39th NATIONAL CARDIOLOGY CONGRESS

ORAL PRESENTATIONS

THE ANATOLIAN JOURNAL OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Interventional Cardiology / Coronary

OP-001

The determinants of delays and their impact on clinical end points in acute ST-segment elevation myocardial infarction: A single center experience

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Background and Aim: The purpose of this study was to determine the factors that cause delay time in patients admitted to the hospital with ST-segment elevation myocardial infarction (STEMI). In addition, the effect of this delay on the patient's prognosis has also been investigated.

Methods: 301 patients diagnosed with STEMI treated with primary percutaneous coronary intervention were included into the study. Re-infarction, revascularization, cerebrovascular event, and cardiac death were determined as major cardiac clinical events. The follow-up period of our study was 475 ± 193 days.

Results: In the univariate analysis of the factors that determine the system delay time (120 min) was the education level, diabetes, and variability in pain intensity. In multivariate logistic regression analysis, diabetes, variation in pain intensity, and infarct related artery other than left anterior descending artery (LAD) were found as the independent factors which increased delay times. We determined the cut-off values predicting the composite endpoint as 122.5 minutes for patient delay, 95.5 minutes for system delay and 371 minutes for total ischemic time. It was observed that the in-hospital N-terminus pro-B-type natriuretic peptide (NT-pro-BNP) values of the patients presenting early were lower (181 vs. 594 pg/mL p<0.001), had a higher ejection fraction at the first measurement and even improved at the 6th week of follow-up (p=0.047).

Conclusions: The causes of prolong ischemic duration were hypertension, diabetes mellitus, intermittent angina pectoris, ejection fraction 40%, culprit vessel other than LAD, multi-vessel disease, body mass index, smoking, previous admission to hospital with angina, level of education. This study investigated the causes of the delay time and to determine their relationship with major cardiac clinical events.

Chart 1: Major Cardiac Clinical Events In Early And Late Group According to Total Ischemic Time

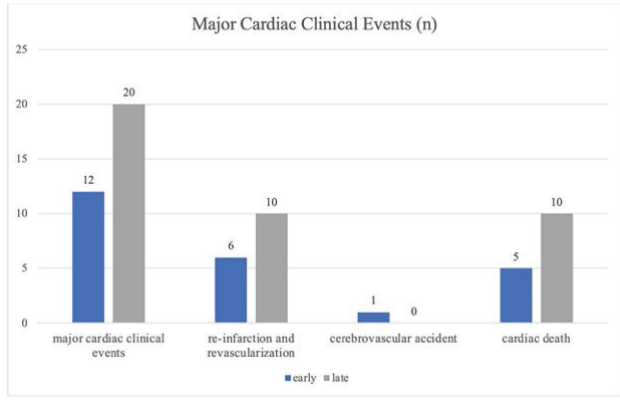


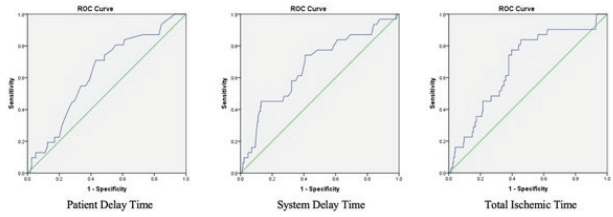
Figure 1. Major cardiac clinical events in early and late group according to total ischemic time.

Table 1. ROC analysis of the ischemic time

Table ROC analysis of the ischemic time

	Area under the curve (95% confidence interval)	Cut-off value (min)	p- value	Sensitivity (%)	Specificity (%)
Patient delay time	0.632 (0.536-0.762)	122.5	0.016	58.1	38.9
System delay time	0.672 (0.569-0.775)	95.5	0.002	61.3	61.5
Total ischemic time	0.677 (0.581-0.772)	371	0.001	64.5	62.2

Picture 1: ROC curves



Interventional Cardiology / Coronary

OP-002

Single long stent and overlapping multiple stents in long coronary lesions: Which is more beneficial in long-term follow-up?

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Background and Aim: Percutaneous coronary intervention (PCI) with single long stents (SLS) and multiple overlapping stents (OMS) is performed to treat long coronary lesions. The aim of our study is to compare the long-term follow-up results of patients who underwent SLS and OMS.

Methods: Patients who were admitted to the hospital with a diagnosis of non-ST-segment elevation myocardial infarction between January 2018 and February 2020, were under the age of 75, and had a drug-eluting stent of at least 40 mm in length were retrospectively examined. Patients with left main coronary artery lesions, shock, severe renal and liver failure, malignancy and hematological disorders were not included in the study. The patients were followed for 3 years. All patients received ticagrelor and acetyl salicylic acid (ASA) for the first year. Afterwards, they only received ASA. Study endpoints were stent thrombosis, stent restenosis, and mortality.

Results: The mean age of the patients was 62.1 ± 8.1 and 64.9% were male. SLS was placed in 52 of the patients, while OMS was placed in 59 of them. There was no significant difference between the groups in terms of age, gender and comorbidities. Although the rate of stent restenosis was found to be lower in the SLS group, there was no statistically significant difference (5.8% vs. 11.9%, p=0.331). Mortality and stent thrombosis were also similar between the groups. The basic demographic characteristics of the patients are summarized in Table 1. Total stent length and contrast volume were significantly higher in the OMS group (42.1 ± 2.6 vs. 48.2 ± 2.9 p<0.001 and 197.1 ± 31.7 vs. 256.9 ± 30.5 p<0.001, respectively). Kaplan-Meier analysis was performed to examine the relationship between the SLS and OMS groups and mortality, stent restenosis, and stent thrombosis during a mean follow-up of 36 months. Accordingly, there was no significant difference between the groups in terms of mortality, stent restenosis and stent thrombosis during the follow-up period (Log-rank: 0.398, p=0.528, Log-rank: 1.142, p=0.285 and Log-rank: 0.932, p=0.761, respectively) (Figure 1).

Conclusions: SLS and OMS showed similar clinical results in long lesions of the coronary arteries. There were similar rates of mortality, stent restenosis, and stent thrombosis. The SLS group had lower contrast volume and shorter stent length. These results show that OMS can be preferred instead of SLS in anatomically difficult and complex lesions. Multicenter studies with larger number of patients are needed to support our findings.

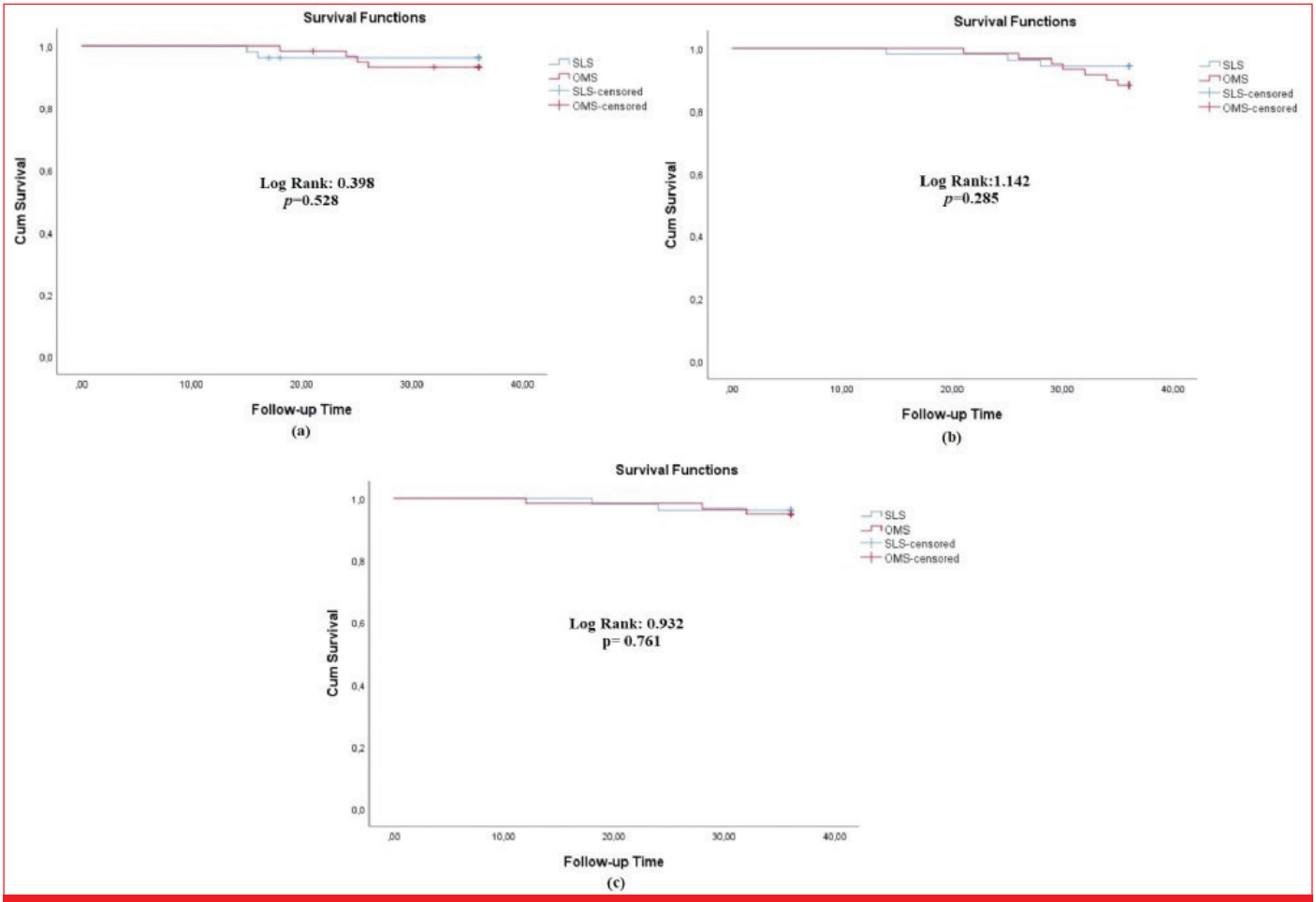

Figure 1. Kaplan-Meier analysis. Mortality (a), stent restenosis (b), stent thrombosis (c).

Table 1. Basic demographic characteristics of patients

	Single long stents (n=52)	Overlapping multiple stents (n=59)	p
Gender (male), n (%)	32 (61.5)	40 (67.8)	0.491
Age, years	61.5 ± 8.2	62.6 ± 8.0	0.483
Body mass index, kg/m^2	26.8 ± 3.8	27.0 ± 3.9	0.857
Hypertension, n (%)	21 (40.4)	21 (35.6)	0.603
Diabetes mellitus, n (%)	14 (26.9)	19 (32.2)	0.544
Dyslipidemia, n (%)	8 (15.4)	12 (20.3)	0.498
Smoking, n (%)	17 (32.7)	22 (37.3)	0.613
Chronic renal failure, n (%)	10 (19.2)	9 (15.3)	0.579
Ejection fraction, %	52.4 ± 8.4	51.3 ± 6.6	0.473
Creatinine, mg/dL	0.94 (0.81-1.19)	0.86 (0.79-1.07)	0.350
Total cholesterol, mg/dL	190.5 ± 56.1	185.9 ± 45.9	0.637
Triglyceride, mg/dL	158 (128-208)	154 (96-215)	0.559
LDL, mg/dL	111.9 ± 39.9	105.3 ± 34.5	0.356
HDL, mg/dL	39.8 ± 10.0	40.7 ± 13.1	0.707
PCI applied artery			
LAD, n (%)	34 (65.4)	39 (66.1)	0.937
CX, n (%)	5 (9.6)	6 (10.2)	0.922
RCA, n (%)	13 (25.0)	14 (23.7)	0.876
Total stent length, mm	42.1 ± 2.6	48.2 ± 2.9	<0.001
Contrast volume, mL	197.1 ± 31.7	256.9 ± 30.5	<0.001
Stent restenosis, n (%)	3 (5.8)	7 (11.9)	0.331
Stent thrombosis, n (%)	2 (3.8)	3 (5.1)	0.754
Mortality, n (%)	2 (3.8)	4 (6.8)	0.683

Interventional Cardiology / Coronary

OP-003

Evaluative value of apelin in STEMI presentations

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Background and Aim: In acute myocardial infarction, hypoxia, through the hypoxia inducible factor pathway, increases the level of apelin, protecting the myocardium from ischemia-reperfusion injury. To evaluate level of apelin-12 in early and late phase of acute myocardial infarction to confirm the positive effects of apelin-APJ axis in patients with myocardial infarction.

Methods: In this study we included ninety eight patients with following criteria: chest pain lasting for more than 30 minutes, a 12-lead electrocardiography characterized with ST-segment elevation (measured at the J-point) in at least two contiguous leads ≥2.5 mm in men <40 years, ≥2 mm in men ≥40 years, or ≥1.5 mm in women in leads V2-V3 and/or ≥1 mm in other leads, rise of cardiac troponin I, and patients who have undergone primary percutaneous coronary intervention (PCI) within period of 12h. Blood samples were collected on the first day (early phase) and on the seventh day (late phase) after reperfusion therapy.

Results: Apelin-12 values in the early and late phase of myocardial infarction were analyzed; the difference was statistically significant (p=0.003). There was variability in apelin values on the first day (Kruskal-Wallis test) relative to segmental wall motion abnormalities (SWMAs) (p=0.043) and on the seventh day relative to different numbers of coronary lesions and stenoses, which was statistically significant (p<0.001). The Mann-Whitney test of the post PCI final thrombolysis in myocardial infarction (TIMI) flow grade (patients without reperfusion injury with thrombolysis in myocardial infarction (TIMI) flow grade 3 and in patients with reperfusion injury with TIMI flow grade ≤2) and the apelin-12 levels during the early phase of myocardial infarction revealed a statistically significant difference (p=0.002).

Conclusions: The increased level of apelin during early phase of myocardial infarction indicates a protective effect from reperfusion injury while low level of apelin during late phase of myocardial infarction is found to be inversely associated with the number of coronary stenoses. Difference in apelin-12 between early and late phase may confirm the effect of apelin-APJ axis in patients with myocardial infarction.

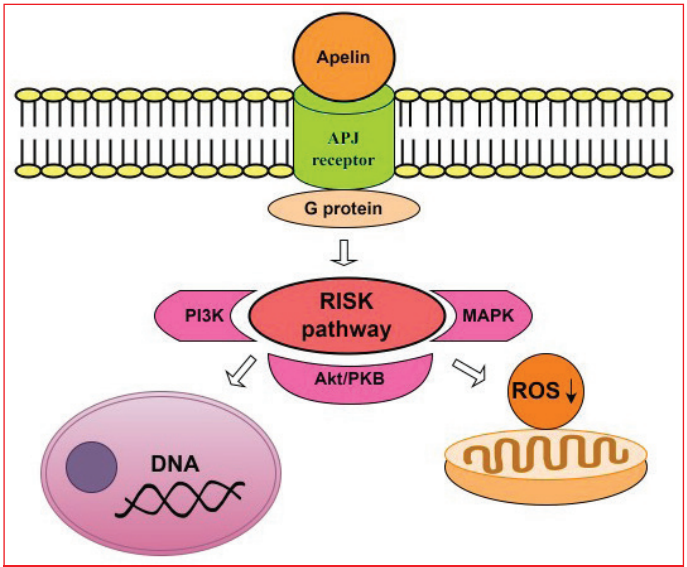

Figure 1. Protective effect of apelin in myocardial ischemia-reperfusion injury.

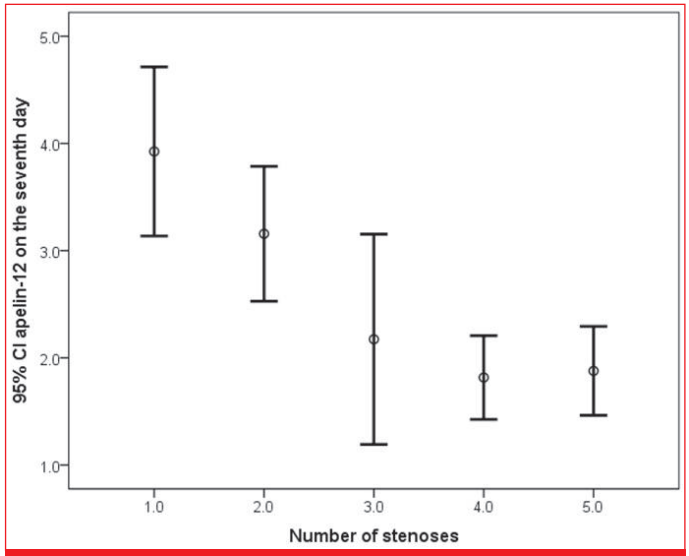

Figure 3. Multiple comparisons graph showing the proportional value of apelin-12 on the 7th day based on the number of stenoses.

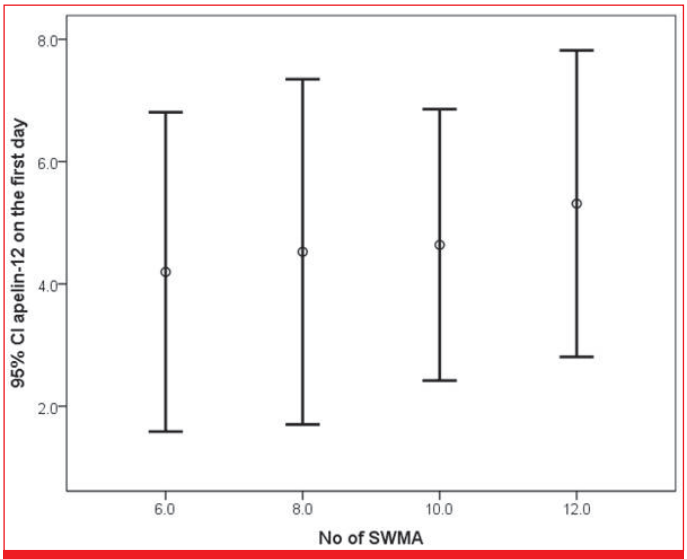

Figure 2. Multiple comparisons graph showing the proportional value of apelin-12 on the 1st day based on the number of SWMAs. SWMA: Segmental wall motion abnormalities.

Table 1. Main characteristics and apelin-12 levels.

	Apelin-12 1st day	p	Apelin-12 7th day	p
No of SWMA median (range)				
5, 6	5.47 (1.80-5.90)	0.043	2.33 (2.30-4.14)	0.43
7, 8	5.60 (0.46-7.08)	0.043	2.97 (1.87-5.28)	0.43
9, 10	2.40 (1.30-13.15)	0.043	2.10 (0.49-6.98)	0.43
11, 12	4.00 (0.0.56-15.25)	0.043	1.95 (0.26-3.53)	0.43
No of stenosis median (range)				
1	3.37 (0.45-11.05)	0.059	3.31 (0.83-10.9)	<0.001
2	4.85 (0.60-13.15)	0.059	2.74 (0.47-6.98)	<0.001
3	1.80 (0.46-12.00)	0.059	1.70 (0.26-9.25)	<0.001
4	1.93 (1.00-6.96)	0.059	2.04 (0.49-2.4)	<0.001
5	2.80 (1.14-8.39)	0.059	1.8 (1.18-2.68)	<0.001
Final TIMI grade flow median (range)				
≤ 2	1.80 (0.46-9.25)	0.002	1.94 (0.26-8.88)	0.01
3	3.93 (0.45-15.25)	0.002	2.42 (0.49-10.90)	0.01
Age median (range)				
≥65	3.15 (0.45-15.25)	0.78	2.20 (1.44-10.90)	0.96
<65	2.92 (0.51-13.50)	0.78	2.36 (0.26-8.88)	0.96
Gender median (range)				
M	2.35 (0.45-13.50)	0.34	2.33 (0.26-10.90)	0.91
F	3.65 (0.50-15.25)	0.34	2.35 (0.55-9.75)	0.91
Hypertension median (range)				
Yes	2.8 (0.45-15.25)	0.55	2.10 (0.26-10.90)	0.058
No	3.17 (0.50-12.00)	0.55	2.55 (0.83-9.75)	0.058
Diabetes mellitus median (range)				
Yes	3.57 (0.56-9.33)	0.94	1.90 (0.55-4.63)	0.029
No	2.80 (0.45-15.25)	0.94	2.40 (0.26-10.90)	0.029
Smoking median (range)				
Yes	3.70 (0.45-13.15)	0.3	2.33 (0.47-10.90)	0.76
No	2.15 (0.46-15.25)	0.3	2.35 (0.26-9.75)	0.76

SWMAs: Segmental wall motion abnormalities, TIMI: Thrombolysis in myocardial infarction.

Interventional Cardiology / Coronary

OP-004

Comparison of glycoprotein IIb/IIIa inhibitor injection route in patients with ST-segment elevation myocardial infarction

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Background and Aim: Glycoprotein (Gp) 2b3a inhibitors are widely available intravenous antiaggregant therapy which are recommended in patients with ST-segment elevation myocardial infarction (STEMI) who have high thrombus burden and/or no-reflow phenomenon during the primary percutaneous coronary intervention. Previous studies have demonstrated that the beneficial effects of Gp2b3a inhibitors have been counterbalanced with excess bleeding. In this study, we aimed to investigate the impact of Gp2b3a inhibitors infused directly to the infarct-related artery on jeopardized myocardium and clinically overt bleeding events.

Methods: We prospectively enrolled patients with STEMI who were treated with primary percutaneous coronary intervention complicated with no-reflow phenomenon and/or presence of high thrombus burden. Weight-adjusted tirofiban was administered according to guideline recommendations. Patients were divided to three groups; intracoronary infusion (group 1), intravenous infusion

(group 2) and control group (group 3) (whom Gp2b3A inhibitors did not used). Intracoronary infusion of Gp2b3A bolus dose was achieved via aspiration catheter placed beyond to the culprit lesion and maintenance dose was infused via peripheral vein. Presence of microvascular obstruction and infarct size were decided according to cardiac magnetic resonance imaging.

Results: We prospectively included 75 patients: 30 patients in group 1, 30 patients in group 2 and 15 patients in group 3. Age and gender were comparable between groups. Comorbidities were similar between groups besides diabetes which was significantly more prevalent in group 1 patients (p=0.006). Thrombus burden was significantly higher in group 1 and 2 compared to group 3 (p<0.001). Angiographic features were similar between groups. There was no significant difference between groups with respect to left ventricular ejection fraction and troponins (p=0.111 and p=0.359). Presence of microvascular obstruction and infarct size were significantly lower in group 1 compared to group 2 and 3 (p=0.048 and p=0.030, respectively). Mean follow-up duration 383 days and new-onset heart failure was significantly lower in group 1 (p=0.035). BARC-3 bleeding event was similar between groups (p=0.841).

Conclusions: Our study has demonstrated that intracoronary infusion of Gp2b3A inhibitors is associated with lower rates of microvascular obstruction, smaller infarct size and lower rates of new-onset heart failure. There was no significant difference with respect to BARC-3 bleeding event between groups. Further studies with higher study population are needed to confirm our results.

Interventional Cardiology / Valve and Structural Heart Disease

OP-005

Comparison of self and balloon-expandable THVs for TAVI in terms of in-hospital and long-term outcomes: Single-center experience

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Background and Aim: Transcatheter aortic valve implantation (TAVI) is an effective interventional therapy that has been used for many years for the treatment of patients with severe aortic stenosis (AS) at high surgical risk. To date, two different types of transcatheter heart valves (THVs) have been used in TAVI: the self-expandable valve (SEV) and the balloon-expandable valve (BEV). Although the efficacy and safety of each of these valves are well established, there are not enough clinical studies directly comparing the different valve types. In this study, we compared in-hospital and 2-year long-term outcomes of patients who underwent TAVI using 4 different THVs in our center.

Methods: The study included 403 patients [mean age 78.2 ± 8.4, female 205 (50.9%)] who underwent TAVI using 4 different brands of THVs, BEV (SAPIEN S3/XT and Myval) and SEV (CoreValve/EvolutR and Portico). Patients were divided into 2 groups, BEV (N=163, 40.4%) and SEV (N=240, 59.6%), and all analyses were performed retrospectively.

Results: Baseline demographic, clinical, echocardiographic characteristics and STS scores did not differ between the two groups (p>0.05 for all). All BEVs and SEVs were implanted from transfemoral access under general anesthesia or conscious sedation with high success rates (99.4% and 97.1%). SEVs were more frequently preferred in valve-in-valve TAVI cases (6.3% vs. 1.2%, p=0.014). In terms of implantation technique, predilatation did not differ between BEV (30.1%) and SEV (30.4%), whereas the need for postdilatation was significantly higher in the SEV (36.7%) group than in the BEV (6.7%) group (p<0.001). In terms of periprocedural adverse events, in-hospital mortality was significantly higher in the BEV (N=21, 12.9%) than in the SEV group (N=15, 6.3%), (p=0.022) whereas the rates of stroke, major and minor bleeding, vascular complications, and permanent pacemaker implantation (PPI) did not differ significantly between the two groups (p>0.05 for all). In the post-procedure follow-up period, both the rates of mortality at follow-up (N=28, 17.2% for BEV vs. N=25, 10.4%, for SEV p=0.049) and the 2-year overall all cause mortality rates were higher in the BEV group (N=49, 30.1% for BEV vs. N=40, 16.7% for SEV, p=0.001). Mean transvalvular gradients measured by echo at follow-up were lower in the SEV group (10.6 ± 5.6 mmHg) than in the BEV group (12.7 ± 8.6 mmHg), (p=0.005) but rates of moderate-to severe paravalvular aortic regurgitation (PVAR) were higher in the SEV group (27.5% vs. 17.8%, p=0.024). No statistically significant differences were observed between the two THV groups with respect to the composite endpoints of in-hospital death, stroke, proton-pump inhibitors, and moderate-to severe PVAR (N=61, 37.4% for BEV, N=82, 34.2%, p=0.502) (Table 1).

Conclusions: Both BEV and SEV THVs are feasable options for the treatment of severe AS with similar high success and low complication rates. However, more randomized and prospective studies are needed to demonstrate the superiority of these THVs over each other.

Table 1.

Variable	BEV (N=163, 40.4%)	SEV (N=240, 59.6%)	Overall Group (N=403)	p value
Baseline Characteristics				
Age	79.1±7.2	77.6±9.1	78.2±8.4	0.200
Gender (female)	75 (46%)	130 (54.2%)	205 (50.9%)	0.108
Hypertension	148 (90.8%)	200 (83.3%)	348 (86.4%)	0.032
Diyabetes	51 (31.3%)	93 (38.8%)	144 (35.7%)	0.125
Atrial Fibrillation	59 (36.2%)	79 (32.9%)	138 (34.2%)	0.496
COPD	63 (38.7%)	92 (38.3%)	155 (38.5%)	0.949
Previous CVA	14 (8.6%)	9 (3.8%)	23 (5.7%)	0.040
CAD	110 (67.5%)	165 (68.8%)	275 (68.2%)	0.789
Previous Cardiac Surgery	31 (19%)	32 (13.3%)	63 (15.6%)	0.123
STS score	9 (6-14)	8 (6-12)	8 (6-13)	0.556
Echocardiographyc Measures				
LVEF (%)	50 (45-60)	55 (44.2-60)	52 (45-60)	0.347
Max Gradient (mmHg)	74.7±21.8	72.5±19.6	73.4±20.5	0.300
Mean Gradient (mmHg)	48.5±14.6	46.3±12.7	47.2±13.5	0.125
Aortic Valve Area (m^2)	0.8 (0.6-0.85)	0.7 (0.6-0.8)	0.73 (0.6-0.8)	0.127
AR ≥2 degree	71 (44.9%)	110 (47.2%)	181 (46.3%)	0.658
MR ≥2 degree	106 (67.1%)	159 (68.2%)	265 (67.8%)	0.811
TR ≥2 degree	128 (80.5%)	184 (79.3%)	312 (79.8%)	0.773
sPAP (mmHg)	43.6±11.2	44.4±11.2	44.1±11.2	0.486
Procedural Features				
VIV TAVI	2 (1.2%)	15 (6.3%)	17 (4.2%)	0.014
THV Size (mm)	26 (26-29)	29 (26-29)	27 (26-29)	<0.001
THV Implantation Success	162 (99.4%)	233 (97.1%)	395 (98%)	0.104
Sheat size (F)	18 (16-20)	14 (14-16)	16 (14-18)	<0.001
Predilatation	49 (30.1%)	73 (30.4%)	122 (30.3%)	0.939
Postdilatation	11 (6.7%)	88 (36.7%)	99 (24.6%)	<0.001
Hospitalisation (days)	4 (3-7)	4 (3-7)	4 (3-7)	0.264
Outcomes				
In-hospital mortality	21 (12.9%)	15 (6.3%)	36 (8.9%)	0.022
Follow-up mortality	28 (17.2%)	25 (10.4%)	53 (13.2%)	0.049
Overall 2-year mortality	49 (30.1%)	40 (16.7%)	89 (22.1%)	0.001
Follow-up mean TVG (mmHg)	12.7±8.6	10.6±5.6	11.5±7.1	0.005
Minor vascular complication	15 (9.2%)	30 (12.5%)	45 (11.2%)	0.302
Major Vascular complication	17 (10.4%)	24 (10%)	41 (10.2%)	0.889
Major bleeding	19 (11.7%)	40 (16.7%)	59 (14.6%)	0.163
Minor bleeding	21 (12.9%)	41 (17.1%)	62 (15.4%)	0.251
Stroke	7 (4.3%)	5 (2.1%)	12 (3%)	0.200
Permanent pacemaker implantation	15 (9.2%)	16 (6.7%)	31 (7.7%)	0.348
Moderate-to severe PVAR	29 (17.8%)	66 (27.5%)	95 (23.6%)	0.024
Composite end-point	61 (37.4%)	82 (34.2%)	143 (35.5%)	0.502

BEV: Balloon-expandable valve, SEV: Self-expandable valve, COPD: Chronic obstructive pulmonary disease, CVA: Cerebrovascular attack, CAD: Coronary artery disease, STS: Society of Thoracic Surgeons, LVEF: Left ventricle ejection fraction, AR: Aortic regurgitation, MR: Mitral regurgitation, TR: Tricuspid regurgitation, sPAP: Systolic pulmonary artery pressure, VIV TAVI: Vale-in-valve transcatheter aortic valve implantation, THV: Transcatheter heart valve, F: French, TVG: Transvalvular gradient, PVAR: Paravalvular aortic regurgitation.

Interventional Cardiology / Coronary

OP-006

5-year clinicAL outcomes and Procedural characteristics of isolated non-left main Side Branch Osteal stenosis (The ALP-SBO registry)

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Background and Aim: Side branch (SB) osteal stenosis is a frequent bifurcation lesion and its optimal treatment strategy is still debated. Although the efficacy of the optimal medical therapy is well known, the comparison of the medical treatment and percutaneous coronary intervention (PCI) is controversial.

Methods: A total of 357 patients with isolated SB osteal stenosis (Medina 0,0,1 classification) were retrospectively evaluated. Demographic, clinical, and angiographic parameters were recorded from the hospital database. Patients were divided into two groups; patients with only medical therapy (n=305) and patients undergoing PCI (n=52). Target vessel revascularization (TVR), myocardial infarction (MI), and mortality were evaluated as major adverse cardiovascular outcomes (MACE).

Results: The mean follow-up time was 59 months while the mean age was 58.2 ± 10.3 years. 102 patients (28.6%) were female. 279 patients (78.2%) had diagonal lesion while 78 (21.8%) had obtuse marginal lesion. 172 patients (48.2%) had acute coronary syndrome. The SB stenosis ratio (70 ± 16; 88 ± 13, p<0.001) and SB lesion length (7.5 ± 6.6; 14.7 ± 6.0, p<0.001) were higher in the PCI group. SB osteal stenting [38 patients (73.1%)] was the common PCI technique. There were no significant differences in terms of TVR [1.3%; 1.9%, p=0.277, medical vs. PCI groups respectively], MI [8.2%; 7.7%, p=0.302, medical vs. PCI groups respectively], mortality [10.2%; 9.6%, p=0.095, medical vs. PCI groups respectively] and MACE [18.0%; 15.4%, p=0.113, medical vs. PCI groups respectively] between groups. Additionally, in Kaplan-Meier survival analysis, there were no differences in TVR (Log-rank p=0.247), MI (Log-rank p=0.295), mortality (Log-rank p=0.086), and MACE (Log-rank p=0.107) between groups.

Conclusions: Medical therapy instead of PCI seems to be an appropriate and optimal treatment strategy in patients with non-left main SB osteal stenosis.

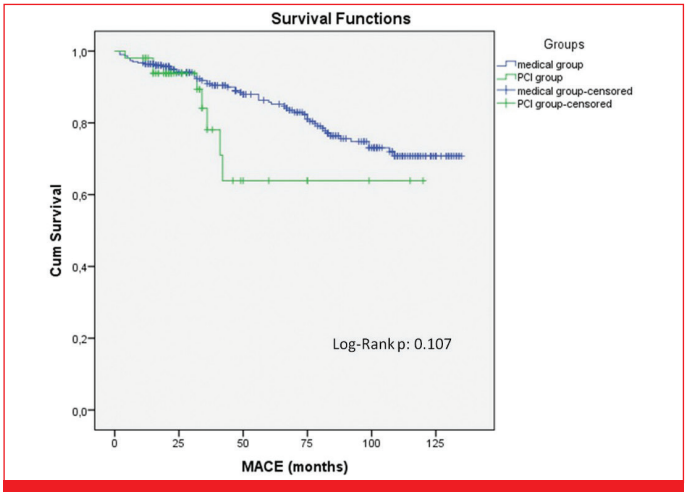

Figure 1. Kaplan-Meier survival curve for MACE. MACE: Major adverse cardiovascular outcome.

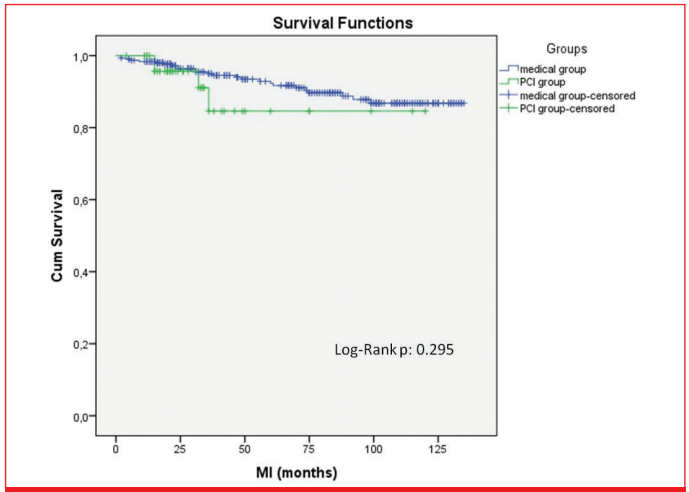

Figure 2. Kaplan-Meier survival curve for MI. MI: Myocardial infarction.

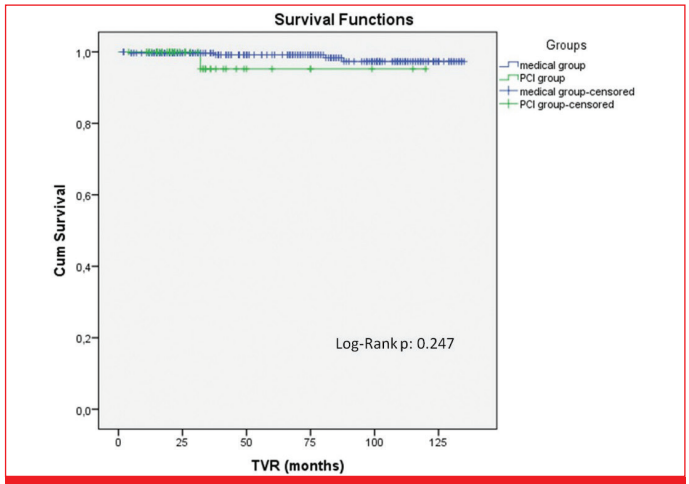

Figure 3. Kaplan-Meier survival curve for TVR. TVR: Target vessel revascularization.

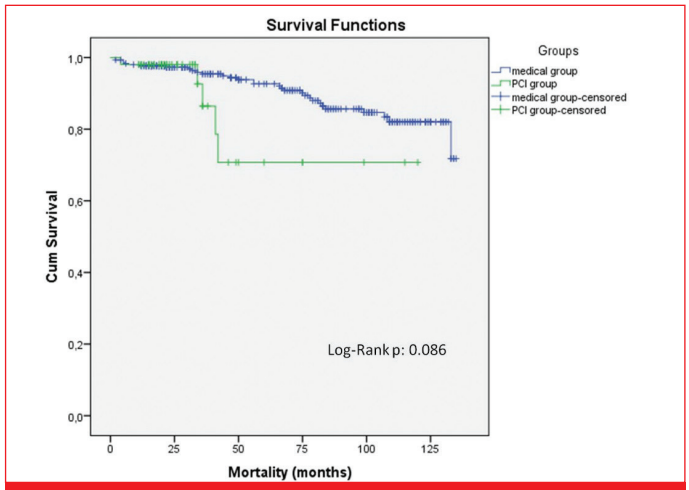

Figure 4. Kaplan-Meier survival curve for mortality.

Table 1. Baseline clinical and demographic parameters of the study population

	Medical group (n=305)	PCI group (n=52)	p
Age, years	58.4 ± 10.4	57.1 ± 9.7	0.386
Gender (female), n (%)	95 (31.1)	7 (13.5)	0.009
Diabetes mellitus, n (%)	85 (27.9)	18 (34.6)	0.321
Hypertension, n (%)	124 (40.7)	18 (34.6)	0.411
Chronic obstructive lung disease, n (%)	31 (10.2)	4 (7.7)	0.580
Previous CVA, n (%)	6 (2.0)	2 (3.8)	0.329
PAD, n (%)	8 (2.6)	0 (0)	0.280
Smoking, n (%)	51 (16.7)	16 (30.8)	0.016
Previous PCI, n (%)	30 (9.8)	10 (19.2)	0.047
Previous MI, n (%)	21 (6.9)	8 (15.4)	0.043
Atrial fibrillation, n (%)	14 (4.6)	1 (1.9)	0.330
Ejection fraction, %	60 (50-60)	60 (50-60)	0.063

CVA: Cerebrovascular accident, MI: Myocardial infarction, PAD: Peripheral arterial disease, PCI: Percutaneous coronary intervention.

Table 2. Angiographic evaluation of the patients

	Medical group (n=305)	PCI group (n=52)	p
Group, n (%)			
Diagonal	250 (82.0)	29 (55.8)	<0.001
OM	55 (18.0)	23 (44.2)	
Clinical presentation, n (%)			
Stable	133 (43.6)	10 (19.2)[a]	<0.001
USAP	17 (5.6)	4 (7.7)	
NONSTEMI	85 (27.9)	27 (51.9)[b]	
STEMI	30 (9.8)	9 (17.3)	
Silent ischemia	40 (13.1)	2 (3.8)	
SB stenosis ratio, %	70 ± 16	88 ± 13	<0.001
MV proximal diameter, mm	3.18 ± 0.59	3.32 ± 0.56	0.128
MV distal diameter, mm	2.90 ± 1.52	2.71 ± 0.45	0.370
SB diameter, mm	2.33 ± 0.29	2.52 ± 0.33	<0.001
SB lesion length, mm	7.5 ± 6.6	14.7 ± 6.0	<0.001
Bifurcation angle, °	58.4 ± 11.5	61.3 ± 11.4	0.257
SB osteal thrombosis, n (%)	4 (1.3)	1 (1.9)	0.547
Multivessel disease, n (%)			
Only SB	188 (61.6)	28 (53.8)	
1-vessel	109 (35.7)	20 (38.5)	0.215
2-vessel	7 (2.3)	3 (5.8)	
3-vessel	1 (0.3)	1 (1.9)	
Syntax score	6.9 ± 3.9	7.7 ± 4.0	0.201

a: Significantly lower compared to medical group, b: Significantly higher compared to medical group. MV: Main vessel, NONSTEMI: Non-ST-segment elevation myocardial infarction, OM: Obtuse marginal, PCI: Percutaneous coronary intervention, SB: Side branch, STEMI: ST-segment elevation myocardial infarction, USAP: Unstable angina pectoris.

Table 3. Clinical outcomes of the patients

	Medical group (n=305)	PCI group (n=52)	Hazard Ratio (95% CI)	p
Primary composite endpoint (MACE), n (%)	55 (18.0)	8 (15.4)	1.852 (0.865-3.964)	0.113
TVR, n (%)	4 (1.3)	1 (1.9)	3.544 (0.363-34.630)	0.277
MI, n (%)	25 (8.2)	4 (7.7)	1.765 (0.600-5.186)	0.302
Mortality, n (%)*	31 (10.2)	5 (9.6)	2.298 (0.864-6.110)	0.095
Secondary endpoint, n (%) Stroke	4 (1.3)	0 (0)	0.042 (0.0-540)	0.740

* all-cause death and mortality. MACE: Major adverse cardiovascular outcome, MI: Myocardial infarction, PCI: Percutaneous coronary intervention, TVR: Target vessel revascularization.

Interventional Cardiology / Valve and Structural Heart Disease

OP-007

Predictors of pacemaker implantation following TAVR

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Background and Aim: Transcatheter aortic valve replacement (TAVR) is a minimally invasive procedure employed for the treatment of aortic valve disease in patients who are considered high-risk or ineligible for traditional open-heart surgery. It entails the percutaneous insertion of a bioprosthetic valve through a catheter, typically via the femoral artery or another access point. During the TAVR procedure, various factors have the potential to affect the patient's electrical conduction system and disrupt the heart's inherent rhythm. As the frequency of procedures increases, the need for complete atrioventricular (AV) block and permanent pacemaker also increases. These factors encompass the positioning of the transcatheter valve, the proximity of the valve to the electrical pathways, and the manipulation of the catheter within the cardiac structure. Consequently, the likelihood of conduction abnormalities, such as heart block, may increased during or following the TAVR procedure. Anticipating the necessity for pacemaker implantation before the TAVR procedure may enable the medical team to take precautionary measures to minimise the risk of conduction abnormalities. The present study aimed to evaluate the relationship between the development of av complete block after TAVR and possible predictive parameters.

Methods: The study population consisted of 191 consecutive patients undergoing TAVR for severe aortic valve stenosis between January 2021 and June 2022. Baseline clinical characteristics and clinical information were recorded. The patients were divided into two groups according to the development of complete AV block. Multivariate logistic regression analysis was performed to identify predictors of complete AV block.

Results: Among the participants, 13 (6.8%) developed complete AV block. In the group with AV complete block, the pre-procedural QRS complex was found to be wider (96.4 ± 21.1 vs. 129.9 ± 26.2 ms; p<0.001). Bradycardia and bundle branch block (BBB) on the pre-procedural electrocardiogram were significantly more common in the complete AV block group (p=0.001). The aortic valve has a significantly smaller area in the complete AV block group (p=0.033). In addition, the prosthetic valve/aortic annulus ratio is higher (p=0.015). In multivariate logistic regression analysis, preoperative QRS complex was found to be an independent predictor of complete AV block. Preoperative bradycardia, aortic valve area and prosthetic valve/aortic annulus ratio were other independent predictors.

Conclusions: In summary, advancements in TAVR technology have facilitated the development of valve designs specifically tailored to minimize interference with the electrical conduction system. These designs aim to reduce the requirement for pacemaker implantation after the procedure and enhance patient outcomes. Therefore, it is crucial to investigate the predictive factors that determine the necessity of pacemaker implantation in patients undergoing TAVR.

Table 2. Multivariate logistic regression analysis

Factor	Multivariable odds ratio (95% CI)	p
Bradycardia	6.22 (3.234-8.565)	0.010
QRS complex	1.110 (1.032-1.211)	0.005
Aortic valve area	1.015 (1.001-3.014)	0.050
Prosthetic valve/aortic annulus ratio	1.288 (1.024-1.456)	0.030

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

Variables	Complete AV block (-) (n=178)	Complete AV block (+) (n=13)	p
Age	76.8 ± 7.9	75.8 ± 8.1	0.643
Diabetes mellitus	76 (43%)	7 (53%)	0.568
Hypertension	146 (82%)	11 (85%)	0.653
Coronary heart disease	112 (63%)	6 (46%)	0.282
Chronic kidney disease	53 (30%)	6 (46%)	0.045
Chronic obstructive pulmonary disease	49 (28%)	2 (15%)	0.269
Peripheral artery disease	10 (6%)	1 (8%)	0.558
Atrial fibrillation	36 (20%)	6 (46%)	0.035
STS score	4.6 ± 2.9	4.2 ± 2.4	0.823
Body mass index	29.2 ± 6.2	27.9 ± 3.2	0.451
Bradycardia	10 (6%)	5 (38%)	0.001
Bundle branch block	89 (50%)	12 (92%)	0.001
PR interval, ms	163.5 ± 30.7	167.2 ± 29.4	0.756
QRS complex, ms	96.3 ± 21.1	129.9 ± 26.2	<0.001
Mitral annular calcification	114 (64%)	9 (69%)	0.001
Aortic valve area, cm^2	0.7 ± 0.1	0.6 ± 0.1	0.033
Aortic mean gradient, mmHg	48.4 ± 11.1	50.8 ± 15.6	0.474
Aortic valve calcium score	2857.2 ± 3264.0	1574.6 ± 1965.1	0.398
Prosthetic valve/aortic annulus ratio	2.6 ± 5.1	6.6 ± 10.3	0.015
Left ventricular ejection fraction, %	51.5 ± 12.8	49.4± 8.7	0.440

AT: Atrioventricular.

Other

OP-008

The effects of immune check point inhibitors on cardiac systolic, diastolic and atrial electromechanics functions

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Background and Aim: Immune checkpoint inhibitors (ICI) are one of the most remarkable agents used as a monotherapy or combination therapy in recent years. We aimed to investigate potential cardiac toxic effects, systolic, diastolic, and atrial functions of patients who were treated with ICI for different cancer types.

Methods: Thirty-three patients who received immunotherapy as an anti-cancer therapy included the study. Echocardiographic and electrocardiographic assessment were performed at first visit before ICI administration and after three months the end of therapy.

Results: N-terminal brain natriuretic peptide (NT-pro-BNP) levels were statistically higher during the post-treatment period (273.12 ± 224.60 pg/mL, 812.32 ± 1676.42 pg/mL; p=0.034; pre- and post-treatment periods). Left ventricular stroke (LVSV) volume and cardiac output (CO) of the patients were statistically lower in the post-treatment control (61.19 ± 13.72 mL and 57.80 ± 10.15 mL; p=0.049; 5.00 ± 1.14 L/min and 4.69 ± 0.85 L/min, p=0.048; LVSV and CO, pre-post-treatment values). Left atrial ejection fraction was detected higher in the post-treatment period (68.26 ± 13.75, 70.17 ± 11.98; p=0.036; pre- and post-treatment values). The QTc interval was prolonged in the post-operative period (433.57 ± 26.19 msec and 451.86 ± 39.41 msec; p=0.029; pre- and post-treatment). Total atrial conduction time was shorter in the post-treatment period (106.87 ± 17.76 msec and 101.19 ± 14.74 msec; p=0.009; pre- and post-treatment).

Conclusions: This study was performed using clinical, electrocardiographic, and echocardiographic follow-up data from patients who were treated with ICI. The most relevant findings are described below: No deaths from cardiac toxicity occurred during the follow-up period. Anterior myocardial infarction occurred in only one patient. The troponin values of the patients did not increase significantly in contrast to the NT-pro-BNP values. While cardiac output and LV stroke volume decreased, no significant changes were observed in diastolic parameters and LVEF. A reduction in the right atrium LA maximum volume was observed, whereas LAVI did not change. Total atrial conduction time was decreased, and the QTc interval was prolonged. In conclusion, our study showed that cardiac systolic and diastolic function did not change, QTc interval was prolonged, and total atrial conduction time decreased in short-term follow-up post-ICI treatment. One patient encountered acute anterior wall myocardial infarction in the study population. Long-term follow-up studies with larger numbers of patients are needed to clarify the clinical significance of these parameters in patients receiving ICI treatment.

Table 1. Demographic data of patients

	Patients (n=33)
Gender, male	27, (81.82%)
Age, years	62.76 ± 9.74
DM	8, (24.24%)
HT	6, (18.18%)
CAD	5, (15.15%)
History of CABG	2, (6,06%%)
Smoking	24, (72.73%)
Alcohol consuming	10, (30.30%)
Cerebral metastases	9, (27.27%)
Palliative radiotherapy	12, (36.36%)
Mediastinal radiotherapy	8, (24.24%)
Immunotherapy for adjuvant therapy	3, (9.09%)
Immunotherapy for metastatic disease	30, (90,91%)
Drug of choice	
Nivolumab	26 (78,79%)
Atezolizumab	5 (15,15%)
Pembrolizumab	2 (6,06%)
Stage of disease	
Stage 3	3, (9,09%)
Stage 4	30, (90,91%)
Prior treatment	
Absent	4, (12,12%)
Chemotherapy	13, (39,39%)
Chemotherapy + Radiotherapy	6, (15,15%)
Tyrosine kinase inhibitor	8, (24,24%)

Table 2. Laboratory findings, weight and BSA of patients

Laboratory Findings	Pre-Treatment	Post-Treatment	p value
Weight, kg	70.10 ± 12.66	69.68 ± 13.25	0.313
BSA, kg/m2	1.81 ± 0.18	1.80 ± 0.19	0.23
Troponin (ng/L) *	47.63 ± 109.19	70.42 ± 172.33	0.181
Creatinine (mg/dl) *	1.04 ± 0.55	1.07 ± 0.87	0.518
ALT (IU/L) *	31.13 ± 66.19	19.87 ± 17.65	0.782
TSH (mU/L) *	2.11 ± 2.35	14.79 ± 37.33	0.227
CRP (mg/L) *	33.39 ± 35.29	47.32 ± 66.02	0.316
NT-pro BNP (ng/L) *	273.12 ± 224.60	812.32 ± 1676.42	**0.034****

Abbreviations: ALT; alanine amino transferase, BSA; body surface area, TSH; thyroid stimulant hormone, CRP; c reactive protein, NT-pro BNP; n terminal brain natriuretic peptide
*p value was calculated with Wilcoxon signed-rank test due to abnormal distribution of data **p<0.05

Table 3. Echocardiographic data of the patients

	Pre-Treatment	Post-Treatment	p value
Diastolic diameter of LV, mm	46.62 ± 4.00	46.84 ± 4.17	0.555
Systolic diameter of LV, mm	30.16 ± 3.17	30.25 ± 3.03	0.790
RWT	0.41 ± 0.06	0.41 ± 0.04	0.204
LV Mass, gr	159.81 ± 34.40	158.66 ± 41.97	0.739
LV stroke volume, mL	61.19 ± 13.72	57.80 ± 10.15	**0.049****
Cardiac output, L/min	5.00 ± 1.14	4.69 ± 0.85	**0.048****
Cardiac index, L/min/m2	2.76 ± 0.66	2.61 ± 0.50	0.085
LV end-systolic volume, mL	22.69 ± 7.86	22.41 ± 7.25	0.783
LV septum thickness, mm *	10.06 ± 1.19	9.84 ± 1.42	0.124
LV posterior wall thickness, mm *	9.59 ± 1.10	9.50 ± 0.95	0.405
LVEF *	61.40 ± 2.51	61.07 ± 2.98	0.336
MPI *	0.58 ± 0.26	0.59 ± 0.18	0.813
DT, msec*	139.72 ± 46.48	135.29 ± 39.09	0.240
TAS, cm/s*	13.32 ± 2.52	12.84 ± 1.97	0.202
TAPSE, mm	19.39 ± 2.72	19.55 ± 2.78	0.620
e/e' ratio	8.65 ± 3.15	9.24 ± 3.18	0.111

Abbreviations: LV; Left ventricle, RWT; Relative wall thickness, LVEF; Left ventricle ejection fraction, MPI; myocardial performance index, DT; Deceleration time, TAS Myocardial systolic velocity, TAPSE; Tricuspid annular plane systolic excursion *p value was calculated with Wilcoxon signed-rank test due to abnormal distribution of data**p<0.05

Table 4. Atrial measurements of the patients

	Pre-Treatment	Post-Treatment	p value
LA vertical diameter, mm	49.06 ± 7.08	48.78 ± 6.40	0.707
LA horizontal diameter, mm*	37.31 ± 3.14	37.06 ± 3.85	0.363
LA maximum volume, mL *	42.80 ± 14.80	39.48 ± 13.30	**0.04****
LA pre-contraction volume, mL	24.22 ± 9.65	22.20 ± 8.14	0.122
LA minimum volume, mL	13.52 ± 7.17	11.73 ± 6.21	0.065
LA total stroke volume, mL*	29.27 ± 12.40	27.75 ± 11.41	0.953
LA active stroke volume*	10.70 ± 6.56	10.47 ± 5.59	0.456
LA passive stroke volume*	18.58 ± 12.41	17.28 ± 10.24	0.984
LAEF*	68.26 ± 13.75	70.17 ± 11.98	**0.036****
LAVI*	23.66 ± 7.67	21.92 ± 6.40	0.082
RA vertical diameter, mm*	28.61 ± 4.95	27.70 ± 3.96	**0.043****
RA horizontal diameter, mm*	41.22 ± 5.07	40.48 ± 4.96	**0.049****

Abbreviations: LA; Left atrium, LAEF; Left atrium ejection fraction, LAVI; Left atrium volume index, RA; Right atrium. * p value was calculated with Wilcoxon signed-rank test due to abnormal distribution of data. ** p<0.05

Table 5. Electrocardiographic measurements and electromechanics delays of the patients

Measurements	Pre-Treatment	Post-Treatment	p value
PR interval, msec	148.50 ± 20.29	139.50 ± 19.32	0.053
QRS duration, msec	85.69 ± 15.38	88.46 ± 17.25	0.16
QTC interval, msec	433.57 ± 26.19	451.86 ± 39.41	**0.029****
Heart rate, bpm	79.64 ± 14.01	87.43 ± 19.28	0.104
Total atrial conduction time, msec*	106.87 ± 17.76	101.19 ± 14.74	**0.009****
PA septal, msec*	41.65 ± 12.25	40.84 ± 12.64	0.672
PA lateral, msec*	55.23 ± 16.70	53.19 ± 14.93	0.404
PA tricuspid, msec*	28.03 ± 11.84	26.26 ± 9.44	0.351
Intra-atrial electromechanical delay, msec *	13.61 ± 8.89	14.58 ± 8.83	0.11
Inter-atrial electromechanical delay, msec	27.19 ± 13.38	26.94 ± 12.40	0.841

Abbreviations: PA; the time interval from the onset of the P wave on surface electrocardiogram to the beginning of A wave obtained by tissue Doppler imaging *p value was calculated with Wilcoxon signed-rank test due to abnormal distribution of data. **p<0.05

Epidemiology

OP-009

Assessment of aortic indexes among diabetics and non-diabetic metabolic syndrome patients

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Background and Aim: Aging process in vascular beds is associated with an increase in vascular stiffness which is augmented by atherosclerosis, hypertension and diabetes mellitus (DM). It has been revealed in many studies that increased aortic stiffness (AS) is a risk factor for cardiovascular disease (CVD). As well as AS, another parameters of assessment of aortic elasticity are aortic distensibility and aortic strain which both have been reported as useful predictors of CVD. Alterations of elasticity of aorta has been shown in metabolic syndrome (Met-S) before however comparisons of these parameters between diabetic and non-diabetis Met-S patients never evaluted before so we aimed to assessment of aortic elasticity between diabetic and non-diabetic patients.

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 International Diabetes Federation diagnostic criteria. Patients with history of coronary heart disease, aortic disease, valvular heart disease, treated with hypertension, hyperlipidemia and diabetes and chronic renal disease were excluded from the study. Biochemical analysis were included many laboratory parameters. However we compared only fasting glucose, uric acid, and creatinine. Aortic stiffness, aortic distensibility and and strain were assessed with echocardiographic analysis with a same experienced cardiologist who was blind to study. Echocardiographic assessment of parameters of aortic elasticity were analysed according to previous studies.

Results: Patients were divided into 3 group as group 0: normal group, group 1: non-diabetic group and group 2: diabetic group. Baseline features and parameters of aortic elasticity were shown in Table 1. Fasting glucose, creatinine and uric acid were found to be significantly different between groups. Among the parameters of aortic elasticity only aortic distensibility and strain were found to be different between groups. Post-hoc analysis of variables were shown in Table 2.

Conclusions: Alterations in elasticity of aorta which has been shown to be an independent predictor of coronary heart disease, stroke and mortality in general population and Met-S. In a study, patients with DM also exhibited significantly greater aortic stiffness expressed by higher AS value than in the control group. In a previous study aortic stiffness was found to be significantly higher in the Met-S group as well as chronic renal disease, DM, congenitel heart disease. Aortic indexes of elasticity were never evaluated between diabetic and non-diabetic with Met-S. In our comparison only aortic distensibility and strain were found to be significant differences between groups. Their importance for chronich inflammatory systemic diseases like Met-S are needed to be investigated in more studies.

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 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						0.827$^{\psi}$
Woman	(count$^{\&}$ percent in total)	25 (16.3%)	22 (14.3%)	26 (16.9%)	73 (47.8%)	
Male	(count$^{\&}$ percent in total)	28 (18.3%)	27 (17.6%)	25 (16.3%)	80 (52.2%)	
Age	Mean ± SD 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 ± SD 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	<0.0001$^{\beta}$
Creatinine, mg/dL	Mean ± SD Median (25%-75%)	0.7 ± 0.1 0.7 (0.6-0.9)	0.8 ± 0.1 0.8 (0.7-1.0)	0.8 ± 0.2 0.8 (0.7-0.9)	0.8 ± 0.2	0.013$^{\&}$
Aortic stiffness, n	Mean ± SD Median (25%-75%)	0.030 ± 0.025 0.023 (0.14-0.38)	0.019 ± 0.016 0.138 (0.009-0.25)	0.027 ± 0.026 0.196 (0.004-0.40)	0.026 ± 0.02	0.946$^{\beta}$
Aortic distensibility ($kPa^{-1}\cdot10^{-3}$)	Mean ± std Median (25%-75%)	2.073 ± 1.44 1.370 (0.98-3.67)	1.517 ± 0.88 1.340 (0.74-2.17)	1.898 ± 1.76 1.167 (0.73-2.55)	1.822 ± 1.53	0.043$^{\beta}$
Aortic strain, %	Mean ± SD Median (25%-75%)	6.201 ± 5.60 5.275 (1.74-8.96)	4.93 ± 3.72 3.342 (2.27-7.87)	5.578 ± 6.02 3.439 (2.07-7.42)	5.720 ± 5.35	0.032$^{\beta}$
Uric acid, mg/dL	Mean ± SD Median (25%-75%)	4.8 ± 0.8 4.8 (3.9-5.3)	5.3 ± 1.4 5.3 (4.4-6.2)	4.9 ± 1.1 4.8 (4.3-5.5)	5.12 ± 1.3	0.004$^{\&}$

&: One-Way ANOVA, β: Independent samples non-parametric Kruskal-Wallis test, ψ: Chi-square test. DM: Diabetes mellitus, SD: Standard deviation.

Table 2. Post-hoc analysis

Variables	Group 0 vs. 1	Group 0 vs. 2	Group 1 vs. 2
Uric acid	NS$^{\alpha}$	0.025$^{\alpha}$	NS$^{\alpha}$
Creatinine	NS$^{\alpha}$	NS$^{\alpha}$	NS$^{\alpha}$
Aortic distensibility	NS$^{\mu}$	0.031$^{\mu}$	<0.001$^{\mu}$
Aortic strain	NS$^{\mu}$	0.029$^{\mu}$	NS$^{\mu}$
Glucose	NS$^{\mu}$	<0.001$^{\mu}$	<0.001$^{\mu}$

α: Tukey HSD post- hoc analysis, μ: Mann-Whitney U test, NS: Non-significant

Epidemiology

OP-010

Calculation of the effect of diabetes on the age at first ACS, taking into account the interaction between diabetes and smoking

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Background and Aim: The study evaluates the effects of tip 2 diabetes mellitus (DM) on the age of the first acute coronary syndromes (ACS) age.

Methods: This prospective observational study enrolled consecutive adult patients (>18 years) who were diagnosed with ACS for the first time and admitted to coronay care unit between 2014 and 2022. The exclusion criteria were determined on three main bases. The first is receiving certain medications and situations that may have confounded the age at first ACS (regular use of antiplatelets, or anticoagulants; cardiovascular diseases; chronic renal disease; chronic obstructive pulmonary disease; malignancy). Secondly, the cases, which the diagnosis of ACS cannot be fully ascertained (situations where oral communication with the patient is impossible and where coronary angiography could not be performed) were excluded. Third, there is suspicion whether the ACS is the first or repetitive ACS (Patients with findings that might be related to previous myocardial infarction on electrocardiography or echocardiography, total occlusion other than "culprit" lesion and no critical stenosis on coronary angiography were also excluded). During the hospitalization period, face-to-face interviews and physical examinations were performed, and laboratory findings and cardiovascular risk factors were determined. Independent variables affecting the age of first ACS were examined by linear regression analysis. In the regression model, the interaction factors of smoking-gender and smoking-diabetes were taken into account.

Results: A total of 1823 patients (mean age, 57.31 ± 11.7 years; 18.9% female) were included the study. 33.4 percent of the patients (n=608) were diabetic. The mean time between the diagnosis of diabetes and the first ACS was 7.7 ± 7.7 years (median 5 years Q1-Q3: 2-10 years). The linear regression analysis showed that DM is an independent risk factor, reducing the age at first ACS. In the regression model, gender, smoking, body mass index, family history, non-HDL were also included as independent variables (p<0.001). DM reduced the age of first ACS episode by 2.2 years (p=0.003). This effect was not observed in subjects for smoking. There was no difference in age at first ACS between smoking-diabetic and non-diabetic patients. In the calculations made with the assumption that there are no risk factors other than smoking and diabetes, it was seen that the diabetes risk factor in smokers could not make an angulation that could cause the ACS threshold to earlier age (Figure 1).

Conclusions: Patients with DM experience first ACS episode 2.2 years earlier than those with non-diabetic patients. Due to the interaction between smoking and diabetes, the effect of diabetes on age can only be calculated in the non-smoking patient population.

Table 1. Baseline characteristics of the study population

	Diabetic (n=608)	Non-diabetic (n=1215)	p
Female, n (%)	149 (24.5)	196 (16.1)	<0.001
HTN, n (%)	310 (51.0)	411 (33.9)	<0.001
Family history of CAD	567 (46.7)	238 (39.1)	0.003
Current smoker	296 (48.7)	742 (61.1)	0.381
Total cholesterol, mg/dL, mean SD Mean (Q1-Q3)	201.9 ± 50.0	199.8 ± 48.9	0.381
HDL, mg/dL, mean SD Mean (Q1-Q3)	41.1 ± 9.5 40 (35-46)	42.1 ± 10.2 41 (35-48)	0.068
LDL-C, mg/dL, mean SD Mean (Q1-Q3)	128.0 ± 41.0 124 (99-152)	129.8 ± 39.3 128 (104-151)	0.381
Non-HDL, mg/dL, mean SD Mean (Q1-Q3)	158.9 ± 46.7 153 (128-187)	158.9 ± 74.7 155 (126-183)	0.984
Trigliseride, mg/dL, mean SD Mean (Q1-Q3)	176.1 ± 133.6 140 (98-202)	149.0 ± 116.4 120 (84-178)	<0.001
Body mass index	28.9 ± 4.5	27.4 ± 4.1	<0.001
Glucose, mg/dL, mean SD Mean (Q1-Q3)	207.5 ± 77.3 155 (147-173)	108.8 ± 8.2 118 (114-123)	<0.001

CAD: Coronary artery disease, HDL: High density lipoprotein, HTN: Hypertension, LDL: Low density lipoprotein.

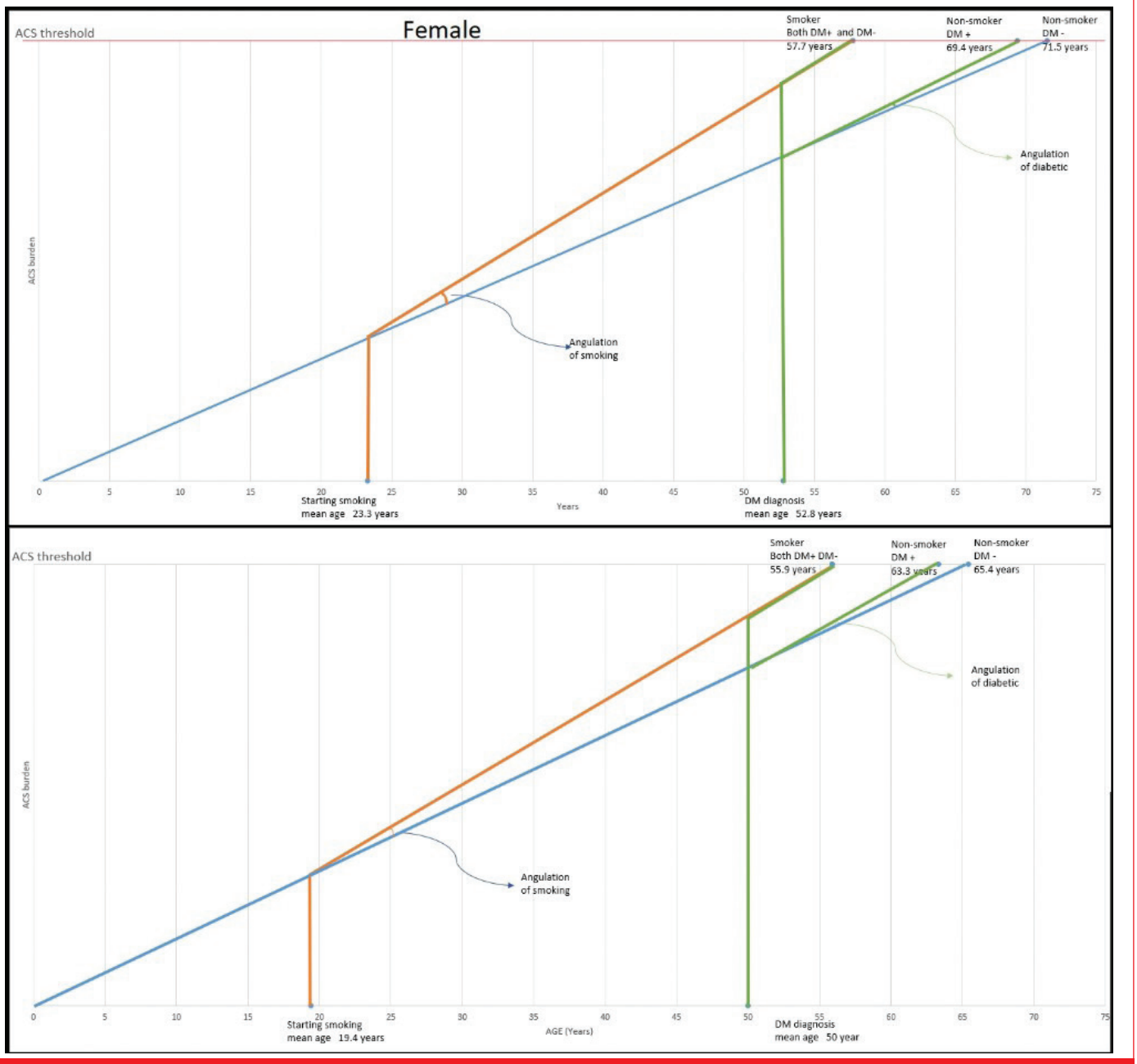

Figure 1. Age to reach hypothetical ACS threshold.
DM: Diabetes mellitus

Other

OP-011

Utilization of artificial intelligence tools in the diagnosis of heart failure with preserved ejection fraction (HFpEF) and determination of the weight of specific clinical parameters in diagnosis

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Background and Aim: Heart Failure with Preserved Ejection Fraction (HFpEF) accounts for nearly half of the entire heart failure patient population. Even for cardiologists, diagnosing HFpEF can be challenging when compared to heart failure with reduced ejection fraction (HFrEF). The lack of consensus on HFpEF diagnostic criteria further complicates this situation. In a retrospective study titled "Artificial Intelligence for the Diagnosis of Heart Failure" published in the journal Digital Medicine in 2020, the use of machine learning achieved diagnostic accuracies of 100% for HFrEF and

HFmrEF, whereas this rate remained at 78.9% for HFpEF. The challenging nature of HFpEF diagnosis and the promising nature of existing studies on AI-assisted diagnosis underscore the necessity of our conducted study.

Methods: Since our study involves making predictions based on labeled data, a supervised learning algorithm has been utilized. This algorithm creates a model using training data composed of input features and target outputs. It learns from examples in the training dataset to make predictions for new inputs. For our study, a dataset was compiled for use in the artificial intelligence program, consisting of 301 patients who visited our center between 2018 and 2022. These patients were randomly selected based on specific criteria. The group is evenly divided between HFpEF and non-HFpEF patients. A total of 56 different parameters were defined, including demographic information, comorbidities, laboratory results, and echocardiography findings. The generated dataset was introduced into MATLAB. The Classification Learner app in MATLAB was employed to determine the classification method with the highest accuracy. The dataset was split into training (n=240) and testing (n=61) sets in an 80%-20% ratio.

Results: During the training using MATLAB-Classification Learner, the system achieved the highest accuracy of 98.3%. In the testing phase, the accuracy rate varied between 96.7% and 98.3% depending on the usage of different parameters. Using the ANOVA, Chi-square, and Kruskal-Wallis statistical methods available within the software package, the diagnostic weight assigned by artificial intelligence to different parameters was measured. For the top 6 parameters, diagnostic weight ratios were found as follows using ANOVA: Pwd: 76.95, Age: 66.71, IVSd: 59.69, EGFR: 38.74, AF: 38.47, NT-pro-BNP: 36.46, LVMASS: 35.62. Considering the experiments conducted within the study and taking literature into account, increasing both the quantity and quality of data leads to improved accuracy.

Conclusions: This study aimed to confirm the hypothesis that HFpEF diagnosis can be achieved with high accuracy using an AI tool, and the findings are consistent with the literature. Few studies in the literature focus solely on HFpEF diagnosis and use real-world data. Therefore, there's a need for more comprehensive studies involving higher sample sizes, including other subgroups of heart failure, to achieve a more accurate assessment.

Other

OP-012

Cardiology assistants' perceptions of the specialty education and training environment in Turkey

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Background and Aim: The learning environment is one of the core components of specialty training. Accurate evaluation of learning environments holds significant importance as it enables the identification of educational deficiencies and allows for a targeted approach to enhance the effectiveness of education. Assessing how learners perceive specialty education and educational environments in the field of medicine is invaluable for identifying the necessary modifications required to attain desired learning objectives. However, limited data exists regarding cardiology residency training in both our country and globally. The aim of this study was to evaluate the perceptions of cardiology residents in Turkey regarding the specialty education and training environment and investigate the relationship between these perceptions and various variables.

Methods: The study included 133 cardiology residents from 44 cardiology clinics in Turkey. Stratified sample selection and quantitative data collection methods were employed. The Postgraduate Hospital Educational Environment Measure (PHEEM) and an education questionnaire were utilized, and data were collected electronically. The study examined the effects of age, gender, medical school graduation, professional year, residency level, type of institution, weekly training hours, weekly night shifts, and geographic region on the residents' perceptions of the educational environment.

Results: This study found that view the cardiac education environment as positive but needing improvement according to residents. Most favorable influences on educational environment perception: Foundation university, first year assistantship, and 2 or more weekly training hours. PHEEM scores were unaffected by age, gender, university of graduation,

Table 1. Resuls of train and test

ALGORITHM	MODEL TYPE	GROUP A						GOUP B					
		TRAİN			TEST			TRAİN			TEST		
		ACCURACY(%)	SENSITIVITY(%)	SPECİFİCİTY(%)	ACCURACY(%)	SENSITIVITY(%)	SPECİFİCİTY(%)	ACCURACY(%)	SENSITIVITY(%)	SPECİFİCİTY(%)	ACCURACY(%)	SENSITIVITY(%)	SPECİFİCİTY(%)
Tree	Fine Tree	96,3	96,7	95,9	96,3	96,7	96,7	96,3	95,2	97,4	95	96,6	95,3
	Medium Tree	96,3	96,7	95,9	96,7	96,7	96,7	96,3	95,2	97,4	95	96,6	93,5
	Coarse Tree	96,3	96,7	95,9	96,7	96,7	95,9	95,4	94,4	96,6	96,7	96,7	96,7
Ensemble	Boosted Trees	97,1	96,7	97,5	98,3	96,8	100	54,8	52,6	92,3	50	50	---
	Bagged Trees	95,9	95,5	95,8	96,7	96,7	96,7	95,4	95,8	95	98,3	96,8	100
	Subspace Discriminant	50,2	50,2	---	50	50	---	68,5	62,2	89,3	60	55,6	100
	RUSboosted Trees	95,9	95,9	95,8	96,7	96,7	96,7	96,3	95,2	97,4	96,7	96,7	96,7
Neural Network	Medium Neural Network	50,2	50,2	---	50	50	---	50,2	50,2	---	50	50	---
	Narrow Neural Network	50,2	50,2	---	50	50	---	50,2	50,2	---	50	50	---
KNN	Fine KNN	50,2	50,2	---	50	50	---	50,2	50,2	---	50	50	---
	Medium KNN	50,2	50,2	---	50	50	---	50,2	50,2	---	50	50	---
	Coarse KNN	50,2	50,2	---	50	50	---	50,2	50,2	---	50	50	---

Group A: Consists of 56 parameters. Group B: consists of 39 parameters. There is a difference between the two groups in terms of echocardiographic findings used.

weekly shift hours, or geography. Many speciality clinics lack assistant handbooks and adaption training. Residents need training lectures, but some clinics don't offer enough. Clinics require better shift lunches, security, and assistant rooms. The Mediterranean region is significantly lower than other regions, especially in education. The study identified several problematic areas, including the physical environment, working hours and workload, training, and the inadequacy of training hours.

Conclusions: These findings provide valuable insights into the perspectives of Turkish cardiology residents regarding the educational environment and highlight areas that require improvement within the educational process. Curriculum designers, clinic and hospital administrators, and health policymakers can utilize these findings to identify current issues in general cardiology education and conduct further research to initiate improvement measures.

Interventional Cardiology / Coronary

OP-013

Frequency of contrast-induced nephropathy in patients with a history of coronary angiography and SGLT-2 inhibitor use

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Background and Aim: Presence of diabetes mellitus is accepted as a risk factor for the development of contrast-induced nephropathy (CN) after coronary angiography. Sodium-glucose Cotransporter 2 Inhibitors (SGLT2i) regulate intraglomerular hemodynamics and reduce glomerulosclerosis and tubulointerstitial fibrosis. In this study, we aimed to investigate the effectiveness of SGLT2i on CN in patients who had previously undergone coronary angiography.

Methods: Our retrospective study included 270 patients who underwent coronary angiography, were diagnosed with type 2 diabetes mellitus and had a glomerular filtration rate (GFR) >30 mL/min/1.73 m^2. Creatinin level of the patients were recorded preprocedural and 48th hours after the procedure. A 25% or 0.5 mg/dL increase in creatinine level compared to pre-procedure was defined as contrast nephropathy. The patients were divided into two groups as those who use SGLT-2i and those who do not. These groups were compared with each other in terms of clinical demographic and laboratory parameters. In addition, the change in creatinine levels according to the HbA1c levels of these groups was examined.

Results: There was no difference in age, gender and body mass index between the patients who took the SGLT2i and those who did not. No statistical difference was observed in the development of CN between the two groups(p=0.447). Compared with the baseline creatinine level, the 48th hour creatinine levels were statistically significantly increased in all patient population (change rate 6.22%), in those taking SGLT-2 (change rate 6.92%) and in those with HbA1c levels above 6.4% (change rate 6.56%) (p=0.006; p=0.032 and p=0.008, respectively). When re-divided into groups according to HbA1c levels, patients with low HbA1c levels (≤6.4%) did not have a significant increase in 48th hour creatinine levels compared with baseline values whether they received SGLT-2i or not. In patients with high HbA1c levels (≥6.4%), there was a significant increase in creatinine in the group receiving SGLT-2i.

Conclusions: In our study, the use of SGLT-2i did not cause a significant increase in CN, but may cause significant increases in creatinine progression in patients with uncontrolled diabetes. Further large-scale studies are needed to determine the benefits or harms of these drugs in CN.

Table 1. Demographic characteristics of the study populations

	Patients using SGLT-2 inhibitors (n=143)	Patients not using SGLT-2 inhibitors (n=127)	p
Age, year	63.4 ± 8.8	62.6 ± 8.7	0.425
Female, n (%)	61 (42.7)	54 (42.5)	0.982
BMI, kg/m^2	28.8 ± 4.9	28.6 ± 5.1	0.590
Indication of coronary angiography			
CCS, n (%)	58 (40.6)	71 (55.9)	0.015
USAP, n (%)	11 (7.7)	15 (11.8)	0.015
NSTEMI, n (%)	48 (33.6)	26 (20.5)	0.015
STEMI, n (%)	26 (18.2)	15 (11.8)	0.015
Risk factors			
HT, n (%)	111 (77.6)	97 (76.4)	0.808
CAD, n (%)	59 (41.3)	68 (53.5)	0.044
HL, n (%)	66 (46.2)	65 (51.2)	0.409
Stroke, n (%)	17 (11.9)	9 (7.1)	0.182
PAD, n (%)	7 (4.9)	15 (11.8)	0.038
Smoking, n (%)	46 (32.2)	40 (31.5)	0.906
Medication			
ACEi/ARB	71 (49.7)	66 (52.0)	0.704
Beta-blocker	51 (35.7)	47 (37.0)	0.819
Diuretics	56 (39.2)	53 (41.7)	0.667
NSAID	15 (10.5)	11 (8.7)	0.611

p value of less than 0.05 was statistically significant. Continuous variables were expressed as mean ± SD, and categorical variables were expressed as median and range. BMI: Body mass index, CCS: Chronic coronary syndromes, USAP: Unstabl angina pectoris, NSTEMI: Non-ST-elevated myocardial infarctus, STEMI: ST-elevated myocardial infarctus, HT: Hypertension, CAD: Coronary artery disease, HL: Hyperlipidemia, PAD: Peripheral artery disease, ACEi: Angiotensin converting enzyme inhibitör, ARB: Angiotensin reseptor blocker, NSAID: Non-steroidal anti-inflamatuar drugs, SD: Standard deviation.

Table 2. Laboratory findings of the study populations

	Patients using SGLT-2 inhibitors (n=143)	Patients not using SGLT-2 inhibitors (n=127)	p
Sodium, mEq/L	136.7 ± 3.9	136.4 ± 3.2	0.460
Potasium, mEq/L	4.31 ± 0.5	4.20 ± 0.44	0.062
Creatinine preprocedural, mg/dL	0.86 (0.31-1.92)	0.78 (0.36-1.65)	0.068
Troponin I, ng/L	100 (1-26495)	45 (2-27000)	0.664
Uric acid, mg/dL	5.62 ± 1.79	4.94 ± 1.45	0.001
CRP, mg/L	4.8 (0-194)	4.0 (0-177)	0.756
Hb, g/dL	13.0 ± 1.94	13.3 ± 2.0	0.364
WBC, 10^3/µL	9.52 ± 3.1	9.19 ± 4.0	0.447
PLT, 10^3/µL	269 ± 92	260 ± 81	0.379
HbA1c, %	7.6 ± 2	8.21 ± 1.9	0.012
LDL-C, mg/dL	105 ± 39.2	103 ± 43.5	0.696
EF, %	51.3 ± 9.7	52.5 ± 8.5	0.254
Contrast volume, mL	120 (35-450)	120 (40-400)	0.902
Contrast-induced nephropathy, n (%)	21 (14.7)	23 (18.1)	0.447

p value of less than 0.05 was statistically significant. Continuous variables were expressed as mean ± SD, and categorical variables were expressed as median and range. CRP: C-reactive protein, Hb: Hemoglobin, WBC: White blood cell, PLT: Platelet, HbA1c: Glycosylated hemoglobin, LDL-C: Low density lipoprotein cholesterol, EF: Ejection fraction, SD: Standard deviation.

Table 3. Comparison of change in creatinine level in subgroups

	Baseline creatinine, mg/dL	48th hour creatinine, mg/dL	Rate of change	p
All patients	0.85 ± 0.26	0.89 ± 0.30	6.22%	0.006
Patients not using SGLT-2 inhibitors	0.88 ± 0.26	0.92 ± 0.34	5.61%	0.074
Patients using SGLT-2 inhibitors	0.82 ± 0.24	0.85 ± 0.23	6.92%	0.032
HbA1c ≤6.4%	0.9 ± 0.3	0.94 ± 0.37	5.33%	0.360
HbA1c >6.4%	0.83 ± 0.23	0.87 ± 0.26	6.56%	0.008

p value of less than 0.05 was statistically significant. SGLT-2i: Sodium-glucose cotransporter 2 inhibitors, HbA1c: Glycosylated hemoglobin.

Table 4. Chane in creatinine levels according to subgroup HbA1c level

	Baseline creatinine, mg/dL	48th hour creatinine, mg/dL	Rate of change	p
HbA1c ≤6.4%				
Patients not using SGLT-2 inhibitors	0.92 ± 0.33	0.96 ± 0.42	6.1%	0.365
Patients using SGLT-2 inhibitors	0.86 ± 0.21	0.87 ± 0.20	3.4%	0.726
HbA1c >6.4%				
Patients not using SGLT-2 inhibitors	0.86 ± 0.21	0.90 ± 0.29	5.3%	0.130
Patients using SGLT-2 inhibitors	0.81 ± 0.25	0.85 ± 0.24	7.6%	0.026

P value of less than 0.05 was statistically significant. SGLT-2i: Sodium-glucose cotransporter 2 inhibitors, HbA1c: Glycosylated hemoglobin.

Interventional Cardiology / Valve and Structural Heart Disease

OP-014

The relationship between myocardial bridging and fatal ventricular arrhythmias in patients with hypertrophic cardiomyopathy: The HCM-MB study

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Background and Aim: Myocardial bridging (MB) and hypertrophic cardiomyopathy (HCM) are associated with risk of fatal ventricular arrhythmias (VAs). The goal of the study was to determine the relationship between MB and fatal VAs in HCM patients with implantable cardiac defibrillators (ICD).

Methods: A total of 108 HCM patients (mean age: 46.6 ± 13.6 years; male: 73) were enrolled in this retrospective study. All patients underwent transthoracic echocardiography and coronary computed tomography angiography. Fatal VAs including sustained ventricular tachycardia and ventricular fibrillation which were documented from ICD records.

Results: There were documented fatal VAs in 29 (26.8%) patients during a mean follow-up time of 71.3 ± 30.9 months. Sudden cardiac death (SCD) risk score (HR: 1.153; 95% CI: 1.024-1.300; p=0.019), presence of MB (HR: 10.778; 95% CI: 1.065-109.056; p=0.044), depth of MB (HR: 4.356; 95% CI: 1.469-12.918; p=0.008), and length of MB (HR: 1.133; 95% CI: 1.046-1.228; p=0.002) were found to be independent predictors of fatal VAs in HCM patients. The prevalence of fatal VAs was significantly higher in patients with MB ≥ 21.5 mm (62.9 vs. 9.6%; p<0.001). This is the first study in the scientific literature describing the effects of MB on fatal VAs in HCM patients with ICD.

Conclusions: The current data suggest that the presence of deep and long MB, and SCD risk score were independent risk factors for fatal VAs in patients with HCM. In addition to conventional risk factors, coronary anatomical course can provide clinicians with valuable information when assessing the risk of fatal VAs in HCM patients.

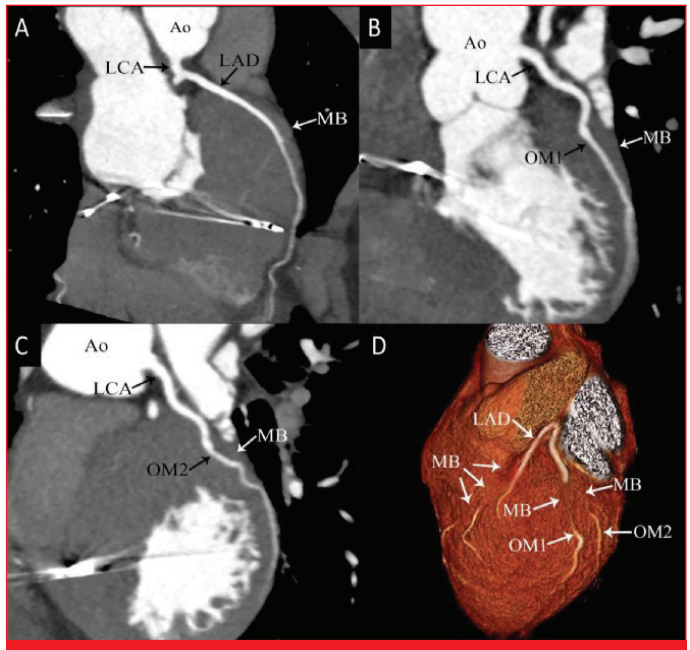

Figure 1. Example of myocardial bridging on cardiac coronary computed tomography angiography in a HCM patient. Respectively, modified 2 chamber and short axis view demonstrating myocardial bridging of the left anterior descending artery (depth:4.5 mm, length: 45 mm) (A) and the first (depth:4.8 mm, length: 35 mm) and second (depth:4.1 mm, length: 22 mm) obtuse marginal arteries (B, C). Three dimensional reconstruction showing myocardial bridging of the left anterior descending artery and obtuse marginal arteries (white arrow) (D). Ao: Aort, LCA: Left main coronary artery, LAD: Left anterior descending, MB: Myocardial bridging, OM1: The first obtuse marginal artery, OM2: Second obtuse marginal artery.

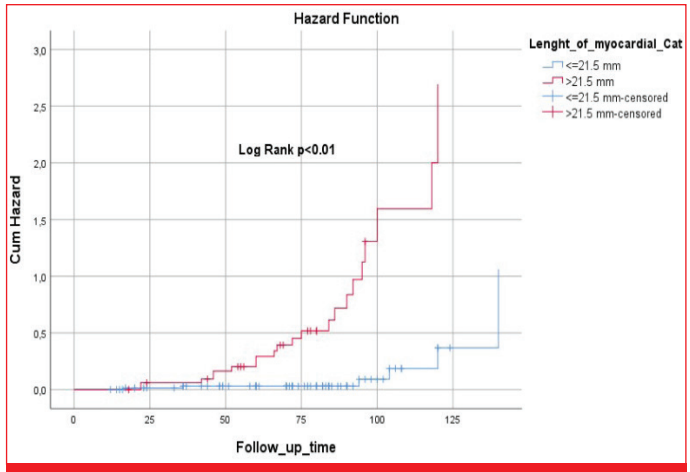

Figure 2. Kaplan-Meier survival analysis revealed that long term cumulative hazard ratio of fatal VAs was found to be significantly increased in patients with higher than >21.5 mm length of MB (MB: Myocardial bridging, VA: Ventricular arrhythmias).

Table 1. Comparison of the demographic and cardiac imaging characteristics of HCM patients with and without fatal ventricular arrhythmias.

Variables	HCM patients without fatal VAs (n=79)	HCM patients with fatal VAs (n=29)	p
Age, years	47.47 ± 14.14	44.31 ± 12.26	0.290[a]
Gender (male), n (%)	56 (70.9)	17 (58.6)	0.165[b]
Hypertension, n (%)	28 (35.4)	8 (27.6)	0.299[b]
Diabetes mellitus, n (%)	7 (8.9)	4 (13.8)	0.334[b]
Current smoker, n (%)	21 (26.6)	6 (20.7)	0.360[b]
CKD, n (%)	9 (11.4)	2 (6.9)	0.390[b]
Hyperlipidemia, n (%)	29 (36.7)	9 (31.0)	0.378[b]
Family history of SCD, n (%)	27 (34.2)	12 (41.4)	0.319[b]
History of SCA, n (%)	17 (21.5)	7 (24.1)	0.479[b]
Syncope, n (%)	35 (44.3)	13 (44.8)	0.566[b]
Obstructive cardiomyopathy, n (%)	50 (63.3)	19 (65.5)	0.509[b]
Atrial fibrillation, n (%)	10 (12.7)	2 (6.9)	0.322[b]
Marrow surgery, n (%)	6 (7.6)	1 (3.4)	0.393[b]
Alcohol septal ablation, n (%)	9 (11.4)	2 (6.9)	0.390[b]
Follow-up time (months)	67.94 ± 27.59	80.38 ± 37.81	0.113[a]
SCD risk score	5.31 ± 1.72	9.11 ± 3.49	<0.001[c]
NYHA class, n (%)			
I	26 (32.9)	8 (27.6)	0.389[b]
II	45 (57.0)	18 (62.1)	0.401[b]
III	7 (8.9)	3 (10.3)	0.535[b]
Medications Used, n (%)			
Antiplatelet agents	13 (16.5)	9 (31.0)	0.084[b]
Beta-blockers	79 (100.0)	29 (100.0)	>0.05
CCB	14 (17.7)	5 (17.2)	0.600[b]
Disopyramide	11 (13.9)	9 (31.0)	0.044[b]
Sotalol	5 (6.3)	5 (17.2)	0.091[b]
ACEi/ARB	25 (31.6)	3 (10.3)	0.019[b]
Statin	17 (21.5)	8 (27.6)	0.336[b]
VKA	5 (6.3)	2 (6.9)	0.607[b]
DOAC	6 (7.6)	2 (6.9)	0.633[b]
Diuretics	6 (7.6)	2 (6.9)	0.633[b]
Echocardiography			
LVEF, %			
>50	74 (93.7)	28 (96.6)	0.487[b]
40-50	2 (2.5)	1 (3.4)	0.613[b]
<40	3 (3.8)	-	0.387[b]
LV diastolic function			
Normal	15 (19.0)	11 (37.9)	0.040[b]
Stage I	31 (39.2)	10 (34.5)	0.413[b]
Stage II	32 (40.5)	7 (24.1)	0.088[b]
Stage III	1 (1.3)	1 (3.4)	0.467[b]
LA diameter, mm	42.86 ± 5.75	40.59 ± 5.32	0.099[c]
LVEDD, mm	43.32 ± 3.89	43.34 ± 3.53	0.028[a]
LVESD, mm	25.89 ± 4.55	25.48 ± 3.59	0.854[c]
IVST, mm	24.25 ± 4.54	25.61 ± 4.01	0.159[a]
PWT, mm	13.84 ± 3.05	13.21 ± 2.04	0.457[c]
LVOT gradient, mmHg	55.41 ± 39.08	59.03 ± 35.57	0.467[c]
SAM, n (%)	45 (57.0)	17 (58.6)	0.528[b]
Apical aneurysm, n (%)	2 (2.5)	1 (3.4)	0.613[b]
>moderate MR	7 (8.9)	-	0.104[b]
MVR, n (%)	4 (5.1)	2 (6.9)	0.513[b]

CCTA			
Indications for coronary imaging, n (%)			
Angina pectoris	22 (27.8)	15 (51.7)	0.019[b]
Atypical chest pain	30 (38.0)	9 (31.0)	0.333[b]
Pre-ablation	8 (10.1)	2 (6.9)	0.465[b]
Pre-surgery	8 (10.1)	1 (3.4)	0.246[b]
Other	11 (13.9)	2 (6.9)	0.263[b]
Agatston score	55.22 ± 104.58	65.14 ± 106.66	0.676[c]
Patients with score, n (%)			
0	44 (55.7)	16 (55.2)	0.566[b]
1-99	21 (26.6)	6 (20.7)	0.360[b]
100-399	11 (13.9)	7 (24.1)	0.165[b]
>400	2 (2.5)	-	0.533[b]
Obstructive coronary artery disease, n (%)	10 (12.7)	5 (17.2)	0.371[b]
Myocardial bridging, n (%)	30 (38.0)	24 (82.8)	<0.001[b]
Depth of myocardial bridging	0.66 ± 0.89	3.32 ± 1.92	<0.001[b]
Depth of myocardial bridging, n (%)			
Superficial (1-2 mm)	23 (29.1)	2 (6.9)	0.011[b]
Deep (2-5 mm)	5 (6.3)	18 (62.1)	<0.001[b]
Very deep (>5 mm)	0	7 (24.1)	<0.001[b]
Length of myocardial bridging, mm	8.06 ± 11.18	22.00 ± 10.78	<0.001[c]
Long myocardial bridging, n (%)	9 (11.4)	19 (65.5)	<0.001[b]
Patients with >1 myocardial bridging, n (%)	0	5(17.2)	<0.001[b]
Coronary localization of myocardial bridging, n (%)			
LAD	14 (17.7)	23 (79.3)	<0.001[b]
LCX	9 (11.4)	5 (17.2)	0.306[b]
RCA	6 (7.6)	2 (6.9)	0.633[b]
Segmental localization of myocardial bridging, n (%)			
Proximal	15 (19.0)	20 (69.0)	<0.001[b]
Mid	10 (12.7)	16 (55.2)	<0.001[b]
Distal	9 (11.4)	2 (6.9)	0.390[b]
Treatment of myocardial bridging, n (%)			
Medical	26 (32.9)	21 (72.4)	<0.001[b]
PCI	-	1 (3.4)	0.269[b]
CABG	-	2 (6.9)	0.070[b]
LGE, n (%)	26 (32.9)	17 (58.6)	0.014[b]
Mortality, n (%)	2 (2.5)	1 (3.4)	0.613[b]

ACEi: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, CABG: Coronary artery bypass graft, CCB: Calcium channel blocker, CKD: Chronic kidney disease, CCTA: Coronary computed tomography angiography, DOACs: Direct oral anticoagulants, HCM: Hypertrophic cardiomyopathy, IVST: Interventricular septal thickness, LA: Left atrium, LAD: Left anterior descending, LCX: Left circumflex, LGE: Late gadolinium enhancement, LVEDD: Left ventricular end diastolic diameter, LVEF: Left ventricular ejection fraction, LVESD: Left ventricular end systolic diameter, LVOT: Left venticular outflow tract, MB: Myocardial bridging, MR: Mitral regurgitation, MVR: Mitral valve replacement, PCI: Percutaneous coronary intervention, PWT: Posterior wall thickness, RCA: Right coronary artery, SAM: Systolic anterior motion of mitral valve, SCA: Sudden cardiac arrest, SCD: Sudden cardiac death, VAs: Ventricular arrhythmias, VKA: Vitamin K antagonist.

Table 2. Cox proportional hazard analysis showing independent predictors of fatal ventricular arrhythmias in HCM patients

Parameters	HR	95% CI	p
SCD risk score	1.153	1.024-1.300	0.019
LGE	0.586	0.257-1.336	0.204
Myocardial bridging	10.778	1.065-109.056	0.044
Length of myocardial bridging	1.133	1.046-1.228	0.002
Depth of myocardial bridging	4.356	1.469-12.918	0.008

CI: Confidence interval, HCM: Hypertrophic cardiomyopathy, HR: Hazard ratio, LGE: Late gadolinium enhancement, SCD: Sudden cardiac death.

Table 3. Comparison of the demographic and cardiac imaging characteristics of HCM patients with MB 21.5 mm cut off value.

Variables	HCM patients with MB <21.5 mm	HCM patients with MB ≥21.5 mm	p
Baseline demographics	47.41 ± 13.31	44.97 ± 14.47	0.388[a]
Age, years			
Gender (male), n (%)	51 (69.9)	22 (62.9)	0.303[b]
Hypertension, n (%)	25 (34.2)	11 (31.4)	0.475[b]
Diabetes mellitus, n (%)	4 (5.5)	7 (20.0)	0.026[b]
Current smoker, n (%)	21 (28.8)	6 (17.1)	0.142[b]
CKD, n (%)	7 (9.6)	4 (11.4)	0.504[b]
Hyperlipidemia, n (%)	26 (35.6)	12 (34.3)	0.535[b]
Family history of SCD, n (%)	27 (37.0)	12 (34.3)	0.479[b]
History of SCA, n (%)	17 (23.3)	7 (20.0)	0.452[b]
Syncope, n (%)	36 (49.3)	12 (34.3)	0.103[b]
Obstructive cardiomyopathy, n (%)	46 (63.0)	23 (65.7)	0.479[b]
Atrial fibrillation, n (%)	9 (12.3)	3 (8.6)	0.412[b]
Marrow surgery, n (%)	6 (8.2)	1 (2.9)	0.272[b]
Alcohol septal ablation, n (%)	8 (11.0)	3 (8.6)	0.496[b]
Follow-up time, months	71.52 ± 31.33	70.77 ± 30.69	0.907[a]
SCD risk score	5.97 ± 2.50	7.09 ± 3.42	0.134[c]
NYHA class, n (%)			
I	21 (28.8)	13 (37.1)	0.254[b]
II	43 (58.9)	20 (57.1)	0.512[b]
III	8 (11.0)	2 (5.7)	0.310[b]
Medications used, n (%)			
Antiplatelet agents	13 (17.8)	9 (25.7)	0.240[b]
Beta-blockers	73 (100.0)	35 (100.0)	>0.05
CCB	11 (15.1)	8 (22.9)	0.231[b]
Disopyramide	7 (9.6)	13 (37.1)	0.001[b]
Sotalol	3 (4.1)	7 (20.0)	0.013[b]
ACEi/ARB	23 (31.5)	5 (14.3)	0.044[b]
Statin	14 (19.2)	11 (31.4)	0.122[b]
VKA	5 (6.8)	2 (5.7)	0.593[b]
DOAC	5 (6.8)	3 (8.6)	0.512[b]
Diuretics	5 (6.8)	3 (8.6)	0.512[b]

Echocardiography	68 (93.2)	34 (97.1)	0.364[b]
LVEF, %	2 (2.7)	1 (2.9)	0.695[b]
>50	3 (4.1)	-	0.305[b]
40-50	16 (21.9)	10 (28.6)	0.299[b]
<40	26 (35.6)	15 (42.9)	0.302[b]
LV diastolic function	29 (39.7)	10 (28.6)	0.180[b]
Normal	2 (2.7)	-	0.455[b]
Stage I	42.68 ± 5.92	41.34 ± 5.18	0.309[c]
Stage II	43.58 ± 3.90	42.80 ± 3.52	0.321[a]
Stage III	26.14 ± 4.42	25.03 ± 3.99	0.300[c]
LAD, mm	24.03 ± 4.51	25.83 ± 4.04	0.047[a]
LVEDD, mm	13.94 ± 3.20	13.11 ± 1.73	0.433[c]
LVESD, mm	54.08 ± 36.70	61.17 ± 40.84	0.361[c]
IVST, mm	44 (60.3)	18 (51.4)	0.253[b]
PWT, mm	-	3 (8.6)	0.032[b]
LVOT gradient, mmHg	6 (8.2)	1 (2.9)	0.272[b]
SAM, n (%)	4 (5.5)	2 (5.7)	0.636[b]
Apical aneurysm, n (%)			
>moderate MR			
MVR, n (%)			
CCTA Indications for coronary imaging, n (%)			
Angina pectoris	19 (26.0)	18 (51.4)	0.009[b]
Atypical chest pain	29 (39.7)	10 (28.6)	0.180[b]
Pre-ablation	7 (9.6)	3 (8.6)	0.586[b]
Pre-surgery	8 (11.0)	1 (2.9)	0.145[b]
Other	10 (13.7)	3 (8.6)	0.336[b]
Agatston score	59.49 ± 107.63	54.51 ± 99.88	0.584[c]
Patients with score, n (%)			
0	38 (52.1)	22 (62.9)	0.198[b]
1-99	21 (28.8)	6 (17.1)	0.142[b]
100-399	11 (15.1)	7 (20.0)	0.350[b]
>400	2 (2.7)	-	0.455[b]
Obstructive coronary artery disease, n (%)	9 (12.3)	6 (17.1)	0.344[b]
Myocardial bridging, n (%)	20 (27.4)	34 (97.1)	<0.001[b]
Depth of myocardial bridging	0.50 ± 1.01	3.18 ± 1.46	<0.001[c]
Depth of myocardial bridging, n (%)			
Superficial (1-2 mm)	14 (19.2)	11 (31.4)	0.122[b]
Deep (2-5 mm)	4 (5.5)	19 (54.3)	<0.001[b]
Very deep (>5 mm)	1 (1.4)	6 (17.1)	0.004[b]
Patients with >1 myocardial bridging, n (%)	-	5 (14.3)	0.003[b]
Coronary localization of myocardial bridging, n (%)			
LAD	8 (11.0)	29 (82.9)	<0.001[b]
LCX	6 (8.2)	8 (22.9)	0.038[b]
RCA	4 (5.5)	4 (11.4)	0.233[b]
Segmental localization of myocardial bridging, n (%)			
Proximal	7 (9.6)	28 (80.0)	<0.001[b]
Mid	4 (5.5)	22 (62.9)	<0.001[b]
Distal	6 (8.2)	5 (14.3)	0.257[b]
Treatment of myocardial bridging, n (%)			
Medical	15 (20.5)	32 (91.4)	<0.001[b]
PCI	1 (1.4)	-	0.676[b]
CABG	-	2 (5.7)	0.103[b]
LGE, n (%)	26 (35.6)	17 (48.6)	0.141[b]
Fatal Vas, n (%)	7 (9.6)	32 (62.9)	<0.001[b]
Mortality, n (%)	2 (2.7)	1 (2.9)	0.695[b]

ACEi: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, CABG: Coronary artery bypass graft, CCB: Calcium channel blocker, CKD: Chronic kidney disease, CCTA: Coronary computed tomography angiography, DOACs: Direct oral anticoagulants, HCM: Hypertrophic cardiomyopathy, IVST: Interventricular septal thickness, LA: Left atrium, LAD: Left anterior descending, LCX: Left circumflex, LGE: Late gadolinium enhancement, LVEDD: Left ventricular end diastolic diameter, LVEF: Left ventricular ejection fraction, LVESD: Left ventricular end systolic diameter, LVOT: Left venticular outflow tract, MB: Myocardial bridging, MR: Mitral regurgitation, MVR: Mitral valve replacement, PCI: Percutaneous coronary intervention, PWT: Posterior wall thickness, RCA: Right coronary artery, SAM: Systolic anterior motion of mitral valve, SCA: Sudden cardiac arrest, SCD: Sudden cardiac death, VAs: Ventricular arrhythmias, VKA: Vitamin K antagonist.

