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Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-001

Incidence and predictors of clinical outcomes in real-life patients with atrial fibrillation treated with oral factor Xa inhibitors: The follow-up results of the ANATOLIA-AF study

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Background and Aim: The main objective of this study is to determine the incidence and predictors of clinical outcomes in patients with atrial fibrillation (AF) treated with factor Xa inhibitors in a real-world setting.

Methods: The ANATOLIA-AF study is a multicenter and observational study that included outpatients with AF. The primary outcome was the risk-benefit balance between major bleeding, thromboembolism, and mortality as reflected by the ''net clinical outcome''. The net clinical outcome is defined as the composite outcome of ischemic stroke, transient ischemic attack, systemic embolism, major bleeding, and all-cause mortality.

Results: A total of 1162 patients with AF treated with factor Xa inhibitors from 26 cardiology centres were included in the present study, with a median age of 72 years (range: 28-96 years). During the median 12 months (IQR=2) follow-up period, 43 patients (3.7%) had ischemic stroke, TIA, and/or systemic embolism and 36 patients (3.1%) had major bleeding. A total of 140 (12.0%) patients died. Overall, the frequency of net clinical outcomes (composite of ischemic stroke, TIA, systemic embolism, major bleeding, and/or all-cause mortality) was 16.8% (195 patients) among the study population. Treatment with rivaroxaban compared with apixaban and edoxaban showed a lower rate of ischemic stroke, TIA, and/or systemic embolism during the follow-up period (2.2% vs. 4.7% vs. 6.5%, respectively, p=0.014). The major bleeding rate was similar between all three factor Xa inhibitors. The all-cause mortality rate in the rivaroxaban group was lower compared with apixaban and edoxaban group (9.8% vs. 15.1% vs. 12.4%, respectively, p=0.042) (Figure 2). Overall, the frequency of net clinical outcomes was 13.8% for patients treated with rivaroxaban, 19.6% for patients treated with apixaban, and 20.6% for patients treated with edoxaban (p=0.019) (Figure 3, 4). In the logistic regression model, the presence of advanced age (OR: 2.01; 95% CI, 1.07-3.79, p=0.030 for patients between 65 and 74 years, and OR: 2.76; 95% CI, 1.43-5.33, p=0.002 for patients ≥75 years), male sex (OR: 1.83; 95% CI, 1.27-2.64, p=0.001), low body weight (≤60 kg) (OR: 2.35; 95% CI, 1.31-4.21, p=0.004), high bleeding risk (HAS-BLED score ≥3) (OR: 1.95; 95% CI, 1.24-3.08, p=0.004), chronic heart failure (OR: 1.99; 95% CI, 1.39-2.84, p<0.001), hypertension (OR: 1.80; 95% CI, 1.10-2.95, p=0.018), chronic liver failure (OR: 4.98; 95% CI, 1.24-19.88, p=0.023), and treatment with apixaban 2.5 mg b.i.d. (OR: 2.59; 95% CI, 1.13-5.92, p=0.023) were independently associated with the development of ischemic stroke, TIA, systemic embolism, major bleeding, and/or all-cause mortality among study population (Figure 5).

Conclusions: The follow-up data from the ANATOLIA-AF study provides evidence from clinical practice to support the safety and effectiveness of factor Xa inhibitors and the incidence and predictors of clinical outcomes in patients with AF.

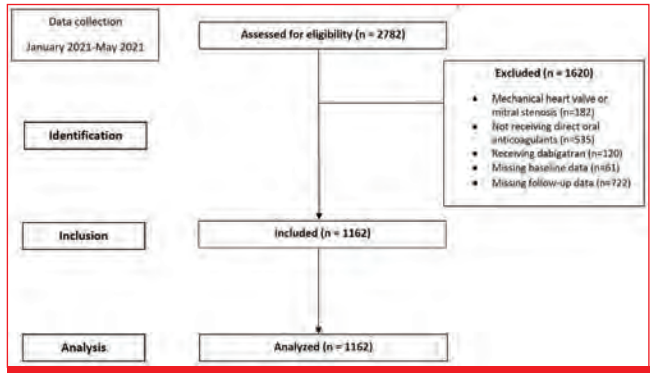

Figure 1. STROBE flow chart (STROBE, Strengthening the Reporting of Observational Studies in Epidemiology).

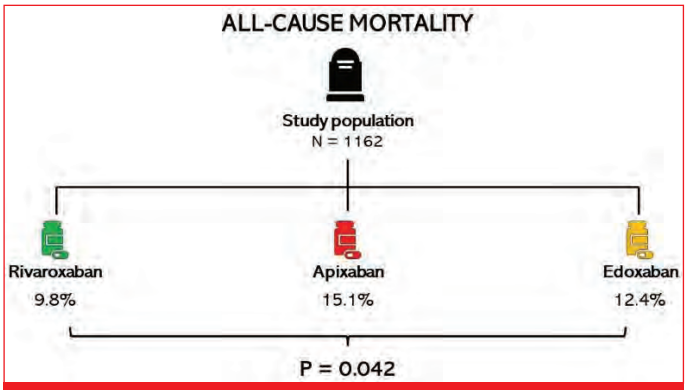

Figure 2. The all-cause mortality rate in the rivaroxaban group was lower compared with apixaban and edoxaban group (9.8% vs. 15.1% vs. 12.4%, respectively, p=0.042).

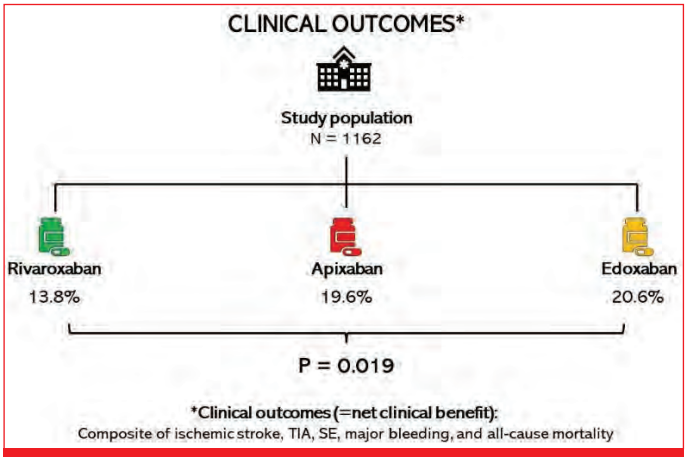

Figure 3. The frequency of net clinical outcomes was 13.8% for patients treated with rivaroxaban, 19.6% for patients treated with apixaban, and 20.6% for patients treated with edoxaban (p=0.019).

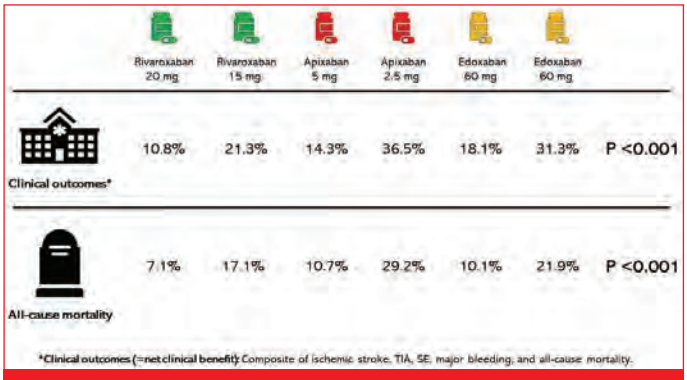

Figure 4. There was a significant difference between standard and reduced doses of each factor Xa inhibitor regarding clinical outcomes. While the rate of clinical outcomes was 21.3% for patients receiving rivaroxaban 15 mg o.d., the same rate was 36.5% and 31.3% for patients receiving apixaban 2.5 mg b.i.d. and edoxaban 30 mg o.d., respectively (p<0.001). Similarly, the all-cause mortality rate was 17.1% for patients treated with rivaroxaban 15 mg o.d., 29.2% for patients treated with apixaban 2.5 mg b.i.d., and 21.9% for patients treated with edoxaban 30 mg o.d., respectively (p<0.001).

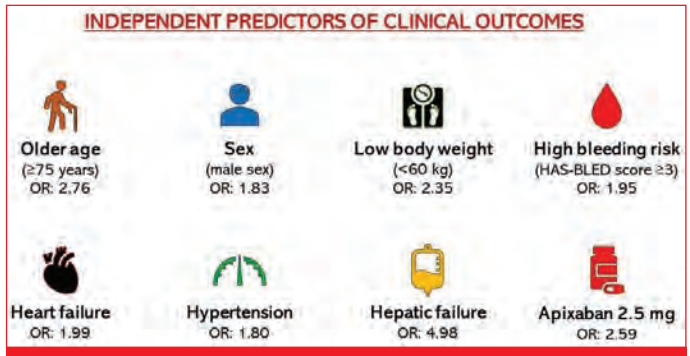

Figure 5. The independent clinical predictors of net clinical outcomes among the study population.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-002

The association of electrical risk score with prognosis in patients with non-ST elevation myocardial infarction

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Background and Aim: Acute coronary syndromes are the leading cause of mortality worldwide. Electrical risk score (ERS) is a novel electrocardiographic risk scoring system. There is no study evaluating the prognostic importance of ERS in patients with non-ST elevation myocardial infarction (NSTEMI). The aim of this study is to determine the association of ERS with in-hospital prognosis in NSTEMI patients.

Methods: 427 consecutive NSTEMI patients were included in this study. Six simple electrocardiographic parameters composed ERS: heart rate >75, presence of left ventricular hypertrophy according to Sokolow-Lyon criteria, QRS transition zone ≥V4, frontal QRS-T angle >90°, prolonged QTc interval (>450 for men, >460 women) and Tp-e interval >89 msec. The ERS was calculated according to the number of abnormal findings in the ECG. Patients were divided into two groups as ERS <3 and ≥3.

Results: No significant difference was found between ERS ≥3 and <3 groups in terms of demographic characteristics. However, patients with ERS ≥3 had significantly higher maximum troponin (p<0.001), TIMI (p=0.002) and GRACE (p<0.001) risk scores and 3-vessel disease frequency (p=0.001), whereas lower left ventricular ejection fraction (p<0.001). These patients had also higher frequency of in-hospital mortality (p<0.05) and adverse events. Logistic regression analysis demonstrated that ERS (OR: 1.790, 95% CI: 1.036-3.095, p=0.037) was an independent predictor of in-hospital mortality.

Conclusions: In conclusion, the frequency of in-hospital adverse events and mortality were significantly higher in

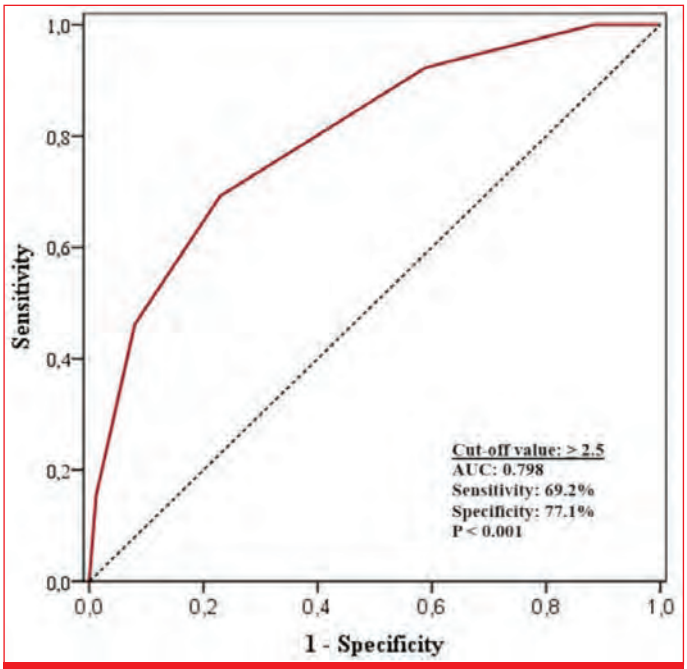

Figure 1. ROC curve analysis of electrical risk score for predicting in-hospital mortality

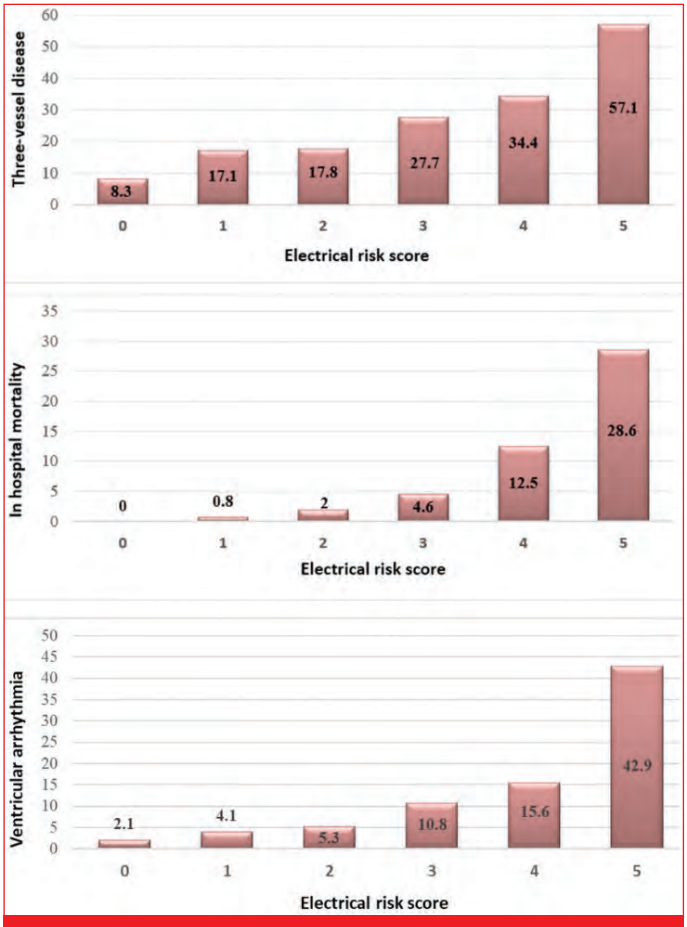

Figure 2. The frequency of three-vessel disease, in-hospital mortality and ventricular arrhythmia according to the number of electrocardiographic abnormalities.

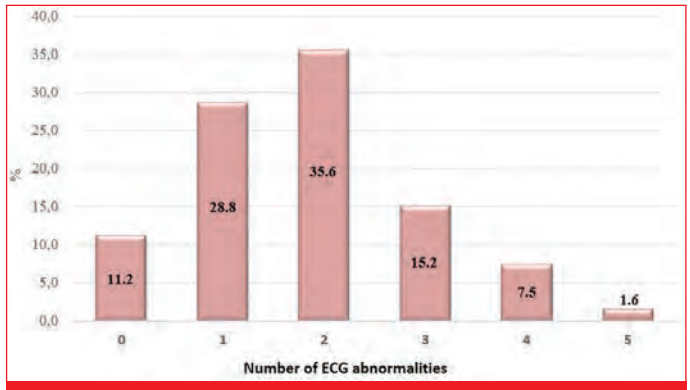

Figure 3. The number of electrocardiographic abnormalities included in the study.

NSTEMI patients with an ERS ≥3 at admission. This simple electrocardiographic risk marker may help identify patients at higher cardiac risk in patients presenting with NSTEMI and identify patients who may need early intervention.

Table 1. Comparison of baseline and laboratory characteristics of patients with ERS <3 and ≥3

Variables	ERS <3 (n=323)	ERS ≥3 (n=104)	p
Age (years)	60.3 ± 10.8	62.7 ± 13	0.093
Gender, Male (%)	217 (67.2)	65 (62.5)	0.380
BMI (kg/m^2)	26.5 ± 4.2	27 ± 4.2	0.316
SBP (mmHg)	130.3 ± 19.6	129.3 ± 22.7	0.681
DBP (mmHg)	78.6 ± 9.9	77.1 ± 11.2	0.204
HT (%)	159 (49.2)	60 (57.7)	0.133
DM (%)	156 (48.3)	60 (57.7)	0.096
Smoking (%)	217 (67.2)	61 (58.7)	0.112
Previous history of CAD (%)	104 (32.2)	31 (29.8)	0.648
Previous history of CVA (%)	7 (2.2)	5 (4.8)	0.156
TIMI score on admission	4.3 ± 1.4	4.6 ± 1.3	0.030
GRACE score on admission	113.8 ± 25.1	133.0 ± 30.6	<0.001
Glucose (mg/dL)	118 (97–166)	145.5 (108.5-234)	0.001
BUN (mg/dL)	34.2 (27.8-42.8)	38.5 (30.0-47.1)	0.006
Creatinine (mg/dL)	0.8 (0.7-0.9)	0.9 (0.7-1.0)	0.008
Total cholesterol (mg/dL)	177.7 ± 42.0	183.1 ± 38.5	0.250
Triglyceride (mg/dL)	138 (94-207)	149.5 (110.5-241.75)	0.100
HDL-Cholesterol (mg/dL)	32 (26-38)	32 (26-40)	0.701
LDL- Cholesterol (mg/dL)	107 (85.2-131.4)	111.8 (92.05-132.3)	0.452
CRP (mg/dL)	0.5 (0.2-1.3)	0.6 (0.2-1.8)	0.257
Albumin (g/dL)	4.2 ± 0.4	4.2 ± 0.5	0.306
Maximum CK-MB (ng/mL)	16.2 (4.9-49.2)	35.0 (10.6-96.8)	<0.001
Maximum Troponin (pg/mL)	5249.1 (816.8-17099.4)	15258.8 (5939.4-25000)	<0.001
Hemoglobin (g/dL)	14.2 ± 1.8	13.9 ± 1.9	0.204
Leukocytes (10x3/uL)	10.4 (8.24-13.03)	11.3 (9.0-14.5)	0.011
Thrombocyte (10x3/uL)	250 (206-301)	251 (223-315)	0.084

BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HT: Hypertension; DM: Diabetes mellitus; CAD: Coronary artery disease; CVA: Cerebrovascular accident; TIMI: Thrombolysis in myocardial infarction; GRACE: Global registry of acute coronary events; BUN: Blood urea nitrogen; CRP: C-reactive protein.

Table 2. Comparison of electrocardiographic and angiographic characteristics of patients with ERS <3 and ≥3

Variables	ERS <3 (n=323)	ERS ≥3 (n=104)	p
Heart rate (/min.)	77.5 ± 15.4	86.4 ± 13.9	<0.001
QRS duration (ms)	91.5 ± 14.4	100.1 ± 19.8	<0.001
QTc interval (ms)	416.7 ± 26.6	437.4 ± 34.7	<0.001
Tp-e interval (ms)	70 (60-80)	80 (80-100)	<0.001
Frontal QRS-T angle (°)	36 (18-80)	100 (47.5-123.0)	<0.001
LVEF (%)	49.6 ± 8.6	42.7 ± 11.1	<0.001
Three-vessel disease (%)	52 (16.1)	33 (31.7)	0.001
Number of stents used	1 (1-2)	1 (1-1.8)	0.671
Stent diameter (mm)	3.0 (2.5-3.0)	3.0 (2.5-3.0)	0.881
Stent length (mm)	29 (18-43)	31 (18-38)	0.997
SYNTAX-2 score	27.3 (20.3-36.9)	37.5 (30.4-53.8)	<0.001

LVEF: Left ventricular ejection fraction; SYNTAX: Synergy between PCI with TAXUS and Cardiac Surgery.

Table 3. Comparison of in-hospital complications

Variables	ERS <3 (n=323)	ERS ≥3 (n=104)	p
All arrhythmias (%)	23 (7.1)	18 (17.3)	0.002
Ventricular arrhythmias (%)	14 (4.3)	15 (14.4)	<0.001
Acute renal failure (%)	26 (8)	23 (22.1)	<0.001
Major bleeding (%)	1 (0.3)	2 (1.9)	0.087
Inotrope requirement (%)	27 (8.4)	25 (24)	<0.001
NIMV requirement (%)	9 (2.8)	19 (18.3)	<0.001
In-hospital mortality	4 (1.2)	9 (8.7)	<0.001

NIMV: Non-invasive mechanical ventilation.

Table 4. Independent predictors of in-hospital mortality

	OR	95% CI	p
GRACE risk score	1.030	1.010-1.051	0.004
Ventricular arrhythmia	7.057	1.755-28.378	0.006
Electrical risk score	1.790	1.036-3.095	0.037

Included variables: Age, gender, body mass index, hypertension, diabetes mellitus, three-vessel disease, electrical risk score, TIMI, GRACE risk score, ventricular arrhythmia.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-003

Investigating long-term outcomes and predictors of primary outcomes in patients treated for cardiac implantable electronic device infections

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Background and Aim: The current estimated infection rate following implantation of cardiac implantable electronic devices (CIEDs) is between 1% and 2%, with variations in the literature ranging from 0.13% to 12.6%. The increase in CIED implantation rates over the years has led to an increase in the incidence of device-related infections. This trend is exacerbated by the increase in comorbidities and life expectancy of patients. The aim of this study was to evaluate the primary endpoints (death, myocardial infarction (MI), cerebrovascular accident (CVA), and reinfection) in patients treated for CIED infection and to identify predictors of the primary endpoints.

Methods: A single-center retrospective analysis was performed. The study included 2322 patients admitted to the arrhythmia clinic of our institute between March 2011 and July 2020 who underwent CIED implantation. After excluding 20 patients with active infection and 3 patients with postoperative cardiac arrest, 36 patients were diagnosed with CIED infection (1.55% incidence). The diagnosis was based on the new 2019 International Criteria for CIED Infection. Patients were stratified according to the occurrence of all-cause death, MI, LVO and reinfection during the follow-up period.

Results: Median follow-up after presentation with CIED infection was 42.5 (7-141) months. Table 1 shows patient characteristics, procedure details and management-related parameters in groups stratified by primary endpoint. 58.3% of patients had undergone a previous device exchange procedure. Among patients with CIED infection, reinfection was observed in 20.0% of those undergoing a de novo procedure and 38.1% of those undergoing a second procedure (p=0.25). Cox regression analysis revealed that pre-procedure C-reactive protein (CRP) level and CHA2DS2-VASc score were independent predictors of the primary endpoint [OR: 1.03 (1.00-1.06); p=0.035, OR: 1.34 (1.03-1.88); p=0.030] (Table 2).

Conclusions: In conclusion, long-term outcomes in patients with CIED infection appear to be associated with pre-procedural CRP levels, which indicate the severity of infection, and the CHA2DS2-VASc score, which provides valuable information about the patient's comorbidities. These factors seem to outweigh the impact of the specific procedure and the antibiotic regimens administered. Management of patients should not only focus on the history of CIED infection during follow-up, but also prioritize the management of existing comorbidities to improve outcomes. Given the limitations of the study, such as its small sample size and retrospective nature, further research is warranted to confirm these findings.

Table 1.

Table 1: Baseline Demographic Characteristics , demographic characteristics, management strategies and laboratory parameters of Study Groups

	Primary Outcome (-) (n:18)	Primary Outcome (+) (n:18)	P value
Age (years)	61 (21-93)	78(52-93)	0.011
Male(%)	10 (55.6%)	12 (33.3%)	0.270
Hypertension, n (%)	11(61.1%)	14(77.8%)	0.278
Diabetes Mellitus, n (%)	4 (22.2%)	10 (55.6%)	0.040
Hyperlipidemia, n (%)	3 (16.7%)	11 (61.1%)	0.006
AF, n (%)	3 (16.7%)	10 (55.6%)	0.015
CAD, n (%)	6 (33.3%)	9 (50.0%)	0.310
LV-EF< 40% n(%)	5 (27.8%)	12 (66.7%)	0.019
CHA2DS2VASC	2 [0-5]	5 [2-6]	0.013
Type of Device			
Pacemaker, n(%)	8 (44.4%)	7 (38.9%)	
VVI/DDD-ICD, n(%)	8 (44.4%)	9 (50.0%)	0.939
CRT-D, n(%)	2 (11.1%)	2 (11.1%)	
Number of Lead >1, n(%)	12 (66.7%)	7 (38.9%)	0.095
Blood Culture(+), n(%)	1 (5.6%)	2 (11.1%)	0.546
Pocket Culture(+), n(%)	7 (38.9%)	8 (44.4%)	0.735
Treatments, n(%)			
Medical, n(%)	4 (22.2%)	4 (22.2%)	
Only Battery Removal, n(%)	8 (44.4%)	7 (38.9%)	0.931
All System Removal, n(%)	6 (33.3%)	7 (38.9%)	
Long Antibiotherapy, n(%)	13 (61.9%)	9 (50.0%)	0.091
GFR	78 (60-120)	60 (29-101)	0.001
CRP	7.9 (0.7-90.0)	11.4 (1.2-82.0)	0.446
ESR	14.5 (5-112)	17 (6-97)	0.556
Hemoglobin	12.5 (9.9-16)	12.7 (9.5-15.5)	0.866
HCT	37.6 (28.7-49.7)	37.4 (28.2-46.7)	0.767
WBC	8950 (3400-12030)	7900 (5100-14300)	0.181
PLT	217 (132-319)	202 (121-365)	0.787

CAD: coronary artery disease , CHA2DS2-VASc score , Congestive heart failure, Hypertension, Age >_ 75 (doubled), Diabetes, Previous stroke/transient ischaemic attack/thromboembolism (doubled), Vascular disease, Age: 65–74, sex (female) . Long Antibiotherapy: Antibiotic therapy for more than 14 days before the procedure in a patient whose culture is negative or negative with treatment . GFR: Glomerular Filtration Rate Using the Cockcroft-Gault Formula

Table 2.

Table 2: Independent Predictors of Clinical Outcome

	OR	%95 CI	p value
Number of Lead ≥ 2	0,625	0,236-1,656	0,345
Preprocedural CRP Level	1,029	1,002-1,058	**0,035**
Sedimentation Level	0,972	0,938-1,007	0,112
CHA2DS2-VASc score	1,393	1,033-1,878	**0,030**

Table 2: Independent Predictors of Clinical Outcome
Clinical outcome : All Cause of Death and Reenfection CI: confidence interval, CRP: C-reactive protein CHA2DS2-VASc score : Congestive heart failure, Hypertension, Age >_ 75 (doubled), Diabetes, Previous stroke/transient ischaemic attack/thromboembolism (doubled), Vascular disease, Age: 65–74, sex (female)

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-004

A new scoring system to predict the risk of late recurrence in extended follow-up after atrial fibrillation catheter ablation: APCEL score

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Background and Aim: In studies where risk scores used to determine the risk of late recurrence after atrial fibrillation (AF) catheter ablation were defined, significant differences were observed in terms of parameters such as post-procedural follow-up time, pre-procedural AF time, energy sources used for ablation, and cut-off values of left atrium (LA) diameter. Considering all these factors, we aimed to develop a new recurrence risk score for prolonged follow-up after AF ablation.

Methods: The study included 206 patients who underwent index AF catheter ablation for paroxysmal or persistent AF. Independent predictors of late recurrence were identified at a median follow-up of 40 months (range 21-57), and a risk score was created. The predictive ability of this score for late recurrence was compared with that of other risk scores.

Results: During the follow-up period of 40.0 (21.0-57.0) months, early recurrence developed in 32 patients (15.5%), and late recurrence developed in 63 patients (30.6%). The frequencies of chronic obstructive pulmonary disease (COPD) (14.3% vs. 4.9%), persistent AF (38.1% vs. 18.2%), and early recurrence (31.7% vs. 8.4%) were significantly higher in those who developed late recurrence than in those who did not (p=0.020, 0.004, and <0.001, respectively). AF duration before ablation [28.0 (21.0-36.0) vs. 18.0 (8.5-24.0) months], left ventricle (LV) mass index [99.4 (86.3-116.0) vs. 93.1 (81.0-104.3) g/m^2], LAVI [36.0 (30.5-44.0) vs. 29.0 (23.0-36.5) mL/m^2], and systolic pulmonary artery pressure (SPAP) [27.0 (21.0-32.0) vs. 25.0 (20.0-29.0) mmHg] were significantly higher in those who developed late recurrence than in those who did not (p<0.001, p=0.018, p<0.001, and p=0.011, respectively) (Table 1). Independent predictors of late recurrence development included pre-ablation AF duration >19 months, persistent AF, early recurrence, chronic obstructive pulmonary disease, and LA volume index >32 mL/m^2 (Table 2). The APCEL risk score, derived from these factors, demonstrated good predictive performance for late recurrence [AUC: 0.837 (95% CI: 0.780-0.885)] (Figure 1). The median APCEL score was significantly higher in patients with late recurrence [3.0 (2.0-4.0)] than in those without [2.0 (0-2.0)] (p<0.001) (Table 1). The APCEL score outperformed APPLE, ATLAS, BASE-AF2, and MB-LATER risk scores in predicting late recurrence (p<0.001 for all) (Figure 2). In general, the risk of late recurrence increased in proportion to the patients' APCEL scores (Figure 3).

Conclusions: The APCEL score, calculated at the end of the blanking period for patients who underwent AF ablation, can effectively identify those at high risk of late recurrence during extended follow-up. This allows for closer monitoring of high-risk individuals and better management of modifiable risk factors.

Variables		Score
A	AF duration >19 months	2
P	Persistent AF	1
C	COPD	1
E	Early recurrence	1
L	LAVI >32 ml/m^2	1

Figure 1. APCEL score.

AF: Atrial fibrillation; COPD: Chronic obstructive pulmonary disease; LAVI: Left atrium volume index.

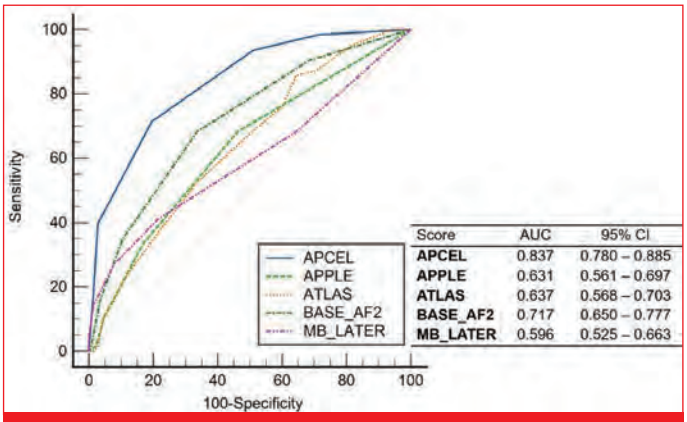

Figure 2. ROC curves of various risk scores to predict late recurrence after AF catheter ablation.

ROC curves of various risk scores to predict late recurrence after AF catheter ablation. AF: Atrial fibrillation, ROC: Receiver operating characteristic.

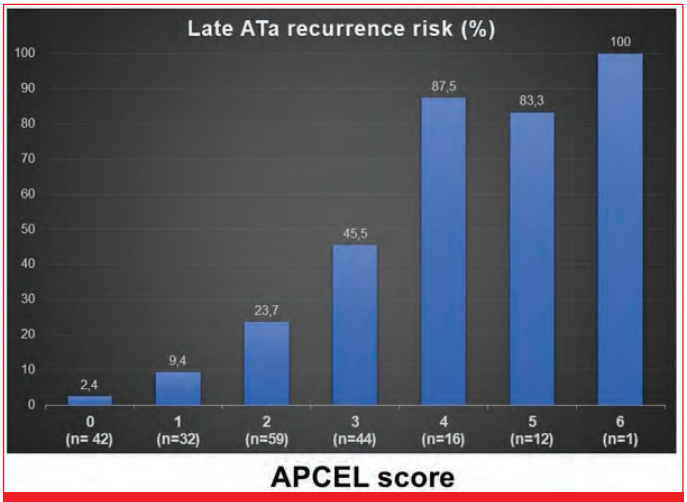

Figure 3. Frequency of each APCEL risk score in the study population and the corresponding risk of late recurrence for each score at long-term follow-up.

APCEL: AF duration, persistent AF, early recurrence, COPD, and LAVI; AF: Atrial fibrillation; COPD: Chronic obstructive pulmonary disease; LAVI: Left atrium volume index.

Table 1. Baseline characteristics of the study population

	Late recurrence (-) (n=143)	Late recurrence (+) (n=63)	p
Age (years)	59.0 (50.0-63.5)	58.0 (52.0-63.0)	0.948
Sex (female) (n, %)	63 (44.1)	36 (57.1)	0.083
BMI (kg/m^2)	28.0 (25.8-32.0)	29.0 (27.3-32.6)	0.101
Hypertension (n, %)	70 (49.0)	36 (57.1)	0.278
COPD (n, %)	7 (4.9)	9 (14.3)	0.020
OSAS (n, %)	12 (8.4)	8 (12.7)	0.480
AF duration (months) (n, %)	18.0 (8.5-24.0)	28.0 (21.0-36.0)	<0.001
Persistent AF (n, %)	26 (18.2)	24 (38.1)	0.004
Early recurrence (n, %)	12 (8.4)	20 (31.7)	<0.001
APCEL score	2.0 (0-2.0)	3.0 (2.0-4.0)	<0.001
LV EF (%)	61.0 (58.0-64.0)	61.0 (58.0-64.5)	0.983
LV mass index (g/m^2)	93.1 (81.0-104.3)	99.4 (86.3-116.0)	0.018
LAVI (mL/m^2)	29.0 (23.0-36.5)	36.0 (30.5-44.0)	<0.001
SPAP (mmHg)	25.0 (20.0-29.0)	27.0 (21.0-32.0)	0.011

AF: Atrial fibrillation; BMI: Body mass index; COPD: Chronic obstructive pulmonary disease; EF: Ejection fraction; LA: Left atrium; LAVI: Left atrium volume index; LV: Left ventricle; OSAS: Obstructive sleep apnea syndrome; SPAP: Systolic pulmonary artery pressure; APCEL: AF duration, persistent AF, early recurrence, COPD, and LAVI.

Table 2. Multiple Cox regression analysis results for prediction of ATa recurrence after AF ablation

	Beta	HR	95%CI	P
AF duration >19 months	1.295	3.652	1.841-7.244	<0.001
Persistent AF	0.724	2.062	1.126-3.775	0.019
COPD	0.954	2.597	1.258-5.364	0.010
Early Recurrence	1.074	2.926	1.606-5.329	<0.001
LAVI >32	0.961	2.613	1.466-4.659	0.001

AF: Atrial fibrillation; ATa: Atrial tachyarrhythmia; CI: Confidence interval; COPD: Chronic obstructive pulmonary disease; HR: Hazard ratio; LAVI: Left atrium volume index.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-005

Comprehensive analysis of recurrence factors in cryoballoon af ablation: Integrating clinical, biomarkers, and echocardiographic parameters

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Background and Aim: Atrial fibrillation (AF) poses substantial challenges in cardiovascular diseases, impacting patient health and economic burden. Understanding the mechanical effects of AF on the left atrium (LA) and assessing the influence of treatment modalities on LA functions are critical. This study aims to assess the efficacy of echocardiographic and biochemical parameters in predicting AF recurrence following second generation cryoballoon ablation (CB-2).

Methods: Ninety-two patients with symptomatic AF, treated with CB-2 at İstanbul University-Cerrahpaşa, Faculty of Medicine, Department of Cardiology, were prospectively examined from January 2021 to July 2023. The study endeavors to develop a predictive model for AF recurrence, investigating the relationship between echocardiographic measurements and serum biomarkers with recurrence. The follow-up duration for echocardiographic assessments and biochemical analyses was systematically documented.

Results: The study revealed a significant enhancement in LA mechanical functions during echocardiographic follow-ups three months post-procedure. Specifically, LA strain parameters emerged as significant predictors of recurrence (LAsr: 95% CI: 1.004-1.246, p=0.047; LAsct: 95% CI: 1.040-1.750, p=0.024). Biochemical analyses demonstrated a correlation between elevated PRO-BNP levels and an increased risk of recurrence (95% CI: 1.000-1.003, p=0.012). Moreover, specific biomarkers such as MYBPHL, which demonstrated increased levels post-procedure, were deemed indicative of atrial damage, suggesting potential additional atrial substrate modification beyond PVI. Consequently, improvements in LA function post-cryoballoon ablation and biochemical markers have surfaced as potential indicators for predicting AF recurrence.

Conclusions: This study elucidates the effectiveness of CB-2 in treating AF and its impact on LA functions. Notably, LA strain measurements and PRO-BNP levels have emerged as reliable indicators for predicting recurrence. Beyond clinical implications, our research establishes a foundation for a deeper understanding of the role of CB-2 in AF management and factors associated with recurrence.

Table 1. Univariate and Multivariate Analysis with Logistic Regression

	Univariate			Multivariate		
	CI	OR(ExpB)	P-value	CI	OR(ExpB)	P-value
Demographic Data						
AF type (Paroxysmal:1, persistant:2)	1,679-15,32	5,073	**0,004**	0,072-52	1,940	0,693
CHA2DS2-VASc	1,048-2,208	1,521	**0,027**	0,928-3,231	1,731	0,085
Biochemical Finding						
PRO-BNP	1,000-1,002	1,001	**0,022**	1,000-1,003	1,002	**0,012**
Echocardiographic Results						
LA dimension	1,007-1,211	1,104	**0,035**	0,825-1,221	1,004	0,970
LAsr	0,901-0,990	0,945	**0,018**	1,004-1,246	1,113	**0,047**
LAsct	1,056-1,369	1,202	**0,005**	1,040-1,750	1,349	**0,024**
LV endo average strain	0,987-1,281	1,124	0,078	0,794-1,479	1,084	0,611
LA-EMD lateral	1,006-1,069	1,037	**0,018**	0,963-1,066	1,013	0,622
E wave velocity	1,002-1,058	1,030	**0,033**	0,960-1,056	1,007	0,782

Cardiovascular Nursing / Technician

OP-006

Nursing care in a surgically treated refractory atrial tachycardia: A case report

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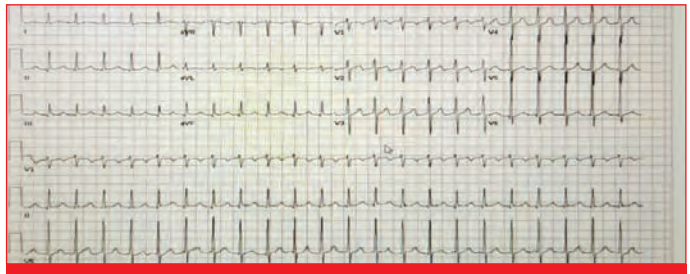
Background and Aim: Atrial tachycardia (AT) usually responds well to catheter ablation therapy. In rare cases, it may be resistant and cause tachycardia-induced cardiomyopathy (CMP), and surgical treatment is an important option in this patient group. The nurse and nursing care have a critical role in the follow-up and treatment of patients admitted to the intensive care unit with a tachycardia attack.

Methods: A 34-year-old male patient was admitted to our emergency department with a complaint of palpitations, the initial ECG was compatible with Atrial Tachycardia (AT). Echocardiography in the emergency department revealed normal left ventricular (LV) systolic functions with an ejection fraction (EF) of 50%. The rate control could not be achieved despite intravenous (IV) beta blocker, adenosine and calcium channel blockers, he was admitted to our intensive care unit for rhythm/rate control with amiodarone infusion or, if necessary, electrical cardioversion or catheter ablation.Rate/rhythm control could not be achieved and control EF was found to be 40% with LV diameters at their upper limit, the tachycardia-induced CMP diagnosis was clarified, and catheter ablation was planned.

Results: Under general anesthesia, tachycardia could not be terminated in the electrophysiology laboratory despite three-dimensional mapping and multiple radiofrequency ablation applications. Tachycardia could not also be terminated with right atrial appendage isolation and cryoballoon ablation. Ultimately, ablation with the epicardial approach was also unsuccessful. In the imaging performed with contrast, an existing diverticulum was detected in the right atrial appendage, and a decision was made for surgical treatment. The patient underwent surgical excision of the right atrial appendage, tachycardia stopped immediately. The patient was discharged on the third day without any problems, and at his 1st-month follow-up, 24-hour rhythm Holter was found to be normal and EF was found to be 60%.

Conclusions: Atrial tachycardia (AT) usually responds well to catheter ablation therapy. However, rarely, and especially in cases originating from the right atrial appendage, surgical treatment results have a high success rate. Nursing care is critical in resistant atrial tachyarrhythmias that require a multidisciplinary approach.



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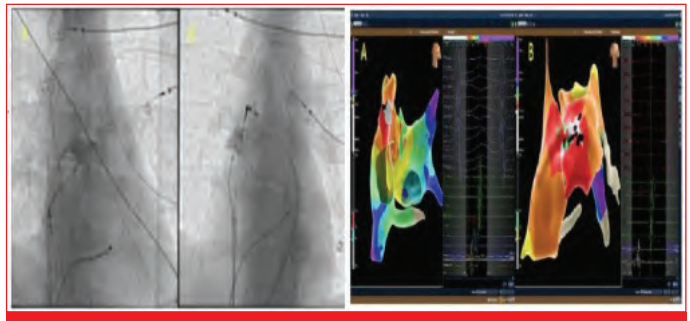

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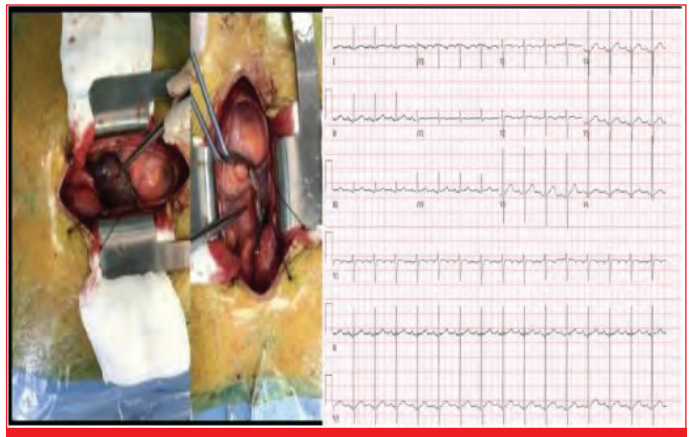

Figure 3.

Other

OP-007

Do orally ingested nanoplastics affect cardiac tissue in rats?

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[2]Department of Biotechnology, Ankara University, Biotechnology Institute, Ankara
[3]Department of Anatomy, Ordu University Faculty of Medicine, Ordu
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Background and Aim: The increasing use of oil-derived plastic materials also leads to the dissemination of their waste in the natural environment. The release of micro and nanoplastics from plastic waste presents an important risk to the environment. We aim in this study investigate the potential accumulation of oral polystyrene nanoplastic (PS-NP) in the cardiac tissue and determine their deleterious effects on rats.
Methods: A total of 30 Wistar albino rats, which included 15 males and 15 females, aged between 6 and 8 weeks, were used in the research.The rats were divided into three groups each consisting of five males and five females. The control group of rats did not undergo any experimental procedures during the study. They were fed with standard food and water. In addition to normal nutrition, the rats in the low-dose group received 25 mg/kg fluorescent labeled PS-NP at the same time every day by oral gavage. The rats in the high-dose nanoplastic group received 50 mg/kg fluorescent labeled PS-NP at the same time every day by oral gavage in addition to normal nutrition. The heart tissue were removed and subjected to histopathological and biochemical analyses.
Results: In immunofluorescence analysis, in both images, there is no morphologic deformation and nanoplastics within the myocardium tissue (Figure 1). In immunofluorescence analysis, green fluorescent dots were detected in the cardiac tissue of low and high dose groups rats receiving fluorescent labeled nanoplastics (Figure 2, 3). Histopathological examination revealed that there was a lack of structure in the muscle fibers and displacement of the nucleus in the heart tissue sections of both male and female rats in the low dose group (Figure 4). In the high dose group rats, vacuolization, bleeding, and edema were observed in the tissue sections. Additionally, there was disorder in the muscle fibers and nucleus, which was not observed in the other groups. Catalase activity was significantly higher in male rats in the high dose group compared to the control group (p=0.036) (Table 1). Additionally, peroxidase activity was significantly higher in both male rats (p=0.031) and female rats (p=0.004) in the high dose group compared to the control group. The activity of Glutathione-S-Transferase in male rats was shown to be decreased in the high dose group compared to both the control group and the low dose group (p=0.023).
Conclusions: Immunofluorescence microscopy showed that orally ingested nanoplastics accumulated in the cardiac tissue of rats. At the same time, that nanoplastics caused histological changes in the cardiac muscle tissue. Furthermore, during chemical examination, it was noted that nanoplastics induced alterations in the activity of antioxidant enzymes.

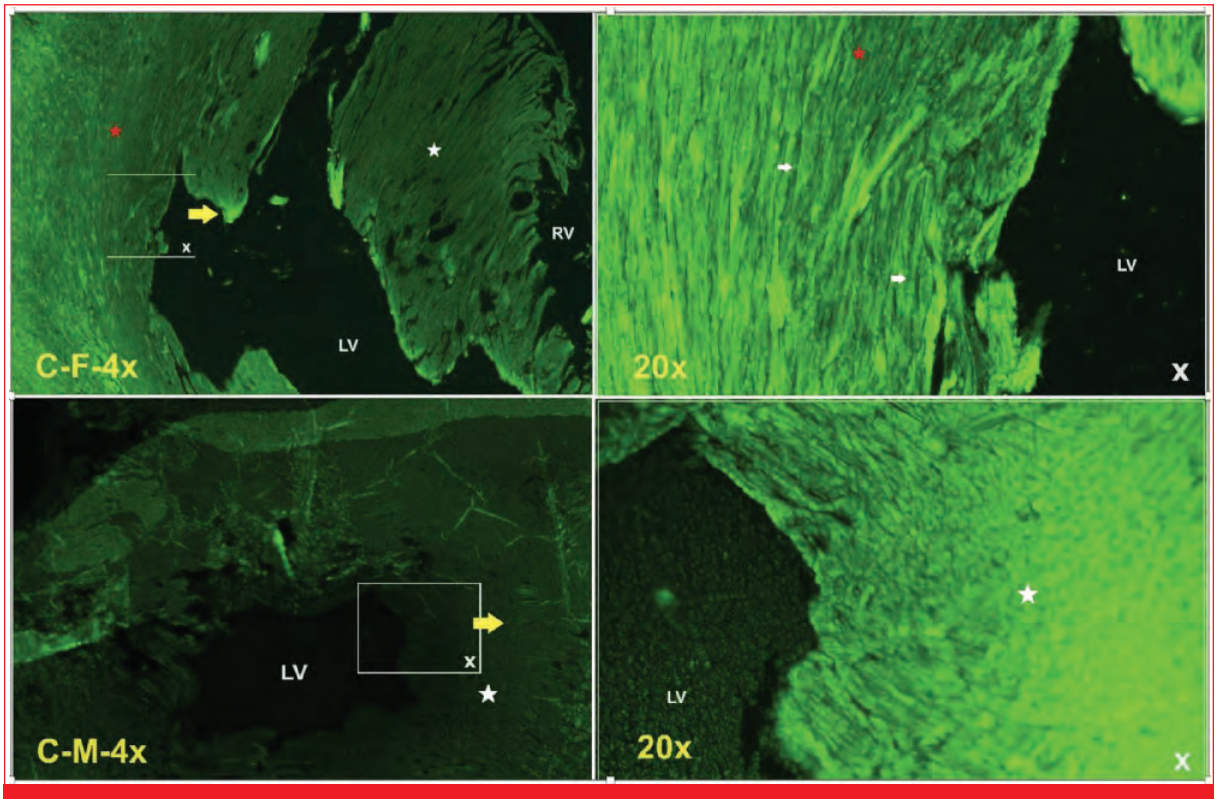
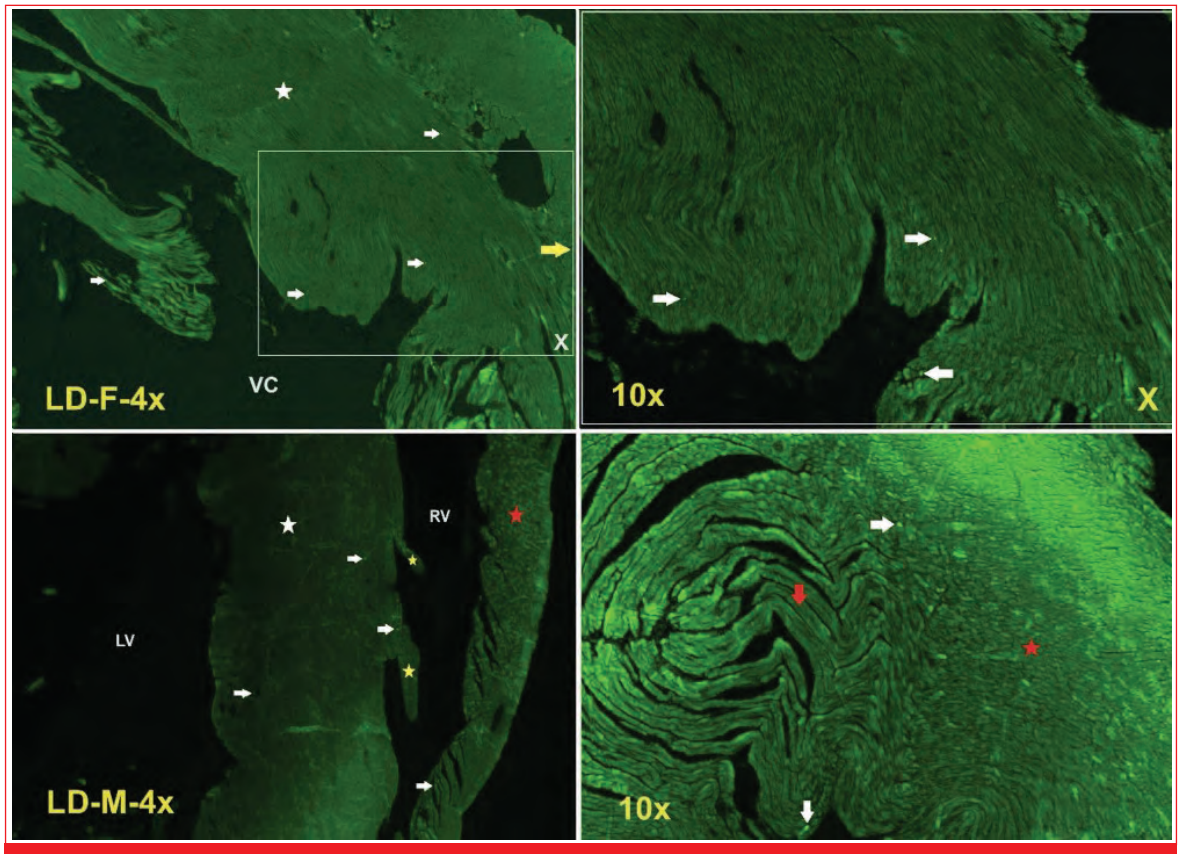


Figure 1.



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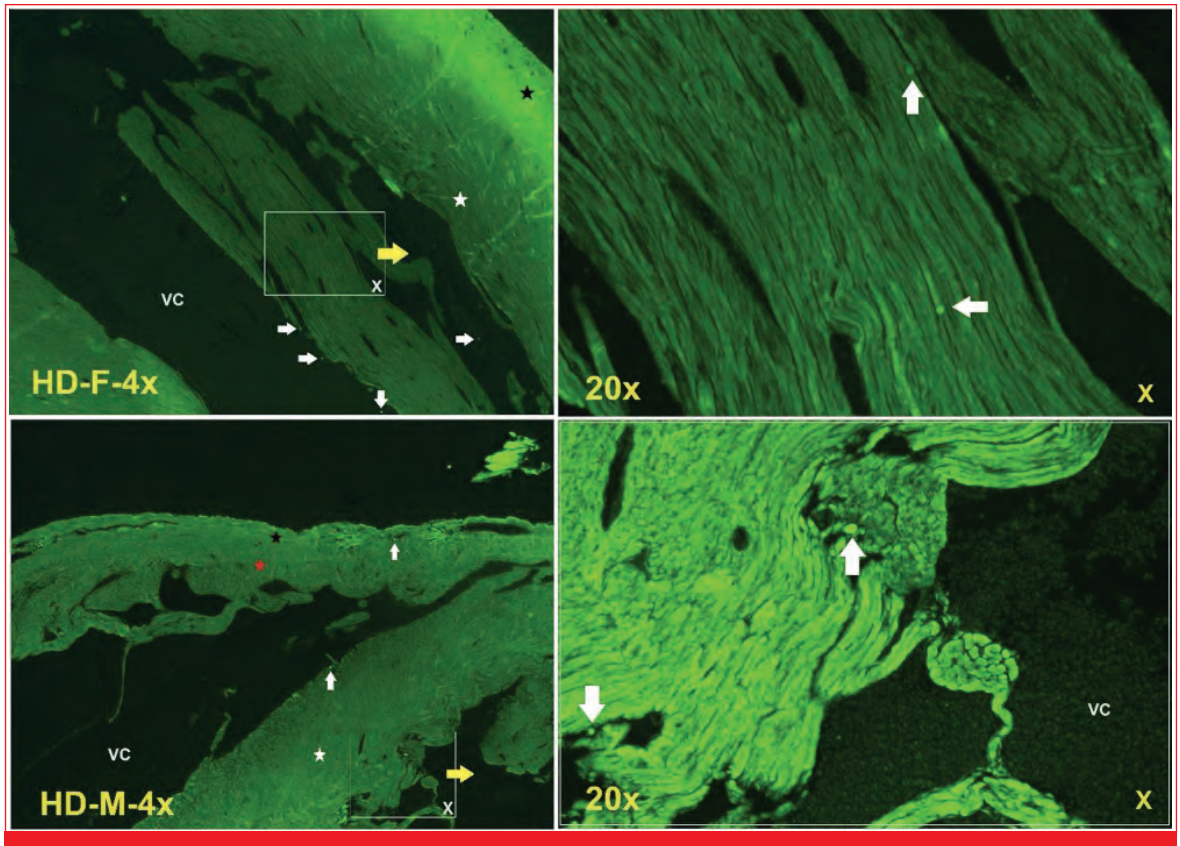

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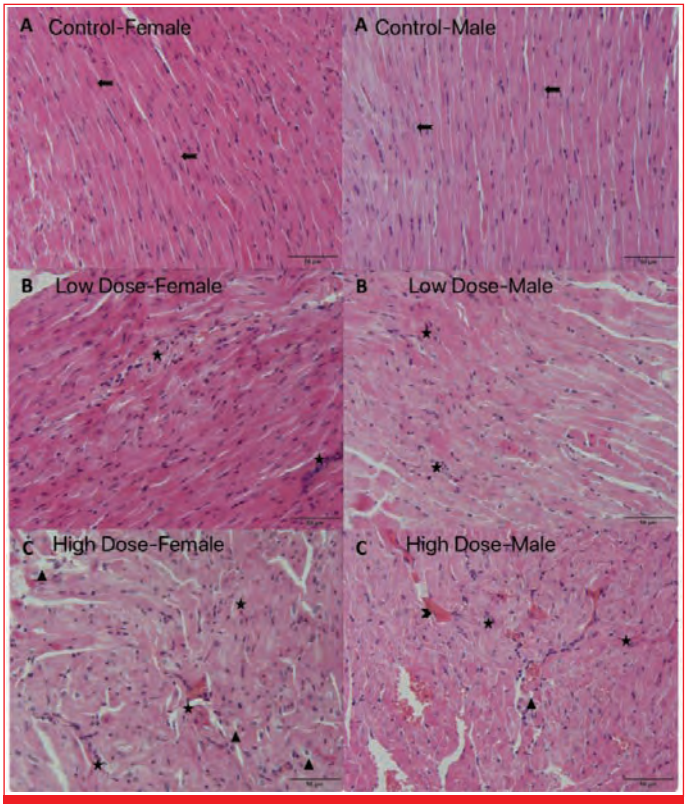

Figure 4.

Other

OP-008

The impact of automatic history-taking software on data quality in the cardiology outpatient clinic

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Adapazari Enka Schools Anatolian High School, Sakarya

Background and Aim: Healthcare delivery now mandates shorter visits despite the need for more data entry, under-mining patient-provider interaction. Furthermore, enhancing access to the outcomes of prior tests and imaging conducted on the patient, along with accurately documenting medication history, will significantly elevate the quality of healthcare service delivery. To enhance the efficiency of clinic visits, we have devised a patient-provider portal that systematically gathers symptom and clinical data from patients through a computer algorithm known as Automated Assessment of Cardiovascular Examination (AACE). We intended to assess the quality of computer-generated Electronic Health Records (EHRs) with those documented by physicians.

Methods: We conducted a cross-sectional study employing a paired-sample design, focusing on individuals seeking assessment for active cardiovascular symptoms at outpatient adult cardiovascular clinics. Participants initially completed the AACE, and subsequently, in the first protocol, patients were subjected to routine care without providing the AACE forms to examining physicians. In the second protocol, the AACE form was presented to the physician before the examination, and participants were subjected to routine care. We assessed the impact of AACE forms generated through computerized history-taking method on the examination, considering various clinical outcomes and satisfaction surveys.

Results: We included non-randomized eligible patients who visited seven general cardiology outpatient clinics between September 18, 2023, and October 27, 2023. These clinics were staffed by the same physicians who were unaware of the content and details of the study. A total of 762 patients (394 patients in protocol 1 and 368 patients in protocol 2) were included in the study. The mean overall impression score for computer-generated EHRs was higher versus physician EHRs (4.2 vs. 2.6; p<0.001). Our study demonstrated that EHRs created by physicians' exhibit inaccuracies or deficiencies in various pieces of information. In the second protocol, in which the AACE form was presented to the physician before the examination, it was determined that the examination time was shorter, the number of tests requested, and the number of new drugs prescribed were less.

Conclusions: We observed that the patient-provider portal, systematically collecting symptom and clinical data from patients through a computer algorithm known as AACE, yielded records that were of higher quality, more comprehensive, better organized, and more relevant compared to those documented by physicians.

Other

OP-009

The correlation and agreement between SCORE2 and PREVENT 10-year ASCVD risk scores: insights from coronary computed tomographic angiography

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Background and Aim: Cardiovascular diseases (CVD) are still one of the major causes of morbidity and mortality and predicting CVD risk is crucial. Predicting Risk of CVD EVENTs (PREVENT) scoring system was developed in the United States that predicts 10-and 30-year risk of total CVD. SCORE2 and SCORE2-OP systems are currently used in our country to assess CVD risk. This study aimed to evaluate the correlation and agreement between these two risk scores in a known ASCVD-free cohort, incorporating computed tomographic coronary angiography (CCTA) data to assess whether there are differences between the groups and the scoring systems regarding CCTA parameters.

Methods: We retrospectively enrolled 171 consecutive patients who presented to the outpatient clinic with chest

pain and underwent CCTA because of considered to have a low clinical likelihood of CAD. Patients aged between 30 and 79 were included to calculate the PREVENT risk score. Patients with a high likelihood of CAD, current usage of lipid-lowering agents, familial hypercholesterolemia, known ASCVD, left ventricular ejection fraction of <50%, and estimated GFR (eGFR) < 45 mL/min/1.73 m^2 were excluded from the study. The PREVENT risk score was determined by the PREVENTTM online calculator. The SCORE2 risk score was used for patients aged 40-69 years and the SCORE2-OP risk score was used for patients aged 70-79 years without known ASCVD and DM. Coronary atherosclerotic burden was assessed by coronary artery calcium (CAC) score. CAC scores were expressed as absolute values and graded according to these values as no evidence of CAD (0), minimal (0-9), mild (10-99), moderate (100-399), severe (400-999), and extensive (≥1000).

Results: The median age of all cohort was 53.7 ± 13.3 years and 52 (30.4%) of the patients were female. PREVENT risk score were available in all patients (n=171) whereas, SCORE2 and SCORE2-OP were available in 113 of the patients. PREVENT 10-year ASCVD risk scores were strongly correlated with SCORE2 and SCORE2-OP 10-year CVD risk scores (r=0.85, p<0.001) and Bland-Altman plot analysis showed a bias of -3.71 points and the limits of agreement were -16.06 and 8.64 with 95% CI (Figure 1). The correlation and agreement between two scoring systems were good however, the risk categories were significantly different (p<0.0001) (Table 1). Among CCTA data, the total CAC score and grade of CAD according to this score were significantly different across PREVENT risk score categories (p<0.001 for all). However, these CCTA findings were similar across SCORE2 & SCORE2-OP risk groups (p=0.3 and p=0.051, respectively).

Conclusions: Adding variables such as body mass index, estimated glomerular filtration rate, and use of antihypertensive drugs, which are not in the SCORE2 risk system but are in the PREVENT risk system, did not significantly change individual scores of patients. However, the total CAC score and grade of CAD according to this score are significantly different across PREVENT and SCORE2 risk systems.

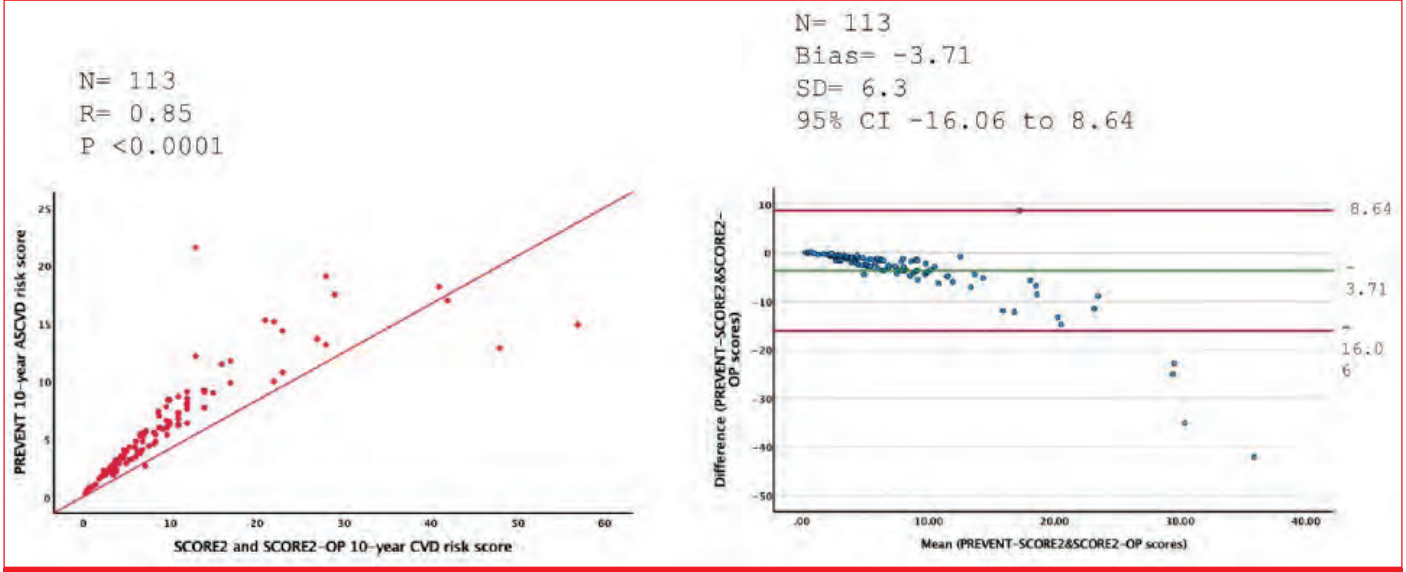

Figure 1. The correlation and Bland-Altmann analysis between PREVENT 10-year ASCVD and SCORE2-SCORE2-OP 10-year CVD risk scores.

Table 1. Patient classification according to PREVENT and SCORE2 risk categories

	SCORE2 and SCORE2-OP risk category (n, %)	SCORE2 and SCORE2-OP risk category (n, %)	SCORE2 and SCORE2-OP risk category (n, %)
PREVENT risk category	**Low-to-moderate risk (n=32)**	**High risk (n=39)**	**Very high risk (n=42)**
Low risk (n=58, 51.3%)	32 (55.2)	22 (37.9)	4 (6.9)
Borderline risk (n=22, 20.4%)	0 (0)	15 (65.2)	8 (34.8)
Intermediate risk (n=31, 27.4%)	0 (0)	2 (6.5)	29 (93.5)
High risk (n=1, 0.9%)	0 (0)	0 (0)	1 (100)

Other

OP-010

Artificial intelligence (Chat GPT) ready to evaluate ECG in real life? Not Yet!

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Cardiology Clinic, Medical Park Göztepe Hospital, İstanbul

Background and Aim: This study aims at evaluating if ChatGPT based artificial intelligence (AI) models are effective in interpreting electrocardiograms (ECGs) and determine their accuracy as compared to those of cardiologists. The purpose is therefore to explore if ChatGPT can be employed for clinical setting, particularly where there are no available cardiologists.

Methods: A total of 107 ECG cases classified according to difficulty (simple, intermediate, complex), were analyzed using three AI models (ECG reader, ECG analyzer, ECG interpreter) and compared with the performance of two cardiologists. The statistical analysis was conducted using chi-square and Fisher exact tests using scikit-learn library in Python 3.8.

