)	
Severe	6 (1.1%)	5 (2%)	1 (0.5%)	-	
Tricuspid Regurgitation n (%)					0.78
Mild	264 (49.6%)	121 (48.6%)	108 (50.7%)	35 (50%)	
Moderate	115 (21.6%)	53 (21.3%)	48 (22.5%)	14 (20%)	
Severe	46 (8.6%)	25 (10%)	13 (6.1%)	8 (11.4%)	
TAPSE mm	17±5	16±5	165±5	18±7	0.24
RVsm (TDI) m/sec	10±3	10±3	10±3	10±2	0.87
TRV m/sec	2.9±1	3.0±0.9	2.9±1.3	2.8±0.5	0.57
SPAP mmHg	44±13	45±13	44±12	43±12	0.58

Values are mean ±SD or n (%) p value<0.05.
ICD= Implanted cardioverter-defibrillator; CRT-D= Cardiac resynchronization therapy-Defibrillator; LVEDd= Left ventricular end diastolic diameter; LVESd= Left ventricular end systolic diameter; LAd= Left atrial diameter; LVEF= Left ventricular systolic ejection fraction; TAPSE= Tricuspid annuler plane systolic excursion; RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure

Table 3. Cox univariate and multivariate analysis of clinical parameters non-device group

	Univariate analysis Device (-) Group		Multivariate analysis Device (-) Group	
	HR (95% CI)	P-value	HR (95% CI)	P-value
Age years	1.009 (0.993-1.026)	0.28	-	-
Gender	1.025 (0.673-1.561)	0.90	-	-
Arterial hypertension	0.774 (0.467-1.281)	0.31	-	-
Diabetes mellitus	0.638 (0.380-1.072)	0.08	-	-
Dyslipidemia	1.440 (0.697-2.974)	0.32	-	-
Smoking history	1.168 (0.772-1.767)	0.46	-	-
COPD	1.645 (0.666-4.068)	0.28	-	-
Renal Disease	0.257 (0.036-1.848)	0.17	-	-
Atrial fibrillation	1.156 (0.702-1.904)	0.56	-	-
Family History	0.573 (0.250-1.316)	0.18	-	-
ACEI or ARB	0.78 (0.489-1.244)	0.29	-	-
Beta blocker	0.44 (0.234-0.836)	0.012	-	-
Aldosterone antagonist	0.779 (0.488-1.245)	0.29	-	-
Loop diuretic	1.767 (1068-2.921)	0.02	3.087 (1.495-6.377)	0.002
Anticoagulant	0.853 (0.540-1.347)	0.49	-	-
Antiplatelet	0.79 (0.521-1.199)	0.26	-	-
If channel blocker	0.797 (0.472-1.348)	0.39	-	-

Cox univariate and multivariate model, P value<0.05.
HR= Hazard ratio; CI= Confidence interval
COPD= Chronic obstructive pulmonary disease; ACEi=Angiotensinogen is converting enzyme inhibitor; ARB=Angiotensin receptor blocker

Table 4. Cox univariate and multivariate analysis of echocardiographic parameters non- device group

	Univariate analysis Device (-) Group		Multivariate analysis Device (-) Group	
	HR (95% CI)	P-value	HR (95% CI)	P-value
LVEDd	1.035 (1.013-1.058)	0.002	-	-
LVESd	1.046 (1.024-1.068)	<0.001	-	-
LAd	1.055 (1.033-1.077)	<0.001	-	-
LVEF	0.891 (0.851-0.931)	<0.001	0.89 (0.84-0.94)	0.004
Mitral Regurgitation		0.002	-	-
Mild	2.721 (0.651-11.377)	0.17	-	-
Moderate	5.356 (1.296-22.134)	0.02	-	-
Severe	6.293 (1.462-27.081)	0.01	-	-
Aortic Regurgitation		0.02	-	-
Mild	1.363 (0.822-2.226)	0.23	-	-
Moderate	0.536 (0.074-3.868)	0.53	-	-
Severe	4.221 (1.526-11.679)	0.006	-	-
Tricuspid Regurgitation		<0.001	-	-
Mild	1.890 (0.965-3.704)	0.064	-	-
Moderate	3.113 (1.536-6.309)	0.002	-	-
Severe	5.875 (2.715-12.713)	<0.001	-	-
TAPSE mm	0.909 (0.869-0.950)	<0.001	-	-
RVsm (TDI) m/sec	0.844 (0.772-0.923)	<0.001	-	-
TRV m/sec	1.030 (0.736-1.440)	0.86	-	-
SPAP mmHg	1.014 (0.997-1.032)	0.10	-	-

Values are mean ±SD or n (%) p value<0.05.
LVEDd= Left ventricular end diastolic diameter; LVESd= Left ventricular end systolic diameter; LAd= Left atrial diameter; LVEF= Left ventricular systolic ejection fraction; TAPSE= Tricuspid annuler plane systolic excursion; RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure

Table 5. Cox univariate and multivariate analysis of clinical parameters ICD group

	Univariate analysis ICD Group		Multivariate analysis ICD-Group	
	HR (95% CI)	P-value	HR (95% CI)	P-value
Age years	1.000 (0.986-1015)	0.97	-	-
Gender	1.151 (0.757-1.752)	0.51	-	-
Arterial hypertension	0.711 (0.440-1.150)	0.16	-	-
Diabetes mellitus	0.913 (0.579-1.439)	0.69	-	-
Dyslipidemia	0.678 (0.354-1.298)	0.24	-	-
Smoking history	1.198 (0.838-1.714)	0.32	-	-
COPD	1.332 (0.674-2.631)	0.40	-	-
Renal Disease	0.495 (0.069-3.551)	0.48	-	-
AF	1.068 (0.069-1.639)	0.76	-	-
Family History	1.204 (0.720-2.011)	0.47	-	-
ACEI or ARB	0.93 (0.596-1.461)	0.76	-	-
Beta blocker	0.522 (0.272-1.002)	0.051	-	-
Aldosterone antagonist	0.85 (0.495-1.468)	0.56	-	-
Loop diuretic	3.674 (1.922-7.024)	<0.001	3.087 (1.495-6.377)	0.002
Anticoagulant	1.046 (0.707-1.546)	0.82	-	-
Antiplatelet	0.98 (0.692-1.413)	0.95	-	-
If channel blocker	0.797 (0.501-1.348)	0.33	-	-

Cox univariate and multivariate model, P value<0.05.
HR= Hazard ratio; CI= Confidence interval
ICD= Implanted cardioverter-defibrillator; COPD = Chronic obstructive pulmonary disease; AF= Atrial fibrillation; ACEi=Angiotensinogen was converting enzyme inhibitor; ARB=Angiotensin receptor blocker

Table 6. Cox univariate and multivariate analysis of echocardiographic parameters ICD Group

	Univariate analysis ICD Group		Multivariate analysis ICD- Group	
	HR (95% CI)	P-value	HR (95% CI)	P-value
LVEDd	1.027 (1.006-1.048)	0.01	-	-
LVESd	1.034 (1.014-1.053)	0.001	-	-
LAd	1.049 (1.025-1.073)	<0.001	-	-
LVEF	0.890 (0.850-0.931)	<0.001	0.89 (0.84-0.94)	<0.0001
Mitral Regurgitation		0.002		
Mild	0.77 (0.409-1.478)	0.44		
Moderate	1.558 (0.841-2.887)	0.15		
Severe	1.943 (0.997-3.787)	0.05		
Aort Regurgitation		0.53		
Mild	1.106 (0.668-1.836)	0.69		
Moderate	1.048 (0.332-3.312)	0.93		
Severe	4.322 (0.594-31.444)	0.14		
Tricuspid Regurgitation		<0.001		0.019
Mild	1.416 (0.572-2.429)	0.22	-	-
Moderate	3.210 (1.810-5.691)	<0.001	-	-
Severe	4.232 (1.953-9.172)	<0.001	3.00 (1.198-7.533)	0.019
TAPSE mm	0.943 (0.909-0.979)	0.002	-	-
RVsm (TDI) m/sec	0.864 (0.806-0.927)	<0.001	-	-
TRV m/sev	0.949 (0.809-1.113)	0.52	-	-
SPAP mmHg	1.030 (1.013-1.048)	0.001	-	-

Values are mean ±SD or n (%) p value<0.05.
ICD= Implanted cardioverter-defibrillator; LVEDd= Left ventricular end diastolic diameter; LVESd= Left ventricular end systolic diameter; LAd= Left atrial diameter; LVEF= Left ventricular systolic ejection fraction; TAPSE= Tricuspid annuler plane systolic excursion; RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure

Table 7. Cox univariate and multivariate analysis of clinical parameters CRT-D Group

	Univariate analysis CRT-D Group		Multivariate analysis CRT-D Group	
	HR (95% CI)	P-value	HR (95% CI)	P-value
Age years	1.017 (0.989-1.046)	0.22	-	-
Gender	1.073 (0.584-1.972)	0.82	-	-
Arterial hypertension	0.807 (0.420-1.550)	0.51	-	-
Diabetes mellitus	0.505 (0.242-1055)	0.06	-	-
Dyslipidemia	1.251 (0.446-3.509)	0.67	-	-
Smoking history	1.554 (0.854-2.828)	0.14	-	-
COPD	2.125 (0.279-16.191)	0.46	-	-
Renal Disease	0.046 (0.000-49.511)	0.38	-	-
AF	1.308 (0.504-3.397)	0.58	-	-
Family History	1.030 (0.248-4.268)	0.96	-	-
ACEI or ARB	0.58 (0.29-1.14)	0.11	0.36 (0.135-0.985)	0.047
Beta blocker	0.352 (0.122-1.015)	0.051	-	-
Aldosterone antagonist	1.202 (0.507-2.852)	0.67	-	-
Loop diuretic	2.142 (0.766-5.986)	0.14	-	-
Anticoagulant	0.705 (0.370-1.341)	0.28	-	-
Antiplatelet	1.368 (0.763-2.452)	0.29	-	-
If channel blocker	0.521 (0.242-1.120)	0.09	-	-

Cox univariate and multivariate model, P value<0.05.
HR= Hazard ratio; CI= Confidence interval
CRT-D= Cardiac resynchronization therapy; COPD = Chronic obstructive pulmonary disease;
AF= Atrial fibrillation; ACEi=Angiotensinogen was converting enzyme inhibitor;
ARB=Angiotensin receptor blocker

Table 8. Cox univariate and multivariate analysis of echocardiographic parameters CRT-D Group

	Univariate analysis CRT-D Group		Multivariate analysis CRT-D Group	
	HR (95% CI)	P-value	HR (95% CI)	P-value
LVEDd	0.994 (0.970-1.017)	0.59	-	-
LVESd	1.003 (0.982-1.025)	0.77	-	-
LAd	1.008 (0.971-1.047)	0.67	-	-
LVEF	0.959 (0.896-1.027)	0.22	-	-
RVEF	0.979 (0.955-1.004)	0.09	-	-
Mitral Regurgitation		0.01	-	-
Mild	0.063 (0.011-0.343)	0.001	-	-
Moderate	0.067 (0.012-0.358)	0.002	-	-
Severe	0.081 (0.013-0.485)	0.006	-	-
Aort Regurgitation		0.38	-	-
Mild	1.608 (0.817-3.166)	0.16	-	-
Moderate	1.273 (0.387-4.184)	0.69	-	-
Severe	-	-	-	-
Tricuspid Regurgitation		0.59	-	-
Mild	1.416 (0.572-3.502)	0.45	-	-
Moderate	1.107 (0.400-3.064)	0.84	-	-
Severe	1.907 (0.658-5.527)	0.23	-	-
TAPSE	0.983 (0.930-1.038)	0.53	-	-
RVsm (TDI) m/sec	0.864 (0.755-0.990)	0.03	0.85 (0.741-0.988)	0.034
TRV	1.383 (0.434-4.403)	0.58	-	-
SPAP	1.003 (0.974-1.034)	0.81	-	-

Values are mean ±SD or n (%) p value<0.05.
CRT-D= Cardiac resynchronization therapy; LVEDd= Left ventricular end diastolic diameter;
LVESd= Left ventricular end systolic diameter; LAd= Left atrial diameter; LVEF= Left ventricular systolic ejection fraction; TAPSE= Tricuspid annuler plane systolic excursion;
RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure

Heart failure

OP-029

The Ferric Carboxymaltose Treatment Impacts on Tp-e Interval, Tp-e/QT Ratio and Tp-e/QTc Ratio in Heart Failure Patients with Iron Deficiency

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Background and Aim: In heart failure (HF) patients with iron deficiency, cardiac electrical irregularity might be a cause of arrhythmia in HF. The aim of our study was to evaluate the effect of ferric carboxymaltose (FCM) treatment on T wave peak to end (Tp-e) interval, Tp-e/QT ratio and Tp-e/corrected QT (QTc) ratio reflecting transmural dispersion of repolarization in HF patients with iron deficiency.

Methods: Forty HF patients with iron deficiency treated with FCM were included in our single center observational study. Repolarization parameters on electrocardiography recorded before and 12 weeks after FCM treatments of these patients were compared. Additionally, these parameters were compared with ventricular repolarization parameters of the control group consisting of 40 healthy age and gender matched individuals and another group of 40 HF patients without iron deficiency.

Results: The clinical data, laboratory parameters, ECG parameters and echocardiography parameters of the patients are presented in Table 1. In the HF patients with iron deficiency, the Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio before FCM treatment were 103.72 ± 19 ms, 0.25 ± 0.04, 0.23 ± 0.04, respectively. These values were higher compared to the healthy control group and HF patient group without iron deficiency (p<0.001) (Table 1). In the HF patients with iron deficiency, the Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio after FCM treatment were lower compared to pre-treatment (89.35 ± 18.57 ms, 0.22 ± 0.04, 0.20 ± 0.04, respectively; p<0.001) (Table 2). It was determined that among the iron deficient HF patients, at the 12th week follow-up after FCM treatment, there was no statistically significant difference between the patients with and those without iron deficiency in terms of the Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio (p= 0.42, p= 0.36, p= 0.38, respectively). However, it was observed that the difference between the HF patient

group with iron deficiency who received FCM treatment and the healthy control group in terms of the Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio at the 12th week follow-up after the treatment was statistically significant (p=0.02, p=0.01, p=0.009 respectively) (Table 3). In the HF patients included in our study, there was a negative correlation between the ferritin level and Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio [r= - 285 (p= 0.002), r= - 0.280 (p= 0.002), r= - 0.304 (p= 0.001), respectively] (Figure 1).

Conclusions: In HF patients, iron deficiency causes prolonged Tp-e interval, high Tp-e/QT and Tp-e/QTc ratios. FCM treatment given to HF patients with iron deficiency corrects prolonged Tp-e interval and high Tp-e/QT ratio and Tp-e/QTc ratio, which are risk factors for ventricular arrhythmia. By this means FCM treatment may improve electrical negative effects of iron deficiency in HF patients with iron deficiency.

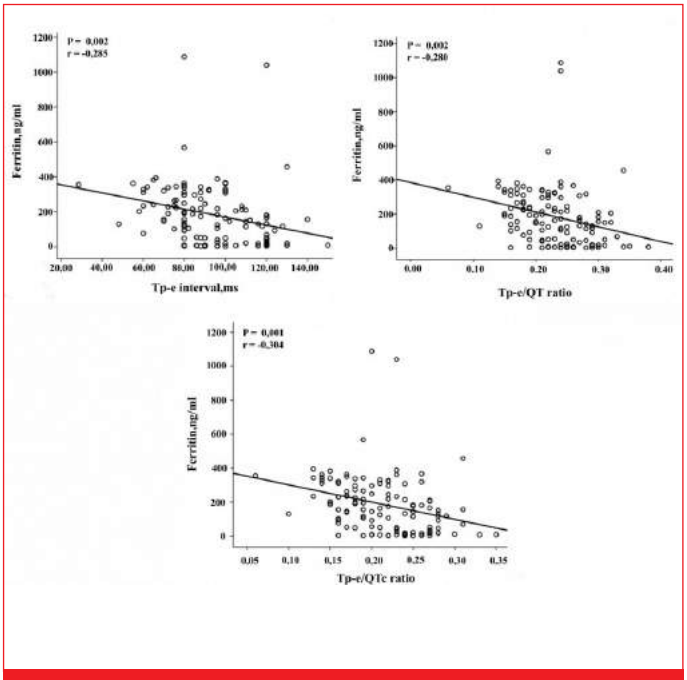

Figure 1. Correlation of Tp-e interval, Tp-e/QT ratio, Tp-e/QTc ratio with ferritin level

QT: QT interval; QTc: Corrected QT interval; Tp-e: T wave peak to end interval.

Table 1. Clinical characteristics, electrocardiographic, echocardiographic, and laboratory findings of the study groups

Variables	Healthy control group (n=40)	Heart failure patients without iron deficiency (n=40)	Heart failure patients with iron deficiency before ferric carboxymaltose treatment (n=40)	Heart failure patients with iron deficiency at 12 weeks after ferric carboxymaltose treatment (n=40)	P-value
Age, years	60 ± 7.04	61.1 ± 13.03	64.45 ± 16.39	64.45 ± 16.39	0.34
Gender, female (n, %)	17, 42.5%	14, 35%	17, 42.5%	17, 42.5%	0.87
LVEF, %	59.1 ± 3.55	33.83 ± 12.44	42.65 ± 15.29	42.87 ± 15.12	<0.001
LVEDD, mm	43.25 ± 2.55	58.05 ± 9.67	53.1 ± 8.14	52.9 ± 8.08	<0.001
Heart rate, beat/min	74.85 ± 13.33	73.8 ± 10.3	79.62 ± 19.66	76.32 ± 16.72	0.49
QT interval, ms	405.2 ± 38.39	404.1 ± 40.3	395.55 ± 47.27	398.40 ± 50.38	0.73
QTc interval, ms	431.7 ± 30.71	429.5 ± 31.76	427.15 ± 34.18	424.2 ± 35.92	0.77
QT dispersion, ms	25.90 ± 5.12	28.53 ± 7.74	33.82 ± 10.31	29.20 ± 8.26	<0.001
Tp-e interval, ms	78.7 ± 16.47	85.76 ± 21.9	103.72 ± 19.08	89.35 ± 18.57	<0.001
Tp-e/QT ratio	0.19 ± 0.04	0.21 ± 0.05	0.25 ± 0.04	0.22 ± 0.04	<0.001
Tp-e/QTc ratio	0.17 ± 0.04	0.19 ± 0.05	0.23 ± 0.04	0.20 ± 0.04	<0.001
Hemoglobin, g/dl	13.93 ± 0.53	13.52 ± 1.73	10.92 ± 2.29	12.58 ± 1.59	<0.001
Ferritin, ng/ml	227.15 [115.2-691]	221.70 [102.1-394]	17.95 [2-132]	242.6 [105-1087]	<0.001
NT-pro BNP, pg/ml	56 [50-75]	1025 [72-10000]	1034 [75-10000]	634 [50-10000]	<0.001
Creatinine, mg/dl	0.73 [0.52-1.1]	0.94 [0.46-6.21]	0.88 [0.43-2.67]	0.91 [0.35-2.57]	0.007
eGFR, mL/min/1.73 m^2	107.28 ± 36.25	78.37 ± 31.85	89.51 ± 41.09	78.65 ± 34.29	0.001
Transferrin saturation, %	25.01 ± 3.24	24.39 ± 3.50	11.91 ± 3.80	24.39 ± 2.78	<0.001

Normally distributed values are presented as mean ± standard deviation, non-normally distributed values are presented as median (minimum-maximum), and categorical values are presented as number of patients. The means of the groups were compared using the one-way ANOVA test. P value of < 0.05 shows statistical significance. eGFR: estimated Glomerular filtration rate according to Modification of Diet in Renal Disease formula; LVEDD: left ventricular end-diastolic diameter; LVEF: left ventricular ejection fraction; NT-pro BNP: N-terminal pro-B-type natriuretic peptide; QTc: Corrected QT interval; Tp-e: T wave peak to end interval.

Table 2. Comparison of the baseline data of the heart failure patients with iron deficiency and 12 weeks after ferric carboxymaltose treatment

Variables	Heart failure patients with iron deficiency before ferric carboxymaltose treatment (n=40)	Heart failure patients with iron deficiency at 12 weeks after ferric carboxymaltose treatment (n=40)	P-value
Tp-e interval, ms	103.72 ± 19.08	89.35 ± 18.57	<0.001
Tp-e/QT ratio	0.25 ± 0.04	0.22 ± 0.04	<0.001
Tp-e/QTc ratio	0.23 ± 0.04	0.20 ± 0.04	<0.001
QT dispersion, ms	33.82 ± 10.31	29.20 ± 8.26	<0.001
LVEF, %	42.65 ± 15.29	42.87 ± 15.12	0.68
LVEDD, mm	53.10 ± 8.14	52.9 ± 8.08	0.41
NT-pro BNP, pg/ml	1034 [75-10000]	634 [50-10000]	0.001

Normally distributed values are presented as mean±standard deviation, non-normally distributed values are presented as median (minimum-maximum). P value of < 0.05 shows statistical significance. LVEDD: left ventricular end-diastolic diameter; LVEF: left ventricular ejection fraction; NT-pro BNP: N-terminal pro-B-type natriuretic peptide; QTc: Corrected QT interval; Tp-e,:T wave peak to end interval.

Table 3. Comparison of the parameters of the heart failure patients with iron deficiency at 12 weeks after ferric carboxymaltose treatment with those of the healthy control group and heart failure patients without iron deficiency

Variables	Healthy control group (n=40)	Heart failure patients without iron deficiency (n=40)	Heart failure patients with iron deficiency at 12 weeks after ferric carboxymaltose treatment (n=40)	P-value
Tp-e interval, ms	78.7 ± 16.47	85.76 ± 21.9	89.35 ± 18.57	0.02* 0.42#
Tp-e/QT ratio	0.19 ± 0.04	0.21 ± 0.05	0.22 ± 0.04	0.01* 0.36#
Tp-e/QTc ratio	0.17 ± 0.04	0.19 ± 0.05	0.20 ± 0.04	0.009* 0.38#
QT dispersion,ms	25.90 ± 5.12	28.53 ± 7.74	29.20 ± 8.26	0.08* 0.73#

Normally distributed values are presented as mean ± standard deviation. P value of < 0.05 shows statistical significance. *p = Comparison between healthy control group and heart failure patients with iron deficiency at 12 weeks after ferric carboxymaltose treatment. #p = Comparison between heart failure patients without iron deficiency and heart failure patients with iron deficiency at 12 weeks after ferric carboxymaltose treatment. QTc: Corrected QT interval; Tp-e: T wave peak to end interval.

Heart failure

OP-030

Survival and cardiac magnetic resonance case-control trial of peripartum cardiomyopathy

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Background and Aim: Peripartum cardiomyopathy(PPCM) is rare cardiomyopathy with unknown etiology. The diagnosis is made by excluding other identifiable causes of heart failure. It occurs towards the end of the pregnancy or during the postpartum period of five months presenting as heart failure clinical spectrum with left ventricular systolic dysfunction of left ventricle ejection fraction (LVEF) < 45%. The purpose of this study is to retrospectively evaluate the clinical characteristics, cardiac magnetic resonance(CMR) imaging features, and relationship with end-points that consist of left ventricle recovery, left ventricular assist device implantation, heart transplantation, and all-cause mortality.

Methods: Outpatient heart failure records between 2008 to 2020 were screened. Thirty-seven patients were defined as PPCM. Twenty-five patients had cardiac MRI evaluation at the time of diagnosis, and six patients were re-evaluated during outpatient follow-up at least one year after the diagnosis.

Results: The mean age was 30.5 ± 5.6 years and the mean baseline LVEF was 28.2 ± 6.7%. In thirteen (35.7%) patients, left ventricular systolic function recovered during the follow-up course. The median recovery time was 281 [IQR (78-358)] days. Patients' LVEF that been evaluated with CMR was 35.3 ± 10.5. Three patients exhibited late gadolinium enhancement (LGE) patterns. Sub-endocardial and mid-wall uptake pattern types were detected. 18 (75%) patients met the Petersen non-compaction cardiomyopathy criteria. Patients that have NC/C ratio lower than 2.3 had lower LVED-V(i) and LVESV(i) (124.9 ± 35.4, 86.4 ± 7.5, p=0.003; 86.8 ± 34.6, 52.6±7.6, p=0.006), respectively. The median follow-up time was 2129 [IQR (911-2634)] days. The primary end-point free one-year survival was 88.9% (event rate 11.1%), and five-year survival was 75.7%.

Conclusions: In a retrospective cohort of PPCM patients, 35.7% of patients' LVEF recovered, and the primary end-point free five-year survival was 75%. 25 patients were assessed with CMR, and three of four patients met the CMR-derived Petersen criteria of left ventricle non-compaction cardiomyopathy (LVNC).

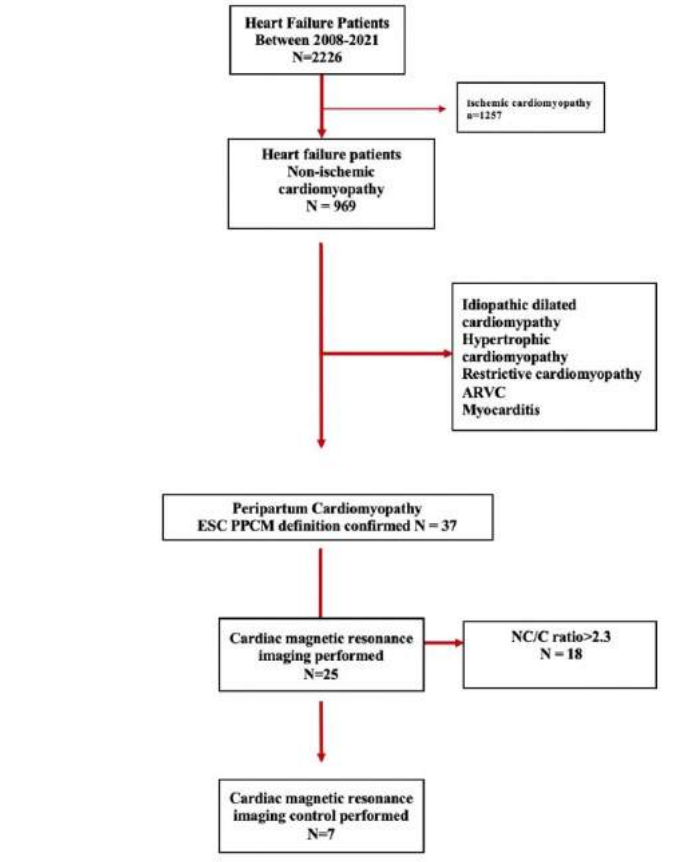

Figure 1. Flow chart of study patients/consort diagram

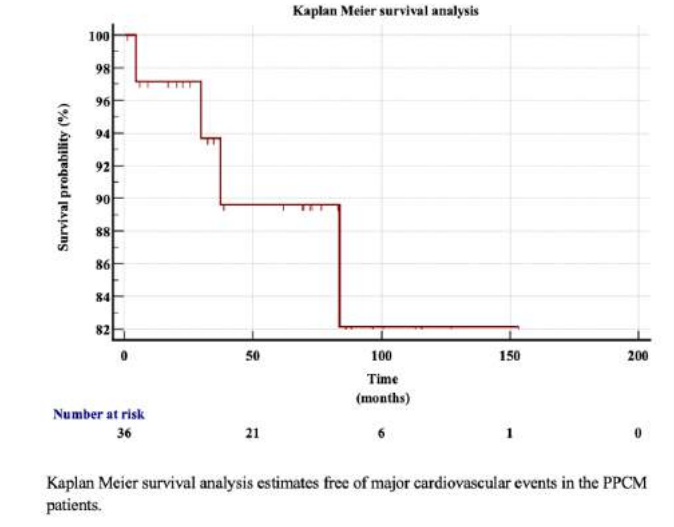

Figure 2. Kaplan Meier survival analysis of the peripartum cardiomyopathy patients

Heart failure

OP-031

Demographic, clinical, and disease characteristics of patients with atrial fibrillation on edoxaban therapy: What are the impacts of heart failure? A subgroup analysis from ETAF-TR Study

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Background and Aim: The incidence and prevalence of both heart failure (HF) and atrial fibrillation (AF) are getting higher in older people. Although HF is a component of CHADS-VASc score, recent studies demonstrated that atrial myopathy was common in patients with HF with preserved ejection fraction (HFpEF) and anticoagulation should be thought of in these patients independent of CHADS-VASc score.

This analysis aims to determine of demographic, clinical and disease characteristics of patients with AF on real-life edoxaban therapy and to assess the impacts of heart failure on treatment strategy.

Methods: This was a subgroup analysis from ETAF-TR Study, which was a national, multicenter, prospective, observational study that enrolled 1053 patients from 50 centers in Turkey. The primary outcome of the ETAF-TR study was any overt bleeding (consisting of major bleeding or clinically relevant nonmajor bleeding or any bleeding that does not meet this definition but is considered as overt bleeding by the participating physician).

HF was defined by and classified into three groups by left ventricular ejection fraction (low, mildly reduced and preserved ejection fraction) according to the latest heart failure guideline of the European Society of Cardiology. Demographic, clinical and disease characteristics of patients with AF were compared between HF groups.

Results: Of 1038 patients enrolled to the study, 305 (35.5%) had HF. The mean age was similar between the total and HF population. Coronary artery disease and arterial hypertension were prevalent in the HF population. CHA2DS2-VASc score was higher in patients with HF (3.2 ± 1.4 vs. 4.2 ± 1.5, p<0.0001). A proper dose selection was similar between the two populations (p=0.69). The rate of overtreatment was 8.7% in patients without HF and so was 11.8% in patients with HF (p=0.13). Moreover, the rate of undertreatment was 7.8% and 7.5% in patients with and without HF, respectively

(p=0.9) (Table 1). Of 305 HF patients, 129 (42.3%) was HFrEF, 87 (28.5%) were HFmrEF and 86 (28.2%) were HFpEF. Whilst male was predominant [85 (65.9%) of patients] in the HFrEF group, the female was predominant [54 (62.8%) of patients] in the HFpEF group. In the HFmrEF group, both sexes were nearly equally represented (Table 2). BMI was higher in HFpEF. Diabetes, arterial hypertension, and permanent atrial fibrillation were similar among HF groups. The rate of previous coronary artery disease was lower in HFpEF. The mean CHA2DS2-VASc scores were 3.9, 4.5, and 4.4 in patients with HFrEF, HFmrEF and HFpEF, respectively. The main laboratory findings were not significantly different across HF groups (Table 2).

Conclusions: One-third of the study population had HF. The patients with HF with reduced, mildly reduced and preserved ejection fractions were nearly equally represented in the present study. HF did not affect the appropriate dose selection of edoxaban. A one-year follow-up will demonstrate the impact of heart failure on edoxaban therapy in real life in Turkey.

Table 1. The comparison of the baseline demographic findings of patients with and without heart failure

Variables	No Heart Failure (n=733)	With Heart Failure (n=305)	P value
Female, n (%)	470 (64.1)	144 (47.2)	<0.0001
Age, mean ± SD	70 ± 11	70 ± 12	0.2
Age ≥75, n (%)	259 (35.3)	128 (42)	0.10
BMI, mean ± SD	29.1 ± 5.3	29.2 ± 5.6	0.72
Smoking, active, n (%)	49 (6.7)	22 (7.2)	0.01
Alcohol, ≥1 drink per week, n (%)	21 (2.9)	11 (3.6)	0.23
HT, n (%)	0 (0)	305 (100)	<0.0001
DM, n (%)	187 (25.5)	91 (29.8)	0.15
CAD, n (%)	169 (24.3)	132 (49.6)	<0.0001
CABG, n (%)	25 (15.0)	42 (32.1)	0.005
Stroke, n (%)	107 (14.6)	31 (10.2)	0.06
Valve repair, n (%)	1 (0.1)	3 (1.0)	0.04
Biological heart valve, n (%)	2 (0.3)	3 (1.0)	0.13
PMBV, n (%)	2 (0.3)	0(0)	0.36
TAVR, n (%)	3 (0.4)	1 (0.3)	0.85
PAD, n (%)	22 (3.2)	11 (4.0)	0.53
Bleeding history, n (%)	62 (8.5)	28 (9.2)	0.71
CHA2DS2-VASc score, mean ± SD	3.2 ± 1.4	4.2 ± 1.5	<0.0001
SBP, mean ± SD	131 ± 17	129 ± 18	0.16
DBP, mean ± SD	78 ± 11	77 ± 11	0.23
Overtreatment, n (%)	64 (8.7)	36 (11.8)	0.13
Undertreatment, n (%)	57 (7.8)	23 (7.5)	0.9
Proper Dosage, n (%)	577 (78.7)	244 (80)	0.6

BMI - body mass index; CABG- coronary artery bypass grafting; CAD- coronary artery disease; DBP- diastolic blood pressure; DM- diabetes mellitus; PAD- peripheral arterial disease; PMBV- percutaneous mitral balloon valvuloplasty; SBP- systolic blood pressure; SD- standard deviation; TAVR- transcatheter aortic valve replacement

Table 2. The comparison of the baseline demographic and laboratory findings of patients with heart failure according to left ventricle ejection fraction

Variables	HFrEF (n=129)	HFmrEF (n=87)	HFpEF (n=86)	P value
Female, n (%)	44 (34.1)	44 (50.6)	54 (62.8)	0.0001
Age, mean ± SD	68 ± 13	72 ± 10	71 ± 14	0.01
Age ≥75, n (%)	43 (33.3)	40 (46.0)	43 (50.0)	0.11
BMI, n (%)	28.1 ± 4.7	28.8 ± 4.7	31.1 ± 6.9	0.007
Smoking, n (%)	12 (9.3)	7 (8.0)	3 (3.5)	0.002
Alcohol, n (%)	5 (3.9)	5 (5.7)	1 (1.2)	0.39
HT, n (%)	98 (76)	76 (87)	72 (84)	1.0
DM, n (%)	40 (3.1.0)	26 (0.1)	24 (28.3)	0.89
AF, permanent, n (%)	86 (66.7)	54 (62.1)	60 (69.8)	0.33
CAD, n (%)	57 (44.2)	50 (57.5)	25 (29.1)	0.001
CABG, n (%)	15 (11.6)	17 (19.5)	10 (11.6)	0.47
CHA2DS2-VASc score, mean ± SD	3.9 ± 1.6	4.5 ± 1.4	4.4 ±1.6	0.002
Concomitant antiplatelet use, n(%)	24 (18.6%)	15 (17.2%)	7 (8.1%)	0.09
SBP, mm Hg, mean ± SD	126 ± 20	135 ± 17	128 ± 16	0.004
DBP, mm Hg, mean ± SD	76 ± 11	81 ± 10	76 ± 12	0.001
Heart rate, bpm, median (IQR)	88 (76-107)	87 (75-99)	82 (74-94)	
Serum creatinine clerance, ml/min,	75.7 ± 31.13	77.9 ± 35.48	77.9 ± 33.85	0.94
Creatinine Clearance 15-50 ml/min, n (%)	29 (22.5%)	20 (23.0%)	18 (20.9%)	67 (22.2%)
Hematocrit, %,, mean ± SD	39.5 ± 6.13	40.4 ± 4.92	37.7 ± 5.80	0.006
Fasting blood glucose, mg/dL, mean ± SD	126.3 ± 55.62	128.5 ± 43.62	122.0 ± 45.63	0.10
SGPT, mg/dL, mean ± SD	24.5 ± 21.31	23.1 ± 17.32	20.5 ± 10.68	0.30

BMI - body mass index; bpm- beat per minute; CABG - coronary artery bypass grafting; CAD - coronary artery disease; DBP- diastolic blood pressure; DM - diabetes mellitus; HFmrEF - heart failure with mildly reduced ejection fraction; HFpEF - heart failure with preserved ejection fraction; HFrEF- heart failure with reduced ejection fraction; SBP - systolic blood pressure; SD- standard deviation; SGPT- glutamic-pyruvic transaminase

Heart failure

OP-032

Effect of short-term cardiac rehabilitation on beck depression scale in patients with heart failure

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Background and Aim: The coexistence of heart failure (HF) with depression is common and may worsen the prognosis. Lack of functional capacity, excessive accompanying morbid conditions, high mortality and complicated course of disease are important sources of depression in patients. Patient education, psychopharmacological and non-pharmacological treatment as well as exercise programs have been reported to be effective in the treatment of depression in HF. In our study, we investigated whether cardiac rehabilitation (CR) treatment causes variability on the 'Beck Depression Scale (BDI)' in patient with HF receiving medical therapy.

Methods: Patients with heart failure who were symptomatic under medical treatment, were included in the cardiac rehabilitation program and completed 30 sessions of cardiac reahbilitation program were screened retrospectively. Demographic features, laboratory results, 6-minutes walking tests, responses to the Beck Depression Scales before and after the CR program, and scale scores were obtained from the patient files. NCSS (Number Cruncher Statistical System) program (2007 Kaysville, Utah, USA) was used for statistical analysis. Statiscally, $p<0.05$ values were considered significant.

Results: The study was carried out between 2007-2020, Dr. Siyami Ersek Hospital Cardiac Rehabilitation Clinic. 54.8% (n=17) of the cases were male and 45.2% (n=14) were female. The ages of the cases included in the study ranged from 21 to 79 years, and the mean was 52.19 ± 11.99 years. Fourteen cases had 45.2% ischemic cardiomyopathy. The mean ejection fraction of the cases were 29 ± 8. Body mass index (BMI) of patients with heart failure before CR was found as 30.91 ± 5.25, while this value was calculated as 30.34 ± 5.03 after CR (p=0.008). In the 6-minute walking test of the patients, walking distances before the CR were found 353 ± 64.43 metres, after CR were found 379.81 ± 55.48 metres (p=0.009). Similar improvement was observed in WATT values, which was 58.06 ± 29.85 before CR and WATT value was measured as 71.45 ± 26.59 after CR (p=0.045). While the BDI score before CR was 13.35 ± 7.89, it was determined that this value decreased to 10.16 ± 7.9 after CR. The reduction of -3.19 ± 8.85 in BDI was statistically significant (p=0.045).

Conclusions: Our study findings, it shows that CR treatment in people with HF causes a significant increase in the effort capacity of our patients and positively varies of BDI scores. This result, it suggests that CR programs which we will plan in addition to our medical treatment approaches in patient with HF, may have a positive effect on the quality of life of our patients.

Table 1. Results before and after cardiac rehabilitation

	Before CR	After CR	P value
EF (%)	31.29 ± 9	31.45 ± 10	>0.05
BMI	30.91 ± 5.25	30.34 ± 5.03	0.008
Six minute walk test (m)	353 ± 64.43	379.81 ± 55.48	0.009
WATT	58.06 ± 29.85	71.45 ± 26.59	0.045
BDI	13.35 ± 7.89	10.16 ± 7.9	0.045

BDI, Beck depression scale; BMI, body mass index; CR, cardiac rehabilitaion; EF, ejection fraction

Table 2. Demographic characteristics of the patients

Demographic Features	n (%)
Age	52.19 ± 11.9
Male sex	17 (55%)
Diabetes mellitus	10 (32%)
Arterial hypertension	12 (38%)
Dislipidemia	17 (54%)
Smoking	5 (16%)
Coronary artery disease	14 (45.2%)
Medications	
Beta blocker	31 (100%)
ACEI/ARB	31 (100%)
CCB	10 (32%)
Statin	13 (41%)
MRA	16 (51%)
Nitrate	13 (41%)
Acetylsalicylic acid/Clopidogrel	31/10 (100/32%)

ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker; MRA, mineralocorticoid receptor antagonist

Heart failure

OP-033

Serum albumin to creatinine ratio as predictor for long-term mortality in patients with acute heart failure

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Background and Aim: The long-term association between serum albumin-to-creatinine ratio (sACR) and poor patient outcomes in acute heart failure (HF) remains unclear. This study aimed to determine whether sACR was a predictor of poor long-term survival in patients with acute decompensated heart failure with reduced ejection fraction (HFrEF).

Methods: This single-center retrospective study included 399 patients hospitalized for acute decompensated heart failure with reduced ejection fraction from January 2015 through January 2020. We noted detailed clinical data of the patients, including demographic data (age, gender), comorbidities [hypertension, diabetes mellitus (DM), atrial fibrillation, etc.], vital signs (heart rate, blood pressure, etc.), laboratory tests (blood routine, electrolytes, etc.) and echocardiographic features. Data on 1 and 5 year all-cause mortality was obtained from hospital electronic record system. The patients were categorized into tertiles (T1, T2, and T3) based on the sACR. The patients were grouped to tertile 1-3 with decreased sACR levels for analysis: the first tertile was sACR value < sACR < 2.64, second tertile was sACR value between ≥2.64, <3.75, third tertile was higher than 3.75. We visually demonstrated the relationship between sACR and patient's survival through the Kaplan-Meier (K-M) curve and used Log-rank tests for hypothesis testing.

Results: A total of 399 HFrEF patients hospitalized for acute decompensated heart failure were included, predominantly male 273 (68.4%) and with a mean age of 67.2 ± 11.5 years. In all population, 43.9% had DM and 58.9% had hypertension. The mean sACR was 3.21 ± 1.33. The presence of hypertension and chronic obstructive pulmonary disease was similar among tertiles. But, the presence of coronary artery disease and DM were significantly higher in the first tertile.Patients in first tertile, compared to those in second and third tertiles, had significantly higher NTproBNP (p<0.001), creatinin (p <0.001), troponin (p<0.001) and had significantly lower haemoglobin (p<0.001) and albumin levels (p<0.001). There was no significant difference in left ventricular ejection fraction between the groups (p=0.369). Overall, the 1-year survival rate was 24.1% and the 5-year mortality rate was 58.4%. Kaplan-Meier analysis showed that patient mortality in the first tertile was 78.2%, 63.6% in the second tertile and 33.6% in the third tertile at 5 years (Long-rank p value <0.001).

Conclusions: In conclusion, the sACR was independently associated with long term mortality (1 and 5 year) in HFrEF patients hospitalized with acute decompensated heart failure, indicating that baseline sACR was a useful biomarker to identify high-risk patients in acute heart failure at an early term.

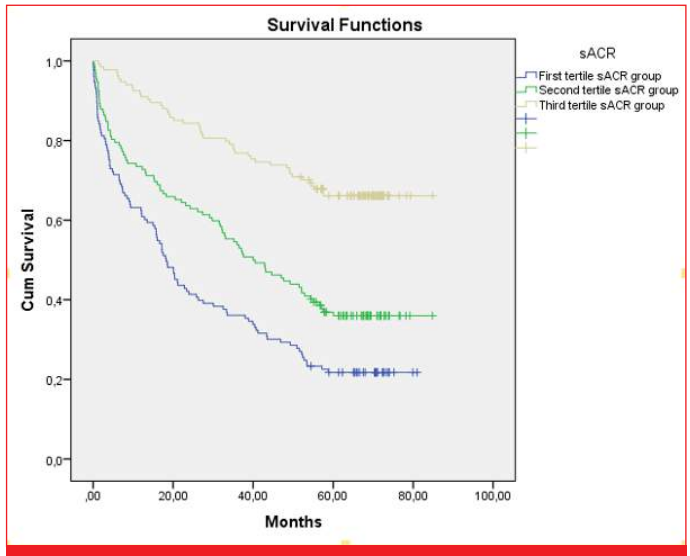

Figure 1. Kaplan-Meier survival analysis for tertiles according to sACR

Table 1. Baseline characteristics, laboratory values and mortality datas of the patients

	sACR < 2.64 (n=133)	sACR ≥2.64, < 3.75 (n=132)	sACR ≥3.75 (n=134)	P value
Age, years	70.4 ± 12.5	68.6 ± 10.2	62.6 ± 11.1	<0.001
Female, n (%)	39 (29.3%)	33 (25.0%)	54 (40.3%)	0.022
HT, n (%)	86 (64.7 %)	75 (56.80%)	74 (55.2%)	0.246
DM, n (%)	68 (51.1%)	60 (45.5%)	47 (35.1%)	0.027
CAD, n (%)	100 (75.2%)	95 (72.0%)	80 (59.7%)	0.016
COPD, n (%)	44 (33.1%)	40 (30.3%)	30 (22.4%)	0.133
HR, beats/minute	80.2 ± 19.8	77.3 ± 15.4	76.6 ± 18.9	0.131
Hemoglobin, g/dl	11.3 ± 1.89	12.6 ± 1.8	13.2 ± 1.8	<0.001
NT-proBNP, pg/ml	11774 (4341.0-24821.0)	3526.5 (1211.0-9551.7)	1359.0 (509.0-3009.0)	<0.001
Albumin, g/dl	3.37 ± 0.58	3.68 ± 0.45	4.04 ± 0.51	<0.001
Creatinin, mg/dL	2.26 ± 1.14	1.15 ± 0.16	0.88 ± 0.15	<0.001
GFR	34.3 ± 14.07	63.9 ± 20.9	85.7 ± 24.02	<0.001
Glucose, mg/dL	124 (91.5-193.5)	115.0 (88.0-155.0)	100 (81.0-126.2)	<0.001
Sodium, mmol/l	136.8 ± 5.2	137.8 ± 4.7	139.4 ± 4.1	<0.001
Troponin-T, ng/L	0.069 (0.004-0.14)	0.033 (0.019-0.054)	0.020 (0.012-0.033)	<0.001
LVEF, %	25.1 ± 7.6	25.0 ± 8.3	26.2 ± 7.7	0.369
Potassium,, mEq/L	4.54 ± 0.5	4.47 ± 0.54	4.48 ± 0.50	0.611
LA diameter, mm	45.6 ± 6.6	46.2 ± 6.5	48.1 ± 16.2	0.073
SPAP, mm Hg	53.0 ± 14.9	48.4 ± 16.0	50.6 ± 15.8	0.028
1 year all-cause mortality, n (%)	50 (37.6%)	34 (25.8%)	12 (9.0%)	<0.001
5 year all-cause mortality, n (%)	104 (78.2%)	84 (63.6%)	45 (33.6%)	<0.001

HT:Hypertension, DM: diabetes mellitus, CAD: coronary artery disease, COPD: Chronic obstructive pulmonary disease, HR: heart rate, GFR: glomerular filtration rate, LVEF: left ventricular ejeciton fraction, LA: left atrium, SPAP: systolic pulmonary artery pressure

Heart failure

OP-034

Aspirin use and the survival in the non-ischemic heart failure patients

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Background and Aim: Aspirin use in non-ischemic heart failure patients is still a matter of debate. Patients with atherosclerosis or high cardiovascular risk have some reasons to use it,but in the non-ischemic heart failure patients,it has not been proven yet by robust studies. Aspirin is off-label prescribed to prevent intracardiac thrombus formation. However, aspirin may decrease the ACE inhibitors' beneficial effects,the primary drugs that prolong survival and it increases bleeding risk. This study aims to investigate the two-way aspirin effect on composite primary end-points (mechanical circulatory support, heart transplantation or all-cause mortality, ventricular arrhythmia or heart failure-related hospitalization).

Methods: This study is retrospective observational study performed between 2008-2021,outpatient heart failure medical records were reviewed,and 582 patients were included after exclusion criterias were applied. Ischemic etiology of heart failure patients had been proved by coronary angiography; without documented normal coronary artery anatomy of coronary computed tomography results for suspected acute coronary artery disease, myocardial perfusion scintigraphy with positive ischemia results or cardiac magnetic resonance imaging with transmural late gadolinium enhancement were excluded from the study. According to Felker et al criteria, over 50% of proximal LAD artery or two coronary segments stenosis over 50% were agreed to be probable cause for ischemic heart failure etiology. Patients with idiopathic dilated,non-compaction, peripartum, arrhythmogenic, hypertrophic and restrictive cardiomyopathy were included in the study without applying any LVEF restriction. Patients taking anticoagulants were excluded from the study.

Results: 80 (38.3%) patients were female, and the mean age was 45.2 ± 12.4 years. 62 (22.5%) patients had a diagnosis of diabetes mellitus (DM), 73 (27.7%) had hypertension 114 (43.2%) patients had a smoking history. The mean LVEF was 27.4 ± 10.2. Patient characteristics are shown in Tables 1, 2. Kaplan Meiers' analysis showed that aspirin using patients had the worst survival rate than non-aspirin users (log rank test p value=0.012) Figure 1. Univariate Cox regression analysis revealed that aspirin positively affected patients with DM and smoking history. RAAS blockers and beta-blockers had a positive impact on composite survival in aspirin-using group.Multivariate cox regression analysis revealed that aspirin was effective for the patients who had DM and who had higher LVEF (Tables 3, 4).

Conclusions: Aspirin using patients' survival was worse than non-users in non-ischemic cardiomyopathy patients.As previously claimed, using aspirin with RAAS blockers did not reduce the effects of RAAS blockers on survival, and similar findings were seen with beta-blockers. Non-ischemic cardiomyopathy patients with high cardiovascular risk factor like DM had beneficial effects when aspirin was used.Therefore,aspirin can be preferred in patients with non-ischemic cardiomyopathy,particularly in those with DM.

Table 1. Characteristics of the patients with non-ischemic cardiomyopathy that use aspirin

	Total
Age years	45.2 ± 12.4
Female n (%)	80 (30.3%)
ICD n (%)	100 (37.9%)
CRT-D n (%)	32 (12.1%)
Arterial hypertension n (%)	73 (27.7%)
Diabetes mellitus n (%)	62 (23.5%)
Dyslipidemia n (%)	26 (9.8%)
Smoking history n (%)	114 (43.2%)
Cerebrovascular event n (%)	20 (6.2%)
COPD n (%)	23 (4.4%)
Renal Disease n (%)	8 (3.0%)
AF n (%)	66 (25.0%)
Family history of heart failure n (%)	27 (10.2%)
ACEi or ARB n (%)	212 (80.3%)
Beta blocker n (%)	252 (95.5%)
Aldosterone antagonist n (%)	196 (74.2%)
Loop diuretic n (%)	269 (98.1%)
Hydrochlorothiazide n (%)	1 (0.4%)
If channel blocker n (%)	109 (20.5%)

*Values are mean ± SD or n (%) p value<0.05. LVEF, Left ventricular ejection fraction; ICD, Implanted cardioverter-defibrillator; CRT-D, Cardiac resynchronization therapy-Defibrillator ACEi, Angiotensinogen-converting enzyme inhibitor; ARB, Angiotensin receptor blocker; AF, Atrial fibrillation; BMI, body mass index; BSA, body surface area; COPD, Chronic obstructive pulmonary disease

Table 2. Echocardiographic parameters of the patients with non-ischemic cardiomyopathy that use aspirin

	Total
LVEDd	63.3 ± 10.3
LVESd mm	53.6 ± 12.7
LAd mm	46.9 ± 7.7
LVEF %	27.4 ± 10.2
Mitral Regurgitation n (%)	
Mild	114 (43.2%)
Moderate	96 (36.4%)
Severe	33 (12.5%)
Aortic Regurgitation n (%)	
Mild	47 (17.8%)
Moderate	7 (2.7%)
Severe	1 (0.4%)
Tricuspid Regurgitation n (%)	
Mild	136 (51.5%)
Moderate	48 (18.2%)
Severe	24 (9.1%)
TAPSE mm	17.6 ± 5.9
RVsm (TDI) m/sec	10.4 ± 2.81
TRV	2.75 ± 0.58
SPAP	42.4 ± 12.3
Outcomes n %	264 (49.6%)

Values are mean ±SD or n (%) p value<0.05. LVEDd, Left ventricular end diastolic diameter; LVESd, Left ventricular end systolic diameter; LAd, Left atrial diameter; LVEF, Left ventricular systolic ejection fraction; TAPSE, Tricuspid annuler plane systolic excursion; RVsm, Right ventricular systolic motion tissue doppler imaging; TRV, Tricuspid regurgitation velocity; SPAP, Systolic pulmonary artery pressure

Table 3. Cox univariate and multivariate analyses of clinical parameters in patients with non-ischemic dilated cardiomyopathy that use aspirin

	Univariate analysis	Univariate analysis	Multivariate analysis	Multivariate analysis
	HR (95% CI)	P value	HR (95% CI)	P value
Age years	1.006 (0.991-1.020)	0.44	-	-
Gender	0.77 (0.54-1.11)	0.17	-	-
Arterial hypertension	0.91 (0.63-1.31)	0.61	-	-
Diabetes mellitus	0.64 (0.43-0.97)	0.036	0.61 (0.38-0.98)	0.042
Dyslipidemia	1.02 (0.60-1.72)	0.92	-	-
Smoking history	1.41 (1.01-1.96)	0.043	-	-
COPD	1.20 (0.56-2.58)	0.62	-	-
Renal Disease	1.09 (0.40-2.98)	0.85	-	-
Atrial fibrillation	0.92 (0.67-1.42)	0.98	-	-
Family History	1.04 (0.62-1.75)	0.87	-	-
ACEI or ARB	0.64 (0.43-0.95)	0.03	-	-
Beta blocker	0.38 (0.20-0.71)	0.03	-	-
Aldosterone antagonist	1.64 (0.44-0.96)	0.31	-	-
Loop diuretic	0.77 (0.24-2.43)	0.66	-	-

Cox univariate and multivariate model, p value<0.05. HR, Hazard ratio; CI, Confidence interval ACEi, Angiotensinogen converting enzyme inhibitor; ARB, Angiotensin receptor blocker; AF, Atrial fibrillation; COPD, Chronic obstructive pulmonary disease

Table 4. Cox univariate and multivariate analyses of echocardiographic parameters of the patients with non-ischemic dilated cardiomyopathy that use aspirin

	Univariate analysis	Univariate analysis	Multivariate analysis	Multivariate analysis
	HR (95% CI)	**P value**	**HR (95% CI)**	**P value**
LVEDd	1.025 (1.008-1.043)	0.003	-	-
LVESd	1.025 (1.010-1.041)	0.001	-	-
LAd	1.035 (1.015-1.056)	< 0.001	-	-
LVEF	0.96 (0.93-0.98)	0.002	0.94 (0.92-0.97)	0.001
Mitral Regurgitation				
None-Mild	2.721 (0.651-11.377)	0.96	-	-
Severe	0.97 (0.49-1.92)	0.94	-	-
Aortic Regurgitation				
None-Mild	1.0 (0.66-1.51)	0.23	-	-
Severe	4.59 (0.63-33.3)	0.006	-	-
Tricuspid Regurgitation				
None-Mild	1.09 (0.71-1.67)	0.69	-	-
Severe	1.79 (0.99-3.24)	0.052	-	-
TAPSE	0.95 (0.91-0.98)	0.004	-	-
RVsm (TDI) m/sec	0.88 (0.81-0.92)	< 0.001	-	-
TRV	1.22 (0.77-1.92)	0.86	-	-
SPAP	1.032 (1.015-1.050)	< 0.001	-	-

Values are mean ±SD or n (%) p value <0.05. HR, Hazard ratio; CI, Confidence interval LVEDd, Left ventricular end diastolic diameter; LVESd, Left ventricular end systolic diameter; LAd, Left atrial diameter; LVEF, Left ventricular systolic ejection fraction; TAPSE, Tricuspid annuler plane systolic excursion; RVsm, Right ventricular systolic motion tissue doppler imaging; TRV,Tricuspid regurgitation velocity; SPAP, Systolic pulmonary artery pressure

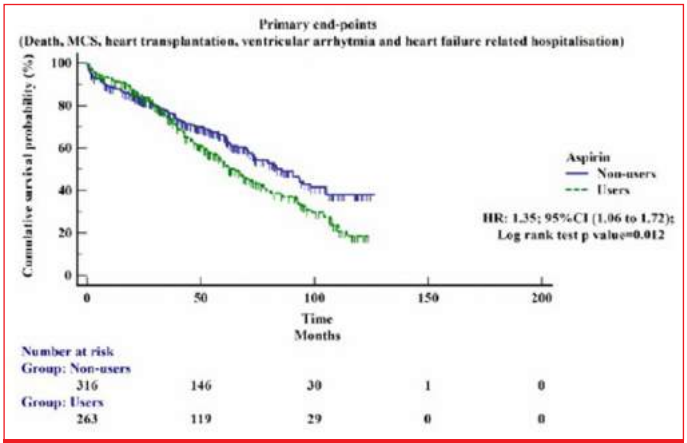

Figure 1. Comparison of Kaplan Meier survival analyses of patients with non-ischemic cardiomyopathy between aspirin users and non-users

Heart failure

OP-035

The effect of education on hospitalization and quality of life in patients with heart failure

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Background and Aim: Heart failure (HF) is a chronic and progressive clinical syndrome characterized by increased neurohormonal activity and various organ dysfunctions, in which the heart cannot pump the required amount of blood to the tissues to meet the body's metabolic needs or can only do so with increased cardiac filling pressures. Heart failure disease, which has heavy individual, social and economic obligations; increasing the fight power of patients by recognizing heart failure, reducing recurrent hospitalizations that increase morbidity and mortality, increasing the strength and awareness of patients by recognizing the symptoms that may develop due to heart failure, recognizing the drugs used by the patients, knowing the possible side effects and benefits, thus increasing their participation in the treatment is importantIn this context, our study was conducted to examine the efficacy of education given to patients followed up with heart failure on quality of life and recurrent hospitalizations.

Methods: A total of 100 participant diagnosed with heart failure were included in our study. 50 of the participants were randomly assigned to the control group and 50 to the intervention group.Routine nursing care was given to the control group patients, and heart failure patient education was given to the intervention group. All patients were evaluated with the 'Minnesota Living With Heart Failure Questionnaire' before and after treatment.

Results: Initial and post-treatment of the control group; When the median mean scores of the intervention group were evaluated at baseline and after treatment and heart failure training;In the intervention group given heart failure education, all symptoms improved from baseline with a highly significant p value ($p<0.001$). In the control group, improvement was observed in the symptoms of 'Eating less foods I like' ($p=0.048$), 'Hospitalization' ($p<0.001$) and 'Side effects from medications' ($p=0.042$). However; In the control group, there was a statistically significant worsening of 'Swelling in your ankles, legs' symptom ($p=0.041$). When the difference in the power of the recovery level of the same symptom (delta symptom: ds) between the control and intervention groups is compared; The power of the level of recovery in the intervention group was found to be stronger than the control group, with the p-value being highly significant ($p=0.000$) in all symptoms, except for the "treatments you received caused side effects" ($p=0.979$).

Conclusions: Consequently; It was seen that the median mean scores of the Minnesota Living With Heart Failure Questionnaire were significantly higher in the intervention group, which received patient education for heart failure, compared to the control group that did not receive training, and that the patients' repeated hospitalizations decreased. We think that simple and low-cost patient education in heart failure will be highly effective on patients' quality of life.

Table 1. Minnesota Living With Heart Failure Questionnaire (MLHFQ)

	Symptom
S1	Swelling in your ankles, legs
S2	Resting during days
S3	Walking or climbing stairs difficult
S4	Working around house difficult
S5	Being away from home difficult
S6	Sleeping difficult
S7	Relating to or doing things with friends or family difficult
S8	Working to earn a living difficul
S9	Recreational activities difficult
S10	Sexual activities difficult
S11	Eating less foods I like
S12	Shortness of breath
S13	Fatigue
S14	Hospitalization
S15	Medical costs
S16	Side effects from medicationsı
S17	Feeling burden to family or friends
S18	Feeling a loss of self-control
S19	9. Being worried
S20	Difficulty concentrating or remembering
S21	Being depressed

S: Symptom, MLHFQ = Minnesota Living With Heart Failure Questionnaire.

Table 2. The difference in the level of improvement of the same symptom between the control and intervention groups with Mann Whitney U test (delta symptom:ds)

	Mann-Whitney U	Wilcoxon	P
ds1	328.500	1409.500	<0.001
ds2	348.500	1429.500	<0.001
ds3	349.500	1430.500	<0.001
ds4	356.000	1437.000	<0.001
ds5	249.000	1330.000	<0.001
ds6	373.500	1454.500	<0.001
ds7	270.500	1351.500	<0.001
ds8	35.500	90.500	0.003
ds9	294.500	1375.500	<0.001
ds10	63.000	141.000	0.003
ds11	324.500	1405.500	<0.001
ds12	352.500	1433.500	<0.001
ds13	360.000	1441.000	<0.001
ds14	812.000	1893.000	0.50
ds15	460.000	1541.000	<0.001
ds16	1056.000	2137.000	0.979
ds17	335.500	1416.500	<0.001
ds18	343.000	1424.000	<0.001
ds19	363.000	1444.000	<0.001
ds20	349.500	1430.500	<0.001
ds21	293.000	1374.000	<0.001

ds: power of symptoms to change (delta symptom) Comparing the power of symptoms (delta symptom) between both groups (Table 2); The effectiveness of regression of symptoms in the intervention group was found to be stronger than the control group, with the p value being quite significant (p <0.001) in all symptoms, except S16 (Side effects from medications) (p=0.979).

Heart failure

OP-036

Non-ischemic cardiomyopathy patient's survival analyses by CHAID algorithm

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Background and Aim: This long-term study aimed to evaluate, patients with non-ischemic cardiomyopathy patients' main predictors of survival by the CHAID (chi-square automatic interaction detection) algorithm. CHAID algorithm is a composite automatic analysis of non-dependent variables according to dependent variables. This algorithm uses chi-square for categorical and F-test for scale variables. Higher probability variables classify patients according to the dependent variable.

Methods: Between 1997 and 2020, eight hundred fifty-nine non-ischemic cardiomyopathy patients were included in the study. Primary end-points were challenging and consisted of heart replacement therapy or all-cause mortality.

Results: Patients' mean age was 42 ± 13 years, 33.8% were female, and the mean LVEF was 31.7 ± 14.3%. Non-ischemic cardiomyopathy patients 88.4% were DCM, 6.5% were HCM, 1.2% were ARCM and 1.6% were RCM. The primary end-points have occurred in 333 (38.8%) patients at a median follow-up of 3.96 years. Kaplan Meier's survival analysis showed that non-ischemic cardiomyopathy patients' primary end-point free five survival was 66%, and ten years survival at 35% - Figure 1. CHAID algorithm divided non-ischemic cardiomyopathy patients into 16 nodes and three branch depths. The main driver of the prognosis was loop diuretic usage in non-ischemic cardiomyopathy patients. The sub-category drivers were left ventricle ejection fraction (LVEF), NYHA functional class (NYHA FC), systolic pulmonary arterial pressure (SPAP), and antiplatelet usage -Figure 2. For patients who did not need loop diuretics, LVEF classifies patients into two groups. LVEF under 24% had a 38% probability of MACE, and ¬LVEF over 24% were divided into two groups by SPAP. SPAP over 30 mm Hg had a 19.4% probability of MACE. Loop diuretic users were classified according to NYHA FC, NYHA FC I to II patients who had ICD or CRT had higher MACE, and NYHA FC III-IV patients, LVEF under 34% patients had 89% probability of MACE. Antiplatelet usage in NYHA FC II to IIII patients was protective. The CHAID algorithm could classify primary end-point occurring non-ischemic cardiomyopathy patients with 61.6% probability and classify non-occurring patients with 87.5% probability.

Conclusions: In non-ischemic cardiomyopathy, patients with CHAID algorithm risk of MACE were analyzed. CHAID algorithm identified the main determinants, and this algorithm was more successful in estimating non-MACE patients.

Figure 1. Non-ischemic cardiomyopathy patients with five and ten years primary end-point survival

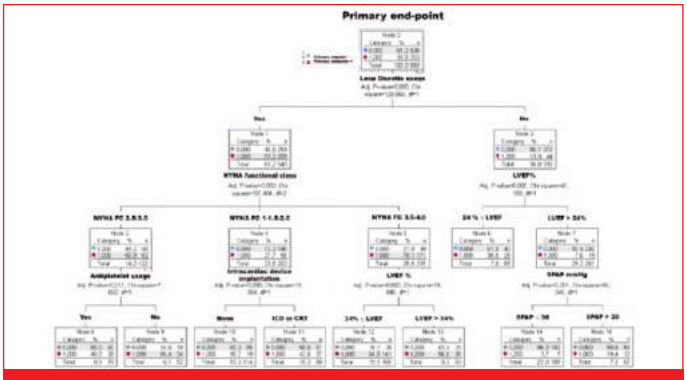

Figure 2. Non-ischemic cardiomyopathy patients CHAID algorithm analyses.

Coronary artery disease / Acute coronary syndrome

OP-037

Serum endocan levels in Minoca syndrome

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Background and Aim: MINOCA syndrome (MI with non-obstructive coronary artery) is a condition, although the diagnostic criteria for myocardial infarction were met, the coronary angiography revealed stenosis of less than 50%. It is believed that endothelial dysfunction is one of the earliest alterations in atherosclerosis and has an important role in the pathophysiology of MINOCA syndrome. Endocan, also known as endothelial cell-specific molecule-1, is a soluble proteoglycan secreted mainly by endothelial cells. The aim of our study was to compare the serum endocan levels of patients diagnosed with MINOCA syndrome with the healty controls.

Methods: 29 MINOCA syndrome patients were enrolled to the study between December 2020 - April 2022 from patients who were performed coronary angiography due to acute coronary syndrome. Of these 29 patients, 12 had myocardial infarction with ST elevation, while 17 had myocardial infarction with non-ST elevation. Serum endocan levels of these patients were measured. 30 Patients who showed similar demographic characteristics such as age, gender, BMI similar to the study group were included in the study as control group. The serum endocan levels were determined as previously reported using an enzyme-linked immunosorbent commercial assay (Aviscera Bioscience, Santa Clara, California).

Results: Endocan levels were significantly higher in patients compared to the healthy control group (1.27 ± 0.36 ng/ml vs. 0.89 ± 0.41 ng/ml, p < 0.001). When we divided the patients with MINOCA syndrome into groups with or without elevation, there was no statistically significant difference.

Conclusions: As far as we know, our study is the first study conducted with the endocan level in MINOCA syndrome. Guidelines indicate that MINOCA is a group of heterogeneous diseases with different mechanisms of pathology. There are studies that shows the endothelial dysfunction plays an important role in the pathophysiology of MINOCA syndrome. In MINOCA syndrome, endocan levels, which are a marker of endothelial dysfunction, are elevated. Large-scale studies are needed to evaluate the role of the endocan level in the diagnosis and prognosis of this disease.

Table 1. Endocan levels of groups

Endocan	Minoca	Healthy group	P value
	1.27 ± 0.36 ng / ml	0.89 ± 0.41 ng / ml	0.001

Table 2. Comparison of demographic characteristics of groups

	Minoca	Healthy control	P value
Age	51.2 ± 12.3	53.1 ± 8.8	0.283
Gender (F/M) (%)	13/16	14/16	0.602
BMI (kg/m^2)	27.7 ± 5.1	27.1 ± 4.8	0.429
Smoking (%)	15 (52%)	15 (50%)	0.464

Coronary artery disease / Acute coronary syndrome

OP-038

Blood pressure recovery ratio after exercise test is associated with SYNTAX score in patients with stable coronary artery disease

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Background and Aim: Previous studies investigating the relation between blood pressure recovery ratio (BPRR) and coronary artery disease(CAD) depended on subjective data. We aimed to investigate the association of BPRR with SYNTAX score (SS) in patients with CAD.

Methods: Among the 142 patients who underwent coronary angiography after the exercise stress test, 98 patients (79 males, 19 females; mean age 57.3 ± 7.3 years) who had at least > 50% occlusion in one coronary artery were included in our study. Patients were divided into two groups as the low SYNTAX score group (SS ≤22) and the intermediate-high SYNTAX score group (SS >23). The BPRR was calculated by dividing the third minute systolic blood pressure (SBP) by the peak exercise SBP.

Results: The BPRR was 0.87 ± 0.06 and 0.96 ± 0.05 in in low and intermediate-high SS group, respectively (p<0.001). EF, lateral e′, E/e′, LA volume, BPRR were correlated with SS.BPRR was negatively correlated with EF and positively correlated with E/e', LA volume, and SS. In multivariate analysis, BPRR (OR: 1.446, p<0.001) and EF (OR: 0.802, p=0.005) were independent predictors of intermediate-high SS. Every 0.01 unit increase in BPRR was associated with a 44.6% increase in the risk of intermediate-high SS. The cut-off value of BPRR obtained by ROC curve analysis was 0.928 for the prediction of intermediate-high SS (sensitivity: 86.1%, specificity: 82.3%). The area under the curve (AUC) was 0.892 (p<0.001).

Conclusions: BPRR, which can be easily obtained during the exercise test, may increase the predictive ability of the exercise test for prediction of the extent of CAD.

Table 1. Comparison of the baseline clinical and demographic features

	Low Syntax Score ≤ 22 n=62	Intermediate-high Syntax Score > 22 n=3	P
Age (years)	56.3 ± 7.8	58.9 ± 6.3	0.100
Gender (Male,%)	51 (82)	28 (78)	0.589
Systolic blood pressure (mm Hg)	132.9 ± 11.8	135.8 ± 13.3	0.270
Diastolic blood pressure (mm Hg)	76.6 ± 8.5	77.1 ± 9.9	0.787
DM (n, %)	32 (51)	23 (63)	0.238
HT (n, %)	43 (69)	31 (86)	0.063
Hyperlipidemia (n, %)	23 (37)	19 (52)	0.130

Table 1. Comparison of the baseline clinical and demographic features (Continued)

	Low Syntax Score ≤ 22 n=62	Intermediate-high Syntax Score > 22 n=3	P
Smoking status (n, %)	26 (42)	17 (47)	0.611
Body mass index (kg/m^2)	28.9 ± 4.4	29.9 ± 4.2	0.083
ACEi or ARB (n, %)	26 (42)	21 (58)	0.117
Beta-blockers (n, %)	21 (33)	8 (22)	0.223
Asetilsalisilikasit (n, %)	27 (43)	8 (22)	0.034
Statin (n, %)	17 (27)	16 (44)	0.086
Calcium channel blocker (n, %)	12 (19)	9 (25)	0.511
Ejection Fraction (%)	58.8 ± 4.0	54.7 ± 4.0	<0.001
LVEDD (mm)	48.5 ± 1.6	50.0 ± 1.2	0.002
E wave deceleration time (s)	182.0 ± 28.0	183.5 ± 21.9	0.784
MV E/A ratio	1.20 ± 0.18	1.24 ± 0.25	0.429
Lateral annular e′velocity (cm/s)	11.5 ± 2.2	10.2 ± 2.3	0.009
Septal annular e′velocity (cm/s)	9.5 ± 1.7	8.6 ± 1.8	0.024
Mitral E/e′ratio	8.8 ± 1.6	10.0 ± 1.9	0.004
Posterior wall thickness (mm)	10.3 ± 1.8	10.9 ± 1.7	0.036
IVS thickness (mm)	10.9 ± 2.5	11.1 ± 1.7	0.142
LA diameter (mm)	31.9 ± 2.8	32.8 ± 2.6	0.045
LA volume (mL)	35.1 ± 5.3	37.3 ± 4.9	0.016
LA volume index (mL / m^2)	23.2 ± 3.6	23.5 ± 2.6	0.487
Myocardial performance index	0.42 ± 0.03	0.42 ± 0.03	0.641
SYNTAX Score	9.6 ± 4.9	32.9 ± 7.6	<0.001
LMCA Disease (n, %)	0 (0)	1 (3)	0.187

LMCA – left main coronary artery; LA - left atrium; LVEDD – Left ventricular end diastolic diameter, IVS - Interventricular septum; MV – mitral valve

Table 2. Comparison of laboratory and exercise stress testing parameters

	Low Syntax Score ≤ 22 n=62	Intermediate-high Syntax Score > 22 n=36	P
Hemoglobin (g/dl)	13.5 ± 1.4	13.0 ± 1.5	0.089
Creatinine (mg/dl)	0.86 ± 0.20	0.95 ± 0.29	0.225
Total cholesterol (mg/dl)	190.5 ± 34.1	197.6 ± 66.1	0.765
LDL cholesterol (mg/dl)	116.2 ± 30.3	121.5 ± 57.6	0.867
HDL cholesterol (mg/dl)	40.8 ± 10.3	38.6 ± 9.5	0.327
Triglycerides (mg/dl)	187.4 ± 109.0	186.9 ± 87.3	0.545
Fasting blood glucose (mg/dl)	138.5 ± 70.2	137.5 ± 56.3	0.693
HsCRP (mg/L)	6.22 ± 6.71	7.34 ± 8.86	0.196
Resting HR (beat/min)	80.5 ± 11.7	81.1 ± 12.8	0.941
Peak HR (beat/min)	139.6 ± 16.4	147.0 ± 15.6	0.011
HR at 1 minute (beat/min)	115.4 ± 16.2	124.5 ± 25.6	0.001
HRR (beat/min)	24.1 ± 10.6	22.5 ± 22.0	0.025
Resting SBP (mm Hg)	134.4 ± 13.0	135.4 ± 15.3	0.728
Peak SBP (mm Hg)	170.9 ± 18.5	175.0 ± 20.2	0.047
SBP at 3 minutes (mm Hg)	149.0 ± 18.9	168.8 ± 19.2	<0.001
BPRR	0.87± 0.06	0.96 ± 0.05	<0.001
Metabolic equivalent	9.86 ± 1.5	9.50 ± 1.4	0.341

LDL - low density lipoprotein; HDL - high density lipoprotein; HsCRP: high sensitive c reactive protein,SBP; systolic blood pressure; BPRR; blood pressure recovery ratio; HR:heart rate;HRR: heart rate recovery

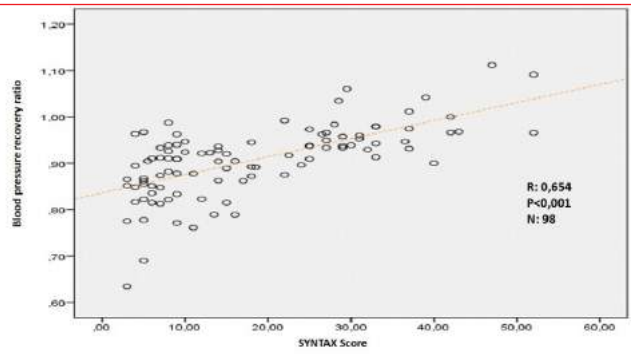

Figure 1. Scatter plot diagram of the relationship between blood pressure recovery ratio and SYNTAX score

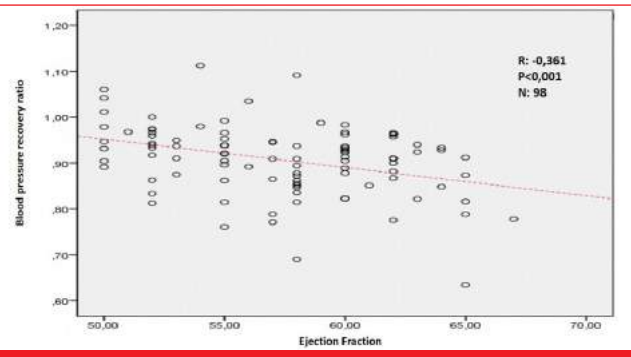

Figure 2. Scatter plot diagram of the relationship between blood pressure recovery ratio and ejection fraction

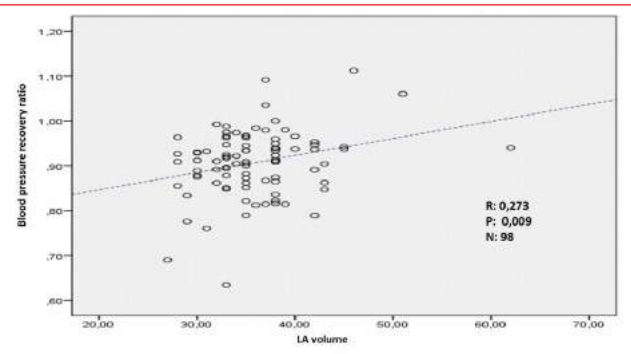

Figure 3. Scatter plot diagram of the relationship between blood pressure recovery ratio and left atrial volume

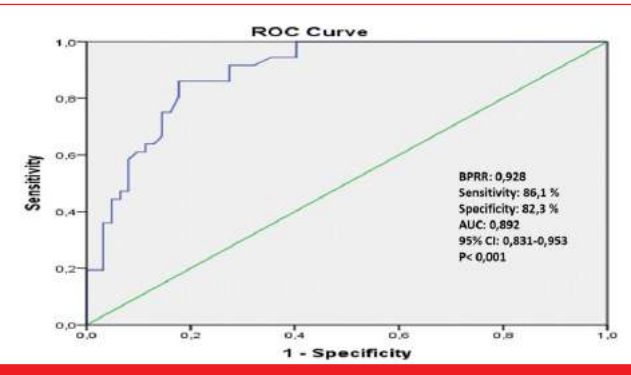

Figure 4. ROC curve analysis to determine predictive value of blood pressure recovery ratio for predicting intermediate-high SYNTAX score

Table 3. The correlation analysis of clinical continuous variables associated with SYNTAX score

	EF	e′ lateral	E/ e′	LA volume	BPRR	HRR	SYNTAX
EF (r)	--	.376***	-.299**	-.185	-.361***	.171	-.482***
MV lateral annular e′ velocity (cm/s) (r)	.376***	--	-.687***	-.132	-.131	.052	-.265**
MV E/e′ (r)	-.299**	-.687***	--	.308**	.245*	.039	.357***
LA volume (r)	-.185	-.132	.308**	--	.273**	-.051	.239*
BPRR (r)	-.361***	-.131	.245*	.273**	--	-.123	.654***
HRR (r)	.171	.052	.039	-.051	-.123	--	-.130
SYNTAX score (r)	-.482***	-.265**	.357***	.239*	.654***	-.130	--

HRR: Heart rate recovery, BPRR: Blood pressure recovery ratio, MV: mitral valve, e′ lateral: mitral valve lateral annular e′ velocity, LA: left atrium. *p<0.05, **p<0.01, ***p<0.001

Coronary artery disease / Acute coronary syndrome

OP-039

The relationship between pre-procedural TIMI flow grade and CHA_2DS_2-VASc score in patients admitted for the first primary percutaneous intervention with acute myocardial infarction and the effect of this relationship on in-hospital mortality

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Background and Aim: Ischemic heart disease is the most common cause of death worldwide. The CHA_2DS_2-VASc score, which includes the main risk factors for coronary artery disease, is a practical scoring system used to determine the risk of stroke and thromboembolism in patients with non-valvular atrial fibrillation (AF). The association of CHA_2DS_2-VASc score with cardiovascular events has been investigated in many studies. However, there is no study in the literature examining the relationship between TIMI flow in the infarct-related artery before primary PCI and the CHA_2DS_2-VASc score. In this study, it was aimed to determine the relationship between TIMI flow grade in the infarct-related artery and CHA_2DS_2-VASc score before primary PCI and the effect of this relationship on in-hospital clinical outcomes in patients presenting with the first diagnosis of acute myocardial infarction clinic.

Methods: This was a crossecitonal, single center, observational study. 750 consecutive patients who met the inclusion criteria were included in the study. Patients were divided into two groups as TIMI 0-1 and TIMI 2-3 according to TIMI flow classification in the artery associated with infarct before the procedure and these groups were compared with CHA_2DS_2-VASc score. In addition, the initial TIMI flow rate and CHA_2DS_2-VASc score were compared with the post-procedure TIMI flow rate. Also, the effect of these parameters on in-hospital clinical outcomes was investigated. In-hospital clinical outcomes were defined as cardiovascular death, acute stent thrombosis, cerebrovascular event, symptomatic heart failure.

Results: TIMI 0-1 flow was detected in 425 (56.7%) patients, and TIMI 2-3 flow was detected in 325 (43.3%) patients. In TIMI 0-1 group, CHA_2DS_2-VASc score was significantly higher (p<0.001) (Table 1). In a receiver operating characteristic curve analysis, the best cut-off value for CHA_2DS_2-VASc score was 3 in predicting the TIMI flow grade in the infarct-related artery before the procedure (Figure 1). As a result of regression analysis, CHA_2DS_2-VASc score was found to be an independent variable in predicting TIMI 0-1 grade in the infarct-related artery before the procedure (Table 2). Furthermore, diagnosis of STEMI, heart failure, and presence of DM were other independent predictors of impaired TIMI flow before the procedure. There was no statistically significant difference between the TIMI flow groups in terms of in-hospital clinical outcomes. The median CHA_2DS_2-VASc score was 4 in the in-hospital mortality group and CHA_2DS_2-VASc scores were significantly higher in this group (p<0.001). The CHA_2DS_2-VASc score was also an independent predictor of in-hospital mortality.

Conclusions: Based on the high predictive value of the CHA_2DS_2-VASc score in predicting thromboembolism, this score was shown to be an independent predictor of preprocedural TIMI flow and in-hospital mortality. This relationship may have positive effects on the success of the procedure and in-hospital mortality in AMI patients.

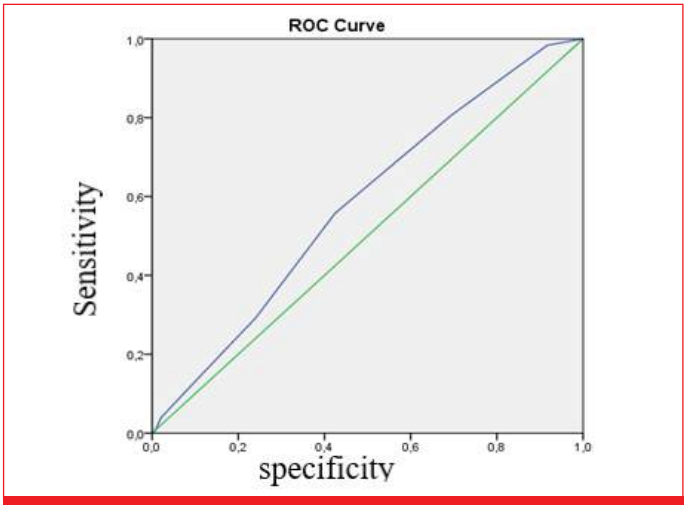

Figure 1. The ROC curve of CHA_2DS_2-VASc score for TIMI flow grade (AUC: 0.581, sensitivity 55.8%, specifity 57.5%)

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

Variables	TIMI 0-1 (n=425)	TIMI 2-3 (n=325)	P value
Age, years	60.3 ± 11.9	60.3 ± 12.1	0.991
Male gender, n (%)	342 (80.5)	245 (75.4)	0.094
DM, n (%)	227 (53.4)	130 (40.0)	<0.001
HT, n (%)	333 (78.4)	238 (73.2)	0.103
HL, n (%)	130 (30.6)	101 (31.1)	0.886
Smoking, n (%)	201 (47.3)	159 (49.2)	0.600
STEMI, n (%)	345 (81.2)	122 (37.5)	<0.001
NSTEMI, n (%)	80 (18.8)	203 (62.5)	<0.001
IRA-LAD, n (%)	192 (45.2)	61 (49.5)	0.048
IRA-CX, n (%)	80 (18.8)	74 (22.8)	0.048
IRA-RCA, n (%)	153 (36.0)	90 (27.7)	0.048
CHA_2DS_2-VASc	3.0 (0-8.0)	2.0 (0-7.0)	<0.001
Troponin, ng/dL	0.71 (0.01-103)	0.28 (0.03-91.8)	<0.001
CK-MB, ng/dL	47.0 (4.3-747.0)	28.5 (9.0-351.0)	<0.001
HDL-C, mg/dL	38.0 (13.0-93.0)	38.0 (14.0-105.0)	0.976
LDL-C, mg/dL	117.0 (30.0-256.0)	115.0 (30.0-229.0)	0.805
Creatinine, mg/dL	0.90 (0.20-6.7)	0.86 (0.42-5.90)	0.041
e-GFR, ml/min/1.73 m^2	88.4 ± 25.7	89.6 ± 23.9	0.545
Glucose, mg/dL	125.0 (69.0-481.0)	106.0 (70.0-399.0)	0.001
WBC,10^3/dL	11.5 (2.6-23.8)	10.2 (4.5-28.9)	<0.001
Hemoglobin, g/dL	14.1 (7.1-20.1)	14.3 (7.8-18.8)	0.123
Platelet count,10^3/dL	236.0 (34.0-997.0)	243.0 (35.0-470.0)	0.293
Lymphocyte, cells/μL	1.80 (0.07-9.8)	1.7 (1.2-2.5)	<0.001
Neutrophils, cells/μL	8.7 (0.86-20.3)	6.8 (2.2-20.7)	<0.001
In-hospital Mortality, n(%)	15 (3.5)	6 (1.8)	0.246
In-hospital CVA, n(%)	6 (1.4)	2 (0.6)	0.477
Acute stent trombosis, n(%)	11 (2.6)	3 (0.9)	0.162
Symptomatic heart failure, n(%)	37 (8.7)	15 (4.6)	0.041
LVEF < 50%	281 (66.1)	136 (41.8)	<0.001

P <0.05 was considered statistical significance. Abbreviations: CK-MB, creatine phosphokinase isoenzyme MB; CX, circumflex artery; CVA, cerebrovascular accident; DM, diabetes mellitus; e-GFR, estimated glomerular filtration rate; HDL-C, high-density lipoprotein cholesterol; HT, hypertension; HL, hyperlipidemia; IRA, infarct related artery; LDL-C, low-density lipoprotein cholesterol; LAD, left anterior desending artery; NSTEMI, non-ST elevation myocardial infarction; RCA, right coronary artery; STEMI, ST elevation myocardial infarction; WBC, white blood cell

Table 2. Logistic Regression Analysis of the parameters predicting TIMI 0-1 flow before PCI

	OR (95% CI)	Univariate Logistic Regression P value	Adjusted OR (95% CI)	Multivariate Logistic Regression P value
CHA_2DS_2-VASc	1.708 (1.276-2.287)	<0.001	1.502 (1.105-2.041)	0.009
Troponin T	1.065 (1.030-1.101)	<0.001	1.049 (1.019-1.079)	0.001
CK-MB	1.009 (1.006-1.012	<0.001		
Glucose	1.003 (1.001-1.006)	0.002		
Creatinine	1.312 (0.865-1.989)	0.202		
Hemoglobin	0.943 (0.876-1.015)	0.117		
WBC	1.105 (1.057-1.154)	<0.001		
Neutrophils	1.166 (1.111-1.223)	<0.001	1.144 (1.089-1.202)	<0.001
Lymphocyte	0.837 (0.736-0.952)	0.007		
3 vessel disease (or 1-2)	1.521 (1.091-2.121)	0.013	1.403 (0.990-1.987)	0.057
Age (<65 age)	1.061 (0.731-1.541)	0.755		
STEMI (or NSTEMI)	7.176 (5.154-9.991)	<0.001	6.299 (4.494-8.830)	<0.001
Gender (men or women)	1.345 (0.950-1.906)	0.095		
LVEF < 50% (or >50)	2.71 (2.01-3.65)	<0.001	2.071 (1.448-2.882)	<0.001
Hypertension	1.323 (0.945-1.853)	0.103		
Diabetes Mellitus	1.720 (1.284-2.304)	<0.001	1.444 (1.038-2.009)	0.029
IRA (or LAD/ Cx)	1.469 (1.074-2.009)	0.016		

CK-MB, creatine phosphokinase isoenzyme MB; IRA, infarct related artery; LDL-C, low-density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; NSTEMI, non-ST elevation myocardial infarction; STEMI, ST elevation myocardial infarction; WBC, white blood cell

Lipid / Preventive cardiology

OP-040

Comparison of serum lipoprotein(a) levels in young and middle-aged patients presenting for the first time with ST-elevation myocardial infarction

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Background and Aim: Lipoprotein(a) [Lp(a)] is associated with coronary artery disease due to its atherogenic and thrombogenic nature.In this study, we aimed to compare the level of Lp(a) in young and middle-aged patients with ST-elevation myocardial infarction (STEMI).

Methods: In this retrospective study included 287 patients aged 20-65 years who presented to the emergency department for the first time due to STEMI. The patients were divided into two groups as 20-45 years (young group, n=111) and 46-65 years (middle-age group, n=176). Both groups were compared in terms of demographic characteristics, comorbidities and laboratory findings.

Results: In the young group, smoking [99 (89.2%) vs. 130 (73.9%), p=0.001), family history [75 (67.6%) vs. 80 (45.5), p<0.001], serum Lp(a) level (38.1 ± 27.9 vs. 23.5 ± 23.2 mg/dl, p<0.001), triglyceride (219.1 ± 231.9 vs. 170.2 ± 105.6 mg/dl, p=0.018), ejection fraction (52.4 ± 6.1 vs. 47.2 ± 7.7, p=0.004) and single vessel disease [83 (74.8%) vs. 110 (62.5%), p=0.031] were higher than middle-age group. In multivaribale logistic regression analyses, family history (OR:2.073, 95% CI [1.210-3.549], p=0.008), low HDL-C level (OR:1.032, 95% CI [1.003-1.062], p=0.029), Lp(a) elevation (OR:1.981, 95% CI [1.871-3.991], p<0.001) were a possible independent risk factor for STEMI in young patients.

Conclusions: Lp(a) level was found to be a higher and possible independent risk factor in young patients who presented with STEMI for the first time, compared to the middle-aged patient group. Lp(a) is a highly atherogenic molecule and it has been associated with stroke, heart failure, aortic stenosis, as well as coronary artery disease. Measurement of Lp(a) level is maybe recommended in young patients with high cardiovascular risk.

Arrhytmia / Electrophysiology / Pacemaker / CRT-ICD

OP-042

Predictive value of supraventricular short runs for new-onset atrial fibrillation in patients with ischemic stroke

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Background and Aim: The clinical importance of supraventricular run (SVR) is uncertain in the management of patients with previous cerebrovascular events. We aim to evaluate the role of SVRs in the development of future AF in patients diagnosed with ischemic stroke. A total of 694 patients were included in the analysis. SVRs was detected in 104 (14.9%) patients in the study group. Seventy-one (10.2%) patients were diagnosed with atrial fibrillation in the follow-up. SVRs were more prevalent among patients with AF (p<0.001). The median atrial run duration was 5.96 (2.02-17.84) seconds in AF absent group vs. 8.76(3.78-17.62) seconds in AF present group (p<0.001). Best predictive cut-off duration of an atrial run was 8 seconds (sensitivity = 61.5% and specificity= 74.4%, AUC = 0.708). Age (OR: 1.03, 95% CI: 1.00-1.060, p=0.008), presence of short atrial run (OR: 0.49, 95% CI 0.28-0.870, p=0.015) and left atrial diameter (OR: 1.13 95% CI: 1.07-1.19, p<0.001) were the independent predictors of AF development in the follow-up.

Methods: We retrospectively evaluated patients who underwent 24-hour Holter monitoring for the evaluation of possible AF after ischemic cerebrovascular events. The presence and their duration of SVR was noted. Subsequent diagnosis of AF was searched in patients with sinus rhythm

Results: A total of 694 patients were included in the analysis. SVRs was detected in 104 (14.9%) patients in the study group. Seventy-one (10.2%) patients were diagnosed with atrial fibrillation in the follow-up. SVRs were more prevalent among patients with AF (p<0.001). The median atrial run duration was 5.96 (2.02-17.84) seconds in AF absent group vs. 8.76(3.78-17.62) seconds in AF present group (p<0.001). Best predictive cut-off duration of an atrial run was 8 seconds (sensitivity = 61.5 % and specificity= 74.4%, AUC = 0.708). Age (OR: 1.03, 95% CI: 1.00-1.060, p=0.008), presence of short atrial run (OR: 0.49, 95% CI 0.28-0.870, p=0.015) and left atrial diameter (OR:1.13 95% CI: 1.07-1.19, p<0.001) were the independent predictors of AF development in the follow-up.