Interventional Cardiology / Coronary

OP-016

Clinical implication of totally occluded infarct related coronary artery in non-ST-segment elevation myocardial infarction: The TOTAL-NSTEMI study

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Background and Aim: A subset of patients found to have total occlusion of the culprit artery (TOCA), present with non-ST-segment elevation myocardial infarction (NSTEMI) and elevated biomarkers. The aim of this study is to assess the effect of the TOCA in patients presenting with NSTEMI.

Methods: This multicenter observational study was retrospectively conducted between 2015 and 2019. Thrombolysis in myocardial infarction (TIMI) flow grade 0 to 1 was defined as the TOCA. The primary endpoint included a combination of all-cause death, myocardial infarction, target vessel revascularization, stent thrombosis, and stroke.

Results: Of 3272 patients, TIMI 0 to 1 flow in the culprit artery was present in 488 (14.9%) patients. TOCA was more likely to be of thrombotic origin (54.1% vs. 10.3%, p<0.001) and visible collaterals (22.5% vs. 4.4%, p<0.001). The rates of 30-day (14.3% vs. 7.2%, p<0.001), 2-year (25% vs. 19.1%, p=0.003) primary end-points were significantly higher in TOCA patients. Fatal arrhythmias were remarkably higher at 30-day and 2-year follow-up (8.6% vs. 4%, p<0.001), (9% vs. 5.2%, p=0.001) respectively. Mechanical complications were also higher in patients with TOCA at 30-day (0.8% vs. 0.2%, p=0.013). Moreover, TOCA (OR: 1.379; p=0.001) was one of the independent predictors of MACCE in NSTEMI patients.

Conclusions: The current data suggest that patients with TOCA in the context of NSTEMI are under higher risk of MACCE, fatal arrhytmias, and mechanical complications.

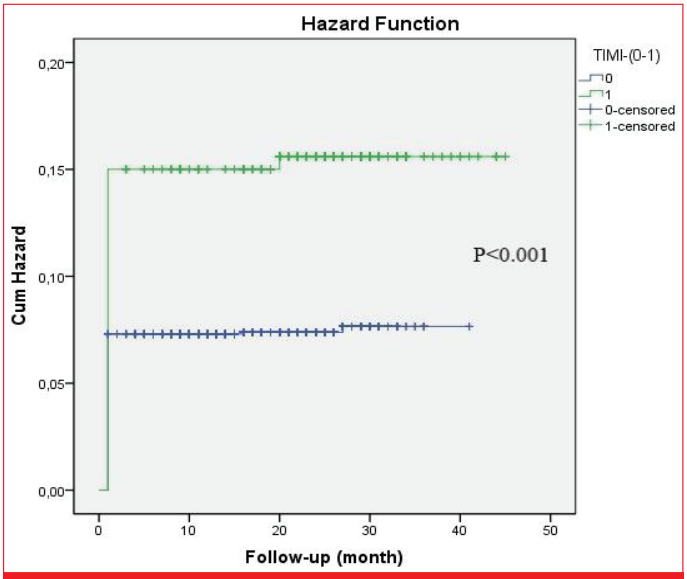

Figure 1. Kaplan-Meier estimates of primary end point (MACCE) at 2-year.

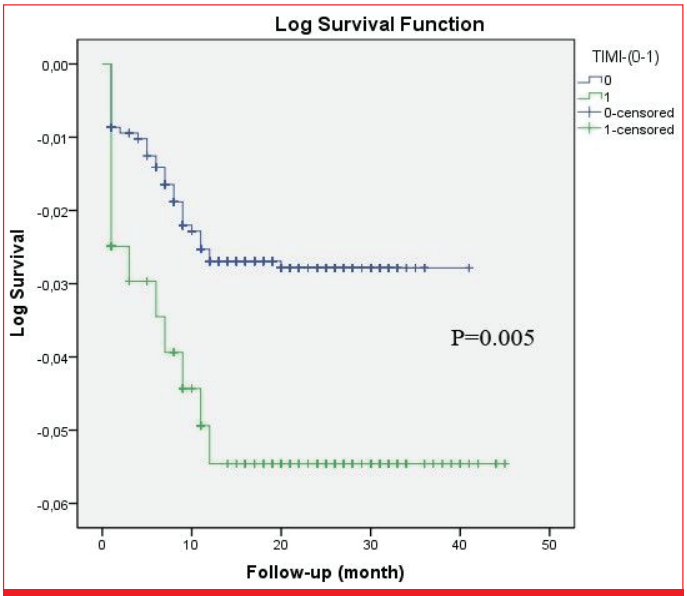

Figure 2. Kaplan-Meier survival analysis revealed that 2-year survival was found to be significantly decreased in patients with total occlusion of the culprit artery.

Table 1. Univariate and multivariate Cox Regression analysis showing independent predictors of MACCE in non-ST-segment elevation myocardial infarction patients

	Univariate Level		Multivariate Level	
	OR (95% CI)	p value	OR (95% CI)	p value
Age	0.992 (0.986-0.999)	0.023	0.993 (0.986-0.999)	**0.039**
Dyspnea	1.339 (1.018-1.761)	0.037	1.310 (0.995-1.723)	0.054
Pseudo-normalization	0.288 (0.072-1.154)	0.079	3.701 (0.923-14.837)	0.065
Atrial fibrillation	1.815 (1.272-2.590)	0.001	0.541 (0.379-0.772)	**0.001**
TOCA	0.731 (0.600-0.890)	0.002	1.379 (1.132-1.680)	**0.001**

Abbreviations: CI: Confidence Interval; MACCE: Major cardiovascular and cerebral events; TOCA: Total occlusion of the culprit artery

Table 2. Baseline demographic, electrocardiographic, and and clinical characteristics of patients with or without TOCA presenting with non-ST-segment elevation myocardial infarction

Variables	TIMI-0/1 (n=488)	TIMI-2/3 (n=2784)	p
Age, years	55.62 ± 11.34	57.25 ± 11.66	0.004
Gender (male), n (%)	398 (81.6)	1758 (63.1)	<0.001
Diabetes mellitus, n (%)	126 (25.8)	654 (23.5)	0.266
Hypertension, n (%)	286 (58.6)	1510 (54.2)	0.074
Current smoker, n (%)	188 (38.7)	1226 (44.1)	0.026
Hyperlipidemia, n (%)	172 (35.2)	982 (35.3)	0.991
History of cerebral embolism, n (%)	10 (2)	48 (1.7)	0.616
Chronic kidney disease, n (%)	70 (14.3)	610 (21.9)	<0.001
Previous MI, n (%)	98 (20.1)	592 (21.3)	0.555
Previous PCI, n (%)	80 (16.4)	548 (19.7)	0.089
Heart failure, n (%)	54 (11.1)	312 (11.2)	0.927
Peripheral arterial disease, n (%)	30 (6.1)	130 (4.7)	0.163
Clinical presentation, n (%)			
Chest pain	472 (96.7)	2580 (92.7)	<0.001
Dyspnea	64 (13.1)	292 (10.5)	0.086
Syncope	4 (0.8)	24 (0.9)	0.925
Baseline troponin T, ng/mL	0.59 (0.30-1.32)	0.60 (0.29-1.31)	0.663
Hemoglobin, g/dL	14.14 ± 1.96	13.90 ± 1.95	0.013
LV ejection fraction, %	55.50 ± 7.96	54.73 ± 8.09	0.053
Baseline electrocardiographic features, n (%)			
ST-segment depression ≥1 mm	212 (43.4)	848 (30.5)	0.001
T wave inversion	110 (22.5)	384 (13.8)	<0.001
Pseudo-normalization	8 (1.6)	24 (0.9)	0.108
LBBB	32 (6.6)	160 (5.7)	0.482
RBBB	28 (5.7)	248 (8.9)	0.010
Atrial fibrillation	12 (2.5)	86 (3.1)	0.451
TIMI risk score, n (%)			<0.001
0-2	59 (12.1)	894 (32.1)	
3-4	163 (33.4)	1337 (48)	
5-7	266 (54.5)	565 (20.3)	

LV: Left ventricle, LBBB: Left bundle branch block, MI: Myocardial infarction, PCI: Percutaneous coronary intervention, RBBB: Right bundle branch block, TIMI: Thrombolysis in myocardial infarction, TOCA: Total occlusion of the culprit artery.

Table 3. Angiographic characteristics of patients with or without TOCA presenting with non-ST-segment elevation myocardial infarction

Variables	TIMI-0/1 (n=488)	TIMI-2/3 (n=2784)	p
Culprit coronary artery, n (%)	104 (21.3)	1162 (41.7)	<0.001
-LAD	200 (41)	974 (35)	
-LCX	184 (37.7)	648 (23.3)	
-RCA			
Lesion morphology, n (%)	264 (54.1)	288 (10.3)	<0.001
-Thrombus	12 (2.5)	82 (2.9)	0.559
-Spasm			
Presence of collaterals, n (%)	110 (22.5)	122 (4.4)	<0.001
Myocardial blush, n (%)	184 (37.7)	116 (4.2)	<0.001
-0	72 (14.8)	394 (14.2)	
-1	232 (47.5)	2274 (81.7)	
-2			
-3			
Treatment timing (hours)	10.20 (2.65)	10.25 (2.64)	0.661
Treatment, n (%)	456 (93.4)	2700 (97)	<0.001
-Stent	32 (6.6)	94 (3.4)	
-Stent+ mechanical thrombectomy			
Stent type, n (%)	476 (97.5)	2714 (97.5)	0.942
-Drug	12 (2.5)	70 (2.5)	
-eluting			
-Bare metal			
Tirofiban use during PCI, n (%)	42 (8.6)	278 (10)	0.344

LAD: Left anterior descending, LCX: Left circumflex, PCI: Percutaneous coronary intervention, RCA: Right coronary artery.

Table 4. Clinical outcomes of study population

Variables	TIMI-0/1 (n=488)	TIMI-2/3 (n=2784)	p
30-day outcomes			
Primary end-point (MACCE), n (%)	70 (14.3)	200 (7.2)	0.001
-All cause mortality	8 (1.6)	14 (0.5)	0.005
-Myocardial infarction	42 (8.6)	130 (4.7)	<0.001
-Target vessel revascularization	40 (8.2)	48 (1.7)	<0.001
-Stent thrombosis	8 (1.6)	16 (0.6)	0.011
-Ischemic stroke	4 (0.8)	28 (1)	0.700
Secondary end-point, n (%)	62 (12.7)	134 (4.8)	<0.001
-TIMI-major bleeding	20 (4.1)	24 (0.9)	<0.001
-Fatal ventricular arrhythmias	42 (8.6)	112 (4)	<0.001
Mechanical complications, n (%)	4 (0.8)	5 (0.2)	0.013
-Acute severe MR due to PMR	1 (0.2)	3 (0.1)	0.571
-VSD	2 (0.4)	4 (0.1)	0.205
-LV Pseudoaneursym	2 (0.4)	2 (0.1)	0.108
2-year outcomes			
Primary end-point (MACCE), n (%)	122 (25)	532 (19.1)	0.003
-All cause mortality	24 (4.9)	72 (2.6)	0.005
-Myocardial infarction	64 (13.1)	356 (12.8)	0.842
-Target vessel revascularization	78 (16)	268 (9.6)	<0.001
-Stent thrombosis	10 (2)	48 (1.7)	0.616
-Ischemic stroke	6 (1.2)	44 (1.6)	0.560
Secondary end-point, n (%)	66 (13.5)	238 (8.5)	<0.001
TIMI-major bleeding	24 (4.9)	110 (4)	0.320
Fatal ventricular arrhythmias	44 (9)	144 (5.2)	0.001

LV: Left ventricle, MR: Mitral regurgitation, PMR: Papillary muscle rupture, TIMI: Thrombolysis in myocardial infarction, VSD: Venticular septal defect.

Table 5. Baseline demographic, electrocardiographic, and and clinical characteristics of patients with or without MACCE presenting with non-ST-segment elevation myocardial infarction

Variables	MACCE (-) (n=2618)	MACCE (+) (n=654)	p
Age (years), mean ± SD	57.26 ± 11.58	56.0 ± 11.79	0.009[a]
Gender (male), n (%)	1710 (65.3)	446 (68.2)	0.089[b]
Diabetes mellitus, n (%)	616 (23.5)	164 (25.1)	0.217[b]
Hypertension, n (%)	1442 (55.1)	354 (54.1)	0.347[b]
Current smoker, n (%)	1136 (43.5)	278 (42.5)	0.341[b]
Hyperlipidemia, n (%)	932 (35.6)	222 (33.9)	0.228[b]
History of cerebral embolism, n (%)	48 (1.8)	10 (1.5)	0.370[b]
Chronic kidney disease, n (%)	540 (20.6)	140 (21.4)	0.348[b]
Previous MI, n (%)	552 (21.1)	138 (21.1)	0.515[b]
Previous PCI, n (%)	496 (18.9)	132 (20.2)	0.252[b]
Heart failure, n (%)	302 (11.5)	64 (9.8)	0.114[b]
Peripheral arterial disease, n (%)	132 (5.0)	28 (4.3)	0.243[b]
Clinical presentation, n (%)	2436 (93.0)	616 (94.2)	0.170[b]
Chest pain	300 (11.5)	56 (8.6)	0.018[b]
Dyspnea Syncope	26 (1.0)	2 (0.3)	0.061[b]
Baseline troponin T, ng/mL	0.96 ± 1.01	0.95 ± 0.98	0.807[a]
Hemoglobin, g/dL	13.97 ± 1.93	13.84 ± 2.04	0.237[a]
Thrombotic culprit lesions, n (%)	424 (16.2)	124 (19.0)	0.052[b]
Baseline electrocardiographic features, n (%)			
ST-segment depression ≥1 mm	852 (32.5)	208 (31.8)	0.378[b]
T wave inversion	396 (15.1)	98 (15.0)	0.492[b]
Pseudo-normalization	30 (1.1)	2 (0.3)	0.031[b]
LBBB	154 (5.9)	38 (5.8)	0.516[b]
RBBB	210 (8.0)	66 (10.1)	0.054[b]
Atrial fibrillation	66 (2.5)	32 (4.9)	0.002[b]
Culprit coronary artery, n (%)			
LAD	1012 (38.7)	254 (38.8)	
LCX	942 (36.0)	232 (35.5)	0.969[c]
RCA	664 (25.4)	168 (25.7)	
TOCA, n (%)	366 (14.0)	122 (18.7)	0.002[b]

LAD: Left anterior descending, LCX: Left circumflex, LV: Left ventricle, LBBB: Left bundle branch block, MACCE: Major cardiovascular and cerebral events, MI: Myocardial infarction, PCI: Percutaneous coronary intervention, RBBB: Right bundle branch block, RCA: Right coronary artery, TIMI: Thrombolysis in myocardial infarction, TOCA: Total occlusion of the culprit artery, SD: Standard deviation. a: Mann-Whitney U Test, b: Fischer's Exact Test, c: Chi-Square Test.

Interventional Cardiology / Coronary

OP-017

Cardiovascular outcomes after mini-crush or double kissing crush stenting techniques for complex bifurcation lesions: The EVOLUTE-CRUSH registry

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Background and Aim: Comparison of clinical outcomes of double kissing crush (DKC) and mini-crush (MC) techniques in patients with complex coronary bifurcation lesions is lacking. This study sought to determine the clinical results of DKC and MC stenting techniques in mid-term follow-up.

Methods: This retrospective study included a total of 269 consecutive patients with complex bifurcation lesions who underwent percutaneous coronary intervention; 132 (49.1%) of them treated with MC technique, whereas 137 (50.9%) treated with DKC technique. The primary endpoint was target lesion failure (TLF), defined as the combination of cardiac death, target vessel miyocardial infarction (MI), or clinically driven-target lesion revascularization (TLR). This is the first study to compare the cardiovascular outcomes of DKC and MC stenting techniques in patients with complex bifurcation lesions.

Results: The SYNTAX scores were similar in both groups [23 (20-30) vs. 23 (19-28), p=0.631). The number of number of balloons (6.31 ± 1.80 vs. 4.42 ± 0.87, p<0.001) and guidewires (3.55 ± 0.83 vs. 2.86 ± 0.74, p<0.001) used, fluoroscopy time (21.55 ± 7.05 vs. 16.66 ± 4.19 min, p<0.001), and procedure time (80.42 ± 27.95 vs. 69.61 ± 18.97 min, p<0.001) were significantly higher in the DKC group. The rate of composite TLF was similar in complex bifurcation patients treated with MC than those treated with the DKC technique (13.6 vs. 12.4%, p=0.453). Moreover, both groups had similar rates in terms of cardiac death or all-caused death, target vessel related-MI, clinically driven TLR, and stent thrombosis.

Conclusions: The present study showed that both techniques of bifurcation treatment met high procedural success with low complication and similar TLF rates.

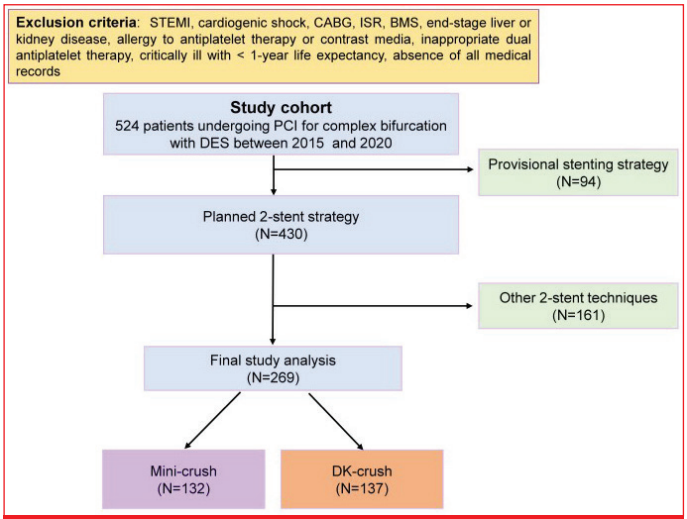

Figure 1. Flow chart for patient selection
BMS: Bare-metal stent, CABG: Coronary artery bypass grafting, ISR: In-stent restenosis, PCI: Percutaneous coronary intervention, DES: Drug-eluting stent, DK: Double kissing, STEMI: ST-elevation myocardial infaction.

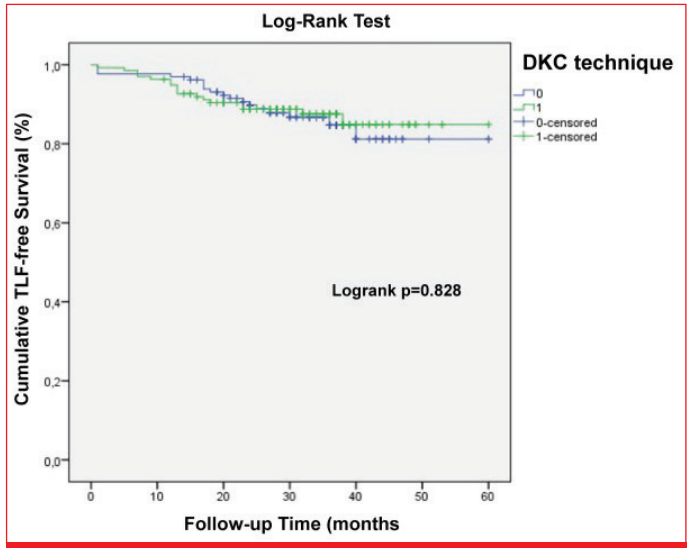

Figure 2. Kaplan-Meier survival analysis for primary endpoint during follow-up.
DKC: Double kissing crush, TLF: Target lesion failure.

Table 1. Baseline demographic and clinical characteristics of the study patients.

Variables	Mini-crush (n=132)	DK-crush (n=137)	p
Age, years	59.89 ± 10.93	60.40 ± 10.94	0.784
Gender (male), n (%)	103 (78.0)	114 (83.2)	0.178
Body mass index, kg/m^2	26.89 ± 3.45	27.64 ± 3.99	0.094
Diabetes mellitus, n (%)	50 (37.9)	51 (37.2)	0.506
Hypertension, n (%)	86 (65.2)	79 (57.7)	0.128
Current smoker, n (%)	63 (47.7)	71 (51.8)	0.291
Hyperlipidemia, n (%)	77 (58.3)	58 (42.3)	0.006
History of stroke, n (%)	1 (0.8)	5 (3.6)	0.116
Chronic kidney disease, n (%)	21 (15.9)	30 (21.9)	0.136
Previous PCI, n (%)	50 (37.9)	29 (21.2)	0.002
History of MI, n (%)	47 (35.6)	34 (24.8)	0.036
Heart failure, n (%)	17 (12.9)	14 (10.2)	0.311
Clinical presentation, n (%)			
CCS	51 (38.6)	64 (46.7)	0.112
NSTEMI	65 (49.2)	53 (38.7)	0.052
USAP	5 (3.8)	18 (13.1)	0.005
LV ejection fraction, %	52.35 ± 10.20	54.87 ± 8.38	0.056
Moderate-severe valve disease, n (%)	20 (15.2)	16 (11.7)	0.256
Laboratory measurements			
White blood cell count, 10^9/L	9.56 ± 2.91	9.29 ± 2.39	0.882
Hemoglobin, g/dL	13.56 ± 1.71	14.04 ± 1.70	0.037
Platelet count, 10^9/L	245.43 ± 69.18	250.98 ± 57.23	0.272
Creatinine, mg/dL	1.06 ± 0.87	0.92 ± 0.27	0.365
Total cholesterol, mg/dL	193.80 ± 57.88	189.15 ± 48.31	0.992
Medications used, n (%)			
Acetylsalicylic acid	132 (100.0)	137 (100.0)	-
Clopidogrel	59 (44.7)	61 (44.5)	0.538
Ticagrelor	62 (47.0)	65 (47.4)	0.518
Prasugrel	11 (8.3)	11 (8.0)	0.552
Beta-blockers	124 (93.9)	127 (92.7)	0.436
CCB	12 (9.1)	20 (14.6)	0.114
ACEi/ARB	115 (87.1)	120 (87.6)	0.527
Statin	125 (94.7)	125 (91.2)	0.193
Diuretics	25 (18.9)	14 (10.2)	0.031
Insulin	40 (30.3)	38 (27.7)	0.371

Table 2. Lesions characteristics per study group

Variables	Mini-crush (n=132)	DK-crush (n=137)	p
SYNTAX score	23 (20-30)	23 (19-28)	0.631
≤22, n (%)	63 (47.7)	50 (36.5)	0.041
23-32, n (%)	41 (31.1)	57 (41.6)	0.047
≥33, n (%)	29 (22.0)	30 (21.9)	0.553
Multi-vessel disease, n (%)	95 (72.0)	97 (70.8)	0.470
Locations of bifurcation lesions, n (%)			0.089
LMCA	6 (4.5)	13 (9.5)	0.408
LAD-Diagonal	82 (62.1)	88 (64.2)	0.128
LCx-OM	43 (32.6)	35 (25.5)	0.742
PDA-PL	1 (0.8)	1 (0.7)	
Type of Medina classification, n (%)			
0.1.1	34 (25.8)	28 (20.4)	0.187
1.1.1	97 (73.5)	109 (79.6)	0.151
Reference vessel diameter, mm			
MV	2.92 ± 0.21	3.08 ± 0.40	0.001
SB	2.59 ± 0.16	2.61 ± 0.20	0.482
SB reference vessel diameter ≥2.5 mm, n (%)	128 (97.0)	127 (92.7)	0.096
Assessment of complex bifurcation lesions, n (%)			
SB diameter stenosis ≥70% or 90%	127 (96.2)	121 (88.3)	0.013
Moderate-to-severe calcification	50 (37.9)	51 (37.2)	0.506
Multiple lesions	95 (72.0)	97 (70.8)	0.470
Bifurcation angle <450 or >700	81 (61.4)	74 (54.0)	0.137
MV reference diameter <2.5 mm	1 (0.8)	4 (2.9)	0.197
Thrombus identified by angiography	19 (14.4)	18 (13.1)	0.451
Lesion length, mm			
MV	22.47 ± 6.54	24.50 ± 6.38	0.024
SB	14.56 ± 4.58	12.98 ± 4.48	<0.001
SB lesion length ≥10 mm, n (%)	126 (95.5)	110 (80.3)	<0.001
MV, n (%)			
TIMI flow grade <3	16 (12.1)	20 (14.6)	0.339
Chronic total occlusion	5 (3.8)	4 (2.9)	0.477
Thrombus-containing lesion	9 (6.8)	11 (8.0)	0.443
SB, n (%)			
TIMI flow grade <3	27 (20.5)	17 (12.4)	0.053
Chronic total occlusion	1 (0.8)	2 (1.5)	0.514
Thrombus-containing lesion	14 (10.6)	7 (5.1)	0.073

LAD: Left anterior descending, LCx: Left circumflex, LMCA: Left main coronary artery, MV: Main vessel, OM: Obtuse marginal artery, PDA: Posterior descending artery, PL: Posterolateral artery, SB: Side branch, TIMI: Thrombolysis in myocardial infarction.

Table 3. Procedural characteristics and in-hospital complications of the study population.

Variables	Mini-crush (n=132)	DK-crush (n=137)	p
Access site, n (%)			
Femoral	126 (95.5)	131 (95.6)	0.590
Radial	6 (4.5)	6 (4.4)	0.590
Tirofiban use during PCI, n (%)	23 (17.4)	26 (19.0)	0.432
Performed IVUS, n (%)	7 (5.3)	16 (11.7)	0.048
Thrombus Aspiration, n (%)	1 (0.8)	2 (1.5)	0.514
Pre-dilation			
MV	78 (59.1)	84 (61.3)	0.402
SB	117 (88.6)	120 (87.6)	0.470
Kissing balloon inflation			
1st	129 (97.7)	137 (100.0)	0.117
2nd	-	135 (98.5)	-
MV Stent number, n	1.20 ± 0.40	1.26 ± 0.44	0.253
Stent diameter, mm	2.97 ± 0.28	3.03 ± 0.33	0.096
Stent length, mm	28.75 ± 7.62	30.94 ± 7.94	0.031
SB Stent number, n	1.06 ± 0.24	1.01 ± 0.12	0.047
Stent diameter, mm	2.61 ± 0.21	2.59 ± 0.22	0.789
Stent length, mm	19.64 ± 5.52	19.64 ± 5.38	0.657
Proximal side-branch optimization, n (%)	115 (87.1)	120 (87.6)	0.527
Final POT, n (%)	127 (96.2)	131 (95.6)	0.610
Resource utilization, n	1.12 ± 0.33	1.09 ± 0.34	0.286
Guiding catheter	2.86 ± 0.74	3.55 ± 0.83	<0.001
Guidewire Balloon	4.42 ± 0.87	6.31 ± 1.80	<0.001
Intraprocedural complication, n (%) Abrupt occlusion			
MV	1 (0.8)	4 (2.9	0.197
SB	7 (5.3)	3 (2.2)	0.152
Intraprocedural complication, n (%) TIMI <3			
MV	6 (4.5)	4 (2.9)	0.352
SB	5 (3.8)	6 (4.4)	0.526
Intraprocedural complication, n (%) Dissection			
MV	4 (3.0)	7 (5.1)	0.292
SB	4 (3.0)	1 (0.7)	0.174
Intraprocedural complication, n (%) Thrombus formation			
MV	3 (2.3)	4 (2.9)	0.521
SB	5 (3.8)	3 (2.2)	0.341
Procedure time, min	69.61 ± 18.97	80.42 ± 27.95	<0.001
Fluoroscopy time, min	16.66 ± 4.19	21.55 ± 7.05	<0.001
Contrast media volume, mL	212.42 ± 63.27	222.26 ± 65.57	0.292
Angiographic success, n (%)			
MV	126 (95.5)	131 (95.6)	0.590
SB	127 (96.2)	134 (97.8)	0.341
In-hospital complications, n (%)			0.679
Death	3 (2.3)	2 (1.5)	0.197
TIMI-major bleeding	1 (0.8)	4 (2.9)	0.640
Pseudoaneurysm	3 (2.3)	3 (2.2)	0.341
Fatal ventricular arrhythmias	5 (3.8)	3 (2.2)	0.491
Stent thrombosis	1 (0.8)	0	0.240
Myocardial infarction	2 (1.5)	0	0.311
Contrast-induced AKI	14 (10.6)	17 (12.9)	

AKI: Acute kidney injury, IVUS: Intravascular ultrasound, PCI: Percutaneous coronary intervention, MV: Main vessel, POT: Proximal optimization technique, SB: Side branch, TIMI: Thrombolysis in myocardial infarction.

Table 4. Clinical outcomes per study group

Variables	Mini-crush (n=132)	DK-crush (n=137)	p
Follow-up time, month	31.41 ± 10.91	30.71 ± 10.86	0.816
Primary end-point (TLF), n (%)	18 (13.6)	17 (12.4)	0.453
Cardiac death	9 (6.8)	8 (5.8)	0.468
Myocardial infarction	9 (6.8)	8 (5.8)	0.468
Target lesion revascularization	7 (5.3)	9 (6.6)	0.429
Secondary end-point, n (%)	19 (14.4)	18 (13.1)	0.451
All-cause death	11 (8.3)	10 (7.3)	0.464
Myocardial infarction	9 (6.8)	8 (5.8)	0.468
Target lesion revascularization	7 (5.3)	9 (6.6)	0.429
Stent thrombosis	4 (3)	3 (2.1)	0.567

AKI: Acute kidney injury, DK: Double kissing, TLF: Target lesion failure, TIMI: Thrombolysis in myocardial infarction.

Interventional Cardiology / Coronary

OP-018

Long-term evaluation of revascularization strategies for medina 0.1.0 left main bifurcation lesions: The LM-CROSSOVER registry

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Background and Aim: The current study aimed to compare long-term clinical outcomes of patients with medina 0.1.0 LM bifurcation lesions treated by crossover stenting (COS) versus accurate ostial stenting (AOS).

Methods: A total of 229 consecutive eligible patients with medina 0.1.0 LM bifurcation lesions were enrolled, and were stratified according to the stenting techniques. The primary end-point was major cardiovascular and cerebral events (MACCE), defined as the combination of all-cause death, target vessel releated-miyocardial infarction (MI), clinically driven target lesion revascularization (TLR), stroke, and stent thrombosis.

Results: The COS and AOS were applied to 78 (34%) and 151 (66%) patients, respectively. During a mean of 40.56 ± 21.1 months of follow-up, the rate of composite MACCE (27.8 vs. 12.8%; p=0.007) was higher in medina 0.1.0 LM bifurcation patients treated with AOS than those treated with COS technique, mainly driven by more frequent all-caused death (13.9 vs. 3.8%, p=0.013) and TLR (6.4 vs. 15.9%; p=0.029). In the multivariable Cox regression analysis, the ostial stenting strategy was one of the independent predictors of MACCE (OR: 2.166; 95% CI, 1.080-4.340; p=0.029).

Conclusions: The current data suggests that COS was associated with a better long-term MACCE and lower mortality rates compared with AOS in patients with medina 0.1.0 LM bifurcation disease.

Table 1. Univariate and multivariate Cox Regression analysis showing independent predictors of MACCE in patients with ostial LAD stenosis

	Univariate Level		Multivariate Level	
	OR (95% CI)	p value	OR (95% CI)	P value
Accurate Ostial Stenting	2.304 (1.155-4.595)	0.018	2.166 (1.080-4.340)	**0.029**
Percentage of Ostial LAD Stenosis	1.020 (0.994-1.048)	0.135	1.030 (1.002-1.058)	**0.035**
Thrombus Identifed by Angiography	1.995 (1.148-3.468)	0.014		
LMCA Stenosis <20%	0.384 (0.138-1.067)	0.066		
Performed IVUS	0.115 (0.016-0.836)	0.033	0.122 (0.016-0.897)	**0.039**
Thrombus Aspiration	3.286 (1.286-8.398)	0.013		
Maximum post-dilatation balloon diameter	0.476 (0.257-0.883)	0.019		
Jailed semiinflated technique	2.161 (0.518-9.028)	0.291		
Tirofiban use during PCI	2.788 (1.185-6.557)	0.019		

Abbreviations: CI: Confidence Interval; IVUS: Intravascular ultrasound, LAD: Left anterior descending artery, LMCA: Left main coronary artery, MACCE: Major cardiovascular and cerebral events; PCI: Percutaneous coronary intervention, OR: Odds ratio

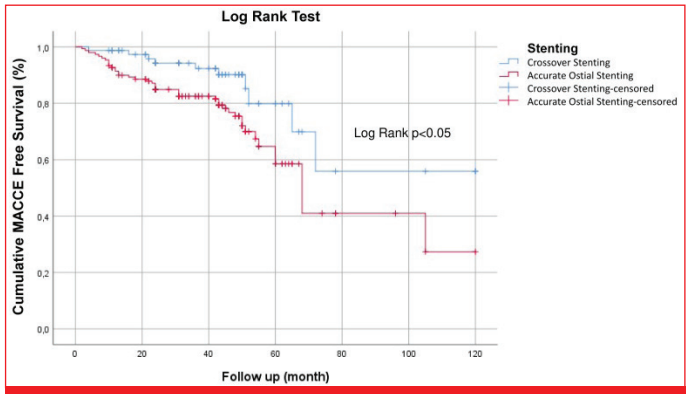

Figure 1. Kaplan-Meier survival analysis revealed that long-term MACCE free survival was found to be significantly decreased in accurate ostial stenting group.

Table 2. Baseline demographic and clinical characteristics of the study patients

Variables	Crossover stenting (n=78)	Accurate ostial stenting (n=151)	p
Age, years	53.21 ± 15.55	57.23 ± 13.00	0.072
Gender (male), n (%)	57 (73.1)	125 (82.8)	0.062
Body mass index, kg/m^2	26.90 ± 3.02	26.57 ± 2.57	0.503
Diabetes mellitus, n (%)	33 (42.3)	72 (47.7)	0.263
Hypertension, n (%)	56 (71.8)	93 (61.6)	0.082
Current smoker, n (%)	33 (42.3)	65 (43.0)	0.514
Hyperlipidemia, n (%)	48 (61.5)	100 (66.2)	0.288
History of stroke, n (%)	1 (1.3)	5 (3.3)	0.334
Chronic kidney disease, n (%)	13 (16.7)	29 (19.2)	0.390
Previous PCI, n (%)	21 (26.9)	45 (29.8)	0.384
Multi-vessel disease, n (%)	32 (41.0)	70 (46.4)	0.265
Clinical presentation, n (%)			
CCS	43 (55.1)	99 (65.6)	0.081
NSTEMI	25 (32.1)	46 (30.5)	0.460
USAP	9 (11.5)	8 (5.3)	0.077
LV ejection fraction, %	47.06 ± 10.52	44.65 ± 11.29	0.092
Moderate-severe valve disease, n (%)	5 (6.4)	13 (8.6)	0.381
Medications used, n (%)			
Antiplatelet agents	78 (100.0)	151 (100.0)	>0.05
Beta-blockers	70 (89.7)	133 (88.1)	0.445
CCB	11 (14.1)	12 (7.9)	0.109
ACEi/ARB	71 (91.0)	126 (83.4)	0.083
Statin	73 (93.6)	133 (88.1)	0.138
Diuretics	25 (32.1)	47 (31.1)	0.500
Nitrate	6 (7.7)	15 (9.9)	0.384
Insulin	12 (15.4)	36 (23.8)	0.092

Table 3. Angiographic and procedural characteristics of the study population

Variables	Crossover stenting	Accurate ostial stenting	p
Access site, n (%)			
Femoral	76 (97.4)	148 (98.0)	0.555
Radial	2 (2.6)	3 (2.0)	0.555
LMCA diameter, mm	4.54 ± 0.40	4.54 ± 0.43	0.077
Ostial LAD diameter, mm	4.17 ± 3.90	3.40 ± 0.30	<0.001
Ostial LCx diameter, mm	3.28 ± 0.44	2.99 ± 0.33	<0.001
Percentage of ostial LAD stenosis, %	91.54 ± 10.75	88.60 ± 12.01	0.122
Thrombus identifed by angiography, n (%)	32 (41.0)	67 (44.4)	0.366
LMCA stenosis <30%, n (%)	16 (20.5)	27 (17.9)	0.376
Non-target lesion intervention, n (%)	24 (30.8)	41 (27.2)	0.335
Intra-aortic balloon pump support, n (%)	6 (7.7)	3 (2.0)	0.044
Performed IVUS, n (%)	14 (17.9)	13 (8.6)	0.034
Rotablator use, n (%)	2 (2.6)	3 (2.0)	0.555
Thrombus Aspiration, n (%)	1 (1.3)	7 (4.6)	0.179
Direct stenting, n (%)	27 (34.6)	40 (26.5)	0.130
Stent diameter, mm	3.57 ± 0.32	3.11 ± 0.23	<0.001
Total stent length, mm	22.95 ± 4.52	23.19 ± 6.76	0.514
Maximum post-dilatation balloon diameter, mm	4.29 ± 0.37	3.43 ± 0.30	<0.001
Procedure time, min	39.29 ± 10.23	37.85 ± 12.87	0.117
Fluoroscopy time, min	13.48 ± 4.09	13.13 ± 5.77	0.457
LCx narrowing >50%, n (%)	13 (16.7)	15 (9.9)	0.105
Side branch intervention, n (%)			
Kissing balloon inflation	9 (11.5)	11 (7.3)	0.201
POT-side-POT	3 (3.8)	0	0.039
Jailed semi-inflated balloon technique	3 (3.8)	0	0.039
LCx stenting	5 (6.4)	8 (5.3)	0.471
Tirofiban use during PCI, n (%)	4 (5.1)	9 (6.0)	0.529
Contrast media volume, mL	126.19 ± 54.40	124.17 ± 64.20	0.497

IVUS: Intravascular ultrasound, LAD: Left anterior descending, LCx: Left circumflex, LMCA: Left main coronary artery, PCI: Percutaneous coronary intervention, POT: Proximal optimization technique.

Table 4. Clinical outcomes of study patients

Variables	Crossover stenting	Accurate ostial stenting	p
Follow-up time, month	41.91 ± 22.16	39.86 ± 20.59	0.621
Primary end-point (MACCE), n (%)	10 (12.8)	42 (27.8)	0.007
All-cause death	3 (3.8)	21 (13.9)	0.013
Myocardial infarction	4 (5.1)	13 (8.6)	0.251
Target lesion revascularization	5 (6.4)	24 (15.9)	0.029
Stent thrombosis	1 (1.3)	4 (2.6)	0.445
Stroke	1 (1.3)	3 (2.0)	0.580
Secondary end-point, n (%)	13 (16.7)	23 (15.2)	0.458
TIMI-major bleeding	2 (2.6)	3 (2.0)	0.555
Fatal ventricular arrhythmias	3 (3.8)	8 (5.3)	0.449
Contrast-induced AKI	8 (10.3)	18 (11.9)	0.445

AKI: Acute kidney injury, MACCE: Major cardiovascular and cerebral events, TIMI: Thrombolysis in myocardial infarction.

Epidemiology

OP-019

Evaluation of cardiovascular risk factors, prevalence and determinants of coronary artery disease in renal transplant patients: A single center experience

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Background and Aim: Cardiovascular (CV) disease is the leading cause of morbidity and mortality in renal transplant patients. In our study, we aimed to determine the CV risk factors, the prevalence and determinants of coronary artery disease (CAD) in patients who underwent kidney transplantation in our center.

Methods: One hundred sixty nine patients who underwent kidney transplantation in our center were included in the study retrospectively. Demographic and clinical characteristics of the patients, cardiac evaluation findings and further examination results were scanned from the database of our center.

Results: The mean age of the patients was 42.86 ± 12.97 years and 43.19% were female. The most common etiological factors for the development of end-stage renal disease were hypertension (HT) and diabetes mellitus (DM). Ninety seven patients (57.4%) were undergoing dialysis, 4 of whom were on peritoneal dialysis. Renal transplant was performed from a cadaver in two patients and from a living donor in the other patients. CAD was detected in 29 patients (17.15%). The most prevalent CV risk factors were HT and hyperlipidemia (HL). Multivariate logistic regression analysis revealed that age, DM, HL and dialysis history were independent risk factors for the development of CAD. In the postoperative follow-ups, no death or acute coronary syndrome was observed during the hospitalization period.

Conclusions: Prevalence of CV risk factors is high in renal transplant candidates. Our findings support the need for a detailed cardiac evaluation and effective management of CV risk factors in patients preparing for kidney transplantation.

Table 1. Etiologies of end stage renal disease in kidney recipients

Etiology	
Hypertension n (%)	21 (12.42)
Diabetes mellitus type 1 n (%)	4 (2.36)
Diabetes mellitus type 2 n (%)	20 (11.83)
Polycystic kidney disease n (%)	14 (8.28)
Idiopathic n (%)	21 (12.42)
Ig A nephropathy n (%)	15 (8.87)
Focal segmental glomerulosclerosis n (%)	14 (8.28)
Glomerulonephritis n (%)	9 (5.32)
Vasculitis n (%)	12 (7.1)
Amyloidosis n (%)	6 (3.55)
Nephrolithiasis n (%)	6 (3.55)
Others n (%)	27 (15.97)

Table 2. Demographic and clinical characteristics of the study group

Parameter	Study group (n=169)
Age	42.86±12.97
Female, % (n)	43.19 (73)
BMI, (kg/m^2)	25.67±5.57
Hypertension, % (n)	83.43 (141)
Hyperlipidemia, % (n)	35.50 (60)
Diabetes mellitus, % (n)	14.20 (24)
Smoking, % (n)	30.76 (52)
CAD, % (n)	17.15 (29)
CHF, % (n)	6.50 (11)
Antiaggregant therapy, % (n)	17.75 (30)
ACEI /ARB, % (n)	15.38 (26)
Beta blockers, % (n)	46.15 (78)
CCBs, % (n)	60.94 (103)
Statin, % (n)	15.38 (26)
Dialysis, % (n)	57.4 (97)

Table 3. Laboratory and echocardiographic characteristics of the patients

Parameter	Study group (n=169)
Glucose mg/dl	117.97±59.01
BUN mg/dl	66.75±25.46
Creatinine mg/dl	7.30± 2.62
eGFR (ml/min/1.73m2)	8.49±3.95
Sodium (mmol/L)	139.07±4.13
Potassium (mmol/L)	4.96±0.75
Total cholesterol (mg/dl)	195.90±51.14
LDL (mg/dl)	128.37±46.78
HDL (mg/dl)	45.93±17.06
Triglycerides (mg/dl)	171.08±114.19
LV EF (%)	58.22±6.84
sPAP (mmHg)	28.34±7.17
LVH % (n)	40.2 (68)

Table 4. Findings of preoperative non-invasive and invasive tests

Parameter	Study group (n=169)
Treadmill exercise test, (n)	130
Ischemia negative	82
Ischemia positive	28
Non-diagnostic	20
MPS, (n)	19
Ischemia negative, (n)	14
Ischemia positive, (n)	5
Coronary CTA, (n)	21
Non-obstructive CAD, (n)	2
Obstructive CAD, (n)	4
Coronary angiography, (n)	61
Normal coronary arteries, (n)	32
Medical treatment, (n)	12
PCI, (n)	10
CABG, (n)	5
Patients with previous CABG, (n)	2
Coronary artery disease, % (n)	17.15 (29)

Table 5. Comparisons of patients according to presence of coronary artery disease

Parameter	Patients with CAD (n=29)	Patient without CAD (n=140)	p value
Age	52±11	41±12.55	<0.001
Male, % (n)	87 (20)	52.1 (76)	0.002
BMI, (kg/m^2)	27.44±4.62	25.30±5.69	0.060
Hypertension, % (n)	85.1 (23)	80 (118)	0.021
Hyperlipidemia, % (n)	65.2 (15)	12.3 (18)	<0.001
Diabetes mellitus, % (n)	43.5 (10)	9.6 (14)	<0.001
Smoking, % (n)	56.5 (13)	26.7 (39)	0.004
Dialysis, % (n)	81.4 (22)	52.8 (75)	0.009
eGFR, (ml/min/1.73m^2)	7±3.75	8.7±3.93	0.128
Creatinine, mg/dl	8.6±3.39	7.1±2.37	0.025
LV EF, %	55±8.77	59±6.35	<0.001
sPAP, mmHg	29.3±8.37	28.2±6.94	0.436
LVH, % (n)	62.1 (18)	35.7 (50)	0.008

Table 6. Logistic regression analyses for the presence of coronary artery disease

	Univariate analysis		
	p value	OR	95% Confidence interval
Age	<0.001	1.076	1.037-1.118
HT	0.068	0.149	0.019-1.148
DM	<0.001	0.064	0.024-0.173
HL	<0.001	0.073	0.029-0.184
Smoking	0.075	0.475	0.239-1.079
Creatinine	0.052	1.155	0.999-1.326
GFR	0.103	0.897	0.787-1.022
Dialysis	0.004	0.227	0.082-0.629
LV EF	0.015	0.941	0.896-0.988
sPAP	0.296	1.534	0.897-2.623
LVH	0.010	1.028	1.976-1.083
	Multivariate analysis		
Age	0.007	1.065	1.018-1.115
DM	0.001	0.122	0.034-0.433
HL	0.001	0.127	0.039-0.410
Dialysis	0.044	0.245	0.062-0.962
LV EF	0.260	0.953	0.877-1.036
LVH	0.448	1.572	0.489-5.049

Other

OP-020

Comparison of vibration and flow mediated vasodilator responses in diabetic patients

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Background and Aim: Growing frequency and life expectancy of the diabetic patients' population in all over the world and in our country increases the share of diabetic vascular complications in cardiology practice. As current treatment and prevention methods are less effective in this patient group, there is a need to for new treatment methods in that area. Exercise, which reduces metabolic and vascular problems associated with diabetes, often becomes impossible especially in advanced stage patients who need treatment the most. Since exercise and flow mediated dilation (FMD) are effective by stimulating mechanotransduction mechanisms on the endothelium, it can be expected that the same mechanisms can also be stimulated by direct vibration.

Methods: In order to test this hypothesis, in the presented study, a group of 20 type-2 diabetic patients (11 males, age 56.80 ± 11.05 and diagnosed for 15.35 ± 8.61 years) were examined via application of a duly FMD and VMD (by performing vibration for 5 minutes with 20 Hz frequency and 3 mm vertical amplitude) to the same side forearm, with a 30 minutes interval. Using a 10 MHz linear echo probe, brachial artery diameter and flow velocities were recorded before and later of FMD and VMD applications at 2-minute intervals for 10 minutes. Then brachial artery flow and resistances has been calculated for each stage.

Results: In the first minute after FMD and VMD applications, BA diameter and flow velocities increased evidently and significantly and vascular resistance decreased in a similar way. None of the corresponding FMD or VMD parameters in the first minute was different. The artery diameters after FMD and VMD, in the first minute, were increased by 6.04 ± 5.29 and 5.49 ± 5.21% respectively. At the tenth minute, these figures decreased to 1.73 ± 3.21 and 2.05 ± 3.31%. In the FMD series, all parameters except artery diameter returned to their baseline values after the fourth minute. After VMD, all parameters also tended to approach their baseline values after the first minute, but the recovery was much slower. At each stage after the first minute, the VMD averages were higher than the baseline value and their corresponding FMD values.

Conclusions: The results of the study indicate that vibration may be a powerful, long- lasting and feasible treatment option in patients with peripheral perfusion failure developed due to diabetic macro and microvascular complications.

Other

OP-021

Cardiac power index is a novel parameter for prediction of myocardial injury during liver transplantation

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Background and Aim: Despite comprehensive preoperative cardiovascular assessment, cardiovascular events remain a leading cause of postoperative mortality, due to the complexity of liver transplantation surgery. Therefore hemodynamic monitoring during liver transplantation is crucial. With Pulse Index Continuous Cardiac Output (PICCO) technology, hemodynamic parameters such as cardiac power index (CPI) can be follwed during surgery. The aim of this study is to investigate the relationship between CPI and postoperative secondary myocardial infarction.

Methods: A total of 53 patients were included in the study. Postoperative myocardial injury (PMI) was observed in 28.3% (n=15) of these patients. Patients divided into two groups according to the presence or absence of myocardial injury following liver transplantation.

Results: ΔCPI was significantly lower in patients with PMI (-0.27 ± 0.11 W/m^2) than those without PMI (0.08 ± 0.18 W/m^2) (p<0.05). The multivariate analysis showed that the only independent predictor of PMI was ΔCPI (HR: 2.245, 95% CI: 1.145-4.387, p=0.032) (Table 1). ROC analysis that revealed ΔCPI values lower than -0.15 W/m^2 were significantly associated with PMI. Peak troponin level, hospital stay and myocardial infaction prevelance were significantly higher in ΔCPI ≤ -0.15 W/m^2 group (p<0.05 for all).

Conclusions: Our data shows that ΔCPI which constitutes the decrease in CPI during transition from the anhepatic phase to the neohepatic phase, can be used as a marker of poor cardiac prognosis in patients who underwent liver transplantation.

Table 1. Univariate and multivariate logistic regression analyses to determine ındependent predictors of myocardial injury following liver transplantation

Variable	Univariate Analysis		
	Odds Ratio	95% Confidence Interval	p value
Age	2.071	0.805 – 5.313	0.046
LVEF	0.970	0.946 – 0.995	0.135
BNP level	1.040	1.223 – 1.064	0.442
ΔCPI	2.326	1.511 – 3.716	0,012
Operation Time	2.651	1.104 – 6.317	< 0.001
Vasopressor Need	1.916	1.225 – 2.293	0.081
	Multivariate Analysis		
	Odds Ratio	95% Confidence Interval	p value
Age	1.242	1.012 – 1.472	0.121
ΔCPI	2.245	1.145 – 4.387	0.032
Operation Time	2.180	1.349 – 3.511	0.008

LVEF: Left Ventricular Ejection Fraction, BNP: Brain Natriuretic Peptide, CPI: Cardiac Power Index

Other

OP-022

A comprehensive evaluation of national cardiology congresses abstracts on the basis of the 2016 academic criteria

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Background and Aim: This study aimed to describe the effects of new academic criteria established in 2016 on the abstracts of the National Congress of the Turkish Society of Cardiology.

Methods: The abstracts presented at 13 consecutive annual congresses were obtained. A literature search was conducted with PubMed, Google Scholar, and Web of Science databases to analyze whether the abstract was published in a scientific journal. The study was divided into 2 time groups according to the new academic criteria published in 2016. Group 1 included 4828 abstracts accepted at National Congress of the Turkish Society of Cardiology between 2009 and 2016, while Group 2 included 2284 abstracts accepted at National Congress of the Turkish Society of Cardiology between 2017 and 2021.

Results: A total of 7112 abstracts were accepted for the 2009-2021 National Congress of the Turkish Society of Cardiology meetings scientific program. The publication rate (43.2% vs. 23.9%, p<0.001), number of authors [7 (5-9) vs. 4 (3-6), p<0.001], and rate of original investigation (72.3 vs. 56.5%, p<0.001) were significantly lower in group 2 than in group 1. Among the quality parameters of the journals in which the abstracts were published, the impact factor (0.59 ± 1.71 vs. 0.26 ± 1.09, p<0.001), the rate of journals in science citation index or science citation index-expanded indexes (70.4 vs. 57.9%, p<0.001), and the rate of the second or third-quartile class (24.2 vs. 16.1%, p<0.001) were significantly lower in group 2 as compared to group 1. Being in group 1, oral presentation, original investigation, and cardiac imaging were identified as independent predictors for publication in scientific journals.

Conclusions: This study showed that the 2016 new academic criteria negatively affected the publication processes of accepted abstracts in National Congress of the Turkish Society of Cardiology.

Table 1. Baseline findings of the abstracts presented at the Turkish Cardiology National Congress

Parameters	All abstracts (n=7112)	Group 1 (2009-2016) (n=4828)	Group 2 (2017-2021) (n=2284)	p
Presentation type, n (%)				
Oral	2811 (39.5)	1671 (34.6)	1140 (49.9)	<0.001
Poster	4301 (60.5)	3157 (65.4)	1144 (50.1)	
Number of authors	6 (4-8)	7 (5-9)	4 (3-6)	<0.001
Abstract type, n (%)				
Case	2330 (32.8)	1336 (27.7)	994 (43.5)	<0.001
Original investigation	4782 (67.2)	3492 (72.3)	1290 (56.5)	
First author gender, male, n (%)	5869 (82.5)	4055 (84.0)	1814 (79.4)	<0.001
Institution of the authors, n (%)				
University hospital	4769 (67.1)	3388 (70.2)	1381 (60.5)	<0.001
Training and research hospital	3098 (43.6)	2150 (44.5)	948 (41.5)	0.016
State hospital	1281 (18.0)	978 (20.3)	303 (13.3)	<0.001
Private hospital	644 (9.1)	456 (9.4)	188 (8.2)	0.096
Multicenter	1783 (25.1)	1434 (29.7)	349 (15.3)	<0.001
Disciplines, n (%)				
Cardiology	6919 (97.3)	4666 (96.6)	2253 (98.6)	<0.001
Cardiovascular surgery	430 (6.0)	342 (7.1)	88 (3.9)	<0.001
Chest diseases	104 (1.5)	64 (1.3)	40 (1.8)	0.163
Internal medicine	271 (3.8)	200 (4.1)	71 (3.1)	0.033
Nephrology	55 (0.8)	33 (0.7)	22 (1.0)	0.209
Anesthesia	40 (0.6)	32 (0.7)	8 (0.4)	0.100
Pediatric cardiology	46 (0.6)	32 (0.7)	14 (0.6)	0.807
Nuclear medicine	22 (0.3)	18 (0.4)	4 (0.2)	0.161
Public health	46 (0.6)	34 (0.7)	12 (0.5)	0.380
Genetic	64 (0.9)	45 (0.9)	19 (0.8)	0.676
Radiology	182 (2.6)	108 (2.2)	74 (3.2)	0.012
Pathology	24 (0.3)	10 (0.2)	14 (0.6)	0.006
Other	375 (5.3)	297 (6.2)	78 (3.4)	<0.001
Biochemistry	245 (3.4)	181 (3.7)	64 (2.8)	0.041
Country of origin of abstract, n (%)				
Türkiye	6845 (96.2)	4604 (95.4)	2241 (98.1)	<0.001
European Union	108 (1.5)	93 (1.9)	15 (0.7)	<0.001
United State of America	19 (0.3)	8 (0.1)	11 (0.5)	0.016
Turkish Republic of Northern Cyprus	31 (0.4)	5 (0.1)	26 (1.1)	<0.001
Middle East	54 (0.8)	47 (1.0)	7 (0.3)	0.002
Turkic Republics	21 (0.3)	16 (0.3)	5 (0.2)	0.414
Other	59 (0.8)	56 (1.2)	3 (0.1)	<0.001
Cardiovascular medicine field, n (%)				
Structural and congenital heart disease	394 (5.5)	20 (4.6)	174 (7.6)	<0.001
Coronary artery disease	1996 (28.1)	1356 (28.1)	640 (28.0)	0.954
Arrhythmia	868 (12.2)	588 (12.2)	280 (12.3)	0.923
Peripheral artery disease	334 (4.7)	162 (3.4)	172 (7.5)	<0.001
Hypertension	249 (3.5)	186 (3.9)	63 (2.8)	0.019
Heart valve disease	565 (7.9)	404 (8.4)	161 (7.0)	0.055
Heart failure	462 (6.5)	306 (6.3)	156 (6.8)	0.432
Cardiac imaging	1084 (15.2)	731 (15.1)	353 (15.5)	0.730
Pulmonary vascular disease	217 (3.1)	105 (2.2)	1123 (4.9)	<0.001
Lipidemia	167 (2.3)	93 (1.9)	74 (3.2)	0.001
Epidemiology	201 (2.8)	150 (3.1)	51 (2.2)	0.038
Other	848 (11.9)	617 (12.8)	231 (10.1)	0.001
Original investigation type, n (%)				
Meta-analysis	8 (0.1)	1 (0.0)	7 (0.3)	0.002
Retrospective	798 (11.2)	451 (9.3)	347 (15.2)	<0.001
Prospective	1081 (15.2)	772 (16.0)	309 (13.5)	0.007
Case-control	2000 (28.1)	1651 (34.2)	349 (15.3)	<0.001
Cross-sectional	760 (10.7)	517 (10.7)	243 (10.6)	0.930
Questionnaire	56 (0.8)	38 (0.8)	18 (0.8)	0.996
Publication rate, %	2633 (37.0)	2087 (43.2)	546 (23.9)	<0.001

Table 2. Comparison of the publication success of the abstracts presented at the Turkish Cardiology National Congress per study groups

Parameters	Group 1 (2009-2016) (n=2087)	Group 2 (2017-2021) (n=546)	p
Publication time, month	6.1 ± 13.2	1.4 ± 3.8	<0.001
Publication language, n (%)			
Turkish	57 (2.7)	28 (5.1)	0.005
English	2030 (97.3)	518 (94.9)	
Academic journal, n (%)			
National	649 (31.1)	213 (39.0)	<0.001
International	1438 (68.9)	333 (61.0)	
Journal Index, n (%)			
SCI/SCIE	1469 (70.4)	316 (57.9)	<0.001
ESCI	268 (12.8)	67 (12.3)	0.722
IPRJ	146 (7.0)	59 (10.8)	0.003
ULAKBIM	186 (7.1)	84 (15.4)	<0.001
Impact factor of the journal	0.593 ± 1.719	0.269 ± 1.099	<0.001
Quartiles class of the journal, n (%)			
Q1	84 (4.0)	13 (2.4)	0.069
Q2/3	505 (24.2)	88 (16.1)	<0.001
Q4	867 (41.5)	210 (38.5)	0.192
Changed component, n (%)			
Title	824 (39.5)	154 (28.2)	<0.001
Number of authors	1116 (53.5)	248 (45.4)	0.001
First author	333 (16.0)	72 (13.2)	0.110
Order of authorship	1238 (59.3)	230 (42.1)	<0.001
Author removal	797 (38.2)	117 (21.4)	<0.001
Author adding	680 (32.6)	196 (35.9)	0.143
Quantitative results	414 (19.8)	64 (11.7)	<0.001
Conclusion	17 (0.8)	18 (3.3)	<0.001

Table 3. Regression analysis of the abstract potential predictors for publication in an academic journal

Parameters	Odds ratio	95% CI (lower-upper)	p
Oral presentation	1.212	1.092-1.346	<0.001
Original investigation	2.022	1.795-2.278	<0.001
Being in group 1	2.199	1.946-2.484	<0.001
Multicenter study	1.045	0.931-1.173	0.457
>1 disciplines	1.123	0.997-1.265	0.057
Structural and congenital heart disease	0.989	0.786-1.243	0.923
Peripheral artery disease	0.987	0.763-1.276	0.919
Cardiac imaging	1.317	1.147-1.514	<0.001
Number of authors	1.016	0.999-1.034	0.072

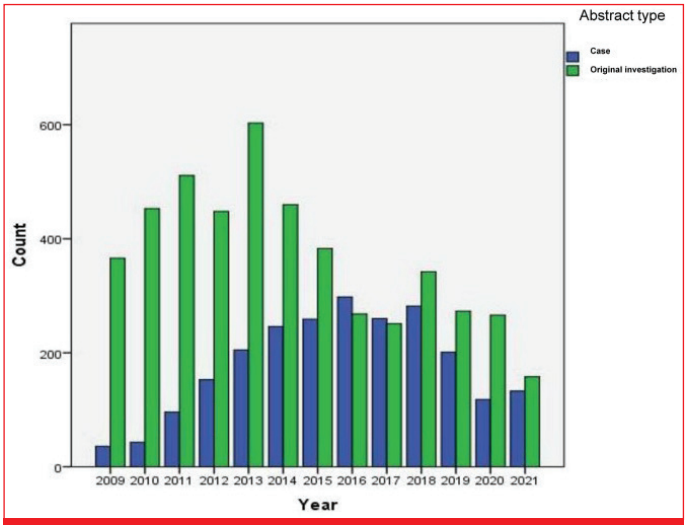

Figure 1. Numerical distribution of the case and original investigation abstracts accepted at the National Congress of the Turkish Society of Cardiology between 2009 and 2021.

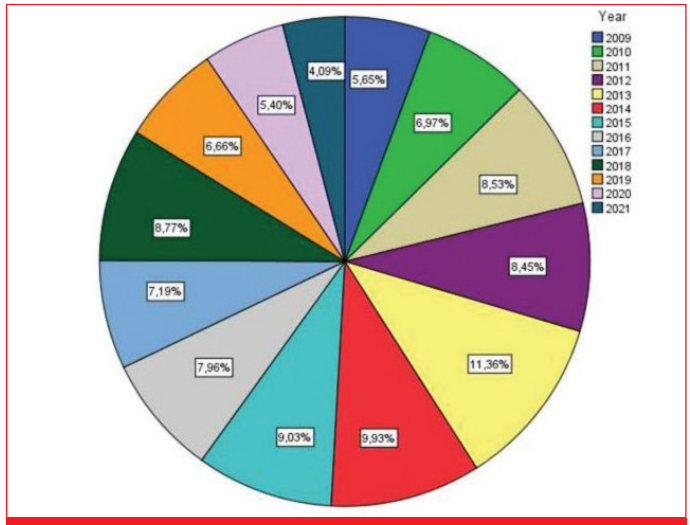

Figure 2. Percentage distribution of a total of 7112 abstracts for each National Congress of the Turkish Society of Cardiology between 2009 and 2021.

Lipid / Preventive Cardiology

OP-024

The relationship between inflammation-oxidative stress and lipoprotein (a) levels in healthy males

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Background and Aim: Atherosclerosis and related diseases are leading causes of death in all around the world. Atherosclerosis is accepted as a chronical inflammatory state highly effected by oxidative stress. High levels of lipoprotein (a) increase likelihood of having atherosclerotic cardiovascular diseases. Monocyte to high-density lipoprotein (HDL) ratio (MHR) indicates inflammation and oxidative stress based on the anti-inflammatory and antioxidant effects of high-density lipoprotein cholesterol (HDL-C), as well as the proinflammatory effect of monocytes. ın this study we analysed the relationship between lipoprotein (a) levels and MHR in otherwise healthy males.

Methods: In this prospective study, patients were selected from the consecutive otherwise healthy males who were admitted to the cardiology outpatient clinic. After exclusion criteria 81 patients included. Demographic, clinical data of included pateints were obtained and biochemical analysis were done. Revealed data statistically analysed.

Results: We divided study population into high risk and low risk group in response to the lipoprotein (a) levels. Lipoprotein (a) <25 formed low risk group. Lymphocyte (2.41 ± 0.54 vs. 2.94 ± 0.67; p=0.008) monocyte (0.48 ± 0.012 vs. 0.57 ± 0.027; p=0.002), LMR (4.14 ± 0.29 vs. 4.6 ± 0.56; p=0.021) and MHR (0.0186 ± 0.003 vs. 0.024 ± 0.0012; p<0.0001) levels were significantly higher in high risk group. Furthermore, lipoprotein (a) levels were not significantly different between smokers and non smokers. When we analysed ll included patients' data, although there were no significant correlations between Lipoprotein (a) and total cholesterol, LDL, HDL and triglyceride levels there were significant correlations between lymphocyte, monocyte, lymphocyte to monocyte ratio (LMR), and monocyte to HDL ratios (MHR) (r=0.688, p<0.0001; r=0.410, p<0.001; r=0.464, p<0.0001; r=0.499, p<0.0001 respectively).

Conclusions: In this prospective study, we showed that inflammation-oxidative stress parameters calculated from the biochemical analysis are significantly higher in high atherosclerotic risk group otherwise healthy males. Besides, there is a significant correlation between lipoprotein (a) levels and inflammation-oxidative stress parameters.

Cardiac Imaging / Echocardiography

OP-025

The effect of body weight on left atrial function determined by longitudinal strain analysis in young adults

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Background and Aim: Obesity is a risk factor for various cardiovascular disorders. Left atrial (LA) function is vital for predicting adverse outcomes in many diseases. LA strain was recently proposed as a noninvasive and valuable parameter for LA functional evaluation. We investigated the effect of body mass index (BMI) values on left atrial functions determined by longitudinal strain analysis in young adults without concomitant disease.

Methods: We prospectively included 134 subjects in our study. Participants were categorized into three subgroups, obese, overweight, and control, according to their BMI. Conventional echocardiographic measurements and strain analysis were performed on all patients.

Results: There were 41 patients (30.5%) in the obesity group, 46 patients (34.3%) in the overweight group, and 47 patients (35.0%) in the control group. Obese patients had significantly larger LA volume (46.9 ± 12.1 mL; p<0.001) compared to overweight and control subjects; however, LA volume index (21.4 ± 6.1 mL/m^2 vs. 22.4 ± 6.1 mL/m^2 vs. 22.4 ± 5.0 mL/m^2; p=0.652) were similar between groups. In the LA strain analysis, obese patients were found to have lower left atrial reservoir longitudinal strain (LASr) compared to both the overweight and control group (44.2 ± 5.8% vs. 39.1 ± 3.7% vs. 36.5 ± 4.9%; p<0.001); moreover obese patients had significantly worse left atrial contraction phase longitudinal strain (LASct) (-15.1 ± 3.1% vs. -13.1 ± 2.5%; p=0.007) and left atrial conduit phase longitudinal strain (LAScd) (-29.0 ± 7.1% vs. -23.3 ± 5.4%; p<0.001) values compared to the control group. However, LASct and LAScd values did not differ between overweight and obese patients.

Conclusions: LA function determined by LA strain analysis was impaired in obese and overweight individuals compared to the control group, even in the early stages of life. The prognostic significance of this finding should be investigated in prospective studies.

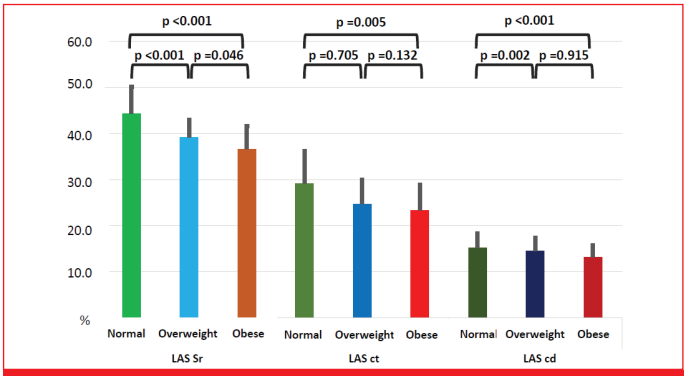

Figure 1. Average left atrial reservoir longitudinal strain (LASr), left atrial conduit phase longitudinal strain (LAScd), left atrial contraction phase longitudinal strain (LASct) with standard deviations among groups.

Lipid / Preventive Cardiology

OP-026

The prevalence of myocardial bridging in coronary CT angiography and its association with atherosclerosis: A retrospective study

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Background and Aim: Myocardial bridging (MB) is a congenital coronary anomaly in which a segment of the epicardial coronary artery courses through the myocardium for a portion of its length. The mechanical forces exerted by MB on the coronary artery can potentially influence its function over time, leading to concerns about the development and progression of atherosclerosis. Thus, this study aims to investigate the occurrence and significance of MB in coronary CT angiography and explore its potential association with atherosclerosis.

Methods: A retrospective analysis was conducted on symptomatic patients who had undergone coronary computed tomography angiography (CCTA) between January 2020 and December 2021 at our institution.

Results: Out of the 852 patients enrolled in the study, 206 (24.2%) were diagnosed with MB. Among these MB patients, the mean age was 56.69 ± 11.68 years, with 124 (60.2%) being male. Notably, 96 (46.6%) of the MB patients exhibited some degree of coronary plaques on CCTA. Of those, 56 (27.2%) had mild plaques, 20 (9.7%) had moderate plaques, and 20 (9.7%) had obstructive plaques. However, statistical analysis did not reveal a significant association between the presence of MB and the occurrence of plaques (p=0.576). Moreover, the assessment of plaque severity and MB showed no significant association (p=0.444). The severity and extent of plaques are illustrated in the figure.

Conclusions: In this study, we found no significant association between MB and the presence of coronary plaques. Although coronary CT angiography is an invaluable tool in clinical practice, it can sometimes incidentally detect various findings, including MB. Our results suggest that MB detected by coronary CT angiography may not be directly related to the presence of coronary artery disease (CAD). To strengthen these findings and gain a deeper understanding of the clinical implications of MB on cardiovascular health, further research and larger-scale studies are warranted. Clinicians should be aware of the potential presence of MB in patients undergoing coronary CT angiography and consider its implications in the context of individualized patient care.

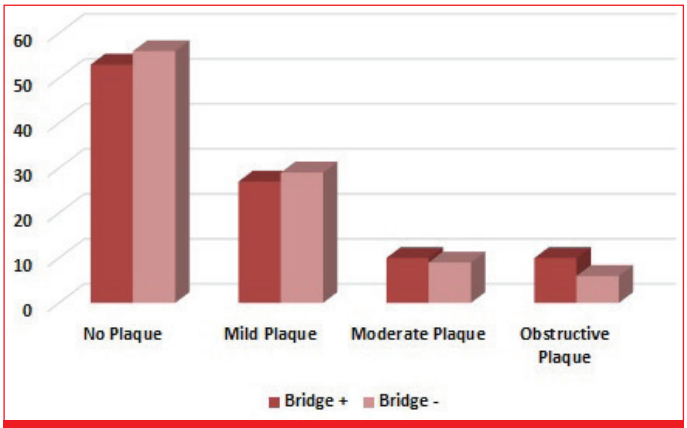

Figure 1. Plaque's characteristic of patients.

Coronary Artery Disease / Acute Coronary Syndrome

OP-027

Patients with acute myocardial infarction with higher platecrit levels have a more unfavorable prognosis than normal patients despite successful intervention to the infarct related artery

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Background and Aim: Platelets with larger volumes are more active and have more intense granule contents, and thus they are a topic of interest in the development of atherosclerosis and pathogenesis of acute coronary syndrome. The main purpose of our study is to investigate the relationship between platecrit levels and 1-month prognosis in patients with acute myocardial infarction who underwent primary percutaneous intervention.

Methods: The study was a cross-sectional study and included 156 patients (114 males, 42 females, mean age 57.0 ± 13.1 years) who applied to the emergency department of our hospital and were diagnosed with acute myocardial infarction and underwent percutaneous coronary intervention. All patients underwent electrocardiogram as well as other laboratory tests. In addition to the complete blood count at the time of admission, the troponin levels of the patients were also recorded. In addition to 300 mg of acetylsalicylic acid, the patients were given 90 mg of loading dose ticagrelor or 600 mg of clopidogrel. All patients underwent coronary percutaneous intervention. According to the hemogram table of our hospital, the normal platecrit value was 0.108-0.282. None of the patients included in the study had a platecrit value below 0.108. Groups were created according to platecrit levels; those with high platelet levels (≥0.282) (group 1; n=63, 41 males, 22 females, mean age: 56.8 ± 14.6 years) and those with normal platecrit levels (<0.282) (group 2; n=93, 73 males, 20 women, mean age: 57.0 ± 12.1 years). The negative endpoint was the death of the patients within 1 month from the date of the procedure, intubation, failure to open the infarct related artery, connection to a respiratory support device, and development of heart failure (EF<40%). It was confirmed from the death registration system whether the patients were dead or not within 1 month.

Results: Comparison of demographic data and laboratory values between groups is shown in Table 1. The rates

of non-ST-elevation MI and ST-elevation MI were similar between the groups (Table 1). In group 1, number of the patients whose infarct related artery could not be opened as a result of the interventionwas 1 and while it was 2 in group 2; p>0.05). Platelet count was significantly higher in group 1 compared to group 2. The exitus rate in group 1 was significantly higher than in group 2 [11 (17.5%) vs. 5 (5.4%); p=0.015)]. The proportion of patients with an adverse endpoint was again significantly higher in group 1 than in group 2 [35 (55.6%) vs. 30 (32.3%)].

Conclusions: Patients with acute myocardial infarction with elevated platecrit levels -a relatively new marker of platelet activation, are associated with a more unfavorable prognosis. Platecrit levels may be an indicator of negative endpoints in these patients despite intervention and opening of the infarct related artery.

Table 1. Comparison of demographic and laboratory parameters of the groups

	Group 1 (n=63)	Group 2 (n=93)	p
Age, years	56.8 ± 14.6	57.0 ± 12.1	0.914
Gender (female), n	22	20	0.065
Hypertension, n	25	40	0.603
Diabetes mellitus, n	24	28	0.288
Smoking, n	39	63	0.535
Hyperlipidemia, n	22	33	0.961
Family history, n	22	29	0.610
Chronic kidney disease, n	5	10	0.560
STEMI, n (%)	51	73	0.710
Troponin T, ng/L	295.7 ± 764.9	348.8 ± 815.2	0.687
Glucose, mg/dL	199.2 ± 107.0	162.2 ± 76.1	0.021
Urea, mg/dL	33.8 ± 10.1	38.2 ± 23.5	0.170
Creatinine, mg/dL	0.91 ± 0.17	1.05 ± 0.75	0.076
Total cholesterol, mg/dL	182.8 ± 46.0	180.8 ± 88.8	0.816
Triglyceride, mg/dL	180.4 ± 116.2	153.2 ± 101.0	0.190
LDL cholesterol, mg/dL	115.5 ± 48.5	114.0 ± 36.8	0.856
HDL cholesterol, mg/dL	38.3 ± 8.4	40.3 ± 10.5	0.310
Ejection fraction, %	43.6±8.7	45.8 ± 7.8	0.125
WBC, 10^9/L	13.11 ± 3.89	10.34 ± 2.76	<0.001
Hemoglobin, g/dL	14.20 ± 1.94	14.15 ± 1.99	0.895
Platelet, 10^9/L	323.9 ± 74.4	217.5 ± 51.8	<0.001
Platecrit, %	0.345 ± 0.062	0.229 ± 0.039	<0.001

STEMI: ST-segment elevation myocardial infarction, LDL: Low density lipoprotein, HDL: Density lipoprotein, WBC: White blood cell.

Coronary Artery Disease / Acute Coronary Syndrome

OP-028

The efficacy and safety of sodium-glucose cotransporter 2 inhibition in patients with acute ST-segment elevation myocardial infarction

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Background and Aim: The landmark studies of sodium-glucose co-transporter-2 (SGLT-2) inhibitors demonstrated cardiovascular protection, and current guidelines recommended SGLT-2 inhibitors. However, in patients with acute ischemia the efficacy is not well established. We aimed to investigate the efficacy and safety of SGLT-2 inhibitors in patients with recent ST-segment elevation myocardial infarction (STEMI).

Methods: Patients with STEMI who underwent successful primary percutaneous coronary intervention were included. Patients treated with SGLT-2 inhibitors during the index hospitalization constituted group 1, and patients with other classes of antidiabetic therapy were included in group 2. Patients' demographics, comorbidities and clinical features were recorded. Cardiovascular outcomes, including cardiovascular death and new-onset heart failure were also recorded.

Results: After the exclusion, there were 145 patients in group 1 and 135 patients in group 2. There was no significant difference between groups in terms of hypertension (p=0.064), dyslipidemia (p=0.826), previous cerebrovascular accident (p=0.348) and smoking habits (p=0.871). There was also no significant difference in terms of multivessel disease (p=0.259) and left main coronary artery disease (p=0.062). Left ventricular ejection fraction was similar between groups (p=0.297). Pro-BNP and peak troponin levels were also comparable between groups (p=0.668 and p=0.088, respectively), however, follow-up pro-BNP levels were significantly lower in group 1 patients (p=0.042). Cardiovascular death and new onset heart failure were both lower in group 1 patients (p=0.033 and p=0.002, respectively). There was no drug-related adverse effect requiring the drug cessation indicating the good safety profile.

Conclusions: Our results indicated that SGLT-2 inhibitors are efficacious in patients with recent STEMI. Further studies with longer follow-up and higher study populations are required to validate our results.

Coronary Artery Disease / Acute Coronary Syndrome

OP-029

Long term impact of clinical pharmacist-led theory based discharge education service in patients with acute coronary syndrome: A randomized controlled trial

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Background and Aim: Patients with acute coronary syndrome have an increased risk of re-infarction and death. To reduce this risk, secondary prevention (including cardioprotective medications and healthy lifestyle behaviors) is recommended. Despite the importance of secondary prevention on patients outcomes, it is stated that patients' adherence to cardioprotective medications is low. In this study, it is aimed to evaluate the clinical pharmacist-led theory-based discharge education and counseling services on clinical outcomes of patients with acute coronary syndrome.

Methods: In this prospective parallel randomized controlled trial (NCT05153707), adult patients who were hospitalized due to acute coronary syndrome in the cardiology clinic of a university hospital were recruited for a year. The patients were allocated to the control group or group that received clinical pharmacist-led theory-based discharge education service by permutated block randomization. The clinical pharmacist, within the scope of the education services, provided medication reconciliation, medication review, individually prepared patient pill card, theory-based patient education [based on the Health Belief Model by utilizing written material and video, and verbally (by using Teach back method, especially for patients with low health literacy)] and counseling [utilizing behavior change techniques based on The Capability, Opportunity, and Motivation Behavior (COM-B) model]. The control group received standard care.

Results: In this study, 167 patient was recruited. There was no statistical difference in the characteristics of patients between the control group (n=87) and the group that received education service (n=80) except for sex. The rate of female patients was significantly lower in the control group (p<0.05). In the control group, 13 patients were readmitted to the hospital for cardiovascular reasons in 360 days compared to 3 patients in the group that received education service. When compared with the control group (14.9%), the rate of 360-day readmission for cardiovascular reasons was statistically lower in the group that received education service (3.8%) (p<0.05). In the control group, 14 patients were readmitted to the hospital for all causes in 360 days compared to 4 patients in the group that received education service. When compared with the control group (16.1%), the rate of 360-day readmission for all causes was statistically lower in the group that received education service (5%) (p<0.05). In the control group, four (4.6%) patients died due to cardiovascular reasons, and six patients (6.9%) died due to all causes within 360 days after discharge. In the group that received education service, there was no all-cause or cardiovascular death within 360 days of discharge.

Conclusions: The findings of our study demonstrate that theory-based education service given to patients with acute coronary syndrome within the scope of clinical pharmacy services may have positive contributions to long-term clinical outcomes.

Coronary Artery Disease / Acute Coronary Syndrome

OP-030

Relationship between fibrinogen to albumin ratio and thrombus burden in patients with acute coronary syndrome

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Background and Aim: Increased coronary thrombus burden is known to be a strong predictor of adverse cardiovascular (CV) outcomes. Fibrinogen to albumin ratio (FAR) can be used as a surrogate marker of pro-inflammation which is closely related to prothrombotic state. We aimed to evaluate the association between FAR and coronary thrombus burden in patients who presented with acute coronary syndrome (ACS). Patients who presented with ACS and treated with percutaneous coronary intervention (PCI) were included in the study.

Methods: 205 ACS (NSTE-ACS 48.7% and STEMI 51.3%) patients who underwent PCI between June 2017 and June 2021 were included in the study. Angiographic coronary thrombus load was performed according to the classification of 'thrombolysis in myocardial infarction (TIMI). The patients were divided into two groups as low thrombus burden (LTB, TIMI thrombus burden grade 0-1-2) and high thrombus burden (HTB, TIMI thrombus burden grade 3-4-5).

Results: The FAR was significantly higher in those with HTB than those with LTB (92.7 ± 21.8 vs. 61.1 ± 19.3, p<0.001). In multivariate logistic regression analysis, high FAR found to be an independent predictor of high thrombus load (OR: 0.930, 95% CI: 0.971-1.139; p<0.001). In ROC analysis, a cut-off value of 57.5 for FAR had an 74.7% sensitivity and 67.8% specificity for predicting high coronary thrombus burden in ACS patients with PCI (AUC: 81.4, p<0.001).

Conclusions: As a result, the increased FAR is an independent parameter that predicts high coronary thrombus burden ACS patiens with PCI.

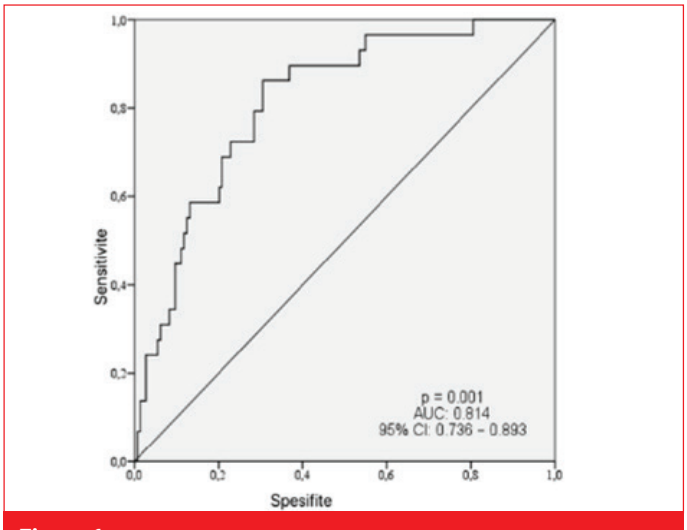

Figure 1.

Table 1. Demographic and laboratory findings

Variables	LTB (n=85)	HTB (n=120)	p value
Age	50.6 ± 12.3	49.6 ± 12.8	0.058
Gender, Male, n (%)	51	58	0.661
HTN, n	45	49	0.411
DM, n	31	28	0.061
Hyperlipidemia, n	14	15	0.549
Smoking, n	42	46	0.413
WBC, 10^3 uL	6.5 ± 3.2	6.3 ± 1.6	0.152
Neutrophil, 10^3 uL	4.5 ± 2.4	4.1 ± 2.1	0.124
Platelet, 10^3 uL	245 ± 83	233 ± 75	0.337
Hemoglobin, g/dl	12.8 ± 1.7	12.7 ± 1.6	0.354
Glucose, mg/dl	99.0 ± 9.5	96.5 ± 12.2	0.566
Aspartate aminotransferase, IU/l	28.9 ± 7.6	24.7 ± 8.7	0.135
Alanine aminotransferase, IU/l	28.5 ± 9.4	25.2 ± 15.5	0.278
Creatinin mg/dl	0.85 ± 0.25	0.89 ± 0.22	0.875
LDL cholesterol, mg/dL	91.8 ± 16.1	75.4 ± 15.7	0.085
Fibrinogen, mg/dL	245 ± 21	334 ± 26	**<0.001**
Albumin, mg/dL	4 ± 1.1	3.6 ± 0.8	**<0.001**
CRP, mg/L	6.4 ± 3.3	6.9 ± 2.8	0.056
FAR	61.1± 19.3	92.7± 21.8	**<0.001**

DM: Diabetes Mellitus; HTN: Hypertension; LDL, Low density lipoprotein; CRP: C-reactive protein, FAR: , Fibrinogen to albumin ratio

Table 2. Multivariate logistic regression analysis showing independent thrombus burden preditive variables

Analysis	Univariate			Multivariate		
Variables	Odds ratio	95% CI	p value	Odds ratio	95% CI	p value
Age	1.063	1.037-1.703	1.171			
FAR	1.044	0.930-1.053	**<0.001**	1.029	1.013-1.045	**<0.001**
DM	1.051	0.971-1.139	0.216			
HT	1.284	0.638-2.585	0.483			
LDL	1.534	1.377-1.073	0.059			
CRP	1.105	1.005-1.204	0.061			

DM: Diabetes Mellitus; HTN: Hypertension; LDL, Low density lipoprotein; CRP: C-reactive protein, FAR: , Fibrinogen to albumin ratio

Coronary Artery Disease / Acute Coronary Syndrome

OP-031

Is aortic knob related to Framingham score in individuals with stenosis on coronary angiography?

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Background and Aim: The aim of our study; to investigate whether aortic knob width (AKW) is associated with Framingham risk score (FRS) in individuals who have visually detected coronary artery disease in individuals who have undergone coronary angiography.

Methods: 147 patients (84 males, 63 females, 54.9 ± 124 years) who applied to the cardiology outpatient clinic with any complaint and had coronary angiography indication according to the examinations were included in the study. Angiography results of the patients were evaluated visually. The presence of coronary artery disease was evaluated visually by 2 separate operators who are blinded to each other. Demographic data and laboratory values of all patients were recorded. Aortic knob width was measured along the

straight imaginary line from the lateral edge of the trachea to the widest point of the aortic knob on the left lateral wall of the aortic arch on the posteroanterior chest X-ray. The patients' 10-year risk of having a heart attack was obtained by FRS. FRS; age, gender, smoking, total and high-density lipoprotein (HDL) cholesterol, systolic blood pressure, and history of drug use due to hypertension were calculated from the calculation system (https://www.mdcalc.com/calc/38/framingham-risk-score-hard-coronary-heart-disease).

Results: As a result of angiography, coronary artery disease was detected visually in 97 patients (67 men, 30 women; 60.1 ± 10.5 years) (group 1), while coronary artery disease was not detected in 50 patients (33 men, 17 women; 45.4 ± 9.1 years) (group 2). FRS, AKW were significantly higher in group 1 compared to group 2. Age, gender, systolic-diastolic blood pressure, and low-density lipoprotein cholesterol values were similar between the groups (Table 1). Glucose levels, body mass index, triglyceride, and total cholesterol levels were significantly higher in group 1 compared to group 2, while HDL cholesterol levels were significantly lower (Table 1). The number of smoking and statin use was higher in group 1 than in group 2. The rates of presence of diabetes, presence of hypertension, use of antihypertensives and presence of coronary artery disease in the family were similar. In the correlation analysis, there was a significant positive relationship between AKW and FRS, age, systolic blood pressure, glucose, and a negative relationship with HDL. There was no relationship between total cholesterol and low-density lipoprotein (Table 2). In the case of the group with only coronary artery disease, there was again a weak correlation between AKW and FRS (r=0.28, p=0.007).

Conclusions: Aortic knob width and FRS are significantly higher in patients detected visual CAD on coronary angiography than in those without. Aortic knob width, which can be easily obtained in the posteroanterior chest X-ray, shows a significant correlation with FRS in individuals with coronary artery disease. It may be effective in determining long-term risk in individuals with AKW.

Table 2. Correlation analysis of aortic knob width between the parameters

	rho	p
FRS	0.60	<0.001
Age	0.48	<0.001
Systolic BP	0.46	<0.001
Glucose	0.24	0.004
HDL-C	-0.32	<0.001
Total C	0.14	0.083
LDL-C	0.02	0.840

Table 1. Comparison of the demographic, laboratory features and aortic knob width, Framingham risc score of the groups

	Group 1 (n=97)	Group 2 (n=50)	p
Age, years	61.6 ± 10.9	58.7 ± 10.2	0.176
Gender (female), n	30	17	0.252
Hypertension, n	30	30	0.227
Smoking, n	44	19	<0.001
Diabetes mellitus, n	20	21	0.565
Family history, n	28	19	0.724
Antihypertensive drug, n	26	28	0.100
Statin use, n	22	11	0.029
Systolic blood pressure, mmHg	142.2 ± 7.8	137.2 ± 16.8	0.221
Diastolic blood presure, mmHg	77.8 ± 13.7	75.6 ± 10.6	0.369
Heart rate, beat/min	83.4±12.6	80.1±11.9	0.701
BMI, kg/m^2	29.5 ± 5.5	22.6 ± 1.7	<0.001
FRS	23.8 ± 13.1	4.6 ±4.3	<0.001
AKW, mm	37.2 ± 5.4	30.2 ±3.2	<0.001
Glucose, mg/dL	121.2 ± 50.5	91.2 ± 8.4	<0.001
Total cholesterol, mg/dL	210.1 ± 46.5	189.8 ± 32.5	0.007
Triglyceride, mg/dL	146.6 ± 78.5	83.6 ± 50.7	<0.001
LDL cholesterol, mg/dL	115.8 ± 43.7	122.5 ± 33.0	0.343
HDL cholesterol, mg/dL	43.1 ± 9.8	55.1 ± 13.3	<0.001

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-032

The effect of ivabradine on ventricular arrhythmias in heart failure with reduced ejection fraction patients

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Background and Aim: Heart failure patients with reduced ejection fraction (HFrEF) are at high risk for ventricular arrhythmias and sudden cardiac death. Ivabradine, a specific inhibitor of the if current in the sinoatrial node, provides heart rate reduction in sinus rhythm and angina control in chronic coronary syndromes. The effect of ivabradine on ventricular arrhythmias in HFrEF patients has not been fully elucidated. In this study, we aimed to investigate the effect of ivabradine use on life-threatening arrhythmias and long-term mortality in HFrEF patients.

Methods: In this retrospective study, 1639 patients with HFrEF were included. Patients were divided into two groups as ivabradine users and non-users. Patients presenting with ventricular tachycardia, presence of ventricular extra systole and ventricular tachycardia in 24-hour rhythm monitoring, appropriate ICD shocks and long-term mortality outcomes were evaluated according to ivabradine use.

Results: After adjustment for all possible variables, admission with ventricular tachycardia was 3.0 times higher in ivabradine non-users (95% CI: 1.5-10.2). Presence of premature ventricular contractions and ventricular tachycardias in 24-h holter rhythm monitoring were notably higher in ivabradine non-users. According to the adjusted model for all variables, 4.1 times more appropriate ICD shocks were observed in the ivabradine non-users than the users (95% CI: 1.8-9.6). Long-term mortality did not differ between these groups after adjustment for all covariates.

Conclusions: The use of ivabradine has been shown to reduce appropriate ICD therapy in patients with HFrEF. The use of ivabradine in HFrEF patients may have potential in preventing ventricular arrhythmias.

Table 1. Comparison of demographic, clinical characteristics, laboratory and echocardiography parameters of patients according to ivabradine usage in patients with heart failure with reduced ejection fraction

	Overall (n=1639)	Patients not using ivabradine (n=1363)	Patients using ivabradine (n=276)	P value
Age, y	71 (63 – 79)	71 (62 – 79)	74 (65 – 82)	<0.001
Male gender	946 (57.7%)	797 (58.5%)	149 (54.0%)	0.169
Hypertension	969 (59.1%)	809 (59.4%)	160 (58.0%)	0.670
Diabetes mellitus	596 (36.4%)	486 (35.7%)	110 (39.9%)	0.186
Hyperlipidemia	469 (28.8%)	393 (29.1%)	76 (27.7%)	0.657
Smoking	156 (9.6%)	130 (9.6%)	26 (9.5%)	0.937
Chronic renal failure	408 (25.0%)	345 (25.4%)	63 (22.8%)	0.370
COPD	186 (11.5%)	152 (11.3%)	34 (12.4%)	0.592
Cerebrovascular accident	26 (1.6%)	21 (1.6%)	5 (1.8%)	0.791
Hypothyroidism	63 (3.9%)	51 (3.8%)	12 (4.4%)	0.645
Hyperthyroidism	37 (2.3%)	28 (2.1%)	9 (3.3%)	0.249
Coronary artery disease	1142 (69.7%)	942 (69.1%)	200 (72.5%)	0.269
Heart failure etiology				
Ischemic	1086 (66.3%)	913 (67.0%)	173 (62.7%)	0.168
Non-ischemic	553 (33.7%)	450 (33.0%)	103 (37.3%)	0.168
Device types				
ICD	281 (17.2%)	212 (15.6%)	69 (25.1%)	<0.001
CRT-D	105 (6.4%)	83 (6.1%)	22 (8.0%)	0.259
All defibrillators	386 (23.7%)	295 (21.8%)	91 (33.2%)	<0.001
Laboratory values				
Creatinine, mg/dL	1.0 (0.8–1.3)	1.0 (0.8–1.3)	1.0 (0.8–1.3)	0.350
Potassium, mEq/L	4.5 (4.2–4.8)	4.5 (4.2–4.8)	4.4 (4.1–4.7)	0.108
Magnesium, mEq/L	2.1 (1.9–2.3)	2.1 (1.9 – 2.3)	2.1 (1.9 – 2.3)	0.330
Calcium, mEq/L	9.3 (8.9– 9.6)	9.3 (8.9 – 9.6)	9.3 (9.0 – 9.6)	0.252
Echocardiography data				
LVEF, %	30(25-35)	30(25–35)	30(25– 35)	0.475
LVEDD, mm	60(54–68)	60(54– 68)	61(56– 67)	0.114
LVESD, mm	48(41– 56)	48(41– 56)	50(42– 57)	0.154
LAAP, mm	44(40–49)	44.0 (40–50)	45(40– 48)	0.949
Out-hospital medication				
Beta-blockers	1624 (99.1%)	1350 (99.0%)	274 (99.3%)	0.715
ACEIs or ARBs	1105 (67.4%)	921 (67.6%)	184 (66.7%)	0.770
Spironolactone	1026 (62.6%)	845 (62.0%)	181 (65.6%)	0.262
Furosemide	1516 (92.5%)	1256 (92.1%)	260 (94.2%)	0.238

Table 2. Distribution of patients' ventricular arrhythmias, appropriate ICD treatments, and long-term mortality according to ivabradine use

	Patients not using ivabradine (n=1363)	Patients using ivabradine (n=276)
Admission with ventricular tachycardia	44(3.2%)	4(1.4%)
Presence of premature ventricular contractions>5% in 24-hour rhythm Holter monitoring	169 (36.2%)	60(21.7%)
Ventricular tachycardia in 24-hour rhythm Holter monitoring	12(2.6%)	2(0.7%)
Appropriate ICD shock in follow-up	64(21.7%)	7(7.7%)
Long-term mortality	143 (10.5%)	22(8%)

Abbreviations: ICD, implantable cardioverter defibrillator.

Table 3. Multivariate analysis for admission with ventricular tachycardia, presence of premature ventricular contractions>5% in 24-holter rhythm monitoring, ventricular tachycardia in 24-holter rhythm monitoring, appropriate ICD shock in follow-up and long-term mortality by ivabradine usage

	Patients Not Using Ivabradine	Patients Using Ivabradine
Admission with ventricular tachycardia, HR (95% CI)		
Model 1: unadjusted	3.6 (1.3 – 10.2)	1[Reference
Model 2: adjusted for all covariates[a]	3.0 (1.5 – 7.4)	1[Reference]
Long-term mortality, HR (95% CI)		
Model 1: unadjusted	1.9 (1.2 – 3.0)	1[Reference]
Model 2: adjusted for all covariates[a]	1.4 (0.8 – 2.8)	1[Reference]
Presence of premature ventricular contractions in 24-h rhythm Holter monitoring, HR (95% CI)		
Model 1: unadjusted	2.9 (2.1 – 3.9)	1[Reference]
Model 2: adjusted for all covariates[a]	2.4 (1.1 – 3.1)	1[Reference]
Ventricular tachycardia in 24-h rhythm Holter monitoring, HR (95% CI)		
Model 1: unadjusted	6.7 (1.4 – 30.4)	1[Reference]
Model 2: adjusted for all covariates[a]	4.2 (1.9 – 12.1)	1[Reference]
Appropriate ICD shock in follow-up, HR (95% CI)		
Model 1: unadjusted	4.3 (2.0 – 9.5)	1[Reference]
Model 2: adjusted for all covariates[a]	4.1 (1.8 – 9.6)	1[Reference]

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-033

The clinical monitoring effect of intracardiac morphology changes in patients with defibrillator device

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Background and Aim: The aim of our study was to demonstrate the relationship between intracardiac morphological changes and clinical parameters and patient characteristics, as well as to how whether this value could predict patient fallow-up and clicial outcomes in outpatient monitoring. According to our hypothesis, device morphology should detect ongoing cardiac remodelling, which could have a role in HF monitoring.

Methods: 84 patients who applied to our clinic for device control were included in the study. Morphological changes, SF-12 quality of life score, comorbities, current laboratuary tests and current echocardiograms were performed as part of the device control. Every patient who were device owner, regardless of heart falure, was included in the study. In the control of St. Jude/Abbott devices, the patients' device EGM recordings were examined and captured as photographs while the device was in normal rhythm. During the recording of morphologies, approximately 6-10 beats' average of recorded morphologies on the device screen were noted. Additionally, the therapy records of the patients were also reviewed. In our study, the patients' device screen images were captured by us, and the arithmetic average of morphology values was obtained, resulting in a numeric value between 0 and 100.

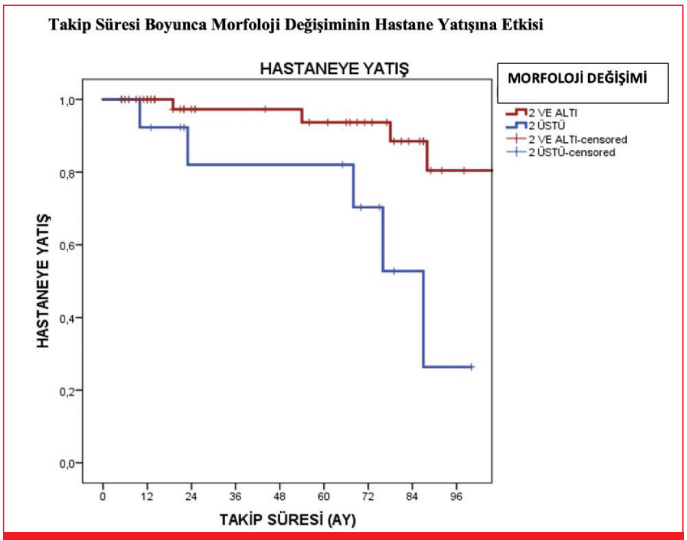
Results: It was shown that patients with high BNP levels in laboratory values (p=0.058) might have lower morphologies approaching statistical significance. Significant correlation between valve insufficiencies and morphological changes could not demonstrated in the echocardiograms performed for each patient. Although the EF value was higher on average in patients with a morphology score of 100 (%39.3-%35.2), it did not reach statistical significance (p=0.18) Nevertheless, it was shown that in echocardiograms, the diameters obtained by dividing the diastolic and systolic diameters by the body surface area were larger in patients with lower morphology scores (p=0.03). As a secondary endpoint, when the hospitalization rates of patients due to decompensation after device implantation were examined, patients with a morphology change of 2 or above during the control had 5.6 times higher hospitalization rates (p=0.02). This result was consistent when evaluated with multiple regression analysis. This indicates that morphological changes could be involved in the follow-up of patients with implantable cardioverter-defibrillator devices.

Conclusions: The quality of life score of patients with a high average value of morphology is significantly higher in a statistical sense (p=0.015). Hospitalization rates for patients due to congestive heart failure (CHF) requiring intravenous diuretic therapy after device implantation, it was shown that patients with a morphology change of 2 or above during the control had 5.6 times higher hospitalization rates (p=0.02).

