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Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PP-001

Effect of noise on the electrocardiographic parameters

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Background and Aim: Noise, defined as any sound that is unpleasant, is one of the most important environmental problems. Prolonged exposure to noise has been shown to be associated with the development of cardiovascular diseases. No study investigated the effect of noise on surface electrocardiography (ECG). We hypothesized that some ECG changes may be seen on the surface ECG before overt heart disease developed in subjects exposed to the noise. The aim of our study is to investigate the effect of noise on surface ECG parameters including P-wave dispersion (PWD), QT intervals, corrected QT interval (QTc), T-wave peak to end (Tp-e) interval, and Tp-e/QT and Tp-e/QTc ratios.

Methods: Fifty-one individuals (46 males, mean age, 35.6 ± 6.3 years) afected the noise and 43 volunteers without any disease (37 males; mean age: 36.0 ± 6.7 years) were included in this study. The average noise level in the textile factory was 112 dB. A 12-lead ECG was obtained from all individuals. PR interval, PWD, QRS duration, QT interval, QTc interval, Tp-e interval, and Tp-e/QT and Tp-e/QTc ratios were calculated for all individuals. In addition, noise group was divided into two groups: those exposed ≥10 years and those exposed <10 years to the noise

Results: Baseline characteristics of study population are presented in Table 1, white blood count (WBC) (p=0.018), neutrophil (p=0.007) and high sensitive C-reactive protein (hsCRP) (p=0.004) were signifcantly higher in the noise group compared to the control group. Electrographic parameters of the study groups are listed in Table 1. When compared to the control group, the noise group had signifcantly prolonged PWD (p=0.029), QT interval (p=0.001), QTc interval (p=0.045), Tp-e interval (p<0.001). In addition, Tp-e/QT (0.24 ± 0.03 vs. 0.23 ± 0.02, p=0.015) and Tp-e/QTc (0.22 ± 0.02 vs. 0.21 ± 0.02, p<0.001) ratios were signifcantly higher in the noise group. To better elucidate the efect of working duration on electrocardiographic parameter, our noise group was divided into two groups as follows: those exposed ≥10 years to the noise (group I, n=26) and those exposed <10 years to the noise (group II, n=25). It was found that individuals in group I had signifcantly prolonged PWD (p<0.001), QT interval (p=0.043), QTc interval (p=0.048), Tp-e interval (p<0.001) and Tp-e/QTc (p=0.020) ratio compared to individuals in group II (Table 2). In addition, the duration of working was positively correlated with PWD (r=0.468, p=0.001), QT (r=0.299, p=0.033), Tp-e interval (r=0.543, p<0.001), and Tp-e/QTc (r=0.328, p=0.019). Multiple linear regression analysis was found that noise was the independent predictor of both PWD (ß=0.244, p=0.032) and Tp-e/QTc (ß=0.319, p=0.003) (Table 2).

Conclusions: We showed that noise signifcantly increased PWD, QT and Tp-e interval measurements, and the duration of noise has an important role on these ECG parameters. In addition, noise was the independent predictor of both PWD and Tp-e/QTc.

Table 1. Basal characteristics and electrocardiography of study population

Variables	Control Group n=43	Noise group n=51	p
Age, years	36.0 ± 6.7	35.6 ± 6.3	0.799
Male gender, %	37 (86)	46 (90)	0.533
BMI, kg/m^2	25.9 ± 4.2	25.8 ± 4.0	0.960
Smoking, %	24 (56)	30 (58)	0.740
Systolic Blood pressure, mmHg	125 ± 9.6	126 ± 10.9	0.601
Diastolic Blood pressure, mmHg	77 ± 8.1	75 ±7.9	0.855
Haemoglobin (g/dL)	14.6 ± 1 .7	14.5 ±1 .9	0.737
WBC (10^3/µL)	8.8 (7.5-10.4)	9.6 (8.6-12.2)	0.018
Platelet (10^3/µL)	215 ± 35	229 ± 41	0.072
Neutrophil (10^3/µL)	4.4 ± 1.4	5.5 ± 2.1	0.007
Lenfosit (10^3/µL)	2.44 ± 0.9	2.7 ± 1.1	0.222
hs-CRP (mg/dL)	0.24 ± 0.11	0.45 ± 0.34	0.004
LVEF, %	55 ±9	56±8	0.987
Left atrium, mm	34 (30-36)	34 (30-35)	0.759
Heart rate, per/min	73 ± 9.8	77 ± 11.5	0.219
PR interval (ms)	152 ± 25	151 ± 23	0.895
P max (ms)	97.6 ± 10	101.3 ± 12	0.122
P min (ms)	68.2 ± 7.5	68.4 ± 6.5	0.899
PWD (ms)	28.0 ± 4.1	33.2 ± 9.2	0.034
QRS duration (ms)	96 ± 10.3	93 ± 10.7	0.140
QT interval (ms)	359.3 ± 2.74	373.5 ± 27.3	0.001
QTc interval (ms)	403 ± 13	409 ± 21	0.045
Tp-e interval (ms)	83.5 ± 7.3	90.6 ± 6.0	<0.001
Tp-e/QT	0.23 ± 0.02	0.24 ± 0.03	0.015
Tp-e/QTc	0.21 ± 0.02	0.22 ± 0.02	<0.001

Aberrations: WBC, white blood count; BMI, body mass index; LVEF, Left ventricular ejection fraction; hsCRP, high sensitive C reactive protein

Table 2. Comparison of ECG parameters between according to noise exposure time, linear regression analysis showing independent predictor of the PWD

Table 2 Comparison of ECG parameters between according to noise exposure time

Variables	Group I n=26	Group II n=25	P
PR interval (ms)	154.7 ± 25.5	147.5 ± 19.8	0.264
PWD (ms)	38.7 ± 7.4	30.0 ± 7.3	<0.001
QRS duration (ms)	91.2 ± 10.5	94.2 ±10.9	0.343
QT interval (ms)	381.3 ± 28.1	365.9 ± 24.7	0.043
QTc interval (ms)	415.8 ± 25.7	404.4 ± 11.1	0.048
Tp-e interval (ms)	94.0 ± 5.9	87.3 ± 3.9	<0.001
Tp-e/QT	0.25 ± 0.02	0.24 ± 0.02	0.216
Tp-e/QTc	0.23 ± 0.02	0.21 ± 0.01	0.020

Group I: exposed ≥10 years to the noise, **Group II:** exposed <10 years to the noise.

Table 3 Univariate linear regression analysis showing independent predictor of the PWD

	Unstandardized coefficients		Standardized coefficients		
	B (Estimate parameter)	SE (Standard error)	β	t	P
Age	0.236	0.138	0.176	1.712	0.090
Gender	3.561	2.858	0.129	1.246	0.216
BMI	0.196	0.228	0.089	0.860	0.216
Smoking	0.142	1.877	0.008	0.076	0.940
hsCRP	3.231	3.297	0.102	0.980	0.330
Left atrium	0.204	0.298	0.071	0.685	0.495
LVEF	0.125	0.110	0.118	1.137	0.259
Noise	4.856	1.789	0.272	2.714	0.008

Table 4 Univariate linear regression analysis showing independent predictor of the Tp-e/QTc interval

	Unstandardized coefficients		Standardized coefficients		
	B (Estimate parameter)	SE (Standard error)	β	t	P
Age	8.356	0.001	0.029	0.280	0.780
Gender	0.005	0.006	0.092	0.885	0.378
BMI	0.001	0.001	0.116	1.122	0.265
Smoking	0.008	0.004	0.205	1.988	0.050
hsCRP	0.015	0.007	0.221	2.174	0.032
Left atrium	0.001	0.001	0.033	0.321	0.749
LVEF	0.001	0.001	0.152	1.476	0.143
Noise	0.014	0.004	0.361	3.713	0.006

Aberrations: β, Standardized β coefficient; BMI, Body mass index; LEVF, left ventricular ejection fraction; hsCRP, high sensitive C reactive protein

ular tachycardia. These studies predominantly advance by examining variables associated with normal cardiac cycles, such as QT interval, QTc interval, and Tpe interval. There is a limited number of studies that explore the relationship between the time intervals of ventricular beats in individuals with ventricular extrasystole and the intervals of normal beats.

Methods: In this study, our aim was to determine the utility of utilizing intervals specific to ventricular extrasystole to predict ventricular tachycardia in individuals with ventricular extrasystole. The study design was retrospective in nature. Holter recordings were retrospectively reviewed, and individuals with solely ventricular extrasystole (Group I) as well as those with both ventricular extrasystole and ventricular tachycardia (defined as a minimum of 4 consecutive ventricular beats) (Group II) were identified. The study variables are presented in Figure 1. Novel variables were derived from the relationships between these variables. To indicate intergroup differences in statistical analysis, the Mann-Whitney U test was employed. The predictive capacity of each parameter for ventricular tachycardia was determined using ROC analysis. A significance threshold of 0.05 was considered for p values.

Results: A total of 202 patients were included in the study (age: 57 ± 17 years; 91 female). Group I comprised 146 patients, while Group II consisted of 56 patients. The p values for variables with significant intergroup differences and the area under the ROC curve values resulting from the ROC analysis are presented in Figure 2. Accordingly, variables with significant differences were identified as coupling time, Tvpa, Tpe-vpa, Tpe1-Tpe2, Tpe-vpa/(Tpe1+Tpe2), and R-on-T (p<0.05).

Conclusions: In conclusion, the utilization of temporal interval values associated with ventricular extrasystole holds potential for predicting ventricular tachycardia. These intervals appear to be applicable, especially within algorithms based on artificial intelligence.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PP-002

Temporal characteristics of ventricular extrasystole predict sustained/non-sustained ventricular tachycardia

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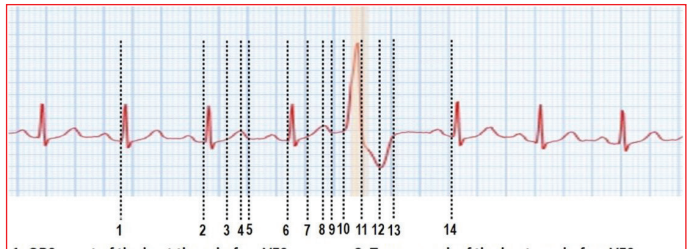
Background and Aim: Ventricular tachycardia is situated within the most severe arrhythmia category in terms of prognosis. It poses a high risk of mortality due to hemodynamic compromise and/or progression to cardiac arrest. Numerous studies are currently being conducted to predict ventricular tachycardias and identify individuals at high risk for ventric-



1: QRS onset of the beat three before VES
2: QRS onset of the beat two before VES
3: T-wave onset of the beat two before VES
4: T-wave peak of the beat two before VES
5: T-wave end of the beat two before VES
6: QRS onset of the beat one before VES
7: T-wave onset of the beat one before VES
8: T-wave peak of the beat one before VES
9: T-wave end of the beat one before VES
10: QRS onset of the VES
11: T-wave onset of the VES
12: T-wave peak of the VES
13: T-wave end of the VES
14: QRS onset of the postextrasystolic beat

RR1: From 1 to 2
RR2: From 2 to 6
Coupling time: From 6 to 10
Compensatory pause: From 10 to 14
T1: From 3 to 5
T2: From 7 to 9
Tpe1: From 4 to 5
Tpe2: From 8 to 9
Tvpb: From 11 to 13
Tpe-vpb: From 12 to 13
R-on-T: From 8 to 10

Figure 1. Definitions of working variables.

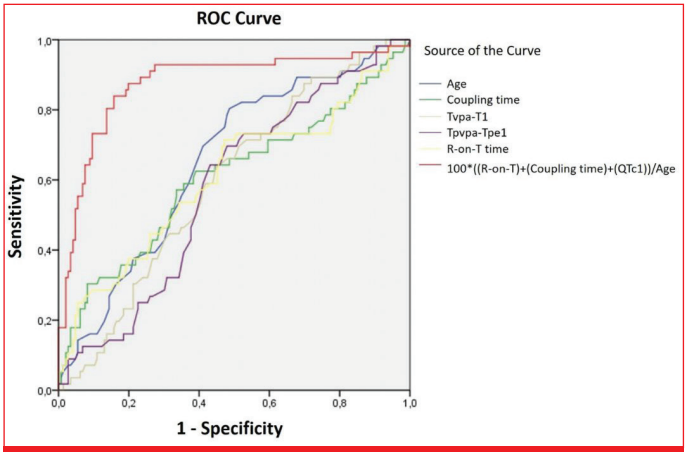


Figure 2. ROC curves for the six parameters with the highest areas under the ROC curve.

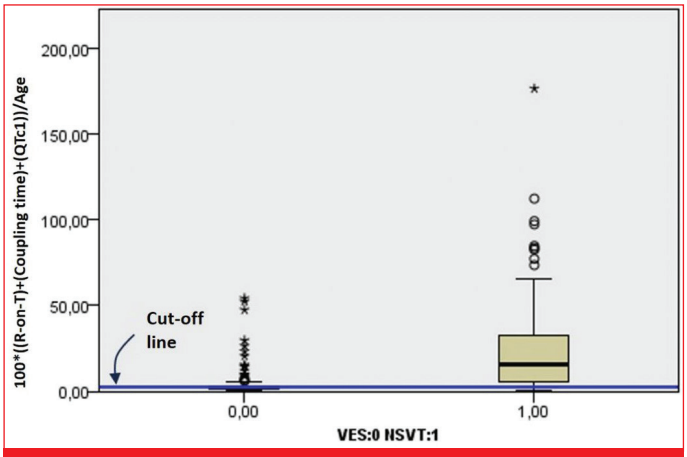


Figure 3. Optimal cut-off value for the variable 100*[(RonT)+(coupling)+(Qtc1)]/age (4.21) and distribution of groups.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PP-003

Balloon-guided subclavian puncture via the median cubital vein for the axillary vein spasm during cardiac electronic device implantation

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Background and Aim: The subclavian or axillary vein is preferred for venous access during cardiac implantable electronic devices (CIED). Puncture of these veins may be difficult in some cases. For this reason, radiopaque imaging can be performed using a vein on the same side as the intervention site. In rare cases, injury to the surrounding tissues or the vein itself may trigger reflex vasospasm in the venous vein. In this case, we performed a balloon-guided subclavian puncture via the median cubital vein, and we implanted a CIED in a patient who developed venous spasms during axillary venous puncture procedures.

Methods: A 55-year-old man with a previous diagnosis of heart failure, hypertension, and diabetes mellitus had short episodes of monomorphic VT on 24-hour Holter monitoring due to palpitations. Echocardiography revealed a left ventricular ejection fraction of 30. Electrocardiography (ECG) showed a QRS width of 130 ms. The patient was taken to the catheterization laboratory for DDD-ICD implantation. The axillary vein was initially preferred for venous access. Despite repeated attempts, venous access was not successful. The road mapping was applied by administering 20 cc of radiopaque material from the median cubital vein. However, after imaging, it was observed that the axillary vein was 100%. Considering venous spasm, 200 µg perlinganit was administered. However, distal flow was not achieved in the occluded lesion. In addition, venous puncture could not be performed despite repeated attempts before occlusion. Subsequently, a 0.014 hydrophilic wire was sent from the median left cubital vein. The total occlusion site was crossed. Then, a 2.5 x 20 mm balloon was sent to dilate the occlusion site (Figure 1). After balloon dilatation, some blood flow was observed distal to the occlusion. It was determined that the venous vasospasm was slightly opened. The balloon was re-inflated, and a successful puncture was performed distal to the balloon marker (Figure 2). Subsequently, atrial and ventricular leads were implanted in the appropriate position, and DDD-ICD was performed.

Results: The most common cause of blood flow obstruction during CIED implantation procedures is anatomical abnormalities at the subclavicular level. Mechanical damage to the subclavian vein caused by venous puncture attempts or vascular spasms can cause slowed or complete obstruction of blood flow. Vascular spasm is usually asymptomatic and resolves spontaneously. Despite the use of nitroglycerin applications, venous spasms may not resolve. Similar to arterial vasospasm, anesthetic agents can be employed to treat venous spasms. Our study showed that balloon dilatation can resolve the spasm and guide us for subclavian venous puncture.

Conclusions: During CIED, axillary or subclavian venous spasms may be observed, although rarely. A median cubital vein approach may be considered to relieve venous spasms and facilitate venous puncture access.

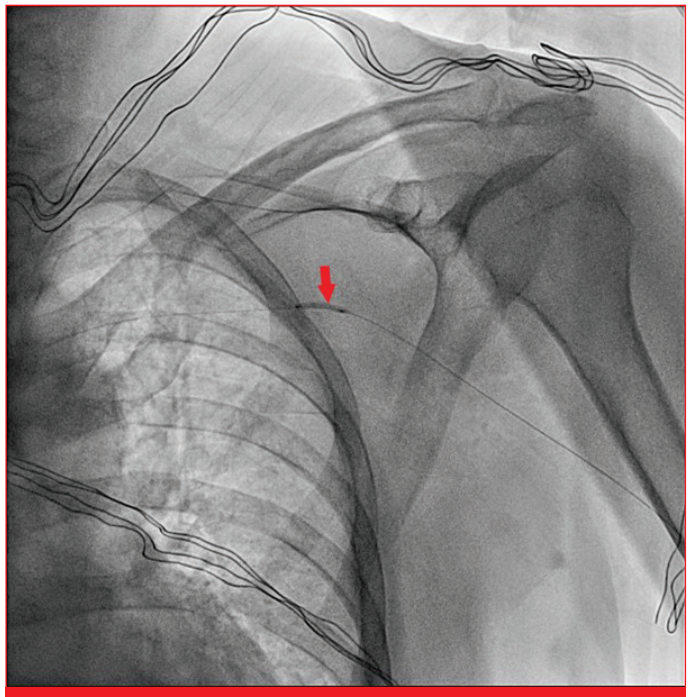

Figure 1. Balloon dilatation of the axillary vein.

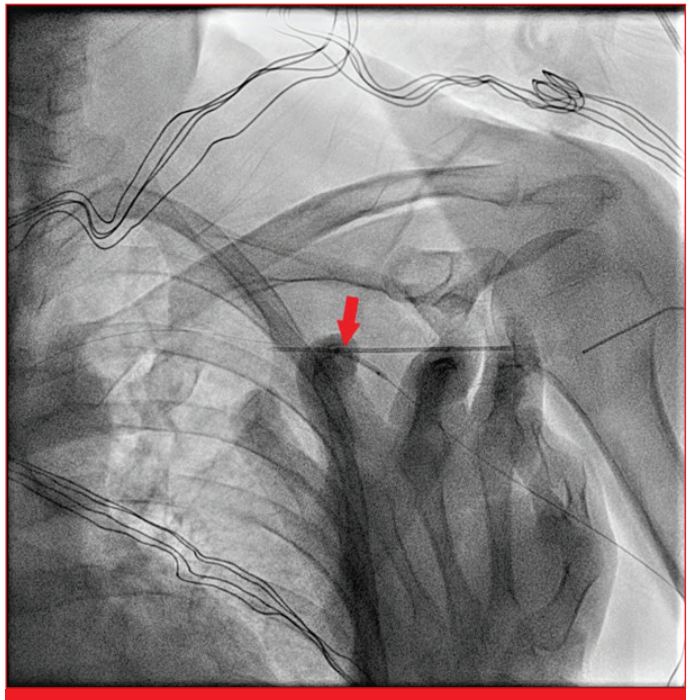

Figure 2. Subclavian venous puncture from distal balloon marker.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PP-004

A novel predictor behind conventional risk factors of new-onset atrial fibrillation after off-pump coronary artery bypass graft surgery: The triglyceride-glucose index

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Background and Aim: Atrial fibrillation (AF), which frequently occurs after coronary artery bypass graft surgery (CABG), raises the risk of adverse events. Therefore, it may be important to identify patients at high risk of postoperative AF (POAF) before cardiac surgery. Insulin resistance (IR) is a major risk factor for developing POAF, and the triglyceride-glucose (TyG) index offers a more precise and reliable assessment of IR compared to the homeostatic model. This study aimed to investigate the correlation between preoperative TyG index levels and POAF in patients undergoing off-pump CABG.

Methods: This cross-sectional study enrolled 416 patients who received off-pump CABG at the Faculty of Medicine, Karabük University, from January 2015 to February 2023 and had no previous history of AF. The inclusion criteria included being 18 years or older, undergoing their first off-pump CABG procedure after acute coronary syndrome or elective coronary angiography, and having normal sinus rhythm. Cardiac rhythm assessment was performed using bedside monitors and twelve-lead electrocardiograms (ECGs) immediately after the surgery and daily until patients were discharged. All ECG recordings obtained from patient files and clinical observation records were used. Postoperative AF was described as AF lasting at least 5 minutes or requiring cardioversion with electrical or antiarrhythmic drugs. Categorical variables were assessed through the chi-square test. The TyG index's predictive threshold for POAF was established via receiver operating characteristic (ROC) analysis. Significant univariate analysis variables ($p<0.05$) were included in multivariate logistic regression models employing the backward method. The TyG index's predictive ability for POAF was compared in two models: one incorporating conventional risk factors from literature, and the other including those factors plus the TyG index. A significance level of $p<0.05$ determined statistical significance.

Results: Four hundred sixteen patients undergoing isolated off-pump CABG were included in the study. The preoperative TyG index and the presence of POAF were evaluated in all patients. POAF was detected in 106 patients (25.5%), with significantly higher TyG index levels observed in POAF patients compared to those without POAF [9.8 (8.0-11.7) vs. 9.0 (7.7-11.1), $p<0.001$]. The TyG index was found to be an independent predictor of POAF ($p<0.001$) with moderate predictive power (AUC=0.767). Adding the TyG index to the model, which included age and left atrial diameter, significantly improved its predictive capacity for POAF ($p=0.038$).

Conclusions: In the patients undergoing off-pump CABG, the TyG index was a notable independent predictor for POAF, regardless of their diabetic status. However, more large-scale multicenter and prospective studies are needed to confirm its efficacy before it can be used in clinical practice.

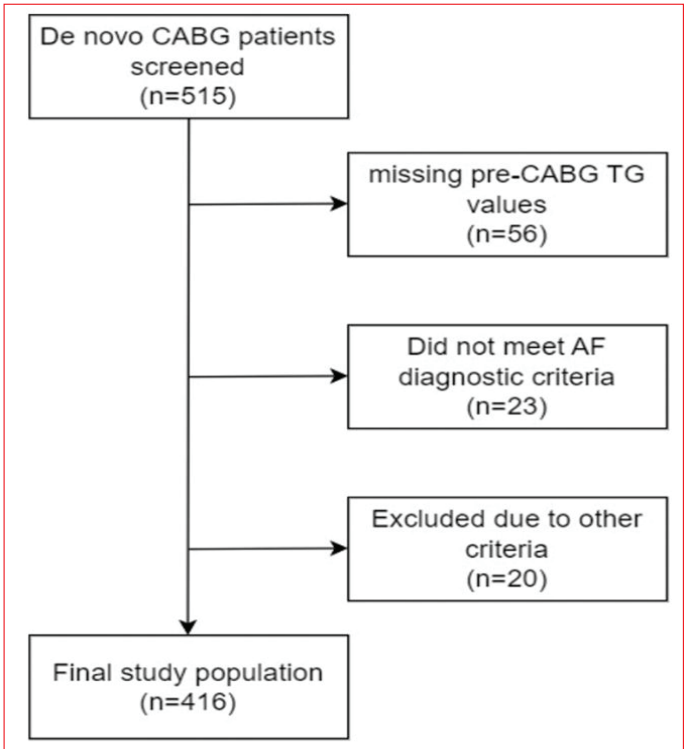

Figure 1. Flowchart.

Table 1. Clinical characteristics of the study population

Variables	Whole cohort (n=416)	No POAF (n=310)	POAF (n=106)	*P*-value
Age (years)	64 (40–86)	62 (41–86)	70 (40–82)	<0.001
Male, n(%)	325 (78.1)	245 (79.0)	80 (75.5)	0.191
Hypertension, n(%)	295 (70.9)	202 (65.2)	93 (87.7)	<0.001
Diabetes mellitus, n(%)	220 (52.9)	150 (48.4)	70 (66)	0.002
Hyperlipemia, n(%)	237 (57.0)	167 (53.9)	70 (66)	0.029
Body mass index (kg/m²)	28.0 (17.0–51.0)	27.8 (17.0–44.1)	29.4 (19.7–51.0)	0.010
Timing of CABG				
Within the first week following ACS, n(%)	78 (18.8%)	36 (11.6%)	42 (39.7%)	<0.001
1-4 Weeks Following ACS, n(%)	246 (59.1%)	211 (68.1%)	35 (33.0%)	0.001
After Elective Coronary Angiography, n(%)	92 (22.1%)	63 (20.3%)	29 (27.3%)	0.072
Laboratory factors				
Creatinine (mg/dL)	1.0 ± 0.4	1.0 ± 0.3	1.0 ± 0.5	0.953
eGFR (mL/min/1.73 m²)	79.9 ± 18.2	79.9 ± 17.7	79.6 ± 19.5	0.897
Glucose (mg/dL)	126.0 (76.0–380.0)	121.0 (76.0–380.0)	158.5 (80.0–371.0)	<0.001
HbA1c (%)	7.4 ± 2.0	6.9 ± 1.7	8.7 ± 2.5	<0.001
Low-density lipoprotein (mg/dL)	129.7±39.9	125.5 ± 37.9	141.9 ± 43.5	<0.001
Triglyceride (mg/dL)	154.0 (48.0–968.0)	139.0 (48.0–968.0)	198.0 (52.0–758.0)	<0.001
Triglyceride glucose index	9.1 (7.7–11.7)	9.0 (7.7–11.1)	9.8 (8.0–11.7)	<0.001
Echocardiographic variables				
Left atrial diameter (mm)	38.5 ± 4.1	37.4 ± 3.5	41.6 ± 4.2	<0.001
Left ventricular ejection fraction (%)	52.4 ± 9.2	53.3 ± 8.9	49.7 ± 9.7	0.001
Mild or moderate mitral regurgitation, n(%)	362 (87.0)	266 (85.8)	96 (90.6)	0.157
Number of distal anastomotic vessels				
1 vessel, n(%)	41 (9.9%)	34 (11.0%)	7 (6.6%)	0.001
2 vessels, n(%)	114 (27.4%)	99 (31.9%)	15 (14.2%)	<0.001
3 vessels, n(%)	261 (62.7%)	177 (57.1%)	84 (79.2%)	<0.001
Postoperative data				
Mechanical ventilation time (hours)	8.0 (2.0–46.0)	7.0 (3.5–46.0)	9.0 (2.0–39.0)	<0.001
Postoperative ICU stay (days)	3 (1–29)	3 (1–12)	4 (1–29)	<0.001
Postoperative hospital stay (days)	7 (4–104)	7 (4–22)	9 (5–104)	<0.001

Abbreviations: ACS; acute coronary syndrome, CABG; coronary artery bypass grafting, eGFR; estimated glomerular filtration rate, HbA1c; glycated hemoglobin A1c, ICU; intensive care unit, POAF; postoperative atrial fibrillation.