Conclusions: Age, left atrial diameter and the presence of SVRs are associated with an increased risk of future AF after ischemic stroke. SVR duration may be an important parameter in risk stratification. Close rhythm monitoring should be implemented when SVRs are detected.

Table 1. Multivariate logistic regression analysis for the detection of future atrial fibrillation episodes

Table 1. Multivariate logistic regression analysis for the detection of future atrial fibrillation episodes.			
Parameters	Multivariate		
	OR	CI %95	p
Presence of SVR	0.49	0.28-0.87	0.015*
Age	1.03	1.00-1.06	0.008*
CHA_2DS_2-VASc score	1.10	0.91-1.31	0.301
HF	1.28	0.37-4.38	0.691
Coronary Artery Disease	0.72	0.37-1.39	0.330
LVEF	1.00	0.95-1.05	0.842
Left Atrial Diameter	1.13	1.07-1.19	<0.001*

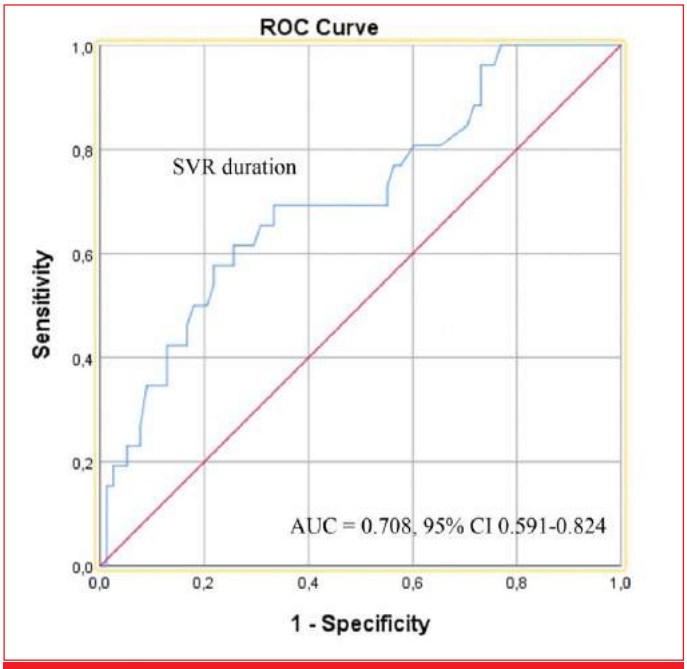

Figure 1. Receiver operating characteristic(ROC) curve of supraventricular run (SVR) duration

Arrhytmia / Electrophysiology / Pacemaker / CRT-ICD

OP-043

An easy measurement to predict the left vs. right premature ventricular contractions: CS distal delay interval

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Background and Aim: We aimed to detect the predictive value of CS distal delay interval for differentiating the left from right sided PVCs.

Methods: We included 137 patients with symptomatic frequent premature ventricular contractions (PVCs) who underwent successful catheter ablation retrospectively (67 male, 70 female; mean age 46.0 ± 16.2 years). Patients were classified into two groups as left sided and right sided PVCs. Decapolar catheters were placed in the coronary sinus before the procedure. CS distal delay interval (Q-CSd) was measured as the interval from onset of earliest QRS complex of premature ventricular contractions in 12 lead ECG to distal CS EGM signal.

Results: CS distal delay interval was found to be significantly lower in left sided PVCs. The cutoff value of CS distal delay interval obtained by ROC curve analysis was 48,5 ms for prediction of right sided origin (sensitivity: 91.5%, specificity: 85.9%). The area under the curve (AUC) was 0.911 (p<0.001).

Conclusions: CS distal delay interval is a novel and simple measurement for accurately differentiating the left from right sided PVCs. The use of this simple measurement could be beneficial for decreasing ablation duration, radiation exposure and the number of arterial or venous punctures.

Figure 1. Measurement of the CS distal delay interval. CS distal delay interval was measured as the interval from onset of earliest QRS complex of premature ventricular contractions in 12 lead ECG to distal CS far field ventricle EGM signal

Table 1. Comparison of the baseline parameters

	Left sided and summit n=78	Right sided n=59	P
Age (years)	40.6 ± 15.7	50.1 ± 15.5	0.001
Gender (Male, %)	33 (42.3)	34 (57.6)	0.076
Hemoglobin (g/dl)	13.2 ± 1.6	13.6 ± 1.4	0.235
Creatinine (mg/dl)	0.73 ± 0.28	0.69 ± 0.17	0.396
LV ejection fraction (%)	54.0 ± 12.9	60.0 ± 7.3	0.033
QRS duration (ms)	163.5 ± 23.0	167.0 ± 23.5	0.402
Maximum deflection index (ms)	94.7 ± 20.7	90.4 ± 14.9	0.109
Q-CS distal interval (ms)	31.6 ± 20.5	69.2 ± 20.7	<0.001
PVC burden (%)	19.0 ± 10.5	18.8 ± 9.2	0.950
TICMP, n (%)	8 (10.3)	1 (1.7)	0.077
Treated with 3D Mapping n (%)	77 (98.7)	59 (100.0)	0.383
Coronary artery disease n (%)	12 (15.9)	6 (10.2)	0.371
Beta blockers, n (%)	47 (60.3)	28 (47.5)	0.136
Amiodarone, n (%)	10 (12.8)	4 (6.8)	0.248
Calcium channel blockers, n (%)	8 (10.3)	4 (6.8)	0.476
Propafenone, n (%)	4 (5.1)	3 (5.1)	0.991

LV, left ventricular, TICMP, tachycardia induced cardiomyopathy

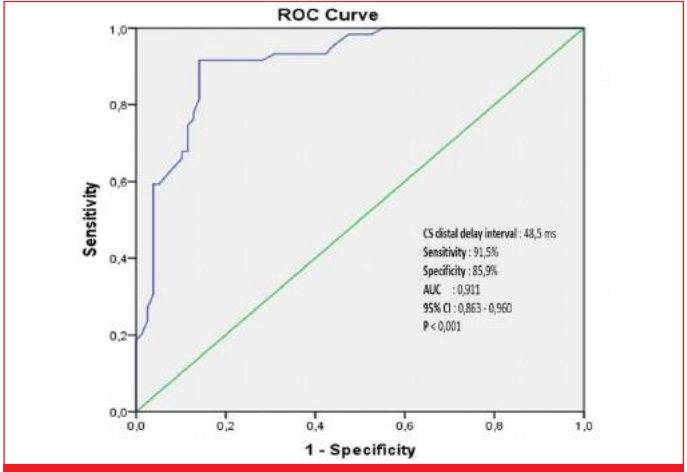

Figure 2. ROC curve analysis to determine predictive value of CS distal delay interval for differentiating the left from right sided PVCs

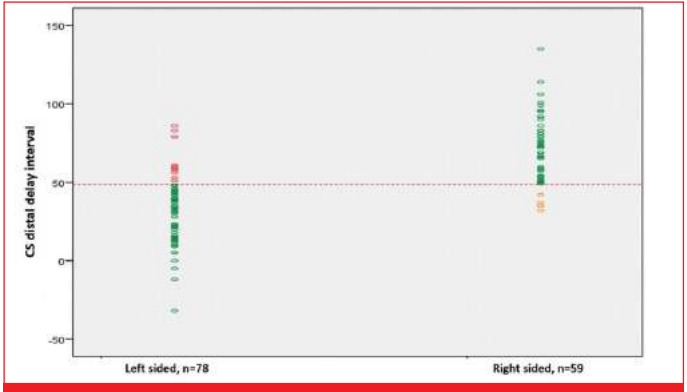

Figure 3. Scatter plot diagram CS distal delay interval for the left and right sided PVCs

Arrhytmia / Electrophysiology / Pacemaker / CRT-ICD

OP-044

Characteristics of patients with atrial fibrillation on edoxaban treatment in Turkey: Does health provider type matter? A report from ETAF-TR Study

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Background and Aim: Safety and effectiveness of edoxaban was demonstrated in a phase III trial and is being confirmed in the post-authorization Edoxaban Treatment in routiNe clinical prActice for patients with Atrial Fibrillation in Europe (ETNA-AF-Europe) study in patients with atrial fibrillation (AF). The Evaluation of Treatment Safety in Patients with Atrial Fibrillation on Edoxaban Therapy in Real-Life in Turkey (ETAF-TR) study is designed to evaluate the safety and effectiveness of edoxaban treatment in AF in routine practice. This sub-study evaluates the baseline demographic, clinical, and laboratory characteristics of ETAF-TR Study according to the health provider type.

Methods: The ETAF-TR study (NCT04594915) is a national, multicenter, prospective, observational study that included 1053 cases from 50 centers. The primary outcome of the study is any overt bleeding. Enrollment process had been completed in May 2022. The overall duration of follow-up will be 1 year. Enrolled patients divided to two groups according to the health provider type as state and private health provider.

Results: Mean age was 70.1 years and 59% were female. Mean CHA2DS2VASc and HASBLED scores were 3.5 and 1.6, respectively. Baseline demographic, clinical, and laboratory characteristics of study population summarized in Table. Majority of patients (824 of 1053 pts, 78.2%) were enrolled by state hospitals. Patients who enrolled by state providers were younger than private providers (69.2 ± 11.0 vs. 73.2 ± 11.3 years, respectively, p<0.001) and had lower risk of stroke (mean CHA2DS2VASc score: 3.4 ± 1.5 vs. 3.9 ± 1.4, respectively, p<0.001). Dramatically, inapproprite low dose prescription of edoxaban (improper usage of edoxaban 30 mg od) was higher in private providers than state providers (12.8% vs. 6.6%, respectively, p=0.003).

Conclusions: Edoxaban has been used wide spectrum of patients with AF in daily routine practice with a good overall

adherence to the product label. However bleeding fear may not be underestimated factor for undertreatment, particularly for physicians who working in private providers.

Table 1. The comparison of the socio-demographic profile, clinical features, medical history, and laboratory data of patients according to the health provider type

Variable	Overall (N = 1053)	State Hospital (N = 824)	Private Hospital (N = 229)	*P* – value
Socio-demographics				
• Age, mean ± SD, years	70.1 ± 11.2	69.2 ± 11.0	73.2 ± 11.3	<0.001
• Age group, n (%)				
▪ <65 years	265 (25.2)	223 (27.1)	42 (18.3)	<0.001
▪ 65 – 74 years	397 (37.7)	325 (39.4)	72 (31.4)	
▪ ≥75 years	391 (37.1)	276 (33.5)	115 (50.2)	
• Female sex, n (%)	621 (59.0)	481 (58.4)	140 (61.1)	0.452
• Education level, n (%)				
▪ Not educated or primary school	711 (67.5)	599 (72.7)	112 (48.9)	<0.001
▪ Secondary school	284 (27.0)	189 (22.9)	95 (41.5)	
▪ High school	58 (5.5)	36 (4.4)	22 (9.6)	
Physical examination				
• BMI, mean ± SD, kg/m^2	29.1 ± 5.3	29.2 ± 5.4	28.5 ± 5.2	0.064
• Body weight, n (%)				
▪ <90 kg	948 (90.0)	751 (91.1)	197 (86.0)	0.022
▪ ≥90 kg	105 (10.0)	73 (8.9)	32 (14.0)	
• Systolic BP, mean ± SD, mmHg	130 ± 17	129 ± 17	133 ± 18	<0.001
• Diastolic BP, mean ± SD, mmHg	77 ± 11	77 ± 11	77 ± 11	0.783
AF-related information				
• AF type, n (%)				
▪ Paroxysmal AF	311 (29.5)	203 (24.6)	108 (47.2)	<0.001
▪ Persistent AF	82 (7.8)	72 (8.7)	10 (4.4)	
▪ Long-standing persistent AF	59 (5.6)	53 (6.4)	6 (2.6)	
▪ Permanent AF	601 (57.1)	496 (60.2)	105 (45.9)	
• CHA_2DS_2-VASc score, mean ± SD	3.5 ± 1.5	3.4 ± 1.5	3.9 ± 1.4	<0.001
• HAS-BLED score, mean ± SD	1.6 ± 0.9	1.6 ± 0.9	1.5 ± 1.0	0.530
• History of cardioversion, n (%)	74 (7.7)	55 (7.3)	19 (9.0)	0.407
• History of AF ablation procedure, n (%)	21 (2.1)	20 (2.5)	1 (0.4)	0.053
Medical history				
• Previous stroke and/or TIA, n (%)	139 (13.2)	106 (12.9)	33 (14.4)	0.540
• Coronary artery disease, n (%)	303 (31.3)	236 (31.3)	67 (31.2)	0.969
• Congestive heart failure, n (%)	305 (29.4)	244 (30.0)	61 (27.0)	0.372
• Hypertension, n (%)	748 (71.0)	580 (70.4)	168 (73.4)	0.380
• Diabetes mellitus, n (%)	282 (26.8)	226 (27.4)	56 (24.5)	0.368
• Dyslipidemia, n (%)	194 (18.9)	136 (17.0)	58 (25.4)	0.004
• Peripheral artery disease, n (%)	33 (3.4)	19 (2.5)	14 (6.3)	0.006
• History of cancer, n (%)	40 (3.9)	36 (4.5)	4 (1.8)	0.062
• Chronic gastrointestinal disease, n (%)	139 (14.2)	120 (15.8)	19 (8.8)	0.009
• Previous bleeding history, n (%)	90 (8.5)	72 (8.7)	18 (7.9)	0.674
• Usage of antiplatelet therapy	118 (11.2)	93 (11.3)	25 (10.9)	0.875
• Current smoker, n (%)	71 (6.7)	55 (6.7)	16 (7.0)	0.107
Laboratory data				
• Serum creatinine, mean ± SD, mg/dL	0.9 ± 0.3	1.0 ± 0.3	0.9 ± 0.2	0.905
• CrCl mean ± SD, mL/min	80 ± 31	82 ± 32	74 ± 28	0.001
• CrCl 15–50 mL/min, n (%)	174 (16.5)	126 (15.3)	48 (21.0)	0.041
• Hemoglobin, mean ± SD, mg/dL	12.9 ± 1.8	13.0 ± 1.9	12.8 ± 1.6	0.138
• FBG, mean ± SD, mg/dL	120 ± 44	121 ± 44	116 ± 44	0.065
Appropriate or inappropriate dose prescription [available data for 1015 patients]**				
• Appropriate dose, n (%)	834 (82.2)	659 (83.5)	175 (77.4)	0.035
• Inappropriate low dose, n (%)	81 (8.0)	52 (6.6)	29 (12.8)	0.002
• Inappropriate high dose, n (%)	100 (9.9)	78 (9.9)	22 (9.7)	0.946

SD, standard deviation; BMI, body mass index; BP, blood pressure; AF, atrial fibrillation; TIA, transient ischemic attack; CrCl, creatinine clearance; FBG, fasting blood glucose.

Arrhytmia / Electrophysiology / Pacemaker / CRT-ICD

OP-045

Demographic, clinical, and disease characteristics of patients with atrial fibrillation on edoxaban therapy according to the stroke risk: A report from evaluation of treatment safety in patients with atrial fibrillation on edoxaban therapy in real-life data

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Background and Aim: In our study, we aimed to define the ischemic risk of the cases under edoxaban treatment in Turkey and the factors affecting the ischemic risk by means of the CHADsVASc score.

Methods: The ETAF-TR study (NCT04594915) is a national, multicenter, prospective, observational study that included 1053 cases from 50 centres. Study inclusion criteria; being over the age of 18, being under edoxaban treatment, and patients giving consent were accepted. It aims to complete the ENGAGE-AF study, in which the efficacy of edoxaban has been proven, with real-life data. In our study, the ischemic risks of the patients in the ETAF-TR study were classified by the CHADsVASc score. CHADsVASc were defined as C Congestive heart failure, H Hypertension, A>75 years (2 points), D diabetes, S stroke (2 points), V vascular disease, A 65-74 years, Sc gender. The cases were classified as low [0 (male)-1 (female) score], moderate [1 (male)-2 (female) score] and high [≥ 2 (male)-≥ 3 (female) points] risk using the CHADsVASc score, according to the ESC 2020 atrial fibrillation diagnosis and menagement guideline recommendations. The characteristics of the cases between the groups were evaluated, including their previous ischemic history, HASBLED scores, drug doses, GFR values, and bleeding tendency.

Results: The mean CHADsVASc score of the population was 3.5 (1.5), while the mean score between the groups was 0.7 (0.5), 1.6 (0.5), 3.9 (1.3) in the low-intermediate and high-risk groups, respectively. The median CHADSVASC score was 3 (2.0-4.0) in the whole population and 1.0 (0.0, 1.0), 2.0 (1.0, 2.0), 4.0 (3.0, 5.0) between the groups, respectively. The distribution of cases according to risk groups was determined as 3.4% (n=36) (low risk), 13.2% (n=139) (medium risk group), 83.3% (n=878) (high risk group). The use of 60 mg edoxaban was 91.7% (n=33) in the low-risk group, 92.8% (n=129) in the intermediate-risk group, and 77.6% (n=681) in the high-risk group. In the high-risk group, 39.3% (n=345) of the cases aged 65-74 years, 44.5% (n=391) of the cases over 75 years of age are included, and 58.7% (n=515) of the high-risk group consists of women. In the high-risk group, the frequencies of congestive heart failure, hypertension, diabetes, and stroke were defined 32.8% (n=288), 82.9% (n=728), 39.3% (n=271), 15.8% (n=139), 27.9% (n=245) respectively. All of the cases (n=26) with a high HASBLED score are in the high-risk group according to the CHADsVASc score. Among all cases, thromboembolic events were detected in 14.2% (n=150), ischemic stroke in 10.6% (n=112), pulmonary embolism in 0.4% (n=4), and dvt in 0.8% (n=8) patients, while the majority of these cases were in the high-risk group [respectively 16.9% (n=148), 12.8% (n=112), 0.3% (n=3), 0.8 (n=7)].

Conclusions: Our results, in the light of follow-up data, will guide the evaluation of the efficacy of edoxaban according to the risk of ischemic events in cases under edoxaban treatment in Turkey.

Table 1. CHADSVASC score and parameters

	All. (n=1053)	Low (n=36)	Intermediate (n=139)	High (n=878)
CHADSVASC (median and IQR)	3.0 (2.0, 4.0)	1.0 (0.0, 1.0)	2.0 (1.0, 2.0)	4.0 (3.0, 5.0)
CHADSVASC (mean and std)	3.5 (1.5)	0.7 (0.5)	1.6 (0.5)	3.9 (1.3)
Congestive heart failure	305 (29.0%)	1 (2.8%)	16 (11.5%)	288 (32.8%)
Hypertension	805 (76.4%)	6 (16.7%)	71 (51.1%)	728 (82.9%)
Age <65	265 (25.2%)	36 (100.0%)	87 (62.6%)	142 (16.2%)
Age 65-74	397 (37.7%)		52 (37.4%)	345 (39.3%)
Age >=75	391 (37.1%)			391 (44.5%)
Age >=85	72 (6.8%)			72 (8.2%)
Diabetes Mellitus	282 (26.8%)	1 (2.8%)	10 (7.2%)	271 (30.9%)
Stroke	139 (13.2%)			139 (15.8%)
Vascular disease	253 (24.0%)		8 (5.8%)	245 (27.9%)
Female	621 (59.0%)	24 (66.7%)	82 (59.0%)	515 (58.7%)
Male	432 (41.0%)	12 (33.3%)	57 (41.0%)	363 (41.3%)

Table 2. Ischemic history

	All (n=1053)	Low (n=36)	Intermediate (n=139)	High (n=878)
Thromboembolic event	150 (14.2%)		2 (1.4%)	148 (16.9%)
Ischemic stroke	112 (10.6%)			112 (12.8%)
Pulmonary embolism	4 (0.4%)		1 (0.7%)	3 (0.3%)
Deep vein thrombosis	8 (0.8%)		1 (0.7%)	7 (0.8%)

Table 3. Edoxaban dose end HASBLED parameters

	All (n=1053)	Low (n=36)	Intermediate (n=139)	High (n=878)
Edoxaban dose 30 mg	210 (19.9%)	3 (8.3%)	10 (7.2%)	197 (22.4%)
Edoxaban dose 60 mg	843 (80.1%)	33 (91.7%)	129 (92.8%)	681 (77.6%)
Creatinine clearance 15-30	17 (1.6%)			17 (1.9%)
30-50	157 (14.9%)	1 (2.8%)	3 (2.2%)	153 (17.4%)
50-80	388 (36.8%)	3 (8.3%)	32 (23.0%)	353 (40.2%)
>80	491 (46.6%)	32 (88.9%)	104 (74.8%)	355 (40.4%)

Table 3. Edoxaban dose end HASBLED parameters (Continued)

	All (n=1053)	Low (n=36)	Intermediate (n=139)	High (n=878)
HASBLED low risk	537 (51.0%)	34 (94.4%)	119 (85.6%)	384 (43.7%)
HASBLED intermediate risk	490 (46.5%)	2 (5.6%)	20 (14.4%)	468 (53.3%)
HASBLED high risk	26 (2.5%)			26 (3.0%)
Uncontrolled hypertension	118 (11.2%)		9 (6.5%)	109 (12.4%)
Abnormal renal function	86 (8.2%)	1 (2.8%)	3 (2.2%)	82 (9.3%)
Abnormal hepatic function	5 (0.5%)			5 (0.6%)
Bleeding	75 (7.1%)	1 (2.8%)	5 (3.6%)	69 (7.9%)
Major bleeding	18 (1.7%)		1 (0.7%)	17 (1.9%)
Intracranial bleeding	4 (0.4%)		1 (0.7%)	3 (0.3%)
Labil INR (TTR < 60%)	111 (10.5%)	1 (2.8%)	9 (6.5%)	101 (11.5%)
Drugs and alcohol	271 (25.7%)	3 (8.3%)	28 (20.1%)	240 (27.3%)
Antiplatelet	118 (11.2%)	1 (2.8%)	7 (5.0%)	110 (12.5%)
Alcohol 1/week	32 (3.0%)		6 (4.3%)	26 (3.0%)
Alcohol 1-7 week	12 (1.1%)		2 (1.4%)	10 (1.1%)

Table 4. AF history and laboratory parameters

	All. (n=1053)	Low (n=36)	Intermediate (n=139)	High (n=878)
Paroxysmal	311 (29.5%)	16 (44.4%)	49 (35.3%)	246 (28.0%)
Persistent	82 (7.8%)	1 (2.8%)	14 (10.1%)	67 (7.6%)
Long standing persistent	59 (5.6%)	1 (2.8%)	12 (8.6%)	46 (5.2%)
Permanent	601 (57.1%)	18 (50.0%)	64 (46.0%)	519 (59.1%)
Left atrial appendage closure	1 (0.1%)			1 (0.1%)
Cancer	40 (3.8%)		4 (2.9%)	36 (4.1%)
HB (g/dL)	12.9 (1.9)	13.0 (2.0)	13.4 (1.8)	12.8 (1.9)
PLT (mm^3)	241484.9 (73298.8)	245111.1 (62353.3)	243744.6 (66628.5)	240978.5 (74762.9)

Arrhytmia / Electrophysiology / Pacemaker / CRT-ICD

OP-046

PrevAleNce and Associated factors of inappropriaTe dosing of direct Oral anticoaguLants In pAtients with Atrial Fibrillation: The ANATOLIA-AF Study

Umut Kocabaş[1], Işıl Ergin[2], Veysel Yavuz[3], Selda Murat[4], İbrahim Özdemir[5], Ömer Genç[6], Cihan Altın[7], Haşim Tüner[8], Bengisu Keskin Meriç[9], Ali Çoner[10], Sinem Çakal[11], Sercan Çayırlı[12], Arda Güler[13], Dilay Karabulut[14], Onur Dalgıç[15], Osman Uzman[16], Bektaş Murat[17], Şeyda Şahin[18], Umut Karabulut[19], Tarık Kıvrak[20], Muharrem Said Coşgun[21], Ferhat Özyurtlu[22], Mehmet Kaplan[23], Emre Özçalık[1]

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Background and Aim: Inappropriate dosing of direct oral anticoagulants is associated with an increased risk of stroke, systemic embolism, major bleeding, cardiovascular hospitalization, and death in patients with atrial fibrillation. The main goal of the study was to determine the prevalence and associated factors of inappropriate dosing of direct oral anticoagulants in real-life settings.

Methods: This study was a multicenter, cross-sectional, observational study that included 2,004 patients with atrial fibrillation. The study population was recruited from 41 cardiology outpatient clinics between January and May 2021. The main criteria for inappropriate direct oral anticoagulant dosing were defined according to the recommendations of the European Heart Rhythm Association (Table 1).

Results: The median age of the study population was 72 years and 58% were women. Nine hundred and eighty-seven patients were prescribed rivaroxaban, 658 apixaban, 239 edoxaban, and 120 dabigatran (Figure 1). A total of 498 patients (24.9%) did not receive the appropriate dose of direct oral anticoagulants (Figures 2, 3). In a logistic regression model, advanced age, presence of chronic kidney disease, presence of permanent atrial fibrillation, prescription of reduced doses of direct oral anticoagulants, prescription of edoxaban treatment, concomitant use of amiodarone treatment, and non-use of statin treatment were significantly associated with potentially inappropriate dosing of direct oral anticoagulants (Table 2).

Conclusions: The study demonstrated that the prevalence of inappropriate direct oral anticoagulant dosing according to the European Heart Rhythm Association recommendations was 24.9% in patients with atrial fibrillation. Several demographic and clinical factors were associated with the inappropriate prescription of direct oral anticoagulants.

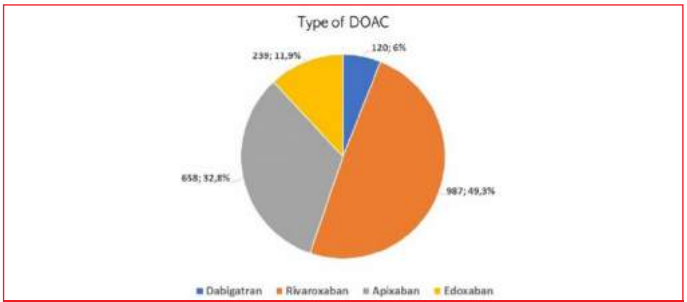

Figure 1. Frequency of direct oral anticoagulants among study population

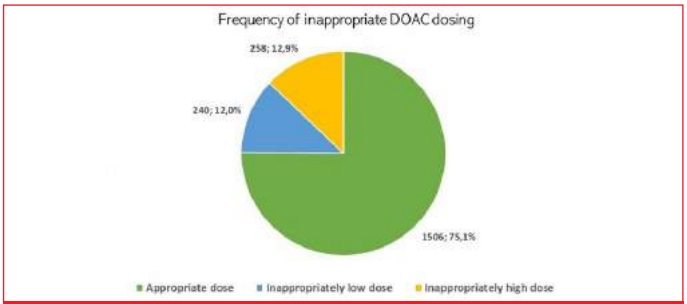

Figure 2. Frequency of appropriate dose of direct oral anticoagulant, inappropriate low dose of direct oral anticoagulant, and inappropriate low dose of direct oral anticoagulant prescription for the entire cohort

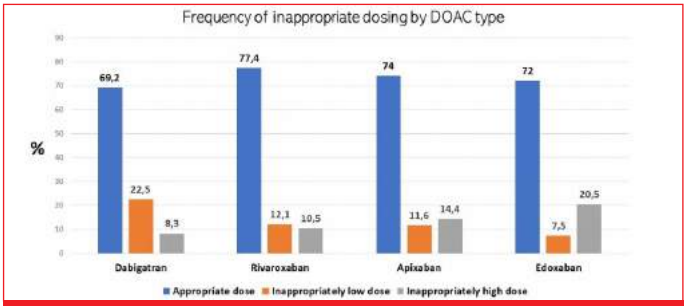

Figure 3. Frequency of inappropriate low and high dosing by each direct oral anticoagulant type

Table 1. Criteria for defining a patient as requiring a reduced dose of direct oral anticoagulants in the ANATOLIA-AF study

For patients receiving dabigatran
• Age ≥80 years • Concomitant usage of verapamil • ≥2 of the following criteria: age 75–79 years, GFR 30 – 50 mL/min/1.73 m², HAS-BLED score ≥3, concomitant usage of antiplatelet therapy, concomitant usage of amiodarone, or body weight ≤60 kg
For patients receiving rivaroxaban
• GFR 15 – 49 mL/min/1.73 m² • ≥2 of the following criteria: age ≥75 years, HAS-BLED score ≥3, concomitant usage of antiplatelet therapy, concomitant usage of amiodarone, or body weight ≤60 kg
For patients receiving apixaban
• ≥2 of the following criteria: age ≥80 years, serum creatinine ≥1.5 mg/dL, or body weight ≤60kg • GFR 15 – 29 mL/min/1.73 m² • ≥2 of the following criteria: age ≥75 years, HAS-BLED score ≥3, concomitant usage of antiplatelet therapy, concomitant usage of amiodarone, or concomitant usage of diltiazem
For patients receiving edoxaban
• GFR 15 – 49 mL/min/1.73 m² • Body weight ≤60kg • Concomitant usage of strong P-glycoprotein inhibitor (e.g. dronedarone, ketoconazole, erythromycin) • ≥2 of the following criteria: age ≥75 years, HAS-BLED score ≥3, concomitant usage of antiplatelet therapy, concomitant usage of amiodarone, or concomitant usage of verapamil
GFR = glomerular filtration rate; HAS-BLED = uncontrolled hypertension, abnormal renal and liver function (1 point each), stroke, bleeding, labile international normalized ratios, elderly (age >65 years), drugs or alcohol (1 point each) (concomitant use of antiplatelet agents or non-steroidal anti-inflammatory drugs, alcohol abuse).

Table 2. Unadjusted variables and age-adjusted logistic regression analysis/models of factors associated with inappropriate dose of direct anticoagulants

	Unadjusted OR (95% CI)	Model 1[†] OR (95% CI)	Model 2[‡] OR (95% CI)
Demographic features			
Age			
• <65 years (reference group)			
• 65 – 74 years	2.07 (1.44-2.96) **	2.02 (1.41-2.90) **	1.57 (1.03-2.39) *
• ≥75 years	4.40 (3.12-6.20) **	4.16 (2.92-5.93) **	2.32 (1.47-3.65) **
Marital status			
• Married (reference group)			
• Single or widowed or divorced	1.61 (1.30-1.99) **	1.16 (0.92-1.45)	1.05 (0.81-1.37)
		Nagelkerke R2 = 0.075	
Medical history and laboratory findings			
BMI, kg/m²	0.97 (0.95-0.99) *	0.99 (0.97-1.01)	0.99 (0.96-1.01)
Presence of CHF	1.49 (1.21-1.83) **	1.28 (1.02-1.60) *	1.08 (0.82-1.42)
Presence of PAD	1.70 (1.05-2.76) *	1.44 (0.84-2.45)	1.46 (0.83-2.58)
Absence of AF ablation procedure history	3.01 (1.37-6.61) *	1.62 (0.71-3.68)	1.90 (0.77-4.67)
History of major and/or CRNM bleeding	1.71 (1.21-2.41) *	1.30 (0.89-1.90)	1.15 (0.77-1.71)
GFR group			
• GFR 15 – 29 mL/min/1.73 m² (reference group)			
• GFR 30 – 59 mL/min/1.73 m²	2.58 (1.13-5.91) *	3.23 (1.40-7.50)*	4.25 (1.79-10.11) **
• GFR ≥60 mL/min/1.73 m²	1.20 (0.56-2.59)	2.39 (1.03-5.56) *	3.73 (1.55-8.99) *
Hemoglobin, mg/dL	0.88 (0.83-0.92) **	0.96 (0.90-1.01)	0.98 (0.92-1.04)
		Nagelkerke R2 = 0.096	
AF-related features			
AF type			
• New-onset AF (reference group)			
• Paroxysmal AF	1.33 (0.68-2.62)	1.44 (0.72-2.87)	1.52 (0.73-3.16)
• Persistent or long-standing persistent AF	1.44 (0.71-2.90)	1.57 (0.67-2.81)	1.60 (0.75-3.44)
• Permanent AF	2.17 (1.12-4.20) *	1.98 (1.01—3.89) *	2.16 (1.06-4.41) *
CHA-DS2-VASc score	1.28 (1.19-1.37) **	1.07 (0.98-1.16)	1.07 (0.96-1.18)
HAS-BLED score	1.43 (1.27-1.59) **	1.12 (0.97-1.28)	1.022 (0.87-1.21)
		Nagelkerke R2 = 0.088	
Treatments			
DOACs			
• Rivaroxaban (reference group)			
• Edoxaban	1.34 (0.97-1.84)	1.50 (1.07-2.12) *	1.66 (1.11-2.31) *
• Dabigatran	1.53 (1.01-2.31) *	1.26 (0.81-1.97)	1.19 (0.75-1.91)
• Apixaban	1.20 (0.96-1.51)	1.25 (0.96-1.57)	1.16 (0.89-1.50)
Dosage of DOAC			
• Standard dose (reference group)			
• Reduced dose	3.48 (2.80-4.31) **	2.68 (2.12-3.40) **	2.54 (1.96-3.29) **
Usage of antiplatelet therapy	1.57 (1.16-2.12) *	1.43 (1.02-2.00) *	1.34 (0.91-1.96)
Usage of amiodarone	1.66 (1.06-2.60) *	2.02 (1.25-3.28) *	2.79 (1.63-4.79) **
Usage of diuretics	1.46 (1.19-1.79) *	1.20 (0.96-1.49)	1.01 (0.78-1.32)
Usage of NSAIDs	1.35 (0.99-1.84)	1.18 (0.84-1.65)	1.20 (0.83-1.75)
Non-use of statins	1.31 (1.01-1.71) *	1.57 (1.18-2.10) *	1.63 (1.19-2.21) *
		Nagelkerke R2 = 0.145	**Nagelkerke R2 = 0.168**

Arrhytmia / Electrophysiology / Pacemaker / CRT-ICD

OP-047

Real-world evaluation Of anticoagulant Treatment patterns in patients with Atrial fibrillation: Data from multicenter ROTA study

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Background and Aim: Oral anticoagulant therapy is the cornerstone of atrial fibrillation (AF) management to prevent stroke and systemic embolism. However, there is limited real-world information regarding stroke and systemic embolism prevention strategies in patients with AF. The aim of the ROTA study is to obtain the real-world data of anticoagulant treatment patterns in patients with AF.

Methods: The ROTA study is a cross-sectional, multicenter, and observational study that included 2,597 patients with AF. The study population was recruited from 41 cardiology outpatient clinics between January 2021 to May 2021.

Results: The median age of the study population was 72 years and 57.4% were female. The median CHA2DS2-VASc and HAS-BLED scores were 4 and 1, respectively. Vitamin K antagonists (VKAs) and direct oral anticoagulants (DOACs) were used in 15.9% and 79.4% of patients, respectively. The mean time-in-therapeutic range (TTR) was 52.9% for patients receiving VKAs, and 76% of those patients had an inadequate TTR with <70%. The most common prescribed DOACs were rivaroxaban (38.1%), apixaban (25.5%), and edoxaban (11.2%). The rate of overuse of VKAs and DOACs was high (76.1%) in patients with a low stroke risk and more than one-fourth of patients on DOAC therapy were receiving a reduced dose of DOACs. Among patients who were on DOAC treatment, patients with apixaban treatment were older, had higher CHA2DS2-VASc and HAS-BLED scores, and had lower creatinine clearance than the patients receiving other DOACs.

Conclusions: The ROTA study provides important real-world information about anticoagulant treatment patterns in patients with AF.

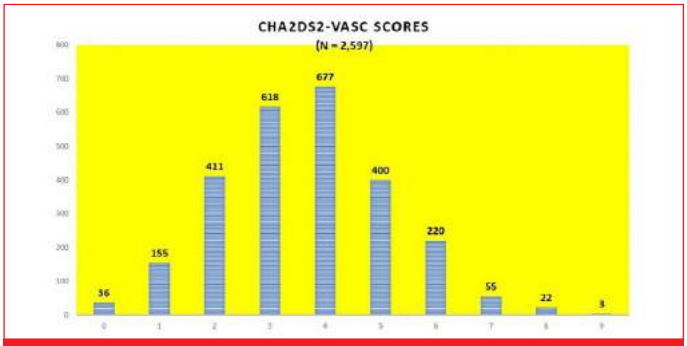

Figure 1. Number of patients according to the CHA2DS2-VASc score

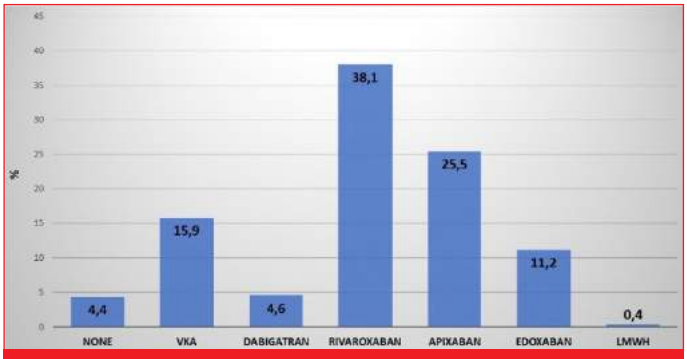

Figure 2. Frequency of anticoagulant treatments among study population

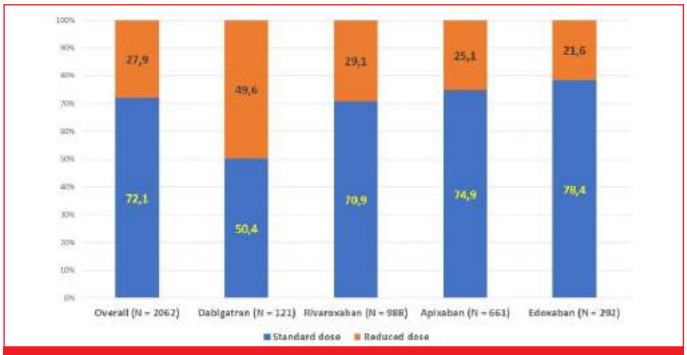

Figure 3. Frequency of standard versus reduced doses of direct oral anticoagulants

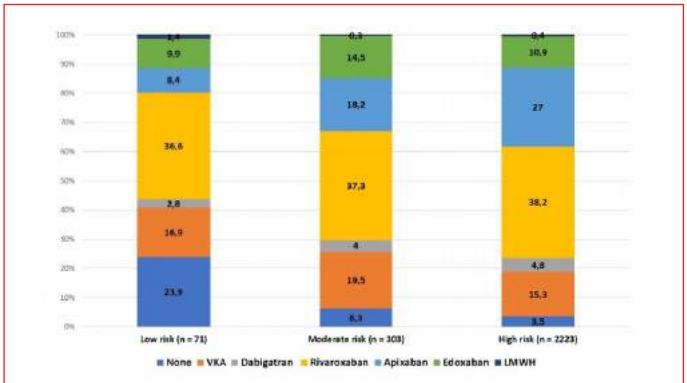

Figure 4. Anticoagulant treatment patterns according to the stroke risk profile

Interventional cardiology / Carotid and peripheral vascular

OP-048

A comprehensive meta-analysis of the literature comparing radial versus femoral access for carotid artery stenting

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Background and Aim: Carotid artery stenting has become an effective therapy option in the treatment of carotid artery stenosis. Although the femoral access is the preferred approach for carotid artery stenting, the radial access is especially preferred to reduce the risk of bleeding and to reduce the length of bed rest. However, no evidence suggests the superiority of these two methods over each other. Thus, we aimed to conduct a meta-analysis that investigates the comparison of radial access versus femoral access in carotid artery stenting.

Methods: We searched PubMed, Google Scholar, and Cochrane libraries for eligible studies. After reviewing all potential studies, this meta-analysis was conducted with remained 9 studies.

Results: In total, 9 studies with 7267 patients (652 in the radial group and 6615 in the femoral group) were enrolled in this meta-analysis (Table 1). There were no differences between the groups with respect to MACCE (composite of myocardial infarction, stroke, and death), fluoroscopy time, procedure time, hospital stay, contrast volume, and vascular complications (Figure 1). The heterogeneities between studies were lower than an acceptable level for outcomes except for fluoroscopy time, in which 96% heterogeneity was observed. The study reported by Gao et al was detected as an outlier and influential study for this outcome. The random-effect model was repeated after removing this study but the heterogeneity did not change meaningfully. The results for fluoroscopy time should be evaluated in future meta-analyses. The comparisons of composites outcomes of cerebellar events including stroke and transient ischemic attack were also similar between the groups.

Conclusions: This meta-analysis showed that there was no superiority of radial or femoral access over each other in carotid artery stenting.

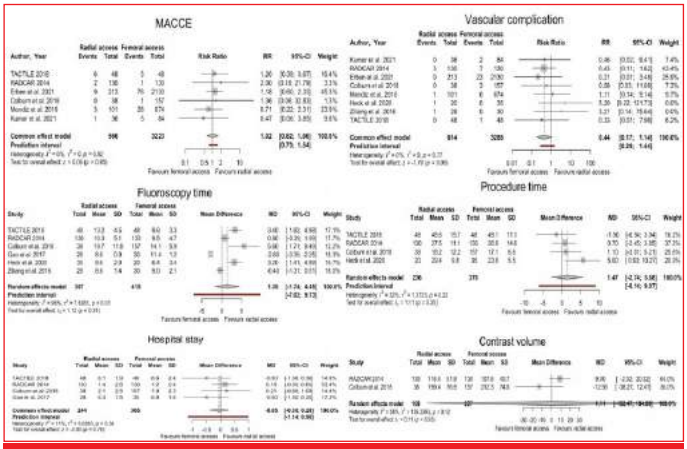

Figure 1. The composite outcomes of MACCE (composite of myocardial infarction, stroke, and death), fluoroscopy time, procedure time, hospital stay, contrast volume, and vascular complications according to radial access versus femoral access

Table 1. All studies included in this meta-analysis

Study	Year	Region	Radial Access (size)	Femoral Access (size)	Age (radial access)	Age (femoral access)	Male (radial access)	Male (femoral access)	Study design
TACTILE	2018	Russian	48	48	64.6(8)	63(6.7)	40(83)	36(75)	RCT
RADCAR	2014	Hungary	130	130	66.8(8.9)	66.7(10.2)	73(60.8)	85(65.4)	RCT
Erben et al.	2021	USA	213	5427	68.6(7.9)	67.6(8.2)	149(69.9)	3372(63.4)	Cohort
Colburn et al.	2018	USA	38	157	29.2(3.9)	29.6(7.1)	31(81.6)	97(61.8)	Cohort
Mendiz et al.	2016	Argentina	101	674	71(10.6)	69.7(9.4)	80(79.2)	478(70.9)	Cohort
Kumar et al.	2021	USA	36	84	NA	NA	NA	NA	Cohort
Gao et al.	2017	China	28	30	72(5.5)	70(6.7)	13(46.2)	14(46.6)	Cohort
Heck et al.	2020	USA	20	35	NA	NA	NA	NA	Cohort
Ziliang et al.	2016	China	38	30	NA	NA	NA	NA	Cohort

Interventional cardiology / Carotid and peripheral vascular

OP-049

The relationship between platelet indices and ischemic brain lesions after carotid artery stenting

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Background and Aim: Carotid artery stenting (CAS) is being increasingly used as an alternative treatment to carotid endarterectomy; however, ischemic brain lesions after CAS remain as a matter of concern. Studies have revealed that many risk factors trigger cerebrovascular events that develop after the procedure. Platelet indices, such as platelet volume (MPV), platelet distribution width (PDW) and plateletcrit (PCT) indicators of platelet activation and shown in previous studies as predictive factors of the atherosclerotic process. Hence, we aimed to investigate the relationship between platelet indices and ischemic brain lesions involving both silent and overt cerebral ischema in patients who underwent carotid artery stenting.

Methods: We prospectively evaluated pre- and post-procedure diffusion-weighted imaging (DWI) examinations of 130 patients who underwent CAS in our tertiary referral center for carotid artery revascularization. The primary endpoint of the study was designed as the presence of new hyperintense lesion on DWI with or without any neurological findings. Patients were classified into two groups DWI (+) and DWI (-) groups. Blood samples were drawn before procedure. The relationship between platelet indices including MPV, PDW and PCT and ischemic brain lesions developed after CAS was analyzed.

Results: Among 130 patients, 40 patients (30.7%) were found to have ischemic brain lesions.We found silent brain ischemia at 30 of these 40 patients, ischemic stroke was detected in 7 patients and TIA in 3 patients. Except for the presence of chronic kidney disease (CKD), there was no significant difference between those with and without ischemic lesions in terms of basic demographic characteristics, while the rate of patients with groupra CKD with ischemic lesion was higher (10% vs. 25%, p=0.043). In terms of procedural features, the fluoroscopy duration was significantly longer in the group with ischemic lesions [14.2 (10.8-19.1) vs. 16.0 (13.3-26.9), p=0.044]. In biochemical analysis, CRP [9 (5-14) vs. 14.5 (11-20), p<0.001], MPV [9.6 (8.2-10.4) etc. 10.6 (9.4-11.0), p=0.001], PDW [12.6 (10.9-15.0) vs. 15.1 (13.5-18.0), p<0.001], PCT [0.215 (0.16-0.26) etc. 0.265 (0.18-0.34), p=0.012] was found to be significantly higher in the ischemic lesion group than those without. There was no significant difference in platelet count between the two groups. There was no significant difference in mortality between the two groups. In multivariate analysis, age, CRP, PCT, MPV and PDW were independent predictors of ischemic brain lesions that may occur after CAS. In ROC curve analysis, 10.750 for MPV, 13.05 for PDW and 0.235 for PCT were determined as the cut-off values for predicting ischemic brain lesions after CAS.

Conclusions: Platelet indices including MPV, PDW and PCT seems to be a simple and feasible indicators to predict the risk of cerebral ischemia during CAS procedure, which might be of clinical value in tailoring the best treatment options in patients with carotid artery stenosis.

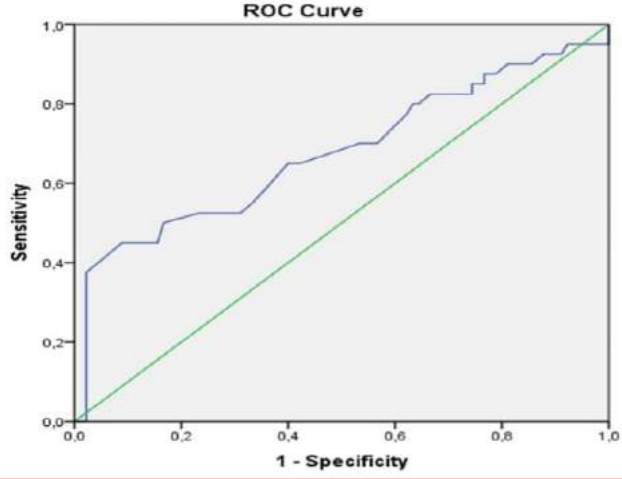

Figure 1. MPV ROC curve analyses for ischemic brain lesions

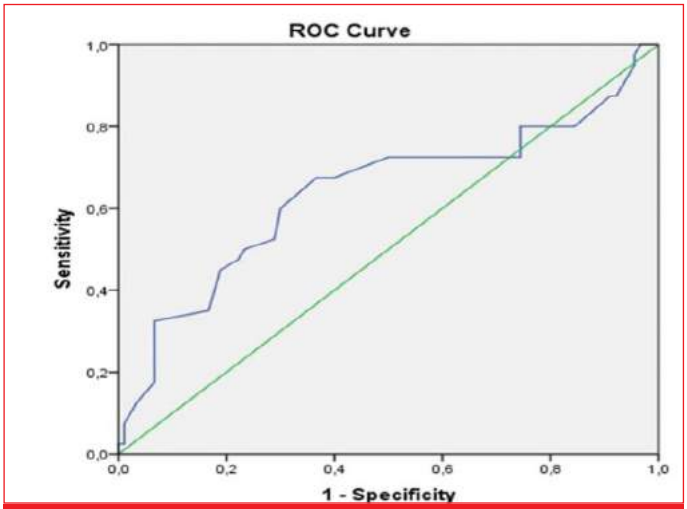

Figure 2. PCT ROC curve analyses for ischemic brain lesions

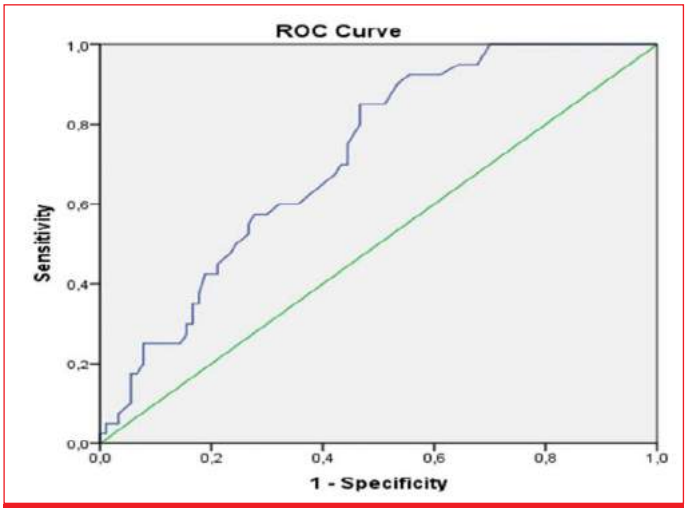

Figure 3. PDW ROC curve analyses for ischemic brain lesions

Table 1. Demographic, clinical and laboratuary findings of the patients

	All patients (n=130)	Ischemic Brain Lesion – (n=90)	Ischemic Brain Lesion + (n=40)	p
Age, years	69.3±6.8	68.4±6.6	71.4±6.8	0.018
Gender, male, n (%)	91 (70.0)	58 (64.4)	33 (82.5)	0.038
HF, n (%)	9 (6.9)	4 (4.4)	5 (12.5)	0.101
HT, n (%)	90 (69.2)	66 (73.3)	24 (60.0)	0.128
DM, n (%)	46 (35.4)	33 (36.7)	13 (32.5)	0.647
Stroke, n (%)	47 (36.2)	29 (32.2)	18 (45.0)	0.162
CKD, n (%)	20 (15.4)	10 (11.1)	10 (25.0)	0.043
Presence of symptom, n (%)	81 (62.3)	54 (60.0)	27 (67.5)	0.415
Hb, g/dL	13.2±1.70	13.2±1.6	13.4±1.9	0.492
WBC, 10^9/L	8.5 (6.9-10.3)	8.4 (6.7-10.5)	8.5 (6.9-10.1)	0.992
Neutrophil, 10^9/L	4.9 (4.1-6.25)	4.9 (4.1-6.4)	5.3 (4.2-6.0)	0.449
Lymphocyte, 10^9/L	2.3 (1.6-3.1)	2.3 (1.7-3.2)	1.9 (1.6-2.9)	0.179
Total cholesterol, mg/dL	242.2±40.04	241.6±39.1	244.3±42.6	0.723
Albumin, g/dL	4.0 (3.6-4.5)	4.1 (3.7-4.5)	4.0 (3.5-4.4)	0.078
CRP, mg/dL	11 (6.75-16)	9 (5-14)	14.5 (11-20)	<0.001
PLT, 10^9/L	262±94	259±93	272±96	0.443
PCT, %	0.22 (0.18-0.29)	0.215 (0.16-0.26)	0.265 (0.18-0.34)	0.012
MPV, fl	9.7 (8.87-10.6)	9.6 (8.2-10.4)	10.6 (9.4-11.0)	0.001
PDW, %	13.9 (11.3-16.0)	12.6 (10.9-15.0)	15.1 (13.5-18.0)	<0.001

HF: Heart Failure, HT: Hypertension, DM: Diabetes Mellitus, CKD: Chronic Kidney Disease, Hb: Hemoglobine, WBC: White Blood Cell, CRP: C-reactibe protein, PLT: Platelet, PCT: Plateletcrit, MPV: Mean Platelet Volume, PDW: Platelet distribution width

Table 2. Procedural findings and outcomes of the patients

Contralateral stenosis, n (%)	35 (26.9)	24 (26.7)	11 (27.5)	0.921
Stenosis degree %	90 (80-95)	90 (80-95)	90 (80-90)	0.554
Lesion lenght, mm	18 (13.5-20)	15 (10-20)	20 (15-24)	0.074
Aortic Arch type, n (%)				
1	102 (78.5)	79 (87.8)	23 (57.5)[a]	
2	10 (7.7)	2 (2.2)	8 (20.0)[b]	<0.001
3	13 (10.0)	5 (5.6)	8 (20.0)[b]	
4	5 (3.8)	4 (4.4)	1 (2.5)	
Predilatation, n (%)	30 (23.1)	18 (20.0)	12 (30.0)	0.212
Postdilatation, n (%)	90 (69.2)	62 (68.9)	28 (70.0)	0.899
Contrast volume, ml	150 (100-200)	150 (110-200)	150 (100-200)	0.585
Fluoroscopy time, min	14.7 (11.1-21.5)	14.2 (10.8-19.1)	16.0 (13.3-26.9)	0.044
Access site complications, n (%)	6 (4.6)	6 (6.7)	0 (0)	0.104
MI, n (%)	0 (0)	0 (0)	0 (0)	
Mortality, n (%)	1 (0.8)	0 (0)	1 (2.5)	0.308
Intracranial bleeding, n (%)	0 (0)	0 (0)	0 (0)	
Silent ischemia, n (%)	30 (23.1)	0 (0)	30 (75,0)	<0.001
Stroke, n (%)	7 (5.4)	0 (0)	7 (17.5)	<0.001
TIA, n (%)	3 (2.3)	0 (0)	3 (7.5)	0.028

MI: Myocardial Infarction, TIA: Transient Ischemic Attack, [a] statistically lower than group 1, [b] statistically higher than group 1

Table 3. Univariable and multivariable logistic regression analysis of the variables for ischemic brain lesions

	Univariate analysis			Multivariate analysis		
	Odds ratio	95% C.I. (Lower-Upper)	p	Odds ratio	95% C.I. (Lower-Upper)	p
Age	1.072	1.011-1.136	0.020	1.101	1.021-1.187	0.013
Gender, male	2.601	1.034-6.545	0.042	2.459	0.832-7.268	0.104
CKD	2.667	1.009-7.047	0.048	1.600	0.456-5.606	0.463
CRP	1.055	1.014-1.097	0.008	1.059	1.011-1.109	0.015
PCT	312.486	4.330-22.549	0.009	824.476	4.678-14.523	0.011
MPV	1.417	1.063-1.890	0.018	1.466	1.049-2.050	0.025
PDW	1.267	1.114-1.442	<0.001	1.315	1.119-1.545	0.001

CKD: Chronic Kidney Disease, , CRP: C-reactibe protein, PLT: Platelet, PCT: Plateletcrit, MPV: Mean Platelet Volume, PDW: Platelet distribution width

Interventional cardiology / Coronary

OP-050

The effect of symptom-to-reperfusion time on left ventricular function, strain, and infarct characteristics in patients with ST-segment elevation myocardial infarction: novel insights from cardiovascular magnetic resonance imaging

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Background and Aim: Data on the relationship between time-to-reperfusion and post-ST-segment elevation myocardial infarction (STEMI) left ventricular (LV) function and infarct characteristics are limited, inconsistent and not up to date. Besides, the interplay between time-to-reperfusion and post-STEMI LV strain remains unknown. We examined the association between the symptom-to-reperfusion time and cardiovascular magnetic resonance (CMR)-derived LV ejection fraction (LVEF), strain, and infarct characteristics in STEMI patients.

Methods: The study included 108 STEMI patients who underwent primary percutaneous coronary intervention (PPCI). Patients were subcategorized according to median of the symptom-to-reperfusion-time: shorter (<160 min, n=54) and longer (>160 min, n=54). CMR was performed 2-7days after PPCI and at 1 month. CMR cine imaging was performed for functional assessment and late gadolinium enhancement to evaluate infarct size and microvascular obstruction (MVO). Myocardial feature tracking technique was used for strain analysis.

Results: Baseline clinical characteristics were comparable between the groups. Baseline and follow-up LVEF were higher in patients with shorter reperfusion time (p=0.02 for both). Patients with shorter reperfusion time had a more favorable baseline and follow-up global LV circumferential and radial strain values, while global LV longitudinal strain was similar for both baseline and follow-up (Figure 1). Baseline and follow-up infarct size were smaller in patients with shorter reperfusion time (p=0.04, p=0.01, respectively). The presence and extent of MVO were similar between the groups.

Conclusions: In STEMI patients, time-to-reperfusion was significantly associated with improvement in LVEF, LV circumferential and radial strain, but not with longitudinal strain. Furthermore, time-to-reperfusion showed a significant relation with infarct size but not with microvascular injury.

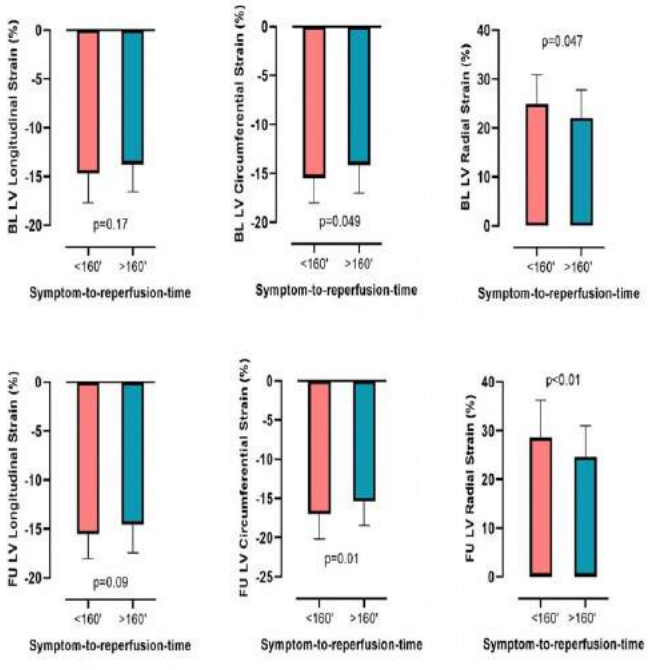

Figure 1. Comparison of baseline and follow-up global LV longitudinal, circumferential, and radial strain between patients with shorter and longer symptom-to-reperfusion-time
Data are shown as mean and standard deviation.

Interventional cardiology / Coronary

OP-051

The effect of bioresorbable stent on long-term clinical outcome in patients with chronic total occlusion

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Background and Aim: Bioresorbable vascular scaffolds (BVS) represent a novel therapeutic option for the treatment of coronary artery disease. There is limited data on clinical and angiographic outcomes following implantation of BVS in patients with complex coronary lesions, particularly chronic total occlusions (CTO) for long term. Therefore, in the present study we aimed to investigate the comparative long-term clinical outcomes of BVS in patients with CTO versus non-CTO lesions.

Methods: In our study, we investigated long-term clinical outcome in which BVS (Absorb, Abbott) was implanted. The demographic and clinical data and as well as long-term follow-up of 214 patients who were implanted Absorb were compared in CTO versus non-CTO lesions.

Results: Of the study population, mean age was 57.4 ± 9.0, majority of them were male (83.5%), 58.3% had hypertension and 30.5% had diabetes. There was no difference in the demographic data of 87 patients who were in the CTO and 127 patients who were in the non-CTO group, exept CTO group had more patients with stable angina (p=0.014) (Figure 1). During the mean follow-up of 72.1 ± 21.2 months, there were no difference between the CTO and non-CTO groups in terms of in-stent restenosis (p=0.601), target lesion revascularization requirement (p=0.097), target vessel revascularization requirement (p=0.524), stent thrombosis (p=0.712), myocardial infarction (p=0.322) and overall survival (log rank p=0.691), (Figure 2).

Conclusions: There were no difference in terms of survival and other clinical outcomes between the CTO and non-CTO groups in a long follow-up period of approximately 6.5 years, who were implanted Absorb stent. Our results demonstrate that BVS implantation in CTO lesions can be performed with good procedural success and reasonable clinical long-term outcome as in non-CTO lesions.

Table 1. Characteristics of the study population

	Non-CTO n = 127	CTO n= 87	p value
HT	75, (%59,06)	49, (%56,32)	0,778
DM	40, (%31,5)	26, (%29,89)	0,881
Family History	7, (%5,51)	8, (%9,2)	0,414
Smoking	16, (%12,6)	17, (%19,54)	0,182
Dyslipidemia	37, (%29,13)	26, (%29,89)	1
CABG History	4, (%3,15)	5, (%5,75)	0,491
PCI History	38, (%29,92)	26, (%29,89)	0,995
MI History	14, (%11,02)	11, (%12,64)	0,96
Access, Femoral	55, (%43,31)	41, (%47,13)	0,829
Access, Radial	72, (%56,69)	45, (%51,72)	0,829
Clinic			0,014
SAP	54, (%42,86)	55, (%63,22)	
UAP	63, (%50)	31, (%35,63)	
NSTEMI	8, (%6,35)	1, (%1,15)	
STEMI	1, (%0,79)	0, (%0)	
Indication			<0,001
Elektive PCI	116, (%91,34)	21, (%24,14)	
CTO	3, (%2,36)	66, (%75,86)	
In-stent restenosis	3, (%2,36)	0, (%0)	
MI	5, (%3,94)	0, (%0)	
Bifurcation	17, (%13,49)	8, (%9,3)	
Age	58 ± 11	56 ± 9	0,712
Survival, years	5,94 ± 1,85	5,84 ± 1,72	0,153
Gender, male	104 (%81,9)	77 (%88,5)	0,248

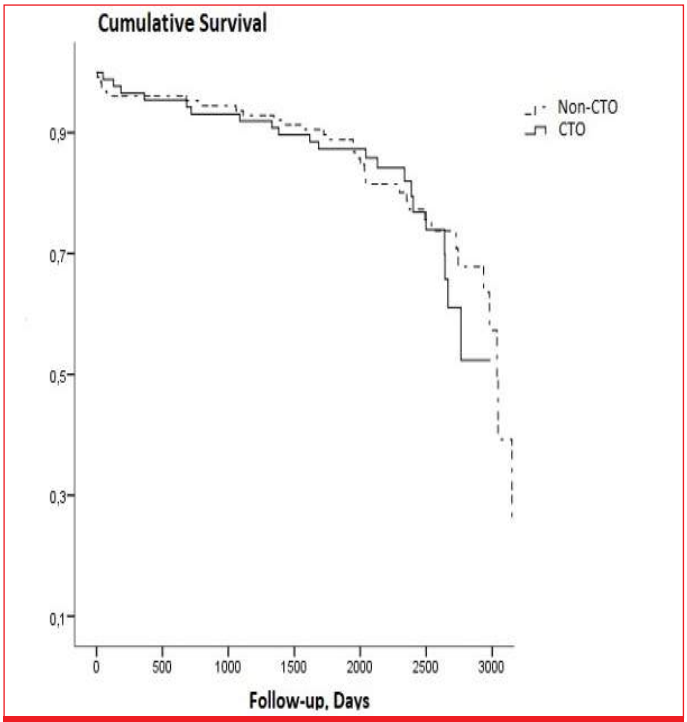

Figure 1. Cumulative Survival

Interventional cardiology / Coronary

OP-052

Association of CABG SYNTAX score with long term clinical outcomes in patients with acute myocardial infarction undergoing SVG PCI

Özge Çakmak Karaaslan[1], Orhan Maden[1], Yücel Kanal[2], İdris Yakut[1], Nezaket Merve Yaman[1], Hasan Can Könte[3], Mehmet Timur Selçuk[1], Hatice Selçuk[1]

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Background and Aim: The CABG SYNTAX score (CSS) has been recommended as an objective and quantitative evaluation tool for coronary anatomic complexity after CABG. We aimed to evaluate the long-term prognostic value of the CSS and its relationship with the composite criteria of all-cause death, cerebrovascular accident (CVA) and/or non-fatal myocardial infarction (MI) in patients who underwent percutaneous coronary intervention (PCI) of saphenous vein graft (SVG).

Methods: We retrospectively evaluated 232 patients who admitted with MI and underwent PCI of SVGs, between 2012 and 2018. The study population was divided into two groups according to the results of the median pre-PCI CSS.

Results: The composite criteria of all-cause death/CVA/non-fatal MI were observed in 107 patients (46.1%). The incidence of the primary endpoint was significantly higher among the patients with a high pre-PCI CSS (p<0.001). Multivariable Cox regression analyses demonstrated that both pre-PCI CSS (HR=1.678, 95% CI: 1.082–2.602, p=0.021) and post-PCI CSS (HR= 1.663, 95% CI: 1.066–2.596, p=0.025) were significantly associated with the primary endpoint. The Kaplan–Meier cumulative curves divided by the median of the pre-PCI CSS demonstrated that, compared with the low pre-PCI CSS group, the high-score group was associated at five years with higher composite criteria of all-cause death/CVA/non-fatal MI (low, 40.3%; high, 57.8%; p=0.015).

Conclusions: Pre-PCI CSS is a significant prognostic factor for the long-term clinical outcomes in patients with previous CABG who underwent PCI of SVG.

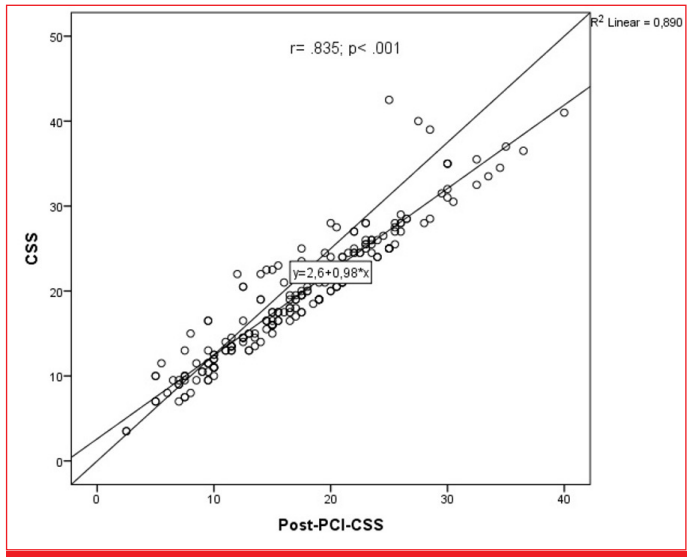

Figure 1. Correlation between pre-PCI CSS and post-PCI CSS

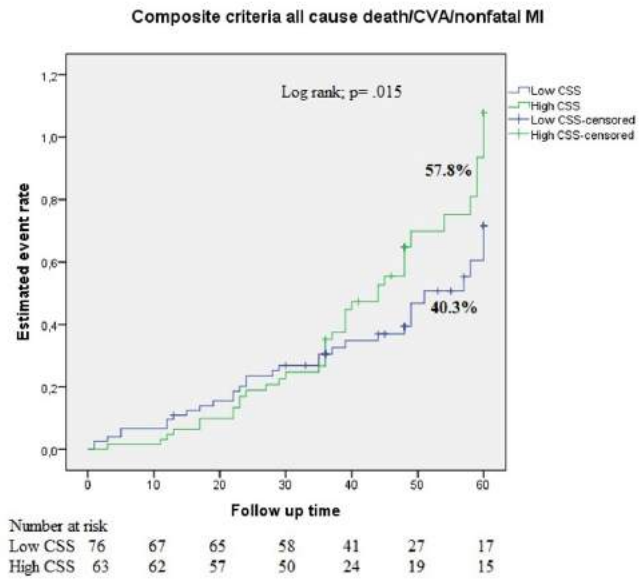

Figure 2. Kaplan-Meier curve showing composite criteria all cause death/CVA/MI rate through follow up time

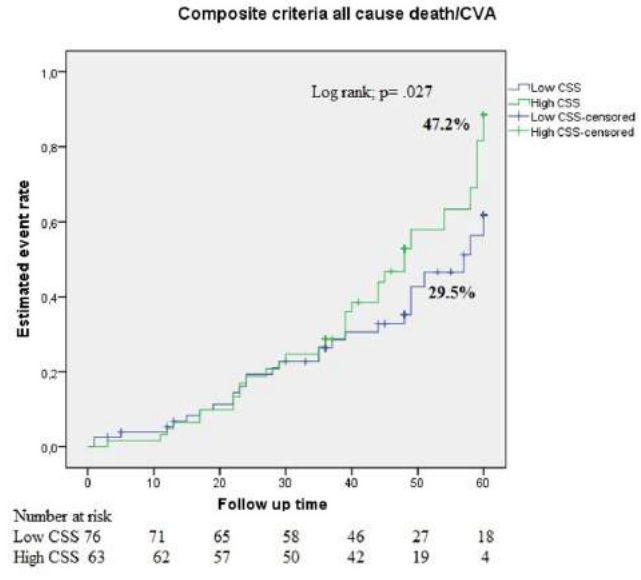

Figure 3. Kaplan-Meier curve showing composite criteria all cause death/CVA rate through follow up time

Epidemiology

OP-053

Mortality trends from ischemic heart disease in Turkey: 2009–2019

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Background and Aim: Cardiovascular diseases still play an important role in public health and epidemiology as the leading cause of death worldwide. Ischemic heart disease (IHD) is the most common reason in this group. This study aims to analyze the latest trends in IHD mortality rates in Turkey by age, gender, and region using the Turkish Statistical Institute (TUIK) mortality data and evaluate the results.

Methods: We have obtained IHD mortality data (2009-2019, as 12 regions) for Turkey from the mortality database of the TUIK. Joinpoint analysis was used to estimate the annual percentage change (APC), and an average annual percentage change (AAPC) to identify significant changes in trends.

Results: The mean mortality rate for IHD in Turkey was in an increasing trend from 2009 to 2019 [APC=1.7 (-0.8; 4.3), p=0.166]. This increase was more pronounced in women [APC=2.2 (-0.7; 5.2), p=0.121] compared to men [APC=1.4 (-1.1; 3.9), p=0.235]. When the period between 2015-2019 was evaluated, it was determined that IHD mortality was in a decreasing trend in the groups over 65 years of age. Also, the death rate due to IHD is almost 2 times higher in men than in women in Turkey, and this rate ratio is highest in the İstanbul region. Between 2009 and 2015 breakout increase was detected in TR3-TR4-TR12 region [APC: 8.9 (2.3; 15.9), p=0.016], [APC: 5.2 (0.4; 10.1), p=0.036], [APC: 4.4 (1.5; 7.3), p=0.01] in men. Also, there was a regular significant increase in the TR2 region [AAPC: 3.5 (0.8; 6.3), p=0.017].

Conclusions: Although IHD mortality trends have decreased globally, our country's average is still on an increasing trend. However, significant decreases have been observed in IHD mortality rates, especially in the group over 65 years of age, in the last 5 years.

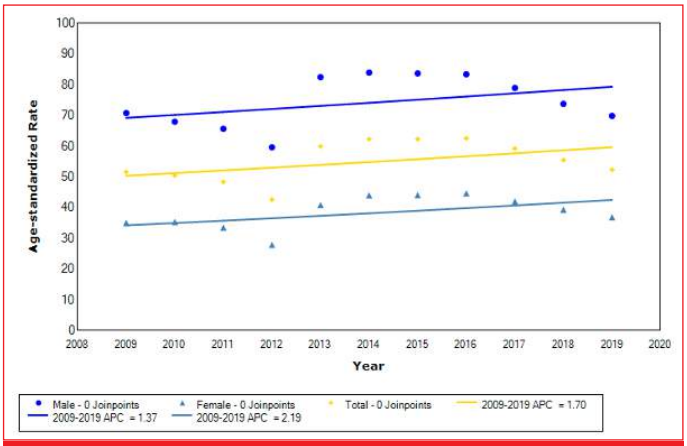

Figure 1. Trends in mortality from ischemic heart diseases in Turkey, by genders, 2009–2019.

Table 1. Number of deaths, distribution of standardized mortality rates by gender

Region	Deaths			ASR			Male/Female Ratio
	Male	Female	Total	Male	Female	Total	
TR1	48836	32889	81725	74.37	34.41	52.48	2.61
TR2	23078	17213	40291	82.07	42.82	61.55	1.92
TR3	59657	46671	106328	80.33	42.86	60.45	1.87
TR4	38078	27273	65351	83.67	43.46	62.30	1.93
TR5	30576	22425	53001	71.82	36.73	52.77	1.96
TR6	42974	31690	74664	76.40	42.52	58.58	1.80
TR7	19979	14717	34696	79.13	41.39	58.71	1.91
TR8	27824	20520	48344	73.81	37.89	54.63	1.95
TR9	13966	10034	24000	65.20	28.57	45.44	2.28
TR10	7925	5709	13634	69.64	36.78	52.13	1.89
TR11	10986	8061	19047	66.94	34.99	49.62	1.91
TR12	19178	15193	34371	71.22	39.76	53.89	1.79
Total	343057	252395	595452	74.55	38.51	55.21	1.95

ASR: Age-standardized rate TR1-Istanbul: Istanbul; TR2-Western Marmara: Balıkesir, Canakkale, Edirne, Kırklareli, Tekirdağ; TR3-Ege: Afyon, Aydin, Denizli, Izmir, Kutahya, Manisa, Mugla, Usak; TR4-Eastern Marmara: Bursa, Eskisehir, Bilecik, Kocaeli, Sakarya, Duzce, Bolu, Yalova; TR5-Western Anadolu: Ankara, Konya, Karaman; TR6-Akdeniz: Adana, Antalya, Burdur, Hatay, Isparta, Mersin (İcel), Kahramanmaras, Osmaniye; TR7-Central Anadolu: Kirikkale, Aksaray, Nigde, Nevsehir, Kırsehir, Kayseri, Sivas, Yozgat; TR8-Western Karadeniz: Zonguldak, Karabuk, Bartin, Kastamonu, Cankırı, Sinop, Samsun, Tokat, Corum, Amasya; TR9-Eastern Karadeniz: Trabzon, Ordu, Giresun, Rize, Artvin, Gumushane; TR10-Northeastern Anadolu: Erzurum, Erzincan, Bayburt, Agri, Kars, Igdir, Ardahan; TR11-Central Eastern Anadolu: Malatya, Elazig, Bingol, Tunceli, Van, Mus, Bitlis, Hakkari; TR12-Southeastern Anadolu: Gaziantep, Adiyaman, Kilis, Sanliurfa, Diyarbakir, Mardin, Batman, Sirnak, Siirt

Epidemiology

OP-054

Oral anticoagulant use and long-term follow-up results in patients with nonvalvular atrial fibrillation in Turkey-AFTER-2 study

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Background and Aim: The aim of this study was to investigate the frequency of oral anticoagulant drugs and time in therapeutic range (TTR) in patients receiving warfarin in addition to the epidemiological trial of non-valvular AF (NVAF) previously conducted in Turkey (AFTER trial). Furthermore, prevalence of major adverse events and mortality rates of the patients were evaluated during the long-term follow-up period.

Methods: We created a national data registry for NVAF patients, reflecting all geographic regions by population density. In that context, the study included all consecutive atrial fibrillation (AF) patients older than 18 years of age who were admitted to the cardiology outpatient clinic except for patients those with prosthetic heart valves and rheumatic mitral valve stenosis. The AFTER-2 study was designed as a prospective, observational and multicenter epidemiological study with 1- and 5-year patient follow-ups. The percentage of time in the therapeutic INR range was calculated according to the Roosendaal method, assuming that the changes (at least 6) between consecutive INR measurements were linear with time. The target TTR level was considered >60% as recommended by the guidelines.

Results: 2592 patients from 35 different centers were included in the study. According to CHA2DS2-VASc score risk classification; 349 patients were in the low-intermediate risk group, and 2243 patients were in the high-risk group. The mean age was 68.7 ± 11.1 years, 55.5% of the patients were female. The most common comorbid diseases were chronic kidney disease (69%) and hypertension (65.5%) (Table 1). The TTR rate in the general population was 40%. In addition, in the general population; the mortality rate at one-year follow-up was 9.1%, and the mortality rate at five-year follow-up was 29.4% (Table-2). Kaplan-Meier analysis performed to examine the survival time during 5 years of follow-up according to their CHA2DS2-VASc risk score classifications. As the risk class increased, lower survival rates were observed during the follow-up period (log rank: 68.6; p<0.001) (Figures 1, 2).

Conclusions: The AFTER-2 study showed higher use of anticoagulation in NVAF patients than in previous national studies. Furthermore, this study demonstrated that most of the NVAF patients are in the high-risk group and the TTR rates is still low in Turkey. As a result, this is a significant reason for switching from warfarin to non-K vitamin dependent oral anticoagulant (NOAC) treatments.

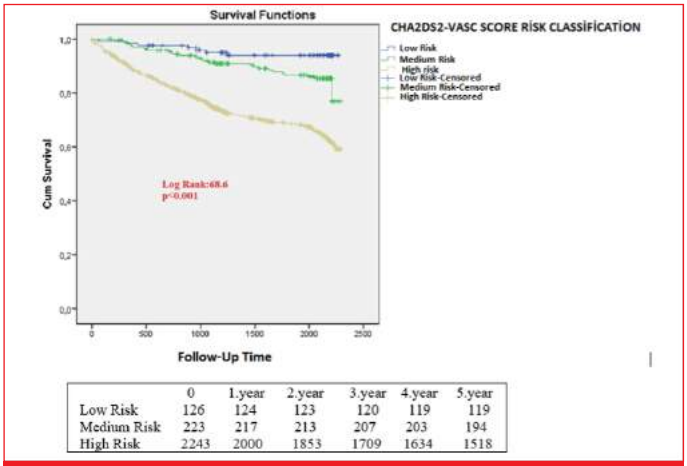

Figure 1. Kaplan-Meier analysis between CHA2DS2-VASC score risk classification and mortality during 5-year follow-up

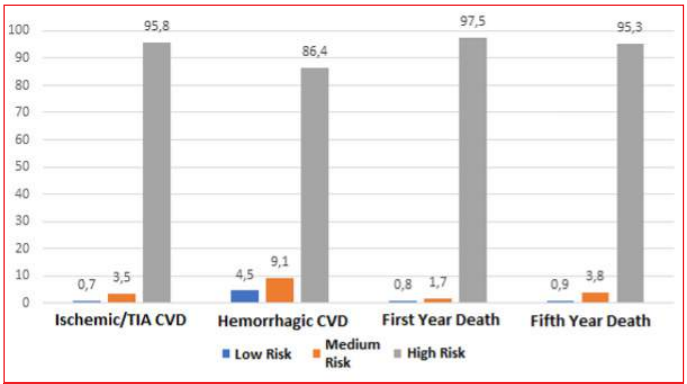

Figure 2. Relationship between primary endpoints and CHA2DS2-VASC score risk classification

Table 1. Demographic characteristics and echocardiographic results

CHA2DS2-VASC score risk classification	Low-Medium Risk (n=349)	High risk (n=2243)	P value	Total (n=2592)
Gender (Female), n (%)	105 (30.1)	1333 (59.4)	<0.001	1438 (55.5)
Age, (years)	56.2 ± 11.6	70.5 ± 9.4	<0.001	68.7 ± 11.1
Body mass index, (kg/m^2)	27.9 ± 3.6	28.2 ± 4.0	0.135	28.2 ± 3.9
Heart rate (minute)	85.7 ± 17.7	87.9 ± 18.2	0.035	87.6 ± 18.2
Systolic blood pressure (mm Hg)	124.5 ± 15.2	128.5 ± 17.6	<0.001	128 ± 17.4
Diastolic blood pressure	76.3 ± 10.0	77.9 ± 11.6	0.014	77.7 ± 11.4
Ischemic cardiomyopathy, n (%)	29 (8.3)	644 (28.7)	<0.001	673 (26)
Dilated cardiomyopathy, n (%)	11 (3.2)	123 (5.5)	0.067	134 (5.2)
Hypertrophic cardiomyopathy, n (%)	2 (0.6)	18 (0.8)	0.649	20 (0.8)
Chronic obstructive pulmonary disease, n (%)	35 (10)	417 (18.6)	<0.001	452 (17.4)
Deep venous thrombus, n (%)	0	6(0.3)	0.420	6 (0.2)
Pulmonary embolism, n (%)	1 (0.3)	13 (0.6)	0.419	14 (0.5)
Thyroid dysfunction, n (%)	13 (3.7)	77 (3.4)	0.782	90 (3.5)
Glomerular filtration rate, (mL/min)	58.4 (42.8-94)	41.0 (28.5-64.8)	<0.001	43.8 (29.9-68)
Chronic renal failure, n (%)	180 (51.6)	1609 (71.7)	<0.001	1789 (69)
Smoker, n (%)	37 (10.6)	114 (5.1)	<0.001	151 (5.8)
Hypertension, n (%)	79 (22.6)	1620 (72.2)	<0.001	1699 (65.5)
Diyabetes mellitus, n (%)	16 (4.6)	560 (25)	<0.001	576 (22.2)
Ischemic cerebrovascular disease/transient ischemic attack, n (%)	4 (1.1)	204 (9.1)	<0.001	208 (8)
Hemorrhagic cerebrovascular disease, n (%)	3 (0.9)	19 (0.8)	0.585	22 (0.8)
Ejection fraction, (%)	55.5 ± 7.8	49.8 ± 10.9	<0.001	50.5 ± 10.7
Left atrium diameter	41.8 ± 6.6	45.4 ± 6.8	<0.001	44.9 ± 6.9
Left atrium volume	62.9 ± 33.9	71.6 ± 31.3	<0.001	70.5 ± 31.8
Left atrium thrombus, n (%)	8 (2.3)	16 (0.7)	0.004	24 (0.9)

Table 2. Follow-up results, HASBLED score and TTR results

CHA2DS2-VASC score risk classification	Low-medium risk (n=349)	High risk (n=2243)	P value	Total (n=2592)
Follow-up time, (day)	2072 (1197-2188)	1626 (880-2130)	<0.001	1920 (939-2133)
Ischemic cerebrovascular disease/transient ischemic attack in follow-up, n (%)	6 (1.7)	136 (6.1)	0.001	142 (5.5)
Hemorrhagic cerebrovascular disease in follow-up, n (%)	1 (0.3)	11 (0.5)	0.505	12 (0.5)
Death at first-year follow-up, n (%)	6 (1.7)	231 (10.3)	<0.001	237 (9.1)
Death at fifth-year follow-up, n (%)	36 (10.3)	725 (32.3)	<0.001	761 (29.4)
HASBLED score	0 (0-1)	2 (1-2)	<0.001	1 (1-2)
TTR (time in therapeutic range), (%)	49.0 (26-85.4)	40 (22.8-70)	0.163	40 (23-70)

Epidemiology

OP-055

Long-term efficacy and safety results of patients using warfarin with effective time in therapeutic range and patients using non vitamin-K antagonist oral anticoagulants (AFTER-2 study subgroup analysis)

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Background and Aim: In this multicenter study we conducted in Turkey, we aimed to compare the long-term follow-up results of patients with non-valvular atrial fibrillation (NVAF) who use warfarin (VKA) and have an effective time in therapeutic range (TTR) with those who use non-vitamin K antagonist oral anticoagulant (NOAC).

Methods: We created a national data registry for NVAF patients that reflects all geographic regions by population density. In this context, all consecutive AF patients older than 18 years of age who applied to the cardiology outpatient clinic, except for patients with prosthetic heart valve and rheumatic mitral valve stenosis, were examined. A total of 1140 patients from 35 different centers who met the criteria were included in the study. Patients who were switched between OACs were excluded from the study. While determining the TTR value, the results with no more than 100 days interval between international normalized ratio (INR) values were evaluated during the follow-up period. During the follow-up period, at least 4 INR values per year were taken and TTR value was calculated according to the Roosendaal method. The effective TTR level was accepted as >60% as recommended by the guidelines. There were 254 patients in the effective TTR group and 886 patients in the NOAC group. Primary endpoints; ischemic cerebrovascular disease (CVD), hemorrhagic CVD, and death at one and five years were determined.

Results: Demographic, echocardiographic and laboratory findings of the patients in the effective TTR and NOAC groups were compared in Table 1 and Table 2. There was no significant difference between the two groups in terms of ischemic CVD, hemorrhagic CVD, and death at during one and five years of follow-up (Tables 3-6, Figures 1a-1d and 2).

Conclusions: We observed similar results in terms of primary endpoints in patients with NVAF who used VKA and had effective TTR and patients using NOAC. It is important in terms of being a large-participant study conducted in our country on this subject. The higher cost of NOAC group drugs compared to VKA is a problem. However, considering the density of patients other than effective TTR in our country, we can conclude that the use of NOAC may be more cost-effective in terms of overall health costs. In patients with NVAF and no contraindications, NOAC should be considered instead of VKA, even if they have effective TTR.

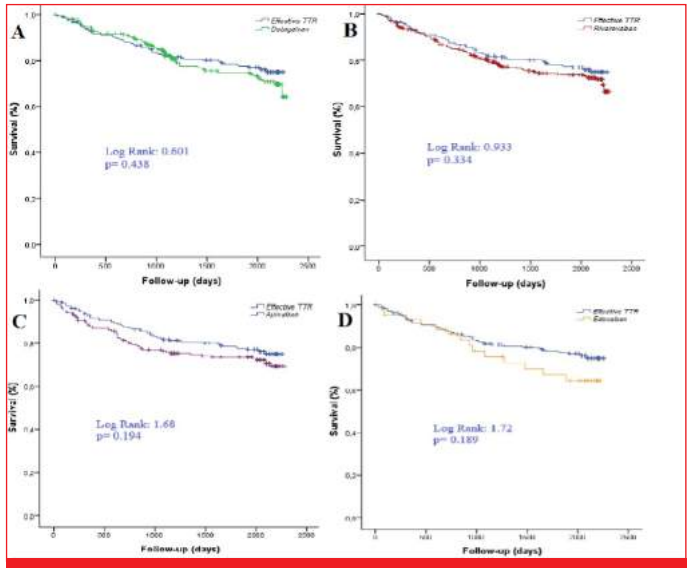

Figure 1. a, b, c, d. Comparison of patients with effective TTR using warfarin and patients using NOAC. Kaplan-Meier analysis between 5-year follow-up and mortality.

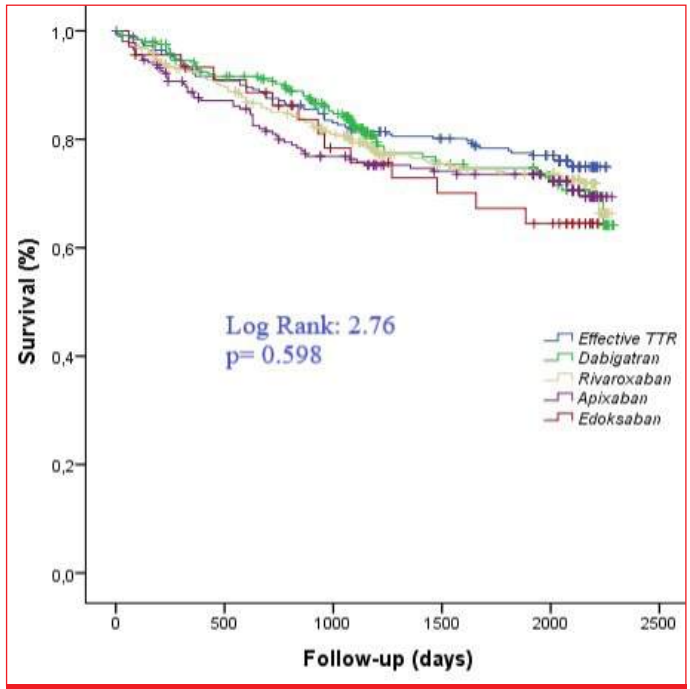

Figure 2. Comparison of patients with effective TTR using warfarin and patients using NOACs. Kaplan-Meier analysis between 5-year follow-up and mortality.