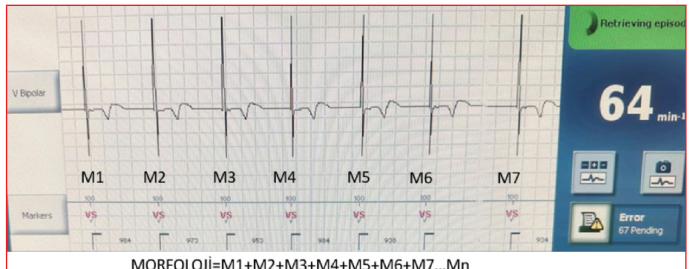
Tablo 1. Demografik özellikler

Demografik Veriler	
Yaş⁺ yıl	67,5±7
Boy⁺ cm	172±5
Kilo ⁺kg	77±9
BMI *kg/m2	26,3
Cinsiyet , kadın, n(%)	17 (%20,2)
,erkek (%)	67 (%79,8)
Takip Süresi , ay	56
KAH , n(%)	44 (%52,4)
Diyabet , n(%)	35 (%41,7)
Cihaz tipi , n(%)	
VVI-ICD n(%)	42 (%50)
DDD-ICD n(%)	14 (%16,7)
CRT-ICD n(%)	28 (%33,3)
Primer profilaksi n(%)	61 (%72,6)
Epizod görülen n(%)	28 (%33,3)
VT terapi alan n(%)	4 (%4,8)
ACE-i kullananlar n(%)	45(%53,6)
ARB kullananlar n(%)	15 (%17,9)
ARNI kullananlar n(%)	12 (%14,3)
BB kullananlar	79 (%94)
MRA kullananlar n(%)	53 (%63,1)
SGLT-2 inh. kullananlar n(%)	23 (%27,4)
Diüretik kullananlar n(%)	29 (%34,5)
İvabradin kullananlar n(%)	15 (%17,9)
Antiaritmik kullananlar n(%)	27 (%32,1)

Ortalama* (± *standart deviasyon*) , Ortanca⁺ (çeyrekler arası aralık)



Şekil 1. Hastaneye yatışın morfoloji değişimi ile ilişkisi. 2 birim ve üzeri morfoloji değişimi olan hastaların takip süresince hastaneye yatışının değerlendirilmesi



MORFOLOJİ=M1+M2+M3+M4+M5+M6+M7...Mn / n

Şekil 2. Morfoloji diskriminasyonu. Morfoloji ortalamasının hesaplaması.

Tablo 2. Morfoloji değişiminin etkisi. EGM morfolojisi değişenler ile değişmeyenlerin karşılaştırılması

	Morfoloji=100	Morfoloji<100	P değeri
YAŞ+	64,5	67,5 (58-73)	0,8
SF12*	49.7±5.2	46.5±5.9	0.026
EF*,%	39,3±14,7	35,2±11,5	0.18
LVEDC*,mm/m2	28,4±4,5	31,4±5.6	0.03
LVESC *,mm/m2	21,9±5,4	24,7±6.2	0.065
BNP+ ,pg/mL	180 (70-283)	187 (62-454)	0,058
ALB+ ,g /dL	4,3 (4-4,4)	4,2 (4,1-4,4)	0,976
K *, mmol/L	4,47±0,37	4,49±0,43	0,89
NA+ ,mmol/L	139 (138,141,2)	140 (137-140)	0,83
HB+ ,g /dL	14 (13-15,1)	13,7 (12,3-14,5)	0,066
WBC*·10*3/µL	8,7±1,7	7,9±1,87	0,68
PLT+·10*3/µL	223000 (184000-267000)	211000 (179500-261500)	0,72
GFR*, ml/dak/ m2	80,7±22,3	69,7±24,6	0,066
BMI* ,kg/m2	26,2±3,5	26,4±4,3	0,878
PAB+ ,mmHg	25 (25-32,5)	32,5 (25-40)	0,1
BSA+ ,m2	1,97 (1,9-2,1)	1,89 (1,75-2,03)	0,22

Ortalama* (± standart deviasyon) , Ortanca+ (çeyrekler arası aralık)

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-034

Evaluation of natriuretic peptide levels and echocardiography findings in patients with micro AF

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Background and Aim: Micro-AF, defined as brief episodes of rapid irregular atrial activity, is a predictor for atrial fibrillation (AF), which is an important cause of mortality and morbidity. However, micro-AF may be overlooked diagnosis. The aim of our study is to investigate the predictability of laboratory, electrocardiographic and echocardiographic findings for the diagnosis of micro-AF.

Methods: Micro-AF was defined as sudden onset of irregular tachycardia with episodes of ≥5 consecutive supraventricular beats and total absence of p-waves, lasting less than 30 s. Two groups were formed as patients with and without Micro-AF detected in 24-hour rhythm Holter analysis. Laboratory parameters, electrocardiographic and echocardiographic findings of the two groups were compared.

Results: A total of 90 patients, 45 patients with micro-AF and 45 patients in the control group, were included in our study. Serum troponin T (13.09 ± 11.45 and 4.41 ± 2.46 ng/dL; p<0.001) and pro-BNP (375.57 ± 636.68 and 63.19 ± 56.82 pg/mL; p<0.001) micro it was found to be higher in the AF group. Serum ANP values (160.19 ± 83.94 and 278.63 ± 196.51 ng/L; p<0.001) were higher in the control group. We found a 63.4 pg/dL pro-BNP cut-off value for the diagnosis of micro-AF with a sensitivity of 91.1% and a specificity of 73.3%. Each 1 pg/dL increase in serum pro-BNP level increased the risk of micro-AF by 1.8%. In ECG evaluation, P-max (113.00 ± 10.25 and 98.00 ± 10.47 ms; p<0.001), P-min (73.89 ± 5.53 and 70.00 ± 6.39 ms; p<0.001) and P-dd (39.11 ± 7.93 and 28.00 ± 7.64 ms; p<0.001) were longer in the micro-AF group. In echocardiographic findings, interventricular septum wall thicness (11.14 ± 1.37 and 9.65 ± 2.65 mm; p=0.003), posterior wall thicness (12.13 ± 6.58 and 9.78 ± 2.63 mm; p=0.002) and left atrial diameter (35.92 ± 1.30 and 34.62 ± 1.77 mm; p<0.001) were found to be higher in the micro-AF group. In the micro-AF group, mitral A wave velocity was higher (0.79 ± 0.18 and 0.71 ± 0.13 cm/h; p=0.019). e' and s' wave velocities measured were lower, and E/e' ratios were higher from 6 regions. Atrial electro-mechanical delay times (PA) were significantly higher in the micro-AF group. Lateral tricuspid PA cut-off value of 18.5 seconds was determined for micro-AF with a sensitivity of 93.3% and a specificity of 91.1%; lateral septal PA cut-off value of 11.5 seconds was determined with 95.6% sensitivity and 75.6% specificity.

Conclusions: In conclusion, in our patients who applied to the outpatient clinic with the complaint of palpitation, serum pro-BNP, serum troponin T levels, P-max, P-dd times in ECG evaluation, left atrial diameter in echocardiographic evaluation, E wave velocity, A wave velocity, e' measurement, E/e' rate and measurement of AEMD durations may allow us to identify AF patients early.

Tablo 1. Ekokardiyografi parametrelerinin karşılaştırılması

	Mikto AF	Kontrol Grubu	İstatistiksel Analiz
EF (%)	61,62±4,86	61,53±5,06	t=0,083 p=0,934
LVDSÇ (mm)	47,67±4,12	47,56±3,11	Z=-0,811 p=0,417
LVSSÇ (mm)	31,52±4,18	31,59±2,79	Z=-0,569 p=0,569
Septum (mm)	11,14±1,37	9,65±2,65	Z=-2,964 **p=0,003**
Arka Duvar (mm)	12,13±6,58	9,78±2,63	Z=-3,073 **p=0,002**
LA (mm)	35,92±1,30	34,62±1,77	Z=-3,577 **p<0,001**
Aort Kökü (mm)	19,35±1,39	18,58±2,89	Z=-1,417 p=0,156
E (cm/s)	0,76±0,16	0,74±0,12	Z=-0,408 p=0,683
A (cm/s)	0,79±0,18	0,71±0,13	Z=-2,350 **p=0,019**
E/A	1,01±0,30	1,07±0,25	Z=-1,429 p=0,153
PASP (mm Hg)	30,91±6,68	17,64±3,59	Z=-7,824 **p<0,001**
Lateral (TE-e') (ms)	37,87±22,34	39,67±21,67	t=-0,388 p=0,699
Septal (TE-e') (ms)	42,36±20,14	45,38±27,26	Z=-0,440 p=0,660
Lateral e' (cm/s)	8,30±1,47	13,24±2,68	t=-10,866 **p<0,001**
Septal e' (cm/s)	7,12±1,19	11,37±2,11	t=-11,794 **p<0,001**
Lateral E/e'	9,35±2,04	5,68±1,05	Z=-7,904 **p<0,001**
Septal E/e'	10,92±2,49	6,58±1,13	Z=-7,767 **p<0,001**
Lateral PA (ms)	108,44±9,63	89,07±6,56	Z=-7,674 **p<0,001**
Septal PA (ms)	92,44±7,90	79,18±6,70	t=8,589 **p<0,001**
Trikuspit PA (ms)	82,76±6,79	74,84±6,56	t=5,614 **p<0,001**
Lateral Septal PA (ms)	16,20±3,65	9,80±2,79	Z=-6,907 **p<0,001**
Septal Trikuspit PA(ms)	9,69±3,16	4,33±1,39	Z=-7,775 **p<0,001**
Lateral Trikuspit PA (ms)	25,84±5,64	14,13±3,32	Z=-7,811 **p<0,001**

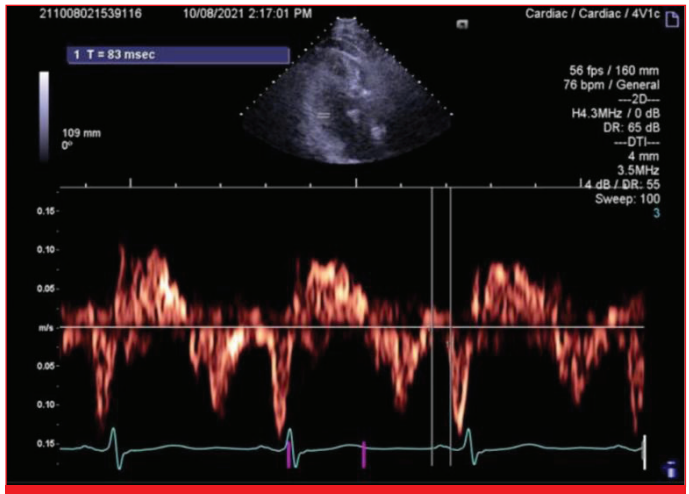
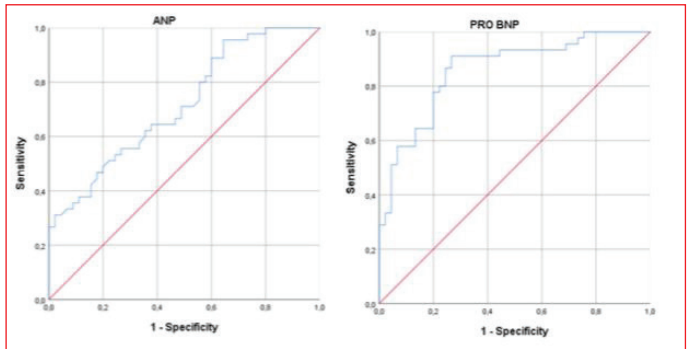
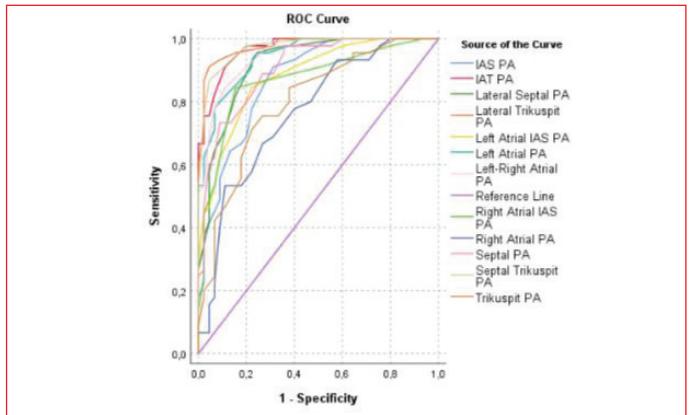


Figure 1. Measurement of atrial electromechanical delay by echocardiography.



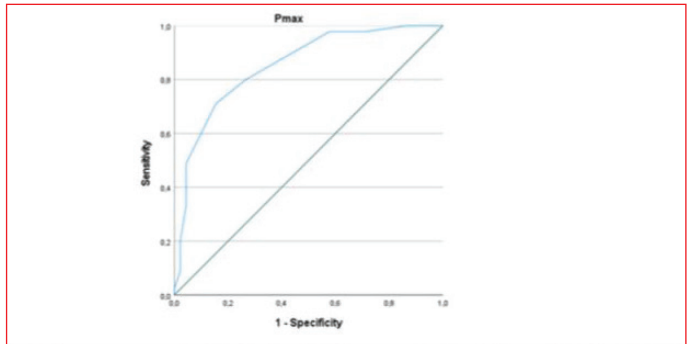
Değişken	Alan	Standart hata	p	%95 güven aralığı		Cut-off
				Alt sınır	Üst sınır	
ANP	0,716	0,053	p<0,001	0,612	0,820	147,4
PRO BNP	0,860	0,039	p<0,001	0,783	0,936	63,4

Şekil 2. Mikro-AF durumuna göre ANP ve pro-BNP cut-off değerinin belirlenmesi.



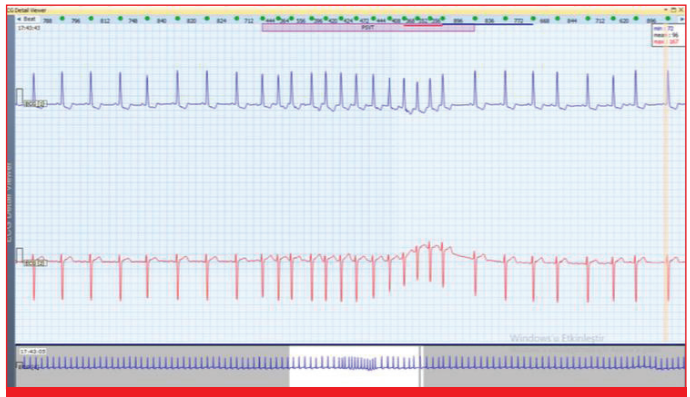
Değişken	Alan	Standart hata	p	%95 güven aralığı		Cut-off
				Alt sınır	Üst sınır	
Lateral PA	0,969	0,014	p<0,001	0,941	0,997	97,5
Septal PA	0,906	0,030	p<0,001	0,847	0,965	85,5
Trikuspit PA	0,800	0,046	p<0,001	0,709	0,891	78,5
Lateral Septal PA	0,921	0,028	p<0,001	0,866	0,976	11,5
Septal Trikuspit PA	0,973	0,014	p<0,001	0,946	1,000	5,5
Lateral Trikuspit PA	0,977	0,013	p<0,001	0,953	1,000	18,5
Left Atrial PA	0,944	0,021	p<0,001	0,902	0,986	90,5
IAS PA	0,873	0,036	p<0,001	0,802	0,943	78,5
Right Atrial PA	0,767	0,050	p<0,001	0,669	0,865	72,5
Left Atrial IAS PA	0,883	0,034	p<0,001	0,816	0,950	9,5
Right Atrial IAS PA	0,872	0,039	p<0,001	0,796	0,948	5,5
Left-Right Atrial PA	0,947	0,021	p<0,001	0,905	0,988	15,5

Şekil 3. Mikro-AF durumuna göre PA değerlerinin cutt-off değerinin belirlenmesi.



Değişken	Alan	Standart hata	p	%95 güven aralığı		Cut-off
				Alt sınır	Üst sınır	
Pmax	0,852	0,040	p<0,001	0,773	0,931	102,5

Şekil 4. Mikro-AF durumuna göre P-max cut-off değerinin belirlenmesi.



Şekil 5. Ritim holterde örnek mikro-AF kaydı.

Tablo 2. Biyokimya ve elektrokardiyografi bulgularının karşılaştırılması

	Mikro-AF	Kontrol grubu	İstatistiksel analiz
Kreatinin	0,82 ± 0,22	0,76 ± 0,17	p=0,390
Pro-BNP	375,57 ± 636,68	63,19 ± 56,82	p<0,001
ANP	160,19 ± 83,94	278,63 ± 196,51	p<0,001
TSH	1,52 ± 1,15	1,81 ± 0,94	p=0,071
Hemoglobin	13,38 ± 1,51	13,59 ± 1,73	p=0,125
Pmax	113,00 ± 10,25	98,00 ± 10,47	p<0,001
Pmin	73,89 ± 5,53	70,00 ± 6,39	p=0,009
Pdd	39,11 ± 7,93	28,00 ± 7,64	p<0,001

Tablo 3. Çalışma grubu demografik verileri

	Mikro-AF	Kontrol		Mikro-AF	Kontrol
Cinsiyet			ACEi		
Kadın	23	22	Kullanıyor	9	16
Erkek	22	23	Kullanmıyor	36	29
Diabetes mellitus			ARB		
Var	13	12	Kullanıyor	20	11
Yok	32	33	Kullanmıyor	25	34
Hipertansiyon			Kalsiyum kanal blokeri		
Var	34	28	Kullanıyor	9	10
Yok	11	17	Kullanmıyor	36	35
Koroner arter hastalığı			Beta-bloker		
Var	8	9	Kullanıyor	23	9
Yok	37	36	Kullanmıyor	22	36
SVH öyküsü			Oral antidiyabetik		
Var	23	6	Kullanıyor	13	11
Yok	22	39	Kullanmıyor	32	34
Sigara			İnsülin		
Kullanıyor	16	21	Kullanıyor	6	3
Kullanmıyor	29	24	Kullanmıyor	39	42
Alkol			Statin		
Kullanıyor	10	13	Kullanıyor	13	9
Kullanmıyor	35	32	Kullanmıyor	32	36

Tablo 4. Çalışma gruplarının karşılaştırılması

	Mikro-AF	Kontrol grubu
CHA2DS2-VASc skoru	3,09 ± 1,49	2,22 ± 1,46
Yaş	62,84 ± 8,22	60,36 ± 10,01
Beden kitle indeksi	29,71 ± 4,35	28,77 ± 5,11
Sistolik kan basıncı (mmHg)	130,42 ± 15,26	126,47 ± 14,92
Diyastolik kan basıncı (mmHg)	80,53 ± 9,50	78,11 ± 9,20
Nabız (vuru/dakika)	72,40 ± 10,01	73,00 ± 8,43

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-035

Clinical implication of early repolarization pattern in potentially serious congenital coronary artery anomalies

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Background and Aim: Congenital coronary artery anomalies (CCAAs) have serious life-threatening complications such as fatal ventricular arrhythmias, syncope, and sudden cardiac death (SCD). Omreover, early repolarization pattern (ERP) has been associated with an increased risk of ventricular fibrillation and SCD. In this study, we aimed to evaluate the relationship between ERP and poor cardiovascular clinical outcomes in patients with potentially serious CCAAs.

Methods: This retrospective study included 106 potentially serious CCAA patients (mean age: 55 ± 13 years; male: 54) who were diagnosed with conventional and coronary computed tomography angiography (CCTA). All patients underwent transthoracic echocardiography and 12-lead surface electrocardiography. Cardiac events were defined as sustained ventricular tachycardia or fibrillation, syncope, cardiac arrest, and SCD.

Results: The presence of interarterial course (IAC) was confirmed by CCTA in 48 (44.3%) patients. During a median follow-up time of the 24 (18-50) months, a total of 12 (11.3%) patients experienced cardiac events. The presence of IAC was significantly more frequent and ERP were significantly greater in patients with poor clinical outcomes. Moreover, the presence of IAC and ERP were found to be independent predictors of poor clinical outcomes and decreased long term cardiac event free survival in these patients.

Conclusions: The current study shows that the ERP and IAC may be associated with poor cardiovascular clinical outcomes in potentially serious CCAA patients.

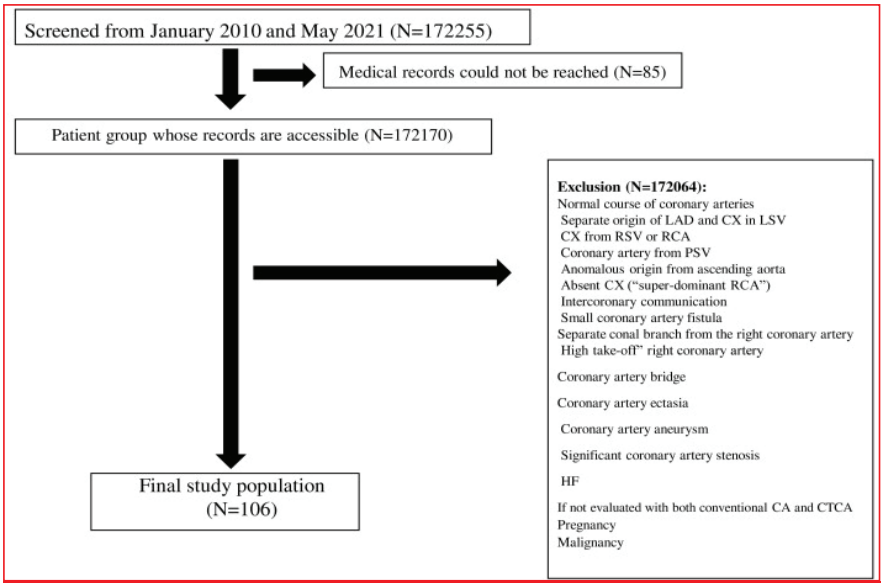

Figure 1. Flow chart for patient selection.

Table 1. The distribution of coronary artery anomalies among study population

Parameters	All patients (n=106)	Patients with poor outcomes (n=12)	Patients without poor outcomes (n=94)	p
Coronary artery anomalies				
LMCA from RSV, n (%)	20 (18.9)	3 (25.0)	17 (18.1)	0.403
LAD from RSV, n (%)	9 (8.5)	0	9 (9.6)	0.324
RCA from LSV, n (%)	32 (30.2)	4 (33.3)	28 (29.8)	0.518
LMCA from PA, n (%)	2 (1.9)	0	2 (2.1)	0.785
SCA from RSV, n (%)	15 (14.2)	1 (8.3)	14 (14.9)	0.466
SCA from LSV, n (%)	16 (15.1)	4 (33.3)	12 (12.8)	0.081
Large / multiple CAF, n (%)	7 (6.6)	0	7 (7.4)	0.420
Interarterial course, n (%)	48 (45.3)	37 (91.7)	11 (39.4)	0.001

CAF: Coronary artery fistulae, LAD: Left anterior descending coronary artery, LMCA: Left main coronary artery, LSV: Left sinus of valsalva, PA: Pulmonary artery, RCA: Right coronary artery, RSV: Right sinus of valsalva, SCA: Single coronary artery.

Table 2. Comparison of demographic, echocardiographic and electrocardiographic parameters between patients with and without poor cardiovascular clinical outcomes

Parameters	Patients without poor outcomes (n=94)	Patients with poor outcomes (n=12)	p
Age, years	56 ± 14	52 ± 9	0.342
Gender, male	47 (50.0)	7 (58.3)	0.587
Diabetes mellitus, n (%)	14 (14.9)	1 (8.3)	0.466
Hypertension, n (%)	37 (39.4)	2 (16.7)	0.109
Dyslipidemia, n (%)	25 (26.6)	1 (8.3)	0.151
Smoking, n (%)	20 (21.3)	2 (16.7)	0.527
Chronic renal disease, n (%)	25 (26.6)	0 (0)	0.032
Non-critical CAD, n (%)	36 (38.3)	5 (41.7)	0.528
LVEF, %	60 (55-65)	65 (60-65)	0.008
LAD, mm	37 (34-40)	35 (34-38)	0.314
LVEDD, mm	46 (44-50)	45 (44-45)	0.103
LVESD, mm	30 (26-32)	27 (26-29)	0.070
PWT, mm	10 (9-11)	10 (9-10)	0.081
IVST, mm	11 (11-12)	11 (11-12)	0.449
Diastolic dysfunction, n (%)	20 (21.3)	1 (8.3)	0.263
P wave duration, msec	100 (94-108)	100 (93-109)	0.753
QRS duration, msec	88 (80-92)	86 (81-87)	0.349
T wave duration, msec	158 (148-165)	160 (143-166)	0.944
PR interval, msec	166 (144-184)	166 (148-178)	0.881
Pathological Q wave, n (%)	6 (6.4)	0 (0)	0.477
Early repolarization, n (%)	11 (11.7)	8 (66.7)	<0.001
T wave inversion, n (%)	7 (7.4)	4 (33.3)	0.021
ST depression > 1 mm, n (%)	5 (5.3)	0 (0)	0.542
Antiplatelet agents, n (%)	31 (33.0)	2 (16.7)	0.211
B-blockers, n (%)	22 (23.4)	1 (8.3)	0.213
CCB, n (%)	18 (19.1)	2 (16.7)	0.597

CAD: Coronary artery disease, CCB: Calcium channel blocker, CV: Cardiovascular, IVST: Interventricular septal thickness, LAD: Left atrial diameter, LVEDD: Left ventricular end diastolic diameter, LVEF: Left ventricular ejection fraction, LVESD: Left ventricular end systolic diameter, PWT: Posterior wall thickness.

Table 3. Multivariate logistic regerssion analysis showing independent predictors of poor cardiovascular clinical outcomes in patients with potentially serious coronary artery anomalies

Parameters	OR	95% CI	p
Early repolarization	14.971	2.402-93.316	0.004
Interarterial course	10.056	1.059-95.510	0.044

CI: Confidence Interval, OR: Odds ratio.

Congenital Heart Diseases

OP-039

Long-term follow-up results of adult ASD population

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Background and Aim: Atrial septal defect (ASD) is an abnormal communication between left and right atrium due to defective development of interatrial septum (IAS). As ASD can remain undiagnosed until adulthood, it is the most common congenital heart defect (CHD) in adults. In this study we aimed to evaluate demographics, clinical characteristics, survival and mortality predictors of adult ASD population.

Methods: All adult ASD patients regardless of treatment status who are followed by adult congenital heart disease (ACHD) outpatient of İstanbul University Cerrahpaşa Institute of Cardiology between 1980 and 2023 were included in this analysis.

Results: The study group consisted of 456 patients and most of them (71.3%) were female. The mean age was 48.4 ± 16.6 (min: 17, max: 98) years-old. The ASD type was ostium primum, secundum and other (sinus venosus or unroofed coronary sinus type) in 2.4%, 91.7% and 5.9% of patients, respectively. While 11 patients had ostium primum type ASD, just 2 patients had unroofed coronary sinus type defect. More than half of patients (51.5%) had undergone surgical closure and percutaneous closure was the treatment choice in 66 patients (14.5%). The most common rhythm was sinus rhythm in 81.4% patients and AF was the main rhythm in 10.7% of patients. Two-thirds of patients had either complete or incomplete right bundle branch block (RBBB). On echocardiography, the mean systolic pulmonary artery pressure (sPAP) was 37.4 ± 14.9 mmHg, the mean tricuspid annular plane systolic excursion (TAPSE) was 21.1 ± 3.8 mm. Less than half of patients (47.6%) had right heart catheterization (RHC) and hemodynamic data before closure of ASD. On RHC, the mPAP was 27.6 ± 12.2 mmHg, the mean PVR was 1.9 ± 3.5 WU, the mean right atrial pressure (RAP) was 11.2 ± 7.8 mmHg, the mean cardiac index was 2.5 ± 0.9 mL/ps/m^2, the mean pulmonary capillary wedge pressure (PCWP) was 12.4 ± 5.3 mmHg. The mortality rate was 9.2%. In univariate analysis, primum type ASD, surgical closure and medical follow-up without closure, AF, RV hypertrophy, right or left axis deviation, RBBB, age, sPAP, TAPSE, PVR, mPAP, RAP, CI, PCWP were predictors of mortality. Cox regression analysis revealed age, TAPSE, PVR and PCWP as the predictors of mortality (Table 1). Figure 1 is showing Kaplan Meier survival analysis according to ASD type (54.5% for primum ASD, 91.8% for secundum ASD, and 88.9% for others, p=0.000).

Conclusions: In this long-term follow-up analysis of adult ASD patients, age, TAPSE, PVR and PCWP were found independent predictors of mortality. The mortality rate was 9.2% in almost half a century follow-up. Patients with primum ASD had worse outcome than the patients with other ASD types.

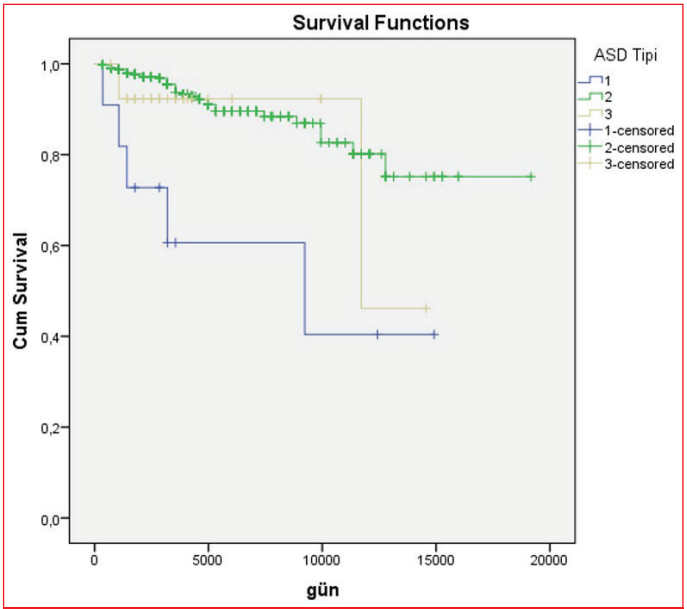

Figure 1. Figure 1 is showing Kaplan Meier survival plots of different ASD types.

Pulmonary Hypertension / Pulmonary Vascular Disease

OP-040

The effect of low diffusion capacity for carbon monoxide on prognosis in patients with pulmonary hypertension

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Background and Aim: Recently, a new lung phenotype was defined in patients with idiopathic pulmonary arterial hypertension (IPAH) which is characterized by a low diffusion capacity for carbon monoxide (DLCO) and smoking history. IPAH patients with predicted DLCO of less than 45% had a similar prognosis with Group 3 PAH and had a worse prognosis compared with classical IPAH. We studied the prognosis of patients with Group 1 PAH and chronic thromboembolic pulmonary hypertension (CTEPH) with low predicted DLCO.

Methods: Patients with treatment-naive IPAH, congenital heart disease-associated PAH, and CTEPH were included in this study. Patients with connective tissue disease-associated PAH were excluded. We compared survival in patients with predicted DLCO <45% and predicted DLCO >45%. Patients were followed up until January 31, 2023.

Results: We included 24 (39.3%) patients with IPAH, 6 (9.8%) patients with congenital heart disease-associated PAH, and 31 (50.8%) patients with CTEPH. The mean age of patients with group 1 PAH was 54 ± 14 years (63.3% female), and the mean age of patients with CTEPH was 57 ± 16 years (67.7%

female). The median follow-up time was 2.8 (interquartile range, 0.6-4.8) years, and 15 patients (50%) in group 1 PAH and 11 patients (35.5%) in the CTEPH group died in this period. Survival was not significantly different between predicted DLCO <45% and predicted DLCO >45% groups for group 1 PAH (long-rank p=0.056) (Figure 1). In contrast, survival was significantly lower in patients with predicted DLCO <45% in patients with CTEPH (Log-rank p<0.027) (Figure 2). Survival rates for patients with predicted DLCO <45% in group 1 PAH were as follows: 69% (1 year) and 59% (3 years), 37% (5 years), and 18% (8 years) respectively. Survival rates for patients with predicted DLCO<45% in the CTEPH group were as follows: 58% (1 year) and 44% (3 years), and 29% (5 years), respectively.

Conclusions: The predicted DLCO <45% is associated with a worse prognosis in patients with CTEPH. The effect of this observation on decision-making in patients with CTEPH needs further research.

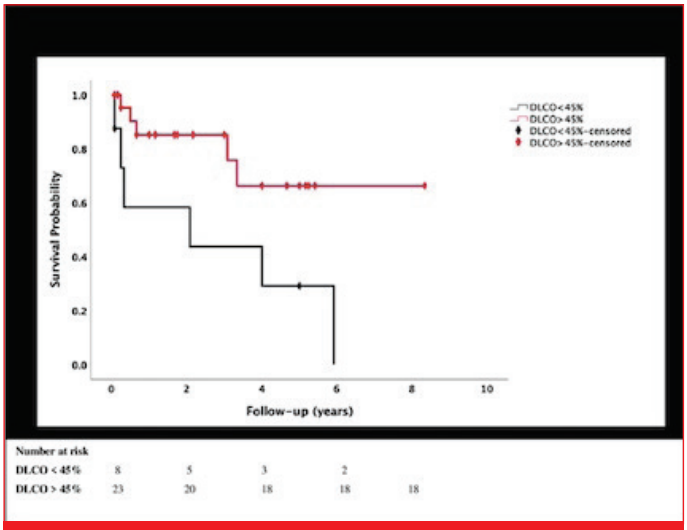

Figure 1. Kaplan-Meier survival curves for predicted DLCO <45% and DLCO >45% groups in CTEPH patients.

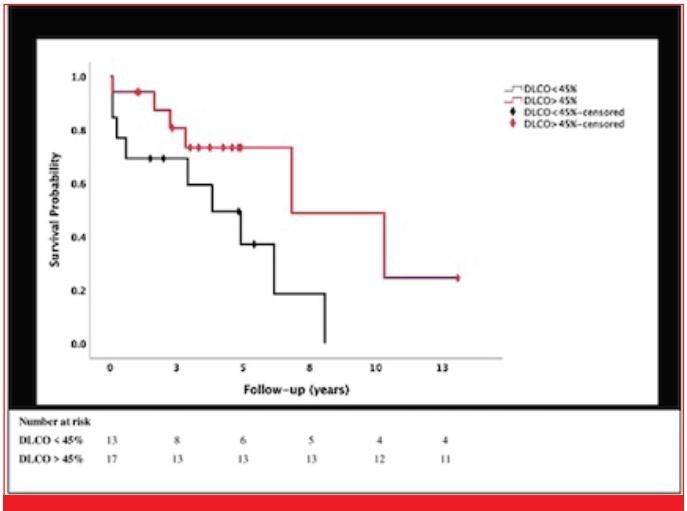

Figure 2. Kaplan-Meier survival curves for predicted DLCO <45% and DLCO >45% groups in group 1 PAH patients.

Pulmonary Hypertension / Pulmonary Vascular Disease

OP-041

A scoring system for prediction of HITT in pulmonary embolism patients: A single-center study

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Background and Aim: Heparin-induced thrombocytopenia (HIT) is an immune system disorder that arises after exposure to either unfractionated (UFH) or low-molecular-weight heparin (LMWH), manifesting as potentially life-threatening thrombocytopenia and thrombosis. The incidence of HIT varies between 1% to 5% in patients receiving heparin. The decrease in platelet count begins 5-14 days after starting heparin due to the effect of PF4/H antibodies. Thrombosis also occurs in 20-50% of HIT patients. The prothrombotic state caused by HIT can significantly impact prognosis, posing a 30-fold higher thrombosis risk compared to the general population, with a mortality risk ranging from 17% to 30% in thrombosis cases. The most important diagnostic feature of HIT is an absolute thrombocytopenia and the time of onset of thrombocytopenia, occurrence of thrombosis with thrombocytopenia, and the exclusion of criteria that could lead to other causes of thrombocytopenia. Drugs, infections, cancer, and the presence of mechanical valves can cause thrombocytopenia. Therefore, Warkentin and colleagues developed the 4T scoring system in 2006 to define HIT. Total score ≥6 indicates a high probability of HIT, 4-5 points indicate moderate probability, and ≤3 points indicate low probability (Table 1). Pulmonary embolism (PE) is a cardiovascular disease characterized by acute thrombosis of pulmonary artery branches with a mortality of 5-6% within the first 30 days. Treatment for PE involves the use of thrombolytic and anticoagulant drugs during the acute phase. Heparin commonly used in the acute and chronic phases of these patients. The development of HIT in these patients can increase the already high mortality associated with PE. In our study, we aimed to develop a model predicting HIT development in PTE patients to reduce morbidity and mortality associated with HIT. To the best of our knowledge, there are no studies on this topic in the literature.

Methods: Data from 324 hospitalized PE patients were retrospectively analyzed. The 4T score was calculated for each patient. A total of 28 patients with a 4T score of 6 or higher and/or HIT diagnosis by a certified hematologist were identified. In univariate analysis, 11 parameters were found to correlate with HIT development. To prevent overfitting due to the small sample size, a lasso regression analysis was used to develop a predictive model for HIT development in PE patients using the most efficient 6 parameters (Table 2).

Results: The modeling (Figure 1) statistically significantly predicted HIT development in PE patients (Figure 2).

Conclusions: PE is the third most common cardiovascular syndrome worldwide after myocardial infarction and stroke, and thrombosis is responsible for its pathophysiology. The

development of HIT due to the use of heparin and its derivatives can paradoxically lead to an increase in thrombosis and associated mortality. With our model, we aimed to detect patients at high risk of developing HIT early and thereby reduce mortality and morbidity.

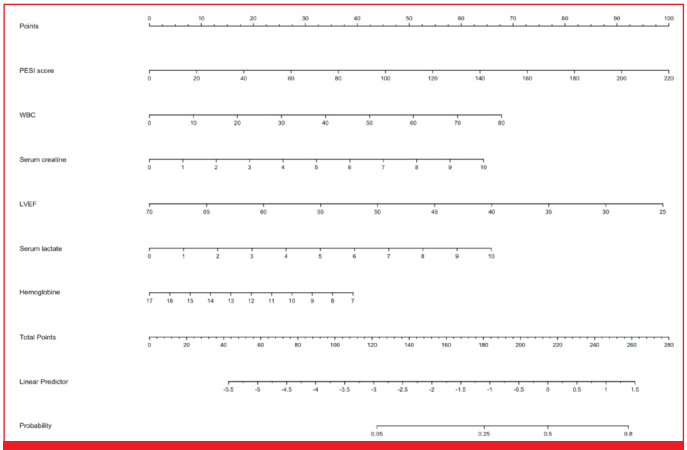

Figure 1.

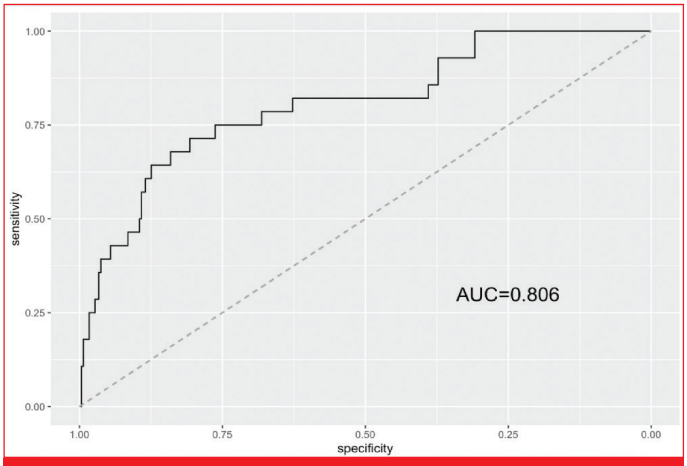

Figure 2.

Table 2.

Variables	OR	95% CI	p
PESI score (78-122)	1.894	1.056-3.401	0.032
WBC (8-13)	1.145	1.011-1.297	0.034
Serum creatinine (0.87-1.15)	1.059	1.005-1.116	0.032
LVEF (25-67)	0.052	0.009-0.309	0.001
Serum lactate (1.2-2.4)	1.287	0.953-1.737	0.09
Hemoglobine (10-13)	0.686	0.383-1.232	0.208

Multivariable logistic regression analysis for detecting HITT.

Pulmonary Hypertension / Pulmonary Vascular Disease

OP-042

Nursing diagnosis for pulmonary hypertension patients in cardiology intensive care unit

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Background and Aim: Pulmonary hypertension (PH) is a disease characterized by progressive vascular changes such as proliferation and vasoconstriction in the pulmonary arteries. Despite all the medical advances, the mortality rate is still high. Echocardiography and right heart catheterization are the two most important modalities for the diagnosis of PH. In the most recent guideline, the diagnosis is made with a resting mean pulmonary artery pressure (mPAP) >20 mmHg in right heart catheterization. (2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension) PH is clinically divided into 5 classes:

I: Pulmonary arterial hypertension

II: PH due to left heart diseases

III: PH due to lung diseases and/or hypoxia

Table 1.

Category	2 points	1 point	0 points
1. Thrombocytopenia	Platelet count fall >50% and platelet rare ≥20 x 10^9/L	Platelet count fall 30%-50% or platelet rare 10^9 L x 10^9/L	Platelet count fall <30% or platelet rare < 10 x 10^9/L
2. Timing of platelet count fall	Clear onset between days 5 and 10 or platelet fall ≤1 day (prior heparin exposure within 30 days)	Consistent with days 5-10 fall, but not clear (e.g. missing platelet counts) or onset after day 10 or fall ≤1 day (prior heparin exposure 30-100 days ago)	Platelet count fall <4 days without recent heparin exposure
3. Thrombosis or other sequelae	New thrombosis (confirmed) or skin necrosis at heparin injection sites or acute systemic reaction after intravenous heparin bolus	Progressive or recurrent thrombosis or nonnecrotizing (erythematous) skin lesions or suspected thrombosis (not proven)	None
4. Other causes for thrombocytopenia	None apparent	Possible	Definite

4Ts scoring system Total score of ≥6 points, high probability of HIT; 4-5 points, intermediate probability of HIT; ≤3 points, low probability of HIT.

IV: Chronic thromboembolic PH

V: PH with unclear mechanisms or caused by multiple factors

Methods: To provide treatment and care of patients diagnosed with PH in the intensive care unit, to reduce the mortality and morbidity rates of patients, to improve their living conditions and to establish nursing diagnoses.

Results: Intensive care nurses have a coordination role between the patient and the physician in the care of PH patients. Nurse; it has an important role in drug compliance, nutrition, exercise, mental health and palliative care.

Conclusions: Objectives of nursing care; maximizing cardiopulmonary functions to evaluate the effects of pharmacological treatment, maintaining skin integrity to educate the patient and his/her family about heart failure.

Nursing care:

1. Ineffective breathing: Close sPO_2 monitoring is required in patients with PH who have difficulty in breathing. In cases such as dyspnea, cyanosis, tachypnea, wheezing, hypoventilation, oxygen support is applied according to the doctor's request (nasal, nebula, mask, non-invasive ventilation). The patient is placed in the semifowler or fowler position.

2. Evaluation and intervention: Evaluate right heart insufficiency, intervene appropriately evaluating response to medications, observing and managing side effects helps with critical thinking, problem solving, and application of patient needs.

3. Risk of decreased cardiac output: Decrease of the blood pumped from the heart to meet the tissue requirement and causing impaired heart function; the patient is observed in terms of dyspnea, orthopnea, tachycardia, restlessness, change in mental status, capillary filling time. Vital signs are followed. The indication and symptoms of hypoxemia are given the appropriate position. Fluid follow-up, amount of fluid taken and output, signs of dehydration are evaluated.

4. Risk of bleeding: Depending on the patient's use of anticoagulant medication, high INR, aPTT, PT value and a tendency to bleeding: Bleeding signs and symptoms are followed (melena, hematemesis, ecchymosis, GIS bleeding, petechiae). Platelet count, htc, hb, plt, aptt are monitored. If any change is detected, the physician is notified.

Pulmonary Hypertension / Pulmonary Vascular Disease

OP-043

Could impedance cardiography be a non-invasive alternative method of measuring cardiac output in patients with pulmonary hypertension?

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Background and Aim: Current guidelines recommend right heart catheterization (RHC) and invasive cardiac output (CO) measurement for the diagnosis and clinical follow-up in pulmonary hypertension (PH). The aim of our study was to compare non-invasive impedance cardiography (ICG) with eFick and thermodilution (TD) techniques for cardiac index (CI) measurements and mortality prediction in patients with PH.

Methods: Between 2008-2018, 284 RHCs were performed for the diagnosis for PH in our institution. Among these patients, 215 patients with a mean pulmonary artery pressure (mPAP) of >25 mmHg in whom at least two methods were used for CO measurement were retrospectively enrolled. Patients were evaluated with Pearson correlation in 3 groups according to the used methods, eFick and TD (Group 1), Fick and ICG (Group 2), TD and ICG (Group 3). Bland-Altman Analysis was conducted to test the interchangeability of the tests with each other. We also compared the predictive power of CI measured by different methods for 1-year overall mortality and cardiovascular hospitalizations.

Results: eFick vs. ICG had moderate correlation (r=0.390 p=0.01), eFick vs. TD and TD vs. ICG had good correlations (r=0.634 p<0.001, and r=0.534 p<0.01, respectively). The mean difference (bias) between eFick vs. ICG, ICG vs. TD, and eFick vs. TD were 0.6 mL/min, 0.47 mL/min, and -0.2 mL/min respectively, but limits of agreement were wide. CI of <2.5 L/min/m^2 measured by TD in both groups significantly predicted the 1-year mortality. However, CI measured by eFick method failed to predict mortality either in the group compared with TD or in Group 3 compared to ICG. CI measured by ICG, significantly predicted 1-year mortality compared to eFick (p=0.02) (Figure 1).

Conclusions: Our single center real-life data showed that for CO and CI measurements, ICG provides moderate correlation with TD and fair with eFick methods. Bland-Altman analysis showed that these tests were not interchangeable. Moreover, TD appeared superior to both eFick and ICG, while ICG was even better than eFick in predicting 1-year adverse events, including total mortality and hospitalizations, in patients with PH. Therefore, hemodynamic assessment with ICG might be used as a non-invasive, low-cost, easy prognostic predictor of survival in patients with PH.

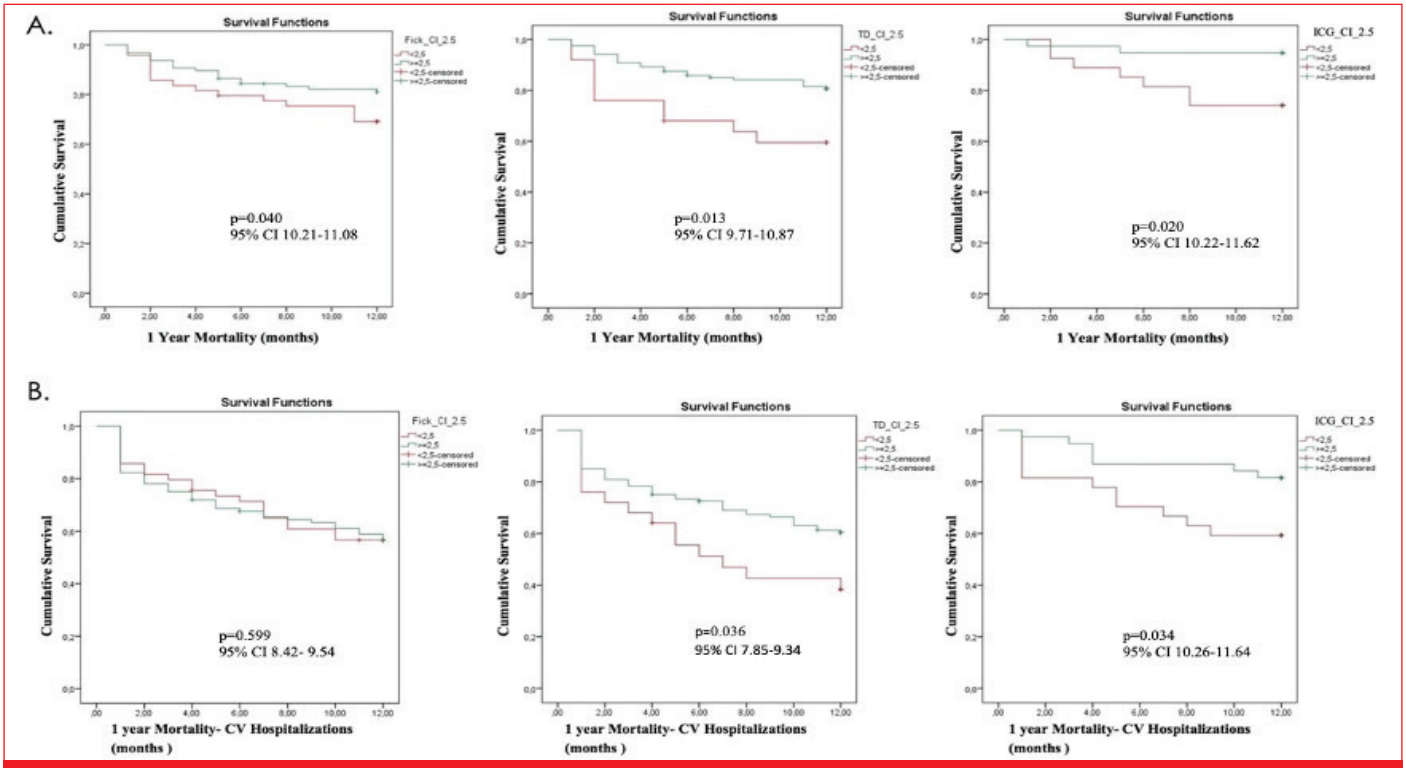

Figure 1. Kaplan-Meier analysis for 1-year mortality (A) and 1- year mortality and cardiovascular hospitalizations (B) according to the 3 different methods.
CI, cardiac index; CV: Cardiovascular; ICG, impedance cardiography; TD, thermodilution

Pulmonary Hypertension / Pulmonary Vascular Disease

OP-044

The relationship between pulmonary artery pulsatility index and vasoreactivity test

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Background and Aim: The pulmonary artery pulsatility index is a hemodynamic parameter that provides insight into the function of the pulmonary circulation. The vasoreactivity test, also known as the acute vasodilator challenge, is a diagnostic procedure performed to evaluate the responsiveness of the pulmonary vasculature to vasodilator medications. The PAPi and the vasoreactivity test are interconnected through their implications for pulmonary vascular function. There is no study on the relationship between pulmonary artery pulsatility index and vasoreactivity test.

Methods: Patients who underwent right heart catheterization and subsequent vasoreactivity test in our center between 2020 and 2022 were included in the study. PAPi is calculated by measuring the difference between the systolic and diastolic pressures in the pulmonary artery and dividing it by the mean pulmonary artery pressure. PAPi = (systolic pressure-diastolic pressure) / right atrial pressure. Vasoreactivity test was done with inhaled iloprost.

Results: A total of 46 patients were evaluated. PAPi value was found to be statistically significantly higher in the group with positive vasoreactivity test. When the baseline demographic, echocardiographic and laboratory findings were evaluated between the two groups, only the eGFR value was higher in the group with positive vasoreactivity test.

Conclusions: PAPi and the vasoreactivity test are interconnected in assessing the function of the pulmonary vasculature. The PAPi reflects the pulsatile nature of the pulmonary artery pressure waveform, while the vasoreactivity test evaluates the responsiveness of the pulmonary arteries to vasodilator treatment. Both these parameters contribute to a comprehensive understanding of pulmonary vascular health and guide the management of conditions like pulmonary arterial hypertension.

Table 1. Results of the study

	VR negative (n=38)	VR positive (n=8)	P value
Age, years	61.31 ± 16.96	52.75 ± 20.21	0.215
Male sex, n (%)	2 (25)	13 (34.2)	0.648
eGFR, ml/min	72.13 ± 25.65	101.85 ± 25.37	0.007
Hemoglobin, g/dL	12.76 ± 2.50	12.75 ± 1.38	0.989
TAPSE, mm	19.25 ± 4.06	21.50 ± 4.14	0.168
PCWP, mmHg	9.83 ± 3.65	11.83 ± 4.78	0.337
Cardiac index,	2.00 ± 0.01	2.18 ± 0.58	0.673
PVR, WU	7.96 ± 4.72	8.40 ± 4.03	0.848
SVR, WU	21.34 ± 7.89	23.00 ± 3.39	0.650
MVO2 sat, %	56.66 ± 14.36	41.00 ± 15.55	0.162
PAPi	5.31 ± 3.19	8.90 ± 3.29	0.006

Congenital Heart Diseases

OP-045

Demographics and clinical charactersitics of adult operated ToF patients

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Background and Aim: Tetralogy of fallot (ToF) is characterized by the following four features: a nonrestrictive VSD; overriding aorta (but <50%); infundibular, valvular, supravalvular RVOTO and/or branch PA stenosis; and consequent RV hypertrophy (RVH). It is the most common cyanotic congenital heart defect (CHD). Most of the patients will not reach into adulthood without surgery depending on the degree of RVOTO. So most of adult ToF patients had history of total correction surgery. Major consequences of adult operated ToF patients are pulmonary regurgitation (PR), residual RVOTO, residual VSD, aortic complication and RV and LV dysfunction. We aimed to describe demographic, clinical, echocardiographic and CMR characteristics of adult ToF patients.

Methods: Adults mostly operated ToF patients are followed at ACHD outpatient of our University Institute of Cardiology were analyzed.

Results: There was 91 adult ToF patients (86 operated, 5 non-operated) and almost half of them was male (49.5% vs. 50.5%). Seventeen of 46 women (18.7%) had a history of delivery. The mean age was 36.3 ± 9.9 (IQR: 19-71 years). The functional capacity was I, II, and III in 64.8%, 28.6% and 6.6%, respectively. On ECG, the mean QRS duration was 140.6 ± 26.5 msec. While most of patients (93.4%) was in sinus rhythm, there was 4 patients with AF/Aflutter and 2 patients with pace rhythm. On Holter monitoring, 7 patients had NSVT, 12 patients had SVT. On echocardiography, the mean LVEF was 57.8 ± 6.9%, the mean RV diameter on parasternal long axis view was 31.8 ± 7.2 mm, and the mean TAPSE was 18.1 ± 3.0 mm. Patient with moderate-severe PR consisted 40.7% of whole study population. The mean PA and RVOT diameter on echocardiography were 30.6 ± 5.2 and 31.7 ± 9.6 mm. Table 1 is showing EF, RV and LV volumes and PR regurgitation volumes on CMR. Eighteen patients had fibrosis on CMR especially in RVOT.

Conclusions: Here we present demographics, clinical, ECG, echocardiographic and CMR characteristics of adult mostly operated ToF patients. Severe PR is one of the major concerns which necessitates re-intervention. Moderate to severe PR was found 40% in our study population. CMR is the gold standard imaging modality in these patient population to evaluate of PR severity and RV volumes which determines prognosis and re-intervention. RV fibrosis on CMR also should be investigated as fibrosis is a negative predictor of survival.

Table 1. EF and volumes derived from CMR

Parameter	Mean	Std deviation
LVEF, %	51.1	9.3
RVEF, %	41.6	9.7
RVEDVI, mL/m^2	161.5	61.9
RVESVI, mL/m^2	96.4	44.7
PR RVI, mL/m^2	49.1	36.2

Pulmonary Hypertension / Pulmonary Vascular Disease

OP-046

The impact of the new hemodynamic definition on the prevalence of pre-capillary pulmonary hypertension

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Background and Aim: The current 2022 ESC pulmonary hypertension (PH) guideline suggests mean pulmonary artery pressure (mPAP) >20 mmHg, pulmonary arterial wedge pressure (PAWP) <15 mmHg, and pulmonary vascular resistance (PVR) >2 WU as new hemodynamic definition of pre-capillary PH. We aimed to investigate the impact of the new ESC 2022 PH definition on number of pre-capillary PH patients.

Methods: The results of right heart catheterization (RHC) performed with various clinical indications between 2017 and 2023 were analyzed. Both 2015 and 2022 European Society of Cardiology (ESC)/European Respiratory Society (ERS) PH guidelines were used to identify PH patients.

Results: One hundred twenty-three RHC procedures were performed in a 6-year period. Most of them were female (72.4%). The clinical indications for RHC were suspicion of congenital heart disease associated pulmonary arterial hypertension (APAH-CHD) in 32.5% of patients, idiopathic PAH in 43.9% of patients, PH due to left heart disease in 17.9% of patients and chronic thromboembolic pulmonary hypertension (CTEPH) in 5.7% of patients. The mean age was 53.1 ± 16.6 years. The RHC results revealed a mean PAP of 35.4 ± 17.8 mmHg, PAWP of 13.3 ± 6.0 mmHg, and PVR of 5.2 ± 6.3 WU. While 20% of patients had no PH, almost 10% of patients had pre-capillary PH according to new 2022 ESC/ERS guideline hemodynamic PH definition criteria which was not able to be classified as pre-capillary PH according to previous ESC/ERS guideline. There was 8.1% patients had combined pre and post-capillary PH according to current guideline. Finally 15.4% of patients had undefined PH, defined as mPAP >20 mmHg, but PVR <2 WU, which was a novel definition for the first-time mentioned in 2022 guideline.

Conclusions: Current ESC/ERS guidelines for the diagnosis and treatment of PH will increase almost 10% our PAH patients population who are going to be treated with PAH specific drug therapy.

Table 1. Table 1 is showing the ratio of PH patients according to previous and current guideline

Definition	Patients, N (%)
Current pre-capillary PH (PVR >2 WU, mPAP >20 mmHg and PCWP ≤15 mmHg)	12 (9.8%)
Previous pre-capillary PH (PVR >3 WU, mPAP >25 mmHg and PCWP ≤15 mmHg)	35 (28.5%)
No PH (mPAP <20 mmHg)	25 (20.3%)
Current combined pre and post-capillary PH (PVR >2WU, mPAP >20 mmHg and PCWP >15mmHg)	10 (8.1%)
Previous combined pre and post-capillary PH (PVR >3WU, mPAP >25 mmHg and PCWP >15 mmHg)	20 (16,3%)
Previous isolated post-capillary PH (PVR <3WU, mPAP >20 mmHg and PCWP >15mmHg)	2 (1.6%)
Undefined PH (mPAP >20 mmHg, but PVR <2WU)	19 (15.4%)

Abbreviations: PAP; pulmonary artery pressure, PCWP; pulmonary capillary wedge pressure, PH; pulmonary hypertension, PVR; pulmonary vascular resistance.

Pulmonary Hypertension / Pulmonary Vascular Disease

OP-047

Low dose thrombolytic therapy versus unfractionated heparin in patients with intermediate-high risk pulmonary embolism

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Background and Aim: The patients with intermediate-high risk pulmonary embolism (PE) who have acute right ventricular dysfunction and myocardial injury without overt hemodynamic compromise may be candidates for thrombolytic therapy (TT). In this study, we aimed to compare the clinical outcomes of low-dose prolonged TT and unfractioned heparin (UFH) in intermediate-high risk PE patients.

Methods: This study enrolled 83 [female: 45 (54.2%), mean age: 70.07 ± 10.7 years] retrospectively evaluated patients with the diagnosis of acute PE who were treated with low-dose and slow-infusion of TT or UFH. The primary outcomes of the study was defined as a combination of death from any cause and hemodynamic decompensation, and severe or life-threatening bleeding. Secondary endpoints were recurrent PE, pulmonary hypertension, and moderate bleeding.

Results: The initial management strategy of intermediate-high risk PE was TT in 41 (49.4%) patients and UFH in 42 (50.6%) cases. Low-dose prolonged TT was successful in all patients. While the frequency of hypotension decreased significantly after TT (22 vs. 0%, p<0.001), it did not decrease after UFH (2.4 vs. 7.1%, p=0.625). The proportion of hemodynamic decompensation was significantly lower in the TT group (0 vs. 11.9%, p=0.029). The rate of secondary endpoints was significantly higher in UFH group (2.4 vs. 19%, p=0.016). Moreover, prevalance of pulmonary hypertension was significantly higher in UFH group (0 vs. 19%, p=0.003).

Conclusions: Prolonged TT regimen with low dose, slow infusion of t-PA was found to be associated with a lower risk of hemodynamic decompensation and pulmonary hypertension in patients with acute intermediate-high-risk PE compared to UFH.

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

Parameters	Low dose TT group (n=41)	UFH group (n=42)	p
Age, years	70.8 ± 10.2	69.4 ± 11.2	0.541
Gender (female), n (%)	22 (53.7)	23 (54.8)	0.920
Hypertension, n (%)	30 (73.2)	28 (66.7)	0.518
Diabetus mellitus, n (%)	9 (22.0)	14 (33.3)	0.247
COPD, n (%)	8 (19.5)	12 (28.6)	0.335
Surgical history, n (%)			
Arthroplasty	3 (7.3)	0	0.116
Femur fixation	1 (2.4)	3 (7.1)	0.317
Gastrectomy	0	1 (2.4)	0.506
Hysterectomy	1 (2.4)	0	0.494
Prior DVT, n (%)	23 (56.1)	23 (54.8)	0.903
Malignancy, n (%)	2 (4.9)	1 (2.4)	0.491
Clinical presentation, n (%)			
Dyspnea	41 (100.0)	42 (100.0)	
Tachypnea	16 (39.0)	15 (35.7)	0.755
Syncope	5 (12.2)	5 (11.9)	0.615
Chest pain	4 (9.8)	4 (9.5)	0.630
Troponin (ng/mL)	1.379 ± 0.587	1.251 ± 0.430	0.259
HASBLED score, n (%)			
1	21 (51.2)	23 (54.8)	0.746
2	20 (48.8)	18 (42.9)	0.588
Heart rate (bpm)	111 (109-117)	110.5 (109-117)	0.497
O_2 saturation, %	87 (86-88)	87 (86-88)	0.952
PTE location, n (%)			
Bilateral	31 (75.6)	36 (85.7)	0.243
Saddle	5 (12.2)	5 (11.9)	0.615
MPA	5 (12.2)	1 (2.4)	0.095
LVEF, %	60 (58-60)	60 (58-62)	0.509
RV/LV ratio	1.2 (1.0-1.2)	1.13 (1.0-1.2)	0.625
TAPSE, cm	1.6 (1.5-1.7)	1.6 (1.5-1.7)	0.585
SPAP, mmHg	53 (43-60)	41.5 (37-45)	<0.001
PESI score	112 (108-121)	111.5 (105-120)	0.565
PESI class, n (%)			
3	9 (22.0)	12 (28.6)	0.488
4	32 (78.0)	30 (71.4)	

COPD: Chronic obstructive pulmonary disease, DVT: Deep vein thrombosis, LV: Left ventricle, LVEF: Left ventricular ejection fraction, MPA: Main pulmonary artery, PESI: Pulmonary embolism severity index, PTE: Pulmonary thromboembolism, RV: Right ventricle, SPAP: Systolic pulmonary artery pressure, TAPSE: Tricuspid annular plane systolic excursion [Continuous variables with normal distribution were expressed as mean ± standard deviation and continuous variables without normal distribution were expressed as median (25^{th}-75^{th} percentiles)]

Table 2. The list of 3-month clinical outcomes per study group

Parameters	TT group (n=41)	UFH group (n=42)	p
Primary endpoint, n (%)	1 (2.4)	5 (11.9)	0.106
All-cause death	0 (0)	2 (4.8)	0.253
Hemodynamic decompensation	0 (0)	5 (11.9)	0.029
Severe or life-threatening bleeding	1 (2.4)	0 (0)	0.494
Secondary endpoint, n (%)	1 (2.4)	8 (19.0)	0.016
Recurrent pulmonary embolism	0 (0)	2 (4.8)	0.253
Pulmonary hypertension (≥40 mmHg on echocardiography)	0 (0)	8 (19.0)	0.003
Moderate bleeding	1 (2.4)	0 (0)	0.494
Minor bleeding, n (%)	5 (12.2)	5 (11.9)	0.615

TT: Thrombolytic therapy, UFH: Unfractioned heparin.

Table 3. Comparison of pre- and post-treatmant echocardiographic and parameters according to treatment strategy

	TT Group			UFH Group		
	Baseline	3th month	P value	Baseline	3th month	P value
Heart rate (bpm)	111 (109-118)	80 (77.5-86)	<0.001	110.5 (109-117)	80 (75-82.5)	<0.001
O_2 Saturation	87 (86-88)	96 (95-96)	<0.001	87 (85.75-88)	94 (92-95)	<0.001
Tachypnea, n(%)	16 (39.0)	0 (0)	<0.001	15 (35.7)	4 (9.5)	0.013
Hypotension, n(%)	9 (22.0)	0 (0)	<0.001	1 (2.4)	3 (7.1)	0.625
RV/LV ratio	1.2 (1.0-1.2)	0.66 (0.63-0.70)	<0.001	1.1 (1.0-1.2)	0.71 (0.65-0.84)	<0.001
SPAP, mmHg	53 (43-60)	24 (23-25)	<0.001	41.5 (37-45)	32 (29.5-37.25)	<0.001
TAPSE, mm	16 (15-17)	24 (21.5-25)	<0.001	16 (15-17.1)	20.5 (19-24)	<0.001

Abbrevetion: LV: Left ventricle; RV: Right ventricle, SPAP: Systolic pulmonary artery pressure, TAPSE: Tricuspid Annular Plane Systolic Excursion, TT: Thrombolytic therapy, UFH: Unfractioned heparin

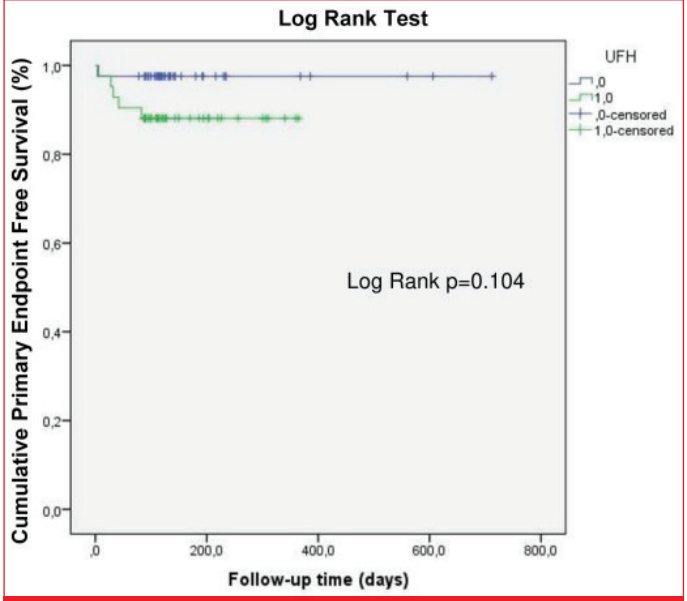

Figure 1. Kaplan-Meier survival analysis indicates that short-term cumulative primary end-points free survival ratio was not found to be significantly decreased in patients with intermediate-high risk PE. UFH: Unfractionated Heparin.

Pulmonary Hypertension / Pulmonary Vascular Disease

OP-048

Detection of pulmonary hemodynamics using pulmonary tomography based on artificial intelligence in patients with pulmonary hypertension

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Background and Aim: Pulmonary hypertension (PH) is a progressive condition characterized by high blood pressure in the pulmonary vasculature, leading to slow blood flow and increased pressure due to vascular narrowing and stiffening. Early detection of PH is crucial to prevent complications such as heart failure. Therefore, this study proposes a novel automatic classification model based on deep feature extraction to achieve early and accurate diagnosis of PH.

Methods: In this study, we collected CT images for PH detection and this dataset contains 807 CT images. We developed a new automatic classification architecture, called EfDenseNet, for PH detection and tested it using the collected data. We utilized pre-trained deep network architectures, namely EfficientNetb0 and DenseNet201, as feature extractors. To enhance the performance of the feature extractors, we segmented the CT images into patches and extracted deep features from both the patches and the original image. The generated features from each patch has been merged. The presented EfDenseNet generates four feature vectors by deploying the feature extraction layers (fully connected layer and global average pooling layers) of the EfficientNetb0 and DenseNet201. We used Neighborhood Component Analysis (NCA), ReliefF, and Chi-Square (Chi2) to select the most informative features from the feature vectors, resulting in 45 different selected feature vectors. In the classification phase, we employed support vector machine (SVM) and k-nearest neighbor (kNN) algorithms to classify the features. Finally, we applied a mode function-based iterative majority voting to the classification results to obtain generalized classification results.

Results: The proposed self-organized EfDenseNet architecture produced 90 classification results and 178 voted results. We selected the best result from the 178 voted results and achieved the highest classification accuracy for all four classes. We validated all results using k-fold cross-validation and obtained an accuracy of 97.27% on the new PH dataset collected by us, demonstrating the high classification performance of our proposed method.

Conclusions: Our proposed automatic classification model based on deep feature extraction achieved accurate and early diagnosis of PH with high classification performance. The results of this study suggest that the proposed self-organized EfDenseNet architecture is a promising approach for PH classification. Additionally, our study gives us ideas about which patient to refer to right heart catheterization.

Table 1. Characteristics of the gatheredcollected thorax CT angiography dataset

No	Group	Number of Images
1	Group 1 (20≤mPAP≤25)	80
2	Group 2 (25≤mPAP≤30)	130
3	Group 3 (mPAP>30)	387
4	Healthy	210
Total		807

Table 2. Demographic characteristics of the gathered CT image dataset

Group	Number of participants	Age	Female	Male
Group 1	43	39.6±4.5	21	22
Group 2	65	41.3±5.3	39	26
Group 3	91	45.7±7.5	55	36
Healthy	114	42.7±6.1	68	46
Overall	313	42.3±5.9	183	130

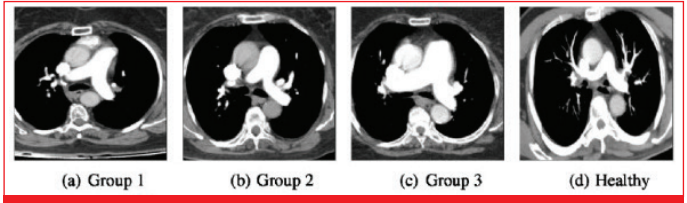

Figure 1. The sample CT images of the gathered CT image dataset.

Table 3. Performance of EfDenseNet archietecture on PH dataset (%)

Class	Accuracy	UAR	UAP	F1
Group 1	-	91.25	92.41	91.82
Group 2	-	94.62	96.85	95.72
Group 3	-	98.45	97.94	98.20
Healthy	-	99.05	98.11	98.58
Average	97.27	95.84	96.33	96.08

*UAR=Unweighted average recall, UAP=Unweighted average precision

The developed EfDenseNet model with self-organized architecture achieved 97.27% accuracy on the dataset. Testing was performed by applying a 10-fold CV strategy. In addition, when the other performance metrics are analyzed, it is seen that the proposed method achieves more than 95% in all metrics.

Figure 2. Confusion matrix of the proposed model.

Heart Failure

OP-049

EUCOR-CMP trial; comparison of clinical characteristics and survival between ischemic and non-ischemic heart failure patients with reduced ejection fraction: An observational study

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Background and Aim: This study aims to thoroughly scrutinize and contrast the clinical disparities and survival determinants among patients who suffer from ischemic cardiomyopathy (ICM) and non-ischemic cardiomyopathy (NICM).

Methods: This study is a retrospective observational analysis carried out at a single center. It investigated a total of 1614 patients who were diagnosed with outpatient heart failure between 2008 and 2022. Among these patients, 910 (56%) were diagnosed with ischemic cardiomyopathy (ICM), and 704 (44%) were diagnosed with non-ischemic cardiomyopathy (NICM). The primary endpoint of the study was a composite of all-cause mortality, left ventricular assist device (LVAD) implantation, and heart transplantation.

Results: The average age was 46 ± 12 years, and ICM patients were older (48.3 ± 12.0, 43.0 ± 13, respectiveley, p<0.0001). Additionally, ICM patients exhibited a higher incidence of comorbidities related to cardiovascular disease. Out of the patients in the group, the majority-81.4% were taking ACE

inhibitors or ARBs, while only 8% were taking ARNIs. Almost all patients-98% were taking beta-blockers, and 92.5% were taking MRA. Only a small percentage-14.6% were using SGLT2-i. No significant differences were found between the groups. In 30.2% of the patients, an ICD was implanted, and a CRT-D was implanted in 8.7%. The ICM patients had a higher rate of ICD implantation, while the NICM group had a higher rate of CRT-D implantation (p<0.0001). The average LVEF was 28.0 ± 10.9% with no notable variations between the groups. 60.3% of the patients had moderate to severe mitral regurgitation (MR), which was more prevalent in the ICM patients (p<0.0001). The TAPSE, which is an indicator of right ventricular function, was found to be worse in the ICM group. Over a median follow-up period of 6.2 years, 446 patients (27.6%) reached the primary endpoint, with 253 (27.7%) being ICM patients and 193 (27.4%) being NICM patients. The multivariate analysis revealed that ischemic etiology independently predicted the primary endpoint. Additionally, advanced age, low blood pressure, NYHA functional class 3-4, moderate or severe MR, and increasing NT-pro-BNP levels were all poor prognostic indicators for all heart failure patients.

Conclusions: Our study highlights the importance of considering ischemic etiology when managing HFrEF patients. Further research can enhance the prognosis of this patient group by gaining a better understanding of the mechanisms between ischemic etiology and survival and creating more efficient treatment options.

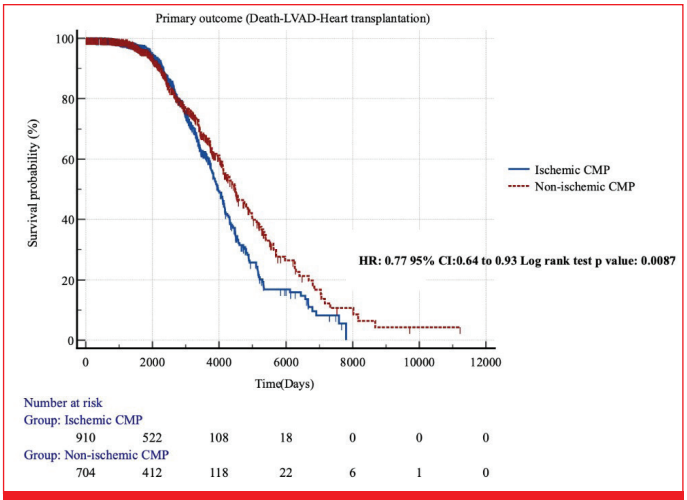

Figure 1. Primary endpoint free survival according to ischemic or non-ischemic etiology of heart failure.
LVAD: Left ventricular assist device, CMP: Cardiomyopathy.

Table 1. Baseline characteristics

	Total	Ischemic CMP	Non-ischemic CMP	p
Age, years	46.0 ± 12.8	48.3 ± 12.0	43.0 ± 13.0	<0.0001
Male, %	72.8	80.9	62.3	<0.0001
BMI, kg/m^2	26.5 ± 3.9	26.5 ± 4.0	26.5 ± 3.91	0.45
BSA, m^2	1.91 ± 0.18	1.91 ± 0.18	1.90 ± 0.18	0.47
SBP, mmHg	113.9 ± 18.3	113.8 ± 17.9	114.1 ± 18.7	0.41
DBP, mmHg	69.5 ± 11.9	69.0 ± 10.8	70.0 ± 13.0	0.056
NYHA FC				0.007
I	10.6	9.1	12.5	
II	39.7	36.9	43.2	
III	26.6	29	23.5	
IV	5.6	5.6	5.6	
Arterial hypertension, n (%)	32.2	38.5	24.0	<0.0001
Diabetes mellitus, n (%)	26.8	34.1	17.3	<0.0001
Dyslipidemia, n (%)	15.7	19.7	10.5	<0.0001
Smoking history, n (%)	36.5	46.7	23.3	<0.0001
Cerebrovascular event, n (%)	5.1	5.9	4.1	0.10
COPD, n (%)	5.7	7.2	3.6	0.002
Renal disease, n (%)	3.3	3.7	2.8	0.33
Chronic liver disease, n (%)	0.7	0.6	0.9	0.50
Peripherial arterial disease, n (%)	2.3	3.9	0.8	0.001
AF, n (%)	17.2	16.5	18.1	0.042

*Values are mean ± SD or n (%) p value <0.05. CPM: Cardiomyopathy, BMI: Body mass index, BSA: Body surface area, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, NYHA FC: New York Heart Association functional capasity, COPD: Chronic obstructive pulmonary disease, AF: Atrial fibrillation.

Table 1. Baseline characteristics (continue)

	Total	Ischemic CMP	Non-ischemic CMP	p
Intracardiac device, n (%)	51.2			<0.0001
Pacemaker, n (%)	11.5	14.4	7.0	
ICD n (%)	30.2	33.2	26.3	
CRT-D, n (%)	8.7	6.3	11.7	
Family history of heart failure, n (%)	12.4	6.5	20.1	<0.0001
NT-pro-BNP, pg/mL				0.080
ACEi or ARB, n (%)	81.4	82.3	80.3	0.29
ARNI, n (%)	8.0	7.2	9.0	0.18
Beta-blocker, n (%)	98.0	98.6	97.6	0.14
Aldosterone antagonist, n (%)	92.5	92	93.1	0.41
SGLT2-i, n (%)	14.6	14.0	15.5	0.40
Loop diuretic, n (%)	77.0	81.7	70.8	<0.0001
Anticoagulant, n (%)	28.3	28.9	27.6	0.59
Antiplatelet, n (%)	53.7	54.3	52.9	0.56
If channel blocker, n (%)	30.7	28.3	33.9	0.016
Digoxine, n (%)	17.7	16.2	19.7	0.065
Antihyperlipidemic, n (%)	32.0	46.6	13.2	<0.0001
Antiarrhytmic, n (%)	9.7	10.1	9.2	0.63
Primary end-point, n (%)	27.6	27.7	27.4	0.98
Follow-up time median IQR, days	2265	2255	2286	0.003
LVEDd, mm	62.7 ± 10.5	63.7 ± 9.4	61.4 ± 11.7	0.80
LVESd, mm	52.5 ± 12.5	53.5 ± 11.3	51.1 ± 13.8	0.89
LAd, mm	46.4 ± 7.6	47 ± 7.5	45.7 ± 7.8	<0.0001
LVEF, %	28.0 ± 10.9	26.9 ± 9.3	29.5 ± 12.4	0.27
Mitral regurgitation, n (%)				<0.0001
Mild	27.5	26.1	28.5	
Moderate	40.6	44.0	34.7	
Severe	19.7	19.0	20.1	
Aortic regurgitation, n (%)				0.24
Mild, %	17	18.3	15.2	
Moderate, %	5.7	5.4	6.1	
Severe, %	1.2	0.9	1.6	
Tricuspid regurgitation, n (%)				0.12
Mild, %	37.0	38.0	35.7	
Moderate, %	32.0	32.9	30.7	
Severe, %	15.0	14.9	15.1	
TAPSE, mm	17.1 ± 5.0	16.8 ± 4.9	17.6 ± 5.0	<0.0001
RVsm (TDI), m/sec	10.1 ± 2.72	9.9 ± 2.67	10.4 ± 2.76	<0.0001
TRV, m/sec	2.84 ± 0.54	2.89 ± 0.55	2.78 ± 0.55	<0.0001
SPAP, mmHg	35.9 ± 15.4	37.3 ± 15.8	34.1 ± 14.7	<0.0001

*Values are mean ± SD or n (%) p value <0.05. CMP: Cardiomyopathy, ICD: Intracardiac device, CRT-D: Cardiac resynchronization therapy with defibrillation, ACEi: Angiotensin converting enzyme inhibitors, ARB: Angiotensin receptor blockers, ARNI: Angiotensin receptor/neprilysin inhibitors, SGLT2-i: Sodium glucose co-transporter-2 inhibitors, IQR: The interquartile range. LVEDd: Left ventricular end diastolic diameters, LVESd: Left ventricular end systolic diameters, LAd: Left atrium diameters, LVEF: Left ventricular ejection fraction, TAPSE: Tricuspid annular plane systolic excursion, RVsm (TDI): Right ventricular peak systolic myocardial velocity by tissue doppler imagining, TRV: Tricuspid regurgitation velocity, SPAP: Systolic pulmonary artery pressure.

Table 2. Overall univariate and multivariate analysis for predictors of primary outcomes

	Univariate analysis	Univariate analysis	Multivariate analysis	Multivariate analysis
	HR (95% CI)	p	HR (95% CI)	p
Age, years	1.036 (1.027-1.046)	<0.0001	1.017 (1.005-1.028)	0.004
Gender, male	1.482 (1.17-1.86)	0.001	-	-
BMI, kg/m^2	0.99 (0.95-1.039)	0.80	-	-
BSA, m^2	1.166 (0.432-3.145)	0.76	-	-
SBP, mmHg	0.981 (0.975-0.986)	<0.0001	0.990 (0.984-0.996)	0.001
DBP, mmHg	0.978 (0.969-0.987)	<0.0001	-	-
NYHA FC		<0.0001		0.001
I	-	-	-	-
II	1.822 (1.031-3.222)	0.039	1.172 (0.653-2.104)	0.59
III	4.721 (2.699-8.258)	<0.0001	1.874 (1.023-3.433)	0.042
IV	6.509 (3.589-11.804)	<0.0001	2.366 (1.1198-4.675)	0.013
Arterial hypertension	1.118 (0.922-1.355)	0.25	-	-
Ischemic etiology	1.287 (1.065-1555)	0.009	1.284 (1.025-1.609)	0.03
Diabetes mellitus	1.271 (1.046-1.545)	0.016	-	-
Dyslipidemia	1.236 80.971-1.573)	0.085	-	-
Smoking history	1.291 (1.067-1.561)	0.009	-	-
COPD	1.135 (0.787-1.635)	0.49	-	-
Renal disease	2.026 (1.352-3.036)	0.001	-	-
Chronic liver disease	1.834 (0.758-4.437)	0.17	-	-
Peripherial arterial disease	0.945 (0.504-1.770)	0.85	-	-
Atrial fibrillation	1.130 (0.894-1.427)	0.307	-	-
Device				<0.0001
Pacemaker	0.138 (0.051-0.371)	<0.0001	0.438 (0.162-1.189)	0.10
ICD	0.822 (0.671-1.008)	0.060	0.576 (0.459-0.724)	<0.0001
CRT-D	0.554 (0.408-0.753)	<0.0001	0.362 (0.259-0.505)	<0.0001
Family history	0.694 (0.514-0.935)	0.016	-	-
ACEi or ARB	0.546 (0.447-0.67)	<0.0001	-	-
ARNI	1.165 (0.850-1.596)	0.34	-	-
Beta-blocker	0.317 (0.208-0.484)	<0.0001	-	-
Aldosterone antagonist	0.475 (0.362-0.625)	<0.0001	-	-
SGLT2-i	0.914 (0.715-1.168)	0.47	-	-
Loop diuretic	3.120 (2.010-4.842)	<0.0001	-	-
Anticoagulant	0.984 (0.808-1.200)	0.87	-	-
Antiplatelet	1.158 (0.959-1.398)	0.12	-	-
If channel blocker	1.080 (0.890-1.309)	0.43	-	-
Digoxine	0.991 (0.804-1.221)	0.93	-	-
Antiarrhytmic	0.778 (0.585-1.036)	0.08	-	-
Antihyperlipidemic	1.050 (0.869-1.269)	0.61	-	-

Cox univariate and multivariate model, p value <0.05. HR: Hazard ratio, CI: Confidence interval, BMI: Body mass index, BSA: Body surface area, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, NYHA FC: New York Heart Association functional capasity, COPD: Chronic obstructive pulmonary disease. ICD: Intracardiac device, CRT-D: Cardiac resynchronization therapy with defibrillation, ACEi: Angiotensin converting enzyme inhibitors, ARB: Angiotensin receptor blockers, ARNI: Angiotensin receptor/neprilysin inhibitors, SGLT2-i: Sodium glucose co-transporter-2 inhibitors

Table 2. Overall univariate and multivariate analysis for predictors of primary outcomes (continue)

	Univariate analysis	Univariate analysis	Multivariate analysis	Multivariate analysis
	HR (95% CI)	p	HR (95% CI)	p
LVEDd, mm	1.022 (1.011-1.033)	<0.0001	-	-
LVESd, mm	1.022 (1.013-1.032)	<0.0001	-	-
LAd, mm	1.048 (1.035-1.060)	<0.0001	-	-
LVEF, %	0.934 (0.918-0.951)	<0.0001	-	-
Mitral regurgitation		<0.0001		0.001
Mild	0.549 (0.370-0.814)	0.003	0.786 (0.501-1.233)	0.29
Moderate	1.392 (0.992-1.954)	0.056	1.106 (0.764-1.602)	0.59
Severe	2.229 (1.562-3.182)	<0.0001	1.589 (1.092-2.311)	0.015
Tricuspide regurgitation		<0.0001		0.037
Mild	1.699 (1.1150-2.509)	0.008	1.372 (0.877-2.146)	0.16
Moderate	3.556 (2.445-5.171)	<0.0001	1.443 (0.911-2.286)	0.11
Severe	4.230 (2.835-6.311)	<0.0001	0.988 (0.581-1.678)	0.96
Aort regurgitation		<0.0001	-	-
Mild	1.518 (1.206-1.910)	<0.0001	-	-
Moderate	1.954 (1.399-2.729)	<0.0001	-	-
Severe	2.856 (1.271-6.419)	0.011	-	-
TRV, m/sec	1.702 (1.421-2.039)	<0.0001	-	-
RVsm (TDI)	0.853 (0.821-0.886)	<0.0001	-	-
TAPSE, mm	0.905 (0.886-0.924)	<0.0001	-	-
SPAP, mmHg	1.022 (1.016-1.027)	<0.0001	-	-
NT-pro-BNP (log)	2.698 (2.281-3.192)	<0.0001	1.976 (1.548-2.522)	<0.0001

Cox univariate and multivariate model, p value <0.05. HR: Hazard ratio, CI: Confidence interval, LVEDd: Left ventricular end diastolic diameters, LVESd: Left ventricular end systolic diameters, LAd: Left atrium diameters, LVEF: Left ventricular ejection fraction, TRV: Tricuspid regurgitation velocity, RVsm (TDI): Right ventricular peak systolic myocardial velocity by tissue doppler imagining, TAPSE: Tricuspid annular plane systolic excursion, SPAP: Systolic pulmonary artery pressure.

Heart Failure

OP-050

Evaluation of clinical results of remote patient monitoring in the care of patients with heart failure

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Background and Aim: In the follow-up and management of HF patients, weight, heart rate and blood pressure monitoring via smartphone application; To determine whether it reduces death, emergency service admissions and hospitalization due to HF and to evaluate the usability of this system.

Methods: This study was planned as a prospective, randomized controlled open label, single center cohort study. Patients with a diagnosis of HFrEF for at least 6 months with optimal medical treatment were included in the study. Patients with LVEF above 35%, patients with COPD requiring treatment, and patients with renal failure requiring hemodialysis were excluded from the study. 158 patients were randomized into two arms in an age-matched pair design (usual care (UC) and telemonitoring (TM) groups). The primary endpoint was death due to HF, emergency service admission, and hospitalization composite result. Patients in the TM group were asked to fill in the MHFQL scale to detect the change in comfort of life in patients who were regularly followed up. An open-ended telephone questionnaire was conducted to find out the reasons for discontinuing telemonitoring of patients who stopped following TM follow-up in the first 90 days.

Results: The mean (± SD) age and follow-up time were 52.4 (± 10.4) years and 288.9 (± 37) days, respectively. No significant difference was observed between TM and UC in patients who reached the primary endpoint (95% CI: 0.61-1.89, Log-rank p=0.7924, HR: 1.07). 38% (n=30) of the patients in the TM group stopped telemonitored follow-up in the first 90 days. The most common reason for discontinuing this follow-up

was the increase in anxiety caused by the remote monitoring system (37%, n=11). This was followed by insufficient social support (23%, n=7). There was no significant difference in the baseline characteristics of the group that left the TM follow-up (TMS, n=30) and the group that continued regularly (TMC, n=49). There was no significant difference between UC, TMC and TMS patients who reached the primary endpoint (According to 95% CI UC, TMC and TMS HR: 0.83, 0.43-1.59, HR: 1.48, 0.39-3.14, respectively). In 29 TMC patients who answered the MLHFQ scale, there was no statistically significant change in physical function, emotional state, and total score before and after TM follow-up (respectively, 95% CI, p=0.6787, p=0.4115, p=0.6752).

Conclusions: The smartphone remote monitoring assisted care approach did not show any measurable improvement in the clinical condition of the patients. Although the study did not create statistically significant differences in terms of primary results, it is encouraging enough to both develop new different perspectives and support further research.

Heart Failure

OP-051

Comparison of short-term and long-term predictive abilities of the Intermountain Score and ACEF score in patients with heart failure, and their comparison with each other

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Background and Aim: Heart failure (HF) is a disease with high morbidity and mortality among cardiovascular diseases. Predicting the short-term and long-term mortality risk of these patients is critically important for clinical management. In this context, several risk scores have been developed. Scoring systems like the Intermountain Risk Score (IMRS) and ACEF (Age, Creatinine, and Ejection Fraction) are used as short-term mortality predictors for various chronic diseases, as they rely on patients' demographic characteristics and laboratory results. Both IMRS and ACEF are simple and cost-effective measurements that calculate scores using factors such as patient characteristics and laboratory results. However, determining which score is a better predictor for HF patients and assessing their predictive power for long-term mortality are of great significance to understand the clinical role of these scores. In our study, we aimed to compare IMRS and ACEF for short-term mortality and assess their discriminative ability for predicting long-term mortality. To the best of our knowledge, there is no study in the literature that compares these two scores for short-term mortality or examines their predictive capacities for long-term mortality.

Methods: For this study, heart failure patients retrospectivelty demographic and clinical characteristics such as age, gender, ejection fraction (EF) value, and serum creatinine levels were recorded. Both IMRS and ACEF scores were calculated for each patient.

Results: A total of 269 individuals were studied (mean age: 72.3, 38.2% female). Patients were divided into two groups: short-term (0-30 days) and long-term (0-2 years) mortality. The demographic characteristics of patients are shown in Table 1. The 30-day IMRS (HR: 1.105, 95% CI: 1.024-1.191, p=0.009) and ACEF (HR: 1.589, 95% CI: 1.045-2.414, p=0.031) predicted 30-day mortality (Figure 1). Elevated BNP levels (p=0.001) and male gender (p=0.043) were identified as independent predictors of 30-day mortality (Table 3). There was no statistically significant difference between IMRS and ACEF in predicting short-term mortality. For long-term (0-2 years) mortality, cerebrovascular events were identified as independent predictors. Neither IMRS (HR: 1.022, 95% CI: 0.974-1.071, p=0.379) nor ACEF (HR: 0.823, 95% CI: 0.614-1.104, p=0.194) scores were statistically significant in predicting long-term mortality (Figure 2).

Conclusions: Previous studies have found that IMRS and ACEF scores predict 30-day mortality in patients with acute myocardial infarction and heart failure, consistent with our findings. In comparing these two scores, IMRS was found to be superior to ACEF, although not statistically significant. However, for predicting long-term mortality, neither score was statistically significant. This suggests the need for the development of new scores for predicting long-term mortality in the follow-up of patients with chronic conditions like HF.

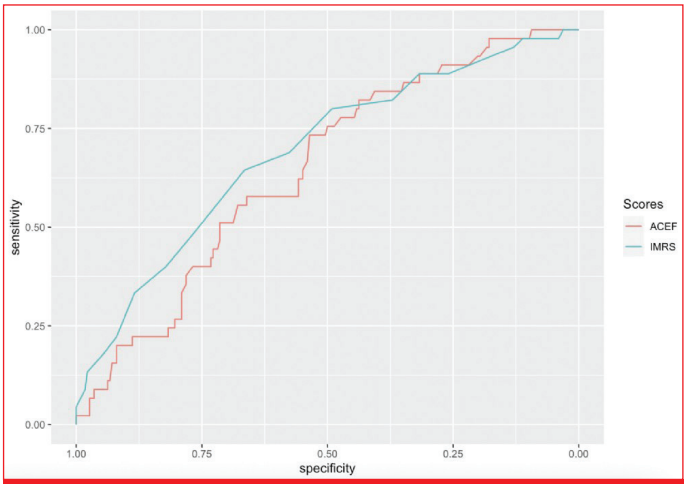

Figure 1.

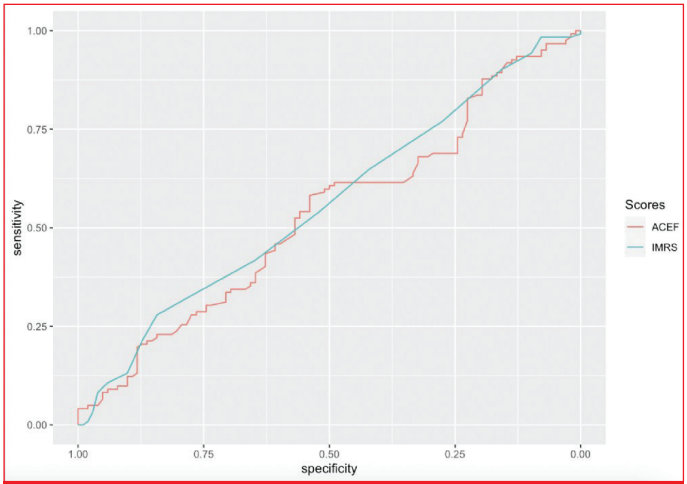

Figure 2.

Table 1.

	30-day mortality		
	Mortality(-) ***(N=224)***	**Mortality(+)** ***(N=45)***	**p- value**
ACEF score	1.91 [1.64;2.60]	2.46 [1.91;2.76]	0.002
IMRS for 30-day	18.0 [14.0;20.0]	20.0 [18.0;23.0]	<0.001
IMRS grades for 30 day:			0.004
Low	58 (25.9%)	5 (11.1%)	
Medium	76 (33.9%)	10 (22.2%)	
High	90 (40.2%)	30 (66.7%)	
	24-months mortality		
	Mortality(-) (N=102)	***Mortality(+) (N=122)***	***p-value***
ACEF score	1.86 [1.68;2.51]	1.96 [1.62;2.62]	0.382
IMRS for 1-year	15.0 (4.26)	14.1 (4.18)	0.102
IMRS grades for 1-year			0.182
Low	11 (10.8%)	24 (19.7%)	
Medium	52 (51.0%)	58 (47.5%)	
High	39 (38.2%)	40 (32.8%)	

Table 2.

	Mortality(-) ***(N=102)***	**Mortality(+)** ***(N=167)***	**p-value**
Age	71.0 (64.0-82.0)	74.0 (65.5-82.0)	0.183
Male gender	69(67.6%)	100(59.9%)	0.251
Hypertension	75 (73.5%)	135 (80.8%)	0.210
Diabetes mellitus	54 (52.9%)	89 (53.3%)	1.000
Cigarette smoking	42 (41.2%)	72 (43.1%)	0.853
CAD	75 (73.5%)	125 (74.9%)	0.923
Hyperlipidemia	46 (45.1%)	80 (47.9%)	0.748
CVA	27 (26.5%)	40 (24.0%)	0.750
CKD	27 (26.5%)	54 (32.3%)	0.379
COPD	35 (34.3%)	52 (31.1%)	0.685
WBC	9.35 (7.09-11.9)	8.90 (6.60-11.4)	0.190
Hemoglobin	11.9 (2.36)	12.0 (2.20)	0.763
Hct	36.4 (7.06)	36.3 (6.69)	0.917
Lymphocyte	1.81 (1.15-2.49)	1.51 (0.96-2.10)	0.019
Monocyte	0.56 (0.38-0.77)	0.53 (0.40-0.68)	0.343
Neutrophil	6.22 (4.74-8.27)	5.90 (4.37-8.66)	0.565
MCV	86.3 (81.2-89.7)	86.3 (81.9-90.1)	0.577
MCHC	32.7 (32.0-33.7)	32.7 (31.7-34.0)	0.725
MPV	9.30 (8.27-10.2)	9.80 (8.60-10.7)	0.030
Platelets	244 (187-313)	212 (174-262)	0.008
RDW	14.2 (13.1-16.1)	14.8 (13.5-16.5)	0.109
Serum creatinine	1.13 (0.90-1.39)	1.19 (0.98-1.58)	0.346
Glucose	126 (103-170)	125 (96.5-180)	0.718
CRP	26.7 (4.93-59.8)	22.0 (4.42-75.7)	0.983
Serum albumin	32.0 (27.4-38.2)	32.0 (25.1-38.0)	0.835
HCO3	24.9 (22.4-29.4)	26.7 (23.0-30.7)	0.100
Sodium	137 (133-140)	138 (134-140)	0.368
Potassium	4.30 (3.80-4.70)	4.20 (3.60-4.70)	0.383
BNP	2073 (661-3676)	1446 (612-3644)	0.957
LVEF	40.0 (31.5-45.0)	38.0 (30.0-45.0)	0.056
LVSD	44.0 (37.0-52.0)	43.0 (38.0-52.0)	0.853
TPAP	40.0 (30.0-45.0)	45.0 (35.0-55.0)	0.001
Stay in ICU, days	0(0-3)	2(0-5)	0.004

Heart Failure

OP-052

The use of SGLT-2 inhibitors and hemoconcentration in patients with heart failure

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Background and Aim: SGLT-2 inhibitors (SGLT-2i) in heart failure patients reduce heart failure-related hospitalizations and increase survival as unevinced, alleged particular mechanisms. SGLT-2i are announced as smart osmotic diuretics.

Methods: Outpatient heart failure 177 patients using SGLT-2i between 2019-2023 were included in the study. The initial examination comprised a group of ninety-eight patients who commenced SGLT-2i treatment following admission to the heart failure outpatient clinic. Hemoconcentration readings were monitored, provided they were taken at least two weeks after treatment initiation. The Wilcoxon rank and paired tests were used to compare outcomes that did not adhere to a normal distribution. As part of the analysis, the study also included 79 patients who had been prescribed SGLT-2i prior to their admission to the heart failure outpatient clinic. A mixed linear model was employed with the patients to measure the hemoconcentration effects of SGLT-2i during routine follow-up sessions at varying intervals.

Results: A study was conducted on 177 heart failure patients with a mean age of 51.9 ± 12.0. Out of the total patients,74.3% were male, and the mean LVEF was 32.3 ± 13.4%. The comparison time median was 70 days, ranging from 34 to 168 days. The study showed that after initiating SGLT-2i, there was a significant increase in hemoglobin and hematocrit levels (Δd-Hb: 0.51, 95% CI: 0.30-0.70, Z-score: -4.250, p<0.0001; Δd-Hct: 1.74, 95% CI: 0.32-1.08, Z-score: -4.758, p<0.0001; respectively). During the initial treatment of heart failure patients with SGLT-2i, there was a significant decrease in the majority of patients' NT-pro-BNP levels (Δd-NT-pro-BNP: -392, 95% CI: -978-193), Z-score: -3.604, p<0.0001). SGLT-2i did not lead to any impairment of renal function or electrolyte imbalance. Through continuous analysis of heart failure patients who have not received treatment before and are currently prescribed SGLT-2i, laboratory parameters were used for mixed linear model repeated measures during 789 follow-up visits. Patients undergoing SGLT2i therapy showed significantly lower levels of hemoglobin and hematocrit than those not using SGLT-2i in the long term (Intercept estimate: 13.8, Δd-estimate: -0.26, F test: 21.6, p<0.001; Intercept estimate: 42.3, Δd-estimate: -0.97, F test: 41.5, p<0.001; respectively). Mean while, NT-pro-BNP levels increased and did not change significantly (Intercept estimate: 2248, Δd-estimate: 143, F test: 0.6, p=0.41). These effects were also analyzed with different loop diuretic doses in each patient. The hemoconcentration parameters were significantly lower (Intercept estimate: 13.7, Δd-estimate: -0.25, F test: 18.3, p<0.001; Intercept estimate: 42.1, Δd-estimate: -0.97, F test: 41.3, p<0.001; respectively).

Conclusions: Although the hemoconcentration properties of this treatment show noticeable effects in the initial stages, this effect tends to disappear in the long run. Instead, there is a rise in the values of hemoglobin and hematocrit, which may result from heart failure progression.

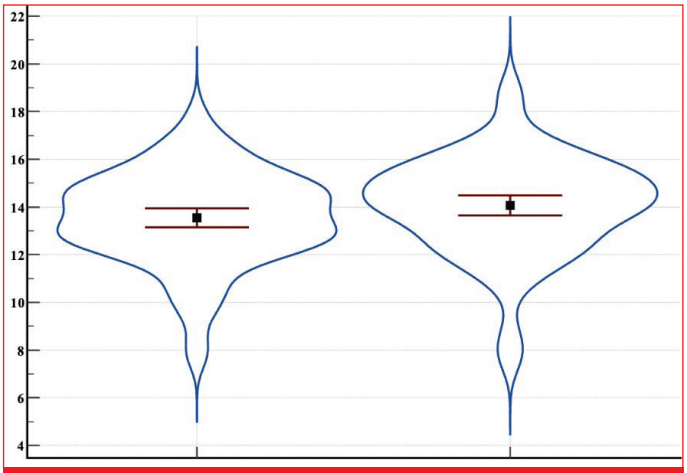

Figure 1. Hemoglobin change after initiation of SGLT-2 inhibitors.