Table 2. Logistic regression analysis for identifying predictors of POAF

Characteristics	OR	95% CI	*P*-Value
Univariate logistic regression analysis			
Age	1.087	1.056–1.119	<0.001
Male	1.407	0.843–2.350	0.191
Body mass index	1.078	1.027–1.131	0.002
Hypertension	0.261	0.140–0.489	<0.001
Diabetes mellitus	0.482	0.304–0.763	0.002
Hyperlipidemia	0.601	0.379–0.951	0.030
Left atrial diameter	1.344	1.250–1.446	<0.001
Left ventricular end-diastolic diameter	1.059	1.005–1.115	0.031
Left ventricular ejection fraction	0.959	0.937–0.982	0.001
Mild or moderate mitral regurgitation	0.030	0.007–0.126	0.060
Mechanical ventilation time	1.130	1.056–1.209	<0.001
TyG index	5.402	3.557–8.202	<0.001
Multivariate logistic regression analysis			
Age	1.047	1.006–1.090	0.025
Left atrial diameter	1.373	1.243–1.518	<0.001
TyG index	6.824	3.511–13.264	<0.001

Abbreviations: POAF; postoperative atrial fibrillation, TyG; Triglyceride glucose index.

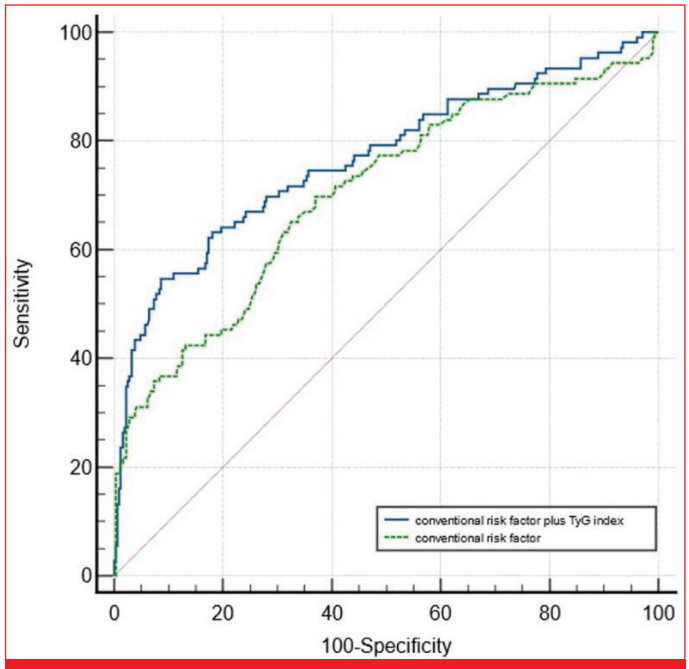

Figure 2. ROC curves of the conventional risk factor model and the conventional risk factor plus TyG index model. The conventional risk factor model includes age and left atrial diameter.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PP-005

Association of uric acid albumin ratio with recurrence of atrial fibrillation after cryoballoon catheter ablation

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Background and Aim: The catheter ablation (CA) of atrial fibrillation (AF) has gained popularity over the last two decades. Despite improvements in this technology and the better understanding of pathophysiological mechanisms, freedom from AF after a successful ablation procedure can still be as low as 57%. The recurrence rate after the CA of AF is estimated to be between 25-45%, recently, changes in the atrial architecture of atrial cardiomyopathy have been established to be related with inflammation. Many trials have clearly demonstrated the linkage between hyperuricemia and cardiac diseases or risk factors. In this study, our goal was to evaluate the role of UAR for the prediction of AF recurrence after CA.

Methods: The endpoint of the study was the late recurrence of AF after successful CA, which developed after a months blanking period. 24-h rhythm monitoring was routinely performed to all patients in control visits after the procedure. Symptomatic attacks requiring intervention or asymptomatic episodes of AF recorded on 24-h rhythm monitoring were defined as recurrences. Demographic characteristics, laboratory parameters, procedural features, complications, in hospital mortality and long term follow-up data were collected from medical records.

Results: The study population consisted of 170 subjects who had undergone CA for AF. The median follow-up duration was 22 months after procedure. The whole population was divided into two groups, namely recurrence and no recurrence. The frequency of the recurrence after ablation was 26% (n=53). Groups compared for demographic and clinical features and these data were depicted in Table 1. The mean UA levels were higher and the albumin levels were lower in the recurrence (+) group (p<0.01). According to the Pearson correlation analyses, uric acid and albumin levels were negatively and significantly correlated (p<0.01). The mean UAR was higher in the recurrence (+) group compared to the other group (2.4 ± 0.9 vs. 1.8 ± 0.7, p<0.01). When we entered these variables into the multivariable regression analysis, only LA diameter (HR: 1.08, 95% CI: 1.01-1.16, p=0.01) and the UAR (HR: 1.36, 95% CI: 1.06-1.75, p=0.01) were ascertained as independent predictors of recurrence (Figure 1). In ROC analysis, the UAR >1.67 predicted recurrence with a sensitivity of 77% and a specificity of 57% (AUC: 0.68, p<0.01). We also plotted the relationship between the probability of recurrence and UAR.

Conclusions: In conclusion, we found that a higher UAR was associated with the increased frequency of AF recurrence after CA. Although CA is a safe and effective treatment strategy for AF, identifying patients at high risk for developing recurrence is of utmost importance. In this manner, as an easily calculable laboratory index, UAR may be utilized to predict the recurrence of AF after CA.

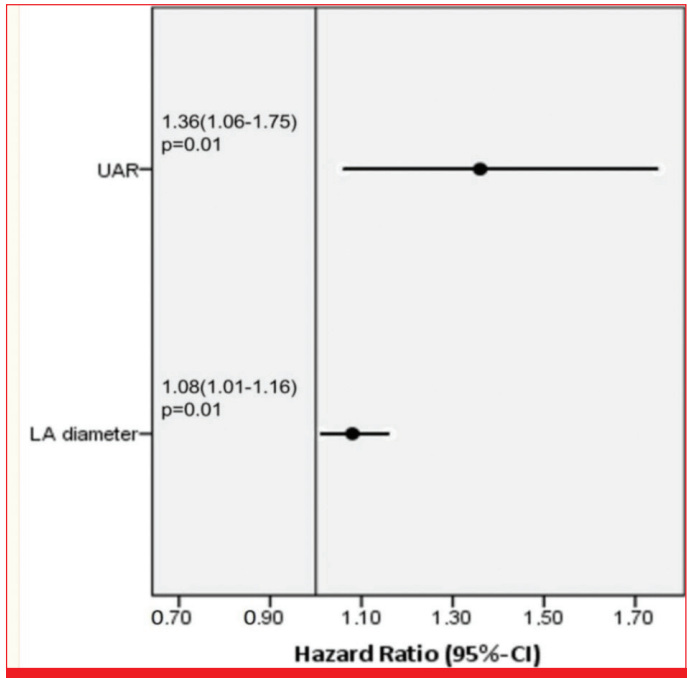

Figure 1. Forest plot graphic of independent predictors of atrial fibrillation recurrence after ablation according to the multivariable regression analyses.

Table 1. Comparison of the demographic and clinical parameters between study groups.

VARIABLE	All Patient	Recurrence (+)	Recurrence (-)	P VALUE
Age,yıl	59,1±11,7	61,3±11,5	57,6±11,4	0,07
Male, n(%)				
Hypertension, n(%)	93(54,7)	43(81,1)	50(42,7)	<0,01
Coronary Artery Disease, n(%)	41(24,1)	26(49,1)	15(12,8)	<0,01
Heart Failure, n(%)	29(17,1)	24(45,3)	5(4,3)	<0,01
COPH, n(%)	15(8,8)	6(11,3)	9(7)	0,10
Diabetes Mellitus, n(%)	24(14,1)	12(22,6)	12(10,3)	0,03
Chronic Kidney Disease, n(%)	10(5,9)	3(5,6)	7(6)	0,50
Smoking, n(%)	54(31,8)	16(30,2)	38(32,5)	0,76
Chadsvasc,	1,8±0,9	2,9±1	1,4±0,9	<0,01
AF Duration, month	17[30]	36[43]	12[22,5]	<0,01
AF Pattern, (PAF)	121(71,2)	22(41,5)	99(84,6)	<0,01
Hemoglobin, g/L	14,2±5,6	14,7±9,7	13,9±1,8	0,40
Serum Creatinin , mg/dl	0,9±0,2	0,9±0,2	0,8±0,2	0,17
LDL, mg/dl	116±34,5	116,2±31,1	115,8±36,1	0,94
TSH, mIU/l	2,1±1	2,3±1	1,8±0,9	0,25
CRP, mg/l	0,2[0,25]	0,2[0,3]	0,2[0,2]	0,81
Operation Duration, minute	123,9±22,5	121,8±26,7	124,8±20,3	0,41
Ü/A ratio	2,03±0,9	2,4±0,9	1,8±0,7	<0,01
LVEF	57,6±13,7	48,4±11	61,7±8,9	<0,01
LA AP Diameter	39,7±6,3	44,1±5,5	37,7±5,6	<0,01
Left Ventriculer Hypertrophy, n(%)	18(10,6)	7(13,2)	11(9,4)	0,45

VARIABLE	Unadjusted HR	CI	P VALUE	Adjusted HR	CI	P VALUE
LA Diameter	1,15	1,08-1,21	<0,01	1,08	1,01-1,16	0,01
U/A ratio	1,59	1,25-2,02	<0,01	1,36	1,06-1,75	0,01
Chadsvasc	1,34	1,13-1,59	<0,01	1,02	0,83-1,25	0,81
AF Pattern	0,29	0,16-0,51	<0,01	0,55	0,27-1,1	0,09
AF Duration	1,01	1,01-1,02	<0,01	1,02	0,99-1,02	0,19

COPD: Chronic obstructive pulmonary disease, AF: Atrial fibrillation, LDL: Low density lipoprotein, TSH: Thyroid stimulating hormone, UAR: Uric acid albumin ratio, LVEF: Left ventricle ejection fraction, LA: Left atrial.

Other

PP-006

The effect of obesity on PR interval and QRS complex duration in electrocardiogram

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Background and Aim: Obesity has both direct and indirect (comorbidities) effects on myocardial morphology. It has been observed that various cells in the heart are transformed into fat cells in obese patients. Therefore, atrial and ventricular conduction disturbances are expected to be more common in obese patients. On the other hand, as is well known, increased QRS is associated with poor cardiovascular outcomes. The aim of this study was primarily to investigate and evaluate the effect of body mass index (BMI) on PR interval and QRS duration in electrocardiogram (ECG).

Methods: A total of 158 adults (62 women, 96 men) with a mean age of 40.59 ± 10.24 years and a BMI of 30.03 ± 8.76 kg/m^2 were retrospectively analyzed. According to BMI values, 56 of the patients were classified as underweight and healthy, 43 as overweight and 59 as obese patients. Adults without arrhythmia, significant heart disease and not taking medication were included in the studied group. The characteristics of the patients are also shown in Tables 1 and 2. The correlation between BMI and PR interval and QRS duration was analyzed by multiple linear regression analysis.

Results: There was no statistically significant correlation between PR interval and BMI (p>0.05). Similarly, no statistically significant correlation was found between gender, age, blood pressure and PR interval (p>0.05). On the contrary, as expected, a significant negative correlation (p<0.05) was shown between PR interval and pulse rate. On the other hand, no correlation was found between BMI and pulse rate in the group analyzed by regression analysis (p=0.0927). A significant positive correlation (p<0.05) was observed between QRS duration and BMI. In other words, the duration of the QRS complex increased with increasing BMI. The mean QRS was 89.85 ± 11.18 ms. Similarly, a significant correlation (p<0.05) was calculated between QRS and gender. It was observed that the QRS duration was less in women with 86.42 ± 13.36 ms (in men 92.06 ± 8.82 ms). On the other hand, no significant correlation was found between QRS duration and age, pulse rate and blood pressure. These results are summarized in Tables 3 and 4.

Conclusions: In this study, contrary to expectations, there was no statistically significant effect of BMI on PR interval. On the other hand, it was once again confirmed in this study that the PR interval shortens with increasing pulse rate. Since there was no correlation between BMI and pulse rate in the group analyzed, it was difficult to establish an indirect relationship between PR interval and BMI via pulse rate. In contrast, there was a significant positive correlation between QRS interval and BMI and gender. In other words, as BMI increases, QRS duration is prolonged. Increased QRS duration with obesity is a warning for poor cardiovascular outcomes. It is perhaps worth noting that the QRS is narrower in women. Investigation of the cardiological reasons for the increase in QRS duration in obesity would be appropriate for further research.

Table 1. Electrographic, echocardiographic and general characteristics of all patients

Parameter	Value ± Standard deviation
Number of patients	158
Age	40.59 ± 10.24
BMI (kg/m^2)	30.03 ± 8.76
Gender (male/female)	96/62
Systolic blood pressure, mmHg Diastolic blood pressure, mmHg	117.41 ± 15.27 77.41 ± 10.68
Heart rate, (L/min)	75.27 ± 13.89
LA, cm	3.702 ± 0.30 x 4.26 ± 0.5
LVEDD, cm	4.81 ± 0.45
LVESD, cm	3.72 ± 0.42
IVST, cm	0.89 ± 0.13
RV, cm	3.62 ± 0.44
MV mean pressure gradient, mmHg	1.38 ± 0.49
AV mean pressure gradient, mmHg	2.88 ± 1.08
MR (trivial), n (%)	71 (44.9)
AR (trivial), n (%)	8 (5.1)
LVEF, %	60.39 ± 1.41
Systolic PAB, mmHg	21.08 ± 2.65
Diastolic dysfunction (stage I), n (%)	21 (13.3)
Insulin resistance, n (%)	13 (8.2)
Smoking history, n (%)	85 (53.8)
PR interval, ms	153.55 ± 19.60
QRS complex duration, ms	89.85 ± 11.18

BMI: Body mass index, LVEDD: Left ventricular end-diastolic dimension, LVESD: Left ventricular end-systolic dimension, IVST: Interventricular septum thickness, LVEF: Left ventricular ejection fraction, LA: Left atrial, RV: Right ventricul, MR: Mitral regurgitation, AR: Aort regurgitation, MV: Mitral vave, AV: Aort vave.

Table 2. Distribution of patients according to their BMI

BMI range, kg/m^2	Weight range	Number of patients
<18.5	Underweight	2
18.5-24.9	Healthy weight	54
25.0-29.9	Overweight	43
30.0-34.9	Class 1 Obesity	26
35.0-39.9	Class 2 Obesity	15
>40	Class 3 Obesity	18
Total		158

BMI: Body mass index.

Table 3. Multiple linear regression analysis for PR interval and QRS duration

Variable	BMI	Systolic blood presure	Diastolic blood presure	Age	Gender	Heart rate
PR interval, p value	0.0590	0.2053	0.2592	0.4680	0.3186	0.0100
QRS duration, p value	0.0441	0.4696	0.2986	0.3195	0.0015	0.2476

BMI: Body mass index.

Table 4. The effect of BMI on heart rate

Variable	BMI
Heart rate, p value	0.0927

BMI: Body mass index.

Other

PP-007

Depression status and quality of life in patients aged 60 and over with heart failure: A pilot study

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Background and Aim: In those aged 60+, heart failure often leads to decreased quality of life, especially due to symptoms such as fatigue and social isolation. Furthermore, an increased prevalence of depression in these patients compounds their suffering. Depression not only intensifies heart failure symptoms but also further affects life quality. Hence, a comprehensive approach combining pharmacological and psychotherapeutic treatments is crucial. To investigate the relationship between depression and quality of life in individuals aged 60+ with heart failure and understand the role of gender and other demographic factors.

Methods: This cross-sectional study was approved by the Ethics Committee of Koşuyolu High Specialization Training and Research Hospital. Data were gathered from heart failure patients aged 60+ at the same hospital between 25.07.2023 and 25.08.2023. The pilot study included 41 participants, with the main study targeting 800. Tools utilized were the geriatric depression scale, the SF-12 quality of life scale, and the Minnesota living with heart failure Qquestionnaire. Data analysis was performed using SPSS 22.0, employing non-parametric tests like the Kruskal-Wallis H test.

Results: Of the 41 patients, 43.9% showed no depression, 39% had mild symptoms, 4.9% moderate, and 12.2% severe depression. Women showed significantly higher depression rates (p=0.039). No major differences were observed based on age, education, and other demographic factors (p>0.05). However, depression severity was significantly tied to quality of life, general health, mental health, and vitality (p<0.05). There were significant differences in the quality of life (p=0.014), general health (p=0.001), mental health (p=0.002), and vitality (p=0.011) of those living with heart failure based on their depression status. It was determined that those with severe depression have lower quality of life, general health, mental health, and vitality compared to those without depression.

Conclusions: In this pilot study involving 41 patients, the prevalent mood was either a lack of depression or only mild depressive symptoms. However, there was a marked gender disparity in depression rates, with women manifesting higher incidences and tending towards more severe categories. While demographic factors such as age, education, and employment characteristics didn't significantly impact depression status, the mental well-being of heart failure patients was intrinsically linked with their depression severity. Those experiencing severe depression notably had compromised quality of life, general health, mental health, and vitality. The findings underscore the importance of psychological assessments in conjunction with medical evaluations in older patients with heart failure.

Interventional Cardiology / Valve and Structural Heart Disease

PP-008

Short-term outcomes after transcatheter aortic valve implantation; risk factors for acute kidney injury and impact of right heart failure

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Background and Aim: Aortic stenosis is one of the most common valvular heart diseases. Surgical aortic valve replacement or transcatheter aortic valve implantation (TAVI) is recommended to improve survival in symptomatic patients with severe aortic stenosis TAVI is an effective treatment option for patients who are considered inoperable or at high risk for surgical aortic valve replacement. Conclusive evidence of the safety and efficacy of TAVI has been demonstrated in relation to the prognostic benefits and outcomes of treated patients. However, most studies in TAVI show

that despite increased clinician experience and advances in technology, procedural complications and co-morbidities remain a significant problem affecting patient outcomes. Acute kidney injury (AKI), which affects clinical response and occurs specifically after TAVI is a common complication and has been associated with poor prognosis. Multifactorial causes play a role in the pathogenesis of AKI. Right heart failure, one of these causes, leads to renal venous congestion and decreased renal perfusion pressure, leading to AKI and often cardiorenal syndrome. In our study, we planned to evaluate the risk factors and especially the presence of right heart failure before TAVI and its effect on the development of AKI in patients undergoing TAVI.

Methods: It is a retrospective single center observational study. Patients who were diagnosed with severe aortic stenosis according to clinical, echocardiographic and hemodynamic criteria in the cardiology clinic of Dr. Siyami Ersek Cardiovascular Surgery Hospital between January 2015 and December 2020 who were at high risk for surgical repair were included in the study. Demographic and clinical characteristics, laboratory and echocardiographic data aswellas complications during the procedure were recorded. AKI was defined according to the increase in serum creatinine with in the first 48 hours using AKI Network criteria. Demographic characteristics, renal function and mortality rates of patients classified. Tricuspid annular plane systolic excursion (TAPSE) and inferior vena cava (IVC) measurements were recorded for right ventricular dysfunction. TAPSE <17 mm was considered as the definition of right ventricular dysfunction.

Results: In our study the rate of patients who developed AKI as a complication in patients undergoing TAVI was 42%, the mortality rate was 11.1%, the mean age of the patients was 78.49 ± 8.01 years. Female patiens were more dominant 56.1%. When the characteristics of patients who developed AKI were analyzed, AKI was significantly higher in patients with a higher mean age (80.90 ± 6.8). The mean hemoglobin level before TAVI was significantly lower in patients who developed AKI. In our study there was no significant difference between right ventricular dysfunction in patients with and without AKI.

Conclusions: Careful evaluation of risk factors in high risk patients together with the TAVI planning team (Heart-Kidney Team) including a nephrologist as a multidisiplinary team will help improve outcomes.

Table 1. Demographic characteristics of patients

Age	78.49 ± 8.01
Gender	Female: 111 (56.1%), male: 87 (43.9%)
DM	74 (37.4%)
HT	165 (83.3%)
CKD Stage 1-2: GFR: ≥60 mL/min/1.73 m^2 Stage 3: GFR: 30-59 mL/min/1.73 m^2 Stage 4: GFR <29 mL/min/1.73 m^2	 97 (49%) 87 (43.9%) 14 (7.1%)
PHT (>35 mmHg)	136 (68.7%)
TAPSE (<17 mm)	49 (24.7%)
IVC diameter (>21 mm)	107 (54%)
Rate of AKI	83 (41.9%)

Table 2. Demographic characteristics of patients who develop and do not develop AKI

Patient characteristics	Patients who develop AKI (n=83)	No AKI (n=115)	p
Age	80.90 ± 6.8	76.75 ± 8.4	<0.000***
Gender	Female: 57 (68.7%) Male: 26 (31.3%)	Female: 54 (47%) Male: 61 (53%)	<0.002***
HT	74 (89.2%)	91 (79.1%)	<0.062
CKD Stage 1-2: Stage 3: Stage 4:	83 (41.9%) 19 (22.8%) 50 (60.2%) 14 (17%)	115 (58.1%) 77 (67%) 38 (33%) 0	<0.000***
Hb before the procedure	83 (41%) Hb: 10.8 ± 1.55	115 (58%) Hb: 11.65 ± 1.60	<0.000
Death	16 (19.2%)	6 (5%)	<0.002***

Table 3. Evaluation of echocardiographic right heart measurements (IVC, TAPSE and PH) in patients with and without AKI

ECHO measurements	AKI (n=83, 41%)	No AKI (n=115)	p
IVCI >21 mm	42 (39.3%)	65 (60.7%)	<0.607
TAPSE <17 mm	19 (38.8%)	30 (61.2%)	<0.139
PH >35 mmHg	61 (44.9%)	75 (55.1%)	<0248

IVC: Inferior vena cava collapsing index, TAPSE: Tricuspid annular plane systolic excursion, PH: Pulmonary hypertension.

Interventional Cardiology / Carotid and Peripheral Vascular

PP-010

Lower extremity peripheral arterial interventions and contrast induced nephropathy

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Background and Aim: Atherosclerosis is the most significant risk factor for the impairment of arterial blood circulation. Peripheral arterial disease (PAD) is generally more prevalent in diabetic and smoking men. PAD lesions have a long procedural duration and often require a considerable amount of contrast medium usage. In this study, the development of contrast-induced nephropathy (CIN) was evaluated in patients with PAD after interventional procedures.

Methods: The study included 144 patients who presented to our hospital between January 2019 and July 2023 and underwent interventional methods for PAD treatment. Of these patients, 65 were male, and the mean age was calculated as 63.5 ± 11.9 years. It was a prerequisite to perform the procedure on a single leg during each session. CIN was defined as an increase in serum creatinine of >0.5 mg/dL or >25% compared to baseline levels within 48-72 hours after the interventional procedure. Patients were divided into two groups: those who developed CIN and those with normal kidney function (NKF). Patients with a baseline creatinine level >2.5 mg/dL and/or a GFR <30 were not included in the study. Patients with a GFR <50 received an intravenous infusion of N-acetylcysteine-prepared saline solution at least 1 hour before the procedure and continued for 24 hours after the procedure.

Results: CIN was observed in a total of 32 patients. There were no significant differences between the two groups in terms of baseline creatinine, Mehran score, and GFR levels (Table 1). The amount of contrast medium used in each patient was recorded. Significant differences were found between the CIN and NKF groups in terms of creatinine, GFR, CV/GFR, and CV/CrCl ratios. The CV/GFR ratio was evaluated using ROC analysis, and a CV/GFR ratio of >3.5 was determined to predict CIN development with 76% sensitivity and 79% specificity.

Conclusions: It was determined that there is a CIN development rate of >20% after interventional procedures due to PAD. Therefore, it is believed that determining the amount of contrast medium used during the procedure based on GFR would be beneficial (Table 2). To achieve this, it is essential to emphasize the importance of not exceeding the maximum CV ratios calculated based on GFR before starting the procedure. According to our study, a CV/GFR ratio of >3.5 predicts the development of CIN.

Table 2. Multivariate analysis of the patients. Predictors of the CIN development

Variable	OR (95% CI)	p
Baseline creatinine	3.3 (1.0-6.9)	0.005
Baseline GFR	3.0 (0.8-5.7)	0.008
Baseline Mehran score	1.9 (0.7- 3.9)	0.012
Contrast volume	3.9 (1.3-7.7)	<0.001
CV/GFR	4.1 (1.5-8.0)	<0.001
Diabetes mellitus	1.7 (0.6-4.1)	0.042
Smoking	1.6 (0.4-3.8)	0.048

CV: Contrast volume, GFR: Glomerular filtration rate.

Table 1. General characteristics of the patients

Variables	CIN group (n=32)	NKF group (n=112)	p
Age, years	64.3 ± 9.2	62.9 ± 9.9	0.424
Baseline creatinine, mg/dL	1.18 ± 0.49	1.12 ± 0.54	0.715
Baseline GFR, mL/min/1.73 m^2	62.3 ± 17.4	70.1 ± 24.4	0.085
Coronary artery disease	16 (50%)	48 (42.8%)	0.076
Diabetes mellitus	19 (59.3%)	42 (37.5%)	0.002
Contrast volume	252 ± 101	164 ± 72	<0.001
CV/GFR	4.5 ± 1.7	3.4 ± 1.3	<0.001
Baselime Mehran score	13.8 ± 4.8	10.1 ± 4.2	<0.001
Renal protection rate	8 (25%)	26 (23.2%)	0.814

CV: Contrast volume, GFR: Glomerular filtratio rate, CIN: Contrast induced nephropathy, NKF: Normal kidney functions.

Interventional Cardiology / Coronary

PP-011

Beginners in the cath lab: Catheter kinking and management in the patient presenting with acute coronary syndrome

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Background and Aim: Fracture or kinking of the diagnostic catheter during coronary angiography procedures is a rare but serious complication. If the catheter cannot be removed percutaneously, it may result in surgical interventions. We present a case of catheter fracture during right coronary artery cannulation in a patient with acute inferior myocardial infarction. The catheter fractured after right femoral access was removed by percutaneous left (contralateral) femoral access.

Methods: Our patient with diabetes mellitus and no previous known coronary artery disease presented to the emergency department with typical chest pain and was admitted to our cardiology clinic with a diagnosis of inferior myocardial infarction. Emergency percutaneous coronary angiography was performed with a 7 Fr sheath from the right femoral artery. Imaging of the left coronary arteries was performed. During right coronary artery imaging with a diagnostic catheter, catheter manipulation was not reflected on the catheter tip. In addition, there was no opaque material passage through the distal end of the catheter. The bending of the catheter was observed (Figure 1). Despite contralateral manipulation of the catheter, the bent segment did not straighten. 0.038-inch and 0.014-inch guidewires were sent. As the wires did not pass through the fractured segment, we planned to remove the fractured catheter percutaneously from the left femoral artery. We used a 9 Fr sheath to access the left femoral artery. We captured the catheter tip in repeated attempts with a snare catheter (Figure 2). We cut the proximal part of the catheter in the right femoral artery and removed the entire catheter from the left femoral sheath (Figure 3). We then performed coronary angiography and found 100% occlusion in the mid-RCA. The procedure was terminated with stenting of the right coronary artery.

