

40th NATIONAL CARDIOLOGY CONGRESS

POSTER PRESENTATIONS

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PP-001

The impact of predictors used in differentiation between paroxysmal supra ventricular tachycardia and ischemic heart disease in young patients with elevated high sensitive troponin

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Background and Aim: Cardiac troponin T (cTnT) is a highly sensitive and specific biomarker for the diagnosis of acute myocardial infarction (AMI). However, elevated cTnT is also seen in other cardiac and noncardiac conditions. This phenomenon potentially leading to mismanagement of PSVT cases. Therefore, it becomes crucial to delineate the kinetic effects of cTnT elevation. This understanding is imperative for ensuring an accurate clinical diagnosis and effectively differentiating PSVT-related troponin elevation from underlying ischemic conditions. The aim of this study is to determine the pattern of changes in hs-cTnT levels in patients under 50 years of age with low risk of cardiovascular disease PSVT patients, who had elevated hs-cTnT levels upon presentation at the emergency department.

Methods: A total of 139 patients, under the age of 50, with low cardiovascular disease risk and admitted to the emergency department with chest pain and cTnT elevation during follow-up were analyzed. We subsequently assessed the presence of IHD in patients exhibiting elevated cTnT levels, and compared the variations in cTnT levels between those with IHD and PSVT. The presence of IHD was evaluated by means of diagnostic coronary angiography (CA) or cardiac computed tomographic angiography (CTA) after biomarker sampling. Electrophysiological study (EPS) was performed in patients whose CA or CTA did not reveal significant coronary artery stenosis. Pre-procedural transthoracic echocardiography was performed on all patients

Results: In 99 out of the 139 patients included in the study, a coronary artery lesion responsible for infarction was detected on CA or CTA. Forty patients, who did not have a coronary artery lesion responsible for infarction, underwent EPS. Among these patients, atrioventricular nodal reentrant tachycardia was induced in 31, atrioventricular reentrant tachycardia in 5, and atrial tachycardia in the remaining 4 during EPS. In the patient group where PSVT, the coexistence of chest pain with palpitation symptoms was significantly more observed compared to the NSTEMI group (70% vs. 14.4%, $p < 0.001$). The

troponin value at admission and the peak troponin value were found to be significantly higher in the NSTEMI group compared to the PSVT group (30 pg/mL vs. 155.5 pg/mL, $p < 0.001$ and 100 pg/mL vs. 345 pg/mL, $p < 0.001$). The time to peak troponin, on the other hand, was earlier in the PSVT group compared to the NSTEMI group (6 h vs. 7 h, $p = 0.072$).

Conclusions: We found that despite a higher likelihood of elevated enzyme levels, the admission and maximum cTnT levels were higher in patients with IHD. However, the time to peak troponin was significantly earlier in patients with PSVT. Further studies with a larger population are necessary to elucidate the exact changes in cardiac enzyme. No significant difference was observed between the two groups in the analysis of echocardiographic data.

Table 1.

	SVT, n=40	NSTEMI, n=99	p value
Male, %	52.2	82.7	0.001
Age	44.3 ± 10.8	44.55 ± 5.1	0.133
HT %	47.5	22.4	0.007
DM %	17.5	18.5	0.558
HPL %	20	14.3	0.277
CHF %	5.1	7.5	0.423
Smoking, %	68.6	87.3	0.030
Family History, %	34.3	46.9	0.175
Pain + palpitation, %	70	14.4	<0.001
0. hour troponin	30 (12.75-80)	155.5 (37.75-512.75)	<0.001
Peak troponin	100 (50-196)	345.5 (78-129)	<0.001
Troponin time-to-peak, hour	6 (1-7)	7 (3-15.75)	0.072
HR at presentatin, /min	79 (74-102)	78	0.142
LA diameter, mm	35.5 (30-39)	37	0.084
Medial E'	9.2 (6.9-11.6)	7.5 (34-39)	0.018
Lateral E'	11 (8.75-13.8)	11 (9-12)	0.179
Pro-BNP	275 (70-940)	188 (75.5-389)	0.082

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PP-002

Glomerular hyperfiltration is associated with poor prognosis in patients with atrial fibrillation

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Background and Aim: Atrial fibrillation (AF) is the most common chronic arrhythmia with high mortality and morbidity. Clinical risk factors are associated with poor outcomes in AF patients. Glomerular hyperfiltration, variably defined as an abnormally high renal glomerular filtration rate (GFR), increased filtration fraction, or increased filtration per nephron, is linked to the pathogenesis of chronic diseases like diabetes mellitus (DM) and atherosclerotic cardiovascular diseases (ASCVD). This study aimed to elucidate the relationship between glomerular hyperfiltration and adverse outcomes in AF patients.

Methods: Between 1 January 2019 and 1 January 2020, a total of 1060 patients with a diagnosis of AF in cardiology outpatient clinic evaluations were evaluated for the study. After excluding patients with GFR < 60 mL/min/1.73 m² according to the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula, eGFR was defined as values above the 95th percentile (≥ 107 mL/min/1.73 m²), while the normal filtration group included patients with eGFR between 60 and 105 mL/min/1.73 m². The primary outcome was all-cause death and the secondary outcome was ischaemic stroke during follow-up.

Results: During a mean follow-up period of 436 days, all-cause death occurred in 98 patients (16.1%) and ischaemic stroke in 40 patients (6.6%). Survival was worse in the hyperfiltration group compared to normofiltration [odds ratio (OR): 3.21, 95% confidence interval (CI): 1.83-5.62, $p=0.040$]. Compared to the normofiltration group, patients in the hyperfiltration group were younger (66.4 ± 11.3 vs. 58.8 ± 13.3 , $p<0.001$) and had lower left ventricular ejection fraction (LVEF) (53.8 ± 11.8 vs. 49.1 ± 13.8 , $p=0.021$). Although not reaching statistical significance, female gender and DM were more common in the hyperfiltration group. When CHADS-VASC scores were compared, the normofiltration group had a higher mean score (3.2 ± 1.4 vs. 2.9 ± 1.3 , $p=0.105$). High glomerular filtration rate was associated with a higher risk of mortality [odds ratio (OR): 5.29, 95% confidence interval (CI): 2.45-11.43, $p<0.001$] compared with normal glomerular filtration rate. Advanced age, chronic obstructive pulmonary disease (COPD) and smoking were other independent risk factors for poor prognosis in multivariate regression analysis.

Conclusions: A few pathophysiological pathways are responsible for the effects of glomerular hyperfiltration on cardiovascular diseases. The main pathophysiological pathways are RAS and sympathetic nervous system activation triggered by increased inflammation. In AF patients, increased adrenergic tone and neurohormonal activation during the hyperfiltration process adversely affect the course of the disease. Glomerular hyperfiltration, defined as eGFR ≥ 105 mL/min/1.73 m², is closely associated with all-cause mortality in patients with AF. This patient group may need to be approached carefully in terms of long-term risk assessment in patients with AF.

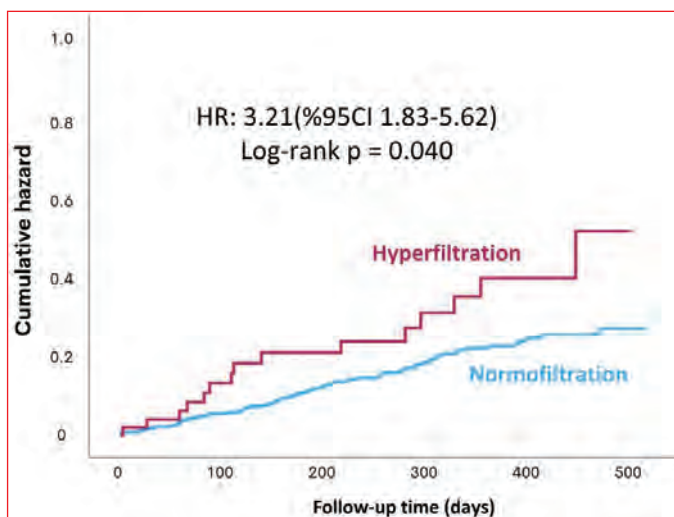


Figure 1. Cumulative hazard curves for all-cause mortality in patients with atrial fibrillation, comparing those with hyperfiltration to those with normofiltration.
HR, hazard ratio.

Table 1. Baseline demographic and clinical characteristics of the patients included in the study according to glomerular filtration groups

	Normofiltration (n=556)	Hyperfiltration (n=52)	p
Clinical features			
Age (years)	66.4 ± 11.3	58.8 ± 13.3	<0.001
Female, n (%)	257 (46.2)	30 (57.7)	0.113
Hypertension, n (%)	465 (83.6)	37 (71.2)	0.038
Previous MI, n (%)	133 (23.9)	12 (23.1)	0.523
Heart failure, n (%)	184 (33.1)	16 (30.8)	0.852
Diabetes mellitus, n (%)	239 (43.0)	26 (50.0)	0.329
Pulmonary embolism, n (%)	39 (7.0)	6 (11.5)	0.261
Hypothyroidism, n (%)	101 (18.2)	8 (15.4)	0.756
Obesity, n (%)	29 (5.2)	3 (5.8)	0.748
Hyperlipidaemia, n (%)	291 (52.3)	25 (48.1)	0.556
COPD, n (%)	224 (40.3)	19 (36.5)	0.598
Smoking, n (%)	193 (34.7)	18 (35.3)	0.933
eGFR (CKD-EPI)	82.6 ± 12.7	117.9 ± 14.7	<0.001
CHADS-VASC	3.2 ± 1.4	2.9 ± 1.3	0.105
Echocardiographic findings			
LVEF (%)	53.8 ± 11.8	49.1 ± 13.8	0.021
LA diameter (mm)	40.0 ± 5.7	39.8 ± 5.3	0.877
LVEDC (mm)	44.2 ± 10.5	44.7 ± 8.9	0.757
Severe TR, n (%)	63 (11.3)	5 (9.6)	0.277
sPAB (mmHg)	35 (15-48)	40 (30-58)	0.147
Medical treatment			
Beta blockers, n (%)	300 (54.0)	19 (36.5)	0.016
Digoxin, n (%)	65 (11.7)	2 (3.8)	0.135
Statins, n (%)	275 (49.5)	15 (28.8)	0.004
RASI, n (%)	256 (46.0)	17 (32.7)	0.064
Oral anticoagulation, n (%)	489 (87.9)	41 (78.8)	0.097
Amiodarone, n (%)	31 (5.6)	5 (9.6)	0.221
Outcomes			
Mortality, n (%)	83 (14.9)	15 (28.8)	0.040
Ischaemic stroke, n (%)	36 (6.5)	4 (7.7)	0.767

COPD: Chronic obstructive pulmonary disease; eGFR: Estimated glomerular filtration rate; LA: Left atrium; LVEDC: Left ventricular end-diastolic diameter; LVEF: Left ventricular ejection fraction; MI: Myocardial infarction; RASI: Renin angiotensin system inhibitor; sPAB: Systolic pulmonary artery pressure; TR: Tricuspid regurgitation.

Table 2. Independent risk factors associated with death in multivariate regression analysis

Variables	Adjusted Odds Ratio (95% CI)	p
Age	1.09 (1.06-1.12)	<0.001
Hyperfiltration	5.21 (2.45-11.43)	0.001
COPD	1.72 (1.07-2.74)	0.024
Smoking	1.84 (1.13-3.00)	0.015

COPD: Chronic obstructive pulmonary disease.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD**PP-005****TAPSE/sPAP ratio predicts whether heart failure patients will respond well to LBBAP therapy**

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Background and Aim: Tricuspid annular plane systolic excursion/systolic pulmonary artery pressure (TAPSE/sPAP) ratio is the validated non-invasive estimation of right ventricular-pulmonary arterial coupling easily obtained during a standard Doppler echocardiography. Left bundle branch area pacing (LBBAP) is a pacemaker method applied to provide cardiac resynchronization therapy (CRT). Appropriate patient selection is crucial for those undergoing LBBAP. The aim of this study is to evaluate the relationship between the TAPSE/sPAP ratio and the group of patients who respond well to LBBAP therapy.

Methods: In this study, 28 patients from our center with a left ventricular ejection fraction (LVEF) of less than 35% and a QRS duration of more than 150 ms who exhibited clinical symptoms of heart failure and underwent LBBAP-CRT therapy were included. Patients were evaluated for their response to CRT therapy at 0, 1, and 6 months. A good response to LBBAP therapy was defined as a 15% increase in LVEF compared to baseline as determined by follow-up echocardiography. TAPSE and sPAP values were measured echocardiographically and recorded prior to the LBBAP procedure, and the ratio was calculated. Patients were divided into two groups: those who responded well to LBBAP therapy and the other group.

Results: A total of 28 heart failure patients were included [mean age 65.3 ± 10 years, 50% (n=13) male]. A good response to LBBAP-CRT therapy was observed in 15 patients (53.6%). Among the patient population, 38.5% had coronary artery disease, 71.4% were hypertensive, and 53.6% were diabetic. In both the group that responded well to LBBAP and the other

group, hypertension, diabetes, and coronary artery disease were observed similarly ($p=0.410$, $p=0.136$, $p=0.705$). In the group that responded well to LBBAP, the follow-up ejection fraction was significantly higher compared to the other group (42.7 ± 9.1 vs. 26.3 ± 7.6 , $p<0.001$). The TAPSE/sPAP ratio was also significantly higher in the group that responded well (0.77 ± 0.30 vs. 0.54 ± 0.24 , $p=0.044$). In the TAPSE/sPAP ratio (ROC) curve analysis, the area under the curve (AUC) was calculated as 0.731 with 66.7% sensitivity and 76.9% specificity ($p=0.017$). A TAPSE/sPAP ratio value >0.72 was determined as the cut-off value for indicating a good response to LBBAP pacing therapy.

Conclusions: The TAPSE/sPAP ratio may serve as a predictor of echocardiographic response in heart failure patients planned for LBBAP. It could contribute to patient selection in the LBBAP procedure.

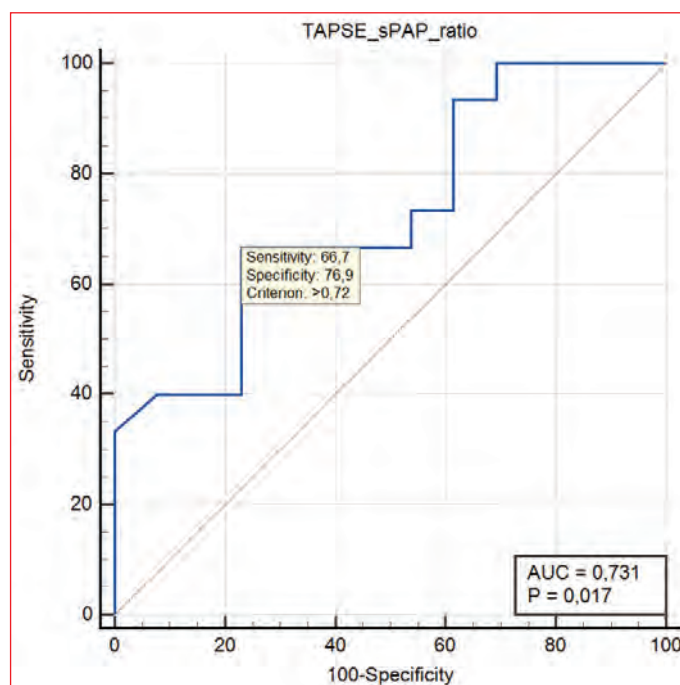


Figure 1. ROC curve analysis.

Table 1. Clinical and laboratory characteristics between the group that responded well to LBBAP therapy and the other group

	Responded well to LBBAP therapy (n=15)	The other group (n=13)	p value
Age \pm SD	65 \pm 11.3	66.6 \pm 7.8	0.647
Hypertension, n (%)	12 (80.0%)	8 (61.5%)	0.410
Diabetes mellitus, n (%)	10 (66.7%)	5 (38.5%)	0.136
CAD, n (%)	6 (40.0%)	4 (30.8%)	0.705
Non-ischemic dilated cardiomyopathy, n (%)	9 (60.0%)	9 (69.2%)	0.705
NT-proBNP, pg/mL, (IQR)	2430 (386-6462)	1957 (562-7147)	0.650
Albumin, g/dL, (IQR)	4.2 (3.7-4.4)	4.0 (3.6-4.4)	0.618
Creatinin, mg/dL, (IQR)	1.0 (0.8-1.4)	1.1 (0.6-1.5)	0.892
Bazal EF, \pm SD, \pm SD	26.5 \pm 5.6	24.4 \pm 6.4	0.376
Follow-up EF, \pm SD	42.7 \pm 9.1	26.3 \pm 7.6	<0.001
sPAP, \pm SD	29.7 \pm 11.9	40.9 \pm 19.0	0.070
TAPSE/sPAP ratio, \pm SD	0.77 \pm 0.30	0.54 \pm 0.24	0.044

CAD: Coronary arteria disease; EF: Ejection fraction; sPAP: Systolic pulmoner artery pressure.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PP-009

Assessment of awareness levels regarding anticoagulant use in patients with atrial fibrillation

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Background and Aim: Oral anticoagulants (OACs) should be used carefully due to their narrow therapeutic range and potential serious side effects. It has been observed in daily practice that patients have a low level of understanding regarding the reasons for using anticoagulants and their side effects. This survey aims to assess patients' knowledge and experiences regarding their disease and medication.

Methods: A survey was administered to 50 consecutive patients using oral anticoagulants due to atrial fibrillation (AF) who were admitted to a tertiary cardiology outpatient clinic.

Results: Of the 50 patients surveyed, 56% (28) were male. The mean age was 64 (\pm 12.6) years. Among the study group, 42% had graduated from primary school, and 20% had a university degree. 60% were retired, and 6% were office workers. 80% lived in urban areas. The median CHA₂DS₂-VASc score was 3. The duration of AF in patients was 72.98 months (min 1 month, max 300 months). The duration of oral anticoagulant use due to arrhythmia was 64.82 months (min 1 month, max 336 months). 34% percent were using rivaroxaban, 26% were using warfarin, 28% were using apixaban, 10% were using edoxaban, and 2% were using dabigatran. 96% percent were aware of their arrhythmia, but only 30% of patients knew the name of their arrhythmia. All patients were aware that they were using an anticoagulant. 40% (20) had never used any anticoagulant before, 17 (34%) had used aspirin, 7 (14%) had used warfarin, and 6 (12%) had used some form of anticoagulant but did not know which one. 90% reported using their medication regularly. When asked why they were using oral anticoagulants, 10% (5) said they did not know, 18% (9) said it was to correct the arrhythmia, 6% (3) said it was to prevent a heart attack, and 66% (33) said it was to prevent clot formation in the hear (Figure 1). 54% percent (27) of patients believed that the use of oral anticoagulants had positively affected their quality of life. Thirty-four percent (17) of patients reported that they had not received any information about their medication or did not remember being given information (Figure 2). 52% (26) indicated that they had no knowledge about side effects. 68% (34) had no information about food interactions. 84% (42) of patients had no knowledge about drug interactions. 60% (30) of patients indicated that they needed more information. Among those using direct oral anticoagulants (DOACs), 10% (4) were found to have unnecessary food restrictions. 92% of patients did not know when an OAC dose change was necessary. Despite the lack of knowledge, 68% of patients (34) reported not experiencing any side effects. Among the remaining 75% (12) reported minor bleeding, and 25% (4) reported a cerebrovascular event.

Conclusions: AF patients have a low level of understanding of the reasons for using oral anticoagulants and their side effects. In this context, it is necessary to improve cardiology clinics and develop specialized patient education programs.

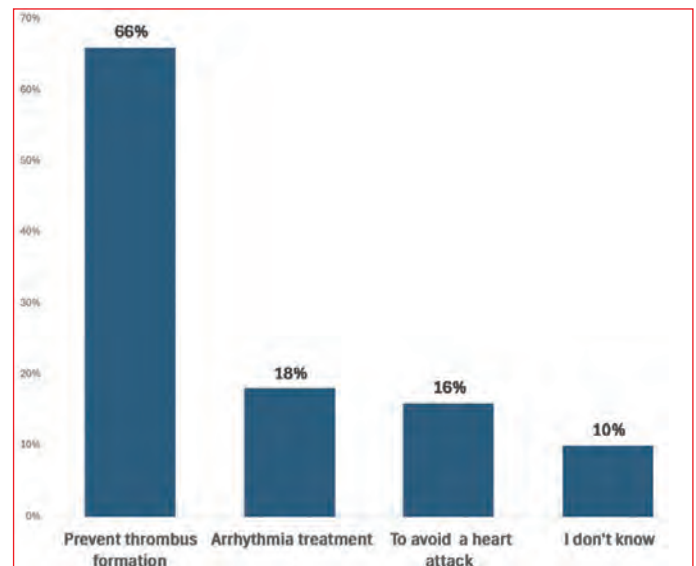


Figure 1. Why do you use blood thinners?

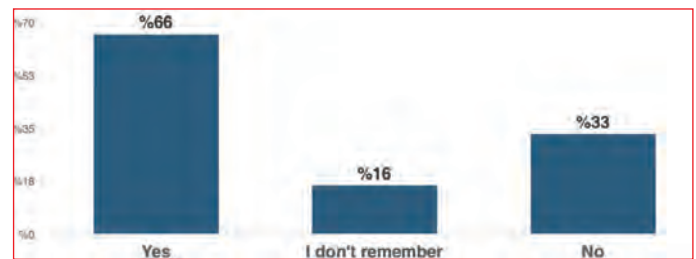


Figure 2. Were you given information about blood thinners before starting them?

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PP-012

Evaluation of frontal QRS-T angle as a predictor of mortality in hemodialysis patientsÇağlar Kaya¹, Mustafa Ebik¹, Merve Akbulut Çakır¹, İlhan Kılıç², Cihan Öztürk¹, Emirhan Çakır¹¹Department of Cardiology, Trakya University, Faculty of Medicine, Edirne²Department of Nephrology, Trakya University, Faculty of Medicine, Edirne

Background and Aim: The anterior Frontal QRS-T angle is one of the markers of the repolarization of the ventricles. In this study, we investigated whether or not frontal QRS-T angle could be a predictor of mortality in hemodialysis patients with 7-year follow-up.

Methods: 110 hemodialysis patients were enrolled in the study. Wide Frontal QRS-T angle was defined as its angle of

> 90°. Two different groupings were evaluated according to the width of the QRS-T angle and occurrence of death.

Results: 37 patients (34%) had wide Frontal QRS-T angle, and 73 patients (66%) had normal Frontal QRS-T angle. The mean age was found to be higher in the Wide Frontal QRS-T group and in the deceased group. Ejection fraction was found to be lower in the deceased group. It was also found that the Frontal QRS-T angle was wider and wider in the deceased group [94 (31-113), 33 (16-80) $p < 0.001$]. In univariate, multivariate and stepwise logistic regression analyses, being in the wider group was found to increase mortality (OR: 8.08, CI: 2.75-23.74, $p < 0.001$). Fragmented QRS was also found to increase mortality (OR: 11.25, CI: 2.98-42.49, $p < 0.001$).

Conclusions: Our results show that wide frontal QRS-T angle and being in the wide are associated with mortality in patients with hemodialysis patients.

Table 1. Univariate, multivariate and stepwise binary logistic regression analysis of the mortality

Log Reg Variables	Univariate Model			Multivariate Model			Stepwise Model		
	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p
Age	1,07	1,03-1,11	<0,001	1,04	0,98-1,10	0,129	0,03	1	0,138
DM	1,08	1,02-1,16	0,049	0,83	0,25-2,70	0,758	--	--	--
Width	9,38	3,75-23,45	<0,001	8,60	1,05-70,02	0,044	8,08	2,75-23,74	<0,001
Frontal QRS-T	1,02	1,00-1,03	<0,001	1,00	0,97-1,02	0,969	--	--	--
Fragmented QRS	11,04	3,59-33,86	<0,001	12,17	2,90-51,09	<0,001	11,25	2,98-42,49	<0,001
EF	0,96	0,91-1,02	0,24	1,01	0,93-1,09	0,755	--	--	--

DM, Diabetes Mellitus; EF, Ejection Fraction; CI, Confidence Interval.

Epidemiology

PP-013

Assessment of medical students' knowledge on familial hypercholesterolemia: insights from principal component analysis

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Background and Aim: Familial hypercholesterolemia (FH) is a genetic condition that can lead to severe cardiovascular diseases at an early age. This study aims to evaluate the knowledge level of medical students about FH and analyze the strengths and weaknesses of their education.

Methods: The study was conducted on 263 medical students at Ege University Medical School using a 24-item questionnaire. Principal Component Analysis (PCA) was applied to the student responses to identify distinct knowledge clusters: PC1 to PC5, each representing different areas of information.

Results: The PCA results and correct answer (CA) percentages are as follows:

- PC1: General disease understanding and clinical applications (36.51% variance, 49.74% CA)
- PC2: Diagnostic criteria and management strategies (10.06% variance, 26.42% CA)
- PC3: Biochemical markers and lipid profiles (8.17% variance, 32.12% CA)
- PC4: Treatment approaches and lifestyle impacts (7.59% variance, 8.61% CA)
- PC5: Cardiovascular risks associated with cholesterol levels (6.08% variance, 88.87% CA)

Conclusions: The analysis indicates strong knowledge and consensus in PC1 and PC5. PC1 shows sufficient understanding of FH's general characteristics and clinical practices among students; PC5 indicates a high accuracy in understanding the cardiovascular risks associated with cholesterol levels. In contrast, lower CA percentages in PC2 and PC3 and a very low CA with high variance in PC4 point to uncertainties and knowledge gaps among students in these areas. This suggests a need for more in-depth education on diagnosis, biochemical markers, and particularly on the effects of treatment approaches and lifestyle changes. Strengthening the curriculum in these areas will provide students with a more comprehensive and effective understanding of FH.

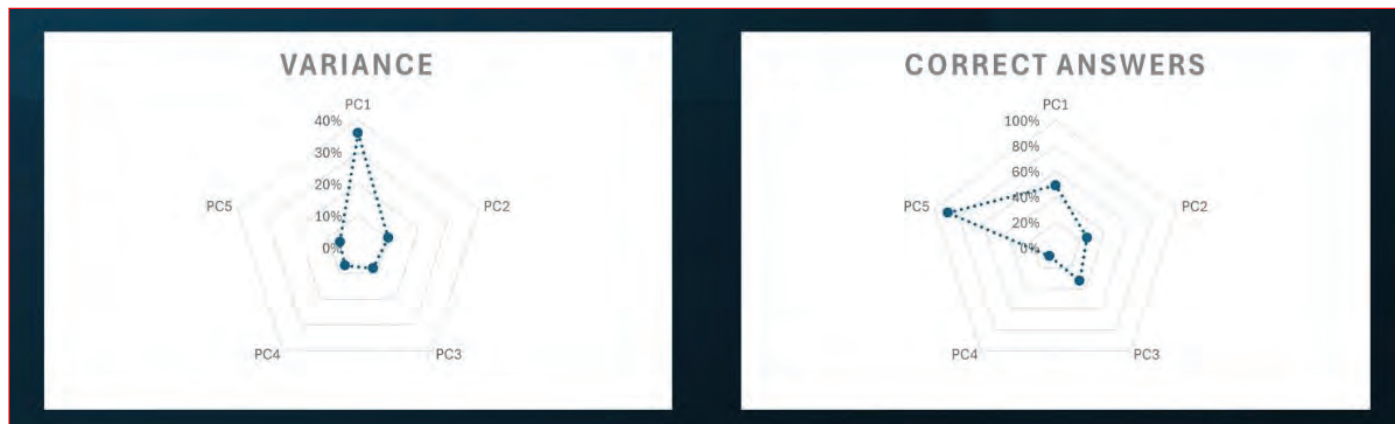


Figure 1. Radar Graph of Components

Epidemiology

PP-014

Evaluation of the relationship between glycemic control and aortic stiffness and arrhythmia frequency in diabetic patients with heart failure

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Background and Aim: Heart failure and diabetes are common and serious health issues in modern societies. The coexistence of these two conditions increases cardiovascular risks and complicates patient management. Dysregulation of blood sugar in diabetes can negatively impact the course of heart failure, yet there are gaps in our understanding of this interaction. This study aims to investigate the effects of blood sugar regulation on arterial stiffness and arrhythmias in patients with both diabetes and heart failure. The findings could provide valuable insights for optimizing the management of these conditions.

Methods: A total of 86 patients were included in our study, with the inclusion of patients who had an implanted cardiac device to accurately assess the frequency of arrhythmias. The study, which was initiated with the approval of the Ethics Committee, included patients selected based on inclusion and exclusion criteria from cardiology and endocrinology clinics. Inclusion criteria were symptomatic heart failure (NYHA class I-III), a diagnosis of diabetes, implantation of a cardiac device, and being in sinus rhythm, while exclusion criteria included NYHA class IV, pregnancy, breastfeeding, permanent atrial fibrillation, and inability to tolerate supine position. After obtaining written consent, the patients were evaluated with a prospective observational study design, with assessments of aortic stiffness, echocardiographic examination, and device checks conducted at baseline and at 6 months.

Results: At baseline, 86 patients were divided into two groups based on HbA1c levels: “controlled diabetes” (HbA1c <7.0%, n=29) and “uncontrolled diabetes” (HbA1c ≥7.0%, n=57). The heart failure types were 84.9% with reduced ejection fraction (HFrEF), 4.7% with preserved ejection fraction (HFpEF), and 10.5% with mid-range ejection fraction (HFmrEF). Pulse wave velocity was significantly lower in the controlled diabetes group compared to the uncontrolled group [9.4 (8.3-11.5) vs. 12.0 (9.6-15.0), p=0.009]. A moderate positive relationship was found between HbA1c levels and KF-PWV (r=0.250, p=0.01) and between KF-PWV and diabetes duration (r=0.296, p=0.006). At baseline and 6 months, patients with non-sustained/sustained VT and subclinical atrial fibrillation had significantly lower baseline HbA1c levels compared to those without these conditions

(VT: 6.8 ± 0.6 vs. 7.8 ± 1.5 , p=0.005; atrial fibrillation: 6.9 ± 0.7 vs. 7.9 ± 1.5 , p=0.007).

Conclusions: In conclusion, our study reveals that tight control of HbA1c levels has both positive effects and potential risks on cardiovascular health. It is concluded that HbA1c targets should be individualized for patients with diabetes and heart failure, and treatment should account for potential hypoglycemia risks. The limitations of our study underscore the need for further research on larger patient populations.

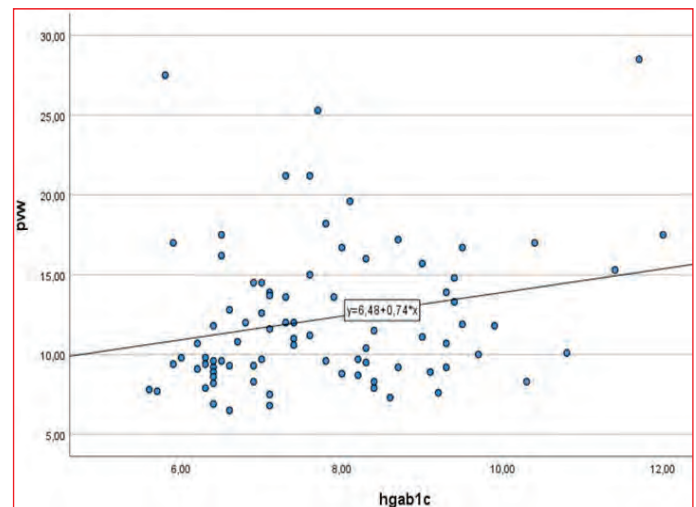


Figure 1.

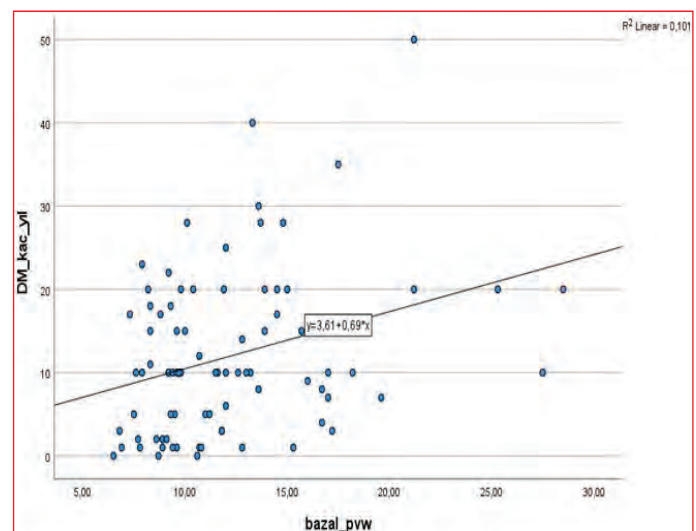


Figure 2.

Table 1. Clinical and laboratory characteristics between controlled and uncontrolled diabetes groups

	Controlled diabetes group	Uncontrolled diabetes group	p value
Age, years	65.0 (60.5-69.0)	65.0 (59.0-70.0)	0.574
HT, n (%)	17 (58.6)	31 (54.4)	0.709
Creatinin, mg/dL	1.0 (0.8-1.3)	1.1 (0.9-1.5)	0.078
HbA1c, %	6.4 (6.2-6.5)	8.3 (7.6-9.0)	<0.001
LVEF, %	28.0 (21.5-41.5)	30.0 (22.2-35.7)	0.671
CF-PWV	9.4 (8.3-11.5)	12.0 (9.6-15.0)	0.009
AP	12.0 (8.2-15.7)	10.0 (6.0-16.0)	0.640
SEVR	162.5 (151.0-193.5)	155.0 (139.0-174.0)	0.180
Subclinical atrial fibrillation	8 (27.6%)	5 (8.8%)	0.029

SEVR: Sub-endocardiyl viability ratio; HT: Hypertension; LVEF: Left ventricule ejection fraction; AP: Aortic augmentation; CF-PWV: Carotis-femoral pulse wave velocity.

Interventional Cardiology / Valvular and Structural Heart Disease

PP-016

Short- and mid-term outcomes of early alcohol septal ablation therapy for patients with mildly symptomatic hypertrophic obstructive cardiomyopathy: A tertiary center experience

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Background and Aim: According to the AHA guideline for HCM (2020), in patients with HOcm, earlier surgical myectomy performed at comprehensive HCM centers was recommended as a Class 2b indication in the presence of additional clinical risk factors, including poor functional capacity attributable to LVOTO as documented through treadmill exercise testing, or young adults with very high resting LVOTO. It is seen that the role of alcohol septal ablation (ASA) in patients with mildly asymptomatic HOcm is scarce. In this study, we aimed to identify the efficacy and safety of earlier ASA procedures among patients with mildly symptomatic HOcm having poor functional capacity as documented through exercise testing, as well as adults with very high resting or a provokable LVOT gradient (>50 mmHg) despite maximal tolerated medical therapy.

Methods: 47 patients with mildly symptomatic HOcm having poor functional capacity as documented through exercise testing, and patients with resting or provokable LVOT gradients >50 mmHg despite maximal tolerated medical therapy, were included. In this study, we aimed to determine the clinical outcomes according to targets for the post-ASA procedure described in the 2020 AHA guidelines for HCM: 30-day mortality; 30-day adverse complications (tamponade, LAD dissection, infection, major bleeding); 30-day complete heart block resulting in need for permanent pacemaker; more than moder-

ate residual mitral regurgitation; repeat procedure rate; improvement NYHA class; rest or provoked LVOT gradient <50 mmHg.

Results: Clinical and echocardiographic characteristics at baseline and six-month follow-up of the patients are shown in Table 1. The classification of clinical endpoints according to targets for post-ASA procedure described in the 2020 AHA guidelines for the diagnosis and treatment of patients with HCM are summarized in Table 2.

Conclusions: Recently, the Euro-ASA registry results reported that long-term survival after ASA in mildly symptomatic patients with HCM was similar to the expected survival of an age- and sex-matched general population; moreover, patients who underwent ASA had LVOT gradient reduction and symptomatic relief with a low risk of developing heart failure. The reason why the mortality rate in our study was lower than in the existing literature may be explained by the fact that the patients included in our study were younger than the patients included in other studies; moreover, these patients were mildly symptomatic, and their structural heart changes were not obviously evident. Conclusions; in patients with HOcm, earlier ASA performed at comprehensive centers may be a reasonable solution in the presence of poor functional capacity attributable to LVOTO, as documented through treadmill exercise testing in terms of short- and mid-term clinical outcomes. We believe that as a result of large-scale similar and consistent studies on this subject, the recommendation contents will be changed in future guidelines.

Table 1. Clinical and echocardiographic characteristics at baseline and six-month follow-up of patients with HOcm treated with ASA

	Baseline (n = 47)	Follow-Up (n = 47)	p Value
NYHA class	2 ± 0	1.2 ± 0.5	<0.01
LV gradient at rest, mmHg *	43 (20-140)	14 (0-20)	<0.01
LV gradient after provocation, mmHg *	84 (50-156)	26 (5-35)	<0.01
LV ejection fraction, %	60 ± 8	63 ± 5	0.340
Basal septum thickness, mm *	18 (16-23)	16 (14-18)	<0.01
Left atrium diameter, mm	44 ± 0.3	42 ± 0.2	0.03
Pulmonary artery systolic pressure, mmHg	25 ± 7	14 ± 5	<0.01

* Mann-Whitney U test was used for non-normally distributed variables and expressed by median (minimum-maximum). LV: Left ventricular; NYHA: New York Heart Association.

Table 2. Example targets for alcohol septal ablation outcomes according to 2020 ACC/AHA HCM guidelines (+)

Events	ASA (%)	Our Procedure, n (%)	p Value
30-day mortality	≤1%	0%	<0.01
30-day adverse complications (*)	≤10%	0%	<0.01
30-day complete heart block resulting in the need for permanent pacemaker	≤10%	2 (4.2%)	<0.01
More than moderate residual mitral regurgitation (**)	≤5%	1 (2.1%)	<0.01
Repeat procedure rate (***)	≤10%	2 (4.2%)	<0.01
Improvement NYHA ≥ class (**)	>90%	45 (95.7%)	<0.01
Rest and provoked LVOT gradient <50 mmHg (**)	>90%	46 (97.8%)	<0.01

Interventional Cardiology / Valvular and Structural Heart Disease

PP-018

Contrast echocardiography with cocktailed fluid for alcohol septal ablation

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Background and Aim: Septal reduction therapy (SRT), alcohol septal ablation (ASA) has been recommended in patients with recurrent exertional syncope caused by a resting or maximum provoked left ventricle outflow obstruction gradient ≥ 50 mmHg despite optimal medical therapy in obstructive hypertrophic cardiomyopathy (HOCM). Selective injection of alcohol into a septal perforator artery leads to create a localized septal scar. Angiographic identification could not be sufficient detect correct positioning of alcohol in the target myocardial area due to excessive anatomic variety and potential collateralization in the septal perforatory artery. Therefore, myocardial contrast echocardiography is necessary to test each target septal branch. The echocardiographic contrast agent is injected through the balloon catheter with simultaneous transthoracic echocardiography and compared with the same views recorded at baseline. However, these contrast agents (Levovist, Gelafundin) are expensive and not widely available. Hence, we have replaced these agents with a simple, inexpensive technique.

Methods: 13 patients (7 male, 6 female) with symptomatic HOCM underwent ASA between the years 2017 and 2024. The principles of Declaration of Helsinki were followed in this study. Written informed consent was obtained from all the patients. A guidewire was passed into the first septal branch. A 2.5-mm over-the-wire balloon was advanced to the first perforatory and was inflated. To confirm the target myocardial area in the first perforatory, we used new technique as myocardial contrast echocardiography. This technique included cocktail fluid (agitated 5 mL of saline plus 0.5

mL of blood of patient, shaken in a 3-way-stopcock). We used 2 mL of cocktailed fluid into the first perforatory artery. Transthoracic echocardiography showed the enhancement of the basal septum. Then, 1-2 mL of ethanol was injected to the first perforatory artery.

Results: We saw clearly related target area in echocardiography. Further, we didn't see complications related to its use as backflow of bubbles to the left anterior descending artery.

Conclusions: This technique is a feasible alternative for septal ablation in places where synthetic contrast agents are not available.

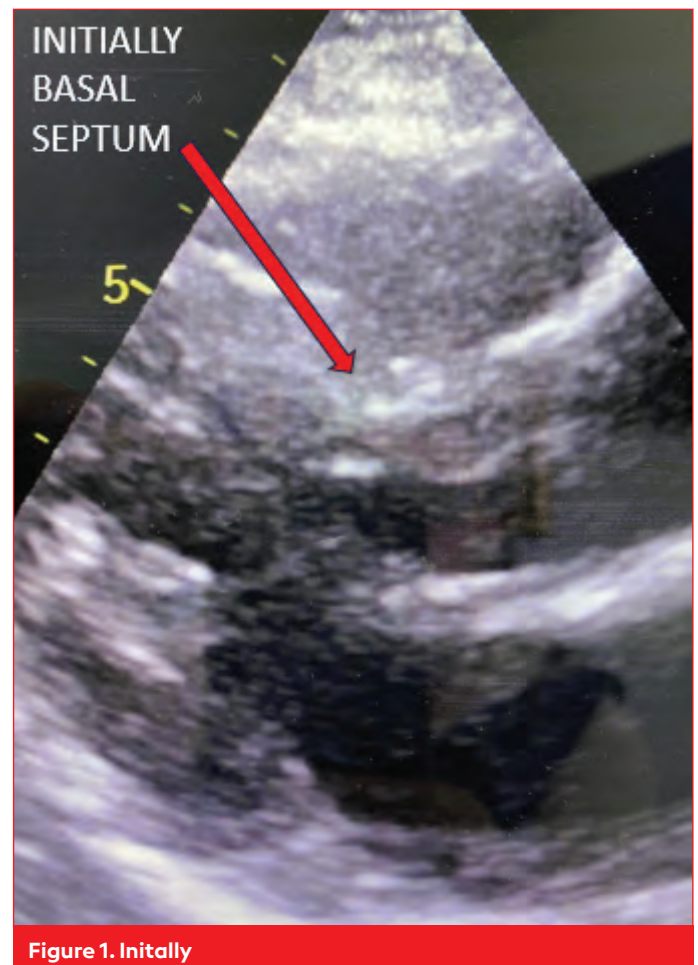


Figure 1. Initially

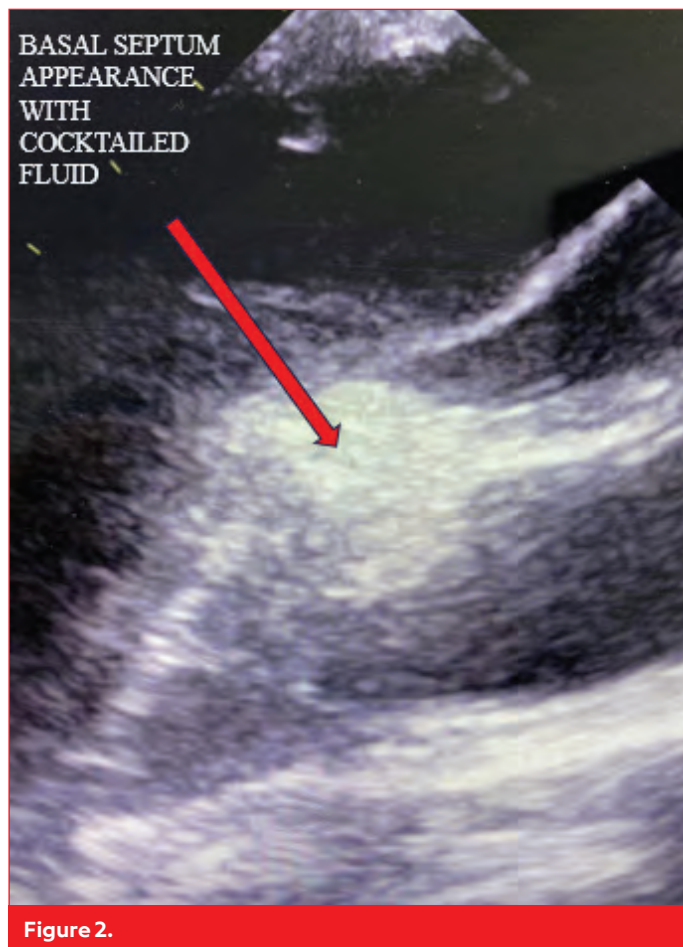


Figure 2.

Interventional Cardiology / Valvular and Structural Heart Disease

PP-019

Our valve in valve TAVI results

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Objective: Symptomatic severe aortic valve stenosis is increasingly treated with bioprosthetic valves. Transcatheter valve-in-valve (VIV) implantation has emerged as a new treatment option for patients with degenerative bioprosthetic aortic valves who are at high surgical risk. Initial cases utilized first-generation TAVI devices within bioprosthetic aortic valves, which resulted in high post-procedural gradients and insufficient orifice areas. With advancements in devices and operator expertise, their feasibility has improved. We report the feasibility of VIV implantation using these new devices and our clinical outcomes in a series of patients with degenerated aortic bioprostheses.

Method: Between 2012 and 2024, a total of 678 TAVI procedures were performed at the University of Health Sciences Ahi Evren Chest, Heart, and Vascular Surgery Training

and Research Hospital. Patients who underwent VIV procedures were identified through hospital system records. Our first VIV case was performed in 2017. All patients were evaluated by the heart team and considered high-risk for surgical aortic valve replacement (mean logistic EuroSCORE 7.2%). Patients met the VARC-3 criteria for prosthetic valve dysfunction. Pre-procedural transesophageal echocardiography was performed in all patients to rule out endocarditis and thrombus.

Results: VIV procedures were performed in a total of 8 patients. The mean age of the patients was 86.6 years. The mean body mass index was 28 kg/m². Five patients were female, and three were male. Pre-procedural maximum gradients averaged 68.75 mmHg, and mean gradients averaged 43.3 mmHg. The mean Doppler velocity index was 0.24, and the effective orifice area was 0.65 cm². No in-hospital or 1-month mortality was observed. Within six months, 1 patient died due to pulmonary edema. Between 6 months and 1 year, there was no mortality. After 1 year, 3 patients died (2 cardiac and 1 non-cardiac causes). The femoral approach was used in all VIV cases. Four patients had surgical bioprostheses, and four had dysfunctional TAVI prostheses. Seven cases underwent VIV due to severe AS, and 2 due to severe AR. One patient (St. Jude Epic Porcine) underwent VIV due to structural valve dysfunction. The mean duration of dysfunction after initial bioprosthetic implantation was 9 years. All VIV procedures used 1 Proglide for closure. One patient experienced a peripheral complication and underwent femoral artery graft stent implantation. No patients required permanent pacemaker implantation. Post-procedural maximum gradients averaged 19.8 mmHg, and mean gradients averaged 12 mmHg.

Discussion: Aortic bioprostheses with small inner diameters remain a challenging pathology for TAVI due to high residual gradients and small aortic orifice areas. However, advances in valve technologies and operator experience have facilitated successful valve-in-valve procedures.

Table 1. Demographic Data

Mean Age	86.6
Mean BMI	28.2
Gender	5 Female 3 Male
Mean EOA	0.65
Mean DVI	0.22
Duration of Dysfunction/Years	9
Surgical Bioprosthetic Valve Dysfunction Duration/Years	10.5
TAVI Bioprosthetic Valve Dysfunction Duration/Years	7.5

Table 2. Pre- and Post-Procedure Valve Data

Valve size	Pre-Procedure	Post-Procedure
23 mm	5	3
26 mm	3	5
Types of Valve		
Edwards Saphien XT	4	1
St Jude Epic Porcine	1	
Perceval Large	1	
Myval		3
Medtronic Evolute R		4
Medtronic Freestyle	2	

Table 3. Pre- and Post-Procedure Valve Data

	Pre-Procedure	Post-Procedure	6 Months
Moderate and Severe PVL	2	0	0
Maximum Gradient (Mean) mmHg	68.75	19.8	22
Mean Gradient (Mean) mmHg	43.3	12	13

Table 4. VARC-3 Data

Parameter	Value
In-hospital Mortality	%0
Periprocedural Death	%0
Early Death	%12.5 (1)
Late Death	%37.5 (3)
Technical success	%100
Device success	%87.5
Early safety	%87.5
Clinical efficacy	%62.5

Interventional Cardiology / Carotid and Peripheral Vascular

PP-020

Increased serum CRP-albumin ratio is independently associated with in-stent restenosis after carotid artery stenting

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Background and Aim: Atherosclerotic stenosis of the carotid artery is responsible for 10-15% of ischemic strokes. Carotid artery stenting (CAS) has emerged as one of the standard options for managing symptomatic carotid stenosis and is considered an alternative to carotid endarterectomy (CEA). However, studies have reported an incidence of in-stent restenosis (ISR) ranging from 3.3-21% after a follow-up period of 6 months to 2 years. The presence of ISR can significantly impact the long-term benefits and safety profile of CAS. Inflammation's well-established role in the pathogenesis of carotid atherosclerosis necessitates investigation into various inflammatory cells and mediators involved in carotid disease and stroke to better identify high-risk patients. C-re-

active protein (CRP), a widely used clinical inflammatory marker, plays a significant role in the inflammatory response to atherosclerosis. The present study was designed with the aim of investigating the correlation between CAR and ISR.

Methods: We performed a single-center, retrospective, study of 529 patients who underwent carotid artery stenting were evaluated between the years 2010 and 2020. The patients were divided into 2 groups according to ISR after carotid artery stenting. Demographic, clinical characteristics and laboratory parameters were collected from hospital records and follow-up data were obtained from the national health registration system or phone calls. The blood parameters were evaluated before carotid intervention. We included patients who developed ISR after carotid artery stenting. Patients with conditions that could affect CRP levels, such as multiple organ failure, systemic inflammatory response syndrome or malignancy were excluded from the study.

Results: When we evaluated the laboratory parameters, creatinine, glucose, leucocytes, complete blood counts, triglycerides, liver enzymes and lipid panel were similar between groups, but uric acid ($p<0.01$), Alb ($p<0.01$), CRP ($p<0.01$) and CAR ($p<0.01$) were higher in the restenosis (+) group. The mean CAR was 31.8 mg/dL. In addition, we found that eGFR was lower in the restenosis (+) group. We entered these variables into the multivariable regression analysis, and found that CAR [Hazard Ratio (HR): 1.13, 95% Confidence Interval (CI): 1.03-1.24 $p=0.01$], diabetes mellitus (HR: 2.24, 95% CI: 1.29-3.90 $p<0.01$), smoke (HR: 18.8, 95% CI: 9.24-38.4 $p<0.01$) were independent predictors of carotid artery ISR. In ROC analysis, CAR >0.28 predicted ISR after CAS with sensitivity 93% and a specificity of 89%.

Conclusions: Our study highlights the prognostic importance of CAR in CAS patients with ISR, being an independent risk factor. This inexpensive and easy to assess biochemical parameter may be helpful in improving ISR prediction and the selection of the patients that could benefit from a prevention strategy.

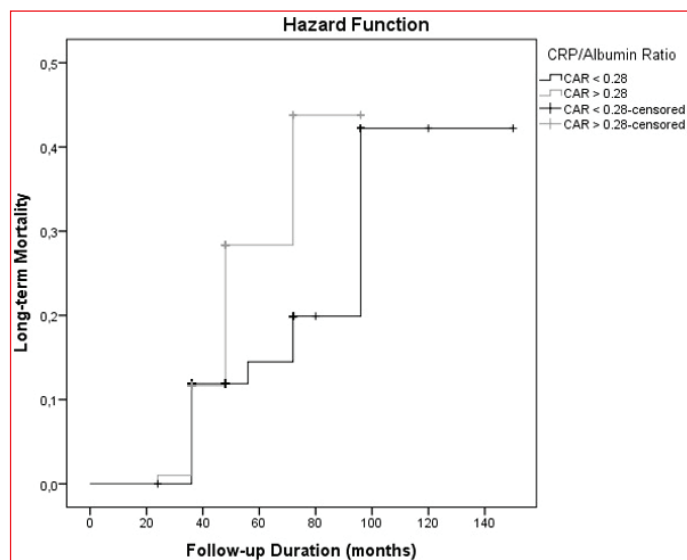


Figure 1. Kaplan-Meier curve for long term mortality. On a Kaplan-Meier curve, patients with CAR >0.28 had significantly higher risk for long-term mortality (log-rank, $p<0.01$).