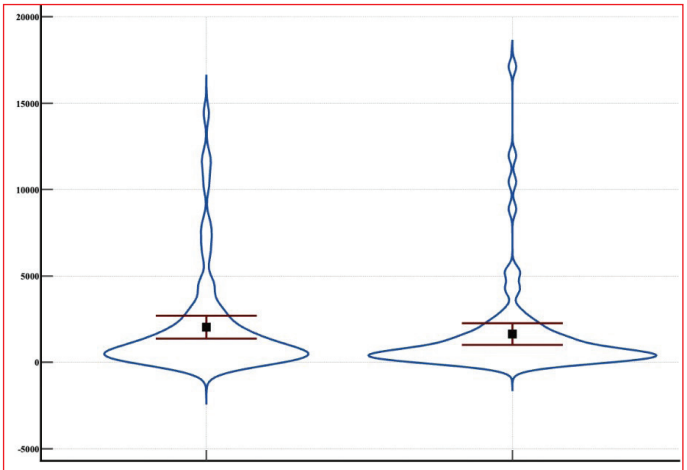

Figure 2. NT-pro-BNP change after initiation SGLT-2 inhibitors.

Table 1. Baseline characteristics

Baseline characteristics	First visit	Second visit	Z score	p
Age, years	51.9 ± 12.0	-	-	-
Male, %	74.3	-	-	-
BMI, kg/m^2	29.7 ± 5.1	29.7 ± 5.1	-	-
SBP, mmHg	117.8 ± 23.5	116.4 ± 23.9	-	-
DBP, mmHg	73.1 ± 13.6	73.1 ± 13.2	-	-
NYHA FC, %	-	-	-	-
I	35.7	-	-	-
II	51.2	-	-	-
III-IV	13.1	-	-	-
Ischemic etiology, %	47.5	-	-	-
Arterial hypertension, %	32.8	-	-	-
Diabetes mellitus, %	48.3	-	-	-
Dyslipidemia, %	31.9	-	-	-
Smoking history, %	32.2	-	-	-
Cerebrovascular event, %	4.3	-	-	-
COPD, %	11.9	-	-	-
Renal disease, %	0.9	-	-	-
Chronic liver disease, %	0.9	-	-	-
Peripherial arterial disease, %	0.9	-	-	-
AF, %	10.7	-	-	-
Intracardiac device	-	-	-	-
CRT, %	13.5	-	-	-
ICD, %	36.9	-	-	-

BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, NYHA: New York Heart Association, COPD: Chronic obstructive pulmonary disease, AF: Atrial fibrillation, CRT: Cardiac resynchronization therapy, ICD: Implantable cardioverter defibrillator.

Table 2. Medication

Medication	First visit	Second visit	Z score	p
ACEi or ARB, %	49.1	-	-	-
ARNI, %	21.6	-	-	-
Beta-blocker, %	82.8	-	-	-
Aldosterone antagonist, %	67.2	-	-	-
Loop diuretic, %	64.7	-	-	-
Anticoagulant, %	20.7	-	-	-
Antiplatelet, %	48.3	-	-	-
If channel blocker, %	19	-	-	-
Digoxine, %	12.9	-	-	-
Antihyperlipidemic, %	31	-	-	-

ACEi: Angiotensin-converting enzyme inhibitors, ARB: Angiotensin II receptor blockers, ARNi: Angiotensin receptor/neprilysin inhibitör.

Table 3. Electrocardiography

Electrocardiography	First visit	Second visit	Z score	P
HR, beat/min	77 ± 15	74 ± 11	-	-
PR interval, msec	170 ± 31	170 ± 31	-	-
QRS duration, msec	125 ± 29	123 ± 27	-	-
QT interval, msec	448 ± 53	444 ± 55	-	-

HR: Heart rate

Table 4. Echocardiography

Echocardiography	First visit	Second visit	Z score	p
LVEDd, mm	59.3 ± 0.8	-	-	-
LVESd, mm	47 ± 1.1	-	-	-
LAd, mm	43 ± 0.9	-	-	-
LVEF, %	32.3 ± 13.4	-	-	-
TAPSE, mm	17.8 ± 5.7	-	-	-
RVsm TDI, m/sec	10.2 ± 3.1	-	-	-
TRV, m/sec	2.53 ± 0.57	-	-	-
SPAP, mmHg	48 ± 15	-	-	-
Mitral regurgitation, %	-	-	-	-
None	14.9	-	-	-
Mild	36	-	-	-
Moderate	4.3	-	-	-
Severe	1.7	-	-	-
Aort regurgitation, %	-	-	-	-
None	76.5	-	-	-
Mild	17.4	-	-	-
Moderate	4.3	-	-	-
Severe	1.7	-	-	-
Tricuspide regurgitation, %	-	-	-	-
None	26.1	-	-	-
Mild	40.9	-	-	-
Moderate	19.1	-	-	-
Severe	13.9	-	-	-

LVEDd: Left ventricular end-diastolic diameter, LVESd: Left ventricular end-systolic diameter, LAd: Left atrium diameter, LVEF: Left ventricular ejection fraction, TAPSE: Tricuspid annular plane systolic excursion, RVsm TDI: Right ventricular peak systolic myocardial velocity (RVSm) by tissue, Doppler imaging; TRV: Tricuspid regurgitation velocity, SPAP: Systolic pulmonary artery pressure.

Table 5. Laboratory tests

Laboratory tests	First visit	Second visit	Z score	p
SGOT (IQR), U/L	18 (14-22)	17 (14-22)	-0.222	0.82
SGPT (IQR), U/L	18 (13-27)	18 (12-29)	-0.449	0.65
ALP (IQR), U/L	80 (58-125)	87 (64-117)	-0.195	0.84
GGT (IQR), U/L	35 (20-71)	29 (18-89)	-1.245	0.21
Total protein, g/L	58.1 ± 22.1	56.1 ± 12	-0.105	0.91
Albumin, g/L	36.6 ± 13.7	39.1 ± 12	-0.105	0.91
Total bilirubin (IQR), mg/L	0.58 (0.44-0.94)	0.62 (0.39-0.95)	-0.304	0.76
Direct bilirubin (IQR), mg /L	0.26 (0.20-0.44)	0.26 (0.20-0.46)	-0.509	0.61
NT-pro-BNP, pg/mL (IQR), ng/L	953 (353-2392)	658 (285-1798)	-3.604	<0.0001
Total cholosterol, mg/dL	167 ± 47	169 ± 53	-0.159	0.87
Triglyceride, mg/dL	149 ± 82	169 ± 99	-2.21	0.027
HDL cholosterol, mg/dL	42 ± 13	42 ± 17	-1.181	0.23
LDL cholosterol, mg/dL	97 ± 34	93 ± 41	-0.963	0.33
Fasting glucose, mg/dL	128 ± 59	126 ± 53	-0.518	0.604
Urea, mg/dL	43 ± 20	46 ± 27	-0.676	0.49
Creatinin, mg/dL	1.00 ± 0.27	1.07 ± 0.33	-1.323	0.18
Sodium, mEq/L	137 ± 3.8	137 ± 4.2	-0.686	0.49
Potassium, mEq/L	4.5 ± 0.5	4.6 ± 0.5	-0.706	0.48
Chloride, mEq/L	98 ± 4.2	99 ± 4.8	-1.126	0.26
Hemoglobin, g/dL	13.5 ± 1.9	14.1 ± 2.0	-4.250	<0.0001
Hematocrit, %	41.5 ± 5.4	43.2 ± 5.4	-4.758	<0.0001

SGOT: Serum glutamic-oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase, ALP: Alkaline phosphatase, GGT: Gamma glutamyl transferase, HDL: High-density lipoprotein, LDL: Low-density lipoprotein.

Heart Failure

OP-053

Relationship between iron levels and heart failure related events in patients with ST-segment elevation myocardial infarction

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Background and Aim: Worldwide, ischaemic heart disease is the most common cause of death, and its frequency is increasing. ST-segment elevation myocardial infarction (STEMI) is the most catastrophic type of ischaemic heart disease. Despite the increase in the prevalence of primary percutaneous coronary intervention (pPCI) and the improvement of the local techniques used, some patients still develop heart failure after STEMI. It is also known, as previous studies suggest, serum iron levels and heart failure event are strictly correlated; if iron deficiency is present in patients with chronic heart failure, iron replacement reduces symptomatic heart failure and rehospitalizations. Our study aims yo investigate the relationship between serum iron levels in patients with STEMI and future heart failure related events.

Methods: We enrolled retrospectively 347 STEMI patients treated with pPCI and who have iron deficiency workup done in the index hospitalization. Patients divided into two groups regarding to the existence of iron deficiency. Iron deficiency is described as either a serum ferritin concentration <100 ng/mL or 100-299 ng/mL with transferrin saturation <20%.

Results: Mean age was 56 (± 11,5) and 80% of patients were male. 49% of patients were admitted to the hospital with anterior STEMI and 51% were non-anterior localization. There was no statistically significant difference between groups in case of basal characteristics and procedural characteristics (Table 1, 2, 3). Mean follow-up was 24 months. Our study demonstrated patients with STEMI who have iron deficiency in the index hospitalization are more likely to develop symptomatic heart failure requiring hospitalization [16.8% vs. 8.7% (HR: 1.93, p=0.028)]. Furthermore, patients with iron deficiency at 12 months assessed by transthoracic echocardiography, using biplane Simpson method [42% vs. 45% (p=0.34)] (Table 4).

Conclusions: Our study revealed iron deficiency is a predictor of future heart failure related hospitalizations in STEMI patients treated with pPCI.

Table 1. Baseline characteristics

	Iron Deficiency (n:139)	Control (n:208)	p value
Age	57,6±11,4	55,7±11,7	0,140
Male %	75,5	88,3	**0,002**
Hypertension%	36,5	26,2	**0,044**
Diabetes mellitus %	31,6	22,8	0,070
Chronic renal failure %	10,1	6,8	0,300
Coronary artery disease (previously known) %	19,1	17,2	0,659
Cerebrovascular Event %	5,1	2,5	0,196
Hyperlypidemia%	14,0	9,0	0,148

Table 2. Laboratory results

	Iron Deficiency (n:139)	Control (n:208)	p value
Pro-BNP (pg/mL)	265 (1490)	258 (1092)	0,696
Troponin peak time (hour)	6,0 (8,0)	9,0 (6,0)	0,394
Troponin-T (max)	4,03 (7,98)	4,05 (6,72)	0,713
Hgb (g/dL)	13,1 (2,9)	13,9 (1,9)	**<0,001**
WBC count	11,000 (4,400)	11,900 (5,300)	0,093
PLT count	248,000 (78,400)	239,000 (99,500)	0,462
Urea (mg/dL)	31,0 (16)	33 (13)	0,434
Creatinin (mg/dL)	0,84 (0,30)	0,83 (0,26)	0,597
eGFR	113 (38,8)	112 (56,6)	0,440
Albumin (g/dL)	3,9 (0,5)	4,0 (0,5)	**0,010**
HbA1c%	6,1 (1,0)	5,9 (1,3)	0,107
Glucose (g/dL)	129 (70)	130 (69)	0,971
CRP (mg/L)	3,9 (10,4)	4,3 (7,3)	0,382
LDL (mg/dL)	126 (51)	125 (45)	0,942

Table 3. Procedural data

	Iron Deficiency (n:139)	Control (n:208)	p value
Anterior localization	%53,4	%42,2	**0,046**
AHA Lesion type			0,133
A	%20,6	%27,4	
B	%35,3	%38,8	
C	%44,1	%33,8	
Thrombus grade			0,380
1	%0	%1,5	
2	%13,3	%12,9	
3	%46,7	%51,2	
4	%40	%34,3	
No-reflow %	%20	%17,8	0,615

Table 4. Primary and secondary endpoints

	Iron Deficiency (n:139)	Control (n:208)	HR	p value
HF hospitalization	%16,8	%8,7	1.93	**0,028**
Ejection Fraction (12th month)	%42 (%15)	%45 (%14)		**0,034**
All cause mortality	%17,4	%16,8		0,893

Heart Failure

OP-054

Effect of atrial fibrillation on frailty in patients with heart failure with reduced ejection fraction (HFrEF)

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Background and Aim: Atrial fibrillation (AF) is a frequently encountered comorbidity in patients with reduced ejection fraction heart failure (HFrEF). The aim of this study is to investigate the impact of AF on frailty parameters in patients with HFrEF.

Methods: In our single-center retrospective observational study, 89 patients diagnosed with chronic heart failure, having functional capacity of NYHA class 2 or 3, and not requiring positive inotropic support were divided into two groups: 45 in sinus rhythm (SR) and 44 in AF rhythm. Frailty assessment included gait speed, 6-minute walk test (6MWT), Timed Up and Go (TUG) test, handgrip strength test, PRISMA-7 questionnaire, and the Minnesota Living with Heart Failure questionnaire (MLHFQ).

Results: Similar rates of diabetes, hypertension, and chronic kidney disease were observed in both groups. The NT-pro-BNP level was statistically higher in the AF group (Table 1). Frailty parameters were compared in two groups. 6MWT, TUG, Handgrip test, Minnesota Heart Failure Living Questionnaire Emotional Dimension Score was found to be statistically significant (Table 2). Using the Charlson Comorbidity Index, a frailty index was constructed based on frailty parameters. Cluster analysis divided participants into four groups based on frailty severity: no frailty, mild, moderate, and severe. Multinomial logistic regression analysis revealed that the presence of AF in HFrEF patients increased the risk of moderate frailty by 3.4 times and severe frailty by 5.5 times. High NT-pro-BNP levels and low GFR increased the risk of severe frailty by 2 times (Table 3).

Conclusions: Our study identified that the presence of AF in HFrEF patients is an independent predictor of frailty.

Table 1. Comorbidities, medications, blood tests and echocardiographic parameters

Parameters	SR group (n=45)	AF group (n=44)	p
Ischemic HF, n (%)	38 (62.3)	23 (52.2)	0.001
DM, n (%)	12 (26.6)	19 (43.1)	0.102
HT, n (%)	26 (57.7)	19 (43.1)	0.169
CKD, n (%)	13 (28.8)	17 (38.6)	0.331
NYHA, n (%) NYHA 2 NYHA 3	 24 (53.3) 21 (46.7)	 20 (45.4) 24 (54.6)	 0.457 0.414
BB, n (%)	44 (97.7)	44 (100.0)	1.00
RAAS inhibitors, n (%) ACEi ARB ARNI	 29 (64.4) 9 (20.0) 0	 29 (65.9) 3 (6.8) 3 (6.8)	 0.101
Loop diuretics, n (%)	25 (55.5)	38 (86.3)	0.001
Thiazide diuretics, n (%)	7 (15.5)	18 (40.9)	0.008
MRA, n (%)	33 (73.3)	33 (75.0)	0.857
SGLT-2i, n (%)	9 (20.0)	22 (50.0)	0.003
GFR, mL/min/1.73 m^2	84.90 ± 35.45	71.38 ± 28.22	0.050
NT-pro-BNP, pg/mL	1565.37 ± 1398.47	2668.95 ± 1771.85	0.001
Albumin, g/dL	4.020 ± 0.377	3.763 ± 0.449	0.004
LVEF, %	29.71 ± 6.583	28.77 ± 7.563	0.534
LAVI, mL/m^2	32.389 ± 10.963	38.47 ± 21.164	≤0.001
TR velocity, m/sec	2.60 ± 0.433	3.19 ± 0.51	≤0.001
SPAP, mmHg	32.53 ± 9.45	40.95 ± 13.71	≤0.001
TAPSE, mm	16.311 ± 3.182	14.5 ± 4.112	0.022
Tricuspid S velocity, cm/sec	12.11 ± 2.647	9.59 ± 2.275	≤0.001
RAA, cm^2	16.824 ± 4.641	23.64 ± 5.258	≤0.001
IVC diameter, cm	2.171 ± 0.241	2.351 ± 0.314	0.015
IVC collapse <%50, n (%)	7 (26.9)	19 (73.1)	0.004

AF: Atrial fibrilation, BB: Beta-blocker, min: Minute, DM: Diabetes mellitus, GFR: Glomerular filtration rate, HT: Hypertension, CKD: Chronic kidney disease, HF: Heart failure, LA: Left atrium, LAVI: Left atrium volume, MRA: Mineralocorticoid receptor antagonist, NT-pro-BNP: N terminal pro brain natriuretic peptide, OR: Odds ratio, RAA: Right atrium area, RAS: Renin angiotensin aldosterone system, SPAP: Systolic pulmonary artery pressure, TAPSE: Tricuspid annular plane systolic excursion, TR: Tricuspid regurgitation, IVC: Inferior vena cava.

Table 2. Comparison of frailty among groups

Frailty parameters	Frailty confirmed in SR group (n=45)	Frailty confirmed in AF group (n=44)	Total (n=89)	p
Walking speed, n (%)	17 (44.7)	21 (55.3)	38 (42.7)	0.343
6MWT, n (%)	20 (37.7)	33 (62.3)	53 (59.6)	0.003
TUG test, n (%)	28 (43.1)	37 (56.9)	65 (73.0)	0.020
Hand grip, n (%)	21 (38.9)	33 (61.1)	54 (60.7)	0.006
PRIZMA-7, n (%)	28 (45.2)	34 (54.8)	62 (69.7)	0.123
The minnesota living with heart failure questionnaire	23.00 ± 13.636	25.023 ± 8.42	-	0.401
The minnesota living with heart failure questionnaire emotional extent score	11.82 ± 7.152	14.386 ± 4.325	-	0.044

AF: Atrial fibrilation, 6MWT: Six-minute-walk-test, SR: Sinus rhythm, TUG: Time up and go test.

Table 3. Assessment of frailty determinants utilizing the frailty index and charlson comorbidity index

	Frailty	OR	Lower limit	Upper limit	p
Age	Mild	1.018	0.940	1.102	0.663
	Moderate	1.086	0.990	1.192	0.082
	Severe	1.082	1.002	1.167	0.043
Rhythm (AF)	Mild	2.000	0.332	12.046	0.449
	Moderate	3.436	1.001	5.793	0.038
	Severe	5.684	1.084	8.803	0.040
GFR, mL/min/1.73 m^2	Mild	0.936	0.882	1.031	0.615
	Moderate	1.475	0.962	1.917	0.427
	Severe	1.975	1.195	2.998	0.037
NT-pro-BNP, pg/mL	Mild	1.000	0.989	1.041	0.811
	Moderate Severe	1.401	1.145	1.907	0.043
		2.051	1.516	2.701	0.002
Albumin, g/dL	Mild	0.890	0.501	1.348	0.186
	Moderate	1.447	0.914	4.828	0.638
	Severe	4.193	1.927	9.081	0.047
LAVI, mL/m^2	Mild	2.789	0.832	9.350	0.097
	Moderate	6.060	1.478	24.850	0.012
	Severe	11.344	3.154	40.797	<0.001
TR velocity, m/sec	Mild	1.432	0.635	5.699	0.405
	Moderate	3.274	1.228	9.521	0.032
	Severe	6.566	3.279	14.316	0.002
SPAP, mmHg	Mild	0.977	0.892	1.069	0.607
	Moderate	1.351	0.960	1.651	0.279
	Severe	2.103	1.418	3.195	0.017
TAPSE, mm	Mild	0.753	0.654	1.160	0.753
	Moderate	1.129	0.824	1.451	0.121
	Severe	2.272	1.616	3.921	0.006
Tricuspid S wave velocity, cm/sec	Mild	0.762	0.511	1.027	0.612
	Moderate	0.997	0.776	1.202	0.170
	Severe	1.719	1.548	1.944	0.017
IVC diameter, cm	Mild	0.645	0.296	1.350	0.652
	Moderate	1.435	0.793	2.450	0.597
	Severe	2.050	1.144	3.312	0.005

AF: Atrial fibrilation, GFR: Glomeruler filtration rate, HF: Heart failure, LA: Left atrium, LAVI: Left atrium volume index, NT-pro-BNP: N terminal pro brain natriuretic peptide, OR: Odds ratio, SPAP: Systolic pulmonary artery pressure, TAPSE: Tricuspid annular plane systolic excursion, TR: Tricuspid regurgitation, IVC: Inferior vena cava.

Heart Failure

OP-055

Is tinnitus useful for detection of worsening heart failure?

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Background and Aim: There are many causes of tinnitus, from iatrogenic, genetics, vascular to neurological diseases. Tinnitus and heart failure are seen frequently together, since they have similar risk factors. There is not enough data about tinnitus and related parameters in patients with heart failure. In our study, we aimed to investigate the tinnitus in heart failure reduced ejection fraction (HFrEF) patients and the related clinical parameters.

Methods: A total of 64 HFrEF patients with tinnitus and 56 HFrEF patients without tinnitus were included in this study. All patients with tinnitus were evaluated with this Tinnitus Handicap Inventory Scale. With this scale, patients' tinnitus was graded from one to five. All patients' history, physical examination, echocardiography and laboratory parameters were evaluated. Clinical determinants of the tinnitus in HFrEF patients were established using univariate and multivariable logistic regression analyses.

Results: There was no significant difference in terms of age (p=0.660, 60.4 ± 12.6 vs. 61.4 ± 10.9), body mass index (p=0.505), diabetes mellitus (p=0.192), hypertension (p=0.184) and coronary artery disease (p=0.348) in HFrEF patients with and without tinnitus. Female gender was more common in patients with tinnitus (p=0.009, 45.3% vs. 23.2%). Systolic blood pressure (p=0.542), diastolic blood pressure (p=0.067), heart rate (p=0.231) and pulse pressure (p=0.592) were similar between groups. BNP levels (p=0.003) were higher in patients with tinnitus. The use of beta-blockers (p=0.015) and ASA (p=0.003) were higher in patients without tinnitus. In terms of ecocardiographic parameters, it was observed that patients with tinnitus had lower EF (p=0.027) and higher left ventricular end diastolic diameter (p=0.009), left ventricular end systolic diameter (p=0.022) and left atrial diameter (p=0.048). Atrial fibrillation (p<0.001, 37.2% vs. 17.9%) was more common in patients with tinnitus. There was a positive correlation between BNP and tinnitus grade in correlation analysis (r= 0.232, p=0.020). Female gender (p=0.013), atrial fibrillation (p=0.01), ejection fraction (p=0.019), BNP (p=0.039), beta-blocker (p=0.021) and ASA (p=0.006) were significant in univariate regression analysis. BNP (p=0.020) and atrial fibrillation (p=0.004) were significant in multivariate regression analysis.

Conclusions: The presence of tinnitus was associated with higher levels of BNP and the tinnitus grade was correlated with BNP positively. Also, AF was independent risk factor for tinnitus in HFrEF patients.

Table 1. Baseline characteristics, echocardiographic, laboratory parameters and medication in HFrEF patients

Characteristics	Tinnitus (+) (n=64)	Tinnitus (-) (n=56)	p
Age	60.4 ± 12.6	61.4 ± 10.9	0.660
Gender (Female), %	45.3	23.2	0.009
Body mass index, kg/m^2	27.3 ± 0.48	26.7 ± 4.1	0.505
NYHA classifcation	2.9 ± 0.5	3 ± 0.2	0.075
Hypertension	46 (71.9%)	35 (62.5%)	0.184
Diabetes mellitus	23 (35.9%)	23 (41.1%)	0.192
Coronary artery disease	48 (75%)	37 (66.1%)	0.348
Atrial fibrillation	16 (37.2%)	10 (17.9%)	<0.001
Systolic blood pressure, mmHg	120 (120-130)	120 (110-130)	0.542
Diastolic blood pressure, mmHg	80 (70-80)	70 (60-80)	0.067
Ejection fraction, %	30 (20-35)	30 (30-35)	0.027
Left ventricular end diastolic diameter, mm	61.3 ± 8.5	56.6 ± 6.3	0.009
Left atrial diameter, mm	45.8 ± 6.4	43 ± 4.8	0.048
e/e' ratio	11.5 (8.3-14)	12.3 (9.7-15.9)	0.161
Hemoglobin, g/dL	12.8 ± 1.8	13 ± 1.8	0.523
Creatinine, mg/dL	1.16 ± 0.61	1.19 ± 0.5	0.769
C-reactive protein, mg/dL	0.79 (0.22-2.3)	0.9 (0.5-1.7)	0.380
BNP, pg/mL	1367 (704-2872)	1040 (361-1680)	0.003
Total cholesterol, mg/dL	173 ± 41	167 ± 45	0.453
LDL cholesterol, mg/dL	106 ± 36	1.16 ± 0.61	0.406
HDL choltesterol, mg/dL	39 ± 12	36 ± 11	0145
Trigliseride, mg/dL	146 ± 9	163 ± 92	0.283
Acetyl salicylic acid	43 (67.2%)	50 (89.3%)	0.003
ARB	9 (14.1%)	15 (26.8%)	0.066
ACE inhibitor	36 (56.3%)	40 (71.4%)	0.062
Beta-blocker	46 (71.9%)	50 (89.3%)	0.015
Furosemide	42 (65.6%)	41 (77.4%)	0.117
Tiazide	28 (43.8%)	30 (58.5%)	0.078
Spironolactone	19 (44.2%)	29 (51.8%)	0.292

Table 2. Significant univariate and multivariate analysis of tinnitus in HFrEF patients

Variables	Univariate regression coefficient (95% CI)	p	Multivariate regression coefficient (95% CI)	p
Gender (Female)	0.365 (0.165-0.805)	0.013	0.379 (0.090-1.605)	0.188
Atrial fibrillation	0.218 (0.091-0.518)	0.01	0.098 (0.020-0.483)	0.004
Ejection fraction, %	0.920 (0.858-0.986)	0.019	0.906 (0.807-1.017)	0.093
BNP, pg/mL	1.000 (1.000-1.001)	0.039	1.000 (1.000-1.001)	0.020
Beta-blocker	3.261 (1.191-8.926)	0.021	0.769 (0.104-5.659)	0.796
Acetyl salicylic acid	4.070 (1.505-11.004)	0.006	1.548 (0.208-11.538)	0.670

Heart Failure

OP-056

Single-centre experience with tafamidis in patient with transthyretin cardiac amyloidosis

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Background and Aim: Transthyretin cardiac amyloidosis (ATTR-CA) is a progressive disease with a poor prognosis. Tafamidis improves prognosis in patients with ATTR-CA. However, real-world data on the use of tafamidis in ATTR-CA are limited. In this case series; we presented the clinical features and follow-up data of patients diagnosed with ATTR-CA treated with tafamidis in our center.

Methods: We initiated tafamidis treatment in 10 patients with the diagnosis of ATTR-CA (9 patients wTTR-CA, 1 patient mTTR-CA) between 2020-2023. All patients received 80 mg of tafamidis daily until March 2023 and then tafamidis meglumine 61 mg was started after this date as it was approved by the national health system. Data on baseline were collected. Composite outcomes of HF related hospitalization and mortality after tafamidis treatment were evaluated. Clinical data before and after 6 months of tafamidis treatment have also been examined.

Results: The mean age of patients was 71.1 ± 11.6 years, and 5 (50%) were male. At baseline, 7 patient was NYHA class III, 3 patients were NYHA class II. Three patients had atrioventricular (AV) conduction system disease and pacemaker implantation was performed in one patient due to complete AV block. Median (IQR) NT-pro-BNP value was 2468 pg/mL (961-7331). Mean left ventricular (LV) ejection fraction ± SD was 58.4 ± 5.5%, mean LV global longitudinal strain was -9.2 ± 2.9%, and mean LV wall thickness (LVWT) ± SD was 15.9 ± 3.0 mm. The median duration of tafamidis treatment was 13 months. Four patients were hospitalized due to HF and 1 patient was hospitalized due to acute stroke. Also, three patients died (2 of them due to HF, 1 of them due to acute stroke). One of the patients died due to HF in the second month of treatment. One patient was not using the treatment regularly because the patient complained of digestive symptoms during treatment with tafamidis. NYHA was worsened in 3 patients, remained unchanged in 6 patients and improved in 1 patients. There was significant difference in terms of LVWT (16.3 ± 2.9 mm vs. 15.8 ± 2.9, p=0.015) at 6-month. No significant difference was also found in NT-pro-BNP (2364 vs. 1700, p=0.222), troponin (0.037 vs. 0.055, p=0.370) and creatinin (0.95 vs. 1.01, p=0.266) levels from baseline to 6 months. None of the patients receiving tafamidis had worsening in kidney and liver functions. The medication was not discontinued due to any side effects. Composite outcome of HF related hospitalization and mortality occurred in 5 patients. LVEF was lower (55 ± 5.61 vs. 61.8 ± 2.9, p=0.043) and NT-pro-BNP was higher [4818 (888-19225) vs. 2364 (1058-2996), p=0.038] in the group with the composite outcome (Table 1).

Conclusions: The results of this study suggested that patients diagnosed with ATTR-CA showed poor outcomes with a 30% mortality and 40% hospitalization rates during 13-month follow-up. In our real-life clinical practice, tafamidis, the only guideline-recommended treatment option for ATTR-CA, was generally well tolerated and showed a good safety profile.

Table 1. Clinical features of the ATTR-CA patients with and without composite outcomes

	Total (n=10)	Composite outcome (+)	Composite outcome (-)	p
Age, years	71.1 ± 11.6	76.8 ± 7.3	65.4 ± 12.9	0.126
Male, %	5 (50%)	2 (40%)	3 (60%)	0.300
NYHA functional class Class I Class II Class III Class IV	 0 3 (30%) 7 (70%) 0	 0 2 (40%) 3 (60%) 0	 0 1 (20%) 4 (80%) 0	0.500
Atrial fibrillation, %	4 (40%)	2 (40%)	2 (40%)	0.738
Pericardial effusion, %	1 (10%)	-	1 (20%)	0.500
Cardiac pacemaker, %	1 (10%)	-	1 (20%)	0.500
Troponin, ng/mL	0.058 ± 0.06	0.068 ± 0.07	0.034 ± 0.033	0.570
Serum NT-pro-BNP, pg/mL	2468 (961-7331)	4818 (888-19225)	2364 (1058-2996)	0.038
Serum creatinine, mg/dL	1.05 ± 0.41	1.27 ± 0.48	0.84 ± 0.18	0.105
LVEF, %	58.4 ± 5.5	55 ± 5.61	61.8 ± 2.9	0.043
LV-GLS, %	9.2 ± 2.9	7.6 ± 2.4	10.8 ± 2.5	0.122
LAVI, mL/m^2	35.7 ± 9.4	33.2 ± 11.1	38.2 ± 7.2	0.435
LASr, %	11.1 ± 3.6	11.5 ± 2.8	10.6 ± 4.6	0.761
LVWT, mm	15.9 ± 3.0	15.2 ± 3.6	16.6 ± 2.6	0.520
Granular sparkling, %	7 (70%)	3 (60%)	4 (80%)	0.500

Hypertension

OP-057

The relationship between triglyceride-glucose index (TyG) with dipper hypertension and non-dipper hypertension

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Background and Aim: Blood pressure exhibits a specific circadian rhythm. The lack of adequate nocturnal blood pressure decline due to the disruption of this circadian rhythm is defined as the non-dipper hypertension pattern. Long-term studies comparing dipper and non-dipper patient groups have shown that the non-dipper group has a worse prognosis in terms of cardiovascular events and mortality. The triglyceride-glucose index (TyG index) is a low-cost and easily obtainable marker indicating insulin resistance. Numerous studies have also shown its association with endothelial dysfunction and autonomic dysfunction. The relationship between the mechanisms involved in the circadian rhythm of blood pressure and the TyG index is evident. However, it has been observed that there is a lack of sufficient studies demonstrating the relationship between the circadian rhythm of blood pressure and the TyG index. In our study, we aimed to demonstrate the relationship between the non-dipper hypertension pattern, associated with higher cardiovascular morbidity and mortality, and the TyG index.

Methods: This single-center and retrospective study included 150 patients by examining the electronic medical records of patients who presented to the Cardiology Clinic of Kırıkkale University Faculty of Medicine. Based on the 24-hour ambulatory blood pressure monitoring (ABPM) data, the patients were categorized into three groups: dipper (n=48), non-dipper (n=58), and normotensive control group (n=44). The TyG index was calculated using the formula Ln [fasting triglycerides (mg/dL) x fasting glucose (mg/dL)/2].

Results: According to the obtained data from the study, a significant difference was observed between the TyG index of normotensive and hypertensive groups (p<0.001). Furthermore, there was a statistically significant difference in TyG index values between dipper and non-dipper patients (p=0.033). The TyG index demonstrated a negative correlation with the nocturnal blood pressure dipping rate (r=-0.199, p=0.015, Spearman correlation analysis), and an increase in TyG index value was associated with a decrease in nocturnal blood pressure dipping rates.

Conclusions: TyG index can be used as an indicator of the disruption in the circadian rhythm of blood pressure. High TyG index values in hypertensive patients may indicate the presence of a non-dipper hypertension pattern. These findings were obtained from a single-center and retrospective study and might be influenced by regional variations. Further support for these results is needed through studies involving larger patient groups and multiple centers.

Hypertension

OP-060

Morning surge in blood pressure and cardiovascular risk: Exploring gender differences

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Background and Aim: Normal morning blood pressure (BP) surge is a physiological phenomenon, and this surge is believed to be influenced by the body's circadian rhythm, hormonal fluctuations, and the natural activation of the sympathetic nervous system upon waking. While some degree of morning blood pressure elevation is normal, excessive surges have been associated with heightened cardiovascular risk. This surge may place additional strain on the heart and blood vessels during a period when the body is transitioning from a resting state to increased activity. Recent research has highlighted the significance of the "morning surge" in blood pressure, shedding light on its implications for cardiovascular risk. Moreover, the link between the morning surge and cardiovascular risk might not be uniform. Genetic predisposition, lifestyle factors, and underlying health conditions can all modulate the relationship between morning blood pressure elevation and cardiovascular outcomes. Interestingly, emerging evidence suggests that gender differences may play a role in the morning surge's impact on cardiovascular risk.

Methods: We analyzed 210 adult patients, ages 20-86 years old. The ambulatory blood pressure monitoring (ABPM) was recorded automatically. We defined morning BP surge as follows: a sleep-trough surge defined as the morning BP (2-hour average of four 30-minute BP readings just after wake-up) minus the lowest nocturnal BP (1-hour average of the 3 BP readings centered on the lowest nighttime reading); a prewaking surge defined as the morning BP minus the prewaking BP (2-hour average of 4 BP readings just before wake-up); a rising BP surge defined as the morning BP measured on rising minus the BP in a supine position 30 minutes before rising; and morning-evening difference defined as morning SBP-evening SBP (Figure 1).

Results: Sleep through surge and a morning-evening difference were significantly higher in men compared to women ($p<0.05$ for all). Although there was no statistically significant difference between the two groups in prewaking surge and rising blood pressure surge parameters, these values were higher in men (Table 1).

Conclusions: This study suggested that men may experience a more pronounced morning surge in BP compared to women. By considering gender differences in morning BP patterns and addressing associated risk factors, healthcare professionals can take proactive steps towards reducing the burden of cardiovascular disease and enhancing overall heart health.

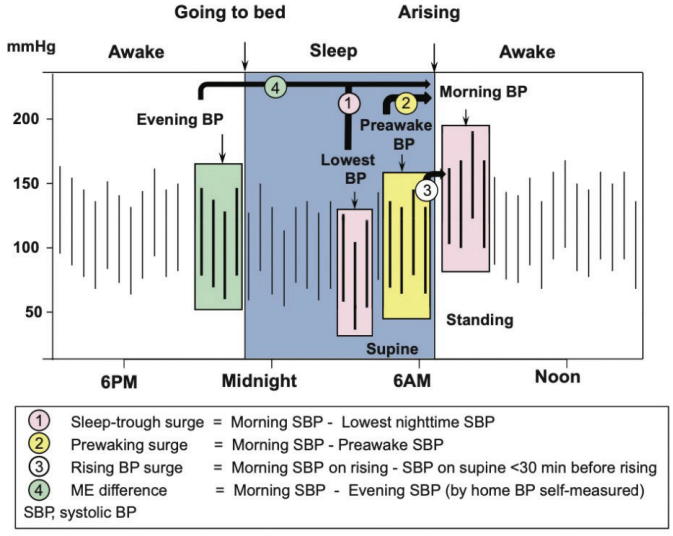

Figure 1. Definition of morning surge in BP.

Table 1. Morning BP surge parameters by gender

	Male (n =107)	Female (n=104)	P value
Sleep through surge	17.34 ± 13.53	13.45 ± 11.86	0.028
Prewaking surge	8.26 ± 14.91	4.57 ±13.80	0.064
Rising blood pressure surge	6.78 ± 18.06	2.81 ± 15.37	0.088
Morning evening difference	9.00 ± 10.05	6.20 ± 8.37	0.029

Hypertension

OP-061

The effect of night shift on blood pressure in healthcare workers

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Background and Aim: The global burden of hypertension is increasing due to an aging population. Today, we see an increase in blood pressure due to various internal and external reasons, not only in the advanced age group, but also in young people. There is an active effect of diet, physical and mental stressors, and social lifestyle on arterial pressure. In this study, we aimed to observe the susceptibility to high blood pressure in occupational groups who work at night.

Methods: We designed our study with participants who have at least 6 night shifts per month. Doctors, nurses and other health workers included the study group. Volunteers who did the same job as the study group, during daylight hours were evaluated as the control group. Participants without a diagnosis of hypertension, atherosclerotic disease, kidney disease or any vascular history were included in the study. Also participants with chronic diseases or

who received antihypertensive medical treatment for any reason were not included in the study. A total of 114 participants were included in the study. We made the follow-ups using a blood pressure holter device. Holter recordings were made at least 48 hours.

Results: A total of 114 participants, 55 of them in the study group, 59 of them were followed as the control group. When we compared the two groups, no significant difference was observed between daylight, night and mean of systolic and diastolic pressures and body mass index. The statistical difference between the ages is remarkable (p=0.001). While the age is 31.3 ± 7.2 in the group working without shift, it is 27.2 ± 2.2 in the group working on shifts. Other significant statistical differences are in the differences between the rates of daylight and night blood pressure (systolic and diastolic p values respectively, 0.006 and 0.005). Systolic daylight-night difference was -5.7 ± 5.5% in the study group and -9.0 ± 7.0% in the control group. Diastolic daylight-night difference was -7.9 ± 9.6% in the study group and -12.7 ± 8.2% in the control group.

Conclusions: The current literature suggests that poor sleep conditions may be an essential risk factor implicated the pathophysiological abnormalities of hypertension and related comorbid states. The occurrence of hypertension is intricately linked to the dysregulation of the autonomic nervous system. Sleep plays an important role in maintaining homeostasis to regulate the stress system. Poor sleep quality may affect the development of hypertension through the activation of the sympathetic nervous system and proinflammatory pathways. The negative effect of night shift on blood pressure cannot be evaluated alone; it should be evaluated as a cumulative and a major effect for the future with other risk factors. Night shifts are an unavoidable reality in the modern world. However, with more scientific studies on this subject in the future, the risk factors for this condition can be revealed more clearly. Thereby introducing a new element in cardiovascular risk assessment.

Table 1. Comparative data of the two groups.

Variables	NIGHT SHIFT		P value
	NO Mean ±Standard Deviation	YES Mean ±Standard Deviation	
Age	31,3±7,2	27,2±2,2	**0,001**
Systolic daylight, mmhg	117,3±10,9	114,8±7,2	0,165
Systolic night, mmhg	107,6±15,9	108,1±8,3	0,831
Systolic mean, mmhg	114,5±10,4	112,6±6,7	0,262
Diastolic daylight, mmhg	73,3±9,4	72,5±6,6	0,573
Diastolic night, mmhg	63,7±8,4	66,4±7,0	0,064
Diastolic mean, mmhg	71,1±9,1	69,9±6,2	0,406
Systolic daylight-night difference (dipper) (%)	-9,0±7,0	-5,7±5,5	**0,006**
Diastolic daylight-night difference (dipper) (%)	-12,7±8,2	-7,9±9,6	**0,005**
Body-mass index	22,0±2,3	22,0±3,2	0,935

Other

OP-062

A telemetry monitoring system for postoperative surgical patients to predict rhythm disorders

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Background and Aim: The aim of this study is to demonstrate the utility of telemetry rhythm monitoring in detecting clinically inapparent episodes of rhythm disorders in postoperative cardiac surgical patients. Rhythm disorders occurs in 10% to 65% of patients after cardiac surgery, usually on the second or third postoperative day. Postoperative rhythm disorders such as atrial fibrillation is associated with increased morbidity and mortality and longer, more expensive hospital stays.

Methods: The data for this research have been drawn from 201 cardiac surgery patients in Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Education and Research Hospital, Division of Cardiovascular Surgery during October 2021-March 2022. The monitoring was conducted using a wearable telemetry ECG device, a mobile application, and a web application. The wearable telemetry ECG device known as iQardi, is designed & developed by Hayriya Information Technology and Health Technologies Inc., a Vestel Company, under the brand name iSina and was individually provided to patients in conjunction with the iQardi mobile application. The web application developed within the scope of the study enabled the monitoring of all patients in the ward on a single screen. Our intention was to offer a clinical decision support system that would be able to early predict rhythm disorders by using machine learning algorithms.

Results: Totally 204 individuals who participated in the study, 172 (84%) had various medical histories. Of these patients, 119 (69%) were male and 53 (31%) were female. Among the patients participating in the study, those with hypertension as a comorbidity constitute 56% of all patients. Of these patients, 76 (68%) are male and 38 (32%) are female. Arrhythmias were more frequently observed in these patients compared to others. Thirty-one patients have chronic atrial fibrillation (AF) (14%). Women comprise 62% of these patients, while men comprise 38%.

Conclusions: The number of patients who had normal sinus rhythm in the pre-op period and developed arrhythmia in the post-op period was 26 (12.74%). In the post-op period, 17 (8.3%) patients were diagnosed with arrhythmia and given an early warning alarm. The outputs of the developed iSina CPMS (Central Patient Monitoring System) artificial intelligence algorithm were compared with physician opinion and conventional methods and then for the atrial fibrillation 98.2% accuracy rate was detected. The findings show that acute rhythm disorders such as atrial fibrillation can be predicted accurately by telemetry rhythm monitoring systems.

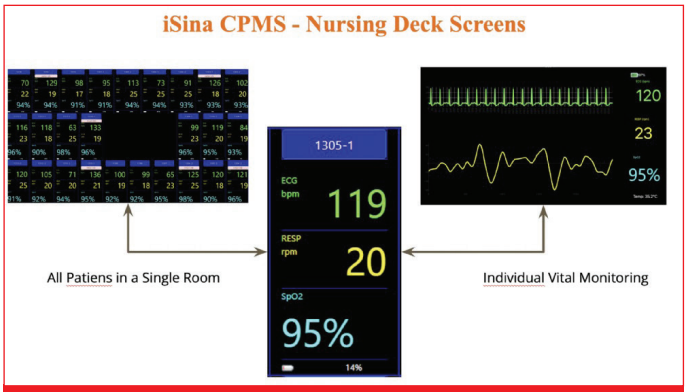

Figure 1. iSina CPMS-Nursing deck screen.
A web-based software was developed to monitor the health status of 26 patients at the same time in the cardiovascular surgery ward. This software, located at the nurse's desk station, allows for the monitoring of data for all patients on the ward through a single screen. The layout of the information on the screen is organized according to the floor plan of the ward. Data is updated at 10-second intervals. The system generates an alarm when it detects an abnormal health status in patients and displays this alarm on the screen.

Table 1. Demographic and BMI characteristics of the participants

Features	Frequency (n)	Percent (%)
Male	144	70.5
Female	60	29.5
Adults (18-65)	120	58.8
Older Adults (65+)	84	41.2
Underwieght (<18.5)	1	0.4
Normal (18.5-24.9)	51	25.0
Overweight (25-29.9)	93	45.5
Obese (>30)	59	28.9

A total of 204 patients who underwent open-heart surgery and were over 18 years of age participated in the study. The ages of the patients ranged from 33 to 81. The distribution of the study participants according to demographic variables and BMI is shown in Table 1.

Table 2. Distribution of comorbid diseases of the participants by gender

	Male	Female	Total	Total
Comorbidities	Frequency (n)	Frequency (n)	Frequency (n)	Percent (%)
Hypertension	76	38	114	55.8
Diabetes	64	38	102	50.0
COPD	7	5	12	5.8
Chronic renal failure	4	3	7	3.4
Atrial fibrillation	12	19	31	15.1

Out of the 204 individuals who participated in the study, 172 (84%) had various medical histories. Of these patients, 119 (69%) were male and 53 (31%) were female. The distribution of comorbidities and their breakdown by gender are shown in Table 3.

Other

OP-063

HEARRT-C score: Mortality predictor in COVID-19 patients

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Background and Aim: In this study, the goal is to determine predictors of mortality using fundamental clinical, biochemical and radiological parameters in patients who require hospitalization due to COVID-19.

Methods: Between 01/03/2020 and 31/12/2021, 1068 patients admitted to Dokuz Eylül University Pandemic Clinic with positive COVID-PCR test were retrospectively screened and 743 patients were excluded due to missing data. The study included 325 hospitalized patients. Demographic data, clinical features at presentation and thorax computed tomography (CT) parameters were analyzed. The weston scoring method, which has been shown to correlate with the Agatston score, was used for the visual coronary calcium score (CACs) (Figure 1). Aortic calcification was defined as involvement above 130 Hounsfield units (HU) density and a minimum volume of 0.5 mm 3 from the ascending aorta to the diaphragm. Patients were followed for in-hospital and post-discharge mortality. Predictors of mortality were determined by multivariate logistic regression analysis and scoring was performed according to risk coefficients. The cut-off value for the score was determined by ROC analysis.

Results: The 325 patients were followed up for a mean of 12.9 ± 8.9 months. Mortality occurred in 104 (32%) patients during follow-up. Demographic and clinical data of the patients are presented in Table 1. The predictors of all-cause mortality were; age >67 years (OR: 2.8, p=0.012), hsTroponin >9.5 ng/mL on admission (OR: 6, p=0.001), heart rate >100 v/min on admission (OR: 4, p=0.002), saturation <90% (OR: 3.5, p=0.001), visual CAC score >1+ presence of aortic calcification (OR: 3.8, p=0.001), hemoglobin <12 mg/dL (OR: 3.8, p<0.001), not taking ASA on admission (OR: 15.6, p<0.001) (Table 2). All prognostic predictors were scored according to 'Odds Ratio' values and HEARRT-C score was created. According to other mortality predictors, hsTroponin >9.5 ng/mL on admission increased the risk 2-fold and not receiving ASA on admission increased the risk 5-fold (Table 3). As a result of ROC analysis the predictive value for the HEARRT-C score was determined as 8. It was found to be significant in all-cause mortality with 80% sensitivity and 86% specificity [AUC: 0.90 (95% CI: 0.86-0.94) p≤0.001]. HEARRT-C score ≥8 showed a 26-fold increase in all-cause mortality (OR: 26.7 95% CI: 14.96-49.77, p<0.001).

Conclusions: Mortality is high in COVID-19 patients, therefore early diagnosis and treatment is important. The developed HEARRT-C score has a high predictive value in all-

cause mortality due to COVID-19. In addition to classical risk factors, CAC score, aortic calcification and hsTroponin level at presentation have been shown to be effective on all-cause mortality. ASA has been shown to be highly protective in these patients. This may be due to the fact that ASA is effective against complications of COVID-19 such as prothrombosis, inflammation and endothelial dysfunction. The HEARRT-C score needs to be validated with large-scale studies.

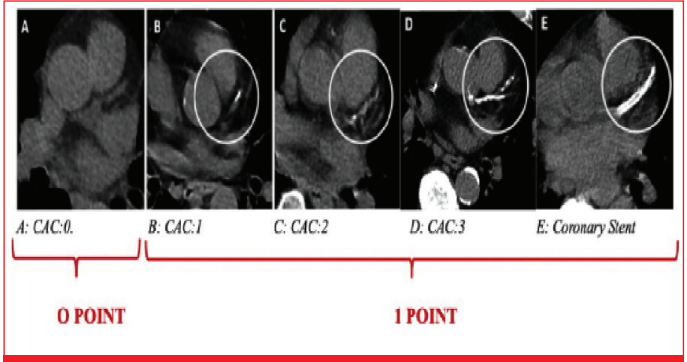

Figure 1. Visual Coronary Calcium Score Calculation with Weston Score: Visual CAC Calculation for LAD (Area within the white ring)

Table 1. Demographic and Clinical Data of the Patients Included in the Study

Demographical and Clinical Data	
Age, Years	63±16
Sex, Female, /n(%)	135(%41.5)
Hypertension, n(%)	192 (%59.1)
Diyabetes Mellitus, n(%)	101 (%31.1)
Chronic Kidney Disease, n(%)	66 (%20.3)
Hyperlipidemia, n(%)	51 (%15.7)
Smoking, n(%)	148 (%45.5)
Coronary Artery Disease, n(%)	43 (%13.2)
ECG, Sinus Rythm, n(%)	277 (%85.2)
Heart Rate>100 bpm, n(%)	86 (%26.5)
ASA users, n(%)	56 (%17.2)
Thorax CT-Covid (moderate-heavy)	345 (%75)
Thorax CT - CAC Score>1	184 (%56)
Thorax CT T-Aort calcification+	191 (%58)
Hemoglobin	12±2.2 gr/dl
hsTroponin (on admission)	7±1438 ng/L
D-Dimer	1.4±6.8µg/mL
Saturation	%92±4.9

Table 2. Results Of Univariate And Multivariate Logistic Regression Analysis For All-cause Mortality Predictors

	UNIVARIATE LOGISTIC REGRESSION ANALYSIS			MULTIVARIATE LOGISTIC REGRESSION ANALYSIS		
Variables	**OR**	**%95 CI**	**p**	**OR**	**%95 CI**	**p**
Age >67 years	5.67	3.39-9.46	<0.001	2.8	1.25-6.24	=0.012
HT	1.89	1.15-3.1	<0.011			
CKD	4.31	2.44-7.58	<0.001			
CAD	2.05	1.06-3.93	=0.031			
ECG, SR+/-	2.87	1.54-5.33	=0.001			
Heart Rate >100 bpm,	4.43	2.67-7.42	<0.001	4.02	1.78-9.06	=0.001
Saturation < %90	4.63	2.81-7.63	<0.001	3.52	1.72-7.19	=0.001
Thorax CT-Covid	2.47	1.33-4.59	<0.001			
Thorax CT - CAC +/-	5.0	2.9-8.8	<0.001			
Thorax CT -Aort +/-	5.47	3.08-9.70	<0.001			
Thorax CT +/- ve Aort +/-	5.28	3.13-8.91	<0.001	3.85	1.69-8.78	=0.001
Thorax CT - EFT +/-	2.07	1.26-3.25	=0.003			
Hemoglobin<12	5.32	3.19-8.86	<0.001	3.80	1.87-7.68	<0.001
hsTrop>9.5 (on admission)	9.82	5.68-16.98	<0.001	6.03	2.90-12.52	<0.001
ASA -/+ (not receiving ASA on admission)	7.69	2.70-21.90	<0.001	15.61	4.46-54.62	<0.001

Table 3. HEARRT-C Scoring System

Hemoglobin <12mg /dL	**1 point**
Elderly (age) >67	**1 point**
ASA not receiving	**5 point**
Rate (heart rate >bpm)	**1 point**
Respiration (saturation <%90)	**1 point**
hs**Trop**≥9.5 (on admission)	**2 point**
CAC>1 +Aort calcification+	**1 point**
Total Point:	**>8**

Heart Valve Diseases

OP-064

Comparison of SYNTAX II Score, EUROSCORE and STS scores for perioperative and in-hospital mortality prediction in patients who underwent TAVI (transaortic valve implantation) procedure for severe aortic stenosis

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Background and Aim: Aortic stenosis (As) is a common valvular disease and transcatheter aortic valve implantation (TAVI)

is increasingly used to treat patients at high risk for conventional surgery. As the procedure becomes widespread and the number of cases increases, it necessitates a predictive scoring against complications that may develop during and after the procedure. In this study, we compared Syntax Score II (SS II) which are used in the diagnosis, staging and treatment of coronary artery disease with conventional risk scores; EUROSCORE and STS, in terms of predicting in-hospital mortality.

Methods: Seventy patients who underwent TAVI between 2011 and 2018 in our hospital for aortic stenosis were included in the study. Patients were divided into two groups according to in-hospital mortality development and the relationship between risk scores and mortality was investigated.

Results: 58.6% of the patients were male and the mean age was 77.4 ± 10.1 years. The patients were divided into two groups who died in the hospital and were discharged safely. In univariate analysis, STS score, EUROSCORE and SS II, left ventricular systolic diameter and mean aortic gradient was high in the group at which patients were death. The incidence of postoperative complications was similar in both groups (p=0.22 for stroke, p=0.50 for acute renal failure, p=0.49 for pacemaker implantation). In the regression analysis, EUROSCORE was found to be an independent predictor of in-hospital mortality in TAVI patients (p=0.05). The other two scores were not statistically significant.

Conclusions: EUROSCORE has significantly better predictive power among the risk scores commonly used in predicting perioperative and in-hospital mortality in patients undergoing TAVI. However, we believe that there is a need for new risk scoring systems which can make better predictions specific to TAVI.

Cardiovascular Surgery

OP-065

Systemic inflammation index as a predictor of saphenous vein graft disease in patients with coronary bypass

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Background and Aim: The systemic inflammation index (SII) has been considered a good index that reflects the local immune response and systemic inflammation. This study aimed to observe the relationship between SII and saphenous vein graft disease.

Methods: A total of 422 patients have saphenous vein graft were retrospectively included in our study. 222 patients were with saphenous vein graft disease (SVGD) and 200 of them were with patent saphenous vein graft.

Results: SII was higher in the SVGD group than the control group (631.55 ± 397.84, 421.71 ± 351.07, p=0.001). ROC analysis was performed to identify the optimal cut-off point with the highest sensitivity and specificity. The optimal cut-off point for SII was defined as 430. Using a cutoff level of >430, SII predicted SVGD with a sensitivity of 73% and specificity of 56%.

Conclusions: SII can be used to predict saphenous vein disease and patency time in patients with saphenous grafts. SII is an inexpensive, easily accessible and highly reliable parameter for patients with saphenous vein graft.

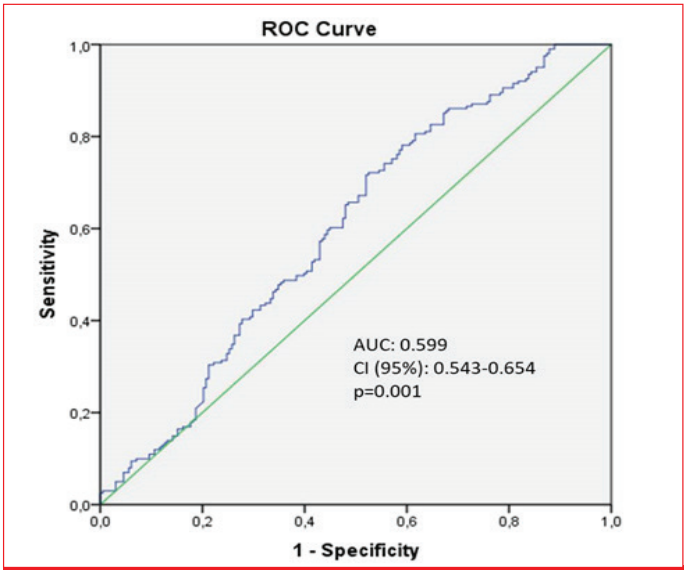

Figure 1. Receiver–operating characteristic (ROC) curves of SII predicting saphenous graft vein disease

Table 1. Basal characteristics of the patients during the index coronary angiography

	SVGD (222)	Control Group[a] (200)	P value
Male Gender, n (%)	192 (86.5)	175 (87.5)	0.757
Age, years (mean±SD)	66.32+9.48	67.38+10.38	0.329
Hypertension, n (%)	187 (84.2)	160 (80)	0.256
Diabetes Mellitus, n (%)	121 (54.4)	91 (45.5)	0.065
Hyperlipidemia, n (%)	148 (66.7)	114 (57)	0.041
Peripheral Arterial Disease, n (%)	20 (9)	22 (11)	0.495
Chronic Kidney Disease, n (%)	65 (29.3)	40 (20)	0.028
Cerebrovascular accident, n (%)	14 (6.3)	19 (9.5)	0.222
Smoking, n (%)			
Active	70 (31.5)	48 (24)	0,312
Ex-smoker	20 (9)	12 (6)	
Never	132 (59.5)	140 (70)	
Medical Treatment, n (%)			
Antiaggregan	*201 (90.5)*	182 (91)	0.817
Anticoagulant	*25 (11.3)*	17 (8.5)	0.344
Beta Blocker	*178 (80.2)*	149 (74.5)	0.163
ACE/ARB	*148 (66.7)*	93 (46.5)	0,001
Spironolactone	*46 (20.7)*	17 (8.5)	0,001
Statin	*140 (63.1)*	97 (48.5)	0.003
Calcium Channel Blocker	*48 (21.6)*	35 (17.5)	0.228

Abbreviations: ACE/ARB, angiotensin converting enzyme/ angiotensin receptor blocker ; SVGD, saphenous vein graft disease.

[a]Patients with patent saphenous vein grafts without degeneration.

Table 2. Graft Details and Laboratory Parameters of Groups.

	SVDG	Control Group[a]	P value
Age of saphenous vein graft (mean±SD)	8.67±6.76	7.20±5.90	**0.018**
Saphenous Graft (mean±SD)	2.09±0.84	1.49±0.82	
Degenerated Saphenous (mean±SD)	1.50±0.697	0	
Hemoglobin, mg/dl (mean±SD)	13.27±1.89	13.75±1.65	0.099
WBC, x 1000µL (median,IQR)	7.57 (3.01)	7.5 (2.12)	0.274
Platelet, x 1000µL (median,IQR)	207 (90)	210.50 (75.5)	0.329
Total cholesterol, mg/dl (median,IQR)	160 (53.5)	168 (51)	0.224
LDL, mg/dl (median,IQR)	90 (35.5)	100.5 (33.5)	0.205
HDL, mg/dl (mean±SD)	43.3 ± 12.58	43.62 ± 12.31	0.875
Triglyceride, mg/dl (mean±SD)	136.09 ± 68.68	149.69 ± 78.92	0.258
SII (mean±SD)	631.55 ± 397.84	421.71 ± 351.07	**0.001**

Abbreviations: HDL, high density lipoprotein; LDL, low density lipoprotein; SII, systemic inflammation index ;SVGD, saphenous vein graft disease; WBC, white blood cell

[a] Patients with patent saphenous vein grafts.

Table 3. Multivariate Logistic Regression Analysis for Saphenous Vein Graft Disease

Possible Confounding Factor	Odds Ratio	95% Confidence Interval	P value
Diabetes Mellitus	1.319	0.858-2.025	0.207
Hyperlipidemia	1.309	0.845-2.028	0.227
Chronic Kidney Disease	1.497	0.910-2.461	0.112
ACE/ARB	2.034	1.312-3.152	**0.001**
Spironolactone	1.703	0.883-3.284	0.112
Statins	1.678	1.089-2.585	**0.019**
Age of saphenous vein graft	1.036	1.001-1.072	0.042
Active smoking	1.088	0.671-1.765	0.732
SII > 430	2.074	1.337-3.219	**0.001**

Abbreviations: ACE/ARB, angiotensin converting enzyme/ angiotensin receptor blocker; SII, systemic inflammation index

Other

OP-066

The relationship of cardiac syndrome X and obstructive sleep apnea syndrome and the effects of sleep apnea treatment on myocardial ischemia

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Background and Aim: Cardiac syndrome X is a disease that does not have an obvious effect on long-term mortality but creates serious physical and psychiatric morbidity. Cardiac syndrome X shows significant heterogenity in etiopathogenesis. Obstructive sleep apnea syndrome (OSAS) is common in the community and has close relations with many cardiovascular diseases. Although OSAS is associated with many cardiovascular diseases, its relation with cardiac syndrome X is not clear. Our aim is to investigate the relationship of OSAS with cardiac syndrome X and the effects of CPAP treatment on myocardial ischemia.

Methods: 27 patients which diagnosed as cardiac syndrome X after coronary angiography were included. Seventeen patients with OSAS detected in their polysomnography and agreed to participate to the treatment phase of the study were given CPAP treatment for 3 months. After 3 months of treatment, myocardial perfusion scintigraphy (MPS) was performed again and patients were evaluated whether there was a decrease in myocardial ischemia compared to the pre-treatment scintigraphy. A 20-segment left ventricular model was used to evaluate the extensity of myocardial ischemia. Chi-square and Fisher's precision test were applied to independent groups for categorical data in the comparison of variables. The independent samples t-test was used for independent groups with normal distribution, and bilateral Mann-Whitney U-test was used for independent groups that did not show normal distribution. Logistic regression analysis was performed to identify independent predictors of ischemia recovery.

Results: In cardiac syndrome X patients who participated in our study, OSAS was detected in 24 of 27 (88.9%) polysomnographic examinations. After 3 months of treatment, myocardial ischemia was decreased in 13 of 17 patients (76.4%) who accepted to continue with treatment. A statistically significant correlation was found between the patient's diagnosis of hypertension, higher serum HDL cholesterol level, and adherence to CPAP therapy and reduction in myocardial ischemia.

Conclusions: The frequency of OSAS is prominently high in cardiac syndrome X patients. In cardiac syndrome X patients who have OSAS, obvious improvement in myocardial ischemia detected in MPS's after CPAP treatment. There is a need for larger studies to investigate the relationship between OSAS, treatment effect and cardiac syndrome X.

Heart Valve Diseases

OP-067

Assessment of the multiple genetic variants associated with prosthetic valve thrombosis

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Background and Aim: The risk of prosthetic valve thrombosis (PVT) may increase with inadequate anticoagula-

tion, the thrombogenicity of the valve, and hemodynamic abnormalities. Also, some inherited genetic problems, such as MTHFR A 1298 C and heterozygous mutations of fibrinogen and 455 G-A, may cause recurrent PVT. We aim to assess the possible contribution of genetic abnormalities to the occurrence of PVT with one of the largest series in the literature.

Methods: This single-center retrospective study has two groups, including patients with normal prosthetic valves and PVT. All patients were evaluated with 3D-RT transesophageal echocardiography. The genetic panel was examined from blood samples in all cases; however, the specific composition of the panel differed depending on the kit utilized. The local ethics association approved the study, and the patients were enrolled after informed consent.

Results: Among the genetic panels examined, Factor II/Prothrombin (G20210A), Factor V Leiden (G1691A), MTHFR (C677T), MTHFR (A1298C), Factor XIII (G103T) (V34L), PAI-I (5G>4G), ACE I/D (I/D alleles), APO E GENOTIP (E2, E3, E4 isoforms), ApoE112, and APOE158 provided Hardy-Weinberg Equilibrium consistency. Beta fibrinogen (455 G>A), HPA-1 GPIIIa, APO B-100, and Factor V R2 did not provide Hardy-Weinberg equilibrium consistency. Significant differences were detected in the Factor II/Prothrombin (G20210A), HPA-1 GPIIIa (T196C), and Factor V R2 (A4070G) (H1299R) genotype ratios of the thrombosis and control patient groups (p=0.044, p=0.034, p=0.016). The factor II/prothrombin (G20210A) heterozygous ratio in thrombosis patient groups was statistically significantly higher than the control group. Univariate Logistic Regression Analysis found heterozygous Factor V R2 was a protective factor (p=0.012, OR: 0.263). Factors with p<0.250 (Protrombin G20210A, FactorVR2 detected) in univariate analysis were corrected for age and gender; female gender was found to be 2.194 times the risk factor compared to male gender. Older age was 0.968 times, and being heterozygous for Factor V R2 was 0.203 times the protective factor compared to those with normal Factor V R2.

Conclusions: PVT might be associated with inherited genetic abnormalities. Our study finds that the male gender, older age, and heterozygote Factor V R2 polymorphism were protective factors in developing PVT. Factor V-R2 is a genetic anomaly associated with arterial and venous thrombosis. However, the carriers of the heterozygote FV2 polymorphism had a decreased risk of developing PVT in our study. This might be attributed to the fact that Factor V R2 did not provide Hardy-Weinberg equilibrium consistency. Also, in the thrombosis patient groups, the ratio of heterozygous factor II/prothrombin was higher than in the control group. All of the thrombosis patients who had homozygous HPA-1 GPIIIa were in the PVT group. This study is one of the largest series in the literature. Moreover, larger studies are needed to comprehensively detect genetic abnormalities in PVT.

Table 1. The table explains the previous diagnosis, valve types, and clinical features on the admission of the patients.

		Tromboz Grup	Kontrol Grup	
		n / N (%)	n / N (%)	p#
Cinsiyet	Kadın	125 / 175 (71,4)	57 / 101 (56,4)	0,011
	Erkek	50 / 175 (28,6)	44 / 101 (43,6)	
Yaş Ort.±SD Min-Maks (Median)		49,8±13,1 18-77 (52)	55,4±12,2 29-79 (58)	0,002*
AVR		49 (27,9)	49 (48,5)	0,003
MVR		157 (89,7)	72 (71,2)	<0,001
Kapak	STJ	51 / 100 (51,0)	24 / 100 (44,4)	0,357
	ML	1 / 100 (1,0)	1 / 100 (1,9)	
	MEDTRONIC	5 / 100 (5,0)	0 / 100 (0,0)	
	CM	24 / 100 (24,0)	19 / 100 (35,2)	
	ATS	9 / 100 (9,0)	7 / 100 (13,0)	
	Sorin	9 / 100 (9,0)	3 / 100 (5,6)	
ETSVS Ort.±SD Min-Maks (Median)		122,9±89,1 6-566 (95)	94,1±55,8 22-252 (81)	0,110*
Clexane		8 / 132 (6,1)	2 / 54 (3,7)	0,726
INR Ort.±SD Min-Maks (Median)		2,59±0,86 1-6,09 (2,52)	2,81±1,04 1,17-7,3 (2,735)	0,172*
Gebelik		12 / 124 (9,7)		
OT		103 / 172 (59,2)		
NOT		69 / 174 (40,1)		

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Table 2. Table shows Hardy-Weinberg Equilibrium consistency of genetic panels.

Genetik Paneller		n	%	Hardy-Weinberg Equilibrium
Faktor II/Protrombin (G20210A)	Normal	248	95,0%	X^2:3,711
	Homozigot	1	0,4%	p=0,055
	Heterozigot	12	4,6%	
Faktor V Leiden(G1691A) (R506Q)	Normal	219	84,6%	X^2:2,903
	Homozigot	4	1,5%	p=0,088
	Heterozigot	36	13,9%	
MTHFR(C677T)	Normal	125	47,9%	X^2:1,415
	Homozigot	19	7,3%	p=0,234
	Heterozigot	117	44,8%	
MTHFR(A1298C)	Normal	126	48,5%	X^2:0,000
	Homozigot	24	9,2%	p=0,999
	Heterozigot	110	42,3%	
Faktor XIII (G103T) (V34L)	Normal	146	70,9%	X^2:1,458
	Homozigot	8	3,9%	p=0,227
	Heterozigot	52	25,2%	
Beta Fıbrınogen-455 G>A)	Normal	126	64,3%	X^2:4,828
	Homozigot	2	1,0%	p=0,028
	Heterozigot	68	34,7%	
PAI-I (5G>4G)	Normal	73	30,3%	X^2:0,694
	Homozigot	43	17,8%	p=0,405
	Heterozigot	125	51,9%	
HPA-1 GPIIIa (T196C)	Normal	147	73,9%	X^2:10,238
	Homozigot	11	5,5%	p=0,001
	Heterozigot	41	20,6%	
ACE I/D (I/D allels)	D/D	60	30,5%	X^2:0,008
	I/I	39	19,8%	p=0,929
	I/D	98	49,7%	
APO B-100 (G107084A)	Normal	195	98,5%	X^2:125,911
	Homozigot	2	1,0%	p<0,001
	Heterozigot	1	0,5%	
APO E GENOTIP (E2, E3, E4 isoforms)	E3/E3	125	74,9%	X^2:3,455
	E3/E4	17	10,2%	p=0,063
	E2/E3	25	15,0%	
ApoE112	Normal	24	96,0%	X^2:0,010
	Heterozigot	1	4,0%	p=0,919
APOE158	Normal	24	96,0%	X^2:0,010
	Heterozigot	1	4,0%	p=0,919
Faktor V R2(A4070G) (H1299R)	Normal	206	91,6%	X^2:5,120
	Homozıgot	2	0,9%	p=0,024
	Heterozıgot	17	7,6%	

Table 5. Thrombosis risk effects of genetic panels multivariate logistic regression analysis.

		p	OR	95% C.I.	
Cinsiyet (Ref:Erkek) Kadın		0,015	2,194	1,168	4,120
Yaş		0,013	0,968	0,943	0,993
Faktor II Protrombin G20210A (Ref:Normal)		0,440			
	Homozigot	1,000			.
	Heterozigot	0,200	3,984	0,481	33,010
FaktorVR2 (Ref:Normal)		0,019			
	Homozigot	1,000			.
	Heterozigot	0,005	0,203	0,067	0,617

Hosmer and Lemeshow Test Chi-square:9,661 p=0,290 Cox & Snell R Square:0,119

Table 3. Thrombosis risk effects of genetic panels.

		Tromboz Grup		Kontrol Grup		
		n	%	n	%	p
Faktor II/Protrombin (G20210A)	Normal	149	92,5%	99	99,0%	0,044
	Homozigot	1	0,6%	0	0,0%	
	Heterozigot	11	6,8%	1	1,0%	
Faktor V Leiden(G1691A) (R506Q)	Normal	130	82,3%	89	88,1%	0,204
	Homozigot	4	2,5%	0	0,0%	
	Heterozigot	24	15,2%	12	11,9%	
MTHFR(C677T)	Normal	77	47,8%	48	48,0%	0,515
	Homozigot	14	8,7%	5	5,0%	
	Heterozigot	70	43,5%	47	47,0%	
MTHFR(A1298C)	Normal	80	49,7%	46	46,5%	0,451
	Homozigot	17	10,6%	7	7,1%	
	Heterozigot	64	39,8%	46	46,5%	
Faktor XIII (G103T) (V34L)	Normal	89	67,9%	57	76,0%	0,449
	Homozigot	6	4,6%	2	2,7%	
	Heterozigot	36	27,5%	16	21,3%	
BETA FIBRINOGEN-455 G>A)	Normal	78	61,4%	48	69,6%	0,387
	Homozigot	2	1,6%	0	0,0%	
	Heterozigot	47	37,0%	21	30,4%	
PAI-I (5G>4G)	Normal	48	31,8%	25	27,8%	0,507
	Homozigot	29	19,2%	14	15,6%	
	Heterozigot	74	49,0%	51	56,7%	
HPA-1 GPIIIa (T196C)	Normal	93	72,7%	54	76,1%	0,034
	Homozigot	11	8,6%	0	0,0%	
	Heterozigot	24	18,8%	17	23,9%	
ACE I/D (I/D allels)	I/I	25	20,2%	14	19,2%	0,880
	I/D	60	48,4%	38	52,1%	
	D/D	39	31,5%	21	28,8%	
APO B-100 (G107084A)	Normal	123	98,4%	72	98,6%	0,284
	Homozigot	2	1,6%	0	0,0%	
	Heterozigot	0	0,0%	1	1,4%	
APO E GENOTIP (E2, E3, E4 isoforms)	E3/E3	94	75,8%	31	72,1%	0,885
	E3/E4	12	9,7%	5	11,6%	
	E2/E3	18	14,5%	7	16,3%	
ApoE112	Normal	4	100%	20	95,2%	1,000
	Heterozigot	0	0,0%	1	4,8%	
APOE158	Normal	4	100,0%	20	95,2%	1,000
	Heterozigot	0	0,0%	1	4,8%	
Faktor V R2(A4070G) (H1299R)	Homozigot	2	1,4%	0	0,0%	0,016
	Heterozigot	6	4,1%	11	14,1%	
	Normal	139	94,6%	67	85,9%	

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Table 4. Univariate logistic regression analysis of thrombosis risk effects of genetic panels.

	p	OR	95,0% C.I.	
Faktor II Protrombin G20210A (Ref:Normal)	0,168			
Homozigot	1,000			.
Heterozigot	0,059	7,309	0,929	57,507
Faktor V Leiden G1691AR506Q (Ref:Normal)	0,710			
Homozigot	0,999			.
Heterozigot	0,407	1,369	0,651	2,880
MTHFRC677T (Ref:Normal)	0,523			
Homozigot	0,313	1,745	0,591	5,155
Heterozigot	0,778	0,928	0,554	1,556
MTHFRA1298C (Ref:Normal)	0,454			
Homozigot	0,492	1,396	0,539	3,618
Heterozigot	0,404	0,800	0,473	1,352
Faktor XIIIG103TV34L (Ref:Normal)	0,453			
Homozigot	0,434	1,921	0,375	9,850
Heterozigot	0,290	1,441	0,733	2,834
Beta fibrınogen455GA (Ref:Normal)	0,607			
Homozigot	0,999			.
Heterozigot	0,317	1,377	0,735	2,580
PAII5G4G (Ref:Normal)	0,508			
Homozigot	0,361	1,323	0,726	2,413
Heterozigot	0,340	1,428	0,687	2,965
HPA1GPIIIaT196C (Ref:Normal)	0,859			
Homozigot	0,999			.
Heterozigot	0,581	0,820	0,405	1,661
ACE allels (Ref:I/I)	0,880			
I/D	0,754	0,884	0,409	1,910
D/D	0,927	1,040	0,448	2,415
APOB100G107084A (Ref:Normal)	1,000			
Homozigot	0,999			.
Heterozigot	1,000	0,000	0,000	.
APOEAPO E (Ref:N E3/E3)	0,885			
E3/E4	0,737	1,179	0,450	3,088
E2/E3	0,921	0,933	0,239	3,638
FaktorVR2 (Ref:Normal)	0,041			
Homozigot	0,999			.
Heterozigot	0,012	0,263	0,093	0,741

Heart Valve Diseases

OP-068

Transcatheter closure or surgery for symptomatic paravalvular leaks: The multicenter KISS registry

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Background and Aim: The optimal treatment of symptomatic paravalvular leaks (PVL) remains controversial between transcatheter closure (TC) and surgery. This study aimed to retrospectively evaluate the long-term outcomes of large-scale patients who underwent re-operation or TC of PVLs.

Methods: A total of 335 [male: 209 (62.4%), mean age: 58.15 ± 12.77 years] patients who underwent treatment of PVL at the three tertiary centers between January 2002 and December 2021. Echocardiographic features, procedure details, technical success, and in-hospital and long-term results were assessed. Technical success was defined as a reduction of regurgitation to no greater than mild PVL with an absence of death, stroke, and device-related prosthetic valve dysfunction. Procedural success was defined as the reduction of regurgitation to no greater than mild PVL with the absence of death, stroke, and any major complications. The long-term primary endpoint was defined as the combination of all-cause death, stroke, or rehospitalizations. The biologically plausible variables as covariates were further entered into the Cox hazard regression analysis, with backward selection, to determine any independent associates of in-hospital mortality and long-term primary endpoint. The cumulative incidence of mortality was calculated with the Kaplan-Meier survival test. The effect of the treatment group on long-term primary endpoint rates was investigated using the Log-rank test. The significance level was accepted as $p<0.05$ in all statistical analyses.

Results: The initial management strategy was TC in 171 (51%) patients and surgery in 164 (49%) cases. Three hundred (89.6%) of all cases were mitral PVL and 35 (10.4%) were aortic PVL. Mean left ventricular ejection fraction was 52.03

± 10.79%. Technical (78.9 vs. 76.2%, p=0.549) and procedural success (73.7 vs. 65.2%, p=0.093) were similar both groups. In-hospital (15.9 vs. 4.7%, p=0.001) and log-term all-cause mortality (43.9 vs. 31.6%, p=0.02) were higher in surgery group. However, the long-term primary endpoint rate was not significant between both groups (48% vs. 57.9%, p=0.068). Being in the surgery group (HR: 3.222, 95% CI: 1.441-7.205; p=0.004) was found to be one of the independent predictors of in-hospital mortality. Moreover, estimated glomerular filtration rate ≤30 (HR: 2.874, p<0.001) and residual greater than mild PVL (HR: 2.690, p<0.001) were also found to be independent predictors of long-term primary endpoint.

Conclusions: This retrospective observational study suggests that percutaneous closure of PVLs was associated with lower early and long-term mortality compared to surgery. Moreover, effective management of these patients requires an integrated team approach that includes both percutaneous and surgical treatment and weighs early versus late treatment on the basis of clinical and anatomic factors. Nevertheless, multicenter randomized trials with clearly defined inclusion and exclusion criteria would raise the level of evidence available to guide the management of patients with PVLs.

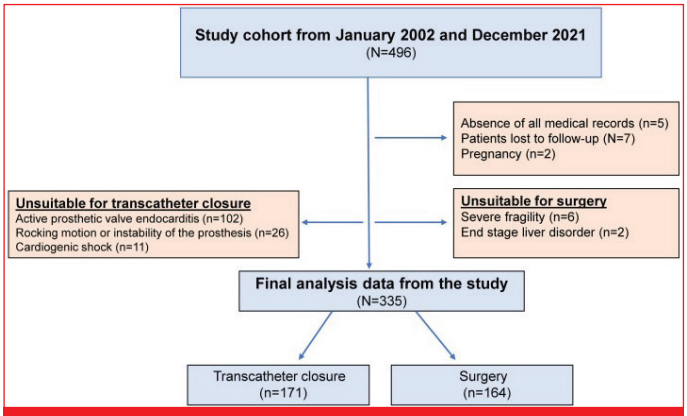

Figure 1. Flow chart for patient selection.

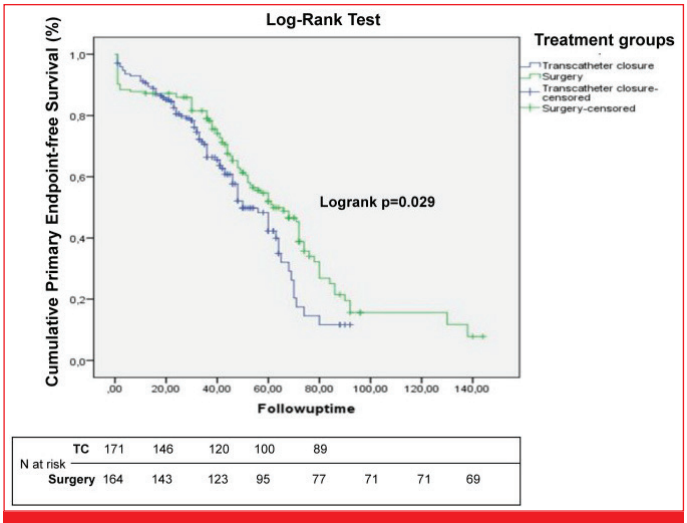

Figure 2. Kaplan-Meier survival analysis for primary endpoint during follow-up.

Table 1.

Variables	Transcatheter closure (n=171)	Surgery (n=164)	p
Valve position of PVL, n (%)			
Aortic	24 (14)	11 (6.7)	0.028
Mitral	147 (86)	153 (93.3)	<0.001
Age, years	63.7 ± 10.03	52.36 ± 12.76	<0.001
Gender (male)	110 (64.3)	99 (60.4)	0.454
Comorbidities, n (%)			
Hypertension	88 (51.5)	49 (29.9)	0.001
Atrial fibrillation	110 (64.3)	77 (47)	0.001
Diabetes mellitus	39 (22.8)	30 (18.3)	0.307
Hyperlipidemia	52 (30.4)	32 (19.5)	0.021
Prior PCI	18 (10.5)	20 (12.2)	0.630
Prior CABG	32 (18.7)	24 (14.6)	0.317
Chronic kidney disease	60 (35.1)	56 (34.1)	0.856
Heart failure	45 (26.3)	48 (29.3)	0.546
Previous endocarditis	14 (8.2)	22 (13.4)	0.123
Chronic pulmonary disease	33 (19.3)	21 (12.8)	0.106
Previous reoperation	29 (17)	35 (21.3)	0.308
History of stroke	14 (8.2)	24 (14.6)	0.063
Smoker	54 (31.6)	45 (27.4)	0.406
Number of sternotomies	1.41 ± 0.62	1.45 ± 0.59	0.391
ETSVS, months	93.91 ± 57.78	109.87 ± 79.03	0.160
STS score (mortality)	4.18 ± 3.29	2.02 ± 1.13	<0.001
Presenting symptoms, n (%)			
HF (NYHA class ≥3)	145 (84.8)	146 (89)	0.252
Hemolysis	137 (80.1)	144 (86)	0.154
HF + Hemolysis	118 (69)	124 (75.6)	0.177
ES transfusion, n (%)	82 (48)	104 (63.4)	0.004
Hospitalization	151 (92.1)	115 (67.3)	<0.001
Valve type, n (%)			
Mechanical	160 (93.6)	155 (94.5)	0.715
Bioprosthetic	11 (6.4)	9 (5.5)	

Baseline demographic and clinical characteristics per study group. Abbreviations: CABG: Coronary artery bypass grafting, ES: Erythrocyte suspension, ETSVS: Elapsed time since valve surgery, HF: Heart failure, NYHA: New York Heart Association, PCI: Percutaneous coronary intervention, PVL: Paravalvular leak, STS: Society of Thoracic Surgeon.

Table 2. Comparison of baseline echocardiographic and laboratory findings between the groups.

Variables	Transcatheter closure (n=171)	Surgery (n=164)	p
Number of aortic PVL, n (%)*			
I	15 (62.5)	7 (63.6)	0.424
II	6 (25)	4 (36.4)	
III	3 (12.5)	0	
Number of mitral PVL, n (%)*			
I	92 (62.6)	91 (59.5)	0.885
II	47 (32)	51 (33.3)	
III	9 (5.9)	6 (4.1)	
IV	2 (1.4)	2 (1.3)	
Aortic PVL grade, n (%)*			
Mild	0	0	-
Moderate	6 (25)	0	0.068
Severe	23 (95.8)	11 (100)	0.492
Number of PVLs ≥2, n (%)*	64 (37.4)	65 (39.6)	0.678
Mitral PVL grade, n (%)*			
Mild	7 (4.8)	8 (5.2)	0.853
Moderate	15 (10.2)	21 (13.7)	0.348
Severe	142 (96.6)	153 (100)	0.021
Location of the aortic PVL, n (%)*			
Non-coronary sinus	11 (6.4)	6 (3.7)	0.247
Left coronary sinus	17 (9.9)	6 (3.7)	0.023
Right coronary sinus	6 (3.5)	3 (1.8)	0.342
Location of the mitral PVL, n (%)*			
Medial	38 (22.2)	44 (26.8)	0.327
Posterior	45 (27.4)	39 (22.8)	0.328
Anterior	62 (36.3)	64 (39)	0.601
Lateral	66 (38.6)	64 (39)	0.936
LVEF, %	52.71 ± 10.96	51.31 ± 10.59	0.145
LAD, mm	49.53 ± 7.38	48.03 ± 5.14	0.633
LVEDD, mm	54.16 ± 7.49	53.17 ± 6.5	0.020
LVESD, mm	37.57 ± 8.01	37.77 ± 8.28	0.888
Estimated sPAB, mmHg	45.85 ± 15.99	43.26 ± 16.31	0.046
Severe TR, n (%)	72 (42.1)	65 (39.6)	0.646
RV dysfunction, n (%)	67 (39.2)	49 (29.9)	0.074
TAPSE, mm	18.43 ± 3.42	19.11 ± 3.07	0.048
Laboratory measurements			
Creatinine, mg/dL	1.11 ± 0.58	1.14 ± 0.52	0.262
LDH, U/L	898.58 ± 567.67	922.12 ± 559.28	0.569
Total bilirubin, mg/dL	1.64 ± 1.02	1.61 ± 0.98	0.928
White blood cell count, 10^9/L	7.48 ± 2.14	7.94 ± 2.79	0.212
Hemoglobin, g/dL	10.13 ± 1.87	9.71 ± 1.57	0.069
Platelet, 10^9/L	228.94 ± 82.25	246.47 ± 97.43	0.125
CRP, mg/dL	8.25 ± 6.96	7.45 ± 4.54	0.969

* "n" donates the number of PVLs rather than the number of patients with PVLs Abbreviations: CRP: C-reactive protein, LAD: Left atrial diameter, LDH: Lactate dehydrogenase, LVEDD: Left ventricular end diastolic diameter, LVEF: Left ventricular ejection fraction, LVESD: Left ventricular end systolic diameter, PVL: Paravalvular leak, RV: Right ventricle, sPAP: Systolic pulmonary artery pressure, TAPSE: Tricuspid annular plane systolic excursion.

Table 3. Procedure details of transcatheter PVL closure

Parameters	Transcatheter closure (n=171)
Technical success	135 (78.9)
Mitral closure technique, n (%)*	
Transseptal without	80 (46.8)
VA rail Transseptal with	58 (33.9)
VA rail Transapical	9 (5.3)
Aortic closure technique, n (%)*	
Retrograd	24 (14)
Transcatheter closure device, n (%)	
AVP II	11 (6.4)
AVP III	140 (81.9)
AVP IV	3 (1.8)
ADO II	1 (0.6)
ADO III	11 (6.4)
Amplatzer mVSD occluder	1 (0.6)
OCCLUTECH	12 (7)
ASO	10 (5.8)
Number of device placed	1.43 ± 0.60
Procedure time, min	132.30 ± 44.31
Contrast media volume, mL	19.65 ± 49.46

Footnote: * "n" donetes the number of patients with PVL rather than the number of PVL. ADO: Amplatzer ductal occluder, AVP: Amplatzer vascular plug, ASO: Amplatzer septal occluder, VA: Venoarterial, mVSD: Muscular ventricular septal defect.

Table 4. Procedure details of surgical PVL closure

Parameters	Surgical PVL closure (n=164)
Technical success	125 (76.2)
Surgical PVL closure technique, n (%)	
Valve replacement	107 (65.2)
Patch repair	14 (8.5)
Two-layer suture repair	9 (5.5)
Single-layer pledgeted repair	34 (20.7)
Cross-clamp time, min	72.29 ± 16.68
Cardiopulmonary bypass time, min	145.27 ± 31.83
Concomitant valve surgery	55 (33.5)

PVL: Paravalvular leak.

Table 6. Cox proportional hazard analysis showing independent predictors of in-hospital mortality in patients undergoing PVL treatment.

Parameters	HR	95% CI	p
Being in surgery group	3.222	1.441-7.205	0.004
Previous endocarditis	3.038	1.421-6.496	0.004
Heart failure	2.446	1.156-5.161	0.019
Right venticle dysfunction	2.130	1.035-4.383	0.040
Chronic kidney disease	2.679	1.326-5.416	0.006

CI: Confidence Interval, HR: Hazard ratio, PVL: Paravalvular leak.

Table 5. Clinical outcomes per study group

Variables	Transcatheter closure (n=171)	Surgery (n=164)	p
Procedural success	126 (73.7)	107 (65.2)	0.093
Technical success	135 (78.9)	125 (76.2)	0.549
In-hospital outcomes, n (%)			
All cause death	8 (4.7)	26 (15.9)	0.001
Residual greater than mild PVL	29 (17)	12 (7.3)	0.007
Stroke	2 (1.2)	4 (2.4)	0.381
Vascular complications	10 (5.8)	4 (2.4)	0.119
Prolonged ventilation	3 (1.8)	6 (3.7)	0.281
AKI with a need for renal replacement therapy	6 (3.5)	24 (14.6)	<0.001
Pneumonia	3 (1.8)	16 (9.8)	0.002
Tamponade	2 (1.2)	9 (5.5)	0.027
Pleural effusion requiring drainage	1 (0.6)	15 (9.1)	<0.001
Hemothorax	1 (0.6)	1 (0.6)	0.976
Coronary obstruction	1 (0.6)	0 (0)	0.327
Cardiac rupture	1 (0.6)	2 (1.2)	0.538
Procedure-related PHV dysfunction	3 (1.8)	0 (0)	0.088
Major bleeding	5 (2.9)	9 (5.5)	0.241
Hospital length of stay, days	7.04 ± 5.24	13.12 ± 13.64	<0.001
Follow-up time, months	36.87 ± 20.1	49.30 ± 28.2	<0.001
Long-term outcomes, n (%)			0.020
All cause death	54 (31.6)	72 (43.9)	0.438
Stroke	3 (1.8)	5 (3)	0.229
Endocarditis	2 (1.2)	5 (3)	0.834
Rehospitalization	55 (32.2)	51 (31.1)	0.263
Reintervention	21 (12.3)	14 (8.5)	0.927
Transcatheter	9 (5.3)	9 (5.5)	0.098
Surgery	12 (7)	5 (3)	
Time to reintervention, months	2.04 ± 7.37	0.78 ± 3.40	0.107
Primary endpoint, n (%)	82 (48)	95 (57.9)	0.068

AKI: Acute kidney injury, PHV: Prosthetic heart valve, PVL: Paravalvular leak, TIA: Transient ischemic attack.

Table 7. Cox proportional hazard analysis showing independent predictors of long-term primary endpoint in patients undergoing PVL treatment

Parameters	HR	95% CI	p
Age	1.007	0.994-1.021	0.277
Estimated GFR ≤30	2.874	1.956-4.224	<0.001
Left ventricle EF ≤35%	1.476	0.934-2.331	0.095
Hemoglobin	0.965	0.878-1.061	0.461
Residual greater than mild PVL	2.690	1.826-3.962	<0.001

CI: Confidence Interval, EF: Ejection fraction, GFR: Glomerular filtration rate, HR: Hazard ratio, PVL: Paravalvular leak.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-069

The effect of sodium-glucose cotransporter-2 inhibitors on atrial electromechanic conduction time

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Background and Aim: The frequency of atrial fibrillation (AF) in patients with diabetes has been shown to be significantly higher than in the general population. Atrial electromechanical delay is known as an important determinant of atrial fibrillation. In this study, it is aimed to investigate the effects of sodium-glucose cotransporter-2 (SGLT-2) inhibitors, which are relatively new oral antidiabetic drugs, on atrial electromechanical delay (EMG) time in patients with type 2 diabetes mellitus (DM).

Methods: Thirty type 2 DM patients (53.3% female, mean age 60.07 ± 10.03 years) who were going to start SGLT-2 inhibitor were included in the study. The patients were evaluated with echocardiography at baseline and 6 months later. Basic echocardiographic findings and atrial electromechanical delay times were calculated with tissue Doppler.

Results: While no significant decrease was observed in intraatrial EMG times, a significant shortening was observed in interatrial EMG times. It was found that interatrial EMG times, which were 15.13 ± 5.87 ms, decreased to 13.20 ± 6.12 ms in the control (p=0.029). In lateral PA times (58.73 ± 6.41 ms to 54.37 ± 6.97 ms, p<0.001), septal PA times (50.90 ± 6.02 ms to 48.23 ± 5). Statistically significant shortenings were observed in the duration of the tricuspid PA (from 43.60 ± 6.28 ms to 41.30 ± 5.60 ms, p=0.003) to 88 ms, p<0.001). There was a significant decrease in e/e' values from 8.13 ± 4.0 to 6.50 ± 2.37 (p=0.003)

Conclusions: This study showed that; SGLT-2 inhibitors may have curative effects on electromechanical conduction of the atrium and thus protect from DM-related functional impairment and arrhythmia, particularly AF.

Table 1. Basic characteristics of patients

Basic characteristics (n=30)	
Age, mean+SD	60,07±10,03
Gender	
Male, number (%)	14 (46,7)
Female, number (%)	16 (53,3)
Hypertension, number (%)	20 (66,7)
Cerebrovascular accident, number (%)	2 (6,7)
Coronary artery disease, number (%)	10 (33,3)
Dapagliflozin users, number (%)	25 (83,3)
Empagliflozin users, number (%)	5 (16,6)

SD = standard deviation

Table 2. Laboratory findings

Biochemical Parameters	N	Pre-treatment	Post-treatment	p
eGFR (mL/min/1.73m2)	30	87,4±15,6	86,8±17,1	0,757
Creatinine (mg/dL)	30	0,83±0,15	0,83±0,20	0,821
BUN (mg/dL)	29	13,57±2,59	14,06±3,69	0,296
HbA1c (%)	27	9,33±1,41	8,14±1,39	<0.001
Fasting plasma glucose (mg/dL)	30	207,8±77,7	180,0±62,1	0,035
Total cholesterol (mg/dL)	21	175.0±60,1	158,1±40,2	0,159
LDL (mg/dL)	28	109,8±44,6	95,0±29,4	0,062
HDL (mg/dL)	23	41.0±8,1	42,2±9,3	0,445
Sodium (mmol/L)	30	138,5±3,1	138,8±3,3	0,556
Potassium (mmol/L)	30	4,60±0,34	4,57±0,36	0,423
LDH (mg/dL)	27	206,8±32,3	206,0±26,7	0,887
AST (U/L)	30	20,1±7,9	18,8±5,6	0,206
ALT (U/L)	30	24,1±14,9	20,4±10,3	0,027
GGT (U/L)	26	34,8±24,8	34,3±25,8	0,672

ALT; alanine aminotransferase, AST; aspartate aminotransferase, BUN; blood urea nitrogen, eGFR; estimated glomerular filtration rate, GGT; gamma glutamyl transferase, HBA1c; Glycosylated hemoglobin, HDL; High-density lipoprotein, LDH; Lactate dehydrogenase, LDL; Low-density lipoprotein, Data presented as mean±standard deviation.

Table 3. Echocardiographic parameters

	Pre-treatment	Post-treatment	p
Aortic root (mm)	21,30±2,38	21,37±2,41	0,677
LVDD (mm)	46,03±5,54	45,80±5,70	0,379
IVS (mm)	11,23±1,61	11,07±1,38	0,096
PW (mm)	10,07±1,23	9,73±1,08	0,023
E (m/s)	0,62±0,13	0,64±0,14	0,238
A (m/s)	0,87±0,17	0,83±0,18	0,065
E/A	0,72±0,14	0,80±0,23	0,021
E /e'	8,13±4,0	6,50±2,37	0,003
TAPSE (mm)	26,53±3,04	26,70±3,03	0,556
Ejection fraction (%)	59,00±5,68	59,10±5,16	0,756
LA area in A4C (cm2)	15,00±4,04	14,72±3,68	0,230
Heart rate (beats/min)	83,93±13,25	76,50±11,44	<0.001
PAP (mmHg)	6,50±9,66	6,33±10,08	0,899

A; late transmitral flow velocity, A4C; apical 4 spaces, E; early transmitral flow velocity, e': early diastolic mitral annular velocity, IVS; interventricular septum, LVDD; left ventricular end-diastolic diameter, LVSD; left ventricular end-systolic diameter, PAP; pulmonary artery pressure, PW; posterior wall, TAPSE; tricuspid annular plane systolic excursion. Data are presented as mean±standard deviation.

Table 4. Atrial electromechanical conduction times

	Pre-treatment	Post-treatment	p
Lateral PA (ms)	58,73±6,40	54,37±6,97	<0,001
Septal PA (ms)	50,90±6,02	48,23±5,87	<0,001
Triküspit PA (ms)	43,60±6,28	41,30±5,59	0,003
İntra-atriyal EMG (ms)	7,13±5,48	6,67±5,27	0,527
İnteratriyal EMG (ms)	15,13±5,87	13,20±6,12	0,029

EMG = Electromechanical delay, PA = atrial electromechanical conduction time, SGLT-2 = Sodium-glucose cotransporter-2. Data are presented as mean±standard deviation.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-070

Evaluation of risk factors for long-term atrial fibrillation development in patients undergoing typical atrial flutter ablation: A multi-center pilot study

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Background and Aim: Atrial flutter (AFL) and atrial fibrillation (AF) are the most commonly detected supraventricular arrhythmia and share similar pathophysiological mechanisms. After the successful ablation of AFL, AF frequently occurs in such patients in the long-term follow up. As emphasized in some studies, some mechanisms seem to predispose to the development of AF in AFL patients and approximately 20% of such kind of patients have accompanying AFL. We aim to analyze independent risk factors that predict the development of AF in patients who underwent typical AFL ablation.

Methods: Our study was designed as multi-center, cross-sectional and retrospective. A total of 442 patients who underwent typical AFL ablation at 3 different centers between January 1, 2018 and January 1, 2022 were included retrospectively. The patients after the ablation procedure were divided into those who developed AF and those who did not. The patients were followed up for an average of 12 (4-20) months. In the post-procedure period, atrial arrhythmias were investigated with 24-hour Holter and ECG at 1st month, 6th month, 12th month and at 6-month intervals thereafter.

Results: AF developed in 206 (46.6%) patients in the long-term follow-up. Age, hypertension (HT), obstructive sleep apnea syndrome (OSAS), previous cerebrovascular accident (CVA), left atrium antero-posterior diameter, severe mitral regurgitation, hemoglobin, blood glucose and HbA1c values were found to be significant in univariable analysis. According to multivariable analysis, HT [p=0.014, HR: 1.483 (1.084-2.030)], OSAS [p=0.008, HR: 1.520 (1.117-2.068)] and previous CVA [p=0.038, HR: 1.749 (1.031-2.968)] were independently associated with the development of AF in AFL patients who underwent ablation procedure.

Conclusions: In the present study, we found that HT, OSAS and previous CVA were independently correlated with the development of AF in the long-term follow-up of patients who underwent typical AFL ablation. We consider that AFL patients with such risk factors should be followed-up closely following cavo-tricuspid isthmus ablation for the development of AF.