Table 1. Demographic characteristics and echocardiographic results

Parameters	Overall n=1140	Effective TTR n=254	NOAC n=886	P value
Gender, female, n (%)	650 (57)	140 (55.1)	510 (57.6)	0.488
Age, (years)	69.37 ± 10.38	68.56 ± 10.43	69.60 ± 10.37	0.161
Persistant-permanent AF	859 (75.4)	210 (82.7)	649 (73.3)	0.002
EHRA 1-2	943 (82.7)	213 (83.9)	730 (82.4)	0.586
Treatment strategy, rhythm control	281 (24.6)	47 (18.5)	234 (26.4)	0.010
Body mass index, (kg/m^2)	28.46 ± 3.94	28.23 ± 3.94	28.52 ± 3.94	0.306
Ischemic cardiomyopathy, n (%)	303 (26.6)	57 (22.4)	246 (27.8)	0.090
Dilated cardiomyopathy, n (%)	51 (4.5)	14 (5.5)	37 (4.2)	0.364
Chronic obstructive pulmonary disease, n (%)	191 (16.8)	47 (18.5)	144 (16.3)	0.397
Deep venous thrombus, n (%)	5 (0.4)	1 (0.4)	4 (0.5)	0.690
Pulmonary embolism, n (%)	8 (0.7)	3 (1.2)	5 (0.6)	0.255
Thyroid dysfunction, n (%)	37 (3.2)	5 (2)	32 (3.6)	0.193
Glomerular filtration rate, (mL/min)	44 (31)	41.79 (39)	44 (28)	0.358
Chronic renal failure, n (%)	749 (65.7)	170(66.9)	579 (65.3)	0.640
Smoker, n (%)	63 (5.5)	24 (9.4)	39 (4.4)	0.002
Hypertension, n (%)	789 (69.2)	165 (65)	624 (70.4)	0.096
Diyabetes Mellitus, n (%)	263 (23.1)	45 (17.7)	218 (24.6)	0.022
Ischemic CVD/transient ischemic attack, n (%)	84 (7.4)	13 (5.1)	71 (8.0)	0.119
Hemorrhagic CVD, n (%)	9 (0.8)	2 (0.8)	7 (0.8)	0.997
Ejection fraction, (%)	50.86 ± 11.02	52.68 ± 10.92	50.33 ± 11.0	0.003
Left atrium diameter, mm	45.40 ± 6.70	45.22 ± 6.42	45.45 ± 6.78	0.624
Left atrium thrombus, n (%)	5 (0.4)	1 (0.4)	4 (0.5)	0.902

Table 2. Laboratuar Parameters Results

Parameters	Overall n=1140	Effective TTR n=254	NOAC n=886	P value
Hg, (gr/dL)	12.98 ± 1.89	12.82 ± 1.94	13.03 ± 1.87	0.111
Hct, (%)	39.89 ± 5.13	39.13 ± 5.20	40.10 ± 5.10	0.007
Plt, (10^3/Ul)	233.07 ± 69.97	228.40 ± 70.54	234.41 ± 69.79	0.228
WBC, (10^3/uL)	7.92 ± 2.37	7.74 ± 2.34	7.98 ± 2.37	0.164
Neutrophil, (%)	62.94 ± 10.08	61.85 ± 10.10	63.25 ± 10.06	0.052
Lymphocyte, (%)	25.78 ± 8.80	26.0 ± 8.75	25.72 ± 8.82	0.646
Monocyte, (%)	7.37 ± 2.60	7.35 ± 2.45	7.37 ± 2.65	0.919
MPV, (%)	9.10 ± 1.53	9.04 ± 1.61	9.11 ± 1.50	0.515
Glucose, (mg/dL)	118.43 ± 41.66	116.1 ± 40.15	119.0 ± 40.15	0.318
Urea, (mg/dL)	30 (20.5)	28 (20)	30.7 (20)	0.003
Creatinine, (mg/dL)	0.88 (0.38)	0.90 (0.35)	0.87 (0.37)	0.029
ALT, (U/L)	18 (12)	18.5 (11)	17 (12)	0.130
AST, (U/L)	23.95 ± 11.05	24.0 ± 10.9	23.93 ± 11.07	0.927
Albumin, (gr/dL)	4.03 ± 0.53	3.96 ± 0.54	4.05 ± 0.52	0.025
Total Cholesterol, (mg/dL)	178.73 ± 41.34	179.49 ± 44.76	178.51 ± 40.33	0.740
Triglyceride, (mg/dL)	129.73 ± 61.46	132.81 ± 65.76	128.84 ± 60.17	0.364
LDL, (mg/dL)	108.27 ± 34.94	103.83 ± 37.65	109.55 ± 34.04	0.021
HDL, (mg/dL)	44.92 ± 12.50	44.18 ± 11.86	45.13 ± 12.67	0.283
Uric acid, (mg/dL)	6.2 (2.3)	6.6 (2.3)	6.1 (2.2)	0.021

Table 3. Comparison of the results effective TTR group and the patients using dabigatran

Parameters	Effective TTR n=254	Dabigatran n=243	P value
Ischemic CVD/transient ischemic attack in follow-up, n (%)	10 (3.9)	14 (5.8)	0.343
Hemorrhagic CVD in follow-up, n (%)	1 (0.4)	2 (0.8)	0.616
Death at first-year follow-up, n (%)	18 (7.1)	13 (5.3)	0.424
Death at fifth-year follow-up, n (%)	59 (23.2)	58 (23.9)	0.969
Primer endpoint, n (%)	62 (24.4)	71 (29.2)	0.226
HASBLED score, median (IR)	1 (1)	1 (1)	0.062
CHA2DS2-VASC score, median (IR)	3 (2)	3 (2)	0.553

Table 4. Comparison of the results effective TTR group and the patients using rivaroxaban

Parameters	Effective TTR n=254	Rivaroxaban n=393	P value
Ischemic CVD/transient ischemic attack in follow-up, n (%)	10 (3.9)	27 (6.9)	0.117
Hemorrhagic CVD in Follow-up, n(%)	1 (0.4)	2 (0.5)	0.833
Death at first-year follow-up, n (%)	18 (7.1)	32 (8.1)	0.623
Death at fifth-year follow-up, n (%)	61 (24.0)	103 (26.2)	0.531
Primer endpoint, n (%)	62 (24.4)	117 (29.8)	0.137
HASBLED score	1 (1)	1 (1)	0.037
CHA2DS2-VASC score	3 (2)	3 (2)	0.310

Table 5. Comparison of the results effective TTR group and the patients using apixaban

Parameters	Effective TTR n=254	Apixaban n=205	P value
Ischemic CVD/transient ischemic attack in follow-up, n (%)	10 (3.9)	11 (5.4)	0.466
Hemorrhagic CVD in follow-up, n(%)	1 (0.4)	0	0.553
Death at first-year follow-up, n(%)	18 (7.1)	23 (11.2)	0.123
Death at fifth-year follow-up, n(%)	61 (24.0)	57 (27.8)	0.356
Primer endpoint, n (%)	62 (24.4)	59 (28.8)	0.291
HASBLED score	1 (1)	2 (1)	0.005
CHA2DS2-VASC score	3 (2)	3 (2)	0.119

Table 6. Comparison of the results effective TTR group and the patients using edoxaban

Parameters	Effective TTR n=254	Edoxaban n=45	P value
Ischemic CVD/transient ischemic attack in follow-up, n (%)	10 (3.9)	3 (6.7)	0.309
Hemorrhagic CVD in Follow-up, n (%)	1 (0.4)	0	0.849
Death at first-year follow-up, n (%)	18 (7.1)	4 (8.9)	0.429
Death at fifth-year follow-up, n (%)	61 (24.0)	15 (33.3)	0.186
Primer endpoint, n (%)	62 (24.4)	15 (33.3)	0.207
HASBLED score	1 (1)	2 (1)	0.008
CHA2DS2-VASC score	3 (2)	3 (1)	0.109

Epidemiology

OP-056

Dapagliflozin improves cardiac autonomic function measures in type 2 diabetic patients with cardiac autonomic neuropathy

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Background and Aim: Cardiac autonomic neuropathy (CAN) is a frequent complication of type 2 diabetes mellitus. CAN, in which sympathetic tone predominates over parasympathetic activity, increases both cardiovascular morbidity and mortality and unfortunately has no definitive treatment. Sodium-glucose cotransporter-2 (SGLT2) inhibitors have been suggested to reduce sympathetic nervous system activity, based on the results from previous studies. In this study, we aimed to investigate the effect of 24-week treatment with dapagliflozin, an SGLT2 inhibitor, on cardiac autonomic function measures in patients with type 2 diabetes mellitus and CAN.

Methods: A total of 246 type 2 diabetic patients naïve to SGLT2 inhibitors and who were planned to add dapagliflozin 10 mg/day or other oral antidiabetic drug(s) (except glucagon-like peptide-1 analogs or other SGLT2 inhibitors) to their treatment due to suboptimal glycemic control with their existing medications were included. All underwent cardiovascular autonomic reflex tests. Of these, 114 had definite or confirmed CAN according to these tests. Thirty-four patients were exluded due to exclusion criteria. Dapagliflozin 10 mg/day (n=42) or non-SGLT2 inhibitor oral antidiabetic(s) (n=38) was added to the treatment of the remaining patients. All patients underwent 24-hour Holter-electrocardiogram recordings to obtain heart rate variability and heart rate turbulence parameters before starting additional medication and after a 24-week treatment period.

Results: In-group analyses showed that dapagliflozin 10 mg/day for 24 weeks improved heart rate variability and heart rate turbulence parameters and decreased the frequency of ventricular premature beats relative to their baseline values. No such findings were observed in the control group despite similar glycemic control (Table 1). The adjusted difference of hemoglobin A1c between the groups was -0.037% (95% CI: -0.197% to 0.122%, p=0.645). However, intergroup comparisons revealed that adjunctive therapy with dapagliflozin resulted in an increase (improvement) of 8.79 ms in SDNN (p=0.005), 6.11 ms in SDANN (p=0.036), 4.16 ms in SDNN index (p=0.010), 6.95 ms in RMSSD (p=0.019), and 1.58% in pNN50 (p=0.003) relative to the non-SGLT2 inhibitor antidiabetics (Table 1). Similarly, in-group comparisons pointed out that heart rate turbulence parameters were found to be significantly improved only in dapagliflozin group. Intergroup analysis showed that add-on therapy with dapagliflozin for 24 weeks increased the turbulence slope values and decreased the turbulence onset values compared to therapy without an SGLT2 inhibitor in type 2 diabetic patients with cardiac autonomic neuropathy (Table 1).

Conclusions: Dapagliflozin improves measures of cardiac autonomic function compared to the control group in type 2 diabetic patients with CAN. This intergroup benefit, demonstrated for the first time, may be promising for the regression of CAN with SGLT2 inhibition.

Table 1. Baseline versus 24-week comparisons and intergroup analysis of 24-hour holter-ECG findings, time-domain heart rate variability, and heart rate turbulence parameters between dapagliflozin and control groups

	Dapagliflozin Group (n=42)			Control Group (n=38)			Adjusted Difference Between Groups	
	Baseline	24 Weeks	P	Baseline	24 Weeks	P	Mean (95% CI)	P
24-hour Holter-ECG findings								
PVBs per hour, n	15.9 ± 20.7	11.3 ± 17.6	<0.001	15.1 ± 21.1	14.4 ± 20.1	0.519	-3.89 (-7.61, -0.16)	0.041
PVBs percent	0.329 ± 0.424	0.233 ± 0.351	<0.001	0.315 ± 0.426	0.304 ± 0.423	0.280	-0.084 (-0.158, -0.011)	0.025
Mean heart rate, beats/minute	81.6 ± 6.0	82.4 ± 6.2	0.414	81.0 ± 5.4	81.6 ± 5.9	0.417	0.083 (-2.244, 2.409)	0.944
Heart rate variability parameters								
SDNN, ms	102.1 ± 25.1	113.1 ± 29.5	<0.001	100.3 ± 24.1	102.6 ± 23.4	0.067	8.79 (2.76, 14.82)	0.005
SDANN, ms	85.1 ± 22.4	92.3 ± 27.4	0.006	82.9 ± 23.7	84.1 ± 23.3	0.380	6.11 (0.41, 11.81)	0.036
SDNN index, ms	31.2 ± 15.4	35.9 ± 17.4	0.003	31.0 ± 13.2	31.5 ± 12.6	0.141	4.16 (1.01, 7.32)	0.010
RMSSD, ms	41.6 ± 28.3	49.9 ± 28.4	0.001	39.2±27.6	40.6 ± 26.2	0.099	6.95 (1.19, 12.70)	0.019
pNN50, %	4.02 ± 3.33	5.8 ± 4.59	<0.001	4.13 ± 2.96	4.34 ± 2.67	0.174	1.58 (0.57, 2.58)	0.003
Heart rate turbulence parameters								
Turbulence onset	-0.0454 ± 0.0995	-0.0673 ± 0.1044	<0.001	-0.0486 ± 0.0876	-0.0458 ± 0.0903	0.104	-0.025 (-0.039, -0.010)	0.001
Turbulence slope, ms/RR	3.64 ± 4.77	5.22 ± 5.56	<0.001	3.97 ± 4.61	4.21 ± 5.14	0.129	1.348 (0.705, 1.991)	<0.001

Data presented as mean ± standard deviation.
ECG, electrocardiogram; n, numbers; pNN50, percentage of successive normal to normal (NN) intervals that differ by more than 50 milliseconds; PVBs, premature ventricular beats; RMSSD, root mean square of successive RR interval difference; SDANN, standard deviation (SD) of the average NN intervals for each 5-minute segment of the 24-hour recording; SDNN, SD of NN intervals; SDNN index, mean of the 5-minute SD of the NN interval calculated over 24 hours.

Epidemiology

OP-057

Impact of sodium-glucose cotransporter-2 inhibitors on sympathetic nervous system activity detected by sympathetic activity index and LF/HF ratio in patients with type 2 diabetes mellitus

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Background and Aim: Cardiac autonomic neuropathy is a serious microvascular complication of type 2 diabetes mellitus that affects a significant portion of patients. Due to decreased parasympathetic activity, the sympathetic nervous system becomes dominant, causing several problems that lead to increased cardiovascular morbidity and mortality. Sodium-glucose cotransporter-2 inhibitors have been shown to reduce sympathetic nervous system activity previously. The aim of this study is to evaluate the effect of at least 6 months of sodium-glucose cotransporter-2 inhibitor treatment on sympathetic nervous system activity with sympathetic activity index and heart rate variability parameters in patients with type 2 diabetes mellitus.

Methods: Holter-electrocardiogram recordings of 50 patients who were using a sodium-glucose cotransporter-2 inhibitor (empagliflozin or dapagliflozin) for at least 6 months and 50 patients who did not were analyzed retrospectively. The sympathetic activity index and heart rate variability parameters of these 2 groups, which were similar in terms of age, gender, hemoglobin A1c, and duration of diabetes, were compared. The sympathetic activity index relies on an appropriate combination of Laguerre coefficients derived from the use of Laguerre functions, which have unique properties in the time and frequency domains of heart rate variability (Figure 1).

Results: The ratio of low-frequency to high-frequency (LF/HF) power reflecting the sympathovagal balance [–1.495 (–2.165/–1.196) vs. –1.224 (–1.619/–0.863), p=0.008] and sympathetic activity index [1.44 (1.06/2.76) vs. 2.47 (1.42/3.68), p=0.009] was lower in the sodium-glucose cotransporter-2 inhibitor group than in the control group (Table 1). In addition, the sympathetic activity index was correlated with the LF/HF ratio (r=0.418, p<0.001).

Conclusions: Sodium-glucose cotransporter-2 inhibitor treatment for at least 6 months was found to result in lower values of sympathetic activity index and the LF/HF ratio in patients with type 2 diabetes mellitus. These findings indicate lower sympathetic nervous system activity, which supports the sympathoinhibitor effects of sodium-glucose cotransporter-2 inhibitors. This is a promising finding for restoring the impaired sympathovagal balance in cardiac autonomic neuropathy.

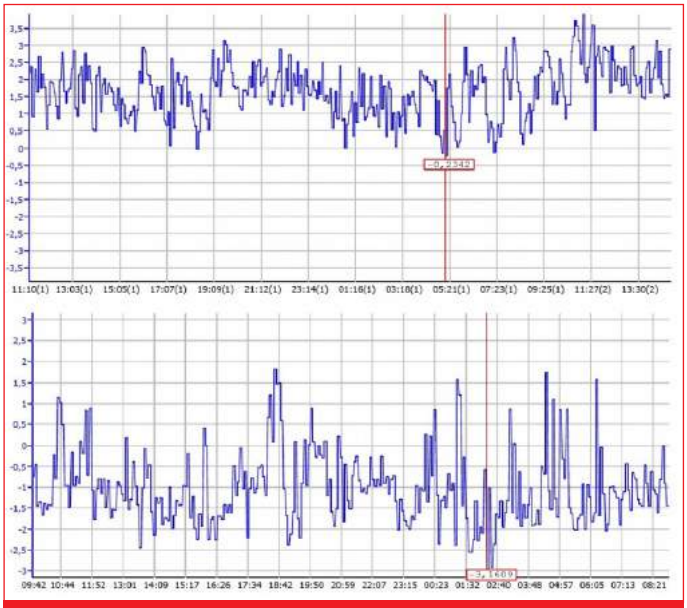

Figure 1. The y-axis shows the sympathetic activity index, and the x-axis shows the time in hours. During the entire recording, it is seen that the sympathetic activity is higher in the upper graph and lower in the below one. The lowest SAI values of the patients in the graphics (seen in the red box) were used for statistical analyses.

Table 1. Comparison of the clinical and laboratory characteristics, heart rate variability parameters, and sympathetic activity index between SLGT2 inhibitor and control groups

	SGLT2 Inhibitor Group (n=50)	Control Group (n=50)	P
Age, years	58.9 ± 10.5	60.1 ± 11.4	0.593
Gender, male/female, numbers (%)	22/28 (44/56)	26/24 (52/48)	0.423
Duration of diabetes, years	7.3 ± 2.2	7.7 ± 2.5	0.642
Fasting plasma glucose, mg/dL	143 ± 44.2	147.6 ± 39.7	0.228
Hemoglobin A1c, %	7.41 ± 1.80	7.81 ± 1.95	0.263
Resting heart rate, beats/minute	71.7 ± 8.0	74.8 ± 11.8	0.093
Left ventricular ejection fraction, %	63.0 ± 5.5	62.4 ± 5.0	0.327
Time-domain heart rate variability parameters			
SDNN, milliseconds	126.9 ± 29.4	110.7 ± 34.7	0.013
SDANN, milliseconds	106.6 ± 30.0	92.1 ± 32.9	0.023
SDNNI, milliseconds*	32 (25/46.75)	25.5 (18.75/38)	0.021
RMSSD, milliseconds*	61 (36/85.25)	56 (37/66)	0.442
pNN50, %*	5 (3/9.5)	4 (2.75/7)	0.211
Frequency-domain heart rate variability parameters			
LF power, ms^2*	348.5 (178.5/549.4)	512.5 (317.4/915.9)	0.004
HF power, ms^2*	193.5 (103.5/345.2)	207.3 (111.1/422.8)	0.549
LF/HF ratio*	1.44 (1.06/2.76)	2.47 (1.42/3.68)	0.009
Sympathetic activity index*	-1.495 (-2.165/-1.196)	-1.224 (-1.619/-0.863)	0.008

*Data are presented as median (25%/75% interquartile ranges).
P-value < 0.05 is statistically significant.
HF, high frequency; LF, low frequency; pNN50, percentage of successive RR intervals that differ by >50 milliseconds; RMSSD, root mean square of successive RR interval differences; SDANN, standard deviation of the average normal-to-normal intervals for each 5-minute segment of a 24-hour recording; SDNN, standard deviation of normal-to-normal intervals; SDNNI, mean of the standard deviations of all the normal-to-normal intervals for each 5-minute segment of a 24-hour recording; SGLT2, sodium-glucose cotransporter-2.

Epidemiology

OP-058

Effects of empagliflozin on left atrial electromechanical functions in patients with type 2 diabetes mellitus

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Background and Aim: Sodium-dependent glucose co-transporter 2 (SGLT2) inhibitors cause a decrease in extravascular and intravascular volume, like a diuretic agent with osmotic diuresis and natriuresis, as well as preventing glucose reabsorption from the renal tubules. Positive effects of SGLT2 inhibitors on atrial electromechanical functions have been demonstrated in animal experiments. The point of this study to investigate the effects of Empagliflozin on left atrial mechanical functions and atrial electromechanical delay (AEMD) in patients with type 2 diabetes mellitus (T2DM).

Methods: 62 patients (40.3% female, mean age 50.5 ± 8.6 years old) with type 2 DM were enrolled to the study. Participants were used a SGLT2 inhibitor (empagliflozin 10-25 mg/daily) for 6 weeks. Patients were examined initially and after six weeks with echocardiography. LA volume was recorded, atrial conduction times were measured using tissue doppler imaging (TDI).

Results: There were no significant differences was observed in chamber diameters and left ventricular ejection fraction in 6 weeks. A statistically significant decrease was ob-

served in mitral inflow flow peak E velocity and mitral E/A ratio (71.5 ± 11.4 vs. 68.5 ± 13.2, p=0.008; 1.09 ± 0.26 vs. 1.03 ± 0.27, p=0.014). Left atrium passive emptying volume and passive emptying fraction were significantly increased (9.66 ± 3.2 vs. 10.0 ± 3.3, p=0.048; 28.7 ± 8.8 vs. 29.9 ± 0.9, p=0.018). A decrease was observed in all AEMD but the decrease in left intra-atrial and right intra-atrial AEMD were statistically significant (7.5 ± 1.6 vs. 6.3 ± 1.5, p=0.042; 1.25 ± 1.67 vs. 1.87 ± 1.53, p=0.036).

Conclusions: The improvement in left atrial structural and electrical conductions in a short period of 6 weeks is promising in terms of protection from arrhythmias and heart failure, which is common in patients with T2DM. Larger randomized studies with longer follow-up periods are needed to demonstrate the positive effects of SGLT 2 inhibitors on left atrial electromechanical functions.

Table 1. Demographics and basic echocardiographic parameters

Variables	Pre-treatment	Post-treatment	P value
Age, years	50.5 ± 8.6		N/A
Gender, female, n (%)	25 (40.3)		N/A
BMI, kg/m^2	30.5 ± 5.52	30.3 ± 5.3	< 0.001
Body weight, kg	80.2 ± 15.6	79.6 ± 15.0	< 0.001
LV end-diastolic diameter, mm	43.7 ± 3.4	43.6 ± 2.9	0.660
LV end-systolic diameter, mm	27.9 ± 3.8	27.7 ± 3.5	0.150
LV EF, %	65.7 ± 1.2	65.7 ± 1.0	0.450
LA diameters, mm			
Antero-posterior	32.5 ± 3.2	32.5 ± 3.3	0.576
Medio-lateral	35.4 ± 3.7	34.9 ± 3.5	0.067
Apico-basal	41.0 ± 3.6	40.5 ± 3.3	0.003
Right atrial diameters, mm			
Medio-lateral	33.4 ± 4.3	33.1± 4.0	0.136
Apico-basal	40.6 ± 4.6	40.4 ± 3.9	0.290
RV end-diastolic diameter, mm			
Basal	29.8 ± 5.5	29.0 ± 5.8	0.216
Mid	24.0 ± 4.3	23.4 ± 3.3	0.797
Mitral inflow peak E velocity (m/s)	71.5 ± 11.4	68.5 ± 13.2	0.008
Mitral inflow peak A velocity (m/s)	68.5 ± 15.3	67.3 ± 13.6	0.177
Mitral E/A ratio	1.09 ± 0.26	1.03 ± 0.27	0.014
Mitral DT (ms)	0.20 ± 0.04	0.20 ± 0.41	0.584

LV, left ventricular, LA, left atrial, RV, right ventricular, DT, deceleration time. P<0.01 compared with before and after empagliflozin

Table 2. Effect of empagliflozin on AEMD and mechanical functions of left atrium

Variables	Pre-treatment	Post-treatment	P value
LA maximal volume at end-systole (Vmax, mL/m^2)	33.9 ± 6.6	33.8 ± 6.4	0.525
LA minimal volume at end-diastole (Vmin, mL/m^2)	16.0 ± 4.9	15.8 ± 4.9	0.329
Volume at the begining of atrial systole (Vp Ml/m^2)	24.2 ± 6.1	23.8 ± 6.0	0.026
Total emptying volume (mL/m^2)	17.9 ± 3.7	17.9 ± 3.7	0.869
Total emptying fraction (%)	53.4 ± 8.2	53.5 ± 8.4	0.627
Passive emptying volume (mL/m^2)	9.66 ± 3.2	10.0 ± 3.3	0.048
Passive emptying fraction (%)	28.7 ± 8.8	29.9 ± 0.9	0.018
Active emptying volume (mL/m^2)	8.3 ± 2.9	7.9 ± 3.0	0.057
Active emptying fraction (%)	34.4 ± 9.6	33.4 ± 1.0	0.127
PA lateral (ms)	69.87 ± 19.75	68.00 ± 17.60	0.052
PA septal (ms)	62.37 ± 12.28	61.75 ± 10.73	0.412
PA tricuspid (ms)	63.62 ± 14.08	63.62 ± 13.65	0.244
Left intra-atrial (PA lateral – PA septum) (ms)	7.5 ± 1.6	6.3 ± 1.5	0.042
Right intra-atrial (PA septum – PA tricuspid) (ms)	1.25 ± 1.67	1.87 ± 1.53	0.036
Inter-atrial (PA lateral – PA tricuspid) (ms)	6.25 ± 1.92	4.43 ± 1.72	0.085

LA, left atrial, AEMD, atrial electromechanical delay

Epidemiology

OP-059

How did the population characteristics of ENGAGE-AF trial transcribed real life conditions in Turkey: An external validation test with evaluation of treatment safety in patients with atrial fibrillation on edoxaban therapy in real-life in Turkey (ETNA)

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Background and Aim: ENGAGE-AF (Effective Anticoagulation with Factor Xa Next Generation in Atrial Fibrillation – Thrombolysis in Myocardial Infarction 48) trial showed the efficacy and safety of edoxaban therapy in patients with atrial fibrillation ETNA AF Study confirmed the results of ENGAGE-AF Trial in real life conditions. However as one of the biggest European country, Turkey had not been included the ETNA-AF Study. The Evaluation of Treatment Safety in Patients with Atrial Fibrillation on Edoxaban Therapy in Real-Life in Turkey (ETAF-TR) study is designed to evaluate the safety and effectiveness of edoxaban treatment in atrial fibrillation in routine practice in Turkey. In this study, it was aimed to compare the basal characteristics of the patient groups included in the ENGAGE-AF, ETNA-AF and ETAF-TR study.

Methods: ETAF-TR study, in which 1053 cases from 50 centers were included, is a national, multicenter, prospective observational study. The criteria for study inclusion are; being over 18 years of age, already under edoxaban treatment and patients giving consent, and the first patient was included in August 2020. The babaselin demographics of 13638 patients included in ETNA-AF and 2123 patients included in the ENGAGE-AF study from Europe were compared in this study.

Results: Patients included in the ETAF-TR study; according to the patients included in the ETNA-AF and TIMI ENGAGE 48 study; are younger [70.1 (11.3) vs. 73.6 (9.52),72.7 (8.09) p<0.001], and have lower BMI [29.1 (5.4) vs. 28.1 (5.14), 29.8 (5.39) p<0.001]. The CHA2DS2-VASc score of the ETAF TR group was lower than of ENGAGE AF TIMI 48 Trial [5 (1.5) vs. 4.2 (1.31) p<0.001], higher than the population of the ETNA-AF study [3.5 (1.5) vs. 3.1 (1.40) p<0.001]. The HASBLED score of the ETAF-TR study group and the TIMI were similar to the ENGAGE 48 group [1.6 (0.92) vs. 1.6 (1.0) p=1, 1.6 (1.0) vs. 2.6 (1.13) p <0.001]. The patients included in the ETAF TR study have; less hypertension, diabetes, heart failure, and renal failure history; however, they have more myocardial infarction history and higher permanent AF rates than TIMI ENGAGE 48 trial population (p<0.001) (Table 1). According to the patients included in the ETNA-AF study; there were higher history of diabetes, ischemic stroke, TIA, heart failure, myocardial infarction and permanent AF history, while fewer renal diseases were present (p<0.001) in the ETAF-TR study group (Table 1).

Conclusions: Edoxaban therapy with the ETAF-TR study was performed with a population less risky in terms of ischemic events and similar population in terms of bleeding complications than in the phase 3 study TIMI ENGAGE 48 study. Compared to the ETNA-AF population, the ETAF-TR population has higher risk of ischemic events and a lower risk of bleeding complications. The results of the ETAF – TR study are important in terms of showing the results of a population at different risk in terms of ischemic-embolic events and bleeding risk as well as external validation of previous studies.

Table 1.

	ETNA-AF-Europe (n=13,638)	ENGAGE AF-TIMI 48a Corresponding ETNA-AF countries (n=2123)	ETAF-TR (n=1053)	ETNA-AF-EUROPE vs. ETAF-TR P value	ENGAGE AF-TIMI vs. ETAF-TR P value
Age (y), mean (SD)	73.6 (9.52)	72.7 (8.09)	70.1 (11.3)	<0.001	<0.001
By age sub-groups, n (%)					
< 65	2096 (15.4)	344 (16.2)	265 (25.2)	<0.001	0.122
65–75 years	4598 (33.7)	764 (36.0)	397 (37.7)		
≥ 75 years	6939 (50.9)	1015 (47.8)	391 (37.1)		
Male, %	56.6	62.4	41.0	<0.001	<0.001
BMI (kg/m^2), mean (SD)	28.1 (5.14)	29.8 (5.39)	29.1 (5.4)	<0.001	0.0006
Weight (kg), mean (SD)	81.0 (17.34)	85.4 (17.47)	79.1 (15)	0.0005	<0.001
CrCl (mL/min), mean (SD)	69.4 (24.23)	75.5 (28.96)	80.5 (31.5)	<0.001	<0.001
CHA2DS2-VASc, calculated, mean (SD)	3.1 (1.40)	4.2 (1.31)	3.5 (1.5)	<0.001	<0.001
HAS-BLED, mean (SD)	2.6 (1.13)	1.6 (0.92)	1.6 (1.0)	<0.001	1.00
Hypertension, %	76.9	92.4	76.4	0.7109	<0.001
Diabetes, %	21.9	39.1	26.8	0.0002	<0.001
Myocardial infarction, %	4.3	2.9	49.5	<0.001	<0.001
Ischaemic stroke, %	5.9	15.5	13.2	<0.001	0.0852
Transient ischaemic attack, %	3.3	11.9	13.2	<0.001	0.2943
Congestive heart failure, %	5.8	48.2	29	<0.001	<0.001
Paroxysmal AF, %	53.6	26.6	29.5	<0.001	0.0851
Persistent AF, %	24.4	24.1	7.8	<0.001	<0.001
Permanent AF, %	19.6	49.4	57.1	<0.001	<0.001
Renal disease (including dialysis), %	27.0	11.9	7.6	<0.001	<0.001

Hypertension

OP-060

Convinient novel method for diagnosing diastolic dysfunction: Electrocardiographic diastolic index

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Background and Aim: Diastolic dysfunction (DD) is the relaxation defect of the left ventricular myocardium. It can show a broad clinical course, simple impaired myocardial relaxation to end-stage heart failure (HF). Left ventricular (LV) DD is the primary pathophysiology in patients with preserved ejection heart failure. DD is commonly observed in patients with dysregulated blood pressure. DD can be considered an intermediate clinical stage in the progression to HF. Early diagnostic methods can detect the development of diastolic dysfunction and slow the progression to HF. Many patients with diastolic dysfunction are asymptomatic before clinical symptoms of HF. Consequently, cost-effective diagnostic methods come to the fore in diagnosing DD. Various studies on the relationship between ECG and DD are under scope because it is both easily accessible and more cost-effective in predicting diastolic dysfunction.

In this study, we aimed to investigate the predictor of EDI in detecting DD in patients followed up with HT.

Methods: The single-center, retrospective observational study included 202 consecutive patients with hypertension who applied to the cardiology policlinic between January 2022 and March 2022. The patients were divided into groups with and without DD by TTE parameters. Baseline characteristics, TTE and ECG findings, and EDI were compared between the two groups. The EDI is formulated as [aVL R amplitude × (V1S amplitude + V5R amplitude)/PWLI amplitude]. EDI values of the patients were calculated by two experienced cardiologists who were unaware of the patients' TTE parameters.

Results: Two hundred two patients followed with a diagnosis of hypertension were included in the study. Basal characteristics are given in Table 1. The patients were divided into two groups according to the presence of left ventricular diastolic dysfunction (105 patients without LVDD, group 1; 97 patients with LVDD, group 2). The EDI value of the patients included in the study was 8.5 ± 7.3. EDI value in group 2 was significantly higher than in group 1 (p<0.005).

Conclusions: This study is research that determines the role of the electrocardiographic diastolic index in predicting DD in hypertensive patients. A higher EDI value was found in patients with LVDD than those without LVDD. A higher EDI value predicted SVDD, and the optimal cut-off value was calculated at 7.4 mV. These results show that changes in cardiac diastolic parameters can be detected in the 12-lead surface ECG in the patient population without coronary artery disease followed by hypertension. An ECG examination, Cornell criterion, and Sokolow-Lyon voltage criteria were calculated. In our study, evaluating the components of both criteria in a single formula in the electrocardiographic examination increases the index's predictiveness. In this study, the EDI value was significantly higher in patients with LVDD, suggesting that ECG can be used as a critical diagnostic parameter in predicting diastolic dysfunction.

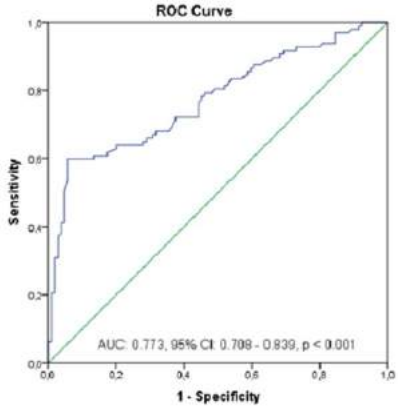

Figure 1. ROC curve
A receiver operating curve (ROC) analysis showed that the optimal cut-off value of the electrocardiographic diastolic index (EDI) to predict diastolic dysfunction (DD) was 7.4 mV with 63.6% sensitivity and 79.8% specificity (area under the curve [AUC] 0.773; 95% confidence interval [CI] 0.708 – 0.839; p<0.001).

Table 1. Baseline clinical characteristics, echocardiographic and electrocardiographic findings of all patients

	All Population (n=202)	LVDD (-) (n=105)	LVDD (+) (n=97)	P value
Age, years	50 ± 14	47 ± 14	53 ± 13	0.019
Male, n (%)	86 (42.6)	35 (33.3)	51 (52.6)	0.007
Female, n (%)	116 (57.4)	70 (66.7)	46 (47.4)	0.007
Diabetes mellitus, n (%)	33 (16.3)	11 (10.5)	22 (22.7)	0.023
Smoking, n (%)	93 (46)	44 (41.9)	49 (50.5)	0.259
BMI, kg/m2	30 ± 10	28.5 ± 11	32 ± 9	0.005
LVEDD, mm	46 ± 3	46 ± 3	46 ± 3	0.124
LVESD, mm	29 ± 4	28 ± 3	29 ± 4	0.051
IVST, mm	1.0 ± 0.2	1.0 ± 0.1	1.1 ± 0.2	< 0.001
PWT, mm	1.0 ± 0.1	1.0 ± 0.10	1.0 ± 0.11	0.002
LVEF, %	61 ± 5	62 ± 5	60 ± 3.5	0.032
LA, mm	35 ± 3	35 ± 4	36 ± 4	0.031
E, cm/sec	70 ± 10	80 ± 10	70 ± 10	< 0.001
A, cm/sec	60 ± 20	60 ± 10	80 ± 30	< 0.001
E/A ratio	1.2 ± 0.5	1.4 ± 0.3	0.9 ± 0.5	< 0.001
e'Lateral, cm/sec	10 ± 4	12 ± 2	8 ± 2	< 0.001
D1 P wave amplitude, mV	0.1 ± 0.06	0.1 ± 0.04	0.1 ± 0.05	0.181
aVL R amplitude, mV	0.4 ± 0.3	0.3 ± 0.3	0.5 ± 0.3	< 0.001
V1S amplitude, mV	0.7 ± 0.3	0.7 ± 0.4	0.7 ± 0.5	0.043
V5R amplitude, mV	1.0 ± 0.6	1.0 ± 0.5	1.1 ± 0.7	0.093
V1S amplitude+V5R amplitude, mV	1.7 ± 0.7	1.7 ± 0.7	2.0 ± 0.9	0.005
EDI	8.5 ± 7.3	5.2 ± 3.7	10.6 ± 8.5	<0.005

BMI: Body mass index, LVEDD: Left ventricular end-diastolic dimension, LVESD: Left ventricular end-systolic dimension, IVST: Interventricular septum thickness, PWT: Posterior wall thickness, LVEF: Left ventricular ejection fraction, LA: Left atrial, EDI: Electrocardiographic Diastolic Index.

Table 2. Univariate logistic regression analysis for left ventricular diastolic dysfunction

	Odds Ratio (95% CI)	P value
EDI	1.248 (1.159 - 1.345)	< 0.001
Age	1.025 (1.005 - 1.047)	0.016
DM	2.507 (1.144 – 5.495)	0.022
BMI	1.060 (1.015 - 1.106)	0.009

CI: Confidence Interval. EDI: Electrocardiographic Diastolic Index, DM: Diabetes mellitus; BMI: Body mass index

Table 3. Multivariate logistic regression analysis for left ventricular diastolic dysfunction

	Odds Ratio (95% CI)	P value
Model 1		
EDI	1.253 (1.161 - 1.352)	< 0.001
Age	1.015 (0.986 - 1.044)	0.322
DM	2.666 (1.071 – 6.638)	0.035
BMI	1.025 (0.968 - 1.086)	0.395
Model 2		
EDI > 7.4	7.262 (3.771 – 13.985)	< 0.001
Age	1.012 (0.986 - 1.040)	0.369
DM	2.263 (0.986 – 1.040)	0.078
BMI	1.033 (0.987 - 1.091)	0.247

CI: Confidence Interval. EDI: Electrocardiographic Diastolic Index, DM: Diabetes mellitus; BMI: Body mass index

Hypertension

OP-061

The value of optic nerve sheath diameter in predicting resistant hypertension

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Background and Aim: Cerebral perfusion pressure (CPP) depends on the difference between mean arterial pressure (MAP) and intracranial pressure (IP) [CPP:MAP-IP].

If the mean arterial pressure remains consistently high, as in resistant hypertension (RHT), it is possible for the IP to remain high to maintain a constant cerebral perfusion pressure. İncreasing systolic blood pressure is strongly associated with lower cerebrospinal fluid flow dynamics. Therefore, it may be possible to estimate the probability of the presence of RHT by the measurement of optic nerve sheath diameter (ONSD), which is a noninvasive indicator of IP. Rapid methods are needed to predict RHT.

Methods: A total of 290 subjects, including the control group (n:102) without any additional disease including HT, the group with HT (n=106) and RHT diagnoses (n=82) for at least 1 year, were included in the study. These subjects had undergone magnetic resonance imaging (MRI) scans in the last 3–6 months. The groups had similar age and sex ratios. Basic biochemical data were recorded. ONSD and ocular globe transverse diameter (Glob_dia) measurements from MR images were performed three times and averaged. Data were evaluated by Kruskal-Wallis + post-hoc tests and the sensitivity of ONSD in predicting RHT was evaluated by ROC analysis. Intra-observer reliability analyses were performed.

Results: In the control, HT and RHT groups, ONSD measurements were [median, IQR] 4.7 (4.4-5), 4.7 (4.4-5.1), 5.3 (4.7-5.5) mm, respectively (p(1-2): 0.762, p(1-3): 0.000, p(2-3): 0.000). The ONSD/Glob_dia ratio was found to be 0.21 (0.2–0.22), 0.21 (0.19–0.23), 0.24 (0.21–0.25) in the groups, respectively. [p(1-2): 0.860, p(1-3): 0.000, p(2-3): 0.001]. The sensitivity of ONSD at 4.75 mm in predicting RHT was determined as 77% (CI: 71–83%).

Conclusions: Optic nerve sheath diameter can be used to predict resistant hypertension with good sensitivity.

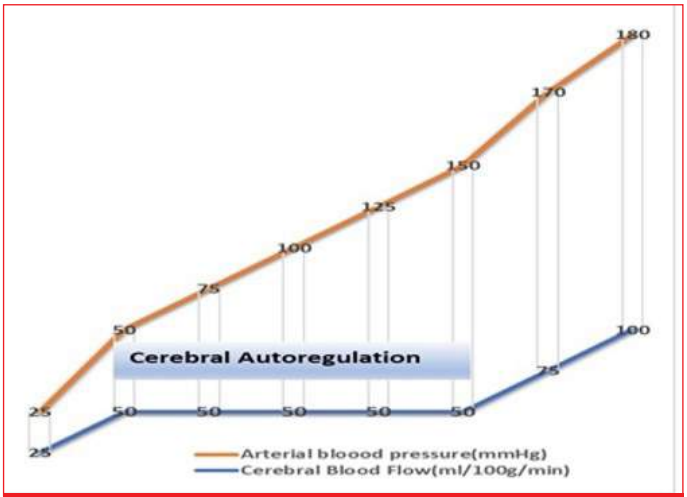

Figure 1. Arterial blood pressure and cerebral autoregulation

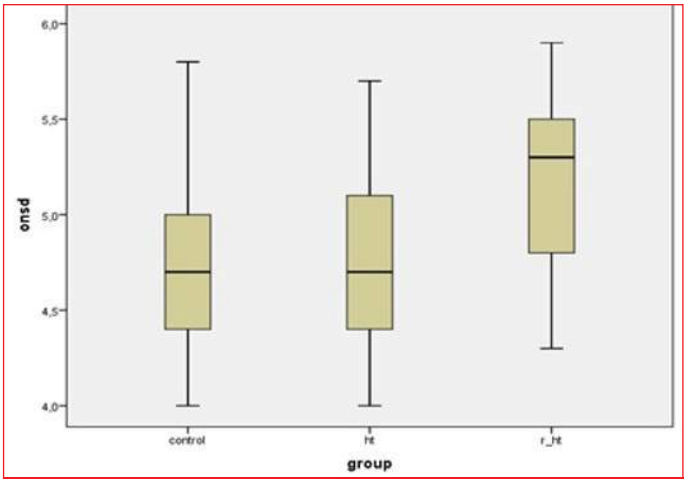

Figure 2. ONSD values between groups

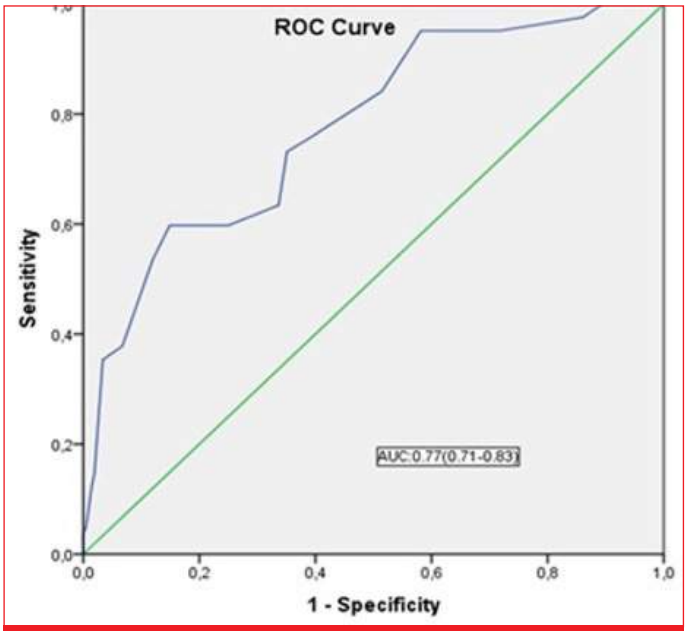

Figure 3. ROC curve showing sensitivity of ONSD (4.75 mm and above)in predicting RHT

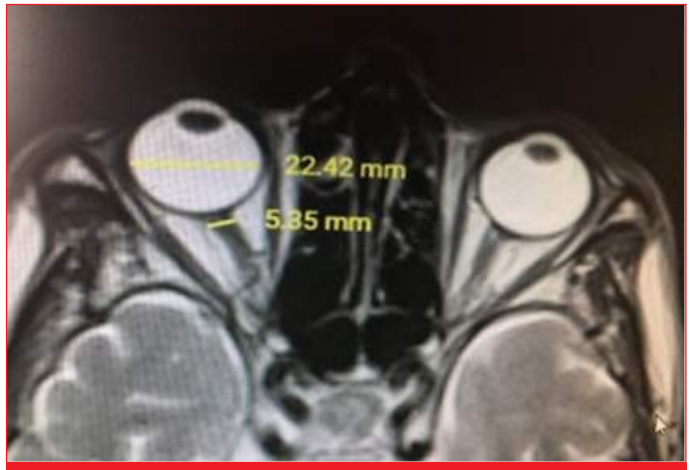

Figure 4. Ocular globe transvers diameter and ONSD measurement

Table 1.Comparison of baseline characteristics and optic nerve sheat,ocular globe diameter measurements between groups

	Control (group 1, n=102)	HT (group 2, n=106)	RHT (group 3, n=82	P (1-2)#	P (1-3)#	P (2-3)#	P
Age*	54 (51-61)	55 (51-62)	56 (52-60)	0.266	0.128	0.715	0.294†
Female/Male(%)	51/51 (50%)	55/51 (51%-48%)	41/41 (50%)	0.083	1	0.368	0.953§
BMI (kg/m^2)	26.5 (24.7-28.5)	26.5 (24.7-28.5)	26.7 (25.6-29.4)	0.709	0.085	0.018	0.06†
Pulse (bpm)	68 (65-76)	70 (64-80)	72 (65-79)	0.676	0.128	0.340	0.336†
Sys BP (mm Hg)	110 (100-115)	140 (138-146)	160 (154-162)	0.000	0.000	0.000	0.000†
Dia BP(mm Hg)	65 (60-75)	90 (80-92)	110 (100-110)	0.000	0.000	0.000	0.000†
Glucose (mg/dl)	84 (77-92)	83 (75-90)	87 (75-91)	0.169	0.619	0.550	0.419†
Creatinin (mg/dl)	0.8 (0.7-0.9)	0.8 (0.7-1)	0.9 (0.8-1.2)	0.512	0.000	0.000	0.000†
Haemoglobin (g/dl)	12 (11-14)	12 (11-14)	12 (11-14)	0.158	0.935	0.238	0.311†
LDL (mg/dl)	90 (78-115)	95 (84-111)	98 (85-120)	0.584	0.101	0.154	0.199†
HT duration (year)	-	5 (3-8.2)	7 (0-12)	0.000	0.000	0.517	0.000†
HT retinopathy	-	Grade 1: 49 (46%) Grade 2: 16 (15%) Grade 3: 4 (3.7%)	Grade 1: 29 (35%) Grade 2: 16 (19%) Grade 3: 5 (6%)	-	-	0.000§	-
ONSD	4.7 (4.4-5)	4.7 (4.4-5.1)	5.3 (4.7-5.5)	0.762	0.000	0.000	0.000†
Glob_dia	22.5 (21.8-23)	22.5 (21.8-23)	22.5 (21.7-23)	0.634	0.647	0.955	0.862†
ONSD/Glob_dia	0.21 (0.2-0.22)	0.21 (0.19-0.23)	0.24 (0.21-0.25)	0.860	0.000	0.000	0.000†
Female n=147	n=51	n=55	n=41	0.798	0.000	0.000	0.000
ONSD	4.7 (4.4-5.1)	4.5 (4.4-5)	5.2 (4.7-5.5)	0.706	0.875	0.911	0.939
Glob_dia	22.5 (22-22.9)	22.5 (21.5-23)	22.5 (21.6-23.2)	1	0.003	0.000	0.000
ONSD/Glob_dia	0.21 (0.20-0.23)	0.20 (0.19-0.22)	0.23 (0.21-0.25)				
				0.349	0.000	0.000	0.000
Male n=143	n=51	n=51	n=41	0.806	0.560	0.928	0.881
ONSD	4.7 (4.4-4.9)	4.7 (4.4-5.1)	n=5.3 (4.9-5.5)	0.458	0.000	0.000	0.000
Glob_dia	22.5 (21.8-23.1)	22.5 (22-23.1)	22.5 (21.8-22.9)				
ONSD/Glob_dia	0.21 (0.20-0.22)	0.21 (0.20-0.23)	0.24 (0.21-0.25)				

*Median (ınterquartile range, IQR)
BMI: Body mass index Sys BP: Systolic blood pressure Dia BP: Diastolic blood pressure HT: Hypertension RHT: Resistant Hypertension ONSD: Optic nerve sheat diameter Glob_dia: Ocular globe diameter p†(Kruskal Wallis test)§ (Chi-square test) #Mann Whitney U test

Hypertension

OP-062

Hidden enemy: Masked hypertension in women with a history of preeclampsia

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Background and Aim: Women with a history of preeclampsia during pregnancy have four times higher risk of developing chronic hypertension after pregnancy than those with a healthy pregnancy, but cases of masked hypertension may not be diagnosed with clinical evaluation and office blood pressure measurements alone.Twenty-four hour ambulatory blood pressure monitoring (ABPM) is the reference-standard for confirmation of hypertension diagnoses or detection of masked hypertension outside of clinical settings. The aim of our study is to investigate the frequency of masked hypertension in women with a history of preeclampsia.

Methods: A total of 70 young women with a history of preeclampsia (n=35) and a healthy pregnancy (n=35) underwent blood pressure monitoring with office measurements and 24-hour ABPM 1 to 3 years postpartum. Women with chronic hypertension and using antihypertensive drug were excluded. Hypertension was defined according to ESC/ESH 2018 Guidelines for the Management of Hypertension (the diagnostic threshold for hypertension is ≥130/80 mm Hg over 24 h, ≥135/85 mm Hg for the daytime average, and ≥120/70 for the nighttime average).

Results: Women with a history of preeclampsia did not differ from controls in age (p=0.18), body mass index (p=0.15), or postpartum duration (p=0.73). Office blood pressure values of all patients were within normal limits. The rate of postpartum masked hypertension was higher in patients with a history of preeclampsia compared to those with a healthy pregnancy history (38% vs. 25%, p<0.05). Isolated diastolic hypertension was the most common finding (60%) in women diagnosed with hypertension in ABPM results. Nondipping pattern was observed more frequently in women with a history of preeclampsia (25% vs. 17%, p<0.05). Masked hypertension was associated with non-dipper hypertension (p=0.03).

Conclusions: In our study, we showed that the frequency of masked hypertension is higher in women with a history of preeclampsia than healthy control group. Only office blood pressure measurements are not sufficient to diagnose chronic hypertension, particularly cases of isolated diastolic hypertension in this patient group, and it is important to evaluate the patients by using 24-hour ABPM, together with closer follow-up.

Hypertension

OP-063

The role of left atrial volume index and silent embolic cerebral infarction in predicting cardiovascular and cerebrovascular events in hypertensive patients

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Background and Aim: Hypertension is associated with silent embolic cerebral infarction (SECI) and many structural heart changes such as left atrial (LA) enlargement. This study examined the relationship between LA enlargement and SECI in patients with hypertension and major adverse cardiac and cerebrovascular events (MACCE) in long-term follow-up.

Methods: Three hundred sixty hypertensive patients and 101 controls were included. Left atrial volume indexes (LAVI) were calculated by using transthoracic echocardiography with the biplane area length method and abnormal LAVI was defined as ≥ 34 mL/m^2. SECIs were defined on the deep white matter using MRI. All patients were followed up for an average of 29.3 months, and MACCE were evaluated.

Results: Higher LAVI values were measured in the hypertensive group than in the control group (31.3 ± 5.1 vs. 27.1 ± 3.7, p<0.001) and there was a significant association between abnormal LAVI and the development of MACCE (p=0.026). The incidence of MACCE in patients with abnormal LAVI (14.6%) was significantly higher than the patients with normal LAVI (6.4%) and the control group (4.0%). There was a significant association between the presence of SECI and the development of MACCE (p=0.048). In patients with SECI, 11.9% developed MACCE during the follow-up time. This incidence was significantly higher than the patients without SECI (5.5%).

Conclusions: Increased LAVIs and SECI were associated with various adverse cardiovascular events. Especially in hypertensive individuals without any arrhythmia, changes in LA volumes are powerful predictors of future cardiac events.

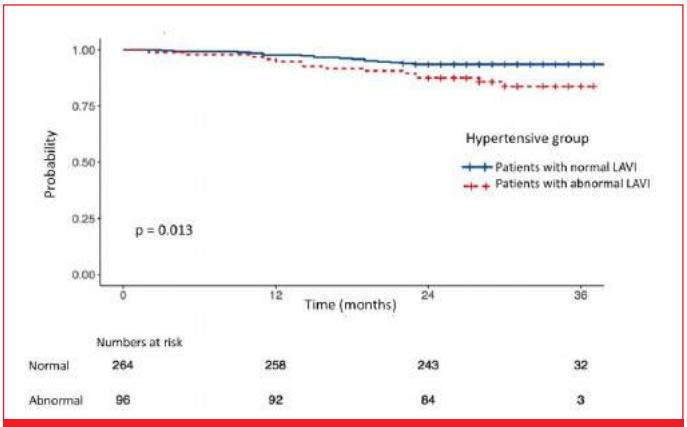

Figure 1. Kaplan-Meier plot of time to the MACCE probability in patients with abnormal LAVI compared with the patients with normal LAVI (p=0.013).

Table 1. Demographic and clinical characteristics of the study groups.

	Hypertensive group (n=360)	Control group (n=101)	p value
Age (year) [†]	51.8 ± 8.1	44.8 ± 8.7	**<0.001***
Sex [‡]			
Female	262 (72.8)	61 (60.4)	**0.023****
Male	98 (27.2)	40 (39.6)	
BMI (kg/m²) [†]	31.3 ± 5.4	28.7 ± 5.1	**<0.001***
Smoking [‡]			
Non-smoker	245 (68.1)	56 (55.4)	**0.041****
Ex-smoker	52 (14.4)	17 (16.8)	
Smoker	63 (17.5)	28 (27.7)	
Alcohol [‡]	1 (0.3)	1 (1.0)	0.391**
Diabetes mellitus [‡]	35 (9.7)	4 (4.0)	0.102**
Drugs [‡]			
ACE inhibitors	73 (20.3)	--	--
ARB	89 (24.7)	--	--
Calcium channel blockers	87 (24.2)	--	--
Diuretics	90 (25.0)	--	--
Beta-blockers	27 (7.5)	--	--
Laboratory parameters			
Total cholesterol [†]	197.3 ± 42.7	178.8 ± 33.7	**<0.001***
LDL [§]	121.0 [52.0- 247.0]	118.0 [76.0- 206.0]	0.360**
Triglyceride [§]	176.0 [45.0- 497.0]	177.0 [69.0- 344.0]	0.211**
HDL [†]	43.2 ± 10.6	38.7 ± 6.5	**<0.001***
LAVI (mL/m²) [†]	31.3 ± 5.1	27.1 ± 3.7	**<0.001***
SECI [‡]	177 (49.2)	-	-
MACCE [‡]	31 (8.6)	4 (4.0)	0.178***

[†]: mean ± standard deviation, [‡]: n (%), [§]: median [min-max].
BMI: Body mass index, ACE: Angiotensin-converting enzyme, ARB: Angiotensin type2 receptor blockers, LDL: Low-density lipoprotein cholesterol, HDL: High-density lipoprotein cholesterol. LAVI: Left atrial volume index, SECI: Silent embolic cerebral infarction, MACCE: Major adverse cardiac and cerebrovascular events
**: Independent Samples t-test*
***: Mann-Whitney U test*
****: Pearson Chi-Square or Fisher's Exact test*

Table 2. Comparison of hypertensive patients with normal and abnormal LAVI.

	Hypertensive group		
	Patients with normal LAVI (n=264)	Patients with abnormal LAVI (n=96)	p value
Age (year) [†]	50.7 ± 8.2	54.6 ± 7.4	**<0.001***
Sex [‡]			
Female	195 (73.9)	67 (69.8)	0.526**
Male	69 (26.1)	29 (30.2)	
BMI (kg/m²) [†]	31.0 ± 5.1	31.9 ± 6.2	0.237*
Smoking [‡]			
Non-smoker	177 (67.0)	68 (70.8)	0.675**
Ex-smoker	38 (14.4)	14 (14.6)	
Smoker	49 (18.6)	14 (14.6)	
Alcohol [‡]	1 (0.4)	0 (0.0)	0.999**
Diabetes mellitus [‡]	24 (9.1)	11 (11.5)	0.639**
Drugs [‡]			
ACE inhibitors	56 (21.2)	17 (17.7)	0.560**
ARB	63 (23.9)	26 (27.1)	0.625**
Calcium channel blockers	60 (22.7)	27 (28.1)	0.358**
Diuretics	59 (22.3)	31 (32.3)	0.074**
Beta blockers	21 (8.0)	6 (6.2)	0.751**
Laboratory parameters			
Total cholesterol (mg/dL) [†]	198.7 ± 43.8	193.3 ± 39.5	0.272*
LDL (mg/dL) [§]	121.0 [52.0- 247.0]	120.5 [65.0- 225.0]	0.918**
Triglyceride (mg/dL) [§]	176.0 [51.0- 497.0]	177.0 [45.0- 425.0]	0.488**
HDL (mg/dL) [†]	43.7 ± 11.1	41.9 ± 9.1	0.119*
SECI [‡]	122 (46.2)	55 (57.3)	0.082**
MACCE [‡]	17 (6.4)	14 (14.6)	**0.026****

[†]: mean ± standard deviation, [‡]: n (%), [§]: median [min-max].
BMI: body mass index, ACE: angiotensin-converting enzyme, ARB: angiotensin type2 receptor blockers, LDL: low-density lipoprotein cholesterol, HDL: high-density lipoprotein cholesterol, SECI: silent embolic cerebral infarction, MACCE: Major adverse cardiac and cerebrovascular events
**: Independent Samples t-test*
***: Mann-Whitney U test*

Table 3. Comparison of the incidences of MACCE in the hypertensive group with normal and abnormal LAVI and the control group.

	Patients with normal LAVI (n=264)	Patients with abnormal LAVI (n=96)	Control group (n=101)	p value
MACCE [‡]	17 (6.4)	14 (14.6)	4 (4.0)	**0.011****

[‡]: n (%).
LAVI: left atrial volume index. MACCE: Major adverse cardiac and cerebrovascular events
****: Pearson Chi-Square or Fisher's Exact test*

Table 4. Comparison of hypertensive patients with and without SECI.

	SECI		
	Positive (n=177)	Negative (n=183)	p value
Age (year) [†]	53.4 ± 7.3	50.1 ± 8.6	**<0.001***
Sex [‡]			
Female	131 (74.0)	131 (71.6)	0.690**
Male	46 (26.0)	52 (28.4)	
BMI (kg/m²) [†]	31.4 ± 5.5	31.1 ± 5.3	0.631*
Smoking [‡]			
Non-smoker	132 (74.6)	113 (61.7)	**0.023****
Ex-smoker	18 (10.2)	34 (18.6)	
Smoker	27 (15.3)	36 (19.7)	
Alcohol [‡]	1 (0.6)	0 (0.0)	0.492**
Diabetes mellitus [‡]	15 (8.5)	20 (10.9)	0.543**
Drugs [‡]			
ACE inhibitors	36 (20.3)	37 (20.2)	0.999**
ARB	45 (25.4)	44 (24.0)	0.856**
Calcium channel blockers	41 (23.2)	46 (25.1)	0.754**
Diuretics	50 (28.2)	40 (21.9)	0.201**
Beta blockers	9 (5.1)	18 (9.8)	0.131**
Laboratory parameters			
Total cholesterol (mg/dL)	195.0 ± 40.7	199.4 ± 44.6	0.327*
LDL (mg/dL) [§]	123.0 [56.0- 245.0]	120.0 [52.0- 247.0]	0.436**
Triglyceride (mg/dL) [§]	178.0 [55.0- 340.0]	168.0 [45.0- 497.0]	**0.035****
HDL (mg/dL)	40.9 ± 9.8	45.4 ± 10.9	**<0.001***
LAVI (mL/m²) [†]	31.8 ± 5.5	30.6 ± 4.6	**0.027***
MACCE [‡]	21 (11.9)	10 (5.5)	**0.048****

[†]: mean ± standard deviation, [‡]: n (%), [§]: median [min-max].
SECI: silent embolic cerebral infarction, BMI: body mass index, ACE: angiotensin converting enzyme, ARB: angiotensin type2 receptor blockers, LDL: low-density lipoprotein cholesterol, HDL: high-density lipoprotein cholesterol, LAVI: Left atrial volume index, MACCE: Major adverse cardiac and cerebrovascular events
**: Independent Samples t test*
***: Mann-Whitney U test*
****: Pearson Chi-Square or Fisher's Exact test*

Heart valve diseases

OP-064

Combination strategy during first trimester and continuation therapy in pregnant patients with mechanical heart valves: The KYBELE Study

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Background and Aim: The prothrombotic state of pregnancy increases the risk of thromboembolic complications and death in women with mechanical heart valves (MHV). This study aims to determine the optimal anticoagulation regimens for pregnant women with MHVs.

Methods: All women were allocated to one of three treatment options during first trimester including, enoxaparin only (Group 1), enoxaparin plus fixed dose of warfarin 2.5 mg (Group 2), and enoxaparin plus fixed dose of warfarin 4 mg (Group 3). Inclusion and exclusion criteria, and the flow chart of the study are summarized in Figure 1. Patients clearly declaring that their concern about fetal risks outweighs the maternal thromboembolic risk were allocated to Group 1. All other patients were allocated to Group 2 or Group 3. The choice between low dose warfarin 2.5 and 4 mg was based on pre-pregnancy need of warfarin dose. For women whose pre-pregnancy warfarin dose was 5 to 7.5 mg, Group 2 was opted, and whose pre-pregnancy warfarin dose was more than 7.5 mg, Group 3 was opted in the first trimester. The target anti-Xa was 0.8-1.2 for Group 1 similar to guideline recommendations. However, combination therapy patients, the target anti-Xa was 0.7-1.A combination of death from any cause and non-fatal events was used as the maternal outcome measure. An overview of the study algorithm was demonstrated in Figure 2. The non-fatal events included thromboembolism, severe bleeding (intracranial or blood transfusion requirement), need for thrombolytic therapy or need for in-hospital intravenous unfractionated heparin therapy. Due to the observational nature of the study, formal comparative statistics were not performed; hence, P values were not reported.

Results: A total 78 pregnancies of 65 women with MHV's were included in the study. The position of MHV was mitral in 47 (60.3%), aortic in 15 (19.2), and combined mitral and aortic in 17 (20.5) patients (Table 1). Group 1 had the high rates of high thrombus formation or increase (52%), MVT requiring treatment (24%), cerebral embolism (16%), and combined outcome (36%) in the first trimester (Table 2). Moreover, Group 1 had also the highest incidence of combined maternal outcome (40%) throughout pregnancy, followed by Group 2 (12.5%). The lowest rate was in Group 3 (3.4%) (Table 3). When only the first trimester was considered, the corresponding values were 36%, 4.2%, and 3.4%, respectively. Of note, 11 (78.5%) of 14 events occurred within the first trimester. Maternal death occurred in only 2 cases, one being in Group 1 and the other in Group 2. Fetal loss occurred in a total of 27 (34.6%) pregnancies. The incidences of fetal loss did not differ between the groups. However, early abortion occurred in only 1 (12.5%) case in Group 1, in 5 (62.5%) cases in Group 2, and 9 (81.8%) in Group 3 patients (Table 3).

Conclusions: Combined anticoagulation strategy during first trimester is associated with low maternal complication and acceptable fetal outcomes in patients with MHVs.

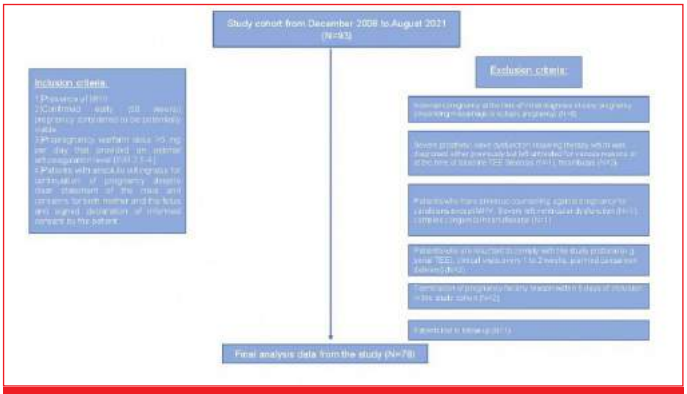

Figure 1.

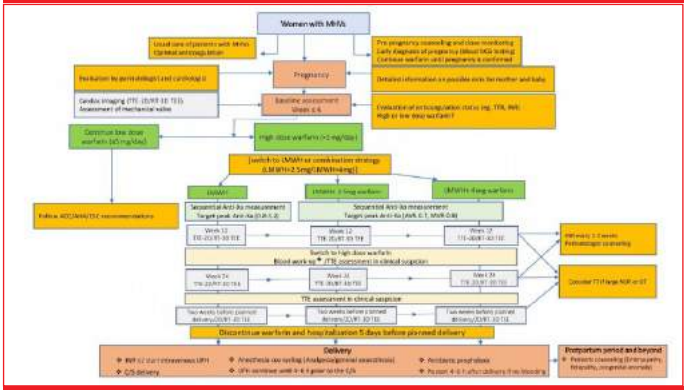

Figure 2. An overview of the study algorithm was demonstrated in Figure 2

Table 1. Maternal clinical, obstetric and echocardiographic characteristics as per study groups

Maternal clinical, obstetric and echocardiographic characteristics	Group 1 (n=25)	Group 2 (n=24)	Group 3 (n=29)
Age, median (IQR)	31 (26.5-35)	32 (30-37)	31 (27-35)
Mechanical valve position, n (%) Aortic Mitral Combined	4 (16) 15 (60) 6 (24)	4 (16.7) 16 (66.7) 4 (16.7)	7 (24.1) 16 (55.2) 6 (20.7)
Redo surgery, n (%)	3 (12)	4 (16.7)	1 (3.4)
Elapsed time since valve surgery (months)	89 (49-148)	111 (64-181)	137 (93-189)
Atrial fibrillation, n (%)	3 (12)	2 (8.3)	1 (3.4)
Diabetes Mellitus, n (%)	0 (0)	1 (4.2)	0 (0)
Hypertension, n (%)	2 (8)	0 (0)	1 (3.4)
Smoking, n (%)	1 (4)	2 (8.3)	3 (10.3)
Coronary artery disease, n (%)	2 (8)	1 (4.2)	0 (0)
Left ventricular dysfunction, n (%)	0 (0)	2 (8.3)	0 (0)
Thyroid dysfunction, n (%) Hypothyroidism Hyperthyroidism	3 (12) 1 2	1 (4.2) 1 0	3 (10.3) 3 0
Previous thromboembolism, n (%)	5 (20)	5 (20.8)	9 (31)

Table 1. Maternal clinical, obstetric and echocardiographic characteristics as per study groups (Continued)

Maternal clinical, obstetric and echocardiographic characteristics	Group 1 (n=25)	Group 2 (n=24)	Group 3 (n=29)
Congenital anomaly, n (%)	0 (0)	2 (8.3)	1 (3.4)
Previous fetal loss, n (%) One loss Two losses Three losses Four losses	17 (68) 9 4 2 2	17 (70.8) 4 12 1 0	18 (62.1) 7 7 3 1
Gravida	2 (1.5-3.5)	3 (2-3)	2 (1-3)
Para	1 (1-2)	1 (1-2)	1 (1-2)
Abortion	1 (0.5-1.5)	1 (0-2)	1 (0-1.5)
Gestational week at enrollment	5 (4-6)	4 (4-5)	5 (4-6)
Non-severe paravalvular leakage	1 (4)	1 (4.2)	2 (6.9)
Non-obstructive MHV Pannus	3 (12)	3 (12.5)	1 (3.4)
Prosthesis patient mismatch	0 (0)	1 (4.2)	1 (3.4)
Nonobstructive MHV thrombus Ring thrombosis Large thrombus	17 (68) 16 1	17 (70.8) 16 1	17 (58.6) 15 2

Table 2. Maternal and fetal 1st trimester events as per study groups *Four patients were treated with slow infusion 25 hours) of low dose (25 mg) alteplase. The remaining two patients were treated with 2-4 weeks of unfractionated heparin infusion

Endpoints	Group 1 (n=25)	Group 2 (n=24)	Group 3 (n=29)
Thrombus formation or increase, n (%)	13 (52)	2 (8.3)	0 (0)
Obstructive thrombus, n (%)	3 (12)	0 (0)	0 (0)
Treatment for MHV thrombus, n (%)	6 (24)*	1 (4.2)	0 (0)
Bleeding, n (%)	2 (8)	2 (8.3)	4 (13.8)
Intracranial haemorrhage, n (%)	1 (4)	0 (0)	0 (0)
Cerebral embolism, n (%)	4 (16)	0 (0)	1 (3.4)
Maternal death, n (%)	1 (4)	0 (0)	0 (0)
Combined outcome, n (%)	9 (36)	1 (4.2)	1 (3.4)
Fetal loss (early abortion), n (%)	1 (4)	5 (20.8)	9 (31)

Table 3. Maternal and fetal whole pregnancy outcomes as per study groups

Endpoints	Group 1 (n=25)	Group 2 (n=24)	Group 3 (n=29)
New thrombus formation or increase in thrombus burden, n (%)	15 (60)	3 (12.5)	3 (10.3)
Obstructive thrombus, n (%)	4 (16)	0 (0)	0 (0)

Table 3. Maternal and fetal whole pregnancy outcomes as per study groups (Continued)

Endpoints	Group 1 (n=25)	Group 2 (n=24)	Group 3 (n=29)
Treatment for MHV thrombus, n (%)	7 (28)	1 (4.2)	0 (0)
Minor bleeding, n (%)	2 (8)	2 (8.3)	5 17.2)
Major bleeding, n (%)	1 (4)	0 (0)	0 (0)
C/S related major bleeding, n (%)	0 (0)	0 (0)	1 (3.4)
Fetal loss, n (%) Early abortion Late abortion Still birth	8 (32) 1 (4) 5 (20) 2 (8)	8 (33.3) 5 (20.8) 1 (4.2) 2 (8.3)	11 (37.9) 9 (31) 0 (0) 2 (6.9)
Warfarin-related congenital anomaly, n (%)	1 (4)	2 (8.3)	0 (0)
Polyhydramnios, n (%)	0 (0)	1 (4.2)	0 (0)
Placenta previa, n (%)	2 (8)	0 (0)	0 (0)
Preeclampsia, n (%)	3 (12)	0 (0)	0 (0)
Gestational age at birth (week)	37 (35.5-38)	37.5 (37-38)	38 (37-38)
Weight at birth (kg)	2.96 (2.41-3.05)	3 (2.46-3.34)	2.9 (2.68-3.18)
Atrial arrhythmia, n (%)	0 (0)	2 (8.3)	2 (6.9)
Ventricular arrhythmia, n (%)	0 (0)	4 (16.7)	0 (0)
Intracranial haemorrhage, n (%)	1 (4)	0 (0)	0 (0)
Cerebral embolism, n (%)	6 (24)	1 (4.2)	1 (3.4)
Maternal death, n (%)	1 (4)	1 (4.2)	0 (0)
Combined outcome, n (%)	10 (40)	3 (12.5)	1 (3.4)

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-065

Evaluation of V2S/V3R index algorithm in patients who underwent successful ablation for symptomatic premature ventricular complex originated from outflow tract

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Background and Aim: Premature Ventricular Complex (PVC) is the most common ventricular arrhythmia in the community. Idiopathic PVCs originate mostly from the right (RVOT) and left ventricular outflow tract (LVOT). V2S/V3R index is one of the RVOT-LVOT differentiating algorithms. This algorithm predicts that PVC originates from LVOT If the ratio of the amplitude of the S wave in lead V2 and the amplitude of the R wave in lead V3 is less than or equal to 1.5 on the 12-lead ECG. The sensitivity and the specificity are 89% and 94% respectively. In our study, we investigated the diagnostic accuracy of this algorithm in patients undergoing PVC ablation in our center.

Methods: In this study, we analyzed ECG recordings of 505 patients underwent catheter ablation for PVC between 01.01.2015 and 20.01.2022. Non-outflow tract PVCs, underlying structural heart disease, bundle branch block in baseline ECG, unsuccessful ablation, polymorphic and presence of prior catheter ablation history were exclusion criteria. Remaining 17 of 217 patients have right bundle branch block (RBBB) pattern PVCs (R>S in V1). 190 patients with left bundle branch (LBBB) and inferior axis (R<S in V1 and positive QRS complexes in D2, D3 and aVF) were analyzed with their preprocedural 12 derivation ECG recordings. Prucka CardioLab™ Electrophysiology Recording System (GE Healthcare) was used to record and analyze PVC templates saved before the procedure. Antiarrhythmic drugs were discontinued for at least 5 half-lives before the procedure and written informed consent was taken from each patient. Statistical analysis was made with IBM SPSS 24.0.

Results: At the end of the procedure, RVOT PVC ablation was performed in 94 of 190 patient. LVOT PVC ablation count was 96. PVC V2 S amplitude was significantly lower in LVOT group (RVOT: 2.01 ± 0.21, LVOT: 1.22 ± 0.53 p<0.001). PVC V3 R amplitude was significantly higher in LVOT group (RVOT: 0.71 ± 0.22, LVOT: 1.39 ± 0.65). V2S/V3R index was significantly lower in LVOT group (LVOT: 1.38 ± 0.56, RVOT: 4.37 ± 0.89). Specifity and sensitivity of V2S/V3R in our group were 89.4% and 67.7% respectively. Positive and negative predictive values were 84% and 73% respectively.

Conclusions: In our study with patients underwent ablation procedure for outflow tract PVC with LBBB morphology and inferior axis, the S wave in lead V2 is smaller in LVOT PVC than in RVOT PVC, and the R wave in lead V3 is larger in LVOT PVC than in RVOT PVC. The V2S/V3R algorithm was found to have lower sensitivity in our study population, but similar specificity. Although this algorithm can give false positive or false negative results in patients with transition zone V3, it is an easy-to-use algorithm that is not complicated to measure and implement. New technologies like artificial intelligence instead of traditional methods can be used to predict the site of origin more precisely.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-066

The high-risk coexistence of atrial fibrillation and hypertension in Middle Eastern patients: Analysis from the Jordan Atrial Fibrillation Study

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Background and Aim: Clinical studies on the impact of hypertension (HTN) on the outcome of patients with atrial fibrillation (AF) in the Middle East are scarce. The aim of this contemporary multicenter study is to evaluate the effect of coexisting HTN on the baseline clinical profiles and one-year prognosis in a cohort of Middle Eastern patients with AF.

Methods: Consecutive AF patients in 29 hospitals and cardiology clinics were enrolled in the Jordan AF (May 2019 - December 2020). Patients were followed up for one year. We compared clinical features, use of medications and 1-year prognosis in patients with AF/HTN compared with AF/no HTN.

Results: Of 2020 AF patients enrolled; 1506 (74.6%) had HTN. Patients with HTN were 10 years older than those with no HTN (mean age ± SD 70.3 ± 10.4 years vs. 60.7 ± 16.6 years, p<0.0001), were more likely to be women and had significantly higher prevalence of diabetes, dyslipidemia, coronary heart disease and left ventricular hypertrophy. Valvular AF was less prevalent in HTN vs. non HTN patients (6.2% vs. 15.2%, p<0.0001). Mean CHA2DS2-VASc and HAS-BLED scores were higher in HTN patients than those with no HTN (4.1 ± 1.6 vs. 2.1 ± 1.6, p<0.0001), and (1.8 ± 1.1 vs. 1.2 ± 1.0, p<0.0001), respectively. More eligible HTN patients received oral anticoagulant agents (87.8% vs. 68.5%, p<0.0001). At one year, HTN patients, compared with no HTN patients, had significantly higher rates of incident stroke/systemic embolism (5.1% vs. 2.7%, p=0.04), acute coronary syndrome (2.4% vs. 0.6%, p=0.02), hospital admission for cardiac cause (15.3% vs. 11.0%, p=0.03%), and major bleeding events (3.0% vs. 1.2%, p=0.05). Intracranial bleeding occurred in higher, but not statistically significant, number in HTN patients (0.6% vs. 0.2%, p=0.48). Multiple logistic regression analysis showed that HTN was associated with higher incidence of stroke and systemic embolism (odds ratio 1.9, 95% confidence interval 1.0-3.8, p=0.05).

Conclusions: In this cohort of Middle Eastern patients with AF, the coexistence of HTN was associated with worse baseline clinical profile and one-year outcome.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-067

Evaluation of cardiac electrophysiological balance according to admission and discharge NIHSS score in acute ischemic stroke patients: A pilot study

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Background and Aim: The main objectives of this investigation were to determine whether there were any relationships between corrected cardiac-electrophysiological balance (iCEBc) value and admission and discharge National Institutes of Health Stroke Scale Score (NIHSS) scores in patients with acute ischemic stroke (AIS), and whether iCEBc value was an independent predictor of high NIHSS scores (NIHSS score ≥ 5).

Methods: In this retrospective and observational study, 231 consecutive adult patients with AIS were evaluated. The iCEBc value was obtained by dividing the corrected QT interval (QTc) by the QRS duration measured from surface electrocardiography. An experienced neurologist used the NIHSS score to determine the severity of the stroke at the time of admission and before discharge from the neurology care unit. The participants in the research were categorized into two groups: those with minor AIS (NIHSS score = 1-4) and those with moderate to severe AIS (NIHSS score ≥ 5).

Results: AIS patients with NIHSS score ≥ 5 had higher heart rate, QT, QTc, Tp-e/QTc, iCEB, and iCEBc values compared with those with NIHSS 1-4. iCEBc value was shown to be independently related to NIHSS score ≥ 5 (OR:1.102, 95%CI:1.036–1.172, p<0.001). There was a moderate correlation between iCEBc and admission (r = 0.333, p<0.001) and discharge (r = 0.329, p<0.001) NIHSS scores.

Conclusions: The findings of this study demonstrated that iCEBc value was related to NIHSS admission and discharge scores. Furthermore, elevated iCEBc value was found to be an independent predictor of NIHSS score ≥ 5.

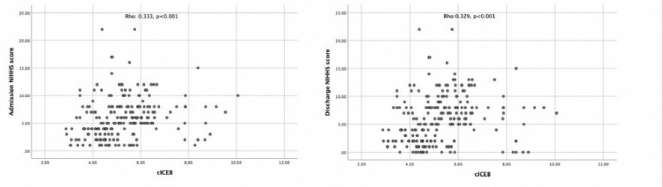

Figure 1.

Table 1. Univariable and multivariable model for prediction of admission National Institutes of Health Stroke Scale Score ≥ 5

	Univariable analysis		Multivariable analysis	
	P value	OR (95%CI)	P value	OR (95%CI)
Age	0.017	1.024 (1.004 – 1.043)	0.004	1.032 (1.010 – 1.054)
Smoking	0.020	2.345 (1.145 – 4.804)	0.011	2.835 (1.272 – 6.320)
LAVI	0.078	1.051 (0.994 – 1.111)	0.034	1.068 (1.005 – 1.135)
Heart rate	0.005	1.022 (1.007 – 1.038)	-	-
QRS	0.072	0.987 (0.974 – 1.001)	-	-
QTc	<0.001	1.016 (1.011 – 1.022)	-	-
iCEBc	<0.001	1.971 (1.503 – 2.587)	<0.001	1.102 (1.036 – 1.172)

All clinically relevant parameters were included in the model.

OR, Odds ratio; CI, confidence interval.

Abbreviations: LAVI, left atrium volume index

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-068

Single nucleotide polymorphism at chromosome 4q25 may predict long-term recurrence after successful electrical cardioversion for persistent atrial fibrillation

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Background and Aim: Genom-wide association studies have demonstrated that a single nucleotide polymorphism (SNP) at chromosome 4q25 are associated with atrial fibrillation (AF) recurrence after successful direct-current cardioversion (DCCV). However, there is no data related to the genetic predictors of AF recurrence after DCCV in the Turkish population, and there is not enough data related to other SNPs in this topic. We aimed to investigate whether 11 common AF-related SNPs predicted AF recurrence after successful DCCV in Turkish patients (Table 1).

Methods: A total of 75 patients with persistent AF who obtained sinus rhythm after DCCV were enrolled in the study. Patients were prospectively followed for AF recurrence after DCCV. Baseline clinical features and SNPs were compared between patients with AF recurrence and those without recurrence. Association between SNPs and AF recurrence was assessed by the additive model (wild type vs. heterozygous variant vs. homozygous variant), dominant model (wild type vs. heterozygous and homozygous variant), and recessive model (homozygous variant vs. heterozygous variant and wild type).

Results: AF recurrence developed in 37 patients (50.7%) in the follow-up of 17.0 (11.0-25.0) months. The percentage of female sex was higher (55.3% vs. 32.4%), and AF duration was longer [9.2 (5.5-11.6) vs. 5.0 (2.7-11.0) months] in patients with AF recurrence than in those without recurrence (p=0.046 and 0.027, respectively) (Table 2). Drug use was similar between the groups. One SNP in PITX2 locus (rs17570669_T: OR 9.00, 95% CI 1.28-63.02, p=0.027) and one SNP in ZFHX3 locus (rs2106261_T: OR 8.96, 95% CI 1.03-77.66, p=0.047) were significantly associated with AF recurrence in additive model (Table 1). Cox regression analysis demonstrated that one SNP (rs17570669_T) was found to be independently associated with AF recurrence following DCCV in the additive model (OR: 3.75, 95% CI: 1.10-12.77, p=0.034) (Table 3). A Kaplan-Meier curve showed significantly lower survival without AF recurrence in carriers of the rs17570669_T SNP in the additive model (Figure 1).

Conclusions: A SNP in the PITX2 locus (rs17570669_T) is an independent predictor of long-term AF recurrence after successful DCCV in Turkish patients with persistent AF.

Table 1. Relationship between studied SNPs and AF recurrence after successful electrical cardioversion in a binary logistic regression analysis.

			Dominant model		Additive model		Recessive model	
Chromosome band	**Nearest gene**	**SNP**	**OR (95% CI)**	**P**	**OR (95% CI)**	**P**	**OR (95% CI)**	**P**
4q25	PITX2	rs2200733	1.42 (0.46-4.32)	0.534	1.55 (0.50-4.79)	0.443	0.46 (0.08-2.67)	0.385
4q25	PITX2	rs10033464	0.80 (0.19- 3.24)	0.755	0.85 (0.20- 3.53)	0.834	0.60 (0.15- 2.35)	0.472
4q25	PITX2	rs6838973	0.75 (0.24- 2.28)	0.615	2.33 (0.19- 27.56)	0.501	3.08 (0.30- 31.10)	0.339
4q25	PITX2	rs3853445	0.80 (0.19- 3.24)	0.755	0.40 (0.03- 6.17)	0.512	0.47 (0.04-5.45)	0.548
4q25	PITX2	rs17570669	1.09 (0.97- 1.23)	0.124	9.00 (1.28-63.02)	0.027	4.66 (0.91- 23.68)	0.063
16q22	ZFHX3	rs2106261	8.63 (1.01-74.10)	0.049	8.96 (1.03- 77.66)	0.047	0.97 (0.25- 3.67)	0.964
8q21	EPHX2	rs751141	0.53 (0.20- 1.36)	0.189	0.50 (0.19- 1.30)	0.158	Not analyzed	
7q31	CAV1	rs3807989	3.21 (0.78- 13.25)	0.105	5.33 (0.89– 31.91)	0.067	2.20 (0.60– 8.05)	0.234
12q24	TBX5	rs10507248	0.58 (0.17– 1.99)	0.392	0.25 (0.50- 1.25)	0.092	0.31 (0.90– 1.12)	0.076
19q13	TGF-1	rs1800469	1.02 (0.19- 5.46)	0.973	1.03 (0.19- 5.59)	0.966	0.96 (0.30– 3.09)	0.956
3q22	SCN10A	rs6795970	0.54 (0.17– 1.68)	0.290	0.50 (0.15- 1.62)	0.252	1.20 (0.33- 4.33)	0.781

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

Table 2. Baseline and follow-up characteristics of the study population according to AF recurrence.

	AF recurrence (-) (n=37)	**AF recurence (+) (n=38)**	**P**
Age (years)	60.4 ± 12.8	63.3 ± 10.0	0.279
Gender (female)	12 (32.4)	21 (55.3)	0.046
BMI (kg/m^2)	27.7 (26.2-31.9)	28.6 (26.6–33.0)	0.203
Hypertension (n, %)	23 (62.2)	27 (71.1)	0.414
Diabetes mellitus (n, %)	10 (27.0)	6 (15.8)	0.235
Stroke/ TIA history (n, %)	3 (8.1)	3 (7.9)	1.000
Heart failure with reduced EF (n, %)	15 (40.5)	12 (31.6)	0.419
CHA2DS2VASc score	3.0 (1.0-4.0)	2.0 (2.0–3.2)	0.807
AF duration (months)	5.0 (2.7–11.0)	9.2 (5.5–11.6)	0.029
Follow-up duration (months)	14.0 (9.5–24.0)	20.0 (11.7–28.0)	0.124
Hemoglobin (g/dl)	14.3 ± 1.8	13.7 ± 1.8	0.144
eGFR (mL/ min/1.73 m^2)	80.0 (59.5–90.0)	74.2 (59.7–90.0)	0.491
LV EF (%)	55.0 (40.0–60.0)	58.5 (41.5–62.0)	0.488
LA dimeter (mm)	42.4 ± 6.7	44.1 ± 4.6	0.202

AF: Atrial fibrillation, BMI: Body mass index, EF: Ejection fraction, eGFR: Estimated glomerular filtration rate, LA: Left atrium, LV: Left ventricle, TIA: Transient ischemic attack.

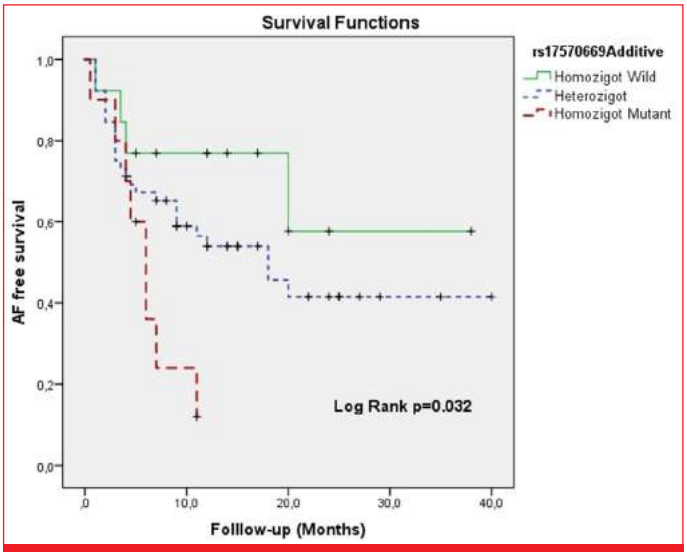

Figure 1. Kaplan-Meier curve showing AF-free survival during the follow-up period. Single nucleotide polymorphism (rs17570669_T) in additive model and cumulative survival. Green: Wild type, Blue: Heterozygous carriers, Red: Homozygous carriers. AF: Atrial fibrillation.

Table 3. Parameters predicting AF recurrence using Cox regression analysis.

	Univariate			Multivarate		
	Beta	**HR (95% CI)**	**P**	**Beta**	**HR (95% CI)**	**P**
Female	0.691	1.99 (1.05–3.79)	0.035	0.637	1.89 (0.99 – 3.61)	0.053
AF duration	0.028	1.02 (0.97–1.08)	0.313			
Hypertension	0.315	1.37 (0.67–2.77)	0.380			
Body mass index	0.040	1.04 (0.98–1.10)	0.154			
SNP rs2106261_T	1.632	5.11 (0.69–37.50)	0.108			
SNP rs17570669_T	1.427	4.16 (1.22–14.11)	0.022	1.322	3.75 (1.10 – 12.77)	0.034

AF: Atrial fibrillation, HR: Hazard ratio, SNP: Single nucleotide polymorphism.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-069

Cardioneuroablation is associated with improved health-related quality of life in patients with cardioinhibitory type vasovagal syncope

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Background and Aim: Cardioneuroablation(CNA) emerged as a safe and feasible alternative in treatment of vasovagal syncope (VVS). The aim of this study was to demonstrate if this novel treatment results in improvement in quality of life (QoL) of the patients.

Methods: Patients with documented cardioinhibitory type VVS in tilt table testing, who underwent CNA in our center, were enrolled in this study. ECGs were obtained prior to procedure, and at 6 month follow-up visit. QoL was assessed with the use of SF-36 and EQ VAS questionnaires.

Results: Twenty-seven patients (age: 34 ± 14 years, 48% female) were enrolled in this study. ECG data were available in 25 patients while QoL data were available in 27 patients. At 6-month follow-up, heart rate significantly increased (74 ± 15 bpm to 84 ± 14 bpm, p=0.003). QoL assessed by SF-36 score improved significantly in postprocedural follow-up (92 ± 9 and 96 ± 11, p=0.016). Similarly, significant improvements in mobility, self-care and usual activity domains of EQ-5D was observed (mean scores of 3.0 ± 1.5 and 2.1 ± 1.3, p<0.001; 1.3 ± 0.9 and 1.2 ± 0.6, p=0.041; 1.7±1.0 and 1.4±0.8 respectively). EQ-VAS score also improved significantly (39 ± 24 to 77 ± 18, p<0.001).

Conclusions: Our findings suggest that CNA might be associated with improvement in QoL in patients with VVS.

Table 1. Baseline characteristics, and change in ECG and quality of life measures at 6 month follow-up

	Valid Cases	Pre-CNA	Post-CNA	p
Demographics				
Female,n(%)	25	13(48)	-	-
Age,years	25	34±14	-	-
Higher education,n(%)				
Electrocardiography				
Heart rate,bpm	**25**	**74±15**	**84±14**	**0.003**
PR,ms	25	160±62	162±56	0.62
QT,ms	25	387±40	372±45	0.10
Quality of Life				
SF-36 Score	27	92 ± 9	96±11	0.06
PCS				
MCS				
EQ-VAS Score	**27**	**39±24**	**77±18**	**<0.001**
Mobility	**27**	**3±2**	**2±1**	**<0.001**
Self-Care	27	1±1	1±1	0.041
Usual Activity	**27**	**2±1**	**1±1**	**0.021**
Pain	27	2±1	2±1	0.11
Anxiety	27	2±2	2±1	0.17

CNA: cardioneuroablation

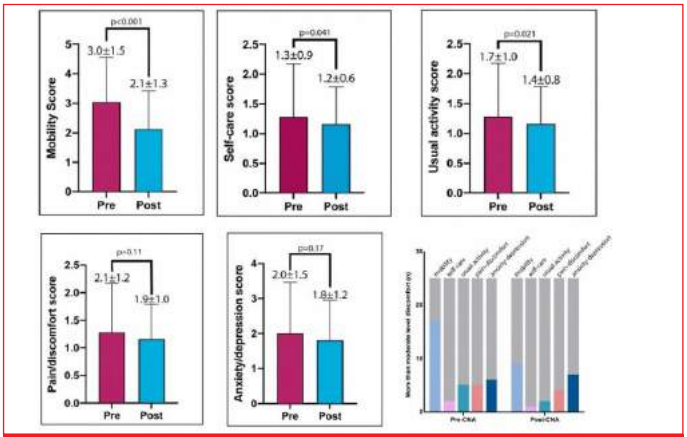

Figure 1. Change in EQ-5D survey domain scores at 6-months follow-up

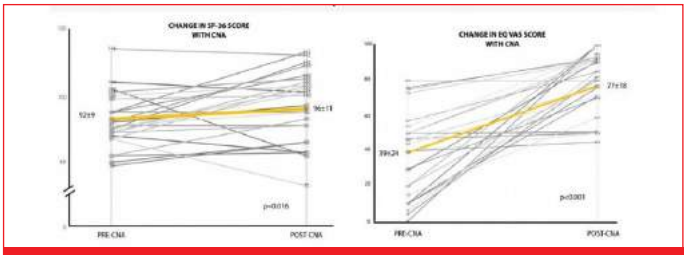

Figure 2. Change in Quality of Life assessed with SF-36 score and EQ-VAS score at 6 month follow-up compared with baseline

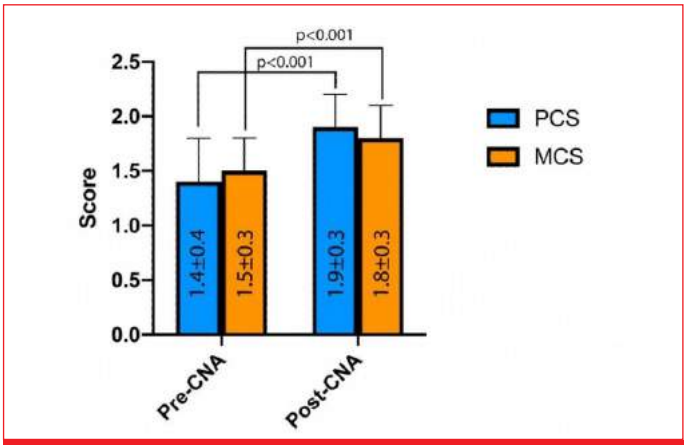

Figure 3. Changes in EQ-5D physical and mental component scores at 6-months follow-up

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-070

Demographic, clinical, and disease characteristics of patients with atrial fibrillation on edoxaban therapy according to kidney functions: A report from evaluation of treatment safety in patients with atrial fibrillation on edoxaban therapy in real-life

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Background and Aim: The efficacy of non-vitamin K antagonist (VKA) oral anticoagulants (NOACs) in preventing systemic embolism and stroke in patients with atrial fibrillation have been proven. Available data have indicated the reliability of NOACs. However, no prospective post-authorization safety and efficacy studies have been conducted in Turkey. The ETAF-TR, a real-life study investigating the safety and efficacy of edoxaban in atrial fibrillation patients in Turkey was designed for this purpose. In this study, we aimed to present the demographic, clinical and disease characteristics of patients according to their kidney functions.

