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OP-001 [Interventional Cardiology / Coronary]

Underlying mechanism of second-generation drug eluting stent thrombosis; an optical coherence tomography study

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Background and Aim: Optimal percutaneous coronary intervention (PCI) for the treatment of stent thrombosis (ST) depends on the mechanism of the ST. Since the conventional invasive angiography has limited value for the identification of underlying aetiology, intravascular imaging is particularly important. Aims to demonstrate the optical coherence tomography (OCT) findings in patients presenting with early, late and very late drug-eluting ST.

Methods: Angiography and OCT images of patients with ST were reviewed. OCT findings including malapposition, neoatherosclerosis, neointimal hyperplasia, stent expansion index, minimal lumen area and stent edge disease were assessed.

Results: There were 6 patients with early ST and 68 patients with late stent thrombosis (28 late ST and 40 very late ST). Among patients with early ST, there were 4 cases with stent edge dissection, 1 case with significant tissue/thrombus prolapse and there was no plausible cause of ST in 1 patient. There was no significant difference between patients with late and very late ST in terms of clinical features. The mechanism of ST in overall patient group was neoatherosclerotic plaque rupture (42.6%), neointimal erosion (20.5%), stent-edge disease (22.0%) and malapposition/uncovered strut (10.2%). No plausible pathology of ST (4.4%) was detected in 3 patients. The most common OCT findings were neoatherosclerosis (70.4%), stent-edge disease (61.4%) underexpansion (55%) and malapposition/uncovered strut (48%).

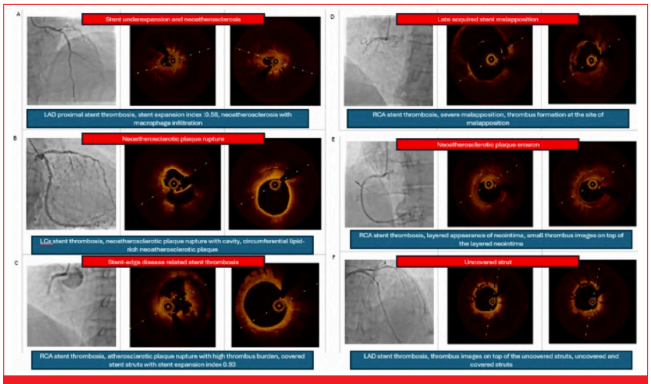

Figure 1. Mechanism.

Conclusions: OCT is a feasible and safe procedure which clearly identifies the underlying aetiology in majority of patients with stent thrombosis. Furthermore, OCT is of clinical importance regarding individualizing and optimizing the PCI procedure.

OP-002 [Interventional Cardiology / Coronary]

The relationship between triglyceride-glucose index and 1-year mortality in patients with ST-segment elevation myocardial infarction

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Background and Aim: Insulin resistance (IR) is a key risk factor for cardiometabolic diseases, and the triglyceride-glucose (TyG) index has emerged as a reliable surrogate marker. Compared to triglyceride or glucose levels alone, the TyG index offers superior predictive accuracy for cardiovascular events. Early identification of high-risk ST-segment elevation myocardial infarction (STEMI) patients is essential for improving outcomes. Although the TyG index has been studied in various cardiovascular settings, its prognostic value in STEMI remains inadequately explored. This study aims to assess the TyG index as a predictor of one-year mortality in STEMI patients undergoing percutaneous coronary intervention (PCI).

Methods: This retrospective single-center study included 573 consecutive STEMI patients admitted between January 1, 2019, and January 1, 2020. Patients were divided into low (≤9.17, n=284) and high TyG (>9.17, n=289) groups based on Youden's index. Clinical variables were compared, and one-year mortality predictors were analyzed using logistic regression.

Results: Demographic characteristics were similar between groups (age, p=0.453; gender, p=0.541). However, significant differences were noted in clinical and laboratory parameters. The high TyG group showed a higher prevalence of cardiogenic shock (p=0.044), pulmonary edema (p=0.040), diabetes mellitus (p<0.001), and dyslipidemia (p=0.010). Laboratory findings revealed significantly elevated fasting glucose, total cholesterol, and triglyceride levels (all p<0.001), along with higher creatinine (p=0.004) and LDL (p=0.018), and lower HDL levels (p=0.005). Mortality rates were also significantly higher in the high TyG group, including in-hospital, one-year, and five-year mortality (all p<0.001). These results suggest that an elevated TyG index may be a significant marker of both short- and long-term mortality risk. Cox regression analysis showed that the TyG index was independently associated with one-year mortality. It remained a significant predictor in both univariate (HR: 2.56, 95% CI: 1.66–3.86, p<0.001) and multivariate analyses (HR: 2.67, 95% CI: 1.70–4.18, p<0.001). In the multivariate model, DM (HR: 2.50, 95% CI: 1.19–5.25, p=0.015), hypertension (HR: 3.20, 95% CI: 1.63–6.29, p=0.001), prior myocardial infarction (HR: 3.21, 95% CI: 1.93–5.33, p<0.001), and chronic kidney disease (HR: 1.97, 95% CI: 1.21–3.21, p=0.006) were also independent predictors. Other variables were not statistically significant.

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

Variables	All Patients (n=573)	Low TyG ≤9.17 (n=284)	High TyG >9.17 (n=289)	p value
Gender (female), n (%)	162 (28.3)	77 (27.1)	85 (29.4)	0.541
Age, (years)	61.48 ± 14.02	61.92 ± 14.42	61.04 ± 13.62	0.453
Smoking, n (%)	394 (68.8)	193 (68.0)	201 (69.6)	0.681
LVEF (%)	44.20 ± 10.29	44.67 ± 10.61	43.72 ± 9.95	0.279
HT, n (%)	335 (58.5)	168 (59.2)	167 (57.8)	0.739
DM, n (%)	190 (33.2)	74 (26.1)	116 (40.1)	<0.001
Dyslipidemia, n (%)	257 (44.9)	112 (39.4)	145 (50.2)	0.010
CKD, n (%)	34 (5.9)	12 (35.3)	22 (7.6)	0.086
Previous MI, n (%)	112 (19.5)	57 (20.1)	55 (19.0)	0.754
Previous PCI, n (%)	92 (16.1)	41 (14.4)	51 (17.6)	0.295
Previous CABG, n (%)	29 (5.1)	13 (4.6)	16 (5.5)	0.601
Recurrent MI, n (%)	47 (8.2)	21 (7.4)	26 (9.0)	0.485
Cardiogenic shock, n (%)	36 (6.3)	12 (4.2)	24 (8.3)	0.044
Pulmonary oedema, n (%)	41 (7.2)	14 (4.9)	27 (9.3)	0.040
Chronic heart failure, n (%)	88 (15.4)	50 (17.6)	38 (13.1)	0.139
Killip > 2	108 (18.8)	59 (20.8)	49 (17.0)	0.242
VT, VF	49 (8.6)	23 (8.1)	26 (9.0)	0.701
WBC (x10^3/uL)	12.67 ± 2.62	12.49 ± 2.90	12.86 ± 2.31	0.090
Lymphocyte (x10^3/uL), median (Q1-Q3)	1.75 (1.21-2.55)	1.76 (1.18-2.48)	1.75 (1.22-2.65)	0.635
Neutrophil (x10^3/uL)	8.62 ± 2.75	8.48 ± 2.82	8.75 ± 2.68	0.230
Hemoglobin (gr/L)	13.47 ± 1.82	13.43±1.84	13.50±1.82	0.665
Platelet (10^3/uL)	259.80 ± 68.27	258.00 ± 69.71	261.57 ± 66.90	0.531
Glucose, (mg/dL)	165.10 ± 80.85	132.80 ± 41.70	196.83 ± 96.10	<0.001
Creatinine (mg/dL), median (Q1-Q3)	0.83 (0.72-1.03)	0.81 (0.71-0.96)	0.87 (0.73-1.09)	0.004
CRP (mg/dL), median (Q1-Q3)	0.58 (0.28-1.16)	0.55 (0.21-1.12)	0.60 (0.34-1.21)	0.152
Total cholesterol (mg/dL)	178.33 ± 41.57	170.60 ± 39.90	185.95 ± 41.83	<0.001
HDL cholesterol (mg/dL)	37.05 ± 11.06	38.35 ± 12.13	35.78 ± 9.76	0.005
LDL cholesterol (mg/dL)	127.03 ± 38.43	123.21 ± 36.22	130.86 ± 40.23	0.018
Triglyceride (mg/dL) median (Q1-Q3)	127 (86-204)	90 (67-116)	201 (143-228)	<0.001

LVEF: Left ventricular ejection fraction; HT: Hypertension; DM: Diabetes mellitus; CKD: Chronic kidney disease; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CABG: Coronary artery by-pass graft; VT: Ventricular tachycardia, VF: Ventricular fibrillation; WBC: White blood cells (106/L), CRP: C-reactive protein; HDL-C: High-density lipoprotein cholesterol (mg/dL); LDL-C: 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.

Table 2. Comparison of baseline angiographic characteristics and short- and long-term mortality in patients with low and high triglyceride-glucose index

Variables	All Patients (n=573)	Low TyG ≤9.17 (n=284)	High TyG >9.17 (n=289)	p value
Type of MI, n (%)				0.353
Anterior MI	228 (39.8)	118 (41.5)	110 (38.1)	
Inferior MI	222 (38.7)	112 (39.4)	110 (38.1)	
Other	123 (21.5)	54 (19.0)	69 (23.9)	
Culprit artery, n (%)				0.581
LAD	278 (48.5)	133 (46.8)	145 (50.2)	
Cx	112 (19.5)	58 (20.4)	54 (18.7)	
RCA	155 (27.1)	76 (26.8)	79 (27.3)	
Other (IMA, diagonal)	28 (4.9)	17 (6.0)	11 (3.8)	
Follow-up duration (months)	39.28 ± 16.83	40.55 ± 16.21	38.04 ± 17.35	0.074
In-hospital mortality, n (%)	60 (10.5)	16 (5.6)	44 (15.2)	<0.001
First-year mortality, n (%)	104 (18.2)	31 (10.9)	73 (25.3)	<0.001
Five-year mortality, n (%)	150 (26.2)	56 (19.7)	94 (32.5)	<0.001

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

Table 3. Independent predictors of 1-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	0.199			
Gender	0.82	0.52-1.29	0.404			
Smoking	7.42	3.02-18.27	<0.001	1.51	0.55-4.09	0.416
LVEF	0.93	0.91-0.95	<0.001	0.97	0.95-1.00	0.058
Cardiogenic shock	1.83	0.89-3.79	0.100			
Pulmonary oedema	1.11	0.45-2.73	0.821			
Previous MI	6.44	4.30-9.63	<0.001	3.21	1.93-5.33	<0.001
HT	5.63	3.08-10.29	<0.001	3.20	1.63-6.29	0.001
DM	11.00	6.15-19.75	<0.001	2.50	1.19-5.25	0.015
CKD	5.64	3.68-8.64	<0.001	1.97	1.21-3.21	0.006
WBC	1.00	0.93-1.08	0.902			
Hemoglobin	1.02	0.92-1.14	0.614			
LDL-C	1.00	0.99-1.01	0.927			
TyG	2.56	1.66-3.86	<0.001	2.67	1.70-4.18	<0.001

LVEF: Left ventricular ejection fraction; MI: Myocardial infarction; HT: Hypertension; DM: Diabetes mellitus; CKD: Chronic kidney disease; WBC: White blood cells (106/L); LDL-C: Low-density lipoprotein cholesterol (mg/dL); TyG: Triglyceride-glucose index. Statistical significance is considered at a p-value of less than 0.05.

Conclusions: This study demonstrates that the TyG index may be a valuable biomarker for predicting one-year mortality in STEMI patients undergoing PCI. In addition to reflecting insulin resistance, it may facilitate early risk stratification and guide treatment decisions. Nevertheless, larger prospective studies are needed to confirm its independent prognostic value in this population.

OP-003 [Interventional Cardiology / Coronary]

Proximal side-branch optimization and re-intervention: A pooled analysis of EVOLUTE-CRUSH registries

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Background and Aim: The impact of proximal side-branch optimization (PSBO) after PCI with systematic 2-stent techniques for complex coronary bifurcation lesions on re-intervention remains unclear. The aim of this multicentric study was to evaluate the association between target lesion revascularization (TLR) and PSBO following complex coronary bifurcation PCI.

Methods: The study comprised 871 patients [male: 667 (76.5%), mean age: 60.44 ± 10.31 years] who underwent bifurcation PCI including mini-crush (n=302), double kissing-crush (n=218), nano-crush (n=129), T and small protrusion (TAP) (n=125), and mini-culotte (n=99) between January 2014 and January 2024 were included in the study from six tertiary centers. The primary endpoint was clinically driven TLR during follow-up. The secondary endpoint was defined as the major adverse cardiovascular events (MACE), cardiac death, target vessel myocardial infarction (TVMI), stent thrombosis, stroke, or all-cause death. The study group was divided into two groups: PSBO (+) and PSBO (-) (n=152) and PSBO (+) (n=719).

Results: Patients with PSBO (+) had older age, had more baseline comorbidities (eg. history of stroke, heart failure, and valve disease ≥ moderate). SYNTAX scores [23.66 ± 5.90 vs. 26.36 ± 7.02, p<0.001] was significantly lower in the PSBO (+) group than in the PSBO (-) group. However, the frequency of multiple lesions (71.3 vs. 61.8%, p=0.020) was notably higher in the PSBO (+) group compared to the PSBO (-) group. There was no difference between systematic 2-stent techniques in both groups. Moreover, the PSBO (+) had a longer follow-up time compared to the PSBO (-) group (31.38 ± 15.08 vs. 28.84 ± 14.76 months, p=0.049). The incidence of TLR (8.9 vs. 16.4%, p=0.005) was significantly higher in the PSBO (-) group compared to the PSBO (+) group and this was mainly driven side-branch-TLR (6.7 vs. 12.5%, p=0.014). Other endpoints were comparable between the two groups. The mid-term TLR (adjusted HR (IPW): 0.750, [95% CI: 0.422-1.333], p=0.327) rate in the overall population did not differ between the two groups. High SYNTAX score (adjusted HR (IPW): 1.077, p<0.001), non-fatal intra-procedural complications (adjusted HR (IPW): 6.767, p<0.001), utilization TAP technique (adjusted HR (IPW): 2.075, p=0.010), and left main localization of bifurcation lesion (adjusted HR (IPW): 2.710, p=0.002) were found to be independent predictors of primary endpoint.

Conclusions: This study does not show a stark association between the utilization of PSBO and side-branch TLR for planned 2-stent techniques for complex coronary bifurcation disease. Moreover, High SYNTAX score, non-fatal intra-procedural complications, utilization of the TAP technique, and left main bifurcation lesion were independent predictors of TLR.

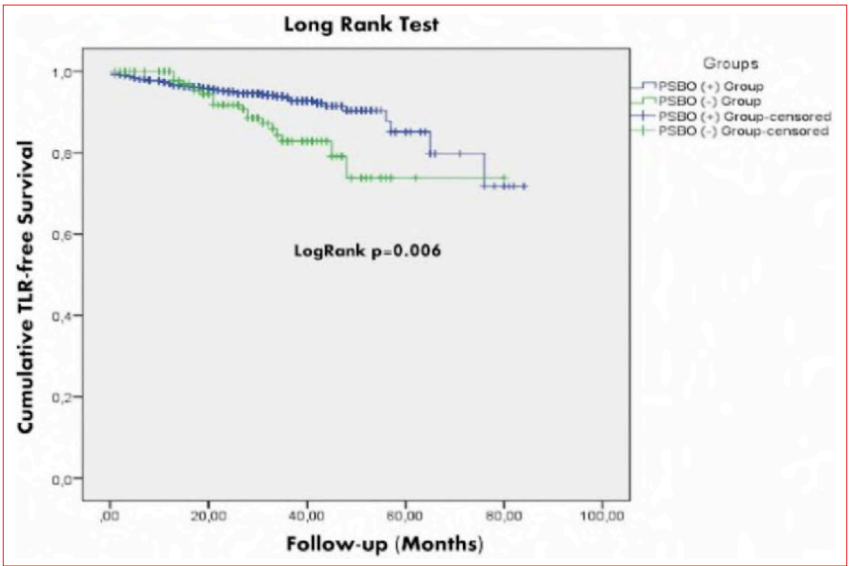

Figure 1. Kaplan Meier.

Table 1. Baseline demographic and clinical characteristics of per study group

Variables	PSBO (+) Group (n=719)	PSBO (-) Group (n=152)	P
Age (years)	60.76±10.44	58.93±9.70	**0.048**
Male, sex n (%)	551 (76.6)	116 (76.3)	0.933
Comorbidities, n(%)			
Hypertension	469 (65.2)	91 (59.9)	0.210
Diabetes mellitus	280 (38.9)	62 (40.8)	0.672
Hyperlipidemia	388 (54.0)	75 (49.3)	0.300
Chronic kidney disease	140 (19.5)	38 (25.0)	0.125
Current Smoker	368 (51.2)	76 (50.0)	0.791
History of Stroke	25 (3.5)	0 (0.0)	**0.014**
Prior PCI	236 (32.8)	44 (28.9)	0.353
Prior MI	224 (31.2)	43 (28.3)	0.486
Heart Failure	112 (15.6)	12 (7.9)	**0.014**
LV Ejection Fraction (%)	53.97±9.38	55.10±8.71	0.398
Moderate-severe valve disease, n (%)	86 (12.0)	8 (5.3)	**0.014**
Laboratory measurements			
White blood cell count, (10^9/L)	9.30±2.69	9.44±2.55	0.548
Hemoglobin, (g/dL)	13.48±1.95	13.93±1.74	**0.009**
Creatinine, (mg/dL)	.98±.55	1.07±.82	0.186
Platelet count, (10^9/L)	251.09±63.53	234.72±63.02	**0.004**
Total cholesterol, (mg/dL)	182.75±53.22	182.08±52.20	0.887
Clinical Presentation, n (%)			
CCS	310 (43.1)	73 (48.0)	0.268
NSTEMI	327 (45.5)	67 (44.1)	0.867
USAP	80 (11.1)	12 (7.9)	0.239
Medications Used, n (%)			
Acetylsalicylic acid	719 (100.0)	152 (100.0)	-
Clopidogrel	340 (47.2)	70 (46.1)	0.830
Ticagrelor	330 (45.9)	62 (40.8)	0.250
Prasugrel	49 (6.8)	20 (13.2)	**0.009**
Beta Blockers	682 (94.9)	139 (91.4)	0.101
CCB	104 (14.5)	24 (15.8)	0.675
ACEI/ARB	599 (83.3)	120 (78.9)	0.198
Statin	679 (94.4)	146 (96.1)	0.418
Diuretics	122 (17.0)	18 (11.8)	0.118
Insulin	180 (25.0)	41 (27.0)	0.618

*Independent Samples T-test **Chi-squared test ***Fisher's exact test †Mann–Whitney U test

ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, CCB: Calcium channel blocker; CCS: Chronic coronary syndrome, DK: Double kissing; LV: Left ventricle; MI: Myocardial infaction; NSTEMI: Non-ST segment elevation myocardial infarction; PCI: Percutaneous coronary intervention; USAP: Unstable angina pectoris.

Table 2. Lesions characteristics per study group

Variables	PSBO (+) Group (n=719)	PSBO (-) Group (n=152)	P
Multi-vessel disease, n(%)	475 (66.1)	88 (57.9)	0.056
SYNTAX score	23.66±5.90	26.36±7.02	**<0.001**
SYNTAX score ≤22, n(%)	316 (43.9)	45 (29.6)	**0.001**
SYNTAX score 23-32, n(%)	300 (41.7)	58 (38.2)	0.435
SYNTAX score ≥33, n(%)	103 (14.3)	49 (32.2)	**<0.001**
Locations of bifurcation lesions, n(%)			
LMCA	62 (8.6)	15 (9.9)	0.623
LAD-Diagonal	448 (62.3)	95 (62.5)	0.939
LCx-OM	187 (26.0)	38 (25.0)	0.796
PDA-PL	22 (3.1)	4 (2.6)	1.00
Medina classification, n(%)			
0.1.1	186 (25.9)	43 (28.3)	0.491
1.1.1	533 (74.1)	109 (71.7)	0.434
Lesion length, mm			
Main vessel	25.10±8.06	25.36±7.22	0.275
Side branch	16.20±5.25	16.03±6.07	0.203
Reference vessel diameter, mm			
Main vessel	3.48±10.20	3.24±2.32	0.729
Side branch	2.60±.25	2.61±.22	0.164
Assessment of complexity, n(%)			
Calcification ≥ moderate	250 (34.8)	72 (47.4)	**0.003**
Multiple lesions	513 (71.3)	94 (61.8)	**0.020**
Thrombus identified by angiography	82 (11.4)	26 (17.1)	0.053
SB stenosis ≥70% or 90%	670 (93.2)	143 (94.1)	0.688
Bifurcation angle<45° or >70°	358 (49.8)	95 (62.5)	**0.004**
MV reference diameter < 2.5mm	15 (2.1)	5 (3.3)	0.371
Main vessel, n(%)			
TIMI flow grade <3	84 (11.7)	22 (14.5)	0.339
Chronic total occlusion	20 (2.8)	5 (3.3)	0.788
Thrombus-containing lesion	43 (6.0)	13 (8.6)	0.240
Side branch, n(%)			
TIMI flow grade <3	89 (12.4)	18 (11.8)	0.855
Chronic total occlusion	8 (1.1)	2 (1.3)	0.689
Thrombus-containing lesion	41 (5.7)	14 (9.2)	0.106

*Chi-squared test **Mann–Whitney U test ***Fisher's exact test

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

Table 3. Procedural characteristics of the study groups

Parameters	PSBO (+) Group (n=719)	PSBO (-) Group (n=152)	P
Access site, n(%)			
Femoral	691 (96.1)	145 (95.4)	0.685
Radial	28 (3.9)	7 (4.6)	0.652
Tirofiban use during PCI, n (%)	95 (13.2)	26 (17.1)	0.207
Utilization of IVUS, n(%)	61 (8.5)	9 (5.9)	0.329
Thrombus Aspiration, n(%)	5 (0.7)	4 (2.6)	0.055
Pre-dilation			
Main vessel	550 (76.5)	108 (71.1)	0.156
Side branch	603 (83.9)	133 (87.5)	0.261
2-stent Techniques			
Mini-culotte	79 (11.0)	20 (13.1)	0.320
TAP	110 (15.3)	15 (9.9)	0.083
DK-crush	171 (23.8)	45 (29.6)	0.131
Mini-crush	253 (35.2)	49 (32.2)	0.487
Nano-crush	106 (14.7)	23 (15.1)	0.932
Final kissing balloon inflation	704 (97.9)	148 (97.4)	0.676
Main vessel			
Stent number, n	1.18±.38	1.20±.40	0.453
Stent diameter, mm	3.04±.40	3.04±.43	0.864
Stent length, mm	30.50±8.67	30.36±8.02	0.761
Side branch			
Stent number, n	1.03±.18	1.03±.18	0.954
Stent diameter, mm	2.56±.46	2.60±.38	0.187
Stent length, mm	20.91±6.22	20.56±6.58	0.358
Final POT, n (%)	698 (97.1)	144 (94.7)	0.144
Resource utilization, n (%)			
Total guiding catheter number	1.08±.33	1.11±.32	0.156
Total guidewire number	2.73±.84	2.89±.84	**0.013**
Total balloon number	5.85±2.12	5.81±2.02	0.785
Total stent number	2.20±.40	2.25±.49	0.366
Procedure time, min	68.22±24.29	68.56±23.36	0.587
Fluoroscopy time, min	21.71±7.40	20.94±7.33	0.214
Contrast media volume (mL)	209.28±66.95	205.56±62.28	0.533
Angiographic success, n(%)			
Main vessel	700 (97.4)	148 (97.4)	0.994
Side branch	701 (97.5)	147 (96.7)	0.583

*Chi-squared test **Fisher's exact test ***Mann–Whitney U test

AKI: Acute kidney injury; DK: Double kissing; IVUS: Intravascular ultrasound; PCI: Percutaneous coronary intervention; POT: Proximal optimization technique; TIMI: Thrombolysis in myocardial infarction.