Results: Cardiologists demonstrated superior accuracy (92.52%) compared to ChatGPT-based models (READER: 57.94%, INTERPRETER: 62.62%, ANALYZER: 62.62%). Statistically significant differences were observed between cardiologists and AI models ($p<0.05$). ChatGPT models exhibited enhanced performance with female patients; however, the differences found were not statistically significant. Cardiologists significantly outperformed AI models across all difficulty levels. When it comes to diagnosing patients with arrhythmia (A) and cardiac structural disease (CSD) ECG patterns, cardiologists gave the best results though there was no any statistical difference between them and AI models in diagnosing people with normal (N) ECG patterns.

Conclusions: ChatGPT based models have potential in ECG interpretation; however, they currently lack adequate reliability beyond oversight from a doctor. Additionally, further studies that would improve the accuracy of these models especially in intricate diagnoses are needed.

Other

OP-011

Assessing radiation dose variability across different imaging modalities and views in hemodynamic laboratory procedures: A comparative analysis

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Department of Cardiology, Gülhane Training and Research Hospital, Ankara

Background and Aim: In cardiology, optimizing imaging techniques is essential for balancing diagnostic accuracy and patient safety. This study investigates the variations in radiographic parameters across different imaging views and modalities to assess their impact on radiation exposure.

Methods: To evaluate how different imaging views (AP, AP-cranial, Left-cranial, Right-cranial, AP-caudal, Left-caudal, Right-caudal, LAO, RAO) and frame rates (15 FPS vs. 30 FPS) affect kilovoltage (kV), milliampere-seconds (mAs), exposure time (ms), and dose area product (DAP) in hemodynamic procedures, data from 147 patients who underwent hemodynamic laboratory procedures over a 1-month period were analyzed. Parameters kV, mAs, ms, and DAP were compared across various imaging views. Statistical analyses were performed to identify significant differences.

Results: The study highlights significant differences in radiation exposure parameters across various imaging views and frame rates. Left-caudal exhibited the highest kV (85.60 ± 12.70), while AP had the lowest (68.81 ± 6.24). AP-caudal had the highest mAs (865.22 ± 611.65), and AP had the lowest (500.89 ± 266.46). AP-caudal showed the longest exposure time (7.41 ± 0.86 ms), and LAO the shortest (4.82 ± 1.49 ms). DAP was highest in Left-caudal (3888.43 ± 2422.80) and lowest in AP (1948.57 ± 1483.94). Higher frame rates (30 FPS) lead to significantly higher DAP compared to lower frame rates (15 FPS) and Fluoroscopy. Cerebral procedures at 2 FPS have as higher KV values (83.65 ± 9.47) as at Left Coronay 30 FPS. Furthermore, Cerebral at 2 FPS have higher DAP values (7766.02 ± 7248.75) compared to others.

Table 1. Pairwise comparison of the ECG image diagnosis methods

Method-1 (Accuracy %)	Method-2 (Accuracy %)	p
READER (57.94%)	CARDIOLOGIST (92.52%)	1.19e-08
INTERPRETER (62.62%)	CARDIOLOGIST (92.52%)	3.77e-07
ANALYZER (62.62%)	CARDIOLOGIST (92.52%)	3.77e-07
READER (57.94%)	INTERPRETER (62.62%)	5.76e-01
READER (57.94%)	ANALYZER (62.62%)	5.76e-01
INTERPRETER (62.62%)	ANALYZER (62.62%)	1.00e+00

Table 1 presents a comparison of diagnostic methods for ECG images in pairs. Significant differences were observed between cardiologists (92.52%) and ChatGPT-based READER (57.94%), INTERPRETER (62.62%), and ANALYZER (62.62%) models. In all these comparisons, p-values were found to be below 0.05, indicating that cardiologists were significantly more accurate in their diagnoses.

Conclusions: Higher kV, mAs, and exposure times are associated with increased radiation dose, particularly in views such as Left-caudal and AP-caudal. Adjusting kV, mAs, and exposure times based on specific imaging views can enhance image quality while minimizing radiation dose. Cardiologists should continuously review and optimize imaging protocols to adhere to the ALARA (As Low As Reasonably Achievable) principle, ensuring patient safety and diagnostic accuracy.

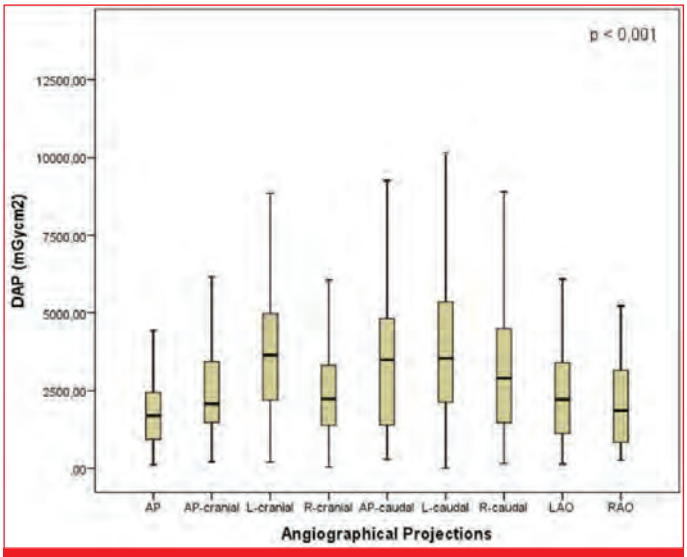

Figure 1. Dose area product values between different angiographic views obtained at 15 FPS.

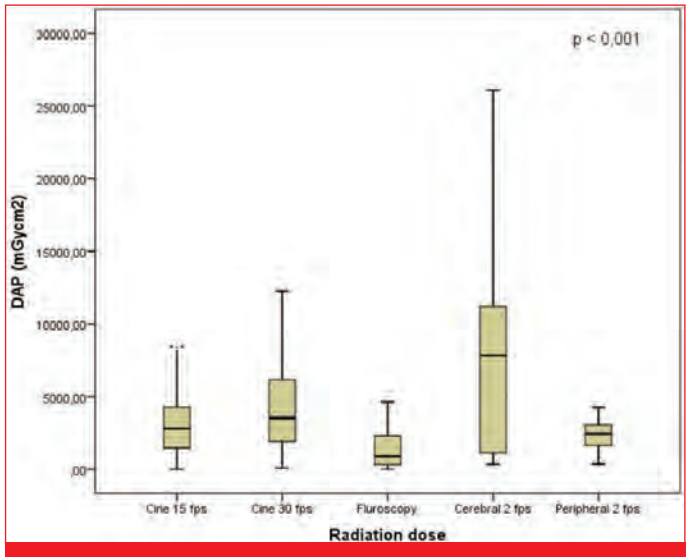

Figure 2. *Dose area product values between different cardiovascular procedures.*

Table 1. Radiation dose parameters between different angiographic views obtained at 15 FPS

Left coronary 15 FPS

	AP (n=69)	AP-cranial (n=83)	Left-cranial (n=347)	Right-cranial (n=273)	AP-caudal (n=186)	Left-caudal (n=282)	Right-caudal (n=310)	LAO (n=258)	RAO (n=35)	P value
kV	68,81 ± 6,24	76,56 ± 7,33	80,83 ± 8,94	75,37 ± 6,02	85,25 ± 8,57	85,60 ± 12,70	78,78 ± 9,24	73,50 ± 5,83	72,40 ± 4,23	< 0,001
m(A)s	500,89 ± 266,46	742,26 ± 129,65	814,05 ± 102,02	755,01 ± 127,32	865,22 ± 611,65	780,79 ± 12,75	769,35 ± 12161	713,71 ± 156,08	700,94 ± 199,47	< 0,001
ms	4,82 ± 1,49	6,34 ± 0,99	6,93 ± 1,05	6,17 ± 0,90	7,41 ± 0,86	7,28 ± 1,20	6,60 ± 1,14	6,12 ± 4,51	5,74 ± 0,85	< 0,001
DAP	1948,57 ± 1483,94	2462,68 ± 1618,89	3836,53 ± 3174,14	2423,75 ± 1538,20	3394,94 ± 2201,04	3888,43 ± 2422,80	3145,01 ± 2247,66	2360,64 ± 1464,21	2027,40 ± 1437,89	< 0,001

Table 2. Radiation dose parameters between fluoroscopy, 15 FPS and 30 FPS

	Fluroscopy (n=115)	Left coronary 15 FPS (n=1854)	Left coronary 30 FPS (n=110)	P value
kV	75,94 ± 14,24	78,99 ± 9,89	83,89 ± 10,87	< 0,001
m(A)s	17,40 ± 84,90	766,36 ± 241,99	716,62 ± 111,36	< 0,001
ms	0,06 ± 0,65	6,62 ± 2,04	7,13 ± 1,11	< 0,001
DAP, ($mGycm^2$)	2231,46 ± 4931,48	3091,22 ± 2331,67	4624,57 ± 4931,48	< 0,001

Cardiovascular Nursing / Technician

OP-013

Assessing the psychometric properties of the pulmonary arterial hypertension symptom scale

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Background and Aim: Pulmonary hypertension (PH) is a condition that can encompass many clinical conditions and can further complicate cardiovascular and respiratory diseases. PH is often characterised by symptoms, and these symptoms negatively affect patients physically and psychologically, leading to a reduction in their health-related quality of life and self-care. Knowledge of the symptoms experienced and their severity will help healthcare professionals to identify the issues that need to be focused on in the patient's care, thereby improving the patient's health-related quality of life and self-care. The aim of this study was to analyse the psychometric properties of the Turkish version of the Pulmonary Arterial Hypertension Symptom Scale (PAHSS).

Methods: The study sample consisted of 130 patients who were followed up at the pulmonary hypertension outpatient clinic in the cardiology department of a university hospital. The PAHSS was developed as a scale that assesses 17 symptoms and their severity that patients experience specific to PH. The scale focuses on the frequency and severity of symptoms experienced by patients in the last month. The data were analysed by the researchers using SPSS 25 and AMOS 24 in a computer environment. For scale validity, linguistic and conceptual equivalence, content validity, face validity, construct validity were assessed; for scale reliability, internal consistency reliability coefficient (Cronbach's alpha coefficient), item total score analysis, test-retest analysis, Tukey's test analysis were applied.

Results: The mean age of the patients included in the study was 60.81 ± 15.39 years and 70% (n=91) of the patients were female. 60.8% (n=79) of the patients experienced symptom exacerbation. The content validity index calculated according to the Lawshe technique applied to determine the content validity of the scale was found to be 0.94. Factor loadings for the cardiac sub-dimension of the PAHSS ranged from 0.553-0.901 and for the pulmonary sub-dimension from 0.457-0.811. The Cronbach's alpha coefficient of the scale was found to be 0.945. The Cronbach alpha coefficient of the cardiac subscale was 0.930 and the Cronbach alpha coefficient of the pulmonary subscale was 0.868. Item-total correlations were between 0.548-0.866 for the cardiac subscale and between 0.430-0.759 for the pulmonary subscale.

Conclusions: As a result of the analyses, the PAHSS was accepted as a valid and reliable scale introduced into the Turkish culture for the assessment of symptoms in patients with PH. The PAHSS is a scale that can be easily administered during routine outpatient follow-up, and by using the scale, it is possible to know what symptoms patients have experienced in the past month and to plan the treatment and care of patients accordingly.

Table 1. Fit Index Results of PAHSS

Fit Index Values	Reference Values	Research Results
CMIN/DF (Chi-Square / Degrees of Freedom)	<5	2.01
GFI (Goodness of Fit Index)	>0.70	0.84
NFI (Normed Fit Index)	>0.85	0.86
IFI (Incremental Fit Index-IFI)	>0.85	0.92
CFI (Comparative Fit Index)	>0.85	0.92
RMSEA (Root Mean Square Error of Approximation)	<0.08	0.08

Table 2. Confirmatory Factor Analysis Factor Loadings of PAHSS

	Symptoms	Factor Loadings
CARDIAC	Dizzy/Lightheaded	0.717
	Passing Out	0.849
	Awaken at Night Short of Breath	0.901
	Swelling of Ankles /Feet	0.553
	Cough	0.701
	Hoarseness	0.778
	Abdominal Swelling	0.705
	Nausea	0.861
	Loss of Appetite	0.769
	Numb, Painful Hands or Feet with Cold and Stress (Raynaud's Phenomena)	0.674
PULMONARY	Fatigue	0.457
	Chest Pain/Discomfort	0.755
	Fast Heart Beat/Palpitations	0.555
	Shortness of Breath at Rest	0.811
	Shortness of Breath with Exertion	0.554
	Shortness of Breath when Lying Down	0.773
	Difficulty Sleeping	0.778

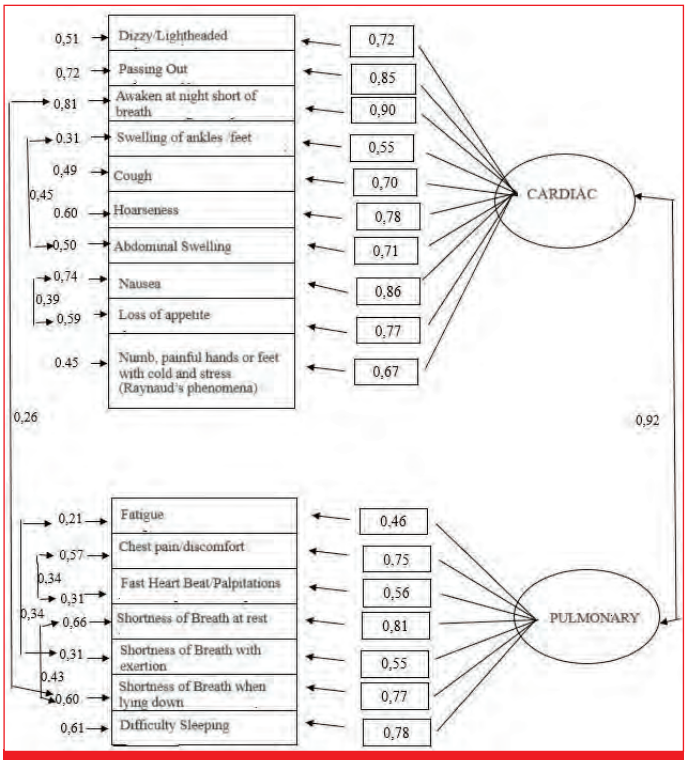

Figure 1. Confirmatory Factor Analysis Results of PAHSS.

Interventional Cardiology / Valvular and Structural Heart Diseases

OP-014

Long-term outcomes of balloon-expandable transcatheter aortic valve implantation according to Varc-3 in Turkish patients: A single-center study

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Background and Aim: Transcatheter aortic valve implantation (TAVI) is an established treatment for severe aortic stenosis (AS). There is limited data in the literature on balloon-expandable transcatheter heart valve (THV) long-term outcomes using VARC-3 criteria in Turkish patients.

Methods: This study was designed as a retrospective single-center study. The study included 270 consecutive patients who underwent TAVI with balloon-expandable Sapien XT valves for symptomatic severe AS between January 2012 and March 2017, and follow-up data were collected until April 2024. Clinical and procedural outcomes were evaluated according to VARC-3 criteria.

Results: The mean age of the patients was 82.5 ± 7.1 years, and 73.3% were women. The Society of Thoracic Surgeons (STS) mean score of the patients was 6.4% ± 3.1%. Transfemoral access was used in 99.3% of patients. Technical success was observed in 260 (96.3%), device success was observed in 245 (90.7%), and early safety was observed in 189 (70%) patients. All cause and cardiovascular mortality rates were 53.7% and 27.8% at 5-year follow-up, and 70,4% and 35,2% at 7-year follow-up, respectively. A total of 9.9% of patients required a permanent pacemaker implantation (PPI) at 30 days after implantation. The moderate (stage 2) or severe (stage 3) structural valve deterioration (SVD) rate at 7 years was 8.7%. The rate of bioprosthetic valve failure (BVF) at 7 years was 4.5%.

Conclusions: Patients with inoperable or high surgical risk treated with Sapien XT had a high 7-year mortality. Acceptable SVD and BVF rates indicate that Sapien XT valve performance is not poor at 7-year follow-up.

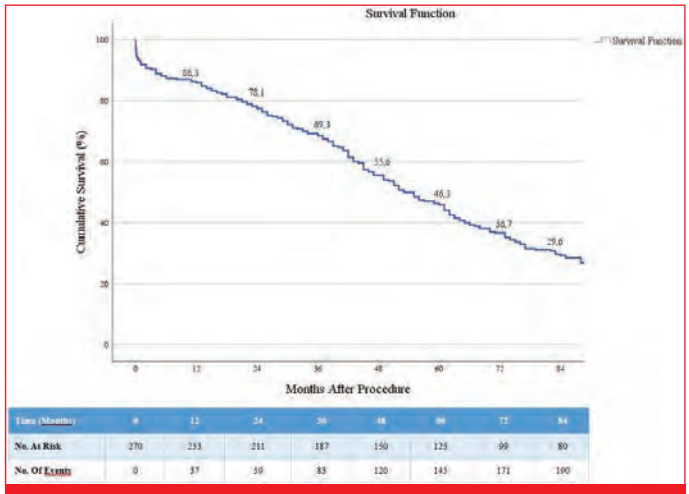


Figure 1. Cumulative survival rate.

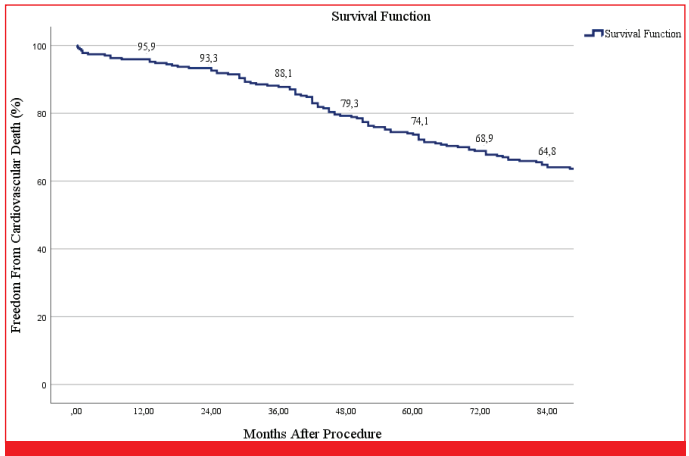


Figure 2. Freedom from cardiovascular death.

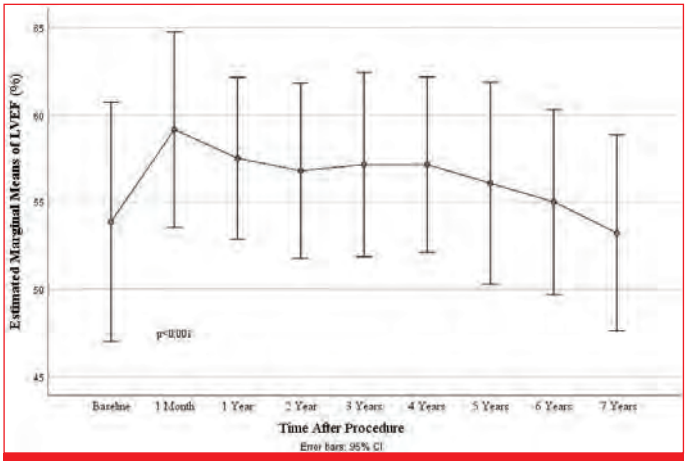
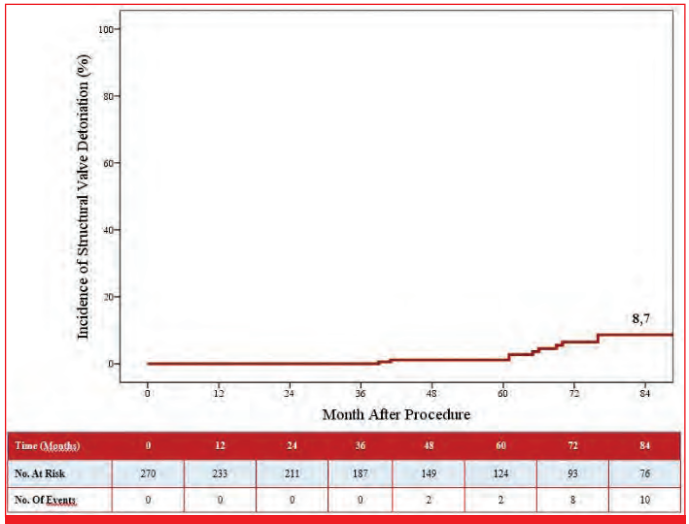


Figure 3. Progression of LVEF (%) from baseline through 7 years of follow-up.



Time (Months)	0	12	24	36	48	60	72	84
No. At Risk	270	233	211	187	149	124	93	76
No. Of Events	0	0	0	0	2	2	8	10

Figure 6. Cumulative incidence of structural valve deterioration.

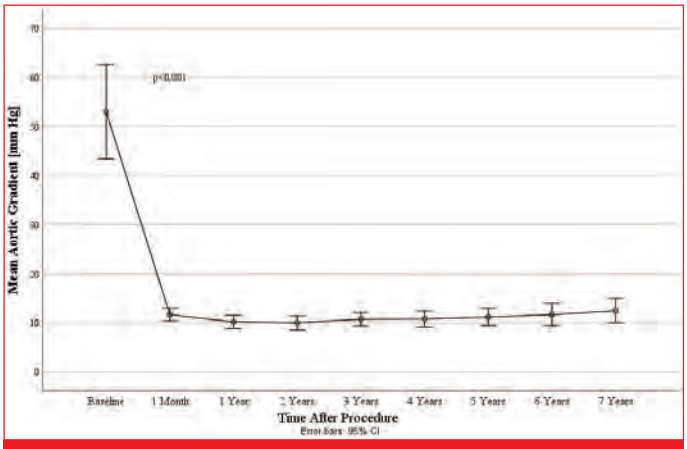
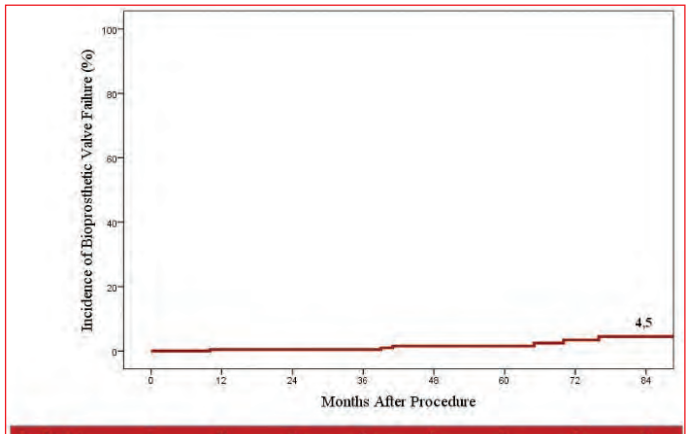


Figure 4. Progression of mean aortic valve gradient (mmHg) from baseline through 7 years of follow-up.



Time (Months)	0	12	24	36	48	60	72	84
No. At Risk	270	233	211	187	149	123	96	79
No. Of Events	0	1	1	1	3	3	5	6

Figure 7. Cumulative incidence of bioprosthetic valve failure.

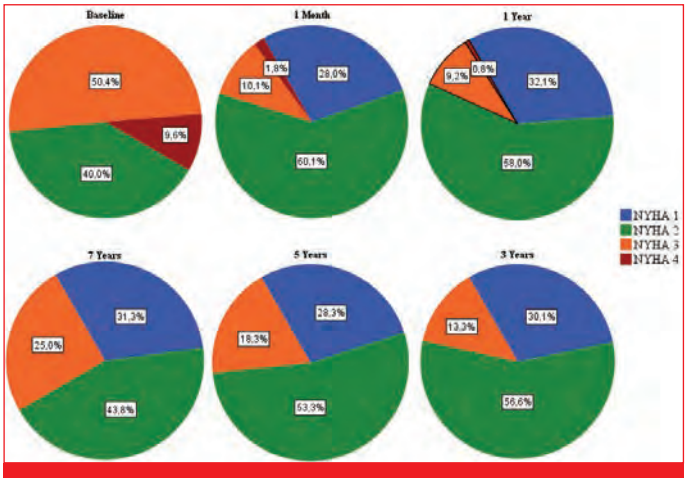


Figure 5. Change in NYHA functional class.

Table 1. Baseline demographic and clinical characteristics of the 270 patients

Parameters	All Patients (n=270)
Age (year)	82.5 ± 7.1
Female, n (%)	198 (73.3)
Body mass index (kg/m^2)	28.8 ± 6.4
Body surface area (m^2)	1.8 ± 0.2
Diabetes mellitus type II, n (%)	74 (27.4)
Arterial hypertension, n (%)	241 (89.2)
Dyslipidaemia, n (%)	82 (30.3)
Family history of coronary artery disease, n (%)	16 (5.9)
Current smoking, n (%)	36 (13.3)
Percutaneous coronary intervention, n (%)	51 (18.9)
Coronary artery bypass grafting, n (%)	45 (16.7)
Myocardial infraction, n (%)	40 (14.8)
Mitral valve replacement, n (%)	3 (1.1)
Hemodialysis, n (%)	2 (0.7)
Chronic obstructive pulmonary disease, n (%)	61 (22.6)
Ischemic stroke, n (%)	21 (7.8)
STS score %	6.4 ± 3.1
EuroSCORE II %	8.8 ± 4.7
Logistic EuroSCORE %	26.3 ± 14.6
Left ventricular ejection fraction %	53.1 ± 10.5
Aortic valve area (cm^2)	0.7 ± 0.2
Aortic valve area index (cm^2/m^2)	0.4 ± 0.1
Aortic mean gradient (mmHg)	46.5 ± 13.8
Serum glucose (mg/dL)	138 ± 54.1
Creatinine (mg/dL)	1.1 ± 0.7
Glomerular filtration rate ml/min/1.73 m2	60.9 ± 23.3
Hemoglobin (mg/dL)	11.7 ± 1.8
Atrial fibrillation, n (%)	64 (23.7)
Pacemaker, n (%)	8 (3)
Left bundle branch block, n (%)	30 (11.1)
Right bundle branch block, n (%)	23 (8.5)
Porcelain aorta, n (%)	2 (0.7)
Dextrocardia, n (%)	1 (0.4)
Classification of aortic stenosis n (%)	
High-gradient	235 (87)
Low-flow, low-gradient with reduced ejection fraction	24 (8.9)
Low-flow, low-gradient with preserved ejection fraction	11 (4.1)
Stages of heart failure, n (%)	
NYHA I	0 (0)
NYHA II	108 (40)
NYHA III	136 (50.4)
NYHA IV	26 (9.6)

Table 2. Procedural characteristics and periprocedural complications at 30-day follow-up

Procedural parameter	
Access site, n (%)	
Transapical	2 (0.7)
Transfemoral	268 (99.3)
Valve size mm, n (%)	
23	91 (33.7)
26	134 (49.6)
29	45 (16.7)
Predilatation, n (%)	207 (76.7)
Postdilatation, n (%)	9 (3.3)
Simultaneous TAVI and EVAR	3 (1.1)
Periprocedural (30-day) complication	
Major cardiac structural complicationa, n (%)	3 (1.1)
Multiple valves (ViV), n (%)	1 (0.4)
Valve migration/embolization, n (%)	4 (1.5)
Ischemic stroke, n (%)	5 (1.9)
Acute kidney injury (type 2-4), n (%)	15 (5.6)
Major vascular complication, n (%)	17 (6.3)
New permenant pacemakerb, n (%)	26 (9.9)
≥ Moderate paravalvular leak, n (%)	9 (3.3)
Bleeding (type 2-4), n (%)	47 (17.4)

TAVI: Transcatheter aortic valve implantation; EVAR: Endovascular aneurysm repair aAnnular rupture, cardiac tamponade, coronary obstruction (Left main coronary artery) bEight patients with pacemakers at baseline were not included.

Table 3. Primary end point(s) at 30-day, 1-year, 3-year, 5-year and 7-year follow-up, timing of mortality and composite endpoints of the 270 patients

Variable	30 days	1 year	3 years	5 years	7 years
All-cause mortality, yes (%)	19 (7)	37 (13.7)	83 (30.7)	145 (53.7)	190 (70.4)
Cardiovascular mortality, yes (%)	15 (5.6)	26 (9.6)	51 (18.9)	75 (27.8)	95 (35.2)
Non-cardiovascular mortality, yes (%)	4 (1.5)	11 (4.1)	32 (11.9)	70 (25.9)	95 (35.2)
Timing of mortality, n (%)					
Periprocedural mortality		19 (7)			
Early mortality		18 (6.7)			
Late mortality		153 (56.7)			
Composite endpoints, n (%)					
Technical success		260 (96.3)			
Device success		245 (90.7)			
Early safety		189 (70)			

Table 4. Causes and timing of all deaths (n=190)

Causes of death	Patients, n (%)	≤3 days	3-30 days	1-12 months	12-24 months	24-36 months	36-48 months	48-60 months	60-72 months	72-84 months
Cardiovascular	95 (50)									
Heart Failure	37 (19.5)	2	1	4	6	4	6	6	5	3
Acute myocardial infarctiona	13 (6.8)	1			3	2	2	1	2	2
Strokeb	11 (5.8)			2	1	2	2	3	1	
Prosthetic valve malposition	4 (2.1)	3	1							
Femoral access cite bleeding	3 (1.6)	2	1							
Arrhythmiac	2 (1.1)				1				1	
Acute pulmonary embolism	2 (1.1)				1					1
Cardiac tamponade	1 (0.5)	1								
Endocarditis	1 (0.5)			1						
Annular rupture	1 (0.5)	1								
Acute TAVI device faild	1 (0.5)	1								
Abdominal aortic rupture	1 (0.5)	1								
Acute mesenteric ischemia	1 (0.5)			1						
Sudden death	8 (4.2)			2	2		1	1		2
Unknown	9 (4.7)			1	1	2	2		3	
Non-cardiovascular	95 (50)									
Pneumonia	25 (13.2)			3	4	5	5	3	4	1
Coronavirus disease 2019	6 (3.2)						2	3		1
Other infection/sepsise	13 (6.8)		1	1	1	1	2	3	3	1
COPDf	5 (2.6)			1			3	1		
Traumag	10 (3.7)				1	1	2	1	3	2
Kidney diseaseh	11 (5.8)		3	1		1	2		2	2
Alzheimer disease/dementia	8 (4.2)					4	2	1		1
Gastrointestinal bleeding	5 (2.6)				1		2		1	1
Non-cardiac surgery related complicationsi	3 (1.6)						1	1		1
Diffuse alveolar hemorrhagej	1 (0.5)									1
Hepatic failure	1 (0.5)							1		
Malignancyk	7 (3.7)			1		2	3		1	

a One patient developed acute myocardial infarction due to left main coronary artery obstruction during the procedure. The other patients died due to acute myocardial infarction unrelated to the procedure. b One patient died of hemorrhagic stroke, the other patients died of ischemic stroke. c One patient died due to high-degree AV block at 13 months, and one patient died in hospital due to ventricular arrhythmia at 62 months. d After the first valve implantation, TAVI-in-TAVI was performed as a bailout therapy due to severe aortic regurgitation and hemodynamic collapse. The patient died intraprocedurally. e The origin of infection/sepsis was urinary in 3 patients; gastrointestinal in 1 patient. 7 patients had an unknown origin of infection (without signs of endocarditis). f End-stage chronic obstructive pulmonary disease with acute or chronic respiratory failure. g Fall, road accident and building collapse. h Acute kidney injury or chronic kidney disease. i Wound infection, wound dehiscence, bleeding. j Diffuse alveolar hemorrhage due to anticoagulant therapy. k Three patients died of lung cancer, one patient each died of colorectal cancer, pancreatic cancer, breast cancer and gastric cancer.

Table 5. Details of patients with structural valve deterioration and bioprosthetic valve failure at 7-year follow-up

Patients	Age, years	Gender	Valve size, mm	SVD	BVF	Stages of BVD	Timing of SVD, years	Treatment	Vital status
#1	84	Male	26	Yes	Yes	Stage 3	3.4	Medication	Dead
#2	75	Female	26	Yes	No	Stage 2	5.5	Medication	Dead
#3	84	Female	23	Yes	Yes	Stage 3	5.8	Medication	Dead
#4	62	Female	23	Yes	No	Stage 2	6.3	Medication	Alive
#5	77	Female	23	Yes	Yes	Stage 3	3.3	ViV	Dead
#6	85	Male	29	Yes	No	Stage 2	5.4	Medication	Dead
#7	85	Male	26	Yes	Yes	Stage 2*	5.1	Medication	Dead
#8	92	Female	23	Yes	No	Stage 2	5.1	Medication	Alive
#9	85	Female	26	Yes	Yes	Stage 3	6.3	ViV	Alive
#10	78	Male	29	Yes	No	Stage 2	5.8	Medication	Alive
#11	78	Female	26	No	Yes	Stage 1&	-	Medication	Dead

TAVI: Transcatheter aortic valve implantation; ViV: Valve-in-valve; SVD: Structural valve deterioration; BVF: Bioprosthetic valve failure; BVD: Bioprosthetic valve deterioration. *The patient with valve-related symptoms died of unknown cause 2 months after the diagnosis of stage 2 SVD. We have recorded this event as a valve-related death. &Endocarditis without significant haemodynamic changes.

Interventional Cardiology / Valvular and Structural Heart Diseases

OP-015

Evaluating the 30-day performance of the Myval valve compared to the Portico self-expanding valve

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Background and Aim: The Myval balloon-expandable (BE) valve has shown encouraging early clinical data in terms of safety and efficacy. Comparative data with other well-established contemporary valves are nonetheless still scarce. This study aims to compare the performance of the Myval BE valve with the Portico self-expanding (SE) valve. This study aims to compare the performance of the Myval BE valve with the Portico self-expanding (SE) valve.

Methods: In this retrospective single-center study, 266 patients with symptomatic severe aortic stenosis (AS) who underwent transcatheter aortic valve replacement were included and treated with the Myval BE valve (n=122) or with the Portico SE valve (n=144). The primary endpoint is a composite of device success, technical success and early safety according to the Valve Academic Research Consortium-3 criteria at 30-day follow-up.

Results: There were no significant differences between the two groups in terms of demographic data except diabetes mellitus. STS scores and echocardiographic features were similar. Hospital length of Stay was significantly longer in the Portico group. (p=0.020) Especially Valve malposition (p=0.004) and Need for a second valve (p=0.005) were significantly higher in the Portico group. Predilatation and postdilatation requirement were significantly higher in the Portico group (p<0.001). Therefore, it was observed that Contrast volume was higher in the Portico group (p<0.001). Paravalvular regurgitation was also significantly higher in the Portico group (p=0.005). No significant difference was observed in mortality (Myval: 7.4% - Portico: 10.4%, p=0.38), stroke (4.1% - 2.8%, p=0.73) and new permanent pacemaker (23% - 21%, p=0.66) at 30-day follow-up.

Conclusions: In our study, although there was no difference in mortality and stroke at 30 days in either valve group, the Myval valve demonstrated superior device success and paravalvular regurgitation compared to the Portico valve. A large randomized trial is needed to confirm these findings.

Table 1. Baseline characteristics of the study population

Parameters	Total (N=266)	Myval (N=122)	Portico (N=144)	p value
Demographic and clinical characteristics				
Age (years)	79.6 ± 6.1	79.0 ± 6.2	80.2 ± 5.9	0.115
Male	102 (38.3)	49 (40.2)	53 (36.8)	0.575
ypertension	205 (77.1)	99 (81.1)	106 (73.6)	0.145
Diabetes mellitus	121 (45.5)	64 (52.5)	57 (39.6)	0.036
Coronary artery disease	202 (75.9)	99 (81.1)	103 (71.5)	0.067
Coronary bypass	52 (19.5)	22 (18)	30 (20.8)	0.566
Stroke/TIA	8 (3)	5 (2.5)	5 (3.5)	0.730
Chronic kidney disease	73 (27.4)	31 (25.4)	42 (29.2)	0.494
eripheral artery disease	33 (12.4)	9 (7.4)	24 (16.7)	0.022
Pulmonary disease	60 (22.6)	21 (17.2)	39 (27.1)	0.055
Pacemaker	10 (3.8)	4 (3.3)	6 (4.2)	0.704
Atrial fibrillation	56 (21.1)	25 (20.5)	31 (21.5)	0.836
STS score	6.0 (4.1-8.2)	6.3 (4.8-8.2)	5.0 (4.0-8.1)	0.155
Echocardiographic characteristics				
LVEF (%)	56.5 (50.0-60.0)	55.5 (48.7-60.0)	59 (50.0- 60.0)	0.311
Aortic valve area (cm^2)	0.75 ± 0.20	0.69 ± 0.16	0.80 ± 0.27	0.304
Aortic mean gradient (mmHg)	47.5 ± 13.2	47.5 ± 14.4	47.4 ± 12.2	0.962
Aortic peak gradient (mmHg)	79.3 ± 42.5	80.9 ± 54.0	77.4 ± 22.6	0.497
Aortic peak velocity (m/s)	4.4 ± 0.6	4.4 ± 0.6	4.3 ± 0.55	0.879

Table 2. Procedural data and technical valve-related complications

Parameters	Total (N=266)	Myval (N=122)	Portico (N=144)	p value
Conscious anesthesia	237 (89.1)	108 (88.5)	129 (89.1)	0.782
Procedure time* (min)	78.7 ± 30.0	75.6 ± 34.7	81.3 ± 25.1	0.124
ICU length of Stay (days)	2.0 (1.0-4.0)	2.0 (1.0-5.0)	2.0 (1.0-3.0)	0.201
Hospital length of Stay (days)	7.0 (5.0-11.7)	8.0 (5.0-13.7)	7.0 (4.2-10.0)	0.020
Contrast volume, mL	150 (120-200)	150 (100-180)	150 (120-200)	<0.001
Predilatation	144 (54.1)	21 (17.2)	123 (85.4)	<0.001
Postdilatation	105 (39.6)	33 (27)	72 (50)	<0.001
Valve malposition	9 (3.4)	0 (0)	9 (6.2)	0.004
Need for a second valve	13 (4.9)	1 (0.8)	12 (8.3)	0.005
Coronary compression or obstruction	1 (0.4)	0 (0)	1 (0.7)	1.0
Annular rupture/Aortic dissection	2 (0.8)	2 (1.6)	0 (0)	0.209
Conversion to open surgery	2 (0.8)	1 (0.8)	1 (0.7)	1.0
Cardiac tamponade	5 (1.9)	1 (0.8)	4 (2.8)	0.379
Paravalvular regurgitation				0.005
None/trace	94 (35.7)	56 (45.9)	38 (27)	
Mild	159 (60.5)	63 (51.6)	96 (68.1)	
Moderate	10 (3.8)	3 (2.5)	7 (5)	
Severe	0 (0)	0 (0)	0 (0)	

Table 3. Composite endpoints.

Parameters	Total (N=266)	Myval (N=122)	Portico (N=144)	p value
Technical success	239 (89.8)	112 (91.8)	127 (88.2)	0.331
Freedom from mortality	263 (98.9)	122 (100)	141 (97.9)	0.252
Successful access, delivery of the device, and system retrieval	262 (98.5)	120 (98.4)	142 (98.6)	1.0
Correct positioning of a single valve in the proper location	253 (95.1)	121 (99.2)	132 (91.7)	000.5
Freedom from surgery or intervention related to procedural complications	255 (95.9)	115 (94.3)	140 (97.2)	0.227
Device success (at 30 days)	199 (74.8)	99 (81.1)	100 (69.4)	0.028
Technical success	239 (89.8)	112 (91.8)	127 (88.2)	0.331
Freedom from mortality	242 (91)	113 (92.6)	129 (89.6)	0.389
Freedom from surgery or intervention related to procedural complications	243 (91.4)	114 (93.4)	129 (89.6)	0.264
Intended valve performance*	249 (94.7)	117 (95.9)	132 (93.6)	0.410
Early safety (at 30 days)	173 (65)	75 (61.5)	98 (68.1)	0.262
All-cause mortality	24 (9.0)	9 (7.4)	15 (10.4)	0.389
All stroke	9 (3.4)	5 (4.1)	4 (2.8)	0.736
VARC type 2–4 bleeding	37 (13.9)	21 (17.2)	16 (11.1)	0.152
Major vascular, access-related, or cardiac structural complication	23 (8.6)	8 (6.6)	15 (10.4)	0.264
Acute kidney injury stage 3 or 4	14 (5.3)	5 (4.1)	9 (6.2)	0.434
Moderate or severe aortic regurgitation	10 (3.8)	3 (2.5)	7 (5)	
New permanent pacemaker	60 (22.6)	29 (23.8)	31 (21.5)	0.663
Surgery or intervention related to the device	243 (91.4)	114 (93.4)	129 (89.6)	0.264

Interventional Cardiology / Valvular and Structural Heart Diseases

OP-016

Role of CT in predicting in-one year mortality for TAVI: Multicenter TAVI registry

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Background and Aim: In patients undergoing surgical aortic valve replacement due to severe aortic valve stenosis (AS), the presence of right ventricular damage markers (RVDM) determined by echocardiography is a poor prognosis indicator and is associated with increased mortality. Such data is not available in the patient group who underwent transcatheter aortic valve replacement (TAVI). The aim of this study is to investigate the prognostic value of RVDM determined by cardiac CT in the patient group undergoing TAVI.

Methods: This multicenter, retrospective study included 184 patients who underwent TAVI between 2020-2022 and had at least 1-year follow-up data. TAVI due to severe AS was taken as the inclusion criterion. Exclusion criterias were defined as previous tricuspid or pulmonary interventions and deaths from procedural complications. Baseline clinical and echocardiographic characteristics of the patients were recorded. In CT, pulmonary artery diameter (PAD) and right ventricular outflow tract myocardial thickness (RVOTMT) in mid-systole and maximal right and left ventricular (RV/LV) diameter ratios in mid-diastole were meausered as indicators of RVDM. Meauserements were made double-blindly in the core laboratory by a radiologist and a cardiologist and their averages were taken (Figure 1). The primary end point of the study was determined as death at 1-year follow-up.

Results: Of the 184 patients included in the study, 54.1% were female (n=99), mean age of the patients was 76.9 ± 7.3. Before the procedure, the mean aortic valve gradient was 52 ± 10. Death at 1-year follow-up, which was the primary endpoint of the study was observed in 42 patients (22%). Among the clinical features, the rates of DM, HT and COPD were similar in both groups, while the coronary artery disease rate and age were higher in the group with 1-year death. Among the laboratory parameters GFR was lower in the death group, and the STS score was similar in both groups. While EF, one of the echocardiographic parameters, was similar in both groups, sPAP and right ventricular dilatation rate were higher in the group with death. Among the CT parameters, PAD, RVOTMT and maxial RV/LV diameter ratio were observed to be significantly higher in the group with death (p<0.001) (Table). In the ROC analysis, PAD of 30.5 and above has 78% sensitivity and 82% specificity (AUC: 0.87 95% CI: 0.82-0.93, p<0.001), RVOT MT of 4 mm and above has 90% sensitivity and 87% specificity (AUC: 0.93 95% CI: 0.87-99, p<0.001), maximal RV/LV diameters ratio of 0.91 and above showed 90% sensitivity and 92%

specificity (AUC: 0.94 95% CI: 0.89-0.99, p<0.001) to predict 1-year mortality (Figure 2).

Conclusions: This study showed that RVDM determined by Cardiac CT (PAD, RVOTMT and Maximal RV/LV diameter ratio) were strong predictors of 1-year mortality in the patient group undergoing TAVI. As far as we have concerned our study is the first in the litreture showing preoperative cardiac CT can be used to 1-mortality in severe AS treated with TAVI.

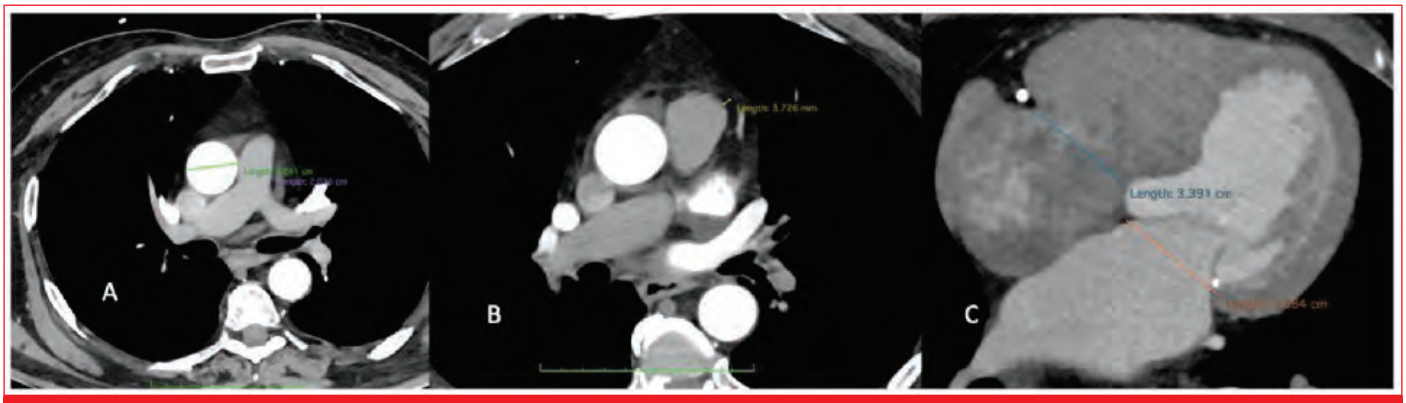

Figure 1. Cardiac tomography measurements. A: Pulmonary artery diameter in mid-systole, B: Right ventricular outflow tract thickness in mid-systole, C: Maximal right and left ventricular diameter ratios in mid-diastole.

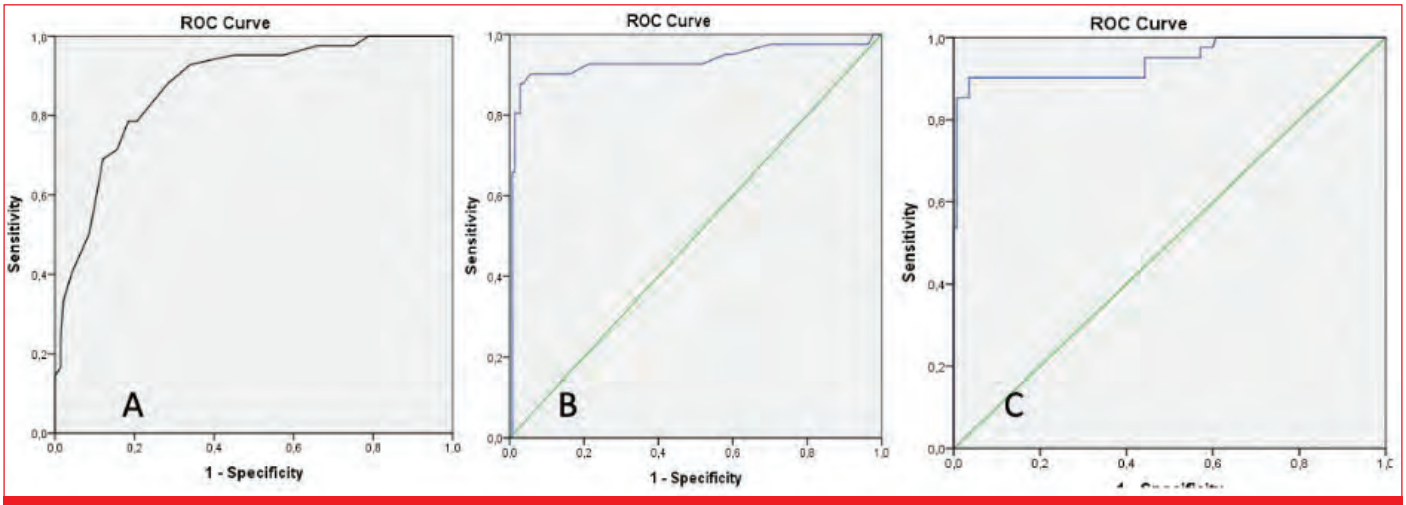

Figure 2. ROC curves representing the relationship between the right ventricular damage markers and one year mortality. A: Pulmonary artery diameter, B: Right ventricular outflow tract thickness, C:maximal right and left ventricular diameter ratios.

Table 1.

	Surviving group (n=142)	Exitus Group (n=42)	p
Age (year) [Median (IQR)]	76 (9)	81 (9.5)	0.002
GFR [Median (IQR)]	71 (32)	52 (33)	<0.001
sPAB mmHg [Median (IQR)]	40 (27)	45 (33)	0.014
STS score [Median (IQR)]	12 (5.7)	12.1 (3.4)	0.155
PA diameter mm [Median (IQR)]	25 (13)	34.5 (7.25)	<0.001
RVOT thickness (mm) [Median (IQR)]	3.5 (0.8)	6.4 (1.85)	<0.001
Maximal RV/LV diameter (Mean ± SD)	0.77 ± 0.12	1.15 ± 0.19	<0.001
Coronary artery disease n (%)	102 (72.3%)	36 (84%)	0.04
RV dilatation n (%)	22 (22.9%)	18 (48.6%)	0.03

GFR: Glomerular filtration rate; sPAB: Systolic pulmonary artery pressure; PA: Pulmonary artery; IQR: Interquartile range; SD: Standart deviation; RV: Right ventricle; LV: Left ventricle.

Interventional Cardiology / Valvular and Structural Heart Diseases

OP-018

The safety and efficiency of pericardial window by using uniportal video-assisted thoracoscopic surgery for the treatment of pericardial effusion: A single-center experience

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Background And Aim: In a healthy individual, the pericardium contains between 15 and 50 mL of serous fluid. Pericardial effusion (PE) is an acute or chronic accumulation of fluid over 50 mL in the pericardial cavity. Pathologies that can cause increased production or impaired fluid absorption in the pericardium result in PE, which can lead to clinical conditions ranging from asymptomatic cases to life-threatening cardiac tamponade. Subxiphoid fenestration, pericardial window (PW) opening with thoracotomy, and PW opening with video-assisted thoracoscopic surgery (VATS) are the three surgical methods used in the surgical treatment of PE. PW is the surgical procedure in which, a part of the pericardium is removed so that the effusion can flow into the chest cavity, and the mass effect caused by recurrent PE (usually malignant) is prevented. Another advantage is that it will provide a definitive treatment by limiting the occurrence of PE and/or tamponade.

Methods: 35 patients were enrolled. Patients were analyzed for gender, age, symptom, operation side, discharge time, complications, and pathologic specimens. Pre-operative thoracic computed tomography was used to determine other chest pathologies and the operation side. The localization and amount of PE determined by pre-operative transthoracic echocardiography (TTE) were used to choose the level of the intercostal incision. Based on those views, mid-axillary lines 4, 5, or 6 intercostal spaces were preferred. On the first post-operative day, chest X-ray was taken and TTE was performed. Patients were referred to their relevant clinics for follow-up. Other patients were discharged at 24 hours for outpatient control after ten days. Operative mortality was defined as mortality within 30 days after surgery.

Results: Of the 35 patients; 25 (71.4%) were male, 10 (28.6%) were female, and the mean age was 60.1 ± 15.4 years. The left side was preferred for 28 patients (80%) and the right for 7 (20%). The median BMI was 25.5 kg/m^2 (24.0-28.1). The median day of stay in our clinic and/or referred clinic was 1 (1-2). Four patients (11.4%) who were previously diagnosed with malignancy died within the first month postoperatively. The mortalities were not attributed to the pericardial procedure. There was no microbial growth in the culture samples. Histopathological diagnoses were reported as chronic non-specific inflammation in 21 patients (60%), hyalinization in 3 (8.5%), malignancy in 9 (25.7%), and acute inflammation in 2 (5.7%). Pericardial fluids were checked by performing TTE on the wound site on the 10th postoperative day. Pericardial fluid accumulation and any postoperative complications were not observed. The median follow-up period of the patients was 385 (322-485) days. We did not encounter any recurrence of PE.

Conclusions: PW opening using uniportal VATS seems to be a safe method for patients with PE without any need to one-lung ventilation. In addition, uniportal VATS can be considered as the first surgical option in obese patients.

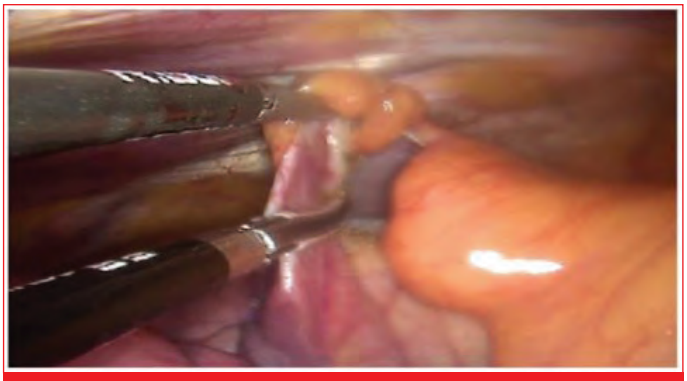

Figure 1.Window created by surgical energy device.

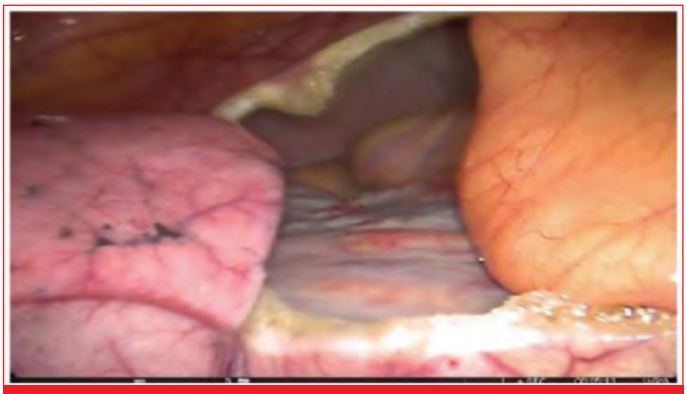

Figure 2. Pericardial window created with pericardiectomy.

Table 1. Data on patients' gender, operation side and type of intubation

Variables	
Gender	25 male (71.4%) 10 female (28.6%)
Operation side	28 left (80%), 7 right (20%)
Type of intubation	11 OLV (31.4%), 10 DLV (28.6%), 14 NI (40%)

Table 2. Data on patients' age, BMI, and length of stay in the clinic

Variables	
Age, years	60.1 ± 15.4
BMI (kg/m^2)	25.5 (24.0-28.1)
Stay in clinic, days	1 (1-2)

Interventional Cardiology / Carotid and Peripheral Vascular

OP-019

Balloon embolization of bronchial artery: A new technique in massive hemoptysis

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Background and Aim: Massive hemoptysis, characterized by severe respiratory bleeding, requires immediate medical attention due to its high mortality rate. We described our experience with the parachute balloon embolization procedure for life-threatening massive hemoptysis in this article.

Methods: The study was planned for 14 patients who presented with massive hemoptysis, had a high surgical risk, and failed medical and bronchoscopic treatment. The bleeding focus was embolized by placing the balloon, which was cut in half and separated from its shaft, using the new parachute technique. Bronchial artery embolization was performed in all cases by entering through the femoral artery. Diagnostic angiography of the aortic arch and descending thoracic aorta was performed to view the bronchial artery by advancing a multihole catheter (Pigtail Cook, Bloomington, Indiana) over a 0.035-inch hydrophilic guidewire (Terumo, Japan) into the thoracic aorta. Afterwards, the artery was engaged with 7F Amplatz Left or right 2 catheters. The embolization process was planned with the method we call the parachute technique. Balloon diameters between 2.0 and 4.0 were selected depending on the target artery (Solaris, medtronic). Ballons were inflated to a pressure of 4 atm. Downloaded after 30 seconds. Then the balloon part was separated by cutting from the distal shaft part. The balloon was cut in half. The two formed parts (the main balloon shaft and the cut distal part of the balloon) were separately loaded on the floppy wire with the cut side of the balloon opposite to the flow direction (parachute effect). Using 0.014 wire, it was attempted to be moved to the lesion region. The cut shaft of the balloon was pushed with its half, and the material to be left was stabilized by keeping the balloon shaft fixed to the proximal bronchial artery. Because of the unidirectional artery flow, the balloon was expanded slightly and positioned in the lesion area as a result. And the wire was pulled slowly and retracted. The balloon shaft was then pulled back from the remaining piece. If satisfactory embolization was not achieved, the same process was repeated in the bleeding pulmonary artery branch.

Results: Hemorrhage was effectively controlled in all individuals. One patient died during the in hospital follow-up. 5 of our patients needed intensive care for 2-6 days (average 3.6 days). The number of hospitalization days, including the intensive care and service, of the patients, excluding the patient with Exitus, was between 2 and 10 days, and the patient was discharged with recovery in an average of 5.6 days. During the 6-month follow-up examination of the patients, there were no instances of complications or recurrence of bleeding.

Conclusions: The use of balloon embolization using the parachute approach can be an effective and safe bail out treatment option for those with severe hemoptysis. Furthermore, this technique may facilitate the development of new treatment modalities and the creation of novel devices in the future.

Figure 1. A: Ballons were inflated to a pressure of 4 atm. Downloaded after 30 seconds, B: Then the balloon part was separated by cutting from the distal shaft part, C: The balloon was cut in half, D: The main balloon shaft and the cut distal part of the balloon were prepared separately load on the floppy wire with the cut side of the balloon opposite to the flow direction (parachute effect).

Figure 2. The balloon main shaft and the cut balloon distal part are loaded on 0.14 wire to be transported to the target artery.

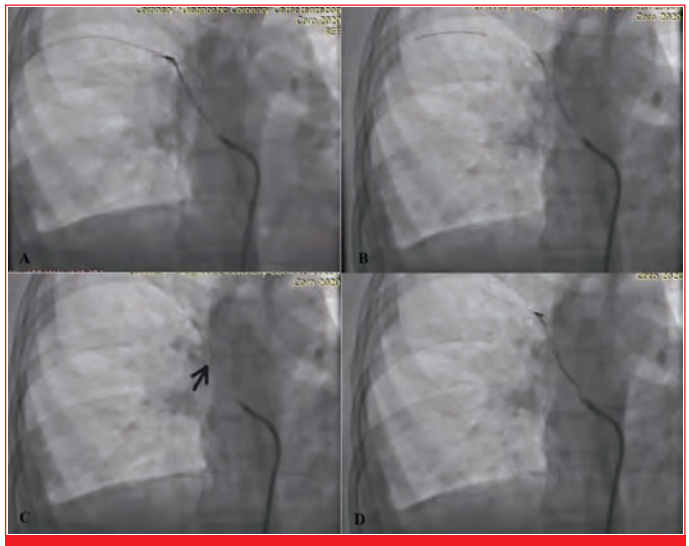

Figure 3. A: Bronchial artery was engaged with 7F Amplatz Left or Amplatz right 2 catheters, B: The cut shaft of the balloon was pushed with its half, and the material to be left was stabilized by keeping the balloon shaft fixed to the proximal bronchial artery. Due to the unidirectional flow of the artery, the balloon was slightly expanded and placed in the location of the lesion, C: After the wire is retracted, the balloon is observed inside the artery (arrow), D: Control injection confirms that the vessel is occluded.

Cardiovascular Nursing / Technician

OP-020

Nursing in percutaneous coronary and valvular interventions care

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Background and Aim: Coronary heart diseases today affect the health of people in productive ages. It is the most important health problem that threatens. The mortality rate due to heart diseases has increased despite all preventive and therapeutic developments and despite new methods, it is one of the leading causes of death. These patients this is because it continues to be the most important cause of mortality and morbidity. The awareness of intensive care nurses on this issue is important. Responsibilities of the nurse in the care of the patient receiving interventional treatment; preventing possible complications, early recognition. Patient/family education and it is rehabilitation prevention and early recognition of possible complications, individualized care and patient/family education, modification of risk factors. Necessary lifestyle changes and prognosis in interventional treatments in cardiology are important factors affecting and structured.

Methods: Nurse's support for individualized and structured care and patient/family education Following the latest developments and literature and attend nursing seminars It is important to do.

Results: Care is provided in line with the nursing process. Medical, physical examination, hemodynamic monitoring and diagnostic evaluation of the patient by analyzing and interpreting the data including the tests, most of the common nursing diagnoses are made and care is planned, implemented and evaluated.

Conclusions: We can reduce mortality and morbidity with good nursing care.

Interventional Cardiology / Coronary

OP-022

Clinical outcomes of double kissing culotte and mini-culotte stenting in non-left main coronary bifurcation lesions: The OPTIMUM trial

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Background and Aim: Culotte stenting is one of the most commonly used techniques in coronary bifurcation lesion (CBL). Double kissing mini-culotte (DKC) stenting is the most updated and modified form of culotte stenting recommended by current guidelines. We aimed to compare the DKC with the mini-culotte (MC) in non-left main true CBL.

Methods: A total of 193 patients with non-left main true CBL undergoing percutaneous coronary intervention were categorized as MC stenting (n=92) and DKC stenting (n=101) groups. The primary endpoint of the study was target lesion failure (TLF) which was a composite outcome of cardiac death, target vessel myocardial infarction (TVMI) and target lesion revascularization (TLR) at 1-year and 3-year follow up.

Results: There was no difference in the TLF between MC and DKC techniques at 1-year follow-up. At 3-year, the incidence of TLF was lower in DKC compared to MC [18 (19.6%); 6 (5.9%), p=0.007] that was mainly driven by the TLR [13 (14.1%); 5 (5.0%), p=0.035]. The number of patients with TVMI [4 (4.3%); 3 (3.0%), p=0.617] and cardiac death [5 (5.4%); 1 (1.0%), p=0.118] was also lower in the DKC group.

Conclusions: In the true CBLs, the DKC technique was superior compared to MC technique in terms of TLF and TLR at 3-year follow-up. There were no differences in cardiac death and TVMI between DKC and MC techniques at 1-year and 3-year follow-up.

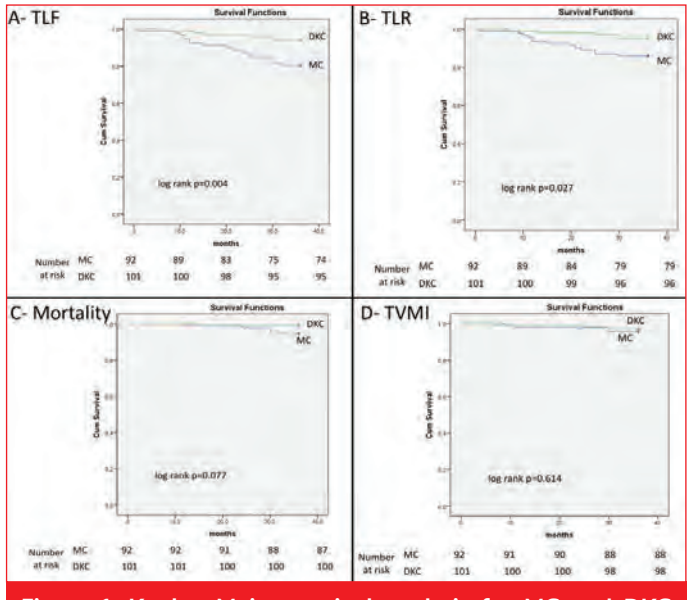

Figure 1. Kaplan Meier survival analysis for MC and DKC stenting techniques in terms of A- TLF, B- TLR, C- Cardiac death, D- TVMI.

DKC: Double kissing culotte, MC: Mini-culotte, TLF: Target lesion failure, TLR: Target lesion revascularization, TVMI: Target vessel myocardial infarction

Table 1. Baseline clinical and laboratory parameters of patients

	mini-Culotte (n=92)	DK-Culotte (n=101)	p
Age (years)	62±10.2	63.3 ± 8.4	0.319
Gender (female), n (%)	23 (25.0)	21 (20.8)	0.486
Hemoglobin (g/dL)	13.82 ± 1.71	13.51 ± 1.6	0.193
Thrombocyte x10^3/mm^3	245.5 (206-291.5)	244 (211-285)	0.868
Leukocyte x10^3/mm^3	8.27 (6.97-10.61)	8.3 (6.53-10.0)	0.193
Creatinine (mg/dL)	0.86 (0.71-1.0)	0.85 (0.79-0.98)	0.789
Total cholesterol (mg/dL)	190.7 ± 54	185.7 ± 54.9	0.545
LDL cholesterol (mg/dL)	116.5 ± 45.7	115.9 ± 44.0	0.927
HDL cholesterol (mg/dL)	42 (36-50)	44 (38-52)	0.248
Triglyceride (mg/dL)	151 (103-201)	155 (125-198)	0.052
Glucose (mg/dL)	115 (98.5-180)	110 (90-147)	0.062
Diabetes mellitus, n (%)	41 (44.6)	48 (47.5)	0.680
Hypertension, n (%)	50 (54.3)	61 (60.4)	0.396
Smoking, n (%)	30 (32.6)	39 (38.6)	0.385
Previous PCI, n (%)	19 (20.7)	25 (24.8)	0.498
Atrial fibrillation, n (%)	4 (4.3)	6 (5.9)	0.434
Oral anticoagulation, n (%)	5 (5.4)	6 (5.9)	0.880
Beta blocker, n (%)	87 (94.6)	93 (92.1)	0.491
ACEI/ARB, n (%)	82 (89.1)	90 (89.1)	0.996
Statin, n (%)	90 (97.8)	99 (98.0)	0.654
Calcium channel blocker, n (%)	17 (18.5)	21 (20.8)	0.686
Clinic presentation, n (%) Stable USAP/NONSTEMI	 42 (45.7) 50 (54.3)	 45 (44.6) 56 (55.4)	0,878
Ejection fraction (%)	52.5 ± 9.8	51.7 ± 7.9	0.548

ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, DK: Double kissing, HDL: High density lipoprotein, LDL: Low density lipoprotein, NONSTEMI: Non ST segment elevation myocardial infarction, PCI: Percutaneous coronary intervention, USAP: Unstable angina pectoris.

