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PP-001 [Heart Failure]

Frontal QRS-T angle and cardiac electrophysiological balance as predictors of disability severity in relapsing-remitting multiple sclerosis

Çağatay Tunca[1], Betül Akıncıoğlu[1], Etkin Elifoğlu[1], Leyli Can Aynal[2], Alperen Taş[3], Ali Sezgin[1], Yusuf Bozkurt Şahin[1], Veysel Ozan Tanık[1], Bülent Özlek[4], Bülent Güven[2], Kürşat Akbuğa[1]

[1]Department of Cardiology, Ankara Etlik City Hospital, Ankara
[2]Department of Neurology, Ankara Etlik City Hospital, Ankara
[3]Department of Cardiology, Ahi Evran University Training and Research Hospital, Kırşehir
[4]Department of Cardiology, Muğla Sıtkı Koçman University, Faculty of Medicine, Muğla

Background and Aim: Multiple sclerosis (MS) is a chronic immune-mediated disorder of the central nervous system, increasingly recognized as a systemic disease with cardiovascular implications. Individuals with MS have a heightened risk of arrhythmias, myocardial infarction, and sudden cardiac death, even after adjustment for conventional cardiovascular risk factors. A major contributor to this excess risk appears to be autonomic nervous system (ANS) dysfunction caused by demyelinating lesions in the brainstem and spinal cord. These alterations can lead to impaired heart rate variability (HRV), QT interval prolongation, and increased susceptibility to arrhythmias. In this context, accessible and reliable ECG-based markers are needed to detect subclinical cardiac involvement and guide clinical risk stratification in MS. The frontal QRS-T angle (fQRS-T), an emerging marker of ventricular repolarization heterogeneity, has shown prognostic value in diverse populations. The index of cardiac electrophysiological balance (iCEB), calculated as the QT interval divided by QRS duration, offers an integrative reflection of repolarization-conduction dynamics and arrhythmic risk. However, their utility in patients with MS, particularly in association with disability severity, remains poorly defined. This study aimed to assess fQRS-T angle and iCEB values in relapsing-remitting MS (RRMS) and to evaluate their relationship with Expanded Disability Status Scale (EDSS) scores.

Methods: This retrospective, cross-sectional study included 101 clinically stable RRMS patients and 51 age- and sex-matched healthy controls who underwent standard 12-lead ECG. Patients were stratified into EDSS <6 (n=81) and EDSS ≥6 (n=20) subgroups. ECG parameters included QT, QTc, Tp-e, Tp-e/QTc, fQRS-T angle, and iCEB (plus corrected iCEBc). ECGs were manually analyzed using digital calipers by two blinded cardiologists, with the average of three consecutive beats recorded. Interobserver discrepancies >5 ms were resolved by joint review. Statistical analyses included univariate testing, multivariable logistic regression, and ROC analysis.

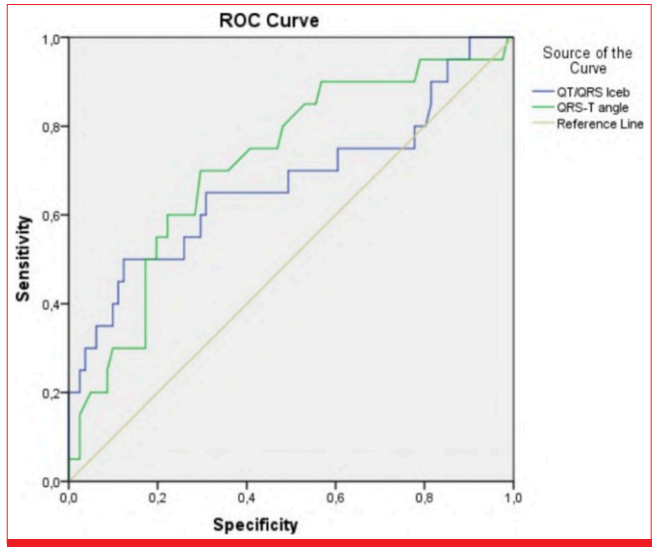

Figure 1. Receiver operating characteristic (ROC) curves for predicting severe disability (EDSS ≥6) in patients with relapsing-remitting multiple sclerosis. The green line represents the frontal QRS-T angle (AUC=0.718; cut-off: 32°), and the blue line represents the index of cardiac electrophysiological balance (iCEB=QT/QRS) (AUC=0.671; cut-off: 4.37). The diagonal dashed line indicates the reference line for no-discrimination. Both ECG-derived markers demonstrated moderate discriminative power, with the QRS-T angle showing slightly superior diagnostic performance.

Table 1. Clinical and laboratory differences between MS and control groups

Variable	MS, N=101	Control, N=51	P value
Age, years	35.19 ± 9.12	36.64 ± 9.29	0.108
LV EF, %	61.10 ± 2.27	61.30 ± 2.27	0.625
Hb, g/dl	13.67 ± 2.29	13.37 ± 2.66	0.469
Creatinine, mg/dl	0.70 ± 0.16	0.68 ± 0.16	0.424
Glucose, mg/dl	87.36 ± 8.81	87.84 ± 10.84	0.768
LDL, mg/dl	116.20 ± 35.05	122.27 ± 37.10	0.388
Sodium,mmol/l	139.91 ± 4.24	139.86 ± 5.69	0.954
WBC, 10^3/L	6.70 ± 2.27	6.40 ± 1.92	0.417
Heart rate, atım/dk	77.09 ± 11.80	74.25 ± 10.50	0.157
S wave max amplitüde, mV	10 (5-22)	10 (5-19)	0.605
S wave min amplitüde, mV	2.0 (1.0-8.0)	2.0 (1.0-8.0)	0.974
RS max, mV	13.65 ± 3.83	13.52 ± 3.59	0.837
R vawe pik time, msn	35.41 ± 5.15	35.40 ± 4.93	0.986
RWPT V1, msn	15 (10-45)	20 (10-45)	0.516
RWPT V4-6, msn	30 (20-45)	35 (25-45)	0.171
RWPT D3, msn	25 (10-45)	22 (10-45)	0.953
QT, msn	372.89 ± 36.88	361.44 ± 27.28	0.033
QTc, msn	398.47 ± 28.69	391.18 ± 22.30	0.089
QRS, msn	86.48 ± 9.32	90.0 ± 11.44	0.045
iCEB	4.35 ± 0.64	4.08 ± 0.59	0.012
iCEBc	4.66 ± 0.64	4.42 ± 0.65	0.031
Tp-e,msn	88.26 ± 18.99	77.40 ± 14.15	0.001
Tp-e/QT	0.23 ± 0.04	0.21 ± 0.03	0.004
Tp-e/QTc	0.22 ± 0.04	0.19 ± 0.03	0.002
PR, msn	140.02 ± 20.79	140.09 ± 21.80	0.986
RR, msn	792.68 ± 118.83	788.86 ± 127.05	0.864
P min, msn	100 (30-150)	80 (40-140)	0.141
Pmax, msn	70 (30-140)	80 (40-140)	0.158
P wave dispersion, msn	56.55 ± 14.31	56.13 ± 14.97	0.876
QRS-T angle	26 (2-106)	18 (2-65)	0.024

Abbreviations: Hb, Hemoglabin ; LV EF, Left Ventricüle Ejection Fraction; WBC, White Blood Cell; RWPT, R wave pik time; iCEB, Index of Cardiac Electrophysiological Balance; Tp-e, T wave peak to T wave end interval

Results: RRMS patients exhibited significantly wider fQRS-T angles and higher iCEBc compared to controls (p<0.05). Among RRMS patients, those with EDSS ≥6 had further increases in fQRS-T angle and iCEB (p<0.01). Both variables independently predicted severe disability in multivariable analysis. The fQRS-T angle demonstrated good discriminatory ability (AUC: 0.718; cut-off: 32°; 70% sensitivity, 71% specificity).

Conclusions: Frontal QRS-T angle and iCEB are independently associated with disability severity in RRMS, likely reflecting underlying autonomic dysfunction or myocardial involvement. These inexpensive, noninvasive ECG markers may serve as practical tools for early cardiovascular risk assessment. Incorporating them into clinical evaluation may support earlier intervention and improve outcomes in patients with MS.

Table 2. Comparison based on EDSS severity scores in multiple sclerosis patients

Variable	EDSS<6, N=81	EDSS>=6, N=20	P value
Age, years	34.66 ± 8.85	37.35 ± 10.10	0.241
EDSS	1.87 ± 1.26	6.82 ± 0.65	<0.001
LV EF, %	61.07 ± 2.30	61.25 ± 2.22	0.759
Hb, g/dl	13.73 ± 2.39	13.43 ± 1.85	0.601
Creatinine, mg/dl	0.71 ± 0.15	0.66 ± 0.17	0.219
Glucose, mg/dl	87.10 ± 8.56	88.47 ± 9.94	0.545
LDL, mg/dl	116.07 ± 34.18	116.76 ± 39.59	0.942
Sodium,mmol/l	139.65 ± 4.60	140.95 ± 2.01	0.222
WBC, 10^3/L	6.81 ± 2.33	6.22 ± 1.67	0.293
Heart rate, atım/dk	85.69 ± 11.03	84.0 ± 13.39	0.789
S wave max amplitüde, mV	10 (5-22)	8 (5-19)	0.148
S wave min amplitüde, mV	2.0 (1.0-8.0)	2.0 (1.0-4.0)	0.471
RS max, mV	13.84 ± 3.97	12.90 ± 3.19	0.329
R vawe pik time, msn	35.82 ± 5.29	33.75 ± 4.25	0.107
RWPT V1, msn	15 (10-45)	15 (10-25)	0.520
RWPT V4-6, msn	35 (20-45)	30 (25-45)	0.596
RWPT D3, msn	25 (10-45)	20 (10-40)	0.055
QT, msn	365.79 ± 29.40	401.65 ± 49.34	0.005
QTc, msn	393.13 ± 22.33	420.10 ± 40.25	0.009
QRS, msn	86.92 ± 9.24	84.70 ± 9.65	0.341
iCEB	4.24 ± 0.49	4.82 ± 0.94	0.016
iCEBc	4.57 ± 0.53	5.04 ± 0.90	0.035
Tp-e,msn	84.75 ± 17.10	102.50 ± 20.03	<0.001
Tp-e/QT	0.23 ± 0.04	0.25 ± 0.04	0.035
Tp-e/QTc	0.21 ± 0.04	0.24 ± 0.03	0.014
PR, msn	139.52 ± 19.60	142.71 ± 27.07	0.601
RR, msn	802.98 ± 117.13	736.78 ± 116.33	0.055
P min, msn	100 (30-150)	90 (40-140)	0.993
Pmax, msn	60 (30-140)	60 (40-100)	0.746
P wave dispersion, msn	57.10 ± 14.21	53.57 ± 14.99	0.399
QRS-T angle	23 (2-95)	42.50 (4-106)	0.003

Abbreviations: EDSS, Expanded Disability Status Scale; Hb, Hemoglabin ; LV EF, Left Ventricüle Ejection Fraction; WBC, White Blood Cell; RWPT, R wave pik time; iCEB, Index of Cardiac Electrophysiological Balance; Tp-e, T wave peak to T wave end interval

Table 3. Regression analysis for identifying parameters associated with multiple sclerosis

	Univariable regression		Multivariable regression	
Variables	OR (%95 CI)	P value	OR (%95 CI)	P value
QT, msn	1.027 (1.012-1.042)	<0.001		
QTc, msn	1.033 (1.014-1.053)	0.001		
iCEB	3.587 (1.625-7.921)	0.002	3.544 (1.572-7.878)	0.001
iCEBc	2.971 (1.365-6.466)	0.006		
Tp-e,msn	1.056 (1.023-1.090)	0.001		
Tp-e/QT	1.012 (1.007-1.017)	0.040	1.002 (0.991-1.013)	0.095
Tp-e/QTc	1.016 (1.006-1.026)	0.018		
QRS-T angle	1.034 (1.010-1.057)	0.004	1.036 (1.009-1.063)	0.010

Abbreviations: iCEB, Index of Cardiac Electrophysiological Balance; Tp-e, T wave peak to T wave end interval

Table 4. Receiver operating characteristic (ROC) analysis of MS-related predictive markers

Variables	AUC	Cut off	P	Sensitivity	Specifity
Iceb	0.671	4.37	0.018	65	65
QRS-T angle	0.718	32	0.003	70	71

PP-002 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

The impact of delta QRS on biventricular strain in patients with RVOT ventricular extrasystoles

Ahmet Özderya

Department of Cardiology, Trabzon Kanuni Training and Research Hospital, Trabzon

Background and Aim: Ventricular extrasystoles (VES) adversely affect both the right and left ventricles due to atrioventricular and ventricular dyssynchrony. Right ventricular outflow tract (RVOT) VES are generally considered relatively benign. However, to date, no study in the literature has specifically investigated the impact of QRS duration in these VES on cardiac function. This study aimed to evaluate the effect of delta QRS on both left and right ventricular function using strain imaging in patients with RVOT VES.

Methods: Eighty-four patients with electrocardiographically confirmed RVOT VES and ≥5000 VES episodes on 24-hour Holter monitoring were included. Delta QRS was calculated as the difference between the QRS duration of VES beats and that of baseline sinus rhythm. Patients were divided into two groups based on the median delta QRS value: supramedian and inframedian. Echocardiographic parameters of left and right ventricular function were compared between the two groups.

Results: Patients in the supramedian delta QRS group had a higher prevalence of hypertension, as well as elevated levels of creatinine, platelet count, LDL, and total cholesterol, while showing lower MCV and albumin levels (Table 1). Echocardiographic parameters including LV-EF, LV-EDD, aortic velocity, TAPSE, LV global longitudinal strain (GLS) (p<0.001), and RV total strain (p<0.001) differed significantly between the two groups (Table 2). In univariable regression analysis, increased delta QRS was found to be a predictor of reduced LV-GLS [OR: 0.975 (0.952–0.999), p=0.042] and reduced RV total strain [OR: 0.922 (0.887–0.958), p<0.001].

Conclusions: Our study demonstrated that increased delta QRS in patients with RVOT VES is associated with impaired left and right ventricular function. These findings highlight the clinical importance of QRS duration in VES during patient follow-up.

Table 1. Clinical characteristics of the study population

	Supramedian delta QRS duration n=42	Inframedian delta QRS duration n=42	p
Age (years)	56.1 ± 14.47	49.9 ± 16.97	0.076
Gender (F/M) (n, %)	21 / 21	16 / 26	0.272
BMI (kg/m^2)	30.08 ± 4.59	30.46 ± 5.63	0.733
Hypertension (n)	22	12	0.026
Diabetes mellitus (n)	5	4	0.724
Hyperlipidemia (n)	1	4	0.156
Fasting blood glucose (mg/dL)	94.28 ± 10.36	90.95 ± 17.71	0.296
Creatine (mg/dL)	0.88 ± 0.23	0.79 ± 0.12	0.034
Albumin (g/dL)	42.36 ± 3.28	44.4 ±2.37	0.002
LDL (mg/dL)	135.16 ± 29.4	111. 09 ± 29.81	<0.001
ALT (IU/L)	20 (10-44)	21 (12-38)	0.202
AST (IU/L)	20 (13-59)	21 (9-27)	0.064
Total Cholesterol (mg/dL)	194.35 ± 39.01	174.8 ± 42.56	0.037
Hemoglobin (g/dL)	14.07 ± 1.74	14.14 ± 1.41	0.832
Hematocrit (%)	42.37 ± 6.26	43.2 ± 3.44	0.455
White Blood Cell (x10^9/L)	7.91 (4.1-11.4)	8.4 (5.5-11.9)	0.060
Platelet (x10^9/L)	252.07 ± 42.56	206.76 ± 41.44	<0.001
MCV (fL)	86.3 (67-96.2)	90 (64-97)	<0.001

Table 2. Comparison of echocardiography parameters

	Supramedian delta QRS duration n= 42	Inframedian delta QRS duration n= 42	p
LV-EF (%)	60 (52-68)	64 (55-69)	0.016
LA	37.4 ± 5.67	36.5 ± 4.11	0.405
LV-EDD	49.52 ± 4.47	42.19 ± 8.06	<0.001
LV-ESD	31 (22-38)	30 (22-46)	0.104
Aortic velocity	156.45 ± 28.82	133.26 ± 23.41	<0.001
IVS	10.47 ± 1.51	10.09 ± 1.7	0.283
E	87.38 ± 13.96	80.08 ± 28.2	0.148
A	82.8 ± 22.54	82.57 ± 16.42	0.956
E'	10.67 ± 7.28	9.98 ± 4.31	0.480
E/E'	6.55 ± 2.19	7.91 ± 3.01	0.200
TAPSE	22.61 ± 2.74	24.57 ± 1.86	<0.001
LV-GLS	18.5 ± 2.22	20.44 ± 1.71	<0.001
RV Total Strain	17.56 ± 3.4	20.05 ± 1.91	<0.001

PP-003 [Cardiac Imaging / Echocardiography]

Myocardial deformation in pregnancy: A comparative echocardiographic analysis with non-pregnant women

Sibel Çatalkaya, Eda Özcan

Department of Cardiology, Bursa City Hospital, Bursa

Background and Aim: Pregnancy induces profound cardiovascular adaptations, including increased preload, reduced afterload, and structural remodeling of the cardiac chambers. Speckle-tracking echocardiography enables sensitive evaluation of myocardial function beyond conventional parameters. While left ventricular (LV) strain has been widely investigated in pregnancy, data on right ventricular (RV) and left atrial (LA) strain remain limited. In particular, normative values for pregnant populations are scarce, especially when using modern automated strain analysis software. This study aimed to assess myocardial deformation parameters in healthy pregnant women using automated speckle-tracking echocardiography.

Methods: In this cross-sectional observational study, 36 healthy pregnant women (24–37 weeks gestation, age 27.2 ± 4.3 years) and 28 healthy non-pregnant women (age 28.7 ± 5.6 years) underwent transthoracic echocardiography between May 2024 and June 2025. Participants with inadequate image quality for strain analysis were excluded. Examinations used a Philips EPIQ 7 with an X5-1 cardiac probe. In addition to conventional parameters, myocardial deformation imaging assessed left ventricular global longitudinal strain (LV GLS), right ventricular free wall strain (RV

Table 1. Comparison of echocardiographic parameters between pregnant women in gestational weeks 24–37 and healthy non-pregnant controls

Parameters	Pregnant (n=36)	Control (n=28)	p value
LVEDd (mm)	46.3 ± 5.8	42.4 ± 3.0	0.005*
LVESd (mm)	31.4 ± 5.4	26.9 ± 3.8	0.002*
IVSd (mm)	9.5 ± 1.0	7.9 ± 1.0	<0.001*
PWd (mm)	8.4 ± 1.1	6.8 ± 1.0	<0.001*
RWT	0.37 ± 0.07	0.31 ± 0.06	0.08
LVEF (%)	60.5 ± 5.0	64.7 ± 5.5	0.011*
LAd (mm)	32.9 (4–43)	31.2 (25–36)	0.354
E wave (m/s)	0.9 ± 0.1	1.0 ± 0.1	0.02*
A wave (m/s)	0.7 ± 0.2	0.7 ± 0.1	0.34
E/A ratio	1.3 ± 0.3	1.5 ± 0.3	0.002*
E/E' ratio	0.083 (0.05–0.14)	0.076 (0.06–0.1)	0.011*
RAd (mm)	33.1 (3.8–40)	33.5 (26–40)	0.83
RVd basal (mm)	32 (4.4–40)	33 ± 2.7	0.77
RV E wave (m/s)	0.7 ± 0.3	0.7 ± 0.1	0.97
RV A wave (m/s)	0.6 ± 0.3	0.5 ± 0.1	0.27
TR Vmax (m/s)	2.4 ± 0.4	2.1 ± 0.3	0.015*
TR pmax (mmHg)	20.9 ± 5	17.6 ± 4.5	0.031*
RV FWS (%)	-25.8 ± 6.6	-30.9 ± 4.2	0.002*
RV GLS (%)	-20.9 ± 3.2	-25.3 ± 3.4	<0.001*
LV GLS (%)	-19 ± 2.8	-24.8 ± 2.2	<0.001*
LASr 4CH (%)	41.6 (25.9–72.3)	53.1 (16.1–76.1)	0.01*
LASr 2CH (%)	39.1 (21.8–65.1)	50.7 (25.4–63.7)	0.03*
LAScd 4CH (%)	-24 (-55.9–-16.4)	-36 (-55.9–-11.6)	0.001*
LAScd 2CH (%)	-23.2 (-36.5–-14.3)	-33 (-50.6–-18.6)	<0.001*
LASct 4CH	-15.4 (-40,4–-4.1)	-14.8 (-24.2–-0.4)	0.76
LASct 2CH	-18 (-33.5–-7.4)	-14.7 (-29.9–-5.3)	0.36

Data are presented as mean ± standard deviation or median (minimum–maximum). Statistically significant differences (p<0.05); LVEDd: Left ventricular end-diastolic diameter; LVESd: Left ventricular end-systolic diameter; IVSd: Interventricular septal diameter; PWd: Posterior wall diameter; RWT: Relative wall thickness; LVEF: Left ventricular ejection fraction; LAd: Left atrial diameter; E/A: Early to late diastolic transmitral flow ratio; E': Early diastolic mitral annular velocity; RAd: Right atrial diameter; RVd: Right ventricular basal diameter; TR: Tricuspid regurgitation; Vmax: Maximum velocity; pmax: Maximumn pressure gradient; FWS: Free wall strain; GLS: Global longitudinal strain; LASr: Left atrial reservoir strain; LAScd: Left atrial conduit strain; LASct: Left atrial contraction strain; 4CH: Four-chamber view; 2CH: Two-chamber view.

FWS) and global longitudinal strain (RV GLS), as well as left atrial reservoir (LASr), conduit (LAScd), and contraction strain (LASct). Strain analysis was performed offline using vendor-specific software (QLAB, Philips). Statistical analysis applied Student's t-test or Mann–Whitney U test; p<0.05 was considered significant. The study had institutional ethics approval.

Results: Pregnant participants showed mild but significant increases in LV dimensions and wall thickness, consistent with volume overload adaptation, accompanied by a modest reduction in LVEF. These structural changes provide context for the observed alterations in myocardial deformation. Strain analysis revealed significantly lower LV GLS (-19.0% vs. -24.8%, p<0.001), RV FWS (-25.8% vs. -30.9%, p=0.002), RV GLS (-20.9% vs. -25.3%, p<0.001), as well as reduced left atrial reservoir strain (4CH: 41.6% vs. 53.1%, p=0.01; 2CH: 39.1% vs. 50.7%, p=0.03) and conduit strain (4CH: -24.0% vs. -36.0%, p=0.001; 2CH: -23.2% vs. -33.0%, p<0.001). Left atrial contraction strain did not differ significantly. A detailed overview is provided in Table 1.

Conclusions: This study provides normative myocardial deformation reference values for women between 24 and 37 weeks of gestation using automated QLAB software. Despite preserved conventional function, pregnancy induces significant strain alterations detectable by speckle-tracking echocardiography. These findings highlight the need for gestation-specific reference ranges to improve cardiovascular assessment in maternal care.

PP-004 [Cardiac Imaging / Echocardiography]

The predictive significance of the echocardiographic ratio of tricuspid annular plane systolic excursion to systolic pulmonary arterial pressure (TAPSE/sPAP) in systemic sclerosis

Ömer Kertmen[1], Abdulkadir Kara[2]

[1]Department of Cardiology, Amasya University Sabuncuoğlu Şerefeddin State Hospital, Amasya
[2]Department of Cardiology, Samsun Training and Research Hospital, Samsun

Background and Aim: Pulmonary hypertension (PH) is a complex pathophysiological condition that can manifest in various clinical contexts and exacerbate numerous systemic diseases. Among connective tissue diseases (CTDs), Systemic Sclerosis (SSc) is most frequently associated with pulmonary arterial hypertension (PAH). Although SSc is an uncommon disorder, it is linked to higher rates of morbidity and early mortality compared to other rheumatological diseases, primarily due to the development of interstitial pulmonary disease (ILD) and/or PAH. The importance of early diagnosis in improving prognosis is well-recognized. The tricuspid annular plane systolic excursion to systolic pulmonary arterial pressure (TAPSE/sPAP) ratio is an echocardiographic parameter used to assess the interaction between the right ventricle and the pulmonary artery In this study, we aim to investigate the early alterations in the pulmonary vascular bed by assessing the TAPSE/sPAP ratio in patients with SSc who do not present with overt PAH.

Methods: Forty five SSc patients who fulfilled the American College of Rheumatology criteria for diagnosis and forty five gender and age-matched, healthy subjects enrolled in this cross-sectional observational study. Exclusion criteria were; congenital heart disease, portal hypertension, HIV infection, coronary artery disease, structural heart disease or heart failure, heart valve disease (more than mild), chronic obstructive pulmonary disease (COPD), obstructive sleep apnea syndrome, other CTD or drugs which are involved in PH etiology, and chronic thromboembolic PH (CTePH). Echocardiographic parameters were assessed and compared to the control group. The results were evaluated within a 95% confidence interval. A p value <0.05 was considered to indicate statistical significance.

Results: The right-ventricular–pulmonary-artery coupling index—expressed as the TAPSE/sPAP ratio—was markedly lower than in healthy controls (1.18 ± 0.48 vs. 1.65 ± 0.61; p<0.001). This reduction reflects both diminished TAPSE (22.1 ± 4.4 mm vs. 24.4 ± 2.6 mm; p<0.001) and elevated systolic pulmonary artery pressure (26.4 ± 8.4 mmHg vs. 23.1 ± 5.5 mmHg; p<0.001), indicating early right-ventricular systolic dysfunction in the setting of increased pulmonary afterload.

Conclusions: The TAPSE/sPAP ratio, as an index of RV/PA coupling, is an affordable predictor of cardiovascular involvement and PAH in SSc. So may improve the prognostic stratification of SSc patients.

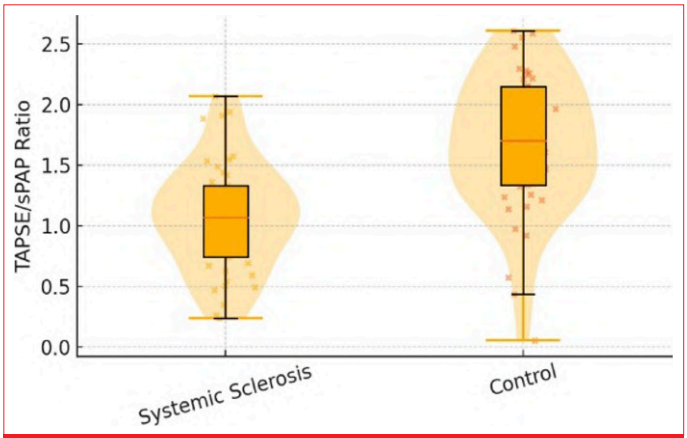

Figure 1.

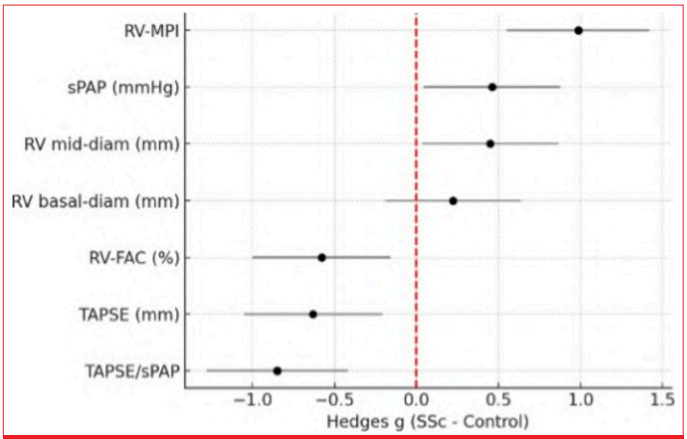

Figure 2.

Table 1. Echocardiographic characteristics of study population

Parameters	Scleroderma group (n=45)	Control group (n=45)	p value
Heart beats, bpm	70 ± 8	71 ± 6	0.475
LVEF, %	61.5 ± 1.4	61.7 ± 1.2	0.254
LVESD, mm	30.1 ± 1.7	31.1 ± 1.3	0.135
LVEDD, mm	44.2 ± 3.3	43.7 ± 3.4	0.118
LVSWT, mm	10.2 ± 0.7	10 ± 0.6	0.302
PWT, mm	9.2 ± 0.5	9 ± 0.6	0.136
LAD (a-p), mm	31.8 ± 4.7	30.8 ± 4.2	0.099
sPAP, mmHg	26.4 ± 4.4	23.1 ± 5.5	**<0.001**
E/e' (septal) ratio	9.1 ± 2.7	8.9 ± 2.6	0.041
RV mid-diameter, mm	27.1 ± 3.1	25.9 ± 2.1	**<0.001**
RV basal-diameter, mm	31.3 ± 3.3	30.6 ± 2.9	0.045
RV-FAC, %	40.4 ± 4.4	43.4 ± 5.8	**<0.001**
RV S' cm/s	11.3 ± 2.2	12.1 ± 2.3	0.008
Tissue doppler RV-MPI	0.50 ± 0.03	0.44 ± 0.08	**<0.001**
TAPSE, mm	22.1 ± 4.4	24.4 ± 2.6	**<0.001**
Tapse/sPAP	1,18 ± 0,48	1,65 ± 0,61	**<0.001**

PP-005 [Heart Failure]

Electrocardiographic markers predicting 1-year mortality in patients with heart failure with preserved ejection fraction: Insights from atrial and ventricular electrical abnormalities

Halit Emre Yalvaç[1], Bektaş Murat[1], Selda Murat[2], Ezgi Çamlı[1]

[1]*Eskişehir City Hospital, Eskişehir*
[2]*Department of Cardiology, Eskişehir Osmangazi University, Faculty of Medicine, Eskişehir*

Background and Aim: Heart failure with preserved ejection fraction (HFpEF) constitutes nearly half of all heart failure cases and carries substantial morbidity and mortality. Despite preserved left ventricular ejection fraction, HFpEF patients exhibit structural and electrical remodeling that predisposes to adverse outcomes. Electrocardiographic (ECG) indices reflecting atrial myopathy, ventricular depolarization, and repolarization heterogeneity may provide a simple and cost-effective means for risk stratification, yet data on their prognostic role in HFpEF are scarce. To determine the predictive value of selected ECG parameters for 1-year all-cause mortality in patients with HFpEF.

Methods: We retrospectively analyzed 158 consecutive patients diagnosed with HFpEF between January 2022 and December 2023. Baseline 12-lead ECG parameters included P-wave axis, Tp-e/QTcmax, and Tp-e/JTc. The primary endpoint was 1-year all-cause mortality. Variables with p<0.10 in univariate analysis were entered into a multivariable penalized logistic regression model with bootstrap resampling (n=1000) to estimate odds ratios (OR) and 95% confidence intervals (CI). Model discrimination was assessed using receiver operating characteristic (ROC) curve analysis.

Results: During follow-up, 6 patients (2.9%) died. Non-survivors had significantly higher P-wave axis values (median difference, p<0.001) and lower Tp-e/QTcmax ratios (p=0.007). In multivariable analysis excluding atrial fibrillation status, the following independent predictors emerged:

P-wave axis: OR 1.89 (95% CI: 1.65–2.19)

Tp-e/QTcmax: OR 0.91 (95% CI: 0.86–0.95)

Tp-e/JTc: OR 0.89 (95% CI: 0.82–0.97)

The final model demonstrated excellent discrimination with an AUC=0.892. At a probability cut-off of 0.5, sensitivity was 100% and specificity was 79%. ROC analysis for the model is shown in Figure 1. Patients stratified into the high-risk group had a markedly higher observed 1-year mortality compared with the low-risk group.

Conclusions: In HFpEF, ECG-derived markers of atrial abnormality (P-wave axis) and ventricular repolarization heterogeneity (Tp-e/QTcmax, Tp-e/JTc) independently predict 1-year all-cause mortality. A multivariable model incorporating these indices provides excellent prognostic discrimination and may guide early, targeted interventions. These findings underscore the utility of simple ECG measures for risk stratification in HFpEF, offering an inexpensive and widely available tool for clinical practice.

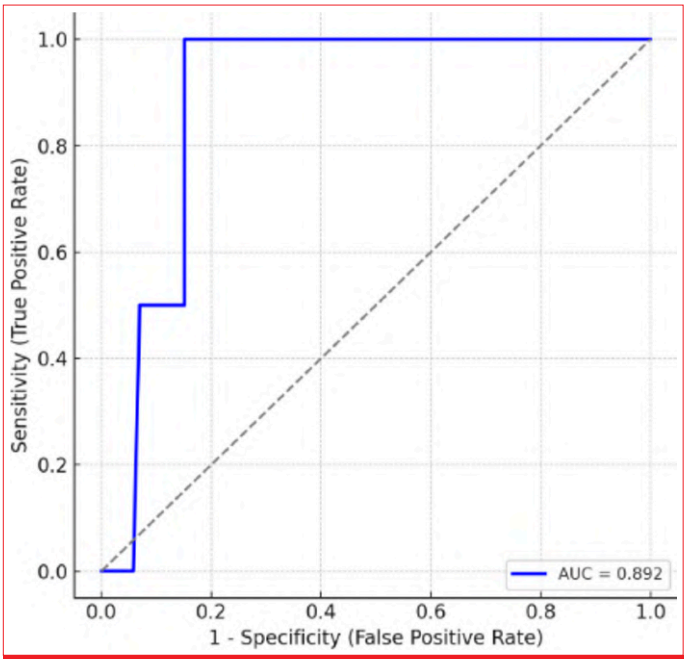

Figure 1. Prediction of 1-year mortality-ROC curve. Receiver operating characteristic (ROC) curve for the multivariable model excluding atrial fibrillation in predicting 1-year all-cause mortality among patients with heart failure with preserved ejection fraction (HFpEF). The model incorporates P-wave axis, Tp-e/QTcmax, and Tp-e/JTc as predictors, demonstrating excellent discrimination with an area under the curve (AUC) of 0.892.

Table 1. Univariate and multivariate logistic regression analysis for prediction of 1-year all-cause mortality in patients with HFpEF

Variable	Univariate OR (95% CI)	p	Multivariate OR (95% CI)	p
P-wave axis	7.49 (0.32–173.31)	0.209	1.89 (1.65–2.19)	<0.001
Tp-e/QTcmax	0.00 (0.00–344.77)	0.156	0.91 (0.86–0.95)	0.020
Tp-e/JTc	0.00 (0.00–86.41)	0.177	0.89 (0.82–0.97)	0.030

OR: Odds ratio; CI: Confidence interval; HFpEF: Heart failure with preserved ejection fraction; Tp-e–T-peak to T-end interval; QTc: Corrected QT interval; JTc: Corrected JT interval.

PP-006 [Cardiac Imaging / Echocardiography]

Uncovering PFO and ASD with coronary CT angiography: A single-center analysis

Ziya Gökalp Bilgel[1], Sefa Okar[1], Caner İncekaş[2]

[1]*Başkent University, Faculty of Medicine, Adana Research and Application Center, Adana*
[2]*Department of Biostatistics, Başkent University, Faculty of Medicine, Ankara*

Background and Aim: Patent foramen ovale (PFO) and atrial septal defect (ASD) are congenital cardiac anomalies strongly associated

with cryptogenic stroke. While transthoracic echocardiography (TTE) is the standard for prevalence estimation, coronary computed tomography angiography (CCTA) offers a potential for incidental detection during routine coronary evaluations. This study aimed to determine the prevalence of PFO and ASD in a patient cohort undergoing CCTA and compare these findings with TTE.

Methods: A retrospective analysis was performed on CCTA images from 1,128 consecutive patients (37.2% female, 62.8% male; mean age 53.95 ± 11.42 years) assessed for coronary artery disease between November 2022 and September 2024. Clinical profiles encompassed diabetes (19.0%), hypertension (45.7%), hyperlipidemia (18.0%), smoking history (42.6%), previous coronary artery disease (8.3%), and family history of cardiovascular disease (54.2%). PFO and ASD were identified using standardized CCTA protocols based on the classification framework by Williamson et al. All patients also underwent TTE. Prevalence rates and corresponding 95% confidence intervals (CI) were calculated for each modality.

Results: CCTA identified PFO in 51 patients (4.52%; 95% CI: 3.46–5.88%) and ASD in 9 patients (0.80%; 95% CI: 0.42–1.50%). Conversely, TTE detected PFO in 2 patients (0.18%; 95% CI: 0.05–0.64%) and ASD in 1 patient (0.09%; 95% CI: 0.02–0.50%).

Conclusions: CCTA demonstrated a significantly higher prevalence of PFO (4.52%) and ASD (0.80%) compared to TTE, a finding consistent with transesophageal echocardiography and autopsy data. These results suggest that CCTA may serve as a valuable supplementary imaging modality for identifying cardiac septal defects during coronary evaluations, potentially enhancing the detection of cryptogenic stroke risk. Further prospective research is recommended to validate these outcomes.

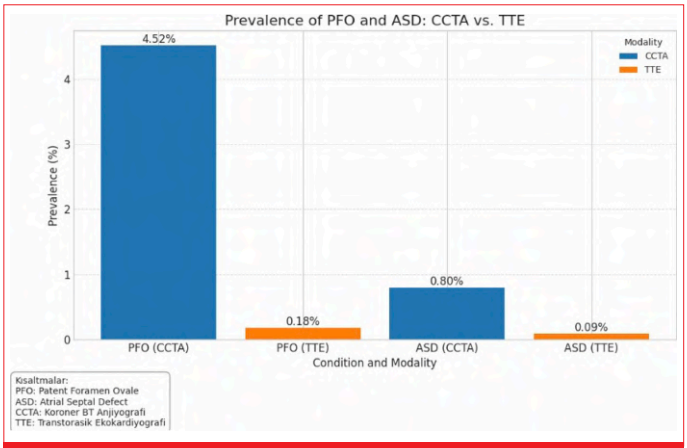

Figure 1. Comparison of PFO and ASD prevalence detected by CCTA and TTE. PFO and ASD Prevalence This figure presents a comparative analysis of the prevalence of Patent Foramen Ovale (PFO) and Atrial Septal Defect (ASD) using two different imaging modalities: Coronary CT Angiography (CCTA) and Transthoracic Echocardiography (TTE). The graph highlights that CCTA detected a substantially higher prevalence of PFO (4.52%) and ASD (0.80%) compared to TTE, which found prevalence rates of just 0.18% for PFO and 0.09% for ASD. This visual evidence supports the potential of CCTA as a more sensitive tool for identifying these cardiac septal defects, suggesting its utility as a valuable complementary method in clinical practice.

PP-007 [Cardiac Imaging / Echocardiography]

Assessment of left ventricular, right ventricular, and left atrial functions using strain echocardiography in patients with pseudoexfoliation syndrome

Mücahit Tan[1], Rumeysa Bilmez Tan[2], Yüksel Kaya[1], Muhammed Batur[2]

[1]Department of Cardiology, Yüzüncü Yıl University, Faculty of Medicine, Van
[2]Department of Ophthalmology, Yüzüncü Yıl University, Faculty of Medicine, Van

Background and Aim: Pseudoexfoliation syndrome (PEX) is a systemic disorder characterized by the abnormal production of fibrillar material in the extracellular matrix and its progressive accumulation in various organs, primarily in ocular tissues but also including the myocardium. Strain echocardiography, a quantitative imaging modality that has gained increasing clinical use in recent years, enables the early detection of subclinical myocardial dysfunction. The present study aimed to evaluate potential subclinical right and left ventricular dysfunction as well as impairment of left atrial functions in patients with PEX using strain echocardiography.

Methods: This cross-sectional case-control study included 27 asymptomatic patients with pseudoexfoliation syndrome (PEX) without known cardiac disease and 27 healthy volunteers as the control group. All participants underwent comprehensive echocardiographic examinations, including left ventricular, right ventricular, and left atrial strain analyses.

Results: Among standard echocardiographic parameters, there were significant differences between the two groups in terms of lateral e' (0.07 ± 0.02 vs. 0.09 ± 0.02, p=0.002), mean e' (0.06 ± 0.01 vs. 0.08 ± 0.02, p=0.001), E/A ratio (0.68 ± 0.13 vs. 0.84 ± 0.27, p=0.012), and deceleration time (193.07 ± 45.70 vs. 144.23 ± 47.56, p=0.001). No significant difference was observed in global longitudinal strain (GLS) values between the PEX and control groups (−23.12 ± 2.11 vs. −20.49 ± 9.41, p=0.163). Similarly, no significant differences were found in right ventricular free-wall longitudinal strain (RVFWSL) and right ventricular four-chamber strain (RV4CSL) values (p=0.149 and p=0.460, respectively). In the evaluation of left atrial strain parameters (LASr, LAScd, LASct), a statistically significant difference was detected in left atrial reservoir strain between the PEX and control groups, whereas no significant differences were found in left atrial conduit strain (LAScd) and left atrial contractile strain (LASct) (p=0.068 and p=0.284, respectively).

Conclusions: In pseudoexfoliation (PEX) syndrome, where standard echocardiographic techniques may remain inconclusive, subclinical atrial involvement can be detected using strain echocardiography. PEX is associated with impairment of left atrial deformation parameters. Therefore, systemic involvement should be considered in patients with PEX, and these patients should be monitored for cardiac dysfunction and arrhythmias.

Table 1. Characteristics of the PEX and control groups

	PEX (n=27)	Control (n=27)	p
Age (years)	70.74 (7.5)	58.96 (5.8)	0.001
Female n (%)	15 (55.6)	13 (48.1)	0.586
DM n (%)	5 (18.5)	8 (29.6)	0.340
HT n (%)	9 (33.3)	10 (37)	0.776
HR (beat/min)	76 (11)	78 (12)	0.384
SBP (mmHg)	131 (15)	125 (26)	0.339
DBP (mmHg)	76 (8)	84 (9)	0.001
WBC (10^3/uL)	7.01 (2.4)	7.29 (1.3)	0.996
Hemoglobin (g/dL)	14.31 (1.5)	15.1 (1.4)	0.446
Platelet (10^3/uL)	268.96 (110)	277.14 (78.5)	0.771
Albumin (Alb, g/dL)	39.8 (3.4)	43.84 (2.4)	0.001
Trigliseride (mg/dL)	210.65 (174.2)	181.68 (190.8)	0.597
Total Cholesterol (mg/dL)	210.57 (47.4)	218.68 (45.6)	0.567
LDL (mg/dL)	125.13 (41.1)	133.68 (38.2)	0.475
HDL (mg/dL)	46.36 (11.8)	49.60 (15.4)	0.433
HbA1C (%)	6.25 (1.9)	6.73 (1.9)	0.426
Sodium (Na, mmol/L)	139.00 (2.7)	138.36 (2.2)	0.395
Potassium (K, mmol/L)	4.2 (0.3)	4.3 (0.3)	0.013
Creatinine (mg/dL)	0.81 (0.1)	0.84 (0.1)	0.645
eGFR (ml/m^2/min)	82.44 (12.1)	89.05 (11.4)	0.063
CRP (mg/L)	5.83 (10.9)	5.11 (4.6)	0.777
TSH (mUI/mL)	1.66 (1)	1.75 (0.8)	0.754

DM: Diabetes mellitus, HT: Hypertansion, HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, WBC: White blood count, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, HbA1C: Hemoglobin A1C, eGFR: Estimated glomerular filtration rate, CRP: C-reactive protein, Alb: Albumin, TSH: Thyroid stimulating hormone. Bold values indicate

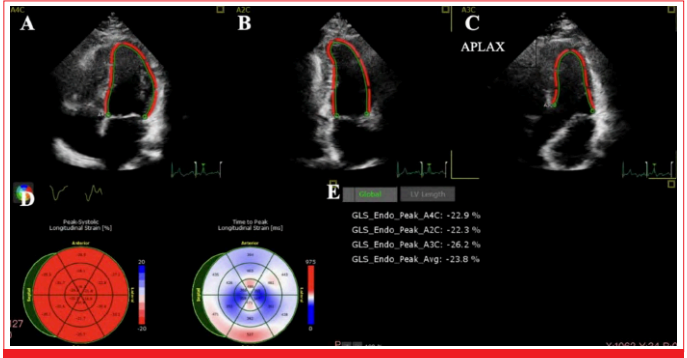

Figure 1. Left ventricle global longitudinal strain (LV GLS) measurement. (A) Left ventricular focused apical four-chamber, (B) two-chamber, (C) long axis (APLAX) view, (D) Left ventricular bullseye plots; peak systolic longitudinal strain and time to peak longitudinal strain, (E) Measured left ventricle strain values.

Table 2. Echocardiographic findings of the PEX and control groups

	PEX Mean (SD)	Control Mean (SD)	p
LVEF (%)	61.52 (2.80)	61.23 (2.86)	0.722
LVEDD (cm)	4.50 (0.42)	4.59 (0.36)	0.455
LVESD (cm)	3.22 (0.45)	3.19 (0.46)	0.822
IVS (cm)	0.96 (0.18)	1.01 (0.15)	0.312
PW (cm)	0.82 (0.15)	0.87 (0.15)	0.240
LA diameter (cm)	3.57 (0.39)	3.47 (0.25)	0.563
RV diameter (cm)	3.21 (0.39)	3.22 (0.53)	0.920
LA min. volume (mL)	16.21 (8.48)	12.76 (4.88)	0.097
LA max. volume (mL)	37.66 (14.58)	34.85 (12.72)	0.481
E (m/s)	0.55 (0.11)	0.61 (0.18)	0.137
A (m/s)	0.79 (0.13)	0.75 (0.12)	0.233
S' lateral (m/s)	0.08 (0.01)	0.08 (0.01)	0.553
E' lateral (m/s)	0.07 (0.02)	0.09 (0.02)	0.002
A' lateral (m/s)	0.14 (0.17)	0.11 (0.02)	0.427
E' mean	0.06 (0.01)	0.08 (0.02)	0.001
E/E'	8.76 (2.02)	7.64 (1.98)	0.059
E/A	0.68 (0.13)	0.84 (0.27)	0.012
IVRT	93.78 (14.10)	89.05 (14.76)	0.258
DT	193.07 (45.7)	144.23 (47.56)	0.001

SD: Standard deviation; LVEF: Left ventricular ejection fraction; LVEDD: Left ventricle end-diastolic diameter; LVESD: Left ventricle end-systolic diameter; RV: Right ventricle; PW: Posterior wall; LA: Left atrium; RV: Right ventricle; E: Mitral E-wave velocity; A: Mitral A-wave velocity; S': Mitral annulus positive systolic wave; E': Early diastolic mitral annular wave; IVRT: Isovolumic relaxation time; DT: E-wave deceleration time.

Table 3. Strain echocardiographic findings of the PEX and control groups

	PEX Mean (SD)	Control Mean (SD)	p
LV GLS	-23.12 (2.11)	-20.49 (9.41)	0.163
LASr	34.51 (4.99)	41.29 (6.37)	0.001
LAScd	-15.65 (5.15)	-19.56 (9.56)	0.068
LASct	-18.89 (4.99)	-20.74 (7.36)	0.284
RVFWSL	-28.78 (2.80)	-29.94 (3.00)	0.149
RV4CSL	-24.24 (2.60)	-24.78 (2.80)	0.460

LV GLS: Left ventricle global longitudinal strain; LASr: Left atrium reservoir strain; LAScd: Left atrium conduit strain; LASct: Left atrium contractile strain; RVFWSL: Right ventricular free-wall longitudinal strain; RV4CSL: Right ventricular four-chamber strain.

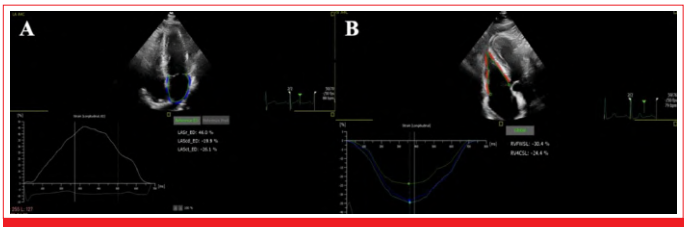

Figure 2. (A) Left atrium strain measurement (LASr, LAScd, LASct), (B) Right ventricular strain measurement (RVFWSL, RV4CSL).

PP-008 [Epidemiology]

Physical activity and sexual function after myocardial infarction: A cross-sectional survey from Türkiye

Şahhan Kılıç[1], Süha Asal[1], Fahri Çakan[1], Büşra Zehra Büyükkılıç[2], Mert Babaoğlu[3], Süleyman Kürşad Özel[4], Samet Yavuz[3], Mehmet Şeker[5], Murat Selçuk[4], Tufan Çınar[6], Mehmet Uzun[3]

[1]*Department of Cardiology, Çorlu State Hospital, Tekirdağ*
[2]*Department of Internal Medicine Nursing, Health Sciences University, School of Nursing, İstanbul*
[3]*Department of Cardiology, Sultan Abdülhamid Han Hospital, İstanbul*
[4]*Department of Cardiology, Sancaktepe Şehit Prof. Dr. İlhan Varank Training and Research Hospital, İstanbul*
[5]*Department of Cardiology, Ümraniye Education and Research Hospital, İstanbul*
[6]*Department of Medicine, University of Maryland Midtown Campus, USA*

Background and Aim: Post-myocardial infarction (MI) recovery encompasses both physical activity and sexual health, yet these domains are often overlooked during routine follow-up, particularly in culturally conservative societies. This study aimed to evaluate the levels of physical activity and sexual function in patients after MI in Türkiye, assess the quality of lifestyle counseling at discharge, and identify factors influencing the successful resumption of these activities.

Methods: In this cross-sectional survey, 204 patients with a history of MI attending follow-up at three tertiary hospitals in Istanbul between November 2024 and May 2025 were enrolled. Participants completed validated questionnaires on physical activity, sexual function, medication adherence, psychological concerns, and counseling experiences. Clinical data, including left ventricular ejection fraction and treatment modalities, were also recorded. Statistical analysis included descriptive statistics and correlation testing.

Results: The median age was 57 years; 85.3% of participants were male. Only 46.1% reported receiving comprehensive counseling at discharge. The median time to resume sexual activity was 34 days, with 43.1% reporting infrequent sexual encounters (≤3 times/month). Erectile dysfunction affected 29.3% of men. Physical activity levels declined post-MI in 46.6% of patients (Table 1), despite a median walking time of 55 minutes per day. Physician-initiated discussions on sexual health were reported by only 7.4%. Counseling quality correlated positively with earlier sexual resumption and improved exercise tolerance (Table 2, 3).

Conclusions: There is a substantial gap between clinical guidelines and real-world practice in addressing physical activity and sexual function after MI in Türkiye. Culturally sensitive rehabilitation programs and structured counseling are urgently needed.