Table 4. Comparison of non-fatal intraprocedural complications according to 2-stent techniques

Parameters	Mini-Culotte (N=99)	DK-crush (N=216)	Mini-crush (N=302)	Nano-crush (N=129)	TAP (N=125)	P value
Non-fatal Intraprocedural complication, n(%)	14 (14.1)	29 (13.4)	46 (15.2)	13 (10.1)	34 (27.2)	**0.002**
Abrupt occlusion						
MV	2 (2.0)	4 (1.9)	6 (2.0)	1 (0.8)	4 (3.2)	0.742
SB	2 (2.0)	4 (1.9)	8 (2.6)	3 (2.3)	8 (6.4)	0.145
TIMI<3						
MV	3 (3.0)	4 (1.9)	11 (3.6)	3 (2.3)	8 (6.4)	0.227
SB	3 (3.0)	10 (4.6)	9 (3.0)	4 (3.1)	8 (6.4)	0.472
Dissection						
MV	6 (6.1)	8 (3.7)	11 (3.6)	2 (1.6)	4 (3.2)	0.495
SB	1 (1.0)	1 (0.5)	7 (2.3)	3 (2.3)	6 (4.8)	0.090
Thrombus formation						
MV	2 (2.0)	4 (1.9)	3 (1.0)	2 (1.6)	2 (1.6)	0.924
SB	0 (0.0)	6 (2.8)	10 (3.3)	4 (3.1)	4 (3.2)	0.508
Coronary Perforation						
MV	0 (0.0)	0 (0.0)	1 (0.3)	0 (0.0)	0 (0.0)	0.757
SB	0 (0.0)	0 (0.0)	1 (0.3)	0 (0.0)	0 (0.0)	0.757

*Chi-squared test

Table 5. In-hospital and long-term outcomes per study group

Parameters	PSBO (+) Group (n=719)	PSBO (-) Group (n=152)	P
In-hospital complications, n (%)			
Death	9 (1.3)	2 (1.3)	1.00
Major bleeding	16 (2.2)	5 (3.3)	0.392
Pseudoaneurysm	20 (2.8)	5 (3.3)	0.788
Fatal arrhythmias	14 (1.9)	4 (2.6)	0.536
Stent thrombosis	5 (0.7)	1 (0.7)	1.00
Spontaneous MI	7 (1.0)	2 (1.3)	0.661
Contrast-induced AKI	79 (11.0)	17 (11.2)	0.944
Stroke	1 (0.1)	0 (0.0)	1.00
Follow-up time, month	31.38±15.08	28.84±14.76	**0.049**
Mid-term Outcomes, n (%)			
TLR (Primary endpoint)	64 (8.9)	25 (16.4)	**0.005**
MV-TLR	24 (3.3)	8 (5.3)	0.240
SB-TLR	48 (6.7)	19 (12.5)	**0.014**
Cardiac death	30 (4.2)	9 (5.9)	0.386
TVMI	50 (7.0)	17 (11.2)	0.075
Stent thrombosis	23 (3.2)	9 (5.9)	0.149
Stroke	7 (1.0)	1 (0.7)	1.00
All-cause death	42 (5.8)	12 (7.9)	0.340

AKI: Acute kidney injury; MACE: Major adverse cardiac events; MV: Main vessel; MI: Myocardial infarction; SB: Side branch; TLR: Target lesion revascularization: TVMI: Target vessel myocardial infarction.

Table 6. Cox regression analysis of potential predictor factors for primary endpoint (all-cause death)

Parameters	Unadjusted HR	95% CI	P value	Adjusted HR (IPW)	95% CI	P value
Intraprocedural complications (+) group	5.434	[3.134-9.433]	<0.001	3.067	[1.686-5.586]	**<0.001**
Age	1.036	[1.010-1.062]	0.006	1.022	[0.995-1.050]	0.109
SYNTAX Score	1.133	[1.085-1.185]	<0.001	1.074	[1.026-1.125]	**0.002**
Diabetes Mellitus	2.206	[1.277-3.811]	0.005	1.336	[0.743-2.402]	0.333
Chronic Kidney Disease	3.203	[1.851-5.542]	<0.001	1.536	[0.846-2.788]	0.159
LV Ejection fraction	0.959	[0.935-0.983]	0.001	0.970	[0.944-0.997]	**0.030**
Prior PCI	1.494	[0.860-2.597]	0.154	0.906	[0.501-1.638]	0.743
Bifurcation Localization (LMCA)	4.393	[2.339-8.253]	<0.001	2.988	[1.470-6.077]	**0.003**
Proximal side branch optimization	0.277	[0.160-0.479]	<0.001	0.331	[0.181-0.607]	**<0.001**
SB stentdiameter mm	1.052	[0.829-1.333]	0.678	1.098	[0.749-1.610]	0.632

OR: Odds ratio, CI: Confidence interval.

OP-004 [Interventional Cardiology / Coronary]

Comparing BCIS-JS and SYNTAX-II scores in predicting one-year mortality in stable angina patients: A study on efficiency and predictive accuracy

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Background and Aim: The SYNTAX-II score's application complexity in clinical settings continues to be an issue, despite its established predictive value in mortality. The British Cardiovascular Intervention Society's BCIS-JS score stands out as a practical substitute, providing a streamlined method that takes coronary grafts and myocardial risk into account. This study assesses the predictive power of the BCIS-JS and SYNTAX-II scores for all-cause 1-year mortality in patients with stable angina undergoing elective invasive coronary angiography.

Methods: We retrospectively assessed 567 patients presenting between May 2021 and May 2022 to our tertiary care hospital outpatient cardiology clinic with stable angina without coronary grafts. Calculations for BCIS-JS and SYNTAX-II scores were performed, followed by mortality outcome analyses to determine the predictive accuracy of each score. Statistical procedures included chi-square, Mann-Whitney U tests, Spearman correlation, and ROC curve analysis, which were conducted using IBM SPSS version 27.

Results: Among the cohort (289 males, median age 64), significant associations were found between higher scores and increased 1-year mortality for both BCIS-JS (median 10 (IQR 4-12) in deceased vs. 4 (IQR 0-6) in survivors; p<0.0001) and SYNTAX-II (median 20 (IQR 14-26) in deceased vs. 9 (IQR 5-16) in survivors; p<0.0001) (Table 1). The mean calculation time was markedly less for BCIS-JS at 44 seconds compared to 114 seconds for SYNTAX-II. ROC analysis identified a BCIS-JS score >6 with 61% sensitivity and 81% specificity (AUC=0.781) and a SYNTAX score >15 with 72% sensitivity and 73% specificity (AUC=0.764) as optimal thresholds for mortality prediction. In analysis with the Spearman test, a high level (r=0.718) positive correlation was detected between the BCIS-JS score and the SYNTAX-II score (p<0.001). Multivariate regression analysis demonstrated that BCIS-JS (OR: 1.2443, 95% CI: 1.1205-1.3817, p<0.0001) and SYNTAX-II (OR: 1.0553, 95% CI: 1.0083-1.1045, p=0.0207) were independent predictors of 1-year mortality (Table 2).

Conclusions: It is possible to accurately predict 1-year mortality using the BCIS-JS and SYNTAX-II scores. BCIS-JS is a viable risk stratification method because of its much-reduced time to calculation, which highlights its effectiveness and usability in hectic clinical settings. This efficiency, combined with its predictive accuracy, supports the adoption of BCIS-JS in clinical settings for improved patient management and outcomes.

Table 1. *Baseline characteristics and comparisons between survivors and non-survivors*

	Survivors (n=479)	**Non-survivors** (n=88)	**P value**
Sex (male), n (%)	235 (%49,4)	52 (%59,1)	0.094
Age (years)	62,1±9,4	63,3±10,9	0.323
BMI, kg/m^2	28,3±4,5	29,2±4,8	0.316
Risk factors, n (%)			
Hypertension	279 (%58,2)	52 (%59,1)	0.883
Diabetes mellitus	203 (%42,4)	49 (%55,7)	**0.021**
Coronary artery disease	181 (%37,8)	35 (%39,8)	0.724
Smoking	117 (%24,5)	13 (%14,8)	**0.047**
Family history	30 (%6,3)	4 (%4,5)	0.533
Previous medications, n (%)			
Antiaggregant	216 (%45,1)	42 (%47,7)	0.648
Anticoagulant	39 (%8,1)	5 (%5,7)	0.428
ACE/ARB	229 (%47,8)	48 (%54,5)	0.245
Beta Blocker	192 (%40,1)	39 (%44,3)	**0.047**
Calcium Channel Blocker	96 (%20,1)	15 (%17)	0.509
Statins	126 (%26,3)	21 (%23,9)	0.631
Diuretics	86 (%18)	23 (%26,1)	0.073
Laboratory data			
Glucose, mg/dL	107 (93-146)	122 (93-175)	0.205*
Creatinine, mg/dL	0,97 (0,81-1,13)	1,03 (0,87-1,27)	**0.015***
Triglyceride, mg/dL	139 (101-196)	146 (103-237)	0.172*
HDL-c, mg/dL	40 (33-48)	39 (32-48)	0.496*
Total cholesterol, mg/dL	180 (152-215)	190 (157-222)	0.147*
LDL-c, mg/dL	109 (84-137)	120 (89-143)	0.255*
Coronary artery score			
BCIS Jeopardy	4 (0-6)	10 (4-12)	**<0.001**
SYNTAX II	9 (5-16)	20 (14-26)	**<0.001**

*Mann Whitney U Test

Table 2. *Multivariate logistic regression analysis*

	Multivariate regression analysis		
	OR	%95 GA	P value
Diabetes mellitus	1,3463	0,7860-2,3060	0.2789
Smoking	0,5719	0,2796-1,1696	0.1258
Beta Blocker	1,1178	0,6520-1,9162	0.6856
Creatinine	1,3304	0,9296-1,9039	0.1186
BCIS Jeopardy	1,2443	1,1205-1,3817	**<0.0001**
SYNTAX II	1,0553	1,0083-1,1045	**0.0207**

OP-005 [Interventional Cardiology / Coronary]

The impact of CMR-guided revascularization on left ventricular recovery in patients with severe systolic dysfunction: a comparative analysis of CABG and PCI approaches

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Background and Aim: Cardiac magnetic resonance (CMR) imaging is a well-established modality for assessing myocardial viability in patients with significantly reduced left ventricular ejection fraction (LVEF). This study investigates the influence of a CMR-based revascularization strategy on long-term LV functional recovery and compares outcomes between coronary artery bypass grafting (CABG) and percutaneous coronary intervention (PCI).

Methods: We included 416 patients with LVEF <35%. In Group I (n=192), revascularization strategy was determined based on CMR viability assessment. Group II (n=224) underwent revascularization based on angiographic complexity. Both groups were further stratified according to anatomical revascularization completeness (complete vs. incomplete). LVEF was evaluated at 12, 18, and 24 months.

Results: At 12 months, both groups showed modest and statistically non-significant increases in LVEF (Group I: from 31.18 ± 2.58% to 34.74 ± 3.98%, p=0.6533; Group II: from 31.08 ± 2.53% to 34.97 ± 5.61%, p=0.1280). By 18 months, LVEF significantly improved in the CMR-guided group (to 41.91 ± 4.16%, p=0.0009), while Group II showed a non-significant trend (p=0.0897). At 24 months, Group I reached 46.8 ± 2.92% (p=0.00001), outperforming Group II (42.71 ± 4.99%, p=0.0727). In subgroup analyses, both CABG and PCI demonstrated similar improvements in LVEF over time, with no statistically significant differences (p>0.05). Additionally, anatomical completeness of revascularization did not correlate with superior outcomes, suggesting that functional revascularization may suffice even in anatomically incomplete cases.

Conclusions: CMR-guided viability assessment provides a more effective strategy for improving long-term LV function in patients with severely reduced LVEF compared to angiography-guided decisions. Both CABG and PCI offer comparable benefits when tailored to viability imaging, and full anatomical revascularization may not be essential for functional recovery. These findings highlight the value of CMR in optimizing individualized treatment strategies in advanced heart failure.

OP-006 [Interventional Cardiology / Coronary]

Impact of non-fatal intraprocedural complications of systematic 2-stent techniques on adverse events

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Background and Aim: The effect of non-fatal intraprocedural complications (NIC) during PCI with systematic 2-stent techniques for complex coronary bifurcation lesions on mid-term mortality and major adverse events has not been described. This multicenter study aimed to assess the association between NIC and mid-term mortality and adverse events after complex coronary bifurcation lesions (CBLs) defined by DEFINITION criteria revascularization by 2-stent techniques.

Methods: The study comprised 871 patients [male: 667 (76.5%), mean age: 60.43 ± 10.34 years] who underwent bifurcation PCI including mini-crush (n=302), double kissing-crush (n=218), nano-crush (n=129), T and small protrusion (TAP) (n=125), and mini-culotte (n=99) between January 2014 and January 2024 were included in the study from six tertiary centers. Non-fatal

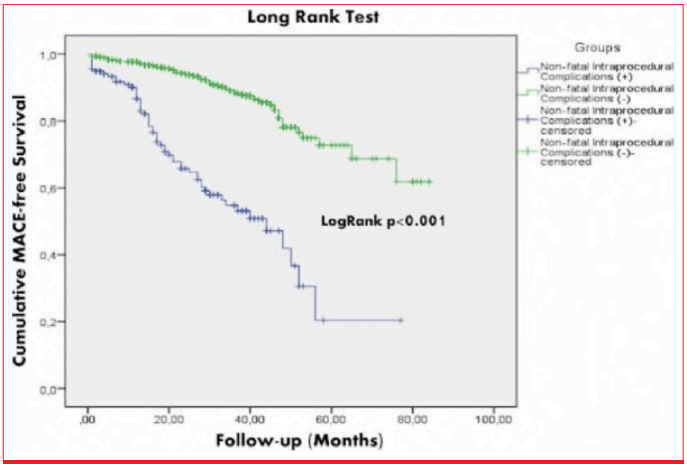

Figure 1. Kaplan Meier MACE.

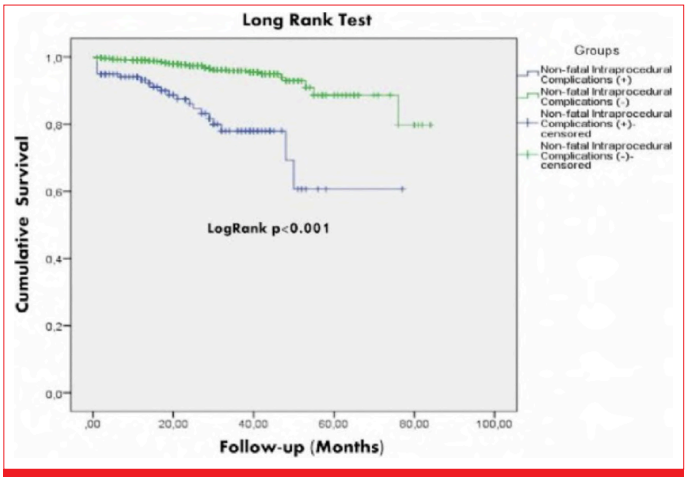

Figure 2. Kaplan Meier Mortality.

intraprocedural complications (NIC) included abrupt occlusion, worsening of TIMI flow grade (TIMI < 3), major dissection (greater than type B), occurrence of thrombus formation, and perforation of the main vessel or side branch. The primary endpoint was all-cause mortality during follow-up. The secondary endpoint was defined as the major adverse cardiovascular events (MACE), which include cardiac death, target vessel myocardial infarction (TVMI), or clinically driven target lesion revascularization (TLR). The study cohort was divided into 2 groups as NIC (+) (n=136) and NIC (-) (n=735).

Results: One or more NIC occurred in 136 of 871 patients (15.6%) after PCI. Patients with NIC (+) had more baseline comorbidities (eg. diabetes mellitus and chronic kidney disease). SYNTAX scores [26.69 ± 6.73 vs. 23.67 ± 5.98, p<0.001] was significantly higher in the NIC (+) group than in the NIC (-) group. However, angiographic success rates main vessel (89 vs. 98.9%, p<0.001) and side branch (90.4 vs. 98.6%, p<0.001) were notably lower in the NIC (+) group compared to the NIC (-) group. The incidence of all-cause death (16.2 vs. 4.2%, p<0.001), MACE (40.4 vs. 11.7%, p<0.001), cardiac death (14 vs. 2.7%, p<0.001) TVMI (22.8 vs. 4.9%, p=0.030), and clinically driven TLR (27.9 vs. 6.5%, p<0.001) were significantly higher in the NIC (+) group compared to the NIC (-) group. The mid-

Supplementary Table 1. Cox regression analysis of potential predictor factors for secondary endpoint (MACE)

Parameters	Unadjusted HR	95% CI	P value	Adjusted HR (IPW)	95% CI	P
Intraprocedural complications (+) group	5.050	[3.597-7.092]	<0.001	3.378	[2.331-4.901]	**<0.001**
Age	0.997	[0.981-1.013]	0.734	0.989	[0.973-1.005]	0.190
SYNTAX Score	1.097	[1.069-1.126]	<0.001	1.052	[1.024-1.081]	**<0.001**
Diabetes Mellitus	1.626	[1.168-2.263]	0.004	1.208	[0.851-1.716]	0.290
Chronic Kidney Disease	2.405	[1.699-3.404]	<0.001	1.540	[1.059-2.239]	**0.024**
LV Ejection fraction (%)	0.988	[0.971-1.005]	0.164	0.997	[0.980-1.015]	0.768
Prior PCI	1.265	[0.895-1.789]	0.183	0.954	[0.663-1.372]	0.798
Bifurcation localization (LMCA)	2.820	[1.814-4.383]	<0.001	2.575	[1.583-4.189]	**<0.001**
Proximal side branch optimization	0.311	[0.222-0.437]	<0.001	0.451	[0.313-0.650]	**<0.001**
SB stent diameter, mm	1.033	[0.870-1.227]	0.709	1.032	[0.795-1.339]	0.812

CI: Confidence interval; HR: Hazard ratio; IPW: Inverse probability weighting; LMCA: Left main coronary artery; LV: Left ventricle; MACE: Major adverse cardiac events; PCI: Percutaneous coronary intervention; SB: Side branch.

Table 1. Baseline demographic and clinical characteristics of per study group

Variables	Non-fatal Intraprocedural Complications (+) Group (n=136)	Non-fatal Intraprocedural Complications (-) Group (n=735)	P
Age (years)	59.57±10.61	60.59±10.29	0.342
Male, sex n (%)	101 (74.3)	566 (77.0)	0.488
Comorbidities, n(%)			
Hypertension	88 (64.7)	472 (64.2)	0.913
Diabetes mellitus	70 (51.5)	273 (37.1)	**0.002**
Hyperlipidemia	74 (54.4)	389 (52.9)	0.750
Chronic kidney disease	47 (34.6)	130 (17.7)	**<0.001**
Current Smoker	63 (46.3)	380 (51.7)	0.249
History of Stroke	4 (2.9)	21 (2.9)	0.957
Prior PCI	49 (36.0)	232 (31.6)	0.306
Prior MI	46 (33.8)	221 (30.1)	0.383
Heart Failure	49 (36.0)	232 (31.6)	0.306
LV Ejection Fraction (%)	53.45±9.66	54.34±9.18	0.290
Moderate-severe valve disease, n (%)	11 (8.1)	83 (11.3)	0.269
Laboratory measurements			
White blood cell count, (10^9/L)	9.46±2.65	9.32±2.71	0.609
Hemoglobin, (g/dL)	13.41±1.92	13.58±1.93	0.308
Creatinine, (mg/dL)	1.04±0.45	0.98±0.63	**0.017**
Platelet count, (10^9/L)	246.88±61.43	248.52±64.11	0.941
Total cholesterol, (mg/dL)	180.17±44.44	183.04±54.45	0.652
Clinical Presentation, n (%)			
CCS	70 (51.5)	313 (42.5)	0.051
NSTEMI	54 (39.7)	341 (46.4)	0.150
USAP	12 (8.8)	81 (11.0)	0.446
Medications Used, n (%)			
Acetylsalicylic acid	136 (100)	735 (100)	-
Clopidogrel	56 (41.2)	352 (47.9)	0.149
Ticagrelor	64 (47.1)	328 (44.6)	0.600
Prasugrel	16 (11.8)	53 (7.2)	0.071
Beta Blockers	123 (90.4)	698 (95)	0.037
CCB	20 (14.7)	108 (14.7)	0.997
ACEI/ARB	106 (77.9)	613 (83.4)	0.123
Statin	121 (89.0)	704 (95.8)	**0.001**
Diuretics	21 (15.4)	118 (16.1)	0.858
Insulin	42 (30.9)	180 (24.5)	0.116

*Mann–Whitney U test **Chi-squared test ***Fisher's exact test †Independent Samples T-test

ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, CCB: Calcium channel blocker; CCS: Chronic coronary syndrome, DK: Double kissing; LV: Left ventricle; MI: Myocardial infaction; NSTEMI: Non-ST segment elevation myocardial infarction; PCI: Percutaneous coronary intervention; USAP: Unstable angina pectoris.

Table 2. Lesions characteristics per study group

Variables	Non-fatal Intraprocedural Complications (+) Group (n=136)	Non-fatal Intraprocedural Complications (-) Group (n=735)	P
Multi-vessel disease, n(%)	89 (65.4)	475 (64.6)	0.855
SYNTAX score	26.69±6.73	23.67±5.98	**<0.001**
SYNTAX score ≤22, n(%)	33 (24.3)	328 (44.6)	**<0.001**
SYNTAX score 23-32, n(%)	59 (43.4)	297 (40.4)	0.517
SYNTAX score ≥33, n(%)	44 (32.4)	110 (15.0)	**<0.001**
Locations of bifurcation lesions, n(%)			
LMCA	13 (9.6)	64 (8.7)	0.748
LAD-Diagonal	85 (62.5)	457 (62.2)	0.943
LCx-OM	34 (25.0)	192 (26.1)	0.809
PDA-PL	4 (2.9)	22 (3.0)	1.00
Medina classification, n(%)			
0.1.1	40 (29.4)	188 (25.6)	0.450
1.1.1	96 (70.6)	547 (74.4)	0.387
Lesion length, mm			
Main vessel	24.62±7.13	25.23±8.06	0.638
Side branch	15.51±4.26	16.29±5.58	0.239
Reference vessel diameter, mm			
Main vessel	3.07±.51	3.06±.47	0.465
Side branch	2.61±.25	2.60±.24	0.677
Assessment of complexity, n(%)			
Calcification ≥ moderate	74 (54.4)	248 (33.7)	**<0.001**
Multiple lesions	85 (62.5)	523 (71.2)	**0.043**
Thrombus identified by angiography	28 (20.6)	79 (10.7)	**0.001**
SB stenosis ≥70% or 90%	126 (92.6)	687 (93.5)	0.724
Bifurcation angle<45° or >70°	84 (61.8)	370 (50.3)	**0.014**
MV reference diameter < 2.5mm	7 (5.1)	13 (1.8)	**0.025**
Main vessel, n(%)			
TIMI flow grade <3	11 (8.1)	94 (12.8)	0.122
Chronic total occlusion	7 (5.1)	18 (2.4)	0.093
Thrombus-containing lesion	13 (9.6)	42 (5.7)	0.090
Side branch, n(%)			
TIMI flow grade <3	12 (8.8)	94 (12.8)	0.194
Chronic total occlusion	2 (1.5)	8 (1.1)	0.660
Thrombus-containing lesion	14 (10.3)	40 (5.4)	0.031

*Chi-squared test **Mann–Whitney U test ***Fisher's exact test

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

Table 3. Procedural characteristics of the study groups

Parameters	Non-fatal Intraprocedural Complications (+) Group (n=136)	Non-fatal Intraprocedural Complications (-) Group (n=735)	P
Access site, n(%)			
Femoral	128 (94.1)	708 (96.3)	0.228
Radial	8 (5.9)	27 (3.7)	0.234
Tirofiban use during PCI, n (%)	43 (31.6)	77 (10.5)	**<0.001**
Utilization of IVUS, n(%)	12 (8.8)	58 (7.9)	0.713
Thrombus Aspiration, n(%)	6 (4.4)	3 (0.4)	**0.001**
Pre-dilation			
Main vessel	93 (68.4)	565 (76.9)	**0.034**
Side branch	113 (83.1)	623 (84.8)	0.620
2-stent Techniques			
Mini-culotte	14 (10.3)	85 (11.6)	0.668
TAP	34 (25.0)	91 (12.4)	**<0.001**
DK-crush	29 (21.3)	187 (25.4)	0.307
Mini-crush	46 (33.8)	256 (34.8)	0.821
Nano-crush	13 (9.6)	116 (15.8)	0.061
Final kissing balloon inflation	132 (97.1)	720 (98.0)	0.509
Main vessel			
Stent number, n	29 (21.3)	131 (17.8)	0.333
Stent diameter, mm	3.02±.45	3.41±10.04	0.360
Stent length, mm	30.34±8.06	30.50±8.65	0.791
Side branch			
Stent number, n	4 (2.9)	24 (3.3)	1.00
Stent diameter, mm	2.54±.54	2.60±.93	0.671
Stent length, mm	20.44±6.16	20.92±6.30	0.165
Proximal side-branch optimization, n **(%)**	95 (69.9)	624 (84.9)	**<0.001**
Final POT, n (%)	129 (94.9)	713 (97.0)	0.198
Resource utilization, n (%)			
Total guiding catheter number	1.12±.32	1.08±.33	0.111
Total guidewire number	2.92±.81	2.73±.84	**0.003**
Total balloon number	5.59±2.01	5.89±2.12	0.072
Total stent number	2.24±.48	2.20±.41	0.485
Procedure time, min	70.72±25.77	67.81±23.80	0.387
Fluoroscopy time, min	22.36±8.52	21.43±7.16	0.361
Contrast media volume (mL)	218.93±66.90	206.73±65.86	0.064
Angiographic success, n(%)			
Main vessel	121 (89.0)	727 (98.9)	**<0.001**
Side branch	123 (90.4)	725 (98.6)	**<0.001**

*Chi-squared test **Fisher's exact test ***Mann–Whitney U test

DK: Double kissing; IVUS: Intravascular ultrasound; PCI: Percutaneous coronary intervention; POT: Proximal optimization technique; TIMI: Thrombolysis in myocardial infarction.