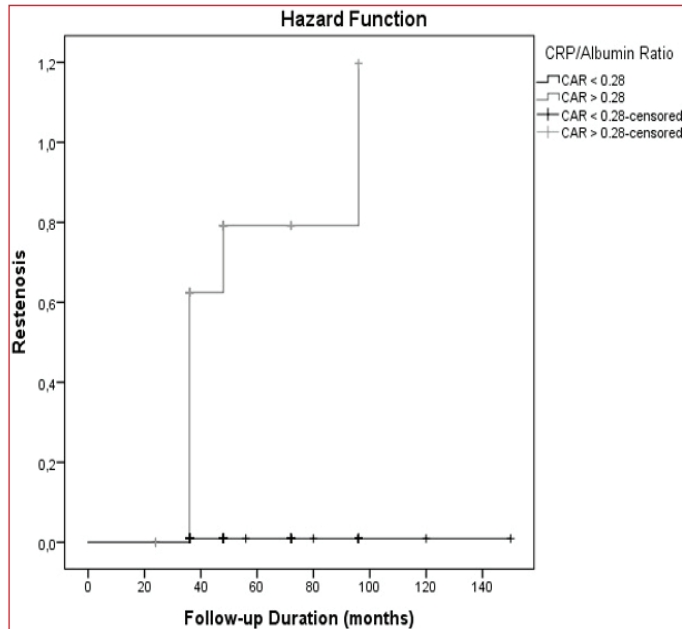


Figure 2. Kaplan-Meier curve for ISR. On a Kaplan-Meier curve, patients with CAR >0.28 had significantly higher risk for restenosis (log-rank, p<0.01).

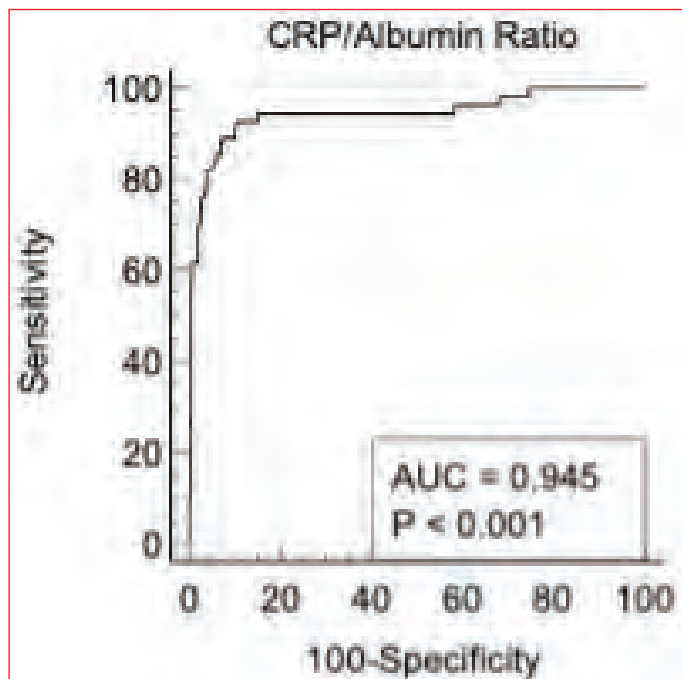


Figure 3. The relationship between C-reactive protein-albumin ratio (CAR), C-reactive protein (CRP) and albumin (Alb) levels in in-stent restenosis (ISR). In Receiver Operating Characteristic (ROC) analysis, CRP-Albumin ratio >0.28 predicted ISR with a sensitivity of 93% and a specificity of 89%. The area under the receiver curve (AUC) of CAR for predicting carotid stent restenosis was 0.945 [95% confidence interval (CI): 0.923-0.963, p<0.001]

Interventional Cardiology / Coronary

PP-026

Obesity paradox and long-term cardiovascular outcomes in acute coronary syndrome patients undergoing percutaneous coronary intervention

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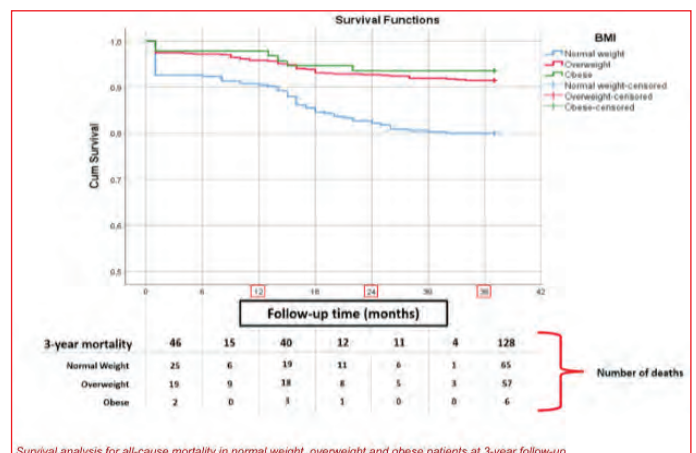
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Background and Aim: Obesity is a major risk factor for cardiovascular disease, and current treatment guidelines recommend maintaining a normal BMI range of 20-25 to reduce the risk of cardiovascular diseases. However, there is evidence to suggest that obese patients with acute coronary syndrome (ACS) may have better survival rates than non-obese patients during hospitalization and long-term follow-up, known as the obesity paradox. Nevertheless, the impact of the obesity paradox specifically in ACS patients who received only routine percutaneous coronary intervention (PCI) has not been studied. Therefore, the aim of our study was to investigate the impact of the obesity paradox in ACS patients who underwent only routine PCI during their hospitalization and for a period of 3 years of follow-up.

Methods: Kaplan-Meier analysis/Log-rank test, evaluated survival times among patients over a 3-year follow-up based on BMI categories (normal-overweight-obese). Univariate and multiple logistic regression analyses (Backward LR method) were performed to construct long-term mortality prediction models.

Results: The group of obese patients had the lowest rate of in-hospital mortality (n=2; 2.2%) compared to the other BMI groups ($\chi^2=12.448$; p=0.002). The overweight group also had a more favourable in-hospital mortality rate (n=19; 2.8%) compared to the normal weight group (n=24; 7.4%) ($\chi^2=12.448$; p=0.002). Although the three-year survival rates were similar between the obese and overweight groups, both groups had

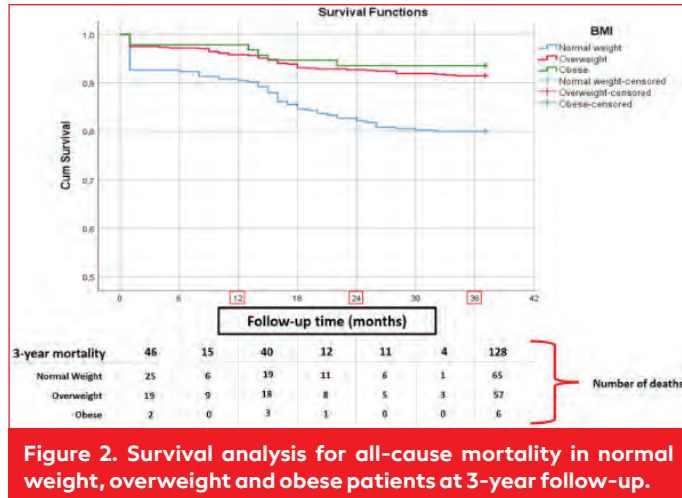


Survival analysis for all-cause mortality in normal weight, overweight and obese patients at 3-year follow-up

Figure 1.

significantly better survival rates than the normal weight group [6 (6.5%), 57 (8.5%), 65 (20.1%), $\chi^2=30.662$, $p<0.001$] (Figure 1).

Conclusions: Obesity and being overweight are established risk factors for the development of cardiovascular disease. However, the obesity paradox suggests that obese and overweight patients with acute coronary syndrome who undergo percutaneous coronary intervention (PCI) have a lower mortality risk compared to non-obese patients. This paradoxical effect should be considered when considering the appropriate treatment strategies for these patients.



Interventional Cardiology / Coronary

PP-028

Association of syntax score with B-type natriuretic peptide in elderly patients undergoing percutaneous coronary intervention

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Background and Aim: Elderly patients (≥ 75 years) with coronary artery disease became an increasing patient population in real life practice. In our study, we aimed to assess the relation of SYNTAX score with pro B-type natriuretic peptide (PRO-BNP) in elderly patients undergoing percutaneous coronary intervention (PCI).

Methods: A total of 236 elderly patients undergoing PCI between January 2017 and April 2023 from our university hospital enrolled to the analysis. Eighty-two non-ST segment elevation myocardial infarction patients, 90 ST segment elevation myocardial infarction patients, 12 unstable angina patients and 52 chronic coronary syndromes patients were enrolled. Clinical and follow-up data was obtained from the medical records. The SYNTAX score (SS) was calculated due to the web site calculator and patients were divided into 2 groups: Low SS group (<22) and high SS group (≥ 22).

Results: Out of 236 elderly patients; 193 patients were assigned to the low SS group and 43 patients were assigned

to the high SS group. There was no significant difference between 2 groups in terms of diabetes, hypertension, dyslipidaemia, coronary artery disease and clinical presentation. Mean SYNTAX score was 12 in low SS group and 28 in high SS group. While median baseline PRO-BNP level was 2213 pg/mL in low SS group, it was 3521 pg/mL in the high SS group ($p=0.032$). There was no significant difference in terms of coronary calcification severity, ratio of thrombus in culprit lesion and TIMI III flow after PCI. Ejection fraction was significantly decreased in high SS group (46.7 vs. 40.7, $p=0.006$).

Conclusions: Our study showed that PRO-BNP level was associated with higher SS syntax score and lower ejection fraction in elderly patients undergoing PCI in setting of different clinical scenarios.

Table 1.

	SYNTAX Score <22	SYNTAX Score ≥ 22	p
Pro-BNP (pg/mL)	2213	3521	0.032
Troponin T (ng/mL)	0.79	3	0.002
LVEF (%)	46.7	40.78	0.006

Interventional Cardiology / Coronary

PP-029

The triglyceride glucose index may predict significant coronary stenosis in moderate left main coronary artery lesions: An intravascular ultrasonography study

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Background and Aim: There may be severe difficulties in determining the severity of left main coronary artery (LMCA) lesions. The use of intravascular ultrasound (IVUS) facilitates the decision about the lesion severity in these patients. Previous studies have shown a close association of the triglyceride glucose (TyG) index with coronary atherosclerosis. The aim of this study was to investigate the relationship between TyG index and lesion severity in patients who have undergone LMCA IVUS.

Methods: The study included 180 patients who were determined with intermediate coronary stenosis (ICS) in LMCA and underwent an IVUS procedure. The patients were separated into two groups according to the TyG index values as those <9.83 and ≥ 9.83 . In the IVUS measurements of these patients, the plaque burden (PB) and the minimal lumen area (MLA) showing lesion severity were measured. Lesions calculated as $MLA < 6 \text{ mm}^2$ or $PB \geq 65\%$ were considered critical lesions.

Results: The patients comprised 136 (75.6%) males and 44 (24.4%) females with a median (IQR) age of 59 (53-68) years

(Table 1). In the group with high TyG index, MLA values were significantly lower (7.74 ± 4.1 vs. 6.24 ± 2 , $p=0.032$) and PB percentages were significantly higher (54.5 ± 15.2 vs. 59.8 ± 11.4 , $p=0.05$). In addition, the number of patients with critical lesions detected in IVUS measurements was significantly higher in the group with high TyG index (69 vs. 19, $p=0.005$) (Table 2). SYNTAX score was significantly higher in patients with critical lesions detected on IVUS (17 ± 9 vs. 6 ± 8 , $p<0.001$) (Table 3).

Conclusions: The results of this study suggested that there was a significant association between the TyG index and lesions evaluated as critical on IVUS, which may predict anatomically important lesions in patients with moderate degree of LMCA stricture. TyG index can be a surrogate marker for invasive treatment modality for the management of patients with lesions of LMCA in the grey zone for intervention.

Table 1. The baseline characteristics and laboratory investigations of all patients according to TyG index

	All patients (n=180)	TyG index <9.83 (n=155)	TyG index \geq 9.83 (n=25)	p value
Demographic characteristics				
Age, y	59 (53-68)	59 (52-68)	62 (58-68)	0.051
Male sex, %	136 (75.6)	115 (74.2)	21 (84)	0.451
Body mass index, kg/m ²	26.8 (24.7-29)	26.8 (24.5-29)	26.2 (25.2-28.1)	0.978
Comorbidities				
Hypertension, %	112 (62.2)	97 (62.6)	15 (60)	0.827
Diabetes mellitus, %	42 (23.3)	31 (20)	11 (44)	0.019
Hyperlipidemia, %	46 (25.6)	41 (26.5)	5 (20)	0.624
Smoking, %	114 (63.3)	97 (62.6)	17 (68)	0.661
Chronic kidney disease, %	27 (15)	22 (14.2)	5 (15)	0.544
Medications				
Acetylsalicylic acid use, %	179 (99.4)	154 (99.4)	25 (100)	1.000
P2Y12 Inh use, %	144 (80)	123 (79.3)	21 (84)	0.717
B-blocker use, %	169 (93.9)	144 (92.9)	25 (100)	0.366
ACE Inh use, %	154 (85.6)	131 (84.5)	23 (92)	0.539
Statin use, %	176 (97.8)	152 (98.1)	24 (96)	0.453
OAD use, %	46 (25.6)	32 (20.6)	14 (56)	0.001
Laboratory assessment				
Hemoglobin, g/dL	13.2 (12-14.5)	13.2 (12-14.6)	13.2 (11.8-13.9)	0.610
eGFR, mL/min/1.73 m ²	90.3 (70.2-101.9)	92.3 (70.1-103.3)	88.2 (74-99.2)	0.450
Total cholesterol, mg/dL	167 (144-206.5)	166 (138-204)	183 (159-232)	0.047
Triglycerides, mg/dL	145.5 (100-204)	135 (95-173)	302 (232-345)	<0.001
LDL cholesterol, mg/dL	90.7 (68-126)	89.9 (69-127.6)	95.9 (62.2-124.1)	0.675
Albumin, g/L	42 (39-44)	42.1 (39.1-44.3)	41.6 (38.8-44.9)	0.849
FBG, mg/dL	117 (98-171.5)	110 (95-159)	175 (124-229)	0.001
Uric acid, μ mol/L	5.4 (4.7-6)	5.4 (4.7-6.1)	5.2 (4.5-5.9)	0.928
TyG index	9.2 (8.7-9.5)	9.04 (8.6-9.4)	10.1 (10-10.3)	<0.001

Data are presented as median (interquartile range) or number (percentage) of patients. Abbreviations: ACE: Angiotensin converting enzyme; eGFR: Estimated glomerular filtration rate; FBG: Fasting blood glucose; IQR: Interquartile range; LDL: Low-density lipoprotein; OAD: Oral antidiabetic; TyG: Triglyceride glucose

Table 2. Angiographic and procedural status of patients according to TyG index

Angiographic parameters	All patients (n=180)	TyG index <9.83 (n=155)	TyG index ≥9.83 (n=25)	p value
Procedural data				
SYNTAX score	11 ± 10	11 ± 10	13 ± 10	0.361
IVUS-based volume parameters in LMCA				
EEM volume, mm ³	149.2 ± 48.8	151.1 ± 47.6	137.5 ± 55.1	0.253
Lumen volume, mm ³	67.2 ± 37	69.3 ± 38.2	54.2 ± 24.2	0.011
Plaque volume, mm ³	82 ± 33.1	81.7 ± 32	83.5 ± 40	0.833
MLA, mm ²	7.56 ± 3.9	7.74 ± 4.1	6.24 ± 2	0.032
MLA <6 mm ²	80 (44.4)	65 (41.9)	15 (60)	0.071
Plaque burden, %	55.2 ± 14.8	54.5 ± 15.2	59.8 ± 11.4	0.050
Plaque burden ≥65%	64 (35.6)	52 (33.5)	12 (48)	0.181
Critical lesion, n	88 (48.9)	69 (44.5)	19 (76)	0.005

Data are presented as mean ± SD or number (percentage) of patients. Abbreviations: CABG: Coronary artery bypass surgery; CAG: Coronary angiography; EEM: External elastic membrane; IVUS: Intravascular ultrasound; MLA: Minimal lumen area; PCI: Percutaneous coronary intervention; SD: Standard deviation; SYNTAX: The synergy between percutaneous coronary intervention with taxus and cardiac surgery; TyG: Triglyceride glucose.

Table 3. Basic characteristics and laboratory examinations of all patients according to critical lesions on IVUS

	All patients (n=180)	Critical lesion (+) (n=88)	Critical lesion (-) (n=92)	p value
Age	59 (53-68)	59 (55-70)	59 (51-68)	0.059
Male gender	136 (75.6)	67 (76.1)	69 (75)	0.864
Hypertension, %	112 (62.2)	54 (61.4)	58 (63)	0.878
Diabetes mellitus, %	42 (23.3)	22 (25)	20 (21.7)	0.725
Hyperlipidemia, %	46 (25.6)	26 (29.5)	20 (21.7)	0.238
Chronic kidney disease, %	27 (15)	14 (15.9)	13 (14.1)	0.835
Hemoglobin, g/dL	13.2 (12-14.5)	12.9 (11.9-14.3)	13.6 (12.2-14.6)	0.218
eGFR, mL/min/1.73 m ²	90.3 (70.2-101.9)	91.4 (70.1-101.9)	89.3 (71.1-101.9)	0.941
Total cholesterol, mg/dL	167 (144-206.5)	162.5 (133.5-204)	175.5 (151-209.5)	0.073
LDL cholesterol, mg/dL	90.7 (68-126)	84.2 (61-120)	97.5 (71-135)	0.064
Triglycerides, mg/dL	145.5 (100-204)	146 (112-197.5)	144.5 (94.5-205.5)	0.439
FBG, mg/dL	117 (98-171.5)	160 (114.5-194)	105 (89.5-123)	<0.001
TyG index	9.2 (8.7-9.5)	9.39 (8.96-9.72)	8.98 (8.51-9.33)	<0.001
SYNTAX score	11 ± 10	17 ± 9	6 ± 8	<0.001

Data are presented as median (interquartile range), mean ± SD or number (percentage) of patients. Abbreviations: eGFR: Estimated glomerular filtration rate; FBG: Fasting blood glucose; LDL: Low-density lipoprotein; SD: Standard deviation; SYNTAX: The synergy between percutaneous coronary intervention with taxus and cardiac surgery; TyG: Triglyceride glucose

Interventional Cardiology / Coronary

PP-032

Coexistence of coronary artery ectasia and coronary slow flow triggers platelet activation

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Background and Aim: Coronary Slow Flow (CSF) is an angiographic finding characterized by delayed distal vessel opacifi-

cation. Coronary Artery Ectasia (CAE) is one of the secondary causes of CSF. In both conditions, the Thrombolysis In Myocardial Infarction (TIMI) frame count is significantly elevated. They may rarely be the cause of life-threatening arrhythmias, myocardial ischemia, sudden cardiac death, or recurrent acute coronary syndrome. SCUBE-1 (Signal Peptide-CUB-EGF Domain-Containing Protein-1) increases platelet-endothelial cell adhesion molecule levels, platelet aggregation, and platelet adhesion to the subendothelial matrix. Studies have shown that it is associated with endothelial dysfunction and is significantly elevated in diseases such as acute coronary syndrome (ACS) and acute ischemic stroke. In our study, we evaluated serum SCUBE-1 levels as a marker of platelet activation in patients with CSF, with and without CAE.

Methods: The study we included 60 patients (25 females, 35 males; mean age 59 ± 7) who underwent coronary angiography

Table 1. Comparison of parameters related to the coagulation cascade

Variables	CSF and CAE n=32	CSF n=28	Control n=27	p value
Platelet, 10 ³ /μL	247 ± 51	239 ± 41	272 ± 61	0.06
D-dimer, ng/mL	365 ± 305★	316 ± 295	192 ± 165	<0.05
Fibrinogen, mg/dL	280 ± 73★	265 ± 62	230 ± 35	<0.05
D-dimer/Fibrinogen ratio	1.29 ± 1.07	1.23 ± 1.14	0.84 ± 0.77	0.20
MPV, fL	8.4 ± 1.1	8.6 ± 0.9	8.3 ± 1.0	0.68
SCUBE-1, ng/mL	11.7 ± 6.7★	8.7 ± 6.6	6.8 ± 4.3	<0.05

MPV: Mean platelet volume; SCUBE-1: Signal peptide-CUB-EGF domain containing protein-1 ★: p<0.05 compared to the control group.

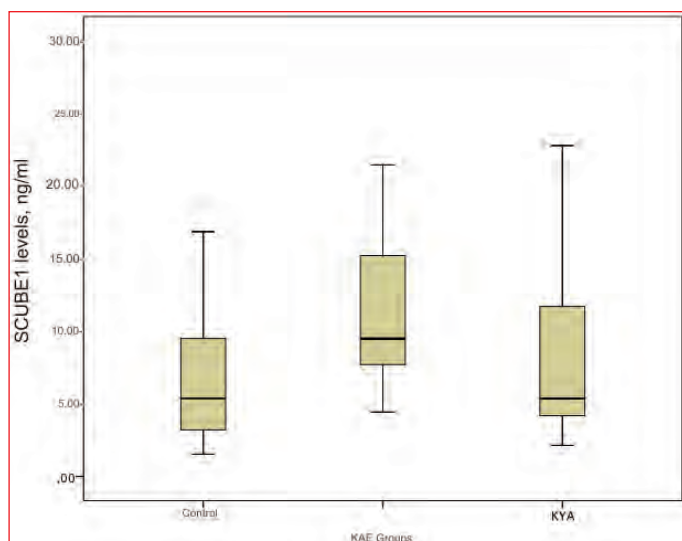


Figure 1. SCUBE-1 levels in CAE, CSF patients, and control group. SCUBE-1 levels are higher in patients with CAE compared to the other groups (p < 0.05).

and had CSF in at least one coronary artery according to the TIMI frame count method. The patients were randomized as 32 CSF patients with CAE (13 women, 19 men; mean age 60 ± 6) and 28 CSF patients without CAE (12 women, 16 men; mean age 58 ± 7). What's more, a control group of 27 people (19 women, 8 men; mean age 59 ± 10) who were similar in terms of age, gender and cardiac risk factors and whose coronary arteries and coronary blood flow were found to be normal, was formed for comparison. Routine laboratory parameters associated with the prothrombotic process and SCUBE-1 (Signal peptide-CUB-EGF domain-containing protein-1) levels were evaluated.

Results: The corrected TIMI (Thrombosis in myocardial infarction) frame count were significantly higher for all three coronary arteries in the patient groups (p<0.01). SCUBE-1 which is associated with cardiac enzyme levels (11.7 ± 6.7, 8.7 ± 6.6, and 6.8 ± 4.3 ng/mL, p<0.05), D-dimer and fibrinogen levels were significantly higher in the CSF and CAE group compared to the control group.

Conclusions: In our study, patients with both CAE and CSF had higher levels of cardiac enzymes, d-dimer, fibrinogen, and serum SCUBE-1. Therefore, we believe that platelet activation and a prothrombotic state are more pronounced in patients with CSF and CAE.

Interventional Cardiology / Coronary

PP-034

Early term outcomes of drug-coated balloons for small and diffuse coronary vessels

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Background and Aim: Small vessel size, diffuse coronary lesions especially in presence of diabetes mellitus are independent predictors for stent restenosis. Such patients still have a challenging for drug eluting stenting with an increase in long-term adverse events. In recent years, drug-coated balloons (DCB) have been introduced as promising alternative device to prevent such potential stent restenosis cases. We have also evaluated the patients underwent DCB in terms of early term outcomes in our clinic.

Methods: This prospective study included 35 of 60 patients underwent percutaneous coronary intervention with DCB from September 2023 to March 2024 in Medicana International Ankara Hospital cardiology clinic. The study patients (35 patients) had de novo lesions including small coronary artery size (<3 mm), and long lesions (>20 mm). The principles of Declaration of Helsinki were followed in this study. We followed these patients early term at 6 months. Optimal lesion preparation was achieved using balloons with a balloon-to-vessel ratio of 0.8-1:1. DCB were inflated to their nominal pressure for at least 90 seconds, taking care to extend the DCB at least 2-3 mm beyond the predilation balloon length. DCB were coated with sirolimus combined with either phospholipids (Magic Touch; Concept Medical, Gujarat, India) or biodegradable polymer (Selution; Med Alliance, Nyon, Switzerland) or paclitaxel coated (Agent; Boston Scientific, Marlborough, Massachusetts) as a carrier for the drug. After lesion preparation, bailout stenting was performed in the presence of residual flow-limiting dissections or residual stenosis >30%. Then, we repeated control angiography after 4 minutes in terms of presence of recoil, residual stenosis, dissections.

Results: The patients had de novo lesions including small coronary artery size (<3 mm), and long lesions (>20 mm). Mean age was 59.7 ± 11.3 years, 81.8% of patients were male, and 52.6% were diabetic. Mean vessels diameters were 2.61 ± 3.2 mm, lesion lengths were 34 ± 11.4 mm. The patients underwent DCB as sirolimus combined with phospholipids in the 20 patient, sirolimus biodegradable polymer in the 8 patients, and paclitaxel coated in the 7 patients. We performed DCB interventions in left anterior descending artery (12 patients), diagonal branch (4 patients), circumflex artery (12 patients), right coronary artery (7 patients). Two patients underwent the hybrid approach of combining DCB with drug eluting stents in the LAD vessel. Procedural success was 98%. One patient underwent bailout stenting procedure due to flow limiting dissection. Procedural complications was low. Residual dissections developed in 5 patients, which were no flow limiting. We not found clinic ischemic case abnormality at 6 months.

Conclusions: We observed that DCB interventions appeared effective and safely in small size and long-diffuse coronary lesions in the early term.

Interventional Cardiology / Coronary

PP-036

In coronary angiography, transradial versus transfemoral access: What are patients' perspectives?

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Background and Aim: Current guidelines in percutaneous coronary interventions recommend using radial access (TRA) over femoral access (TFA). TRA is associated with reduced major bleeding, early mobilization, and patient comfort. This study aims to compare patient satisfaction and complications between the two methods to determine the most ideal access route in daily practice.

Methods: A total of 152 patients who presented to the Dokuz Eylül University and underwent coronary angiography (CAG) between February 1 and June 30, 2024, were included in the study. The choice of access site was determined by the operator. Patients were divided into two groups based on whether they underwent TRA or TFA. A satisfaction survey was administered 24 hours post-procedure, and patients were monitored for complications over a one-month period. The primary endpoint was defined as patient satisfaction. Complications were categorized as minor bleeding (BARC 1-2), major bleeding (BARC 3-5), pseudoaneurysm, hematoma, and spasm.

Results: Of the 152 patients included in the study, 50 (33%) underwent TRA, and 102 (67%) underwent TFA. Minor bleeding occurred in 24 (16%) patients, and major bleeding occurred in 3 (0.02%) patients. There were no significant

differences between the groups in terms of gender, comorbidities, body mass index (BMI), antiplatelet use and complications. Survey results showed no difference between the groups in pre-procedure anxiety, satisfaction with CAG via the access route, or awareness of TRA. However, post-procedure pain was reported more frequently in the TRA group [23 (46%) vs. 16 (15%), $p < 0.001$]. SBP was also higher in this group (126 ± 13 vs. 124 ± 16 , $p = 0.033$). Pre-procedure anxiety was more common in women [27 (46%) vs. 18 (19%), $p < 0.001$]. There was no difference in the choice of access site [21 (42%) vs. 52 (50%), $p = 0.29$] and minor ($p = 0.12$) or major bleeding ($p = 0.51$) in elderly patients (≥ 65 years). In obese patients (BMI ≥ 30), there was no difference in minor ($p = 0.75$) or major bleeding ($p = 0.28$) or pseudoaneurysm ($p = 0.21$), but obesity was more common in female patients ($p = 0.028$).

Conclusions: Current data advocate for "radial first", but our study found no difference in satisfaction between patients undergoing TRA and TFA. Personal experiences of patients may influence this outcome. Previous studies show patients who underwent TFA prefer it for subsequent procedures, while those with experience in both TRA and TFA prefer TRA. Increased use of closure devices and appropriate compression may improve femoral experience, while smaller radial arteries and a propensity for spasms in female patients may lead to higher anxiety and postoperative pain with TRA. The learning curve of radial access may also contribute to these results. In summary, a joint decision by the operator and patient on vascular access appears to be the most ideal approach. Other factors potentially influencing patient satisfaction should be investigated.

Table 1. Demographic, clinical, and laboratory data of patients included in the study

	(n:152)
TRA, n(%)	50
Patients experiencing preoperative anxiety, n(%)	45
Patients experiencing moderate to severe perioperative pain, n(%)	19
Patients experiencing postoperative pain, n(%)	39
Satisfaction with CAG, n(%)	146
Patients recommending CAG from the site of the procedure, n(%)	147
Awareness of TRA, n(%)	102
ASA users, n(%)	61
Clopidogrel users, n(%)	21
OAK users, n(%)	7
Patients undergoing PCI, n(%)	44
Minor bleeding, n(%)	24
Major bleeding, n(%)	3
Pseudoaneurysm, n(%)	4
Hematoma, n(%)	10
Spasm, n(%)	3
Age, years	63 ± 11
Systolic Blood Pressure (SBP), mmHg	125 ± 15
Diastolic Blood Pressure (DBP), mmHg	74 ± 10
BMI, kg/m ²	$23 (17-24)$
LVEF, %	$57 (50-60)$
Creatine, mg/dl	$0.85 (0.67-1)$
Hemoglobin, g/dl	$12.5 (10.8-14.2)$
PLT, 10 ³ /UL	$254 (203-304)$

Table 2. Comparison of patients undergoing TRA versus TFA

	PATIENTS UNDERGOING		P VALUE
	TRA n:50	PATIENTS UNDERGOING TFA n:102	
Gender, Female, n(%)	18 (36)	40 (39)	0.70
Hypertension, n(%)	31	71	0.34
Diabetes, n(%)	19	42	0.70
Age, years	61.7±12	64 ±10	0.28
SBP, mmHg	126±13	124±16	0.03
DBP, mmHg	75±11	73±10	0.22
Heart Rate, v/dk	71±10	71±12	0.26
PP, 10 ³ /dL	255±77	271±101	0.24
BMI, kg/m ²	24 (17–29)	23 (17–27)	0.98
LVEF, %	40 (30–60)	35 (15–60)	0.079
Creatinin, mg/dl	0.83 (0.74–1)	0.86 (0.66–1)	0.72
Hemoglobin, g/dl	12.5 (11.4–14.4)	12.6 (10.3–14)	0.51
ASA users, n(%)	13 (30)	46 (45)	0.074
Clonidogrel users, n(%)	3 (6)	18 (18)	0.05
Patients experiencing preoperative anxiety, n(%)	16	29	0.65
Patients experiencing moderate to severe perioperative pain, n(%)	9	10	0.15
Patients experiencing postoperative pain, n(%)	23	16	<0.001
Satisfaction with CAG, n(%)	48	98	0.98
Patients recommending CAG from the site of the procedure, n(%)	48	99	0.73
Awareness of TRA, n(%)	38	64	0.10
Patients undergoing PCI, n(%)	19	25	0.8
Minor bleeding, n(%)	6	18	0.37
Major bleeding, n(%)	1	2	0.98
Pseudoaneurysm, n(%)	1	3	0.73
Hematoma, n(%)	2	8	0.36

Table 3. Comparison of survey results of female and male patients

	FEMALE PATIENTS		P VALUE
	n:58	MALE PATIENTS n:94	
Patients experiencing preoperative anxiety, n(%)	27	18	<0.001
Patients experiencing postoperative pain, n(%)	19	20	0.11
Awareness of TRA, n(%)	43	59	0.14

Interventional Cardiology / Coronary

PP-037

Myocardial infarction after blunt chest trauma: A case report

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Background and Aim: After blunt trauma to the chest, patients may present with various cardiac pathologies such as myocardial contusion, left ventricular rupture, pericardial effusion/pericarditis, valve damage, aortic dissection and coronary artery damage. Although post-traumatic chest pain is generally thought to be due to chest wall injury, other pathologies must be excluded. Taking a detailed history from the patient, observing an ECG, and evaluating with echocardiography are our most important diagnostic tools.

Methods: We present a case who was admitted to the emergency department with anterior myocardial infarction after a motor vehicle accident.

Case Report: A 31-year-old man admitted to a hospital after a blunt chest trauma due to a motorcycle accident. No pathological findings were found in physical examination or CT scans. When he was monitoring he described tightening chest pain radiating to his left arm. He has no history of any illnesses or use of medications. The only cardiovascular risk factor he had was smoking. His ECG featured anterior wall MI (Figure 1). His heart rate of 95 bpm blood pressure at 135/85 mmHg. Transthoracic echocardiography showed severe hypokinesis of the septum and the anterior wall, ejection fractions approximately 30%. No pericardial effusion or pathology in ascending aorta was observed. While we preparing patient for angiography, he developed VF. He was defibrillated, and

was quickly taken for angiography. Coronary angiography showed complete occlusion and dense thrombus of the left anterior descending coronary artery (LAD) (Figure 2). No coronary artery disease was observed in his other arteries. A drug induced stent implanted the lesions, intracoronary tirofiban was administered and TIMI 3 flow was achieved (Figure 3). The procedure was completed without complications.

Conclusions: In cases of chest pain that develops after chest trauma, we must definitely suspect injuries that may develop in coronary arteries or other heart structures (aorta, myocardium). For this reason, one of the most important tools in diagnosis is echocardiography. Before making a diagnosis, pericardial effusion and ascendant aorta must be checked and pathologies such as aortic dissection or free wall rupture must be excluded. Although the LAD is the most commonly injured artery in most cases, cases of injuries to other coronary arteries have also been reported. When looking at the pathophysiology of myocardial infarction, it is seen that damage and dissection develop in the arterial intima secondary to trauma, and subsequently intraluminal thrombosis develops. What needs to be done in its management is to perform coronary angiography and implanted a stent. Since such lesions contain dense thrombus, thrombus aspiration and/or intracoronary tirofiban application is another effective treatment method. In our patient, due to the dense thrombus, the local dissection line in the artery could not be clearly seen. However, since the other vessels were not atherosclerotic, we think that this pathophysiology is also related to our case.

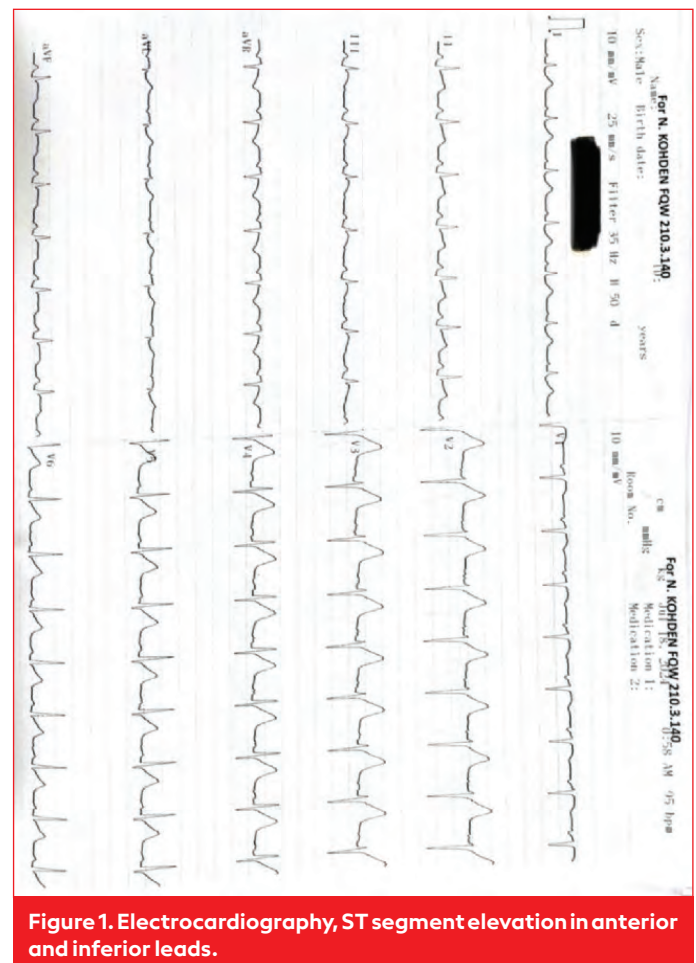
**Figure 1. Electrocardiography, ST segment elevation in anterior and inferior leads.**



Figure 2. Total occlusion of LAD from the ostial region.

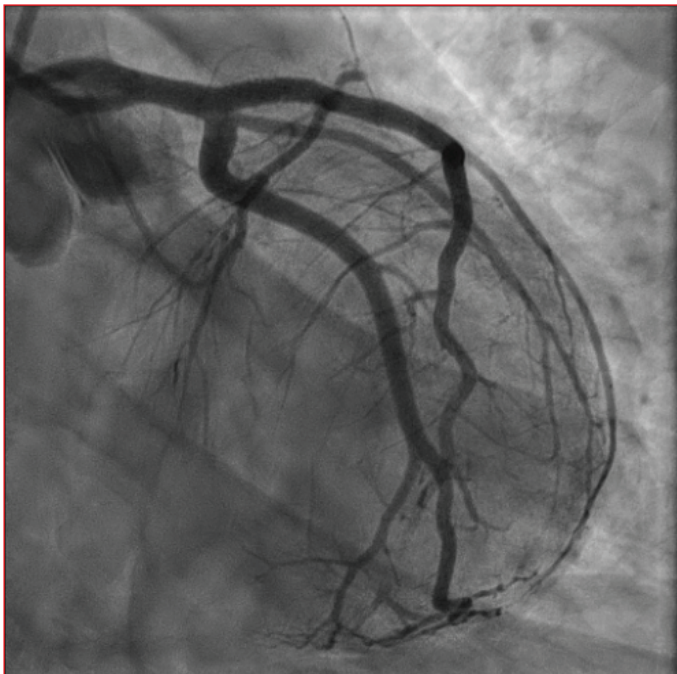


Figure 3. Flow provision after LAD percutaneous coronary intervention.

Interventional Cardiology / Coronary

PP-038

The relationship between FFR value and left ventricular mass index (LVMI) in patients with coronary artery disease

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Background and Aim: Percutaneous coronary interventions are the major treatment method for coronary artery disease (CAD). Although the degree of stenosis and plaque morphology in the coronary artery can be visually evaluated by angiography additional tests indicating ischemia are needed to make an optimal intervention decision in moderate lumen stenosis (50-70%). Fractional Flow Reserve (FFR) is one of the basic tests we use to show myocardial ischemia. However, many factors and clinical conditions, such as left ventricular hypertrophy (LVH), and the presence of multiple and consecutive lesions, can affect the results and reliability of this test. In this study, we aimed to investigate the relationship between FFR results and left ventricular mass index (LVMI) in patients who underwent FFR due to intermediate coronary artery stenosis.

Methods: A total of 668 patients [age 65 (IQR: 58-72) years; 73.7% male] who underwent FFR due to intermediate coronary artery stenosis were included in the study. The patients were divided into two groups FFR >80 and FFR ≤80. The FFR results, echocardiography and laboratory findings of the patients were analyzed. There was no significant difference between the groups in terms of clinical and demographic characteristics other than male gender ($p=0.02$), LVH ($p<0.001$) and smoking ($p=0.03$). In the FFR ≤80 groups, LV mass, LVMI, Relative wall thickness (RWT) and syntax score were statistically significantly higher, while eGFR was statistically significantly lower. FFR values showed a significant negative correlation with LVMI ($r=-0.245$, $p<0.001$) and syntax score ($r=-0.344$, $p<0.001$).

Results: In univariate analysis, male gender, LV mass, LVMI, syntax score, smoking, LVH, RWT, and eGFR were found associated with FFR ≤80. In multivariate analysis, LVMI ($p<0.001$), syntax score ($p<0.001$) and LVEF ($p=0.033$) are found independent predictors of FFR ≤80. Receiver operating characteristic analyses indicated a cut-off value of 95.52 g/m² for LVMI (AUC=0.628; 95% CI: 0.586–0.670; $p<0.001$) and a cut-off value of 10.75 for syntax score (AUC=0.632; 95% CI: 0.589–0.676; $p<0.001$) to best identify patients with FFR value ≤80.

Conclusions: LVMI and syntax scores are associated with FFR value ≤80 in patients with CAD. While interpreting the FFR results, it should be kept in mind that LVMI and syntax scores may have effects on the results.

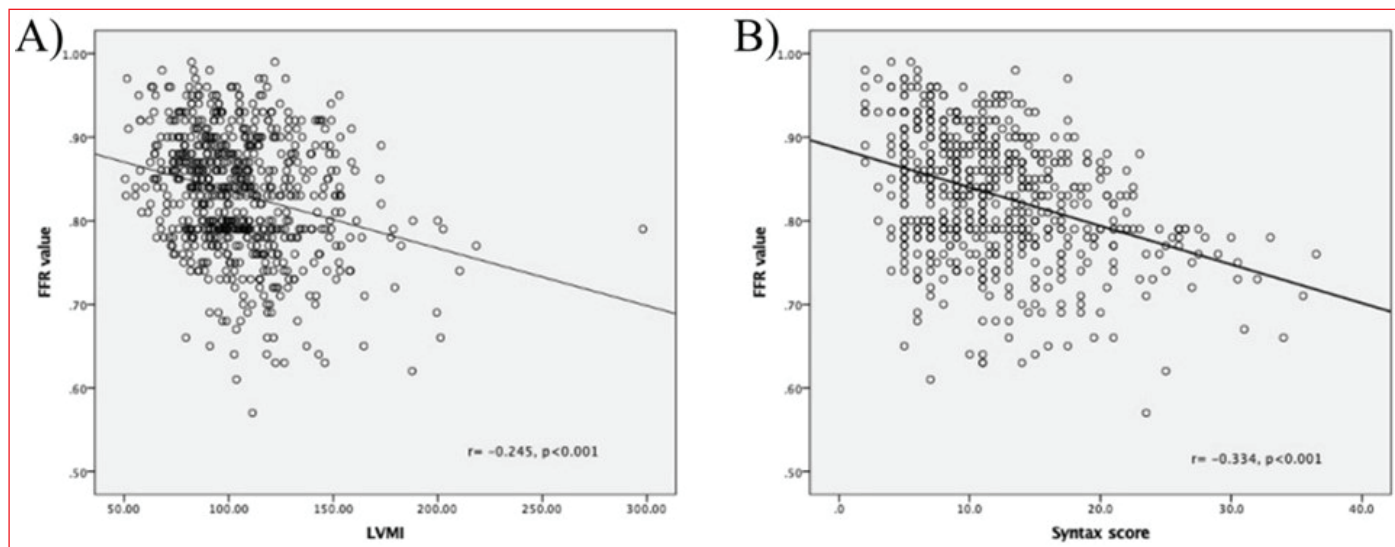
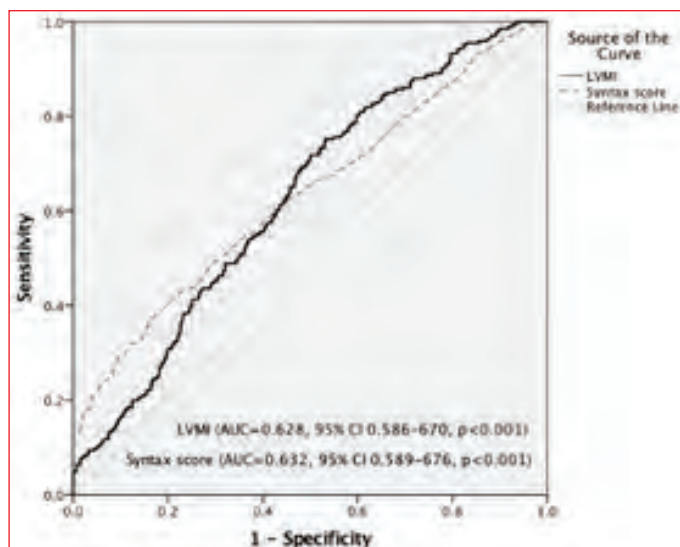


Figure 1. A) Plot of relationship between FFR values and left ventricular mass index (LVMI) B) Plot of relationship between FFR values and Syntax score.

Variables	All patients n=668	FFR >80 group n=402 (60.2%)	FFR ≤80 group n=266 (39.8%)	p value
Age, years	65 (58-72)	67 (60-73)	67 (58-74.5)	0.926
Gender (male), n (%)	492 (73.7)	283 (74)	209 (78.6)	0.02
History of CAD, n (%)	469 (70.2)	276 (68.7)	193 (72.6)	0.3
History of MI, n (%)	192 (28.7)	108 (26.9)	84 (31.6)	0.191
Hypertension, n (%)	492 (73.7)	293 (72.9)	199 (74.4)	0.592
Diabetes mellitus, n (%)	270 (40.4)	170 (42.3)	100 (37.6)	0.229
Dislipidemia n (%)	144 (21.6)	80 (19.9)	64 (24.1)	0.212
COPD, n (%)	93 (13.9)	58 (14.4)	35 (13.2)	0.732
Atrial Fibrillation, n (%)	60 (9)	40 (10)	20 (7.5)	0.334
Smoking, n (%)	151 (22.6)	79 (19.7)	72 (27.1)	0.03
LVH, n (%)	208 (31.1)	93 (23.1)	115 (43.2)	<0.001
Post-MI FFR, n (%)	70 (10.5)	34 (8.5)	36 (13.5)	0.39
FFR value	0.84 (0.79-0.89)	0.87 (0.84-0.91)	0.77 (0.73-0.79)	<0.001
LVEF, %	60 (52-60)	60 (55-60)	60 (55-60)	0.937
LV Mass, g	187.5 (158.8-217.9)	180.7 (152.5-207.1)	199.6 (170.2-227.7)	<0.001
LVMI, g/m ²	101.9 (87.1-120.3)	96.9 (83.3-114.9)	107.6 (94.7-124)	<0.001
RWT	0.43 (0.39-0.48)	0.42 (0.38-0.47)	0.44(0.40-0.47)	0.015
FFR applied lesion, n (%)				
LAD	593 (88.8)	349 (86.8)	244 (91.7)	0.107
CX	56 (8.4)	41 (10.2)	15 (5.6)	
RCA	19 (2.8)	12 (0.3)	7 (2.6)	
Syntax score	11 (7-15)	10 (7-13.5)	12 (8-17.5)	<0.001
eGFR, mL/min/1.73 mm ²	75.5 ± 27.8	69.9 ± 1.9	66.6 ± 2.4	0.034
Glucose, mg/dL	133 (110-184)	134 (110-192)	136 (114-190)	0.238
Hemoglobin, g/dL	14 (12.8-15.2)	13.7 (12.3-15.1)	13.8 (12.4-15.1)	0.738
Platelets, (x10 ³ /)	234 (196-279)	234 (198-277)	241 (202-279)	0.411
Total Cholesterol, mg/dL	192 (156-225)	198 (158-231)	192 (158-222)	0.093
HDL-C, mg/dL	42 (36-50)	43 (36-50)	42 (36-51)	0.697
LDL-C, mg/dL	119 (87.2-147.1)	122 (89-152)	117 (88-147)	0.111
Triglyceride, mg/dL	142 (103-211)	151 (109-220)	157 (108-224)	0.314

Table 2. Univariate and multivariate logistic regression analysis to detect the independent predictors of FFR value ≤ 80 in patients undergoing FFR procedure.

	Univariate Analysis			Multivariate Analysis		
	OR	(95% CI)	p value	OR	(95% CI)	p value
Age	1.000	0.985-1.015	0.962	0.989	0.969-1.008	0.262
Gender	1.542	1.073-2.216	0.019	0.934	0.621-1.405	0.743
Left ventricular Mass	1.010	1.007-1.014	<0.001			
LVMI	1.018	1.012-1.024	<0.001	1.018	1.010-1.025	<0.001
eGFR	0.994	0.988-0.999	0.033	0.997	0.990-1.005	0.448
Syntax score	1.111	1.078-1.145	<0.001	1.104	1.070-1.139	<0.001
Hypertension	1.105	0.776-1.574	0.580	0.969	0.643-1.462	0.881
LVEF	0.989	0.971-1.007	0.230	1.025	1.002-1.048	0.033
Diabetes Mellitus	0.822	0.599-1.129	0.226	0.829	0.584-1.177	0.295
History of MI	1.256	0.894-1.765	0.188			
Smoking	1.517	1.053-2.187	0.025	1.389	0.933-2.067	0.106
CAD	1.207	0.857-1.699	0.281			
LVH	0.395	0.283-0.553	<0.001			
RWT	15.672	1.432-171.511	0.024			

**Figure 2. Receiver operating characteristic graphic to detect the best cut-off value of LVMI and syntax score for FFR value ≤ 80 .**

Interventional Cardiology / Coronary

PP-039

Exploring the role of the fibrosis-4 index in patients with coronary slow flow

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Background and Aim: Coronary slow flow (CSF) is a clinically significant condition characterized by delayed progression of contrast material during coronary angiography in the

absence of significant stenosis (<40% obstruction) in the coronary arteries. This condition can cause chest pain during rest or exercise. The Fibrosis-4 (FIB-4) index is an effective, reliable, and non-invasive tool used to indicate liver fibrosis during the progression of certain chronic illnesses and infectious conditions. Recently, the FIB-4 index has been shown to have a relationship with concomitant coronary artery disease in patients with hepatic steatosis. In this study, we investigated the relationship between coronary slow flow and the Fibrosis-4 index.

Methods: A total of 87 patients were enrolled for this study. The distributional normality of the data was assessed. Depending on the normality of the data, either the T-test or the Mann-Whitney U test was employed to compare pairs of groups. Chi-square test was used to compare categorical variables. The FIB-4 index is calculated by multiplying the patient's age with the aspartate aminotransferase (AST) level and then dividing the result by the product of the platelet count and the square root of the alanine aminotransferase (ALT) level.

Results: The CSF group consisted of 24 patients, while the NCS group consisted of 63 patients. The groups were similar in terms of gender and comorbidities. The CSF group had higher values of red blood cell count (5.1 ± 0.5 vs. 4.8 ± 0.6 ; $p=0.024$), hemoglobin (15.3 ± 1.7 vs. 13.5 ± 1.7 ; $p<0.001$), hematocrit (45.1 ± 3.7 vs. 40.6 ± 4.5 ; $p<0.001$), MCV [87.9 ($86.0-92.6$) vs. 86 ($80.5-89.6$); $p=0.012$], and MCH (30.2 ± 2.2 vs. 28.5 ± 2.9 ; $p=0.010$) compared to the NCA group. The FIB-4 index was statistically similar between the groups [0.96 ($0.77-1.37$) for the CSF group and 1.00 ($0.80-1.79$) for the NCA group; $p=0.352$].

Conclusions: In our study, we found no relationship between the FIB-4 index and coronary slow flow. However, we observed an association between coronary slow flow and elevated levels of red blood cell count, hemoglobin, hematocrit, mean cell volume, and mean cell hemoglobin. This relationship may be related to increased viscosity due to higher cell count and volume.

Table 1. Demographic and laboratory parameters of the study groups

	Coronary Slow Flow Group (n=24)	Normal Coronary Arteries Group(n=63)	p
Age	51.6 +/- 11.0	54.1 +/- 13.2	0.425
Gender	21 (87.5%)	31 (49.2%)	0.001
Hypertension	9 (37.5%)	15 (23.8%)	0.749
Diabetes Mellitus	4 (16.7%)	13 (20.6%)	0.677
Heart Failure	0 (0%)	3 (4.8%)	0.277
Atrial Fibrillation	1 (4.2%)	7 (11.1%)	0.316
Fasting Blood (mg/dl)	98 (89.5 – 107.5)	101 (88 - 126)	0.575
Creatinine (mg/dl)	0.9 (0.7 – 1.0)	0.79 (0.68 – 0.91)	0.031
Total Cholesterol (mg/dl)	177 (158 - 209)	183 (159 - 219)	0.562
High Density Lipoprotein (mg/dl)	41 (35 - 49)	45 (39 - 53)	0.092
Low Density Lipoprotein (mg/dl)	105 (94 - 120)	109 (88 - 136)	0.981
Triglyceride (mg/dl)	140 (78 - 214)	121 (90 - 205)	0.848
Uric Acid (mg/dl)	5.9 (5.1 – 7.1)	5.2 (4.3 – 6.2)	0.100
Albumin (g/L)	43.6 (40.8 – 45.7)	42.5(39.8 – 44.8)	0.219
Aspartat Transaminase (IU/L)	20 (17 - 26)	22 (16 - 27)	0.750
Alanin Aminotransferase (u/L)	22 (18 - 30)	20 (15 - 27)	0.161
White Blood Count (10 ³ /uL)	7.5 (6.3 – 8.5)	7.4 (6.1 – 9.1)	0.864
Red Blood Count (10 ⁶ /uL)	5.1 +/- 0.5	4.8 +/- 0.6	0.024
Hemoglobin (g/dl)	15.3 +/- 1.7	13.5 +/- 1.70	<0.001
Hematocrite (%)	45.1 +/- 3.7	40.6 +/- 4.5	<0.001
Mean Corpuscular Volume (fL)	87.9 (86.0 – 92.6)	86 (80.5 – 89.6)	0.012
Mean Corpuscular Hemoglobin (pg)	30.2 +/- 2.2	28.5 +/- 2.9	0.010
Mean Corpuscular Hemoglobin Concentration (%)	33.8 +/- 1.6	33.3 +/- 1.9	0.214
Platelet (10 ³ /uL)	224.5 (197.3 – 274.7)	234 (195 - 278)	0.736
Red cell Distribution With (%)	12.1 (11.8 – 12.4)	12.4 (11.7 – 13.9)	0.143
Mean Platelet Volume (fL)	8.1 (6.7 – 9.1)	7.5 (6.7 – 8.4)	0.277
Neutrophile (10 ³ /uL)	4.3 (3.5 – 5.4)	4.6 (3.7 – 5.8)	0.393
Lymphocyte (10 ³ /uL)	2.3 (1.9 – 3.2))	2.1 (1.7 – 2.6)	0.069
FIB-4*	0.96 (0.77 – 1.37)	1.00 (0.80 – 1.79)	0.352

Abb: *Fibrosis-4 Index

Hypertension

PP-041

Association of blood pressure variability with contrast nephropathy in patients with STEMI undergoing primary percutaneous coronary intervention

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Background and Aim: Contrast-induced nephropathy (CIN), a common complication after primary percutaneous coronary intervention (PCI) in patients with ST-elevation myocardial infarction (STEMI), is associated with increased

short and long-term mortality, increased cardiovascular events and prolonged hospitalisation. Blood pressure variability (BPV), which refers to changes in blood pressure (BP) over time, has been shown to be associated with cardiovascular events, stroke, impaired renal function and target organ damage (TOD), independent of blood pressure level. The relationship of this variability with CIN is not known. In this study, we investigated the relationship between invasively measured short-term BPV and the development of CIN in haemodynamically stable patients with STEMI undergoing PCI.