Table 1. Likert classification of patients answers to lifestyle questions (categorical variables are reported in percentages)

Questions	Completely disagree	Partially disagree	Undecided	Partially agree	Completely agree
I did not miss my follow-up appointments.	6 (%2,9)	11 (%5,4)	9 (%4,4)	30 (%14,7)	146 (%71,6)
I took my medication regularly.	3 (%1,5)	4 (%2)	8 (%3,9)	33 (%16,2)	156 (%76,5)
I have concerns about my medications.	119 (%58,3)	19 (%9,3)	21 (%10,3)	35 (%17,2)	10 (%4,9)
I think my cholesterol lowering medications are beneficial for me.	11 (%5,4)	29 (%14,2)	38 (%18,6)	46 (%22,5)	80 (%39,2)
I can continue my physical activity at the same level it was before the heart attack.	21 (%10,3)	28 (%13,7)	26 (%12,7)	34 (%16,7)	95 (%46,6)
I can continue sexual activity at the same level it was before the heart attack.	44 (%21,6)	47 (%23)	23 (%11,3)	24 (%11,8)	65 (%31,9)
I had reservations about having sex after the heart attack.	100 (%49)	15 (%7,4)	11 (%5,4)	41 (%20,1)	36 (%17,6)
I think my cardiac medications are affecting my sexual performance.	104 (%51)	26 (%12,7)	24 (%11,8)	23 (%11,3)	26 (%12,7)
I had reservations about having strenuous exercise (soccer, fitness, martial arts, swimming, weightlifting etc.) after the heart attack.	35 (%17,2)	29 (%14,2)	47 (%23)	33 (%16,2)	60 (%29,4)
I realized the value of my health after the heart attack.	6 (%2,9)	7 (%3,4)	13 (%6,4)	24 (%11,8)	153 (%75)
I am following a healthier diet after the heart attack.	13 (%6,4)	14 (%6,9)	27 (%13,2)	57 (%27,9)	93 (%45,6)
I can have another heart attack.	28 (%13,7)	23 (%11,3)	37 (%18,1)	42 (%20,6)	73 (%35,8)
My desire to continue my career did not decrease after the heart attack.	30 (%14,7)	27 (%13,2)	22 (%10,8)	16 (%7,8)	109 (%53,4)
I am not worried about driving after the heart attack.	13 (%6,4)	17 (%8,3)	35 (%17,2)	8 (%3,9)	128 (%62,7)
I am not worried about being alone in a place after the heart attack.	17 (%8,3)	15 (%7,4)	12 (%5,9)	5 (%2,5)	155 (%76)
I am not worried about boarding a boat/plane/train after the heart attack.	11 (%5,4)	14 (%6,9)	14 (%6,9)	7 (%3,4)	158 (%77,5)
I am confident I can engage in extreme sports after the heart attack (parachute, hiking, rowing, martial arts etc.).	103 (%50,5)	21 (%10,3)	28 (%13,7)	13 (%6,4)	38 (%18,6)

Table 2. Answers to questions about physical and sexual activity and counseling (categorical variables are reported in percentages, numerical variables are reported as median [interquartile range])

Questions	
How long do you take walks in a day? (minutes)	55 (30-90)
How soon after the heart attack did you first have sex? (days)	34 (10-60)
How often do you have sex in a week?	
Once a day or more	3 (%1,5)
3-6 times a week	22 (%10,8)
1-2 times a week	91 (%44,6)
2-3 times a month or less	88 (%43,1)
Did you have any problems in your sex life after the heart attack? If yes, which one of the following?	
I did not have any problems.	56 (%27,5)
I have low sexual drive.	50 (%24,5)
I did not climax.	1 (%0,5)
(Men only) I climaxed early.	21 (%12,1)
I had physical pain.	2 (%1)
I had shortness of breath.	4 (%2)
I had chest pain.	3 (%1,5)
I did not enjoy sex.	6 (%2,9)
I was worried about my sexual performance.	8 (%3,9)
(Men only) I had problems with erection.	51 (%29,3)
(Women only) I had problems with lubrication.	1 (%3,3)
How did you obtain information about sex life after the heart attack?	
My doctor	30 (%14,7)
From television	2 (%1)
From the internet	14 (%6,9)
From my social circle	9 (%4,4)
I did not obtain any information.	149 (%73)
(Men only) Did you ever take any sexual performance enhancing medications after the heart attack?	27 (%15,5)
Who initiated the conversation about sex life during your follow-up visit?	
My doctor	15 (%7,4)
Myself	25 (%12,3)
We both did	1 (%0,5)
No one did	163 (%79,9)
What did the doctor recommend?	
Recommended restrictions.	14 (%6,9)
Recommended to aim for a lower heart rate during sex.	0 (%0)
Recommended a more passive position.	1 (%0,5)
Recommended medication for enhancing sexual performance.	8 (%3,9)
Recommended relaxing exercises.	3 (%1,5)
Recommended limiting the times of sex I have.	2 (%1)
Did not recommend any restrictions.	14 (%6,9)
This topic was not discussed.	158 (%77,5)
Were you satisfied by your doctor's recommendations on your sexual life?	
Not at all satisfied.	5 (%2,9)
Partially satisfied.	25 (%2,5)
Mostly satisfied.	9 (%4,4)
Completely satisfied.	6 (%2,9)
This topic was not discussed.	159 (%77,9)

PP-009 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

SGLT2 inhibitors may improve survival in high ventricular-pacing CIED patients with preserved ejection fraction: A retrospective cohort study

Hidayet Ozan Arabacı, Şükrü Arslan, Muhammed Heja Geçit, Mustafa Yıldız

Department of Cardiology, İstanbul University Cerrahpaşa, Cardiology Institute, İstanbul

Background and Aim: Initially developed as antidiabetic agents, SGLT2 inhibitors have become essential in cardiovascular medicine due to their proven benefits in reducing morbidity and mortality in heart failure, regardless of ejection fraction. Beyond glycemic control, these agents have demonstrated antiarrhythmic properties, including reductions in sudden cardiac death, atrial arrhythmias, and atrial fibrillation. However, evidence remains limited regarding their effect in patients with cardiac implantable electronic devices (CIEDs), particularly those with high ventricular pacing and preserved EF. This study aimed to evaluate the impact of SGLT2 inhibitor therapy on all-cause mortality in this specific population.

Methods: This retrospective, observational single-center cohort study included patients who underwent CIED implantation between January 2018 and June 2024. Of 2176 screened individuals, 540 patients were eligible: aged >18 years, with preserved LVEF (≥50%), and CIED implanted for conduction defects (AV blocks, non-AV block conduction defects [bifasicular and trifasicular blocks], or slow ventricular response AF). Exclusion criteria included reduced LVEF, congenital AV block, myocarditis-related conduction defects, sinus node dysfunction, or incomplete data. Demographic, clinical, ECG, echocardiographic, laboratory, and medication data were collected from institutional records. Associations between SGLT2i use and all-cause mortality were analyzed using multivariate Cox regression and Kaplan-Meier survival curves.

Results: In this retrospective cohort of 540 patients with preserved ejection fraction who underwent CIED implantation due to conduction system disorders, the mean follow-up duration was 59.2 months, during which all-cause mortality occurred in 34.3% of patients. Patients with mortality were significantly older and had higher rates of comorbidities including diabetes, hypertension, stroke, chronic kidney disease, and atrial fibrillation. They were more likely to have received VR pacemakers or ICDs, whereas those implanted with DR devices demonstrated more favorable survival outcomes. Multivariate Cox regression identified advanced age, heart failure, atrial fibrillation, lower GFR, greater IVS thickness, and elevated NT-proBNP levels as independent predictors of all-cause mortality. Importantly, SGLT2 inhibitor use emerged as the only independent variable associated with a significant reduction in mortality, suggesting a potential cardioprotective effect in this population.

Conclusions: In patients with cardiac conduction defects, preserved EF, and high ventricular pacing rates implanted with CIEDs, SGLT2 inhibitor use significantly reduced all-cause mortality. Although limited, existing evidence suggests SGLT2 inhibitors may mitigate negative outcomes linked to ventricular pacing-induced dyssynchrony. Prospective, large-scale studies are needed to confirm these findings.

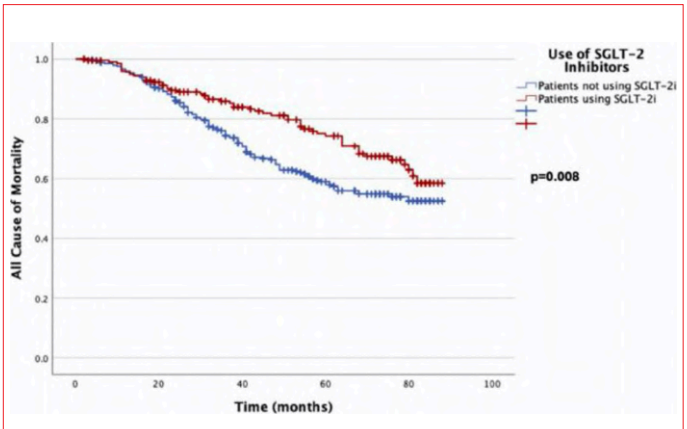

Figure 1. Kaplan-Meier curves demonstrating the impact of SGLT-2 inhibitors on survival outcomes.

Table 1. Clinical and demographic characteristics of patients with all-cause mortality

	All Cause of Mortality (-) (n: 355)	All Cause of Mortality (+) (n: 185)	P value
Age, (year)*	76 (24-97)	86 (60-102)	**<0.001**
Gender, n (%)			
• Female	195 (54.9)	97 (52.4)	0.581
Indications for Implantation, n (%)			
• AV Blocks#	224 (63.1)	116 (63.7)	
• Bundle Branch Conduction Defects## (out of AV block)	43 (12.1)	19 (10.3)	0.770
• AF with Slow Ventricular Rate	88 (24.8)	50 (27.0)	
Types of Implanted CIEDs, n (%)			
• VR pacemaker	101 (28.5)	81 (43.8)	
• DR pacemaker	214 (60.3)	82 (44.3)	**<0.001**
• VR-ICD	20 (5.6)	18 (9.7)	
• DR-ICD	20 (5.6)	4 (2.2)	
Heart Failure; n (%)			
• Patients without HF	152 (42.8)	48 (25.0)	**0.003**
• Patients with HF and EF %50 <	203 (57.2)	137 (75.0)	
DM, n (%)	121 (34.1)	82 (44.3)	**0.020**
HT, n (%)	285 (80.3)	166 (89.2)	**0.005**
Stroke, n (%)	83 (23.4)	61 (33.0)	**0.017**
CAD**, n (%)	91 (25.6)	59 (31.9)	0.123
CKD***, n (%)	76 (21.4)	85 (45.9)	**<0.001**
AF, n (%)	152 (42.8)	118 (63.8)	**<0.001**
Senkop-presenkop, n (%)	275 (77.5)	128 (69.2)	**0.016**
Smoker, n (%)	68 (19.2)	28 (15.1)	0.246
Use of Medical Treatment; n (%)			
• Beta Blocker	244 (68.7)	139 (75.1)	0.121
• Non-DHP Calcium Channel Blocker	65 (18.3)	47 (25.4)	0.054
• Amiodarone	20 (5.6)	21 (11.4)	**0.017**
• Class I Anti-Arythmic Drugs	11 (3.0)	7 (3.8)	0.715
• Digoxin	65 (18.3)	49 (26.5)	**0.027**
• ACE-i / ARB	300 (84.5)	163 (88.1)	0.256
• SGLT-2 inhibitor	151 (42.5)	55 (29.7)	**0.004**
• ARNI	8 (2.3)	4 (2.2)	0.946
• MRA	96 (27.0)	91 (49.2)	**<0.001**
• Diuretic	285 (80.3)	178 (90.2)	**<0.001**
• Oral Anticoagulant Drugs	249 (70.1)	152 (82.2)	**0.002**

ACE-i: angiotensin-converting enzyme inhibitor, ARB: angiotensin receptor blocker, AF: atrial fibrillation, ARNI: angiotensin receptor/neprilysin inhibitor, AV: atrioventricular, CAD: coronary artery disease, CIED: cardiac implantable electronic device, CKD: chronic kidney disease, DM: diabetes mellitus, DR: atrial and ventricular double leads, HF: heart failure, HT: hypertension, ICD: implantable cardioverter-defibrillator, MRA: aldosterone receptor antagonist, Non-DHP: non dihydropyridine calcium channel blocker, SGLT-2: sodium-glucose cotransporter-2, VR: ventricular single lead.
*Median(Min-Max)
**CAD was defined as the presence of significant coronary artery stenosis of 50% or more on invasive coronary angiography, percutaneous coronary intervention and/or coronary artery by-pass surgery.
***CKD is defined as the presence of glomerular filtration rate (GFR < 60 ml/min).
AV Blocks; 2:1 AV block and above, high grade AV block, AV complete block are defined as the general indication for all patients with intracardiac device implantation.
Bundle Branch Conduction Defects (out of AV block): It is defined as the general indication of all patients implanted with intracardiac device with the indications of bifascicular block and symptomatic bradycardia, trifascicular block and symptomatic bradycardia.

Table 2. Electrocardiographic and echocardiographic characteristics of patients with all-cause mortality

	All Cause of Mortality (-) (n: 355)	All Cause of Mortality (+) (n: 185)	P value
PR Interval, (msn)	200 (106-400)	210 (138-340)	0.484
QRS Segment, (msn)	132 (65-240)	140 (81-180)	**0.001**
QTc Interval, (msn)	435 (328-536)	440 (370-535)	**0.018**
AV Complete Blocks, n (%)	115 (32.4)	70 (37.7)	0.092
High Degree AV Blocks#, n (%)	54 (15.2)	32 (17.3)	0.149
LVEF, (%)*	60 (50-65)	60 (50-64)	0.389
LA, mm*	44 (29-60)	45 (32-62)	**0.001**
LVEDd, mm*	50 (37-59)	51 (36-60)	0.169
IVS, mm*	12 (7-18)	12 (8-20)	**0.009**
PW, mm*	11 (6-17)	11 (8-19)	0.062
TAPSE, mm*	20 (10-27)	20 (14-28)	0.349
sPAP, mmHg*	35 (20-100)	38 (22-100)	**0.003**

AV: atrioventricular, IVS: interventricular septum, ICD: implantable cardioverter-defibrillator, LA: left atrium, LVEDd: left ventricular end-diastolic diameter, LVEF: left ventricular ejection fraction, LVEDd; left ventricular end-diastolic diameter,
PW: posterior wall of left ventricule sPAP: systolic pulmonary arterial pressure, TAPSE: tricuspid annular plane systolic excursion,
*Median(Min-Max)
#High Degree AV Blocks are defined as AV blocks with cardiac conduction defects above 2:1 AV block (e.g. 3:1, 4:1 AV blocks) but AV complete block is not detected on electrocardiograms.

Table 3. Laboratory and biochemical characteristics of patients with all-cause mortality

	All Cause of Mortality (-) (n: 355)	All Cause of Mortality (+) (n: 185)	P value
Hb, g/dl*	13.2 (6.5-19.0)	12.2 (5.2-17.0)	**<0.001**
HCT, (%)*	38.3 (19.6-56.0)	36.3 (16.2-50.0)	**<0.001**
PLT, 10^3 mm ^{3}*	221.0 (50.2-535.0)	211.5 (42.0-676.0)	0.067
WBC, 10^3 mm ^{3}*	7.2 (3.2-14.7)	7.6 (2.3-15.6)	0.179
NEUT, 10^3 mm ^{3}*	4.3 (0.3-11.4)	4.8 (1.5-10.8)	**0.001**
Lymphocyte, 10^3 mm ^{3}*	1.8 (0.3-4.5)	1.5 (0.2-4.2)	**<0.001**
Monocyte, 10^3 mm ^{3}*	0.6 (0.1-2.0)	0.6 (0.1-1.3)	**0.012**
Creatinine, mg/dl*	0.9 (0.5-5.6)	1.1 (0.5-8.8)	**<0.001**
GFR, ml/min*	67.1 (12.0-128.2)	51.5 (8.2-120.0)	**<0.001**
Na (Sodium), mg/dl*	140.1 (127.5-152.0)	141.0 (126.3-147.7)	0.898
K (Potassium), mg/dl*	4.5 (3.3-5.7)	4.5 (3.0-5.9)	0.974
LDL, mg/dl*	108.0 (15.0-251.0)	112.0 (46.0-216.0)	0.315
Troponin T, ng/ mL*	0.012 (0.001-0.340)	0.016 (0.001-0.430)	**<0.001**
NT-pro BNP, pg/ml*	167.7 (3.5-6989.4)	425.0 (56.5-18877.0)	**<0.001**
CRP, mg/dl*	3.9 (0.1-75.0)	7.8 (0.3-86.0)	**<0.001**

CRP: c reactive protein, GFR: glomerular filtration rate, Hb: haemoglobin, HCT: haemotocrit, LDL: low density lipoprotein, NEUT: neutrophil, NT-pro BNP: N-terminal protein brain natriuretic peptide, PLT: platelet, WBC: white blood cell,
*Median(Min-Max)

Table 4. Multivariate Cox regression model independently predicting mortality development attributable to all causes

All Cause of Mortality	Hazard Ratio (HR)	Lower – Upper (95% Confidence Interval (CI))	P Value
Age	1.05	1.03-1.07	**<0.001**
HF	2.12	1.12-5.65	**0.029**
DM	1.09	0.80-1.50	0.561
HT	0.65	0.38-1.09	0.110
CKD	0.97	0.86-1.38	0.891
AF	1.41	1.09-1.98	**0.044**
AV Complete Block	1.32	0.97-1.80	0.077
LVEDd	1.00	0.97-1.46	0.669
IVS	1.16	1.05-1.28	**0.004**
TAPSE	0.99	0.93-1.05	0.782
sPAP	1.00	0.98-1.02	0.618
Hb	0.94	0.86-1.03	0.229
WBC	1.00	0.99-1.01	0.151
GFR	0.98	0.97-0.99	**0.004**
Troponin	0.48	0.04-1.42	0.074
NT-proBNP	1.00	1.00-1.00	**<0.001**
LDL	1.00	0.99-1.01	0.281
CRP	1.01	1.01-1.12	**0.045**
Beta Blocker	1.14	0.78-1.65	0.488
ACE-I/ARB	1.01	0.62-1.64	0.957
SGLT-2i	0.50	0.37-0.69	**<0.001**

ACE-i: angiotensin-converting enzyme inhibitor, ARB: angiotensin receptor blocker, AF: atrial fibrillation, AV: atrioventricular, CKD: chronic kidney disease, CRP: c reactive protein, DM: diabetes mellitus, GFR: glomerular filtration rate, Hb: haemoglobin, HF: heart failure, HT: hypertension, IVS: interventricular septum, LDL: low density lipoprotein, LVEDd: left ventricular end-diastolic diameter, NT-pro BNP: N-terminal protein brain natriuretic peptide, SGLT-2: sodium-glucose cotransporter-2, sPAP: systolic pulmonary arterial pressure, TAPSE: tricuspid annular plane systolic excursion, WBC: white blood cells.

PP-010 [Coronary Artery Disease / Acute Coronary Syndrome]

Prediction of major adverse cardiovascular events using DANCAMI in patients with acute myocardial infarction

Bektaş Murat[1], Selda Murat[2], Esra Can Önder[2]

[1]Department of Cardiology, Eskişehir Osmangazi University, Faculty of Medicine, Eskişehir
[2]Eskişehir City Hospital, Eskişehir

Background and Aim: Major adverse cardiovascular events (MACE) following acute myocardial infarction (AMI) are associated with poor prognosis and increased mortality. The comorbidity burden plays a critical role in determining long-term outcomes. The Danish Comorbidity Index for Acute Myocardial Infarction (DANCAMI) was designed as a mortality prediction tool for AMI patients. This study

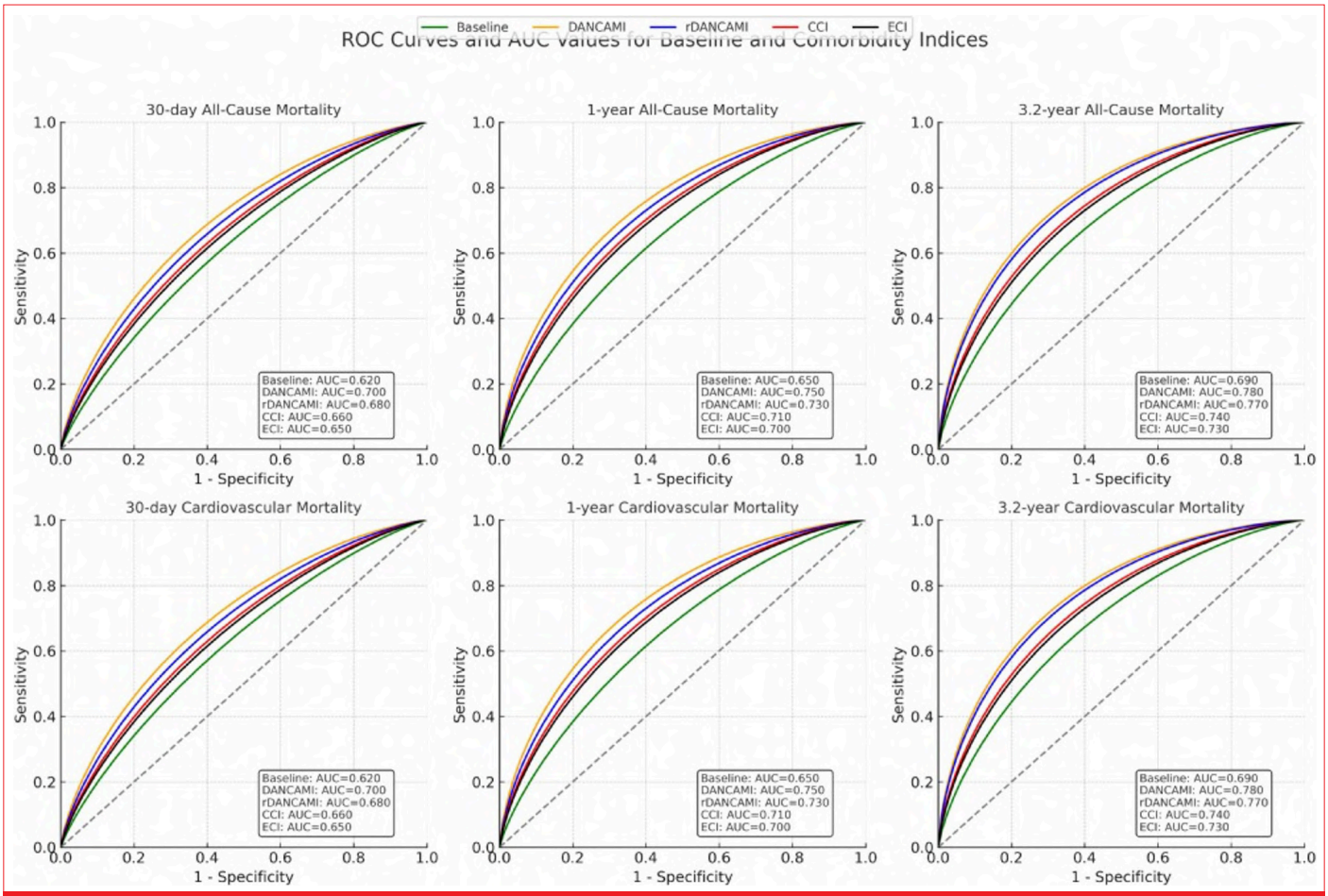

Figure 1. ROC analysis of comorbidity inces for mortality prediction

evaluated the predictive performance of DANCAMI and compared it with other widely used indices, including the revised DANCAMI (rDANCAMI), Charlson Comorbidity Index (CCI), and Elixhauser Comorbidity Index (ECI).

Methods: We performed a retrospective analysis of 851 patients diagnosed with ST-segment elevation myocardial infarction (STEMI) from two centers. The mean follow-up duration was 39.9 months. Demographic characteristics, comorbidity profiles, and MACE occurrence were recorded. For each patient, DANCAMI, rDANCAMI, CCI, and ECI scores were calculated. The discriminatory ability of each score for predicting MACE was analyzed using receiver operating characteristic (ROC) curves and area under the curve (AUC) metrics.

Results: Of the study population, 34.9% were classified as low, 53.9% as moderate, 10% severe and 1.0% as very severe according to DANCAMI scores (Table 1). The most frequent comorbidities were hypertension (58.1%), diabetes mellitus (43.2%), and atrial fibrillation (25.7%). Cumulative incidence curves indicated a stepwise increase in MACE risk with higher comorbidity burden. ROC analysis demonstrated that DANCAMI outperformed rDANCAMI, CCI, and ECI in discriminating patients at risk of all-cause mortality and cardiovascular mortality (Figure 1).

Conclusions: DANCAMI and other comorbidity indices effectively predict MACE in STEMI patients. Among these, DANCAMI exhibited superior discriminatory power for all-cause and cardiovascular mortality. These findings emphasize the prognostic significance of

Table 1. Demographic and clinical characteristics of STEMI patients

Characteristic	Number (%)
Total patients	851 (100%)
Female sex	204 (23.96%)
Age (median, IQR)	63.9 ± 10.8
DANCAMI category - Low	297 (34.9%)
DANCAMI category - Moderate	459 (53.9%)
DANCAMI category - Severe	85 (10.0%)
DANCAMI category - Very Severe	8 (1%)
Charlson category - Low	463 (54.4%)
Charlson category - Moderate	229 (26.9%)
Charlson category - Severe	93 (10.9%)
Charlson category - Very Severe	66 (7.8%)
Elixhauser category - Low	509 (59.8%)
Elixhauser category - Moderate	238 (28.0%)
Elixhauser category - Severe	82 (9.6%)
Elixhauser category - Very Severe	22 (2.6%)
Hypertension	406 (47.7%)
Diabetes	332 (39.0%)
Coronary artery disease	198 (23.3%)
Atrial fibrillation	19 (2.2%)

comorbidity assessment in AMI and highlight DANCAMI's clinical utility as a risk stratification tool. While the retrospective design poses inherent limitations, the results provide a robust foundation for prospective studies exploring its application in diverse patient populations.

PP-011 [Heart Failure]

Investigation of the effects of SGLT-2 inhibitor use on echocardiographic parameters in patients with heart failure with preserved ejection fraction

Selen Cansu Altun, Nil Özyüncü, Müge Akbulut, Türkan Seda Tan

Department of Cardiology, Ankara University, Faculty of Medicine, Ankara

Background and Aim: Heart failure with preserved ejection fraction is a growing clinical challenge due to its increasing prevalence and limited treatment options. SGLT-2 inhibitors represent the first pharmacological class shown to improve cardiovascular outcomes in this patient population. However, data on their short-term effects, particularly on left atrial function, remain limited. This study aims to evaluate the impact of SGLT-2 inhibitors (empagliflozin or dapagliflozin) on diastolic function in patients with heart failure with preserved ejection fraction who were naive to this therapy. Beyond conventional diastolic indices, the study incorporated advanced echocardiographic parameters including left atrial strain analysis, left atrial stiffness index, and the left atrioventricular coupling index to provide a broader understanding of the atrial-specific effects of SGLT-2 inhibition.

Methods: This prospective study included 50 patients diagnosed with heart failure with preserved ejection fraction who were newly initiated on SGLT-2 inhibitor therapy between April 1, 2024, and April 1, 2025, at Ankara University Faculty of Medicine outpatient clinics. Baseline and 3-month follow-up assessments included NYHA functional class, laboratory markers (including NT-proBNP, hemoglobin, and uric acid), and echocardiographic measurements (left atrial volume index, E/e' ratio, left atrial strain parameters, left atrial stiffness index, and left atrioventricular coupling index).

Results: After three months of treatment, there was a significant reduction in the E/e' ratio, reflecting improved left ventricular filling pressures (12 ± 3 vs. 9.8 ± 2.8; p<0.001). A meaningful reduction in left atrial volume index was also observed (34.63 ± 5.5 vs. 32.6 ± 5.7; p<0.001). Left atrial reservoir strain (18.58 ± 4.2% vs. 20.86 ± 5.0%; p<0.001) and pump strain (–9.18 ± 3.2% vs. –11.6 ± 3.1%; p<0.001) both significantly increased. The left atrial stiffness index decreased substantially (0.64 ± 0.26 vs. 0.47 ± 0.20; p<0.001). A significant improvement was also observed in the left atrioventricular coupling index (32.4% ± 7.4 vs. 29.0% ± 6.6; p<0.001). NT-proBNP levels declined significantly (367.2 ± 240.8 vs. 307.8 ± 177.9; p=0.007), accompanied by a notable improvement in NYHA functional class (2.24 ± 0.63 vs. 1.92 ± 0.60; p<0.001).

Table 1. Baseline characteristics of the study population

Characteristic	Value
Age (years)	63.88 ± 8.08
Sex	
• Female	35 (70%)
• Male	15 (30%)
Body Mass Index (kg/m²)	30.7 ± 3.9
NYHA Class	
• I	5 (10%)
• II	28 (56%)
• III	17 (34%)
• IV	0 (0%)
Type 2 Diabetes Mellitus	41 (82%)
Hypertension	46 (92%)
Coronary artery disease	21 (42%)
Smoking	19 (38%)
SGLT2 Inhibitor	
• Dapagliflozin	39 (78%)
• Empagliflozin	11 (22%)
Beta-blocker use	37 (74%)
ACEi/ARB use	38 (76%)
Insulin use	13 (26%)
Metformin use	40 (80%)
GLP-1 RA use	2 (4%)
Statin use	35 (70%)
HbA1c (%)	7.8 ± 1.9
Fasting blood glucose (mg/dL)	125 ± 25.4
Hemoglobin (g/dL)	12.6 ± 1.8
Hematocrit (%)	38.2 ± 5.65
Creatinine (mg/dL)	1.0 ± 0.3
eGFR (mL/min/1.73 m²)	69.8 ± 19.3
NT-proBNP (pg/mL)	367 ± 241
Uric acid (mg/dL)	5.7 ± 1.5
CRP (mg/L)	6.25 ± 5.9

Table 2. Baseline echocardiographic parameters

Echocardiographic Parameter	Value
Left Ventricular Ejection Fraction (LVEF, %)	56.2 ± 4.3
Left Ventricular End-Diastolic Diameter (cm)	4.9 ± 0.5
Left Ventricular End-Systolic Diameter (cm)	3.3 ± 0.4
Interventricular Septum Thickness (IVS, cm)	1.08 ± 0.14
Left Atrial Volume Index (LAVI, mL/m²)	34.63 ± 5.5
E/A Ratio	1.1 ± 0.4
E/e' Ratio	12 ± 3
Tricuspid Regurgitation Velocity (TRV, m/s)	2.6 ± 0.4
Left Atrial Stiffness Index	0.64 ± 0.26
Left Atrial Reservoir Strain (%)	18.58 ± 4.2
Left Atrial Pump Strain (%)	–9.2 ± 3.2
Left Atrioventricular Coupling Index (LACI, %)	32.4 ± 7.4

Table 3. Comparison of parameters at baseline and at 3-month follow-up

Parameter	Baseline	3-Month Measurements	p value
NYHA Class	2.24 ± 0.63	1.92 ± 0.60	<0.001
E/e' ratio	12 ± 3	9.8 ± 2.8	<0.001
Left atrial volume index (ml/m²)	34.63 ± 5.5	32.6 ± 5.7	<0.001
Left atrial reservoir strain (%)	18.58 ± 4.2	20.86 ± 5.0	<0.001
Left atrial pump strain (%)	–9.2 ± 3.2	–11.6 ± 3.1	<0.001
Left atrial stiffness index	0.64 ± 0.26	0.47 ± 0.20	<0.001
Left atrioventricular coupling index (LACI) (%)	32.4 ± 7.4	29 ± 6.6	<0.001
NT-proBNP (pg/mL)	367.2 ± 240.8	307.8 ± 177.9	0.007
Uric Acid (mg/dl)	5.65 ± 1.5	5 ± 1.55	<0.001
Hemoglobin (g/dl)	12.6 ± 1.78	12.79 ± 1.8	0.007
Hematocrit (%)	38.2 ± 5.65	39.1 ± 5.32	0.008

Conclusions: This study demonstrated that SGLT-2 inhibitors lead to early and significant improvements in diastolic function and left atrial parameters in patients with heart failure with preserved ejection fraction. The observed improvements in E/e′ ratio, left atrial volume index, left atrial strain, stiffness index, and left atrioventricular coupling index suggest a positive effect on atrial function. Notably, this is the first study to report a significant improvement in the left atrioventricular coupling index with SGLT-2 inhibitor therapy. These advanced echocardiographic indices may be useful for early diagnosis and treatment monitoring.

PP-012 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Is it safe to perform direct-current cardioversion during left atrial appendage occlusion with thrombus?

Nihan Bahadır, Osman Bektaş, Fatih Akkaya, İdris Buğra Cerik, Seçkin Dereli, Ahmet Kaya

Department of Cardiology, Ordu University, Faculty of Medicine, Ordu

Background and Aim: Percutaneous left atrial appendage occlusion (LAAO), which is a device-based therapy, stands out as the main therapy approach in AF patients who have intolerance or recurrent stroke under anticoagulant therapy. Approximately ninety percent of strokes in patients with atrial fibrillation (AF) occur due to thrombus formation in the left atrial appendage (LAA). Warfarin and new oral anticoagulants (NOACs) are commonly used to prevent thromboembolic events due to AF. Although some AF patients have high thromboembolic risk, anticoagulant therapy can be discontinued because of high bleeding risk, major contraindications, or intolerance. According to the increasing number of LAAO procedures, direct current cardioversion (DCCV) can occur as an alternative approach to relieve clinical symptoms in AF patients who have previous catheter ablation or contraindications. We describe the first cases of DCCV performed on AF patients with LAA thrombus during the LAAO procedure after the device was released. To demonstrate the feasibility and safety of performing DCCV during the LAAO procedure after the device was released when clinically necessary with absolute indications even though patients had LAA thrombus.

Methods: This study was approved by Ordu University Training and Research Hospital, and consent was waived due to the retrospective nature of this case report. All three patients had thrombus formation at the mid and distal regions of the left atrial appendage before the LAAO. In these cases, the reasons for undergoing the LAAO procedure were despite being on anticoagulant therapy, experiencing recurrent strokes with haemorrhagic transformation, complicated intraocular haemorrhage, and tachycardiomyopathy caused by atrial fibrillation with a persistent LAA thrombus that prevented ablation. After device deployment, AF with a high ventricular response causing hemodynamic instability appeared in all three patients, and a single 200-J DCCV was performed. Following DCCV, LAA device placement was controlled by TEE and fluoroscopy.

Results: At the same time, during the LAAO procedure and after the device release, immediately performing DCCV didn't result in device migration, device embolization, peridevice leak (>3 mm), or any neurological symptoms. There were no complications or device migrations as a result of the TEE assessment during the procedure, and the transthoracic echocardiographic assessment was performed 72 hours and 1 month after the procedure.

Conclusions: These three cases demonstrate the feasibility and safety of uncomplicated direct-current cardioversion during the LAAO procedure with patients who had thrombus at LAA. The purpose of this case presentation was to demonstrate that percutaneous LAAO procedure can be performed safely with absolute indications, even though patients had LAA thrombus, and DCCV can be used immediately after the device is released at the time of procedure when it is necessary.

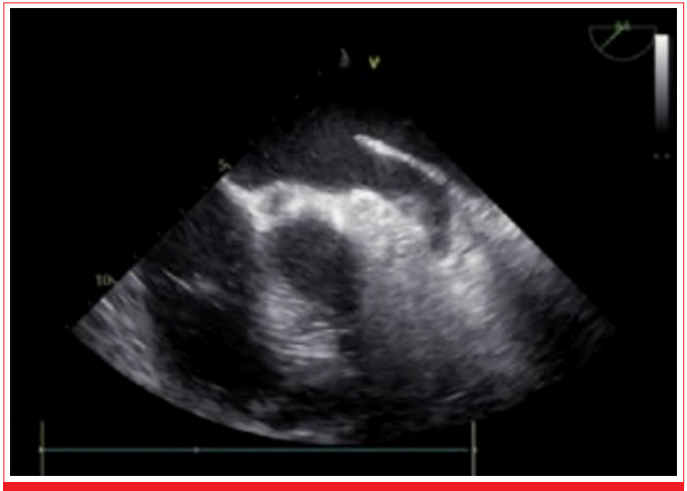

Figure 1. 6 x 9 mm Thrombus Formation at Mid LAA on TEE Image.

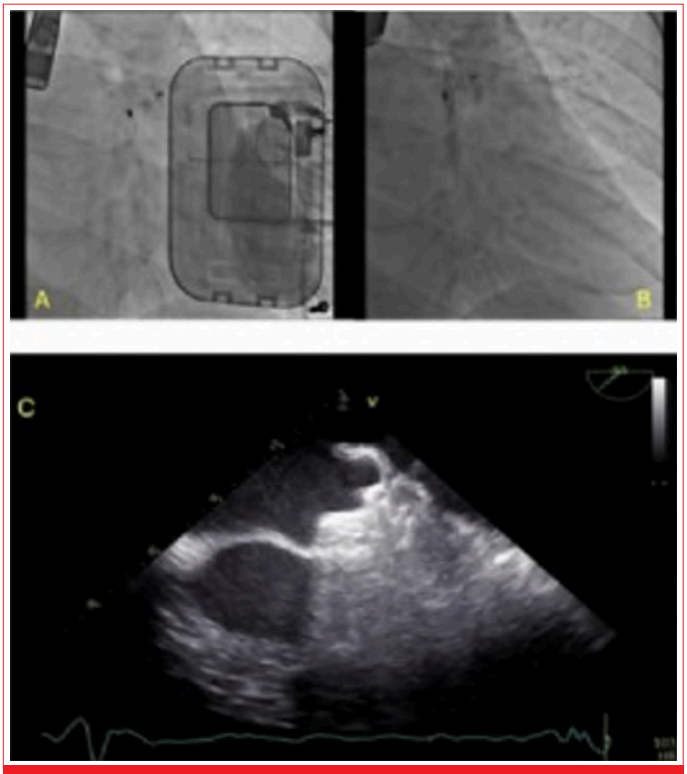

Figure 2. A) Fluoroscopy Image During DCCV; B,C) LAAO Device Is Seen in LAA After DCCV At Fluoroscopy Images and TEE Image.

PB-013 [Coronary Artery Disease / Acute Coronary Syndrome]

The relationship between plasma atherogenic index and long-term mortality in patients with ST-segment elevation myocardial infarction

Ali Evsen[1], Adem Aktan[2]

[1]Department of Cardiology, Dağkapı State Hospital, Diyarbakır 2Department of Cardiology, Artuklu University, Faculty of Medicine, Mardin

Background and Aim: ST-segment elevation myocardial infarction (STEMI) is typically caused by the sudden and complete occlusion of coronary artery blood flow. Dyslipidemia is a well-established risk factor for cardiovascular diseases (CVD). The prognosis of the disease is influenced by existing risk factors and angiographic findings. The atherogenic index of plasma (AIP), calculated as the logarithm of the TG/HDL-C ratio, is considered a simple and alternative marker of plasma atherogenicity. The prognostic value of AIP in this population remains unclear. The objective of this study is to evaluate the association between the AIP and long-term mortality in patients with STEMI undergoing primary percutaneous coronary intervention (pPCI).

Methods: A total of 651 consecutive patients diagnosed with STEMI at a single center between January 1, 2018, and January 1, 2020, were retrospectively included in the study. Based on the optimal cut-off value of 0.578 for the AIP, determined using Youden's index, patients were divided into two groups: low AIP (≤0.57, n=319) and high AIP (>0.57, n=332). Patients were followed up retrospectively for an average of 39.3 ± 16.9 months.

Table 1. Comparison of clinical, laboratory, in-hospital adverse cardiovascular events and comorbid conditions of patients with low and high plasma atherogenic index

Variables	All Patients (n=651)	Low AIP (n=319)	High AIP (n=332)	p value
Gender (Female), n (%)	188 (28.9)	106 (33.2)	82 (24.7)	0.016
Age (years)	61.94 ± 13.91	63.35 ± 13.75	60.59 ± 13.95	0.011
Smoking, n (%)	430 (66.1)	198 (62.1)	232 (69.9)	0.035
HT, n (%)	371 (57.0%)	194 (60.8%)	177 (53.3%)	0.053
DM, n (%)	195 (30)	78 (24.5)	117 (35.2)	0.003
Dyslipidemia, n (%)	272 (41.8)	94 (29.5)	178 (53.6)	<0.001
CKD, n (%)	37 (5.7)	16 (5.0)	21 (6.3)	0.471
Previous MI, n (%)	125 (19.2)	54 (16.9)	71 (21.4)	0.149
Previous PCI, n (%)	98 (15.1)	40 (12.5)	58 (17.5)	0.079
Previous CABG, n (%)	31 (4.8)	20 (6.3)	11 (3.3)	0.077
LVEF (%)	44.55 ± 10.19	45.32 ± 9.87	43.82 ± 10.45	0.065
Recurrent MI, n (%)	50 (7.7)	19 (6.0)	31 (9.3)	0.105
VT, VF	51 (7.8)	18 (5.6)	33 (9.9)	0.041
Killip >2	115 (17.7)	47 (14.7)	68 (20.5)	0.055
Chronic heart failure, n (%)	105 (16.1)	44 (13.8)	61 (18.4)	0.112
Pulmonary oedema, n (%)	53 (8.1)	21 (6.6)	32 (9.6)	0.154
Cardiogenic shock, n (%)	45 (6.9)	17 (5.3)	28 (8.4)	0.119
WBC (x103/uL)	12.80 ± 2.61	12.26 ± 3.34	13.33 ± 1.44	<0.001
Lymphocyte (x103/uL)	1.91 ± 0.97	1.85 ± 0.97	1.97 ± 0.96	0.114
Neutrophil (x103/uL)	8.78 ± 2.80	8.54 ± 3.04	9.01 ± 2.54	0.034
Hemoglobin (gr/L)	13.47 ± 1.90	13.33 ± 2.09	13.61 ± 1.70	0.059
Platelet (103/uL)	260.40 ± 68.18	256.53 ± 71.61	264.12 ± 64.60	0.156
Glucose (mg/dL), median (Q1-Q3)	139 (115-207)	133 (112-191)	150 (117-221)	0.002
Creatinine (mg/dL), median (Q1-Q3)	0.84 (0.72-1.05)	0.83 (0.72-1.03)	0.84 (0.73-1.05)	0.266
CRP (mg/dL), median (Q1-Q3)	0.59 (0.29-1.19)	0.60 (0.20-1.08)	0.58 (0.31-1.33)	0.226
Total cholesterol (mg/dL)	179.02 ± 42.59	174.0 ± 42.15	183.72 ± 42.53	0.004
HDL cholesterol (mg/dL)	37.05 ± 11.14	39.32 ± 11.91	34.95 ± 9.95	<0.001
LDL cholesterol (mg/dL)	12.45 ± 38.58	121.65 ± 42.22	132.72 ± 34.17	<0.001
Triglyceride (mg/dL), median (Q1-Q3)	134 (89-218)	116 (80-189)	149 (98-227)	<0.001

HT: Hypertension; DM: Diabetes mellitus; CKD: Chronic kidney disease; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CABG: Coronary artery by-pass graft; LVEF: Left ventricular ejection fraction; VT: Ventricular tachycardia; VF: Ventricular fibrillation; WBC: White blood cells (106/L), CRP: C-reactive protein; HDL cholesterol: High-density lipoprotein cholesterol (mg/dL); LDL cholesterol: Low-density lipoprotein cholesterol (mg/dL); Data are presented as mean ± SD, median (Q1-Q3) or n (%). Statistical significance is considered at a p-value of less than 0.05.

Results: In the high AIP group, the proportion of female patients was lower (p=0.016), and patients were younger (p=0.011). The prevalence of diabetes mellitus (DM) (p=0.003), dyslipidemia (p<0.001), and smoking (p=0.035) was significantly higher in the high AIP group. White blood cell count (p<0.001), neutrophil count (p=0.034), glucose (p=0.002), total cholesterol (p=0.004), LDL (p<0.001), and triglyceride levels (p<0.001) were significantly higher in the high AIP group, whereas HDL levels were lower (p<0.001). Additionally, the incidence of ventricular tachycardia/ventricular fibrillation (VT/VF) (p=0.041), in-hospital mortality (p<0.001), one-year mortality (p<0.001), and five-year mortality (p<0.001) were significantly higher in the high AIP group. The mean follow-up duration was also shorter compared to the low AIP group (p=0.044). In the Cox regression analysis evaluating variables associated with five-year mortality, univariate analysis identified DM (p<0.001), hypertension (HT) (p<0.001), chronic kidney disease (CKD) (p<0.001), reduced ejection fraction (EF) (p<0.001), and elevated AIP levels (p<0.001) as significant risk factors. In the multivariate analysis, DM (HR: 3.40, 95% CI: 2.21–5.23, p<0.001), CKD (HR: 1.56, 95% CI: 1.03–2.35, p=0.034), reduced EF (HR: 0.97, 95% CI: 0.96–0.99, p=0.004), and elevated AIP levels (HR: 1.65, 95% CI: 1.16–2.34, p=0.005) were identified as independent predictors of five-year mortality.

Conclusions: This study demonstrates that the AIP predicts long-term mortality in STEMI patients undergoing pPCI. As a parameter that can be easily calculated using routine lipid profiles, AIP may serve as a useful marker for risk stratification, guiding early treatment strategies, and ultimately reducing mortality in patients with STEMI.

Table 2. Comparison of angiographic characteristics, follow-up duration, and mortality outcomes between patients with low and high plasma atherogenic indices

Variables	All Patients (n=651)	Low AIP (n=319)	High AIP (n=332)	p value
Type of MI, n (%)				
-Anterior MI,	265 (40.7)	129 (40.4)	136 (41.0)	
-Inferior MI,	244 (37.5)	116 (36.4)	128 (38.6)	0.681
-Other	142 (21.8)	74 (23.2)	68 (20.5)	
Culprit artery, n (%)				0.721
-LAD	307 (47.2)	146 (45.8)	161 (48.5)	0.044
-Cx	118 (18.1)	56 (17.6)	62 (18.7)	<0.001
-RCA	192 (29.5)	98 (30.7)	94 (28.3)	<0.001
-Other (IMA,Diagonal)	34 (5.2)	19 (6.0)	15 (4.5)	<0.001
Follow-up Duration (months)	39.3 ± 16.9	40.7 ± 14.5	37.9 ± 18.8	
In-hospital Mortality, n (%)	51 (7.8)	13 (4.1)	38 (11.4)	
First-year Mortality, n (%)	95 (14.6)	29 (9.1)	66 (19.9)	
Five-year Mortality, n (%)	159 (24.4)	52 (16.3)	107 (32.2)	

LAD: Left anterior descending; Cx: Circumflex; RCA: Right coronary artery; MI: Myocardial infarction; IMA: Internal mammary artery. Statistical significance is considered at a p-value of less than 0.05.

Table 3. Independent predictors of 5-year mortality in univariate and multivariate Cox regression analysis models

Variables	Univariate analysis	Univariate analysis	Univariate analysis	Multivariate analysis	Multivariate analysis	Multivariate analysis
	HR	95% CI	p	HR	95% CI	p
Age	1.00	0.99-1.02	0.556			
Gender	0.71	0.49-1.03	0.075			
HT	2.62	1.81-3.80	<0.001	1.40	0.90-2.17	0.132
DM	5.58	3.89-8.01	<0.001	3.40	2.21-5.23	<0.001
CKD	3.98	2.75-5.77	<0.001	1.56	1.03-2.35	0.034
LDL-C	1.00	0.99-1.01	0.719			
LVEF	0.92	0.90-0.94	<0.001	0.97	0.96-0.99	0.004
WBC	1.00	0.94-1.06	0.995			
Hemoglobin	1.07	0.98-1.17	0.123			
AIP	1.82	1.31-2.55	<0.001	1.65	1.16-2.34	0.005

HT: Hypertension; DM: Diabetes mellitus; CKD: Chronic kidney disease; LDL-C: Low-density lipoprotein cholesterol (mg/dL); LVEF: Left ventricular ejection fraction; WBC: White blood cells (10^6/L), AIP: Plasma atherogenici. Statistical significance is considered at a p-value of less than 0.05.

PP-014 [Other]

Prognostic nutritional index and frailty after ischemic stroke: Mid-term follow-up results

Sibel Çatalkaya, Eda Özcan

Department of Cardiology, Bursa City Hospital, Bursa

Background and Aim: The Prognostic Nutritional Index (PNI), a biomarker reflecting systemic inflammation and nutritional status, has been associated with morbidity and mortality in oncology, cardiology, and critical care settings. Frailty, characterized by reduced physiological reserve and heightened vulnerability to stressors, is a well-established predictor of adverse outcomes after stroke. While frailty has been shown to affect both early and long-term recovery and survival after stroke, its association with PNI in patients with ischemic stroke remains underexplored. This study aimed to investigate the relationship between PNI measured at the time of stroke and mid-term frailty status assessed during follow-up.

Methods: We retrospectively analyzed patients admitted with ischemic stroke between 2021 and 2023. Patients with malignancies or chronic systemic diseases were excluded. PNI was calculated at admission using the formula: PNI=10 x serum albumin (g/dL) + 0.005 x total lymphocyte count (/mm³). Patients were stratified into two groups based on a literature-based PNI cutoff value of 49.75: malnourished (PNI ≤49.75) and well-nourished (PNI >49.75). Frailty was assessed during outpatient visits or via structured telephone interviews using the Frailty Scale (FS), a 0–5 scoring tool evaluating daily activity, mobility, and general health. A score ≥3 was defined as frail.

Results: A total of 86 patients (mean age: 66 ± 11.1 years; 64.6% male) were included, with a median follow-up duration of 16 months (range: 3–25 months). The prevalence of comorbidities was high, including hypertension (77.9%), diabetes (45.4%), and coronary artery disease (36.1%). Frailty was significantly more common among patients with lower PNI values (35.3% vs. 15.7%; p=0.047). Similarly, mean frailty scores were higher in the malnourished group (2.1 ± 1.6 vs. 1.3 ± 1.5; p=0.017). There was an inverse and statistically significant correlation between PNI and frailty scores (Spearman r=-0.53; p<0.001). Patients with low PNI also exhibited higher neutrophil and CRP levels and lower hemoglobin, triglyceride, and albumin values (Detailed estimates are presented in Table 1).