Table 4. Comparison of non-fatal intraprocedural complications according to 2-stent techniques

Parameters	Mini-Culotte (N=99)	DK-crush (N=216)	Mini-crush (N=302)	Nano-crush (N=129)	TAP (N=125)	P value
Non-fatal Intraprocedural complication, n(%)	14 (14.1)	29 (13.4)	46 (15.2)	13 (10.1)	34 (27.2)	**0.002**
Abrupt occlusion						
MV	2 (2.0)	4 (1.9)	6 (2.0)	1 (0.8)	4 (3.2)	0.742
SB	2 (2.0)	4 (1.9)	8 (2.6)	3 (2.3)	8 (6.4)	0.145
TIMI<3						
MV	3 (3.0)	4 (1.9)	11 (3.6)	3 (2.3)	8 (6.4)	0.227
SB	3 (3.0)	10 (4.6)	9 (3.0)	4 (3.1)	8 (6.4)	0.472
Dissection						
MV	6 (6.1)	8 (3.7)	11 (3.6)	2 (1.6)	4 (3.2)	0.495
SB	1 (1.0)	1 (0.5)	7 (2.3)	3 (2.3)	6 (4.8)	0.090
Thrombus formation						
MV	2 (2.0)	4 (1.9)	3 (1.0)	2 (1.6)	2 (1.6)	0.924
SB	0 (0.0)	6 (2.8)	10 (3.3)	4 (3.1)	4 (3.2)	0.508
Coronary Perforation						
MV	0 (0.0)	0 (0.0)	1 (0.3)	0 (0.0)	0 (0.0)	0.757
SB	0 (0.0)	0 (0.0)	1 (0.3)	0 (0.0)	0 (0.0)	0.757

*Chi-squared test

Table 5. In-hospital and long-term outcomes per study group

Parameters	Non-fatal Intraprocedural Complications (+) Group (n=136)	Non-fatal Intraprocedural Complications (-) Group (n=735)	P
In-hospital complications, n (%)			
Death	8 (5.9)	3 (0.4)	**<0.001**
Major bleeding	5 (3.7)	16 (2.2)	0.354
Pseudoaneurysm	4 (2.9)	21 (2.9)	1.00
Fatal arrhythmias	11 (8.1)	7 (1.0)	**<0.001**
Stent thrombosis	6 (4.4)	0 (0.0)	**<0.001**
Spontaneous MI	9 (6.6)	0 (0.0)	**<0.001**
Contrast-induced AKI	19 (14.0)	77 (10.5)	0.232
Stroke	0 (0.0)	1 (0.1)	1.00
Follow-up time, month	23.48±15.39	32.31±14.74	**<0.001**
Mid-term Outcomes, n (%)			
All-cause death	22 (16.2)	31 (4.2)	**<0.001**
MACE	55 (40.4)	86 (11.7)	**<0.001**
Cardiac death	19 (14.0)	20 (2.7)	**<0.001**
TLR	38 (27.9)	48 (6.5)	**<0.001**
TVMI	31 (22.8)	36 (4.9)	**<0.001**
Stent thrombosis	18 (13.2)	14 (1.9)	**<0.001**
Stroke	1 (0.7)	6 (0.8)	1.00

AKI: Acute kidney injury; MACE: Major adverse cardiac events; MI: Myocardial infarction; TLR: Target lesion revascularization: TVMI: Target vessel myocardial infarction.

Table 6. Cox Regression analysis of potential predictor factors for primary endpoint (all-cause death)

Parameters	Unadjusted HR	95% CI	P value	Adjusted HR (IPW)	95% CI	P value
Intraprocedural complications (+) group	5.434	[3.134-9.433]	<0.001	3.067	[1.686-5.586]	**<0.001**
Age	1.036	[1.010-1.062]	0.006	1.022	[0.995-1.050]	0.109
SYNTAX Score	1.133	[1.085-1.185]	<0.001	1.074	[1.026-1.125]	**0.002**
Diabetes Mellitus	2.206	[1.277-3.811]	0.005	1.336	[0.743-2.402]	0.333
Chronic Kidney Disease	3.203	[1.851-5.542]	<0.001	1.536	[0.846-2.788]	0.159
LV Ejection fraction	0.959	[0.935-0.983]	0.001	0.970	[0.944-0.997]	**0.030**
Prior PCI	1.494	[0.860-2.597]	0.154	0.906	[0.501-1.638]	0.743
Bifurcation Localization (LMCA)	4.393	[2.339-8.253]	<0.001	2.988	[1.470-6.077]	**0.003**
Proximal side branch optimization	0.277	[0.160-0.479]	<0.001	0.331	[0.181-0.607]	**<0.001**
SB stentdiameter mm	1.052	[0.829-1.333]	0.678	1.098	[0.749-1.610]	0.632

OR: Odds ratio, CI: Confidence interval.

term mortality (adjusted HR (IPW): 3.067, [95% CI: 1.686-5.586], p<0.001) and MACE (adjusted HR (IPW): 3.378, [95% CI: 2.331-4.901], p<0.001) rates in the overall population notably differed between the NIC (+) group and the NIC (-) group.

Conclusions: This multicenter study suggests that NICs were strongly associated with mortality and MACE after planned 2-stent techniques for complex coronary bifurcation disease.

OP-007 [Other]

AI-driven digital health history system for perioperative cardiac risk assessment in non-cardiac surgery

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Background and Aim: Digital health history devices offer promising tools to enhance the quality and efficiency of medical consultations. This study evaluated the effectiveness of DHHDs, specifically the CAPE software (Comprehensive AI-Assisted Preoperative Evaluation), in perioperative cardiac risk assessment and management for patients undergoing non-cardiac surgery

Methods: The CAPE software was developed for perioperative cardiac risk assessment in non-cardiac surgery patients, based on the latest guidelines. A total of 405 patients were assigned to two groups: 205 received routine cardiology consultations, and 200 were assessed using the CAPE software. Consultation notes for both groups were uploaded to the electronic health record. A key feature of CAPE is an interactive questionnaire completed by patients before their consultation. Using yes/no/uncertain options, this structured approach minimizes subjective interpretation and confusion. The software is developed in Turkish, making it accessible for patients with a primary school education level. The software analyzes the patient's medical history and calculates the Revised Cardiac Risk Index score according to the latest guidelines. It also determines the need for tests such as cardiac biomarkers, echocardiography, and coronary CT angiography, following recommendations from ESC, AHA, and CCS guidelines. Based on the evaluation, one of two recommendations is made: No additional postoperative monitoring is required. A consultation note is prepared, recommending daily

troponin testing for 48-72 hours and ECG monitoring in the PACU. Patients are stratified into low-, intermediate-, and high-risk categories based on surgical bleeding risk. The software assesses thrombotic and bleeding risks and provides recommendations for managing antithrombotic therapy. It also offers guidance for the perioperative management of other cardiovascular medications.

Results: The CAPE software group demonstrated a significant reduction in hypertensive crises (p=0.010) and a decreasing trend in hypotension (p=0.064) and arrhythmias (p=0.081). Overall cardiovascular and cerebrovascular complication rates were significantly lower in this group (p<0.01). Among non-cardiac complications, a significant reduction in major bleeding was noted (p=0.048). The time to complete all assessments and finalize consultation notes was shorter in the CAPE software group (1.53 ± 1.12 days vs. 6.38 ± 8.50 days, p<0.01). Blinded raters scored computer-generated notes higher in overall impression, thoroughness, usefulness, organization, and adherence to guidelines (all adjusted p<0.001).

Conclusions: CAPE software-generated consultation notes were more comprehensive, organized, and guideline-consistent than physician-documented notes. The software also reduced perioperative complications, including hypertensive crises, hypotension, arrhythmias, and major bleeding. These findings suggest that, under physician oversight, DHHDs like CAPE can improve consultation quality and clinical outcomes.

A 58-year-old male is scheduled for transurethral resection of the prostate (TURP). He had an acute myocardial infarction 4 years ago and underwent primary percutaneous coronary intervention (PCI) with drug-eluting stent placement in the left anterior descending artery (LAD). No critical lesions requiring intervention were found in other coronary vessels. The patient reports no cardiac symptoms and describes his exercise capacity as greater than 4 meters. He has a diagnosis of hypertension (HT), which is well-controlled on treatment. He does not have diabetes mellitus (DM) and quit smoking 4 years ago. His last echocardiogram (09/19/2022) showed an ejection fraction (EF) of 50%, anteroapical hypokinesis, and grade 1 diastolic dysfunction.
Current Medications: Aspirin 100 mg 1×1, Atorvastatin 20 mg 1×1, Perindopril-indapamide 5/1.25 mg 1×1, Metoprolol succinate 50 mg 1×1
The planned surgery is categorized as high surgical bleeding risk, while the patient's individual thrombotic and bleeding risks are assessed as low. The Revised Cardiac Risk Index (RCRI) score is 1. Given the RCRI score ≥1, an NT-proBNP test was conducted, which returned a value of <300 mg/L. **Based on these findings, the patient is recommended for surgery with the following management plan:**
Cardiovascular Testing: No additional cardiovascular testing is deemed necessary preoperatively.
Postoperative Monitoring: No routine postoperative cardiovascular monitoring is indicated.
Medications: Aspirin should be withheld 7 days before surgery and restarted as soon as possible postoperatively. Metoprolol should be continued perioperatively, but if the patient's systolic blood pressure is low preoperatively, the dose may need to be reduced or stopped prior to surgery. Perindopril-indapamide should be discontinued 24 hours before surgery and restarted on the second postoperative day if the patient remains hemodynamically stable. Atorvastatin should be continued throughout the perioperative period.
Early Mobilization: Early mobilization should be encouraged to promote recovery, reduce the risk of thromboembolic complications, and enhance overall patient outcomes.

Figure 1. Representative consultation note created by the CAPE system for one of the enrolled patients.

OP-008 [Other]

Effects of SGLT-2 inhibitors on interatrial conduction delay in patients with diabetes mellitus

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Background and Aim: Sodium-glucose cotransporter-2 (SGLT2) inhibitors have been shown to reduce the incidence of atrial fibrillation (AF) in patients with Diabetes Mellitus (DM). Atrial electromechanical delay (EMD) is an electrophysiological parameter associated with the development of AF. This study was designed to investigate whether there is a change in atrial EMD in patients with type 2 DM who had SGLT2 inhibitors added to their existing metformin therapy. Specifically, the aim was to compare atrial EMD measurements taken before the initiation of SGLT2 inhibitors with those measured at the 4-month follow-up.

Methods: This study is a retrospective analysis conducted on patients diagnosed with type 2 DM. Patients aged between 35 and 75 years with a diagnosis of type 2 DM were included in the study. The diagnosis and treatment of DM were determined and applied according to current clinical guidelines. Exclusion criteria were: age over 75, insulin-dependent DM, coronary artery disease, heart failure, moderate to severe valvular heart disease, any arrhythmia, obstructive sleep apnea syndrome, chronic lung disease, liver failure, hypertension, systemic inflammatory diseases, acute renal failure, malignancy, history of cerebrovascular events, history of pulmonary embolism, hypothyroidism and hyperthyroidism, and body mass index (BMI) >35. Clinical data, medications used, and laboratory characteristics of the patients were obtained from hospital records. After applying the exclusion criteria, 50 patients were included in the study. Baseline measurements were compared with measurements taken at the 4th month follow-up. The study was approved by the Ethics Committee of Dışkapı Yıldırım Beyazıt Training and Research Hospital with decision number 114/15 dated 28/06/2021. The study was conducted in accordance with the ethical principles and standards of the Helsinki Declaration and Good Clinical Practice Guidelines.

Results: Left intraatrial EMD (19.58 ± 7.53 vs. 16.76 ± 7.53; p<0.001), right intraatrial EMD (14.42 ± 5.81 vs. 12.46 ± 8.05; p=0.016), and interatrial EMD (34 ± 9.41 vs. 29.22 ± 9.44; p<0.001) were found to have significantly decreased at follow-up. Subgroup analyses conducted in the dapagliflozin and empagliflozin groups revealed no statistically significant differences.

Conclusions: In diabetic patients who were started on SGLT2 inhibitors, a significant reduction in atrial EMD was observed over a 4-month follow-up period, along with favorable metabolic effects. These findings contribute significantly to the literature regarding the potential protective effects of SGLT2 inhibitors in reducing the incidence of atrial fibrillation.

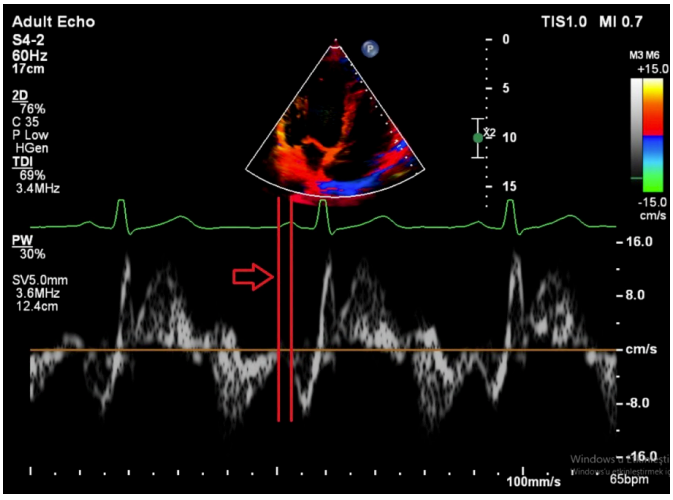

Figure 1. Measurement of EMD (PA interval) by tissue doppler imaging.

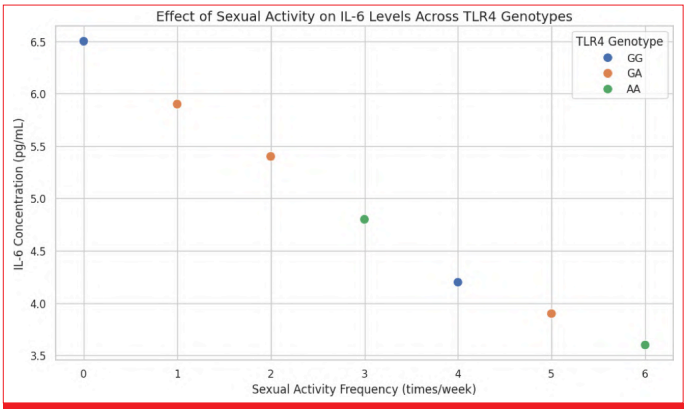

Figure 1. Correlation between TLR4 polymorphism (rs4986790) and IL-6 levels under stress. Individuals carrying the G allele show significantly higher IL-6 expression, suggesting enhanced inflammatory responses. Data simulated for illustration.

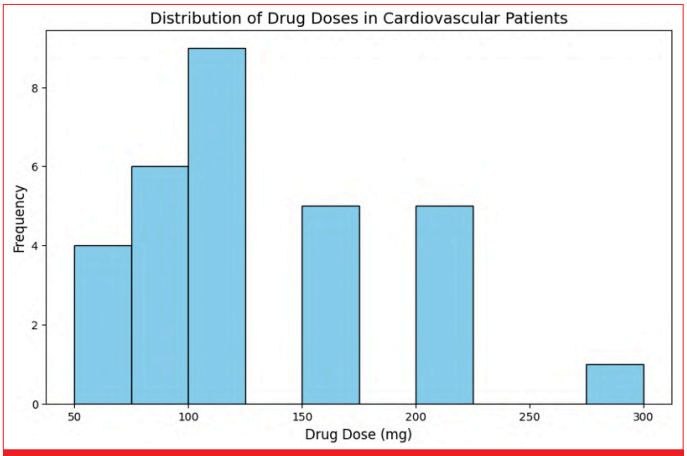

Figure 2. Histogram depicting the distribution of cardiovascular drug dosage among patients, highlighting variability and trends in medication consumption across different patient groups.

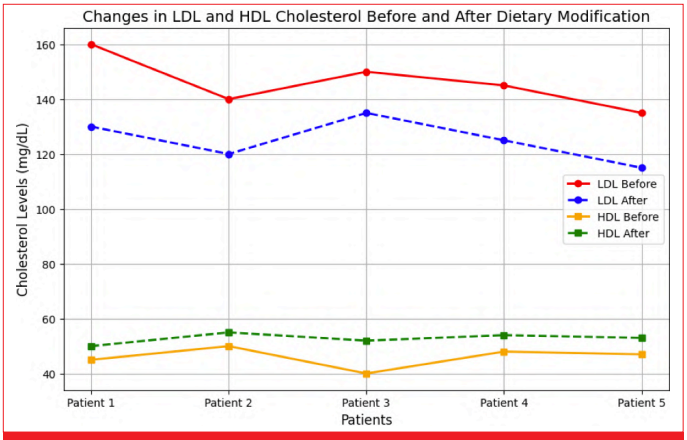

Figure 3. Dietary intervention reduces LDL and increases HDL levels in patients with cardiovascular risk (n=1,200; *p<0.001). Data demonstrate the synergistic effects of nutritional modification on lipid profiles in genetically susceptible individuals (e.g., PCSK9 variants).

OP-009 [Other]

Stress, sex, and immunogenetics: Cardiovascular and transgenerational effects

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Background and Aim: Cardiovascular diseases (CVDs) are influenced by complex interactions between genetic and environmental factors, with stress and sexual activity playing key roles in modulating immune responses. This study aimed to investigate polymorphisms in genes such as TLR4 rs4986790, IL-1β, and HLA-DQA1, which regulate immune responses to stress and sexual activity, and their impact on transgenerational CVD risk.

Methods: This multicenter study (N=1,506, Iran/Turkey) analyzed inflammatory markers (IL-6, TNF-α, CRP), epigenetic modifications (DNA methylation), and hormonal dynamics. We genotyped participants for key variants and measured inflammatory and hormonal responses, including IL-6 methylation and sex hormone levels.

Results: Hyperinflammatory genotypes enhanced stress-induced vascular inflammation (↑IL-6 +58%, p<0.001), while regular sexual activity reduced inflammation (↓hs-CRP 35%, p=0.01). Epigenetic markers (such as IL-6 methylation, β=0.15, p=0.004) linked maternal stress to increased CVD risk in offspring. Testosterone increased inflammation, while estrogen exerted a protective effect.

Conclusions: Sexual activity improves endothelial function and reduces systemic inflammation, offering a potential protective effect against CVD. This study proposes a new immunogenetic model for CVD risk stratification, integrating genetic susceptibility, epigenetic inheritance, and lifestyle factors. Epigenetic editing and genetic screening could enable preventive and personalized approaches.

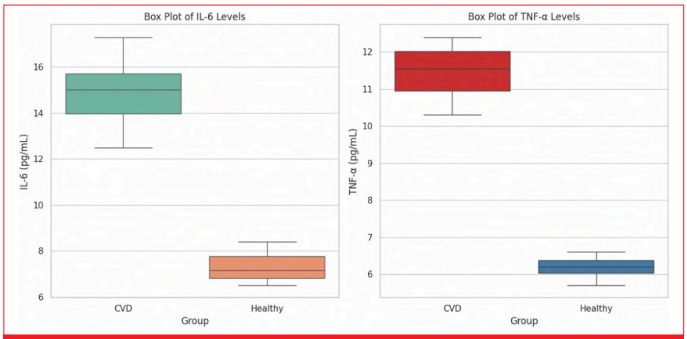

Figure 4. Comparison of serum IL-6 and TNF-α levels between patients with cardiovascular disease and healthy controls. The box plots show significantly higher levels of IL-6 and TNF-α in the patient group compared to the control group (p<0.001).

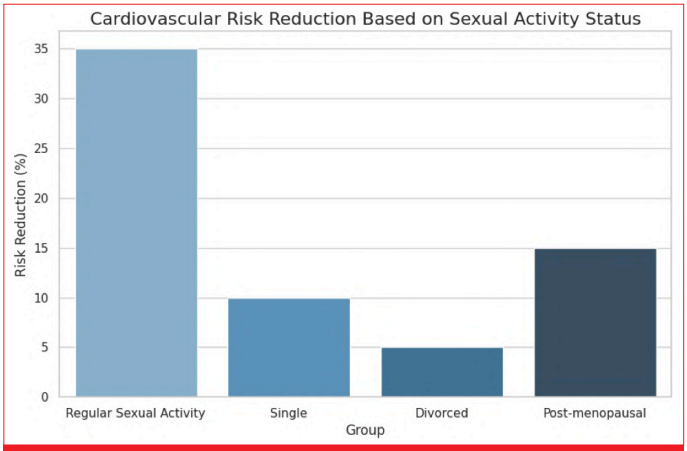

Figure 5. Bar chart showing the reduction in cardiovascular disease risk across different groups: individuals with regular sexual activity, singles, divorced, and postmenopausal women.

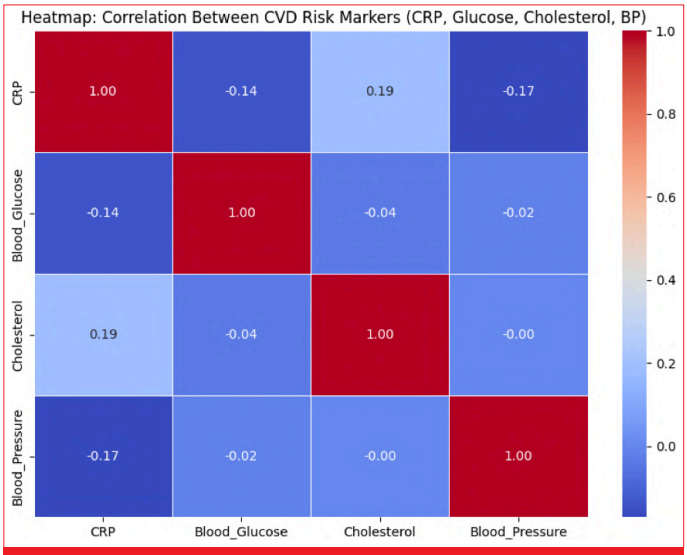

Figure 6. Correlation matrix of cardiovascular risk factors. Pearson coefficients (r) illustrate relationships between hs-CRP, fasting glucose, total cholesterol, and systolic blood pressure. Red indicates strong positive correlations (synergistic risk), while blue/white reflects weak or independent associations.

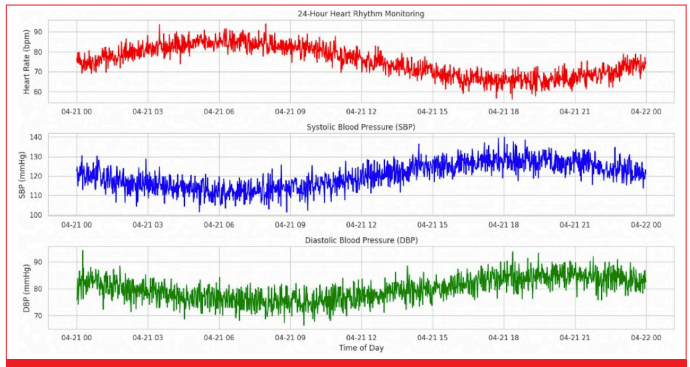

Figure 7. 24-hour fluctuations in blood pressure and heart rhythm (Holter monitoring data). Circadian variations and stress-triggered episodes are marked with arrows.

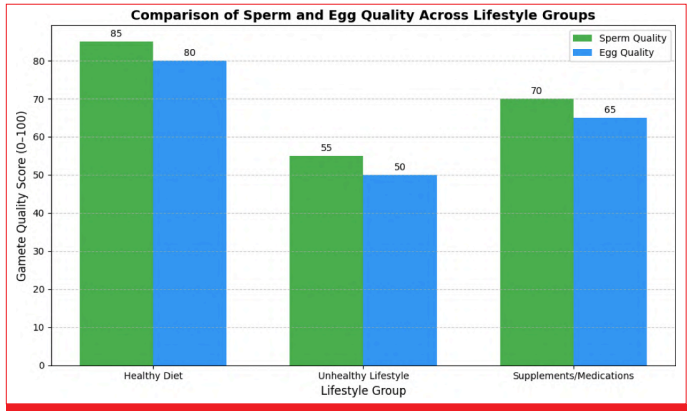

Figure 8. Gamete quality index scores across lifestyle groups. Healthy diet associates with superior gamete metrics versus unhealthy lifestyle (*p<0.05). Data shown as mean ± SD.

Table 1. Climatic conditions of study sites

City	Climate Type	Max Temp (°C)	Key Stressors
Şanlıurfa	Hot, arid	42	Heat-induced oxidative stress
Bandar Abbas	Hot, humid	38	Humidity-driven inflammation
Samsun/Tabriz	Temperate	32	Seasonal variability

Table 2. Medication classes, examples with dosage, and corresponding monitoring parameters for clinical management

Medication Class	Examples (Dosage)	Monitoring Parameters
Antihypertensives	Bisoprolol (2.5–10 mg/day)	BP, heart rate variability
Immunomodulators	Hydroxychloroquine (200–400 mg)	CRP, IL-6 levels
Anticoagulants	Rivaroxaban (10–20 mg/day)	D-dimer, bleeding risk
Thyroid Agents	Levothyroxine (25–150 mcg/day)	TSH, free T4

Table 3. Laboratory tests, methods, devices/platforms, and reference ranges used for analysis

Test	Method	Device/Platform	Reference Range
Fasting glucose	Glucose oxidase	Cobas 6000 (Roche)	70-100 mg/dL
HbA1c	HPLC	Bio-Rad D-10	4-6%
Lipid panel	Enzymatic colorimetry	Architect i2000 (Abbott)	LDL <100 mg/dL*, HDL >40
hs-CRP	Immunoturbidimetry	Cobas c501 (Roche)	Low risk: <1 mg/L
NT-proBNP	Electrochemiluminescence	Elecsys (Roche)	<125 pg/mL

Table 4. Laboratory tests and their clinical relevance in cardiovascular disease (CVD) risk assessment

Test	Method	Key Clinical Relevance
TSH/fT4	CLIA	Thyroid-CVD interplay
Cortisol (AM)	CLIA (Elecsys)	Stress axis activation
Testosterone/Estradiol	LC-MS/MS	Sex-specific CVD risk stratification

Table 5. Genetic variants and their associated methods, along with their relevance to cardiovascular diseases (CVD)

Gene	Variant	Method	CVD Relevance
IL1B	rs16944 (-511C/T)	PCR-RFLP	Plaque instability
HLA-DQA1	*05:01	SSP-PCR	Autoimmune modulation

Table 6. Overview of diagnostic tests, protocols used, and corresponding outcome measures

Test	Protocol	Outcome Measures
2D Echocardiography	Philips EPIQ 7 (LVEF by Simpson's biplane)	Diastolic dysfunction grading
Coronary CTA	Siemens Force 256-slice (SCCT guidelines)	CAC scoring, plaque characterization
24-hr ABPM	Spacelabs 90217 (AHA 2022 criteria)	Nocturnal BP dipping patterns

Table 7. Regional variations in cardiovascular biomarkers (mean ± SD)

City	TC (mg/dL)	hs-CRP (mg/L)	NT-proBNP (pg/mL)	TnT (ng/mL)	Glucose (mg/dL)	K+ (mmol/L)
Şanlıurfa	210 ± 42*	4.5 ± 1.7*	145 ± 32*	0.045 ± 0.016*	118 ± 22*	4.4 ± 0.4
Adana	208 ± 41*	4.2 ± 1.6*	142 ± 31*	0.044 ± 0.015*	116 ± 21*	4.3 ± 0.4
Tehran	198 ± 35	3.2 ± 1.1	130 ± 25	0.038 ± 0.012	112 ± 18	4.1 ± 0.3
Urmia	185 ± 28†	2.8 ± 0.9†	120 ± 20†	0.030 ± 0.010†	105 ± 15†	4.0 ± 0.2

Table 8. Genetic polymorphisms in CVD patients

Gene	Variant	CVD Cohort (%)	Controls (%)	OR (95% CI)	Phenotypic Association
AGT	rs699 (M235T)	12.3*	5.1	2.6 (1.9–3.6)	Resistant hypertension
FBN1	rs137854421	7.5*	0.3	26.4 (8.1–85.7)	MVP, aortic root dilation
SCN5A	rs7626962	5.0*	0.2	27.1 (6.3–116.3)	Ventricular arrhythmias

*FDR-corrected p<0.001.