Methods: The ETAF-TR (NCT04594915) is a national, multicenter, prospective, observational study that enrolled 1053 patients from 49 centers. Efficacy, treatment persistence, and posology were evaluated in an explorative manner. The baseline characteristics, demographic data and diseases of all patients were recorded. The overall duration of follow-up was one year and the first patient was enrolled in August 2020.

Results: A total of 1053 patients were enrolled in the study, of whom 621 (59%) were female, and 432 (41%) were male. The mean patient age was 70.1 years. The patients were grouped based on creatinine clearance as 15-30 (n=17), 30-50 (n=157) 50-80 (n=388) and > 80 (n=491). Among all patients, hypertension was detected in 155 (14.7%) patients. In the patients with hypertension, there were 21 (13.4%) patients in the CrCl 30-50 group, 57 (14.7%) patients in the CrCl 50-80 group, and 77 (15.7%) patients in the CrCl > 80 group. Diabetes was detected in 282 (26.8%) patients in the cohort. Among those with diabetes, 4 (23.5%) patients were in the CrCl 15-30 group, 42 patients (26.8%) were in the CrCl 30-50 group, 101 (26.0%) patients were in the CrCl 50-80 group and 135 (27.5%) patients were in the CrCl > 80 group. Heart failure was detected in 305 (29.0%) patients. Among these patients, there were 5 (29.4%) patients in the CrCl 15-30 group, 63 (40.1%) patients in the CrCl 30-50 group, 116 (29.9%) patients in the CrCl 50-80 group, and 121 (24.6%) patients in the CrCl > 80 group. A total of 140 (13.3%) patients were detected to have a history of cerebrovascular event. The classification of these patients according to the CrCl revealed 1 (5.9%) patients were in the CrCl 15-30 group, 21 patients (13.4%) were in the CrCl 30-50 group, 56 (14.4%) patients were in the CrCl 50-80 group and 62 (12.6%) patients were in the CrCl > 80 group. Vascular disease was detected in a total of 253 (24%) patients, of those, there were 3 (17.6%) patients in the CrCl 15-30 group, 51 (32.5%) patients in the CrCl 30-50 group, 96 (24.7%) patients in the CrCl 50-80 group and 103 (21.0%) patients in the CrCl > 80 group.

Conclusions: The data obtained in the ETAF-TR study will support the ENGAGE AF and ETNA-AF studies. The results of our study will enable the comparison of demographic and clinical data of Turkey with the results of these studies under the conditions of our country.

Other

OP-071

Characteristics of patients with atrial fibrillation on edoxaban treatment in Turkey: Baseline data from evaluation of treatment safety in patients with atrial fibrillation on edoxaban therapy in real-life in Turkey (ETAF-TR) study

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Background and Aim: Safety and effectiveness of edoxaban was demonstrated in a phase III trial and is being confirmed in the post-authorization Edoxaban Treatment in routiNe clinical prActice for patients with Atrial Fibrillation in Europe (ETNA-AF-Europe) study in patients with atrial fibrillation (AF). However, any post-authorization safety study focusing on the safety of edoxaban treatment in Turkey with prospective design has not been performed yet. The Evaluation of Treatment Safety in Patients with Atrial Fibrillation on Edoxaban Therapy in Real-Life in Turkey (ETAF-TR) study is designed to evaluate the safety and effectiveness of edoxaban treatment in AF in routine practice. This sub-study describes the baseline demographic, clinical, and laboratory characteristics of ETAF-TR Study.

Methods: The ETAF-TR study (NCT04594915) is a national, multicentre, prospective, observational study that included 1053 cases from 50 centres. The primary outcome of the study is any overt bleeding. Effectiveness, treatment persistence, and posology will also be evaluated in an explorative manner. Enrolment process had been completed in May 2022. The overall duration of follow-up will be 1 year.

Results: Mean age was 70.1 years and 59% were female. Mean CHA2DS2-VASc and HAS-BLED scores were 3.5 and 1.6, respectively. Baseline demographic, clinical, and laboratory characteristics of ETAF-TR study is summarized in Table 1.

Although, 82% of patients received an edoxaban dose in line with the summary of product characteristics (SmPC) (proper posology), 10% and 8% of patients had been taking improperly higher (60 mg, od, over treatment) and lower (30 mg, od, under treatment) doses of edoxaban respectively.

Conclusions: Edoxaban has been used wide spectrum of patients with AF in daily routine practice with a good overall adherence to the SmPC. As the biggest national pharmacovigilance study to date, ETAF-TR Study will provide detailed insight to safety of edoxaban treatment.

Table 1. Baseline demographic, clinical, and laboratory variables of ETAF-TR study population

Variable	All Patients (n=1053)	Edoxaban 30 mg (n=210, 19.9%)	Edoxaban 60 mg (n=843, 80.1%)
Sex (Female) (n,%)	621 (59.0%)	137 (65.2%)	484 (57.4%)
Age (years) (mean, SD)	70.1 (11.3)	77.4 (11.0)	68.2 (10.6)
Age≥75 years (n, %)	391 (37.1%)	151 (71.9%)	240 (28.5%)
Body Mass Index (kg/m²) (mean, SD)	29.1 (5.4)	27.8 (5.4)	29.4 (5.3)
Systolic Blood Pressure (mmHg) (mean, SD)	130.3 (17.5)	131.9 (17.3)	129.9 (17.6)
Diastolic Blood Pressure (mmHg) (mean, SD)	77.5 (11.0)	77.0 (11.1)	77.7 (11.0)
Heart Failure (n, %)	305 (29.0%)	72 (34.3%)	233 (27.6%)
Hypertension (n, %)	805 (76.4%)	166 (79.0%)	639 (75.8%)
Diabetes Mellitus (n, %)	282 (26.8%)	56 (26.7%)	226 (26.8%)
Stroke & TIA History (n, %)	139 (13.2%)	29 (13.8%)	110 (13.0%)
Vascular Disease (n, %)	253 (24.0%)	63 (30.0%)	190 (22.5%)
CHA_2DS_2VASc Score (Mean, SD)	3.5 (1.5)	4.2 (1.4)	3.3 (1.5)
HASBLED Score (Mean, SD)	1.6 (1.0)	2.0 (1.0)	1.4 (0.9)
AF Pattern (Paroxysmal) (n %)	311 (29.5%)	49 (23.3%)	262 (31.1%)
AF Pattern (Permanent) (n %)	601 (57.1%)	139 (66.2%)	462 (54.8%)
AF Pattern (Persistent) (n %)	82 (7.8%)	14 (6.7%)	68 (8.1%)
AF Pattern (Long Standing Persistent) (n %)	59 (5.6%)	8 (3.8%)	51 (6.0%)
History of AF Ablation (n, %)	21 (2.0%)	3 (1.4%)	18 (2.1%)
Implantable Devices (CIED) (n, %)	51 (4.8%)	15 (7.1%)	36 (4.3%)
Left Atrial Appendage Closure (n, %)	1 (0.1%)	–	1 (0.1%)
Coronary Artery Disease (n, %)	301 (28.8%)	64 (30.5%)	239 (28.4%)
Bleeding History (n, %)	90 (8.5%)	30 (14.3%)	60 (7.1%)
Bleeding Type (CRNM, n, %)	19 (1.8%)	7 (3.3%)	12 (1.4%)
Bleeding Type (Major, n, %)	18 (1.7%)	6 (2.9%)	12 (1.4%)
Bleeding Type (Overt, n, %)	9 (0.9%)	5 (2.4%)	4 (0.5%)
Bleeding Type (Minor, n, %)	44 (4.2%)	12 (5.7%)	32 (3.8%)
Thromboembolic Event History (n, %)	150 (14.2%)	30 (14.3%)	120 (14.2%)
Ischemic Stroke (n, %)	112 (10.6%)	19 (9.0%)	93 (11.0%)
Transient Ischemic Attack (n, %)	24 (2.3%)	6 (2.9%)	18 (2.1%)
Creatinine Clearance (ml/min) (Mean, SD)	80.5 (31.5)	55.2 (23.2)	86.8 (30.1)
Creatinine Clearance (ml/min) (Median, IQR)	77.8 (57.8, 98.7)	49.7 (38.8, 62.5)	83.7 (66.0, 103.2)
Haemoglobin (g/dl) (Mean, SD)	12.9 (1.9)	12.1 (1.9)	13.1 (1.8)
Antiplatelet Use	118 (11.2%)	29 (13.8%)	89 (10.6%)
Creatinine Clearance 15-50 ml/min (n, %)	174 (16.5%)	106 (50.5%)	68 (8.1%)
Off Label Usage (n, %)	38 (3.6%)	3 (1.4%)	35 (4.2%)
Overtreatment (n, %)	100 (10%)	–	100 (11.8%)
Undertreatment (n, %)	81 (8%)	81 (38.6%)	–
Proper Posology (n, %)	834 (82%)	129 (61.4%)	743 (88.2%)

Other

OP-072

Could zonulin and presepsin be biomarkers and therapeutic targets for acute myocarditis?

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Background and Aim: Acute myocarditis is an acute inflammation of the heart muscle with clinical presentations ranging from mild clinical to dilated cardiomyopathy, often seen in young adult and childhood. The diagnosis of myocarditis is usually made with clinical and laboratory parameters. This can sometimes be confused with diseases presenting with similar clinical features, making the diagnosis difficult. Therefore, the use of more specific biomarkers in addition to the classically used biomarkers such as troponin will accelerate the diagnosis. In addition, these biomarkers may help us to understand the mechanism of myocarditis development and thus predict unpredictable clinical outcomes. The aim of this study is to reveal the possible relationship between intestinal permeability and acute myocarditis.

Methods: In this study we wanted to evaluate serum levels of serum zonulin and presepsin in 69 consecutive subjects, including 34 patients with myocarditis and and another 35 were the control group, matched for age, gender, and cardiovascular risk factors.

Results: Zonulin and presepsin levels were statistically higher in the patient group than in the control group (Figure 1). We divided the patients into 3 groups according to their zonulin tertiles. Those who described gastrointestinal complaints within the last 4 weeks of admission were significantly more common in tertile 3 than in tertile 1 and 2. In addition, Peak CK-MB, peak Troponin-I, presepsin values were significantly higher in tertile 3 compared to other tertiles.In the patient group, zonulin levels were positively correlated with presepsin, peak CK-MB, and peak troponin levels (Figure 2) and zonulin and presepsin were found to be independent predictors of acute myocarditis in logistic regression analysis. When ROC Curve analysis was performed, the optimal cut-off value of presepsin to predict acute myocarditis is presepsin ≥584.13; was predictive for acute myocarditis with 79.4 % sensitivity and 80% specificity (Figure 3).

Conclusions: Myocarditis may present similarly to ischemic heart disease with chest pain, abnormalities in electrocardiograms, and elevated cardiac biomarkers, and may be confused with these cardiac pathologies that are similar in symptoms and laboratory findings, including cardiac amyloidosis and hypertrophic cardiomyopathy. Therefore, suspected cases of myocarditis remain a challenging diagnosis for clinicians in terms of its presentation, features, and course. In this respect, when gold standard diagnostic tools such as EMB and cMRI are not available, auxiliary biomarkers that can be used in addition to CK-MB and cardiac troponin for rapid diagnosis of myocarditis cases and exclusion of other confounding cardiac causes. In addition, increased respiratory tract and intestinal permeability may be one of the main mechanisms triggering the development of myocarditis. Therefore, zonulin and presepsin may be promising biomarkers for both diagnosis and follow-up treatment in myocarditis patients.

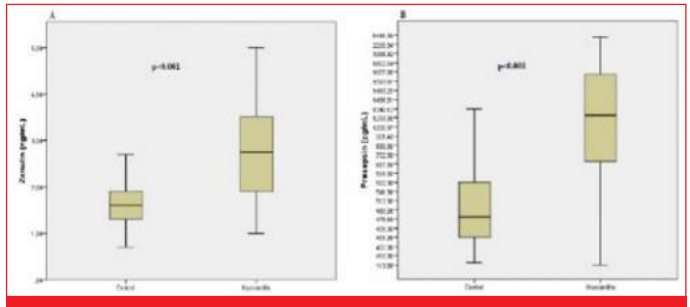

Figure 1. Zonulin (A) and presepsin (B) levels in patients with myocarditis compared to the control group

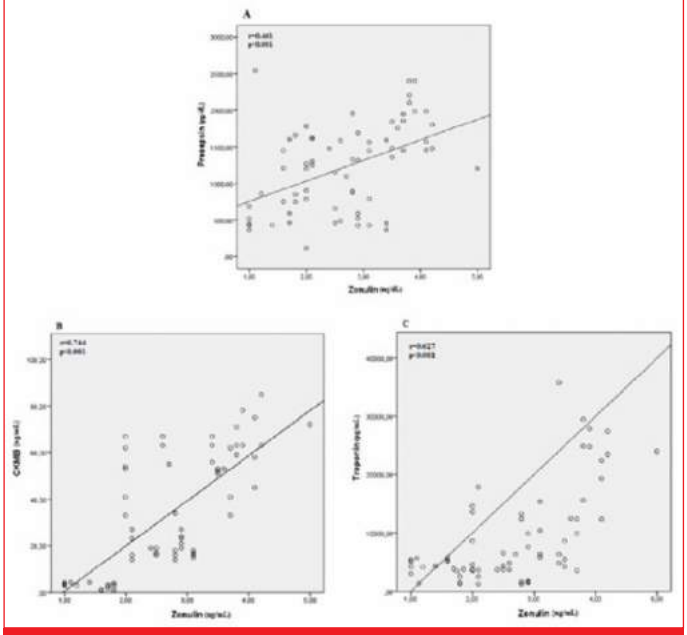

Figure 2. Positive correlation between Zonulin and Presepsin (A), Zonulin and CK-MB (B), Zonulin and Troponin (C) in patients with myocarditis

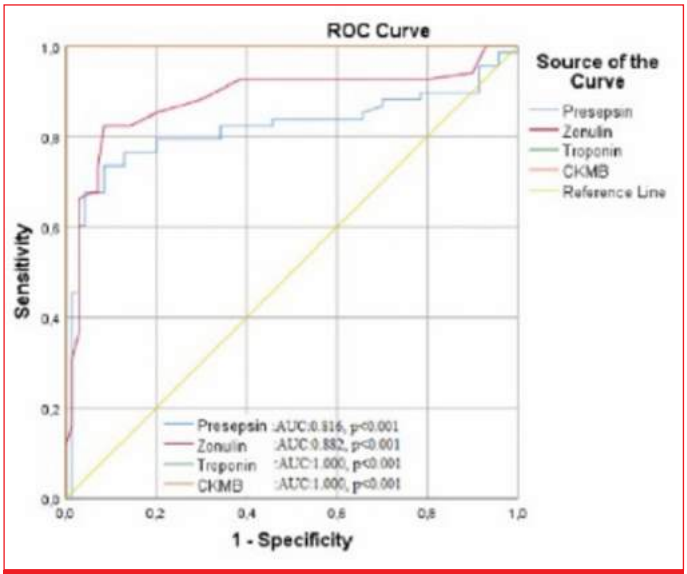

Figure 3. Receiver operating characteristics (ROC) curve of presepsin, zonulin, troponin and CKMB for predicting the acute myocarditis.

Table 1. Independent predictors of acute myocarditis

	Odds ratio	95% CI	P value
Zonulin	12.331	4.261-35.689	<0.001
Presepsin	1.001	1.000-1.002	0.025

Entered variables: C- reactive protein, History of COVID-19 or COVID-19 vaccination in the last 6 months, Fibrinogen, GIS complaints, Rhythm disturbances, Left ventricular ejection fraction, Presepsin, Zonulin

Other

OP-073

Admission troponin levels were associated with disease severity and recent cardiac injury shown by MRI in Covid-19 inpatients

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Background and Aim: Troponin and some inflammation biomarkers were associated with Covid-19 severity and intensive care unit (ICU) admissions related with cardiac injury. But cardiac magnetic resonance imaging (CMRI) remains to be the gold standard for revealing myocardial involvement. In this study, we aim to investigate whether admission troponin levels were associated with both clinical severity and cardiac injury shown using CMRI.

Methods: The study sample consisted of 51 recovered patients who had needed in-hospital follow-up, either in COVID-19 ICU (Group 1, n=16) and COVID-19 clinics (Group 2, n=35). Hs-cTnT, CRP, PCT, NLR, D-dimer, ferritin levels and SII and were measured at admission to hospital. All of these participants were referred to electrocardiography (ECG), transthoracic echocardiography (TTE) and CMRI simultaneously on an average of 4-6 weeks after discharge. Study groups were compared according to these findings.

Results: Among study population, 25 patients (49%) had SARS-CoV-2 variants, including the Alpha variant [n=16, (31%)], the Beta [n=5, (10%)] and the Delta variant [n=4, (8%)]. Respiratory distress as initial symptom was higher in group 1 compared to group 2. Group 1 had higher respiratory rate, lower SpO2 levels and higher supplemental oxygen requirement compared with group 2, which would explain respiratory distress. Hs-cTnT levels and inflammatory biomarkers were significantly higher in ICU patients (p<0.05). ROC curve revealed significant correlation between Hs-cTnT, NLR, D-dimer, ferritin, CRP, SII levels and ICU admission (p<0.05) (Figure 1). ECG and TTE features of groups were similar. Functional parameters were also similar for both groups in CMRI findings. But, a total of 32 patients had any kind of injury on CMRI, including at least one of the following: myocardial oedema (n=7), pericardial effusion (n=13) and right ventricular failure (n=12), ischemic (n=8) or non-ischemic fibrosis (n=27) on late gadolinium enhancement imaging. CMRI images of various myocardial injury patterns were shown in Figure 2. There was a significant difference between patients who followed up in Group 1 vs. Group 2 for non-ischemic fibrosis [n=12 (75%) vs. n=15 (43%); p=0.03]. ICU patients had more common non-ischemic fibrosis on CMRI (p=0.03). ROC curve exposed a significant correlation between Hs-cTnT and SII levels with any injury shown on CMRI (p<0.05) (Figure 3).

Conclusions: Admission troponin and SII levels were associated with disease severity and cardiac injury shown by CMRI

even if echocardiographic evaluation is normal. Both of them could be used to have an idea about both the need of ICU and serious cardiac involvement.

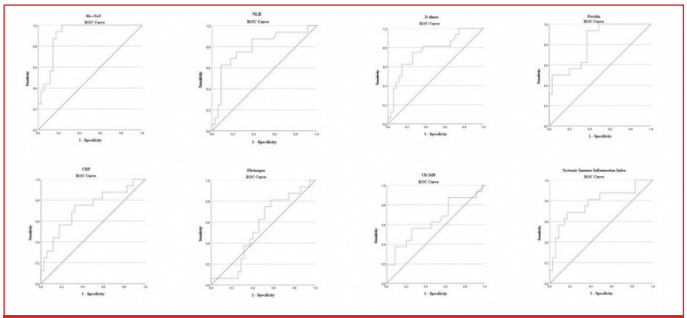

Figure 1. The analysis of the ROC curve for correlation between biomarkers and ICU admission

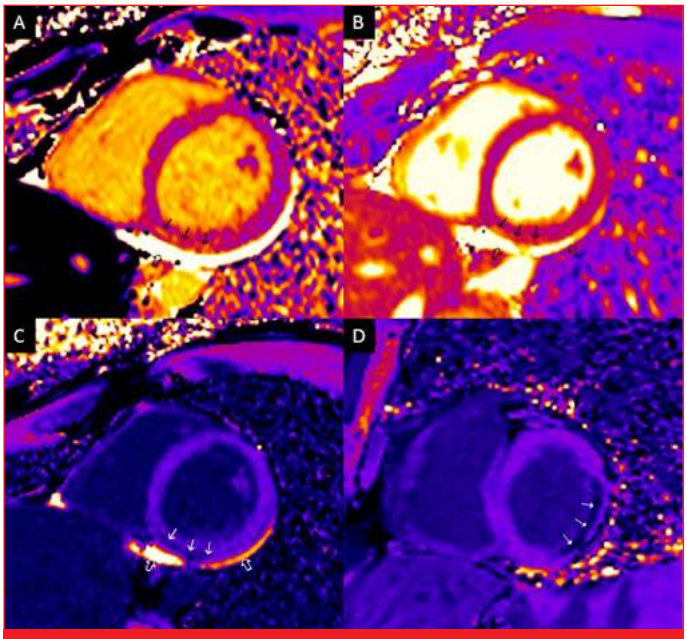

Figure 2. Native T1 (A), T2 (B), and post-contrast T1 (C) map images in short axis plane shows signal changes consistent with acute myocarditis (arrows) and mild pericardial effusion (open arrows). In another patient, subendocardial T1 shortening in post-contrast T1 map (D) indicates left circumflex territory infarction (striped arrows).

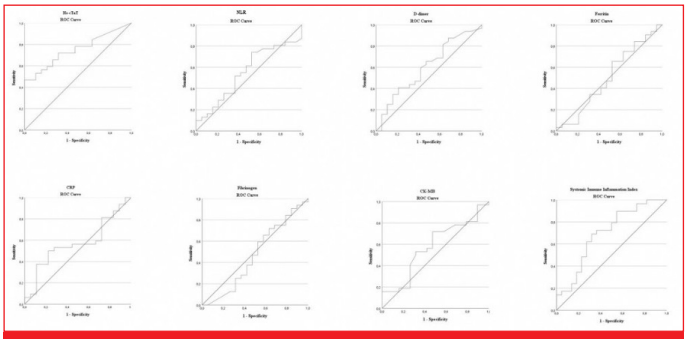

Figure 3. The analysis of the ROC curve for correlation between biomarkers and CMRI.

Other

OP-074

Perspectives on the use of digital health technologies among Turkish cardiologists: Results from a survey

Duygu Koçyiğit[1], Nurgül Keser[2], Evrim Şimşek[3], Serdar Bozyel[4], Yalçın Dalgıç[4], Mehmet Şeker[2], Yetkin Korkmaz[2], Arda Güler[5], Tufan Çınar[2], Mehmet Ertürk[5]

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Background and Aim: Digital health technologies (DHTs) have the potential of facilitating both physicians' and patients' lives with regards to the diagnosis, treatment and follow-up of cardiovascular disease. A huge acceleration in relevant research has been noted particularly after the COVID-19 pandemic. The goal of this study was to determine the reasons for using DHTs in cardiology, as well as the perceived barriers to its use, among Turkish cardiologists.

Methods: An electronic survey with 43 question multiple-choice questionnaire was conducted between January 10-March 3, 2022. Turkish Society of Cardiology member cardiology fellows in training and specialists were contacted via e-mail (n=2789).

Results: 308/2789 (11.04%) subjects responded to the survey (72.40% males, 62.01% aged 30-44 years). 42.53% and 44.81% were affiliated with university hospitals and state hospitals, respectively. 88/297 (29.63%) stated having at least good understanding of DHTs in cardiology. 44.16% utilized smart devices to track their own health status. 117/290 (40.34%) and 193/299 (64.55%) used social media platforms to share medical information with their patients and other physicians, respectively. WhatsApp and Instagram were the most popular platforms for sharing with patients (92/117, 78.63% and 48/117, 41.03%), while WhatsApp and Twitter were the most popular platforms for sharing with other physicians (151/193, 78.24% and 91/193, 47.15%). Considerations and recommendation/utilization patterns of DHTs by physicians are summarized in Table 1. Perceived barriers to the use of DHTs in cardiology is shown on Figure 1 (A: physician-related, B: patient-related, C: technical).

Conclusions: Findings suggest that nearly half of the physicians use DHTs to collect their own health data and use social media to disseminate health information. The majority of physicians believe that DHT is beneficial to both themselves and their patients, and that DHT use in cardiology has increased as a result of the COVID-19 pandemic. To overcome the challenges to the use of DHTs in cardiol-

ogy, a multilayered collaborative effort involving patient and professional organizations, as well as technical stakeholders and lawmakers, is required.

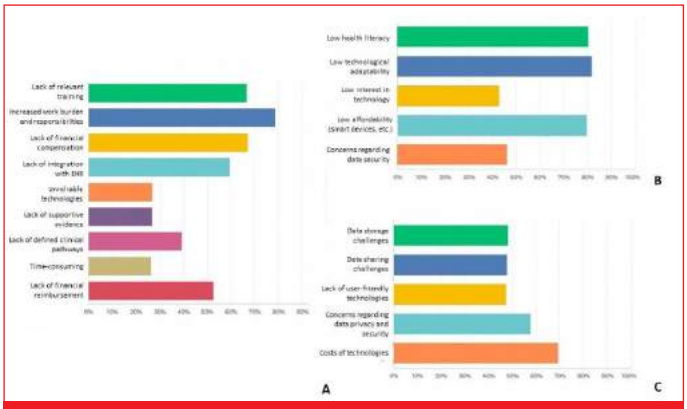
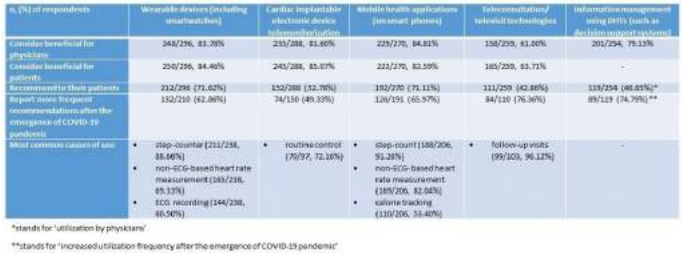


Figure 1.

Table 1.



*stands for 'utilization by physicians'

**stands for 'increased utilization frequency after the emergence of COVID-19 pandemic'

Other

OP-075

A new marker for doxorubicin cardiotoxicity: Paraoxonase-1; ecocardiographic, biochemical and histological study

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Background and Aim: Paraoxonase-1 (PON-1) is an antioxidant,cardioproctevite enzyme that associates with high-density lipoprotein and helps to prevent the formation of oxidized low density lipoprotein. There are a little evidence that show association between PON-1 levels and doxorubicin (DOX) cardiotoxicity, but there's not any basic science study about this issue. The aim of this study to relive correlation between PON-1 levels and histological changes in DOX cardiotoxicity.

Methods: Eight teen male Sprague Dawley rats (350–500 g) were randomized into two groups. The first (control) group (n=9) received serum physiologic (1 ml) via orogastric gavage (OG) and intraperitoneally (i.p.) for 14 days. The second (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. On the 15th day, the rats were anesthetized and cardiac functions were evaluated by echocardiography. Then blood samples were taken from directly heart to evaluate biochemical parameteres and heart tissues were excised to evaluate histopathological findings. PON-1 activity was measured using commercially available kits. The rate of paraoxon hydrolysis was measured by monitoring the increase of absorption at 412 nm at 37 °C. The amount of generated p-nitrophenol was calculated from the molar absorption coefficient at pH 8.5, which was 18.290 M–1 cm–1.

Results: In DOX group; left ventricle end diastolic and end systolic diameters were enlarged; therefore ejection fraction and fractional shortening were lowered than the control group (p<0.001). Histological analyses showed increased karyolisys, karyorheksis decreased normal cell morphology in DOX group. Biochemical analysis relieved decreased PON-1 levels in DOX group than control group (p<0.01) (Table 1). Moreover PON-1 levels were correlated with histological finding (Pearson r: -0,6; p<0.01) (Table 2).

Conclusions: Our results showed that decreased PON-1 levels is associated with DOX cardiotoxicity, morover PON-1 levels can reflect DOX cardiotoxicity in histological levels. To our knowledge; this is the first study presenting that PON-1 level is as valuable as histological data in DOX cardiotoxicity. On the basis of this study; with PON-1 level, DOX cardiotoxicity can be diagnosed at an early stage without the need for biopsy and prevention can be taken.

Table 1. Echocardiographic, histologic, biochemical findings of groups

	Control (n=9)	DOX (n=9)	P
LVEDD mm, Median (IQR)	53 (12)	71 (9)	0.007
LVESD mm, Median (IQR)	29 (6.5)	53 (9.5)	<0.001
EF (%) (mean ± SD)	74.7 ± 5.3	35.2 ± 10.5	<0.001
FS (mean ± SD)	50.5 ± 6.5	13.7 ± 6.2	<0.001
Normal myocytes ratio (number/100 myocyte)	0.95 ± 0.04	0.45 ± 0.02	<0.001
Karyolysis ratio (number/100 myocyte)	0.015 ± 0.012	0.255 ± 0.028	<0.001

Table 1. Echocardiographic, histologic, biochemical findings of groups (Continued)

	Control (n=9)	DOX (n=9)	P
Karyorrhexis ratio (number/100 myocyte)	0.030 ± 0.027	0.293 ± 0.025	<0.001
Infiltrative cell quantity (number/per field)	2.6 (2.2)	11.8 (1.4)	<0.001
PON-1 level U/L serum (mean ± SD)	1064.8 ± 106.3	772.4 ± 239.3	0.004

EF: Ejection Fraction, FS: Fractional Shortening, LVEDD: Left ventricle end diastolic diameter, LVESD: Left ventricle end systolic diameter, IQR: Interquartile range, PON-1: Paraoxonase-1

Table 2. PON-1 correlation with histological markers

	Pearson r	P
Infiltrative cell quantity	-0.606	0.008
Karyolysis ratio	-0.632	0.005
Karyorrhexis ratio	-0.657	0.003

Interventional cardiology / Valve and structural heart diseases

OP-076

Left atrial appendix closure experience of a tertiary centre: procedure success, complications and follow-up results

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Department of Cardiology, İzmir Health Sciences University Tepecik Training and Research Hospital, İzmir

Background and Aim: Oral anticoagulants (OAC) are the preferred treatment options of patients with atrial fibrillation in order to diminish the risk of thromboembolic events. However, patients who are unable to use an OAC for a long time (because of a contraindication) and patients who experienced major bleeding or recurrent thromboembolic events despite of taking an OAC are good candidates of percutaneous LAA (left atrial appendix) closure. Here we present our centre's experience of such cases undergone this procedure in three-years time period.

Methods: We retrospectively analyzed 39 percutaneous LAA closure procedure using Amulet™ occluder device in Cardiac Catheter Laboratory of Tepecik Training and Research Hospital. Indications were major gastrointestinal bleeding (n=15), intracranial hemorrhage (n=14) and ischemic cerebrovascular event (n=10) under OAC therapy. We analyzed procedure success/complication rates and also major cerebrovascular events in follow-up period.

Results: Mean age of study population was 75.75 (±8.76) and 58.9% (n=23) of patients were female. Mean CHA2DS2-VASc and HAS-BLED scores of patients were 4.79 (±1.63) and 3.03 (±1.23) respectively. Success rate of procedures was 100% and only in one case we observed thrombus on device after 1 month which resolved with intravenous heparin therapy. Six patients died during follow up; but only one of them (2.5%) died because of a major cerebrovascular event (intracranial hemorrhage). We did not observe any ischemic cerebrovascular event even in patients who have not continued OAC therapy (n=8) in three years follow-up.

Conclusions: Percutaneous LAA closure is a viable option in carefully selected patients with a high success rate and acceptable complication rates.

Interventional cardiology / Valve and structural heart diseases

OP-077

Is transcatheter aortic valve ımplantation in patients with low to moderate risk as safe as in high risk patients?

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Background and Aim: Transcatheter aortic valve implantation (TAVI) and surgical aortic valve replacement are treatments that can be chosen interchangeably in high risk patients with severe aortic stenosis (AS). However, there is still a lack of data about low risk patients. We investigated the impact of patient's risk status on mortality in TAVI procedure.

Methods: A total of 289 consecutive symptomatic severe AS patients who underwent TAVI were retrospectively evaluated. Baseline clinical, demographic and procedural variables were recorded. Then, patients were divided into two groups according to their STS-prom score; patients with low to moderate STS-prom score (≤8) as group 1 (96 patients) and patients with STS-prom high score (>8) as group 2 (193 patients).

Results: There was no significant difference in terms of postoperative aortic regurgitation, coronary occlusion, stroke, minor and major vascular complications, bleeding, tamponade and cardiac pacemaker implantation between groups. The incidence of acute kidney injury was higher in high risk patients [10 (10.4%), 42 (21.8%); p=0.018]. In hospital mortality [9 (9.4%), 26 (13.5%); p=0.315] and 1-year mortality rates [15 (15.6%), 37 (19.2%); p=0.460] were similar in both groups. Long term survival rates were not different in both groups in Kaplan-Meier survival analysis (log rank, p=0.215). In multivariate logistic regression analysis, permanent pacemaker implantation (OR: 2.583, 95% CI: 1.083-6.160; p=0.032) and atrial fibrillation (OR: 2.069, 95% CI: 1.106-3.873; p=0.023) were independent predictors of mortality.

Conclusions: TAVI is safe and effective treatment modality in symptomatic severe AS patients with low to moderate risk.

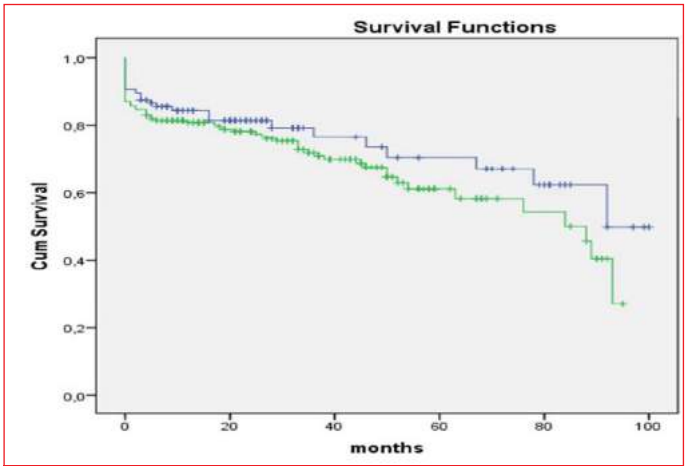

Figure 1. Kaplan-Meier survival. Long term survival rates were not different in both groups in Kaplan-Meier survival analysis (Log Rank, p=0.215)

Table 1. Baseline clinical and demographic variables of the whole study group

	All patients (n=289)	Low-moderate risk (n=96)	High risk (n=193)	P
Age	78.8 ± 7.6	80 ± 7	78 ± 8	0.094
Gender (female), n (%)	178 (61.6)	65 (67.7)	113 (58.5)	0.132
Coronary artery disease, n (%)	181 (62.6)	59 (61.5)	122 (63.2)	0.772
Smoking, n (%)	75 (26.0)	27 (28.1)	48 (24.9)	0.552
Body mass index (kg/m^2)	27 (24-29.5)	27.8 (24.3-30.2)	26.7 (24.0-29.0)	0.280
COPD, n (%)	172 (59.5)	58 (60.4)	114 (59.1)	0.826
Diabetes mellitus, n (%)	116 (40.1)	37 (38.5)	79 (40.9)	0.696
Hypertension, n (%)	208 (72.0)	65 (67.7)	143 (74.1)	0.255
Dyslipidemia, n (%)	85 (29.4)	32 (33.3)	53 (27.5)	0.302
CABG, n (%)	64 (22.1)	20 (20.8)	44 (22.8)	0.705
Peripheral arterial disease, n (%)	96 (33.2)	30 (31.3)	66 (34.2)	0.616
Previous cerebrovascular disease, n (%)	8 (2.8)	0 (0)	8 (4.1)	0.038
Atrial fibrillation, n (%)	55 (19.0)	16 (16.7)	39 (20.2)	0.470
Creatinine (mg/dl)	1.10 (0.90-1.40)	1.0 (0.8-1.29)	1.1 (0.95-1.40)	<0.001
Hemoglobine (g/dl)	11.4 (10.1-12.6)	11.2 (10.0-12.45)	11.4 (10.3-12.6)	0.199
Leukocytes × 10^3/mm^3	7.8 (6.64-9.20)	7.75 (6.45-9.26)	7.82 (6.77-9.2)	0.790
Thrombocyte × 10^3/mm^3	229 (197-279)	230 (196-258)	229 (197-290)	0.345

Table 1. Baseline clinical and demographic variables of the whole study group (Continued)

	All patients (n=289)	Low-moderate risk (n=96)	High risk (n=193)	P
Vavle type, n (%) Balloon exp Self exp Mech exp	206 (71.3) 57 (19.7) 26 (9.0)	68 (70.8) 26 (27.1) 2 (2.1)	138 (71.5) 31 (16.1)a 24 (12.4)b	0.003
Valve size, n (%) 23 mm 25 mm 26 mm 27 mm 29 mm 34 mm	72 (24.9) 19 (6.6) 91 (31.5) 24 (8.3) 82 (28.4) 1 (0.3)	23 (24.0) 3 (3.1) 30 (31.3) 9 (9.4) 30 (31.3) 1 (1.0)	49 (24.5) 16 (8.3) 61 (31.6) 15 (7.8) 52 (26.9) 0 (0)	0.479
Ejection fraction (%)	60 (46.5-60)	60 (48-60)	60 (48-60)	0.586
Aortic valve area (cm^2)	0.71 ± 0.15	0.70 ± 0.14	0.72 ± 0.16	0.244
Maximum gradient (mm Hg)	81.3 ± 17.2	81.4 ± 16.2	81.3 ± 17.7	0.974
Mean gradient (mm Hg)	50.8 ± 11.07	49.9 ± 10.3	51.3 ± 11.4	0.340
Postoperative discharging time (days)	7.3 ± 4.8	8 ± 5	7 ± 4	0.602
STS PROM score	9.83 ± 4.60	5.13 ± 1.42	12.18 ± 3.78	<0.001

a= lower than low-moderate group, b= higher than low-moderate group
CABG: coronary artery bypass grafting, COPD: chronic obstructive pulmonary disease, STS-PROM: Society of Thoracic Surgeons-predicted risk of mortality

Table 2. Procedural and follow-up characteristics of patients

	All patients (n=289)	Low-moderate risk (n=96)	High risk (n=193)	P
Postoperative AR, n (%) None Mild Moderate	225 (77.9) 54 (18.7) 10 (3.5)	68 (70.8) 24 (25.0) 4 (4.2)	157 (81.3) 30 (15.5) 6 (3.1)	0.123
Coronary occlusion, n (%)	2 (0.7)	1 (1.0)	1 (0.5)	0.555
Stroke, n (%)	5 (1.7)	1 (1.0)	4 (2.1)	0.462
Acute kidney injury, n (%)[a]	52 (18.0)	10 (10.4)	42 (21.8)	0.018
Major vascular complication, n (%)	18 (6.2)	9 (9.4)	9 (4.7)	0.119
Minor vascular complication, n (%)	28 (9.7)	9 (9.4)	19 (9.8)	0.899
Bleeding, n (%)	60 (20.8)	23 (24.0)	37 (19.2)	0.345
Tamponade, n (%)	7 (2.4)	2 (2.1)	5 (2.6)	0.573
Cardiac pacemaker implantation, n (%)	44 (15.2)	12 (12.5)	32 (16.6)	0.363
In-hospital 30-day mortality, n (%)	35 (12.1)	9 (9.4)	26 (13.5)	0.315
In-hospital mortality time (days)	26.9 ± 8.5	27 ± 8	27 ± 9	0.507
1-year mortality, n (%)	52 (18.0)	15 (15.6)	37 (19.2)	0.460

AR: aortic regurgitation

Table 3. Multivariate logistic regression analysis about independent predictors of mortality

	Multivariate analysis		
	Odds ratio	95% CI (Lower-Upper)	P
Permanent pacemaker imp	2.583	1.083-6.160	0.032
Atrial fibrillation	2.069	1.106-3.873	0.023
STS PROM	1.091	1.029-1.156	0.003
Creatinine	1.364	0.941-1.976	0.101

Interventional cardiology / Valve and structural heart diseases

OP-078

Comparison of left ventricular guidewire pacing versus right ventricular rapid pacing among patients undergoing TAVI with Portico valve

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Background and Aim: Since the first Transcatheter Aortic Valve Implantation (TAVI) on a person in 2002, operations have become progressively minimalist. Because balloons are used in self-expandable valves, rapid ventricular pacing is required. The purpose of this study was to evaluate conventional RV rapid pacing versus guidewire-mediated left ventricular (LV) rapid pacing in patients who had balloon valvuloplasty from TAVI procedures using a self-expandable Portico valve (Abbott).

Methods: A total of 147 patients with symptomatic severe aortic stenosis who underwent TAVI with a Portico valve were included in the study, retrospectively. In the conventional group, balloon-tipped temporary pacing catheters (5-F Pacel, Abbott) were inserted in RV after 6 Fr sheaths were put by femoral access. In the LV pacing group, 0.035-inch Innowi (Symedrix GMBH) was utilized in conjunction with the transcatheter valve delivery system. Using a clip, one of the electrodes was linked to the wire at one end and the pacing generator at the other. The other electrode was attached to a short wire that was either inserted in a sheath or applied to the skin with a local anesthetic. The opposite end of the electrode was linked to the pacing generator once more. At a rate of 160-200 bpm, rapid ventricular pacing was used. Aortic balloon valvuloplasty (pre- and post-dilatation) was performed once the systolic blood pressure reached <50 mm Hg. During valve implantation, no pacing was used.

Results: The RV pacing group (n=80) had a mean age of 77.6 ± 7.0, whereas the LV pacing group (n=67) had a mean age of 76.2±8.2 (p=0.280). Except for the STS score (6.39 ± 3.17 vs. 5.20 ± 1.75, p=0.007), which was lower in the LV pacing group, other demographic and laboratory characteristics were comparable in both groups. The aortic valve (AV) area was lower (0.66 ± 0.16 vs. 0.58 ± 0.15, p=0.002), mean AV gradient (47 ± 11 vs. 52 ± 13, p=0.005), and max AV gradient (71 ± 17 vs. 78 ± 20, p=0.04) were greater in the LV pacing group compared to the RV pacing group. The total procedure time (66.2 ± 15.9 vs. 58.2 ± 10.1, p<0.001), total fluoroscopy time (30.1 ± 9.0 vs. 27.7 ± 5.3, p=0.045), and contrast volume (275 ± 69 vs. 246 ± 50, p=0.004) were all longer in the RV pacing group. Both groups experienced similar procedural complications. The LV pacing group had fewer peripheral complications. 30-day outcomes such as all-cause mortality, cardiovascular mortality, bleeding, stroke, acute kidney injury, and the requirement for a permanent pacemaker were likewise comparable.

Conclusions: The following are the key findings of the current study; (i) There was no significant difference in 30-day clinical outcomes between the two pacing groups, (ii) Procedural times and the opaque amount may be less in the LV pacing group because it does not require additional venous vascular or right ventricular access, is quickly applicable, and is simple to use, (iii) As a consequence, LV pacing with the Portico valve appears to be as reliable, safe, simple, and effective as traditional RV pacing.

Table 1. Demographic and baseline data

	RV Pacing Group (n=80)	LV Pacing Group (n=67)	P-value*
Age (years)	77.6 ± 7.0	76.2 ± 8.2	0.280
Female gender, n (%)	52 (65.0)	45 (67.0)	0.783
Hyperlipidemia, n (%)	19 (23.8)	14 (20.9)	0.680
Coronary artery disease, n (%)	33 (41.3)	28 (41.8)	0.947
Previous CABG, n (%)	17 (21.3)	14 (20.9)	0.958
Previous atrial fibrillation, n (%)	15(18.8)	12 (17.9)	0.896
NYHA Class III-IV, n (%)	22 (27.5)	16 (23.9)	0.618
Diabetes mellitus, n (%)	28 (35.0)	19 (28.4)	0.390
Hypertension, n (%)	46 (57.5)	38 (56.7)	0.924
Prior stroke, n (%)	8 (10)	3 (4.5)	0.205
Peripheral artery disease, n (%)	10 (12.5)	12 (17.9)	0.360
Chronic kidney disease, n (%)	15 (18.8)	6 (9.0)	0.091
STS score (%)	6.39 ± 3.17	5.20 ± 1.75	0.007
< 4, n (%)	17 (21.3)	17 (25.4)	0.555
4 –7,9, n (%)	44 (55.0)	42 (62.7)	0.346
> 8, n (%)	19 (23.8)	8 (11.9)	0.066
Left ventricular systolic dysfunction (LV EF < 50%), n (%)	20 (25.0)	16 (23.9)	0.875
Left ventricular ejection fraction (%)	51.6 ± 11.4	54.5 ± 9.0	0.890
Mean aortic valve gradient (mm Hg)	47 ± 11	52 ± 13	0.005
Maximum aortic valve gradient (mm Hg)	71 ± 17	78 ± 20	0.040
Aortic valve maximum velocity (m/sn)	4.3 ± 0.5	4.4 ± 0.5	0.288
Aortic valve area (cm^2)	0.66 ± 0.16	0.58 ± 0.15	0.002
Bicuspid aortic valve morphology, n (%)	14 (17.5)	11 (16.4)	0.862
Mitral insufficiency (moderate/severe), n (%)	18 (22.5)	14 (20.9)	0.814

Table 1. Demographic and baseline data (Continued)

	RV Pacing Group (n=80)	LV Pacing Group (n=67)	P-value*
Aortic insufficiency (moderate/severe), n (%)	15 (18.8)	10 (14.9)	0.539
Creatinine, mg/dL	0.97 ± 0.43	0.91 ± 0.25	0.540
e-GFR**, ml/min/1.73 m^2	69 ± 23	70 ± 19	0.688
LDL-C, mg/dL	116 ± 29	120 ± 37	0.477
Hemoglobin, mg/dL	11.5 ± 1.8	11.6 ± 1.6	0.967

Data are presented as number (percentage) or mean ± standard deviation. *P-value < 0.05 was considered significant. **calculated according to the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.
CABG: coronary artery bypass surgery, e-GFR: estimated glomerular filtration rate, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, NYHA: New York Heart Association, STS: Society of Thoracic Surgeons

Table 2. Procedural data

	RV Pacing Group (n=80)	LV Pacing Group (n=67)	P-value*
Prosthesis implant success, n (%)	80 (100)	67 (100)	1.000
Valve size, n (%)			
23 mm	1 (1.3)	2 (3.0)	
25 mm	9 (11.3)	14 (20.9)	
27 mm	26 (32.5)	16 (23.9)	
29 mm	44 (55.0)	35 (52.2)	
Pre-dilatation n (%)	75 (93.9)	75 (88.1)	0.226
Post-dilatation, n (%)	38 (47.5)	36 (53.7)	0.452
Total procedure time (min)	66.2 ± 15.9	58.2 ± 10.1	< 0.001
Total fluoroscopy time (min)	30.1 ± 9.0	27.7 ± 5.3	0.045
Contrast volume (ml)	275 ± 69	246 ± 50	0.004
Acute device success**, n (%)	76 (95.0)	63 (94.0)	0.796
Procedural mortality, n (%)	0 (0)	0 (0)	NA
Conversion to open heart surgery, n (%)	0 (0)	0 (0)	NA
Conversion to AVR, n (%)	0 (0)	0 (0)	NA
Need for second Portico valve***, n (%)	3 (3.8)	3 (4.5)	0.824
Coronary obstruction, n (%)	1 (1.3)	0 (0)	0.360
Major vascular complications, n (%)	8 (10.0)	5 (7.5)	0.589
A-V Fistula, n (%)	1 (1.3)	0 (0)	0.358
Pseudoaneurysm, n (%)	3 (3.8)	3 (4.5)	0.824
>2 U ES replacement, n (%)	9 (11.5)	4 (6.0)	0.242
Graft stent, n (%)	4 (5.0)	4 (6.0)	0.796
Mild-Severe inguinal hematoma, n (%)	8 (10.0)	2 (3.0)	0.093
Iliofemoral dissection, n (%)	2 (2.5)	2 (3.0)	0.857

Data are presented as number (percentage) or mean ± standard deviation. AVR: aortic valve replacement, ES: erythrocyte suspension, NA: non-available.
*P-value < 0,05 was considered significant. ** Acute device success is achieved if the subject met all four Valve Academic Research Consortium criteria (successful vascular access, delivery and deployment of the device, and successful retrieval of the delivery system; only 1 valve implanted in the proper anatomic location; the correct position of the device in the proper anatomic location; and intended performance of the prosthetic heart valve (defined as aortic valve area >1.2 cm^2, mean aortic gradient <20 mm Hg, or peak velocity <4 m/s, without moderate or severe prosthetic valve aortic regurgitation). *** On the LV pacing group 29 mm Valve 3/3 vs RV pacing group 25 mm 1/3 and 29-mm 2/3

Table 3. 30-day outcomes

	RV Pacing Group (n=80)	LV Pacing Group (n=67)	P-value*
All-cause mortality, n (%)	6 (7.5)	2 (3.0)	0.229
Cardiovascular mortality, n (%)	3 (3.8)	1 (1.5)	0.402
>72 h Myocardial infarction, n (%)	0 (0)	0 (0)	NA
Stroke, n (%)	4 (5.0)	2 (3.0)	0.539
Acute kidney injury, n (%)	13 (16.3)	8 (11.9)	0.457
Life-threatening bleeding, n (%)	2 (2.5)	1 (1.5)	0.667
New pacemaker implantation**, n (%)	8 (12.9)	11 (16.4)	0.574

Data are presented as number (percentage) or mean ± standard deviation. *P-value < 0.05 was considered significant. ** Only individuals who had recently had a pacemaker placed were included

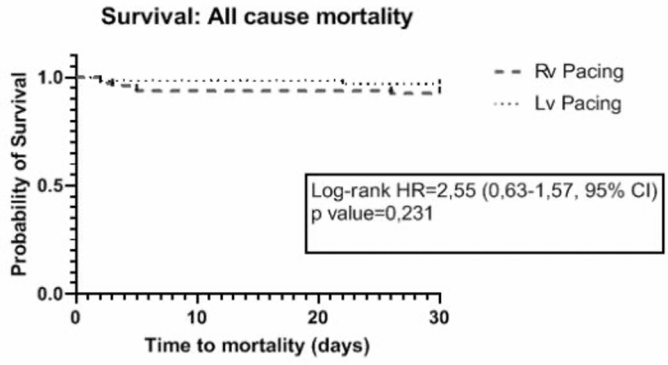

Figure 1. Mortality analysis. Kaplan-Meier curve analysis between LV guidewire pacing group and RV pacing group.

Interventional cardiology / Valve and structural heart diseases

OP-079

Impact of preprocedural pulmonary artery systolic pressure on transcatheter aortic valve replacement-related acute kidney injury

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Background and Aim: Acute kidney injury (AKI) after transcatheter aortic valve replacement (TAVR) is associated with poorer short- and long-term outcomes. The aim of this study was to examine the predictive value of pulmonary artery systolic pressure (PASP) for the development of AKI after TAVR in an attempt to better define risk assessment for this growing population.

Methods: Patients (n=90) with severe aortic stenosis who underwent TAVR were included in this single center retrospec-

tive study. Patients were divided into two groups according to whether TAVR-related AKI developed or not. Logistic regression analysis was used to identify predictors of TAVR-related AKI. Receiver operating characteristic analysis was used to evaluate the performance of PASP for discriminating risk of AKI.

Results: For all patients, the incidence of TAVR-related AKI was 25.6 %. When comparing the value of baseline PASP between the two groups, the TAVR-related AKI (+) group showed a higher values of PASP than the AKI (-) group (55.4 ± 14.0 vs. 37.1 ± 16.3 mm Hg, p<0.001). In the multivariate logistic regression analysis, the independent predictors of AKI were PASP (OR 1.076, 95% CI 1.017-1.139, p=0.011), EuroSCORE (OR 1.238, 95% CI 1.093-1.401, p=0.001), and hypertension (OR 3.544, 95% CI 1.438-5.738, p=0.017). ROC curve analysis revealed that a PASP value higher than 39 mm Hg predicted TAVR-related AKI with a sensitivity of 82.6% and a specificity of 70.7% (AUC: 0.822; p<0.001).

Conclusions: Preprocedural value of PASP was independently associated with TAVR-related-AKI development. Thus, a higher PASP value could be a promising marker of AKI in these patients.

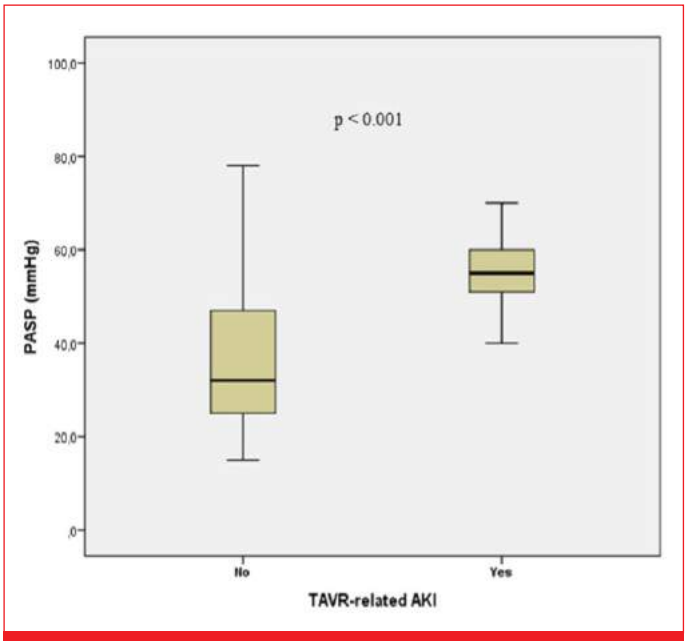

Figure 1.

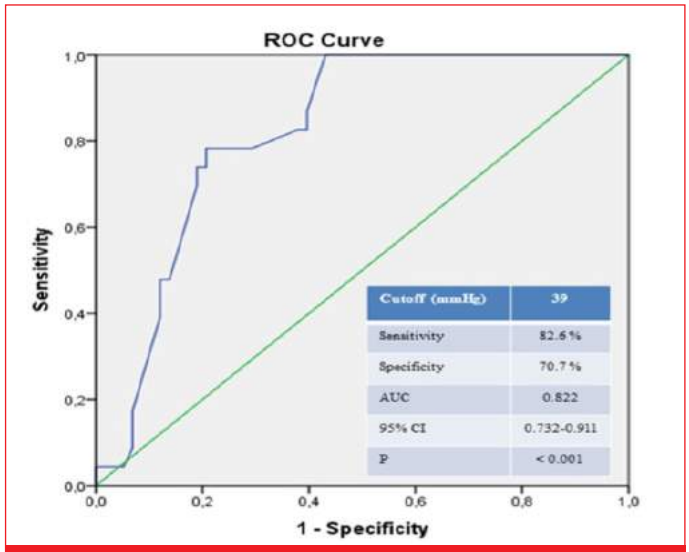

Figure 2.

Table 1. Comparison of data of patients with and without acute kidney injury in TAVI patients

Variables	Acute kidney injury		p Value
	No (n=67)	Yes (n=23)	
Age, years	78 ± 7	80 ± 8	0.228
Female, %	35(52.2%)	16 (69.6%)	0.148
Previous Stroke, %	4(6.0%)	0 (0%)	0.231
Diabetes mellitus, %	18 (26.9%)	8 (34.8%)	0.470
Hypertension, %	52(77.6%)	21 (91.3%)	0.148
Hyperlipidemia, %	23 (34.3%)	7 (30.4%)	0.734
Chronic obstructive pulmonary disease, %	6 (9.0%)	6 (26.1%)	0.037
Coronary artery disease, %	32(47.8%)	12 (52.2%)	0.715
Left ventricular ejection fraction (%)	52.1 ± 11.3	48.8 ± 13.2	0.252
AV area (cm^2)	0.68 ± 0.18	0.63 ± 0.15	0.315
Maximum Aortic gradients, mm Hg	76± 27	79 ± 23	0.569
Mean Aortic gradients, mm Hg	45 ± 17	47 ± 14	0.689
Peak transaortic valve velocity (m/s)	4.3 ± 0.7	4.3 ± 0.7	0.724
Systolic pulmonary artery pressure, mm Hg	37.1 ± 16.3	55.4 ± 14.0	<0.001
Interventricular septum thickness, mm	12.7 ± 2.4	12.7 ± 3.8	0.990
Posterior wall thickness, mm	11.9 ± 2.0	12.0 ± 3.3	0.980
Severe AR, %	20(29.9%)	9(39.1%)	0.414
Low-density lipoprotein cholesterol (mg/dL)	92± 31	104 ± 57	0.246
Triglyceride (mg/dL)	114 ± 47	116 ± 39	0.877
Hemoglobin (g/dL)	11.0 ± 1.6	10.8 ± 1.5	0.753
White blood cell count ($x10^9$/L)	8234 ± 3540	10210± 4273	0.032
Platelet count ($x10^9$/L)	213 ± 82	219 ± 86	0.768
First Creatinine (mg/dL)	1.0 ± 0.33	1.0 ± 0.25	0.470
EuroSCORE (%)	12 ± 11	24 ± 10	0.0001
Prosthesis size, mm	27.3 ± 3.2	26.6 ± 2.2	0.382
Size of pre-dilatation balloon, mm	21.9 ± 2.6	21.1 ± 2.2	0.284
Total amount of contrast volume (ml)	148+25	158+33	0.453
Life-threatening bleeding, %	14(20.9%)	10 (43.5%)	0.035
Cardiac tamponade, %	2(3.0%)	0 (0 %)	0.402
Stroke, %	1(1.5%)	0 (0.0%)	0.556
Permanent pacemaker implantation, %	6(19 %)	0 (0 %)	0.237
New-onset atrial fibrillation/flutter, %	19(28.4%)	11 (47.8%)	0.087

Table 2. Multivariate logistic regression analysis of potential predictors for acute kidney injury

Variable	Clinical covariates adjusted		
	Odds ratio	95% Confidence interval	p Value
Gender	0.195	0.571-18.728	0.198
Hypertension	3.444	1.438 - 5.738	0.017
Chronic obstructive pulmonary disease	0.198	0.017 - 2.295	0.195
Systolic pulmonary artery pressure	1.002	1.017 - 1.139	0.011
White blood cell count ($x10^9$/L)	1.000	1.000 – 1.000	0.814
Life-threatening bleeding	0.397	0.073 – 2.152	0.284
New-Onset Atrial fibrillation	0.448	0.079 – 2.535	0.364
EuroSCORE	1.238	1.093 - 1.401	0.001

Interventional cardiology / Carotid and peripheral vascular

OP-080

Prognostic role of the atherogenic index of plasma on mortality in patients with critical limb ischemia undergoing endovascular revascularization for below-the-knee ischemic lesions

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Background and Aim: Endovascular interventions have been increasingly used for the treatment of patients suffering from below-the-knee (BTK) ischemic lesions. Yet, there is a paucity of data regarding long-term mortality in patients with critical limb ischemia (CLI) undergoing endovascular revascularization for BTK lesions. Recently introduced the atherogenic index of plasma (AIP) has been established for evaluation of plasma atherogenicity and is strongly associated with adverse cardiovascular events. Herein, we aimed to investigate the prognostic role of the AIP on mortality in patients with CLI undergoing endovascular revascularization for BTK lesions.

Methods: The records of 142 patients with symptomatic CLI undergoing endovascular revascularization for BTK lesions between January 2015 and December 2021 were analyzed. Patients were divided into groups with low and high AIP values based on an AIP cut-off value derived from a ROC analysis. For each group, procedural details and follow-up outcomes were analyzed.

Results: The mean follow-up time was 50.3 ± 12.9 months. Regarding follow-up outcomes, major and minor amputation rates were comparable between the two groups (p>0.05). On the other hand, patients with high AIP values had higher rates of mortality compared to patients with low AIP values (57.2% vs. 31.2%, p<0.001). ROC curve is constructed to evaluate the predictive value of long-term mortality of the AIP. The area under the curve of the AIP is 0.850 [95% confidence interval (CI): 0.772–0.928]. The optimal cut-off value is calculated as 0.6285 with a sensitivity of 78.6% and specificity of 80.5%.

Conclusions: AIP is an independent predictor of mortality among patients with CLI who underwent endovascular revascularization for BTK lesions.

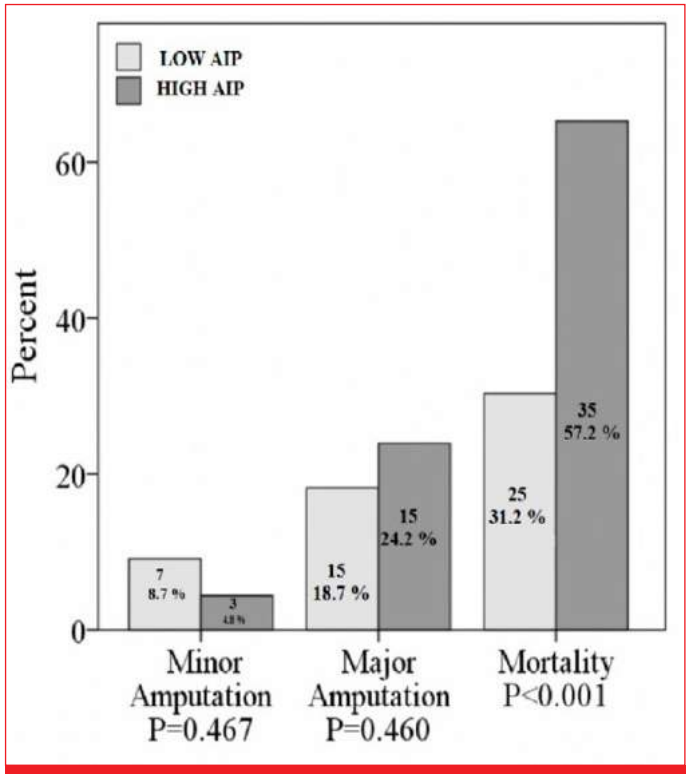

Figure 1. Follow-up outcomes
AIP: atherogenic index of plasma

Table 1. Baseline demographics and clinical characteristics of the study population

Variables	All patients (n=142)	Low AIP (n=80)	High AIP (n=62)	P-value
Age, years	62.8 ± 8.9	62.0 ± 8.6	63.6 ± 9.3	0.465
Male gender, n (%)	109 (76.8)	58 (72.5)	51 (82.2)	0.223
Hypertension, n (%)	120 (84.5)	70 (87.5)	50 (80.6)	0.429
Diabetes mellitus, n (%)	83 (58.4)	42 (52.5)	41 (67.7)	0.079
Hyperlipidemia, n (%)	44 (30.9)	23 (28.8)	21 (33.8)	0.501
Current smoking, n (%)	68 (47.8)	38 (47.5)	30 (48.4)	0.435
Prior CAD, n (%)	83 (58.4)	48 (60.6)	35 (56.5)	0.666
CHF, n (%)	10 (7.1)	7 (9.1)	3 (5.3)	0.119
History of stroke, n (%)	17 (11.9)	6 (7.1)	11 (17.4)	0.068

Table 1. Baseline demographics and clinical characteristics of the study population (Continued)

Variables	All patients (n=142)	Low AIP (n=80)	High AIP (n=62)	P-value
CKD, n (%)	42 (33.9)	18 (22.7)	24 (39.1)	0.061
Atrial fibrillation, n (%)	20 (14.1)	12 (15.2)	8 (13.0)	0.754
Previous contralateral major amputation, n (%)	6 (5.4)	3 (4.5)	3 (6.5)	0.688
Previous ipsilateral minor amputation, n (%)	12 (8.4)	8 (10.0)	4 (6.5)	0.522
Fontaine classification stage III, n (%)	48 (33.8)	32 (40.2)	16 (27.2)	0.141
Fontaine classification stage IV, n (%)	87 (61.2)	44 (55.5)	43 (69.6)	0.076
Rutherford classification stage IV, n (%)	55 (38.7)	36 (45.5)	19 (30.6)	0.109
Rutherford classification stage V, n (%)	54 (38.0)	29 (36.4)	25 (41.3)	0.597
Rutherford classification stage VI, n (%)	31 (21.8)	14 (18.2)	17 (28.3)	0.208
Lesion localization, n (%)				
Popliteal artery n (%)	39 (27.5)	20 (25.8)	19 (30.4)	0.586
Anterior tibial artery, n (%)	76 (52.1)	42 (53.0)	34 (56.5)	0.715
Tibioperoneal truncus, n (%)	11 (7.7)	4 (5.0)	7 (11.2)	0.170
Posterior tibial artery, n (%)	57 (40.1)	30 (37.9)	27 (43.5)	0.552
Peroneal artery, n (%)	35 (24.6)	19 (24.2)	16 (26.1)	0.824
Concomitant PTA above the knee, n (%)	34 (23.9)	13 (16.7)	21 (30.4)	0.085

CAD: coronary artery disease, CHF: congestive heart failure. CKD: chronic kidney disease, PTA: percutaneous transluminal angioplasty,

Table 2. Laboratory parameters and medications

Variables	All patients (n=142)	Low AIP (n=80)	High AIP (n=62)	P-value
Hemoglobin, g/dL	12.1 ± 2.3	12.9 ± 2.1	11.0 ± 2.2	<0.001
WBC, 10^6/L	9.6 (8.1-11.1)	9.1 (7.5-10.2)	10.6 (9.1-12.8)	<0.001
eGFR, mL/min/1.73 m^2	81 (56-95)	89 (62-96)	72 (28-92)	0.007
Total cholesterol, mg/dL	174 ± 46	169 ± 44	180 ± 47	0.225

Table 2. Laboratory parameters and medications (Continued)

Variables	All patients (n=142)	Low AIP (n=80)	High AIP (n=62)	P-value
HDL-c, mg/dL	39.0 (30.5-74.5)	44.0 (32.5-77.0)	28.5 (21.5-32.0)	<0.001
LDL-c, mg/dL	104 ± 37	99 ± 37	112 ± 37	0.066
Triglyceride, mg/dL	115 (87-176)	94 (68-155)	136 (105-198)	0.002
AIP	0.21 ± 0.53	0.14 ± 0.52	0.77 ± 0.23	<0.001
CRP, mg/L	14 (7-43)	12 (6-29)	37 (10-92)	<0.001
Serum glucose, mg/dl	127.8 ± 69.0	113.5 ± 55.5	142.9 ± 80.6	0.077
Neutrophil count, 109/L	6.79 ± 2.87	5.47 ± 1.58	8.69 ± 3.24	<0.001
Lymphocyte count, 109/L	2.11 ± 0.85	2.46 ± 0.82	1.60 ± 0.61	<0.001
Platelet count x 103/mm3	239.5 ± 59.7	225.2 ± 58.3	255.9 ± 60.2	0.035
Medication, n (%)				
Aspirin	107 (75.3)	62 (77.5)	45 (72.5)	0.712
Clopidogrel	18 (12.6)	11 (13.7)	7 (11.2)	0.860
Cilastozol	11 (7.8)	7 (8.7)	4 (6.5)	0.517
Statin treatment	65 (45.7)	38 (47.0)	27 (43.5)	0.780
ACEi/ARB	31 (21.8)	22 (27.3)	9 (15.2)	0.164
Beta-blocker	67 (47.1)	40 (50.0)	27 (43.5)	0.429
Calcium channel blocker	39 (27.4)	23 (28.8)	16 (26.1)	0.711

ACEi/ARB: angiotensin-converting enzyme inhibitor/ angiotensin receptor blocker, AIP: atherogenic index of plasma, CRP: C-reactive protein, eGFR: estimated glomerular filtration rate, HDL-c: high density lipoprotein cholesterol, LDL-c: low-density lipoprotein cholesterol, WBC: white blood cell,

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-081

A novel method: Efficacy and safety of atrial fibrillation ablation without fluoroscopy and protection lead apron after transseptal puncture

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Background and Aim: Fluoroscopy is routinely used for Radiofrequency (RF) catheter ablation atrial fibrillation (AF). Fluoroscopy has severe effects on the human body such as radiation exposure and indirect orthopaedic problems due to protective equipment. Despite the increasing popularity of zero or near fluoroscopy strategies, they are limited by long

procedure times and the need for additional equipment such as intracardiac echocardiography. There is a need for a novel method for atrial fibrillation compatible and cost-effective with daily workflow. We aimed to compare the efficacy and safety of atrial fibrillation ablation without protection lead apron after the transseptal puncture method with the conventional fluoroscopic ablation method.

Methods: All consecutive patients who had undergone RF catheter AF ablation were included in the study retrospectively. After transseptal puncture, protection lead apron had taken off just before 3D reconstruction of the left atrium (LA). Flouroscopy was lock and standby position. Pulmonary vein isolations (PVI) were performed using a multielectrode catheter and smart-touch contact force (CF)-sensing catheter with the mapping system with zero-flouro method after transeptal. In conventional method group,there was used a multielectrode catheter and a smart-touch contact force (CF)-sensing catheter with the mapping system with fluoroscopy entire the operation.

Results: Consecutively 116 patients were included in the study. Of these 71 was in the lead apron free (LAF) group and 45 was in the fluoroscopic conventional ablation (FCA) group. Total fluoroscopy time (6.9 ± 3.6 vs. 13.1 ± 6.7 min, p<0.001) and dose area product (DAP) values (15.4 ± 12.1 vs. 31.5 ± 17.4 G/m^2, p=0.004) were significantly lower in the LAF group. Total procedure times (83.9 ± 21.1 vs. 80.8 ± 22.5 min, p=0.46) were found to be similar between groups. There were only 4 (5.6%) procedures needed to lead apron re-worn. The reason for re-worn were catheters dropped to the right side from LA (n=2), and the need to multiple times re-map due to map shift (n=2). All re-worn protection lead apron requirement was seen in the first twenty cases. There were no procedure-related complications were observed by the new method. Pulmonary vein isolation was achieved for all patients.

Conclusions: The method in Radiofrequency AF ablation that without the protection lead apron after transseptal puncture is safe and effective compared to the conventional method and has similar procedure times. The need for re-worn lead aprons was very low.

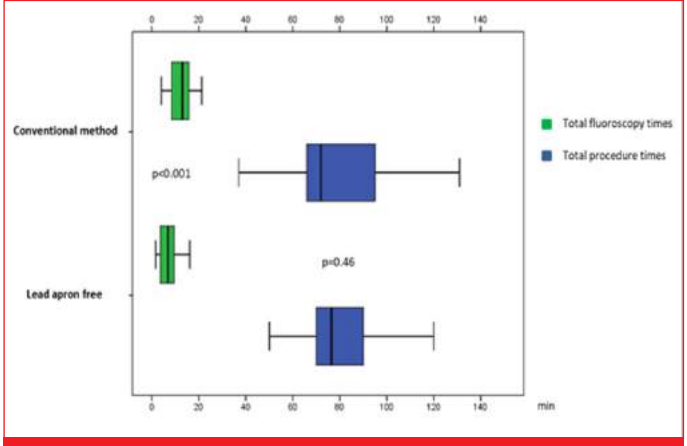

Figure 1. Total procedure and fluoroscopy time Comparation between two groups: total procedure time and flouroscopy time

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-082

Effects of SGLT2 inhibitors as an add-on therapy to metformin on index of cardiac electrophysiological balance in type 2 diabetes mellitus patients

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Background and Aim: Sodium-glucose cotransporter 2 (SGLT-2) inhibitors have metabolic and cardioprotective benefits in patients with DM. Large-scale clinical trials investigating the effects of SGLT-2 inhibitors in patients with DM have shown favorable outcomes in terms of preventing cardiovascular death, nonfatal myocardial infarction, nonfatal stroke, heart failure-related hospitalizations, and progression of chronic kidney disease. Index of cardiac elecctrophysiological balance (ICEB) is a new eletrocardiographical parameter which reflects the balance between ventricular depolarization and repolarization and provides more valuable information about ventricular arrhythmogenesis than other electrocardiography parameters like QT, QTc, QT dispersion. Aim of this studty is to analyse effect of SGLT-2 inhibitors as add-on therapy to metformin on ICEB

Methods: In this prospective cross-sectional study patients were selected fromn consecutive type 2 DM patients who were admitted to the cardiology outpatient clinic between 1st January 2022 and 31st July 2022. After exclusion, 83 patiens who were on metformin monotherapy formed contol group and 75 age and sex matched patients who were switched from metformin monotherapy to metformin+SGLT-2 inhibitor combination therapy due to inadequate glycemic control formed SGLT group. All pharmacological therapies were initiated by endocrinologists who were unaware of the study protocol. Basal and 6th month ECG data of both groups were analysed by 3 experienced cardiologists who were blinded to the patient group. ICEB defined as QT/QRS and ICEBc defined as QTc/QRS.

Results: Although there were no significant differences between SGLT and control groups interms of basal ECG parameters, QT (440 ± 43.91 vs. 450 ± 24.64; p<0.001), QTc (456.38 ± 43.99 vs. 467 ± 32.71; p<0.001), QRS (95.13 ± 12.27 vs. 97.41 ± 21.69; p=0.003), ICEB (4.61 ± 0.552 vs. 4.73 ± 0.482; p<0.0001), ICEBc (4.78 ± 0.558 vs. 4.89 ± 0.439; p<0.0001) values at 6th month were signiicantly lower in SGLT group.

Conclusions: SGLT-2 inhibitors as an add-on therapy to metformin significantly lower ICEB and ICEBc which would improve ventricular susceptibility to arrhythmias in type 2 DM patients as early as the 6th month of treatment.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-083

Stroke and bleeding risks according to age caterogories in patients on edoxaban

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Background and Aim: Elderly non-valvular atrial fibrillation (NVAF) patients are at increased risk for stroke and bleeding. The risk profile of these patients may influence oral anticoagulant (OAC) drug prescription. Evaluation of Treatment Safety in Patients with Atrial Fibrillation on Edoxaban Therapy in Real-Life in Turkey (ETAF-TR) study aimed to investigate real-life edoxaban use in Turkey. We aimed to investigate the relationship among age categories and patient characteristics in this sub-study.

Methods: The data from ETAF-TR study was analysed according to age categories; <65 years old, 65-75 years old, and ≥75 years old.

Results: Of the 1053 edoxaban treated patients, 265 (25.1%) were <65 years old, 397 (37.7%) were 65-75 years old, and 391 (37.1%) were >75 years old. Older patients were more symptomatic according to EHRA classification. Besides NTProBNP levels were higher in these patients. A total of 210 patients were on low dose edoxaban of whom 151 (71.9%) were older than 75 years old. Although CHA2DS2VASc score was higher in elderly comorbidities were evenly distributed among age categories. Bleeding history was not much different according to age categories but risk of bleeding assessed by HASBLED score was higher in the elderly. Antiplatelet and NSAID use were higher and creatinine clearance was lower in older patients. Also, hemoglobin levels were lower in older patients. A comparison of patient characteristics and medications among age categories were given in Table 1.

Conclusions: Older patients with NVAF had higher risk for thromboembolic and bleeding events. The analysis of patient characteristics of ETAF-TR study has shown most of the edoxaban 30 mg users were patients ≥75 years old. Although these patients did not have a more prevalent history of bleeding they had a higher HASBLED score and more risk factors of bleeding. Clinicians should be aware of the need for a tailored oral anticoagulant approach in elderly patients.

Table 1. Patient characteristics according to age categories

	All (n=1053)	<65 years (n=265)	65-74 years (n=397)	≥75 years (n=391)
Male (%)	432 (41)	131 (49.4)	158 (39.8)	143 (36.6)
EHRA Class (%)				
Class I	224 (21.3)	69 (26.0)	101 (25.4)	54 (13.8)
Class II A-B	727 (69.0)	182 (58.7)	264 (66.5)	281 (71.8)
Class III-IV	102 (9.7)	14 (5.3)	32 (8.1)	56 (14.3)
Edoxaban 30 mg	210 (19.9)	13 (4.9)	46 (11.6)	151 (38.6)
Coronary heart disease (%)	303 (28.8)	73 (27.5)	113 (28.5)	117 (29.9)
Congestive heart failure (%)	305 (29.0)	75 (28.3)	102 (25.7)	128 (32.7)
Hypertension (%)	805 (76.4)	188 (70.9)	314 (79.1)	303 (77.5)
Diabetes Mellitus (%)	282 (26.8)	78 (29.4)	112 (28.2)	92 (23.5)
Stroke history (%)	139 (13.2)	36 (13.6)	47 (11.8)	56 (14.3)
Vascular disease (%)	253 (24.0)	62 (23.4)	86 (21.7)	105 (26.9)
Bleeding history (%)	90 (8.5)	20 (7.5)	36 (9.1)	34 (8.7)
CHA2DS2VASc score (mean±SD)	3.5 ± 1.5	2.2 ± 1.2	3.3 ± 1.2	4.5 ± 1.3
HAS-BLED score (mean±SD)	1.6 ± 1.0	0.8 ± 0.9	1.7 ± 0.9	1.9 ± 0.9
Antiplatelet use (%)	128 (12.1)	23 (8.7)	47 (11.8)	58 (14.9)
NSAID use (%)	84 (8.0)	23 (8.7)	21 (5.3)	40 (10.2)
Creatinine Clearance (mean ± SD) ml/min	80.5 ± 31.5	107.6 ± 33.3	81.9 ± 24.7	60.7 ± 20.5
Hemoglobin (mean ± SD) mg/dL	12.9 ± 1.9	13.4 ± 1.8	13.1 ± 1.8	12.3 ± 1.8
NTProBNP (median and IQR) pg/ml	1370.0 (544.0, 3226.0)	847.0 (365.0, 3226.0)	863.0 (174.0, 1848.0)	1753.5 (909.0, 3792.0)

EHRA: European Heart Rhythm Association, IQR: Interquartile range, NSAID: Non-steroidal anti inflammatory durg, SD: Standart deviation

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-084

Demographic, clinical, and disease characteristics of patients with atrial fibrillation on edoxaban therapy according to the bleeding risk: A report from evaluation of treatment safety in patients with atrial fibrillation on edoxaban therapy in real

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Background and Aim: Patients with atrial fibrillation (AF) have been demonstrated by phase III, ENGAGE-AF Trial. ETNA-AF Study confirmed the results of the trial in real life conditions. However, no prospective studies presenting real-life data on edoxaban treatment in Turkey have been performed. Therefore, the ETAF-TR Study was designed to evaluate the safety and efficacy of Edoxaban therapy in routine practice. This substudy aimed to evaluate initial demographic, clinical, and laboratory characteristics of the patients included in the ETAF-TR study according to the bleeding risk.

Methods: The ETAF-TR study (NCT04594915) is a national, multicenter, prospective, observational study that included 1053 cases from 50 centres. Study inclusion criteria; being over the age of 18, being under edoxaban treatment, and giving consent were accepted. Patients were divided into three groups according to the their bleeding risk: low (HASBLED Score: 0-1), medium (HASBLED Score: 2-3) and high (HASBLED Score: 4-6). The primary study endpoint was the composite end point of any overt bleeding (major bleeding, clinically significant non-major bleeding, bleeding that does not meet these criteria but is defined by the clinician as overt bleeding). Bleeding histories, factors causing bleeding tendency, CHADSVASC scores and drug doses were evaluated between the groups.

Results: The mean HASBLED scores of all patients were found to be 1.6 (1.0), and the distribution in low-intermediate and high-risk groups was 0.8 (0.4), 2.3 (0.5), and 4.2 (0.5), respectively. In addition, the median HASBLED scores of the patients were 1.0 (1.0, 2.0) in the whole population, 1.0 (1.0, 1.0) in the low-risk population, 2.0 (2.0, 3.0) in the intermediate-risk population, and 4.0 (4.0,4.0) in the high-risk population. The cases were distributed in the population as 51% (n=537) low risk group, 47% (n=490) medium risk group, 2.4% (n=26) high risk group. The rates of cases over 65 years of age in low, medium and high risk groups were found to be 54.7% (n=294), 86.9% (n=426) and 96.2% (n=25), respectively. The rate of use of 60 mg edoxaban was 86.8% (n=466) in the low-risk group, 74.7% (n=366) in the intermediate-risk group, and 42.3% (n=11) in the high-risk group. Uncontrolled hypertension was found in 3.2% (n=17) in the low-risk group, 18.8% (n=92) in the medium-risk group, and 34.6% (n=9) in the high-risk group. In the high risk group, abnormal kidney function 57.7% (n=15), stroke 46.2% (n=12), bleeding tendency 30.8% (n=8), labile INR 42.3% (n=11), drugs and excessive alcohol drinking 84.6% (n=22) was determined. 8.5% of all cases had a history of bleeding. Intracranial bleeding in 4 (0.4%) cases, upper GIS bleeding in 10 (0.9%) cases, and lower GIS bleeding in 3 (0.3%) cases were detected. In addition, the CHADSVASC score was found to be higher in the group with a high HAS-BLED score (2.8, 4.1, 5.2, respectively).

Conclusions: The substudy obtained clinical and laboratory details of ETAF-TR Study population according to the bleeding risk tertiles.

Table 1. HASBLED score and parameters

	All (n=1053)	Low (n=537)	Intermediate (n=490)	High (n=26)
HASBLED median and IQR	1.0 (1.0, 2.0)	1.0 (1.0, 1.0)	2.0 (2.0, 3.0)	4.0 (4.0, 4.0)
HASBLED mean and std	1.6 (±1.0)	0.8 (±0.4)	2.3 (±0.5)	4.2 (±0.5)
Edoxaban Dose 30 mg	210 (19.9%)	71 (13.2%)	124 (25.3%)	15 (57.7%)
Edoxaban Dose 60 mg	843 (80.1%)	466 (86.8%)	366 (74.7%)	11 (42.3%)
Uncontrolled Hypertension	118 (11.2%)	17 (3.2%)	92 (18.8%)	9 (34.6%)
Abnormal Renal Function	86 (8.2%)	5 (0.9%)	66 (13.5%)	15 (57.7%)
Abnormal Hepatic Function	5 (0.5%)		3 (0.6%)	2 (7.7%)
Stroke	139 (13.2%)	16 (3.0%)	111 (22.7%)	12 (46.2%)
Bleeding	75 (7.1%)	5 (0.9%)	62 (12.7%)	8 (30.8%)
Labil INR (TTR >60%)	111 (10.5%)	10 (1.9%)	90 (18.4%)	11 (42.3%)
Elderly (age >65)	745 (70.8%)	294 (54.7%)	426 (86.9%)	25 (96.2%)
Drugs and Alcohol	271 (25.7%)	40 (7.4%)	209 (42.7%)	22 (84.6%)
Antiplatelet	118 (11.2%)	37 (6.9%)	76 (15.5%)	5 (19.2%)
Alcohol 1/week	32 (3.0%)	18 (3.4%)	12 (2.4%)	2 (7.7%)
Alcohol 1-7 week	12 (1.1%)	3 (0.6%)	9 (1.8%)	

Table 2. Bleeding history

	All (n=1053)	Low (n=537)	Intermediate (n=490)	High (n=26)
Major Bleeding	18 (1.7%)	2 (0.4%)	14 (2.9%)	2 (7.7%)
Minor Bleeding	44(4.2 %)	6 (1.1%)	35 (7.1%)	3 (11.5%)
Intracranial Bleeding	4 (0.4%)		4 (0.8%)	
Upper GIS Bleeding	10 (0.9%)	2 (0.4%)	7 (1.4%)	1 (3.8%)
Lower GIS Bleeding	3 (0.3%)		2 (0.4%)	1 (3.8%)
Soft tissue, muscle, skin bleeding	1 (0.1%)		1 (0.2%)	

Table 3. Ischemic parameters

	All (n=1053)	Low (n=537)	Intermediate (n=490)	High (n=26)
CHADSVASC mean and std	3.5 (1.5)	2.8 (1.3)	4.1 (1.4)	5.2 (1.5)
Congestive Heart Failure	305 (29.0%)	135 (25.1%)	156 (31.8%)	14 (53.8%)
Hypertension	805 (76.4%)	367 (68.3%)	413 (84.3%)	25 (96.2%)
Age <65	265 (25.2%)	209 (38.9%)	55 (11.2%)	1 (3.8%)
Age 65-74	397 (37.7%)	185 (34.5%)	201 (41.0%)	11 (42.3%)
Age >=75	391 (37.1%)	143 (26.6%)	234 (47.8%)	14 (53.8%)
Age >=85	72 (6.8%)	29 (5.4%)	43 (8.8%)	
Diabetes Mellitus	282 (26.8%)	130 (24.2%)	142 (29.0%)	10 (38.5%)
Stroke	139 (13.2%)	16 (3.0%)	111 (22.7%)	12 (46.2%)
Vascular disease	253 (24.0%)	95 (17.7%)	147 (30.0%)	11 (42.3%)
Female	621 (59.0%)	317 (59.0%)	292 (59.6%)	12 (46.2%)
Male	432 (41.0%)	220 (41.0%)	198 (40.4%)	14 (53.8%)

Table 4. AF History and laboratory parameters

	All (n=1053)	Low (n=537)	Intermediate (n=490)	High (n=26)
Paroxysmal	311 (29.5%)	178 (33.1%)	128 (26.1%)	5 (19.2%)
Persistent	82 (7.8%)	39 (7.3%)	41 (8.4%)	2 (7.7%)
Long standing persistent	59 (5.6%)	30 (5.6%)	25 (5.1%)	4 (15.4%)
Permanent	601 (57.1%)	290 (54.0%)	296 (60.4%)	15 (57.7%)
Left atrial appendage closure	1 (0.1%)	1 (0.2%)		
HB(g/dL)	12.9 (1.9)	13.1 (1.9)	12.7 (1.9)	12.2 (2.2)
PLT (mm^3)	241484.9 (73298.8)	244473.2 (73677.2)	238396.5 (72539.7)	237969.2 (79900.7)

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-085

Efficiency of MVP ECG risk score for prediction of long-term atrial fibrillation in patients with ICD for heart failure with reduced ejection fraction

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Background and Aim: The Morphology – Voltage – P – Wave duration (MVP) ECG risk score is a newly defined scoring system that has recently been used for atrial fibrillation prediction. To our knowledge, there are currently no studies evaluating the MVP ECG risk score's ability to predict AF in patients with heart failure with reduced ejection fraction (HFrEF) with an implanted ICD. The aim of this study was to evaluate the ability of the MVP ECG risk score to predict AF in patients with an implantable cardioverter defibrillator and heart failure with reduced ejection fraction in long-term follow-up.

Methods: The study used a single-center, and retrospective design. The study included 328 patients who underwent ICD implantation in our hospital between January 2010 and April 2021, diagnosed with heart failure and had ECG's that could be evaluated in the hospital health data system. Participants who had an ICD implanted for other pathology than HFrEF (such as hypertrophic cardiomyopathy, Brugada syndrome, long QT syndrome) were excluded. The patients were divided into low, intermediate and high-risk categories according to the MVP ECG risk scores. The long-term development of atrial fibrillation was compared among these three groups.

Results: The low-risk group included 191 patients, the intermediate-risk group 114 patients, and the high-risk group 23 patients. The long-term AF development rate was 12.0% in the low-risk group, 21.9% in the intermediate risk group, and 78.3% in the high-risk group. In multivariable analysis, age, left atrial anterior-posterior diameter, and MVP ECG risk score were found to be independent predictors of long-term AF. Patients in the high-risk group were found to have 5.2 times higher rates of long-term AF occurrence compared to low risk group after adjustment for confounding factors.

Conclusions: To best of our knowledge, this is the first study in which the MVP ECG risk score was evaluated to predict AF in patients with an ICD implanted with HFrEF. The MVP ECG risk score, which is an in expensive, simple and easily accessible tool, was found to be a significant predictor of the development of AF in the long-term follow-up of patients with an ICD with heart failure with reduced ejection fraction. This risk score may be used to identify patients who require close follow-up for development and management of AF.

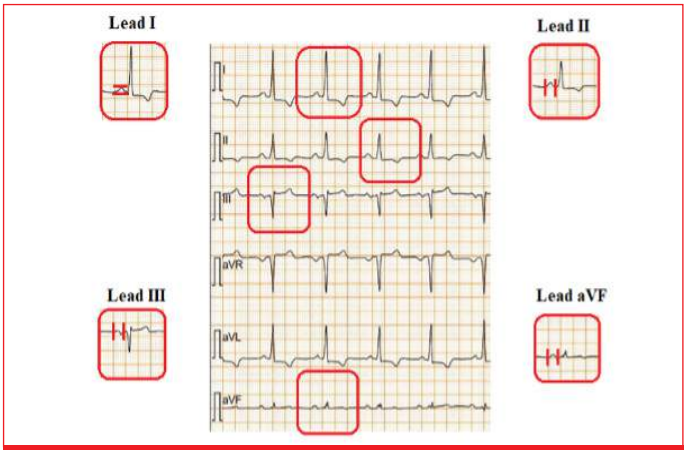

Figure 1. Evaluation of P wave variables on a sample ECG. The P wave voltage is measured from lead D1. PWD is measured from leads D2, D3 and aVF.

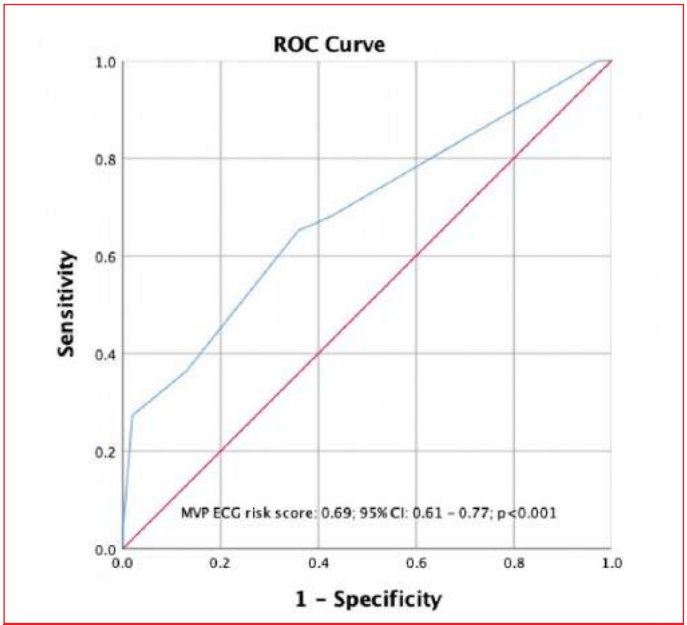

Figure 2. ROC analysis showing that the MVP ECG risk score is 3 with an optimal cut-off value of 3 with 66% sensitivity and 65% specificity to predict long-term atrial fibrillation.

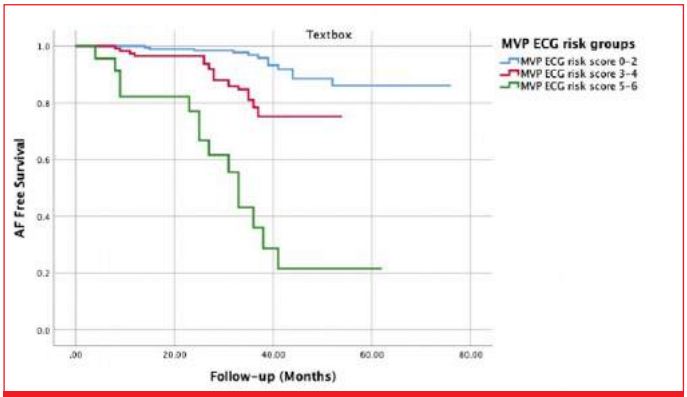

Figure 3. Shown is the cumulative incidence curve of AF free survival according to MVP ECG risk score group, as calculated by means of the Kaplan–Meier method.