Methods: Our study was designed as a single-centre, prospective, observational study. Between 2020 and 2023, 220 patients who presented to the emergency department of our hospital with ischaemic symptoms, were diagnosed with STEMI and underwent PCI were included in the study. The primary endpoint was development of CIN. Admission and 48-72 hour laboratory values, demographic

and clinical information, length of hospital stay, contrast volumes used, and clinical outcomes of the study group were recorded. In the coronary intensive care unit after percutaneous coronary intervention, intra-arterial blood pressure was monitored continuously for 6 hours through the existing femoral sheath and evaluated with short-term BPV; standard deviation (SD), average real variability (ARV) and delta parameters. Patients in the study were divided into two groups according to whether or not they developed CIN. Multivariate logistic regression analysis was used to determine the independent predictors associated with CIN.

Results: The mean age of the 220 patients included in the study was 60.82 ± 13.10 years and 49 (22.3%) of them were female. CIN developed in 20% of patients. Mean age, admission creatinine, peak troponin, length of hospital stay and contrast volume were higher in the group that developed CIN. In addition, left ventricular ejection fraction and glomerular filtration rate were lower in the same group. It was observed that systolic SD, systolic ARV and systolic delta were higher in the group that developed CIN. Multivariate logistic regression analysis revealed that systolic SD and systolic ARV parameters were independent predictors of the development of CIN.

Conclusions: In STEMI patients, short-term BPV measured intra-arterially was shown to be associated with the development of CIN. In particular, the finding that systolic SD and systolic ARV are independent predictors of CIN may have clinical importance in terms of early diagnosis and preventive interventions.

Table 1. Clinical characteristics and treatments between those with and without CIN

Comparison of clinical characteristics between groups with and without CIN				
	Overall (n=220)	CIN (+) (n=44)	CIN (-) (n=176)	P value
Age	60,82 ± 13,10	66,04 ± 12,40	59,51 ± 12,98	<0,01
Gender (female) n, %	49 (22,3)	8 (18,2)	41 (23,3)	0,46
BMI (kg/m ²)	28,15 ± 3,06	28,17 ± 3,04	28,15 ± 3,08	0,97
DM n, %	70 (31,8)	20 (45,5)	50 (28,4)	0,03
HT n, %	115 (52,3)	24 (54,5)	91 (51,7)	0,73
HL n, %	70 (31,8)	10 (22,7)	39 (22,2)	0,93
CKD n, %	12 (5,5)	5 (11,4)	7 (4)	0,054
CAD n, %	55 (25)	12 (27,3)	43 (24,4)	0,69
Smoke n, %	108 (49,1)	18 (40,9)	90 (51,1)	0,22
TREATMENT USED BEFORE HOSPITALISATION				
ASA n, %	87 (39,5)	22 (50)	65 (36,9)	0,11
BB n, %	57 (25,9)	14 (31,8)	43 (24,4)	0,31
ACE inh. n, %	103 (46,8)	22 (50)	81 (46)	0,63
CCB n, %	45 (20,5)	13 (29,5)	32 (18,1)	0,09
Statin n, %	60 (27,3)	15 (34,1)	45 (25,6)	0,25

n: Sample size; CIN: Contrast-induced nephropathy; BMI: Body mass index; DM: Diabetes mellitus; HT: Hypertension; HL: Hyperlipidemia; CKD: Chronic kidney disease; CAD: Coronary artery disease; ASA: Acetylsalicylic acid; BB: Beta blocker; ACE inh: Angiotensin converting enzyme inhibitor; CCB: Calcium channel blocker.

Table 2. Comparison of laboratory values between groups with and without CIN

Comparison of laboratory values between groups with and without CIN				
	Overall (n=220)	CIN (+) (n=44)	CIN (-) (n=176)	P value
Leukocyte (x 10 ³ /μL)	12,71 ± 4,51	13,34 ± 4,96	12,55 ± 4,39	0,295
haemoglobin (g/dL)	13,65 ± 1,72	13,75 ± 1,73	13,22 ± 1,65	0,081
Platelet (x 10 ³ /μL)	258,66 ± 77,90	238,29 ± 59,99	263,75 ± 81,11	0,062
Urea (mg/dL)	36,15 ± 16,21	41,71 ± 22,14	34,76 ± 14,10	0,01
Creatinine (mg/dL)	0,93 ± 0,37	1,09 ± 0,52	0,9 ± 0,32	<0,01
eGFR ml/dk/1.73m ²	85,32 ± 22,38	71,13 ± 23,46	88,95 ± 20,69	<0,01
Peak troponin(ng/mL)	3235,50 [6437,75]	5669 [6954,5]	2888 [5830]	<0,01
CRP (mg/L)	4,93 [9,42]	8,89 [14,14]	4,38 [7,33]	0,043
AST (IU/L)	51,5 [110,75]	94,5 [116,0]	44,5 [91,5]	0,044
ALT (IU/L)	26 [21]	30,5 [27,25]	25 [22]	0,195
Na (mEq/L)	135,24 ± 3,83	134,31 ± 4,32	135,47 ± 3,68	0,07
K (mEq/L)	4,15 ± 0,53	4,30 ± 0,54	4,11 ± 0,52	0,04
Additional Features				
Duration of hospitalisation (hours)	89 [53,75]	99 [75,75]	82,5 [53,5]	<0,01
LVEF %	44,46 ± 8,92	40,61 ± 9,18	45,43 ± 8,61	<0,01
Contrast volume (mL)	267,04 ± 106,16	301,13 ± 140,79	258,52 ± 94,17	0,017
Systolic BP (mmHg)	128,98 ± 16,2	128,56 ± 15,64	129,09 ± 16,38	0,848
Diastolic BP (mmHg)	72,34 ± 11,78	73,75 ± 12,04	71,99 ± 11,72	0,377

n: Sample size; CIN: Contrast-induced nephropathy; eGFR: Glomerular filtration rate; CRP: C reactive protein; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; Na: Sodium; K: Potassium; LVEF: Left ventricular ejection fraction; BP: Blood pressure.

Table 3. Blood pressure variability values in the group with and without CIN

	Overall (n=220)	CIN (+) (n=44)	CIN (-) (n=176)	P value
	Mean ± SD	Mean ± SD	Mean ± SD	
sySD	10,11 ± 6,33	12,15 ± 8,62	9,61 ± 5,53	0,017
diSD	7,20 ± 3,70	7,51 ± 3,53	7,12 ± 3,75	0,536
syARV	8,31 ± 4,78	10,05 ± 6,06	7,87 ± 4,32	<0,01
diARV	6,06 ± 3,44	7,01 ± 3,92	5,83 ± 3,28	0,041
syDELTA	29,22 ± 18,88	35,02 ± 26,20	27,77 ± 16,33	0,02
diDELTA	20,83 ± 10,86	22,34 ± 10,88	20,45 ± 10,85	0,304

n: Sample size; BPV: Blood pressure variability; CIN: Contrast-induced nephropathy; SD: Standard deviation; sySD: Systolic standard deviation; diSD: Diastolic standard deviation; syARV: Systolic average real variability; diARV: Diastolic average real variability; syDELTA: Systolic delta value; diDELTA: Diastolic delta value.

Table 4. Multivariate logistic regression analyses of sySD and syARV

Multivariate logistic regression analysis to identify independent determinants of CIN

(sySD model)			
Variable	OR	%95 CI	P value
sySD	1,055	1,003 - 1,110	0,04
Age	1,036	1,006 - 1,067	0,02
DM	1,500	0,695 - 3,237	0,302
Creatinine	2,279	0,961 - 5,407	0,062
Peak troponin	1,000	1,000 - 1,000	0,188
Contrast volume	1,005	1,002 - 1,008	<0,01
LVEF	0,952	0,911 - 0,995	0,029

CIN: Contrast-induced nephropathy; sySD: Systolic standard deviation; OR: Odds ratio; CI: confidence interval; DM: Diabetes mellitus; LVEF: Left ventricular ejection fraction.

Multivariate logistic regression analysis to identify independent determinants of CIN

(syARV model)			
Variable	OR	%95 CI	P value
syARV	1,084	1,011 - 1,162	0,024
Age	1,036	1,005 - 1,067	0,021
DM	1,498	0,692 - 3,239	0,305
Creatinine	2,160	0,913 - 5,110	0,079
Peak troponin	1000	1000 - 1000	0,213
Contrast volume	1,005	1,001 - 1,008	<0,01
LVEF	0,955	0,913 - 0,998	0,04

CIN: Contrast-induced nephropathy; syARV: Systolic average real variability; OR: Odds ratio; CI: confidence interval; DM: Diabetes mellitus; LVEF: Left ventricular ejection fraction.

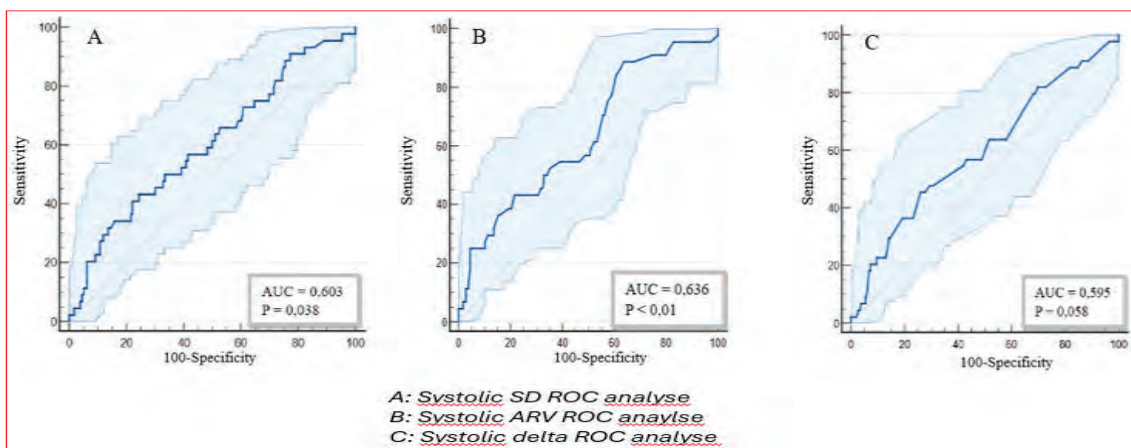


Figure 1. ROC analyses of sySD syARV and sydelta.

Hypertension

PP-042

Relationship between total body water percentage and dipper/non-dipper blood pressure

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Background and Aim: The aim of this study is to investigate the relationship between the total body water percentage calculated with the TANITA device and 24-hour blood pressure (BP) changes (dipper/non-dipper).

Methods: 129 acute coronary syndrome patients were included in our study. After clinical stabilization was achieved during the patients' intensive care hospitalization, body water amount parameters were calculated with the TANITA BC-601 device and hourly BP monitoring was performed for 24 hours using the automatic BP measurement device. A

decrease of less than 10% in the mean systolic BP measured at night compared to the daytime was defined as non-dipper. The patients were divided into two groups as dipper and non-dipper BP and statistical analysis was performed.

Results: When dipper (41 patients; age=59.88 ± 11.23 years) and non-dipper (88 patients; age=61.5 ± 11.42 years) groups were compared, acute subendocardial myocardial infarction (NSTEMI; p=0.015), left ventricular ejection fraction (LV-EF; p=0.009), left ventricular diastolic (LV-EDD; p=0.016) and systolic (LV-ESD; p=0.014) diameters and total body water percentage (p<0.001) were found to be statistically different. Regression analysis was performed to determine the predictors of non-dipper BP. In univariable regression analysis, NSTEMI (p=0.013), LV-EF (p=0.012), LV-EDD (p=0.019), LV-ESD (p=0.016) and total water percentage (p<0.001) were found to be dependent predictors; in multivariable regression analysis, only total water percentage (OR: 1.356, 95% CI: 1.205-1524, p<0.001) was found to be an independent predictor of non-dipper BP.

Conclusions: In this study, we found a close relationship between non-dipper pattern BP, which is closely related to cardiovascular mortality, and body water percentage. In this way, we indicate the importance of a simple water factor in protection from cardiovascular patients.

Table 1. Clinical characteristics, laboratory and echocardiographic parameters of the study population

	Dipper (n=41)	Non-dipper (n=88)	p value
Age (years)	59.88 ± 11.23	61.5 ± 11.42	0.452
Gender (F/M) (n)	(11/30)	(20/68)	0.661
ACS (NSTEMI/STEMI) (n)	(7/34)	(35/53)	0.015
Hypertension n (%)	20 (49)	43 (49)	0.999
Diabetes Mellitus n (%)	10 (24)	25 (28)	0.677
Furosemid/HTZ n (%)	8 (19)	13 (15)	0.609
MRA n (%)	1 (2)	2 (2)	0.520
Hemoglobin (g/dL)	13.96 ± 1.75	14.08 ± 1.74	0.729
Creatine (mg/dL)	0.94 (0.34-1.7)	0.88 (0.63-1.8)	0.532
GFR	84 (34-114)	89.3 (32.9-118)	0.903
Total Protein (g/L)	71.3 (54.7-86.7)	69.5 (30.6-79.3)	0.124
Albumin (g/dL)	39.5 (31.5-46.6)	40.3 (27-47)	0.646
LV-EF (%)	55 (35-65)	52.5 (25-65)	0.009
LV-EDC (mm)	45.8 ± 4.47	48.1 ± 4.77	0.016
LV-ESD (mm)	30.63 ± 5.89	33.42 ± 5.83	0.014
LA (mm)	43.92 ± 4.72	37.49 ± 4.54	0.215
E wave (cm/s)	67.37 ± 22.43	62.15 ± 19.32	0.203
A wave (cm/s)	68.92 ± 14.39	69.95 ± 17.87	0.828
Total Water Weight (kg)	53.66 ± 6.24	50.23 ± 5.56	0.206
Total Water Percentage (%)	59.34 ± 5.24	50.54 ± 6.36	<0.001

Table 2. Univariable and multivariable regression analysis showing the relationship non-dipper pattern and parameters

Variables	Univariable			Multivariable		
	OR	95% CI	p	OR	95% CI	p
ACS (NSTEMI/STEMI)	0.312	0.124-0.781	0.013	1.019	0.294-3.531	0.977
LV-EF	1.065	1.014-1.118	0.012	1.084	0.992-1.184	0.074
LV-EDD	0.901	0.825-0.983	0.019	0.789	0.587-1.062	0.118
LV-ESD	0.918	0.856-0.984	0.016	1.134	0.873-1.473	0.346
Total Water Percentage	1.275	1.167-1.392	<0.001	1.356	1.205-1.524	<0.001

Heart Valve Diseases

PP-044

Improvement in atrial functional and ventricular functional tricuspid regurgitation following catheter ablation or cardioversion of atrial fibrillation

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Background and Aim: The majority of cases of significant tricuspid regurgitation (TR) are secondary to tricuspid annular dilation and leaflet tethering in the context of RV remodeling due to pressure or volume overload, as seen in patients with

pulmonary hypertension (primary or secondary to left-sided heart disease) or dilated cardiomyopathies. Secondary TR induced by tricuspid valve annular dilatation secondary to right atrial dilatation is referred to as atrial functional TR, while secondary TR caused by RV remodeling is referred to as ventricular functional TR. In current guidelines, GDMT (Guideline-Directed Medical Therapy) is effective for secondary TR attributable to HF with reduced LVEF. Normal sinus rhythm should be restored for secondary TR caused by AF-related annular dilatation. In patients with ventricular functional TR, however, AF may also accompany the clinical condition. It is unknown whether restoring sinus rhythm will reduce tricuspid regurgitation in these patients. The objective of this study was to investigate the degree of tricuspid regurgitation change in patients with atrial and ventricular functional tricuspid regurgitation after sinus rhythm restoration.

Methods: A retrospective cohort study of patients undergoing AF ablation and cardioversion at a single center between 2019 and 2023 was performed. Patients with at least grade 1

TR on echocardiography and a baseline echocardiogram and a follow-up echocardiogram after ablation were included. A-FTR and V-FTR were defined according to the latest ACC/AHA guidelines. According to the latest guidelines, patients with mild to severe FTR were classified as A-FTR if they had atrial fibrillation, left ventricular ejection fraction >60%, pulmonary artery systolic pressure (PASP) <50 mmHg, no left-sided valve disease, and normal-appearing tricuspid leaflets. FTR patients who did not meet at least one of these three criteria were defined as ventricular FTR (V-FTR).

Results: A total of 88 patients were enrolled. The prevalence of A-FTR in our cohort was 62% (53 patients), and 38% of the patients were in the V-FTR group. There was no significant difference between the A-FTR and V-FTR groups regarding age, gender, and NYHA class. 21 of the patients underwent DCCV, and 67 underwent catheter ablation. The prevalence of severe FTR was similar (17% in A-FTR vs. 8.6% in V-FTR, p=0.205). There was no difference in preintervention TR grade between the two groups. TR severity improved significantly from baseline to follow-up in both V-FTR and A-FTR patients. These outcomes were observed in patients who underwent both DCCV and catheter ablation.

Conclusions: The degree of tricuspid regurgitation is reduced with sinus rhythm restoration in patients with both ventricular functional and atrial functional TR.

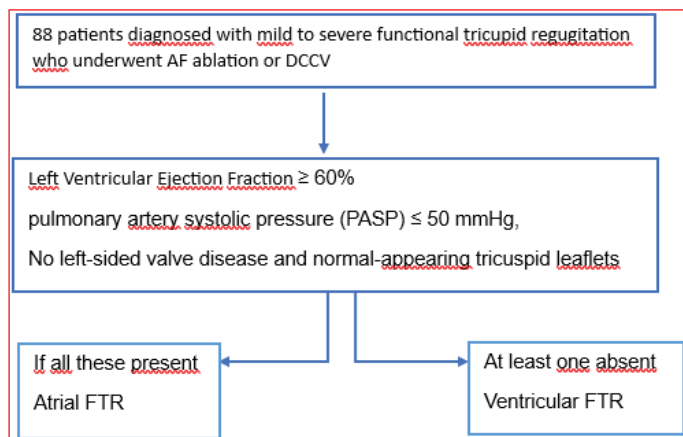


Figure 1. Study groups.

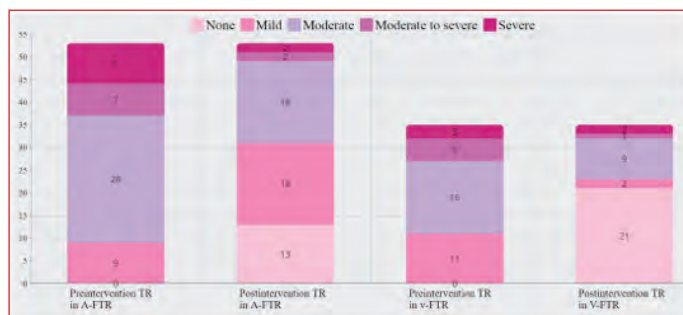


Figure 2. Figure 2 shows changes in the severity of TR from baseline to after sinus rhythm restoration in groups.

Table 1. Table 2, shows the pre-intervention TR grades of the patients in the horizontal row and the post-intervention TR grades in the vertical row.

			Postintervention TR					P Value
			None-Trivial	Mild	Moderate	Moderate-Severe	Severe	
A-FTR	Preintervention TR	Mild	7	2	0	0	0	<0.001†
		Moderate	5	12	9	0	2	
		Moderate-Severe	1	2	4	0	0	
		Severe	0	2	5	2	0	
V-FTR	Preintervention TR	Mild	9	1	1	0	0	<0.001†
		Moderate	10	1	4	0	1	
		Moderate-Severe	2	0	3	0	0	
		Severe	0	0	1	1	1	

† Marginal Homogeneity Test

Heart Valve Diseases

PP-045

Regression of mitral insufficiency due to marantic endocarditis in a patient with systemic lupus erythematosus after use of anticoagulants

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Background and Aim: Cardiac involvement can occur in Systemic Lupus Erythematosus (SLE). Among valve involvements, mitral valve regurgitation is one of the most common findings. The observed abnormalities may be due to atypical verrucous endocarditis. Additionally, valvulitis, fibrosis, and mucoid degeneration in these structures have been identified as significant factors in the etiology. I will discuss a case of mitral insufficiency associated with valve involvement in a patient diagnosed with lupus, and the improvement observed following anticoagulant therapy and monitoring.

Methods: A 25-year-old female patient with a diagnosis of systemic lupus erythematosus (SLE) presented to the emergency department with dyspnea. Echocardiographic examination revealed moderate-severe mitral insufficiency. A regular, hypoechoic vegetation was observed at the tips of the mitral valve. Transesophageal echocardiography (TEE) identified 5 mm and 4 mm vegetations on the posterior and anterior leaflets of the mitral valve, respectively. Given that the patient's infectious parameters were negative and there was no fever, marantic endocarditis was considered. Evaluated by the cardiovascular surgery-cardiology council, the patient was planned for follow-up with warfarin therapy, targeting an INR range of 2-3 for one month. After one month of warfarin treatment, follow-up echocardiography showed no vegetation on the mitral valve. The severity of mitral regurgitation was assessed as mild. Due to the improvement in symptoms and the reduction in both the appearance and severity of the mitral valve vegetation, continued monitoring with anticoagulation therapy was deemed appropriate, and the need for valve surgery was no longer indicated.

Results: Mitral insufficiency can be observed in patients diagnosed with systemic lupus erythematosus (SLE) due to valve involvement. In cases where the severity of the insuffi-

ciency might necessitate surgical intervention, a reduction in the severity of the valve insufficiency may be observed following treatment with anticoagulants and subsequent monitoring. The need for valve surgery may be eliminated.

Conclusions: Systemic lupus erythematosus (SLE) is one of the most well-known systemic inflammatory diseases. In SLE, all cardiac structures can be affected, and venous thromboembolic complications may develop. Valve disease in SLE is classified as either Libman-Sacks endocarditis or nonbacterial thrombotic endocarditis. Libman-Sacks endocarditis is characterized by non-infectious vegetations and is the most distinctive valve lesion associated with SLE. While any valve may be involved in SLE, the mitral valve is the most frequently affected. These valve lesions have been associated with increased morbidity and mortality in patients with SLE. Depending on the severity of the condition, valve surgery may be required in some cases. However, after anticoagulant therapy, the need for surgical intervention may be eliminated.

Heart Valve Diseases

PP-047

The relationship between rheumatic valvular heart disease and serum lipoprotein(a) levels

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Background and Aim: Rheumatic valvular heart disease (RVHD) is a significant consequence of rheumatic fever, a complication of untreated streptococcal throat infections. It primarily affects the heart valves, leading to conditions such as mitral stenosis, aortic regurgitation, and others. While the pathophysiology of RVHD has traditionally been linked to immune-mediated damage following streptococcal infection, recent research has illuminated the role of various biochemical markers in its development and progression. One such marker is lipoprotein(a) [Lp(a)], a lipid particle that has gained attention for its potential role in cardiovascular diseases. Lp(a) is structurally similar to low-density lipoprotein (LDL) but contains an additional protein component called apolipoprotein(a). Elevated levels of Lp(a) have been associated with an increased risk of atherosclerosis and calcific aortic valvular disease, suggesting a potential link to valvular heart disease. We aimed to elucidate the relationship between the serum Lp(a) levels and RVHD.

Methods: In this cross-sectional study, we included patients with rheumatic valvular heart disease and healthy patients. We performed serum Lp(a) analysis in all patients. We analyzed the demographic, echocardiographic parameters and analyzed the relationship between the serum Lp(a) concentrations and echocardiographic parameters.

Results: We included 33 patients diagnosed with rheumatic valvular disease and 33 control patients. Mean age of the patient population was 50 ± 12 years, and 24 (73%) were female. Hypertension and diabetes mellitus were similar

between groups. Lp(a) was higher in the RVHD group than in the control group (31 ± 26 mg/dL). Serum Lp(a) positively correlated with left atrial diameter ($\rho=0.479$; $p=0.005$), estimated pulmonary artery systolic pressure ($\rho=0.419$; $p=0.024$), Wilkins score ($\rho=0.417$; $p=0.020$) and negatively correlated with mitral valve area ($\rho=-0.361$; $p=0.039$).

Conclusions: Serum Lp(a) concentrations were higher in patients with the rheumatic valvular heart disease than in healthy controls, and its concentration positively correlated with left atrial diameter, Wilkins score, estimated pulmonary artery systolic pressure and negatively correlated with mitral valve area.

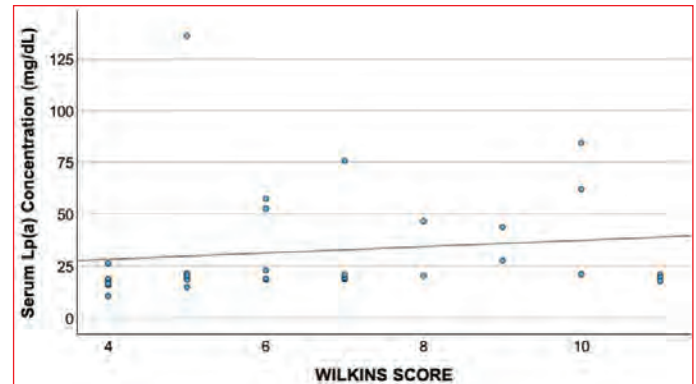


Figure 1.

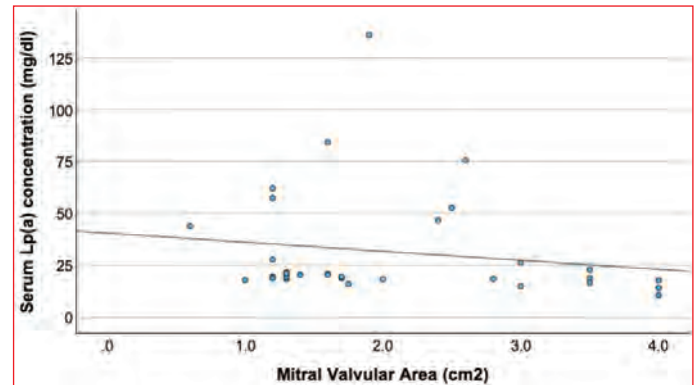


Figure 2.

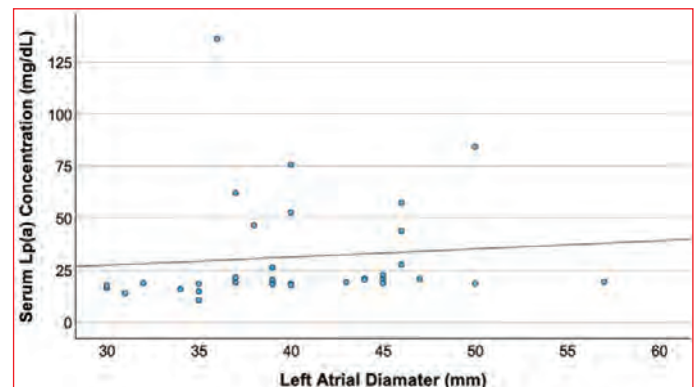


Figure 3.

Table 1. The comparative analysis of demographics and comorbidities of the study population

	All patients (n=33)	RVHD group (n=33)	Control group (n=33)	p value
Age, years	50 ± 11	51 ± 12	49 ± 11	0.531
Female sex	47 (71)	24 (73)	23 (70)	0.786
Body mass index, kg/m ²	28.8 ± 5.3	28.3 ± 5.3	29.3 ± 5.4	0.491
Hypertension	22 (34)	8 (24)	14 (45)	0.078
Diabetes mellitus	7 (11)	5 (15)	2 (7)	0.265
Serum lp(a) concentration, mg/dL	26 ± 20	31 ± 26	20 ± 5	0.016

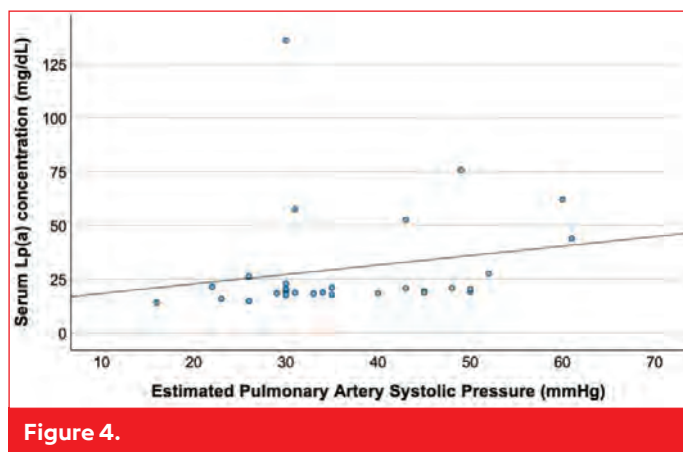


Figure 4.

Table 2. The echocardiographic features of the patients with the rheumatic valvular heart disease

	Value
Left ventricular ejection fraction, %	60 (55-65)
Left atrial diameter	42 (39-45)
Mitral valve area, cm ²	1.4 (1.3-2.0)
Wilkins score	6 (5-9)
Mitral regurgitation	
None	2 (3)
+1	14 (42)
+2	10 (30)
+3	4 (12)
+4	3 (9)
Aortic regurgitation	
None	16 (49)
+1	10 (30)
+2	6 (18)
+3	1 (3)
+4	0 (0)
Aortic stenosis	
None	30 (46)
Mild	0 (0)
Moderate	2 (6)
Severe	1 (3)
Tricuspid regurgitation	
None	8 (24)
Mild	15 (46)
Moderate	9 (27)
Severe	1 (3)
Estimated pulmonary artery systolic pressure, mmHg	31 (28-42)

Heart Valve Diseases

PP-048

Evolving language in heart valve disease research: A computational analysis of terminological trends

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Background and Aim: Recent advances in heart valve disease research have highlighted the increasing prevalence and complexity of valve pathologies, particularly underlining the impact of aging populations. This has spurred an increased need for comprehensive data analysis to understand trends in medical terminology and the dissemination of knowledge within the field. The emergence of large-scale data analysis tools, including natural language processing capabilities like ChatGPT, has further transformed our approach to mining and interpreting vast amounts of scientific literature. By leveraging these advancements, researchers are now better equipped to track changes in the discourse surrounding heart valve diseases, providing insights into how scientific communication evolves in response to new discoveries and technologies.

Methods: PubMed API was utilized to extract all abstracts containing the phrase "heart valve". The retrieved data were processed using Python, employing libraries such as Pandas, collections defaultdict, and re, to count and categorize the frequency of all words within these abstracts. Subsequently, Microsoft Excel was used to compute total word counts and analyze trends before and after the introduction of ChatGPT. The words were ranked based on the ratio of increase in their usage. For the top 20 non-technical terms that had alternative expressions, a comparative statistical analysis was conducted using Python, juxtaposed with ChatGPT popularity graphs to assess changes in term usage over time.

Results: A total of 6.087 abstracts were extracted, comprising 917 case reports, 943 reviews, 10 editorials, and 4.217 research articles. Analysis identified 17.743 unique words, phrases, and abbreviations, from which frequencies were calculated. The top 20 words that gained popularity exhibited a 55.56% (± 8%) increase in usage following the rise of ChatGPT. A correlation coefficient of 0.63 was observed between the total count of the top 20 popularity-gaining

words and ChatGPT popularity graphs. Further, ANOVA test results indicated a significant correlation ($p < 0.05$) between the words “delve”, “aimed”, “significant”, and “robust” – all of which are reported in the literature as ChatGPT’s favorite words – and the popularity of ChatGPT.

Conclusions: The study demonstrates a significant correlation between the evolving terminology in heart valve disease literature and the rising popularity of advanced computational tools like ChatGPT. The marked increase in specific non-technical terms reflects a broader trend in the scientific community towards integrating these technologies in research dissemination. Our findings underline the potential of AI-driven analyses to not only enhance our understanding of disease trends and terminologies but also to predict and shape the future discourse in medical research. This intersection of traditional medical research with cutting-edge technology opens new avenues for the dynamic and responsive advancement of cardiovascular science.

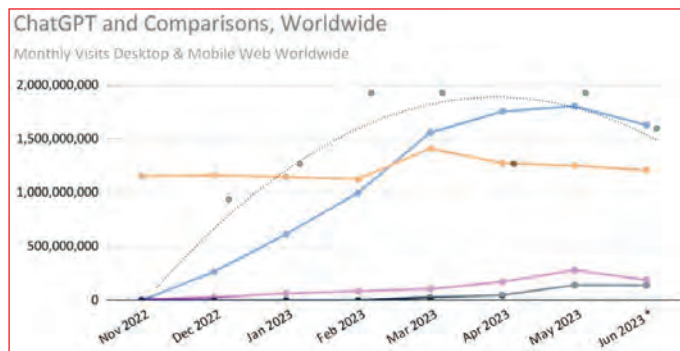


Figure 1. The graph is showing the popularity of chatGPT (blue), use of top 20 popularity-gaining words in heart valve articles (dashed green), use of search engines (orange), use of Grammarly (pink) and use of Bard (grey).

Heart Valve Diseases

PP-049

Change in plasma whole blood viscosity in patients undergoing percutaneous/surgical intervention for aortic stenosis

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Background and Aim: In our study, the aim is to determine how whole blood viscosity (WBV), considered as an inflammatory marker, changes after percutaneous and surgical interventions in cases of aortic stenosis.

Methods: The study was hospital-based, single-center, observational, cross-sectional, and retrospective. It included 200 patients aged 18 and above who underwent bioprosthetic AVR/TAVI between 2019 and 2022 and attended routine outpatient clinics at our Health Sciences

University Koşuyolu Specialized Training and Research Hospital. WBV, hematocrit (HCT %), and total plasma protein concentration (g/dL) were used to calculate both low shear rate (LSR, 0.5 s^{-1}) and high shear rate (HSR, 208 s^{-1}) using the validated formulas by DeSimone and colleagues before and three months after bioprosthetic AVR and TAVI. $\text{HSR} (208 \text{ s}^{-1}) = (0.12 \times \text{hematocrit}) + 0.17 (\text{total protein} - 2.07)$
 $\text{LSR} (0.5 \text{ s}^{-1}) = (1.89 \times \text{hematocrit}) + 3.76 (\text{total protein} - 78.42)$

Results: There is a moderate negative correlation between aortic valve area and HSR ($r = -0.340$, $p < 0.01$). Similarly, there is a moderate negative correlation between aortic valve area and LSR ($r = -0.358$, $p < 0.01$). HSR viscosity values decreased by an average of 0.95 ± 0.31 units between the preoperative period and 3 months postoperatively ($p < 0.001$). LSR viscosity values also decreased by an average of 18.92 ± 6.32 units between the preoperative period and 3 months postoperatively ($p < 0.001$). Due to the observational nature of the study, there are differences among variables between the two groups. To balance these differences, prevent potential confounders, and reduce bias, propensity score analysis (PSA) and “inverse probability weighting” (IPW) method have been used. After balancing the variables, their effects on WBV at 3rd month were modeled using multiple linear regression. It has been found that TAVI has a greater effect on the reduction in WBV at 3 months compared to surgery [Estimate: $-0.137 (-0.262; -0.011)$ $p = 0.003$]. It was also found that valve areas in the TAVI group were larger compared to the other group. Additionally, it has been shown that (effective orifice area) EOA is an independent predictor of WBV at 3 months, and an increase in EOA leads to a reduction in WBV [Estimate: $-0.006 (-0.12 - -0.003)$ $p = 0.04$].

Conclusions: Both the TAVI and bAVR groups showed a statistically significant decrease in HSR and LSR whole blood viscosity values between the preoperative period and 3 months postoperatively. The comparison of preoperative and 3-month postoperative WBV values did not show a statistically significant difference between the TAVI and bAVR groups. In this study, we demonstrated that WBV is an important parameter in patients with aortic valve stenosis. In patients who underwent TAVI, a greater reduction in WBV compared to surgery was observed. We concluded that a significant reason for this reduction is the greater increase in EOA in TAVI patients compared to those who underwent surgery.

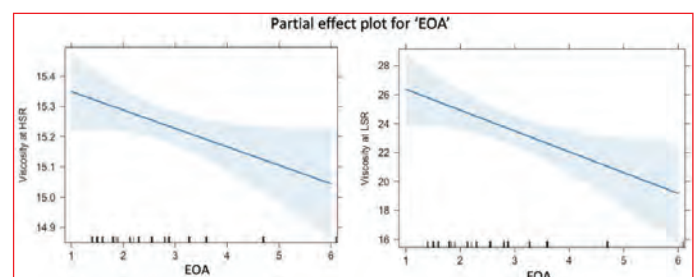


Figure 1. Partial effect plot for EOA.

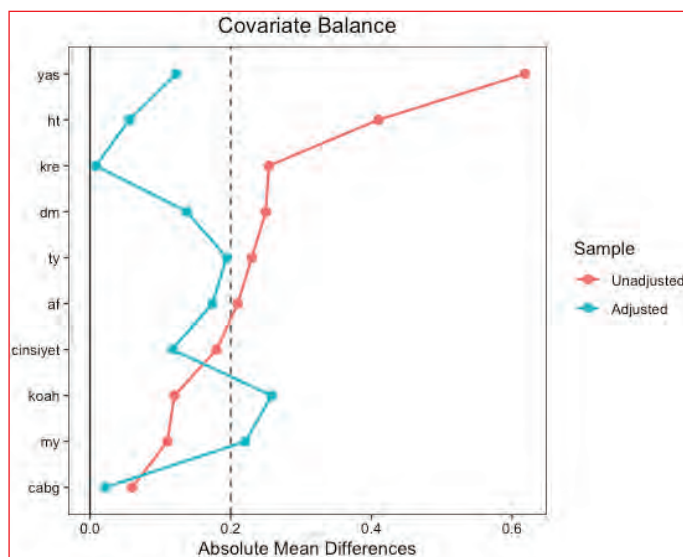


Figure 2. Propensity score covariate balance. Inverse probability weighted covariate balance compared with unadjusted covariates.

Heart Failure

PP-050

Transfer learning for echocardiographic detection of heart failure with preserved ejection fraction: preliminary results of TALE-HFpEF Study

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Background and Aim: Heart failure with preserved ejection fraction (HFpEF) is a heterogeneous syndrome with increasing prevalence. The diagnosis of HFpEF is a complex one that has not yet reached a consensus in current guidelines, and attempts are being made to diagnose it through various algorithms and scoring systems. However, the uncertainties in the diagnostic process and the inherent complexity continue to pose significant barriers to practical implementation. The use of artificial intelligence on single apical 4-chamber transthoracic echocardiography video clips for HFpEF detection has shown success, but knowledge from readily available models trained for different tasks is not utilized. This study aims to utilize transfer learning, an artificial intelligence method, to detect HFpEF using echocardiography images.

Methods: In this preliminary analysis, echocardiography video clips were collected from 40 healthy volunteers and

53 HFpEF patients, all over 18 years old. The diagnosis of HFpEF was made in accordance with the current ESC guidelines. Apical 4-chamber transthoracic echocardiography images of the patients and volunteers included in the study were obtained and analyzed. Patients with chronic obstructive pulmonary disease, recent myocardial infarction (last 6 months), or recent stroke/cerebrovascular disease (last 3 months) were excluded. Transfer learning was applied using a video ResNet model, adapted for left and right ventricle ejection fraction (LVEF and RVEF) prediction tasks, along with a non-medical video classification task (Kinetics 400). A 5-fold cross-validation schema was used, and models were compared using balanced accuracy with a right-tailed t-test.

Results: When comparing with the control group, the HFpEF group shows higher rates of hypertension, diabetes, and atrial fibrillation, as well as higher NT-proBNP levels. The paired one-tailed t-test confirmed significant superiority of all transfer learning models over the baseline model ($p < 0.005$). The model transferred from the LVEF regression task achieved an AUC of 0.95 ± 0.04 and F1 score of 0.93 ± 0.04 (Figure 1), demonstrating superior performance. Statistical analysis indicated no significant variation in balanced accuracy among models ($p > 0.05$). Figure 1 also depicts ROC curves of the models initialized with different task weights. Figure 2 illustrates the locations where models focus before and after training using the Grad-CAM method. The LVEF model has achieved 92% accuracy in identifying HFpEF patients with 95% sensitivity and 90% specificity.

Conclusions: The preliminary results of our study are promising in the diagnosis of HFpEF patients through echocardiographic clips with transfer learning. Throughout our study, as the sample size grows, this model could become a key tool in clinical practice for detecting HFpEF patients, potentially enhancing AI's role in diagnosing this challenging patient group.

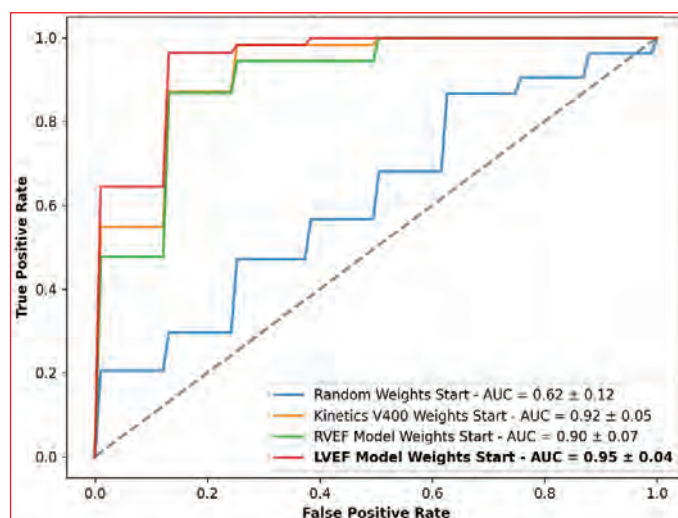


Figure 1. Receiver Operating Characteristic (ROC) Curve of Different Starting Weights on HFPEF Detection Task.

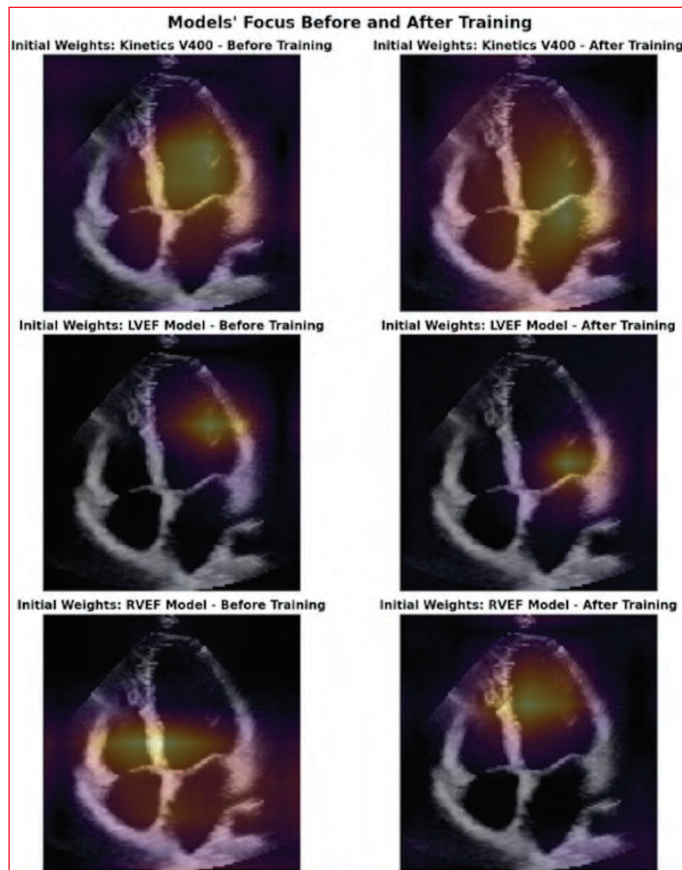


Figure 2. Figure-2 illustrates the locations where models focus before and after training using the Grad-CAM method.

Table 1. Each artificial intelligence model's performance in diagnosing HFpEF

HFpEF Classification Performance				
Initial Model Task	Sensitivity	Specificity	F1 Score	Balanced Accuracy
No Prior Task	1.00 ± 0.00	0.07 ± 0.10	0.74 ± 0.02	0.54 ± 0.05
Non-Medical Video Classification	0.92 ± 0.07	0.88 ± 0.08	0.91 ± 0.04	0.90 ± 0.04
LVEF Regression	0.95 ± 0.07	0.90 ± 0.05	0.93 ± 0.04	0.92 ± 0.04
RVEF Regression	0.88 ± 0.11	0.88 ± 0.11	0.89 ± 0.06	0.88 ± 0.06

Heart Failure

PP-052

Evaluation of serum zonulin levels indicating intestinal permeability in symptomatic heart failure patients

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Background and Aim: There are few studies evaluating serum zonulin levels, which indicate intestinal permeability, in symptomatic HF patients. Our aim in this study was to

evaluate serum zonulin levels and echocardiographic parameters in symptomatic HF patients, regarding their etiology and after current congestion treatment.

Methods: Our study was conducted among patients who applied to cardiology outpatient clinics or emergency departments with chronic HF complaints between November 2022 and November 2023. 32 patients with ischemic origin and 28 patients with non-ischemic HF were prospectively examined. It was simultaneously compared with a group of 45 healthy controls. Demographic data of both groups were taken. Blood samples from all patients included in the study were collected in EDTA-containing blood tubes and echocardiographic evaluation was performed.

Results: Serum zonulin levels [22.9 (12-59), 20.4 (13-78) and 10.1 (6-26) ng/mL $p < 0.01$] were significantly higher in the ischemic and non-ischemic HF patient group compared to the control group before treatment. During the treatment period, ischemic [22.9 (12-59) and 20.9 (10-57) ng/mL, $p = 0.03$] and non-ischemic HF [20.4 (13-78) and 18.2 (12-57) ng/mL, $p = 0.01$]. It was observed that serum zonulin levels decreased significantly in the groups. No significant difference was observed between ischemic and non-ischemic HF groups before ($p = 0.47$) and after ($p = 0.29$) treatment. Echocardiographic data showed a dependent relationship between pre-treatment serum zonulin level and left ventricular EF, right ventricular basal diameter, and pulmonary artery systolic diameter. Serum zonulin level was negatively correlated with left ventricular EF ($b = -0.42$, 95% CI: -0.103-0.22, $p = 0.003$). There was a negative correlation between serum zonulin level and left ventricular EF ($b = -0.42$, 95% CI: -0.103-0.22, $p = 0.003$), a positive correlation with right ventricular basal diameter ($b = 0.43$, 95% CI: 0.25-1.23, $p = 0.004$), and C-reactive protein value ($b = 0.42$, 95% CI: 0.19-0.38, $p < 0.001$). Zonulin levels were also negatively correlated with left ventricular EF ($p = 0.002$) after treatment.

Conclusions: In our study, serum zonulin levels associated with intestinal permeability were found to be significantly

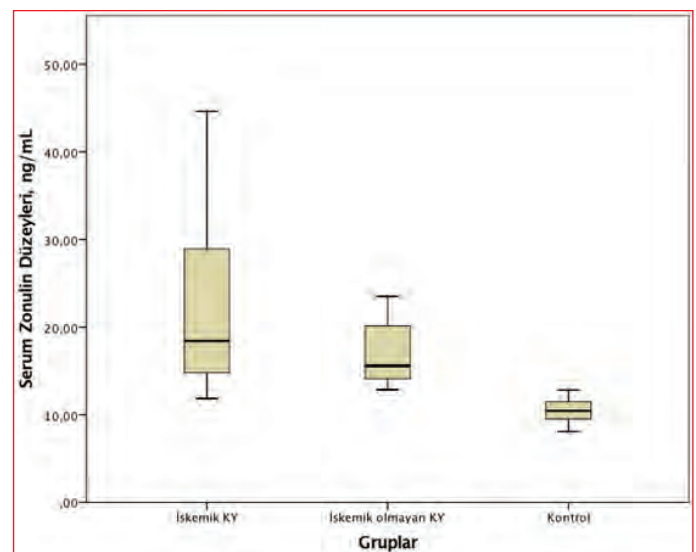


Figure 1. Comparison of serum zonulin levels between pretreatment ischaemic and non-ischaemic HF patients and control groups.

elevated in both HF patient groups, independent of ischemic etiology, and decreased significantly with HF treatment for congestion. Moreover, a significant negative correlation was found between serum zonulin levels and LV EF before and after treatment. These results suggest that serum zonulin levels may be used as a biomarker for both diagnostic and therapeutic follow-up in symptomatic HF patients.

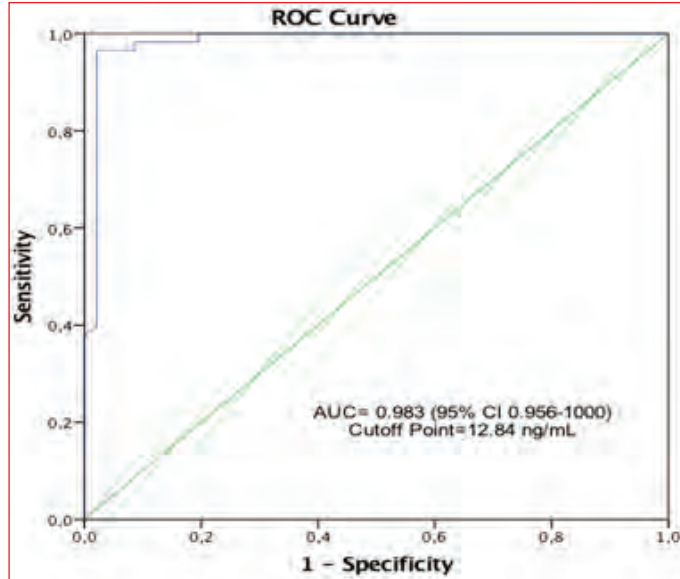


Figure 2. ROC curve analysis of plasma zonulin values in heart failure patients.

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

	İskemik KY n= 32	Non-İskemik KY n= 28	Kontrol n= 45	P Değeri
Yaş Ortalaması, yıl	69±10 *	71±11*	66±7	0.12
Erkek/ Kadın, n/n	25/7 *	19/9 *	33/12	0.67
Sistolik KB, mmHg	146±24 *	137±24 *	121±9	<0.01
Diastolik KB, mmHg	90±16 *	84±13 *	75±6	<0.01
Kalp Hızı, vuru/dk	84±13 *	88±18 *	73±13	<0.01
Vücut Kitle İndeksi, kg/m ²	28±4 *	27±4	26±2	0.18
Bel çevresi, cm	97±13 *	97±14 *	90±6	<0.01
Hipertansiyon, n(%)	26 (81) *	22 (78) *		0.99
Diyabetes Mellitus, n(%)	15 (47) *	15 (53) *		0.39
Hiperlipidemi, n(%)	26 (81) *	9(32) *		<0.01
Obezite	11 (34)	7 (25)		0.57
FK II/III, %/%	50/ 50	46/ 50		0.80
Yoğun bakım yatış süresi, gün	2.5 (0-10)	1.8 (0-12)		0.31
Servis yatış süresi, gün	6.8 (2-14)	5.0 (2-8)		0.02
Cihaz implantasyonu, n (%)	9 (28)	2 (7)		0.04
IV Furosemid miktarı, mg	756	510		0.08
IV Furosemid tedavi süresi, gün	7.0±3.9	5.1±2.0		0.06

FK: fonksiyonel Kapasite, IV: İntravenöz, KB: Kan Basıncı. P: Gruplar arası tedavi öncesi değerlerin karşılaştırılması. P <0.05 değeri istatistiksel olarak anlamlı kabul edildi.

Heart Failure

PP-053

The impact of using SGLT-2 inhibitor on left ventricular longitudinal strain and NT-proBNP levels during six-month follow-up in diabetic patients with and without coronary artery disease with preserved ejection fraction

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Background and Aim: Optimal glycemic control is necessary to prevent cardiovascular events in patients with type 2 diabetes. The positive impact of sodium-glucose cotransporter-2 inhibitors (SGLT2i) on cardiovascular events and mortality in these patients has been demonstrated by previous studies although the mechanism is unclear. We aimed to compare the influence of SGLT2i on left ventricular remodeling and strain in diabetic patients with coronary artery disease (CAD) and without CAD during 6-month follow-up.