Table 9. Inflammatory markers in CVD vs. controls

Marker	CVD Patients	Controls	p-value	Clinical Implication
IL-6	12.5 ± 4.2 pg/mL	3.8 ± 1.2	<0.001	Predicts HFpEF progression (AUC=0.82)
TNF-α	9.8 ± 3.1 pg/mL	2.6 ± 1.0	<0.001	Correlates with CAD severity (r=0.68)
hs-CRP	4.2 ± 1.5 mg/L	1.8 ± 0.6	<0.001	ASCVD risk stratification

Table 10. Pathogen seroprevalence in CVD

Pathogen	CVD (%)	Controls (%)	OR (95% CI)	Proposed Mechanism
H. pylori	53.2*	20.1	4.5 (3.1–6.5)	Chronic endothelial inflammation
C. pneumoniae	30.1*	10.5	3.7 (2.3–5.8)	Foam cell formation
CMV	45.8*	12.2	6.1 (4.0–9.3)	T-cell senescence → immunosenescence

Table 11. Hormonal dynamics following sexual activity

Activity Pattern	Testosterone (ng/dL)	Progesterone (ng/mL)	TSH (mIU/L)	Free T3 (pg/mL)	Free T4 (ng/dL)
Regular (pre-coital)	455 ± 35	1.2 ± 0.3	2.1 ± 0.5	3.1 ± 0.4	1.2 ± 0.2
Regular (24h post)	520 ± 42*	1.5 ± 0.4*	2.3 ± 0.6	3.3 ± 0.5	1.3 ± 0.3
Irregular (pre-coital)	430 ± 30	1.1 ± 0.2	2.2 ± 0.5	3.0 ± 0.4	1.0 ± 0.2
Irregular (24h post)	510 ± 40*	1.6 ± 0.5*	2.5 ± 0.7	3.5 ± 0.6	1.4 ± 0.3

Table 12. Cardiovascular and inflammatory parameters across different physical activity patterns

Parameter	Regular Activity	Irregular Activity	Abstinence	p-trend
CAD Incidence (/1,000)	3.2	5.8	7.1	<0.001
Systolic BP (mmHg)	-8.5*	-2.1	+3.2	0.003
hs-CRP (mg/L)	1.4 ± 0.6	2.9 ± 1.1	3.5 ± 1.3	<0.001
Heart Rate Variability	↑ RMSSD 28%	↔	↓ LF/HF ratio	0.008

OP-010 [Other]

The predictive value of prognostic immune-nutritional scores for in-hospital mortality in infective endocarditis: Insights from a multicenter study

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Background and Aim: Infective endocarditis (IE) remains associated with significant in-hospital mortality despite advances in diagnosis and treatment. In recent years, prognostic immune-nutritional scores have emerged as valuable tools in predicting outcomes in various diseases. However, their prognostic utility in IE has not been fully explored.

Methods: We retrospectively analyzed 582 patients diagnosed with definite or possible IE. Patients were divided into two groups based on in-hospital mortality status. Demographic, clinical, laboratory, and echocardiographic data were compared. Prognostic scores including the Osaka Prognostic Score (OPS), CALLY index, PNI, and HALP were calculated. Univariable and multivariable logistic regression analyses were performed to identify independent predictors of in-hospital mortality. ROC curve analysis was used to assess the predictive accuracy of key variables.

Results: In-hospital mortality occurred in 159 patients (27.3%). Non-survivors had significantly higher OPS and lower CALLY Index and PNI scores. In multivariable analysis, PCI history (OR=2.122, p=0.011), cancer history (OR=2.140, p=0.049), >10 mm vegetation (OR=2.658, p<0.001), SPAP (OR=1.027, p=0.003), WBC (OR=1.043, p=0.024), platelet count (OR=0.997, p=0.039), AST (OR=1.003, p=0.023), and OPS (OR=2.470, p<0.001) were identified as independent predictors of in-hospital mortality. ROC curve analysis demonstrated that OPS had good discriminative ability (AUC=0.689), with a cut-off value of 2.5 yielding 67% sensitivity and 68% specificity.

Conclusions: In conclusion, immune-nutritional indices, especially the Osaka Prognostic Score (OPS), provide valuable prognostic information in patients with infective endocarditis. OPS was independently associated with in-hospital mortality and offers a simple, accessible tool for early risk assessment. Future prospective studies should explore the utility of integrating immune-nutritional status into treatment algorithms for infective endocarditis.

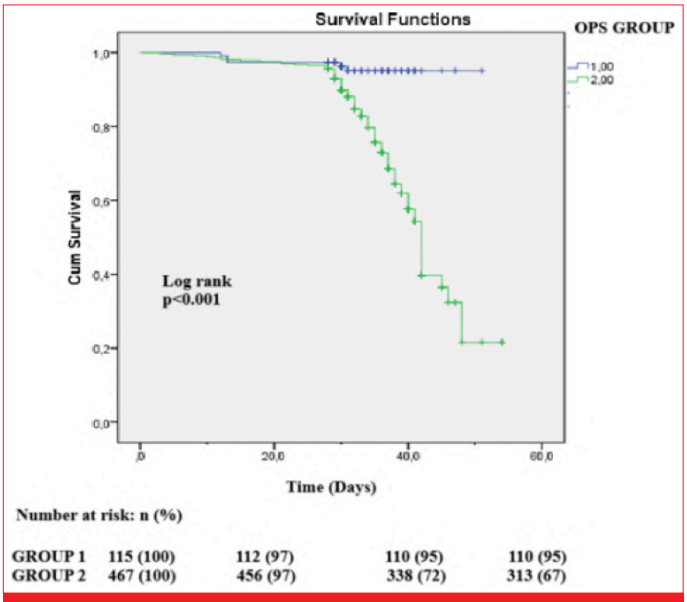

Figure 1. Kaplan–Meier analysis of mortality stratified by OPS cut-off.

Table 1. Comparison between patients with and without in-hospital mortality

Variables	Survivor, n=423	Non-Survivor, n=159	P Value
Age,years	57.39 ± 16.16	57.30 ± 14.12	0.953
Gender (male), n (%)	268 (%63.4)	94 (%59.1)	0.348
DM, n (%)	165 (^%39)	85 (%53.5)	0.002
HT, n (%)	232 (%54.8)	103 (%64.8)	0.031
Smoking, n (%)	101 (%23.9)	36 (%22.6)	0.754
COPD, n (%)	39 (%9.2)	13 (%8.2)	0.694
CRF, n (%)	115 (%27.2)	62 (%39)	0.006
Pacemaker history, n (%)	34 (%8.1)	7 (%4.4)	0.125
ICD history, n (%)	27 (%6.4)	8 (%5.1)	0.548
CRT history, n (%)	15 (%3.6)	2 (%1.3)	0.141
Cancer history, n (%)	28 (%6.6)	20 (%12.6)	0.020
Stroke history, n (%)	62 (%14.7)	34 (%21.5)	0.048
PAH, n (%)	18 (%4.3)	16 (%10.1)	0.008
PCI history, n (%)	83 (%19.7)	55 (%34.6)	<0.001
İE history, n (%)	38 (%9)	13 (%8.2)	0.759
Valve surgery history, n (%)	99 (%23.4)	38 (%23.9)	0.900
Culture positive, n (%)	311 (%73.5)	140 (%88.1)	<0.001
Blood culture, n (%)	Negative, 109 (%25.8) Coag.Neg.Staph, 68 (%16.1) Staph. Aureus, 148 (%35) Streptococcus, 44 (%10.4) Ent. Feacalis, 20 (%4.7) Gram negative, 23 (%5.4) Brucella, 2 (%0.5) Candida, 9 (%2.1)	Negative, 19 (%11.9) Coag.Neg.Staph, 26(%16.4) Staph. Aureus, 62 (%39) Streptococcus, 13 (%8.2) Ent. Feacalis, 13 (%8.2) Gram negative, 22 (%13.8) Brucella, 1(%0.6) Candida, 3 (%1.9)	0.001
>10 cm vegetation,n(%)	194 (%46.1)	118 (%74.2)	<0.001
Native valve İE,n (%)	263 (%62.2)	110 (%69.2)	0.116
Mechanic valve İE, n (%)	114 (%27)	38 (%23.9)	0.455
Lead related İE, n (%)	33 (%8.2)	9 (%5.7)	0.374
Catheter related İE, n (%)	14 (%3.3)	2 (%1.3)	0.177
LV EF, %	52.70 ± 12.18	51.85 ± 11.24	0.446
SPAP, mmHg	33 (10-94)	40 (15-86)	<0.001
GFR	68 (3-150)	43 (5-124)	<0.001
WBC, $10^3/mm^3$	8.71 (0.45-41.30)	10.6 (3.80-74.0)	<0.001
HGB, g/dL	10.47 ± 2.24	9.69 ± 2.10	<0.001
HCT, %	32.35 ± 6.74	29.52 ± 6.53	<0.001
Platelets, $10^3/mm^3$	228 (8-2360)	194 (27-617)	<0.001
Neutrophils, $10^3/mm^3$	6.20 (0.20-92.20)	8.69 (0.06-95.60)	<0.001
Lymphocytes, $10^3/mm^3$	1.25 (0.04-27.60)	1.50 (0.30-67.0)	0.282
AST, U/L	23 (3-1028)	29 (3.35-5266)	<0.001
ALT, U/L	22 (2-1290)	36 (3-3143)	0.001
CRP, mg/L	75.30 (0.40-468.20)	124 (1.10-423)	<0.001
Sodium, mmol/L	136.58 ± 4.26	135.33± 12.43	0.216
Potassium	4.29 ± 0.62	4.36 ± 0.73	0.256
Albumin, g/dL	33.53 ± 6.78	30.20 ± 6.22	<0.001
Glucose, mg/dL	108 (63-358)	127 (19-460)	0.001
HbA1C	6.15 ± 1.53	6.48 ± 1.74	0.103
BUN, mg/dL	40 (1.98-330)	67 (8-385)	<0.001
Creatinine, mg/dL	1.04 (0.36-13.70)	1.35 (0.45-9.89)	<0.001
Triglycerides, mg/dL	137 (56-734)	145 (52-562)	0.990
HDL, mg/dL	33 (3-210)	30 (8-71)	0.086
LDL, mg/dL	86 (6-206)	90 (40-164)	0.073
CALLY İNDEX	0.05 (0.000-15.36)	0.03 (0.002-3.82)	<0.001
OPS	2.07 ± 0.89	2.67 ± 0.55	<0.001
HALP	19.22 (0.92-967.72)	20.62 (2.12-2010)	0.355
PNI	40.30 (22.80-177.20)	36.40 (22-356)	<0.001
Hemodialysis, n (%)	71 (%16.8)	67 (%42.1)	<0.001
İn-hospital stroke, n (%)	37 (%8.8)	37 (%23.3)	<0.001
Periferic emboli, n (%)	21 (%5.0)	19 (%11.9)	0.003
Major Bleeding, n (%)	22 (%5.2)	15 (%9.4)	0.062
İ.V. İnotrop, n (%)	68 (%16.1)	138 (%86.8)	<0.001
Pacemaker Requirement, n (%)	24 (%5.7)	7 (%4.4)	0.535
İntubation, n (%)	5 (%1.2)	151 (%95)	<0.001
İE surgery, n (%)	166 (%39.2)	64 (%40.3)	0.825
İn Hospital follow up, Days	35.28 ± 4.81	32.39 ± 8.40	<0.001

Abbreviations: DM, Diabetes Mellitus; HT, Hypertension; COPD, Chronic Obstructive Pulmonary Disease; CRF, Chronic Renal Failure; PCI, Percutane Coronary Intervention; GFR, Glomerular Filtration Rate; LV EF, Left Ventricle Ejection Fraction; SPAP, Systolic Pulmonary Artery Pressure; IE, Infective endocarditis; WBC, White Blood Cell; HGB, Hemoglobin; HCT, Hematocrit; MCV, Mean Corpuscular Volume; CRP, C-Reactive Protein; BUN, Blood Urea Nitrogen; AST, Aspartate Aminotransferase; ALT, Alanine Aminotransferase; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; OPS, Osaka Prognostic Score; CALLY INDEX, C-reactive protein–Albumin–Lymphocyte index; HALP, Hemoglobin, Albumin, Lymphocyte, and Platelet score; PNI, Prognostic Nutritional Index

Table 2. ROC curve analysis for predicting in-hospital mortality

Variables	AUC	P Value	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
SPAP	0.614	<0.001	0.553	0.714
WBC	0.654	<0.001	0.601	0.708
AST	0.626	<0.001	0.565	0.687
Platelets	0.632	<0.001	0.574	0.690
OPS	0.689	<0.001	0.644	0.734

Abbreviations: AUC, Area under the Curve; SPAP, Systolic Pulmonary Artery Pressure; WBC, White Blood Cell; AST, Aspartate Aminotransferase; OPS, Osaka Prognostic Score

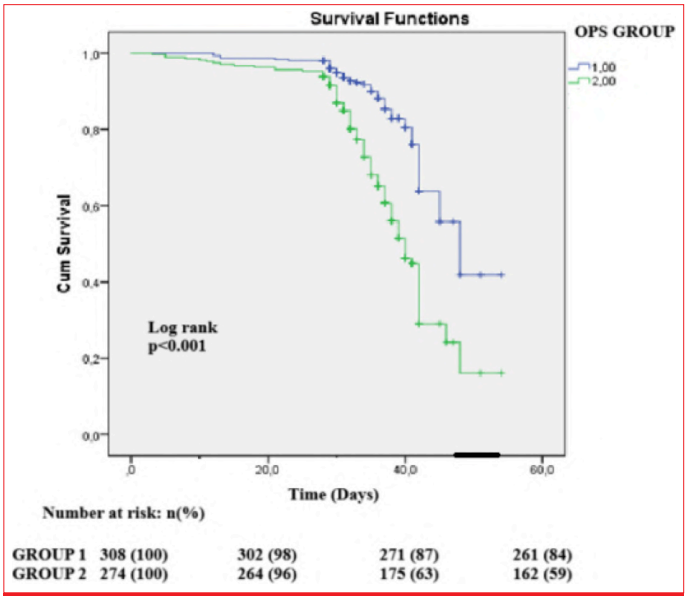

Figure 2. Kaplan–Meier survival curves for mortality according to OPS group.

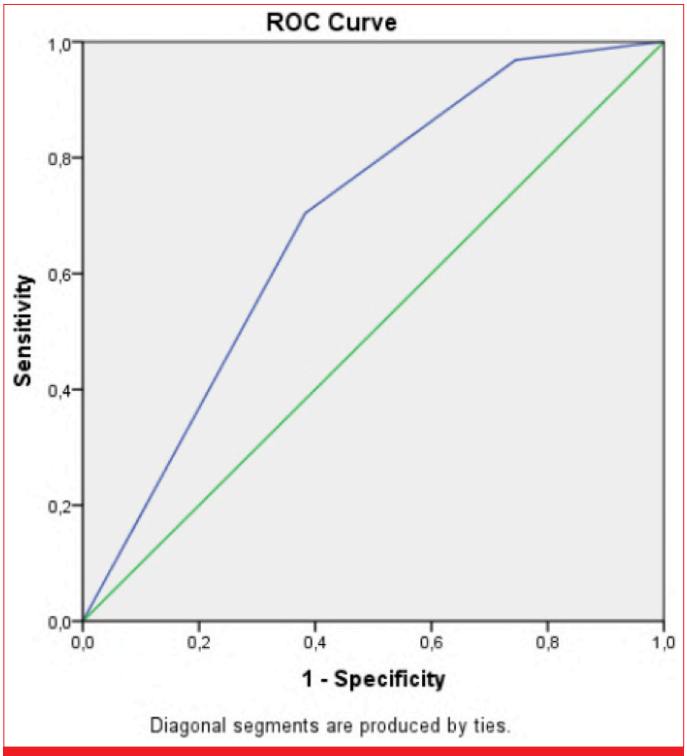

Figure 3. ROC curve analysis for predicting in-hospital mortality.

OP-011 [Other]

Development and validation of first Turkish aAI-based device to guide optimal echocardiographic probe positioning

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Background and Aim: The importance of artificial intelligence (AI) in cardiac imaging is increasing day by day, and it is beginning to play an active role in clinical practice. Our primary goal was to develop an AI-based application that guides the user to the correct probe position during transthoracic echocardiography and assists in the training of cardiology residents and medical students.

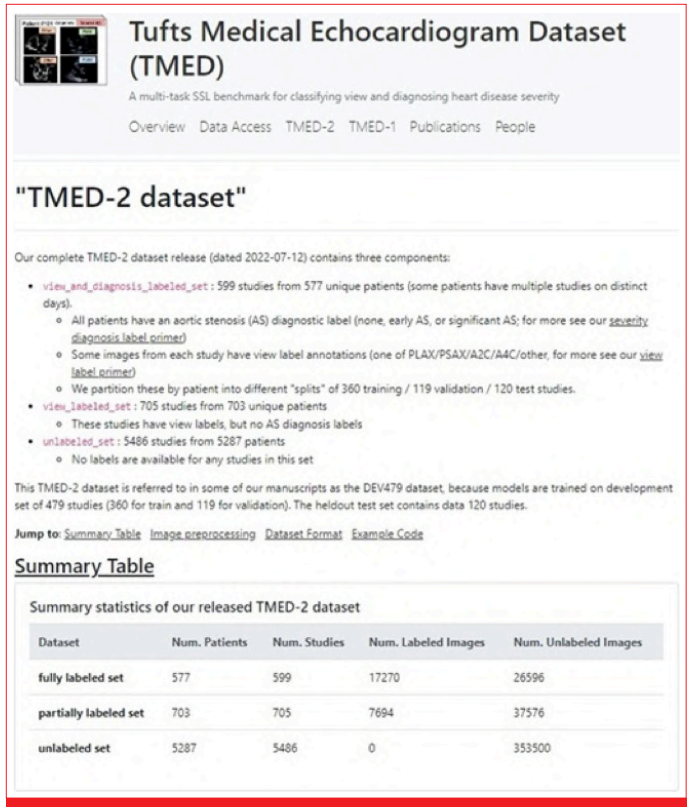

Figure 1. TMED-2 dataset, published by Tufts University in July 2022.

Figure 2. InceptionV3 Model The link to the model architecture study: https://arxiv.org/pdf/1512.00567.pdf

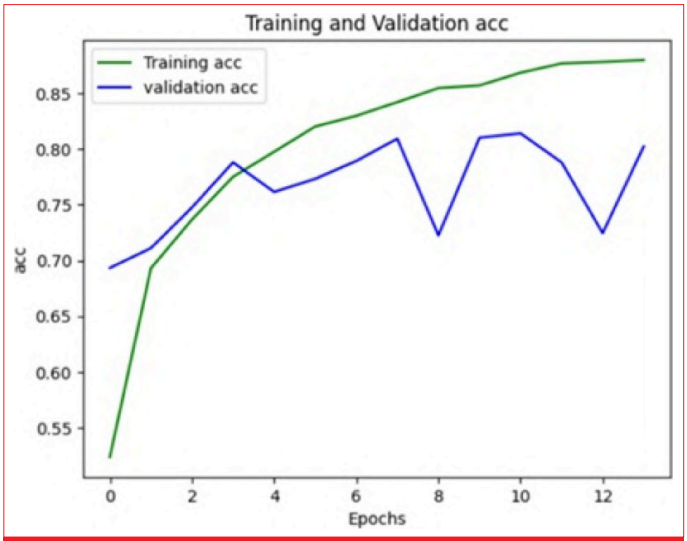

Figure 3. a) The most challenging factors encountered during the development of the artificial intelligence model were the limited number of available usable data, the low quality of even the usable data, and the significant class imbalance in the target views. b) During the model development process, the outcome of each experiment was analyzed, and theories for model optimization were developed based on the results and parallel research. These theories were then applied in subsequent experiments. As a result of the conducted research and experiments, an AI model was developed that can predict PLAX, PSAX, A2C, A3C, A4C, and A5C views with an accuracy rate of over 85% and can process an average of 12 images per second.

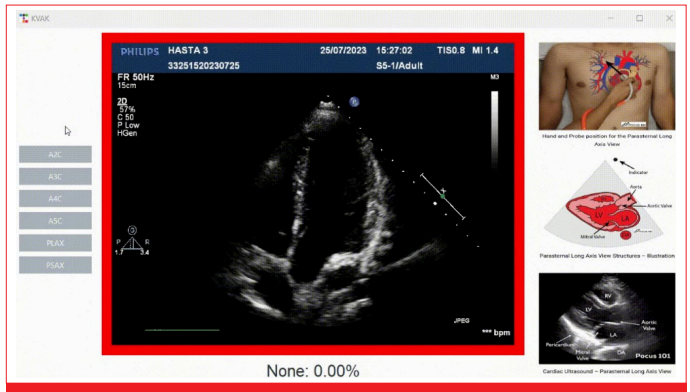

Figure 4. A5C measurement to improve visualization and user-friendliness, we developed a dedicated application for our model. In this application, the user can select the desired echocardiographic view using the buttons on the left panel. On the right side, we included reference images to assist students while reviewing the selected view. The central area displays the real-time echocardiographic image. A guiding frame appears around the image: green if the correct view is approached, and red if the current view is far from the target, helping guide the user toward the correct position. At the bottom of the interface, the model's prediction and its confidence percentage are displayed. The video format of the file could not be loaded. A measurement video is available. It can be sent upon request.

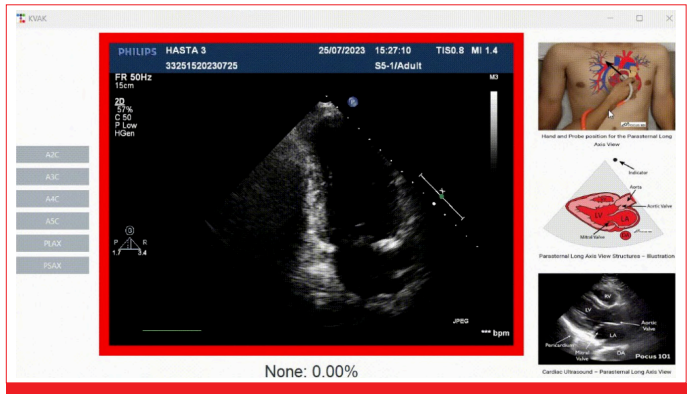

Figure 5. A2C measurement to improve visualization and user-friendliness, we developed a dedicated application for our model. In this application, the user can select the desired echocardiographic view using the buttons on the left panel. On the right side, we included reference images to assist students while reviewing the selected view. The central area displays the real-time echocardiographic image. A guiding frame appears around the image: green if the correct view is approached, and red if the current view is far from the target, helping guide the user toward the correct position. At the bottom of the interface, the model's prediction and its confidence percentage are displayed. The video format of the file could not be loaded. A measurement video is available. It can be sent upon request.

Methods: In the first stage, we aimed to train the AI model using both standard and incorrect echocardiographic images. Subsequently, we obtained access to the TMED-2 dataset, published by Tufts University in July 2022 (Figure 1), by submitting a request. This dataset included 353,500 unlabelled images from 5,287 patients. To label which standard echocardiographic view each image corresponded to, we formed a team of 10 cardiologists. One of the key features considered in the labeling system was the ability for approximately 10 physicians to simultaneously and efficiently label images. For this purpose, a Heroku-based web server was rented (Figures 2 and 3). Due to their clinical importance and frequent use, we selected the following views as labels: PLAX, PSAX, A2C, A3C, A4C, and A5C. Images that did not fit these categories or were deemed poor quality were classified as unusable. A total of 44,840 images were reviewed, and the model was trained on 11,515 suitable images. The AI application was developed using the InceptionV3 architecture as its backbone (Figure 4). As a result of this process, we developed an AI model capable of predicting PLAX, PSAX, A2C, A3C, A4C, and A5C

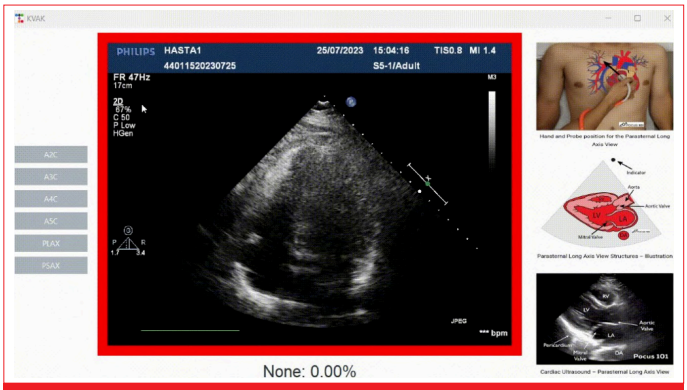

Figure 6. A4C measurement to improve visualization and user-friendliness, we developed a dedicated application for our model. In this application, the user can select the desired echocardiographic view using the buttons on the left panel. On the right side, we included reference images to assist students while reviewing the selected view. The central area displays the real-time echocardiographic image. A guiding frame appears around the image: green if the correct view is approached, and red if the current view is far from the target, helping guide the user toward the correct position. At the bottom of the interface, the model's prediction and its confidence percentage are displayed. The video format of the file could not be loaded. A measurement video is available. It can be sent upon request.