Conclusions: Lower PNI values at the time of ischemic stroke were significantly associated with increased frailty during mid-term follow-up. These findings suggest that PNI may serve not only as a biochemical marker but also as a valuable indicator of functional decline after stroke. Moreover, early identification of at-risk individuals through PNI may enable tailored therapeutic strategies, including more intensive rehabilitative follow-up, to improve clinical outcomes in this vulnerable population. Given its simplicity, low cost, and objectivity, PNI could be a valuable tool in routine clinical practice.

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

	Study population (n=86)	PNI <=49,75 (n=34)	PNI >49,75 (n=51)	p value
Age (years)	66 ± 11.1	68.5 ± 9.8	61 ± 11.8	0.07
Gender (male), n (%)	62 (64.6%)	20 (58.8%)	37 (72.5%)	0.19
Hypertension, n (%)	67 (77.9%)	25 (73.5%)	42 (82.4%)	0.33
Diabetes mellitus, n (%)	39 (45.4%)	15 (44.1%)	24 (47.1%)	0.83
Coronary artery disease, n (%)	31 (36.1%)	11 (32.4%)	20 (39.2%)	0.52
Heart failure, n (%)	20 (23.3%)	9 (26.5%)	11 (21.6%)	0.60
Chronic kidney disease, n (%)	18 (20.9%)	9 (26.5%)	9 (17.6%)	0.33
Ejection fraction (%)	55 ± 7.5	55 ± 8.4	55.1 ± 7	0.96
Frailty Scale (FS)	1.7 ± 1.6	2.1 ± 1.6	1.3 ± 1.5	0.017*
Frail (FS >=3), n (%)	20 (23.3%)	12 (35.3%)	8 (15.7%)	0.047*
Laboratory parameters				
Leukocytes (10^3/μL)	8.8 ± 2.9	8.8 ± 3.2	8.8 ± 2.7	0.44
Neutrophils (10^3/μL)	6.1 ± 3	7.4 ± 3.5	5.3 ± 2.1	0.002*
Lymphocytes (10^3/μL)	3.1 ± 7.9	3.6 ± 12.4	2.8 ± 1.1	0.63
Hemoglobin (g/dL)	13.3 ± 2	12.7 ± 1.9	13.7 ± 1.9	0.02*
Creatinine (mg/dL)	1 ± 0.4	1 ± 0.6	0.9 ± 0.3	0.29
GFR (mL/min/1.73 m²)	80.4 ± 23.7	81 ± 26.4	88.9 ± 21.3	0.13
Triglycerides (mg/dL)	144.5 (20-1058)	145.1 (20-342)	167 (52-1058)	0.03*
LDL (mg/dL)	113.8 ± 33.5	113.5 ± 31.2	106 ± 31.9	0.29
HDL (mg/dL)	41.5 ± 10.4	39 ± 8.8	42.1 ± 10.7	0.17
Albumin (g/L)	41 ± 4.3	37.5 ± 3.9	42.7 ± 3.1	<0.001*
CRP (mg/dL)	3.9 (0.4-302)	8 (0.7-301)	2.7 (0.4-302)	0.04*

Data are presented as n (%), mean ± standard deviation, or median (min–max). *Statistically significant (p<0.05).

PP-015 [Epidemiology]

Stratification of HCM patients based on genetic variant classification from a single center

Rabia Okumuş[1], Selda Murat[1], Fatih Enes Durmaz[2], Sinem Kocagil[3], Yüksel Çavuşoğlu[1]

[1]*Department of Cardiology, Eskişehir Osmangazi University, Faculty of Medicine, Eskişehir*
[2]*Department of Cardiology, Muş State Hospital, Muş*
[3]*Department of Medical Genetics, Eskişehir Osmangazi University, Faculty of Medicine, Eskişehir*

Background and Aim: Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiac disease, marked by left ventricular hypertrophy in the absence of abnormal loading conditions. Genetic testing has become increasingly important for diagnosis, risk stratification, and family screening. However, the clinical significance of variants of uncertain significance (VUS) and the phenotypic distinctions among genetic subgroups remain areas of investigation. This study aimed to evaluate the genetic distribution and associated clinical characteristics of patients diagnosed with HCM.

Methods: This single-center, descriptive study prospectively included 177 consecutive patients diagnosed with HCM between April 2023 and April 2025. HCM was defined as unexplained LV wall thickness ≥15 mm on echocardiography. Genetic analysis was performed using next-generation sequencing-based clinical exome panels. Based on genetic results, patients were divided into four groups: genotype-negative (n=78), VUS (n=53), likely pathogenic (n=20), and pathogenic (n=26).

Results: Of the 177 patients, 99 (55.9%) carried at least one reportable genetic variant, while 78 (44.1%) were genotype-negative. Among genotype-positive patients, 53 (30.0%) had VUS, 20 (11.3%) had likely pathogenic variants, and 26 (14.7%) had pathogenic variants. Among gene-specific findings, MYBPC3 was the most frequently observed gene mutation (n=14), followed by TTR and MYH7. In the VUS group (n=53), the most frequently observed genes were TTN (n=6), FLNC (n=5), LAMA4 (n=4), and DSP (n=3). Several genes appeared in two patients each, including ABCC6, MYH6, MYBPC3, RYR2, and VCL, while the remaining genes were detected in single individuals.In the likely pathogenic group (n=20), the gene with the highest prevalence was MYBPC3 (n=6), followed by DES (n=2) and TNNT2 (n=2). The remaining genes—including NDUFAF1, MYH6, TTR, SCN5A, PLEC, LAMP2, ACTA1, MYH7, MYBP3, and TTN—were each detected in a single case. In the pathogenic group (n=26), MYBPC3 was again the most frequently encountered gene, detected in 9 patients. TTR was found in 6 patients, followed by MYH7 in 5 and TNNT2 in 3. Additionally, DMD, ILDR1, and TNNC1 were each found in one patient. This distribution emphasizes the predominance of sarcomeric genes—particularly MYBPC3 and MYH7—in the pathogenic subgroup, whereas VUS mutations were more heterogeneous and included a broader spectrum of genes with lower recurrence.

Conclusions: This study demonstrates the importance of integrating genetic data into the clinical evaluation of HCM patients. Mean while, the genotype-negative and VUS groups encompassed a broader and more heterogeneous population, often presenting with milder or less distinct features. The high frequency of sarcomeric gene involvement, particularly MYBPC3 and MYH7, reinforces their central role in HCM pathogenesis. These findings underscore the utility of genetic testing for refining diagnosis, guiding follow-up strategies, and informing familial risk assessment in patients with HCM.

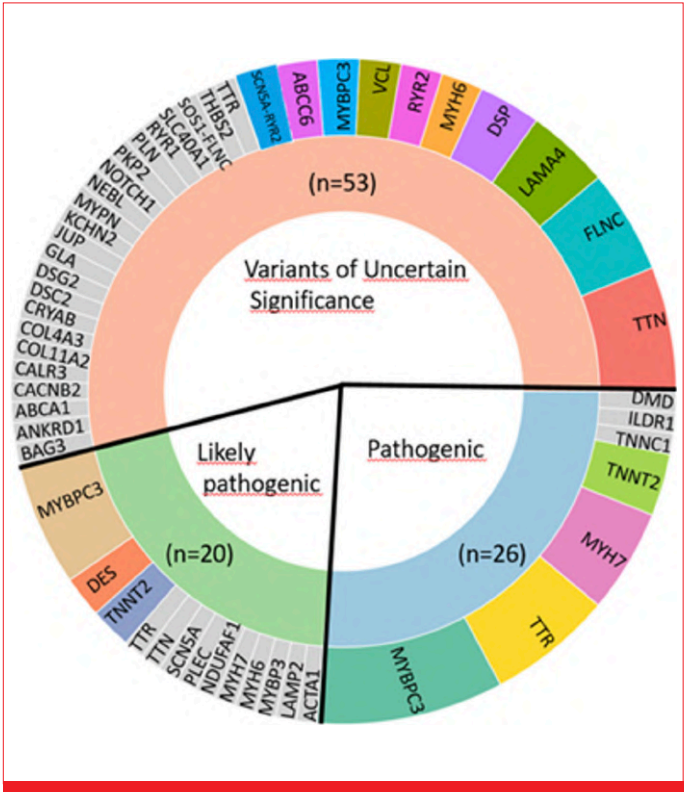

Figure 1. Distribution of Genetic Variants in HCM Patients Categorized as Pathogenic, Likely Pathogenic, and VUS, with Detailed Gene-Specific Findings.

Table 1. Clinical and demographic characteristics of the overall cohort and genetic subgroup

	Overall (n=177)	Genotype Negative	Genotype Positive Total (n=99)	VUS (n=53)	Likely Pathogen (n=20)	Pathogen (n=26)
Age (year)						
Mean (SD)	55.8 ± 13.3	57.5 ± 12.5	54.4 ± 13.8	56.8 ± 12.4	52.8 ± 16.1	50.7 ± 14.3
Median (min-max)	56 [19-96]	58 [25-93]	54 [19-96]	60 [30-82]	56 [19-73]	50 [27-96]
Gender, n (%)						
Female	78 (44.1%)	38 (48.7%)	40 (40.4%)	20 (37.7%)	6 (30.0%)	14 (53.8%)
Male	99 (55.9%)	40 (51.3%)	59 (59.6%)	33 (62.3%)	14 (70.0%)	12 (46.2%)

Table 2. Clinical, laboratory, and echocardiographic characteristics by genetic group

	Genotype (-) (n=78)	VUS (n=53)	Pathogen + Likely pathogen (n=46)	p value
Age, years, ± SD	57.5 ± 12.5	56.8 ± 12.4	51.6 ± 15.0	0.049*
Body Mass Index (kg/m²)	28.3 ± 5.2	27.9 ± 3.6	27.9 ± 4.5	0.905
NT-proBNP, pg/mL [IQR]	745 [201-1849]	287 [70-1179]	689 [119-2126]	0.183
LVEF, %	57.1 ± 8.0	58.3 ± 7.0	59.9 ± 8.1	0.151
IVS (mm)	15.8 ± 2.4	15.5 ± 3.5	17.1 ± 4.4	0.041**
PWT (mm)	13.4 ± 2.0	13.3 ± 2.2	13.9 ± 2.9	0.319
Left atrium volume index, mL/m³	31.4 ± 13.2	28.6 ± 13.5	35.3 ± 14.4	0.070
Septal e', cm/s	5.9 ± 1.8	5.5 ± 2.1	5.5 ± 2.2	0.480
Lateral e', cm/s	7.5 ± 2.7	7.4 ± 2.7	7.3 ± 3.1	0.883
RA diameter, mm,	37.0 ± 5.9	36.2 ± 3.3	37.3 ± 4.7	0.515
TJV, m/s	2.2 ± 0.5	2.1 ± 0.4	2.2 ± 0.5	0.567

* There is a statistically significant difference between the genotype-negative group and the likely pathogenic/pathogenic group. ** There is a statistically significant difference between the VUS group and the likely pathogenic/pathogenic group.

PP-016 [Heart Failure]

Left atrioventricular coupling index in heart failure: Diagnostic and discriminative role across heart failure subtypes

Ezgi Çamlı Babayiğit[1], Selda Murat[2], Erdi Babayiğit[2], Bülent Görenek[2]

[1]*Eskişehir City Hospital, Eskişehir*
[2]*Department of Cardiology, Eskişehir Osmangazi University, Faculty of Medicine, Eskişehir*

Background and Aim: The Left Atrioventricular Coupling Index (LACI), defined as the ratio of left atrial to left ventricular end-diastolic volume, reflects the mechanical interplay between the atrium and ventricle. It has been proposed as a novel marker of atrioventricular remodeling and is associated with diastolic dysfunction severity and is an independent predictor of outcomes in patients with HF. This study aimed to assess the utility of LACI in detecting HF and differentiating among its subtypes based on left ventricular ejection fraction (LVEF).

Methods: A total of 144 patients were evaluated and stratified into two major groups based on clinical and echocardiographic findings: patients with heart failure (HF+, n=79) and those without heart failure (HF–, n=65). The HF+ group was further categorized as HFrEF (n=30), HFmrEF (n=20), and HFpEF (n=29). Demographic, clinical, and echocardiographic parameters including LA volume, LVEF, LAEF, and LACI were compared across groups.

Results: LACI was significantly higher in the HF+ group compared to HF– (0.23 ± 0.15 vs. 0.14 ± 0.08, p<0.001). Among HF subtypes, LACI was highest in HFpEF patients (HFpEF: 0.32 ± 0.18 vs. HFrEF: 0.17 ± 0.10, HFmrEF: 0.17 ± 0.09, p<0.001). LA volume, LAVI, and LAEF showed significant differences between HF+ and HF–, supporting the presence of atrial remodeling in HF patients. GLS and NT-proBNP levels were also significantly worse in the HF+ group.

Conclusions: LACI is significantly elevated in patients with heart failure and demonstrates the highest values in HFpEF, indicating its potential as a non-invasive marker of left atrioventricular uncoupling. These findings support the role of LACI as a novel parameter to distinguish heart failure subtypes, especially in the evaluation of diastolic dysfunction.

Table 1. Comorbities ve demographics in all population and HF groups

	All Population (n=144)	HF+ (n=79, SD)	HF- (n=65, SD)	p value
Female gender, n, (%)	57 (%40)	22 (%27,8)	35 (%53,8)	0,002
Age (mean years)	62,5 (24-90)	67 (35-90)	57 (24-65)	0,06
BMI (kg/m2)	28,4 (17-41)	27,5 (±4,5)	29,5(±5,31)	0,018
HT (%)	112 (%77,8)	66 (%83,5)	46 (%70,8)	0,067
DM (%)	81 (%56,3)	45 (%57)	36 (%55,4)	0,84
CVD (%)	40 (%27,8)	19 (%24,1)	21 (%32,3)	0,27
Vascular Disease (%)	93 (%64,6)	62 (%78,5)	31 (%47,7)	<0,001
CRF (%)	52 (%36,1)	36 (%45,6)	16 (%24,6)	0,009
KOAH (%)	20 (%13,9)	15 (%19)	5 (%7,7)	0,05
Dyslipidemia (%)	35 (%23,5)	20 (%25,3)	11 (%16,9)	0,24
Smoking (%)	62 (%43,1)	39 (%49,4)	23 (%35,4)	0,09
Nt-ProBNP (pg/ml)	3455 (10-35000)	5036 (1250-35000)	196 (10-550)	<0,001

Table 2. Echocardiographic parameters in all population and HF groups

Variables	All Population (n=144)	HF+ (n=79, SD)	HF- (n=65, SD)	p value
LA volume (ml)	57,1 (21-145)	67,8 (±22,7)	44,4(±12,0)	<0,001
LA diameters (mm)	39,4 (33-50)	43,4 (±5,5)	37,6 (±4,14)	<0,001
LAVI (ml/m2)	30,5 (11-77)	36,8(±12,9)	23,01(±5,6)	<0,001
LAEF (%)	58 (14-89)	51,4 (±15,1)	68,1 (±8)	<0,001
LVEF (%)	52,8 (17-74)	44,3 (±13,6)	63,2(±4,8)	<0,001
LACI	0,19 (0,02-0,79)	0,23 (±0,15)	0,14 (±0,08)	<0,001
LAVmaks (ml)	58,6 (12,4-177)	68,5 (±28,8)	46,2 (±14,2)	<0,001
LAVmin (ml)	26,9 (3-109))	35,8 (±21,4)	16,1 (± 6,7)	<0,001
LV diastolic volume (ml)	151,4 (47-406)	176,3 (±67)	121,6 (±40,8)	<0,001
LV systolic volume (ml)	74,7 (14-205)	98,7 (±54,4)	45,9 (±18,9)	<0,001
Mitral E/e'	10,3 (4,4-38)	11,9 (±6,5)	8,3 (±2.5)	<0,001
GLS (-%)	16,4 (4-24)	13,7 (±2,9)	<0,001	<0,001

Table 3. Echocardiographic parameters between HF subgroups

Variables , SD	HFrEF (n=30)	HFmrEF (n=20)	HFpEF (n=29)	p value
LA volume (ml)	69,9 (±22,2)	61,8 (±21,7)	69,9 (±22,7)	0,38
LA diameters (mm)	43,8 (± 5,8)	41,6 (±5,0)	44,2 (±5,5)	0,22
LAVI (ml/m2)	37,1 (±12,2)	31,8 (±11,3)	42,9 (±14,0)	0,04
LAEF (%)	50,5 (± 17,9)	57,6 (±12,1)	48,5 (±12,8)	0,09
LVEF (%)	30,1 (± 6,4)	44,2 (±3,06)	59,0 (± 5,4)	<0,001
LACI	0,17 (±0,1)	0,17 (±0,09)	0,32 (±0,18)	<0,001
LAVmaks (ml)	67,8 (± 26,2)	56,5 (±21,03)	77,4 (±33,3)	0,043
LAVmin (ml)	36,4 (±22,3)	25,6 (±14,0)	42,3 (± 22,6)	0,024
LV diastolic volume (ml)	207 (±74)	157 (±52,9)	156,3 (±55,3)	0,004
LV systolic volume (ml)	138,2 (± 56,6)	84,7 (±33,9)	66,3 (±35,09)	<0,001
Mitral E/e'	12,2 (± 6,9)	11,7 (±6,7)	11,8 (±6,1)	0,9
GLS (-%)	11,8 (±3,09)	14,2 (±2,1)	15,2 (±2,03)	<0,001

PP-017 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Effect of cytisine use in smoking cessation treatment on ventricular repolarization parameters

Cahit Coşkun[1], Derya Tosun[3], Bilal Çakır[2], Burak Çetinkaya[3]

[1]*Department of Cardiology, Manisa Demirci State Hospital, Manisa*
[2]*Department of Cardiology, İstanbul Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul*
[3]*Department of Cardiology, Manisa Demirci State Hospital, Manisa*

Background and Aim: Cytisine is a a pharmacological agent widely used for smoking cessation, acting as a partial agonist of the α4β2 nicotinic acetylcholine receptor. While varenicline, a drug with a similar mechanism of action, has been associated with electrocardiographic (ECG) alterations, the electrophysiological safety profile of cytisine remains unclear. This study aimed to evaluate the effects of cytisine use on electrocardiographic parameters, particularly QT, QTc, Tp-e and the Tp-e/QTc ratio.

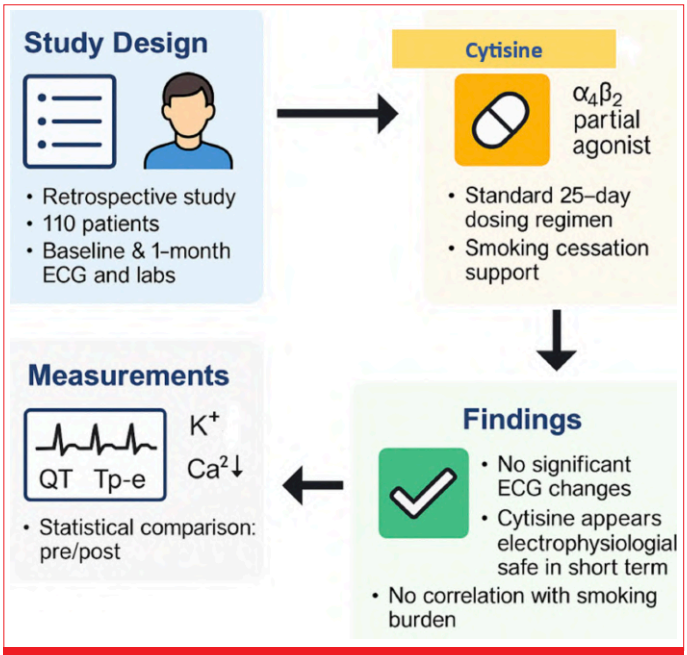

Figure 1. Study design.

Table 1. Comparison of laboratory and electrocardiographic parameters before and after cytisine treatment

	Initial	Follow-up	P Value
Leukocyte (x 10^3 /µL)	8,49 ± 2,31	8,46 ± 3,03	0,84
Haemoglobin (g/dL)	14,92 ± 1,60	14,86 ± 1,85	0,64
Platelet (x 10^3 /µL)	232 ± 43	243 ± 33	0,65
Creatinine (mg/dL)	0,92 ± 0,34	0,96 ± 0,43	0,67
AST (IU/L)	22,1 ± 6,2	22,5 ± 6,1	0,48
ALT (IU/L)	26,3 ± 7,8	26,1 ± 8,0	0,51
Na (mEq/L)	138,85 ± 15,89	136,52 ± 22,79	0,89
K (mEq/L)	4,14 ± 0,30	4,2 ± 0,41	**0,03**
Ca (mg/dl)	9,10 ± 0,40	8,8 ± 0,44	**0,02**
Mg (mg/dl)	1,97 ± 0,22	1,87 ± 0,22	0,16
Systolic BP (mmHg)	128 ± 18	130 ± 20	0,06
Diastolic BP (mmHg)	75 ± 11	76 ± 13	0,09
Heart rate	76 ± 11	77 ± 12	0,10
QT, ms	363,4 ± 25,6	364,1 ± 26,6	0,45
QTc, ms (Fridericia)	392 [21,5]	390 [29,5]	0,36
QRS, ms	94 ± 10	95 ± 9	0,52
Tp-e, ms	80 [14]	80 [15,5]	0,07
Tp-e/QTc	0,20 ± 0,03	0,20 ± 0,03	0,26

AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; Na: Sodium; K: Potassium; Ca: Calsium; Mg: Magnesium; BP: Blood pressure; QTc: Corrected QT interval; Tp-e: Tpeak-Tend

Methods: A retrospective analysis was conducted on 110 patients who completed a 25-day cytisine regimen for smoking cessation. Patients with structural heart disease, arrhythmias, QT-prolonging drugs, electrolyte disturbances, or incomplete follow-up were excluded. Standard 12-lead ECGs and serum biochemistry were assessed before treatment and at the 1-month follow-up. Statistical analyses included paired tests and correlation analysis.

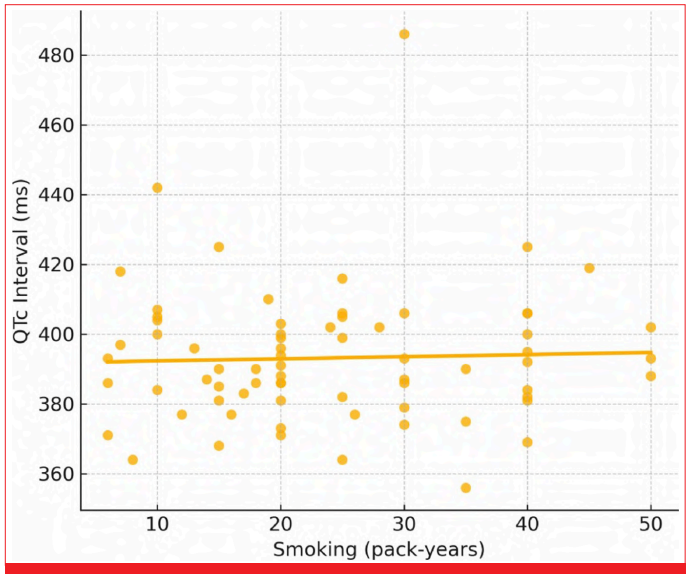

Figure 2. Correlation between cumulative smoking exposure (pack-years) and baseline Tp-e/QTc ratio.

Results: No statistically significant changes were observed in QT, QTc, Tp-e, or Tp-e/QTc intervals following cytisine treatment (all p>0.05). A modest increase in serum potassium and decrease in calcium levels were noted, though these remained within normal limits. No correlation was found between smoking exposure (pack-years) and baseline Tp-e/QTc.

Conclusions: Cytisine treatment did not adversely affect ECG markers of ventricular repolarization in the short term. Cytisine appears to have a favorable electrophysiological safety profile, supporting its use in smoking cessation. However, further prospective, randomised and long-term studies are warranted to confirm these findings, particularly in patients with pre-existing cardiovascular conditions.

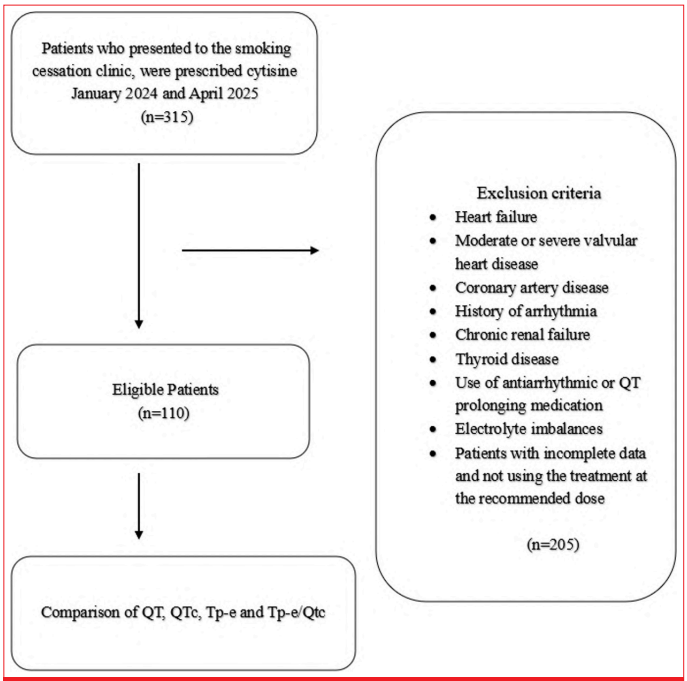

Figure 3. Exclusion criteria.

PP-018 [Hypertension]

The relationship between VAT/SAT ratio and 24-hour ambulatory blood pressure monitoring parameters in newly diagnosed hypertensive patients

Hilal Feyza Filiz[1], Turgut Karabağ[2]

[1]Department of Internal Medicine, İstanbul Education and Research Hospital, İstanbul
[2]Department of Cardiology, İstanbul Education and Research Hospital, İstanbul

Background and Aim: Visceral ve subcutaneous adipose tissue ratio (VAT/SAT) contribute to obesity but may have different metabolic and atherosclerosis risk profiles. VAT/SAT ratio is a correlate of cardiometabolic risk, above and beyond BMI and VAT only. In this study we have investigated the relationship of VAT/

Table 1. Demographic, laboratory, and blood pressure monitoring parameters between the groups

	Group 1 (controls) n=47	Group 2 (newly diagnosed hypertensives) n=82	P
Age (years)	45.4 ± 11.3	44.6 ± 10.2	0.668
Gender (female)	33 (70.2)	33 (40.2)	0.001
BMI (kg/m^2)	28.0 ± 6.2	29.4 ± 5.4	0.110
Waist circumference (cm)	94.81 ± 15	100.19 ± 11.95	0.027
Hip circumference (cm)	107.3 ± 11.7	108.1 ± 14.5	0.273
Smoking (n)	19	41	0.294
History of CAD (n)	18	39	0.308
Hyperlidipemia (n)	5	3	0.139
VAT (mm)	39.8 (29.8-49.5)	49 (38.68-58.08)	0.002
SAT (mm)	16.8 (13.8-23.4)	17.9 (13.98-22.6)	0.534
VAT/SAT	2.16 (1.82-2.78)	2.75 (2.03-3.5)	0.016
SBP (mmHg)	136.15 ± 17.24	157.7 ± 15.06	<0.001
DBP (mmHg)	83.34 ± 12.01	97 ± 11.88	<0.001
Glucose (mg/dL)	96 ± 15.36	95.45 ± 11.71	0.930
Ure (mg/dL)	26.68 ± 6.9	29.55 ± 9.11	0.127
Creatinine (mg/dL)	0.73 ± 0.12	0.81 ± 0.18	0.008
HbA1C (%)	5.51 ± 0.38	5.58 ± 0.4	0.366
HOMA IR	2.73 ± 2.3	3.08 ± 2.44	0.197
Total cholesterol (mg/dL)	204.5 ± 42.19	209.8 ± 43.4	0.497
TG (mg/dL)	144.0 ± 92.3	158.2 ± 99.4	0.225
LDL (mg/dL)	122.9 ± 35.1	129.0 ± 32.8	0.322
HDL (mg/dL)	52.49 ± 14.59	48.38 ± 11.95	0.167
24-h Max SBP (mmHg)	152.9 ± 11.6	184.5 ± 24.1	<0.001
24-h Min SBP (mmHg)	97 ± 9.1	113.6 ± 15.3	<0.001
24-h Mean SBP (mmHg)	122.1 ± 7.9	145.3 ± 12.6	<0.001
24-h Max DBP (mmHg)	104.1 ± 13.4	125.0 ± 24.4	<0.001
24-h Min DBP (mmHg)	53.5 ± 8.1	63.9 ± 12.3	<0.001
24-h Mean DBP (mmHg)	74.9 ± 7.1	90.2 ± 11.5	<0.001

BMI; Body mass index, CAD; Coronary artery disease, VAT; Visceral adipose tissue, SAT; Subcutaneous adipose tissue, HOMA IR; Homeostatic Model Assessment of Insulin Resistance, TG; Triglyceride, LDL; Low denasity lipoprotein, HDL; High denaity lipoprotein, SBP; Systolic blood pressure, DBP; Diastolic blood pressure.

SAT ratio with 24-hour ambulatory blood pressure monitoring in patients with newly diagnosed hypertensive patients.

Methods: Total 129 patients included to the study (82 newly diagnosed hypertensives, 49 male, 33 female; mean age=44.6 ± 14.1 years; 47 controls with no overt disease,14 male, 33 female 45.4 ± 11.3 years). Detailed anamnesis of all participants, height, weight, body mass index, waist and hip circumference, office blood pressure measurement, and 24-hour ambulatory blood pressure measurement were performed and recorded. After 8-12 hours of fasting in both groups, venous blood samples were taken to measure fasting blood sugar, lipid profile, urea, creatinine, glycated hemoglobin, HOMA-IR and CRP, and the resulting laboratory parameters were recorded. Dipper and non-dipper patterns were identified.

Results: Ambulatory blood pressure parameters were significantly higher in patients with hypertensive compared to controls as expected. No significant relationship was detected with VAT/SAT between hypertensive participants with dipper and non-dipper patterns. Seventy patients were nondipper pattern, 59 patients were dipper pattern. A similar and significant relationship was found between the VAT/SAT ratio and 24-hour mean SBP, 24-hour mean DBP and night mean SBP values (r=0.188, r=0.175 and r=0.186, respectively). VAT and VAT/SAT ratios of patients were found to be higher than controls (p=0.002, p=0.016, respectively).

Conclusions: In newly diagnosed hypertension patients, the effect of the ratio of visceral fat tissue thickness to subcutaneous fat tissue thickness on the dipper/non-dipper pattern of hypertension was not determined. VAT/SAT ratio was related with 24-hour mean systolic and diastolic blood pressures.

Table 2. Spearman correlation analysis betweet VAT/SAT ratio and 24 hour anbulatory monitoring parameters

	VAT r	SAT r	VAT/SAT r
24-h Max SBP	0.243**	0.042	0.188*
24-h Min SBP	0.172	-0.056	0.175*
24-h Mean SBP	0.246**	0.008	0.186*
24-h Max DBP	0.188*	-0.103	0.218*
24-h Min DBP	0.228**	0.071	0.164
24-h Mean DBP	0.138	-0.031	0.137

*p<0.05, **p<0.01.

PP-020 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Sex-related differences in patients with atrial fibrillation treated with factor Xa inhibitors

Umut Kocabaş[1], Işıl Ergin[2], Veysel Yavuz[3], Selda Murat[4], İbrahim Halil Özdemir[5], Faruk Aydınyılmaz[6], Mehtap Yeni[7], Halil İbrahim Biter[8], Ümit Yaşar Sinan[9], Veysel Özgür Barış[10], Özcan Vuran[11], Sefa Okar[12]

[1]Department of Cardiology, Başkent University, Faculty of Medicine, İzmir Hospital, İzmir
[2]Department of Public Health, Ege University, Faculty of Medicine, İzmir
[3]Department of Cardiology, Manisa Akhisar Mustafa Kirazoğlu State Hospital, Manisa
[4]Department of Cardiology, Eskişehir Osmangazi University, Faculty of Medicine, Eskişehir
[5]Department of Cardiology, Manisa City Hospital, Manisa
[6]Department of Cardiology, Erzurum Regional Training and Research Hospital, Erzurum
[7]Isparta State Hospital, Isparta
[8]Department of Cardiology, Haseki Training and Research Hospital, İstanbul
[9]Department of Cardiology, İstanbul University, Institute of Cardiology, İstanbul
[10]Department of Cardiology, Gaziantep Dr. Ersin Arslan Training and Research Hospital, Gaziantep
[11]İzmir Kardiya Medical Center, İzmir
[12]Başkent University, Faculty of Medicine, Adana Research and Application Center, Adana

Background and Aim: Real-world data on sex-related differences in patients with atrial fibrillation (AF) are inconsistent. This study aimed to investigate sex-related differences in the clinical characteristics, management, and outcomes of patients with AF treated with factor Xa inhibitors in a real-world context.

Methods: The present study was a national, multicenter, observational study that enrolled patients with AF at 41 cardiology centers between January 2021 and May 2021. The primary outcome was the rate of all-cause mortality, and the secondary outcomes were cardiovascular mortality, ischemic events, bleeding rates, and myocardial infarction rates.

Results: This study included a cohort of 1,159 patients diagnosed with AF and treated with factor Xa inhibitors. The median age of the participants was 72 years, and 59.7% (692 patients) of the cohort were female. Female participants were older, with a higher proportion of patients aged ≥75 years than male participants. Compared with male patients, female patients had lower levels of education and household income. The proportion of non-married, widowed, or divorced individuals was higher in female than in male patients. Male patients were more likely to have coronary artery disease, a history of myocardial infarction, heart failure, dyslipidemia, peripheral artery disease, and chronic kidney disease, whereas female patients were more likely to have diabetes and anemia than male patients. Although the median CHA_2DS_2-VASc

score was higher in female patients, the proportion of patients with a high stroke risk was similar between female and male patients. The history of pharmacological and/or electrical cardioversion was higher in men than in that in women. Although the history of AF ablation was higher in men than in women, the difference was not statistically significant. Concomitant antiplatelet therapy was more frequently used in male than in female patients (Figure 1). Over a median follow-up period of 13 months, 140 patients (12.1%) died during the study period. The all-cause mortality rate was significantly higher in men than in women (15.2% vs. 10.0%, p=0.007). Although the cardiovascular mortality rate was comparable between the sexes, male patients exhibited a higher rate of death from non-cardiovascular causes (8.8% vs. 4.8%, p=0.020). During the follow-up period, the incidence of ischemic events, such as myocardial infarction, stroke, and systemic embolism, was similar between the two groups. Conversely, the rates of major bleeding and intracranial hemorrhage were higher in men than in women (3.4% vs. 1.4%, p=0.025; and 1.9% vs. 0.4, p=0.018, respectively, Figure 2).

Conclusions: The present study provides contemporary data on sex-related differences in the clinical characteristics, management, and outcomes of patients with AF treated with factor Xa inhibitors.

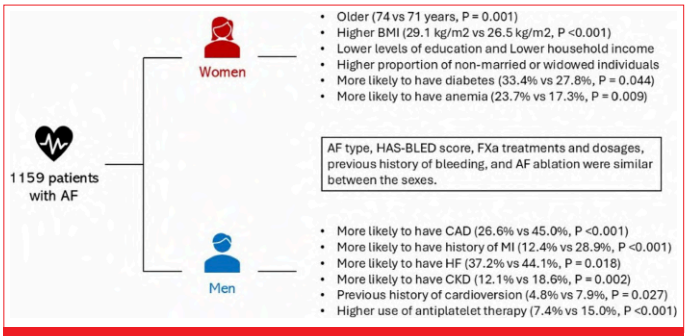

Figure 1.

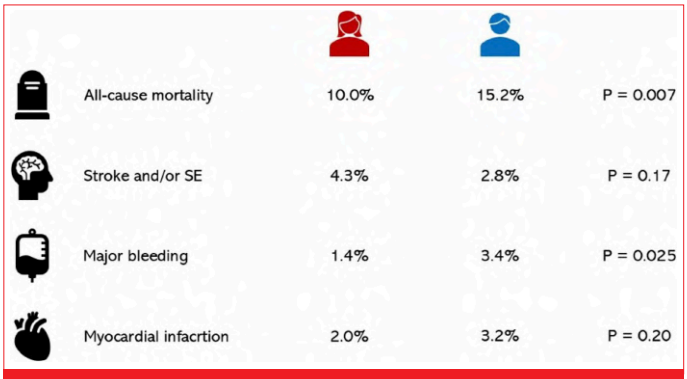

Figure 2.

PP-021 [Interventional Cardiology / Valvular and Structural Heart Disease]

Prognostic value of FIB-4 and FIB-5 scores for in-hospital mortality after transcatheter aortic valve implantation

Alperen Taş[1], Veysel Ozan Tanık[2], Muhammed Erzurum[3], Çağatay Tunca[2], Buse Çuvalcıoğlu[2], Emine Cansu Yücel[2], Bülent Özlek[4]

[1]*Department of Cardiology, Ahi Evran University Training and Research Hospital, Kırşehir*
[2]*Department of Cardiology, Ankara Etlik City Hospital, Ankara*
[3]*Department of Cardiology, Karabük University, Faculty of Medicine, Karabük*
[4]*Department of Cardiology, Muğla Sıtkı Koçman University, Faculty of Medicine, Muğla*

Background and Aim: Risk prediction in transcatheter aortic valve implantation (TAVI) remains challenging, particularly in elderly, frail patients. Conventional surgical scores do not incorporate systemic inflammation or nutritional status. Fibrosis-4 (FIB-4) and Fibrosis-5 (FIB-5) scores, derived from routine laboratory parameters, may reflect these domains, but their prognostic value in TAVI is unclear.

Methods: In this retrospective single-center cohort, 271 patients with severe symptomatic aortic stenosis undergoing transfemoral TAVI (January 2023–March 2025) were analyzed. Patients with advanced liver disease, malignancy, systemic inflammation, or missing data were excluded. FIB-4 and FIB-5 were calculated from admission labs. The primary endpoint was all-cause in-hospital mortality. Multivariable Cox regression identified independent predictors; ROC and Kaplan–Meier analyses assessed discriminative performance and survival.

Results: In-hospital mortality occurred in 29 patients (10.7%). Non-survivors had higher FIB-4 (3.61 vs. 1.95, p<0.001) and lower FIB-5 (–7.57 vs. –1.31, p=0.001) scores. Multivariable analysis identified EuroSCORE II (HR 1.145; p=0.039), vascular complications (HR 1.101; p<0.001), FIB-4 (HR 2.734; p=0.001), and FIB-5 (HR 0.190;

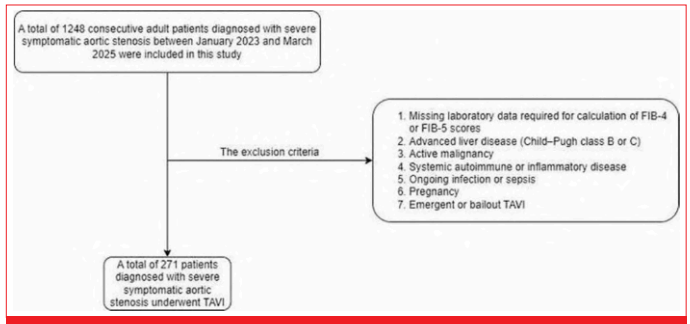

Figure 1. Flowchart of patient selection. Among 1,248 patients with severe symptomatic aortic stenosis, 271 underwent Transcatheter Aortic Valve Implantation (TAVI) after applying exclusion criteria, including missing laboratory data, advanced liver disease, malignancy, systemic inflammation, infection, pregnancy, or emergent/bailout procedures.

p=0.017) as independent mortality predictors. ROC analysis showed AUC 0.735 for FIB-4 (cut-off 2.50) and 0.695 for FIB-5 (cut-off –4.80). Kaplan–Meier curves demonstrated significantly lower survival with higher FIB-4 or lower FIB-5 (both log-rank p<0.001).

Conclusions: FIB-4 and FIB-5 scores, readily obtainable from routine labs, independently predict in- hospital mortality after TAVI, with FIB-4 showing the highest discriminative ability. These indices may complement existing risk models, enhancing preprocedural risk stratification in elderly, high-risk candidates.

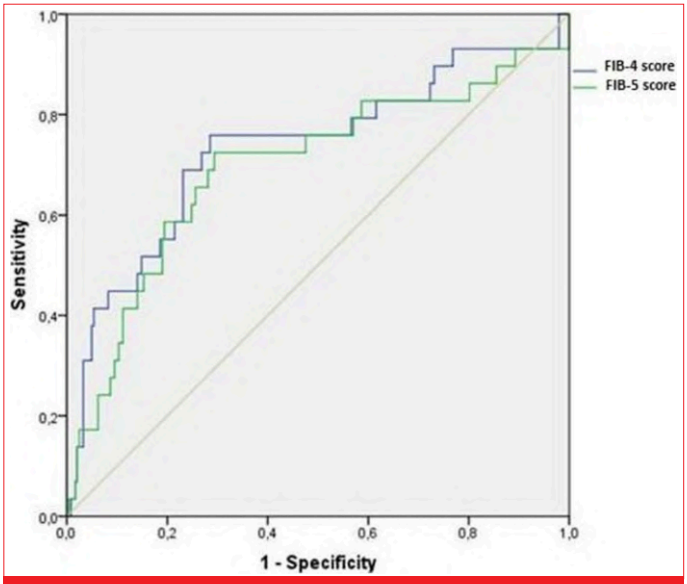

Figure 2. Receiver Operating Characteristic curves for predicting in-hospital all-cause mortality using FIB-4 (blue line) and FIB-5 (green line) scores. The area under the curve was 0.735 for FIB-4 (p<0.001) and 0.695 for FIB-5 (p=0.001), indicating moderate discriminative ability for both indices.

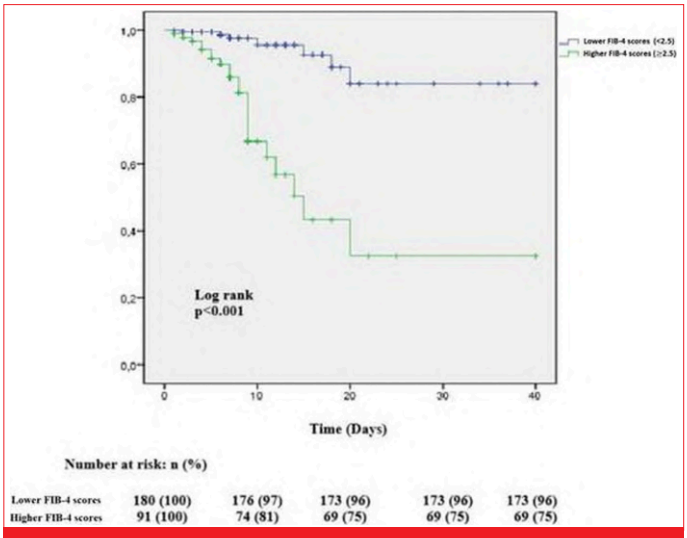

Figure 3. Kaplan–Meier survival curves showing significantly lower survival rates in patients with higher FIB-4 scores (≥2.5) compared to those with lower scores (<2.5) (log-rank p<0.001).

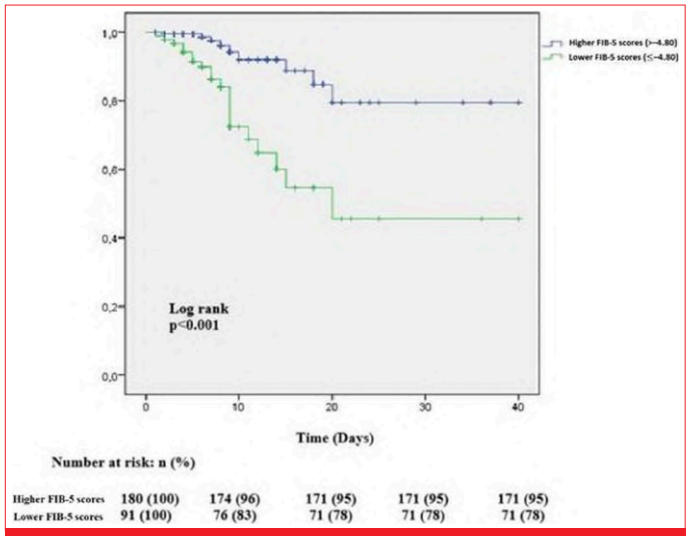

Figure 4. Kaplan–Meier survival curves demonstrating significantly lower survival rates in patients with lower FIB-5 scores (≤4.80) compared to those with higher scores (>4.80) (log rank p<0.001).

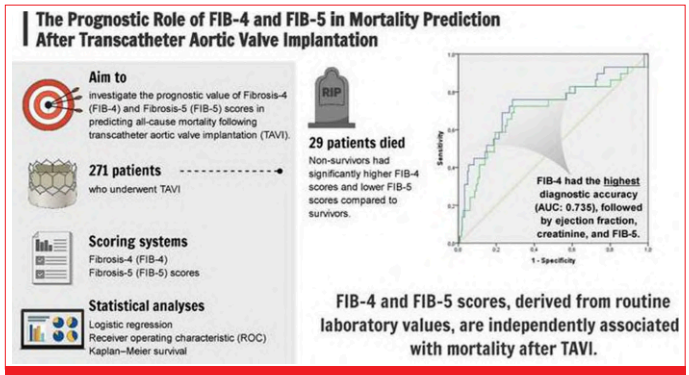

Figure 5.

Table 1. Comparison of clinical, demographic, and laboratory characteristics between deceased and surviving patients

	Survivors (n=242)	Deceased (n=29)	p-value
Age, years	78.5 ± 8.1	87.9 ± 3.1	<0.001
Gender, male, n (%)	120 (49.6)	11 (37.9)	0.235
Comorbidities, n (%)			
Hypertension	197 (81.4)	21 (72.4)	0.249
Diabetes mellitus	95 (39.3)	18 (62.1)	0.019
Atrial fibrillation	56 (23.1)	10 (34.5)	0.197
COPD	24 (9.9)	9 (31.0)	0.001
PCI history	55 (22.7)	6 (20.7)	0.804
CABG history	40 (16.5)	7 (24.1)	0.306
Peripheral artery disease	46 (19.0)	8 (27.6)	0.274
Drug use on admission, n (%)			
ACEi/ARB	150 (62.0)	15 (51.7)	0.285
Beta-blocker	125 (51.7)	15 (51.7)	0.994
Acetylsalicylic acid	114 (47.1)	15 (51.7)	0.638
Statins	81 (33.5)	11 (37.9)	0.632
Anticoagulation	47 (19.4)	6 (20.7)	0.871
Periprocedural data			
LVEF, %	52.6 ± 11.9	44.2 ± 9.3	<0.001
Aortic valve mean gradient, mmHg	47 (35 - 102)	44 (40 - 75)	0.055
sPAP, mmHg	38.6 ± 12.2	46.6 ± 7.5	<0.001
STS score	9.9 (4.4 - 29.7)	13.4 (6.5 - 18.4)	0.021
EuroSCORE II	4.6 (1.7 - 21.2)	7.1 (2.5 - 28.5)	0.001
Valve size, mm	27.0 ± 4.1	28.1 ± 2.8	0.163
Self-expanding valve, n (%)	172 (71.1)	25 (86.2)	0.101
Balloon-expandable valve, n (%)	67 (27.7)	4 (13.8)	0.101
Vascular complication, n (%)	37 (15.3)	15 (51.7)	<0.001
Stroke, n(%)	8 (3.3)	4 (13.8)	0.010
Major bleeding, n (%)	2 (% 0.8)	7 (%24.1)	<0.001
Contrast-induced nephropathy, n (%)	15 (6.2)	12 (41.4)	<0.001
Hemodialysis, n (%)	6 (2.5)	6 (20.7)	<0.001
Complete atrioventricular block, n (%)	32 (13.2)	2 (6.9)	0.298
Length of hospitalization, (days)	6 (1 - 40)	8 (1 - 40)	0.073
Laboratory data			
White blood cells, x10³/mm³	7.3 (2.9 - 18.0)	6.8 (3.8 - 12.5)	0.766
Hemoglobin, g/dL	11.9 (6.9 - 15.8)	10.3 (8.5 - 13.1)	<0.001
Platelets, x10³/mm³	224 (87 - 619)	219 (65 - 460)	0.403
Neutrophils, x10³/mm³	4.5 (1.4 - 8.4)	5.1 (2.5 - 9.8)	0.242
Lymphocytes, x10³/mm³	1.6 (0.4 - 4.9)	1.4 (1.0 - 3.1)	0.804
Monocytes, x10³/mm³	0.61 (0.01 - 1.35)	0.60 (0.20 - 1.12)	0.604
Serum albumin, mg/dL	38 (25 - 49)	39 (21 - 43)	0.750
Blood glucose, mg/dL	118 (70 - 327)	183 (61 - 200)	0.010
Uric acid, mg/dL	6.5 (2.2 -12.8)	7.8 (4.8 - 11.8)	0.002
Urea, mg/dL	53 (14 - 228)	79 (35 - 193)	<0.001
Serum creatinine, mg/dL	1.1 (0.4 - 5.2)	1.8 (0.7 - 11.4)	<0.001
Glomerular filtration rate, mL/min/1. 73m²	58 (7 - 108)	37 (12 - 92)	<0.001
Aspartate aminotransferase, U/L	19 (6 - 87)	22 (11 - 81)	0.051
Alanine aminotransferase, U/L	13 (3 - 34)	12 (6 - 23)	0.148
C-reactive protein, mg/L	6.6 (0.1 – 10.1)	8.0 (0.4 – 11.4)	0.363
Serum sodium, mmol/L	138.9 ± 4.1	138.0 ± 2.5	0.278
Alkaline phosphatase, IU/L	97.3 ± 23.5	63.5 ± 7.9	0.089
Serum potassium, mmol/L	4.5 ± 0.6	4.6 ± 0.5	0.398
HbA1c, %	6.1 ± 1.2	6.9 ± 1.5	0.009
Total cholesterol, mg/dL	161.3 ± 40.6	154.8 ± 38.2	0.448
Triglycerides, mg/dL	115 (49 - 426)	110 (66 - 246)	0.898
LDL-cholesterol, mg/dL	97 (39 - 218)	81 (33 - 164)	0.134
HDL-cholesterol, mg/dL	44 (16 - 170)	45 (21 - 60)	0.434
TSH, µIU/mL	1.7 (0.1 - 18.3)	1.8 (0.5 - 8.4)	0.738
Troponin, ng/mL	22.4 (0.1 – 149.0)	24.2 (0.1 – 275.0)	0.548
NT-pro-BNP, pg/mL	1453 (49 - 32815)	1405 (432 - 7755)	0.624
FIB-4 score	1.95 (0.45 - 9.81)	3.61 (0.77 - 8.06)	<0.001
FIB-5 score	-1.31 ([-26.87] - [+12.66])	-7.57 ([-25.72] - [+17.10])	0.001

Abbreviations: COPD, Chronic Obstructive Pulmonary Disease; PCI, Percutaneous Coronary Intervention; CABG, Coronary Artery Bypass Graft; ACEi, Angiotensin-Converting Enzyme Inhibitor; ARB, Angiotensin II Receptor Blocker; LVEF, Left Ventricular Ejection Fraction; sPAP, Systolic Pulmonary Artery Pressure; STS, Society of Thoracic Surgeons; HbA1c, Hemoglobin A1c; LDL, Low-Density Lipoprotein; HDL, High-Density Lipoprotein; TSH, Thyroid Stimulating Hormone; FIB-4, Fibrosis-4; FIB-5, Fibrosis-5; NT-pro-BNP, N-terminal pro-B-type Natriuretic Peptide.