Results: The presence of a foreign body in the vessel is a very serious condition due to thromboembolism and infection. Catheter fracture is a complication that may occur during coronary angiography. Catheter fracture usually occurs during RCA cannulation, as in our case. Loss of pressure waves, failure to transmit catheter movements to the tip, and obstruction of opaque material passage should suggest catheter fracture. The location of the fracture segment can be determined by fluoroscopy. The kink can usually be opened by reversal or by sending a 0.038-inch guide wire. The fractured catheter should not be aggressively pulled into the sheath. If this is done, vascular injury and catheter rupture may occur. To prevent such an event, hemodynamic monitoring, careful manipulation of 0.038-inch wires, and preference for the radial method are among the precautions.

Conclusions: As in every case during cardiac catheterization, preventing complications is easier than managing them. Therefore, it is vital to be careful, especially in tortuous vessels, to recognize its complication early to avoid catheter fracture.

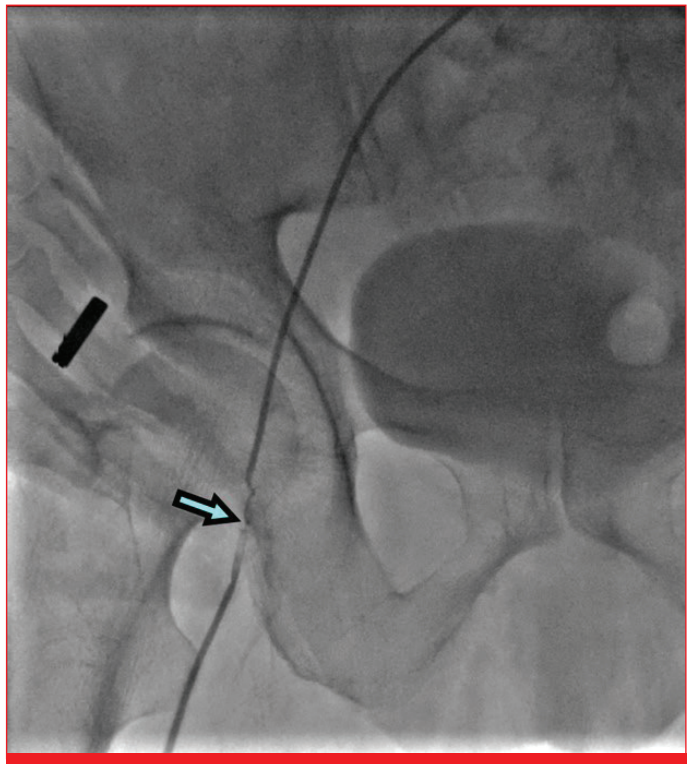

Figure 1. Kinked diagnostic catheter (blue arrow: kinked catheter segment).

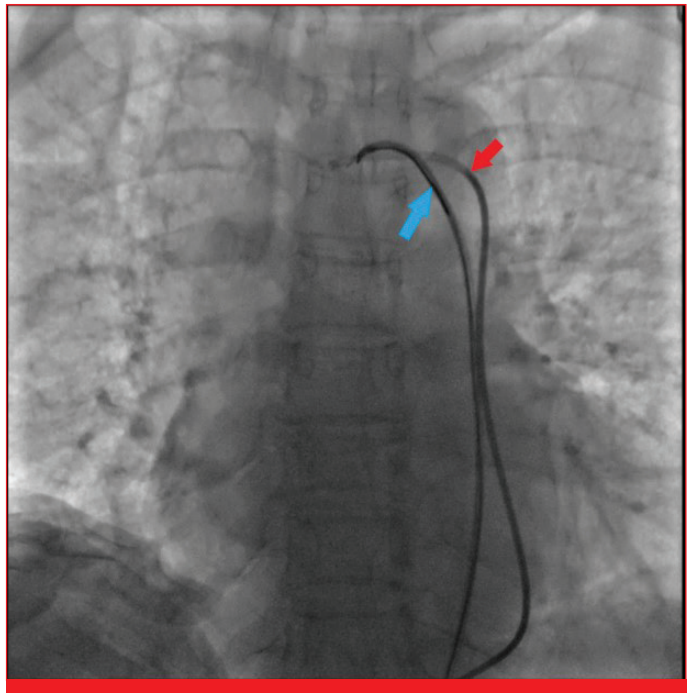

Figure 2. A snare capture of kinked guiding catheter.

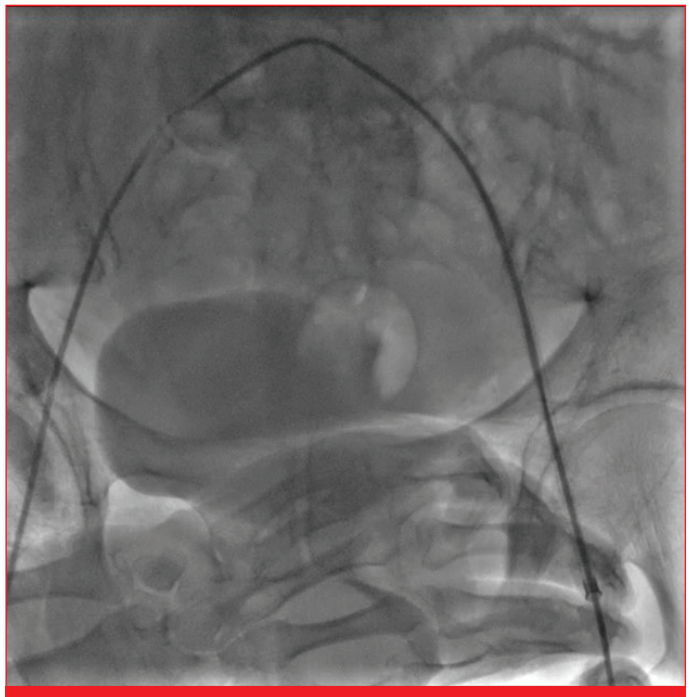

Figure 3. Withdrawal of the kinked catheter from the left femoral artery.

Interventional Cardiology / Coronary

PP-012

Progression of the atherosclerosis in native coronary arteries in patients with previous coronary revascularization

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Background and Aim: Repeat revascularization and target lesion failure are the most common observed adverse outcomes in patients who underwent revascularization either via coronary artery bypass surgery (CABG) or percutaneous coronary intervention. Besides technical failure, progression of atherosclerosis is a major issue in patients who were treated with CABG initially. Hence prediction of patients who are prone to develop new atherosclerotic disease is essential and may have impact on initial revascularization strategy. In this study, we aimed to investigate the predictors of the progression of atherosclerosis in patients who underwent coronary revascularization. Moreover, we also search for the impact of the atherosclerosis progression on cardiovascular outcomes.

Methods: We retrospectively collected the data of patients with recent coronary angiography who were previously underwent coronary revascularization via CABG or PCI. Patients were divided into two groups; group 1 included patients with previous PCI and group 2 patients with previous CABG. Patients' demographics, comorbidities and biochemical analyses were gathered.

Results: There were 142 patients in group 1 and 104 patients in group 2. Mean duration between revascularization and coronary angiography were longer in group 2 (p=0.006). There was no significant difference between groups with respect to age and gender (p=0.410 and p=0.677, respectively). Comorbidities including hypertension (p=0.148), diabetes (p=0.741), dyslipidemia (p=0.119) and chronic renal failure (p=0.682) were also similar between groups. Progression of atherosclerosis was statistically more prevalent among group 2 patients (42.6% vs. 63%, p=0.005). However, despite the higher rates of progression of the atherosclerosis in native coronary arteries, cardiovascular outcomes including heart failure (p=0.374), myocardial infarction (p=0.874) and cardiovascular death (p=0.214) were comparable between groups. Univariate analyses demonstrated that none of the comorbidities or angiographic features are independently associated with progression of atherosclerosis.

Conclusions: Our study have demonstrated that progression of atherosclerosis is a major issue in patients with previous revascularization and more common among patients who were initially treated with CABG. However, we could not able to define any predictors of atherosclerosis progression and hence further studies are required.

Interventional Cardiology / Coronary

PP-013

The effect of sandbag-free bed rest after manual compression in patients who underwent transfemoral angiography

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Background and Aim: Femoral access site complications (ASC) are an important condition that is frequently seen after any percutaneous intervention procedure, and affects patient-doctor comfort. In most clinics, after the removal of the femoral sheath, pressure is applied with a sandbag for 3-5 hours on the insertion site, followed by manual compression. There is not enough data that this sandbag application, which has a very negative effect on patient comfort, reduces ASC. In this study, we aimed to compare the ASC rates between standard sandbag application and bed rest without sandbag following manual compression in patients with femoral access.

Methods: Patients who were underwent transfemoral any percutaneous intervention procedure (mostly coronary interventions) in our clinic between April 2019 and May 2023 were included. Patients were classified as being followed without sandbag (n=160) and followed with sandbag (n=158), and ASC rates (ecchymosis, pseudoaneurysm, hematoma, bleeding) between the two groups were compared.

Results: A total of 318 patients were included in our study, 160 of which were without sandbags and 158 with sandbags. There was no difference between the two groups in the gender, age, sheath size and bed rest times. The rate of diabetes mellitus (DM) in patients who did not use sandbags was found to be statistically significantly higher than in patients who used sandbags (p=0.044). Complications were detected in 16.9% (n=27) of patients who did not use sandbags, and 25.3% (n=40) of patients with sandbags. The most common complications were recorded as ecchymosis with 10.6% (n=17) in the without sandbag group and 13.9% (n=22) in the sandbag group. There was no statistically significant difference in the distribution of complication and complication development time between the groups (p>0.05).

Conclusions: Femoral ASC tends to be less common in patients who are followed up with only bed rest after removal of the femoral sheath, followed by manual compression, without sandbag. In addition, not putting sandbags provides a significant increase in patient comfort.

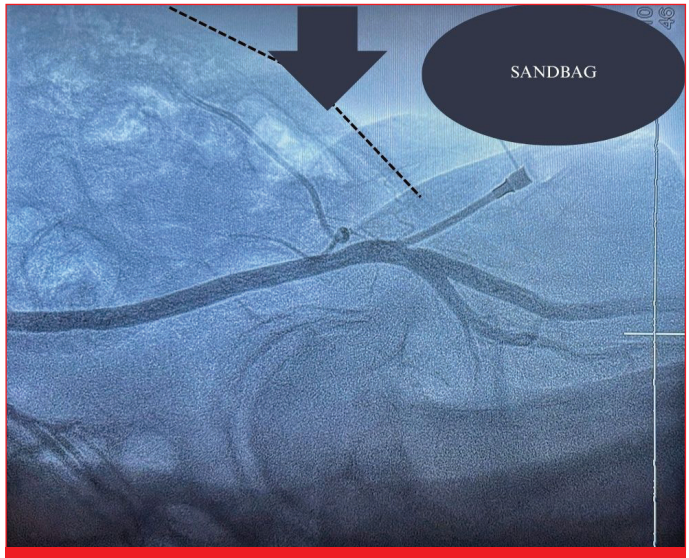

Figure 1. Fluoroscopic view of femoral artery entry and correct compression site. ---: Line showing abdominal contours in overweight patients, ->: Line showing where the sandbag should compress.

Table 1. Comparison of Demographic Characteristics, Chaornic Disseases and Smoking

	Group 1 (n=160)	Group 2 (n=158)	p
Sex; n (%)			
Female	43 (26.9)	49 (31.0)	0.416*
Male	117 (73.1)	109 (69.0)	
Age (years)	60.86 ± 12.58	62.50 ± 11.62	0.327**
BMI (kg/m²)	28.21 ± 4.27	28.20 ± 4.35	0.897**
Central Obesity; n (%)	37 (23.1)	43 (27.2)	0.401*
HT; n (%)	73 (45.6)	85 (53.8)	0.145*
DM; n (%)	67 (41.9)	49 (31.0)	**0.044***
Atrial Fibrilation; n (%)	13 (8.1)	14 (8.9)	0.814
CRF; n (%)	15 (9.4)	11 (7.0)	0.432*
Smoking; n (%)	43 (26.9)	43 (27.2)	0.946*

*: Pearson Chi-square test
**: Mann Whitney U test
BMI: Body Mass Index, HT: Hypertension, DM: Diabetes Mellitus CRF: Chronic Renal Failure

Table 2. Comparison of Procedure-Related Features

		Group 1 (n=160)	Group 2 (n=158)	p
Apply to the Hospital				
ACS		85 (53.1)	90 (57.0)	0.492*
Elective Procedure		75 (46.9)	68 (43.0)	
Antiaggregant-Anticoagulant Treatment Before Procedure #				
ASA		118 (73.8)	106 (67.1)	
Clopidogrel		49 (30.6)	46 (29.1)	
Ticagrelor		6 (3.8)	4 (2.5)	-
Prasugrel		3 (1.9)	1 (0.6)	
Varfarin		6 (3.8)	8 (5.1)	
NOAC		10 (6.3)	8 (5.1)	
PCI (n, %)		116 (72.5)	107 (67.7)	0.352*
Sheath				
6F		83 (51.9)	89 (56.3)	0.425*
7F		77 (48.1)	69 (43.7)	
Medication Given in the Procedure				
None		42 (26.3)	50 (31.6)	
Heparin		81 (50.6)	68 (43.0)	0.380*
Heparin+Tirofiban		37 (23.1)	40 (25.3)	
Sheath Removal Time (mn)		203.75 ± 173.79	169.62 ± 123.36	0.058**
Compression Time (dk)		24.25 ± 13.39	22.29 ± 13.53	0.069**
Activated Clotting Time		135.01 ± 16.70	120.89 ± 24.91	**<0.001****
6F Sheath Sedantery Follow-up Time (mn)		235.96 ± 64.22	225.56 ± 38.33	0.176**
7F Sheath Sedantery Follow-up Time (mn)		348.64 ± 54.03	346.09 ± 38.53	0.760**
Vital signs before sheath removal	**Systolic BP**	126.01 ± 14.87	124.77 ± 14.66	0.429**
	Diastolic BP	75.21 ± 10.02	75.15 ± 10.63	0.893**
	Heart Rate	75.22 ± 13.00	77.06 ± 13.12	0.265**

#: Some patients have more than one treatment.
*: Pearson Chi-square test
**: Mann Whitney U test
ACS: Acute Coronary Syndrome, ASA: Acetyl Salicylic Acid, PCI: Percutaneous Coronary Intervention, BP: Blood Pressure

Table 3. Distribution of Hematological Parameters Before and After the Procedure

		Group 1 (n=160)	Group 2 (n=158)	p
BEFORE PROCEDURE	**APTT**	30.88 ± 3.25	28.96 ± 3.69	**<0.001***
	INR	1.06 ± 0.19	1.06 ± 0.13	0.897*
	PLT (10³/µL)	255.54 ± 79.78	272.67 ± 88.59	0.100*
	Hemoglobin (g/dl)	13.65 ± 2.01	13.80 ± 1.84	0.472**
	Hematocrit (%)	41.10 ± 5.92	41.73 ± 5.27	0.318**
AFTER PROCEDURE	**Hemoglobin (g/dl)**	12.65 ± 1.86	12.87 ± 1.69	0.397*
	Hematocrit (%)	38.16 ± 5.48	39.01 ± 5.01	0.151**

*:Mann Whitney U test
**: Student's T Test
APTT: Activated partial thromboplastin time, INR: International Normalized Ratio, PLT: Platelet

Table 4. Comparison of Complications Between Groups

	Group 1 (n=160)	Group 2 (n=158)	p
Complication			
No	133 (83.1)	118 (74.7)	0.065*
Yes	27 (16.9)	40 (25.3)	
Complication type			
Ecchymosis	17 (10.6)	22 (13.9)	0.340*
Hematoma	5 (3.1)	7 (4.4)	
Re-bleeding	5 (3.1)	10 (6.3)	
Pseudoaneurysm	0 (0.0)	1 (0.6)	
Complication Development Time (mn)	83.87 ± 192.08	121.39 ± 348.33	0.711**

*: Pearson Chi-square test
**: Mann Whitney U test

Interventional Cardiology / Coronary

PP-014

The impact of ticagrelor therapy on CABG-related bleeding in patients with STEMI managed with pPCI

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Background and Aim: Coronary artery bypass grafting (CABG) in patients with recent ST-segment elevation myocardial infarction (STEMI) carries higher risk particularly due to increased bleeding rates. In this study, we aimed to investigate the impact of ticagrelor therapy on cardiovascular outcomes in patients with recent STEMI treated with primary percutaneous coronary intervention (pPCI) and subsequent CABG surgery.

Methods: Patients with a clear diagnosis of STEMI who were treated pPCI and underwent subsequent CABG surgery were included. Patients who were preloaded with ticagrelor 180 mg during the diagnosis were studied. Patients' demographics, clinical variables and short-term cardiovascular outcomes were recorded.

Results: After the exclusion of patients, 98 patients were included in the analyses. 44 patients (44.8%) of patients underwent CABG surgery within 3 days of pPCI procedure and 42 (43%) patients underwent between days 3 and 7. Left main coronary artery involvement was 26.5% and the presence of chronic total occlusion in the non-infarct related artery was 25.5% among the study population. CABG-related bleeding occurred in 22 (22.4%) patients. There was no significant difference with respect to ticagrelor dose and timing of the surgery between patients with or without CABG-related bleeding (p=0.165 and p=0.142). Multivariate analyses demonstrated that only preoperative haemoglobin level is associated with CABG-related bleeding (OR: 0.720, p=0.034). There were 3 deaths within the 30 days of surgery which of all occurred in patients with CABG-related bleeding. However, CABG-related bleeding did not increase long-term cardiovascular events during the follow-up.

Conclusions: Our results indicated that cessation of ticagrelor therapy within 3 days prior to surgery does not associate with an increased risk of CABG-related bleeding. Moreover, early CABG following STEMI does not increase the risk of long-term cardiovascular events.

Table 1. Comparison of patients with/without bleeding

	Major bleeding (-) (74 patients)	Major Bleeding (+) (22 patients)	p value
Age	60.9±10.6	61.1±10.0	0.937
Gender	77%	79%	0.892
HT	64.5%	50%	0.220
DM	43.5%	45.5%	0.866
CVA	4.1%	3.9%	0.243
CRF	6.7%	22.7%	**0.028**
Preoperative HGB	13.0±2.0	11.3±1.7	**0.001**
LVEF	46.0±9.3	47.0±6.6	0.613
Troponin	2.53(0.4-10)	3.9(0.3-10)	0.314
Pro-BNP	500(200-18960)	1081(450-35.000)	**0.026**
Additional Ticagrelor	90 (0-450)	180 (0-630)	0.165
ES replacement	1.5(0-4)	7 (2-26)	**<0.001**
Early CABG	32 (43%)	10 (45%)	0.142
ICU stay	2 (1-10)	6(1-20)	**<0.001**
Hospital stay	7(3-27)	11.5(3-90)	**0.011**

Table 2. Predictors of major adverse cardiovascular outcomes

	Univariate analyses			*Multivariate analyses*		
	OR	95% CI	p value	OR	95% CI	p value
Age	1.026	0.986 – 1.068	0.211			
HT	0.727	0.321 - 1650	0.446			
DM	1.440	0.645 – 3.219	0.374			
CRF	1.370	0.361 – 5.191	0.644			
Non-IRA CTO	**2.950**	**1.100 – 7.912**	**0.032**	**1.714**	**0.281 – 8.452**	**0.559**
LMCA disease	1.044	0.425 – 2.565	0.925			
Peak Hs-TnT	**1.306**	**1.132 – 1.506**	**<0.001**	**1.312**	**1.018 – 1.691**	**0.036**
Mitral regurgitation	**2.976**	**1.285 – 6.896**	**0.011**	**2.517**	**1.170 – 5.687**	**0.006**
LVEF	**0.710**	**0.626 – 0.806**	**<0.001**	**0.692**	**0.592 – 0.811**	**<0.001**
Major bleeding	1.080	0.417 – 2.799	0.874			
Early surgery	0.806	0.602 – 1.078	0.146			

Table 3. Univariate and multivariate analyses for predicting CABG-related bleeding

	Univariate analyses			*Multivariate analyses*		
	OR	95% CI	p value	OR	95% CI	p value
Age	0.998	0.952 – 1.046	0.936			
HT	0.551	0.211 -1.437	0.223			
DM	1.086	0.418 -2,819	0.866			
CRF	**4.176**	**1.085 -9.076**	**0.038**	0.698	0.445 – 1.094	0.643
Additional ticagrelor	0.574	0.221 -1.493	0.255			
Hgb	**0.661**	**0.506 -0.862**	**0.002**	**0.720**	**0.531 – 0.976**	**0.034**
Early surgery	0.739	0.517 – 1.058	0.098	0.698	1,094 – 8.651	0.117

Table 4. Demographics and clinical variables of study population

	Mean
Age	61
Gender (male)	78%
Hypertension	61.2%
Diabetes mellitus	43.9%
Chronic renal failure	10.2%
LMCA involvement	26.5%
Non-IRA CTO	25.5%
Ischemia duration, minutes	120 (60-1080)
Hemoglobin, g/L	12.6
Hs-TnT level (peak, ng/L)	4.1
Pro-BNP, pg/L	760 (200-35000
LVEF	46.3
ICU stay, days	3
Postoperative major bleeding	22

Interventional Cardiology / Coronary

PP-016

The relationship between the modified Glasgow prognostic and SYNTAX scores in patients with non-ST elevation myocardial infarction

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Background and Aim: This study aimed to investigate the predictive value of the newly defined the modified Glasgow prognostic score (mGPS) in determining the extent and severity of coronary artery disease (CAD) in comparison with the other inflammatory markers such as neutrophil to lymphocyte ratio (NLR) and C-reactive protein to albumin ratio (CAR) in patients with non-ST-elevated myocardial infarction (NSTEMI).

Methods: This study included 295 consecutive patients with NSTEMI undergoing coronary angiographic examination. mGPS was calculated for each patient at admission. The SYNTAX scoring system was used to assess the severity and extent of CAD

Results: A total of 295 patients, 68 women (23.1%), 227 men (76.9%), with a minimum age of 31, a maximum age of 85, and a mean age of 61.2 ± 10.9 years, were included in the study. The mean SYNTAX score of the patients was 7.3 ± 10.4 (min=0-max=40).The mean body mass index, diabetes mellitus, hyperlipidemia, cerebrovascular disease, smoking rates, left ventricular hypertrophy, heart valve disorder rates of patients with SYNTAX score. >22 were statistically significantly higher than those with SYNTAX score 22 and below (p=0.015, p=0.008, p=0.003, p=0.039, p<0.001, p<0.001, p<0.001). The CRP, CRP/albumin mean mGPS 1-2 ratios of patients with SYNTAX score >22 were statistically significantly higher than those with SYNTAX score ≤22 (p<0.001 for all). The smoking (OR: 3.341, 95% CI: 1.531-7.294, p=0.002), CRP/albumin ratio (OR: 4.958, 95% CI: 1.335-18.418, p=0.017) and mGPS score of 1-2 (OR: 3.121, 95% CI: 1.430-6.814, p=0.004) were found to be independent associates of high SYNTAX score.

Conclusions: mGPS may be associated with high SYNTAX score and may be used to estimate the extent and complexity of CAD in NSTEMI patients.

Table 1. Regression analysis of potential predictor factors for the high SYNTAX Score

	Univariate analysis				Multivariable analysis			
	p	OR	%95 CI		p	OR	%95 CI	
Age	0,136	1,021	0,994	1,049	0,345	1,017	0,982	1,054
Body Mass Index	0,007	1,178	1,046	1,328	0,258	1,098	0,934	1,290
Hypertension	0,074	1,846	0,941	3,620	0,959	0,978	0,421	2,270
Diabetes Mellitus	0,009	2,218	1,220	4,033	0,588	1,278	0,526	3,105
Hyperlipidemia	0,004	2,454	1,329	4,531	0,704	1,183	0,496	2,821
Smoking	0,001	3,033	1,613	5,703	0,002	3,341	1,531	7,294
Left ventricular ejection fraction	0,003	0,932	0,889	0,977	0,237	0,965	0,911	1,023
Crp/albumin	<0,001	14,892	4,867	45,567	0,017	4,958	1,335	18,418
Neu/Lym	0,621	0,984	0,924	1,048	0,364	0,941	0,826	1,073
mGPS (reference:0)								
1-2	<0,001	5,299	2,718	10,333	0,004	3,121	1,430	6,814

Interventional Cardiology / Coronary

PP-017

Which biomarker(s) augment the diagnostic value of the positive exercise electrocardiography test: Systemic inflammatory index, plasma atherogenic index, or monocyte/HDL ratio?

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Background and Aim: The exercise electrocardiography test (EET) is still used before coronary angiography (CAG) in the diagnosis of chronic cardiac syndromes (CCS). In recent years, many biomarkers have been found to be associated with coronary artery disease (CAD), the foremost of which are the systemic inflammatory index (SII), plasma atherogenic index (PAI), and the monocyte/HDL ratio (MHR). The aim of this study was to determine the value of the combination of a positive EET with these 3 biomarkers in the determination of obstructive CAD.

Methods: This single-centre, retrospectve study included 540 patients who underwent CAG after ETT between 2018 and 2021. The patients were separated as Group 1, comprising 434 patients with normal coronary arteries and non-obstructive CAD, and Group 2 including 106 with obstructive CAD. The SII, MHR, and PAI values were calculated for all the patients. In Group 2, the SYNTAX scores were also calculated and the patients were separated into 2 groups of SYNTAX <22 or ≥23.

Results: Advanced age, diabetes mellitus (DM), and male gender were determined at a higher rate in Group 2 (Table 1). Glucose, low-density lipoprotein (LDL), white blood cells, and MHR were determined to be significantly higher in Group 2, and the glomerular filtration rate (GFR) and high-density lipoprotein (HDL) values were determined to be lower (p<0.05) (Table 2). According to the multivariate logistic regression analysis, age, gender, DM, and LDL were determined to be independent predictors of CAD (Table 3). In the ROC curve analysis, a cutoff value of 12 for the MHR in the determination of obtructive CAD had sensitivity of 60.4%, specificity of 53.0%, negative predictive value (NPV) of 84.6%, and positive predictive value (PPV) of 23.9%. No difference was determined between the subgroups of SYNTAX <22 and ≥23 in respect of the MHR, PAI, and SII values (Table 4).

Conclusions: The main result of this study was that a high MHR is an indicator of obstructive CAD in patients with positive EET and suspected CAD.

Table 1. Patient characteristics

	Group 1 (n=434) (NCA and n-obsCAD)	Group 2 (n=106) (obsCAD)	p
Age	54.4 ± 9.6	59.4 ± 9.4	<0.001
Gender (F/M)	190/244 (43.8%/56.2%)	21/85 (19.8%/80.2%)	<0.001
BMI	30.1 ± 5.2	29.1 ± 4.4	0.067
SBP, mmHg	117.6 ± 13.6	120.1 ± 15.1	0.128
DBP, mmHg	76.5 ± 7.8	77.4 ± 7.3	0.292
Pulse, pulse/min	86.4 ± 15	87.3 ± 13.8	0.562
HT	88 (20.3%)	16 (15.1%)	0.282
DM	60 (13.8%)	27 (25.5%)	0.003

BMI: Body mass index, DBP: Diastolic blood pressure, DM: Diabetes mellitus, F: Female, M: Male, HT: Hypertension, NCA: Normal coronary arteries, n-obsCAD: Non-obstructive coronary artery disease, obsCAD: Obstructive coronary artery disease, SBP: Systolic blood pressure.