Table 1. Basic clinical characteristics and echocardiographic findings of all patients according to MVP ECG risk score.

		MVP ECG risk score		
Basic Clinical Characteristics	**MVP ECG risk score 0-2, n=191**	**MVP ECG risk score 3-4, n=114**	**MVP ECG risk score 5-6, n=23**	**P value**
Age, years	60 (49–70)	62 (54–70)	67 (58–74)	0.067
Gender, Male, n (%)	154 (80.6)	97 (85.1)	17 (73.9)	0.384
Hypertension, n (%)	102 (53.4)	73 (64.0)	18 (78.3)	0.028
Diabetes Mellitus, n (%)	52 (27.2)	45 (39.5)	17 (73.9)	<0.001

Table 1. Basic clinical characteristics and echocardiographic findings of all patients according to MVP ECG risk score. (Continued)

		MVP ECG risk score		
Basic Clinical Characteristics	**MVP ECG risk score 0-2, n=191**	**MVP ECG risk score 3-4, n=114**	**MVP ECG risk score 5-6, n=23**	**P value**
Hyperlipidemia, n (%)	60 (31.4)	47 (41.2)	7 (30.4)	0.198
Chronic renal failure, n (%)	30 (15.7)	19 (16.7)	5 (21.7)	0.773
Coronary artery disease, n (%)	135 (70.7)	82 (71.9)	15 (65.2)	0.812
Percutaneous coronary intervention	81 (42.4)	48 (42.1)	9 (39.1)	0.956
Coronary artery bypass grafting	36 (18.8)	27 (23.7)	2 (8.7)	0.192
Cerebrovascular accident, n (%)	7 (3.7)	6 (5.3)	6 (26.1)	<0.001
ICD Indication				
Primary	76 (39.8)	49 (43.0)	8 (34.8)	0.725
Secondary	115 (60.2)	65 (57.0)	15 (65.2)	0.725
Device Types				
VVI-ICD	167 (87.4)	102 (89.5)	20 (87.0)	0.852
DDD-ICD	24 (12.6)	12 (10.5)	3 (13.0)	0.852
Echocardiographic parameters				
Left ventricular ejection fraction, %	28 (25–35)	27 (20–35)	24 (23–26)	0.043
Left ventricular end-diastolic dimension, mm	56 (51–64)	57 (52–65)	58 (51–65)	0.901
Left ventricular end-systolic dimension, mm	43 (32–54)	44 (35–54)	45 (37–53)	0.893
Left atrial anteroposterior diameter (mm)	37 (35–40)	37 (35–40)	41 (40–44)	<0.001
Out-hospital medication				
Acetylsalicyclic acid	78 (40.8)	60 (52.6)	10 (43.5)	0.133
Beta-blockers	127 (66.5)	83 (72.8)	14 (60.9)	0.378
Statins	52 (27.2)	32 (28.1)	5 (21.7)	0.823
ACEIs or ARBs	91 (47.6)	61 (53.5)	9 (39.1)	0.375
Long-term atrial fibrillation, %	23 (12.0)	25 (21.9)	18 (78.3)	<0.001
Follow-up, Months	39 (31–47)	36 (29–45)	32 (29–34)	

Table 2. Laboratory variables and electrocardiography findings of all patients according to MVP ECG risk score.

		MVP ECG risk score		
Laboratory variables	**MVP ECG risk score 0-2, n=191**	**MVP ECG risk score 3-4, n=114**	**MVP ECG risk score 5-6, n=23**	**P value**
Hemoglobin (g/dl)	13.2 (11.6–14.6)	13.1 (12.1–14.5)	12.9 (11.4–14.7)	0.906
Lymphocytes (%)	2.1 (1.4–2.7)	2.1 (1.4–2.6)	2.2 (1.4–2.7)	0.703
WBC (cells/μL)	9.4 (6.8–11.0)	8.9 (6.6–10.3)	9.9 (7.2–10.9)	0.242
Platelet count (/mm^3)	234 (184–268)	246 (198–295)	219 (182–257)	0.214
Creatinine (mg/dL)	1.2 (0.8–1.2)	1.0 (0.8–1.1)	1.4 (1.0–1.6)	0.141
Urea (mg/dL)	22 (15–27)	21 (15–25)	40 (26–52)	<0.001
TSH (mIU/L)	1.6 (0.8–2.1)	1.7 (0.7–2.0)	2.2 (0.9–2.8)	0.144
Albumin (mg/dl)	3.9 (3.6–4.2)	3.9 (3.7–4.2)	4.0 (3.8–4.4)	0.163
Glucose (mg/dl)	136 (93–140)	133 (93–146)	137 (99–147)	0.842
Electrocardiography parameters				
Morphology in inferior leads				
Non-biphasic (<120 ms)	185 (96.9)	0 (0.0)	0 (0.0)	<0.001
Non-biphasic (>120 ms)	4 (2.1)	91 (79.8)	0 (0.0)	<0.001
Biphasic	2 (1.0)	23 (20.2)	23 (100.0)	<0.001
Voltage in lead I, mV	1.30 (1.11–1.43)	1.30 (1.16–1.48)	0.96 (0.92–1.16)	<0.001
P-wave duration, ms	108 (102–115)	130 (125–134)	138 (128–144)	<0.001
MVP ECG risk score	1.0 (1.0–1.0)	3.0 (3.0–4.0)	5.0 (5.0–5.0)	<0.001

Table 3. Univariable analysis and multivariable model for long-term atrial fibrillation prediction according to admission demographic and clinical characteristics, laboratory parameters, echocardiography and electrocardiography variables.

Univariable Analysis	**P value**	**HR (95% CI)**
Age	0.007	1.027 (1.007–1.048)
Diabetes Mellitus	0.028	1.718 (1.059–2.788)
Cerebrovascular accident	<0.001	4.227 (2.150–8.312)
Left atrium antero-posterior diameter	<0.001	1.151 (1.099–1.207)
Left ventricle ejection fraction	0.042	0.966 (0.935–0.999)
MVP ECG risk score	<0.001	1.733 (1.459–2.057)
Multivariable analysis	P value	HR (95% CI)
Age	0.006	1.031 (1.009–1.053)
Diabetes Mellitus	0.762	0.923 (0.547–1.556)
Cerebrovascular accident	0.043	2.177 (1.025–4.626)
Left atrium antero-posterior diameter	<0.001	1.146 (1.084–1.211)
Left ventricle ejection fraction	0.156	0.976 (0.944–1.009)
MVP ECG risk score	<0.001	1.442 (1.209–1.719)

Table 4. Cox- regression models for long-term atrial fibrillation incidence by Morphology-Voltage-P wave duration (MVP) electrocardiography (ECG) risk score.

		MVP ECG risk score	
	MVP ECG risk score 0-2, n=191	**MVP ECG risk score 3-4, n=114**	**MVP ECG risk score 5-6, n=23**
Long-term atrial fibrillation			
Number of patients	23	25	18
Case rate, %	12.0	21.9	78.3
Long-term atrial fibrillation, HR (95% CI)			
Model 1: unadjusted	1[Reference]	2.2 (1.4–6.2)	6.6 (4.3–24.2)
Model 2: adjusted for all covariatesa	1[Reference]	1.6 (1.1–4.1)	5.2 (2.3 -14.1)

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PB-001 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Clinical outcome in nonagenarians undergoing permanent pacemaker implantation

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Background and Aim: The advanced age and presence of multiple comorbidities necessitate an increased need for permanent pacemakers (PPM) in older age groups. Although life expectancy is a significant determinant in PPM applications, many nonagenarians (age ≥90) continue their lives with PPMs, but the data is scarce. This study aimed to evaluate the clinical characteristics and outcomes in these patients.

Methods: This chart-review was conducted at a private hospital cardiology department and included patients who underwent PPM implantation between December 2005 and December 2019. General characteristics and outcomes of patients older than 90 years-of-age were evaluated retrospectively.

Results: A total of 32 nonagenarians were included in the study. The median age was 91 years, and 59.4% were females. The most common comorbidities were chronic renal failure (25%), hypertension (21.9%), and previous coronary artery disease (21.9%). Half of the patients presented with presyncope (50%); other common symptoms were dizziness (37.5%) and bradycardia (31.3%). Primary etiology was unexplained in 59.4% of cases, and the most common etiological factor was chronic ischemic heart disease (18.8%), followed by sick sinus syndrome in 12.5% of patients. Only four patients (12.5%) had ecchymosis as an acute complication. For chronic complications, two patients (6.2%) had isolation defects, and one patient (3.1%) had pacemaker syndrome. The temporary pace was indicated for ten patients (31.3%), and one patient (3.1%) had a revision upgrade to pacemaker-ICD. The overall mortality rate was 65.6% (Table 1). The median overall survival was 39.3 months (SE: 9.5; 95% CI: 20.8 – 58.0 months). One-, three- and five-year survival probabilities were 77%, 53%, and 37%, respectively (Figure 1).

Conclusions: Advanced age, particularly over 90 years, is a challenging factor for invasive cardiac procedures. But, our results showed a significant survival benefit of PPM implantation for those patients. Both short and long-term survival probabilities were satisfactory, which reached almost 40% in nonagenarians.

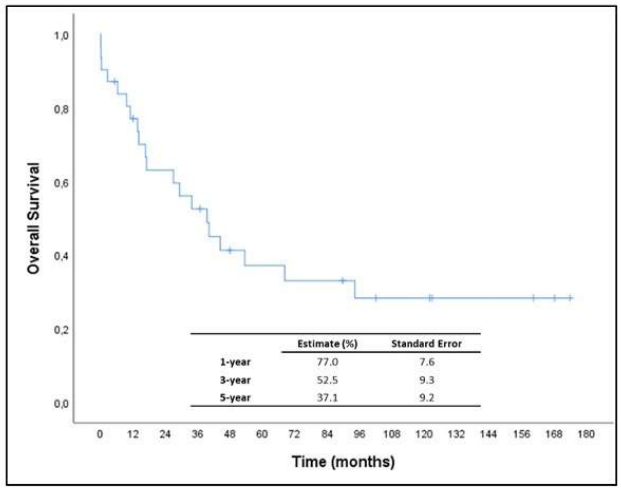

Figure 1. Survival of nonagenarians with permanent pacemaker implantation

Table 1. General characteristics of patients

	Median	Min-Max
Age (years)	91	90 - 99
	n	%
Sex		
Female	19	59.4
Male	13	40.6
Comorbidities		
Chronic renal failure	8	25.0
Hypertension	7	21.9
Previous coronary artery disease	7	21.9
Diabetes	6	18.8
Congestive heart failure	5	15.6
Hyperlipidemia	5	15.6
Left ventricle hypertrophy	2	6.3
Previous Stent	2	6.3
Previous CABG	2	6.3
Carotid arterial disease	2	6.3
Lower extremity peripheral arterial disease	2	6.3
Smoking	2	6.3
Dialysis	1	3.1
Previous MI	1	3.1
Dementia	1	3.1
Presenting symptoms		
Presyncope	16	50.0
Dizziness	12	37.5
Bradycardia	10	31.3
Syncope	9	28.1
Dyspnea	8	25.0
Chest pain	4	12.5

Table 1. General characteristics of patients (Continued)		
	Median	Min-Max
Recurrent syncope	4	12.5
Palpitation	3	9.4
Heart failure	3	9.4
Primary etiology		
Unexplained	19	59.4
Chronic ischemic heart disease	6	18.8
Sick sinus syndrome	4	12.5
Valvular disease	2	6.2
Post-infarct	1	3.1
Dilated cardiomyopathy	1	3.1
Acute complications		
Ecchymosis	4	12.5
Long-term complications		
Isolation defects	2	6.2
Need for temporary pace	10	31.3
Revision upgrade to pacemaker-ICD	1	3.1
Mortality	21	65.6

PB-002 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Effective Therapeutic intervention for LAA thrombus: percutaneous LAA closure

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Background And Aim: Left atrial appendage occlusion is a feasible and effective therapeutic intervention for thromboembolic prevention in patients with non-valvular AF. Some procedural difficulties, such as thrombus formation in LAA, can be challenging for operators and increase periprocedural complication risk. In this clinical study, outcomes of LAA closure in patients with LAA thrombus were evaluated.

Methods: One hundred and fifty consecutive patients had undergone percutaneous LAA closure in our clinic between 2015 and 2021. Sixteen patients had thrombus formation in LAA before occlusion and enrolled in the study. Patients were apprised of a paravalvular leak and thrombus formation on the device with transesophageal echocardiography 1, 6, and 12 months after the procedure. One year after the closure, evaluation with transesophageal echocardiography was done only under clinical suspicion. Major adverse clinical events during follow-up, including disabling stroke, clinically relevant hemorrhage, myocardial infarction, and all-cause mortality, were recorded.

Results: Sixteen patients had been followed for a median of 36 months (1-60 months). The median age was 71.1 +/- 6.7 years. Nine patients were male (56.3%). CHA2DS2-VASc and HAS-BLED scores were calculated at 5 (2-8) and 5 (1-6), respectively. In four patients, LAA occlusion was indicated due to malign LAA. LAA occlusion was performed with Amplatzer Amulet Device in all patients. Postprocedural antiplatelet treatment was decided on clopidogrel, DAPT, or oral anticoagulant plus clopidogrel in 3, 9, and 4 patients, respectively. Five patients died during follow-up. Covid-19 related respiratory failure was responsible for death in three patients. Five patients were hospitalized due to heart failure, and 2 of them died during the hospitalization. Any clinically significant cerebrovascular event or major bleeding was not observed during follow-up. The first month and sixth-month echocardiographic evaluations were done on all patients. Peridevice leak or thrombus formation was not observed in any patients.

Conclusions: LAA closure in patients with LAA thrombus is a feasible and effective method to reduce thromboembolic risk. It can be performed as an alternative therapy to OACs in patients who have contraindications to OACs or malign LAA.

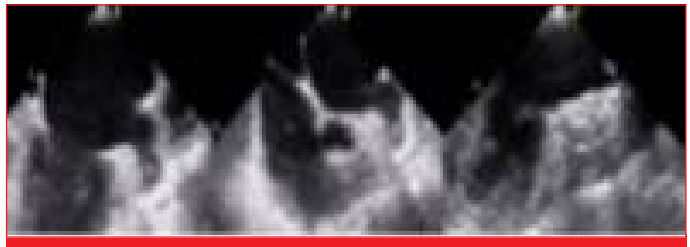

Figure 1. LAA closure in patient with LAA thrombus

PB-003 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

The Impact of PlasmaBlade on Cardiac Implantable Electronic Device Generator Replacement Procedures

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Background and Aim: The pulsed electron avalanche knife (PEAK) PlasmaBlade (Medtronic Inc., Minneapolis, MN, USA) is a low-thermal-injury electrosurgical device that uses brief, precise pulses of radiofrequency energy to cut and coagulate soft tissue. The PEAK PlasmaBlade applies electrical plasma with a lower peak temperature which may reduce the risk of lead insulation damage. The aim of this study was to compare our experience in cardiac implantable electronic device (CIED) generator replacement procedures performed with the PEAK PlasmaBlade with conventional replacement technique.

Methods: We retrieved our data about patients undergoing CIED generator replacement procedure at our center between December 2019 and December 2021. Patients who underwent a CIED generator replacement procedure using with the PlasmaBlade constituted the study group. Patients

who underwent a CIED generator replacement by conventional replacement technique constituted the control group.

Results: There were 58 patients (Mean age 62.51±17.81) in study group and 104 patients (Mean age 63.82±14.65) in the control group. Procedure time in the study group was significantly shorter than in control group (40.43±9.74 vs. 48.91±9.87 minutes; P < 0.001). There were no damaged leads in the study group in comparison to 4 (3.8%) the control group. Device features, procedure durations and outcomes of the groups were shown in tables 1 and 2.

Conclusions: Use of PlasmaBlade for generator replacement resulted in significantly reduced procedure time while avoiding lead damage.

Table 1. Device types and durations

Variables	Study Group (n=58)	Control Group (n=104)	p value
ICD, n (%)	46(79.3%)	80(76.9%)	0.726
Single chamber, n (%)	6(10.3%)	10(9.6%)	0.881
Dual chamber, n (%)	20(34.5%)	38(36.5%)	0.794
Three chamber, n (%)	32(55.2%)	56(53.8%)	0.871
Pace Maker, n (%)	12(20.7%)	24(23.1%)	0.726
Number of previous device procedures, n (%)			
1	50(86.2%)	92(88.5%)	0.676
2	8(13.8%)	12(11.5%)	0.676
Procedure duration, (min)	40.43±9.74	48.91±9.87	<0.001
Length of hospital stay, (day)	1.62±.072	1.49±0.72	0.273

*p<0.05 statistically significant. Continues variables are reported (mean±SD). Categorical variables are reported n (%).
Abbreviations: ICD; Implantable Cardioverter Defibrillator

Table 2. Outcomes of both replacement techniques

Variables	Study Group (n=58)	Control Group (n=104)	p value
Damage to Device Lead, n (%)	0(0%)	4(3.8%)	0.298
Dislocation of generator/Pocket erosion, n (%)	0(0%)	2(1.9%)	0.537
Post-operative hematoma, n (%)	2(3.4%)	7(6.7%)	0.381
Post-operative infections, n (%)	1(1.7%)	2(1.9%)	0.928

*p<0.05 statistically significant. Continues variables are reported (mean±SD). Categorical variables are reported n (%).

PB-004 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Comparing to indexes of ventricular arrhythmia between diabetics and non-diabetic metabolic syndrome patients

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Background and Aim: On surface ECG, measuring the time between the peak and end of the T wave, which is called as Tp-Te interval and reflects transmural dispersion of repolarization and QTc, Q-Tpeak (QTp), Tp-Te interval, and Tp-Te/QT have been proposed as predictors of risk for ventricular arrhythmia or mortality in various clinical scenarios like in HF patients. Tp-Te interval, Tp-Te/QT ratio end Tp-Te/QTc ratio were found to be prolonged in diabetic patients. In another study these indexes were found to be significantly increased in Met-S patients. Comparisons of these parameters between diabetic and non-diabetis patients with Met-S never evaluted before so we aimed to assess this.

Methods: The study was conducted with 100 patient with a new diagnosis of Met-S (diabetic group n: 51 [33,3%], non-diabetic group n: 49 [32%]) and normal control group (n: 53 [34,6%]). The diagnosis of Met-S was made with the IDF diagnostic criteria. Patients with history of coronary heart disease, valvular heart disease, hypertension, hyperlipidemia and chronic renal disease were excluded from the study. QTc interval and Tp-Te/QTc ratio were assessed with electrocardiographic analysis with paper speed of 50 mm/s by a same experienced cardiologist who was blind to study.

Results: Patients were divided into 3 group as normal, non-diabetic and diabetic group. Baseline features were shown in table 1. Fasting glucose, creatinine and uric acid were found to be significantly different between groups. Only Tp-Te/QTc ratio was found to be significantly different between groups and post-hoc analysis of Tp-Te/QTc and glucose were shown in table-2. Tp-Te/QTc was found to be different only between diabetic and control group.

Conclusions: Measuring the time between the peak and end of the T wave, which is called as Tp-Te interval and reflects transmural dispersion of repolarization and also QTc, Q-Tpeak (QTp), Tp-Te interval, and Tp-Te/QT have been proposed as predictors of risk for ventricular arrhythmia or mortality in various clinical scenarios. In previous studies these indexes were found to be significantly increased in Met-S as well as diabetic patients. However, comparing these indexes between diabetic and non-diabetic patients in Met-S population has been never performed before. Our study showed the only Tp-Te/QTc ratio was significantly difference among groups. Only diabetic and control groups showed difference, but non-diabetic and diabetic groups did not. In other terms non-diabetic and diabetic patients may have nearly same risk level for arrhythmia. But to clarfy the differences for arrhythmia risk level between these groups, many other predictors are needed to study. So, predictors of ventricular arrthymia may be prolonged only diabetics in Met-S patients comparing to control group and non-diabetic and diabetic patients have similar risk levels. Diabetic patients with Met-S have more predictors of ventricular arrthymia than non-diabetic patients with Met-S.

Table 1. Baseline characteristics and comparisons of groups

Variables		Normal Group 0 n:53 (34,6%)	Non-DM Group 1 n:49 (32%)	DM Group 2 n:51 (33,3%)	Total n:153 (100%)	P
Gender						
Woman	(count & percent in total)	25 (16,3%)	22 (14,3%)	26 (16,9%)	73 (47,8%)	0,827ψ
Male	(count & percent in total)	28 (18,3%)	27 (17,6%)	25 (16,3%)	80 (52,2%)	
Age	Mean±std Min: 35 Max: 97	56,2 ± 7,8	59,0 ± 8,1	59,8 ± 8,1	57,8 ± 8,5	0,723&
Glucose (mg/dL)	Mean±std Median (25%-75%)	94,2 ± 8,7 93,5 (88 – 99,5)	98,0 ± 7,9 97,0 (92,5 – 102,5)	155,2 ± 54,8 143,0 (117,5 – 182,5)	112,6 ± 38,9	<0,0001β
Tp-Te/ QTc	Mean±std Median (25%-75%)	0,195 ± 0,04 0,194 (0,178 – 0,208)	0,188 ± 0,03 0,192 (0,166 – 0,206)	0,181 ± 0,04 0,182 (0,149 – 0,200)	0,026 ± 0,02	0,046 &
QTc (msn)	Mean±std Median (25%-75%)	415,2 ± 29,4 421 (400 – 436)	410,9 ± 35,9 410 (400 – 436)	423,7 ± 34,1 425 (403 – 447)	1,822 ± 1,53	0,089 &
CRP (mg/dL)	Mean±std Median (25%-75%)	2,6 ±2,5 1,7 (0,8 – 4,1)	3,3 ± 2,4 2,25 (1,5 – 5,6)	3,6 ± 2,6 2,4 (1,4 – 5.9)	5,720 ± 5,35	0,168 &

Variables		Normal Group 0 n:53 (34,6%)	Non-DM Group 1 n:49 (32%)	DM Group 2 n:51 (33,3%)	Total n:153 (100%)	p
Gender						
Woman	(count & percent in total)	25 (16,3%)	22 (14,3%)	26 (16,9%)	73 (47,8%)	0,827ψ
Male	(count & percent in total)	28 (18,3%)	27 (17,6%)	25 (16,3%)	80 (52,2%)	
Age	Mean±std Min: 35 Max: 97	56,2 ± 7,8	59,0 ± 8,1	59,8 ± 8,1	57,8 ± 8,5	0,723&
Glucose (mg/dL)	Mean±std	94,2 ± 8,7	98,0 ± 7,9	155,2 ± 54,8	112,6 ± 38,9	<0,0001β
	Median (25%-75%)	93,5 (88 – 99,5)	97,0 (92,5 – 102,5)	143,0 (117,5 – 182,5)		
Tp-Te/ QTc	Mean±std	0,195 ± 0,04	0,188 ± 0,03	0,181 ± 0,04	0,026 ± 0,02	0,046 &
	Median (25%-75%)	0,194 (0,178 – 0,208)	0,192 (0,166 – 0,206)	0,182 (0,149 – 0,200)		
QTc (msn)	Mean±std	415,2 ± 29,4	410,9 ± 35,9	423,7 ± 34,1	1,822 ± 1,53	0,089 &
	Median (25%-75%)	421 (400 – 436)	410 (400 – 436)	425 (403 – 447)		
CRP (mg/dL)	Mean±std	2,6 ±2,5	3,3 ± 2,4	3,6 ± 2,6	5,720 ± 5,35	0,168 &
	Median (25%-75%)	1,7 (0,8 – 4,1)	2,25 (1,5 – 5,6)	2,4 (1,4 – 5.9)		

&: One-Way ANOVA β: Independent samples non-parametric Kruskal-Wallis test ψ: Chi-square test

Table 2. Post-hoc analysis

Variables	Group 0 vs 1	Group 0 vs 2	Group 1 vs 2
Tp-Te/QTc	Ns. α	0,037 α	Ns. α
Glucose	Ns. μ	<0,001 μ	<0,001 μ

α: Tukey HSD post- hoc analysis μ: Mann-Whitney U test

Table 3. Correlations

Variables		Spearman's rho	Group 0	Group 1	Group 2
	CRP	r	0,05	-0,001	-0,009
		p	Ns	Ns	Ns
Tp-Te/QTc	Glucose	r	-0,045	-0,166	0,177
		p	Ns	Ns	Ns
	Age	r	-0,122	-0,203	-0,141
		p	Ns	Ns	Ns

PB-005 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Arrhythmia predictive ECG parameters in Takayasu arteritis patients

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Background and Aim: Takayasu arteritis (TA) is a systemic disease characterized by large vessel vasculitis. Concomitant coronary artery disease, increased afterload and aortic insufficiency are not rarely and among the causes of abnormal coronary perfusion in TA patients. Our aim in this study was to investigate the predictive ECG parameters of arrhythmia potential in patients with takayasu arteritis.

Methods: Forty consecutive patients (mean age: 40 ± 11 years, 9 male) who applied cardiology outpatient clinic and had Takayasu diagnosis were included in the study. Forty healthy subjects were included as controls. All patients underwent 12-lead electrocardiography (ECG) to determine P wave (PW), QT interval, PW and QT dispersions, and Tp-e interval.

Results: The characteristics and ECG parameters of the patients and controls are listed in Table-1. Takayasu patients had similar heart rates (79 ± 16 vs. 75 ± 11 p:0.281) and QT dispersion compared to controls (37 ± 12ms vs. 37 ± 19 ms p:0.397). Takayasu patients had significantly longer Tp-e interval compared to controls (85 ± 13 msn vs. 68 ± 10 msn p<0.001). QTc durations of the patients were significantly longer compared to controls (QTcmax: 437 ± 24 ms vs. 419 ± 31 ms p:0.002; QTcmin: 394±21 ms vs. 378±29 ms p:007). while there were not any significant differences in QTc dispersion (42 ± 15ms vs. 43 ± 20 msn p:0.617). Takayasu patients had higher PWD compared to controls (54 ± 15ms vs. 32± 14 ms p<0.001).

Conclusions: Takayasu causes prolongation in PWD, QTc and Tp-e intervals. The presence of high-risk ECG parameters for arrhythmias in TA patients should be kept in mind for possible arrhythmias in the follow-up and treatment of these patients.

Table 1. The characteristics of ECG Parameters the patients and controls

	Patient (n= 40)	Controls (n= 40)	P
Age (years)	40 ± 11	45 ± 12	0.167
Male sex (n – %)	9 (37.5 %)	10 (25 %)	0.793
Heart rate (/min)	79 ±16	75 ± 11	0.281
RR (msec)	785 ± 161	762 ± 183	0.556
PR (msec)	145 ± 20	150 ± 18	0.190
QRS (msec)	86 ± 10	84 ± 7	0.855
QTmax (msec)	385 ± 39	376 ± 31	0.500
QTmin (msec)	347 ± 38	338 ± 28	0.288
QTDisp (msec)	37 ± 12	37 ± 19	0.397
QTcmax (msec)	437 ± 24	419 ± 31	0.002
QTcmin (msec)	394 ± 21	378 ± 29	0.007
QTcDisp (msec)	42 ± 15	43 ± 20	0.617
Tp-e (msec)	85 ±13	68 ± 10	<0.001
PW max (msec)	104 ± 13	98 ± 13	0.069
PW min (msec)	50 ± 10	67 ± 9	<0.001
PWD (msec)	54 ± 15	32 ± 14	<0.001

QTc: Corrected QT, QTc disp: Corrected QT dispersion, PWD: P Wave dispersion

PB-006 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

The Effect of mild-moderate C-reactive protein elevation to cardiac implantable electronic device infection

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Background and Aim: CIED implantation is one of the procedures in which preoperative antibiotic prophylaxis standards are not applied within the scope of preventive measures in surgical infections due to the high mortality rate due to infection complications. The present study aimed to discuss the effect of mild to moderately high levels of CRP without systemic infection symptoms on CIED infections.

Methods: The study was designed as a single-center observational study. The patients were grouped as CRP levels < 10 mg/dl, 10-60 mg/dl. The presence of pocket infection, infective endocarditis, and demographic/clinical characteristics of the patients are compared statistically between the groups.

Results: The total number of patients enrolled in the study was 158. There was no statistical difference between the groups on demographic and clinical characteristics. (Table 1) Laboratory findings were similar between the groups except for white blood count (WBC). WBC was statistically significantly higher than group 1 in group 2 patients. (p<0.05) (Table 2) CIED infection was not statistically differentiated between groups. (p=0.685)

Conclusions: CIED procedures can be performed with standard preoperative AB prophylaxis in patients with incidentally detected mild to moderately high CRP levels without systemic signs of infection.

Table 1. Demographic and clinical characteristics of the patients

		CRP		P
		Group I (n=100)	Group II (n=58)	
Sex	Female	36 (%36,0)	18 (%31,0)	0,645 [a]
	Male	64 (%64,0)	40 (%69,0)	
Age(year)		69,0 ± 12,1	66,4 ± 10,5	0,166 [b]
Antibiotics	Cefazol	100 (%100,0)	58 (%100,0)	1,000 [c]
Type of CIED	Pacemaker	28 (%28,0)	23 (%39,7)	0,297 [d]
	ICD	53 (%53,0)	27 (%46,6)	
	BIV-ICD	19 (%19,0)	8 (%13,8)	
Type of Procedure	Initial	68 (%68,0)	39 (%67,2)	0,575 [d]
	Battery Replacement	26 (%26,0)	13 (%22,4)	
	Battery Replacement + Lead Revision	6 (%6,0)	6 (%10,3)	
CIED Pocket Infection	No	100 (%99)	55 (%96,5)	0,685 [c]
	Yes	1 (%0,1)	2 (%3,5)	
Type of Suture	Aesthetic	97 (%97,0)	57 (%98,3)	1,000 [c]
	Matrix	3 (%3,0)	1 (%1,7)	
Indications	Ischemic CMP	43 (%43,0)	21 (%36,2)	--
	Non-Ischemic CMP	16 (%16,0)	7 (%12,1)	
	AV Block	20 (%20,0)	20 (%34,5)	
	Syncope	4 (%4,0)	1 (%1,7)	
	Heart Failure Resen. Therapy	11 (%11,0)	7 (%12,1)	
	Multi Indication	3 (%3,0)	2 (%3,4)	
	Symptomatic Pause	2 (%2,0)	0 (%0,0)	
	Lead Revision	1 (%1,0)	0 (%0,0)	
EF	<35	64 (%64,0)	31 (%53,4)	0,288 [d]
	35-50	5 (%5,0)	6 (%10,3)	
	>50	31 (%31,0)	21 (%36,2)	
PHT	35-45 mmhg	78 (%78,0)	49 (%84,5)	0,435 [a]
	45-55 mmhg	22 (%22,0)	9 (%15,5)	
Medications	ASA	47 (%47,0)	31 (%53,4)	0,435 [d]
	NOACs	16 (%16,0)	9 (%15,5)	1,000 [a]
	Clopidogrel	24 (%24,0)	12 (%20,7)	0,778 [a]
	Warfarin	9 (%9,0)	7 (%12,1)	0,732 [a]
	Beta-blocker	75 (%75,0)	38 (%65,5)	0,276 [a]
	ACEI	48 (%48,0)	28 (%48,3)	0,973 [d]
	ARB	24 (%24,0)	8 (%13,8)	0,182 [a]
	Statin	51 (%51,0)	24 (%41,4)	0,243 [d]
	Spirinolactone	46 (%46,0)	18 (%31,0)	0,093 [a]

Group I: Crp <10 mg/dl, Group II: Crp ≥10 mg/dl
a: Yates' Corrected Chi-Square Test, b: Independent Samples t Test, c: Fisher's Exact Test, d: Pearson's Chi-Square Test,

Table 2. Laboratuar findings and Comorbidities of the patients

		CRP		P
		Group I (n=100)	Group II (n=58)	
WBC		7.971,2 ± 2.027,3	8.798,1 ± 2.740,4	**0,032** [b]
	<10000	90 (%90,0)	44 (%75,9)	**0,031** [a]
	≥10000	10 (%10,0)	14 (%24,1)	
Neutrophil		66,4 ± 9,6	66,1 ± 11,0	0,849 [b]
Lymphocyte		23,7 ± 8,5	20,9 ± 7,9	0,51 [b]
Creatinin		1,1 ± 0,4	1,2 ± 0,9	0,169 [b]
Crcl		70,1 ± 20,8	65,8 ± 21,9	0,226 [b]
Comorbidities	AF	28 (%28,0)	16 (%27,6)	1,000 [a]
	Hypertension	67 (%67,0)	36 (%62,1)	0,650 [a]
	Diabetes Mellitus	24 (%24,0)	14 (%24,1)	1,000 [a]
	Hyperlipidemia	47 (%47,0)	24 (%41,4)	0,494 [d]
	COLH	5 (%5,0)	6 (%10,3)	0,214 [c]
	Malignancy	2 (%2,0)	0 (%0,0)	0,532 [c]
	Smoking	20 (%20,0)	12 (%20,7)	1,000 [a]
	Heart Failure	70 (%70,0)	37 (%63,8)	0,530 [a]
	Cerebrovascular Disease	1 (%1,0)	2 (%3,4)	0,555 [c]
	Ateroscleroti c Vascular Disease	63 (%63,0)	33 (%56,9)	0,556 [a]

Group I: CRP <10 mg/dl, Group II: CRP≥10 mg/dl
a: Yates' Corrected Chi-Square Test, b: Independent Samples t-Test, c: Fisher's Exact Test, d: Pearson's Chi-Square Test,
ACEI: Angiotensin-converting enzyme inhibitör, ASA: Acetylsalicylic acid, CRP: C-Reactive Protein, NOACs: Novel Oral Anticoagulants, ARB: Angiotensin-converting enzyme inhibitör, PHT: Pulmonary Hypertension, WBC: White Blood Cells

PB-007 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Fragmented QRS and stroke severity in patients with acute ischemic stroke

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Background and Aim: Acute ischemic stroke results from cerebral deficient blood supply caused by cerebral vascular thrombosis, cerebral embolism or other causes.The cardiovascular manifestations of acute ischemic stroke have been well known. Apart from preexisting cardiac disorders, cardiovascular complications are most likely mediated by an increased sympathetic activity. Therefore, severity of myocardial damage detected through electrocardiogram (ECG) may reflect the severity of primary cerebral injury to a certain extent. Electrocardiographic changes, which are frequently encountered in stroke patients, have also been shown to be associated with long-term prognosis. The presence of fragmented QRS (fQRS) on electrocardiogram (ECG) has been increasingly examined in recent years and is a depolarization abnormality caused by the presence of myocardial regional scar. However, it is uncertain fQRS are related with stroke severity. In this study, we aimed to investigate the relationship between fQRS and stroke severity in patients with acute ischemic stroke.

Methods: We retrospectively studied 97 adult patients (57 men, 40 women, 65 ± 12 years) with acute ischemic stroke (≤24 h of symptom onset) admitted to the neurology care unit, between 1 September 2021 and 30 May 2022. NIHSS scores were calculated. Patients were divided into 2 groups according to the NIHS score (Group 1; NIHSS < 16, Group 2; NIHSS ≥ 16). Demographic, clinical, and laboratory data were collected for all patients. Cardiac evaluation with two-dimensional echocardiography was performed within 48 hours of admission to neurology care unit. ECG was performed at admission to neurology care unit. A diagnosis of fQRS was made based on following criteria; (I) an RSR' pattern with or without Q waves in two consecutive leads (QRS duration < 120 ms), (II) an additional R wave (R' wave) or notch in the S wave, and (III) presence of more than one R wave without typical bundle branch block.

Results: There were a significant difference among some laboratory parameters of patients (Table 1). LVEF was significantly lower in Group 2 patients than Group 1 patients. Electrocardiographic parameters were significantly higher in Group 2 patients than Group 1 patients. fQRS was significantly higher in Group 2 patients than Group 1 patients. (Table 2).

Conclusions: Our results suggested that fQRS score is associated with stroke severity on admission in patients with acute ischemic stroke.

Table 1. Clinical characteristics of patients

Variables	Group-1 (NIHSS <16) n=69	Group-2 (NIHSS ≥ l6 Group) n=28	p Value
Age (year)	56.9 ± 11.8	69.3 ± 14.1	NS
Gender (F/M)	28 / 41	12 / 16	NS
Hypertension	24 (35%)	10 (38%)	NS
Diabetes Mellitus	22 (33%)	10 (36%)	NS
Smoking	20 (29%)	9 (32%)	NS
Hyperlipidemia	24 (36%)	11 (39 %)	NS
SBP (mm-Hg)	139.4±15.8	165.1±16.3	<0.05
DBP (mm-Hg)	71.4±10.3	87.5±12.4	<0.05
Glucose (mg/dL)	142.7±32.5	203.7±48.5	<0.05
Creatinine (mg/dL)	1.1±0.5	1.8±0.9	<0.05
HbA1c (%)	6.4±1.63	9.7±1.72	<0.05
Infarct volume (mL)	19.2 ± 3.4	47.5 ± 5.2	<0.05
Troponin (ng/L)	6.7 ± 2.4	14.8 ± 5.7	<0.05
LVEF (%)	59.2 ± 6.4	51.6 ± 5.8	<0.05

Table 2. Electrocardiographic parameters of patients

Variables	Group-1 (NIHSS <16) n=69	Group-2 (NIHSS ≥ l6 Group) n=28	p Value
Heart rate	89.3 ± 11.5	98.1 ± 13.9	<0.05
QRS duration (ms)	92.3 ± 10.5	96.7 ± 12.2	>0.05
QTc (ms)	481±46.5	541±67.3	<0.05
QTd (ms)	63.2±4.3	87.4±3.9	<0.05
QTcd (ms)	64.7±3.2	92.8±3.7	<0.05
fQRS, n (%)	11 (16 %)	9 (32 %)	<0.05

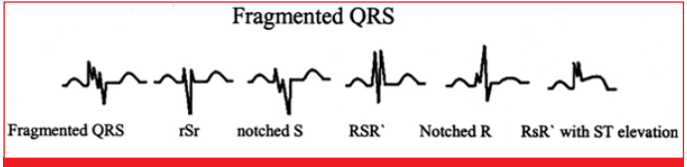

Figure 1. Fragmented QRS

PB-008 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Estimation of atrial fibrillation from lead-I ECGs: Comparison with cardiologists and machine learning model (CurAlive), a clinical validation study

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Background and Aim: Electrocardiogram (ECG) recognition of cardiac arrhythmias is critical for cardiac abnormality diagnosis. Because of their strong prediction characteristics, artificial neural networks (ANNs) are the preferred method in medical diagnosis systems (MDS). This study presents a method to detect atrial fibrillation with lead-I ECGs using artificial intelligence. The aim of the study is to compare the accuracy of the diagnoses estimated by cardiologists and artificial intelligence over lead-I ECGs using 12-lead ECGs as references.

Methods: The clinical study aimed to investigate how much information can be extracted from the ECG using only lead-I, and to compare the diagnostic performance of the artificial intelligence supported ECG analysis system of CurAlive, a product of Notrino Research, with the diagnostic performance of cardiologists on lead-I. Thus, the role of dry electrode and single lead mobile ECG devices in AFIB diagnosis will be examined in the future. Using the image annotation tool zillin.io, all 328 ECG files prepared for cardiologists' evaluation were evaluated separately from one another according to the AFIB, NOT- SURE, NSR, and OTHER groups. All of the same ECGs were also analyzed by CurAlive for comparison.

Results: The precision performance averages of the cardiologists participating in the study ranged from 79.1% to 84.6%. When the diagnostic performances performed by using the lead-I derivation were compared, the average precision of the diagnostics performed by the cardiologists was obtained 82.2%. CurAlive's average precision was found as 94.1%. The average F1 score of the cardiologists for AFIB was found to be 85.1%, whereas that of CurAlive was obtained as 97.0%. Moreover, while the average F1 score for NSR of cardiologists was determined as 44.1%, CurAlive's F1 score average was 83.6%. The average of OTHER F1-score was calculated as 54.2%, while CurAlive's average score was %95.3. It was determined that the accuracy of the cardiologists

was 54.6%, while the accuracy of CurAlive was 93.6%.

Conclusions: CureAlive identified AFIB using lead-I ECGs, with nearly 12-lead ECGs sensitivity and significantly higher diagnostic power than cardiologists which use lead-I.These results are promising in increasing the early diagnosis capacity of remote patient monitoring systems. In this way, it can be said that CureAlive can contribute to the diagnosis of cardiac emergencies practically, early, quickly and cheaply. With future enhancements, it is estimated that CurAlive will also be able to detect other abnormal rhythms and beats that can only be identified with 12-lead ECGs using lead-I ECGs. Based on these results, it is planned to conduct a research on diagnosis of MI from lead-I ECGs using prospective patient data. It can be speculated that CurAlive will be capable of diagnosing other abnormalities thanks to its flexible design, so it is likely to be used for the diagnosis of other heart, kidney, and endocrinological diseases in the early- future.

PB-009 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Usefulness of the systemic immune inflammation index to predict atrial fibrillation recurrence after cryoballoon-based catheter ablation

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Background and Aim: Catheter-based atrial fibrillation (AF) ablation is an effective and reliable therapy in the treatment of paroxysmal atrial fibrillation patients. However, postprocedural AF recurrences are still remaining a major clinical problem. Several factors and non-invasive clinical markers have been investigated to predict the success rate of catheter ablation and appropriate patient selection for interventional

strategy, but results have not been consistent. Inflammation is a well-established risk factor in the initiation and perpetuation of atrial fibrillation. The systemic immune inflammation index (SII),) has emerged as a new predictor of inflammation and is determined using the following equation: neutrophil (N) × platelet (P) ÷ lymphocyte (L). To the best of our knowledge, the impact of pre- procedural SII level, as a pro-inflammatory marker, on AF recurrence following cryoballoon-based AF ablation has never been studied before. The aim of this study was to establish whether there is a relationship between SII level and recurrence of AF after catheter ablation.

Methods: This study was a non-randomized single center trial. All data including clinical, laboratory and procedural were retrospectively examined and prospectively analyzed. The study population consisted of 370 consecutive patients (mean age 56.1±12.3 years, 50.5% male) with drug-resistant symptomatic AF who underwent initial PV isolation with cryoballoon technique for documented AF. Patients were categorized into two groups according to their pre-procedural SII levels. Post-ablation blanking period was observed for 3 months.

Results: At a mean follow-up of 25.0±6.7 months, 77 patients (20.8%) had developed AF recurrence. On multivariate Cox regression analysis, pre-ablation SII level (HR: 2.27, 95% CI: 1.33–3.86, P < 0.001), left ventricular ejection fraction (HR: 0.96, 95% CI: 0.94–0.99, P: 0.025), CHADS-VASC Score (HR: 1.16, 95% CI: 1.01–1.33, P: 0.034) and EHRA Score (HR: 1.98, 95% CI: 1.32–2.99, P: 0.001) were independent predictors of AF recurrence after cryoablation. Using a cut-off level of 532, the pre- ablation SII level predicted AF recurrence during follow up with a sensitivity of 71.4% and a specificity of 67.9%.

Conclusions: Our findings revealed that, in patients with paroxysmal AF undergoing cryoablation, increased pre-ablation SII levels were associated with a higher rate of AF recurrence. As a readily available, inexpensive, and easy to obtain marker of inflammation, pre-ablation SII level has well-predicted AF recurrence after cryoablation.

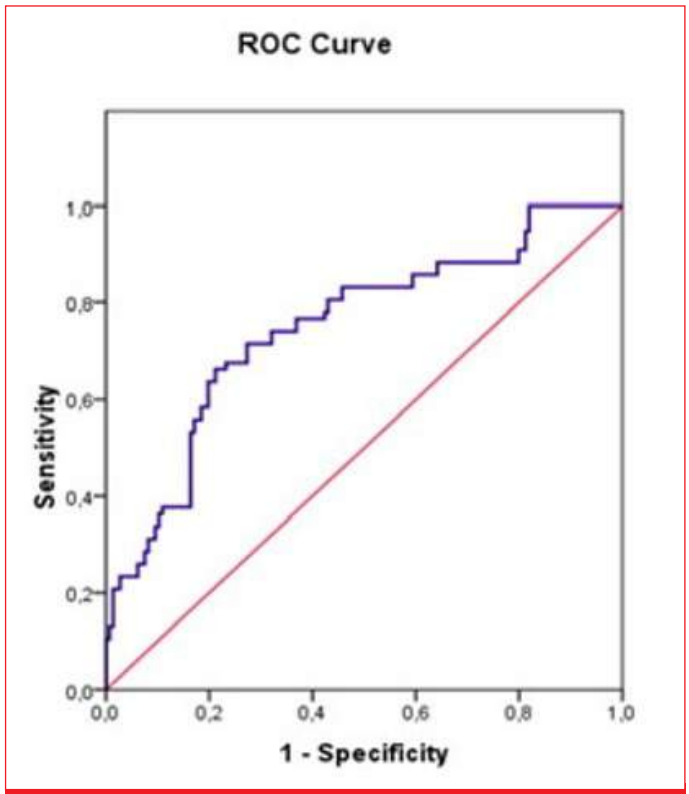

Figure 1. Receiver-operating characteristic curve of the pre-ablation level of systemic inflammation index for predicting AF recurrence in patients with paroxysmal AF undergoing catheter ablation

Table 1. Univariate and multivariate Cox proportional hazard modelling results of the AF recurrence after cryoballoon-based catheter ablation

Variable	Univariate analysis			Multivariate analysis		
	HR	95 % CI	P Value	Adjusted OR	95 % CI	p value
Age	1.018	0.999-1.038	0.070			
Gender (female)	0.690	0.438-1.087	0.108			
Hypertension	1.558	0.985-2.463	0.058			
Hyperlipidemia	0.366	0.087-1.534	0.169			
Smoking	0.988	0.424-2.299	0.977			
Diabetes mellitus	1.256	0.758-2.082	0.376			
Coronary artery disease	1.528	0.900-2.595	0.117			
OSAS	1.423	0.447-4.529	0.550			
CHADS Score	1.366	1.150-1.623	**0.000**			
CHADS VASC Score	1.320	1.987-4.226	**0.000**	1.163	1.011-1.337	0.034
EHRA Score	2.898	0.433-17.358	**0.000**	1.166	1.093-1.805	0.021
Echocardiographic parameters						
LVEF	0.949	0.928-0.970	**0.000**	0.968	0.941-0.996	0.025
LA Diameter	2.537	1.544-4.169	**0.000**	1.301	0.709-2.387	0.395
Labaratory parameters						
Hemoglobin	0.945	0.833-1.073	0.385			
WBC	1.089	0.984-1.205	0.099			
Neutrophil count	1.333	1.185-1.499	**0.000**			
Lymphocyte count	0.708	0.510-0.984	**0.040**			
Platelet count	1.003	0.999-1.006	0.163			
Creatinine	1.810	0.660-4.960	0.243			
Urea	1.005	0.991-1.021	0.474			
eGFR	0.988	0.976-1.001	0.077			
LDL	0.988	0.991-1.006	0.653			
HDL	0.997	0.975-1.020	0.827			
HsCRP	1.035	0.995-1.077	0.089			
SII > 532	3.334	2.027-5.483	**0.000**			
				2.270	1.334-3.86	0.003

PB-010 [Other]

Bioinformatic-based study on genes and micrornas predisposing Covid-19-related cardiovascular injuries

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Background and Aim: Apart from the direct and immediate invasion of coronavirus to vital tissues such as the heart, the virus is also capable of damaging these tissues based on the host's genetic susceptibility. The present bioinformatic-based study aimed to determine the genes and related microRNAs that most likely to be associated with susceptibility rates for virus-induced cardiovascular vulnerabilities.

Methods: A deep search was scheduled in databases including Pubmed (Medline), Google Scholar, Web of Science, and Scopus databases to assess all microRNAs and targeted genes related to cardiovascular defects induced by the coronavirus. The bioinformatic professional software systems were employed to assess gene-microRNAs interactions and mechanisms involved in cardiovascular injury.

Results: The coronavirus can induce cardiovascular defects by the three mechanisms of inducing cardiac fibrosis (by up-regulating miR-367-3p and down-regulating hsa-miR-5692a), inducing hypertension (by up-regulating miR-18b-5p), and inhibiting microvascular angiogenesis (by up-regulation of miR-18b-5p and down-regulating hsa-miR-5692a). Such processes can be triggered by the effects on NFAT5, CD69, and HGF expression.

Conclusions: Considering the central role of the revealed microRNAs and their targeted genes in cardiovascular injuries induced by coronavirus, such microRNAs can be applied for finding a way to stabilize the host against virus attacks as well as genetically based treatment for the affected host.

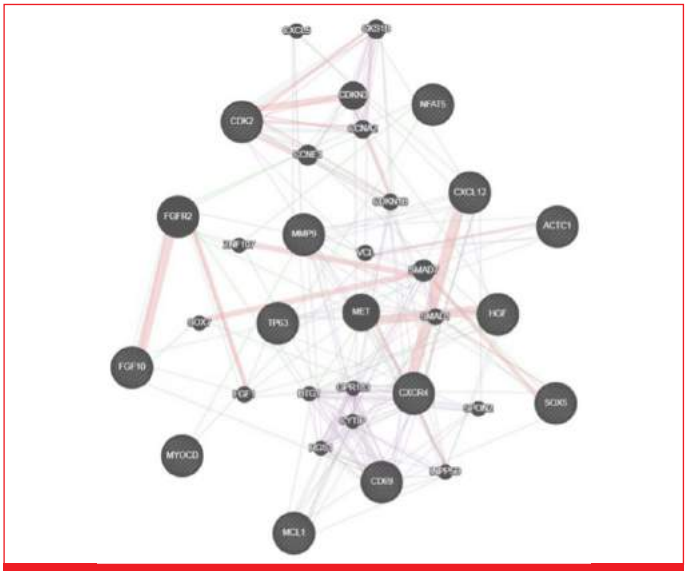

Figure 1. The pathways and gene to gene interactions involved in cardiovascular destruction by invasion of coronavirus COVID-19

Table 1. Reviewing the literatures on microRNAs and the target genes involved in heart injuries due to COVID-19

microRNA	Targeted gene	Regulation nature	Protective role
miR-18b-5p	NFAT5	Up-regulated	Decreasing endothelial nitric oxide synthase expression and inducing hypertension, anti-angiogenic effect
miR-193a-3p	MCL1	Up-regulated	Facilitating normal mitochondrial function, reducing the risk for dilated cardiomyopathy
miR-203a-5p	TP63	Up-regulated	Reducing the risk for Arrhythmogenic cardiomyopathy
miR-208b-5p	MYOCD	Up-regulated	A transcriptional co-activator of serum response factor (SRF) inducing cardiogenesis
miR-219a-5p	SOX5	Up-regulated	Reducing the risk for arrhythmia and ventricular hypertrophy
miR-367-3p	CD69 ACTC1	Down-regulated	Inducing post myocardial infarction cardiac fibrosis
miR-668-3p	CXCL12 CXCR4	Up-regulated	Anti-apoptotic effects and induces angiogenesis and inhibits fibrosis
miR-5582-5p	CDK2 FGF10	Up-regulated	Inducing Cdk2 signaling pathways, regulation of cardiac I/R injury pathway
miR-5692a	HGF MMP9	Up-regulated	Inducing microvascular angiogenesis

Table 2. Categories of gene-gene interactions related to heart injuries due to COVID-19 based on FDR

Type of Interaction	Percentage of Overall Interaction
Co-expression	56.88%
Physical interactions	41.14%
Pathway	1.57%
Genetic interactions	0.41%

Table 3.

Pathways	Genes	FDR	Coverage
Tissue morphogenesis	FGF10, HGF, ACTC1	3.57e-5	8/212
Morphogenesis of epithelium	FGF10, HGF	4.35e-4	5/67
Transforming growth factor receptor	CDK2	1.45e-3	3/10
Regulation of cell migration	FGF10, HGF, CXCL12	2.20e-3	6/199
Muscle tissue development	FGF10, MYOCD, ACTC1	6.89e-3	5/150
Cell chemotaxis	HGF, CXCL12, CXCR4	8.04e-3	5/157
Muscle organ development	FGF10, MYOCD, ACTC1	9.67e-3	5.172
Cardiac muscle development	FGF10, MYOCD, ACTC1	1.09e-2	4/85
Cardiac muscle tissue morphogenesis	FGF10, ACTC1	2.47e-2	3/38
Cellular response to ROS	HGF, CDK2	5.01e-2	3/58
Muscle cell differentiation	FGF10, MYOCD, ACTC1	5.01e-2	4/151
Growth factor activity	FGF10, HGF,	5.01e-2	3/54
Ventricular septum morphogenesis	FGF10	7.50e-2	2/14
Muscle contraction	MYOCD, ACTC1	7.89e-2	4/190
Heart development	FGF10, MYOCD, ACTC1	9.26e-2	4/203

The main pathways which activated in the background of gene cluster based on FDR values

PB-011 [Other]

Vitamin D supplementation improves SIRT1, Irisin, and glucose indices in overweight or obese type 2 diabetic patients: a doubleblind randomized placebo-controlled clinical trial

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Background and Aim: Vitamin D (VD) may increase sirtuin 1 (SIRT1) and subsequently PPAR-γ coactivator 1α (PGC-1α) and irisin levels and these improvements may reduce insulin resistance (IR). The aim was to assess the effects of vitamin D supplementation on SIRT1, irisin, and IR in overweight/obese type 2 diabetes (T2D) patients.

Methods: Ninety T2D males and females were recruited as a clinical trial study (mean of age and body mass index (BMI) of intervention and placebo groups were 50.05 ± 10.17 and 50.36 ± 10.2 yrs. and 31.37 ± 3.4 and 30.43 ± 3.2 kg/m2, respectively). The inclusion criteria were T2D, VD deficient, BMI > 25 kg/m2, and serum HbA1c<8.5%. The exclusion criteria were using vitamin and mineral supplements, having any acute disease, recent modifying dose or type of drugs. The supplementation was 50,000 IU/week VD or placebo for 8 weeks. The demographic characteristics, anthropometrics, dietary intakes and physical activity status, sun exposure status, fasting blood sugar (FBS) and insulin, glycosylated hemoglobin (HbA1c), irisin, SIRT1, 25-hydroxy D3 (25(OH)VD), homeostasis model assessment of insulin resistance (HOMA-IR), and quantitative insulin sensitivity check index (QUICKI) were determined. The significant P-value was ≤0.05.

Results: The increase of serum VD, SIRT1, and irisin in the intervention group was significant (p < 0.001). HbA1c was decreased significantly by 1%. The changes in the other glucose indices (FBS, insulin, and IR) were non-significant.

Conclusions: VD supplementation may improve T2D by decreasing HbA1c and increasing SIRT1 and irisin in VD deficient T2D patients. Further trials are suggested.

PB-012 [Other]

The relationship between cardio-ancle vascular index and atherogenic index in patients with diabetes mellitus

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Background and Aim: Atherogenic lipids play a role in various cellular functions such as decreased nitric oxide activity in endothelial cells and increased inflammation. This role may increase or accelerate atherosclerosis in diabetic patients. Cardio-ankle vascular index (CAVI) is accepted as an important indicator of atherosclerosis. The relationship between atherogenic index (AIP) calculated from atherogenic lipids and CAVI levels in patients with type 2 diabetes mellitus (T2DM) has not yet been demonstrated. This study aimed to investigate the relationship between AIP and CAVI in T2DM patients.

Methods: Fifty type 2 diabetic patients who came to the cardiology clinic for cardiovascular risk assessment were included in the study. CAVI measurements were made after 3 minutes of rest and a subclinical atherosclerosis was considered with (CAVI ≥8). The atherogenic index (AIP) was calculated as log (triglyceride/HDL).

Results: The mean AIP level was 0.5±0.2 and the mean CAVI level was 7.6±2.1 in T2DM patients. A positive correlation was found between AIP and CIMT levels according to CAVI levels (r = 0.385, p < 0.001). The mean AIP level was found to be higher in patients without subclinical atherosclerosis (0.89 ± 0.20 vs 0.62 ± 0.13, p < 0.001). Increased AIP values were found to be a co-independent predictor of the presence of subclinical atherosclerosis (OR = 5.46, p<0.001) and CAVI levels (± SE = 0.13 ± 0.04, p < 0.001). In the ROC curve analysis, the threshold value of the AIP index in predicting the presence of subclinical atherosclerosis was determined as 0.75 (sensitivity 74.8%, specificity 76.3%, AUC = 0.852; p < 0.001).

Conclusions: Increased API levels in T2DM patients were an independent predictor of increased CAVI levels and the presence of subclinical atherosclerosis. API, which can be easily evaluated using blood parameters, can be an important screening tool for the presence of atherosclerosis in T2DM patients.

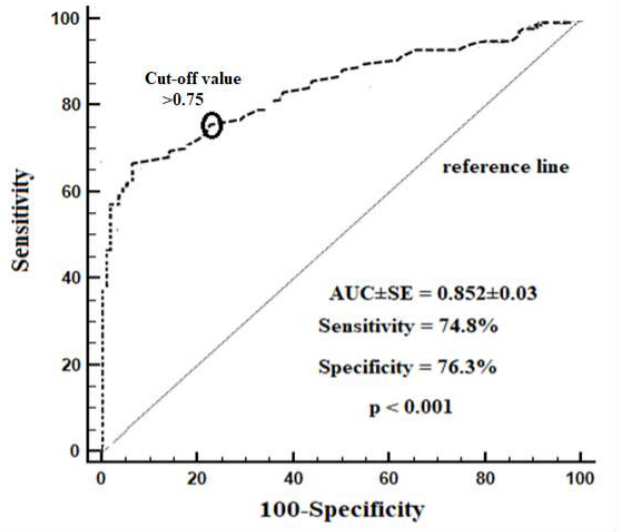

Figure 1. CAVI and API

PB-013 [Other]

Is there a relationship between the helical pattern of hypertrophy and atrial fibrillation in patients with hypertrophic cardiomyopathy?

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Background and Aim: Atrial fibrillation (AF) is commonly seen in patients with hypertrophic cardiomyopathy (HCM) and is associated with left atrial enlargement, fibrosis and diastolic dysfunction. Cardiac magnetic resonance (CMR) imaging provides important information about the geometric pattern of hypertrophy that may related to worse clinical outcomes such as arrhythmic events. The purpose of this study was to determine whether helical pattern is associated with increased risk for AF and diastolic dysfunction in patients with HKM.

Methods: A total of 50 consecutive patients with HCM referred for CMR imaging were enrolled into the study. The helical pattern was evaluated by measurement of the maximal left ventricle (LV) wall thickness (LVWT) for each of the 17 classical LV segments.

Results: A spiral pattern was observed in 20 patients (40%). We found significantly higher incidence of AF in patients who detected helical distribution than in those who did not (40% vs. 10%, p = 0.012). Left atrial volume index (LAVI) was also higher in patients with helical pattern compared to non-helical pattern (32.5 mL/m2 ± 16.9 vs. 23.7 mL/m2 ± 5.8; P < 0.001).

Conclusions: Our study suggests that presence of helical pattern is associated with significantly increased risk of development AF and LAVI in HCM patients.

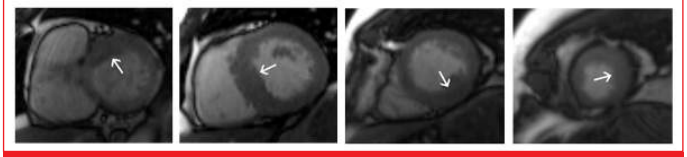

Figure 1.

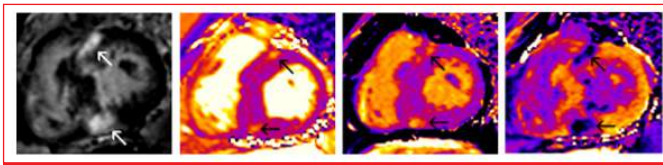

Figure 1. CAVI and API

PB-014 [Other]

Prognostic value of the C-reactive protein-to-albumin ratio in patients with infective endocarditis

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Background and Aim: The morbidity and mortality remain high in infective endocarditis (IE) due to its impact on cardiac functions and septic embolic complications. Systemic inflammation plays a critical role in the pathogenesis of IE. Recently, C-reactive protein-to-albumin ratio (CAR), a novel indicator of inflammatory response, has been shown to be an important marker for evaluating various inflammatory conditions. The relationship between the CAR and prognosis in patients with IE is not yet clear. Therefore, we aimed to investigate whether serum CAR values at hospital admission are associated with prognosis and mortality in patients with IE.

Methods: Patients diagnosed with definite IE according to the modified Duke criteria were collected from our tertiary hospital from 2016 to 2021 in the present study. Patients with severe chronic inflammatory or autoimmune disease, hematological disease, malignancy, severe renal failure, or chronic liver disease, patients receiving immunosuppressive therapy, and patients with missing laboratory data were excluded from the study. The study population was classified into 2 groups: patients with a primary clinical outcome (n=64) and those without (n=132). The primary clinical outcome consisted of the need for intensive care unit (ICU) treatment and in-hospital mortality. For all patients, the CAR was calculated as the ratio of serum C-reactive protein level to serum albumin level at hospital admission.

Results: In this retrospective study, a total of 196 patients were included (67% male, mean age 52.7 ± 14.9 years). During hospitalization, 53 (27%) patients died, and 34 (17.3%) patients were admitted to the ICU. Ultimately, 64 patients had the

primary composite endpoint. Acute renal failure occurred in 20.4% of patients, congestive heart failure in 8.7%, and septic embolism in 24.5% as a complication. Patients with a primary clinical outcome had significantly higher CAR values at admission compared with those without (40.4 [0.2–166.9] vs 17.9 [0.4–141.6], respectively; p <0.001) (Figure 1). In the Cox regression analysis, acute renal failure and CAR values were found to be independent predictors of mortality (HR=3.605, p=0.008; HR=1.015, p=0.02, respectively). In the ROC analysis, a cutoff value of > 20.24 for CAR values at admission predicted primary clinical outcome with 82.4% sensitivity and 70.3% specificity (AUC=0.838, 95% CI 0.734–0.941, p <0.001). According to the Kaplan-Meier survival analysis, the survival rate was found to be significantly lower in patients with acute renal failure than in those without (p=0.025; Figure 2).

Conclusions: Our study showed that higher CAR values were associated with poor prognosis and mortality in IE patients. As a result, it is of great importance to identify high-risk patients needing more aggressive treatment at the time of hospital admission. Serum admission CAR, as an easily obtainable biomarker, could be a useful predictor for prognostic implications in patients with IE.

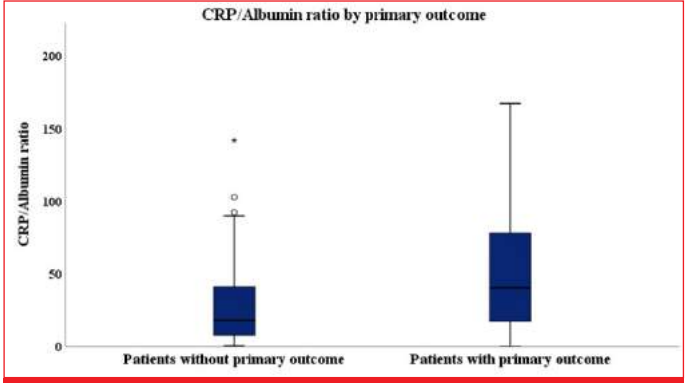

Figure 1. Serum C-reactive protein to albumin ratio values according to primary clinical outcome of the patients with infective endocarditis

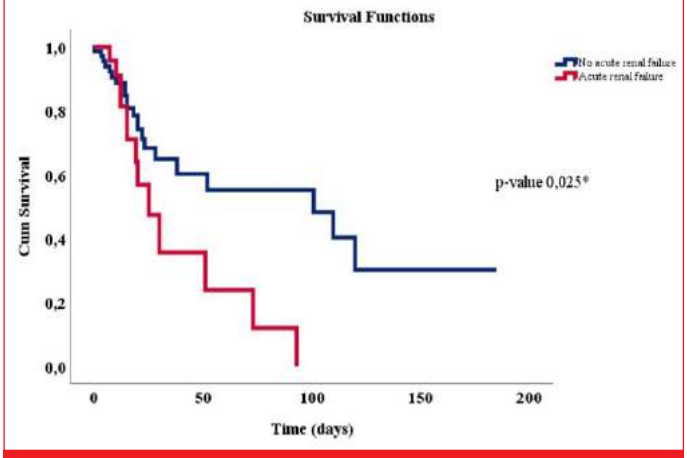

Figure 2. Kaplan-Meier survival analysis according to acute renal failure in patients with infective endocarditis

PB-015 [Other]

The effect of coronary angıography procedure and results on smoking cessation alone with doctor's advice

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Background and Aim: This study aimed to analysis of effect of coronary angiography procedure and results on smoking cessation alone with doctor's advice.

Methods: Our study is a prospective study and the active smoking patients who had coronary anjiyography between January in 2018 and January in 2021 350 patients included after the approval of ethics committee. The patients were divided into three groups as the patients without coronary artery disease (CAD), patients with significant CAD and medical treatment and patients with significant CAD and percutaneous coronary intervention (PCI) or coronary artery bypass greft (CABG). For the diagnosis of significant CAD, the presence of 50% or more stenosis in at least one of the epicardial coronary arteries was accepted. fagenstörm nicotine addiction score calculated every patients. After the patients were discharged, their smoking condition was reported by phoning them 180 days later.

Results: Six months later, the number of the patients who stopped smoking was only 21 (6%). At the evaluation made six months later 94% of the patients are observed to go on smoking. There was no statistical difference between the groups. Any statistical difference wasn't found gender and age in the 3 groups. No meaningful difference among 3 groups was evaluated patients cronic disease related to diabetes, hypertension vs. no significant difference was evaluated usage of package and year when they started smoking and the level of fagenstörm nicotine addiction.

Conclusions: It has been shown that even in individuals diagnosed with coronary artery disease after coronary anjiography, the doctor's advice does not have an effect on smoking.

addiction in the long term, and patients do not provide sufficient motivation and education to quit smoking. And doctors don't have enough attention this situation. This situation suggests that smoking cessation programs should be widespread in hospitals for long-term follow-up and treatment of smokers and that patients need multidisciplinary support treatments.

Table 1.

gender	male	102	80	76	2,13
	famale	38	24	30	
Marital status	maried	88	64	81	0,78
	single	52	40	24	
Education status	Secondary education	118	75	86	0,24
	High school	22	29	20	
Diabetes mellitus	existing	40	50	26	0,36
	absent	100	74	80	
hypertension	existing	50	55	55	0,45
	absent	90	69	71	
Smoking starting age		17,5	20,1	18,7	0,32
Package year		32	37	41	0,69
FAGENSTROM Ratio		5	5	4,5	0,29

CAD:CORONARY ARTERY DISEASE

Table 2.

	No serius CAD	Serius coronary artery disease and medical treatment	Serius coronary artery disease and interventrions	P
Keep continue smoking	132	100	97	0,68
Quit smoking	8	4	9	

PB-016 [Other]

The prognostic value of systemic immune inflammation index to predict in-hospital mortality in patients with infective endocarditis

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Background and Aim: Infective endocarditis (IE) is a life-threatening disease associated with significant mortality. Inflammation and inflammatory markers constitute the main pathophysiology of endocarditis process. The systemic immune inflammation index (SII), based on serum platelet, neutrophil, and lymphocyte concentration, is an inflammation-based score that has been shown to be a prognostic determinant in various populations. The aim of this study was to investigate the impact of SII on in –hospital mortality in patients with infective endocarditis (IE).

Methods: This was a population based retrospective cohort study. A total of 85 patients with IE were enrolled in this retrospective study. Data of IE hospital admissions in patients aged ≥18 years in a tertiary hospital during June 2020-April 2022 and in-hospital all-cause mortality data were retrospectively collected from hospital database system. Baseline serum platelet, neutrophil, and lymphocyte levels were used to calculate SII. The study population was categorized as log SII <3.29 low group, ≥3.29 high group according to SII. A prediction model was created for independent predictors of in-hospital mortality.

Results: A total 23 (27.4%) of endocarditis patients died during hospitalization. History of chronic heart failure, hypertension and prosthetic valve implantation, high CRP, wbc, neutrophil level and low e-GFR were observed more frequently in the high SII group. In-hospital mortality rate was also higher in the high SII group. In the univariate regression analysis, SII (OR, 6.339; 95% CI, 2.475-16.237, *P* <.001), vegetation size (OR, 0.122; 95% CI, 0.050-0.296), *P* <.001), CRP (OR, 1.005; 95% CI, 1.000-1.009, *P* =.033) and procalcitonin (OR, 1.054; 95% CI, 1.030-1.078, *P* <.001) were predictors of in-hospital mortality. Multivariate analysis also showed that SII (OR, 1.005; 95% CI, 1.167-7.833, *P* =.023), CRP (OR, 0.595; 95% CI, 0.995-1.008, *P* =.033), procalcitonine (OR, 1.046; 95% CI, 1.019-1.073, *P* =.001), vegetation size (OR, 0.122; 95% CI, 0.122-0.842, *P* =.021), history of prosthetic valve implantation (OR, 4.402; 95% CI, 1.622-11.945, *P* =.004) were independent predictors of in-hospital mortality for endocarditis patients. In the receiver operating characteristic curve, the optimal cutoff value of SII to predict in-hospital mortality was 3.29, with 73.9% sensitivity and 78.7% specificity (area under the curve: 0.772). A graph was revealed to show the SII values of the study population broken down by daily survive.

Conclusions: SII was a novel, available, easily measurable marker of inflammation seemed to be an independent predictor of in-hospital mortality in patients diagnosed with infective endocarditis.

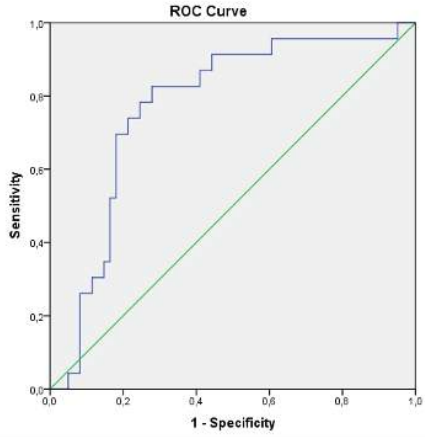

Figure 1. The ROC curve of SII for in-hospital mortality (AUC: 0.772, cut: 3.29, sensitivity: 73.9%, spesifity: 78.7%)

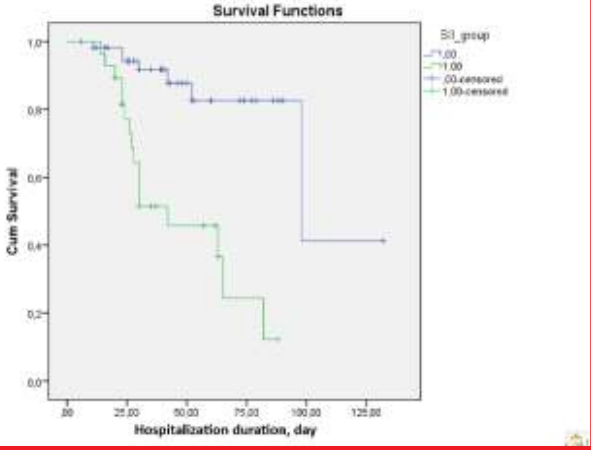

Figure 2. In-hospital survive according to SII

Table 1. Baseline characteristics of study population

Variable	Overall Group (n = 85)	SII Low Group (n = 57)	SII High Group (n = 28)	P
Age, years	56 (44.5-67.75)	55 (45-67)	58 (43-69)	.404
Male, n (%)	45 (52.9)	29 (50.9)	16 (57.1)	.296
DM-OAD, n (%)	16 (18.8)	11 (19.3)	5 (17.9)	.167
DM-Insulin, n (%)	20 (23.5)	14 (24.6)	6 (21.4)	
HT, n (%)	48 (56.5)	32 (56.1)	16 (57.1)	.008
HL, n (%)	24 (28.6)	17 (30.4)	7 (25.0)	.263
CAD, n (%)	28 (32.9)	17 (29.8)	11 (39.3)	.761
MI, n (%)	6 (7.1)	3 (5.3)	3 (10.7)	.850

Table 1. Baseline characteristics of study population (Continued)

Variable	Overall Group (n = 85)	SII Low Group (n = 57)	SII High Group (n = 28)	P
CHF, n (%)	9 (10.6)	6 (10.5)	3 (10.7)	<.001
CKD-Non-HD, n (%)	20 (23.5)	16 (28.1)	10 (17.5)	.121
CKD-HD, n (%)	20 (23.5)	4 (14.3)	10 (35.7)	
LVEF %, Median [IQR]	60 (55-60)	60 (55-60)	60 (55-60)	.666
Creatinine, mg/dl; Median [IQR]	1.13 (0.82-3.75)	1.08 (0.78-2.46)	2.45 (0.9-5.13)	.014
e-GFR, ml/min; Median [IQR]	61 (13.25-91)	66.5 (26.5-97.75)	37 (11.25-79.25)	.013
CRP, mg/dl; Median [IQR]	89.1 (41.2-156.4)	69.2 (28.95-101.35)	149.7 (93-188.9)	<.001
Glucose, mg/dL; Median [IQR]	104 (89-130)	112 (98.5-168.5)	120.5 (105.2-174.2)	.237
WBC,103/dL; Median [IQR]	10.81 (7.64-14.98)	9.49 (6.98-12.48)	14.89 (12.37-15.82)	<.001
Hemoglobin, mg/dL; Median [IQR]	9.8 (8.45-11.55)	9.7 (8.2-11.55)	10.45 (8.62-11.87)	.256
Platelet count,103/dL; Median [IQR]	201 (152.5-278.5)	193 (140-257)	224.5 (166.75-286)	.106
Lymphocyte, cells/μL, Median [IQR]	1.23 (0.81-1.83)	1.51 (1.05-2.04)	0.81 (0.47-1.23)	<.001
Monocytes, cells/μL; Median [IQR]	0.81 (0.55-1.25)	0.77 (0.5-1.2)	0.99 (0.62-1.62)	.233
Neutrophils, cells/μL; Median [IQR]	10.81 (7.64-14.98)	6.56 (4.92-9.79)	12.47 (10.34-14.37)	<.001
Albumin, g/dL; Median [IQR]	32 (29-38)	33 (29-38)	32 (26.5-35.75)	.286
Vegetation size <10 mm	62 (72.9)	39 (68.4)	23 (82.1)	.181
Vegetation size >10 mm	23 (27.1)	18 (31.6)	5 (17.9)	
Native valve, n (%)	59 (64.9)	40 (70.2)	19 (67.9)	.048
Prosthetic valve, n (%)	26 (30.6)	17 (29.8)	9 (32.1)	
In-hospital mortality,n (%)	23 (27.4)	7 (12.5)	16 (57.1)	<.001

P <.05 was considered statistical significance.
CHF, congestive heart failure; CKD, chronic kidney disease; DM, diabetes mellitus; e-GFR, estimated glomerular filtration rate; HD, hemodialysis; HT, hypertension; HL, hyperlipidemia; IQR, interquartile range; LVEF, left ventricular ejection fraction; OAD, oral anti-diabetic; WBC, white blood cell.

Table 2. Univariate predictors of total in-hospital mortality

	Univariate Analysis	
	OR (95% CI)	P
Vegetation size, mm	0.122 (0.050-0.296)	<.001
Age, year	1.011 (0.986-1.037)	.399
Gender, male	0.831 (0.365-1.892)	.658
Diabetes mellitus	1.055 (0.649-1.713)	.830
Hypertension	0.563 (0.240-1.317)	.185
SII	6.339 (2.475-16.237)	<.001
Sedimentation, mm/h	1.005 (0.897-1.022)	.590
CRP, mg/dL	1.005 (1.000-1.009)	.033
Procalcitonin, ng/mL	1.054 (1.030-1.078)	<.001
WBC, 103/dL	1.011 (0.079 -1.044)	.510
HGB, mg/dL	0.868 (0.714-1.055)	.155

CRP, body mass index; CI, confidence interval; HGB,hrmoglobine; WBC,White blood cell; OR, odds; SII, systemic immune inflammation index

Table 3. Multivariate predictors of total in-hospital mortality

	Multivariate analysis	
	OR (95% CI)	P
Vegetation size, mm	0.122 (0.122-0.842)	.021
Prosthetic valve	4.402 (1.622-11.945)	.004
SII	1.005 (1.167-7.883)	.023
CRP, mg/dL	0.595 (0.995-1.008)	.033
Procalcitonin, ng/mL	1.046 (1.019-1.073)	.001

CRP, body mass index; CI, confidence interval; OR, odds; SII, systemic immune inflammation index.

PB-017 [Other]

The effect of sleep quality and anxiety level on blood pressure variability

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[1]*Burdur State Hospital Cardiology Clinic, Burdur*
[2]*Department of Cardiology, Gülhane Research and Education Hospital, Ankara*

Background and Aim: We aimed to investigate the effects of sleep quality and level of anxiety on the cardiovascular system with blood pressure variability (BPV) based on ambulatory blood pressure holter data.

Methods: A total of 88 patients underwent ambulatory blood pressure monitoring were included in the study. BPV was evaluated by using standard deviation (SD) of 24-hour, day, and night systolic, diastolic blood pressure values. Coefficient variation of blood pressure (CV) was calculated with the formula of (standard deviation of blood pressure/mean arterial pressures) X 100%. The Pittsburgh Sleep Quality Index (PSQI) was used to evaluate the sleep quality of individuals and the Beck Anxiety Inventory (BAI) was used to measure the frequency and severity of anxiety symptoms.

Results: Total sleep time was decreased in poor sleep quality group, as expected. There was a statistically significant and positive relationship between the PSQI scores and BAI scores

of patients who underwent ambulatory blood pressure monitoring. According to the PSQI scores, there was no statistically significant difference between the groups in terms of blood pressure values, but there was a difference in terms of the parameters indicating BPV, especially in terms of SD of SBP and CV of SBP. Similarly, a statistically significant increase in SD of SBP and CV of SBP values were observed with increasing anxiety level. When we divided the patients into four groups according to PSQI and BAI scores; SD of SBP and CV of SBP values were statistically significantly differed between groups, and we observed that this difference was more evident between patients with PSQI ≤5 and BAI ≤16 and patients with PSQI >6 and BAI >16.

Conclusion: Blood pressure variability parameters are adversely affected with increasing levels of anxiety and poor sleep quality.

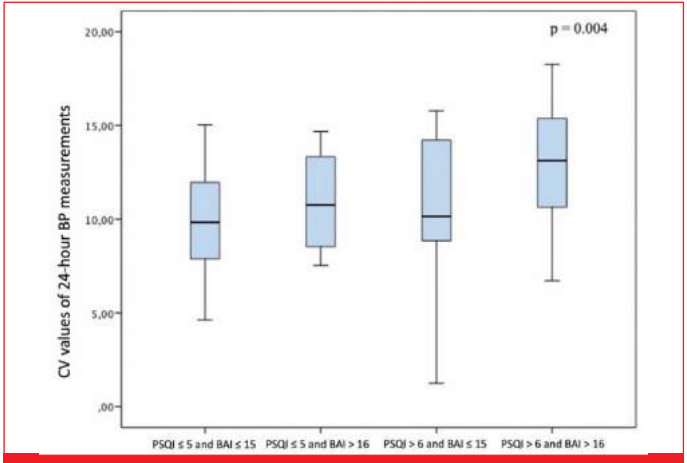

Figure 1. 24-hour SBP CV values according to sleep quality and anxiety levels

PB-018 [Interventional Cardiology / Carotid and Peripheral Vascular

Outcomes of TAVI procedure in North Cyprus

Alptekin Özkoç[1], Cenk Conkbayır[2]

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Background and Aim: Complication rates may not always match up with real life in transcatheter aortic valve implantation (TAVI). Due to this reason, small volume centers should compare their findings with those of other randomized studies. In this study we aimed to investigate mortality and complication rates of TAVI in a small island country.

Methods: This study analyzed 94 TAVI procedures, which were performed at a state hospital in Northern Cyprus between 2016 and 2021, and calculated mortality and morbidity rates from admission to hospital discharge.

Results: 63.8% of the patients, who underwent TAVI, were female, mean age was 78.64 years and mean aortic gradient was 51.6 mmHg. NYHA Class IV heart failure was evident in 10 patients. 29.8% of the patients had percutaneous coronary intervention (PCI) and 8.5% had coronary bypass histories. 8.5% of the patients had pacemaker or ICD. 15.9% of the patients had left and 4.25% had right bundle branch block. Eighty nine of the 94 TAVI procedures were implanted in the right femoral vein, 4 were implanted in the left femoral vein and 1 was implanted in the axillary vein. 3 of the transfemoral TAVI procedures were closed by surgery and 91 were closed percutaneously (ProGlide). Self-expandable valve was used in 58.52% of the patients whereas balloon expandable valve was used in the remaining 41.48%. One TAVI procedure was valve-in-vale implementation. Mean EuroSCORE 2 score was 14.17. Four patients died during the procedure. 3 deaths occurred due to coronary obstruction whereas one death occurred due to valve dislocation. Perforation due to temporary pacemaker lead in the right ventricle and consequent pericardial tamponade developed in 6 patients and 2 patients died due to this reason. 1 patient died due to acute renal failure and related metabolic causes. Peripheral vascular complications were the most frequent complications with a percentage of 20.2%. The 68.4% of these complications were treated percutaneously and 31.6% were treated surgically. Mild cerebrovascular accident occurred in 1 patient. Pacemaker was implanted in 9 (9.6%) patients due to advanced heart block that developed after TAVI. Pacemaker implantation percentages in self-expandable and balloon-expandable TAVI patients were 10.9% and 7.7%, respectively.

Conclusions: In our study 85.1% of patients were high risk patient. Vascular complications with a percentage of 20.21% were the most frequent complications observed in TAVI patients. Heart blocks that require pacemaker implantation were the second most frequent complications. Besides, parallel to the literature, pacemaker demand was lower in patients with balloon expandable valves. Perforation due to temporary pacemaker lead in the right ventricle and consequent pericardial tamponade was another complication. Mortality and morbidity rates in our case was similar to the literature. Findings of this study may be used to develop strategies to reduce mortality and morbidity rates in TAVI procedures in relatively low volume loaded hospitals.

Table 1. Demographic characteristics and medical histories

Patients underwent TAVI	94	
Mean age (years)	76.8	
Comorbidities	No	Yes
Coronary artery disease	61.7%	29.8% (PCI) 8.5% (CABG)
Diabetes Mellitus Type I and II	26.6%	73.4%
Hypertension	13.9%	86.1%
Heart failure	NYHA 3 89.3%	NYHA 4 10.7%
Mean LVEF	56.2%	
P mean	51.6	
Mean EuroSCORE II	14.17	
Comorbid valve disease	Severe mitral stenosis	Severe mitral insufficiency
	1.05%	8.5%

Table 2. Procedural information

Aortic valve type	Balloon expandable %41.5(39)	Self-expandable %58.5 (55)
Access site	Femoral right %94.7 (89) left %4.25	axillary %1.05
TAVI valve-in-valve	%2.12 (2)	
Rapid ventricular pacing	Temporary PM %91.5	Permanent PM/ICD %8.5
Vascular closure method	Surgical %3.2	Percutaneous (ProGlide) %96.8

Table 3. Complications and Mortality Rates	
In-Hospital mortality	5.31% (5)
Coronary obstruction	2.1% (2)
Valve dislocation	1.05% (1)
Pericardial tamponade	6.4% (6)
Peripheral complications	20.2% (19)
Cerebrovascular accident	1.05% (1)
Permanent Pacemaker implantation	9.6% (9) -SE % 11, BE 7.7%

PB-019 [Interventional Cardiology / Carotid and Peripheral Vascular]

Outcomes of percutaneous PFO closure: Single centre experience

Cem Çöteli, Ahmet Yetkin, Yusuf Ziya Şener, Uğur Nadir Karakulak, Levent Şahiner, Barış Kaya, Ahmet Hakan Ateş, Hikmet Yorgun, Kudret Aytemir

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Background and Aim: Patent foramen ovale closure aims to reduce recurrent stroke risk in a specific group of patients. On the other hand, absolute risk reduction with PFO closure compared to antiplatelet treatment is still under investigation due to procedural complication risks.

Methods: The objective of this trial is to report short and long-term results of PFO closure in our center.

Results: In our study group (mean age 43.7, SD: 11.12), 217 (58.8%) of them were female, the most common comorbidity was hypertension (in 87 pts, 23.6%), atrial fibrillation was present in 9 patients (2.4%), and 46 pts (12.5%) had migraine. The median rope score of patients was 6 (2-9). The percutaneous PFO closure was performed without complications in 352 patients (95.4%). Pericardial effusion, access-related bleeding, dislocation, and supraventricular arrhythmia were observed in 5, 9.1, and 2 patients, respectively. Pericardiocentesis, urgent surgery, and blood transfusion were not needed in any patient. The device migrated after releasing in one patient and was collected with a snare catheter. The transseptal atrial septal puncture was required in 15 (4.1%) patients. 327 patients (88.6%) used DAPT, and 42 patients (11.4%) used SAPT (ASA or clopidogrel) or oral anticoagulant after procedure. The median follow-up duration was 12.7 months (3-147 months). Mortality was not observed in the study group, but 18 patients (5.7%) presented with recurrent stroke during follow-up. Clinically disabling stroke was present in 3 patients (0.81%). Device malposition or thrombus formation on the closure device were not observed at the third-month transthoracic echocardiography.

Conclusions: PFO closure is a safe, feasible, and effective therapeutic option for thromboembolic risk reduction in selected patients. Procedure-related complication risk and postprocedural recurrent stroke are low. Further randomized controlled trials and cost-effectivity analysis are needed to understand the absolute outcomes of the procedure.

Table 1. Baseline characteristics	
Age (years)	43.7 ± 11.12
Female	217 (58.8%)
Hypertension	87 (23.6%)
T2DM	30 (8.1%)
CAD	13 (3.5%)
AF	9 (2.4%)
Migraine	46 (12.5%)
LVEF	63.8±4.15%
ROPE Score	6 (2-9)
Follow-up Duration (Month)	12.7 (3-147)

T2DM, type 2 diabetes mellitus; CAD, coronary artery disease; AF, atrial fibrillation; LVEF, left ventricular ejection fraction; ROPE Score, risk of paradoxical embolism score

Table 2. Procedural and follow-up outcomes	
DAPT	327 (88.6%)
SAPT	33 (8.9%)
OAC	9 (2.4%)
Device Size	
• 18 mm	34 (9.2%)
• 25 mm	283 (76.7%)
• 28 mm	18 (4.9%)
• 30 mm	34 (9.2%)
Procedural Success	369 (100%)
Transseptal Puncture	15 (4.1%)
Complication	17 (4.6%)
Pericardial effusion	5 (1.4%)
Access-related bleeding	9 (2.4%)
Supraventricular arrhythmia	2 (0.5%)
Dislocation	1 (0.3%)
Mortality	0 (0%)
Recurrent Stroke	3 (0.8%)

DAPT, dual anti-platelet; SAPT, single anti-platelet; OAC, oral anticoagulant

PB-020 [Interventional Cardiology / Carotid and Peripheral Vascular]

Percutaneous stenting treatment in symptomatic carotid artery disease: A single-center experience

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Background and Aim: Carotid endarterectomy has been included in the guidelines as the main treatment modality in severe symptomatic carotid artery disease. Carotid artery stenting (CAS) is the alternative treatment option in patients with high morbidity and high comorbidity. The aim of this study was to present demographic data and short-term clinical outcomes of symptomatic severe carotid artery patients treated with CAS who were admitted to tertiary invasive cardiology clinic.

Methods: This single-center, retrospective, observational study included 72 symptomatic patients who underwent CAS procedures between 2010-2019. Clinical features and procedure details of the patients were obtained from the hospital information system. Mortality and morbidity data after discharge were obtained from hospital registration system and telephone visits.

Results: 72 patients who met the inclusion criteria were included in the study. 26% of the patients were female gender and the mean age was 67.6 ± 8.6 years. 50.7% of the patients had coronary artey disease. CAS was performed on the left carotid artery of 31 patients and on the right carotid artery of 41 patients, and there was no CAS procedure on bilateral internal carotid artery. 71.2% of patients were used with distal filter devices and 28.8% of them were treated with MOMA. Mortality occurred in 8 patients in the median 3-year follow-up. Female gender, coronary artery disease, low-density lipoprotein levels were statistically higher in patients with mortality, while hemoglobin values were significantly lower.

Conclusions: CAS is a successful and safe treatment method in the prevention of stroke when performed in experienced centers. The medium-term clinical results of CAS are very satisfactory.

Table 1.

Age, year Mean ± SS	67.55 ± 8.6
Female gender, n (%)	18 (25)
DM, n (%)	32 (43.8)
HT, n (%)	44 (61.6)
HL, n (%)	33 (46)
ASKH, n (%)	36 (50)
Presence of coronary artery bypass graft, %	18 (25)
Creatinine, mg/dL Median (IQR)	0.95 (0.32)
Hemoglobin g/dL Average ± SS	13.22 ± 2.06
Leukocyte Median (IAA)	8.18 (2.91)
Platelets, cell/mL Mean ± SS	265.1 ± 78.30
MPV, fL Mean ± SS	9.06 ± 1.85
LDL, mg/dL Median (IQR)	117.0 (59.0)

Clinical characteristics of patients at the time of admission and laboratory data

Table 2.

Degree of stenosis, % Median (IQR)	80 (10)
Lesion side (left/right), %	42.5 / 57.5
Predilation, n (%)	19 (26)
Postdilatation, n (%)	71 (97.3)
Using a Distal Filter Device (Spider), %	71.2
Using a MOMA Device, %	28.8
Periprocedural Complication, %,	0

Features related to carotid artery stenting

Table 3.

	Living (n = 64)	Mortality (n = 8)	P
Age, year Mean ± SS	68.08 ± 8.43	63.25 ± 9.31	.135
Female Gender (%)	21.5%	62.5%	.013
Diabetes (%)	47.7%	12.5%	.58
Hypertension (%)	61.5%	62.5%	.99
Hyperlipidemia (%)	43.1%	75%	.88
Coronary Artery disease (%)	44.6%	100%	.003
Predilated (%)	26.2%	25%	.94
Postdilated (%)	96.9%	100%	.61
Proximal protection device (%)	29.2%	25%	.80
Creatinin Median (IQR)	0.95 (0.31)	0.96 (0.83)	.49
Hemoglobin Mean ± SD	13.44 ± 2.04	11.41 ± 1.15	.007
WBC Mean ± SD	8.48 ± 3.16	9.70 ± 3.09	.31
Platelet count Mean ± SD	258.15 ± 77.66	321.50 ± 61.97	.03
MPV Mean ± SD	9.02 ± 1.64	9.30 ± 3.24	.69
LDL Mean ± SD	124.23 ± 35.80	167.0 ± 40.16	.005
Degree of stenosis (%)	83.9 ± 0.09	81.8 ± 0.12	.54

Comparative data of patients who developed mortality at follow-up

PB-021 [Interventional Cardiology / Coronary]

Heart rate variability can be a predictor for Mace in no-reflow phenomenon patients with non-stemi

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Background and Aim: It is already known that major adverse events (MACE) are more common in patients who develop the no-reflow phenomenon in NSTEMI. However, it is still a

subject of research to predict which patient groups will be more at risk of developing MACE. The current study aims to investigate the relationship between Heart Rate Variability (HRV) and MACE in no-reflow phenomenon patients.