Table 2. Clinical outcomes

		mini-Culotte (n=92)	DK-Culotte (n=101)	Hazard Ratio (95% CI)	p
1-year outcome					
TLF, n (%)		7 (7.6)	1 (1.0)	0.128 (0.016-1.036)	0.054
	TLR, n (%)	6 (6.5)	1 (1.0)	0.149 (0.018-1.240)	0.078
	Target vessel MI, n (%)	2 (2.2)	1 (1.0)	0.457 (0.041-5.038)	0.522
	Cardiac death, n (%)	1 (1.1)	0 (0)	0.014 (0-137)	0.603
Secondary endpoint, n (%)					
	Definite stent thrombosis, n (%)	1 (1.1)	0 (0)	0.014 (0-137)	0.603
3-year outcome					
TLF, n (%)		18 (19.6)	6 (5.9)	0.282 (0.112-0.711)	0.007
	TLR, n (%)	13 (14.1)	5 (5.0)	0.330 (0.118-0.927)	0.035
	Target vessel MI, n (%)	4 (4.3)	3 (3.0)	0.682 (0.153-3.049)	0.617
	Cardiac death, n (%)	5 (5.4)	1 (1.0)	0.180 (0.021-1.541)	0.118
Secondary endpoint, n (%)					
	Definite stent thrombosis, n (%)	1 (1.1)	1 (1.0)	0.906 (0.057-14.484)	0.944

DK: Double kissing, MI: Myocardial infarction, TLF: Target lesion failure, TLR: Target lesion revascularization.

Table 3. Bifurcation lesion, coronary artery disease and procedural characteristics

	mini-Culotte (n=92)	DK-Culotte (n=101)	p
MV proximal stenosis (%)	69.6 ± 14.7	69.4 ± 12.9	0.887
MV distal stenosis (%)	72.9 ± 13.1	73.2 ± 13.2	0.880
SB stenosis (%)	88.8 ± 5.8	87.9 ± 7.1	0.319
MV reference vessel diameter (mm)	3.01 ± 0.32	3.0 ± 0.26	0.767
SB reference vessel diameter (mm)	2.87 ± 0.26	2.85 ± 0.23	0.599
MV stent length (mm)	26.9 ± 6.7	25.0 ± 8.2	0.086
SB stent length (mm)	21.2 ± 5.3	22.0 ± 6.6	0.342
Bifurcation angle (°)	60 (51-66)	61 (51-71)	0.494
Multivessel disease, n (%)	41 (44.6)	55 (54.5)	0.308
SYNTAX score	16.1 ± 4.6	16.5 ± 4.9	0.552
Complex bifurcation, n (%)	60 (65.2)	72 (71.3)	0.365
MV lesion length (mm)	25.89 ± 6.67	24.03 ± 8.17	0.086
SB lesion length (mm)	16.3 ± 4.7	19.1 ± 2.5	<0.001
Calcification MV, n (%)	5 (5.4)	7 (6.9)	0.067
Calcification SB, n (%)	5 (5.4)	3 (3.0)	0.310
Guiding catheter size, n (%) 6F 7F	 3 (3.3) 89 (96.7)	 0 (0) 101 (100.0)	0.106
Arterial access, n (%) Femoral Radial	 91 (98.9) 1 (1.1)	 101 (100.0) 0 (0)	0.477
MV predilatation, n (%)	63 (68.5)	72 (71.3)	0.671
SB predilatation, n (%)	35 (38.0)	42 (41.6)	0.616
Final kissing balloon, n (%)	87 (94.6)	101 (100.0)	0.023
POT, n (%)	89 (96.7)	97 (96.0)	0.552
Procedural time (min)	33 (14-53)	26 (13-43)	0.310

CXA: Circumflex artery, DK: Double kissing, LAD: Left anterior descending artery, MV: Main vessel, RCA: Right coronary artery, POT: Proximal optimization technique, SB: Side branch.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-023

Gender related clinical characteristics and supraventricular arrhytmias in patients with Brugada syndrome and effects on prognosis in a large cohort of patients: A single center observational study

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Background and Aim: Brugada Syndrome (BS) is a significant cause of sudden cardiac death (SCD) in otherwise healthy young adults. Although BS is more commonly observed in males, female patients are also at considerable risk. Supraventricular tachyarrhythmias (SVT) have been detected at notably higher rates in BS patients compared to the general population, with significant gender-related differences in clinical outcomes. Our study aims to evaluate the prevalence and clinical characteristics of SVTs in BS patients, focusing on gender-related prognostic differences.

Methods: We conducted a single-center, prospective observational study involving 82 BS patients (52 males, 30 females) diagnosed in the last 2 years with a mean follow-up of 12 ± 5.3 months (median 7 months). The study excluded patients with structural heart disease or those who had undergone prior epicardial ablation. Baseline ECG parameters, SVT recurrences, and clinical outcomes, including ICD interventions and SCD events, were recorded and analysed.

Results: Baseline parameters together with basal ECG parameters were recorded (Tables 1-3). On a mean follow

up of 12 ± 5.3 months, the clinical events and symptoms were determined (Table 4). 18 (22%) of them had an ICD implanted. 3rd and 6th months of ICD controls were done in the available patients (Table 4). The analyses have shown that males were highly symptomatic, demonstrating more spontaneous type-1 and previous history of atrial fibrillation (AF). There was a trend in women with BS to demonstrate more SND, more SVT diagnosis and more history of AV-nodal reentry tachycardia ablations. ICD implantation rates were similar between each gender. There was a trend towards longer PR and HV intervals in men and longer QRS and QTc durations in women (Table 2). When we analysed the age groups, there was also a trend towards the younger subjects on the presumably higher reproductive state demonstrating significantly prominent ST segment/j-point elevations on Ajmalin Drug Challenge Test than the older individuals, together with higher PR, QRS and QTc intervals (Table 3). In the follow up, none of the patients suffered a SCD event, while there was a trend towards the women demonstrating significantly more high atrial rate episodes on ICD controls and towards males demonstrating more appropriate ICD shock events (Table 4).

Conclusions: Women with BS appear to be more susceptible to symptomatic dysrhythmic/SVT events, while males demonstrate a higher likelihood of ventricular events requiring ICD interventions and significantly more atrial fibrillation episodes. ECG and drug challenge tests seem to be affected by the age and reproductive stage. These findings suggest that gender and age might be regarded as a critical consideration in the risk stratification and management of BS patients highlighting the need for tailored therapeutic strategies. Limited data on hormonal and gender specific modulation on cardiac ion channels might be regarded as a probable mechanism explaining our results.

Table 1. Clinical Characteristics

Parameters	Overall (n=82)	Men (n=52)	Women (n=30)	p-value
Age at Diagnosis (years, Mean ± SD)	36.5 ± 15	31.0 ± 12	44.5 ± 14.5	0.01*
Proband n (%)	77 (94.2%)	47 (90.4%)	30 (100%)	0.08
Family History of SCD n (%)	40 (48.8%)	26 (50%)	14 (46.7%)	0.75
Asymptomatic n (%)	47 (57.3%)	31 (59.6%)	12 (40%)	0.10
Syncope n (%)	55 (67.1%)	37 (71.2%)	18 (60%)	0.30
Aborted SCD (ASCD) n (%)	3 (3.7%)	2 (3.8%)	1 (3.3%)	0.90
Age at 1st Symptom (years)	20 ± 19.32	19.2 ± 19.69	21 ± 17.52	0.65
Previous AF n (%)	19 (23.2%)	15 (28.8%)	4 (13.3%)	0.04*
Previous SND n (%)	6 (7.3%)	1 (1.9%)	5 (16.7%)	0.01*
Spontaneous Type I ECG n (%)	7 (8.5%)	6 (11.5%)	1 (3.3%)	0.01*
Diagnosis by DCT n (%)	74 (90.2%)	45 (86.5%)	29 (96.7%)	0.10
EPS n (%)	78 (95.1%)	49 (94.2%)	29 (96.7%)	0.65
EPS Inducible n (%)	3 (3.7%)	2 (3.8%)	1 (3.3%)	0.90
AVNRT Ablation n (%)	15 (18.3%)	7 (13.5%)	8 (26.7%)	0.15
AVNRT Recurrence n (%)	2 (2.4%)	1 (1.9%)	1 (3.3%)	0.65
Any SVT n (%)	46 (56.1%)	24 (46.2%)	22 (73.3%)	0.03*
ICD Implantation n (%)	16 (19.5%)	12 (23.1%)	4 (13.3%)	0.20

Abbreviations: SCD: Sudden cardiac death, ASCD: aborted sudden cardiac death, AF: atrial fibrillation, SND: sinus node dysfunction, ECG: electrocardiogram, DCT: drug challenge test, EPS: electrophysiological study, ICD: implantable cardioverter defibrillator, AVNRT: Atrio Ventricular Nodal Reentry Tachycardia*Percentages are calculated among patients in each gender or overall group and values are presented as (mean ±SD)

Table 2. ECG Characteristics of the Study Population at Diagnosis

Parameters	Overall (n=82)	Men (n=52)	Women (n=30)	p-value
PR Interval (ms, Mean ± SD)	164.54 ± 31.27	172.94 ± 32.02	154.47 ± 30.81	0.02*
QRS Duration (ms, Mean ± SD)	94.16 ± 26.89	93.56 ± 19.66	99.00 ± 29.23	0.15
QTc in DII (ms, Mean ± SD)	408.77 ± 35.58	400.59 ± 40.37	418.57 ± 33.72	0.01*
ST Elevation after DCT (mm, Mean ± SD)	3.45 ± 1.05	3.90 ± 1.08	2.91 ± 0.98	0.003*
EPS HV Interval (ms, Mean ± SD)	44.45 ± 9.94	45.17 ± 8.70	44.20 ± 10.36	0.40

***Percentages are calculated among patients in each gender or overall group and values are presented as (mean ±SD)**

Abbreviations: EPS: electrophysiological study, HV: HV interval

Table 3. ECG Characteristics of the Study Population According to Age

Parameters	Overall (n=82)	Younger Adults (18-24 years, n=26)	Older Adults (25-54 years, n=56)	p-value
PR Interval (ms, Mean ± SD)	164.54 ± 31.27	174.54 ± 32.50	157.82 ± 30.00	0.02*
QRS Duration (ms, Mean ± SD)	94.16 ± 26.89	98.16 ± 28.00	91.74 ± 25.00	0.10
QTc in DII (ms, Mean ± SD)	408.77 ± 35.58	414.00 ± 36.00	405.26 ± 34.00	0.05
ST Elevation after DCT (mm, Mean ± SD)	3.45 ± 1.05	3.75 ± 1.10	3.30 ± 1.00	0.03*
EPS HV Interval (ms, Mean ± SD)	44.45 ± 9.94	46.00 ± 10.00	43.48 ± 9.50	0.10

Abbreviations: EPS: electrophysiological study, HV: HV interval

***Percentages are calculated among patients in each gender or overall group and values are presented as (mean ±SD)**

Table 4. Clinical Cardiac Events of the Study Population with ICD at Follw-up

Parameters	Overall (n=16)	Men (n=12)	Women (n=4)	p-value
Mean Follow-up (months, Mean ± SD)	12 ± 5.3	12 ± 5.3	10 ± 6.4	-
Median Follow-up (months)	7	8	6	-
Patients with High Atrial Rate Episodes on ICD n (%)	9 (56.3%)	6 (50%)	3 (75%)	0.25
Patients with Appropriate ICD Shock n (%)	3 (18.8%)	3 (25%)	0 (0%)	0.30
SCD n (%)	0 (0%)	0 (0%)	0 (0%)	-

Abbreviations: SCD: Sudden cardiac death, **ICD**: implantable cardioverter defibrillator

***Percentages are calculated among patients in each gender or overall group and values are presented as (mean ±SD)**

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-024

The safety of cryobaloon based atrial fibrillation ablation strategy: A single centre retrospective cohort study

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Background and Aim: Cryoballoon based pulmonary vein isolation/ablation technique (CBA) is an established method for the invasive management of atrial fibrillation (AF) worldwide. However there are concerns about its safety in elderly population. Our study aims to investigate the safety of cryoablation in elderly population.

Methods: Procedural and clinical data of 134 patients who underwent CBA of AF from 2022 to 2023 in our center were retrospectively reviewed. Patients with missing data were excluded. The number of isolated pulmonary veins (PV), time to isolation (TTI), number of cryo-applications, the lowest temperature reached, application time for each PVs and total fluoroscopy times were obtained. Complications during the peri-procedural period were noted. Data of the patients before and after 65 years of age were statistically compared.

Results: Clinical and baseline characteristics are shown in Table 1-3. Patients ≥65 years of age had significantly higher coronary artery disease [(25.8% vs. 46.3%) p=0.018], chronic kidney disease [4.8% vs. 16.4%) p=0.047] and antiplatelet use [(1.6% vs. 11.9%) p=0.034]. However, there were no statistically significant difference between the elderly and the remaining population regarding total number of CBA applications, the lowest temperature reached, total ablation time, total fluoroscopy time or phrenic (16.1% vs. 0%, p=0.089)/mechanical (4.8% vs. 5.9%, p=0.78) complication rates.

Conclusions: CBA of AF can be performed without safety concerns in elderly population.

Table 1.

	Baseline Patient Characteristics				
	<65 years		>65 years		
	n	%	n	%	p
Male	46	74,2	39	58,2	0,065
DM	7	11,3	23	34,3	0,003
HT	43	69,4	48	71,6	0,848
CAD	16	25,8	31	46,3	0,018
HL	30	48,4	41	61,2	0,16
Obesity	13	21	16	23,9	0,833
COPD	0	0	3	4,5	0,245
CV disease	7	11,3	8	11,9	0,989
CKD	3	4,8	11	16,4	0,047
Cardiac surgery	1	1,6	5	7,5	0,21

Table 2-3.

	Procedural Characteristics		
	<65 years	>65 years	
	mean±std dev	mean±std dev	p
Total number of applications	4,97±1,568	4,9±1,156	0,768
Total ablation time (Sec)	923,76±272,8	958,39±229,47	0,439
Total fluoroscopy time (Sec)	778,23±288,59	791,78±392,33	0,823
Radiaton dose (mGy)	44,69±22,32	45,25±32,49	0,908

	Overal Complications				
	<65 years		>65 years		
	n	%	n	%	p
Phrenic nerve palsy (Temporary)	10	16,1	4	6	0,089
Phrenic nerve palsy (Permanent)	4	6,5	3	4,5	0,71
Mechanical complications	3	4,8	4	5,9	0,78

Interventional Cardiology / Coronary

OP-026

The relationship between serum growth differantiation factor 15 levels and coronary collateral circulation in stable coronary artery patients with chronic total occlusion

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Background and Aim: Collateral vessels are channels that provide anastomosis connecting epicardial coronary arteries. CCC provides an alternative resource to myocardium compromised by occlusive coronary artery disease. It has positive effects in reducing the frequency of myocardial infarction, protecting left ventricular function by limiting the infarct area and reducing coronary mortality. GDF-15 levels increase rapidly in response to cardiovascular damage such as pressure load, heart failure, ischemia/reperfusion and atherosclerosis. In our study, we aimed to compare the serum GDF-15 levels of patients with stable CAD and patients without CAD on angiographic imaging and to investigate the relationship between well or poorly developed collaterals according to Rentrop coronary collateral circulation classification and serum GDF-15 levels in patients with CTO.

Methods: A total of 140 age- and gender-matched consecutive patients who were found to have total occlusion (n=70) and no significant stenosis in epicardial coronary arteries (n=70) who underwent coronary angiography due to stabil coronary artery disease between June 2021 and June 2022 were included in the study. GDF-15 levels were measured using enzyme-linked immunosorbent assay. Coronary collateral circulation was graded using Rentrop's classification of collateral filling. we firstly aimed to compare stable coronary artery patients with CTO with the control group, then to classify the level of coronary collateral circulation as poor and well-developed collateral circulation according to Rentrop coronary collateral circulation classification and to compare GDF-15 levels between these groups.

Results: GDF-15 levels were 343.24 (89.11-3155.92) ng/L in the CTO group and 310.41 (48.07-631.12) ng/L in the control group and the difference between the two groups was clinically significant high (p=0.001). In the binary and multivariate regression analysis, GDF-15 levels were found to be independent predictors (p=0.014). GDF-15 levels were measured as 426.724 (278.03-3155.92) ng/L in the group with good collateral development and 262.390 (89.115-784.657) ng/L in

the group with poor collateral development. This difference was considered clinically significant (p<0.001). GDF-15 levels were found to be an independent predictor of coronary collateral development (p=0.015).

Conclusions: In conclusion, serum GDF-15 levels were found to be higher in patients with CTO compared with the control group. GDF-15 levels were also found to be higher in the well-developed collateral group compared to the poorly developed collateral group. In this study, we thought that GDF-15 may be effective in the evaluation of collateral circulation. It was thought that patient-based evaluation according to GDF-15 levels could shed light on the medical or invasive evaluation of CTO lesions.

Table 1. Basic demographic, clinical, laboratory parameters of the study population

	Chronic Total Occlusion Group (n:70)	Control Group (n: 70)	p value
Clinical parameters			
Gender, male, n (%)	57(81.4)	55(78.6)	0.853
Age, year	62.96±8.79	61.87±9.64	0.487
BMI, kg/m^2	28.53±4.06	27.75±3.46	0.220
Hypertension, n (%)	45(64.3)	37(52.9)	0.230
Diyabetes Mellitus, n (%)	23(32.9)	22(31.4)	1
Hyperlipidemia, n (%)	42(60)	24(34.3)	0.004*
Smoking, n (%)	48(68.6)	33(47.1)	0.009*
Family history (related to CAD), n (%)	22(31.4)	25(35.7)	0.721
Medical drug use			
The use of antiplatelet, n (%)	63(90)	40(57.1)	<0.001*
Use of statins, n (%)	53(75.7)	19(27.1)	<0.001*
The use of ACEi/ARB, n (%)	37(52.9)	29(41.4)	0.236
Use of beta blockers, n (%)	46(65.7)	30(42.9)	0.011*
The use of calcium channel blockers, n (%)	20(28.6)	8(11.4)	0.027*
Laboratory parameters			
Wbc (×10^3/L)	8.30(4.00-15.41)	8.24(3.22-25.90)	0.331
Hemoglobin count (g/dl)	14.15±1.73	13.91±1.97	0.457
Number of platelet (×10^3/L)	234.50(121.00-437.00)	246.00(107.00-422.00)	0.952
GFR (ml/dk/1.73m^2)	84.32(42.80-120.170)	93.61(51.75-138.040)	0.002*
LDL (mg/dl)	88(19-200)	109.30(14-204)	0.114
HDL (mg/dl)	39(22-64)	46.50(25-79)	0.001*
Total cholesterol (mg/dl)	161.50(88-289)	190.50(84-318)	0.018*
Triglycerides (mg/dl)	161(56-354)	137(39-776)	0.655
CRP (mg/l)	25.15(4.12-0.30)	3(2-45)	0.450
TSH (µIU/ml)	1.40(0.01-9.81)	1.72(0.02-15.00)	0.012*
Blood pressure, systolic (mmhg)	117.57±11.34	115.98±13.06	0.458
Blood pressure, diastolic (mmhg)	73.21±7.37	72.30±7.10	0.470
GDF-15 (ng/l)	343.24(89.11-3155.92)	310.41(48.07-631.12)	0.001*

*p<0.05 was statistically significant BMI: body mass index; CAD: coronary artery disease; ACEi/ARB: angiotensin-converting enzyme inhibitor/angiotensin receptor blockers; WBC: white blood cell; GFR: Glomerular filtration rate; LDL: Low-density cholesterol; HDL: high-density cholesterol; CRP: C reactive protein; TSH: thyroid-stimulating hormone; GDF-15: Serum Growth Differentiation Factor 15.

Table 3. Dual logistic regression analyses to determine the independent predictors of chronic total occlusion according to the Rentrop Collateral classification

	Univariate		Multivariate	
	OR (95% CI)	p değeri	OR (95% CI)	p value
Family History	0.135-1.069	0.067	0.088 (0.011-0.714)	0.023*
GDF-15 (ng/l)	1.003-1.012	0.003*	1.007 (1.001-1.012)	0.015*
Number of platelet (×10^3/L)	0.987-1.002	0.187	0.991 (0.978-1.003)	0.149
Duration of angina pectoris, week	1.152 (1.051-1.262)	0.002	1.178 (1.051-1.322)	0.006*

*p<0.05 was statistically significant.

Table 2. Basic demographic and clinical characteristics of the patient population according to the Rentrop coronary collateral classification

	Poor Collateral n:30	Good Collaterals n:40	p value
Gender, male, n (%)	24(80)	33(82.5)	0.790
Age, year	61.6±9.41	63.98±8.26	0.266
BMI, kg/m^2	28.38±4.27	28.65±3.95	0.793
Hypertension, n (%)	17(56.7)	28(70)	0.249
Diyabetes Mellitus, n (%)	12(40)	11(27.5)	0.271
Hyperlipidemia, n (%)	20(66.7)	22(55)	0.324
Smoking, n (%)	22(73.3)	26(65)	0.176
Family history (related to CAD), n (%)	13(43.3)	9(22.5)	0.063
Duration of angina pectoris, week	12 (4-36)	22 (6-104)	<0.001*
Syntax score	21.75(10-44.5)	20.75(3-42)	0.789
Number of patient vessels 1, n (%) 2, n (%) 3, n (%)	 12(40) 8(26.7) 10(33.3)	 19(47.5) 13(32.5) 8(20)	0.450
CTO vessels LAD, n (%) CX, n (%) RCA, n (%)	 12(40) 6(20) 12(40)	 11(27.5) 7(17.5) 22(55)	0.435
The use of antiplatelet, n (%)	26(86.7)	37(92.5)	0.421
Use of statins, n (%)	24(80)	29(72.5)	0.469
The use of ACEi/ARB, n (%)	15(50)	22(55)	0.678
Use of beta blockers, n (%)	17(56.7)	29(72.5)	0.167
The use of calcium channel blockers, n (%)	7(23.3)	13(32.5)	0.634
Wbc (×10^3/L)	8.86±2.53	8.73±2.47	0.826
Hemoglobin count (g/dl)	14.02±1.27	14.24±2.02	0.591
Number of platelet (×10^3/L)	261.46±63.38	240.725±64.90	0.186
Neutrophils (x10^3/L)	4.67(3.1-12.3)	5.2(2-12.4)	0.840
Lymphocyte (x10^3/L)	2.25±0.98	2.22±0.76	0.888
GFR (ml/dk/1.73m^2)	80.51±19.98	85.15±13.05	0.246
LDL (mg/dl)	91.05±34.49	96.68±44.89	0.569
HDL (mg/dl)	41.35±9.75	40.75±7.66	0.776
Total cholesterol (mg/dl)	165.41±42.35	169.08±51.27	0.751
Triglycerides (mg/dl)	165.21±72.54	158.48±60.46	0.673
CRP (mg/l)	22.73(1-79.1)	27.45(0.30-136)	0.711
TSH (µIU/ml)	1.65±1.65	1.48±1.05	0.690
Blood pressure, systolic (mmhg)	116.67±9.58	118.25±12.58	0.567
Blood pressure, diastolic (mmhg)	72.67±7.39	73.63±7.42	0.594
GDF-15 (ng/l)	262.390(89.115-784.657)	426.724(278.03-3155.92)	<0.001*

*p<0.05 was statistically significant BMI: body mass index; CAD: coronary artery disease; ACEi/ARB: angiotensin-converting enzyme inhibitor/angiotensin receptor blockers; WBC: white blood cell; GFR: Glomerular filtration rate; LDL: Low-density cholesterol; HDL: high-density cholesterol; CRP: C reactive protein; TSH: thyroid-stimulating hormone; GDF-15: Serum Growth Differentiation Factor 15.

Cardiovascular Nursing / Technician

OP-027

Effect of Killip class on intensive care anxiety disorder in acute myocardial infarction

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Background and Aim: During coronary intensive care follow-up, environmental factors as well as the clinical condition of patients can affect their psychosocial health status. Especially hemodynamic and respiratory problems can increase the development of intensive care anxiety and depression. Bad experiences in intensive care may also negatively affect the post-intensive care prognosis of the primary disease. Early detection and prevention of causes

that will increase the development of anxiety and depression may improve this prognosis. In this study, we investigated the effect of the Killip class on anxiety in patients admitted to intensive care due to acute myocardial infarction (AMI).

Methods: Killip classes during hospitalization of 110 patients admitted to coronary intensive care unit due to AMI were recorded. Interventions and treatments applied to patients during the intensive care unit were recorded. After the intensive care follow-ups were completed, the patients were administered a "Hospital Anxiety Depression Scale (HADS)" survey. In HADS, the lower limit of anxiety was accepted as 10 points and the lower limit of depression was accepted as 7 points. **Results:** Among 110 patients 76 (69%) of those were men. Mean age of these patients was 61.9 ± 12.5 (min: 31, max: 89) years. While 77 (70%) of the patients were Killip class 1, the others were Killip 2, 3 and 4. The mean anxiety score was 7.85 ± 4.9 and the mean depression score was 7.58 ± 4.92. Anxiety was detected in 37 (34%) of the patients and depression was detected in 60 (55%). Patients having a class other than Killip 1 and non-invasive mechanical ventilation (NIV) application significantly increased intensive care anxiety (p=0.009 and 0.043). It was determined that NIV application also significantly increased the development of depression, but Killip class did not affect this situation (p=0.037 and p=0.095) (Table 1). Laboratory data of the patients are presented in Table 2. In the univariate and multivariate logistic regression analysis, it was determined that being outside Killip class 1 was an independent predictor for intensive care anxiety (OR: 2.857, 95% CI: 1.200, 6.798; p=0.018) (Table 3).

Conclusions: Environmental factors such as noisy and intense light environment, distance from family, insomnia and inactivity, as well as clinical conditions such as pain, shortness of breath and hemodynamic disorders are effective in the development of intensive care anxiety and depression. In previous studies, similar to our study results, the rate of anxiety disorder was found to be 12-44% and the rate of depression was 10-47% in patients staying in the intensive care unit. Killip classification is a fast, effective and safe scoring system that evaluates respiratory and hemodynamic parameters in AMI patients. As the killip class increases, in-hospital mortality increases dramatically. Additionally, in our study, a score other than Killip 1 was found to be an independent predictor for the development of intensive care anxiety disorder.

Table 1. Demographic and clinical characteristics of patients according to groups

Variables n (%)		Anxiety Score score<10 n= 73 (66%)	10<score<21 n=37 (34%)	P value	Depression Score score<7 n=50 (45%)	7<score<21 n=60 (55%)	P value
Age, years (mean ±SD)		63.08± 12.3	59.57±12.8	0.166	60.26±11	63.3±13.7	0.212
Male gender		51 (69.9)	25 (67.6)	0.806	37 (74)	39 (65)	0.309
Hypertension		38 (52.1)	21 (56.8)	0.640	25 (50)	34 (56.7)	0.485
Diabetes mellitus		32 (43.8)	14 (37.8)	0.547	19 (38)	27 (45)	0.459
Hyperlipidemia		40 (54.8)	20 (54.1)	0.941	30 (60)	30 (50)	0.294
CKD		4 (5.5)	5 (13.5)	0.146	1 (2)	8 (13.3)	**0.031**
CVD		2 (2.7)	0	0.549	0	2 (3.3)	0.50
COPD		8 (11)	4 (10.8)	0.981	4 (8)	8(13.3)	0.372
Smoking		32 (43.8)	23 (62.2)	0.069	24 (48)	31 (51.7)	0.702
Alcohol		13 (17.8)	6 (16.2)	0.835	13 (26)	6 (10)	**0.027**
Marital status-married		48 (65.8)	26 (70.3)	0.633	32 (64)	42 (70)	0.504
Educational background	0	5 (6.8)	2 (5.4)	0.883	3 (6)	4 (6.7)	0.172
	1	42 (57.5)	24 (64.9)		25 (50)	41 (68.3)	
	2	15 (20.5)	7 (18.9)		12 (24)	10 (16.7)	
	3	11 (15.1)	4 (10.8)		10 (20)	5 (8.3)	
CAG history		24 (32.9)	18 (48.6)	0.108	16 (32)	26 (43.3)	0.223
Previous hospitalization		53 (72.6)	28 (75.7)	0.730	35 (70)	46 (76.7)	0.429
Previous intensive care unit history		29 (39.7)	16 (43.2)	0.723	20 (40)	25 (41.7)	0.859
NIV		1 (1.4)	4 (10.8)	**0.043**	0	5 (8.3)	**0.037**
Mechanical ventilator		0	2 (5.4)	0.111	0	2 (3.3)	0.50
CPR		0	1 (2.7)	0.336	0	1 (1.7)	1
Use of inotropic agents		2 (2.7)	1 (2.7)	1	0	3 (5)	0.249
Intensive care period		2.06±1.5	2.64±2.5	0.133	1.84±0.9	2.61±2.4	**0.033**
Type of MI	NONSTE	45 (61.6)	24 (64.9)	0.268	34 (68)	35 (58.3)	0.761
	Inferior	17 (23.3)	4 (10.8)		8 (16)	13 (21.7)	
	Anterior	10 (13.7)	7 (18.9)		7 (14)	10 (16.7)	
	Others	1 (1.4)	2 (5.4)		1 (2)	2 (3.3)	
Type of treatment	Medical	13 (17.8)	4 (11.1)	0.132	9 (18)	8 (13.6)	0.506
	PCI	56 (76.7)	26 (72.2)		38 (76)	44 (74.6)	
	Surgery	4 (5.5)	6 (16.7)		3 (6)	7 (11.9)	
Type of Access	Radial	24 (32.9)	14 (38.9)	0.536	18 (36)	20 (33.9)	0.819
	Femoral	49 (67.1)	22 (61.1)		32 (64)	39 (66.1)	
Killip classification	1	57 (78.1)	20 (54.1)	**0.009**	39 (78)	38 (63.3)	0.095
	2,3,4	16 (21.9)	17 (45.9)		11 (22)	22 (36.7)	

Abbreviations: SD, standard deviation; n, number of patients; CAG, coronary angiography; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; CPR, Cardiopulmonary resuscitation; CVD, cerebrovascular disease; MI, myocardial infarction; NIV, noninvasive mechanical ventilator; PCI, percutaneous coronary intervention. (Educational background; 0: illiterate, 1: primary education, 2: high school, 3: university)

Table 2. Laboratory characteristics of patients according to groups

Variables (mean ± SD)	Anxiety Score score<10 n= 73 (66%)	10<score<21 n=37 (34%)	P value	Depression Score score<7 n=50 (45%)	7<score<21 n=60 (55%)	P value
FBG, mg/dl	153.16±64.6	138.81±52.5	0.245	149.2±64.5	147.61±58.3	0.893
Urea, mg/dl	38.62±17.6	45.81±30.7	0.122	34.87±11.8	46.18±28.3	**0.010**
Creatinine, mg/dl	1.03±0.8	1.48±1.4	0.079	1.03±0.9	1.31±1.2	0.171
Sodium, mEq/L	139.65±3.9	138.4±3.6	0.109	139.28±3.4	139.18±4.2	0.900
Potassium, mg/dl	4.36±0.6	4.49±0.9	0.349	4.32±0.5	4.47±0.8	0.280
Calcium, mg/dl	9.1±0.6	8.97±0.5	0.261	9.19±0.7	8.94±0.4	**0.024**
AST, IU/L	51.8±60	39.05±35.1	0.236	54.9±68.3	41.36±35.5	0.185
ALT, IU/L	28.61±25.9	24±16.6	0.327	30.46±29.4	24.23±16.1	0.162
Total cholesterol, mg/dl	196.21±49.3	197.41±56.5	0.910	207.27±52	187.8±49.9	0.053
HDL-cholesterol, mg/dl	38.61±10.2	37.14±12.1	0.511	39.37±10.4	37.07±11.1	0.279
LDL-cholesterol, mg/dL	123.53±57.7	121.51±44.1	0.856	133.45±65.8	114.11±38.7	0.065
Triglycerides, mg/dl	193.1±166.7	171.9±80.2	0.475	187±111.3	184.9±165.9	0.943
WBC, × 10^9 /L	10.29±3.3	10.8±3.4	0.452	10.11±2.8	10.76±3.7	0.318
Hemoglobin, g/Dl	13.55±2.1	12.94±2.8	0.207	14.08±1.7	12.74±2.7	**0.003**
Hematocrit, %	42.9±5.7	41.21±8.3	0.217	44.41±4.3	40.6±7.8	**0.003**
Platelet count, × 10^9 /L	244.15±75.7	279.5±95.1	0.054	256.14±74.9	255.96±91.5	0.991
Hs-cTnT	588.13±1124	801±1491	0.404	376.8±800.8	892.5±1501	**0.033**
HbA1C, %	7.21±2.2	6.45±1.5	0.092	7.13±2.3	6.79±1.8	0.422
TSH, mU / L	2.2±2.4	1.74±1.9	0.304	2.48±2.7	1.68±1.8	0.071
T4, mU / L	1.29±0.6	1.14±0.3	0.228	1.21±0.4	1.27±0.6	0.649
LVEF, %	52.93±8.4	50.45±10.8	0.218	52.39±8.9	51.8±9.8	0.758

Abbreviations: SD, standard deviation; n, number of patients; ALT, alanine aminotransferase; AST, aspartate transaminase; FBG, Fasting blood glucose; HDL, high density lipoprotein; Hs-cTnT, high-sensitive cardiac troponin T; LDL, low density lipoprotein; LVEF,left ventricle ejection fraction; TSH, tiroid stimülan hormon.

Table 3. Effects of variables on the development of anxiety disorder in univariate and multivariate logistic regression analysis

Variables	Univariate Logistic Regression		Multivariate Logistic Regression	
	OR (95% CI)	P Value	OR (95% CI)	P Value
Killip classification (2,3,4)	3.028(1.292- 7.097)	**0.011**	2.857(1.200- 6.798)	**0.018**
Smoking	2.105(0.937- 4.729)	0.072		
NIV	8.727(0.939- 81.139)	0.057		
Creatinine	1.536(0.962-2.453)	0.072		
Platelet count	1.005(1.000-1.010)	**0.042**	1.005(1.000- 1.010)	0.069
HbA1C	0.799(0.610- 1.046)	0.102		

Heart Failure

OP-029

Clinical trajectories in non-ischemic cardiomyopathy: Insights into predictors of myocardial recovery and heart failure progression

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Background and Aim: Heart failure (HF) patients exhibit diverse clinical trajectories, broadly categorized into stabilization, deterioration, and recovery. This study aims to identify predictors of myocardial recovery in non-ischemic cardiomyopathy (NICM) patients, emphasizing the role of biomarkers, imaging parameters, and clinical features.

Methods: An observational cohort study included 439 patients with NICM who underwent cardiac magnetic resonance imaging (CMR) between January 2008 and March 2024. Clinical, echocardiographic, and CMR data were analyzed to identify predictors of myocardial recovery. Patients were stratified into three groups: those who reached the primary endpoint (all-cause mortality, left ventricular assist device implantation, or heart transplantation), those who maintained stability under medical therapy, and those who exhibited clinical and myocardial recovery. Heart failure improved ejection fraction (myocardial recovery) patients defined as a documented history of LVEF ≤40, ≥10% increase of LVEF, and improved LVEF at least 40%.

Results: The study cohort had a mean age of 42 ± 14.3 years, with a predominance of male patients (63.6%). Myocardial recovery was observed in 21.2% (n=93) of patients, with 46.2% (n=43) achieving an LVEF >50%. The primary predictors of myocardial recovery were NYHA functional class (FC) and left ventricular end-systolic volume (LVESV). Patients classified as NYHA I-II with an LVESV <148 mL had an 89.8% likelihood of recovery, whereas those with NYHA III-IV and LVESV >148 mL had minimal recovery potential. Elevated NT-proBNP levels were significantly correlated with adverse outcomes. Despite the high utilization of evidence-based HF medications, optimal dosing was achieved in a minority of patients. The decision tree model demonstrated an overall predictive accuracy of 83.9%. Notably, recovery occurred in various clinical contexts, including post-peripartum CM, atrial fibrillation ablation or cardioversion, kidney replacement therapy and cessation of oncological treatments, underscoring the heterogeneous nature of recovery pathways. The median duration of myocardial recovery was 565 days, and following recovery, patients were monitored for a median of 864 days. The initial LVEF recorded

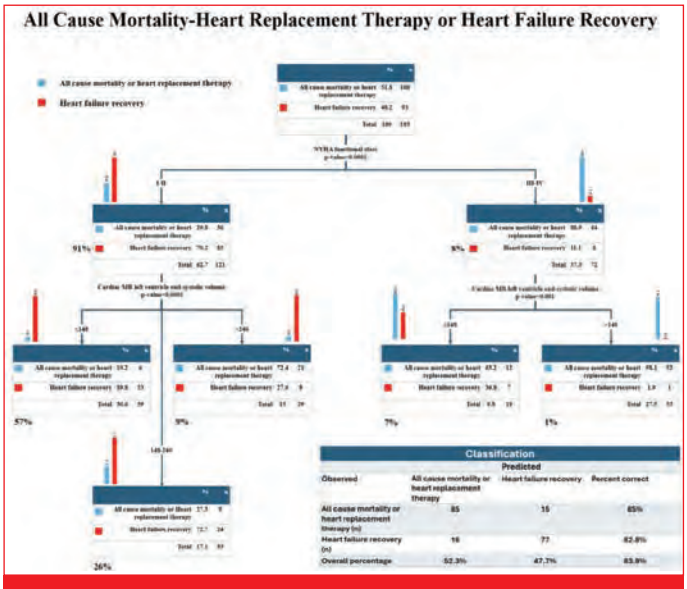

Figure 1. CHAID analysis of main predictors of all cause mortality-heart replacement therapy and myocardial recovery.

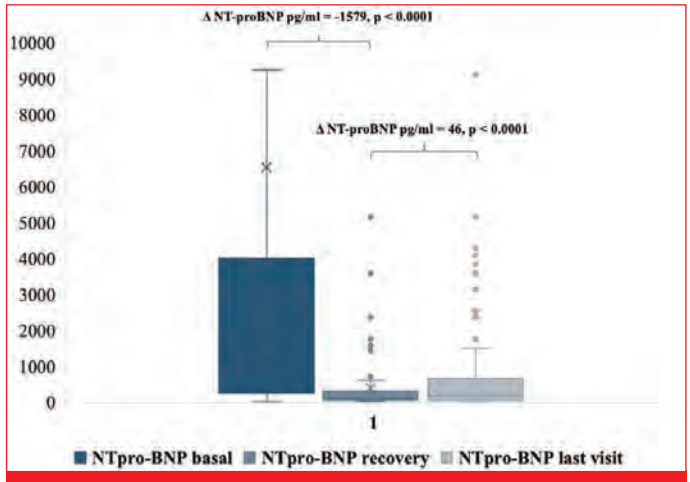

Figure 2. NT-proBNP changes in myocardial recovery patients.

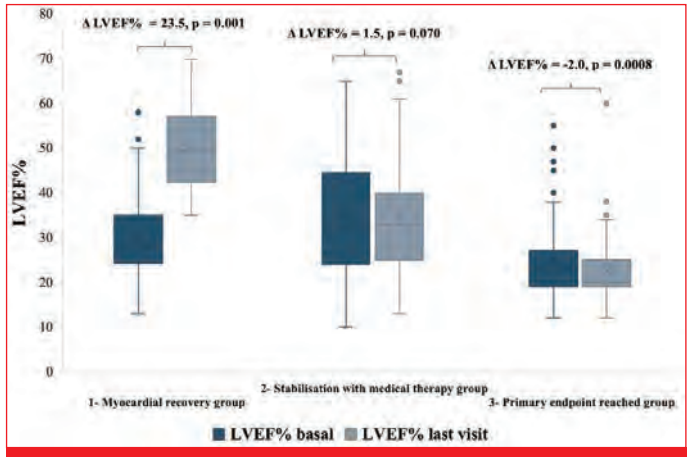

Figure 3. Left ventricle ejection fraction changes comparison in all cause mortality or heart replacement, myocardial recovery and stabilization with medical therapy.

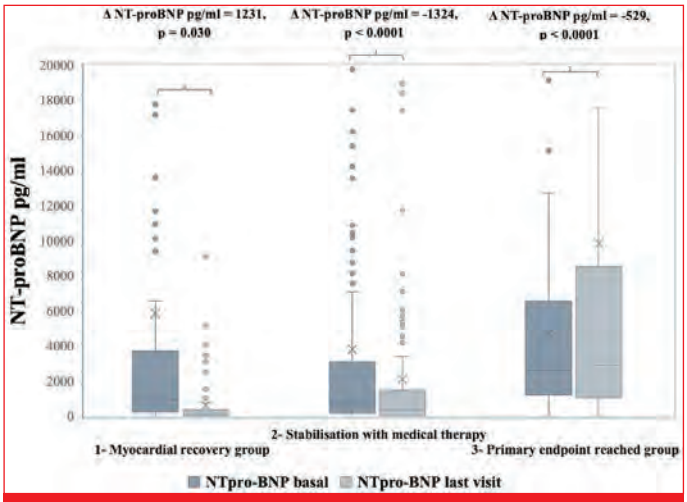

Figure 4. NT-proBNP changes comparison in all cause mortality or heart replacement, myocardial recovery and stabilization with medical therapy.

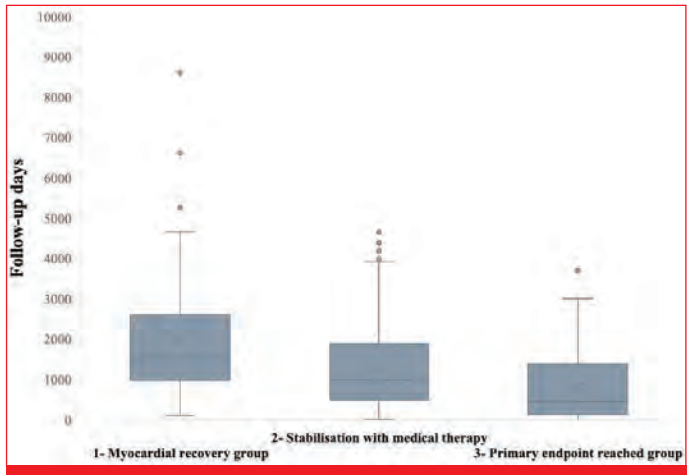

Figure 5. Follow-up times comparison in all cause mortality or heart replacement, myocardial recovery and stabilization with medical therapy.

Table 1. Clinical characteristics of patients and comparison of all-cause mortality or heart replacement therapy, myocardial recovery, and stabilization with medical therapy patients

	Total	All-cause mortality or heart replacement therapy	All-cause mortality or heart replacement therapy	Stabilization with medical therapy	p value
	n=439	n=100	n=93	n=246	
Age years	42.0 ± 14.3	40.3 ± 13.9	41.5 ± 13.9	42.9 ± 14.7	0.29
Male n (%)	279 (63.6)	62 (62)	52 (55.9)	165 (55.9)	0.15
BMI kg/m^2	27.7 ± 5.6	26.8 ± 5.3	26.8 ± 5.4	28.3 ± 5.8	0.022
BSA m^2	1.94 ± 0.23	1.91 ± 0.20	1.88 ± 0.23	1.97 ± 0.24	0.001
NYHA FC III-IV n (%)	93 (22.7)	60 (64.5)	6 (6.6)	27 (12.1)	<0.0001
Arterial hypertension n (%)	100 (22.8)	16 (16)	24 (25.8)	60 (24.4)	0.17
Diabetes mellitus n (%)	82 (18.7)	9 (9)	20 (21.5)	53 (21.5)	0.018
Dyslipidemia n (%)	30 (6.8)	5 (5)	7 (7.5)	18 (7.3)	0.70
Smoking history n (%)	119 (27.1)	28 (28)	29 (31.2)	62 (25.2)	0.52
Cerebrovascular event n (%)	19 (4.3)	4 (4)	7 (7.5)	8 (3.3)	0.22
Lung disease n (%)	-	-	-	-	0.29
COPD n (%)	34 (7.7)	8 (8)	9 (9.8)	17 (6.9)	-
Pulmonary embolism n (%)	8 (1.8)	4 (4)	2 (2.2)	2 (0.8)	-
Connective tissue disease n (%)	9 (2.1)	1 (1)	5 (5.4)	3 (1.2)	0.038
Renal disease n (%)	23 (5.2)	1 (1)	8 (8.6)	14 (5.7)	0.054
Malignancy n (%)	18 (4.1)	7 (7.1)	3 (3.2)	8 (3.3)	0.24
AF n (%)	61 (14.1)	12 (12.4)	19 (20.4)	30 (12.4)	0.14
Intracardiac device	-	-	-	-	-
ICD n (%)	100 (22.8)	34 (34)	12 (12.9)	54 (22)	0.002
CRT n (%)	21 (4.8)	4 (4)	4 (4.3)	13 (5.3)	0.85
Family History of heart failure n (%)	60 (14)	18 (18.4)	10 (11)	32 (13.3)	0.31
SBP mmHg	116 ± 16	106 ± 14	119 ± 15	119 ± 15	<0.0001
DBP mmHg	73 ± 11	66 ± 9	74 ± 11	75 ± 11	<0.0001
NTpro-BNP pg/mL	1166 (313-3621)	2624 (1281-6559)	998 (221-3604)	770 (221-3101)	<0.0001
Follow-up time Days (IQR)	1423 (618-2463)	824 (193-2217)	2055 (1088-2931)	1395 (663-2401)	<0.0001

* Values are mean ± SD or n (%) p<0.05. ACEi: Angiotensin converting enzyme inhibitor; AF: Atrial fibrillation; ARB: Angiotensin receptor blocker; BMI: Body mass index; BSA: Body surface area; COPD: Chronic obstructive pulmonary disease; CRT: Cardiac resynchronization therapy; ICD: Implanted cardioverter-defibrillator; DBP: Diastolic blood pressure; IQR: Interquartile range; LVEF: Left ventricular ejection fraction; New York Heart Association Functional Class: NYHA FC; SBP: Systolic blood pressure.

was 26.1 ± 7.2%, which improved to 49.5 ± 7.9%, reflecting a mean increase of 23.3 ± 8.8% (p<0.0001). NT-proBNP levels normalized after recovery, decreasing from 1338 (IQR: 338-4250) pg/mL to 116 (IQR: 63-337) pg/mL (p<0.0001). However, during follow-up, levels increased from 116 (IQR: 63-337) pg/mL to 170 (IQR: 65-671) pg/mL (p<0.0001).

Conclusions: NYHA FC and LVESV are critical predictors of myocardial recovery in NICM patients. Early and aggres-

Table 2. Medical therapy of patients and comparison of all-cause mortality or heart replacement therapy, myocardial recovery, and stabilization with medical therapy patients

	Total	All-cause mortality or heart replacement therapy	Myocardial recovery	Stabilization with medical therapy	p value
	n=439	n=100	n=93	n=246	
ACEi or ARB n (%)	323 (73.6)	74 (74)	69 (74.2)	180 (73.2)	0.97
ACEi or ARB dose	-	-	-	-	0.043
Exact dose n (%)	85 (20.2)	12 (12.9)	24 (27)	49 (20.5)	-
Half dose n (%)	113 (26.8)	20 (21.5)	26 (29.2)	67 (28)	-
Quarter dose n (%)	110 (26.1)	35 (37.6)	16 (18)	59 (24.7)	-
Beta-blocker n (%)	414 (94.3)	97 (97)	90 (96.8)	227 (92.3)	0.11
Beta-blocker dose	-	-	-	-	0.57
Exact dose n (%)	30 (7.0)	5 (5.3)	6 (6.5)	19 (7.9)	-
Half dose n (%)	125 (29.3)	29 (30.5)	29 (31.5)	67 (27.9)	-
Quarter dose n (%)	248 (58.1)	58 (61.1)	54 (58.7)	136 (56.7)	-
Aldosterone antagonist n (%)	323 (73.6)	89 (89)	61 (65.6)	173 (70.3)	<0.0001
Aldosterone antagonist dose	-	-	-	-	0.001
Exact dose n (%)	11 (2.5)	6 (6.1)	1 (1.1)	4 (1.6)	-
Half dose n (%)	303 (69.8)	81 (81.8)	57 (62.6)	165 (67.6)	-
Quarter dose n (%)	4 (0.9)	1 (1)	1 (1.1)	2 (0.8)	-
ARNI n (%)	63 (14.4)	11 (11)	15 (16.1)	37 (15)	0.53
ARNI dose	-	-	-	-	0.11
Exact dose n (%)	16 (3.7)	7 (7.1)	2 (2.2)	7 (2.9)	-
Half dose n (%)	17 (3.9)	1 (1)	6 (6.5)	10 (4.1)	-
Quarter dose n (%)	26 (6)	3 (3)	5 (5.4)	18 (7.4)	-
SGLT2-i n (%)	91 (20.7)	12 (12)	19 (20)	60 (24.4)	0.036
Anticoagulant n (%)	115 (26.2)	25 (25)	36 (38.7)	54 (22)	0.007
Antiplatelet n (%)	178 (40.5)	44 (44)	30 (32.3)	104 (42.3)	0.17
If channel blocker n (%)	89 (20.3)	35 (35)	14 (15.1)	40 (16.3)	<0.0001

* Values are mean ± SD or n (%) p<0.05. ACEi: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker; ARNI: Angiotensin receptor blocker-neprilysin inhibitor; SGLT2-i: Sodium-glucose transport protein two inhibitors.

Table 3. Echocardiography parameters of patients and comparison of all-cause mortality or heart replacement therapy, myocardial recovery, and stabilization with medical therapy patients

	Total	All-cause mortality or heart replacement therapy	Myocardial recovery	Stabilization with medical therapy	P-value
	n=439	n=100	n=93	n=246	
LVEDd mm	60 ± 9.7	65.3 ± 9.4	57.8 ± 8.5	58.4 ± 9.5	<0.0001
LVESd mm	49.3 ± 12.3	57 ± 11.2	47.7 ± 10	46.6 ± 12.3	<0.0001
LAd mm	43.1 ± 7.3	47 ± 6.4	42.6 ± 6.4	43.4 ± 7.5	<0.0001
LVEF %	31.7 ± 12.7	24.8 ± 8.9	30.1 ± 9.1	34.8 ± 13.4	<0.0001
Mitral regurgitation moderate or severe n (%)	174 (41.8)	60 (60)	35 (38)	79 (35.3)	<0.0001
Aortic regurgitation moderate or severe n (%)	18 (4.3)	4 (4)	4 (4.3)	10 (4.4)	0.98
Tricuspid regurgitation moderate or severe n (%)	99 (22.3)	40 (38.8)	21 (20.4)	42 (18.8)	0.002
TRV m/sec	2.7 ± 0.75	2.85 ± 0.45	2.79 ± 0.58	2.62 ± 0.88	0.08
TDI RVsm m/sec	10.7 ± 2.7	9.4 ± 2.5	11 ± 2.5	11.1 ± 2.7	<0.0001
TAPSE mm	18.7 ± 5.2	16.4 ± 4.7	18.9 ± 5	19.6 ± 5.2	<0.0001
SPAP mmHg	37.9 ± 15.8	41.2 ± 13.8	41.2 ± 13.5	35.1 ± 16.3	0.003

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

sive management is essential for patients with favorable prognostic indicators. The findings underscore the need for personalized treatment strategies and highlight the importance of comprehensive clinical and imaging assessments in HF management. Further research is warranted to validate these predictors and refine therapeutic approaches.

Table 4. Cardiac magnetic resonance imaging parameters of patients and comparison all-cause mortality or heart replacement therapy, myocardial recovery and stabilization with medical therapy patients

	Total	All-cause mortality or heart replacement therapy	Myocardial recovery	Stabilization with medical therapy	p value
	n=439	n=100	n=93	n=246	
IVS mm	9.8 ± 2	8.9 ± 2	9.6 ± 2.3	9.6 ± 2.1	0.002
LVEDd mm	61.5 ± 10.5	67.4 ± 10.4	59.7 ± 8.6	59.8 ± 10.3	<0.0001
LVESd mm	52 ± 12.4	59.8 ± 11.5	50 ± 10.3	49.7 ± 12.1	<0.0001
LAd mm	46.6 ± 9.2	52.3 ± 9.7	44.5 ± 7.8	45 ± 8.5	<0.0001
LVEDV mm	249 ± 100	310 ± 106	220 ± 81	235 ± 94	<0.0001
LVESV mm	178 ± 92	248 ± 97	147 ± 68	162 ± 85	<0.0001
LVEF %	30 ± 11	22 ± 10	32 ± 10	32 ± 11	<0.0001
SV mL	69.3 ± 27.7	64.5 ± 26.6	69.1 ± 32.9	71.5 ± 25.9	0.10
CO L.min	5.3 ± 2.0	5.2 ± 2.1	5.2 ± 2.4	5.4 ± 1.9	0.69
CI L.min/m^2	2.73 ± 1.05	2.76 ± 1.14	2.72 ± 1.27	2.72 ± 0.73	0.95
LVmass gr	163 ± 57	162 ± 50	154 ± 57	168 ± 60	0.26
Late Gadolinium Enhancement	119 (27.1)	30 (30)	19 (20.4)	70 (28.5)	0.25
Subendocardial LGE n (%)	23 (5.2)	7 (7)	1 (1.1)	15 (6.1)	0.12
Midwall LGE n (%)	76 (17.3)	17 (17)	14 (15.1)	45 (18.3)	0.77
Subepicardial LGE n (%)	20 (4.6)	6 (6)	4 (4.3)	10 (4.1)	0.73

* Values are mean ± SD or n (%) p<0.05. CI: Cardiac index; CO: Cardiac output; IVS: Interventricular septum; LAd: Left atrial diameter; LVEDd: Left ventricular end-diastolic diameter; LVEDV: Left ventricular end-diastolic volume; LVESd: Left ventricular end systolic diameter; LVESV: Left ventricular end-systolic volume; LVEF: Left ventricular ejection fraction; LV: Left ventricle; SV: Stroke volume.

Table 5. Clinical characteristics of patients Cox univariate and multivariate analysis of all-cause mortality or heart replacement therapy or myocardial recovery patients

	Univariate analysis	p value	Multivariate analysis	p value
	HR (95% CI)		HR (95% CI)	
Age years	0.99 (0.98-1.01)	0.75		
Male %	1.33 (0.87-2.01)	0.17		
BMI kg/m^2	0.99 (0.96-1.03)	0.88		
BSA m^2	1.59 (0.66-3.86)	0.29		
NYHA FC III-IV	5.53 (3.62-8.42)	<0.0001	4.75 (2.01-11.2)	<0.0001
Arterial hypertension	0.63 (0.37-1.08)	0.09		
Diabetes mellitus n (%)	0.47 (0.24-0.94)	0.035		
Dyslipidemia n (%)	0.77 (0.31-1.91)	0.58		
Smoking history n (%)	0.86 (0.55-1.33)	0.50		
Cerebrovascular event n (%)	0.52 (0.16-1.65)	0.27		
COPD n (%)	0.74 (0.36-1.53)	0.42		
Pulmonary embolism	2.61 (0.95-7.16)	0.06		
Connective tissue disease n (%)	0.27 (0.03-1.96)	0.19		
Renal disease n (%)	0.15 (0.02-1.01)	0.06		
AF n (%)	0.70 (0.38-1.28)	0.24		
ICD n (%)	1.51 (0.99-2.30)	0.051		
CRT n (%)	0.70 (0.25-1.91)	0.49		
Family history of heart failure n (%)	1.42 (0.85-2.38)	0.17		
SBP mmHg	0.96 (0.94-0.97)	<0.0001		
DBP mmHg	0.95 (0.93-0.97)	<0.0001		
Log-NT-proBNP	1.96 (1.50-2.56)	<0.0001		

* Values are mean ± SD or n (%) p<0.05. ACEi: Angiotensin converting enzyme inhibitor; AF: Atrial fibrillation; ARB: Angiotensin receptor blocker; BMI: Body mass index; BSA: Body surface area; COPD: Chronic obstructive pulmonary disease; CRT: Cardiac resynchronization therapy; ICD: Implanted cardioverter-defibrillator; DBP: Diastolic blood pressure; IQR: Interquartile range; LVEF: Left ventricular ejection fraction; New York Heart Association Functional Class: NYHA FC; SBP: Systolic blood pressure.

Table 6. Medical therapies of patients Cox-regression univariate and multivariate analysis of all-cause mortality or heart replacement therapy or myocardial recovery patients

	Univariate analysis	p value	Multivariate analysis	p value
	HR (95% CI)		HR (95% CI)	
ACEi or ARB n (%)	0.86 (0.55-1.35)	0.51		
Exact dose n (%)	0.56 (0.28-1.11)	0.09		
Half dose n (%)	0.67 (0.37-1.2)	0.18		
Quarter dose n (%)	1.24 (0.74-2.0)	0.40		
Beta-blocker n (%)	1.18 (0.37-3.78)	0.77		
Exact dose n (%)	1.27 (0.30-5.42)	0.74		
Half dose n (%)	1.03 (0.31-3.42)	0.95		
Quarter dose n (%)	1.19 (0.37-3.86)	0.76		
Aldosterone antagonist n (%)	2.54 (1.35-4.76)	0.004		
Exact dose n (%)	3.59 (1.32-9.74)	0.012		
Half dose n (%)	2.53 (1.35-4.76)	0.004		
Quarter dose n (%)	3.06 (0.39-23.8)	0.28		
Loop diuretic n (%)	4.39 (2.39-8.06)	<0.0001		
Anticoagulant n (%)	0.73 (0.46-1.14)	0.17		
Antiplatelet n (%)	1.21 (0.81-1.8)	0.34		
If channel blocker n (%)	1.97 (1.30-2.98)	0.001		
ARNI n (%)	0.82 (0.43-1.54)	0.54		
Exact dose n (%)	1.41 (0.65-3.07)	0.37		
Half dose n (%)	0.24 (0.03-1.78)	0.16		
Quarter dose n (%)	0.93 (0.29-2.98)	0.91		
SGLT-2i	0.86 (0.47-1.59)	0.64		

* Values are mean ± SD or n (%) p<0.05. ACEi: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker; ARNI: Angiotensin receptor- neprilysin inhibitor; SGLT2-i: Sodium-glucose transport protein two inhibitor.

Table 7. Echocardiographic parameters of patients Cox-regression univariate and multivariate analysis of all-cause mortality or heart replacement therapy or myocardial recovery patients

	Univariate analysis	p value	Multivariate analysis	p value
	HR (95% CI)		HR (95% CI)	
LVEDd mm	1.04 (1.02-1.06)	<0.0001		
LVESd mm	1.04 (1.02-1.05)	<0.0001		
LAd mm	1.08 (1.05-1.11)	<0.0001		
LVEF %	0.93 (0.91-0.96)	<0.0001		
Mitral regurgitation moderate or severe	2.17 (1.44-3.25)	<0.0001		
Aortic regurgitation moderate or severe	1.04 (0.38-2.84)	0.93		
Tricuspid regurgitation moderate or severe	2.29 (1.52-3.45)	<0.0001		
TRV m/sec	1.10 (0.69-1.76)	0.67		
RVsm m/sec	0.81 (0.74-0.89)	<0.0001		
TAPSE mm	0.93 (0.89-0.98)	0.005		
SPAP mmHg	1.002 (0.98-1.01)	0.79		

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

Table 8. Cardiac magnetic resonance parameters of patients Cox-regression univariate and multivariate analysis of all-cause mortality or heart replacement therapy or myocardial recovery patients

	Univariate analysis	p value	Multivariate analysis	p value
	HR (95% CI)		HR (95% CI)	
IVS mm	0.94 (0.84-1.03)	0.22		
LVEDd mm	1.04 (1.02-1.05)	<0.0001		
LVESd mm	1.04 (1.02-1.06)	<0.0001		
LAd mm	1.04 (1.02-1.05)	<0.0001		
LVEF %	0.94 (0.92-0.96)	<0.0001		
LVEDV mL	1.005 (1.003-1.006)	<0.0001		
LVESV mL	1.006 (1.004-1.008)	<0.0001	1.004 (1.000-1.007)	0.028
SV mL	0.99 (0.98-1.003)	0.30		
CO L.min	0.99 (0.91-1.08)	0.98		
CI L.min/m^2	0.98 (0.84-1.16)	0.89		
LVmass gr	1.005 (1.001-1.010)	0.019		
Late gadolinium enhancement	1.74 (1.13-2.69)	0.012		
Subendocardial LGE	3.38 (1.55-7.34)	0.002		
Midwall LGE	1.42 (0.84-2.40)	0.18		
Subepicardial LGE	1.18 (0.51-2.72)	0.68		

* Values are mean ± SD or n (%) p<0.05. CI: Cardiac index; CO: Cardiac output; IVS: Interventricular septum; LAd: Left atrial diameter; LVEDd: Left ventricular end-diastolic diameter; LVEDV: Left ventricular end-diastolic volume; LVESd: Left ventricular end systolic diameter; LVESV: Left ventricular end-systolic volume; LVEF: Left ventricular ejection fraction; LV: Left ventricle; SV: Stroke volume.

Heart Failure

OP-030

Real-world data on Empaglifozin and Dapaglifozin use in patients with HEART failure: The RED–HEART Study

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Background and Aim: Although sodium-glucose cotransporter-2 inhibitors (SGLT2i) reduce the risk of rehospitalization and cardiovascular death in randomized clinical trials, there is limited real-world data about the current implementation of SGLT2i. We aimed to determine the use of SGLT2i and to identify clinical factors associated with their use in patients with heart failure (HF) in a real-life setting.

Methods: Real-world data on empaglifozin and dapaglifozin use in patients with HEART failure: The RED-HEART study is a multicentre, cross-sectional, and observational study that included outpatients with HF regardless of ejection fraction from 19 cardiology centres between August 2023 and December 2023.

Results: The study population consisted of 1923 patients with HF, predominantly men (61.2%), with a median age of

66 (range: 19-101) years. Overall, 925 patients (48.1%) were receiving SGLT2i. Among study population, 22.1% had HF with preserved ejection fraction, 21.5% had HF with mildly reduced ejection fraction, and 56.4% had HF with reduced ejection fraction, and the use of SGLT2i was 42.0%, 47.9%, and 50.6%, respectively (p=0.012) (Figure 1). Although the use of SGT2i was 76.6% in patients with HF and diabetes, it was only 19.8% in patients with HF and chronic kidney disease, and 26.8% in patients with HF without diabetes and/or chronic kidney disease (p<0.001) (Figure 2). More than two-thirds of patients with HF with reduced ejection fraction were using quadruple (37.6%) or triple (37.7%) medical therapy for HF (Figure 3). Clinical inertia, reimbursement regulations, and cost issues were the main reasons for not using SGLT2i. Higher education level (OR: 1.80; 95% CI: 1.06-3.05; p=0.027), higher household income (OR: 3.46; 95% CI: 1.27-9.42; p=0.015), New York Heart Association functional class IV (OR: 2.72; 95% CI: 1.16-6.35; p=0.021), diabetes (OR: 9.42; 95% CI: 6.72-13.20; p<0.001), and the use of angiotensin receptor-neprilysin inhibitor (OR: 4.09; 95% CI: 2.39-7.01; p<0.001), mineralocorticoid receptor antagonists (OR: 2.02; 95% CI: 1.49-2.75; p<0.001), loop diuretics (OR: 1.62; 95% CI: 1.18-2.22; p=0.003), and thiazide diuretics (OR: 1.72; 95% CI: 1.30-2.29; p<0.001) were independently associated with the use of SGLT2i (Figure 4). Conversely, atrial fibrillation (OR: 0.63; 95% CI: 0.45-0.88; p=0.008), chronic kidney disease (OR: 0.53; 95% CI: 0.37-0.76; p=0.001), the use of non-dihydropyridine calcium channel blockers (OR: 0.68; 95% CI: 0.48-0.98; p=0.042), and the use of statin (OR: 0.67; 95% CI: 0.49-0.91; p= 0.010) were independently associated with the non-use of SGLT2i (Figure 4).