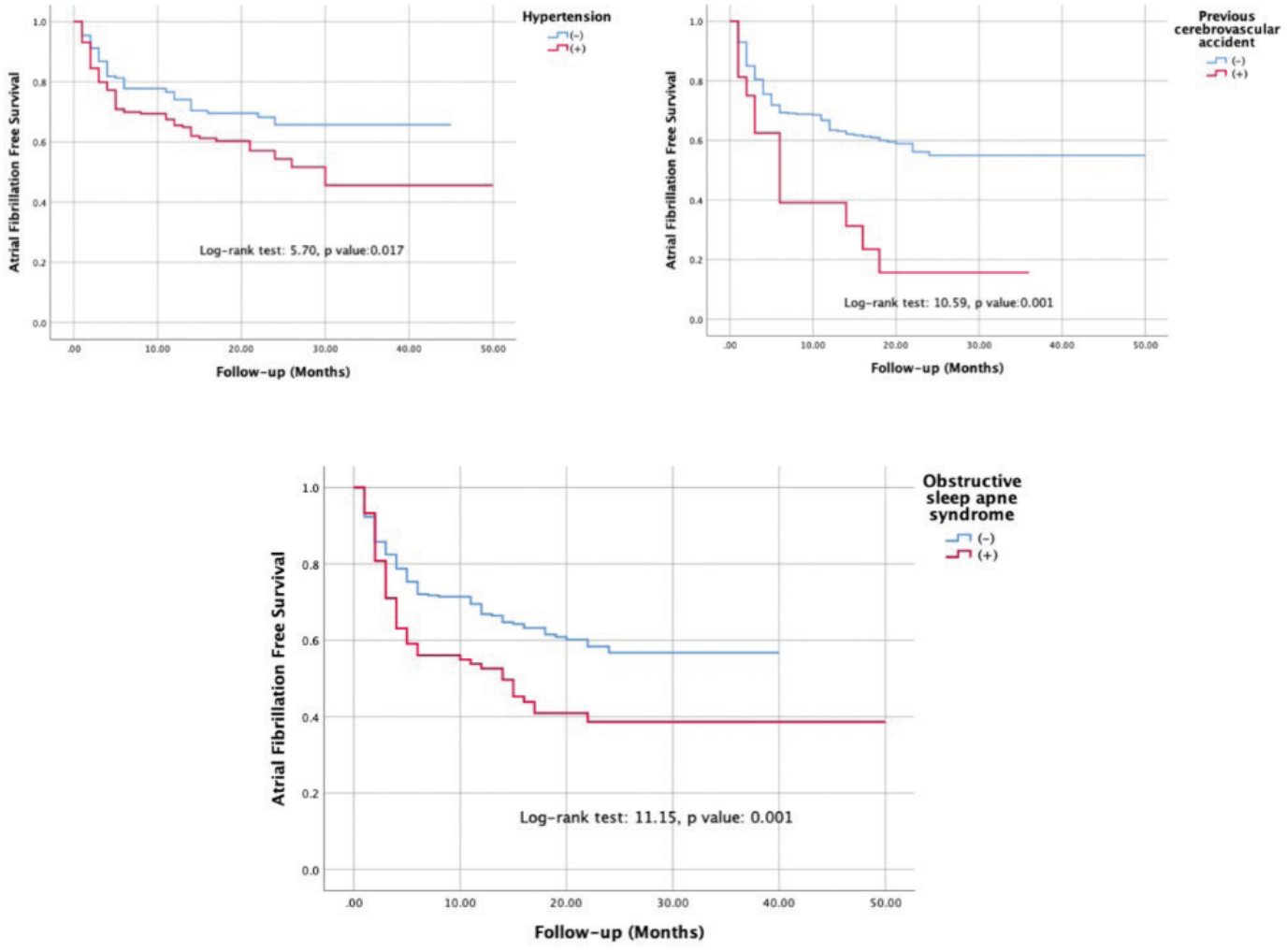

Figure 1. Kaplan - Meier Curves

Table 1. Comparison of demographic and clinical characteristics and echocardiographic parameters of patients according to incidence of atrial fibrillation in long-term follow-up

	Atrial Fibrillation (-) in long-term follow-up n=236	Atrial Fibrillation (+) in long-term follow-up n=206	P value
Age (years)	58 (50 – 68)	65 (58 – 70)	<0.001
Male gender, n (%)	162 (68.6)	147 (71.4)	0.535
Body mass index, kg/m^2	27.4 (24.6 – 29.7)	27.0 (24.4 – 30.1)	0.939
Hypertension, n (%)	110 (46.6)	137 (66.5)	<0.001
Diabetes Mellitus, n (%)	75 (31.8)	75 (36.4)	0.305
Chronic renal failure, n (%)	31 (13.1)	31 (15.0)	0.564
Chronic obstructive pulmonary disease, n (%)	21 (8.9)	13 (6.3)	0.306
Obstructive sleep apne syndrome, n (%)	41 (17.4)	63 (30.6)	0.001
Coronary artery disease, n (%)	66 (28.0)	61 (29.6)	0.703
Previous cerebrovascular accident, n (%)	4 (1.7)	15 (7.3)	0.004
Congestive heart failure, n (%)	54 (22.9)	43 (20.9)	0.611
Echocardiography variables at admission			
Left ventricle ejection fraction	60 (50 – 68)	60 (50 – 60)	0.005
LVEDD, mm	47 (45 – 51)	48 (45 – 52)	0.140
LVESD, mm	31 (28 – 37)	31 (28 – 37)	0.977
LA anterior-posterior diameter, mm	39 (36 – 44)	42 (37 – 46)	<0.001
MR ≥+3	32 (13.6)	48 (23.3)	0.008
TR ≥+3	53 (22.5)	33 (16.0)	0.088
LAA peak emptying velocity, cm/s	48 (35 – 60)	40 (33 – 50)	0.030

Table 2. Comparison of laboratory parameters of patients according to incidence of atrial fibrillation in long-term follow-up

	Atrial Fibrillation (-) in long-term follow-up n=236	Atrial Fibrillation (+) in long-term follow-up n=206	P value
Laboratory variables at admission			
Hb (g/dl)	13.8 (12.5 – 15.0)	13.2 (12.0 – 14.5)	0.005
RDW	14.3 (13.3 – 17.0)	14.4 (13.3 – 16.6)	0.588
MCV	88.3 (85.3 – 90.4)	88.2 (84.9 – 91.8)	0.914
WBC (cells/μL)	7.7 (6.3 – 8.9)	7.3 (6.2 – 8.8)	0.175
Neutrophils	4.7 (3.9 – 5.7)	4.5 (3.4 – 5.6)	0.201
Platelet count (/mm^3)	233 (185 – 277)	228 (182 – 268)	0.489
MPV	9.6 (8.6 – 10.8)	9.9 (9.1 – 10.7)	0.126
Lymphocytes	14.7 (12.5 – 14.8)	14.7 (12.5 – 14.8)	0.579
Creatinine (mg/dL)	0.89 (0.78 – 1.04)	0.90 (0.77 – 1.10)	0.622
BUN (mg/dL)	32.1 (28.0 – 39.0)	35.0 (26.0 – 44.0)	0.236
TSH	2.10 (1.47 – 2.90)	1.60 (1.00 – 2.63)	0.008
AST	21.0 (17.0 – 28.0)	21.0 (15.0– 27.0)	0.438
ALT	20.0 (14.0 – 29.0)	19.0 (13.0 – 28.0)	0.265
Glucose (mg/dl)	100.0 (89.0 – 121.0)	110.0 (93.0 – 139.0)	0.002
HbA1c	6.2 (5.7 – 6.7)	6.5 (6.0 – 7.1)	0.001
Triglyceride	104 (77 – 154)	115 (80 – 179)	0.047
HDL	45 (38 – 52)	44 (37 – 54)	0.652
LDL	107 (81 – 141)	113 (90 – 137)	0.271
Albumin	44 (40 – 46)	41 (38 – 44)	0.156
Medical treatment			
Class I Anti-arrhythmic drug, n (%)	52 (22.0)	89 (43.2)	<0.001
Sotalol, n (%)	3 (1.3)	7 (3.4)	0.200
Amiodarone, n (%)	35 (14.8)	44 (21.4)	0.074
Acetylsalicyclic acid, n (%)	52 (22.0)	45 (21.8)	0.962
Beta-blockers, n (%)	136 (57.6)	141 (68.4)	0.019
Ca-channel blockers, n (%)	50 (21.2)	73 (35.4)	0.001
Statin, n (%)	55 (23.3)	50 (24.3)	0.812
Ace inhibitors/ ARBs, n (%)	90 (38.1)	111 (53.9)	0.001
Spironolactone, n (%)	25 (10.6)	28 (13.6)	0.333
Furosemide, n (%)	50 (21.2)	40 (19.4)	0.645
Warfarin, n (%)	33 (14.0)	33 (16.0)	0.549
New oral anticoagulant, n (%)	102 (43.2)	159 (77.2)	<0.001

Table 3. Univariable analysis and multivariable model for long-term incidence of atrial fibrillation

	Univariable analysis		Multivariable analysis	
	P value	HR (95% CI)	P value	HR (95% CI)
Age	0.002	1.017 (1.006 – 1.029)	0.146	1.009 (0.997 – 1.022)
Hypertension	<0.001	1.819 (1.360 – 2.434)	*0.014*	1.483 (1.084 – 2.030)
Obstructive sleep apne syndrome	0.012	1.468 (1.087 – 1.984)	*0.008*	1.520 (1.117 – 2.068)
Previous cerebrovascular accident	0.011	1.981 (1.169 – 3.357)	*0.038*	1.749 (1.031 – 2.968)
LA anterior-posterior diameter	0.003	1.035 (1.011 – 1.059)	0.268	1.014 (0.989 – 1.039)
MR ≥+3	0.011	1.519 (1.099 – 2.100)	0.196	1.251 (0.891 – 1.755)
Hb	0.004	0.898 (0.834 – 0.967)	0.174	0.947 (0.875 – 1.024)
Glucose	0.010	1.003 (1.001 – 1.005)	0.349	1.001 (0.998 – 1.005)
HbA1c	0.005	1.177 (1.050 – 1.318)	0.539	1.051 (0.897 – 1.232)

Abbreviations: HR, Hazard ratio; CI, confidence interval

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

OP-071

Assessment of NOAC dosing patterns in Turkish patients with non-valvular atrial fibrillation: A multicenter, cross-sectional study with insights from the ASPECT-NOAC study

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Background and Aim: We aimed to assess the real-world label adherence of non-vitamin K antagonist oral anticoagulant (NOAC) dosing patterns, including apixaban, edoxaban, and rivaroxaban, as well as their clinical outcomes in Turkish patients with atrial fibrillation (AF).

Methods: This was an observational, prospective, multicenter study. Patients with AF who were prescribed NOACs within the last 4 months were recruited from 34 cardiology clinics in Turkey. Baseline data were initially collected, and patient awareness was evaluated at 3-4 weeks. The final study visit was conducted at 12 months.

Results: A total of 903 patients were enrolled in the study. The mean age was 72.84 ± 10.17 years, and 475 patients (52.6%) were female. We found that 140 (15.5%), 721 (79.8%), and 42 patients (4.7%) were prescribed off-label low, on-label, and off-label high dosing, respectively. The age of the patients in the on-label group was significantly lower than that of those in the off-label low and off-label high groups (both p<0.001). Additionally, female patients were more frequently observed in the off-label high group (p=0.019), and patients in the off-label low and on-label groups were significantly more obese than those in the low-label group (p<0.001) (Table 1 and 2). The perception of income levels also revealed significant differences between the groups (p=0.010) (Table 3). Furthermore, the HAS-BLED scores were significantly lower in the on-label group than in the other groups (both p<0.001). Similarly, the CHA2DS2-VASc scores were significantly lower in the on-label group than in the off-label group (p<0.001).

Conclusions: Off-label low-dose NOAC prescriptions were relatively common in Turkish patients; however, this was much lower than in previously reported results. Increasing clinician awareness of appropriate NOAC dosing may help identify at-risk patients.

Table 1. Demographic and clinical characteristics of the study groups

		Groups			
		Off-label low (n=140)	On-label (n=721)	Off-label high (n=42)	p
Age (year)		74.2 ± 9.6	68.1± 9.8	73.4 ± 12.1	<0.001
Sex	Female	63 (45.0)	383 (53.1)	29 (69.0)	0.019
	Male	77 (55.0)	338 (46.9)	13 (31.0)	
BMI (kg/m^2)		28.9 ± 5.0	29.7 ± 5.7	25.9 ± 6.1	<0.001
Educational status	Illiterate	38 (27.1)	167 (23.2)	14 (33.3)	0.358
	Primary school	77 (55.0)	387 (53.7)	24 (57.1)	
	Middle School	11 (7.9)	69 (9.6)	2 (4.8)	
	High school	6 (4.3)	69 (9.6)	1 (2.4)	
	University	8 (5.7)	28 (3.9)	1 (2.4)	
	Post-University (Master's-Doctorate)	0 (0.0)	1 (0.1)	0 (0.0)	
Perception of income level	On average, my income covers my expenses.	94 (67.1)	496 (68.8)	26 (61.9)	0.010
	Below average, I am having trouble meeting my essential needs	34 (24.3)	190 (26.4)	15 (35.7)	
	Above average, I can save beyond meeting my needs.	11 (7.9)	16 (2.2)	1 (2.4)	
	Poor, I cannot meet my needs at all.	1 (0.7)	19 (2.6)	0 (0.0)	
Drug discontinuation	Those who continued	106 (93.0)	576 (91.1)	30 (90.9)	0.807
	Those who discontinued	8 (7.0)	56 (8.9)	3 (9.1)	
HAS-BLED Score		1.94 ± 1.1	1.5 ± 1.08	2. ± 1.1	<0.001
CHA_2DS_2-VASc Score		3.471 ± 1.42	2.9 ± 1.5	3.3 ± 1.2	<0.001

BMI: Body Mass Index; HAS-BLED: Hypertension, Abnormal liver/renal function, Stroke history, Bleeding history or predisposition, Labile INR, Elderly, Drug/alcohol usage; CHA2DS2-VASc congestive heart failure, hypertension, age ≥75 (doubled), diabetes, stroke (doubled), vascular disease, age 65 to 74 and sex category (female)

Table 2. Dosing patterns of NOACs in the study groups

	Prescription Patterns n (%)			
NOAC	Off-label Low	On-Label	Off-label High	Total
Apixaban	68 (17.5%)	309 (79.6%)	11 (2.8%)	388 (100%)
Edoxaban	15 (9%)	138 (82.6%)	14 (8.4%)	167 (100%)
Rivaroxaban	57 (16.4%)	274 (78.7%)	17 (4.9%)	348 (100%)
Total	140 (15.5%)	721 (79.8%)	42 (4.7%)	903 (100%)

1. Low dose: apixaban less than 10mg daily; rivaroxaban less than 20mg daily; edoxaban less than 60 mg daily.
2. Standard dose: apixaban 10mg daily; rivaroxaban 20mg daily; edoxaban 60 mg daily.
3. High dose: apixaban more than 10mg daily; rivaroxaban more than 20mg daily; edoxaban more than 60 mg daily.

Table 3. Association between the knowledge levels of NVAF and NOACs and dosing pattern

	Off-label low (n=132)	On-label (n=692)	Off-label high (n=39)	p
AF Knowledge	48.8 ± 23.0	49.3 ± 23.3	45.1 ± 24.6	0.506
NOAC Knowledge	74.7 ± 20.3	73 ± 19.4	72.2 ± 20.5	0.520

AF: Atrial Fibrillation; NOAC: Non-vitamin K Antagonist Oral Anticoagulant

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-072

Predictors of late term sustained ventricular arrhythmias in patients with LVAD

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Background and Aim: Left ventricular assist devices (LVADs) implantation is increasingly used in patients with end-stage heart failure (HF). Although these devices effectively improve survival, atrial and ventricular arrhythmias are common, they may predispose these patients to additional risk, and complicate patient management. Identifying determinants of late term sustained ventricular arrhythmias (sVA) in patient with LVAD.

Methods: The data of patients who had an LVAD implantation in a single tertiary center between 2012-2022 were reviewed retrospectively. 495 consecutive patients with docemented rhythm information whose arrhythmia records could be accessed were included in the study. After the first-year follow-up, the patients were divided into two groups as those with and without late sVA. Factors that may affect the occurrence of late sVA were compared between the groups.

Results: 495 patients (mean age was 54 ± SD and 87.7% male) included in the study. 34 (6.9%) of them had late sVA. The 49.1% had ischemic etiology and 72.1% had heart ware. Mean follow up was 35 ± 34 months, late sVA group had longer follow up (p=0.01) (Table 1). Patients with late sVA were older, intracardiac defibrillator, heart ware use and pump thrombosis were more common. Late sVA occurred in 34 (56.7%) of 60 post-LVAD patients (p=0.0001). History of sVA in the first month after LVAD was not associated with late sVA. Kaplan Meier survival analysis revealed patients with late sVA had better survival.

Conclusions: Late sVA is a more common late term complication in HF patients with LVAD. Since patients with early complications die earlier, those surviving had long lived LVAD could have more late sVA. This could be an explanation of the better survival of the late sVA group (immortal time bias). Also late sVA patients had more pump thrombosis which might not be associated with a cause-effect relationship.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-073

CHA2DS2-VASc and CHADS2 scores, indicators of clinical outcomes in patients with atrial high rate episodes

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Background and Aim: Atrial high rate episode is a type of arrhythmia that is associated with cerebrovascular events and is detected in patients with an intracardiac device with an atrial lead. There is no indicator that can show the short-term clinical outcomes in patients with AHRE, and there is not enough data to reveal the relationship between CHA_2DS_2-VASc and $CHADS_2$. In this study, we examined the effect of CHA_2DS_2-VASc and $CHADS_2$ score on all-cause death, atrial fibrillation development and cerebrovascular event development at 18 months in patients who applied to the cardiology outpatient clinic and had AHRE detected in routine pacing control.

Table 1. Baseline demographics of all groups

	All patients (n=495)	Late VA post-LVAD (n=34)	No-late VA post-LVAD (n=461)	p
Age, years, mean ± SD	54.48 ± 12.3	56.44 ± 10.93	54.34 ± 12.47	0.422
Male, n (%)	434 (87.7)	30 (88.2)	404 (87.6)	0.918
Heart failure etiology, n (%) - Ischemic CMP - Dilated CMP	 243 (49.1) 251 (50.7)	 17 (50) 17 (50)	 235 (51) 227 (49.2)	0.913 0.932
LVAD Heart mate II Heart mate III Heart ware	 57 (11.5) 80 (16.2) 357 (72.1)	 9 (10.4) 7 (20.6) 18 (52.9)	 48 (26.5) 74 (16.4) 339 (73.5)	0.009
LVAD with intracardiac defibrillator, n (%)	280 (56.5)	27 (79.4)	253 (54.9)	0.005
LVAD with CRT, n (%)	51 (10.3)	4 (11.8)	47 (10.2)	0.77
Follow-up, months, mean ± SD	35 ± 34	61.35 ± 38.14	33.53 ± 33.7	0.01

CMP: Cardiomyopathy, CRT: Cardiac resynchronization therapy, LVAD: Left ventricular assist device, VA: Ventricular arrhythmia.

Methods: 61 patients with atrial leads (CRT-P/D, DDR-KPM and DDR-ICD), older than 18 years of age, and AHRE detected in the event records, who applied to the cardiology outpatient clinic between June 2020 and January 2022, were included. We performed a retrospective observational study enrolling consecutive patients with AHRE detected by an intracardiac electronic device. AHRE traces recorded by the CIED were visually inspected and adjudicated independently by two cardiologists to avoid false positives. The only exclusion criteria are clinical AF documented by surface ECG (12-lead ECG) or an ECG strip lasting ≥30 seconds. CHA_2DS_2-VASc and $CHADS_2$-score were calculated appropriately according to patients' congestive heart failure, hypertension, diabetes, stroke, vascular disease, gender and age. Patients were followed at 18 months for cerebrovascular event, conversion to permanent atrial fibrillation, and all-cause mortality. Groups with and without clinical outcomes were compared in terms of clinical, laboratory and CHA_2DS_2-VASc and $CHADS_2$ scores.

Results: 61 patients with AHRE detected by intracardiac device, with an average age of 71.4 ± 12.2 years, were included in the study. Mean left ventricular ejection fraction values were 51.9 ± 13.3. 70.5% of the patients were hypertension, 27.9% diabetes mellitus, 16.4% heart failure. During the 18th month follow-up, 5 of the patients (8.2%) were rehospitalized due to cerebrovascular event. Mortality was observed in 8 (13.1%) of the patients. Between the groups with and without clinical outcomes, CHA_2DS_2-VASc score (4.3 ± 1.8 vs. 2.9 ± 1.3, p=0.001) and $CHADS_2$ score (2.53 ± 1.3 vs. 1.4 ± 1.1, p=0.001) was significantly higher.

Conclusions: Studies have reported that as the CHA_2DS_2-VASc score increases, the incidence of AHRE increases and this relationship is stronger with longer AHREs. CHA_2DS_2-VASc score was found to be a predictor of atrial fibrillation development in AHRE patients. Difficult conditions such as death or cerebrovascular event were not included in clinical outcomes. In our study, however, cerebrovascular event and all-cause death were included in clinical outcomes, and the correlation of scores with clinical outcomes was demonstrated at the end of 18-month follow-up. In patients with AHRE, the calculated scores can be helpful in predicting the development of clinical outcomes, patient follow-up, and decision-making.

Table 1. Clinical and scoring characteristics of the study device

	Clinical outcome (-) (n=42)	Clinical outcome (+) (n=19)	p
Age, years	69.9 ± 12.6	74.8 ± 11.1	0.144
Diabetes, n (%)	9 (21)	8 (42)	0.174
Hypertension, n (%)	27 (64)	16 (84)	0.202
LVEF, %	53.8 ± 12.2	47.8 ± 15.2	0.132
Creatinine, mg/dL	0.9 ± 0.6	1.1 ± 0.3	0.425
CHA_2DS_2-VASc	4.3 ± 1.8	2.9 ± 1.3	0.001
$CHADS_2$	2.5 ± 1.3	1.4 ± 1.1	0.001

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-074

Effects of premature ventricular complex burden on left ventricular global longitudinal strain in patients without structural heart disease

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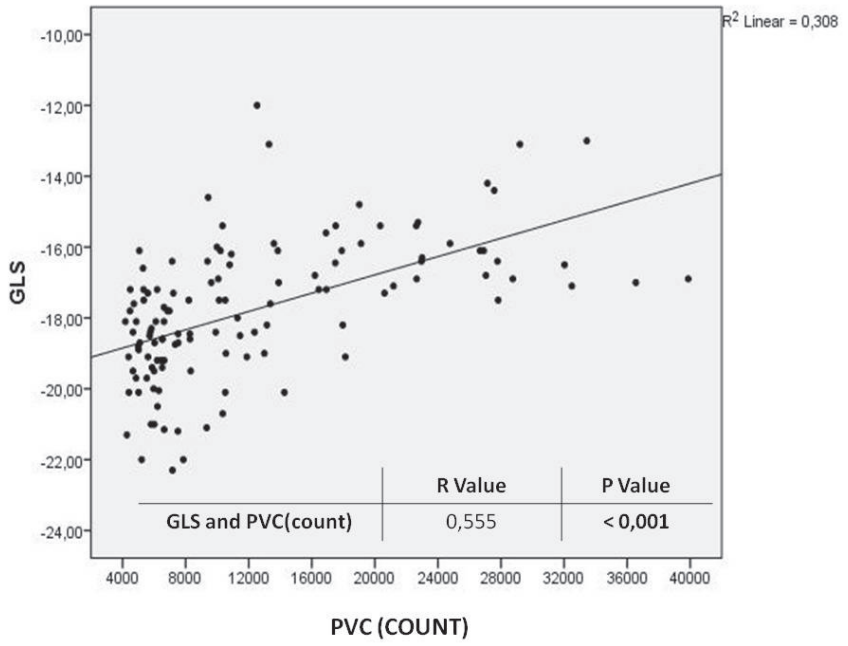
Background and Aim: Idiopathic PVCs, frequently encountered rhythm disorders, can have significant clinical implications. Although LVEF is commonly preserved in patients without structural heart disease, assessing detailed LV function remains crucial. Speckle-tracking echocardiography can provide insight into subclinical LV disturbances, enabling the evaluation of LVGLS. This study aimed to examine the impact of PVC burden on LV functions (GLS and MPI) in patients with preserved LVEF.

Methods: A total of 122 patients with idiopathic frequent PVCs were included and divided into three groups according to PVC burden: group 1 5%<PVC<10%, group 2 10%<PVC<15%, and group 3 15%<PVC. Electrocardiography and holter recordings were analyzed to assess PVC characteristics. Transthoracic echocardiography was performed to evaluate LV parameters, including LV global longitudinal strain (LV-GLS) and myocardial performance index (MPI).

Results: In our study, no significant difference was found between the groups determined according to PVC burden in terms of baseline demographic and laboratory parameters. More than 70% of our patients had cardiac MRI evaluations. LVGLS value was found to be significantly higher in groups 2 and 3 with high PVC burden (Table 1) (-18.89 ± 1.4, -17.55 ± 2.1, -16.26 ± 1.3; p<0.001, respectively). Correlation analysis (Figure 1) revealed a positive and significant correlation between PVC count and PVC burden with deterioration in left ventricular global longitudinal strain (LV-GLS) values (r=0.555; p<0.001, r=0.536; p<0.001, respectively). The ROC curve analysis demonstrated that (Figure 2) different PVC burden cut-off values were associated with GLS deterioration greater than certain thresholds with varying levels of specificity and sensitivity.

Conclusions: In our study, it was observed that there was a significant deterioration in LV-GLS values in groups with high PVC burden. Even lower levels of PVC burden were associated with left ventricular function impairments in patients with preserved LVEF and normal hearts. Our study suggests that permanent treatments such as ablation may come to the fore at an early stage, especially in this group of patients, even if the LVEF value is normal, and it may be appropriate to follow up these patients not only with standard ECHO but also with strain echo. The effects of these data on clinical outcomes are important, therefore more comprehensive follow-up studies are needed.



Roc Curve Analysis

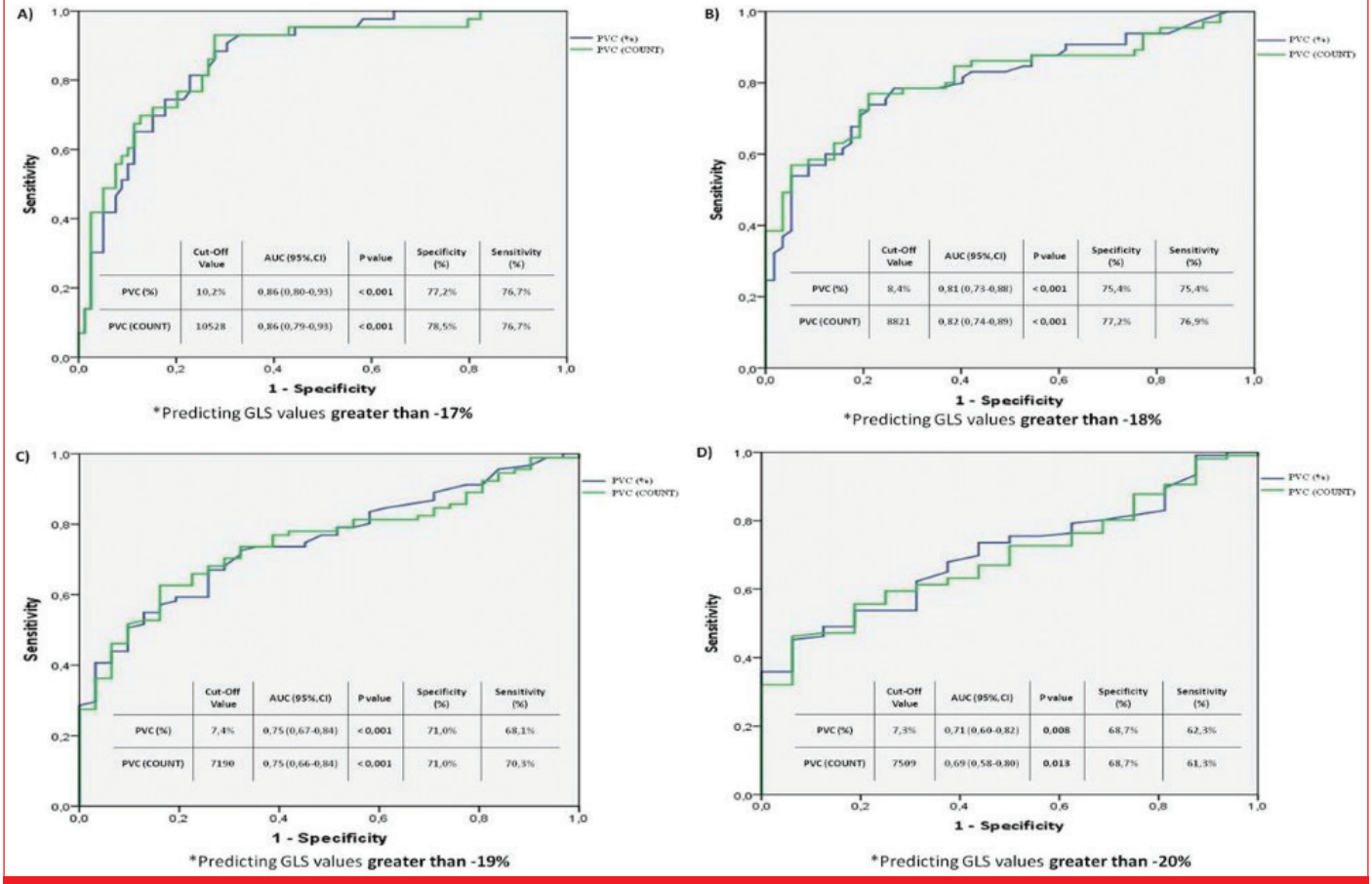


Figure 1. Correlation analysis.

Table 1. Baseline clinical characteristics of patients according to PVC burden

	Group 1 (n=54)	Group 2 (n=33)	Group 3 (n=35)	p
Age, years	46.83 ± 12.6	46.94 ± 8.8	42.83 ± 14.8	0.280
Male, n (%)	20 (37.0%)	16 (48.5%)	17 (48.6%)	0.445
Hypertension, n (%)	23 (42.6%)	10 (30.3%)	11 (31.4%)	0.407
Diabetes mellitus, n (%)	12 (22.2%)	5 (15.2%)	4 (11.4%)	0.392
Sudden death in family, n (%)	10 (18.5%)	5 (15.2%)	12 (34.3%)	0.114
Drug usage, n (%)				
Beta-blockers	35 (64.8%)	23 (69.7%)	30 (85.7%)	0.093
CCB	9 (16.7%)	5 (15.2%)	11 (31.4%)	0.163
Class Ic AAD	9 (16.7%)	5 (15.2%)	12 (34.3%)	0.084
Class III AAD	4 (7.4%)	2 (6.1%)	4 (11.4%)	0.694
BMI	25.4 ± 2.7	26.6 ± 3.5	24.7 ± 2.7	0.029
Cardiac MRI, n (%)	6 (11.1%)	4 (12.1%)	16 (45.7%)	<0.001
Haemoglobin, mg/dL	13.84 ± 1.4	14.18 ± 1.5	14.23 ± 1.6	0.400
LDL cholesterol, mg/dL*	124.37 ± 30.4	127.30 ± 27.4	130.94 ± 38.3	0.641
HbA1c, %	5.8 ± 0.9	5.8 ± 0.8	5.9 ± 1.1	0.678
TSH	2.39 ± 1.3	2.59 ± 1.9	2.53 ± 1.8	0.924
fT4	1.16 ± 0.2	1.28 ± 0.6	1.17 ± 0.2	0.742
Creatinine, mg/dL	0.78 ± 0.16	0.77 ± 0.1	0.81 ± 0.2	0.491
Potasium, mmol/L	4.48 ± 0.4	4.61 ± 0.4	4.67 ± 0.4	0.021
PVC count/day	5890 ± 1064	10778 ± 2069	23689 ± 6452	<0.001
QTc (msc)	419.74 ± 40.1	425.12 ± 32.8	422.40 ± 30.3	0.789
BBB, n (%)	10 (18.5%)	10 (30.3%)	10 (28.6%)	0.377
Average HR/min	74.59 ± 8.2	75.79 ± 9.5	76.34 ± 7.2	0.598
NSVT, n (%)	8 (14.8%)	13 (39.4%)	15 (42.9%)	0.006
LVd, mm	48.02 ± 3.3	50.45 ± 4.9	49.94 ± 7.7	0.066
MPI index	0.50 ± 0.06	0.48 ± 0.08	0.47 ± 0.06	0.143
GLS %	-18.89 ± 1.4	-17.55 ± 2.1	-16.26 ± 1.3	<0.001

* median (min-max) AAD: Antiarrhythmic drug, BBB: Bundle branch block, BMI: Body mass index, CA: Coronary angiography, CCB: Calsium canal blockers, GLS: Global longitudinal strain, fT4: Free thyroxine four, LVd: Left ventricular end-diastolic diameter, MPI: Myocardial performance index, NSVT: Non-sustained ventricular tachycardia, PVC: Premature ventricular complex, QTc: Corrected QT intervale, TSH: Thyroid stimulating hormone.

Heart Failure

OP-075

Determination of left ventricular recovery rate and predictors in heart failure patients with low ejection fraction

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Background and Aim: Determination of myocardial recovery rates and predictors in the patient group with low ejection fraction.

Methods: A total of 1436 patients who met the inclusion criteria were included in the study. The study is single-center, observational, retrospective, study. Distribution of all patients included in our study in terms of hypertension, diabetes mellitus, hyperlipidemia, cerebrovascular accident, lung disease, liver disease, renal disease, smoking, electrocardiographic findings (classified as sinus rhythm, atrial fibrillation, pace rhythm), echocardiographic findings (left and right ventricular ejection fraction values, left ventricular systolic and end-diastolic diameters, left atrium diameters, degrees of valve regurgitation, TAPSE, RSVM, SPAP values related to right ventricular functions), blood pressure values at presentation to the clinic, baseline, NYHA functional class values, NT-pro-BNP follow-up values and CİED's data were evaluated. The data obtained were recorded in the case report form. Binary logistic

regression analyzes were applied to determine the predictors in the study.

Results: The mean age (± SD) of the patients in the study was 46.89 (12.28). 27.2% of the patients were women. When the patients were classified according to the type of cardiomyopathy, 57.7% (n=837) of the patients had ischemic and 42.3% (n=599) had non-ischemic etiology. Recovery was detected in 157 (10.9%) patients in total. This rate was 18.7% for the non-ischemic group and 5.3% for the ischemic group. Recovery rates were significantly higher in the group followed with non-ischemic etiology. Presence of atrial fibrillation (OR: 3.177, CI: 1.409-7.162, p=0.005), use of loop diuretics (OR: 2.673, CI: 1.690-7.688, p<0.0001), higher NT-pro-BNP values (OR: 1.000, CI: 1.000-1.000, p=0.006), higher NYHA classes (OR: 4.793, CI: 2.230-9.988, p<0.0001), increased LVEDD diameters (OR: 1.085, CI: 1.055-1.117, p<0.0001), the presence of severe tricuspid regurgitation (OR: 2.930, CI: 1.023-8.392, p<0.0001) were found to be negative predictors that reached statistical significance in multivariate analysis.

Conclusions: In conclusion, in this study, which aimed to determine the recovery rates and predictors, the total recovery rate was found to be 10.9%. The factors that may be related are; non-ischemic etiology, lower BNP values, lower NYHA classes, no presence of atrial fibrillation, not using loop diuretics, smaller LVEDD diameters, and no severe tricuspid regurgitation.

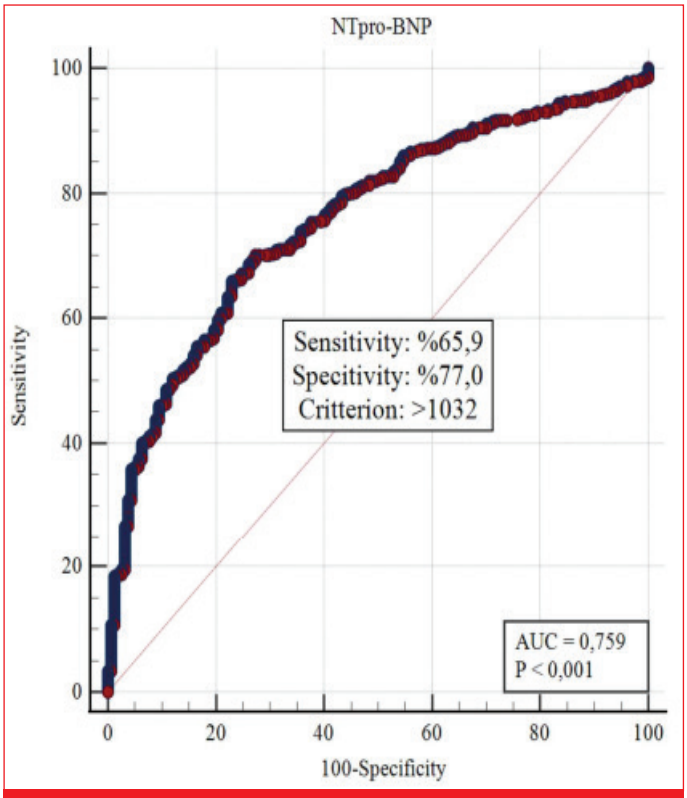

Figure 1. BNP values ROC analysis.
We found with %65,9 sensitivity and with %77 specitivity that those patients who have BNP values higher than 1032, we do not expect recovery for patients above this value.

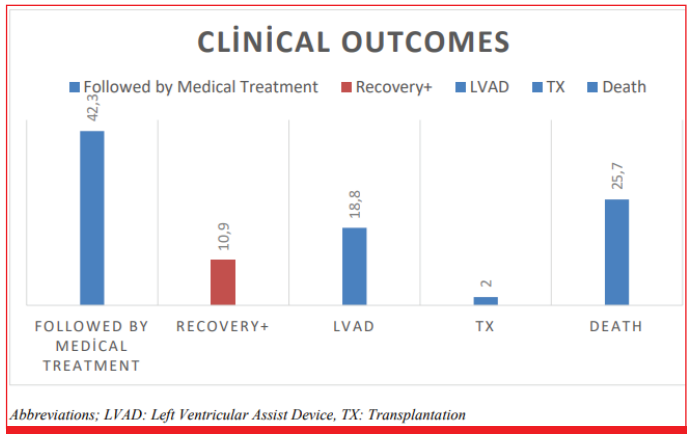

Figure 2. Clinical outcomes.

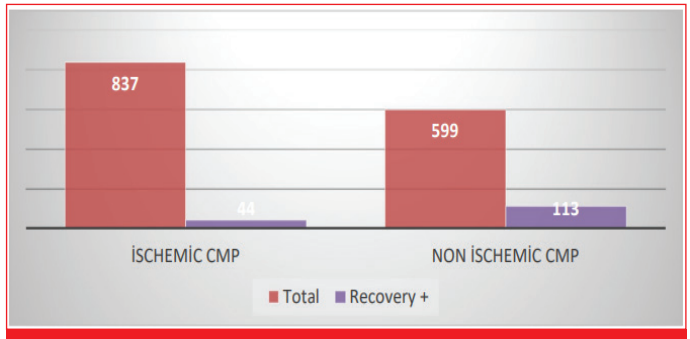

Figure 3. Clinical outcomes accoring to etiology.

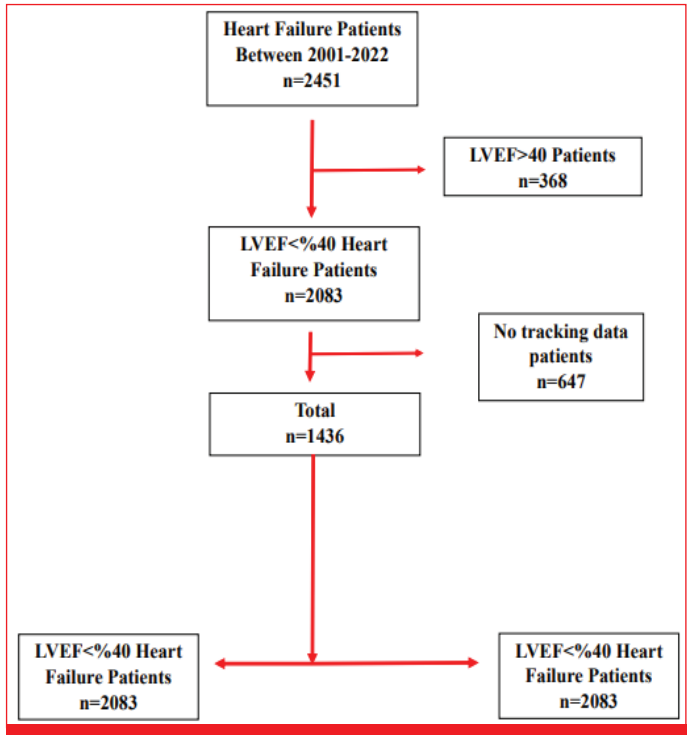

Figure 4. Flow chart of study patients.

Table 1. Basal characteristical analysis

	Univariate analysis		Multivariate analysis	
	OR (95% CI)	P value	OR (95% CI)	P value
Age	1.013(1.000-1.027)	**0.04**	-	-
Woman %	0.938 (0.825-1.067)	0.33	-	-
BMI kg/m^2	0.999(0.955-1.046)	0.97	-	-
Smoker	0.751(0.527-1.068)	0.11	-	-
Hypertension	1.132 (0.792-1.619)	0.49	-	-
Diabetes mellitus	1.382 (0.934-2.044)	0.10	-	-
Hyperlipidemia	1.065 (0.682-1.661)	0.78	-	-
CVA	1.778 (0.706-4.474)	0.22	-	-
Lung Dis.	1.348 (0.610-2.981)	0.46	-	-
Renal Dis.	1.343(0.475-3.792)	0.57	-	-
SBP	0.977 (0.968-0.985)	**<0.0001**	-	-
DBP	0.979 (0.966-0.992)	**0.001**	-	-
BNP	1.001 (1.000-1.001)	**<0.0001**	1.000 (1.000-1.000)	**0.006**
NYHA				
I	-	-	-	-
II	1.649 (1.081-2.515)	**0.02**	-	-
III	12.097 (6.123-23.902)	**<0.0001**	4.793 (2.230-9.988)	**<0,0001**
IV	-	-	-	-
AF	3.946 (1.981-7.860)	**<0.0001**	3.177 (1.409-7.162)	**0.005**
Family History	0.772 (0.484-1.230)	0.27	-	-
ACEi or ARB	1.094 (0.734-1.632)	0.65	-	-
Full dose	0.412 (0.247-0.687)	**0.001**		
Half dose	1.197 (0.690-2.076)	0.52		
Quarter dose	2.861 (1.281-6.394)	**0.01**		
ARNİ	0.564 (0.345-0.923)	**0.02**	-	-
Full dose	0.381 (0.160-0.904)	**0.03**		
Half dose	1.584 (0.484-5.183)	0.44		
Quarter dose	0.406 (0.208-0.789)	**0.008**		
ACEi/ARB/ARNİ	0.656 (0.370-1.164)	0.14	-	-
Beta bloker	0.285 (0.038-2.106)	0.21	-	-
Full dose	0.169 (0.022-1.308)	0.08		
Half dose	0.233 (0.031-1.729)	0.15		
Quarter dose	0.424 (0.058-3.312)	0.42		
MRA	0.919 (0.494-1.711)	0.79	-	-
Full dose	0.717 (0.384-1.337)	0.29		
Half dose	0.999 (0.881-2.300)	0.81		
SGLT 2 inh	0.605 (0.405-0.905)	**0.01**	-	-
Loop diuretics	4.197 (2.938-5.997)	**<0.0001**	2.673 (1.690-7,688)	**<0.0001**
Acetyl salicylic acid	1.011 (0.727-1.412)	0.94	-	-
Antiplatelets		0.84	-	-
Anticoagulants	0.916 (0.641-1.309)	0.63	-	-
Ivabradin	0.677 (0.482-0.951)	**0.02**	-	-
Digoxin	3.213 (1.756-5.879)	**<0.0001**	-	-
Statin Therapy	0.733 (0.523-1.026)	0.07	-	-
CRT	1.757 (0.973-3.174)	0.06	-	-
Antiarithmye Therapy	3.014 (1.298-6.999)	**0.01**	-	-

Table 2. Echocardiography parameter analysis

	Univariate Analysis		Multivariate Analysis	
	Total			
	OR (95% CI)	P value	OR (95% CI)	P value
LVEF %	0.909(0.883-0.935)	<0.0001	-	-
LVEDD mm	1.070 (1.049-1.092)	<0.0001	1.085 (1.055-1.117)	<0.0001
LVESD mm	1.077 (1.057-1.096)	<0.0001	-	-
LA mm	1.098 (1.069-1.127)	<0.0001	-	-
MR				
Mild	0.922(0.525-1.621)	0.77	-	-
Moderate	1.539 (0.885-2.678)	0.12	-	-
Severe	4.356 (1.170-6.053)	<0.0001	-	-
AR				
Mild	0.604 (0.171-2.130)	0.43	-	-
Moderate	1.245 (0.587-2.640)	0.31	-	-
Severe	1.442 (0.879-2.365)	0.14	-	-
TR				
Mild	2.257 (1.473-3.460)	<0.0001	-	-
Moderate	3.125 (1.984-4.924)	<0.0001	-	-
Severe	4.678 (1.292-26.932)	<0.0001	2.930 (1.023-8.392)	<0.0001
PR				
Mild	2.862 (0.898-4.324)	0.91		
Moderate	6.893 (1.615-27.147)	0.09		
Severe	8.380 (2.684-31.567)	0.99		
TAPSE* mm	0.972 (0.960-0.984)	<0.0001		
RVsm m/sn	0.898(0.865-0.932)	<0.0001	-	-
SPAP mmHg	1.040 (1.026-1.054)	<0.0001	-	-

Heart Failure

OP-076

Abdominal perfusion pressure is an indicator of short-term re-admission in patients hospitalized with acute decompensated heart failure

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Background and Aim: There is growing interest in the measurement of intra-abdominal pressure (IAP) in critical patients, which is associated with intra-abdominal organ dysfunction. Elevated IAP in patients with decompensated heart failure has been associated with a worsening of renal function. Also, there is no data regarding clinical outcomes of the abdominal IAP and perfusion pressure (APP) in in patients hospitalized with acute decompensated heart failure (HF). In this study, we aimed to evaluate the effect of IAP and APP values measured in the early period on clinical outcomes in patients hospitalized with acute decompensated HF at the first month.

Methods: The study was conducted prospectively in a single center. Between March 2022 and May 2023, 45 patients who were hospitalized with acute decompensated HF. To obtain a precise IAP value, the pressure is measured with the transvesical method. Briefly, IAP is measured via a standard Foley catheter, which is connected with a pressure transducer placed in-line with the iliac crest at the midaxillary line. The Foley catheter is flushed with a maximal instillation volume of 25 mL sterile saline via the aspiration port of the Foley catheter with the drainage tube clamped to allow a fluid-filled column to develop up into the bladder. A pressure transducer is then inserted in the aspiration port, and the pressure is measured. The IAP is expressed in mmHg and is measured atend-expiration in the supine position, ensuring that abdominal muscle contractions are absent. In this study, the IAP is measured on admission (within the first 8 hours). We used a cut-off value of ≥8 for the elevated IAP. The patients were followed up in the first month in terms of re-admission, hospitalization, and mortality due to heart failure and all-cause mortality. Groups with and without clinical outcomes were compared IAP, APP, and MAP measurements.

Results: Forty-five patients with a mean age of 75 ± 8.7 years were included in the study. There were 25 (55.5%) patients with an IAP value >8 mmHg. Total length of stay is longer in patients with IAP value >8 mmHg (8.7 ± 4.5 vs. 5.4 ± 3.2 days, p=0.009). And, creatinine value was significantly higher in patients with higher IAP value (1.44 ± 0.57 vs. 1.02 ± 0.28 mg/dL). In the first month follow-up, 10 (22%) of the patients were re-hospitalized due to HF. HF related mortality was observed in 2 (4.4%) of the patients. In the group with clinical outcomes, APP (72.9 ± 6.2 vs. 83.2 ± 12.8, p=0.019), MAP (83.7 ± 8.9 vs. 93.2 ± 13.2, p=0.040), and DBP (65.7 ± 8.4 vs. 74.3 ± 12, p=0.041) was significantly lower than the group without clinical outcomes.

Conclusions: This study revealed that low APP values measured at admission in patients with acute decompensated HF are associated with short-term readmission, and high IAP values are associated with longer length of stay. Although the interim analysis results of our ongoing study are presented, studies with more patients are needed to evaluate the relationship between HF classifications, comorbidities, IAP and APP.

Table 1. Comparison of general demographic, echocardiographic and laboratory results of the patients

	Clinical outcome (-) (n=35)	Clinical outcome (+) (n=10)	p
Age, years	75.7 ± 8.4	72.7 ± 9.8	0.341
Gender, male, n (%)	16 (46)	3 (30)	0.481
Diabetes, n (%)	11 (31)	5 (50)	0.451
Hypertension, n (%)	23 (69)	7 (70)	0.981
Systolic blood pressure, mmHg	43.9 ± 18.2	41.9 ± 18.6	0.761
LAVI, mL/m^2	48.9 ± 19.2	43.7 ± 18.2	0.463
TAPSE, mm	18.0 ± 3.1	20.3 ± 5	0.100
LVEF, %	39 ± 14.7	38.3 ± 16.6	0.901
Creatinine, mg/dL	1.2 ± 0.5	1.4 ± 0.4	0.445

TAPSE: Tricuspid annular plane systolic excursion, LVEF: Left ventricle ejection fraction, LAVI: Left atrium volume index.

Table 2. Comparison of hemodynamic and IAP, APP measurements

	Clinical outcome (-) (n=35)	Clinical outcome (+) (n=10)	p
SBP, mmHg	131 ± 21.6	119.8 ± 15.6	0.134
DBP, mmHg	74.3 ± 12.0	65 7 ± 8.4	0.041
MAP, mmHg	93.2 ± 13.2	83.7 ± 8.9	0.040
IAP, mmHg	10 ± 4.3	10.8 ± 3.5	0.580
APP, mmHg	83.2 ± 12.8	72.9 ± 6.2	0.019

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, IAP: Intra abdominal pressure, APP: Abdominal perfusion pressure.

Heart Failure

OP-077

The relationship between the MAGGIC score and cardiorenal syndrome in patients with acute decompensated heart failure with reduced ejection fraction

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Background and Aim: The Meta-Analysis Global Group in Chronic Heart Failure (MAGGIC) is a scoring system that is easy to use in outpatient clinics or at the bedside and was developed to predict the survival of heart-failure patients after hospitalization. This study aims to evaluate the relationship between the MAGGIC score and cardiorenal syndrome (CRS) in patients with acute decompensated heart failure with reduced ejection fraction (HFrEF).

Methods: For this retrospective, single-center study, 706 patients includes with New York Heart Association (NYHA) II-IV who were hospitalized and discharged due to acute decompensated heart failure between 2016 and 2021. CRS type 1 is defined an acute worsening of cardiac function leading to renal dysfunction. Patients were divided into two groups: those with CRS and those without. The MAGGIC score of all patients was determined. The primary outcome of this study was the occurrence of CRS.

Results: CRS developed in 132 patients. MAGGIC scores were observed to be higher in CRS (+) patients compared to CRS (-) patients (30.70 ± 8.09 vs. 23.96 ± 5.59, p<0.001). After a multivariable analysis, MAGGIC score (OR: 3.92, p<0.001), sodium (OR: 0.92, p=0.003), NT-pro-BNP (OR: 1.78, p=0.009), hs troponin (OR: 1.28, p=0.044), MRA (OR: 0.61, p=0.019) and furosemide dose (OR: 1.03, p=0.001) were found to be independent predictors of CRS development. The MAGGIC score was associated CRS development (AUC: 0.778).

Conclusions: The MAGGIC score can be associated with CRS in HFrEF patients.

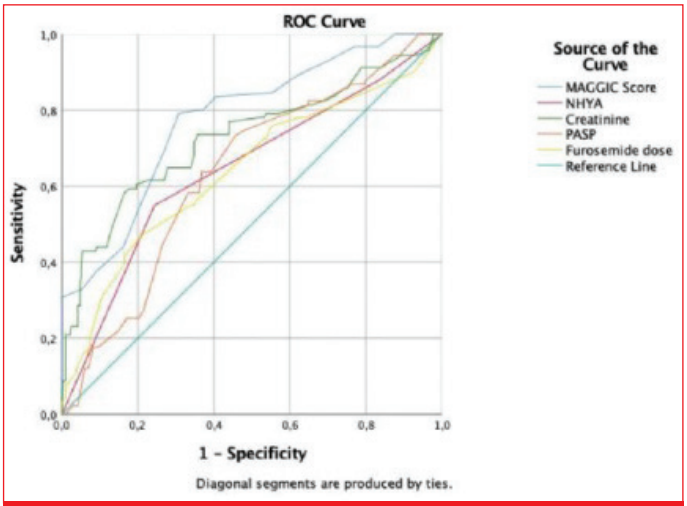

Figure 1. The ROC curves for MAGGIC scores, NYHA Class, Creatinine, PASP and furosemide dose.

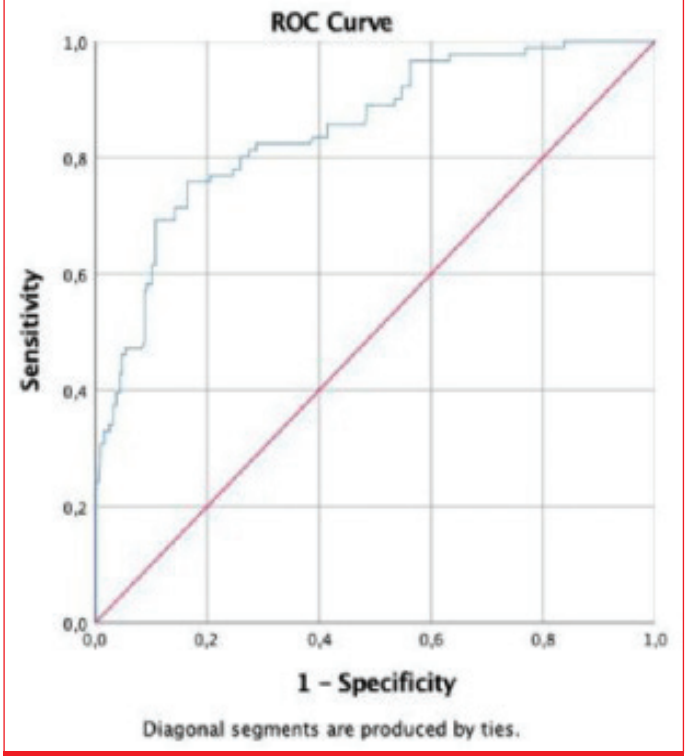

Figure 2. ROC analysis with a single model created with all of the combine data (MAGGIC score, NYHA, creatinine, PASP, furosemide dose)

Table 1. Univariate and multivariate logistic regression analyzes to identify independent predictors of CRS development in HFrEF patients.

Variable	Univariate			Multivariate		
	OR	95% CI	p	OR	95% CI	p
MAGGIC score	2.23	1.68-3.79	<0.001	2.75	1.73-4.04	<0.001
NYHA Class	2.45	1.82-3.29	<0.001	1.76	1.02-3.03	0.040
LVEF	0.36	0.21-0.52	<0.001	0.03	0.01-3.10	0.140
SBP (mmHg)	0.98	0.97-0.98	0.001	0.99	0.97-1.01	0.270
Hemoglobin	0.80	0.73-0.88	<0.001	0.97	0.83-1.12	0.680
HT	0.72	0.49-1.05	0.090			
DM	1.67	1.14-2.44	0.008	0.55	0.28-1.08	0.080
Creatinine	1.66	1.19-2.56	<0.001	1.75	1.15-3.31	<0.001
Sodium	0.87	0.84-0.90	<0.001	0.86	0.81-0.91	<0.001
CRP	1.03	1.01-1.05	0.010	1.00	0.99-1.02	0.960
Albumin	0.66	0.49-0.89	0.008	0.68	0.39-1.21	0.192
PASB (mmHg)	1.02	1.01-1.04	0.001	1.04	1.01-1.06	0.002
Furosemide dose	1.03	1.02-1.05	<0.001	1.03	1.01-1.05	0.005
ACEi /ARB	0.34	0.23-0.50	<0.001	0.46	0.29-0.58	0.020
Statin	0.61	0.41-0.89	0.012	0.75	0.36-1.58	0.450

Abbreviations: CRS, Cardiorenal syndrome; NYHA, New York Heart Association; LVEF, Left ventricular ejection fraction; SBP, Systolic blood pressure; HT, Hypertension; DM, Diabetes mellitus; CRP, C-reactive protein; PASP, Pulmonary artery systolic pressure; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin-receptor blockers.

Heart failure

OP-078

An updated meta-analysis of randomized controlled trials investigating omecamtiv mecarbil in the management of heart failure patients

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Background and Aim: Omecamtiv mecarbil (OM) is a selective cardiac myosin activator that improves cardiac contractility by enhancing cardiac sarcomere function without increasing intracellular myocyte calcium levels. We aimed to conduct an updated meta-analysis of randomized controlled trials (RCTs) investigating the effect of OM in heart failure (HF) patients by adding the recently published study METEORIC-HF.

Methods: We searched PubMed, ClinicalTrials.gov, and Cochrane libraries for eligible RCTs that compared OM with placebo in HF patients. All pooled effect sizes were calculated with a fixed-effect model based on low heterogeneity between studies and represented as risk ratio (RR) with a 95% confidence interval.

Results: Five RCTs with 9640 patients were included. Patients were followed up for longest 24 months. There were no differences between OM and placebo with respect to all-cause death (RR: 1.0; 0.93-1.07, p=0.96), cardiac death (RR: 1.01; 0.92-1.10, p=0.86), heart failure event (RR: 0.95; 0.89-1.01, p=0.1), heart failure hospitalization (RR: 0.97; 0.90-1.04, p=0.35).

Conclusions: This updated meta-analysis with 5 RCTs showed that omecamtiv mecarbil was not superior to placebo for all-cause mortality, cardiac death, HF event, and HF hospitalization in heart failure patients.

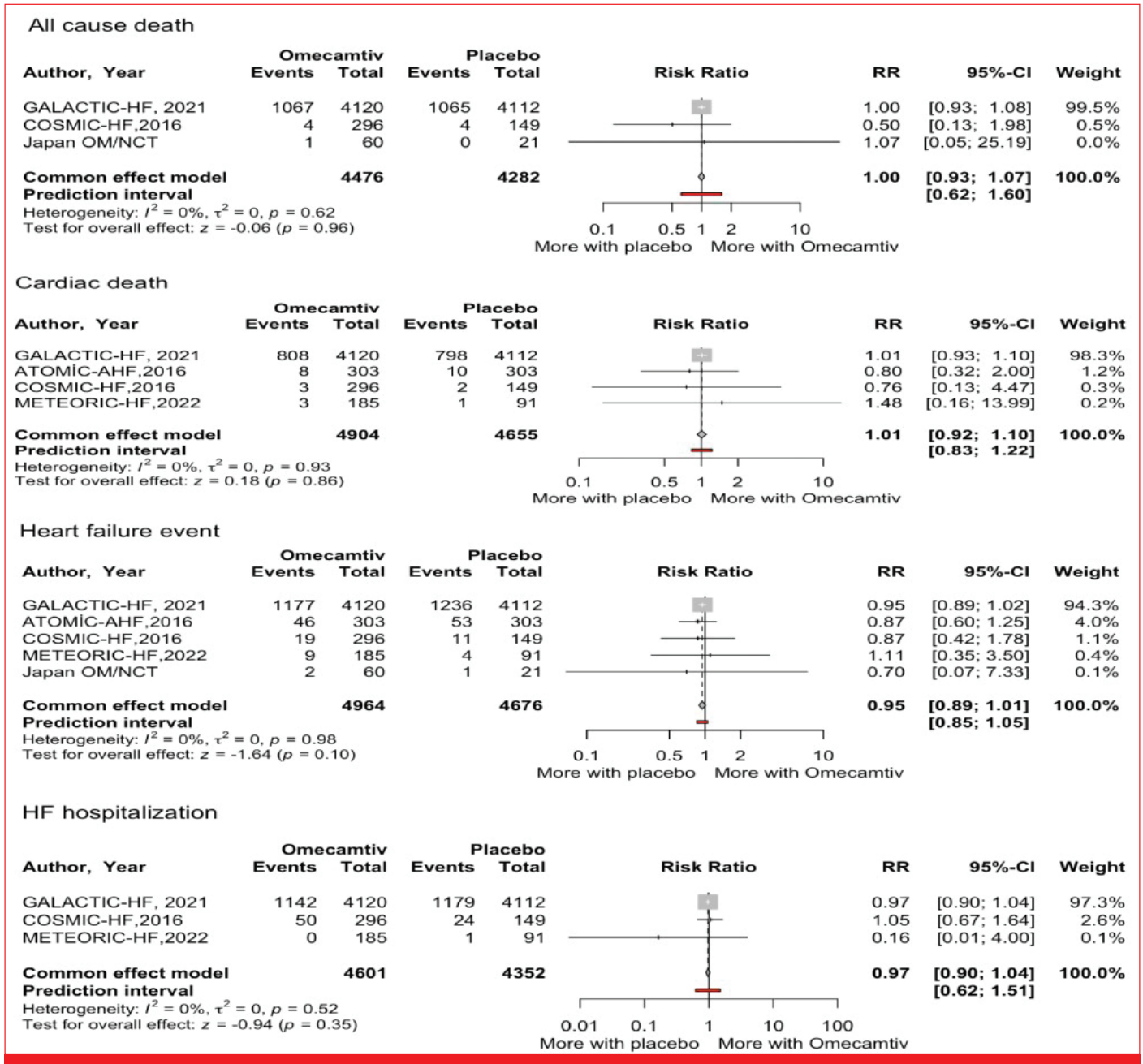

Figure 1. Forest plots of outcomes including all-cause death, cardiac death, heart failure event, and hospitalization.

Heart Failure

OP-079

Urinary sodium excretion dynamics in heart failure patients

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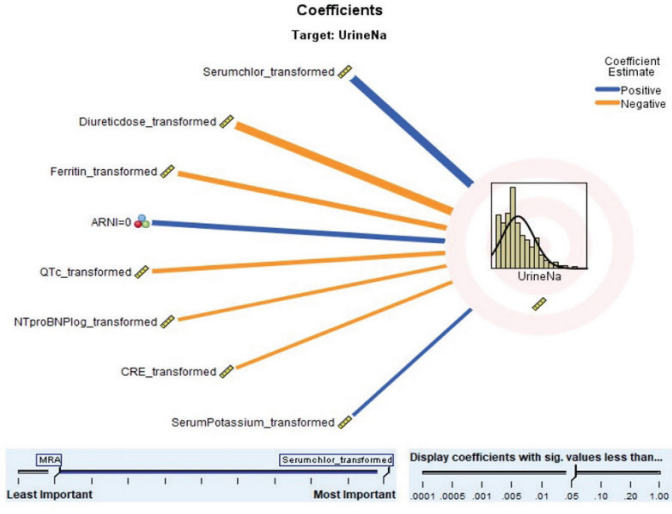
Background and Aim: Excessive sodium and water retention in heart failure patients leads to congestion, hospitalization, and reduced survival rates. Understanding sodium retention and intervention impacts is lacking. Thus, this prospective observational study seeks to identify factors influencing urinary sodium excretion.

Methods: The study examined outpatient heart failure patients tracked from 2020 to 2023. Follow-up duration varied by patients' heart failure status. Analysis covered 335 patients with 791 follow-up records. During visits, urine sodium was categorized into: low (<50), low-normal (50-80), normal-high (80-100), and high (>100) excretion. Noted is no baseline-patient urine sodium group comparison. Regular follow-up tests used one-way ANOVA, categorical variables employed chi-square tests. Long-term dependent variables on urine Na were investigated using automatic linear modeling regression; model excluded colinear variables.

Results: The mean age was years old, and were male. The mean LVEF was %, and 791 follow-up median intervals were 84 (28-180) days. Urinary sodium excretion is increased by systolic blood pressure increments. NYHA functional class has significantly differed urinary sodium excretion; nevertheless, NYHA functional class I heart failure patients had a higher ratio of low urinary sodium excretion. Heart failure guideline-directed medical therapies had non-linear characteristics for urine sodium excretion. According to an automatic linear regression model, eight variables influence urine sodium excretion. The variable with the most significant impact on this process is serum chlor with interaction (OR: 0.369, 95% CI: 2.75-4.54, p<0.0001). As the dose of loop diuretic increases, urinary excretion decreases (OR: -0.388, 95% CI: -0.45 to -0.32, p<0.0001). Patients taking angiotensin receptor blockers and neprisilin inhibitors have a negative correlation (OR, 95% CI: -35.2 to -17.9, p<0.0001). In the multivariable regression analysis, diuretic dose, taking ARNI, increasing QTc interval, NT-pro-BNP, and ferritin levels were negatively related to urine Na. However, heart failure patients with high serum chlor levels had increased urine Na excretion. To examine the interaction between NYHA functional class and diuretic dose, a linear mixed regression model was used for each heart failure patient. The fixed effect variables were NYHA functional class, diuretic dose, and NYHA functional class diuretic dose, and each follow-up was selected as a random effect. In the linear mixed regression model, diuretic dose according to NYHA functional class did not show a significant relationship with urine Na (F test: 2.009, p=0.34).

Conclusions: Urinary sodium excretion is regulated in a complex manner in patients with heart failure. Increasing the diuretic dose does not simply increase urinary sodium; the degree of heart failure determined by different variables had interactions. Joint modeling regression endpoint-free survival analyses could clarify the issue.



Nodes	Importance	V5
Serum Potassium	0.0473	0.0473
Creatinin	0.0492	0.0492
NTproBNP (log)	0.0536	0.0536
QTc	0.079	0.0790
ARNI	0.0886	0.0886
Ferritin	0.1079	0.1079
Diuretic dose	0.209	0.2090

Figure 1. Automatic linear regression modeling, urine Na most important variables, p<0.05.

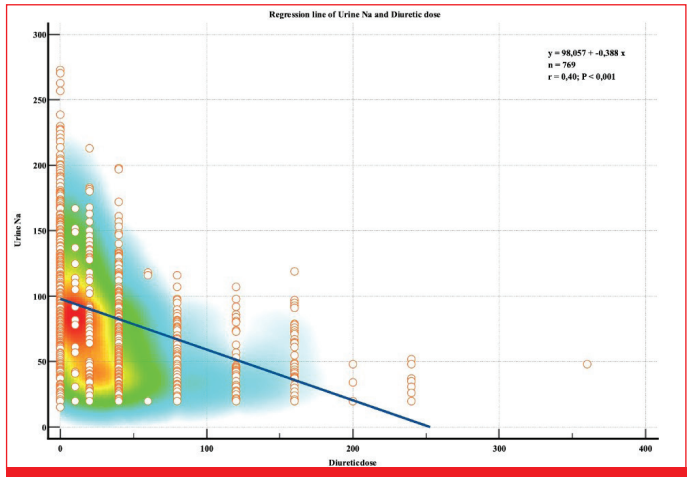


Figure 2. Regression line of urine Na and diuretic dose.

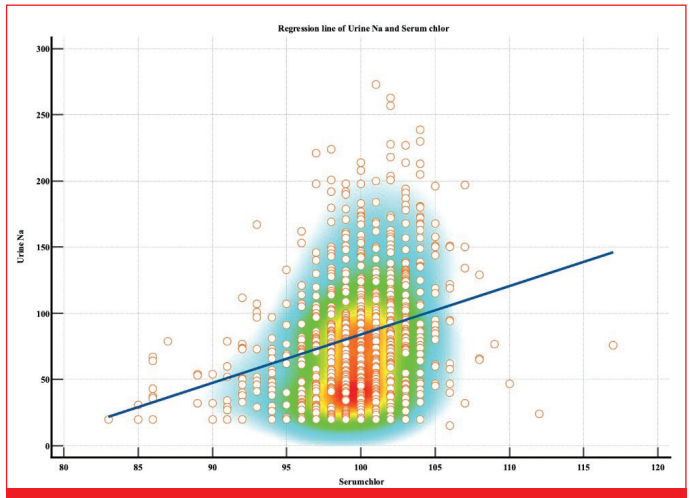


Figure 3. Regression line of urine Na and serum chlor.

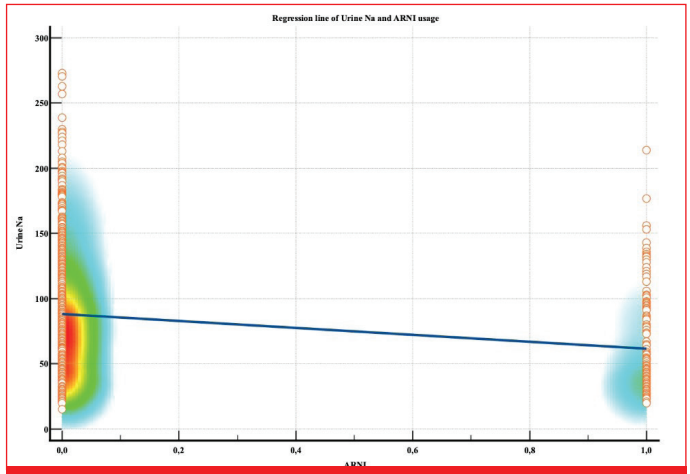


Figure 4. Regression line of urine Na and ARNI usage.

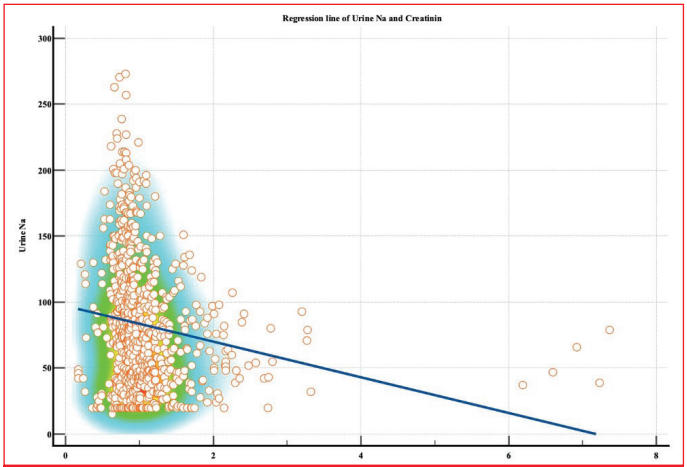


Figure 5. Regression line of urine Na and creatinine.

Table 1. Patient characteristics, repeated measures of follow-up variables and comparison followed-up parameters One-way ANOVA

Urine Na excreters		**Low**	**Low-Normal**	**Normal-High**	**High**	
Urine sodium, mEq/L	**Total**	**<50**	**50-80**	**80-100**	**≥100**	**p**
Age, years*	50.5±13.2	-	-	-	-	-
Male, %*	74.9	-	-	-	-	-
BMI, kg/m^2	27.6±4.6	-	-	-	-	-
SBP, mmHg	117 ± 21	112 ± 21	116 ± 21	120 ± 22	121 ± 20	<0.0001
DBP, mmHg	74 ± 13	72 ± 13	73 ± 13	74 ± 12	75 ± 13	0.06
NYHA FC, %	-	-	-	-	-	<0.0001
I	46.1	25.6	19.4	14.4	40.6	-
II	40.2	37.7	22.2	15.4	24.7	-
III-IV	13.7	8.5	3.3	6.2	2.1	-
Ischemic etiology, %*	35.2	-	-	-	-	-
Arterial hypertension, %*	26.9	-	-	-	-	-
Diabetes mellitus, %*	23.6	-	-	-	-	-
Dyslipidemia, %*	21.2	-	-	-	-	-
Smoking history, %*	46.6	-	-	-	-	-
Cerebrovascular event, %*	4.5	-	-	-	-	-
COPD, %*	12.5	-	-	-	-	-
Renal disease, %*	0.6	-	-	-	-	-
Chronic liver disease, %*	1.8	-	-	-	-	-
Peripherial arterial disease, %*	0.6	-	-	-	-	-
AF, %*	13.2	-	-	-	-	-
Intracardiac device	-	-	-	-	-	-
CRT-D, %*	7.8	-	-	-	-	-
ICD, %*	34.9	-	-	-	-	-

Na:Sodium, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, NYHA FC: New York Heart Association functional classification, COPD: Chronic obstructive pulmonary disease, AF: Atrial fibrillation, CRT-D: Cardiac resynchronization therapy defibrillator, ICD: Implantable cardioverter-defibrillator. *Denotes single and first measurement of variables of patients.

Table 2. Medication, repeated measures of follow-up variables and comparison followed-up parameters One-way ANOVA

Urine Na excreters		**Low**	**Low-Normal**	**Normal-High**	**High**	
Urine sodium, mEq/L	**Total**	**<50**	**50-80**	**80-100**	**≥100**	**p**
Medication	-	-	-	-	-	-
ACEi or ARB, %	71.5	25.7	21.7	15.5	37.2	<0.0001
ARNI, %	9.3	53.8	16.7	13.5	16.0	<0.0001
Beta-blocker, %	88.3	32.7	20.1	14.4	32.8	0.033
Aldosterone antagonist, %	59.2	36	18.3	15	30.7	0.085
SGLT-2 inhibitor, %	11.7	36.6	19.9	22	21.5	0.001
Loop diuretic, %	49.5	46.7	21.1	14.5	17.6	<0.0001
Loop diuretic dose	39 ± 52	65 ± 61	43 ± 53	33 ± 41	11 ± 21	<0.0001
Thiazide diuretic, %	21.6	28.6	25	15.3	31.1	0.22
Anticoagulant, %	24	36.4	22.1	13.4	28.1	0.42
Antiplatelet, %	45.1	32.5	21.8	16.4	29.3	0.45
If channel blocker, %	16.7	41.4	21.8	15.8	21.1	0.030
Digoxine, %	5.7	42.4	22.7	4.5	30.3	0.067
Antihyperlipidemic, %	31.3	30.6	21.8	17.3	30.3	0.34

ACEi:Angiotensin-converting enzyme inhibitors, ARB: Angiotensin receptor blockers, ARNI: Angiotensin receptor/neprilysin inhibitor, SGLT-2: Sodium-glucose cotransporter-2.

Table 3. Electrocardiography, repeated measures of follow-up variables and comparison followed-up parameters One-way ANOVA

Urine Na excreters		**Low**	**Low-Normal**	**Normal-High**	**High**	
Urine sodium, mEq/L	**Total**	**<50**	**50-80**	**80-100**	**≥100**	**p**
Electrocardiography	-	-	-	-	-	-
HR, beat/min	74 ± 15	77 ± 15	74 ± 15	73 ± 15	73 ± 14	0.017
PR interval, msec	169 ± 29	169 ± 32	174 ± 32	171 ± 29	165 ± 23	0.063
QRS duration, msec	121 ± 27	123 ± 28	119 ± 25	123 ± 28	121 ± 28	0.38
QT interval, msec	455 ± 46	466 ± 49	454 ± 50	457 ± 44	442 ± 36	<0.0001

HR: Heart rate.

Table 4. Echocardiography, repeated measures of follow-up variables and comparison followed-up parameters One-way ANOVA

Urine Na excreters		**Low**	**Low-Normal**	**Normal-High**	**High**	
Urine sodium, mEq/L	**Total**	**<50**	**50-80**	**80-100**	**≥100**	**p**
Echocardiography	-	-	-	-	-	-
LVEDd, mm*	5.91 ± 1.05	-	-	-	-	-
LVESd, mm*	4.68 ± 1.26	-	-	-	-	-
LAd, mm*	4.25 ± 0.84	-	-	-	-	-
LVEF, %*	36.1 ± 14.2	-	-	-	-	-
TAPSE, mm*	19.5 ± 5.4	-	-	-	-	-
RVsm TDI, m/sec*	10.6±2.8	-	-	-	-	-
TRV, m/sec*	2.57 ± 0.65	-	-	-	-	-
SPAP, mmHg*	50.2 ± 16.9	-	-	-	-	-
Mitral regurgitation, %*	-	-	-	-	-	-
None	22.7	-	-	-	-	-
Mild	43	-	-	-	-	-
Moderate	25.4	-	-	-	-	-
Severe	9	-	-	-	-	-
Aort Regurgitation %*	-	-	-	-	-	-
None	80	-	-	-	-	-
Mild	16.4	-	-	-	-	-
Moderate	0.9	-	-	-	-	-
Severe	2.7	-	-	-	-	-
Tricuspide Regurgitation %*	-	-	-	-	-	-
None	31	-	-	-	-	-
Mild	51.6	-	-	-	-	-
Moderate	11	-	-	-	-	-
Severe	6.3	-	-	-	-	-

LVEDd: Left ventricular end-diastolic diameter, LVESd: Left ventricular end-systolic diameter, LAd: Left atrium diameter, LVEF: Left ventricular ejection fraction, TAPSE: Tricuspid annular plane systolic excursion, RVsm TDI: Right ventricular systolic velocity tissue doppler imaging, TRV: Tricuspid regurgitation peak velocity, SPAP: Systolic pulmonary artery pressure. * Denotes single and first measurement of variables of patients.

Table 5. Laboratory tests, repeated measures of follow-up variables and comparison followed-up parameters One-way ANOVA

Urine Na excreters		**Low**	**Low-Normal**	**Normal-High**	**High**	
Urine sodium, mEq/L	**Total**	**<50**	**50-80**	**80-100**	**≥100**	**p**
Laboratory tests	-	-	-	-	-	-
SGOT, U/L (IQR)	19.8 ± 10	21.3 ± 12	18.4 ± 8.1	18.2 ± 7.1	19.9 ± 11.5	0.015
SGPT, U/L (IQR)	22.6 ± 19	23.6 ± 21	20.9 ± 18.4	23.4 ± 23.1	22.4 ± 14.9	0.54
ALP, U/L (IQR)	83.7 ± 36	88.1 ± 40	87.2 ± 39	81.9 ± 36	77.6 ± 26.8	0.006
GGT, U/L (IQR)	44.1 ± 59	56.3 ± 74	47.5 ± 60	46.2 ± 64	27.7 ± 22.6	<0.0001
Total bilirubin, mg/dL (IQR)	0.70 ± 0.43	0.80 ± 0.48	0.76 ± 0.51	0.63 ± 0.36	0.58 ± 0.29	<0.0001
Indirect bilirubin, mg/dL (IQR)	0.37 ± 0.25	0.42 ± 0.27	0.38 ± 0.30	0.33 ± 0.18	0.32 ± 0.21	<0.0001
NT-pro-BNP, pg/mL (IQR)	2613 ± 5853	3915 ± 7018	3103 ± 7387	2705 ± 5215	827 ± 1749	<0.0001
Total cholosterole, mg/dL	162 ± 42	163 ± 46	153 ± 42	166 ± 40	166 ± 39	0.018
Triglyceride, mg/dL	145 ± 114	147 ± 155	129 ± 72	147 ± 89	154 ± 93	0.21
HDL cholosterole, mg/dL	43.7 ± 13.4	43.9 ± 13.5	41.8 ± 11.5	44.0 ± 15.2	44.6 ± 13.5	0.22
LDL cholosterole, mg/dL	91.1 ± 33.8	90.4 ± 36	86.5 ± 33.5	93.2 ± 32	93.9 ± 32.2	0.17
Fasting glucose, mg/dL	112 ± 41	112 ± 41	117 ± 51	117 ± 44	105 ± 30	0.016
Urea, mg/dL	45 ± 31	50 ± 33	54 ± 37	45 ± 35	34 ± 15	<0.0001
Creatinine, mg/dL	1.08 ± 0.63	1.15 ± 0.75	1.22 ± 0.84	1.08 ± 0.48	0.90 ± 0.26	<0.0001
Sodium, mEq/L	138 ± 3.2	138 ± 3.3	137 ± 3.1	138 ± 2.5	138 ± 3.3	0.005
Potassium, mEq/L	4.5 ± 0.47	4.4 ± 0.49	4.6 ± 0.54	4.6 ± 0.41	4.5 ± 0.39	0.004
Chlorur, mEq/L	99.5 ± 3.7	98.4 ± 4.1	99.1 ± 4.3	100 ± 2.7	100 ± 2.7	<0.0001
Serum osmality, mOsm/kg	290 ± 12	289 ± 18	291 ± 7	291 ± 9	289 ± 7	0.33
WBC, 10^3/µL	8156 ± 2227	8414 ± 2396	7891 ± 2093	8103 ± 2102	8077 ± 2165	0.11
Hemoglobulin, g/dL	14.1 ± 1.86	14.1 ± 1.83	13.7 ± 2.0	14.0 ± 1.96	14.3 ± 1.7	0.009
Hematocrite, %	42.7 ± 5.1	42.8 ± 5.1	41.7 ± 5.5	42.6 ± 5.6	43.2 ± 4.4	0.052
Thrombocyte, 10^3/µL	231 ± 66	235 ± 72	224 ± 67	235 ± 59	231 ± 61	0.36
Serum iron, µg/dL	78 ± 30	78 ± 31	76 ± 27	77 ± 31	81 ± 30	0.40
TIBC, µg/dL	328 ± 60	324 ± 58	336 ± 61	336 ± 61	326 ± 59	0.27
Ferritin, µg/dL	152 ± 167	182 ± 191	144 ± 154	134 ± 129	135 ± 160	0.009
TSAT, %	24 ± 11	25 ± 10	24 ± 11	23.5 ± 11	25 ± 11	0.43

Na: Sodium, SGOT: Serum glutamic-oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase, ALP: Alkaline phosphatase, GGT:Gamma-glutamyl transferase, NT-pro-BNP=N-terminal pro-B-type natriuretic peptide, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, WBC: White blood cells, TIBC: Total ıron-binding capacity, TSAT: Transferrin saturation, IQR: Interquartile range.

Table 6. Laboratory tests, repeated measures of follow-up variables and comparison followed-up parameters One-way ANOVA

Urine Na excreters		**Low**	**Low-Normal**	**Normal-High**	**High**	
Urine sodium, mEq/L	**Total**	**<50**	**50-80**	**80-100**	**≥100**	**p**
Laboratory tests	-	-	-	-	-	-
Urine ceratine, mg/dL	131 ± 87	146 ± 103	121 ± 93	109 ± 74	133 ± 67	0.004
Urine potassium, mmol/L	53.6 ± 29	49 ± 27	46 ± 26	49.6 ± 28	65 ± 31	<0.0001
Urine chlorur, mmol/L	91 ± 63	38 ± 22	67 ± 28	98 ± 32	158 ± 56	<0.0001
Urine sodium, mmol/L	82.8 ± 50	38 ± 22	67 ± 28	98 ± 32	143 ± 36	-

Na: Sodium.

Table 7. Linear regression repeated measures follow-up variables

	Univariate analysis		Multivariate analysis	
	Beta coefficient 95% CI	p	Beta coefficient 95% CI	p
NYHA FC	-18.6 (-24.6 to -12.6)	<0.0001		
Systolic BP	0.391 (0.22-0.56)	<0.0001		
Diuretic döşe, mg	-0.388 (-0.45 to -0.32)	<0.0001	-0.17 (-.25 to -0.088)	<0.0001
RAAS inhibitor	23.4 (15.9-30.9)	<0.0001		
Beta-blocker	16.9 (3.4-30.4)	0.014		
MRA	-8.44 (-15.8 to -1.06)	0.025		
SGLT-2i	-12.9 (-21.2 to -0.47)	0.002		
ARNI	-26.6 (-35.2 to -17.9)	<0.0001	-13.08 (-21.8 to -4.3)	0.004
QTC	-0.25 (-032 to -0.17)	<0.0001	-0.11 (-0.19 to -0.03)	0.007
Heart rate	-0.434 (-0.66 to -0.19)	<0.0001		
NT-pro-BNP (log)	-24.07 (-28.8 to -19.3)	<0.0001	-7.44 (-13.8 to -1.0)	0.023
Hemoglobulin	1.72 (-0.19-3.64)	0.064		
SGOT	-0.245 (-0.57-0.09)	0.15		
Creatinin	-13.5 (-19 to -8.0)	<0.0001		
Serum Na	2.37 (1.28-3.46)	<0.0001		
Chlorur	0.369 (2.75-4.54)	<0.0001	2.48 (1.54-3.43)	<0.0001
Ferritin	-0.43 (-0.65 to -0.21)	<0.0001	-0.031 (-0.052 to -0.011)	0.002

NYHA FC: New York heart association functional classification, BP: Blood pressure, RAAS: Renin-angiotensin-aldosterone system, MRA: Mineralocorticoid receptor antagonists, SGLT-2i: Sodium/glucose cotransporter-2 inhibitors, ARNI: Angiotensin receptor/neprilysin inhibitor, NT-pro-BNP: N-terminal pro-B-type natriuretic peptide, SGOT: Serum glutamic oxaloacetic transaminase, N: Sodium, log: logarithm.

Heart Failure

OP-080

EUCOR-ICD trial; the benefit of implantable cardioverter defibrillator in ischemic and non-ischemic heart failure patients

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Background and Aim: Patients suffering from heart failure are at high risk of ventricular arrhythmia. While ICD implantation has been proven to increase the survival rate of ischemic HF patients, the same cannot be stated for non-ischemic HF patients. This study aims to investigate the efficacy of ICD in both ischemic and non-ischemic heart failure patients.

Methods: This study evaluates 1280 patients who received outpatient treatment for HF between 2008 and 2022. The study involves in ishemic or non-ischemic HF patients with LVEF 35% or less, excluding those with hypertrophic or restrictive cardiomyopathy. This study is a retrospective observational research. The primary outcome is a combination of all-cause death, LVAD implantation, heart transplantation, and ventricular arrhythmia.

Results: The patients' average age was 46.7 ± 12.1 years. The majority of patients (76.2%) were male, and 61.6% had ischemic HF. Almost half (46.3%) of the patients received an ICD, and 9.8% of those had CRT. The median follow-up period was 56.5 (28.4-87.7) months. The mean LVEF for both the ICD and control groups was similar (24.9 ± 5.5; 24.3 ± 4.9, respectively; p=0.72). 305 (51.4%) patients in the ICD group experienced the primary outcome, while 356 (51.8%) patients in the control group experienced the same. The hazard ratio for the primary outcome in the ICD group was 0.72 (95% CI: 0.61-0.84; p<0.0001). When analyzing subgroups based on the cause of HF, it was found that patients with ischemic HF continued to benefit from ICD (HR: 0.72, 95% CI: 0.60-0.87, p=0.006). However, for patients with non-ischemic HF, there was no significant benefit from ICD (HR: 0.77, 95% CI: 0.58-1.02, p=0.07). For the primary outcome in the Cox regression model, beta-blocker, loop diuretic, TAPSE, and NT-pro-BNP were found to be independent predictors in patients without an ICD. In the multivariate model for ICD-implanted patients, systolic blood pressure, ischemic HF, NYHA functional class, CRT function, mitral regurgitation, TAPSE, and NT-pro-BNP were all independent predictors for the primary outcome. In this study, we calculated the predictive values of LVEF, TAPSE, and NT-pro-BNP levels using ROC analysis. Additionally, we calculated the combined predictive probability along with NYHA functional class and other factors. Our results

showed that NT-pro-BNP had a higher predictive value than LVEF and TAPSE in a single analysis (AUC: 0.75 vs. 0.69 and 0.70, respectively). However, when we considered the combined predictive probability, it had a superior predictive value (AUC: 0.80, p<0.0001).

Conclusions: The study shows a significant discrepancy for primary outcome benefit of ICD between ischemic and non-ischemic HF patients. Ischemic group exhibited significantly lower benefit compared to non-ischemic patients.According to the findings, it has been found that the combination of predictive probabilities derived from LVEF, TAPSE, NTpro-BNP, and NYHA functional class results in a significantly more accurate prediction of the primary outcome.

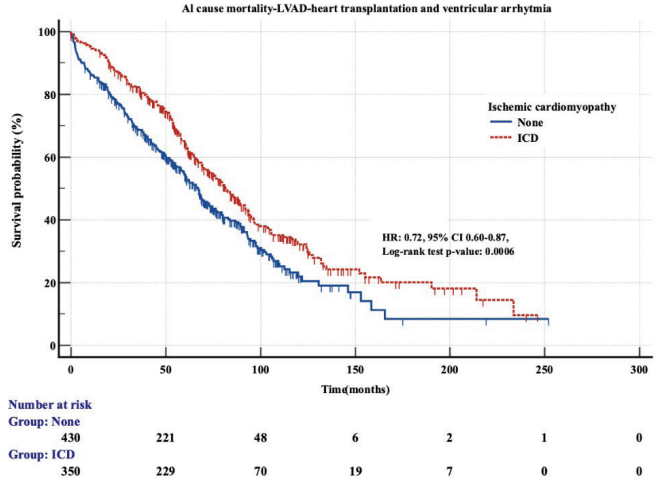

Figure 1. All-cause mortality-LVAD-heart transplantation-ventricular arrythmia (ischemic cardiomyopathy--none/ICD).

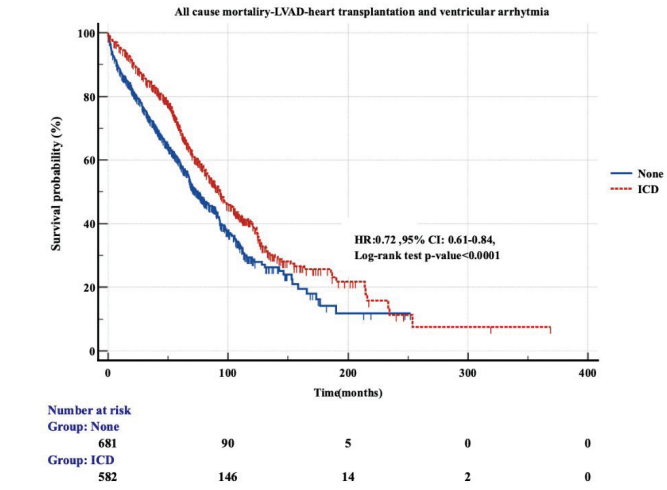

Figure 2. All-cause mortality-LVAD-heart transplantation-ventricular arrythmia (none/ICD).

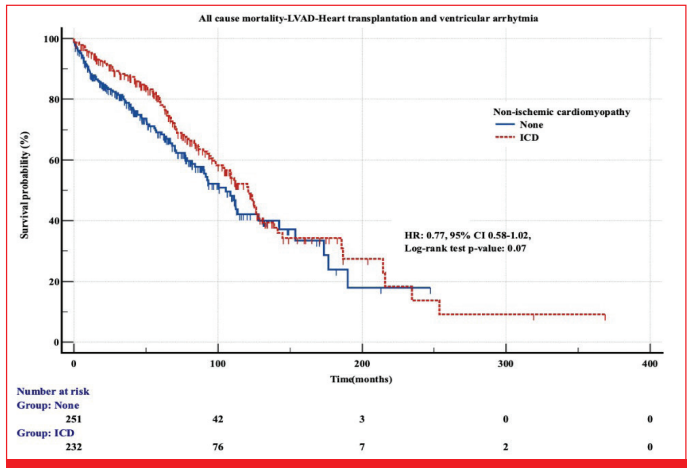

Figure 3. All-cause mortality-LVAD-heart transplantation-ventricular arrythmia (non-ischemic cardiomyopathy--none/ICD).

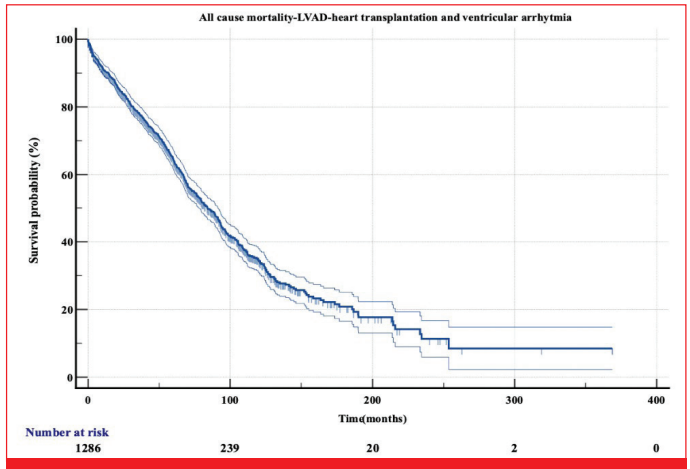

Figure 4. All-cause mortality-LVAD-heart transplantation-ventricular arrythmia (overall).

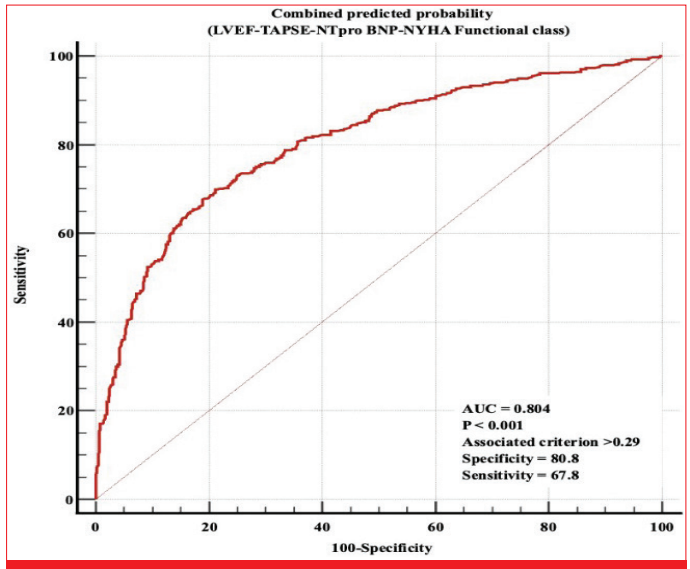

Figure 5. Combined predicted Probability (LVEF-NT-pro-BNP-TAPSE-NYHA functional class).

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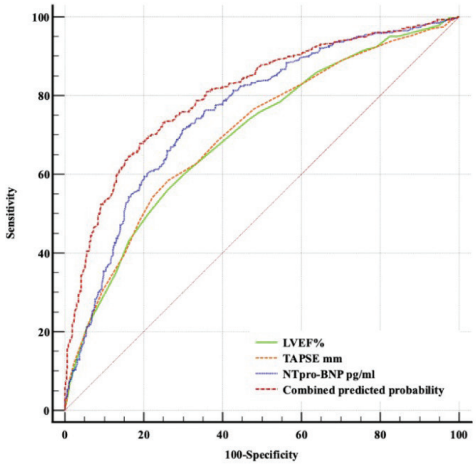

	AUC and 95%CI	LVEF %	TAPSE mm	NTpro-BNP pg/ml	Combined predicted probability
LVEF %	0.69 (0.66-0.72)	-	0.77	0.0011	<0.0001
TAPSE mm	0.70 (0.67-0.73)	0.77	-	0.003	<0.0001
NTpro-BNP pg/ml	0.75 (0.72-0.78)	0.0011	0.003	-	0.0002
Combined predicted probability	0.80 (0.77-.082)	<0.0001	<0.0001	0.0002	-

Figure 6. Comparison of predictive values of LVEF, TAPSE, NT-pro-BNP and combined evaluation.

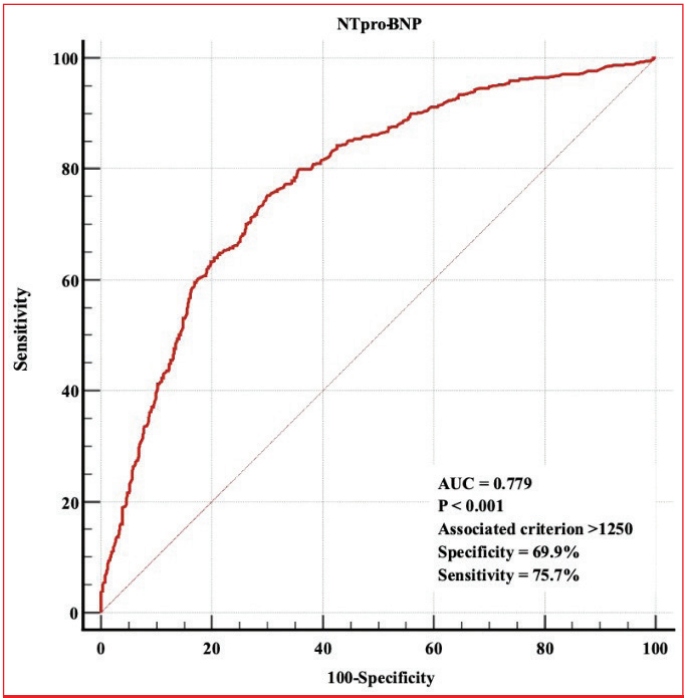

Figure 8. Predictive evaluation of NT-pro-BNP using ROC analysis.

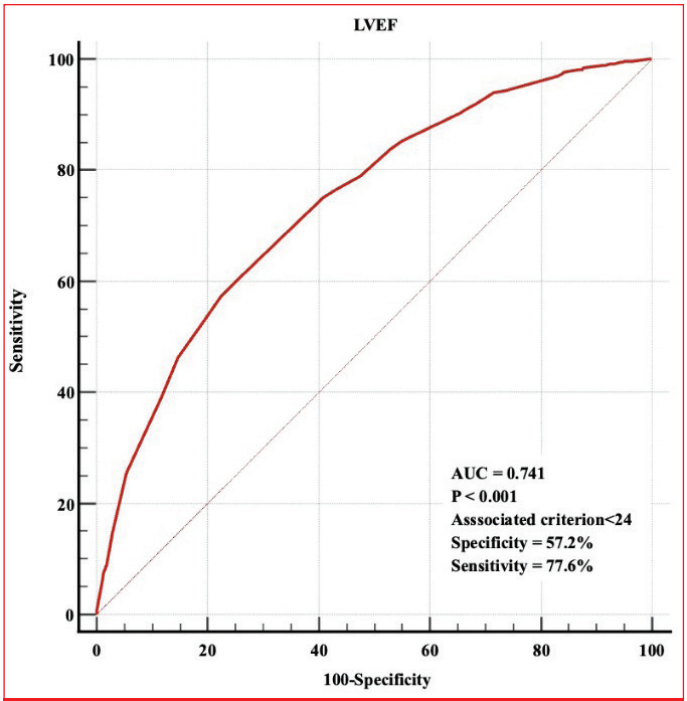

Figure 7. Predictive evaluation of LVEF using ROC analysis.

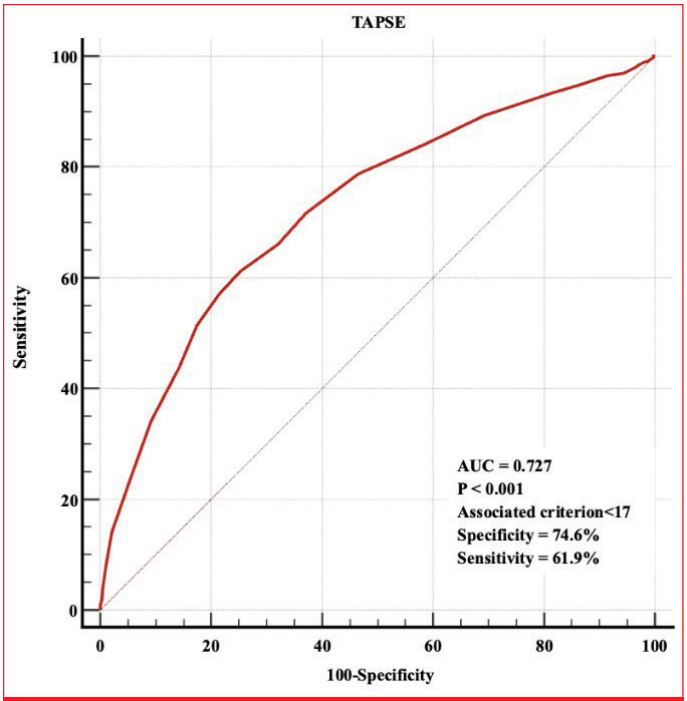

Figure 9. Predictive evaluation of TAPSE using ROC analysis.