Table 2. Univariable and multivariable Cox regression analysis for predicting mortality

	Univariable regression		Multivariable regression	
	HR (95% CI)	p-value	HR (95% CI)	p-value
Age	1.284 (1.173-1.405)	<0.001		
Diabetes mellitus	1.395 (1.179-1.873)	0.022		
COPD	3.034 (1.224-7.516)	0.017		
Vascular complication	1.171 (1.076-1.384)	<0.001	1.101 (1.029-1.356)	<0.001
LVEF	0.952 (0.926-0.979)	0.001		
sPAP	1.051 (1.010-1.083)	0.001		
STS score	1.041 (0.971-1.116)	0.254		
EuroSCORE II	1.132 (1.047-1.223)	0.002	1.145 (1.007-1.303)	0.039
Hemoglobin	0.683 (0.552-0.846)	<0.001	0.813 (0.573-1.154)	0.247
Creatinine	2.421 (1.501-3.906)	<0.001		
FIB-4 score	1.591 (1.281-1.976)	<0.001	2.734 (1.544-4.840)	0.001
FIB-5 score	0.917 (0.869-0.967)	0.001	0.190 (0.032-0.372)	0.017

Abbreviations: HR, Hazard Ratio; CI, Confidence Interval; COPD, Chronic Obstructive Pulmonary Disease; LVEF, Left Ventricular Ejection Fraction; sPAP, Systolic Pulmonary Artery Pressure; STS, Society of Thoracic Surgeons; FIB-4, Fibrosis-4; FIB-5, Fibrosis-5.

Table 3. Receiver Operating Characteristic analysis performed to predict mortality

	AUC	p-value	95% Confidence Interval		Cut-off	Sensitivity (%)	Specificity (%)
			Lower Bound	Upper Bound			
Age	0.711	<0.001	0.615	0.806	81.50	65	65
EuroSCORE II	0.681	0.001	0.579	0.784	5.35	65	63
Creatinine	0.721	<0.001	0.619	0.822	1.23	69	68
LVEF	0.726	<0.001	0.637	0.814	49	70	71
FIB-4 score	0.735	<0.001	0.622	0.848	2.50	72	73
FIB-5 score	0.695	0.001	0.577	0.813	-4.80	69	70

Abbreviations: AUC, The Area Under the Curve; LVEF, Left Ventricular Ejection Fraction; FIB-4, Fibrosis-4; FIB-5, Fibrosis-5.

PP-023 [Heart Failure]

A new ratio for the assessment of right heart function in heart failure: Correlation between mean TR gradient/ IVC diameter and urinary sodium

Volkan Kozluca, Dilşah Sevik, Gözde Cansu Yılmaz, Nail Burak Özbeyaz, İrem Dinçer

Department of Cardiology, Ankara University, Faculty of Medicine, Ankara

Background and Aim: Right heart function is crucial for evaluating congestion and predicting diuretic response in heart failure (HF). Conventional echocardiographic parameters, such as tricuspid annular plane systolic excursion (TAPSE), right ventricular outflow tract velocity-time integral (RVOT VTI), and right atrial pressure (RAP), are commonly used to assess right ventricular (RV) performance. The Pulmonary Artery Pulsatility Index (PAPI), measured invasively, reflects RV–arterial coupling by integrating pressure and flow. Our proposed ratio—Mean TR Gradient / Maximum IVC Diameter—was designed as a non-invasive surrogate to the physiological concept of PAPI, combining an estimate of RV systolic pressure with a marker of venous return and preload. We evaluate this novel parameter and its correlation with spot urinary sodium levels.

Methods: This single-center, observational study enrolled 45 patients with decompensated HF. The following parameters were measured: mean tricuspid regurgitation (TR) gradient (mmHg), maximum inferior vena cava (IVC) diameter (mm), and spot urinary sodium concentration (mmol/L). A novel ratio was calculated by dividing the mean TR gradient by the IVC diameter (Mean TR Gradient / IVC). A urinary sodium threshold of 50 mmol/L was used, and receiver operating characteristic (ROC) analysis (Figure 1) was performed to evaluate the predictive performance of the new ratio. The mean age was 69.2 ± 11.5 years; 33.3% were female (n=15) and 66.7% were male (n=30). The mean left ventricular ejection fraction was 35.8 ± 18.7%, mean estimated glomerular filtration rate (eGFR) was 49.0 ± 27.5 mL/min/1.73 m², and the median BNP level was 9,513 pg/mL (Table 2).

Results: A significant positive correlation was observed between the mean TR gradient/IVC ratio and spot urinary sodium levels (r=0.55, p<0.001; Figure 4). ROC analysis yielded an AUC of 0.78

(Figure 1), with an optimal cut-off value of 1.02, demonstrating 80% sensitivity and 75% specificity. TR gradient values (Figure 2) and TR gradient/IVC ratios (Figure 4) were significantly higher in patients with urinary sodium ≥50 mmol/L compared to those with <50 mmol/L ($p<0.05$), whereas IVC diameters did not differ significantly (Figure 3). Echocardiographic and laboratory comparisons are presented in Table 1.

Conclusions: Non-invasive assessment of right ventricular function and preload is crucial in the management of heart failure. In our study, we proposed the TY gradient/IVC ratio as a novel parameter, which,similar to the invasive Pulmonary Artery Pulsatility Index, demonstrated significant predictive value for spot urine sodium levels. This approach offers a low-cost, echocardiography-based, non-invasive alternative for clinical evaluation, potentially aiding in the assessment of congestion and guiding diuretic therapy. Nevertheless, further studies involving larger patient cohorts,serial measurements,and comprehensive evaluation of its impact on diuretic response are warranted to validate its clinical utility.

Table 1. Comparison of echocardiographic and laboratory parameters according to urinary sodium levels

Parameter	Urinary Na <50 mmol/L Mean ± SD	Urinary Na ≥50 mmol/L Mean ± SD	p value
TR Gradient (mmHg)	21.1 ± 7.9	26.9 ± 6.3	0.015
RVOT VTI (cm)	12.6 ± 4.4	12.5 ± 3.4	0.969
TAPSE (cm)	1.50 ± 0.43	1.40 ± 0.30	0.424
PAB (mmHg)	46.8 ± 9.4	53.6 ± 10.4	0.041
Peak TR Velocity (m/s)	2.8 ± 0.5	3.1 ± 0.4	0.052
Peak TR Gradient (mmHg)	32.5 ± 10.7	39.5 ± 11.0	0.052
IVC Diameter (cm)	2.5 ± 0.5	2.3 ± 0.4	0.220
Urinary Sodium (mmol/L)	31.3 ± 12.8	92.3 ± 26.5	<0.001
TR Gradient / IVC Diameter	0.8 ± 0.3	1.2 ± 0.3	0.006
TAPSE / PAB	0.20 ± 0.10	0.20 ± 0.12	0.557

Table 2. Patient characteristics according to spot urinary sodium levels

Parameter	Urinary Na <50 mmol/L	Urinary Na ≥50 mmol/L
EF (Mean ± SD)	26.3 ± 17.3	40.5 ± 17.8
Age (Mean ± SD)	65.5 ± 11.7	71.0 ± 11.2
Women, n (%)	0 (0.0%)	15 (50.0%)
Man, n (%)	15 (100.0%)	15 (50.0%)
GFR (Mean ± SD)	55.2 ± 28.4	46.0 ± 27.1
BNP (Median)	9513.0	9355.5

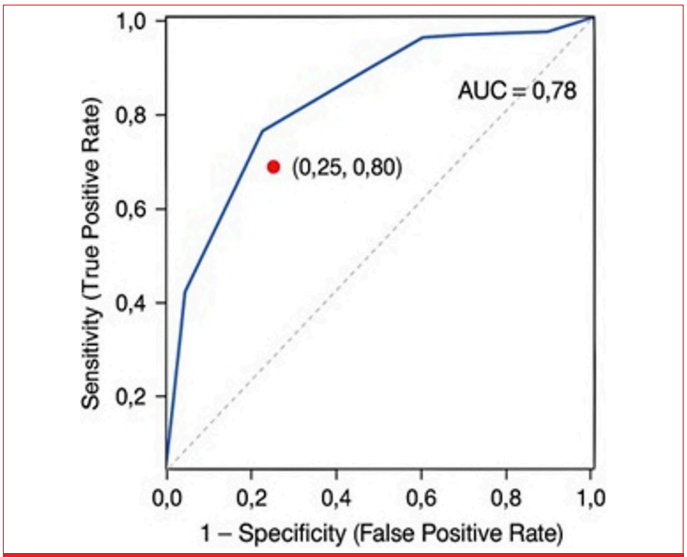

Figure 1. ROC analysis of the mean TR gradient / IVC diameter ratio for predicting urinary sodium levels (<50 mmol/L) (AUC=0.78). The optimal cutoff value was determined with 80% sensitivity and 75% specificity.

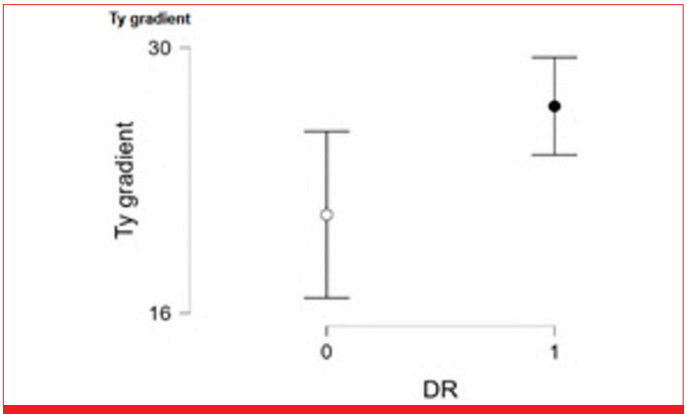

Figure 2. Comparison of tricuspid regurgitation (TR) gradient values between patients with urinary sodium <50 mmol/L (Group 0) and ≥50 mmol/L (Group 1), presented as means with error bars.

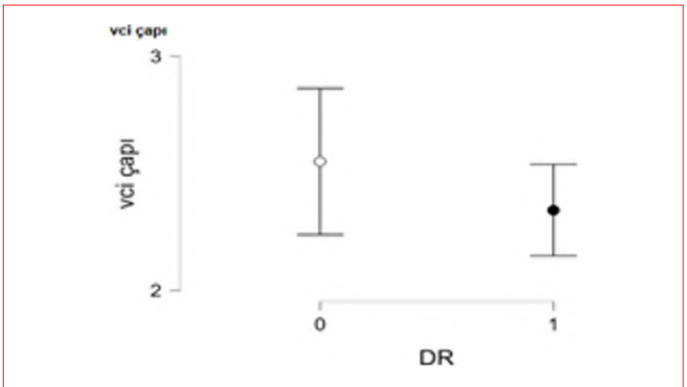

Figure 3. Comparison of IVC diameter values between patients with urinary sodium <50 mmol/L (Group 0) and ≥50 mmol/L (Group 1), presented as means with error bars.

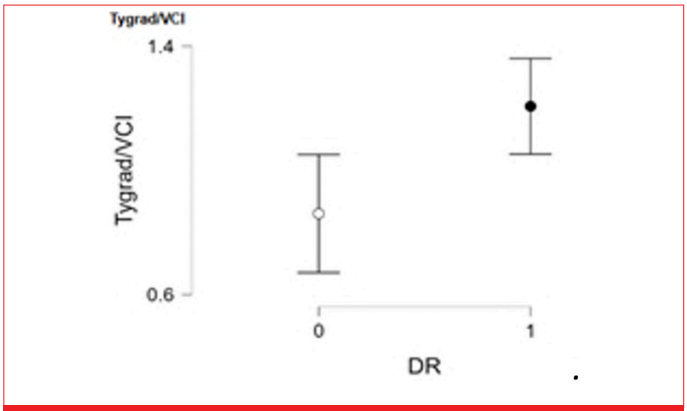

Figure 4. Comparison TR Gradient / IVC Diameter values between patients with urinary sodium <50 mmol/L (Group 0) and ≥50 mmol/L (Group 1), presented as means with error bars.

PP-024 [Interventional Cardiology / Valvular and Structural Heart Disease]

Peripheral acute hemodynamic success criteria in mitraClip: Arterial wave ejection time evaluation

Erdoğan İlkay[1], Ersin Sarıçam[1], Alev Kılıçgedik[2], Gazi Akkurt[1], Zeynettin Kaya[3]

[1]*Department of Cardiology, Medicana International Ankara Hospital, Ankara*
[2]*İstanbul Başakşehir Çam and Sakura City Hospital, İstanbul*
[3]*Department of Cardiology, Private Foundation (ASV) Life Hospital, Antalya*

Background and Aim: Moderate-to-severe secondary mitral regurgitation (MR) has been associated with a poor prognosis in chronic HFrEF patients. According to COAPT trial, secondary mitral regurgitation correction with MitraClip confirmed a significant reduction in hospitalization and all-cause mortality compared with effective medical therapy alone. Currently practically, the success of mitraClip has commonly been evaluated via two criteria. First is to obtain normalized pulmonary vein Doppler findings, second method is measurement of the height of the V-wave before the procedure and compare it with the pressure curves after implantation of the MitraClip. Left ventricular ejection time (LVET) is a cardiovascular marker, which characterizes ventricular performance parameters. Invasive arterial blood pressure (ABP) tracing composed of the tidal wave (TDW) and the dicrotic wave (DCW) (Figure 1). The objective of this study was to compare with the changes of pulmonary vein Doppler findings and radial arterial wave ejection time measurements in the patients underwent mitraClip.

Methods: We studied symptomatic 15 patients underwent mitraClip. The mean age was 70 ± 0.8 years and 8 patients (53%) were male. All patients had symptomatic with moderate to severe (3-4) MR and abnormal LV systolic function (left ventricular ejection fraction (LVEF, <30). The procedure was performed under general anesthesia using transesophageal echocardiography guidance and fluoroscopy. Vascular access was established from the femoral vein. The MitraClip device was advanced following an echocardiography-guided transseptal approach to the LA and across the MV to the LV. We obtained all patients pulmonary vein Doppler changes before and after mitraClip procedure. Mean while, arterial pressures were measured from the radial artery. Transducers were balanced by determining zero level at the mid-axillary line. Patients were compared pulmonary vein Doppler findings with LVET calculation by obtaining radial artery patterns.

Results: We analyzed pulmonary Doppler findings and LVET by radial artery waveform features before and after mitraClip in 15 patients (Table). We obtained arterial traces during mitraClip procedure. While we monitored radial artery traces during clip closing time and realized longer LVET on time closing clip (Figure 2). We realized that longer LVET levels were associated with normalized pulmonary vein Doppler findings. There was significant association between LVET measurements with invasive hemodynamics and pulmonary vein Doppler changes.

Conclusions: We observed that longer arterial ET is associated with normalized pulmonary vein findings. We think that longer LVET could be acute peripheral success criteria in mitraClip. The evaluation of radial artery waveform could be effective as a peripheral predictor marker of successful Mitraclip in assessing MR.

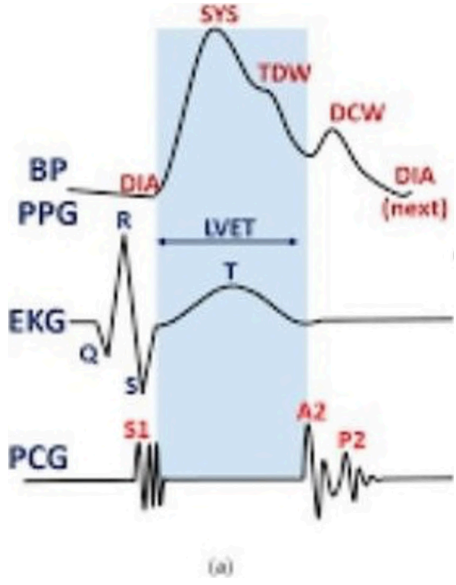

Figure 1. Left ventricular ejection time (LVET) measurement.

Table 1. Comparison of LVET and pulmonary vein Doppler changes in patients before and after MitraClip treatment

	Before mitraClip	After mitraClip	p
Pulmonary vein features	S<D	S>D	
LVET by radial artery	0.16 ± 0.02 ms	0.28 ± 0.03 ms	<0.05

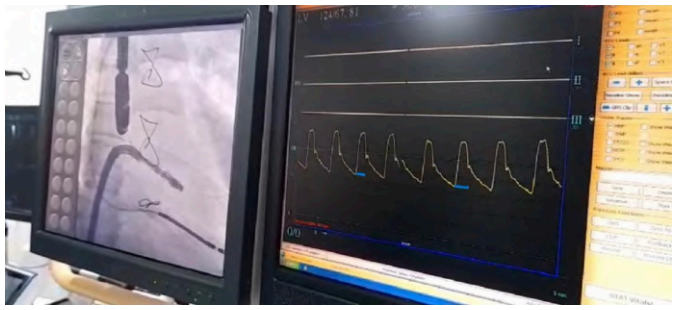

Figure 2. Longer LVET on time closing clip in radial artery trace. Short blue line, LVET; before closure of clip longer blue line, LVET; after closure of clip.

PP-025 [Cardiac Imaging / Echocardiography]

A rare case of pneumohydropericardium caused by Streptococcus mitis/oralis in a patient with prior esophageal cancer

Muhammed Ali Şahin[1], Gökçe Softaoğlu[1], İrfan Özgen[1], Mustafa Ozan Gürsoy[1], Ali Kemal Çabuk[2], Emine Güner[1]

[1]*Department of Cardiology, İzmir Health Sciences University Tepecik Training and Research Hospital, İzmir*
[2]*Department of Cardiology, Izmir City Hospital, İzmir*

Background and Aim: Pneumohydropericardium, characterized by the simultaneous accumulation of air and fluid in the pericardial cavity, is an extremely rare clinical entity that may arise secondary to trauma, invasive procedures, esophageal perforation, or infections. In malignancy-related cases, fistula formation between the esophagus and pericardium may facilitate the introduction of both air and infected material. We present a rare case of pneumohydropericardium associated with Streptococcus mitis/oralis infection in a patient with a history of esophageal cancer and longstanding dysphagia requiring liquid diet.

Methods: A 78-year-old female with a known history of esophageal cancer treated with chemoradiotherapy one year prior presented with dyspnea, fever, and palpitations. Physical examination revealed hypotension, tachycardia, and muffled heart sounds. Chest X-ray and thoracic CT revealed a pericardial air-fluid level. Transthoracic echocardiography (TTE) demonstrated pericardial effusion

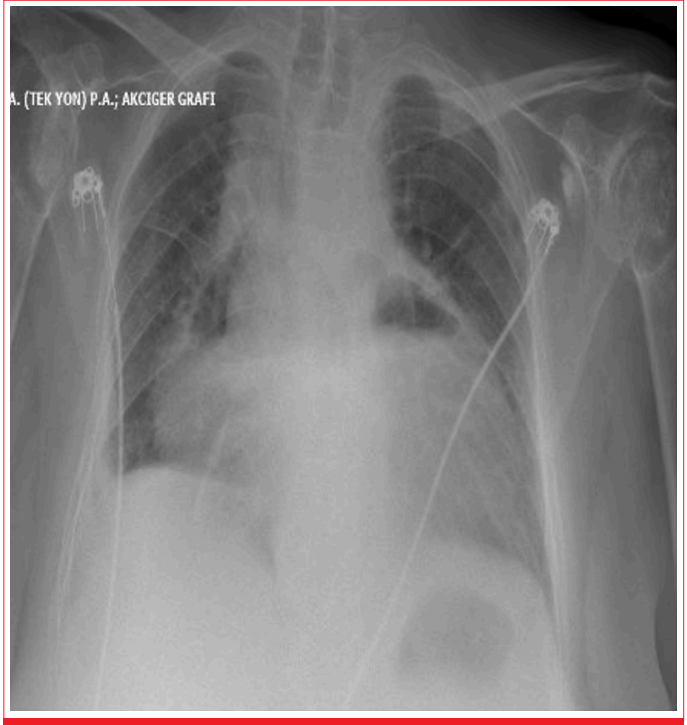

Figure 1. Chest X-ray demonstrating pericardial air-fluid level. The posteroanterior chest radiograph shows a prominent air-fluid level surrounding the heart

Figure 2. Pericardiocentesis procedure and drainage of serous fluid with air bubbles. The image captured during pericardiocentesis demonstrates the aspiration of serous pericardial fluid containing visible air bubbles using a syringe.

compressing the right ventricle, consistent with cardiac tamponade. Emergent pericardiocentesis was performed, yielding 350 mL of serous fluid with visible air bubbles. Microbiological and biochemical analysis of the fluid was conducted.

Results: Pericardial fluid analysis revealed an exudative profile (LDH: 1311 U/L, glucose: <1 mg/dL, albumin: 2.6 g/dL) and leukocytosis with polymorphonuclear predominance. Gram stain showed abundant leukocytes. S. mitis/oralis was isolated on culture, although no growth was seen in blood cultures. Despite the absence of dental or oropharyngeal pathology, the presence of S. mitis/oralis and prior history of esophageal malignancy raised suspicion of an esophagopericardial fistula. Endoscopy did not reveal a visible fistulous tract. The patient was treated empirically with imipenem due to the critical clinical condition, presumed gastrointestinal flora involvement, and known activity of carbapenems against viridans streptococci. The patient responded clinically and completed a 14-day antibiotic course with full recovery.

Conclusions: This case highlights pneumohydropericardium as a rare but life-threatening complication in patients with prior esophageal malignancy. Infectious causes, particularly from gastrointestinal flora, should be considered even in the absence of direct fistula visualization. Prompt recognition, pericardial drainage, and appropriate empiric antimicrobial therapy are critical for favorable outcomes. Reporting such rare cases contributes to raising clinical awareness and may aid in early identification in similar scenarios.

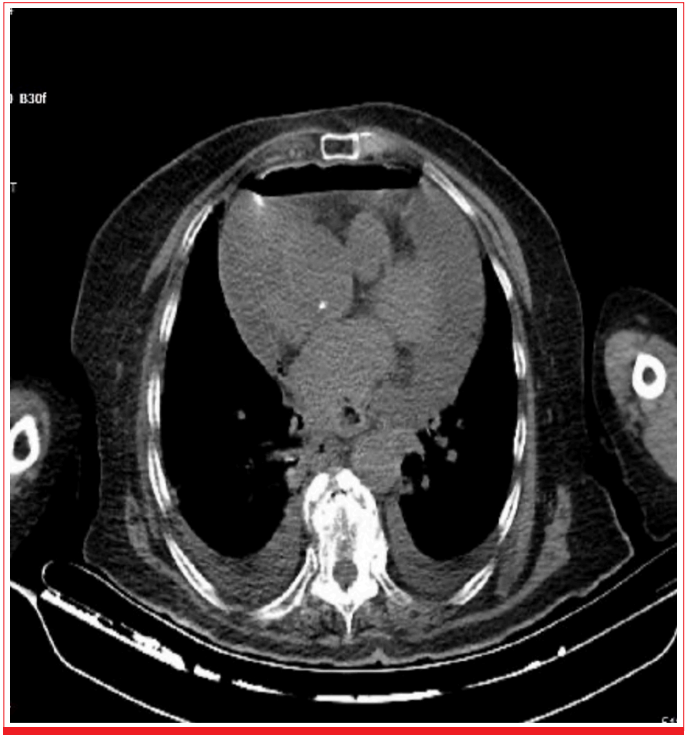

Figure 3. Thoracic CT showing pneumohydropericardium. Axial thoracic computed tomography (CT) image reveals air and fluid accumulation within the pericardial cavity, consistent with pneumohydropericardium. The presence of both hypodense (air) and hyperdense (fluid) areas encasing the heart is clearly visible, supporting the diagnosis in the appropriate clinical context.

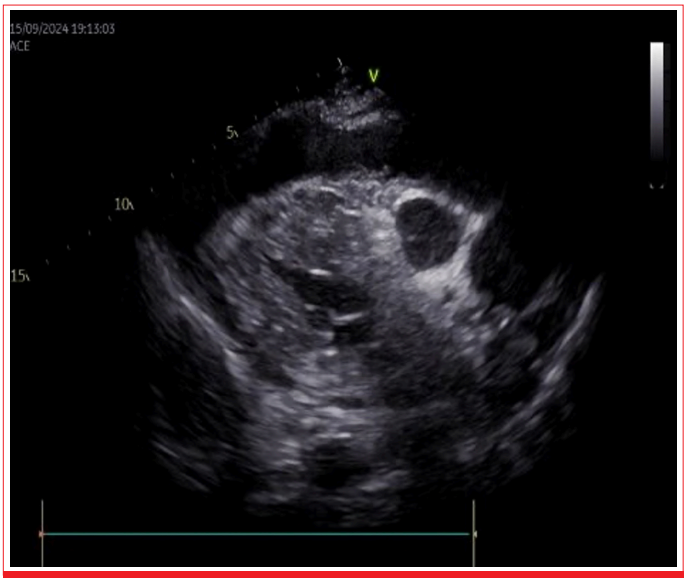

Figure 4. Transthoracic echocardiography demonstrating pericardial effusion with air. Transthoracic echocardiographic image (subcostal view) showing a large circumferential pericardial effusion surrounding the heart. The irregular echogenicity and echolucent areas suggest the presence of both fluid and intrapericardial air, consistent with pneumohydropericardium.

PP-028 [Cardiac Imaging / Echocardiography]

The impact of mitral annular calcification on left atrial elasticity: The role of left atrial stiffness as a diagnostic marker

Muhammed Ali Şahin[1], İrfan Özgen[1], Utku Zeybey[1], Ali Kemal Çabuk[2], Mustafa Ozan Gürsoy[1], Dilara Zorlu Demir[1]

[1]*Department of Cardiology, İzmir Health Sciences University Tepecik Training and Research Hospital, İzmir*
[2]*Department of Cardiology, İzmir City Hospital, İzmir*

Background and Aim: Mitral annular calcification (MAC) is a degenerative process involving the fibrous support structure of the mitral valve and has been increasingly recognized for its association with adverse cardiovascular outcomes. Left atrial stiffness (LAS), reflecting atrial compliance and remodeling, has emerged as a novel echocardiographic marker of subclinical atrial dysfunction. This study aimed to investigate the effect of MAC on left atrial elasticity and to evaluate the relationship between MAC severity and LAS.

Methods: This prospective, single-center study included a total of 112 subjects who underwent transthoracic echocardiographic evaluation. Participants were divided into two groups: 58 patients with echocardiographically confirmed MAC and 54 age- and sex-matched controls without MAC. Echocardiographic measurements included left atrial reservoir strain (LASr), LAS (calculated as E/e′ divided by LASr), left atrial volume index (LAVI), and E/e′ ratio. MAC severity was graded as mild, moderate, or severe. Correlation and multivariate regression analyses were performed to determine independent predictors of LAS. Receiver operating characteristic

(ROC) curve analysis was used to assess the diagnostic performance of LAS in identifying MAC.

Results: LAS was significantly higher in the MAC group compared to controls (0.57 ± 0.30 vs. 0.34 ± 0.20, p<0.001). LAS values increased in parallel with MAC severity (median LAS: no MAC=41.3; mild=59.9; moderate=81.9; severe=94.8; p<0.001). LAS showed a strong negative correlation with LASr (r=–0.750, p<0.001), and positive correlations with LAVI (r=0.501, p<0.001), E/e′ (r=0.793, p<0.001), and age (r=0.371, p<0.001). In multivariate regression, independent predictors of LAS included LASr (β=–0.502, p<0.001), LAVI (β=0.088, p=0.025), and E/e′ (β=0.586, p<0.001). ROC analysis revealed that LAS had a good diagnostic accuracy in distinguishing MAC (AUC=0.762, 95% CI: 0.674–0.850).

Conclusions: MAC significantly impairs left atrial elasticity, as evidenced by elevated LAS values. LAS was markedly higher in the MAC group compared to controls and showed a progressive increase with greater MAC severity. LAS was independently associated with LASr, LAVI, and E/e′, underscoring its complex interplay with atrial mechanics and diastolic function. As a noninvasive and integrative index, LAS may serve as a promising biomarker for early detection of subclinical atrial dysfunction in patients with MAC.

PP-029 [Other]

Impact of preoperative cardiac evaluation and recommendations on morbidity and mortality in patients undergoing elective non-cardiac surgery a retrospective cohort study

Süha Asal[1], Şahhan Kılıç[1], Emrecan Uygun[2], Mert Babaoğlu[2], Mustafa İnceoğlu[2], Samet Yavuz[2], Yetkin Korkmaz[2], Tufan Çınar[3], Ahmet Lütfullah Orhan[2]

[1]Department of Cardiology, Çorlu State Hospital, Tekirdağ
[2]Department of Cardiology, Sultan Abdülhamid Han Hospital, İstanbul
[3]University of Maryland Midtown Campus, Department of Medicine, USA

Background and Aim: Preoperative cardiac evaluation is routinely performed to reduce cardiovascular risk in non-cardiac surgery. However, the benefit of routine cardiology consultations remains uncertain and may increase healthcare burden without improving outcomes. This study aims to evaluate the impact of such consultations on morbidity and mortality in patients undergoing non-cardiac surgery between March and September 2021, assess the effectiveness of risk stratification strategies, and identify potentially unnecessary consultations.

Methods: This retrospective cohort study included 482 patients referred to a cardiology outpatient clinic for preoperative cardiac evaluation before non-cardiac surgery from March 2021 to September 2021. Data collected included demographics, cardiovascular risk factors, functional capacity (metabolic equivalents [METs]), electrocardiography (ECG), echocardiography, preoperative recommendations, new cardiac diagnoses, postoperative cardiac complications, hospital stay and mortality. Statistical analysis involved chi-square, Mann-Whitney U, and multivariable logistic regression (significance set at p<0.05).

Table 1. Demographic and clinical data

Preoperative patient data	n=482
Age(year) median(IQR)	69 (60-76)
Sex	
Female	163 (%33,8)
Male	319 (%66,2)
Type of Surgery	
Bronchoscopy/EBUS	30 (%6,2)
Endoscopy/Colonoscopy	153 (%31,7)
Genitourinary surgery	93 (%19,3)
Cholecystectomy/gastrointestinal surgery	78 (%16,2)
Orthopedic surgery (hip fracture)	23 (%4,8)
CNS surgery	27 (%5,6)
Thoracic surgery	16 (%3,3)
Cataract, ophthalmic surgeries	42 (%8,7)
Oral and nasal cavity procedures	20 (%4,1)
Risk Factors	
Coronary artery disease	235 (%48,8)
Nonobstructive coronary artery disease	71 (%14,8)
Percutaneous coronary intervention	94 (%19,5)
CABG	60 (%12,4)
CABG+ Percutaneous coronary intervention	10 (%2,1)
Diabetes mellitus	182 (%37,8)
Insülin	25 (%13,7)
Hypertension	236 (%48,9)
Cerebrovascular disease	18 (%3,7)
Chronic renal failure	10 (%2,1)
Hyperlipidemia	23 (%4,8)
Smoking	43 (%8,9)
Cancer	4 (%0,8)
Family history	1 (%0,2)
Previous myocardial infarction	15 (%3,1)
Medical Treatment	
ASA	222 (%46)
ADP receptor inhibitor	91 (%18,9)
Warfarin	24 (%5)
NOAC	42 (%8,7)
Beta blocker	190 (%39,4)
Calcium channel blocker	51 (%10,6)
ACE-inhibitor / ARB	144 (%29,9)
Mineralocorticoid receptor antagonist	15 (%3,1)
Diuretic	12 (%2,5)
SGLT-2 inhibitor	7 (%1,5)
Statin	132 (%27,4)
Ranolazin	9 (%1,9)
Trimetazidin	5 (%1)
Nitrate	10 (%2,1)

Abbreviations: IQR: interquartile range, EBUS: endobronchial ultrasound, CNS: central nervous system, CABG: coronary arter bypass greft, ASA: acetylsalicylic acid, ADP: adenosine diphosphate, NOAC: non–vitamin K antagonist oral anticoagulants, ACE: angiotensin converting enzyme, ARB: angiotensin receptor blocker, SGLT-2: sodium glucose cotransporter-2

Results: The median age was 69 years (IQR: 60-76), with 66.2% male patients. The most prevalent risk factors were coronary artery disease (48.8%), hypertension (48.9%), and diabetes mellitus (37.8%). Most patients (96.5%) were asymptomatic, with 79.9% having a functional capacity of 4-10 METs and 8.1% <4 METs. ECG revealed sinus rhythm in 88.8% and atrial fibrillation/flutter in 11.2%; echocardiography showed EF >50% in 88.1%. Preoperative recommendations included discontinuation of warfarin/NOACs (10%), antiplatelets (31.1%), surgery deferral (3.1%), and initiation of statins (0.8%); however, 54.8% of consultations resulted in no additional recommendations. New cardiac diagnoses were made in 3.7% of cases (coronary artery disease 1.4%, heart failure 0.8%, atrial fibrillation 0.6%). Postoperative cardiac complications occurred in 2.9% (n=14), with in-hospital mortality of 0.6% (n=3) and 1-year mortality of 1.7% (n=8). Low functional capacity (<4 METs) was associated with increased complication risk (OR: 3.4; 95% CI: 1.1-10.5; p=0.04). Complication rates were similar regardless of whether cardiology recommendations were made (p=0.78).

Conclusions: Preoperative cardiac evaluations led to new diagnoses or management changes in few cases, with most consultations in low-risk patients likely unnecessary. Low complication and mortality rates support the efficacy of risk-based approaches per ACC/AHA and ESC/ESAIC guidelines. Enhanced use of risk stratification tools, such as the Revised Cardiac Risk Index (RCRI), could reduce unnecessary consultations, alleviate cardiology clinic workload, and

optimize healthcare resource allocation. These findings underscore the need for revising and standardizing perioperative risk assessment protocols, with a recommendation for other specialties to implement risk stratification prior to requesting cardiology consultations.

Table 2. Cardiac evaluation and recommendation

Preoperative patient data	n=482
Are there any cardiac symptoms?	
Dyspnea	11 (%2,3)
Chest pain	3 (%0,6)
Weakness	1 (%0,2)
Swollen feet	2 (%0,4)
No active symptoms	465 (%96,5)
Functional capacity	
<4 Mets	39 (%8,1)
4-10 Mets	385 (%79,9)
>10 Mets	58 (%12)
Electrocardiography	
Sinus rhythm	428 (%88,8)
Atrial fibrillation/flutter	54 (%11,2)
Ejection fraction on echocardiography	
EF <%40	21 (%4,4)
EF %40-50	36 (%7,5)
EF>%50	425 (%88,1)
Valvular pathology	
AVR	5 (%1)
MVR	1 (%0,2)
Bioprosthetic valve	4 (%0,8)
Moderate or severe mitral stenosis	3 (%0,6)
Moderate or severe aortic stenosis	13 (%2,7)
Moderate or severe mitral regurgitation	3 (%0,6)
Moderate or severe aortic insufficiency	5 (%1)
Pre-operative advice	
Warfarin or NOAC discontinuation	48 (%10)
ASA or ADP receptor inhibitor discontinuation	150 (%31,1)
Blood pressure regulation	2 (%0,4)
Heart failure medication regulation	3 (%0,6)
Statin initiation	4 (%0,8)
Lifestyle change	0 (%0)
Surgical delay	15 (%3,1)
No additional recommendations	264 (%54,8)
Was a new cardiac diagnosis made?	
Coronary artery disease	7 (%1,4)
Heart failure	4 (%0,8)
Moderate or severe valve disease	1 (%0,2)
Hypertension	3 (%0,6)
Atrial fibrillation/flutter	3 (%0,6)
Other arrhythmias	2 (%0,4)
No new diagnoses	464 (%96,3)
Did any cardiac complications occur post-procedure? Was a cardiology reconsultation required? (Yes)	14 (%2,9)
Length of hospital stay (days) median (IQR)	1 (0-3)
Was there any periprocedural or in-hospital mortality? (Yes)	3 (%0,6)
Was there any mortality within the past year? (Yes)	8 (%1,7)

Abbrevations: Mets: metabolic equivalents, EF: ejection fraction, AVR: aortic valve replacement, MVR: mitral valve replacement, NOAC: non-vitamin K antagonist oral anticoagulants, ASA: acetylsalicylic acid, ADP: adenosine diphosphate,

PP-031 [Interventional Cardiology / Coronary]

Comparison of the noncompliant balloon and stent balloon double inflation postdilatation techniques

Uygur Şimşek, Kurtuluş Karaüzüm, Didar Mirzamidinov, Ebrar Gencer, Beyza Kalaş, Hacer Doğan, Mehmet Kadir Ceylan, Salah Abdo, İrem Yılmaz, Tayfun Şahin, Ertan Ural, Göksel Kahraman

Department of Cardiology, Kocaeli University, Faculty of Medicine, Kocaeli

Background and Aim: Post-dilatation after stent implantation is an almost routine step of percutaneous coronary interventions (PCI) and this procedure is typically performed with non-compliant (NC) balloons. The present study aims to compare the conventional post-dilatation strategy using a NC balloon with an alternative approach that utilizes the stent balloon through a double inflation technique during PCI.

Methods: A total of 58 chronic coronary syndrome (CCS) patients were included in this prospective, randomised and single-centre study between October 2023 and May 2024. Patients were randomised in a 1:1 fashion to recieve either NC post-dilatation or stent balloon double inflation post-dilatation after stent implantation in the non-small (stent size ≥3 mm), non-diffuse (stent length ≤18 mm) and non-calcific lesions. The primary outcome was the composite of major cardiac events (MACE) at the 1-year follow-up, including cardiac death, target vessel-related myocardial infarction (TVMI) or clinically driven target lesion revascularization (TLR). The secondary outcomes were radiation exposure, contrast-induced nephropathy (CIN), total procedure time, and procedural complications including vessel perforation, stent deformation, stent edge dissection or distal TIMI ≤2 coronary flow.

Results: During the 1-year follow-up, MACE rates were statistically similar between the NC balloon post-dilatation and stent balloon double inflation post-dilatation groups (p=0.754). Among secondary outcomes, radiation exposure and total procedure time were significantly lower in the stent balloon group patients than those NC balloon group (759.2 ± 450.8 mGy vs. 931.3 ± 495.4 mGy; p=0.034 and 18.7 ± 13.2 min vs. 25.7 ± 14.5 min; p=0.024, respectively). Procedural complications and CIN did not show statistical difference in the two groups.

Conclusions: The stent balloon double inflation post-dilatation strategy shows similar clinical outcomes compared with the conventional NC balloon post-dilatation technique in CCS patients undergoing non-complex PCI, while promising secondary benefits by reducing total procedure time and radiation exposure. Additionally, by eliminating the need for an extra balloon, it provides a cost-effective alternative.

PP-032 [Interventional Cardiology / Valvular and Structural Heart Disease]

Prognostic value of the CALLY index for predicting mortality in patients undergoing transcatheter aortic valve implantation (TAVI)

Muhammet Ali Ekiz[1], Muhammed Musa Yıldız[1], Bayram Ali Kılınç[1], Ali Bağcı[1], Mustafa Karabacak[1], Şakir Arslan[2], Fatih Aksoy[1]

[1]Department of Cardiology, Süleyman Demirel University, Faculty of Medicine, Isparta
[2]Department of Cardiology, Antalya Education and Research Hospital, Antalya

Background and Aim: Aortic stenosis (AS) is one of the most common valvular heart diseases associated with high morbidity and mortality. Transcatheter aortic valve implantation (TAVI) has become the standard of care in high-risk patients; however, accurate prediction of postprocedural mortality remains a challenge. The C-reactive protein–albumin–lymphocyte (CALLY) index is a novel marker integrating inflammation, nutritional status, and immune competence. This study aimed to evaluate the prognostic value of the CALLY index in predicting 2-year mortality after TAVI.

Methods: This retrospective study included 123 patients (60 males, 63 females) who underwent TAVI between February 2023 and April 2025. Preprocedural CRP, albumin, and lymphocyte levels were obtained, and the CALLY index was calculated as (Albumin × Lymphocyte) / CRP). Patients were divided into survivor and non-survivor groups based on 2-year mortality. Laboratory, echocardiographic, and clinical variables were compared (Table 1, Table 2, Table 3). Multivariate logistic regression identified independent predictors (Table 4). ROC analysis and pairwise comparisons assessed discriminative performance (Table 5, Table 6, Figure 1). Kaplan–Meier curves evaluated survival differences (Figure 2).

Results: The CALLY index was significantly lower in non-survivors compared to survivors (0.76 ± 1.06 vs. 2.77 ± 4.26; p<0.001; Table 1). Higher CRP-to-albumin ratio, neutrophil-to-lymphocyte ratio, and systemic immune-inflammation index were observed in the mortality group (all p<0.05; Table 1). Multivariate analysis identified the CALLY index (OR: 0.512; 95% CI: 0.308–0.851; p=0.010), left atrial diameter, and left ventricular systolic diameter as independent predictors of mortality (Table 4). ROC analysis demonstrated good discriminative ability for the CALLY index (AUC: 0.751; 95% CI: 0.665–0.825; Table 5, Figure 1). Pairwise ROC comparisons showed no statistically significant differences between inflammatory markers (Table 6). Kaplan–Meier analysis confirmed significantly lower survival in patients with low CALLY index values (Figure 2).

Conclusions: The findings of this study suggest that the CALLY index is a simple, inexpensive, and reliable biomarker that can effectively predict early mortality in patients undergoing TAVI. By integrating inflammation, immunity, and nutritional status into a single parameter, it may provide additional prognostic information beyond traditional risk scores. The incorporation of the CALLY index into preprocedural risk assessment algorithms could assist clinicians in identifying high-risk patients and optimizing treatment strategies. Prospective, multicenter studies are warranted to validate these results and further explore its clinical utility.

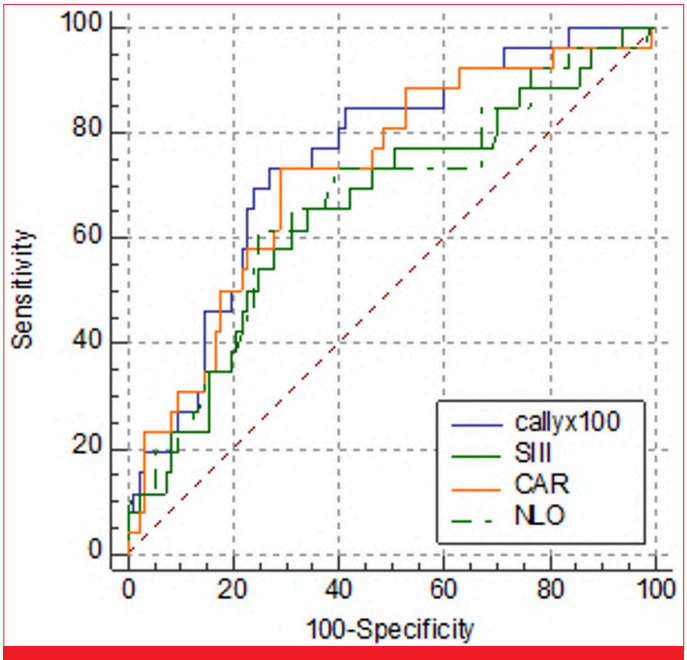

Figure 1. Receiver operating characteristic (ROC) curve analyses.

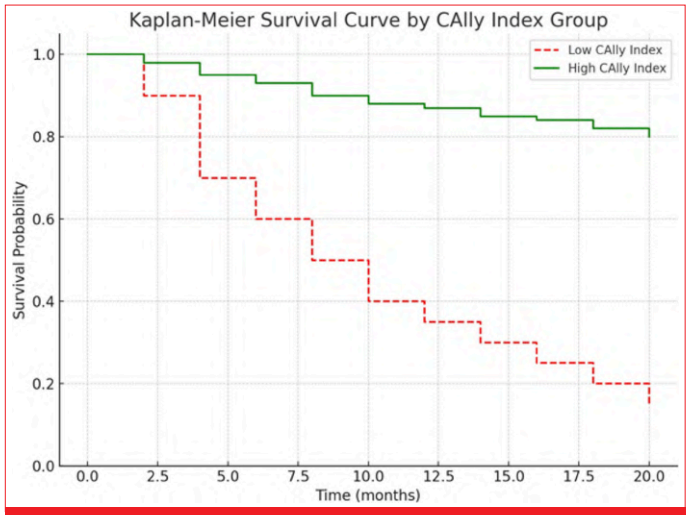

Figure 2. Kaplan–Meier survival analysis.

Table 1. Comparison of laboratory parameters between survivor and non-survivor groups

Parameter	survivor (n=97) Mean ± SD	non-survivor (n=26) Mean ± SD	p-value
CALLY index	2.7658 ± 4.2614	0.7558 ± 1.0592	**<0.001***
SII (x10^3)	691.0 ± 507.1	1100.7 ± 901.1	**0.034***
CAR	3.12 ± 6.09	8.54 ± 14.14	**0.004***
NLR	3.35 ± 2.26	5.55 ± 4.78	**0.001***
Age (years)	79.3 ± 5.08	77.0 ± 7.59	0.154
ICU stay (days)	1.97 ± 1.11	2.46 ± 2.92	**<0.001***
AKI (yes/no)	0.2239 ± 0.3357	0.2818 ± 0.4093	0.406
BMI (kg/m²)	23.8 ± 2.22	23.9 ± 1.79	0.812
HBG (g/dL)	11.54 ± 1.79	11.33 ± 1.81	0.584
PLT (10^3/µL)	205.71 ± 73.59	211.04 ± 74.14	0.744
WBC (10^3/µL)	7.32 ± 2.62	7.83 ± 2.45	0.375
Neutrophil (10^3/µL)	4.88 ± 2.61	5.58 ± 2.44	0.218
Monocyte (10^3/µL)	0.740 ± 0.462	0.700 ± 0.185	0.666
Glucose (mg/dL)	113.78 ± 43.56	128.19 ± 54.51	0.159
Uric Acid (mg/dL)	6.71 ± 1.89	6.88 ± 2.14	0.693
Preop Creatinine (mg/dL)	1.20 ± 0.86	1.48 ± 0.96	0.153
Postop Day 2 Creatinine (mg/dL)	1.44 ± 0.88	1.87 ± 1.22	**0.046***
BUN (mg/dL)	58.05 ± 25.40	70.11 ± 32.91	0.092
LDL (mg/dL)	99.77 ± 33.95	93.62 ± 30.72	0.404
HDL (mg/dL)	48.78 ± 14.08	43.47 ± 11.70	0.081
Triglyceride (mg/dL)	116.5 ± 60.32	117.1 ± 45.14	0.965
Total Cholesterol (mg/dL)	171.3 ± 46.25	160.9 ± 41.57	0.301
ALT (U/L)	17.49 ± 19.69	23.62 ± 33.33	0.233
AST (U/L)	21.23 ± 15.61	23.25 ± 18.98	0.377
Albumin (g/dL)	3.42 ± 0.35	3.15 ± 0.37	**0.021***
Lymphocyte (10^3/µL)	1.69 ± 0.65	1.35 ± 0.63	**0.023***
CRP (mg/L)	10.5 ± 17.88	25.83 ± 36.74	**0.003***

CALLY index: index based on CRP, albumin, and lymphocyte; SII: systemic immune-inflammation index; CAR: CRP/albumin ratio; NLR: neutrophil/lymphocyte ratio; ICU stay: intensive care unit stay duration; AKI: acute kidney injury; BMI: body mass index; HBG: hemoglobin; PLT: platelet count; WBC: white blood cell;LDL: low-density lipoprotein; HDL: high-density lipoprotein; ALT: alanine aminotransferase; AST: aspartate aminotransferase; CRP: C-reactive protein. Values are presented as mean ± standard deviation. p<0.05 was considered statistically significant.

Table 2. Comparison of echocardiographic parameters between survivor and non-survivor groups

Parameter	survivor (n=97) Mean ± SD	non-survivor (n=26) Mean ± SD	p-value
EF (%)	48.96 ± 11.04	45.77 ± 10.07	0.184
Left Atrium Diameter (mm)	43.41 ± 5.85	46.88 ± 5.95	**0.011***
Diastolic Diameter (mm)	48.73 ± 5.52	52.31 ± 7.19	**0.007***
Systolic Diameter (mm)	31.51 ± 7.01	36.04 ± 7.81	**0.011***
IVS (mm)	14.02 ± 2.40	13.65 ± 2.38	0.148
PW (mm)	12.23 ± 1.69	11.58 ± 1.81	0.089
MR (grade)	1.55 ± 0.68	1.65 ± 0.64	0.641
AR (grade)	1.07 ± 0.71	1.15 ± 0.61	0.594
Postop Aortic Mean Gr (mmHg)	7.65 ± 2.56	7.54 ± 2.18	0.840
TR (grade)	1.73 ± 0.60	1.93 ± 0.79	0.191
PAP (mmHg)	50.13 ± 14.46	52.69 ± 15.44	0.263

EF: ejection fraction; IVS: interventricular septal thickness; PW: posterior wall thickness; MR: mitral regurgitation grade; AR: aortic regurgitation grade; Postop Aortic Mean Gr: postoperative aortic valve mean gradient; TR: tricuspid regurgitation grade; PAP: pulmonary artery pressure. Values are presented as mean ± standard deviation. p<0.05 was considered statistically significant.

Table 3. Comparison of clinical categorical variables between survivor and non-survivor groups

Parameter	survivor n (%)	non-survivor n (%)	Total n (%)	p-value
Female sex	54 (85.7%)	9 (14.3%)	63 (51.2%)	0.056
Postop AF	4 (57.1%)	3 (42.9%)	7 (5.7%)	0.147
Transfusion	39 (83.0%)	8 (17.0%)	47 (38.2%)	0.379
Postop Pacemaker	9 (64.3%)	5 (35.7%)	14 (11.4%)	0.156
Hypertension	27 (84.4%)	5 (15.6%)	32 (26.0%)	0.375
Diabetes Mellitus	57 (79.2%)	15 (20.8%)	72 (58.5%)	0.922
Hyperlipidemia	52 (81.3%)	12 (18.8%)	64 (52.0%)	0.499
Coronary artery disease	50 (80.6%)	12 (19.4%)	62 (50.4%)	0.625
Congestive heart failure	37 (88.1%)	5 (11.9%)	42 (34.1%)	0.071
Smoking	63 (82.9%)	13 (17.1%)	76 (61.8%)	0.164

Postoperative atrial fibrillation (Postop AF); requirement for permanent pacemaker after the procedure (Postop Pacemaker); Values are expressed as percentages. p<0.05 was considered statistically significant.