Methods: Between October 2021 and June 2022, 100 diabetic patients with preserved ejection fraction (HbA1c levels 6.5-10%) were started on SGLT2i (empagliflozin or dapagliflozin) and were prospectively followed up. Conventional and speckle-tracking echocardiography was performed by blinded sonographers, at baseline and then at 1 month and 6 months of treatment. After 6 months, the initial and biochemical blood tests were administered, and N-terminal pro-B-type natriuretic peptide levels of the patients were measured.

Results: Patients with CAD were older (p=0.008), more frequently hypertensive (p=0.035), and had dyslipidemia (p=0.021). N-terminal pro-B-type natriuretic peptide levels did not change significantly after treatment in both groups. Left ventricular ejection fraction, global, 2 chamber, and 3-chamber strain values were improved significantly following SGLT2i administration for the overall patient cohort, regardless of CAD status (p<0.05 for all groups).

Conclusions: Treatment with SGLT2i resulted in improvement in left ventricular strain parameters, which indicates that they might have a positive impact on outcomes for diabetic patients with preserved EF.

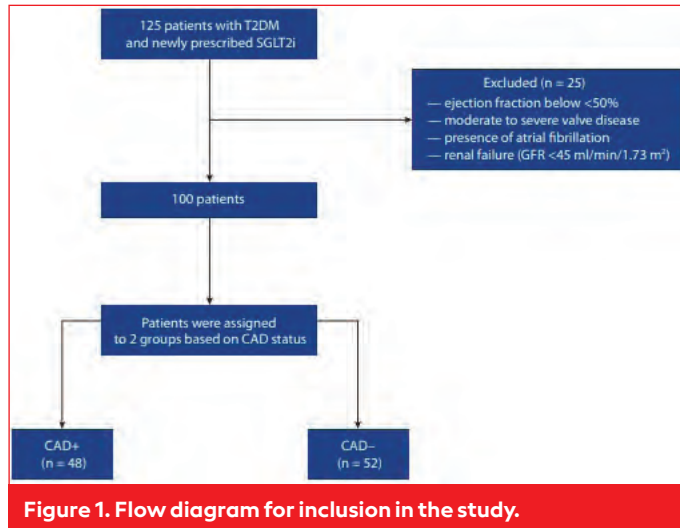


Figure 1. Flow diagram for inclusion in the study.

Table 1. Demographic, clinical and laboratory parameters of the study cohort grouped according to the presence of coronary artery disease

Variables	All population (n = 100)	CAD+ (n = 48)	CAD- (n = 52)	P-value
Female gender, n (%)	71 (71)	37 (77.1)	34 (65.4)	0.2
Age, years	58.7 (9.9)	61.4 (8.6)	56.3 (10.4)	0.01
BMI, kg/m ²	32.0 (4.5)	31.2 (3.1)	32.7 (5.4)	0.11
HT, n (%)	69 (69)	38 (79.2)	31 (59.1)	0.04
Dyslipidemia, n (%)	59 (59)	34 (70.8)	25 (48.1)	0.02
Smoking, n (%)	27 (27)	16 (33.3)	11 (21.2)	0.17
Family history, n (%)	26 (26)	14 (29.2)	12 (23.1)	0.49
CRF, n (%)	7 (7)	3 (6.3)	4 (7.7)	0.78
Stroke history, n (%)	1 (1)	0 (0)	1 (1.9)	0.33
COPD, n (%)	4 (4)	0 (0)	4 (7.7)	0.05
Medications				
β-blockers, n (%)	39 (39)	29 (60.4)	10 (19.2)	<0.001
CCBs, n (%)	41 (41)	24 (50)	17 (32.7)	0.08
RAAS-blockers, n (%)	66 (66)	39 (81.3)	27 (51.9)	0.002
MRA, n (%)	5 (5)	3 (6.3)	2 (3.8)	0.58
Statins, n (%)	38 (38)	24 (50.2)	12 (23.1)	0.001
Empagliflozin, n (%)	66 (66)	31 (64.6)	35 (66.5)	0.91
Metformin, n (%)	82 (82)	40 (83.3)	42 (80.8)	0.74
Laboratory tests				
Creatinine, mg/dl	0.85 (0.28)	0.89 (0.31)	0.82 (0.27)	0.41
TC, mg/dl	209 (42)	212 (47)	207 (47)	0.61
LDL-C, mg/dl	133 (33)	134 (27)	132 (38)	0.75
HDL-C, mg/dl	41.8 (8.6)	41.1 (8.6)	42.4 (8.7)	0.46
Triglyceride, mg/dl	163 (121-252)	189 (124-288)	153 (116-229)	0.04
NT-proBNP baseline, pg/ml	100 (55.3-160)	125 (77-163.8)	78 (45.6-158.3)	0.76
NT-proBNP sixth month, pg/ml	88 (57.3-130)	92.5 (58.5-127.5)	80.5 (51.3-146)	0.43
Hemoglobin, g/dl	13.3 (1.7)	13.1 (1.4)	13.5 (1.8)	0.44
CRP, mg/dl	3.30 (1.40-5.70)	3.80 (1.10-6.30)	3.10 (1.90-5.10)	0.04

Continuous variables are given as means and standard deviations or medians and first and third quartiles (IQR). Categorical variables are presented as numbers and percentages. Abbreviations: BMI, body mass index; CCBs, calcium channel blockers; COPD, chronic obstructive pulmonary disease; CRF, chronic renal failure; CRP, C-reactive protein; HDL-C, high-density lipoprotein cholesterol; HT, hypertension; LDL-C, low-density lipoprotein cholesterol; MRA, mineralocorticoid receptor antagonists; RAAS, renin-angiotensin system; TC, total cholesterol

Table 2. Echocardiographic parameters of the all study cohort

Variables	Findings	P-value	ANOVA
Echocardiographic parameters			
LV end-diastolic volume _{ul} , ml	51 (49-53)	<0.001	
LV end-diastolic volume _{ml} , ml	50 (48.25-52)		
LV end-systolic volume _{ul} , ml	30 (29-32)	<0.001	
LV end-systolic volume _{ml} , ml	29 (27-31)		
E/a _o	11.8 (2.25)	0.28	
E/e' _a	11.7 (2.33)		
LAV _{ul} , ml/m ²	34.74 (2.33)	0.04	
LAV _{ml} , ml/m ²	33.41 (2.8)		
LVEF _{ul} , %	56.3 (4.7)	0.004	<0.001
LVEF _{ml} , %	58.1 (7.6)		
LVEF _{ul} , %	59.3 (5.8)		
Global longitudinal strain _{ul}	17.9 (2.2)		<0.001
Global longitudinal strain _{ml}	18.6 (2.6)		
Global longitudinal strain _{ul}	18.9 (2.6)		
Two-chamber strain _{ul}	17.9 (2.2)		<0.001
Two-chamber strain _{ml}	18.2 (2.7)		
Two-chamber strain _{ul}	18.6 (3.0)		
Three-chamber strain _{ul}	18.0 (2.7)		0.003
Three-chamber strain _{ml}	18.5 (2.8)		
Three-chamber strain _{ul}	18.8 (2.5)		
Four-chamber strain _{ul}	17.8 (2.5)		<0.001
Four-chamber strain _{ml}	19.0 (3.3)		
Four-chamber strain _{ul}	19.3 (2.1)		

Baseline, First month follow-up, Sixth month follow-up; P= Comparison between baseline and sixth month follow-up; P= Global strain, vs Global strain, P= Global strain, vs Global strain. Data are mean (standard deviation) for normally distributed data and median and interquartile range for non-normally distributed data. Repeated measures of ANOVA, assessing for differences in change — in LVEF and strain values when all time points are considered

Abbreviations: LAV_{ul}, left atrial volume index; LV, left ventricle; LVEF, left ventricular ejection fraction

Heart Failure

PP-054

Type and use of loop diuretics can affect mortality in outpatients with heart failure

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Background and Aim: Although some studies have suggested a potential benefit of torasemide over furosemide for hard-endpoints in outpatients with heart failure (HF), there has been no striking evidence of the superiority of torasemide compared with furosemide in this patient group due to relatively small studies of torasemide versus furosemide. Therefore, we aimed to investigate the association between mortality and both the use and type of different loop diuretic (LD) strategies, especially in outpatients with HF.

Methods: This study was a retrospective observational study that included 378 outpatients with HF who were admitted to the Cardiology Outpatient Clinic of Karabük University, Faculty of Medicine, between January 1, 2022, and December 31, 2022. We stratified all included outpatients with HF into three distinct groups based on LD strategy: furosemide, torasemide, and none LD groups. The primary outcome was 1-year all-cause mortality. We conducted Kaplan-Meier analysis and Cox regression analysis to estimate the unadjusted 1-year all-cause mortality rate and the adjusted 1-year all-cause mortality rate in the three different groups of LD strategies, respectively. We created a model including age, sex, atrial fibrillation, LVEF, BNP, eGFR, combined diuretic treatment strategy, beta blockers, RASi, MRA, and NOAC for adjusted 1-year all-cause mortality in Cox regression analysis.

Results: The baseline characteristics of the patients were similar between the three LD strategies except LVEF, the presence of atrial fibrillation, the use of beta blockers, RASi, and MRA, the use of combined diuretic treatment strategy, and BNP levels (Table 1). Primary outcome occurred in 30 of 188 patients (16%) in the furosemide, 7 of 70 patients (10%) in the torasemide, and 17 of 120 patients (14.2%) in the none LD, respectively (Table 2). The number of 1-year total emergency department visits and the number of 1-year cardiology outpatient clinic visits were shown in Table 2 based on the three LD strategies. Overall mean survival day was 330.24 ± 4.86 (95% CI: 320.71-339.77). The mean survival was 327.60 ± 7.18 (95% CI: 313.52-341.68) days in the furosemide, 337.37 ± 10.11 (95% CI: 317.55-357.19) days in the torasemide, and 330.22 ± 8.52 (95% CI: 313.50-346.94) days in the none LD, respectively (log-rank p=0.49) (Figure 1). In the Cox regression analysis, the overall survival rate was the worst in the furosemide, followed by in the none LD strategy and the torasemide, respectively (Log rank p<0.001) (Figure 2).

Conclusions: Compared with furosemide-treated outpatients with HF, torasemide and none LD strategy were less likely to cause 1- year all-cause mortality. Our data are hypothesis-generating, and randomized controlled trials are needed to investigate the optimal LD type.

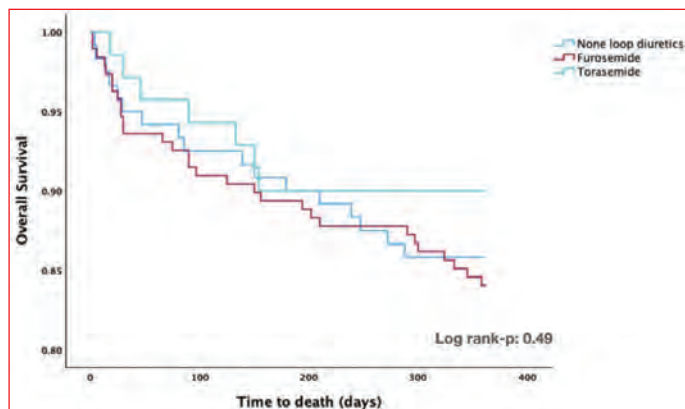


Figure 1. Kaplan-Meier analysis showed the overall survival of outpatients with heart failure according to loop diuretic strategy.

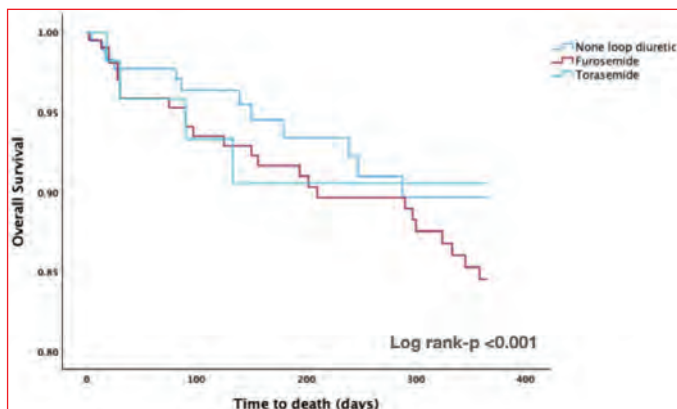


Figure 2. Overall survival according to loop diuretic strategy adjusted by Cox regression analysis in outpatients with heart failure. *Model was adjusted by age, sex, atrial fibrillation, left ventricular ejection fraction, B-type natriuretic peptide, estimated glomerular filtration rate, beta blocker usage, renin-angiotensin-aldosterone system inhibitors usage, mineralocorticoid receptor antagonist usage, new oral anticoagulant usage, and the use of combined diuretic therapy in the Cox regression analysis.

Table 1. Baseline characteristics of the outpatients with heart failure according to loop diuretic strategies

	Furosemide (n=188)	Torasemide (n=70)	None LD (n=120)	P
Age (years)	72.00 (65.25-78.75)	69.00 (59.00-79.25)	71.00 (61.00-80.00)	0.57
Sex (female/male)	103/85	36/34	56/64	0.38
LVEF (%)	50.00 (35.00-55.00)	42.50 (35.00-50.00)	50.00 (40.00-55.00)	0.04
Hypertension (n,%)	173 (92.0)	63 (90)	104 (86.7)	0.31
Diabetes mellitus (n,%)	74 (39.4)	25 (35.7)	47 (39.2)	0.86
Previous MI (n,%)	47 (25)	14 (20)	24 (20)	0.50
Hyperlipidemia (n,%)	98 (52.1)	38 (54.3)	58 (48.3)	0.67
Atrial fibrillation (n,%)	121 (64.4)	46 (65.7)	50 (41.7)	<0.05
Medical therapy				
Beta blockers (n,%)	172 (91.5)	65 (92.9)	71 (59.2)	<0.05
RASI (n,%)	149 (79.3)	59 (84.3)	73 (60.8)	<0.05
MRA (n,%)	124 (66.0)	53 (75.7)	28 (23.3)	<0.05
SGLT2i (n,%)	37 (19.7)	12 (17.1)	18 (15.0)	0.57
VKA (n,%)	20 (10.7)	9 (12.9)	6 (5.0)	0.13
NOAC (n,%)	102 (54.3)	39 (55.7)	37 (30.8)	<0.05
Combined DT (n,%)	74 (39.4)	25 (35.7)	0 (0.0)	<0.05
Device therapy				
ICD (n,%)	19 (10.1)	11 (15.7)	7 (5.8)	0.08
Laboratory variables				
BNP (pg/mL)	269.24 (159.00-556.97)	415.26 (141.14-943.16)	192.69 (61.89-525.69)	0.03
eGFR (mL/min/1.73m ²)	56 (50-89)	66 (53-88)	12.80 (10.85-14.10)	0.88
Hemoglobin (gr/dL)	12.80 (11.20-13.92)	12.50 (11.45-13.80)	1.91 (1.15-3.52)	0.38
TSH (mIU/L)	1.70 (0.90-2.78)	1.97 (0.80-3.01)		

LD, loop diuretics; LVEF, left ventricular ejection fraction; MI, myocardial infarction; RASI, renin-angiotensin-aldosterone system inhibitors; MRA, mineralocorticoid receptor antagonists; SGLT2i, sodium-glucose cotransporter-2 inhibitors; VKA, vitamin K antagonists; NOAC, new oral anticoagulants; DT, diuretic therapy; ICD, intracardiac defibrillator; BNP, brain natriuretic peptide; eGFR, estimated glomerular filtration rate; TSH, thyroid stimulating hormone.

Table 2. Outcomes of outpatients with heart failure according to loop diuretic strategies

	Furosemide (n=188)	Torasemide (n=70)	None LD (n=120)	p
Number of emergency department visits at 1-year	1 (0-3)	1 (0-3)	1 (0-3)	0.99
Number of cardiology outpatient clinic at 1-year	3 (1-5)	2 (1-4)	2 (1-3)	<0.05
All-cause mortality at 1-year (n,%)	30 (16.0)	7 (10.0)	17 (14.2)	0.48

LD, loop diuretics.

Heart Failure

PP-056

Evaluation of the relationship between plasma miRNA level and cardiotoxicity in patients diagnosed with cancer and started anthracycline chemotherapy

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Background and Aim: Breast cancer is one of the most common types of cancer among women worldwide. Anthracycline derivative chemotherapeutic agents are frequently used in the treatment of sarcoma and breast cancer. Recent studies have evaluated the association of microRNA-1 (miR-1) expression with cardiotoxicity. The aim of our study is to determine whether miRNAs have superiority in terms of predicting cardiotoxicity compared to high-sensitivity cardiac troponin T and N-terminal pro-brain natriuretic peptide,

Table 1. Comparison of the serum NT-proBNP, Hs-cTn and plasma miR-1 variables of the patients in the study group before CT and 24-48 hours after CT

Parameters	Pre-chemotherapy (Mean ± SD) (1)	Post-chemotherapy 24-48 hours (Mean ± SD) (2)	1-2 [§] (p)
NT-ProBNP (pg/mL)	76.4 ± 55.1	315.3 ± 195.1	<0.001*
Hs-cTnT (ng/mL)	0.005 ± 0.002	0.06 ± 0.003	<0.001*
MiR-1 DeltaCT	15.9 ± 4.0	14.5 ± 3.9	0.048

Hs-cTnT: High sensitivity cardiac troponin T; NT-proBNP: N-terminal Pro-brain natriuretic peptide. * Wilcoxon test, [§] Changes in serum and plasma parameters measured 24-48 hours after chemotherapy compared to baseline.

which are currently used in the determination of cardiac damage.

Methods: The study included 51 cancer patients (a total of 102 samples before and after KT) who applied to the oncology outpatient clinic of Eskişehir Osmangazi University Faculty of Medicine Training Practice and Research Hospital between 10.02.2020 and 30.06.2022 and agreed to participate in the study, and it was conducted prospectively. mi-RNA1, Hs-cTnT and NT-proBNP were measured at baseline and 24-48 hours after chemotherapy.

Results: In our study, we examined whether miR-1 can be used predictively in determining cardiotoxicity. In our study, when compared to pre-chemotherapy, high-sensitive cardiac troponin T (0.06 ± 0.003 vs. 0.005 ± 0.002 , $p < 0.001$) and N-terminal pro-brain natriuretic peptide (315.3 ± 195.1 vs. 76.4 ± 55.1 , $p < 0.001$) taken 24-48 hours after chemotherapy statistically significant increase was revealed. In DeltaCT (14.5 ± 3.9 vs. 15.9 ± 4.0 , $p = 0.048$), whose decrease expresses an increase in miR-1 expression, there was a significant decrease after chemotherapy. In the multivariate analysis we performed to determine the variables associated with subclinical cardiotoxicity, we found that the first-year left atrial contractile

strain and high-sensitivity cardiac troponin in the blood at 24-48 hours significantly changed compared to the baseline values as independent predictors of >15% decrease in global longitudinal strain. When evaluated according to baseline echocardiography, in the first year after chemotherapy, left atrial ejection fraction (10.8 vs. -5.8, $p = 0.006$) and left atrial contractile strain (-5.4 vs. 0.3, $p = 0.022$) change decreased significantly more in the group that developed subclinical cardiotoxicity.

Conclusions: It was determined that there was a significant increase in miR-1 levels after chemotherapy compared to the pre-chemotherapy value. As a result, increases in miR-1 levels may occur in the early period following anthracycline exposure and may be useful in detecting cardiomyocyte damage.

Heart Failure

PP-057

Intravesical pressure measurement: A simple method to predict diuresis in patients with congestive heart failure

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Background and Aim: In patients with congestive heart failure, intra-abdominal pressure (IAP) may increase due to ascites or increased fluid within the splanchnic system. It is associated with renal venous congestion, decreased renal perfusion pressure and impaired renal function in these patients. The goal of our study is to assess the capability of IAP in predicting the diuresis in patients who were hospitalized due to decompensated heart failure.

Methods: This single-center study comprised of 83 patients (female 58.9% and mean age 71.6 ± 13.6 years) with symptomatic heart failure who were admitted to the intensive care unit (ICU). Patients with refractory shock or those requiring ultrafiltration were excluded. The patients were treated with intravenous loop diuretics along with guideline-directed medical therapy to achieve euvolemic status. IAP was intra-vesical measured according to guidelines using a urinary catheter connected to a pressure transducer in the ICU prior to the initiation of diuretic treatment. Elevated IAP

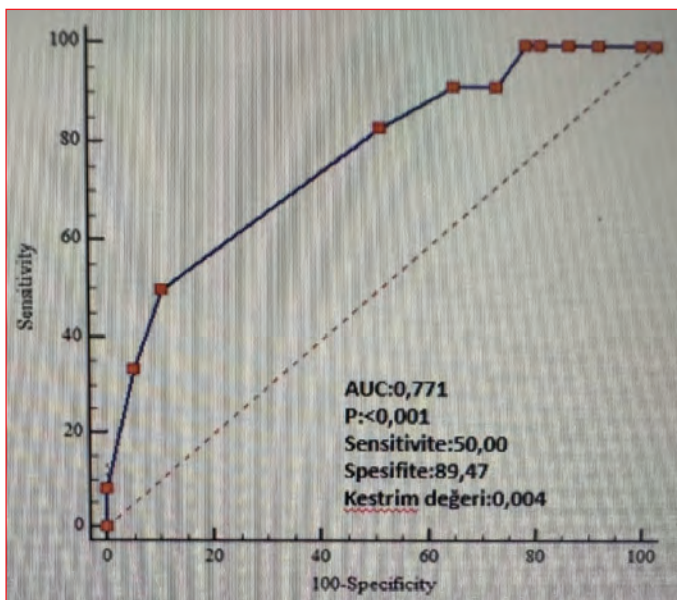


Figure 1. ROC analysis curve showing the diagnostic validity of Hs-cTnT values after chemotherapy in the study group, according to the determination of a 15% decrease in GLS after chemotherapy.

is defined as >8 mm Hg. The abdominal perfusion pressure (APP) is calculated by: Mean arterial pressure-IAP.

Results: Patients were stratified into two groups: elevated IAP (n=44), and normal IAP (n=39). Baseline demographic, clinical, laboratory results were similar between the groups. The APP was lower in high IAP group [65.3 (56.8-78.5) vs. 74 (64-78.5); p=0.008]. Multiple linear regression was performed on diuresis volume, adjusting for admission creatinine, intravenous diuretic dosage, and the need for inotropes. IAP predicted both the initial 24-hour diuresis and the cumulative 48-hour diuresis (R2=0.192 and 0.131, respectively.) A one-unit increase in IAP (measured in mmHg) was associated with a decrease in urinary output of 213 mL (95% CI: 355-71) over 24 hours and 310 mL (95% CI: 569-51) over 48 hours.

Conclusions: In patients admitted with decompensated heart failure, intra-vesical measured IAP predicts the initial 48-hour urinary output. Measuring IAP by this simple method may be helpful in predicting response to diuretic treatment. Identifying patients with increased IAP and decreased APP may guide clinicians to take more aggressive treatment measures earlier such as high-dose-diuretics or ultrafiltration to achieve decongestion.

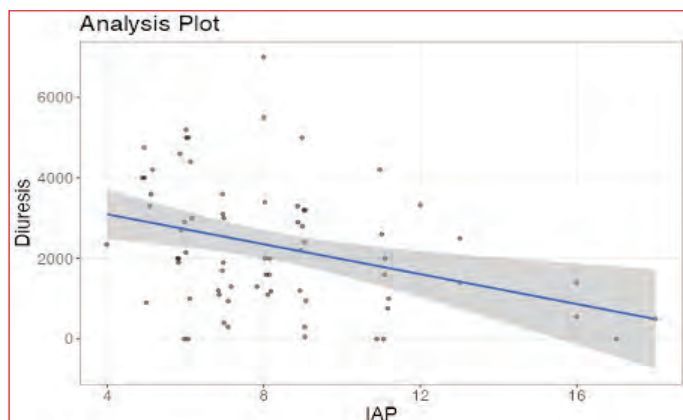


Figure 1. Relationship between diuresis and increasing intra-abdominal pressure.

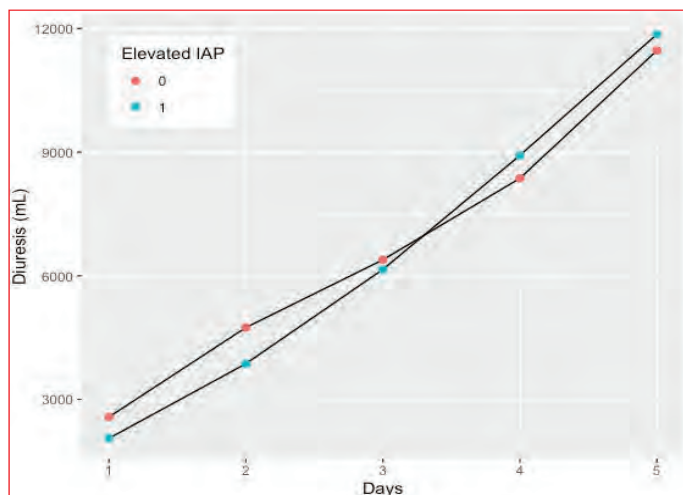


Figure 2. Presence of the increased intra-abdominal pressure is associated with decreased diuresis.

Table 1.

Table-1: Demographic, clinical and laboratory data of the patients according to the presence of elevated intra-abdominal pressure

Variables	Patients with IAP ≥ 8 mm Hg (n =44)	Patients with IAP < 8 mmHg (n = 39)	p Value
Age, years	73 (11.5)	70.1 (15.5)	0.568
BMI, kg/m ²	24.6 (2.36)	25.1 (3.35)	0.476
Hypertension, %	31 (70.5)	23 (59)	0.274
Diabetes, %	20 (45.5)	15 (38.5)	0.520
Atrial fibrillation, %	20 (45.5)	13 (33.3)	0.260
Ischemic, %	29 (65.9)	19 (48.7)	0.113
Previous CABG, %	8 (18.2)	4 (10.3)	0.362
Previous PCI, %	4 (9.1)	3 (7.7)	0.819
Stroke, %	3 (6.8)	1 (2.6)	0.619
COPD, %	11 (25)	12 (30.8)	0.558
Malignancy, %	0	2 (5.1)	0.218
Medication on admission, %			
Beta blocker	31 (70.5)	22 (56.4)	0.184
ACEI	30 (68.2)	21 (53.8)	0.180
MRA	20 (45.5)	12 (30.8)	0.170
Loop diuretic	28 (63.6)	22 (56.4)	0.502
Statin	23 (52.3)	16 (41)	0.306
Ejection Fraction, %	30 (20-45)	30 (25-45)	0.107
Systolic BP, mm Hg	110 (35.1)	116 (32)	0.279
SpO ₂ , %	88.6 (8.47)	90.4 (6.89)	0.445
IVC, cm	2.14 (0.6)	2.15 (0.19)	0.983
Estimated SPAP, mm Hg	41.6 (10.4)	40.4 (10.8)	0.640
Hemoglobin, g/dL	12.2 (2.49)	12.4 (2.87)	0.725
CRP	13.6 (6-50)	22 (9.6-46)	0.483
BNP, ng/L	9576 (4379-33109)	8711 (3803-26053)	0.523
Pleural effusion, %	39 (88.6)	33 (84.6)	0.590
Ascites, %	16 (36.4)	11 (28.2)	0.428
Length of stay, days	10 (7-14)	8 (5-11)	0.149

Table 2.

Variables	Patients with IAP ≥ 8 mm Hg (n = 44)	Patients with IAP < 8 mm Hg (n = 39)	p Value
Creatinine on admission, mg/dL	1.39 (1.11-1.94)	1.42 (0.99-1.88)	0.544
Worsening renal function, %	12 (27.3)	7 (17.9)	0.313
Lactate, mmol/L	2.21 (1.55-3.3)	1.83 (1.13-2.32)	0.087
IAP, mm Hg	9 (9-11)	6 (6-7)	<0.001
MAP, mm Hg	80.1 (22.3)	86.9 (21.5)	0.151
Perfusion pressure, mm Hg	65.3 (56.8-78.5)	74 (64-78.5)	0.008
HCO ₃ , mmol/L	23.4 (5.22)	22.7 (5.53)	0.578
Sodium, mEq/L	137 (4.51)	137 (3.89)	0.783
Potassium mEq/L	4.75 (0.7)	4.83 (0.9)	0.539
IV furosemide dose, mg	160 (100-200)	120 (80-200)	0.209
Inotrope, %	6 (13.6)	3 (7.7)	0.490
In-hospital mortality, %	4 (9.1)	5 (12.8)	0.728

Table-2: Multiple linear regression analysis in predicting diuresis

Predictor	Estimate	SE	95% Confidence Interval		p
			Lower	Upper	
Intercept	3835.17	766.46	2302.02	5368.32	<.001
IAP	-213.54	70.81	-355.19	-71.88	0.004
Admission creatinine	-63.25	217.16	-497.65	371.14	0.772
IV diuretic dosage	3.01	2.01	-1.01	7.02	0.140
Requirement of inotropes:					
1 - 0	-757.58	793.83	-2345.48	830.33	0.344

Heart Failure

PP-058

Effects of intra-abdominal pressure and abdominal perfusion pressure on the development of infections: A study in heart failure patients

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Background and Aim: There is growing interest in the measurement of intra-abdominal pressure (IAP) in critical patients, as it is associated with intra-abdominal organ dysfunction. As intra-abdominal pressure increases, the pressure on arteries, capillaries, and veins also rises. This increase in pressure reduces blood flow and disrupts venous return. The increase in bacterial translocation can lead to the development of sepsis. Bacterial translocation affects morbidity and mortality in critically ill patients, such as those with heart failure. In this study, we evaluated the relationship between the IAP and abdominal perfusion pressure (APP) values of patients and the development of infectious processes during their hospital stay.

Methods: The study was conducted prospectively in a single center. Between March 2022 and June 2024, 93 patients who were hospitalized with acute decompensated HF. To obtain a precise IAP value, the pressure is measured with the transvesical method. Briefly, IAP is measured via a standard Foley catheter, which is connected with a pressure transducer placed in-line with the iliac crest at the midaxillary line. The Foley catheter is flushed with a maximal instillation volume of 25 mL sterile saline via the aspiration port of the Foley catheter with the drainage tube clamped to allow a fluid-filled column to develop up into the bladder. A pressure transducer is then inserted in the aspiration port, and the pressure is measured. The IAP is expressed in mmHg and is measured at end-expiration in the supine position, ensuring that abdominal muscle contractions are absent. In this study, IAP was measured on admission (within the first 8 hours). The abdominal perfusion pressure, calculated as MAP minus IAP [APP=Mean arterial pressure (MAP)-IAP]. We used a cut-off value of ≥ 8 for elevated IAP. Patients were divided into two groups based on whether or not they developed infectious processes during their hospital stay. Groups with and without clinical outcomes were compared in terms of IAP, APP, and MAP measurements.

Results: Ninety-three patients with a mean age of 74.5 ± 8.6 years were included in the study. There were 58 (62.1%) patients with an IAP value ≥ 8 mmHg. The total length of stay was longer in patients with an IAP value ≥ 8 mmHg (7.7 ± 6.0 vs. 5.4 ± 2.7 days, $p=0.014$). During their hospital stay, a total of 24 (38%) patients developed infectious processes and were started on antibiotic treatment. In the group with clinical outcomes, APP (75.9 ± 9.4 vs. 81.8 ± 12.3 , $p=0.035$) was significantly lower than in the group without clinical outcomes. IAP (11.2 ± 3.7 vs. 9.0 ± 3.7 , $p=0.016$) was significantly higher in the group with clinical outcomes compared to the group without clinical outcomes.

Conclusions: In this study, patients admitted with decompensated heart failure who developed infections had higher IAP and lower APP. Abdominal pressure measurements can provide insights into which patients are more likely to develop infections, which may help in taking preventive measures.

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

	Clinical outcome (-) (n=69)	Clinical outcome (+) (n=24)	p value
Age, years	74.6 \pm 8.4	71.9 \pm 9.2	0.190
Gender, male, n (%)	36 (52.2)	10 (41.7)	0.478
Diabetes, n (%)	29 (42.0)	13 (54.2)	0.347
Hypertension, n (%)	46 (66.7)	17 (70.8)	0.803
RA, diameter, mm	44.0 \pm 8.4	45.7 \pm 12.2	0.551
TAPSE, mm	18.2 \pm 3.8	20.0 \pm 4.0	0.345
LVEF, %	39.9 \pm 15.4	39.0 \pm 13.8	0.841
Creatinine, mg/dL	1.4 \pm 0.7	1.7 \pm 1.0	0.199
SBP, mmHg	126.2 \pm 19.6	125.2 \pm 18.0	0.835
DBP, mmHg	73.2 \pm 11.3	68.1 \pm 10.9	0.057
MAP, mmHg	90.9 \pm 12.3	87.1 \pm 9.8	0.183
IAP, mmHg	9.0 \pm 3.7	11.2 \pm 3.7	0.016
APP, mmHg	81.8 \pm 12.3	75.9 \pm 9.4	0.035

TAPSE: Tricuspid annular plane systolic excursion; LVEF: Left ventricle ejection fraction; RA: Right atrium; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure; IAP: Intraabdominal pressure; APP: Abdominal perfusion pressure

Heart Failure

PP-060

A new parameter that predicts mortality in heart failure: HALP scoreSelim Aydemir¹, Onur Altinkaya¹, Sidar Şiyar Aydın²¹Department of Cardiology, Erzurum Regional Education and Research Hospital, Erzurum²Department of Cardiology, Atatürk University, Faculty of Medicine, Erzurum

Background and Aim: Heart failure (HF) is a chronic, progressive clinical syndrome resulting from structural or functional cardiac abnormalities. HF has a high incidence and mortality rate worldwide. Therefore, prognostic assessment is important for the management of these patients. The combination of haemoglobin, albumin, lymphocytes and platelets (HALP) is a novel measure used to assess prognosis in many diseases including cancer, cerebrovascular and cardiovascular. HALP components indicating these parameters have also been reported to be associated with prognosis in HF patients. Anemia, lymphopenia, and low serum albumin are associated with death and adverse cardiac outcomes in HF

patients. In addition, platelet activation and hypercoagulability are evident in patients with heart failure. However, trials on the relationship between HALP and the prognosis of patients with heart failure have been less reported. Therefore, in our study, we aimed to evaluate the relationship between HALP score and mortality in patients with reduced and mildly reduced ejection fraction heart failure.

Methods: Our study was a retrospective observational study and included patients admitted to our clinic with heart failure EF <50% between 2017 and 2022. The mean follow-up period of the patients in terms of mortality was 6 years. HALP Score was defined as haemoglobin level (g/L) x albumin level (g/L) x

lymphocyte count (10⁹/L) / platelet count (10⁹/L). Univariate and multivariate Cox regression analysis was used to determine the association between HALP score and mortality. Receiver operating characteristic (ROC) curves were drawn to evaluate the predictive power of independent variables for mortality and analyzed using the area under the curve (AUC). P value <0.05 was considered significant.

Results: 886 patients were included in our study. Mortality rate was 9.81%. The mean age of the patients was 68.7 ± 12.6 years and 529 (59.7%) of them were male. HT was present in 69.9%, DM in 25.3%, and coronary artery disease in 75.2% of the patients. The mean EF was 35.8 ± 9.8. Age (OR: 95%

Table 1. Demographic data of groups according to mortality development status

Variables	Total Group (n=886)	Mortality (+) (n=87)	Mortality (-) (n=799)	p value
Age (year)	68.71 ± 12.6	73.93 ± 12.5	68.14 ± 12.5	<0.001
Gender (male, %)	529 (59.7)	61 (70.1)	468 (58.6)	0.037
HT (number, %)	620 (69.9)	62 (71.3)	558 (69.8)	0.126
DM (number, %)	224 (25.3)	26 (29.9)	198 (24.8)	0.299
CAD (number, %)	666 (75.2)	61 (70.1)	605 (75.7)	0.251
AF (number, %)	271 (30.6)	30 (34.5)	241 (30.2)	0.407
Ejection fraction (%)	35.83 ± 9.82	36.16 ± 10.32	35.79 ± 9.77	0.744
ASA (number, %)	437 (49.3)	39 (44.8)	398 (49.8)	0.377
Statins (number, %)	248 (28)	29 (33.3)	219 (27.4)	0.243
NOAC (number, %)	203 (22.9)	24 (27.6)	179 (22.4)	0.275
ACEI/ARB (number, %)	506 (57.1)	49 (56.3)	457 (57.1)	0.846
MRA (number, %)	250 (28.2)	24 (27.5)	226 (28.2)	0.648
BB (number, %)	453 (51.1)	46 (52.9)	407 (50.9)	0.732
Diuretics (number, %)	366 (41.3)	37 (42.5)	329 (41.1)	0.550
Ivabradin (number, %)	131 (14.8)	8 (9.2)	123 (15.4)	0.122
Hb (g/dL)	14.10 ± 2.27	13.14 ± 2.79	14.21 ± 2.18	<0.001
WBC (10 ³ /μL)	9.54 ± 4	11.77 ± 5.9	9.26 ± 3.7	<0.001
Platelet count (10 ³ /μL)	250.1 ± 82.1	257.9 ± 111	249.2 ± 78.3	0.679
AST (U/L)	53.7 (6-1295)	152.1 (9-1295)	38.3 (6-1000)	<0.001
CRP (mg/L)	25.1 (1-272)	45.5 (1-253)	23.1 (1-272)	<0.001
Creatinine (mg/dL)	1.20 ± 0.78	1.71 ± 1.1	1.15 ± 0.71	<0.001
Na (mmol/L)	139.5 ± 4.1	140.6 ± 6.5	139.4 ± 3.8	0.141
K (mmol/L)	4.47 ± 0.65	4.68 ± 0.85	4.55 ± 0.63	0.030
Glucose (mg/dL)	156.3 ± 93.7	176.9 ± 95.6	154 ± 93.3	0.001
Albumin (g/L)	35.7 ± 13.3	36.1 ± 8.9	35.6 ± 13.7	0.001
Total cholesterol (mg/dL)	163.9 ± 48.9	150.8 ± 49.6	165.4 ± 48.7	0.063
TG (mg/dL)	145.8 ± 74.3	127.9 ± 66.4	147.8 ± 74.9	0.053
LDL (mg/dL)	118.5 ± 84.1	117.3 ± 52.1	118.6 ± 87.1	0.819
HALP Score	58.3 (2.4-252)	42.1 (2.4-196)	60.6 (12-252)	<0.001

HT: Hypertension; DM: Diabetes mellitus; CAD: Coronary artery disease; AF: Atrial fibrillation; ASA: Acetylsalicylic acid; NOAC: Novel oral anticoagulants; ACEI: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker; MRA: Mineralocorticoid receptor antagonist; BB: Beta blocker; Hb: Haemoglobin; Wbc: White blood cell; AST: Aspartate aminotransferase; CRP: C-reactive protein; Na: Sodium; K: Potassium; TG: Triglycerides; LDL: Low density lipoprotein.

Table 2. Regression analysis according to mortality development status

Variables	Univariate OR, 95% CI	p value	Multivariate OR, 95% CI	p value
Age	1.040 (1.020-1.060)	<0.001	1.032 (1.004-1.062)	0.025
Sex	0.706 (0.634-0.996)	0.037	4.470 (1.937-10.316)	<0.001
WBC	1.120 (1.077-1.166)	<0.001	1.058 (0.982-1.141)	0.140
CRP	1.008 (1.004-1.013)	<0.001	1.001 (0.991-1.006)	0.713
AST	1.003 (1.002-1.004)	<0.001	1.002 (1.001-1.004)	0.002
Glucose	1.002 (1.001-1.004)	0.026	1.001 (0.997-1.004)	0.777
Creatinine	1.399 (1.251-1.564)	<0.001	1.364 (1.099-1.692)	0.005
HALP Score	0.857 (0.790-0.929)	<0.001	1.849 (0.756-0.955)	0.006

Hb: Haemoglobin; WBC: White blood cell; CRP: C-reactive protein; AST: Aspartate aminotrasferase

CI: 1.004-1.062, $p=0.025$), gender (OR: 4.470, 95% CI: 1.937-10.316, $p<0.001$), AST (OR: 1.002, 95% CI: 1.001-1.004, $p<0.001$), creatinine (OR: 1.364, 95% CI: 1.099-1.692, $p=0.005$) and HALP score (OR: 1.849, 95% CI: 0.756-0.955, $p=0.006$) were independently associated with mortality. In the ROC curve analysis, the predictive value of the HALP score was 42 and the AUC value for predicting mortality was 0.645 (95% CI: 0.576-0.715, $p<0.001$).

Conclusions: In our study, we observed that the HALP Score can be used as a parameter to predict mortality in patients with reduced or mildly reduced heart failure. While inflammation plays a role in the pathogenesis of heart failure patients, nutritional status also plays an important role in the prognosis of patients. Since the HALP score includes indicators of immune response and nutritional status, it can be used as a marker in predicting the prognosis of patients with heart failure.

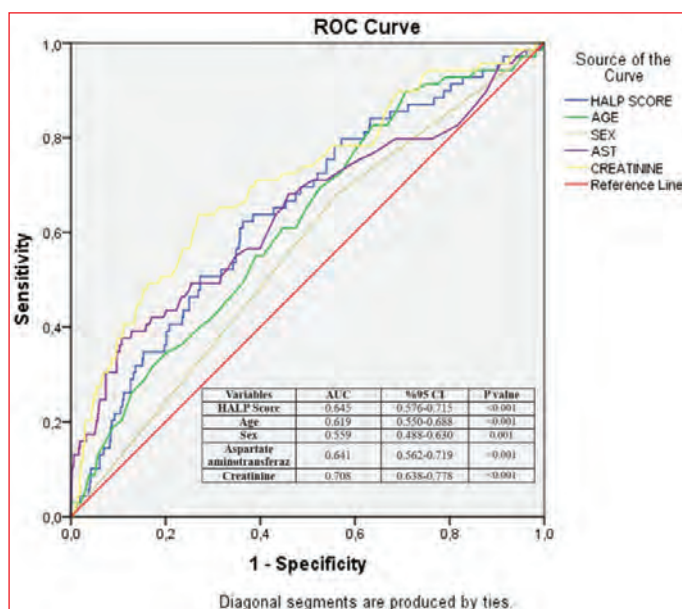


Figure 1. Receiver operating characteristic (ROC) curve analysis and Area under the curve values (AUC) of parameter.

Cardiac Imaging / Echocardiography

PP-063

Prognostic predictors in constrictive pericarditis, 19-year experience at a tertiary care hospital

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Background and Aim: Constrictive pericarditis is a chronic inflammatory entity that may progress with thickening and calcification in the pericardial tissue, resulting in decreased pericardial elasticity and subsequent increase in ventricular filling pressures. This rare treatable cause of HFpEF, is associated with various aetiologies, depending on the development level of the countries. Several algorithms have been developed for the diagnosis, whereas studies evaluating the prognostic factors are limited. Our study aimed to examine the clinical, laboratory, electrocardiographic, imaging and catheterization parameters that determine the prognosis of constrictive pericarditis.

Methods: 60 patients diagnosed with constrictive pericarditis based on echocardiography, computed tomography and right heart catheterization parameters were included retrospectively in the study. Clinical presentation, laboratory, and electrocardiographic findings were assessed. Volumetric measurement, tissue Doppler, mitral inflow velocity analysis, and pericardial evaluation were made via 2D echocardiography. Pericardial thickness, effusion, and calcium score were obtained via thorax CT, while right-sided pressure measurements were acquired via catheterization. The primary clinical outcome was determined as mortality, and the effect of these parameters was evaluated by Cox regression analysis.

Results: Median follow-up was 3.5 years (0–19) and mean age was 52.7 ± 17.5 years. 38 of the patients (63.3%) were male,

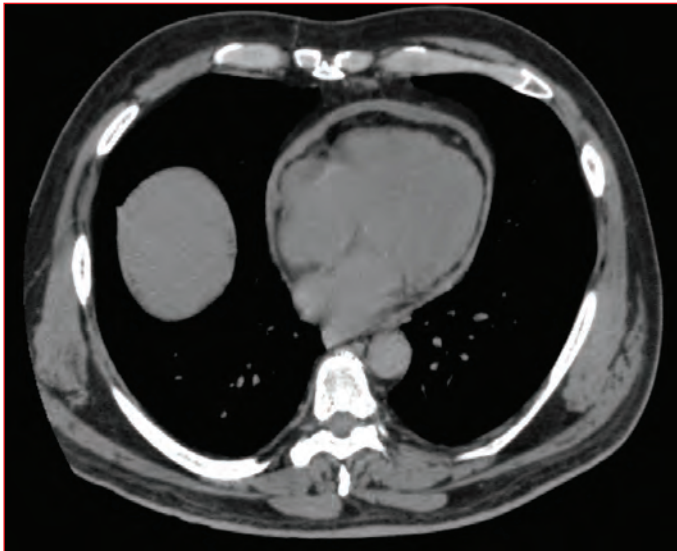


Figure 1. Computed tomography showing constrictive pericarditis.

the most common aetiologies were malignancy (21.4%) and other systemic diseases (21.4%). 7 patients were lost to follow-up and 18 (31.6%) patients died during the process. When different models were evaluated with Cox regression analysis, ascites, hemoglobin and elevated proBNP values were prognostic predictors (HR: 10.991 p value 0.006, HR: 0.513 p value 0.010 and HR: 3.904 p value 0.045, respectively). ROC analysis indicated the highest AUC value for the model that included hemoglobin (AUC 0.953).

Conclusions: Constrictive pericarditis is one of the treatable causes of HFpEF. While imaging and catheterization parameters play a crucial role in the diagnosis, clinical and laboratory parameters affect the prognosis.

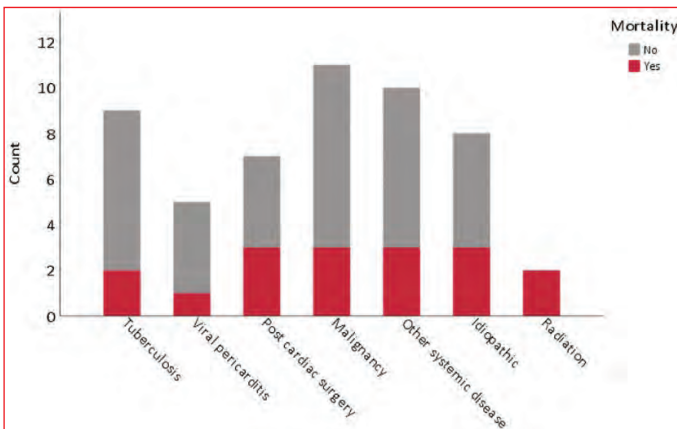


Figure 2. Figure showing etiology and mortality distribution of constrictive pericarditis cases.

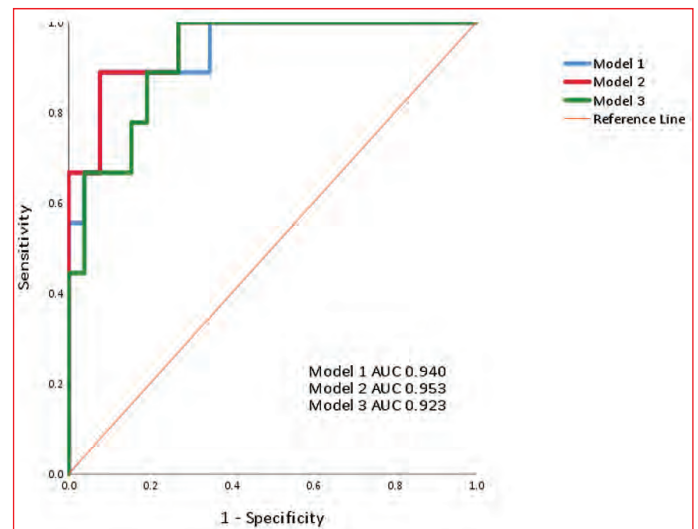


Figure 4. ROC analysis showing AUC values for models.

	Univariate			Model 1			Model 2			Model 3		
	HR	95% CI	p-value	HR	95% CI	p-value	HR	95% CI	p-value	HR	95% CI	p-value
Age	1.035	1.002-1.069	0.040	1.046	0.972-1.125	0.233						
Male Gender	2.425	0.690-8.522	0.167									
Ascites	4.820	1.696-13.697	0.003	10.991	2.021-59.780	0.006*						
Peripheral edema	1.397	0.524-3.726	0.504									
Hgb	0.622	0.472-0.819	0.001				0.513	0.308-0.852	0.010*			
Albumin	0.640	0.336-1.217	0.173									
Pro-BNP (quartile 4)	2.631	1.234-5.611	0.012							3.904	1.033-14.751	0.045*
LVEDV	1.013	1.001-1.026	0.039	1.007	0.989-1.024	0.442	0.997	0.978-1.016	0.759	1.009	0.991-1.029	0.328
LVEF	0.998	0.923-1.080	0.966									
sPAP-Echocardiography	1.027	0.986-1.071	0.203									
Pericardial thickness-CT	0.934	0.643-1.357	0.721									
Pericardial calcium score (ln)	1.032	0.602-1.768	0.910									
RVEDP-catheter	0.971	0.814-1.158	0.743									
sPAP-catheter	1.118	0.990-1.263	0.073									
Heart rate	1.017	0.982-1.052	0.351									
Sinus rhythm	0.764	0.232-2.518	0.658									
Low voltage	3.366	0.823-13.766	0.091	0.720	0.126-4.123	0.712	1.363	0.236-7.860	0.729	0.947	0.184-4.881	0.948
T wave inversion	0.575	0.151-2.186	0.417									

Figure 3. Cox regression analysis showing prognostic predictors.

Cardiac Imaging / Echocardiography

PP-064

Left ventricular hypertrophy findings on electrocardiogram predict impaired left atrial functionsUğur Karagöz¹, Nihan Kahya Eren², Emre Özdemir², Volkan Emren², Mustafa Ozan Gürsoy³, Mehmet Tokaç²¹Cardiology Clinic, Torbalı State Hospital, İzmir²İzmir Katip Çelebi University Atatürk Training and Research Hospital, İzmir³Cardiology Clinic, İzmir Tepecik Training and Research Hospital, İzmir

Background and Aim: Electrocardiographic left ventricular hypertrophy (ECG LVH) holds significant clinical importance in cardiovascular disease. Pathological processes that lead to left ventricular hypertrophy (LVH) also induce remodeling and impair left atrial (LA) function. Atrial function can be assessed using speckle-tracking echocardiography. This study investigates the potential impact of ECG LVH on LA strain.

Methods: A total of 62 individuals diagnosed with LVH, based on the echocardiographic left ventricular mass index, were included. ECG LVH was assessed using established

protocols: the Sokolow-Lyon voltage criteria ($SV1 + RV5/RV6 > 35$ mm), Cornell voltage criteria ($RaVL + SV3 > 28$ mm for men and > 20 mm for women), and the Cornell product criteria [$(SV3 + RaVL + (\text{for women } 8 \text{ mm})) \times \text{QRS duration} > 2440$ mm x ms]. Participants were categorized into two groups based on the presence or absence of ECG LVH. The relationship between LA strain measures and ECG characteristics was explored.

Results: The study population had a median age of 58.3 ± 10.1 years, with 40.3% being female, 91.9% hypertensive, and 35.5% diabetic. Nineteen patients (30.6%) were identified with ECG LVH based on Sokolow-Lyon voltage, Cornell voltage, or Cornell product criteria. These patients exhibited significantly reduced LA reservoir, conduit, and contraction strains ($p < 0.001$). Statistically significant correlations were observed between all three phases of LA strain measures and Sokolow-Lyon voltage (reservoir $r = -0.389$, $p < 0.01$; conduit $r = -0.273$, $p < 0.05$; contraction $r = -0.359$, $p < 0.01$), Cornell voltage (reservoir $r = -0.49$, $p < 0.001$; conduit $r = -0.432$, $p < 0.001$; contraction $r = -0.339$, $p < 0.01$), and Cornell product (reservoir $r = -0.471$, $p < 0.001$; conduit $r = -0.387$, $p < 0.01$; contraction $r = -0.362$, $p < 0.01$).

Conclusions: ECG LVH is associated with impaired LA strain, validating its use as an effective tool for predicting LA dysfunction.