Table 2. Laboratory data

	Group 1 (n=434) (NCA and n-obsCAD)	Group 2 (n=106) (obsCAD)	p
Glucose, mg/dL	102.5 (93-122)	109.5 (95.5-146.8)	0.006
GFR, mL/min/1.73 m^2	95.3 ± 14.5	90.1 ± 13.5	0.001
LDL, mg/dL	117.7 ± 35.8	125.9 ± 34.6	0.033
Triglyceride, mg/dL	157 (112.8-223)	163 (126.8-249.5)	0.160
HDL, mg/dL	43 (37-51)	40.5 (34-47)	0.010
WBC, /µL	7254.8 ± 1823.8	7666.04 ± 2047.0	0.043
Neutrophil, /µL	4304.2 ± 1421.3	4541.23 ± 1542.2	0.131
Lymphocyte, /µL	2210.1 ± 627.9	2305.3 ± 748.7	0.179
Monocyte, /µL	545.1 ± 163.6	577.0 ± 193.5	0.120
Hemoglobin, g/L	14.6 ± 1.7	14.9 ± 1.7	0.162
Platelet; 10^3/µL	255.6 ± 55.5	261.3 ± 70.9	0.446
MHR	13.1 ± 5.5	14.7 ± 6.3	0.019
SII	479 (361.8-632.3)	478 (345.3-660.8)	0.848
PAI	0.21 ± 0.269	0.259 ± 0.274	0.094

CAD: Coronary artery disease, GFR: Glomerular filtration rate, HDL: High density lipoprotein, LDL: Low density lipoprotein, MHR: Monocyte HDL ratio, NCA: Normal coronary arteries, n-obsCAD: Non-obstructive coronary artery disease, obsCAD: Obstructive coronary artery disease, PAI: Plasma atherogenic index, SII: Systemic inflammatory index, WBC: White blood cell.

Table 3. Multiple logistic regression analysis results for Table 1-2

	OR (95% CI)	p
Age	1.058 (1.034-1.084)	<0.001
Gender (male vs. female)	3.652 (2.137-6.239)	<0.001
DM	2.239 (1.285-3.903)	0.004
LDL	1.009 (1.002-1.015)	0.007

DM: Diabetes mellitus, LDL: Low density lipoprotein.

Table 4.Comparison of PAI, SSI, MHR values according to SYNTAX score in patients with major CAD

Major CAD	SYNTAX <22 (n=82)	SYNTAX 23≤ (n=24)	p
PAI	0.251 ± 0.275	0.290 ± 0.273	0.543
SII	478 (369-652)	471 (293.5-815.3)	0.711
MHR	14.5 ± 6.1	15.3 ± 7.1	0.616

CAD: Coronary artery disease, MHR: Monocyte HDL ratio, PAI: Plasma atherogenic index, SII: Systemic inflammatory index.

Cardiovascular Surgery

PP-018

Long-term survival on LVAD support

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Background and Aim: Preoperative variables can predict short-term survival with a left ventricular assist device (LVAD). However, predictors of long-term survival remain poorly characterised. Therefore, patients with long-term follow-up after LVAD in our centre were evaluated.

Methods: Data on patients who underwent LVAD implantation at a single tertiary centre between 2012 and 2022 were retrospectively reviewed. 495 consecutive patients were four grouped according to time on support 1, short-term (<1 year, n=173), 2. mid-term (1-3 years, n=124) and 3. mid-term (3-5 years, n= 82), 4. long-term (>5 years, n=126). Mortality rates and causes of death were compared according to groups.

Results: 495 patients (mean age was 54 ± SD and 87.7% male) included in the study. The 49.1% had ischemic etiology and 72.1% had heart ware. Mean follow up was 35 ± 34 months, no significant difference was found between the groups in terms of survival time and device type. In group 4 patients with long-term follow-up, early mechanical ventilation duration after LVAD was short and per-operative IABP, ECMO and levetronix were not used. There was no significant correlation between severity of mitral and aortic regurgitation and follow-up time (p=0.522). There was a 22.6% mortality rate in the study. The majority of these deaths occurred within 1 year of short-term follow-up (14.7%), and the rate decreased to 2.8% at long-term follow-up (p=0.001). When analysing causes of death, it was found that the most common long-term cause of death was pump thrombosis (p=0.006).

Conclusions: Prolonged survival is severely limited by the incidence of infection and postoperative end-organ dysfunction. Long-term pump thrombosis is most frequently life-threatening. The broadening of the therapeutic target makes it imperative that future LVAD trials be designed with adequate follow-up to capture outcomes beyond 24 months.

Table 1. Comparison of preoperative characteristics and causes of death between groups

	All patients (n=495)	Group 1 (<1 year, n=124)	Group 2 (1-3 years, n=82)	Group 3 (3-5 years, n=103)	Group 4 (>5 years, n=13)	p
Male, n (%)	434 (87.7)	151 (30.5)	108 (21.8)	66 (13.3)	109 (22)	0.041
Heart failure etiology, n (%)						
-Ischemic CMP	243 (49.1)	82 (16.6)	62 (12.5)	40 (8.1)	59 (11.9)	0.943
- Dilated CMP	251 (50.7)	91 (18.4)	61 (12.3)	42 (8.5)	57 (11.5)	0.922
LVAD						
Heart mate II	57 (11.5)	15 (3)	15 (3)	10 (2)	17 (3.4)	0.356
Heart mate III	80 (16.2)	23 (4.6)	19 (3.8)	18 (3.6)	20 (4)	
Heart ware	357 (72.1)	135 (23.7)	90 (18.2)	54 (10.9)	78 (15.8)	
Exitus, n (%)	112 (22.6)	73 (14.7)	17 (3.4)	8 (1.6)	14 (2.8)	0.001
DI	81 (1.8)	17 (3.7)	16 (3.4)	20 (4.3)	28 (6)	0.001
GMB	51 (11.2)	8 (1.8)	10 (2.2)	10 (2.2)	23 (5)	0.001
PT	122 (2.6)	30 (6.4)	29 (6.2)	21 (4.5)	42 (9)	0.006

CMP: Cardiomyopathy, DI: Driveline infection, GMB: Gastrointestinal-mucocutaneous bleeding, PT: Pump thrombosis.

Heart Valve Diseases

PP-019

Does valvulo-metabolic disease exist: Meta analysis of the current literature

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Background and Aim: Obesity has emerged as a pandemic of the 21st century, with significant implications for cardiovascular disease. While its impact on heart valve disease has been a subject of interest, the existing literature remains inconclusive. Therefore, this meta-analysis aims to explore the potential effects of obesity on different types of heart valve disease and their prevalence, based on the latest available evidence.

Methods: A comprehensive and meticulous literature review was conducted. Specific search terms such as "obesity", "prevalence", and the name of each valve type (mitral, aortic, tricuspid) were used in the inquiry on the MEDLINE database. Our inclusion criteria were limited to studies that involved both control and obesity groups and reported valve disease prevalence. Statistical analyses were performed using the Jamovi program.

Results: The initial search yielded a total of 1036 studies. After excluding irrelevant studies, only six studies comprising a total of 12.143.168 patients were included in this analysis. Among the selected studies, two reported on aortic stenosis, one on aortic insufficiency, one on general aortic valve disease, and two on mitral disease. The synthesis of data indicated that obesity has a significant impact on aortic stenosis (95% CI: 0.00637-0.0112, I2: 28.7%). However, it did not show a significant association with general aortic valve disease (95% CI: -0.00269-0.0108, I2: 96.6%), and mitral regurgitation stenosis (95% CI: -0.124-0.0394, I2: 83.9%). Interestingly, an inverse association between obesity and both aortic and mitral regurgitation was observed, but it did not reach statistical significance.

Conclusions: This meta-analysis found a positive association between obesity and aortic stenosis, suggesting that obesity may play a role in its pathogenesis. However, no significant association was observed between obesity and aortic or mitral regurgitation. It is essential to note that the current body of evidence on the relationship between obesity and valve disease prevalence is limited. Further research and larger studies are warranted to provide more definitive conclusions in this area.

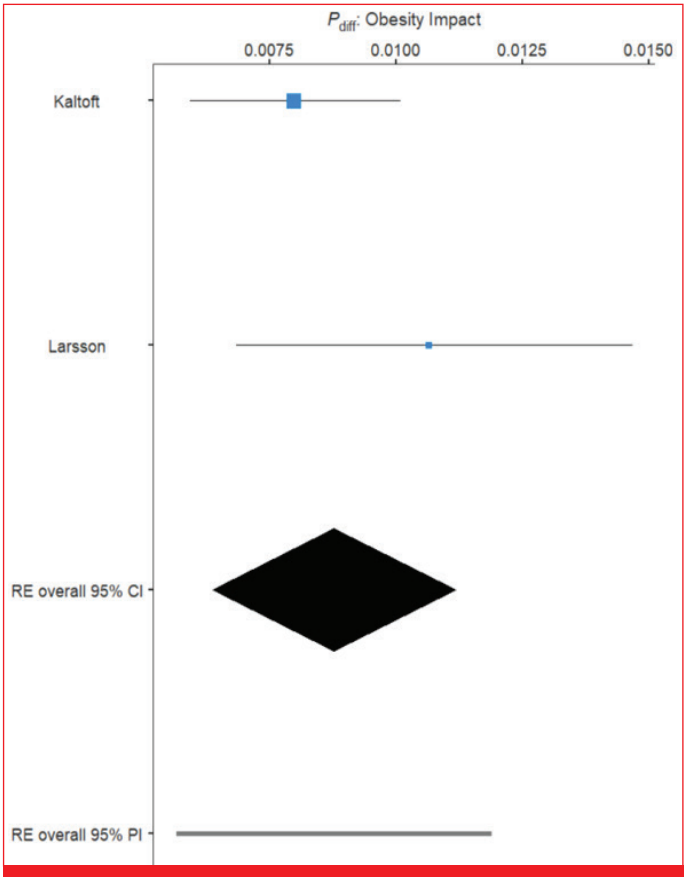

Figure 1. Forest plot of obesity impact on aortic stenosis.

Table 1. Heart valve disease prevalence in patients with obesity

Author	Year	Disease	Patient size	Valve disease prevalence in patients with obesity	Valve disease in control
Kaltoft	2020	Aortic stenosis	107.916	1.8%	1.0%
Larsson	2017	Aortic stenosis	71.917	2.8%	1.7%
Lebowitz	2000	Aortic regurgitation	3501	9.2%	11.1%
Haseefa	2022	Aortic valve diseases	11.958.944	1.2%	1.2%
Bartowiak	2021	Mitral regurgitation	681	1.3%	18.0%
Alpert	1993	Mitral regurgitation	195	1.1%	10.0%

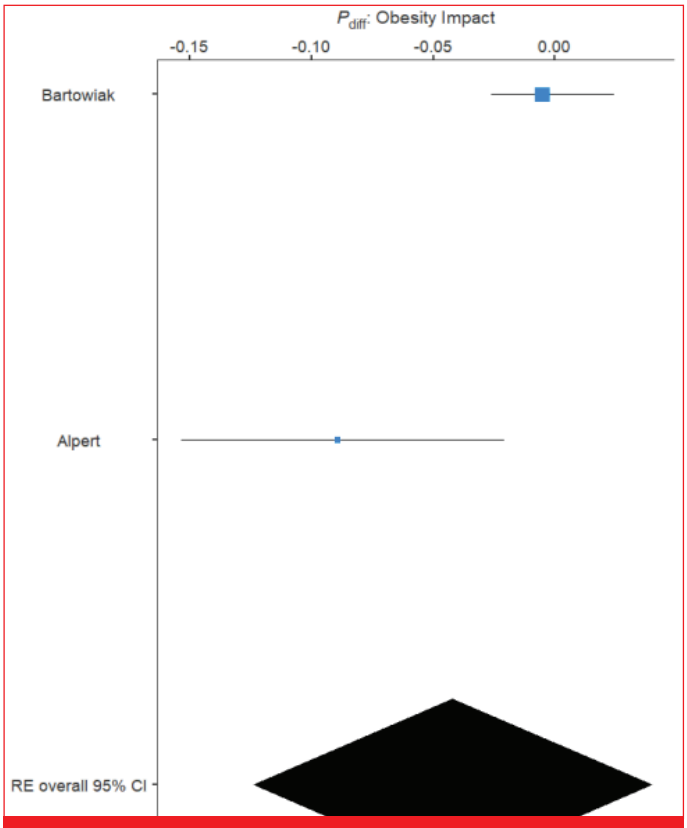

Figure 2. Forest plot of obesity impact on mitral regurgitation.

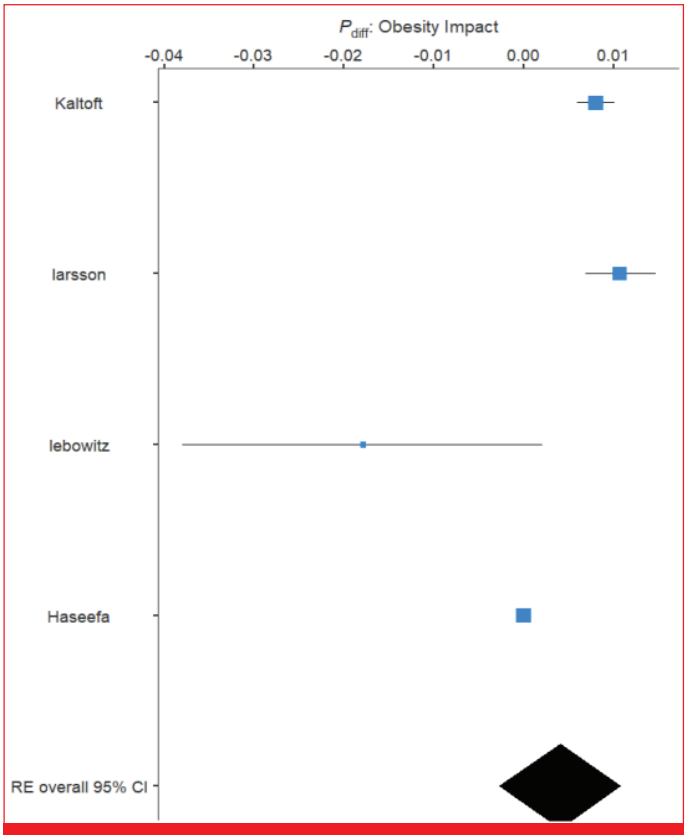

Figure 3. Forest plot about obesity impact on both aortic regurgitation and stenosis.

Heart Failure

PP-020

Evaluation of the effect of prognostic nutritional index on mortality in acute heart failure

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Background and Aim: Acute heart failure (AHF) constitutes a critical medical condition that necessitates immediate therapeutic intervention and continuous monitoring. Despite advancements in pharmacological and device-based therapies, AHF remains a major public health issue with high morbidity, hospital readmission rates, and substantial mortality. An emerging area of research that has gained significant attention is the potential role of nutritional status as an independent prognostic factor in AHF outcomes. The prognostic nutritional index (PNI), initially designed to evaluate preoperative nutritional status and systemic inflammatory response in oncologic patients, has been increasingly explored in the context of cardiovascular diseases. However, comprehensive data elucidating the relationship between PNI and mortality risk in AHF patients are notably sparse. This study aims to bridge this gap by investigating the impact of the PNI on mortality rates among patients admitted with acute heart failure.

Methods: This study is a single-center, retrospective analysis aimed at investigating the prognostic implications of the PNI in patients hospitalized for acute heart failure (AHF). A total of 245 patients admitted to a tertiary medical center for AHF were included in the analysis. PNI was calculated as 10 x serum albumin (g/dL) + 0.005 x total lymphocyte count (per mm^3). Patients were followed for an average duration of one year after hospital admission. The primary endpoint for this study was all-cause mortality. Statistical analyses were conducted to evaluate the independence and robustness of PNI as a predictor of mortality. Multivariate logisctic regression models were employed to adjust for potential confounders.

Results: 245 patients were included in the study retrospectively. The mean age was 60.5 ± 14.2 years and 173 (70%) of them were male. In the group with mortality, PNI and albumin were lower (p<0.001, p≤0.001); age were higher (p≤0.001); etiology, other comorbidities, hemoglobin and creatinine levels were similar (Table 1). Both groups were similar in terms of prescribed medications, as outlined in Table 2. Multivariable regression analysis showed; age and PNI were determined as independent risk factors for all couse mortality (age; OR: 1.044, 95% CI: 1.018-1.070, p≤0.001) (PNI; OR: 0.953, 95% CI: 0.918-0.990, p=0.013) (Table 3).

Conclusions: In summary, our investigation elucidates the PNI as an independent predictor of all-cause mortality in patients hospitalized for AHF. Remarkably, the predictive capacity of PNI remained robust even after adjustment for sex, estimated glomerular filtration rate (eGFR), left ventricular ejection fraction (LVEF), prescribed medications and hemoglobin levels. Derived from routine biochemistry and hemogram tests, PNI stands as a cost-effective and easily accessible metric that holds significant promise for clinical utility.

Table 1.

	All patients (n=245)	Patient with primer endpoint (n=94)	Patient without primer endpoit (n=151)	p
Age	60.51 ± 14.2	66.4 ± 13.9	56.9 ± 13.1	<0.001
Male	173 (68%)	65 (69%)	108 (71%)	0.692
DM	90 (36%)	35 (37%)	55 (36%)	0.898
HT	155 (63%)	70 (74%)	85 (54%)	0.004
Ischemic etiology	113 (46%)	50 (53%)	63 (40%)	0.08
Creatinine, mg/dL	1.8 ± 6.0	1.9 ± 6.7	1.7 ± 5.5	0.741
Na, mEq/L	136.5 ± 4.1	136.4 ± 4.7	136.5 ± 3.8	0.866
K, mEq/L	4.5 ± 3.0	4.4 ± 0.6	4.5 ± 3.8	0.642
Albumin, g/dL	3.6 ± 0.5	3.4 ± 0.5	3.7 ± 0.5	<0.001
Hb, g/dL	12.4 ± 1.9	11.9 ± 2.0	12.7 ± 1.7	0.002
PNI	45.6 ± 9.0	42.4 ± 9.0	47.7 ± 8.4	<0.001
LVEF	27.6 ± 8.1	28.4 ± 7.9	27.1 ± 8.2	0.226

Table 2. Medication utilization

	All patiens (n=245)	Patient with primer endpoit (n=94)	Patient without primer endpoit (n=151)	p
ACE inhibitors	198 (71%)	73 (77%)	125 (82%)	0.322
ARB	18 (7%)	5 (5%)	13 (8%)	0.337
Beta-blocker	243 (99%)	93 (99%)	150 (99%)	0.734
Statine	103 (42%)	40 (42%)	63 (41%)	0.898
Antiplatelet agent	143 (58%)	51 (54%)	92 (60%)	0.303
Anticoagulant	126 (51%)	54 (57%)	72 (47%)	0.137
Aldosterone antagonist	142 (57%)	51 (54%)	91 (60%)	0.354

Table 3. Univariate-multivariate regression analysis

	Univariate	Regression	Analysis	Multivariate	Regression	Analysis
	OR	95% CI	p	OR	95% CI	p
Age	1.054	1.032-1.077	<0.001	1.044	1.018-1.070	<0.001
HT	0.442	0.251-0.776	0.005	0.972	0.483-1.955	0.936
Hb	0.801	0.695-0.922	0.002	0.897	0.763-1.055	0.190
PNI	0.925	0.892-0.958	<0.001	0.953	0.918-0.990	0.013

Heart Failure

PP-021

The value of HFA-PEFF and H2FPEF scores in distinguishing underlying cardiac amyloidosis in patients with heart failure and preserved ejection fraction

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Background and Aim: Heart failure with preserved ejection fraction (HFpEF) is a complex clinical syndrome with heterogeneous underlying etiologies. Transthyretin (TTR) cardiac amyloidosis (CA) is an underestimated and underdiagnosed cause of HFpEF. The HFA-PEFF and H2FPEF scores have been developed to diagnose HFpEF. The value of these scores in distinguishing patients with HFpEF caused by CA is not clear. In this study, we aimed to reveal the value of these scores in distinguishing the underlying diagnosis of CA in patients with HFpEF.

Methods: This single-center, prospective study included 213 patients who had a diagnosis of HFpEF according to 2016 ESC HF guidelines. Patients with severe primary valvular diseases, previous myocardial infarction, sarcomeric hypertrophic cardiomyopathy or myocardial storage diseases were excluded from the study. The H2FPEF and HFA-PEFF score are calculated. Thus, according to the H2FPEF and HFA-PEFF score, patients were ranked as having a low, intermediate and high probability of HFpEF when H2FPEF score was 0-1, 2-5 and >5, respectively, HFA-PEFF score was 0-1, 2-4 and 5-6, respectively. 99mTc-PYP cardiac scintigraphy was performed in 167 patients who have ≥2 red flags for CA. In the absence of monoclonal protein in the serum and urine, Grade

2 to 3 myocardial uptake or a Heart/Lung (H/L) ratio of ≥1.5 post injection of 99mTc-PYP on planar and SPECT images is considered positive for TTR-CA.

Results: Mean age of study population was 69.33 ± 11.1 years. 124 patients (74.3%) had a prior hypertension (HT), left ventricular (LV) hypertrophy (IVS ≥12 mm) was present in 73.1% of patients (n=122). Mean LV ejection fraction was 60.5% ± 5.3, mean left ventricular global longitudinal strain was -14.3% ± 4.7. 99mTc-PYP cardiac scintigraphy showed that 31 (18.5%) of the patients have had a grade 2 or 3 cardiac uptake. In 25 (14.9%) of these patients, the H/L ratio was ≥1.5 and concordance was positive. 136 (81.4%) patients were evaluated as Grade 1 or 0. There were 1 (0.6%) patient with an HFA-PEFF score of 0-1, 12 (7.2%) patients with a score of 2-4, and 154 (92.2%) patients with a score of 5-6. According to the H2FPEF score; 8 (4.8%), 87 (52.1%) and 72 (43.1%) patients were classified as low (0-1), moderate (2-5) or high (6-9), respectively (Figure 1). In the whole population, the mean HFA-PEFF score was 5.6 ± 0.8, the mean HFA-PEFF score of the grade 2-3 group was 5.7 ± 0.5, while the mean HFA-PEFF score of the grade 0-1 group was 5.6 ± 0.8, and there was no significant difference between the two groups (p=0.884). In addition, the mean H2FPEF score of all patients was 5 ± 2.2, the mean H2FPEF score of the grade 2-3 group was 5.4 ± 2.5, while the mean H2FPEF score of the grade 0-1 group was 4.9 ± 2.2, and there was no significant difference between the two groups (p=0.385) (Table 1).

Conclusions: The H2FPEF and HFA-PEFF scores used to diagnose HFpEF are not effective in predicting CA in the underlying etiology. Simple and easy methods to predict CA at first glance are needed for early diagnosis.

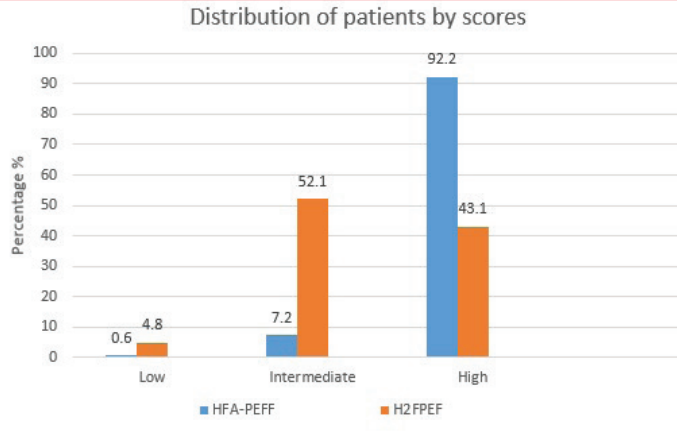

Figure 1. Distribution of patients by scores.

Table 1. The comparison of H2FPEF and HFA-PEFF scores according to grade groups

	Grade 0-1	Grade 2-3	p
HFA-PEFF score, mean ± SD	5.6 ± 0.8	5.7 ± 0.5	0.884
H2FPEF score, mean ± SD	4.9 ± 2.2	5.4 ± 2.5	0.385

Heart Failure

PP-022

The effect of changes in ferritin levels on NT-pro-BNP levels in the presence or absence of anemia in heart failure patients

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Background and Aim: Iron deficiency is a common and serious comorbidity in heart failure patients, regardless of whether it causes anemia. Iron deficiency is linked to reduced exercise capacity, frequent hospitalization for heart failure-related issues,and poorer outcomes. The European Society Cardiology Heart Failure Guideline, published in 2021, defines iron deficiency as having a ferritin level below 100 ng/mL or a ferritin level between 100-299 ng/mL with transferrin saturation below 20% and recommends intravenous replacement of iron despite the absence of anemia. This study aims to investigate whether changes in ferritin levels have an effect on NT-pro-BNP in heart failure patients, with or without anemia.

Methods: This observational study prospectively examines 335 patients with heart failure who were followed at an outpatient heart failure clinic. Between 2019 and 2023, this study investigated 1593 regular clinic follow ups. Linear mixed regression modeling was used for repeated serum ferritin measures on NT-pro-BNP changes and split to the presence of anemia.

Results: The group had an average age of 50.5 years, with a standard deviation of 13.2 years. Additionally, 73.1% of the group consisted of male individuals. The mean LVEF for heart failure patients was 36.1 ± 14.2%, and the majority of the patients 86.3%, were classified as NYHA functional class I-II. During the initial visit of patients, it was discovered that 21.2% of them had anemia, as per the WHO guidelines that consider the gender of each patient. During their follow-up appointments, 27 patients were given an intravenous iron replacement. However,the measurements taken after this treatment were not included in the analysis. Regarding multiple measurements of iron metabolism parameters, 51.3% of ferritin levels were below 100 ng/mL, 39.9% were within the 100-299 ng/mL range, and 8.7% were over 300 ng/mL. Among the ferritin levels within the 100-299 ng/mL range, 25% showed transferrin saturation levels below 20%. To conduct a linear mixed model analysis of repeated measures, the NT-pro-BNP levels change in relation to ferritin, and the patient's heart failure status should be considered as the dependent variable. This analysis should be performed for each subject and visited separately. For patients with anemia and ferritin changes, there was a notable influence on their NT-pro-BNP levels (F=47.652, p=0.001). However, there was no significant effect on patients without anemia and ferritin changes on their NT-pro-BNP levels (F=0.63, p=0.99).

Conclusions: Based on these findings, it appears that heart failure patients without anemia do not experience any impact on heart failure-related biomarker NT-pro-BNP from changes in their ferritin levels. However, heart fail-

ure patients with anemia and corresponding ferritin level changes were found to have a correlation with NT-pro-BNP levels. These results suggest that intravenous iron infusion may be beneficial for treating iron deficiency with anemia, although further investigation is needed to confirm this hypothesis.