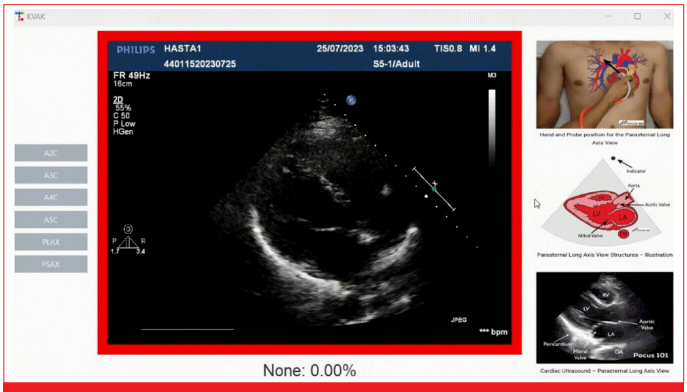

Figure 7. PLAX measurement to improve visualization and user-friendliness, we developed a dedicated application for our model. In this application, the user can select the desired echocardiographic view using the buttons on the left panel. On the right side, we included reference images to assist students while reviewing the selected view. The central area displays the real-time echocardiographic image. A guiding frame appears around the image: green if the correct view is approached, and red if the current view is far from the target, helping guide the user toward the correct position. At the bottom of the interface, the model's prediction and its confidence percentage are displayed. The video format of the file could not be loaded. A measurement video is available. It can be sent upon request.

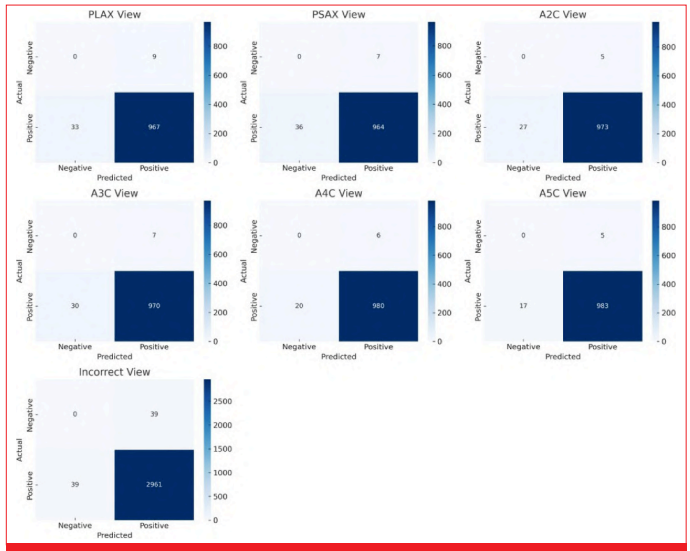

Figure 8. Confusion matrix.

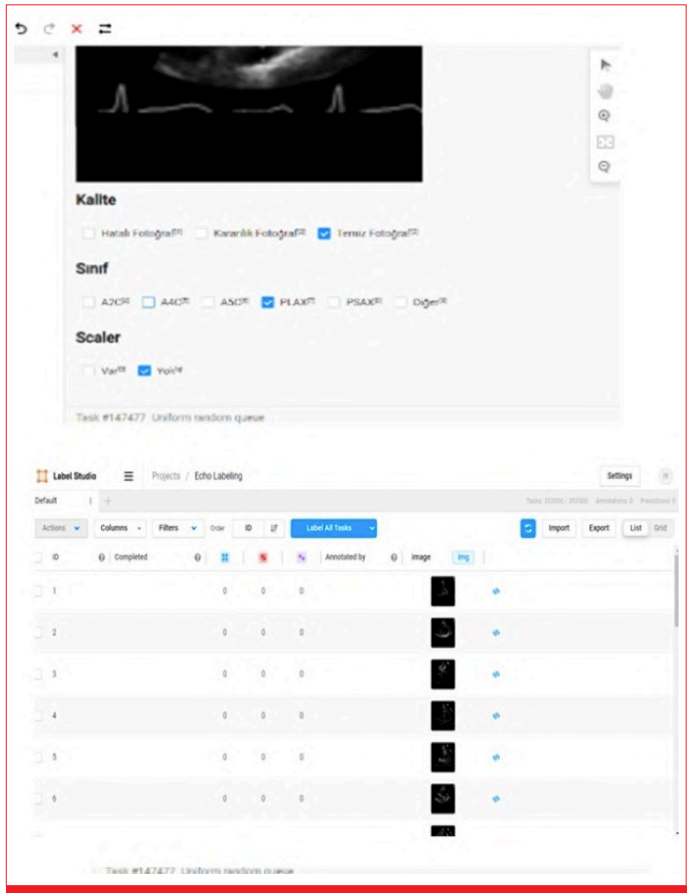

Figure 9. Labeling interface.

views with over 85% accuracy, processing an average of 12 images per second (Figure 5). Following model training, we developed the user interface of the application. The interface visually indicates whether the current probe position corresponds to the selected echocardiographic view (Figures 6, 7, 8, and 9). Next, we collected echocardiographic images of PLAX, PSAX, A2C, A3C, A4C, and A5C

views from 1,000 patients in the cardiology outpatient clinic, along with 3,000 additional incorrectly positioned images. These images were stored in DICOM format (Philips EPIQ). The validation process was carried out by five independent cardiologists who were blinded to the original measurements. The device was considered accurate if it output a prediction with greater than 99% confidence when the image was truly in the correct position.

Results: The AI model achieved the following correct prediction rates: PLAX: 96.7%; PSAX: 96.4%; A2C: 97.3%; A3C: 97.0%; A4C: 98.0%; A5C: 98.3%. Among the 3,000 incorrectly positioned images, the model correctly identified 98.71% as incorrect views.

Conclusions: The software we developed represents the first Turkish AI software to initiate echocardiographic view classification and segmentation. To our knowledge, this is also the first study from Turkey in this field. Further development of the application is ongoing, including integration of ejection fraction estimation and advanced myocardial segmentation features.

Table 1. Baseline clinical and echocardiographic characteristics of the patients

Parameters	(n=1000)
Age, years	56.5 ± 10.6
Female, n (%)	49.5 (49.5%)
Body Mass Index, kg/m²	26.3 ± 3.5
Hypertension, n (%)	251 (25.1%)
Congestive Heart Failure, n (%)	65 (6.5%)
Diabetes Mellitus, n (%)	145 (14.5 %)
Hyperlipidemia, n (%)	156 (15.6%)
Hypertrophic Cardiomyopathy, n (%)	11 (1.1%)
Ejection Fraction (%)	55.5 ± 10.2
Left Atrium Diameter, mm	33.5 ± 3.5
Left Ventricular Diastolic Diameter, mm	41.5 ± 2.07
Left Ventricular Systolic Diameter, mm	28 ± 1.12
Intraventricular Septumthickness, mm	10.2 ± 1.12

Table 2. Performance metrics of the AI model for echocardiographic view classification

View	True Positives (TP)	False Negatives (FN)	False Positives (FP)	Accuracy	Precision	Recall (Sensitivity)	F1 Score
PLAX	967	33	9	0.958	0.991	0.967	0.979
PSAX	964	36	7	0.957	0.993	0.964	0.978
A2C	973	27	5	0.968	0.995	0.973	0.984
A3C	970	30	7	0.963	0.993	0.980	0.981
A4C	980	20	6	0.974	0.994	0.983	0.987
A5C	983	17	5	0.978	0.995	0.983	0.989
Incorrect	2961	39	39	0.974	0.987	0.987	0.987

Accuracy: Proportion of correct predictions (TP / (TP + FN + FP)). Precision: How many predicted positives were truly positive (TP / (TP + FP)). Recall (Sensitivity): Proportion of actual positives correctly identified (TP / (TP + FN)). F1 Score: Harmonic mean of precision and recall; reflects the balance between the two. The AI model demonstrated high performance across all standard transthoracic echocardiographic views, achieving accuracy above 95%, precision greater than 98%, and recall levels between 96–98%. The F1 scores, ranging from 0.978 to 0.989, indicate excellent balance between precision and sensitivity. The confusion matrix further supports the robustness of the model for clinical application.

OP-012 [Other]

Contrast nephropathy preventive effects and molecular biological mechanism of thymoquinone zein nanoparticles given after exposure to iohexol carbon nanodots and alternative contrast nanoparticles in podocyte cells

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Background and Aim: One of the most common causes of hospital-acquired acute kidney injury is contrast-induced nephropathy (CIN), which develops following the administration of contrast agents for diagnostic or therapeutic purposes.This clinical condition not only prolongs hospitalization and increases healthcare costs but is also associated with significant long-term morbidity and mortality. In this study, the prevention of potential contrast-induced nephropathy in podocyte cells, exposed to contrast agents and alternative contrast

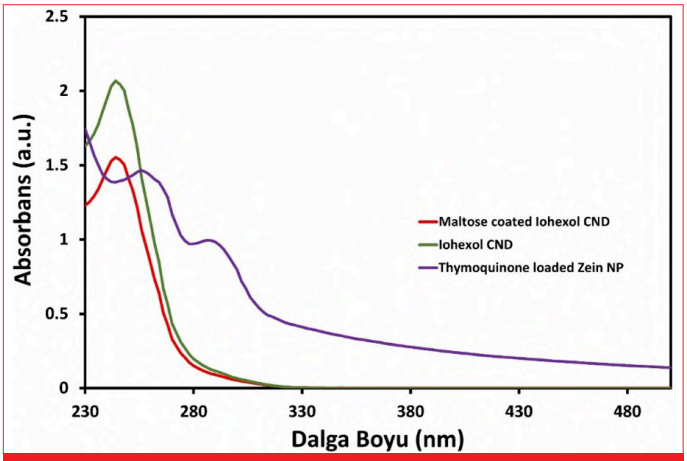

Figure 1. UV-vis spectrum results.

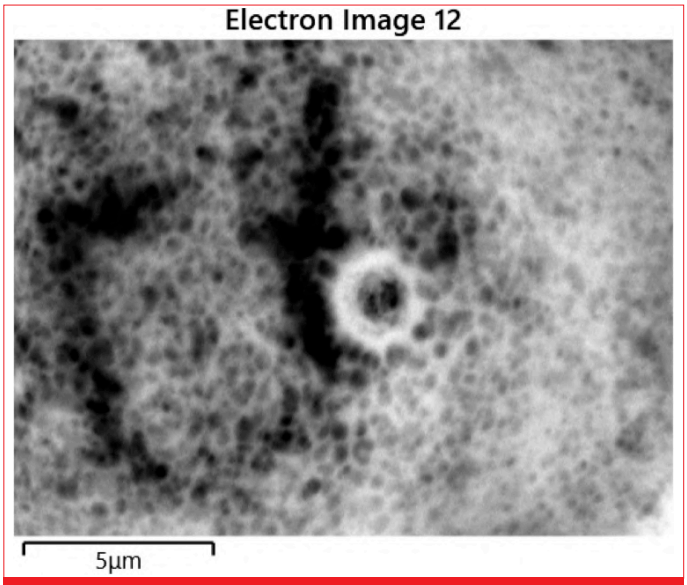

Figure 2. SEM image of Iohexol carbon nanodot.

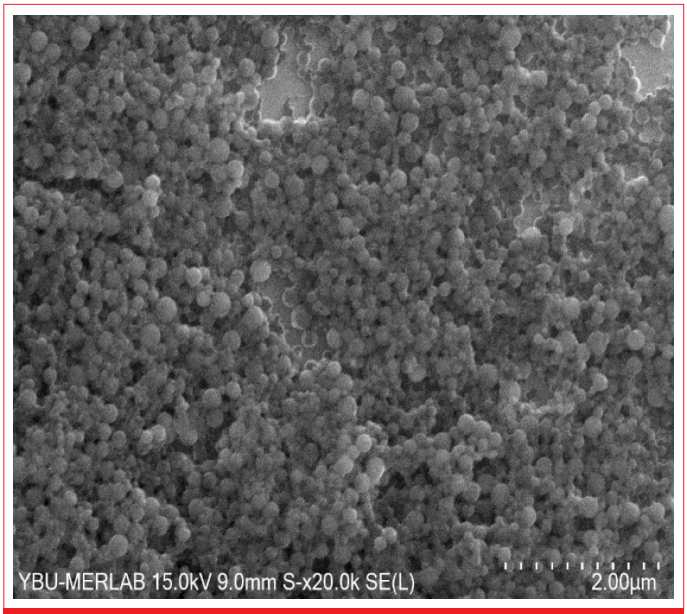

Figure 3. SEM image of thymoquinone-ioaded zein nanoparticle.

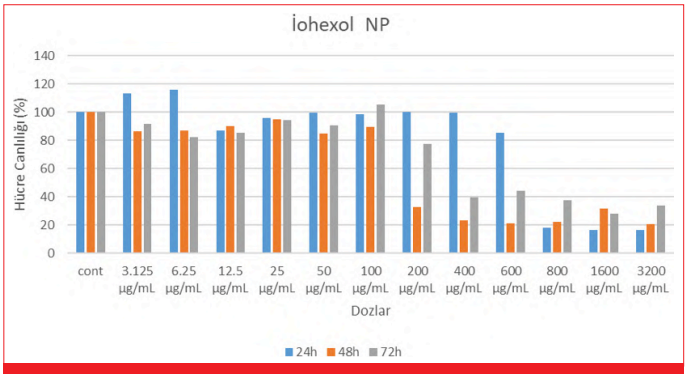

Figure 4. Dose and time dependent effects of Iohexol nanoparticles on cell viability.

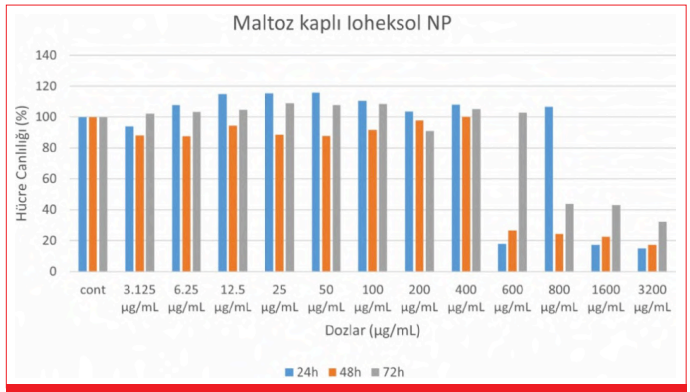

Figure 5. Dose and time dependent effects of maltose coated iohexol nanoparticles on cell viability.

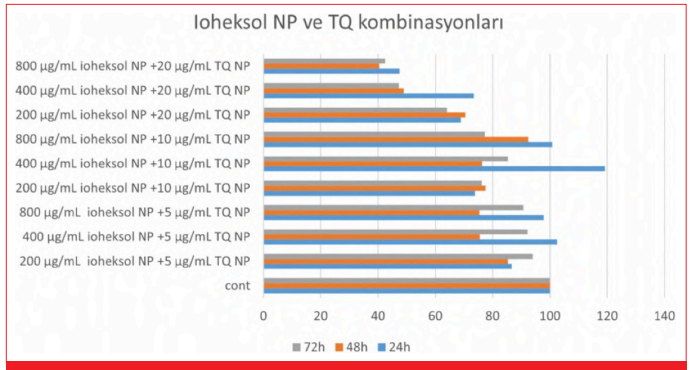

Figure 6. Dose and time dependent effects on cell viability after combination with TQ (Iohexol nanoparticle).

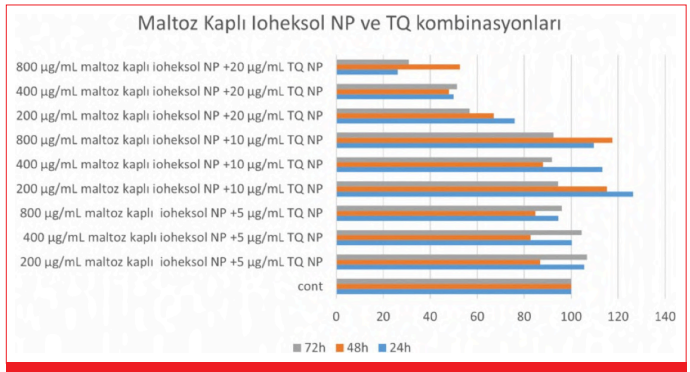

Figure 7. Dose and time dependent effects on cell viability after combination with TQ (maltose-coated Iohexol nanoparticle).

nanomaterials in a cell culture environment, was investigated using thymoquinone metal–organic framework nanoparticles.

Methods: Initially, carbon nanodots (CNDs) and maltose-coated Iohexol-CND contrast agents were synthesized.Thymoquinone-loaded zein nanoparticles (TQ-ZNPs) were prepared using the anti-solvent precipitation method. Podocyte cells were cultured in vitro, and dose concentrations were determined based on the literature. Cytotoxicity assays were conducted using the XTT method in a dose- and time-dependent manner at 24, 48, and 72 hours.In subsequent steps, total RNA was isolated using the TRIzol method, followed by cDNA synthesis. Gene expression changes related to oxidative

stress and genotoxicity BAX, BCL2, NRF2, NF-κB, SOD2, Catalase, H2AX, and GPX1 were analyzed at the mRNA level by real-time PCR. Relevant statistical analyses were performed.

Results: The synthesis and characterization of TQ-ZNPs were successfully performed using techniques such as UV–visible spectroscopy, scanning electron microscopy (SEM), and particle size analysis, demonstrating that they are suitable candidates for targeted drug delivery applications (Figures 1–3). The cytotoxic effects of Iohexol nanoparticles and maltose-coated Iohexol nanoparticles at concentrations ranging from 3.125 µg/mL to 3200 µg/mL were evaluated using the XTT method at 24, 48, and 72 hours (Figures 4, 5). In groups co-treated with TQ, a dose- and time-dependent reduction in cytotoxicity was observed (Figures 6, 7). TQ treatment was shown to attenuate cellular damage by modulating the expression of genes associated with oxidative stress and genotoxicity. Specifically, the upregulation of NF-κB, SOD2, Catalase, and GPX1 indicated antioxidant and protective activity ($p<0.05$).

Conclusions: This study is significant because it is the first to demonstrate, in vitro, the effects of the newly synthesized and original TQ-ZNPs compound on contrast-induced nephropathy using podocyte cells. In conclusion, the use of TQ-containing nanodots was shown to reduce Iohexol-induced toxicity in podocyte cells at the in vitro level. Furthermore, the regulation of gene expression associated with oxidative stress, genotoxicity, and cell survival suggests that multiple intracellular signaling pathways may have been activated, thereby exerting a potential mechanistic effect. The findings obtained from this study are expected to guide future, more comprehensive studies and contribute to the current scientific literature.

OP-013 [Other]

Myocardial remodeling under strenuous exercise in transgenic mice with familial hypertrophic cardiomyopathy

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Background and Aim: Familial hypertrophic cardiomyopathy (HCM) is an autosomal dominant genetic disease, which is presented with ventricular hypertrophy, myofibrillar disarray, and interstitial fibrosis. D166V is one of the myosin ventricular regulatory light chain 2 mutations which are associated with malignant outcomes such as heart failure or sudden cardiac death. Hypertrophy as determined by the heart weight to body weight ratio has not been found before even in the old animals with D166V mutation. The aim was to induce left ventricular hypertrophy (LVH) development with strenuous exercise, to evaluate the effect of stress on LV remodeling, to determine of global LVH and LVH type in Tg-HCM and normal mice and to evaluate myocardial function using tissue Doppler imaging (TDI).

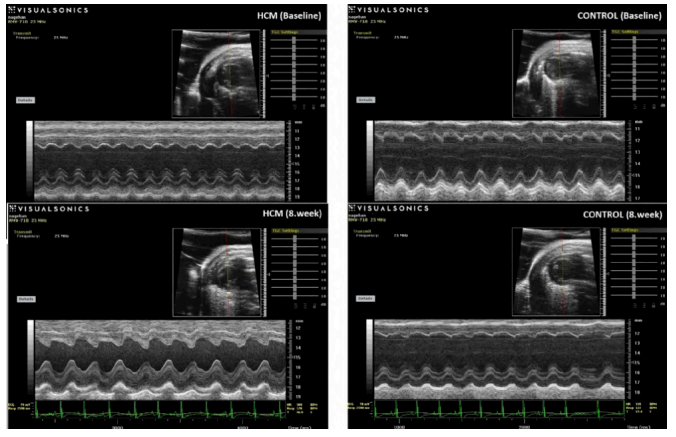

Figure 1. M-mode echocardiographic images of HCM vs NTg mice at baseline and after 8 weeks of exercise.

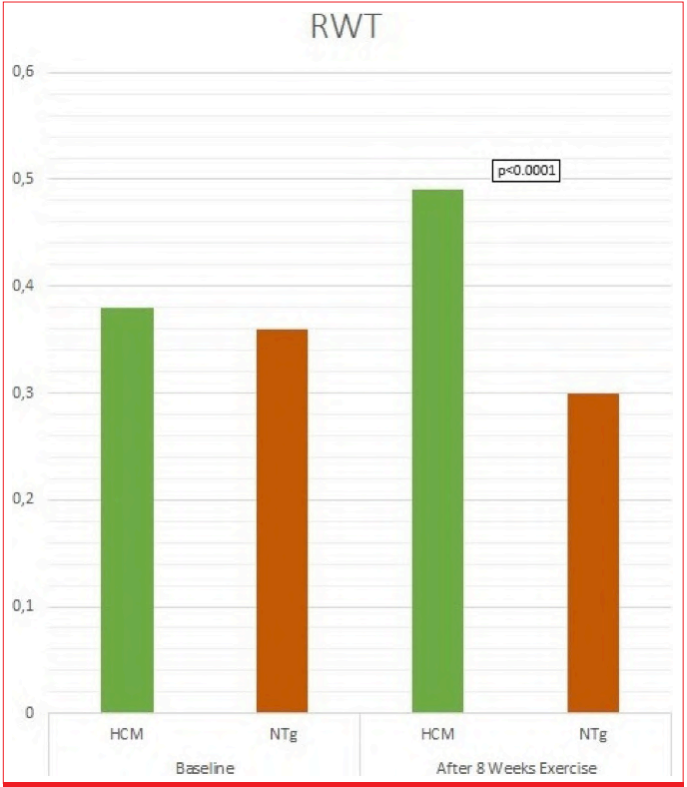

Figure 2. Comparison of relative wall thickness measurements of HCM vs NTg mice at baseline and after 8 weeks of exercise.

Methods: 7 D166V mutant male mice and 10 weight matched control (non-transgenic: NTg) mice forced to treadmill exercise (5 days a week, one hour a day, 20 m/min) and underwent B-mode echocardiography and TDI at baseline and after 8 weeks. Animals were euthanized 24 hours after their last exercise, then their hearts were removed, the left ventricles of each heart were dissected and weighed individually, tibial lengths were measured.

Results: The findings of echocardiographic examination were presented in Table 1. LVEF (55 ± 6.6 mm vs. 58 ± 6.8 mm, ns) was not statistically different between two groups after exercise period. Relative wall thickness (RWT) was same at baseline (0.38 ± 0.03 vs. 0.36 ± 0.04, ns)

Figure 3. Tissue doppler imaging echocardiographic images of HCM vs NTg mice after 8 weeks of exercise.

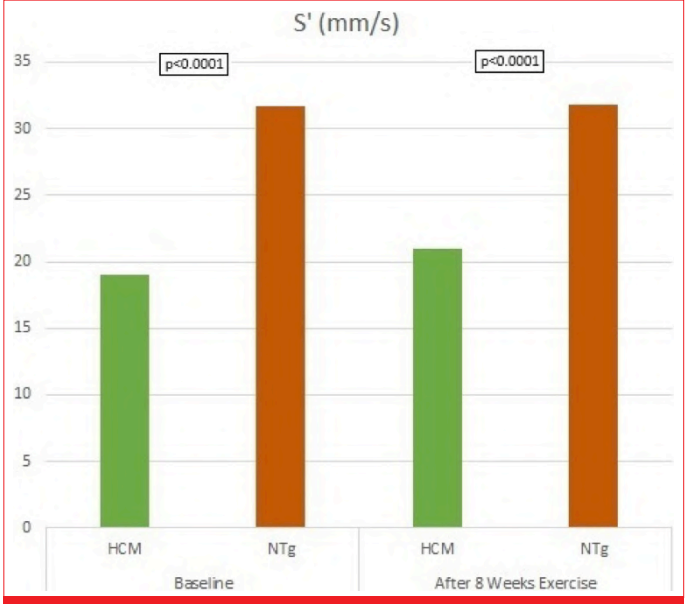

Figure 4. Comparison of septal wall S' by tissue Doppler imaging of HCM vs NTg mice at baseline and after 8 weeks of exercise.