Conclusions: The RED-HEART study provided comprehensive real-world data about the implementation of SGLT2i in patients with HF. These results suggest that there is a need for organized action and close collaboration between the healthcare providers to improve the implementation of SGLT2i, especially in patients with HF preserved ejection fraction and chronic kidney disease.

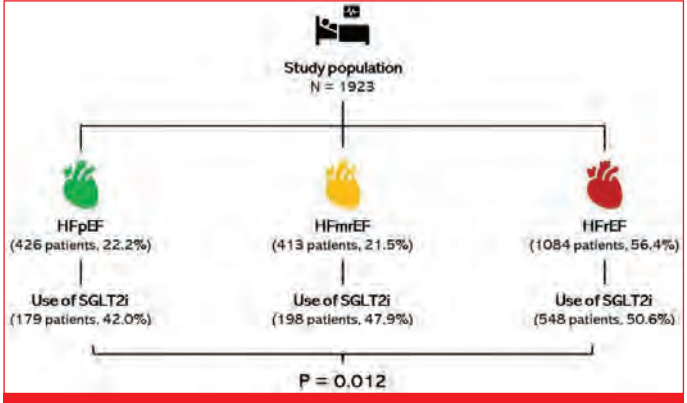

Figure 1. The use of SGLT2i according to the HF types.

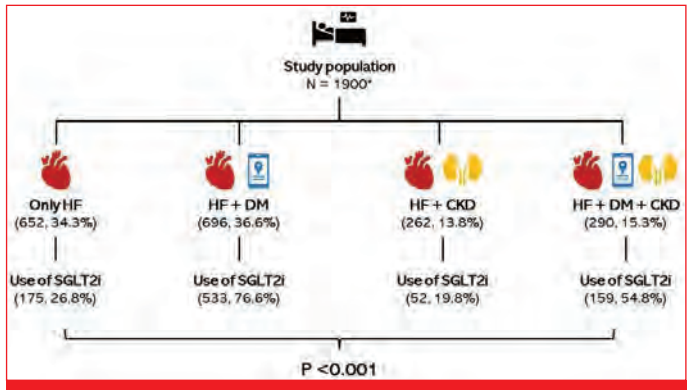

Figure 2. The use of SGLT2i according to the presence or absence of diabetes and chronic kidney disease.

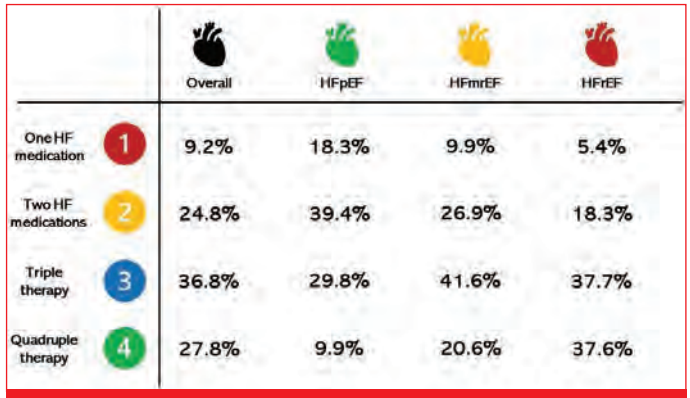

Figure 3. The use HF medications comprising ACEi or ARBs or ARNi, beta-blockers, MRAs, and SGLT2i among study population and different HF types.

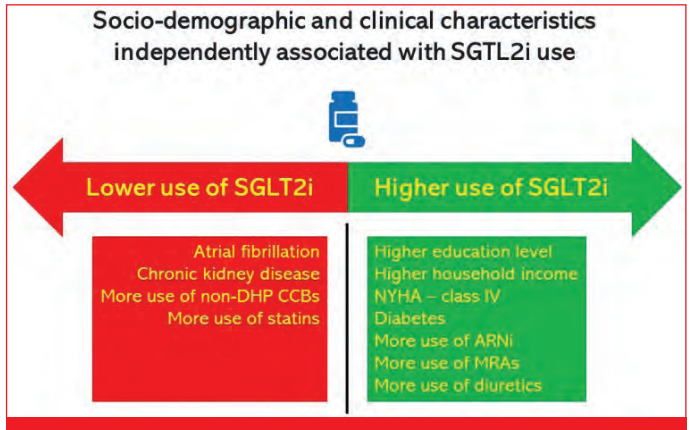

Figure 4. Independent predictors of the use or non-use of SGLT2i among study population regardless of ejection fraction.

Pulmonary Hypertension / Pulmonary Vascular Diseases

OP-031

Whole exome sequencing of Turkish patients with idiopathic pulmonary arterial hypertension and chronic thromboembolic pulmonary hypertension

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Background and Aim: Pulmonary hypertension (PH) is characterized by increased pulmonary vascular load-induced right ventricular hypertrophy and remodeling. Idiopathic PAH (IPAH) and chronic thromboembolic PH (CTEPH) are rare subtypes of PH, and the genetic background is still not completely elucidated. This study aims to identify the genes with the causal genetic variants of the development of CTEPH and IPAH.

Methods: DNA samples were isolated from the peripheral blood samples of the 16 IPAH and 15 CTEPH patients. DNA library preparation was performed by following the Sureselect V6 kit procedure, and whole exome sequencing (WES) was performed on the Illumina NovaSeq X instrument. All obtained variants from Variant Call Format (VCF) file were filtered by a panel of PAH-related genes including 14 WSPH-listed genes (BMPR2, ATP13A3, AQP1, ABCC8, KCNK3, SMAD9, SOX17, CAV1, TBX4, EIF2AK4, KDR, ENG, ACVRL1, and GDF2), and 27 non-WSPH-listed genes (KCNA3, KLF2, SMAD1, SMAD4, KCNA5, BMPR1A, BMPR1B, ABCA3, NOTCH1, NOTCH2, NOTCH3, BMP10, FBLN2, JAG1, JAG2, PTGIS, PDGFD, GGCX, SMAD5, KLK1, SARS2, SERPINE1, SIRT3, THBS1, TNIP2, TopBP1, and TET2).

Results: Among 14 WSPH-listed genes, six distinct rare variants in KDR, GDF2, AQP1, and ACVRL1 genes were found in 6 IPAH patients. These variants were classified as variants of uncertain significance (VUS) according to ACMG classification. A total of 20 variants in non-WSPH-listed PAH genes were identified in 11 IPAH patients. 85% of the found variants were missense variants, and the others were stop-gain (5%), frameshift (5%), and inframe deletion (5%) variants. 90% of the identified variants were classified as VUS, while 10% were classified as likely pathogenic (LP) (a missense variant in the NOTCH3 gene and a frameshift variant in the PTGIS gene). Moreover, IPAH patients with rare variants in non-WSPH-listed PAH-related genes had higher levels of proBNP (p=0.030) and lower ejection fraction (p=0.019). Among CTEPH patients, two missense variants classified as VUS were found in ACVRL1 and KDR genes in two patients. In addition, twelve missense variants in PAH-related non-WSPH-listed genes classified as VUS were identified in seven CTEPH patients.

Conclusions: The findings suggest that rare PAH-related gene variant carriage in IPAH patients are associated with more severe clinical features, highlighting the need for further research to clarify the pathogenic roles of these genetic variants and their impact on disease progression.

Pulmonary Hypertension / Pulmonary Vascular Diseases

OP-032

Clinical and hemodynamic correlates of the ratio of mixed venous oxygen saturation to pulmonary capillary wedge pressure in patients underwent to cardiac catheterization due to suspicion of pulmonary hypertension

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Background and Aim: Pulmonary hypertension (PH) is a severe condition, which can lead to significant cardiovascular and functional impairments. The ratio of mixed venous oxygen saturation to pulmonary capillary wedge pressure (RSW) can be a potential marker for assessing disease severity and guiding treatment. We aim to evaluate the clinical, demographic, laboratory, echocardiographic, and hemodynamic parameters in patients with suspected pulmonary hypertension categorized into four groups based on their RSW levels.

Methods: This study included 82 patients who underwent to cardiac catheterization due to suspicion of pulmonary hypertension, stratified into four groups based on their RSW levels: Group 1 (lowest RSW), Group 2, Group 3, and Group 4 (highest RSW). We assessed various parameters, including demographic characteristics, 6-minute walk distance (6MWD), laboratory biomarkers, echocardiographic findings, and right heart catheterization metrics.

Results: Significant differences were observed across the four groups: Age decreased significantly with increasing RSW (67.10 ± 9.35 years in Group 1 to 47.23 ± 11.35 years in Group 4, p<0.001). Body mass index (BMI) did not differ significantly (p=0.147). 6MWD significantly improved with higher RSW (314.75 ± 57.86 meters in Group 1 to 447.61 ± 68.01 meters in Group 4, p<0.001). eGFR increased significantly with RSW levels (62.20 ± 24.52 mL/min/1.73 m^2 in Group 1 to 91.57 ± 23.28 mL/min/1.73 m^2 in Group 3, p=0.001). Serum uric acid and hemoglobin levels also varied significantly (serum uric acid p=0.009; hemoglobin p=0.014). RA increased with RSW levels (48.88 ± 10.68 mm in Group 1 to 46.41 ± 7.94 mm in Group 4, p=0.036). No significant differences were observed in LV EF, LA, and TAPSE. PAPmean (p<0.001) and PCWP (p<0.001) decreased significantly with increasing RSW levels. CI and PVR also showed significant variations (CI: 2.40 ± 0.58 L/sec/m^2 in Group 1 to 3.00 ± 0.72 L/sec/m^2 in Group 4, p<0.001; PVR: 6.70 ± 3.89 WU in Group 1 to 3.52 ± 2.04 WU in Group 4, p<0.001).

Conclusions: This study demonstrates that the RSW ratio is a valuable marker in categorizing patients with PH. Higher RSW levels are associated with improved functional capacity and significant variations in clinical, laboratory, echocardiographic, and hemodynamic parameters. These findings highlight the potential of RSW as a tool for assessing disease severity and guiding management strategies in PH.

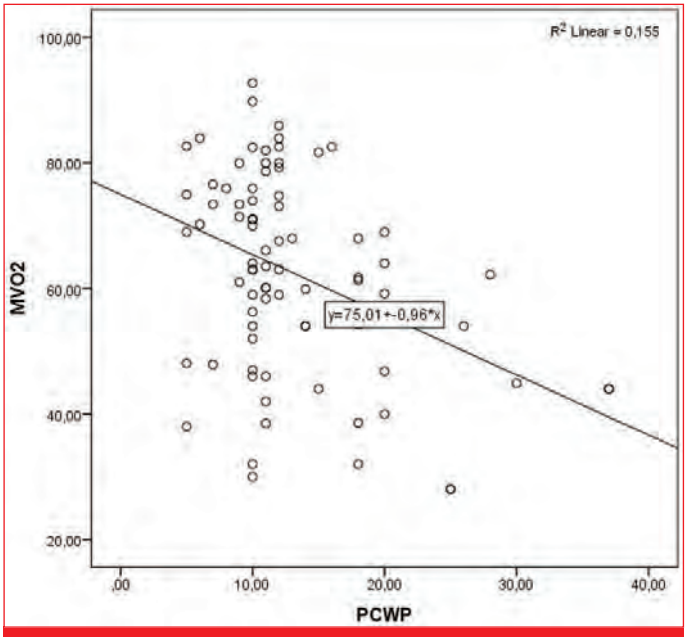

Figure 1. Scatter plot showing the ratio of mixed venous oxygen saturation (SvO_2) to pulmonary capillary wedge pressure (PCWP) across different patients. The trend line indicates the correlation between the two variables.

Table 1. Results of the study

	Group 1 (n=20)	Group 2 (n=20)	Group 3 (n=21)	Group 4 (n=21)	P value
RSW	2,33 ± 0,81	4,64 ± 0,72	6,50 ± 0,51	9,88 ± 2,79	< 0,001
Demographical Features					
Age, year	67,10 ± 9,35	57,55 ± 18,45	59,57 ± 14,50	47,23 ± 11,35	< 0,001
BMI, kg/m²	31,95 ± 6,29	27,14 ± 7,67	29,30 ± 6,34	27,49 ± 7,74	0,147
6MWD, m	314,75 ± 57,86	357,20 ± 114,24	361,33 ± 122,74	447,61 ± 68,01	< 0,001
Laboratory Parameters					
proBNP, pg/ml	753,58 ± 596,33	1269,05 ± 1418,21	580,62 ± 745,96	770,75 ± 1117,38	0,251
eGFR, ml/sec/m²	62,20 ± 24,52	81,25 ± 20,01	91,57 ± 23,28	88,80 ± 25,24	0,001
Uric acid, mg/dl	7,02 ± 2,07	7,26 ± 2,16	5,57 ± 2,15	5,52 ± 1,56	0,009
Hemoglobin, g/dL	11,17 ± 1,96	14,02 ± 2,12	13,63 ± 2,59	13,60 ± 2,71	0,014
Echocardiographic Parameters					
LV EF, %	58,63 ± 3,00	59,47 ± 2,83	58,25 ± 5,19	59,65 ± 3,81	0,619
LA, mm	41,21 ± 7,72	40,22 ± 9,04	38,30 ± 6,29	37,00 ± 7,31	0,317
RA, mm	48,88 ± 10,68	54,31 ± 15,07	44,05 ± 8,49	46,41 ± 7,94	0,036
TAPSE, mm	19,95 ± 4,98	17,00 ± 4,24	19,77 ± 4,64	20,38 ± 5,12	0,126
TJV, m/sec	3,97 ± 0,43	4,23 ± 0,79	3,49 ± 0,76	3,46 ± 0,58	0,001
Right Heart Catheterization Parameters					
RAP, mmHg	14,94 ± 9,27	12,56 ± 7,43	7,52 ± 4,83	7,15 ± 3,32	0,001
PAP_{mean}, mmHg	46,15 ± 14,48	53,68 ± 17,77	34,61 ± 12,51	35,23 ± 16,65	< 0,001
PCWP, mmHg	21,60 ± 7,33	12,70 ± 2,81	10,61 ± 1,28	8,09 ± 2,36	< 0,001
$Aorta_{mean}$, mmHg	111,45 ± 19,55	91,85 ± 16,95	99,66 ± 12,47	96,85 ± 13,79	0,002
CI, L/sec/m²	2,40 ± 0,58	1,97 ± 0,62	2,45 ± 0,71	3,00 ± 0,72	< 0,001
PVR, WU	6,70 ± 3,89	12,50 ± 7,83	5,36 ± 3,74	3,52 ± 2,04	< 0,001
SVR, WU	22,08 ± 6,37	23,51 ± 5,75	21,87 ± 6,04	18,09 ± 4,84	0,024

Pulmonary Hypertension / Pulmonary Vascular Diseases

OP-033

Correlating dicrotic notch index with hemodynamic, echocardiographic, and clinical parameters in pulmonary hypertension

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Background and Aim: Pulmonary hypertension (PH) is characterized by increased pulmonary artery pressure (PAP) and pulmonary vascular resistance (PVR) due to loss of arterial elasticity and vascular obstruction. The Dicrotic Notch Index (DNI) captures reflected wave characteristics and vascular elasticity, potentially serving as an important marker in PH assessment. This study evaluates the relationship between DNI, PVR, PAPs, and other clinical parameters in PH patients.

Methods: A retrospective analysis was conducted on 56 patients diagnosed with PAH, CTEPH, or normal PAPs who underwent right heart catheterization (RHC). PAP waveforms were analyzed to determine DNI. Correlations between DNI and hemodynamic, clinical, and echocardiographic parameters were assessed using Pearson correlation coefficients. Logistic regression and ROC analysis were performed to evaluate DNI's predictive value.

Results: DNI showed significant positive correlations with sPAP (r=0.972, p<0.001), dPAP (r=0.876, p<0.001), mPAP (r=0.987, p<0.001), RA pressure (r=0.741, p=0.018), and PVR (r=0.814, p<0.001). Significant negative correlations were observed with CI (r=-0.573, p=0.012) and pSO2 (r=-0.516, p=0.043). Univariable logistic regression identified DNI as a significant predictor of PH (OR: 1.100, 95% CI: 1.048-1.155, p<0.001). ROC analysis showed an AUC of 0.922 for DNI, indicating excellent predictive value.

Conclusions: DNI is strongly correlated with key hemodynamic parameters such as PVR and mPAP, underscoring its potential as a crucial marker in the assessment of PAH and

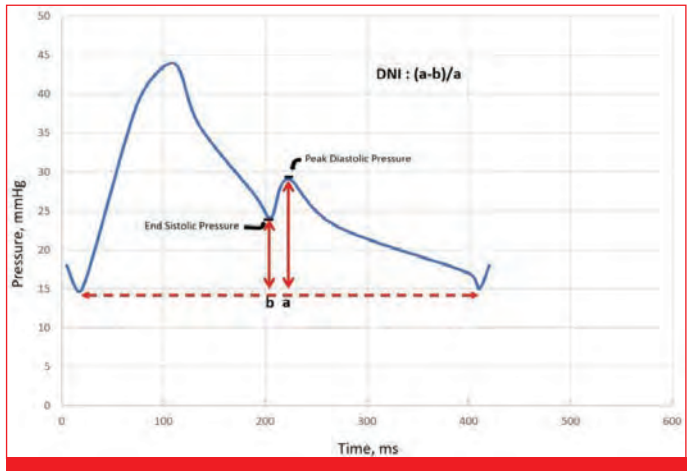

Figure 1.

CTEPH patients. Additionally, DNI exhibited significant correlations with other RHC parameters, echocardiographic measurements, and clinical indicators, suggesting its utility in evaluating pulmonary arterial stiffness and resistance. Further research is needed to validate these findings in larger cohorts and to establish standardized protocols for DNI measurement in clinical practice.

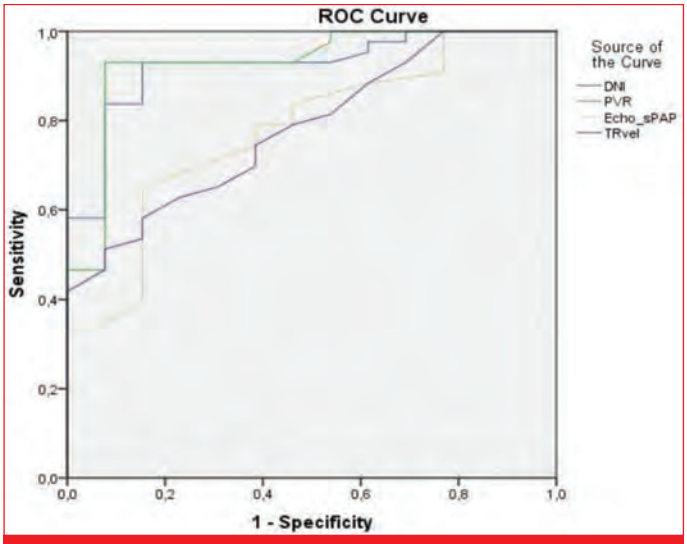

Figure 2.

Table 1. Baseline Clinical Characteristics of Study Patients

Characteristic	Value (n = 56)
Age, years	52.4 ± 15.2
Gender, male, %	23 (41.06%)
Body mass index, kg/m²	23.6 ± 3.1
HT, no. (%)	20 (35.7%)
DM, no. (%)	8 (14.2%)
Beta Blocker, no. (%)	15 (26.7%)
Non-DPN KK, no. (%)	4 (7.1%)
DPN KK, no. (%)	9 (16.07%)
Diuretic, no. (%)	26 (46.4%)
ASA, no. (%)	3 (5.3%)
OAC, no. (%)	12 (21.4%)
MRA, no. (%)	8 (14.2%)
WBC, 10^9/L	8.43 ± 2.69
Hemoglobin, g/dL	13.2 ± 1.40
Platelets, 10^9/L	238 ± 88
Creatinine, mg/dL	0.87 ± 0.32
NT-ProBNP, pg/mL	686.4 ± 950
6MWT, m	372 ± 145
Normal patients (based on RHC), no. (%)	10(17.8%)
PAH subgroup, no. (%)	34 (60.7%)
- Idiopathic	22 (64.7%)
- Associated with:	
- Connective tissue disease	8 (23.5%)
- Congenital heart disease	4 (11.1%)
CTEPH subgroup, no. (%)	12 (21.4%)
Rheumatologic Disease, no. (%)	
- RA	2 (3.6%)
- Scleroderma	5 (8.9%)
Procedure Site, no. (%)	
- Femoral	18 (32.1%)
- Jugular	38 (67.8%)

Abbreviations: HT: Hypertension, DM: Diabetes Mellitus, Non-DPN: Non-Dihydropyridine-derived calcium channel blockers, DPN: Dihydropyridine-derived calcium channel blockers, ASA: Acetylsalicylic Acid, OAC: Oral Anticoagulant, MRA: Mineralocorticoid Receptor Antagonist, RA: Rheumatoid Arthritis, WBC: White Blood Cell, 6MWT: 6-Minute Walk Test, Pro-BNP: Pro-Brain Natriuretic Peptide

Table 2. Echocardiographic and Catheterization Measurements

Characteristic	Mean ± Std. Deviation	Minimum	Maximum
EF, %	60.22 ± 5.12	55	67
Echo sPAB, mmHg	49.18 ± 22.52	36	160
TR vel, m/s	3.62 ± 0.88	2.8	7.8
TAPSE, mm	16.20 ± 5.16	10.0	24.0
PaccT, ms	107.89 ± 32.937	61	156
Right Heart Catheterization Parameters			
PA Systolic, (mmHg)	58.12 ± 26.2	28	167
PA Mean, (mmHg)	34.76 ± 17.2	14	100
PA Diastolic, (mmHg)	22.50 ± 14.0	6	70
Systemic sBP, (mmHg)	122.8 ± 24.4	86	187
Systemic dBP, (mmHg)	80.8 ± 16.1	54	108
Systemic mBP, (mmHg)	90.89 ± 17.5	64	132
PWCP, (mmHg)	10.10 ± 3.1	6	15
RV Systolic, (mmHg)	56.34 ± 29.5	0	162
RA Mean, (mmHg)	11.56 ± 6.2	4	17
sSO2, %	94.88 ± 4.9	79.2	100.0
pSO2, %	65.32 ± 11.3	42.0	94.0
mvSO2, %	67.86 ± 8.16	48.0	78.0
CO (Fick), L/min	4.92 ± 1.65	1.96	7.84
CI (Fick), L/min/m²	2.72 ± 0.88	1.22	5.16
PVR, Wood units	5.20 ± 6.2	1.04	35.04

Abbreviations: EF: Ejection Fraction, sPAP: Systolic Pulmonary Artery Pressure, TY reg jet: Tricuspid Jet Velocity, TAPSE: Tricuspid Annular Plane Systolic Excursion, PaccT: Pulmonary Acceleration Time, PA: Pulmonary Artery, BP: Blood Pressure, PWCP: Pulmonary Capillary Wedge Pressure, RV: Right Ventricule, RA: Right Atrium sSO2: Systemic Oxygen Saturation, pSO2: Pulmonary Artery Oxygen Saturation, mvSO2: Mixed Venous Oxygen Saturation, CO: Cardiac Output, CI: Cardiac Index, SVR: Systemic Vascular Resistance, PVR: Pulmonary Vascular Resistance

Table 3. Correlation of Right Heart Parameters with Dicrotic Notch Index

Variables	Dicrotic Notch Index	
	r-value	p-value
RHC parameters		
sPAP (mmHg)	0.972	**<0.001**
dPAP (mmHg)	0.876	**<0.001**
mPAP (mmHg)	0.987	**<0.001**
RA (mmHg)	0.741	**0.018**
PCWP (mmHg)	0.077	0.224
PVR (Wood units)	0.814	**<0.001**
CO (Fick) (L/min)	-0.207	0.063
CI (Fick) (L/min/m²)	-0.573	**0.012**
pSO2	-0.516	**0.043**
ECHO parameters		
TR vel (m/s)	0.770	**0.018**
sPAP (mmHg)	0.701	**0.006**
TAPSE (mm)	-0.171	0.308
AccT (ms)	-0.072	0.718
TAPSE/SPAP ratio	-0.106	0.342
Other parameters		
NT-ProBNP, pg/mL	0.684	**0.003**
6MWT, m	-0.305	0.292

RHC: Right Heart Catheterization ,sPAP: Pulmonary Artery Systolic, dPAP: Pulmonary Artery Diastolic, mPAP: Pulmonary Artery Mean, RA: Right Atrium Mean Pressure, PCWP: Pulmonary Capillary Wedge Pressure, PVR: Pulmonary Vascular Resistance, CO: Cardiac Output, CI: Cardiac Index, TR reg vel: Tricuspid Regurgitation Velocity, AccT: Pulmonary Artery Acceleration Time, 6MWT: 6-Minute Walk Test,

Table 4. Univariable and Multivariable Analysis of Predictors for Pulmonary Hypertension

Parameter	Univariable Analysis OR (95% CI)	p-value	Multivariable Analysis OR (95% CI)	p-value
DNI	1.100 (1.048-1.155)	<0.001	1.061 (1.007-1.118)	0.025
Echo sPAP, mmHg	1.066 (1.025-1.108)	<0.001	0.996 (0.985-1.007)	0.482
NT-proBNP	1.000 (1.000-1.000)	0.067	1.000 (0.999-1.000)	0.461
TR vel, m/s	1.187 (1.053-1.338)	0.005	1.192 (0.895-1.588)	0.223

DNI: Dicrotic Notch Index, TAPSE: Tricuspid Annular Plane Systolic Excursion, sPAP: Systolic Pulmonary Artery Pressure, NT-proBNP: N-terminal pro b-type Natriuretic Peptide

Heart Failure

OP-035

Comparison of different formulations in the calculation of pulmonary elastance on mortality

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Background and Aim: Heart failure (HF) is a clinical syndrome resulting from structural or functional abnormalities of the heart that lead to a decrease in cardiac output and an increase in cardiac pressures at rest or during exercise. In systolic heart failure, right ventricular afterload increases due to pulmonary vascular structures and haemodynamic changes. This leads to right ventricular hypertrophy, reduced right ventricular ejection fraction (EF), reduced stroke volume, reduced cardiac output (CO) and eventually progressive right heart failure. Right ventricular function is important for prognosis; therefore, it is essential to understand and assess right ventricular afterload. In our study, we compared the effect of different formulae for calculating pulmonary elastance, one of the parameters used to assess right ventricular afterload, on prognosis.

Methods: The single-centre retrospective study included 352 patients who underwent right heart catheterisation (RHC) for heart failure with low ejection fraction (HFrEF) at Dr. Siyami Ersek Thoracic Heart and Vascular Surgery Training and Research Hospital between 2018 and 2022. Medical history, demographic characteristics, cardiovascular risk factors, comorbid diseases, New York Heart Association functional class, echocardiography results and haemodynamic data of right heart catheterisation were recorded in the retrospectively reviewed patient files. Mortality data were obtained from the national registry. Patients were divided into two groups according to mortality status. Pulmonary elastance was calculated by different formulae adjusted according to stroke volume and stroke volume index: Ea (PV)= pulmonary artery systolic pressure/stroke volume, Ea*(PV)=(pulmonary artery mean pressure-pulmonary capillary end pressure)/stroke volume, PP/SV= (pulmonary artery systolic pressure-pulmonary artery diastolic pressure)/stroke volume, Ea(PV)φ=pulmonary artery systolic pressure/stroke volume index, Ea*(PV)φ=(pulmonary artery mean pressure-pulmonary capillary end pressure)/stroke volume index, PP/SVφ=(pulmonary artery systolic pressure-pulmonary artery diastolic pressure)/stroke volume index. The results were compared.

Results: The study included 352 patients with HFrEF, and mortality developed in 139 (39.1%) patients during the follow-up period. The mean Ea (PV), Ea*(PV), PP/SV, Ea(PV)φ, Ea*(PV)φ, PP/SVφ of the patients who developed mortality were higher than the surviving patients and the difference was statistically significant. When the effect of echocardiography and RHC data of the patients on mortality risk was evaluated by multivariate analysis, it was observed that Ea (PV) and PP/SV predicted mortality and continued to predict mortality in the patient group with normal PVR.

Conclusions: Different formulae used in the calculation of pulmonary elastance have been shown to be effective in determining mortality. According to the results of the study, it can be said that Ea (PV) and PP/SV are more successful predictors of mortality than PVR.

Heart Failure

OP-036

The ratio of tricuspid annular plane systolic excursion to pulmonary artery systolic pressure may predict poor outcomes following interatrial shunting device implantation in patients with heart failure

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Background and Aim: Previous studies have demonstrated that implanting an interatrial shunting device may improve outcomes in patients with heart failure, irrespective of left ventricle ejection fraction. However, its efficacy varies among individuals. This study aimed to explore whether the tricuspid annular plane systolic excursion (TAPSE) /systolic PA pressure ratio can be useful in predicting clinical outcomes after implantation of the interatrial shunting device.

Methods: Our study was the exploratory analysis of the AFR-PRELIEVE study. AFR-PRELIEVE was a prospective, non-randomized, multicenter, first-in-man study in symptomatic heart failure patients regardless of the left ventricle ejection fraction. Patients with pulmonary capillary wedge pressure (PCWP) ≥15 at rest received an 8-mm device; patients with resting PCWP <15 and exercise-induced increase ≥25 received the 10-mm device. A total of 106 HF patients, n=62 with reduced ejection fraction (HFrEF), n=44 with preserved ejection fraction (HFpEF), were prospectively enrolled in the AFR-PRELIEVE registry with the Atrial Flow Regulator (AFR)

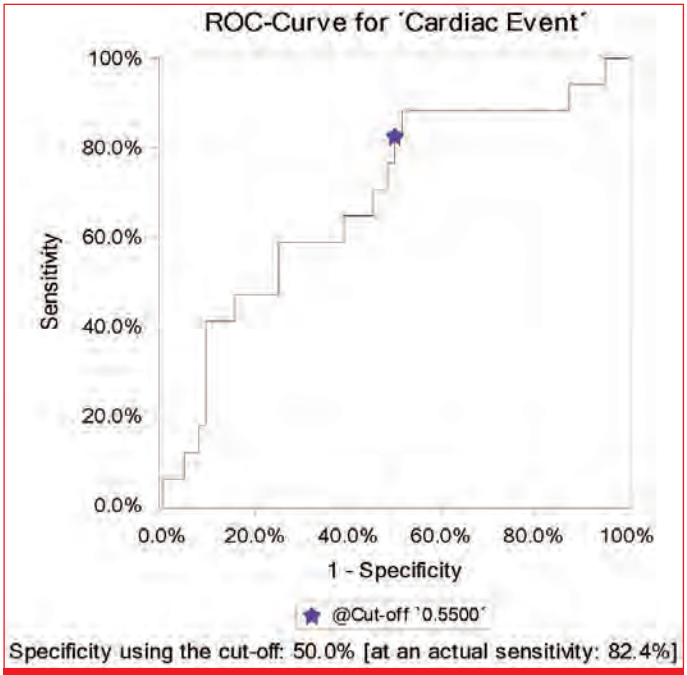

Figure 1. ROC curve for predicting of serious adverse effects following interatrial shunt device.

implantation. Serious adverse events included cardiovascular mortality and worsening HF (WHF).

Results: Serious adverse events (SAEs) occurred in a total of 19 patients (18%). There were no significant differences in baseline comorbidities, KCCQ scores, or the 6-minute walking distance between patients who experienced SAEs and those who did not (Table). Patients who experienced SAEs exhibited higher systolic pulmonary arterial pressure, NT-proBNP levels, and serum creatinine, as well as a lower TAPSE/PASP ratio. The incidence of SAEs was significantly higher among patients in the first TAPSE/PASP tertile compared to those in the third tertile (59% vs. 12%, p=0.026). A TAPSE/PASP ratio of <0.55 was identified as having the highest sensitivity for predicting these events (AUC=0.683, 95% CI: 0.533-0.833) (Figure).

Conclusions: The reduced TAPSE/PASP ratio was associated with higher rates of serious adverse events in patients with heart failure undergoing controlled inter-atrial shunting. Our result that decreased TAPSE/PASP ratio could indicate potential clinical non-responders to left atrial decompression warrants further prospective validation.

Table 1. Baseline characteristics of patients with and without developing serious adverse events

Parameters	Serious adverse events (-) n=87	Serious adverse events (+) n=19	p value
Age, years, Median (IQR)	67.0 (60.0, 73.0)	71.0 (67.0, 74.0)	0.09
Male, n (%)	48 (55.2)	12 (63.2)	0.52
BMI, Median (IQR)	29.4 (25.1, 33.1)	26.0 (22.9, 31.6)	0.12
Hypertension, n (%)	56 (64.4)	13 (68.4)	0.74
Diabetes mellitus, n (%)	34 (39.1)	8 (42.1)	0.81
Coronary artery disease, n (%)	29 (33.3)	9 (47.4)	0.25
Atrial fibrillation, n (%)	30 (34.5)	7 (36.8)	0.85
Permanent pacemaker, n (%)	12 (13.8)	4 (21.1)	0.42
HFrEF (LV EF ≤40%), n (%)	48 (55.2)	14 (73.7)	0.14
HFpEF (LV EF >40%), n (%)	39 (44.8)	5 (26.3)	0.14
KCCQ-OSS, median (IQR)	41.7 (27.1, 59.1)	39.6 (30.7, 64.1)	0.84
6MWD, m, median (IQR)	200 (110, 300)	160 (85, 200)	0.053
Mitral E/E', m/s, median (IQR)	12.0 (8.1, 16.0)	18.1 (10.6, 25.4)	0.02
TAPSE, mm, median (IQR)	19.0 (17.0, 24.0)	17.0 (15.0, 22.0)	0.08
SPAP, mmHg, median (IQR)	35.0 (26.5, 40.0)	40.0 (36.5, 50.0)	0.04
TAPSE (mm)/PASP (mmHg), median (IQR)	0.54 (0.43, 0.84)	0.43 (0.33, 0.53)	0.02
CO, L/min, median (IQR)	4.41 (3.56, 5.60)	3.94 (3.01, 4.28)	0.07
PVR, W, median (IQR)	1.62 (0.46, 2.65)	2.77 (0.00, 4.99)	0.28
Serum creatinine, mg/dL, median (IQR)	0.96 (0.82, 1.23)	1.15 (0.95, 1.35)	0.03
NT-proBNP, pg/mL, median (IQR)	317 (114, 1200)	1261 (567, 1790)	0.01

6MWD: 6-minute walking distance; BMI: Body mass index; BUN: Blood urine nitrogen; CO: Cardiac output; HFpEF: Heart failure with preserved ejection fraction; HFrEF: Heart failure with reduced ejection fraction; IQR: Interquartile range; KCCQ-OSS: Kansas City Cardiomyopathy Questionnaire Overall Summary Score; LVEF: Left ventricle ejection fraction; PVR: Pulmonary vascular resistance; SD: Standard deviation; SPAP: Systolic pulmonary arterial pressure; TAPSE: Tricuspid annular plane systolic motion.

Heart Failure

OP-037

Clinical management strategies of cardiologists in heart failure with reduced ejection fraction in Türkiye

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Background and Aim: This survey aims to determine the current opinions of cardiologists practicing in Türkiye about the management of heart failure with reduced ejection fraction (HFrEF).

Methods: The survey material was composed of twenty-two individual questions and published on the SurveyMonkey platform.

Results: Overall, 177 cardiologists (mean age: 39.5 years, 73.3% male) completed the survey, of which 38.7% were practicing in an education and research hospital and 10.2% were HF-specialists. The cut-off EF value to define HFrEF was ≤40% for 80.1% of the cardiologists (Figure 1A). Although ARNi treatment was considered the most efficient HF medication for 52.6% of physicians, 62.7% of the study participants would initiate to HF treatment with an ACEi instead of ARNi due to reimbursement regulations and cost issues (Figure 1B). The present survey revealed that starting medical therapy with an ARNi instead of ACEi in patients with de novo HFrEF was more common in HF specialists than non-specialists (72.2% vs. 33.3%, p=0.001) (Figure 1B). The majority of participants who are practicing in a university hospital with an academic rank would start medical therapy with an ARNi (university hospital 54.0% vs. state hospital 10.0%, p=0.008) (Figure 1C-D). More than half of the cardiologists (52.3%) declared that adding another class of HF medication is more important than up-titrating those already started. Although 69.5% of the study participants stated that it is possible to prescribe all four classes of HF medications during the index hospitalization period, the majority of cardiologists preferred the sequential approach which starts with ACEi/ARNi first, beta-blockers second, MRAs third, and SGLT2i fourth. Although more than 40% of participants with academic rank declared that the most realistic time to reach the maximal up-titration was 3 months, 50% of research assistants thought that the maximal up-titration time was only 1 month (p=0.028) (Figure 2A). HF specialists were more dedicated to optimizing and up-titration of HF medications compared to the non-specialists (p=0.013) (Figure 2B). To the question 'if you had to choose only one heart failure medication class for a patient with HFrEF, which medication class would you choose?', 44.1% of cardiologists answered ARNi, followed by beta–blockers (23.7%), ACEi (22.0%), SGLT2i (7.3%), and MRAs (2.8%) (Figure 3A). In the subgroup analysis, 83.3% of the HF specialists answered ARNi versus 39.6% among non-HF specialists (p=0.009) (Figure 3B). Only one-fourth of cardiologists (n = 43, 24.3%) choose to start MRAs even if the glomerular filtration rate is <30 mL/min (Figure 4A-B).

Conclusions: The present survey demonstrated that there are significant gaps between guideline recommendations and real-life clinical practice of cardiologists in Türkiye. These results suggest that there is a need for organized action by healthcare providers to improve implementation of guideline recommendations.

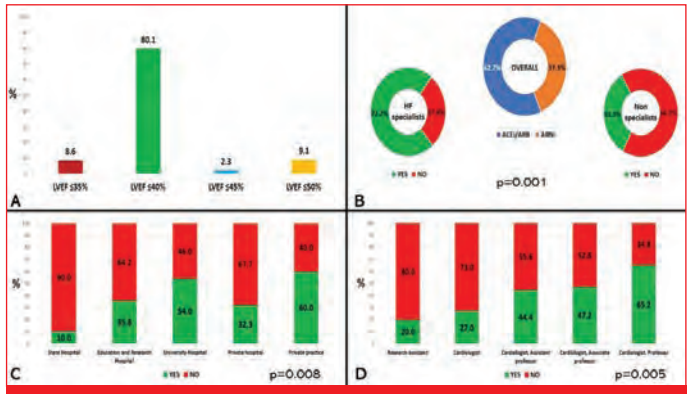

Figure 1. The accepted ejection fraction cut-off value to define HFrEF among survey participants (A); the answers to the question "ACEi or ARNi choice in patients with de novo HFrEF: Which one is the first?" (B); the answers to the question "do you start HF treatment with an ARNi instead of ACEi in patients with de novo HFrEF" according to the HF specialists versus non-specialists (B), location of practice (C), and academic rank (D).

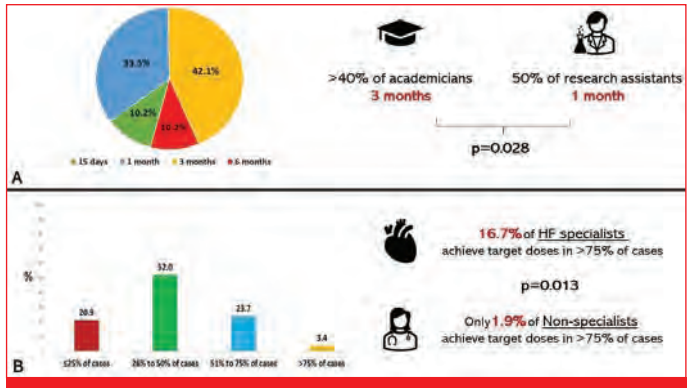

Figure 2. The most realistic time to reach the maximal up-titration of HF medications and the difference between the participants with an academic rank and research assistants (A); the answers to the question "how many patients do you achieve titration to full doses in clinical routine?" and the difference between the HF specialists versus non-specialists (B).

Figure 3. The answers to the question "if you had to choose only one heart failure medication class for a patient with HFrEF, which medication class would you choose?" (A); and 83.3% of the HF specialists answered ARNi to the same question versus only 39.6% among non-HF specialists (B).

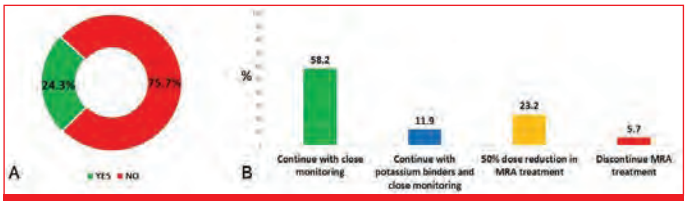

Figure 4. Do you introduce MRAs in HFrEF patients with GFR < 30 mL/min (A); and the answers to the question ''what is your management strategy in patients with serum potassium level 5.0–5.5 µmol/L and receiving MRA treatment?'' (B).

Heart Failure

OP-038

Left atrio-ventricular global longitudinal strain as a predictor of mortality in heart failure with preserved ejection fraction

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Background and Aim: Despite advances in the treatment of heart failure with preserved ejection fraction (HFpEF), mortality rates remain high, underscoring the importance of identifying high-risk groups. Left atrio-ventricular global longitudinal strain (LAVGLS) provides comprehensive information about left atrial and left ventricular strain. The aim of this study is to investigate the use of echocardiographically assessed LAVGLS values as a predictor of 2-year all-cause mortality in heart failure with preserved ejection fraction.

Methods: This single-center prospective study involved 206 patients diagnosed with preserved ejection fraction heart failure between March 2020 and November 2022. Left atrio-ventricular global longitudinal strain (LAVGLS) was calculated as the sum of peak LASr and the absolute value of peak LVGLS (LAVGLS=LASr+|LVGLS|). Patients were divided into two groups according to the median of the LAVGLS (31.1): low (≤31.1) and high (>31.1) levels. Detailed clinical data, including demographic information (age, gender), comorbidities (hypertension, diabetes mellitus, atrial fibrillation, etc.), laboratory tests (blood routine, electrolytes, etc.), and echocardiographic features, were recorded. Information on 2-year all-cause mortality was obtained from the hospital's electronic record system.

Results: A total of 206 HFpEF patients (69.1 mean age ± 10.9 years, %65.5 female) were enrolled. In the overall population, %30.1 had coronary artery disease, and %75.2 had hypertension. Among comorbidities, the presence of hypertension, diabetes, and chronic renal failure were similar in both. In the group with to low LAVGLS group, the H2FPEF score, which aids in making a clinical diagnosis, was significantly higher (6.0 ± 2.1 vs. 4.2 ± 2.1, p<0.001). Systolic pulmonary artery pressure, right atrium diamater, right atriyum area, TAPSE, LAVmin and RV strain was significantly different between groups. Other echocardiographic parameters were observed similarly. In the low LAVGLS group, proBNP was significantly elevated. In the low LAVGLS group, 1-year all-cause mortality was %19, and 2-year all-cause mortality was %29.3 (Table 1). Kaplan-Meier analysis demonstrated higher 2-year all-cause mortality in HFpEF patients in the low LAVGLS group (log-rank p=0.008).

Conclusions: Kaplan-Meier survival curves for 2-year all-cause mortality in HFpEF patients according to low or high LAVGLS provides information about left atrial function and structure, as well as right heart functions, and may offer insights into mortality in patients with heart failure with preserved ejection fraction.

Table 1. Relationships between clinical and laboratory data and the LAVGLS in patients with HFpEF

	LAVGLS ≤31.1 (n=104)	LAVGLS >31.1 (n=102)	p value
Age, years, ± SD	71.1 ± 9.6	66.5 ± 11.6	<0.001
Female, n(%)	68 (65.4%)	67 (65.7%)	0.964
Body mass index (kg/m^2)	28.3 ± 4.7	30.6 ± 6.7	0.005
HT, n (%)	73 (47.1%)	82 (52.9%)	0.090
DM, n (%)	39 (49.4%)	40 (50.6%)	0.800
Chronic renal failure, n (%)	42 (40.4%)	31 (30.4%)	0.134
H2FPEF score	6.0 ± 2.1	4.2 ± 2.1	<0.001
NT-proBNP, pg/mL, (IQR)	2389 (1193-4440)	727 (403-1471)	<0.001
Hemoglobin, g/dL, ± SD	12.1 ± 2.1	12.7 ± 2.2	0.116
Creatinine, mg/dL, ± SD	1.0 (0.8-1.4)	0.9 (0.7-1.3)	0.121
Glucose, mg/dL, (IQR)	115 (96.0-151)	110 (95-155)	0.971
Sodium, mmol/L, ± SD	139.1 ± 3.7	138.9 ± 3.6	0.668
Potassium, mEq/L, ± SD	4.3 ± 0.5	4.5 ± 0.5	0.019
1 year all-cause mortality, n (%)	19 (19%)	11 (10.8%)	0.101
2 year all-cause mortality, n (%)	29 (29.3%)	14 (13.9%)	0.008

LA: Left atrial; LAVmin: Left atrial volume minimum; TAPSE: Tricuspid annular plane systolic excursion; LVEF: Left ventricular ejection fraction; LAEF: Left atrial ejection fraction; RV: Right ventricule; RA: Right atrial; SPAP: Systolic pulmonary arterial pressure.

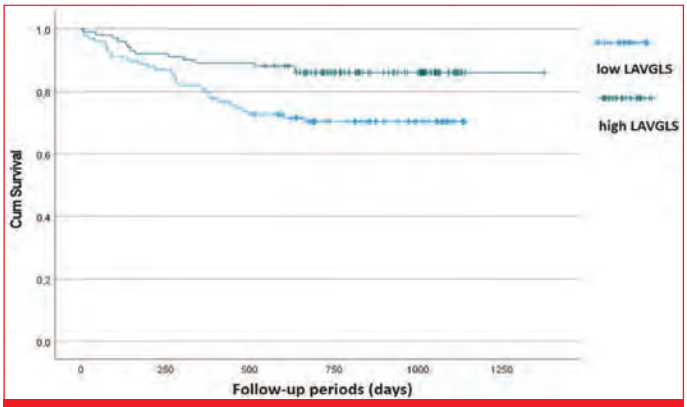

Figure 1. Kaplan-Meier survival curves for 2-year all-cause mortality in HFpEF patients according to low or high LAVGLS (Log-rank test < 0.008).

Table 2. Relationships between echocardiographic features the LAVGLS in patients with HFpEF

	LAVGLS ≤31.1 (n=104)	LAVGLS >31.1 (n=102)	p value
LVEF, %, ± SD	60.1 ± 5.6	61.0 ± 4.9	0.248
RA diameter, mm, ± SD	44.8 ± 4.0	38.6 ± 5.3	<0.001
RA area, mm^2, ± SD	19.7 ± 6.1	15.2±4.5	<0.001
SPAP, mm Hg, ± SD	48.7 ± 17.8	37.4 ± 13.6	<0.001
Left atrium volume index, mL/m^3 ± SD	45.8 ± 13.5	31.9 ± 9.3	<0.001
TAPSE diameter, mm, ± SD	20.7 ± 4.1	22.0 ± 3.4	0.012
LAEF, %, ± SD	38.2 ± 10.0	56.3 ± 10.5	<0.001
LAVmin, ml, [IQR]	54.4 (37.2-74.5)	27.1 (19.8-36.8)	<0.001
RV strain, %	-15.8±5.3	-22.2±4.7	<0.001

LA: Left atrial; LAVmin: Left atrial volume minimum; TAPSE: Tricuspid annular plane systolic excursion; LVEF: Left ventricular ejection fraction; LAEF: Left atrial ejection fraction; RV: Right ventricule; RA: Right atrial; SPAP: Systolic pulmonary arterial pressure.

Heart Failure

OP-039

Predicting worsening in chronic heart failure using artificial intelligence (PROHEART AI); Interim analysis

Zekeriya Doğan[1], Murat Sünbül[1], Erkan Çeçen[2], Tarık Kıvrak[2], Selda Murat[3], İstiklal Özkaya[3], Cihat Çalışkan[3], Bektaş Murat[4], Mevlüt Demir[5], Taner Şen[5], Cabir Vural[6], Berrin Aysevinç[6], Songül Çeçen Düzel[6], Şevket Gündüz[6], Ali Serdar Fak[6]

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Background and Aim: Early detection of worsening in heart failure (HF) might lead to timely interventions for a better course. The purpose of our study is to develop an AI algorithm to predict worsening in patients with chronic HF using biological data coming from a smart textile.

Methods: This is a multicenter, prospective, clinical observational study in 5 cardiology centers. Stable patients with reduced ejection fraction (HFrEF) or with improved ejection fraction (HFimpEF) are included. All patients are supplied with a smart textile (ST) (Hexoskin, Carre Technologies Inc. Canada). After a brief training period, patients are asked to wear the ST daily during pre-specified time periods. ECG, respiration, and acceleration signals are derived from the embedded sensors. Data is collected weekly and analysed in the coordinating center. Patients with emergency visit or hospitalization due to heart failure or patients with an increase of at least >30% in serum NT-proBNP measurement are considered to have worse outcomes compared to patients with none of the above. The AI model developed by convolutional neural network architecture has 6 convolutional layers and 2 artificial neural layers. The model is trained by using the data sets derived from the data recorded at 1 week, 4 weeks, and within 1 month before

Figure 1. A sample of simultaneous recordings of ECG, acceleration X, acceleration Y and acceleration Z signals, and raw respiration (abdominal and thoracic) signals.

Table 1.

Dataset Type	Accuracy	Recall (sensitivity)	Precision	F1-Score
ECG	0.94* 0.96** 0.98***	0.94 0.96 0.97	0.93 0.96 0.97	0.93 0.96 0.98
Acceleration-x	0.54 0.40 0.59	0.55 0.40 0.62	0.54 0.40 0.59	0.52 0.39 0.56
Acceleration-y	0.50 0.40 0.54	0.51 0.40 0.53	0.50 0.40 0.53	0.48 0.39 0.53
Acceleration-z	0.65 0.50 0.57	0.66 0.51 0.56	0.65 0.50 0.57	0.63 0.48 0.56
Respiration Thoracic	0.72 0.78 0.69	0.72 0.79 0.73	0.71 0.77 0.69	0.71 0.77 0.67
Respiration abdominal	0.61 0.77 0.51	0.61 0,80 0.51	0.60 0,77 0.50	0.60 0.76 0.47
Heart Rate	0.94 0.70 0.88	0.94 0.71 0.88	0.94 0.70 0.88	0.94 0.69 0.88
Heart Rate Quality	0.55 0.67 0.65	0.56 0.78 0.77	0.54 0.68 0.65	0.50 0.63 0.60
Activity	0.43 0.53 0.53	0.41 0.55 0.57	0.42 0.53 0.53	0.39 0.49 0.46
Breathing Rate	0.63 0.61 0.62	0.64 0.61 0.64	0.63 0.61 0.62	0.62 0.60 0.60
Breathing Rate Quality	0.63 0.61 0.62	0.64 0.61 0.64	0.63 0.60 0.62	0.62 0.60 0.61
Minute Ventilation	0.71 0.54 0.46	0.71 0.54 0.45	0.70 0.53 0.46	0.70 0.53 0.43
Minute Ventilation Adjusted	0.64 0.56 0.57	0.74 0.57 0.60	0.64 0.55 0.58	0.59 0.53 0.55
Tidal Volume	0.67 0.56 0.52	0.67 0.56 0.53	0.67 0.55 0.52	0.66 0.55 0.46

Validation performance of the one-channel model fed by raw and processed data measured while patients are at rest in different time periods (# of epoch: 20, Uncertainty: 0.01) *The first lines in each row represent the results obtained from training the model with the dataset created with the data at 4 weeks before the end point. **The second lines in each row represent the results obtained from training the model with the dataset created with the data recorded at one week before the end point. *** The third lines in each row represent the results obtained from training the modelwith the dataset created with the data within the last one-month data before the end point.

the worsening event. The model performs binary classification. Accuracy, precision, recall (sensitivity) and F1-score are used as performance metrics. The performance value above 0.7 is accepted as meaningful.

Results: A total of 33 patients (age 61.3 ± 12.2 years, F: 6), have been recruited since October 2023 and 6 patients are considered to have worse prognosis as of May 2024. An example of multichannel biological signals is given in Figure 1. ECG and heart rate signals during rest periods are found to be independently effective in classify worsening and stable patients (Table 1). When assessed as multichannel data, the highest performance belongs to the model trained with ECG signals together with thoracic respiration signals (Table 2). Validation performance of the two-channel model during activity is highest when ECG signals together with abdominal respiration signals are used (Table 3).

Conclusions: Biological data derived from a smart textile may classify worsening and in remission heart failure patients earlier. Training the model with ECG signals alone seems to be sufficient and could further be reinforced with the addition of respiration and acceleration signals during activity. AI might help in early detection of worsening in chronic heart failure patients.

Table 2.

Input Dataset Type: (Inputl1 + Input2)	Accuracy	Recall (sensitivity)	Precision	F1-Score
(ECG+Accelerationx)	0.94*	0.94	0.94	0.94
	0.96**	0.96	0.96	0.96
	0.98	0.98	0.98	0.98
(ECG+Accelerationy)	0.95	0.95	0.95	0.95
	0.95	0.95	0.95	0.95
	0.98	0.98	0.98	0.98
(ECG+Accelerationz)	0.97	0.97	0.97	0.97
	0.95	0.95	0.95	0.95
	0.98	0.98	0.98	0.98
(ECG+Respiration Thoracic)	0.99	0.99	0.99	0.99
	0.97	0.97	0.97	0.97
	0.99	0.99	0.99	0.99
(ECG+Respiration abdominal)	0.89	0.89	0.89	0.89
	0.96	0.96	0.96	0.95
	0.81	0.84	0.81	0.80
(ECG+Heart Rate)	0.96	0.96	0.96	0.96
	0.97	0.97	0.97	0.97
	0.97	0.97	0.97	0.97
(ECG+Heart Rate Quality)	0.94	0.95	0.94	0.94
	0.96	0.96	0.96	0.96
	0.98	0.98	0.98	0.98
(ECG+Activity)	0.96	0.96	0.96	0.96
	0.96	0.96	0.96	0.96
	0.98	0.98	0.98	0.98
ECG+Breathing Rate	0.94	0.96	0.94	0.94
	0.96	0.98	0.96	0.96
	0.98		0.98	0.98
ECG+Breathing Rate Quality	0.94	0.94	0.94	0.94
	0.95	0.96	0.96	0.95
	0.98	0.98	0.98	0.98
ECG+Minute Ventilation	0.96	0.96	0.96	0.96
	0.95	0.96	0.95	0.95
	0.98	0.98	0.98	0.98
ECG+Minute Ventilation Adjusted	0.97	0.97	0.97	0.97
	0.96	0.96	0.96	0.96
	0.99	0.99	0.99	0.99
ECG+Tidal Volume	0.96	0.96	0.96	0.96
	0.96	0.96	0.95	0.95
	0.98	0.98	0.98	0.98

Validation performance of the two- channel model fed by raw and processed data measured while patients are at rest in different time periods (# of epoch: 20, Uncertainty: 0.01). *The first lines in each row represent the results obtained from training the model with the dataset created with the data at 4 weeks before the end point. **The second lines in each row represent the results obtained from training the model with the dataset created with the data recorded at one week before the end point. *** The third lines in each row represent the results obtained from training the model with the dataset created with the data within the last one-month data before the end point.

Table 3. Validation performance of the two-channel model fed by raw and processed data measured while patients are active (# of epoch: 20, Uncertainty: 0.01)

Dataset Type	Accuracy	Recall (sensitivity)	Precision	F1-Score
ECG+ Acceleration-x	0.95	0.94	0.94	0.94
ECG+ Acceleration-y	0.97	0.97	0.97	0.97
ECG+ Acceleration-z	0.96	0.96	0.96	0.96
ECG+ Thoracic Respiration	0.96	0.96	0.95	0.95
ECG+ Abdominal Respiration	0.99	0.99	0.99	0.99
ECG+ Heart Rate	0.96	0.96	0.96	0.96
ECG+ Activity	0.94	0.95	0.94	0.94
ECG+ Breathing Rate	0.97	0.97	0.97	0.96
ECG+ Minute Ventilation	0.96	0.96	0.96	0.96
ECG+ Tidal Volume	0.96	0.96	0.96	0.96

*The first lines in each row represent the results obtained from training the model with the dataset created with the data at 4 weeks before the end point. **The second lines in each row represent the results obtained from training the model with the dataset created with the data recorded at one week before the end point. *** The third lines in each row represent the results obtained from training the model with the dataset created with the data within the last one-month data before the end point.

Heart Failure

OP-040

Timing of depression matters in heart failure: Results from a nationwide cohort

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Background and Aim: The presence of depression in heart failure (HF) has been linked to increased risk for morbi-mortality. However, depression might exist before HF or commence following the course of HF. Besides, the effect of specific antidepressants on prognosis remains unclear. In this study, we aimed to investigate the impact of different phenotypes of depression along with different classes of antidepressants on overall mortality in HF.

Methods: This subgroup analysis included data from 2,701,099 patients with HF, derived from the Nationwide Electronic Healthcare Database between January 1, 2016 and December 31, 2022. Herein, HF patients with a documented diagnosis of depression were considered along with index timing of depression diagnosis and classified as depression before index HF (DBHF group) and depression after the index diagnosis of HF (DAHF group). The main outcome was defined as all-cause mortality. Propensity score matching analysis and Cox regression analysis were employed to assess the impact of specific antidepressants on the main outcome, with adjustments made to mitigate the influence of confounding factors.

Results: Overall, HF patients with depression had greater proportion of females (60.8% vs. 47.5%) compared to HF patients without depression. Hospitalization rates (59.5% vs. 59.4%, p=0.152,) were similar, whereas, emergency department admissions during the 7-year follow-up were higher in the DBHF group compared to the DAHF group [17 (9-32) vs. 14 (7-26) days (p<0.001)]. Among 348,211 depressed HF patients with available timing data, 226,401 patients had index diagnosis of depression before the index diagnosis of HF (DBHF group) and 121,810 patients had index diagnosis of depression after the index diagnosis of HF (DAHF group) (Figure 1). Cumulative survival rate up to 7 years of follow up in HF patients on quadruple GDMT was worse in DBHF group than DAHF group and HF patients without depression [HR: 1.61 (95% CI: 1.39-1.87) vs. 1.04 (95% CI: 0.88-1.22), respectively (Figure 2)]. In the propensity matched cohort, DBHF had significantly poorer survival compared to DAHF (HR: 1.62, 95% CI: 1.60-1.64, p<0.001) (Figure 3). Specific antidepressant class effect on mortality was sought in HF patients with DBHF and DAHF subtypes. Second line adjunctive medications alone or in combination was associated with higher mortality irrespective of subtype of depression. Of note, neither SSRIs nor TCAs alone was not associated with higher mortality (Figure 4A, 4B).

Conclusions: There are prognostically distinct two phenotypes of depression in HF patients. Depression before the index diagnosis of HF portends poorer prognosis than depression after the index diagnosis of HF. Second line adjunctive medications alone or in combination with SSRIs, Benzodiazepine derivatives and TCAs are linked to higher mortality irrespective of subtype of depression designating severity of treatment resistant depression per se is linked to excess mortality.

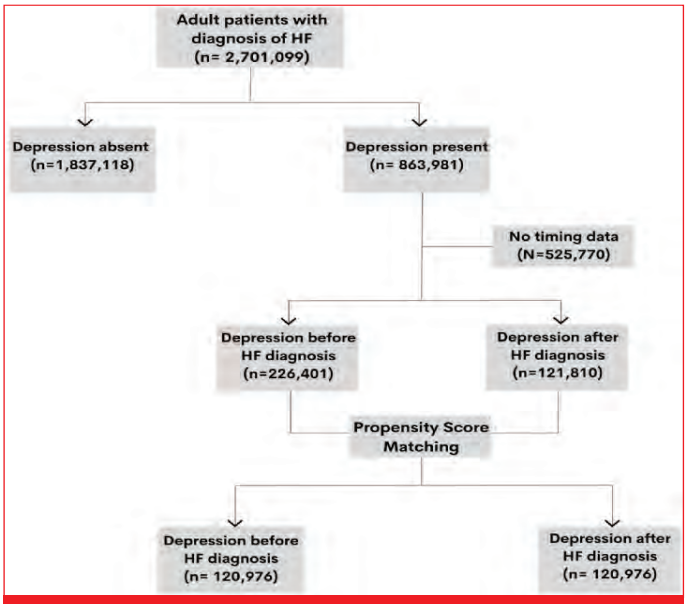

Figure 1. Flow-chart of the study population.

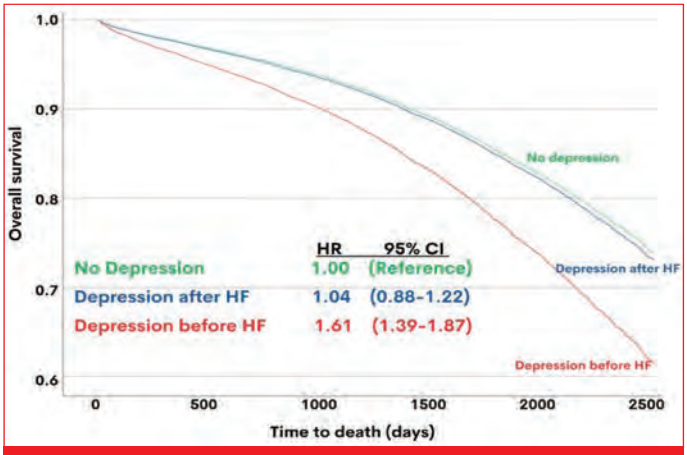

Figure 2. Survival in HF patients with DBHF versus DAHF versus no depression on quadruple guideline-directed medical therapy.

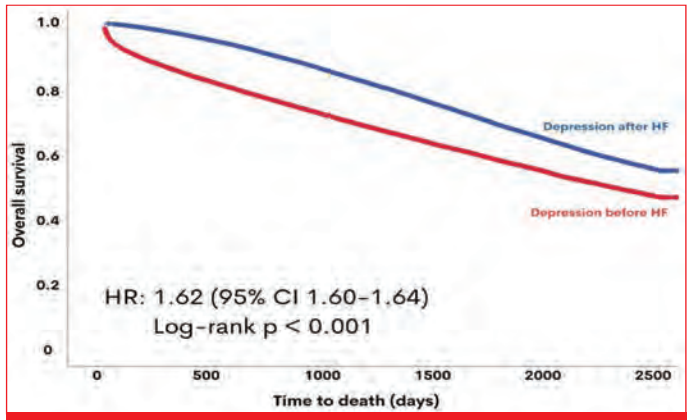

Figure 3. The overall survival rate in HF patients with DBHF versus DAHF after propensity score matching.

Table 1. Baseline characteristics of the study population according to the presence of depression diagnosis at the index date of heart failure diagnosis

	Depression absent (n= 1,837,118)	Depression present (n= 863,981)	Total population (n= 2,701,099)	p	Cohen
Age, years	69 (60-78)	71 (62-79)	70 (61-78)	<0.001	0.11
Sex, Female	872,374 (47.5)	525,681 (60.8)	1,398,055 (51.8)	<0.001	0.27
Comorbidities					
Hypertension	1,793,321 (97.6)	854,540 (98.9)	2,647,861 (98.0)	<0.001	0.10
Diabetes Mellitus	791,239 (43.1)	438,594 (50.8)	1,229,833 (45.5)	<0.001	0.15
Prior MI	408,932 (22.3)	182,974 (21.2)	591,906 (21.9)	<0.001	0.03
COPD	778,628 (42.4)	406,870 (47.1)	1,185,498 (43.9)	<0.001	0.09
Anemia	664,579 (36.2)	433,389 (50.2)	1,097,968 (40.6)	<0.001	0.28
Atrial fibrillation	674,980 (36.7)	334,673 (38.7)	1,009,653 (37.4)	<0.001	0.04
Anxiety	615,686 (33.5)	691,026 (80.0)	1,306,712 (48.4)	<0.001	0.98
Selected Laboratory Parameters					
BNP, pg/ml	878 (213-3349)	836 (205-3290)	864 (210-3328)	0.041	0.01
NT-proBNP, pg/ml	1327 (333-4691)	1291 (318-4618)	1316 (327-4665)	0.571	0.01
eGFR, (CKD-EPI)	74.7 (52.7-91.2)	71.5 (50.5-89.1)	73.7 (52.0-90.5)	<0.001	0.07
Haemoglobin, g/dL	11.7 (9.0-13.6)	11.3 (9.0-13.1)	11.5 (9.0-13.4)	<0.001	0.05
Medications					
Beta blocker	1,532,311 (83.4)	732,756 (84.8)	2,265,067 (83.9)	<0.001	0.04
RASi	949,360 (51.7)	478,291 (55.4)	1,427,651 (52.9)	<0.001	0.07
MRA	721,777 (39.3)	329,927 (38.2)	1,051,704 (38.9)	<0.001	0.02
SGLT-2i	197,269 (10.7)	101,312 (11.7)	298,581 (11.1)	<0.001	0.03
ICD	16,389 (0.9)	6,635 (0.8)	23,024 (0.9)	<0.001	0.01
CRT	6,296 (0.3)	2,594 (0.3)	8,890 (0.3)	<0.001	0.01
Any Antidepressant	608,101 (33.1)	804,961 (93.2)	1,413,062 (52.3)	<0.001	1.39
Outcomes					
Hospitalization	1,092,159 (59.4)	514,426 (59.5)	1,606,585 (59.5)	0.152	0.15
Emergency department visit	14 (7-26)	17 (9-32)	15 (7-28)	<0.001	0.13

MI, myocardial infarction; COPD, chronic obstructive pulmonary disease; BNP, brain natriuretic peptide; NT-proBNP, N-terminal pro brain natriuretic peptide; eGFR, estimated glomerular filtration rate; RASi, renin angiotensin system inhibitors; MRA, mineralocorticoid receptor antagonists; SGLT-2i, sodium glucose cotransporter-2 inhibitors; ICD, intracardiac defibrillator; CRT, cardiac resynchronization therapy.