Table 4. Results of logistic regression analysis for predictors of post-TAVI mortality

Parameter	Exp(B)	95% CI	p-value
CALLY INDEX	0.512	0.308 – 0.851	0.010
CAR	1.066	1.008 – 1.128	0.024
NLR	1.235	1.062 – 1.436	0.006
SII	1.001	1.000 – 1.002	0.009
Postop Day 2 Creatinine	1.474	0.995 – 2.184	0.053
Left Atrium Diameter	1.101	1.022 – 1.187	0.012
Systolic Diameter	1.080	1.020 – 1.144	0.008

CALLY index: CRP, albumin, and lymphocyte-based index; SII: systemic immune-inflammation index; CAR: CRP/albumin ratio; NLR: neutrophil/lymphocyte ratio; Exp(B): odds ratio; CI: confidence interval. Statistically significant values are shown in bold (p < 0.05).

Table 5. ROC analysis of inflammatory markers for mortality prediction

Parameter	AUC	95% CI	p-value	Youden Index (J)	Cut-off Value	Sensitivity (%)	Specificity (%)
CALLY index	0,751	0.665–0.825	< 0.0001	0,4627	≤ 0.6343	73,08	73,2
CRP	0,718	0.630–0.796	0.0001	0,4318	> 6.9	73,08	70,1
SII	0,656	0.565–0.739	0.0131	0,3136	> 687.304	65,38	65,98
CAR	0,725	0.637–0.801	0.0001	0,4421	> 1.8904	73,08	71,13
NLO	0,668	0.577–0.750	0.0078	0,368	> 4.1	61,54	75,26

AUC = Area under the ROC curve; CI = Confidence Interval; Youden Index (J) = Sensitivity + Specificity – 1; Cut-off Value = Optimal threshold calculated using the Youden Index; Sensitivity = True positive rate (%); Specificity = True negative rate (%);
CALLY = C-reactive protein–albumin–lymphocyte index; CRP = C-reactive protein; SIII = Systemic immune-inflammation index; CAR = C-reactive protein to albumin ratio; NLO = Neutrophil to lymphocyte ratio.

Table 6. Pairwise AUC comparison of inflammatory markers

Comparison	Difference in AUC	95% CI	z statistic	p-value
CALLY vs SII	0,095	-0.0330 to 0.223	1,454	0,1459
CALLY vs CAR	0,0262	-0.0322 to 0.0846	0,878	0,3798
CALLY vs NLO	0,0833	-0.0441 to 0.211	1,281	0,2
SII vs CAR	0,0688	-0.0726 to 0.210	0,953	0,3404
SII vs NLO	0,0117	-0.0607 to 0.0841	0,317	0,7515
CAR vs NLO	0,0571	-0.0956 to 0.210	0,733	0,4635

AUC: Area Under the Curve; CI: Confidence Interval; CALLY: C-reactive protein–Albumin–Lymphocyte Index; SII: Systemic Immune-Inflammation Index; CAR: C-reactive protein to Albumin Ratio; NLO: Neutrophil-to-Lymphocyte Ratio.

PP-033 [Interventional Cardiology / Coronary]

Rota Cell: New marker of platelet activation marker for rotational atherectomy

Erdoğan İlkay[1], Ersin Sarıçam[1], Selin Merih Urlu[2], Arslan Öcal[3]

[1]*Department of Cardiology, Medicana International Ankara Hospital, Ankara*
[2]*Department of Internal Medicine, Medicana International Hospital, Ankara*
[3]*Department of Cardiology, Gülhane Training and Research Hospital, Ankara*

Background and Aim: Rotational atherectomy (rotablation) assists percutaneous coronary intervention (PCI) for de novo severely calcified or unexpandable lesions, which are increasingly prevalent in the elderly population. During plaque ablation, the rotating burr causes damage to endothelial cell barrier which leads to collagen exposure. But this calcium debulking technique has some severe complications, including slow-flow, distal embolization, and increase in cardiac enzymes due to interaction between platelets and atheromatous debris. This process triggers platelet activation. Peripheral blood reactions are not well known with optimal speed of rotablation. We investigated blood cell changes in the bloodstream following rotational atherectomy by using peripheral blood smear.

Methods: This study is a randomized double-blind controlled study that compares rotational atherectomy plus coronary intervention (balloons plus stenting technique) and only coronary intervention. Rotational atherectomy group (20 patients) had at least one de novo highly calcified lesion eligible for rotablation procedure. The coronary intervention group (20 patients) had stable coronary plaque with at least >70% luminal narrowing. Both group baseline features were similar. The patients with high troponin levels, active infection findings, hematologic malignancy were excluded. All patients took standard antithrombotic and antiplatelet therapy. Rotablation was performed with a Rotablator coronary system (Rotablator®, Boston Scientific Corp., Natick, MA, USA). Following ablation, balloon predilatation and stent implantation were performed. After 6-12 hours procedure, we collected all patients' peripheral blood smear with Wright-Giemsa combination stain. After staining, the monolayer is viewed under a microscope using magnification up to 1000 times. Individual cells, which erythrocyte, platelets, and white blood cells are examined by expert hematologists.

Results: We observed normal findings in control group. However, we realized abnormal peripheral blood smear findings. In red blood cells, we saw schistocytes, which are fragmented erythrocytes (all rotational atherectomy group). Secondly, we observed

hypersegmented neutrophils as morphologic abnormalities of mature granulocytes in 2 patients. Lastly, we saw giant granules platelets in four patients. These patients were associated with 1.75 burr size and high calcific lesion. We defined giant platelets as rota cell (Figure). Rota cell was referred to as a powerful activated platelet effect, in which large platelets are more active than small platelets.

Conclusions: Activated platelet effects could be observed in peripheral smear tests in rotational atherectomy techniques. We saw abnormal giant platelet appearance in peripheral smear test and defined as 'rota cell'. We think that this cell is a powerful activated platelet marker. This finding could be indirect peripheral sign of effective calcium debulking.

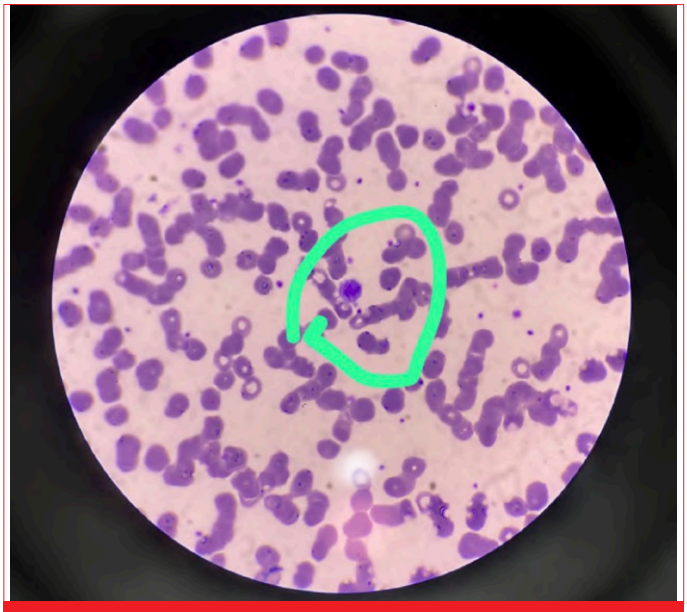

Figure 1. Giant granules platelets; rota cell.

PP-034 [Other]

Does the VFRisk score, a new risk prediction score for sudden cardiac death, contribute to risk classification in the hypertrophic cardiomyopathy population?

Büşra Kuru Görgülü, Arda Güler, Utku Yartası, Gamze Babür Güler

Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul

Background and Aim: Implantable cardioverter-defibrillators (ICDs) have been proven effective in the primary prevention of sudden cardiac death (SCD) among Hypertrophic cardiomyopathy (HCM) patients. However, appropriate patient selection remains a clinical challenge, largely due to the use of differing risk stratification tools across various guidelines. The VFRisk score is a clinical prediction algorithm developed to identify patients presenting with shockable rhythms, ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT), who are at risk of SCD. The score is composed of 13 clinical, electrocardiogram, and echocardiographic variables derived from the patient's lifetime clinical records. 8 clinical (history of diabetes, heart failure, stroke, atrial fibrillation, myocardial infarction, COPD, seizure disorder, syncope), 4 ECG variables (resting heart rate, QTc interval, T peak-T end interval, delayed intrinsic deviation) and 1 echocardiographic marker (left ventricular hypertrophy). In our study, we wanted to investigate the prognostic power of this score or its parameters in the HCM population and whether it contributes to risk stratification.

Methods: This was a retrospective observational study that included all adult patients with a diagnosis of hypertrophic cardiomyopathy (HCM) who were consecutively evaluated over a 16-year period at a large tertiary HCM referral center. In total, 558 patients were included in the final analysis. Subsequent analyses examined the association of this score with clinical parameters such as ICD implantation, presence of late gadolinium enhancement (LGE) on cardiac MRI, and the HCM-SCD risk score.

Results: Baseline characteristics of the final study population, consisting of 558 patients, are summarized in Table 1. The mean age of the patients was 52.3 years. Among the cohort, 143 patients were categorized in the extensive LGE group, while 422 were in the non-extensive LGE group. Male sex, a history of congestive heart failure, previous myocardial infarction, and the presence of an implanted ICD were all significantly more prevalent in the extensive LGE group. Similarly, NT-ProBNP levels, prolonged QT interval, and atrial fibrillation rhythm were significantly higher in patients with extensive LGE. As shown in Figure 1, VFRisk scores were significantly higher in patients with extensive LGE compared to those without (6.60 ± 2.11 vs. 5.77 ± 2.24, p<0.001). As shown in Figure 2, VFRisk score was significantly higher among patients who had received an ICD compared to those without an ICD (7.13 ± 2.11 vs. 5.85 ± 2.24; p=0.001). This finding suggests that the VFRisk score retrospectively aligns with clinical decisions regarding ICD implantation.

Conclusions: The VFRisk score, a new sudden cardiac risk predictor, has been found to be statistically associated with late gadolinium enhancement and ICD implantation in patients with hypertrophic cardiomyopathy. This score may be useful in SCD risk stratification in sarcomeric HCM patients.

Table 1. Demographic, clinical, and electrocardiographic characteristics of patients

Baseline Characteristics	*n* (%) or Mean ± SD	<15% LGE (n:422)	≥ 15% LGE (n:143)	p- Value
Male sex	395 (69,9%)	283 (67,0%)	112 (78,3%)	0,01
Congestive heart failure	333 (68,7%)	262 (62,1%)	126 (88,1%)	<0,001
HCMP type Obstructive Nonobstructive Apical		 228 (54,8%) 156 (37,5%) 32 (7,7%)	 55 (38,7%) 66 (46,5%) 21 (14,8%)	 0,001
ICD	31	25 (80,6%)	6 (19,4%)	0,01
Alcohol ablation	10	8 (80,0%)	2 (20,0%)	0,70
Myectomy	12 (1,2%)	10(2,4%)	2 (1,4%)	0,48
NT-ProBNP	1258	1162	1499	<,001
Disopyramide	55 (9,7%)	43 (10,3%)	12 (8,5%)	0,51
Betablocker [b]	452 (79,5%)	321 (78,3%)	117 (83,0%)	0,29
CCB [b]	114 (20,1%)	96 (24,0%)	18 (12,9%)	0,01
Sinus	514 (90,9%)	391 (92,7%)	123 (86,0%)	0,01
AF	39 (6,9%)	23 (5,5%)	16 (11,3%)	0,01
Pace rhythm	12 (2,1%)	7 (1,7%)	5 (3,5%)	0,19
Heart rate	72,8 ⋆ 12,8	73,1 ⋆12,9	72,2 ⋆12,5	0,36
Heart rate>75	222 (39,3%)	174 (41,2%)	48 (33,6%)	0,12
Prolonged QT	183 (32,4%)	125 (29,6%)	58 (40,6%)	0.02
T peak to t end	114 (20,2%)	78 (18,5%)	36 (25,2%)	0,10
Delayed intrinsicoid	42 (7,4%)	32 (7,6%)	10 (7,0%)	0,96

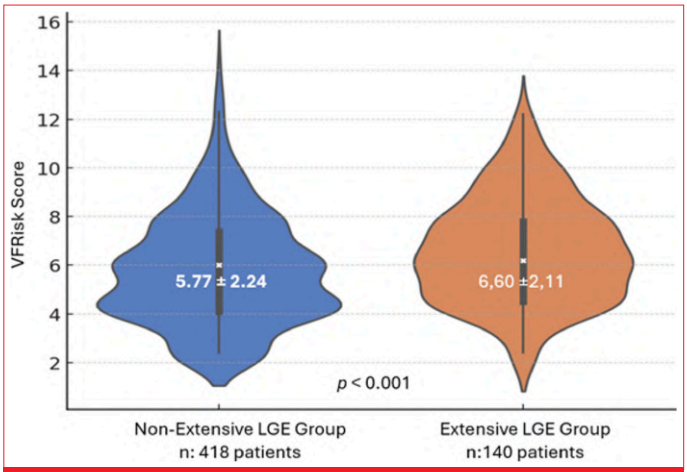

Figure 1. Distribution of VFRisk score according to extent of LGE.

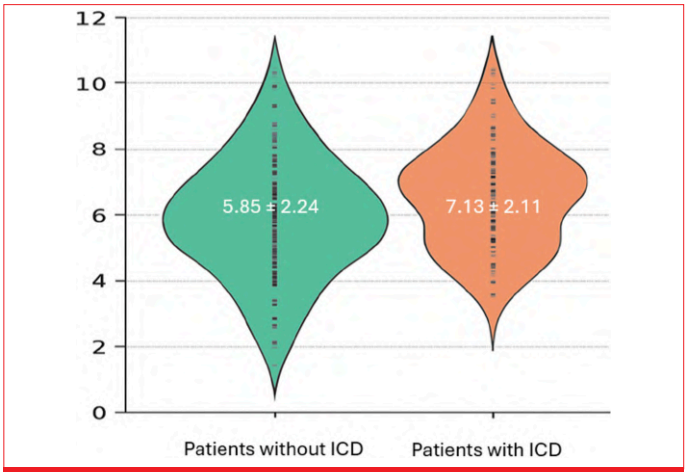

Figure 2. Distribution of VF risk scores according to ICD implantation status.

PP-035 [Coronary Artery Disease / Acute Coronary Syndrome]

Clinical outcomes of chronic coronary artery disease patients receiving colchicine: A multicenter observational study

Ecem Gürses[1], Mustafa Doğduş[2], Umut Uyan[4], Mehdi Zoghi[3]

[1]Department of Cardiology, Bakırçay University, Çiğli Training and Research Hospital, İzmir
[2]Department of Cardiology, Medical Park Hospital, İzmir
[3]Department of Cardiology, Ege University, Faculty of Medicine, İzmir
[4]İzmir Ödemiş State Hospital, İzmir

Background and Aim: Inflammation plays a pivotal role in the initiation and progression of atherosclerosis. Acute coronary syndrome (ACS), particularly myocardial infarction (MI), induces a systemic inflammatory response, promoting plaque destabilization and increasing the risk of recurrent cardiovascular events. Colchicine, an anti-inflammatory agent that inhibits multiple steps of the inflammatory cascade and modulates endothelial activation and platelet function, has shown prognostic benefit in post-MI and ACS patients in the COLCOT and LoDoCo2 trials. This study aimed to evaluate the clinical outcomes of colchicine therapy in patients with chronic coronary artery disease (CAD) who remained at high risk despite optimal medical therapy.

Methods: Between September 1, 2024, and July 15, 2025, patients with chronic CAD from multiple centers who continued to have uncontrolled risk factors or recurrent cardiac events despite guideline-directed medical therapy were enrolled. Colchicine was initiated at 0.5 mg/day for secondary prevention. Gastrointestinal intolerance was assessed at one month. Functional capacity, angina frequency and severity, and quality of life were assessed at baseline and at six months using the Modified Seattle Angina Questionnaire (SAQ) and the Canadian Cardiovascular Society (CCS) classification. Paired t-tests were used for continuous variables, and chi-square tests for categorical variables; $p<0.05$ was considered statistically significant.

Results: A total of 312 patients (mean age 60.4 ± 9.3 years; 68.9% male) were included. Comorbidities included hypertension (83.8%), diabetes mellitus (34.9%), hyperlipidemia (47.4%), and chronic obstructive pulmonary disease (19.0%). Smoking status: current smokers (48.4%), former smokers (44.6%), never smokers (7.1%). At one month, gastrointestinal intolerance occurred in 9.0% of patients. At six months, significant improvements were observed compared with baseline: heavy lifting capacity (3.76 → 4.04; p=0.001), quality of life score (3.42 → 3.87; p=0.03), reduced nitrate use in the previous four weeks (4.13 → 4.38; p<0.005), higher patient satisfaction scores (1.70 → 2.28; p<0.005), and lower CCS angina class (2.52 → 2.01; p=0.023). Hospitalization and coronary revascularization rates at six months were both 3.5%.

Conclusions: In this multicenter observational study, colchicine use in chronic CAD patients was associated with significant improvements in angina symptoms, functional capacity, and patient-reported quality of life measures over a six-month follow-up period. The low rates of gastrointestinal intolerance and hospitalization further suggest its safety in this population. While these findings are consistent with prior large-scale randomized trials in post-MI and ACS populations, the current study adds real-world data for stable CAD patients who remain at high risk despite optimal therapy.

Table 1. Basic clinical characteristics of the patients

Demographic Characteristics	
Age (years)	60.4
Male (%)	68.9%
Smoking status	
Current smokers	48.4%
Former smorkers	44.6%
Never smoked	7.1%
Medical History	
Hypertension (%)	83.8%
Diabetes mellitus (%)	34.9%
Hyperlipidemia (%)	47.4%
Chronic obstructive pulmonary disease (%)	19%

Table 2. Comparisons of pre- and post-treatment assessments using the modified seattle angina questionnaire and CCS classification

Parameter	Baseline	6 months	p value
Heavy lifting capacity	3.76	4.04	0.001
Quality of life scores	3.42	3.87	0.03
Nitrate use (past 4 weeks)	4.13	4.38	<0.005
Patient satisfaction score	1.7	2.28	<0.005
CCS angina class	2.52	2.01	0.023

CCS: Canadian Cardiovascular Society, SAQ: Seattle Angina Questionnaire.

Table 3. Adverse events

Adverse Events	Incidence (%)
Gastrointestinal intolerance (1 month)	9.0
Hospitalization (6 months)	3.5
Coronary revascularization (6 months)	3.5

PP-037 [Coronary Artery Disease / Acute Coronary Syndrome]

A novel indicator of all-cause mortality in acute coronary syndrome: the CALLY index

Barış Güven[1], Muhammed Furkan Deniz[2], Neziha Aybüke Geylan[3], Veysel Oktay[3]

[1]*Department of Cardiology, İdil State Hospital, Şırnak*
[2]*Department of Cardiology, Bağcılar Education and Research Hospital, İstanbul*
[3]*Department of Cardiology, İstanbul University, Cerrahpaşa Institute of Cardiology, İstanbul*

Background and Aim: Acute coronary syndrome (ACS) is often the first clinical presentation of cardiovascular disease, which is the leading cause of mortality and morbidity worldwide. Inflammation plays a fundamental role in the pathogenesis of atherosclerosis and acute coronary events. It is also well established that dietary and nutritional habits are significant determinants of cardiovascular risk. Considering these factors, ACS patients should be approached from a holistic perspective, as immunonutritive and inflammatory markers may be associated with clinical outcomes. The CALLY index, which is based on serum CRP, serum albumin, and lymphocyte count, is a novel biomarker believed to be important in reflecting the balance of nutrition, immunity, and inflammation. This score, which we hypothesize could provide significant predictive results in ACS patients, has not been previously studied in this patient group, nor has its prognostic value for long-term mortality been tested. In this study, we aimed to investigate the prognostic effect of the CALLY index on long-term mortality in ACS patients who have undergone primary percutaneous coronary intervention (pPCI).

Methods: This retrospective cohort study included 505 patients who presented with ACS and underwent pPCI in single center. Data were obtained from patient records and the hospital database and were used anonymously. CALLY index and other five prognostic scores were calculated. The median follow-up was 40 months. All-cause mortality was defined as the primary endpoint.

Results: The median age of the patients was 59 years, 23.4% were female. The CALLY index was categorized into low (<0.7) and high (≥0.7). Age (p=0.038), concomitant atrial fibrillation (p=0.023), previous CABG (p=0.001), ACE-I/ARB/ARNI use (p=0.015), diuretic use (p=0.021), and a low-CALLY index (p<0.001) were identified as independent predictors of all-cause mortality in multivariate cox regression analysis. When compared to other prognostic scores according to AUC in ROC analysis, the CALLY index demonstrated the best ability to predict all-cause mortality. Additionally, patients with a high-CALLY index exhibited significantly better survival outcomes compared to those with a low-CALLY index (log-rank: p<0.001).

Conclusions: Our study suggest that the CALLY index can be used as an independent prognostic tool for assessing the outcomes of ACS patients who have undergone primary PCI. It appears to offer greater predictive value compared to some existing scoring systems used for patient evaluation. Consequently, the CALLY index may be a valuable biomarker for risk assessment, guiding treatment decisions, determining follow-up schedules, and improving prognosis. To thoroughly evaluate the CALLY index's potential as a prognostic factor, further research with larger sample sizes and multicenter trials is necessary.

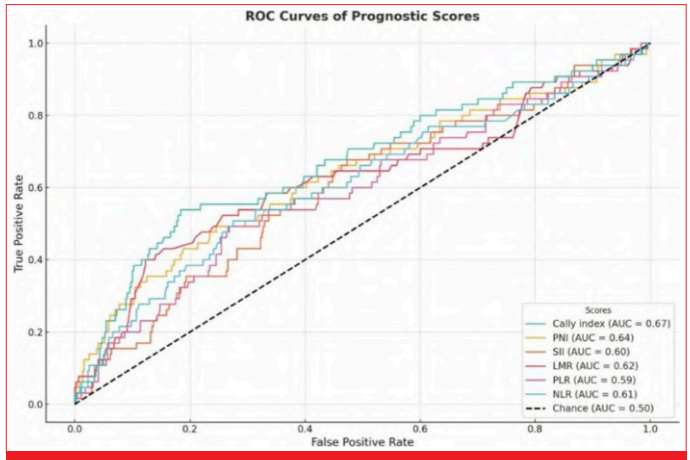

Figure 1. ROC curves of prognostic scores.

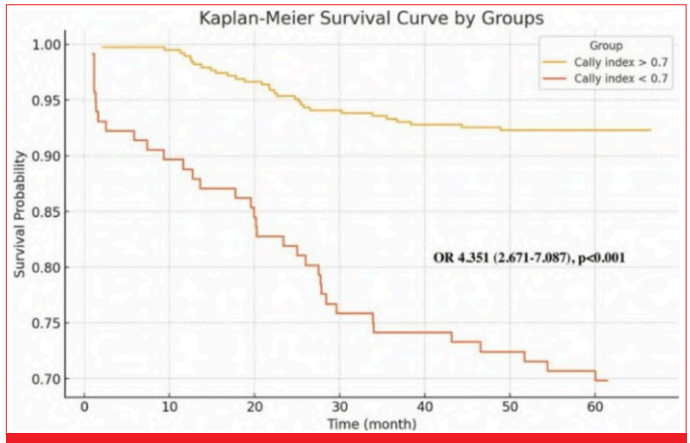

Figure 2. Kaplan-Meier survival curve by groups.

Table 1. Demographic and clinical characteristics of study population

Variables	All patients (n = 505)
Age (year)	59 (52–68)
Sex, n (%), (female)	118 (23.4)
HT, n (%)	234 (46.3)
DM, n (%)	181 (35.8)
CKD, n (%)	61 (12.1)
Anemia, n (%)	94 (18.6)
Hyperlipidemia, n (%)	203 (40.2)
Smoking, n (%)	299 (59.2)
AF, n (%)	35 (6.9)
Femoral access, n (%)	482 (95.4)
Previous PCI, n (%)	184 (36.4)
Previous CABG, n (%)	61 (12.1)
Previous stroke, n (%)	21 (4.2)
Previous major bleeding, n (%)	4 (0.8)
STEMI, n (%)	189 (37.4)
LVEF, (%)	52 (45–60)
Follow-up (month)	40 (34–50)
Death, n (%)	65 (12.9)

AF: atrial fibrillation; CKD: chronic kidney disease; DM: diabetes mellitus; HT: hypertension; LVEF: left ventricular ejection fraction.

Table 2. Treatment at discharge

Variables	All patients (n = 505)
ASA use, n (%)	505 (100)
Clopidogrel use, n (%)	182 (36)
Ticagrelor use, n (%)	312 (61.8)
Prasugrel use, n (%)	8 (1.6)
Statin use, n (%)	501 (99.2)
Beta-blocker use, n (%)	476 (94.3)
ACE-I/ARB/ARNI use, n (%)	452 (89.5)
MRA use, n (%)	49 (9.7)
Diuretic use, n (%)	61 (12.1)

ACE-I: angiotensin-converting enzyme inhibitors; ARB: angiotensin receptor blockers; ARNI: angiotensin receptor-neprilysin inhibitor ASA: acetylsalicylic acid; MRA: mineralocorticoid receptor antagonist.

Table 3. Laboratory findings and values of prognostic scores

Variables	All patients
Haemoglobin level (g/dL)	14.4 (13.0–15.4)
Platelet count (per nL)	231(194–271)
WBC level (10^3/μL)	9.5 (7.9–11.8)
Lymphocyte count (10^3/μL)	2.1 (1.5–2.7)
Neutrophil count (10^3/μL)	6.0 (4.7–8.0)
Monocyte count (10^3/μL)	0.7 (0.6–0.9)
Creatinine level (mg/dL)	0.90 (0.77–1.06)
C-reactive protein level (mg/L)	4.4 (2.0–9.3)
Albumin level (g/dL)	3.99 (3.74–4.22)
Total cholesterol level (mg/dL)	170 (143–205)
NLR	2.86 (2.04–4.60)
LMR	2.88 (2.11–3.86)
PLR	111 (84–158)
PNI	82.45(57.98–110.64)
SII	675 (440–1117)
CALLY index	1.96 (0.74–4.06)

CALLY index: C-reactive protein-albumin-lymphocyte index; LMR: lymphocyte-to-monocyte ratio; NLR: neutrophil-to-lymphocyte ratio; PLR: platelet-to-lymphocyte ratio; PNI: prognostic nutritional index; SII: systemic immune-inflammation index; WBC: white blood count.

Table 4. Univariate and multivariate cox regression analysis for predicting all cause mortality

	Univariate analysis		Multivariate analysis	
Variables	HR (95% CI)	p-value	HR (95% CI)	p-value
Age	1.053 (1.030–1.077)	<0.001	1.028 (1.002–1.054)	**0.038**
Sex (male)	0.771 (0.448–1.328)	0.348		
HT	1.245 (0.766–2.025)	0.377		
DM	1.682 (1.033–2.736)	0.036	1.087 (0.631–1.872)	0.763
CKD	2.343 (1.315–4.173)	0.004	0.906 (0.458–1.791)	0.776
Anemia	2.359 (1.410–3.947)	0.001	1.338 (0.758–2.362)	0.315
Hyperlipidemia	0.825 (0.493–1.381)	0.464		
Smoking	1.026 (0.624–1.685)	0.920		
AF	4.414 (2.439–7.987)	<0.001	2.192 (1.113–4.317)	**0.023**
Femoral access	1.449 (0.354–5.926)	0.606		
Previous PCI	0.917 (0.551–1.525)	0.738		
Previous CABG	2.790 (1.603–4.856)	<0.001	2.956 (1.545–5.655)	**0.001**
Previous stroke	1.939 (0.777–4.835)	0.156		
Previous major bleeding	2.656 (0.368–19.190)	0.333		
STEMI	1.151 (0.700–1.891)	0.580		
LVEF	0.956 (0.935–0.977)	<0.001	0.978 (0.953–1.004)	0.103
ASA use	0.499 (0.069–3.601)	0.491		
Clopidogrel use	1.426 (0.874–2.326)	0.155		
Ticagrelor use	0.638 (0.392–1.038)	0.070		
Prasugrel use	2.102 (0.513–8.608)	0.302		
Statin use	0.485 (0.067–3.498)	0.473		
Beta-blocker use	0.527 (0.227–1.221)	0.135		
ACE-I/ARB/ARNI use	0.487 (0.260–0.912)	0.025	0.428 (0.216–0.849)	**0.015**
MRA use	1.741 (0.887–3.415)	0.107		
Diuretic use	4.537 (2.711–7.594)	<0.001	2.238 (1.132–4.424)	**0.021**
Low-CALLY Index	4.351 (2.671–7.087)	<0.001	2.945 (1.676–5.176)	**<0.001**

ACE-I: angiotensin-converting enzyme inhibitors; AF: atrial fibrillation; ARB: angiotensin receptor blockers; ARNI: angiotensin receptor-neprilysin inhibitor; ASA: acetylsalicylic acid; CALLY index: C-reactive protein-albumin-lymphocyte index; CKD: chronic kidney disease; DM: diabetes mellitus; HT: hypertension; LVEF: left ventricular ejection fraction; MRA: mineralocorticoid receptor antagonist.

PB-038 [Other]

The role of education and screening criteria in the diagnosis of transthyretin cardiac amyloidosis: Preliminary findings from a multicenter national study

Işık Tekin[1], Yalın Tolga Yaylalı[1], Barış Düzel[2], Sena Kaplan[3], Hatice Solmaz[3], Sevil Gülaştı[9], Ömer Faruk Çırakoğlu[6], Muhammet Raşit Sayın[6], Zehra Erkal[4], Nermin Bayar[4], Gönül Açıksarı[7], Pelin Karaca Özer[8], Cafer Zorkun[8], Lütfü Bekar[5], Cansu Öztürk[10], Önder Öztürk[10], Taner Şen[12], Emre Demir[11], Mehdi Zoghi[11], Oktay Ergene[13]

[1]Department of Cardiology, Pamukkale University, Faculty of Medicine, Denizli
[2]Department of Cardiology, Mersin City Hospital, Mersin
[3]Department of Cardiology, Izmir City Hospital, İzmir
[4]Department of Cardiology, Antalya Education and Research Hospital, Antalya
[5]Department of Cardiology, Hitit University, Faculty of Medicine, Çorum
[6]Ahi Evren Cardiovascular Surgery Education and Research Hospital, Trabzon
[7]Department of Cardiology, İstanbul Medeniyet University, Göztepe Education and Research Hospital, İstanbul
[8]Department of Cardiology, İstanbul University, İstanbul Faculty of Medicine, İstanbul
[9]Department of Cardiology, Adnan Menderes University, Faculty of Medicine, Aydın
[10]Department of Cardiology, Diyarbakır Education and Research Hospital Hospital, Diyarbakır
[11]Department of Cardiology, Ege University, Faculty of Medicine, İzmir
[12]Department of Cardiology, Kütahya Health Sciences University, Evliya Çelebi Training and Research Hospital, Kütahya
[13]Department of Cardiology, İzmir Katip Çelebi University, Atatürk Training and Research Hospital, İzmir

Background and Aim: Although transthyretin (TTR) cardiac amyloidosis is a treatable disease when diagnosed correctly, it is

often underdiagnosed or misdiagnosed due to its rarity and the insufficient recognition of disease-specific findings (red flags). This study aimed to evaluate the impact of cardiologist-targeted educational interventions and structured diagnostic criteria on the screening and diagnosis rates of TTR cardiac amyloidosis. In particular, the effectiveness of the screening criteria of age ≥60 years and left ventricular hypertrophy (LVH) >13 mm was assessed.

Methods: This multicenter study was conducted in two phases: pre-education (June 2023–2024) and post-education (June 2024–2025). Cardiologists participating in the study received online education covering red flag findings, diagnostic algorithms, and structured screening strategies. Emphasis was placed on identifying red flags in patients aged ≥60 years with LVH >13 mm, such as elevated NT-proBNP, atrial enlargement, conduction abnormalities, and pseudo-Q waves. Educational materials included printed and visual brochures with diagnostic algorithms and treatment tracking forms based on ESC guidelines. The number of screened and diagnosed patients before and after the education was compared. Chi-square and Fisher's exact tests were used for statistical analysis.

Results: A total of 11 centers and 19 investigators participated in the study. Following the educational interventions and implementation of structured screening materials, a statistically significant increase was observed in both the number of screened patients and diagnosed TTR cardiac amyloidosis cases. The number of screened patients increased by 190%, and the number of diagnosed cases rose by 536% after education (Table 1). The most frequent red flag findings were elevated NT-proBNP and atrial enlargement. Additionally, the number of patients who met the age ≥60 & LVH >13 mm criteria and had at least one red flag increased by 230% (from 80 to 264 patients) in the post-education period.

Conclusions: The use of structured educational programs and screening criteria significantly improved the diagnosis rate of TTR cardiac amyloidosis. Notably, the increase in diagnosis rate from 4.5% to 9.8% highlights the critical role of clinical suspicion and targeted screening in identifying the right patients. This finding indicates that nearly 1 out of every 10 screened patients received a confirmed diagnosis—an outcome that underscores the diagnostic value of focused clinical training and standardized criteria.

Table 1. Comparison of screened and diagnosed patient numbers before and after clinical education

Period	Screened patients	Diagnosed patients	Diagnosis rate (%)	p
2023–2024 (pre-education)	246	11	4.5	<0.01
2024–2025 (post-education)	713	70	9.8	

PP-039 [Interventional Cardiology / Coronary]

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

Eser Durmaz, Murat Çimci, Damla Raimoglou, Gündüz İncesu, Alpin Mert Tekin, Metehan Miroğlu, Sedat Güney, Ahmet Atıl Aygün, Işıl Firdevs Sarıbay İncesu, Zeynep Soybay, Mahmut Kaan Temiz, Utku Raimoğlu, Bilgehan Karadağ

Department of Cardiology, İstanbul University, Cerrahpaşa Faculty of Medicine, İstanbul

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,

Table 1. Comparison of lifestyle modification

	Combined follow-up group 243 patients	Centre-based follow-up group 299 patients	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

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).

Table 2. Comparison of symptom control

	Combined follow-up group 243 patients	Centre-based follow-up group 299 patients	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 3. Patient demographics and clinical features

	Combined follow-up group 243 patients	Centre-based follow-up group 299 patients	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
HbA1C (%) Baseline Follow-up	 6 (5,6-6,7) 6,1 (5,6-6,7)	 6 (5,7-6,8) 6,2 (5,7-7)	 0,933 0,248
LDL-cholosterol (mg/dl) Baseline Follow-up Reduction (%)	 129,6±44,4 78±30,6 41±17,4	 127,1±41,9 91,1±35,8 35,9±17,6	 0,621 <0.001 0.020
Pro-BNP (ng/dl) Baseline Follow-up	 431 (136- 1288) 191 (69-580)	 566 (100-1517) 543 (155-2420)	 0,573 <0.001
LVEF (%) Baseline Follow-up	 47,6±10,7 51,9±9	 43,1±10,6 48,8±10,4	 <0,001 0.003

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 healthy lifestyle and symptom control in comparison to conventional centre-based follow-up strategy in which patients were evaluated only at the outpatient clinic.

PP-040 [Hypertension]

The role of the EASIX score in patients with hypertension: A cross-sectional study

Ender Murat[1], Mehmet Sadık Karpat[1], Yusuf Öztürk[1], Hatice Taşkan[2], Ozan Köksal[3], Ayşe Saatçi Yaşar[1], Murat Çelik[1], Uygar Çağdaş Yüksel[1], Cem Barçın[1]

[1]*Department of Cardiology, Gülhane Training and Research Hospital, Ankara*
[2]*Department of Cardiology, Haseki Training and Research Hospital, İstanbul*
[3]*Department of Cardiology, Zonguldak Atatürk State Hospital, Zonguldak*

Background and Aim: Hypertension (HT) is a leading cause of cardiovascular morbidity and mortality, and ambulatory blood pressure monitoring (ABPM) provides superior risk stratification compared to office measurements. The endothelial activation and stress index (EASIX), a biomarker derived from lactate dehydrogenase (LDH), creatinine (CRE), and platelet (PLT) counts, has been increasingly recognized as a predictor of vascular dysfunction and adverse outcomes. This study aimed to investigate the association between EASIX score and 24-hour ABPM parameters in patients with HT without significant comorbidities.

Methods: This retrospective cross-sectional study included 192 patients aged 18–70 years who were diagnosed with HT and had no additional comorbidities, ensuring a homogeneous study population focused solely on isolated hypertension (Figure 1). The EASIX score was calculated as [LDH × CRE] / PLT and analyzed after Log_2 transformation. Patients were categorized according to achievement of target BP values on 24-h ABPM. Comparative analyses, correlation tests, and logistic regression were performed to identify independent predictors of BP control. Receiver operating characteristic (ROC) analysis determined the discriminative performance of EASIX (Graphical Abstract).

Results: Patients without target-range BP had significantly higher EASIX scores compared to those with target-range BP (p<0.05) (Figure 2). ROC analysis identified a threshold EASIX score of 0.48 (AUC=0.755, 95% CI=0.685–0.825), with 80% sensitivity and 68% specificity for predicting inadequate BP control (Figure 3). Logistic regression revealed that ascending aortic diameter, serum sodium, serum albumin, and Log_2 (EASIX) were independent predictors of BP control status (Table 1 and Table 2). Higher EASIX scores were significantly associated with uncontrolled hypertension, reflecting greater endothelial stress and potential cardiovascular risk.

Conclusions: The EASIX score, based on routine laboratory parameters, demonstrated strong predictive ability for identifying hypertensive patients with poor BP control on ABPM. These findings suggest that EASIX may serve as a practical, low-cost biomarker reflecting vascular endothelial stress in HT. Incorporating EASIX into clinical evaluation could help identify high-risk patients, guide the use of ABPM, and support timely therapeutic adjustments. Prospective studies are warranted to validate its prognostic value and explore integration into clinical decision-making algorithms for HT management.

PP-041 [Cardiac Imaging / Echocardiography]

Relationship between left ventricular multilayer global longitudinal strain and left atrial strain obtain by 'speckle tracking echocardiography' specket tracing method and heart rate variability in patients with hypertrophic cardiomyopathy

Okan Tepeli, Veysel Oktay, Emir Özgür Barış Ökçün

Department of Cardiology, İstanbul University Cerrahpaşa Cardiology Institute, İstanbul

Background and Aim: Our aim in this study is to evaluate systolic and diastolic functions with LV multilayer GLS and LA strain obtained by the'Speckle Tracking Echocardiography'method in HCM patients and to examine ventricular arrhythmias and heart rate variability obtained from holter recordings.

Methods: In the crosssectional study, 51 people diagnosed with HCM and followed up with this diagnosis for at least one year were included as the patient group. 33 healthy individuals, similar in age and gender to the patient group, were included as the control group. Echo examination was performed on all patients with the Philips Epiq (Philips Medical Systems,USA) echo device. Patients were divided into two groups according to the presence/absence of LVOT (left ventricular outflow tract obstruction) 17 LVOT (+),34 LVOT (-) Strain analysis was performed with the help of QLAB13.0 Software version; Philips. All patients and healthy controls underwent echo examination and were fitted with a 24 hour 5 channel rhythm Holter device, Cardioscan12 DMS. The records obtained were evaluated in a computer environment using CardioScan12 software.

Results: In the HCM group, a statistically significant decrease was detected in endo, mid, epiGLS strain values compared to the control group consisting of healthy individuals. EndoGLS %-16.6 vs. %-21.0, p=0.000, MidGLS %-15 vs. %-18.8, p=0.00, EpiGLS %-13.7 vs. %-17.8, p=0.000. The strain values we obtained showed a positive correlation with proBNP values. In our study, statistically significantly more NSVT was detected in the patient group than in the control group p=0.026. The reason for the decrease in multi layer GLS and the frequency of NSVT may be the fibrotic scar tissues and the early effects on systolic functions in HCM patients. LA strain examination plays an important role in the diagnosis of diastolic dysfunction, which plays a role in pathogenesis in HCM patients. In our thesis study, we detected a statistically significant absolute decrease in LARS, LARSP, LACD, LACDP values in LA Strain. HCM patients compared to the control group LARS, LARSP, LACD, LACDP p=0.00. LA size and LAVI measurements were evaluated to be significantly larger p=0.00. In addition, a correlation was observed in many of the LA strain parameters with diastolic dysfunction diagnostic criteria used in conventional echo, such as LAVI and E/e'. In this thesis, we also examined the relationship of HRV parameters with arrhythmias and multi layer GLS and LA strain. No statistically significant difference was found between the control group and the patient group, except for the HF parameter p=0.002 and the difference was not seen in other parameters. Considering the publications on the HRV corrective effect of beta blocker treatment, the lack of a statistically significant difference may be related to the intensive beta blocker use rate of the patients.

Conclusions: Our study is important in terms of the evaluation of the early effects of systolic and diastolic functions in patients diagnosed with HCM by speckle tracking echo and its relationship with ventricular arrhythmias.

Table 1. Comparison of patient and control groups according to left ventricular echocardiography parameters

EF	Min	Maks.	$\bar{x}$	Ss	M	Z	p
HASTA	58,00	67,00	60,6294	1,79168	60,0000	-2.845	.004
KONTROL	57,00	63,00	59,4818	1,37692	59,0000		
Toplam	57,00	67,00	60,1786	1,72704	60,0000		
LVDD	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**p**
HASTA	35,00	55,00	46,9216	4,53803	47,0000	-1.924	.054
KONTROL	38,00	52,00	45,0909	4,24532	45,0000		
Toplam	35,00	55,00	46,2024	4,49036	46,0000		
LVSD	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	21,00	36,00	29,7843	3,18317	30,0000	-.521	.602
KONTROL	26,00	36,00	30,3939	2,48671	30,0000		
Toplam	21,00	36,00	30,0238	2,92878	30,0000		
IVSD	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	14,00	30,00	17,1961	3,01343	16,0000	-7.786	.000
KONTROL	7,00	13,00	10,1515	1,27772	10,0000		
Toplam	7,00	30,00	14,4286	4,25196	15,0000		
PWD	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**p**
HASTA	10,00	18,00	12,7059	1,84710	12,0000	-6.903	.000
KONTROL	7,00	11,00	9,7576	1,11888	10,0000		
Toplam	7,00	18,00	11,5476	2,15320	11,0000		
LVMi(g/m2)	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**p**
HASTA	103,23	261,42	150,9000	32,27935	143,5300	-7.515	.000
KONTROL	49,11	123,00	83,4312	18,66342	84,2000		
Toplam	49,11	261,42	124,3944	43,13714	124,6300		
E(CM/S)	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**p**
HASTA	34,00	104,00	62,1137	15,27419	62,0000	-2.739	.006
KONTROL	29,00	94,00	53,2727	14,04740	53,0000		
Toplam	29,00	104,00	58,6405	15,34567	56,0000		
A	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**p**
HASTA	29,60	125,00	65,4471	20,98752	63,0000	-0.504	.614
KONTROL	35,00	115,00	62,7576	19,27276	63,0000		
Toplam	29,60	125,00	64,3905	20,25583	63,0000		
E'(CM/S)	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	3,10	12,00	7,5333	1,93635	7,7000	-4.628	.000
KONTROL	4,90	14,00	10,0000	2,13146	10,0000		
Toplam	3,10	14,00	8,5024	2,34073	8,5000		
A'	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	4,80	14,00	8,5922	2,09798	8,2000	-1.786	.061
KONTROL	3,00	16,00	9,3273	2,35057	9,3000		
Toplam	3,00	16,00	8,8810	2,21634	9,0000		
E/E'	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	3,40	19	8,32	2,99	8,4000	-4.67	.000
KONTROL	2,90	13,80	5,6152	2,41973	4,8000		
Toplam	2,90	19	14,3288	3,06	6,5000		

Table 2. Holter heart rate variability

ORTKH	Min	Maks.	$\bar{x}$	Ss	M	Z	P
HASTA	54,00	90,00	71,3725	9,13227	70,0000	-.119	.905
KONTROL	52,00	90,00	71,3030	10,33043	72,0000		
Toplam	52,00	90,00	71,3452	9,55957	71,5000		
SDNN24	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	69,00	496,00	134,8235	62,45277	120,0000	-	.085
KONTROL	75,00	213,00	140,0303	32,92936	136,0000	1.722	
Toplam	69,00	496,00	136,8690	52,67078	130,0000		
SDNNİ	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	26,00	147,00	52,8431	21,56884	48,0000	-	.288
KONTROL	33,00	114,00	56,0303	18,96293	53,0000	1.063	
Toplam	26,00	147,00	54,0952	20,52657	50,0000		
rMSSD	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	12,00	75,00	30,2745	14,41538	27,0000	-	.598
KONTROL	11,00	55,00	27,2727	9,14560	27,0000	0.527	
Toplam	11,00	75,00	29,0952	12,63350	27,0000		
Pnn50	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	,00	34,00	8,4510	8,69785	5,0000	-.193	.847
KONTROL	,00	28,00	6,9697	6,23240	5,0000		
Toplam	,00	34,00	7,8690	7,81531	5,0000		
TOTAL POWER	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	627,00	6269,00	1986,2128	1228,97444	1664	-.591	.554
KONTROL	753,00	4012,00	1687,9091	757,07894	1678		
Toplam	627,00	6269,00	1863,1625	1064,64463	1671		
LF	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	52,00	1446,00	536,1200	352,47797	459,5000	-.442	.658
KONTROL	102,00	1085,00	470,6667	265,67457	455,0000		
Toplam	52,00	1446,00	510,0964	320,66275	458,0000		
HF	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	34,00	18484,00	2647,2000	3696,49682	708,5000	-	.002
KONTROL	18,00	3495,00	372,1818	604,07871	205,0000	3.052	
Toplam	18,00	18484,00	1742,6747	3092,29585	303,0000		

Table 3. Left atrium, LAVI, left atrial strain findings (evaluation according to R-R and P-P)

LA	Min	Maks.	$\bar{x}$	ss	M	Z	P
HASTA	34,00	49,00	42,1373	3,48723	42,0000	-6.180	.000
KONTROL	30,00	42,00	36,0606	3,08159	36,0000		
Toplam	30,00	49,00	39,7500	4,46101	40,0000		
LAVI	**Min**	**Maks.**	$\bar{x}$	**ss**	**M**	**Z**	**P**
HASTA	19,63	57,32	36,0798	9,10297	35,1000	-5.431	.000
KONTROL	12,69	43,08	24,5712	6,34738	24,0300		
Toplam	12,69	57,32	31,5586	9,87033	30,9800		
LA reservoir strain ED	**Min**	**Maks.**	$\bar{x}$	**ss**	**M**	**Z**	**P**
HASTA	9,40	72,00	33,9490	12,75179	33,4000	-3.683	.000
KONTROL	24,30	67,50	44,8000	11,46356	44,6000		
Toplam	9,40	72,00	38,2119	13,30581	38,3500		
LA CONDUİT STRAİN ED	**Min**	**Maks.**	$\bar{x}$	**ss**	**M**	**Z**	**P**
HASTA	-40,60	-5,20	-19,3490	9,67085	-16,6000	-4.071	.000
KONTROL	-50,90	-12,30	-29,7758	10,25137	-28,4000		
Toplam	-50,90	-5,20	-23,4452	11,09509	-23,5000		
LA CONTRACTİLE STRAİN ED	**Min**	**Maks.**	$\bar{x}$	**ss**	**M**	**Z**	**P**
HASTA	-33,70	18,80	-13,8608	8,73996	-13,2000	-.421	.674
KONTROL	-33,40	-1,70	-15,0182	8,25770	-14,1000		
Toplam	-33,70	18,80	-14,3155	8,52230	-13,6000		
LA RESERVOIR STRAIN PREA	**Min**	**Maks.**	$\bar{x}$	**ss**	**M**	**Z**	**P**
HASTA	9,40	54,70	29,2431	9,89534	28,2000	-4.016	.000
KONTROL	23,40	55,50	38,8515	8,77208	37,4000		
Toplam	9,40	55,50	33,0179	10,53278	34,0000		
LA CONDUIT STRAIN PREA	**Min**	**Maks.**	$\bar{x}$	**ss**	**M**	**Z**	**P**
HASTA	-34,00	-4,80	-16,9294	8,38991	-14,2000	-3.930	.000
KONTROL	-49,70	-9,30	-25,7697	8,98855	-23,9000		
Toplam	-49,70	-4,80	-20,4024	9,61346	-20,5000		
LA CONTRACTILE STRAIN PREA	**Min**	**Maks.**	$\bar{x}$	**ss**	**M**	**Z**	**P**
HASTA	-25,40	,60	-12,3059	5,70920	-12,0000	-.256	.798
KONTROL	-26,80	14,30	-12,2182	8,14480	-12,1000		
Toplam	-26,80	14,30	-12,2714	6,72409	-12,0000		

Table 4. Left ventricular multilayer GLS patient and control group findings

ENDOGLS	Min	Maks.	$\bar{x}$	Ss	M	Z	P
HASTA	-24,00	-10,10	-16,6157	2,94716	-16,4000	-5.730	.000
KONTROL	-30,20	-16,00	-21,0182	3,10307	-20,4000		
Toplam	-30,20	-10,10	-18,3452	3,69100	-18,3000		
MIDGLS (TRANSMURAL)	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	-20,90	-9,30	-15,0392	2,55844	-14,9000	-5.817	.000
KONTROL	-26,00	-14,90	-18,8424	2,38210	-18,5000		
Toplam	-26,00	-9,30	-16,5333	3,10201	-16,7000		
EPIGLS	**Min**	**Maks.**	$\bar{x}$	**Ss**	**M**	**Z**	**P**
HASTA	-19,30	-8,30	-13,7431	2,44608	-13,8000	-6.404	.000
KONTROL	-22,90	-14,00	-17,8333	1,84555	-17,6000		
Toplam	-22,90	-8,30	-15,3500	2,99266	-15,1000		

Table 5. Table summarizing the beta-blocker use and other demographic characteristics of the patient and control groups and the results of the presence of NSVT on rhythm holter between the groups

Özellikler	HASTA GRUBU Min-Maks x̄±SS (M)		KONTROL GRUBU Min-Maks x̄±SS (M)		Toplam Min-Maks x̄±SS (M)		Ki Kare	P
	N	%	n	%	N	%	χ2	P
NSVT							**4.941**	**.026**
• YOK	44	86,3%	33	100,0%	77	91,7%		
• VAR	7	13,7%	0	0,00%	7	8,3%		
SVT	**N**	**%**	**n**	**%**	**N**	**%**	**2.021**	**.158**
• YOK	31	60,8%	25	75,8%	56	66,7%		
• VAR	20	39,2%	8	24,2%	28	33,3%		

Özellikler	HASTA GRUBU Min-Maks x̄±SS (M)		KONTROL GRUBU Min-Maks x̄±SS (M)		Toplam Min-Maks x̄±SS (M)		Z*	P
Yaş	29-80 55,58±12,90 (57)		24-74 53,57±13,29 (54)		24-80 54,79±13,01 (56.5)		*-,646*	*,518*
Boy	154-190 170.47±9,49 (170)		153-183 166.36±8,32 (165)		153-190 168.85±9,22 (169.5)		*-1.904*	*,057*
Kilo	55-108 83,09±12.41 (84)		52-105 78,60±12.23 (78)		52-108 81,33±12,47 (80)		*-1,724*	*,085*
VKI	2220-3810 2855.6±376.2 (2780)		2220-4893 2895.6±550.4 (2750)		2220-4893 2871.3±449.9 (2770)		*-0,412*	*,680*
	N	**%**	**N**	**%**	**N**	**%**	**χ2**	**P**
Cinsiyet							*2.021*	*.155*
• Kadın	14	27,5%	14	42,4%	28	33,3%		
• Erkek	37	72,5%	19	57,6%	56	66,7%		
Sigara	**N**	**%**	**N**	**%**	**N**	**%**	*16.803*	*.000*
• Hayır	24	47,1%	30	90,9%	54	64,3%		
• Evet	16	31,4%	2	6,1%	18	21,4%		
• Exsmoker	11	21,6%	1	3,0%	12	14,3%		
Betabloker kullanımı								
Evet	44	%86,27	1	%3,03				
Hayır	7	%13,73	32	%96,97				

Table 6. The correlation analysis findings table of left ventricular multilayer gls and Pro-BNP. The correlation analysis results of the parameters evaluating diastolic function (LAVI and E/e') related to left atrial strain examination

			PROBNP	ENDOGLS	MİDGLS	EPİGLS
Spearman's rho	PROBNP	Korelasyon Katsayısı	1,000	,314*	,373**	,353*
		P	.	,026	,008	,012
		N	50	50	50	50
	ENDOGLS	Korelasyon Katsayısı	,314*	1,000	,933**	,831**
		P	,026	.	,000	,000
		N	50	51	51	51
	MİDGLS	Korelasyon Katsayısı	,373**	,933**	1,000	,930**
		P	,008	,000	.	,000
		N	50	51	51	51
	EPİGLS	Korelasyon Katsayısı	,353*	,831**	,930**	1,000
		P	,012	,000	,000	.
		N	50	51	51	51

Correlations

			E/e'	LARS	LACD	LACS	LARSP	LACDP	LACSP
Spearman's rho	E/e'	Correlation Coefficient	1,000	**-,380****	**,310****	**,220***	**-,368****	**,275***	**,150**
		Sig. (2-tailed)		**,000**	**,004**	**,044**	**,001**	**,011**	**,172**
	LARS	Correlation Coefficient		1,000	-,805**	-,568**	,975**	-,735**	-,513**
		Sig. (2-tailed)			,000	,000	,000	,000	,000
	LACD	Correlation Coefficient			1,000	,039	-,871**	,984**	-,022
		Sig. (2-tailed)				,727	,000	,000	,845
	LACS	Correlation Coefficient				1,000	-,454**	-,062	,928**
		Sig. (2-tailed)					,000	,573	,000
	LARSP	Correlation Coefficient					1,000	-,824**	-,399**
		Sig. (2-tailed)						,000	,000
	LACDP	Correlation Coefficient						1,000	-,134
		Sig. (2-tailed)							,225
	LACSP	Correlation Coefficient							1,000
		Sig. (2-tailed)							.