Table 1. Baseline characteristics according to anemia status in heart failure patients

	Total	Anemia (-)	Anemia (+)	p
Age, years	50.5 ± 13.2	49.7 ± 13.2	53.9 ± 12.9	0.016
Male, %	73.1	73.6	81.7	0.15
BMI, kg/m^2	28.8 ± 20.7	27.7 ± 4.5	27.2 ± 5	0.51
BSA, m^2	1.9 ± 0.19	1.9 ± 0.19	1.9 ± 0.19	0.26
SBP, mmHg	118 ± 20	118 ± 20	117 ± 19	0.63
DBP, mmHg	75 ± 13	75 ± 12	73 ± 15	0.24
HR, beat/min	76 ± 14	76 ± 14	74 ± 15	0.48
NYHA FC, %	-	-	-	0.14
I	46.3	49	35.8	-
II	40	38.3	46.3	-
III-IV	13.8	12.6	17.9	-
Ischemic etiology, %	35.3	33.8	40.8	0.14
Arterial hypertension, %	26.9	27.4	25.4	0.73
Diabetes mellitus, %	23.7	20.9	33.8	0.023
Dyslipidemia, %	21.3	19.4	28.2	0.10
Smoking history, %	46.7	46.4	47.9	0.82
Cerebrovascular event, %	4.5	4.9	2.8	0.56
COPD, %	12.6	12.9	11.3	0.70
Renal disease, %	0.6	0.8	0	0.46
Chronic liver disease, %	1.8	1.1	4.2	0.08
Peripherial arterial disease, %	0.6	0.6	0	0.46
AF, %	13.3	12.5	17.5	0.51
Intracardiac device	-	-	-	0.29
CRT, %	7.8	9.1	2.8	-
ICD, %	34.7	34.2	36.6	-
ACEi or ARB, %	73.9	62	71.4	0.048
ARNI, %	9.3	10	7.0	0.45
Beta-blocker, %	88.3	90	81.7	0.053
Aldosterone antagonist, %	59	61.3	50.7	0.10
SGLT2-i, %	11.7	12.6	8.5	0.33
Loop diuretic, %	49.7	47.1	59.2	0.07
Anticoagulant, %	24.1	23.8	25.4	0.78
Antiplatelet, %	45.2	47.9	35.2	0.56
If channel blocker, %	16.5	16.7	15.5	0.80
Digoxine, %	5.7	6.5	2.8	0.23
Antihyperlipidemic, %	31.4	31.2	32.4	0.84

* Values are mean ± SD or n (%) p<0.05. Anemia; Hb <12 g/dL female, Hb <13 g/dL male. BMI: Body mass index, BSA: Body surface area, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, HR: Heart rate, NYHA FC: New York Heart Association functional classification, COPD: Chronic obstructive pulmonary disease, AF: Atrial fibrillation, CRT: Cardiac resynchronization therapy, ICD: Implanted cardioverter-defibrillator, ACEi:Angiotensinogen was converting enzyme inhibitor, ARB: Angiotensin receptor blocker, ARNI: Angiontensin receptor neprilysin inhibitor, SGLT2-i: Sodium-glucose co-transporter-2 inhibitors.

Table 3. Echocardiographic parameters of the heart failure patients according to anemia status

	Total	Anemia (-)	Anemia (+)	p
LVEDd, mm	5.91 ± 1.05	5.88 ± 1.07	5.97 ± 0.93	0.57
LVESd, mm	4.68 ± 1.26	4.67 ± 1.26	4.68 ± 1.21	0.96
LAd, mm	4.25 ± 0.84	4.28 ± 0.83	4.12 ± 0.88	0.15
LVEF, %	36.1 ± 14.2	35.8 ± 14.3	37.4 ± 13.6	0.41
TAPSE, mm	19.8 ± 4.7	19.3 ± 5.7	20.2 ± 4.4	0.30
RVsm TDI, m/sec	10.6 ± 2.8	10.5 ± 2.9	10.9 ± 2.5	0.45
TRV, m/sec	2.57 ± 0.65	2.61 ± 0.66	2.42 ± 0.57	0.62
SPAP, mmHg	50.2 ± 16.9	50.5 ± 16.9	49.4 ± 16.8	0.62
Mitral regurgitation, %	-	-	-	0.66
None	22.8	24.0	18.3	-
Mild	43.1	42.2	46.5	-
Moderate	25.1	25.5	23.9	-
Severe	9	8.4	11.3	-
Aort regurgitation, %	-	-	-	0.79
None	79.9	80.2	78.9	-
Mild	16.5	16.0	18.3	-
Moderate	0.9	0.8	1.4	-
Severe	2.7	3.0	1.4	-
Tricuspide regurgitation, %	-	-	-	0.22
None	30.8	30.0	33.8	-
Mild	51.8	55.5	38	-
Moderate	11.1	9.1	18.3	-
Severe	6.3	5.3	9.9	-

* Values are mean ± SD or n (%) p<0.05. Anemia; Hb <12 g/dL female, Hb <13 g/dL male. LVEDd: Left ventricular end diastolic diameter, LVESd: Left ventricular end systolic diameter, LAd: Left atrial diameter, LVEF: Left ventricular systolic ejection fraction, TAPSE: Tricuspid annuler plane systolic excursion, RVsm: Right ventricular systolic motion tissue doppler imaging, TRV: Tricuspid regurgitation velocity, SPAP: Systolic pulmonary artery pressure.

Table 4. Biochemical and hemogram parameters of the heart failure patients according to anemia status

	Total	Anemia (-)	Anemia (+)	p
Hemoglobin, g/dL	13.9 ± 1.89	14.6 ± 1.2	11.3 ± 1.4	-
Hematocrit, %	42.2 ± 5.08	44 ± 3.6	35.5 ± 4.0	-
WBC, /µL	8069 ± 2169	8272 ± 2145	7295 ± 2414	0.001
PLT, 10^3/µL	237 ± 71	235 ± 63	243 ± 97	0.52
Ferritin, ng/mL (IQR)	96.4 (50.9-176)	105 (52.3-177)	91.7 (45.7-180)	0.75
Serum iron, µg/dL	79.9 ± 34.4	85.3 ± 33.7	53.8 ± 24.4	<0.0001
TSAT, %	24.8 ± 11.8	26.6 ± 11.4	17.8 ± 10.7	<0.0001
NT-pro BNP, pg/mL (IQR)	653 (186-1753)	569 (170-1393)	1172 (361-5229)	0.001
Creatinine, mg/dL (IQR)	0.91 (0.76-1.12)	0.91 (0.76-1.08)	0.96 (0.70-1.32)	0.086

*Values are mean ± SD or n (%) p<0.05. Anemia; Hb <12 g/dL female, Hb <13 g/dL male. Hb: Hemoglobin, WBC: White blood cell, PLT: Platelet, TSAT: Transferrin saturation, NT-pro-BNP: N-terminal pro b-type natriuretic peptide, IQR: Interquartile range.

Heart Failure

PP-023

Cardiologists' attitudes towards worsening heart failure

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Background and Aim: Worsening heart failure (WHF), although no consensus has been established on definition yet, might be defined as deterioration of heart failure (HF) signs and symptoms in a patient with chronic HF (despite previous stable background guideline directed medical therapy) or requires urgent escalation of therapy: including hospitalization, emergency department visit, or outpatient IV diuretic therapy (± outpatient oral therapy). Because of lack of distinct criteria on definition and robust evidence for guideline recommendations (data come from inclusion and exclusion criteria of recent clinical trials, and these are even changeable), awareness level of clinicians on this entity is not well-known. Our aim was to evaluate cardiologists' attidutes to WHF.

Methods: We performed a questionare which was costructed spesifically on WHF definition and adjunctive therapeutic options in order to evaluate the awareness level of clinicians nationwide in Türkiye between May 2023 and July 2023. The survey contains a group of questions which were questioning the participant's work experience, his/her center's experience on advanced heart failure therapies, the participant's approach in certain circumstances such as initial treatment preference and follow-up strategy. We also asked for how the participant describe WHF pnenomenon and whether he/she was aware of advanced treatment options for particular situations and also how often refers such patient population to centers capable of applying advanced therapies for heart failure.

Results: We analyzed a total of 240 cardiologists' survey results. Most of participants have been working as a cardiology specialist for more than 5 years (n=177, 74%), and also 74% of them have been working in a tertiary center. However, only 26% (n=63) of participants' center was capable of applying advanced therapies such as left ventricular asist device (LVAD), heart transplantation, MitraClip© and TriClip©. We found that most of physicians were implementing guideline directed medical therapy in patients with de novo and "stable" heart failure (beta-blocker, ARNI/ACE inhibitor, SGLT-2 inhibitor and MRA; 89% of participants initiates at least two of them at the same time) and appropirate follow-up intervals. Half of participants declared that they first up-titrate beta-blockers (51%) whereas 41% of participants' first choice for uptitration was ARNI/ACE inhibitor. The answers about: the definition of WHF, the risk of mortality in patients with HF and also in patients with LVAD were presented in Table 1. Lastly, we asked which patients should be referred to a center specified in advanced HF therapies, and results were shown in Table 1.

Conclusions: We found in this survey that majority of cardioloists' awareness level for WHF was low, and we may offer that a cosensus report need to be published by our National Heart Failure Working Group about which particular patient population should be referred to a center specified in advanced HF therapies.

Table 1. Survey results

Question/Options	Number of participants (%)
Which of them defines WHF? (you can choose more than one)	
Hospitalization for HF	225 (95)
IV diuretic therapy in ED	198 (83)
IV diuretic therapy in outpatient clinic	155 (65)
Need to uptitrate oral loop diuretic dose or adding HCTZ/acetazolamide	137 (57)
Need to change furosemide into torasemide	53 (22)
How many heart failure patients have you referred to an advanced center for last 6 months?	
0-2	136 (57)
3-5	56 (24)
6-10	18 (8)
>10	27 (11)
What is the 5-year mortality rate of a patient with de novo HF?	
25%	37 (15)
30%	67 (28)
50%	123 (51)
70%	12 (5)
What is the 5-year survival rate of a HF patient after LVAD implantation?	
30%	119 (50)
60%	89 (38)
80%	29 (12)
Which patient do you refer to a center capable of advanced HF therapies? (you can choose more than one)	
Needing positive inotropic therapy	151 (66)
Persistent NYHA class 4 and/or persistent elevated BNP/NT-pro-BNP levels	161 (70)
Always with systolic blood pressure <90 mmHg	105 (46)
Ejection fraction <20%	97 (42)
Recurrent and appropirate ICD shocks	135 (59)
More than one hospitalization for decompensated HF within 1 year	108 (47)
Persistent hypervolemia and/or need for uptitration of diuretic dose	85 (37)
Inability to uptitrate or contraindication to GDMT	74 (32)

WHF: Worsening heart failure, IV: Intravenous, ED: Emergency department, HCTZ: Hydrochlorothiazide, HF: Heart failure, LVAD: Left ventricular asist device, BNP: Brain natriuretic peptide, NT-pro-BNP: N-terminal pro-brain natriuretic peptide, ICD: Implantable cardioverter defibrillator.

Cardiac Imaging / Echocardiography

PP-024

Demonstration of subclinical left ventricular electrical and mechanical dysfunction in overweight subjects by frontal QRS-T angle and 3D-speckle tracking echocardiography

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Background and Aim: Overweightness is a considerable step in the process leading to obesity. There are no sufficient studies on the effect of cardiomyopathy defined in obese patients about overweight subjects. We thought that it may be useful to examine the myocardial involvement in overweight individuals electro-mechanically with more sensitive techniques before the development of obesity cardiomyopathy. The aim of the present study was to demonstrate whether or not there are subclinical left ventricular (LV) electrical and mechanical dysfunctions in overweight patients using frontal QRS-T (fQRS-T) angle (electrically) and 3D-speckle tracking echocardiography (mechanically).

Methods: A total of 80 overweight patients and 80 age- and sex-matched normal weight individuals were enrolled into the study. 3D-STE examinations of the patients were performed. Electrocardiographic recordings were obtained for fQRS-T angle assessment.

Results: The LV-GLS and LV-GCS were significantly depressed in the overweight group than in the normal weight group (-14.5 ± 3.4 vs. -21.7 ± 3.6, p<0.001; -15.2 ± 4.6 vs. -24.3 ± 4.8, p<0.001, respectively). The fQRS-T angle was found to be increased in the overweight group (142.5 ± 39.2 vs. 114.7 ± 43.5, p≤0.001). Statistically significant positive linear correlations were observed between BMI with LV-GLS, LV-GCS, and fQRS-T angle. LV-GLS and LV-GCS were found to be disrupted linearly as BMI increased (r=0.718 for BMI and LV-GLS, r=0.653 for BMI and LV-GCS). As BMI increased, it was found that the fQRS-T angle increased (r=0.692 for BMI and fQRS-T angle).

Conclusions: Our results support that, overweight individuals, despite their being apparently healthy, may have subclinical LV myocardial mechanical and electrical dysfunction.

Table 1. Clinical, electrocardiographic and laboratory data of the study population

	Normal weight group (n=80)	Overweight group (n=80)	p
Age	53.6 ± 10.4	54.8 ± 9.8	0.487
Gender (female), n (%)	41 (51.2)	42 (52.5)	0.862
BMI, kg/m^2	22.3 ± 2.5	27.4 ± 2.2	0.019
Heart rate, bpm	74 ± 13	79 ± 15	0.443
Frontal QRS-T angle, °	114.7 ± 43.5	142.5 ± 39.2	<0.001
QT interval, ms	429 ± 48	436 ± 45	0.316
QTc interval, ms	457 ± 40	468 ± 38	0.207
Hypertension, n (%)	55 (68.7)	61 (76.2)	0.023
Hyperlipidemia, n (%)	27 (33.7)	29 (36.2)	0.762
Diabetes mellitus, n (%)	25 (31.2)	26 (32.5)	0.803
Smoking, n (%)	19 (23.7)	23 (28.7)	0.065
Fasting glucose, mg/dL	127.4 ± 39.7	132.5 ± 42.2	0.218
Creatinine, mg/dL	0.89 ± 0.3	0.92 ± 0.3	0.647
Hemoglobin, g/dL	13.8 ± 1.2	14.2 ± 1.5	0.371
Platelet, K/uL	292000 ± 113000	295000 ± 117000	0.512
TC, mg/dL	168.4 ± 43.5	171.3 ± 42.4	0.562
LDL-C, mg/dL	107.8 ± 42.8	120.3 ± 41.6	0.054
HDL-C, mg/dL	46.3 ± 9.2	41.4 ± 9.9	0.415

BMI: Body mass index, QTc: Corrected QT interval, TC: Total cholesterol, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol.

Table 2. Two and three dimensional echocardiographic data

	Normal weight group (n=80)	Overweight group (n=80)	p
LVSWT, mm	10.3 ± 1.5	10.7 ± 1.8	0.144
PWT, mm	9.2 ± 1.6	9.8 ± 1.5	0.681
LVEDD, mm	45.9 ± 4.6	47.2 ± 4.8	0.206
LVESD, mm	32.3 ± 3.5	33.4 ± 3.2	0.715
Left atrium diameter, mm	36.4 ± 4.4	38.8 ± 4.6	0.103
LAVI, mL/m^2	24.9 ± 6.3	26.8 ± 6.9	0.079
LV mass index, g/m^2	80.4 ± 17.5	86.9 ± 18.2	0.053
E/A	1.33 ± 0.3	1.27 ± 0.3	0.518
Lateral e', cm/s	10.5 ± 3.5	10.2 ± 3.6	0.825
Septal e', cm/s	8.6 ± 1.4	8.5 ± 1.3	0.913
dT, ms	239.8 ± 40.1	234.1 ± 39.7	0.262
TAPSE, mm	23.4 ± 2.8	22.5 ± 2.7	0.638
LVEF, %	64.6 ± 4.9	63.8 ± 4.8	0.322
LVEDV, mL	94.8 ± 21.6	95.5 ± 22.1	0.643
LVESV, mL	35.7 ± 8.1	39.4 ± 7.9	0.218
LV-GLS, %	-21.7 ± 3.6	-14.5 ± 3.4	<0.001
LV-GCS, %	-24.3 ± 4.8	-15.2 ± 4.6	<0.001
LV-GAS, %	-35.3 ± 4.4	-32.1 ± 4.5	0.106
LV-GRS, %	44.6 ± 5.5	42.3 ± 5.9	0.479

LVSWT: Left ventricular septal wall thickness, PWT: Posterior wall thickness, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, LAVI: Left atrial volume index, dT: Deceleration time, TAPSE: Tricuspid annular plane systolic excursion, LVEF: Left ventricular ejection fraction, LVEDV: Left ventricular end-diastolic volume, LVESV: Left ventricular end-systolic volume, LV: Left ventricular, GLS: Global longitudinal strain, GCS: Global circumferential strain, GAS: Global area strain, GRS: Global radial strain.

Table 3. Correlations between BMI with 3D-STE parameters and fQRS-T angle

	BMI r	P
LV-GLS	0.718	<0.001
LV-GCS	0.653	<0.001
LV-GAS	0.221	0.314
LV-GRS	-0.224	0.143
fQRS-T angle	0.692	<0.001

Spearman's correlation coefficient, Hosmer and Lemeshow TesT p=0.209. BMI: Body mass index, STE: Speckle tracking echocardiography, fQRS-T: Frontal QRS-T, LV: Left ventricular, GLS: Global longitudinal strain, GCS: Global circumferential strain, GAS: Global area strain, GRS: Global radial strain.

Cardiac Imaging / Echocardiography

PP-025

Periaortic adipose tissue index: A new approach to the relationship between coronary stenosis severity/lesion complexity and periaortic adipose tissue

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Background and Aıim: Periaortic adipose tissue (PAT) is associated with atherosclerosis. The severity of coronary stenosis with PAT has not been evaluated with conventional coronary angiography (CAG). The aim of the study is to determine the relationship between PAT and coronary stenosis severity/complexity, and to evaluate it with the periaortic adipose tissue index (PATI), a new index derived from PAT. Patients who underwent CAG and thoracic computed tomography (CT) between January 2017 and January 2022 were included in the study.

Methods: PAT volume was calculated by evaluating CT images, and PATI was calculated by dividing the PAT volume by the circumference of the descending aorta. Patients were divided into two groups according to the presence of ≥50% stenosis on CAG. The correlation of PAT and PATI with the SYNTAX score was evaluated. In our study, 263 patients [mean age 64.5 (54/72), male 164 (62.4%)] were evaluated. Severe coronary artery disease (CAD) was observed in 181 patients (68.8%). PAT volume and PATI were significantly higher in patients with severe stenosis (p<0.001, for both). When PAT and PATI were evaluated alongside CAD risk factors, an independent association between PATI and severe CAD was discovered (ß=0.581, p=0.97; ß=0.968, p=0.006, respectively). No correlation was found between SYNTAX score and PAT and PATI (r: -0.026, p=0.73; r: -0.019, p=0.19, respectively).

Results: In our study, PAT and PATI were higher in patients with severe coronary stenosis, and there was an independent relationship between PATI and severe stenosis.

Conclusions: We found no relationship between PAT and PATI and the SYNTAX score.

Cardiovascular Nursing / Technician

PP-027

Importance of nurses in recognizing complications in transcatheter aortic valve implantation (TAVI) patients

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Background and Aim: Transcatheter aortic valve implantation (TAVI) is an alternative treatment modality for patients

with symptomatic advanced aortic stenosis. Early recognition of complications that occur within the first 24 hours after TAVI and prompt diagnosis and treatment save lives.

Methods: This study was conducted with the nurse's early recognition of complications in the post-procedure period of the patient who underwent TAVI between 20.04.2023 and 22.06.2023 in Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Hospital Intensive Care Unit and service. It is a retrospective study that includes file scanning for rapid diagnosis and evaluation. The data obtained were evaluated in line with the determined nursing diagnoses and within the scope of the literature.

Results: In the post-procedure period, the patients' stay in the intensive care unit and service was monitored. In this process, a total of 15 nurses diagnosis has been made. Major nursing diagnoses; risk of infection, risk of bleeding, decreased cardiac output, lack of self-care, ineffective respiratory pattern, acute pain, acute confusion, nutritional imbalance-undernutrition, activity intolerance, lack of knowledge, fear, constipation. It was determined as disturbance in sleep pattern, risk of trauma, risk of falling, risk of deterioration of skin integrity, and nursing interventions were evaluated in this direction.

Conclusions: Post-procedure follow-up and treatment process of TAVI patients is very important. During their shifts in the wards, nurses are alone with the patients. Nurses have a great responsibility here. It should not be forgotten that it is vital for patients to have knowledge, skills and experience regarding special complications.

Coronary Artery Disease / Acute Coronary Syndrome

PP-029

The effect of platelet functions on short-term mortality and morbidity in ST-elevation myocardial infarction

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Background and Aim: Despite the transfer of the treatment process to pre-hospital settings, shortening of intervention times, and implementation of recommended treatment options, ST-elevation myocardial infarction (STEMI) remains the leading cause of in hospital death. In our study, the contribution of the reperfusion strategy, clopidogrel loading doses, and adjunctive therapy on platelet aggregation to mortality and morbidity was investigated.

Methods: Between April 2015 and July 2016, 58 patients aged 18-80 years admitted to our clinic with a diagnosis of STEMI were included in the study. Peripheral venous blood samples were collected at 2nd, 4th, and 24th hours after antiplatelet therapy to evaluate platelet functions using the "multiplate" device. Patients were divided into two groups based on residual platelet aggregation levels in the 4th-hour blood samples: those with aggregation level ≥47 U were designated as group A, and those with <47 U were designated as group B. The contribution of high platelet reactivity and the treatment strategy used to clinical outcomes was evaluated.

Results: Our study revealed that patients with high residual platelet aggregation at the 4th hour (group A) received fibrinolytic therapy, underwent a 300 mg clopidogrel loading dose, and used morphine due to persistent pain (p=0.003, p=0.036, p<0.001). It was found that in hospital events increased 6.637-fold in those who received fibrinolysis compared to those who did not (OR: 6.637, 95% CI: 1.391-31.658; p=0.018). When examining the effectiveness of morphine alone on platelet aggregation, it was observed that it was significantly associated with high residual platelet aggregation at 2nd and 4th hours, while this effect decreased at 24th hour (p<0.001, p=0.070). The use of morphine was found to be associated with morbidity at one month (p=0.030), but no direct relationship was found with an increase in morbidity. No significant difference was observed in clinical outcomes related to high platelet reactivity.

Conclusions: Based on the findings, the inadequacy of medical reperfusion strategy and delayed effect of clopidogrel with morphine use were detected. Although this delay did not have clinical significance, it led to a significant increase in hospital admissions with chest pain and the need for repeat revascularization during three-month follow-up. Long-term follow-up results are required.

Table 1. General clinical characteristics of the patients

Male*	45 (77.6)
Age, years	58.07 ±11.39
Body mass index, kg/m^2	27.85 ± 5.32
Diabetes mellitus*	12 (20)
Hypertension*	21 (35)
Current smoking*	46 (76.4)
Hyperlipidemia*	7 (11.7)
Family history of coronary artery disease*	22 (36.7)
Coronary artery disease*	2 (3.3)
Chronic obstructive pulmonary disease*	1 (1.7)
Chronic renal failure (GFR >35)*	2 (3.3)
Coronary artery bypass grafting*	0
Peripheral artery disease*	0

* n (%), other data are given as mean ± SD.

Table 2. Effect of antiaggregant loading doses and reperfusion therapy on platelet aggregation

		Group A (≥47 U) (n=28)	Group B (<47 U) (n=30)	p
Clopidogrel	300 mg	15 (65.2)	8 (34.8)	0.036
	600 mg	13 (37.1)	22 (62.9)	
Fibrinolytics		15 (75)	5 (25)	0.003
Primary PCI		13 (35.1)	24 (64.9)	0.008

Coronary Artery Disease / Acute Coronary Syndrome

PP-030

Clinical significance of monocyte chemoattractant protein-1 and CC chemokine receptor type 2 gene polymorphisms in young patients with acute coronary syndrome

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Background and Aim: Acute coronary syndrome (ACS) is the primary reason of death global. There is a special concern in identifying risk factors and underlying causes of ACS in young population. Genetic factors have currently become a popular study topic in terms of disease prevention and early diagnosis. In this study, we aimed to investigate the clinical importance of monocyte chemoattractant protein-1 (MCP-1)(A-2518G) and C-C chemokine receptor type 2 (CCR2) (G190A) gene polymorphisms in patients with young ACS.

Methods: This study was planned as a cross-sectional case-control study. 63 with YACS (<40 years), and normal coronary arteries have 103 patients were included in this study. However, the use of a similar control group against YACS may adversely affect the results. Since it cannot be guaranteed that people in the control group will not have myocardial infarction in the future, the control group consisting of people who have reached the age limit (over 40 years in our study) were included 63 patients with young ACS (<40 years), and 103 patients who have normal coronary arteries were included in this study (Table 1). MCP-1(A-2518G) and CCR2(G190A) gene polymorphisms were measured in all patients. All patients underwent coronary angiography via femoral or radial access using the standard Judkins technique.

Results: Baseline demographical characteristics of study population were presented in Table 1. Significant difference existed between the groups for age (p=0.001). Laboratory parameters of study groups were showed in Table 1. hsCRP, ESR and HDL (p<0.001, p=0.003 and p=0.001, respectively) were significantly different between the two groups. Genotype and allele distribution of MCP-1(A-2518G) and CCR2(G190A) polymorphism in the study groups were showed in Tables 2, 3. MCP-1 gene polymorphism compared with two groups were significantly different. However, CCR2 gene polymorphism was not statistically different. The relationship between MCP-1(A-2518G) gene GG (12.7% vs. 3.7%, respectively), AG (33.3% vs. 50.5%, respectively), AA (54% vs. 45.8%, respectively) genotypes is significant in YACS compared to the control group. Significant difference was found (p=0.021). However, no correlation was found between allele frequencies (Table 2). It was determined that MCP-1 GG genotype was significantly higher in YACS than the control group and increased the risk of disease. But there was no important difference among the CCR2(G190A) gene AA (85.7% vs. 85.0%, respectively), AG (14.3% vs. 13.1%, respectively), AA (0% vs. 1.9%, respectively) genotypes when compared with the control group (p=0.543). In addition, no correlation was found between allele frequencies between the two groups (Table 3).

Conclusions: In this study, it was determined that the MCP-1(A-2518G) GG genotype was high in the young patient with ACS and was associated with coronary artery risk.

Table 1. Baseline laboratory and demographical characteristics of study population

Variables	YACS group n=63	Control group n=107	P
Age, years	38.1 ± 3.4	51.1 ± 7.9	0.001
Male gender, %	38 (60)	66 (62)	0.860
BMI, kg/m^2	27.4 ± 2.3	26.9 ± 2.8	0.382
Diabetes mellitus, %	14 (22)	16 (15)	0.230
Hypertension, %	18 (29)	20 (19)	0.135
Smoking	17 (27)	21 (20)	0.266
SBP,mmHg	128.2 ± 11. 9	126.9 ± 12.5	0.524
DBP,mmHg	75.5 ± 7.3	75.8 ± 8.1	0.804
Total cholesterol, mg/dl	169.2 ± 53.2	169.4 ± 37.1	0.971
Low density cholesterol, mg/dl	102.78 ± 32.4	101.7 ± 30.1	0.828
High density cholesterol, mg/dl	34 (30-40)	40 (34-52)	0.001
Triglyceride, mg/dl	168 (103-219)	148 (102-209)	0.198
Glucose, mg/dl	93 (90-101)	95 (90-100)	0.937
Urea, mg/dl	29.9 ± 10.1	30.8 ± 10.6	0.474
Creatinine, mg/dl	0.86 ± 0.2	0.84 ± 0.2	0.445
Sodium, mmol/l	139.9 ± 4.0	139.1 ± 3.7	0.174
Potassium, mmol/l	4.3 ± 0.42	4.2 ± 0.44	0.854
Hemoglobin, gr/dl	13.6 ± 1.9	13.5 ± 1.7	0.731
White blood count,10^3/µl	10.8 ± 3.4	10.6 ± 2.9	0.999
Platelet, 10^3/µl	251.9 ± 56.8	244.0 ± 57.7	0.386
ESR, mm/hour	27(11-42)	13(6-27)	0.003
hsCRP, mg/dl	6.7(2.5-10)	2.9(0.8-7.0)	<0.001
Ejection fraction, %	53.5 ± 5.0	57.1 ± 6.9	<0.001

Abbreviations: ESR: Erythrocyte sedimentation Rate, BMI: body mass index; hsCRP:high sensitive C-reactive protein, DBP: diastolic blood pressure; SBP: systolic blood pressure.