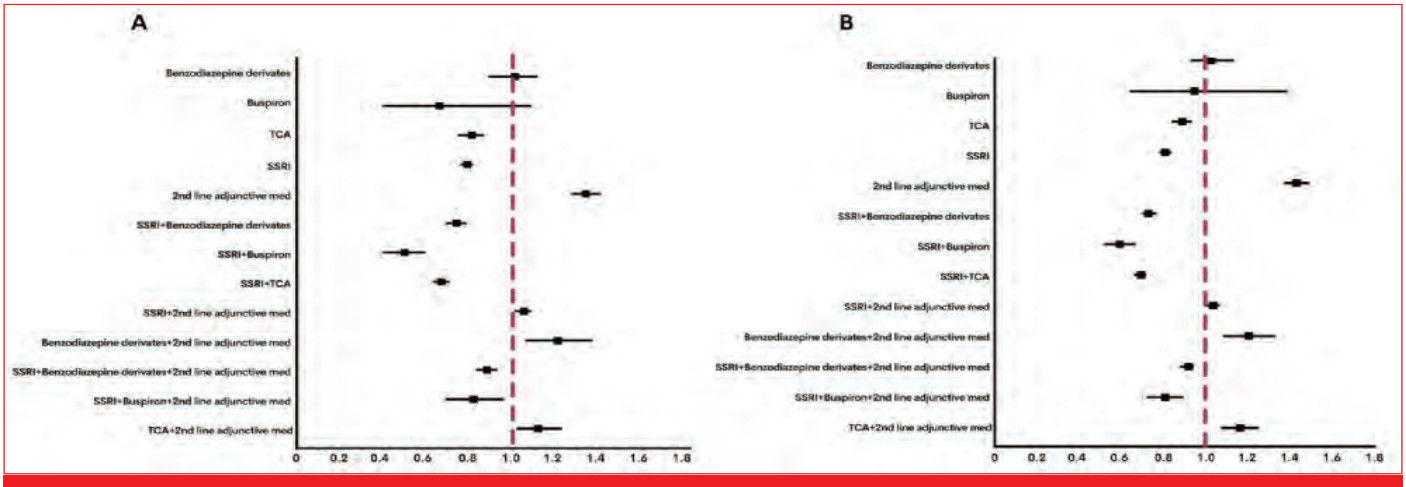

Figure 4. Overall effect of individual antidepressant classes on all-cause mortality rate in HF patients with DAHF (A) and DBHF (B), respectively.

Heart Failure

OP-041

Evaluation of soluble ST2 and galectin 3 levels in patients with heart failure

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Background and Aim: Heart failure (HF) is a clinical syndrome which is defined as a structural and/or functional abnormality of the heart resulting in increased intra-cardiac pressures and/or insufficient cardiac output. Many biomarkers have been used so far to evaluate the diagnosis and prognosis in patients with HF and their clinical importance has been demonstrated. Soluble Stromelysin-2 (sST2) and Galectin-3 have been shown to correlate with prognosis in patients with HF. However, there is no study evaluating the clinical significance of sST2 and galectin-3 in HF classification according to ejection fraction (EF). In this study, we aimed to examine the diagnostic value of sST2 and galectin-3 in HFclassification based onEF.

Methods: Forty-one HF with reduced EF (HFrEF), 41 HF with mildly reduced EF(HFmrEF), 41 HF with preserved EF (HFpEF) patients and 41 controls who applied to the cardiology outpatient clinic and whose consents were obtained were included in the study. Left ventricular EF ≤40% was defined as HFrEF, between 41% and 49% as HFmrEF, and ≥50% as HFpEF. The sST2 and Galectin-3 levels of the patients were measured and comparisons were made.

Results: A total of 164 individuals, including 123 patients diagnosed with HF (HFrEF: 41, HFmrEF: 41 and HFpEF: 41) and 41 controls, were included in the study. In terms of basal characteristics; there was a significant difference among the four groups regarding gender (p=0.003) and body mass index (BMI) (p=0.001). In post hoc analyses, the frequency of female gender and BMI were found to be significantly higher in HFpEF patients compared to HFrEF and HFmrEF groups (Table 1). In terms of laboratory parameters; significant differences were found among the groups regarding urea (p=0.014), creatinine (p<0.001), uric acid (p=0.007) and NT-pro BNP. In post hoc analyses, HFrEF patients had significantly higher urea, creatinine and uric acid levels compared to the other groups (Table 2). In addition, there was a significant difference among the groups in terms of sST2 (p<0.001) and Galectin-3 (p=0.007) (Table 3). In post hoc analyses, sST2 and galectin-3 levels were found to be significantly higher in the HFmrEF and HFrEF groups compared to the control group. However, no significant difference was found between HFpEF and the control group in terms of sST2 and galectin-3 (Figure 1 and 2). In the correlation analysis, sST2 level was positively correlated with BNP (r=0.240, p=0.002), whereas negatively correlated with EF (r=−0.403, p<0.001). In addition, galectin-3 level was positively correlated with BNP (r=0.172, p=0.028), whereas negatively correlated with EF (r=−0.295, p<0.001) (Figure 3).

Conclusions: sST2 and galectin-3 levels were significantly higher in patients with HFrEF and HFmrEF, and the blood levels of these markers increase further as EF decreases. Both biomarkers may have a role in predicting patients with HFrEF and HFmrEF. Further studies with larger participants are needed to better understand the diagnostic importance of these biomarkers.

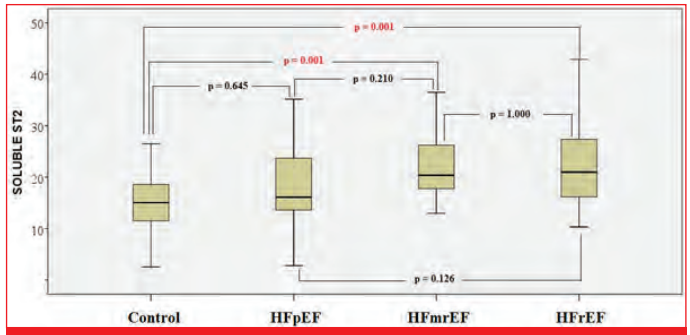

Figure 1. Comparison of soluble ST2 among the heart failure subtypes and control group.

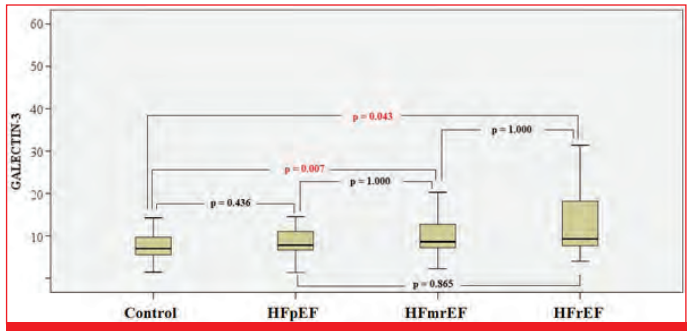

Figure 2. Comparison of galectin-3 among the heart failure subtypes and control group.

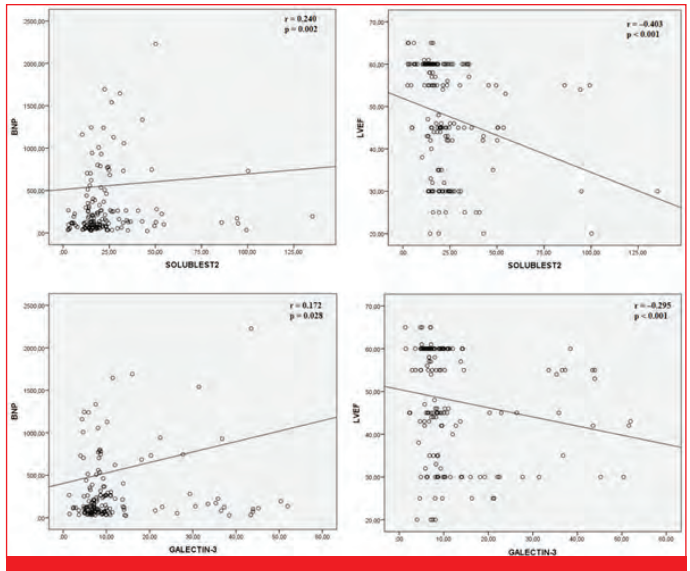

Figure 3. Correlation of sST2 and galectin-3 with BNP and LVEF.

Table 1. Comparison of baseline characteristics of heart failure subtypes and control patients

	Control (n=41)	HFpEF (n=41)	HFmrEF (n=41)	HFrEF (n=41)	p
Age, year	56.6 ± 8.9	56.6 ± 9.6	56.6 ± 12.6	58.6 ± 14.8	0.831
Sex, female (%)	20 (48.8)	26 (63.4)	10 (24.4)	15 (36.6)	0.003
BMI, kg/m^2	29.4 ± 4.4	32.2 ± 6.7	28.3 ± 3.7	27.9 ± 4.6	0.001
SBP (mmHg)	125.9 ± 23.9	127.3 ± 20.1	123.4 ± 25.0	117.2 ± 20.5	0.192
DBP (mmHg)	69.0 ± 15.3	70.3 ± 12.2	69.8 ± 12.9	68.8 ± 13.3	0.955
HT (%)	17 (41.5)	24 (58.5)	24 (58.5)	18 (43.9)	0.244
DM (%)	12 (29.3)	12 (29.3)	7 (17.1)	8 (19.5)	0.425
HL (%)	7 (17.1)	9 (22)	10 (24.4)	2 (4.9)	0.088
Rhythm (%) SR (%) AF (%)	 41 (100) 0 (0)	 41 (100) 0 (0)	 37 (90.2) 4 (9.8)	 36 (87.8) 5 (12.2)	0.021
LBBB (%)	2 (4.9)	1 (2.4)	6 (14.6)	27 (65.9)	<0.001
LVEF (%)	60.0 ± 2.2	57.7 ± 2.7	44.3 ± 1.5	29.2 ± 4.2	<0.001
LVDSÇ (cm)	4.8 ± 0.4	5.0 ± 0.5	5.1 ± 0.5	6.2 ± 0.7	<0.001
LVSSÇ (cm)	3.3 ± 0.2	3.5 ± 0.4	3.9 ± 0.4	5.1 ± 0.6	<0.001
IVS (cm)	0.9 ±0.1	1.0 ± 0.3	1.0 ± 0.1	0.9 ± 0.2	0.042
LA (cm)	3.4 ± 0.2	3.7 ± 0.3	3.6 ± 0.5	4.1 ± 0.5	<0.001
Ascending aorta (cm)	3.4 ± 0.3	3.6 ± 0.4	3.4 ± 0.3	3.6 ± 0.4	0.104
RV (cm)	3.5 ± 0.2	3.5 ± 0.2	3.6 ± 0.3	3.7 ± 0.5	0.026
E/e'	7.0 ± 0.9	14.6 ± 0.9	12.0 ± 2.1	15.9 ± 1.0	<0.001

BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HT: Hypertension; DM: Diabetes mellitus; HL: Hyperlipidemia; SR: Sinus rhythm; AF: Atrial fibrillation; LBBB: Left bundle branch block, LVEF: Left ventricular ejection fraction; LVDSÇ: Left ventricular end diastol diamater; LVSSÇ: Left ventricular end sistol diamater; IVS: Interventricular septum; LA: Left atrium; RV: Right ventricle.

Table 2. Comparison of laboratory characteristics of heart failure subtypes and control patients

	Control (n=41)	HFpEF (n=41)	HFmrEF (n=41)	HFrEF (n=41)	p
Urea (mg/dL)	32.1 (24.6-38.5)	29.9 (26.3-38.5)	29.9 (25.6-36.3)	38.5 (29.9-52.4)	0.014
Creatinine (mg/dL)	0.79 ± 0.14	0.8 ± 0.19	0.89 ± 0.19	0.95 ± 0.20	<0.001
Glucose (mg/dL)	112 (93.5-135.0)	103 (92.5-130.5)	100 (93.5-125.5)	103 (90.5-130.0)	0.667
Uric acid (mg/dL)	4.9 ± 1.2	5.3 ± 1.4	5.3 ± 1.4	6.0 ± 1.5	0.007
Sodium (mmol/L)	139.9 ± 3.0	140 ± 2.1	139.9 ± 2.1	140.1 ± 2.0	0.976
Potassium (mmol/L)	4.3 ± 0.3	4.4 ± 0.3	4.3 ± 0.4	4.3 ± 0.4	0.942
Albumin (g/dL)	4.4 ± 0.2	4.4 ± 0.2	4.3 ± 0.3	4.3 ± 0.2	0.584
LDL-Cholesterol (mg/dl)	101.8 (83.0-132.2)	103 (81.3-131.3)	91 (72.4-103.8)	105.8 (73.1-123.6)	0.104
TSH (uIU/dL)	1.3 (0.9-1.6)	1.3 (0.9-2.2)	1.1 (0.7-1.7)	1.0 (0.7-2.0)	0.255
CRP (mg/dL)	0.4 (0.1-0.6)	0.7 (0.2-1.1)	0.3 (0.1-0.8)	0.5 (0.2-1.0)	0.105
WBC (x10^3/uL)	8.4 (6.7-10.8)	7.6 (6.6-9.2)	7.7 (6.4-9.7)	8.6 (7.4-9.9)	0.188
Hematocrit (%)	41.9 ± 5.5	42.6 ± 4.1	43.7 ± 3.9	43.2 ± 4.9	0.329
Hemoglobin (g/dL)	13.4 ± 1.8	14.1 ± 1.5	14.2 ± 1.6	14.1 ± 1.6	0.148
Platelet (x10^3/uL)	288 (236.0-331.5)	275 (217.0-344.0)	245 (211.5-327.5)	255 (232.0-312.5)	0.254
NT pro-BNP (pq/ml)	88.8 (53.2-167.0)	85.8 (50.4-162.5)	138 (105.0-510.0)	769 (321.0-1289.5)	<0.001

LDL: Low density lipoprotein; TSH: Thyroid stimulating hormone; CRP: C-Reactive protein; WBC: White blood cell.

Table 3. Comparison of sST2 and Galectin-3 levels ofheart failure subtypes and control patients

	Control (n=41)	HFpEF (n=41)	HFmrEF (n=41)	HFrEF (n=41)	p
Soluble ST2, ng/L	15.0 (9.6-20.2)	16.1 (13.5-26.9)	20.3 (17.1-26.6)	20.9 (16.1-28.4)	<0.001
Galectin-3, ng/mL	7.0 (5.4-9.7)	7.8 (6.4-12.4)	8.6 (6.9-13.2)	9.2 (7.5-18.6)	0.007

Cardiac Imaging / Echocardiography

OP-042

A predictive model for differentiating causes of elevated mechanical prosthetic aortic valve gradient

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Background and Aim: Evaluating aortic prosthetic valves (APV) is often challenging, and multimodality assessment is most beneficial for these patient groups. In the presence of high-gradient aortic prosthetic valves, the use of transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), cinefluoroscopic assessment, and computed tomography (CT) is a requirement. The diagnosis in the type of obstruction is mostly made using CT as guidelines recommended. However, CT usage for aortic prosthetic valves requires experience and is not always readily accessible. In this study, we aimed to investigate the diagnostic value of easily accessible parameters, such as echocardiography and cinefluoroscopy, in the differential diagnosis of patients with high-gradient APVs.

Methods: Patients evaluated for the presence of high-gradient APVs between February 2020 and April 2024 were included in our study. In addition to their clinical and laboratory findings, standard TTE evaluation and cinefluoroscopy were routinely performed, with TEE and CT used as needed by the physician. Following this diagnostic assessment, patients were categorized into three diagnostic groups: non-obstructive (mismatch) and obstructive conditions (pannus or thrombus). Patients for whom optimal cinefluoroscopic evaluation could not be performed and those with detected subaortic membrane were excluded from the study.

Results: A total of 159 patients with high-gradient aortic stenosis were included in the study (median age: 52.0 years, 46.5% male). Of these, 102 had mismatch, 22 had thrombus, and 35 had pannus. The mismatch group exhibited significantly shorter acceleration times and lower indexed EOA compared to the thrombus and pannus groups. The PHV opening angle was also significantly lower in the mismatch group. The number of effective in time in therapeutic range (TTR) was numerically lower in the thrombus group. A multivariate multinomial logistic regression model was employed to illustrate the predicted probabilities of the diagnostic groups. According to partial chi-square values, the importance of variables in the model ranked as follows: PHV opening angle, AT, age of PHV (>5 years), size of PHV, and effective TTR. When the PHV age was less than 5 years, there was no apparent relationship between the opening angle and the probability of pannus, whereas the probability of mismatch decreased with increasing opening angle, regardless of PHV age. As the opening angle increased, the probability of thrombus tended to increase in both PHV age groups, but this trend was more pronounced when PHV age was less than 5 years.

Conclusions: In this study, we developed a clinical prediction model with good performance using 5 variables in patients with high-gradient aortic prosthetic valve disease which will be beneficial even CT is not present.

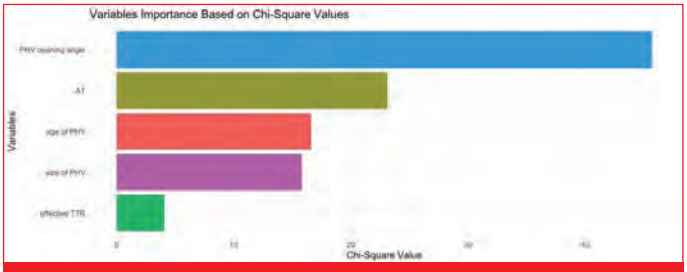

Figure 1. Variables Importance Based on Chi-Square Values.

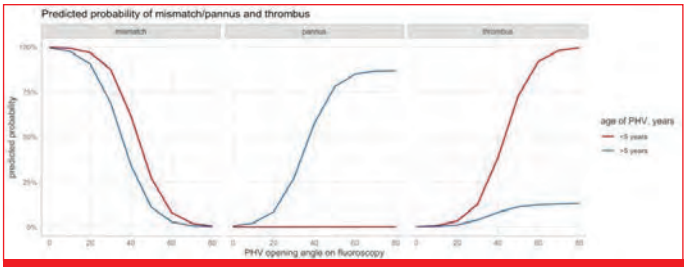

Figure 2. The predictive probability of diagnostic groups as a function of PHV opening angle, stratified by PHV age.

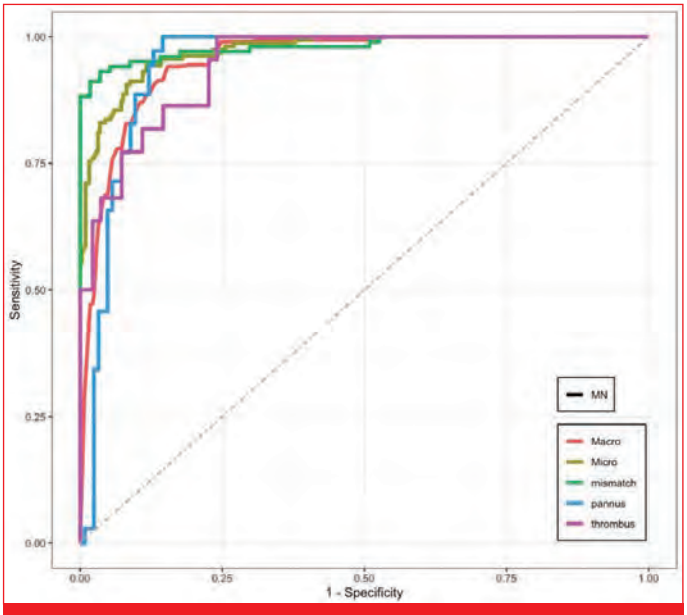

Figure 3. The model's macro-average multi-class plots averaging all diagnostic groups against the rest.

Table 1. Baseline characteristics according to etiology (mismatch, thrombus and pannus) of high aortic gradient

Characteristic	Mismatch, N = 102	Thrombus, N = 22	Pannus, N = 35	p-value
Sex, male	54 (53%)	9 (41%)	11 (31%)	0.075
Age, years	52 (42, 62)	52 (47, 61)	54 (44, 68)	0.547
AF, %	31 (32%)	8 (36%)	11 (33%)	0.909
Heart rate, beat/min	72 (66, 85)	74 (68, 86)	74 (66, 80)	0.623
Hemoglobin	12.0 (9.90, 13.2)	12.1 (9.93, 13.6)	12.5 (10.0, 13.6)	0.735
Platelet count	220 (193, 275)	217 (187, 250)	226 (178, 280)	0.899
C-reactive protein	4 (3, 13)	4 (3, 10)	3 (2, 6)	0.286
Creatinine	0.80 (0.68, 1.00)	0.89 (0.68, 1.06)	0.85 (0.67, 1.02)	0.916
Estimated GFR	97 (74, 111)	97 (73, 110)	83 (61, 107)	0.318
Valve type				0.187
AVR	44 (43%)	12 (55%)	11 (31%)	
AVR+MVR	57 (56%)	9 (41%)	23 (66%)	
Other	1 (1.0%)	1 (4.5%)	1 (2.9%)	
REDO operation	5 (4.9%)	1 (4.5%)	0 (0%)	0.463
StJude valve	58 (62%)	11 (52%)	14 (41%)	0.112
Bileaflet valve	99 (97%)	22 (100%)	32 (91%)	0.239
Brand name				0.021
ATS	17 (17%)	10 (45%)	12 (35%)	
Carbomedics	12 (12%)	1 (4.5%)	5 (15%)	
Monoleaflet	3 (3.0%)	0 (0%)	3 (8.8%)	
Sorin	8 (7.9%)	0 (0%)	0 (0%)	
St Jude	61 (60%)	11 (50%)	14 (41%)	
PHVsize (no)				0.002
19	41 (46%)	5 (24%)	9 (29%)	
21	40 (45%)	6 (29%)	15 (48%)	
23	8 (9.0%)	8 (38%)	6 (19%)	
25	0 (0%)	2 (9.5%)	1 (3.2%)	
Age of PHV	12 (5, 16)	6 (3, 14)	11 (9, 16)	0.050
LVEF	60 (50, 60)	55 (55, 60)	60 (55, 60)	0.549
V max	3.60 (3.18, 3.94)	3.56 (3.08, 4.21)	3.61 (3.45, 4.35)	0.332
Mean gradient	31 (25, 37)	32 (24, 50)	34 (29, 50)	0.075
Acceleration time	77 (70, 85)	100 (90, 109)	92 (86, 102)	<0.001
DVI	0.33 (0.30, 0.37)	0.24 (0.21, 0.25)	0.24 (0.22, 0.26)	<0.001
EOA	1.27 (1.18, 1.35)	1.60 (1.47, 1.67)	1.43 (1.30, 1.59)	<0.001
BSA	1.82 (1.70, 1.93)	1.82 (1.65, 1.92)	1.70 (1.60, 1.80)	<0.001
Indexed EOA	0.70 (0.64, 0.77)	0.87 (0.82, 0.91)	0.86 (0.79, 0.91)	<0.001
Transvalvular AR	8 (7.8%)	3 (14%)	2 (5.7%)	0.570
Paravalvular AR	12 (12%)	4 (18%)	5 (14%)	0.615
MVR	58 (57%)	10 (45%)	25 (71%)	0.131
TVR	1 (1.0%)	0 (0%)	1 (2.9%)	0.590
Effective TTR	49 (48%)	6 (27%)	16 (46%)	0.204
Impaired fluoroscopy angle	6 (5.9%)	22 (100%)	35 (100%)	<0.001
PHV opening angle	15 (13, 20)	43 (37, 56)	45 (39, 50)	<0.001

AF: Atrial fibrillation, AR: Aortic regurgitation, AVR: Aortic valve replacement, BSA: Body mass index, DVI: Dimensional velocity index, EOA: Effective orifice area, GFR: Glomeral filtration rate, LVEF: Left ventricular ejection fraction,MVR: Mitral valve replacement, PHV: Prosthetic heart valve, TTR: Time in therapeutic range

Table 2. Multinomial logistic regression estimates to predict the etiology (mismatch, thrombus and pannus) of high aortic gradient

Variable	Regression coefficient (β), 95% CI	p value
Pannus vs Mismatch		
Intercept	-41.7	
PHV opening angle on fluoroscopy°	0.14 (0.08, 0.21)	<0.001
Acceleration time (msec)	0.22 (0.08, 0.37)	0.003
Size of PHV (mm)	0.47 (-0.01, 0.95)	0.055
Age of PHV>5 year (yes)	8.31 (-2.26, 18.9)	0.123
Presence of effective TTR (yes)	-1.21 (-2.81, 0.39)	0.137
Thrombus vs Mismatch		
Intercept	-52.5	
PHV opening angle on fluoroscopy°	0.15 (0.07, 0.22)	<0.001
Acceleration time (msec)	0.30 (0.14, 0.46)	<0.001
Size of PHV (mm)	1.05 (0.46, 1.64)	<0.001
Age of PHV>5 year (yes)	-0.97 (-3.43, 1.50)	0.442
Presence of effective TTR (yes)	-1.96 (-3.96, 0.05)	0.055

Cardiac Imaging / Echocardiography

OP-043

Exploring the heart's shape: The sphericity index in myocarditis patients and its prognostic significance

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Background and Aim: Myocarditis, a leading cause of non-ischemic dilated cardiomyopathy, has increased in prevalence during the COVID-19 pandemic. Although imaging is vital for diagnosing myocarditis, its prognostic value is not well understood. The sphericity index, which assesses the roundness of the left ventricle (LV), may help in both diagnosis and prognosis of various diseases. This study examines the prognostic significance of the sphericity index in myocarditis patients.

Methods: We analyzed clinical and imaging data from 30 myocarditis patients diagnosed using the Revised Lake Louise criteria and a control group of 20 age- and gender-matched individuals. The primary outcomes were all-cause mortality, sudden cardiac death, heart failure hospitalization, and ventricular arrhythmia. We compared cardiac magnetic resonance (CMR) and transthoracic echocardiography (TTE) parameters, including the CMR-derived sphericity index, between groups and used regression analysis to identify factors influencing outcomes.

Results: The mean age was 39.28 ± 15.2 years, with 72% of patients being male. LV ejection fraction (EF) values from TTE and CMR were significantly lower in the primary outcome group, while the sphericity index was significantly higher (0.51 ± 0.1 vs. 0. 3 ± 0.1 and 0.39 ± 0.1, p value 0.001). The sphericity index showed good intra- and interobserver reliability (ICC 0.95 and 0.88, respectively) and sphericity index >0.4 predicted primary outcomes with 88.89% sensitivity and 71.43% specificity (AUC 0.825, p<0.001). Regression analysis identified the sphericity index as a significant prognostic factor (OR: 6.987, p=0.012).

Conclusıons: The CMR-derived sphericity index is a straightforward imaging parameter that plays a crucial role in predicting the prognosis of myocarditis.

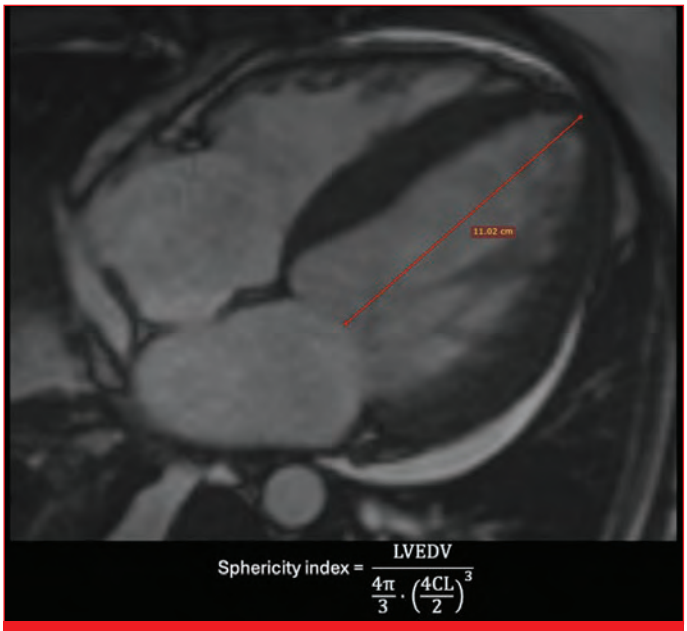

Figure 1. Measurement of sphericity index: LVEDV- left ventricular end-diastolic volume, 4CL-4 chamber length (red line).

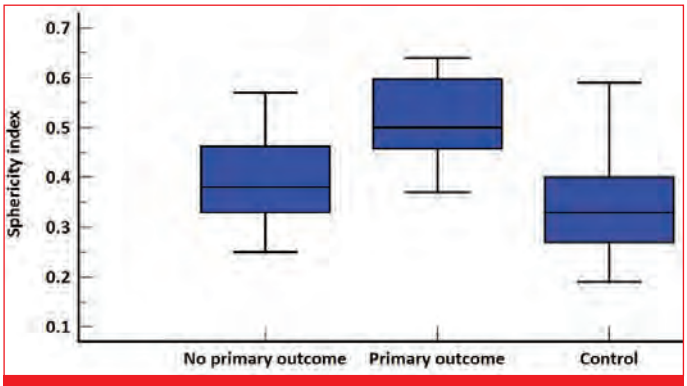

Figure 2. Comparison of sphericity index in patients with and without primary outcome, and controls.

Cardiac Imaging / Echocardiography

OP-044

Evaluation of left atrial function in patients treated with electrical cardioversion and radiofrequency catheter ablation for atrial fibrillation rhythm control by two-dimensional speckle tracking echocardiography

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Background and Aim: The effects of radiofrequency catheter ablation (RFCA) on left atrial (LA) mechanical function are not fully understood. Some previous studies have suggested that thermal modalities such as RF cause coagulation necrosis including edema, intramural hemorrhage and microvascular damage, and in the chronic phase, these lesions transform into areas of reparative fibrosis, leading to increased scar tissue and decreased compliance after ablation. The effect of sinus rhythm restoration on left atrial reservoir strain is not clearly known in patient with atrial fibrillation (AF). In this study, we aimed to compare the changes in myocardial deformation after catheter ablation and electrical cardioversion (DCCV) procedures for rhythm control in patients with atrial fibrillation and to investigate whether the ablation procedure decreases atrial reservoir function.

Methods: In this study, we retrospectively analyzed echocardiographic parameters of left ventricular and left atrial function and strain measurements with two-dimensional STE before and after the procedure in 94 patients who underwent catheter ablation or cardioversion for atrial fibrillation between November 2022 and May 2024. Patients with a left ventricular EF ≥50% and a suitable image for LA strain were included in the study. Patients younger than 18 years of age, AF recurrence in the first 3 months, left ventricular EF <50%, history of mechanical mitral valve replacement, mitral stenosis, mitral regurgitation grade 2 or higher were excluded. The procedure performed on the patients was not known by the cardiologist performing the echocardiographic evaluation.

Results: Our study was performed in 45 DCCV patients and 49 catheter ablation patients who met the inclusion criteria. LA reservoir strain (LARS) significantly improved after the procedure in the ablation group (4 chamber LARS 15.1 ± 8.2, 19.6 ± 7.1 p<0.001, respectively). The mean LARS value also showed a significant improvement after the procedure compared to the pre-procedure in patients who underwent DCCV (4 chamber LARS 12.2 ± 6.2, 17.3 ± 8.1 p<0.001, respectively). There was no significant difference in strain change between the groups (p=0.7).

Conclusions: In our study, the improvement in the reservoir strain of patients who underwent RFCA was similar with DCCV group. These findings suggest that restoration of sinus rhythm by RFCA at the expense of fibrosis in the lesion areas leads to improvement in left atrial reservoir function.

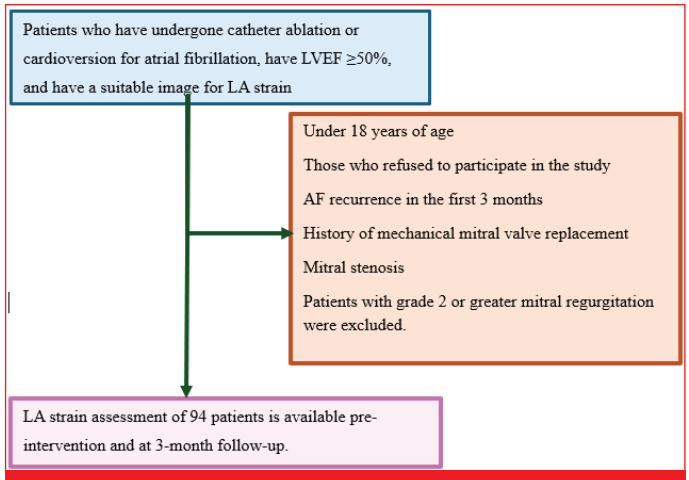

Figure 1. Participant involvement flowchart.

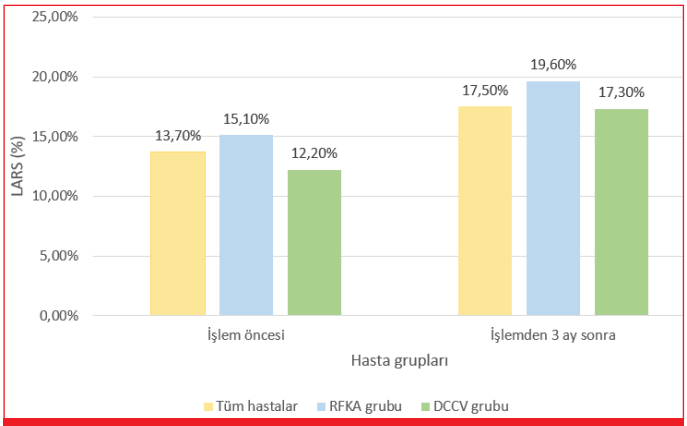

Figure 2. Change in 4-chamber LARS value of the groups before and after the procedure.

Table 1. Comparison of change in baseline and 3-month measurements in patients undergoing DCCV and RFCA

İşlem tipine göre iki grup degişkenleri		İşlem öncesi ölçümler	3. ay ölçümleri	P değeri
DCCV	LAD (mm)	46.3±6.0	45.1±6.7	0.33
RFKA		44.6±8.2	41.1±12.1	
DCCV	LA alanı (cm^2)	22.8±4.8	22.4±5.3	0.3
RFKA		21,8±5,7	20.8±5.4	
DCCV	LAVİ (ml/m^2)	32.7±11.1	32.5±10.9	0.7
RFKA		32±9.3	30.4±9.5	
DCCV	LAVmax (ml/m^2)	62.6±20.6	62.3±20.6	0.6
RFKA		62±20	58.6±20	
DCCV	LVEDD (mm)	50.3±6	50.2±6.12	0.62
RFKA		49.3±6.06	48.3±8.8	
DCCV	LVESD (mm)	33.8±6.2	34.2±7.5	0.6
RFKA		34±7.5	33.3±7.8	
DCCV	LVEF (%)	57.8±7	72±84.6	0.3
RFKA		59.3±6.2	60.3±5.2	
DCCV	TAPSE (cm)	2.3±1.9	2.01±0.4	0.3
RFKA		2.0±0.33	2.1±0.3	
DCCV	TRvmax (m/sn)	3.2±3.3	2.6±0.5	0.7
RFKA		2.6±0.5	2.5±0.5	
DCCV	SPAB (mmHg)	37.8±13.3	36.2±10	0.58
RFKA		35.2±10.7	31.8±10	
DCCV	Mitral E (m/sn)	0.84±0.24	0.80±0.22	0.32
RFKA		0.8±0.21	0.8±0.23	
DCCV	Mitral A (m/sn)	0.65±0.16	0.59±0.16	0.50
RFKA		0.69±0.19	0.66±0.2	
DCCV	Mitral E/e' ortalama	10.7±3.8	9.6±3.7	0.9
RFKA		9.7±2.5	8.8±3	
DCCV	4 Boşluk LARS (%)	12.2±6.2	17.3±8.1	0.4
RFKA		15.1±8.2	19.6±7.1	

Cardiac Imaging / Echocardiography

OP-045

Cardiovascular magnetic resonance imaging identification of different phenotypic features of hypertrophic cardiomyopathy and the evaluation of the relationship with genotype

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Background and Aim: This study aimed to identify the phenotypic features contributing to the development of left ventricular outflow tract obstruction (LVOTO) in patients with hypertrophic cardiomyopathy (HCM) and evaluate the genotype-phenotype relationship.

Methods: This cross-sectional study included 96 patients diagnosed with HCM (mean age: 56.9 ± 13.5 years, 32.3% female). Patients were divided into the hypertrophic non-obstructive cardiomyopathy (HNCM; n=60) and hypertrophic obstructive cardiomyopathy (HOCM; n=36) groups. All patients underwent cardiovascular magnetic resonance imaging. Patients (n=77) who had previously provided formal approval underwent genetic examination that included 18 genes. Patients who underwent genetic testing were categorized into the variant-negative (n=48), variant of unknown significance (VUS; n=13), and likely pathogenic/pathogenic variant (LP/P; n=16) groups.

Results: The basal interventricular septum (IVS) thickness and anterior mitral leaflet (AML) and posterior mitral leaflet (PML) lengths were higher and the LVOT diameter and ventricular arterial coupling were lower in the HOCM group. Mitral regurgitation, residual leaflets, abnormal chordal attachment, and bifid papillary muscle (PM) were observed more frequently in the HOCM group. The anterolateral (AL) PM-septum distances measured at the end of diastole and systole were lower in the HOCM group, whereas the AL-PM mobility calculated using these parameters was higher in the HOCM group. In the multivariate logistic regression model, the AML length/LVOT diameter ratio, PML length/LVOT diameter ratio, and AL-PM mobility were associated with LVOTO, independent of the basal IVS thickness, abnormal chordal attachment, and bifid PM. An AML length/LVOT diameter ratio of ≥2.30, PML length/LVOT diameter ratio of ≥1.83, and AL-PM mobility of ≥57.7% were predictors of LVOTO, with good sensitivity and specificity. Positive variants (VUS, LP, and P) were found in 37.7% (29 of 77) of patients who underwent genetic testing. The LP/P variant was detected in 20.8% (16 of 77) of patients, which enabled confirmation of the final molecular diagnosis. The most commonly identified genes were MYBPC3 and MYH7. The three groups had significant differences in the LVOT diameter, AML length, AML length/LVOT diameter ratio, PML length/LVOT diameter ratio, and abnormal chordal attachment.

Conclusions: The AML length/LVOT diameter ratio, PML length/LVOT diameter ratio, and AL-PM mobility were associated with LVOTO, independent of other phenotypic changes, especially the basal IVS thickness. The rate of positive variants in patients with HCM in our study population was lower than that in other populations, and our data were compatible with those of other studies conducted in the Turkish population. Genetic testing results may affect the phenotypic expression of patients with HCM; however, to make an ultimate decision regarding this situation, further comprehensive research is needed.

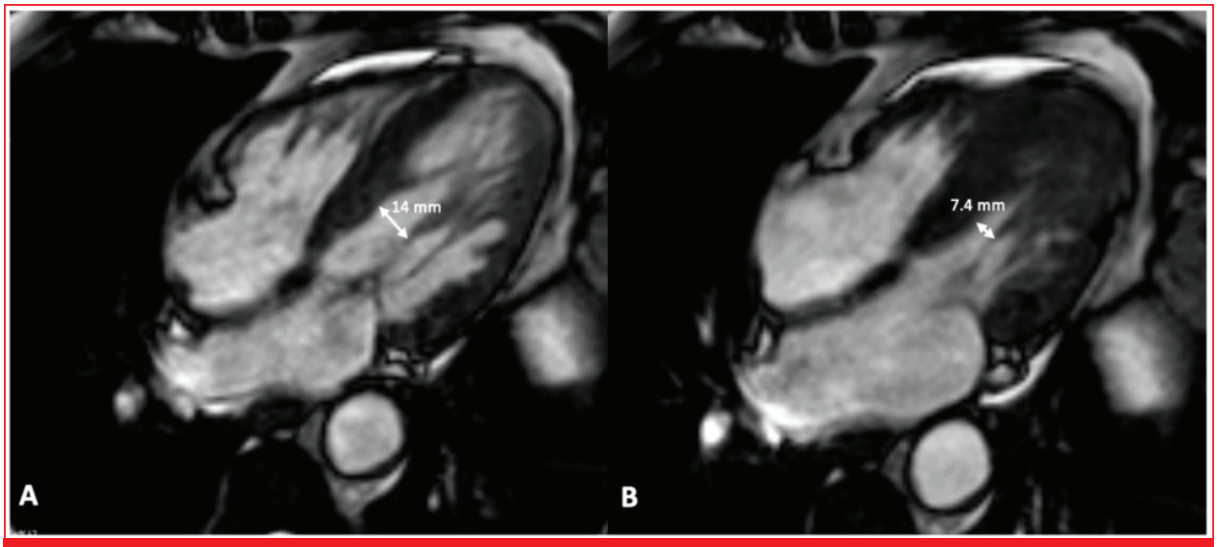

Figure 1. Assessment of AL-PM mobility. A) Distance of AL-PM to septum at end-diastole, B) Distance of AL-PM to septum at end-systole.

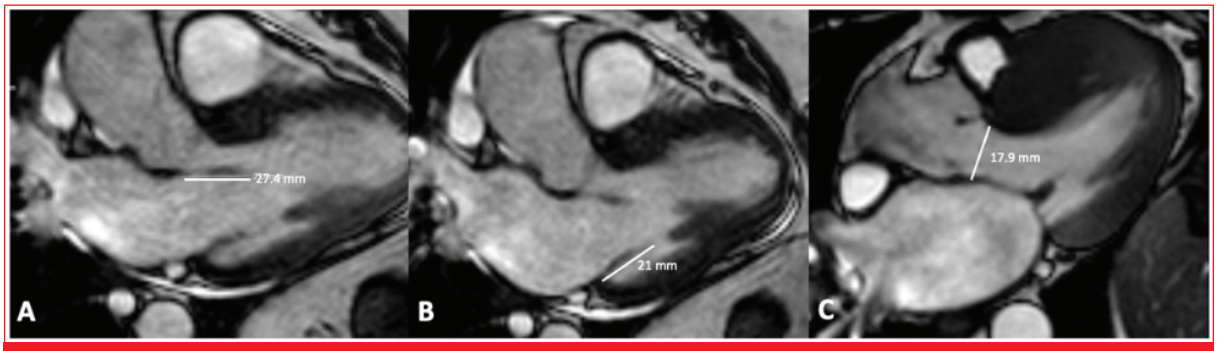

Figure 2. Measurement of mitral valve leaflet lengths and LVOT diameter. A) AML length, B) PML length, C) LVOT diameter.

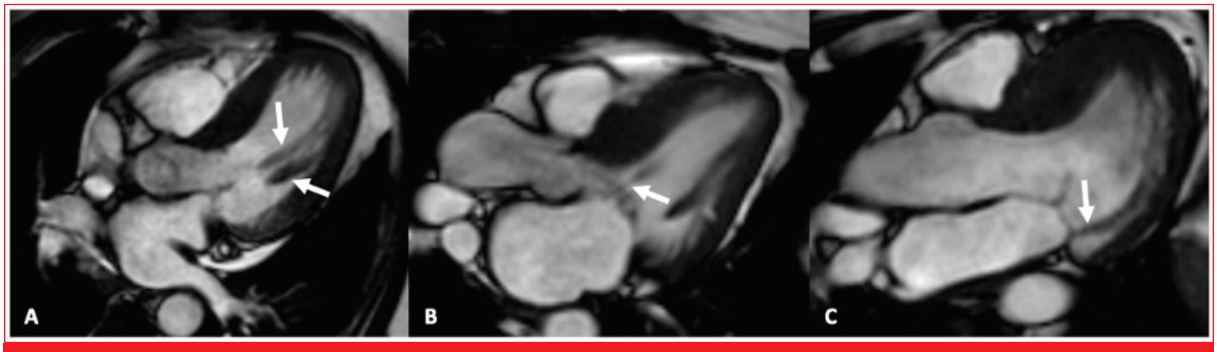

Figure 3. Changes in the submitral apparatus. A) Bifid PM, B) Abnormal chordal attachment of AL-PM to AML, C) Direct posteromedial PM insertion into the PML.

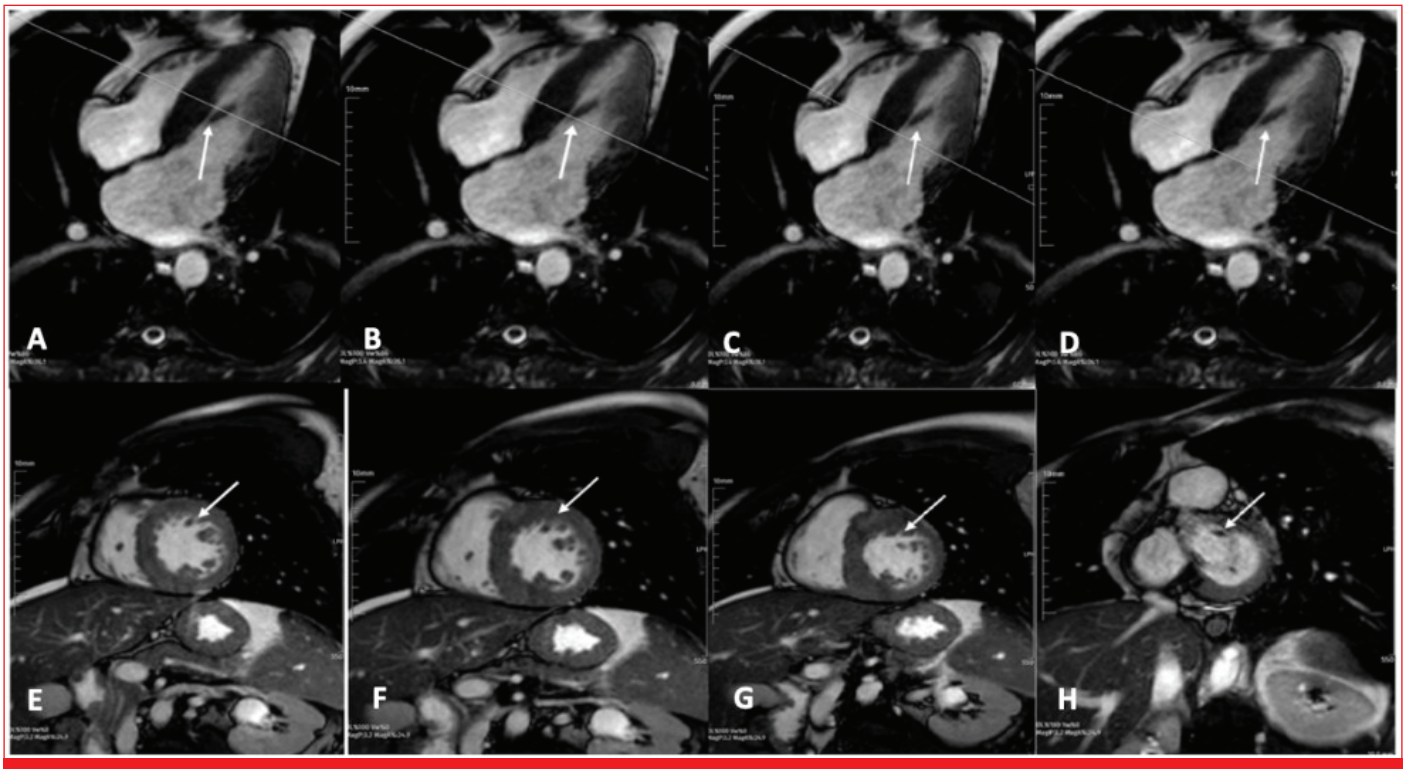

Figure 4. Identification of the accessory apical-basal muscle bundle in the four-chamber (A, B, C, D) and short axis views (E, F, G, H).

Table 1. Multivariable logistic regression model for predicting the presence of LVOTO (Model 1)

Variables	p-value	Odds Ratio	95% CI
AML length/LVOT diameter	**<0.001**	4.739	2.007–11.192
AL-PM mobility	**0.016**	1.037	1.007–1.068
Basal IVS thickness	0.169	1.112	0.956–1.293
Abnormal chordal attachment	0.173	3.324	0.591–18.708
Bifid PM	0.452	2.019	0.324–12.577

AML: anterior mitral valve; AL-PM: anterolateral papillary muscle; IVS: interventricular septum; LVOT: left ventricular outflow tract; LVOTO: left ventricular outflow tract obstruction; PM: papillary muscle

Table 2. Multivariable logistic regression model for predicting the presence of LVOTO (Model 2)

Variables	p-value	Odds Ratio	95% CI
PML length/LVOT diameter	**<0.001**	11.321	3.237–39.594
AL-PM mobility	**0.019**	1.037	1.006–1.068
Basal IVS thickness	0.174	1.119	0.951–1.317
Abnormal chordal attachment	0.086	5.173	0.793–33.747
Bifid PM	0.513	1.924	0.271–13.682

AML: anterior mitral valve; AL-PM: anterolateral papillary muscle; IVS: interventricular septum; LVOT: left ventricular outflow tract; LVOTO: left ventricular outflow tract obstruction; PM: papillary muscle

Table 3. Genetic distribution according to gene and variant pathogenicity

HCM Variant<	VUS	LP/P	Total (VUS/LP/P)
MYBPC3	3	8	11 (34.4%)
MYH7	2	6	8 (25%)
MYL2	1	0	1 (3.1%)
MYL3	1	0	1 (3.1%)
TPM1	0	1	1 (3.1%)
TNNI3	1	0	1 (3.1%)
TNNT2	0	1	1 (3.1%)
TNNC1	1	0	1 (3.1%)
ACTC1	0	1	1 (3.1%)
VCL	1	0	1 (3.1%)
HCM Phenocopies			
FLNC	4	0	4 (12.5%)
PRKAG2	1	0	1 (3.1%)
Total	15 (47%)	17 (53%)	32 (100%)

HCM: hypertrophic cardiomyopathy; LP: likely pathogenic; P: pathogenic; VUS: variants of uncertain significance

Cardiac Imaging / Echocardiography

OP-046

Optimization and accurate projections in cinefluoroscopic evaluation of mechanical prosthetic valves

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Background and Aim: The accurate evaluation of mechanical prosthetic heart valves (PHV) is critical in ensuring their proper function and longevity. Cinefluoroscopy, a dynamic imaging technique, plays a vital role in assessing these prosthetic devices by providing real-time visualization of their motion and functionality. However, the quality and diagnostic value of cinefluoroscopic imaging are highly dependent on the optimization of the roentgen tube angles and the accuracy of the projections used. This study aims to explore and refine these technical parameters to enhance the precision and reliability of cinefluoroscopic evaluations of mechanical prosthetic valves, thereby contributing to better patient outcomes and more effective clinical practices.

Methods: In our study, patients with aortic and mitral PHVs, who were evaluated using cinefluoroscopy between October 2021 and June 2024, were assessed. The method for optimal evaluation is illustrated in Figure 1, while the determination of opening and closing angles is shown in Figure 2. The manufacturer and type of PHV were recorded from hospital records. Two experienced cardiologists conducted the cinefluoroscopic evaluation of the patients. Various tube projections were attempted until an optimal image was obtained. Patients were positioned supine, and in right and left lateral decubitus positions to achieve the best image. If optimal imaging could not be achieved in these three positions and four different projections, it was recorded as non-optimal.

Results: In our study, a total of 331 patients were evaluated, comprising 250 patients with aortic PHV and 81 patients with mitral PHV. The demographic characteristics of the aortic PHV patients are summarized in Table 1. The optimal projections for both mitral and aortic PHVs are shown in Figure 3. For aortic PHVs, optimal imaging was achieved in 94.8% of cases, with 87.8% of these obtained in the supine position. The most frequently achieved optimal projection for aortic PHVs was the left cranial projection. The distribution of optimal projections for aortic PHVs is summarized in Figure 4. For mitral PHVs, the rate of optimal imaging was significantly lower than for aortic PHVs, achieved in 66.7% of cases, with 61.1% of these obtained in the supine position. The demographic characteristics of mitral PHV patients are summarized in Table 2. The projection most commonly associated with optimal imaging for mitral PHVs was the right cranial projection. The distribution of projections for mitral PHVs is shown in Figure 5.

Conclusions: This study highlights the importance of optimizing tube angles and projections in cinefluoroscopic evaluation of PHVs. The findings demonstrate that optimal imaging is more easily achieved in aortic PHVs, particularly in the supine position with left cranial projection. However, mitral PHVs present greater challenges, with lower rates of optimal imaging. These results emphasize the need for tailored approaches to improve diagnostic accuracy in cinefluoroscopy.

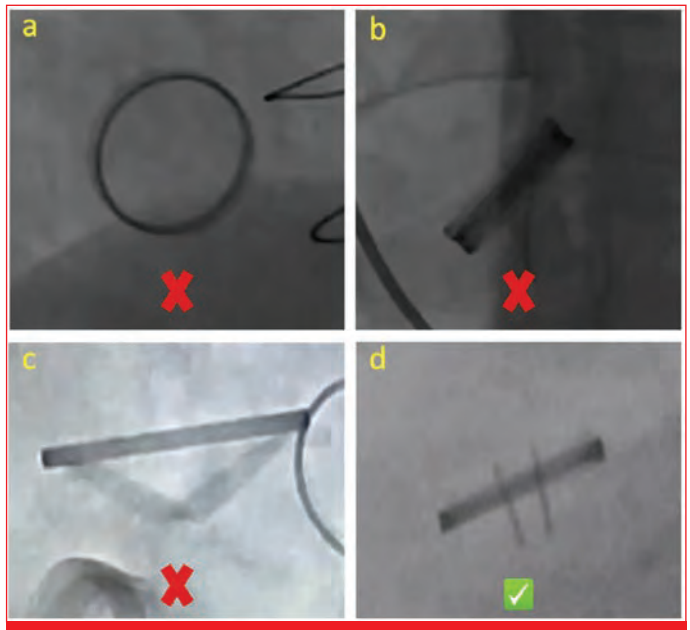

Figure 1. Appropriate and optimal cinefluoroscopic evaluation of mechanical prosthetic valves. In images a, b, and c, non-optimal views that do not allow for angle measurement are shown, whereas in image d, a perpendicular optimal view that enables angle measurement is presented.

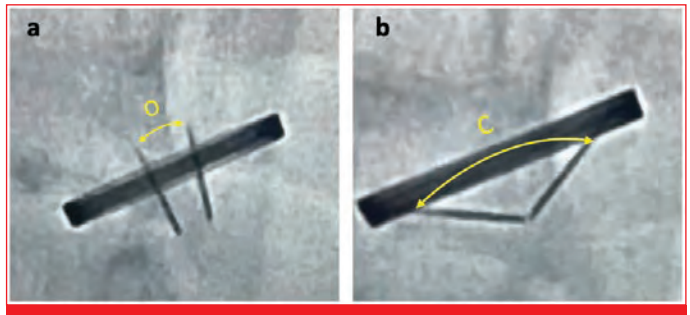

Figure 2. Demonstration of how cinefluoroscopic opening and closing angles are measured in a mechanical prosthetic valve. (O: opening angle, C: closing angle).

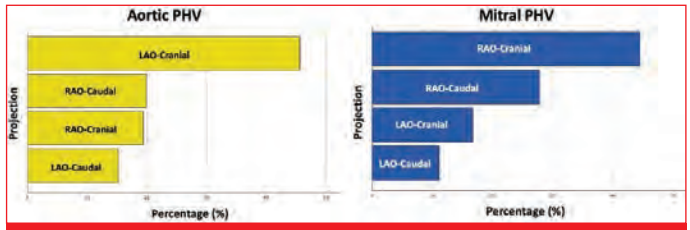

Figure 3. The optimal cinefluoroscopic projections for aortic and mitral prosthetic valve patients and their distribution within our study population are shown (PHV: prosthetic heart valve).

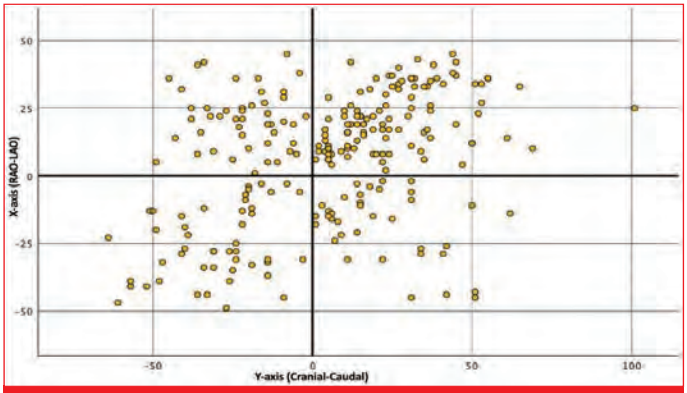

Figure 4. The distribution of optimal images obtained from various projections in patients with aortic prosthetic valves is shown.

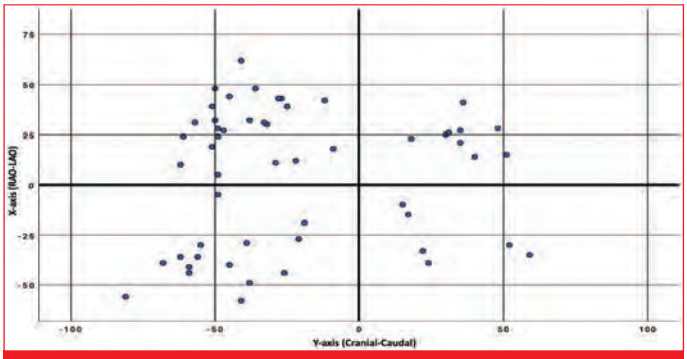

Figure 5. The distribution of optimal images obtained from various projections in patients with mitral prosthetic valves is shown.

Table 1. Baseline characteristics of aortic prosthetic heart valve patients

	Total aortic PHV patients, n=250)
Age, years	56.0 (45.0-65.0)
Male gender, n (%)	114 (45.6)
Operation type, n (%)	
AVR	119 (47.6)
AVR + MVR	125 (50)
Others	6 (2.4)
Leaflet type, n (%)	
Monoleaflet	16 (6.4)
Bileaflet	231 (93.6)
Manufacturer of aortic PHV, n (%)	
St Jude	131 (57.7)
ATS	50 (22)
Carbomedics	26 (11.5)
Sorin	10 (4.4)
Aortic PHV number	21.0 (19.0-21.0)
PHV duration, years	10.0 (5.0-16.0)
Patient position, n (%)	
Supine	208 (87.8)
Left lateral decubitus	11 (4.6)
Right lateral decubitus	18 (7.6)
Aortic PHV opening angle, degree	19.0 (13.0-37.0)
Optimal image for aortic PHV, n (%)	
yes	237 (94.8)
no	13 (5.2)
Optimal projection of aortic PHV, n (%)	
LAO-Cranial	108 (45.6)
RAO-Cranial	46 (19.4)
LAO-Caudal	36 (15.2)
RAO-Caudal	47 (19.8)

Table 2. Baseline characteristics of mitral prosthetic heart valve patients

	Total mitral PHV patients, n=81)
Age, years	57.0 (46.25-65.0)
Male gender, n (%)	39 (48.1)
Operation type, n (%)	
MVR	11 (13.6)
AVR + MVR	53 (65.4)
Others	17 (21)
Leaflet type, n (%)	
Monoleaflet	13 (16)
Bileaflet	68 (84)
Manufacturer of mitral PHV, n (%)	
St Jude	24 (46.2)
ATS	12 (23.1)
Carbomedics	9 (17.3)
Sorin	5 (9.6)
Mitral PHV number	29.0 (27.0-31.0)
PHV duration, years	14.0 (7.0-22.0)
Patient position, n (%)	
Supine	33 (61.1)
Left lateral decubitus	11 (20.4)
Right lateral decubitus	10 (18.5)
Mitral PHV opening angle, degree	18.4 (12.2-26.3)
Optimal image for mitral PHV, n (%)	
yes	54 (66.7)
no	27 (33.3)
Optimal projection of mitral PHV, n (%)	
LAO-Cranial	9 (16.7)

Cardiac Imaging / Echocardiography

OP-047

Relationship between symptom burden and myocardial fibrosis in patients with hypertrophic cardiomyopathy

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Background and Aim: In patients with hypertrophic cardiomyopathy (HCM), determining the risk of sudden cardiac death is crucial for prognosis. In addition to traditional parameters, late gadolinium enhancement (LGE) detected by cardiac magnetic resonance imaging (CMR) is often used to identify fibrosis and assess the risk of sudden cardiac death. The latest guidelines from the American Heart Association (AHA) recommend an ICD for primary prevention in HCM patients with high fibrosis (over 15% LGE) detected by CMR. However, LGE quantification is often challenging and requires specialized software to achieve precise values. Many centers have limited access to and use of these software tools. Since symptom severity is frequently correlated with disease severity, these two parameters are closely

related. This study aims to investigate the relationship between symptom burden and high fibrosis rates in HCM patients.

Methods: Patients with HCM evaluated in our center's cardiomyopathy outpatient clinic and who underwent CMR between October 2021 and May 2023 were included in the study. The symptom burden of the patients was determined using the Kansas City Cardiomyopathy Questionnaire (KCCQ). The KCCQ-12 score was obtained through a licensed provider and routinely administered to HCM patients. According to this scoring system, patients with low scores are considered to have a high symptom burden. The presence of high fibrosis was determined by analyzing post-contrast images from CMR, with evaluations conducted by two experienced radiologists. LGE of 15% or more was considered indicative of high fibrosis (Figure 1).

Results: A total of 193 patients were evaluated in our study. Demographic data are summarized in Table 1, and imaging features are summarized in Table 2. High fibrosis was detected in 57 patients. There was no difference in basic demographic characteristics between the two groups. The KCCQ-12 score was significantly lower in the high fibrosis group. Troponin and NT-proBNP levels were higher in the high fibrosis group. Patients in the high fibrosis group had lower LVEF, higher maximal wall thickness, and higher LV mass index. In logistic regression analysis, interventricular septum thickness detected on TTE, LVEF, MWT, and LV mass index detected on CMR were identified as independent predictors of high fibrosis. Additionally, the KCCQ-12 score was also identified as a significant independent predictor of high fibrosis. Table 3 shows the logistic regression analysis in predicting high fibrosis. In the ROC curve analysis (Figure 2), a cut-off value of 57.9 for the KCCQ-12 score predicts the presence of high fibrosis with a sensitivity of 77.1% and a specificity of 33.3% (AUC: 0.717).

Conclusions: In conclusion, our study highlights that a lower KCCQ-12 score, indicating higher symptom burden, is a significant independent predictor of high fibrosis in HCM patients. The score's predictive value, with moderate sensitivity, underscores its potential utility in assessing fibrosis risk.

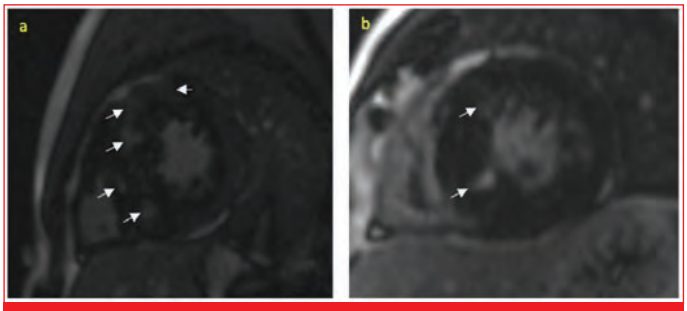

Figure 1. Short-axis post-contrast images from cardiac MRI of two patients, one with high fibrosis (a) and one without (b), white arrows show fibrosis areas.