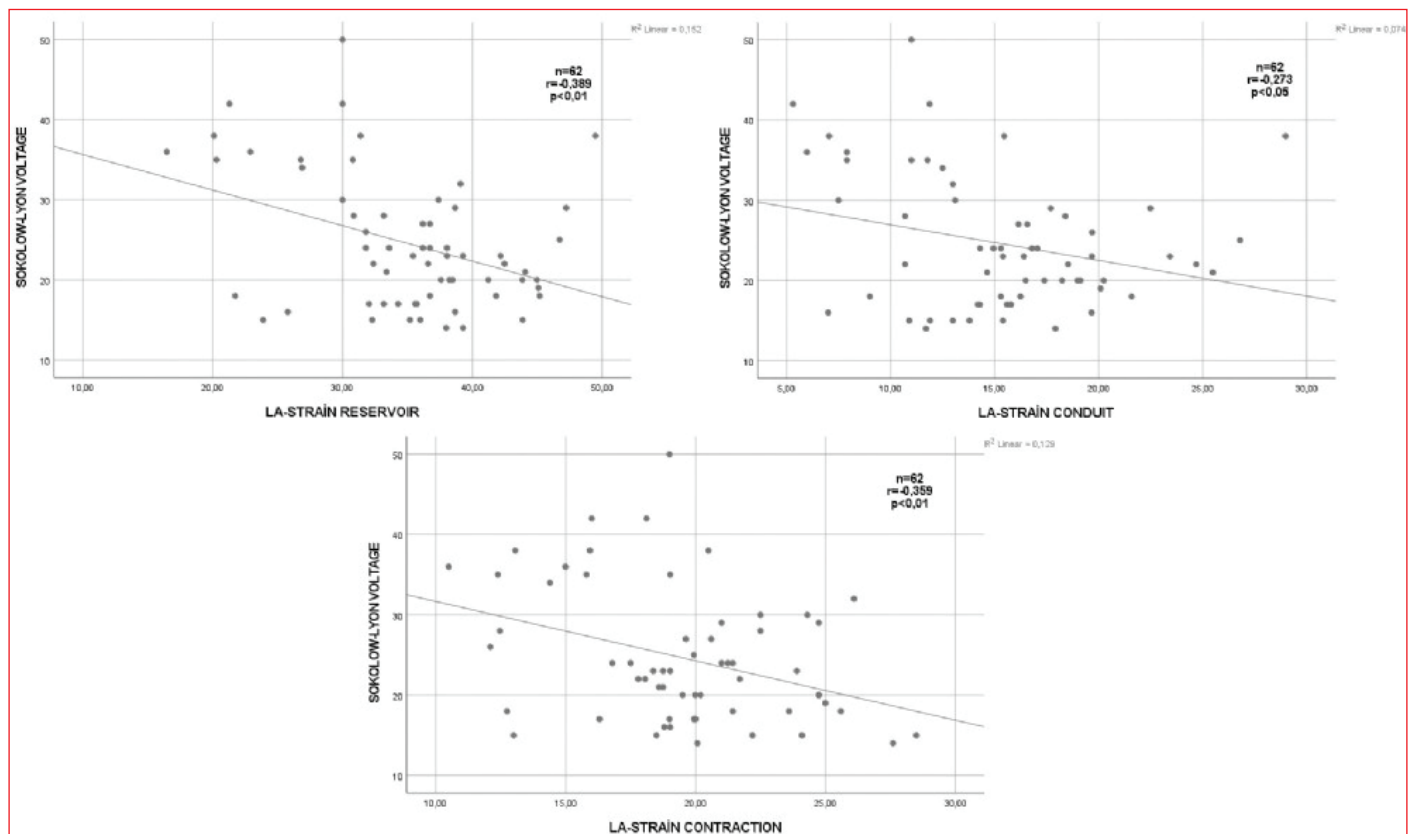


Figure 1. The correlation between Sokolow-Lyon voltage and LA strain values.

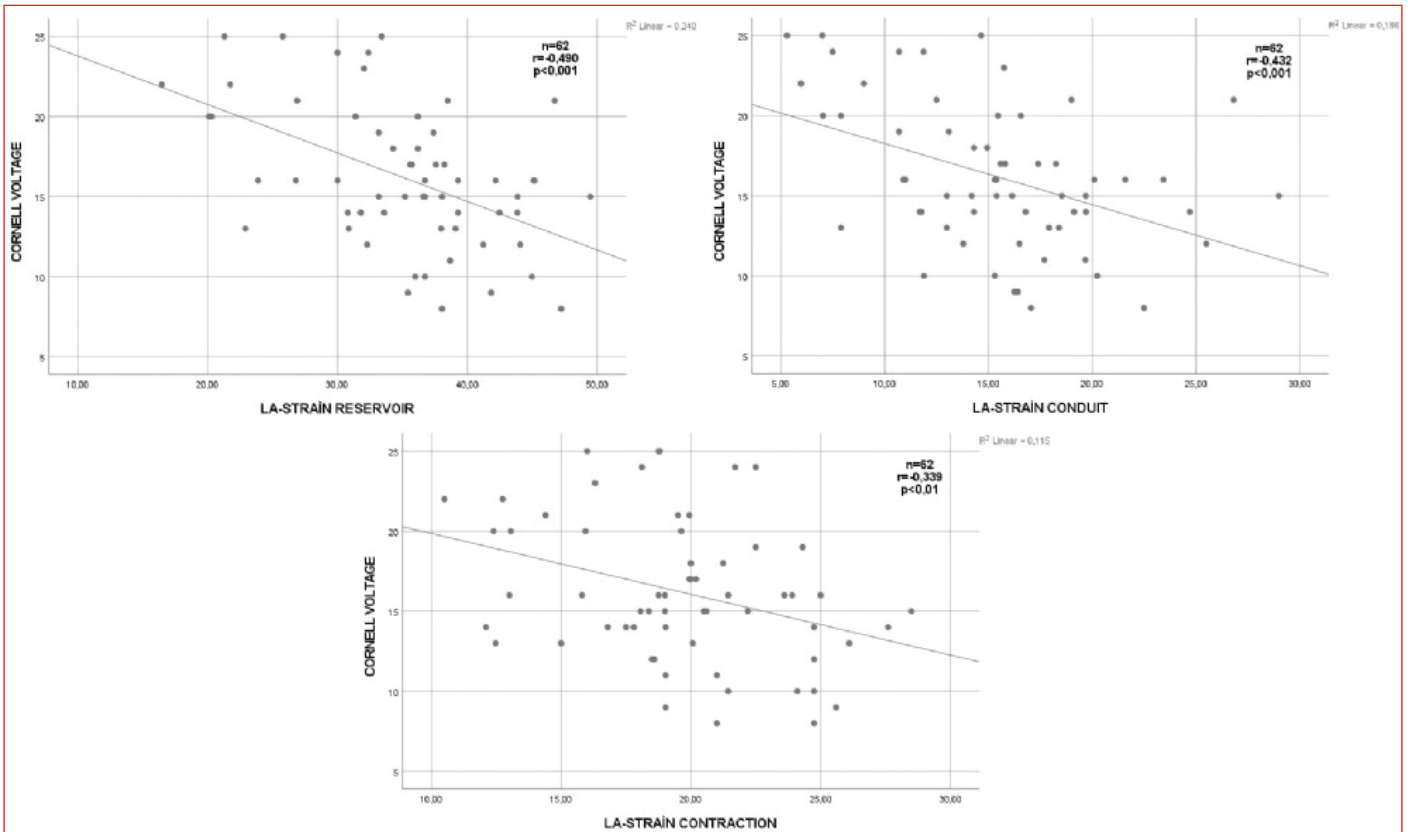


Figure 2. The correlation between Cornell voltage and LA strain values.

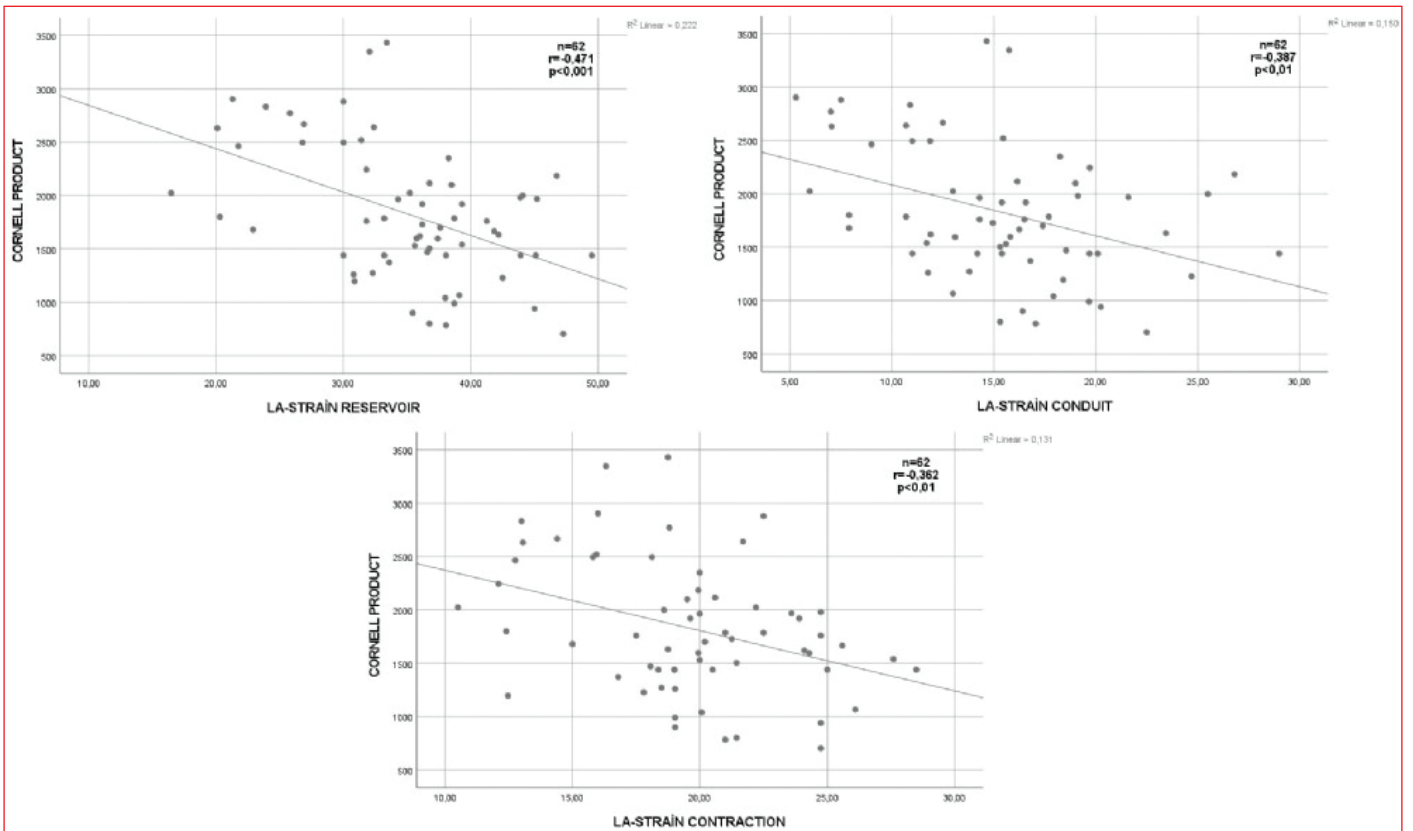


Figure 3. The correlation between Cornell product and LA strain values.

Cardiac Imaging / Echocardiography

PP-065

Prognostic significance of Tp-e interval in mitral valve prolapse and mitral annular disjunction

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Background and Aim: Recently studies highlighted various phenotypes of mitral valve prolapse (MVP) associated with clinically significant ventricular arrhythmias. In this context, the role of mitral annular dysjunction (MAD) is particularly important. The anatomical and arrhythmic substrate of MAD remains unclear. We evaluated the distribution of ventricular repolarization using Tp-e interval, Tp-e/QT and Tp-e/QTc ratio in patients with MAD and MVP and assessed their prognostic value.

Methods: Patients with MVP were retrospectively screened. Diabetes mellitus, coronary artery disease, hypertension, congenital heart disease, rheumatic heart disease, Marfan syndrome, atrial fibrillation, and patients whose ECG and 24-hour ambulatory ECG records could not be accessed were excluded. After the exclusion, patients were divided into two groups: those with MAD (age 35.5 ± 14.1 , n=28) and those without MAD (age 39.7 ± 16.1 , n=239). The two groups were compared with healthy controls (age 40.84 ± 12 , n=25). ECG, 24-hour ambulatory ECG, and transthoracic echocardiography were evaluated for all subjects. The standard 12-lead electrocardiograms were analyzed; QT, QTc, Tp-e, Tp-e/QT and Tp-e/QTc were calculated. Mortality, hospital admission, ventricular arrhythmia, heart failure and mitral valve surgery were determined as clinical outcome.

Results: There was no difference between the groups in terms of age. Female gender was more frequent in the patients with MAD group compared to those without MAD (89% vs. 61%; p=0.013). Mean MAD distance measured by echocardiography was 7.6 ± 2.1 mm. Tp-e interval (p<0.001), Tp-e/QT ratio (p<0.001) and Tp-e/QTc ratio (p=0.031) were significantly prolonged in MVP patients with MAD compared to those without MAD and the controls. The frequency of inferior lead T wave inversion (p=0.041) and premature ventricular contractions/24 hour were higher in patients with MAD compared to those without MAD and the controls (p=0.003). When primary clinical outcomes were examined, sudden cardiac death (p=0.003) and ventricular tachycardia (p=0.021) were more common in the patients with MAD compared to those without MAD. Mitral regurgitation severity (HR: 2.039, 95% CI 1.066-3.904, p=0.031) and Tp-e interval (HR: 1.103, 95% CI 1.018-1.194, p=0.016) were independent predictors of the primary clinical outcome. A Tp-e interval of >71 ms predicted the primary outcome with 72% sensitivity and 78% specificity (AUC 0.816, 95% CI 0.716-0.915, p<0.001). According to Kaplan-Meier survival analysis for 150 months, the patients with a Tp-e interval of <71 ms have higher risk for mortality than the patients with a Tp-e interval of ≥ 71 ms (p<0.001).

Conclusions: In MVP patients with MAD, Tp-e interval was prolonged and Tp-e/QT and Tp-e/QTc ratios were increased. Tp-e interval was an independent predictor of primary clinical outcome. ECG is a practical study to evaluate the prognosis and possible arrhythmias. Tp-e interval might be a useful marker of cardiovascular morbidity and mortality in MVP patients with MAD.

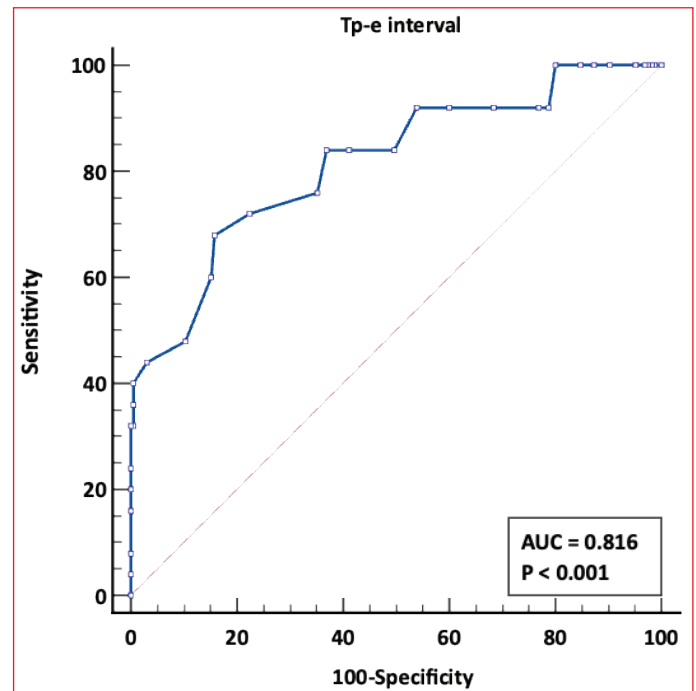


Figure 1. ROC curve analysis showing a Tp-e interval of >71 ms predicted the primary outcome with 72% sensitivity and 78% specificity.

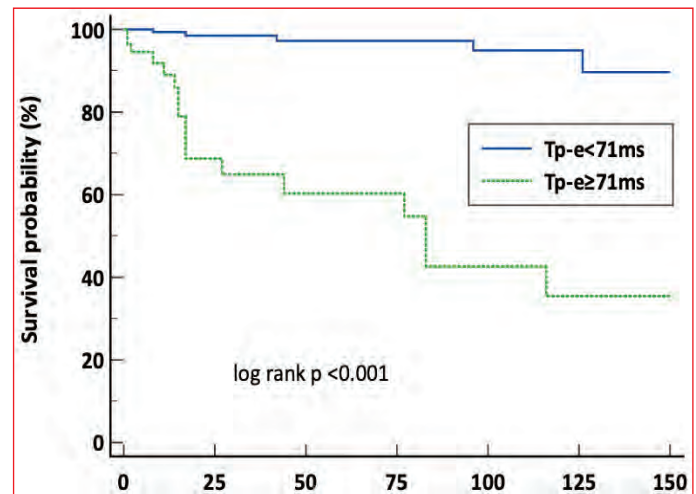


Figure 2. Kaplan-Meier survival analysis for 150 months showing the patients with a Tp-e interval of <71 ms have higher risk for mortality than the patients with a Tp-e interval of ≥ 71 ms.

Cardiac Imaging / Echocardiography

PP-066

Low heart rate variability is associated with cerebrovascular accident in patent foramen ovale

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Background and Aim: The clinical implications of patent foramen ovale (PFO) and its relationship with cerebrovascular accident (CVA) are still under debate. Demonstrating a certain association between PFO and CVA is challenging. This observational study intended to explore relevant associations between HRV parameters and CVA in patients with PFO.

Methods: We included 164 patients with PFO detected by transesophageal echocardiography (TEE) in our study. Two or more of the following features were considered as high anatomic risk PFO: long-tunnel PFO ≥ 10 mm, hypermobile interatrial septum or interatrial septal aneurysm, prominent eustachian valve or Chiari's network, large RL shunt during Valsalva maneuver, low-angle PFO ≤ 10 . Patients were divided into two groups as those with cryptogenic stroke or transient ischemic attack (symptomatic) and those without (asymptomatic). Clinical, echocardiographic, HRV parameters with 24 h rhythm Holter ECG of the patients were compared.

Results: Patients who underwent TEE due to CVA and were found to have PFO (n=44, age: 46.3 ± 16.1) were compared with consecutive asymptomatic patients with incidental PFO found with TEE (n=120, age: 49.6 ± 17.1). Age, gender and BMI were similar between the two groups. While the frequency of hypertension, diabetes and coronary artery disease was similar, smoking was higher in the symptomatic group (40% vs. 19%, p=0.007). The presence of high anatomic risk PFO was higher in the symptomatic group (51% vs. 23%, p<0.001). When 24 h Holter ECG findings were compared, SDNN 24 h, SDANN index, SDNN index, Rmssd, VLF and HF (p<0.001, p<0.001, p<0.001, p<0.001, p=0.015; respectively) of HRV parameters were significantly lower in the symptomatic PFO group. In multivariate logistic regression analysis, VLF, Rmssd, LF and smoking were found to be independent predictors of CVA (p<0.001, p=0.018, p=0.002, p=0.039; respectively). VLF <1020 predicted CVA with the highest AUC value, 78% specificity and 73% sensitivity (AUC 0.779 95% CI 0.694-0.863).

Conclusions: Low heart rate variability was independently associated with the risk of having a cerebrovascular accident in patients with patent foramen ovale.

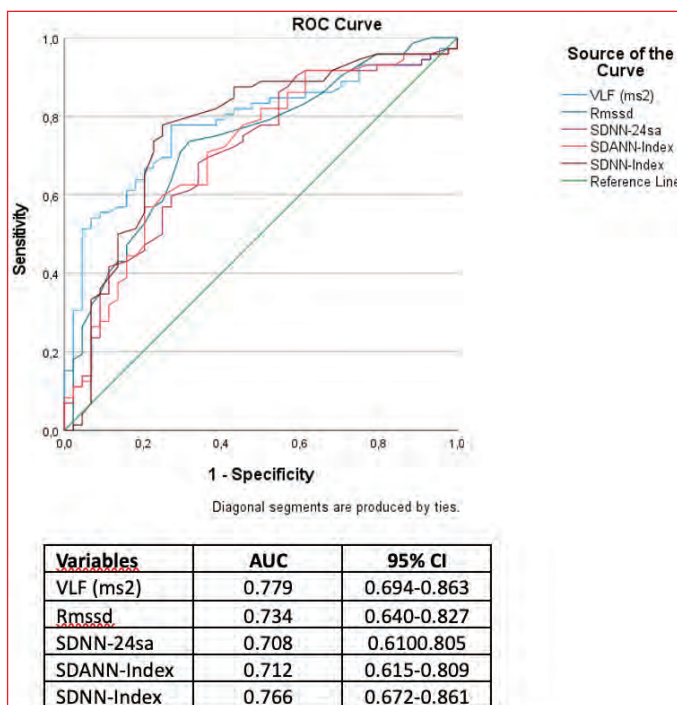


Figure 1. ROC curve analysis showing the specificity and sensitivity of HRV parameters predicting CVA.

Cardiac Imaging / Echocardiography

PP-070

Left ventricular dysfunction in patients with ulcerative colitis

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Background and Aim: Although ulcerative colitis (UC) resembles other inflammatory disorders, cardiac effects have not been fully studied. The objective of our research was to assess left ventricular (LV) function in individuals with UC using tissue doppler imaging (TDI).

Methods: We conducted a cross-sectional case protocol study between December 2021 and March 2022. In this study, 50 UC patients (24 females and 26 men) were age- and sex-matched with healthy volunteers. Detailed transthoracic echocardiography (TTE) and TDI measured systolic and diastolic function.

Results: UC patients had reduced early/late diastolic myocardial velocities, mitral annular plane systolic excursion, and end-diastolic distance from the mitral annulus to the LV apex compared to controls. Higher systolic myocardial velocity (Sm), isovolumetric relaxation time, and displacement index were observed (p<0.001). The correlation coefficient between echocardiographic parameters and UC was meaningful (p<0.001).

Conclusions: The results of our study indicate that individuals with UC saw a decline in their TDI values. The use of TDI has the potential to detect and assess early functional LV abnormalities in UC.

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

	Controls (n=50)	Patients with UC (n=50)	p value
Age, years	36.9 ± 5.5	37.4 ± 5.6	0.902
Gender, male, n (%)	24 (48)	26 (52)	0.364
BSA (m ²)	1.76 ± 0.15	1.78 ± 0.14	0.503
Diabetes (n)	3	5	0.482
Hypertension (n)	4	7	0.672
Hyperlipidemia (n)	5	7	0.424
Smoking (n)	7	10	0.529
Topical 5-ASA (n, %)	-	24.5	-
Oral 5-ASA (n, %)	-	34.0	-
Salazopyrin (n, %)	-	4.8	-
Corticosteroids (n, %)	-	10.2	-
Anti-TNF/AZA (n, %)	-	11.2	-
Mayo	-	2.6 ± 2.1	-
Duration (years)	-	3.0	-

Table 2. Echocardiographic parameters of the study population

	Controls (n=50)	Patients with UC (n=50)	p value
Ejection fraction, %	60.2 ± 2.6	59.8 ± 2.4	0.856
Ejection time, ms	277.3 ± 30.2	282.4 ± 30.0	0.698
LVEDD, mm	43.7 ± 0.3	43.1 ± 0.5	0.798
LVESD, mm	30.7 ± 0.2	31.2 ± 0.2	0.556
IVS, mm	11.4 ± 0.5	11.7 ± 0.4	0.899
PW, mm	9.0 ± 0.1	8.9 ± 0.1	0.897
LAD, mm	31.4 ± 2.1	37.2 ± 2.5	<0.001
E, cm/s	74.2 ± 8.4	78.5 ± 7.4	0.081
A, cm/s	50.8 ± 7.4	79.4 ± 8.7	<0.001
E/A	1.42 ± 0.1	1.08 ± 0.2	<0.001
dT, ms	183.1 ± 15.0	189.2 ± 18.0	0.262
IVRT, ms	78.5 ± 4.7	94.2 ± 7.3	<0.001
IVCT, ms	41.7 ± 5.0	42.0 ± 5.8	0.754
MAPSE, cm	1.58 ± 0.2	1.22 ± 0.1	<0.001
Em, cm/s	13.0 ± 1.2	7.2 ± 1.2	<0.001
Am, cm/s	8.5 ± 1.6	8.7 ± 1.5	0.758
E/Em	5.1 ± 1.2	9.6 ± 2.2	<0.001
Sm, cm/s	5.7 ± 1.1	9.5 ± 1.4	<0.001
LVMI, g/m	115.2 ± 14.8	120.2 ± 15.8	0.293
DI, %	0.34 ± 0.02	0.56 ± 0.10	<0.001
S-VTI, cm	1.56 ± 0.12	1.62 ± 0.15	0.656
L0, cm	5.72 ± 0.29	4.22 ± 0.28	<0.001

Table 3. Correlations between ulcerative colitis and echocardiographic parameters

E/A	-0.642	<0.001
IVRT	0.784	<0.001
MAPSE	-0.552	<0.001
E/Em	0.742	<0.001
Sm	0.884	<0.001
DI	-0.646	<0.001
L0	-0.522	<0.001

Cardiac Imaging / Echocardiography

PP-071

Evaluation of the myocardial performance index in coronary slow flow patients with and without coronary artery ectasia

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Background and Aim: Late clearance of epicardial coronary arteries during angiographic imaging without significant stenosis of the coronary arteries is defined as coronary slow flow (CSF). Coronary artery ectasia (CAE) is among secondary reasons of CSF which causes similar clinical and angiographic findings. Its pathophysiological mechanisms remains unclear. However, in some studies, it has been suggested that endothelial dysfunction and microvascular dysfunction are responsible. Myocardial performance index (MPI) is defined as an alternative method that quantitatively reflects global ventricular function. In this study, we aimed to evaluate the parameters associated with echocardiographically biven-tricular functions in CAE patients with or without CSF.

Methods: The study we included 60 patients (25 females, 35 males; mean age 59 ± 7) who underwent coronary angiography and had CSF in at least one coronary artery according to the TIMI frame count method. The patients were randomized as 32 CSF patients with CAE (13 women, 19 men; mean age 60 ± 6) and 28 CSF patients without CAE (12 women, 16 men; mean age 58 ± 7). What's more, a control group of 27 people (19 women, 8 men; mean age 59 ± 10) who were similar in terms of age, gender and cardiac risk factors and whose coronary arteries and coronary blood flow were found to be normal, was formed for comparison. Pulse wave and tissue Doppler echocardiography were performed to measure systolic and diastolic parameters on the left and right ventricles of the patients.

Results: The corrected TIMI frame count was significantly higher for all three coronary arteries in the patient groups (p<0.01). Left ventricle isovolumic relaxation (IVRT), contraction time (IVCT) and MPI values were statistically higher in the patient groups significantly compared to the control group in pulse wave Doppler examination. Geç diyastolik doluş mitral annüler dalga (Aa) wave, early diastolic filling mitral annular

wave (Ea)/Aa, and early diastolic peak flow velocity(E)/Ea ratio were similar among the groups in left ventricle septal wall tissue Doppler echocardiography examination. However, the Em wave was lower in the CSF and CAE groups compared to the control group ($p<0.05$). On the other hand, MPI was statistically higher in both patient groups compared to the control group ($p<0.05$). There were no significant differences between the groups in terms of right ventricular tissue Doppler echocardiography values (IVRT, ET, MPI).

Conclusions: In our study, we found that there was greater deterioration in the MPI value, an indicator of global left ventricular (LV) functions, in patients with slow coronary flow, independent of coronary ectasia. However, there were no significant differences between the groups in terms of right

ventricular tissue Doppler echocardiography values (IVRT, ET, MPI).

Table 1. Comparison of TIMI frame counts between groups

Variables	CSF and CAE (n=32)	CSF (n=28)	Control (n=27)	p value
LAD (CTFC)	31 ± 11	36 ± 17	21 ± 7	<0.01
LAD (TFC)	53 ± 18	60 ± 30	36 ± 11	<0.01
CX (TFC)	28 ± 8	31 ± 14	15 ± 4	<0.01
RCA (TFC)	33 ± 15	41 ± 18	18 ± 3	<0.01

LAD: Left anterior descending artery; Cx: Circumflex artery; RCA: Right coronary artery; CAE: Coronary artery ectasia; CSF: Coronary slow flow; CTFC: Corrected TIMI frame count.

Table 2. Echocardiographic parameters comparison between groups

Variables	CSF and CAE (n=32)	CSF (n=28)	Control (n=27)	p value
LV EF, %	65.3 ± 2.3	65.8 ± 1.0	65.5 ± 1.8	0.56
LV EDD, mm	44.7 ± 3.8	45.3 ± 3.6	43.5 ± 3.5	0.20
LV ESD, mm	27.8 ± 4.0	27.2 ± 3.3	26.3 ± 3.2	0.28
Left Atrium, mm	34.3 ± 2.7	34.3 ± 2.2	32.9 ± 2.8	0.07
Mitral E Wave, m/s	0.75 ± 0.22	0.73 ± 0.14	0.75 ± 0.12	0.87
Mitral A Wave, m/s	0.84 ± 0.26	0.78 ± 0.17	0.74 ± 0.15	0.15
Mitral E/A Ratio	1.0 ± 0.5	1.0 ± 0.3	1.0 ± 0.3	0.69
Mitral DZ, ms	206 ± 30	218 ± 37	212 ± 30	0.08
Septal Ea Wave, cm/s	9.2 ± 1.4*	9.7 ± 1.5	10.5 ± 1.8	<0.05
Septal Aa Wave, cm/s	9.8 ± 1.9	9.5 ± 1.8	10 ± 1.0	0.56
Septal Ea/Aa Ratio	0.97 ± 0.20	1.04 ± 0.19	1.05 ± 0.16	0.18
Septal E/Ea Ratio	8.2 ± 2.2	7.6 ± 1.1	7.2 ± 0.8	0.06
Septal Sa Wave, cm/s	8.8 ± 1.4	9.1 ± 1.2	8.9 ± 0.8	0.57
LV IVRT, ms	98 ± 11*	90 ± 13W	83 ± 10	<0.05
LV IVCT, ms	50 ± 6*	50 ± 4*	45 ± 3	<0.05
LV EZ, ms	273 ± 31*	273 ± 29*	295 ± 25	<0.05
LV MPI	0.54 ± 0.07*	0.52 ± 0.07*	0.43 ± 0.05	<0.05
LV IVRTa, ms	95 ± 9*	89 ± 12*	82 ± 9	<0.05
LV IVCTa, ms	49 ± 6	50 ± 5	47 ± 3	0.09
LV EZa, ms	270 ± 29*	271 ± 25*	295 ± 22	<0.05
LV MPIa	0.53 ± 0.06*	0.51 ± 0.06*	0.44 ± 0.05	<0.05
RV IVRTa, ms	84 ± 13	76 ± 10W	82 ± 10	<0.05
RV IVCTa, ms	48 ± 4	47 ± 4	49 ± 3	0.18
RV EZa, ms	293 ± 34	283 ± 29	292 ± 31	0.12
RV MPIa	0.45 ± 0.05	0.44 ± 0.04	0.45 ± 0.04	0.47
RV Thickness, mm	2.8 ± 0.4*	2.7 ± 0.4*	2.4 ± 0.5	<0.05
TAPSE, mm	22.9 ± 1.8	22.5 ± 1.9	22.0 ± 1.3	0.09

A: Late diastolic filling; Aa: Late diastolic filling mitral annular wave; E: Rapid early diastolic filling; Ea: Early diastolic filling mitral annular wave; EZ: Ejection time; IVCT: Isovolumetric contraction time; IVRT: Isovolumetric relaxation time; CAE: Coronary artery ectasia; CSF: Coronary slow flow; MPI: Myocardial performance index; Sa: Systolic mitral annular wave; SV EF: Left ventricular ejection fraction; SV ED: Left ventricular end-diastolic diameter; SV ES: Left ventricular end-systolic diameter; RV: Right ventricle; LV: Left ventricle; TAPSE: Tricuspid annular plane systolic excursion; TDI: Tissue doppler imaging. *: $p<0.05$ compared to control group W: $p<0.05$ compared to the slow flow group with coronary ectasia

Cardiac Imaging / Echocardiography

PP-072

The effects of ferric carboxymaltose treatment on left ventricle functions during the acute treatment period

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Background and Aim: Guidelines recommend routine use of parenteral ferric carboxymaltose (FCM) therapy for patients with heart failure (HF) and iron deficiency anemia (IDA). However, the effects of iron treatment on myocardial functions in the acute period have not been clearly demonstrated yet. The present study aims to examine the acute effects of parenterally administered FCM treatment on myocardial functions in patients with acute HF.

Methods: The study population consisted of patients admitted with a diagnosis of acute HF and IDA. All patients underwent baseline echocardiographic examination and then parenteral FCM was administered. After the FCM treatment, all patients underwent echocardiographic examination again and the effects of FCM treatment on the echocardiographic results were examined.

Results: The mean age of the patients was found to be 56.7 ± 5.1 years, and 75.4% of the patients were male. Before FCM treatment, left ventricular ejection fraction (LVEF) was found to be 32.1%, and LVEF was found to be 34.8% after the FCM treatment. In the control echocardiographic examination, LV-EF (p=0.042), systolic pulmonary artery pressure (p=0.014), e/e' (p=0.035), left ventricular global longitudinal strain (p=0.011), strain rate S (p=0.050) and strain rate E (p=0.007) were found to be statistically significantly higher after the administration of the treatment.

Conclusions: FCM treatment yielded a significant improvement in myocardial functions in the acute period. In addition to the critical role of iron in hemoglobin production in red blood cells, iron plays an important role in metabolism since it also has other roles in many other enzymatic steps.

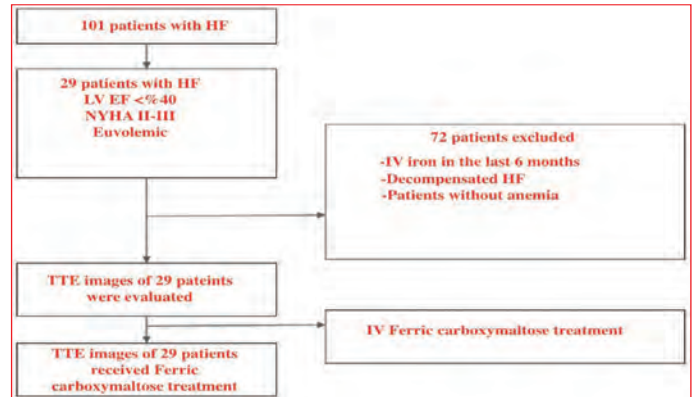


Figure 1. Study flow diagram.

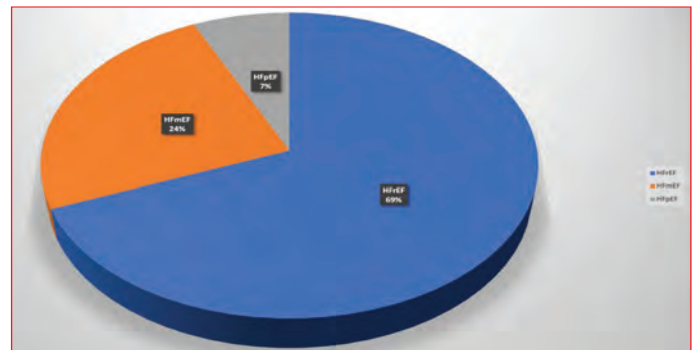


Figure 2. The distribution of the study population according to HF groups.

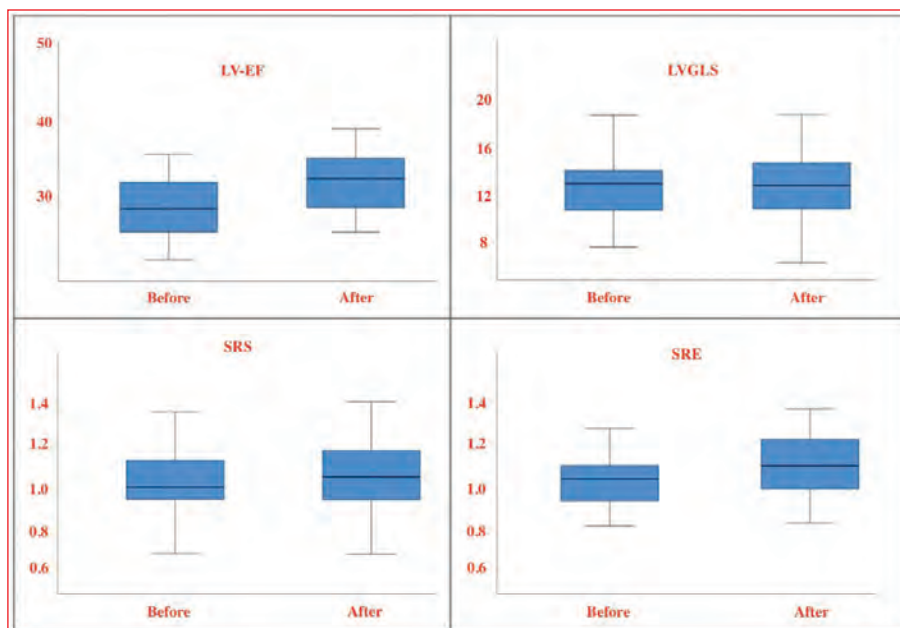


Figure 3. Box graph of echocardiographic parameters.

Cardiac Imaging / Echocardiography

PP-073

Cardiac effects of inflammation in rheumatoid arthritis and spondyloarthritis patients

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Background and Aim: Rheumatoid arthritis (RA) and ankylosing spondylitis (AS) are common diseases in the community with chronic inflammation. In addition to joint involvement, extra-articular involvement can also be seen in both groups of diseases. One of the most important of these is cardiac involvement. In addition to direct cardiac involvement, rheumatological diseases may also result in cardiac damage due to increased inflammation. This inflammatory damage can be observed in diseases with different mechanisms, albeit exhibiting similar characteristics. Myocardial performance index (MPI) is a parameter that can be easily measured by Doppler echocardiography, obtained by dividing the sum of isovolumetric relaxation time (IVRT) and isovolumetric contraction time (IVHR) by ejection time (EZ). It is important for the evaluation of cardiac functions. In this study, our aim is evaluate the cardiac involvement in the active inflammation period in patients with RA and AS, and evaluate the change in cardiac involvement with the suppression of inflammation after the treatment echocardiographically and compare it with the pre-treatment.

Methods: The study involved 30 newly diagnosed, active RA and 31 AS patients in need of biological treatment. The patients were evaluated by the same cardiologist using pulse wave and Tissue doppler echocardiography at the beginning of the study and after 3 months of treatment. Myocardial performance index (MPI), a parameter that can provide information about both systolic and diastolic functions and can be easily measured by echocardiography, was used to evaluate the cardiac effects of inflammation.

Results: MPI values were found to be higher than normal values in both groups during the active disease period (when inflammation was high). These values were determined as 0.5 ± 0.07 for RA patients and 0.51 ± 0.1 for AS patients. Although disease activity decreased in the 3rd month of treatment in both patient groups, no change was detected in MPI values and the values remained above the normal range, similar to pre-treatment values. In a study including 46 patients diagnosed with RA, in which DDE findings were compared, a shortening of the Ea wave, a lengthening of the Aa wave, and a lower Ea/Aa ratio were found in RA patients compared to the control group, and with these results, it was thought that diastolic dysfunction was present in RA patients. In our study, DDE showed that diastolic dysfunction was present at the time of diagnosis, and in the evaluation made after treatment, diastolic dysfunction continued despite the regression in inflammatory values and joint findings.

Conclusions: Inflammation-induced cardiac involvement may develop in both RA and AS patients, despite their different pathophysiologic pathways. Longer follow-up periods

are necessary for the improvement of inflammation-induced diastolic dysfunction and MPI values compared to joint findings used in activation parameters.

Table 1. General echocardiography and pulse wave Doppler echocardiography findings in RA and AS patients

Features	RA	AS	p
	Average \pm SS (n=30)	Average \pm SS (n=31)	
IVGZ Before Treatment	85.13 \pm 10.93	86.52 \pm 11.4	0.631
IVGZ After Treatment	88.27 \pm 9.96	86.03 \pm 11.39	0.419
IVKZ Before Treatment	49.7 \pm 9.27	50.58 \pm 12.12	0.752
IVKZ After Treatment	46.63 \pm 10.31	49.1 \pm 9.64	0.339
EZ Before Treatment	272.87 \pm 31.31	274.23 \pm 35.33	0.874
EZ After Treatment	266 \pm 27.69	263.81 \pm 25.08	0.747
MPI Before Treatment	0.5 \pm 0.07	0.51 \pm 0.1	0.697
MPI After Treatment	0.51 \pm 0.08	0.52 \pm 0.07	0.761

*: Significant at 0.05 level according to independent group Student's t-test RA: Rheumatoid arthritis; AS: Ankylosing spondylitis; IVGZ: Isovolumetric relaxation time; IVKZ: Isovolumetric contraction increase; EZ: Ejection time; MPI: Myocardial performance index

Table 2. Tissue Doppler Echocardiography findings in RA and AS patients

Features	RA	AS	p
	Average \pm SS (n=30)	Average \pm SS (n=31)	
Ea Before Treatment	9.54 \pm 2.41	9.2 \pm 3.2	0.638
Ea After Treatment	8.63 \pm 2.24	9.25 \pm 2.51	0.320
Aa Before Treatment	8.84 \pm 1.93	8.12 \pm 3.01	0.276
Aa After Treatment	10.87 \pm 13.14	7.51 \pm 2.53	0.168
Ea/Aa Before Treatment	1.14 \pm 0.38	1.2 \pm 0.38	0.521
Ea/Aa After Treatment	1.09 \pm 0.47	1.37 \pm 0.58	0.044*
DDE MPI Before Treatment	0.54 \pm 0.27	0.49 \pm 0.11	0.324
DDE MPI After Treatment	0.5 \pm 0.08	0.5 \pm 0.09	0.951

*: Significant at 0.05 level according to independent group Student's t-test RA: Rheumatoid arthritis; AS: Ankylosing spondylitis; Ea: Early diastolic filling mitral annular wave; Aa: Late diastolic filling mitral annular wave; DDE-IVGZ, IVKZ, MPI: Isovolumetric relaxation time, isovolumetric contraction time and myocardial performance index measured by tissue Doppler echocardiograph.

Coronary Artery Disease / Acute Coronary Syndrome

PP-077

Effect of meteorological parameters on the frequency of acute myocardial infarction

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Background and Aim: Environmental factors (temperature, air pollution, humidity, wind, etc.) directly affect the frequency of acute myocardial infarction. This relationship has been shown in both observational and epidemiological studies. Weather events such as a sudden drop or sudden rise in air temperature affect this frequency. In this study, we aimed

to investigate the effects of meteorological parameters on the frequency of acute myocardial infarction.

Methods: In this retrospective study, a total of 5234 patients who were admitted to Amasya University Faculty of Medicine SSEAH with a diagnosis of acute myocardial infarction (AMI) and underwent emergency coronary angiography between January 2018 and February 2024 were evaluated. Meteorological parameter values such as maximum temperature (°C), minimum temperature (°C), average temperature (°C), temperature difference (°C), maximum relative humidity (%), minimum relative humidity (%), average relative humidity (%), maximum current pressure (hPa), minimum current pressure (hPa), average current pressure (hPa) were obtained from Amasya Meteorology Directorate. Data were analyzed with IBM SPSS V23 and R program. Analysis results are presented as mean \pm standard deviation and median (minimum-maximum). The significance level was taken as $p < 0.050$.

Results: Over a period of approximately 6 years, 5234 patients with a diagnosis of acute myocardial infarction were included in the study. It was observed that a total of 1503 patients in winter, 1237 in spring, 1244 in summer and 1250 in autumn underwent coronary angiographic procedures with the diagnosis of acute myocardial infarction (STEMI, NSTEMI, USAP) (Table). It was determined that there was a significant increase in the frequency of AMI, especially in the winter season when the weather was significantly cold. In the correlation analyses, it was seen that there was a statistically significant positive moderate relationship ($r=0.542$; $p=0.014$) between the minimum temperature and USAP number; and a mild-moderate relationship ($r=-0.459$; $p=0.042$) between the temperature difference and USAP number in the winter season (Figure). In the summer season, a statistically significant positive moderate relationship was observed between the mean actual pressure (hpa) and the number of NSTEMIs ($r=0.500$; $p=0.035$); When all variables are compared, one unit increase in the temperature difference reduces the USAP number by 1.116 units ($p < 0.001$), while one unit increase in the minimum actual pressure (hpa) value reduces the USAP number by 0.540 units ($p=0.021$).

Conclusions: Among the meteorological parameters investigated in our study, it was observed that variables related to air temperature in particular were closely related to the risk of acute myocardial infarction. The frequency of AMI increases especially in winter months when the average temperature is low. Establishing awareness to avoid long-term exposure to cold weather will be extremely beneficial in reducing the risk of low temperature-related AMI.

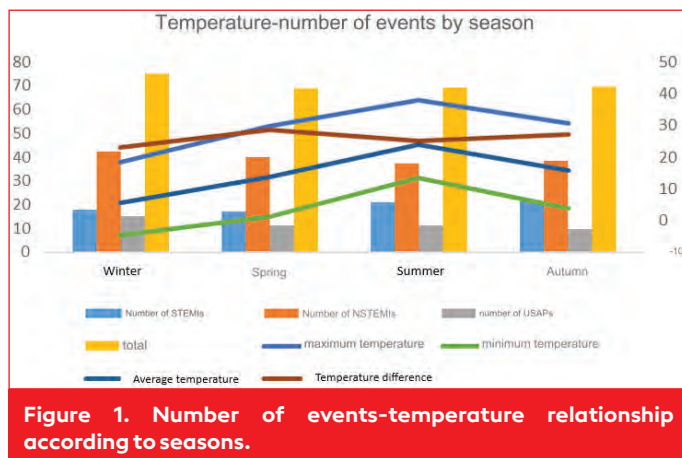


Figure 1. Number of events-temperature relationship according to seasons.

Table 1. Number of events by season on an annual basis

Year/Season	Winter	Spring	Summer	Autumn
2018	187	135	154	153
2019	280	241	233	233
2020	244	158	141	151
2021	142	211	207	189
2022	168	208	233	260
2023	250	284	276	264
2024	232			
TOTAL	1503	1237	1244	1250
Total Patient	STEMI 1423	NSTEMI 2932	USAP 879	5234

Coronary Artery Disease / Acute Coronary Syndrome

PP-078

Age shock index as an early predictor of atrial fibrillation in acute coronary syndrome patients

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Background and Aim: The development of atrial fibrillation (AF) is a common occurrence in the process of acute coronary syndrome. It has been demonstrated that in patients with acute coronary syndrome complicated by AF, there is an increased risk of sudden, short-term, or long-term mortality, heart failure, impaired left ventricular function, a decrease in quality of life, and an elevated incidence of thromboembolic events. In our study, we examined the predictive abilities of the "bedside shock index (SI)", "modified SI (MSI)", "age-SI (A-SI)", and "age-MSI" indices for the development of AF in individuals hospitalized due to acute coronary syndrome.

Methods: This study included patients diagnosed with acute coronary syndrome who underwent primary percutaneous coronary intervention (PCI) at a tertiary cardiac center. The Bedside SI was calculated as the ratio of heart rate (HR) to systolic blood pressure (SBP). A-SI was calculated as the product of age and SI. MSI was calculated as the ratio of HR to mean arterial pressure (MAP), where MAP is calculated using the formula $(2 \times \text{diastolic blood pressure} + \text{SBP}) / 3$. Age-MSI (A-MSI) was calculated as the product of age and MSI. During their hospital stay, patients were monitored for the development of AF. Patients were divided into two groups based on the development or non-development of AF during the follow-up.

Results: A total of 463 patients were included out of which 78.8% ($n=365$) were male and mean age was 50.3 ± 16.8 years. Other demographic, laboratory and clinical characteristics are presented in Table 1. In-hospital AF was observed in 4.4% ($n=20$) patients. The AF patient group exhibited higher 1-year all-cause mortality rates [5 (25%) vs. 34 (7.7%) $p=0.020$], along with prolonged periods of intensive care

and total hospitalization. The ROC curve analysis for A-SI (AUC: 0.705, sensitivity 65.0%, specificity 76.3%, p=0.001), and A-MSI (AUC: 0.700, sensitivity 75.0%, specificity 63.0%, p=0.001), predicting AF are shown in Figure 1.

Conclusions: A-SI and A-MSI can be used to determine the risk of developing AF in hospital for patients with acute coronary syndrome. These indices can be considered effective screening tools in identifying high-risk patients.

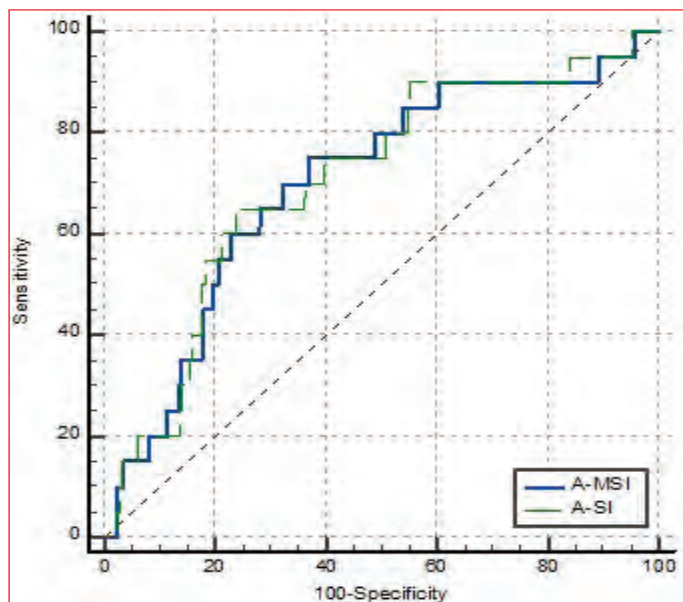


Figure 1. The ROC curve analysis for A-SI and A-MSI.

Table 1. Clinical features of acute coronary syndrome according to AF

	AF (-) (n=443)	AF (+) (n=20)	p value
Age, years, ± SD	499 ± 16.7	57.7 ± 17.8	0.045
Male, n (%)	348 (78.6)	17 (85.0)	0.779
HT, n (%)	150 (33.9)	7 (35.0)	0.916
DM, n (%)	109 (24.6)	4 (20.0)	0.793
Smoking, n (%)	248 (56.2)	4 (20)	0.001
HR, beats/minute, (IQR)	80 (74-91)	84 (76-116)	0.082
Hemoglobin, g/dL, ± SD	14.9 ± 1.7	14.0 ± 2.2	0.016
Creatinine, mg/dL, (IQR)	0.9 (0.7-1.1)	1.0 (0.8-1.2)	0.075
LVEF, %, ± SD	50.7 ± 10.2	46.8 ± 13.8	0.229
SI, (IQR)	0.65 (0.58-0.79)	0.75 (0.59-1.12)	0.078
MSI, (IQR)	0.91 (0.80-1.08)	1.03 (0.83-1.47)	0.097
A-SI, (IQR)	31.3 (24.2-42.3)	47.0 (32.3-53.0)	0.002
A-MSI, (IQR)	42.8 (32.9-59.5)	62.9 (45.7-75.4)	0.003
Coronary Angiography Duration, minute, (IQR)	32 (25-40)	35 (26-54)	0.316
Intensive care stay, (IQR)	2.0 (2.0-3.0)	3.0 (2.0-9.0)	0.017
Service stay, (IQR)	2.0 (1.0-2.0)	2.5 (1.0-4.5)	0.056
Total length of stay, ± SD	4.0 (3.0-5.0)	5 (3.0-9.5)	0.013
1 year all-cause mortality, n (%)	34 (7.7)	5 (25)	0.020

Coronary Artery Disease / Acute Coronary Syndrome

PP-082

Predictive value of adiponectin on long term MACE in STEMI patients

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Background and Aim: Decreased level of adiponectin is known to identified patients at risk of adverse left ventricular remodeling and major adverse cardiac events (MACE). We evaluated long term MACE after ST-segment elevation myocardial infarction (STEMI) according to level of adiponectin.

Methods: Prospective study population consisted of 73 consecutive patients with STEMI of onset < 12 h who underwent primary percutaneous coronary intervention (pPCI). The levels of creatine kinase (CK), the MB fraction of creatine kinase (CK-MB), troponin I, adiponectin, CRP, and other routine laboratory parameters were measured. Measurement of adiponectin is done during hospitalization with enzyme-linked immunosorbent assay (ELISA) method (Phoenix Pharmaceuticals, Belmont, CA, USA). The study subjects were divided in two groups according to the level of serum adiponectin.

Results: The follow-up period was 12 months. In total 22 (30.12%) patients suffered from MACE, 18 (24.65%) among patients with adiponectin value ≤1.8 ng/mL as well as 4 (5.47%) to those with value >1.8 ng/mL (p<0.013) (Table 1). Kaplan-Meier curves were used to show the number of MACE and the proportion of patients that survived at each even time point based on the cut-off value of adiponectin during hospitalization (1.8 ng/mL) (Figure 1). The log-rank test for the difference in survival resulted in a p value of 0.013. A receiver operating characteristics (ROC) curve plots the true positive rate against the false positive rate at different cut-off points, AUC: 0.77 (95% CI, 0.66-0.89), p=0.01 (Figure 2). Table 2 presents the area under the curve for biomarkers (troponin I, creatine kinase, creatine kinase MB, adiponectin and C-reactive protein).