Table 2. Genotype and allele distribution of CCR2 (G190A) polymorphism in the study population

CCR2		MI n=63	Control Group n=107	P
Gen	GG (normal)	54 (85.7)	91 (85)	0.543
	AG (hetero)	9 (14.3)	14 (13.1)	
	AA (homo)	0(0)	2 (1.9)	
Allel	A	117 (92.9)	196 (91.6)	0.676
	G	9 (7.1)	18 (8.4)	

Table 3. Genotype and allele distribution of MCP-1 (A-2518G polymorphism in the study population

MCP-1		MI n=63	Control Group n=107	P
Genotip	AA (normal)	34 (54)	49 (45.8)	0.021
	AG (hetero)	21(33.3)	54 (50.5)	
	GG (homo)	8 (12.7)	4 (3.7)	
Allel	A	89(70.6)	152 (71)	0.939
	G	37(29.4)	62(29)	

Coronary Artery Disease / Acute Coronary Syndrome

PP-031

Unraveling the prognostic potential of the castelli risk index for coronary artery ectasia

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Background and Aim: Coronary artery ectasia (CAE) is a vascular condition characterized by localized or diffuse

enlargement of one or more coronary arteries. The castelli risk index (CRI) is a cardiac risk assessment tool used to predict the risk of developing coronary artery disease (CAD). These ratios are thought to be better predictors of CAD risk than individual lipid levels alone. The relationship between coronary artery ectasia (CAE) and atherosclerosis has been a topic of considerable interest and investigation in the field of cardiovascular research. Therefore, in this study, we aimed to investigate the relationship between CAE and the CRI, a known predictor of atherosclerosis.

Methods: The study included a control group with normal coronary arteries and patients with confirmed CAE without any invasive interventions. Patients with ST-elevation myocardial infarction were excluded from the study. The CRI values were calculated for each group. CRI is determined by the assessment of two ratios: the total cholesterol (TC) to high-density lipoprotein (HDL) cholesterol ratio (TC/HDL-C) and the low-density lipoprotein (LDL) cholesterol to HDL cholesterol ratio (LDL-C/HDL-C).

Results: A total of 112 patients were included in the study, comprising 56 individuals in the control group with normal coronary arteries and 56 in the CAE group. In the CAE group, the mean platelet level, mean cholesterol level, median triglyceride level, median CRI-I level, and median creatinine level were found to be higher, whereas the mean HDL level was lower (Table 1). However, no significant association was observed between CRI-II and the presence of CAE. Increased platelet levels (OR: 1.04, 95% CI: 1.02-1.07; p=0.008), elevated CRI-I levels (OR: 1.40, 95% CI: 1.13-1.70; p=0.015), and higher creatinine levels (OR: 1.08, 95% CI: 1.01-1.17; p=0.038) were identified as independent predictors for CAE. The predictive threshold value for CRI-I in CAE detection was determined to be >4.5, with a sensitivity of 74.2% and specificity of 67.8% (Figure 1).

Conclusions: Our study provides valuable insights into the relationship between CAE and the CRI as a predictor of atherosclerosis. Moreover, our findings reveal that increased platelet levels, elevated CRI-I values, and higher creatinine levels are independent predictors for CAE. The CRI-I threshold value of >4.5 demonstrated promising sensitivity and specificity in predicting CAE presence. These results underscore the importance of CRI as a valuable tool for assessing the risk of CAE and contribute to a better understanding of the pathophysiological mechanisms underlying this vascular condition. Further studies and clinical validations are warranted to corroborate these findings and explore the clinical implications for patient management and risk stratification in coronary artery ectasia.

Table 1. Characteristic findings of the research cohort

Variables	Control (n=56)	CAE (n=56)	p
Age, years	61.1 ± 7.6	60.5 ± 9.6	0.697
Gender, n (%)			
Female	34 (60.7)	16 (28.6)	
Male	22 (39.3)	40 (71.4)	0.318
Drugs, n (%)			
ACEi	47 (83.9)	49 (87.5)	0.788
Loop diuretics	28 (50.0)	31 (55.4)	0.675
CCB	11 (19.6)	17 (30.4)	0.275
Laboratuar findings			
Hemoglobin, g/dL	13.7 ± 1.2	14.2 ± 1.9	0.100
WBC, (count, x10^3)	8.2 ± 1.8	8.2 ± 2.2	0.958
Platelets, (count, x10^3)	258.7 ± 67.4	294.1 ± 72.3	0.001*
Cholesterol, mg/dL	183.8 ± 44.2	198.9 ± 50.5	0.008*
HDL, mg/dL	45.4 ± 7.6	40.6 ± 9.7	0.004*
LDL, mg/dL	122 (97-144.5)	110.5 (87-135)	0.414
Triglyceride, mg/dL	146.5 (109.5-168)	162.5 (126.5-189.5)	0.001*
CRI-I	4.0 (3.6-5.2)	4.6 (4.0-6.2)	0.006*
CRI-II	2.6 (2-3.3)	2.7 (2.1-3.7)	0.225
Glucose, mg/dL	94.6 ± 14.6	97.4 ± 12.4	0.283
Sedimentation, mm/hour	5.5 (4-10)	4.1 (3-12)	0.157
C-reactive protein	4.2 (2.8-6.9)	4.7 (1.3-8.7)	0.670
Creatinine, mg/dL	0.8 (0.7-0.9)	0.9 (0.8-1.0)	0.012*
Uric acid, mg/dL	5 (4.2-5.5)	5.7 (4.7-6.7)	0.111
GFR, mL/min/1.73 m^2	89.4 ± 14.7	82.5 ± 24	0.067

Data are mean ± standard deviation or median (IQR), or number (%). *p<0.05 indicates statistical significance. Abbreviations: CAE, coronary artery ectasia; WBC, white blood cell; HG, hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride; GFR, glomerular filtration rate; ASA, acetylsalicyclic acid; BB, beta blocker; CRI-I, Castelli Risk Index I; CRI-II, Castelli Risk Index II.

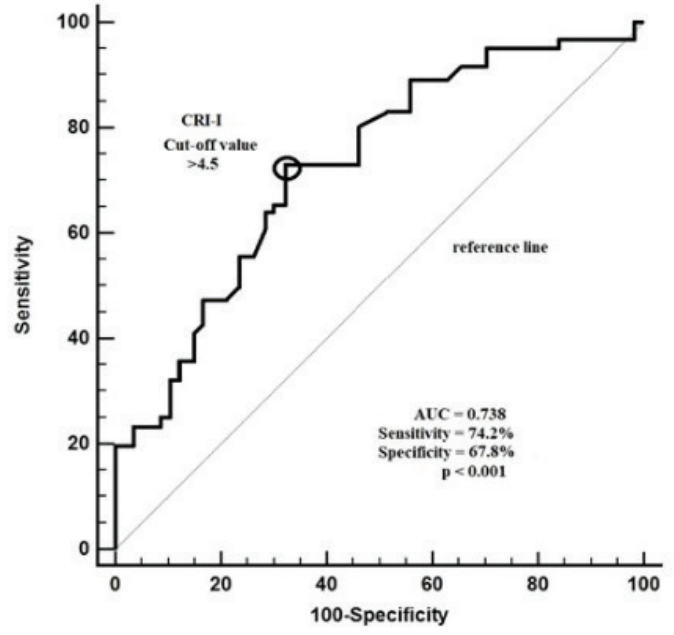

Figure 1. The diagnostic performance of CRI-I in predicting CAE.

Coronary Artery Disease / Acute Coronary Syndrome

PP-032

Left dominant coronary circulation is associated with poorer left ventricular function but not long term mortality after ST-elevation myocardial infarction

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Background and Aim: To evaluate the effect of coronary dominance on the left ventricular systolic function in patients with first ST-elevational myocardial infarction (STEMI), and to evaluate relationship between coronary dominance (CD) and long-term mortality. There is considerable variation between individuals in respect to CD.

Methods: We included 471 patients with first STEMI. The patients were categorized as right and left dominance according to their CD pattern. The left ventricular wall motion score index (WMSI) and left ventricular ejection fraction (LVEF) were used to evaluate the extent of left ventricular systolic dysfunction. The Cox regression analysis was used to assess the relationship between CD and long-term mortality.

Results: Left CD was present in 41 (8.7%) of the 471 patients. WMSI was significantly higher in the left dominant group than of the right dominant group (1.74 ± 0.38 vs. 1.56 ± 0.35, p=0.002). The frequency of LVEF <40% was significantly higher in patients with left CD group compared to right CD group (39% vs. 15.8%, p<0.001). The patients with left CD had higher peak CK and CK-MB levels (3269 ± 2988 U/L vs. 2355 ± 1511 U/L, p=0.007; 390 ± 303 U/L vs. 241 ± 172 U/L, p<0.001, respectively). Nevertheless, mortality was similar between the left and right dominance groups (13 (%40.1) vs. 85 (%30.7), p=0.201). In cox-regression analysis, CD was not related to long-term mortality.

Conclusions: Patients with left dominance had significantly lower left ventricular systolic function early after STEMI. However, long-term mortality was similar in patients left and right dominant circulation.

Table 1. Demographic and clinical characteristics of the study population

	Right Dominant Group	Left Dominant Group	p
Patients n (%)	430 (91.3)	41 (8.7)	
Age (Years) n(%)	56.2±11.0	59.2±11	**0.095**
Gender (Male) n (%)	357 (83)	36 (87.8)	**0.431**
Family History n (%)	103 (24.0)	7 (17.1)	**0.302**
Smoker n(%)	278 (64.7)	23 (56.1)	**0.276**
Hypertension n(%)	131 (30.5)	16 (39)	**0.258**
Diabetes n (%)	78 (18.1)	6 (14.6)	**0.575**
Total Cholesterol (mg/dl)	192±42	182±43	**0.142**
Creatinine (mg/dl)	1.07±0.50	1.04±0.22	**0.723**
Primary PCI or Trombolytic therapy n(%)	339 (78.8)	29 (70.7)	**0.230**
Time from symptom onset (minutes)			
To hospital admission	149(75-257)	195(90-330)	**0.108**
To reperfusion therapy	165(110-270)	210(130-250)	**0.382**
Time to anjiography (days) (median, 25th – 75th percentile)	1 (0-3)	0 (0-4)	**0.449**
Time to echocardiography (days) (median, 25th – 75th percentile)	**2 (1-3)**	**2 (1-3)**	**0.876**

Values are expressed as mean ± SD or numbers of patients (percent value). IHD: ischemic heart disease, PCI: percutaneous coronary intervention, TT: thrombolytic therapy, CK: creatine kinase)

Table 2. Angiographic findings

	Right Dominant Group n(%)	Left Dominant Group n(%)
	430 (91.3)	**41 (8.7)**
Vessel disease		
1	257 (59.8)	**23 (56.1)**
2	117 (27.2)	**15 (36.6)**
3	56 (13)	**3 (7,3)**
IRA		
LAD	219 (50.9)	**31 (75.6)***
RCA	175 (40.7)	**2 (4.9)**
Cx	36 (8.4)	**8 (19.5)**
TIMI 2/3 flow	361 (84)	**34 (82.9)**
Location of culprit lesion		
Proximal	112 (26)	**10 (24.4)**
Distal	318 (74)	**31 (75.6)**
Presence of collaterals to IRA	**58 (13.5)**	**3 (7.3)**

Values are expressed as numbers of patients (percent value). CAD: coronary artery disease IRA: infarct related artery, LAD: left anterior descending, RCA: right coronary artery, Cx: circumflex
* p<0.001

Table 3. Echocardiographic left ventricular systolic indexes and peak CK/CKMB values

	Right Dominant Group	Left Dominant Group
WMSI	1.56±0.35	**1.74±0.38***
LVEF, %	49±9.3	**45.1±11.1***
LVEF <40%	68 (15.8)	**16 (39)****
Pik CK (U/L)	2355±1511	**3269±2988***
Pik CKMB (U/L)	**241±172**	**390±303****

Values are expressed as mean ± SD or numbers of patients (percent). WMSI: wall motion score index, LVEF: left ventricular ejection fraction, CK: creatine kinase, CKMB: creatine kinase-MB.
*p<0.05, ** p<0.001

Table 4. Cox regression analysis for the very long term mortality

	Exp(B)	95,0% CI for Exp(B)		
		Lower	Upper	P
Age	1,055	1,034	1,076	**<,001**
Gender (Male)	0,655	,386	1,113	,118
Diabetes Mellitus	1,051	,590	1,874	,866
Hypertension	,959	,612	1,501	,854
Coronary Dominance(Left)	1,536	,795	1,342	,201
Receiving Emergent Reperfusion	,848	,535	1,118	,481
IRA				,425
RCA vs LAD	1,183	,754	1,857	,465
CX vs LAD	,676	,295	1,546	,353
EF<40%	1,800	1,069	3,031	**,027**

CX: circumflex artery, EF: ejection fraction, IRA: infarct related artery, LAD: left anterior descending artery, RCA: right coronary artery

Coronary Artery Disease / Acute Coronary Syndrome

PP-033

Bendopnea is a strong predictor of the presence and severity of the coronary artery disease in patients without known coronary artery disease and with normal left ventricular systolic function

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Background and Aim: Bendopnea as a relatively novel type of dyspnea, has been first described in patients with heart failure (HF) and is associated with an increased left ventricular end-diastolic pressure (LVEDP) in these patients. Studies have been suggested that HF patients with bendopnea have a worse clinical outcomes including advanced symptoms, rehospitalization, more severe disease and even death. However, there is no study investigating the association between bendopnea and coronary artery disease (CAD), yet. The aim of this study was to evaluate the relationship bendopnea and CAD in stable patients without known CAD and with normal left ventricular systolic function.

Methods: A total of 240 patients who underwent coronary angiography were included in the study. Patients were divided into two groups: with bendopnea and without bendopnea. Clinical and angiographic characteristics of two groups were compared. Logistic regression analysis was used to determine predictors of presence and severity of CAD in present study population.

Results: CAD was significantly more common in patients with bendopnea than those without bendopnea (56.8% vs. 3.3%, p<0.001). The patients with bendopnea had a higher rates of two-vessels disease (30.4% vs. 11.5%), three-vessels disease (33.7% vs. 4.7%), and left main disease (%9.8 vs. 0.7%) compared to those without bendopnea. LVEDP and Gensini score were found markedly higher in patients with bendopnea compared to those without bendopnea [13.3 ± 2.8 vs. 6.6 ± 2.2, p<0.001 and 48 (22-77) vs. 2 (0-14), p<0.001; respectively]. In multivariable analysis, male gender (p=0.005, OR: 2.804, 95% CI: 1.368-5.746), diabetes (p=0.041, OR: 2.214, 95% CI: 1.035-4.738), bendopnea (p<0.001, OR: 23.344, 95% CI: 6.792-80.227), LVEDP (p=0.029, OR: 1.147, 95% CI: 1.021-1.309), and smoking (p=0.028, OR: 3.229, 95% CI: 1.131-9.215) were independent predictors of the CAD. Additionally, these predictors other than male gender, were found to be associated with multivessel CAD.

Conclusions: Bendopnea can be easily detected during physical examination in patients with suspected CAD and it may help to clinicians in the diagnostic work-up of CAD.

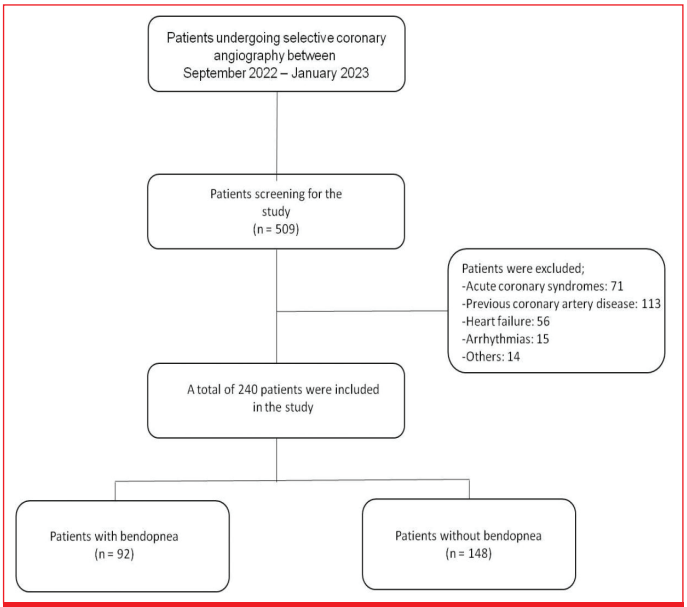

Figure 1. The flow chart diagram of the study patients enrollment.

Coronary Artery Disease / Acute Coronary Syndrome

PP-034

Demographic and anamnestic predictors for coronary artery disease in patients with ST-T alterations

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Background and Aim: Electrocardiography (ECG) is an inevitable inquiry in patients with suspected coronary artery disease (CAD). Its role is particularly established for diagnosis of acute coronary artery disease, whereas for chronic artery disease, although it is recommended as initial investigation, its findings are not specific. The main ECG alterations in this setting are ventricular repolarization abnormalities, mainly presented as ST segment depression and/or T wave alterations. The specificity of ST-T changes in conventional ECG for the diagnosis of coronary heart disease in patients undergoing coronary angiography is shown to be 33.7% and the sensitivity 66.0%. The aim of this research was to identify demographic and history data that can predict the occurrence of CAD in patients with ST-T changes who were undergoing elective coronary angiography (ECA).

Methods: This was a prospective cross-sectional study that included 84 consecutive patients with ST-T changes in ECG that underwent ECA in our tertiary medical center. Patients with horizontal or descending ST depression ≥0.5 mm at the J point in ≥2 contiguous leads and/or T wave alterations were included in the study. Exclusion criteria included patients with previously known CAD, severe anaemia, <18 years of age, atrial fibrillation and poor echocardiographic window. The criterion for detecting substantial coronary artery disease through coronary angiography was established as a stenosis of over 70% in the coronary arteries and/or greater than 50% in the case of the left main coronary artery.

Results: Forty eight (57.14%) patients resulted without significant CAD forming the first group, while 36 (42.86%) patients had significant CAD forming the second group. Older patients, males, smokers and those with typical chest pain showed statistical significance between the groups (Table 1). Simple regression analysis of the number of affected vessels in relation to demographic and history data demonstrated the following: age: r=0.34, p=0.001, male gender: r=0.57, p<0.00001, smoking: r=0.41, p=0.0001, typical chest pain: r=0.26, p=0.02. Multiple regression analysis showed that all these parameters were significant predictors, male gender (p<0.00001) being the strongest.

Conclusions: Relying solely on ST-T changes in an ECG is inadequate for confirming the existence of chronic CAD. Nonetheless, when ECG ventricular repolarization irregularities are coupled with factors such as older age, male gender, smoking, and typical chest pain, they become indicative of the presence and extent of chronic CAD. Notably, among these factors, male gender emerges as the most influential predictor.

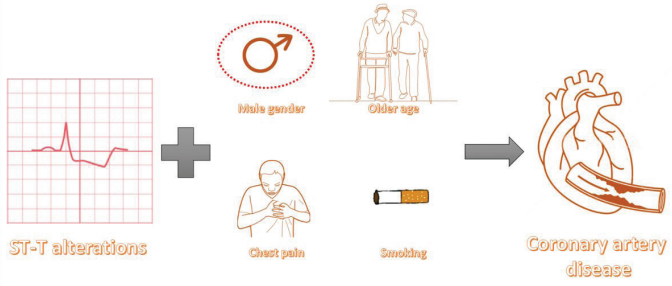

Figure 1. Diagram of the predictors for CAD in patients with ST-T alterations.

When ECG ventricular repolarization irregularities are coupled with factors such as older age, male gender, smoking, and typical chest pain, they become indicative of the presence and extent of chronic CAD.

Table 1. Comparison of demographic data between the two groups

	No CAD (n=48)	CAD (n=36)	p
Age, years	58.54 ± 7.31	68.86 ± 8.95	<0.0001
Gender (males), %	9/48 (18.7)	26/32 (72.2)	<0.0001
Hypertension, %	38/48 (79.17)	28/36 (77.78)	0.88
Diabetes mellitus, %	9/48 (18.75)	12/36 (33.3)	0.13
Dyslipidemia, %	16/48 (33.3)	12/36 (33.3)	1.0
BMI, kg/m^2	28.85 ± 5.03	26.8 ± 4.58	0.06
Family history, %	17/48 (35.42)	15/36 (41.67)	0.56
CCS grading, average	1.06 ± 0.73	1.78 ± 0.74	0.08
Typical chest pain, %	18/48 (37.5)	26/36 (72.2)	0.002
Smokers, %	7/48 (14.58)	15/36 (41.67)	0.005

CAD: Coronary artery disease, BMI: Body mass index, CCS: Canadian Cardiovascular Society grading of angina pectoris.

Table 2. Multiple regression analysis of the affected coronary artery vessels in relation to demographic and history data

	Coefficient	Standard error	t	p
Age	0.03	0.02	2.53	0.007
Male gender	0.52	0.1	5.39	<0.00001
Smoking	0.34	0.11	3.18	0.001
Typical chest pain	0.18	0.09	1.9	0.03

Standard error of estimate 0.8; coefficient of determination (R-squared): 0.48; adjusted for degrees of freedom: 0.45

Coronary Artery Disease / Acute Coronary Syndrome

PP-036

The role of new inflammatory markers in determining the development of contrast-induced nephropathy in patients undergoing percutaneous coronary angiography due to STEMI

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Background and Aim: Contrast induced nephropathy is an important complication that develops after angiographic procedures. It is an important morbidity and mortality problem that causes patients to stay in hospital for a long time. This study aims to investigate the role of pre-procedural new immune-inflammation markers in predicting CIN development in patients undergoing percutaneous coronary intervention (PCI) due to ST segment elevation myocardial infarction (STEMI).

Methods: A total of 417 STEMI patients were included in the study. CIN was defined as an increase of at least 0.5 mg/dL or 25% in serum baseline creatinine level 72 hours after the procedure. Patients were divided into two groups: those with and without CIN. Baseline characteristics, angiographic features, laboratory data, pan-immune-inflammation, systemic immune inflammation index (SII) and systemic response index were compared between the two groups.

Results: STEMI patients who developed CIN, glucose level (p<0.01), platelet count (p=0.017), neutrophil count (p=0.017), white blood cell (WBC) count (p<0.001), monocyte count (p<0.001), neutrophil-to-lymphocyte ratio (NLR) (p<0.001), systemic immune inflammation index (SII) score (p<0.001), SRI (p<0.001), PIV (p<0.001) were higher compared to those without CIN. The receiver operating characteristic curve analysis (ROC) showed a cut off value of 468.6 with a sensitivity of 83.3% and a specificity of 72% in patients with PIV CIN (AUC: 0.83). SRI >2.19 had 83% specificity and 77% sensitivity in detecting CIN (AUC: 0.834). SII >838.9 had 81% specificity and 75% sensitivity in detecting CIN (AUC: 0.82). NLR >838.9 had 72% specificity and 81% sensitivity in detecting CIN (AUC: 0.81).

Conclusions: Our study shows that pre-procedural PIV and SRI values are a strong predictor among new inflammatory markers in determining the development of contrast-induced nephropathy in STEMI patients.

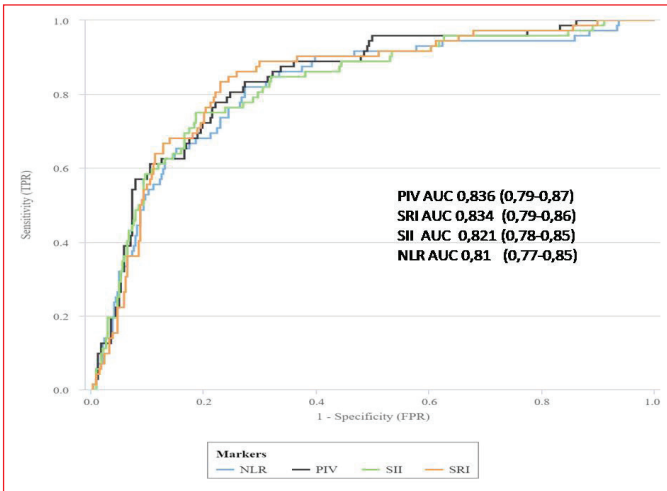

Figure 1. ROC analysis.
Effect of pan-immune inflammation value, systemic immune inflammation index, systemic response index and neutrophil/lymphocyte ratio values on contrast induced nephropathy.

Table 1. Demographic characteristics of the study populations

	CIN (+)	CIN (-)	p
Variables	(n=72)	(n=345)	
Age, years	66.8 ± 10.6	64.5 ± 9.9	0.07
Gender (male), n (%)	44 (61.1)	205 (59.6)	0.69
Hypertension, n (%)	40 (54.8)	187 (54.4)	0.94
Diabetes mellitus, n (%)	47 (64.4)	172 (50)	0.02
Current smoker, n (%)	52 (71.2)	210 (61)	0.1
Hypercholesterolemia, n (%)	20 (27.4)	103 (29.9)	0.6
Left ventricular ejection fraction, %	54.2 ± 5.2	54.79 ± 5.1	0.4
Systolic blood pressure, mmHg	126.9 ± 6.4	127.1 ± 6.4	0.75
Diastolic blood pressure, mmHg	82.2 ± 6.5	81.6 ± 6.5	0.4
Heart rate, beats/min	76.3 ± 7.7	78.2 ± 7.8	0.07

Table 2. Laboratory findings of the study populations

Laboratory findings	CIN (+) (n=72)	CIN (-) (n=345)	p
Glucose, mg/dL	148.5 (116.2-225.7)	124.9 (102-190)	<0.001
Creatinine, mg/dL	1.03 (0.75-1.3)	0.84 (0.71-0.96)	<0.001
Glomerular filtration rate, mL/min/1.73 m^2	77.1 (48-98.4)	90.4 (75-100)	0.001
AST, U/L	27 (20-30)	24 (19-34)	0.22
ALT, U/L	20 (15.2-28)	18 (13-26)	0.3
Total cholesterol, mg/dL	164.4 ± 35.8	166.6 ± 45.2	0.8
LDL-C, mg/dL	121 (100.8-144.8)	120 (100-147)	0.92
Triglycerides, mg/dL	140 (129-160)	132 (91.7-189)	0.273
CRP, mg/dL	8 (5-10.2)	5.6 (2.5-11.4)	0.075
Hemoglobin, mg/dL	12.8 ± 2.1	13.2 ± 2	0.145
Platelet count, 10^3/µL	250 (205.5-289.5)	225 (188-271.75)	0.017
WBC count, 10^3/µL	11.2 (9.5-13.5)	8.5 (6.8-9.9)	<0.001
Neutrophil count, 10^3/µL	12.68 (7.03-21.9)	4.8 (3.8-8.4)	0.017
Lymphocyte count, 10^3/µL	1.65 (1-2)	1.89 (1.6-2.1)	0.381
Monocyte, 10^3/µL	0.75 (0.6-0.96)	0.62 (0.5-0.76)	<0.001
NLR	5.5 (3.3-7.9)	2.1 (1.5-3.2)	<0.001
SII	1309 (791.9-2001.4)	487 (349.6-729)	<0.001
SRI	4.17 (2.4-6.02)	1.36 (0.92-2.06)	<0.001
PIV	1110 (528.7-1390.8)	313.7 (199.6-493.8)	<0.001

ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, CIN: Contrast-induced nephropathy, HDL-C: High-density lipoprotein cholesterol, HsCRP: High sensitivity C-reactive protein, LDL-C: Low-density lipoprotein cholesterol, NLR: Neutrophil/lymphocyte ratio, SII: Systemic immune-inflammation index, SRI: Systemic response index, PIV: Pan-immune-inflammation, WBC: White blood cell.