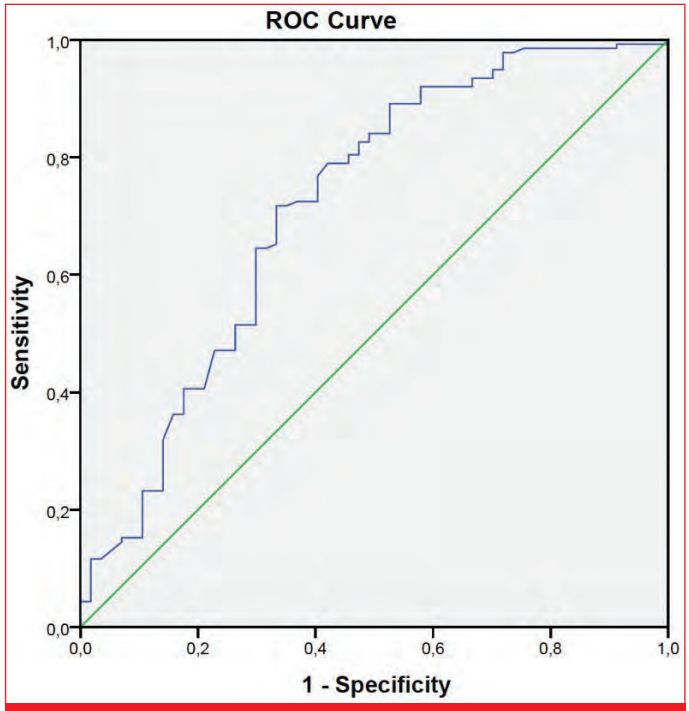

Figure 2. ROC curve of Kansas City Cardiomyopathy Questionnaire Score in prediction of high fibrosis.

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

Variable	Total population (n=195)	High fibrosis group (n=57)	Non-fibrosis group (n=138)	P-value
Age, months	53 (20.0, 76.0)	54 (20.0, 71.0)	52.5 (21.0, 76.0)	0.814
Gender (male)	142 (72.8)	47 (82.5%)	95 (68.8%)	0.077
BMI, kg/m2	26.7 (19.0-39.7)	27 (19.0, 35.4)	26,29 (19.8, 39.7)	0.564
NYHA Class, n (%)				0.3
1	*52 (26.6)*	13 (22.8%)	76 (55.1%)	
2	*105 (53.8)*	29 (50,9%)	39 (28.3%)	
3	*33 (16.9)*	12 (21.1%)	21 (15.2%)	
4	*5 (2.5)*	3 (5.3%)	2 (1.4%)	
KCCQ	63.5 (7.8, 96.8)	45.4 (9.3, 91.1)	68.7 (7.8, 96.8)	**<0.001**
Syncope, n (%)	33 (16.9)	8 (14%)	25 (18,1%)	0.6
Smoking, n (%)	92 (47.1)	27(47.4%)	65 (47.1%)	0.97
AF, n (%)	39 (20)	15 (26.3%)	24 (17.4 %)	0.22
Stroke, n (%)	6 (3)	1 (1.8%)	5 (3.6 %)	0.6
CAD, n (%)	42 (21.5)	11 (19.3%)	31 (22.5%)	0.7
DM, n (%)	35 (17.9)	8 (14 %)	27 (19.6%)	0.4
HT, n (%)	104 (53.3)	30 (52.6%)	74 (53.6%)	0.9
Family history of SCD, n (%)	23 (11.7)	7 (12.3%)	16 (11.6%)	1
Hgb, g/dL	14.6 (8.7, 18.4)	15 (9.7, 18.2)	14.4 (8.7,18.4)	0.289
Creatinine, mg/dL	0.88 (0.5-3.19)	0.89 (0.6, 1.8)	0.88 (0.5, 3.19)	0.803
Troponin, ng/mL	12.5 (2.4, 245.0)	16.4 (3.0, 245.0)	11.4 (2.4, 79.0)	**0.001**
CKMB, ng/mL	3 (0.5, 11.7)	3.3 (1.1, 10.9)	2.9 (0.5, 11.7)	**0.018**
NT-proBNP, pg/dL	420 (11, 5527)	738 (11, 5527)	302 (11, 3698)	
Logtrop	1.09 (0.39, 2.39)	1.22 (0.48, 2.39)	1.06 (0.39, 1.90)	**0.001**
LogNTPROBNP	2.62 (1.04-3.74)	2.87 (1.04, 3.74)	2.48 (1.04, 3.57)	**0.001**
Scd score	1.94 (1, 11)	2.13 (0.95, 6.5)	1.825 (0.68, 10.9)	**0.229**
ICD implantation, n (%)	19 (9.7)	6 (10.5%)	13 (9.4%)	0.8
Myectomy, n (%)	5 (2.5)	2 (3.5%)	3 (2.2%)	0.6
SAA, n (%)	10 (5.1)	3 (5.3%)	7(5.1%)	1
HCM type, n (%)				0.4
Septal	*99*	24 (42.1 %)	75 (54.3%)	
Non-obstructive	*72*	26 (45.6%)	46 (33.3%)	
Apical	*18*	5 (8.8%)	13 (9.4%)	
Mid-cavity	*6*	2 (3.5%)	4 (2.9%)	

Table 2. Echocardiographic and CMR characteristics of the patients

Variable	Total population (n=195)	High fibrosis group (n=57)	Non-fibrosis group (n=138)	P-value
Echo features				
LVEF, %	65 (35-78)	65 (36, 72)	65 (35, 78)	0.423
IVS, mm	17 (10-36)	18 (11.3, 36)	16.8 (10, 34)	**0.034**
PW, mm	12.1 (8-33.6)	13 (8, 33.6)	12 (8, 22)	**0.047**
LAD, mm	43 (33-63)	45 (34, 55)	42.4 (33, 63)	**0.046**
Rest gradient, mmHg	27.5 (10-122)	25 (10, 101)	28 (10, 122)	0.647
Provoked gradient, mmHg	57.5 (30-170)	55 (31, 134)	62 (30, 170)	0.208
TAPSE, mm	22 (12-32)	22 (12, 32)	22 (15.5, 32)	0.681
CMR features		8 (14%)	25 (18,1%)	0.6
CMR-LVEF, %	68 (29-87)	66 (29, 87)	70 (39, 86)	**0.002**
LV mass index, g/m^2	80 (43.15-172.5)	82.37 (53.89, 172.50)	77 (43.16, 162.5)	**0.016**
Indexed SV, ml/m^2	49 (14-136)	48 (25, 86)	49 (14, 136)	0.889
Indexed ESV, ml/m^2	22.5 (5-72)	24 (10,72)	21 (5,70)	**0.001**
Indexed EDV, ml/m^2	71 (30-190)	75 (44, 120)	71 (30, 190)	**0.028**
MWT, mm	18.5 (13.5-36)	20 (13.5, 36)	18.5 (13.5, 32)	**0.028**
Apical aneurysm, n (%)	3 (1.5)	2 (3.5%)	1 (0.7%)	0.2

Table 3. Logistic regression analysis of predictive factors for high fibrosis in HCM patients

Variable	Odds ratio	95% C.I.	P value
NT-proBNP	1.000	0.999 - 1.001	0.945
CK-MB	1.057	0.819 - 1.365	0.669
Troponin	1.011	0.979 - 1.043	0.502
LVEF	1.052	0.946 - 1.170	0.353
IVS	0.565	0.435 - 0.734	**<.001**
CMR-LVEF	0.845	0.757 - 0.944	**0.003**
MWT	2.094	1.563 - 2.806	**<.001**
LV mass index	1.091	1.042 - 1.143	**<.001**
KCCQ score	0.965	0.939 - 0.992	**0.010**

Cardiac Imaging / Echocardiography

OP-048

The effect of disopyramide treatment on functional capacity improvement in patients with obstructive hypertrophic cardiomyopathy

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Background and Aim: In hypertrophic cardiomyopathy (HCM), the presence of left ventricular outflow tract obstruction (LVOTO) is associated with symptoms and adverse outcomes. Medical treatment options are limited in patients with HOCM, and disopyramide is a frequently used agent in this context. Due to the difficulties in obtaining disopyramide in our country and the limited data on this subject, we aimed to investigate the effect of disopyramide on functional capacity in HCM patients.

Methods: Between October 2021 and May 2024, symptomatic HCM patients who used disopyramide due to obstructive HCM and those who were monitored under beta-blocker therapy because they could not obtain disopyramide were retrospectively evaluated. Patients were divided into two groups based on disopyramide use. Treatment response was defined as at least a 1-stage improvement in NYHA functional capacity. Clinical and laboratory benefits such as functional class improvement, change in NT-proBNP, and change in LVOT gradient were compared between the two groups.

Results: A total of 127 patients were evaluated revealing an average age of 54.2 years (±11), with 74 of them being male (58.2%). Table 1 presents the baseline characteristics and laboratory parameters of the study groups, categorized based on disopyramide administration. The initial provocative gradient of the patients was 67 mmHg (IQR: 55-89 mmHg), and their functional capacity was classified as NYHA class 2 in 79% of the cohort. Table 2 summarizes the imaging and laboratory findings after follow-up. A statistically significant improvement in functional capacity was observed in 40 patients (62%) within the disopyramide treatment group, compared to 16 patients (26%) in the control group not receiving disopyramide. Also, Disopyramide use (p=0.001, OR: 6.009, 95% CI: 2.094-17.239) and baseline NYHA class (p=0.035, OR: 4.485, 95% CI: 1.111-18.105) were identified as independent predictors for improvement in functional capacity (Table 3). Despite the improvement in functional capacity, there was no difference in clinical outcomes between the two groups. The side effects related to disopyramide were consistent with the literature, and none of the patients required discontinuation of the drug due to QT prolongation.

Conclusions: In this retrospective study, the use of disopyramide in patients with obstructive hypertrophic cardiomyopathy (HOCM) was associated with significant improvements in functional capacity. Specifically, 62% of patients in the disopyramide group experienced at least a 1-stage improvement in NYHA functional capacity, compared to only 26% in the control group. Additionally, disopyramide use and baseline NYHA class were identified as independent predictors for functional capacity improvement. These findings suggest that disopyramide may still be an effective therapeutic option for enhancing functional capacity in HOCM patients, underscoring the need for better accessibility to this medication in regions where it is difficult to obtain.

Table 1. Baseline clinical. laboratory. electrocardiographic and imaging characteristics of the patients

	Non-Disopyramide group (n:62)	*Disopyramide group (n:65)*	*Total (n:127)*	*P value*
Age, years	52.7±10.6	55.7±11.3	54.2±11	0.139
Male gender, n (%)	43 (69.3)	31 (47.6)	100 (78.7)	0.013
NYHA *2* *3* *4*	54 (87.1) 8 (12.9) 0 (0)	46 (70.7) 18 (27.6) 1 (1.5)	100 (78.7) 26 (20.6) 1 (0.79)	0.067
High-dose beta-blocker, n (%)	26 (42)	24 (37)	50 (39)	0.713
Heart rate, bpm	76.85±14.5	73.8±11.9	75.3±13.3	0.210
AF, n (%)	10 (16)	15 (23)	25 (20)	0.376
QTc, msn	450.1±32.6	448.9±24.8	449.5±28.9	0.819
QRS, msn	100.9±18.1	95.2±17	98±17.7	0.081
LVEF. %	60.0 (60.0 – 65.0)	60.0 (60.0 – 65.0)	60.0 (60.0 – 65.0)	0.912
MWT, mm	18±4.3	18.2±3.4	18.1±3.9	0.751
Resting gradient, mmHg	54.0 (44.0-74.2)	60.0 (42.0-74.5)	56.0 (44.0-74.0)	0.556
Provocable gradient, mmHg	62.0 (51.7-87.2)	59.0 (73.0-93.5)	67.0 (55.0-89.0)	0.031
LAD, mm	43.7±7.4	43.3±4.9	43.5±6.2	0.727
E/A	0.8 (0.7-1.2)	0.9 (0.7-1.3)	0.81 (0.71 – 1.28)	0.203
Septal e', m/sn	5.8±2.2	5.2±1.7	5.5±2	0.305
Lateral e', m/sn	8.5±3	8.5±3	8.5±2.9	0.989
E/e'	10.7 (8.0-15.3)	14.7 (12.7- 21.3)	13.2 (9.1 – 19.5)	0.006
sPAB, mmHg	27 (24.5- 30.5)	29.5 (24- 33.3)	27 (24 – 32)	0.415
CMR-LVEF, %	70 (67- 75)	67 (65- 75)	69 (65 – 75)	0.089
CMR- MWT, mm	19.2±4.4	19.7±3.9	19.4±4.1	0.522
Extent LGE, n(%)	7 (11.2)	9 (13.8)	16 (12.6)	0.393
NT-proBNP, pg/L	484.8 (110.7-1192.3)	876.4 (466 - 1785)	676 (222 – 1593)	0.024
Hs-TnT, ng/L	12.2 (7.6- 18)	14 (8.8- 19.4)	12.9 (8.3 – 18.3)	0.417
eGFR, ml/min	99.2 (83.1- 108)	95.2 (80.2- 106.7)	97.7 (82.5 – 106.9)	0.439

Table 2. The clinical, laboratory, electrocardiographic, and imaging characteristics of the patients at the end of the follow-up period

	Non-disopyramide group (n:62)	*Disopyramide group (n:65)*	*Total (n:127)*	*P value*
Follow up, months	15.8±7.3	15.2±6.0	15.5±6.7	0.682
NYHA *1* *2* *3*	13 (21) 28 (43) 13 (21)	28 (43) 33 (51) 4 (6)	41 (32) 69 (54) 13 (12)	0.067
NYHA Improvement, n (%)	16 (26)	40 (62)	56 (44)	0.001
Heart rate, bpm	72.4±11.7	71.6±10	72±10.8	0.690
AF, n(%)	6 (9.8)	8 (12.3)	14 (11)	0.426
QTc, msn	450.5±28.2	459±34.6	454.8±31.7	0.171
QRS, msn	99.7±21.1	101.6±20.0	100.7±20.5	0.653
NT-ProBNP, pg/L	566.5 (110.7- 1169.8)	654.5 (262.5- 1564)	635 (177 – 1450)	0.138
Hs-TnT, ng/L	11.5 (9.6- 17.5)	12.3 (6.9- 20)	15.7±12.5	0.733
LVEF, %	61.2±3.3	61.2±11.8	61.2±8.1	0.642
Resting gradient, mmHg	46.5 (36- 70.3)	40 (24.5- 60)	67 (55 – 89)	0.031
Provoked gradient, mmHg	57 (45.8- 86.5)	51 (35- 77)	55 (43 – 81)	0.018
LAD, mm	44.1±6.2	43.8±6.1	43.9±6.1	0.842
Δ QRS, msn	1.65±14.5	7.6±15.4	4.6±15.2	0.045
Δ QTc, msn	-1.6±25.3	18.3±65	8.7±50.8	0.044
Δ NT-proBNP, pg/L	-7.3 (-51.3- 60.8)	-44.4 (-244.5- 2.3)	-16.8 (-151.6-29)	0.003
Δ Resting gradient, mmHg	-4 (-7, -1.8)	-14 (-27, -8)	-8 (-17, -4)	<0.001
Δ Provoked gradient, mmHg	-4 (-9- 2.3)	-20 (-30.5- -12)	-11 (-10, -4)	<0.001
Outcomes *Myectomy* *Septal alcohol ablation* *Mortality*	11 (17.7) 4 (6.4) 5 (8) 2 (3.2)	8 (12.3) 3 (4.6) 4 (6.1) 1 (1.5)	19 (14.9) 7 (5.5) 9 (7) 3 (2.3)	0.391

Table 3. Logistic Regression Analysis to Identify Predictors of NYHA Class Improvement

Variables	Univariate analysis				Multivariate analysis			
	95% C.I.for OR		OR	P value	95% C.I.for OR		OR	P value
	Lower	Upper			Lower	Upper		
Disopyramide use	2,16	9,81	4,60	0,00	2,09	17,24	6,01	<0.001
Age	0,99	1,05	1,02	0,23	0,99	1,11	1,05	0.09
Initial NYHA	1,62	9,83	3,99	0,00	1,11	18,10	4,48	0.04
Initial NT-proBNP	1,00	1,00	1,00	0,24	1,00	1,00	1,00	0.15
Initial Rythm	0,41	2,40	1,00	0,99				
Gender	0,47	1,94	0,95	0,89				
CMR-MWT	0,89	1,08	0,98	0,71				
High dose beta-blocker use	0,66	2,83	1,37	0,40				
Constant			0,67	0,10			0,00	0,02

Cardiac Imaging / Echocardiography

OP-049

Unveiling the prognostic power of a new marker: Right ventricular scalloping index in ARVC patients-a multimodality imaging study

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Background and Aim: Arrhythmogenic right ventricular cardiomyopathy (ARVC), a rare form of cardiomyopathy associated with a dismal prognosis, involves the replacement of cardiac myocytes with fibrofatty tissue. Multiple criteria are utilized for diagnosis and risk assessment. The right ventricular scalloping index (RVSI) has emerged as a newly proposed parameter for ARVC diagnosis, yet its prognostic implications remain unexplored in the literature. Our study seeks to investigate the prognostic impact of cardiac magnetic resonance imaging (MRI) derived index (RVSI-MR) alongside other parameters.

Methods: The study retrospectively enrolled 27 patients diagnosed with ARVC at our clinic. Primary outcomes encompassed all-cause mortality, sudden cardiac death, sustained ventricular arrhythmia, and ablation necessitated by ventricular arrhythmia. In cardiac MRI, the epicardial contour of the RV was traced from the apex to the atrioventricular groove, with its length measured as the curve length from end-systolic phase images. A straight line connecting the two endpoints of the RV epicardial contour was measured as the straight distance. RVSI-MR was calculated by dividing the curve length by the straight distance. Comparative analysis, particularly focusing on the RVSI, was conducted on clinical, imaging, and electrocardiographic parameters within primary outcome groups. Additionally, Cox regression analysis was employed to identify parameters influencing prognosis.

Results: The average age was 42.59 ± 16.8 years, with males constituting 81.5% of the patient cohort. The median follow-up period was 134 months (0-236). Notably, in patients

with the primary outcome, RVSI-MR was significantly higher (1.32 ± 0.1 vs. 1.46 ± 0.2, p=0.007), while RV ejection fraction (EF)-MR was notably lower (54.91 ± 10.1 vs. 41.32 ± 12.9, p=0.007). Furthermore, RVSI-MR exhibited a positive correlation with the RV mid diameter-transthoracic echocardiography (TTE) and a negative correlation with RVEF-MR (r=0.428, p=0.029, r=-0.477, p=0.016, respectively). Cox regression analysis identified RV dysfunction at diagnosis and RVEF-MR as predictors of prognosis (HR 29.364, p=0.004, HR 0.863, p=0.021, respectively). Additionally, ROC analysis revealed that an RVSI-MR value exceeding 1.43 predicted the primary outcome with 72.73% sensitivity and 87.50% specificity (AUC 0.609, p=0.002), while an RVEF-MR value of ≤ 49.71% predicted the primary outcome with 70% sensitivity and 73.33% specificity (AUC 0.787, p=0.004).

Conclusions: The right ventricular scalloping index represents a novel and easily applicable cardiac MRI parameter useful for ARVC diagnosis and risk stratification.

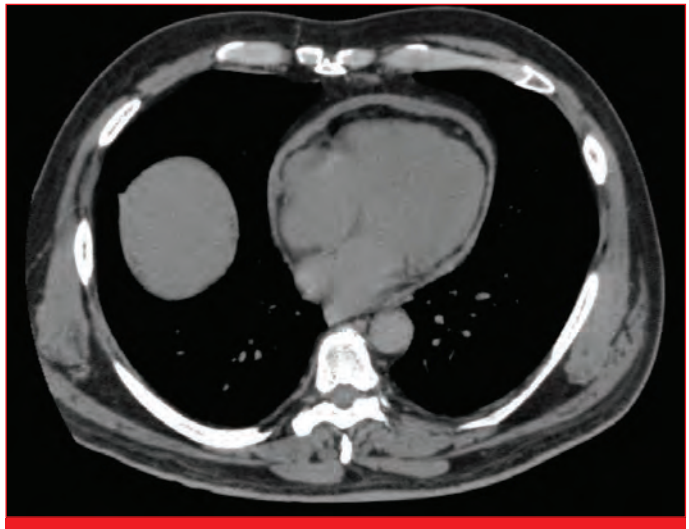

Figure 1. Right ventricular scalloping index, calculated by dividing the curve length by the straight distance.

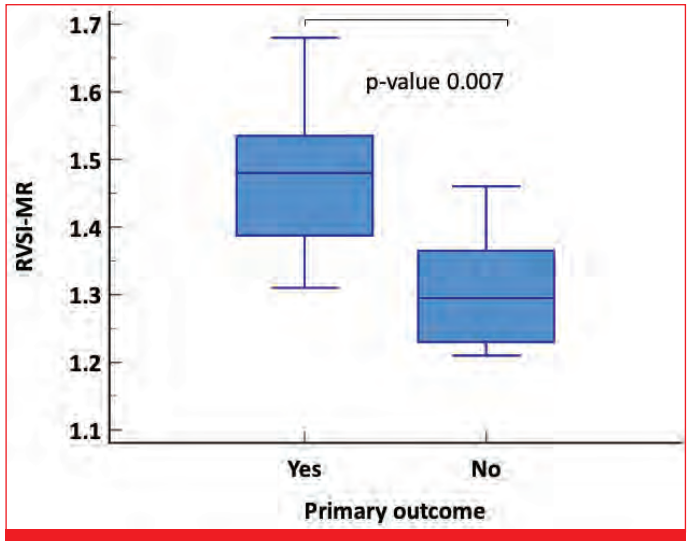

Figure 2. Comparison of RVSI in patients with primary outcome and without primary outcome.

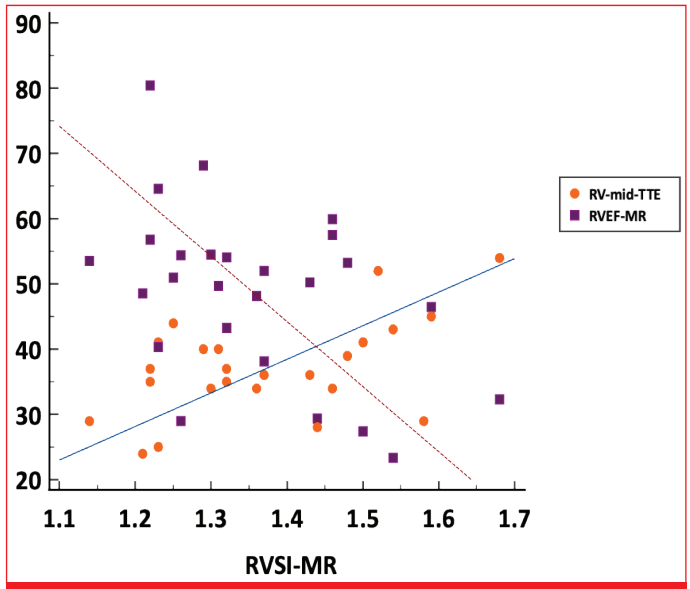

Figure 3. Correlation of RVSI with RV-mid-TTE and RVEF-MR.

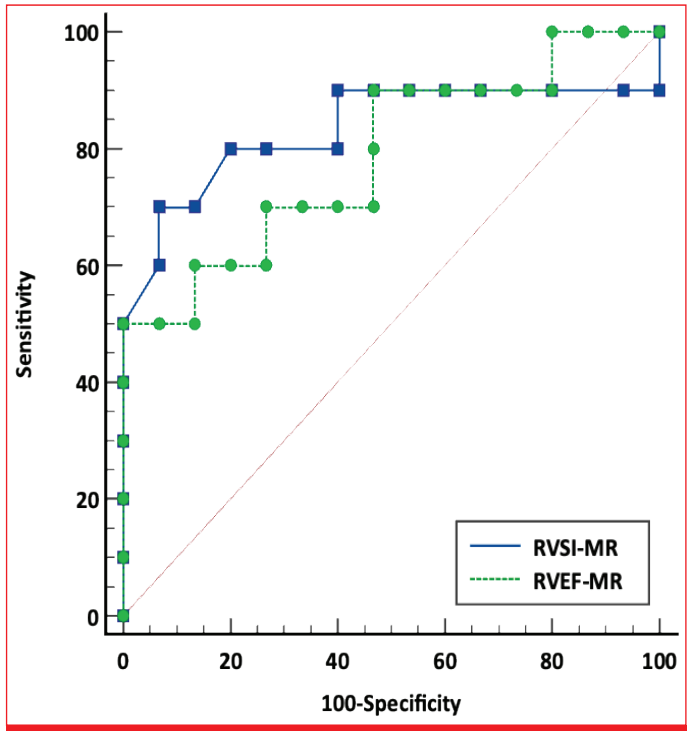

Figure 4. ROC analysis of RVSI-MR and RVEF-MR for primary outcome.

Cardiac Imaging / Echocardiography

OP-050

Assessment of cryptogenic stroke risk in patients with atrial septal defect: A single-center transesophageal echocardiography study

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Background and Aim: Atrial septal defect (ASD) and patent foramen ovale (PFO) are common congenital heart defects, each carrying distinct risks for cryptogenic stroke (CS). While PFO is often implicated in paradoxical embolism and subsequent stroke, the risk associated with isolated ASD remains less clear. This study aims to evaluate the prevalence of CS in patients with isolated ASD and ASD with PFO detected using transesophageal echocardiography (TEE).

Methods: A retrospective, single-center study was conducted on patients who underwent TEE at a tertiary care center between January 2016 and December 2022. Patient data, including imaging results and medical records, were obtained from the hospital's archives. The study analyzed 362 patients diagnosed with ASD, including 291 patients with isolated ASD and 71 patients ASD with PFO. Key variables such as age, gender, hemoglobin (Hb) levels, LDL cholesterol levels, glomerular filtration rate (GFR), left ventricular ejection fraction (LV EF), diabetes mellitus (DM), smoking, hypertension (HT), hyperlipidemia (HPL), chronic kidney disease (CKD), coronary artery disease (CAD), and the incidence of cryptogenic stroke (CS) were evaluated. All statistical analyses were performed using SPSS software, version 26 (SPSS Inc., Chicago, IL, USA), and R software. Statistical significance was determined with a p value of <0.05.

Results: No statistically significant differences were found between the isolated ASD and ASD with PFO groups in terms of age, gender distribution, hemoglobin levels, LDL cholesterol levels, LV EF, DM, smoking, HT, HPL, CKD, or CAD (p>0.05 for all) (Figure 1). However, GFR was significantly lower in the ASD with PFO group compared to the isolated ASD group (61.28 ± 19.72 vs. 74.77 ± 13.85 mL/min/1.73 m^2, p=0.002). Moreover, the incidence of CS was significantly higher in the ASD with PFO group (32.39% vs. 5.50%, p<0.001). Multivariate analysis revealed that PFO was a significant predictor of increased CS risk with an odds ratio of 7.40 (95% CI 3.26-16.82; p<0.001).

Conclusions: This study underscores the significant role of PFO as an independent predictor of CS within the ASD patient population. The presence of PFO in patients with ASD was strongly correlated with an elevated risk of CS, highlighting the critical clinical implications for this particular group. These findings emphasize the necessity for heightened clinical vigilance and potentially more aggressive management strategies in ASD patients harboring a PFO. The association between PFO and increased CS risk in this cohort advocates for further exploration of the underlying pathophysiological mechanisms and supports the development of tailored preventive measures to mitigate stroke risk in this vulnerable population.

Table 1. Demographic, Clinical and Laboratory Characteristics of Study Groups

Variable	ASD Group (n=291)	ASD with PFO Group (n=71)	p
Age (years) (mean ± SD)	39.98 ± 15.05	38.87 ± 16.08	0.640
Gender; Male (n, %)	104 (35.74%)	26 (36.62%)	0.879
Hb (g/dL) (mean ± SD)	12.85 ± 1.07	12.52 ± 1.18	0.155
LDL-C (mg/dL) (mean ± SD)	112.26 ± 28.95	116.58 ± 30.38	0.343
GFR (mL/min/1.73 m^2) (mean ± SD)	74.77 ± 13.85	61.28 ± 19.72	0.002
LV EF (%) (mean ± SD)	55.12 ± 5.78	54.87 ± 6.05	0.721
DM (n, %)	20 (6.87%)	5 (7.04%)	0.947
Smoking (n, %)	34 (11.68%)	14 (19.72%)	0.123
HT (n, %)	56 (19.24%)	15 (21.13%)	0.745
HPL (n, %)	25 (8.59%)	9 (12.68%)	0.315
CKD (n, %)	7 (2.41%)	0 (0.00%)	0.245
CAD (n, %)	25 (8.59%)	5 (7.04%)	0.698
CS (n, %)	12 (4.12%)	16 (22.54%)	<0.001

ASD: Atrial septal defect; PFO: Patent foramen ovale; Hb: Hemoglobin; LDL-C: Low density lipoprotein cholesterol; GFR: Glomerular filtration rate; LV EF: Left Ventricular ejection fraction; DM: Diabetes mellitus; HT: Hypertension; HPL: Hyperlipidemia; CKD: Chronic kidney disease; CAD: Coronary artery disease; CS: Cryptogenic stroke.

Cardiac Imaging / Echocardiography

OP-052

Effect of iron deficiency and intravenous supplementation on myocardial deformation in patients without cardiovascular risk

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Background and Aim: Although iron deficiency is one of the most common metabolic disorders worldwide and a major cause of anemia, it is often overlooked and untreated for long periods of time unless anemia develops and worsens. There are studies suggesting that iron deficiency is associated with impaired deformation parameters by strain echocardiography in healthy females. However, these studies have not shown whether subclinical myocardial cardiac function improves after iron replacement. The aim of the study was to evaluate the effect of iron deficiency and supplementation on myocardial deformation parameters.

Methods: In this study, female patients who were diagnosed with iron deficiency anemia in the internal medicine outpatient clinic and were planned to receive intravenous iron replacement therapy within medical indications were included in the study. Conventional and strain echocardiography were performed before intravenous iron therapy and 4-8 weeks after the anemia resolved. Patients were compared with age-matched female controls without iron deficiency or anemia.

Results: The study population consisted of 53 female patients with iron deficiency anemia without cardiovascular risk (mean age: 35.5 ± 9.9 years) and 47 controls (mean age: 38.8 ± 7.7 years). Pre-treatment baseline conventional echocardiography parameters were similar in the anemia and control groups, while left ventricular global longitudinal strain (LV-GLS) was significantly lower in the anemia group (-17.08 ± 2.2 vs. -19.72 ± 2.0, p<0.001). Intravenous iron therapy was administered to patients with anemia. Ferritin and hemoglobin levels increased significantly after treatment (222.6 ± 208.6 vs. 7.28 ± 7.15 ng/mL, p<0.001; 12.86 ± 1.2 vs. 9.32 ± 1.7, p<0.001; respectively). Post-treatment myocardial deformation parameters were significantly higher in the anemia group compared to pre-treatment baseline measurements (-18.98 ± 1.8 vs. -17.08 ± 2.2, p<0.001). Baseline ferritin and hemoglobin levels were negatively correlated with baseline LV-GLS (r= -0.51, p<0.001; r= -0.38, p<0.001, respectively). Baseline ferritin level was an independent predictor of baseline impaired LV-GLS (OR: 0.94, 95% CI 0.89-0.98, p=0.008). Ferritin level < 8.8 ng/mL predicted baseline impaired LV-GLS with 77% sensitivity and 86% specificity.

Conclusions: Intravenous iron supplementation has beneficial effects on myocardial deformation parameters in young women with anemia without cardiovascular risk. Moreover, baseline ferritin level is a predictor of impaired LV-GLS.

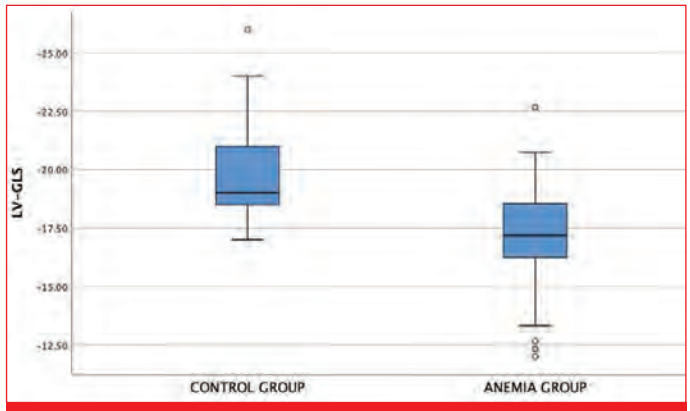

Figure 1. Comparison of LV-GLS between anemia and control groups.

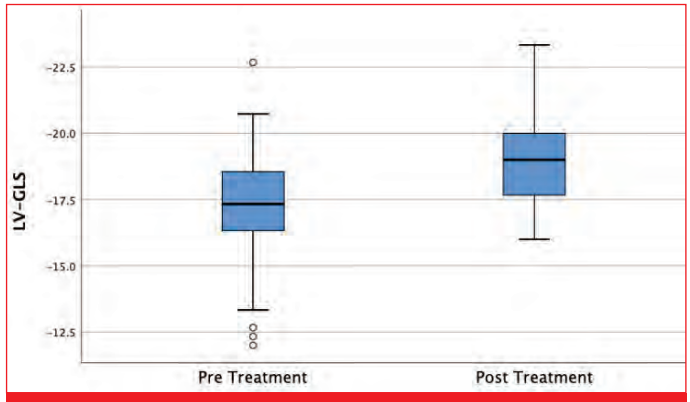

Figure 2. Comparison of LV-GLS before and after intravenous iron treatment in the anemia group.

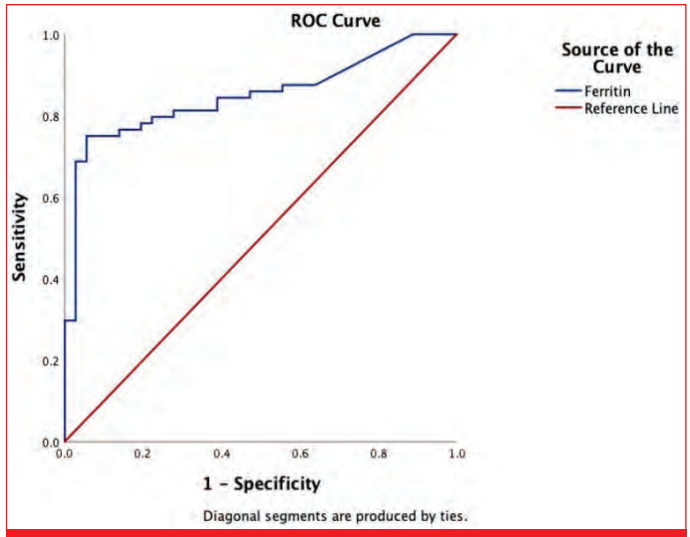

Figure 3. ROC curve analysis showing the specifcity and sensitivity of the ferritin level in predicting impaired LV-GLS.

Cardiac Imaging / Echocardiography

OP-053

Comparison of left atrial functions between medical therapy and cryoablation in patients with paroxysmal atrial fibrillation: An advanced echocardiography study

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Background and Aim: The improvement of left atrial structure and mechanics, gained by treatment strategies, are closely related with prognosis in atrial fibrillation (AF) patients. The aim of this study was to evaluate the long-term effects of medical therapy or cryoablation (CA) on the size and functions of the left atrium (LA) and left atrial appendage (LAA) in AF patients with advanced echocardiography techniques.

Methods: A total of 60 paroxysmal AF patients were examined in two groups: 30 who had history of CA and 30 who did not undergo CA and were followed up medically (beta blockers and/or other antiarrhythmic drugs). A standard 12-lead electrocardiogram was taken upon admission. All patients were required to be in sinus rhythm at the time of inclusion. Transthoracic echocardiography examination, including detailed tissue Doppler measurements and 2D speckle tracking echocardiography (Figure 1A), was performed. Transesophageal echocardiography (TEE) imaging, including Doppler measurements and 3D orifice area measurement (Figure 1B), was performed to evaluate LAA functions.

Results: The average age of the patients was 61.5 ± 8.6 years and 55% were male. The median follow-up time from AF diagnosis was 39.3 (24-52) months in the study population. The two groups were similar in terms of LA reservoir strain, conduit strain and contractile strain values (p=0.47, p=0.89 and p=0.27, respectively). In the TEE examination, no significant difference was observed between the medical therapy and CA groups in terms of early and late emptying velocities, which indicate LAA contractile functions (p=0.63 and p=0.09, respectively). While the average LAA orifice area change was 40.9 ± 15.4 in the medical therapy group, it was 47.3 ± 17.1 in the CA group, but this difference was not statistically significant (p=0.13). There was no difference between the two groups in terms of LAA tissue Doppler characteristics and pulmonary vein flow velocities (p<0.05).

Conclusions: There was no superiority between medical therapy and CA in terms of LA and LAA size and functions in paroxysmal AF patients, evaluated by advanced echocardiography techniques.

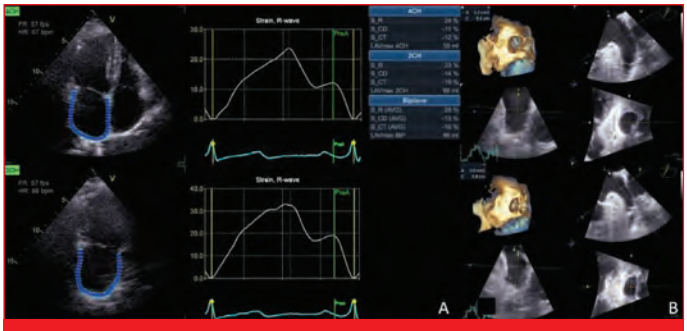

Figure 1. 2D speckle tracking evaluation of the left atrium and 3D orifice area measurement of left atrial appendage.

Cardiac Imaging / Echocardiography

OP-054

May recovered COVID-19 patients have impaired myocardial Work?

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Background and Aim: The aim of this study was to investigate whether there are sequelae in left ventricular (LV) systolic function by comparing LV function in fully recovered COVID-19 patients with pulmonary involvement and healthy controls without COVID-19 by conventional echocardiography and myocardial work.

Methods: 55 healthy volunteers and 61 patients hospitalized with COVID-19 with pulmonary involvement were included. Patients did not need non-invasive or invasive mechanical ventilation support during hospitalization. Patients were included in the study if they were asymptomatic for at least six months after recovery from COVID-19. Transthoracic echocardiography (TTE) was performed. Demographic and clinical characteristics and laboratory test results were collected. Clinical characteristics, blood tests, TTE, speckle-tracking echocardiography (STE) and myocardial work results were compared.

Results: No statistically significant differences were found in the longitudinal strain parameters of the LV among the study groups. The patient group exhibited notably reduced levels of global work index (GWI), global constructed work (GCW), and global work efficiency (GWE). The patient group exhibited a notably elevated global wasted work (GWW).

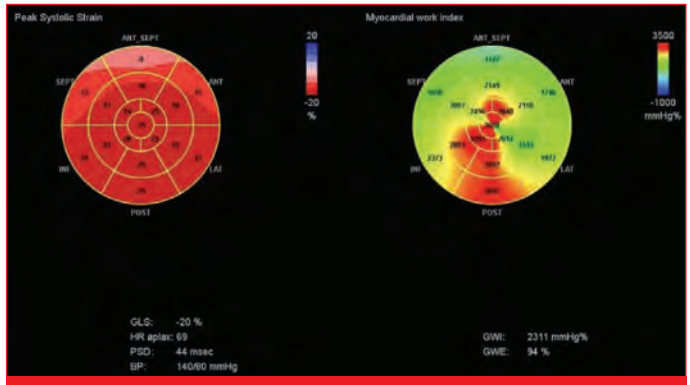

Figure 1.

Conclusions: The group of fully recovered asymptomatic COVID-19 patients with pulmonary involvement showed significantly lower values for GWI, GCW, and GWE, while GWW showed a significantly higher value. Myocardial work parameters may be useful in determining myocardial sequelae.

Table 1. Clinical characteristics of the patiens, and laboratory data

	Patient group (n=61)	Control group (n=55)	p value
Patient characteristics			
Age (years)	41.6 ± 10.3	42.0 ± 9.9	0.809
Gender			0.318
Male (n, %)	39 (72.2%)	34 (61.8%)	
Female (n, %)	15 (27.8%)	21 (38.2%)	
BMI (kg/m^2)	25.92 ± 2.72	26.1 ± 4.34	0.444
Systolic blood pressure (mmHg)	116.5 ± 13.3	119.5 ± 10.6	0.198
Diastolic blood pressure (mmHg)	75.0 ± 7.2	76.8 ± 7.4	0.340
Laboratory Data			
Hemoglobin, g/dL	14.3 ± 1.2	13.7 ± 1.4	0.068
White blood cell count (10^3/µl)	7.1 ± 1.5	7.5 ± 3.3	0.423
Serum creatinine (mg/dL)	0.80 ± 0.05	0.80 ± 0.14	0.219
Glucose (mg/dL)	97.0 ± 25.6	105.6 ± 21.1	0.659
Sodium (mEq/L)	139.3 ± 2.2	136.4 ± 1.5	0.155
AST (unit/L)	21.0 ± 7.5	23.9 ± 7.9	0.259
ALT (unit/L)	24.2 ± 12.8	25.3 ± 8.7	0.130
Thyroid stimulating hormone (TSH) (mIU/L)	1.6 ± 1.3	1.6 ± 0.7	0.999

BMI, body mass index

Table 2. Echocardiography results

	Patient group (n=61)	Control group (n=55)	p value
LVEDD (mm)	45.4 ± 3.3	45.3 ± 2.9	0.890
LVESD (mm)	30.52 ± 2.57	27.40 ± 3.00	**<0.001**
LAV max	40.7 ± 10.1	38.8 ± 9.6	0.288
LVEF (%)	59.8 ± 9.4	61.2 ± 4.2	0.261
IVS (mm)	10.0 ± 1.1	9.2 ± 0.9	0.378
E/A ratio	1.1 ± 0.5	1.1 ± 0.3	0.665
Em lateral (cm/s)	0.15 ± 0.04	0.18 ± 0.02	0.074
Am lateral (cm/s)	0.12 ± 0.04	0.14 ± 0.13	0.197
TAPSE (mm)	23.4 ± 3.4	24.1 ± 3.2	0.091
LV-LS 4 chamber (%)	19.60 (19.00 - 20.47)	19.10 (18.00 - 20.05)	0.051
LV-LS 2 chamber (%)	21.63 (20.00 - 22.10)	21.10 (19.30 - 23.00)	0.767
LV-LS 3 chamber (%)	18.17 (18.00 - 21.30)	19.60 (18.10 - 21.70)	0.400
LV-GLS (%)	20.00 (20.00 - 20.00)	20.17 (18.71 - 20.92)	0.923
GWI (%mmHg)	1584 (1481 - 2017)	2406 (2270 - 2540)	**<0.001**
GCW (%mmHg)	2305 (1889 - 2526)	2719 (2604 - 2885)	**<0.001**
GWW (%mmHg)	223 (215 - 353)	213 (203 - 226)	**0.009**
GWE (%mmHg)	88 (85 - 92)	93 (91 - 94)	**<0.001**

LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LAV max, left atrium maximum volume; LVEF, left ventricular ejection fraction; IVS, interventricular septum thickness; E, mitral inflow early diastolic velocity; A, mitral inflow late diastolic velocity; Em, mitral inflow early diastolic tissue velocity; Am, mitral inflow late diastolic tissue velocity; TAPSE, tricuspid annular plane systolic excursion; LV, left ventricle; LS, longitudinal strain; GLS, global longitudinal strain; GWI, global work index; GCW global constructed work; GWW, global wasted work; GWE, global work efficiency

Cardiac Imaging / Echocardiography

OP-055

Evaluation of the relationship between the presence of scar on cardiac MRI and the selvester score in patients with hypertrophic cardiomyopathy

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Background and Aim: The Selvester score is a grading system used to assess the extent of scar tissue resulting from ischemic or non-ischemic causes through electrocardiographic data. When utilized in conjunction with advanced imaging techniques such as cardiac magnetic resonance, it plays a crucial role in guiding diagnostic and therapeutic decisions. In this study, we aim to evaluate the relationship between the presence of scar on cardiac MRI and the Selvester score in patients with hypertrophic cardiomyopathy.

Methods: In the past year, 36 patients who presented to our hospital and underwent cardiac MRI with a diagnosis of hypertrophic cardiomyopathy were retrospectively reviewed. The patients' ECG, cardiac MRI, and laboratory findings were recorded. The Selvester score was calculated to assess the presence of myocardial scarring and was compared with the cardiac MRI results.

Results: A total of 36 patients with hypertrophic cardiomyopathy (HCM) were included in the study. Of these, 28 (77.7%) had late gadolinium enhancement (LGE) on cardiac MRI. There was no significant difference between the two groups in terms of characteristic features. Laboratory findings revealed that the pro-BNP levels were significantly higher in the group with LGE (p=0.003) (Table 1). The Selvester score was also significantly higher in the group with LGE (p=0.031). A Selvester score ≥3 was found to be associated with LGE in HCM patients with a sensitivity of 57% and specificity of 85% (AUC: 0.69, 95% CI, p=0.03) (Figure 1).

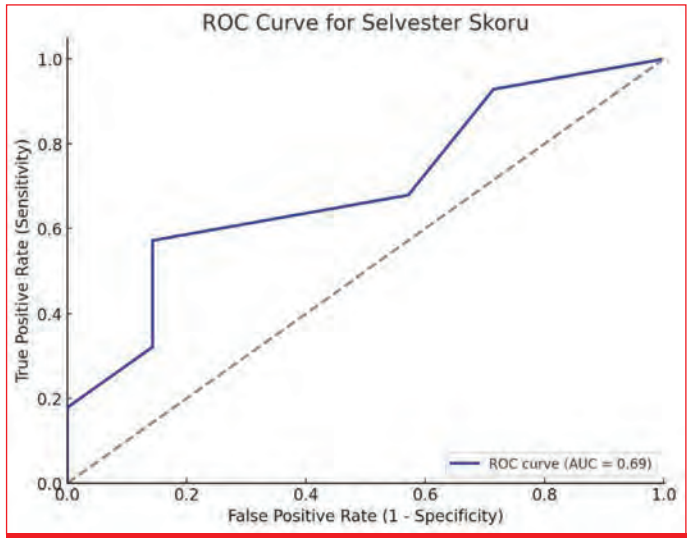

Figure 1. ROC curve for Sylvester score.

Table 1. Demographic and Clinical Data According to Hypertrophic Cardiomyopathy

	Study population (n=36)	Cases with LGE (n=28)	Cases without LGE (n=8)	p value
Age, year	51.28 ± 13.61	52.29 ± 13.39	47.75 ± 14.71	0.450
Gender, female, n (%)	10 (27.8)	7 (25)	3 (37.5)	0.658
Comobidities, n (%)				
DM	7 (19.4)	5 (17.9)	2 (25)	0.639
HT	17 (47.29	13 (46.4)	4 (50)	1.000
HL	8 (22.2)	7 (25)	1 (12.5)	0.651
Laboratory findings				
Neutrophil, 10^3/uL	4.9 ± 2.8	4.6 ± 2.1	6.07 ± 4.3	0.409
Lymphocyte, 10^3/uL	2.2 ± 1.06	2.2 ± 0.9	2.5 ± 1.3	0.590
Plt, 10^3/uL	243 ± 63	238 ± 60	257 ± 75	0.529
Hs CRP, mg/L	4.4 ± 3.5	4.1 ± 3.9	3.7 ± 1.9	0.772
Pro-BNP, pg/mL	742 ± 952	972 ± 1000	50 ± 28	0.003
Troponin, ng/L	191 ± 571	236 ± 633	12 ± 9.3	0.131
Albumin, g/L	43.9 ± 3.7	43.7 ± 3.7	44.5 ± 3.9	0.656
Echocardigraphic findings				
LVEF, %	59.7 ± 4.6	59.5 ± 5.2	60.6 ± 1.7	0.359
LAD, cm	39.7 ± 5.6	40.5 ± 5.7	37.1 ± 4.4	0.095
PASP, mmHg	26.2 ± 10.9	27.1 ± 12.1	23.2 ± 4.1	0.162
SAM, n (%)	29 (80.6)	23 (82.1)	6 (75)	0.639
LVOT gradient ≥30 mm Hg, n (%)	17 (47.2)	13 (46.4)	4 (50)	1.000
Drugs, n (%)				
Beta blockers	27(77.1)	20 (74.1)	7 (87.5)	0.648
CCB	3(8.6)	2 (7.4)	1 (12.5)	0.553
Selvester QRS score	3.25 ± 2.7	3.71 ± 2.82	1.63 ± 1.99	0.031

BNP: Brain natriuretic peptide; CCB: Calcium channel blockers; DM: Diabetes mellitus; HL: Hyperlipidemia; Hs-CRP: High sensitive C-reactive protein; HT: Hypertension, LVEF: Left ventriculer ejection fraction; LVOT: Left ventriculer outflow tract; PASP: Pulmonary systolic artery pressure; PLT: Platelet; SAM: Systolic anterior motion.

Conclusions: The Selvester score is an effective electrocardiographic finding for evaluating the presence of scar tissue in patients with hypertrophic cardiomyopathy. Our study supports previous research by demonstrating the success of this scoring system in identifying scar presence.

Epidemiology

OP-056

TURK-HEART a study designed to determine strategies for achieving recommended dyslipidemia treatment goals in very high CVD risk patients in Türkiye

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Background and Aim: Atherosclerotic cardiovascular disease (ASCVD) remains the leading global cause of death. Modifiable riskfactors like elevated LDL-C, high blood pressure, smoking, and diabetes significantly contribute to ASCVD. Real-world data indicate challenges in meeting LDL-C goals, with only very few of very high-risk patients achieving European guidelines targets. Turkey's secondary prevention efforts are suboptimal, highlighting the need for improved coordination and implementation of evidence-based recommendation. The study aim is; how lipid lowering therapy is managed in real-world clinical practice and effect on mortality and morbidity in very-high- risk patients. This prospective, multi-center study encompasses 42 months, including a 6-month recruitment period and three-years follow-up.

We planned to enroll more than 1500 adult patients with very high risk ASCVD in NUTS-2 subregions of Turkey. Data collection would cover demographics, medical history, lifestyle, blood biochemistry, lipid-modifying treatments, major cardiovascular events, total deaths, and health economics. Follow-up intervals will align with routine clinical practice, occurring at 3-month, 6-month, and subsequent 6 month intervals. Data collected will be transferred to an electronic Case Report Form. Descriptive statistics will be expressed as numbers and percentages and as mean or median for numerical variables. The chi-square test will be used for categorical variables, and change in each follow-up time point according to baseline will be investigated with paired T-Test or Wilcoxon Signed-Rank test.

Results: Primary endpoints are percentage of patients with LDL- C reduction ≥50% from baseline and LDL-C goal <55 mg/dL in three-tear follow-up period in very-high- risk patients. Secondary endpoints are rate of composite MACE per person-year, rate of adherence to prescribed lipid lowering medications, percentage of patients having follow-up visit in every 5-7 months during follow-up period. **Conclusions:** Our research will shed a light on the implementation of guidelines into real-life practice. We will also plan to set-up a lipid clinic network across Turkey for better management of dyslipidemia.

Epidemiology

OP-057

Lyme disease seroprevalence in patients with a pacemaker

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Background and Aim: Lyme disease, the most common zoonosis in the Northern Hemisphere, is a disease transmitted by tick bite, that can lead to a wide range of clinical manifestations. The heart is also one of the organs that can be affected by this disease. As a result of Lyme carditis, patients can develop varying degrees of atrioventricular block, and some may require the implantation of a pacemaker. Due to the reversible nature of heart block, permanent pacemaker implantation is not recommended for these cases except in very rare circumstances. Clinical studies and seropositivity research related to Lyme disease and Lyme carditis in Turkey are insufficient. In this study, our primary aim was to determine the seropositivity of Lyme disease in patients with a pacemaker and to identify how many of the patients with a permanent pacemaker had Lyme disease.

Methods: Our study was conducted on patients who received outpatient or inpatient treatment at the cardiology clinic of Ankara Bilkent City Hospital between November 2022 and May 2024. Among adult patients who had undergone temporary and/or permanent pacemaker implantation or had previously been implanted with a pacemaker, 168 patients whose IgG and IgM tests against *Borrelia burgdorferi* were studied using the ELISA method were included in the study. Cases were evaluated for whether the pacemaker was implanted during treatment at our hospital or previously, ECG rhythms and ventricular pacing percentages. Lyme disease seropositivity was examined across all cases, and it was analyzed which characteristics were associated with higher seropositivity rates.

Results: 82 (48.8%) of the patients in the study were male and 86 (51.2%) were female. The average age at which pacemakers were implanted in patients is 66.80 ± 16.61 years. 97 of the patients (57.7%) were newly implanted with a pacemaker, while 71 (42.3%) had an existing pacemaker. The seropositivity rate for Lyme disease among all cases was found to be 9.5%. In cases without pacing rhythm, IgG test positivity was significantly higher (p=0.013). When evaluated among patients with newly implanted pacemakers, significantly higher IgG positivity was found in cases without pacing rhythm compared to the other group (p=0.048).

Conclusions: The seroprevalence of Lyme disease in patients with pacemakers is non-negligible. Considering the seroprevalence studies of Lyme disease conducted in our country, it has been found that the seropositivity among patients with pacemakers is higher than that in the general population. Therefore, the possibility that patients with pacemakers have had or currently have Lyme disease should not be overlooked. Especially patients whose heart rhythm is not in a paced rhythm are more likely to have experienced Lyme carditis.

Epidemiology

OP-058

Usefulness of the systemic immune inflammation ındex on predicting adverse cardiac events in patients with hypertrophic cardiomyopathy

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Background and Aim: Hypertrophic cardiomyopathy (HCM) is one of the most common genetic myocardial diseases. The clinical course of HCM is highly heterogenious, ranging from a normal lifespan with asymptomatic status to advanced heart failure, systemic embolic events, and sudden cardiac death. The major challenge in the clinical management of HCM is identifying those at increased risk for life-threatening ventricular arrhythmias and SCD. In recent decades, much attention has been given to evaluate the role of inflammation and oxidative stress in both for pathogenesis and determining prognosis of HCM. The systemic immune inflammation index (SII) has emerged as a new predictor of inflammation and is determined using the following equation: neutrophil (N) × platelet (P) ÷ lymphocyte (L). The prognostic significance of SII in the clinical outcomes of HCM remains to be evaluated. The aim of the present study was to evaluate of SII on the clinical endpoints in patients with HCM.

Methods: This study investigated consecutive 403 patients with HCM (177 women; 51.1 ± 14.9 years old). Data regarding

clinical features, risk profiles, echocardiographic and laboratory parameters of all patients were obtained from clinical follow-up visits, patients' files, and the electronic database. The primary endpoint was defined as the occurrence of composite cardiac events that included cardiovascular death or malignant arrhythmic events (SCD, sustained VT or VF or appropriate ICD shock).

Results: After a median [interquartile range (IQR)] follow-up period of 6.0 (5.0-8.0) years, composite primary endpoint was developed in 51 (7.9%) patients. ROC analysis showed that using a cut-off level of 532, SII predicted the primary endpoint with a sensitivity of 71 % and specificity of 68% (Figure 1). Univariate Cox regression analyses showed that atrial fibrillation, NSVT, the mean HCM risk-SCD according to ESC risk score, CRP and high SII were significantly associated with the primary endpoint (for all, $p < 0.05$) (Table 4). However, in the multivariate model, SII (HR: 3.1, 95% CI: 1.6-5.9; $p<0.001$, was the only significant predictor of the primary endpoint (Table 1).

Conclusions: This study showed that SII is an independent predictor of malignant arrhythmia and death in patients with HCM. This parameter may be used to establish patients at high risk for adverse cardiac events and guiding selection for aggressive therapy in patients with HCM.

Table 1.

Variable	Univariate analysis			Multivariate analysis		
	HR	95% CI	p value	Adjusted HR	95% CI	p value
Age	0.992	0.974-1.010	0.391			
Gender, male	0.854	0.493-1.479	0.573			
Heart Failure	1.924	0.820-4.519	0.133			
Atrial fibrillation	2.099	1.195-3.687	0.010	1.428	0.765- 2.668	0.263
LVEDD (mm)	1.034	0.986-1.034	0.166			
Maximal wall thickness	1.049	0.995-1.107	0.078			
LVEF (%)	0.981	0.952-1.012	0.231			
LVOT Gradient (mmHg)	0.996	0.987-1.005	0.350			
LA Diameter (mm)	1.013	0.969-1.059	0.567			
SCD at 5 years - HCM risk-SCD	1.087	1.009-1.171	0.027	1.011	0.893-1.095	0.830
Family history of SCD	0.776	0.414-1.454	0.428			
NSVT at 24- h Holter monitoring	2.485	1.413-4.371	0.002	1.784	0.864-3.684	0.118
Maximal LV wall thickness ≥30 mm	1.522	0.602-3.848	0.374			
Syncope	0.973	0.457-2.069	0.943			
Abnormal blood pressure response	0.700	0.095-5.174	0.727			
Hemoglobin (g/dL)	0.968	0.858-1.094	0.606			
Platelet ($x10^3$ µL)	0.999	0.995-1.002	0.496			
WBC ($x10^3$ µL)	1.001	0.943-1.063	0.966			
Neutrophil (x103 µL)	1.051	0.920-1.201	0.465			
Lymphocyte (x103 µL)	1.021	0.732-1.426	0.901			
Monocyte (x103 µL)	1.002	1.001-1.003	<0.001			
Glucose (mg/dL)	0.996	0.989-1.004	0.325			
Creatinine (mg/dL)	1.185	0.801-1.753	0.395			
Uric acid (mg/dL)	1.041	0.913-1.187	0.548			
HDL-C (mg/dL)	0.993	0.973-1.013	0.470			
TSH, UI/mL	1.025	1.000-1.051	0.051			
hsCRP (mg/dL)	1.043	1.018-1.068	0.001	1.017	0.988-1.046	0.246
SII ≥532	3.941	2.142-7.249	<0.001	3.148	1.661-5.966	<0.001

Univariate and multivariate cox regression analysis for prediction of primary composite endpoint. (Bolded values indicate statistically significant odds ratio. CI: confidence interval; CRP: C-reactive protein; HDL-C: High-density lipoprotein cholesterol; LA: Left atrium; LVEDD: Left ventricular end diastolic diameter; LVEF: Left ventricular ejection fraction; LVOT: Left ventricular outflow tract; NSVT: Non sustained ventricular tachycardia; NYHA: New York Heart Association; SCD: Sudden cardiac death; SII: Systemic immune inflammation index; TSH: Thyroid stimulating hormone; WBC: White blood cell)

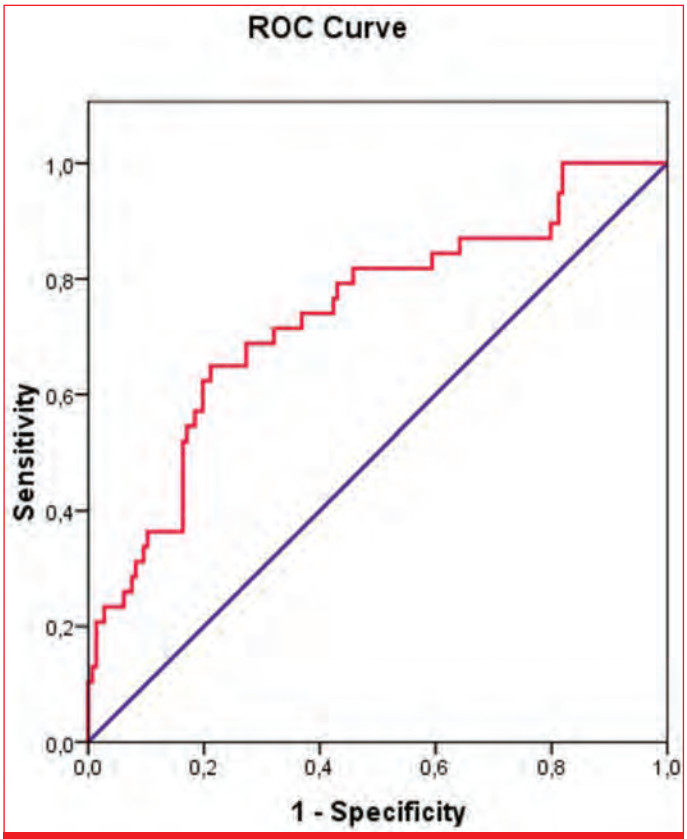

Figure 1. Receiver-operating characteristic curve of the systemic inflammation index for predicting adverse cardiac events in patients with hypertrophic cardiomyopathy.

mean age of 59.3 ± 10.5 years, with 333 (32%) females. Upon admission, mGPS was calculated using blood samples collected from the patients, and their data were obtained from hospital record. Following stent implantation, patients were classified according to the TIMI flow score into two groups: the no-reflow (TIMI 0, 1, 2) group (n=264) and the normal flow (TIMI3) group (n=861). The mGPS classification was determined according to these criteria: Score 0 indicated an albumin level >3.5 g/dL and CRP <1 mg/dL; Score 1 indicated an albumin level >3.5 g/dL and CRP >1 mg/dL; and Score 2 indicated an albumin level <3.5 g/dL and CRP >1 mg/dL.

Results: Significant statistical differences were found among the groups for smoking status (p=0.039), systolic blood pressure (SBP) (p<0.001), ischemia duration (p=0.007), LDL levels (p<0.001), the incidence of RCA as the culprit artery (p=0.033), troponin (p<0.001), BNP (p=0.013), albumin (p<0.001), CRP levels (p<0.001) and mGPS (p<0.001). SBP (OR: 1.012, 95% CI: 1.006-1.018; p<0.001), ischemia duration (OR: 0.996, 95% CI: 0.994-0.998; p<0.001), LDL levels (OR: 0.968, 95% CI: 0.962-0.975; p<0.001), RCA as the culprit artery (OR: 2.318, 95% CI: 1.375-3.912; p=0.002), troponin (OR: 0.996, 95% CI: 0.993-0.999; p=0.002), albumin (OR: 6.593, 95% CI: 3.026-14.365; p<0.001), and mGPS (OR: 27.720, 95% CI: 9.301-82.619; p<0.001) were identified as independent predictors for coronary no-reflow.

Conclusions: The mGPS is an emerging predictor of coronary no-reflow in STEMI patients and could be valuable for risk stratification in those undergoing PCI.

Coronary Artery Disease / Acute Coronary Syndrome

OP-060

Modified glasgow prognostic score predicted coronary flow grade in ST-elevation myocardial infarction

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Background and Aim: The Modified Glasgow Prognostic Score (mGPS), a new immune inflammatory marker based on C-reactive protein (CRP) and albumin levels, has demonstrated prognostic significance in heart diseases. This study investigated the prognostic value of mGPS in predicting thrombolysis in myocardial infarction (TIMI) flow grades in patients presenting with ST-elevation myocardial infarction (STEMI).