Table 1. Echocardiographic findings of the groups at baseline and after 8 weeks

	Baseline			After 8 Weeks Exercise		
	HCM	NTg	p	HCM	NTg	p
HR (bpm)	445±35	416±42	ns	413±75	432±42	ns
LVAW (mm)	0.73±0.06	0.74±0.06	ns	0.93±0.03	0.78±0.08	<0.0001
LVPW (mm)	0.77±0.02	0.72±0.06	0.044	1.0±0.7	0.74±0.7	<0.0001
IVS (mm)	0.72±0.05	0.71±0.04	ns	0.98±0.06	0.72±0.03	<0.0001
LVDD (mm)	4.09±0.3	3.96±0.3	ns	4.14±0.5	4.61±0.2	0.016
RWT	0.38±0.03	0.36±0.04	ns	0.49±0.04	0.3±0.02	<0.0001
LV Area (mm^2)	11.6±1.3	10.3±1.1	ns	11.5±0.8	12.5±1.7	0.02
LVEF (%)	63±6	59±5	ns	55±6.6	58±6.8	ns
E (mm/s)	980±40	1008±83	ns	968±42	715±85	0.006
A (mm/s)	603±49	607±42	ns	472±59	497±70	ns
E/A	1.63±0.2	1.69±0.2	ns	2.01±1.3	1.45±0.1	0.005
S' (mm/s)	19.0±4	31.7±7	<0.001	21±3.4	31.8±3.7	<0.0001
E' (mm/s)	18.8±6	41±10	<0.001	14.1±3.8	32.8±3.7	<0.0001
A' (mm/s)	18.8±5	30±6	0.001	19.4±4.3	28.9±5.3	0.001
E'/A'	0.85±0.4	1.36±0.2	0.005	0.76±0.3	1.05±0.2	0.03
E/E'	58.8±4	25.5±6	0.001	60.3±3	23.6±6	<0.0001

HR: Heart rate, LVAW: Left ventricular anterior Wall, LVPW: Left ventricular posterior wall, IVS:Interventricular septum, LVDD: Left ventricular diastolic dimension, RWT: Relative wall thickness, LV:Left ventricle, LVEF: Left ventricular Ejection Fraction, ns:non-significant

but significantly higher in HCM than NTg after exercise (0.49 ± 0.04 vs. 0.3 ± 0.02, p<0.0001) (Figure 2). E/A ratio became higher in HCM than in NTg after 8 weeks. Septal s' was decreased and E/E' was increased in mutants at baseline and after exercise.As shown in Table 2, HW/BW (6.1 ± 0.5 mg/g vs. 5.1 ± 0.1 mg/g, p=0.05) and LVW/BW (4.5 ± 0.4 mg/g vs. 3.9 ± 0.5 mg/g, p=0.04) was significantly higher in HCM group.

Table 2. Heart weight, left ventricular weight, body weight, tibial length measurements and the ratios of the groups

	HCM	NTg	p
Body Weight (g)	32.4±1.9	33.8±1.3	0.145
Tibial Length (mm)	17.5±0.7	17.5±0.2	0.968
Heart Weight (mg)	196±16	173±38	0.199
LV Weight (mg)	146±11	132±21	0.201
HW/BW (mg/g)	6.1±0.5	5.1±0.1	0.05*
HW/TL (mg/mm)	11.2±1.3	9.8±2.1	0.201
LVW/BW(mg/g)	4.5±0.4	3.9±0.5	0.04*
LVW/TL(mg/mm)	8.3±0.8	7.5±1.1	0.214

HW: Heart weight, LVW: Left ventricular weight, BW: Body weight, TL: Tibial length, *: statistically significant

Conclusions: In our study we detected concentric hypertrophy in those mutant mice after exercise period. Importantly although there was no LVH in those mutant mice, TDI parameters of septal base were different than non-transgenic mice before exercise. In a study in which septal corner tissue velocities were used to predict cardiovascular events in asymptomatic or mildly symptomatic patients with HCM, a decrease was observed in all myocardial velocities obtained from the septal corner of patients experiencing cardiovascular events, while an increase in the septal E/E' value was observed. This study demonstrated that forced exercise further induces LVH in the heart with D166V mutation. TDI may be beneficial for early detecting functional impairment of myocardium in familial HCM before hypertrophy development. Therefore, it can be recommended that individuals with a family history of HCM be evaluated with TDI even if they have normal echocardiographic findings. Additionally, restricting strenuous exercise in individuals with a family history may be an important warning to reduce the development of hypertrophy.

OP-014 [Cardiac Imaging / Echocardiography]

Not all that swells is heart failure: A hidden paraneoplastic culprit

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Background and Aim: Heart failure with preserved ejection fraction (HFpEF) presents significant diagnostic challenges due to its nonspecific clinical presentation and symptom overlap with various extracardiac pathologies. Misdiagnosis may result in delays or misdirection in management. This case aims to emphasize the importance of considering alternative, non-cardiac etiologies in patients with preserved LVEF and atypical features. Specifically, we present a rare paraneoplastic syndrome secondary to a gastrointestinal stromal tumor (GIST) mimicking HFpEF.

Methods: A 66-year-old male with a history of hypertension and type 2 diabetes mellitus, and a prior normal coronary angiogram performed ten years earlier, was admitted with complaints of progressive dyspnea, bilateral lower extremity edema, and abdominal distension. His body mass index was consistent with morbid obesity (170 cm, 160 kg, BSA: 2.57 m^2). Transthoracic echocardiography (TTE) revealed preserved left ventricular ejection fraction (LVEF: 55%) and no significant valvular pathology. ECG demonstrated sinus rhythm, and laboratory parameters were unremarkable. Based on the initial clinical assessment, the patient was diagnosed with HFpEF and diuretic therapy was initiated. Due to insufficient clinical response, further evaluation including NT-proBNP measurement and systemic examination was performed.

Results: The patient's symptoms persisted despite optimized diuretic therapy. NT-proBNP level was measured at 70 pg/mL, which was not consistent with a diagnosis of HFpEF. Additionally, inferior vena cava diameter and respiratory variability were within normal limits. Dermatologic examination revealed nodular, erythematous skin lesions on the lower extremities and abdomen, which were initially attributed to stasis dermatitis. Dermatology consultation suggested a paraneoplastic etiology. Abdominal computed tomography revealed a hypodense, lobulated mesenteric mass, consistent with a gastrointestinal stromal tumor (GIST). Peripheral edema was attributed to impaired lymphatic drainage due to both the tumor's mass effect and the patient's obesity. The patient was subsequently referred to gastrointestinal surgery.

Conclusions: In morbidly obese patients with volume overload and preserved LVEF, HFpEF is often considered the primary diagnosis. However, clinicians should maintain a high index of suspicion for alternative etiologies in the presence of atypical findings such as low natriuretic peptide levels and unexplained dermatologic manifestations. This case illustrates the critical role of integrating echocardiographic findings with systemic evaluation and highlights the diagnostic value of a multidisciplinary, multimodality approach in differentiating true cardiac pathology from extracardiac mimickers such as paraneoplastic syndromes. Early recognition of such entities is essential for appropriate management and improved outcomes.

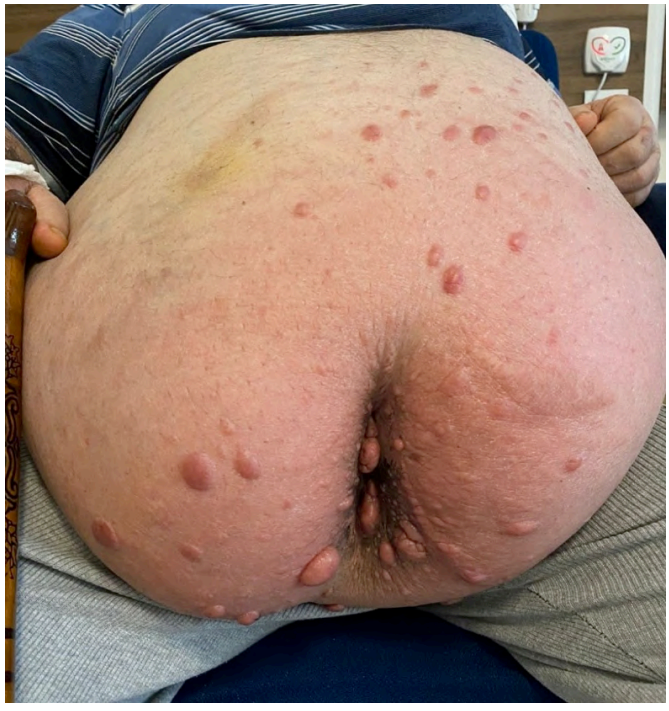


Figure 1. The morbidly obese patient presented with significant abdominal distension and nodular erythematous lesions on the anterior abdomen.

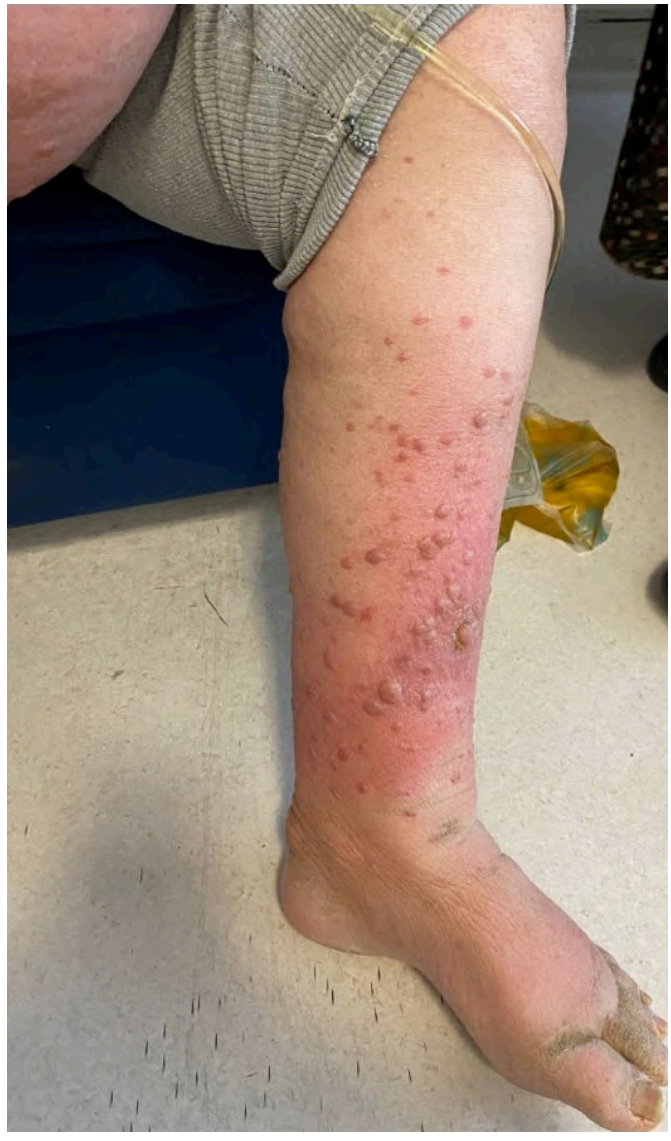


Figure 2. The patient presented with bilateral lower extremity edema accompanied by erythematous skin lesions on the legs.

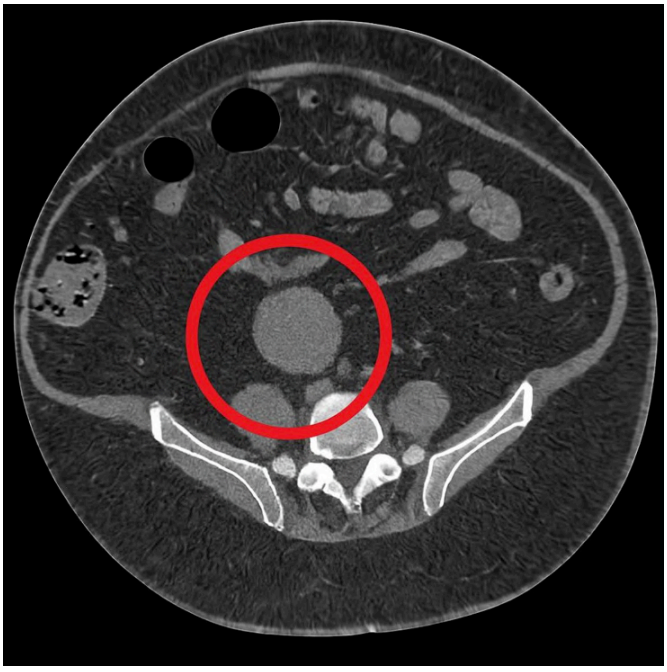
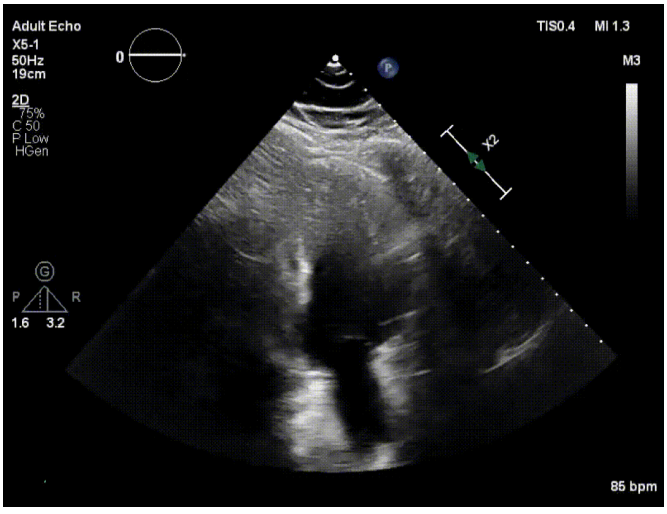
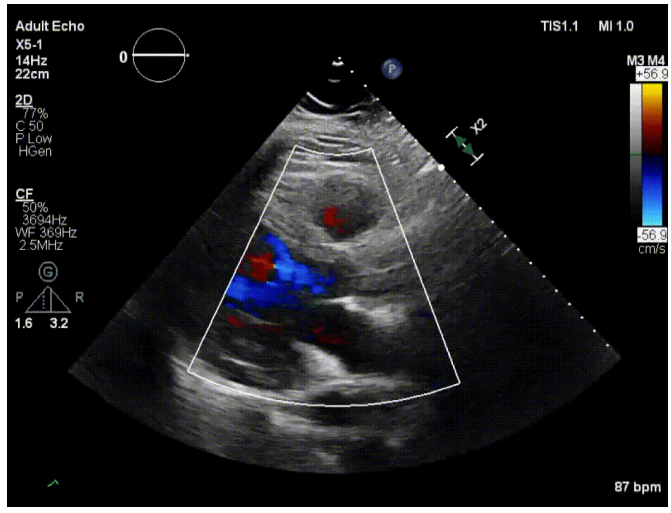


Figure 3. A hypodense, mildly lobulated lesion measuring approximately 43x37 mm, extending into the mesenteric fat tissue, suggestive of a GIST.



Video 1. Transthoracic echocardiography was technically limited due to the patient's habitus; however, image quality was sufficient to demonstrate preserved left ventricular systolic function. Color Doppler evaluation revealed no evidence of significant valvular pathology.



Video 2. Transthoracic echocardiography was technically limited due to the patient's habitus; however, image quality was sufficient to demonstrate preserved left ventricular systolic function. Color Doppler evaluation revealed no evidence of significant valvular pathology.

OP-015 [Cardiac Imaging / Echocardiography]

Evaluation of predictive factors for mitral regurgitation improvement after transcatheter aortic valve implantation (TAVI)

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Background and Aim: Mitral regurgitation (MR) is frequently encountered in patients undergoing transcatheter aortic valve implantation (TAVI). Identifying the changes in MR severity following the procedure and the predictive factors of such changes is of clinical significance for patient management. This study retrospectively evaluated clinical and echocardiographic parameters predicting MR improvement after TAVI.

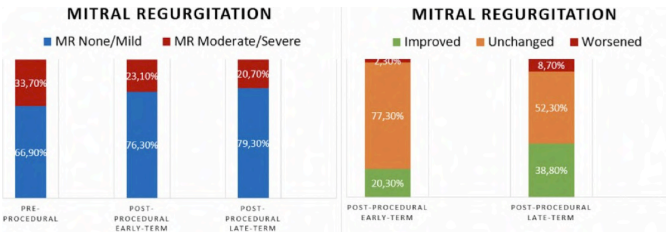

Figure 1. Change in mitral regurgitation.

Table 1. Baseline charecteristics

Parameters	Total N:183	MR None/Mild N:120 66,3%	MR Moderate/ Severe N:61 33,7%	P value
Age (years) Mean ±ss	77,6±6	77±6,7	78,8±6,4	*0,08*
Gender/ M, n(%)	81 (44,3%)	47 (39,2%)	32 (52,5%)	*0,11*
BMI kg/m^2 Ortalama ±ss	27,8±5,8	28,9±5,9	27,4±5,7	*0,87*
BSA m^2 Mean ±ss	1,8±0,1	1,8±0,1	1,8±0,2	*0,16*
STS Score Ortalama ±ss	5,3±3	4,5±2,8	6,9±3,4	***0,001***
NYHA 1-2, n(%)	92 (50,3%)	70 (58,3%)	21 (34,4%)	***0,003***
NYHA 3-4, n(%)	91 (49,7%)	50 (41,7%)	40 (65,6%)	
CAD, n (%)	135 (75,8%)	84 (73%)	49 (80,3%)	*0,35*
LVEF <50, n(%)	43 (24,7%)	19 (16,7%)	24 (41,4%)	***0,001***
AF, n(%)	37 (20,4%)	18 (15%)	18 (31%)	***0,01***
NT-proBNP ng/L Median, Q1-Q3	1747 (47-60000)	1361(47-35335)	3583(114-60069)	***0,01***
Device	23 (12,6%)	14 (11,7%)	9 (14,8%)	*0,91*
PPM, n(%)	18 (9,8%)	11 (9,2%)	7 (11,5%)	
ICD, n(%)	3 (1,6%)	2 (1,7%)	1 (1,6%)	
CRT-D, n(%)	2 (1,1%)	1 (0,8%)	1 (1,6%)	

* Values are mean ±SD or n (%) p value<0.05.

MR= Mitral Regurgitation; TR= Tricuspid Regurgitation; BMI=body mass index; BSA= Body Surface Area; STS= Society of Thoracic Surgeons; NYHA= New York Heart Association; CAD= Coronary Artery Disease; LVEF=Left ventriculer ejection fraction; AF= Atrial fibrilation; PPM= Permanent pacemaker; ICD= Implanted cardioverter-defibrillator; CRT-D= Cardiac resynchronization therapy-Defibrillator

Table 2. Echocardiography characteristics of the patients before the procedure

Parameters	Total n:183	MR None/Mild n:120 66,3%	MR Moderate/ Severe n:61 33,7%	P value
LVEF, %	54,2±12	56,4±11,6	49,7±13,5	***0,001***
RVEF, %	54,1±3,2	54,2±2,9	53,7±3,6	*0,27*
LVEDD, (cm)	4,9±2,6	4,9±3,2	5,0±0,7	*0,87*
LVESD, (cm)	3,1±0,8	3,0±0,6	3,4±0,9	***0,001***
LA, (cm)	4,3±0,7	4,1±0,6	4,7±0,6	***0,001***
RV, (cm)	2,4±0,3	2,5±0,3	2,4±0,3	*0,62*
IVS, (cm)	1,3±0,2	1,3±0,2	1,3±0,2	*0,17*
PW, (cm)	1,1±0,1	1,1±0,1	1,1±0,2	*0,13*
RWT	0,51±0,12	0,52±0,12	0,47±0,13	***0,01***
LVMI, (g/m2)	129,2±37	125,7±32	137,8±44	*0,06*
AVA, (cm2)	0,7±0,1	0,7±0,1	0,7±0,1	*0,47*
AVmax(m/sn)	4,3±0,7	4,3±0,6	4,3±0,7	*0,74*
Aort Max Gradient(mmHg)	77,8±19	76,8±18,4	79,7±20,3	*0,34*
Aort Mean Gradient (mmHg)	48±12	48,2±12,5	47,7±13,8	*0,83*
TAPSE (mm)	21±3,6	21,2±3,4	20,7±3,9	*0,46*
RVSM (cm/san)	11,9±4	11,6±1,9	12,3±6,9	*0,38*
TRV (m/san)	2,7±0,6	2,5±0,6	3,1±0,6	***0,001***
sPAP (mmHg)	39,1±16	35,1±15	47,2±15,5	***0,001***

* Values are mean ±SD or n (%) p value<0.05.
MR= Mitral Regurgitation; TR= Tricuspid Regurgitation; LVEF= Left ventricular systolic ejection fraction; RVEF; Right ventricular systolic ejection fraction; LVEDD= Left ventricular end diastolic diameter; LVESD= Left ventricular end systolic diameter; LA= Left atrial diameter; RV= Right ventricular diameter; IVS= Interventricular septum; PW= Posterior wall; RWT= Relative wall thickness; LVMI= Left ventricular mass index; AVA= Aort valve area; TAPSE= Tricuspid annuler plane systolic excursion; RVsm= Right ventricular systolic motion tissue doppler imaging; TRV=Tricuspid regurgitation velocity; SPAP=Systolic pulmonary artery pressure

Table 3. Univariate and multivariate analysis for prediction of mitral regurgitation improvement

Parameters	Univariate analysis		Multivariate analysis	
	OR (95% CI)	P value	OR (95% CI)	P value
Age	0.971 (0.920-1.025)	*0,28*	-	-
STS Score	0.938 (0.821-1.071)	*0,34*	-	-
CAD	1.434 (0.619-3.326)	*0,40*	-	-
LVEF <50	0.660 (0.282-1.544)	*0,33*	-	-
NT-proBNP	0.808 (0.435-1.503)	*0,35*	-	-
Diabetes mellitus	1.450 (0.668-3.149)	*0,34*	-	-
Renal disease	2.059 (0.741-5.719)	*0,16*	2.013 (0.650-6.232)	*0,22*
AF	1.277 (0.483-3.377)	*0,62*	-	-
ACEi or ARB	0.708 (0.318-1.577)	*0,39*	-	-
Beta bloker	0.274 (0.107-0.702)	***0,007***	0.261 (0.098-0.691)	***0,007***
LVEF	1.020 (0.992-1.049)	*0,15*	0.999 (0.954-1.046)	*0,96*
LVESD	0.716 (0.460-1.115)	*0,14*	0.920 (0.435-1.898)	*0,75*
LA	0.410 (0.229-0.734)	***0,003***	0.406 (0.219-0.755)	***0,004***
sPAP	0.980 (0.959-1.002)	*0,07*	0.990 (0.965-1.015)	*0,43*
Aortic Regurgitation	0.564 (0.182-1.752)	*0,16*	1.589 (0.285-8.862)	*0,38*
Tricuspid Regurgitation	0.824 (0.299-2.267)	*0,06*	0.821 (0.052-1.223)	*0,35*
Prothesis valve size	0.860 (0.759-0.974)	***0,01***	0.921 (0.684-1.241)	*0,59*
Sinus valsalva diameter	0.865 (0.771-0.972)	***0,01***	0.825 (0.718-0.948)	***0,006***
PCI before the procedure	3.157 (0.705-14.124)	*0,13*	4.822 (0.976-23.833)	*0,054*
Post-procedure PPM	0.256 (0.033-2.019)	*0,19*	3.556 (0.423-29.902)	*0,26*

Logistic univariate and multivariate model, P value<0.05.
OR= Odds ratio; CI= Confidence interval
STS= Society of Thoracic Surgeons; CAD= Coronary Artery Disease; AF= Atrial fibrilation; ACEi=Angiotensinogen was converting enzyme inhibitor; ARB=Angiotensin receptor blocker; LVEF= Left ventricular ejection fraction; LVESD= Left ventricular end-sistolic diameter; LA= Left atrial diameter; SPAP=Systolic pulmonary artery pressure; PCI= Percutaneous coronary intervention; PPM= Permenant pacemaker

Methods: We retrospectively analyzed data of 183 patients who underwent TAVI between December 2012 and October 2021 at the Department of Cardiology, Ege University Faculty of Medicine. Transthoracic echocardiographic parameters were evaluated at three time points: baseline (pre-TAVI), early post-procedural (0–3 months), and late post-procedural (3–12 months). Based on pre-procedural echocardiography, patients were stratified into two groups according to mitral regurgitation (MR) severity: those with none to mild MR and those with moderate to severe MR, following guideline-based criteria. MR improvement was defined as a reduction of at least one grade in MR severity compared to baseline, as assessed during follow-up. Logistic regression analyses were performed to determine independent predictors of MR improvement.

Results: The mean age of the study population was 77.6 ± 6 years, and 44.3% were male (Table 1). MR improvement was observed in 35 patients (20.3%; p<0.001) in the early period and in 49 patients (38.8%; p=0.001) in the late period (Figure 1). Multivariate analysis identified beta-blocker use (OR: 0.26; p=0.007), LA dilation (OR: 0.41; p=0.004), and increased sinus valsalva diameter (OR: 0.82; p=0.006) as independent predictors (Table 3).

Conclusions: Previous studies have shown that MR improvement after TAVI is more likely in patients with functional/secondary MR. Doldi et al. demonstrated higher MR regression rates in patients with atrial and ventricular functional MR. Although our study found a significant relationship between LA dilation and MR improvement, we did not categorize MR subtypes. Interestingly, we report for the first time that an increased sinus of Valsalva diameter is a prognostic factor for MR improvement. This may be related to reduced mechanical stress on the mitral apparatus and preserved aorto-mitral geometry. Prior literature has also identified beta-blocker use, absence of atrial fibrillation, lower systolic pulmonary artery pressure, and higher LVEF as contributing factors. However, the limited number of patients and heterogeneity in existing studies highlight the need for further research. MR improvement is frequently observed after TAVI. Identifying predictive factors may aid in patient selection and clinical decision-making. The presence of moderate or severe MR should not be considered a contraindication to TAVI; instead, patients with high likelihood of improvement should be evaluated comprehensively.