Conclusions: The cut-off value for the adiponectin levels measured during hospitalization (1.8 ng/mL) identified patients at risks for MACE.

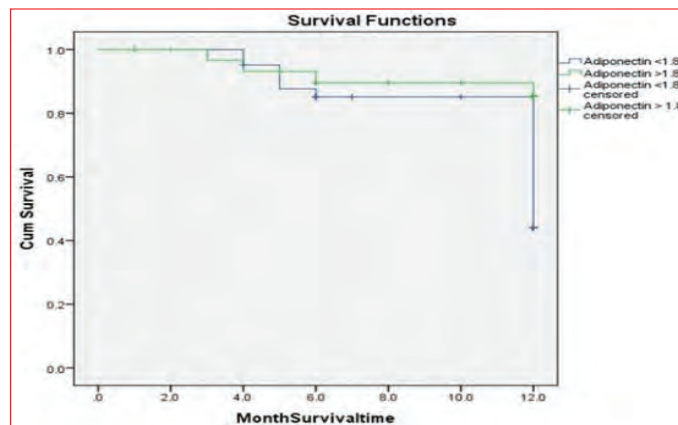


Figure 1. Kaplan-Meier estimates showing higher rates of MACE recurrence among patients with adiponectin ≤1.8 compared to higher adiponectin levels >1.8 (p=0.013).

Table 1. Baseline characteristics of patients

Characteristics	Adiponectin ≤ 1.8 ng/mL (n=41)	Adiponectin >1.8 ng/mL (n=32)	p value
Age (year), mean (\pm SD)	59.68 (\pm 10.68)	60.14 (\pm 11.75)	0.78
Gender (male), n (%)	20 (27.39)	25 (34.24)	0.42
Body mass index, mean (\pm SD)	26.66 (3.57)	29.00 (4.05)	0.003
Medical history			
Hypertension, n (%)	18 (24.65)	25 (34.24)	0.99
Cholesterol, mean (\pm SD)	5.15 (1.09)	5.37 (1.07)	0.24
Smoking, n (%)	16 (21.91)	21 (28.76)	0.91
Ejection fraction, mean (\pm SD)	51.59 (\pm 9.19)	53.34 (\pm 8.69)	0.64
MACE, n (%)	18 (24.65)	4 (5.47)	0.013
Laboratory values			
Haemoglobin, mean (\pm SD)	137.68 (\pm 14.82)	135.61 (\pm 14.41)	0.80
Creatine kinase, median (range)	1502.0 (42.0-7550.0)	1298 (245.0-4764.0)	0.94
Creatine kinase-MB, median (range)	161.0 (15.0-929.0)	152.0 (19.5-500.0)	0.88
Troponin I, median (range)	45.01 (0.01-180.0)	5.46 (0.01-137.0)	0.039
C-reactive protein, mean (\pm SD)	8.49 (\pm 13.84)	25.68 (\pm 54.47)	0.17
Multiple coronary artery stenoses/culprit lesions, n (%)	4 (5.47)	11 (15.06)	0.54
DMT2	6 (8.21)	5 (6.84)	0.82
Final TIMI grade flow ≤ 2 , n (%)	7 (9.58)	5 (6.84)	0.15

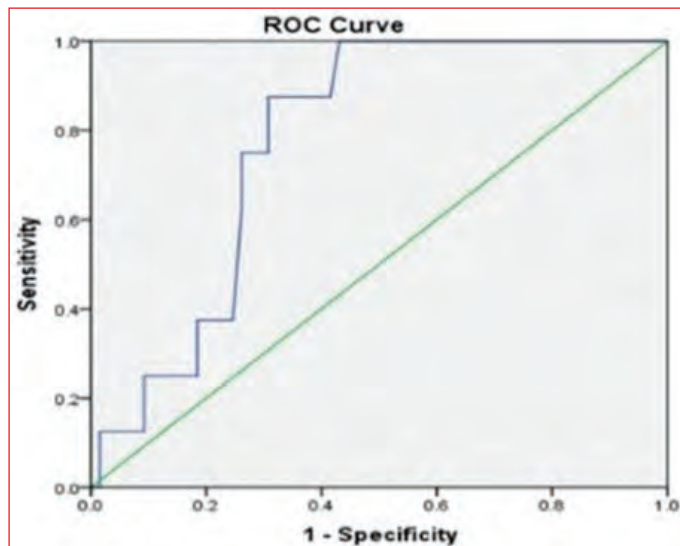


Figure 2. ROC curve analysis of adiponectin for the prediction of MACE (95% CI, 0.66-0.89), p=0.01.

Table 2. Area under the curve values for biomarkers

Biomarker	AUC (95% CI)	Cut-off value	p value
Adiponectin	0.77 (0.66-0.89)	1.80	0.01
HbA1c	0.74 (0.54-0.95)	6.35	0.02
Troponin I	0.60 (0.39-0.81)	31.75	0.32
Creatine kinase	0.60 (0.44-0.77)	1405	0.32
Creatine kinase-MB	0.51 (0.33-0.69)	169.5	0.90
Hemoglobin	0.60 (0.41-0.79)	140.5	0.32
C-reactive protein	0.78 (0.59-0.98)	7.0	0.008

Coronary Artery Disease / Acute Coronary Syndrome

PP-087

The prognostic effect of diabetes mellitus on elderly acute coronary syndrome patients in-hospital and long-term follow-up

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Background and Aim: Diabetes mellitus (DM) is a well-known major risk factor for cardiovascular, cerebrovascular and renovascular diseases as a result of macrovascular and microvascular complications, the prevalence of which is increasing all over the world. Moreover, it is associated with increased cardiovascular mortality and morbidity in patients with acute coronary syndrome (ACS) due to specific pathophysiological mechanisms affecting metabolic pathways. In recent years, as a result of increasing life expectancy all over the world, we have more often encountered elderly ACS patients who are more fragile and need more specific follow-up and treatment. In this study, we aimed to evaluate the intra-hospital and long-term prognostic effect of DM in elderly patients with ACS >75 years of age.

Methods: From December 2016 to January 2022 ACS patients >75 years of age who were admitted to the coronary intensive care unit and treated were included in the study. The baseline clinical, demographic characteristics and laboratory findings of the patients, as well as the treatment and follow-up processes applied were recorded. In addition, patients were followed up in terms of in-hospital and at least 1-year mortality.

Results: A total of 682 patients >75 years of age with ACS were included in the study. 34.8% of the patients (n=237) were diabetic patients. The average age of the patients was 82.18 ± 4.88 and 50.7% of them were female patients. The patients were divided into two groups with and without DM (non-DM vs. DM). The age and female gender were significantly higher in non-DM group compared to the DM group (p<0.001, p=0.003 respectively). Hypertension, history of PCI, blood sugar and triglycerides were found to be significantly higher in the DM group (p<0.001, p=0.038, p<0.001, p<0.001 respectively), while eGFR was found to be significantly higher in the non-DM group (p<0.001). There was no statistically significant difference between the two groups in terms of in-hospital and long-term mortality (p=0.605, p=0.746 respectively) (Tables 1 and 2). In the Univariate and multivariate Cox regression analysis, age, eGFR, hemoglobin, LVEF and acute renal failure were found to be an independent risk factor for mortality. There was no statistically significant relationship between DM and mortality in both univariate Cox regression analysis and multivariate Cox regression analysis (HR: 1.07, 95% CI 0.844-1.357, p=0.58; HR: 0.99, 95% CI 0.760-1.298, p=0.96) (Table 3). The Kaplan-Meier analysis also showed that there was no statistically significant difference between the all-cause mortality rate in elderly ACS patients with DM and without DM (log-rank test; p=0.57; Figure 1).

Conclusions: In our study, we found that DM is not a risk factor for mortality in ACS patients aged >75. Elderly

patients represent a special group and we think that risk modification, treatment and follow-up plans should be made accordingly.

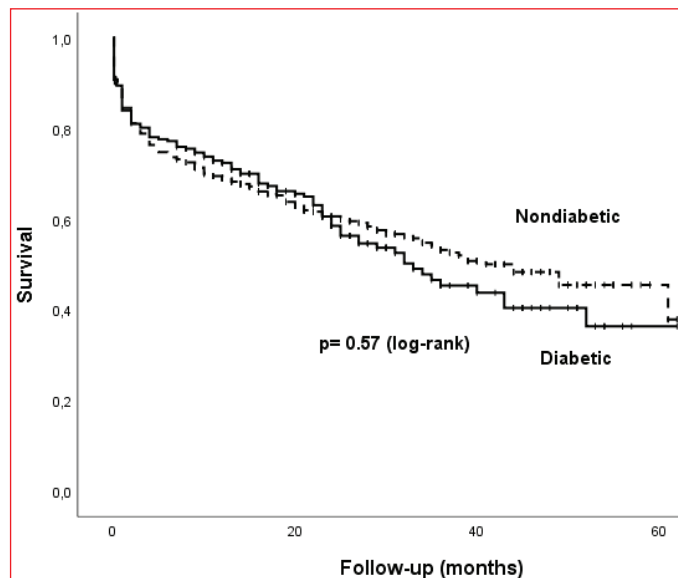


Figure 1. Kaplan-Meier survival curves (all-cause mortality) of acute coronary syndrome patients with or without diabetes mellitus.

Variables	Non-DM (n=445)	DM (n=237)	p value
Age (years)	82.00 (79.00, 86.00)	80.00 (77.00, 84.00)	<0.001
Gender (female), n (%)	233 (52.4)	103 (43.5)	0.03
Hypertension n (%)	309 (70.4)	222 (93.7)	<0.001
Atrial fibrillation, n (%)	123 (27.6)	64 (27.0)	0.928
History of PCI, n (%)	83 (18.8)	61 (26.0)	0.038
History of CABG, n (%)	58 (13.1)	40 (16.9)	0.207
Acute renal failure, n (%)	106 (24.4)	72 (30.6)	0.082
Revascularization (PCI), n (%)	281 (63.1)	165 (69.6)	0.092
In-hospital mortality, n (%)	46 (10.3)	28 (11.8)	0.605
Long-term mortality, n (%)	193 (43.4)	106 (44.7)	0.746

Variables	Non-DM (n=445)	DM (n=237)	p value
LVEF, %	45.00 (36.00, 55.00)	50.00 (40.00, 60.00)	0.089
eGFR (mL/min)	61.23 (44.85, 79.35)	56.00 (39.00, 72.87)	0.003
Glucose (mg/dL)	131.00 (111.00, 156.00)	192.00 (148.00, 279.00)	<0.001
CRP (mg/dL)	7.80 (2.90, 32.95)	9.65 (4.22, 40.15)	0.065
Hemoglobin (g/dL)	12.57 (5.07)	12.07 (1.98)	0.067
Maximum Troponin I (ng/mL)	98.51 (8.11, 4116.00)	236.50 (7.03, 6260.00)	0.613
Total Cholesterol (mg/dL)	168.00 (138.00, 205.50)	167.00 (142.50, 202.00)	0.960
HDL-C (mg/dL)	42.00 (35.00, 50.00)	39.00 (34.00, 48.50)	0.050
LDL-C (mg/dL)	103.00 (77.00, 137.00)	98.50 (77.00, 130.75)	0.251
Triglycerides (mg/dL)	97.00 (75.50, 126.50)	120.00 (92.00, 159.50)	<0.001

Table 3. Univariate and multivariate Cox regression analysis to detect the independent predictors of mortality in patients with acute coronary syndrome

	Univariate			Multivariate		
	HR	(95% CI)	p value	HR	(95% CI)	p value
Age	1.08	1.051-1.099	<0.001	1.06	1.040-1.095	<0.001
Gender	0.94	0.751-1.182	0.60	1.06	0.829-1.374	0.61
Diabetes Mellitus	1.07	0.844-1.357	0.58	0.99	0.760-1.298	0.96
Hypertension	1.03	0.786-1.369	0.79	1.07	0.780-1.464	0.67
eGFR	0.99	0.980-0.990	<0.001	0.99	0.985-0.997	0.02
Atrial fibrillation	1.14	0.889-1.474	0.29	1.10	0.847-1.448	0.45
Hemoglobin	0.88	0.832-0.936	<0.001	0.91	0.857-0.982	0.01
LVEF	0.96	0.950-0.971	<0.001	0.96	0.954-0.975	<0.001
Max Troponin	1.00	1.000-1.000	0.43	1.00	1.000-1.000	0.60
Revascularization	0.82	0.654-1.040	0.10	0.97	0.754-1.252	0.82
Acute Renal Failure	1.84	1.444-2.352	<0.001	1.34	1.025-1.770	0.03

Coronary Artery Disease / Acute Coronary Syndrome

PP-088

Features of clinical course and risk of developing comorbid conditions in coronary artery disease and chronic obstructive pulmonary disease

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Background and Aim: To assess the clinical course, risk of development, and prediction of comorbid conditions using the Charlson Index in patients with coronary artery disease (CAD), as well as to determine the relationship between chronic obstructive pulmonary disease (COPD).

Methods: Our study included 333 patients with comorbid conditions of CAD and COPD, of whom 183 (55.0%) were men and 150 (45.0%) were women. The study was conducted among patients who sought consultation at the outpatient clinic. The work was carried out in accordance with the Helsinki Declaration and was approved by the local ethics committee. Statistical analysis was performed using the Statistica 12.0 software package (Statsoft Inc., USA). Qualitative values were presented in absolute numbers (n) and percentages (%), and compared using Pearson's χ^2 test.

Results: Our study included 333 patients (mean age 63.53 ± 10.06) with CAD and COPD. The analysis showed that with increasing age (from 44 years to 60-74 years), the frequency of comorbid conditions proportionally increased (from 2.7% to 54.95%, respectively). In old age,

there is a tendency for comorbid conditions to decrease (to 13.2%). Among patients under 44 years of age, smokers accounted for $66.7 \pm 16.7\%$, which is 3.3 times higher than in the group of patients over 75 years old. This figure is 3.3 times higher than in the group of patients over 75 years old. With increasing age, the percentage of smokers among patients decreases ($\chi^2=23.501$; $p<0.01$). The relationships between the severity of CAD and COPD and the impact of COVID-19 were analyzed. According to the study, an analysis 180 (54%) patients had COVID-19. As can be seen, the highest prevalence of COVID-19 (66.6%) was observed in the younger age group under 44 years. With increasing age, there was a tendency for COVID-19 prevalence to decrease (43.2%) ($\chi^2=8.360$; $p<0.05$). A sedentary lifestyle was prevalent in all groups. The Charlson Comorbidity Index in the study group was 4.37 ± 0.99 points. In the first age group under 44 years, the average comorbidity index was 2.88 ± 0.60 points, corresponding to $77.2 \pm 3.6\%$ survival. In the age group of 75 years and older, the average comorbidity index was 5.70 ± 0.66 points, corresponding to $9.27 \pm 1.5\%$ survival. It was noted that the average values of C-reactive protein (CRP) increased with the older age group of patients. In the group of patients under 74 years, the average CRP levels did not differ between groups.

Conclusions: With increasing age, there is a tendency for the growth of comorbid conditions. The peak prevalence of CAD with COPD most commonly occurred in the 60-74 age group. As age increases, the percentage of smokers among patients decreases. It was found that the Charlson Comorbidity Index averaged 2.88 points in patients under 44 years, while in 75-year-olds, it was almost twice as high at 5.7 points. The average values of ESR and CRP in the blood increased with the older age group of patients.

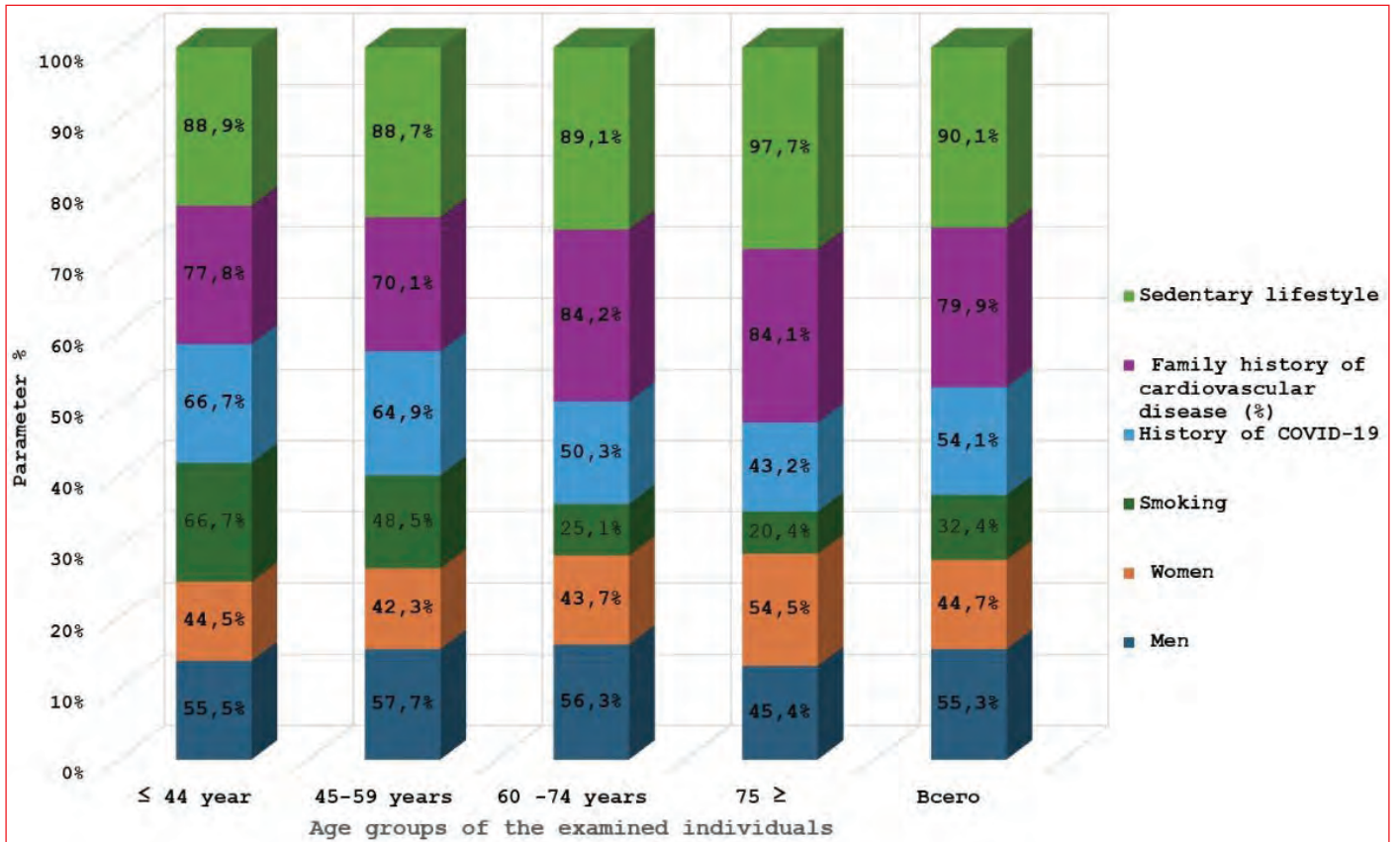


Figure 1. Gender structure and development risks in comorbid patients with ischemic heart disease and chronic obstructive pulmonary disease.

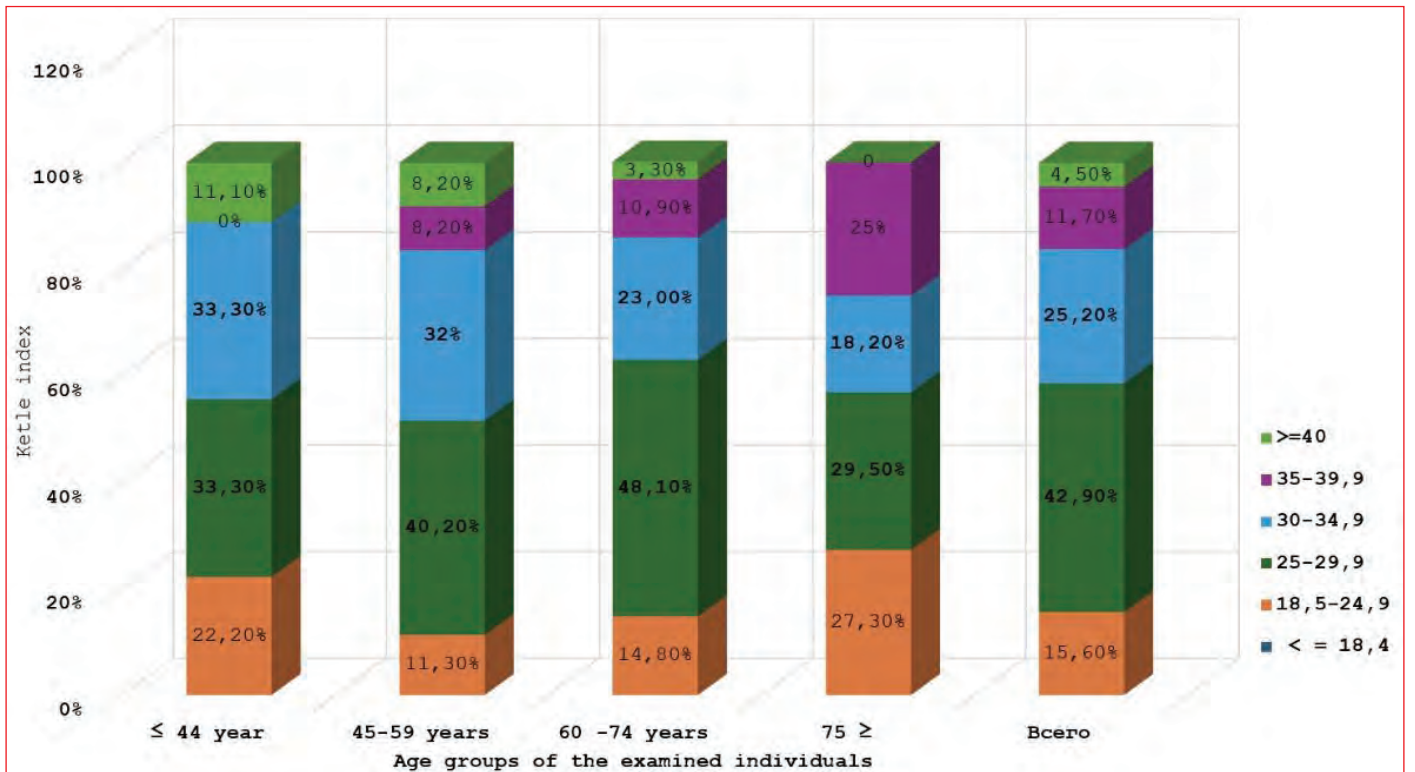


Figure 2. Prevalence of excess body weight and/or obesity among comorbid patients with CAD and COPD.

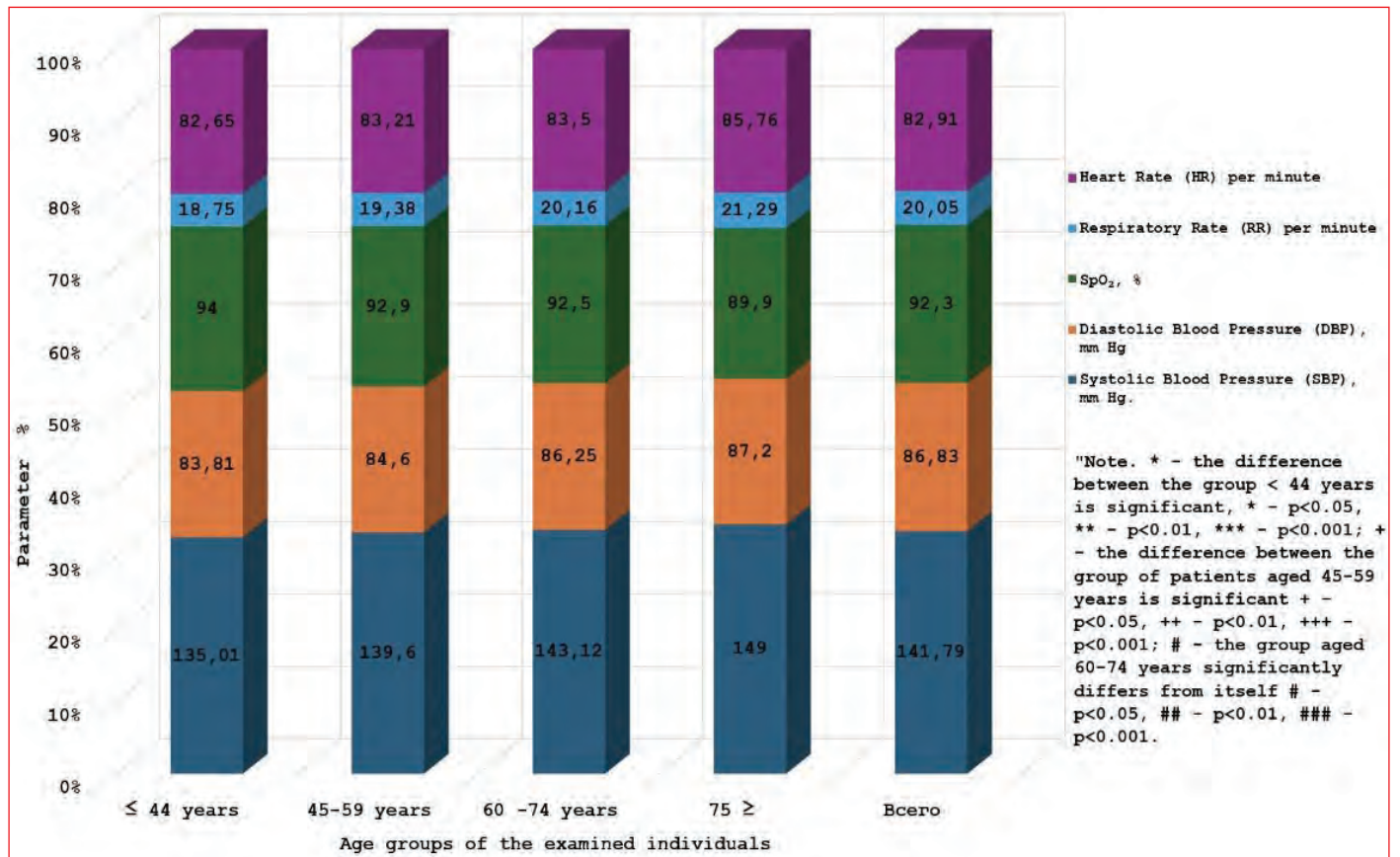


Figure 3. Clinical characteristics and prognosis based on the Charlson comorbidity index of examined patients with CAD and COPD.

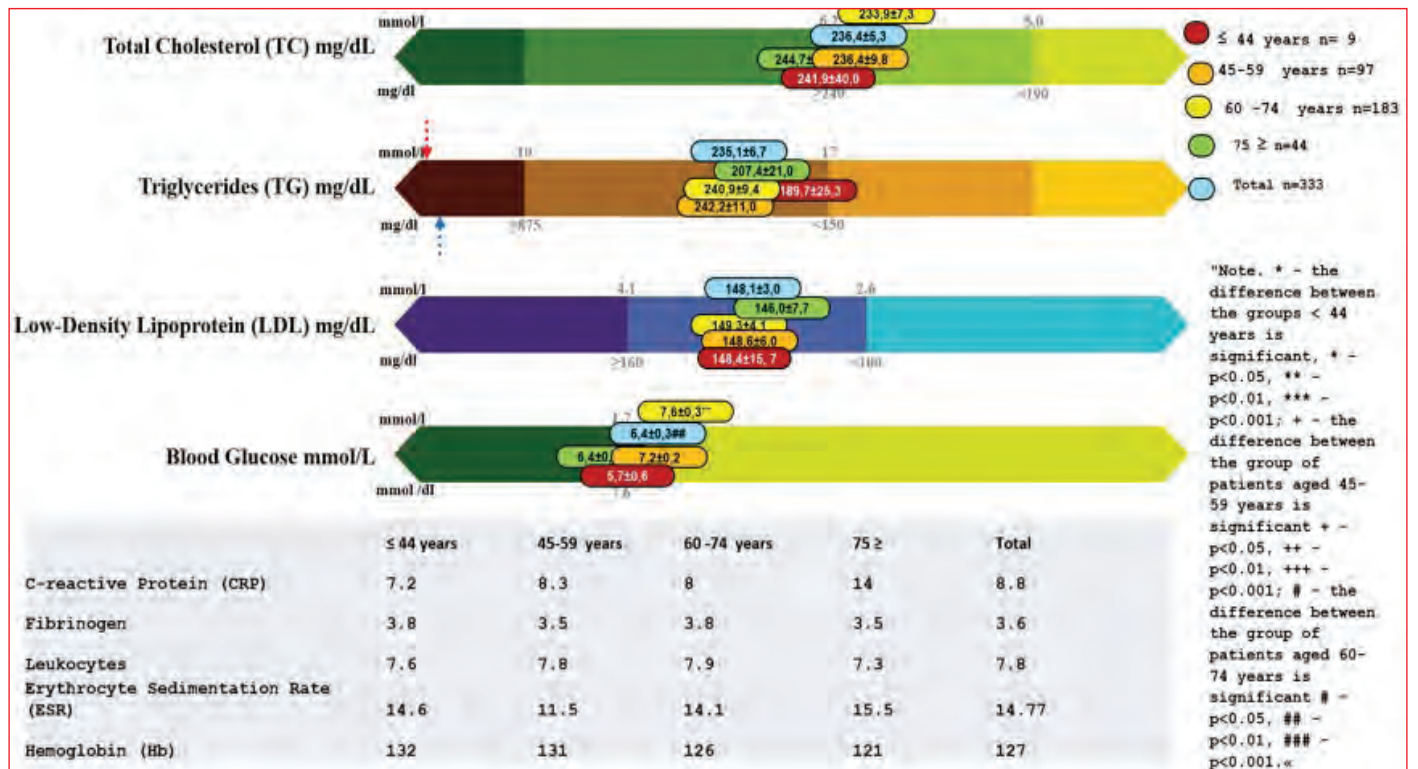


Figure 4. Main indicators of biochemical analysis in comorbid patients with CAD and COPD.

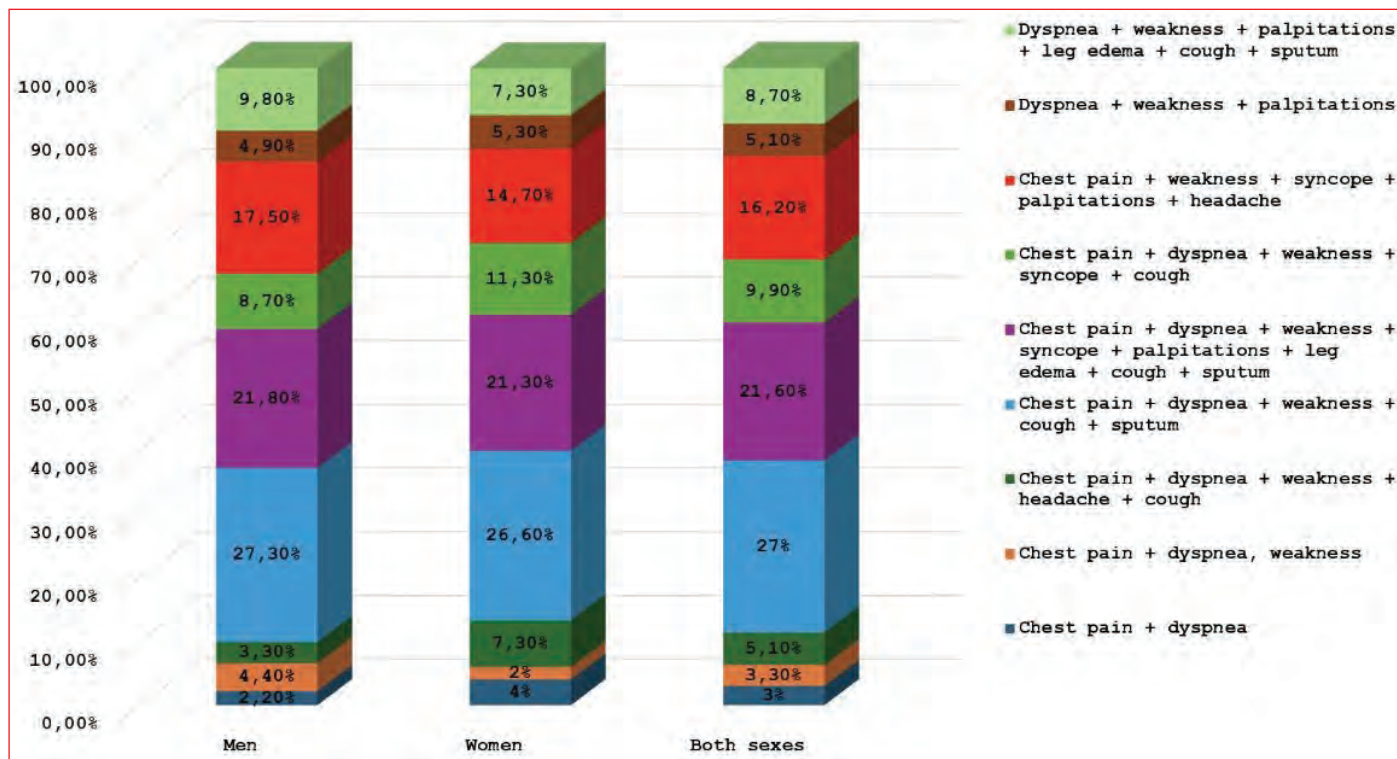


Figure 5. Features of gender manifestation of symptoms in comorbid patients with CAD and COPD.

Coronary Artery Disease / Acute Coronary Syndrome

PP-089

Triglyceride-glucose index as a biomarker in patients with acute coronary syndrome

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Background and Aim: The triglyceride-glucose (TyG) index is an indicator used to evaluate insulin resistance and has been shown to be a reliable biomarker for atherosclerosis and arterial stiffness. It is also associated with the severity of coronary artery disease. Additionally, the TyG index is a predictor of cardiovascular and all-cause mortality. Biomarkers like troponin are essential for the diagnosis of acute coronary syndromes because it is important to differentiate these patients using non-invasive techniques before applying coronary angiography. In this study, we aimed to define the relationship of another biomarker, the TyG index, with coronary artery disease and to determine if the TyG index can be used as a biomarker for acute coronary syndrome patients.

Methods: A total of 641 consecutive patients were enrolled in this study, including 473 with ST elevation myocardial infarction (STEMI), 81 with non-ST elevation myocardial infarction (NSTEMI), and 87 with normal coronary artery (NCA) patients. Demographic and laboratory data were retrospectively recorded from hospital medical records. Fasting blood glucose and triglyceride levels were obtained from the early admission to the hospital. The triglyceride index

was calculated as $\ln(\text{triglycerides} \times \text{fasting blood glucose} / 2)$. The distributional normality of the data was assessed. Depending on the normality of the data, either the T-test or the Mann-Whitney U test was employed to compare pairs of groups. Chi-square test was used to compare categorical variables.

Results: The triglyceride index was higher in the STEMI group compared to the NCA group [9.025 (8.611-0.635) vs. 8.889 (8.329-9.321); $p=0.005$]. Additionally, white blood cell count, hemoglobin levels, LDL-C levels, and fasting blood glucose were statistically higher, while HDL-C levels were lower in the STEMI group compared to the NCA group. When comparing the NSTEMI and NCA groups, the triglyceride index was higher in the NSTEMI group [9.123 (8.721-9.702) vs. 8.889 (8.329-9.321); $p=0.006$]. Moreover, WBC count, hemoglobin, glucose, and LDL levels were higher, and HDL levels were lower in the NSTEMI group. No statistically significant differences were found between the STEMI and NSTEMI groups in terms of laboratory data.

Conclusions: The findings suggest that the TyG index can be used as a noninvasive, accessible, and cost-effective biomarker for diagnosing acute coronary syndrome. The precise reason for the correlation between the TyG index and acute coronary syndrome remains unclear. However, it is hypothesized that the underlying mechanism may be related to insulin resistance. As is well known, insulin resistance is a significant determinant of cardiovascular disease risk factors, such as hypertension, hyperlipidemia, and hyperglycemia. Our study demonstrated that the TyG index is higher in patients with NSTEMI and STEMI compared to the normal population, and this finding may be beneficial for the differential diagnosis of acute coronary syndromes.

Table 1. Demographic and laboratory parameters of the study groups

	STEMI (n=473)	NSTEMI (n=81)	NCA (n=87)	p (STEMI vs. NSTEMI)	p (STEMI vs. NCA)	p (NSTEMI vs. NCA)
Age (year)	61.1 ± 11.9	59.9 ± 11.3	53.5 ± 2.6	0.406	<0.001	0.001
Gender	369 (78.8%)	57 (70.4%)	52 (60.0%)	0.132	<0.001	0.150
Hypertension	321 (67.9%)	64 (79.0%)	35 (40.2%)	0.044	<0.001	<0.001
Diabetes mellitus	224 (47.3%)	37 (45.7%)	17 (19.5%)	0.780	<0.001	<0.001
Atrial fibrillation	17 (3.6%)	2 (2.5%)	8 (9.2%)	0.607	0.020	0.066
White blood cell count (10 ³ /μL)	11.6 (9.7-14.3)	8.2 (11.0-13.9)	7.36 (6.1-8.7)	0.037	<0.001	<0.001
Hemoglobin (g/dL)	14.7 (13.3-15.8)	14.6 (13.4-15.9)	14.0 (12.9-15.2)	0.861	0.006	0.037
Platelet (10 ³ /μL)	245 (201-291)	237 (196-278)	232 (196-277)	0.221	0.101	0.816
Total cholesterol (mg/dL)	187 (161-215)	195 (166-217)	183 (158-219)	0.206	0.705	0.185
High density lipoprotein (mg/dL)	39 (34-46)	38 (33-46)	43 (37-51)	0.564	<0.001	0.002
Low density lipoprotein (mg/dL)	119 (96-143)	119 (97-226)	107 (90-127)	0.492	0.028	0.025
Triglyceride (mg/dL)	123 (80-187)	136 (97-226)	133 (88-205)	0.070	0.189	0.547
Fasting glucose (mg/dL)	137 (112-200)	131 (107-174)	100 (89-123)	0.198	<0.001	<0.001
Triglyceride-glucose index (TyG)	9.025 (8.611-0.635)	9.123 (8.721-9.702)	8.889 (8.329-9.321)	0.393	0.005	0.006

NCA: Normal coronary artery; STEMI: ST-elevation myocardial infarction; NSTEMI: Non-ST-elevation myocardial infarction

Coronary Artery Disease / Acute Coronary Syndrome

PP-090

Effect of percutaneous coronary intervention on sleep measures

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Background and Aim: This study aims to investigate the impact of interventional treatment for coronary artery disease on sleep by assessing sleep quality and duration before and after the procedure in patients undergoing elective coronary angiography and percutaneous coronary intervention (PCI).

Methods: The research was conducted in our cardiology clinic between May 1 and September 30, 2023. The sample comprised 56 patients who underwent coronary angiography. The "Pittsburgh Sleep Quality Index (PSQI)" was utilized to assess sleep quality. Data were collected through face-to-face interviews at the beginning and by phone interviews at the 3 month follow-up.

Results: The mean PSQI score of the study participants was 5.80 ± 3.72. Approximately half of the patients (26 patients, 47.3%) were identified to have poor sleep quality. For the 18 patients who underwent coronary angiography and were eligible for medical treatment follow-up, no significant differences were observed in any of the PSQI components during the 3 month follow-up. In the case of the 38 patients who underwent coronary angiography and received percutaneous coronary intervention, the analysis revealed a

significant improvement in Sleep Disturbance (PSQI component 5) (p<0.01), Daytime Dysfunction (PSQI component

Name: _____ Date: _____

Pittsburgh Sleep Quality Index (PSQI)

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. **Please answer all questions.**

- During the past month, what time have you usually gone to bed at night? _____
- During the past month, how long (in minutes) has it usually taken you to fall asleep each night? _____
- During the past month, what time have you usually gotten up in the morning? _____
- During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.) _____

5. During the <u>past month</u> , how often have you had trouble sleeping because you...	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe:				

- During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?
- During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

	No problem at all	Only a very slight problem	Somewhat of a problem	A very big problem
8. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?				

	Very good	Fairly good	Fairly bad	Very bad
9. During the past month, how would you rate your sleep quality overall?				

Figure 1. The Pittsburgh Sleep Quality Index (PSQI).

7) ($p=0.001$), and the total PSQI score ($p=0.001$) during the follow-up. The overall analysis of 56 patients showed a significant improvement in Sleep Latency (PSQI component 2) ($p=0.02$), Sleep Disturbance (PSQI component 5) ($p<0.01$), Daytime Dysfunction (PSQI component 7) ($p=0.005$), and the total PSQI score ($p=0.001$) over a 3 month period. Only 2 out of 6 patients (33%) without CAD improved after angiography, whereas this rate in CAD patients was 68% (34/50 patients) ($p=0.04$).

Conclusions: The results of this study indicate that sleep quality is low in individuals with CAD, and PCI has a positive impact on sleep.

Coronary Artery Disease / Acute Coronary Syndrome

PP-093

Relation between the triglyceride-HDL-C ratio and SYNTAX-II score in non-STEMI

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Background and Aim: Cardiovascular disease (CVD) is a leading cause of morbidity and mortality worldwide. Although several risk factors for CVD have been established, including age, male sex, hypertension, diabetes mellitus, obesity, hypercholesterolemia, and recent studies have showed that some patients without these risk factors may also develop CVD. Also, despite the development of advanced techniques and the primary and secondary prevention measures, patients with CVD remain at increased risk of recurrent adverse cardiovascular events. Therefore, identifying persons at early risk for CVD will have remarkable clinical significance for improving risk stratification and therapeutic management. Triglyceride and HDL-C, comprehensively reflects the balance between proatherogenic and antiatherogenic factors. However, evidence of the impact of the triglyceride-HDL-C ratio on the severity of coronary artery disease (CAD) is limited. This study investigated the association between the triglyceride-HDL-C ratio and SYNTAX-II score in non-STEMI patients.

Methods: This study included eighty seven (53 male-34 female, mean age 67.37 ± 12.67 years), with diagnosis of non-STEMI patients. Diagnostic coronary angiography was performed all patients. All sociodemographic characteristics, medical history, medical imaging data, and blood sample analysis results of the participants were collected from the medical records. SYNTAX-II score, as an indicator of the severity of coronary artery disease (CAD), was calculated. SYNTAX-II score was calculated using the online calculator (www.syntaxscore.com), and included two anatomical variables (anatomical SS and unprotected left main coronary artery disease) and six clinical variables [age, creatinine clearance (CrCl), left ventricular ejection fraction (LVEF), sex, chronic obstructive pulmonary disease (COPD), and peripheral arterial disease (PAD)]. All patients were divided into two groups according to SYNTAX-II score. Group A (SYNTAX-II score: 1-32, $n=66$), Group B (SYNTAX-II score: >32 , $n=21$).

Results: There is a significant difference between groups according to age, hypertension, diabetes mellitus, hyperlipidemia, COPD and biochemical parameters. Left ventricular ejection fraction was significantly lower in Group B patients than Group A patients. Also, Triglyceride/HDL-C ratio was significantly higher in Group B patients (Table 1).

Conclusions: In this study, we found that there is a relation between SYNTAX-II score and triglyceride/HDL-C ratio in non-STEMI patients. However, further studies are required.

Table 1. Clinical and laboratory parameters of patients

Variables	Group A (SYNTAX- Score II: 1-32) (n=66)	Group B (SYNTAX- Score II: >32) (n=21)	p value
Age (year)	62.98 ± 9.44	81.19 ± 11.71	<0.001
Gender (F/M)	25/41	9/12	0.798
Diabetes mellitus	26	15	0.033
Smoking	45	12	0.431
Hypertension	29	18	<0.001
Hyperlipidemia	22	17	<0.001
COPD	8	9	0.004
Leucocyte	8.69 ± 1.71	9.55 ± 2.42	0.136
Platelet	216.66 ± 47.76	319.16 ± 64.18	<0.001
Hematocrit	42.14 ± 4.72	38.51 ± 4.27	0.002
Glucose	154.09 ± 84.38	253.52 ± 173.41	0.001
Creatinine	0.77 ± 0.11	1.07 ± 0.12	<0.001
e-GFR	90.62 ± 7.08	57.46 ± 11.04	<0.001
Calcium	8.91 ± 0.43	8.40 ± 0.52	<0.001
Magnesium	2.0 ± 0.17	2.11 ± 0.08	0.015
Sodium	138.48 ± 1.85	136.04 ± 0.66	<0.001
Potassium	4.18 ± 0.38	4.40 ± 0.38	0.027
HbA1c	6.64 ± 2.22	9.20 ± 1.90	<0.001
Total cholesterol	159.47 ± 36.99	195.32 ± 51.94	0.005
LDL cholesterol	92.95 ± 21.71	141.06 ± 58.99	<0.001
HDL cholesterol	42.07 ± 10.05	32.68 ± 5.51	<0.001
Triglyceride	121.57 ± 40.52	126.55 ± 37.60	0.612
Triglyceride/HDL	3.12 ± 1.08	3.91 ± 1.51	0.013
LVEF (%)	54.51 ± 5.29	50.0 ± 9.21	0.006

Coronary Artery Disease / Acute Coronary Syndrome

PP-094

Association between serum albumin-to-creatinine ratio and long term outcomes in patients with ST-elevation myocardial infarction

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Background and Aim: The decreasing trend in ST-segment elevation myocardial infarction (STEMI) mortality is

mainly attributed to the greater use of reperfusion therapy, especially primary percutaneous coronary intervention (PCI). Actually, the prognosis of post-STEMI following PCI is influenced by clinical, echocardiographic, and biochemical factors. Therefore, early risk stratification is extremely essential for clinicians to determine treatment strategy and long-term management. Serum albumin is an important biomarker associated with inflammation and platelet activation, while serum creatinine is an indicator used to assess kidney function and overall systemic health conditions. The aim of this study is to evaluate the relationship between the serum albumin/creatinine ratio (sACR) and adverse cardiovascular events occurring during hospitalization in patients who have undergone PCI due to STEMI.

Methods: In this study, 851 patients diagnosed with STEMI and who underwent PCI at our two centers between January 2018 and January 2024 were retrospectively included. Adverse cardiovascular events were defined as reinfarction, malignant arrhythmia, cerebrovascular events, major bleeding, and in-hospital death after PCI. Laboratory records of the patients were examined through the health information system, and if death occurred after discharge, the dates were recorded. The serum sACR is defined by dividing serum albumin by serum creatinine. Patients were divided into two groups based on the median sACR value (35.4): group with high sACR (>35.4) and group with low sACR (≤35.4).

Results: A total of 851 STEMI patients [mean age 63.9 ± 10.7 years, 76% (n=647) male] who underwent percutaneous coronary intervention were included in the study. In the patient population, 23.3% had coronary artery disease, 47.7% were hypertensive, and 36.8% were diabetic. It was observed that 16.1% (n=137) of the patients experienced adverse cardiovascular events during hospitalization, and according to the data obtained from the follow-up system, 5.3% of the patients died within the first month, 6.2% within six months,

and 9.5% within one year. The one-year all-cause mortality rate was significantly different between the groups (12.3% vs. 6.8%, p=0.006). In the ROC curve analysis of sACR, the area under the curve (AUC) was calculated as 0.628 with 67.9% sensitivity and 56.4% specificity (p<0.001). The predictive value of sACR for the development of in-hospital adverse events in STEMI patients who underwent PCI was determined to be ≤34. Patients were followed for an average of 37 months. In the Kaplan-Meier curve analysis (Figure 2), all-cause mortality was higher in the group with a low sACR ratio (p<0.001).

Conclusions: The serum sACR ratio is an easy and practical value that can be calculated immediately after patient admission. It can be a valuable predictor for adverse cardiovascular events that may occur during hospitalization and for all-cause mortality in the long term, thereby providing benefits in daily clinical practice.

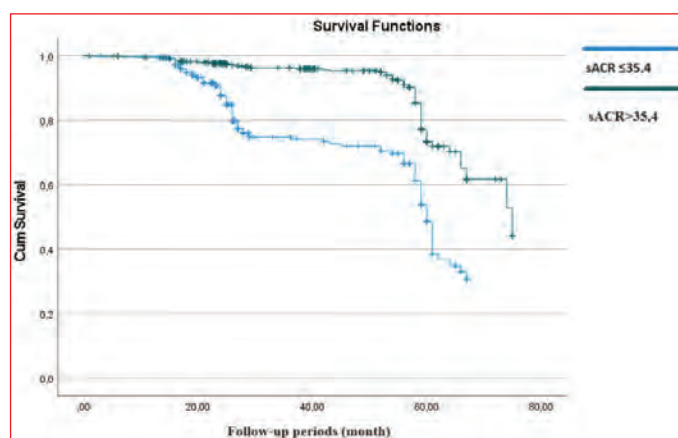
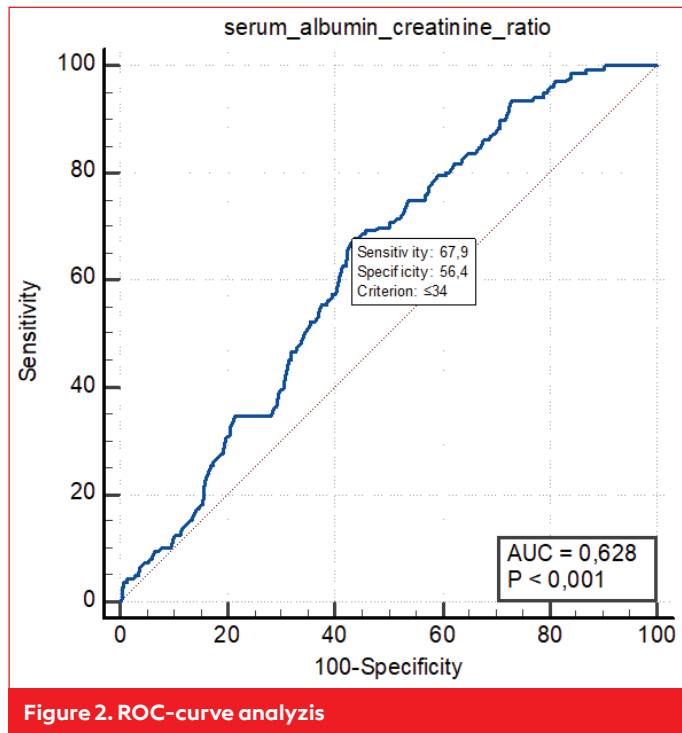


Figure 1. Kaplan-Meier curve showing all-cause mortality

Table 1. Relationships between clinical and laboratory data and the sACR in patients with STEMI

	sACR ≤35.4 (n=422)	sACR >35.4 (n=429)	p value
Age, years ± SD	64.9 ± 11.0	62.7 ± 10.4	0.003
Hypertension, n (%)	226 (53.6)	178 (41.9)	<0.001
Diabetes mellitus, n(%)	168 (39.8)	144 (33.9)	0.074
CAD, n (%)	103 (24.4)	93 (21.9)	0.384
Heart rate, beats/minute, ± SD	75 ± 17.1	74.7 ± 17.1	0.765
Albumin, g/L, ± SD	39.5 ± 4.5	40.4 ± 3.5	0.002
Creatinine, mg/dL, (IQR)	1.52 (1.37-1.67)	0.90 (0.81-0.97)	<0.001
Adverse cardiovascular events, n (%)	95 (22.5)	42 (9.9)	<0.001
Hospitalization duration, days	7.4 ± 3.9	7.1 ± 3.9	0.177
Reinfarction, n (%)	46 (10.9)	2 (0.5)	<0.001
Malign arrhythmia, n (%)	34 (65.4)	18 (34.6)	0.018
Development of cerebrovascular events, n (%)	5 (1.2)	0 (0)	0.030
Major bleeding, n (%)	4 (0.9)	8 (1.9)	0.257
In-hospital all-cause mortality, n (%)	17 (4)	18 (4.2)	0.902
One-month all-cause mortality, n (%)	25 (5.9)	20 (4.7)	0.411
Six-month all-cause mortality, n (%)	33 (7.8)	20 (4.7)	0.057
One-year all-cause mortality, n (%)	52 (12.3)	29 (6.8)	0.006

CAD: Coronary Arter Disease.

**Coronary Artery Disease / Acute Coronary Syndrome**

PP-095

Association of neutrophil to albumin ratio (NAR) with SYNTAX score in patients with acute coronary syndromeGülay Uzun¹, Ahmet Özdeyrya², Yahya Dağcan Bıçakçı¹, Faruk Kara¹, Ömer Faruk Çirakoğlu¹, Muhammet Raşit Sayın¹¹Ahi Evren Cardiovascular Surgery Training and Research Hospital, Trabzon²Cardiology Clinic, Trabzon Kanuni Training and Research Hospital, Trabzon

Background and Aim: Coronary artery disease (CAD) is a leading cause of morbidity and mortality worldwide. The SYNTAX score is an established tool used to assess the complexity of coronary artery lesions in patients undergoing revascularization procedures. Recent studies suggest that the neutrophil-to-albumin ratio (NAR) could serve as a novel marker of inflammation, potentially correlating with the severity of CAD. This study aimed to investigate the association between NAR and SYNTAX scores in patients with acute coronary syndrome (ACS) to evaluate NAR as a potential marker for assessing coronary artery disease severity.