Methods: The study involved 1125 STEMI patients who had TIMI 0 flow in the culprit coronary artery and underwent primary percutaneous coronary intervention (PCI) between February 2020 and February 2024. The study cohort had a

Table 1. The Modified Glasgow Prognostic Score

Modified Glasgow Prognostic Score	
0	albumin >3.5 g/dL and CRP <1 mg/dL
1	albumin >3.5 g/dL and CRP >1 mg/dL
2	albumin <3.5 g/dL and CRP >1 mg/dL

Table 2. Baseline and procedural characteristics of thestudy population.

	no-reflow (TIMI 0 1 2) n=179	normal flow (TIMI 3) n=849	p value
Age (years)	60 (58-64)	59 (51-66)	0.0201
Sex (m/%)	123 (68.7)	572 (67.4)	0.727
Diabetes mellitus (n/%)	59 (33)	271 (32)	0.786
Hypertension (n/%)	74 (41.3)	358 (42.2)	0.839
Smoking (n/%)	71 (39.7)	269 (31.7)	0.039
Family history (n/%)	55 (30.7)	275 (32.4)	0.0655
Hyperlipidemia (n/%)	28 (15.6)	151 (17.8)	0.492
BMI (kg/m^2)	25.7 (±3.6)	25.2 (±3.8)	0.149
Systolic P (mmHg)	119.7 (±42.2)	132 (±30.1)	<0.001
Diastolic P (mmHg)	71.5 (±24.2)	74.1 (±15.5)	0.081
Heart Rate (beat/mn)	81.1 (±15.9)	80.5 (±13.2)	0.584
Killip Class III/IV (n/%)	61 (34.1)	224 (26.4)	0.37
CAD history (n/%)	40 (22.3)	161 (19)	0.3
Total ischaemia time (mn)	238 (124-280)	192 (112-270)	0.007
Hemoglobin (g/dL)	13.2 (12.1-15)	13.4 (12.1-14.4)	0.315
WBC count ($10^3/\mu L$)	11.9 (9-14.3)	12.2 (8.3-14.4)	0.940
Platelet count ($x10^9/L$)	255 (170-304)	255 (187-300)	0.824
Glucose (mg/dL)	134 (92-158)	125 (91.4-168)	0.119
Creatinine (mg/dL)	1.02 (0.68-1.06)	0.88 (0.68-1.02)	0.64
LDL-C (mg/dL)	131 (52-168)	102 (68-128)	<0.001
HDL-C (mg/dL)	30 (19.4-36)	30.5 (20-37)	0.661
Triglyceride (mg/dL)	105 (81-151)	113 (81-150)	0.603
CRP (mg/L)	0.92 (0.54-0.97)	0.7 (0.54-0.81)	<0.001
Albumin (mg/dL)	3.78 (3.6-4)	4 (3.7-4.1)	<0.001
Troponin (ng/uL)	173	150	<0.001
BNP (pg/mL)	231 (194-303)	218 (174-267)	0.013
Culprit lesion n (%) LMCA LAD Cx RCA	2.2 36 19.6 41.9	3.1 41.7 26.4 30.6	0.033
Modified Glasgow Prognostic Score (n, %) 0 1 2	 83.8 8.4 7.8	 94.7 3.4 1.9	 <0.001

Table 3. Predictors of no-reflow on Multivariable Analysis

Variables	Univariable p value	OR (95% CI)	Multivariable p value
smoking	0.039		
systolic blood pressure	<0.001	1.012 (1.006-1.018)	<0.001
ischemia duration	0.007	0.996 (0.994-0.998)	<0.001
LDL levels	<0.001	0.968 (0.962-0.975)	<0.001
RCA Occlusion	0.033	2.318 (1.375-3.912)	0.002
troponin	<0.001	0.996 (0.993-0.999)	0.002
BNP	0.013		
albumin	<0.001	6.593 (3.026-14.365)	<0.001
CRP	<0.001		
mGPS (0 vs. ≥1)	<0.001	27.720 (9.301-82.619)	<0.001

Coronary Artery Disease / Acute Coronary Syndrome

OP-061

The prognostic role of residual Syntax score in older patients with acute coronary syndrome

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Background and Aim: Approximately half of the patients presenting with acute coronary syndrome have multivessel disease. There has been conflicting data especially with older people, regarding the residual coronary artery disease burden and its impact on mortality. Therefore, we aimed to assess all-cause mortality and residual coronary artery disease burden in older patients with acute coronary syndrome.

Methods: Patients over 75 years of age who presented with acute coronary syndrome and underwent percutaneous coronary intervention were retrospectively included in the study. After the index procedure, residual SYNTAX scores were calculated and patients were divided into two groups as residual SYNTAX > 8 and < 8. In-hospital and long-term all-cause mortality were defined as the end-point of the study.

Results: Overall, 352 patients were included in the study. Mean age was 82.0 ± 4.8 years and 188 (53.4%) patients were female. Median follow-up was 35 (3-57) months. Both in-hospital and long-term mortality were significantly higher in patients with residual SYNTAX sore >8 (33.9% vs. 12.0% and 70.1% vs. 48.4% both p<0.01 respectively). In Kaplan-Meier analysis

survival curves continued to separate showing increased mortality for patients with residual SYNTAX score >8 (p<0.01). In multivariate Cox regression analysis; high residual coronary artery disease burden [rSS >8, HR: 1.83 (1.30-2.56 95% CI), p<0.01], age, diabetes mellitus, left ventricular ejection fraction and renal insufficiency were associated with long-term allcause mortality.

Conclusions: Elderly patients with residual SYNTAX score >8 had higher in-hospital and long-term all-cause mortality. Strategies aiming to reduce residual coronary artery disease burden by revascularization seems reasonable.

Table 1. Baseline clinic and laboratory

	Original Cohort				Propensity Score Match			
	Total N=352	Syntax<8 N=225 (63.9%)	Syntax>8 N=127 (36.1%)	P value	Syntax<8 N= 85	Syntax>8 N= 85	P value	SMD
Age (y)	82.0 (4.8)	81.8 (4.7)	82.5 (4.8)	0.14	82.5 (4.7)	82.5 (5.0)	0.96	<0.01
Sex (female)	188 (53.4%)	122 (54.2%)	66 (52.0%)	0.68	42 (49.4%)	42 (49.4%)	1.00	<0.01
Diabetes Mellitus	132 (37.5%)	82 (36.6%)	50 (40.3%)	0.49	29 (34.5%)	36 (42.9%)	0.34	0.17
Hypertension	239 (69.1%)	152 (68.2%)	87 (70.7%)	0.62	58 (69.0%)	59 (71.1%)	0.86	0.04
Chronic renal failure	166 (47.3%)	93 (41.5%)	73 (57.5)	<0.01	38 (44.7%)	37 (43.5%)	1.00	0.02
eGFR (ml/min)	61.8 (26.4)	65.0 (25.7)	56.2 (27.6)	<0.01	64.7 (25.6)	66.7 (29.1)	0.47	0.15
Previous PCI	92 (27.5%)	62 (29.0%)	30 (24.8%)	0.41	19 (23.8%)	25 (28.4%)	0.59	0.10
Previous stroke	26 (7.6%)	16 (7.2%)	10 (8.2%)	0.74	8 (9.5%)	9 (9.8%)	1.00	<0.01
STEMI	194 (55.1%)	118 (52.4%)	76 (59.8%)	0.18	56 (65.9%)	50 (58.8%)	0.679	0.146
NSTEMI	127 (36.1)	83 (36.9%)	44 (34.6%)		24 (28.2%)	29 (34.1%)		
UA	31 (8.8%)	24 (10.7%)	7 (5.5%)		5 (5.9%)	6 (7.1%)		
Moderate/severe aortic stenosis	9 (2.9%)	7 (3.4%)	2 (1.9%)	0.73	3 (3.8%)	2 (2.9%)	1.00	0.05
Previous MI	25 (7.1%)	17 (7.8%)	8 (6.4%)	0.64	3 (3.6%)	7 (8.3%)	0.32	0.20
CKD	98 (27.8%)	52 (23.1%)	46 (36.2%)	<0.01	32 (37.6%)	29 (34.1%)	0.74	0.07
Atrial fibrillation	82 (24.0%)	54 (24.7%)	28 (22.8%)	0.69	23 (27.4%)	21 (25.6%)	0.86	0.04
Inotropic therapy	63 (17.9%)	27 (12.0%)	36 (28.3%)	<0.01	15 (17.6%)	24 (28.2%)	0.14	0.25
In-hospital bleeding	38 (11.8%)	19 (10.8%)	34 (33.6%)	0.48	11 (12.9%)	11 (12.9%)	1.00	<0.01
LVEF (%)	45.4 (11.4)	46.4 (11.3)	43.6 (11.3)	0.04	44.1 (11.8)	44.8 (11.2)	0.69	0.06
CRUSADE score	40.0 (35.0)	37.5 (34.1)	43.9 (35.4)	<0.01	38.3 (36.2)	41.7 (35.4)	0.16	0.21
TIMI score	4.5 (2.1)	4.1 (2.1)	5.3 (3.0)	<0.01	4.6 (2.4)	5.2 (3.0)	0.11	0.24
Killip 3-4 heart failure	43 (12.2%)	14 (6.2%)	29 (22.8%)	<0.01	7 (10.6%)	19 (22.4%)	0.06	0.32
Glucose (mg/dl)	172.6 (88.1)	165.8 (83.2)	184.8 (95.4)	0.052	179.9 (86.2)	192.7 (101.1)	0.34	0.14
ALT (IU/L)	19.0 (12.0-27.0)	17.0 (12.0-25.0)	21.0 (14.0-36.5)	<0.01	17.0 (12.0-25.2)	19.5 (14.0-35.0)	0.32	0.21
AST (IU/L)	30.0 (21.0-69.0)	27.5 (20.0-57.0)	36.5 (22.5-85.5)	0.02	32.7 (21.0-66.0)	35.0 (22.0-59.0)	0.89	0.13
CRP (mg/L)	7.9 (3.5-23.3)	6.8 (3.1-15.7)	16.3 (4.5-53.0)	<0.01	6.4 (1.9-18.0)	15.6 (4.8-81.7)	<0.01	0.55
Admission Creatinine (mg/dl)	1.3 (1.0)	1.2 (0.7)	1.5 (1.3)	<0.01	1.22 (0.73)	1.49 (1.34)	0.11	0.24
Max Creatinine (mg/dl)	1.8 (1.1)	1.6 (1.3)	2.3 (1.8)	<0.01	1.8 (1.3)	2.0 (1.8)	0.86	0.13
WBC (x10³)	10.4 (4.3)	9.9 (3.7)	12.1 (4.5)	<0.01	10.0 (3.6)	12.0 (5.4)	<0.01	0.43
Hemoglobin (gr/dl)	12.5 (1.9)	12.6 (1.9)	12.5 (2.0)	0.49	12.7 (1.9)	12.4 (1.9)	0.24	0.18
Platelet (x10³)	240 (71)	229 (70)	260 (81)	<0.01	229 (75)	258 (86)	0.02	0.35
Total cholesterol (mg/dl)	182 (47)	186 (49)	174 (41)	0.04	183 (47)	170 (39)	0.07	0.30
Triglyceride (mg/dl)	123 (51)	121 (52)	128 (48)	0.21	123 (52)	127 (50)	0.63	0.08
HDL cholesterol (mg/dl)	41 (11)	43 (12)	39 (8)	<0.01	41 (12)	39 (8)	0.19	0.26
LDL cholesterol (mg/dl)	119 (42)	122 (44)	113 (36)	0.15	123 (44)	108 (34)	0.10	0.32

Abbreviations: CIN, contrast induced nephropathy; CRP, C reactive protein; HDL, high density lipoprotein; HF, heart failure; LDL, low density lipoprotein; WBC white blood cell

CRUSADE (Can Rapid risk stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation of the ACC/AHA guidelines)

TIMI Thrombolysis In Myocardial Infarction

SMD Standardized mean difference

Table 2. Angiographic characteristics

	Original Cohort				Propensity Score Match			
	Total N=352	Syntax<8 N=225 (63.9%)	Syntax>8 N=127 (36.1%)	P value	Syntax<8 N=85	Syntax>8 N=85	P value	SMD
Multivessel disease	260 (73.9%)	145 (64.4%)	115 (90.6%)	<0.01	71 (83.5)	73 (85.9)	0.83	0.06
LMCA lesion	22 (6.5%)	8 (3.7%)	14 (11.8%)	<0.01	8 (9.6%)	4 (5.0%)	0.37	0.17
CTO	64 (18.2%)	8 (3.6%)	56 (44.1%)	<0.01	5 (5.9%)	33 (38.8%)	<0.01	0.86
Admission syntax score	16 [10-22]	13 [7-19]	21 [16-27]	<0.01	20 [17-23]	19 [15-22]	0.18	0.23
Residual syntax score	5 [3-10]	2 [0- 5]	13 [9-19]	<0.01	3.0 [0-5]	12 [9-15]	<0.01	2.54
Total stent length	33[20-54]	33 [18-56]	33 [23-50]	0.83	48 [25-69]	34 [23-48]	0.01	0.48
DES length	33 [20-53]	31 [19-51]	33 [23-51]	0.58	46 [25-67]	33 [23-48]	0.10	0.34
BMS length	20 [14-36]	18 [13-40]	21 [15-35]	0.59	22 [12-41]	21 [15-34]	0.83	0.06
Stent type								
BMS only	61 (17.8%)	29 (13.1%)	32 (26.7%)	0.01	10 (12.2%)	21 (26.2%)	0.051	0.44
DES only	225 (65.8%)	156 (70.3%)	69 (57.5%)		53 (64.6%)	50 (62.5%)		
DES+BMS	33 (9.6%)	22 (9.9%)	11 (9.2%)		14 (17.1%)	6 (7.5%)		
POBA only	23 (6.7%)	15 (6.8%)	8 (6.7%)		5 (6.1%)	3 (3.8%)		

Abbreviations: BMS, bare metal stent; CTO, chronic total occlusion; DES, drug eluting stent LMCA, left main coronary artery; POBA, plain old balloon angioplasty; SMD, standardized mean difference

Table 3. Outcomes

	Original Cohort				Propensity Score Match		
	Total N=352	Syntax<8 N=225 (63.9%)	Syntax>8 N=127 (36.1%)	P value	Syntax<8 N= 85	Syntax>8 N= 85	P value
In-hospital mortality	70 (19.9%)	27 (12.0%)	43 (33.9%)	<0.01	15 (17.6%)	27 (31.8%)	0.05
1-year all-cause mortality	109 (31.0%)	51 (22.7%)	58 (45.7%)	<0.01	28 (32.9%)	36 (42.4%)	0.26
Long-term all-cause mortality	198 (56.3%)	109 (48.4%)	89 (70.1%)	<0.01	49 (57.6%)	55 (64.7%)	0.43

Table 4. Multivariable Cox Regression analysis

	Univariate Analysis		Multivariate Analysis	
	Hazard Ratio (95% CI)	P Value	Hazard Ratio (95% CI)	P Value
Residual syntax >8	1.99 (1.50-2.65)	<0.01	1.83 (1.30-2.56)	<0.01
Gender (male)	1.14 (0.86-1.51)	0.35	1.24 (0.88-1.77)	0.21
Age	1.08 (1.04.1.11)	<0.01	1.07 (1.03-1.11)	<0.01
Diabetes Mellitus	1.40 (1.05-1.86)	0.01	1.47 (1.04-2.08)	0.02
LVEF	0.95 (0.94-0.97)	<0.01	0.96 (0.94-0.97)	<0.01
eGFR	0.98 (0.98-0.99)	<0.01	0.99 (0.98-0.99)	0.02
Significant bleeding	1.30 (0.84-2.02)	0.23	0.87 (0.51-1.49)	0.62
Admission hemoglobin	0.99 (0.92-1.07)	0.94	1.05 (0.96-1.16)	0.22
CIN	1.65 (1.22-2.21)	<0.01	1.09 (0.74-1.60)	0.64

Abbreviations: CI, Confidence interval; CIN, contrast-induced nephropathy; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction

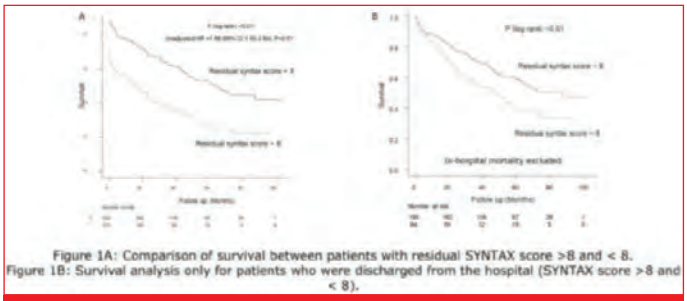

Figure 1A: Comparison of survival between patients with residual SYNTAX score >8 and < 8.
Figure 1B: Survival analysis only for patients who were discharged from the hospital (SYNTAX score >8 and < 8).

Figure 1. Kaplan Meier.

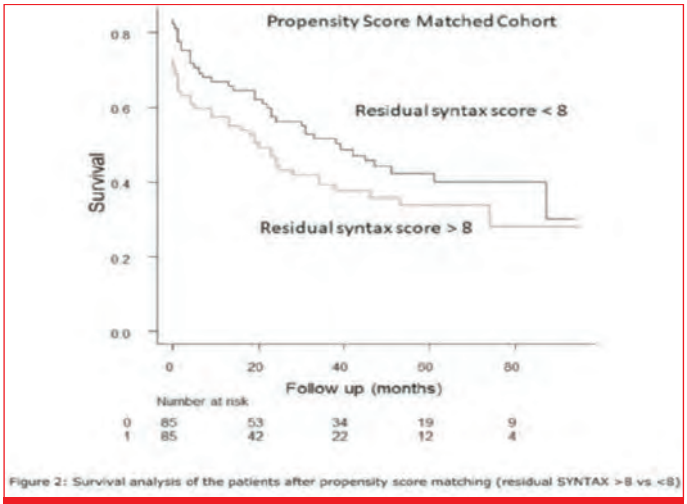

Figure 2: Survival analysis of the patients after propensity score matching (residual SYNTAX >8 vs <8)

Figure 2. Propensity Matched Score Cohort.

Coronary Artery Disease / Acute Coronary Syndrome

OP-062

Evaluation of the relationship between coronary artery disease and retinal vascular density using optical coherence tomography angiography

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Background and Aim: In this study it was aimed to establish a relationship between patients who underwent coronary angiography (CAG) with the suspicion of coronary artery disease (CAD) by determining the severity of CAD with the Gensini score and evaluating the microvascular changes in both CAD and CAD severity and retinal superficial capillary plexus (SCP) with optical coherence tomography angiography (OCTA).

Methods: 120 eyes of 60 patients with coronary artery disease (CAD) who nondiabetic presented with chest pain to the cardiology department's clinic and underwent coronary angiography (CAG) with a diagnosis of stable angina pectoris (SAP) and 117 eyes of 59 patients with normal coronary artery structure were included in the study. The data of the patients included in the study, such as their coronary angiography (CAG) data, demographic characteristics, left ventricular ejection fraction (LVEF), comorbid diseases and laboratory results were retrospectively reviewed. In 3 x 3 mm optical coherence tomography angiography (OCTA) scans, the vascular density (VD) of the superficial capillary plexus (SCP) in different quadrants in the foveal and parafoveal regions, the vascular perfusion percentage (VP) and the width, area, and perfusion rate of the foveal avascular zone (FAZ) were separately scanned and recorded. Coronary artery disease (CAD) (n=60) and control (n=59) groups were established.

Results: When comparing VD (vascular density) parameters between the coronary artery disease (CAD) and control groups, there was a statistically significant decrease in the full, nasal, temporal, and inferior VD parameters in the CAD group (p=0.023, p=0.034, p=0.045, p=0.027 respectively). When VP (vascular perfusion) parameters were compared there was a statistically significant decrease in the inner, full, nasal, temporal, and inferior VP parameters in the CAD group (p=0.039, p=0.029, p=0.042, p=0.034, p=0.041 respectively). In the CAD group, when attempting to establish a relationship between the Gensini score and VD, a significant negative correlation was found between VD inner, full, nasal, temporal, superior, and inferior parameters (p<0.0001, p<0.0001, p=0.020, p=0.001, p=0.002, p=0.002 respectively).

Conclusions: In light of this study and future research, the investigation and diagnosis of retinal vascular diseases in patients diagnosed with CAD (coronary artery disease) can be conducted noninvasively, quickly and practically through OCTA (optical coherence tomography angiography). This will aid in early diagnosis and treatment helping to prevent the reduction or loss of vision. Moreover, coronary artery disease is a common condition in society and one of the leading causes of mortality. The detection of a decrease in retinal VD (vascular density) and VP (vascular perfusion) by OCTA may facilitate the early detection of narrowing in the coronary arteries and, which are structurally similar. Hence, it is concluded that this could be beneficial in reducing mortality related to CAD in the population.

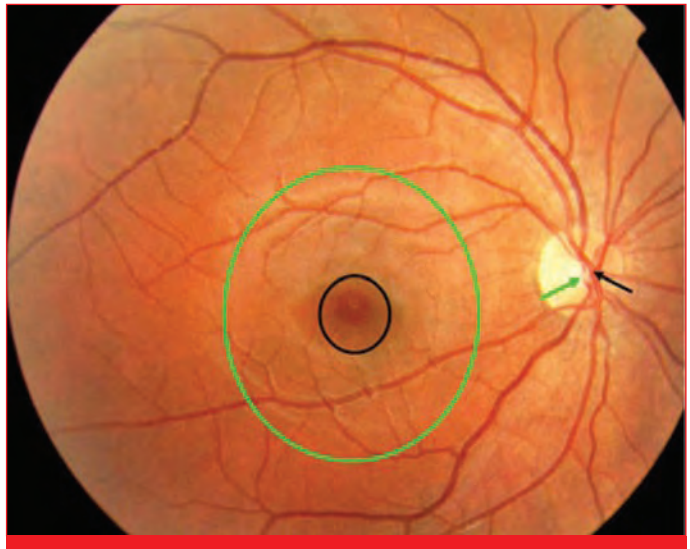

Figure 1. Normal fundus view. Green circle indicates macula, black circle indicates fovea, black arrow indicates central retinal artery, green arrow indicates central retinal vein.

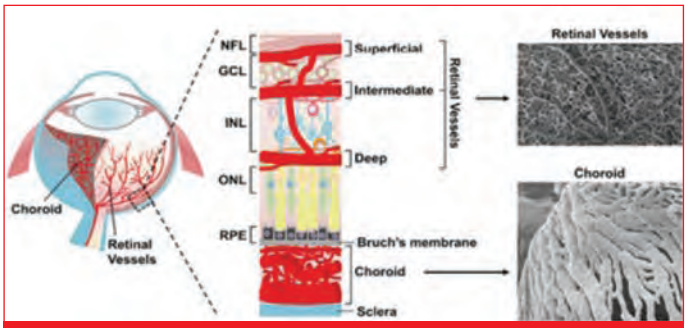

Figure 2. Retinal Microvascular plexus.

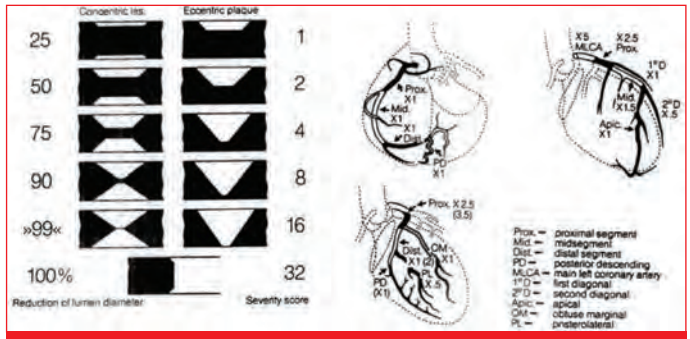

Figure 4. Gensini Score

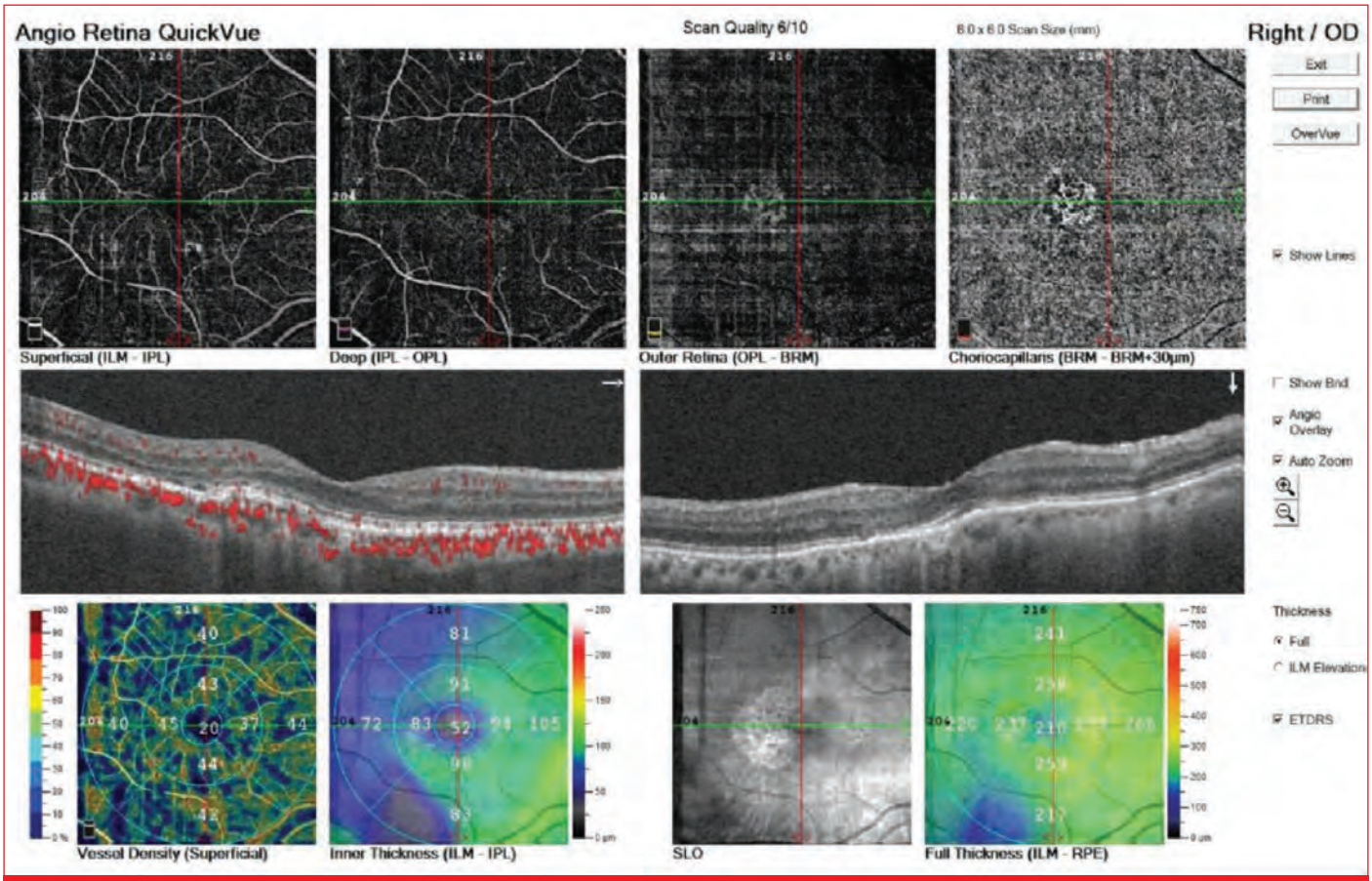

Figure 3. Optical Coherence Tomography Angiography. Examination of the retinal layer and appearance of vascular structures in Optical Coherence Tomography Angiography.

Table 1. Comparison of Gender Characteristics of Control and CAD Group

		Control N (%)	CAD N (%)	p
Gender	Male	25 (42.4%)	33 (55%)	0.168
	Female	34 (57.6%)	27 (45%)	

There is no statistically significant difference between the two groups in terms of gender distribution

Table 2. Comparison of Comorbid Features in Control and CAD Groups

Comorbid		Control N (%)	CAD N (%)	p
HT	Yes	24 (40.7%)	34 (56.7%)	0.081
	No	35 (59.3%)	26 (43.3%)	
Familiy history	Yes	11 (18.6%)	20 (33.3%)	0.068
	No	48 (81.4%)	40 (67.7%)	
Smoking	Yes	24 (25%)	32 (53.3%)	0.167
	No	35 (59.3%)	28 (46.7%)	

There is no statistically significant difference between the two groups in terms of comorbid characteristics

Table 3. Comparison of Age, Gensini score and Laboratory parameters rates between the control and CAD groups

	Control (N=59)		CAD (N=60)		
	Mean	Standard deviation	Mean	Standard deviation	p
Age	54.98	7.396	57.38	8.901	0.113
Gensini score	0.000	0.0000	24.225	18.9671	<0.0001
LDL	121.6017	33.61014	125.7915	39.58506	0.539
HDL	47.4743	9.77148	50.2120	11.30542	0.164
TRIG	143.45	66.417	160.41	97.634	0.275
TK	195.776	40.4189	206.729	53.3970	0.214
HB	13.536	1.6178	13.392	1.5010	0.221
PLT	254.61	67,557	246.92	61.393	0.522
Creatinine	0.7068	0.15606	0.7563	0.14920	0.079
There is no significant difference in age and laboratory parameters between the two groups					

Table 4. Comparison of the rates of VD parameters between the control and CAD groups

	Control (N=59)		KAH (N=60)		
VD (%)	Mean	Standard deviation	Mean	Standard deviation	p
Central	11.4983	3.28792	11.1317	3.36743	0.549
Full	20.8271	1.61865	19.9483	2.43940	0.023
Nazal	22.2636	3.46713	21.0442	2.69334	0.034
Temporal	21.8008	2.06497	20.9650	2.41117	0.045
Superior	21.7246	2.21237	21.0001	2.50047	0.097
	Med [Q1-Q3]		Med [Q1-Q3]		
Inner	21.85 [20.6-23.4]		21.65 [19.1-23.2]		0.132
Inferior	22.75 [20.7-23.5]		21.52 [18.92-22.9]		0.027
There is a statistically significant decrease in VD full, nasal and temporal subparameters in the CAD group					

Table 5. Comparison of the ratios of VP parameters between the control and CAD groups

	Control (N=59)		CAD (N=60)		
VP (%)	Mean	Standard deviation	Mean	Standard deviation	p
Central	19.8186	6.06098	18.8858	6.32253	0.413
Inner	39.1331	2.88185	37.8808	3.60353	0.039
Full	36.9347	3.33467	35.5917	3.29595	0.029
Nazal	38.5669	4.09260	37.0675	3.85060	0.042
Superior	38.5915	4.24835	37.1775	4.00593	0.064
Inferior	39.7441	4.03774	38.2217	4.00887	0.041
	Med [Q1-Q3]		Med [Q1-Q3]		
Temporal	39.75 [37.7-41.45]		38,72 [35.98-40.96]		0.034
There is a statistically significant decrease in VP inner, full, nasal, inferior and temporal subparameters in the CAD group					

Table 6. The relationship between Gensini score and OCTA VD parameters in the CAD group

	Parameters	Gensini Score	
		r	p
VD (%)	Central	-0.229	0.078
	Inner	-0.445	<0.0001
	Full	-0.473	<0.0001
	Nazal	-0.299	0.020
	Temporal	-0.425	0.001
	Superior	-0.390	0.002
	Inferior	-0.391	0.002
According to Pearson correlation, there is a statistically significant inverse relationship between Gensini score and VD internal, complete, nasal, temporal, superior and inferior.			

Table 7. The relationship between Gensini score and OCTA VP parameters in the CAD group

	Parameters	Gensini Score	
		r	p
VP (%)	Central	0.459	<0.0001
	Inner	0.402	0.001
	Full	0.422	0.001
	Nazal	0.265	0.041
	Temporal	0.325	0.011
	Superior	0.363	0.004
	Inferior	0.294	0.022
According to Pearson correlation, there is a statistically significant inverse relationship between Gensini score and VP central, internal, complete, nasal, temporal, superior and inferior.			

Coronary Artery Disease / Acute Coronary Syndrome

OP-063

Genetic variants in EDN1 and PAI-1 and their role in coronary artery disease susceptibility and clinical outcomes

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Background and Aim: Coronary artery disease (CAD) is a condition characterized by the narrowing or blockage of the coronary arteries due to the buildup of atherosclerotic plaques. Genetic factors play a significant role in the progression of CAD by influencing various aspects of lipid metabolism, endothelial function, and inflammation. Endothelin-1 (EDN-1) contributes to CAD by causing vasoconstriction, promoting endothelial dysfunction, and accelerating plaque formation, while Plasminogen Activator Inhibitor-1 (PAI-1) exacerbates CAD by inhibiting fibrinolysis, leading to impaired clot dissolution and increased thrombus risk. Both factors interact to enhance inflammation and plaque instability, further increasing cardiovascular risk. In this study, the objective was to examine the roles of EDN1 rs3087459 and SERPINE1 (PAI-1) 4G/5G (rs1799768) polymorphisms in the development and progression of CAD.

Methods: A group of 1513 individuals who went through coronary angiography were separated into two groups: non-CAD (luminal stenosis ≤30%, n=688) and CAD (luminal stenosis ≥50% in at least one coronary artery, n=845). Peripheral blood samples were taken before the procedure, and DNA was isolated using the salting-out method. The individuals were genotyped for EDN1 rs3087459 and SERPINE1 (PAI-1) 4G/5G (rs1799768) polymorphisms, and statistical analyses were carried out.

Results: In analyses, it was found that myocardial infarction (MI) patients with PAI-1 4G/4G genotype (p=0.016) and carriers of 4G allele (5G/4G and 4G/4G genotypes) (p=0.013) have higher levels of CK-MB compared to patients with 5G/5G genotype. Additionally, CAD patients with the 4G/4G genotype had lower levels of HDL cholesterol compared to 5G/5G genotyped patients (p=0.042). In the analyses that examine the association between CAD and EDN1 rs3087459 genotype, it was found that MI is more prevalent in individuals with CC genotype than AA (p=0.046). In the stable angina pectoris group, the frequency of the CC genotype is significantly higher compared to the non-CAD group (p=0.033).

Conclusions: The study reveals that genetic variants in EDN1 and SERPINE1 are linked to heightened susceptibility and severity of coronary artery disease, with distinct associations observed in myocardial infarction and stable angina pectoris. These insights underscore the potential for integrating genetic testing into CAD risk assessment and management strategies.

Coronary Artery Disease / Acute Coronary Syndrome

OP-064

Dual-faceted risks in coronary artery disease management: Insights from fluid dynamics simulations on left anterior descending artery narrowing

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Background and Aim: Coronary artery disease (CAD) remains a leading cause of morbidity and mortality worldwide, predominantly driven by the development and progression of atherosclerotic plaques. Endothelial shear stress (ESS) has been identified as a crucial factor influencing plaque morphology and stability. Variations in ESS are associated with different phases of plaque development, influencing both the risk of plaque rupture and the propensity for calcification. Understanding the hemodynamic changes induced by plaque progression, particularly in the context of inflammation and narrowing within coronary arteries, is essential for devising more effective therapeutic strategies.

Methods: Coronary angio CT images of a patient with mixed atherosclerotic plaque in the proximal section of the left anterior descending (LAD) artery were utilized for three-dimensional reconstruction of the LAD and left circumflex (LCX) arteries. The reconstructed model served as the basis for computational fluid dynamics (CFD) simulations conducted using Autodesk CFD. Throughout four iterative simulations, inflammation within the LAD was modeled by progressively narrowing the proximal section's diameter by 5 percent per iteration. From these simulations, ESS numerical results were extracted for subsequent analysis (Figure 1).

Results: In the inflamed region (left wall of LAD), ESS was observed to increase with arterial narrowing up to the 10 percent point, reaching a peak mean ESS of 1.8384 Pa (SD ± 0.3915), before decreasing dramatically as the narrowing approached 15 percent, with a mean ESS of 0.7745 Pa (± 0.1619). On the right wall of the LAD, ESS initially increased, peaking at a mean of 1.5841 Pa (± 0.5697) at 5 percent narrowing, and then showed a gradual decline. Notably, the lower quartile ESS values of the right wall (0.95 ± 0.28 Pa) were consistently lower compared to those of the left wall (1.26 ± 0.38 Pa) (Table 1).

Conclusions: The progression of inflammation in the LAD up to a 5 percent narrowing was associated with a significant increase in ESS, suggesting an elevated risk of plaque injury and subsequent emboli formation. Conversely, as narrowing exceeded 10 percent, a notable decrease in ESS was observed, aligning with literature that correlates lower ESS levels with the initiation of calcified, more stable plaque formation. These findings highlight a dual-faceted risk in the management of coronary artery diseases; while reduced ESS at higher degrees of narrowing may decrease the likelihood of embolic events by fostering stable plaque formation, it concurrently solidifies arterial obstructions that diminish blood flow. Thus, the inflammatory progression presents a paradox where initial increases in ESS heighten the risk of embolic events, yet subsequent reductions may lead to the formation of hardened plaques that ultimately restrict arterial patency.

Table 1. Simulation results from different scenarios of narrowing proximal LAD.

Position of the wall	Narrowing percent	Mean ESS (Pa)	Standard deviation	Lower quartile ESS (Pa)
Proximal LAD left wall	0%	1.5307	0.2485	1.4037
Proximal LAD right wall	0%	1.4390	0.4601	1.0204
Proximal LAD left wall	5%	1.7660	0.2947	1.4849
Proximal LAD right wall	5%	1.5841	0.5697	1.0779
Proximal LAD left wall	10%	1.8384	0.3915	1.4646
Proximal LAD right wall	10%	1.4338	0.4285	1.1578
Proximal LAD left wall	15%	0.7745	0.1619	0.6862
Proximal LAD right wall	15%	0.7740	0.2757	0.5341

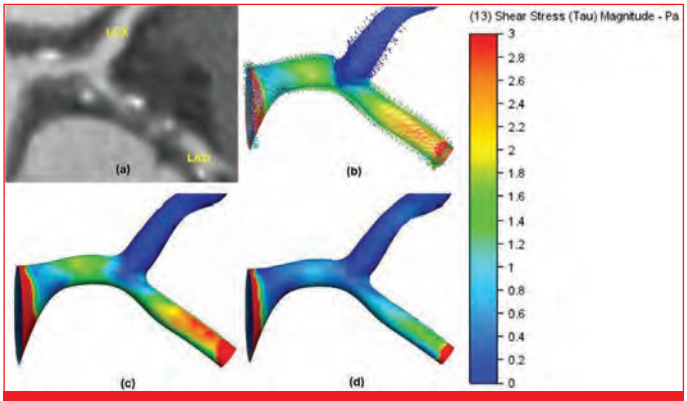

Figure 1. CFD results on proximal LAD: (a) Coronary angio-CT result. (b) ESS results of first iteration including destructive force vectors. (c) ESS results on 10% narrowing scenario. (d) ESS results on 15% narrowing scenario.

Coronary Artery Disease / Acute Coronary Syndrome

OP-065

Evaluating the accuracy of ChatGPT's responses on coronary artery disease

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Background and Aim: Despite developing new treatment options, coronary artery disease is extremely common and is the leading cause of morbidity and mortality worldwide. According to the World Health Organization (WHO), cardiovascular diseases, including coronary artery disease, cause approximately 17.9 million deaths each year. As internet usage continues to rise in the healthcare field, there is growing interest in chatbots. Released on May 13, 2024, ChatGPT version 4o offers significantly advanced features compared to its previous versions, although it requires a monthly subscription fee. The accuracy of ChatGPT's answers regarding frequently asked questions about atrial fibrillation, hypertension, and heart failure has been researched. However, there is no research available in the literature regarding coronary artery disease. In this study, we aimed to evaluate the quality of ChatGPT 4o answers to questions frequently asked by patients about coronary artery disease.

Methods: In this study, fifty commonly asked patient questions about coronary artery disease were identified. The questions were primarily obtained from the frequently asked questions (FAQs) sections on the websites of Cleveland Clinic, Mayo Clinic, and the National Health Service (NHS) UK, based on questions commonly asked by patients. These questions are categorized into five subgroups: basic information, diagnosis, treatment, recovery-operative risks -complications- follow-up, and prevention/dietary aspects. These questions were then posed to the ChatGPT-4o version. The answers were independently rated for accuracy by two experienced cardiologists on a scale of comprehensive/correct (1), incomplete/partially correct (2), a mix of accurate and inaccurate/misleading (3), and completely inaccurate/irrelevant (4). The accuracy of ChatGPT's responses on coronary artery disease has been evaluated.

Results: Overall, the scoring frequencies for ChatGPT-4 were as follows: 14% (7) incomplete/partially correct, and 86% (43) comprehensive/correct. There were no "completely inaccurate/irrelevant" nor a "mix of accurate and inaccurate/misleading" responses. According to category, the model provided "comprehensive/correct" answers to 78.5% of questions regarding "basic knowledge", 80% related to "diagnosis", 80% related to "treatment", 92.3% related to "recovery-operative risks -complications- follow-up", 100% related to "prevention/dietary". Table 1 shows the accuracy levels of ChatGPT's responses by topics.

Conclusions: ChatGPT demonstrated a high accuracy rate in responding to common patient inquiries regarding coronary artery disease. To the best of our knowledge, this is the first study in the literature to evaluate the responses of ChatGPT with coronary artery disease. However, ChatGPT should be used cautiously for collecting medical information about coronary artery disease, as it may occasionally omit important details in its responses.

Table 1. Showing the accuracy levels of ChatGPT's responses by topics

	Comprehensive/Correct	Incomplete/Partially Correct
Basic Knowledge (n=14)	11 (78.5%)	3 (21.5%)
Diagnosis (n=5)	4 (80%)	1 (20%)
Treatment (n=10)	8 (80%)	2 (20%)
Recovery – Operative Risks – Complications – Follow-Up (n=13)	12 (92.3%)	1 (7.3%)
Prevention Dietary (n=8)	8 (100%)	0 (0%)
Total (n=50)	43 (86%)	7 (14%)

Coronary Artery Disease / Acute Coronary Syndrome

OP-066

Relationship with H2FPEF score in MINOCA and INOCA patient groups

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Background and Aim: Myocardial infarction with no obstructive coronary atherosclerosis (MINOCA) is a clinical condition characterized by angiographic evidence of myocardial infarction despite normal coronary arteries or non-obstructive (<50% stenosis) coronary artery disease. MINOCA accounts for 5-10% of all infarctions,with one-year mortality of 3.5% and five-year cardiovascular mortality of 10-25% in MINOCA cases. The underlying pathology may originate from the epicardial coronary arteries (coronary spasm, acute thrombosis due to erosion of non-stenotic plaque, spontaneous coronary artery dissection) or the microvascular system. Ischaemia with no obstructive coronary atherosclerosis (INOCA) is the most common syndrome with symptoms of ischemic heart disease caused by coronary microvascular dysfunction and epicardial coronary vasospasm. Diastolic dysfunction is common in many heart diseases and is an important cause of coronary ischemia. Inflammation is one of the common causes in the pathophysiology of heart failure with preserved ejection fraction (HFpEF), both MINOCA and INOCA, and the comorbidities that trigger microvascular endothelial inflammation are similar in etiology. In our study, we aimed to reveal the relationship between MINOCA and INOCA, which have recently gained importance, and H2FPEF score, which is important in the diagnosis of HFpEF.

Methods: The study included 298 patients who underwent coronary angiography for chronic coronary syndrome or acute myocardial infarction and had normal coronary arteries or coronary stenosis less than 50%. Patients were divided into two groups according to the diagnosis of MINOCA and INOCA and H2FPEF scores were compared.

Results: 150 (50.33%) of the patients included in the study were in the MINOCA group and 148 (49.67%) in the INOCA group. Demographic, angiographic evaluations, echocardiographic findings and laboratory characteristics of the patients are summarized in Table 1 and Table 2. There were significant differences between the groups in terms of hypertension, diabetes mellitus, atrial fibrillation and smoking (Table 1). Troponin (396.24 ± 42.52 vs. 52.51 ± 12.23, p<0.001), NT-proBNP (458 ± 41.35 vs. 153 ± 14.20, p<0.001) and H2FPEF score (6.38 ± 1.3 vs. 4.24 ± 1.4, p<0.001) were significantly higher in the MINOCA group compared to the INOCA group (Table 1). In ROC analysis, the cut-off value of H2FPEF score for MINOCA was determined as 5.3 with 82% sensitivity and 79% specificity (AUC=0.715, 95% CI: 0.648-0.877, p<001) and for INOCA was determined as 3.2 with 73%sensitivity and 72%specificity (AUC=0.681, 95% CI: 0.631-0.853, p<0.001) (Figure 1). Multivariate logistic regression analysis showed that NT-proBNP, troponin, LA size, e/e', LAVI and H2FPEF score were independent predictors in MINOCA and INOCA patients (Table 3).

Conclusions: In the data obtained in our study, the H2FPEF score was found to be significantly higher in the MINOCA group compared to the INOCA group. This scoring system, which is cheap, easy and based on risk factors, should be investigated for HFpEF in MINOCA patients.

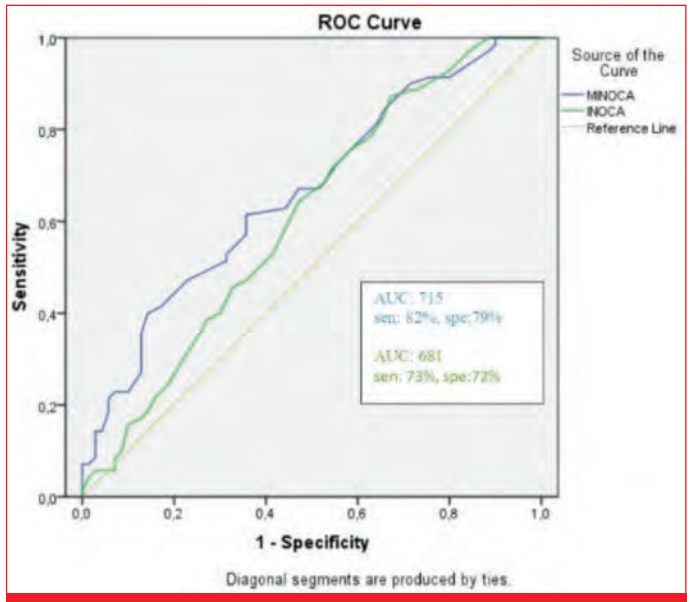

Figure 1. ROC analysis for H2PEF score in MINOCA and INOCA.

Table 1.

variable	MINOCA (n=150)	INOCA (n=148)	p
Demographics features			
Age (years)	64 ± 12.3	65.2 ± 9.2	0.451
Female gender n (%)	63 (42)	61 (41.21)	0.137
BMI kg/m^2	29.22 ± 8.23	28.45 ± 9.3	0.243
CAD n (%)	52 (34.66)	53 (35.81)	0.073
Stroke n (%)	6 (4)	5 (3.37)	0.563
Diabetes mellitus n (%)	66 (44)	59 (39.86)	0.039
Hypertension n (%)	79 (52.66)	66 (44.59)	0.032
Hyperlipidemia n (%)	81 (54)	76 (51.35)	0.130
Smoking n (%)	57 (38)	45 (30.40)	0.025
Atrial fibrillation n (%)	33 (22)	21 (14.18)	0.017
Laboratory findings			
Glucose (mg/dL)	129.23 ± 11.45	131.16 ± 11.23	0.549
Creatinin (mg/dL)	1.13 ± 0.46	1.22 ± 0.37	0.045
BUN (mg/dL)	28.4 ± 11.3	30.3 ± 9.11	0.331
Sodium (mmol/L)	141.32 ± 9.27	142.33 ± 9.34	0.371
Potassium (mmol/L)	4.32 ± 1.20	4.58 ± 0.93	0.383
Albumin (g/dL)	4.29 ± 1.03	4.18 ± 0.99	0.283
ALT (U/L)	25.34 ± 3.45	26.57 ± 4.01	0.174
AST (U/L)	26.26 ± 2.57	27.37 ± 2.18	0.246
TSH (µIU/mL)	1.36 ± 0.87	1.01 ± 0.59	0.124
T4 (µIU/mL)	1.12 ± 0.53	1.25 ± 0.61	0.227
Haemoglobin (g/dL)	10.23 ± 2.27	11.28 ± 1.34	0.479
WBC count (x10^3/µL)	11.56 ± 1.38	11.42 ± 1.83	0.643
LDL cholesterol (mg/dL)	127.18 ± 20.31	129.51 ± 19.54	0.438
Triglyceride (mg/dL)	255.12 ± 11.47	252.12 ± 10.06	0.279
Troponin (ng/mL)	396.24 ± 42.52	52.51 ± 12.23	<0.001
NT-proBNP (pg/mL)	458 ± 41.35	153 ± 14.20	<0.001
H2PEF score	7.38 ± 1.3	4.24 ± 1.4	<0.001

Table 1

variable	MINOCA (n=150)	INOCA (n=148)	p
Demographics features			
Age (years)	64 ± 12.3	65.2 ± 9.2	0.451
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Laboratory findings			
Glucose (mg/dL)			
Creatinin (mg/dL)			
BUN (mg/dL)			
Sodium (mmol/L)			
Potassium (mmol/L)			
Albumin (g/dL)			
ALT (U/L)			
AST (U/L)			
TSH (µIU/mL)			
T4 (µIU/mL)			
Haemoglobin (g/dL)			
WBC count (x10^3/µL)			
LDL-cholesterol (mg/dL)			
Triglyceride (mg/dL)			
Troponin (ng/mL)			
NT-proBNP (pg/mL)			
H2PEF score			

Table 2

variable	MINOCA (n=150)	INOCA (n=148)	p
Angiographic parameters			
LAD lesion n (%)	37 (24.66)	32 (21.62)	0.013
LCX lesion n (%)	45 (30)	43 (29.05)	0.841
RCA lesion n (%)	30 (20)	32 (21.62)	0.714
Three vessel disease n (%)	12 (8)	10 (6.7)	0.170
No lesion n (%)	38 (25.33)	35 (23.64)	0.089
Echocardiographic findings			
LVEF (%)	53.43 ± 11	54.31 ± 9.3	0.231
LA size (mm)	41.25 ± 3.5	37.27 ± 2.1	0.033
sPAP (mmHg)	35.21 ± 5.3	26.11 ± 4.3	<0.001
E/e'	13.45 ± 3.1	9.9 ± 4.3	<0.001
LAVI (mL/m^2)	41.3 ± 5.2	36.5 ± 5.0	<0.001
Medications			
Acetylsalicylic acid n (%)	110 (73.33)	109 (73.64)	0.652
ACE I, ARB n (%)	96 (64)	89 (60.13)	0.090
Beta bloker n (%)	112 (74.66)	100 (67.56)	0.096
Statin n (%)	73 (48.66)	75 (50.67)	0.253
Klopidogrel n (%)	5 (3.33)	3 (2.02)	0.161
Calcium channel blokers n (%)	33 (22)	30 (20.27)	0.198
Oral anticoagulant n (%)	33 (22)	21 (14.18)	0.017

Table 3. Univariate and multivariate regression analysis to identify independent predictors in MINOCA and INOCA patients.

	Univariate Analysis	Univariate Analysis	Multivariate Analysis	Multivariate Analysis
Variable	**Odds Ratio (95% CI)**	**p value**	**Odds Ratio (95% CI)**	**p value**
Hypertension	1.234 (0.652-1.329)	0.032	1.125 (0.965-1.212)	0.129
Smoking	1.312 (0.683-1.432)	0.025	1.237 (1.015-1.323)	0.439
Diabetes mellitus	1.360 (0.982-1.514)	0.039	1.309 (0.987-1.445)	0.010
Atrial fibrillation	1.257 (1.120-1.369)	0.017	1.128 (0.967-1.256)	0.059
Creatinin (mg/dl)	1.236 (0.850-1.324)	0.045	1.157 (0.823-1.245)	0.137
Troponin (ng/mL)	2.173 (1.812-2.310)	<0.001	2.054 (1.649-2.423)	0.037
NT-proBNP (pg/mL)	2.215 (1.818-2.412)	<0.001	2.122 (1.712-2.328)	0.021
LAD lesion	1.347 (1.023-1.432)	0.013	1.241 (1.061-1.345)	0.347
sPAP (mmHg)	1.246 (1.011-1.345)	<0.001	1.234 (1.031-1.324)	0.046
LA size (mm)	1.126 (0.932-1.234)	0.033	1.111 (1.001-1.321)	0.023
E/e'	1.258 (1.102-1.456)	<0.001	1.189 (1.013-1.356)	0.027
LAVI (mL/m^2)	1.312 (1.145-1.478)	<0.001	1.281 (1.019-1.452)	0.011
H2PEF score	2.814 (2.183-3.206)	<0.001	2.736 (2.054-3.162)	<0.001

Coronary Artery Disease / Acute Coronary Syndrome

OP-067

Comparison of percutaneous and surgical revascularization in NSTEMI myocardial infarction patients with chronic kidney failure

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Background and Aim: Renal dysfunction is observed in approximately 40% of patients with acute coronary syndrome (ACS) and chronic kidney failure (CKF). Morbidity and mortality rates increase in this patient group with increased CKF. Within the scope of this research, we aimed to elucidate the effect of percutaneous and surgical revascularization on long-term follow-up results in patients with NSTEMI with CKF.

Methods: A total of 150 patients with a diagnosis of NSTEMI, CKF, and multivessel disease who underwent revascularization therapy (percutaneous or surgical) were enrolled in this prospective research. Chronic kidney failure was analyzed via mild and moderate-severe subgroups. Major adverse cardiac events (MACE) were death, myocardial infarction (MI), and ischemia-driven revascularization.

Results: In our study, 68 patients (45.3%) had percutaneous, and 82 (54.7%) had surgical revascularization. The incidence of mortality in percutaneous and surgical revascularization groups is 7.35% (n=5) and 14.63% (n=12), respectively. In percutaneous and surgical revascularization groups, bleeding rates were 2.94% (n=2) and 20.73% (n=17), respectively. Mortality (p=0.03) and major bleeding (p<0.01) were higher in the CABG group. The patients in Group CABG had significantly higher SYNTAX scores than those in Group PCI (p<0.001). There were significantly more patients with SYNTAX scores ≥33 in Group CABG than in Group PCI (12.3% vs. 8.8%, p<0.001). Long-term outcomes revealed that MACE, mortality, and MI occurred similarly in the two groups, while ischemia-driven revascularization was more frequent in the percutaneous revascularization group (p=0.02). The cumulative incidence of MACE was similar in both groups (p=0.96).

Conclusions: Regarding the outcomes of this research, we found that percutaneous coronary intervention and coronary artery bypass graft surgery performed similar major cardiac adverse event-free survival in the long-term in patients undergoing coronary revascularization for chronic kidney failure and multi-vessel disease accompanied by NSTEMI.

Table 1. Evaluation of groups based on Killip classification, Grace and SYNTAX risk scores, and hemodynamic, electrocardiographic and echocardiographic findings

	Group PCI (n=68)	Group CABG (n=82)	p
Heart rate (beat/minute) §	80.0 [51.0 – 130.0]	88.0 [55.0 – 124.0]	0.001**
Systolic blood pressure (mmHg) †	137.1 ± 19.3	136.1 ± 23.7	0.787***
Diastolic blood pressure (mmHg) †	77.0 ± 12.1	72.2 ± 15.0	0.030***
Changes in ST-segment ‡	44 (64.7)	56 (68.3)	0.772*
Left ventricular ejection fraction (%) ‡			
>50	31 (45.6)	49 (59.8)	0.158*
30-50	31 (45.6)	30 (36.6)	
<30	6 (8.8)	3 (3.7)	
Killip Class 3-4 ‡	4 (5.9)	10 (12.2)	0.298*
GRACE score †	122.7 ± 20.1	112.3 ± 23.7	0.004***
GRACE score categories ‡			
<109	18 (26.5)	38 (46.3)	0.034*
109-140	38 (55.9)	36 (43.9)	
>140	12 (17.6)	8 (9.8)	
SYNTAX score §	16.0 [5.0 – 245.0]	24.0 [10.0 – 45.0]	<0.001**
SYNTAX score categories ‡			
0-22	52 (76.5)	35 (43.2)	<0.001*
23-32	10 (14.7)	36 (44.4)	
≥33	6 (8.8)	10 (12.3)	

‡: n (%), †: mean ± standard deviation, §: median [min-max]
PCI: percutaneous coronary intervention, CABG: coronary artery bypass grafting.
**. Pearson Chi-Square, Fisher's Exact, or Fisher Freeman Halton test.*
***. Mann-Whitney U test.*
****. Independent Samples T-Test.*

Table 2. In-hospital and long-term morbidity and prognostic outcomes of the study groups

	Group PCI (n=68)	Group CABG (n=82)	p
In-hospital outcomes			
Major adverse cardiac event ‡	27 (39.7)	32 (39.0)	0.999*
Reinfarction ‡	2 (2.9)	1 (1.2)	0.590*
Major bleeding ‡	2 (2.9)	17 (20.7)	0.003*
Acute heart failure ‡	4 (5.9)	8 (9.8)	0.570*
Acute renal failure ‡	24 (35.3)	17 (20.7)	0.071*
Length of stay (day) §	6.0 [2.0 – 24.0]	21.0 [7.0 – 99.0]	<0.001**
Mortality ‡	2 (2.9)	10 (12.2)	0.075*
Long-term outcomes			
Duration for follow-up (day) §	517.0 [2.0 – 840.0]	490.5 [11.0 – 1341.0]	0.120**
Major adverse cardiac event ‡	21 (30.9)	18 (22.0)	0.292*
Nonfatal myocardial infarction ‡	14 (20.6)	9 (11.0)	0.162*
Revascularization ‡	13 (19.1)	5 (6.1)	0.028*
Need for hemodialysis ‡	9 (13.2)	9 (11.0)	0.864*
Mortality ‡	3 (4.4)	2 (2.4)	0.659*

‡: n (%), §: median [min-max]
PCI: percutaneous coronary intervention, CABG: coronary artery bypass grafting.
**. Pearson Chi-Square or Fisher's Exact test.*
***. Mann-Whitney U test.*

Coronary Artery Disease / Acute Coronary Syndrome

OP-068

Evaluation of risk factors, diagnosis, treatment and follow-up results in elderly acute coronary syndrome patients; the effect of gender differences on the results

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Background and Aim: With population ageing and rise of life expectancy, older adults represent an increasing proportion of acute coronary syndromes (ACS) patients. Older age has been identified as a major predictor of mortality but patients aged ≥75 years are often under-represented in clinical trials. Older patients have increased prevalence of comorbidities, and also advanced age associated with greater risk of both ischaemic and bleeding events. Therefore advanced age complicate the management of ACS, and there are limited data on the optimal management of older ACS adults. We aimed to determine the risk factors, to evaluate the effect of gender difference, and help to identify treatment strategy for prevention of recurrent cardiovascular events, all cause death in older patients.

Methods: Retrospectively, 1846 patients over the age of 65, who had undergone coronary angiography were screened and we identified 627 patients, ≥75 years of age (34%). Baseline characteristics, risk factors, laboratory analyzes and angi-

Table 1A. Baseline demographic characteristics, clinic presentations, laboratory findings for the study population

Clinical characteristics	Patients with total CAD, n=627	Female patients, n=289	Male patients, n=338	p
Age (years)	81 ± 4.9	81.5 ± 5	80.5 ± 4.7	0.010
BMI (kg/m^2)	28.1 ± 4.8	29.8 ± 5.2	26.7 ± 4	0.001
Smoking, n (%)	114 (18.2)	25 (8.7)	89 (78.4)	<0.001
Diabetes mellitus, n (%)	234 (37.3)	119 (41.2)	115 (34)	0.065
Hypertension, n (%)	414 (66)	214 (74)	200 (59.2)	<0.001
Dyslipidemia, n (%)	292 (51)	129 (48.3)	163 (58.4)	0.221
CKD, n (%)	141 (22.5)	61 (21.1)	80 (23.7)	0.595
Previous CAD, n (%)	175 (28)	50 (17.4)	125 (37)	<0.001
Previous stroke or TIA, n (%)	48 (7.7)	20 (6.9)	28 (8.3)	0.523
Atrial fibrillation, n (%)	120 (19.1)	68 (23.5)	52 (15.4)	0.010
Moderately to severely frail, n (%)	114 (18.2)	66 (22.8)	48 (14.2)	0.006
Clinical presentations, n (%)				
STEMI	208 (33.2)	100 (34.6)	108 (32)	0.483
NSTEMI OR USAP	420 (66.8)	189 (65.4)	230 (68)	0.484
Cardiac arrest	22 (3.5)	10 (3.5)	12 (3.6)	0.855
Dyspnea	182 (28.9)	101 (34.9)	81 (24)	0.001
Angina	336 (53.5)	134 (46.4)	202 (59.8)	0.003
Syncope	11 (1.8)	9 (3.1)	2 (0.6)	0.017
Palpitation	4 (0.6)	3 (1)	1 (0.3)	0.245
Laboratory analysis				
Hemoglobin (mg/dL)	11.98 ± 1.9	11.4 ± 1.7	12.4 ± 2	<0.001
Triglyceride (mg/dL)	131 ± 77	144 ± 89	120 ± 63	<0.001
HDL-C (mg/dL)	44 ± 11	46.6 ± 12	42 ± 11	<0.001
LDL-C (mg/dL)	125 ± 40	129 ± 43	121 ± 36	0.115
CRP (mg/L)	29 ± 44	28 ± 41	31 ± 47	0.428
HsTnI (ng/L)	8892 ± 19424	8817 ± 15570	8958 ± 22348	0.935
NT-pro BNP (ng/L)	8976 ± 14154	10403 ± 14883	7548 ± 13284	0.092
Creatinine	1.3 ± 0.6	1.2 ± 0.5	1.3 ± 0.7	0.011
Glucose	161 ± 84	172 ± 94	152 ± 75	0.003

ography images were initially evaluated. Since 82 patients had no follow-up data, sixth-month and first-year follow-up analysis was performed on 545 patients. Statistical analyses were performed with Statistical Package for Social Sciences version 21.0 software (SPSS Inc, IBM, Armonk, NY).

Results: Mean age was 81 years and median follow-up time was 10.4 ± 10.6 months. The majority (66.8%) of the elderly patients had NSTEMI or USAP followed by STEMI (33.2%) and there was no significant gender difference in terms of ACS type. Baseline demographic characteristics, clinical presentations, laboratory, angiographic findings of the study population are shown in Table 1. When compared with male,female patients had a higher risk of a hospital MACCE (36.4% vs. 27.5%; p=0.019; HR: 1.514; 95% CI: 1.143-2.06) incluiding clinical heart failure (p=0.060), arrhythmia (p=0.067), bleeding especialley access-related p=0.002) and superficial subcutaneous (p=0.089) (Table 2). Clopidogrel was the most preferred P2Y12. Death from any cause occurred in 16.9% patients. There was no significant difference in hospital (11.1% vs. 9.8%; p=0.617) and overall mor-

Table 1B. Angiographic, echocardiographic findings and treatment strategy of the study population

Clinical characteristics	Patients with total CAD, n=627	Female patients, n=289	Male patients, n=338	p
Number of diseased vessels, n (%)				
Single-vessel	187 (29.8)	87 (30.1)	100 (29.6)	0.355
Two-vessel	181 (28.8)	82 (28.4)	99 (29.3)	0.449
Multiple-vessel	186 (29.6)	67 (23.2)	119 (35.2)	0.002
Non-occlusive CAD	73 (11.7)	53 (18.2)	20 (5.9)	<0.001
Coronary reperfusion strategies, n (%)				
PCI	416 (66.3)	176 (60.9)	240 (71)	<0.001
Primary PCI	208 (32.9)	94 (32.5)	112 (33.2)	0.851
Elective or emergent CABG	87 (13.9)	21 (7.3)	57 (16.9)	<0.001
Medical follow-up	172 (27.7)	101 (34.9)	71 (21)	<0.001
Patients undergoing TAVI	33 (5.3)	22 (7.6)	11 (3.3)	0.015
Incomplete revascularization, n (%)	213 (33.9)	99 (34.3)	114 (33.7)	0.871
Access-site (femoral)	432 (68.9)	219 (75.8)	213 (63)	0.001
Medical treatment, n (%)				
Thrombus aspiration	6 (1)	2 (0.7)	4 (1.2)	0.014
Thrombolytic	1 (0.2)	1 (0.3)	0	0.282
Glycoprotein IIb/IIIa inhibitors	15 (2.5)	9 (3.2)	6 (1.8)	0.282
Ticagrelor	98 (16)	39 (13.8)	59 (17.5)	0.158
Prasugrel	1 (0.2)	1 (0.3)	0	0.281
Clopidogrel	437 (71.5)	205 (72.4)	232 (68.6)	0.641
ASA	558 (91.2)	252 (89)	306 (90.5)	0.085
ACEI/ARB	361 (59.7)	169 (60.4)	192 (59.1)	0.844
Beta-blocker	460 (75.5)	200 (70.9)	260 (76.9)	0.014
Statin	498 (79.4)	222 (78.4)	276 (81.7)	0.071
Anticoagulant	79 (12.6)	47 (16.3)	32 (9.5)	0.011
DAT	58 (9.2)	38 (13.2)	20 (5.9)	0.002
Echocardiography (LVEF) %	45.2 ± 11	45.4 ± 11	45 ± 11	0.561

tality (18.3% vs. 15.1%; p=0.288) rates between female and male pateients. There was no decrease in hospital (9.9% in PCI vs. 11.5%; p=0.543) and first-year (2.9% in PCI vs. 1.9%; p=0.459) mortality with PCI but hospital MACCE (36.5% vs. 22%; p=0.000) and sixth-month mortality (4.15% vs. 1.4%; p=0.029) were higher in PCI. In subgroup analysis, hospital mortality (3.5% vs. 8.4%; p=0.040) was reduced with PCI in NSTEMI (Table 3).

Conclusions: Primer PCI has significantly improved hospital mortality, PCI should be considered for all STEMI patients. Frailty was a significant risk factor for in-hospital and long-term mortality and adverse events. Following frailty assessment and comorbidity evaluation it may be reasonable to recommend invasive strategy in addition to an optimal medical therapy taking into account the risks of future cardiovascular events and complications.