Table 1. Basal characteristics of all patients-1

	Total	None	ICD	p
Age, years	46.7 ± 12.1	45.7 ± 12.7	48.0 ± 11.1	0.001
Male, %	76.2	73.2	79.6	0.008
BMI, kg/m^2	26.5 ± 3.9	26.3 ± 4.0	26.7 ± 3.7	0.24
BSA, m^2	1.91 ± 0.18	1.91 ± 0.18	1.92 ± 0.18	0.31
SBP, mmHg	114 ± 18	116 ± 18	111 ± 17	<0.0001
DBP, mmHg	69.5 ± 11.2	70 ± 11	68 ± 10	0.011
NYHA FC	-	-	-	<0.0001
I	12.4	16.9	8.1	-
II	48.9	50.3	47.5	-
III	32.3	28.0	36.4	-
IV	6.4	4.8	7.9	-
Ischemic etiology	61.6	63	60	0.27
Arterial hypertension, n (%)	34.8	32.0	38.0	0.025
Diabetes mellitus, n (%)	29.0	26.8	31.6	0.059
Dyslipidemia, n (%)	17.7	14.4	21.5	0.001
Smoking history, n (%)	40.5	39.1	42.1	0.29
Cerebrovascular event, n (%)	4.9	5.4	4.2	0.32
COPD, n (%)	5.9	6.4	5.3	0.41
Renal disease, n (%)	2.9	3.4	2.4	0.29
Chronic liver disease, n (%)	0.5	0.1	0.4	0.40
Peripherial arterial disease, n (%)	2.5	2.7	1.4	0.74
AF, n (%)	16.5	17.5	15.2	0.29
CRT, %	9.8	-	21.1	-
Family history of heart failure, n (%)	13.3	11.4	15.6	0.025
NT-pro-BNP, pg/mL	1441 (588-4073)	1488 (578-4440)	1409 (610-3568)	0.34

Table 2. Basal characteristics of all patients-2

	Total	None	ICD	p
ACEi or ARB, n (%)	79.3	84.3	73.5	<0.0001
ARNI, n (%)	9.1	8.9	9.3	0.80
Beta-blocker, n (%)	98.2	99.0	97.3	0.024
Aldosterone antagonist, n (%)	92.4	93.0	91.7	0.39
SGLT2-i, n (%)	15.9	14.4	17.5	0.12
Loop diuretic, n (%)	84	78.5	90.4	<0.0001
Anticoagulant, n (%)	29.4	27.9	31	0.22
Antiplatelet, n (%)	58.5	55.7	61.1	0.031
If channel blocker, n (%)	33.6	31.1	36.4	0.046
Digoxine, n (%)	17.8	16.2	19.7	0.096
Antihyperlipidemic, n (%)	38.4	30.6	47.6	<0.0001
Antiarrhytmic, n (%)	9.9	3.9	14.4	<0.0001
Primary end-point, n (%)	51.8 (661)	52.0 (356)	51.7 (305)	0.92
Follow-up time median IQR, days	56.5 (28.4-87.7)	49.1 (21.6-77.9)	64.3 (38.9-99.9)	<0.0001
LVEDd, mm	65.0 ± 8.9	64.3 ± 9.1	65.8 ± 8.5	0.003
LVESd, mm	55.4 ± 10.1	54.7 ± 10.3	56.3 ± 9.7	0.004
LAd, mm	47.1 ± 7.0	47.1 ± 7.5	47.3 ± 6.4	0.58

Table 3. Basal characteristics of all patients-3

	Total	None	ICD	p
LVEF, %	24.6 ± 5.3	24.9 ± 5.5	24.3 ± 4.9	0.72
Mitral regurgitation, n (%)	-	-	-	<0.0001
Mild	25.3	21.2	30.2	
Moderate	45.4	45.4	45.4	
Severe	22.7	13.6	9.1	
Aortic regurgitation, n (%)	-	-	-	0.97
Mild, %	16.9	17.0	16.8	
Moderate, %	5.0	5.3	4.8	
Severe, %	0.6	0.6	0.7	
Tricuspid regurgitation, n (%)	-	-	-	0.078
Mild, %	36.0	33.0	39.4	
Moderate, %	33.9	34.5	33.3	
Severe, %	15.3	17.0	13.4	
TAPSE, mm	16.8 ± 4.8	16.5 ± 4.9	17.1 ± 4.7	0.032
RVsm (TDI), m/sec	9.9 ± 2.6	9.8 ± 2.5	10.1 ± 2.7	0.019
TRV, m/sec	2.87 ± 0.53	2.86 ± 0.52	2.89 ± 0.55	0.38
SPAP, mmHg	36.9 ± 15.5	36.6 ± 1.52	37.3 ± 15.9	0.43

Table 4. Univariate/multivariate analysis of all patients-1

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p	HR (95% CI)	p
Age, years	1.007 (1.000-1.014)	0.40	-	-
Gender (male)	1.170 (0.975-1.404)	0.87	-	-
BMI, kg/m^2	0.954 (0.927-0.981)	0.001	1.048 (1.004-1.095)	0.03
BSA, m^2	0.480 (0.263-0.876)	0.017	-	-
SBP, mmHg	0.981 (0.975-0.986)	<0.0001	0.990 (0.980-1.000)	0.04
DBP, mmHg	0.980 (0.971-0.989)	<0.0001	-	-
NYHA FC	-	<0.0001	-	0.029
I	-	-	-	-
II	1.553 (1.030-2.340)	0.036	1.103(0.577-2.110)	0.76
III	3.842 (2.564-5.756)	<0.0001	1.789 (0.910-3.516)	0.09
IV	5.683 (3.611-8.944)	<0.0001	1.841 (0.763-4.444)	0.17
Arterial hypertension	0.862 (0.732-1.014)	0.07	0.694 (0.499-0.964)	0.029
Ischemic etiology	1.641 (1.390-1.938)	<0.0001	1.915 (1.284-2.857)	0.001
Diabetes mellitus	0.890 (0.838-1.169)	0.9	-	-
Dyslipidemia	0.894 (0.728-1.099)	0.28	-	-
Smoking history	1.225 (1.050-1.429)	0.01	*	*
COPD	1.021 (0.744-1.400)	0.089	-	-
Renal disease	1.195 (0.781-1.828)	0.41	-	-
CVA	1.210 (0.888-1.648)	0.22	-	-
Chronic liver disease	2.742 (1.014-7.311)	0.047	-	-
Peripherial arterial disease	0.815 (0.435-1.525)	0.52	-	-
Atrial fibrilation	1.280 (1.050-1.560)	0.014	*	*
CRT-D	0.571 (0.434-0.752)	<0.0001	*	*
Family history	-	-	-	-
ACEi or ARB	0.973 (0.808-1.171)	0.769	-	-
ARNI	0.718 (0.532-0.969)	0.03	*	*
Beta-blocker	0.471 (0.305-0.727)	0.001	*	*
Aldosterone antagonist	0.768 (0.584-1.011)	0.06	*	*

Table 5. Univariate/multivariate analysis of all patients-2

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p	HR (95% CI)	p
SGLT2-i	0.651 (0.517-0.819)	<0.0001	0.595 (0.394-0.899)	0.014
Loop diuretic	1.832 (1.396-2.404)	<0.0001	2.194 (1.138-4.230)	0.019
Anticoagulant	0.947 (0.802-1.118)	0.52	-	-
Antiplatelet	0.995 (0.853-1.160)	0.94	-	-
If channel blocker	0.908 (0.771-1.069)	0.24	-	-
Digoxine	1.060 (0.882-1.276)	0.53	-	-
Antiarrhytmic	1.356 (1.046-1.757)	0.021	-	-
Antihyperlipidemic	0.633 (0.538-0.744)	<0.0001	0.686 (0.471-1.001)	0.05
LVEDd, mm	1.025 (1.016-1.033)	<0.0001	*	*
LVESd, mm	1.208 (1.021-1.036)	<0.0001	-	-
LAd, mm	1.046 (1.036-1.056)	<0.0001	*	*
LVEF, %	0.908 (0.894-0.923)	<0.0001	*	*
Mitral regurgitation		<0.0001	*	*
Mild	0.767 (0.511-1.151)	0.20	-	-
Moderate	1.720 (1.182-2.503)	0.005	-	-
Severe	2.802 (1.907-4.115)	<0.0001	-	-
Tricuspide regurgitation	-	<0.0001	*	*
Mild	1.581 (1.161-2.153)	0.004	-	-
Moderate	3.185 (2.368-4.285)	<0.0001	-	-
Severe	4.576 (3.337-6.273)	<0.0001	-	-
Aort regurgitation		0.001	*	*
Mild	1.218 (0.999-1.486)	0.051	-	-
Moderate	1.714 (1.259-2.335)	0.001	-	-
SPAP, mmHg	1.023 (1.019-1.028)	<0.0001	*	*
TRV, m/sec	1.668 (1.441-1.931)	<0.0001	-	-
TDI, Rvsm	0.832 (0.806-0.858)	<0.0001	-	-
TAPSE, mm	0.896 (0.881-0.911)	<0.0001	0.954 (0.916-0.993)	0.020
NT-pro-BNP (log)	3.029 (2.641-3.473)	<0.0001	2.541 (1.726-3.742)	<0.0001

Table 6. Univariate/multivariate analysis of patients with ICD implantation-1

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p	HR (95% CI)	p
Age, years	1.023 (1.011-1.034)	<0.0001	*	*
Gender (male)	0.831 (0.625-1.105)	0.20	-	-
BMI, kg/m^2	0.990 (0.946-1.035)	0.64	-	-
BSA, m^2	0.784(0.306-2.008)	0.61	-	-
SBP, mmHg	0.978 (0.971-0.986)	<0.0001	0.991 (0.982-1.000)	0.03
DBP, mmHg	0.970 (0.958-0.982)	<0.0001	-	-
NYHA FC	-	<0.0001	-	0.001
I	-	-	-	-
II	0.798 (0.461-1.382)	0.42	0.585 (0.313-1.093)	0.09
III	2.095 (1.229-3.571)	0.047	1.065 (0.565-2.007)	0.8
IV	3.085 (1.707-5.575)	<0.0001	1.310 (0.627-2.734)	0.47
Arterial hypertension	1.035(0.818-1.309)	0.77	-	-
Ischemic etiology	1.574 (1.239-2.001)	<0.0001	1.631 (1.217-2.186)	0.001
Diabetes mellitus	1.023 (0.801-1.307)	0.85	-	-
Dyslipidemia	0.906 (0.672-1.223)	0.52	-	-
Smoking history	1.081 (0.861-1.357)	0.50	-	-
COPD	0.995 (0.609-1.624)	0.98	-	-
Renal disease	1.470 (0.782-2.763)	0.23	-	-
Chronic liver disease	1.865 (0.462-7.527)	0.38	-	-
Peripherial arterial disease	0.817 (0.363-1.838)	0.62	-	-
Atrial fibrilation	1.272 (0.935-1.730)	0.12	-	--
CRT	0.631 (0.472-.845)	0.002	0.634 (0.454-0.884)	0.007
Family history	0.616 (0.436-.871)	0.006	*	*
ACEi or ARB	0.889 (0.690-1.144)	0.36	-	-
ARNI	0.803 (0.520-1.240)	0.32	-	-
Beta-blocker	0.509 (0.292-.888)	0.017	*	*
Aldosterone antagonist	0.761 (0.508-1.139)	0.18	-	-
SGLT2-i	0.888 (0.652-1.209)	0.45	-	-

Table 7. Univariate/multivariate analysis of patients with ICD implantation-2

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p	HR (95% CI)	p
Loop diuretic	1.943 (1.136-3.325)	0.015	*	*
Anticoagulant	0.902 (0.708-1.150)	0.40	-	-
Antiplatelet	1.043 (0.829-1.313)	0.71	-	-
If channel blocker	1.029 (0.814-1.301)	0.80	-	-
Digoxine	1.172 (0.906-1.515)	0.22	-	-
Antiarrhytmic	1.419 (1.058-1.903)	0.02	-	-
Antihyperlipidemic	0.708 (0.564-0.890)	0.003	*	*
LVEDd, mm	1.020 (1.006-1.033)	0.004	*	*
LVESd, mm	1.022 (1.011-1.034)	<0.0001	-	-
LAd, mm	1.048 (1.030-1.066)	<0.0001	*	*
LVEF, %	0.933 (0.911-0.956)	<0.0001	*	*
Mitral regurgitation	-	<0.0001	-	0.009
Mild	0.804 (0.419-1.543)	0.51	1.088 (0.530-2.236)	0.81
Moderate	1.962 (1.053-3.653)	0.034	1.803 (0.923-3.523)	0.08
Severe	3.010 (1.588-5.705)	0.001	2.054 (1.029-4.102)	0.04
Tricuspide regurgitation	-	<0.0001	*	*
Mild	1.317 (0.858-2.022)	0.20	-	-
Moderate	2.356 (1.550-3.580)	<0.0001	-	-
Severe	3.351 (2.128-5.275)	<0.0001	-	-
Aort regurgitation	-	0.01	*	*
Mild	1.351 (1.004-1.817)	0.047	-	-
Moderate	1.747 (1.103-2.765)	0.017	-	-
Severe	2.548 (0.947-6.857)	0.064	-	-
TRV, m/sec	1.643 (1.319-2.048)	<0.0001	-	-
TDI Rvsm	0.871 (0.831-0.912)	<0.0001	-	-
TAPSE, mm	0.906 (0.882-.930)	<0.0001	0.966 (0.935-0.999)	0.04
SPAP, mmHg	1.020 (1.013-1.027)	<0.0001	*	*
NT-pro-BNP (log)	2.392 (1.958-2.922)	<0.0001	1.615 (1.226-2.127)	0.001

Table 8. Univariate/multivariate analysis of patients without ICD implantation-1

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	**p**	**HR (95% CI)**	**p**
Age, years	0.997 (0.989-1.006)	0.52	-	-
Gender (male)	0.813 (0.63-1.035)	0.09	*	*
BMI, kg/m^2	0.934 (0.901-0.967)	<0.0001	*	*
BSA, m^2	0.308 (0.142-0.668)	0.003	-	-
SBP, mmHg	0.981 (0.972-0.989)	<0.0001	*	*
DBP, mmHg	0.990 (0.977-1.002)	0.11	-	-
NYHA FC	-	<0.0001	*	*
I	-	-	-	-
II	2.867 (1.538-5.343)	0.001	-	-
III	6.949 (3.727-12.958)	<0.0001	-	-
IV	11.340 (5.540-23.213)	0.011	-	-
Arterial hypertension	0.742 (0.589-0.934)	0.011	*	*
Ischemic etiology	1.672 (1.321-2.115)	<0.0001	*	*
Diabetes mellitus	0.972 (0.772-1.223)	0.80	-	-
Dyslipidemia	0.911 (0.680-1.220)	0.53	-	-
Smoking history	1.382 (1.118-1.708)	0.003	*	*
COPD	1.027 (0.678-1.556)	0.89	-	-
Renal disease	0.997 (0.560-1.773)	0.99	-	-
Chronic liver disease	3.374 (0.470-24.217)	0.22	-	-
Atrial fibrilation	1.265 (0.974-1.643)	0.078	-	-
CRT	-	-	-	-
Family history	0.947 (0.674-1.332)	0.75	-	-
ACEi or ARB	1.036 (0.775-1.384)	0.81	-	-
ARNI	0.594 (0.386-0.914)	0.018		
Beta-blocker	0.266 (0.126-0.564)	0.001	0.493 (0.279-0.872)	<0.0001
Aldosterone antagonist	0.757 (0.517-1.108)	0.15	-	-
SGLT2-i	0.470 (0.327-0.677)	<0.0001	-	-
Loop diuretic	1.935 (1.406-2.661)	<0.0001	7.291 (2.050-25.924)	0.02

Table 9. Univariate/multivariate analysis of patients without ICD implantation-2

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p	HR (95% CI)	p
Anticoagulant	1.072 (0.852-1.350)	0.55	-	-
Antiplatelet	0.968 (0.785-1.194)	0.76	-	-
If channel blocker	0.818 (0.649-1.031)	0.08	*	*
Digoxine	0.985 (0.749-1.294)	0.91	-	-
Antiarrhytmic	1.148 (0.602-2.189)	0.67	-	-
Antihyperlipidemic	0.580 (0.455-0.740)	<0.0001	*	*
LVEDd, mm	1.032 (1.020-1.043)	<0.0001	*	*
LVESd, mm	1.036 (1.026-1.047)	<0.0001	-	-
LAd, mm	1.044 (1.033-1.055)	<0.0001	*	*
LVEF, %	0.883 (0.864-0.902)	<0.0001	*	*
Mitral regurgitation	-	<0.0001	*	*
Mild	0.839(0.491-1.432)	0.52	-	-
Moderate	1.716 (1.067-2.758)	0.026	-	-
Severe	2.772 (1.708-4.498)	<0.0001	-	-
Tricuspide regurgitation	-	<0.0001	*	*
Mild	1.993 (1.265-3.139)	0.002	-	-
Moderate	4.381 (2.844-6.749)	<0.0001	-	-
Severe	6.151 (3.911-9.672)	<0.0001	-	-
Aort regurgitation	-	0.12	-	-
Mild	1.129 (0.862-1.479)	0.37	-	-
Moderate	1.644 (1.073-2.520)	0.022	-	-
Severe	1.326 (0.329-5.334)	0.69	-	-
TRV, m/sec	1.781 (1.456-2.178)	<0.0001	-	-
TDI Rvsm	0.792 (0.758-0.828)	<0.0001	-	-
TAPSE, mm	0.889 (0.869-.910)	<0.0001	0.910 (0.849-.974)	0.007
SPAP, mmHg	1.028 (1.022-1.034)	<0.0001	*	*
NT-pro-BNP (log)	3.823 (3.146-4.646)	<0.0001	3.367 (1.814-6.251)	<0.0001

Heart Failure

OP-081

The definition of sarcomeric and non-sarcomeric gene mutations in hypertrophic cardiomyopathy patients: A multicenter diagnostic study across Turkey

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Background and Aim: Hypertrophic cardiomyopathy (HCM) is the most common hereditary cardiomyopathy with a marked clinical heterogeneity that may range from an asymptomatic course to the development of arrhythmias, heart failure and sudden cardiac death. HCM is considered a predominantly monogenic disease, while due to extreme heterogeneity, the disease-causing genes remain unknown in nearly 25-40% of cases. In up to 60% of adults with HCM, mutations in cardiac sarcomere protein genes are responsible for the disease, while in 5-10% of cases, mutations in non-sarcomere genes leads to HCM associated with neuromuscular disease, metabolic disorders, or genetic syndromes. Next generation sequencing (NGS)-based genetic diagnosis study aimed to identify sarcomeric and non-sarcomeric gene mutations and to confirm the final molecular diagnosis in patients diagnosed with HCM.

Methods: A total of 392 patients with HCM were included in this multicenter NGS diagnostic study conducted between January 2022 and June 2022 at 23 centers across Türkiye. Patients with left ventricule (LV) wall thickness ≥15 mm in one or more LV myocardial segments, as measured by any imaging technique. Patients with LVH due to severe hypertension or aortic stenosis, or those with confirmed etiology of HCM were excluded from this study. HCM panel containing 17 genes *(ACTC1, DES, FLNC, GLA, LAMP2, MYBPC3, MYH7, MYL2, MYL3, PLN, PRKAG2, PTPN11, TNNC1, TNNI3, TNNT2, TPM1, TTR)* was investigated by NGS-based genetic diagnostic study. The classification of pathogenicity of variants was based on the American College of Medical Genetics and Genomics (ACMG).

Results: The positive genetic variants (VUS, likely pathogenic or pathogenic) were identified in 121(30.9%) of 392 samples, including 119 (30.4%) samples positive for HCM variants or HCM phenocopies (including 1 sample positive for TTR-related amyloidosis) and 2 samples with GLA variants. Final molecular diagnosis was established in 69 (57.0%) of 121 samples with positive variants, corresponding to 17.6% of total 392 samples analyzed. The diagnostic yield was 17.1% (15.8% for HCM variants) for HCM and HCM phenocopies, and was 0.5% for Fabry disease. Specifically, among 121 samples, 133 variants (94 were different and 43 were novel variants) were identified in 12 genes including *MYBPC3, MYH7, FLNC, MYL3, TNNI3, DES, LAMP2, GLA, TPM1, TTR, TNNT2 and PRKAG2* genes. Likely pathogenic or pathogenic variants were found in 69 (57.0%) of 121 samples with variants (17.6% of all 392 cases), leading to a confirmed diagnosis. These 69 samples with likely pathogenic or pathogenic variants involved MYBPC3 (n=30), MYH7 (n=28), DES (n=4), GLA (n=2), TPM1 (n=2), LAMP2 (n=2), TNNT2 (n=1), and TNNI3 (n=1) genes.

Conclusions: Our study showed that the distribution of genetic mutations, the prevalence of Fabry disease and TTR amyloidosis in the Turkish population diagnosed with HCM was similar to the other populations but the percentage of sarcomeric gene mutations was slightly lower.

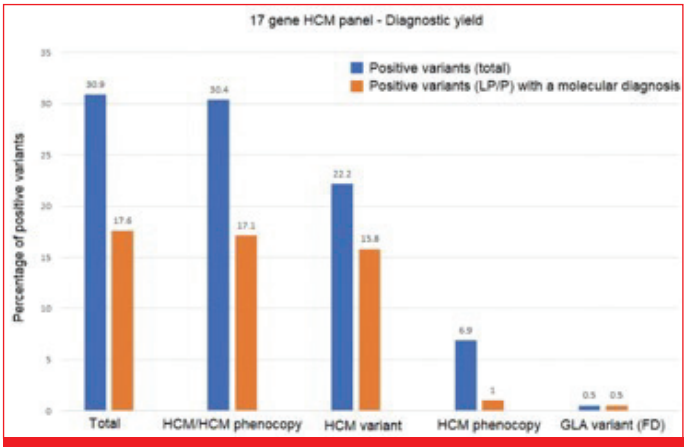

Figure 1. 17 Gene HCM Panel - Diagnostic Yield

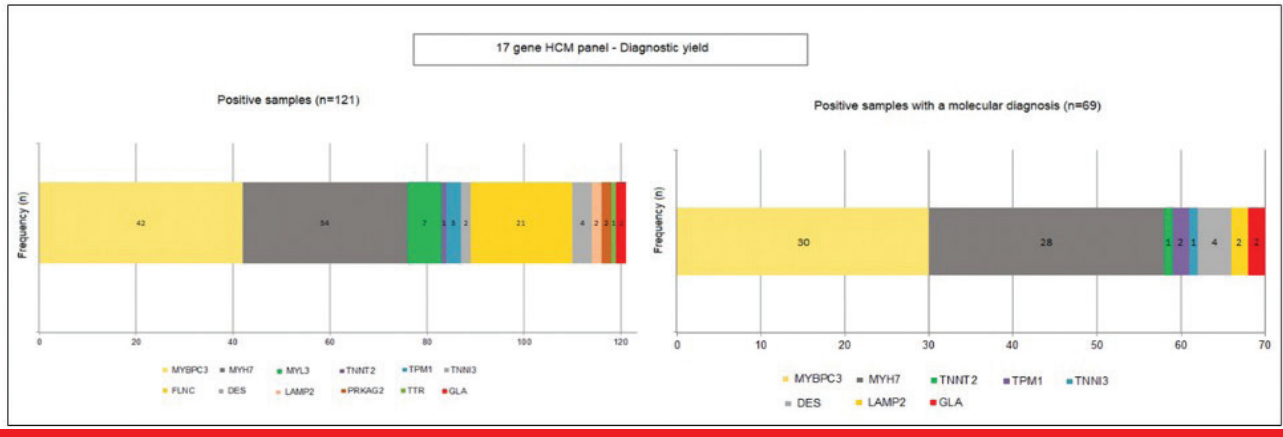

Figure 2. 17 Gene HCM Panel - Diagnostic Yield

Healt Failure

OP-082

Dapagliflozin may protect against the cardiotoxic effects of 5-fluorouracil

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Background and Aim: 5-fluorouracil (5-FU), an antimetabolite chemotherapy drug, is used in the treatment of many various cancers and it can cause potential toxicities such as cardiac ischemia, arrhythmia, vasospasm, and direct myocardial damage. It is known that the incidence of cardiac side effects due to 5-FU is 3-18%. In experimental studies, it has been understood that it causes coronary vasospasm by directly affecting the release of nitric oxide from the endothelium, and that it exerts its cardiotoxic effects by causing vasoconstriction independently of the endothelium via protein kinase C. Also there are alternative theories claiming a direct thrombogenic effect, oxidative damage or an autoimmune event. Dapagliflozin (DAPA) has shown protective effects on cardiovascular diseases. The DAPA-HF study demonstrated that DAPA reduced primary combined outcomes, including cardiovascular mortality and heart failure. DAPA shows anti-oxidative properties by reducing cytosolic and mitochondrial free oxygen radical production and changing Ca2+ dynamics, thus exerting protective effects against cell damage under oxidative stress conditions. Theoretically, DAPA has the potential to inhibit 5-FU induced AIC. This study aimed to compare the possible cardioprotective effects of DAPA on cardiomyopathy in rats.

Methods: A total of 32 male Wistar albino rats were divided into 4 groups consisting of 8 each (control: 8, DAPA: 8, 5-FU: 8, 5-FU + DAPA: 8). Mean while, 5-FU and 5-FU + DAPA groups received 150 mg/kg 5-FU at the beginning of the study intraperitoneally, DAPA and 5-FU + DAPA groups were gavaged daily with 10 mg/kg DAPA. At the begining and at the second week of the study, rats were examined by echocardiography and electrocardiogram. End of the study histopathological method was used to evaluate the level of cardiotoxicity (Figure 1).

Results: ST elevation was observed in 75% of the 5-FU group and 25% of the 5-FU + DAPA group (p=0.031). Ejection fraction decreased by 21% in the 5-FU group, %7 in 5-FU + DAPA group (p<0.001). Heart rate increased 10% in 5-FU and 2% in 5-FU + DAPA (p<0.001). QRS duration increased 46.6% in 5-FU and 7% in 5-FU + DAPA (p<0.001). QTc increased by 13.7% in 5-FU, while increased by 2.9% in 5-FU + DAPA (p<0.001) (Table 1, Figure 2-4). Histopathological results showed that the cardiac tissue histology was normal in control (Figure 1A) and DAPA groups (Figure 1B). On the other hand, the 5-FU group showed high levels of cardiac intoxication and severe histopathological findings, including hyperemia, necrosis, inflammatory cell infiltration, and hyaline formation compared with the control group (Figure 1c) (p<0.001 for all findings). A significant decrease was observed in the 5-FU + DAPA group in histopathological findings compared to the 5-FU group (Figure 1D) (p<0.001 for all findings) (Table 2, Figure 5,6).

Conclusions: Our study showed that dapagliflozin has the potential to reduce the effects of 5-FU induced cardiotoxicity.

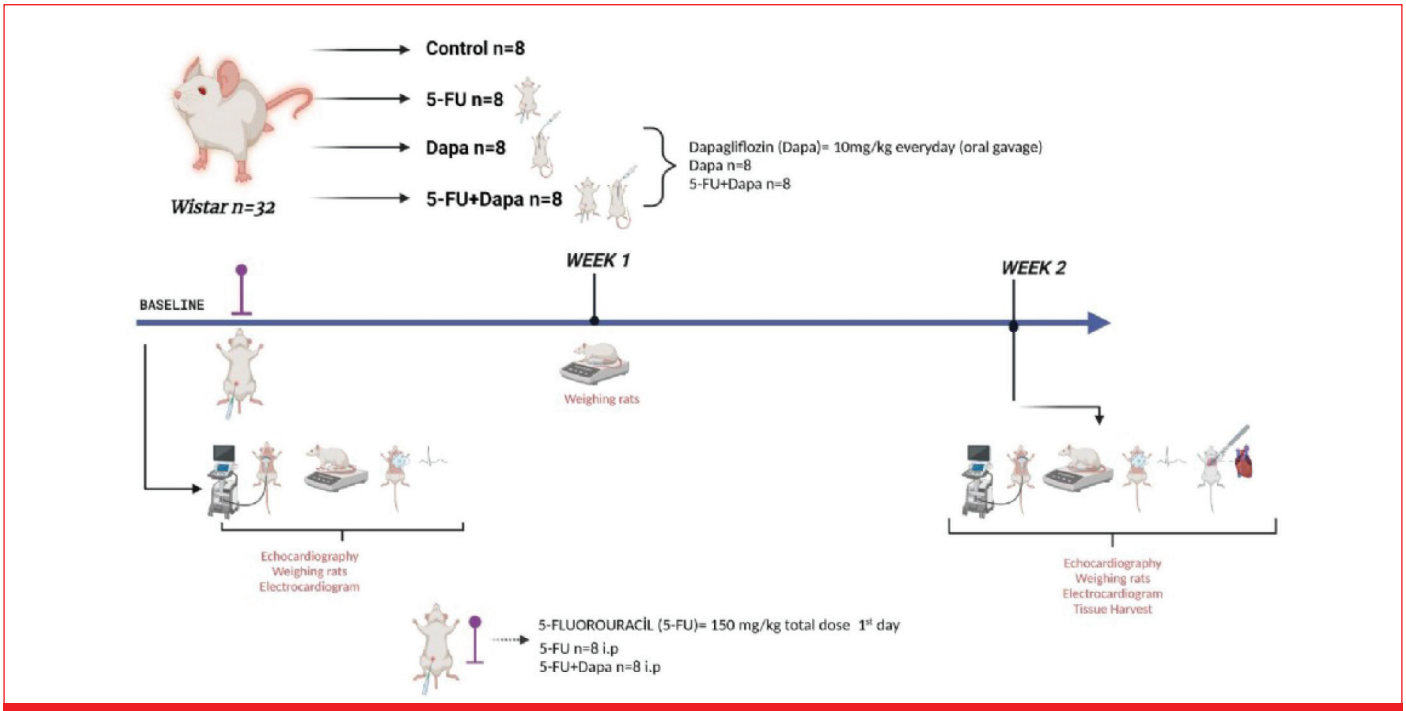


Figure 1. Study design

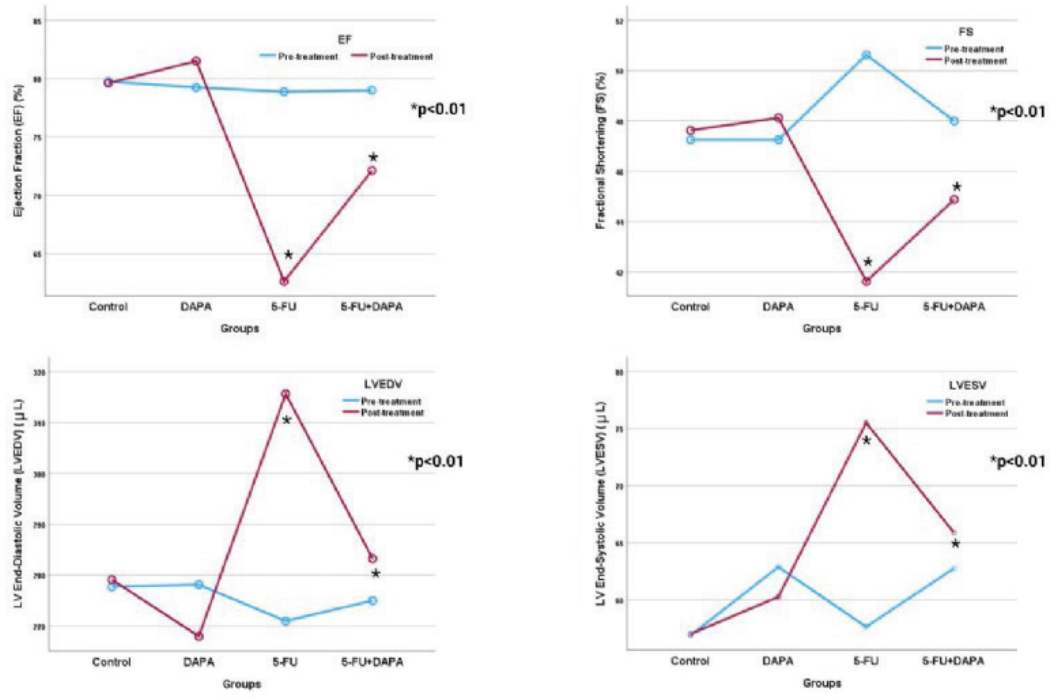


Figure 2. Echocardiographic findings of study groups

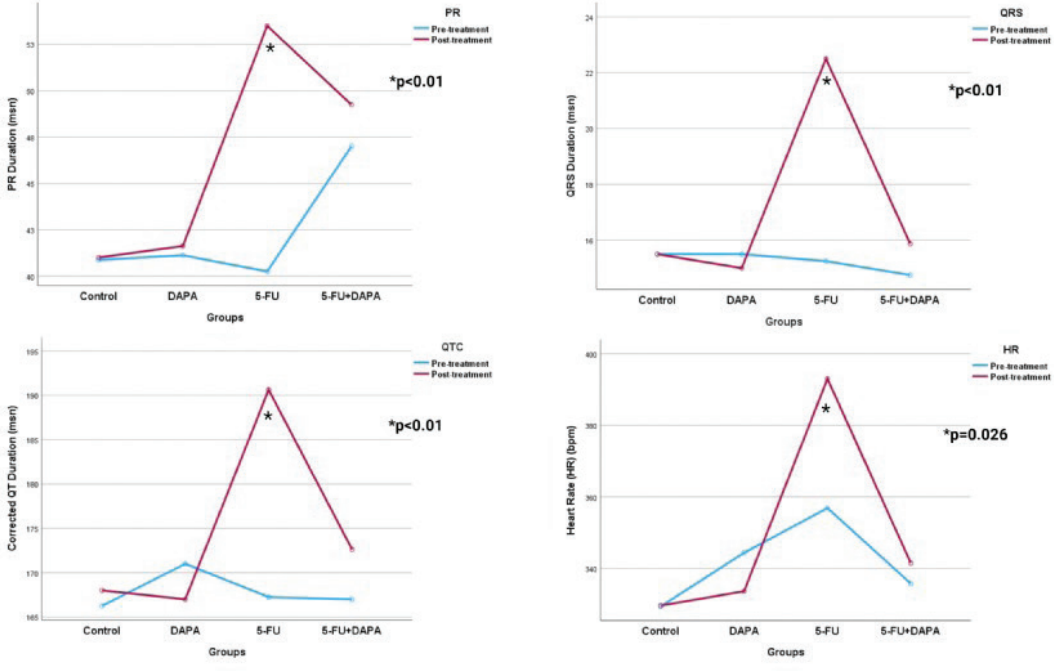
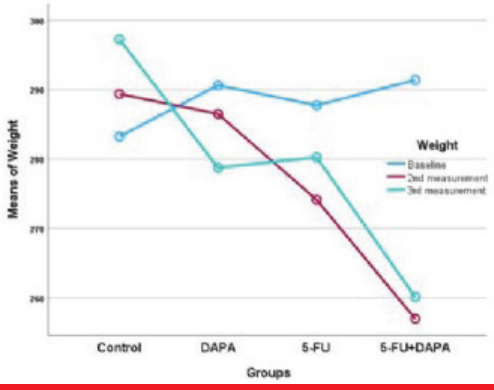


Figure 3. ECG findings of study groups



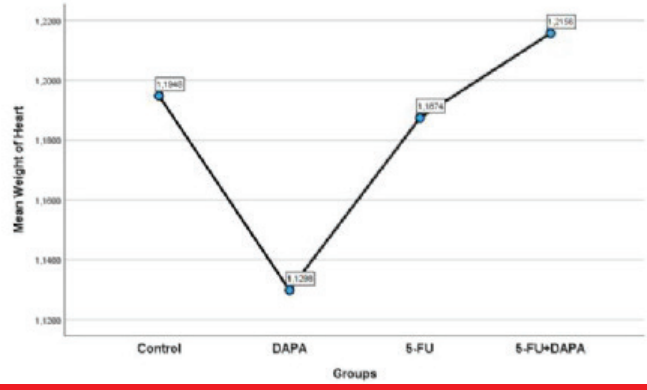


Figure 4. Weight changes of the study groups and heart weights at the end of the study

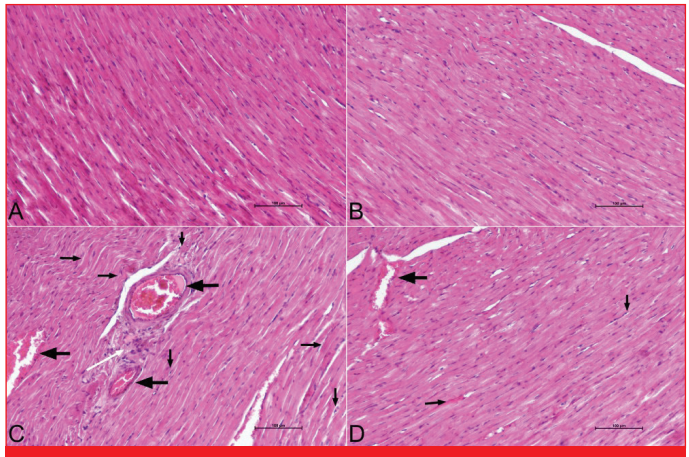


Figure 5. The hematoxylin and eosin staining results showed that the cardiac tissue histology was normal in control (Figure 1A) and DAPA groups (Figure 1B). On the other hand, the 5-FU group showed high levels of cardiac intoxication and severe histopathological findings, including hyperemia, necrosis, inflammatory cell infiltration, and hyaline formation compared with the control group (Figure 1C) (p<0.001 for all findings). A significant decrease was observed in the 5-FU + DAPA group in histopathological findings compared to the 5-FU group (Figure 1D) (p<0.001 for all findings).

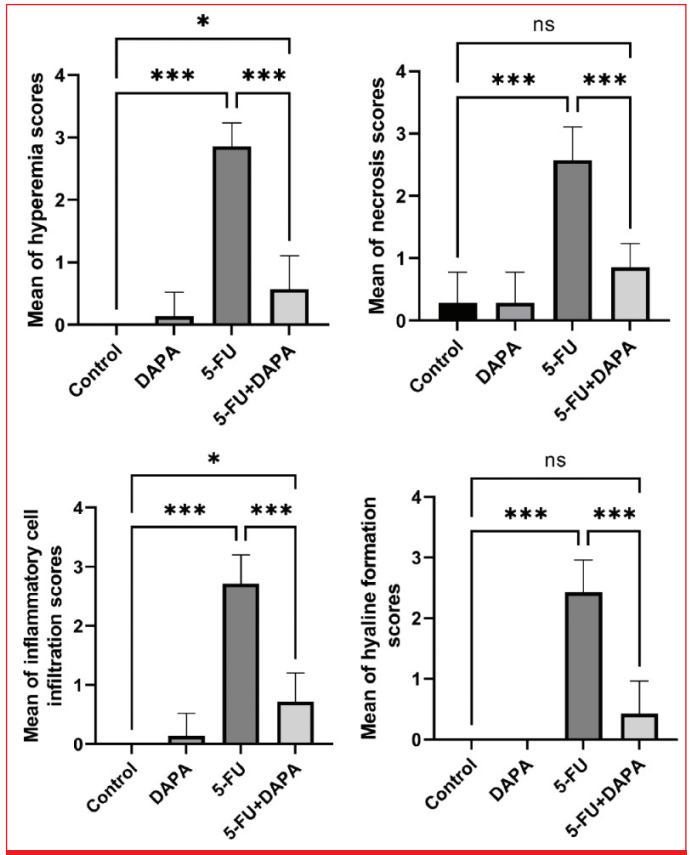


Figure 6.

Table 1. Cardiac and electrocardiography measurements of rats according to study groups

	Control	DAPA	5-FU	5-FU + DAPA	p groups	p measures
First weight measurement, g	283.25 ± 28.71	290.62 ± 26.46	287.75 ± 36.84	291.37 ± 26.02	0.521	<0.001* 1-2 1-3
Second weight measurement, g	289.37 ± 46.94	286.50 ± 24.16	274.12 ± 26.43	257.00 ± 23.82		
Third weight measurement, g	292.25 ± 40.57	278.75 ± 22.44	280.25 ± 23.08	260.12 ± 17.06		
Ejection fraction, baseline, %	79.75 ± 1.03	79.25 ± 1.83	78.87 ± 2.35	79.00 ± 3.25	<0.001* a-c a-d b-d	<0.001*
Ejection fraction, week 2, %	79.62 ± 1.59	81.50 ± 2.92	62.62 ± 6.32	72.12 ± 6.08		
Fractional shortening baseline, %	47.25 ± 3.80	47.25 ± 5.23	50.62 ± 4.62	48.00 ± 4.30	0.848	<0.001*
Fractional shortening week 2, %	47.62 ± 3.85	48.12 ± 5.51	41.62 ± 4.47	44.87 ± 3.44		
End-diastolic volume baseline, µL	277.75 ± 12.41	278.12 ± 10.99	271.00 ± 13.26	275.00 ± 15.00	0.083	0.001
End-diastolic volume week 2, µL	279.12 ± 11.93	268.00 ± 14.24	315.62 ± 35.28	283.25 ± 15.43		
End-systolic volume baseline, µL	56.87 ± 10.13	62.87 ± 4.83	57.62 ± 7.26	62.75 ± 8.53	0.132	<0.001*
End-systolic volume week 2, µL	57.00 ±10.40	60.25 ± 6.04	75.50 ± 11.16	65.87 ± 8.79		
Heart rate baseline, bpm	329.37 ± 23.76	344.37 ± 27.51	358.87 ± 12.95	337.75 ± 20.80	0.002* a-c b-c d-c	0.026*
Heart rate week 2, bpm	329.62 ± 25.25	336.62 ± 23.37	393.00 ± 34.94	341.50 ± 22.97		
QTc baseline, ms	166.25 ± 18.52	171.00 ± 16.54	167.25 ± 15.82	167.00 ± 13.00	0.849	<0.001*
QTc week 2, ms	168.00 ± 18.05	167.00 ± 15.47	190.62 ± 21.36	172.62 ± 14.12		
PR baseline, ms	40.87 ± 5.16	41.12 ± 5.22	40.25 ± 3.19	47.00 ± 5.37	0.007* a-d b-d	<0.001*
PR week 2, ms	41.00 ± 5.01	41.62 ± 4.59	53.50 ± 6.23	49.25 ± 5.49		
QRS baseline, ms	15.50 ± 1.77	15.50 ± 1.60	15.25 ± 1.98	14.75 ± 1.28	0.001* a-c b-c d-c	<0.001*
QRS week 2, ms	15.52 ± 1.81	15.00 ± 1.30	22.50 ± 3.81	15.87 ± 1.26		
Heart weight, g	1.19 ± 0.17	1.12 ± 0.11	1.18 ± 0.10	1.21 ± 0.15	0.643	
ST elevation, N (%)	0	0	6 (75.0)	2 (25.0)	0.031**	

* Significant at 0.05 level according to Two-way ANOVA, ** Significant at 0.05 level according to Chi-square test. a: Control, b: DAPA, c: 5-FU, d: 5-FU + DAPA groups.

Table 2. Histopathological findings of the study groups

Parameter	Control	DAPA	5-FU	5-FU + DAPA
Hyperemia	0 ± 0	0.142 ± 0.378	2.857 ± 0.378^{c}	0.571 ± 0.534^{a,f}
Necrosis	0.285 ± 0.488	0.285 ± 0.488	2.571 ± 0.534^{c}	0.857 ± 0.378^{f}
Inflammatory cell infiltration	0 ± 0	0.142 ± 0.378	2.714 ± 0.488^{c}	0.714 ± 0.488^{a,f}
Hyaline formation	0 ± 0	0 ± 0	2.429 ± 0.534^{c}	0.428 ± 0.534^{f}

Values are expressed as mean ± SD. Abbreviations: DAPA: Dapagliflozin, 5-FU: 5-fluorouracil. a, b, c: Statistically significant differences between control and other groups (a: p<0.05, b: p<0.01, and c: p<0.001). d, e, f: Statistically significant differences between 5-FU and other groups (d: p<0.05, e: p<0.01, and f: p<0.001).

Coronary Artery Disease / Acute Coronary Syndrome

OP-083

Bendopnea predicts high SYNTAX score in patients with coronary artery disease: A Bayesian approach

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Background and Aim: Coronary artery disease (CAD) is one of the major health problems in worldwide. CAD severity, which could be calculated by SYNTAX score (SS), is associated with higher morbidity and mortality. Predicting high SS is prominent for detecting high-risk patients. A new symptom of shortness of breath when bending forward is described as bendopnea and was related to elevated cardiac filling pressure. It is also known that a high SYNTAX score is associated with left ventricular (LV) systolic and diastolic dysfunction which results in a higher LV filling pressure. Thus, we aimed to investigate whether there was an association between bendopnea and high SYNTAX scores in CAD patients using the Bayesian approach in this study.

Methods: A total of 374 CAD patients who had coronary artery stenosis of >50% were enrolled in this prospective study. A high SYNTAX score was defined as having a SYNTAX score >22. Each patient was asked to lean forward at the waist as if wearing shoes while seated and the presence of bendopnea was accepted as occuring shortness of breath within 30 seconds. The association of variables with high SYNTAX scores was quantified using Bayesian logistic regression analysis. Posterior probabilities was calculated using the Markov chain Monte Carlo method with 4 chains and the median posterior odds ratio (OR) and credible intervals (CrI) were expressed. The robustness of the results was tested by calculating the heterogeneity of meta-analysis using different priors (neutral, optimistic, and skeptical).

Results: There were 238 (64%) patients with bendopnea and 136 (36%) patients without bendopnea in this study (Table 1). Bendopnea (+) group had higher SYNTAX score and gensini scores compared to bendopnea (-) group. The presence of bendopnea was independently associated with a high SYNTAX score (OR: 3.82, 95% CrI: 1.93-8.17) (Table 2, Figure 1). At the mean tau value, approximately 23% of the heterogeneity could be defined by the priors, which implies that our results were quite robust to the different priors (Figure 2).

Conclusions: Bendopnea was independently associated with CAD severity and might be used for risk stratifying in those patients.

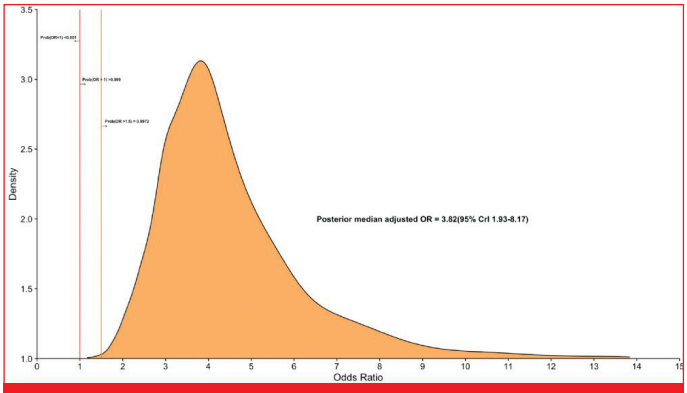

Figure 1. Posterior probability distribution of odds ratio of bendopnea for predicting high syntax score.

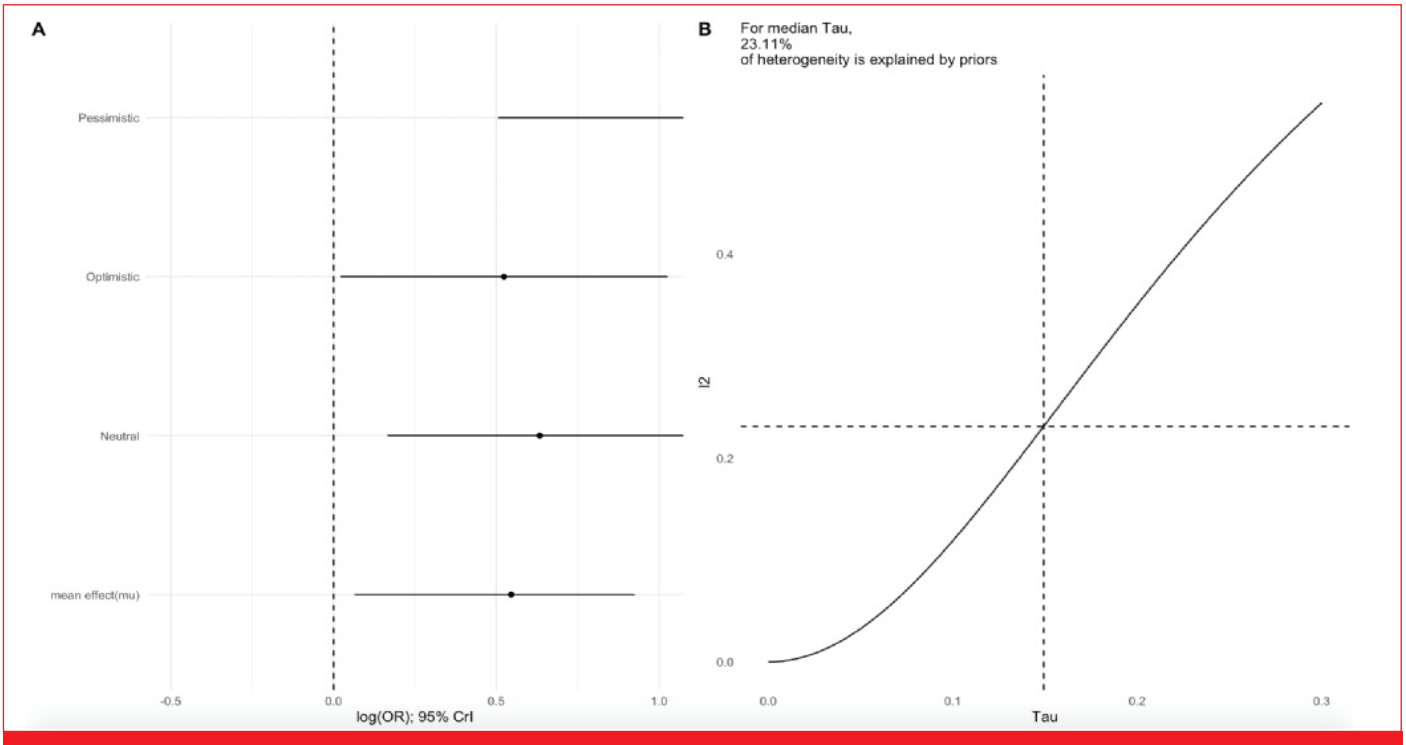

Figure 2. Forest plot of each priors (A) and heterogeneity for all tau distribution (B).

Table 1. Baseline demographic, laboratory, and echocardiographic features of study groups

Variables	Bendopnea (-) (n=136)	Bendopnea (+) (n=238)	Posterior probability of difference from 0
Age, years	52.7 (13.0)	55.7 (15.2)	0.950
Male gender, n (%)	81 (59.6)	119 (50.0)	0.961
Body mass index, kg/m^2	27.4 (3.10)	27.6 (3.33)	0.761
Cigarette smoking, n (%)	82 (60.3)	159 (66.8)	0.890
Diabetes mellitus, n (%)	53 (39.0)	98 (41.2)	0.674
Chronic obstructive pulmonary disease, n (%)	50 (36.8)	60 (25.2)	0.991
Hypertension, n (%)	62 (45.6)	113 (47.5)	0.639
6-minutes walking test, meters	246 (187)	177 (149)	>0.999
White blood cell, x10^3/µL	8.44 (3.06)	8.74 (2.99)	0.818
Hemoglobin, g/dL	14.2 (4.14)	14.3 (3.44)	0.605
Sodium, mmol/L	134 (26.4)	135 (21.9)	0.791
Albumin, g/dL	3.99 (1.56)	3.52 (1.71)	0.994
Triglycerides, mg/dL	270 (209)	226 (179)	0.999
LDL-cholesterol, mg/dL	101 (75.8)	96.6 (60.2)	0.994
Heart rate, beats/min	75.2 (16.9)	74.3 (14.1)	0.819
Left ventricular ejection fraction, %	59.3 (6.46)	57.3 (5.97)	>0.999
Interventricular septum, mm	11.2 (1.99)	12.4 (1.67)	>0.999
LV end-diastolic diameter, mm	52.4 (6.72)	52.3 (6.72)	0.615
LV end-systolic diameter, mm	33.5 (6.92)	31.7 (6.09)	0.995
Mitral E, m/s	1.24 (1.04)	0.92 (0.90)	0.999
Mitral A, m/s	0.79 (0.18)	0.80 (0.18)	0.650
TDI lateral e, cm/sn	10.4 (3.49)	10.1 (3.55)	0.786
TDI septal e, cm/sn	7.93 (2.43)	8.34 (2.74)	0.923
Pulmonary artery systolic pressure (mmHg)	30.4 (5.1)	28.5 (4.2)	>0.999
Deceleration time, ms	181 (44.2)	174 (43.5)	>0.999
Isovolumetric relaxation time, ms	90.4 (21.8)	102 (19.0)	>0.999
Gensini score	31.2 (32.3)	41.3 (27.3)	0.995
SYNTAX score	9.49 (9.24)	13.7 (8.98)	>0.999
High SYNTAX group, n (%)	12 (8.82)	52 (21.8)	0.999

Table 2. Bayesian logistic regression analysis for predicting high syntax score

Variables	Odds ratio	95 % CrI	Posterior probability (OR >1)
Age	1.000	0.980-1020	0.336
Gender	1.877	1.000-3.743	0.974
Hypertension	1.061	0.577-1.954	0.581
Diabetes mellitus	3.743	2.013-7.099	>0.999
Cigarette smoking	1.116	0.606-2.075	0.639
Chronic obstructive pulmonary disease	2.745	1.390-5.473	0.999
Bendopnea	3.819	1.934-8.166	>0.999

Coronary Artery Disease / Acute Coronary Syndrome

OP-084

The nutritional and inflammatory predictors of permanent atrioventricular block in ST segment elevation myocardial infarction

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Background and Aim: High-grade atrioventricular (AV) block (HG-AVB) (complete AV block and Mobitz-type II block) is a common complication of acute myocardial infarction (AMI). The recent studies have showed that there is an increased risk for HG-AVB in patients with inferior wall myocardial infarction, age, prior myocardial infarction, sex, smoking, diabetes mellitus, hypertension and ST-segment elevation myocardial infarction (STEMI). Although HG-AVB is reversible in many patients, it persists in approximately 10% of patients and requires a permanent pacemaker. Inflammation has a pivotal role in the pathogenesis of atherosclerosis and AMI. The evidence supports a central role for both local and systemic inflammation in process of the AMI by destabilizing and rupturing atherosclerotic plaque. In addition, lower albumin level is considered as a risk factor for the coronary artery disease and AMI. The recent studies have showed that inflammation and nutritional statuses are related to a variety of indicators which might be predicted the prognosis of the STEMI. The Naples prognosis score (NPS) is an effective immune and nutritional scoring system based on albumin, cholesterol level, LMR and NLR. NPS was established as a prognostic indicator for long-term outcome in patients with certain cancers, pulmonary embolism and AMI. This study was conducted to evaluate the prognostic role of the NPS in patients with HG-AVB in STEMI patients.

Methods: In this study, 233 STEMI patients with HG-AVB on admission or within the first 24 hours were retrospectively reviewed. The patients were divided into two groups as temporary or permanent AV block. We compared clinical and laboratory parameters of both groups. The NPS was calculated based on serum albumin and total cholesterol concentrations, neutrophil/lymphocyte, and lymphocyte/monocyte ratio (Figure 1).

Results: A total of 233 STEMI patients in HG-AVB were included in the study. The patients were divided into two groups according to whether the AV block was permanent or not [temporary AV block (t-AVblock), permanent AV block (p-AVblock)]. Table 3 shows the demographic, clinical and laboratory parameters of the two groups. Patients with p-AVblock group were older, compared to t-AVblock group and DM and smoking were more common in p-AVblock group. P-AVblock group had a significantly higher prevalence of anterior MI and Killip class ≥3. Total ischaemic time was higher in with p-AVblock group while heart rate, systolic and diastolic blood pressure and EF had lower. In addition, BNP, troponin and NPS levels were higher in p-AVblock group parameters found statistically significant in predicting p-AVBlock in univariate analysis were studied by regression analysis. Age, smoking, BNP, troponin, anterior MI and NPS were found to be independent predictors of p-AVBlock (Table 2).

Conclusions: HG-AVB is a well-recognized complication of AMI and it is clinically important to decide whether it was temporary or permanent. The NPS may help assess the optimal waiting period before permanent pacemaker implantation in patients with HDAVB following STEMI and further studies are needed in this context.

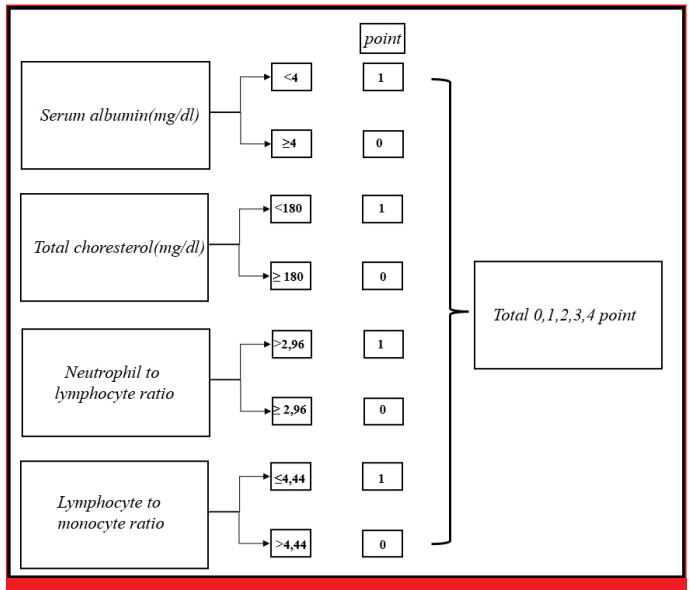

Figure 1. Formulation of NAPLES prognostic score

Table 1. Baseline demographic parameters of two groups

Variables	t-AV Block Group (n: 20)	p-AV Block Group (n: 213)	P value
Age	**67±9**	**57±11**	**<0,001**
Female Sex n (%)	10(50)	88(41)	NS
DM n (%)	**8(40)**	**40(19)**	**0,025**
Ht n (%)	8(40)	87(40)	NS
Dislipidemia n (%)	8(40)	75(35)	NS
Smoking n (%)	**8(40)**	**38(17)**	**0,017**
PCI/CABG History	6(30)	36(16)	0,06
Family History n (%)	7(35)	55(25)	NS
HR, beat/min	**33±5**	**37±5**	**0,002**
SBP, mmHg;	**104±28**	**120±34**	**0,049**
DBP, mmHg	**53±16**	**63±19**	**0,023**
Drugs			
Beta Blocker n (%)	2(10)	35(16)	NS
Statin n (%)	13(65)	36(16)	NS
ACEIs or ARBs n (%)	3(15)	35(16)	NS
ASA n (%)	7(35)	48(22)	NS
P2Y12 rec. inh. n (%)	3(15)	16(7)	NS
CCB n (%)	1(5)	12(5)	NS

Table 2. Clinical and laboratory parameters of two groups

Variables	t-AV Block Group (n: 20)	p-AV Block Group (n: 213)	P value
Hemoglobin, g/dl;	12,9±2,2	13,1±2,1	NS
WBC,10^3 /L	13,3±47	13,1±4,4	NS
Platelet count,103 /µL	274±69	260±78	NS
Lymphocyte, 103 /µL	1,5±0,7	1,8±1,1	NS
Monocytes, 103 /µL	0,58±0,27	0,68±0,4	NS
Neutrophils, 103 /µL	10,6±4,5	10,3±4,3	NS
CRP, mg/l	16,4±1,3	15,4±1,2	NS
Albumin, g/dl	3,42±0,39	3,57±0,54	NS
Total chol, mg/dL	166±23	176±44	NS
Triglycerides, mg/dl	132±60	145±65	NS
HDL-C, mg/dl	39±12	37±12	NS
LDL-C, mg/dl	104±28	113±39	NS
Urea, mg/dl	42±20	44±27	NS
Creatinine, mg/dl	1,13±0,9	0,89±0,61	NS
Glucose, mg/dl;	225±147	173±112	0,057
Uric acid , mg/dl;	6,4±1,9	6,1±2,1	NS
BNP level	**262±36**	**120±37**	**<0,001**
Peak Troponin Level	**332±286**	**135±113**	**<0,001**
Total ischemia time	**258±157**	**199±111**	**0,03**
Killip 3,4; n (%)	**13(65)**	**65(30)**	**<0,001**
Anterior MI n (%)	**13(65)**	**59(27)**	**<0,001**
LVEF %	**41±8**	**47±8,3**	**0,002**
NAPLES score	**3,75±0,55**	**2,67±096**	**0,001**

Table 3.

Variables	Univariate p	Multivariate p	OR (95% CI)
Age	<0.001	0.022	0.85 (0.74-0.97)
DM	0.025	0.960	0.94 (0.08-11.5)
Smoking	0.017	0.023	37.4 (1.6-855.6)
SBP	0.049	0.6	1 (0.95-1.1)
DBP	0.023	0.87	1.01 (0.9-1.2)
HR	0.002	0.132	1.26 (0.93-1.7)
BNP	<0.001	0.022	0.99 (0.97-0.99)
Peak troponin	<0.001	0.016	0.99 (0.98-0.99)
Total ischemia time	0.03	0.720	98 (0.97-1.1)
Killip 3,4 at adm	<0.001	0.056	15.8 (0.93-270.6)
Anterior MI	<0.001	0.008	925 (5.9-144.3)
LVEF %	0.002	0.32	1.08 (0.92-1.27)
NAPLES score	0.001	0.008	0.038 (0.3-0.43)

Independent predictors of permanent AV block in multivariate logistic regression analysis (age, smoking, BNP, troponin, anterior MI and NPS).

Coronary Artery Disease / Acute Coronary Syndrome

OP-085

Relationship between triglyceride-glucose index and coronary complexity in patients with acute coronary syndrome

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Background and Aim: The SYNTAX score is a well recognized scoring system to assess the complexity of coronary artery disease (CAD). Patients with high SYNTAX scores have been found to be at an elevated risk of major adverse cardiac events (MACE). Increasing evidence shown that insulin resistance plays a role in the pathogenesis of CAD. Recently, the triglyceride-glucose (TyG) index has emerged as a novel marker that reliably indicates insulin resistance. Studies have demonstrated that an elevated TyG index serves as an independent predictor for the progression of coronary artery calcification and augments the risk of CAD. Moreover, TyG index is associated with an unfavorable prognosis in patients with acute coronary syndrome (ACS). The aim of our study was to elucidate the relationship between the TyG index and the SYNTAX score in ACS patients.

Methods: This was an observational retrospective study conducted between September 2021 and May 2022. We analyzed a total of 122 patients who were admitted to our hospital with ACS and did not have diabetes mellitus (DM). TyG index was calculated using the formula ln [fasting triglyceride (mg/dL) x fasting glucose (mg/dL)/2]. The SYNTAX score was determined using an online calculation tool (http://syntaxscore.com/), with angiographic images evaluated by two cardiologists. The patients were divided into two groups: Group 1 with a low SYNTAX score (≤22) and Group 2 with a moderate-high SYNTAX score (>22). Intergroup variables were subsequently assessed.

Results: The main characteristics between Group 1 (n=78) and Group 2 (n=34) are summarized in Table 1. In the univariate analysis, a significant difference was observed in the prevalence of previous coronary artery disease (CAD), with 31% (n=24) in the low SYNTAX group and 53% (n=18) in the medium-high SYNTAX group (p=0.026). Regarding laboratory findings, statistically significant differences were found in baseline troponin levels (2423.1 ± 4615.3 ng/mL vs. 7994.1 ± 9987.4 ng/mL, p<0.001), creatinine levels (1 ± 0.6 mg/dL vs 1.4 ± 1.3 mg/dL, p=0.035), and CK-MB levels (17.2 ± 40.1 ng/mL vs. 54 ± 84.5 ng/mL, p=0.005) between the two groups. While fasting triglyceride levels (157.4 ± 88.4 mg/dL vs. 149 ± 180.6 mg/dL, p=0.736) and fasting blood glucose (105.9 ± 28 mg/dL vs. 106.7 ± 19.6 mg/dL, p=0.880) did not show significant variability between the groups, the TyG index was found to be significantly higher in the medium-high SYNTAX group (8.48 ± 1.01 vs. 9 ± 0.77, p=0.009). Furthermore, multivariate regression analysis revealed that the TyG index was an independent predictive factor for a high SYNTAX score (p=0.015), considering the significant parameters identified in the univariate analysis (Table 2).

Conclusions: A high TyG index is associated with a higher presence of coronary complexity (SYNTAX >22) in ACS patients without a diagnosis of DM. The TyG index has the potential to serve as a non-invasive predictor of coronary complexity in ACS patients, thus potentially influencing the therapeutic approach for these individuals.

Table 1. Basal characteristics of patients according to SYNTAX score

Parameters	GROUP-1(SYNTAX≤22) (n=78)	GROUP-2(SYNTAX>22) (n=34)	p-value
Demographic features			
Age	62.5 ± 12.4	66.2 ± 13.4	0.160
Sex (n%)			0.607
Female	22 (73.3)	8 (26.7)	
Male	56 (68.3)	26 (31.7)	
HT (n%)			0.975
No	25 (69.4)	11 (30.6)	
Yes	53 (69.7)	23 (30.3)	
CAD history (n%)			**0.026**
No	54 (77.1)	16 (22.9)	
Yes	24 (57.1)	18 (42.9)	
Smoking status (n%)			0.138
Non-Smoker	44 (75.9)	14 (24.1)	
Smoker	34 (63)	20 (37)	
Laboratory Features			
WBC (10^9/L)	9 ± 3.7	8.9 ± 4.1	0.915
Hemoglobin (mg/dL)	14.3 ± 2.3	13.4 ± 2.6	0.097
Platelet count (10^9/L)	251.7 ± 76.4	243 ± 70	0.574
CRP (mg/L)	15.7 ± 31.3	26.6 ± 38.8	0.133
Fasting blood glucose (mg/dL)	105.9 ± 28	106.7 ± 19.6	0.880
Creatinin (mg/dL)	1 ± 0.6	1.4 ± 1.3	0.035
Triglyceride (mg/dL)	157.4 ± 88.4	149 ± 180.6	0.736
HDL-c (mg/dL)	43.5 ± 31.1	37.1 ± 10.7	0.254
LDL-c (mg/dL)	129.4 ± 47	123.2 ± 45.3	0.525
Total cholesterol (mg/dL)	176.1 ± 45.4	174.3 ± 52.1	0.866
CK-MB (ng/ml)	17.2 ± 40.1	54 ± 84.5	0.005
hs-Troponin (ng/ml)	2423.1 ± 4615.3	7994.1 ± 9987.4	<0.001
TyG index	8.48 ± 1.01	9 ± 0.77	**0.009**
Angiographic Features			
LAD Lesion (n%)			**0.010**
No	46 (80.7)	11 (19.3)	
Yes	32 (58.2)	23 (41.8)	
LAD-Diagonal Lesion (n%)			0.432
No	74 (71.2)	30 (28.8)	
Yes	4 (57.1)	3 (42.9)	
CX lesion (n%)			**<0.001**
No	64 (83.1)	13 (16.9)	
Yes	14 (40)	21 (60)	
CX-OM lesion (n%)			0.418
No	73 (71.6)	29 (28.4)	
Yes	4 (57.1)	3 (42.9)	
RCA lesion (n%)			0.205
No	44 (74.6)	15 (25.4)	
Yes	33 (63.5)	19 (36.5)	

HT: Hypertension, CAD: Coronary artery disease, WBC: White blood cell, CRP: C-reaktif protein, LDL-c: low density lipoprotein cholesterol, HDL-c: High density lipoprotein cholesterol, CK-MB: Creatine kinase-myocardial band, hs-Troponin: High sensitive-troponin, TyG: Trigliserid-Glukoz index, LAD: Left anterior descending artery OM: Obtus marginalis Cx: Circumflex artery, RCA: Right coronary artery.

Table 2. Logistic regression analysis of the parameters predicting SYNTAX score

	OR	95 %CI	p value
Constant			0.004
CAD history	1.92	0.68 – 5.43	0.218
hs- Troponin	1	0.98 – 1.02	0.080
Creatinin	1.78	0.90 – 3.50	0.098
CK-MB	1.01	0.99 – 1.02	0.329
TyG index	2.30	1.18 – 4.51	**0.015**

OR: Odds ratio, CI:Confidence intervals, CAD: Coronary artery disease, hs-Troponin: High sensitive-troponin, CK-MB: Creatine kinase-myocardial band,, TyG: Triglyceride-Glucose index

Coronary Artery Disease / Acute Coronary Syndrome

OP-086

Impact of preloading strategy with ticagrelor on periprocedural myocardial injury in patients with non-ST elevation acute coronary syndromes undergoing early invasive strategy

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Background and Aim: Current guidelines endorsed by the European Society of Cardiology (ESC) do not recommend routine administration of P2Y12 receptor inhibitors in patients with non-ST-elevation acute coronary syndromes (NSTE-ACS) in whom coronary anatomy in unknown and an early invasive strategy (coronary angiography in 24 hours) is planned. However, absence of P2Y12 inhibitors in NSTEMI may potentially increase the risk of thrombotic complications such as periprocedural myocardial injury and stent thrombosis, both before and at the time of percutaneous coronary intervention (PCI). The primary aim of this study is to investigate impact of preloading strategy with ticagrelor on periprocedural myocardial injury in patients with non-ST elevation acute coronary syndromes undergoing early invasive strategy. Secondary aim is to investigate safety outcomes such as rate of bleeding related with preloading strategy.

Methods: NSTE-ACS patients who underwent coronary angiography and subsequent PCI in 24 hours following hospital admission were divided into two groups; the first group (pretreatment group) is composed of patients receiving ticagrelor pretreatment as soon as possible after admission and before PCI, and second groups (no pretreatment group) is composed of patients who received a loading dose of ticagrelor after coronary angiography. Pretreatment group encompassed 232 patients and no-pretreatment group encompassed 87 patients. Periprocedural myonecrosis defined by an increase of >5 times the 99th percentiles of the assay in troponin-negative patients before PCI or a 20% increase compared with pre-PCI value.

Results: The 2 groups were similar regarding baseline characteristics except higher prevalence of hypertension (p=0.014) and higher hemoglobin levels (p=0.01) in preloading group in comparison to no-preloading group. Patients in the ticagrelor pretreatment group had less myonecrosis before the coronary angiography based on troponin measurements sampled between 6th and 12th hour after admission (p=0.025). Patients in the ticagrelor pretreatment group also had less procedural myonecrosis based on troponin measurements sampled between 12th and 24th following the PCI (p=0.022) (Figure 1). The rates of bleeding according to Bleeding Academic Research Consortium at 1-month follow-up was similar between the 2 groups (p=0.938).

Conclusions: Our findings suggest that ticagrelor pretreatment reduces periprocedural myonecrosis in NSTE-ACS patients undergoing PCI in 24 hours following admission. Pretreatment with ticagrelor is not associated with higher bleeding events in comparison to loading at the time of PCI.

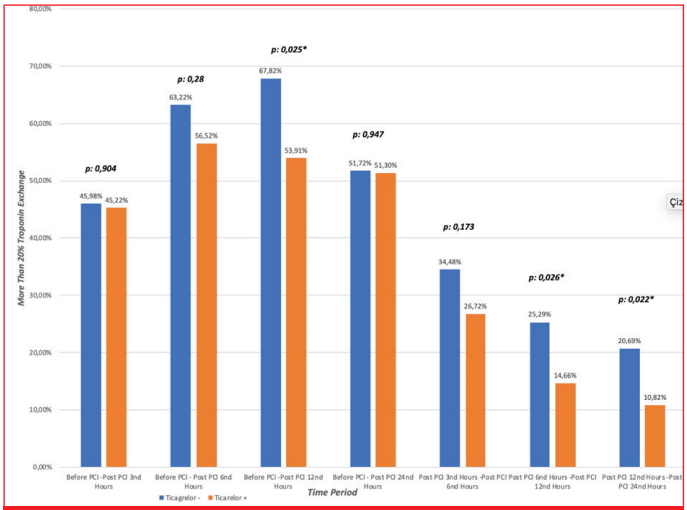

Figure 1. Comparison of the values of pre- and post-procedure troponin between the patient group in which patients in the ticagrelor pretreatment group and non-pretreatment group (*: p<0.05).

Results: Age and other demographic data were similiar in both groups. Although LA maximum volumes at the end-diastole were higher in the CSF group, no statistically significant difference was observed. (34.3 ± 16.7 vs. 33.5 ± 19.6, p=0.108). LA minimal volumes were similar in both groups (16.0 ± 4.9 vs. 14.8 ± 5.4, p=0.083). Volume at the beginning of atrial systole were higher in CSF group than control, however the difference was not statistically significant (16.0 ± 4.9 vs. 14.8 ± 5.4, p=0.083). A statistically significant difference was observed between LA ejection fractions in favor of the control group (53.35 ± 17.35 vs. 55.8 ± 15.11, p=0.03).

Conclusions: Evaluation of LA volume, functions and structure are mandatory for atrial arrhytmias and heart failure with preserved ejection fraction. It is known that atrial arrhythmias and heart failure symptoms are seen more frequently in patients with CSF phenomenon than in healthy individuals. Likewise, diastolic dysfunction was observed more frequently in the echocardiographic evaluation of this group of patients. Although there is no direct decrease in ejection fraction in CSF patients, it can affect atrial functions and make patients symptomatic. Since, it is difficult and challenging to provide an appropriate echocardiographic window for the evaluation of atrial ejection fraction in daily life, considering the effect of CSF on atrial functions is important for early and accurate treatment selection in symptomatic patients.

Epidemiology

OP-087

Echocardiographic evaluation of left atrial functions in coronary slow flow phenomenon

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Background and Aim: Coronary slow flow (CSF) phenomenon is very common during angiography and its adverse effects on both systolic and diastolic parameters of the left ventricle have been shown. In some of the echocardiographic studies conducted in recent years, it has been claimed that CSF is associated with impaired atrial functions and even leads to an increase in the frequency of atrial arrhythmias. In our study, we aimed to evaluate left atrial (LA) functions echocardiographically in CSF patients.

Methods: A total of 75 patients were included in the study. Thirty of the patients were excluded from the study due to the inconsistency in the echocardiography window. Only patients in whom LA could be evaluated in the appropriate echocardiographic window were included in the study (Figure 1). LA volume and ejection fraction (EF) were evaluated according to the Kdynes formula. LA ejection fraction was calculated as: (Vmax-Vmin)/Vmax x 100.

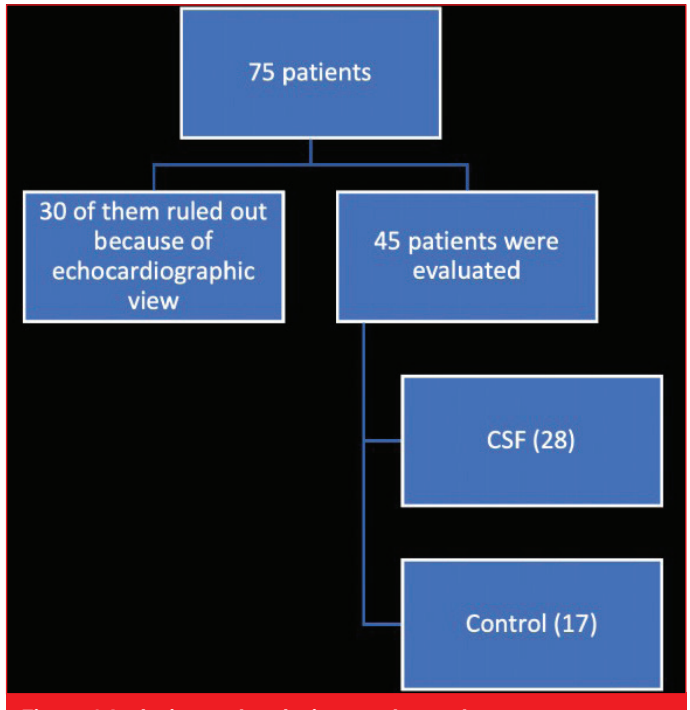

Figure 1. Inclusion and exclusions to the study.

Table 1. Demographics and echocardiographic findings

Variables	CSF (n=28)	Control group (n=17)	p
Age, years	51.14 ± 8.20	49.16 ± 7.72	0.110
Gender (male), % (n)	71.4% (20)	70.5% (12)	0.600
BMI, kg/m^2	24.10 ± 4.06	25.06 ± 4.51	0.456
DM, n (%)	14 (50)	11 (64.7)	0.080
HT, n (%)	15 (53.5)	9 (52.9)	0.332
LVES, mm	28 (25-28)	28.38 ± 3.29	0.602
LVED, mm	48 (46-51)	47.55 ± 2.84	0.150
LVEF, %	64.00 (62.10-65.60)	63.40 ± 4.10	0.178
LA maximal volume at end-systole (Vmax, mL/m^2)	34.3 ± 26.7	33.5 ± 19.6	0.108
LA minimal volume at end-diastole (Vmin, mL/m^2)	16.0 ± 4.9	14.8 ± 5.4	0.083
Volume at the beginning of atrial systole (Vp, mL/m^2)	25.1 ± 6.1	24.2 ± 6.1	0.180
LAEF, %	53.35 ± 17.35	55.8 ± 15.11	0.030

Coronary Artery Disease / Acute Coronary Syndrome

OP-088

Atherogenic plasma index is associated with impaired systolic and diastolic functions in newly diagnosed Type 2 diabetic individuals without macrovascular disease

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Background and Aim: The atherogenic index of plasma (AIP) is a powerful and sensitive index that reflects the interaction between atherogenic and protective lipoproteins. The aim of our study is to investigate whether AIP is associated with systolic and diastolic functions in newly diagnosed type 2 diabetic individuals without overt cardiovascular disease.

Methods: 156 individuals (88 women, 49.2 ± 9.3 years) who applied to the internal medicine and cardiology outpatient clinics with any complaints and were not found to have overt cardiovascular disease in the examinations were included in the study. Of the individuals included, 104 had newly diagnosed type 2 diabetes (group 1; 58 women, 50.4 ± 9.8 years). As a control group, 52 individuals (group 2; 30 women; 46.7 ± 7.4 years) were included in the study. None of the individuals were using antihypertensive drugs, statins and antidiabetic drugs. AIP is calculated as a base 10 logarithmic conversion of the triglyceride to high-density lipoprotein-cholesterol ratio (TG/HDL-C). AIP values <0.11 were considered low risk, 0.11-0.21 intermediate risk, and >0.21 high risk. Laboratory values were analyzed in venous blood taken after 8 hours of fasting for all patients. All individuals underwent transthoracic echocardiography. In addition to left ventricular dimensions, systolic and diastolic functions and tissue Doppler velocities were obtained. Left ventricular myocardial performance index (MPI) as well as epicardial adipose tissue thickness were measured.

Results: Comparison of demographic data between groups is shown in Table 1. AIP was significantly higher in group 1 compared to group 2 (0.24 ± 0.32 vs. 0.01 ± 0.18, p<0.001). Diastolic and systolic functions were significantly impaired in group 1 compared to group 2. When classified according to AIP values, systolic and diastolic functions were found to be significantly impaired in the high-risk group compared to the low- and intermediate-risk groups (Table 2). MPI, which showed both systolic and diastolic functions at the same time, was found to be significantly impaired in the high-risk group compared to the other groups (Table 2). Epicardial fat thickness was significantly thicker in the high-risk group compared to the other groups (Table 2). In the correlation analysis, AIP was significantly correlated with ejection fraction and E/A ratio (r=-0.32; p<0.001, r=-0.31; p<0.001 respectively).

Conclusions: AIP, a marker of increased risk of atherosclerosis and coronary artery disease, is higher in newly diagnosed diabetic individuals than in the control group. Systolic and diastolic functions are associated with more impairment in systolic and diastolic functions in newly diagnosed type 2 diabetic individuals with high-risk AIP values compared to individuals with low- and intermediate-risk AIP values.

Table 1. Comparison of the demographic and laboratory features of the groups

	Group 1 (n=104)	Group 2 (n=52)	p
Age, years	50.4 ± 9.8	46.7 ± 7.3	0.019
Gender (female), n	58	30	0.673
Smoking, n	41	24	0.343
Systolic blood pressure, mmHg	138.3 ± 20.3	127.7 ± 17.7	0.001
Diastolic blood presure, mmHg	82.8 ± 12.8	76.4 ± 14.4	0.006
Heart rate, beat/min	85.0 ± 13.5	80.4 ± 9.9	0.034
BMI, kg/m^2	29.4 ± 5.0	24.8 ± 4.3	<0.001
HbA1c, %	9.1 ± 2.4	5.3 ± 0.3	<0.001
Glucose, mg/dL	191.1 ± 82.0	91.4 ± 5.9	<0.001
Total cholesterol, mg/dL	213.3 ± 72.8	196.2 ± 40.1	0.120
Triglyceride, mg/dL	209.5 ± 169.5	115.4 ± 58.6	<0.001
LDL cholesterol, mg/dL	124.7 ± 40.0	120.8 ± 35.4	0.554
HDL cholesterol, mg/dL	45.2 ± 13.8	55.0 ± 12.2	<0.001
Homa-IR	4.74 ± 2.17	2.29 ± 1.30	0.001
AIP	0.24 ± 0.32	0.01 ± 0.18	<0.001

Table 2. Comparison of the diastolic functions and epicardial fat thickness of the groups according to atherogenic index of plasma risk stratification

	High risk (>0.21)	Intermediate risk (0.11-0.21)	Low risk (<0.11)	p
Age, years	50.4 ± 9.9	48.3 ± 7.5	49.8 ± 8.7	0.688
Systolic blood pressure, mmHg	140.8 ± 19.6	133.8 ± 14.1	133.3 ± 21.3	0.100
Diastolic blood presure, mmHg	83.9 ± 127	84.6 ± 10.9	80.1 ± 14.1	0.206
Mitral E/A	0.83 ± 0.27	1.00 ± 0.28	1.04 ± 0.34	0.002
Mitral EDT, msn	241.7 ± 72.8	238.3 ± 61.7	220.0 ± 55.1	0.148
E/e' lateral	7.1 ± 1.9	6.6 ± 1.8	7.1 ± 2.1	0.659
E/e' septal	10.2 ± 2.5	8.3 ± 1.8	9.8 ± 2.8	0.034
MPI	0.29 ± 0.15	0.19 ± 0.07	0.28 ± 0.11	0.010
Epicardial fat thickness, mm	7.4 ± 2.0	5.5 ± 2.1	5.6 ± 2.1	<0.001
Ejection fraction, %	63.2 ± 2.5	66.1 ± 3.5	65.4 ± 3.7	<0.001

Coronary Artery Disease / Acute Coronary Syndrome

OP-089

The impact of symptom onset time on thrombus burden in patients with acute ST-elevation myocardial infarction

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Background and Aim: Circadian rhythm is involved in the pathogenesis of acute myocardial infarction (AMI) by affecting inflammatory processes that cause atherosclerosis and thrombosis. The circadian variation in the onset of AMI and the effectiveness of reperfusion treatments suggests that there may also be a circadian rhythm in the thrombus burden in the infarct-related artery. This study investigated the effect of symptom onset time on thrombus burden in patients presenting with acute ST-elevation MI (STEMI).

Methods: Between January 2018 and June 2021, 1068 patients with acute STEMI were enrolled retrospectively. The clinical and demographic characteristics of the patients were obtained from the hospital records. Patients were

divided into 4 groups according to the time of onset of chest pain (00:00-05:59, 06:00-11:59, 12:00-17:59, and 18:00-23:59). Angiographic thrombus burden was scored based on thrombolysis in myocardial infarction (TIMI) thrombus grades. After wiring and/or small balloon dilation, patients with thrombus burden grade 4 were categorized as large thrombus burden, while patients with thrombus burden grade <4 were categorized as small thrombus burden.

Results: The mean age was 59.13 (± 11.83) and 80.4% of the patient population was male. Table 1 summarizes baseline clinical characteristics of 4 groups. The peak incidence of MI symptom onset was observed between 06:00 and 11:59 (n=358, 33.5%), whereas the lowest incidence occured between 00:00 and 05:59 (n=196, 18.4%) (Figure 1). The rate of large thrombus burden was found to be statistically significantly higher in the patient group with pain onset between 00:00-05:59 hours than in the other patient groups (p<0.001) (Figure 2).

Conclusions: The present study showed a marked circadian periodicity at the time of onset of acute STEMI with a morning peak, but the thrombus burden was higher in patients with symptom onset between 00:00 and 05:59. This difference in thrombus burden may explain the high mortality in AMI patients with symptom onset between 00:00 and 05:59. Our results suggest that time of the day may be important in the pathophysiology of myocardial infarction.

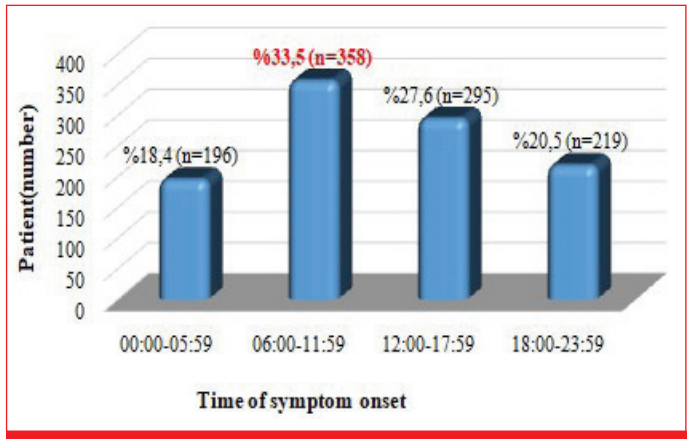

Figure 1. Circadian distribution of symptom onset time.

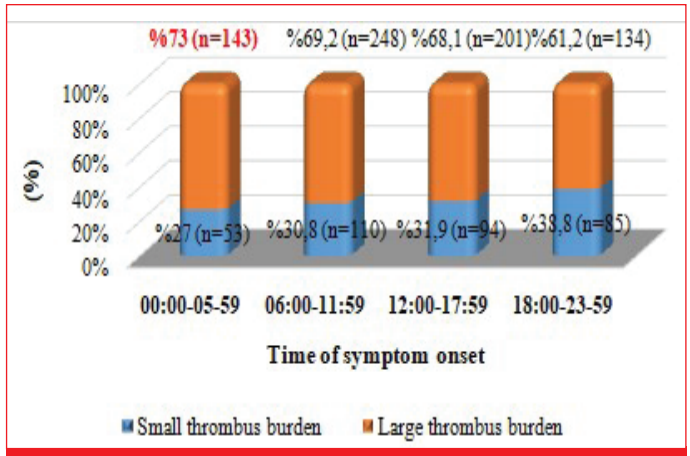

Figure 2. Circadian distribution of large thrombus burden.

Table 1. Baseline clinical characteristics

	00:00-05:59 (n=196)	06:00-11:59 (n=358)	12:00-17:59 (n=295)	18:00-23:59 (n=219)	p
Age, years	57.92 ± 12.27	59.77 ± 11.59	59.68 ± 12.14	58.38 ± 11.31	0.210
Male, n (%)	159 (81.1)	294 (82.1)	232 (78.6)	175 (79.9)	0.727
Hypertension, n (%)	93 (47.4)	129 (35.9)	117 (39.7)	74 (33.8)	0.020
Diabetes mellitus, n (%)	67 (34.2)	102 (28.6)	93 (31.5)	58 (26.5)	0.309
Current smoking, n (%)	71 (36.2)	141 (39.4)	123 (41.7)	88 (40.2)	0.652
Aspirin/p2y12 inhibitors, n (%)	20 (10.2)	40 (11.2)	31 (10.5)	16 (7.3)	0.488
Systolic blood pressure, mmHg	127.70 ± 20.90	124.28 ± 23.69	128.35 ± 24.00	125.37 ± 23.73	0.241
Diastolic blood pressure, mmHg	77.24 ± 13.70	74.62 ± 14.45	75.56 ± 13.99	74.27 ± 14.82	0.241
LVEF, %	44.56 ± 8.18	43.26 ± 8.39	43.96 ± 8.68	43.74 ± 8.27	0.479
Peak troponin, pg/mL	16519 ± 10657	19002 ± 9963	18012 ± 10325	15518 ± 10780	0.002
Peak CK-MB, IU/L	108 ± 94	138 ± 101	142 ± 111	132 ± 107	0.009

Data were expressed as n (%), mean ± standard deviation LVEF, left ventricular ejection fraction.

Table 2. Angiographic findings

Variable	00:00-05:59 (n=196)	06:00-11:59 (n=358)	12:00-17:59 (n=295)	18:00-23:59 (n=219)	p
Infarct related artery					0.514
LAD, n (%)	93 (47.4)	174 (48.5)	127 (43.1)	109 (49.8)	
LCX, n (%)	29 (14.8)	56 (15.7)	44 (14.9)	38 (17.4)	
RCA, n (%)	74 (37.8)	128 (35.9)	124 (42.0)	72 (32.9)	
Diseased vessels					0.123
Single vessel, n (%)	111 (56.6)	201 (56.0)	145 (49.2)	129 (58.9)	
Multi-vessel, n (%)	85 (43.4)	157 (44.0)	150 (50.8)	90 (41.1)	
cTFC after PCI	26.33 ± 18.20	26.16 ± 17.47	25.52 ± 18.21	23.33 ± 12.35	0.390
Thrombus burden					0.012
STB, n (%)	53 (27.0)	110 (30.8)	94 (31.9)	85 (38.8)	
LTB, n (%)	143 (73.0)	248 (69.2)	201 (68.1)	134 (61.2)	

Data were expressed as n (%), mean ± standard deviation. LAD: Left anterior descending artery, LCX: Left circumflex artery, RCA: Right coronary artery, cTFC: Corrected TIMI frame count, STB: Small thrombus burden, LTB: Large thrombus burden.

Coronary Artery Disease / Acute Coronary Syndrome

OP-090

The predictive value of atherogenic index of Plasma levels in the development of new-onset atrial fibrillation among ST-segment elevation myocardial infarction patients

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Background and Aim: New-onset atrial fibrillation (NOAF) affects 2.3% to 21% of acute ST-segment elevation myocardial infarction (STEMI) patients and is associated with poor clinical outcomes. Detecting high-risk individuals early is essential for preventing complications and improving prognosis. The atherogenic index of plasma (AIP) reflects the balance between atherogenic and protective lipoproteins in the blood. AIP has demonstrated strong predictive abilities for atherosclerosis and coronary artery disease (CAD). This study aims to assess the predictive value of AIP levels in the development of NOAF among STEMI patients.

Methods: In our retrospective study, we enrolled a total of 1475 consecutive patients diagnosed with STEMI between January 2016 and July 2021. We excluded the patients who were diagnosed with AF or had documented AF before admission. The patients who underwent emergency coronary artery bypass graft surgery (CABG) or had a history of CABG, and patients with inaccessible data were also excluded. AIP was calculated using the formula log10 (TG/HDL-C). The diagnosis of AF was made based on the consensus guidelines. NOAF was defined as an arrhythmia occurring after hospitalization, characterized by irregular RR intervals on the ECG, absence of identifiable P waves with an unidentified isoelectric line, and an atrial rhythm exceeding 300 bpm.

Results: The study population consisted of 1133 STEMI patients. NOAF developed in 66 patients (5.8%) during the hospitalization. AIP levels were higher in patients with NOAF than in patients without NOAF (0.53 ± 0.27 vs. 0.46 ± 0.28, p=0.044). Compared with patients without NOAF, patients with NOAF were older and had a more frequent history of HT and smoking. To account for the marked differences between the number of patients with and without NOAF and to eliminate the effects of the variables that were previously found to be associated with NOAF, we performed propensity score matching. In all, 64 patients were selected from among the patients without NOAF and matched with 66 patients with NOAF. In the matched population, we found that the AIP levels were significantly higher in patients with NOAF than in patients without NOAF (0.53 ± 0.27 vs. 0.15 ± 0.21, p=0.014). Receiver operating characteristics curve analysis showing the AIP cutoff value of 0.27 that predicted NOAH with 86.4% sensitivity and 84.4% specificity. Univariate logistic regression showed an association between NOAF and risk factors including WBC count, bazal syntax score, AIP (p<0.05 for all). Multivariable logistic regression analysis identified only AIP [31.303 (11.713-83.658)] as independent predictors of new onset AF during hospital stay.

Conclusions: This study highlights the potential of AIP as a valuable clinical tool for risk stratification and prediction of NOAF in STEMI patients. Incorporating AIP assessment into routine clinical practice may aid in optimizing patient management and outcomes in this high-risk population.

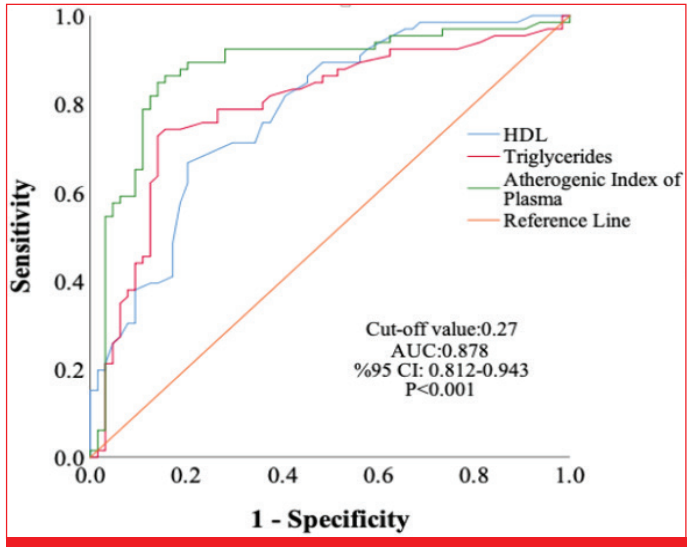

Figure 1. Estimation of NOAF by ROC curve of AIP.

Table 1. Baseline clinical, demographic, and laboratory variables

	Total (n=1133)	NOAF (+) (n=66)	NOAF (-) (n=1067)	p
Age, years	56 ± 12	62 ± 13	56 ± 12	0.000
Gender (female), n (%)	203 (17.9)	16 (1.4)	187 (16.5)	0.168
Diabetes mellitus, n (%)	264 (23.3)	21 (1.9)	243 (21.4)	0.092
Hypertension, n (%)	461 (40.7)	39 (3.4)	422 (37.2)	0.002
Smoking, n (%)	629 (55.5)	28 (2.5)	601 (53)	0.027
Hyperlipidemia, n (%)	524 (46.2)	27 (2.4)	497 (43.9)	0.370
Family history of CAD, n (%)	268 (23.7)	12 (1.1)	256 (22.6)	0.281
Beta-blocker, n (%)	77 (6.8)	9 (1)	68 (6)	0.023
ACEi/ARB, n (%)	228 (20.1)	12 (1.1)	216 (19.1)	0.685
Statin, n (%)	206 (18)	9 (0.1)	197 (17)	0.324
Heart rate, bpm	77 ± 16	85 ± 18	77 ± 16	0.000
Systolic blood pressure, mmHg	132 ± 31	129 ± 37	132 ± 31	0.837
Killip class >1 on admission, n (%)	181 (16)	25 (2.2)	156 (13.8)	0.000
Hemoglobin, g/dL	13.6 ± 1.7	13.1 ± 2.1	13.7 ± 1.8	0.025
WBC count (/10^3)	12.3 ± 3.8	13.4 ± 4.2	12.2 ± 3.8	0.024
Glucose, mg/dL	127 (105-171)	136 (102-209)	127 (105-169)	0.181
eGFR, mL/min	89 ± 25	80 ± 28	89 ± 24	0.004
High-density lipoprotein cholesterol, mg/dL	37 (31-45)	35 (27-40)	38 (31-45)	0.002
Triglycerides, mg/dL	110 (78-156)	111 (83-158)	110 (77-156)	0.801
Total cholesterol, mg/dL	180 ± 44	174 ± 51	181 ± 43	0.175
Low-density lipoprotein cholesterol, mg/dL	117 ± 39	118 ± 45	117 ± 39	0.772
Atherogenic index of plasma	0.47 ± 0.28	0.53 ± 0.27	0.46 ± 0.28	0.044
Peak creatine kinase MB, ng/mL	181 (103-322)	350 (167-408)	176 (101-306)	0.000
Total ischemia time, min	207 ± 122	227 ± 120	206 ± 122	0.093
Baseline TIMI flow (<3, pre), n (%)	1021 (90)	61 (1)	960 (84.7)	0.907
Bazal SYNTAX score	17 ± 5	18 ± 4	17 ±5	0.000
Infarct-related LAD artery, n (%)	586 (51.7)	40 (3.5)	546 (48.2)	0.137
Left ventricular ejection fraction	47 ± 8	15 (1.3%)	120 (10.6%)	0.000
Hospital stay, days	5 ± 3	8 ± 5	5 ± 3	0.000

NOAF: New-onset atrial fibrillation, ACEi: Angiotensin-converting enzyme inhibitors, ARB: Angiotensin receptor blockers, WBC: White blood cells, GFR: Glomerular filtration rate, LAD: Left anterior descending.

Table 2. Propensity score matching for NOAF

	NOAF (-) (n=64)	NOAF (+) (n=66)	Total (n=130)	p
Age, years	62 ± 12	62 ± 13	62 ± 13	-
Gender (female), n (%)	20 (55.6)	16 (44.4)	36 (28%)	-
Diabetes mellitus, n (%)	17 (45)	21 (55)	38 (30)	-
Hypertension, n (%)	28 (42)	39 (58)	67 (51)	-
Smoking, n (%)	18 (39)	28 (61)	46 (35)	-
Hyperlipidemia, n (%)	25 (48)	27 (52)	52 (50)	0.831
Family history of CAD, n (%)	16 (25)	12 (18)	28 (21)	0.346
Beta-blocker, n (%)	4 (7)	9 (13)	13 (10)	0.162
ACEi/ARB, n (%)	17 (27)	12 (22)	29 (22)	0.253
Statin, n (%)	12 (19)	9 (13)	21 (16)	0.430
Heart rate, bpm	79 ± 17	85 ± 18	82 ± 18	0.071
Systolic blood pressure, mmHg	130 ± 34	129 ± 37	129 ± 35	-
Killip class >1, n (%)	12 (32.4)	25 (38.6)	37 (28)	-
Hemoglobin, g/dL	12,7±2	13,1±2,1	12,9±2,1	0.098
WBC Count, /10^3	11,9±4,3	13,4±4,2	12,7±4,3	0.028
Glucose, mg/dL	128 (107-156)	135 (102-209)	130 (105-189)	0.391
eGFR, mL/min	89±27	80±28	84±28	0.074
High-density lipoprotein cholesterol, mg/dL	45 (38-54)	35 (27-40)	40 (31-48)	0.000
Triglycerides, mg/dL	61 (45-82)	111 (83-158)	83 (54-133)	0.000
Total cholesterol, mg/dL	176±44	174±51	175±48	0.694
Low-density lipoprotein cholesterol, mg/dL	115±40	118±45	117±42	0.789
Atherogenic index of plasma	0,15±0,21	0,53±0,27	0,34±0,31	0.000
Peak CK-MB, ng/mL	220 (112-363)	350 (167-408)	248 (139-398)	-
Total ischemia time, min	214±122	228±120	221±120	0.455
Bazal SYNTAX score	17±4	18±4	17±4	0.036
Infarct-related LAD, n (%)	38 (59%)	40 (60%)	78 (60%)	-
Noreflow, n (%)	7(10%)	15(22%)	22(17%)	-
LVEF, %	44±8	41±9	42±8	-
Length of hospital stay, day	6±4	7±5	7±5	0.023

NOAF: New-onset atrial fibrillation, CAD: Coronary artery disease, ACEi: Angiotensin-converting enzyme inhibitors, ARB: Angiotensin receptor blockers, WBC: White blood cells, GFR: Glomerular filtration rate, LAD: Left anterior descending, CK: Creatinin kinase, LVEF: Left ventricular ejection fraction.

Table 3. Independent predictors for NOAF with univariate and multivariate logistic regression analysis

	Univariate analysis	Univariate analysis	Multivariate analysis	Multivariate analysis
	OR (95% CI)	p	OR (95% CI)	p
Hyperlipidemia	1.080 (0.535-2.179)	0.830		
Family history of CAD	0.667 (0.287-1.550)	0.346		
Beta-blocker	2.368 (0.691-8.122)	0.170		
Heart rate	1.018 (0.997-1.039)	0.092		
Hemoglobin	1.104 (0.932-1.309)	0.253		
WBC	1.099(1.009-1.198)	0.031	1.036 (0.923-1.164)	0.548
GFR	0.988 (0.976-1.001)	0.073		
Atherogenic index of plasma	34.2 (12.907-90.618)	<0.001	31.303 (11.713-83.658)	<0.001
Bazal SYNTAX score	1.104 (1.003-1.216)	0.044	1.052 (0.920-1.202)	0.458
Length of hospital stay	1.084 (0.998-1.176)	0.055		

NOAF: New-onset atrial fibrillation, CAD: Coronary artery disease, WBC: White blood cells, GFR: Glomerular filtration rate, OR: Odds ratio.

Coronary Artery Disease / Acute Coronary Syndrome

OP-091

Predictive value of the leuko-glycemic index (LGI) in new-onset atrial fibrillation after coronary artery bypass grafting

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Background and Aim: Postoperative atrial fibrillation (POAF) is a potentially life-threatening complication after coronary artery bypass grafting (CABG) surgery. According to the newer studies; it is estimated to be seen around 20-50% and importance of this situation is mentioned in several studies. A novel inflammatory marker, the leuko-glycemic index (LGI), showed a better predictive value for POAF development compared to its components. We investigated the relationship between LGI and new-onset POAF during in hospital stay.

Methods: In a retrospective study of consecutive patients (n=826) without a history of atrial fibrillation who underwent CABG, patients were divided into 2 groups according to new-onset postoperative atrial fibrillation. The prognostic significance of LGI in new-onset AF after CABG was compared in the two groups.

Results: In the populations studied, the incidence of new-onset POAF was about 11.6% (n=96). We evaluated univariable and multivariable binary logistic regression analyses for all variables in order to identify the independent predictors of atrial fibrillation after CABG in hospital. In univariable regression analyses, leukoglycemic index, age, eGFR, chronic obstructive lung disease, peripheral arterial disease, hyperlipidemia, LAAP diameter, uric asid and CRP were found to be correlated with new onset atrial fibrillation. When we entered these variables into the multivariable regression analysis, leukoglycemic index (OR: 1.22, 95% CI: 1.04-1.44, p=0.01), age (OR: 1.04, 95% CI: 1.01-1.07, p<0.01), CRP (OR: 1.05, 95% CI: 1.01-1.05, p<0.01) and LAAP diameter (OR: 2.12, 95% CI: 1.12-4.68, p<0.01) were ascertained as independent predictors of atrial fibrillation after CABG. In ROC analysis, the leukoglycemic index >1.263 predicted atrial fibrillation after CABG with a sensitivity of 91% and a specificity of 59%. The area under the receiver curve of the LGI for the prediction of new-onset atrial fibrillation after CABG was 0.737 (95% CI: 0.705-0.767, p<0.001).

Conclusions: Our study highlights the prognostic importance of LGI as an independent risk factor for new-onset atrial fibrillation in postoperative CABG patients. This inexpensive and easily assessed biochemical parameter may help to improve the prediction of POAF and the selection of patients who could benefit from a preventive strategy.

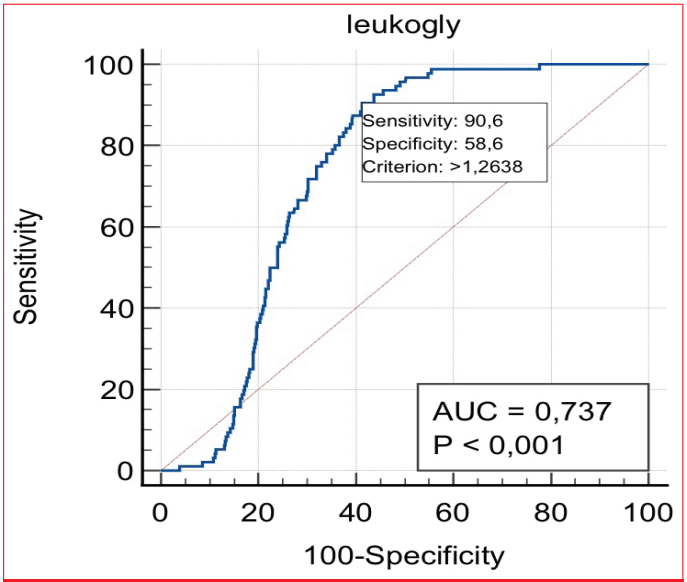

Figure 1. In ROC analysis, the leukoglycemic index >1.263 predicted atrial fibrilation after CABG with a sensitivity of 91% and a specificity of 59%, as shown in Figure 1.