Table 1. Baseline characteristics and laboratory findings of study population according to the SYNTAX SCORE

	All patients (n=715)	Low Syntax Score (<22) (n=633)	Moderate and high Syntax Score (>22) (n=82)	p
Age (years)	63	62.82	65.68	0.048
Male gender, n (%)	538 (75.2)	478 (75.5)	60 (73.2)	0.644
BMI (kg/m ²)	28.73 (25.80-32.87)	28.65 (25.73-32.4)	29.88 (27.1-34.2)	0.032
Hypertension, n (%)	406 (56.8)	341 (53.9)	65 (79.3)	<0.001
Diabetes mellitus, n (%)	188 (26.3)	156 (24.6)	32 (39)	0.005
Hyperlipidemia, n (%)	244 (34.1)	213 (33.6)	31 (37.8)	0.455
Smoking, n (%)	427 (59.7)	377 (59.6)	50 (61)	0.805
WBC	9.31 (7.6-11.5)	9.28	9.85	0.051
Hemoglobin (g/dL)	14 (12.8-15.1)	14.1 (12.9-15.1)	13.6 (12.3-15.1)	0.168
Lymphocyte (10 ³ /mL)	1.95	2	1.51	<0.001
Neutrophil (10 ³ /mL)	6.3	6.16	7.45	<0.001
PLT (x10 ⁹ /L)	219	219	212	0.086
MPV (fl)	8.62	8.60	8.80	0.129
Creatinine (mg/dL)	0.9	0.9	0.88	0.220
Glucose (mg/dL)	116	115.5	124.5	0.022
CRP	2.1	2.02	3.3	0.007
Albumin (g/dL)	4.12	4.15	3.91	<0.001
LDL-C (mg/dL)	139	139	137	0.249
HDL-C (mg/dL)	40	41	40	0.329
Total cholesterol (mg/dL)	199	200	197	0.392
Triglyceride (mg/dL)	121	122	112	0.425
Hs-Troponin I	8776	8156	11535	0.134
LV-EF (%)	55	58	45	<0.001
NLR	3.09	2.93	5.51	<0.001
SII	718.8	686.86	1053	<0.001
NAR	15.24	14.88	19.44	<0.001
SYNTAX Score	11	9	25.5	<0.001

Methods: A total of 715 patients diagnosed with ACS, including 300 with ST-elevation myocardial infarction (STEMI) and 415 with non-ST elevation myocardial infarction (NSTEMI), were retrospectively analyzed. Patients were stratified into two groups based on their SYNTAX scores: low (≤ 22) and moderate high (> 22). Laboratory parameters, including NAR, were measured, and their associations with SYNTAX scores were assessed using univariable and multivariable logistic regression analyses.

Results: Patients with moderate-high SYNTAX scores exhibited significantly higher NAR values compared to those

with low SYNTAX scores [19.44 (12.88-26.91) vs. 14.88 (11.03-20.20); $p < 0.001$]. Multivariable analysis identified NAR as an independent predictor of moderate-high SYNTAX scores (OR: 1.064; 95% CI 1.025-1.104; $p = 0.001$), alongside hypertension.

Conclusions: The findings of this study suggest that elevated NAR is associated with more severe coronary artery disease, as indicated by higher SYNTAX scores. NAR could serve as a valuable, cost-effective marker for risk stratification in ACS patients. Further research is warranted to validate these findings and explore their implications in clinical practice.

Table 2. Comparison of the baseline characteristics and laboratory findings of inframedian and supramedian values of neutrophil to albumin ratio

	Inframedian NAR (n=357)	Supramedian NAR (n=358)	p
Age (years)	63.5 \pm 11.9	62.8 \pm 12.7	0.048
Male gender, n (%)	261 (73.1)	277 (77.4)	0.186
BMI (kg/m ²)	28.7	28.62	0.907
Hypertension, n (%)	204 (57.1)	202 (56.4)	0.846
Diabetes mellitus, n (%)	87 (24.4)	101 (28.2)	0.243
Hyperlipidemia, n (%)	119 (33.3)	125 (34.9)	0.655
Smoking, n (%)	199 (55.7)	228 (63.7)	0.030
WBC (10 ³ /mL)	7.7	11.3	<0.001
Lymphocyte (10 ³ /mL)	2.14	1.68	<0.001
Neutrophil (10 ³ /mL)	4.7	8.32	<0.001
PLT ($\times 10^9$ /L)	217	223	0.112
MPV (fl)	8.5	8.73	0.129
Creatinine (mg/dL)	0.9	0.88	0.778
Glucose (mg/dL)	115	118	0.026
CRP	1.73	2.9	<0.001
Albumin (g/dL)	4.27	3.98	<0.001
LDL-C (mg/dL)	140	136	0.544
HDL-C (mg/dL)	40	41	0.595
Total cholesterol (mg/dL)	203	196	0.049
Triglyceride (mg/dL)	128	112	0.002
Hs-Troponin I	3782	24206	<0.001
LV-EF (%)	60	50	<0.001
NLR	2.1	5.4	<0.001
SII	448	1199	<0.001
CAR	0.40	0.75	<0.001
SYNTAX score	9	13	<0.001

Table 3. Univariable and multivariable analysis showing the association between parameters and moderate-high Syntax Score

	Univariable			Multivariable		
	OR	95% CI	p	OR	95% CI	p
Age	1.019	1.000-1.039	0.049			
BMI	1.047	1.000-1.096	0.051			
HT	3.274	1.877-5.711	<0.001	2.677	1.202-6.811	0.018
DM	1.957	1.212-3.160	0.006	2.037	1.067-3.886	0.031
Lymphocyte	0.574	0.426-0.773	<0.001			
Neutrophil	1.140	1.064-1.220	<0.001			
Glucose	1.002	0.999-1.005	0.153			
CRP	1.016	0.999-1.033	0.072			
Albumin	0.378	0.240-0.596	<0.001			
EF	0.927	0.904-0.951	<0.001	0.933	0.906-0.960	<0.001
NLR	1.101	1.045-1.160	<0.001			
SII	1.038	1.014-1.062	<0.002			
CAR	1.047	0.994-1.102	0.080			
NAR	1.061	1.035-1.088	<0.001	1.064	1.014-1.124	0.013

Multivariable model including: Age, hypertension, diabetes mellitus, lymphocyte, neutrophil lymphocyte ratio (NLR), systemic immun inflammation index (SII), neutrophil albumin ratio (NAR), ejection fraction (EF).

Coronary Artery Disease / Acute Coronary Syndrome

PP-096

Relationship between red cell distribution width (RDW)/lymphocyte ratio and SYNTAX score in patients with ST segment elevation myocardial infarction

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Background and Aim: Coronary artery disease is linked to increased inflammation, making research on inflammatory markers and related hematological parameters intriguing for clinical use. The main objective of this study is to investigate the relationship between the RDW/lymphocyte ratio in patients with STEMI and the anatomical severity of coronary artery lesions using the SYNTAX score. Additionally, other parameters associated with the SYNTAX score in these patients and the relationship of patient outcomes with the SYNTAX score have also been examined.

Methods: Patients who presented to the emergency department between January 1, 2022, and October 1, 2023, and underwent PCI due to STEMI were included in the study. After excluding additional conditions that could affect hematological parameters at admission, comorbidities, as well as hematological and biochemical results obtained at admission and within the first 24 hours after PCI, were recorded. Coronary angiography images of the patients were reviewed together with a cardiologist to calculate the SYNTAX scores.

Additionally, post-PCI left ventricular ejection fraction (LVEF), reperfusion status, and the recommendation for CABG were also noted.

Results: Data from 297 patients who were confirmed to have ACS after coronary angiography were included. The mean age of the patients was 62.1 ± 12.1 years. Reperfusion was achieved in 276 patients (92.9%), while 27 patients (9.0%) were recommended for CABG after angiography. The mean EF after coronary angiography was 43.7 ± 11.3 . 208 patients (70%) were classified into the low SYNTAX score group, and 89 (30%) were classified into the moderate-high SYNTAX score group. While no significant differences were found in demographic data, risk factors, and laboratory values between the groups, post-PCI LVEF ($p < 0.001$) and reperfusion rate ($p = 0.001$) were higher in the low SYNTAX group. The rate of patients recommended for CABG after PCI was higher in the moderate-high SYNTAX group ($p < 0.001$). There was no significant relationship found between laboratory values obtained at admission and SYNTAX Score. However, RLR ($p = 0.032$) and NLR ($p = 0.004$) parameters obtained within the first 24 hours after PCI were found to be correlated with SYNTAX score.

Conclusions: There is no association between RDW/lymphocyte ratio and neutrophil/lymphocyte ratio at admission with SYNTAX Score in STEMI patients. However, RDW/lymphocyte and neutrophil/lymphocyte ratios calculated from samples taken within the first 24 hours after PCI are higher in patients in the moderate-high SYNTAX group compared to those in the low SYNTAX group. Post-PCI LVEF is higher in patients in the low SYNTAX group. While the rate of reperfusion is higher in patients in the low SYNTAX group, patients in the moderate-high SYNTAX group are more often recommended for reperfusion with CABG.

Table 1. Logistic regression analysis**Table 12: Reperfüzyona etki edebilecek faktörlerin lojistik regresyon analizi**

Parametreler	ODD (B)	%95 CI		p
		Alt Sınır	Üst Sınır	
Yaş	0.995	0.949	1.043	0.841
Cinsiyet	7.307	0.912	58.557	0.061
Sigara	1.041	0.377	2.877	0.938
Diyabet	1.474	0.522	4.165	0.464
Hipertansiyon	2.289	0.709	7.389	0.166
KAH	2.296	0.844	6.242	0.103
RLR	1.006	0.976	1.037	0.707
SYNTAX Skoru	0.963	0.915	1,013	0.142

Table 2. Comparison of risk factors in groups with and without reperfusion**Table 11: Reperfüzyon sağlanma durumuna göre hastaların laboratuvar ve risk faktörlerinin karşılaştırılması**

Parametre	Reperfüzyon Sağlananlar	Reperfüzyon Sağlanamayanlar	p
Yaş	62.0 ± 12.1	64.3 ± 11.4	0.398
Cinsiyet (Erkek)	220 (%79.7)	19 (%95)	0.094
Risk Faktörleri (n, %)			
Sigara Kullanımı	96 (34.8)	7 (35)	0.984
Diyabet	82 (29.7)	9 (45)	0.152
Hipertansiyon	143 (51.8)	15 (75)	0.045
KAH* öyküsü	53 (19.2)	8 (40)	0.041
Laboratuvar Verileri 1 (Median, IQR 25-75)			
RDW**	13.9 (13.4-14.7)	14.4 (13.7-16.2)	0.169
Lenfosit	2.4 (1.6-3.6)	3.1 (1.3-4.5)	0.365
Nötrofil	7.9 (5.1-11.2)	7.6 (5.2-9.3)	0.181
Troponin	106 (26.6-1303.5)	451 (25.3-2947.5)	0.707
RLR [†]	5.8 (4-8.6)	5.5 (3.2-10.6)	0.408
NLR [‡]	3.2 (1.7-5.9)	2.6 (1.9-5.6)	0.303
SYNTAX skoru	16,25 (10-22,5)	23,75 (8,25-28,75)	0,209

*KAH: Koroner Arter Hastalığı
** RDW: Eritrosit dağılım genişliği
†RLR: Eritrosit dağılım genişliğinin lenfosit oranı
‡NLR: Nötrofil lenfosit oranı

Table 4. Multivariate linear regression analysis of parameters that may be associated with SYNTAX Scor**Table 10: SYNTAX Skoru ile ilişkili olabilecek parametrelerin çok değişkenli lineer regresyon analizi**

	Pearson Korelasyon Katsayısı	p	Regresyon katsayısı	%95 CI	p
Yaş	0.14	0.008	0.141	0.009 0.195	0.032
Diyabet	0.054	0.177	-2.015	2.601	0.803
Hipertansiyon	0.099	0.045	-1.259	3.247	0.386
Sigara Kullanımı	0.014	0.406	-1.028	3.355	0.297
KAH öyküsü	0.035	0.276	-2.187	2.875	0.789
RLR	0.016	0.392	-0.07	-8.254 3.977	0.492
NLR	0.022	0.355	-3.063	5.944	0.529

Table 3. Comparison of patient characteristics according to SYNTAX groups**Table 9: SYNTAX Gruplarına göre hastaların özelliklerinin karşılaştırılması**

	SYNTAX DÜŞÜK (N=208)	SYNTAX ORTA-YÜKSEK (N=89)	p
Yaş	61,4 ± 12,1	63,8 ± 12,0	0,113
Cinsiyet (Erkek)	173 (83,2)	67 (75,3)	0,114
Risk Faktörleri (n, %)			
Hipertansiyon	103 (49,5)	34 (61,8)	0,052
Diyabet	59 (28,4)	32 (36,0)	0,194
KAH* öyküsü	37 (17,8)	24 (27,0)	0,730
Sigara	75 (36,1)	29 (32,6)	0,565
Laboratuvar Verileri 1 (Median, IQR 25-75)			
RDW**	14 (13,5-14,8)	13,8 (13,4-14,7)	0,589
Lenfosit	2,4 (1,6-3,7)	2,4 (1,4-3,6)	0,909
Nötrofil	8,2 (5,6-11,5)	7,4 (5,3-10,9)	0,255
Troponin	132,5 (28,7-733,2)	116,5 (26,4-1748,0)	0,685
CK-MB	4,8 (2,7-22,1)	4,8 (2,9-21,8)	0,861
RLR [†]	6,0 (3,9-8,9)	5,8 (4,0-8,5)	0,876
NLR [‡]	3,4 (1,8-6,0)	2,9 (1,7-7,1)	0,433

*KAH: Koroner Arter Hastalığı
** RDW: Eritrosit dağılım genişliği
†RLR: Eritrosit dağılım genişliğinin lenfosit oranı
‡NLR: Nötrofil lenfosit oranı

	SYNTAX DÜŞÜK (N=208)	SYNTAX ORTA-YÜKSEK (N=89)	p
Laboratuvar Verileri 2 (Median, IQR 25-75)			
RDW**	14 (13,5-14,6)	13,9 (13,5-14,7)	0,848
Lenfosit	1,8 (1,3-2,3)	1,6 (1,1-2,2)	0,030
Nötrofil	8,9 (6,6-11,9)	10,2 (7,4-12,8)	0,560
Troponin	22609 (8517-53417)	37704 (15049-72972)	0,001
CK-MB	99,5 (10,2-99,5)	124,8 (38,9-288,0)	0,254
RLR [†]	8,3 (6,0-11,3)	8,5 (6,2-14,4)	0,032
NLR [‡]	4,9 (3,2-7,7)	6,5 (3,8-11,0)	0,004
RLR1/RLR2	1,35 (0,9-1,9)	1,5 (1,1-2,2)	0,040
Klinik Sonuçları			
PCI sonrası LVEF	45,8 ± 10,4	38,7 ± 11,9	<0,001
Reperfüzyon oranı	199 (95,7)	77 (87,5)	0,01
CABG Önerisi	7 (3,4)	20 (22,5)	<0,001

*KAH: Koroner Arter Hastalığı
** RDW: Eritrosit dağılım genişliği
†RLR: Eritrosit dağılım genişliğinin lenfosit oranı
‡NLR: Nötrofil lenfosit oranı

Table 5. Risk factors and laboratory data of all patients**Tablo 8: Risk faktörleri ve laboratuvar verileri**

Tüm hastalar (n = 297)	
Risk Faktörleri	n, (%)
Hipertansiyon	158 (53,2)
Diyabet	91 (30,6)
KAH* öyküsü	61 (20,5)
Sigara	104 (35,0)
Laboratuvar Verileri	Ort ± SS
RDW**	14,4±2,4
Lenfosit	2,7±1,6
RLR†	8,6 ± 22,5
Nötrofil	8,7±4,3
Hemoglobin	14,8 ± 8,3
Platelet	263,8 ± 78,5
Kreatinin	1,0 ± 0,4
Troponin	4656,4 ± 32302,9

*KAH: Koroner Arter Hastalığı
** RDW: Eritrosit dağılım genişliği
†RLR: Eritrosit dağılım genişliğinin lenfosit sayısına oranı

Lipid / Preventive Cardiology

PP-099

Implementation of supervised follow-up strategies into usual care in patients with ST-segment elevation myocardial infarction

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Background and Aim: Cardiac rehabilitation programmes have been associated with lower cardiovascular events, however attendance rates and compliance are under expectations. In this study, we aimed to investigate the impact of combining telerehabilitation with the usual care on risk factor control, drug adherence and maintaining a healthy lifestyle.

Methods: Patients who suffered ST-segment elevation myocardial infarction were included. In the combined follow-up strategy patients were allowed to contact a predetermined attending physician via mobile phone and a prescheduled outpatient clinic follow-up programme was arranged. In the conventional centre-based follow-up strategy patients were evaluated only at the outpatient clinic. Lifestyle modification, risk factor control, drug adherence and symptom control were compared at the end of 12 months.

Results: There were 243 patients in the combined follow-up group (group 1) and 299 patients in the centre-based follow-up group (group 2). Patients' demographics and comorbidities

were similar between groups. Lifestyle modifications including smoking cessation, attending regular physical exercise and adoption of a Mediterranean diet were significantly higher in group 1 patients ($p < 0.001$, $p = 0.024$ and $p = 0.003$ respectively). Adherence to dual antiplatelet therapy was higher in group 1; however, statin therapy was similar between groups ($p = 0.008$ and $p = 0.512$ respectively). Angina frequency and severity were lower in group 1. Likewise, functional capacity was also higher in group 1. There was no significant difference between groups concerning myocardial infarction, revascularization and cardiovascular death ($p = 0.450$, $p = 0.354$ and $p = 0.250$ respectively). Although the incidence of new-onset heart failure was similar ($p = 0.137$), hospitalization due to heart failure was lower in group 1 patients ($p = 0.007$).

Conclusions: Our study indicated that combined follow-up strategy with a predetermined attending physician via mobile phone following STEMI resulted in better cardiovascular risk factors, higher adoption of a healthy lifestyle and symptom control in comparison to conventional centre-based follow-up strategy in which patients were evaluated only at the outpatient clinic.

Table 1. Patient demographics and clinical features

	Combined follow-up group (n=243)	Centre-based follow-up group (n=299)	p value
Age (years)	75.6%	80.3%	0.197
Gender (male)	60.7 ± 11.3	62.9 ± 11.8	0.056
HT (%)	86.1	83.3	0.400
DM (%)	40.4	36.8	0.417
CVA (%)	3.7	2.8	0.374
CRF (%)	4.9	3.2	0.211
Dyslipidemia (%)	75.9	77.2	0.760
Anterior STEMI (%)	44.4	44.8	0.928
Smoking (%)	77.5	51.7	<0.001
LDL cholesterol (mg/dL)	129.6 ± 44.4	127.1 ± 41.9	0.621
Pro-BNP (ng/dL)	431 (136-1288)	566 (100-1517)	0.573
LVEF (%)	47.6 ± 10.7	43.1 ± 10.6	<0.001
HbA1c (%)	6 (5.6-6.7)	6 (5.7-6.8)	0.933

Table 2. Comparison of lifestyle modification

	Combined follow-up group (n=243)	Centre-based follow-up group (n=299)	p value
BP under control (%)	86.2	82.6	0.415
Smoking (%)	77.5	51.7	<0.001
Among smokers, attempt to quit (%)	49.2	18.2	<0.001
Among quitters, smoking relapse (%)	16	13.5	<0.001
Exercise (%)	60.1	46.3	0.024
Among exercisers, Appropriate exercise (%)	46.2	64.5	0.003
Reduce in BMI (%)	19.6	11.9	0.046
Diet (%)	64	56.2	0.184

Table 3. LDL-C, HbA1c, pro-BNP, LVEF values during follow-up

	Combined follow-up group (n=243)	Centre-based follow-up group (n=299)	p value
HbA1c (%)	6.1 (5.6–6.7)	6.2 (5.7–7)	0.248
LDL cholesterol (mg/dL)	78 ± 30.6	91.1 ± 35.8	<0.001
Reduction in LDL-C levels (%)	41 ± 17.4	35.9 ± 17.6	0.020
Pro-BNP (ng/dL)	191 (69–580)	543 (155–2420)	<0.001
LVEF (%)	51.9 ± 9	48.8 ± 10.4	0.003

Table 4. Comparison of symptom control

	Combined follow-up group (n=243)	Centre-based follow-up group (n=299)	p value
Angina (%)	24.3	33.3	0.025
CCS class			<0.001
1	85.5%	45.5%	
2	14.5%	32.2%	
3	0%	18.2%	
4	0%	4.1%	
Functional capacity			
NYHA			<0.001
1	73.8%	38.3%	
2	23.3%	32.5%	
3	2.3%	20.8%	
4	0.6%	8.3%	

Table 5. Cardiovascular outcomes

	Combined follow-up group (n=243)	Centre-based follow-up group (n=299)	p value
Myocardial infarction	7.8%	9.8%	0.450
Urgent/planned revascularization	22.7	24.4%	0.354
Timing of revascularization (months)	1 (1–12)	1 (0–3)	0.234
New-onset HF	17.7%	22.9%	0.137
Hospitalization due to HF	11.7%	20.3%	0.007
Cardiovascular mortality	4.2%	5.8%	0.250

Lipid / Preventive Cardiology

PP-100

Gender-specific disparities in familial hypercholesterolemia knowledge among medical students

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Background and Aim: Familial hypercholesterolemia (FH) is an inherited disorder characterized by elevated low-density lipoprotein cholesterol (LDL-C), leading to an increased risk of premature cardiovascular diseases. It is one of the most common genetic disorders, with a prevalence of approximately 1 in 250 individuals worldwide. Historically, gender disparities have influenced educational opportunities in the medical field, potentially affecting the understanding and management of conditions like FH. The extent of knowledge among primary care providers about the diagnosis and management of FH is not well-documented.

Methods: A 24-question survey assessing knowledge about FH was conducted among first and fifth-year medical students at Ege University Medical School, representing naïve and more knowledgeable groups, respectively. The Dutch Lipid Clinic Network (DLCN) questionnaire was appended for in-depth analysis. The survey was distributed to 210 of 418 first-year students and 174 of 353 fifth-year students, using digital QR codes and paper formats for those without internet access. Statistical analysis was performed using Python's libraries including Pandas, Seaborn, Matplotlib, and NumPy.

Results: Out of 263 respondents (140 first year), 131 were female. There were 1465 instances of 'do not know' responses, 679 (46%) from females, with fifth-year students contributing 405 of these, 184 (45%) from females. Females accounted for 450 of 910 incorrect responses overall, and among fifth-year students, 312 of 616 incorrect responses. Additionally, among the 131 female respondents, 60 (46%) had their lipid profiles tested, with 54 (90%) recalling their results; in contrast, 52 of 132 (39%) male respondents had tests, with 39 (75%) recalling their results.

Conclusions: The study identified significant gender disparities in both knowledge and awareness of FH, with female students demonstrating a lower rate of incorrect and uncertain responses compared to male students, especially in the later stages of their medical education. Additionally, female students were more likely to have their lipid profiles tested and to recall these results, indicating a higher engagement with their own health metrics. This proactive behavior in health monitoring among female students highlights a potential strength that educational interventions could build upon to further enhance understanding of FH.

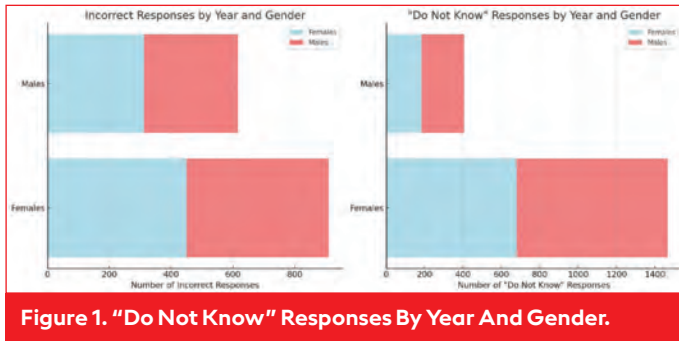


Figure 1. "Do Not Know" Responses By Year And Gender.

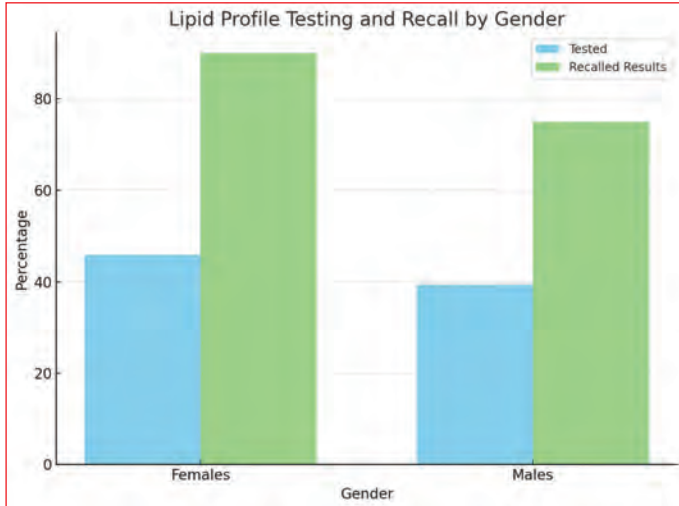


Figure 2. Lipid Profile Testing And Recall By Gender.

Lipid / Preventive Cardiology

PP-101

The effect of smartphone addiction on vessel wall thickness, which is a predictor of atherosclerosis

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Background and Aim: The increasing global popularity of smartphone usage has increased concerns about the negative effects of smartphone addiction, such as lack of sleep, sedentary life, bad eating habits, anxiety, stress, and depression, especially among the young population. These problems caused by smartphone addiction are also well-known risk factors for atherosclerosis. However, according to our observation, there is no research in the literature that directly shows the relationship between smartphone addiction and subclinical atherosclerosis. In this study, carotid intima-media thickness, an important surrogate marker in demonstrating subclinical atherosclerosis, was used to examine the relationship between smartphone addiction and subclinical atherosclerosis.

Methods: This cross-sectional study was conducted on 96 high school students aged between 13 and 22 years. A smart-

phone addiction questionnaire consisting of 33 questions was applied to measure smartphone addiction. Along with the socio-demographic characteristics of the patients, factors such as eating habits, sleep patterns, and activity levels were also questioned. The mean carotid intima-media thickness was measured by an experienced sonographer according to the published and accepted methods.

Results: When we set the threshold for smartphone addiction at over 66 points, we discovered that the group with smartphone addiction had considerably thicker carotid intima-media (0.68 ± 0.2 vs. 0.45 ± 0.1 ; $p < 0.001$). In addition, logistics regression analysis had shown that smartphone addiction level independently affects the carotid intima-media thickness (OR: 1.111; 95% CI 1.057-1.168, $p < 0.001$).

Conclusions: Smartphone addiction may help prediction of subclinical atherosclerosis via carotid intima-media thickness among teenagers.

Table 1. Results of logistic regression analysis for increased carotid intima-media thickness.

Variables	p	odds ratio	95% CI
Gender (female)	0.07	4.524	0.894-22.886
Body mass index (kg/m^2)	0.34	1.168	0.848-1.609
Fragmented family	0.04	0.112	0.013-0.966
Doing regular physical activity	0.17	1.663	0.288-9.592
Sleep time	0.02	0.341	0.138-0.845
Difficulty falling asleep	0.06	1.540	0.297-4.994
Mindful eating questionnaire score	0.04	1.048	1.001-1.110
Smartphone addiction scale score	<0.001	1.111	1.057-1.168

(CMT ≥ 0.6).

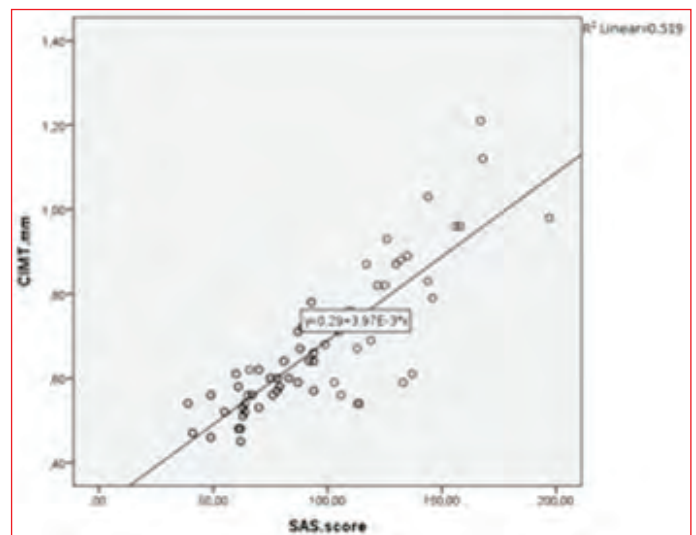


Figure 1. Correlations among carotid intima-media thickness (CIMT, mm) with smartphone addiction scale (SAS) score in all patients.

Lipid / Preventive Cardiology

PP-102

Does the current curriculum enhance knowledge on familial hypercholesterolemia? Insights from medical studentsMeral Kayıkçıoğlu¹, Bekir Kaan Çoşgun², Utku Sarıtaş², Muhammad Arya²¹Department of Cardiology, Ege University, Faculty of Medicine, İzmir
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Background and Aim: Familial hypercholesterolemia (FH) is a genetic metabolic disorder associated with high low-density lipoprotein cholesterol (LDL-C) levels from birth. Studies in Turkey have identified FH prevalence as high as one in every 22 individuals, partially explaining the early onset of heart attacks among the youth. The education of general practitioners and family doctors is crucial for the diagnosis and management of FH. This study assesses the knowledge of medical students on FH, comparing first-year students who represent the general low-knowledge population to fifth-year students who have completed their cardiology rotation.

Methods: A 24-item questionnaire along with the Dutch Lipid Clinic Network questionnaire was administered to the closest training groups of first and fifth-year medical students at Ege university in both digital and paper formats. Statistical analysis was performed using Python's Pandas, Seaborn, Matplotlib, and NumPy libraries. An awareness index was calculated from the questions about general or specific knowledge about FH. ANOVA was utilized to relate continuous variables like scores and response times with categorical variables such as class, gender, and blood lipid monitoring status.

Results: A total of 263 students responded to the survey (140 first year, 131 female). The mean awareness index is 21.5% (± 5). Additionally, 90.87% of the participants scored lower than 30% on the awareness index. Accuracy rates were 35.33% (± 14.47 SD) for first year, 47.15% (± 12.72 SD) for fifth year, 38.99% (± 15.75 SD) for males, and 42.83% (± 13.70 SD) for females. ANOVA results showed strong correlations between class level, score, response time, and incorrect answers ($p < 0.0001$); blood lipid monitoring with scores and incorrect answers ($p < 0.005$); and gender with scores and incorrect answers ($p = 0.036$). Fifth year students achieved higher scores despite more incorrect answers, with no significant time difference between male and female students ($p = 0.389$).

Conclusions: A substantial 90.87% of the participants scored below 30 on the FH Awareness Index, indicating a pervasive lack of knowledge regarding FH among the students. The rise in scores reflects educational efforts, but the persistently low scores and increased incorrect answers indicate a superficial learning depth, fostering a misleading sense of confidence among students. The strong correlation between regular blood lipid monitoring and higher scores suggests that educational interventions are promoting this healthy practice among students. However, the high error rate and reduced use of "I don't know" responses among fifth year students reveal a lack of deep understanding of fundamental concepts. In conclusion, FH education needs to be strengthened with more effective strategies to enable students to accurately identify and manage this critical health issue.

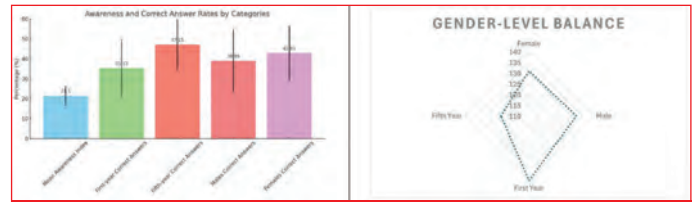


Figure 1. Results

Lipid / Preventive Cardiology

PP-104

Atherogenic plasma index is associated with subclinical atherosclerosis and diastolic functions in patients with newly diagnosed type 2 diabetes mellitusİşıl İşel¹, Turgut Karabağ²¹Department of Internal Medicine, İstanbul Education and Research Hospital, İstanbul²Department of Cardiology, İstanbul Education and Research Hospital, İstanbul

Background and Aim: Individuals with type 2 diabetes (T2DM) have a 1.5 to 2-fold higher risk of developing cardiovascular disease (CVD) compared to those without T2DM. The atherogenic index of plasma (AIP) is a predictor for the severity of coronary artery disease. In this study, we investigated whether AIP is associated with subclinical cardiac involvement in newly diagnosed T2DM patients without overt cardiovascular disease.

Methods: In the study, 108 patients (group 1; 59 men, 49 women, 49.8 ± 9.7 years) who applied to the internal medicine and cardiology outpatient clinics of our hospital with any complaint and were diagnosed with type 2 diabetes as a result of the tests performed, and 49 healthy individuals who were not found to have cardiovascular disease as a control group. (group 2; 15 men, 34 women, mean age 48.1 ± 7.5 years) were included. All tests were performed to exclude overt cardiovascular diseases in the diabetic group. Those who were found to have cardiovascular disease as a result of the tests, those with a positive exercise test, those with any disease and taking medication that could affect cardiac conduction, and those with systemic diseases were excluded from the study. All patients underwent transthoracic echocardiography (TTE). In addition to conventional findings on TTE, epicardial fat thickness from the anterior of the right ventricle in the parasternal long axis window and anterior aortic wall thickness 2-3 cm above the sinotubular junction were measured. Diastolic functions were calculated. Carotid intima media thickness was measured 2-3 cm above the carotid bifurcation with a vascular probe. AIP was calculated with the formula $\text{Log}(\text{TG}/\text{HDL-C})$.

Results: Comparison of demographic and laboratory parameters between groups are shown in Table 1. AIP was significantly higher in group 1 than in group 2. AIP was significantly correlated with CIMT, aortic anterior wall thickness and epicardial fat thickness (Table 2). It was also significantly correlated with parameters indicating diastolic functions (Table 2).

Conclusions: AIP is associated with subclinical atherosclerosis and diastolic functions in newly diagnosed T2DM patients without overt cardiovascular diseases. AIP can be considered as one of the target parameters in treatment in these individuals.

Table 1. Comparison of the parameters of the groups

	Group 1 (n=108)	Group 2 (n=49)	p
Age (years)	49.8 ± 9.7	48.1 ± 7.5	0.257
Gender (F, n)	49	34	0.019
Smoking (n)	43	19	0.393
BMI (kg/m ²)	29.4 ± 4.5	25.6 ± 3.9	<0.001
Systolic BP (mmHg)	138.6 ± 18.9	123.7 ± 13.8	<0.001
Diastolic BP (mmHg)	86.1 ± 12.1	77.9 ± 12.5	<0.001
Glucose (mg/dL)	191.3 ± 79.4	93.3 ± 8.1	<0.001
Urea (mg/dL)	27.4 ± 7.6	26.2 ± 7.4	0.355
Creatinine (mg/dL)	0.73 ± 0.17	0.70 ± 0.12	0.308
Total cholesterol (mg/dL)	206.1 ± 49.3	194.5 ± 33.5	0.091
Triglyceride (mg/dL)	193.3 ± 123.7	103.8 ± 52.4	<0.001
LDL cholesterol (mg/dL)	126.6 ± 42.3	116.5 ± 29.1	0.143
HDL cholesterol (mg/dL)	44.7 ± 13.4	57.6 ± 14.0	<0.001
HOMA IR	6.14 ± 3.88	1.88 ± 1.22	<0.001
Insulin (IU)	14.2 ± 9.2	7.2 ± 3.6	0.002
HbA1c (%)	8.9 ± 2.4	5.4 ± 2.3	<0.001
AIP	1.35 ± 0.71	0.61 ± 0.50	<0.001
CIMT (mm)	1.04 ± 0.24	0.68 ± 0.12	<0.001
Epicardial fat thickness (mm)	0.72 ± 0.19	0.42 ± 0.08	<0.001
Aortic anterior wall thickness (mm)	0.58 ± 0.13	0.41 ± 0.09	<0.001
E/A	0.85 ± 0.28	1.37 ± 0.20	<0.001
E/E' lateral	7.44 ± 2.30	6.32 ± 1.76	<0.001
E/E' septal	10.23 ± 2.50	8.06 ± 1.83	<0.001

BMI: Body mass index; BP: Blood pressure; LDL: Low density lipoprotein; HDL: Density lipoprotein; AIP: Atherogenic plasma index; CIMT: Carotis intima media thickness).

Table 2. Spearman correlation analysis between AIP and several parameters.

	rho	p
CIMT (mm)	0.27	0.001
Epicardial fat thickness (mm)	0.41	<0.001
Aortic anterior wall thickness (mm)	0.33	<0.001
EF	-0.33	<0.001
Mitral E/A	-0.27	<0.001
E/E' (lateral)	0.11	0.161
E/E' (septal)	0.17	0.044
Age (years)	-0.07	0.387
HOMA-IR	0.54	<0.001

CIMT: Carotis intima media thickness; EF: Ejection fraction; HOMA-IR: Homeostatic model assessment for insulin resistance).

Lipid / Preventive Cardiology

PP-105

The effects of lipoprotein (a) and high sensitive CRP on coronary artery disease in patients with severe hypercholesterolemia

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Background and Aim: Lipoprotein(a) [Lp(a)] is a genetically inherited lipoprotein that has emerged as a significant causal risk factor for various forms of cardiovascular disease, notably including coronary artery disease (CAD), peripheral artery disease (PAD), ischemic stroke, and calcific aortic valve stenosis. Epidemiological evidence suggests that elevated levels of Lp(a) can lead to increased atherogenesis, persistent inflammation, and enhanced thrombosis, thereby contributing to the pathophysiology of these cardiovascular conditions. Parallel to Lp(a), high-sensitivity C-reactive protein (hsCRP) has also been identified as a vital biomarker and risk factor for coronary artery disease. Intriguingly, the interaction between these two biomarkers is thought to operate independently of low-density lipoprotein (LDL) cholesterol levels. However, this relationship becomes more complex in individuals with significantly elevated LDL levels, specifically those exceeding 190 mg/dL. While the individual contributions of Lp(a) and hsCRP to the risk of coronary artery disease have been documented, their interplay and unique impact in patients presenting with elevated LDL cholesterol which is also associated with increased risk for coronary artery disease remain inadequately explored. Therefore, this study aims to elucidate the effects of Lp(a) and hsCRP levels on coronary artery disease in patients with severe hypercholesterolemia defined as LDL cholesterol levels greater than 190 mg/dL.

Methods: This single-center, retrospective study included patients with LDL values >190 mg/dL, Lp(a) and hsCRP tests, and imaging of their coronary arteries.

Results: This study encompass an analysis of 62 patients. Among these participants, five individuals reported a clinical history of acute coronary syndrome, while twelve exhibited greater than 50% stenosis in their coronary arteries. The Agatston calcium score was determined for 40 of the patients. Findings revealed that four of these patients displayed scores suggestive of severe atherosclerosis, whereas seven presented with moderate atherosclerosis (Table 1). Furthermore, a moderate and statistically significant correlation was observed between patient age and the levels of LDL, Lp(a), and hsCRP. However, no significant associations were identified between the values of Lp(a) and hsCRP with CAD, acute coronary syndrome and Agatston calcium scores. Contrarily Lp(a) have significant association with the extent of coronary artery disease defined by greater than 50% stenosis (Table 2).

Conclusions: In patients with severe hypercholesterolemia, defined as serum LDL cholesterol levels greater than 190 mg/dL, Lp (a) have significant association but hsCRP have no association with the extent of coronary artery disease.

Table 1.

Table 1. Baseline Characteristics of the Patients	
Characteristics (N=62)	
Age	
Median (IQR) - yr	54 (44-60)
Sex - no. (%)	
Female	34 (54.8)
Male	28 (45.2)
Coexisting conditions - no. (%)	
Smoking	13 (21)
Diabetes	7 (11.3)
Coronary heart disease	35 (56.5)
Hypertensiyon	17 (27.4)
LDL (mg/dl) - mean (\pm SD)	222.4 (\pm 37.4)
Lipoprotein a (mg/dl) - (IQR)	20.2 (11.6-39.6)
High sensitive CRP (mg/L) - (IQR)	1.2 (0.8-2.7)
Agatston calcium score (N=40) - (IQR)	0 (0-108)
No atherosclerosis (AU:0) - no. (%)	22 (35.5)
Mild atherosclerosis (AU:1-99) - no. (%)	9 (14.5)
Moderate atherosclerosis (AU:100-399) - no. (%)	7 (11.3)
Severe atherosclerosis (AU: \geq 400) - no. (%)	4 (6.5)
>%50 coronary stenosis - no. (%)	12 (19.4)
Acute coronary syndrome - no. (%)	5 (8.1)

Table 2.

Table 2. Analysis of the Association of Lp(a) and hsCRP with Comorbidities		
	Lp(a)	hsCRP
Coronary heart disease	p:0.265	p:0.188
Acute coronary syndrome	p:0.148	p:0.736
Agatston calcium score	p:0.088	p:0.155
>%50 coronary artery stenosis	p:0.036	p:0.651

Lipid / Preventive Cardiology

PP-107

Effect of increasing triglyceride-glucose index on blood pressure and heart rate recovery values examined in exercise stress tests

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Background and Aim: The triglyceride-glucose index (TgG) was first used as an important biomarker of insulin resistance. In [fasting triglyceride value x fasting glucose value/2] is the formula used to calculate TgG. The exercise stress test shows changes in heart rhythm and blood pressure at different stages. Heart rate recovery (HRR) is how quickly your heart returns to normal after you stop exercising. It is the difference between your heart rate at its maximum in the treadmill and heart rate at one or three minutes later while at rest. The aim of the study was to observe how an increase in TgG changes HRR and blood pressure values.

Methods: The research was a prospective study including 98 patients. The examined parameters in the exercise stress test and calculated TgG data were obtained from hospital data base. The maximum blood pressure values in treadmill and heart rate recovery values at 1 and 3 minutes were recorded. Examined parameters of 98 patients were compared with calculated TgG values statistically.

Results: Correlation coefficient of TgG with HRR at 1 min, at 3 min, peak systolic pressure, and peak diastolic pressure is (-0.32/-0.46/0.03/0.07) respectively. The mean values

Table 1. Correlation coefficient of TgG with heart rate recovery and peak exercise blood pressure

	TgG	HRR1 Min	HRR3 Min	Peak Exe HR	Peak Exe SBP	Peak Exe DBP
TgG	Correlation Coefficient	1.000	-0.322	-0.464	-0.255	0.027
	P		0.001	0.001	0.001	0.791
	N	98	98	98	98	98

Table 2. TgG values, HRR at first and third minutes of exercise stress test of CAD and non-CAD patients

CAD		TgG	HRR1 Min	HRR3 Min	Peak Exe SBP	Peak Exe DBP	Peak Exe HR	
0	Mean	8.8940	38.5357	61.321	194.2222	82.3333	164.3571	
	Standard deviation	0.49938	12.51176	11.35879	30.24470	13.61048	13.82939	
	Percentiles	25	8.4965	31.0000	54.2500	168.7500	73.7500	153.5000
		50	8.8836	39.5000	61.0000	198.0000	81.5000	165.0000
		75	9.2557	48.7500	68.0000	215.7500	92.0000	176.0000
1	Mean	9.1758	34.2857	55.9643	206.5000	85.8214	154.3571	
	Standard deviation	0.60616	11.36224	9.10223	25.01333	19.09635	14.61408	
	Percentiles	25	8.7315	27.0000	50.0000	188.7500	75.2500	142.5000
		50	9.0607	33.0000	57.0000	207.0000	85.5000	155.0000
		75	9.5286	41.7500	61.7500	226.2500	98.7500	163.5000

of HRR in 3 min were 55.9 and 61.3 in coronary artery disease (CAD) patients and non-CAD patients ($p=0.046$). In patients with CAD first (Q1), second (Q2), third (Q3) quartiles are (50/57/61.7) at HRR in 3 min, whereas the distinctly non-CAD patient quartiles are (54.2/61/68). The mean TgG value of CAD patients is 9.17 and 8.89 in non-CAD patients ($p=0.05$).

Conclusions: TgG and HRR are slightly inversely proportional to 1 min and moderately inversely proportional to 3 min. There was no significant correlation between TgG levels and blood pressure. CAD patients have greater mean TgG values, and their HRR values tend to be longer compared with non-CAD patients.

Lipid / Preventive Cardiology

PP-108

A comparison of an online and in-person innovative method for primary prevention of coronary artery disease

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Background and Aim: Implementing and maintaining primary preventive methods to enhance health-related behavioral outcomes for coronary artery disease (CAD) can be challenging, especially in unexpected circumstances such as the COVID-19 outbreak. The current study compared the feasibility and effectiveness of a longitudinal online-enhanced education and follow-up program to a face-to-face structured program in terms of behavioral outcomes for CAD prevention.

Methods: CAD Prevention Project (SCAD-PP) was designed to utilize medical school students to conduct the project under the supervision of professors. It had two different education and training phases. A series of conferences regarding the primary prevention for CAD were organized for underserved populations. Participants were prospectively assigned to an intervention where pre- and post-conference knowledge was collected, and an education booklet was given. Each participant was followed-up via phone calls for 6 months. At the 6 months follow-up, the impact of the program on behavioral outcomes was assessed. Because face-to-face meetings were forbidden during the COVID-19 outbreak, we restructured the study using Microsoft Teams program. CAD Online Prevention Project (SCAD-OPP) started in April 2020. A series of online conferences on primary prevention for CAD were organized. Each student was asked to enroll participants from local population and assist them during the online intervention. Pre and post conference knowledge were collected and assessed via online tools and an education booklet was mailed to the participants. Each

participant was followed-up via phone calls for 6 months. At the 6-month follow-up, data was collected to evaluate and compare the impact of the SCAD-OPP and the SCAD-PP on behavioral outcomes.

Results: There were 172 participants in the SCAD-PP and 72 in the SCAD-OPP group. SCAD-OPP were older (45 ± 13 vs. 40 ± 11.9 years, $p=0.005$) and had a higher education level. The rest of the demographic characteristics were comparable. Overall knowledge on CAD risk factors, primary prevention measures, diet and daily exercise habits were very poor in both groups. After the enhanced education and follow-up program there was a significant improvement on the knowledge of CAD risk factors and primary prevention measures in both groups. At 6-month follow-up, both groups experienced similar favorable changes in behavioral outcomes. However, the decrease in body mass index was greater in the SCAD-PP ($p=0.019$).

Conclusions: This is the first study to show that a longitudinally structured training program of medical students may be used to administer an online or face-to-face enhanced education and follow-up program for primary prevention of CAD with similar successful outcomes. The use of this method, whether face-to-face or online, benefits not just the public interest but also enhances active interaction of medical students with patients at a very early stage of their career.

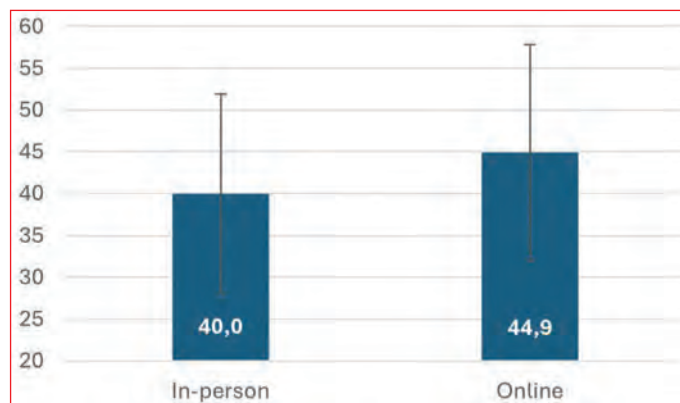


Figure 1. Baseline Mean Age (yrs)

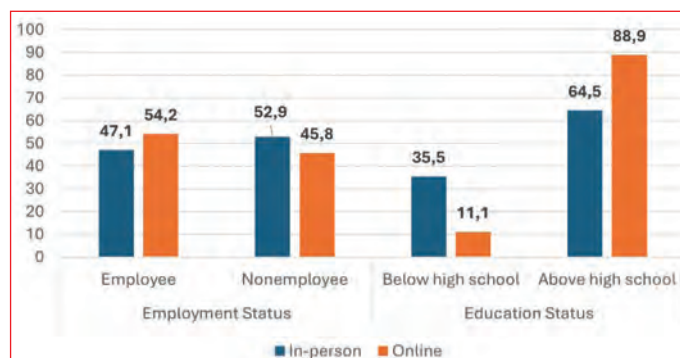


Figure 2. Baseline Employment and Education Status.

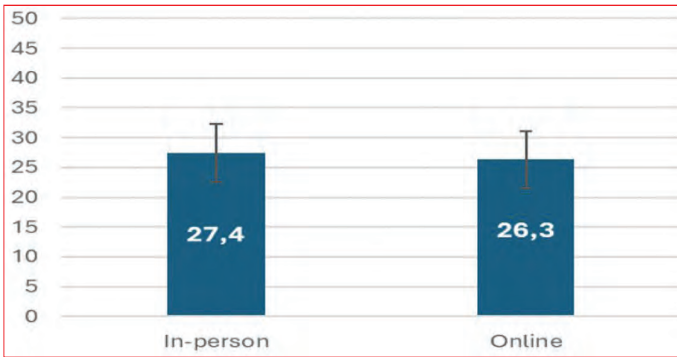


Figure 3. BMI at 6 months.

Nuclear Cardiology

PP-109

Machine learning for prognostic prediction in coronary artery disease with SPECT data: A systematic review

Vedat Çiçek¹, Ezgi Hasret Kozan Çıkrıkçı², Mert Babaoğlu³, Almina Erdem³, Mohammed Laser Mohammed⁴, Tufan Çınar⁴, Hatice Savaş¹, Ulaş Bağcı¹

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Background and Aim: Single-photon emission computed tomography (SPECT) analysis relies on qualitative visual assessment or semi-quantitative measures like total perfusion deficit (TPD) that plays a critical role in the non-invasive diagnosis of CAD by assessing regional blood flow abnormalities (Figure 1). Recently, machine learning (ML)-

based analysis of SPECT images for CAD diagnosis holds promise, its utility in predicting long-term patient outcomes (prognosis) remains an active area of investigation. In this review, we comprehensively examine the current landscape of ML-based analysis of SPECT for CAD prognosis.

Methods: To comprehensively identify relevant studies, we conducted a systematic search across PubMed, Embase, and Cochrane databases up to November 30, 2023. Articles retrieved from the initial search were then screened based on titles and abstracts. Subsequently, full-text review of the shortlisted articles was performed to ensure they met the following inclusion criteria: 1) investigation of SPECT-MPI-based AI models for prognostic prediction in CAD patients, and 2) presentation of original research findings.

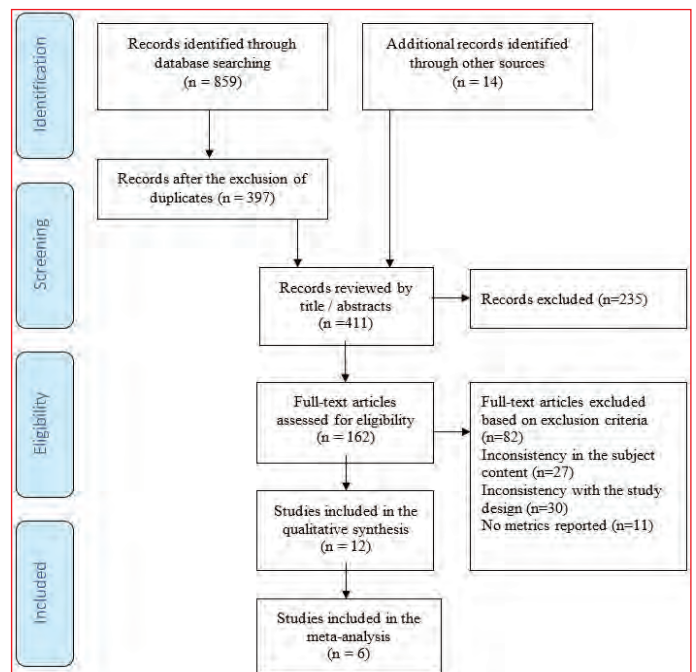


Figure 2. PRISMA flowchart for the study screening and selection.