OP-016 [Cardiac Imaging / Echocardiography]

Non-invasive assessment of plaque characteristics in diffuse and focal coronary artery disease; Plaque vulnerability and high-risk features

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Background and Aim: Recent studies have suggested that focal and diffuse atherosclerotic coronary artery disease (CAD) have significantly different plaque composition, treatment efficacy and outcomes. In this study, we aimed to investigate the plaque characteristics of diffuse and focal lesions in patients who underwent coronary computed tomography angiography (CCTA) for the assessment of obstructive CAD.

Methods: Patients with CAD-RADS score ≥2 were retrospectively reviewed. Plaque characteristics, composition and high-risk features were assessed. Plaques ≥20 mm or ≥25% of total vessel length were accepted as diffuse lesions and otherwise focal lesions. CCTA-derived plaque features and lesion-oriented cardiac outcomes were compared.

Results: After the exclusion of ineligible patients, 597 lesions of 441 patients were evaluated. The mean age of the study population was 55 ± 9.4 years. There were 463 focal lesions and 134 diffuse lesions. Diffuse lesions demonstrated higher calcification (p=0.001). CCTA-derived high-risk features, including spotty calcification (p=0.001), low-attenuation plaque (p=0.041) and positive remodeling (p=0.016), were more prevalent in focal disease. In addition, high-risk plaque was also higher in focal lesions (p=0.022). Moreover, lesions with ≥70% plaque burden were significantly higher in focal lesions (p=0.044). Multivariate analysis demonstrated that plaque burden ≥70% and CCTA-derived high-risk plaque were independently associated with cardiac outcomes. During the follow-up, lesion-oriented myocardial infarction and revascularization were higher in diffuse disease (p=0.015 and p<0.001, respectively).

Conclusions: Diffuse and focal atherosclerotic lesions demonstrate significant differences regarding plaque vulnerability. Moreover, diffuse plaques have a worse prognosis compared to focal lesions.

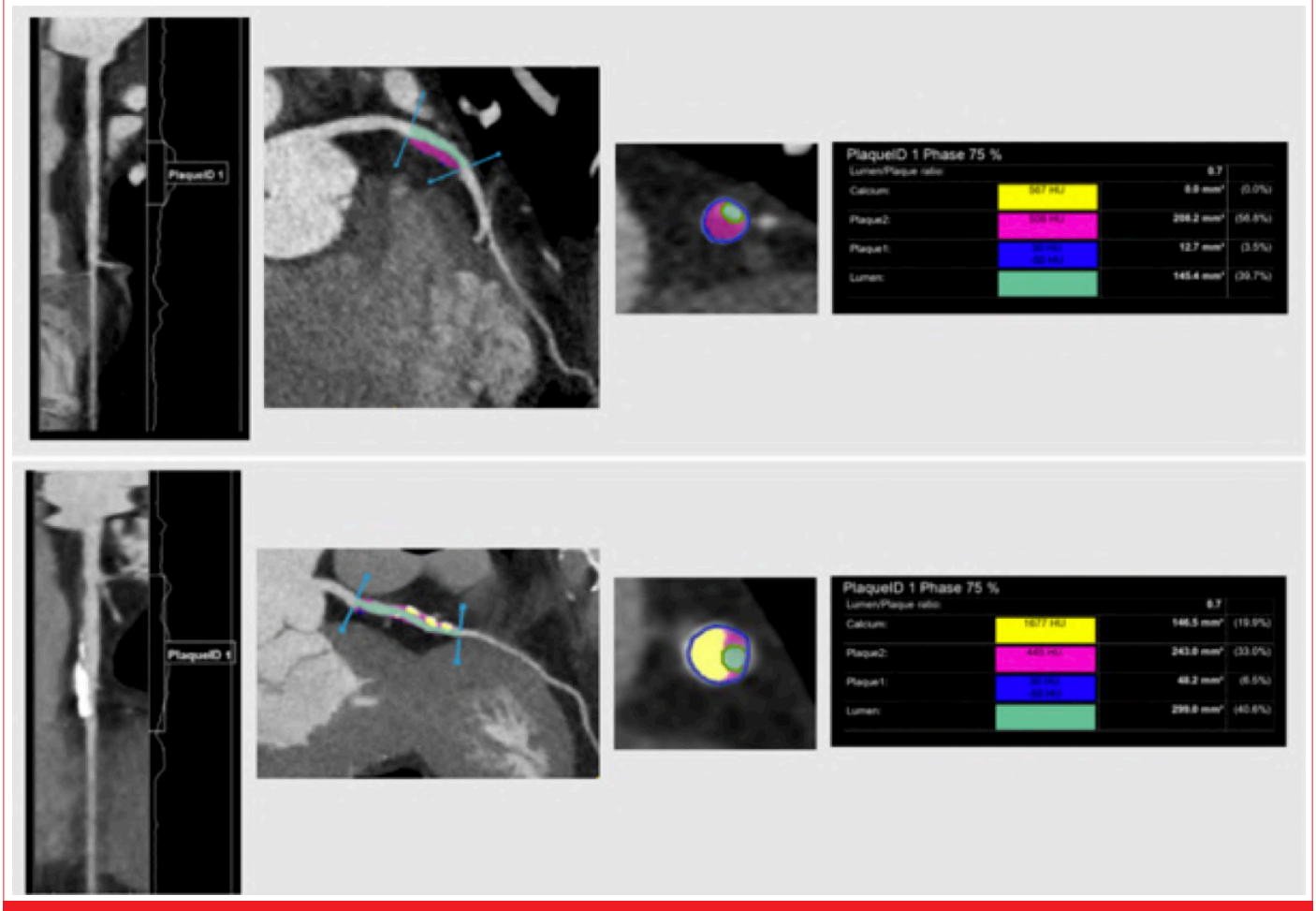

Figure 1. Quantification of coronary artery plaque burden, top panel showing focal atherosclerotic lesion, bottom panel showing diffuse atherosclerotic lesion.

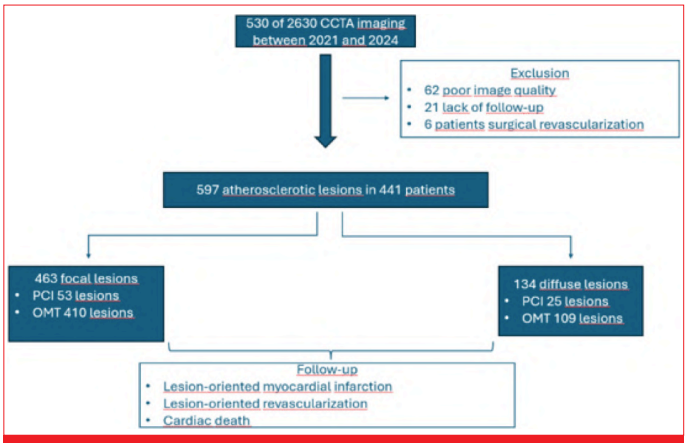

Figure 2. Patient selection and study design.

Table 1. Predictors of lesion-oriented cardiac outcomes

	Univariate analysis for MACE			Multivariate analysis for MACE		
	OR	CI	p value	OR	CI	p value
Diabetes mellitus	1,808	1,243-2,628	**0,002**	1,291	0,766-2,178	0,338
Hypertension	2,949	1,756-4,951	**<0,001**	2,587	1,304-5,131	**0,007**
Dyslipidaemia	3,216	2,063-5,015	**<0,001**	2,038	1,051-3,953	**0,035**
Smoking	1,466	0,986-2,182	0,059			
Plaque burden ≥ 70%	4,213	1,586-11,188	**0,004**	6,201	1,751-21,966	**0,005**
Napkin-ring sign	1,812	0,998-3,291	0,051			
Spotty calcification	1,868	1,043-3,344	**0,035**	1,104	0,464-2,624	0,823
Low attenuation	1,275	0,715-2,275	0,410			
Positive remodeling	1,065	0,724-1,568	0,749			
High-risk plaque	1,781	1,129-2,810	**0,013**	1,897	0,957-3,761	**0,016**

Table 2. Comparison of CCTA-derived plaque characteristics of diffuse and focal lesions

Feature	Focal disease 463 lesions	Diffuse disease 134 lesions	p value
Vessel			0.002
LAD	48.1%	61.3%	
LCx	24.8%	14.9%	
RCA	27.1%	23.8%	
Plaque type (%)			0.001
Calcified	54.4	57.7	
Partially calcified	28.6	31	
Soft	17	11.3	
Plaque volume (mm^3)	216 (71-526)	323 (116-822)	<0.001
Plaque burden (%)	36.9 (24.6-48.9)	38 (24.8-48.7)	0.989
Plaque burden ≥70 (%)	5	2	0.044
High risk features (%)			
Napkin-ring sign	8.1	6.2	0.231
Positive remodelling	42.9	6.4	0.016
Spotty Calcification	13.6	7.4	0.001
Low-attenuation plaque	1.5	34.3	0.041
High-risk plaque (%)	19.5	13.5	0.022

LAD: Left anterior descending artery, LCx: Left circumflex artery, RCA: Right coronary artery

Table 3. Comparison of lesion-oriented cardiac outcomes

	Focal disease	Diffuse disease	p value
Myocardial infarction (%)	1.3	8.3	<0.001
Revascularization (%)	12.6	19.3	0.015
Cardiac death	1.1	1.6	0.410
Composite outcomes	13.1	20	0,015

Table 4. Patients' demographics and clinical features

Age (years)	55 ± 9.4
Gender (male)	80.1
Diabetes mellitus (%)	27.6
Hypertension (%)	67.6
Smoking history (%)	56.3
Dyslipidemia (%)	56.1
Agatsons score	61 (17-228)
Plaque type (%)	
Calcified	61.6
Partially calcified	19.4
Soft	19.0
CCTA-derived high risk features (%)	
High-risk plaque	16.2
Napkin-ring sign	9.8
Positive remodelling	9
Low-attenuation plaque	12.6
Spotty Calcification	39.2
Plaque burden (%)	33.3 (20.4-45.2)
Plaque burden ≥70 (%)	1.3
Follow-up duration (months)	33 (21-40)

OP-017 [Cardiac Imaging / Echocardiography]

Phenotypes and clinical outcomes of hypertrophic cardiomyopathy from a large cohort in Türkiye

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Background and Aim: Hypertrophic cardiomyopathy (HCM) is an inherited myocardial disorder defined by left ventricular

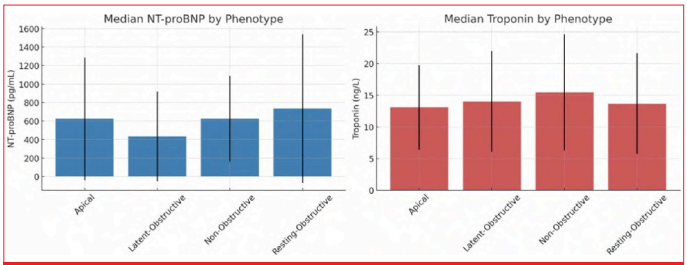

Figure 1. Median NT-proBNP and troponin levels across different hypertrophic cardiomyopathy (HCM) phenotypes. Error bars represent the interquartile range (IQR).

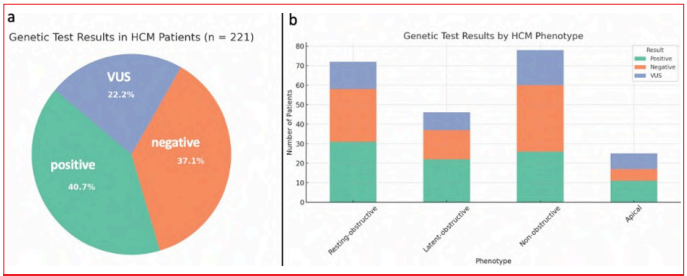

Figure 2. Genetic testing results in patients with HCM. (a) Distribution of genetic test results among 221 patients who underwent genetic testing. Results were categorized as positive (pathogenic or likely pathogenic variants), negative, or variant of uncertain significance (VUS). (b) Genetic test outcomes stratified by HCM phenotype, including resting-obstructive, latent-obstructive, non-obstructive, and apical subtypes.

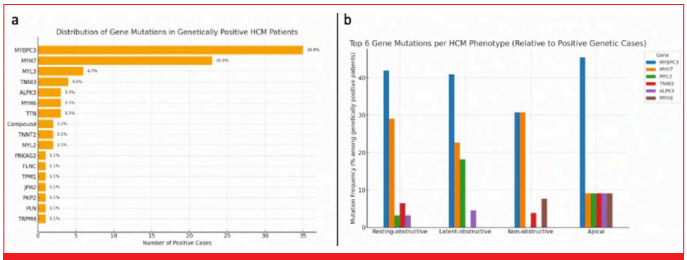

Figure 3. Distribution of gene mutations in genetically positive hypertrophic cardiomyopathy (HCM) patients. (a) Frequency of individual gene mutations among genetically positive cases. MYBPC3 and MYH7 were the most commonly detected mutations, followed by MYL3, TNNI3, ALPK3, and MYH6. (b) Distribution of the top six gene mutations stratified by HCM phenotype (resting-obstructive, latent-obstructive, non-obstructive, and apical), relative to genetically positive cases.

hypertrophy that cannot be fully explained by loading conditions. While its prevalence has been studied extensively through imaging modalities and electronic health records, regional differences in phenotypic expression and prognosis remain underexplored. Genetic predisposition, combined with environmental and geographical factors, highlights the need for population-specific data. Given the limited national data, this study aims to evaluate the epidemiological features, phenotypic characteristics, and prognostic outcomes of HCM patients followed at our center.

Table 1. Baseline demographic and clinical characteristics of the study cohort according to hypertrophic cardiomyopathy phenotypes

Variable	Overall	Resting-obstructive	Latent-obstructive	Non-obstructive	Apical	p-value[2]
	N = 701	N = 228	N = 141	N = 267	N = 65	
Sex (female), n (%)	224 (32)	86 (38)	47 (33)	68 (25)	23 (35)	0.028
Age, years	53.0 (45.0, 62.0)	55.0 (46.0, 63.0)	52.0 (44.0, 60.0)	53.0 (44.0, 62.0)	53.0 (44.0, 61.0)	0.178
BMI	28.4 (25.7, 31.6)	28.5 (25.8, 31.6)	27.8 (26.2, 31.5)	28.5 (25.7, 31.2)	28.0 (25.6, 31.2)	0.975
Family history of CM, n (%)	115 (17)	39 (17)	32 (23)	33 (13)	11 (17)	0.055
Family history of SCD, n (%)	84 (12)	30 (13)	19 (14)	29 (11)	6 (9.4)	0.688
Atrial fibrillation, n (%)	130 (19)	52 (23)	24 (17)	45 (17)	9 (14)	0.220
HT, n (%)	355 (51)	101 (45)	79 (56)	151 (57)	24 (37)	0.003
DM, n (%)	101 (15)	31 (14)	25 (18)	38 (14)	7 (11)	0.547
CAD, n (%)	190 (27)	52 (23)	37 (27)	82 (31)	19 (29)	0.248
Stroke, n (%)	19 (2.7)	4 (1.8)	4 (2.9)	11 (4.1)	0 (0)	0.225
Smoking, n (%)	168 (25)	47 (21)	36 (27)	70 (27)	15 (23)	0.427
NYHA, n (%)						0.066
1	195 (29)	52 (24)	47 (35)	72 (29)	24 (39)	
2	337 (51)	110 (50)	63 (47)	139 (56)	25 (41)	
3	117 (18)	49 (22)	22 (16)	35 (14)	11 (18)	
4	13 (2.0)	7 (3.2)	2 (1.5)	3 (1.2)	1 (1.6)	
Syncope, n (%)	85 (12)	31 (14)	22 (16)	24 (9.1)	8 (13)	0.196
Presyncope, n (%)	101 (15)	40 (18)	25 (18)	31 (12)	5 (7.8)	0.059
Dyspnea, n (%)	298 (43)	107 (47)	65 (46)	102 (38)	24 (38)	0.167
Angina, n (%)	139 (20)	42 (19)	27 (20)	62 (24)	8 (13)	0.215
Palpitation, n (%)	143 (21)	51 (23)	28 (20)	54 (21)	10 (16)	0.701
SCD score	2.2 (1.5, 3.4)	2.7 (2.0, 4.4)	2.2 (1.7, 3.7)	1.8 (1.3, 2.8)	1.9 (1.3, 3.0)	0.000

BMI – Body mass index, CAD- Chronic artery disease, CM- Cardiomyopathy, DM – Diabetes Mellitus, HT – Hypertension, NYHA- New York Heart Association, SCD – Sudden cardiac death

Methods: We retrospectively analyzed adult patients diagnosed with hypertrophic cardiomyopathy HCM guidelines. Patients with secondary LV hypertrophy or incomplete data were excluded. Clinical, laboratory, ECG, and imaging data-including TTE for all and CMR when indicated-were collected. HCM phenotypes were classified as obstructive, non-obstructive, or apical based on LVOT gradients and hypertrophy pattern. Genetic testing was performed in selected cases, with classification per ACMG/AMP 2015 guidelines.

Results: The cohort included 701 patients with a median age of 53 years, of whom 68% were male. The phenotypic distribution comprised 9.3% apical, 38.1% non-obstructive, 32.5% resting obstructive, and 20.1% latent obstructive HCM. ICD implantation history was more frequent among obstructive patients, particularly in the latent obstructive group compared to the non-obstructive group. NT-proBNP levels were numerically highest in the resting-obstructive type. Differences in biomarker profiles among HCM phenotypes are depicted in Figure 1. Although LGE was more frequently observed in apical HCM, post-hoc analysis showed no significant difference in prevalence across subgroups. In contrast, LGE extent was significantly greater in the apical group (p=0.003). Genetic testing, performed in 32% of patients, revealed a 44% positivity rate, with MYBPC3 and MYH7 being the most commonly detected mutations. Genetic testing results in patients with HCM are summarized in Figure 2 and distribution of gene mutations in genetically positive patients are shown in Figure 3. The overall mortality rate was 2.8%, with heart failure identified as the leading cause of death. The findings of our study are summarized in the tables.

Conclusions: This study offers an important epidemiological data on HCM from this regional cohort, highlighting the phenotypic spectrum and clinical features. Obstructive HCM-especially the resting subtype-was the most prevalent, linked to more severe symptoms, higher NT-proBNP levels, and frequent mitral regurgitation. CMR improved detection of apical variants and LGE, key for risk stratification. Genetic testing, performed in about one-third

Table 2. Therapeutic interventions, medication use and laboratory findings according to hypertrophic cardiomyopathy phenotypes

Characteristic	Overall	Resting-obstructive	Latent-obstructive	Non-obstructive	Apical	p-value[2]
	N = 701	N = 228	N = 141	N = 267	N = 65	
Presence of ICD, n (%)	83 (11.8)	31 (14)	25 (18)	22 (8.3)	5 (7.7)	0.022
Alcohol septal ablation, n (%)	37 (5.3)	28 (12)	9 (6.4)	0 (0)	0 (0)	0.000
Surgical myectomy, n (%)	17 (2.4)	9 (3.9)	5 (3.5)	3 (1.1)	0 (0)	0.095
Disopyramide, n (%)	68 (9.9)	51 (22.3)	17 (12)	0 (0)	0 (0)	0.000
Beta blocker, n (%)	566 (80.7)	191 (83.8)	116 (82.3)	213 (79.8)	46 (70.8)	
metoprolol	367 (54)	134 (58.7)	80 (56.7)	121 (45.3)	37 (56.9)	
bisoprolol	114 (17)	41 (17.9)	22 (15.6)	44 (17)	6 (9.2)	
Calcium channel blockers, n (%)	139 (19.8)	41 (17.9)	30 (21.2)	59 (22)	9 (13.8)	
Diuretics, n (%)	223 (31.8)	64 (28)	46 (32.6)	99 (37)	14 (21.5)	
ACEi or ARBs, n (%)	284 (42)	76 (34)	68 (49)	120 (46)	20 (31)	0.004
OACs, n (%)	103 (15)	44 (20)	20 (14)	31 (12)	8 (13)	0.097
eGFR, mL/min/1,73 m²	93.0 (74.3, 105.0)	92.0 (74.2, 105.0)	95.6 (78.0, 108.0)	92.0 (70.4, 103.5)	95.3 (83.1, 107.6)	0.093
CKMB, ng/mL	2.9 (2.0, 4.2)	3.0 (2.0, 4.5)	2.8 (1.8, 4.5)	2.9 (2.0, 4.1)	2.4 (1.8, 3.2)	0.285
Troponin, ng/L	14 (8, 24)	13 (9, 25)	15 (8, 26)	14 (9, 24)	14 (7, 23)	0.553
NT-ProBNP, pg/mL	626.2 (206.9, 1,466.0)	737.0 (212.2, 1,820.0)	433.0 (121.0, 1,091.0)	625.0 (221.0, 1,162.0)	623.8 (302.4, 1,644.5)	0.006

ACE- Angiotensin-converting enzyme inhibitors, ARBs - Angiotensin receptor blockers, CKMB- Creatine Kinase-MB isoenzyme, eGFR- Estimated glomerular filtration rate, ICD- Implantable cardioverter-defibrillator, NT-ProBNP- N-terminal pro B-type natriuretic peptide, OAC- oral anticoagulant

Table 3. Electrocardiographic, echocardiographic, and cardiac magnetic resonance imaging findings across hypertrophic cardiomyopathy phenotypes

Characteristic	Overall	Resting-obstructive	Latent-obstructive	Non-obstructive	Apical	p-value[2]
	N = 701	N = 228	N = 141	N = 267	N = 65	
Electrocardiography						
Heart rate, bpm	73.0 (65.0, 83.0)	73.0 (65.0, 82.5)	74.0 (65.0, 85.0)	72.0 (65.0, 82.0)	73.0 (65.5, 83.5)	0.981
QRS duration, ms	92.0 (84.0, 102.0)	94.0 (85.0, 104.0)	94.0 (84.0, 102.0)	92.0 (84.0, 102.0)	87.0 (80.0, 97.0)	0.043
QTc duration, ms	445 (426, 464)	448 (428, 466)	445 (429, 464)	442 (422, 463)	447 (433, 464)	0.123
Echocardiography						
LVEF, %	60 (60, 65)	60 (60, 65)	60 (60, 65)	60 (55, 65)	60 (57, 60)	0.000
IVS, mm	17.0 (15.0, 20.0)	18.0 (16.0, 21.0)	17.0 (15.7, 20.8)	17.0 (15.0, 20.0)	13.0 (11.7, 15.0)	0.000
PW, mm	12.0 (11.0, 14.0)	12.5 (11.0, 14.0)	12.0 (10.5, 13.0)	13.0 (11.0, 14.0)	11.0 (10.0, 12.0)	0.000
MWT, mm	18.0 (16.0, 21.0)	18.0 (16.0, 21.9)	17.2 (16.0, 21.0)	17.0 (15.3, 20.5)	16.0 (14.0, 18.0)	0.000
E/e'	12.6 (10.0, 16.0)	12.5 (9.0, 15.7)	11.0 (9.2, 16.0)	12.6 (10.0, 16.5)	13.0 (10.5, 16.0)	0.612
LA diameter, mm	41.0 (37.0, 46.0)	43.0 (38.5, 48.0)	41.0 (37.0, 45.0)	40.0 (37.0, 46.0)	39.0 (34.6, 42.0)	0.000
Rest gradient, mmHg	31 (20, 48)	45 (35, 62)	17 (14, 22)	NA (NA, NA)	NA (NA, NA)	0.000
Provoked gradient, mmHg	60 (41, 86)	79 (62, 100)	40 (34, 51)	NA (NA, NA)	NA (NA, NA)	0.000
Mitral regurgitation, n (%)	129 (18.4)	69 (30.2)	18 (12.7)	33 (12.3)	9 (13.8)	0.000
PAPs, mmHg	27.0 (23.0, 35.0)	29.0 (24.0, 35.0)	26.0 (21.0, 30.0)	26.0 (23.0, 37.0)	28.5 (23.5, 39.0)	0.208
TAPSE, mm	21.1 (20.0, 23.2)	22.0 (20.0, 25.0)	21.0 (19.3, 23.0)	21.0 (20.0, 23.0)	20.0 (18.0, 22.0)	0.064
IVC diameter, mm	15.3 (13.0, 19.0)	16.0 (13.0, 20.0)	15.0 (12.0, 18.0)	15.0 (12.8, 19.0)	14.5 (12.4, 18.0)	0.406
Cardiac magnetic resonance imaging						
Presence of CMR, n (%)	587 (84)	179 (79)	120 (85)	223 (84)	65 (100)	0.001
CMR-LVEF, %	60.0 (56.0, 62.0)	60.0 (57.0, 62.0)	60.0 (56.0, 62.0)	60.0 (55.0, 61.0)	60.0 (55.0, 63.0)	0.000
CMR-MWT, mm	18.5 (16.0, 22.0)	19.0 (16.7, 23.0)	18.7 (16.5, 22.0)	18.0 (15.3, 21.2)	18.0 (15.5, 21.7)	0.039
CMR-RVEF, %	60.0 (56.0, 62.0)	60.0 (57.0, 62.0)	60.0 (56.0, 62.0)	60.0 (55.0, 61.0)	60.0 (55.0, 63.0)	0.278
Presence of LGE, n (%)	502 (86)	146 (82)	100 (83)	194 (87)	62 (95)	0.045
Extensive LGE, n (%)	146 (25)	32 (18)	24 (20)	66 (30)	24 (37)	0.003
Apical aneurysm, n (%)	17 (3.0)	7 (4.1)	3 (2.7)	1 (0.5)	6 (9.7)	0.001

CMR- Cardiac magnetic resonance,CMR-LVEF- Cardiac magnetic resonance Left ventricular ejection fraction, CMR-LVEF- Cardiac magnetic resonance Reft ventricular ejection fraction, CMR- MWT- Cardiac magnetic resonance- Mean wall thickness, ,IVC- Inferior vena cava, IVS- interventricular septum, LA- Left atrium, LGE- late gadolinium enhancement, LVEF- Left ventricular ejection fraction, MWT- Mean wall thickness, , PAPs- systolic pulmonary artery pressure, PW- posterior wall, TAPSE- Tricuspid annular plane systolic excursion

of patients, most often identified MYBPC3 and MYH7 mutations. These population-specific findings underscore the importance of regional data in guiding personalized HCM management.