			LAVI	LARS	LACD	LACS	LARSP	LACDP	LACSP
Spearman's rho	LAVI	Korelasyon	1,000	-,334*	,372**	,053	-,331*	,379**	,017
		P		,017	,007	,713	,018	,006	,906
		N	51	51	51	51	51	51	51
	LARS	Korelasyon	-,334*	1,000	-,798**	-,633**	,984**	-,731**	-,600**
		P	,017	.	,000	,000	,000	,000	,000
		N	51	51	51	51	51	51	51
	LACD	Korelasyon	,372**	-,798**	1,000	,141	-,867**	,990**	,055
		P	,007	,000	.	,323	,000	,000	,701
		N	51	51	51	51	51	51	51
	LACS	Korelasyon	,053	-,633**	,141	1,000	-,547**	,034	,931**
		P	,713	,000	,323	.	,000	,812	,000
		N	51	51	51	51	51	51	51
	LARSP	Korelasyon	-,331*	,984**	-,867**	-,547**	1,000	-,807**	-,505**
		P	,018	,000	,000	,000	.	,000	,000
		N	51	51	51	51	51	51	51
	LACDP	Korelasyon	,379**	-,731**	,990**	,034	-,807**	1,000	-,058
		P	,006	,000	,000	,812	,000	.	,688
		N	51	51	51	51	51	51	51
	LACSP	Korelasyon	,017	-,600**	,055	,931**	-,505**	-,058	1,000
		p	,906	,000	,701	,000	,000	,688	.
		N	51	51	51	51	51	51	51

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).

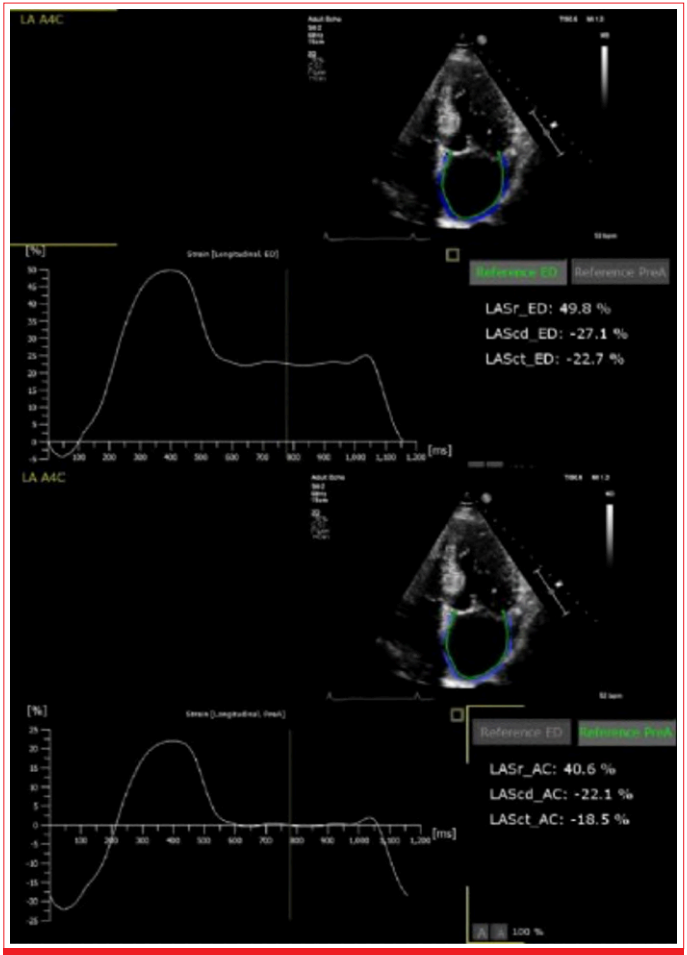

Figure 1. Left atrial strain measurement. [Upper image (A): Left atrial strain value (ED) according to R-R, lower image (B) Left atrial strain value (preA) according to P-P].

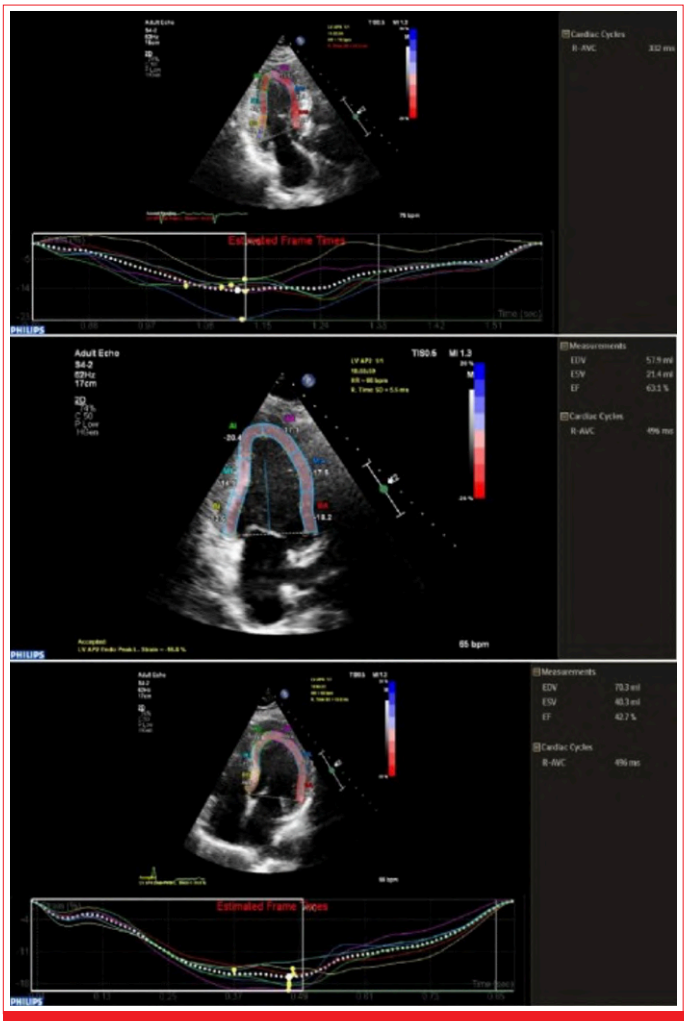

Figure 2. Multilayer GLS image acquisition. Left ventricular multilayer strain imaging: apical 2-chamber and apical 3-chamber, apical 4-chamber images (A: UPPER; 3-chamber image, B: MIDDLE; 2-chamber image, BOTTOM; 4-chamber image).

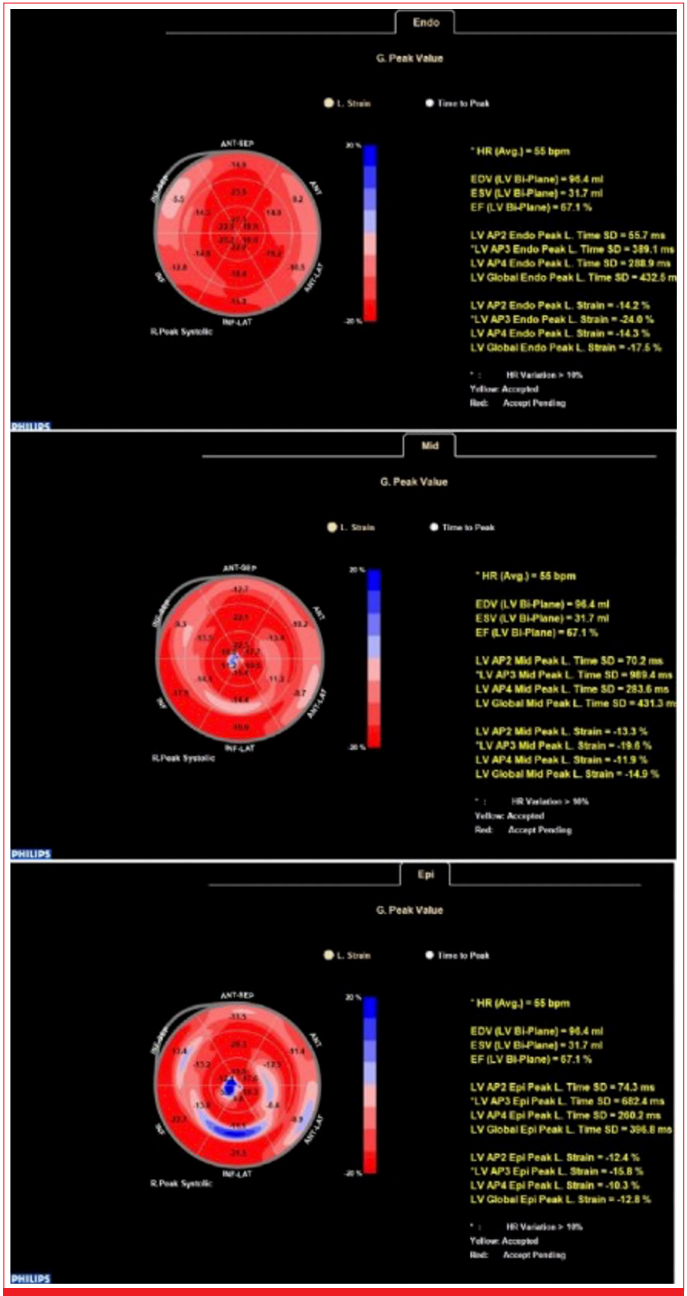

Figure 3. Multilayer gls results table.

PP-042 [Cardiac Imaging / Echocardiography]

Evaluation of the relationship between intrapericardial pressure and echocardiographic findings in patients presenting with cardiac tamponade and/or massive pericardial effusion

Seyda Dereli[1], Tuğba Çetin[2], Furkan Durak[1], Selçuk Yazıcı[1], Sait Terzi[1], Göktuğ Savaş[1]

[1]Department of Cardiology, Dr. Siyami Ersek, Cardiovascular Surgery, Training and Research Hospital, İstanbul
[2]Department of Cardiology, Çorlu State Hospital, Tekirdağ

Background and Aim: Pericardial effusion may be asymptomatic or may present with life-threatening clinical conditions due to increased intrapericardial pressure (IP) leading to cardiac compression. Echocardiography is a rapid and effective imaging modality for the diagnosis of pericardial effusion (PE) and assessment of its hemodynamic consequences. In this study, we aimed to investigate the relationship between IP and echocardiographic and hemodynamic parameters in patients who presented with pericardial effusion and/or cardiac tamponade and underwent therapeutic pericardiocentesis.

Methods: This prospective,single-center study was conducted between October 2023 and March 2025. Patients diagnosed with PE and/or tamponade who underwent successful pericardiocentesis by the attending physicians were included. IP measurements were performed through the catheter tip during echocardiography-guided pericardiocentesis. Echocardiographic findings, invasive blood pressure monitoring data, and electrocardiographic (ECG) measurements obtained before and after the procedure were compared and analyzed in relation to IP.

Results: A total of 27 patients with a mean age of 70 ± 13.5 years were included. The mean pericardial fluid thickness measured prior to pericardiocentesis was 32.9 ± 11.3 mm. IP decreased from 8.9

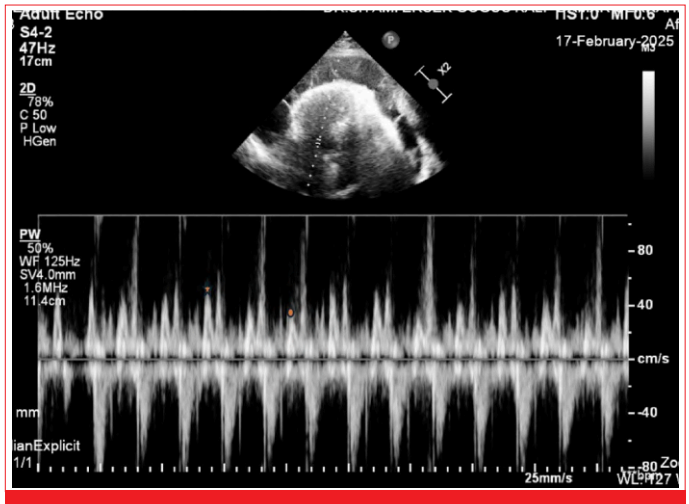

Figure 1. Assessment of Mitral Diastolic E Wave Variation Using Pulsed-Wave Doppler.

± 7.4 mmHg before the procedure to 2.0 ± 8.2 mmHg after the procedure (p=0.001). A significant reduction was observed in both diastolic and mean arterial pressures following pericardiocentesis (p<0.001 for both), while systolic blood pressure showed no significant change (p=0.090). Right atrial pressure, pulmonary artery pressure and inferior vena cava diameter significantly decreased (p<0.001; p=0.044; p<0.001, respectively). Stroke volume, calculated by Doppler echocardiography, increased significantly (p=0.001), and a significant and strong negative correlation was found between stroke volume and IP (r=–0.408; p=0.034). Although a reduction in respiratory variation of mitral E-wave velocity was observed after treatment (p<0.001), this finding did not show a statistically significant correlation with increased IP (r=0.264; p=0.275). In the ECG analyses performed at the 8th hour post-procedure, a significant decrease in heart rate was observed (from 106.7 ± 24.0 bpm to 95.0 ± 18.2 bpm; p=0.001), whereas no statistically significant changes were detected in QRS and P wave amplitudes.

Conclusions: A significant and strong negative correlation was found between increased IP and stroke volume estimated by Doppler echocardiography. On the other hand, no statistically significant relationship was observed between the respiratory variation of the mitral E wave and increased IP or hemodynamic deterioration. The findings of our study indicate that a single echocardiographic parameter is insufficient to assess the severity of elevated pericardial pressure and the resulting cardiac compression. Therefore, clinical, hemodynamic and imaging findings should be evaluated in a comprehensive and patient-specific manner.

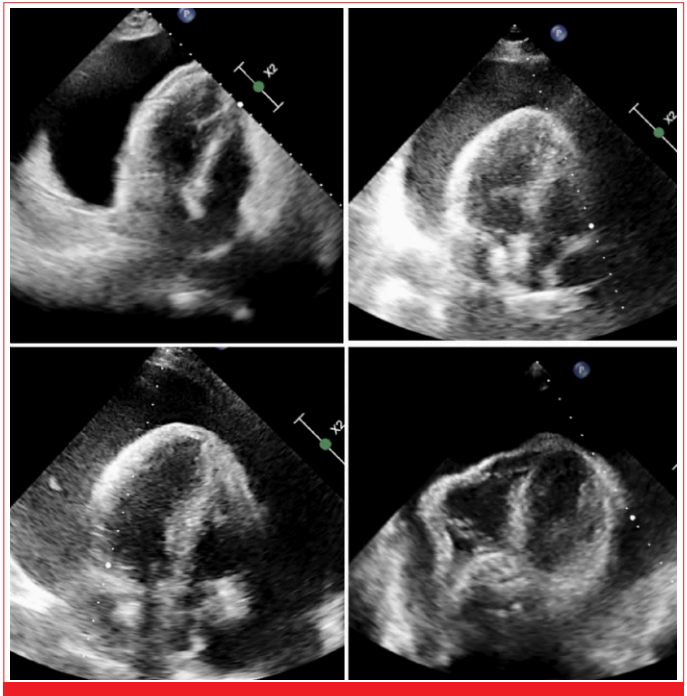

Figure 2. Evaluation of pericardial effusion by two-dimensional echocardiography. (A) Apical four-chamber view showing pericardial effusion accumulated adjacent to the left heart chambers. (B and C) In two different patients, circumferential pericardial effusion is observed causing collapse of the left and right atria. (D) Assessment of pericardial effusion through the subcostal window in a patient with chest wall deformity.

Table 1. Comparison of echocardiographic parameters before and after pericardiocentesis

Parameter	Before Pericardiocentesis	After Pericardiocentesis	p-value
Pericardial fluid amount, mm	32.9 ± 11.3	9.1 ± 8.4	<0.001
IVC diameter, mm	21.9 ± 6.9	16.7 ± 6.1	<0.001
Estimated RA pressure, mmHg	10.8 ± 5.1	4.7 ± 3.4	<0.001
Estimated sPAP, mmHg	40.5 ± 18.0	31.4 ± 10.7	0.044
TAPSE, mm	18.3 ± 4.5	17.3 ± 3.7	0.300
RV S' velocity, cm/s	12.1 ± 3.5	11.0 ± 2.4	0.083
Stroke volume, mL	50.9 ± 17.5	64.0 ± 26.9	0.001
Cardiac output, mL/min	5116 ± 1481	5615 ± 1971	0.157
Mitral E wave (expiration), cm/s	77.8 ± 19.5	76.5 ± 22.6	0.795
Mitral E wave (inspiration), cm/s	57.1 ± 18.8	65.4 ± 20.7	0.097
Respiratory variation of mitral E wave, %	25.3 ± 13.3	13.0 ± 7.4	<0.001
Mitral A wave, cm/s	70.4 ± 26.5	73.6 ± 23.9	0.555
Lateral e' wave, cm/s	9.1 ± 3.9	9.5 ± 3.1	0.596
Lateral a' wave, cm/s	11.3 ± 5.0	11.0 ± 5.3	0.782
Septal e' wave, cm/s	7.2 ± 3.5	8.2 ± 3.0	0.185
Septal a' wave, cm/s	9.2 ± 3.0	8.7 ± 3.6	0.686

IVC: Inferior vena cava; RA: Right atrium; RV S': Right ventricular systolic velocity; sPAP: Systolic pulmonary artery pressure; TAPSE: Tricuspid annular plane systolic excursion.

Table 2. Comparison of hemodynamic findings before and after pericardiocentesis

Parameter	Before Pericardiocentesis	After Pericardiocentesis	p-value
IP, mmHg	8.9 ± 7.4	2.0 ± 8.2	**0.001**
SBP, mmHg	137.9 ± 31.3	130.4 ± 30.6	0.090
DBP, mmHg	71.5 ± 15.9	60.8 ± 11.6	**<0.001**
MAP, mmHg	94.7 ± 20.7	84.7 ± 17.8	**<0.001**
SpO_2, %	97.1 ± 2.6	96.7 ± 2.3	0.287
Heart Rate, bpm	106.7 ± 24.0	95.0 ± 18.2	**0.001**

bpm: Beats per minute; DBP: Diastolic Blood Pressure; IP: Intrapericardial Pressure; MAP: Mean Arterial Pressure; SBP: Systolic Blood Pressure; SpO_2: Peripheral Oxygen Saturation.

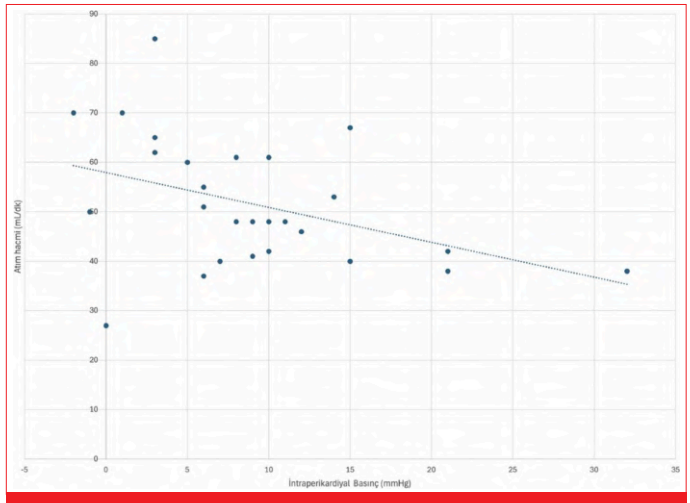

Figure 3. Scatter plot of intrapericardial pressure and stroke volume in patients.

PP-043 [Cardiac Imaging / Echocardiography]

Prognostic value of echocardiographic right ventricular–pulmonary artery coupling parameters for mortality following resuscitated cardiac arrest: A prospective cohort study

Gökhan Ergün, Halime Tanrıverdi, Sefa Gündüz, Yücel Yılmaz, Ömer Sahin, Ziya Şimşek, Selami Demirelli

Department of Cardiology, Kayseri City Training and Research Hospital, Kayseri

Background and Aim: Conventional right ventricular (RV) functions also have prognostic value in patients with cardiac arrest. However, there is a paucity of data concerning the role of RV-Pulmonary artery (RV-PA) coupling parameters as novel indicators of RV function in these patients. The main objective of this study is to investigate the short- and long-term prognostic significance of RV-PA coupling parameters in patients with cardiac arrest.

Methods: This was a single-center, prospective observational study conducted between December 2022 and December 2023. The study population consisted of 49 cardiac arrest patients who were successfully resuscitated. RV-PA coupling parameters were evaluated, and the patients were subsequently followed for mortality outcomes at both one-month and one-year.

Results: One-month and one-year mortality were 48.9% and 59.2%, higher in non-ischemic patients (p=0.006, p=0.025).

Table 1. Comparison of demographic characteristics and laboratory parameters in patients by one-month mortality

Variable	Survival (n=25)	Death (n=24)	p value
Age, years	64 (53-70)	69.5 (58.5-80)	0.207
Gender, male, n (%)	15 (60%)	17 (71%)	0.426
BMI (kg/m²)	27 ± 3.3	27.6 ± 4.4	0.570
SBP (mmHg)	116.3 ± 23.9	108.1 ± 33.1	0.328*
DBP (mmHg)	79 ± 18	69 ± 18	0.082
Heart Rate (beats/min)	86 (76-113)	111 (85-136)	0.078
Diabetes Mellitus, n (%)	12 (48%)	14 (58%)	0.469
Hypertension, n (%)	16 (64%)	19 (79%)	0.240
Coronary Artery Disease, n (%)	12 (48%)	14 (58%)	0.469
Heart Failure, n (%)	8 (32%)	14 (58%)	0.064
Smoking, n (%)	12 (48%)	9 (38%)	0.458
Arrest location (in-hospital), n (%)	21 (84%)	17 (71%)	0.269
Arrest cause (ischemic/non-ischemic), n (%)	20/5 (80/20%)	10/14 (42/58%)	0.006
Glucose (mg/dL)	263 (164-338)	221 (152-322)	0.575
GFR (mL/min/1.73 m²)	65.7 ± 24.6	51.1 ± 25.5	0.046
NT-Pro BNP (ng/L)	959 (189-6332)	4637 (1566-13632)	0.038
CRP (mg/L)	5.5 (3.5-10.5)	16.5 (5.9-57.6)	0.037
Peak Troponin (mU/L)	1136 (143-4264)	435 (168-2420)	0.490

BMI: Body mass index, CRP: C-reactive protein, DBP: Diastolic blood pressure, GFR: Glomerular filtration rate, NT-Pro BNP: N-Terminal pro B-Type natriuretic peptide, SBP: Systolic blood pressure. *: Welch's test.

Table 2. Comparison of echocardiographic parameters based on one-month mortality

Parameter	Survival (n=25)	Death (n=24)	p Value
LV EF (%)	48 ± 12.3	41.2 ± 13.0	0.069
LAVI (mL/m²)	17 (12-24)	23 (14-27)	0.297
E/average e'	8.6 (7.1-9.5)	8.8 (8.0-9.4)	0.435
RV FAC (%)	43.7 ± 7.2	39.8 ± 7.4	0.064
RV s' (cm/s)	10.8 ± 2.6	10.1 ± 3.2	0.429
TAPSE (mm)	33.8 ± 7.4	28.0 ± 7.3	0.008
sPAP (mmHg)	19 (17-25)	36 (22-40)	0.007
pACT (ms)	99 (85-116)	99 (90-115)	0.944
TRV (m/s)	1.6 (1.3-2.1)	2.4 (1.9-2.7)	0.009
TAPSE/sPAP (mm/mmHg)	1.7 (1.2-2.2)	0.8 (0.6-1.4)	<0.001
TAPSE x pACT (mm x ms)	3744 (2574-4176)	2762 (2299-3375)	0.033
TAPSE/TRV (mm/(m/s)	20.0 (14.3-24.6)	11.4 (9.9-16.4)	0.002
FAC/sPAP (%/mmHg)	2.29 (1.62-2.49)	1.17 (0.89-2.2)	0.003
RV s'/sPAP(cm/s/mmHg)	0.52 (0.38-0.61)	0.30 (0.20-0.48)	0.005

LAVI: Left atrial volume index, LV EF: Left ventricle ejection fraction, PACT: Pulmonary acceleration time, RV: Right ventricle, RV FAC: RV fractional area change, RV s': RV systolic velocity, sPAP: Systolic pulmonary artery pressure, TAPSE: Tricuspid annular plane systolic excursion, TRV: Tricuspid regurgitation velocity.

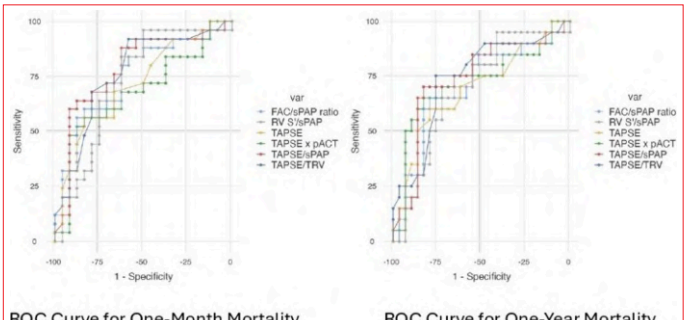

Figure 1.

In patients with higher short- and long-term mortality, RV-PA coupling parameters—including tricuspid annular plane systolic excursion (TAPSE)/systolic pulmonary arterial pressure (sPAP), TAPSE x pulmonary acceleration time (pACT), TAPSE/tricuspid regurgitant velocity (TRV), RV fractional area change (RV FAC) / sPAP, RV systolic tissue doppler velocity of the tricuspid annulus (RV s')/ sPAP, and the conventional marker TAPSE—were lower. Short-term predictors: TAPSE/sPAP, TAPSE x pACT, TAPSE/TRV, RV FAC/sPAP, RV s'/sPAP, TAPSE (p<0.001, p=0.033, p=0.002, p=0.003, p=0.005, p=0.008, respectively); long-term predictors (p=0.005, p=0.008, p=0.007, p=0.018, p=0.022, p=0.013, respectively) were found.

Conclusions: Our findings indicate that RV-PA coupling parameters and TAPSE are strongly and inversely associated with both short- and long-term mortality, highlighting their potential as superior prognostic markers.

Table 3. Comparison of demographic characteristics and laboratory parameters in patients by one-year mortality

Variable	Survival (n=20)	Death (n=29)	p value
Age (years)	64 (53-68)	70 (59-82)	0.057
Gender (Male), n (%)	12 (60%)	20 (69%)	0.517
BMI (kg/m²)	26.5 ± 3.2	27.8 ± 4.2	0.266
SBP (mmHg)	118 ± 23	108 ± 32	0.200*
DBP (mmHg)	80 ± 17	70 ± 19	0.054
Heart Rate (bpm)	86 (84-107)	110 (80-133)	0.238
Diabetes Mellitus, n (%)	10 (50%)	16 (55%)	0.721
Hypertension, n (%)	14 (70%)	21 (72%)	0.854
Coronary Artery Disease, n (%)	10 (50%)	16 (55%)	0.721
Heart Failure, n (%)	6 (30%)	16 (55%)	0.082
Smoking, n (%)	11 (55%)	10 (34%)	0.154
Arrest Location (in-hospital), n (%)	19 (95%)	19 (66%)	0.017°
Arrest Cause (ischemic/ non-ischemic), n (%)	16/4 (80/20%)	14/15 (48/52%)	0.025
Glucose (mg/ dL)	264 (168-316)	217 (153-327)	0.654
GFR (mL/ min/1.73 m²)	72 ± 22.4	49.2 ± 24.2	0.002
NT-ProBNP (ng/L)	685 (176-1808)	4687 (1598-14765)	0.004
CRP (mg/L)	5.0 (3.5-10.5)	10.5 (5.8-54.9)	0.028
Peak Troponin (mU/L)	975 (143-2932)	988 (178-5325)	0.618

BMI: Body mass index, CRP: C-reactive protein, GFR: Glomerular filtration rate, NT-Pro BNP: N-Terminal pro B-Type natriuretic peptide, SBP: Systolic blood pressure. *:Welch's test. °: Fisher's exact test.

Table 4. Comparison of echocardiographic parameters based on one-year mortality

Parameter	Survival (n=20)	Death (n=29)	p value
LV EF (%)	49.0 ± 12.4	41.7 ± 12.8	0.051
LAVI (mL/m²)	18.7 (14.2-24.8)	17.2 (12.6-25.5)	0.864
E/average e'	8.6 (7.5-9.6)	8.7 (7.8-9.2)	0.879
RV FAC (%)	43.6 ± 7.5	40.5 ± 7.5	0.162
RV s' (cm/s)	10.9 ± 2.8	10.2 ± 3.0	0.410
TAPSE (mm)	34.3 ± 7.7	28.7 ± 7.2	0.013
sPAP (mmHg)	19 (17-26)	35 (19-40)	0.037
pACT (ms)	111 (96-116)	95 (88-106)	0.181
TRV (m/s)	1.6 (1.3-2.1)	2.2 (1.7-2.7)	0.021
TAPSE/sPAP (mm/mmHg)	1.7 (1.3-2.0)	2.2 (1.7-2.7)	0.005
TAPSE x pACT (mm x ms)	3870 (3080-4262)	2760 (2300-3366)	0.008
TAPSE/TRV (mm/m/s)	20.5 (16.4-25.9)	12.5 (10-16.7)	0.007
RV FAC/sPAP (%/mmHg)	2.29 (1.61-2.62)	1.27 (0.89-2.2)	0.018
RV S'/ sPAP(cm/s/ mmHg)	0.53 (0.37-0.63)	0.35 (0.21-0.52)	0.022

LAVI: Left atrial volume index, LV EF: Left ventricle ejection fraction, PACT: Pulmonary acceleration time, RV: Right ventricle,RV FAC: RV fractional area change, RV s': RV systolic velocity, sPAP: Systolic pulmonary artery pressure,TAPSE: Tricuspid annular plane systolic excursion, TRV: Tricuspid regurgitation velocity.

PP-044 [Coronary Artery Disease / Acute Coronary Syndrome]

Examination of type-D personality, perceived social support and psychological status in coronary artery disease patients

Burak Çağlar

Department of Cardiology, İstanbul Başakşehir Çam and Sakura City Hospital, İstanbul

Methods: The current descriptive, cross-sectional, correlational study was conducted between May 2023 and January 2024 with 251 patients admitted to the departments of cardiology of a Training and Research Hospital in Istanbul. The Type-D Personality Scale, Multidimensional Scale of Perceived Social Support (MSPSS), and Depression-Anxiety-Stress Scale (DASS-21) were used to collect the data.

Results: The average age of the individuals was 61.48 ± 13.45 years. Of the patients, 55% had a previous MI and 64.5% had additional chronic diseases. The average scores of the total and sub-scales of Type-D Personality Scale (Negative Affection, Social Introversion) were 11.81 ± 5.45, 11.01 ± 5.44, and 12.61 ± 5.46, respectively. The average value of the MSPSS was 73.93 ± 9.79. The average value of Depression was 5.23 ± 3.06, Anxiety was 7.68 ± 3.88, and Stress was 7.4 ± 3.43. A significant negative relationship was detected between Negative Affection and Social Introversion and Support from Private

Person, Family Support, Support from Friends, Support from Friends, and the MSPSS. A significant positive relationship was detected between negative affection and Depression, Anxiety and Stress.

Conclusions: Type-D personality trait was observed in most of the patients. Perceived social support, depression and anxiety levels were high. Type-D personality negatively affects the perception of social support. It was also found that Type-D personality increased depression, anxiety, and stress scores.

PP-045 [Cardiac Imaging / Echocardiography]

Relationship between aortic flow propagation velocity and ankle-brachial index in peripheral artery disease

Habibe Sena Yıldırım, Ünal Güntekin

Department of Cardiology, Akdeniz University, Faculty of Medicine, Antalya

Background and Aim: Peripheral artery disease (PAD) is primarily caused by the progressive narrowing of the arteries in the lower extremities and serves as an indicator of systemic atherosclerosis. The etiopathogenesis of PAD is fundamentally linked to atherosclerosis and its complications. Atherosclerosis is a widespread disease that also affects large vessels, leading to thickening and stiffening of the arterial wall. Consequently, this results in increased arterial resistance and a subsequent decrease in flow propagation velocity within the arterial lumen. Aortic flow propagation velocity (AVP) has been evaluated as a novel parameter reflecting aortic stiffness and, consequently, atherosclerosis. The ankle-brachial index (ABI) is a well-established, simple and noninvasive procedure frequently used in clinical practice for the diagnosis of PAD. The aim of this study was to demonstrate the contribution of ABI and AVP to the noninvasive diagnosis of PAD in patients with angiographically diagnosed PAD and control group patients without PAD.

Methods: The study included 101 patients diagnosed with PAD via conventional peripheral angiography or computed tomographic angiography and 101 control patients without suspected PAD. The maximum ankle arterial pressures were divided by the maximum of the brachial arterial pressures to calculate the ABI. Following routine echocardiographic assessments, from the suprasternal window in supine position the diameter of the descending aorta was measured just distal to the subclavian artery and color M-mode Doppler recordings were obtained by adding cursor parallel to the main flow of direction. AVP was calculated by dividing the distance between the points corresponding to the beginning and end of the propagation slope by the duration between the corresponding time points. The final AVP value was recorded as the mean of at least three measurements (Figure 1-2).

Results: The mean AVP value in the PAD group was 34.65 ± 10.67 cm/s. In contrast, in the control group was 69.5 ± 14.27 cm/s. The AVP values in the PAD group were significantly lower than those in the control group (p<0.0001). In the control group, the mean ABI was 1.19 ± 0.07. In the PAD group, the mean ABI was 0.71 ± 0.18. A statistically significant difference was observed in ABI values between the groups (p<0.0001). A statistically significant moderate positive correlation was found between AVP and ABI (r=0.697, p<0.0001) (Table 1).

Conclusions: In our study, ABI and aortic flow AVP were measured in a group of patients diagnosed with PAD and in a control group without PAD. It was found that AVP was significantly lower in patients with PAD compared to the control group (p<0.0001). Furthermore, AVP was shown to be correlated with ABI, a well-established diagnostic test for PAD (r=0.697, p<0.0001) (Figure 3). This suggests that AVP could serve as a complementary diagnostic tool to ABI and may be particularly useful as an alternative test in cases where ABI measurement is limited or inconclusive.

Table 1. Comparison of AVP and ABI between groups

	PAD (n=101)			Control (n=101)			p
	Mean ± SD	Min	Max	Mean ± SD	Min	Max	
AVP (cm/sn)	34,65±10,67	14,8	77,1	69,5±14,27	30,5	124	<0,0001
ABI	0,71±0,18	0,27	1,2	1,19±0,07	1	1,34	<0,0001

ABI: Ankle-Brachial Index, **AVP**: Aortic Flow Propagation Velocity **SS**: Standard Deviation

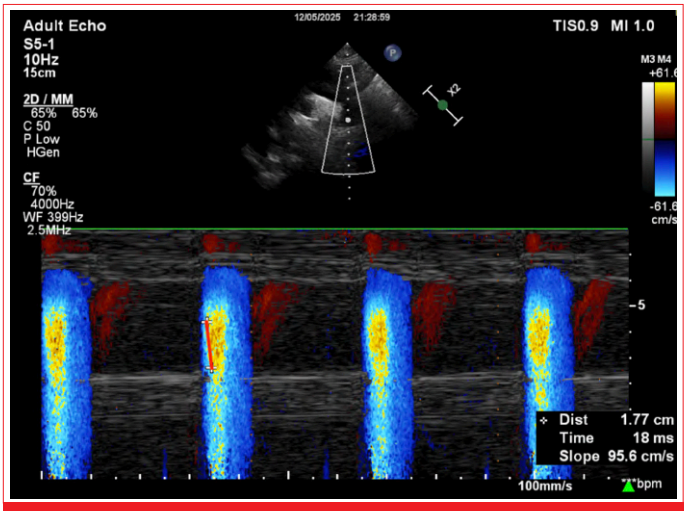

Figure 1. Measurement of descending aortic flow velocity propagation in a subject in the control group.

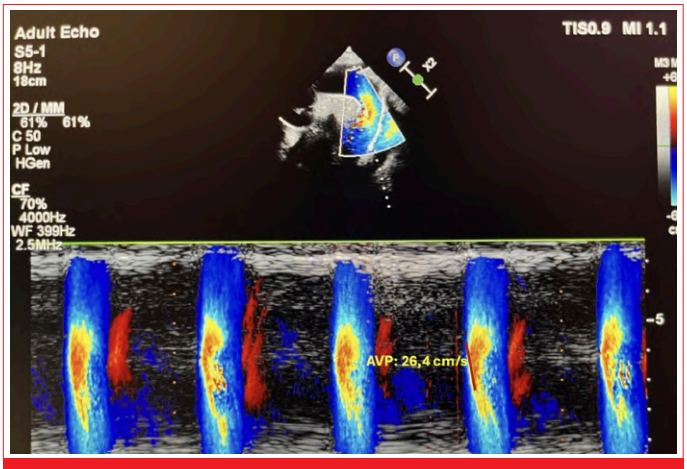

Figure 2. Measurement of descending aortic flow velocity propagation in a subject in a peripheral artery disease.

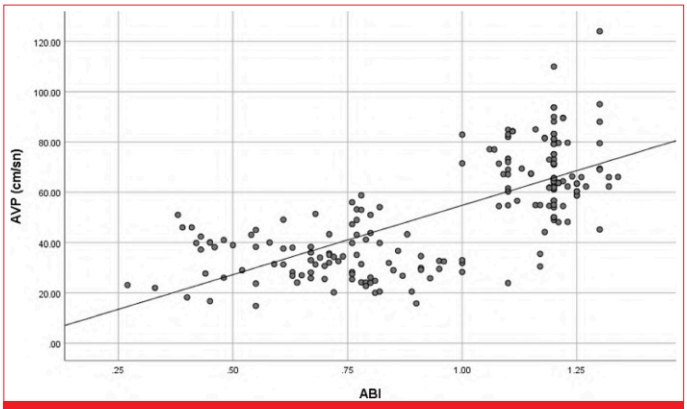

Figure 3. Correlation between AVP and ABI.

PP-046 [Heart Failure]

Evaluation of titration of drugs to target doses in heart failure

İmam Günay[1], Hülya Binokay[2], Abdi Bozkurt[3]

[1]*Department of Cardiology, Yozgat City Hospital, Yozgat*
[2]*Department of Biostatistics, Çukurova University, Faculty of Medicine, Adana*
[3]*Department of Cardiology, Çukurova University, Faculty of Medicine, Adana*

Background and Aim: Beta-blockers (BB), angiotensin-converting enzyme inhibitors/angiotensin receptor blockers (ACEI/ARB), and mineralocorticoid receptor antagonists (MRA) are effective in reducing mortality and morbidity in heart failure (HF). However, in real-world practice, achieving guideline-recommended target doses for these drugs is often limited for various reasons. In this study, we evaluated the use rates of disease-modifying drugs, the levels of achieving target doses, and the reasons for not increasing doses in HF patients based on one-year follow-up data.

Table 1. Use rates of disease-modifying drugs, target dose achievement, and limiting factors

Drug group	Baseline use (%)	Follow-up (%)	Target dose ≥50% (%)	Maximum dose (%)	Limiting factors (%)
Beta blocker	87.5	98.7	76.3	27.6	Bradycardia 33.3% - Hypotension 30.5%
ACEI/ ARB	80.9	93.4	73.7	38.8	Hypotension 47.5% - Elevated creatinine 42.5%
MRA	57.9	75.0	75.0	27.0	Elevated creatinine 57.8% - Hyperkalemia 26.3%

Methods: A total of 152 HF patients with an ejection fraction (EF) ≤40% were prospectively evaluated. The initial and one-year follow-up usage rates of BB, ACEI/ARB, and MRA treatments, the proportion of patients reaching ≥50% of the target dose, the rates of reaching the maximum dose, and the reasons limiting dose escalation were recorded. Data were statistically analyzed ($p<0.05$). The study was conducted in accordance with the Declaration of Helsinki with approval from the relevant ethics committee.

Results: Beta blocker usage increased from 87.5% at baseline to 98.7% at follow-up; ACEI/ARB from 80.9% to 93.4%; and MRA from 57.9% to 75.0% ($p<0.05$). The rates of achieving ≥50% of the target dose were 76.3% for BB, 73.7% for ACEI/ARB, and 75.0% for MRA. The rates of reaching the maximum dose were 27.6% for BB, 38.8% for ACEI/ARB, and 27.0% for MRA. The main reasons for not increasing the doses were bradycardia (33.3%) and hypotension (30.5%) for BB; hypotension (47.5%) and impaired renal function (42.5%) for ACEI/ARB; elevated creatinine (57.8%) and hyperkalemia (26.3%) for MRA.

Conclusions: Real-life data show that HF patients can be treated in accordance with guidelines through regular and individualized follow-up. Bradycardia, hypotension, and impaired renal function are the main obstacles to dose optimization. Although the use of drugs proven to reduce mortality in HF is common in daily practice, the rates of use at recommended doses remain low. It is concluded that gradual dose titration with close follow-up and motivation can help achieve the recommended targets.

PP-047 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

SCALE-CryoAF score and left atrial functions after cryoablation in patients with atrial fibrillation

Kadir Karaçalı

Department of Cardiology, Ankara Gölbaşı State Hospital, Ankara

Background and Aim: The aim of this study is to investigate the SCALE-CryoAF score in predicting late recurrence (>3 months post-ablation) and its relationship with left atrial (LA) functional changes after cryoablation.

Methods: A total of 55 paroxysmal and persistent atrial fibrillation (AF) patients undergoing initial second-generation cryoballoon-based pulmonary vein isolation (CB2-PVI) were prospectively enrolled between February 2024-August 2024. Clinical risk factors and previous medications were obtained directly from the patients. A standard 12-lead electrocardiogram (ECG) was taken and routine blood examination was performed for each patient. All patients underwent transthoracic echocardiography (TTE) including 2D speckle tracking echocardiography before ablation. At the same time transesophageal echocardiography was performed to assess left atrial appendage (LAA) function and rule out intracardiac thrombi. SCALE-CryoAF score and other predictive scores for late recurrence (LR) were calculated. Clinical follow-up with 12-lead ECG and 24-h Holter ECG was regularly carried out at 1, 3, 6, and 12 months and then every 6 months after ablation at the outpatient clinic. TTE was also performed 3 months after ablation to evaluate changes in LA structure and function.

Results: During a median follow-up of 12 (9-12) months, 18 of 55 patients (32.7%) experienced LR. 10 patients (18.2%) had early recurrence (<3 month) and 7 of these patients (70%) also had LR. SCALE-CryoAF score was significantly higher in patients with LR (p<0.001). Other predictive score for LR including MB-LATER, BASE-AF2, CAAP-AF, ATLAS scores were also higher in patients with LR (p=0.002, p=0.013, p=0.035, p=0.007, respectively) while DR-FLASH score was similar between the groups (p=0.310). Receiving operator characteristic (ROC) analysis showed that SCALE-CryoAF score >3.5 predicted LR with a higher sensitivity of 83.3% and specificity of 70.3% than other predictive scores (Area under the curve: 0.817, p<0.001). Kaplan-Meier analysis of 12-month AF/Atrial tachyarrhythmia (ATA)-free survival presented better outcome in patients with SCALE-CryoAF <3.5 (86.9% vs. 37.4%, p<0.001 [Log Rank]). Compared with baseline there were significantly reduction in LA antero-posterior diameter (p<0.001), maximum LA volume index (LAVI) (p<0.001), and minimum LAVI (p<0.001) with increases in LA emptying fraction (LAEF) (p<0.001), LA reservoir strain (p<0.001), LA conduit strain (p<0.001), and LA contractile strain (p=0.006) at 3 months following ablation in patients with SCALE-CryoAF <3.5.

Conclusions: SCALE-CryoAF score provided reliable predictive value for LR after cryoballoon ablation. At the same time, in patients with high SCALE-CryoAF score, there was no improvement in LA functions. Patients with high SCALE-CryoAF score may benefit from more intensive long-term follow-up.

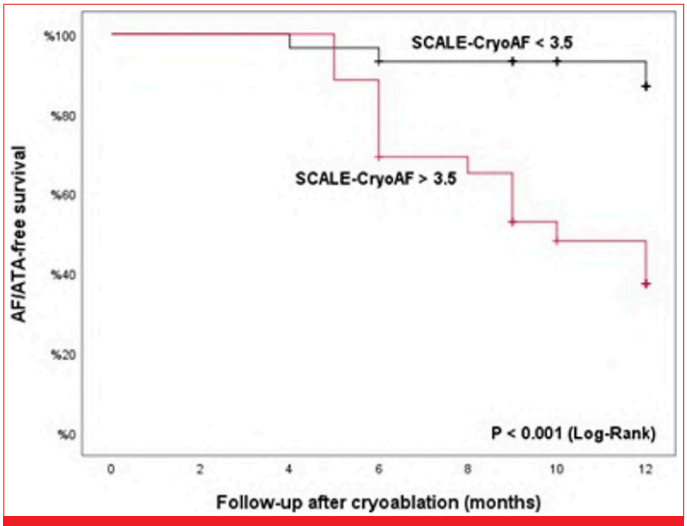

Figure 1. Kaplan-Meier survival analysis of the groups.

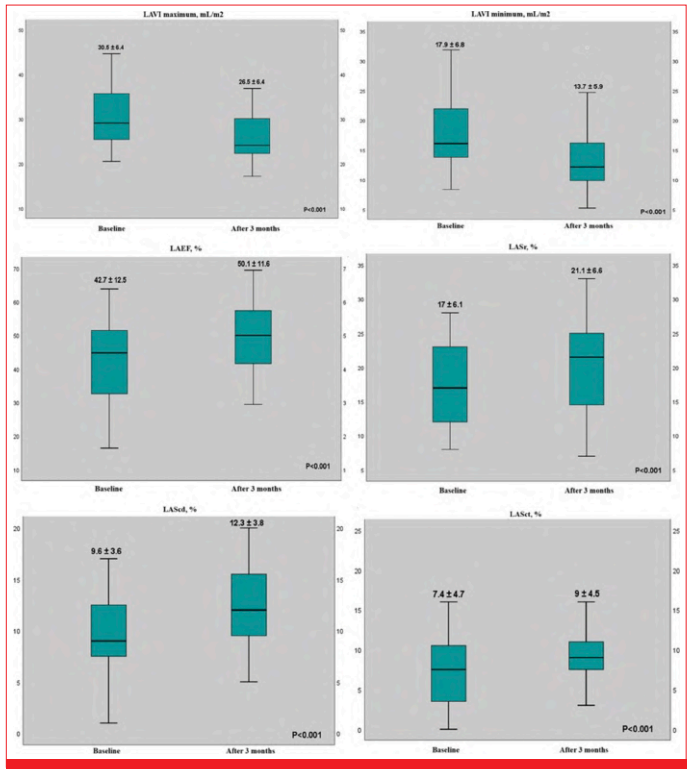

Figure 2. Left atrial functional changes 3 months after cryoablation according to SCALE-CryoAF score.