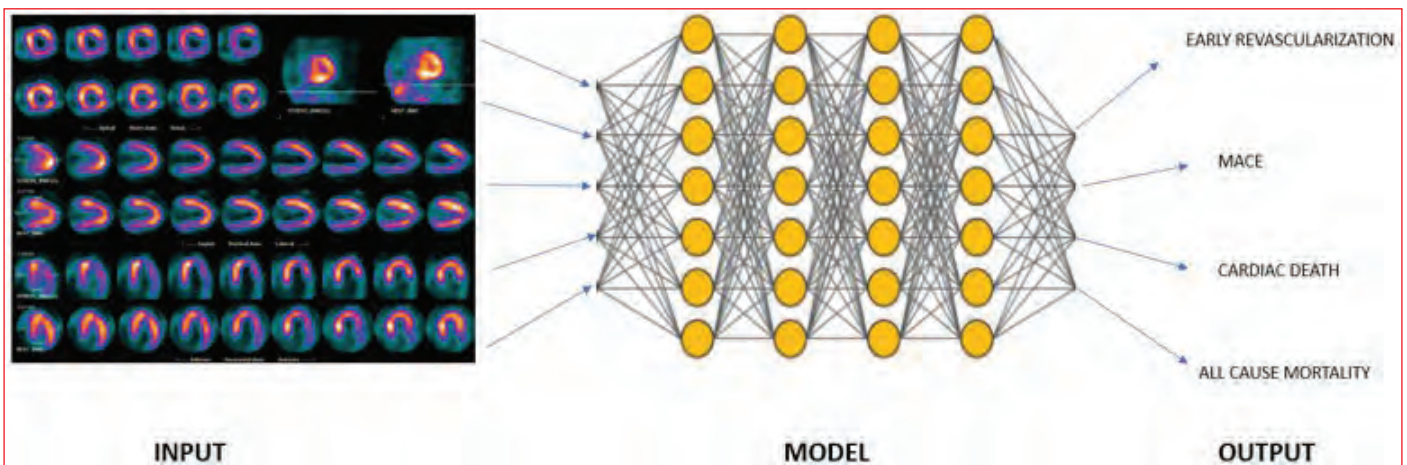


Figure 1. Deep learning techniques allow obtaining different outcomes from imaging images.

FIRST AUTHOR	YEAR	OUTCOME AGE GENDER (MALE) CENTER	PARTICIPANTS OUTCOME	ML MODEL USED	FOLLOW UP TIME	STRESS TPD Vs ML-SPECT MODEL (AUC)	STRESS TPD Vs ML-SPECT+EHR MODEL (AUC)	LR MODEL Vs ML-SPECT MODEL (AUC)	LR MODEL Vs ML-SPECT+EHR MODEL (AUC)
Julian Betancur et al. [18]	2018	MACE 62 ±13 YEARS 48% SINGLE CENTER	2619/239	WEKA	3 YEARS	0.73 Vs 0.78 P <0.05	0.73 Vs 0.81 P <0.05	-	-
David Haro Alonso et al. [19]	2019	CARDIAC DEATH 71 ±12 YEARS 52% SINGLE CENTER	832/1551	AdaBoost	3.13±1.99 YEARS	-	-	0.76 Vs 0.83 P<0.001	-
Lien-Hsin Hu et al. [20]	2020	EARLY REVASCULARIZATION 65 ±11 YEARS 66% MULTICENTER	1970/958	WEKA	6 MONTHS	-	0.71 Vs 0.79 P < 0.001	-	-
Luis Eduardo Juarez-Orozca et al. [21]	2020	MACE 68 ±9 YEARS SINGLE CENTER	1085/159	CNN	385 days	0.84 Vs 0.90 P <0.05	-	0.78 Vs 0.90 P <0.05	-
Valeria Cantoni et al. [22]	2021	MACE 64 ±10 YEARS 73 % SINGLE CENTER	453/41	SVM	2.5±0.5 YEARS	P=0.043 Vs P<0.001	-	-	-
Richard Rios et al. [23]	2021	MACE 65 ±12 YEARS 57% MULTICENTER	2041/3541	XGBoost	4.7±1.5 YEARS	0.69 Vs 0.75 P <0.05	0.69 Vs 0.79 P<0.001	-	-
Ananya Singh et al. [24]	2022	ALL CAUSE MORTALITY 71 YEARS 60% SINGLE CENTER	4735/877	CNN	6 YEARS	0.60 Vs 0.82 P<0.001	-	0.75 Vs 0.82 P <0.05	-
Eero Lehtonen et al. [25]	2023	MACE 62 ±9 YEARS 42% SINGLE CENTER	241/210	XGBoost	4 YEARS	-	-	-	P <0.05**
Luis Eduardo Juarez-Orozca et al. [26]	2023	ALL CAUSE MORTALITY-MI 61 YEARS 43 % SINGLE CENTER	739/46	GBM	6.1 YEARS	-	-	-	p=0.002**
Fares Alahdad et al. [27]	2023	MACE 61.1 ±14.2 YEARS 54% MULTICENTER	956/102	Auto Sklearn	31 months	-	-	-	P<0.001**
Ananya Singh et al.-internal [28]	2023	ALL CAUSE MORTALITY-MI 64 YEARS 57% MULTICENTER	20201/1913	HARD MACE-DL	4.6 YEARS	0.63 Vs 0.76 P<0.001	-	0.72 Vs 0.76 P <0.05	-
Ananya Singh et al.-external [28]	2023	ALL CAUSE MORTALITY-MI 68 YEARS 54% SINGLE CENTER	9019/719	HARD MACE-DL	3.5 YEARS	0.65 Vs 0.73 P<0.001	-	0.70 Vs 0.73 P <0.05	-

Figure 3. Summary of studies in which a prognostic prediction model was developed with ML using SPECT images.

Results: Our systematic search yielded twelve retrospective studies, investigating SPECT-based ML models for prognostic prediction in CAD patients, with a total sample size of 73,023 individuals. Several of these studies demonstrate the superior prognostic capabilities of ML models over traditional logistic regression (LR) models and TDP, especially when incorporating demographic data alongside SPECT imaging. Notably, the integration of demographic information with SPECT imaging in ML frameworks shows statisti-

cally significant improvements in prognostic performance (Figure 2).

Conclusions: Our review suggests that ML models leveraging SPECT imaging data, either independently or in conjunction with demographic information, hold promise for improved prognostic prediction in patients with CAD. These findings warrant further investigation into the development and validation of robust ML models for enhanced clinical decision-making in CAD management.

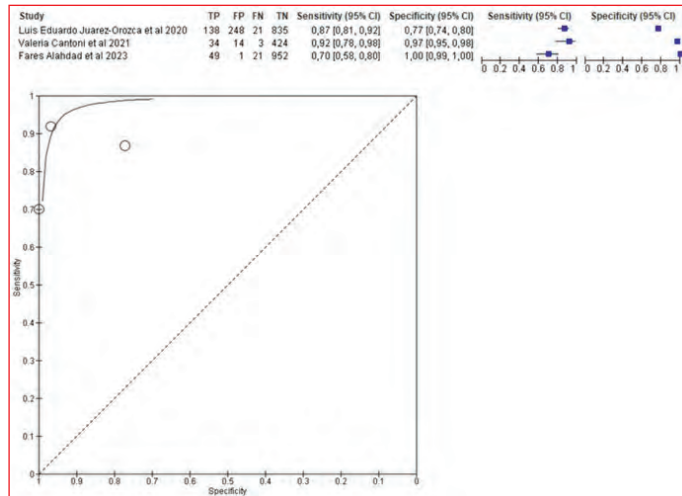


Figure 4. Forest plot and SROC plot of sensitivity and specificity on MACE outcomes with SPECT imaging based ML models.

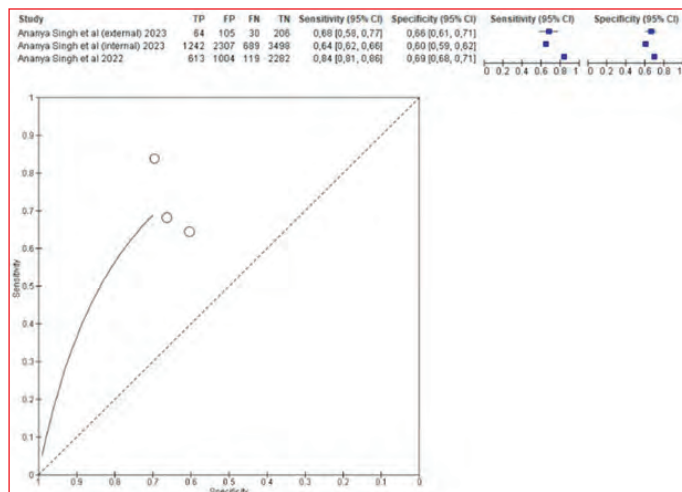


Figure 5. Forest plot and SROC plot of sensitivity and specificity on all cause mortality outcomes with SPECT imaging based ML models.

of 4th, 5th, 6th grade students who are continuing their education at medical faculties in Türkiye.

Methods: In our study, we created questions and multiple choice answers with 10 different ECG images via Google-forms in order to evaluate ECG information. This “ECGLOGY Knowledge Assessment Questionnaire” that we created was applied online to 4th, 5th, 6th grade students in all medical faculties in Türkiye between 08.04.2024 and 08.05.2024.

Results: In the study, the faculties and grades of the students, cardiology internship status were evaluated. 890 students from 45 different medical faculties participated in the study. 52.4% of the participants are women and 47.6% are men. 33.5% of the participants are grade 4 students, 30.1% are grade 5 students and 36.4% are grade 6 students. 89.6% (791) of 890 students have completed cardiology internship, 10.4% (92) have not completed cardiology internship. When the participants were asked about their ECG knowledge level; 52.2% (461) rated their ECG knowledge as moderate, 24.3% (215) rated it as good, 17.8% (157) rated it as bad and 4.3% (38) rated it as very good. When the participants were asked about the importance of ECG in their professional lives; 90.8% (802) found it very important, 8.3% found it moderately important and 0.7% (6) found it less important. In our study, 9 of the participants scored 0 points, 16 scored 1 point, 23 scored 2 points, 48 scored 3 points, 70 scored 4 points, 107 scored 5 points, 118 scored 6 points, 153 scored 7 points, 147 scored 8 points, 129 scored 9 points and 70 scored 10 full points. The most correctly marked question in the survey is the question of the normal ECG image with a ratio of 82.8%. The question that was most incorrectly asked in the survey was the question of 2:1 atrioventricular block with a ratio of 36.4%. According to the survey results; the average score was found to be 6.56 out of 10.

Conclusions: In the study, the effect of ECG education in medical schools in Türkiye on students’ competency in interpreting electrocardiography can be evaluated as sufficient to recognize normal ECG, but it can be mentioned that it is insufficient to recognize pathological ECGs. As a result, our study emphasizes that ECG education, which is a basic and universal skill, should be given more importance in the school of medicine process.

Other

PP-112

Evaluation of electrocardiography knowledge of medical school students before graduation: ecglogy study

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Background and Aim: The aim of this research is to evaluate the competency in interpreting electrocardiography (ECG)

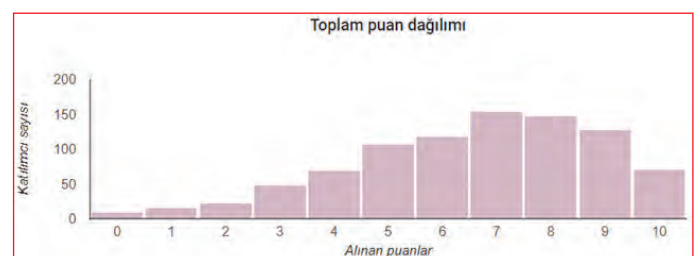
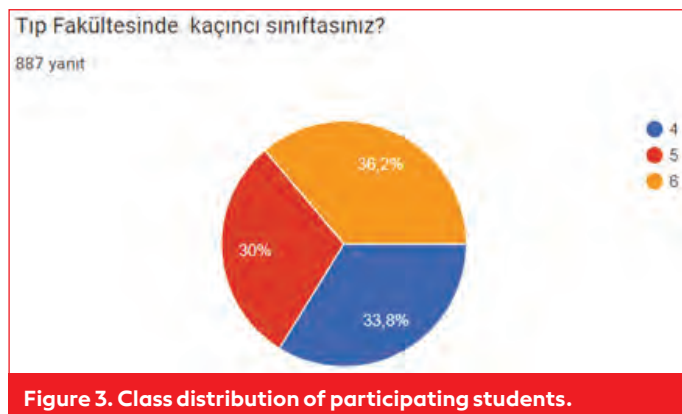
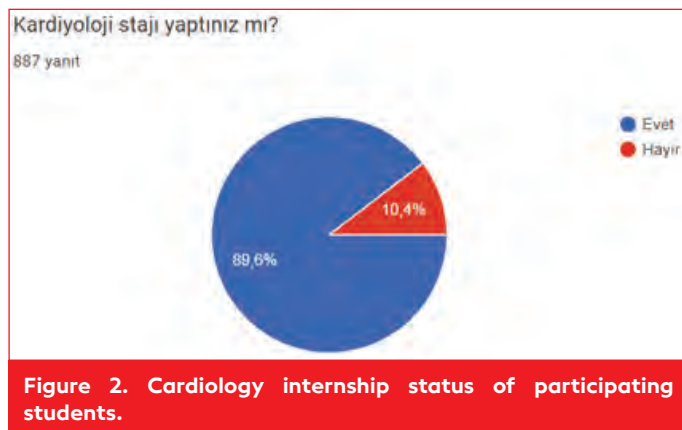


Figure 1. Study results.



Other

PP-114

Cardiac rehabilitation is associated with better long-term outcomes after coronary artery bypass grafting

Hicaz Zencirkiran Ağuş, Dilara Dünder, Gizemnur Coşkun, Serkan Kahraman, Ahmet Güner, Mehmet Altunova, Tuğba Aktemur, Ezgi Gültekin Güner, Ali Kemal Kalkan, Mustafa Yıldız, Mehmet Ertürk

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Background and Aim: Cardiac rehabilitation (CR) is a multidisciplinary, systematic programme to providing evidence-based secondary prevention therapies for patients with cardiovascular disease. Most studies evaluate CR studies of chronic coronary artery disease, percutaneous coronary intervention and coronary artery bypass grafting (CABG) populations; but data of post-CABG CR and long term outcomes are lacking. The aim of our study was to determine the influence of CR participation on major adverse cardiac events (MACE), all cause mortality and morbidity after CABG.

Methods: We enrolled 385 patients who participated in CR program after CABG between 2017 and 2023. CR programme was performed 6-8 weeks after CABG in the CR unit of our

hospital. One group (CR +) included 207 patients who participated in CR program and the control group (CR -) included 178 patients who did not want to, could not be participated or attended <5 days in CR program after CABG surgery. Patients completed nearly 15 sessions. We retrospectively analyzed rehospitalization, revascularization, atrial fibrillation, cerebrovascular events, mortality at first year and long term. In our study we defined MACE as all cause mortality, myocardial infarction and cardiac rehospitalisation.

Results: 23% of patients were female in the CR - group and 28% in CR + group. Median follow-up time was 4 years. The CR + group tended to have similar first year events as CR-group. But there was a lower incidence of long term MACE ($p=0.002$), rehospitalisation ($p=0.036$), revascularisation ($p=0.014$), myocardial infarction ($p=0.043$) and cerebrovascular accident ($p=0.016$) in outpatient CR group compared to no-CR group. Long term all cause and cardiovascular mortality didn't differ between groups ($p=0.091$).

Conclusions: The risk long term MACE was markedly decreased in patients who attended CR compared with those who did not. These findings highlights the importance of patient referral and participation in CR after CABG.

Table 1. Baseline characteristics of the CR group and the No-CR group

	CR - group (n=178)	CR + control group (n=207)	p value
Age	63 ± 9	65 ± 8	0.027
Cigarette (%)	115 (64.6)	70 (33.8)	<0.01
COPD (%)	26 (14.6)	24 (11.6)	0.381
DM (%)	103 (57.9)	124 (59.9)	0.685
HT (%)	102 (57.3)	133 (64.3)	0.163
MI history (%)	38 (21.3)	44 (21.3)	0.982
CVA (%)	13 (7.3)	9 (4.3)	0.213
AF (%)	6 (3.4)	15 (7.2)	0.095
Redo CABG (%)	2 (1.1)	1 (0.5)	0.443
LIMA grafting (%)	157 (88.2)	185 (89.4)	0.716
Creatinine	1.61 ± 7.75	0.94 ± 0.26	0.052
Albumine	42.6 (38.6-44.1)	42 (38.4-44.3)	0.63
T. cholesterol	182 ± 50	183 ± 50	0.783
LDL	106 ± 44	107 ± 39	0.918
HDL	38 (32-44)	39 (34-47)	0.095
Triglyceride	165 (108-242)	149 (102-203)	0.122
Leucosite	9.24 (7.74-11)	8.67 (7.18-10.82)	0.171
Neutrophil	5.81 (4.59-7.07)	5.26 (4.24-7.1)	0.099
Lymphocyte	2.36 (1.84-2.97)	2.31 (1.84-2.91)	0.898
Platelet	255 (205-303)	238 (202-297)	0.093
EF	55 (45-60)	55 (45-60)	0.116

AF: Atrial fibrillation; CABG: Coronary artery bypass grafting; COPD: Chronic obstructive pulmonary disease; CR: Cardiac rehabilitation; DM: Diabetes mellitus; EF: Ejection fraction; HT: Hypertension; LIMA: Left internal mammarian artery; MI: Myocardial infarction; T. cholesterol: Total cholesterol.

Table 2. Clinical outcomes

At 1 year			
	CR -	CR +	p value
AF (%)	14 (7.9)	14 (6.8)	0.678
MACE (%)	14 (7.9)	10 (4.8)	0.22
Rehospitalisation (%)	11 (6.2)	11 (5.3)	0.715
Revascularisation (%)	7 (3.9)	6 (2.9)	0.575
AMI (%)	7 (3.9)	3 (1.4)	0.114
CVA (%)	2 (1.1)	0	0.213
CV mortality (%)	2 (1.1)	2 (1.0)	0.63
All cause mortality (%)	0	1 (0.5)	0.538
Long term follow up			
AF (%)	11 (23.6)	10 (4.8)	0.561
MACE (%)	34 (19.1)	17 (8.2)	0.002
Rehospitalisation (%)	30 (16.9)	20 (9.7)	0.036
Revascularisation (%)	22 (12.4)	11 (5.3)	0.014
AMI (%)	17 (9.6)	9 (4.3)	0.043
CVA (%)	9 (5.1)	2 (1.0)	0.016
CV mortality (%)	2 (1.1)	3 (1.4)	0.571
All cause mortality (%)	9 (5.1)	4 (1.9)	0.091

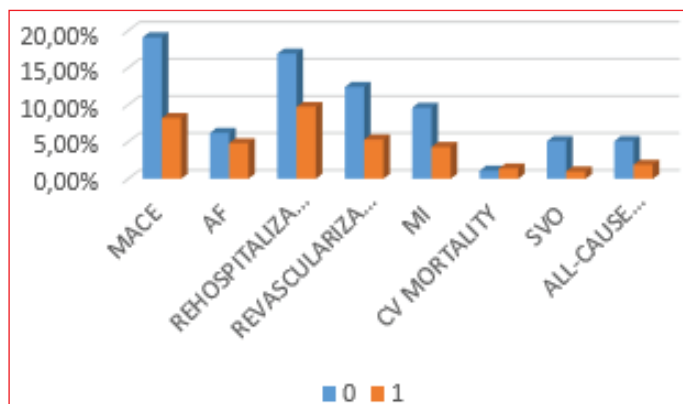


Figure 1. Comparative analysis of long-term outcome for the CR+ and CR- group.

Other

PP-115

Bridging educational gaps in familial hypercholesterolemia: A machine learning model for personalized learning

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Background and Aim: Educators in cardiology are tasked with integrating rapid advancements in cardiovascular medicine into cohesive curricula, necessitating continuous updates to

incorporate the latest clinical trials and evidence-based practices. The integration of research into clinical education further complicates training, as educators must balance teaching responsibilities with research guidance. With limited time and resources, there is a felt need for innovative methods of knowledge distribution among medical students. Machine learning (ML) is increasingly utilized in personalized education, providing tailored learning experiences based on individual performance metrics. This study employs machine learning to classify students based on their educational needs in the specific field of Familial Hypercholesterolemia (FH).

Methods: In this study, a questionnaire comprising 24 questions with 5 different Principal Components (PC) factor (Table 1) was utilized to assess the knowledge and awareness of medical students regarding FH. Data was collected from 263 students at Ege University Medical School. A custom Python code was employed to generate an arbitrary dataset consisting of 5.000 records for 11 different student groups based on their study needs and type (Table 2), resulting in a total of 55.000 training records. FastTreeOva was determined to exhibit superior performance and accuracy for the task and was therefore selected as the final model. This model was utilized to evaluate the answers of each student, producing scores that indicated the degree of fit to specific student groups-ranging from 1 (complete fit) to 0 (no fit). Based on these scores, the group with the highest score was identified as the best fit for each student.

Results: The results of the study revealed distinct educational needs among the medical students regarding FH. The “Risk Comprehension Gap” group was the most fitting, with an average score of 0.19, indicating a significant area of weakness for 139 out of the 263 students assessed. Following this, the “Biochemical Literacy Lapse” group had an average score of 0.13, identifying it as a primary weakness for 23 students. Conversely, the “Clinico-Conceptual Deficient” group received the lowest fitting score at 0.02, with no students identified as having this as a weakness point, suggesting a lesser need for educational focus in this area.

Conclusions: The use of ML in this study highlights its potential to enhance medical education by personalizing learning experiences based on individual needs. By accurately identifying knowledge gaps among students regarding FH, targeted educational strategies can be implemented more effectively. This approach is particularly vital in regions with limited educational resources, where optimizing learning paths and focusing on critical areas can significantly impact educational outcomes.

Table 1. Principal Components Factors

Principal Component	Description
PC1	General disease understanding and clinical applications
PC2	Diagnostic criteria and management strategies
PC3	Biochemical markers and lipid profiles
PC4	Treatment approaches and lifestyle impacts
PC5	Cardiovascular risks associated with cholesterol levels

Table 2. Student groups based on their study needs and type

Group Name	Description of Weakness	Principal Components
Clinico-Conceptual Deficient	Lacks foundational knowledge of FH's general understanding and clinical applications.	PC1
Diagnostic Acumen Deficit	Struggles with understanding diagnostic criteria and management strategies for FH.	PC2
Biochemical Literacy Lapse	Has limited knowledge of biochemical markers and lipid profiles associated with FH.	PC3
Therapeutic Insight Void	Finds it difficult to grasp the impact of treatment approaches and lifestyle changes on managing FH.	PC4
Risk Comprehension Gap	Lacks comprehension of the cardiovascular risks associated with elevated cholesterol levels.	PC5
Foundational Knowledge Void	Struggles with both the general understanding of the disease and the specific diagnostic criteria.	PC1, PC2
Strategic Application Gap	Has difficulties with diagnostic strategies and understanding how lifestyle and treatment influence FH.	PC2, PC4
Lifestyle-Risk Disconnect	Fails to connect the dots between lifestyle interventions and their risk reduction effects on cardiovascular health.	PC4, PC5
Bio-Risk Analysis Deficiency	Finds it challenging to understand and apply biochemical knowledge and to assess broader cardiovascular risks effectively.	PC3, PC5
Integrative Understanding Deficit	Has comprehensive difficulties spanning general knowledge, biochemical insights, and the practical implications of treatments and lifestyle on FH.	PC1, PC3, PC4
Knowledgeably Proficient	Shows a relatively strong grasp of all aspects covered by the PCA.	None (Strong in all PCs)

Other

PP-116

Predictive value of inflammatory scores for left atrium thrombosis in ischemic stroke without atrial fibrillation

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Background and Aim: The primary cause of left atrial thrombus (LAT) in ischemic stroke (IS) patients is atrial fibrillation (AF). Case control studies in the literature indicate that LAT can occur in 6-9% of stroke patients with sinus rhythm during etiological investigations. However, the available methods to predict LAT in these patients are very limited in the current literature. General inflammation markers have been proposed as prognostic tools for predicting IS. This study

compared ten different inflammation scores to identify the most effective score for predicting LAT in patients with IS without AF.

Methods: In this single-center, retrospective study, we included 303 consecutive patients with IS. Each patient underwent a transesophageal echocardiography (TEE) examination within 10 days of admission to detect the presence of left atrial thrombus (LAT). To identify independent predictors of LAT, we conducted a multivariate logistic regression analysis.

Results: A total of 303 patients were confirmed to have IS and included in the analysis. LAT despite 34 patients at the TEE examination. The patients were categorized into two groups based on their having LAT status (Table 1). Based on the results of the univariate regression analysis The Prognostic Nutritional Index (PNI) ($p=0.035$), Systemic Inflammatory Index (SII index) ($p=0.045$), Platelet-lymphocyte ratio (PLR) ($p=0.023$), C-Reactive Protein-Albumin Ratio (CAR) ($p=0.009$), were identified as statistically significant predictors of LAT. Based on the results of the multivariate regression analysis, the CAR ($p=0.003$) emerged as the only statistically significant predictor (Table 2). The Receiver Operating Characteristic (ROC) analysis demonstrated an Area Under the Curve (AUC) of CAR: 0.7409 ($p<0.001$) for predicting Left Atrial Thrombus In ischemic Stroke Without Atrial Fibrillation (Figure 1).

Conclusions: Among the ten inflammation scores evaluated, the PNI, HALP and CAR were statistically significant predictors of LAT in IS patients without AF. CAR was identified as the optimal score.

Table 1. Baseline demographic features of patients.

	Left Atrial Thrombosis(-) (n=269)	Left Atrial Thrombosis (+) (n=34)	P value
Age	69 (58-80)	66 (56-79)	0,433
Gender, male(%)	155(%58)	20(%59)	0,732
HT	197 (%73,5)	21 (%61,8)	0,150
DM	103 (%38,3)	6 (%17,6)	0,018
COPD	20 (%7,7)	0 (%0)	0,388
Dementia	17 (%8,4)	0 (%0)	0,139
CanCer	9 (%4,4)	4 (%16,7)	0,015
CKD	34 (%12,7)	6 (%17,6)	0,422
PAF	11 (%4,5)	11 (%32,4)	<0,001
Hospitalization	5 (3-9)	8,5 (4-19)	0,053
In-Hospital Mortality	15 (%7,4)	4 (%16,7)	0,118

Table 2. Evolution of inflammation scores using univariate and multivariate regression

	<u>Univariate regresyon analysis</u>			<u>Multivariate regresyon analysis</u>		
	OR	%95 GA	P value	OR	%95 GA	P value
PLR	1,0045	1,0006-1,0084	0,023			
PAN index	1,0003	1,0000-1,0007	0,083			
PNI	0,9408	0,8888-0,9958	0,035			
SII index	1,0005	1,0001-1,0008	0,045			
HALP	0,9884	0,9690-1,0082	0,248			
CAR	1,2405	1,0536-1,4607	0,009	2,7001	1,3874-5,2547	0,003
Naples score	1,2496	0,7815-1,9980	0,352			
Naples group	1,6530	0,6324-4,3212	0,305			

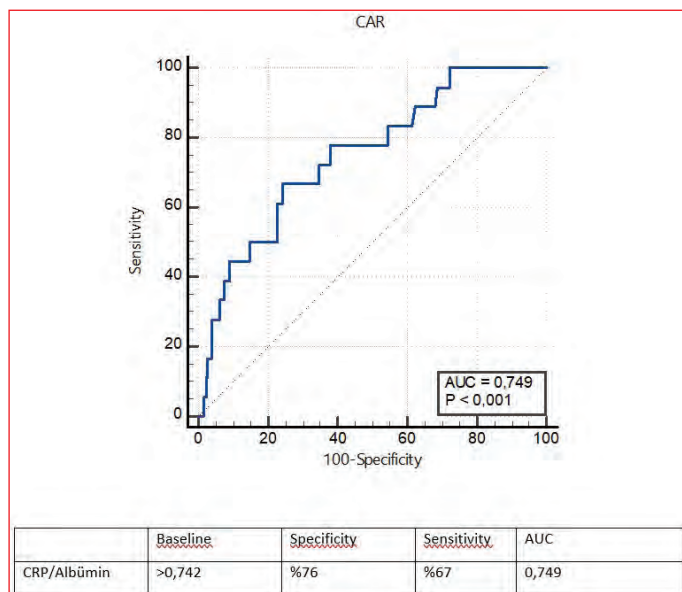


Figure 3. Performance of CAR for predicting Left Atrial Thrombus In ischemic Stroke Without Atrial Fibrillation.

Other

PP-119

The irrevocable point in forming prominent atherosclerotic plaques: Hemodynamic insights from fatty streaks

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Background and Aim: Atherosclerosis is a condition characterized by the accumulation of cholesterol and other plaque components in arterial walls, constituting a significant cause of cardiovascular diseases. This complex process often begins with the appearance of fatty streaks (FS) in the aortic arch and abdominal aorta. Historically, the contributions of FS to atherosclerosis have been studied from molecular and

biochemical perspectives. This study investigates the hemodynamic changes caused by FS.

Methods: This study is based on the CHAOS dataset, which includes CT scans of 40 individuals collected retrospectively from the PACS system of Dokuz Eylül University Hospital. A custom Python script was developed for the 3D reconstruction of the aorta and its branches using libraries such as numpy, PIL, ezdxf, and shapely.geometry. Various thicknesses of small, long, and thin bumps were used to simulate the initiation of atherosclerosis and FS. Autodesk CFD was utilized to simulate blood flow and estimate endothelial shear stress (ESS) and other hemodynamic factors. The boundary conditions set the aortic blood velocity at 0.68 m/s, with velocities for branching arteries-celiac, superior mesenteric, and renal-assigned values of 100 cm/s, 125 cm/s, and 75 cm/s, respectively. The k-epsilon turbulence model was selected, and the modified Petrov-Galerkin method was applied. Numerical results from CFD were visualized and evaluated using Python.

Results: The study indicates that there are quantitative changes in ESS when fatty streaks are present in the aortic wall. The graph of the aorta without fatty streaks is relatively linear concerning position and has an average ESS

value of 3.5 Pa. However, when fatty streaks are added, there is a clear and significant change in ESS, which assumes a double-peaked shape with the first peak at 6.8 Pa and the second peak at 6.3 Pa (Figure 1). Areas with an ESS greater than 5.5 Pa exhibit a distribution resembling a metaphorical dam on the surface of the FS. As atherosclerosis progresses, the metaphorical dam-like structure becomes more pronounced, and the differences in ESS become more evident. A thickness of 1.5 mm is identified as the irrevocable point in forming a prominent atherosclerotic plaque (Figure 2). Upon increasing the blood velocity and pressure in the simulation by 40%, the areas with an ESS under 4.5 decreased in size by 80%. Moreover, areas with an ESS exceeding 5.5 form a continuous shape, effectively eliminating spaces of low ESS that were previously trapped between the high ESS surface (Figure 3).

Conclusions: The presence of an irrevocable point in FS thickness and the effectiveness of the increase in blood flow demonstrate the importance of early preventive measures such as engaging in sports and maintaining a healthy lifestyle during the 30s and early 40s, when the early stages of atherosclerosis are typically forming for most people. Additionally, more frequent consultations with a physician or cardiologist are advisable.

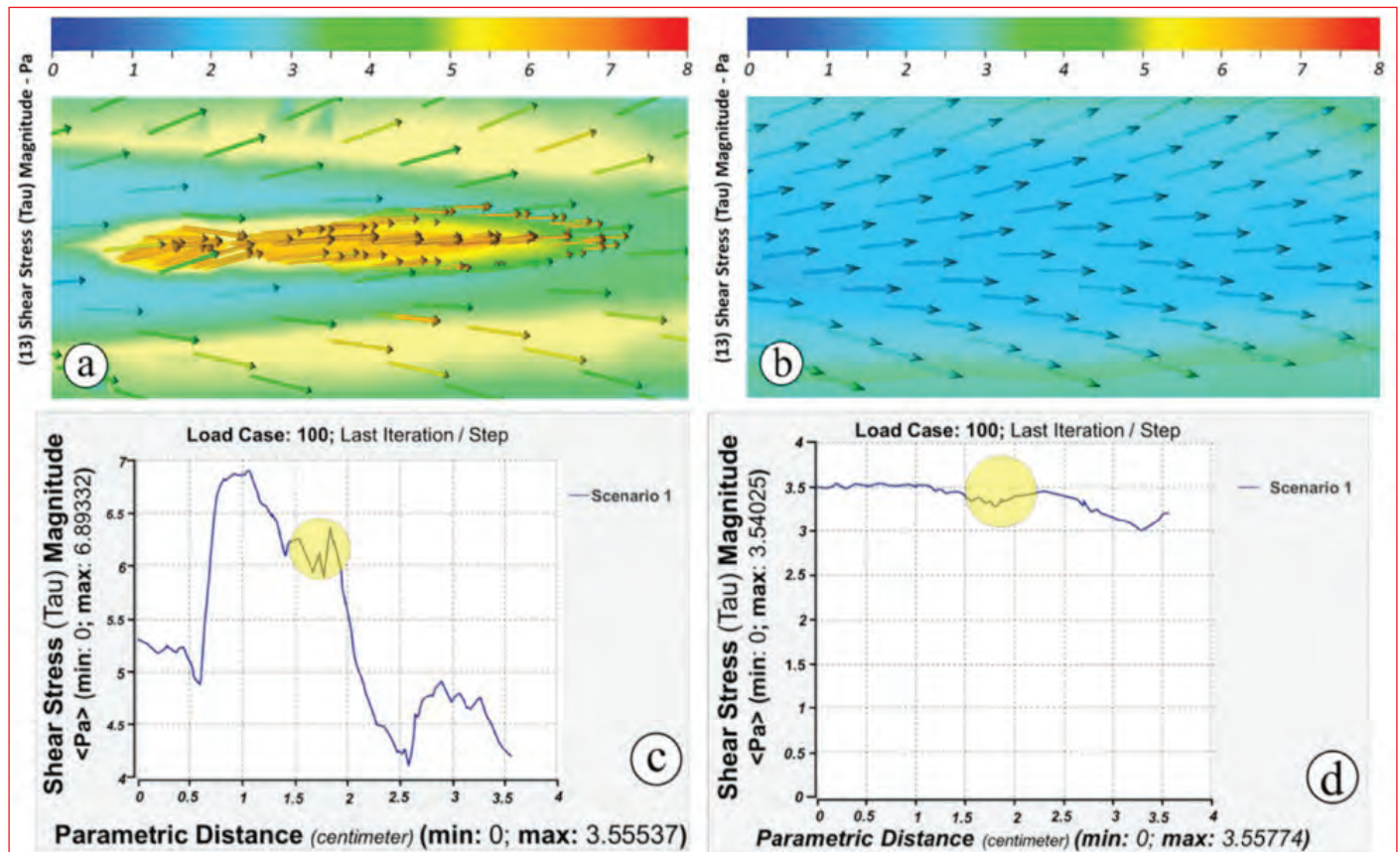


Figure 1. (a) ESS map in the presence of FS. The metaphorical dam candidate is highlighted in yellow with an oval shape; (b) The same geographical area without the presence of fatty streaks. (c) The changes in ESS of the aortic wall in an area with fatty streaks; (d) The changes in ESS of the aortic wall in an area without fatty streaks.

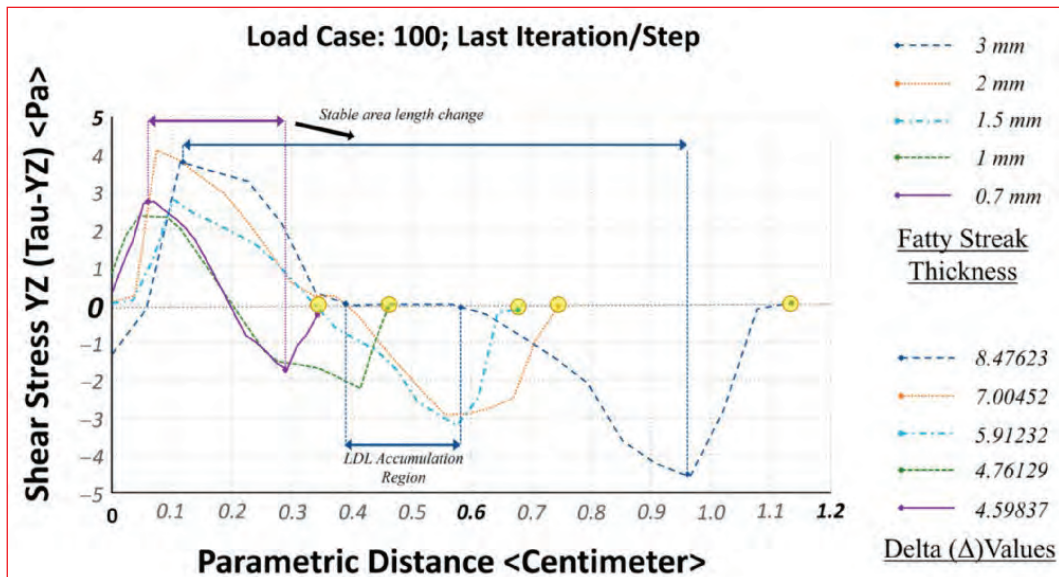


Figure 2. The impact of the increase in the thickness of the aortic wall on ESS distribution. The difference in ESS increases from 2.58 Pa in the original model to 8.48 Pa in a model with a 3 mm thickness increase. The stable area length in terms of ESS increases from ~0.2 cm to ~0.8 cm.

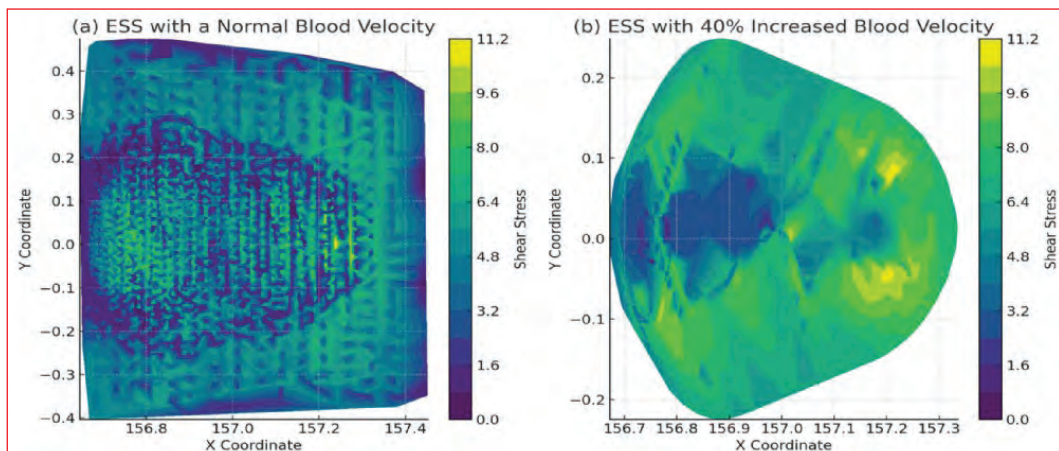


Figure 3. ESS Contours.

Other

PP-124

Comparison of inflammatory scores as prognostic markers for in-hospital mortality in patients with infective endocarditis

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Background and Aim: General inflammation markers have been proposed as prognostic tools for predicting in-hospital mortality in infective endocarditis (IE). Nonetheless, it is unclear whether these markers provide additional prognostic value over established indicators. This study compared nine different inflammation scores to assess their effectiveness in enhancing the prediction of in-hospital mortality.

Methods: Patients with IE diagnosed between 2017 and 2023 at two cardiology centers in İstanbul were included in this study. Pre-treatment inflammation markers were obtained from the clinical laboratory information system. In-hospital mortality prognostication was assessed using Cox proportional hazards models

Results: A total of 146 patients with a preliminary diagnosis of IE were initially considered for this study. After applying exclusion criteria, 102 patients were confirmed to have IE and included in the analysis. 32 patients died during their hospital stay (Table 1). The patients were categorized into two groups based on their In-hospital mortality status. The Prognostic Nutritional Index (PNI) ($p=0.011$), OR: 0.924 (95% CI: 0.870-0.982), the Platelet-to-Lymphocyte Ratio (PLR) ($p=0.009$), OR: 0.993 (95% CI: 0.988-0.998) and the modified Glasgow Prognostic Score (mGPS) ($p=0.05$) OR: 2.579 (95% CI: 0.994-6.643), were identified as statistically significant predictors of In-hospital mortality in IE patients. Based on the results of the multivariate regression analysis, the PNI ($p=0.035$), (OR: 0.921 95% CI: 0.853-0.994) emerged as the only statistically significant predictor (Table 2).

Conclusions: Among the nine inflammation scores evaluated, the PNI, PLR and mGPS were statistically significant predictors of In-hospital mortality in patients with IE. PNI was identified as the optimal score based on its statistical significance and multivariate regression analysis.

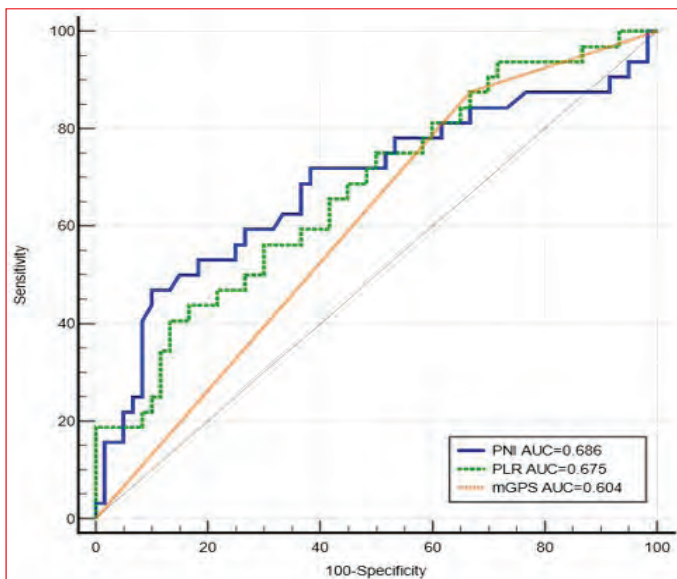


Figure 1. Comparison of scores predicting in-hospital mortality using ROC analysis.

Other

PP-125

Iodide-triggered mumps after fistulogram

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Background and Aim: Iodide mumps is an infrequent adverse reaction to iodine-containing contrast agents administered during angioplasty or contrast-enhanced imaging methods. It usually subsides promptly without complications. The medical literature suggests that this side effect occurs more frequently in patients with renal insufficiency. In this par-

ticular case, the patient was unable to undergo immediate hemodialysis after the procedure due to potential bleeding complications. This may have prolonged the duration of the contrast agent in the plasma and resulted in the chance of side effects. The pathogenesis of this condition has not been definitively determined, and there is no specific treatment. Clinicians should be mindful of its possibility in patients suffering from neck swelling post-interventional treatment.

Methods: A 77-year-old male patient was admitted to our clinic for percutaneous transluminal angioplasty to treat stenosis of the arteriovenous fistula in his right upper extremity for haemodialysis. The patient had a medical history of diabetes, hypertension, and chronic renal failure, requiring haemodialysis three times a week. Balloon angioplasty was successfully performed using an 8.0 mm x 60 mm compliant balloon for stenotic lesion.

Results: The patient reported swelling in his upper neck one day after the procedure. During the physical examination, a painless, symmetrical mass was discovered in the submandibular region. The patient was afebrile with no signs of infection or other symptoms. Laboratory tests were essentially normal excepting blood urea nitrogen and creatinine. The patient underwent a bilateral neck ultrasound which revealed an increase in the size of the submandibular glands accompanied by swollen, homogeneous glandular tissue on both sides. Neck computed tomography was performed and revealed enlarged submandibular glands on both sides. Based on the patient's clinical history and laboratory results, we suspected iodide mumps following administration of non-ionic iodinated contrast medium. The patient underwent haemodialysis 24 hours post-intervention and subsequently submandibular swelling resolved without specific treatment. The patient was completely back to normal 36 hours later. The patient has been discharged from the hospital.

Conclusions: The probability of developing acute sialadenitis is directly proportional to serum iodide levels and inversely related to renal function. Our patient was at an increased risk because of her end-stage renal failure. The delay in dialysis for the first 24 h after the fistulogram might have contributed further to the pathogenesis. The way to prevent recurrence of iodide mumps is to avoid using intravenous iodinated contrast in those patients again, or performing urgent dialysis within 24 h if the use of iodinated dye is essential.

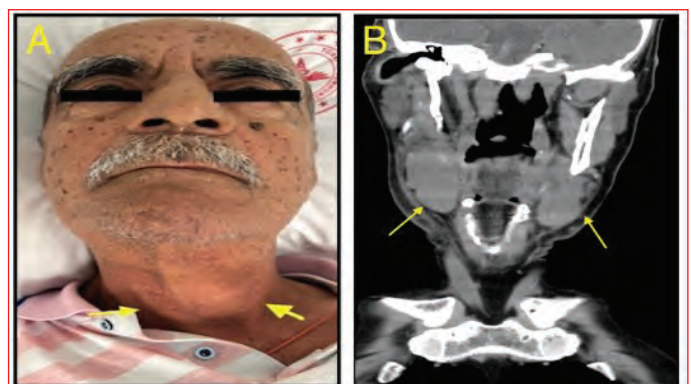


Figure 1. Iodide mumps. Significant swelling neck around the submandibular glands on presentation.

Surname Name	Surname Name	Pub Number	Surname Name	Surname Name	Pub Number
Abanoz, Oğuzhan	Abanoz, O.	PP-107, PP-090	Atmaca, Sezgin	Atmaca, S.	OP-042, OP-046, OP-047, OP-048, OP-051
Abdullaeva, Saodat	Abdullaeva, S.	PP-098	Avcı, Talha	Avcı, T.	PP-105
Ahmadi, Ahmet Şekip	Ahmadi, A. Ş.	OP-004, PP-058	Ayata, Alihan	Ayata, A.	PP-041
Akalin, Aysen	Akalin, A.	PP-067	Aydemir, Selim	Aydemir, S.	PP-060
Akaltun, Faruk	Akaltun, F.	PP-108	Aydın, Esra	Aydın, E.	OP-020
Akbulut, Müge	Akbulut, M.	PP-075, PP-091	Aydın, Fatih	Aydın, F.	PP-094
Akbulut Çakır, Merve	Akbulut Çakır, M.	PP-012	Aydın, Oğuz	Aydın, O.	PP-032, PP-071
Akdeniz, Hilal	Akdeniz, H.	PP-121, PP-122, PP-118	Aydın, Sidar Şiyar	Aydın, S. Ş.	PP-060
Akdoğan, Ali Tolga	Akdoğan, A. T.	OP-038	Aydın, Sinem	Aydın, S.	OP-051, OP-042, OP-047, OP-048
Akgün, Hüseyin	Akgün, H.	PP-047	Ayduk Gövdeli, Elif	Ayduk Gövdeli, E.	OP-043, OP-049, PP-063, PP-065,
Akın, Mustafa	Akın, M.	OP-071, PP-027	Aygün, Ahmet Atıl	Aygün, A. A.	PP-099
Akın, Yeşim	Akın, Y.	PP-054	Ayseviniç, Berrin	Ayseviniç, B.	OP-039
Aksoy, Fatih	Aksoy, F.	PP-083, PP-097	Ayyıldız, Selman	Ayyıldız, S.	PP-118, PP-121, PP-122
Aksu, Derya	Aksu, D.	PP-072	Babaoğlu, Mert	Babaoğlu, M.	PP-109, PP-116
Aksu, Uğur	Aksu, U.	PP-072	Babur Güler, Gamze	Babur Güler, G.	OP-042, OP-046, OP-047, OP-048, OP-051, PP-050
Aksüyek, Soner	Aksüyek, S.	PP-003, PP-069	Bacaksız, Ahmet	Bacaksız, A.	PP-107, PP-090
Aktemur, Tuğba	Aktemur, T.	PP-114	Bağcı, Ulaş	Bağcı, U.	PP-109, PP-116, PP-124
Akyol, Ahmet	Akyol, A.	OP-028	Bağcı, Ali	Bağcı, A.	PP-083, PP-097
Akyürek, Ömer	Akyürek, Ö.	PP-044	Bahtiyar, Burak	Bahtiyar, B.	OP-058
Akyüz, Ali Rıza	Akyüz, A. R.	OP-014, PP-019	Bakalli, Aurora	Bakalli, A.	PP-082
Aladağ, Nesim	Aladağ, N.	PP-106	Bakhshaliyev, Nijad	Bakhshaliyev, N.	OP-036, PP-062
Alhan, Cem	Alhan, C.	OP-028	Balaban, İsmail	Balaban, İ.	PP-049, PP-068
Almasri, Muayad	Almasri, M.	OP-048	Ballı, Mehmet	Ballı, M.	OP-016
Altan, İdil	Altan, İ.	PP-108	Barış, Veysel Özgür	Barış, V. Ö.	OP-016
Altın, Ali Timuçin	Altın, A. T.	PP-044	Barman, Hasan Ali	Barman, H. A.	PP-051
Altın, Cihan	Altın, C.	OP-030, OP-037	Bartunek, Jozef	Bartunek, J.	OP-036
Altın, Eslem	Altın, E.	PP-112	Başaran, Özcan	Başaran, Ö.	OP-056, OP-070, OP-074
Altinkaya, Onur	Altinkaya, O.	PP-060	Baskovski, Emir	Baskovski, E.	OP-044, PP-044
Altınsoy, Meltem	Altınsoy, M.	OP-055	Baştopçu, Murat	Baştopçu, M.	OP-028
Altıparmak, İbrahim Halil	Altıparmak, İ. H.	OP-002, OP-041, PP-007	Bayir Garbioğlu, Duygu	Bayir Garbioğlu, D.	PP-056
Altunkeser, Bülent Behlül	Altunkeser, B. B.	OP-026	Bayraktaroğlu, Selen	Bayraktaroglu, S.	OP-029
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Altuntaş, Seda	Altuntaş, S.	OP-072	Bayram, Zübeyde	Bayram, Z.	PP-068
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Anker, Stefan	Anker, S.	OP-036	Belen, Erdal	Belen, E.	PP-053
Apaydın, Ziya	Apaydın, Z.	PP-053	Beral, Ayberk	Beral, A.	PP-039, PP-078, PP-089
Arı, Hasan	Arı, H.	PP-003, PP-069	Bergman, Martin	Bergman, M.	OP-036
Arı, Selma	Arı, S.	PP-003	Berisha, Blerim	Berisha, B.	PP-082
Arslan, Ayça	Arslan, A.	PP-061	Beşiroğlu, Fatih	Beşiroğlu, F.	OP-023, OP-024
Arslan, Enes	Arslan, E.	OP-046	Bıçakçı, Yahya Dağcan	Bıçakçı, Y. D.	PP-095
Arslan, Şükrü	Arslan, Ş.	OP-003, PP-016	Biçer Yeşilay, Asuman	Biçer Yeşilay, A.	OP-002, OP-041, PP-007
Arslanhan, Gökhan	Arslanhan, G.	OP-028	Bilen, Mehmet Nail	Bilen, M. N.	PP-047
Artaç, İnanç	Artaç, İ.	PP-061	Bilge, Ahmet Kaya	Bilge, A. K.	OP-049, PP-063
Arter, Ertan	Arter, E.	PP-079	Bilgin, Aslıhan Gizem	Bilgin, A. G.	OP-031
Arya, Bekir Kaan	Arya, B. K.	PP-100	Bilgin, Büşra	Bilgin, B.	OP-072
Arya, Helen	Arya, H.	PP-048	Bilgin, Mehmet Emin	Bilgin, M. E.	OP-003
Arya, Muhammad	Arya, M.	OP-064, PP-013, PP-048, PP-100, PP-102, PP-115, PP-119,	Bilir, Gürkan	Bilir, G.	OP-007
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Aslan, Muzaffer	Aslan, M.	PP-025	Biter, Halil İbrahim	Biter, H. İ.	PP-053
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Aslan, Serkan	Aslan, S.	OP-015, OP-022	Bora, Sebnem	Bora, S.	PP-119
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Atalay, Mustafa	Atalay, M.	OP-058	Bozat, Tansin	Bozat, T.	PP-003
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Atmaca, Mert Murat	Atmaca, M. M.	PP-116			

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Bütün, Zeynep Kezban	Bütün, Z. K.	PP-037	Dağlı, Mustafa necati	Dağlı, M. n.	OP-019
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Candemir, Başar	Candemir, B.	PP-044	Dalgıç, Onur	Dalgıç, O.	OP-001
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Cebeci, Ahmet Ceyhan	Cebeci, A. C.	PP-020	Dedeoğlu, Necip Fazıl	Dedeoğlu, N. F.	PP-007
Cebeci, Hüseyin Emre	Cebeci, H. E.	PP-052	Değirmenci, Muhammet	Değirmenci, M.	OP-007
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Cızıgıcı, Ahmet Yaşar	Cızıgıcı, A. Y.	OP-022	Demir, Kenan	Demir, K.	OP-026
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