Table 3. Angiographic data

	CIN (+) (n=72)	CIN (-) (n=345)	p
Total amount of contrast, mL	239.7 ± 37.3	232.6 ± 31.2	0.1
Total time of procedure, min	30 ± 5.06	28.7 ± 5.4	0.09
Culprit coronary vessel, n (%)			
Left anterior descending coronary artery	37 (51.4)	178 (52)	0.9
Left circumflex coronary artery	29 (40.3)	148 (43.3)	0.64
Right coronary artery	35 (48.6)	172 (50.3)	0.7
Left main artery	6 (8.3)	13 (8.3)	0.09
Number of diseased coronary arteries, n (%)			
One-vessel disease	29 (40.3)	173 (50.6)	0.11
Two-vessel disease	18 (25)	67 (19.6)	0.44
Three-vessel disease	4 (5.6)	7 (2)	0.09

Coronary Artery Disease / Acute Coronary Syndrome

PP-037

Missed opportunities in patients with acute myocardial infarction

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Background and Aim: Myocardial infarction is a non-reversible myocardial injury and necrosis that occurs primarily as a complication of coronary artery disease, resulting from severe and prolonged ischemia. Missed opportunities refer to the failure to identify a disease or prevent its occurrence. In this context, when a patient at risk of myocardial infarction is not asked about their condition during a visit, a missed opportunity occurs. According to the literature review, nearly half of the patients who have experienced myocardial infarction have encountered missed opportunities. The aim of our study is to identify missed opportunities in patients who have experienced acute myocardial infarction within the past two years at a public hospital's cardiology clinic. We intend to evaluate whether patients were under doctor's control before their first heart attack in terms of missed opportunities.

Methods: This cross-sectional study was conducted between January 2023 and April 2023 at a public hospital's cardiology outpatient clinic. A face-to-face survey consisting of 25 questions was administered to patients who had experienced myocardial infarction. By considering the responses to the survey questions, a significant relationship between the

age of patients at their first heart attack and other answers was sought using the SPSS program.

Results: Of the 74 participants included in the study, 16.2% were female (n=12) and 83.8% were male (n=62). The mean age of the participants was 60 (standard deviation: ± 9.09), and the mean age at first heart attack was 53 (standard deviation: ± 9.00). A significant association was found between the age at first heart attack and education level (p=0.040), treatment for high cholesterol (p<0.001), diabetes treatment (p=0.037), hypertension treatment (p=0.023), diagnosis of coronary artery disease (p=0.039), diagnosis of high cholesterol (p<0.001), diagnosis of diabetes (p=0.037), hypertension diagnosis (p=0.019), and frequency of smoking (pack-years) (p<0.001).

Conclusions: As expected, the study found that certain chronic diseases and smoking frequency have an impact on the age at first heart attack. It was observed that individuals receiving treatment for hypertension, high cholesterol, and diabetes experienced their first heart attack at a later age. The limited sample size due to logistical issues restricted the study, and obtaining a larger sample would yield more accurate results.

Table 1. Demographic variables of the study group

Cinsiyet	Kadın	12	16,2
	Erkek	62	83,8
Meslek	şoför	12	16,2
	emekli	22	29,70
	serbest meslek	6	8,1
	ev hanımı	10	13,5
	işçi	11	14,9
	öğretmen	2	2,7
	çiftçi	3	4,1
	esnaf	1	1,4
	aşçı	3	4,1
	bahçıvan	1	1,4
	memur	1	1,4
	elektrik teknisyeni	1	1,4
	mali müşavir	1	1,4
Eğitim durumu	okuryazar değil	3	4,1
	ilkokul mezunu	41	55,4
	ortaokul mezunu	16	21,6
	lise mezunu	9	12,2
	üniversite mezunu	5	6,8
	yüksek lisans/doktora	0	0

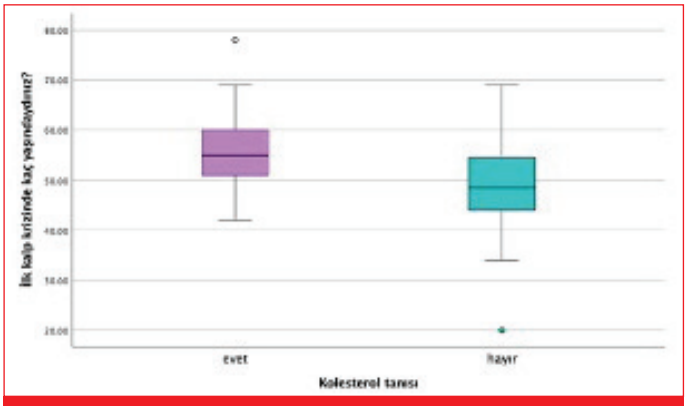

Figure 1. Diagnosis of hypercholesterolemia and age on first MI.

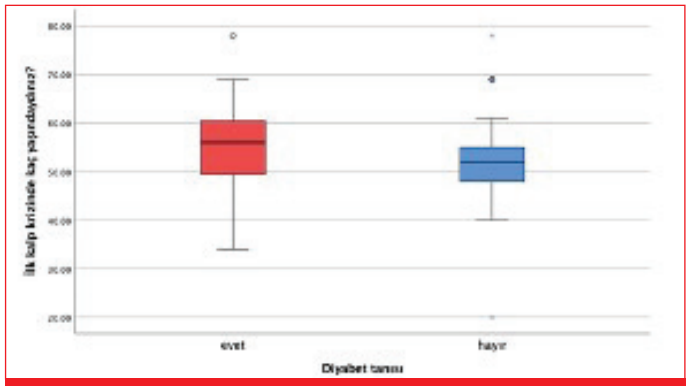

Figure 2. Diagnosis of diabetes and age on first MI.

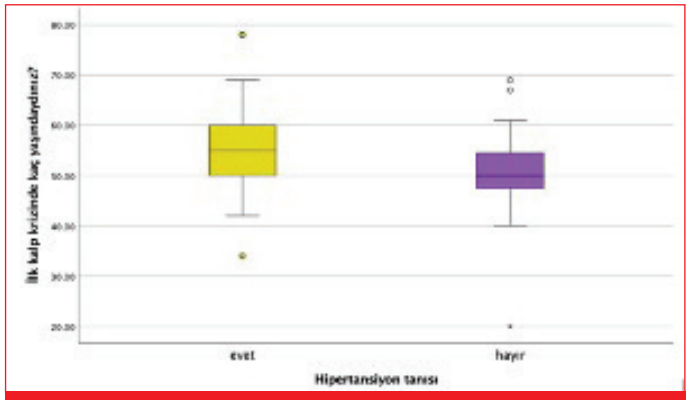

Figure 3. Diagnosis of hypertension and age on first MI.

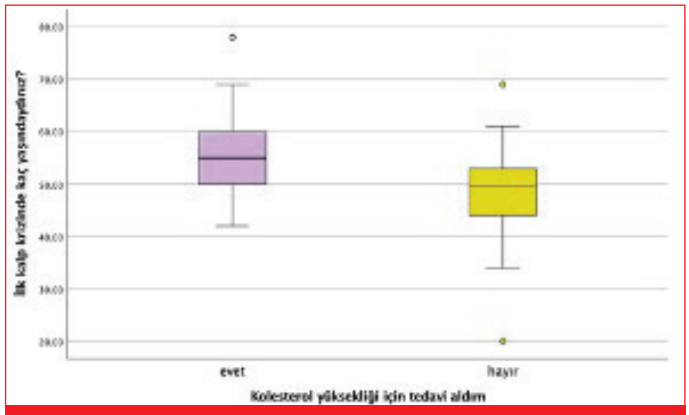

Figure 4. Treatment of hypercholesterolemia and age on first MI.

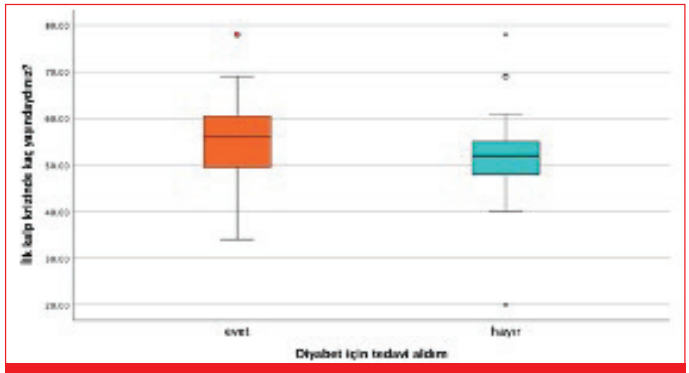

Figure 5. Treatment of diabetes and age on first MI.

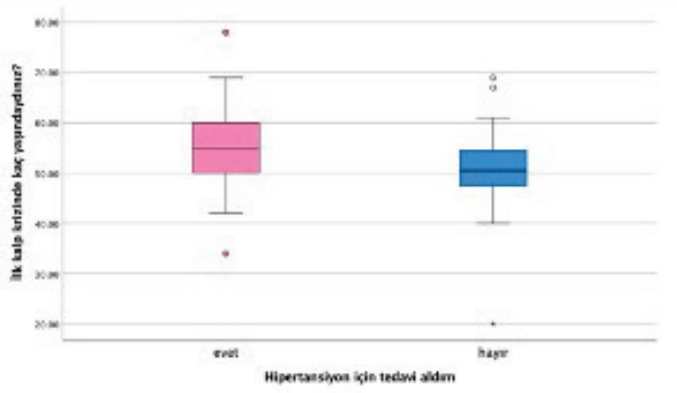

Figure 6. Treatment of hypertension and age on first MI.

Coronary Artery Disease / Acute Coronary Syndrome

PP-038

Could morphine have a negative impact on the anti-platelet effect of clopidogrel in patients with STEMI? A single center pilot study reinforcing an old hypothesis

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Background and Aim: Morphine is still in use for pain relief in acute coronary settings. However, several studies have suggested a possible negative impact of morphine on the effect of antiplatelet agents. Our aim was to evaluate the effect of morphine on antiplatelet effect of clopidogrel and in-hospital mortality and morbidity in patients with ST-segment elevation myocardial infarction (STEMI).

Methods: This study was derived from a prospective observational study to evaluate the impact of platelet functions on mortality and morbidity in patients with STEMI. Platelet reactivity (PR) was assessed at 2-, 4-, and 24-hour blood samples. Patients were divided into two groups as morphine recipients and non-recipients. In-hospital, short-term (30 and 90 days), and long-term (12-month) outcomes were obtained.

Results: A total of 58 consecutive patients [mean age 58 ± 11 years; 77.6% male] with a mean follow-up of 10.2 months were evaluated. Morphine group consisted of significantly more females (p=0.019) and patients with higher platelet count (p=0.015). The use of morphine has significantly increased the PR (p<0.001). The proportions of patients with a platelet aggregation of ≥47 U at the 2nd and 4th hours were significantly higher in the morphine group than the non-morphine group, indicating an increased PR (p<0.001) (Table 1). There was a significant association between morphine administration and morbidity at the first month (p=0.030). 3rd month follow-up revealed increased rates of recurrent admissions, hospitalizations, and revascularizations with insufficient platelet inhibition, and recurrent chest pain however there were no significant differences at the 12th month evaluation.

Conclusions: Our single-center, prospective, observational study, showed a significantly increased PR at the 4th hour, which resolved at 24 hours of clopidogrel treatment, in STEMI patients receiving morphine compared with those not receiving morphine. Furthermore, morbidity increased in patients receiving morphine, both during the hospital stay and in the first month. All these results confirm an old hypothesis that needs further evidence.

Table 1. Evaluation of the effect of morphine use on platelet aggregation

		Patients received morphine (n=28)	Patients received no morphine (n=30)	p
Pain duration at the time of admission	<1 hour 1-3 hours >3 hours	5 (33.3) 10 (62.5) 13 (48.1)	10 (66.7) 6 (37.5) 14 (51.9)	0.267
Killip classification	<2 ≥2	23 (45.1) 5 (71.4)	23 (45.1) 5 (71.4)	0.246
Clopidogrel	300 mg 600 mg	13 (56.5) 15 (42.9)	10 (43.5) 20 (57.1)	0.308
Primary percutaneous coronary intervention		14 (37.8)	23 (62.2)	0.035
Fibrinolytic therapy		14 (70)	6 (30)	0.016
Platelet aggregation				
2nd hours	<47 U ≥47 U	3 (10.7) 25 (89.3)	17 (56.7) 13 (43.3)	<0.001
4th hours	<47 U ≥47 U	6 (21.4) 22 (78.6)	24 (80) 6 (20)	<0.001
24th hours	<47 U ≥47 U	20 (74.1) 7 (25.9)	28 (93.3) 2 (6.7)	0.070

Table 2. In hospital risk stratification and evaluation of follow-up prognosis between groups

	Patients received morphine (n=28)	Patients received no morphine (n=30)	p
TIMI score %, median (IQR)	4.4 (1.9, 9.85)	2.2 (1.6, 16)	0.556
GRACE score			
In hospital mortality, median (IQR)	2.4 (1.45, 4.65)	2.7 (1.1, 4.6)	0.761
6-month mortality, median (IQR)	5.3 (2.6, 10.35)	4.4 (1.6, 7.9)	0.369
In hospital events, n (%)	10 (37)	11 (37.9)	0.945
1st month morbidity, n (%)	9 (34.6)	3 (10.3)	0.030
3rd month morbidity, n (%)	5 (19.2)	2 (7.4)	0.250

Grace: Global registry of acute coronary events, IQR: Interquartile range, TIMI: The thrombolysis in myocardial infarction.

Lipid / Preventive Cardiology

PP-040

Beyond the systolic: Unveiling diastolic blood pressure characteristics in patients undergoing cardiovascular risk assessment using SCORE-2

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Background and Aim: It is important to note that cardiovascular risk assessment is a complex process, and different risk assessment tools and guidelines may have varying approaches to incorporating blood pressure measurements into their calculations. The SCORE-2 (Systematic COronary Risk Evaluation) system is just one of many cardiovascular risk assessment tools available, and it does not include diastolic blood pressure (DBP) as a separate variable in its risk assessment calculations. Instead, it primarily focuses on systolic blood pressure (SBP), which is considered a stronger predictor of cardiovascular risk. This omission might be since SBP has been found to be a stronger predictor of cardiovascular risk compared to DBP.

Methods: Patients aged 40-69 years without a history of diabetes or cardiovascular disease were included in the study. The parameters involved in the use of the SCORE-2 scale were evaluated. Turkey's risk group is high, and the C scale was used. The ambulatory blood pressure monitoring (ABPM) was recorded automatically. DBP parameters in 24-hour AMBP measurements were evaluated. We defined standard deviation (SD) and coefficient of variation (CV) of DBP values as an indicator for blood pressure variability.

Results: We analyzed 99 adult patients (44 male, 44.4%). Since none of the patients were in the high-risk group, the patients enrolled into this study were divided into 2 groups as low and medium risk according to their SCORE-2 risk scores. As expected, there were differences between the groups in terms of SCORE-2 parameters. While no difference was observed between the groups in terms of age, total cholesterol, BMI and eGFR, interestingly, a statistically significant difference was observed between the two groups in terms of serum uric acid levels. According to the measurements of 24-h ABPM, 24-hour, day-time and night-time DBP measurements were statistically higher in the medium-risk group. However, no statistically significant difference was observed between the two groups in terms of DBP variability indices with daytime and nighttime separately.

Conclusions: The DBP might be used to provide information about the overall cardiovascular health of a person's cardiovascular system. Persistent elevated DBP can damage the walls of arteries and weaken the heart muscle over time. So, elevated DBP indicates increased resistance in the arteries, often due to stiffening or narrowing of blood vessels. This can lead to increased workload on the heart and potentially causing damage to blood vessels. Therefore, managing diastolic blood pressure can be crucial in reducing cardiovascular risk and promoting heart health.

Table 1. Basal demographical, clinical and laboratory data.

	Low risk (n=65)	Moderate risk (n=34)	P value
SCORE2 risk score, (%)	2.86 ± 1.31	5.12 ± 1.87	< 0.001
Male sex, n (%)	18 (27.7)	26 (76.5)	< 0.001
Current smoker, n (%)	55 (84.6)	18 (52.9)	0.001
SBP, mmHg	120.93 ± 12.96	132.52 ± 12.51	<0.001
Age, years	53.46 ± 7.21	51.47 ± 8.85	0.232
BMI, kg/m^2	28.58 ± 6.00	30.38 ± 4.82	0.133
Total cholesterol, mg/dL	214.20 ± 46.33	220.02 ± 61.48	0.597
HDL cholesterol, mg/dL	53.66 ± 13.19	41.47 ± 10.68	<0.001
eGFR, ml/min	83.50 ± 17.28	91.04 ± 20.24	0.057
Serum uric acid, mg/dL	5.19 ± 1.24	5.89 ± 1.29	0.018

SBP: systolic blood pressure; eGFR: estimated glomerular filtration rate; BMI: body mass index; HDL: high density lipoprotein.

Table 2. Measurements of 24-h ABPM and DBP indices with 24-hour, day-time and night-time separately

	Low risk (n=65)	Moderate risk (n=34)	P value
Recording time, (hours)	23.13 ± 0.42	23.06 ± 0.38	0.423
24-hour,			
DBP, (mmHg)	75.53 ± 9.37	80.91 ± 11.15	0.013
Mean SD of DBP, (mmHg)	12.60 ± 4.09	12.67 ± 4.64	0.938
CV of DBP, (%)	16.85 ± 5.57	15.66 ± 5.16	0.302
Day-time			
DBP, (mmHg)	77.27 ± 9.86	82.26 ± 11.58	0.027
Mean SD of DBP, (mmHg)	13.87 ± 14.83	12.45 ± 4.86	0.590
CV of DBP, (%)	18.27 ± 19.87	15.17 ± 5.41	0.375
Night-time			
DBP, (mmHg)	69.47 ± 9.06	76.23 ± 11.09	0.002
Mean SD of DBP, (mmHg)	14.02 ± 33.36	11.27 ± 4.60	0.634
CV of DBP, (%)	20.41 ± 48.99	14.82 ± 5.64	0.510

SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; PP: pulse pressure; SD: standard deviation; CV: coefficient variation.

Lipid / Preventive Cardiology

PP-041

Assessment of the risk status and knowledge level of cardiovascular diseases in individuals applying to the cardiology outpatient clinic during the pandemic period

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Background and Aim: Cardiovascular diseases (CVDs) are the leading cause of death globally, and also in Türkiye. It is very important to determine risk situations to take early precautions about cardiovascular disease risk factors, and the level of awareness. The COVID-19 pandemic has deeply affected individuals and social life; as a result of this impact, negative changes in well-being, disruption of routine medical appointments and controls may affect the risk status and management of cardiovascular diseases. The aim of this study was to determine the cardiovascular diseases risk status, knowledge level, well-being in the last 2 weeks, to evaluate how they manage their routine medical controls and to investigate their relationship with each other, who applied to the cardiology outpatient clinic during the pandemic period.

Methods: Our study was cross-sectional design. The questionnaire consisted of sociodemographic characteristics, hospital admission status during the pandemic period, the CARRF-KL Scale for knowledge of cardiovascular disease risk factors, the SCORE-2 System for determining risk status, and the WHO-5 Well-being Index for determining well-being. Data analysis was done using Mann-Whitney U test, Kruskal-Wallis test, chi-square test, Spearman correlation analysis. A $p<0.05$ was considered significant in all analyses except post hoc analyses.

Results: According to the SCORE-2 system, 43.30% of the individuals were in the low-moderate risk group, 46.10% in the high-risk group and 10.60% in the very high-risk group; the mean score from the CARRF-KL scale was 19.75 ± 4.45 and their knowledge level was slightly above the medium level; the mean score of the WHO-5 Well-Being Index was 12.02 ± 5.01 and the rate of below the threshold was 50.10%. It was observed that 36.50% of the participants postponed their medical appointments due to the pandemic. No significant statistical difference and relationship was found between SCORE-2 cardiovascular risk status and "CARRF-KL scores" and "WHO-5 well-being status" (p=0.480; rho=-0.050; p=0.686). A significant difference was found between CARRF-KL scores and WHO-5 well-being status (p=0.042).

Conclusions: It was observed that the majority of the individuals participating in the study were in the high and very high-risk group, their level of knowledge was slightly above the medium level, and more than half of them had well-being below the threshold value. For the prevention and early diagnosis of cardiovascular diseases, it is very important to increase the level of knowledge in the community and to identify risk situations. Effective information, education and screening activities on these issues should be implemented.

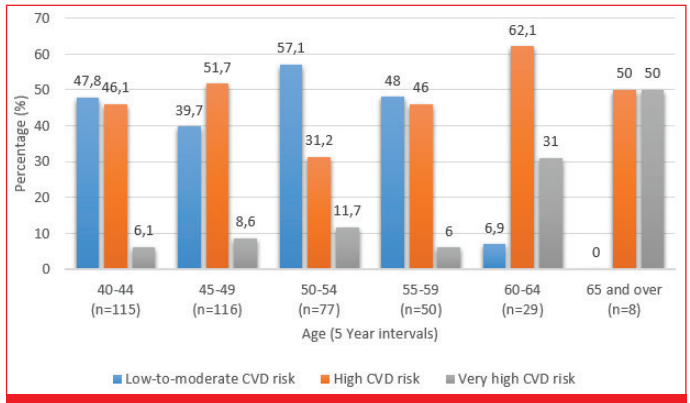

Figure 1. Distribution of 10-year cardiovascular event risk states according to age groups calculated with the SCORE-2 system for high-risk countries.

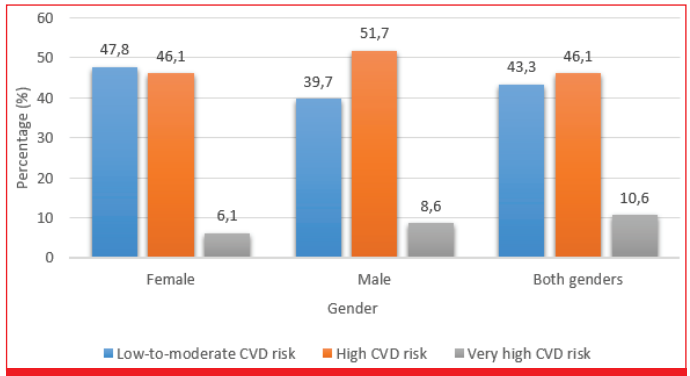

Figure 2. Distribution of 10-year cardiovascular event risks calculated by SCORE-2 system for high-risk countries by gender.

Table 1. Descriptive statistics of participants' WHO-5 index, CARRF-KL scale and SCORE-2 scores

Scale	Mean - SD	Median (Min-Max)
WHO-5	12.02 ± 5.01	12 (0-25)
CARRF-KL	19.75 ± 4.45	20 (1-28)
SCORE2	4.77 ± 3.69	3.80 (0.40-30.60)

Table 2. Comparison of SCORE-2 risk status with CARRIF-BD scores and WHO-5 well-being status

SCORE2 Categories	CARRF-KL Score Median (min – max)	p *	WHO-5 Well-Being Category		p **
			Bad	Good	
Low-Medium Risk	20 (5-28)	0.480	82 (48.00%)	89 (52.00%)	0.686
High risk	20 (1-27)		93 (51.10%)	89 (48.90%)	
Very High Risk	19 (8-27)		23 (54.80%)	19 (45.20%)	

* Kruskal Wallis test , **Chi-Square test, row percentage is given.

Table 3. Comparison of WHO-5 well-being status and CARRF-KL scores

WHO-5 Well-Being Categories	CARRF-KL Score Median (**IQR 25-75)	P*
Good	20 (18-24)	0.042
Bad	20 (17-23)	

*Mann Whitney U test **Interquartile range

Nuclear Cardiology

PP-042

The correlation of reverse redistribution pattern with coronary angiography: A conundrum revisited

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Background and Aim: The reverse redistribution pattern (RR), defined as the new appearance or worsening of an existing defect in resting images compared to stress images, is a phenomenon whose etiology, pathophysiology, and clinical implications are not well understood, but which has continued to be observed since its first report in 1979. Since the studies examining the possible correlation between RR and coronary angiography (CAG) date back to days when dual antiaggregant and anti-ischemic therapies were not widely used as they are today, we aimed to reinvestigate any relationship between RR and CAG findings in today's contemporary clinical settings.

Methods: All patients with a RR pattern on Tc99-MIBI scan done for myocardial ischemia detection between 2021-2023 (first 6 months) years were retrospectively screened. Demographic information, history of previous acute coronary syndrome (ACS; STEMI, NSTEMI, unidentified ACS), revascularization, comorbidities and risk factors (diabetes, hypertension, dyslipidemia, and smoking) were collected. After a primary analysis to identify population characteristics and the ventricular regions where the RR pattern was most common, a secondary analysis was performed to investigate any correlation between RR and the location of coronary stenoses. The physician's decision in the case of the RR pattern was grouped as follows: medical-only treatment, CAG, coronary CT angiography, and cardiac PET scan.

Results: The primary study cohort was comprised of 67 patients (men 73%, aged 63.6 ± 10.5, Table 1). RR pattern was most commonly seen in the inferior-posterior wall (n=41, 31.3%), followed by the apex (n=19, 14.5%) and anterior (n=12, 9.2%). In 40% of the patients (n=27) had RR pattern in more than one region. About half of the study population had a history of some form of revascularization and 1 in 3 had experienced ACS. According to the results of the secondary analysis done on patients with an available coronary angiography, most patients with RR pattern had normal/non-obstructive coronary angiograms (61.1%, n=22); significant stenoses in 1, 2, and 3 vessels were present in 19.4% (n=7), 13.8% (n=5), and 5.5% (n=2) of patients, respectively (Figure 1). There was no correlation between the regions of the RR pattern and significant stenosis detected on coronary angiography (p=0.6, p=0.5, p=0.6, respectively for LAD, CX, and RCA). There was also no difference in terms of gender, history of ACS or revascularization, and comorbidities between those with and without significant stenosis on coronary angiography.