Methods: In this study, 396 patients who had non-stemi were examined between March 2019 to January 2021. 28 patients who developed an angiographically no-reflow phenomenon (TIMI 0-I flow after PCI) were included in this study. A 24-hour rhythm holter and Beck Depression Inventory (BDI) tests were recorded for the patients during their 1st-month follow-up. SDNN timing was used for HRV determination. Curve-fitting and Cox proportional-hazard regression models with a hazard ratio (HR) and 95% confidence interval (CI) were used. Patients were divided into two groups [the high-HRV group (n = 18) and the low-HRV group (n = 10)] based on the SDNN threshold value of 90.36 ms identified through curve fitting.

Results: During a mean follow-up of 258.64 ± 38.27 days, 4 (28.57%) patients died from all causes in the no-reflow subset, 3 (30%) patients died in the low HRV group, and 1 (5.55%) patient died in the high HRV group (*P* =.031). Two (20%) patients had a stroke in the Low-HRV group and 1 (5.55%) patient had a stroke in the high-HRV group (*P* =.001). BDI results were found higher in the no-reflow group (*P* =.023). Three (30%) patients had re-infarction in the Low-HRV group and 2 (11.11%) patients had a stroke in the high-HRV group (*P* =.0024). Univariate analysis showed that the risk of MACE in the Low-HRV group was significantly greater than that in the High-HRV group (HR = 1.35, 95% CI: 1.05 to 2.16).

Conclusion: Our study demonstrated that HRV reduction (>90.36 msec) is associated with MACE in no-reflow patients.

PB-022 [Interventional Cardiology / Coronary]

The effect of kissing on mid-term clinical outcome in patients with bifurcation lesions who were implanted bioresorbable stent with provisional approach

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Background and Aim: In bifurcation lesions, when a drug-eluting stent is implanted with provisional approach, there is no difference in clinical outcome between the groups with and without side-branch kissing. However, there is no study investigating the effect of kissing on the side branch in bifurcation lesions in which a bioresorbable stent is implanted.

Methods: In our study, we investigated whether kissing balloon effected mid-term clinical outcome in bifurcation lesions in which bioresorbable stent (Absorb, Abbott) was implanted. The demographic and clinical data and as well as mid-term follow-up of 62 patients who were implanted Absorb stent with provisional approach in the bifurcation lesion were compared according to presence or absence of kissing procedure.

Results: Of the study population, mean age was 57.1 ± 10.0, majority of them were male (84.5%), 57.9% had hypertension and 30.8% had diabetes. There was no difference in the demographic data of 35 patients who were in the kissing and 27 patients who were in the non-kissing group, except non-kissing group was significantly older (*P* =.027). During the mean follow-up of 72.1 ± 21.2 months, there were no difference between the kissing and non-kissing groups in terms of in-stent restenosis (*P* =.691), target lesion revascularization requirement (*P* =.087), target vessel revascularization requirement (*P* =.614), stent thrombosis (*P* =.912), myocardial infarction (*P* =.892) and overall survival (log rank *P* =.601).

Conclusions: There were no difference in terms of survival and other clinical outcomes between the kissing and non-kissing groups in a long follow-up period of approximately 6.5 years, who were implanted Absorb stent with a provisional approach. Our results demonstrate that kissing procedure does not provide additional clinical benefit in the provisional approach of bifurcation lesions. There findings are in line with the lack benefit of kissing stratgey in drug-eluting stents.

Table 1. Characteristics of the study population

	provisional n=27	kissing n=35	p value
HT	18 (66,67%)	15 (42,86%)	0,077
DM	9 (33,33%)	14 (40%)	0,609
Family History	1 (3,7%)	3 (8,57%)	0,626
Smoking	5 (18,52%)	4 (11,43%)	0,485
Hyperlipidemia	5 (18,52%)	8 (22,86%)	0,76
CABG history	1 (3,7%)	0 (0%)	0,435
PCI history	7 (25,93%)	8 (22,86%)	1
MI history	3 (11,11%)	1 (2,86%)	0,309
Access			0,114
Femoral	17 (62,96%)	15 (42,86%)	
Radial	9 (33,33%)	20 (57,14%)	
Other	1 (3,7%)	0 (0%)	
Clinic			0,877
SAP	14 (51,85%)	17 (48,57%)	
UAP	10 (37,04%)	15 (42,86%)	
NSTEMI	3 (11,11%)	3 (8,57%)	
STEMI	0 (0%)	0 (0%)	
Age	59,56 ± 9,49	54,09 ± 9,33	0,027
Survival, day	2298,19 ± 805,5	2336,91 ± 496,5	0,495
Indication			0,537
MI	1 (3,7 %)	1 (2,86 %)	
CTO	5 (18,52 %)	11 (31,43 %)	
Elective PCI	21 (77,78 %)	23 (65,71 %)	

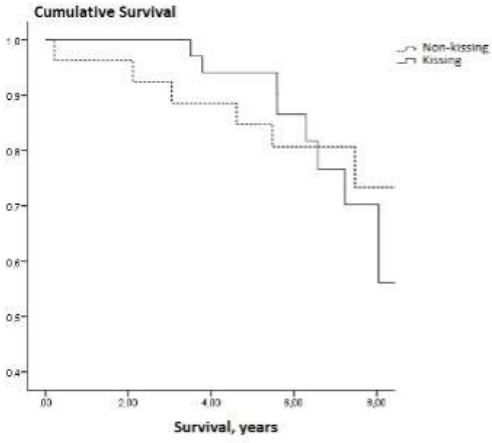

Figure 1. Cumulative survival

PB-023 [Coronary]

The relationship between triglyceride glucose index and multivessel lesion in patients presenting with acute coronary syndrome

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Background and Aim: Triglyceride-glucose index (TGI) is a new parameter that has been used to determine insulin resistance. There are studies reporting that it can also be used to determine cardiovascular risk. Several recent studies have demonstrated the relationship between TGI and vascular disease; however, the role of TGI with multivessel disease has not been extensively evaluated. In this study, we aimed to reveal the predictive value of TGI in patients presenting with acute coronary syndrome (ACS) and diagnosed with multiple vessel lesions.

Methods: We designed our study retrospectively and cross-sectionally. We analyzed 142 patients who applied to our clinic with the diagnosis of ACS and underwent coronary angiography for the first time between January 2018 and January 2020. Two groups were formed as patients with 70% or more stenosis in two or more vessels in their coronary arteries (multi-vessel lesion, n=64) and patients with single vessel lesion or non-critical lesion (n=78). TGI was calculated using the formula fasting triglyceride (mg/dL) x fasting glucose (mg/dL) / 2.

Results: The age ratio of the patients included in the study was significantly higher in the group with multi-vessel disease (60.3 ± 7.9 vs. 65.1 ± 8.3, p<0.001). The most common comorbid diseases detected in the patients were diabetes mellitus (47.2%), hypertension (49.3%) and dyslipidemia (46.5%). TGI was significantly higher in the group with multiple vessel lesions (9.4±0.4 vs. 9.1±0.4 p<0.001). While age, BMI, hypertension, creatinine, uric acid and TGI were found to be significant in the univariable analysis, in the multivariable regression analysis; We found that age (OR:1.06; 95%CI:1.01-1.12, P=0.008), TGI (OR: 4.39; 95%CI:1.54-12.48, p=0.006) parameters were independent predictors of multivessel lesion. In the ROC analysis; TGI predicted multivessel lesion with 64.1% sensitivity and 66.7% specificity (AUC: 0.694; 95%CI: 0.607-0.780, p<0.001).

Conclusions: In recent years, besides inflammation parameters, the effect of some newly defined index parameters and scoring systems in predicting cardiovascular diseases has been investigated. In this respect, we think that TGI can be used as a simple, inexpensive, and rapid test to predict multivessel lesion. In order to generalize the results of the study, it should be supported by multicenter studies with large participation.

Table 1. Demographics and clinical findings

	Overall n: 142	Multi-vessel disease n: 64	Single-vessel disease n:78	p
Age	62.5±8.4	65.1 ± 8.3	60.3 ±7.9	<0.001
Gender, male, n(%)	84 (59.2)	36 (56.3)	48 (61.5)	0.524
BMI, kg/ m^2	26.0±2.2	26.5±2.4	25.6±2.0	0.023
Diabetes mellitus, n(%)	67 (47.2)	39 (60.9)	28 (35.9)	0.003
Hypertension, n(%)	70 (49.3)	40 (62.5)	30 (38.5)	0.004
Smoking, n(%)	69 (48.6)	28 (43.8)	41 (52.6)	0.296
Dyslipidemia, n(%)	66 (46.5)	36 (56.3)	30 (38.5)	0.034
Family history, n(%)	45 (31.7)	18 (28.1)	27 (34.6)	0.408
LVEF, %	53.5±7.9	52.2 ±8.3	54.6 ±7.6	0.074
Hemoglobin, g/dl	12.3±1.9	12.6 ±2.0	12.1 ±1.8	0.177
Leukocytes,10^3/ uL	9.8±1.9	9.9±1.8	9.6±2.1	0.347
Fasting glucose, mg/dl	143.2±46.5	158.8 ±51.3	130.4 ±38.0	<0.001
Creatinine, mg/ dl, IQR	0.86 (0.3)	0.90 (0.3)	0.81(0.3)	0.090
Total cholesterol, mg/dl	187.9±37.5	193.0±39.3	183.7±35.6	0.168
Triglyceride, mg/dl	160.3±46.0	167.1 ±43.1	154.8 ±47.8	0.113
LDL, mg/dl	116.4±32.6	120.9 ±35.0	112.7 ±30.1	0.135
HDL, mg/dl	39.2±8.3	38.3 ±7.1	40.0 ±9.1	0.220
Uric acid	5.5±1.5	5.8 ±1.5	5.2 ±1.4	0.035
TGI	9.2±0.4	9.4±0.4	9.1±0.4	<0.001

BMI, Body mass index; LV EF, Left ventricular ejection fraction; TGI, Triglyceride glucose index

Table 2. Relationship between multivessel disease and clinical variables

	Univariable regression analysis	p value	Multivariable regression analysis	p value
Age	1.07 (1.03-1.12)	0.001	1.06 (1.01-1.12)	0.008
Gender,male	1.24 (0.63-.243)	0.524		
BMI, kg/m^2	1.20 (1.02-1.41)	0.028	1.14 (0.96-1.36)	0.117
Hypertension	2.66 (1.34-5.27)	0.005	1.41 (0.63-3.13)	0.395
Smoking	0.70 (0.36-1.36)	0.296		
LVEF	0.96(0.92-1.0)	0.077		
Hemoglobin	1.13(0.94-1.34)	0.177		
Creatinine	2.41(0.89-6.51)	0.082		
Uric acid	1.27(1.01-1.61)	0.038	1.17(0.91-1.52)	0.214
TGI	5.11(2.08-12.56)	<0.001	4.39(1.54-12.48)	0.006

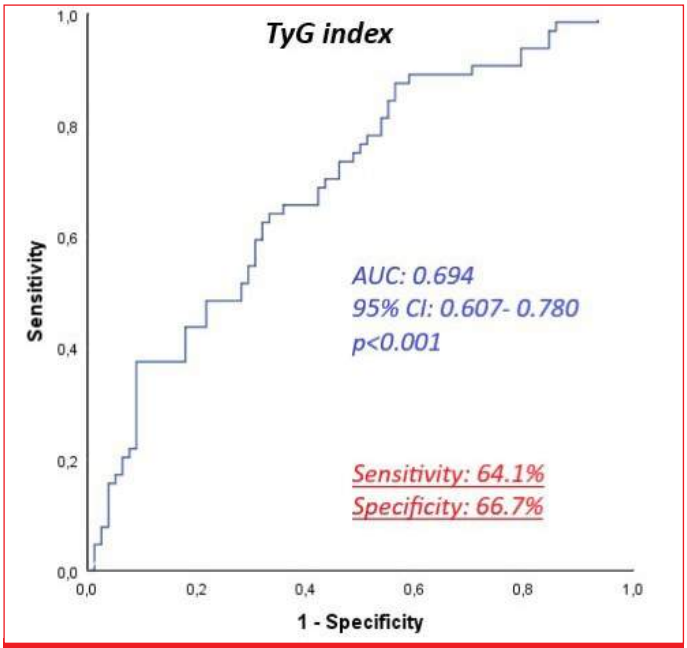

Figure 1. ROC analysis showing the relationship between multivessel disease and triglyceride-glucose index

PB-024 [Hypertension]

Effect of basal autonomic function on dipping pattern

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Background and Aim: Blood pressure changes diurnal during the day and these changes have prognostic significance in cardiovascular events. This change is regulated by neurohormonal mechanisms. Dipping pattern (DP) is defined as the expected decrease in systolic blood pressure in nighttime measurements compared to daytime measurements. The absence of this change is defined as the non-dipping pattern (NDP). Heart rate variability (HRV) indicates fluctuation in heart rate and is based on neurocardiac interaction. It tests the effect of sympathetic and vagal tone on heart rate and pulse variability. It is known that sympathetic and vagal tone are also effective in blood pressure regulation, but research comparing the relationship between HRV parameters and dipping status is limited. Our aim is to determine the effect of basal autonomic activity on dipping status with HRV analysis.

Methods: Patients over the age of 18 who underwent ambulatory blood pressure monitoring (ABPM) and 24-hour Holter ECG at the same admission between February 2017 and September 2022 were evaluated retrospectively. The percentage of systolic blood pressure (BP) reduction in nighttime measurements was calculated from the ABPM recordings relative to the patients' daytime measurements. A decrease of ≥10% was defined as DP, and a decrease of less than 10% was defined as NDP. HRV analysis was performed using appropriate software from 24-h Holter ECG recordings. Demographic and laboratory data were compared between DP and NDP groups. Parameters that could be related to the dipping percentage were evaluated in the correlation analysis. Linear logistic regression analysis was performed to determine independent predictors of systolic dipping percent.

Results: In our study, 129 patients [mean age 57.2±18.2, male 63 (48.8%)) were evaluated. Among the evaluated patients, 32 (24.8%) had DP and 97 (75.2%) had NDP (Table 1). Nocturnal systolic and diastolic BP were significantly higher in the group with NDP (p<0.001, p:0.001, respectively) (Table 2). Among the HRV parameters, low frequency (LF, 0.04-0.15 Hz) / high frequency (HF, 0.15-0.4 Hz) ratio was lower in the group with NDP, and PNN50 was higher (p:0.002, p:0.047, respectively) (Table 2). In the linear regression analysis, an independent relationship was found between the percentage of dipping and the HRV parameters standard deviation (SD) of all NN intervals (SDNN) and SD of the average NN intervals calculated over a 5-min period of the entire recording (SDANN) (p:0.034, p:0.0.15, respectively)(Table 4).

Conclusion: In our study, we could not detect a correlation between the HRV parameters associated with basal autonomic activity and the percentage of dipping. In addition, we found independent associations between SDNN and SDANN and percentage of dipping. This suggests that the decrease in nighttime blood pressure may be independent of basal autonomic activity.

Table 1. Comparison of clinical and laboratory characteristics of the study population according to the dipping pattern

Variables	Dipper (N:32)	Nondipper (N:97)	Total (N:129)	P
Age, years	49.3±17.2	59.8±17.9	57.2±18.2	0.005
Male/Female, N(%)	20 (62.5)/ 12 (37.5)	43 (44.3)/ 54 (55.7)	63 (48.8)/ 66 (51.2)	0.075
Systolic OBP, mmHg	131±19.9	134±19.4	133.3±19.5	0.457
Diastolic OBP, mmHg	80.5 (70/90)	80 (70/82)	80 (70/90)	0.177
CAD, N(%)	5 (15.6)	29 (29.9)	34 (26.4)	0.112

Table 1. Comparison of clinical and laboratory characteristics of the study population according to the dipping pattern (Continued)

Variables	Dipper (N:32)	Nondipper (N:97)	Total (N:129)	P
DM, N(%)	4 (12.5)	17 (17.5)	21 (16.3)	0.504
Smoking, N(%)	5 (15.6)	8 (8.3)	13 (10.2)	0.237
HT, N(%)	12 (37.5)	58 (59.8)	70 (54.3)	0.028
ACEi/ARB, N(%)	10 (31.3)	44 (45.4)	54 (41.9)	0.161
Beta Blocker, N(%)	11 (34.4)	47 (48.5)	58 (45)	0.165
CCB, N(%)	5 (15.6)	26 (26.8)	31 (24)	0.199
Statin, N(%)	32 (12.5)	25 (25.8)	29 (22.5)	0.119
Aspirin, N(%)	7 (21.9)	34 (35.1)	41 (31.8)	0.165
Laboratory:				
Glucose, mg/dl	94 (86.2/108.5)	104 (91/121.5)	101 (89/119)	0.046
GFR, ml/min/1.73 m^2	100.5±20.6	86.7±23.8	90.1±23.7	0.004
Hemoglobin, g/dl	14.1±1.7	13.3±1.9	13.5±1.9	0.046
Platelet, x10^3/µl	239 (208/280)	230 (189/273)	233 (195/275)	0.230
Total cholesterol, mg/dll	199.1±47.5	192±37.7	194.3±41.1	0.451
LDL cholesterol, mg/dl	125.4±41.2	119.2±33.2	121.1±35.7	0.463
HDL cholesterol, mg/dl	49.5 (41.5/61)	48 (38/56)	48 (41/58)	0.541
Triglyceride, mg/dl	133(86/199)	139 (110/163)	136 (102/163)	0.559
Echocardiography:				
LVEF, %	65 (61/65)	65 (61/65)	65 (61/65)	0.787
LVEDD, mm	45.5±4.9	45.4±5.7	45.4±5.4	0.866
IVS, mm	11 (10/12)	11 (10/13)	11 (10/12)	0.069
LA, mm	35.8±7.3	37.1±6.1	36.8±6.4	0.354
Aorta, mm	31.5 (29.2/34)	32 (30/35.5)	32 (30/35)	0.144
LVM, g	176.9±57.8	192.8±53.3	188.6±54.7	0.161

Data are mean±standard deviation or median (25%/75% quartiles) or number (percentage). ACEi; angiotensin converting enzyme inhibitor, ARB; angiotensin receptor blocker, OBP; office blood pressure, CAD; coronary artery disease, CCB; calcium channel blocker, DM; diabetes mellitus, GFR; glomerular filtration rate, HDL-C; high-density lipoprotein cholesterol, HT; hypertension, IVS; interventricular septum thickness, LA; left atrium, LDL-C; low-density lipoprotein cholesterol, LVEDD; left ventricular end-diastolic diameter, LVM; left ventricular mass, LVEF; left ventricular ejection fraction.

Table 2. Comparison of ABPM and rhythm holter results of the study population according to the dipping pattern

Variables	Dipper (N:32)	Nondipper (N:97)	Total (N:129)	p
ABPM:				
24-h systolic BP, mmHg	125.1±14.1	131.3±16.4	129.8±16	0.057
24-h diastolic BP, mmHg	73.7±8.7	73.3±9.6	73.±9.3	0.840
Day systolic BP, mmHg	130.1±13.5	132.3±16.9	131.7±16.1	0.500

Table 2. Comparison of ABPM and rhythm holter results of the study population according to the dipping pattern (Continued)

Variables	Dipper (N:32)	Nondipper (N:97)	Total (N:129)	p
Day diastolic BP, mmHg	78±9.1	74.5±10.2	75.4±10	0.090
Night systolic BP, mmHg	111.1±12.5	129.5±17.8	124.9±18.4	<0.001
Night diastolic BP, mmHg	63.5±9.2	70.1±9.3	68.5±9.6	0.001
Systolic dipping, %	14.5±3.9	2.1±6.5	5.1±8	<0.001
Diastolic dipping, %	18.3±7.3	56±8.7	8.7±10	<0.001
24h rhythm Holter:				
Mean heart rate, bpm	80.3±13.3	73.5±11.5	75.2±12.5	0.006
Minimum heart rate, bpm	48.5 (44/52.7)	46 (42/50)	46 (42/51)	0.033
Maximum heart rate, bpm	13.5±27.8	121.5±27.6	123.8±27.8	0.115
LF, ms^2	594 (245/775)	346 (206/705)	417 (230/731)	0.106
HF, ms^2	130 (67/328)	150 (78/300)	144 (74/318)	0.697
LF/HF	3.63 (2.33/5.94)	1.52 (2.42/3.74)	2.62 (1.6/4.03)	0.002
SDNN, ms	134.3±38.5	127.3±40.7	129.1±40.1	0.396
SDANNi, ms	129.4±47.9	118.2±42.2	121±43.8	0.209
SDNNi, ms	49.4±16.3	50±19.1	49.8±18.4	0.875
RMSSD, ms	22 (17/34.2)	28 (19/39)	27 (18.5/39)	0.134
PNN50, %	3 (1/9.75)	6 (2/15)	5 (2/14)	0.047

Data are mean±standard deviation or median (25%/75% interquartile range) or number (percentage). BP; blood pressure, HF; high-frequency component, LF; low-frequency component, pNN50; the proportion of adjacent RR intervals differing by>50ms in the 24-h recording, rMSSD; the square root of the mean squared differences of successive normal-to-normal intervals, SDANN; the standard deviation of the average normal-to-normal intervals calculated over the 5-min period of the entire recording, SDNN; the standard deviation of all normal-to-normal.

Table 3. Correlation analysis of systolic dipping percent and other clinical and laboratory parameters

Variables	r	p	Variables	r	p
Age	-0.328	<0.001	Laboratory:		
Gender	0.095	0.285	Glucose	-0.148	0.093
OBP systolic	-0.164	0.063	GFR	0.375	<0.001
OBP systolic	0.053	0.554	Hemoglobin	0.216	0.017
CAD	-0.322	<0.001	Platelet	0.142	0.121
DM	0.137	0.122	Total cholesterol	0.055	0.618
Smoking	-0.026	0.771	LDL cholesterol	0.040	0.711
HT	-0.224	0.011	HDL cholesterol	0.026	0.813
ACEi/ARB	-0.220	0.012	Triglyceride	-0.041	0.711
Beta Blocker	-0.221	0.012	Echocardiography:		
CCB	-0.175	0.048	LVEF	0.191	0.191
Statin	-0.211	0.016	LVEDD	0.057	0.530

Table 3. Correlation analysis of systolic dipping percent and other clinical and laboratory parameters (Continued)

Variables	r	p	Variables	r	p
Aspirin	-0.150	0.090	IVS	-0.237	0.008
24-h rhythm Holter:			PW	-0.212	0.019
Mean heart rate	0.255	0.003	LA	-0.155	0.089
Minimum heart rate	0.107	0.228	Aorta	-0.129	0.153
Maximum heart rate	0.266	0.003	LVM	-0.137	0.135
LF	0.108	0.221	ABPM:		
HF	0.044	0.620	24-h systolic BP	-0.123	0.164
LF/HF ratio	0.211	0.017	24-h diastolic BP	0.207	0.018
LF/HF ratio	0.180	0.041	Day systolic BP	0.047	0.130
SDANN	0.203	0.021	Day diastolic BP	0.359	<0.001
SDNNi	-0.069	0.437	Night systolic BP	-0.052	0.557
rMSSD	-0.039	0.664	Night diastolic BP	-0.243	0.006
pNN50	-0.047	0.599			

ACEi; angiotensin converting enzyme inhibitor, ARB; angiotensin receptor blocker, BP; blood pressure, CAD; coronary artery disease, CCB; calcium channel blocker, DM; diabetes mellitus, GFR; glomerular filtration rate, HDL-C; high-density lipoprotein cholesterol, HT; hypertension, HF; high-frequency component, IVS; interventricular septum thickness, LA; left atrium, LDL-C; low-density lipoprotein cholesterol, LF; low-frequency component, LVEDD; left ventricular end-diastolic diameter, LVM; left ventricular mass, LVEF; left ventricular ejection fraction, OBP; office blood pressure, PW; posterior wall thickness, pNN50; the proportion of adjacent RR intervals differing by>50ms in the 24-hr recording, rMSSD; the square root of the mean squared differences of successive normal-to-normal intervals, SDANN; the standard deviation of the average normal-to-normal intervals calculated over the 5-min period of the entire recording, SDNN; the standard deviation of all normal-to-normal intervals, SDNNi; SDNN index, the mean of the deviation of the 5-min normal-to-normal intervals over the entire recording.

Table 4. Linear logistic regression analysis to determine independent predictors of systolic dipping percent

Model	Variable	Beta Standardized	p
1	LF/HF ratio	0.160	0.056
	CAD	-0.241	0.006
	GFR	0.315	<0.001
2	CAD	-0.350	0.001
	Aspirin	0.206	0.053
	GFR	0.337	<0.001
	SDNN	0.177	0.034
3	CAD	-0.362	0.001
	Aspirin	0.201	0.055
	GFR	0.116	<0.001
	SADNN	0.203	0.015

Model 1: LF/HF ratio*, age, systolic OBP, CAD, HT, ACEi/ARB, BB, CCB, statin, aspirin, mean HR, glucose*, Hb, GFR, LVEF*, IVS*, LA Model 2: SDNN, age, systolic OBP, CAD, HT, ACEi/ARB, BB, CCB, statin, aspirin, mean HR, glucose*, Hb, GFR, LVEF*, IVS*, LA Model 3: SDANN, age, systolic OBP, CAD, HT, ACEi/ARB, BB, CCB, statin, aspirin, mean HR, glucose*, Hb, GFR, LVEF*, IVS*, LA * Square root transformation applied to nonnormally distributed variables ACEi; angiotensin converting enzyme inhibitor, ARB; angiotensin receptor blocker, BB; Beta blocker, BP; blood pressure, CAD; coronary artery disease, CCB; calcium channel blocker, GFR; glomerular filtration rate, HF; high-frequency component, HT; hypertension, HR; heart rate, LA; left atrium, LF; low-frequency component, Hb; hemoglobin, HT; hypertension, IVS; interventricular septum thickness, LVEDD; left ventricle end diastolic diameter, LVEF; left ventricular ejection fraction, LVM; left ventricular mass, OBP; office blood pressure, pNN50; the proportion of adjacent RR intervals differing by>50ms in the 24-h recording, rMSSD; the square root of the mean squared differences of successive normal-to-normal intervals, SDANN; the standard deviation of the average normal-to-normal intervals calculated over the 5-min period of the entire recording, SDNN; the standard deviation of all normal-to-normal intervals, SDNNi; SDNN index, the mean of the deviation of the 5-min normal-to-normal intervals over the entire recording.

PB-025 [Hypertension]

Comparison of the efficacy of amlodipine, benidipine and lercanidipine in hypertensive patients

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Background and Aim: Hypertension is a global health problem that affects more than 1 billion people worldwide. Various drugs such as ACE inhibitors, calcium channel blockers, and diuretics are used in the treatment of hypertension. Amlodipine, Benidipine and Lercanidipine are calcium channel blockers for the treatment of high blood pressure (hypertension). In this study, we compared the efficacy and safaety of Amlodipine, Benidipine and Lercanidipine in hypertension patients.

Methods: Patient data collected within the scope of the study were analyzed with the IBM Statistical Package for the Social Sciences for Windows 23.0 (SPSS 23.0IBM) package program. Frequency and percentage for categorical data, mean and standard deviation for continuous data were given as descriptive values. Paired Sample T-Test was used to compare pre- and post-treatment values, ANOVA test was used for comparisons between groups, and Post-hoc Benferroni test was used to determine which groups caused the significance in the parameters found significant in the ANOVA test. "Chi-Square Test" was used to compare categorical variables. The results were considered statistically significant when the p value was less than 0.05.

Results: Our study, involved 1157 patients who received Amlodipine (n=461, 39.8%) Amlodipine, Benidipine (n=105, 9.1%) Benidipine and Lercanidipine (n=591,51.1%). The evaluation of the pulse pressures (PP) of the patients before and after the treatment is given in Table 1. When the changes in the pulse pressures of the patients, before and after the treatment were examined, it was observed a statistically significant decrease with all treatments (p<0.05). However differently from Benidipine, Lercanidipine (Table 1) was found to have a higher effect on reducing pulse pressure (p<0.01). This effect was statistically equivalent to that of amlodipine, but significantly lower than that of Benidipine.

The control rates of blood pressure, before and after treatment are given in Table 2. It was observed that the rate of blood pressure control was the highest with Lercanidipine treatment (89.3%), compared with Benidipine (69.5%), while the control rates of blood pressure of Lercanidipine was statistically equivalent to that of amlodipine. A statistically significantly higher clinical efficacy was observed with Lercanidipine and Amlodipine compared to Benidipine. (p<0.05).

Evaluation of blood pressures before and after treatment is given in Table 3. After the treatment, there was a statistically significant difference with both three treatment groups only in terms of systolic blood pressure (p<0.05). Lercanidipine treatment appeared to have a higher blood pressure lowering effect than the other two treatments, while Benidipine had the lowest effect.

Conclusions: The results of our study show that the pulse pressure, the the blood pressure control rates and the blood pressure lowering effect of Lercanidipine were statistically significantly differents from that of Benidipine and equivalent to that of amlodipine.

Table 1. Distribution of blood pressure control rates before and after treatment

Blood Pressure Control (%)	Amlodipine n= 461	Benidipine n= 105	Lercanidipine n=591	p-value
Pretreatment	34.3%	30.5%	29.4%	0.240
Post-treatment	**83.9%**	**69.5%**	**89.3%**	**<0.001**

Table 2. Evaluation of blood pressures before and after treatment

Blood Pressure(mmhg)	Amlodipine n= 461	Benidipine n= 105	Lercanidipine n=591	p-value	Difference*
Pretreatment Systolic	143.5±15.5	144.1±10.2	145.1±16.9	0.249	
Post-treatment Systolic	**128,2±14,1**	**131,8±13.9**	**127,3±15,2**	**0.014**	b-c
p-value	**<0.001**	**<0.001**	**<0.001**		
Pretreatment Diastolic	87.6±10.0	87.0±10.5	88.2±10.1	0.430	
Post-treatment Diastolic	81.1±9.6	81.7±10.9	81.0±10.4	0.790	
p-value	**<0.001**	**0.001**	**<0.001**		

*** a= Amlodipine; b= Benidipine; c= Lercanidipine.**

Table 3. Evaluation of pulse pressures before and after treatment

Pulse Pressure(mmhg)	Amlodipine n= 461	Benidipine n= 105	Lercanidipine n=591	p-value	Difference*
Pretreatment	56.0±14.0	57.1±14.8	56.9±14.8	0.550	
Post-treatment	**47.2±9.9**	**52.2±10.8**	**46.3±8.9**	**<0.001**	a-b, b-c
p-value	**<0.001**	**0.007**	**<0.001**		

*** a= Amlodipine; b= Benidipine; c= Lercanidipine.**

PB-027 [Valvular Heart Diseases]

Diagnosis, treatment & management of stroke in patients with prosthetic valve endocarditis

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Background and Aim: ½Prosthetic valve endocarditis (PVE) is a major complication of heart valve surgery and is becoming more common due to the increasing number of patients undergoing valve replacement surgery. According to a multicenter study, it was determined that PVE constitutes approximately 20% of the total number of IE patients. PVE management is holistic and includes immediate diagnosis, treatment with antibiotics or surgery. In this context, an endocarditis team should be established within the hospital. Studies have shown that in-hospital mortality for PVE patients is up to 22 percent, and this risk increases with concomitant stroke during follow-up or admission. A recent study found that double valve replacement, rheumatic heart disease, and fungal infection are independent risk factors for stroke in PVE patients. In our study, we aimed to define more predictors of stroke in patients with PVE and to contribute to the literature.

Methods: All patients were PVE patients admitted to our center over a period of approximately 16 years (2006-2021). We retrospectively scanned the computer records of all discharged patients diagnosed with PVE and stroke then reviewed them against the new diagnostic criteria for IE (Modified Duke)

Results: The rate of CVA history of the patients followed up with endocarditis and undergoing CVA was statistically significantly higher than the patients without CVA, and the INR level was lower (respectively p=0.028 p=0.045). The success rate of treatment in patients who underwent CVA during follow-up was statistically significantly lower than in patients without CVA (p=0.046). The ESR level of the patients who underwent CVA during the follow-up was statistically significantly higher than the patients without vege area CVA (p=0.043 p<0.001, respectively). In the univariate Logistic Regression Analysis analysis, the risk factors affecting having CVA in the follow-up were found to be a history of CVA, treatment failure, vege area, and level as statistically significant factors (p=0.023 p=0.049 p=0.008). In the model created for risk effects for multivariate Logistic Regression Analysis, the risk factors affecting having CVA at follow-up, the patient's previous history of CVA had the highest odds ratio (OR: 4,558). In the ROC Curve analysis performed for the cut-off value of the vege area, which determines CVA during the follow-up, it was determined as 82.6% sensitivity, 73.7% specificity and 1.7 and above cut-off value.

Conclusions: As a result; In patients with PVE, monitoring of CVA in the in-hospital follow-up is a condition that increases the morbidity and mortality of the patients. In our study, the patient's previous history of CVA, vegetation size and treatment failure were prominent as risk factors in the regression analyzes. It has a sensitivity of 82.6% and a specificity of 73.7% for CVA in the follow-up of vegetation over 1.7 cm2. However, our findings need to be confirmed by larger studies.

PB-028 [Cardiac Insufficiency]

Non-ischemic cardiomyopathy, mildly reduced heart failure characteristics, and survival predictors

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Background and Aim: Heart failure mildly reduced ejection fraction (HFmrEF) patients, defined as left ventricular ejection fraction (LVEF) between 41 to 49% with heart failure symptoms and findings.In this study, we aimed to evaluate non-ischemic-HFmrEF patients' primary end-points(all-cause mortality, heart replacement therapies,ventricular ar-

rhythmia,or heart failure-related hospitalization),and predictors of MACE in the Cox-regression time series.

Methods: Heart failure patients between 2008-2021, 2226 heart failure patients reviewed. HCMP, ARVC, restrictive and ischemic cardiomyopathy patients were excluded. We targeted purging ischemic heart disease effects on HFmrEF patients and decreasing comorbidities' impact on survival.

Results: The 56 HFmrEF patient's enrolled in the study. The mean age was 39.6±12.3 years,and 42.1% were women. The mean LVEF was 45.6±2.78%. As presented in Table 1, hypertension was present in 24.1% of the patients, smoking history in 29.6%, diabetes mellitus in 14.8%,and atrial fibrillation was present in 17% of the patients.Comorbidities of renal failure and chronic obstructive pulmonary disease were low(3.7% and 3.4 %, respectively). The median follow-up time was 1339 days. The five-year primary end-point free survival was 79.5% (the event rate was 20.5%). In a univariate analysis of atrial fibrillation (HR: 4.63; 95%CI 1.29 to 16.8, p=0.0018),loop diuretic usage (HR: 6.16; 95%CI 1.31 to 2898, p=0.0021), left atrial diameter (HR: 1.18; 95%CI 1.07 to 1.30, p<0.001), LVEF (HR: 1.31; 95%CI 1.05 to 1.64, p=0.0015), moderate or severe mitral regurgitation (HR:3.88, 95% CI 1.23 to 12.24, p=0.021) and moderate or severe tricuspid regurgitation (HR: 11.46; 95%CI 1.82 to 71.9, p=0.009) were significant predictors.

Conclusions: HFmrEF patients account for up to 25% of patients with heart failure. HFmrEF is an intermediate HF type between HFpEF and HFrEF. HFmrEF patients have a higher prevalence of ischemic heart disease.In this study, the ischemic heart disease impact was removed from the HFmrEF patients.The 56 patients had low comorbidities.This cohort was almost

pure from the factors that may change the prognosis in the HFrEF and HFpEF patients. In univariate analysis, atrial fibrillation left atrial diameter, and moderate-severe mitral or tricuspid regurgitation were univariate predictors of the primary end-point free survival. The increase of the LVEF was the univariate poor predictor of the primary end-point free survival. So this phenotype of the HFmrEF patients' resembles heart failure preserved ejection fraction patients. CHART-2 Study examined 3480 consecutive HF patients. Patients with HFmrEF, on average, are more similar to HFrEF than HFpEF because they are more often male, younger, and more likely to have CAD, and less likely to have AF and non-cardiac disease. However, the exclusion of the ischemic group in this study made the clinical features of the patient group more evident.

Table 1. Characteristics and echocardiographic parameters of the heart failure mildly reduced non-ischemic cardiomyopathy patients

Anti-platelet n (%)	22 (%39.3)	15 (%26.78)	7 (%12.50)	0.367
Mitral Regurgitation Severe n (%)	3 (%5.4)	1 (%1.78)	2 (%3.6)	0.072
Mitral Regürgitation Modarate n (%)	8 (%14.3)	5 (%8.92)	3 (%5.4)	
Mitral Regurgitation-Mild n (%)	28 (%50)	21 (%37.50)	7 (%12.50)	
Tricuspid Regurgitation-Severe n (%)	2 (%3.6)	0 (%0.0)	2 (%3.6)	**0.023**
Tricuspid Regurgitation-Moderate n (%)	4 (%7.1)	2 (%3.6)	2 (%3.6)	
Tricuspid Regurgitation-Mild n (%)	27 (%48.2)	21 (%37.50)	6 (510.7)	
Pacemaker implantation n (%)	11 (%19.7)	7 (%12.50)	4 (%7.14)	0.445
LVEd mm	54.4 ± 7.28	53.9 ±6.8	55.9 ± 8.8	0.40
LVEs mm	39.6 ± 8.04	39.7 ± 7.0	39.2 ± 7.0	0.83
LAd mm	41.1 ± 7.96	38.7 ± 7.1	48.7 ± 5.3	**<0.0001**
LVEF %	45.61 ± 2.79	45.1 ±2.5	47.2 ± 3.1	**0.043**
TRV m/sec	2.49 ± 0.50	2.38 ± 0.44	3.07 ±0.44	**0.009**
RVsm TDI m/sec	12.27 ± 2.47	12.3 ±2.52	11.8 ±2.40	0.63
TAPSE mm	21.40 ± 4.81	21.1 ±4.66	22.7 ±5.77	0.44
SPAP mmHg	33.86 ± 11.97	30.2±8.4	43.5 ±15.5	**0.091**

* Values are mean ±SD or n (%) p value<0.05.
LVEF=Left ventriculer ejection fraction; ICD= Implanted cardioverter-defibrillator; CRT-D= Cardiac resynchronization therapy-Defibrillator ACEi=Angiotensinogen converting enzyme inhibitor; ARB=Angiotensin receptor blocker; AF= Atrial fibrillation; BMI=body mass index; BSA: body surface area; COPD= Chronic obstructive pulmonary disease
LVEDd= Left ventricular end diastolic diameter; LVESd= Left ventricular end systolic diameter; LAd= Left atrial diameter; TAPSE= Tricuspid annuler plane systolic excursion; RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure

Characteristics and echocardiographic parameters of the heart failure mildly reduced non-ischemic cardiomyopathy patients Table 1-2.

Table 2. Characteristics and echocardiographic parameters of the heart failure mildly reduced non-ischemic cardiomyopathy patients

	Total	**Primary end-point (-)**	**Primary end-point (+)**	**P-value**
Age years		39.9 ± 12.7	38.4 ± 11.2	0.69
Male n (%)	33 (%58.9)	25 (%44.6)	8 (%14.3)	0.827
Hypertension n (%)	13 (%23.2)	8 (%14.28)	5 (%8.92)	0.134
Diabetes Mellitus n (%)	8 (%14.3)	7 (%12.50)	1 (%1.78)	0.667
Hyperlipidemia n (%)	4 (%7.1)	3 (%5.35)	1 (%1.78)	0.646
Smoking History n (%)	16 (%28.6)	11 (%19.64)	5 (%8.92)	0.309
COPD n (%)	6 (%10.7)	5 (%8.92)	1 (%1.78)	0.740
Chronic Kidney Disease n (%)	2 (%3.6)	1 (%1.78)	1 (%1.78)	0.398
Atrial Fibrillation n (%)	9 (%16.1)	5 (%8.92)	4 (%7.14)	0.78
Family History n (%)	9 (%16.1)	8 (%14.28)	1 (%1.78)	0.668
ACEi-ARB n (%)	40 (%71.4)	31 (%55.35)	9 (%16.07)	0.548
Beta-Blocker n (%)	49 (%87.5)	37 (%66.07)	12 (%21.42)	0.481
Mineralocorticoid receptor antagonist n (%)	24 (%42.9)	14 (%25.0)	10 (%17.9)	0.012
Loop Diuretic n (%)	25 (%44.6)	14 (%25.0)	11 (%19.64)	**0.001**
Hydrochlorothiazide n (%)	9 (%16.1)	4 (%7.14)	5 (%8.92)	**0.024**
Ivabradine n (%)	10 (%17.9)	4 (%7.14)	6 (%10.7)	**0.007**
Ventricular Arrhythmia n (%)	2 (%3.6)	0 (%0.0)	2 (%3.6)	**0.009**
Death	12 (%21.4)	1 (%1.78)	11 (%19.6)	**<0.001**
Anti-coagulant n (%)	11 (%19.6)	5 (%8.92)	6 (%10.7)	**0.013**
Vit-K Antagonist n (%)	8 (%14.3)	4 (%7.14)	4 (%7.14)	**0.019**
DOAC n (%)	3 (%5.4)	1 (%1.78)	2 (%3.6)	**0.009**

Characteristics and echocardiographic parameters of the heart failure mildly reduced non-ischemic cardiomyopathy patients Table 1-1.

Table 3. Cox univariate and multivariate analysis of clinical parameters heart failure mid-range non-ischemic cardiomyopathy, patients

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	P-value	HR (95% CI)	P-value
Age years	1.005 (0.958-1.055)	0.17	-	-
Gender	1.031 (0.324-3.284)	0.95	-	-
Arterial hypertension	2.283 (0.692-7.537)	0.17	-	-
Diabetes mellitus	0.701 (0.088-5.573)	0.73	-	-
Dyslipidemia	0.798 (0.100-6.367)	0.83	-	-
Smoking history	1.444 (0.415-5.021)	0.56	-	-
COPD	1.20 (0.56-2.58)	0.62	-	-
Renal Disease	4.392 (0.538-35.88)	0.16	-	-
Atrial fibrillation	**4.635 (1.296-16.58)**	**0.018**	-	-
Family History	0.527 (0.067-4.146)	0.54	-	-
ACEi or ARB	1.318 (0.396-4.389)	0.65	-	-
Beta-blocker	1.398 (0.178-11.01)	0.75	-	-
Aldosterone antagonist	0.302 (0.080-1.133)	0.076	-	-
Loop diuretic	**6.177 (1.314-28.93)**	**0.021**	-	-
Hydrochlorothiazide	1.100 (0.302-4.0)	0.88	-	-
Anticoagulant	1.427 (0.423-4.809)	0.56	-	-
Antiplatelet	2.886 (0.906-9.186)	0.073	-	-
Ivabradine	1.625 (0.477-5.532)	0.43	-	-

Cox univariate and multivariate model, P value<0.05.
HR= Hazard ratio; CI= Confidence interval
ACEi=Angiotensinogen converting enzyme inhibitor; ARB=Angiotensin receptor blocker; AF= Atrial fibrillation; COPD= Chronic obstructive pulmonary disease

Cox univariate and multivariate analysis of clinical parameters heart failure mid-range non-ischemic cardiomyopathy, patients Table 2.

Table 4. Cox univariate and multivariate analysis of echocardiographic parameters heart failure mid-range non-ischemic cardiomyopathy, patients

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	P-value	HR (95% CI)	P-value
LVEDd mm	1.059 (0.971-1.154)	0.19	-	-
LVESd mm	1.030 (0.960-1.105)	0.41	-	-
LAd mm	**1.183 (1.076-1.300)**	**< 0.001**	**1.185 (1.077-1.303)**	**<0.0001**
LVEF %	**1.318 (1.055-1.647)**	**0.015**	-	-
Mitral Regurgitation		0.141		
Mild	3.733 (0.442-30.9)	0.22	-	-
Moderate	5.840 (0.601-56.7)	0.12	-	-
Severe	**15.85 (1.385-181.5)**	**0.026**	-	-
Aortic Regurgitation	3.055 (0.809-11.53)	0.10	-	-
Tricuspid Regurgitation		0.014	-	-
Mild	1.226 (0.288-5.206)	0.78	-	-
Moderate	**7.773 (1.197-50.4)**	**0.032**	-	-
Severe	12.118 (1.883-77.9)	0.009	-	-
TAPSE mm	1.151 (0.977-1.357)	0.093	-	-
RVsm (TDI) m/sec	1.060 (0.728-1.544)	0.762	-	-
TRV m/sec	23.0 (0.999-529)	0.050	-	-
SPAP mmHg	1.051 (0.999-1.106)	0.056	-	-

Values are mean ±SD or n (%) p value<0.05.
LVEDd= Left ventricular end diastolic diameter; LVESd= Left ventricular end systolic diameter; LAd= Left atrial diameter; LVEF= Left ventricular systolic ejection fraction; TAPSE= Tricuspid annuler plane systolic excursion; RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure

Cox univariate and multivariate analysis of echocardiographic parameters heart failure mid-range non-ischemic cardiomyopathy, patients Table 3.

PB-029 [Cardiac Insufficiency]

Prognostic significance of iron deficiency in non-ischemic heart failure patients with low ejection fraction: a retrospective cohort study

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Background and Aim: Iron Deficiency (ID) in Heart Failure (HF) is frequently observed as comorbidity and is associated with poor clinical outcomes. Studies on this subject were mostly conducted in patients with ischemic HF. Therefore, the importance of the presence of ID in non-ischemic patients is not well known. Hence, in the present study, we aimed to investigate the prognostic significance of ID in non-ischemic HF patients with low ejection fraction (HFrEF) than ischemic patients.

Methods: The present study was included 148 patients with ischemic (59 patients) and non-ischemic (89 patients) HFrEF. The patients were divided into four groups according to the presence of ID. In addition to routine laboratory parameters, hematological parameters related to ID were also studied. The average follow-up time was 62.8 (3-144) months

Results: All-cause mortality between ischemic and non-ischemic HF groups was similar (p: 0.40). Ischemic patients with ID were associated with increased mortality (p:0.02) and shorter survival (p:0.04) than non-ischemic patients without ID (Figure 1). Moreover, patients with ID in addition to anemia had shorter survival than those without ID (Figure 2, P: 0.03). In multivariate analysis, there was a significant relationship between mortality and especially poor functional capacity, transferrin saturation, white blood cell, and serum creatinine levels (Table I).

Conclusions: In the present study, we demonstrated that the presence of ID in both HF groups is associated with a worse prognosis. Moreover, we suggested that ID may be a more important prognostic marker than anemia.

Table 1. Results of Univariate and Multivariate Cox's Proportional Hazard Models Regarding mortality

Characteristics	Univariate Analysis		*Multivariate Analysis*	
	OS HR (95% CI)	P Value	*OS HR (95% CI)*	*P Value*
Functional Capacity (2 vs. 3)	0.23 (0.15-0.35)	<0.001	0.31 (0.18-0.54)	<0.001
Diastolic BP	0.98 (0.96-0.99)	0.006		
Creatinine	2.88 (0.96-8.63)	0.04	15.4 (3.9-60.4)	<0.001
WBC	1.16 (1.04-1.28)	0.006	1.22 (1.07-1.38)	0.002
Neutrophyl	1.08 (1.01-1.15)	0.01		
Transferrin Saturation	0.95 (0.93-0.98)	<0.001	0.93 (0.87-0.97)	<0.001
Iron	0.98 (0.97-0.99)	0.01		

BMI: Body Mass Index, BP: blood pressure, HF: Heart Failure, ID: Iron Deficency, NLR: neutrophil/lymphocyte ratio. WBC: White Blood Cell

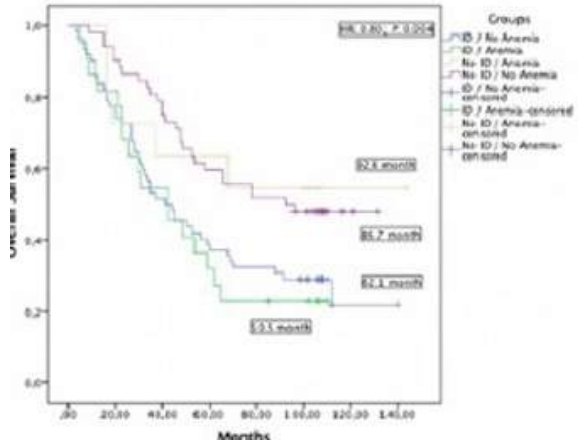

Figure 1. Kaplan-Meier Median Overall Survival curve in HF groups with or without ID

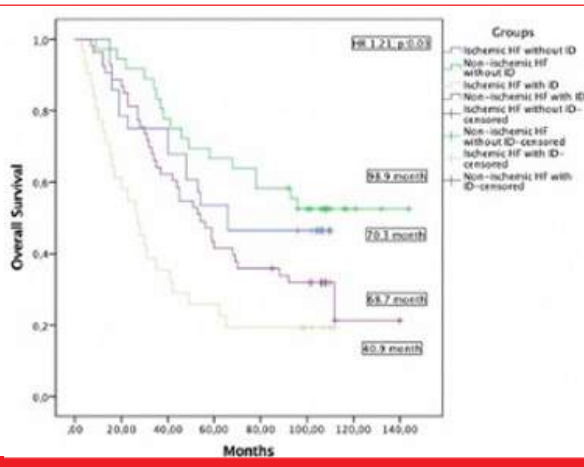

Figure 2. Survival analysis. Kaplan-Meier curves reflecting the difference in survival rates between HF patients with or without ID and anemia

PB-030 [Cardiac Insufficiency]

Adherence to treatments in elderly patients with chronic heart failure

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Background and Aim: Heart failure (HF) is an important cause of mortality and morbidity despite advanced treatments. Most elderly patients with HF also have more than one comorbidity. This further complicates medication and lifestyle, and sometimes leads to reduced adherence to HF treatment. The aim of our study was to show whether elderly HF patients receive treatments known to reduce mortality in adequate doses.

Methods: HF patients aged ≥75 years and ≤50% left ventricular ejection fraction (LVEF) admitted to a tertiary hospital cardiology outpatient clinic between 2020-2021 were included. Demographic, clinical, and laboratory parameters, treatments, and doses were evaluated retrospectively.

Results: 274 patients were included in the study. The median age of patients was 79 years [76–83] and 60.4% (n=165) were male. The median LVEF was 30% [25–35]. Of the patients, 63.5% had hypertension, 34.7% diabetes, 27% chronic kidney disease, 32.1% hyperlipidemia, 12% cerebrovascular disease, 21% chronic obstructive pulmonary disease. 74.5% of the patients were using angiotensin-converting enzyme inhibitor/angiotensin receptor blocker (ACE-I/ARB), 91.6% beta-blockers, 58.8% spironolactone, %1.8 angiotensin receptor/neprilysin inhibitör (Figure 1). In addition, 74.1% were using diuretics, 7.3% were using digoxin, 5.1% were using ivabradine, 35.5% were using statins, 50.7% were using acetylsalicylic acid, 29.2% were using oral anticoagulants. Implantable cardioverter defibrillator/cardiac resynchronization therapy was present in 10.3% of patients. While 14.6% of the patients were using ACE-I/ARB at the maximum dose, 32.5% were using ≥50% of the target dose and 27% were using it at <50% of the target dose. While the rate of using beta-blockers at the maximum dose was 3.6%, the rate of those using ≥50% of the dose was 28.5%, and the rate of using <50% of the dose was 58.8%. The rate of those using the maximum dose for spironolactone was 1.8%, the rate of using at least 50% dose was 36.5%, and the rate of using <50% dose was 20.4% (Figure 2). The rates of ACE-I/ARB use and reaching target doses were similar in men and women (respectively, p=0.744, p=0.963). There was no difference in beta-blockers usage rates (p=0.699), but the rate of those who used at least 50% of the target dose was 41.3% in women and 26% in men (p=0.029). While the use of spironolactone was significantly higher in males, there was no difference in reaching the target doses (p=0.033, p=0.09).

Conclusions: Although elderly heart failure patients receive treatments known to reduce mortality in HF, they do not use these treatments at targeted doses. To improve HF outcomes, it is recommended that patients' treatments be increased to the maximum dose tolerated by the patient.

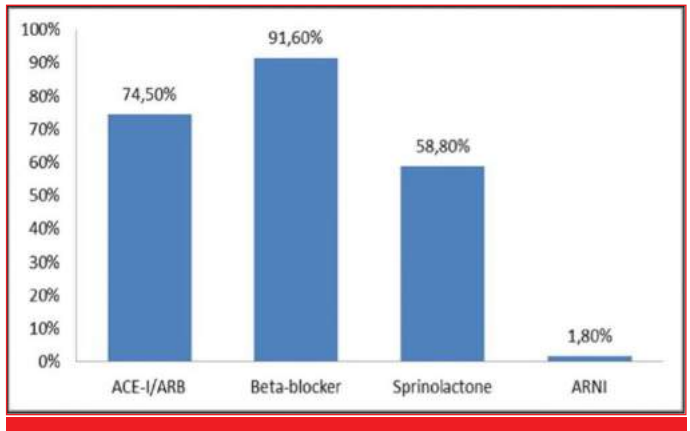

Figure 1. Treatments in elderly patients with chronic heart failure

ACE-I/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; ARNI, angiotensin receptor/neprilysin inhibitor.

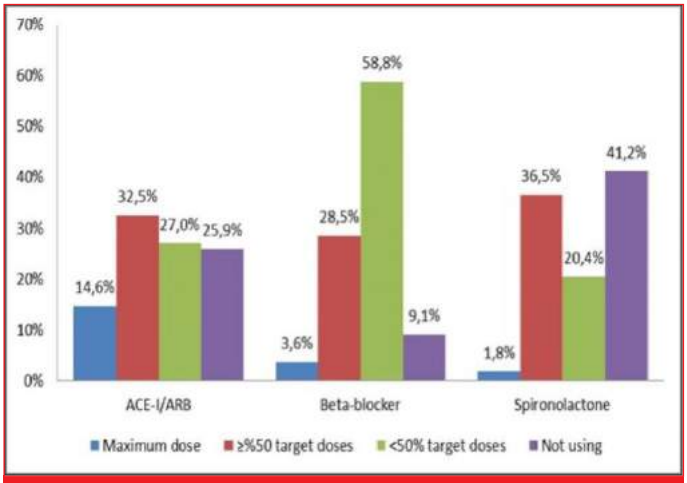

Figure 2. Treatment doses in elderly patients with chronic heart failure
ACE-I/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker.

PB-031 [Cardiac Insufficiency]

The relationship between urinary angiotensinogen and mortality in heart failure patients with reduced ejection fraction

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Background and Aim: Activation of the renin-angiotensin-aldosterone system (RAS) has an important role in the pathophysiology of heart failure with reduced ejection fraction (HFrEF). The effects of systemic RAS activation on HFrEF are well known. However, the effects of the local renin-angiotensin system on HFrEF are not fully known due to limited clinical studies. In this study, we investigated that the effect of urinary angiotensinogen (UAGT), which is accepted as an indicator of local RAS activation, is on all-cause mortality in patients with HFrEF.

Methods: A retrospective, single-center study included 63 patients who were previous UAGT levels. UAGT values were standardized with urinary creatinine (UCre) measured from the same urine sample. UAGT/UCre results were recorded, the median value was calculated and the patients were divided into two groups according to this value. All patients were screened in the national registry systems. Those who died and death date were recorded. The patients whose data could not be reached were called by us and questioned whether they were alive or not.

Results: When the patient group with a UAGT/UCre ratio above the median value was compared with the patient group with a UAGT/UCre ratio equal to or below the median value, 22 (71%) deaths were observed in terms of all-cause mortality over a 4-year period. It was determined that 10 (35.5%) deaths occurred in the other group (p=0.005).

Conclusions: Our research suggests that UAGT can be used as a new biomarker in the prognosis and follow-up of heart failure patients in the future.

Table 1. Basal characteristics of patients groups according to UAGT/UCre ratio

	Group 1 (median UAGT/UCre ≤ 144.01 µg/g) (n= 29)	Group 2 (median UAGT/UCre > 144.01 µg/g) (n= 31)	P
Age (year)	61.20 ± 11.95	67.93 ± 11.22	0.052
Gender (F/M)	9/23	9/22	0.80
Disease History			
Coronary artery disease (n,%)	21, %72.4	20, %65.5	0.55
DM (n,%)	9, %31	15, %48.4	0.15
HT (n,%)	25, %86	17, %54.8	0.19
BMI (kg/m²)	26.64 ± 3.56	27.4 ± 3.31	0.78
SBP (mmHg)	125 ± 23	118 ± 21	0.46
DBP (mmHg)	74 ± 12	71 ± 15	0.48
Heart Rhythm			
Sinus rhythm (n,%)	25, %86.2	20, %64.5	
Atrial fibrillation (n,%)	2, %6.9	9, %29	
Pacemaker rhythm (n,%)	2, %6.9	2, %6.5	
Device History			
ICD	12, %41.3	14, %45.1	0.80
CRT	2, %6.9	2, %6.5	1.0
History of medicine			
Anti-(ARB	27, %93.1	29, %93.5	1.0
Beta-blocker	28, %96.5	30, %96.7	1.0
MRA	27, %93.1	27, %87	0.67
SGLT-2	9, %31	15, %48.4	0.19
Ivabradine	3,%10	3,%9.7	1.0
Biochemical and Hematological Parameters			
Fasting blood glucose (mg/dL)	134.68 ± 64.34	134.38 ± 58.82	0.78
Creatinine (mg/dL)	1.00 (0.60-1.80)	0.98 (0.59-2.1)	0.96
Serum sodium (mEq/L)	139.08 ± 4.37	137.93 ± 2.78	0.14
Serum potassium (mEq/L)	4.67 ± 0.52	4.55 ± 0.63	0.45
AST (U/L)	24.20 ± 10.08	26.92 ± 21.94	0.39
ALT (U/L)	22.40 ± 15.96	15.98 ± 9.41	0.050
WBC (10³/µL)	8.08 ± 2.13	8.08 ± 3.10	0.13
Hg (g/L)	13.18 ± 2.10	12.4 ± 1.78	**0.032**
Hs-CRP (mg/dL)	5.66 ± 5.64	20.21 ± 21.20	**<0.001**
NT-proBNP (pg/mL)	548.5 (67-7071)	11.87 (5.18-1907)	**<0.001**
Fasting total cholesterol (mg/dL)	173.36 ± 58.80	161.17 ± 47.78	0.35
Fasting LDL-cholesterol (mg/dL)	101.44 ± 46.55	92.42 ± 34.49	0.72
Fasting triglyceride (mg/dL)	166.72 ± 128.68	139.71 ± 105.16	0.47

F/M: female/male, DM: diabetes mellitus, HT: hypertension, BMI: body mass index, SBP: systolic blood pressure, DBP: diastolic blood pressure, WBC: White blood cell count, Hg: hemoglobin, Hs-CRP: high sensitive C-reactive protein, LDL: low density lipoprotein, ICD: implantable cardioverter defibrillator, CRT: Cardiac resynchronization therapy, Anti-: Angiotensin-converting enzyme, ARB: Angiotensin II receptor blocker, MRA: Mineralocorticoid receptor antagonist, SGLT-2: Sodium glucose cotransporter-2.

The patients were divided into two groups according to the median value of UAGT/UCre (144.01 µg/g). 29 patients with a UAGT/UCre ratio equal to or lower than the median value were included in group 1, and 31 patients with a UAGT/UCre ratio higher than the median value were included in group 2. The two groups had similar characteristics in terms of age, gender, history, medications and vital signs. When the biochemical parameters were examined, lower hemoglobin (p=0.032), higher Hs-CRP (p<0.001) and NT-proBNP (p<0.001) levels were found to be statistically significant in group 2.

Table 2. Echocardiographic parameters in the groups

	Group 1 (n= 29)	Group 2 (n= 31)	P
Ejection fraction (%)	28.75 ± 5.45	29.38 ± 7.81	0.62
LVEDd (cm)	5.9 ± 0.66	6.01 ± 0.97	0.84
LVESd (cm)	4.73 ± 0.68	4.92 ± 0.9	0.56
PWd (cm)	1.05 ± 0.17	0.97 ± 0.17	0.17
LAd (cm)	4.51 ± 0.76	4.62 ± 1.01	0.77

LVEDd: left ventricular end diastolic diameter; LVESd: left ventricular end systolic diameter; PWd: posterior wall thickness; LAd: left atrial diameter

When the two groups were compared in terms of transthoracic echocardiographic parameters, no significant difference was found.

Table 3. Differences between groups in terms of all-cause mortality

Presence of mortality	Group 1 (n= 29)	Group 2 (n= 31)	P
None	19, %65.5	9, %29	**0.005**
Presence	10, %35.5	22, %71	

When the 4-year all-cause mortality was analyzed, it was found that 22 patients (71%) died in the group 2 with a UAGT/UCre ratio above the median value, and 10 patients (35.5%) died in the group 1 with a lower UAGT/UCre ratio, and this difference was statistically significant.

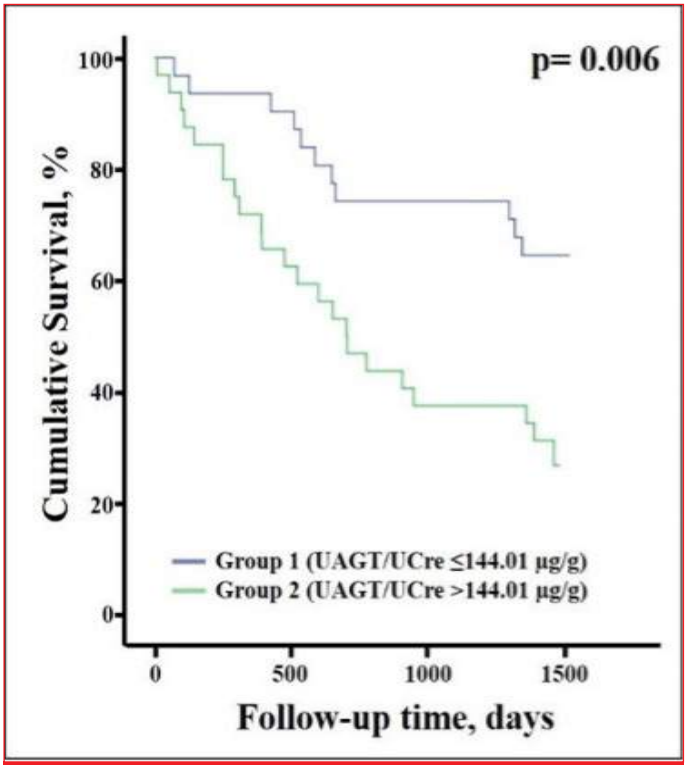

Figure 1. Kaplan-Meier curves of patient groups for all-cause mortality

In the 4-year survival analysis of the study groups, all-cause mortality was found to be higher in group 2 whose UAGT/UCre level was above the median value (log rank p=0.006).

PB-032 [Cardiac Insufficiency]

The effect of different beta-blockers groups on hospitalization and long-term mortality in patients with non-ischemic heart failure

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Background and Aim: Beta-blockers (BBs) are cornerstone agents in heart failure (HF) due to their beneficial effects on the sympathetic nervous system. However, which beta-blockers are more effective and should be the first choice in HF is still a matter of debate. We aimed to investigate the effect of different BBs on hospitalization and long-term mortality in non-ischemic HF with reduced Ejection Fraction (HFrEF).

Methods: The present study was included 106 patients with non-ischemic HFrEF. The patients were divided into three groups as carvedilol (41 patients), nebivolol (33 patients) and metoprolol (32 patients). The average follow-up time was 62.8 (3-144) months.

Results: At the last follow-up, the number of patients who died was 72 (68%). All-cause mortality was similar between BB groups [26 (63%) vs 22 (66%) and 24 (75%), respectively; P:0.57]. However, long-term survival was significantly higher (P: 0.04). Moreover, hospitalization due to HF was significantly lower (P<0.01) in the carvedilol group than the metoprolol group (figure-1). As expected, the patients with LV EF (<33.5%) had worse prognosis.

Conclusions: Carvedilol treatment is associated with longer survival and less hospitalization than metoprolol treatment. Therefore, it may be preferred over metoprolol treatment in patients with non-ischemic HFrEF.

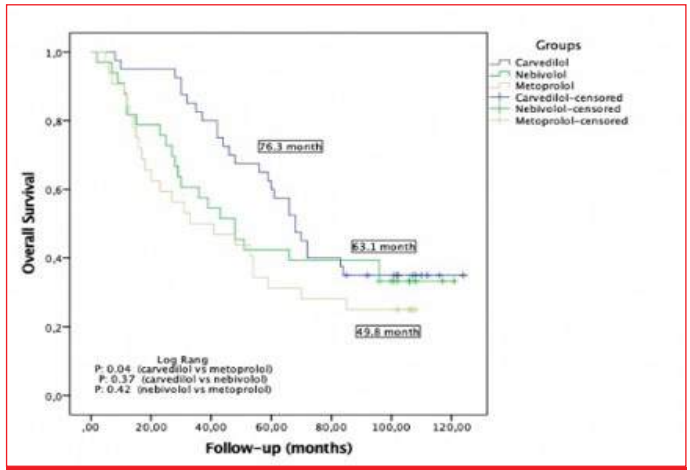

Figure 1. Kaplan-Meier Median Overall Survival curves of the different beta-blocker treatments in non-ischemic heart failure patients

PB-033 [Cardiac Insufficiency]

Effect of sodium-glucose cotransporter 2 (SGLT2) inhibitors on left ventricular volume, function and longitudinal strain in patients with heart failure and preserved ejection fraction: a prospective observational study

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Background and Aim: Sodium-glucose cotransporter 2 inhibitors (SGLT2i) lower cardiovascular events in type 2 diabetes mellitus (T2DM) patients, although the mechanisms underlying these benefits are not clearly understood. Our aim was to study the effects of SGLT2i on left ventricular volume,

function, remodelling and longitudinal strain in patients with heart failure and preserved ejection fraction.

Methods: Between March 2022 and July 2022, we included 32 patients with Tip 2 DM ≥ 18 years old, with HbA1c between 6.5 - 10.0%, and estimated glomerular filtration ≥ 45 ml/min/1.73 m2. Conventional and speckle tracking echocardiography were performed by blinded sonographers, at baseline and after 1 months of treatment.

Results: Among the 32 patients (65.6% females, mean age 57.6 ± 9.3years, mean HbA1c was 8.57 ± 2.53). Mean left ventricular EF was 55,39±8%. Mean change in left ventricular end systolic volume (LVESV) decreased significantly from 34,9 ± 13,5 to 30,3 ± 13 ml (p = 0.033) in the SGLT2i group during 1 month. Absolute value of 4th chamber Global Longitudinal Strain (4CGLS) increased from -17,65±3,8 to -18,67 ± 4,3 (p = 0.040) in the SGLT2i group during 1 month. Absolute value of Global Longitudinal Strain (GLS) increased from -17,7 ±3,4 to -18,2±3,6 (p = 0.017) in the SGLT2i group during 1 month. Absolute value of Left ventricular EF (LVEF) increased from 54,9% ±8,6 to 58,2% ± 9,2 (p = 0.04) in the SGLT2i group during 1 month. We did not find correlations between changes in 2nd chamber and 3rd chamber GLS and other variables like change in HbA1c.

Conclusions: Among patients with T2DM and heart failure with preserved ejection fraction, SGLT2i were associated with a significant reduction in LVESV, LVEF and a significant increment in 4th chamber and Global longitudinal strain measured by speckle tracking echocardiography, which may explain in part the clinical benefits found in clinical trials.

PB-034 [Cardiac Insufficiency]

Renal dysfunction determinants in advanced heart failure patients: pulmonary artery catheterization study

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Background and Aim: Heart and kidney work closely under disrupted neuro-hemodynamic regulation in entire phenotype and etiology in heart failure. Renal dysfunction is multifactorial and asserted the main hemodynamic drivers are decreased renal perfusion and increased renal venous pressure. The advanced HF patients had highly compromised end-organ perfusion, renal functions that could not be explained entirely with two hemodynamic factors. Determinants of renal dysfunction have to be elucidated. This study aimed to clarify the relationship between renal function and hemodynamic factors in advanced heart failure patients.

Methods: Between 2016-2020 hospitalized and pulmonary arterial catheterization (PAC) performed acute heart failure patients whose left ventricle ejection fraction<40% enrolled the study. Cardiac output calculated by the Fick method as central venous and arterial blood gases sampled during PAC(Table-1). Cardiac index and right atrial pressure were categorized into three groups; right atrial pressure groups were analyzed with the Kruskal Wallis test, post hoc analyses were performed with the Conover test. Values of p <0.05 were accepted statistically significant.

Results: Baseline characteristics, echocardiography and PAC results are presented in Table 2. AF is more frequent and righter ventricle systolic work related variables such as RVSWi and TRVPi was higher in renal dysfunction patients. Cardiac index and right atrial mean pressure did not significantly associate with renal dysfunction. Correlation analysis demonstrated sPAP and TRVPi inversely correlated with eGFR. Univariate lineer regression analysis revealed that age, NYHA functional class IV, smoking history, hypertension, AF, NTpro-BNP levels and using ACEi or ARB significantly associated with eGFR. sPAP, PACi and TRVPi siginificantly associated with eGFR (Table-4).In multivariable model smoking history, AF, BMI revealed negative; receiving ACEi or ARB therapy and PACi were positive related variables(Table-5). Mildly reduced CI advanced HF patients had lower eGFR(-Fig-3)Right atrial pressure classified tertiles did not show a significant difference (Fig-4).

Conclusions: Advanced HFrEF patients' prognosis decrease with the decline of renal functions. However, we could not find a relationship between eGFR and cardiac index or right atrial pressure in this study. Smoking history, atrial fibrillation, BMI had a negatively; PACi and ACEi or ARB medication usage positively correlated with the eGFR. Restoring sinus rhythm could benefit to preventing decline eGFR. Decreased PACi at baseline or negative change are prone to renal dysfunction. PACi may be useful as a prognostic marker for renal dysfunction. Moderate tricuspid regurgitation patients with higher right ventricle power are predisposed to renal dysfunction; this finding must be assessed to this patient candidate for percutaneous tricuspid valve intervention. In advanced HF patients, predictors of renal functions and therapeutic interventions have to be elucidated with future studies.

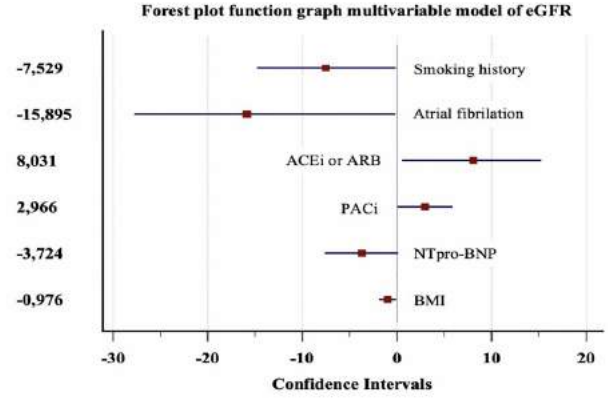

Figure 1. Forest plot function multivariable model of eGFR and B coefficient

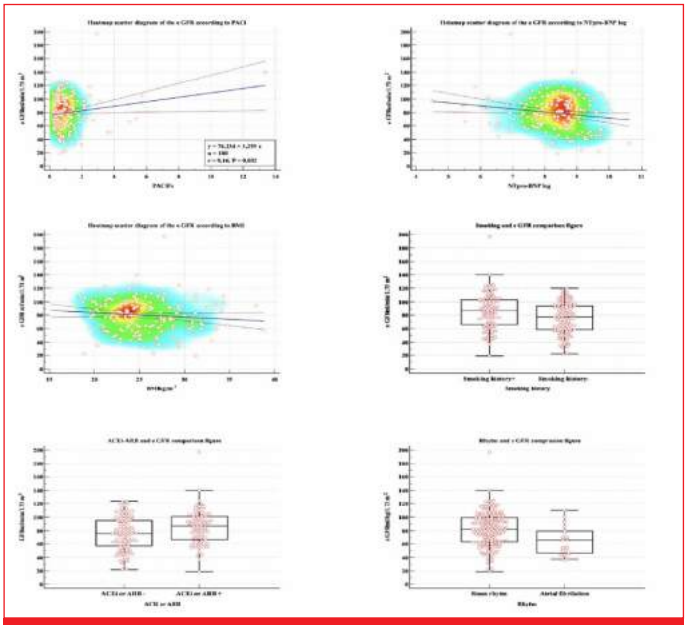

Figure 2. Graphical abstract

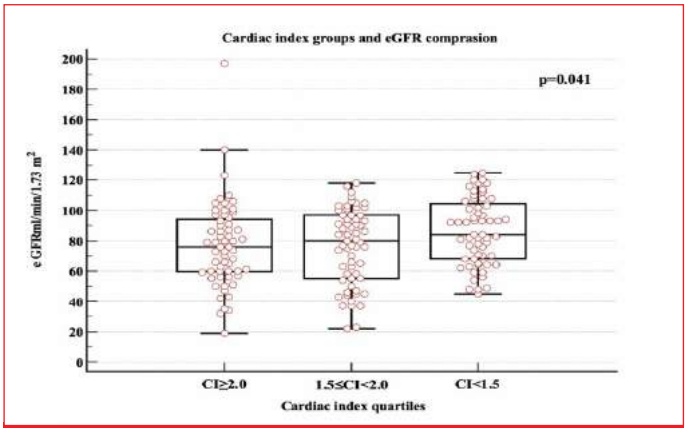

Figure 3. eGFR compared to three cardiac index tertiles

Cardiac indexes were classified into three tertiles. Cardiac index is classified as severely reduced (CI<1.5 L/m2), moderately reduced cardiac index (1.5≤CI<2.0 L/m2), and mildly reduced or normal (CI≥2.0 L/m2). Non-parametric Kruskal Wallis eGFR and cardiac index analysis showed, eGFR significantly differed according to cardiac index. Post hoc Conover analysis indicates mildly reduced cardiac index advanced heart failure patients had lower eGFR. Advanced heart failure patients with the severe and moderately reduced cardiac index did not indicate significance.

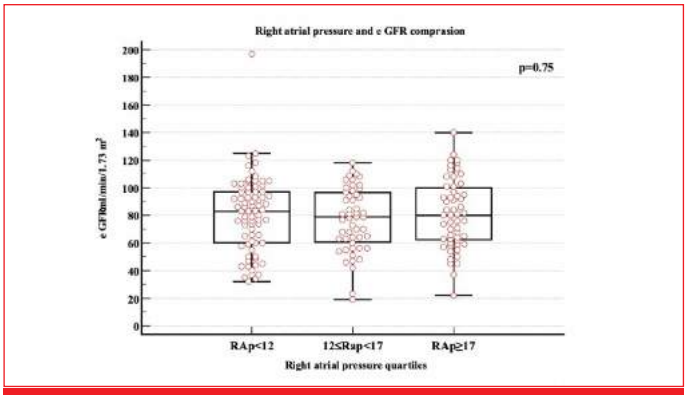

Figure 4. eGFR compared to three right atrial pressure tertiles

Right atrial pressure was classified as mildly increased or normal (Rap<12 mmHg), moderately increased (12≤Rap<17 mmHg), and severely raised (Rap≥17 mmHg). Right atrial pressure classified tertiles did not show a significant difference of eGFR.

PCWP, pulmonary capillary wedge pressure; sPAP, systolic pulmonary artery pressure; dPAP, diastolic pulmonary artery pressure; mPAP, mean pulmonary artery pressure; RVsP, right ventricle systolic pressure; RVedP, right ventricle end-diastolic pressure; RAP, right atrium pressure; SV, stroke volume; CO, cardiac output; CI cardiac index; HF / HFrEF, heart failure / heart failure with reduced ejection fraction; AF, atrial fibrillation; TPG, transpulmonary gradient; DPG, diastolic pulmonary gradient; PVR, pulmonary vascular resistance; SVR, systemic vascular resistance; PAPi, pulmonary artery pulsatility index; PACi, pulmonary artery capacitance; PAE, pulmonary arterial elastance; RVSWi, right ventricle stroke work index; TRV-poweri / TRVPi, total right ventricle power index; LVSWi, Left ventricle stroke work index; BSA, body surface area; BMI, body mass index; NYHA, FC New York Heart Association functional class; eGFR, estimated glomerular filtration rate; TDI-RVsm, tissue Doppler imaging right ventricle systolic motion; TAPSE, tricuspid annuler plane excursion; ACE-i, angiotensinogen converting enzyme inhibitör; ARB, angiotensin receptor blocker

Table 1. Pulmonary arterial catheterization equations

CO (L/min)	**VO2 [SaO2- SvO2] x Hb x 13.4**	**4-8 L/min**
SV (ml)	CO / HR x 1000	60-100 ml/beat
CI (L / min / m^2)	CO / BSA	-
SVI (ml / m^2)	CI / HR x 1000	33 – 47 ml/m^2/beat
TPG (mmHg)	mPAP - PCWP	< 12 mmHg
DPG (mmHg)	dPAP - PCWP	< 7 mmHg
PVR (Wood.unit)	TPG / CO	< 3.125 Wood.unite
SVR (dynes x sec / cm^5)	(SBP - RA) / CO	800- 1200 dynes x sec / cm^5
PP (mmHg)	sPAP - dPAP	-
PAPi	PP / RA	-
PACi (ml/mmHg/m^2)	(SV / PP) / BSA	<1.1 ml/mmHg
PAE (mmHg/ml)	sPAP / SV	-
RVSWi (mmHg.ml/ m^2)	(mPAP - RA) x (SVI) x 0.0136	5-10 mmHg.ml/m^2
Total RV-Poweri (mmHg.ml.beat/ m^2)	(mPAP x CO x 1.3) / BSA	-
LVSWi (mmHg.ml/ m^2)	(SBP - PCWP) x SVI x 0.0136	50 – 62 mmHg.ml/ m^2

Table 2A. Characteristics of patients and comparison according to eGFR

	Total	GFR≥60 ml/min/ 1.73 m²	GFR<60 ml/min/ 1.73 m²	P-value
Age (years)	50.1±12.2	48.3±12.6	56.1 ±8.4	<0.001
Male (%)	83.4	70	30	0.29
NYHA FC III (%)	20.4	83.8	16.2	0.29
NYHA FC IV (%)	79.6	75.7	24.3	
BMI (kg/m2)	25.5±4.16	25.4±4.2	25.8±4.0	0.53
BSA (m2)	1.89±0.20	1.89±0.2	1.88±0.22	0.89
Smoker (%)	53	71.9	28.1	0.062
Arterial hypertension (%)	22.1	70.0	30.0	0.20
Diabetes mellitus (%)	17.1	83.9	16.1	0.34
Dyslipidemia (%)	18.8	73.5	26.5	0.55
AF (%)	10.6	52.6	47.4	0.017
Heart rate (beat/ min)	79.8±14.7	80.3±14.8	78.1±14.3	0.38
Medical History				
ACEi or ARB (%)	45.9	83.1	16.9	0.087
Beta-blocker (%)	76.8	77	23	0.82
Aldosterone antagonist (%)	42.5	77.9	22.1	0.87
Loop diuretic (%)	71.5	76.0	24.0	0.52
HCT (%)	14.9	77.8	22.2	0.95
Positive inotrope (%)	16.6	80	20	0.70

Our study included 181 advanced heart failure with reduced ejection fraction, NYHA functional class III-IV patients who underwent pulmonary artery catheterization. Baseline characteristics, echocardiographic parameters and pulmonary artery catheterization results are presented. Overall, heart failure patients were 50.1±12.2 years old, 79.6% were NYHA functional class IV, and the mean LVEF was 20.9±3.7. The mean glomerular filtration rate was 79.8±25.4 ml/min/1.73 m2, and 22.7% of patients had eGFR was lower than 60 ml/ min/1.73 m2. Reduced renal function patients were older (56.1 ±8.4; 48.3±12.6; p<0.001, respectively), atrial fibrillation were more frequent (52.6%; 47.4%; p=0.017, respectively)

Table 2B. Echocardiographic parameters and comparison according to eGFR

	Total	GFR≥60 ml/min/ 1.73 m²	GFR<60 ml/min/ 1.73 m²	P-value
LVEF (%)	20.9±3.7	20.6±3.6	21.6+4.1	0.13
Moderate or severe mitral regurgitation (%)	73.9	77.0	23.0	0.35
Moderate or severe aortic regurgitation (%)	7.4	61.5	38.5	0.14
Moderate or severe tricuspid regurgitation (%)	64.6	75.7	24.3	0.57
TAPSE (mm)	13.4±5.4	13.7±5.6	12.5±4.7	0.23
TAPSE<16 mm (RV dysfunction) (%)	69.1	73.2	26.8	0.22
RVsm (TDI) m/sec	10.4±4.0	10.5±4.2	10.0±3.7	0.59
TRV m/sec	3.07±0.86	3.0±0.68	3.3±1.29	0.053

Table 2C. Cardiac Catheterization Variables and Comparison according to eGFR

	Total	GFR≥60 ml/min /1.73 m²	GFR<60 ml/min/ 1.73 m²	P-value
PCWP (mmHg)	25.6±8.0	25.3±8.0	26.7±7.7	0.31
sPAP (mmHg)	56.2±18.2	55.2±18.8	59.8±15.5	0.15
dPAP (mmHg)	26.1±8.2	25.6±8.5	27.8±7.3	0.14
mPAP (mmHg)	38.0±15.0	37.5±11.9	37.9±9.9	0.11
RVSP (mmHg)	55.3±17.0	54.3±17.3	58.8±15.4	0.14
RVedp (mmHg)	15.0±7.5	14.9±7.6	15.1±7.3	0.41
RA (mmHg)	13.3±6.6	13.1±6.6	13.7±6.8	0.61
SBP (mmHg)	110.5±19.7	110.4±18.9	111.0±22.5	0.86
DBP (mmHg)	68.0±18.0	67.6±9.5	69.0±14.4	0.51
MBP (mmHg)	81.1±12.1	80.9±11.1	81.8±15.1	0.71
TPG (mmHg)	12.4±6.7	12.2±6.9	13.0±6.0	0.53
DPG (mmHg)	0.55±4.4	0.4±4.1	1.0±5.3	0.39
PAPi	3.2±4.0	3.06±2.76	2.67±6.73	0.39
RA/PCWP	0.54±0.32	0.54±0.35	0.51±0.21	0.60
CO (L/min)	3.55±1.47	3.51±1.50	3.67±1.37	0.55
SV (ml)	46.0±20.8	45.3±21.1	48.6±19.8	0.36
CI (L/min/m²)	1.87±0.70	1.85±0.72	1.94±0.64	0.47
PVR (Wood. Unite)	4.0±2.65	4.05±2.79	3.85±2.12	0.67
SVR (dynes x sec / m⁵)	1700±607	1737±594	1584±641	0.19
PACi (ml/ mmHg/m²)	1.09±1.25	1.13±1.37	0.99±0.73	0.53
PAE (mmHg/ml)	1.48±0.81	1.50±0.84	1.42±.071	0.57
RVSWi (g/m/ beat/m²)	7.83±4.39	7.43±4.04	9.16±5.23	0.027
LVSWi ml/ mmHg/m²	18.8±11.2	18.8±11.3	18.7±11.2	0.70
TRVPi	89.1±36.5	86.0±34.3	99.3±42.0	0.040

Right ventricle systolic work-related variables such as RVSWi (9.16±5.23; 7.43±4.04; p=0.027, respectively) and Total RV-power index (99.3±42.0; 86.0±34.3; p=0.040, respectively) was higher in renal dysfunction patients. Cardiac index (1.85±0.72; 1.94±0.64; p=0.47, respectively) and right atrial mean pressure (13.1±6.6; 13.7±6.8; p=0.61, respectively) did not significantly associated with renal dysfunction in heart failure patients.

Table 2D. Laboratory Parameters and Comparison according to eGFR

	Total	GFR≥60 ml/min/ 1.73 m²	GFR<60 ml/min/ 1.73 m²	P-value
NTpro-BNP pg/ml (IQR)	4575 (2357-6960)	4311 (2147-6596)	5940 (3771-10979)	0.0044
Lactate (mM)	1.52±0.82	1.49±0.71	1.64±1.13	0.34

NTpro-BNP (5940; 4311; p=0.0044, respectively) was significantly higher in renal dysfunction patients.

Table 3. Correlation of Cardiac Catheterization Variables and eGFR

Cardiac catheterization variables	GFR ml/min/ 1.73 m²	
	r coefficient	p value
PCWP (mmHg)	-0.49	0.51
sPAP (mmHg)	-0.147	0.049
dPAP (mmHg)	-0.101	0.177
mPAP (mmHg)	-0.115	0.125
RVSP (mmHg)	-0.131	0.081
RVedp (mmHg)	-0.046	0.542
RA (mmHg)	-0.074	0.324
SBP (mmHg)	0.023	0.785
DBP (mmHg)	-0.069	0.408
MBP (mmHg)	-0.029	0.730
TPG (mmHg)	-0.139	0.063
DPG (mmHg)	-0.101	0.177
PAPi	-0.051	0.500
RA/PCWP	-0.037	0.617
CO (L/min)	-0.024	0.746
SV (ml)	-0.009	0.902
CI (L/min/m²)	-0.030	0.689
PVR (Wood.Unit)	-0.060	0.427
SVR (dynes x sec / m⁵)	0.063	0.451
PACi (ml/mmHg/m²)	0.160	0.032
PAE (mmHg/ml)	-0.012	0.873
RVSWi (g/m/beat/m²)	-0.137	0.068
LVSWi (ml/mmHg/m²)	0.077	0.358
TRVPi	-0.169	0.023

Cardiac index and right atrial pressure were not revealed a significant correlation with the glomerular filtration rate. Correlation analysis demonstrated systolic pulmonary arterial pressure (r -0.147, p=0.049) and total right ventricle power index (-0.169; p=0.023) inversely correlated with glomerular filtration rate. However, these correlations were weak.

Table 4A. Linear regression univariate analysis

		CI 95%	P-value
Age (years)	-0.448	-1.207 to -0.659	<0.0001
Male (%)	-0.046	-13.221 to 6.919	0.53
NYHA FC IV (%)	-0.202	-21.861 to -3.656	0.006
BMI (kg/m2)	-1.446	-1.554 to 0.240	0.150
BSA (m2)	-0.011	-19.615 to 16.794	0.879
Smoking history (%)	-10.30	-17.656 to -2.945	0.006
Arterial hypertension (%)	-9.012	-17.948 to -0.076	0.048
Diabetes mellitus (%)	9.589	-0.260 to 19.437	0.056
Dyslipidemia (%)	-8.585	-18.098 to 0.929	0.077
AF (%)	-16.512	-28.524 to -4.501	0.007
Heart rate (beat/min)	-0.022	-0.277 to 0.234	0.868
Medical History			
ACEi or ARB (%)	9.924	2.545 to 17.303	0.009
Beta-blocker (%)	1.160	-7.718 to 10.038	0.797
Aldosterone antagonist (%)	2.548	-5.024 to 10.121	0.508
Loop diuretic (%)	-5.379	-13.706 to 2.947	0.204
HCT (%)	4.114	-6.390 to 14.619	0.441
Positive inotrope (%)	2.083	-7.993 to 12.159	0.684

Table 4B. Linear regression univariate analysis

Cardiac Catheterization		CI 95%	P-value
PCWP (mmHg)	-0.156	-0.625 to 0.313	0.512
sPAP (mmHg)	-0.206	-0.412 to -0.001	0.049
dPAP (mmHg)	-0.311	-0.765 to 0.142	0.177
mPAP (mmHg)	-0.255	-0.581 to 0.071	0.125
RVsp (mmHg)	-0.916	0.417 to 0.024	0.081
RVedp (mmHg)	-0.155	-0.655 to 0.346	0.542
RA (mmHg)	-0.281	-0.841 to 0.279	0.324
SBP (mmHg)	0.029	-0.183 to 0.242	0.785
DBP (mmHg)	-0.162	-0.547 to 0.224	0.408
MBP (mmHg)	-0.061	-0.408 to 0.286	0.730
TPG (mmHg)	-0.528	-1.085 to 0.029	0.063
DPG (mmHg)	-0.583	-1.432 to 0.265	0.177
PAPi	-0.322	-1.262 to 0.618	0.500
RA/PCWP	-2.918	-14.425 to 8.589	0.617
CO (L/min)	-0.419	-2.970 to 2.232	0.746
SV (ml)	-0.011	-0.192 to 0.169	0.902
CI (L/min/m²)	-1.075	-6.367 to 4.217	0.689
PVR (Wood.Unit)	-0.573	-1.995 to 0.849	0.427
SVR (dynes x sec / m⁵)	0.003	-0.004 to 0.010	0.451
PACi (ml/mmHg/m²)	3.259	0.285 to 6.232	0.032
PAE (mmHg/ml)	-0.374	-5.005 to 4.256	0.873
RVSWi (g/m/beat/m²)	-0.795	-1.647 to 0.058	0.068
LVSWi (ml/mmHg/m²)	0.168	-0.192 to 0.529	0.358
TRVPi	-0.118	-0.220 to -0.016	0.023

A linear univariate regression analysis revealed cardiac index (beta=-1.075, p=0.689) and right atrial pressure (beta=-0.281, p=0.324) did not reach a significant relationship with the eGFR and did not include in the multivariable model.

Table 4C. Linear regression univariate analysis

Echocardiographic Parameters		CI 95%	P-value
LVEF %	-0.125	-1.131 to 0.882	0.807
Moderate or severe mitral regurgitation n (%)	-0.658	-9.589 to 8.274	0.88
Moderate or severe aortic regurgitation n (%)	-3.779	-18.424 to 10.866	0.611
Moderate or severe tricuspid regurgitation n (%)	0.388	-7.558 to 8.334	0.096
TAPSE (mm)	0.251	-0.485 to 0.986	0.502
TAPSE<16 mm (RV dysfunction) (%)	2.592	-6.901 to 11.275	0.556
RVsm (TDI) (m/sec)	0.452	-0.532 to 1.436	0.365
TRV (m/sec)	-2.347	-6.934 to 2.241	0.314
Laboratory Parameters			
NTpro-BNP pg/ml (IQR) log	-4.539	-8.509 to -0.568	0.025
Lactate (mM)	-2.078	-6.674 to 2.509	0.372

Table 5. Linear regression multivariable Backward elimination model

		CI 95%	P-value
Smoking history	-7.529	-14.890 to -0.169	0.045
Atrial fibrillation	-15.985	-27.787 to -0.184	0.008
ACEi or ARB	8.031	0.538 to 15.23	0.036
NTpro-BNP (log)	-3.724	-7.596 to 0.149	0.059
PACi	2.966	0.059 to 5.873	0.046
BMI	-0.976	-1.880 to -0.073	0.036
			R square 0.164

In multivariable model smoking history (beta= -7.529, p= 0.045), atrial fibrillation (beta= -15.985, p= 0.008) and body mass index (beta= -0.976, p= 0.036) revealed negative relation with the eGFR, ACEi or ARB therapy receiving (beta= 8.031, p= 0.036), and PACi (beta= 2.966, p= 0.046) were positive related variables with eGFR. The adjusted R square of the multivariable model was 0.133 indicates that multivariable model parameters explain only 13% of the eGFR variance.