Table 2. Clinical outcomes of patients with ACS during hospital stay and follow-up

Clinical outcomes	Total CAD n=627	Female n=289	Male n=338	p
In hospital outcomes, n (%)				
In-hospital mortality	65 (10.5)	32 (11.1)	33 (9.8)	0.617
Arrhythmia	69 (11)	39 (13.5)	30 (8.9)	0.067
In-hospital MACE	197 (31.7)	104 (36.4)	93 (27.5)	0.019
Bleeding complications, n (%)	49 (8)	35 (12.2)	14 (4.1)	0.001
Access-related bleeding (femoral)	25 (4)	19 (6.6)	6 (1.8)	0.002
Gastrointestinal tract bleeding	19 (3)	11 (3.8)	8 (2.4)	0.525
Hematuria	6 (1)	3 (1)	3 (0.9)	0.987
Retroperitoneal hematoma	2 (0.3)	2 (0.7)	0	0.164
Superficial subcutaneous bleeding	3 (0.5)	3 (1)	0	0.089
CIN	152 (24.5)	78 (27)	74 (22)	0.123
Infection	118 (19)	61 (21.1)	57 (17)	0.179
Acute stent thrombosis	6 (1)	4 (1.4)	2 (0.6)	0.306
Subacute stent thrombosis	20 (3.3)	7 (2.4)	13 (3.8)	0.320
Clinical heart failure (Killip class ≥2)	182 (29)	99 (34.4)	83 (24.8)	0.060
Cardiogenic shock	32 (5.1)	18 (6.2)	14 (4.1)	0.240
Long-term outcomes, n (%)				
Follow-up (months)	9.1 ± 10.5	7.9 ± 9.3	10 ± 11.3	0.014
Overall mortality	104 (16.9)	53 (18.5)	51 (15.1)	0.288
Sixth month mortality	20 (3.2)	14 (4.9)	6 (1.8)	0.031
Non-cardiac death	20 (3.2)	10 (3.5)	10 (3)	0.722
Total MACCE	206 (26.6)	113 (27.4)	93 (25.8)	0.688
Sixth month MACCE	80 (10.3)	47 (11.4)	51 (15.4)	0.073
Recurrent MI	150 (16.9)	38 (13.1)	53 (15.7)	0.388
Target vessel revascularization	69 (11)	24 (8.3)	45 (13.3)	0.042
Chronic stent restenosis	34 (5.4)	5 (1.7)	29 (8.6)	<0.001
Stroke	21 (3.3)	11 (3.8)	10 (3)	0.560
Heart failure	190 (30.3)	94 (32.5)	96 (28.4)	0.201
LVEF (% in third month)	47 ± 13.2	46.4 ± 13.5	47.4 ± 12.9	0.505

Table 3. Independent predictors of death and major adverse cardiac and cerebrovascular events (MACCE) in hospital and follow-up period by cox regression analysis of baseline characteristic, clinical features and angiographic findings in ACS patients aged

Variables	p	Exp(B)	95% CI
In-hospital mortality			
Shock	0.000	3.536	1.981-6.311
Frailty	0.001	2.730	1.511-4.931
CIN	0.004	2.513	1.339-4.719
Infection	0.009	2.054	1.193-3.537
STEMI	0.019	1.885	1.109-3.203
LVEF	0.003	0.957	0.930-0.986
Sixth month mortality			
Frailty	0.005	4.211	1.550-11.439
Infection	0.045	2.641	1.023-6.818
NT-proBNP	0.025	1.000	1.000-1.000
First year mortality			
Frailty	0.000	6.984	2.355-20.712
In-hospital MACCE			
Bleeding	0.000	4.530	2.067-9.928
Infection	0.011	2.129	1.190-3.808
Killip class ≥2	0.008	1.615	1.130-2.307
CIN	0.070	1.756	0.956-3.228
HsTnI (ng/L)	0.057	1.000	1.000-1.000
Sixth month MACE			
History of AF	0.017	2.526	1.181-5.404
Frailty	0.047	2.379	1.013-5.586
CIN	0.031	3.150	1.110-8.940
LVEF	0.005	0.954	0.923-0.986

Coronary Artery Disease / Acute Coronary Syndrome

OP-069

Usefulness of uric acid albumin ratio in predicting contrast-induced nephropathy in patients with acute coronary syndrome undergoing percutaneous coronary intervention

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Background and Aim: Contrast-induced nephropathy (CIN) is one of the frequently encountered complications in patients with acute coronary syndrome who undergo percutaneous coronary intervention (PCI). Serum uric acid/albumin ratio (UAR) is a new marker indicating acute kidney injury in patients in intensive care unit. CIN is diagnosed based on a >0.5 mg/dL or 25% increase in baseline creatinine levels within the next 72 hours after PCI. The aim of our study is to investigate the usefulness of UAR in predicting the development of CIN after PCI in patients with acute coranary sendrom (ACS).

Methods: 96 ACS (non-ST segment elevation myocardial infarction-ACS 100%) patients who underwent PCI between August 2022 and June 2024 were included in the study. The patients were divided into two groups as CIN (+) (29.2%) and CIN (-) (70.8%). Patients with a baseline creatinine value above 2.5 mg/dL were excluded from the study. Demographic and laboratory findings of the patients were taken from their medical records.

Results: When CIN (+) and CIN (-) groups were compared, the parameters with a statistically significant difference between them were as follows; while albumin was high in CIN (-); uric acid, baseline creatinine, and UAR were found to be high in CIN (+). In ROC analysis results for UAR, an AUC value of 0.818 was found. This value indicates that UAR has a good ability to distinguish between the CIN (+) and CIN (-) groups. Additionally, the p value is <0.001, confirming that UAR is statistically significant in ROC analysis. The cut-off value is 0.188, which is an optimal discriminative value. Sensitivity is 82.1% and specificity is 76.5%. These results indicate that UAR is a reliable marker for distinguishing between CIN (+) and CIN (-) groups, providing both high sensitivity and specificity.

Conclusions: As a result, the increased UAR can be an independent marker in predicting contrast-induced nephropathy in patients with ACS undergoing PCI.

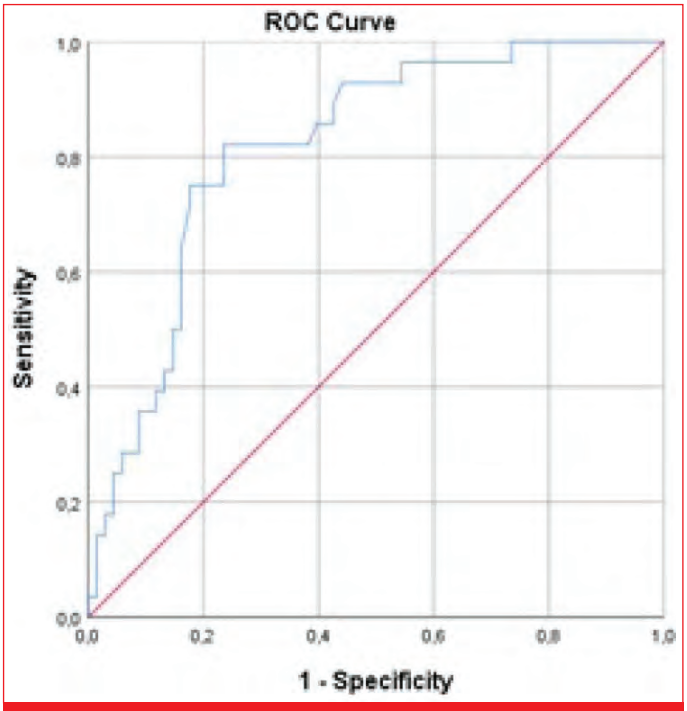

Figure 1. The ROC curve illustrates the performance of UAR. The diagonal red line represents the ROC curve of a random classifier.

Table 1. Comparison of parameters between CIN (+) and CIN (-) groups.

		CIN(+) (N=28)	CIN(-) (N=68)	p-value
		Mean±SD	Mean±SD	
Age		70.29±5.26	66.50±9.05	0.128
Albumin *mg/dl*		32.50±5.65	36.30±6.55	0.007**
Uric Acid *mg/dl*		7.06±0.87	5.78±1.14	<0.001**
1. Day Creatinin *mg/dl*		1.62±0.46	1.30±0.40	0.004**
3. Day Creatinin *mg/dl*		3.28±0.97	1.39±0.46	<0.001**
UAR		0.22±0.05	0.17±0.05	<0.001**
Categorical Parameters		N (%)	N (%)	
GENDER	Female	11 (39.9)	35 (51.5)	0.183
	Male	17 (60.1)	33 (48.5)	
DM	Yes	18 (64.3)	47 (69.1)	0.645
	No	10 (35.7)	21 (30.9)	
HT	Yes	19 (67.9)	54 (79.4)	0.228
	No	9 (32.1)	14 (20.6)	
EF (%)	>50	6 (21.4)	27 (39.7)	0.141
	40-50	8 (28.6)	20 (29.4)	
	<40	14 (50.0)	21 (30.9)	

SD: standard deviation; *: difference is significant at the 0.05 level (two-tailed); **: difference is significant at the 0.01 level (two-tailed).

DM: Diabetes mellitus; HT: Hypertension; EF: Ejection fraction.

Lipid / Preventive Cardiology

OP-070

Determine the status of attitudes and behaviors towards atherosclerosis and dyslipidemia in different clinics across Turkey: Statin Usage (B-Aware dyslipidemia)

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Background and Aim: To examine the attitudes and behaviors of physicians and patients (reflected in physicians) in the clinic during the diagnosis, treatment and follow-up of atherosclerotic cardiovascular disease (ASCVD) and dyslipidemia in order to reveal the current situation in our country in order to support patients to achieve higher rates of targeted outcomes and to work on new solutions.

Methods: In order to investigate the attitudes and behaviors of physicians and patients (as reflected to physicians) towards ASCVD and dyslipidemia treatment, a 26-question questionnaire prepared jointly by endocrinology and cardiology specialist researchers was applied face-to-face to a total of 850 physicians. The Turkish Classification of Territorial Units for Statistics (NUTS-1) was used in order to reflect the results of the survey across Turkey. The number of physicians to participate in the survey was determined according to the weight of the total number of physicians and specialties in the regions. Surveys continued in each region until the targeted number of surveys by specialty was reached. All surveys were completed between November and December 2023.

Results: The opinions and observations of the physicians who participated in the study regarding statin use showed significant differences according to their specialties and the institutions where they worked. Endocrinology and cardiology specialists were the least likely to agree that treatment can be discontinued when cholesterol drops down. Among the reasons for discontinuation of statin use in primary and secondary care hospitals, the responses of patients using too much medication, no benefit from medication and no complaint were reported more frequently.

Conclusions: LDL-C is the primary target of dyslipidemia treatment for cardiovascular protection. Improving patient adherence to statins, which are used as the first choice after lifestyle changes to reduce LDL-C, is an issue that should be prioritized. By analyzing the reasons for low patient compliance with statins in daily life, the proportion of patients reaching the LDL-C target can be increased and cardiovascular events and deaths can be prevented.

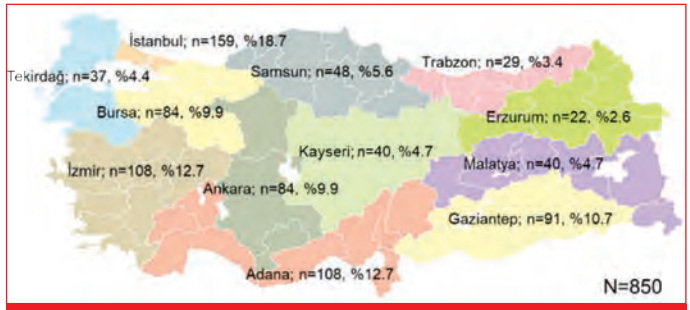

Figure 1.

Figure 2.

		Pratisyen		Aile Hekimi		Dahiliye		Endokrinoloji ve Met.		Kardiyoloji		Diğer		Genel		
		Sıklık	Yüzde	Sıklık	Yüzde	Sıklık	Yüzde	Sıklık	Yüzde	Sıklık	Yüzde	Sıklık	Yüzde	Sıklık	Yüzde	p
Statistin Kullanımı hakkındaki düşünceniz nedir?	Böbrek ve karaciğer hastalığına neden olabilir.	31	12.45%	19	12.10%	52	10.92%	28	14.29%	29	10.10%	23	13.77%	182	12.07%	0.006
	Diabet gelişimini arttırabilir.	31	12.45%	9	5.73%	63	13.24%	27	13.78%	30	10.45%	17	10.18%	177	11.74%	
	Uzun süreli lipid düşürücü tedavi kullanmak etkili ve güvenlidir.	41	16.47%	25	15.92%	122	25.63%	44	22.45%	88	30.66%	37	22.16%	357	23.67%	
	Kolesterol düşünce tedavi kesilebilir.	35	14.06%	23	14.65%	47	9.87%	14	7.14%	20	6.97%	20	11.98%	159	10.54%	
	Statinler yan etkilerinden dolayı uzun dönem kullanılmamalıdır.	39	15.66%	29	18.47%	73	15.34%	27	13.78%	34	11.85%	28	16.77%	216	14.32%	
	Hastanın risk durumuna göre ömür boyu kullanılabilir.	72	28.92%	52	33.12%	119	25.00%	56	28.57%	86	29.97%	42	25.15%	417	27.65%	

Figure 3.

Lipid / Preventive Cardiology

OP-071

Preserved endothelial functions in transgender men: The beneficial role of maintenance dose of testosterone replacement therapy

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Background and Aim: Transgender men (TGM) are individuals assigned female at birth but have a male gender identity. To maintain this phenotypic feature, they have to get a lifelong testosterone replacement therapy. There is controversial data about this therapy impact on vascular functions. We aimed to clarify it by measuring the endothelial functions and compare it with the cisgender healthy counterparts.

Methods: A prospective analysis of TGM was conducted from September 2022 to September 2024. Overall, 38 consecutive TGM admitted for preoperative cardiac evaluation of elective total hysterectomy and bilateral salpingo-oophorectomy were included. At the same time, 10 cisgender women and 17 cisgender men were included for comparison evaluation. After routine biochemical blood tests, electrocardiography and echocardiography were performed. Vascular doppler ultrasonography was used to measure brachial artery flow mediated vasodilatation (FMD) and carotid femoral pulse wave velocity (PWV). A comparative evaluation was done with other cisgender healthy individuals.

Table 1. Demographic features of trans gender men and their healthy counter parts

	Trans gender men (TGM)	Cis gender women (CGW)	Cis gender men (CGM)	p	p TGM vs. CGW	p TGM vs. CGM	p CGW vs. CGM
N	38	10	17				
Age	27.18 ± 6.15	23.50 ± 1.35	22.53 ± 1.58	0.001	0.437	0.001	0.494
Weight (kg)	71.88 ± 13.72	60.8 ± 8.53	75.82 ± 9.46	0.001	0.037	0.804	0.008
Height (m)	1.67 ± 0.048	1.61 ± 0.047	1.77 ± 0.053	0.001	0.009	0.001	0.001
Body mass index	25.61 ± 4.35	23.36 ± 3.84	24.21 ± 2.75	0.212			
Smoking n (%)	26 (68.4)	0	6 (35.3)	0.001	0.001	0.001	0.001
Alcohol n (%)	18 (47.4)	0	1 (5.9)	0.001	0.001	0.001	0.001
Testosterone ester use n (%)	38 (100)						
Time use of Testosterone ester (months)	24 ± 16.97						
Total cholesterol mg/dL	179.89 ± 40.6	170.63 ± 26.68	173 ± 21.26	0.710			
LDL mg/dL	111.42 ± 28.13	93.75 ± 15.7	97.21 ± 24.2	0.035	0.089	0.195	1.000
TG mg/dL	116.53 ± 42.61	74.13 ± 28.15	100.93 ± 50.64	0.041	0.043	0.757	0.499
TSH mU/L	2.57 ± 1.72	2.13 ± 0.58	1.96 ± 0.78	0.571			
Hb g/dL	14.82 ± 1.32	12.77 ± 0.69	15.1 ± 0.67	0.020	0.001	0.673	0.001
Oestradiol pmol/L	96.92 ± 79.69	60.41 ± 40.24	8.94 ± 17.58	0.001	1.000	0.001	0.004
Total testosterone nmol/L	14.01 ± 9.88	1.04 ± 0.54	14.55 ± 2.42	0.001	0.001	0.304	0.001
Free testosterone nmol/L	0.28 ± 0.27	0.013 ± 0.008	0.46 ± 0.09	0.001	0.001	0.002	0.001

Table 2. Echocardiographic features of the study population

	Trans gender men (TGM)	Cis gender women (CGW)	Cis gender men (CGM)	p	p TGM vs. CGW	p TGM vs. CGM	p CGW vs. CGM
N	38	10	17				
LVEF	63.79 ± 2.02	64.7 ± 1.51	63.32 ± 2.13	0.210			
Left ventricle mass index	66.46 ± 12.35	57.35 ± 9.86	70.68 ± 15.34	0.067	0.056	0.232	0.045
LVEDD (cm)	4.4 ± 0.78	4.19 ± 0.43	4.89 ± 0.47	0.003	0.257	0.044	0.003
LVESD (cm)	2.95 ± 0.46	2.64 ± 0.41	3.29 ± 0.43	0.002	0.161	0.037	0.002
IVS (cm)	0.88 ± 0.11	0.76 ± 0.08	0.82 ± 0.15	0.012	0.013	0.343	0.504
Posterior wall (cm)	0.82 ± 0.11	0.76 ± 0.06	0.82 ± 0.11	0.095			

LVEF: Left ventricle ejection fraction; LVEDD: Left ventricle end diastolic diameter; LVESD: Left ventricle end systolic diameter; IVS: Interventricular septum

Table 3. Vascular endothelial function measurement of the study population

	Trans gender men (TGM)	Cis gender women (CGW)	Cis gender men (CGM)	p	p TGM vs. CGW	p TGM vs. CGM	p CGW vs. CGM
N	38	10	17				
FMD	15.55 ± 9.71	17.72 ± 7.48	11.30 ± 9.76	0.179			
Carotid-femoral PWV m/sec	7.45 ± 1.98	6.83 ± 1.69	7.5 ± 2.17	0.775			

FMD: Flow mediated vasodilatation; Carotid femoral PWV: Pulse wave velocity.

Results: TGM were relatively older than cisgender women and cisgender men (27.18 ± 6.15 vs. 23.50 ± 1.35; 22.53 ± 1.58 respectively p<0.001). Body mass index was similar between groups (25.61 ± 4.35 vs. 23.36 ± 3.84; 24.21 ± 2.75 respectively p=0.212). Alcohol use and smoking rate was higher in the TGM group (47.4%, 68.4% respectively). Mean time of testosterone undecanoate use was 24 ± 16.97 months. TGM showed higher levels of low-density lipoprotein (111.42 ± 28.13 vs. 93.75 ± 15.7; 97.21 ± 24.2 mg/dL respectively p<0.035), triglyceride levels (116.53 ± 42.61 vs. 74.13 ± 28.15; 100.93 ± 50.64 mg/dL respectively p<0.041) and hemoglobin (14.82 ± 1.32 vs. 12.77 ± 0.69; 15.1 ± 0.67 respectively p=0.020). Total testosterone levels were higher in the TGM than the cisgender women (14.01 ± 9.88 vs. 1.04 ± 0.54 respectively p<0.001) and similar to the cisgender men (14.01 ± 9.88 vs. 14.55 ± 2.42 nmol/L respectively p=0.34). Left ventricle ejection fraction was normal and similar between the groups (p=0.210). Left ventricle mass index was found normal and similar between the groups. FMD measured from the brachial artery was interestingly found normal and similar with both cisgender women and men (15.55 ± 9.71 vs. 17.72 ± 7.48; 11.30 ± 9.76 respectively p=0.179). In addition, carotid-femoral PWV was found normal and similar to other groups (7.45 ± 1.98 vs. 6.83 ± 1.69; 7.5 ± 2.17 respectively p=0.775).

Conclusions: Physiologic levels of testosterone replacement therapy similar to cisgender men healthy individuals provide preservation of vascular endothelial functions in transgender men.

Lipid / Preventive Cardiology

OP-072

Evaluation of endoplasmic reticulum stress parameters and the relationship of these parameters with cardiovascular risk and coronary artery disease in hypercholesterolemic patients

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Background and Aim: The relationship of endoplasmic reticulum stress (ERS) with dyslipidemia and cardiovascular diseases (CVD) has been tried to be revealed by many different studies. Although it is not fully clarified, the findings suggesting that ERS may play a role in the development of atherosclerotic coronary artery disease (CAD) together with hypercholesterolemia and inflammation are predominantly seen. In this study, it was aimed to examine the ERS parameters in hypercholesterolemia and in individuals with and without CAD [CAD (+)] and [CAD (-)], as well as to evaluate their relationship with the "Systematic Coronary Risk Evaluation 2 (SCORE2)", which indicates CVD risk.

Methods: In our study, 46 dyslipidemic patients diagnosed with hypercholesterolemia and without CAD and not started

on statin therapy yet, 29 healthy control (normocholesterolemic) group who were equal in gender and age and 13 patients with CAD were included in our study. In order to evaluate the endoplasmic reticulum stress state, circulating glucose regulatory protein 78 (GRP78/BiP), inositol dependent kinase 1α (IRE1α), C/EBP homologous protein (CHOP), Caspase-3 levels were measured. CVD risk status of all patients was calculated using the SCORE2 system.

Results: GRP78/BiP, IRE1α, CHOP and Caspase levels were found to be significantly higher of hypercholesterolemic group than normocholesterolemic group, and CAD (+) group than normocholesterolemic and hypercholesterolemic CAD (-) groups. Among the inflammation parameters, interleukin 6 (IL-6) and tumor necrosis factor-α (TNF-α) levels were observed to be significantly higher in the CAD (+) group than in the normocholesterolemic and hypercholesterolemic groups. GRP78, Caspase and TNF-α levels were found to be significantly higher in the SCORE2 high-risk group than in the SCORE2 low- and intermediate-risk groups (p=0.035, p=0.042, p=0.025, respectively). SCORE2 levels showed significant correlations with IRE1α (r=0.22 p=0.037), GRP78 (r=0.33 p=0.002), TNF-α (r=0.34 p=0.001), total cholesterol (r=0.32 p=0.003), LDL-cholesterol (r=0.36 p=0.011) and non-HDL cholesterol (r=0.3 p=0.001) levels. In addition, ERS parameters also showed significant correlations with inflammation and lipid parameters at different levels.

Conclusions: The fact that ERS parameters were significantly higher in CAD (+) patients than in CAD (-) patients and significantly higher in CAD (-) hypercholesterolemia patients than in normocholesterolemic patients supported the finding that ERS may be effective in the development of atherosclerotic CAD. In addition, the fact that the ERS parameters in the high-risk group classified according to SCORE2 were significantly higher than the other low and medium-risk groups, and the significant correlations of ERS parameters with SCORE2 in the entire study group suggest that ERS parameters may contribute to determining the high-risk SCORE2 group.

Anthropometric values of KAH (-) normocholesterolemic and hypercholesterolemic and KAH (+) groups

	CAD (-) normocholesterolemic (n=29)	CAD (-) hypercholesterolemic (n=46)	CAD (+) (n=13)	p
Age (year)	43 (40-46.5)	48[a] (41.5-53)	61[a, b] (53-66)	0.0001
Gender (F/M)	(14/15)	(21/25)	(1/12)	0.03*
SCORE2	2 (1-4)	3 (2-6)	10[a, b] (8.5-14)	0.0001
Smoke (using/not using)	(16/13)	(8/38)[a]	(7/6)[b]	0.001*
Height (cm)	167 (161-176)	168 (162-176)	174 (169-180)	0.106
Weight (kg)	76 (68.5-86)	78 (69-84.5)	80 (67-84)	0.983
BMI (kg/m^2)	26.9 (24.3-30.3)	26.8 (24.4-31.1)	25.7 (24.2-27.5)	0.574
Waist (cm)	90 (79.5-100)	89.5 (80-95)	85 (77.5-94)	0.908
Hip (cm)	103 (100-112)	104 (100-110)	96 (95-108)	0.130
WHR	0.80 (0.80-0.90)	0.85 (0.80-0.90)	0.90 (0.75-0.90)	0.660
Systolic blood pressure (mmHg)	110 (108-120)	120[a] (110-130)	120[a] (120-130)	0.015
Diastolic blood pressure (mmHg)	75 (70-80)	80 (70-80)	80 (70-80)	0.441

p; According to the "Kruskal Wallis" test and the results are given as median [IQR (%25-75)]. (p<0.05) Pairwise group comparison was made according to the Mann-Whitney U test p*: Given according to the "Chi Square" test. Bonferroni correction p<0.017 a: Significantly different compared to the normocholesterolemic group b: Significantly different compared to the hypercholesterolemic group SCORE2: The Systematic Coronary Risk Evaluation 2, F/M: Female/Male; BMI: Body mass index; WHR: Waist hip ratio

Table 2. Endoplasmic reticulum stress and inflammation parameters according to SCORE2 risk status

	Low risk (n=42)	Medium risk (n=32)	High risk (n=14)	p
ERS parameters				
GRP78 (ng/mL)	20 (18.8-27)	20 (19-28.3)	29.5[a, b] (21.8-31.3)	0.035
IRE1α (ng/L)	292 (203-389)	252 (201-391)	394 (300-460)	0.112
CHOP (ng/mL)	11.7 (6.1-17.5)	8.25 (6.23-20.1)	18 (6.9-33.3)	0.321
Caspase (ng/mL)	3.85 (2.98-7.33)	3.45 (2.9-7.58)	7.8[a, b] (3.8-12.7)	0.042
Inflammation Parameters				
IL-6 (ng/L)	114 (75-163)	95 (69-244)	194 (77.5-329)	0.261
TNF-α (pg/mL)	136 (94-241)	150 (106-225)	256[a, b] (144-815)	0.025
CRP (mg/L)	1.95 (1.0-4.15)	1.9 (1.0-3.7)	1.2 (0.645-4.70)	0.342

p; According to the "Kruskal Wallis" test and the results are given as median [IQR (%25-75)]. (p<0.05) Pairwise group comparison was made according to the Mann-Whitney U test a; significant difference compared to the low-risk group b; significant difference compared to the medium-risk group

Table 3. ERS and inflammation parameters of normocholesterolemic, hypercholesterolemic and CAD (+) groups

Parameter	CAD (-) normocholesterolemic (n=29)	CAD (-) hypercholesterolemic (n=46)	CAD (+) (n=13)	p
ERS parameters				
GRP78 (ng/mL)	19 (18-20)	22.5[a] (20-29)	30[a, b] (27.5-38.5)	0.0001
IRE1α (ng/L)	200 (164-268)	349[a] (261-423)	476[a, b] (354-525)	0.0001
CHOP (ng/mL)	6.3 (4.95-11.5)	12.7[a] (6.78-19.2)	31.6[a, b] (21.1-33.4)	0.0001
Caspase (ng/mL)	3.70 (2.90-4.25)	3.70 (2.80-9.20)	11.8[a, b] (7.55-12.6)	0.0001
Inflammation Parameters				
IL-6 (ng/L)	77 (62-118)	141[a] (76.5-241)	323[a, b] (243-332)	0.0001
TNF-α (pg/mL)	124 (87.5-166)	139 (107-221)	667[a, b] (462-883)	0.0001
CRP (mg/L)	1.9 (1.0-3.95)	2.0 (1.0-5.0)	1.4 (0.795-5.25)	0.841
White blood cell (x10^3/μL)	7.3 (6.08-8.24)	6.4 (5.65-7.55)	6.71 (6.14-7.81)	0.228
Leu (x10^3/μL)	2.09 (1.65-2.55)	2.3 (1.9-2.7)	2.0 (1.64-2.61)	0.310
Neu (x10^3/μL)	4.5 (3.3-5.0)	3.7 (2.83-4.35)	3.82 (3.23-5.23)	0.065

p; According to "Kruskal Wallis" test and results are given as median [IQR (%25-75)]. (p<0.05) Pairwise group comparison was made according to Mann-Whitney U test a; significant difference compared to normocholesterolemic group b; significant difference compared to hypercholesterolemic group.

Lipid / Preventive Cardiology

OP-073

Screening for subclinical atherosclerosis in patients with familial hypercholesterolemia

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Background and Aim: Familial hypercholesterolemia (FH) is a monogenic dyslipidemia that leads to early cardiovascular events. Subclinical atherosclerosis is the occurrence of atheromatous plaques in arterial territories before any clinical events. In our study, we investigated the presence, distribution, and independent predictors of subclinical atherosclerosis among patients diagnosed with FH.

Methods: This was a single-center, prospective and cross-sectional study involving a cohort of 215 patients diagnosed with FH. Carotid and femoral ultrasonography were performed, and coronary artery calcium score was measured to screen for subclinical atherosclerosis. Apolipoprotein A-I, apolipoprotein B and Lipoprotein (a) were analyzed using the nephelometric method.

Results: The study cohort comprised 136 females (63%) with a mean age of 54 (43-62) years. The stigmata rate was 18%. Subclinical atherosclerosis was observed in 148 patients (69%), with rates of 48%, 47.5%, and 40.5% in the coronary arteries, carotid bifurcation, and femoral bifurcation, respectively. Advanced age, male sex, high pretreatment LDL-C (Low Density Lipoprotein-Cholesterol) level, diabetes and a low Apo A-I/Apo B ratio were identified as independent predictors of subclinical atherosclerosis. Age, femoral plaque thickness, carotid plaque thickness and CAC (Coronary Artery Calcium) score showed strong correlations with the extent of subclinical atherosclerosis. The rate of statin use during subclinical atherosclerosis screening was 32% and only eight patients (4%) attained LDL-C values <70 mg/dL.

Table 1. Clinical characteristics and laboratory profiles of the study population

Variables	Study Population (n:215)	Definite Clinical Diagnosis (n:120)	Probable Clinical Diagnosis (n:95)	p value
Age (years)	54 (43-62)	55,5 (47,3-62)	51 (41-62)	**0,028**
Gender n (%), (female)	136 (63)	81 (67,5)	55 (58)	0,147
BMI (kg/m²)	27,5 (24,8-30,5)	27,6 (25-30,5)	27,3 (25-30,4)	0,595
Obesity*, n (%)	57 (26,5)	32 (27)	25 (26,5)	0,954
Family CAD History, n (%)	146 (68)	103 (86)	43 (45)	**<0,001**
Smoking, n (%)	113 (53)	66 (55)	47 (49,5)	0,420
Stigmata, n (%)	38 (18)	38 (32)	0 (0)	**<0,001**
Hypertension, n (%)	89 (41,5)	55 (46)	34 (36)	0,138
Diabetes, n (%)	29 (13,5)	22 (18)	7 (7,5)	**0,019**
Pretreatment Total Cholesterol (mg/dl)	319 (290-356)	340 (316-378)	300 (275-311)	**<0,001**
Pretreatment LDL-C (mg/dl)	244 (210-277)	265 (246-293)	211 (200-229)	**<0,001**
Pretreatment nonHDL-C (mg/dl)	263 (234-300)	282 (260-323)	239 (220-260)	**<0,001**
Pretreatment HDL-C (mg/dl)	52 (45-64)	54 (45-64)	50 (46-64)	0,509
Pretreatment Triglyceride (mg/dl)	171 (129-227)	178 (130-229)	170 (129-226)	0,821
Lp (a) (mg/dl)	16 (9-40)	18 (11-45)	14 (6-32)	**0,003**
Apo A-I (mg/dl)	162,9±27,4	162,8±28,9	163±25,4	0,950
Apo B (mg/dl)	126,2±32,1	130,7±35,9	120,6±25,5	**0,016**
Apo A-I/Apo B ratio	1,28 (1,1-1,7)	1,23 (1-1,7)	1,32 (1,1-1,7)	0,138
Lp (a) ≥30 mg/dl, n (%)	67 (31)	41 (34)	26 (28)	0,285
Lp (a) ≥50 mg/dl, n (%)	42 (19,5)	29 (24)	13 (14)	0,054
Apo B ≥130 mg/dl, n (%)	105 (49)	65 (54,2)	40 (42)	0,079
Aortic Valve Disease, n (%)	9 (4)	7 (6)	2 (2)	0,304
Aspirin Use, n (%)	37 (17)	27 (22,5)	10 (10,5)	**0,021**
Statin Use, n (%)	68 (32)	49 (41)	19 (20)	**0,001**
Ezetimibe Use, n (%)	7(3,5)	7 (6)	0	**0,018**
50% reduction in LDL-C level, n (%)	45 (21)	30 (25)	15 (16)	0,099
LDL-C <70 mg/dl, n (%)	8 (4)	3 (2,5)	5 (5)	0,306

BMI: Body Mass Index, CAD: Coronary Artery Disease, Lp (a): Lipoprotein (a), Apo: Apolipoprotein, LDL-C: Low Density Lipoprotein-Cholesterol, HDL-C: High Density Lipoprotein-Cholesterol, Stigmata: (tendon xanthoma, arcus cornealis, xanthelasma), *Obesity: BMI ≥ 30 kg/m²

Conclusions: Subclinical atherosclerosis, as observed in this study, is prevalent among patients with FH. Despite the elevated cardiovascular risk in patients with FH, medication adherence remains suboptimal. Within this high-risk patient group, both the rate of statin use, and the achievement of treatment goals are notably low. Screening for subclinical atherosclerosis may impact the treatment strategies, by the increase physician commitment to treatment protocols and improving patient compliance.

Table 2. Distribution patterns of subclinical atherosclerosis and plaque characteristics

Variables	Study population (n:215)	Definite Clinical Diagnosis (n:120)	Probable Clinical Diagnosis (n:95)	p value
Individuals with Subclinical Atherosclerosis, n (%)	148 (69)	95 (79)	53 (56)	**<0,001**
Subclinical Atherosclerosis Distribution, n (%)	215	120	95	
1 district	53 (25)	32 (27)a	21 (22)a	0,614
2 districts	58 (27)	40 (33)a	18 (19)a	
3 districts	37 (17)	23 (19)a	14 (15)a	
Individuals with Carotid Plaque n (%)	102 (47,5)	63 (52,5)	39 (41)	0,095
Maximum Carotid Plaque Thickness ≥1,5 mm, n (%)	102 54 (53)	63 32 (51)	39 22 (56,5)	0,581
Carotid Plaque Content*, n (%)	102	63	39	
Type 1	19 (18)	9 (14)a	10 (26)a	0,359
Type 2 and 3 (mix)	20 (20)	13 (21)a	7 (18)a	
Type 4	63 (62)	41 (65)a	22 (56)a	
Individuals with Femoral Plaque, n (%)	87 (40,5)	58 (48)	29 (31)	**0,008**
Maximum Femoral Plate Thickness ≥1,5 mm, n (%)	87 47 (54)	58 30 (52)	29 17 (59)	0,543
Femoral Plaque Content*, n (%)	87	58	29	
Type 1	11 (13)	4 (7)a	7 (24)a	0,06
Type 2 and 3 (mix)	14 (16)	11 (19)a	3 (10)a	
Type 4	62 (71)	43 (74)a	19 (66)a	
CAC Score** ≥1, n (%)	188 90 (48)	103 59 (57)	85 31 (36,5)	**0,004**
CAC Score** ≥100, n (%)	188 20 (11)	103 14 (13,5)	85 6 (7)	0,148

CAC: Coronary artery calcium
a: no significant difference was observed in subgroup analysis
*: Plaque characteristics were classified according to the Gray-Weale classification
**: CAC score was calculated over 188 patients.

Table 3. Variables demonstrating strong correlation with the extent of subclinical atherosclerosis

	Study population		Definite Clinical Diagnosis		Probable Clinical Diagnosis	
Variables	r	p value	r	p value	r	p value
Age (years)	0,431	**<0,001**	0,389	**<0,001**	0,404	**<0,001**
Pretreatment LDL-C (mg/dl)	0,328	**<0,001**	0,227	**0,013**	0,262	**0,01**
Pretreatment nonHDL-C (mg/dl)	0,368	**<0,001**	0,283	**0,002**	0,344	**0,001**
Apo A-I (mg/dl)	0,147	**0,031**	0,220	**0,016**	0,058	0,578
Apo B (mg/dl)	0,146	**0,033**	0,146	0,111	0,103	0,322
Lp (a) (mg/dl)	0,084	0,218	0,092	0,320	-0,037	0,724
DLCN Score	0,255	**<0,001**	0,115	0,211	0,175	0,090
Carotid Plaque Thickness (mm)	0,693	**<0,001**	0,653	**<0,001**	0,747	**<0,001**
Femoral Plaque Thickness (mm)	0,724	**<0,001**	0,690	**<0,001**	0,740	**<0,001**
CAC Score (HU)	0,800	**<0,001**	0,754	**<0,001**	0,819	**<0,001**

Apo: Apolipoprotein, Lp (a): Lipoporotein (a), CAC: Coronary artery calcium, DLCN: Dutch Lipid Clinical Network, LDL-C: LDL: Low Density Lipoprotein-Cholesterol, HDL: High Density Lipoprotein-Cholesterol, HU: Hounsfield Unit

Table 4. ROC analysis of variables correlated with the of subclinical atherosclerosis

ROC analysis of variables correlated with the extent of subclinical atherosclerosis					
Variables	AUC (%95 CI)	Cut-off	Sensitivity	Specificity	p value
Age (years)	0,732 (0,652 – 0,812)	≥55,5	%65	%65	**<0,001**
Femoral Plaque Thickness (mm)	0,899 (0,856 – 0,942)	≥0,75 mm	%84	%84	**<0,001**
CAC Score (HU)	0,891 (0,838 – 0,944)	≥19,5	%81	%83	**<0,001**
Carotid Plaque Thickness (mm)	0,872 (0,823 – 0, 921)	≥1,15 mm	%78,4	%79,3	**<0,001**
Pretreatment LDL-C (mg/dl)	0,625 (0,529 – 0,726)	≥246,5	%60	%58	**0,018**
Pretreatment nonHDL-C (mg/dl)	0,665 (0,574 – 0,757)	≥263,5	%60	%61,3	**0,02**
Lp (a) (mg/dl)	0,581 (0,478 – 0,684)	≥17,5	%60	%58	0,127

LDL: Low Density Lipoprotein-Cholesterol, HDL: High Density Lipoprotein-Cholesterol HU:Hounsfield Unit, Lp (a): Lipoporotein (a), CAC: Coronary Artery Calcium

Table 5. Multivariable logistic regression analysis and independent predictors of subclinical atherosclerosis

	Subclinical Atherosclerosis			Coronary Atherosclerosis			Carotid Artery Atherosclerosis			Femoral Artery Atherosclerosis		
Variables	Odds ratio	%95 CI	p value	Odds ratio	%95 CI	p value	Odds ratio	%95 CI	p value	Odds ratio	%95 CI	p value
Age (years)	1,134	1,078 – 1,191	**<0,001**	1,097	1,051 – 1,146	**<0,001**	1,092	1,051 – 1,135	**<0,001**	1,118	1,071 – 1,166	**<0,001**
Male Gender, n (%)	5,225	1,937 – 14,09	**0,001**	3,374	1,361 – 8,360	**0,009**	2,435	1,072 – 5,529	**0,033**	3,250	1,347 – 7,838	**0,009**
BMI (kg/m²)	0,925	0,848 – 1,009	0,078	0,986	0,908 – 1,070	0,730	0,939	0,867 – 1,016	0,118	0,959	0,883 – 1,042	0,325
Smoking, n (%)	1,055	0,512 – 2,174	0,885	1,495	0,748 – 2,989	0,255	1,426	0,746 – 2,726	0,284	2,521	1,261 – 5,040	**0,009**
Hypertension, n (%)	1,125	0,469 – 2,702	0,791	1,675	0,777 – 3,611	0,188	1,666	0,823 – 3,375	0,156	1,408	0,681 – 2,910	0,355
Diabetes, n (%)	4,079	1,006 – 16,55	**0,049**	1,021	0,386 – 2,698	0,967	3,084	1,171 – 8,127	**0,023**	1,285	0,513 – 3,218	0,593
Pretreatment LDL-C (mg/dl)	1,014	1,006 – 1,023	**0,001**	1,011	1,004 – 1,019	**0,004**	1,007	1,002 – 1,013	**0,012**	1,014	1,007 – 1,021	**<0,001**
Lp (a) > 30 mg/dL, n (%)	1,308	0,603 – 2,837	0,497	2,649	1,253 – 5,599	**0,011**	1,494	0,760 – 2,934	0,244	1,149	0,568 – 2,324	0,698
Apo A-I/Apo B ratio	0,442	0,203 – 0,965	**0,040**	0,636	0,311 – 1,303	0,216	0,420	0,210 – 0,842	**0,014**	0,736	0,371 – 1,463	0,382
	Hosmer-Lemeshow Test: 0,542 Nagelkerke R^2 Value: 0,391			Hosmer-Lemeshow Test: 0,280 Nagelkerke R^2 Value: 0,305			Hosmer-Lemeshow Test: 0,166 Nagelkerke R^2 Value: 0,305			Hosmer-Lemeshow Test: 0,521 Nagelkerke R^2 Value: 0,355		

BMI: Body Mass Index, LDL-C: Low Density Lipoprotein Cholesterol, Apo: Apolipoprotein, Lp (a): Lipoprotein (a), CI: Confidence Interval

Lipid / Preventive Cardiology

OP-074

Determine the status of attitudes and behaviors towards atherosclerosis and dyslipidemia in different clinics across Turkey: Length of specialization (B-Aware dyslipidemia)

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Background and Aim: To examine the attitudes and behaviors of physicians and patients (reflected in physicians) in the clinic during the diagnosis, treatment and follow-up of ath-

erosclerotic cardiovascular disease (ASCVD) and dyslipidemia in order to reveal the current situation in our country in order to support patients to achieve higher rates of targeted outcomes and to work on new solutions.

Methods: In order to investigate the attitudes and behaviors of physicians and patients (as reflected to physicians) towards ASCVD and dyslipidemia treatment, a 26-question questionnaire prepared by endocrinology and cardiology specialist researchers was applied face-to-face to a total of 850 physicians. Nomenclature of Territorial Units for Statistics (NUTS-1) was used in order to reflect the results of the survey across Turkey. The number of physicians to participate in the survey was determined according to the weight of the total number of physicians and specialties in the regions. Surveys continued in each region until the targeted number of surveys by specialty was reached. All surveys were completed between November and December 2023. Responses were also analyzed in terms of length of specialty experience (those with more than five years of service in secondary and tertiary hospitals and those with ten years of service or more in other hospitals were defined as having long specialty experience).

Results: The mean frequency of requesting blood lipid measurements, familial hypercholesterolemia differentiation, and prioritization of dyslipidemia assessment in the examination differed according to the length of specialization of the physicians ($p<0.05$). Those with shorter specialty duration were more likely not to routinely order blood lipid values (25.1%), while those with longer specialty duration paid more attention to familial hypercholesterolemia differentiation (67.8% and 47.3%, respectively) than those with shorter specialty duration. The proportion of those who prioritized the evaluation of dyslipidemia in the patient during the examination was 61.4% in those with longer specialty duration and 52.7% in those with shorter specialty duration.

Conclusions: Heart diseases due to atherosclerosis are the leading cause of death in Turkey and worldwide. In our study, the positive correlation between the length of specialization among physicians and measurement of lipid values, differentiation of familial hypercholesterolemia and prioritization of dyslipidemia was remarkable. In order to reduce deaths related to cardiovascular diseases, attitudes and behaviors of physicians and patients should be examined and effective solutions should be developed for the underlying causes.

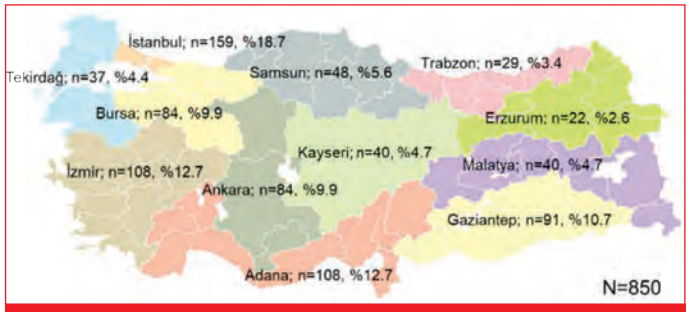

Figure 1.

Figure 2.

		Uzmanlık Süresi Kısa		Uzmanlık Süresi Uzun		Genel		
		Sıklık	Yüzde	Sıklık	Yüzde	Sıklık	Yüzde	p
Kan lipid değerleri ölçümünü ortalama ne sıklıkla istiyorsunuz?	Altı Ayda bir ya da daha sık	89	32.4%	220	38.3%	309	36.4%	0.028
	Yılda bir	84	30.5%	201	35.0%	285	33.5%	
	İki yılda bir ya da az	22	8.0%	43	7.5%	65	7.6%	
	Rutin istemiyorum	69	25.1%	97	16.9%	166	19.5%	
	Hasta Talep Ederse istiyorum	11	4.0%	14	2.4%	25	2.9%	
Ailesel hiperkolesterolemi ayrımına gidiyor musunuz?	Dislipidemi tedavisinde fark olduğunu düşünmüyorum. Ayrıma gitmiyorum.	34	12.4%	74	12.9%	108	12.7%	<0.001
	Ailesel hiperkolesterolemi kriterlerini biliyorum ve kesinlikle dikkat ediyorum.	130	47.3%	390	67.8%	520	61.2%	
	Ayrım için ilgili bölüme sevk ediyorum.	105	38.2%	103	17.9%	208	24.5%	
	Ailesel hiperkolesterolemi hakkında yeterli bilgim yok.	6	2.2%	8	1.4%	14	1.6%	
Muayene sırasında hastada dislipidemi değerlendirmesini önceliklendiriyor musunuz?	Evet	145	52.7%	353	61.4%	498	58.6%	0.002
	Hayır	38	13.8%	52	9.0%	90	10.6%	
	Hasta isterse	28	10.2%	28	4.9%	56	6.6%	
	Anamnez ve fizik muayene bulgularına göre	64	23.3%	142	24.7%	206	24.2%	

Figure 3.

Interventional Cardiology / Valvular and Structural Heart Diseases

OP-017

The role of right heart function in predicting mortality in patients undergoing aortic valve replasment for severe aortic stenosis

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Background and Aim: Aortic valve intervention (AVI) is an indispensable treatment modality for patients with severe aortic valve pathologies. This study aimed to evaluate the contribution of baseline right heart function to classical parameters in determining mortality in patients undergoing AVI for aortic stenosis (AS).

Methods: This was a cross-sectional, retrospective study involving a total of 168 patients who underwent either transcatheter AVI (80 patients) or surgical AVI (88 patients) for severe AS. The main outcome of the study was total mortality monitored during follow-up. Independent predictors of mortality were identified by logistic regression analysis using a baseline model and a full model that included right ventricular function. The performances of the full model and the baseline model were also analyzed.

Results: Mortality was observed in a total of 51 (30%) patients during a median follow-up period of 26 ± 17 months. The mean age of the patients was 69.88 ± 13.55 years. Diabetes mellitus (DM) was present in 75 patients (45%), the mean C-reactive protein level (CRP) was 3 (6), the median tricuspid annular plane systolic excursion (TAPSE) was 20.68 ± 4.15 mm, and the median systolic pulmonary artery pressure (sPAP) was 35.49 ± 15.55 mmHg (p values were 0.009, <0.001, <0.001, <0.001 respectively). Table 1 presented the demographic, clinical, and laboratory characteristics of the study population. Two sets of logistic regression models, baseline and full, were constructed to identify possible determinants associated with total mortality. A baseline model as the reference model was established and adjusted for age, body mass index, DM, coronary artery disease, glomerular filtration rate, CRP, mean aortic valve gradient, aortic valve area, and aortic regurgitation. The final model was revealed by adding TAPSE and sPAP to the baseline model (Table 2). Compared to the baseline model, the inclusion of TAPSE and sPAP in the full model significantly improved model performance (Table 3, Figure 1). Table 2 described parameters associated with mortality. Patients with high sPAP or low TAPSE had a higher mortality rate than patients with low sPAP or high TAPSE (Table 2, Figure 2-3).

Conclusion: Baseline right heart function was a significant predictor of mortality in advanced AS patients undergoing AVI. Consideration of right heart function in the management of patients with severe aortic stenosis will significantly improve survival rates.

Keywords: aortic stenosis, aortic valve intervention, right heart funciton

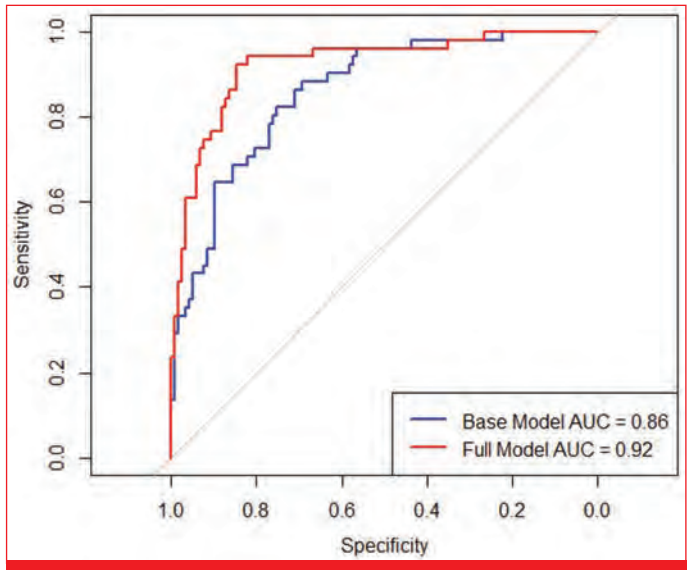

Figure 1. ROC analysis of baseline and full models.

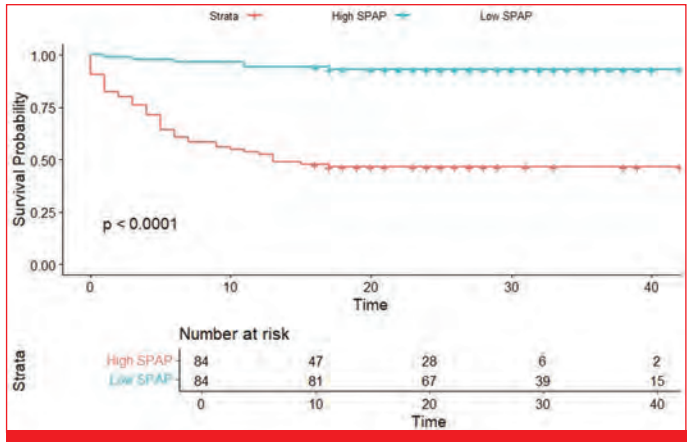

Figure 2. Kaplan-Meier curve showing the probability of survival over time for the two groups with different systolic pulmonary artery pressure levels.

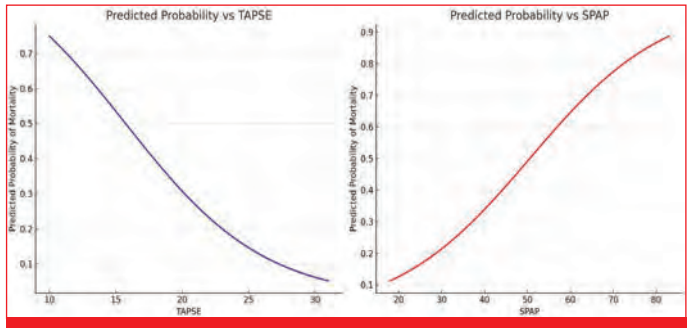

Figure 3. The relationship between TAPSE/ sPAP and mortality probability.

Table 1. Baseline characteristics of the study population

Parameter	All patients (n= 168)	P value
Age, years	69.88 ± 13.55	<0.001
DM, n (%)	75 (45 %)	0.009
HT, n (%)	109 (65 %)	0.885
Smoking, n (%)	50 (28 %)	0.223
AF, n (%)	37 (22 %)	0.033
CVE, n (%)	10 (6 %)	0.081
CAD, n (%)	48 (26 %)	1.000
PAD, n (%)	46 (27 %)	0.905
CKD, n(%)	82 (49 %)	0.087
BMI, kg/m^2	28.59 ± 5.69	0.885
EF, %	55.2 ± 10.30	0.866
Degenerative AS, n (%)	134 (% 79)	0.266
Moderate to severe AR, n (%)	72 (% 43)	0.278
Peak aort velocity, m/s	4.38 ± 0.54	0.561
Peak aort valve gradient, mmHg	78.08 ± 19.83	0.430
Mean aort valve gradient, mmHg	48.28 ± 13.98	0.181
Aort valve area, cm^2	0.71 (0.25)	0.457
TAPSE, mm	20.68 ± 4.15	<0.001
sPAP, mmHg	35.49 ± 15.55	<0.001
Total cholesterol, mg/dL	177.68 ± 46.86	0.073
LDL-C, mg/dL	105.34 ± 42.13	0.112
HDL-C, mg/dL	42.96 ± 12.48	0.843
Triglyceride, mg/dL	141.57 ± 76.68	0.172
eGFR, ml/min/1.73 m^2	70.80 ± 24.00	0.017
CRP, mg/dL	3.00 (6.00)	<0.001
Calcium, mg/dL	9.00 ± 0.58	0.724
Phosphorus, mg/dL	3.78 ± 2.08	0.908

AF; atrial fibrillation, AR; aortic regurgitation, AS, aortic stenosis, BMI; body mass index, CAD; coronary artery disease, CKD; chronic kidney disease, CRP; C- reactive protein, CVE; cerebrovascular events, DM; diabetes mellitus, eGFR; estimated glomerular filtration rate, EF; ejection fraction, HDL- C; high density lipoprotein cholesterol, HT; hypertension, LDL- C; low density lipoprotein cholesterol, PAD; peripheral arterial disease, sPAP; systolic pulmonary artery pressure, TAPSE; tricuspid annular plan systolic excursion.

Table 2. Independent predictors of mortality according to logistic regression analysis

Base Model	OR and 95% CI	p value
Age, years	1.12 (1.035-1.209)	0.005
BMI,kg/m^2	1.05 (0.954 - 1.167)	0.294
DM (yes:no)	2.32 (0.680 - 7.912)	0.179
CAD (yes:no)	1.42 (0.450 - 4.396)	0.542
eGFR, ml/min/1.73 m^2	1.04 (1.001 - 1.090)	0.046
CRP, mg/dL	1.17 (0.908 - 2.048)	0.001
Mean aort valve gradient, mmHg	0.95 (0.317 - 2.857)	0.428
Aort valve area, cm^2	0.93 (0.313 - 2.987)	0.526
AR (Moderate to severe:other)	1.00 (0.552 - 1.816)	0.955
EF, %	1.07 (0.795 - 1.140)	0.030
Full Model		
TAPSE, mm	0.81 (0.704 - 0.926)	0.002
sPAP, mmHg	1.08 (1.038 – 1.124)	<0.001

AR; aortic regurgitation, BMI; body mass index, CAD; coronary artery disease, CI; confidence interval, CRP; C- reactive protein, CVE; cerebrovascular events, DM; diabetes mellitus, eGFR; estimated glomerular filtration rate, EF; ejection fraction, OR; odss ratio, sPAP; systolic pulmonary artery pressure, TAPSE; tricuspid annular plan systolic excursion.

Table 3. Model performance comparison between baseline and full models

Models	AIC	BIC
Base model	205	308
Base model	176	286

AIC; akaike information criterion, BIC; bayesian information criterion

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

OP-034

The Role of Interatrial Septal Thickness for the Prediction of Atrial Tachyarrhythmia Recurrence after Cryoballoon Ablation

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Background and Aim: Interatrial septal thickness (IAST), an indirect marker of interatrial septal fat, and interatrial conduction time (IACT) have been linked to left atrial (LA) fibrosis and atrial fibrillation (AF). This study investigated the potential influence of IAST and IACT on the success of cryoballoon ablation (CBA) for atrial tachyarrhythmia (ATA).

Methods: 75 patients (39 male, mean age 58.3±14.0 years) undergoing their first CBA for pulmonary vein isolation were included. IAST was measured from pre-procedural transesophageal echocardiography (TEE) images, and IACT was calculated from surface ECG and coronary sinus EGM.

Results: The primary endpoint was ATA recurrence (AF, flutter, or tachycardia >30 seconds) after a 3-month blanking period off anti-arrhythmic drugs. During a mean follow-up of 14.2±3.2 months, 7 patients (9%) experienced ATA recurrence. The recurrence group had significantly higher IAST (4.9±0.8 vs. 3.2±0.6 mm, p=0.007) and a higher prevalence of persistent AF (p=0.008). IACT did not differ significantly between the groups (85.8±31.1 vs. 62.2±23.9 ms, p=0.10).

Conclusions: Pre-procedural IAST assessment by TEE may offer prognostic value for CBA patients. Increased IAST is associated with a higher risk of ATA recurrence after CBA, suggesting the need for further investigation.

Keywords: Interatrial septal thickness, atrial tachyarrhythmia, cryoablation

Table 1. Demographic and echocardiographic features of the study population

Variable	ATA Recurrence (-) n=68	ATA Recurrence (+) n=7	P valuev
Sex (male %)	34 (%50)	5 (%72)	0,302
Age	56.2±12.1	62.1±6.7	0,213
BMI (kg/m2)	29.1±0,9	29.2±0,6	0,390
Hypertension	35 (%51)	4 (%57)	0,750
Diabetes Mellitus	15 (%22)	1 (%14)	0,650
CVA history	1 (%1.4)	0 (%0)	0,752
CAD	5 (%7.3)	1 (%14)	0,467
Chronic AF	11 (%16)	4 (%57)	0,008
LA Diameter (mm)	39.6±6,2	43±3,5	0,169
Interatrial conduction time (msn)	85.8±31,1	62.2±23.9	0,102
Interatrial septum thickness (mm)	3.29±0,6	4.97±0,8	0,007

AF; atrial fibrillation, BMI; body mass index CAD; coronary artery disease, CVA; cerebrovascular accident,

Coronary Artery Disease / Acute Coronary Syndrome

OP-059

The Relationship Between The Time of Use of Renin-Angiotensin System Blockers Used in Patients with Acute ST-Segment Elevation Myocardial Infarction and Long-Term Adverse Cardiovascular Events

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Background and Aim: There has been a significant decline in cardiovascular deaths in recent years with the widespread use of primary percutaneous coronary intervention (PPCI) and lifestyle changes in the treatment of ST-segment elevation myocardial infarction (STEMI). Renin-angiotensin system blockers (RAS blockers) are used in the treatment of these patients to reduce long-term adverse cardiovascular events. However, the relationship between administration time of these drugs during the day and cardiovascular outcomes is not fully known. In our study, we aimed to examine the relationship between the administration time (evening vs morning) of RAS blockers during the day and long-term cardiovascular events in patients hospitalized with the diagnosis of STEMI.

Methods: A total of 701 patients who were admitted to our hospital between 2018 and 2020 with a diagnosis of STEMI and underwent PPCI were included in the single-center, retrospective, observational study. Primary endpoints are acute heart failure, non-fatal myocardial infarction, major adverse cardiovascular events (MACE) and long-term mortality. All demographic information, imaging results and clinical outcomes of patients included in the study were recorded, along with their laboratory values during emergency admission and in the first 24 hours after the procedure. Patients were divided into two group according to administration of RAS blockers in the morning (06.00-10.00) and evening (20.00-00.00). Cox regression model was used to identify independent predictors associated with primary outcomes.

Results: The mean age of the total 701 patients included in the study was 57.5±11.9 years and 75.9% were male. After discharge, 485 patients were taking RAS blockers in the morning and 216 patients were taking them in the evening. The frequency of history of heart failure and cardiovascular disease, multivessel disease, history of RAS and beta blocker use and culprit lesion of left anterior descending artery were higher, mean ejection fraction (EF) values and systolic blood pressure were found to be lower and mean heart rate was higher in the group that developed MACE during long-term follow-up after discharge (p<0.01; p<0.01; p<0.01; p=0.01; p=0.01; p=0.03; p<0.01; p<0.01; p=0.01 respectively). In the multivariate COX regression analysis, patient's advanced age, RAS blocker which was taken in the morning, EF measured in the first 24 hours after the procedure and average glucose value independently correlated with MACE. Acute heart failure (AHF), non-fatal myocardial infarction, MACE and death were found to be significantly higher in the group taking RAS blockers in the morning compared to the group taking them in the evening (p<0.01, p=0.02, p<0.01, p <0.01 respectively).

Conclusions: Routine administration of RAS blockers which are included in the long-term treatment of patients with STEMI at bedtime as opposed to upon waking results in significantly diminished acute heart failure, non-fatal myocardial infarction, MACE and death.

Keywords: MACE, RAS blockers, STEMI

Table 1. Comparison of Endpoints by Time of Use of RAS Blockers

Primary Endpoints	All n=701	Awakening n:485 (%69,2)	Bedtime n:216 (%30,8)	P value
Acute Heart Failure n (%)	70 (9,9)	66(9,4)	4(0,5)	<0,01
Nonfatal Myocardial Infarction (%)	55 (7,8)	46(6,6)	9 (1,2)	0,02
MACE n (%)	129 (18,4)	112 (16)	17 (2,4)	<0,01
Death n (%)	73 (10,4)	67 (9,6)	6 (0,8)	<0,01

Table 2. Kaplan-Meier curve showing survival curves for the time of use of Renin-angiotensin Blockers

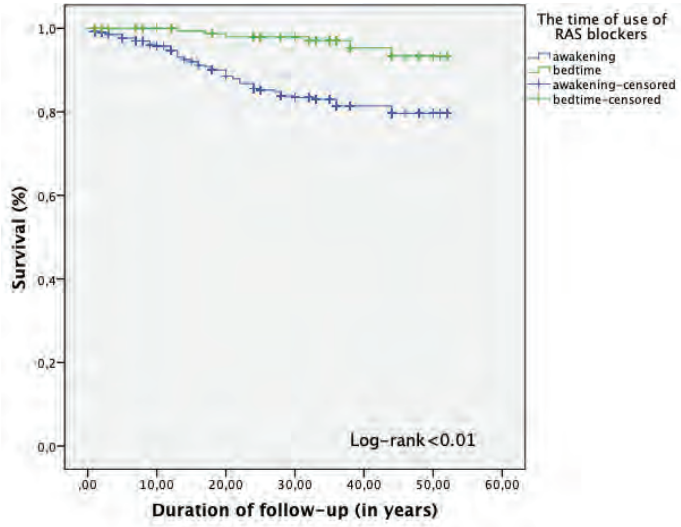

Table 3. Multivariate Cox Regression Analysis for Predicting Long-term Mortality

Variable	HR	CI	P Value
Age	1,10	1,07-1,12	**<0,01**
Multivessel Disease	1,74	1,05-2,88	**0,03**
History of Heart Failure	2,82	1,11-7,12	**0,03**
Time of Use of RAS Blockers*	0,27	0,10-0,68	**<0,01**
EF measured in first 24 hours after the procedure (%)	0,94	0,92-0,96	**<0,01**
Hemoglobin	0,94	0,82-1,07	0,37
CRP	1,002	0,994-1,009	0,67
Glucose	1,004	1,002-1,006	**<0,01**
Sistolic Blood Pressure	0,99	0,98-1,00	0,05
Heart Rate	1,005	0,99-1,01	0,4

*: In SPSS data entry, morning use of RAS blockers are coded as 0 and evening use are coded as 1.

RAS blocker: renin-angiotensin system blocker; CRP: C-reactive protein; EF: Ejection fraction.

(All clinically relevant parameters were included in the model. HR, Hazard Ratio; CI, Confidence Interval)

Table 4. Multivariate Cox Regression Analysis for Predicting MACE

Variable	HR	CI	P Value
Age	1,02	1,01-1,04	**<0,01**
Multivessel Disease	1,28	0,89-1,8	0,17
History of Heart Failure	2,44	1,1-5,4	**0,03**
Time of Use of RAS Blockers*	0,39	0,23-0,68	**<0,01**
EF measured in first 24 hours after the procedure (%)	0,96	0,94-0,97	**<0,01**
Hemoglobin	0,97	0,87-1,07	0,60
CRP	1,003	0,99-1	0,11
Glucose	1,003	1,001-1,004	**<0,01**
Sistolic Blood Pressure	0,98	0,98-0,99	**<0,01**
Heart Rate	1,002	0,99-1,01	0,58

*: In SPSS data entry, morning use of RAS blockers are coded as 0 and evening use are coded as 1.

RAS blocker: renin-angiotensin system blocker; CRP: C-reactive protein; EF: Ejection fraction.

(All clinically relevant parameters were included in the model. HR, Hazard Ratio; CI, Confidence Interval)