Conclusions: In this study, which aimed to examine the relationship between RR patterns and coronary angiographic findings in the current era of widespread use of antiaggregant and anti-ischemic therapy, no evidence of such a link was found. The second major finding was that 60% of the patients with RR pattern had normal/non-obstructive coronaries, so the decision to proceed with coronary angiography should not be made based solely on this finding.

Table 1. Baseline characteristics

Variable	Total (n=67)
Demographics	
Age (years)	63.6 ±10.5
Female	12 (17%)
Clinical history	
Acute coronary syndrome	22 (32.8%)
STEMI	15
NSTEMI	1
Unidentified ACS	6
Revascularization history	28 (41.7%)
Stent-only	16
CABG-only	10
CABG plus stent	2
Imaging parameters	
Echo LVEF (%) [Mdn, (IQR)]	55 (20)
MPI Stress LVEF (%) [Mdn, (IQR)]	47 (18)
MPI Rest LVEF (%) [Mdn, (IQR)]	51.1 ±11.1
Comorbidities and risk factors	
Hypertension	44 (88%)
Diabetes	35 (77.8%)
Dyslipidemia	44 (86.3%)
Smoking	13 (86.7%)

ACS = acute coronary syndrome, CABG = coronary artery bypass grafting, echo = echocardiography, IQR = interquartile range, LVEF = left ventricular ejection fraction, MPI = myocardial perfusion imaging, Mdn = median, NSTEMI = non-st elevation myocardial infarction, STEMI = st elevation myocardial infarction.

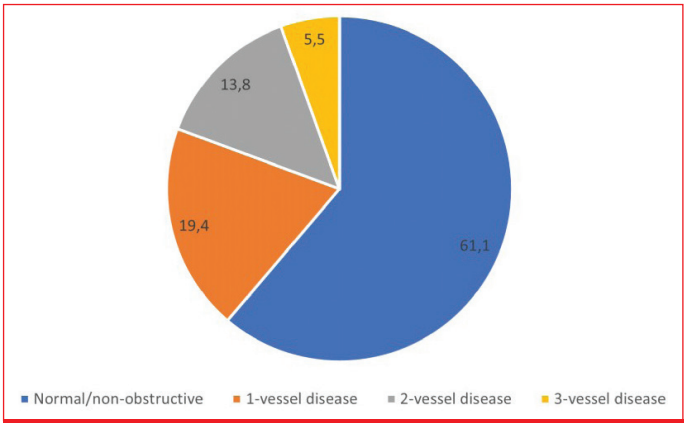

Figure 1. Coronary angiography results (percent).

Pulmonary Hypertension / Pulmonary Vascular Disease

PP-043

Long-term natural course of patients with pulmonary artery pressures in the range of 21-24 mmHg: Insights from a single center study

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Background and Aim: Slightly elevated mean pulmonary artery pressure (mPAP) with right heart catheterization (RHC) was previously termed "borderline PH". Although different mPAP cutoff values have been obtained in studies, its prognostic importance is a matter of debate. However, the long-term natural history of borderline PH is not well addressed. We examined the long-term prognosis of patients with mPAP values in the upper limit of 21 to 24 mmHg, who were referred with a suspicion of PH. METHODS:This retrospective cohort study included patients with moderate-to-high echocardiographic risk who underwent RHC from 2008 to 2021 and met at least 1 year follow-up criteria. Patients were divided into two groups according to the mPAP values in the RHC reports, and a comparison was made between patients with mPAP values of 20 mmHg and below (within normal limits) and those with mPAP values in the range of 21-24 mmHg. The analysis included demographic characteristics, clinical characteristics, and prognoses of the patient groups. Overall mortality was defined as the rate of all-cause mortality in the study population over a mean follow-up of 5 years (minimum 1 year - maximum 13 years).

Results: The study cohort included a total of 140 patients (35.4%) with mPAP values <25 mmHg measured at a total of 395 diagnostic RHCs. Mean age was 53.1 ± 14.8 years, mean follow-up was 4.92 ± 3.13 (min 1-max 13) years, and the majority of patients were women (n=102; 74.5%). Co-morbidity rates were similar between the groups. Among the prognostic clinical markers, NT-pro-BNP and 6-minute walking distance (6MWD) were better in the group with mPAP <21 mmHg. Echocardiographic findings suggestive of PH were more common in the mPAP 21-24 mmHg group [RA area >18 cm^2, 72.7%, (p=0.008), right ventricular ejection fraction >50%, 53.4% (p=0.006)]. Among the clinical prognostic indicators determined by RHC pulmonary artery wedge pressure (PAWP) was significantly higher in mPAP <21 mmHg group (10.97 ± 3.73-8.22 ± 2.76, p=0.0001) and cardiac index (CI) was significantly lower in mPAP <21 mmHg group (3.07 ± 0.74, p=0.001). All-cause mortality rates tended to be higher in the borderline PH group but did not reach to statistical significance during the follow-up. Overall, 23 patients (22.5%) in the mPAP <21 mmHg group and 13 (37.1%) in the mPAP 21-24 mmHg group were died (p=0.063).

Conclusions: In our study, we observed that even mPAP 21-24 mmHg was associated with a worser prognosis than a lower normal range. Although it was performed in a single center and with a limited number of patients, the length of the follow-up period of up to 13 years revealed the natural course patients with a mPAP of 21-24 mmHg range previously called borderline PH. Larger multicenter clinical trials with long term follow-up are warranted to clarify the effect of early treatment options in these high-risk patients.

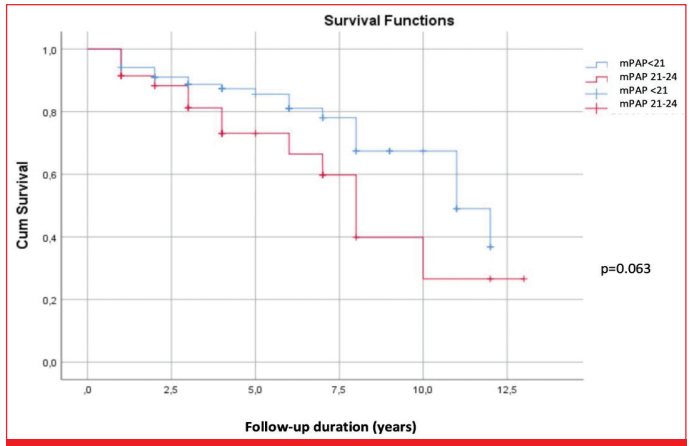

Figure 1. Comparison of survival between groups during follow-up.

mPAP <21 mmHg: Mean 9.571 ± 0.45 (95% CI: 8.68-10.45). Median 11 ± 1.37 (95% CI: 8.30-13.69) mPAP 21-24 mmHg: Mean 7.92 ± 0.92 (95% CI: 6.16-9.69). Median 8.00 ± 0.68 (95% CI: 6.66-9.33)

Table 1. Demographic and clinical characteristics by mPAP Categorization

Characteristics	mPAP <21 mmHg (n:102)	mPAP 21-24 mmHg (n:35)	p
Age (years) mean±SD	51.5 ±15.1	57.97 ± 12.8	0.026
Gender n (%)			
Female	72 (70.6)	30 (87.5)	0.077
Male	30 (29.4)	5 (14.3)	
Follow-up duration, years, mean±SD	5.06± 3.13	4.57 ±3.13	0.429
BMI (kg/m²), mean±SD	25.95 ±4.50	27.84 ±5.48	0.069
Comorbidities n (%)			
Diabetes Mellitus	16 (15.7)	7 (20)	0.556
Hypertension	8 (7.8)	4 (11.4)	0.517
Coronary artery disease	7 (6.9)	0	
WHO-FC (%)			
Class 1	17 (16.7)	1 (2.9)	
Class 2	71 (69.6)	22 (62.9)	0.07 [a]
Class 3	12 (11.8)	12 (34.3)	
Class 4	2 (2)	0	
6MWD m; mean±SD	361±100	319±88	0.281
Laboratories			
NT-proBNP ng/L; mean±SD	201±95	379 ±75	0.37
Hemoglobin, g/dL; mean±SD	12.9±1.2	13+2.3	0.236
GFR, mL/min per 1.73 m^2 (<60) (n, %)	12 (12.4)	3 (9.1)	0.610
Baseline four-strata risk (n, %)			
Low	21 (32.3)	2 (6.7)	
Intermediate-low	25 (38.5)	10 (33.3)	0.05 [a]
Intermediate-high	11 (16.9)	14 (46.7)	
High	8 (12.3)	4 (13.3)	
Follow-up [b]			
Addition of PAH-specific treatment (n, %)	3 (2.9)	9 (27.7)	0.56
Survival, (n, %)			
Alive	79 (77.5)	22 (62.9)	0.063
Excitus	23 (22.5)	13 (37.1)	

AF: Atrial fibrillation, BMI: Body mass index, GFR: Glomerular filtration rate, HR: Heart rate, mPAP: Mean pulmonary artery pressure, NT-pro-BNP: N-terminal pro B-type natriuretic peptide, PH: Pulmonary hypertension, SR: Sinus rhythm, WHO-FC: World Health Organization functional class, 6MWD: 6-minute walking distance.

Table 2. Baseline hemodynamic and echocardiographic characteristics by mPAP categorization

Variable	mPAP <21 mmHg (n:102)	mPAP 21-24 mmHg (n:35)	p
Echocardiography			
LVEF (> %55), n (%)	90 (83.3)	26 (68.6)	**0.036** [a]
RVEF (>%50), n (%)	83 (80)	19 (53.4)	**0.006** [a]
RA area >18 cm^2 n (%)	44 (44.2)	24 (72.7)	**0.008**
TRV m/s mean±SD	3.27 ± 0.69	3.18 ± 0.48	0.484
Echocardiographic signs suggestive of PH, n (%)	42 (44.2)	21 (67.7)	**0.023**
Right heart catheterization			
RA Pressure, mmHg	7.14+3.65	7.71+4.35	0.48
PAWP, mmHg	8.22+2.76	10.97+3.73	**0.0001**
PVR, Woods units n (%)			0.858[a]
• ≤ 2	52 (50.9)	19 (54.2)	
• > 2	47 (46)	16 (45.7)	
Cardiac output, L/min mean±SD	5.36 ± 2.04	5.60 ± 1.41	0.483
Cardiac index, L/min/m^2 mean±SD	3.98 ± 1.15	3.07±0.74	**0.001**

LVEF: Left ventricular ejection fraction, mPAP: Mean pulmonary artery pressure, PAWP: Pulmonary arterial wedge pressure, PH: Pulmonary hypertension, PVR: Pulmonary vascular resistance, RA: Right atrial, RVEF: Right ventricular ejection fraction, TRV: Tricuspid regurgitation velocity.

Table 3. List of patients-initiated PAH specific therapy with the diagnosis of PAH during follow-up

Patient No	Age (years)	Sex	Baseline RHC; mPAP (mmHg)	Flow-up RHC* timing (years)	PAH etiology/group	PAH specific treatment	Survival outcome
1.	44	Male	18	2	CHD	Macitentan	Alive
2.	63	Female	19	2	PTE	Macitentan	Alive
3.	71	Female	18	7	SS	Bosentan	Excitus
4.	70	Female	24	2	PTE	Riociguate	Alive
5.	64	Female	24	1	PTE	Bosentan	Alive
6.	68	Female	22	3	PTE	Riociguate	Alive
7.	73	Female	22	5	SS	Iloprost	Alive
8.	69	Female	21	5	SS	Bosentan	Excitus
9.	73	Female	22	4	SS	Iloprost	Excitus
10.	75	Female	23	3	SS	Macitentan	Excitus
11.	53	Female	23	4	SS	Combined Macitentan & Tadalafil	Alive
12.	67	Female	21	3	SS	Combined Bosentan & Tadalafil	Alive

CHD: Congenital heart disease, mPAP: Mean pulmonary artery pressure, PAH: Pulmonary arterial hypertension, PTE: Pulmonary thromboembolism, RHC: Right heart catheterization, SS: Systemic sclerosis.

Pulmonary Hypertension / Pulmonary Vascular Disease

PP-044

Soluble ST2 in acute pulmonary embolism

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Background and Aim: Suppression of tumorigenicity receptor (ST2) is a member of the IL-1 superfamily. It consists of two components, ST2L, which is the membrane form, and sST2, which is soluble in blood. Soluble ST2 (sST2) is a cardiovascular injury-related biomarker. sST2 is also secreted response to mechanical tension from myocyte and has prognostic value for heart failure. However, biopsy studies have shown that the alveoli are the main source of serum sST2. The extent to which sST2 is elevated in acute pulmonary embolism (PE) and whether sST2 can discriminate between high-risk and low-risk patients are unknown.

Methods: Patients whose diagnosis was confirmed by computed tomography pulmonary angiography (CTPA) were included in the study. Except for PE, other diseases causing sST2 elevation were excluded. Echocardiographic pulmonary artery pressure, CBC, creatinine, CRP, sST2, and D-dimer tests were performed on the patients and the control group at the time of admission.

Results: After pre-assessment, 66 patients confirmed acute PE who met the study criteria and 62 patients for the control group were included. sST2 levels were positively correlated with the pulmonary embolism severity index (PESI). sST2 level was associated with increased mortality and prolonged hospitalization (p<0.05).

Conclusions: Among patients diagnosed with acute PE, sST2 showed superior overall prognostic performance and similar sensitivity over D-dimer. It is a candidate biomarker for predicting high-risk patients for acute PE. Unravelling the role of sST2 may open the door to understanding the acute effects of PE and its chronic complications such as pulmonary hypertension.

Table 1. Distribution of some descriptive features by study groups

	Acute PE (n=66)	Kontrol (n=62)	p
Age (year)	67.9±13.7 (24-93)	62.8±15.7 (22-90)	**<0.01[a]***
Gender			
Male	25 (37.9)	35 (56.5)	**0.035[c]***
Female	41 (62.1)	27 (43.5)	
Hipertansiyon	41 (62.1)	15 (24.2)	**<0.001[c]***
Diyabetes mellitus	11 (16.7)	4 (6.5)	0.073[c]
Sigara kullanımı	14 (21.2)	20 (32.3)	0.157[c]

Sürekli değişkenler "ortalama±standart sapma (minimum-maksimum)", kategorik değişkenler "sayı (sütun yüzdesi)" olarak sunulmuştur
[a]Mann-Whitney U Testi; [b]Student's T Testi; [c]Pearson Ki-Kare Testi; [d]Fisher'in Kesin Testi; *p<0.05

Table 2. Laboratory values according to study groups

	Acute PE (n=66)	Kontrol (n=62)	p
Creatinin	1.09±0.52 (0.39-3.61)	0.95±0.48 (0.58-4.00)	**0.022[a]***
ALT	25.3±27.5 (4.4-140.0)	27.2±19.5 (2.5-120.0)	**0.011[a]***
AST	27.3V20.2 (5.0-107.0)	24.8±10.7 (11.2-73.0)	0.288[a]
Haemoglobin	12.0±2.0 (7.3-17.1)	13.7±2.0 (8.1-17.3)	**<0.001[b]***
WBC	10.6±3.6 (4.0-19.5)	7.2±2.7 (4.0-17.0)	**<0.001[a]***
CRP	66.8±57.3 (0.6-185.0)	3.31±2.38 (0.6-14.9)	**<0.001[a]***
D-Dimer	4977.6±3826.2 (123-10000)	594.1±887.4 (92.9-4126.0)	**<0.001[a]***
sTT2	162.1±182.4 (37.1-704.7)	10.1±6.6 (1.0-21.6)	**<0.001[a]***

Değişkenler "ortalama±standart sapma (minimum-maksimum)" olarak sunulmuştur
[a]Mann-Whitney U Testi; [b]Student's T Testi; *p<0.05

Table 3. Transthoracic echocardiography

	PE (n=66)	Kontrol (n=62)	p
EF	56.6±6.0 (25-70)	57.4±4.4 (45-70)	0.854[a]
PAP	38.3±17.6 (15-120)	18.0±4.4 (10-30)	**<0.001[a]***
RV Diamater	30.1±5.5 (21-46)	24.0±3.7 (16-30)	**<0.001[a]***

Değişkenler "ortalama±standart sapma (minimum-maksimum)" olarak sunulmuştur
[a]Mann-Whitney U Testi; *p<0.05

Table 4. PESI and troponin value of study group

	PE (n=66)
Troponin	241.9±491.5 (0.1-2613.0)
PESI	88.7±35.4 (24-223)

Değişkenler "ortalama±standart sapma (minimum-maksimum)" olarak sunulmuştur
[a]Mann-Whitney U Testi; *p<0.05

Pulmonary Hypertension / Pulmonary Vascular Disease

PP-045

The relationship of systolic pulmonary artery pressure with perioperative mortality and morbidity in patients undergoing non-cardiac surgery: A single center experience

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Background and Aim: Pulmonary hypertension (PH) is associated with adverse perioperative events in patients undergoing non-cardiac surgery. In this study, we aimed to investigate the relationship between systolic pulmonary artery pressure (sPAP), evaluated by transthoracic echocardiography (TTE) before surgery, and perioperative mortality and morbidity in patients who underwent non-cardiac surgery in our center.

Methods: Of the 3425 retrospectively screened patients who underwent non-cardiac surgery, 3049 patients whose estimated sPAP values were previously determined by TTE were included in the study. Patients were classified into 3 groups according to their estimated sPAP levels. sPAP <35 mmHg formed group 1, 35-39 mmHg group 2, and ≥40 mmHg group 3. All demographic and perioperative data obtained from the database of our institute were compared in three groups.

Results: Of the 3049 patients enrolled in the study, 2406 (78.9%) were in group 1, 259 (8.5%) in group 2, and 384 (12.6%) in group 3. Thirty-day all-cause mortality was observed in 82 (2.7%) patients, cardiac mortality occurred in 9 patients (0.3%). In the group with sPAP ≥40 mmHg, cardiac mortality was 0.5% and all-cause mortality was 7.3%. Thirty-day all-cause mortality, acute pulmonary edema, and acute renal failure were significantly higher in group 3 than in the other groups. Cardiac mortality did not differ significantly between the groups.

Conclusions: In conclusion, increased sPAP is associated to adverse postoperative outcomes. Evaluation of sPAP with TTE before non-cardiac surgery in patients whose clinical features and examination findings suggest PH may contribute to the reduction of perioperative mortality and morbidity.

Table 1. Baseline demographic, clinical and echocardiographic characteristics of the study groups

Parameter	Group 1 (sPAP <35 mmHg) (n=2406)	Group 2 (sPAP =35-39 mmHg) (n=259)	Group 3 (sPAP ≥ 40 mmHg (n=384)	p value
Age	59.66±17.01	69.53±15.65	72.92±13.39	**<0.001**
Male, % (n)	50.5 (1214)	49.2 (128)	50.3 (193)	0.929
BMI (kg/m^2)	27.65±5.87	27.52±5.56	26.70±5.65	0.934
HT, % (n)	57.9 (1393)	77.7 (202)	75.8 (291)	**<0.001**
DM % (n)	24.5 (590)	32.3 (84)	28.1 (108)	**0.012**
Smoking, % (n)	30.6 (736)	27.7 (72)	27.6 (106)	0.346
CAD, % (n)	19.2 (461)	20.4 (53)	28.4 (109)	**<0.001**
MI history, % (n)	8.1 (195)	8.8 (23)	19.3 (74)	**<0.001**
CABG history, % (n)	6.9 (166)	6.9 (18)	10.7 (41)	**0.03**
CHF, % (n)	2.5 (59)	6.5 (17)	18.2 (70)	**<0.001**
AF, % (n)	5.1 (123)	16.2 (42)	36.7 (141)	**<0.001**
COPD, % (n)	5 (121)	8.1 (21)	13.3 (51)	**<0.001**
CKD, % (n)	14.1 (339)	21.9 (57)	23.7 (91)	**<0.001**
LV EF (%)	59.34±4.90	57.47±5.66	55.02±9.34	**<0.001**
sPAP (mmHg)	25.85±4.27	36.32±1.61	48.36±9.06	**<0.001**

Table 2. Comparison of groups according to operation risk

Operation risk	Group 1 (sPAP <35 mmHg) (n=2406)	Group 2 (sPAP =35-39 mmHg) (n=259)	Group 3 (sPAP ≥ 40 mmHg (n=384)	p value
Low, % (n)	30.3 (728)	30.4 (79)	26.6 (102)	0.559
Intermediate, % (n)	52.3 (1257)	53.8 (140)	54.2 (208)	
High, % (n)	17.5 (420)	15.8 (41)	19.3 (74)	

Table 3. Comparisons of postoperative adverse events ratios in study groups

Parameter	Group 1 (sPAP <35 mmHg) (n=2406)	Group 2 (sPAP =35-39 mmHg) (n=259)	Group 3 (sPAP ≥ 40 mmHg (n=384)	p value
Cardiovascular mortality, % (n)	0.2 (6)	0.4 (1)	0.5 (2)	0.636
All cause mortality, % (n)	1.9 (46)	3.1 (8)	7.3 (28)	**<0.001**[a]
Acute pulmonary edema, % (n)	0.2 (6)	0 (0)	3.9 (15)	**<0.001**[a]
Atrial fibrillation, % (n)	2.5 (61)	6.2 (16)	6.3 (24)	**<0.001**[b]
Myocardial infarction, % (n)	0.3 (8)	0.4 (1)	0.8 (3)	0.427
Acute renal failure, % (n)	0.7 (16)	1.2 (3)	2.3 (9)	**0.005**[a]
Cerebrovascular event, % (n)	0.2 (4)	0.8 (2)	1.8 (7)	**<0.001**[c]

sPAP, systolic pulmonary artery pressure.
[a] A significant difference was observed between group 3 and other groups, but there was no significant difference between group 1 and 2.
[b] Postoperative AF rate was significantly higher in Groups 3 and 2 compared to group 1. No significant difference was observed between group 2 and 3.
[c] There was a significant difference between group 1 and 3.

Table 4. Multivariable regression analysis for postoperative cardiovascular mortality

	Cardiovascular mortality		
	p value	OR	95% Confidence interval
Age	0.684	1.010	0.961-1.062
sPAP	0.629	0.984	0.921-1.051
LV EF	0.071	0.915	0.830-1.008
HT	0.730	0.743	0.138-4.005
DM	0.665	1.402	0.304-6.467
CAD	0.934	0.928	0.161-5.353
CHF	0.268	0.267	0.026-2.756
AF	0.309	0.427	0.083-2.200
CKD	0.860	0.867	0.176-4.259

Table 5. Multivariable regression analysis for postoperative all-cause mortality

	All-cause mortality		
	p value	OR	95% Confidence interval
Age	**0.005**	1.025	1.007-1.042
sPAP	**0.001**	1.037	1016-1.058
LV EF	0.528	0.986	0.945-1.029
HT	0.443	1.244	0.712-2.172
DM	0.544	0.853	0.511-1.424
CAD	0.118	1.534	0.897-2.623
CHF	0.579	0.741	0.257-2.137
AF	0.676	0.869	0.450-1.678
COPD	**0.005**	2.390	1.303-4.386
CKD	0.116	1.551	0.897-2.680

Pulmonary Hypertension / Pulmonary Vascular Disease

PP-046

Prognostic nutritional index: as a new predictor of mortality in pulmonary arterial hypertension patients

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Background and Aim: The relationship between prognostic nutritional index (PNI) and clinical outcomes as well as mortality in heart failure patients is well known. However, there is no data on the impact of nutritional status on mortality in newly diagnosed and treatment-naive patients with pulmonary arterial hypertension (PAH).

Methods: In this study, a total of 134 consecutive patients (mean age 63.9 ± 28.1) diagnosed with PAH based on right-left heart catheterization since 2013 were retrospectively analyzed, and 52 patients (42 females) who met the criteria were included in the study. The demographic characteristics, biochemical parameters, hematological parameters, echocardiographic parameters, catheterization parameters, 6-minute walk test, and PNI values at the time of initial diagnosis were evaluated. The data of 26 patients who died during the follow-up period and 26 patients who continued PAH-specific treatment were compared.

Results: The PNI value at the time of initial diagnosis was significantly lower in the group of patients who died (35.3 ± 5.4) compared to the group of patients who survived (40 ± 5.4) (p=0.003). According to univariate analysis, female gender, New York Heart Association (NYHA) class, fasting blood glucose, red cell distribution width (RDW), 6-minute walking test distance, albumin, hematocrit, hemoglobin, and PNI were significantly different. After elimination using a multivariate logistic regression model, gender, NYHA class, hemoglobin, fasting blood glucose, and PNI value were independent predictors of mortality in PAH. It was observed that a 1-unit increase in PNI value reduced the risk of developing mortality by 0.835 times. The PNI value was analyzed using receiver operating characteristics (ROC) curve in terms of mortality, and the probable cut-off value was determined to be 38.0 with a sensitivity of 65.4% and specificity of 61.5% (AUC: 0.714, p=0.008).

Conclusions: We first demonstrated that a PNI value above 38 is associated with lower mortality in PAH patients. PNI can be used as a predictor of mortality in newly diagnosed PAH patients undergoing specific treatment.

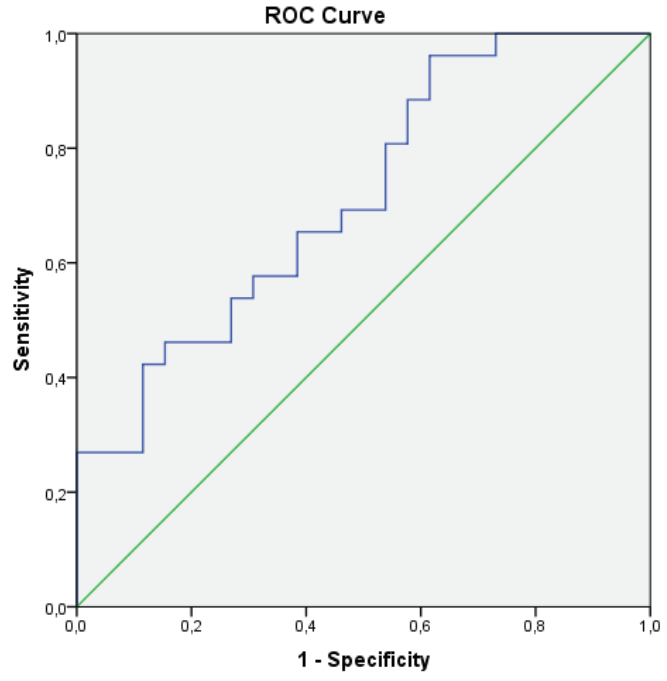

Figure 1. Receiver operating characteristics (ROC) curve.

Table 1. Multivariate logistic regression model

	OR	95% CI Lower Limit	95% CI Upper Limit	p
PNI	0.835	0.721	0.967	0.016
Gender	14.363	0.962	214.483	0.053
NYHA	25.393	1.548	416.467	0.023
Hg	0.976	0.893	1.066	0.588
Fasting blood glucose	1.015	0.999	1.033	0.070

Surname Name	Surname Name	Pub Number
Abacı, Adnan	Abacı, A.	PP-032
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