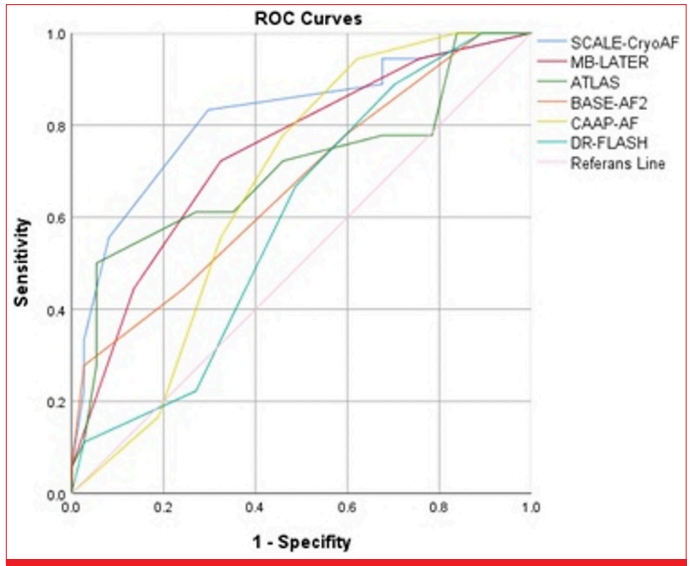

Figure 3. ROC curves of specific risk scores predicting late AF recurrence.

Table 1. Comparison of general and specific risk scores between two groups

Risk scores	All Group	Late recurrence (+) group	Late recurrence (-) group	p value
CHA2DS2-VA	2.0 (1.0, 3.0)	2.0 (1.0, 3.0)	1.5 (1.0, 3.0)	0.379
HASBLED	2.0 (1.0, 2.0)	2.0 (1.0, 2.0)	2.0 (1.0, 2.0)	0.646
SCALE-CryoAF	3.0 (2.0, 4.0)	5.0 (4.0, 7.25)	3.0 (0.5, 4.0)	<0.001
DR-FLASH	3.0 (2.0, 4.0)	3.0 (2.0, 3.25)	2.0 (1.0, 4.0)	0.254
MB-LATER	1.0 (1.0, 2.0)	2.0 (1.0, 3.0)	1.0 (0.5, 2.0)	0.003
BASE-AF2	2.0 (1.0, 3.0)	2.5 (2.0, 3.25)	2.0 (1.0, 2.5)	0.008
ATLAS	9.04 ± 3.67	10.89 ± 3.88	8.14 ± 3.25	0.008
CAAP-AF	4.0 (2.0, 5.0)	5.0 (3.75, 5.0)	3.0 (2.0, 5.0)	0.043

PP-048 [Interventional Cardiology / Coronary]

Relationship between C-reactive protein and uric acid to albumin ratio and coronary collateral circulation in patients with chronic total occlusion

Kadir Karaçalı[1], Mikail Yarlıoğlu[2], Anıl Salman[2]

[1]*Department of Cardiology, Ankara Gölbaşı State Hospital, Ankara*
[2]*Department of Cardiology, Ankara Training and Research Hospital, Ankara*

Background and Aim: C-reactive protein and uric acid to albumin ratio (CUAR) is a recent inflammatory marker associated with cardiovascular disease. We aim to investigate the relationship between CUAR and coronary collateral circulation (CCC) in patients with stable coronary artery disease (CAD) and chronic total occlusion (CTO).

Methods: The patients were divided into two groups; 267 patients with poor CCC formation group and 133 patients with well CCC formation group. CUARlog was calculated using the 'log10 (CRP x UA / Albumin)' formula.

Results: CUARlog levels were significantly higher in patients with poor CCC formation (p<0.001). CUARlog levels above 1.45 predicted poor CCC with a higher sensitivity of 70% and specificity of 67% than uric acid to albumin ratio (UAR), neutrophil to lymphocyte ratio (NLR), and platelet to lymphocyte ratio (PLR) (p<0.001). In the comparison of the AUC values, there was significant difference between the

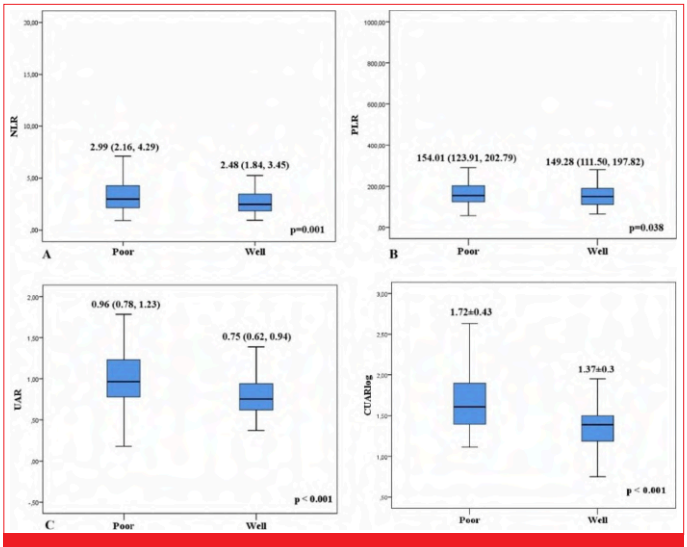

Figure 1. Comparison of CUARlog, UAR, NLR, and PLR levels of study groups.

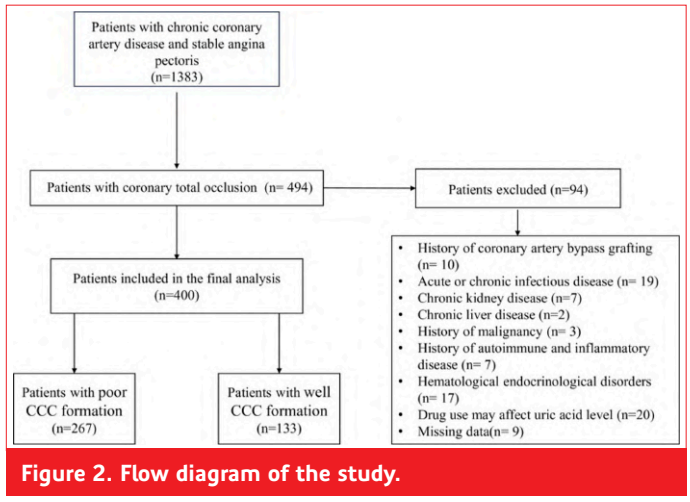

Figure 2. Flow diagram of the study.

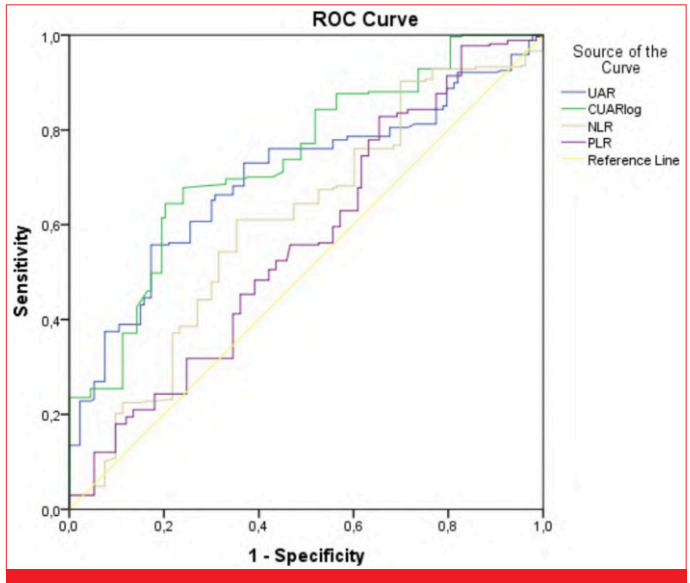

Figure 3. ROC curves of CUARlog, UAR, NLR, and PLR levels to predict poor CCC formation.

CUARlog and UAR (p=0.047), NLR (p=0.001) and PLR (p<0.001). In multivariate regression analysis, CUARlog above 1.45 (p<0.001) was associated independently with poor CCC development.

Conclusions: Our results suggested that CUARlog is more potent and independent marker than other inflammatory markers to predict poor CCC development in CTO patients. It may be useful to identify high-risk patients with poor CCC development.

PP-049 [Coronary Artery Disease / Acute Coronary Syndrome]

Prognostic value of right ventricular function in 1-year mortality after acute coronary syndrome

Selda Murat[1], Fatih Enes Durmaz[2], Rabia Okumuş[1], Bektaş Murat[3]

[1]*Department of Cardiology, Eskişehir Osmangazi University, Faculty of Medicine, Eskişehir*
[2]*Department of Cardiology, Muş State Hospital, Muş*
[3]*Eskişehir City Hospital, Eskişehir*

Background and Aim: Right ventricular (RV) function plays a crucial role in determining clinical outcomes in patients with acute coronary syndrome (ACS). Despite this, RV function has traditionally received less attention than left ventricular parameters in routine echocardiographic assessments. Recent advances in echocardiographic techniques, including strain imaging, have enabled more detailed evaluation of RV performance, which may offer valuable prognostic information. This study aimed to investigate the association between echocardiographic right ventricular function parameters and 1-year mortality in patients with ACS.

Methods: Over a 6-month period, a total of 238 patients diagnosed with STEMI, NSTEMI, or unstable angina pectoris and eligible for a 1-year follow-up were included in the study. Patient selection and exclusion criteria are summarized in the flow chart (Figure 1). Patients were divided into two groups based on 1-year follow-up:

- 1-year survivors (n=117)

- Non-survivors at 1 year (n=10)

Demographic, clinical, and echocardiographic variables were compared between groups.

Results: The overall 1-year mortality rate was 7.9%. Patients who died within one year were significantly older than survivors (77.7 ± 12.3 vs. 64.2 ± 12.0 years, p<0.001) and had higher serum creatinine levels (1.6 [1.0–3.0] vs. 1.0 [0.8–1.2] mg/dL, p=0.003).

RV function was also notably impaired among non-survivors:

- Right atrial diameter was larger (37.5 ± 3.6 vs. 34.9 ± 3.5 mm, p=0.032).
- RV systolic area was greater (9.5 ± 2.8 vs. 7.1 ± 2.2 mm^2, p=0.003).

Table 1. Comparison of 1-year survivors and non-survivors

	1-Year Survivors (n=117)	1-Year Non-Survivors (n=10)	p value
Age, years, ± SD	64.2 ± 12.0	77.7 ± 12.3	<0.001
Male, n (%)	91 (77.8%)	7 (70.0%)	0.694
HT, n (%)	71 (61.2%)	7 (70%)	0.741
DM, n (%)	38 (32.8%)	5 (50%)	0.307
History of CABG, n (%)	11 (9.5%)	3 (30%)	0.082
History of stent, n (%)	35 (30.2%)	2 (20%)	0.722
Body Mass Index (kg/m^2), [IQR]	26.5 [24.4-29.3]	26.6 [24.4-27.7]	0.498
emoglobin, g/dL, ± SD	13.4 [12.1-14.7]	11.4 [10.3-13.8]	0.074
Creatinine, mg/dL, [IQR]	1.0 [0.8-1.2]	1.6 [1.0-3.0]	0.003
LVEF, %, ± SD	49.0 ± 10.2	42.2 ± 13.4	0.054
IVS thickness, ± SD	11.7 ± 1.8	11.5 ± 1.6	0.680
Mitral e', cm/s ± SD	56.9 ± 17.2	74.2 ± 26.0	0.068
Lateral e', cm/s ± SD	7.7 ± 2.3	6.0 ± 1.9	0.027
RA diameter, mm, ± SD	34.9 ± 3.5	37.5 ± 3.6	0.032
RVDA, mm^2, ± SD	12.5 ± 2.9	14.2 ± 2.5	0.085
RVSA, mm^2, ± SD	7.1 ± 2.2	9.5 ± 2.8	0.003
RVFAC, %, ± SD	42.8 ± 9.8	33.8 ± 12.2	0.008
TAPSE, mm, ± SD	21.3 ± 3.0	20.0 ± 4.6	0.280
SPAP, mmHg	24 [20-29]	30 [25-43]	0.073
TJV, m/s	2.1 [1.8-2.4]	2.4 [2.1-3.0]	0.143
Global longitudinal strain, ± SD	-15.4 ± 4.6	-12.6 ± 6.4	0.077
RV 4CSL, % ± SD	-17.2 ± 5.5	-12.4 ± 5.3	0.011
RV FWSSL, % [IQR]	-21.2 [(-26.2)-(-16.3)]	-13.4 [(-17.9)-(-9.3)]	0.005
Length of intensive care unit, days ± SD	2 [1-3]	3.5 [2.-5]	0.004
Total hospital stay, ± SD	4 [3-5]	7 [6.2-9.5]	<0.001

- RV four-chamber longitudinal strain was significantly reduced (–12.4 ± 5.3% vs. –17.2 ± 5.5%, p=0.011).
- RV free wall strain was markedly lower (–13.4% vs. –21.2%, p=0.005).
- RV fractional area change (RV FAC) was significantly lower in non-survivors compared to survivors (33.8 ± 12.2% vs. 42.8 ± 9.8%, p=0.008), suggesting reduced RV systolic performance in the mortality group.

In addition, non-survivors had longer of intensive care unit (ICU) stays (3.5 [2.0–5.0] vs. 2.0 [1.0–3.0] days, p=0.004) and total hospitalizations (7.0 [6.2–9.5] vs. 4.0 [3.0–5.0] days, p<0.001).

Conclusions: Right ventricular strain parameters were significantly associated with 1-year mortality, highlighting their prognostic value in acute coronary syndrome. However, the small size of the non-survivor group requires cautious interpretation of these findings.

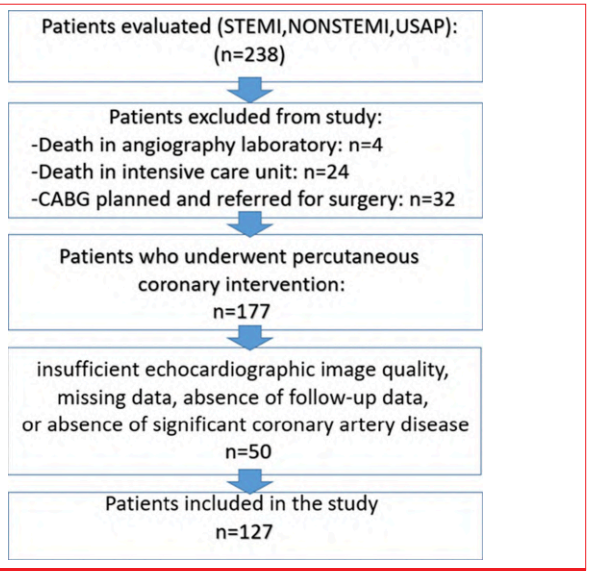

Figure 1. Flow chart of patient selection and exclusion criteria.

PB-050 [Diğer]

Could the Tpeak-Tend interval be an electrocardiographic risk marker for predicting extensive late gadolinium enhancement in patients with hypertrophic cardiomyopathy?

Meltem Tekin, Büşra Kuru Görgülü, Arda Güler, Kadir Sadıkoğlu, İrem Türkmen, Hasan Şahin, Büşra Çörekçioğlu, Sinem Aydın, Gamze Babür Güler

Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul

Background and Aim: Hypertrophic cardiomyopathy (HCM) is a common inherited heart disease. In patients with HCM, extent of myocardial replacement fibrosis with late gadolinium enhancement (LGE) that is assessed by cardiac magnetic resonance (CMR) represents 2-fold increase in sudden cardiac death (SCD) risk. Electrocardiographic markers reflecting transmural dispersion of repolarization, has been used to predict ventricular tachycardia/fibrillation (VT/VF) and sudden cardiac death in different clinical settings. We evaluated the electrocardiogram (ECG) parameters to estimate extensive LGE in patients with HCM.

Methods: Between January 2018 and December 2024, 705 consecutive HCM patients who were diagnosed and followed up at our center were evaluated. Eligibility criteria included ≥18-year-old patients with hypertrophic cardiomyopathy diagnosis that was confirmed by TTE and CMR. Baseline characteristic features and ECG parameters that included heart rate, QRS width, QT and QTc intervals, frontal QRS-T angle, Tpeak-Tend interval, were evaluated for all patients. Extensive myocardial fibrosis was assumed more than 15% LGE on CMR. In this study, ROC analysis was used to determine the appropriate threshold value for the TPTE (Tpeak to Tend) parameter to detect the presence of widespread LGE. Youden index was used to determine the most appropriate threshold value in the ROC curve.

Results: A total of 705 patients with hypertrophic cardiomyopathy were evaluated retrospectively. Of these, 107 patients were excluded due to: insufficient ECG, TTE and/or CMR data (n=33), systemic disorders causing left ventricular hypertrophy (n=42) and left ventricular hypertrophy secondary to severe aortic stenosis (n=65). Patients were stratified into two groups based on LGE burden on CMR; Group 1: Extensive LGE (≥15% of LV mass, n=143; 25.3%), Group 2: Non-extensive LGE (<15%, n=422; 74.7%). QT and QTc intervals, frontal QRS-T angle, and TpTe/QT ratio were not significantly different between the two groups. However, patients with extensive LGE demonstrated a significantly longer QRS duration (103.2 ± 22.5 ms vs. 98.1 ± 22.0 ms, p=0.01) and a longer TpTe interval (76.3 ± 29.8 ms vs. 69.7 ± 28.8 ms, p=0.024). In our study, the performance of TpTe value in predicting the presence of extensive LGE was evaluated by ROC curve analysis. The area under the area (AUC) value obtained as a result of ROC analysis was found to be 0.564. This result shows that TpTe has a low level of discrimination power in distinguishing the presence of extensive LGE. According to the coordinates of the ROC curve, the most appropriate cut-off value was determined as TpTe ≥70 ms. For this cut-off value, sensitivity was calculated as 67.1% and specificity as 47.4%.

Conclusions: In conclusion, the TpTe value has a limited predictive value in predicting the presence of extensive LGE when used alone, and it is thought that it should be evaluated together with other parameters in clinical practice.

Table 1. Electrocardiographic parameters in studied patients groups

	Extensive LGE (n:143)	Non-extensive LGE (n:422)	p- Value
QRS duration	103,2 ±22,5	98,1 ± 22,0	0,01
QT interval	416± 41	408 ± 43	0,07
QTc interval	450 ±33	445 ±33	0,11
P axis	50 (-27,266)	52 (-29,104)	0,38
R axis	14 (-83,270)	13 (-79,212)	0,55
T axis	129 (-79,267)	100 (-85,264)	0,004
TpTe interval	77,5 ± 29,4	69,2 ± 28,4	0,004
Tpte/QTc	0,16 ± 0,06	0,15 ± 0,06	0,057
QRS-T angle	112 ±63	113 ± 59	0,986

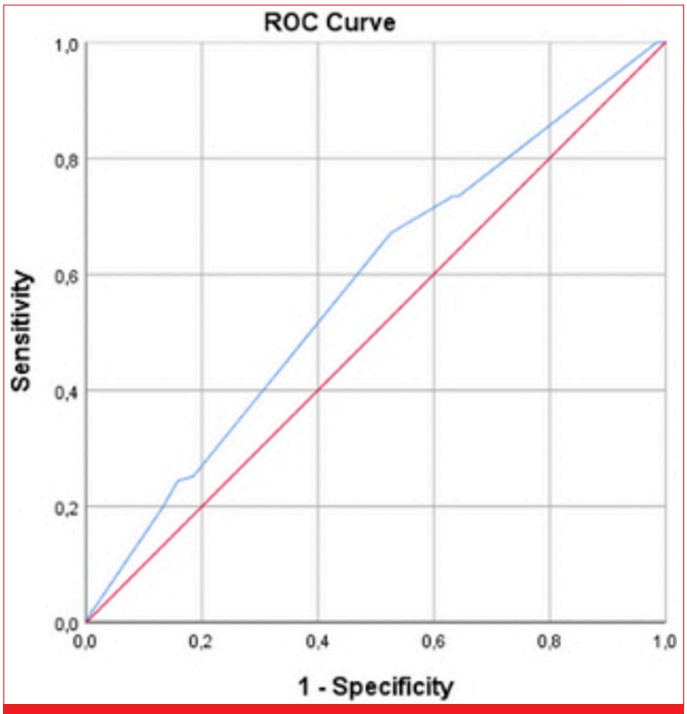

Figure 1. ROC curve analyses.

PB-051 [Cardiac Imaging / Echocardiography]

Evaluation of the effects of SGLT2 inhibitors on transcranial doppler parameters in patients with heart failure

Murat Demirci[1], Burcu Özdemir[2], Beste Özben[1]

[1]*Department of Cardiology, Marmara University, Faculty of Medicine, İstanbul*
[2]*Department of Radiology, Marmara University, Faculty of Medicine, İstanbul*

Background and Aim: Sodium-glucose cotransporter-2 (SGLT2) inhibitors are used in heart failure (HF) for reducing cardiovascular morbidity and mortality. They also have favorable effects on vascular resistance, arterial stiffness, and endothelial function. However, their impact on cerebral circulation remains unclear. Transcranial Doppler (TCD) is a non-invasive method for evaluating cerebral circulation. The Pulsatility Index (PI) and Resistance Index (RI) reflect microvascular resistance and are considered more reliable than flow velocity–based parameters such as maximum systolic velocity (V_{max}) and minimum diastolic velocity (V_{min}). This study aimed to assess the effects of SGLT2 inhibitor therapy on cerebral hemodynamic parameters in HF patients using TCD.

Methods: This study was conducted with an observational design and was approved by the local ethics committee. Consecutive patients with a clinical diagnosis of HF who were initiated on SGLT2 inhibitor therapy were included. Baseline data recorded included ejection fraction (EF), demographic characteristics, comorbidities, medications, and laboratory parameters. Patients were classified into two subgroups according to EF: EF <50% and EF ≥50%. All patients underwent TCD examinations at baseline and at 3 months. Measurements were performed in the middle cerebral artery (MCA) with the sample volume placed parallel to the flow direction, and V_{max}, V_{min}, PI, and RI were traced throughout the cardiac cycle and automatically calculated by the device. Two recordings were obtained from each patient, and their mean values were included in the analysis (Figure 1).

Table 1. Demographic, clinical, and baseline medication characteristics of patients

Parameter	Value
Age (years)	63 ± 9
Male	25 (64.1%)
Body mass index (kg/m²)	30 ± 5
Systolic blood pressure (mmHg)	134 ± 19
Diastolic blood pressure (mmHg)	82 ± 11
Heart rate (beats/min)	72 ± 9
Heart Failure Classification	
HFrEF	9 (23.1%)
HFmrEF	12 (30.7%)
HFpEF	18 (46.2%)
Diabetes Mellitus	13 (33.3%)
Hypertension	17 (43.6%)
History of MI	32 (82.1%)
History of s PCI or PTCA	28 (71.8%)
History of CABG	7 (17.9%)
Medications at Baseline	
Dapagliflozin	24 (61.5%)
Empagliflozin	15 (38.4%)
ACEi	18 (46.1%)
ARB	11 (28.2%)
ARNI	4 (10.2%)
Beta blockers	21 (53.8%)
MRA	10 (25.6%)
Statins	28 (71.8%)
Diuretics	13 (33.3%)

ACEi: Angiotensin-converting enzyme inhibitors, ARB: Sngiotensin receptor blockers, ARNI: Angiotensin receptor–neprilysin inhibitors, CABG: Coronary artery bypass grafting, HFmrEF: Heart failure with mildly reduced ejection fraction, HFpEF: Heart failure with preserved ejection fraction, HFrEF: Heart failure with reduced ejection fraction, MI: Myocardial infarction, MRA: Mineralocorticoid receptor antagonists, PCI: Percutaneous coronary intervention, PTCA: Percutaneous transluminal coronary angioplasty.

Table 2. Baseline laboratory parameters of patients

Parameter	Beginning
Brain Natriuretic Peptide (BNP) (pg/mL)	107 (60–158)
White Blood Cell Count (x10³/μL)	8.4 ± 2.0
Hemoglobin (g/dL)	13.4 ± 1.3
Creatinine (mg/dL)	0.93 ± 0.25
LDL Cholesterol (mg/dL)	94.6 ± 31.8

Values are expressed as mean ± standard deviation or median (interquartile range), as appropriate.

Table 3. Comparison of TCD parameters at baseline and after 3 months of SGLT2 inhibitor therapy in the overall cohort and EF subgroups

Parameter	Baseline	3rd month	p value
Total (n=39)			
Vmax (cm/s)	66.9 ± 18.2	69.2 ± 20.0	0.578
Vmin (cm/s)	23.3 ± 8.1	25.4 ± 8.1	0.198
PI	1.11 ± 0.25	1.02 ± 0.18	0.002
RI	0.62 ± 0.07	0.62 ± 0.06	0.520
EF <50% (n=21)			
Vmax (cm/s)	65.9 ± 20.8	66.7 ± 21.4	0.893
Vmin (cm/s)	22.8 ± 8.5	23.2 ± 7.0	0.467
PI	1.14 ± 0.21	1.05 ± 0.16	0.017
RI	0.63 ± 0.07	0.63 ± 0.05	0.729
EF ≥50% (n=18)			
Vmax (cm/s)	68.1 ± 15.3	72.1 ± 18.2	0.500
Vmin (cm/s)	23.9 ± 7.8	27.9 ± 8.6	0.077
PI	1.09 ± 0.30	0.97 ± 0.19	0.036
RI	0.62 ± 0.08	0.61 ± 0.07	0.266

EF: Ejection Fraction, PI: Pulsatility Index; RI: Resistance Index; Vmax: Maximum systolic velocity; Vmin: Minimum diastolic velocity.

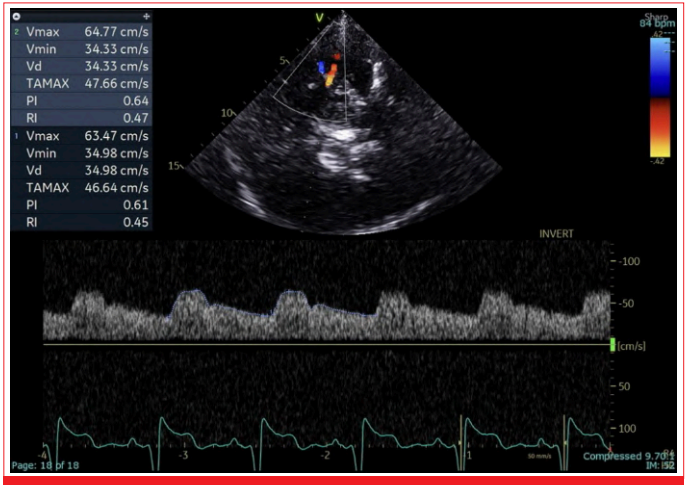

Figure 1. TCD measurement of the middle cerebral artery (MCA), demonstrating the spectral Doppler waveform with simultaneous ECG tracing. Hemodynamic parameters including maximum systolic velocity (Vmax), minimum diastolic velocity (Vmin), pulsatility index (PI), and resistance index (RI) are shown.

Results: A total of 39 patients were included in the study (mean age 63 ± 9 years, 64.1% male). The clinical and demographic characteristics of the patients are summarized in Table 1, and laboratory findings are presented in Table 2. PI showed a significant reduction after 3 months of treatment in the overall cohort (baseline: 1.11 ± 0.25; 3rd month: 1.02 ± 0.18; p=0.002). Although Vmax and Vmin tended to increase, these changes were not statistically significant (p=0.578 and p=0.198, respectively). RI values remained unchanged between baseline and 3 months (baseline: 0.62 ± 0.07; 3rd month: 0.62 ± 0.06; p=0.520). In subgroup analyses, both patients with EF <50% (baseline: 1.14 ± 0.21; 3rd month: 1.05 ± 0.16; p=0.017) and those with EF ≥50% (baseline: 1.09 ± 0.30; 3rd month: 0.97 ± 0.19; p=0.036) demonstrated a significant decrease in PI. In both subgroups, V_{max}, V_{min}, and RI values remained similar (p>0.05). The comparison of baseline and 3rd-month TCD parameters is presented in Table 3.

Conclusions: We found that SGLT2 inhibitors significantly reduced PI values, as measured by TCD in patients with HF, suggesting improvements in cerebral circulation. In our study, this effect was observed in both EF <50% and EF ≥50% subgroups. These findings indicate that SGLT2 inhibitors may be beneficial in reducing cerebrovascular risk in patients with HF.

PP-052 [Interventional Cardiology / Valvular and Structural Heart Disease]

Clinical and biochemical predictors of high-grade atrioventricular block following transcatheter aortic valve implantation: A single-center study

Aykan Çelik

Department of Cardiology, İzmir Atatürk Training and Research Hospital, İzmir

Background and Aim: High-grade atrioventricular block (AVB) is among the most common conduction disturbances following transcatheter aortic valve implantation (TAVI), frequently requiring permanent pacemaker implantation. Prior studies have investigated procedural and anatomical determinants -such as preprocedural electrocardiographic abnormalities, implantation depth, annular dimensions, and balloon predilation- as potential contributors to post-TAVI conduction impairment. However, these parameters are not routinely assessed or may vary significantly between centers. This study aimed to evaluate readily available clinical, echocardiographic, and biochemical parameters as predictors of high-grade AVB occurring within 30 days after TAVI.

Methods: This retrospective, single-center study was conducted at a tertiary academic medical center. A total of 376 patients who underwent TAVI between January 2010 and December 2023 were included. Patients were categorized into two groups based on the occurrence of high-grade AVB within 30 days post-procedure. Demographic, clinical, echocardiographic, and biochemical data were retrieved from electronic health records. Categorical variables were compared using the chi-square test, and continuous variables using the independent samples t-test. Variables with a p<0.10 in univariate logistic regression were included in the multivariate model. Statistical significance was set at p<0.05. Analyses were conducted using SPSS version 30.0 and R version 4.1.2.

Results: High-grade AVB developed in 55 patients (14.6%). Compared to the AVB- group, AVB+ patients had significantly higher rates of hypertension (81.8% vs. 64.2%, p=0.010), diabetes mellitus (45.5% vs. 31.2%, p=0.037), and smoking history (7.3% vs. 2.2%, p=0.038). Echocardiographic assessments revealed higher mean and peak aortic gradients in the AVB+ group (mean: 48.96 vs. 44.78 mmHg, p=0.039; peak: 79.8 vs. 73.5 mmHg, p=0.041). Total cholesterol (187.6 ± 58.6 vs. 167.8 ± 48.1 mg/dL, p=0.007) and LDL-C (113.7 ± 47.4 vs. 99.8 ± 36.4 mg/dL, p=0.041) were also significantly higher in the AVB+ group.

Systemic inflammatory markers—including the systemic immune-inflammation index (SII), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and lymphocyte-to-monocyte ratio (LMR)—did not differ significantly between groups. In multivariate analysis, hypertension (odds ratio [OR]=2.15; 95% confidence interval [CI]: 1.012–4.55; p=0.047) and total cholesterol (OR=1.007; 95% CI: 1.002–1.013; p=0.010) were identified as independent predictors.

Conclusions: Hypertension and elevated total cholesterol were identified as independent predictors of high-grade AVB following TAVI. Integration of these routinely available parameters into preprocedural evaluations may help identify patients at increased risk and improve postprocedural monitoring strategies.

Table 1. Demographic and clinical characteristics of patients by high-grade atrioventricular block status

Characteristics	AVB- (n=321)	AVB+ (n=55)	p value
Age (years)	76.37 ± 8.12	77.78 ± 7.39	0.228
Gender (female), n (%)	159 (49.5%)	31 (56.4%)	0.349
DM, n (%)	100 (31.2%)	25 (45.5%)	0.037
HT, n (%)	206 (64.2%)	45 (81.8%)	0.010
Dyslipidemia, n (%)	88 (27.4%)	11 (20%)	0.249
Smoking, n (%)	7 (2.2%)	4 (7.3%)	0.038
CAD, n (%)	271 (84.4%)	45 (81.8%)	0.626
History of Heart Valve Surgery, n (%)	10 (3.1%)	2 (3.6%)	0.839
Heart Failure, n (%)	72 (22.4%)	6 (10.9%)	0.052
Previous Stroke, n (%)	23 (7.2%)	2 (3.6%)	0.332
CKD, n (%)	86 (26.8%)	16 (29.1%)	0.723
GFR (mL/min)	59.30 ± 18.75	60.38 ± 18.65	0.691
LDL (mg/dL)	99.75 ± 36.42	113.72 ± 47.35	0.041
HDL (mg/dL)	44.84 ± 12.44	42.56 ± 11.10	0.204
TG (mg/dL)	121.32 ± 95.09	161.18 ± 183.73	0.121
Total Cholesterol (mg/dL)	167.81 ± 48.14	187.63 ± 58.57	0.007
WBC (10^3/uL)	7.40 ± 2.38	7.73 ± 2.50	0.357
PLT (10^3/uL)	221.36 ± 83.65	201.05 ± 79.53	0.095
HG (g/dL)	11.87 ± 1.73	11.19 ± 1.94	0.691
NEU (10^3/uL)	6.10 ± 2.87	6.49 ± 2.70	0.348
LYM (10^3/uL)	1.40 ± 0.64	1.50 ± 1.14	0.506
Mono (10^3/uL)	0.74 ± 1.81	0.70 ± 0.25	0.899
SII	1145 ± 895	1091 ± 815	0.680
NLR	5.51 ± 4.61	5.58 ± 3.43	0.925
PLR	182.98 ± 95.62	169.93 ± 98.67	0.352
LMR	2.43 ± 1.43	2.28 ± 1.23	0.463
LVEF (%)	53.47 ± 10.88	55.01 ± 10.85	0.330
Aort Mean Gradient (mmHg)	44.78 ± 14.02	48.96 ± 12.87	0.039

DM: Diabetes mellitus; HT: Hypertension; CAD: Coronary artery disease; CKD: Chronic kidney disease, GFR: Glomerular filtration rate; LDL: Low density lipoprotein; HDL: High density lipoprotein; TG: Triglyceride; WBC: White blood cell; PLT: Platelet; Hg: Hemoglobin; NEU: Neutrophile; LYM: Lymphocyte; Mono: Monocyte, SII: Systemic immune inflammation index; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; LMR: Lymphocyte-to-monocyte ratio; LVEF: Left ventricular ejection fraction.

Table 2. Univariate and multivariate logistic regression results for high-grade atrioventricular block

	Univariate			Multivariate		
Variable	OR	95% CI	p	OR	95% CI	p
Diabetes Mellitus	1.84	1.30-3.30	0.039	1.64	0.89-3.03	0.116
Hypertension	2.51	1.22-5.18	0.012	2.15	1.012-4.55	0.047
Smoking	5.19	1.00-12.45	0.051	3.35	0.882-12.74	0.076
Total Cholesterol	1.007	1.002-1.013	0.008	1.007	1.002-1.013	0.010

OR: Odds ratio; CI: Confidence interval.

PP-053 [Interventional Cardiology / Valvular and Structural Heart Disease]

In-hospital impact of aortic and peripheral tortuosity on procedural success and vascular complications in transcatheter aortic valve implantation

İlker Gül[1], Oktay Şenöz[1], Şahin Bozok[2], Ecem Gürses[1], Ömer Faruk Rahman[2], Ahmet Anıl Başkurt[1], Yusuf Demir[1], İlhan Koyuncu[1], Ferhat Siyamend Yurdam[1], Şirin Şebnem Önür[1], Serhat Agüloğlu[1], İbrahim Duygu[1], Muhammed Rohat Çiçek[1], Mustafa Sezen[1]

[1]Department of Cardiology, Bakırçay University, Çiğli Training and Research Hospital, İzmir
[2]Department of Cardiovascular Surgery, Bakırçay University, Çiğli Training and Research Hospital, İzmir

Background and Aim: Transcatheter aortic valve implantation (TAVI) is a well-established treatment for severe aortic stenosis. Anatomical characteristics of the aorta and peripheral arteries directly influence procedural success. Vascular tortuosity remains an underexplored parameter in TAVI outcomes. This study aimed to investigate the significance of the tortuosity index (TI) in TAVI patients and its relationship with procedural success and vascular complications.

Methods: A total of 262 patients undergoing TAVI were retrospectively analyzed. TI was calculated in two planes from computed tomography angiography: (1) femoral bifurcation to aortic bifurcation, and (2) aortic bifurcation to aortic arch (Figure 1). Patients were classified into low TI (LTI, ≤1.5) and high TI (HTI, >1.5) groups. Demographic, laboratory, imaging, and procedural data were compared. ROC analysis was performed to identify the TI cut-off predicting increased peripheral complications. All TAVI procedures were performed under fluoroscopic guidance with standard closure devices. Procedural time, contrast volume, and device type were recorded. Continuous variables were compared using t-tests or Mann–Whitney U tests, categorical variables with χ^2 or Fisher's exact tests. ROC analysis determined TI cut-off predicting peripheral complications.

Results: Of 262 patients, 172 were in the LTI group and 90 in the HTI group. No significant differences were observed in age, BMI, gender,

or valve type. Vascular closure device failure was significantly higher in HTI patients (8.9% vs. 2.1%, p<0.001), leading to increased need for surgical or endovascular repair. Contrast-induced nephropathy (CIN) was more frequent in the HTI group, as was pericardial effusion (numerical increase) (Table 1 and Table 2). TI positively correlated with contrast volume and need for stiffer guidewires. ROC analysis identified a TI ≥1.8 as the optimal threshold for predicting peripheral complications (sensitivity 77%, specificity 73%). Mortality rates were similar between groups.

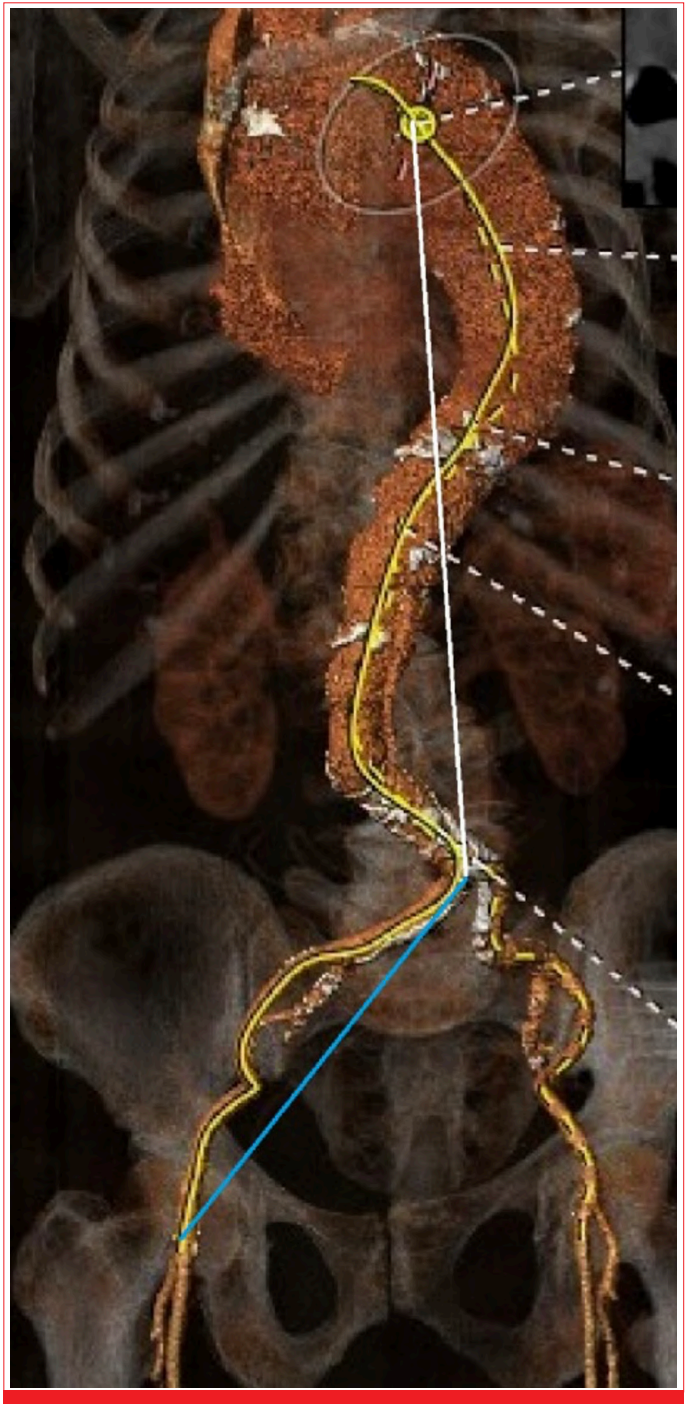

Figure 1. The calculation of aortic, iliac and femoral tortuosity indexes.

Conclusions: Vascular tortuosity significantly impacts procedural outcomes in TAVI. This study demonstrates that higher vascular tortuosity is associated with increased closure device failure, CIN, and procedural challenges in TAVI. The observed TI cut-off of 1.8 may be clinically relevant for pre-procedural planning. Selecting alternative access routes such as transaxillary or transcarotid approaches, or using advanced closure devices, could reduce vascular and other complications in high-TI patients. These findings are consistent with prior vascular interventions literature but highlight the unique technical demands of TAVI procedures.

Table 1. Baseline characteristics of the study population

Variable	LTI (n=172)	HTI (n=90)	p value
Age (years)	78.4 ± 6.5	79.1 ± 6.3	0.32
Male (%)	48.8	51.1	0.72
BMI (kg/m²)	27.3 ± 4.1	27.5 ± 4.3	0.68
Valve type (self-expandable %)	62.2%	60.0%	0.81
GFR	57.2 ± 13.8	56.5 ± 18.9	0.77
Creatinine (mg/dL)	1.12 ± 0.21	1.19 ± 0.33	0.48
Contrast volume (mL)	125.2 ± 38.3	130.1 ± 41.1	0.39

Table 2. Procedural and post-procedural outcomes

Outcome	LTI (%)	HTI (%)	p value
Closure device failure	2.3	8.9	<0.001
CIN	5.8	12.2	0.04
Pericardial effusion	3.4	6.7	0.21
Mortality (in-hospital)	2.7	3.4	0.78

PP-054 [Interventional Cardiology / Coronary]

Diagnostic performance of coronary computed tomography angiography-CT-QFR with invasive coronary angiography-FFR

Erdoğan İlkay[1], Ersin Sarıçam[1], Zeynep Şeyma Turinay[1], Yasemin Sağlam[2], Selin Ardalı Düzgün[3], Tuncay Hazırolan[3]

[1]Department of Cardiology, Medicana International Ankara Hospital, Ankara
[2]Department of Cardiology, Ankara Güven Hospital, Ankara
[3]Department of Radiodiagnostics, Hacettepe University, Faculty of Medicine, Ankara

Background and Aim: Coronary computed tomography angiography (CTA) is recommended for diagnosis of obstructive coronary artery disease (CAD) in patients with low -intermediate pre-test risk as class I recommend according to ESC guidelines. Although an excellent negative predictive value for ruling out obstructive CAD, coronary CTA's positive predictive value remains unsatisfactory. CTA-derived quantitative flow ratio (CT-QFR) is new non-invasive technique like invasive fractional flow reserve (FFR). This high-tech method has lastly been used for on-site evaluation of patients with suspected mild-moderate obstructive coronary artery disease. We

aimed to compare the diagnostic performance of CT-QFR and invasive coronary angiography- FFR in patients with 50%-70% stenosis after coronary CTA.

Methods: In this prospective study, patients were referred to invasive coronary angiography if coronary CTA was ≥50% diameter stenosis. The patient with between <70% and ≥50% on CTA underwent CT-QFR and invasive fractional flow reserve procedure. We compared CT-QFR with coronary invasive FFR. Exclusion criteria included previous percutaneous coronary intervention (PCI)/coronary artery bypass grafting (CABG), contraindication for adenosine. This study included 39 patients between 2024-2025. On-site CT-QFR was analyzed post-hoc blinded to angiographic data and obtained as both distal (MD-QFR) and lesion-specific CT-QFR (LS-QFR). Abnormal CT-QFR was defined as 0.80 diagnostic cut-off. Hemodynamically obstructive CAD was defined as invasive FFR 0.80 as gold-standard.

Results: In the ROC curves of per-vessel analysis of both CT-QFR and invasive FFR, CTQFR were detected as negative predictive value 0.90; positive predictive value 0.62; respectively (Figure).

Conclusions: The diagnostic performance of CT-QFR seems to be effective in the evaluation of the patients with diameter stenosis between 70% and ≥50% on coronary CTA.

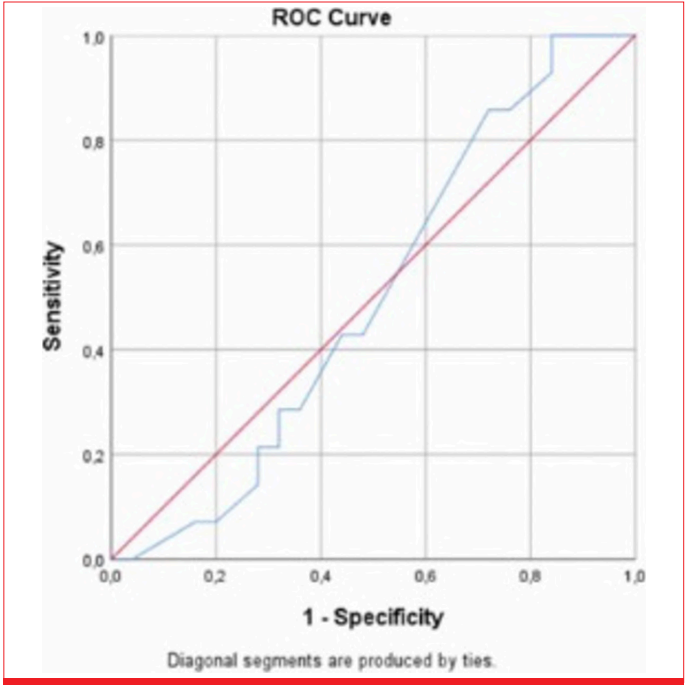

Figure 1. In the ROC curves of per-vessel analysis of both CT-QFR and invasive FFR.

PP-055 [Lipid / Preventive Cardiology]

Lipid lowering therapy use among familial hypercholesterolemia patients: Preliminary results from AHP-Muğla Study

Özcan Başaran[1], Emine Neşe Yeniçeri[2], Elif Aylin Yüce Yörük[3], Gülçin Özkan Onur[2], Aynur Özdemir[2], Aysel Arslan[1]

[1]*Department of Cardiology, Muğla Sıtkı Koçman University, Faculty of Medicine, Muğla*
[2]*Department of Family Medicine, Muğla Sıtkı Koçman University, Faculty of Medicine, Muğla*
[3]*Department of Public Health, Muğla Provincial Health Directorate, Muğla*

Background and Aim: Familial hypercholesterolemia (FH) is a major risk factor for cardiovascular disease. If left untreated it can lead to a significant shortening of life expectancy. However early diagnosis and lifelong management of high cholesterol levels could mitigate this risk and a person with FH could achieve an anticipated lifespan similar to that of a person without the condition. We sought to investigate lipid lowering therapy (LLT) use among patients with FH in southwest of Türkiye.

Methods: We planned to estimate FH prevalance in Muğla with collaboration of family physicians. Patients who were registered to family physicians, >18 years old, agree to participate were randomly enrolled to the study. A total of 10.000 patient enrollment was planned according to geographical distribution. A web-based database to collect patient demographics was developed and Dutch Lipid Clinic criteria was used to identify patients with FH. A score of ≥3 was used for the current analysis. The patients with triglycerides <400 mg/dl were grouped according to LLT use or non-use.

Results: Of the 1100 patients enrolled to date 195 (17.72%) were identified as candidates for FH. Of the patients with FH the mean age was 55.43 ± 12.94, and 111 (56.9%) were female while 56 (28.71%) were on LLT. Patients who were on LLT were older (59.17 ± 11.08 vs. 53.92 ± 13.36, p=0.010) and had higher rate of comorbidities such as hypertension, diabetes and coronary artery disease. However gender, smoking, alcohol use and exercise did not have any impact on LLT use (Table.) Of the patients who were on LLT 46 (82.10%) were on statin therapy alone while the remaining were on any combination with ezetimibe or fibrates. Patients who were on LLT had a better LDL-C control (Table).

Table 1. Demographic and laboratory parameters of FH patients by LLT use

Demographics	All patients (n=195)	LLT use (n=56)	No LLT (n=139)	p
Age	55.43±12.94	59.17±11.08	53.92±13.36	0.010
BMI	28.09±5.25	28.61±4.60	27.88±5.49	0.382
Female (%)	111 (56.9)	30 (53.6)	81 (58.3)	0.549
Smoking (%)	53 (27.2)	11 (19.6)	42 (30.2)	0.133
Alcohol (%)	36 (18.5)	12 (21.4)	24 (17.3)	0.498
Exercise (%)	71 (36.4)	19 (33.9)	52 (37.4)	0.648
HT (%)	81 (41.5)	36 (64.3)	45 (32.4)	<0.001
DM (%)	53 (27.2)	25 (44.6)	28 (20.1)	0.001
CAD (%)	16 (8.2)	13 (23.2)	3 (2.2)	<0.001
MI (%)	9 (4.6)	7 (12.5)	2 (1.4)	0.003
Laboratory parameter				
Total-C	237.90±58.45	254.47±53.85	196.78±48.60	<0.001
LDL-C	150.05±53.49	166.48±47.53	109.28±45.28	<0.001
HDL-C	57.39±13.39	58.85±13.55	53.71±12.34	0.014
Triglyserides	157.61±74.14	165.03±76.79	154.62±73.11	0.376

Conclusions: Heart diseases due to atherosclerosis are the leading cause of death in Türkiye and worldwide. Our study showed LLT use was not at desired level among FH patients. There were even patients who were not on any LLT despite having myocardial infarction or coronary artery disease. There is a need for raising awareness of FH and better management strategies for these patients.

PP-056 [Cardiac Imaging / Echocardiography]

Inflammatory and nutritional indices as predictors of myocardial scar on cardiac MRI in hypertrophic cardiomyopathy: A retrospective, single-center study

Nazlı Turan Şerifler, Emine Şeyma Destici

Department of Cardiology, Ankara Etlik City Hospital, Ankara

Background and Aim: Hypertrophic cardiomyopathy (HCM) is a genetic myocardial disorder characterized by left ventricular hypertrophy and myocardial fibrosis. In recent years, systemic inflammation has been increasingly recognized as an important modulator of disease severity. Chronic low-grade inflammation, frequently observed in HCM, is known to trigger adverse ventricular remodeling processes. This study aims to evaluate the relationship between inflammatory and nutritional indices and the presence of myocardial scar detected by cardiac magnetic resonance imaging in patients with HCM.