Table 1. Variables which was entered into multivariable regression model for atrial fibrillation after CABG

Variable	Adjusted OR (%95 CI)	p value
Age years	1.04 (1.01-1.07)	**<0.01***
PAH	1.58 (0.8-2.8)	0.13
HPL	0.6 (0.3-1.0)	0.09
COPD	1.6 (0.8-3.2)	0.15
CRP	1.05 (1.01-1.05)	**<0.01***
LGI	1.22 (1.04-1.44)	**0.01***
Uric Aside	1.12 (0.9-1.28)	0.06
GFR	1.00 (0.99-1.01)	0.53
LA AP Diameter	2.12(1.12-4.68)	**<0.01***

Lipid / Preventive Cardiology

OP-092

Implementation of primary prevention for coronary artery disease amid the COVID-19 pandemic

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Background and Aim: The implementation and especially the maintenance of primary prevention programs in improving health-related behavioral outcomes for coronary artery disease (CAD) may be very challenging especially during and post-pandemic conditions. The present study was designed to assess the feasibility and effectiveness of a longitudinally structured, online-enhanced education and follow-up program on behavioral outcomes for CAD prevention amid the COVID-19 pandemic.

Methods: Due to COVID-19 restrictions our university suspended face-to-face teaching activities on March 16, 2020, and re-started online education on March 30th via microsoft teams program. Coronary Artery Disease Online Prevention Project (SCAD-OPP) was designed as a model-longitudinal study and utilized medical school students to conduct the entire project under the supervision of professors. It started in April 2020 and had 2 different online education and training phases. In the first phase, 3rd year medical students underwent an online 8-months specially designed training program on primary prevention for CAD. In the second phase, a series of online conferences on primary prevention for CAD were organized by the University. Per inclusion criteria each student was asked to enroll 1 or 2 participants from local population and assist them during the online intervention. Pre and post conference knowledge were collected and assessed via online tools. Every intervention was conducted by specially trained 3rd year medical students and an education booklet which was specifically designed for this study was mailed to the participants. Every other month thereafter, for 6 months, each participant was followed by phone. At the 6 months follow-up, data was collected to assess the impact of enhanced education and follow-up program on behavioral outcomes.

Results: A total of 72 participants were enrolled; 71% were women, mean age was 45 ± 13 years, only 7% had a graduate school degree; 22% were not working. Mean BMI was 26.2 ± 4.6 kg/m^2. Overall knowledge on CAD risk factors, primary prevention measures, diet and daily exercise habits were very poor. After the online-enhanced education and follow-up program there was a significant improvement on the knowledge of CAD risk factors and primary prevention measures (p<0.001). More importantly, the follow-up program led participants to implement those positive changes into their lives and maintain a healthy lifestyle. A separate cost analysis showed significant savings.

Conclusions: This is the first study which showed that a longitudinally structured online-training program of medical students could be utilized to implement an online-enhanced education and follow-up program for primary prevention of CAD with successful outcomes. This model online program is not only cost-effective and beneficial for public interest but also enhances active interaction of medical students with patients at a very early stage of their career.

Coronary Artery Disease / Acute Coronary Syndrome

OP-093

Impact of coronary slow phenomenon on beat-to-beat QT interval variability

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Background and Aim: Coronary slow flow phenomenon (CSF) is associated with increased incidence of ventricular arrhytmias. Abnormality in ventricular repolarization is linked with malign ventricular arrhytmias. Clinical studies demonstrated positive significant linear correlation between CSF and QT dispersion. QT interval was observed higher in CSF patients compared to normal coronary arteries.

Methods: Totally seventy-five patients, forty-six of them diagnosed CSF in coronary angiography were included in this study. Beat-to-beat QT interval variability (QTIV) and QT dispersion were measured on surface electrocardiography.

Results: Mean heart rate was 76.24 ± 12.72 in control group and 75.80 ± 12.60 in CSF (p=0.844). Corrected QT dispersion was statistically higher in CSF patients (41.64 ± 7.72 vs. 36.49 ± 9.23, p=0.001). The heart rate variability (HRV) was higher in CSF patients compared to controls (9.8 ± 9.1 vs. 8.2 ± 7.9, p=0.008). QT variabilty index was not statistically different between CSF and controls (-1.04 ± 0.48 vs. -0.80 ± 0.78, p=0.468) (Table 1).

Conclusions: Differences in repolarization times between leads pose a risk for ventricular arrhythmias. CSF increases the frequency of ventricular arrhythmias by increasing QT dispersion. HRV, which is associated with the autonomic nervous system and adrenergic activity, was higher in the CSF group, and QTVI, one of the current parameters showing healthy repolarization, was found to be similar in both groups. The negative effects of CSF on repolarization are known, and there is a need for extensive studies with reliable and up-to-date predictors of how is the affects and how to manage to decrease the frequency of ventricular arrhythmias.

Table 1. Ventricular repolarization parameters in coronary slow flow phenomenon

Variables	Coronary slow flow (n=46)	Control (n=29)	p
Age, years	75.80 ± 12.60	76.24 ± 12.72	0.844
cQT dispersion, msn	41.64 ± 7.72	36.49 ± 9.23	0.001
Heart rate variability	9.8 ± 9.1	8.2 ± 7.9	0.008
QT variability index	-1.04 ± 0.48	-0.80 ± 0.78	0.468

Lipid / Preventive Cardiology

OP-094

Adherence to current dyslipidemia guldeline in patients utilizing statins According to risk groups: The AIZANOI study

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Background and Aim: Despite very strong evidence and guideline recommendations about decreasing low density lipoprotein cholesterol (LDL-C) to the target levels determined by SCORE risk group, there are difficulties to reach guideline-directed targets. The aim of this study was to assess the adherence to the current European Society of Cardiology dyslipidemia guidelines, the ratio of reaching target values according to risk groups and the reasons for not reaching LDL-C goals in patients on already statin therapy in an outpatient population.

Methods: Adherence to Current Dyslipidemia Guldeline in Patients UtiliZing Statins AccordiNg tO RIsk Groups: AIZANOI Study is a multi-center, cross sectional observational study conducted in 9 cardiology centers between 01.08.2021 and 01.11.2021. A total of 1225 patients using statins of at least 3 months duration before the study were included.

Results: 1225 patients (mean age 62 ± 11 years, 366 female) who were already on statin therapy for at least 3 months were included. Majority of patients (90.8%) had very high SCORE risk and 5.2%, 3.6%, and 0.3% of patients had high, moderate, and low SCORE risk, respectively, according to 2019 European Society of Cardiology (ESC)/European Atherosclerosis Society (EAS) Guidelines for the management of dyslipidemia. More than half (58.2%) of the patients were using high intensity statin regimens as atorvastatin 40-80 mg or rosuvastatin 20-40 mg. Only 26.2% of patients had target LDL-C level according to their risk score. Despite 58.4% of very high-risk patients and 44.4% of high risk patients having been using a high intensity statin regimen, only 24.5% of very high risk patients and 34.9% of high risk patients have reached guideline recommended LDL-C levels. Most prevalent reason for not using target dose statin was physician preference (40.3%). 97.1% of patients were on mono therapy whereas only 2.9% patients were receiving combination therapy. 26.9% of male patients and 24.6% of female patients were on LDL-C targets.

Conclusions: The AIZANOI study showed that we achieved a target LDL-C level in only 26.2% of patients using statin therapy. Although 58.4% of patients with a very high SCORE risk and 44.4% of patients with a high SCORE risk were using a target dose statin regimen, we were only able to achieve guideline-recommended LDL-C levels in 24.5% and 34.9% of them, respectively. Physician inertia is one of the major factors in non-adherence to guidelines. These findings highlight that combination therapy is needed in most of the patients.

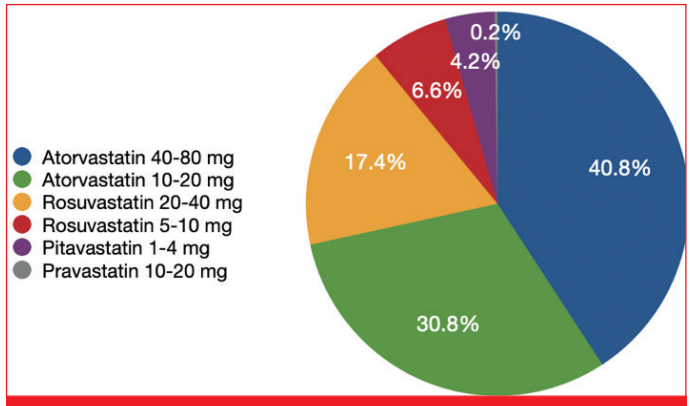

Figure 2. Types and doses of statins.

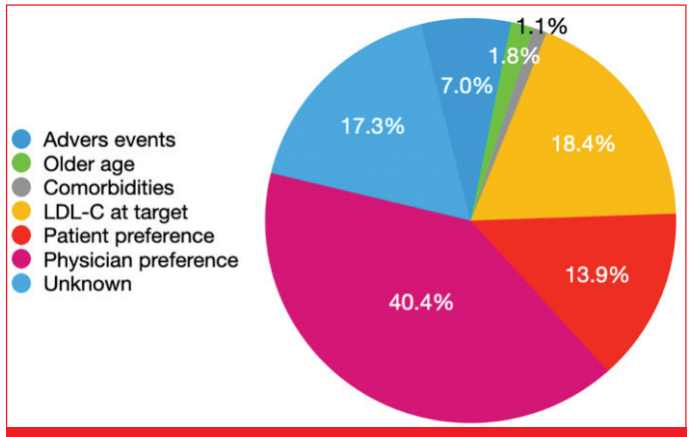

Figure 3. The reasons of not using target dose of statin.

Figure 1. Cities of participating centers.

Table 1. Risk status, target LDL-C levels, type and the intensity of statin therapy, the ratio of achievement of target LDL-C levels, ezetimibe usage of the patients and duration of current statin therapy

Risk status	All patients	Female	Male	p
n (%)	1225	366 (29.9)	859 (70.1)	-
Very high, n (%)	1110 (90.8)	289 (79.2)	823 (96.4)	0.001
High, n (%)	63 (5.2)	40 (11.0)	20 (2.3)	0.001
Moderate, n (%)	44 (3.6)	34 (9.0)	9 (1.1)	0.001
Low, n (%)	4 (0.3)	2 (0.5)	2 (0.2)	0.38
Intensity of statin therapy				
High, n (%)	846 (69.3)	226 (61.9)	620 (72.5)	0.001
Moderate, n (%)	370 (30.3)	138 (37.8)	232 (27.1)	0.001
Low, n (%)	4 (0.3)	1 (0.3)	3 (0.4)	0.83
Type and dose of statin				
Atorvastatin 40-80 mg, n (%)	498 (40.8)	127 (34.7)	371 (43.3)	0.001
Rosuvastatin 20-40 mg, n (%)	213 (17.4)	62 (16.9)	151 (17.6)	0.79
Atorvastatin 10-20 mg, n (%)	376 (30.8)	118 (32.2)	258 (30.1)	0.44
Rosuvastatin 5-10 mg, n (%)	80 (6.5)	35 (9.6)	45 (5.3)	0.001
Pitavastatin 1-4 mg, n (%)	51 (4.2)	23 (6.3)	28 (3.3)	0.02
Pravastatin 10-20 mg, n (%)	3 (0.2)	0	3 (0.4)	0.26
Using target dose statin				
Yes, n (%)	723 (59.2)	206 (56.3)	517 (60.5)	0.2
No, n (%)	498 (40.8)	160 (43.7)	337 (39.4)	0.14
The ratio of LDL-C target level achievement				
LDL-C at target, n (%)	320 (26.2)	90 (24.6)	230 (26.9)	0.43
LDL-C not at target, n (%)	901 (73.8)	276 (75.4)	625 (73.1)	0.34
Ezetimibe				
Yes, n (%)	36 (2.9)	6 (1.6)	30 (3.5)	0.08
No, n (%)	1185 (97.1)	360 (98.4)	825 (96.5)	0.04
Duration of statin therapy				
3-6 months, n (%)	201 (16.5)	53 (14.5)	148 (17.3)	0.23
6-12 months, n (%)	233 (19.1)	65 (17.8)	168 (19.6)	0.46
>1 year, n (%)	787 (64.5)	247 (67.7)	540 (63.1)	0.12

Table 2. The reasons of not using target dose of statin

Reasons	All patients	Female	Male	p
n (%)	1225	366 (29.9)	859 (70.1)	-
Adverse events, n (%)	39 (3.2)	9 (2.5)	30 (3.5)	0.35
Older age, n (%)	10 (0.8)	4 (1.1)	6 (0.7)	0.48
Comorbidities, n (%)	6 (0.5)	3 (0.8)	3 (0.3)	0.28
LDL-C at target, n (%)	102 (8.3)	26 (7.1)	76 (8.8)	0.31
Patient preference, n (%)	77 (6.3)	17 (4.6)	60 (7.0)	0.12
Physician preference, n (%)	224 (18.3)	82 (22.4)	142 (16.5)	0.01
Unknown, n (%)	96 (7.8)	27 (7.4)	69 (8.0)	0.7

LDL-C: Low-density lipoprotein cholesterol.

Table 3. Target dose statin usage ratio of very high and high risk groups and the ratio of achieving target LDL-C level in patients using target dose statin

SCORE risk groups	Target dose statin use ratio	LDL at target
Very high risk	58.4%	24.5%
High risk	44.4%	34.9%

Coronary Artery Disease / Acute Coronary Syndrome

OP-095

Prognostic and predictive performance of immune-nutritional scoring systems in perioperative myocardial infarction/injury following non-cardiac surgery

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Background and Aim: Perioperative myocardial infarction/injury (PMI) is an important complication of non-cardiac surgery that usually has a clinically silent presentation but is associated with a poor prognosis. This study aims to investigate how to predict PMI after non-cardiac surgery and whether preoperative risk scores contribute to the prediction of PMI and prognosis.

Methods: We included 396 consecutive patients for whom cardiology consultation was requested before non-cardiac surgery between December 2022 and March 2023. An absolute increase in high- sensitivity cardiac troponin (hs-cTn) concentration of more than the upper limit of normal on days 1 or 2 after surgery compared to the pre-operative level is defined as PMI. Patients who did not have follow-up of hs-cTn value on days 1 or 2 after surgery were excluded from the study. Immune-inflammatory-nutritional operative risk scores were calculated according to the preoperative blood values of all patients. The total cholesterol (TC) level, serum albumin (Alb) content, neutrophil-to-lymphocyte ratio (NLR) and lymphocyte-to-monocyte ratio (LMR) were determined to calculate the Naples prognostic score (NPS). The sum of the scores of the four parameters is the NPS. The CONUT score consists of Alb and TC concentrations and lymphocyte count. The Prognostic Nutritional Index (PNI) was calculated as follows: (10xAlb+0.005xlymphocyte count). The systemic inflammation score (SIS) score consists of Alb concentration and LMR.

Results: A total of 246 patients were included in the study. PMI was detected in 12.2% of the patients (n=30). Mean age was higher in the PMI group (71.2 ± 14.2 vs. 64.1 ± 13.6, p=0.004), hypertension and peripheral arterial disease were more frequent (p=0.024, p=0.028; respectively). Pre-operative urea, creatinine, pro-BNP, d-dimer, hs-cTn, CRP, neutrophil, monocyte levels were significantly higher than those in the PMI group; Alb, TC levels were lower. The rate of high-risk surgery was higher in the PMI group than in the non-PMI group (47% vs. 18%, p=0.005). PMI group had higher NPS and SIS scores (p<0.001, p=0.008; respectively), while CONUT and PNI scores were similar. The multivariate logistic regression analysis revealed that age (p=0.027), creatinine level (p=0.036), high-risk surgery (p=0.002), and NPS score (p=0.016) were independent predictors of PMI. During the postoperative 30 days, a total of 15 patients died (6.1%). The 30-day mortality rate was higher in the PMI group (20% vs. 4%; p=0.045) than in the non-PMI group. In Cox regression models, NPS (p=0.043), albumin (p=0.027), and PMI (p=0.028) were independent risk factors of 30-day mortality. A NPS ≥3 predicts PMI with 67% sensitivity, 65% specificity, and mortality with 87% sensitivity and 65% specificity.

Conclusions: Among the scores that provide information on immune-nutritional status, NPS can assist in screening patients at high risk for PMI during non-cardiac surgery and can be appropriate for predicting prognosis.

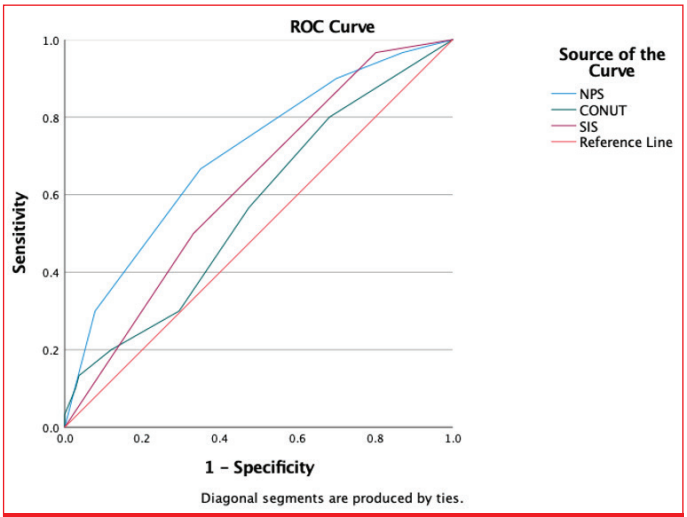

Figure 1. ROC curve showing the sensitivity and specificity of Naples prognostic score in predicting PMI

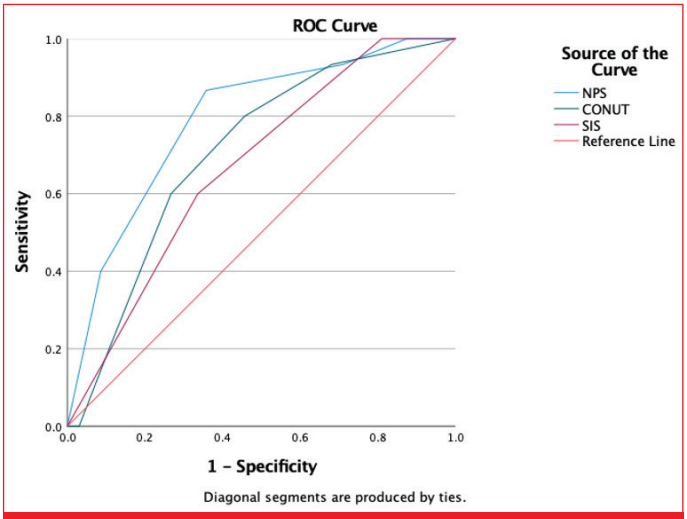

Figure 2. ROC curve showing the sensitivity and specificity of Naples prognostic score in predicting mortality

Cardiac Imaging / Echocardiography

OP-096

Association between epicardial fat volume measured by cardiac magnetic resonance imaging and microvascular obstruction in patients with STEMI

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Background and Aim: Epicardial fat volume (EFV) has been studied extensively and linked to the advanced inflamation and accelerated atherosclerosis. Moreover, previous studies also demonstrated increased fatal and non-fatal myocardial infarction in patients with high EFV. On the other hand, there are also several studies in the literature which concluded higher EFV has been associated with better cardiovascular outcomes in patients with myocardial infarction and called this phenomenon as 'EFV paradox'. In this study, we aimed to investigate the impact of EFV measured by cardiac magnetic resonance (CMR) imaging on myocardial salvage and long term prognosis in patients with first ST-elevation myocardial infarction (STEMI).

Methods: Patients with first STEMI who underwent succesfull primary percutaneous coronary intervention were enrolled. Patients' demographic features, clinical characteristics and biochemical analyses were recorded. CMR imaging was performed between 3rd and 7th days of index myocardial infarction. Epicardial fat borders were traced manually in each slice of short-axis cine images at the end-diastolic phase (Figure 1). The areas measured for each slice were summed and multiplying by slice thickness to obtain EFV. EFV was indexed to body surface area. The primary outcome of the study was microvascular obstruction (MVO).

Results: Sixty-eight patients were included in the analysis. There was male dominance in both groups and comorbidities including diabetes mellitus (p=0.674), hypertension (p=0.674), dyslipidemia (p=0.975) and coronary artery disease (p=0.634) were similar between groups. EFV and EFV index were significantly higher in patients with microvascular obstruction (MVO) (p<0.005) (Table 1). Although univariate analysis for the prediction of MVO demonstrated that EFV is associated with MVO [p=0.042, OR: 1.053 (1.181-2.103)], there was no independent relationship between EFV and MVO according to multivariate analysis [p=0.242, OR: 1.025 (0.984-1.068)] (Table 2). Considering the long term cardiovascular events, univariate analyses demonstrated that EFV is not associated with cardiovascular death [p=0.115, OR: 0.962 (0.918-1.009)].

Conclusions: Although our study have demonstrated that EFV is higher among patients with MVO, there is no independent association between EFV and MVO. Moreover, EFV is not associated with long term adverse cardiovascular events. Further studies with higher study population and long term follow-up are required for the clarification of EFV paradox.

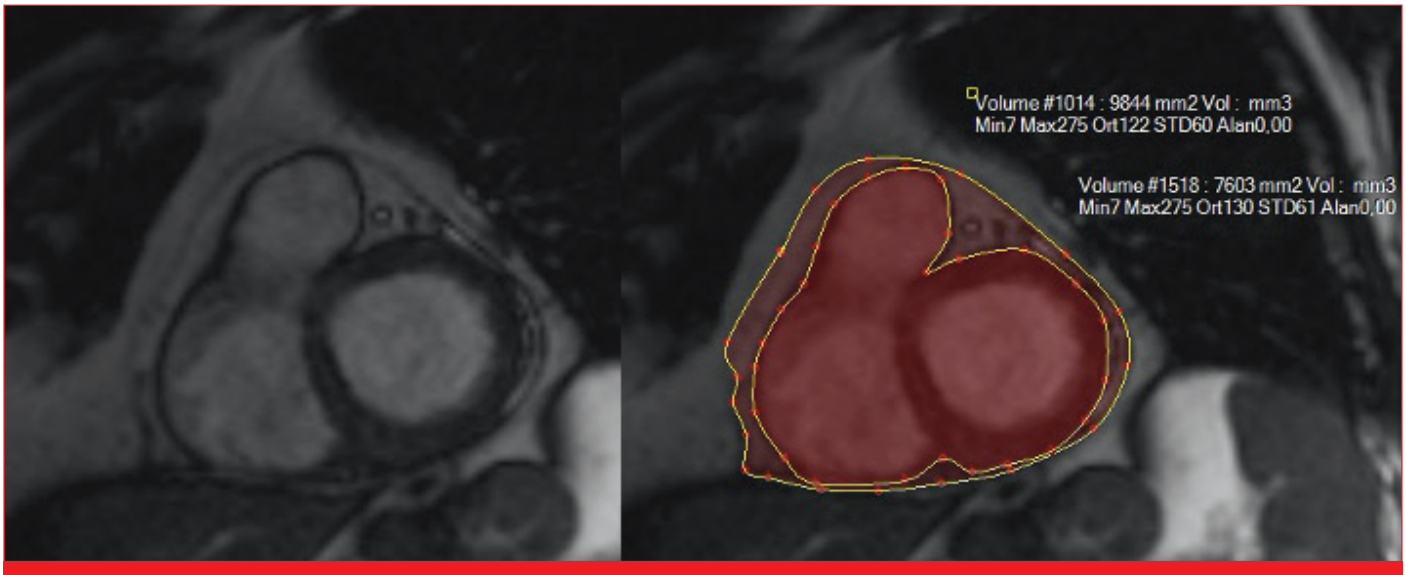

Figure 1. The method used for the quantification of epicardial fat volume by cardiac magnetic resonance imaging.

Table 1. Patients' baseline characteristics

	MVO (+)	MVO (-)	p
Male	100%	86.5%	0.060
Age, years	59.7 ± 9.3	54.4 ± 9.1	0.035
Diabetes mellitus	29.2%	24.3%	0.674
Hypertension	29.2%	24.3%	0.674
Smoking	62.5%	54.1%	0.515
Dyslipidemia	8.3%	8.1%	0.975
Weight, kg	80 (70-90)	80 (76.5-89)	0.951
BMI	1.97 ± 0.2	1.99 ± 1.4	0.688
Coronary artery disease	16.7%	21.6%	0.634
Follow-up time, days	16 (10.25-20)	19 (19-22)	0.095
Atrial fibrilation	0%	0%	
Troponin, pg/dL	3.04 (1.25-6.07)	0.738 (0.307-2.1)	0.001
Symptom-to-distal flow time, minutes	240 (166.2-525)	195 (131.25-330)	0.200
Epicardial fat volume, mL	90.7 (84.7-95.8)	80.6 (73.6-85.0)	<0.001
Epicardial fat volume index, mL/m^2	46.7 (41.7-51.7)	41.65 (36.5-44.7)	0.002
Left ventricle mass, gr	124 ± 32.8	111 ±34.3	0.171
Left ventricle mass index, gr/m^2	61 (50-74)	56 (44.5-65)	0.148
Left ventricle ejection fraction, %	44.2 ± 12.3	51.4 ±10.9	0.024
Left ventricle enddiastolic volume, mL	109.3 (78.9-128.8)	94.9 (78.7-107.4)	0.290
Left ventricle enddiastolic volume index, mL/m^2	52.7 ± 17.1	48.4 ± 12.5	0.271
Left ventricle endsystolic volume, mL	57.5 (33.8-88.9)	43.5 (32-57.5)	0.111
Left ventricle endsystolic volume index, mL/m^2	28.5 (18-42)	21.5 (17-29.7)	0.142
Anterior location	63.2%	56.8%	0.645
Cardiac death	4.2%	0%	
Ventricular arrythmia	4.2%	5.4%	0.827
Heart failure	54.2%	43.2%	0.404
Indication for ICD	25%	27%	0.860
Control LVEF, n (%)	40 (35.2-48)	45 (35-50)	0.346

Quantitative variables are expressed mean ± standard deviation or median (interquartile range). Qualitative variables are expressed as percentages.

Table 2. Univariate and multivariate analysis for the endpoint of microvascular obstruction

	Univariate			Multivariate		
	OR	CI	p	OR	CI	p
Age	1.066	1.003-1.134	0.041	1.050	0.977-1.128	0.188
Troponin level	1.576	1.181-2.103	0.002	1.451	1.041-2.022	0.028
Epicardial fat volume	1.053	1.002-1.107	0.042	1.025	0.984-1.068	0.242
Epicardial fat volume index	1.065	0.989-1.147	0.096			
LVEF	0.946	0.901-0.994	0.029	0.950	0.897-1.005	0.076

Cardiac Imaging / Echocardiography

OP-097

Changes in blood pressure is associated with decrease in left ventricular mass index after kidney transplantation

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Background and Aim: Arterial hypertension has negative impact on both cardiovascular system and transplant survival after kidney transplant (KTx). Left ventricular hypertrophy (LVH) is the most prominent manifestation of cardiovascular organ damage due to arterial hypertension and it is associated with major cardiovascular events. The aim of this study is to investigate the relationship between blood pressure control after KTx and left ventricular mass index (LVMI).

Methods: A total of 49 patients who underwent KTx between 2019 and 2021 were retrospectively investigated. All the participants underwent echocardiography at baseline and one year following the transplantation procedure. Pre- and post-KTx systolic and diastolic blood pressure (SBP/DBP) were measured. We compared postoperative changes in blood pressure (ΔSKB, ΔDKB) with changes in LVMI (ΔLVMI).

Results: The median age of patients was 45.5 ± 7.4 years and the majority were male (63.3%). The mean value of the ΔSBP and ΔDBP were 23 ± 16.6, 7.8 ± 2.8 respectively. The mean ΔLVMI was 9.2 ± 6.7. There was a strong (r=0.836) and significant (p<0.001) positive correlation between SBP change and LVMI change and also there was a moderate (r=0.545) and significant (p<0.001) positive correlation between DBP change and LVMI change.

Conclusions: The findings of the study revealed that the amount of postoperative decrease in systolic and diastolic blood pressure compared to baseline is related to the amount of decrease in left ventricular mass index after KTx.

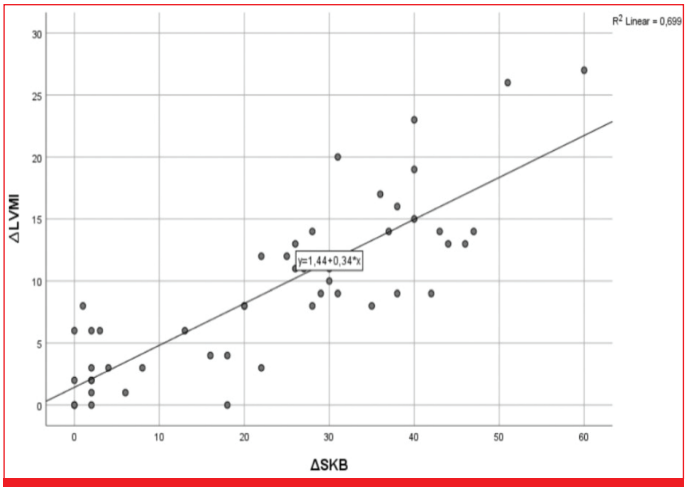

Figure 1. The scatted-dot graph shows correlation of ΔSBP and ΔLVMI. There is a significant relationship between ΔSBP and ΔLVMI (r=0.836, p<0.001).

Table 1. Baseline demographic, clinical, echocardiographic and laboratory features of patients

Variables	Overall population (n=49)
Age, years, mean	45.5 ± 7.4
Gender (male), n (%)	31 (63.3%)
BSA, m^2, mean	1.84 ± 0.24
HT, n (%)	35 (71.4%)
CAD, n (%)	4 (8.2%)
DM, n (%)	7 (14.3%)
CVA, n (%)	2 (4.1%)
PAD, n (%)	2 (4.1%)
Smoking, n (%)	24 (48.9%)
SBP, mmHg, mean	150 ± 28
DBP, mmHg, mean	87 ± 11
LVEF, %, mean	60.3 ± 3.4
LVMI, g/m^2, mean	140 ± 37
Kreatinin, mg/dL, mean	7.1 ± 0.9
BUN, mg/dL, mean	102 ± 13

BSA: Body surface area, HT: Hypertension, CAD: Coronary artery disease, DM: Diabetes mellitus, CVA: Cerebrovascular accident, PAD: Peripheral artery disease, SPB: Systolic blood pressure, DPB: Diastolic blood pressure, LVEF: Left ventricular ejection fraction, LVMI: Left ventricuar mass index, BUN: Blood urea nitrogen.

Table 2. Comparison of the pre-operative and post-operative clinical, echocardiographic and laboratory features

Variables	Pre-operative (n=49)	Post-operative (n=49)	p
Creatinine, mg/dL, mean	7.1 ± 0.9	1.1 ± 0.3	<0.001
BUN, mg/dL, mean	102 ± 13	20.7 ± 6.8	<0.001
LVEF, %, mean	60.3 ± 3.4	61.5 ± 3.6	0.892
LVMI, g/m^2, mean	140 ± 37	131 ± 36	0.040
SBP, mmHg, mean	150 ± 28	127 ± 17	0.029
DBP, mmHg, mean	87 ± 11	79 ± 8	0.046

BUN: Blood urea nitrogen, LVEF: Left ventricular ejection fraction, LVMI: Left ventricuar mass index, SPB: Systolic blood pressure, DPB: Diastolic blood pressure.

Cardiac Imaging / Echocardiography

OP-098

Early echocardiographic changes that may be detected in patients with beta-thalassemia major without cardiac iron deposition

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Background and Aim: One of the main causes of death in patients with beta-thalassemia major (ß-TM) is cardiovascular complications. In addition to cardiac systolic and dia-

stolic dysfunction caused by iron accumulation, arrhythmias, especially atrial fibrillation, are common in this patient group. Atrial cardiomyopathy plays a major role in the development of supraventricular tachyarrhythmias and impaired left ventricular filling pressures. In this pilot study, we compared the left atrial function of ß-TM patients with no myocardial iron deposition on cardiac magnetic resonance imaging (MRI) with normal healthy individuals.

Methods: 22 ß-TM patients who underwent cardiac MRI and showed no myocardial iron deposition and 12, age and sex matched, healthy controls were included in the study. Left ventricular and left atrial dimensions, systolic and diastolic functions were examined by two-dimensional and Doppler transthoracic echocardiography. In addition, left atrial, left and right ventricular strain echocardiography were performed.

Results: Compared to the control group, it was found that the left ventricular ejection fractions of the ß-TM patients were numerically lower, but within the normal limits, and the left ventricular diameters and volumes were higher. MASS-I was higher, left atrial diameter and volume index were higher, mitral valve E/E' ratio was greater and deceleration time was significantly shorter. Right ventricular systolic functions were normal and there was no difference between the two groups. While no difference was observed between the left and right ventricular global longitudinal strain (GLS) values; peak atrial contraction strain (PACS) and active emptying fraction values, which indicate the pump function of the left atrium, were found to be significantly impaired. While the expansion index diminished significantly, peak atrial longitudinal strain (PALS) value was found to have a decreasing trend, although there was no statistically significant difference; which both of them show the reservoir function of the left atrium. There was no difference between the two groups in the PALS-PACS value, which shows the conduit function of the left atrium.

Conclusions: In studies involving ß-TM patients, it has been shown that the deterioration in left atrial strain values is proportional to myocardial iron deposition, occurs long before the LV-GLS values are impaired, and increases the risk of atrial arrhythmia development. In this pilot study, we showed that left atrial dysfunction develops even in ß-TM patients who have not yet determined iron deposition on cardiac MRI. Although clinical studies involving more patients are needed, left atrial deformation parameters may be useful in the early diagnosis of atrial cardiomyopathy. Thus, individuals who appear clinically normal but have a higher risk for cardiac outcomes can be identified, and followed more closely, especially in terms of arrhythmias, and the intensity of chelation therapy can be increased if necessary.

Table 1. Basic demographic characteristics and echocardiographic parameters of beta-TM patients and controls

Parameters	Beta-TM patients (n=22)	Controls (n=12)	p
Age, years	28.68 ± 5.92	30.33 ± 7.22	0.477
Gender (male), n (%)	10 (54.6%)	6 (50.0%)	0.800
Ferritin, ng/mL	1153.23 ± 821.01	72.58 ± 43.29	0.0001
Left ventricular ejection fraction, %	63.77 ± 3.41	67.50 ± 2.81	0.003
Left ventricular global longitudinal strain, %	-20.91 ± 2.36	-22.23 ± 2.62	0.144
Left ventricular end-diastolic diameter, cm	5.12 ± 0.34	4.76 ± 0.44	0.012
Left ventricular end-systolic diameter, cm	3.28 ± 0.31	2.97 ± 0.31	0.009
Left ventricular end-diastolic volume, mL	124.14 ± 16.99	105.58 ± 27.84	0.021
Left ventricular end-systolic volume, mL	44.95 ± 10.12	31.67 ± 10.87	0.001
Left ventricular hypertrophy, n (%) (concentric-hypertrophy/eccentric-hypertrophy/normal)	4 (18.18%)/8 (36.36%)/10 (45.45%)	0 (0%)/0 (0%)/12 (100%)	0.006
MASS-I	110.79 ± 17.45	80.59 ± 9.6	0.0001
Left atrial diameter, cm	3.27 ± 0.42	2.68 ± 0.35	0.0001
Left atrial volume index, mL/m^2	35.49 ± 9.36	17.22 ± 1.79	0.0001
Mitral valve E/A velocity ratio	1.62 ± 0.25	1.64 ± 0.43	0.862
Mitral valve E/E' ratio average	7.56 ± 1.42	6.06 ± 0.95	0.002
Deceleration time, ms	141.05 ± 25.78	194.73 ± 45.78	0.0001
Right ventricular fractional area change (RV-FAC), %	57.51 ± 5.16	57.45 ± 5.16	0.972
TAPSE, cm	2.74 ± 0.26	2.59 ± 0.33	0.172
Right ventricular global longitudinal strain, %	-27.10 ± 4.16	-26.69 ± 3.10	0.767

Table 2. Echocardiographic measurements of the left atrium

	Parameters	Beta-TM patients (n=22)	Controls (n=12)	p
LA dimension-volume	Left atrial diameter, cm	3.27 ± 0.42	2.68 ± 0.35	0.0001
	Left atrial volume index, mL/m^2	35.49 ± 9.36	17.22 ± 1.79	0.0001
LA total function	LA-fractional area change (LA-FAC), %	51.04 ± 4.68	53.88 ± 5.61	0.125
	LA-total emptying volume (LA-T-EV), mL	36.5 ± 12.82	20.58 ± 1.68	0.0001
	LA-total emptying fraction (LA-T-EF), %	59.41 ± 5.93	68.17 ± 5.89	0.0001
LA reservoir function	Peak atrial longitudinal strain (LA-PALS), %	37.86 ± 8.40	42.65 ± 9.87	0.145
	LA expansion index, %	155.86 ± 38.25	222.67 ± 58.28	0.0001
LA pump function	Peak atrial contraction strain (LA-PACS), %	7.18 ± 4.15	12.83 ± 5.65	0.002
	LA-active emptying volume (LA-A-EV), mL	11.09 ± 5.37	6.67 ± 1.67	0.009
	LA-active emptying fraction (LA-A-EF), %	30.55 ± 9.6	39.58 ± 8.74	0.011
	A wave, m/s	0.68 ± 0.12	0.52 ± 0.14	0.001
	E/A	1.62 ± 0.25	1.64 ± 0.43	0.862
LA conduit function	PALS-PACS, %	30.68 ± 8.20	29.82 ± 7.54	0.765
	LA-passive emptying volume (LA-P-EV), mL	25.45 ± 13.63	14.00 ± 2.63	0.0001
	LA-passive emptying fraction (LA-P-EF), %	40.59 ± 9.83	46.25 ± 8.93	0.108
	E wave, m/s	1.09 ± 0.14	0.85 ± 0.12	0.0001
LA stiffness	E/E'	7.56 ± 1.42	6.06 ± 0.95	0.002

Cardiac Imaging / Echocardiography

OP-100

Echocardiographic evaluation of left atrial functions in patients without atrial fibrillation and high CHA2DS2-VASc score

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Background and Aim: The CHA2DS2-VASc score is used to determine the risk of embolism and the need for anticoagulant use in patients with atrial fibrillation (AF). Functional and structural remodeling of the atrium is responsible for AF formation and maintenance. By detecting left atrial functions with echocardiography, atrial dysfunction can be detected early in patients with high CHA2DS2-VASc scores who have not developed AF yet, and its prevalence and morbidity can be reduced by early and effective control of possible risk factors.

Methods: 149 patients without a known or documented diagnosis of AF were included in this study. Patients with severe primary valvular heart disease, acute coronary syndrome, decompensated heart failure (HF), hypertensive pulmonary edema, acute renal failure, thyrotoxicosis were excluded from the study. The mean CHA2DS2-VASc score of the patients was 4.4; 149 patients were divided into two groups of 79 and 70 patients with CHA2DS2-VASc scores of 2-4 and 5-9, respectively. We obtained LA size, areas, LA volumetric measurements [maximal LA volume (LAVmax), pre-A (pre-atrial contraction), LA volume (LAVpre-A), and minimal LA volume (LAVmin)], LA reservoir strain (PALS), LA conduit strain (LAScd), LA contractile strain (LAct), left atrial stiffness (LASt) as well as LV and RV parameters. LA stiffness (LASt) was calculated using the equation: LASt=E/e' ratio/PALS.

Results: A total of 149 patients (mean age: 64.2 ± 12.5 years, 37.5% female) were included in the study. Components of the CHA2DS2-VASc score (congenital HF, hypertension, stroke, diabetes mellitus, vascular disease, and age >65) were more common in the CHA2DS2-VASc (5-9) group, except female gender (p<0.05; Table 1). In the high-score group; LA diameters, volume, volume index (LAVI) and areas, LAVmax, LAVpreA, LAVmin were higher (p<0.05; Table 2). While LA diameters, LA volume, LAVI and LA area were higher in the high-score group than in the low-score group (for all, p<0.05); LA total and active ejection fraction, showing phasic function, were lower in the high-score group (for all, p<0.05; Table 2). In the evaluation of LA strain, PALS, LAScd and LASct were lower in the group with CHA2DS2-VASc (5-9); on the other hand, LASt was higher in the high score group (p<0.001, Table 3). There was a positive high degree correlation between CHA2DS2-VASc score and LA diameter, LA volume and LAst, while a negative moderate correlation was found with PALS and LAScd. Multivariate logistic regression analysis demonstrated that LASt (coeff=4.81, 95% CI: 1.45-15.9, p=0.010) was an independent predictor of high CHA2DS2-VASc score in patients without AF (Table 4).

Conclusions: The present study showed that LA strain values were lower and LASt was more impaired in patients with high CHA2DS2-VASc score without AF. Evaluation of LA structure and functions in addition to the CHA2DS2-VASc score may provide information for atrial remodeling and maybe development of AF, but prospective studies are needed.

Table 1. Baseline demographic characteristics, laboratory parameters, and CHA2DS2-VASc parameters of study groups

	All Population (n=149)	CHA_2DS_2-VASc (2-4) (n=79)	CHA_2DS_2-VASc (5-9) (n=70)	p value
Female gender, n, (%)	56 (%37,5)	29 (%36,7)	27 (%38,6)	0,86
Age (mean years) <65 65-75 >75	64,2 (24-90) 58 (%38,9) 65 (%43,6) 26 (%17,4)	 45 (%57) 28 (%35,4) 6 (%7,6)	 13 (%18,6) 37 (%52,9) 20 (%28,6)	 <0,001
BMI (kg/m2)	28,37(±4,98)	28,7 (±5,5)	27,9(±4,31)	0,29
HT (%)	bu bilgisayar konumuna kaydedildi	62 (%78,5)	66 (%94,3)	0,008
DM (%)	89 (%59,7)	35 (%44,3)	54 (%77,1)	0,000
HF (%)	84 (%56,4)	31 (%39,2)	53 (%75,7)	0,000
CVD (%)	44 (%29,5)	11 (%13,9)	33 (%47,1)	0,000
Vascular Disease (%) CAD (%) PAD (%)	99 (%66,4) 86 (%57,7) 33 (%22.1)	43 (%54,4) 39 (%49,4) 10 (%12,7)	56 (%80,0) 47 (%70) 23 (%32,9)	0,002 0,043 0,006
CHA_2DS_2-VASc	4,4 (2-9)			
Hypo/hyperthyroidism (%)	16 (%10,7)	9 (%11,4)	7 (%10,0)	0,993
Dyslipidemia (%)	35 (%23,5)	16 (20,3)	19 (27,1)	0,34
Smoking (%)	66 (%44,3)	38 (%48,1)	28 (%40,0)	0,32
Alcohol (%)	14 (%9,4)	8 (%10,1)	6 (%8,6)	0,965
Hgb (gr/dl)	13,29 (±2,15)	13,74(±2,13)	12,78(±2,06)	0,001
Creatinin (mg/dl)	1,30 (±1,04)	1,2(±1,1)	1,41(±0,97)	0,032
NT-proBNP (pg/ml)	4592(±8992,93; n=108)	2765,96(±6884; n=53)	6351,96(±10400; n=55)	<0,001

Table 2. Comparison of echocardiographic diameters, volumes, areas, and phasic functions between all population and CHA2DS2-VASc groups

Variables	All population (n=149)	CHA_2DS_2-VASc(2-4) (n=79)	CHA_2DS_2-VASc (5-9) (n=70)	p value
LA volume (ml)	59.59 (±22.69)	55.48(±23.24)	64.24(±21.26)	0.001
LA diameters (mm) -anteroposterior (mm) -LA4C diameter (mm) -LA2C diameter (mm)	 41.51(±5.53) 50.54(±6.88) 50.69(±6.62)	 39.74 (±5.48) 49.31 (±6.74) 49.31(±6.65)	 43.51(±4.91) 51.92(±6.82) 52.24(±6.28)	 <0.001 0.01 0.003
LAVI (ml/m2)	31.69 (±12.52)	29.06(±11.87)	34.65(±12.65)	<0.001
LA4C area (cm2)	18.61(±4.4)	17.75(±4.6)	19.59(±4.03)	0.003
LA2C area(cm2)	18.88(±4.64)	17.88(±4.78)	20.01(±4.24)	<0.001
LAVmaks (ml)	58.85(±23.61)	54.52(±24.22)	63.9(±22.01)	0.006
LAVpreA (ml)	43.64(±21.21)	39.85(±21.02)	48.06(±20.73)	0.009
LAVmin (ml)	27.59(±18.46)	24.08(±17.76)	31.68(±18.57)	0.009
LA total emptying volume(ml)	31.26(±12.0)	30.44(±1.76)	32.22(±9.57)	0.17
LA total emptying fraction (%)	56.14(±18.31)	58.27(±20.54)	53.66(±15.1)	0.02
LA passive emptying volume (ml)	15.21(±7.82)	14.66(±9.1)	15.84(±6.02)	0.17
LA passive emptying fraction (%)	27.31(±15.04)	27.92(±17.95)	26.6(±10.82)	0.18
LA expansion index	161.93(±104.34)	179.29(±115.17)	141.69(±86.72)	0.02
LA conduit volume(ml)	44.53(±27.70)	45.55(±26.72)	43.33(±29.0)	0.49
LA active emptying volume(ml)	16.05(±7.31)	15.77(±7.69)	16.37(±6.89)	0.51
LA active emptying fraction (%)	40.78(±15.14)	43.35(±15.07)	37.77(±14.77)	0.03
LAEF (%)	57.01 (±14.77)	59.62(±14.06)	53.97(±15.11)	0.03

Table 3. Comparison of strain parameters between groups

Variables	All population (n=130)	CHA_2DS_2-VASc (2-4) (n=70)	CHA_2DS_2-VASc (5-9) (n=60)	p value
PALS	25.30(±9.64)	27.69(±9.64)	22.56(±8.97)	0.001
LAScd	12.47(±5.64)	13.95(±6.04)	10.77(±4.64)	0.002
LASct	12.86(± 6.05)	13.8(±5.97)	11.79(±6.01)	0.024
TPLS	395.45(±55.25)	393.25(±53.43)	397.98(±57.61)	0.62
LAst	.56 (± .49)	0.4(±0.34)	0.74(±0.57)	<0.001

Table 4. Univariate and multivariate logistic regression analysis between left atrium echocardiographic parameters and CHA2DS2-VASc score

	Univariate		Multivariate Logistik Regression *	
Variables	OR (%95 CI)	p value	OR (%95 CI)	p value
LA diameter (mm)	1.158 (1.025-1.309)	.019	1.181 (1.052-1.326)	.005
LA volume (mL)	.966 (.904-1.032)	.305	.969 (.941-.997)	.030
LA volume index (mL/m^2)	1.010 (.894-1.141)	.872	-	
PALS	2.807 (.074-106.117)	.578	-	
LASct	.363 (.010-13.749)	.585	-	
LAScd	.349 (.009-13.160)	.570	-	
LAst	5.587 (1.255-24.876)	.024	4.816 (1.451-15.992)	.010

*Logistic regression model was created with Backward Stepwise (Wald) method. OR: Relative risk, 95% CI: Confidence interval

Cardiac Imaging / Echocardiography

OP-101

Evaluation of left atrial volumetric/ mechanical coupling index in hypertrophic cardiomyopathy patients with and without obstructive physiology

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Background and Aim: Hypertrophic cardiomyopathy (HCMP) is characterized by hypertrophy and functional abnormalities of the myocardium. Although left ventricular morphological and functional abnormalities have been investigated in HCMP patients, studies evaluating the left atrial functions are relatively less. Studies showed that measurement of left atrium (LA) functions provides prognostic information and may help clinicians in management of these patients. Left atrial volumetric/mechanical coupling index (LAVMCI) give information about both morphological and hemodynamic functions of LA. We aimed to compare LAVMCI in HCMP patients with and without left ventricular outflow tract (LVOT) obstruction.

Methods: A total of 69 patients with HCMP were included in the study. Patients were divided into two groups; group 0 (n=37) and group 1 (n=32) were composed of patients with left ventricular outflow tract (LVOT) gradient of <30 mmHg, and ≥30 mmHg, respectively. 2-D and tissue Doppler echocardiographic (TDI) assessments of patients were evaluated. LAVMCI was calculated as: ratio of LA volume-index (LAVI) to mitral septal annular TDI-a' wave.

Results: Mean age of the study population was 53.61 ± 15.87 years, BMI was 27.73 ± 5.04 kg/m^2, 40 (58%) of them were male. Patients with obstructive HCMP were older, had higher values of interventricular septum thickness (IVS), LV mass index, LAVMCI and lower values of left ventricular end-diastolic diameter (Table 1). ROC curve analysis demonstrated that LAVMCI value of 5.50 predicted obstructive HCMP with sensitivity of 62.5% and specificity of 75.7% (AUC: 0.769, p<0.001, 95% CI: 0.660-0.878) (Figure 1). Multivariate logistic regression analysis showed that age, IVS and LAVMCI were the independent predictors of obstructive physiology (Table 2).

Conclusions: Hypertrophy of the myocardium which is a characteristic feature of HCMP, is associated with decreased left ventricular (LV) compliance and relaxation with resultant increase in LV filling pressures. Elevated LV pressures are transmitted to LA and cause LA enlargement and dysfunction. Our study showed that patients with obstructive HCMP had worse LAMCI and suggested that LVOT obstruction is one of the factor that further impair the LA functions. Previous studies showed that LA functions had prognostic value in HCMP patients. LA dysfunction has been found to be associated with heart failure, development of atrial fibrillation and major adverse cardiac events. A study found that LA volumes increased and LA ejection fraction decreased in patients with obstructive HCMP compared to patients without obstruction. It is possible that effects of LVOT obstruction further impairs LA functions. LAVMCI is an easily obtainable variable and its predictive value has been shown in other clinical settings including heart failure, stroke, mitral regurgitation and acute coronary syndrome. Our results suggested that obstructive HCMP is associated with worse LA morphological and functional abnormalities.

Table 2. Multivariate logistic regression analysis for the prediction of left ventricular outflow obstruction

	p	OR	95% CI
Age	0.007	1.078	1.021-1.139
IVS	0.022	4.691	1.249-17.617
LAVMCI	0.012	1.288	1.057-1.569

Table 1. Comparison of patients with and without left ventricular obstruction and multivariate logistic regression analysis for the prediction of left ventricular outflow obstruction.

	Group 0 (n=37)	Group 1 (n=32)	p
Age, years	49.51 ± 13.24	58.34 ± 17.49	0.020
Body mass index, kg/m^2	27.64 ± 4.58	27.84 ± 5.59	0.870
Systolic blood pressure, mmHg	126.06 ± 24.80	130.03 ± 23.24	0.515
IVS, cm	2.27 ± 0.60	2.65 ± 0.56	0.010
LVMASS index, g/m^2	209.72 ± 99.81	238.59 ± 77.18	0.029
LVEDD, cm	4.46 ± 0.46	4.17 ± 0.55	0.023
LVESD, cm	2.50 ± 0.42	2.34 ± 0.45	0.149
Ejection fraction, %	55.20 ± 5.05	58.85 ± 7.96	0.142
E/A ratio	1.22 ± 0.42	1.08 ± 0.45	0.255
HR, bpm	73.81 ± 12.90	76.64 ± 11.80	0.400
LAVMCI	4.48 ± 2.54	8.27 ± 5.11	0.001

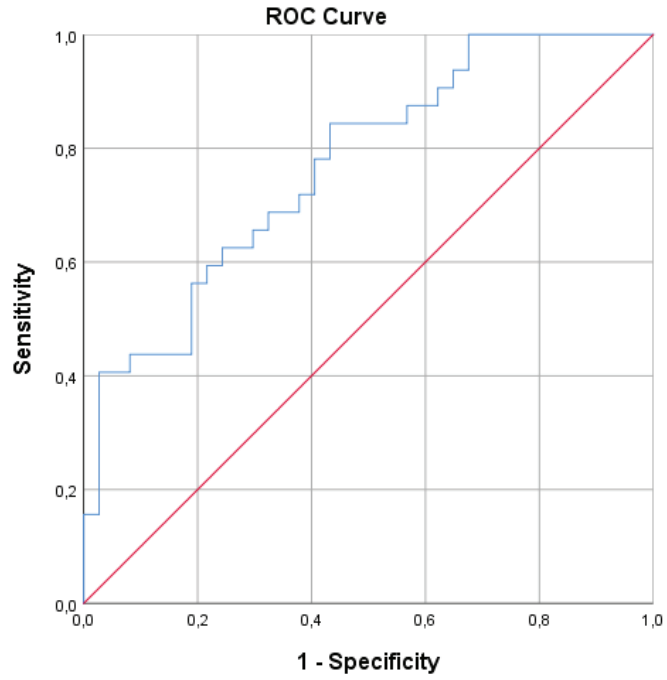

Figure 1. ROC curve analysis of LAVMCI value for prediction of obstructive HCMP.

Heart Valve Diseases

OP-103

Predictive clinical factors of in-hospital systemic embolism in patients with prosthetic valve endocarditis

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Background and Aim: The management of prosthetic valve endocarditis (PVE) includes urgent diagnosis, treatment with antibiotherapy, or surgery and requires the establishment of an endocarditis team. In hospital mortality for patients who suffer from concomitant stroke in PVE was up to 22.8%. We aimed to assess predictors of in hospital systemic embolism in patients with PVE.

Methods: This single-center retrospective study included consecutive patients who previously underwent prosthetic heart valve surgery and were complicated with endocarditis and systemic embolism. 96 consecutive patients with mechanical heart valves and bioprosthetic heart valves were included in the study between 2012 and 2021.

Results: The study included 96 patients (53 males and 43 females; age: 52.4 ± 12.2 years) with PVE. Overall, there were 42 (43.7%) aortic, 47 (48.9%) mitral, 9 (9.5%) aortic and mitral, 1 (1%) tricuspid, and 1 (1%) pulmonary prostheses with PVE, 12 patients underwent redo valve surgery. 86 known patients (98.9%) had bileaflet valves. In the study group, the number of cases of hypertension was 47 (49%), diabetes mellitus was 30 (31.3%), atrial fibrillation was 19 (19.8%), and stroke history was 18 (18.8%). Patients with a stroke history have a significant difference in developing systemic embolism, during hospitalization. The median time after valve surgery was 30.5 (ranging from 12.25 to 84 months). The mean INR on admission was 2.3 ± 0.68. 62 patients (64.6%) had fever on admission, 24 (25%) patients had dyspnea, 17 (17.7%) patients suffered from exhaustion and general condition disorder, 8 (8.3%) patients presented with decompensated heart failure, and 4 (4.1%) patients were admitted with a sternal sternal wound infection with dehiscence. In follow-up, 56 (58.3%) patients showed a regression >50% in the vegetation burden without facing death or with well-managed complications, which was deemed treatment success, and 38 (40%) patients died. Treatment success was found to be a significant protective risk factor for systemic embolism development (p=0.045). The vegetation area was significantly higher in the embolism group (p<0.001). In the univariate logistic regression analysis, stroke history, vegetation area, treatment success, and ESR were significant (p=0.016, p=0.001, p=0.047, p=0.016) (Table 4). Significant univariate regression data were evaluated in the multivariate logistic regression analysis. Only vegetation area was found to be significant again as a risk factor (p=0.037) and had the highest odds ratio (2.34). The ROC curve analysis performed for the vegetation area reveals a cut-off value of 2 cm^2 with 66% sensitivity and 78% specificity.

Conclusions: There is a substantial risk of death and morbidity associated with PVE, and systemic embolism is one of the complications that pose the most significant risk for death. According to our study, having a history of stroke, higher ESR, treatment failure, and vegetation greater than 2.0 cm^2 were risk factors for systemic embolism in PVE.

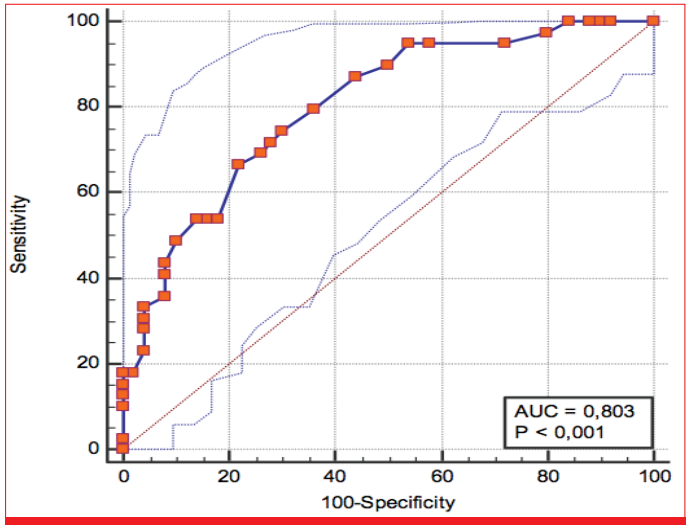

Figure 1. Roc curve analysis of vegetation size

Table 1. The table explains the previous diagnosis, valve types, and clinical features on the admission of the patients.

Variable	All patients (n:96)	Embolism In Hospitalization (n:57)	Embolism In Hospitalization (n:39)	P Value
Age	52,4±12,2	52,1±11,4	52,8±13,4	0,779
Men	53 (55,2)	30 (52,6)	23 (59,0)	0,779
Hypertension	47 (49,0)	27 (47,4)	20 (51,3)	0,706
Diabetes Mellitus	30 (31,3)	15 (26,3)	15 (38,5)	0,207
Stroke History	18 (18,8)	6 (10,5)	12 (30,8)	0,013
TIA History	3 (3,4)	2 (3,5)	1 (3,3)	1,000
Atrial Fibrillation	19 (19,8)	11 (19,3)	8 (20,5)	0,883
Redo Operation	12 (14,6)	6 (11,1)	6 (21,4)	0,322
Fever	62 (64,6)	40 (70,2)	22 (56,4)	0,166
Weakness	10 (10,4)	8 (14,0)	2 (5,1)	0,193
Dyspnea	24 (25,0)	16 (28,1)	8 (20,5)	0,401
Decompansated Heart Failure	8 (8,3)	7 (12,3)	1 (2,6)	0,137
Septic Embolism On Admisssion	3 (3,1)	2 3,5 ()	1 (2,6)	1,000
Wound Site Infection	3 (3,1)	2 (3,5)	1 (2,6)	1,000
Sternal Dehiscence	1 (1,0)	0 (0,0)	1 (2,6)	0,406
NYHA				
1	40 (46,0%)	28 (49,1%)	12 (40,0%)	0,438
2	34 (39,1%)	19 (33,3%)	15 (50,0%)	
3	12 (13,8%)	9 (15,8%)	3 (10,0%)	
4	1 (1,1%)	1 (1,8%)	0 (0,0%)	
Valve				
AVR	51 (58,6)	34 (59,6)	17 (56,7)	0,788
MVR	56 (64,4)	38 (66,7)	18 (60,0)	0,537
TVR	1 (1,1)	1 (1,8)	0 (0,0)	1,000
PVR	1 (1,1)	1 (1,8)	0 (0,0)	1,000
ETSV	30,5 [12,25-84]	36 [14-90]	25 [5-81]	0,366

TIA: Transient Ischemic Attack, NYHA: New York Heart Association (NYHA) Functional Classification, AVR: Aortic Valve Replacement, MVR: Mitral Valve Replacement, TVR: Tricuspid Valve Replacement, PVR: Pulmonary Valve Replacement, ETSV: Elapsed Time Since Valve Surgery

Table 2. The table reveals blood tests of the patients.

Variable	All patients (n:96)	Embolism In Hospitalization (n:57)	Embolism In Hospitalization (n:39)	P Value
HG	10 [9-11,1]	10 [8,95-11]	10 [9,1-11,5]	0,429
WBC (Min-Maks)	13,6±5,2 (3,9-30)	14,0±5,1 (4,8-28)	13,0±5,5 (3,9-30)	0,391
PLT	230 [200-280]	230 [200-275,5]	225 [188,5-301,25]	0,986
ESR (Min-Maks)	67,8±28,2 (11-126)	61,6±28,5 (11-116)	82,3±22,1 (45-126)	0,010
Follow-up ESR	27 [15-68]	22 [15-85,5]	29 [14,75-66,5]	0,785
CRP	6 [2-20,5]	5,5 [1,04-22,25]	8 [2-18,5]	0,606
Follow-up CRP	115 [100-134]	112 [100-130]	115 [100-147,75]	0,434
Glucose	43 [32-56]	42 [32,5-55]	44,5 [29,75-65,25]	0,535
BUN	27 [15-68]	22 [15-85,5]	29 [14,75-66,5]	0,785
Creatinine	0,93 [0,8-1,19]	0,9 [0,72-1,20]	1 [0,8-1,18]	0,488
Sodium	135 [133-138]	135 [133-138]	135 [133-137]	0,559
Potassium	4 [3,8-4,33]	4,05 [3,8-4,45]	4 [3,85-4,2]	0,329
Culture				
Positive	40 (47,1)	26 (45,6)	14 (50,0)	0,703
Negative	45 (52,9)	31 (54,4)	14 (50,0)	
Candida spp.	6 (7,1)	4 (7,0)	2 (7,1)	1,000
Staf Spp.	27 (31,8)	18 (31,6)	9 (32,1)	0,958
Troponin	0,1 [0-0,39]	0,02 [0-0,13]	0,22 [0,03-0,78]	<0,001
Procalsitonin	2,69 [0,31-4595]	2 [0,319-4200]	4,2 [0,30-14015]	0,394
Follow-up HGB	9,75 [7,45-10,68]	9,8 [6,9-11,5]	9,7 [8,1-10,65]	0,834
Follow-up PLT	191,9±126,5 (0-461)	188,7±132,8 (0-461)	197,2±122,8 (0-403)	0,877
INR On Admission	2,3 [1,6-2,98]	2,7 [1,72-3,1]	2,05 [1,4-2,56]	0,026
Blood Type				
0	20 (25,3)	13 (25,5)	7 (25,0)	0,962
A	37 (46,8)	26 (51,0)	11 (39,3)	0,319
B	17 (21,5)	10 (19,6)	7 (25,0)	0,577
AB	5 (6,3)	2 (3,9)	3 (10,7)	0,340
RH				
Negative	12 (15,4)	8 (15,7)	4 (14,8)	0,497
Positive	66 (84,6)	43 (84,3)	23 (85,2)	

HGB, hemoglobin; WBC, white blood cell; PLT, platelet; CRP, C-reactive protein; ESR; Erythrocyte Sedimentation Rate, BUN; Blood Urea Nitrogen; INR, International normalized ratio.

Table 3. The table explains the echocardiography findings, treatment options and the consequences.

Variable	All patients (n:96)	No Embolism In Hospitalization (n:57)	Embolism In Hospitalization (n:39)	P Value
Decreased Valve Area	1,6 [1,1-1,9]	1,6 [1,125-1,98]	1,8 [1-1,9]	0,634
Leaflet				1.000
Monoleaflet	1 (1)	1 (1,8)	0 (0,0)	
Bileaflet	95 (99)	56 (98,2)	39 (100)	
Vegetation Size (cm^2)	1,5 [1-2,5]	1,25 [0,9-2]	2,4 [1,5-3,2]	<0,001
Surgery After Antibiotherapy	27 (32,9)	16 (29,6)	11 (39,3)	0,378
Time of SurgerySurgery				0,703
No surgery	65 (67,7)	37 (64,9)	28 (71,8)	
Early Surgery	7 (7,3)	4 (7,0)	3 (7,7)	
Delayed Surgery	24 (25,0)	16 (28,1)	8 (20,5)	
Antibiotics Amount	2 [2-3]	2 [2-3]	2 [0-3]	0,119
Antiobiotherapy Length (Day)	21 [14-39,75]	21 [14-35]	22 [14-42]	0,383
Use of Unfractioned Heparin	20 (23,0)	13 (22,8)	7 (23,3)	0,956
Exitus	38 (40,0)	18 (32,1)	20 (51,3)	0,061
Treatment Success	56 (58,3)	38 (66,7)	18 (46,2)	0,045

Table 4. Univariate logistic regression analysis shows the factors that increase systemic embolism risk during hospitalization.

Variable	P Value	Oddss Ratio	95,0% CI Min-Maks
Age	0,776	1,005	0,972-1,039
Men	0,540	1,294	0,568- 2,946
Hypertension	0,706	1,170	0,518- 2,643
Diabetes Mellitus	0,209	1,750	0,730- 4,193
Atrial Fibrillation	0,883	1,079	0,390- 2,988
Stroke History	0,016	3,778	1,276- 11,184
TIA History	0,966	0,948	0,082- 10,904
INR on Admission	0,071	0,682	0,450- 1,033
AB Length (Day)	0,237	1,020	0,987- 1,053
Use of UFH	0,956	1,030	0,361- 2,938
Decompensated HF	0,125	0,188	0,022- 1,593
Infected Valve (AVR)	0,788	0,885	0,361- 2,165
Infected Valve (MVR)	0,538	0,750	0,300- 1,872
Decreased Valve Area	0,255	0,550	0,197-1,539
Vegetation Size	<0,001	3,800	2,068- 6,981
Dsypnea	0,403	0,661	0,251- 1,742
Fever	0,168	0,550	0,235- 1,287
Weakness	0,178	0,331	0,066- 1,652
ETSVS (months)	0,521	0,998	0,992- 1,004
Early Surgery	0,991	0,991	0,205- 4,709
Late Surgery	0,407	0,661	0,248- 1,761
Treatment Success	0,047	0,429	0,186- 0,989
Staf spp.	0,958	1,026	0,389- 2,707
Candida spp.	0,983	1,019	0,175- 5,93
HG	0,337	1,154	0,862- 1,545
WBC	0,388	0,962	0,881- 1,051
PLT	0,816	1,001	0,995- 1,006
ESR	0,016	1,031	1,006- 1,056
Follow-up ESR	0,147	1,014	0,995- 1,034
CRP	0,824	0,999	0,992- 1,006
Follow up CRP	0,705	1,002	0,992- 1,012
Troponin	0,989	0,999	0,870- 1,147

TIA: Transient Ischemic Attack, NYHA: New York Heart Association (NYHA) Functional Classification, AVR: Aortic Valve Replacement, MVR: Mitral Valve Replacement, TVR: Tricuspid Valve Replacement, PVR: Pulmonary Valve Replacement, ETSV: Elapsed Time Since Valve Surgery, HF: Heart Failure, AB: Antibiotherapy, UFH: Unfractioned Heparin

Table 5. Multivariate logistic regression analysis with the Hosmer and Lemeshow finds vegetation size is only significant factor for systemic embolism during hospitalization.

Variable	P Value	Oddss Ratio	95,0% CI Min-Max
Vegetation Size (cm^2)	0,037	2,340	1,052-5,204
Treatment Failure	0,730	1,323	0,270-6,478
ESR	0,126	1,024	0,993-1,055
Stroke History	0,722	1,427	0,202-10,10

Heart Valve Diseases

OP-104

Clinical, microbiological, and imaging correlates of in hospital mortality in infective endocarditis: A single-center study

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Background and Aim: Infective endocarditis (IE) is a life-threatening condition with diverse clinical presentations and outcomes. Numerous factors contribute to this continually poor prognosis, including an increasing proportion of older patients with more severe disease, changing epidemiological profiles, antibiotherapy resistance, and an increase in patients with infections related to prosthetic valves or devices.

Methods: We conducted a retrospective analysis of 34 consecutive patients with vegetation on transesophageal echocardiography at our institution. The patients were divided into two groups: those who died during in hospital follow-up and the others. Clinical, microbiological, and biochemical data were collected and analyzed for their significance in relation to in hospital mortality. Imaging findings including echocardiograms were also evaluated for their predictive value.

Results: There was no significant age or gender difference between the two groups (Table 1). A significant number of patients with vegetation on native valve who had in hospital follow-up died (p=0.084) (Table 2). There were no patients who died during in hospital follow-up among those presenting with fever (p=0.033). This was attributed to early antibiotic therapy and focus investigation in patients presenting with fever. The in hospital mortality rate was higher in patients with acute kidney failure (AKF) during hospitalization (p<0.001). This was attributed to the fatal nature of renal failure. Surprisingly, patients presenting with cerebrovascular disease (CVD) had a lower presence in the group of patients who died during in hospital follow-up but it was not statistically significant (p=0.244). This suggests the potential therapeutic contribution of anticoagulation in cases where the differentiation between vegetation and thrombus was not clearly defined (Table 3). The hospital mortality was higher in patients with initial symptom of shortness of breath (p=0.037) (Table 4). This was attributed to valve diseases caused by vegetation, which were thought to lead to symptoms of heart failure. Elevated creatinine (p=0.008), BNP (p=0.028) and low platelet (p=0.007) were associated with in hospital mortality (Table 5). In patients treated with ceftriaxone, in hospital mortality was less (p=0.006). However, it was found that surgery did not reduce in hospital mortality (p=1.000) (Table 6). This was attributed to the patients requiring surgery being associated with more severe disease.

Conclusions: The presence of vegetation increases the risks of CVD, septic emboli, glomerulonephritis and inflammatory processes. As a result, cases of infective endocarditis with observed vegetation tend to have a more fatal course. In our study, we aimed to identify indicators of in-hospital mortality in patients with vegetation. This will enable us to detect in hospital mortality in advance, update treatment modalities, and take necessary precautions.

Table 1. There was no significant age or gender difference between the two groups.

	T1-ex (n=17)	T2 (n=17)	P value
Age	65 ± 14	58 ± 17	0.227
Gender, Male	10 (58.8)	12 (70.6)	0.473
Hypertension	7 (41.2)	10 (58.8)	0.303
Diabetes	4(23.5)	4(23.5)	1.000
Hypertension	1(5.9)	3(17.6)	0.287
Smoking	0 (0)	2(11.8)	0.145
COPD	2 (11.8)	0 (0)	0.145
Dementia	1 (5.9)	1 (5.9)	1.000
Malignancy	6 (35.3)	3 (17.6)	0.244
Coronary Artery Disease	8 (47.1)	6 (35.3)	0.486
Peripheral Artery Disease	1 (5.9)	3 (17.6)	0.287
Chronic Kidney Failure	9 (52.9)	5 (29.4)	0.163
Cerebrovascular Disease	6(35.3)	5 (29.4)	0.714
CABG history	3 (17.6)	5 (29.4)	0.419
Pacemaker history	2 (11.8)	3 (17.6)	0.628
MVR	4 (23.5)	6 (35.3)	0.452

Table 2. Vegetation locations. A significant number of patients with vegetation on native valve who had in-hospital follow-up died (p=0.084).

	T1-ex (n=17)	T2 (n=17)	P value
VEGETATION LOCATION			0.105
ABOVE PROSTHETIC VALVE	4 (23.5)	7 (41.2)	0.271
NATIVE VALVE	12 (70.6)	7 (41.2)	0.084
LEAD	0 (0)	3 (17.6)	0.070
RIGHT HEART CAVITIES	1 (5.9)	0 (0)	0.310

Table 3. Hospitalization reasons. There were no patients who died during in hospital follow-up among those presenting with fever (p=0.033). This was attributed to early antibiotic therapy and focus investigation in patients presenting with fever. The in hospital mortality rate was higher in patients with acute kidney failure (AKF) during hospitalization (p<0.001). This was attributed to the fatal nature of renal failure. Surprisingly, patients presenting with cerebrovascular disease (CVD) had a lower presence in the group of patients who died during in hospital follow-up but it was not statistically significant (p=0.244). This suggests the potential therapeutic contribution of anticoagulation in cases where the differentiation between vegetation and thrombus was not clearly defined.

	T1-ex (n=17)	T2 (n=17)	P value
HOSPITALIZATION REASON			
CVD	3 (17.6)	6 (35.3)	0.244
PNEUMONIA	2 (11.8)	3 (17.6)	0.628
AKF	6 (35.3)	1 (5.9)	0.034
RESPIRATORY FAILURE	1 (5.9)	1 (5.9)	1.000
PRIMARY CARDIOLOGY	3 (17.6)	2 (11.8)	0.628
FEVER ETIOLOGY	0 (0)	4 (23.5)	0.033
POST-CT FEVER	2 (11.8)	0 (0)	0.145
AKF DURING HOSPITALIZATION	12 (70.6)	2 (11.8)	<0.001

Table 5. Laboratory parameters. This was attributed to valve diseases caused by vegetation, which were thought to lead to symptoms of heart failure. Elevated creatinine (p=0.008), BNP (p=0.028) and low platelet (p=0.007) were associated with in hospital mortality.

	T1-ex (n=17)	T2 (n=17)	P value
WBC, cells/µL	12.88 ± 6.46	10.58 ± 4.95	0.260
HGB, g/dL	9.43 ± 1.35	9.77 ± 1.59	0.509
HCT	28.83 ± 4.35	29.63 ± 5.75	0.655
LNF, cells/µL	1.65 ± 1.11	1.26 ± 0.48	0.198
MONOCYTE	0.61 (0.42 – 0.78)	0.41 (0.39 – 0.68)	0.194
NEUTROPHIL	10.51 ± 5.88	8.73 ± 5.04	0.359
MPV	10.07 ± 164	8.72 ± 1.52	0.021
RDW	15.90 (14.80 – 16.30)	16.10 (15.55 – 17.15)	0.332
PCT	0.20 ± 0.17	0.20 ± 0.08	0.983
PLT	155 (119 - 217)	255 (196 - 311)	0.007
KRE	2.10 ± 1.32	1.12 ± 0.44	0.008
UREA	80 ± 35	46 ± 22	0.003
GLUCOSE	117 ± 41	128 ± 49	0.470
AST	24 (15 - 58)	38 (21 - 60)	0.367
ALT	24 (18 - 90)	33 (20 - 59)	0.986
TSH	1.44 ± 1.31	0.87 ± 0.80	0.268
T4	1.13 ± 0.35	1.11 ± 0.17	0.886
TROPONINE	290 (54 - 1006)	55 (32 - 500)	0.244
BNP	2171 (1020 - 21816)	392 (171 - 879)	0.028
D-DIMER	1650 (813 - 4500)	860 (560 - 3180)	0.566
ALBUMIN	28.44 ± 6.03	30.84 ± 5.12	0.246
PROCALCITONIN	1.36 (0.78 – 7.36)	0.26 (0.10 – 0.87)	0.021
SEDIMENTATION	56 ± 37	72 ± 33	0.238
INR	1.25 (1.18 – 1.30)	1.30 (1.22 – 1.96)	0.361

Table 4. Symptoms in application. The hospital mortality was higher in patients with initial symptom of shortness of breath (p=0.037).

	T1-ex (n=17)	T2 (n=17)	P value
SYMPTOMS IN APPLICATION			
WEIGHT LOSS	4 (23.5)	3 (17.6)	0.671
DYSPNEA	10 (58.8)	4 (23.5)	0.037
COUGH	6 (35.3)	3(17.6)	0.244
STROKE	4 (23.5)	6 (35.3)	0.452

Table 6. Treatment. In patients treated with ceftriaxone, in hospital mortality was less (p=0.006). However, surprisingly, it was found that surgery did not reduce in hospital mortality (p=1.000). This was attributed to the patients requiring surgery being associated with more severe disease.

	T1-ex (n=17)	T2 (n=17)	P value
CEPHALOSPORIN	4 (23.5)	12 (70.6)	0.006
RIFAMPICINE	6 (35.3)	6 (35.3)	1.000
VANKOMYCINE	12 (70.6)	9 (52.9)	0.290
GENTAMYCINE	5 (29.4)	3 (17.6)	0.419
MEROPENEM	8 (47.1)	5 (29.4)	0.290
TEICOPLANIN	1 (5.9)	2 (11.8)	0.545
PIPERASILIN/TAZOBACTAM	2 (11.8)	2 (11.8)	1.000
DAPTOMYCINE	3 (17.6)	2 (11.8)	0.628
SURGERY	5 (29.4)	5 (29.4)	1.000

Heart Valve Diseases

OP-105

Biological heart valves versus mechanical heart valves with low-dose warfarin during pregnancy

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Background and Aim: Prosthetic heart valves (PHVs), especially mechanical ones, are highly thrombogenic, and pregnancy-related procoagulant status increases the risk of PHV thrombosis. Current evidence reports that pregnant women with effective INR under low dose warfarin may have optimal maternal and fetal outcomes. While current European guidelines advise to consider implantation of a biological PHV in women with a pregnancy wish, the underlying evidence is limited. The high deterioration rate of biological PHV at young age poses the woman at risk of going through pregnancy with a stenotic or regurgitant PHV. The goal of this study was to evaluate biological heart valves versus

mechanical heart valves with effective anticoagulation with low-dose warfarin during pregnancy.

Methods: The outcomes of anticoagulation regimens were assessed retrospectively in pregnant women (71 women; 71 pregnancies) with PHVs. The study population was divided into two groups according to type of valve; mechanical heart valve with low-dose warfarin (≤5 mg/day) throughout pregnancy (group 1), biological heart valve prosthesis during pregnancy (group 2). Fetal complications included preterm birth, low birth weight, stillbirth, abortion, and congenital anomaly. Besides, maternal outcomes included mortality, valve thrombosis requring therapy, major bleeding, pre-eclampsia, stroke, and endocarditis.

Results: Of the 71 pregnancies, 29 were included in group 1 and 42 were included in group 2. In terms of basic demographic and echocardiographic data, there was no significant difference except valve degeneration. PHV degeneration was statistically significantly higher in group 2 (0 vs. 52.4%, p<0.001). While vaginal delivery was higher in group 2 (3.4% vs. 21.4%, p=0.032), C/S delivery was statistically significantly higher in group 1 (82.6% vs. 59.5%, p=0.037). There was no difference between the two groups in terms of poor fetal outcomes, including preterm birth, low birth weight, abortion, and congenital anomaly. There was no statistical difference between the two groups in terms of major bleeding, endocarditis, valve thrombosis requiring therapy, pre-eclampsia, stroke, maternal mortality.

Conclusions: The current data suggest that pregnant patients with mechanical PHVs on a low-dose warfarin (≤5 mg/day) regimen with therapeutic international normalized ratio levels have maternal-fetal outcomes that are as acceptable as biological valve pregnancy outcomes.

Table 1. Baseline characteristics of pregnancies in patients with prosthetic heart valves

Parameters	Mechanical valve (n=29)	Bioprosthetic valve (n=42)	p
Age, years	29.1 ± 4.6	27.9 ± 5	0.290
ETSVS (months)	62.6 ± 37	70.9 ± 57.6	0.495
BMI, kg/m^2	25.1 ± 2.6	25.6 ± 1.1	0.484
Diabetes mellitus, n (%)	1 (3.4)	2 (4.8)	0.787
Hypertension, n (%)	1 (3.4)	2 (4.8)	0.787
Hyperthyroidism, n (%)	1 (3.4)	1 (2.4)	0.789
Hypothyroidism, n (%)	3 (10.3)	1 (2.4)	0.153
Chronic anemia, n (%)	1 (3.4)	4 (9.5)	0.325
Smoking, n (%)	1 (3.4)	1 (2.4)	0.789
Heart failure, n (%)	2 (6.9)	0	0.084
Atrial fibrillation, n (%)	5 (17.2)	8 (19)	0.847
Valve Position, n (%)			
AVR	6 (20.7)	6 (14.3)	0.479
MVR	14 (48.3)	22 (52.4)	0.734
TVR	2 (6.9)	1 (2.4)	0.353
PVR	0	5 (11.6)	0.054
AVR + MVR	7 (24.1)	8 (19)	0.606
Valve degeneration, n (%)	0	22 (52.4)	<0.001
Previous history of PVT, n (%)	3 (10.3)	1 (2.4)	0.153
Paravalvular leak, n (%)	2 (6.9)	2 (4.8)	0.701
Underlying heart disease			
Rheumatic valve disease	21 (72.4)	31 (73.8)	0.896
ToF	0	4 (9.5)	0.087
Pulmonary stenosis	0	1 (2.4)	0.403
Marfan	1 (3.4)	4 (9.5)	0.325
Other	7 (24.1)	2 (4.8)	0.016
Drugs, n (%)			
ASA 100 mg	5 (17.2)	16 (38.1)	0.058
Beta-blocker	7 (24.1)	8 (19)	0.606

Table 2. Outcomes of pregnancies in the whole series

Variables	Mechanical valve (n=29)	Bioprosthetic valve (n=42)	p
Termination time of pregnancy (weeks)	37.3 ± 2.3	37.5 ± 1.7	0.704
Vaginal delivery, n (%)	1 (3.4)	9 (21.4)	0.032
Caesarian section, n (%)	24 (82.6)	25 (59.5)	0.037
Healthy neonates, n (%)	26 (89.7)	36 (85.7)	0.624
Preterm birth, n (%)	3 (10.3)	6 (14.3)	0.624
Low birth weight, n (%)	3 (10.3)	3 (7.1)	0.634
Abortion, n (%)	5 (17.2)	7 (16.7)	0.949
Stillbirth, n (%)	0	0	-
Congenital fetal abnormalities, n (%)			
-Intrauterine growth retardation	1 (3.4)	0	0.226
-Bicuspid aortic valve	0	1 (2.4)	0.403
-Nasal hipoplasi	1 (3.4)	0	0.226

Table 3. Comparison of feto-maternal complications and pregnancy outcomes between bioprosthetic valve and mechanical prosthetic valve with low dose warfarin

Variables	Mechanical valve (n=29)	Bioprosthetic valve (n=42)	p
Major bleeding, n (%)	1 (3.4)	2 (4.8)	0.787
Endocarditis, n (%)	1 (3.4)	0	0.226
PVT requring therapy, n (%)	1 (3.4)	2 (4.8)	0.787
Preeclampsia, n (%)	0	1 (2.4)	0.403
Stroke, n (%)	0	1 (2.4)	0.403
Maternal mortality, n (%)	0	1 (2.4)	0.403

Heart Valve Diseases

OP-106

The predictors of embolic events in left-sided infective endocarditis

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Background and Aim: Infective endocarditis (IE) is a potentially life-threatening clinical entity with an annual mortality rate of up to 40%. IE is often accompanied by various complications that contribute to morbidity and mortality, primarily cerebral and systemic embolic events (EEs). Although a vegetation size greater than 10 mm is generally recommended as an optimal cut-off for estimating the risk of EEs, it has substantial potential for selection bias. Previous literature on embolic risk in patients with large vegetation has given you varied and conflicting results for vegetation size. Some studies have suggested that the risk of EEs is not increased with larger vegetation sizes. This retrospective study aimed to evaluate the relationship between EEs and clinical, microbiological parameters, and echocardiographic parameters including vegetation size.

Methods: We retrospectively reviewed patients with IE who were admitted to a single institution between November 2011 and August 2018. A total of 235 patients (mean age: 56.0 ± 15.8 years; males: 140) with IE were included in the study. Transesophageal echocardiography detected vegetation in all patients. The study population was divided into two groups based on EEs. The primary endpoint was at least one EE, including coronary, cerebral, splenic, mesenteric, and peripheral artery embolism.

Results: During the hospitalization period, 105 (44.6%) patients had at least one documented EE. Comparison of demographic, echocardiographic, microbiological and clinical parameters between IE patients with and without EEs is included in Table 1. In the ROC curve analysis, vegetation size higher than 13.5 mm predicted the presence of EEs with a sensitivity of 84.8% and a specificity of 63.8% (AUC: 0.780, 95% CI: 0.722-0.839, p<0.001) (Figure 1). The study population

was divided into two groups using a cut-off vegetation size of 13.5 mm. All demographic, echocardiographic and electrocardiographic parameters were compared between these subgroups. The prevalence of EEs was significantly higher in patients with vegetation size of >13.5 mm (65.4 vs. 16.2%; p<0.001), akut kidney injury (27.9 vs. 16.2, p=0.034), newly developed heart failure (36 vs. 13.1%, p<0.001), septic shock (22.8 vs. 9.1%, p=0.006). Moreover, there was no significant relationship between in hospital mortality and vegetation size greater than 13.5 mm. Vegetation size (OR: 9.614, 95% CI: 4.539-20.363), *Staphylococcus aureus* (OR: 2.835, 95% CI: 1.295-6.203), syncope (OR: 4.535, 95% CI: 1.802-11.411), and moderate-severe valve dysfunction (OR: 2.301, 95% CI: 1.089-4.859) were independent risk factors for EEs.

Conclusions: Current data suggest that a vegetation size greater than 13.5 mm as independent risk factors for systemic or cerebral embolism. Hence, early surgery may be considered to prevent EEs for this group.

Table 1. Comparison of the demographic, echocardiographic and clinical characteristics of IE patients with and without embolic events.

Parameters	Embolic event (-) (n=130)	Embolic event (+) (n=105)	p
Age, years	53.3 ± 14.9	59.3 ± 16.4	0.004
Gender (male), n (%)	83 (63.8)	57 (54.3)	0.138
Diabetes mellitus, n (%)	26 (20)	29 (27.6)	0.170
CKD, n (%)	27 (20.8)	12 (11.4)	0.056
AF, n (%)	22 (16.9)	21 (20.0)	0.544
White blood cell count, 10^9/L	10.7 (8.61-14.07)	12.5 (9.85-15.56)	0.003
CRP, mg/dL	58.9 (32.1-132)	78.1 (28.5-179.6)	0.049
ESR, mm/hour	60 (38-79)	74 (57-88)	0.002
Troponin T, ng/mL	0.23 (0.03-0.57)	0.42 (0.1-1.1)	0.001
Procalcitonin, ng/mL	1.06 (0.22-5.67)	3.2 (1.02-16.3)	<0.001
LV ejection fraction, %	60 (55-60)	55 (55-60)	0.003
Infected valve location, n (%)			
Aortic	49 (37.7)	41 (39.0)	0.832
Mitral	60 (46.2)	64 (61.0)	0.024
Infected valve type, n (%)			
Native	102 (78.5)	76 (72.4)	0.280
Bioprosthetic	5 (3.8)	7 (6.7)	0.329
Mechanical prosthetic	22 (16.9)	25 (23.8)	0.189
Size of vegetation, mm	11 (9-16)	19 (15-23)	<0.001
Rupture of chordae, n (%)	19 (14.6)	10 (9.5)	0.238
Leaflet perforation, n (%)	33 (25.4)	23 (21.9)	0.534
Pseudoaneurysms or fistula, n (%)	6 (4.6)	15 (14.3)	0.010
Newly developed moderate or severe regurgitation, n (%)	69 (53.1)	88 (83.8)	<0.001
Causative organism, n (%)			
-*Staphylococcus aureus*	18 (13.8)	37 (35.2)	<0.001
-*Staphylococcus epidermidis*	4 (3.1)	7 (6.7)	0.163
-CNS	21 (16.2)	16 (15.2)	0.848
-Group B streptococcus	10 (7.7)	5 (4.8)	0.361
-*Brucella melitensis*	1 (0.8)	1 (1.0)	0.695
-*Candida albicans*	5 (3.8)	4 (3.8)	0.631
-HACEK	4 (3.1)	4 (3.8)	0.516
-Other organism	25 (19.2)	18 (17.1)	0.681
Surgical treatment, n (%)	97 (74.6)	67 (63.8)	0.073
Medical therapy, n (%)	33 (25.4)	38 (36.2)	0.073
Fatal ventricular arrhythmia, n (%)	10 (7.7)	13 (12.4)	0.229
AKI, n (%)	25 (19.2)	29 (27.6)	0.129
Newly developed HF, n (%)	17 (13.1)	45 (42.9)	<0.001
Septic shock, n (%)	13 (10.0)	27 (25.7)	0.001
3-month death, n (%)	29 (22.3)	38 (36.2)	0.019

AF: Atrial fibrillation, AKI: Acute kidney injury, CKD: Chronic kidney disease, CNS: Coagulase-negative staphylococci, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate, HACEK: Haemophilus species, Aggregatibacter actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae, LV: Left ventricle.

Table 2. Multiple regression analysis showing independent predictors of embolic event in IE patients

Parameters	OR	95% CI	p
Diabetes mellitus	1.768	0.831-3.763	0.139
Vegetation size	9.614	4.539-20.363	<0.001
Staphylococcus aureus	2.835	1.295-6.203	0.009
Moderate-severe valve dysfunction	2.301	1.089-4.859	0.029

Table 3. Comparison of demographic, echocardiographic and clinical features of IE patients with low and high vegetation size

Variables	Vegetation size >13.5 mm (n=136)	Vegetation size ≤13.5 mm (n=99)	p
Age, years	57.7 ± 16.4	53.7 ± 14.8	0.055
White blood cell count, 10⁹/L	11.2 (9.76-15.46)	11.2 (8.9-14.3)	0.124
Hemoglobin, g/dL	9.6 (8.6-10.65)	10.14 (9.1-11.7)	0.010
CRP, mg/dL	76.8 (31.5-161.2)	55.09 (32.1-125.4)	0.116
Troponin T, ng/mL	0.41 (0.09-0.95)	0.17 (0.03-0.57)	0.015
Leaflet perforation, n (%)	41 (30.1)	15 (15.2)	0.008
Pseudoaneurysms or fistula, n (%)	17 (12.5)	4 (4.0)	0.025
Newly developed moderate or severe regurgitation, n (%)	113 (83.1)	44 (44.4)	<0.001
Embolic event, n (%)	89 (65.4)	16 (16.2)	<0.001
Surgical treatment	92 (67.6)	72 (72.7)	0.402
Medical therapy	44 (32.4)	27 (27.3)	0.402
Fatal ventricular arrhythmia, n (%)	16 (11.8)	7 (7.1)	0.232
AKI, n (%)	38 (27.9)	16 (16.2)	0.034
Newly developed HF, n (%)	49 (36.0)	13 (13.1)	<0.001
Septic shock, n (%)	31 (22.8)	9 (9.1)	0.006
3-month death, n (%)	43 (31.6)	24 (24.2)	0.216

AKI: Acute kidney injury, CRP: C-reactive protein.

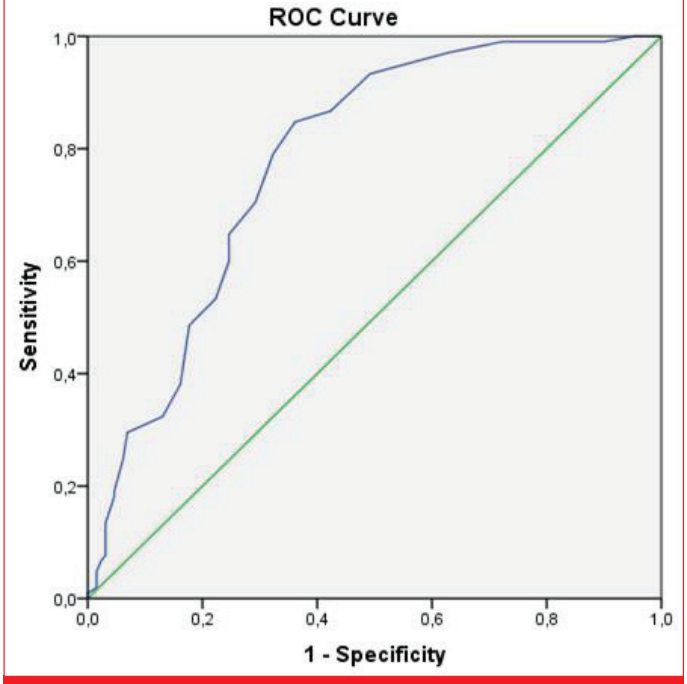

Figure 1.

Heart Valve Diseases

OP-107

Prognostic impact and improvement of mitral and tricuspid regurgitation in patients undergoing transcatheter aortic valve implantation: A retrospective observational study

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Background and Aim: The relationship between concomitant mitral regurgitation (MR) and tricuspid regurgitation (TR) and prognosis in patients undergoing transcatheter aortic valve implantation (TAVI) remains controversial. The aim of our study is to evaluate the changes in MR and TR severity, identify predictors of change, and assess the relationship between the severity of MR and TR and both pre-procedural and post-procedural survival in patients who underwent successful TAVI.

Methods: The study was designed as a single-center, retrospective, observational study and ethical approval was obtained. Retrospective analysis was performed on echocardiographic parameters of 183 patients who underwent successful TAVI between December 2012 and October 2021 in the pre-procedural, early-term (0-3 months) post-procedural and late-term (3-12 months) post-procedural period. Patients were categorized as having no/mild MR (120, 66.3%) or moderate/severe MR (61, 33.7%), and as having no/mild TR (131, 72%) or moderate/severe TR (51, 28%).The primary endpoint was defined as a composite of hospitalization due to heart failure and all-cause mortality.

Results: The median follow-up duration was 2.26 years, during which 68 patients (37.2%) reached the primary endpoint. In the no/mild MR group, the primary endpoint was reached in 37 patients (30.8%), while in the moderate/severe MR group, 30 patients (49.2%) reached the primary endpoint (p=0.02). Among patients in the no/mild TR group, 45 (34.4%) reached the primary endpoint, while 23 (45.1%) patients in the moderate/severe TR group reached the primary endpoint (p=0.23). Adjusted analyses did not show a significant association between the severity of pre-procedural MR or TR and the primary endpoint (HR: 1.75, 95% CI: 0.62-4.92, p=0.28 and HR: 1.35, 95% CI: 0.81-2.23, p=0.24, respectively). In the early post-procedural period, 35 patients (20.3%) showed improvement in MR severity (p<0.001), and in the late post-procedural period, 49 patients (38.8%) showed improvement in MR severity (p=0.001). Similarly, in the early post-procedural period, 31 patients (17.8%) showed improvement in TR severity (p=0.08), and in the late post-procedural period, 39 patients (30.9%) showed improvement in TR severity (p=0.008). Adjusted analyses did not demonstrate a significant association between improvement in MR or TR and survival (HR: 0.54, 95% CI: 0.14-1.08, p=0.08 and HR: 0.61, 95% CI: 0.22-1.61, p=0.34, respectively). In the multivariate analysis, the use of beta-blockers and left atrial dilatation were identified as predictors of MR severity improvement, while elevated TR velocity was identified as a poor prognostic indicator for TR severity improvement.

Conclusions: Our study found that the severity of pre-procedural MR and TR did not serve as independent prognostic markers for the primary endpoint. We observed improvement in MR and TR severity after TAVI, but this improvement did not impact the primary endpoint. Based on these findings, the severity of MR or TR should not be considered contraindications for TAVI.

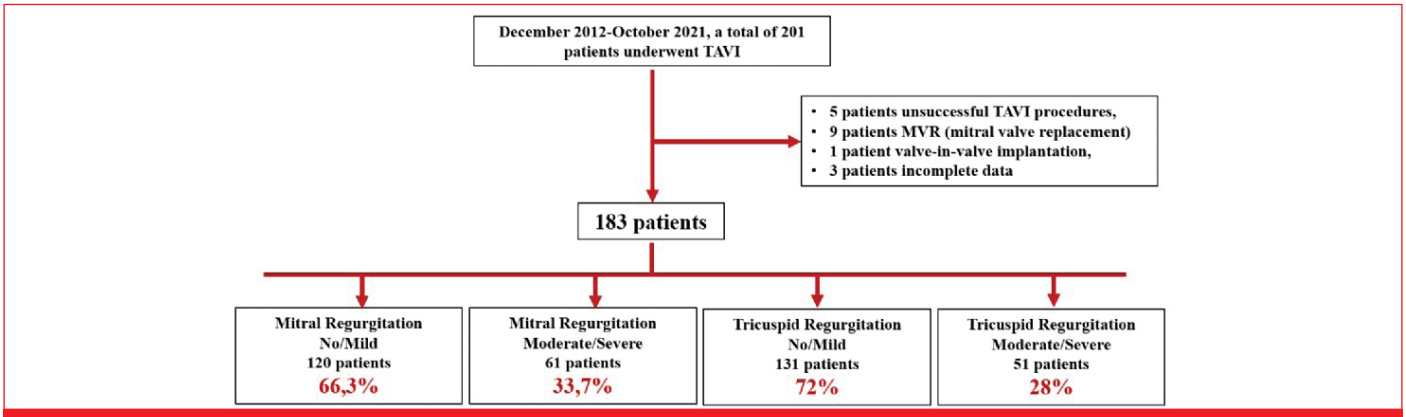

Figure 1. Study population.

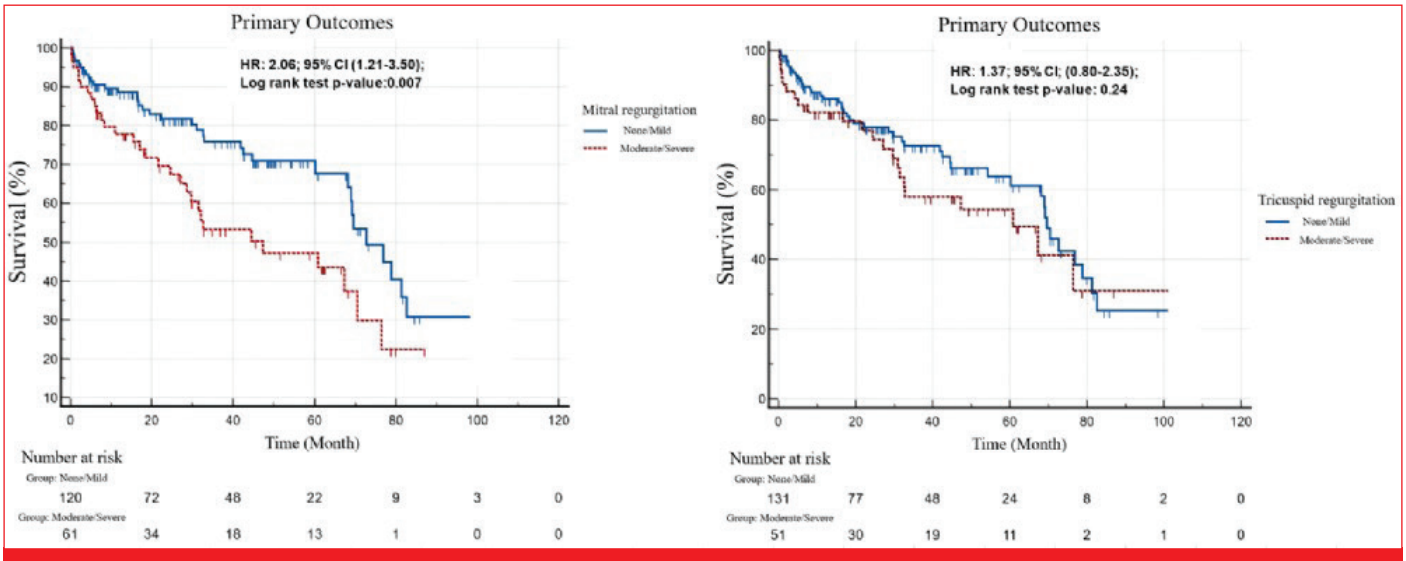

Figure 2. Kaplan Meier primary outcome analysis of patients treated with TAVI according to MR and TR at baseline.