Table 6. Tricuspid regurgitation severities and RVSWi and Total RV-power index

	Right ventricle stroke work index			Total right ventricle power index		
Tricuspid regurgitation	**B coefficient**	**95% CI**	**P-value**	**B coefficient**	**95% CI**	**P-value**
None	-0.063	-0.522 to 0.408	0.79	-0.062	-4.838 to 3.793	0.80
Mild	-0.174	-0.426 to 0.117	0.25	-0.118	-3.299 to 1.474	0.44
Moderate	-0.286	-0.285 to-0.037	0.012	-0.293	-2.483 to-0.352	0.010
Severe	0.071	-0.251 to 0.382	0.67	0.146	-1.125 to 2.827	0.38

eGFR and right ventricle strength variables; RVSWi and TRV-poweri relationship were analyzed with linear regression analyses according to tricuspid regurgitation severity. Patients split into four groups; none, mild, moderate, and severe tricuspid regurgitation. RVSWi and TRV-poweri were calculated by using; mean pulmonary artery pressure, right atrial pressure, stroke volume, and heart rate. Correlation analysis was performed to show the relationship between the variables used to calculate right ventricular strength-related parameters to intercept multicollinearity issues. None of these parameters showed a high correlation (-0.60<r correlation coefficient<0.60). Tricuspid regurgitation severity and right ventricle stroke work-right ventricle power relation with eGFR was not significantly different but eGFR was non-significantly higher in patients with moderate tricuspid regurgitation. Solely moderate tricuspid regurgitation patients eGFR had an inverse relationship with the right ventricle stroke work index (beta=-0.286 p=0.010) and total right ventricle power index (beta=-0.293 p=0.012).

PB-035 [Cardiac Insufficiency]

Whole exome sequence analysis in patients with non-ischemic dilated cardiomyopathy

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Background and Aim: Dilated cardiomyopathy (DCM) is an important health problem with high mortality and hospital admission rates in both adult and pediatric populations. DCM is a serious heart muscle disease characterized by dilatation of the left ventricle (sometimes both ventricles) and decreased left ventricular systolic function. Genetic forms of DCM account for approximately 40% of the disease creates. Although the most important known cause of DCM is ischemic heart disease, most of the patients do not have a narrowing of the coronary arteries that will cause ischemia and the etiology of the disease is still unknown. In these patients, it is possible to identify genetic mutations with new genetic sequencing techniques. In this way, early diagnosis, risk assessment and familial predispositions can be determined by genetic screening. With our study, we aimed to raise awareness about this disease, to identify new mutations associated with the disease and to evaluate its contribution to the disease.

Methods: He was diagnosed with non-ischemic dilated cardiomyopathy in the Department of Cardiology of GU, and his peripheral blood was sent to GU Genetics laboratory for the etiology of dilated cardiomyopathy. The results of genetic analysis were evaluated retrospectively by scanning the archives of the patients who underwent next-generation sequencing (NGS).

Results: As a result, only variants of uncertain clinical significance were detected in 12 (66,67%) of 18 patients, probable pathogenic variants were detected in 5 (27,77%) and pathogenic variant in 1 (5,55%). A variant not previously reported in databases was identified in one patient.

In our study, at least one variant of uncertain clinical significance (VUS) was detected in the TTN gene in 6 of 18 patients (33.33%). In 2 of 5 patients with likely pathogenic (LP) variants detected, these variants were detected in the DSP gene, and one in the LMNA, GATA4 and TTN genes. This gene was found to be the PLP gene in the only patient with a pathogen (P) variant. A variant interpreted as VUS/LP was also found in the TTN gene in this patient.

Conclusions: NIDCM is the most common cardiomyopathy with multiple causes and a clinical picture that includes heart failure and even sudden cardiac death. Therefore, genetic

screening should be made more widespread. In NIDCM patients, preventive health services can be provided to future generations by performing family screenings and determining the genetic transmission possibilities of the disease. In our study, the results of 18 patients who were diagnosed with NIDCM and whose genetic analyzes were performed were analyzed retrospectively. As a result of this examination, at least one variant of uncertain clinical significance was detected in 12 patients (66.67%).

For this reason, variant evaluations should be done more carefully by considering patient clinics as well as studies with more patients. The gene with the highest number of variants of uncertain clinical significance was determined as the TTN gene.

Table 1. Gender information of the patients included in the study

		Sayı	%
Cinsiyet	Erkek	10	55,56
	Kadın	8	44,44
	Toplam	18	100

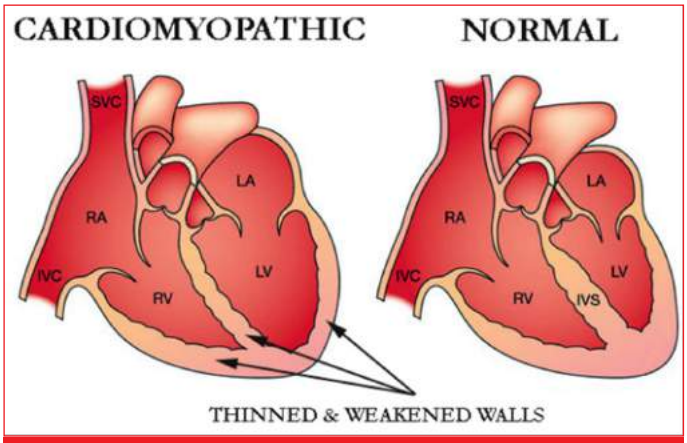

Figure 1. Normal and cardiomyopathic heart image

Table 2. Some genes associated with DCM

Fonksiyonel grup	Gen	Protein
sarkomer	*TTN*	titın
	ACTC1	α-Kardiyak aktin
	MYH7	β-Miyozin ağır zinciri
	TNNC1	Kardiyak troponin C
	TNNT2	Kardiyak troponin T
	TNNI3	Kardiyak troponin I
Nükleer zarf	*LMNA*	laminat klima
	EMD	emerin
Dezmozomlar	*DSP*	desmoplakin
	DSG2	Desmoglein 2
	DSC2	Desmokollin 2
hücre iskeleti	*DES*	Desmin
	VCL	vinkülin
	FLNC	filamin C
Z diski	*ÇANTA3*	BCL2 ile ilişkili atanojen 3
	CSRP3	Kas LIM proteini
	MYPN	miyopaldin
iyon kanalları	*SCN5A*	Sodyum kanal proteini tip 5
Sarkoplazmik retikulum	*PLN*	fosfolamban
	RYR2	Ryanodin reseptörü 2
Diğerleri	*RBM20*	RNA bağlayıcı protein 20

Table 3. NIDKM patients' genetic analysis results

Hasta no	Cinsiyet	Yaş	LV EF	Gen	Varyant	Potein	Mutasyon tipi	Zigosite	Clinvar	Franklin	Varsome	Yorum
H1	K	21	35%	DMD	c.5870G>A	p.Arg1957Gln	yanlış anlamlı	Heterozigot	YOK	VUS	LB	VUS
				DSP	c.2530C>T	p.Gln844Ter	anlamsız	Heterozigot	YOK	LP	LP	LP
				DTNA	c.2077G>A	p.Asp693Asn	yanlış anlamlı	Heterozigot	YOK	VUS	LB	VUS
				MYPN	c.1130G>A	p.Arg377Gln	yanlış anlamlı	Heterozigot	VUS/LB	VUS	VUS	VUS
				TTN	c.52477C>T	p.Pro17493Ser	anlamlı	Heterozigot	YOK	VUS	VUS	VUS
H2	E	50	25%	LDB3	c.1453G>A	p.Ala485Thr	yanlış anlamlı	Heterozigot	VUS	VUS	VUS	VUS
				TTN	c.74932G>C	p.Glu24978Gln	yanlış anlamlı	Heterozigot	YOK	VUS	VUS	VUS
H3	K	57	25%	DSP	c.7000C>T	p.Arg2334Ter	anlamsız	Heterozigot	VUS	LP	LP	LP
				DMD	c.8033G>A	p.Ser2678Asn	yanlış anlamlı	Heterozigot	YOK	VUS	VUS	VUS
				SCN10A	c.T3506A>G	p.M1169R	yanlış anlamlı	Heterozigot	YOK	VUS	VUS/LP	VUS
				TMEM43	c.155_156delCCinsTT	p.Thr52Ile	çerçeve değiştirmeyen	Heterozigot	YOK	VUS	VUS	VUS
H4	E	76	27%	MYOM1	c.64_66delinsCTC	p.V22L	çerçeve değiştirmeyen	Heterozigot	YOK	VUS	VUS	VUS
				TTN	c.3869A>C	p.Glu1290Ala	yanlış anlamlı	Heterozigot	YOK	VUS	VUS	VUS
				TTN	c.14681A>T	p.His4894Leu	yanlış anlamlı	Heterozigot	YOK	VUS	VUS	VUS
H5	E	52	33%	ALPK3	c.662C>G	p.Ala221Gly	çerçeve değiştirmeyen	Heterozigot	YOK	VUS	VUS	VUS
H6	E	42	25%	PLN	c.40_42delAGA	p.Arg14del	çerçeve değiştirmeyen	Heterozigot	P	P	P	P
				PSEN1	c.907C>G	p.Pro303Ala	yanlış anlamlı	Heterozigot	VUS	VUS	VUS	VUS
				TTN	c.44609+1_44609+2	delGTinsCA	splayzing	Heterozigot	YOK	YOK	YOK	VUS/LP
H7	E	58	25%	LMNA	c.1312G>A	p.Gly438Arg	yanlış anlamlı	Heterozigot	VUS	LP	LP	LP
H8	K	61	30%	PKP2	c.1558A>G	p.Ile520Val	yanlış anlamlı	Heterozigot	VUS	VUS	LB	VUS
				PSEN2	c.302T>C	p.Ile520Val	yanlış anlamlı	Heterozigot	YOK	VUS	VUS	VUS
H9	E	36	35%	CACNA1D	c.5107A>G	p.I1703V	yanlış anlamlı	Heterozigot	YOK	VUS	VUS	VUS
				DSP	c.2774G>A	p.Arg925Gln	yanlış anlamlı	Heterozigot	VUS	LB	VUS	VUS
H10	E	49	25%	CRYAB	c.205C>T	p.Arg69Cys	yanlış anlamlı	Heterozigot	VUS	VUS/LP	VUS	VUS
				GATA4	c.487C>T	p.P163S	yanlış anlamlı	Heterozigot	LP	LP	LP	LP
				DES	c.404C>T	p.Ala135Val	yanlış anlamlı	Heterozigot	VUS	LB	VUS	VUS
H11	K	65	30%	TNNT2	c.824G>A	p.Arg275Gln	yanlış anlamlı	Heterozigot	VUS	VUS	VUS	VUS
H12	K	72	20%	FLNC	c.3133C>A	p.His1045Asn	yanlış anlamlı	Heterozigot	VUS	VUS	VUS	VUS
H13	E	62	37%	AKAP9	c.5468A>T	p.Q1823L	yanlış anlamlı	Heterozigot	VUS	VUS	LB	VUS
H14	K	67	38%	ATP2A2	c.2653G>A	p.Val885Met	yanlış anlamlı	Heterozigot	YOK	VUS	LP	VUS
				TNNT2	c.461G>A	p.Arg154Gln	yanlış anlamlı	Heterozigot	VUS	VUS	VUS	VUS
H15	K	66	35%	AKAP9	c.623C>G	p.Thr208Ser	yanlış anlamlı	Heterozigot	YOK	VUS	VUS	VUS
				ANK2	c.10048G>T	p.Val3350Phe	yanlış anlamlı	Heterozigot	YOK	VUS	VUS	VUS
H16	K	54	40%	ANK2	c.9679A>C	p.T3227P	yanlış anlamlı	Heterozigot	VUS	VUS	VUS	VUS
H17	E	34	40%	CACNA1D	c.5414G>A	p.R1805H	yanlış anlamlı	Heterozigot	VUS	VUS	VUS	VUS
				TTN	c.68727_68728del	p.Lys22909AsnfsTer7	çerçeve kayması	Heterozigot	YOK	LP	P	LP
				TTN	c.9052A>G	p.Thr3018Ala	anlamlı	Heterozigot	YOK	VUS	VUS	VUS
H18	E	73	35%	RANGRF	c.355G>A	p.Ala119Thr	yanlış anlamlı	Heterozigot	YOK	VUS	VUS	VUS
				TTN	c.102298C>A	p.Pro34100Thr	yanlış anlamlı	Heterozigot	YOK	VUS	LP	VUS

Table 4. Patients' clinical information

Hasta no	İlk şikayet	EKG	EKO	KAG
H1	nefes darlığı, çarpıntı	sinüs ritmi, nabız 97/dk	EF: %35	normal koroner arterler
		V1-V4 derivasyonlarında T negatifliği	hafif MY-hafif TY	
		nabız 97/dk	LV boşluk artışı	
H2	nefes darlığı	sinüs taşikardisi	EF: %25	LMCA ve CX normal
		nabız 113/dk	LV boşluk artışı	LAD gövde %30, distal %40 darlık
				RCA gövde %40 darlık
H3	göğüs ağrısı	sinüs taşikardisi	EF: %25	normal koroner arterler
		nabız 100/dk	LV boşluk artışı	
			II.° MY	
H4	nefes darlığı	atriyal fibrilasyon (AF)	EF: % 27	LMCA normal,
		sol dal bloğu, nabız 90/dk	LV boşluk artışı	LAD proksimal %40, gövde %30 da
				CX %30 darlık, RCA yaygın plak
H5	göğüs ağrısı	sinüs ritmi, nabız 71/dk	EF % 33	normal koroner arterler
		anteriyor R progresyon gecikmesi	LV boşluk artışı	
		inferiyor ST depresyonu		
H6	nefes darlığı	sinüs ritmi, nabız 91/dk	EF: % 25	LMCA normal, LAD plaklı
		sol aks deviasyonu, inferiyor Q8	LV boşluk artışı	CX plaklı, RCA normal
		anteriyor R progresyon kaybı	I.° MY, I.° TY	
H7	nefes darlığı	AF, nabız 91/dk	EF: % 25	normal koroner arterler
			LV boşluk artışı	
			III.-IV.° MY , III.° TY	
H8	nefes darlığı, çarpıntı	sinüs ritmi, nabız 68/dk	EF: % 30	normal koroner arterler
		sol dal bloğu	LV sistolik fonksiyon bozukluğu	
			II.° MY	
H9	nefes darlığı	sinüs taşikardisi, nabız 100/dk	EF: %35	normal koroner arterler
		sol anteriyor hami blok	LV boşluk artışı	
H10	nefes darlığı	AF, nabız82/dk	EF: % 25	LMCA normal, LAD normal
		sol aks deviasyonu	LV boşluk artışı	Diagonal1 %40 darlık
		lateral derivasyonlarda T negatifliği	I-II.° MY	CX normal, RCA plaklı
H11	nefes darlığı	AF, nabız 85/dk	EF: % 30	normal koroner arterler (sözel)
		sol anteriyor hami blok	LV boşluk artışı	
		sol aks deviasyonu, ventriküler ekstra sist	III.° MY , III.° TY	
H12	nefes darlığı, çarpıntı	AF, nabız 85/dk	EF %20	normal koroner arterler
		sol aks deviasyonu, ventriküler ekstra sist	LV boşluk artışı	
H13	çarpıntı	sinüs bradikardisi, nabız 58/dk	EF%37	ektatik koroner arterler
		anteriyor R progresyon gecikmesi	LV boşluk artışı	
		inferiyor Q8		
H14	göğüste yanma	sinüs ritmi, nabız 96/dk	EF: % 38	normal koroner arterler
		sol dal bloğu	LV hipokinetik	
H15	çarpıntı	pil ritmi, 68/dk	EF: %35	normal koroner arterler
			LV sistolik fonksiyon bozukluğu	
H16	nefes darlığı	sinüs ritmi, nabız 89/dk	EF: % 40	normal koroner arterler
		anteriyor R progresyon kaybı	LV boyut üst sınırda, II.° TY	
		inkomplet sol dal bloğu	Sağ kalp boşluk boyutları artmış	
H17	nefes darlığı, çarpıntı	sinüs ritmi, nabız 75/dk	EF: % 40	normal koroner arterler
		lateral ve inferiyor T negatifliği	LV sistolik fonksiyon bozukluğu	
		ventriküler ekstra sistol		
H18	epigastirk ağrı	sinüs bradikardisi, nabız 51/dk	EF: % 35	normal koroner arterler
			LV sistolik fonksiyon bozukluğu	

PB-036 [Heart Failure]

Medical therapy and survival outcomes of the non-ischemic cardiomyopathy patients

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Background And Aim: This study aimed to evaluate the optimal target doses of the non-ischemic heart failure patients and its effectiveness in this patient group.

Methods: Ege University Cardiomyopathy Observational Research database were reviewed and patients with a left ventricular ejection fraction lower than 50% between 2008-2021 were included in this study. Three main medical therapy of heart failure patients, which are ACEi-ARB, beta-blockers, and mineralocorticoid receptor blockers were assessed. All medical therapies which were maximally tolerated doses were categorized as exact dose, half dose, quarter, or less.

Results: 676 patients were included in the study. 216 (32%) were female, the mean left ventricular ejection fraction was 26.5 ± 7.8 and the mean age was 42.7 ± 12.8 years. Among all patients, 78%, 92% and 75.7% were using angiotensin-converting enzyme inhibitor (ACEI)/angiotensin II receptor blocker (ARB), beta-blockers, and mineralocorticoid receptor antagonist (MRA), respectively.18.4%, %3.8 and 71% of patients reached the maximum doses of ACEI-ARB, beta-blockers and MRA's, respectively.Cox regression analysis revealed that suggested full dose and the half dose of ACEI-ARBs had survival benefits. Beta-blocker use in any dose had survival benefits, too. MRA's did not affect the survival.

Conclusions: Non-ischemic cardiomyopathy patients had benefited from ACEI-ARB and beta-blockers, and suggested exact doses had the maximum benefit for the each patient. MRA's did not affect survival rates in these patients.

Table 1. Cox regression analysis of medical therapy and the optimal doses

HR 95% CI							
	None	Exact Dose	P-value	Half Dose	P-value	Quarter or Less	P-value
ACEI-ARB	-	0.195 (0.108-0.354)	<0.0001	0.627 (0.433-0.907)	0.013	0.933 (0.681-1.277)	0.66
Beta blocker	-	0.474 (0.236-0.953)	0.036	0.299 (0.179-0.500)	<0.0001	0.607 (0.407-0.906)	0.015
MRA	-	1.074 (0.799-1.446)	0.63	0.57 (0.228-1.423)	0.22	-	-

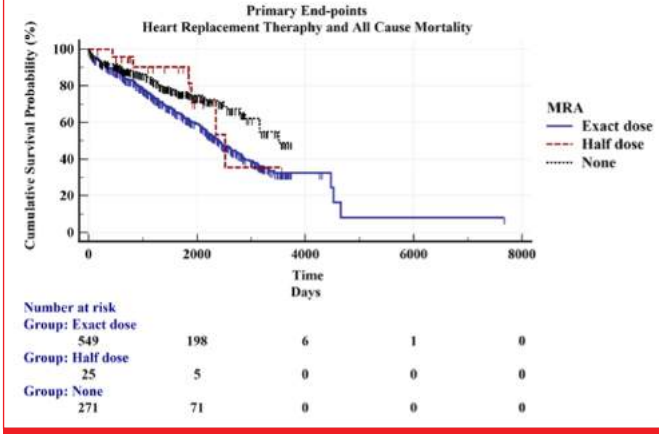

Figure 1. Time-to-event curves for heart replacement therapy or all-cause mortality

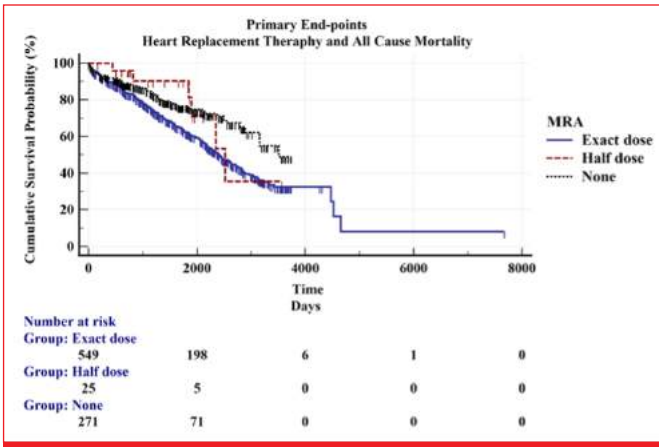

Figure 2. Time-to-event curves for heart replacement therapy or all-cause mortality

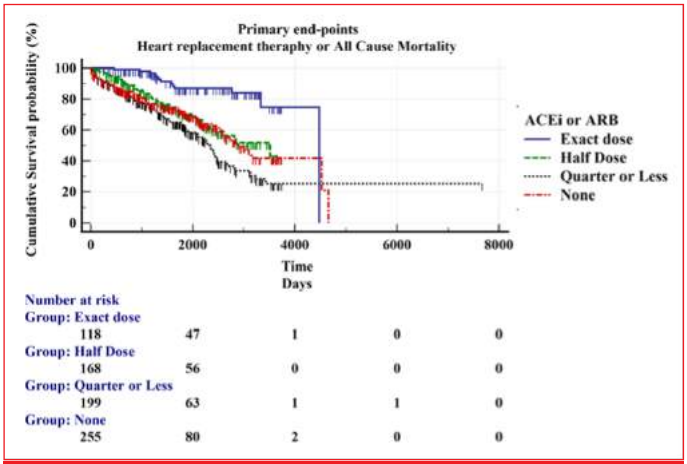

Figure 3. Time-to-event curves for heart replacement therapy or all-cause mortality

PB-037 [Heart Failure]

Non-ischemic cardiomyopathy patient's characteristics, achievement of optimal medical treatment doses, and prognosis in age tertiles. Ege University cardiomyopathy observational research age substudy

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Background and Aim: Non-ischemic cardiomyopathies were generally present in the third to a fourth decade, in early adult life, or the importance of late presentation over the fifties not well known. With the increasing comorbidities, risk factors, and frailty we aimed to evaluate the prognosis and the reaching the optimal medical therapies with increasing age.

Methods: Between 2008-2021 non-ischemic cardiomyopathy patients with systolic dysfunction (LVEF<50%) were classified as three tertiles of age. Tertile 1, 18 to 31.8 years, Tertile 2, 31.8 to 51.9 years, and Tertile 3 over 51.9 years, first heart failure diagnosis age was.

Tertiles were defined as scaled one-third of the age scale. Patients' characteristics and echocardiographic variables, and the composite primary endpoint (all-cause mortality, heart replacement therapy, ventricular arrhythmia, and heart failure-related hospitalization) free survival were evaluated.

Results: Six hundred and seventy-six patients were included in the study, the first tertile had 150, the second tertile had 344 and the third tertile had 182 patients. Non-significantly, the female proportion increased with age. Cardiovascular risk factors and comorbidities increased with the age. Atrial fibrillation significantly was more common in the third tertile (5.6%, 20.2%, 31.3, p<0.0001, respectively) and the family history (14.7%, 9.9%, 5.5%, p<0.020, respectively) was lower in tertile 3. Table 1. Echocardiographic variables were similar in the three groups presented in Table 2. The three optimal medical treatment drugs ACEi-ARB, beta-blocker, and MRA prescription rates were similar in the three tertiles. The rates of reaching target doses were similar in all three treatment groups. Kaplan Meier's survival analysis showed all three tertiles' cumulative primary end-point free survival was similar (p=0.41)

Conclusions: Late phenotypic expression of left ventricular dysfunction of cardiomyopathies' importance is unknown and questioned this debate. Cardiovascular risk factors and comorbidities were more common and so just aspirin medication was frequent with the increasing age.

With the increasing age reaching optimal medical therapy classes and targeted doses did not differ. Late expression of heart failure did not impact survival.

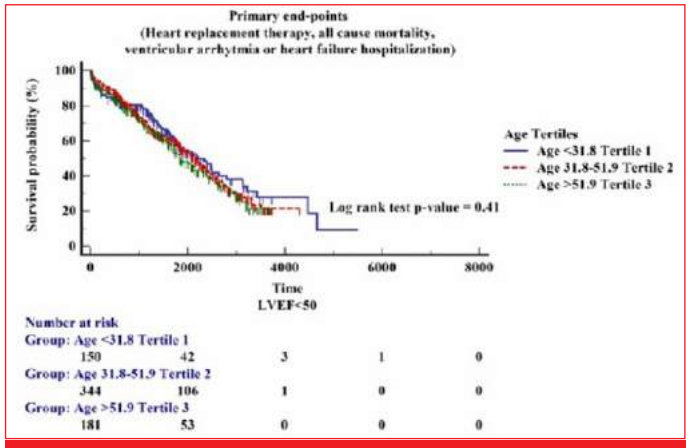

Figure 1. Time-to-event curves for all-cause mortality, heart replacement therapy, ventricular arrhymia or heart failure related hospitalization

Table 1. Characteristics of the patients with non-ischemic cardiomyopathy according to age tertiles

	Total (N:676)	Tertile-1 (N:150)	Tertile-2 (N:344)	Tertile-3 (N:182)	P-value
Age years	42.6±12	24.5±4.4	42.7±5.9	57.9±4.7	-
Female %	216 (32.0%)	44 (29.3 %)	101 (29.4%)	71 (39.0%)	0.058
NYHA FC %					0.57
I	16.5%	14.3%	16.4%	18.2%	-
II	45.8	53.3%	43.5%	43.8%	-
III	29.3%	26.7%	29.7%	30.7%	-
IV	8.4%	5.7%	10.3%	7.3%	-
Arterial hypertension n (%)	152 (22.6%)	14 (9.3%)	77 (22.4%)	61 (33.4%)	<0.0001
Diabetes mellitus n (%)	137 (20.4%)	7 (4.7%)	76 (22.2%)	54 (30%)	<0.0001
Dyslipidemia n (%)	54 (8.0%)	1 (0.7 %)	31 (9.0%)	22 (12.3%)	<0.0001
Smoking history n (%)	272 (40.7%)	38 (25.5%)	154 (44.9%)	80 (45.5%)	<0.0001
Cerebrovascular event n (%)	38 (5.7%)	2 (1.3%)	21 (6.1%)	15 (8.5%)	0.018
COPD n (%)	29 (4.4%)	1 (0,7%)	12 (3.5%)	16 (9.2%)	0.001
Renal Disease n (%)	20 (3%)	7 (4.7%)	12 (3.5%)	1 (0.6%)	0.67
AF n (%)	131 (20%)	8 (5.6%)	65 (20.2%)	55 (31.3%)	<0.0001
Family History of heart failure n (%)	66 (9.8%)	22 (14.7%)	34 (9.9%)	4 (5.5%)	0.020
ACEi or ARB n (%)	527 (78%)	111 (74%)	270 (78.5%)	146 (80.5%)	0.37
Beta blocker n (%)	627 (92.8%)	138 (92%)	320 (93%)	169 (92.9%)	0.92
Aldosterone antagonist n (%)	512 (75.7%)	104 (69.3%)	269 (78.2%)	139 (76.4%)	0.10
Loop diuretic n (%)	476 (70.4%)	90 (60%)	251 (73%)	135 (74.2%)	0.006
Anticoagulant n (%)	190 (28.11%)	36 (24%)	104 (30.2%)	50 (27.5%)	0.35
Antiplatelet n (%)	293 (43.3%)	52 (34.7%)	145 (42.2%)	96 (52.7%)	0.021
If channel blocker n (%)	139 (20.6%)	25 (16.7%)	72 (20.9%)	42 (23.1%)	0.34

*Values are mean ±SD or n (%) p<0.05.

LVEF, Left ventriculer ejection fraction; ICD, Implanted cardioverter-defibrillator; CRT-D, Cardiac resynchronization therapy-Defibrillator ACEi, Angiotensinogen converting enzyme inhibitor; ARB, Angiotensin receptor blocker; AF, Atrial fibrillation; BMI, body mass index; BSA: body surface area; COPD, Chronic obstructive pulmonary disease; NYHA FC, New York Heart Association Functional Class

Table 2. Echocardiographic parameters of the patients with non-ischemic cardiomyopathy according to age tertiles

	Total (N:676)	Tertile-1 Age<31.8 years (N:150)	Tertile-2 Age 31.8-51.9 (N:344)	Tertile-3 Age>51.9 (N:182)	P-value
LVEDd mm	63.8±10.1	61.7±10.2	65.1±10	63.0±9.8	0.91
LVESd mm	54.1±11.9	52.2±12.4	55.5±11.2	52.9±12.3	0.16
LAd mm	45.7±7.8	43.2±7.8	46.3±7.9	46.5±7.3	0.53
LVEF %	26.5±7.8	26.9±8.4	26.1±7.7	26.9±7.5	0.22
Mitral Regurgitation n (%)					0.126
Mild	290 (42.9%)	67 (44.7%)	150 (43.6%)	22 (37,1%)	
Moderate	233 (34.5%)	87 (34,9%)	114 (33.1%)	34 (48,6%)	
Severe	22 (14.7%)	36 (14,5%)	43 (13.5%)	8 (11,4%)	
Aort Regurgitation n (%)					0.022
Mild	109 (16.1%)	19 (12.7%)	47 (13.7%)	43 (23.6%)	
Moderate	15 (2.2%)	3 (2%)	5 (1.5%)	7 (3.8%)	
Severe	7 (1.0%)	1 (0.7%)	4 (1.2%)	2 (1.1%)	
Tricuspid Regurgitation n (%)					0.26
Mild	338 (50%)	75 (50%)	171 (49.7%)	92 (50.5%)	
Moderate	135 (20%)	30 (20%)	60 (17.4%)	45 (24.7%)	
Severe	52(7.7%)	15 (10%)	26 (7.6%)	11 (6%)	
TAPSE mm	17.4±5.4	16.5±5.6	17.6±5.4	17.8±5.2	0.23
RVsm (TDI) m/sec	10.6±3.29	9.7±3.0	10.9±3.4	10.9±3.0	0.60
TRV m/sec	2.88±1.01	2.82±0.56	2.85±0.75	2.98±1.52	0.35
SPAP mmHg	43.8±13.1	43.4±13.3	44.2±13.5	43.5±12.4	0.47

Values are mean ±SD or n (%) p<0.05. LVEDd, Left ventricular end diastolic diameter; LVESd, Left ventricular end systolic diameter; LAd, Left atrial diameter; LVEF, Left ventricular systolic ejection fraction; TAPSE, Tricuspid annuler plane systolic excursion; RVsm, Right ventricular systolic motion tissue doppler imaging; TRV, Tricuspid regurgitation velocity; SPAP, Systolic pulmonary artery pressure

Table 3. Heart failure medical therapy doses according to age **tertiles**

	Total (N:676)	Tertile-1 Age<31.8 years (N:150)	Tertile-2 Age 31.8-51.9 (N:344)	Tertile-3 Age>51.9 (N:182)	P-value
ACEi-ARB n (%)					0.26
Exact dose	107 (18.4%)	18 (13.7%)	55 (18.5%)	34 (21.9)	
Half dose	146 (25%)	27 (20.6%)	75 (25.3%)	44 (28.4)	
Quarter or less dose	181 (31%)	47 (35.9%)	93 (31.3%)	41 (26.5%)	
Beta-blocker n (%)					0.57
Exact dose	26 (4.3%)	6 (4.4%)	15 (5.0%)	5 (3.1%)	
Half dose	123 (20.5%)	24 (17.8%)	71 (23.4%)	28 (17.2%)	
Quarter or less dose	403 (67.1%)	93 (68.9%)	193 (63.7%)	117 (71.8%)	
Aldosterone receptor antagonist n (%)					0.25
Exact dose	480 (72.2%)	97 (65.5%)	255 (75.2%)	128 (71.9%)	
Half dose	21 (3.2%)	5 (3.4%)	9 (2.7%)	7 (3.9%)	

PB-038 [Cardiac Imaging / Echocardiography]

Endothelial functions in transgender females before and after hormone replacement therapy

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Background and Aim: There are fundamental differences in the vascular structure and function between men and women. There are also multiple differences in transsexual individuals with respect to sex-hormone levels and receptors. Polymorphisms in sex steroid receptors have been associated with transsexualism. The objective of this study was to evaluate endothelium-dependent vasodilation in female to male (FtM) transgender patients.

Methods: One hundred consecutive FtM patients (mean age: 26.8±5.4 years) attending endocrinology department were included in the study. Endothelial function was assessed by brachial artery ultrasonography before and one year after hormone replacement therapy. Flow-mediated dilation (FMD) was defined as both the maximum absolute and maximum percentage changes in the vessel diameter during reactive hyperemia.

Results: The brachial artery measures of the patients before and one year after hormone replacement therapy are listed in Table 1. Although FMD as absolute change increased significantly after hormone replacement therapy; the increase in FMD as percentage was not statistically significant.

Conclusions: Endothelial functions may be affected with hormone replacement therapy. Whether there would be more prominent change in FMD in the following years needs to be evaluated.

Table 1. Comparison of brachial artery measures before and after hormone replacement therapy

	Before therapy	One year after therapy	p
Baseline velocity (cm/s)	86.5 ± 22.8	93.9 ± 25.0	0.013
Reactive hyperemia velocity (cm/s)	164.7 ± 43.9	162.2 ± 39.1	0.642
Baseline diameter (mm)	2.94 ± 0.38	3.08 ± 0.42	0.003
Reactive hyperemia diameter (mm)	3.26 ± 0.40	3.44 ± 0.41	<0.001
FMD (absolute – mm)	0.31 ± 0.12	0.36 ± 0.16	0.029
FMD (percentage - %)	10.92 ± 4.71	12.12 ± 6.12	0.134

FMD, flow mediated dilation

Table 2. Comparison of brachial artery measures before and after hormone replacement therapy

	Before therapy	One year after therapy	p
Baseline velocity (cm/s)	86.5 ± 22.8	93.9 ± 25.0	0.013
Reactive hyperemia velocity (cm/s)	164.7 ± 43.9	162.2 ± 39.1	0.642
Baseline diameter (mm)	2.94 ± 0.38	3.08 ± 0.42	0.003
Reactive hyperemia diameter (mm)	3.26 ± 0.40	3.44 ± 0.41	<0.001
FMD (absolute – mm)	0.31 ± 0.12	0.36 ± 0.16	0.029
FMD (percentage - %)	10.92 ± 4.71	12.12 ± 6.12	0.134

PB-039 [Cardiac Imaging / Echocardiography]

Left ventricular longitudinal strain characteristics in patients with newly diagnosed type 2 diabetes mellitus

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Background and Aim: Coronary artery disease (CAD) is one of the main causes of morbidity and mortality in patients with type 2 diabetes mellitus (T2DM). Diabetic complications in the cardiovascular system randomly appear following long standing diabetes mellitus. However, newly diagnosed T2DM can also be associated with cardiac problems. Early diagnosis and treatment of T2DM can reduce the risk of long-term complications, particularly for ischemic heart disease. The degree of myocardial strain, more precisely Global Longitudinal Strain (GLS) measures the systolic function of the left ventricle (LV) and it allows early detection of systolic dysfunction. GLS has also a growing prognostic role in CAD. A cutoff value of LV GLS < | -18.8 | % is suggested for detection of coronary stenosis in patients with angina. Therefore, we aimed to analyze the LV longitudinal strain characteristics in patients with newly diagnosed T2DM.

Methods: This was a prospective cross-sectional study that included 94 patients, 52 patients with newly diagnosed diabetes mellitus that formed the first group and 42 healthy subjects, without history of diabetes mellitus and/or cardiovascular disease, which formed the control group. LV longitudinal strain data were compared between the two groups.

Results: Fifty two patients with newly diagnosed T2DM that entered our study had mean glucose level 16.37 ± 7.43 mmol/L and HbA1c of 8.57 ±2.31 %. There were no differences between the two groups concerning age or gender (55.5 ± 12.25 vs 55.5 ± 10.66, n.s.; females 22/52 (42.31%) vs 26/42 (61.9%), p=0.06). Regarding the LV longitudinal strain, several features derived from these measurements resulted with statistical significance, as presented in table 1. Most notably LV GLS resulted to be lower in patients with newly diagnosed T2DM compared to the healthy subjects (| -19.36 | % ± 2.98 vs | -20.43 | % ± 1.99. p=0.049). Of note, we analyzed the LV longitudinal strain values as absolute numbers in order to avoid confusion. Furthermore, the ratio of patients with LV GLS strain < | -18.8 | % was significantly higher in patients with newly diagnosed T2DM (42.31% vs 21.43%, p=0.03). Likewise, LV end diastolic volume and LV ejection fraction that were derived from LV longitudinal strain measurements resulted with significant statistical difference, as displayed in Table 1.

Conclusions: LV GLS may serve as an important echocardiographic parameter to detect early myocardial changes in asymptomatic patients with newly diagnosed T2DM.

Table 1. Comparison of strain rate parameters between the two groups

	Patients with Diabetes (n=52)	Control group (n=42)	P value
LV 2 chamber LS	-19.46 ± 4.38	-20.6 ± 2.76	0.15
LV 2 chamber LS < | -18.8 | %	21/52 (40.38%)	10/42 (23.81)	0.09
LV 3 chamber LS	-19.08 ± 3.17	-20.63 ± 2.15	0.008
LV 3 chamber LS< | -18.8 | %	21/52 (40.38%)	7/52 (13.46%)	0.004
LV 4 chamber LS	-19.61 ± 2.87	-20.04 ± 2.44	0.44
LV 4 chamber LS < | -18.8 | %	14/52 (26.92%)	9/42 (21.43%)	0.5
LV Global LS	-19.36 ± 2.98	-20.43 ± 1.99	0.049
LV Global LS < | -18.8 | %	22/52 (42.31%)	9/42 (21.43%)	0.03
EDV, ml	94.7 ± 23.57	81.44 ± 18.19	0.0035
EF, %	59.92 ± 5.62	63.13 ± 6.89	0.015

EDV, End Diastolic Volume; EF, Ejection Fraction; LV: Left Ventricle; LS, Longitudinal Strain

PB-040 [Cardiac Imaging / Echocardiography]

A case of gerbode ventricular septal defect confused with infective endocarditis

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Background and Aim: Communication between the left ventricle and right atrium, termed a Gerbode ventricular septal defect (VSD), was first described by Thurman and later explained with varying etiologies, including congenital and acquired forms. A thin part of the ventricular septum may be ruptured and create a direct shunt from the left ventricle into the right atrium.

Methods: Such a complication can result from cardiac surgery, endocarditis, trauma, or myocardial infarction. Here in we present a case with Gerbode VSD confused with infective endocarditis.

Results: A 76-year-old male was admitted to the cardiology outpatient clinic with shortness of breath for 3 days. The patient had atrial fibrillation, hypertension, and diabetes mellitus history. The echocardiography showed enlargement of all heart chambers, normal left ventricular ejection fraction (%50), and presence of a mobile mass suspicious vegetation view on the tricuspid valve (Figure 1). The patient has been admitted to the cardiology clinic with a preliminary diagnosis of infective endocarditis. During the follow-up, acute phase reactants did not rise. No feature was seen in the thorax CT. The complete urinalysis was unremarkable. There was no reproduction observed in blood cultures. The patient's fever has never raised in-hospital follow-up. A tee was made for further examination. In the tee performed on the patient,

mild color doppler flow was observed between the left atrium to the right ventricle (Figure 1). Vegetation was not observed on the tricuspid valve. The diagnosis of infective endocarditis is excluded. Coroner angiography was performed after 24 hour of addition as a part of ischemic work up. Coronary angiography was performed on the patient. Only luminal irregularities were detected. Left to right shunt was also shown on ventriculography (Figure 1F). After all these examinations, the patient is diagnosed with the Gerbode type VSD. The patient was discharged 3 days without any cardiac symptoms. The patient was on a regular follow-up every year.

Conclusions: Our case emphasizes the possibility of unusual İE-like echocardiographic presentations in patients with VSD, with confusion being one of them. It is important for physicians to always consider the diagnosis of Gerbobe VSD in patients presenting with IE or vice versa.

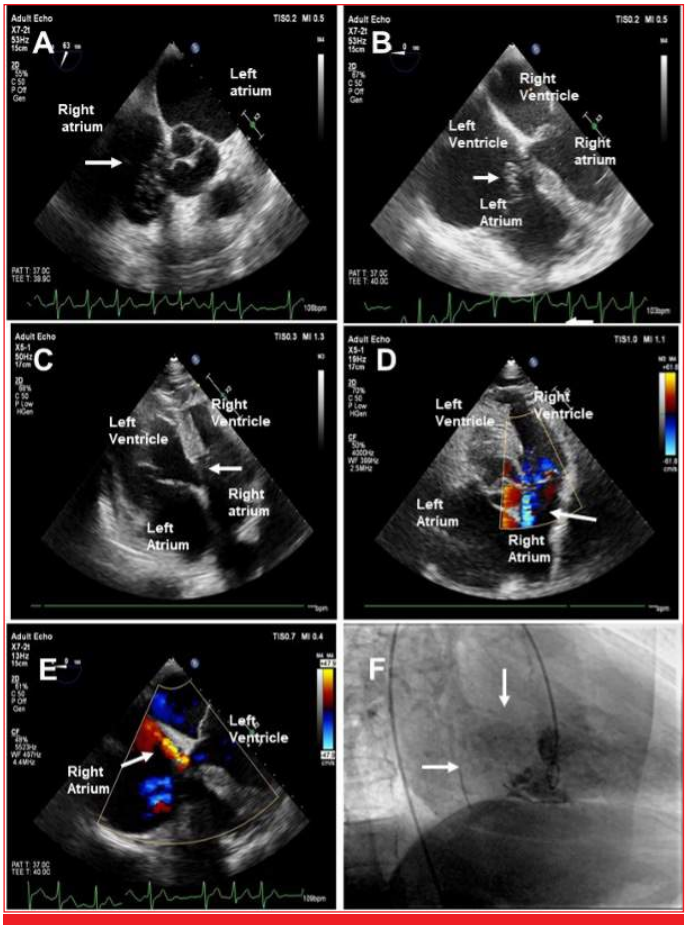

Figure 1. Gerbode type VSD. (A-B) Echocardiography view showed infective endocarditis-like images (C-D) Echocardiography revealed color doppler flow from the left ventricle to the right atrium (E) Ventriculography examination showed contrast view depicted flow for Gerbode type VSD left ventricle to the right atrium

PB-041[Cardiac Imaging / Echocardiography]

Left atrial and right atrial functions in transgender females before and after hormone replacement therapy assessed by 2D-speckle-tracking echocardiography

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Background and Aim: Previous studies have found no gender difference in strain functions unlike volumetric measurements. It is not known whether these findings can be generalized to transgender individuals as there are genetic and epigenetic differences in this subgroup. The objective of this study was to evaluate 2D speckle tracking echocardiography (STE)-derived left atrial (LA) and right atrial (RA) strain parameters in female to male (FtM) subjects before and after gender-affirming medical intervention.

Methods: One hundred consecutive FtM patients (mean age: 26.8±5.4 years) attending endocrinology department were included in the study. All patients underwent 2D STE to determine LA and and RA reservoir and conduit strain functions before and one year after hormone replacement therapy.

Results: The STE measures of the patients before and one year after hormone replacement therapy are listed in Table 1. Both LA and RA reservoir strains decreased significantly while there were not any significant changes in LA and RA conduit strains.

Conclusions: Both LA and RA reservoir functions of transgender females decreased significantly after gender-affirming medical intervention. Whether there would be ongoing decrease in LA and RA reservoir and conduit strains in the following years needs to be elucidated.

Table 1. Comparison of LA and RA functions before and after hormone replacement therapy

	Before therapy	1 year after therapy	p
LA reservoir strain (%)	33.7 ± 7.4	30.2 ± 6.7	<0.001
LA conduit strain (%)	11.4 ± 4.6	10.8 ± 4.9	0.284
LA reservoir strain (%)	40.0 ± 10.5	36.8 ± 10.9	0.025
RA conduit strain (%)	14.0 ± 5.7	13.7 ± 5.0	0.690

PB-042 [Cardiac imaging / Echocardiography]

The relationship between CONUT score and left ventricular function in patients with ischemic heart failure

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Background and Aim: In heart failure, the situation is further complicated by the underlying pathophysiology of chronic inflammation and fluid overload, which lead to nausea, loss of appetite, and early satiety resulting from gastrointestinal edema and hepatic congestion. Heart failure patients have been shown to have higher levels of total energy expenditure and hypercatabolic hormonal status, and are more likely to have negative energy and nitrogen balances compared to healthy controls, thus leading to energy-protein malnutrition. Controlling Nutritional Status (CONUT) score is a new and detailed nutritional and inflammatory score. The aim of this study is to investigate the relationship between CONUT score and left ventricular function in patients with ischemic heart failure.

Methods: A total of 113 patients were included in the study (69.4 ± 12.4 years). Patients were divided into 2 groups according to the left ventricular function (Group 1; LVEF ≤ %30, Group 2; LVEF % 30-49). Demographic, clinical, and laboratory data were collected for all patients. Cardiac evaluation with two-dimensional echocardiography were analyzed. The CONUT score was calculated using a scoring system consisting of serum albumin, lymphocytes, and total cholesterol (range, 0-12; higher = worse), Table 1.

Results: There were a significant differences between groups according to demographic, clinical and echocardiographic parameters. CONUT score was significantly higher in the Group-1 patients than Group-2 patients (Table 1).

Conclusions: In this study, we found that high CONUT score was associated with lower LVEF in ischemic heart failure patients.

Table 1. The CONUT scoring system

Parameters	Normal	Light	Moderate	Severe
Serum albumin (g/L)	≥ 35	30 – 34.9	25 – 29.9	< 25
Score	0	2	4	6
Total lymphocyte count (10^3/μL)	≥ 1.6	1.2 – 1.59	0.8 – 1.19	< 0.8
Score	0	1	2	3
Total cholesterol (mg/dL)	>180	140 - 180	100 - 139	< 100
Score	0	1	2	3
CONUT score (Total)	0-1	2-4	5-8	9-12
Assessment	Normal	Light	Moderate	Severe

Table 2. Clinical characteristics of patients

Variables	LVEF (<%30) n=32	LVEF (%30-49) n=43	P value
Age (years)	67.8±14	59±12.4	0.034
Gender (F/M)	13/19	18/25	>0.05
Heart rate (beats/min)	97.5±15.7	84.1±12.5	0.025
Hypertension	10 (34%)	15 (36%)	0.042
Diabetes Mellitus	8 (26%)	12(29%)	0.072
Smoking	15 (47%)	19 (45%)	0.081
BMI (kg/m2)	29.3±6.4	28.4±5.7	0.065
Hyperlipidemia	11 (36%)	16 (39%)	0.079
Hemoglobin (g/dL)	10.8±3.1	12.1±2.8	0.046
Glucose (mg/dL)	161±69	145±53	0.052
Creatinine (mg/dl)	2.4±1.7	1.6±0.7	0.029
eGFR	46.3±14.2	57.5±12.4	0.039
Lymphocyte count, (103/μL)	1.1±0.5	1.9±0.7	0.028
Platelet count, (103/μL)	205±97	256±75	>0.05
Total protein (g/L)	71±13	82±9	<0.05
Albumin (g/L)	28±5	39±6	<0.05
Total cholesterol (mg/dL)	147.6±35.2	215.9±42.7	<0.05
LDL cholesterol (mg/dL)	78.4±17.6	104.9±26.5	<0.05
HDL cholesterol (mg/dL)	33±5.1	35.2±3.8	>0.05
Triglyceride (mg/dL)	104.3±14.2	138±23.5	>0.05
CONUT score	8	3	0.039

PB-043 [Cardiac imaging / Echocardiography]

Significance of mitral early diastolic inflow velocity to left atrial strain ratio in patients with Acute Coronary Syndrome (AYISIT-ACS study)

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Background and Aim: The ratio of mitral early-diastolic inflow peak velocity (E) to left atrial strain (E/LASr) is a novel parameter of the left ventricle diastolic dysfunction.It was shown that E/LASr was a significant predictor of cardiac outcomes in patients with heart failure. This study aims to evaluate the predictive value of E/LASr for death and hospitalisation due to heart failure following acute coronary syndrome (ACS).

Methods: In total, 232 ACS patients underwent echocardiography following percutaneous coronary intervention. The study endpoints were hospitalisation and mortality because of heart failure (HF).

Results: Mean follow-up was 13 months. During the follow-up period, thirteen patients died and twenty-two patients were hospitalised because of HF. The receiver operating characteristics curve indicated that E/LASr >2,8 predicted these events (logrank, p<0.001).

Conclusions: In conclusion, E/LASr is available as a new echo index which can predict mortality and rehospitalisation because of HF in patients with ACS.

Table 1.

	End point absence(n:197)	End point presence(n:35)	P-values
	Mean±SD or %	Mean±SD or %	
Age, years	64.4±12.6	68.3±12.5	0.291
Male gender, %	79	53	0.275
Hypertension, %	64	81	0.204
Hyperlipidemia, %	49	52	0.795
Smoker, %	53	58	0.851
Diabetes mellitus, %	46	50	0.186
PCI within 24 hours, %	76	60	0.203
Antiagregant	33	35	0.740
ACE inhibitors	24	38	0.195
ARB	15	18	0.346
Beta-blockers	38	39	0.452
LV ejection fraction, %	54.6±12.3	42.1±11.2	**<0.005**
Mitral E velocity, cm/s	74.3±17.6	72.9±18.4	0.361
Mitral A velocity, cm/s	83.5±19.9	89.6±24.1	0.110
Mitral E/A ratio	0.9±0.3	0.8±0.2	0.090
DT, ms	241.9±58.6	220.1±62.8	0.296
LASr, %	32.8±1.8	21.1±1.3	**<0.005**
Peak E' velocity, cm/s	9.4±2.0	8.5±1.9	0.088
Peak A' velocity, cm/s	11.5±2.4	11.1±2.6	0.101
E/E' ratio	8.1±2.3	8.2±2.5	0.851
E/LASr ratio	2.1±0.3	2.7±0.5	**<0.005**

Table 2.

	E/LASr		P values
	2.8>(n:60)	2.8<(n:172)	
	Group A	Group B	
Age, years	67.8±11.9	63.7±12.4	0.05
Male gender, %	52	70	<0.01
BSA, m2	1.7±0.3	1.7±0.3	0.48
Systolic BP, mm Hg	132.6±32.8	129.5±25.6	0.65
Diastolic BP, mm Hg	69.4±18.6	70.3±18.0	0.54
Hypertension, %	76	66	0.09
Hyperlipidemia, %	55	47	0.21
Smoking, %	41	49	0.23
Diabetes mellitus, %	60	42	0.15
Prior MI, %	15	15	0.85

Table 3.

	E/LASr>		P values
	2.8>(n:60)	2.8<(n:172)	
	Group A	Group B	
LV ejection fraction, %	44.1±13.6	54.6±11.5	**<0.01**
LV end-diastolic dimension, mm	48.7±9.8	44.3±5.3	**<0.01**
LV end-systolic dimension, mm	33.8±8.2	32.6±8.6	0.43
Interventricular septum dimension, mm	12.2±2.6	11.9±2.4	0.13
LV posterior wall dimension, mm	12.3±2.8	11.9±2.4	0.21
LASr	24.3±1.7	32.6±2.0	**<0.01**
Mitral E velocity, cm/s	76.9±21.9	72.4±17.3	0.20
Mitral A velocity, cm/s	85.9±21.7	82.8±19.9	0.09
E/A ratio	0.9±0.3	0.9±0.3	0.88
DT, ms	219±51	241±60	0.23
Peak E', cm/s	8.2±2.0	9.4±1.9	**<0.01**
Peak A', cm/s	11.1±2.2	13.3±2.7	**<0.01**
E/E' ratio	10.1±3.9	8.0±2.2	**<0.01**
E/LASr ratio	3.3±0.5	2.0±0.2	**<0.01**

PB-044 [Cardiac imaging / Echocardiography]

The role of three dimensional transeosephageal echocardiography in predicting the effect of cardiac resynchronization therapy on mitral regurgitation in patients with low ejection fraction heart failure

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Background and Aim: Cardiac resynchronization therapy (CRT) positively affects the improvement of functional mitral regurgitation in patients with heart failure with reduced ejection fraction. However, geometric changes in the mitral valve apparatus, subvalvular structures and their contribution to improving mitral regurgitation after CRT have not been clearly defined. Our study aimed to evaluate the geometric parameters of mitral valve apparatus measured with three-dimensional (3D) transesophageal echocardiography (TEE) before CRT implantation and to determine the parameters predicting the improvement of mitral regurgitation after CRT.

Methods: In this prospective study, we included thirty patients with moderate or severe mitral regurgitation and heart failure with low EF planned for CRT implantation who had an indication for TEE. Before CRT implantation, effective regurgitant orifice (ERO) and regurgitant volume (RV) measurements were performed. Detailed quantitative measurements of the mitral valve were done from recorded images by 3D TEE.(Figure1) ERO, RV measurements were repeated to evaluate mitral regurgitation at the end of 3rd month.

Results: There were no significant changes in left ventricular EF and left ventricular diameters at third-month follow-up, whereas ERO and RV values were decreased. The posterior leaflet angle was higher in the non-responder group than the responder group (28.93 ± 8.41 vs 41.25 ± 10.90, p = 0.006). (Figure2) The posterior leaflet angle was an independent predictor of decreased RV and ERO. (Table1)

Conclusions: Among heart failure patients with moderate or severe functional mitral regurgitation who underwent CRT implantation had a lower posterior leaflet angle, which was measured by 3D TEE, in the patient group whose mitral regurgitation improved after CRT

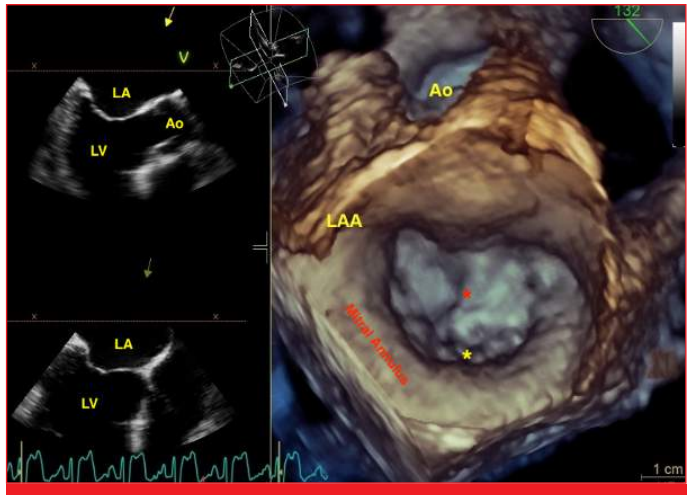

Figure 1.

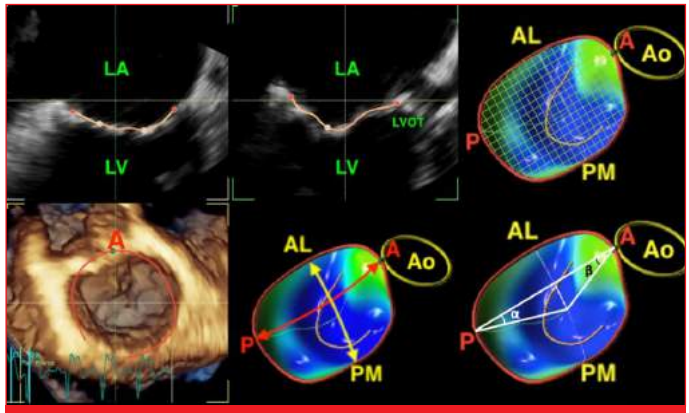

Figure 2.

Table 1.

	Responder	Non-responder	P value
Parameter	**Median Interquartile Range (min – max)**	**Median Interquartile Range (min – max)**	**P**
Mitral annulus area	8,8(6,9-18,7) 4,9	9,0(6,2-10,2) 2,9	0,320
Posteromedial – anterolateral diameter	3,4(2,5-4,6) 0,55	3,3(2,4-4,2) 0,60	0,534
Antero-posterior diameter	3,1(2,2-4,4) 1,03	3,0(2,5-3,4) 0,35	0,597
Anterior leaflet angle	23(13-50) 8,75	23(18-27) 4,75	0,872
Posterior leaflet angle	36(13-60) 18,25	29(19-36) 14,50	0,050**
Tenting Height	0,9(0,5-1,3) 0,2	0,7(0,3-0,9) 0,4	0,070
Interpapiller distance	2,2(1,8-2,9) 0,55	2,1(1,9-2,7) 0,53	0,420

PB-045 [Cardiac imaging / Echocardiography]

Left and right ventricular functions in transgender females before and after hormone replacement therapy assessed by 2D speckle-tracking echocardiography

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Background and Aim: Previous studies have found no clinically relevant gender difference in strain functions unlike volumetric measurements. It is not known whether left ventricular (LV) and right ventricular (RV) strain parameters are affected in transgender individuals. The objective of the present study was to evaluate 2D speckle tracking echocardiography (STE)-derived LV and RV strain parameters in female to male (FtM) subjects before and after gender-affirming medical intervention.

Methods: One hundred consecutive FtM patients (mean age: 26.8±5.4 years) attending endocrinology department were included in the study. All patients underwent conventional echocardiography and 2D STE to determine LV and and RV functions before and one year after hormone replacement therapy.

Results: The conventional echocardiographic and STE measures of the patients before and after hormone replacement therapy are listed in Table 1. While LV ejection fraction did not change significantly; LV global longitudinal strain (GLS) decreased significantly with therapy. Similarly, both tricuspid lateral annular systolic velocity and RV GLS decreased significantly one year after the initiation of hormone replacement therapy.

Conclusions: LV and RV GLS decreased after androgenic hormone-replacement therapy. Whether there would be ongoing decrease in LV and RV GLS in the following years needs to be elucidated.

Table 1. Comparison of echocardiographic parameters before and after hormone replacement therapy

	Before therapy	1 year after therapy	p
LVD (mm)	41.9 ± 3.9	43.2 ± 3.9	0.008
LVS (mm)	25.7 ± 3.9	27.1 ± 4.1	0.006
IVS (mm)	7.7 ± 1.2	8.4 ± 1.3	<0.001
PW (mm)	7.5 ± 1.2	8.2 ± 1.2	<0.001
LVEF (%)	67.2 ± 7.4	66.1 ± 8.7	0.315
LV GLS (-%)	20.6 ± 2.4	19.7 ± 1.7	0.002
E/E'	5.2 ± 1.2	5.6 ± 1.3	0.020
RV fractional area change (%)	49.2 ± 9.8	49.3 ± 10.7	0.946
RVS (cm/s)	12.9 ± 1.8	12.2 ± 1.7	0.002
TAPSE (mm)	24.6 ± 4.0	24.3 ± 3.7	0.572
RV GLS (%)	22.0 ± 3.5	20.9 ± 3.0	0.017

PB-046 [Cardiac imaging / Echocardiography]

Left ventricular rotationonal and ventricular strain parameters in mitral stenosis

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Background and Aim: Rheumatic involvement also occurs on the ventricular myocardium along with the valve structure and atrial tissue. Conflicting results were found in studies evaluating the effect of rheumatic involvement, especially on cardiac torsion. In our study, we investigated the effects of myocardial fibers that provide longitudinal, circumferential, radial and twist movements in patients with moderate and mild rheumatic mitral stenosis by measured Speckle tracking echocardiography.

Methods: 81 patients (age 38.1±12, female n: 49 (61%) (43 moderate mitral stenosis, 38 mild mitral stenosis) and 60 healthy, sex and age matched patients in sinus rhythm were included in the study. All patients' 2D echocardiographic measurements and strain-based GLPS, circumferential, radial, basal, apical torsion and twist measurements were performed using speckle tracking echocardiography Control group and groups with mild and moderate mitral stenosis were compared.

Results: Compared with the control group, GLPS, circumferential and radial strain and basal rotation were significantly higher in the patient group, while no significant difference was observed between apical rotation and twist. In moderate mitral stenosis compared with mild mitral stenosis, Left ventricular ejection fraction, left atrial diameter, left atrial volume index (56.3±14.7 vs. 54.7 ± 15.3, p= 0.6), radial strain (39.6±10.2 vs. 37±7.2, p= 0.2), circumferential strain (14.9 ± 2.2 vs. 14.9 ±2.4, p= 0.9), apical rotation (11.4 ± 3.4 vs. 12.3± 4.3, p= 0.3) and twist (16.8 ± 3.8 vs. 16.6 ± 4.9, p= 0.8) was similar, while basal rotation (5.4± 1.5 vs. 4.3 ± 1.6 p= 0.003), and GLPS (18.6 ± 2.9 vs. 16.6 ± 4.9, p= 0.003) was found to be significantly lower. There was a moderate correlation between the severity of the mitral valve area and basal rotation (r: 0.42, p = < 001), but there was no significant difference between GLPS and apical rotation.

Conclusions: Basal rotation and GLPS were found to be significantly lower in patients with rheumatic mitral stenosis. The rheumatic process especially affects the fibers in the longitudinal and basal parts of the heart. Basal rotation, or GLPS may provide additional information when clinical and echocardiographic conflicts exist, or on optimal operative timing. Studies on these topics are needed.

PB-047 [Coronary Artery Disease / Acute Coronary Syndrome]

High triglyceride-glucose index is associated with poor prognosis in patients with STEMI in long term follow-up

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Background and Aim: Insulin resistance(IR) is the cornerstone in the development of the metabolic syndrome and it has been shown that IR plays an important role in the development and progression of cardiovascular diseases. Calculating IR directly from the HOMA-IR formula is uncommon and complex, and its use in clinical practice is difficult. Instead, the triglyceride-glucose (TyG), index which is calculated from easily accessible triglyceride and fasting glucose levels, is an indirect indicator of IR. TyG index was found to be correlated with HbA1c and HOMA-IR in detecting IR. TyG index is calculated as following formula: Ln [fasting TGs (mg/dL) ×FPG (mg/dL)/2]. This study aims to evaluate the association between the TyG index and MACE independently from other risk factors in patients with the STEMI in long-term follow up.

Methods: We included 646 patients with the diagnosis of STEMI between January 2017 to 2022. All together with the fasting blood glucose and triglyceride, other biochemical variables were measured. The patients were divided into 4 quartiles according to TyG index levels, Q1 (n=180, TyG index≤8.44), Q2 (n=149, 8.45≤TyG index≤8.89), Q3 (n=150, 8.90≤TyG index≤9.42), and Q4 (n=167, TyG index≥9.43). The association between major adverse cardiac events(MACE) that developed in-hospital and at 60-month follow-up and TyG index were compared. Long-term major adverse cardiac events (MACE) were defined as mortality, re-infarction, and target vessel revascularization.

Results: 501(%77,6) of patients were male and 145(%22,4) female. The average age of patients was 61,9±15,0. 121 MACE was observed in 12(1-59) months follow-up. The demographic and clinical characteristics of patients are listed in table 1. A significant increase was found in the MACE ratio compared to quartiles with increased TyG index (p<0.001) (Table 2). Age, presence of DM, presence of CAD, presence of CHF, WBC, hemoglobin, neutrophil, lymphocyte, TyG (Figure 1B), creatinine and CRP levels were determined as potential risk factors associated with MACE. In the multivariate regression model, which included the potential risk factors associated with MACE; age (HR:1.036; p<0.001), WBC (HR:1.089; p<0.001), neutrophil (HR:1.025; p=0.001) and TyG index Q3 (HR:1.87; p=0.044), Q4 (HR:2.918; p=0.002) and CRP level (HR:1.010; p=0.002) were determined as independent risk factors for MACE.

Conclusions: This study demonstrated that MACE rates were higher in STEMI patients with a high TyG index at a mean follow-up of 60 months. In addition, the ROC curve showed that the TyG index had a high predictive value for MACEs in STEMI patients. Adding the TyG index to clinical practice, will provide useful information in predicting clinical outcomes in patients with the STEMI. These findings suggested that TyG index can be used to predict prognosis after STEMI independently from diabetic status.

Table 1. Demographic and clinical characteristics of STEMI patients

Variables	STEMI n=646
Male	501(77,6)
Female	145(22,4)
Age, years	61,9±15,0
Cigarette, n(%)	233(36,1)
Comorbidities, n(%)	
Hypertension	258(39,9)
Diabetes mellitus	180(27,9)
CHD history	68(10,5)
Hyperlipidemia	253(39,2)

Table 1. Demographic and clinical characteristics of STEMI patients (Continued)

Variables	STEMI n=646
Treatment, n(%), PCI	610(94,4)
CABG	21(3,3)
Medical treatment	15(2,3)
Laboratory findings, WBC(10^3 µ/L)	11,2(2,9-25,4)
Hemoglobin(g/dL)	14±1,8
Fasting blood glucose(mg/dl)	112(52-586)
Triglyceride(mg/dl)	123(25-1251)
TyG index	9,0±0,8
Total cholesterol(mg/dl)	192(63-408)
LDL(mg/dl)	116,2(38-262)
HDL(mg/dl)	40,5±10,1
HbA1c	6,6±1,7
Creatinine(mg/dl)	0,9(0,4-3,7)
CRP(mg/dl)	3,7(0-189)
MACE, n(%)	121(18,7)
EX	77(11,9)
TVR	38(5,9)
RMI	34(5,3)
CVE	1(0,2)
Follow-up time, month	12(1-59)

CHD: Chronic heart disease, CHF: Chronic heart failure, CRF: Chronic renal failure, PCI: Percutan coronary intervention, CABG: Coronary artery bypass grefting, WBC: White blood cell, HDL: High density lipoprotein, LDL: Low density lipoprotein, CRP: C-reactive protein, TVR: Target vessel revascularisation, RMI: Re-myocardial infarction, CVE: Cerebrovascular event, CIN: Contrast induced nephropathy. Numerical variables were presented as mean±standard deviation or median (min-max), and categorical variables as numbers (%). Bold characters differ significantly.

Table 2. Distribution of demographic and clinical features according to TyG index in STEMI patients

Variables	Q1 ≤8,44 n=180	Q2 8,45-8,89 n=149	Q3 8,90-9,42 n=150	Q4 ≥9,43 n=167	p
Male	144(80,0)	115(77,2)	117(78,0)	125(74,9)	0,718
Female	36(20,0)	34(22,8)	33(22,0)	42(25,1)	0,718
Age, years	64,3±14,9	64,3±13,5	61,4±15,8	57,7±15,1	<0,001*
Smoking, n(%)	58(32,2)	53(35,6)	58(38,7)	64(38,3)	0,487
Hypertension	68(37,8)	54(36,2)	55(36,7)	81(48,5)	0,075
Diabetes mellitus	29(16,1)	35(23,5)	29(19,3)	87(52,1)	<0,001*
CHD	22(12,2)	14(9,4)	13(8,7)	19(11,4)	0,703
Hyperlipidemia	76(42,2)	65(43,6)	61(40,7)	51(30,5)	0,063
Treatment, n(%)					
PCI	174(96,6	140(94,0)	145(96,7)	151(90,4)	0,061
CABG	2(1,1)	5(3,4)	2(1,3)	12(7,2)	0,061
Medical treatment	4(2,2)	4(2,7)	3(2,0)	4(2,4)	0,061
WBC(10^3 µ/L)	10,1(2,9-20,5)	10,6(4-21,4)	11,3(4,2-19,5)	12,6(5,7-25,4)	<0,001*

Table 2. Distribution of demographic and clinical features according to TyG index in STEMI patients (Continued)

Variables	Q1 ≤8,44 n=180	Q2 8,45-8,89 n=149	Q3 8,90-9,42 n=150	Q4 ≥9,43 n=167	p
Hemoglobin (g/dL)	13,8±1,7	14,1±1,7	14,3±1,7	14,0±1,9	0,190
FBG(mg/dl)	99(52-175)	105(57-370)	111(80-268)	167(88-586)	<0,001*
Triglyceride (mg/dl)	67,5(25-178)	113(39-251)	161(74-247)	239(64-1251)	<0,001*
TyG index	8,1±0,3	8,7±0,1	9,1±0,1	10,0±0,6	<0,001*
Total cholesterol (mg/dl)	174(98-337)	185(63-343)	193(92-360)	210(103-408)	<0,001*
LDL(mg/dl)	108(38-262)	117,8(42-226)	118(38,6-248)	120(40-231)	0,065
HDL(mg/dl)	44,7±10	43,5±9,4	39,0±10,9	37,7±8,2	<0,001*
HbA1c	6,0±1,0	6,1±0,9	6,4±1,3	8,2±2,3	<0,001*
Creatinine (mg/dl)	0,9(0,4-2,5)	0,9(0,4-3,2)	1,0(0,6-2,2)	0,9(0,4-3,7)	0,843
CRP(mg/dl)	4,3(0-122)	3,8(0-189)	3,5(0-144)	3,3(0-95)	0,352
MACE, n(%)	18(10,0)	20(13,4)	25(16,7)	58(34,7)	<0,001*
EX	9(5,0)	14(9,4)	19(12,7)	35(21,0)	<0,001*
TVR	8(4,4)	3(2,0)	8(5,3)	19(11,4)	0,006*
RMI	5(2,8)	6(4,0)	5(3,3)	18(10,8)	0,008*
CVE	0	0	0	1(0,6)	0,720
Follow-up time, month	35,5(1-59)	16(1-59)	12(1-59)	12(1-58)	<0,001*

CHD: Chronic heart disease, CHF: Chronic heart failure, CRF: Chronic renal failure, PCI: Percutan coronary intervention, CABG: Coronary artery bypass grefting, WBC: White blood cell, FBG: Fasting blood glucose, HDL: High density lipoprotein, LDL: Low density lipoprotein, CRP: C-reactive protein, TVR: Target vessel revascularisation, RMI: Re-myocardial infarction, CVE: Cerebrovascular event, CIN: Contrast induced nephropathy. Numerical variables were presented as mean±standard deviation or median (min-max), and categorical variables as numbers (%). Bold characters differ significantly. *P<0.05 indicates statistical significance.

Table 3. Independent predictors of MACE in STEMI patients

Variables	HR	95% CI	p
Age, years	1,036	1,022-1,051	<0.001*
WBC	1,089	1,042-1,139	<0.001*
Neutrophil	1,025	1,011-1,039	<0.001*
Lymphocyte	0,652	0,525-0,810	<0.001*
TyG index,Q1	ref		
Q2	1,401	0,737-2,663	0.304
Q3	1,870	1,016-3,449	0.044*
Q4	2,918	1,578-5,396	0.001*
CRP	1,010	1,004-1,016	0.002*

-2 Log Likelihood=1329,1; p<0.001*

PB-048 [Coronary Artery Disease / Acute Coronary Syndrome]

The correlation of triglyceride / HDL ratio with collateral index in patients with coronary chronic total occlusion

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Background and Aim: As a new atherogenic index, the association of triglyceride to HDL ratio (TG / HDL)with insulin resistance and its significance in risk and prognostic evaluation of cardiovascular diseases have been shown in several studies. In case of TIMI 0 flow in one of the coronary arteries after 3 months or more of complete occlusion, chronic total occlusion (CTO) is mentioned. Coronary collateral circulation (CCD) angiogenesis and narrowing of the coronary arteries Expansion of pre-existing collateral vessels due to the pressure difference before and after result occurs. Coronary collateral development (CHG) is examined in coronary angiography (CAG) according to the Cohen-Rentrop method is evaluated. In this study, we investigated the relationship between the TG/HDL ratio and the development of coronary collateral circulation (CCD).

Methods: This retrospective cross-sectional study was performed in coronary angiography series between 2019-2021 hospital records of patients with chronic total were reviewed. Coronary angiography of 243 patients included in the study pre; triglyceride levels, HDL levels, triglyceride / HDL ratios were measured. Collateral circulation is Rentrop. evaluated according to collateral classification. Rentrop grades 0 and 1 are weak collateral, grades 2 and 3 are good grouped as collateral. The patients' routine blood tests, clinical risk factors, coronary collateral were documented together with the circulatory class and atherogenic plasma indices. Good with weak collateral patient group TG/HDL ratio was compared in collateral advanced patient groups.

Results: There was no significant difference between the two groups in terms of basic clinical and laboratory findings. TG/HDL ratio is poor was higher in the collateral developing coronary artery patient group 6.14±4.31 vs. 3.85±2.61, <.001* According to this a low atherogenic plasma index (AIP) is an independent predictor of good collateral artery development. detected.

Conclusions: High TG/HDL ratio is an independent cardiometabolic marker associated with poor collateral development we revealed.

Table 1. Comparison of basic and laboratory parameters between the weak collateral developing group and the well collateral developing group

	poor collateral group (n: 88)	well collateral group (n:155)	p value
Age (years)	61,51± 10,91	64,45± 9,97	0,034
Sex(n,%) males	80(%90,9)	130(%83,9)	0,172
Hypertension n(%)	63 (% 71,6)	97 (%62,6)	0,163
Diabetes mellitus n (%)	37(%42)	44(%28,4)	0,034
Hyperlipidemia n (%)	38(43,2)	54(34,8)	0,217
Smoking n(%)	35(39,8)	44(28,4)	0,087
Fasting glucose (mg/dl)	163,47±89,39	140,29±59,13	0,016
Creatinine (mg/dl)	1,25±1,22	1,04±0,48	0,059
AST (U/L)	40,64±53,41	33,74±36,34	0,234
ALT(U/L)	28,05±35,69	27,98±21,67	0,984
Fasting HDL cholesterol (mg/dl)	40,70±16,17	43,48±10,09	0,099
Fasting triglyceride(mg/dl)	229,48±162,65	156,34±92,12	<.001*
Hemoglobin (g/dl)	13,87±1,79	13,85±1,91	0,954
Platelet (103/mm3)	244,61±80,9	239,98±76,52	0,657
Fasting triglyceride/ Fasting HDL cholesterol	6,14±4,31	3,85±2,61	<.001*
Left ventricular ejection fraction(%)	47,05 ±10,39	50,10±10,16	0,028

PB-050 [Coronary Artery Disease / Acute Coronary Syndrome]

Relationship between admission SYNTAX score and triglyceride-glucose index in non-diabetic patients with st-segment elevation myocardial infarction evaluated with proportional odds model

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Background and Aim: Insulin resistance is an important risk factor for cardiometabolic diseases. It has been shown to have significant correlation with triglyceride-glucose (TyG) index and thus TyG index has been identified as a simple and easily accessible marker of insulin resistance. Recent studies have reported that TyG index has prognostic value in patients with ST-segment elevation myocardial infarction (STEMI). There are few studies evaluating the effect of TyG index on coronary artery disease severity but there are no studies in patients with STEMI. Therefore, in this study, we aimed to investigate the effect of TyG index on coronary artery disease severity quantified by SYNTAX score in non-diabetic patients with STEMI.

Methods: A total of 1101 non-diabetic patients admitted to our center with STEMI and underwent primary percutaneous

coronary intervention between 01.2018 and 06.2019 were retrospectively included in this study. Demographic, clinical, laboratory and angiographic parameters were collected from medical records. SYNTAX score was determined by all coronary lesions with >50% diameter stenosis in a vessel >1.5 mm. TyG index was calculated as ln [fasting triglyceride (mg/dL)×fasting glucose (mg/dL)/2]. Patients were divided into low (SYNTAX score ≤22) and high (SYNTAX score >22) SYNTAX score groups. Baseline clinical features, laboratory data and in-hospital outcomes of the groups were compared. Independent predictors of high SYNTAX score was determined by multivariate analysis.

Results: Median age of the study group was 57 (49-67) years and 82.5% of the patients were male. 24% of the patients were in the high SYNTAX score group. Patients in the high SYNTAX score group were significantly older and had a higher prevalance of hypertension. They were more frequently presented with anterior myocardial infarction (55.8% vs 35.2%) and Killip class III-IV (13.6% vs 5%). Significantly higher admission creatinine, white blood cell count, troponin and creatine kinase-MB levels were observed whereas hemoglobin level was significantly lower in the high SYNTAX score group. TyG index was similar among groups. In-hospital ventricular arrhytmias, cardiopulmonary arrest and mortality were more commonly observed in high SYNTAX score group (Table 1). Age (OR: 1.38, 95% CI: 1.16-1.63, p=0.003) and white blood cell count (OR: 1.25, 95% CI: 1.12-1.40, p=0.001) were found as independent predictors of high admission SYNTAX score (Table 2). Proportional odds model revealed a significant non-linear inverse relationship between TyG index and SYNTAX score in hypertensive patients (Figure).

Conclusions: TyG index has a non-linear inverse relationship with admission SYNTAX score in hypertensive non-diabetic patients with STEMI. Further studies are required to confirm the results and evaluate the underlying mechanisms of this relation.

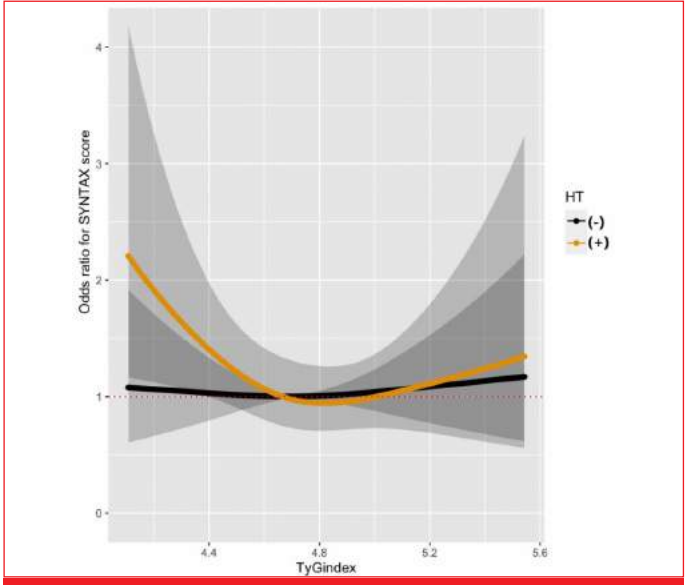

Figure 1. Proportional odds ratio plot showing the effect of TyG index*hypertension on admission SYNTAX score

Table 1. Baseline characteristics of study population

	Overall population (n=1101)	SYNTAX score≤22 (n=836)	SYNTAX score>22 (n=265)	p value
Age (years)	57 (49-67)	57 (49-66.8)	60 (50-70)	0.04
Gender (Female)	193 (17.5)	147 (17.6)	46 (17.4)	0.93
Hypertension	424 (38.5)	305 (36.5)	119 (44.9)	0.014
Hyperlipidemia	51 (4.6)	39 (4.7)	12 (4.5)	0.93
Smoking	870 (79)	668 (79.9)	202 (76.2)	0.2
Previous MI	189 (17.2)	143 (17.1)	46 (17.4)	0.92
Previous Revasc	176 (16)	135 (16.1)	41 (15.5)	0.79
Body mass index (kg/m^2)	27.5 (25-30.3)	27.5 (25.1-30.3)	27.6 (24.1-30.3)	0.34
Killip class III-IV	78 (7.1)	42 (5)	36 (13.6)	<0.000
Anterior MI	441 (40.2)	293 (35.2)	148 (55.8)	<0.000
TyG index	4.71 (4.54-4.89)	4.71 (4.55-4.88)	4.7 (4.51-4.93)	0.95
FBG (mg/dL)	94 (87-103)	94 (87-102)	95 (87-107)	0.09
Admission glucose (mg/dL)	121 (103-144.5)	120 (103-142)	124 (104-152)	0.2
Creatinine (mg/dL)	0.82 (0.74-0.98)	0.81 (0.73-0.97)	0.84 (0.75-1)	0.012
Total cholesterol (mg/dL)	179 (152-207)	179 (152-206)	179 (152-210)	0.79
LDL-cholesterol (mg/dL)	114 (91-138)	114 (91-138.8)	114 (92.5-138)	0.69
HDL-cholesterol (mg/dL)	35 (30-41)	35 (30-41)	34 (30-42)	0.62
Triglyceride (mg/dL)	128 (94-174)	130 (96-173.8)	127 (91-174.5)	0.28
C-reactive protein (mg/L)	0.9 (0.4-2.7)	0.9 (0.4-2.4)	1 (0.3-3.2)	0.37
Troponin (ng/mL)	3.8 (0.54-21.96)	3.21 (0.51-19.65)	5.5 (0.76-29.5)	0.03
CK-MB (U/L)	61.6 (27.3-144.9)	57.1 (25.6-135.3)	76.9 (34.4-169.8)	0.004
WBC (×103/µL)	11.77 (9.5-14.3)	11.61 (9.4-14.1)	12.3 (9.9-15.4)	0.01
Hemoglobin (g/dL)	14.1 (12.8-15.1)	14.1 (12.9-15.1)	14 (12.2-15)	0.04
LVEF (in-hospital)	48 (40-55)	50 (43-58)	40 (33-50)	<0.000
İn-hospital ST	24 (2.2)	17 (2)	7 (2.7)	0.55
İn hospital VT/ VF	125 (11.4)	77 (9.2)	48 (18.1)	<0.000
İn-hospital CPA	131 (11.9)	79 (9.4)	52 (19.6)	<0.000
İn hospital mortality	81 (7.4)	44 (5.3)	37 (14)	<0.000

Categorical data are presented as numbers (percentages) and continuous data are presented as median (interquartile range).
CK-MB, creatin kinase-MB; CPA, cardiopulmonary arrest; FBG,fasting blood glucose; Hb, hemoglobin; LVEF, left ventricular ejection fraction; MI, myocardial infarction; Revasc, Revascularization; ST, stent thrombosis; TyG index, Triglyceride-glucose index; WBC, white blood cell, VF, ventricular fibrillation; VT, ventricular tachycardi

Table 2. Proportional odds model for predictors of admission high SYNTAX score (>22)

Variables	OR (95 CI%)	p value
TyG index	1.02 (0.86-1.19)	0.55
TyG*HT	-	0.01*
Hypertension	0.98 (0.72-1.30)	0.10
Age (years)	1.38 (1.16-1.63)	0.003
Smoking	0.87 (0.67-1.08)	0.14
CRP (mg/L)	1.05 (0.98-1.12)	0.18
WBC (×103/μL)	1.25 (1.12-1.40)	0.001
Creatinine (mg/dL)	0.98 (0.94-1.02)	0.28

CI, confidence interval; CRP, C-reactive protein; OR, odds ratio; TyG, Triglyceride-glucose index; WBC, White blood cell.
*interaction p value

PB-051 [Coronary Artery Disease / Acute Coronary Syndrome]

Potassium level related to myocardial reperfusion injury in delayed STEMI presentation

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Background and Aim: Impaired reperfusion following PCI in patients with delayed STEMI is not fully elucidated. We aimed to analyze the role of electrolyte levels associated with unsuccessful reperfusion, assessed by TIMI flow, in patients presenting with delayed STEMI.

Methods: This was a prospective study that included 103 patients with the following inclusion criteria: total ischemic time > 120 minutes and ≤ 12 hours duration, persistent ST-segment elevation, rise of specific cardiac biomarkers, and those who underwent primary percutaneous coronary intervention. Based on the Thrombolysis in Myocardial Infarction (TIMI) flow grade, myocardial reperfusion injury (MRI) is characterized by a TIMI flow ≤ 2. Patients were divided in two groups, those with TIMI flow 3 and patients with TIMI flow ≤ 2.

Results: The difference between TIMI flow ≤ 2 group (39 patients) and TIMI flow 3 group (64 patients) in regard to electrolytes was: the mean sodium (Na+) level was 137.66 ± 2.80 mmol/L vs 138.57 ± 3.26 mmol/L, potassium (K+) 3.88 ± 0.55 mmol/L vs 3.61 ± 0.49 mmol/L, and the median calcium (Ca++) level 1 (0.41-1.15) mmol/L vs 1.03 (0.27-1.23) mmol/L. According to the Kruskal-Walis test, the difference between potassium levels in the two groups was statistically significant (p=0.016). Based on ROC analysis a cut-off value of 3.6 mmol/L for potassium is determined, showing higher rates of MRI (TIMI flow grade ≤2) among patients with potassium level >3.6 compared to potassium level ≤ 3.6 (p=0.015).

Conclusions: Lower potassium level, within reference range, has an encouraging role in myocardial reperfusion following percutaneous coronary intervention in delayed STEMI patients.

Table 1. Main characteristics and electrolytes.

	Potassium Mean (± SD)	P	Sodium Mean (± SD)	P	Calcium Median (range)	P
Final TIMI grade flow						
≤2	3.88 ± 0.55		137.66 ± 2.80		1 (0.41-1.15)	
3	3.61 ± 0.49	0.016	138.57 ± 3.26	0.26	1.03 (0.27-1.23)	0.43
Age						
≥65	3.69 ± 0.55		138.16 ± 3.79		0.95 (0.5-1.18)	
<65	3.73 ± 0.51	0.88	136.08 ± 16.93	0.47	1.04 (0.27-1.23)	0.04
Gender						
M	3.68 ± 0.46		138.74 ± 3.17		1.02 (0.27-1.28)	
F	3.8 ± 0.68	0.68	136.8 ± 2.49	0.02	0.96 (0.57-1.23)	0.1
Hypertension						
Yes	3.74 ± 0.63		138.47 ± 3.53		0.99 (0.41-1.23)	
No	3.69 ± 0.40	0.51	138.02 ± 2.62	0.64	1.01 (0.27-1.18)	0.21
Diabetes mellitus						
Yes	3.85 ± 0.67		136.47 ± 2.82		0.98 (0.75-1.13)	
No	3.67 ± 0.46	0.14	138.85 ± 3.00	0.001	1.01 (0.27-1.23)	0.51
Smoking						
Yes	3.68 ± 0.50		138.31 ± 2.72		1.00 (0.27-1.15)	
No	3.74 ± 0.56	0.78	138.20 ± 3.55	0.22	1.02 (0.41-1.11)	0.51

*Kruskal-Wallis test, and #Mann-Whitney test.

Table 2. Values of area under the curve for cardiac biomarkers and electrolytes

Cardiac biomarker/electrolyte	AUC (95% CI)	P-value
Troponin T	0.57 (0.36-0.78)	0.5
Creatine kinase	0.48 (0.26-0.70)	0.9
Creatine kinase-MB	0.5 (0.43-0.73)	0.72
C-reactive protein	0.50 (0.35-0.65)	0.56
Sodium	0.43 (0.31-0.54)	0.23
Potassium	0.64 (0.53-0.75)	0.015
Calcium	0.45 (0.34-0.56)	0.43

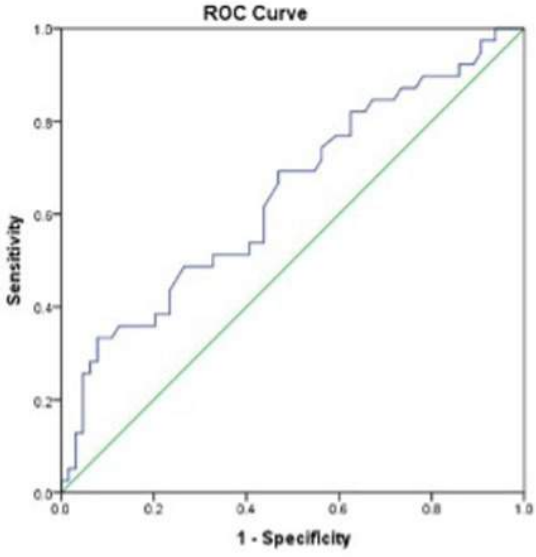

Figure 1. ROC curve analysis of association between the potassium values and myocardial reperfusion injury

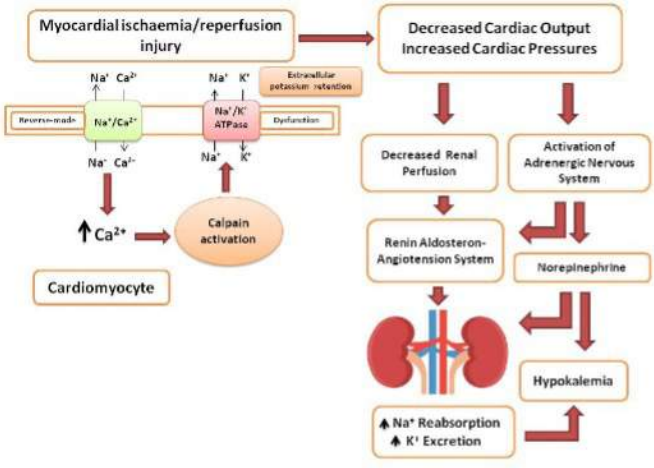

Figure 2. Myocardial reperfusion injury followed by decreased cardiac function, activation of the renin-angiotensin-aldosterone system and the autonomic nervous system leading to hypokalemia

PB-052 [Coronary Artery Disease / Acute Coronary Syndrome]

Is the plasma atherogenic index useful in detecting coronary arteries disesea in primary hyperparathyroidism?

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Background and Aim: The coronary flow reserve (CFR) is a sign of early-stage coronary artery disease (CAD). Plasma atherogenic index (PAI) is related to atherosclerosis and cardiovascular mortality. Therefore, our aim was to determine CFR and PAI in patients with primary hyperparathyroidism (PHPT) and investigate whether PAI can be used in the detection of early-stage CAD.

Methods: The sample was comprised of 44 patients with PTHT and 33 healthy volunteers. We defined CFR as the ratio of the hyperemic diastolic peak velocity to the baseline diastolic peak velocity. PAI values were calculated with the formula of log 10 triglyceride (TG) / high-density lipoprotein (HDL).

Results: The comparison of the groups for PAI and CFR demonstrated that PAI levels were significantly higher while CFR levels were significantly lower in the PTHT patients (p<0.01, p=0.01, respectively). The correlation analysis revealed that CFR was negatively correlated with PAI and TRG (PAI- p<0.0001 r=-0.537). The multivariate logistic regression analysis showed that only a high PAI level (OR: 151.6, 95% confidence interval (CI): 4.1-5480, p=0.006) was an independent predictor of reduction in CFR in PHPT patients.

Conclusions: Overall, we found an independent correlation between PAI and CFR values. Hence, PAI may be useful in identifying PHPT patients facing a high risk of adverse cardiovascular events and may also allow early diagnosis of subclinical atherosclerosis.