Methods: In this retrospective study, 197 patients with hypertrophic cardiomyopathy (HCM) were analyzed. Laboratory parameters included neutrophil, lymphocyte, platelet counts, C-reactive protein (CRP), albumin, and sedimentation rate. Inflammatory indices [SII, SIRI, NLR, MLR, AISI, MI-1, EII, PLR] and nutritional indices [PNI, CAR, albumin] were calculated. Myocardial scar was defined by late gadolinium enhancement (LGE) on cardiac magnetic resonance imaging (CMR). The scar score was determined by assigning 1 point for hs-Troponin T ≥14 ng/L, NT-proBNP ≥125 pg/mL, CAR >0.05, and PNI ≤52. Associations with LGE were assessed using logistic regression and ROC analysis.

Results: LGE was present in 127 patients (64.5%). The LGE-positive group had higher MI-1, CAR, and EII, and lower PNI (all p<0.05). In multivariate analysis, scar score (OR: 1.798, p=0.001), sedimentation rate (OR: 1.115, p=0.006), and interventricular septal thickness (OR: 4.179, p=0.005) independently predicted LGE. ROC analysis showed the scar score had the highest diagnostic performance (AUC=0.751, 95% CI: 0.685–0.810, p<0.001; cut-off ≥2: 52% sensitivity, 87.1% specificity), followed by NT-proBNP ≥125 pg/mL (AUC=0.696), hs-Troponin T ≥14 ng/L (AUC=0.631), CAR (AUC=0.618), and PNI (AUC=0.624) (all p≤0.001).

Conclusions: Higher MI-1, CAR, and EII levels, along with lower PNI, were significantly associated with the presence of myocardial scar detected by LGE in patients with HCM. Among these, the scar score developed in our study [hs-Troponin T ≥14 ng/L, NT-proBNP ≥125 pg/mL, CAR >0.05, PNI ≤52] demonstrated the highest diagnostic value as an independent predictor. Given the gradual progression of myocardial scarring in HCM, incorporating these inflammatory and nutritional markers, particularly the scar score, into early risk assessment may enable timely initiation of targeted therapeutic strategies and potentially slow scar progression.

Table 1. Demographic and clinical data according to negative and positive late gadolinium enhancement (LGE)

Parameter	LGE (-)	LGE (+)	*p-value*
Age (years)	56 ± 19	56 ± 15	0.938
Male (%)	70.0	67.7	0.741
Albumin (g/L)	44.0 ± 4.4	42.0 ± 8.0	<0.001
CRP (mg/L)	2.0 ± 3.3	4.0 ± 4.0	0.003
NT-proBNP	111 ± 293	345 ± 636	<0.001
hs-TnT (ng/L)	11.7 ± 18.3	40.6 ± 110.4	0.005
ESR	4.0 ± 4.0	8.0 ± 8.0	<0.001
IVS (cm, Echo)	1.5 ± 0.4	1.7 ± 0.5	<0.001

Abbreviations: **CRP**, C-reactive protein; **NT-proBNP**, N-terminal pro–B-type natriuretic peptide; **IVS**, interventricular septal; **Scar Score**, composite score based on hs-Troponin T ≥14 ng/L, NT-proBNP ≥125 pg/mL,

Table 2. Inflammatory and nutritional indices according to late gadolinium enhancement (LGE) status

	LGE (-) n=70	LGE (+) n=127	*p-value*
SII	488.3 ± 375.9	492.2 ± 416.0	0.893
SIRI	1.13 ± 1.09	1.14 ± 1.01	0.861
NLR	1.95 ± 1.23	2.00 ± 1.52	0.548
MLR	0.28 ± 0.13	0.25 ± 0.14	0.469
AISI	276.5 ± 302.4	273.0 ± 256.0	0.651
MI-1	4.57 ± 7.43	7.74 ± 11.58	**0.007**
CAR	0.05 ± 0.08	0.09 ± 0.12	**0.001**
EII	256.6 ± 422.5	410.8 ± 498.8	**0.011**
PLR	104.5 ± 43.5	103.4 ± 63.7	0.518
PNI	55.0 ± 6.5	53.5 ± 9.0	**0.004**

Abbreviations: **AISI**, aggregate index of systemic inflammation; **CAR**, C-reactive protein-to-albumin ratio; **EII**, platelet-to-lymphocyte ratio × C-reactive protein; **MI-1**, neutrophil-to-lymphocyte ratio × C-reactive protein; **MLR**, monocyte-to-lymphocyte ratio; **NLR**, neutrophil-to-lymphocyte ratio; **PLR**, platelet-to-lymphocyte ratio; **PNI**, prognostic nutritional index; **SII**, systemic immune-inflammation index; **SIRI**, systemic inflammation response index.

Table 3. Inflammatory indices across LGE burden categories

	LGE <5% n=67	5%≤ LGE <15% n=40	LGE ≥15% n=20	*p-value*
SII	479.1 ± 348.3	526.2 ± 417.8	590.0 ± 745.6	0.174
SIRI	1.12 ± 0.93	1.03 ± 1.21	1.45 ± 1.33	**0.037**
NLR	1.89 ± 1.03	2.11 ± 1.75	2.89 ± 1.87	0.162
MLR	0.26 ± 0.14	0.22 ± 0.15	0.28 ± 0.22	0.565
AISI	260.7 ± 221.3	272.5 ± 269.5	328.8 ± 386.5	0.121
MI-1	6.32 ± 9.15	6.07 ± 7.32	12.86 ± 16.16	**0.002**
CAR	0.08 ± 0.10	0.09 ± 0.10	0.20 ± 0.14	**0.001**
EII	363.8 ± 457.4	361.4 ± 359.7	656.1 ± 419.9	**0.021**
PLR	101.7 ± 62.5	113.2 ± 69.2	103.9 ± 62.7	0.529
PNI	54.0 ± 6.5	53.8 ± 9.9	48.3 ± 7.6	**0.010**

Abbreviations: **AISI**, aggregate index of systemic inflammation; **CAR**, C-reactive protein-to-albumin ratio; **EII**, platelet-to-lymphocyte ratio × C-reactive protein; **MI-1**, neutrophil-to-lymphocyte ratio × C-reactive protein; **MLR**, monocyte-to-lymphocyte ratio; **NLR**, neutrophil-to-lymphocyte ratio; **PLR**, platelet-to-lymphocyte ratio; **PNI**, prognostic nutritional index; **SII**, systemic immune-inflammation index; **SIRI**, systemic inflammation response index.

Table 4. Univariate and multivariate logistic regression analysis showing the independent predictors of positive late gadolinium enhancement (LGE) in hypertrophic cardiomyopathy

	Univariate		Multivariate	
	Odds ratio 95% CI	*p*-value	Odds ratio 95% CI	*p*-value
High native T1	2.074(1.134-3.794)	**0.018**	1.181(0.575-2.425)	0.650
Hs-Troponin T	1.026(1.003-1.049)	**0.027**	1.006(0.993-1.018)	0.380
NT-Pro BNP	1.001(1.000-1.001)	0.017		
Sodium	0.897(0.799-1.008)	0.067		
Scar Score	2.243(1.686-2.983)	**<0.001**	1.798(1.285-2.518)	**0.001**
ESR	1.177(1.090-1.271)	**<0.001**	1.115(1.032-1.206)	**0.006**
IVS	4.373(1.811-10.558)	**0.001**	4.179(1.539-11.347)	**0.005**

Abbreviations: **High native T1**, elevated native T1 mapping values on cardiac magnetic resonance; **hs-Troponin T**, high-sensitivity troponin T; **IVS**, interventricular septal thickness; **NT-proBNP**, N-terminal pro–B-type natriuretic peptide; **Scar Score**, composite score based on hs-Troponin T ≥14 ng/L, NT-proBNP ≥125 pg/mL, CAR >0.05, and PNI ≤52; **Sedim**, erythrocyte sedimentation rate; **Sodium**, serum sodium concentration.

PP-057 [Coronary Artery Disease / Acute Coronary Syndrome]

Prevalence of valvular and ascending aortic calcification in coronary CT angiography: association with left main coronary plaque

Sefa Okar[1], Ziya Gökalp Bilgel[1], Caner İncekaş[2]

[1]Başkent University, Faculty of Medicine, Adana Research and Application Center, Adana
[2]Department of Biostatistics, Başkent University, Faculty of Medicine, Ankara

Background and Aim: Ascending aortic calcification is a recognized risk factor for coronary artery disease (CAD). This study aimed to investigate the prevalence of ascending aortic calcification and its association with left main coronary artery (LMCA) lesions in patients evaluated with coronary computed tomography angiography (CCTA).

Methods: his study included 1128 individuals with a mean age of 53.95 ± 11.42 years, comprising 37.23% females (n=420) and 62.77% males (n=708). Statistical analyses were performed using SPSS version 25.0. Normality of variable distribution was assessed with the Shapiro-Wilk test. Categorical variables were summarized as numbers and percentages, while quantitative variables were presented as mean ± standard deviation. Differences between categorical groups were analyzed using the chi-square test or Fisher-Freeman-Halton Exact Test, with a significance level of α=0.05.

Results: Ascending aortic calcification was detected in 9.1% of cases (n=101). A significant association was found between LMCA lesion status and ascending aortic calcification (p<0.001). Normal LMCA was more prevalent in individuals without aortic calcification (88.37%, n=889) compared to those with calcification (58.42%, n=59). LMCA plaque was significantly more frequent in the calcification group (31.68%, n=32) than in the non-calcification group (8.05%, n=81) (p<0.001). Stenosis below 50% was also higher in the calcification group (8.91%, n=9) compared to the non-calcification group (3.28%, n=33) (p=0.005). Stenosis above 50% was rare in both groups (0.30% without calcification vs. 0.99% with calcification), with no significant difference (p=0.214).

Conclusions: Ascending aortic calcification on CCTA is strongly associated with LMCA plaque and stenosis, indicating its potential as a significant marker for LMCA disease. Patients with this finding warrant comprehensive evaluation, including additional imaging or functional testing, to assess potential LMCA involvement.

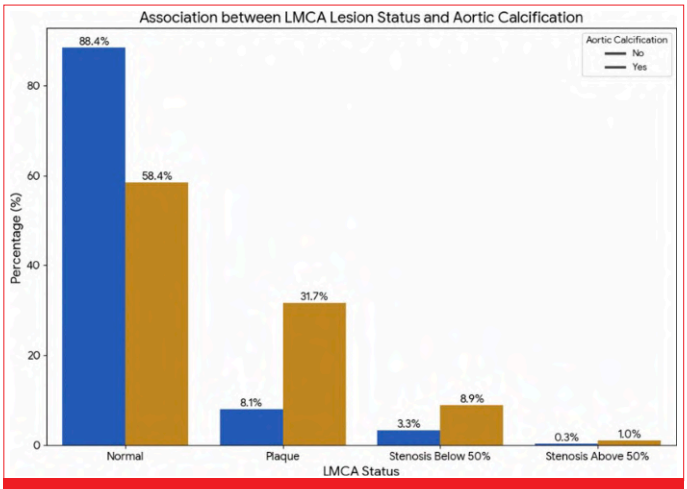

Figure 1. The graph compares the distribution of Left Main Coronary Artery (LMCA) lesion status in groups with and without aortic calcification. The graph shows that the rate of a normal LMCA is significantly higher (88.4%) in individuals without aortic calcification. Conversely, the rates of plaque (31.7%) and stenosis below 50% (8.9%) in the LMCA are statistically significantly higher in the group with aortic calcification. These findings support that aortic calcification in a coronary CT angiography evaluation may be an important marker for the presence of atherosclerotic lesions in the LMCA. LMCA: Left Main Coronary Artery Aortic Calcification: Aortic Calcification Plaque: Plaque Stenosis Below 50%: Stenosis Below 50% Stenosis Above 50%: Stenosis Above 50%.

Table 1. Distribution of left main coronary artery (LMCA) lesion status by presence of aortic calcification

	No aortic calcification (n=1008)	Yes aortic calcification (n=101)	p
LMCA normal	88.37%	58.42%	<0.001
LMCA plaque	8.05%	31.68%	<0.001
LMCA <50% stenosis	3.28%	8.91%	0.005
LMCA >50% stenosis	0.30%	0.99%	–

LMCA: Left main coronary artery, n: Number of patients, p-value: Statistical significance level.

PP-058 [Interventional Cardiology / Valvular and Structural Heart Disease]

Balon expandable TAVR kapaklarda balonların genişleyebilme kapasitelerinin *in vitro* ölçümü

İrem Dilara Can[1], Ezgi Merve Çelik[2], Selin Yöndem[2], Esra Sadıkoğlu[2], Mehmet Akif Erdöl[2], Çağrı Yayla[2], Ahmet Göktuğ Ertem[2], Adnan Burak Akçay[2]

[1]Department of Cardiology, Devrek State Hospital, Zonguldak
[2]Department of Cardiology, Ankara Bilkent City Hospital, Ankara

Background and Aim: In patients undergoing transcatheter aortic valve replacement, the appropriate valve size is selected by measuring the valve area using pre-procedure imaging techniques. A review of the valve user manuals indicates the valve area ranges that can be applied for each fixed valve size and the nominal amount of contrast volume to be used based on the selected valve size. However, the results of inflating the valve balloons to different volumes in the intermediate valve areas are unknown. Our goal was to determine whether balloon volume/diameter changes when the balloons are inflated with a contrast medium volume different from the nominal values.

Methods: After valve implantation, the balloons of the Myvalv balloon-expanded TAVR valve system were inflated separately in vitro with the same contrast medium used during the procedure, to the nominal volume specified by the manufacturer for each valve size, and to volumes 2, 4, and 6 cc above this volume. The diameters were measured using a ruler. If a volume of contrast medium other than the nominal was used during the procedure, a separate in vitro measurement was taken with the same volume of contrast agent. Fluoroscopic recordings taken during the procedure were reviewed, and the proximal, middle, and distal portions of the implanted bioprosthetic valve were measured from the inside out using three valve and valve overlap images, calibrated using the 6F pigtail catheter used.

Results: A total of 22 patients were included in the study. 68.2% of the patients were male, the mean age was 78.8 years, and the mean EF was 51.9%. A total of 22 Myval valves in six different sizes (23, 24.5, 26, 27.5, 29, and 32%) were examined. Fluoroscopically measured proximal, middle, and distal diameters were compared with balloon diameters obtained with different inflation volumes in vitro. In addition to the nominal volume, balloons were inflated to volumes increased by +2 cc, +4 cc, and +6 cc. If a volume other than the nominal volume was used during the procedure, this volume was repeated in vitro. For all valve sizes, increasing the inflation volume resulted in an increase in balloon diameter. Even small volume increases produced measurable changes in diameter. Pre-dilation and post-dilation procedures were performed as necessary.

Conclusions: This study demonstrated measurable increases in balloon diameters when contrast volumes exceeding the nominal inflation volume were used in the balloon-expandable MyvalTAVR valve system. These findings suggest that balloons may have additional expansion capacity beyond the limits specified in the manufacturer's guidelines. This may contribute to the reevaluation of valve selection and implantation strategies, particularly in patients with borderline annulus sizes. However, the in vitro study and limited sample size suggest that further, more comprehensive clinical studies are needed to generalize the results to clinical outcomes.

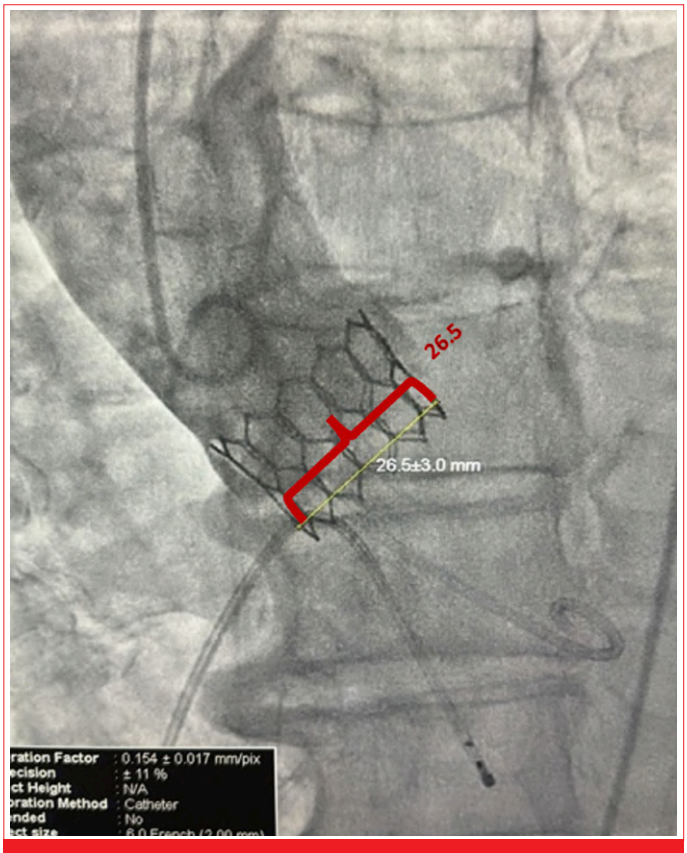

Figure 1. Measurement of the dimensions of the proximal end of the TAVR valve on fluoroscopic images.

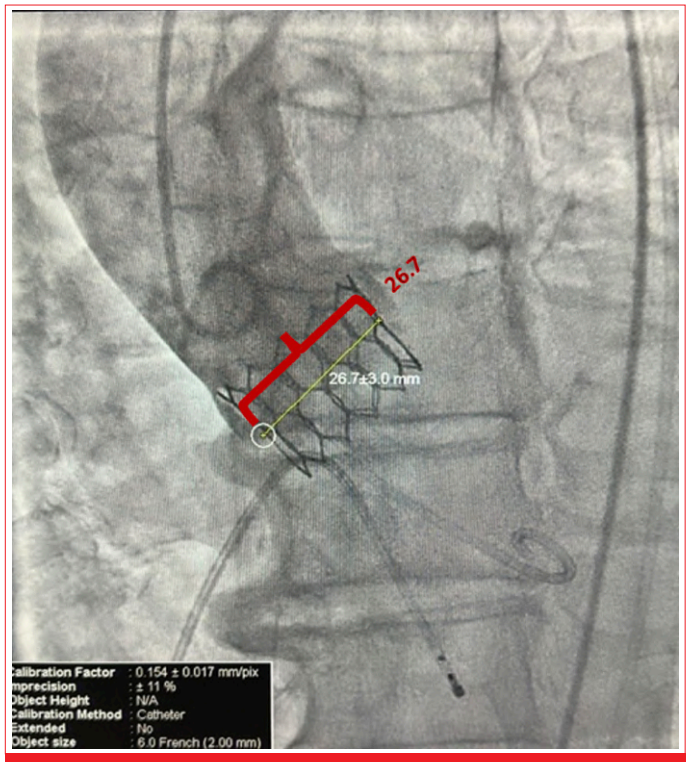

Figure 2. Measurement of the dimensions of the mid-region of the TAVR valve in fluoroscopic images.

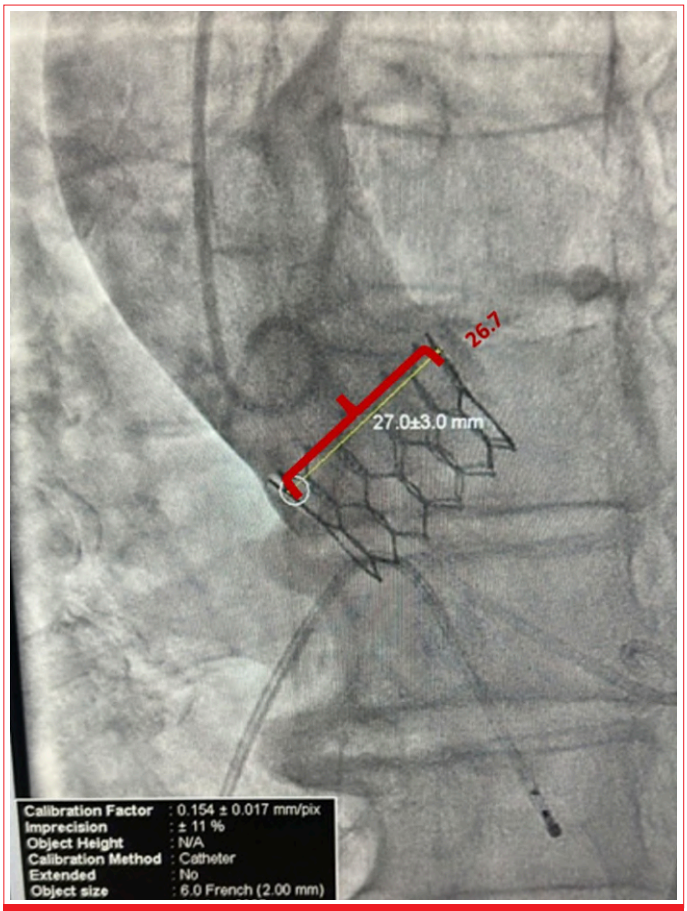

Figure 3. Measurement of the dimensions of the distal end of the TAVR valve on fluoroscopic images.

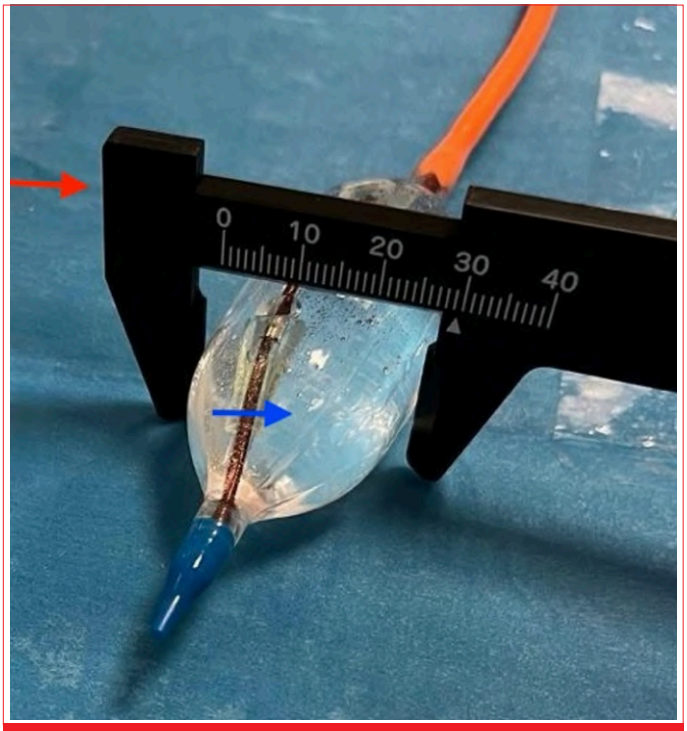

Figure 4. Red arrow: The ruler we used to measure. Blue arrow: The balloon of the TAVR valve, which can be expanded with the balloon used during the procedure.

Table 1. 23 myvalv valve measurements

ID	Flora İle Kapak Ölçümleri Prox (mm)	Flora İle Kapak Ölçümleri Dist (mm)	Flora İle Kapak Ölçümleri Mid (mm)	Nominal Volüm -Balon Ölçüsü	İşlemde Şişlen Volum	16 Cc Ölçüm (mm)	17cc Ölçüm (mm)	18cc Ölçüm (mm	19cc Ölçüm (mm)	20cc Ölçüm (mm)	22cc Ölçüm (mm)	Predilatasyon	Postdilatasyon
1	23.4	22.5	23	18cc	16cc	20.5	-	23	23.5	24.5	25.5	-	-
2	24.6	22.4	25.7	18cc	16cc	20.5	-	22	23	24	25	-	-
3	22.4	21.3	20.5	18cc	18cc	20	22	22.5	23.5	24	25.5	-	-
4	23.4	22	21.5	18c	18cc	20	-	22.5	-	23	24.5	-	-
5	23.4	22	21.8	18cc	18cc	20	-	23	-	23.5	24.	-	-

Table 2. 24.4 myvalv cover measurements

Id	Flora İle Kapak Ölçümleri Prox (mm)	Flora İle Kapak Ölçümleri Dist(mm)	Flora İle Kapak Ölçümleri Mid(mm)	Nominal Volüm-Balon Ölçüsü	İşlemde Şişlen Volum	18cc Ölçüm (mm)	20cc Ölçüm (mm)	21ccÖlçüm (mm)	22cc Ölçüm (mm)	24cc Ölçüm (mm)	Predilatasyon	Postdilatasyon
1	24.6	23.5	24.4	20cc	20cc	22	24		24.5	25.5	+	-
2	26.1	25.3	24.6	20cc	22cc	22.5	24.5	25	25.5	26.5	-	-
3	25.4	25.1	19.1	20cc	24cc	22	24.5		25	25.5	-	-
4	26.7	25.3	23.9	20cc	20cc	23	24		26	26.5	-	-
5	24.4	23.4	23.9	20cc	20cc	23	24		25	26	-	-

Table 3. 26 myvalv cover measurements

ID	Flora İle Kapak Ölçümleri Prox	Flora İle Kapak Ölçümleri Dist (Mm)	Flora İle Kapak Ölçümleri Mid(Mm)	Nominal Volüm	İşlemde Şişlen Volum	22cc Ölçüm (Mm)	23cc Ölçüm (Mm)	25cc Ölçüm (Mm)	27cc Ölçüm (Mm)	28cc Ölçüm (Mm)	29cc Ölçüm (Mm)	Predilatasyon	Postdilatasyon
1	27.2	26.1	24.8	23cc	23cc	24.5	26	27	27.5	28.5	29.5	-	-
2	27.1	25.9	26.2	23cc	23cc	24.4	26	27	27.5	28	29	-	-
3	26.7	25.1	25.4	23cc	23cc	25	26	26.5	27.5	28	29	-	-
4	29,6	28,4	27,6	23	23	-	26	27	28	29	-	-	-

Table 4. 27.5 myvalv cover measurements

Id	Flora İle Kapak Ölçümleri Prox (Mm)	Flora İle Kapak Ölçümleri Dist(Mm)	Flora İle Kapak Ölçümleri Mid(Mm)	Nominal Volüm	İşlemde Şişlen Volum	26cc Ölçüm (Mm)	28cc Ölçüm (Mm)	30cc Ölçüm (Mm)	32cc Ölçüm (Mm)	34cc Ölçüm (Mm)	Predilatasyon	Postdilatasyon
1	28.5	28.7	27.8	28cc	28cc	25	27.5	28	29	29.5	-	+
2	28	26.2	28.1	28cc	28cc	27	28	28.5	29	-	+	-
3	25.1	26.1	26	28cc	28cc	-	27	28	28.5	29.5	-	-

Table 5. 29 myvalv valve measurements

Id	Flora İle Kapak Ölçümleri Prox (Mm)	Flora İle Kapak Ölçümleri Dist (Mm)	Flora İle Kapak Ölçümleri Mid (Mm)	Nominal Volüm	İşlemde Şişlen Volum	30cc Ölçüm (Mm)	32cc Ölçüm (Mm)	34cc Ölçüm (Mm)	36cc Ölçüm (Mm)	38cc Ölçüm (Mm)	Predilatasyon	Postdilatasyon
1	30.5	30.7	29.6	32cc	32cc	27.8	28	28.9	29.1	-	-	-
2	28.8	29.7	29	32cc	32cc	27	29	30	30.5	30.7	-	-
3	28.3	26.9	26.7	32cc	36cc	28	29	30	30.5	31	+	-
4	29.8	29.8	29.9	32cc	36cc	27.5	28.9	29.5	30	21	-	-

Table 6. 32 myvalv valve measurements

Id	Flora İle Kapak Ölçümleri Prox (Mm)	Flora İle Kapak Ölçümleri Dist(Mm)	Flora İle Kapak Ölçümleri Mid(Mm)	Nominal Volüm	İşlemde Şişlen Volum	38cc Ölçüm (Mm)	40cc Ölçüm (Mm)	42cc Ölçüm (Mm)	Ölçüm (Mm)	46cc Ölçüm (Mm)	Predilatasyon	Postdilatasyon
1	32	31.9	30.4	40cc	40cc	31	33	33.5	34	35	-	-

PP-059 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Left stellate ganglion block for prevention of recurrent ICD shocks in LVAD patients: Our clinical experience

Yaprak Tepeli[1], Alper Candemir[1], Ahmet Can Urgancı[1], Elif İrem Yetkin[1], Mustafa Uyar[1], Sanem Nalbantgil[1], Burcu Özalp Horsanalı[2], Can Eyigör[2], Evrim Şimşek[1]

[1]*Department of Cardiology, Ege University, Faculty of Medicine, İzmir*
[2]*Department of Anesthesiology and Reanimation, Ege University, Faculty of Medicine, İzmir*

Background and Aim: Electrical storm and recurrent implantable cardioverter-defibrillator (ICD) shocks are critical clinical challenges in patients with heart failure. In patients with left ventricular assist devices (LVADs), this clinical scenario becomes even more complex. Herein, we present the outcomes of left stellate ganglion block (LSGB) performed in five LVAD patients with VT storm or recurrent ICD shocks.

Methods: Five patients with left ventricular assist devices (LVADs) who experienced refractory ventricular tachycardia (VT) and/or ventricular fibrillation(VF) episodes unresponsive to antiarrhythmic therapy underwent left stellate ganglion block (LSGB) using pulsed radiofrequency ablation under fluoroscopic guidance at different time points. The aim of this intervention was to control refractory arrhythmias through modulation of the sympathetic nervous system. After establishing monitoring in the supine position, procedural sedation and analgesia were administered. Under fluoroscopic guidance, the left stellate ganglion was localized at the level of the C7 vertebra (Figure 1). Following confirmation of needle placement and drug spread via injection of a radiopaque contrast agent, 5 mL of bupivacaine was administered. After performing motor and sensory stimulation testing, pulsed radiofrequency treatment was applied at 42°C for a duration of 6 minutes (Figure 2, 3). Following LSGB, patients were closely monitored in the intensive care unit for hemodynamic status and arrhythmia recurrence.

Results: Five LVAD patients had LSGB (The characteristics of the treated patients are presented in Table 1). Ptosis was not observed after the first day in any patient, and no procedure-related complications occurred. Median follow up time was 4.2 months (3 month- 24 months) VT recurred in one patient on the first day and in another on the fourth day post-procedure. In the remaining three patients, no VT episodes were observed during hospitalization.

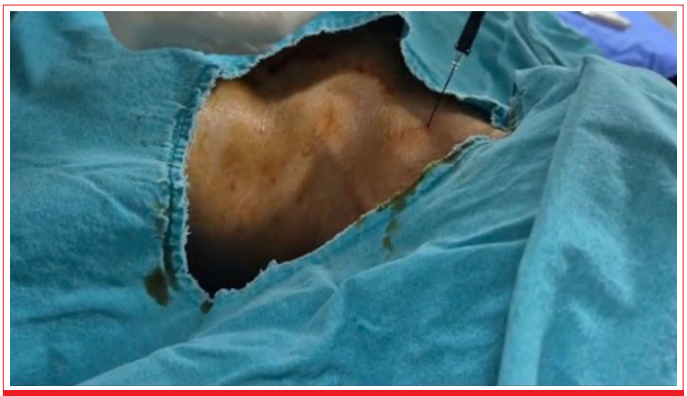

Figure 1.

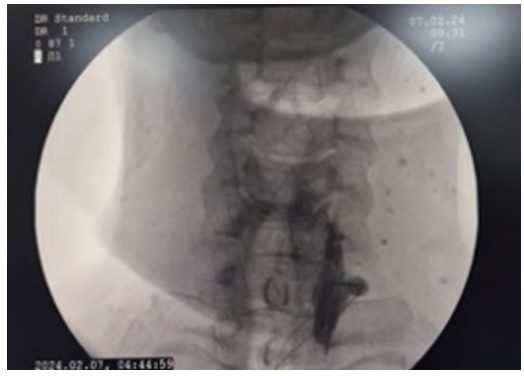

Figure 2.

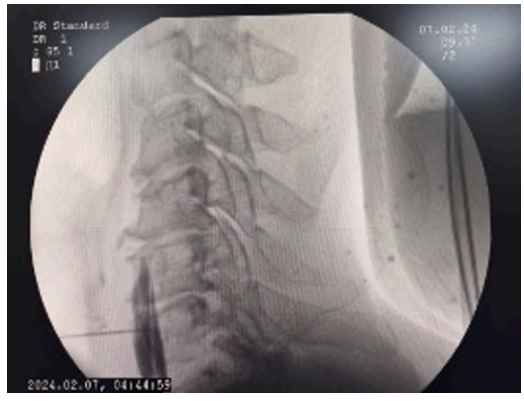

Figure 3.

Table 1.

Case	Sex	Age	Etiology	LVAD year	Device	VT/VF type	AF	Beta-blocker	Antiarrhytmic therapy	LSGB year	VT recurence
1	M	50	NICMP	2016	ICD	M-VT	No	Carvedilol	Amiodarone, Mexiletine	2021	Day 1
2	F	66	NICMP	2020	ICD	M-VT	No	Carvedilol	Mexiletine	2021	Month 5
3	M	52	ICMP	2016	ICD	VF	Yes	Metoprolol	Sotalol, Amiodarone	2022	Day 4
4	M	45	NICMP	2018	CRT	M-VT	No	None	Mexiletine, Amiodarone	2023	None during FU
5	M	28	NICMP	2021	ICD	M-VT	No	Propranolol	Amiodarone, Lidocaine	2024	Day 4

In long-term follow-up, VT recurred in one patient on day 14 and in another at 5 months, while one patient remained free of VT throughout the follow-up period.

Conclusions: Although the degree of effectiveness varied among study patients, unilateral left stellate ganglion block (LSGB) cshould be considered a minimally invasive therapeutic option for reducing or preventing VT storms and recurrent shocks in the complex population of LVAD patients.

PP-060 [Heart Failure]

Relationship between hemodynamic parameters obtained by echocardiography and quality of life, morbidity, and mortality in patients with heart failure with preserved ejection fraction

Esra Duman Acar, Mehmet Arslan, Ahmet Barutçu

Department of Cardiology, Çanakkale Onsekiz Mart University, Çanakkale

Background and Aim: This study aimed to evaluate the relationship between echocardiographically derived hemodynamic and deformation parameters and quality of life, biomarkers, and clinical outcomes in patients with heart failure with preserved ejection fraction (HFpEF).

Methods: Fifty patients diagnosed with HFpEF according to the HFA-PEFF diagnostic algorithm who met the inclusion criteria were followed for 12 months. Quality of life was assessed using the Turkish-validated KCCQ-12 questionnaire, and NT-proBNP levels were measured at regular intervals. Echocardiographic assessments included conventional parameters such as EF, GLS, LAVI, and E/e', as well as twist and untwist parameters evaluated by speckle tracking echocardiography. Clinical outcomes included all-cause mortality and major adverse cardiovascular events (MACE).

Results: Baseline NT-proBNP levels showed a negative correlation with apical rotation (r=-0.316, p=0.025) and twist (r=-0.489, p<0.001), and a positive correlation with basal rotation (r=0.465, p=0.001) and LAVI (r=0.308, p=0.031). No significant relationship was observed between NT-proBNP and EF (r=0.029, p=0.841) or GLS (r=0.223, p=0.120). The E/e' ratio showed a weak positive correlation with peak untwist time (r=0.290, p=0.041) and moderate positive correlations with untwist rate (r=0.389, p=0.005) and corrected untwist time (r=0.331, p=0.019). NT-proBNP levels and changes in KCCQ scores were not VIII significantly associated with mortality or MACE. Similarly, no significant associations were found between KCCQ-12 scores and echocardiographic parameters.

Conclusions: This study demonstrated significant positive correlations between the E/e' ratio, reflecting diastolic filling pressure, and untwist parameters representing left ventricular relaxation mechanics. These findings suggest that untwist mechanics may be a potential marker for the noninvasive evaluation of diastolic dysfunction. Additionally, NT-proBNP appeared to be more closely related to regional mechanical parameters than to conventional systolic function measures. The results underscore the importance of a comprehensive approach that includes mechanical analysis in the diagnosis and follow-up of HFpEF patients.

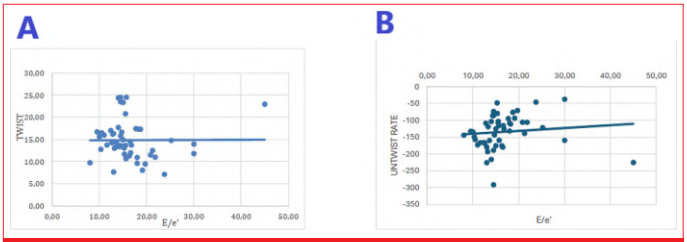

Figure 1. In the correlation analysis between basic transthoracic echocardiography parameters and STE parameters, no correlation was found between E/e' and twist (r=-0.231, p=0.107) (A), whereas a moderate positive correlation was found between E/e' and untwist rate (r=0.389, p=0.005) (B).

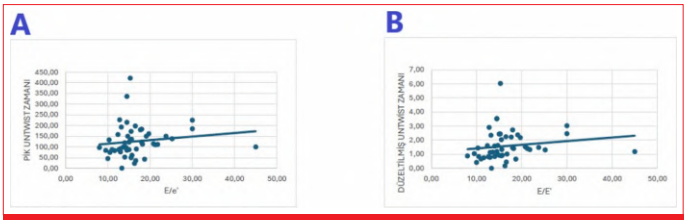

Figure 2. In the correlation analysis between basic transthoracic echocardiography parameters and STE parameters, a weak positive correlation was found between E/e' and peak untwist time (r=0.290, p=0.041) (A), and a moderate positive correlation was found between E/e' and corrected untwist time (r=0.331, p=0.019) (B).

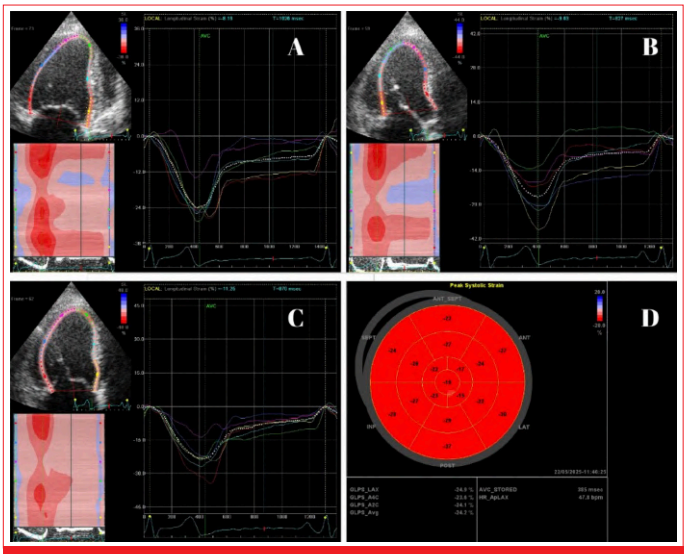

Figure 3. STE-GLS. Apical 4-chamber (A), apical long-axis (B), and apical 2-chamber (C) longitudinal strain analyses of the left ventricle measured by STE: Strain allows for regional assessment of LV wall motion. In D, the 17-segment "bull's eye" map of the LV obtained from the three apical views is shown.

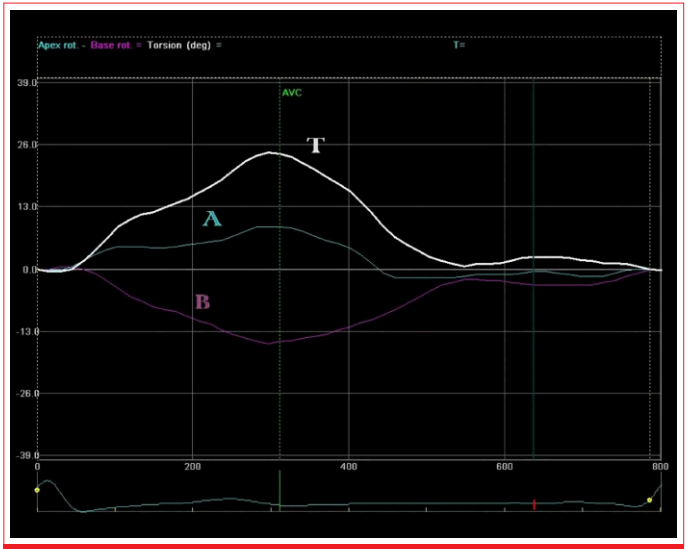

Figure 4. STE-twist. Left ventricular twisting parameters measured by STE (A: apical rotation, B: basal rotation, T: twist [the net difference between apical rotation and basal rotation]. The units of A, B, and T are degrees).

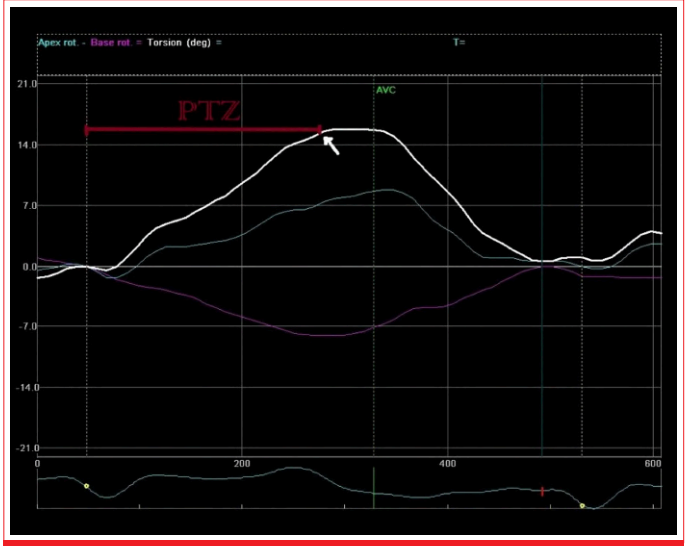

Figure 5. STE-peak twist time. Left ventricular rotation parameters: Time to peak rotation; peak twist time (red line), net difference between apical and basal rotation at the moment of peak rotation; twist (white arrow).

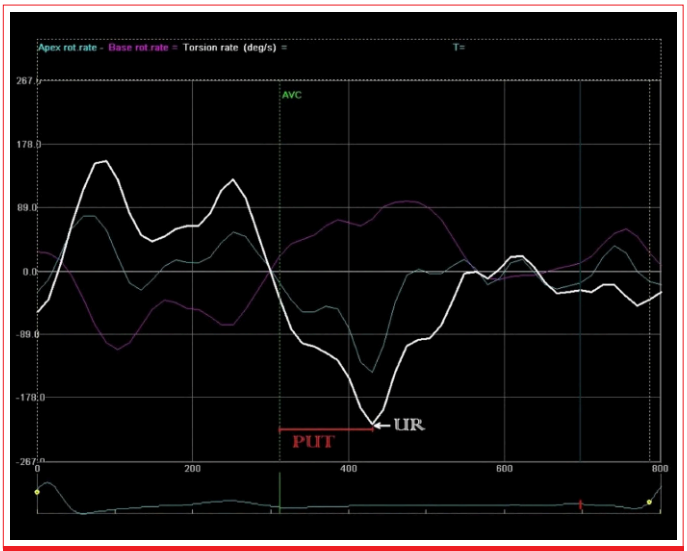

Figure 6. STE-peak untwist time. LV untwisting parameters: Untwist rate (white arrow), time from aortic valve closure to peak untwisting; peak untwist time (red line).

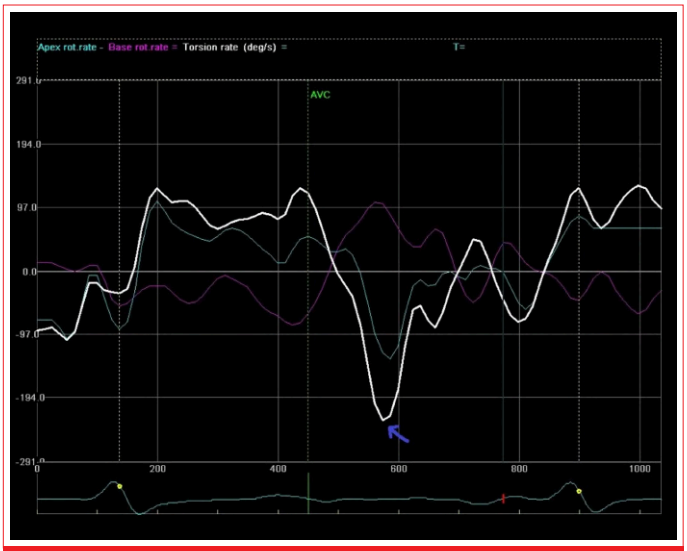

Figure 7. STE-untwist. Left ventricular untwisting parameters measured by STE (untwist rate, indicated by the blue arrow; unit: degrees/s).

Table 1. The baseline NT-proBNP value showed a negative correlation with apical rotation (r=–0.316, p=0.025) and twist (r=–0.489, p<0.001), and a positive correlation with basal rotation (r=0.465, p=0.001) and LAVI (r=0.308, p=0.031), while no statistically significant relationship was found with GLS (r=0.223, p=0.120) or EF (r=0.029, p=0.841). The association between NT-proBNP and rotational parameters persisted for twist and apical rotation at 1 month, whereas it persisted for basal rotation up to 3 months

		BNP -0	BNP-1	BNP-3	BNP-6	BNP-12
APİKAL ROT.	r	**-0,316***	**-0,286***	-0,241	-0,184	-0,062
	p	**0,025**	**0,044**	0,107	0,233	0,691
BAZAL ROT.	r	**0,465****	**0,346***	**0,303***	0,220	0,228
	p	**0,001**	**0,014**	**0,041**	0,151	0,142
TWIST	r	**-0,489****	**-0,394****	-0,274	-0,238	-0,224
	p	**<0,001**	**0,005**	0,065	0,119	0,150
GLS	r	0,223	0,254	0,228	0,288	0,197
	p	0,120	0,076	0,127	0,058	0,206
LVEF	r	0,029	-0,065	-0,188	-0,193	-0,183
	p	0,841	0,652	0,210	0,209	0,240
E/e'	r	0,183	0,275	0,220	0,194	0,234
	p	0,205	0,053	0,142	0,207	0,130
LAVİ	r	**0,308***	0,142	0,086	0,034	0,234
	p	**0,031**	0,331	0,570	0,825	0,126

PP-061 [Cardiovascular Nursing / Technician]

Polypharmacy and frailty in elderly cardiac patients

Melike Çelik[1], Sıdıka Oğuz[1], Zühre Zelal Koyunsever[2], Sedef Tiraki[3]

[1]*Marmara University, Faculty of Health Sciences, İstanbul*
[2]*Aydın State Hospital, Aydın*
[3]*Department of Cardiology, İstanbul Sancaktepe Martyr Prof. Dr. İlhan Varank Training and Research Hospital, İstanbul*

Background and Aim: As the elderly population grows, cardiovascular diseases, along with chronic illnesses, are also on the rise. As a result, older individuals are using multiple medications, resulting in polypharmacy. Polypharmacy is considered a risk factor for frailty. This study was conducted to determine the level of polypharmacy and frailty in elderly individuals with cardiovascular disease and to examine the relationship between polypharmacy and frailty level.

Methods: Elderly individuals aged ≥65 years applying to the cardiology outpatient clinic of a training and research hospital in Istanbul and meeting the inclusion criteria were studied. According to the power analysis, at least 90 patients were targeted (Power: 0.80; β: 0.10; α: 0.05; Effect size: 0.33). Face-to-face interviews, each lasting 15 minutes on average, were conducted with 102 patients. Data were collected using the "Patient Information Form" and the "Edmonton Frailty Scale" and analyzed and reported at p<0.05 significance and 95% confidence levels.

Results: Of the participants, 51% were male, 87.3% were married, 52% were primary/secondary school graduates, and their mean age was 70.9 ± 5.5 years. The mean number of medications they used was 5.7 ± 1.5, and the mean number of chronic conditions was 2.6 ± 1.1. Based on the Edmonton Frailty Scale mean score (6.94 ± 3.37), participants were generally near the moderate frailty level. Females (p<0.0001), singles (p=0.008), those who had never attended school (p<0.0001), and with income less than expenses (p=0.046) had higher mean frailty scores. Frailty increased with age, number of medications used, and number of chronic conditions (p<0.05).

Conclusions: Frailty increases with the number of medications elderly individuals use. Elderly cardiac patients with polypharmacy may be frail, highlighting the importance of early frailty detection and geriatric assessment in comprehensive nursing care.

PP-062 [Cardiovascular Nursing / Technician]

The role of the nurse in cardiac rehabilitation processes

Huriye Arslaner, Dilek Aydın

Department of Cardiology, Health Science University, Kartal Koşuyolu Training and Research Hospital, İstanbul

Background and Aim: Cardiac rehabilitation is a social, physical, and psychological intervention that positively affects underlying risk factors to halt, slow down, or reverse the progression of heart disease, thereby enabling the patient to actively participate in society. Cardiac rehabilitation has positive effects in terms of mortality rates, repeated hospital admissions, quality of life, and increased functional capacity of the individual. A multidisciplinary team effort is required to achieve the desired outcomes in cardiac rehabilitation programs. Nurses play a key role in modifying cardiac risk factors and patient education and are an indispensable member of the team. This paper aims to convey the roles and importance of nurses as members of the team in cardiac rehabilitation processes.

Methods: This paper was compiled based on literature information covering the roles of nurses in cardiac rehabilitation processes.

Results: The importance of providing cardiac rehabilitation as a multidisciplinary team is emphasized in international guidelines. This team consists of members such as a cardiologist, physical therapist, psychologist, dietitian, nurse, occupational therapist, and social worker. Multidisciplinary work requires joint decision-making, sufficient knowledge and communication skills, awareness of team members' responsibilities, a sense of teamwork, and ensuring that the patient and their family play an active role within the team.

Figure 1. Implementation of the exercise.

The nurse's roles include ensuring and maintaining physical well-being, facilitating behavioral change and motivation, preventing complications, adapting to lifestyle changes, increasing the patient's problem-solving skills, and reducing uncertainties related to the disease. Together with team members, the nurse works to maximize the individual's independent functioning. By setting achievable goals with the patient and family, the nurse guides the patient in reaching these goals. The nurse ensures the maintenance of the patient's well-being after discharge and provides the necessary education.

Conclusions: Team work is crucial in cardiac rehabilitation programs, and nurses are an indispensable part of this team. There is a need for more research on nursing practices in cardiac rehabilitation processes.