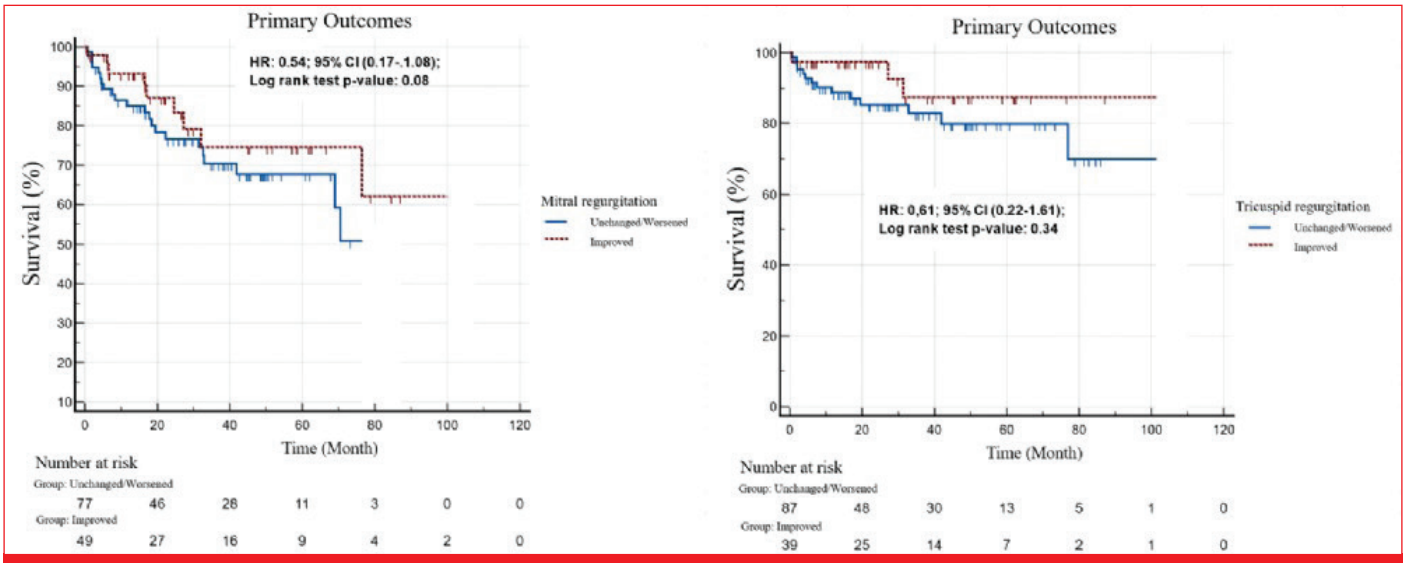

Figure 3. Kaplan Meier primary outcome analysis of patients treated with TAVI according to MR and TR change.

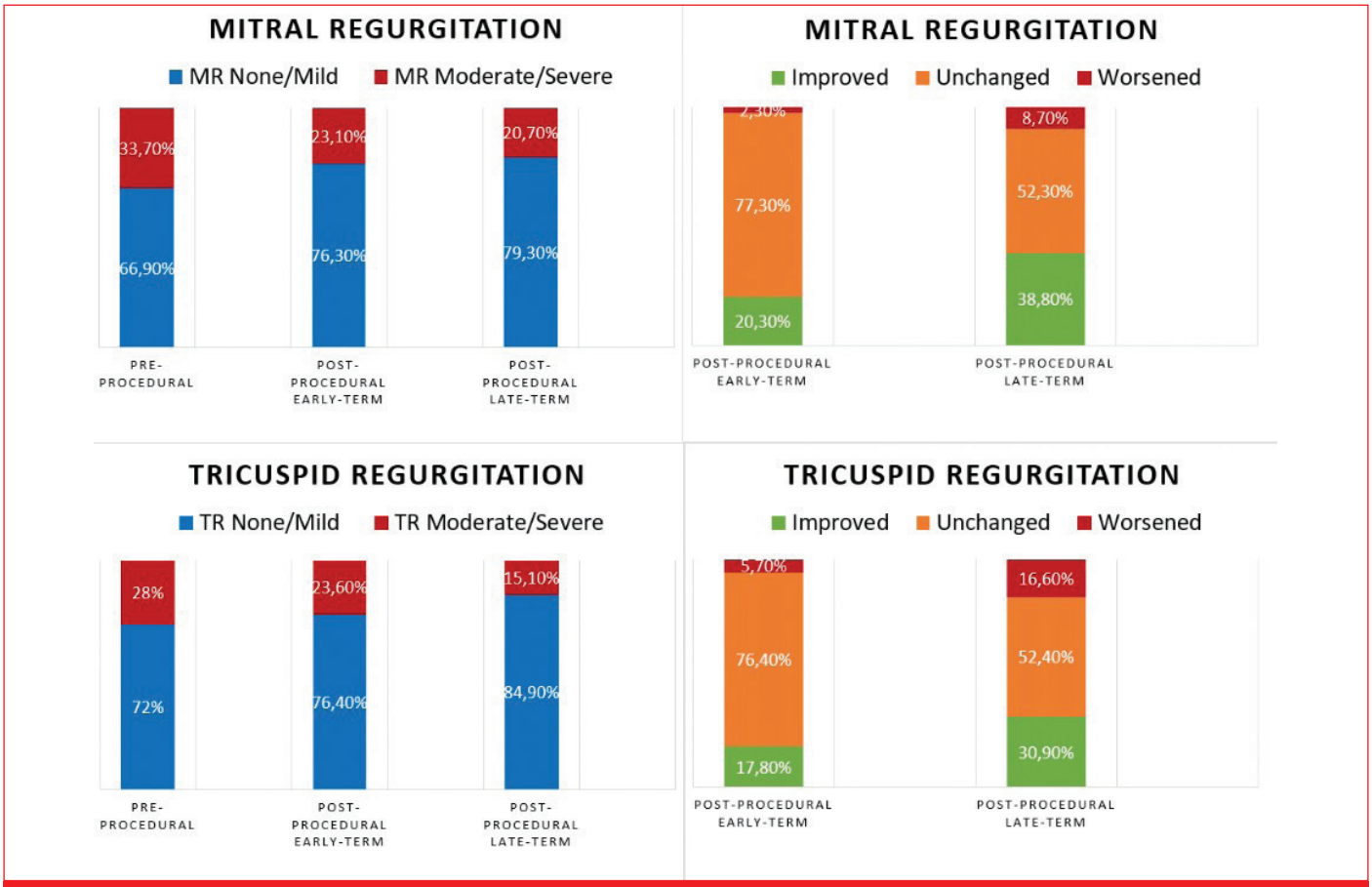

Figure 4. Early and late change in MR and TR after the procedure.

Table 1. Baseline characteristics

Parameters	Total (n=183)	MR None/Mild (n=120, 66.3%)	MR Moderate/ Severe (n=61, 33.7%)	p	TR None/Mild (n=131, 72%)	TR Moderate/ Severe (n=51, 28%)	p
Age, years, Mean ± SD	77.6 ± 6	77 ± 6.7	78.8 ± 6.4	0.08	77.2 ± 6.4	78.7 ± 7.1	0.65
Gender (male), n (%)	81 (44.3)	47 (39.2)	32 (52.5)	0.11	51 (41.2)	26 (51)	0.24
BMI, kg/m^2, Mean ± SD	27.8 ± 5.8	28.9 ± 5.9	27.4 ± 5.7	0.87	28.8 ± 5.9	27.1 ± 5.5	0.62
BSA, m^2, Mean ± SD	1.8 ± 0.1	1.8 ± 0.1	1.8 ± 0.2	0.16	1.8 ± 0.1	1.8 ± 0.2	0.25
STS score, Mean ± SD	5.3 ± 3	4.5 ± 2.8	6.9 ± 3.4	0.001	4.6 ± 2.3	7.1 ± 4.5	0.001
NYHA 1-2, n (%)	92 (50.3)	70 (58.3)	21 (34.4)	-	78 (59.5)	14 (27.5)	-
NYHA 3-4, n (%)	91 (49.7)	50 (41.7)	40 (65.6)	0.003	53 (40.5)	37 (72.5)	0.001
CAD, n (%)	135 (75.8)	84 (73)	49 (80.3)	0.35	94 (74)	40 (80)	0.44
LVEF <50, n (%)	43 (24.7)	19 (16.7)	24 (41.4)	0.001	25 (20.5)	18 (35.3)	0.05
AF, n (%)	37 (20.4)	18 (15)	18 (31)	0.01	16 (12.3)	20 (40.8)	0.001
NT-pro-BNP, ng/L Median, Q1-Q3	1747 (47-60000)	1361 (47-35335)	3583 (114-60069)	0.01	1385 (47-35000)	6616 (114-60069)	0.001
Device	23 (12.6)	14 (11.7)	9 (14.8)	0.91	12 (9.2)	11 (21.6)	0.02
PPM, n (%)	18 (9.8)	11 (9.2)	7 (11.5)	-	11 (8.4)	7 (13.7)	-
ICD, n (%)	3 (1.6)	2 (1.7)	1 (1.6)	-	0	3 (5.9)	-
CRT-D, n (%)	2 (1.1)	1 (0.8)	1 (1.6)	-	1 (0.8)	1 (2)	-
Smoke, n (%)	64 (35)	39 (32.5)	23 (37.7)	0.51	43 (32.8)	20 (39.2)	0.48
Arterial hypertension, n (%)	150 (82.4)	99 (82.5)	49 (81.7)	0.89	109 (84)	40 (78.4)	0.39
Diabetes mellitus, n (%)	78 (42.6)	52 (43.3)	25 (41.0)	0.87	56 (42.7)	21 (41.2)	0.86
Dyslipidemia, n (%)	107 (58.5)	66 (55)	39 (63.9)	0.26	75 (57.3)	31 (60.8)	0.73
Cerebrovascular event, n (%)	9 (5.4)	5 (4.2)	4 (6.6)	0.61	9 (6.9)	0	0.12
COPD, n (%)	33 (18)	23 (20)	9 (14.8)	0.79	26 (19.8)	7 (13.7)	0.84
Renal disease, n (%)	41 (22.4)	27 (22.5)	14 (23)	0.94	10 (7.6)	4 (7.8)	0.55

* Values are mean ± SD or n (%) p value <0.05. MR: Mitral regurgitation, TR: Tricuspid regurgitation, BMI: Body mass index, BSA: Body surface area, STS: Society of Thoracic Surgeons, NYHA: New York Heart Association, CAD: Coronary artery disease, LVEF: Left ventricular ejection fraction, AF: Atrial fibrilation, PPM: Permanent pacemaker, ICD: Implanted cardioverter-defibrillator, CRT-D: Cardiac resynchronization therapy-defibrillator, COPD: Chronic obstructive pulmonary disease, PAD: Peripheral arterial disease.

Table 2. Medication of patients

Parameters	Total (n=183)	MR None/Mild (n=120, 66.3%)	MR Moderate/ Severe (n=61, 33.7%)	p	TR None/Mild (n=131, 72%)	TR Moderate/ Severe (n=51, 28%)	p
ACEi or ARB, n (%)	70 (42.9)	50 (41.6)	19 (31.1)	0.09	53 (43.4)	16 (40)	0.71
Beta-blocker, n (%)	113 (61.7)	66 (55)	47 (77)	0.006	78 (59.5)	35 (68.6)	0.30
Antihyperlipidemic, n (%)	73 (40)	42 (35)	29 (47.5)	0.29	49 (37.4)	23 (45)	0.78
MRAs, n (%)	13 (7.2)	8 (6.8)	5 (8.2)	0.76	7 (5.4)	6 (11.8)	0.19
Loop diüretic, n (%)	28 (16.9)	19 (17.1)	8 (15.1)	0.82	18 (15)	9 (20)	0.48
Tiazid diüretic, n(%)	38 (20.9)	29 (24.4)	9 (14.8)	0.32	30 (23.1)	8 (15.7)	0.21
Calcium channel blockers, n (%)	42 (23.1)	30 (25.2)	11 (18)	0.55	31 (23.8)	11 (21.6)	0.14
Digoxin, n (%)	12 (6.6)	6 (5.1)	6 (9.8)	0.34	8 (6.2)	4 (7.8)	0.74
Antiarrhythmic, n (%)	12 (6.6)	7 (5.9)	5 (8.2)	0.54	10 (7.7)	2 (3.9)	0.51
Anticoagulant, n (%)	33 (18.2)	16 (13.6)	17 (27.9)	0.01	16 (12.4)	17 (33.3)	0.001
VKA, n(%)	14 (7.7)	8 (6.8)	6 (9.8)	-	7 (5.4)	7 (13.7)	-
NOAC, n(%)	19 (10.5)	8 (6.8)	11 (18.1)	-	9 (7)	10 (19.6)	-
Antiplatelet acetylsalicyc acid, n (%)	144 (79.1)	96 (80.7)	46 (75.4)	0.44	107 (82.3)	36 (70.6)	0.10
Klopidogrel, n (%)	137 (75.3)	96 (80.7)	41 (67.2)	0.06	104 (80)	33 (64.7)	0.09

* Values are mean ± SD or n (%) p value <0.05. MR: Mitral regurgitation, TR: Tricuspid regurgitation, ACEi: Angiotensinogen was converting enzyme inhibitor, ARB: Angiotensin receptor blocker, MRAs: Mineralocorticoid receptor antagonists, VKA: Vitamin K antagonists; NOAC: New oral anticoagulants.

Table 3. Characteristics of the patients regarding the TAVI procedure

Parameters	Total (n=183)	MR None/Mild (n=120, 66.3%)	MR Moderate/ Severe (n=61, 33.7%)	p	TR None/Mild (n=131, 72%)	TR Moderate/ Severe (n=51, 28%)	p
Edwards SAPIEN, n (%)	23 (12.6)	10 (8.3)	13 (21.3)	0.02	14 (10.7)	9 (17.6)	0.25
23 mm, n (%)	10 (43.4)	-	-	-	-	-	-
26 mm, n (%)	8 (34.7)	-	-	-	-	-	-
29 mm, n (%)	5 (21.7)	-	-	-	-	-	-
1. Generation corevalve, n (%)	8 (4.4)	8 (6.7)	0	-	7 (5.3)	1 (2)	-
26 mm, n (%)	5 (62.5)	-	-	-	-	-	-
29 mm, n (%)	3 (37.5)	-	-	-	-	-	-
2. Generation evolute R, n (%)	152 (83.1)	102 (85)	48 (78.7)	0.34	110 (84)	41 (80.4)	0.74
20 mm, n (%)	1 (0.7)	-	-	-	-	-	-
23 mm, n (%)	1 (0.7)	-	-	-	-	-	-
26 mm, n (%)	51 (33.6)	-	-	-	-	-	-
28 mm, n (%)	1 (0.7)	-	-	-	-	-	-
29 mm, n (%)	73 (48)	-	-	-	-	-	-
34 mm, n (%)	24 (15.8)	-	-	-	-	-	-
PVL, n (%)	80 (46.5)	56 (49.1)	24 (42)	0.45	56 (44.8)	22 (46.8)	0,73
Mild, n (%)	78 (45.3)	54 (47.4)	24 (42)	-	55 (44)	21 (44.7)	-
Moderate, n (%)	2 (1.2)	2 (1.8)	0	-	1 (0.8)	1 (2.1)	-
PCI before the procedure, n (%)	24 (13.1)	16 (13.3)	8 (13.1)	0.96	19 (14.5)	5 (9.8)	0.47
LAD, n (%)	16 (8.7)	11 (9.1)	5 (8.3)	-	11 (8.3)	5 (9.8)	-
Cx, n (%)	7 (3.8)	5 (4.1)	2 (3.3)	-	6 (4.5)	1 (1.9)	-
RCA, n (%)	5 (3.7)	4 (3.3)	3 (5)	-	6 (4.5)	1 (1.9)	-
Post-procedure PPM, n (%)	17 (9.3)	12 (10)	5 (8.3)	0.79	13 (10)	4 (7.8)	0.78

* Values are mean ± SD or n (%) p value <0.05. MR: Mitral regurgitation, TR: Tricuspid regurgitation, PVL: Paravalvuler leak, PCI: Percutaneous coronary intervention, LAD: Left anterior descending artery, Cx: Circumflex artery, RCA: Right coronary artery, PPM: Permanent pacemaker.

Table 4. Primary outcomes

Parameters	Total (n=183)	MR None/Mild (n=120, 66.3%)	MR Moderate/ Severe (n=61, 33.7%)	p	TR None/Mild (n=131, 72%)	TR Moderate/ Severe (n=51, 28%)	p
Primary outcomes, n (%)	68 (37.2)	37 (30.8)	30 (49.2)	0.02	45 (34.4)	23 (45.1)	0.23
All cause mortality, n (%)	56 (30.6)	30 (25)	25 (41)	0.04	35 (26.7)	21 (41.1)	0.07
Heart failure-related hospitalization, n (%)	20 (10.9)	9 (7.5)	11 (18)	0.04	12 (9.1)	8 (15.6)	0.29
1-year primary outcomes, n (%)	26 (14.2)	13 (10.8)	13 (21.3)	0.008	17 (12.9)	9 (17.6)	0.24
Follow-up, Month, Median, Q1-Q3	27.1 (9.5-50.1)	27.5 (10.2-50.0)	26.5 (7.5-48.3)	-	26.5 (9.9-49.8)	29.7 (9.7-53.7)	-

* Values are mean ± SD or n (%) p value <0.05. MR: Mitral regurgitation, TR: Tricuspid regurgitation.

Table 5. Predictors of primary outcomes

Parameters	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p	HR (95% CI)	p
Mitral regurgitation	1.934 (1.186-3.153)	0.008	1.758 (0.627-4.928)	0.28
Tricuspid regurgitation	1.351 (0.815-2.239)	0.24	-	-
Age	1.028 (0.989-1.068)	0.16	-	-
Gender/Male	1.750 (1.075-2.847)	0.02	1.201 (0.301-1.524)	0.34
STS score	1.254 (1.169-1.345)	<0.001	1.201 (1.036-1.392)	0.01
NYHA	3.239 (1.931-5.433)	<0.001	-	-
Smoker	1.440 (0.881-2.354)	0.14	-	-
Arterial hypertension	1.783 (0.879-3.616)	0.10	-	-
CAD	1.868 (1.013-3.445)	0.04	2.723 (0.945-7.849)	0.06
LVEF <50	1.986 (1.142-3.452)	0.01	1.156 (0.364-3.671)	0.80
NT-pro-BNP	2.029 (1.330-3.095)	0.001	1.560 (0.612-3.977)	0.35
Renal disease	1.830 (1.062-3.154)	0.03	-	-
AF	2.453 (1.453-4.143)	0.001	2.468 (1.059-5.751)	0.03
ACEi or ARB	1.074 (0.623-1.851)	0.79	-	-
Beta-blocker	1.013 (0.610-1.681)	0.96	-	-
LVEF	0.987 (0.969-1.005)	0.16	-	-
RVEF	0.957 (0.899-1.019)	0.16	-	-
LA	1.830 (1.281-2.614)	0.001	1.210 (0.655-2.234)	0.54
TAPSE	0.945 (0.877-1.019)	0.14	-	-
RVSM	0.931 (0.831-1.042)	0.21	-	-
TRV	1.286 (0.914-1.809)	0.14	-	-
sPAP	1.015 (1.001-1.029)	0.03	0.995 (0.966-1.026)	0.76
PCI before the procedure	1.920 (0.932-3.955)	0.07	-	-

Cox univariate and multivariate model, p value <0.05. HR: Hazard ratio, CI: Confidence interval, STS: Society of Thoracic Surgeons, NYHA: New York Heart Association, CAD: Coronary artery disease, LVEF: Left ventriculer ejection fraction, AF: Atrial fibrilation, ACEi: Angiotensinogen was converting enzyme inhibitor, ARB: Angiotensin receptor blocker, RVEF: Right ventricular ejection fraction, LA: 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, PCI: Percutaneous coronary intervention.

Table 6. Pre-procedural, post-procedural 0-3 months, and post-procedural 3 months-1 year follow-up echocardiographic characteristics of all patients

Parameters	Baseline (n=183, 100%)	0-3 month (n=174, 95.1%)	p	>3 month (n=126, 68.9%)	p
LVEF, %	54.2 ± 12	54.9 ± 12	0.001	56.9 ± 10	0.001
RVEF, %	54.1 ± 3.2	54.2 ±2.6	0.11	54.5 ± 2.1	0.31
LVEDD, cm	4.9 ± 2.6	4.6 ± 0.6	0.26	4.7 ± 0.6	0.42
LVESD, cm	3.1 ± 0.8	3.0 ± 0.7	0.46	3.0 ± 0.7	0.10
LA, cm	4.3 ± 0.7	4.2 ± 0.7	0.81	4.2 ± 0.6	0.66
RV, cm	2.4 ± 0.3	2.4 ± 0.4	0.13	2.4 ± 0.4	0.16
IVS, cm	1.3 ± 0.2	1.3 ± 0.2	0.002	1.2 ± 0.2	0.04
PD, cm	1.1 ± 0.1	1.1 ± 0.1	0.056	1.1 ± 0.1	0.07
RWT	0.51 ± 0.12	0.51 ± 0.12	0.95	0.49 ± 0.10	0.26
LVMI, g/m^2	129.2 ± 37	121.0 ± 30	0.001	118.1 ± 31	0.01
AVmax, m/sn	4.3 ± 0.7	1.7 ± 0.1	<0.001	1.8 ± 0.3	<0.001
Aort max gradient, mmHg	77.8 ± 19	15.3 ± 6.8	<0.001	16.4 ± 6.8	<0.001
Aort mean gradient, mmHg	48 ± 12	7.9 ± 4	<0.001	8.6 ± 4.2	<0.001
TAPSE, mm	21 ± 3.6	21.6 ± 3.9	0.007	21.3 ± 3.9	0.30
RVSM, cm/san	11.9 ± 4	11.8 ± 2.0	0.15	12 ± 2.6	0.11
TRV, m/san	2.7 ± 0.6	2.6 ± 0.6	0.002	2.4 ± 0.5	0.001
sPAP, mmHg	39.1 ± 16	35.7 ± 14.6	0.001	31.6 ± 11.7	0.001
Mitral stenosis None/Mild, n (%)	172 (94)	168 (96.6)	-	119 (94.5)	-
Moderate/Severe, n (%)	11 (6)	6 (3.4)	-	7 (5.5)	-
Aortic regurgitation None/Mild, n (%)	145 (80.6)	170 (98.8)	-	120 (97.6)	-
Moderate/Severe, n (%)	35 (19.4)	2 (1.2)	<0.001	3 (2.4)	<0.001
Mitral regurgitation None/Mild, n (%)	120 (66.3)	133 (76.9)	-	100 (79.3)	-
Moderate/Severe, n (%)	61 (33.7)	40 (23.1)	<0.001	26 (20.7)	0.001
Tricuspid regurgitation None/Mild, n (%)	131 (72)	133 (76.4)	-	107 (84.9)	-
Moderate/Severe, n (%)	51 (28)	41 (23.6)	0.08	19 (15.1)	0.008

* Values are mean ± SD or n (%) p value <0.05. LVEF: Left ventricular systolic ejection fraction, RVEF: Right ventricular systolic ejection fraction, LVEDD: Left ventricular end diastolic diameter, LVESD: Left ventricular end systolic diameter, LA: Left atrial diameter, RV: Right ventricular diameter, IVS: Interventricular septum, PW: Posterior wall, RWT: Relative wall thickness, LVMI: Left ventricular mass index, AVA: Aort valve area, 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. Univariate and multivariate analysis for prediction of mitral regurgitation improvement

Parameters	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
Age	0.971 (0.920-1.025)	0.28	-	-
STS Score	0.938 (0.821-1.071)	0.34	-	-
CAD	1.434 (0.619-3.326)	0.40	-	-
LVEF <50	0.660 (0.282-1.544)	0.33	-	-
NT-pro-BNP	0.808 (0.435-1.503)	0.35	-	-
Diabetes mellitus	1.450 (0.668-3.149)	0.34	-	-
Renal disease	2.059 (0.741-5.719)	0.16	2.013 (0.650-6.232)	0.22
AF	1.277 (0.483-3.377)	0.62	-	-
ACEi or ARB	0.708 (0.318-1.577)	0.39	-	-
Beta-blocker	0.274 (0.107-0.702)	0.007	0.261 (0.098-0.691)	0.007
LVEF	1.020 (0.992-1.049)	0.15	0.999 (0.954-1.046)	0.96
LVESD	0.716 (0.460-1.115)	0.14	0.920 (0.435-1.898)	0.75
LA	0.410 (0.229-0.734)	0.003	0.406 (0.219-0.755)	0.004
sPAP	0.980 (0.959-1.002)	0.07	0.990 (0.965-1.015)	0.43
Aortic regurgitation	0.564 (0.182-1.752)	0.16	1.589 (0.285-8.862)	0.38
Tricuspid regurgitation	0.824 (0.299-2.267)	0.06	0.821 (0.052-1.223)	0.35
Prothesis valve size	0.860 (0.759-0.974)	0.01	0.921 (0.684-1.241)	0.59
PCI before the procedure	3.157 (0.705-14.124)	0.13	4.822 (0.976-23.833)	0.054
Post-procedure PPM	0.256 (0.033-2.019)	0.19	3.556 (0.423-29.902)	0.26

Logistic univariate and multivariate model, p value <0.05. OR: Odds ratio, CI: Confidence interval, STS: Society of Thoracic Surgeons, CAD: Coronary artery disease, AF: Atrial fibrilation, ACEi: Angiotensinogen was converting enzyme inhibitor, ARB: Angiotensin receptor blocker, LVEF: Left ventricular ejection fraction, LVESD: Left ventricular end-sistolic diameter, LA: Left atrial diameter, sPAP: Systolic pulmonary artery pressure, PCI: Percutaneous coronary intervention, PPM: Permenant pacemaker.

Table 8. Univariate and multivariate analysis for prediction of tricuspid regurgitation improvement

Parameters	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
Age	0.965 (0.898-1.038)	0.34	0.780 (0.181-3.355)	0.73
CAD	0.336 (0.073-1.548)	0.16	0.282 (0.047-1.683)	0.16
NYHA	0.616 (0.314-1.209)	0.15	2.153 (0.458-10.131)	0.33
LVEF <50	1.324 (0.402-4.367)	0.64	-	-
LVEF	0.963 (0.920-1.008)	0.10	0.967 (0.904-1.034)	0.32
RVEF	0.948 (0.778-1.155)	0.59	-	-
RV	2.144 (0.484-9.499)	0.31	-	-
LA	0.872 (0.462-1.645)	0.67	-	-
TAPSE	1.046 (0.913-1.199)	0.51	-	-
RVSM	0.937 (0.736-1.192)	0.59	-	-
TRV	2.492 (1.146-5.421)	0.02	4.625 (1.436-14.897)	0.01
sPAP	1.035 (0.998-1.073)	0.06	-	-
Aortic regurgitation	0,727 (0.218-2.430)	0.66	-	-
Mitral regurgitation	1.418 (0.508-3.960)	0.50	-	-
Prothesis valve size	1.194 (1.000-1.426)	0.04	1.153 (0.900-1.477)	0.26

Logistic univariate and multivariate model, p value <0.05. OR: Odds ratio, CI: Confidence interval, CAD: Coronary artery disease, NYHA: New York Heart Association, LVEF: Left ventricular ejection fraction, RVEF: Right ventricular ejection fraction, LA: 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.

Other

OP-108

Relationship between mortality and leuko-glycemic index in coronary care unite patients: MORCOR-TURK study

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Background and Aim: Identifying high-risk patients with a poor prognosis in coronary care unite (CCU) patients can assist physicians in providing optimal care and implementing preventive measures. Leuko-glycemic index (LGI), synthesized by multiplying the blood glucose level by the leukocyte count, has gained popularity in risk stratification of myocardial infarction patients. In this context, this study was carried out to investigate the relationship between LGI assessed at admission and in hospital mortality in CCU patients.

Methods: This is a multi-center retrospective observational study. The population of this retrospective study consisted of 3137 consecutive patients admitted to the CCU between 1 February 2023 and 28 February 2023. Age <18 years, chronic inflammatory disease, previously diagnosed with CAD, thyroid disorders, hemolytic disease, malignancy, chronic lung diseases, liver diseases, rheumatic disease, chronic kidney failure and history of hemodialysis, and nonregulated diabetes mellitus were excluded. An additional eight patients with missing data in the hospital's electronic database were excluded from the study. In the end, 2917 patients were included in the study sample. Baseline demographic and clinical characteristics were obtained for each patient from the hospital's electronic database. The LGI scores were calculated for each patient.

Results: Univariate logistic regression analysis revealed clinically and statistically significant predictors age, new onset heart failure (HF), LGI, coronary artery disease, hypertension, diabetes mellitus and new onset atrial fibrillation. Further analysis of these variables using the multivariate logistic regression analysis indicated that age (OR: 1.040, 95% CI: 1.017-1.063, p=0.001), new onset HF (OR: 2.426, 95% CI: 1.419-4.149, p=0.001) and LGI (OR: 1.349, 95% CI: 1.176-1.549, p<0.001), were independent predictors for the development of in-hospital mortality in CCU. LGI score optimal cut-off value of >3.72 predicted in-CCU mortality with 95.56% sensitivity and 49.19% specificity (AUC: 0.659; 95% CI: 0.641-0.676, p<0.001).

Conclusions: LGI, a simple and inexpensive index, was associated with in hospital mortality in CCU patients. Regarding clinical relevance, LGI, a simple index that can be used in any intensive care unit, is easy to calculate and low cost, and can be used for risk stratification and mortality prediction of CCU patients. Patients with high LGI values may benefit from greater monitoring and early therapeutic strategies. Aggressive treatment strategies should be adopted for these patients with higher LGI upon admission. Prospective studies are needed to clarify the prognostic relevance of LGI and CCU patients' mortality in terms of future cardiovascular events.

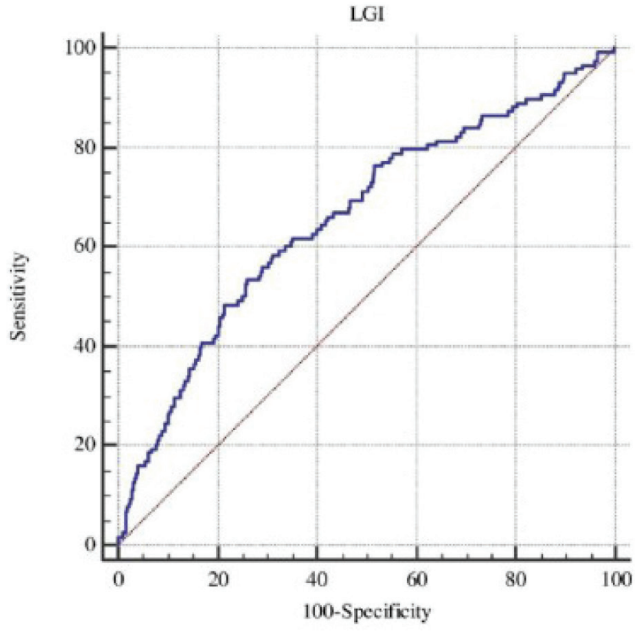

Figure 1. ROC curve analysis of LGI score to predict mortality in CCU.

ROC: Receiver operating characteristic, LGI: Leuko-glycemic index, CCU: Coronary care unit.

Table 1. The baseline demographic and laboratory characteristics of the CCU patients

	Low LGI score <1.23 (n=1458)	High LGI score >1.23 (n=1459)	p
Age, years	64 ± 14	65 ± 12	0.844
Gender (male), n (%)	986 (67.6)	946 (64.8)	0.111
Patients with DM, n (%)	356 (24.4)	734 (50.3)	<0.001
Patients with HT, n (%)	836 (57.3)	898 (61.5)	0.021
Active smokers, n (%)	469 (32.2)	526 (36.1)	0.094
Patient with CAD	667 (45.7)	659 (45.2)	0.753
Patient with AF, n (%)	239 (16.4)	200 (13.7)	0.032
New onset AF, n (%)	449 (30.7)	34 (2.3)	0.002
Patient with HF, n (%) (reduced and preserved)	412 (29.3)	471 (32.3)	0.080
New onset HF, n (%)	902 (61.8)	76 (5.2)	<0.001
Patient with stroke, n (%) (isckemic and hemorrhagic)	48 (3.2)	84 (5.8)	0.001
Patient with CKD, n (%)	185 (12.7)	217 (14.8)	0.091
Killip class >1, n (%)	314 (21.5)	1180 (80.8)	<0.001
SBP	130 ± 23	130 ± 25	0.663
Patient taking positive inotropes, n (%)	101 (7)	142 (9.7)	0.006
Heart rate, bpm	83 ± 23	86 ± 22	<0.001
Ejection fraction, %	50.5 ± 11.4	46.7 ± 11.8	<0.001
Glucose level, mg/dL	105 (94-121)	166 (132-232)	<0.001
Total cholesterol, mg/dL	171 ± 49	181 ± 55	<0.001
HDL, mg/dL	40.9 ± 11.3	41 ± 11.4	0.894
LDL, mg/dL	105.8 ± 39.2	113.2 ± 48.8	<0.001
Triglyceride, mg/dL	114 (81-168)	127 (88-181)	<0.001
Creatine level, mg/dL	0.95 (0.8-1.17)	1 (0.8-1.29)	<0.001
CRP, mg/dL	4.1 (1.65-12)	7.35 (2.5-21.8)	<0.001
Hemoglobin level, mg/dL	13.2 ± 2.2	13.4 ± 2.2	0.049
WBC count, 10^3/mL	7.94 ± 2.06	12.11 ± 3.72	0.001

CCU: Coronary care unit, DM: Diabetes mellitus, HT: Hypertension, CAD: Coronary artery disease, AF: Atrial fibrillation, HF: Heart failure, CKD: Chronic kidney disese, LDL: Low density lipoprotein, HDL: High density lipporotein, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, bpm: beats per minute, WBC: White blood cell, LGI: Leuko-glycemic index.

Table 2. Results of the univariate and multivariate analyses of the variables

	Univariate OR, 95% CI	p	Multivariate OR, 95% CI	p
Age	1.050 (1.035-1.066)	<0.001	1.040 (1.017-1.063)	0.001
New onset HF	2.926 (2.070-4.134)	<0.001	2.426 (1.419-4.149)	0.001
LGI	1.349 (1.222-1.489)	<0.001	1.349 (1.176-1.549)	<0.001
CAD	0.750 (0.433-1.298)	0.304		
Hypertension	0.784 (0.448-1.372)	0.394		
DM	0.909 (0.512-1.613)	0.745		
New onset AF	1.700 (0.778-3.716)	0.183		

HF: Heart failure, LGI: Leuko-glycemic index, OR: Odds ratio, CI: Confidence interval, p: Probability statistic, CAD: Coronary artery disease, DM: Diabetes mellitus, AF: Atrial fibrillation.

Other

OP-109

Prognosis of chronic kidney disease patients and predictors of acute kidney injury in coronary care unit: A subgroup analysis of MORCOR-TURK trial

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Background and Aim: Chronic kidney disease (CKD) patients admitted to coronary care units (CCUs) are at an elevated risk of acute kidney injury (AKI), leading to adverse outcomes and increased mortality. Despite advancements in medical care, the identification of CKD patients at risk of AKI in the CCU remains challenging, and the precise predictors of AKI in this population are not yet fully understood. The aim of this study is to investigate the prognosis of CKD patients admitted to the CCU and to identify predictors of AKI in this cohort.

Methods: This prospective, multicenter study included a total of 615 patients admitted to various hospitals over a one-year period. Among these patients, 288 developed acute kidney injury (AKI+) during their hospital stay, while 327 patients did not experience AKI (AKI-). The study aimed to identify independent predictors of AKI in patients with preexisting chronic kidney disease (CKD) and evaluate their prognosis in coronary care units. Data on demographics, comorbidities, laboratory values, and outcomes were collected prospectively from electronic medical records. Descriptive statistics, chi-square tests, and logistic regression analysis were utilized to identify significant predictors of AKI in CKD patients and improve risk assessment strategies to enhance patient outcomes across multiple medical facilities.

Results: The demographic and clinical characteristics of the study population are presented in Table 1. Among the 615 patients, 288 developed acute kidney injury, while 327 did not experience AKI. The logistic regression analysis revealed several significant predictors of AKI in the study population (Table 2). Patients with CKD showed a substantially higher risk of developing AKI compared to those without CKD (OR: 5.080, p<0.001). Additionally, decompensated heart failure was associated with an increased risk of AKI (OR: 3.103, p=0.003). Conversely, non-primary percutaneous coronary intervention (PCI) was found to be a protective factor against AKI when compared to primary PCI (OR: 0.268, p=0.039). Lower serum albumin levels were significantly associated with an increased risk of AKI (OR: 0.918, p=0.786), while elevated troponin levels were associated with a higher risk of AKI (OR: 1.002, p=0.026). Furthermore, mechanical ventilation (MV) was identified as a significant predictor of AKI (OR: 3.346, p<0.001), as well as bleeding (OR: 5.258, p=0.078).

Conclusions: In conclusion, this study revealed that CKD significantly increased the risk of AKI in patients admitted to coronary care units. Decompensated heart failure, non-primary PCI, lower serum albumin levels, elevated troponin levels, mechanical ventilation, and bleeding were identified as important predictors of AKI. These findings emphasize the importance of targeted risk assessment and preventive measures to improve outcomes in CKD patients in coronary care units.

Table 1. The demographic and clinical data of the study population

Parameters	AKI (+) (n=288)	AKI (-) (n=327)	P value
Age (years)	71. 4 ± 10.3	72.5 ± 10.9	0.221
Male, n(%)	185 (64.2)	180 (55.0)	0.021
Smoker	177 (61.4)	165 (50.4)	0.020
BMI (kg/m^2)	28.4 ± 5.7	28.1 ± 6.1	0.594
CHADVASc Score	3.9 ± 1.2	4.0 ± 1.5	0.828
Killip Class	1.8 ± 0.8	2.2 ± 0.9	<0.001
Systolic Blood Pressure (mmHg)	130.8 ± 25.8	122.8 ± 31.8	0.001
Mean Blood Pressure (mmHg)	94.4 ± 16.9	89.7 ± 22.5	0.004
Heart rate (BPM)	86.5 ± 24.1	92.0 ± 28.2	0.009
Oxygen saturation (%)	93.3 ± 4.9	91.3 ± 6.4	<0.001
Ejection fraction (%)	44.4 ± 13.6	41.9 ± 12.9	0.025
Death, n(%)	14 (4.9)	79 (24.2)	<0.001
Medical History, n(%)			
Ischaemic heart disease	188 (65.3)	180 (55.0)	0.010
PCI	144 (50)	135 (41.3)	0.030
CABG	58 (20.1)	59 (18.0)	0.509
MI history	129 (44.8)	134 (41.0)	0.340
Atrial Fibrillation	80 (27.7)	105 (32.1)	0.245
Heart Failure	168 (58.3)	203 (62.0)	0.426
Hypertension	206 (81.9)	248 (75.8)	0.065
Diabetes mellitus	152 (52.8)	150 (45.9)	0.087
Cerebrovascular Disease	21 (7.2)	32 (9.7)	0.454
Treatment			
ACEi/ARB/ARNI	140 (48.6)	178 (54.7)	0.281
Statin	111 (38.5)	114 (35.0)	0.464
Inotropic drugs	33 (11.5)	115 (35.2)	<0.001
Laboratory Findings			
Fasting Glucose (mg/dl)	160.5 ± 72.6	171.7 ± 82.3	0.087
Creatinine (mg/dl)	2.1 ± 1.4	1.9 ± 1.2	0.056
GFR (mL/min/1.73 m^2)	39.2 ± 17.8	41.1 ± 20.3	0.260
Sodium (mmol/l)	136.9 ± 4.4	135.7 ± 5.9	0.006
Potassium (mmol/l)	4.6 ± 0.6	4.7 ± 0.9	0.151
Albümin (g/dl)	3.8 ± 0.4	3.6 ± 0.6	<0.001
Haemoglobin (g/dl)	11.9 ± 2.3	11.6 ± 2.3	0.129
CRP (mg/l)	10.8 (3.3-30.0)	16.0 (4.4-53.7)	0.011
ALT (IU/l)(25-75p)	17.0 (12.0-29.7)	22.0 (13.6-44.0)	<0.001
AST (IU/l) (25-75p)	24.0 (17.0-40.7)	32.0 (20.0-65.0)	<0.001
Troponin (ng/dl) (25-75p)	51.9 (10.0-361.0)	52.0 (3.3-540.0)	0.895

Table 2. Univariate and multivariate analyses of predictors of acute kidney disease on logistic regression analysis

Variable	Univariate			Multivariate		
	OR	95%CI	p	OR	95%CI	p
Age	1.009	0.994-1.024	0.221			
Gender	1.467	1.060-2.030	0.021	1.782	0.935-3.396	0.079
Hypertension	0.692	0.467-1.024	0.066	0.634	0.287-1.398	0.258
Diabetes Mellitus	0.758	0.552-1.042	0.088	1.232	0.605-2.510	0.566
CAD	0.651	0.470-0.902	**0.010**	0.805	0.380-1.704	0.570
CKD	5.587	3.631-8.598	**<0.001**	5.080	2.274-11.351	**<0.001**
Decompensated HF	1.471	1.017-2.127	**0.041**	3.103	1.476-6.523	**0.003**
Killip Class	1.622	1.333-1.969	**<0.001**	1.029	0.613-1.728	0.913
Primary PCI	1.471	1.017-2.127	**0.041**	3.391	1.496-7.688	**0.003**
Non-primary PCI	0.425	0.260-0.694	**0.001**	0.268	0.177-0.432	**0.039**
SBP	0.991	0.985-0.996	**0.001**	1.009	0.998-1.021	0.116
Heart rate	1.008	1.002-1.014	**0.010**	1.008	0.996-1.019	0.195
sO2	0.935	0.904-0.967	**<0.001**	1.014	0.952-1.081	0.661
LVEF	0.986	0.974-0.998	**0.025**	0.992	0.964-1.021	0.577
MR	1.341	0.964-1.865	0.081	1.340	0.599-2.997	0.475
AF	1.099	0.964-1.252	0.159			
Albumin	0.447	0.324-0.704	<0.001	0.918	0.496-1.700	0.786
CRP	1.006	1.002-1.010	**0.004**	1.003	0.996-1.009	0.418
Troponin	1.002	1.001-1.003	**0.004**	1.002	1.001-1.003	**0.026**
Positive inotropes	4.192	2.733-6.429	**<0.001**	2.139	0.847-5.404	0.108
MV	3.274	2.456-4.363	**<0.001**	3.346	2.052-5.457	**<0.001**
Bleeding	2.395	1.335-4.297	**0.003**	5.258	0.831-23.266	0.078

Epidemiology

OP-110

Short term prognosis of elderly patients admitted to coronary care unit: A subgroup analyis of MORCOR-TURK trial

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Background and Aim: Current data on the factors predicting short-term prognosis and mortality in elderly patients hospitalized and followed up in the coronary care unit (CCU) in Turkey are very limited. In the subgroup analysis of the study of predictors of MORtality in CCUs in Turkey (MORCOR-TURK), it was aimed to determine the short-term prognosis, mortality rates and predictors of elderly patients followed in CCUs in Turkey.

Methods: The MORCOR-TURK trial is a national, non-interventional, multicenter and observational study conducted in Turkey (ClinicalTrials.gov number NCT05296694). The study population include CCUs patients from 50 centers selected from all regions in Turkey (Between 1 and 30 September 2022 prospectively). In the subgroup analysis of the study, patients were divided into 2 groups: group 1 (65 age <75 years, n=923 patients) and group 2 (age 75 years, n=713 patients). The end of the analysis, short-term prognosis, mortality rates and predictors were documented. The statistical significance value was accepted as p<0.05.

Results: The mean age of group 1 was 69 (67-72) years and group 2 was 80 (77-84) years (p<0.001). The percentage of female gender was higher in group 2. The most common reason for admission was chest pain [968 patients (59,16%)] and the most common reason for hospitalization in the CCU was acute coronary syndrome [1053 patients (64%)]. The second cause of hospitalization in the CCU was heart failure (HF) [259 patients (15.83%)] followed by arrhythmias [171 patients (10.45%)]. Atrial fibrillation (AF) was the most common arrhythmia [356 patients (21.76%)]. The mortality rate was 6.1% in elderly patients (4.22% in group 1 and 8.55% in group 2). The highest mortality rate was found in patients with ventricular tachycardia/fibrillation (VT/VF) and decompensated HF at follow-up. Among the patients who died, the time of CCU stays was longer. In the multivariate regression analysis; aortic stenosis (p<0.001), AF in hospital (p<0.017), VT/VF in hospital (p<0.001), and acute renal failure (p<0.001) were found to be the main factors that increased mortality, while ejection fraction (p<0.019) and mean blood pressure (p<0.001) elevation were found to be protective on mortality.

Conclusions: The subgroup analysis of elderly patients in MORCOR-TURK study is the largest and most comprehensive study in Turkey evaluating the short term prognosis of elderly patients admitted to CCUs.

Coronary Artery Disease / Acute Coronary Syndrome

OP-111

Demographic characteristics and short term prognosis of patients admitted to coronary care unit with acute coronary syndrome (MORCOR-TURK ACS)

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Background and Aim: Acute coronary syndromes (ACS) are the most common cause of death in the world which constitute the largest part of cardiovascular emergencies. In this

study, we investigated the incidence of ACS and its predictors and mortality rate in the largest trial which documented the demographic characteristics and mortality data of coronary care unit (CCU) patients in Turkey (MORCOR-TURK ACS).

Methods: In this multicenter, cross-sectional and national study subgroup analysis, we included all consecutive patients admitted to CCU with the diagnosis of ACS in 50 centers. Demographic characteristics, and laboratory data were recorded. Both ST elevation myocardial infarction (STEMI) and non-STEMI patients were included and also compared to each other for in hospital outcomes. Logistic regression (LR) analysis was used to test the predictors of in hospital mortality. A p value less than 0.05 was considered to be statistically significant for all analyses.

Results: All patients with the diagnosis of ACS (2284 patients) were included in the study. In hospital mortality rate was 3.1% (70 patients) and it was slightly higher in STEMI compared to NSTEMI/USAP [4.2% (n=31) vs. 2.5 (n=39), p=0.038]. While cardiac medications including antiplatelet agents, beta-blockers, ACE inhibitors and statins were similar between survivors and nonsurvivors (all p values are >0.05), nonsurvivors were older [72 (59-80) vs. 63 (54-71), p<0.001] and atrial fibrillation (p=0.012), heart failure (p<0.001) and chronic kidney disease (p<0.001) incidences were higher in nonsurvivors (Table 1). Ejection fraction and mean blood pressure (BP) on admission were lower while basal heart rate (HR) was higher in nonsurvivors (all p<0.001). Basal admission characteristics and laboratory data were shown in Table 2.

Table 1. Demographic Characteristics of Acute Coronary syndrome Patients

	All Patients (n=2284)	Nonsurvivors (n=70)	Survivors (n=2214)	p
Clinical Characteristics				
Age (years)	63 (54-71)	72 (59-80)	63 (54-71)	*<0.001*
Male gender, n (%)	1612 (70.6)	73 (53.3)	2014 (66.7)	*0.002*
Main Diagnosis, n (%) STEMI NSTEMI/USAP	 742 (32.5) 1542 (67.5)	 31 (4.2) 39 (2.5)	 711 (95.8) 1502 (97.5)	*0.038*
CAD History, n (%)	1007 (44.1)	34 (3.4)	36 (2.8)	*0.465*
PCI History, n (%)	775 (33.9)	26 (3.4)	44 (2.9)	*0.608*
MI History, n (%)	684 (29.9)	27 (3.9)	43 (2.7)	*0.113*
CABG History, n (%)	253 (11.1)	9 (3.6)	61 (3)	*0.565*
Atrial Fibrillation, n (%)	124 (5.4)	9 (7.3)	61 (2.8)	*0.012*
Heart Failure, n (%)	453 (19.8)	31 (6.8)	39 (2.1)	*<0.001*
Hypertension, n (%)	1264 (55.3)	41 (3.2)	29 (2.8)	*0.626*
Diabetes Mellitus, n (%)	835 (36.6)	31 (3.7)	39 (2.7)	*0.207*
Dyslipidemia, n (%)	818 (35.8)	21 (2.6)	49 (3.3)	*0.375*
Smoking, n (%) Never Ex smoker Active smoker	 738 (32.3) 590 (25.8) 956 (41.9)	 27 (3.7) 17 (2.9) 26 (2.7)	 711 (96.3) 573 (97.1) 930 (97.3)	*0.515*
Family History, n (%)*	835 (36.6)	20 (2.4)	49 (3.5)	*0.166*
Stroke History, n (%) Ischemic Hemorrhagic	 67 (2.9) 11 (0.5)	 2 (3) 1 (9.1)	 65 (97) 10 (90.9)	*0.509*
CKD, n (%)	211 (9.2)	16 (7.6)	54 (2.6)	*<0.001*
Medication on Admission				
Asetilsalisylic Acid, n (%)	992 (43.4)	33 (3.3)	37 (2.9)	*0.542*
Clopidogrel, n (%)	318 (13.9)	5 (1.6)	313 (98.4)	*0.128*
Prasugrel, n (%)	8 (0.4)	0 (0.0)	8 (100)	
Ticagrelor, n (%)	68 (3.0)	0 (0.0)	68 (100)	
Beta blocker, n (%)	873 (38.2)	31 (3.6)	39 (2.8)	*0.318*
ACEI, n (%)	631 (27.6)	21 (3.3)	610 (96.7)	*0.880*
ARB, n (%)	316 (13.8)	10 (3.2)	306 (96.8)	*0.880*
Statin, n (%)	672 (29.4)	26 (3.9)	44 (2.7)	*0.182*
PPI, n (%)**	889 (38.9)	32 (3.6)	36 (2.6)	*0.208*

*37 patients have missing data about family history
**22 patients have missing data about PPI use

Non-sinus rhythm on admission was higher in nonsurvivors [9 (12.9%) vs. 114 (5.1%), p=0.011]. Clinically and statistically significant variables like age, gender, AF history, HF history, CAD, HT and DM histories, mean BP, HR, potassium level on admission were included in the multivariable model. According to backward LR results; age, gender, potassium level, mean BP and heart rate were found as significant predictors of in hospital mortality (Table 3).

Conclusions: Our study, which is one of the largest studies including ACS patients in our country documented upto-date data of short term outcomes. It can be one of the most important cornerstones to reveal the characteristics of ACS patients and improve their management.

Table 2.

	All Patients (n=2284)	Nonsurvivors (n=70)	Survivors (n=2214)	p
EF (%)	50 (45-60)	40 (30-50)	52 (45-60)	*<0.001*
Heart rate (bpm)	81 (71-94)	90 (80-107)	79 (70-88)	*<0.001*
Mean BP (mmHg)	96 (85-106)	78 (68-95)	96 (86-105)	*<0.001*
Glucose (mg/dL)	124 (102-166)	157 (125-225)	125 (103-170)	*0.011*
Creatinine (mg/dl)	0.95 (0.8-1.2)	1.3 (1.0-1.7)	0.9 (0.8-1.1)	*0.719*
GFR (ml/min)	78 (54-95)	52 (32-79)	81 (59-96)	*<0.001*
Sodium (mEq/L)	138 (136-140)	138 (134-141)	138 (136-140)	*0,734*
Potassium (mEq/L)	4.3 (4-4.8)	4.5 (4.1-5.0)	4.3 (4.0-4.6)	*0.006*
Calcium (mEq/L)	9 (8.7-9.4)	8.8 (8.2-9.1)	9.2 (8.8-9.6)	*<0.001*
Magnesium (mEq/L)	1.9 (1.7-2.1)	1.9 (1.6-2.1)	1.9 (1.8-2.1)	*0.946*
AST	27 (19-47)	37 (20-115)	26 (19-46)	*0.041*
ALT	20 (14-32)	26 (16-52)	20 (14-30)	*0.049*
CRP (mg/L)	6 (2-17)	14 (2-84)	4.4 (1.8-12)	*0.002*
Albumin (g/dL)	4 (3,7-4,3)	3.7 (3.3-4.1)	4,1 (3.8-4,4)	*<0.001*
Hematocrit (%)	40,1 (36-44)	37 (34-43)	41 (37-45)	*<0.001*
WBC (10^3/µL)	9.5 (7.5-11.7)	12.7 (9-15.6)	9.5 (7.7-11.8)	*<0.001*
Platelet count (10^3/µL)	233 (192-289)	232 (181-309)	238 (197-287)	*0.862*
Neutrophil count (10^3/µL)	6,3 (4.7-8.7)	9.1 (6.2-12.6)	6.4 (4.8-9)	*0.159*
Lymphocyte count (10^3/µL)	1.9 (1.3-2.6)	1.8 (1-3)	2 (1.4-2.8)	*0.820*
Troponin admission	67 (6-763)	201 (12-5586)	65 (6-727)	*0.050*
Total cholesterol (mg/dL)	165 (134-202)	152 (119-186)	181 (150-211)	*0.002*
Triglyceride (mg/dL)	117 (83-163)	110 (89-160)	126 (88-188)	*<0.001*
HDL (mg/dL)	39 (33-48)	39 (29-48)	40 (34-48)	*0.481*
LDL (mg/dL)	104 (75-133)	93 (61-124)	112 (85-138)	*0.018*
CHA_2DS_2VASc score	2 (1-4)	3 (2-5)	2 (1-4)	*<0.001*

Continuous variables are presented as mean ± SD or median (IQR), categorical variables are presented as frequency (%)
ALT: alanine transaminase, AST: aspartate trnasaminase, BP: blood presure, bpm: beat per minute; CRP: C-reactive protein; EF: ejection fraction, GFR: glomerular filtration rate, HDL: high density lipoprotein, IQR: interquartile range; LDL: low density lipoprotein, SD: standart deviation; WBC: white blood cell

Table 3. Independent predictors of in-hospital mortality in ACS patients

Variables	Univariable analysis		Multivariable analysis	
	OR (95% CI)	p	OR (95% CI)	p
Age	1.053 (1.031 - 1.075)	<0.001	1.027 (1.004 - 1.050)	0.020
Gender	2.071 (1.283 - 3.344)	0.003	2.038 (1.177 - 3.528)	0.011
Heart Failure	3.375 (2.082 - 5.473)	<0.001		
Atrial Fibrillation	2.693 (1.305 - 5.558)	0.007		
CAD	1.205 (0.748 - 1.939)	0.444		
Hypertension	1.146 (0.707 - 1.857)	0.581		
Diabetes Mellitus	1.394 (0.863 - 2.252)	0.175		
Mean BP	0.922 (0.905 - 0.939)	<0.001	0.929 (0.912 - 0.947)	<0.001
Heart rate	1.040 (1.028 - 1.053)	<0.001	1.033 (1.020 - 1.046)	<0.001
Potassium level	2.141 (1.474 - 3.110)	<0.001	1.721 (1.162 - 2.550)	0.007

Other

OP-112

The predictive ability of CONUT score on in hospital mortality in patients admitted to coronary care unit (MORCOR-TURK CONUT)

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Background and Aim: CONUT score was previously described and has been used to predict short- and long-term outcomes in different patient populations. In this study, we wanted to test the relationship between CONUT score and in-hospital mortality in coronary care unit patients (MORCOR-TURK population).

Methods: In this multicenter and national study, all patients with available CONUT score were included in the analysis. Clinical and laboratory data of patients were recorded. CONUT score was calculated according to previously described criteria. The predictive value of CONUT was evaluated by logistic regression analysis. To be able to understand the significance of CONUT score, we constructed two models. Model 1 included age, heart failure, chronic kidney disease, hypertension, diabetes mellitus, and coronary artery disease history. Model 2 included CONUT score and Model 1. Then we compared the performances of two models using -2 log-likelihood ratio, Nagelkerke R2, and area under the curve (AUC). A p value less than 0.05 was considered to be statistically significant for all analyses.

Results: A total of 1018 patients with known CONUT score were included in the analysis. The mean CONUT score was 2.74 ± 1.9 for the whole population. Demographic characteristics were shown in Table. In hospital mortality rate of the whole population was 5.7% (58 patients) and CONUT score was significantly higher in non-survivors [4 (2-6.3) vs. 2 (1-3.8), p<0.001]. Model 1, -2 log-likelihood ratio was 395.995, Nagelkerke R2 0.133, and AUC 0.739 (95% CI: 0.67-0.81). In the second model to which CONUT score is added (Model 2), -2 log-likelihood ratio was 373.743, Nagelkerke R2 0.191, and AUC was 0.787 (95% CI: 0.72-0.85). The area under the curve value of Model 2 was statistically higher than Model 1 (DeLong p=0.01) (Figure 1). A statistically significant correlation was found between death and CONUT score in Model 2 (OR: 1.347; 1.193-1.521, p<0.001).

Conclusions: Our study showed that CONUT score may significantly predict in-hospital mortality in CCU patients.

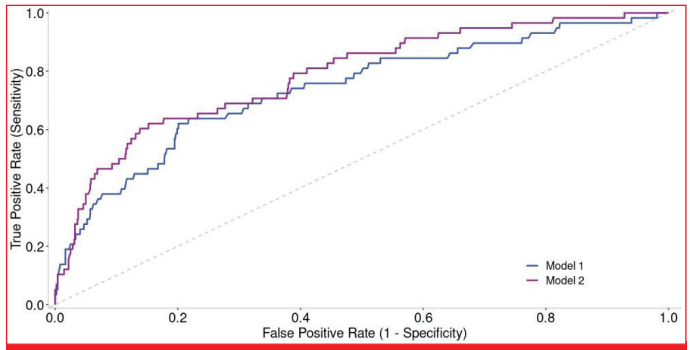

Figure 1. Comparison of Model 1 and Model 2 in predicting in hospital mortality

Model 1: age, heart failure, chronic kidney disease, hypertension, diabetes mellitus, coronary artery disease Model 2: Model 1 + CONUT score.

Table 1. Demographic and clinical characteristics of the patient population

	All patients (n=1018)
Age, years	67 (57-75)
Gender (male), n (%)	663 (65.1)
Hypertension, n (%)	638 (62.7)
Diabetes mellitus, n (%)	376 (36.9)
Dyslipidemia, n (%)	330 (32.4)
Stroke*, n (%)	59 (5.8)
CAD, n (%)	471 (46.3)
AF, n (%)	191 (18.8)
Heart failure, n (%)	355 (34.9)
Chronic renal disease, n (%)	189 (18.36)
EF, %	50 (40-55)
Heart rate, bpm	80 (70-96)
Mean blood pressure, mmHg	94 (83-105)
Glucose, mg/dL	127 (103-173)
Glomerular filtration rate, mL/min	73 (49-93)
Sodium, mEq/L	138 (135-140)
Potassium, mEq/L	4.3 (4-4.8)
Calcium, mEq/L	9 (8.6-9.3)
Magnesium, mEq/L	1.9 (1.7-2.1)
CRP, mg/L	6.8 (2.1-22)
Albumin, g/dL	3.9 (3.5-4.3)
Hematocrit, %	39.4 (35-43.4)
WBC, 10^3/μL	9.3 (7.2-11.6)
Platelet count, 10^3/μL	227 (184-279)
Total cholesterol, mg/dL	152 (125-175)
Triglyceride, mg/dL	108 (77-148)
HDL, mg/dL	38 (32-47)
LDL, mg/dL	90 (67-110)

Continuous variables are presented as mean ± SD or median (IQR), categorical variables are presented as frequency (%). AF: Atrial fibrillation, bpm: Beat per minute, CAD: Coronary artery disease, CRP: C-reactive protein, EF: Ejection fraction, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, WBC: White blood cell. * Includes both ischemic and hemorrhagic stroke cases.

Epidemiology

OP-113

Predictive ability of inflammatory markers on in hospital outcomes in patients admitted to coronary care unit (MORCOR-TURK INFLAME)

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Background and Aim: Inflammatory indices are widely used prognostic markers in acute and chronic disease conditions. Most of the inflammatory markers have been investigated in commonly seen cardiovascular diseases so far. Here, we wanted to investigate the prognostic implications of sytemic immune-inflammatory index (SII), atherogenic index of plasma (AIP), C-reactive protein/albumin ratio (CAR), neutrophil-lymphocyte ratio (NLR), prognostic nutritional index (PNI) and triglyceride/glucose index (TGI) in MORCOR-TURK trial population which is the largest registry of coronary care unit (CCU) patients in Turkey (MORCOR-TURK INFLAME).

Methods: All patients admitted to CCU in 50 different centers in Turkey were included in the analysis of multicenter, cross-sectional and national study. Demographic, clinical and laboratory characteristics of patients were recorded. Besides, all inflammatory indices were calculated according to previously described criteria. The study population was divided into two according to in-hospital survival status. Demographic and clinical characteristics and inflammatory markers were compared between two groups. First, independent samples t-test and chi-square test were used for comparison of numerical and categorical variables. Later, the analysis was continued with model construction method. Model 1 included age, gender, coronary artery disease (CAD), family history of CAD, atrial fibrillation, heart failure, chronic kidney disease, mean blood pressure and heart rate on admission, hypertension and diabetes mellitus. Other models were constructed by adding inflammatory markers to Model 1, respectively (Model 2, Model 3, Model 4, Model 5, Model 6, Model 7). Then we compared the performances of all consecutive models with Model 1 using -2 log-likelihood ratio, Nagelkerke R2 and area under the curve (AUC) method. A p value less than 0.05 was considered to be statistically significant for all analyses.

Results: A total of 3157 patients were investigated and compared according to the basal characteristics and inflammatory markers. One hundred and thirty seven patients (4.3%) died in hospital follow-up. Basal demographic characteristics and laboratory data were shown in Table 1. In Model 1, -2 log-likelihood ratio was 888.439, Nagelkerke R2 0.235 and AUC was 0.814 (95% CI: 0.771-0.858). All other models were constructed by adding each inflammatory marker separately to Model 1. Indeed, statistically significant correlation was found between death and SII, CAR, NLR and PNI but not for AIP and TG/Glucose index in logistic regression (Table 2). But when we compared the AUC change between new models and Model 1, only Model 3 (C-reactive protein/Albumin Ratio + Model 1) AUC was significantly higher than Model 1 (DeLong p=0.01, Figure 1).

Conclusions: Our study showed that CAR but not other inflammatory markers is a significant predictor of in hospital mortality in CCU patients.

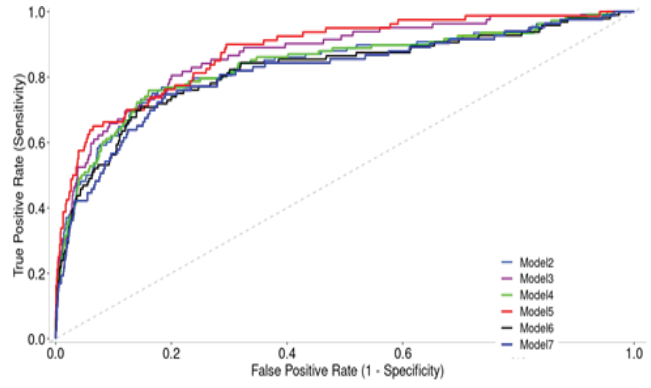

Figure: Comparison of models in which inflammatory markers were added separately

Models	Area Under the Curve	ROC Curve Comparison with Model 1
Model 1	0.814 (95% CI 0.771 - 0.858)	
Model 2	0.836 (95% CI 0.789 - 0.883)	0.61
Model 3	0.869 (95% CI 0.826 - 0.912)	**0.01**
Model 4	0.837 (95% CI 0.790 - 0.884)	0.42
Model 5	0.881 (95% CI 0.841 - 0.921)	0.23
Model 6	0.821 (95% CI 0.769 - 0.873)	0.56
Model 7	0.815 (95% CI 0.759 - 0.871)	0.16

Model 2 : Model 1 + Systemic Immune-Inflammatory Index
Model 3 : Model 1 + C-reactive Protein / Albumin Ratio
Model 4 : Model 1 + Neutrophil-Lymphocyte Ratio
Model 5 : Model 1 + Prognostic Nutritional Index
Model 6 : Model 1 + Atherogenic Index of Plasma
Model 7 : Model 1 + Triglyceride/Glucose index

Figure 1. Comparison of models in which inflammatory markers were added separately.

Table 1. Demographic characteristics of patients according to survival status

	Nonsurvivors (n=137)	Survivors (n=3020)	p
Age, years	73 (63-83)	65 (56-73)	<0.001
Gender (male), n (%)	73 (53.3)	2014 (66.7)	0.002
Hypertension, n (%)	91 (66.4)	1773 (58.7)	0.076
Diabetes mellitus, n (%)	55 (40.1)	1129 (37.4)	0.528
Dyslipidemia, n (%)	46 (33.6)	1074 (35.6)	0.715
Smoking, n (%)			0.043
Never	58 (42.3)	1144 (37.9)	
Ex smoker	45 (32.8)	817 (27.1)	
Active smoker	34 (24.8)	1059 (35.1)	
Family history of CAD, n (%)	37 (27.2)	1064 (35.8)	0.043
CAD, n (%)	74 (54)	1372 (45.4)	0.054
AF, n (%)			0.003
No	103 (75.2)	2571 (85.1)	
Yes	34 (24.8)	449 (14.9)	
Heart failure, n (%)			<0.001
No	61 (44.5)	2118 (70.1)	
Reduced EF HF	60 (43.8)	595 (19.7)	
Preserved EF HF	16 (11.7)	307 (10.2)	
Stroke history, n (%)			0.086
No	126 (92)	2889 (95.7)	
Ischemic stroke	9 (6.6)	116 (3.8)	
Hemorrhagic stroke	2 (1.5)	15 (0.5)	
Prior major bleeding, n (%)	6 (4.5)	124 (4.2)	0.823
Chronic renal disease, n (%)			<0.001
No	92 (67.2)	2631 (87.1)	
Yes	45 (32.8)	3289 (12.9)	
Main admission diagnosis			<0.001
STEMI	31 (22.6)	711 (23.5)	
NSTEMI	31 (22.6)	1156 (38.3)	

Continuous variables are presented as mean ± SD or median (IQR), categorical variables are presented as frequency (%). bpm: Beat per minute, BUN: Blood urea nitrogen, CAD/PAD: Coronary artery disease/peripheral artery disease, CRP: C-reactive protein, IQR: Interquartile range, SD: Standard deviation, WBC: White blood cell.

Table 2. Comparison of Model 1 and constructed models by adding inflammatory markers separately

Models	-2 Log-likelihood ratio	Nagelkerke R2	Odds Ratio*	p*
Model 1	888.439	0.235		
Model 2	667.852	0.277	1.000 (1.000-1.000)	0.002
Model 3	462.730	0.334	1.030 (1.018-1.043)	<0.001
Model 4	665.881	0.280	1.044 (1.019-1.069)	<0.001
Model 5	429.935	0.370	0.901 (0.860-0.944)	<0.001
Model 6	626.390	0.245	1.195 (0.837-1.706)	0.328
Model 7	570.834	0.224	1.000 (1.000-1.000)	0.994

Model 2: Model 1 + Systemic immune-inflammatory Index, Model 3: Model 1 + C-reactive protein / albumin ratio, Model 4: Model 1 + Neutrophil-lymphocyte ratio, Model 5: Model 1 + Prognostic nutritional index, Model 6: Model 1 + Atherogenic index of plasma, Model 7: Model 1 + Triglyceride/glucose index. *Odds ratios and p values specifically indicates the statistical values of the inflammatory markers included in the model.

Other

OP-114

Baseline characteristics and prognosis of atrial fibrillation patients in coronary care unit: A subgroup analysis of MORCOR-TURK trial

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Background and Aim: The MORCOR-TURK trial is a national registry evaluating predictors and rates of in-hospital mortality in coronary care unit (CCU) patients in Turkiye. This report describes the baseline demographic characteristics of patients with atrial fibrillation which is a subgroup analysis of MORCOR-TURK trial.

Methods: The MORCOR-TURK trial is a multicenter, cross-sectional, prospective, and national registry included 50 centers capable of 24-hour CCU service selected from all seven geographic regions in Turkiye. All consecutive patients admitted to CCUs with cardiovascular emergencies between 1-30 September 2022 were prospectively enrolled. Baseline demographic characteristics, admission diagnoses, laboratory data, and CV risk factors of patients with merely AF rhythm were selected and analyzed. Patients were divided into two groups as atrial fibrillation at admission and atrial fibrillation rhythm during hospitalization and groups were compared.

Results: A total of 540 patients with mean age of 71.4 ± 11.6 and 254 (47%) males were included in the analysis. Diabetes mellitus rate was higher in chronic AF group. Other demographic features were similar. Chronic kidney disease was tendence to be higher in AF at admission group. In terms of diagnoses, acute coronary syndrome rate was higher in chronic AF group, whereas heart failure rate was higher in newly diagnosed AF group. Newly diagnosed AF patients admitted with dyspnea complaint more than another group. Decrease in hemoglobin level and increase in CRP were found to be a basis for newly diagnosis of AF, whereas elevation in cholesterol levels was related to chronic AF.

Conclusions: This is the first CCU follow-up registry in Turkiye and in over the world. Considering AF has high morbidity and mortality risk in normal population and even higher in CCU patients, it is crucial to detect precipitator or basis factors to prevent development of this undesired clinical condition. Long term follow-up of patients in this study would provide more clinically applicable results.

Table 1. Atrial fibrillation

	All patients (n:540)	AF at admission (n=413)	AF during hospitalization (n=127)	p
Age (years), St dev.	71.4 11.6	72.7 11.4	67.1 11.4	<0.001
Age (years), IQR	72(65-80)	74(67-81)	67(59-76)	<0.001
Male gender, n (%)	254 (47%)	183(44.3%)	71(55.9%)	0.025
Hypertension, n (%)	383(71%)	300(72.6%)	83(65.4%)	0.119
Diabetes Mellitus, n (%)	217(40.2%)	155(37.5%)	62(48.8%)	0.030
Dyslipidemia, n (%)	174(32.2%)	129(31.2%)	45(35.7%)	0.384
Smoking, n (%)				
Never	314(58.1%)	257(62.2%)	57(44.9%)	<0.001
Ex smoker	152(28.1%)	111(26.9%)	41(32.3%)	
Active smoker	74(13.7%)	45(10.9%)	29(22.8%)	
CAD, n (%)	241(44.6%)	177(42.9%)	64(50.4%)	0.153
PCI, n (%)	169(31.3%)	120(29.2%)	49(38.6%)	0.049
MI, n (%)	148(27.4%)	104(25.2%)	44(34.6%)	0.041
CABG, n (%)	75(13.9%)	58(14%)	17(13.4%)	0.999
PAD, n (%)				
No	338(62.6%)	253(61.3%)	85(66.9%)	0.301
Yes	20(3.7%)	14(3.4%)	6(4.7%)	
Unknown	182(33.7%)	146(35.4%)	36(28.3%)	
Heart failure, n (%)				
No	210(38.9%)	134(32.4%)	76(59.8%)	<0.001
Reduced EF HF	199(36.9%)	160(38.7%)	39(30.7%)	
Preserved EF HF	131(24.3%)	119(28.8%)	12(9.4%)	
Stroke history, n (%)				
No	489(90.6%)	375(90.8%)	114(89.8%)	0.477
Ischemic Stroke	44(8.1%)	34(8.2%)	10(7.9%)	
Hemorrhagic Stroke	7(1.3%)	4(1%)	3(2.4%)	
Prior Major Bleeding, n (%)	27(5%)	21(5.2%)	6(4.8%)	0.999
Chronic Renal Disease, n (%)				
No	406(75.2%)	299(72.4%)	107(84.3%)	0.002
Hemodyalisis (-)	125(23.1%)	109(26.4%)	16(12.6%)	
Hemodyalisis (+)	9(1.7%)	5(1.2%)	4(3.1%)	
Mortality, n(%)	40(7.4%)	29(7%)	11(8.7%)	0.562
Main Admission Diagnosis				
STEMI	47(8.7%)	16(3.9%)	31(24.4%)	<0.001
NSTEMI/USAP	129(23.9%)	76(18.4%)	53(41.7%)	
Decompensated HF	213(39.4%)	191(46.2%)	22(17.3%)	
Arrhythmia	118(21.9%)	102(24.7%)	16(12.6%)	
Cardiac Arrest	3(0.6%)	2(0.5%)	1(0.8%)	
Other diagnoses	30(5.6%)	26(6.3%)	4(3.1%)	
Drug use on Admission				
Asetilsalicylic acid, n (%)	256(52.6%)	89(21.5%)	66(52%)	<0.001
Other Antiplatelets, n (%)	181(33.5%)	27(6.5%)	26(20.5%)	<0.001
Oral Anticoagulants, n (%)	309(57.2%)	292(70.7%)	17(13.4%)	<0.001
Beta blockers, n (%)	407(75.4%)	315(76.3%)	92(72.4%)	0.410
ACE inhibitors/ARB, n (%)	278(51.5%)	207(50.1%)	71(55.9%)	0.401
Calcium Channel Blockers, n (%)	110(20.4%)	91(22%)	19(15%)	0.101
Statins	143(26.5%)	96(23.2%)	47(37%)	0.016
Proton Pump Inhibitors, n (%)	500(92.6%)	259(63.3%)	60(48.4%)	0.003
admission complaint				
chest pain, n (%)	160(29.6%)	84(20.3%)	76(59.8%)	<0.001
dyspnea, n (%)	240(44.6%)	213(51.6%)	28(22%)	
syncope/presyncope, n (%)	24(4.4%)	18(4.4%)	6(4.7%)	
palpitation, n (%)	77(14.3%)	69(16.7%)	8(6.3%)	
sudden death, n (%)	3(0.6%)	1(0.2%)	2(1.6%)	
other, n (%)	34(6.3%)	27(6.5%)	7(5.5%)	

Continuous variables are presented as mean ± SD or median (IQR), categorical variables are presented as frequency (%)

bpm: beat per minute; BUN: blood urea nitrogen; CAD/PAD: Coronary Artery Disease/Peripheral Artery Disease; CRP: C-reactive protein; IQR: interquartile range; SD: standart deviation; WBC: white blood cell

Table 2. Atrial fibrillation 2

	All patients (n:540)	AF at admission (n=413)	AF during hospitalization (n=127)	p
Age (years), St dev.	71.4 11.6	72.7 11.4	67.1 11.4	<0.001
Age (years), IQR	72(65-80)	74(67-81)	67(59-76)	<0.001
Male gender, n (%)	254 (47%)	183(44.3%)	71(55.9%)	0.025
Hypertension, n (%)	383(71%)	300(72.6%)	83(65.4%)	0.119
Diabetes Mellitus, n (%)	217(40.2%)	155(37.5%)	62(48.8%)	0.030
Dyslipidemia, n (%)	174(32.2%)	129(31.2%)	45(35.7%)	0.384
Smoking, n (%)				
Never	314(58.1%)	257(62.2%)	57(44.9%)	<0.001
Ex smoker	152(28.1%)	111(26.9%)	41(32.3%)	
Active smoker	74(13.7%)	45(10.9%)	29(22.8%)	
CAD, n (%)	241(44.6%)	177(42.9%)	64(50.4%)	0.153
PCI, n (%)	169(31.3%)	120(29.2%)	49(38.6%)	0.049
MI, n (%)	148(27.4%)	104(25.2%)	44(34.6%)	0.041
CABG, n (%)	75(13.9%)	58(14%)	17(13.4%)	0.999
PAD, n (%)				
No	338(62.6%)	253(61.3%)	85(66.9%)	0.301
Yes	20(3.7%)	14(3.4%)	6(4.7%)	
Unknown	182(33.7%)	146(35.4%)	36(28.3%)	
Heart failure, n (%)				
No	210(38.9%)	134(32.4%)	76(59.8%)	<0.001
Reduced EF HF	199(36.9%)	160(38.7%)	39(30.7%)	
Preserved EF HF	131(24.3%)	119(28.8%)	12(9.4%)	
Stroke history, n (%)				
No	489(90.6%)	375(90.8%)	114(89.8%)	0.477
Ischemic Stroke	44(8.1%)	34(8.2%)	10(7.9%)	
Hemorrhagic Stroke	7(1.3%)	4(1%)	3(2.4%)	
Prior Major Bleeding, n (%)	27(5%)	21(5.2%)	6(4.8%)	0.999
Chronic Renal Disease, n (%)				
No	406(75.2%)	299(72.4%)	107(84.3%)	0.002
Hemodyalisis (-)	125(23.1%)	109(26.4%)	16(12.6%)	
Hemodyalisis (+)	9(1.7%)	5(1.2%)	4(3.1%)	
Mortality, n(%)	40(7.4%)	29(7%)	11(8.7%)	0.562
Main Admission Diagnosis				
STEMI	47(8.7%)	16(3.9%)	31(24.4%)	<0.001
NSTEMI/USAP	129(23.9%)	76(18.4%)	53(41.7%)	
Decompensated HF	213(39.4%)	191(46.2%)	22(17.3%)	
Arrhythmia	118(21.9%)	102(24.7%)	16(12.6%)	
Cardiac Arrest	3(0.6%)	2(0.5%)	1(0.8%)	
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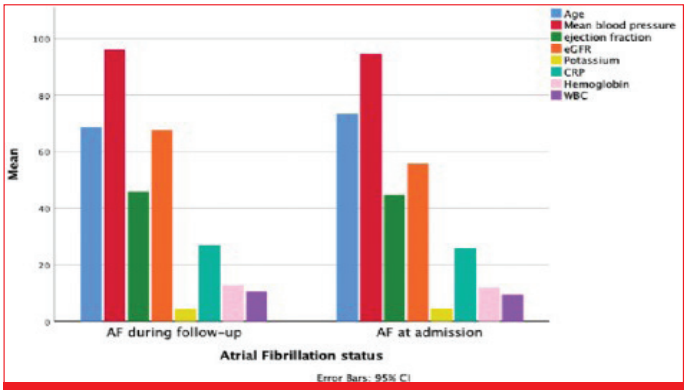

Figure 1. Atrial fibrillation status

Other

OP-115

Predictors of in hospital MORtality in CORonary care patients in Türkiye: MORCOR-TURK trial

İbrahim Ersoy[1], Ahmet Seyda Yılmaz[2], Fatih Kahraman[3], Özgen Şafak[4], Şahin Topuz[5], Faruk Kara[6], Mürsel Şahin[7], Feyza Kurt[8], Fulya Avcı Demir[9], Sefa Erdi Ömür[10], Ertan Aydın[11], Mehtap Yeni[12], Mustafa Begenç Taşcanov[13], Yunus Emre Yavuz[14], İdris Buğra Cerik[15], Şaban Keleşoğlu[16], Yücel Kaçmaz[17], Adem Atıcı[18], Melisa Uçar[19], İshak Yılmaz[20], Sevil Gülaştı[21], Burak Acar[22], İsmail Barkın Işık[23], İbrahim Halil Tanboga[24]

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Background and Aim: Coronary artery disease (CAD) is a leading cause of mortality worldwide, and the coronary care unit (CCU) plays a crucial role in providing intensive care for patients with acute myocardial infarction and other acute coronary syndromes. This study aims to investigate the mortality rates in Turkish CCUs and identify predictors of death among these patients.

Methods: We conducted a national, observational, multicenter, and noninterventional study between September 1, 2022, and September 30, 2022, registered under the Clini-

calTrials.gov number NCT05296694. The study included all patients admitted to CCUs in Turkey with cardiovascular diagnoses. Data on patient demographics, comorbidities, laboratory results, imaging findings, treatments, and outcomes were collected. Multivariate logistic regression analysis was performed to identify predictors of mortality.

Results: A total of 3157 patients were included in the study, and the overall CCU mortality rate was 4.3% (n=137). Male patients accounted for 66.1% of the cohort, and the mean age was 65 years (IQR: 56-73). The most common comorbidities were hypertension (59.8%) and diabetes mellitus (37.5%) (Table 1). Significant differences were observed between nonsurvivors and survivors regarding admission diagnoses (Figure 1). Nonsurvivors had higher proportions of patients admitted with acute pulmonary edema (8% vs. 2.9%, p=0.003), decompensated heart failure (20.4% vs. 10.3%, p=0.001), cardiac arrest (6.6% vs. 0.3%, p<0.001), and non-ST elevation myocardial infarction (NSTEMI) (22.6% vs. 38.3%, p<0.001). Percutaneous coronary intervention (34.5%) was the most frequently used treatment. Multivariate logistic regression analysis revealed that age, male sex, diabetes mellitus, Killip class >I, decreased left ventricular ejection fraction, AF, HF and, CKD were independent predictors of CCU mortality (Table 2).

Conclusions: The CCU mortality rate in Turkey remains relatively low, and several predictors of mortality were identified in this study, including age, male sex, comorbidities such as diabetes mellitus, severity of presentation, and laboratory values. Recognizing these predictors may assist healthcare providers in optimizing treatment strategies and improving outcomes for patients with acute coronary syndromes.

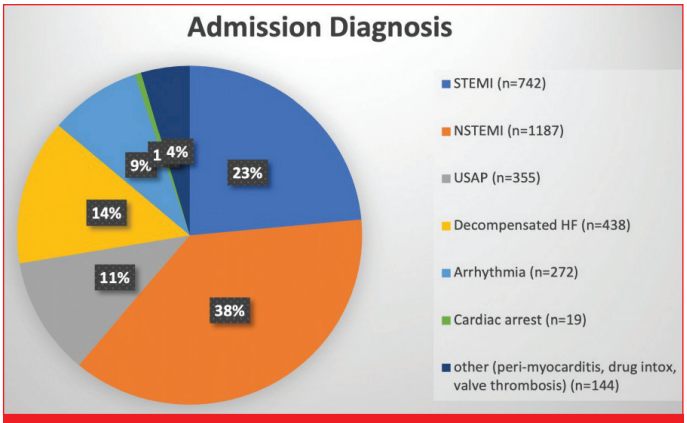

Figure 1. Main admission diagnosis percentages of the study population.

Table 1. Baseline characteristics of the study group

	All patients (n=3157)	Nonsurvivors (n=137)	Survivors (n=3020)	p
Age, years	65 (56-73)	73 (63-83)	65 (56-73)	<0.001
Gender (male), n (%)	2087 (66.1)	73 (53.3)	2014 (66.7)	0.002
Hypertension, n (%)	1864 (59)	91 (66.4)	1773 (58.7)	0.076
Diabetes mellitus, n (%)	1184 (37.5)	55 (40.1)	1129 (37.4)	0.528
Dyslipidemia, n (%)	1120 (35.5)	46 (33.6)	1074 (35.6)	0.715
Smoking, n (%)	1093 (34.6)	37 (24.8)	1059 (35.1)	0.043
Family History of CAD, n (%)	1101 (34.9)	37 (27.2)	1064 (35.8)	0.043
CAD, n (%)	1446 (45.8)	74 (54)	1372 (45.4)	0.054
AF, n (%)	1483 (15.3)	34 (24.8)	449 (14.9)	0.004
Heart failure, n (%)	978 (31)	76 (55.5)	902 (29.9)	<0.001
Stroke history, n (%)	142 (4.5)	11 (8)	131 (4.3)	0.086
Prior major bleeding, n (%)	130 (4.1)	6 (4.5)	124 (4.2)	0.823
Chronic renal disease, n (%)	434 (13.7)	45 (8)	389 (12.9)	<0.001
EF, %	52 (40-57)	40 (30-50)	53 (44-58)	<0.001
Heart rate, bpm	81 (71-94)	96 (80-110)	80 (70-93)	<0.001
Saturation, %	95 (93-98)	94 (86-96)	96 (93-98)	<0.001
Glucose, mg/dL	124 (102-166)	151 (114-212)	123 (101-164)	<0.001
Glomerular filtration rate, mL/min	78 (54-95)	39 (22.7-78)	79 (57-95)	<0.001
AST	27 (19-47)	29 (16-90)	27 (19-46)	0.002
ALT	20 (14-32)	24 (12-73)	20 (14-32)	0.001
CRP, mg/L	6 (2-17)	23.6 (10-120)	5.7 (1.9-16)	<0.001
Troponin	191 (23-3131)	543 (36-8300)	180 (23-2977)	0.088
CHA2DS2VASc score	3 (1-4)	4 (3-5)	3 (1-4)	<0.001

Continuous variables are presented as mean ± SD or median (IQR), categorical variables are presented as frequency (%). bpm: Beat per minute, AF: Atrial fibrillation, CAD: Coronary artery disease, EF: Ejection fraction, AST: Aspartate aminotransferease, ALT: Alanine aminotransferease, CRP: C-reactive protein, IQR: interquartile range, SD: Standard deviation, WBC: White blood cell.

Table 2. Univariate and multivariate logistic regression analysis of predictors for the mortality in coronary care unit

	Univariate			Multivariate		
	OR	95% CI	p	OR	95% CI	p
Age, years	1.050	1.035-1.066	<0.001	1.040	1.010-1.070	0.008
Gender (male)	0.569	0.403-0.803	0.001	0.307	0.146-0.645	0.002
Killip Class	-	-	<0.001	-	-	<0.001
Class I	0.022	0.012-0.041	<0.001	0.031	0.016-0.134	<0.001
Class II	0.058	0.030-0.112	<0.001	0.087	0.031-0.241	<0.001
Class III	0.180	0.096-0.338	<0.001	0.182	0.065-0.503	0.001
Diabetes mellitus	0.891	0.628-1.263	0.516	1.856	1.033-3.335	0.038
Atrial fibrillation	1.894	1.269-2.827	0.002	1.576	1.460-1.721	<0.001
Heart failure	2.929	2.072-4.139	<0.001	10.152	6.315-16.320	<0.001
Chronic kidney disease	3.307	2.279-4.798	<0.001	1.045	1.004-1.504	0.013
Acute renal failure	13.822	9.621-19.858	<0.001	-	-	-
eGFR	0.969	0.962-0.976	<0.001	-	-	-
LV EF	0.943	0.931-0.956	<0.001	0.973	0.948-0.998	0.037

OR: Odds ratio, CI: Confidence interval, eGFR: Glomerular filtration rate, LV EF: Left ventricular ejection failure.

Other

OP-116

The predictors of length of stay and its relationship with in hospital mortality in patients admitted to coronary care unit (MORCOR-TURK TIME)

Alpin Mert Tekin[1], Aslıhan Merve Toprak[2], Faruk Kara[3], İdris Buğra Çerik[4], Mustafa Yenerçağ[5], Nazif Aygül[2], Fatih Kahraman[6], İbrahim Halil Tanboğa[7]

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[5]*Department of Cardiology, Ordu University Faculty of Medicine, Ordu*
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Background and Aim: Prolonged length of stay (LoS) in intensive care unit is significantly associated with mortality and increased resource utilization. In this study, we assessed the independent predictors of LoS and the relation of LoS with in-hospital mortality in coronary care unit (CCU) patients (MORCOR-TURK population).

Methods: Consecutive patients admitted to CCU in 50 centers were included in this study. Demographics characteristics, and laboratory data were recorded. In hospital stay was calculated as days. Poisson regression analysis was used to determine the factors associated with LoS. The relationship between LoS and predictors was evaluated with the risk ratio and 95% confidence interval. A p value less than 0.05 was considered to be statistically significant for all analyses.

Results: A total 2268 patients with known CCU follow up time were included in the analysis. The median length of CCU stay was 1.83 (1-2.37) days (Figure 1). Demographic characteristics were shown in Table 1 and median follow up time and mortality rates according to main admission diagnoses were shown in Figure 2 and Table 2. In hospital mortality rate of whole population was 4.2% (95 patients) and LoS in CCU was significantly higher in non-survivors [2 (0.88-7) days vs. 1.8 (1-2.25) days, p<0.001]. In multivariable poisson regression analysis, main admission diagnosis, heart failure history, blood WBC, CRP and potassium levels, mean blood pressure, chronic kidney disease, age, smoking and HT were found as statistically significant predictors for LoS (Figure 3). The main admission diagnosis, HF history and potassium level can explain at least 50% of the variation in the LoS.

Conclusions: Our study showed that main admission diagnosis, HF history and blood potassium level are most significant predictors of prolonged CCU follow-up. Interventions for these factors may shorten the length of hospital stay.

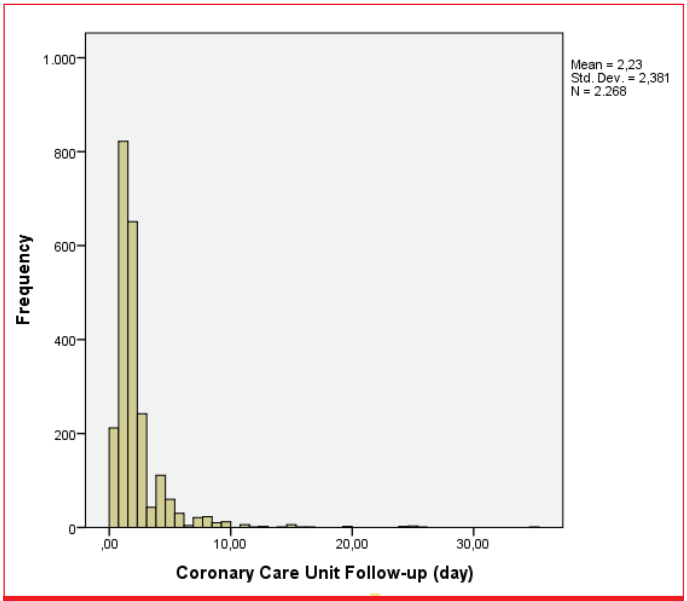

Figure 1. Patient numbers according to their follow-up times in CCU.

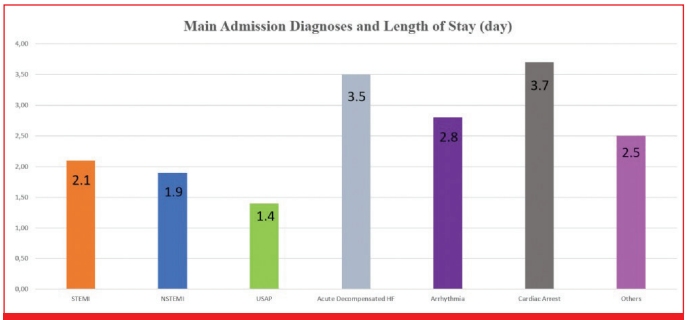

Figure 2. Length of stay (in days) in CCU according to main admission diagnoses.

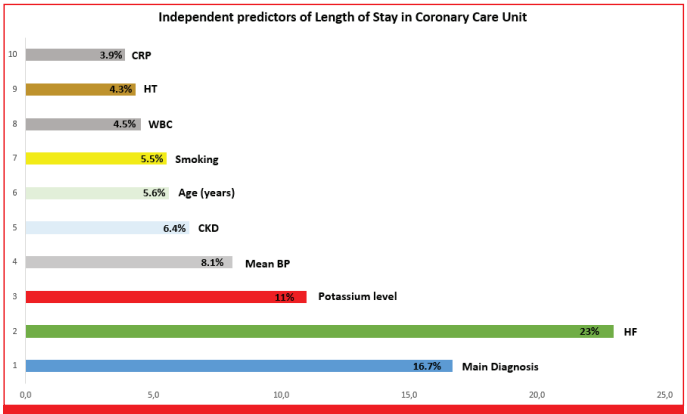

Figure 3. Statistically significant predictors for length of stay in CCU.

Table 1. Demographic and clinical characteristics of patient population (n=2268)	
Age, years	65 (56-74)
Gender (male), n (%)	1524 (67.2)
Hypertension, n (%)	1351 (59.6)
Diabetes mellitus, n (%)	841 (37.1)
Dyslipidemia, n (%)	779 (34.3)
Smoking, n (%)	799 (35.2)
CAD, n (%)	1000 (44.1)
AF, n (%)	329 (14.5)
Heart failure, n (%)	706 (31.1)
Chronic renal disease, n (%)	303 (13.3)
EF, %	52 (40-57)
Heart rate, bpm	80 (70-94)
Mean blood pressure, mmHg	96 (86-108)
Glucose, mg/dL	123 (102-164)
Creatinine, mg/dL	0.95 (0.8-1.2)
Glomerular filtration rate, mL/min	78 (56-95)
Sodium, mEq/L	138 (136-140)
Potassium, mEq/L	4.3 (4-4.8)
Calcium, mEq/L	9.1 (8.7-9.4)
Magnesium, mEq/L	1.9 (1.7-2.1)
CRP, mg/L	5.9 (2-16)
Hematocrit (%)	40.6 (36-44)
WBC, $10^3/\mu L$	9.4 (7.4-11.7)
Albumin, g/dL	4 (3.6-4.3)
Total cholesterol, mg/dL	166 (135-202)
Triglyceride, mg/dL	116 (82-164)
HDL, mg/dL	39 (33-48)
LDL, mg/dL	104 (75-131)
Troponin	141 (20-2362)
CHA2DS2VASc score	3 (1-4)

Table 2. Mortality rates according to the main admission diagnoses	
Diagnosis	**Mortality, n (%)**
STEMI	28/565 (5)
NSTEMI	18/885 (2)
USAP	6/232 (2.6)
Acute decompensated HF	25/292 (8.6)
Arrhythmia	8/185 (4.3)
Cardiac arrest	6/11 (54.5)
Other	4/98 (4.1)

Interventional Cardiology / Coronary

OP-117

Low direct bilirubin levels are associated with contrast induced acute kidney injury in patients with acute coronary syndrome

Onur Kaypaklı

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Background and Aim: Reactive oxygen species have been implicated in the pathogenesis of contrast-induced acute kidney injury. Bilirubin has antioxidant properties as indicated by its ability to scavenge peroxyl radicals. We aimed to investigate the association of bilirubin levels and renal function deterioration after percutaneous coronary intervention for acute coronary syndrome.

Methods: We included 90 patients who underwent percutaneous coronary intervention with the diagnosis of acute coronary syndrome (57 male, 33 female; mean age 59.6 ± 13.0 years). Contrast induced acute kidney injury was defined as an absolute increase in serum creatinine levels by ≥0.5 mg/dL or a relative increase in serum creatinine by ≥25% from baseline within 48 hours after contrast exposure.

Results: Contrast induced acute kidney injury was detected in 17 patients were divided into two groups according to occurrence of CI-AKI. All parameters were compared between the two groups. Contrast volume was significantly higher and direct bilirubin was significantly lower in patients with CI-AKI (p<0.05 for all). Every 0.01 unit increase in direct bilirubin was found to be associated with 10.3% decrease in the risk of CI-AKI. The cut-off value of direct bilirubin obtained by ROC curve analysis was 0.155 for prediction of CI-AKI (sensitivity: 70.6%, specificity: 75.0%). The area under the curve (AUC) was 0.715 (p=0.008).

Conclusions: High levels of direct bilirubin may have a protective role against CI-AKI with its predefined antioxidant effect.

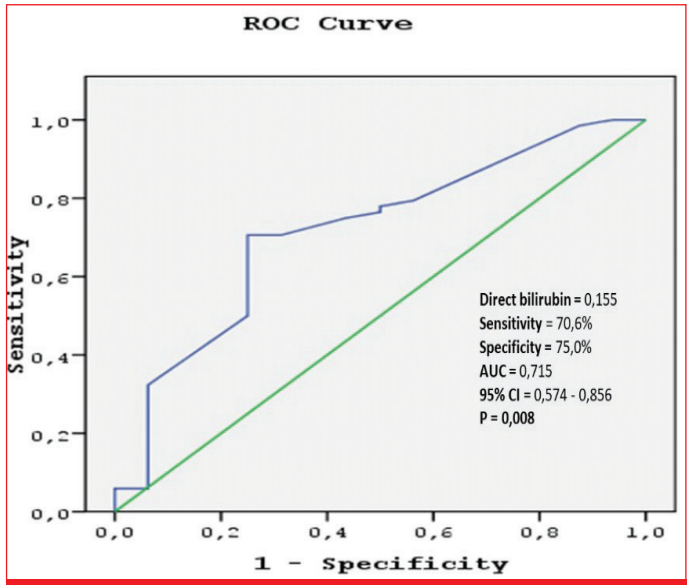

Figure 2. ROC curve analysis to determine predictive value of direct bilirubin for occurrence of contrast induced acute kidney injury

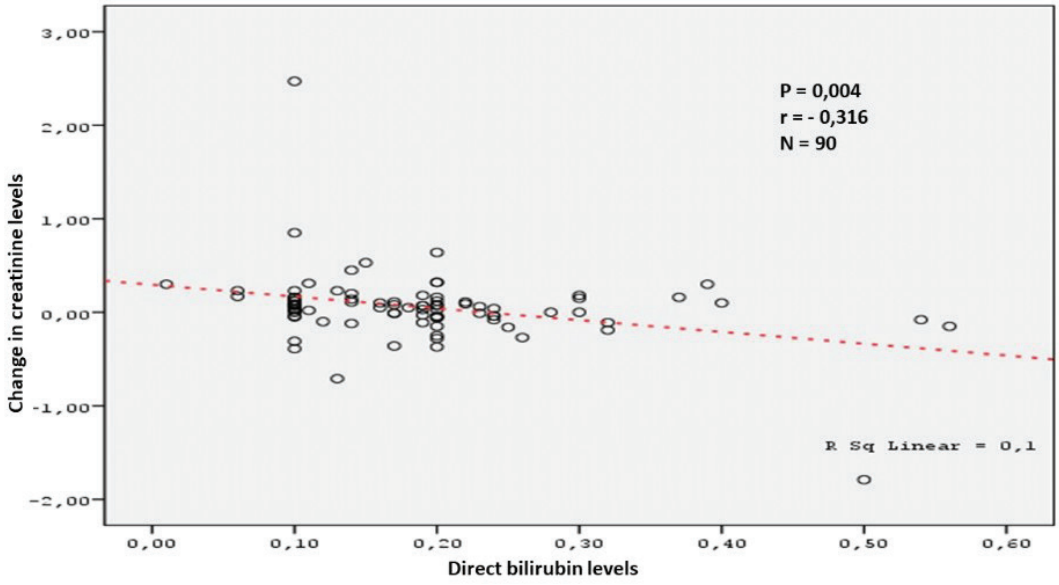

Figure 1. Scatter plot diagram of the relationship of direct bilirubin levels and creatinine increase in 48 hours.

Table 1. Baseline characteristics of patients with and without contrast induced acute kidney injury (CI-AKI)

	Patients without CI-AKI (n=71)	Patients with CI-AKI (n=19)	p
Age, years	58.4 ± 12.7	64.1 ± 13.8	0.093
Gender (female/male	23/48	10/9	0.104
Body mass index, kg/m^2	26.2 ± 4.4	27.1 ± 2.6	0.445
Systolic BP, mmHg	124.4 ± 14.9	135.0 ± 26.8	0.214
Diastolic BP, mmHg	78.4 ± 11.7	81.6 ± 13.3	0.431
Heart rate, bpm	83.6 ± 12.0	85.8 ± 17.8	0.632
Smoking, n (%)	29 (40.8)	5 (26.3)	0.246
Diabetes, n (%)	26 (36.6)	8 (42.1)	0.661
Hypertension, n (%)	45 (63.4)	10 (52.6)	0.393
ACS type (NSTE-AKS/STEMI)	27/44	5/14	0.343
LV ejection fraction, %	44.3 ± 9,9	45.9 ± 7.4	0.513
Contrast volume, mL	161.0 ± 66	228.9 ± 147	0.004
Duration of procedure, min	33.0 ± 12.1	46.5 ± 28.5	0.058
Prior ACEi or ARB use, n (%)	8 (11.3)	2 (10.5)	0.927
Prior beta-blocker use, n (%)	5 (7.0)	3 (15.8)	0.234
Prior ASA use, n (%)	13 (18.3)	1 (5.3)	0.163
Prior ADP antagonist use, n (%)	2 (2.8)	0	0.459
Prior statin use, n (%)	5 (7.0)	1 (5.3)	0.782

ACS: Acute coronary syndrome, ACEi: Angiotensin converting enzyme inhibitors, ARB: Angiotensin receptor blockers, ASA: Acetylsalicylic acid.

Table 2. Laboratory variables of patients with and without contrast induced acute kidney injury (CI-AKI)

	Patients without CI-AKI (n=71)	Patients with CI-AKI (n=19)	p
Hemoglobin, g/dL	14.1 ± 1.84	13.4 ± 2.12	0.141
Baseline GFR, mL/min/1.73 m^2	85.5 ± 24.6	81.2 ± 23.2	0.416
Baseline creatinine, mg/dL	0.94 ± 0.41	0.86 ± 0.23	0.419
48h creatinin increase, mg/dL	0.04 ± 0.26	0.48 ± 0.53	0.000
LDL cholesterol, mg/dL	108.0 ± 34	95.2 ± 30	0.142
HDL cholesterol, mg/dL	38.6 ± 9.1	41.5 ± 13.6	0.417
Triglyceride, mg/dL	165.7 ± 87	163.1 ± 90	0.909
Uric acid, mg/dL	6.35 ± 1.87	6.39 ± 1.60	0.939
Glucose, mg/dL	158.2 ± 80.9	154.0 ± 96.8	0.845
Hb A1c, %	7.1 ± 2.2	7.0 ± 2.2	0.935
Albumin, g/dL	4.04 ± 0.46	3.96 ± 0.35	0.542
Total bilirubin, mg/dL	0.68 ± 0.31	0.59 ± 0.31	0.291
Direct bilirubin, mg/dL	0.20 ± 0.10	0.13 ± 0.08	0.021
Indirect bilirubin, mg/dL	0.49 ± 0.24	0.41 ± 0.23	0.247

LDL: Low density lipoprotein, HDL: High density lipoprotein

Table 3. Multivariate logistic regression analysis to determine the independent predictors of contrast induced acute kidney injury (CI-AKI)

Variables	p	Odds ratio	95% CI	
			Lower	Upper
Direct bilirubin	0.021	0.897	0.818	0.984
Contrast volume	0.615	1.464		