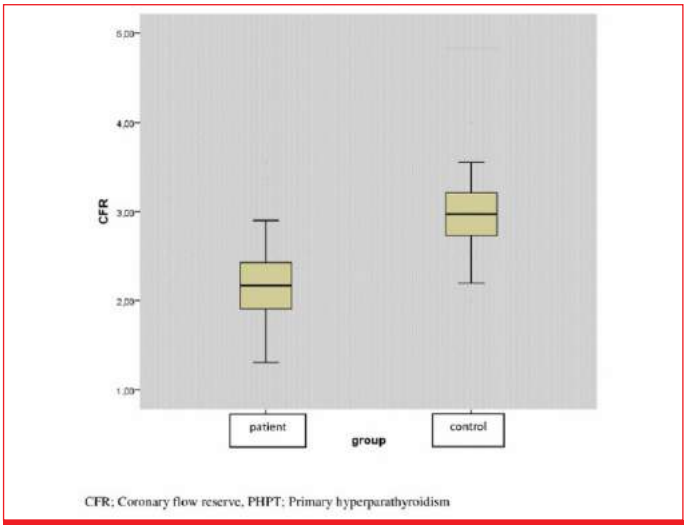
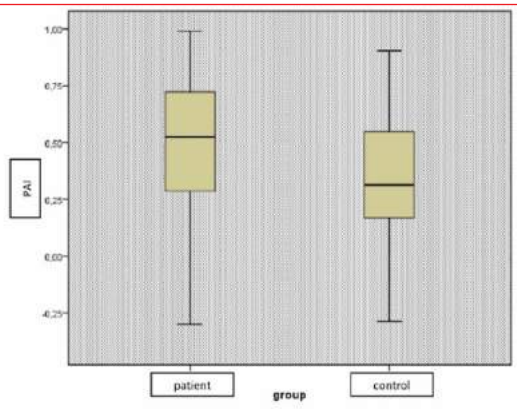
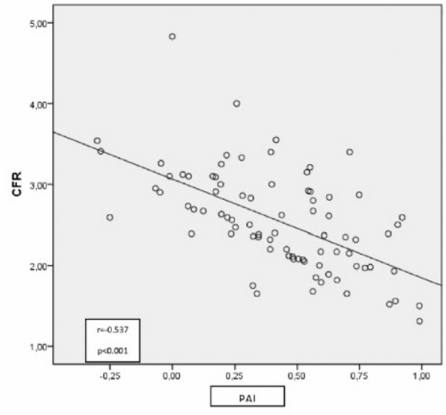


Figure 1. Comparison of CFR levels of PHPT patients and control groups



PAI; Plasma atherogenic index, PHPT; Primary hyperparathyroidism

Figure 2. Comparison of PAI levels of PHPT patients and control groups



CFR; Coronary flow reserve, PAI; Plasma atherogenic index, PHPT; Primary hyperparathyroidism

Figure 3. Relationship between PAI and CFT in patients with PHPT

Table 1. Correlation analysis of non-CFR parameters between CFR in PHPT patients

	CFR r values	CFR P values
PAI	-0,537	<0.001
AC	-0,250	0.023
CRI-1	-0,244	0.026
CRI-2	-0,120	0.282
hsCRP (mg/dl)	-0.409	<0.001
TC	-0,077	0.488
TG (mg/dl)	-0,363	0.001
HDL (mg/dl)	0,135	0,223
LDL (mg/dl)	0.080	0.473
Non-HDL (mg/dl)	-0.133	0.231
Albumin-corrected calcium	-0.610	<0.001
Phosphorus	<0.001	<0.001
PTH	-0,494	<0.001

CFR, Coronary flow reserve; PTH, Parathyroid Hormone; PAI, Plasma atherogenic index; AC, Atherogenic coefficient; CRI, Castelli risk indice; hsCRP, High-sensitivity C-reactive protein; TC, Total cholesterol; TRG, triglyceride; HDL, High-density lipoprotein cholesterol; LDL, Low-density lipoprotein.

Table 2. Comparison of demographic, clinical and laboratory values between subgroups with low and high CFR levels (cut-off value 2 for CFR)

	CFR <2 (n=19)	CFR ≥2 (n=25)	P value
CFR	1.7 ± 0.2	2.39 ± 0.33	<0.001
Age (years)	56.6 ± 11.2	55.6 ± 11.5	0.768
BMI (kg/m2)	25.9 ± 3.7	25.8 ± 2.8	0.94
SBP (mmHg)	129.6 ± 8.2	131.3 ± 5.6	0.459
DBP (mmHg)	78.2 ± 4.1	80.3 ± 4.2	0.130
TC (mg/dl)	186.5 ± 37.1	182.4 ± 37.5	0.706
TG (mg/dl)	170.4 ± 76.3	120.4 ± 65.6	0.019
HDL (mg/dl)	42.3 ± 17.1	43.2 ± 10.6	0.836
LDL (mg/dl)	105.8 ± 33.9	113 ± 26.6	0.416
Non-HDL (mg/dl)	145.1 ± 37.8	139.2 ± 40.4	0.607
PAI	0.66 ± 0.17	0.40 ± 0.25	0.001
AC	3.78 ± 1.3	3.48 ± 1,5	0.484
CRI-1	4.75 ± 1.3	4.4 ± 1.54	0.521
CRI-2	2.74 ± 0.9	2.78 ± 1.04	0.892
hsCRP (mg/dl)	6.6 ± 4.4	3.97 ± 3.7	0.032
PTH	296.7 ± 214	161.5 ± 105.5	0.011
Albumin-corrected calcium	11.3 ± 0.74	10.9 ± 0.46	0.023
Phosphorus	2.44 ± 0.4	2.57 ± 0.4	0.315

CFR, Coronary flow reserve; SBP; Systolic blood pressure; DBP, Diastolic blood pressure; BMI, Body mass index; TC,Total cholesterol; HDL, High-density lipoprotein cholesterol; TG, Triglyceride; LDL, Low-density lipoprotein; PAI, Plasma atherogenic index; CRI, Castelli risk indice; AC, Atherogenic coefficient; CFR, Coronary flow reserve; hsCRP, High-sensitivity C-reactive protein; PTH, Parathyroid Hormone.

Table 3. Univariate and multivariate logistic regression analysis to identify the independent predictors of coronary flow reserve in patients with PHPT

	Univariate analysis	Univariate analysis	Univariate analysis	Multivariate analysis	Multivariate analysis	Multivariate analysis
	Odds Ratio	95% CI	p Value	Odds Ratio	95% CI	P Value
PAI	159.3	7.1-3525.5	0.001	151.6	4.1-5480	0.006
PTH	1.005	1.000-1.009	0.031			
Albumin-corrected calcium	3.397	1.083-10.653	0.036			
hsCRP (mg/dl)	1.172	1.007-1.365	0.041			

PAI; Plasma atherogenic index, PTH;Parathyroid Hormone, hsCRP; High-sensitivity C-reactive protein

PB-053 [Coronary Artery Disease / Acute Coronary Syndrome]

Multi-vessel disease is more common in patients with ST elevation myocardial infarction who underwent angiography for the first time

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Background and Aim: Acute coronary syndromes (ACS) which is consist of ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI) and unstable angina pectoris (USAP) are a group of diseases with acute coronary vascular stenosis or occlusion. STEMI is a type of ACS that causes more severe hemodynamic impairment clinically and requires more urgent invasive intervention. However, ACS type in which cardiac biomarkers deterioration and electrocardiographic (ECG) changes are not seen, such as USAP, is expected to progress with a lower mortality due to its faint clinical presentation. In this article, we aimed to investigate the relationship between ACS types and multivessel disease in patients who underwent revascularization.

Methods: 525 patients, between the age of 30 and 95 years, who were hospitalized for ACS and underwent angiography between July 2019 and June 2022, were retrospectively scanned and analyzed. Patients were divided into 3 groups as USAP, NSTEMI and STEMI according to their clinical, ECG and laboratory data. Demographic characteristics, laboratory data, application forms, revascularization procedures, epicrisis data of the patients in each group were examined. Cardiac coronary catheterization was performed in all patients, and patients who underwent revascularization with percutaneous coronary intervention or coronary artery bypass grafting were included in the study. Patients who did not have laboratory and angiography data within the specified time period, patients with ACS who did not undergo coronary angiography (CAG) and were treated medically, patients who underwent elective CAG, patients with serious co-morbidities such as severe liver or kidney dysfunction, and patients with a previous history of CAD were not included in the study. The number of coronary arteries with critical stenosis was compared with each other. Lesions causing stenosis of 70% or more in the coronary artery lumen were considered critical. Patients with three or more coronary arteries with critical stenosis were named as multivessel patients. A p value below 0.05 was considered statistically significant.

Results: 522 (25.4%) STEMI, 1116 (54.5%) NSTEMI, and 410 (24.1%) USAP patients were included. STEMI patients were younger than the other two groups, male gender was higher in STEMI group. Smoking were higher in STEMI. LDL values were higher in STEMI. Most patients in all 3 ACS groups had single vessel occlusion-stenosis. 3 vessels or more occlusion-stenosis rates were less in all 3 groups. Single vessel disease was higher in the USAP group, while it was lower in the STEMI group. 2 or more vessel disease was higher in the STEMI group, and lower in the USAP group. There was a weak but positive significant correlation between LDL and HbA1c and the number of vessels with coronary occlusion-stenosis

Conclusions: Multiple vessel disease was found to be more common in ACS patients presenting with STEMI.

Table 1. Demographic, laboratory data, cardiovascular risk factors and vessel numbers of patients with acute coronary syndrome

Variables	STEMI (n:522)	NSTEMI (n:1116)	USAP (n:410)	P
Age (year)	57.8 ± 11.9	65.3 ± 11.7	62.5 ± 9.6	*<0.001 ‡0.277 ¥0.043
Male (n, %)	406 (77.7)	836 (74.8)	267 (65.3)	*0.252 ‡<0.001 ¥0.210
DM (n, %)	132 (25.4)	340 (30.4)	130 (31.6)	*0.062 ‡0.065 ¥0.412
HT (n, %)	126 (24.2)	288 (25.7)	98 (23.8)	*0.672 ‡0.673 ¥0.832
Smoking (n, %)	136 (26.2)	236 (21.1)	84 (20.4)	*0.043 ‡0.027 ¥0.712
Obesity (n, %)	122 (23.3)	276 (24.7)	95 (23.1)	*0.582 ‡0.810 ¥0.640
LDL (mg/dL)	126.64 ± 33.7	122.1 ± 37.7	116 ± 39.8	*0.040 ‡<0.001 ¥0.059
HDL (mg/dL)	38.9 ± 11.6	39.8 ± 12.3	41.4 ± 14.6	*0.632 ‡0.887 ¥0.813

Table 1. Demographic, laboratory data, cardiovascular risk factors and vessel numbers of patients with acute coronary syndrome (Continued)

Variables	STEMI (n:522)	NSTEMI (n:1116)	USAP (n:410)	P
TG (mg/dL)	167.4 (153.5-181.2)	166.9 (158.2-175.5)	173.3 (162.0-184.6)	*0.982 ‡0.883 ¥0.753
HbA1c (%)	8.3 ± 3.0	10.1 ± 4.3	6.9 ± 1.9	*0.277 ‡0.053 ¥0.011
Creatinine (mg/dL)	1.1 ± 0.1	1.14 ± 0.2	1.1 ± 0.2	*0.130 ‡0.406 ¥0.416
1 vessel	255 (49%)	656 (58.8%)	335 (81.7%)	*<0.001 ‡<0.001 ¥<0.001
2 vessels	167 (32%)	268 (24%)	55 (13.4%)	*<0.001 ‡<0.001 ¥<0.001
3 or more vessels	100 (19%)	192 (17.2%)	20 (4.9%)	*0.001 ‡<0.001 ¥<0.001

DM, diabetes mellitus; HT, hypertension; NSTEMI, non-ST elevation myocardial infarction; STEMI, ST elevation myocardial infarction; USAP, unstable angina pectoris.
*p value between STEMI and NSTEMI. ‡ p-value between NSTEMI and UA. ¥ p value between STEMI and UA

PB-054 [Coronary Artery Disease / Acute Coronary Syndrome]

Circulating miR-126, miR-210 and let-7g are differentially expressed in MI patients and associated with lipid levels

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Background and Aim: Coronary artery disease (CAD) is one of the most important public health problems. It is of great importance to determine the underlying mechanisms of CAD and non-invasive biomarkers that will provide information about the presence and severity of CAD. Thus, the determination of microRNAs (miRNA) released from cells into the circulation in pathological conditions has emerged as an aim. In this study, it was aimed to determine the circulating levels of miR-126, miR-210, let-7g and miR-326 in CAD and control groups.

Methods: The individuals who underwent coronary angiography and included in the study were classified according to their clinical status as non-CAD (≤30 stenosis, n=55), stable angina pectoris (SAP) (n=48), unstable angina pectoris (UAP) (n=46) and myocardial infarction (MI) (n=36) patients. Circulating miR-126, miR-210, let-7g and miR-326 expression levels were determined in serum samples using the quantitative Real-Time PCR method. Possible target genes of miRNAs and the pathways in which these genes are enriched were investigated by performing bioinformatic analyzes.

Results: Circulating levels of let-7g are found to increase in the MI group compared to UAP, while miR-210 levels are lower in MI patients compared to the other groups. Moreover, miR-126 levels are decreased in UAP and MI patients relative to the non-CAD group. The circulating levels of let-7g, miR-126, and miR-210 are correlated with lipid levels and ratios. In addition, ROC curve analyses of miR-126 and miR-210 are shown promising results as biomarkers. Moreover, in silico analyses revealed that putative targets of the selected miRNAs are associated with CAD development and relevant pathways.

Conclusions: In this study, circulating of let-7g, miR-210, and miR-126 were found differentially expressed in MI group than others and miRNAs were found in correlation with lipid levels and ratios. In conclusion, the results of the study suggest that circulating let-7g, miR-126-3p, and miR-210-3p have potential as biomarkers and their mechanism of action should be elucidated with further functional studies.

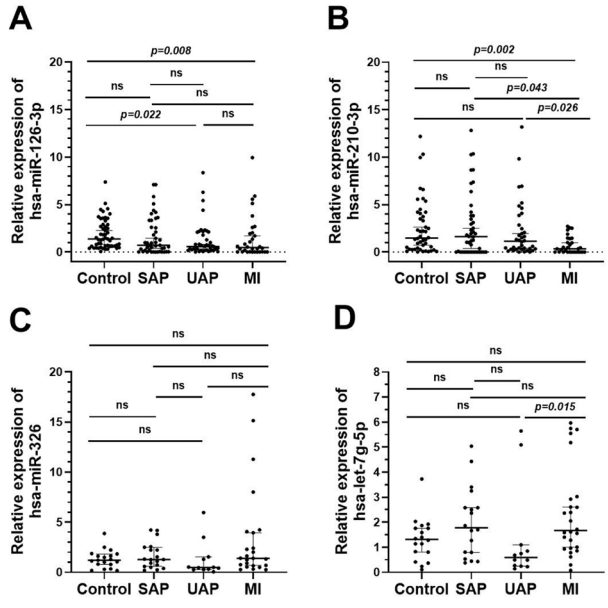

Figure 1. The circulating levels of miR-126-3p, miR-210-3p, miR-326, and let-7g-5p. (A) The circulating levels of miR-126-3p were significantly different in MI and UAP groups compared to non-CAD controls. In pairwise comparisons other than UAP vs. control and MI vs. control, the circulating levels of miR-126-3p were not significantly different. (B) In pairwise comparisons, the circulating levels of miR-210-3p were found significantly different in the MI group compared to UAP, SAP, and non-CAD controls. (C) The circulating levels of miR-326 were not significantly different between groups. (D) In pairwise comparisons, the circulating levels of let-7g-5p were significantly different in the MI group compared to UAP.

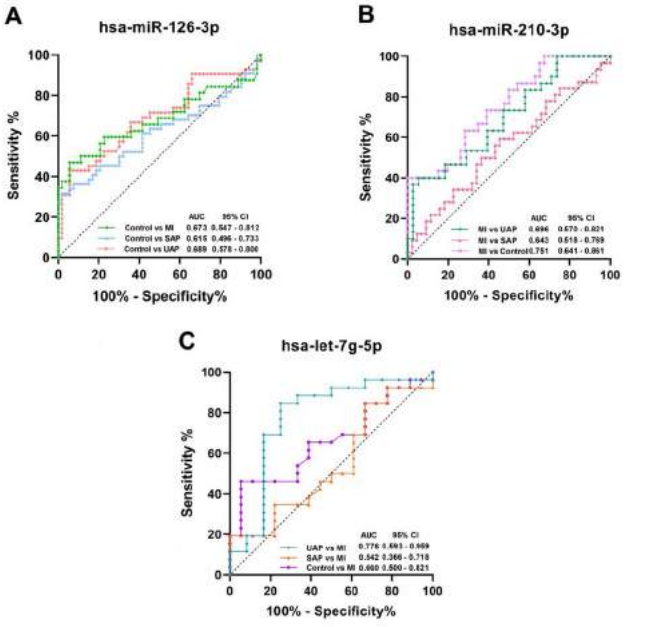

Figure 2. Sensitivity and specificity of circulating miR-126-3p, miR-210-3p, and let-7g-5p. The receiver operating characteristic (ROC) curve analyses were performed for the determination of the discriminative ability of (A) miR-126-3p, (B) miR-210-3p, and (C) let-7g-5p among non-CAD, SAP, UAP, and MI.

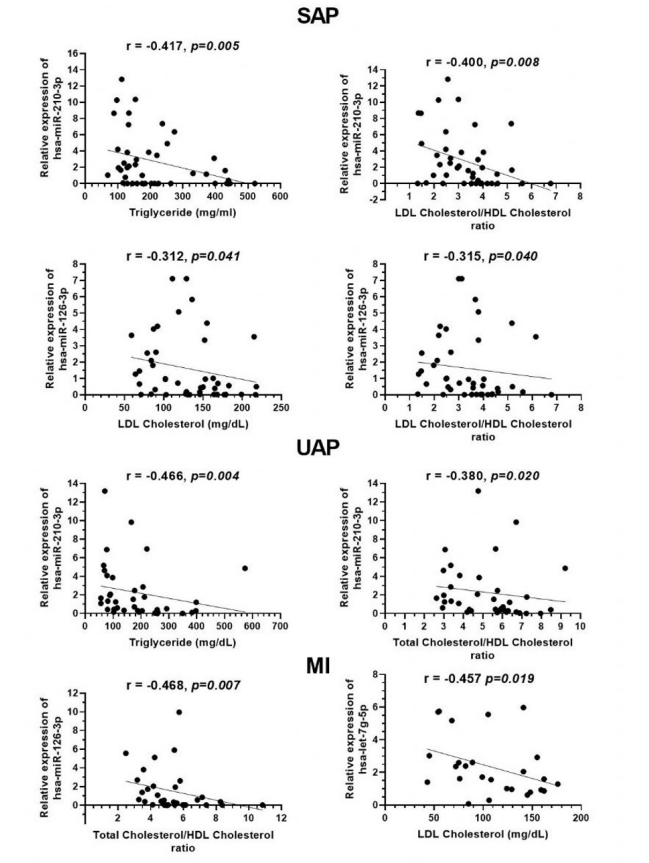

Figure 3. The correlations of miR-126-3p, miR-210-3p and let-7g-5p levels with lipid levels and ratios.

PB-055 [Coronary Artery Disease / Acute Coronary Syndrome]

Blood pressure recovery ratio after exercise test predicts increased carotid intima media thickness in patients with metabolic syndrome

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Background and Aim: Low blood pressure (BP) recovery after exercise is associated with coronary artery disease, left ventricular diastolic dysfunction and pulse wave velocity. We aimed to investigate the association between BP recovery ratio (BPRR) and carotid intima media thickness (CIMT) in patients with metabolic syndrome.

Methods: Among 109 patients who underwent elective exercise stress test, 98 patients with negative exercise test result and metabolic syndrome (MetS) were included (65 males, 44 females; mean age 54.8 ± 8.3 years) in this study. Patients were classified into two groups according to CIMT values: low CIMT (CIMT ≤ 0.9) and high CIMT (>0.9). The BPRR was calculated by dividing the third minute systolic blood pressure (SBP) by the peak exercise SBP.

Results: The BPRR was 0.88 in the low CIMT group, and 0.91 in the high CIMT group (p=0.008). Septal e were significantly lower, creatinine values and statin use rates were significantly higher in the high CIMT group (p <0.05 for all). BPRR was positively correlated with CIMT (r=0.251, p=0.009. Statin use (OR = 5.754, p <0.001) and BPRR (OR = 3.036, p=0.008) were independent parameters for predicting high CIMT. Every 0.1 unit increase in BPRR was associated with 203.6% increase in the risk of high CIMT. The cut-off value of BPRR obtained by ROC curve analysis was 0,921 for prediction of high CIMT. The area under the curve (AUC) was 0.634 (p=0.018).

Conclusions: We found a strong correlation between BPRR and CIMT in patients with metabolic syndrome. BPRR and CIMT, as easy and non-invasive methods, may be used to detect patients with increased risk for cardiovascular events in patients with metabolic syndrome.

Table 1. Comparison of the baseline clinical and demographic features

	Low IMT n=66	High IMT n=43	P
Age (years)	53.9 ± 8.3	56.1 ± 8.2	0.175
Gender (Male, %)	36 (54)	29 (67)	0.180
DM (n, %)	30 (45)	16(38)	0.394
HT (n, %)	42(63)	29(67)	0.684
Smoking status (n, %)	24(36)	15(35)	0.875
Body mass index (kg/m2)	30.7 ± 3.4	30.5 ± 3.4	0.734
Waist circumference (cm)	102.0 ± 7.8	103.5 ± 7.1	0.308

Hemoglobin (g / dl)	13.8 ± 1.4	13.9 ± 1.5	0.779
Creatinine (mg / dl)	0.77 ± 0.14	0.84 ± 0.22	0.049
Total cholesterol (mg / dl)	198.1 ± 30.4	207.0 ± 36.7	0.240
LDL cholesterol (mg / dl)	118.1 ± 30.6	128.8 ± 42.3	0.173
HDL cholesterol (mg / dl)	43.5 ± 11.2	46.6 ± 16.7	0.305
Triglycerides (mg / dl)	175.5 ± 81.0	183.5 ± 111.4	0.958
Fasting blood glucose (mg / dl)	111.5 ± 31.6	120.6 ± 50.5	0.250
Blood urea nitrogen (mg / L)	12.0 ± 4.5	10.1 ± 5.2	0.062
ARB (n, %)	9 (14)	6 (14)	0.963
ACEi (n, %)	14 (21)	9 (21)	0.972
Beta-blockers (n, %)	23 (35)	9 (21)	0.119
Asetilsalisilikasit (n, %)	32 (48)	27 (63)	0.143
Statin (n, %)	13 (20)	24 (56)	<0.001
Calcium channel blocker (n, %)	21 (32)	10 (23)	0.333
HeartScore (%)	4.2 ± 3.6	6.6 ± 6.8	0.062
Carotid intima media thickness (cm)	0.71 ± 0.10	1.04 ± 0.18	<0.001

LDL, Low density lipoprotein; HDL, High density lipoprotein; LA, Left atrium; LVEDD, Left ventricular end diastolic diameter; IVS, Interventricular septum; MV, Mitral valve.

Table 2. Comparison of echocardiographic parameters

	Low IMT n=66	High IMT n=43	P
Ejection Fraction (%)	60.1 ± 2.9	59.6 ± 2.4	0.378
LVEDD (mm)	48.2 ± 1.6	48.4 ± 1.8	0.521
E wave deceleration time (s)	174.5 ± 14.1	171.8 ± 16.0	0.359
MV E/A ratio	1.09 ± 0.16	1.03 ± 0.14	0.062
Lateral annular e'velocity (cm/s)	12.1 ± 2.3	11.4 ± 1.9	0.137
Septal annular e'velocity (cm/s)	10.1 ± 1.7	9.4 ± 1.3	0.035
Mitral E/e'ratio	7.7 ± 1.7	7.9 ± 1.7	0.517
Posterior wall thickness (mm)	9.5 ± 1.0	9.5 ± 1.4	0.740
IVS thickness (mm)	9.8 ± 1.2	9.7 ± 1.2	0.885
LA diameter (mm)	31.1 ± 2.1	31.4 ± 2.7	0.457
LA volume (mL)	32.9 ± 4.1	31.9 ± 4.4	0.232
LA volume index (mL / m^2)	21.9 ± 3.0	22.6 ± 3.4	0.237
Myocardial performance index	0.40 ± 0.03	0.41 ± 0.03	0.277

IMT, Intima media thickness; LA, Left atrium; LVEDD, Left ventricular end diastolic diameter; IVS, Interventricular septum; MV, Mitral valve.

Table 3. Comparison of exercise stress testing parameters

	Low IMT n=66	High IMT n=43	P
Resting SBP (mmHg)	136.9 ± 16.4	142.5 ± 18.5	0.103
Resting DBP (mmHg)	81.5 ± 11.3	85.8 ± 12.1	0.065
Peak SBP (mmHg)	168.7 ± 21.8	170.7 ± 25.5	0.663
SBP at 3 minutes (mmHg)	149.1 ± 19.9	156.4 ± 20.3	0.068
BPRR	0.88 ± 0.07	0.91 ± 0.05	0.018
HRR (beat/min)	30.1 ± 15.2	28.4 ± 15.1	0.569
Metabolic equivalent	10.5 ± 8.0	9.6 ± 2.6	0.468

IMT, Intima media thickness; SBP, Systolic blood pressure; BPRR, Blood pressure recovery ratio; HR, Heart rate; HRR, Heart rate recovery.

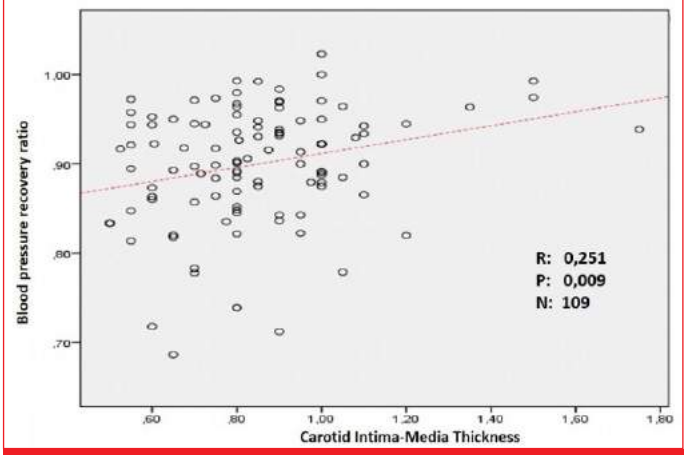

Figure 1. Scatter plot diagram of the relationship between blood pressure recovery ratio and carotid intima media thickness

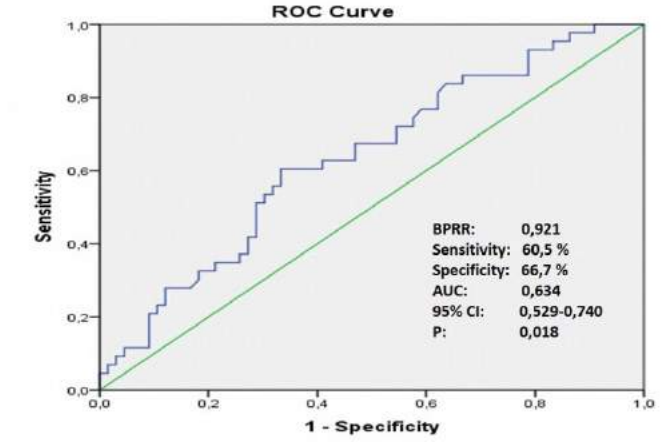

Figure 2. ROC curve analysis to determine predictive value of blood pressure recovery ratio for predicting carotid intima media thickness

Table 4. Multivariate logistic regression analyses to determine the independent predictors of high carotid intima media thickness

Variable	P	Odds Ratio	95% CI -Lower	95% CI -Upper
Statin use	<0.001	5.754	2.237	14.803
BPRR	0.008	3.036	1.336	6.897
Creatine	0.146			
MV Septal annular e'velocity	0.078			

IMT, Intima media thickness; BPRR, Blood pressure recovery ratio; MV, Mitral valve

PB-056 [Coronary Artery Disease / Acute Coronary Syndrome]

The impact of ticagrelor preloading on coronary artery bypass grafting related bleeding in patients with ST-segment elevation myocardial infarction initially managed with primary percutaneous coronary intervention

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Background and Aim: Acute coronary syndrome patients on double antiplatelet treatment who need urgent or early coronary artery bypass grafting (CABG) are at high risk of major bleeding.

In this study, we aimed to investigate the impact of ticagrelor preloading on CABG related bleeding in patients with ST-segment elevation myocardial infarction initially managed with primary percutaneous coronary intervention (pPCI).

Methods: Patients with a clear diagnosis of STEMI who were managed with pPCI and underwent subsequent urgent (0-2 days) or early (3-7 days) CABG surgery were included. All study patients were preloaded with ticagrelor 180 mg at the time of diagnosis with an intention of pPCI. Patients' demographics, clinical variables and short-term cardiovascular outcomes were recorded.

Results: The study cohort included a total of 98 patients who fulfilled the study criteria. Forty four (44.8%) of patients underwent CABG surgery within 3 days of pPCI procedure and 42 (43%) patients underwent between days 3 and 7. Left main coronary artery involvement and presence of chronic total occlusion in the non-infarct related artery were 26.5% and 25.5%, subsequently. CABG-related bleeding occurred in 22 (22.4%) patients. There was no significant difference with respect to total Ticagrelor dose and timing of the surgery between patients with or without CABG-related bleeding (p: 0.165 and p:0.142). Multivariate analyses demonstrated that only preoperative hemoglobin level was an independent predictor of major bleeding (OR:0.720, p: 0.034). There were 3 deaths within the 30 days of surgery, all occurring in patients with major bleeding. However, major bleeding was not associated with long-term cardiovascular events during the follow-up.

Conclusions: Our results indicated that discontinuation of ticagrelor therapy 3 days prior to surgery is sufficient to avoid CABG-related bleeding. Moreover, early CABG following STEMI does not increase the risk of long-term cardiovascular events.

Table 1. Demographics and clinical variables of study population

	Mean
Age	61.0±10.1
Gender(male)	78%
Hypertension	61.2%
Diabetes mellitus	43.9%
Chronic renal failure	10.2%
LMCA involvement	26.5%
Non-IRA CTO	25.5%
Ischemia duration(minutes)	120(60-1080)
Hemoglobin(gr/L)	12.6±2.11
Hs-TnT level(peak, ng/L)	4.1±3.42
Pro-BNP(pg/L)	760(200-35000)
LVEF	46.3±8.80
ICU stay(days)	3(1-20)
Postoperative major bleeding	22.4%

Table 2. Comparision of patients with/without bleeding

	Major bleeding (-) (74 patients)	Major Bleeding (+) (22 patients)	p value
Age	60.9±10.6	61.1±10.0	0.937
Gender	77%	79%	0.892
HT	64.5%	50%	0.220
DM	43.5%	45.5%	0.866
CVA	4.1%	3.9%	0.243
CRF	6.7%	22.7%	**0.028**
Preoperative HGB	13.0±2.0	11.3±1.7	**0.001**
LVEF	46.0±9.3	47.0±6.6	0.613
Troponin	2.53(0.4-10)	3.9(0.3-10)	0.314
Pro-BNP	500(200-18960)	1081(450-35.000)	**0.026**
Additional Ticagrelor	90 (0-450)	180 (0-630)	0.165
ES replacement	1.5(0-4)	7 (2-26)	**<0.001**
Early CABG	32 (43%)	10 (45%)	0.142
ICU stay	2 (1-10)	6(1-20)	**<0.001**
Hospital stay	7(3-27)	11.5(3-90)	**0.011**

Table 3. Univariate and multivariate analysis for predicting major bleeding

	Univariate analyses			*Multivariate analyses*		
	OR	95% CI	p value	OR	95% CI	p value
Age	0.998	0.952 – 1.046	0.936			
HT	0.551	0.211 -1.437	0.223			
DM	1.086	0.418 -2,819	0.866			
CRF	**4.176**	**1.085 -9.076**	**0.038**	0.698	0.445 – 1.094	0.643
Additional ticagrelor Hgb	0.574	0.221 -1.493	0.255			
	0.661	**0.506 -0.862**	**0.002**	**0.720**	**0.531 – 0.976**	**0.034**
Early surgery	0.739	0.517 – 1.058	0.098	0.698	1,094 – 8.651	0.117

Table 4. Predictors of major adverse cardiovascular outcomes

	Univariate analyses			*Multivariate analyses*		
	OR	95% CI	p value	OR	95% CI	p value
Age	1.026	0.986 – 1.068	0.211			
HT	0.727	0.321 - 1650	0.446			
DM	1.440	0.645 – 3.219	0.374			
CRF	1.370	0.361 – 5.191	0.644			
Non-IRA CTO	**2.950**	**1.100 – 7.912**	**0.032**	**1.714**	**0.281 – 8.452**	**0.559**
LMCA disease	1.044	0.425 – 2.565	0.925			
Peak Hs-TnT	**1.306**	**1.132 – 1.506**	**<0.001**	**1.312**	**1.018 – 1.691**	**0.036**
Mitral regurgitation	**2.976**	**1.285 – 6.896**	**0.011**	**2.517**	**1.170 – 5.687**	**0.006**
LVEF	**0.710**	**0.626 – 0.806**	**<0.001**	**0.692**	**0.592 – 0.811**	**<0.001**
Major bleeding	1.080	0.417 – 2.799	0.874			
Early surgery	0.806	0.602 – 1.078	0.146			

PB-058 [Coronary Artery Disease / Acute Coronary Syndrome]

The importance of soluble ST2 level in non-ST segment elevation acute coronary syndromes

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Background and Aim: The aim of this study is to investigate soluble suppression of tumorigenicity 2 (sST2) role in determining the severity of coronary atherosclerosis, its relationship with in-hospital mortality, and its usability as a diagnostic biomarker together with troponin in acute coronary syndromes.

Methods: In this prospective study, we included a total of 188 patients. Patients were divided into 2 groups: patients in whom coronary angiography showed <50% coronary artery stenosis(n=86) and patients with non-ST segment elevation acute coronary syndromes (NSTE ACS) (n=102). sST2 levels were measured by ELISA method.

Results: In the NSTE ACS group male gender ratio (67% vs 33%), age (61.75±12.56 vs 58.57±9.69), dyslipidemia and smoking rate were significantly higher (for all p<0.001), whereas diabetes mellitus rate was higher in the <50% stenosis group (p:0.04). Other demographic characteristics were similar between the two groups. In the NSTE ACS group, low-density lipoprotein cholesterol (LDL) values (129.87±38.23 vs 108.42±26.0) and sST2 values (229.75±70.33 vs 55.02±25.14) were significantly higher, while left ventricular ejection fraction (LVEF) (50.79±10.86 vs. 59.37±4.01) were lower than <50% stenosis group. sST2 value was positive correlated with age, admission troponin, CK and CK-MB values, male gender, dyslipidemia, smoking, LDL cholesterol, Gensini score, in-hospital mortality, electrical and hemodynamic instability, fragmented QRS, QTc value and GRACE score (for all p<0.001), while negatively correlated with LVEF value (p<0.001). In the NSTE ACS group, patient with sST2 values ≥230 ng/L had body mass index (30.57±4.54 vs. 27.95±3.14), Gensini score (64.64±56.64 vs 54.37±38.79), admission troponin value (2843±9584 vs 1275±3296), three-vessel coronary artery disease (CAD) (53.3% vs. 46.7%) and in-hospital mortality rates (71.4% vs. 28.57%) significantly higher compared to those with sST2 value <230 ng/L. Receiver operating characteristic (ROC) curve analysis was used to calculate the threshold values of sST2. We found that sST2 value > 311.78 ng/L (Area under the ROC curve-AUC: 0.927, sensitivity: 57.1%, specificity: 42.9%, p<0.001) was related with in-hospital mortality. sST2 value >218.09 ng/L (AUC: 0.815, sensitivity: 50.0%, specificity: 50.0%, p<0.001) was related with three-vessel CAD. sST2 value >232.56 ng/L (AUC: 0.802, sensitivity: 46.7%, specificity: 53.3%, p<0.001) was related with electrical and hemodynamic instability. In the logistic regression analysis high sST2 value and low LVEF value were defined as independent risk factors for in-hospital mortality.

Conclusions: We found that in NSTE ACS patients sST2 value increased in parallel with admission troponin value, three-vessel CAD, electrical and hemodynamic instability, low LVEF value and in-hospital mortality. Independent risk factors for in-hospital mortality were defined as high sST2 value and low LVEF value. sST2 level can be considered as an important marker for NSTE ACS to determine the time of intervention.

PB-059 [Coronary Artery Disease / Acute Coronary Syndrome]

Cardiovascular disease is the leading cause of mortality in women, are we aware? A survey study on 7920 individuals

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Background and Aim: Cardiovascular disease constitutes the major reason of mortality worldwide for both genders. However, women are overlooked as victims of cardiovascular disease. World Health Organization data underscores ischemic heart disease as the leading cause of death for both men and women, as is the case according to Turkish Statistical Institute data. A 2019 AHA survey found that only 44% of respondents knew that heart disease is the leading cause of female mortality.

Methods: Our study was designed as a survey aiming to seek for awareness of cardiovascular disease as the leading cause of female mortality. Individuals between 18-80 years of age were enrolled and asked to participate in a short survey covering 10 questions.

Results: The study included 7920 individuals aged between 18-80 years. Fifty eight percent (n=4643) of the population were female. Cardiovascular disease was pointed out as the leading cause of women's mortality by 34% (n=1572) of the female and 38% (n=1241) of the male participants. There was statistical significance between two groups. Malignant diseases were declared to be the leading cause by 46% (n=2118) of the women and 42% (n=1404) of the men. Breast cancer was chosen as the major reason by 25% of the study population (n=2015) while 14% reported they had no idea. Besides, education was not found to be a determinant in the awareness of causes of female mortality.

Conclusions: Cardiovascular disease is the leading cause of mortality in women worldwide. Many women live with and die from cardiovascular disease. In contrast to mortality of malignant diseases, cardiovascular mortality in women is not on a decline. Despite this fact there is a neglect in the awareness of cardiovascular disease as the leading cause of female mortality. This misperception may partially be due to sex specific differences in risk factors and clinical presentation, underrepresentation of women in clinical trials, the belief that poor lifestyle habits are the only drive for cardiovascular disease. However, the significant difference in awareness between sex groups may signify that women are more likely to neglect to have or die from cardiovascular disease. This may culminate in inadequate risk factor modification and late diagnosis of cardiovascular disease in women. Another remarkable point is that education level did not differ between groups that pointed out cardiovascular disease and others. This finding may indicate failure in placing health literacy in our education system.

PB-060 [Coronary Artery Disease / Acute Coronary Syndrome]

Predicting the mortality in patients with S-T elevation myocardial infarction by monocyte to high-density lipoprotein ratio

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Background and Aim: Monocytes, which produce a variety of cytokines and molecules, interact with platelets and endothelial cells, causing inflammatory and thrombotic pathways to become worse. Macrophage migration and oxidation of low-density lipoprotein cholesterol molecules are both inhibited by high-density lipoprotein cholesterol (HDL-C). HDL-C neutralizes monocytes' pro-inflammatory and pro-oxidant effects through several mechanisms. As a result, characteristics like the monocyte to HDL-C ratio (MHR) may reveal a patient's inflammatory status. The development of coronary artery disease is influenced by inflammation, oxidative stress, and endothelial dysfunction. Recent research suggests that inflammatory biomarkers are important for assessing mortality in patients with ST-elevation myocardial infarction (STEMI). This study aims to determine the association between MHR and mortality of STEMI patients.

Methods: We enrolled 460 patients who were diagnosed with STEMI between 2019 and 2021 and were sent to our clinic within the first 24 hours of symptom onset. A total of 400 controls with similar ages and gender were included in the study. According to one-year mortality, the patients were divided into two groups. Monocyte counts, HDL-C, and MHR values were compared between the groups.

Results: The patient group had significantly higher monocyte counts and lower HDL levels than the control group, resulting in higher MHR values. In addition, non-surviving patients had a higher monocyte count and MHR value, as well as a lower HDL-C level (p 0.001). In patients with STEMI, the MHR value was also found to be a significant independent determinant of one-year mortality (p 0.001). MHR had the optimum cut-off value of 17.52 (95 % CI 0.95–0.98) for predicting one-year mortality in patients with STEMI.

Conclusions: In patients with STEMI, a high MHR value was found to be an independent predictor of one-year mortality.

Table 1. Comparison of baseline demographic, clinical, and laboratory characteristics of the groups and monocyte count, HDL level, and MHR value according to the one-year mortality

	Control group (n=400)	STEMI group (n=460)	X2 value	P-values
DM Presence, n (%)	150 (37.5%)	148 (32.1%)	1.21	0.27
HT Presence, n (%)	304 (76%)	378 (82.1%)	2.76	0.09
Smoking, n (%)	184 (46%)	170 (36.9%)	3.35	0.07
Age (years)	77.09±6.70	77.35±9.56		0.25
Male, n (%)	184 (46%)	208 (45.2%)	0.01	0.92
HDL-C (mg/dL)	52.81±28.96	45.93±15.61		<0.001
LDL-C (mg/dL)	108.13±7.81	106±7.46		0.58
Creatine (mg/dL)	0.80±0.12	0.78±0.12		0.12
Hb (g/dL)	14.32±1.28	14.18±1.89		0.59
Glucose (mg/dL)	123.42±42.59	127.53±26.95		<0.001
WBC (x109 µL)	6937.26±1935.20	8370.82±3712.09		<0.001
Neutrophil (x109 µL)	4647.06±1275.05	6420.04±3574.70		<0.001
Monocyte (x109 µL)	500.44±94.10	608.03±260.54		0.002
Lymphocyte (x109 µL)	1787.26±510.08	1270.69±708.29		<0.001
Mortality within the one-year period				
	Non-surviving group (n=88)	**Surviving group (n=372)**		
Monocyte (x109 µL)	915.42±53.21	524.03±9.98		<0.001
HDL-C (mg/dL)	37.03±1.16	46.22±0.9		<0.001
MHR	18.86±5.25	12.21±2.21		<0.001

Values are presented as mean ± SD and median [interquartile range]. DM: diabetes mellitus; HT: hypertension; HDL: high-density lipoprotein; LDL: low-density lipoprotein; Hb: hemoglobin; WBC: white blood cell; MHR: monocyte to high-density lipoprotein ratio.

PB-061 [Coronary Artery Disease / Acute Coronary Syndrome]

The association of serum uric acid/albumin ratio with stent trombosis in patients with ST elevation myocardial infarction

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Background and Aim: Despite advances stent technology and medical treatments, the incidence of stent trombosis (ST) is still frequent and a major problem following primer percutaneous coronary intervention (pPCI). Previous studies have shown that high uric acid (UA) and low serum albumin (SA) values were associated with adverse outcome in ST-elevation myocardial infarction (STEMI). This study aimed to investigate the predictive value of the uric acid-to-serum albumin ratio (UAR) for stent trombosis in STEMI patients

Methods: A total of 1887 consecutively STEMI were included in this cross-sectional study. The population sample was classified based on the development of ST during hospitalization. ST was defined as per the standardized definition proposed by the Academic Research Consortium. Acute (0 to 24 hours after stent implantation) and subacute (>24 hours after stent implantation to hospital stay) ST were included. The UAR was calculated by dividing the serum UA level by SA level.

Results: During the hospitalization, 80 pPCI patients (4,2%) were diagnosed with ST. Advanced age, male gender and diabetes mellitus (DM) and smoking were more common in patients who developed ST (Table- 1). ST (+) patients had higher serum UAR levels than ST (-). According to the multivariable logistic regression model, the UAR (OR: 2,6 95% CI: 1.92-3.52, p < 0.001) and DM (OR: 1,6 95% CI: 1.05-2,73, p < 0.029) were independent predictors for ST in STEMI patients. The area under curve (AUC) value of the UAR in a receiver operating characteristics (ROC) evaluation was 0.795. A cutoff value of 1.81 for UAR detected ST development with a sensitivity of 80 % and specificity of 71% (Figure- 1).

Conclusions: As a novel inflammatory marker, UAR was an independent predictor of ST development in STEMI patients underwent pPCI.

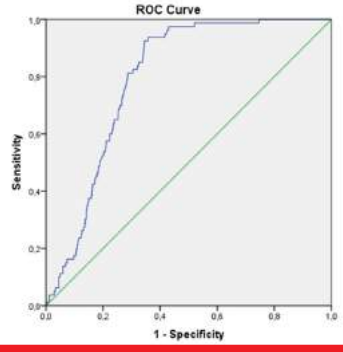

Figure 1. The ROC curve of uric asid-albumin ratio (UAR), for stent trombosis (AUC:0.795 Cut 1.81, sensitivite %80 spesifite %71)

Table 1. Baseline characteristics of the study population

Variables	Overall (n=1887)	ST (-) (n=1807)	ST (+) (n= 80)	P-value
Age, years	59 (51-69)	57 (49-65)	59 (61-69)	0.045
Male gender, n (%)	1458 (77.2)	1386 (76.7)	72 (90)	0.005
DM, n (%)	440 (23.3)	415 (22.9)	25 (32.2)	0.025
HT, n (%)	877 (46.4)	841 (46.5)	36 (46.2)	0.955
HL, n (%)	117 (6.2)	111 (6.1)	6 (7.5)	0.622
Smoking, n (%)	356 (18.8)	336 (18.5)	20 (25.1)	0.039
LMCA stenosis, n (%)	80 (4.2)	77 (4.2)	3 (4.2)	0.824
Stent Type (DES), n (%)	1738 (92.1)	1668 (92.3)	70 (92.5)	0.998
Total cholesterol, mg/dL	176 (152-204)	131 (98-178)	120 (98-170)	0.572
Triglyceride, mg/dL	131 (98-178)	149 (110-208)	123 (94-159)	0.124
HDL-C, mg/dL	35 (30-41)	35 (30-41)	35 (30-41)	0.629
LDL-C, mg/dL	111(90-134)	111 (90-134)	109 (93-135)	0.855
Creatinine, mg/dL	0.82 (0.7-1.0)	0.83 (0.74-0.10)	0.85 (0.7-1.1)	0.913
e-GFR, ml/min/1.73m2	93.3 (74.4-103)	93.7 (73-103)	94.7 (80-103)	0.399
Glucose, mg/dL	131 (108-182)	131 (108-182)	129 (110-185)	0.995
WBC,10^3/dL	11.7 (9.5-14.6)	11.7 (9.5-14.6)	11.8 (10.1-14.7)	0.410
Hemoglobin, g/dL	13 (11-15)	14 (12-15)	14 (13-16)	0.146
Platelet count,10^3/dL	233 (197-278)	235 (197-277)	236 (201-287)	0.315
Lymphocyte, cells/µL	1.73 (1.2-2.3)	1.7 (1.2-2.4)	1.7 (1.2-2.5)	0.743
Neutrophils, cells/µL	8.9 (6.6-11.7)	8.8 (6.6-11.7)	9.3 (6.8-11.5)	0.442
CRP, mg/dL	0.9 (0.4-2.9)	0.9 (0.4-2.8)	1.1 (0.5-3.9)	0.101
Albumin, g/dL	3.8 (3.5-4.1)	3.6 (3.8-4.1)	3.7 (3.5-4.0)	0.140
Uric Asid, mg/dL	5.8(4.8-7.1)	5.9(4.8-7.0)	7.1 (6.4-7.9)	<0.001
UAR, mg/g	1.5(1.2-1.9)	1.5(1.2-1.9)	1.8 (1.7-2.0)	<0.001

p<0.05 was considered statistical significance.
CRP, C-reactive protein; DES, drug-eluting stent; DM, diabetes mellitus; e-GFR, estimated glomerular filtration rate; HDL-C, high-density lipoprotein cholesterol; HT, hypertension; HL, hyperlipidemia; LDL-C, low-density lipoprotein cholesterol; LMCA, left main coronary artery; UAR, uric asid/ albumin ratio; WBC, white blood cell

Table 2. Multivariate logistic regression analysis for ST prediction

Variables	OR	CI	P-value
UAR, mg/g	2.6	1.92-3.52	<0.001
DM	1.6	1.05-2.73	0.029
Age, years	1.01	0.99-1.03	0.71
Smoking	1.2	0.69-2.37	0.282

CI, confidens interval; DM, diabetes mellitus; OR, odds raito; UAR, üric asid-albumin ratio

PB-062 [Lipid / Protective Cardiology]

An assessment of cardiovascular health risks in people working from home during the pandemic period: PANDEV-KALP study

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Background and Aim: The COVID-19 pandemic had multi-faceted impacts on the working population who had to adapt to working from home (WFH). WFH has been reported to increase the rate of depression and anxiety due to social isolation; however, the cardiovascular effects of WFH are not well known yet. We aimed to assess the effects of WFH on cardiovascular risk factors and health behaviors during the COVID-19 pandemic.

Methods: Companies that employ WFH were invited to study by occupational health specialists via respective human resources departments. WFH employees were sent an online self-reported questionnaire which included demographics, WFH conditions, medical history, new complaints during the pandemic, health behaviors during and before the pandemic, and COVID-19 exposure. Participants were also asked to provide data on blood pressure, blood glucose, and lipid levels before and during the pandemic. Data collection started in January 2022 and ended in July 2022. Two hundred forty-five participants were invited, 208 completed the questionnaire (response rate: 84%), and 61 provided biological data (response rate: 25%).

Results: Demographic data are presented in Table 1. Regarding WFH, 72 (34.6%) participants had undergone training for WFH conditions, and 87 (41.8%) were able to give regular breaks. Weekly working hours have increased by 6 hours during the pandemic (49.6±13.8 vs. 43.8±12.1, p=0.001, t-test). Participants reported a median 7.5-point satisfaction regarding WFH on a 10-point scale. One hundred-twelve (53.8%) participants had a new complaint; the most common complaints were weight gain/increased appetite (73, 35.1%), insomnia/anxiety (58, 27.9%), and physical inactivity/musculoskeletal pain (38, 18.3%). One hundred-twenty (57.7%) participants had a weight increase, an increase in median BMI (p=0.001, Wilcoxon signed-rank test), and a shift toward pre-obesity was observed (p=0.001, chi-square test, Table 2). Most participants did not have changes in tobacco or alcohol consumption or exercise during the pandemic. Seventy-nine (37.9%) participants had a history of COVID-19 infection, and 165 (79.3%) had a relative infected with COVID-19. Data from the biological data subgroup did not show significant changes in blood pressure, blood glucose, or lipid levels.

Conclusions: WFH adversely affected modifiable cardiovascular risk factors, and was associated with weight gain, increased work hours, caused a lack of workload planning, and increased anxiety. Previous observational studies have reported increased sedentary behavior, alcohol and food intake, and weight gain. This study adds to the literature that although risk factors are adversely affected by WFH conditions, workers were satisfied with WFH. The training rate regarding WFH conditions was low in our study; therefore, we believe WFH workers must be informed about WFH conditions, and occupational health specialists should focus on reducing specific risk factors that exist during WFH.

Table 1. Demographic data of the participants

Age		40 (23-66)
Gender	Female	82 (39.4%)
	Male	126 (60.6%)
Education	High school	29 (13.9%)
	University	120 (57.7%)
	Higher education	52 (25%)
	Other	7 (3.4%)
Living conditions	Living alone	24 (11.5%)
	Spouse/partner	138 (66.3%)
	Baby/toddler	23 (11.1%)
	Preschool-aged child	26 (12.5%)
	Elementary school-aged child	43 (20.7%)
	High school-aged child	17 (8.2%)
	University-aged child	15 (7.2%)
	Parents	33 (15.9%)
	Other	4 (1.9%)
Comorbidities	Any	96 (46.2%)
	Hypertension	27 (13%)
	Diabetes and prediabetes	25 (12%)
	Musculoskeletal disorders	25 (12%)
	Migraine	23 (11.1%)
	Dyslipidemia	22 (10.6%)
	Coronary artery disease	13 (6.2%)
	Depression	7 (3.4%)
	Cerebrovascular disease	2 (1%)
	Other	7 (3.4%)

Table 2. Median body mass index and body mass index distribution of participants before and during the pandemic

		Before pandemic	During pandemic
BMI	median (interquartile range)	24.8 (15.4-44.4)	25.4 (15.4-46.2)
BMI distribution	Underweight (<18.5)	5 (2.4%)	2 (0.9%)
	Normal weight (18.5-24.9)	96 (46.1%)	88 (42.3%)
	Pre-obesity (25-29.9)	68 (32.6%)	79 (37.9%)
	Obesity class I (30-34.9)	24 (11.5%)	24 (11.5%)
	Obesity class II (35-39.9)	4 (1.9%)	5 (2.4%)
	Obesity class III (≥40)	2 (0.9%)	1 (0.4%)

PB-063 [Lipid / Protective Cardiology]

Acute effect of fast food consumption on hemodynamic parameters and arterial stiffness

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Background and Aim: Healthy diet is utmost important in cardiovascular disease risk management. Fast food contains high energy, sodium,sugar and saturated fat which has detrimental effect on cardiovascular health on long term. Arterial stiffness, as measured by pulse wave velocity, is an independent predictor of cardiovascular events and all-cause mortality. The aim of this study was to investigate whether consumption of fast food and sugar beverages has acute effect on arterial stiffness and hemodynamic parameters.

Methods: Twenty healthy subjects (10 female,10 male) were recruited into the study. They had routine blood tests (lipid profile, fasting blood glucose, complete blood count, kidney and liver function tests, uric acid) done within one week prior to fast food consumption. BMI, waist, hip,neck measurements,blood pressure measurements and EKG were done before fast food as well as baseline arterial stiffness. Total amount of calories were calculated according to basal metabolic rate and female subjects consumed 1420 calories containing fast food including hamburger, sugar beverage and fried potatoes whereas male subjects ingested 2270 calories with the similar content. Ingestion time was recorded. Arterial stiffness was then measured at 30 minutes, and repeated every hour up to 6 hours post prandial in every subject. Arterial stiffness was measured from brachial artery using mobile O-Graph arteriograph system.

Results: The mean age of subjects was 21.6± 1.1years. Systolic blood pressure increased (111.7± 9.1 mmHg vs 121.1±11.5 mmHg, p<0.001), diastolic blood pressure increased (71.4 ± 7.1 mmHg vs 73.8 ± 8.9 mmHg, p<0.001),pulse rate increased (79.7 ± 14.2 vs 85.8 ±12.1, p<0.001), augmentation index increased (14.5 ± 9.7 % vs 21.1± 6.3,% p=0.002) and pulse wave velocity increased (4.67 ± 0.27 m/sec vs 4.92 ± 0.35 m/sec, p=0.01) significantly compared to baseline values with 30 minutes after consumption of high calorie fast food, respectively. The values returned to baseline values after one hour of fast food consumption except pulse rate. The change in pulse wave velocity (arterial stiffness) between baseline and 30 minutes after ingestion of fast food was correlated interestingly with neck circumference (r=0.5,p=0.02). There was also a trend with waist/hip ratio (r= 0.4,p=0.07).

Conclusions: Consumption of fast food increased pulse wave velocity, arterial stiffness, acutely even in healthy young subjects. It may have robust detrimental acute effects on patients with known cardiovascular disease

Table 1. Changes in hemodynamic parameters and arterial stiffness after fast food consumption

	Baseline	30 Minutes PP	1-Hour PP	2-Hour PP	3-Hour PP	4-Hour PP	5-Hour PP	6-Hour PP	P-value
P-SBP	117.9±9.1	121.±11.5	114.±12.4	115.±12.4	114.6±9.6	113.4±9.6	113±10.8	109±13.1	<0.001
P-DBP	71.4±7.1	73.8±8.9	70.7±9.1	69.9±7.6	68.8±7.9	69.7±8.2	68.6±8.4	66.7±7.9	<0.001
Pulse	79.7±14.2	85.8±12.1	89.2±14.8	87.5±13.4	86.9±13.3	85.5±11.6	82.1±11.6	80.1±11.4	<0.001
Central SBP	99.3±6.6	106.4±9.8	99.5±7.3	98.9±6.9	99.2±9.1	99.6±7.2	99.8±7.9	95.1±8.1	<0.001
Central DBP	73±7.1	77.3±9.7	72.4±9.2	71.7±7.6	70.8±8.2	71.4±8.1	70.2±8.7	68.5±8.2	<0.001
SVR	1.1±0.19	1.16±0.08	1.13±0.13	1.11±0.17	1.11±0.16	1.01±0.16	1.08±0.12	1.12±0.17	0.022
AI	14.5±9.7	21.1±6.3	20.9±9.4	20.2±8.4	19.7±9.4	19.8±7.7	15.1±7.5	17.1±7.2	0.002
PWV	4.67±0.27	4.92±0.35	4.72±0.25	4.78±0.39	4.73±0.28	4.73±0.29	4.71±0.32	4.57±0.38	0.010

P, Peripheral; PP, Pst-prandial; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; SVR, Systemic vascular resistance; AI, Augmentation index; pWV, pulse wave velocity

PB-064 [Lipid / Protective Cardiology]

The relationship of serum vaspin level with the extent and severity of coronary artery disease in patients with non-diabetic acute coronary syndrome

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Background and Aim: Atherosclerosis, the most common reason for coronary artery disease, is the leading cause of mortality and morbidity in all over the world. Adipocytokines originating from adipose tissue are thought to be atherosclerosis participants. These adipocytokines can affect vascular wall homeostasis by affecting the function of endothelial cells, arterial smooth muscle cells and macrophages in vascular walls. Vaspin is a recently identified insulin-responsive adipocyte that is released from the visceral fat tissue, a member of the serine protease inhibitor family. It has been shown to be associated with obesity, insulin resistance and diabetes. We aimed to investigate the relationship between serum vaspin level and the diffusiveness end severity of coronary artery disease determined by the SYNTAX score in patients with non-diabetic acute coronary syndrome.

Methods: This study enrolled 165 non-diabetic patients who were admitted to our hospital, due to acute coronary syndrome without ST segment elevation, and who underwent coronary angiography. Serum vaspin levels of patients were measured by ELISA and SYNTAX scores were calculated by examining coronary angiography images. The SYNTAX score was divided into three groups by the interquartile range method (group 1 with SS <10, group 2 with 10 ≤ SS≤22, group 3 with SS> 22).

Results: In group 1 with low SYNTAX score, age was lower than the other two groups (56,4 ± 12,6; 62,5 ± 11,4; 62,5 ± 12,4; p = 0,01). The left ventricular ejection fraction was found to be lower in group 3 with higher SYNTAX score (55.2 ± 6.7, 52.9 ± 7.8, 49.9 ± 8.3, p = 0.002). Total cholesterol, LDL cholesterol and creatinine values were found to be higher in group 3 with high SYNTAX score (p = 0,01, p = 0,008, p = 0,006). Serum vaspin level was highest in group 1 and lowest in group 3 (518,9 ± 66,8; 343,3 ± 45,7; 281,1 ± 26,3 pg/ml; p = 0,03). In the Pearson correlation analysis, there was a weak negative correlation between the SYNTAX score and serum vaspin level (r=-0,207, p=0.008).

Conclusions: There was a weak negative correlation between the serum vaspin level and the diffusiveness and severity of coronary artery disease determined by SYNTAX score in non-diabetic patients with acute coronary syndrome, and as the SYNTAX score increased, serum vaspin level decreased.

PB-065 [Lipid / Protective Cardiology]

The alteration in serum oxidative stress balance in isolated patients with different high-density lipoprotein cholesterol circulating levels

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Background and Aim: High density lipoprotein cholesterol (HDL-C), known as a key player in reverse transport of cholesterol and this function is associated with its athero-protective role. Low HDL-C (LHC) are a strong, independent risk factor for cardiovascular (CV) disease, premature atherosclerosis and increased oxidative stress. However, the clinical benefit of High HDL-C (HHC) through diet or drug therapy is still controversial.

Methods: This study included 'isolated' 50 patients with LHC (≤35 mg/dL), 'isolated' 52 patients with HHC (≥70 mg/dL) and 33 age- and sex-matched control patients with normal HDL-C. 'isolated' was defined as excluding all clinical conditions associated with increased oxidative stress and inflammation which may affect the structural state of HDL-C. In addition to Arylesterase activity and plasma thiol levels, laboratory parameters associated with oxidative stress were also evaluated.

Results: The levels of arylesterase [758 (169-1150) vs. 945 (480-1215); 821 (266-1220); p < 0.01] and total thiols [233±41 vs. 259±46; p = 0.02] were remarkably higher in patients with HHC (Figure 1). More importantly, TAC, OSI, creatine and serum ARES levels were associated with change in serum HDL-C levels (Table I, Figure 2).

Conclusions: In isolated HHC patients, we determined that while serum ARES activity and plasma thiol concentrations were significantly higher, the other markers associated with oxidative stress remarkably decreased. Additionally, the present study demonstrated that the serum oxidative stress state is very important to maintain the positive effects of HDL-C.

Table 1. Linear regression analysis of the effect of TAS, TOS, OSI, creatinine and ARES levels on plasma HDL-C levels as a dependant variable

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
1(constant)	66,528	3,184	-,466	20,895	0,000	60,244	72,831
OSI	-,559	,097		-5,790	0.000	-,750	-,368
2(constant)	142,910	17,257		8,281	0,000	108,741	177,078
OSI	-,536	,090	-,446	-5,958	0.000	-,713	-,358
TAC	-58,296	12,976	-,336	-4,493	0,000	-83,989	-32,604
3(constant)	112,043	19,071		5,875	0,000	74,280	149,806
OSI	-,459	,089	-,383	-5,131	0.000	-,636	-,282
TAC	-53,603	12,559	-,309	-4,268	0,000	-78,471	-28,734
ARES	,027	,008	,246	3,284	0.001	0,11	0,43

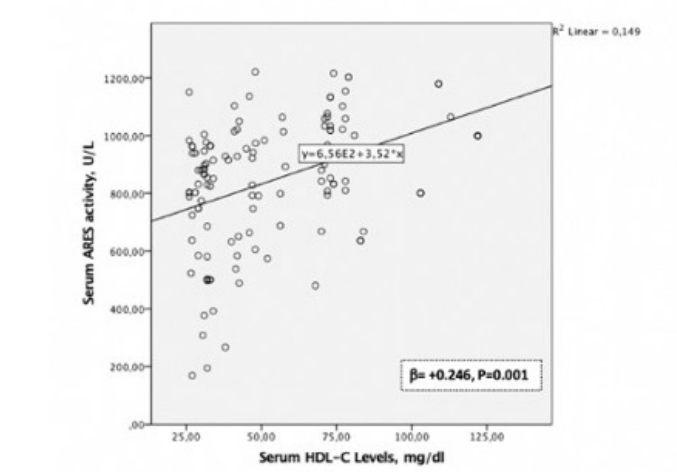

Figure 1. Lineer regression analysis between serum ARES activitiy and HDL-C levels. Serum ARES activity levels (b= +0.246, 95% CI: 0.11-0.43, p=0.001) were dependently associated with HDL-C values

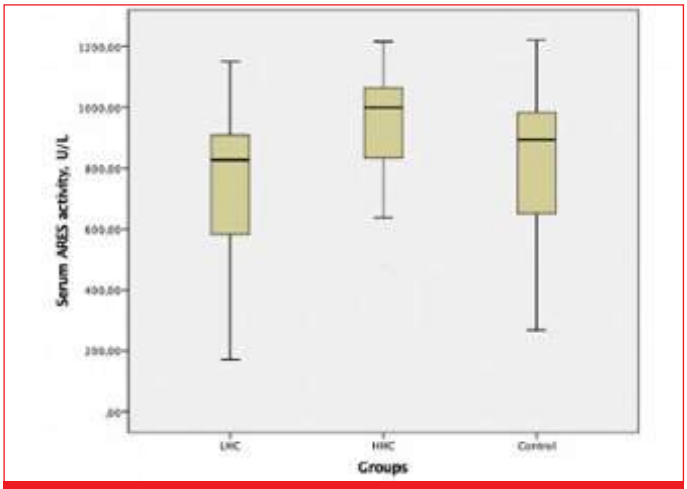

Figure 2. The change in serum ARES activity between LHC, HHC and control groups

PB-066 [Lipid / Protective Cardiology]

Do statins counteract the effect of antidiabetic drugs? Results of the SCEAD study

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Background and Aim: Diabetes and dyslipidemia are a leading cause of mortality and morbidity. According to international guidelines statins are the therapeutic cornerstone in patients with diabetes and/or dislipdemia. However statins and antidiabetic agents have an opposite pharmacological effect, because statins, particularly atorvastatin and rosuvastatin, impair glucose homeostasis, increasing the risk of new-onset diabetes, while antidiabetic drugs improve glycemic homeostasis. The aim of our study was to investigate the interference of atorvastatin, rosuvastatin and pitavastatin on plasma glucose in patients with type 2 diabetes (T2DM) and dyslipidemia, during stable treatment with hypoglycemic drugs.

Methods: The study, was a prospective, randomized, open label, parallel groups, with blinded-endpoints (PROBE design). Of recruited 180 recruited patients with T2DM and dyslipidemia, 131 were randomized to: atorvastatin, (n=44, group ATV), rosuvastatin (n=45, group ROS) and pitavastatin (n=42, group PTV) and completed the 6 months of follow-up.

Results: At end of treatment, patients assigned to atorvastatin and rosuvastatin displayed a marginal, not significant, decrease in fasting plasma glucose (FPG) (FPG: -3.5 and -6.5 mg/dl, both p=NS). On the other hand, in patients treated with pitavastatin FPG decreased significantly (-19.0 mg /dl, p<0.01). Mean glycated hemoglobin A1c (HbA1c%) remained almost stable during treatment with atorvastatin and rosuvastatin (-0.1%, and +0.2%, both p=NS), while it significantly decreased with pitavastatin (-0.7%, p=0.03). Atorvastatin, rosuvastatin and pitavastatin significantly decreased (p<0.001) plasma levels of total cholesterol (TC), low-density lipoprotein-cholesterol (LDL-C), and triglycerides (TG), while high-density lipoprotein-cholesterol (HDL-C) increased significantly (p=0.04) only in the pitavastatin group.

Conclusions: The results of the present study suggest that, at variance from atorvastatin and rosuvastatin, pitavastatin interferes to a lesser extent with hypoglycemic agents in patients with T2DM. This observation has clinical relevance and allow to choose the most appropriate statin therapy in patients with dyslipidemia and diabetes. The efficacy on lipid profile was not significantly different between the three statins, with the exception of HDL-C level which increased significantly (p=0.04) with pitavastatin. The favourable effect of pitavastatin on glucose homeostasis could be associated with the increase of plasma adiponectin level or with a minor attenuations in insulin signaling in adipocytes.

Table1. Changes of Lipids from baseline

Parameters	Drugs	Mean changes (SD) from baseline	P vs baseline	P between drugs
TC	ATV ROS PTV	-74.2 (31.8) -70.9(31.9) -71 (49.5)	<0.001* <0.001* <0.001*	0.38**
LDL	ATV ROS PTV	-65.7 (21.5) -64.6 (28.2) -62.6 (30.5)	<0.001* <0.001* <0.001*	0.58**
HDL	ATV ROS PTV	+0.65 (4.1) +0.38 (8.0) +1.1 (3.1)	0.28* 0.75* 0.04*	0.86**
TG	ATV ROS PTV	-32.8 (71.4) -27.7 (64.4) -33.1 (73.8)	0.001* 0.001* 0.001*	0.92**

TC = Total Cholesterol
LDL= Low Density Lipoproteins
HDL = High Density Lipoproteins
*t Student **ANOVA

Table 2. Demographic, clinical and biochemical characteristics of the study participants at baseline

	Atorvastatin (n= 44)	Rosuvastatin (n= 45)	Pitavastatin (n= 42)	p
Male, n (%)	20 (45.4)	13 (28.8)	14 (26.9)	0.24°
Age, years, mean (SD)	58.8±9.7	58.7±12.0	57.9±9.4	0.91#
Dose	20 mg	10 mg	2 mg	
Hypertension, n (%)	15 (34.1)	17 (37.8)	11 (26.2)	0.50°
Antidibetic drugs n (%) -metformin, n (%) -metformin combinations n (%)* -others (%)**	44 (100) 8 (18.2) 23 (52.2) 4 (9.0)	45 (100) 10 (22.2) 20 (44.4) 2 (4.4)	42 (100) 9 (21.4) 21 (50.0) 3 (7.1)	 0.88° 0.75° 0.68°
FPG (median-interquartiles)** Haemoglobin A1c % (median-interquartiles)	134 (122.5-149.7) 6.0 (6.5-7.8)	134.0 ((121.0-147.0) 6.1 (6.8-8.2)	139.0 ((112.0-185.3) 7.5(6.4-9.4)	0.59°° 0.12°°
Total Cholesterol (mg/dL)	255.9±42.2	256.6±47.8	259.1±39.8	0.99#
LDL-C (mg/dL) mean (SD)	173.8±35.0	174.5±39.4	176.1±33.0	0.98#
HDL-C (mg/dL) mean (SD)	47.2±10.2	48.9±9.1	49.0±8.7	0.60#
Triglycerides (mg/dL) mean (SD)	177.5±64.4	172.6±68.2	179.5±66.9	0.88#

FPG = Fasting plasma glucose;
°° Kruskal Wallis, ° Chi square test, # ANOVA
*metformin+ GLP-1 RA or DPP4 or SGLT-2 inhibitors. **insulin+ metformin or DPP4 inhibitors
** 25%-75% interquartiles

Table 3. Plasma Glucose and HbA1c changes from baseline

Parameters	Drugs	Mean changes from baseline ^^	P vs baseline	P between drugs		
Plasma glucose (mg/dl) (median-IQR)	ATV ROS PTV	-3.5(-21.7, 6.7) - 6.5 (-13.0, 3.0) -19.0 (-40.0, -1.5)	0.42* 0. 17* <0.001*	0.03 **		
HbA1c % (median-IQR)	ATV ROS PTV	-0.10 0.20 -0.75	0.53* 0.40* 0.01*	0.01**		

^^ Mean changes, from baseline, calculated from the difference of individual subject data
*Mann Witheny, **Kruskall-Wallis test, , IQR= 25%-75% interquartile range

PB-067 [Pulmonary Hypertension / Pulmonary Vascular Diseases]

Is It Possible To Predict Mortality By Using Hematologic Parameters In An Emergent Condition?

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Background and Aim: The pulmonary embolism severity index (PESI), is a well-established parameter that could detect early mortality risk in acute pulmonary embolism (PE). PESI is mainly based on clinical parameters, but its sensitivity has been shown to be improved using additional biomarkers. Hemogram parameters such as mean platelet volume (MPV), neutrophil/lymphocyte ratio (NLr), eosinophil, red cell distribution width (RDW) and platelet distribution width (PDW) are widely used as inflammatory markers to assess prognosis in various cardiovascular diseases. In this study, we aimed to investigate the role of hemogram parameters in predicting the mortality in patients presenting with acute PE.

Methods: Patients hospitalized with a diagnosis of PE in our tertiary center between 2016 and 2021 were included in this retrospective study. PE was diagnosed and treated according to current guidelines.Demographic, clinical and laboratory data were reviewed from the hospital database. In-hospital mortality data were documented and patients were divided into two groups according to in-hospital mortality. PE related parameters such as treatment with thrombolytic agents, transfer to intensive care unit or mechanic ventilation requirement were noted.

Results: There was a total of 254 patients (37.4% male). Mean age of the study cohort was 62.7±15.5. There were 77(30.3%) patients that received thrombolytic treatment. 38 patients formed the in-hospital mortality group. History of malignancy and heart failure were significantly higher in the non-survivor group. Regarding labaratory markers; uric acid, BUN, CRP, Tn, NT-proBNP, and D-dimer, leukocyte, neutrophil, MPV, RDW, PDW and NLr were significantly higher and serum sodium, albumine and GFR were significantly lower in non-survivor group. In terms of echocardiography findings, the non-survivor group had significantly lower ejection fraction and higher pulmonary artery pressure and right ventricular dimension. Furthermore, PESI and simplified PESI were significantly higher in non-survivor group.

Multivariate logistic regression analysis revealed that PESI(p<0.0001),RDW(p=0.001) and NLr(p=0.007) were independent risk factors associated with in-hospital mortality. A cut-off value of 141.5 for PESI score was associated with 76.3% sensitivity and 75.9% specificity; 5.9 for NLr was associated with 68.4% sensitivity and 68.1% specificity; 14.1 for RDW was associated with 68.4% sensitivity and 62.6% specificity in predicting in-hospital mortality.

Conclusions: Hematological parameters, assessed by routine blood count analysis on admission may serve as a promising and useful tool for predicting in-hospital mortality of patients presenting with acute PE especially when used in combination with traditional risk scores. Thus, concomitant use of hematological parameters and PESI to define high-risk normotensive and hypotensive PE patients and to predict poor prognosis may be clinically applicable with high specificity and positive probability.

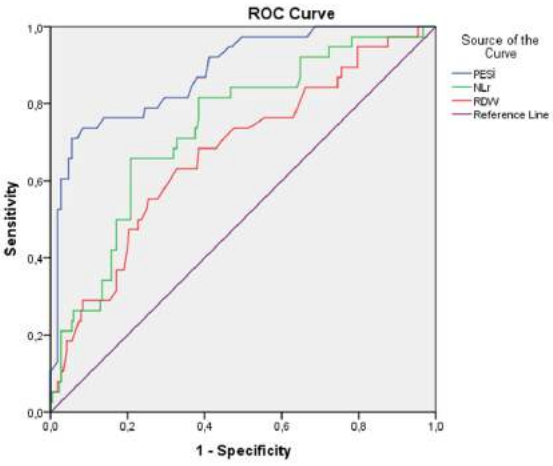

Figure 1. ROC curves of PESI score, RDW and NLr for predicting in-hospital mortality

Table 1. Clinical, demographic and laboratory variables according to in-hospital mortality

Variables	All n=254	Survivor (n=216)	Non-survivor (n=38)	p
Clinical Characteristics				
Age (years)	62.7±15.5	61.9±15.1	66.5±16.9	0.096
Male, n (%)	95 (37.4)	82 (37.9)	13 (34.2)	0.659
Body mass index (kg/m^2)	29.4±5.7	28.7±4.8	29.6±6.1	0.574
Heart Rate>110, beats/min, n (%)	252 (99.2)	214 (99.1)	38 (100)	0.551
Respiratory Rate>30, times/min, n (%)	51 (20.1)	25 (11.6)	26 (68.4)	**<0.0001**
Ssyatolic Arterial Pressure<100, mmHg, n (%)	144 (56.7)	120 (55.6)	24 (63.2)	**0.047**
Saturation O_2<90, n (%)	201 (79.1)	170 (78.7)	31 (81.6)	0.055
Mental status, n (%)	25 (9.8)	9 (4.2)	16 (42.1)	**<0.0001**
Body Temperature (°C)<36, n (%)	8 (3.1)	4 (1.9)	4 (10.5)	**0.019**
Right ventricle/Left ventricle > 1(Computerized Tomography), n (%)	164 (64.6)	131 (60.6)	33 (89.2)	**0.001**
Comorbidity				
Hypertension, n (%)	127 (50.0)	110 (50.9)	17 (44.7)	0.482
Diabetes Mellitus, n (%)	56 (22.0)	49 (22.7)	7 (18.4)	0.559
Previous coronary artery disease, n (%)	34 (13.4)	29 (13.4)	5 (13.2)	0.964
Previous congestive heart failure, n (%)	9 (3.5)	6 (2.7)	3 (7.9)	**0.048**
Previous Malignancy, n (%)	23 (9.1)	12 (5.6)	11 (28.9)	**<0.0001**
Chronic obstructive pulmonary disease, n (%)	33 (13.0)	31 (14.4)	2 (5.3)	0.094
Previous cerebrovascular Accident, n (%)	11 (4.3)	9 (4.2)	2 (5.3)	0.510
Deep venous thrombosis, n (%)	49(19.3)	39(17.8)	10(27.1)	0.202
PESI	132.8±39.7	122.8±29.3	189.4±43.8	**<0.0001**
Simplified PESI≥1	132(52)	104(48.1)	28(73.7)	**<0.0001**
Laboratory findings				
Haemoglobin, (g/dl)	12.2±1.9	12.4±1.9	11.9±2.2	0.185
Platelet, (10^3 /µl)	228.5±79.8	227.9±75.4	231.6±100.3	0.799
Leukocytes, (10^3 /µl)	10.7±3.5	10.2±3.2	13.2±3.8	**<0.0001**
Neutrophile, (10^3 /µl)	7.2±3.3	6.8±3.1	8.9±3.9	**<0.0001**
MPV, fL	8.5±1.2	8.2±2.5	11.2±3.6	**0.024**
Red cell distribution width	14.3±2.1	14.1±1.8	15.6±2.9	**<0.0001**
Platelet distribution width	16.9±9.4	14.2±3.5	17.3±10.1	**0.001**
Neutrophile/lymphocite ratio (NLr)	5.2±4.1	4.7±3.8	7.4±6.1	**0.013**
Serum creatinine, (mg/dl)	1.1±0.8	1.1±0.9	1.2±0.5	0.130
BUN, (mg/dl)	46.4±25.4	41.9±20.1	72.1±35.9	**<0.0001**
Glomerular filtration rate, (mL/dk/1.73m^2)	77.1±27.2	80.6±26.1	57.3±25.1	**<0.0001**
Sodium, (mmol/L)	138.5±4.3	138.8±3.9	136.8±6.1	**0.009**
Potassium, (mmol/L)	4.3±0.5	4.3±0.5	4.5±0.6	0.075
Glucose, (mg/dL)	159.6±75.1	158.3±78.1	167.0±55.3	0.511
Uric acid, (mg/dL)	5.9±2.2	5.7±1.9	7.8±2.9	**<0.0001**
Albumine, (g/dl)	3.7±0.5	3.8±0.5	3.2±0.7	**<0.0001**
C-reactive protein, (mg/dL)	24.4 (0.52-300)	40.5 (0.52-272)	66.1 (6.1-300)	**0.003**
Troponin I, (pg/ml)	120.0 (2-6000)	212.3 (2-1625)	1051.2 (10-6000)	**0.014**
NT-proBNP, (ng/mL)	4328(53.7-14800)	3863.9(53.7-10400)	10800(5890.7-14800)	**0.031**
D-dimer, (ng/mL)	4.8(0.12-35.9)	4.2(0.12-12.8)	10.1(0.13-35.9)	**0.046**
Echocardiography Findings				
Left ventricular ejection fraction, (%)	55.9±4.8	56.5±4.5	53.2±5.7	**<0.0001**
Left ventricular end diastolic dimension, (mm)	44.7±3.6	44.7±3.4	44.2±4.2	0.357
Right ventricular dimension,(mm)	35.6±5.7	34.9±5.6	39.2±4.8	**<0.0001**
RVD/LVD ratio >1	21 (8.3)	14 (6.5)	7 (18.4)	**0.023**
Pulmonary artery systolic pressure, (mmHg)	47.6±10.9	46.3±10.6	54.9±9.7	**<0.0001**
In-hospital Outcomes				
Length of Hospital Stay, n (days)	6.1±4.9	6.6±4.2	5.4±4.8	**0.031**
Intensive care unit admission, n (%)	171(67.3)	136 (62.1)	35 (92.1)	**<0.0001**
Patients receiveing trombolytic therapy, n (%)	77(30.3)	55(25.5)	22(57.9)	**<0.0001**
Advanced Ventilatory Support, n (%)	39(15.4)	11(5.1)	28(73.7)	**<0.0001**

Tablo 2. Univariate and multivariate forward stepwise logistic regression analysis: predictors of in-hospital mortality

	Univariate OR	95% CI	p	Multivariate OR	95% CI	p
PESI	1.043	1.031-1.056	**<0.0001**	1.035	1.018-1.053	**<0.0001**
Leukocytes	1.061	1.005-1.121	**0.034**	0.956	0.737-1.240	0.956
Neutrophil	1.189	1.077-1.314	**0.001**	0.835	0.600-1.161	0.284
RDW	1.329	1.139-1.551	**<0.0001**	1.548	1.186-2.020	**0.001**
PDW	0.936	0.872-1.004	0.064			
MPV	1.339	1.007-1.781	**0.045**	1.152	0.517-2.570	0.729
NLr	1.259	1.140-1.390	**<0.0001**	1.529	1.125-2.078	**0.007**
C-reactive protein	1.009	1.003-1.025	**0.004**	1.001	0.986-1.015	0.919
Troponin I	1.007	1.001-1.016	**0.001**	0.987	0.952-1.002	0.085
NT-proBNP	1.008	0.821-1584	0.997			
D-dimer	0.932	0.843-1.030	0.168			

PB-068 [Pulmonary Hypertension / Pulmonary Vascular Diseases]

The relationship between vitamin D level and pulmonary artery stiffness

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Background and Aim: Vit D has a role in controlling the function of vascular smooth muscle cells and endothelial cells, even in the pulmonary artery. We hypothesized that pulmonary artery elasticity could be affected in patients with relatively low vitamin D levels.

Methods: Young adults with the complaint of shortness of breath were enrolled. Patients were divided into 2 groups according to their vit D levels with a cut-of value of 20 ng/mL. Pulmonary artery stiffness (PAS) was calculated by using the following formula PAS (kHz/sec) = Maximal frequency shift / pulmonary acceleration time. The six-minute walk distance (6MWD) was measured to evaluate the functional exercise capability of subjects.

Results: Subjects with low vit D levels had lower 6MWD compared to those of higher vit D levels (443.58 ± 56.20 m vs 483.20 ± 58.43 m, p = 0.007). The PAS was significantly increased in patients with Vit D level < 20 ng/mL compared with subjects with Vit D level > 20 ng/mL (11.65 ± 3.76 vs. 9.46 ± 2.53, respectively, p = 0.011). In multivariate linear regression analysis, 1,25 Vit D level was found to be predictive of PAS with lower values related to increased PAS (β=-0.280,p=0.009).

Conclusions: We found that PAS might seem to be affected by the low level of vit D. To care screening of vit D deficiency might be used to gain insight into the mechanics of the pulmonary artery vasculature, even in the absence of significant pulmonary artery pressure elevation.

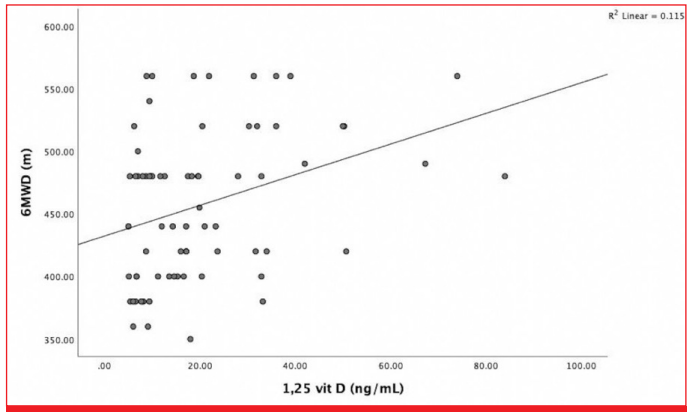

Figure 1. Correlation graph between 1,25 Vit D level and 6MWD

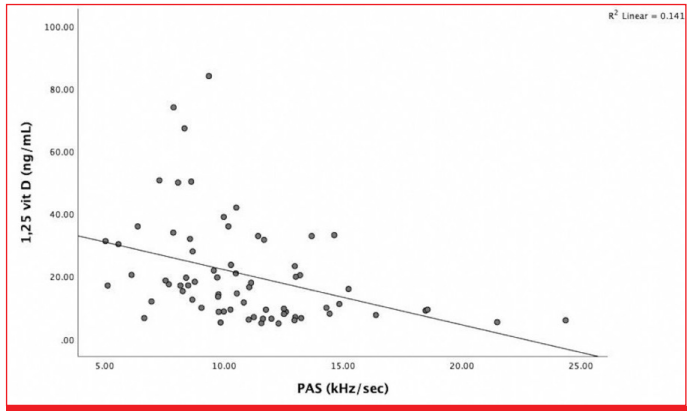

Figure 2. Correlation graph between PAS and 1,25 Vit D level

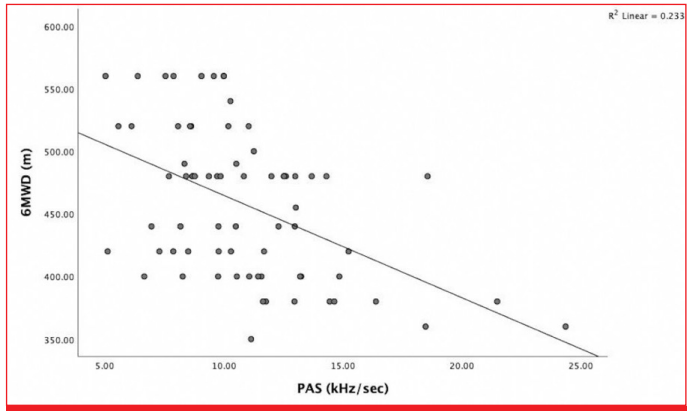

Figure 3. Correlation graph between PAS and 6MWD

PB-069 [Lipid / Protective Cardiology]

The impact of participants' educational level on the successful implementation of a structured-enhanced education and follow-up program focused on the prevention of coronary artery disease

Dilanur Özveri[1], Meyrem Yazdıç[1], Ahmet Kaya[1], Nur Acar[1], Gamze Sofuoğlu[1], Halil İbrahim Deniz[1], Bayan Zatari[1], Pınar Günel[1], Özlem Soran[2]

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[2]*Department of Cardiology, Pittsburgh University, Pittsburgh, PA, USA*

Background and Aim: Primary prevention programs for coronary artery disease (CAD) may be effective in improving health-related behavioral outcomes. However, successful implementation of these programs may depend on the educational level of the participant at the time of the program entry. The primary aim of this study was to assess the impact of the participants' level of education on the effectiveness of a structured, enhanced education and follow-up program for CAD prevention.

Methods: SANKO CAD Prevention Project was designed as a longitudinal study and utilized medical school students to conduct the project under the supervision of professors. It had two different education and training phases. In the first phase, every school year for second year Medical students underwent a year-long, especially designed training program on primary prevention for CAD. In the second phase, which took place in the second year of the study, a series of conferences regarding the primary prevention for CAD were organized by the University and local municipalities for underserved populations. Participants were prospectively assigned to an intervention where pre- and post-conference knowledge was collected and assessed. Every intervention was conducted by specially trained third year Medical students and an education booklet, which was specially designed for the study, was given to the participants. Every other month thereafter, for 6 months, each participant was followed-up via phone calls. At the 6 months follow -up, data was collected to assess the impact of the program on behavioral outcomes.

Results: A total of 172 participants were divided into two groups. Participants in Group A had less than a high school diploma, while those in Group B (n=111) had a high school diploma or higher. Mean age was 40±11.9 years with no significant difference in between the groups. Group A had a higher proportion of women (85% vs. 48%, p<0.001) and a 15% working rate (p<0.001). While BMI rates were higher in Group A (30.09±5.45 kg/m^2, p<0.001), smoking rates were similar in both groups.

Overall, knowledge on CAD risk factors, primary prevention measures were poor in both groups. After the implantation of the program, comparing to baseline, there was a significant improvement on the study measures in both groups (p<0.001). However, there was no statistically significant difference in between the groups. Importantly, the follow-up program led both groups to implement those positive changes into their lives and maintain a healthy lifestyle.

Conclusions: This study results showed that a longitudinally structured training program for medical students could be utilized to implement an enhanced education and follow–up program for primary prevention of CAD. Moreover, the successful outcomes were independent of the participant's levels of education at the time of the program entry. This model program is beneficial for public interest and enhances active interaction of medical students with patients at an early stage of their career.

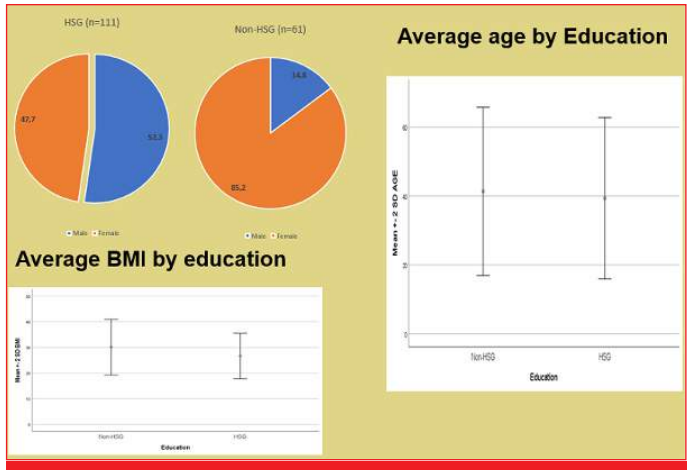

Figure 1.

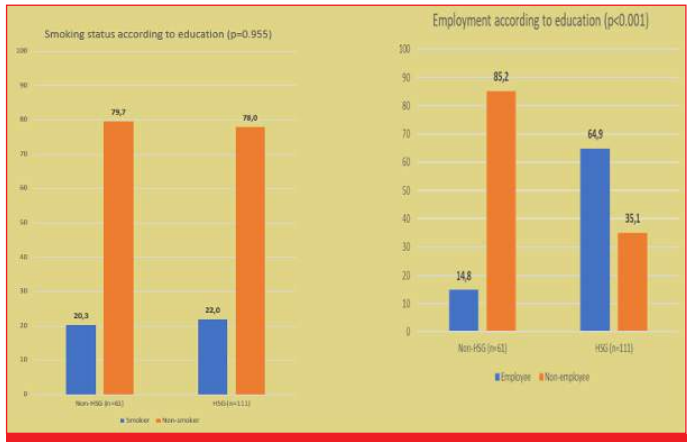

Figure 2.

Surname Name	Surname Name	Pub Number
Acar, Burak	Acar, B.	SB-003
Acar, Emrah	Acar, E.	PB-043, SB-010
Acar, Nur	Acar, N.	PB-069
Ağır, Ayşen	Ağır, A.	SB-003
Agus, Hicaz Zencirkıran	Agus, H. Z.	SB-077
Akbay, Ertan	Akbay, E.	PB-024
Akbulut, Muge	Akbulut, M.	PB-044
Akburak, Fatih	Akburak, F.	PB-063
Akçay, Seckin	Akçay, S.	PB-038, PB-041, PB-045
Akgüllü, Çağdaş	Akgüllü, Ç.	PB-015
Akgün, Didar Elif	Akgün, D. E.	SB-045, SB-084
Akıcı, Ahmet	Akıcı, A.	PB-062
Akıllı, Azem	Akıllı, A.	SB-025
Akın, Kaan	Akın, K.	SB-075
Akıncı, Sinan	Akıncı, S.	PB-024
Akkuş, Oğuz	Akkuş, O.	PB-055, SB-024, SB-038
Aksoy, Fatih	Aksoy, F.	PB-029
Aksoy, Şevket	Aksoy, Ş.	PB-062
Aksu, Ekrem	Aksu, E.	SB-056, SB-057
Aksu, Tolga	Aksu, T.	SB-069
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