Table 4. Clinical outcomes of the HCM patients

Characteristic	Overall	Resting-obstructive	Latent-obstructive	Non-obstructive	Apical	p-value[2]
	N = 701	N = 228	N = 141	N = 267	N = 65	
Follow-up, months	13.0 (4.0, 26.0)	13.0 (4.0, 28.3)	13.0 (3.0, 27.0)	13.0 (4.0, 24.0)	13.5 (3.7, 26.5)	0.496
ICD shock, n (%)	12 (14.4)	4 (12.9)	2 (8)	4 (18.1)	2 (40)	0.219
NSVT, n (%)	117 (17)	37 (16)	23 (16)	44 (16)	13 (20)	0.906
Mortality, n (%)	20 (2.9)	11 (4.8)	2 (1.4)	5 (1.9)	2 (3.1)	0.168
Mortality reasons, n (%)						0.960
Heart failure, n (%)	7 (35)	3 (27)	0 (0)	3 (60)	1 (50)	
Sudden death, n (%)	5 (25)	2 (18)	1 (50)	1 (20)	1 (50)	
ACS, n (%)	2 (10)	0 (0)	1 (50)	1 (20)	0 (0)	
Aortic dissection, n (%)	1 (5.0)	1 (9.1)	0 (0)	0 (0)	0 (0)	
Surgical myectomy, n (%)	2 (10)	2 (18)	0 (0)	0 (0)	0 (0)	
Lung cancer, n (%)	1 (5.0)	1 (9.1)	0 (0)	0 (0)	0 (0)	
Respiratory failure, n (%)	1 (5.0)	1 (9.1)	0 (0)	0 (0)	0 (0)	
Traffic accident, n (%)	1 (5.0)	1 (9.1)	0 (0)	0 (0)	0 (0)	

ACS- Acute coronary syndrome, ICD- Implantable cardioverter-defibrillator, NSVT- Nonsustained ventricular tachycardia

OP-018 [Lipid / Preventive Cardiology]

The impact of the change in SCORE-2 classification with the 2025 ESC/EAS lipid update on cardiovascular risk classification

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Background and Aim: Assessing CV risk is crucial for primary cardiovascular prevention. In 2021, the SCORE algorithm was updated to SCORE2, and different risk assessment limits were established for different age groups. However, the 2025 lipid guideline update returned to a general risk classification range, as in previous years. This analysis examined the impact of this change in risk assessment on classifying patients as very high risk

Methods: A total of 1450 non diabetic patients who experienced their first episode of acute coronary syndrome were included in the study. The CV risk calculations were determined using age, sex, smoking status, blood pressure, total cholesterol, and high density lipoprotein (HDL-C) non-HDL level data according to SCORE-2 2025 ESC lipit update, 2021 prevention guidelines and 2019 ESC dislipidemi guidelines.

Results: The ability to classify patients as very high risk decreased significantly with the 2025 update (14.9%). In fact, the success rate fell even below that achieved with the SCORE risk algorithm in the 2019 ECS guideline (21.6%). SCORE-2 classification, based on the 2021 prevention guideline in which it was first described, is significantly more successful (61.7%) (Figure 1). With the 2025 update, the rate of classification in very high risk in patients aged <50 years has decreased to less than 1% (Figure 2). Age dependence in CV risk classification has increased significantly with this update.

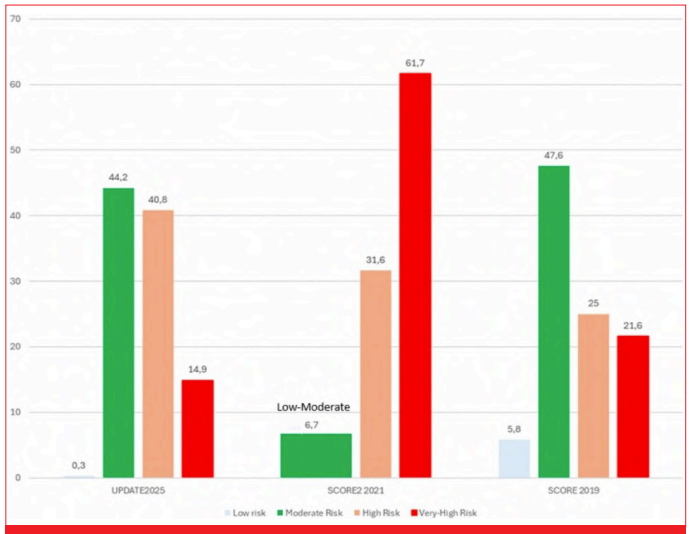

Figure 1. Comparison of cardiovascular risk groups.

Figure 2. According to 2025 ESC lipid update, cardiovascular risk classification by age categories.

Table 1. General characteristics

All patients (n)	1650
Male Gender n, (%)	1369 (83)
Age mean ± SD	56.78 ± 11.98
Systolic blood pressure (mmHg) Mean ± SD	142.50 ± 28.25
Total cholesterol (mg/dL) Mean ± SD	197.4 ± 48.7
Non HDL (mg/dL) Mean ± SD	155.06 ± 46.91
LDL (mg/dL) Mean ± SD	126.69 ± 39.61
TG (mg/dL) Mean ± SD (median Q1-Q3)	154.18 ± 115.57 123 (86-186)
HDL (mg/dL) Mean ± SD	41.63 ± 11.98
Boby mass index, Mean ± SD	27.50 ± 4.17
Family History	41.8 (689)
Smoker	62.1 (1025)

HDL: High density lipoprotein, LDL: Low density lipoprotein, TG: Triglyceride.

Conclusions: With the 2025 ESC lipid update, guideline recommendations for cardiovascular risk classification were regressed. The situation more prominent particularly in younger ages.

OP-019 [Lipid / Preventive Cardiology]

Can a structured, enhanced education and follow-up program, including medical students, be maintained over time in primary prevention for coronary artery disease in an economically challenged population?

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Background and Aim: Previous research suggests that primary prevention programs for coronary artery disease (CAD) can improve health-related behavioral outcomes. However, maintaining these programs can be very challenging and costly mainly due to staffing. Thus, the current study was designed to assess the feasibility, effectiveness, and long-term maintenance of a structured, enhanced education and follow-up program for CAD prevention in an area where the diverse population and economy are major issues.

Methods: Coronary Artery Disease Prevention Project was designed to utilize medical school students to conduct the entire project under the supervision of professors. This longitudinal prospective study began after obtaining ethical committee approval and included two different education and training phases; in the first phase, every year, between 2016 and 2025, third-year medical students underwent a one-year, specially designed, training program on primary prevention for CAD. In the second phase, every year, a series of conferences on primary prevention for CAD were organized by the SANKO University for underserved populations. Participants were prospectively assigned to an intervention where pre and post conference knowledge were collected and assessed. Every intervention was conducted by specially trained 4th year medical students and an education booklet which was specifically designed for this study was given to the participants. Every other month thereafter, for 6 months, each participant was followed by phone. At the 6 month follow up, data was collected to assess the impact of enhanced education and follow-up program on behavioral outcomes.

Results: A total of 245 participants were enrolled; 64% were women, mean age was 41.50 ± 12.39 years, 36% were not working. Mean BMI was 27.30 ± 4.99 kg/m2. Overall, at baseline evaluation, knowledge on CAD risk factors, primary prevention measures, diet and daily exercise habits were very poor. After the enhanced education and follow-up program there was a significant improvement on the knowledge of CAD risk factors and primary prevention measures ($p<0.001$). The follow-up program led the participants to implement those positive changes into their lives and maintain a healthy lifestyle. More importantly, this model program was successfully implemented for almost a decade.

Conclusions: The results of our study indicated that a structured enhanced education and follow-up program, utilizing medical students, can be maintained over time in primary prevention for

coronary artery disease in an economically disadvantaged community, yielding successful outcomes. This model program not only serves the public interest but also fosters early active engagement between medical students and patients throughout their careers.

OP-020 [Interventional Cardiology / Coronary]

Impact of bifurcation angles on long-term outcomes of T stenting and protrusion technique in STEMI

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Background and Aim: T-stenting and Protrusion (TAP) is a frequently employed bailout strategy in coronary bifurcation lesions, yet angle-based recommendations for its use are primarily based on bench testing rather than patient-level outcomes. This study aimed to evaluate the long-term clinical outcomes of TAP stenting according to bifurcation angle in patients with ST-elevation myocardial infarction (STEMI).

Methods: We retrospectively analyzed 193 consecutive STEMI patients who underwent TAP stenting due to side branch (SB) compromise during a provisional strategy between 2015 and 2024. Patients were stratified into three groups based on bifurcation angle: >70°, 50–70°, and <50°. The primary endpoint was target lesion failure (TLF), defined as a composite of cardiac death, target vessel myocardial infarction (TVMI), and clinically driven target lesion revascularization (TLR). Secondary endpoints included all-cause mortality, stent thrombosis (ST), and target vessel revascularization

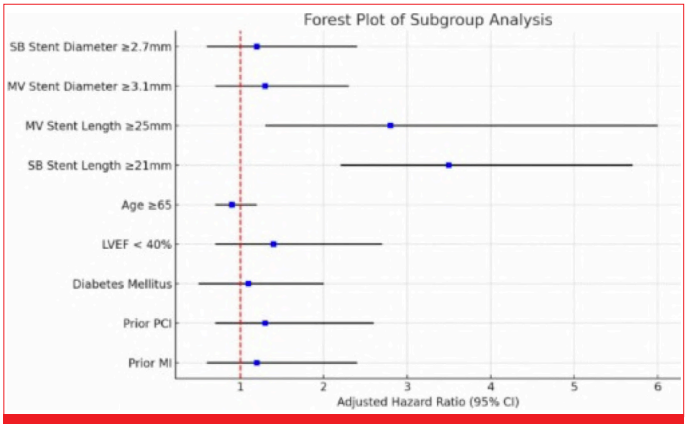

Figure 1. Forest plot of subgroup analysis for the primary outcome. This forest plot displays adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) derived from Cox regression analyses of predefined subgroups. Subgroups include demographic (age ≥65), clinical (prior MI, prior PCI, diabetes mellitus, LVEF <40%), and procedural parameters (MV stent length ≥25 mm, SB stent length ≥21 mm, MV stent diameter ≥3.1 mm, SB stent diameter ≥2.7 mm). Notably, both MV and SB stent lengths above the defined thresholds were significantly associated with an increased risk of TLF, as their 95% CIs did not cross unity. The dashed vertical line indicates the null effect (HR=1.0).

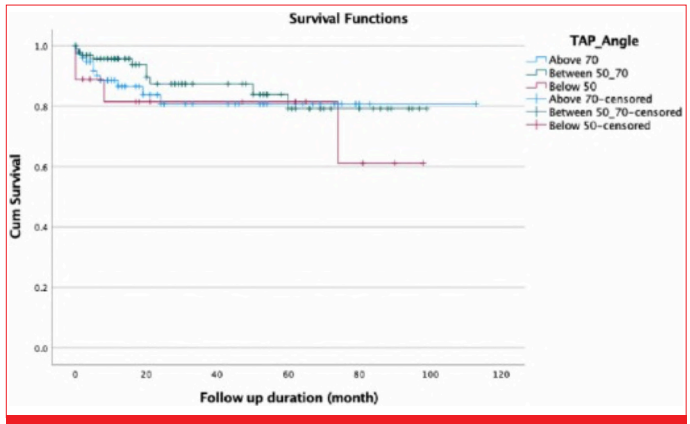

Figure 2. Kaplan–Meier survival curves stratified by bifurcation angle groups. Kaplan–Meier curves demonstrating cumulative survival free from the primary outcome according to post-procedural bifurcation angle groups: >70°, 50°–70°, and <50°. Although numerical differences were observed in estimated mean survival time (93.1, 84.7, and 75.6 months respectively), the overall comparison using the log-rank test did not reach statistical significance (χ^2 = 1.594, p = 0.451).

Table 1. Baseline demographic, clinical, and laboratory characteristics according to bifurcation angle groups

Variable	All n=193 , (%)	Group 1 (>70°, n=77)	Group 2 (50°–70°, n=98)	Group 3 (<50°, n=18)	p-value
Demographics					
Age, years	58.8 ± 11.05	60.4 ± 10.8	58.3 ± 10.3	55.0 ± 15.2	0.13
Male	157 (81.3)	59 (76.6)	82 (83.7)	16 (88.9)	0.34
Risk Factors					
HT	110 (59.5)	50 (69.4)	52 (54.7)	8 (44.4)	0.06
DM	62 (33.3)	30 (41.1)	28 (29.5)	4 (22.2)	0.16
Insulin-Depent	2 (1.1)	2 (2.7)	0 (0.0)	0 (0.0)	0.32
CKD	25 (13.0)	13 (17.1)	11 (11.2)	1 (5.6)	0.31
Hyperlipidemia	45 (23.3)	13 (16.9)	27 (27.6)	5 (27.8)	0.22
Current Smoker	58 (33.9)	20 (29.0)	32 (36.4)	6 (42.9)	0.47
PAD	4 (2.1)	2 (2.7)	2 (2.0)	0 (0.0)	0.77
Medical History					
Prior MI	49 (26.1)	20 (27.4)	26 (26.8)	3 (16.7)	0.63
Prior PCI	72 (37.5)	27 (35.5)	40 (40.8)	5 (27.8)	0.51
Prior Stroke	4 (2.1)	2 (2.7)	1 (1.0)	1 (5.6)	0.42
Heart Failure	33 (19.5)	13 (19.1)	15 (17.4)	5 (33.3)	0.35
LVEF, %	39.9 ± 10.9	39.8 ± 10.7	40.1 ± 11.3	40.3 ± 11.1	0.97
Laboratory					
eGFR, ml/min/1.73m²	83.7 ± 23.3	80.3 ± 23.8	84.9 ± 23.3	92.2 ± 19.4	0.12
Hemoglobin, g/dL	13.64 ± 1.8	13.30 ± 1.77	13.73 ± 1.97	14.48 ± 0.96	**0.05**
LDL, mg/dL	102.4 ± 42.6	95.5 ± 42.5	104.8 ± 42.7	122.2 ± 37.3	0.09
WBC, x10³/µL	9.53 ± 3.2	9.64 ± 3.3	9.43 ± 3.3	9.57 ± 3.0	0.93
CRP, mg/L	5.29 ±14.3	6.41 ± 19.4	3.84 ± 5.9	9.67 ± 24.2	0.37
Creatinine, mg/dL	1.10 ± 1.0	1.18 ± 1.32	1.08 ± 0.88	0.91 ± 0.27	0.63
Platelet count, x10³/µL	246.4 ± 72.2	248.7 ± 75.1	246.2 ± 71.9	239.0 ± 64.9	0.87

CKD; chronic kidney disease, CRP; C-reactive protein, DM; diabetes mellitus, eGFR; estimated glomerular filtration rate, HT; hypertension, LDL; low density lipoprotein, LEVF;

Table 2. Lesion characteristics according to bifurcation angle groups

Variable	All n=193 , (%)	Group 1 (>70°, n=77)	Group 2 (50°–70, n=98)	Group 3 (<50°, n=18)	p-value
Multivessel Disease	137 (71.7)	60 (78.9)	68 (70.1)	9 (50.0)	**0.04**
Complex Bifurcation Lesion	60 (31.1)	18 (23.4)	35 (35.7)	7 (38.9)	0.16
SYNTAX Score	21.02 ± 7.02	20.3 ± 6.8	21.6 ± 7.3	20.9 ± 6.9	0.47
SYNTAX (0-22)	121 (62.7)	52 (67.5)	56 (57.1)	13 (72.2)	0.25
SYNTAX (23-32)	56 (29.0)	20 (26.0)	32 (32.7)	4 (22.2)	0.50
SYNTAX (>32)	16 (8.3)	5 (6.5)	10 (10.2)	1 (5.6)	0.61
Medina 1.0.0	29 (15.0)	7 (9.1)	14 (14.3)	8 (44.4)	**<0.001**
Medina 1.1.0	29 (15.0)	17 (22.1)	10 (10.2)	2 (11.1)	0.08
Medina 0.1.0	34 (17.6)	11 (14.3)	23 (23.5)	0 (0.0)	**0.03**
Medina 1.1.1	41 (21.2)	20 (26.0)	18 (18.4)	3 (16.7)	0.41
Medina 0.1.1	45 (23.3)	18 (23.4)	23 (23.5)	4 (22.2)	0.99
Medina 1.0.1	15 (8.0)	4 (5.3)	10 (10.5)	1 (5.6)	0.42
Left Main Distal Lesion	18 (9.4)	14 (18.2)	4 (4.1)	0 (0.0)	**0.02**
LAD Lesion	123 (63.7)	44 (57.1)	68 (69.4)	11 (61.1)	0.24
LCX Lesion	66 (34.2)	22 (28.6)	36 (36.7)	8 (44.4)	0.33
RCA Lesion	19 (9.8)	10 (13.0)	8 (8.2)	1 (5.6)	0.46

LAD; left anterior descending artery LCX; left circumflex artery RCA; right coronary artery

Table 3. Procedural characteristics and medications according to bifurcation angle groups

Variable	All n=193 , (%)	Group 1 (>70°, n=77)	Group 2 (50°–70°, n=98)	Group 3 (<50°, n=18)	p-value
Main Vessel Stent Length, mm	25.1 ± 7.6	24.4 ± 7.6	25.7 ± 7.3	23.7 ± 9.6	0.53
Main Vessel Stent Diamater, mm	3.15 ± 0.4	3.16 ± 0.4	3.13 ± 0.3	3.15 ± 0.2	0.42
Main Vessel Stent Count	1.12 ± 0.3	1.13 ± 0.4	1.13 ± 0.3	1.06 ± 0.2	0.69
Side Branch Stent Length, mm	21.2 ± 5.9	20.6 ± 5.7	21.4 ± 5.9	21.3 ± 6.5	0.68
Side Branch Stent Diamater, mm	2.77 ± 1.6	2.70 ± 0.2	2.86 ± 2.2	2.53 ± 0.1	0.67
Side Branch Stent Count	1.04 ± 0.2	1.04 ± 0.2	1.03 ± 0.1	1.11 ± 0.3	0.37
POT balloon Size, mm	3.68 ± 0.4	3.71 ± 0.5	3.65 ± 0.4	3.70 ± 0.2	0.56
Medications					
Tirofiban	10 (5.2)	6 (7.8)	2 (2.0)	2 (11.1)	0.11
Clopidogrel	106 (55.8)	40 (52.6)	61 (62.2)	5 (31.3)	**0.05**
Ticagrelor	62 (32.6)	24 (31.6)	30 (30.6)	8 (50.0)	0.29
Prasugrel	25 (13.0)	10(13.2)	10 (10.2)	5(31.3)	0.07
Beta Bloker	164 (86.3)	67 (88.2)	83 (84.7)	14 (87.5)	0.79
Statin	175 (92.1)	70 (92.1)	91 (92.9)	14 (87.5)	0.76
ACE inhibitors / ARB	147 (77.8)	57 (76.0)	78 (79.6)	12 (75.0)	0.82

ACE: Angiotensin Converting Enyzime, ARB: Angiotensin Reseptor Blockor, POT; proximal optimization technique

(TVR). Cox regression analysis was performed to identify independent predictors of adverse outcomes.

Results: TLF occurred in 15.0% of the cohort (Group 1: 15.6%, Group 2: 10.2%, Group 3: 22.2%; p=0.30). While TLF and secondary outcomes were similar between the >70° and 50–70° groups, a numerically higher adverse event rate was observed in the <50° group, without reaching statistical significance. Cardiac death was significantly more frequent in the <50° group (p=0.02). In multivariable Cox regression, longer SB stent length, distal left main involvement, elevated CRP levels, and smaller POT balloon size were independent predictors of TLF. Interaction analysis revealed a significant modifying effect of bifurcation angle on the impact of SB stent length (p<0.001), and

Table 4. Clinical outcomes according to bifurcation angle groups

Outcomes	All n=193 , (%)	Group 1 (>70°, n=77)	Group 2 (50°–70°, n=98)	Group 3 (<50°, n=18)	p-value
In-Hospital					
In-hospital VT/VF	5 (2.6)	2 (2.6)	2 (2.0)	1 (5.6)	0.78
In-hospital Stroke	1 (0.5)	0 (0.0)	0 (0.0)	1 (5.6)	0.09
In-hospital MI	3 (1.6)	2 (2.6)	0 (0.0)	1 (5.6)	0.12
In-hospital Death	5 (2.6)	2 (2.6)	1 (1.0)	2 (11.1)	**0.05**
In-hospital TVR	5 (2.6)	1 (1.3)	4 (4.1)	0 (0.0)	0.40
Access-side complication	3 (1.6)	2 (2.6)	1 (1.0)	0 (0.0)	0.60
CIN	4 (2.1)	4 (5.2)	0 (0.0)	0 (0.0)	**0.04**
Long-Term					
Stroke	1 (0.5)	0 (0.0)	0 (0.0)	1 (5.6)	0.09
Stent Thrombosis	6 (3.1)	3 (3.9)	2 (2.0)	1 (5.6)	0.65
TVMI	15 (7.8)	7 (9.1)	7 (7.1)	1 (5.6)	0.40
TLR	7 (3.6)	5 (6.5)	1 (1.0)	1 (5.6)	0.11
TVR	15 (7.8)	6 (7.8)	7 (7.1)	2 (11.1)	0.35
All-Cause Death	7 (3.6)	3 (3.9)	3 (3.1)	1 (5.6)	1.0
Cardiac Death	7 (3.6)	3 (3.9)	2 (2.0)	2 (11.1)	**0.02**
Primary Outcome (TLF)	29 (15.0)	12 (15.6)	10 (10.2)	4 (22.2)	0.30
Secondary Outcome	24 (12.4)	8 (10.4)	12 (12.2)	2 (11.1)	0.75

Composite outcomes were calculated on a per-patient basis. In patients with multiple component events (e.g., TVR and death), only one event was counted. Therefore, total composite counts may be lower than the sum of individual components.
CIN; contrast-induced nephropathy, MI; myocaridal infarction, TLF; targer-lesion failure TLR; target-lesion revascularization, TVMI; target-vessel myocardial infarction, TVR; target-vessel revascularization, VT; ventricular tachycardia, VF; ventricular fibrillation

Table 5. Univariable and multivariable cox proportional hazards analysis for predictors of the primary outcome

	Univariate			Multivariate		
	HR	95 % CI	P	HR	95 % CI	P
Age	0.99	0.96 – 1.03	0.82			
Insulin Dependent DM	7.27	0.94 – 55.7	**0.05**	9.60	0.83 – 110.7	0.07
Prior PCI	2.04	0.93 – 4.50	0.07	3.25	0.87 – 12.1	0.08
LVEF	0.99	0.96 – 1.03	0.92			
Multivessel Disease	1.35	0.54 – 3.40	0.51			
Complex Bifurcation Lesion	0.58	0.22 – 1.57	0.29			
Left Main Distal Lesion	2.64	0.90 – 7.73	0.07	8.68	2.10 – 35.8	**0.03**
SYNTAX Score	0.97	0.92 – 1.03	0.41			
Bifurcation Angle >70°	1.27	0.58 – 2.81				
Bifurcation Angle 50°-70°	0.95	0.39 – 2.32				
Bifurcation Angle < 50°	1.69	0.53 – 5.43				
Main Vessel Stent Diameter	1.85	0.77 – 4.43	0.16			
Main Vessel Stent Length	0.89	0.83 – 0.96	**0.006**	0.91	0.81 – 1.01	0.09
Side Branch Stent Diameter	0.95	0.69 – 1.31	0.77			
Side Branch Stent Length	1.15	1.08 – 1.22	**<0.001**	1.20	1.08 – 1.34	**<0.001**
POT Balloon Size	0.97	0.96 – 4.52	0.06	0.85	0.72 – 26.3	**0.007**
CRP	1.02	1.00 – 1.04	**0.008**	1.04	1.00 – 1.08	**0.04**

CRP; C-reactive protein, DM; diabetes mellitus, LVEF; left ventricular ejection fraction, PCI; percutan coronary intervention, POT; proximal optimization technique

a borderline effect for MV stent length (p=0.087), with the greatest hazard observed in narrow-angle (<50°) bifurcations.

Conclusions: TAP stenting appears to be a safe and feasible bailout strategy not only for wide (>70°) but also for intermediate (50–70°) bifurcation angles in STEMI patients. However, very narrow bifurcation angles (<50°) may be associated with higher event rates, warranting further investigation through prospective studies.

OP-021 [Interventional Cardiology / Coronary]

DK-crush or mini-crush stenting for complex left main bifurcation lesions: The multicenter EVOLUTE-CRUSH LM registry

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Background and Aim: The comparison of outcomes of mini-crush (MCT) vs double kissing crush (DKC) techniques for complex left main bifurcation (LMB) lesions is still lacking. This investigation aimed to assess the long-term outcomes of patients who underwent MCT or DKC for LMB disease.

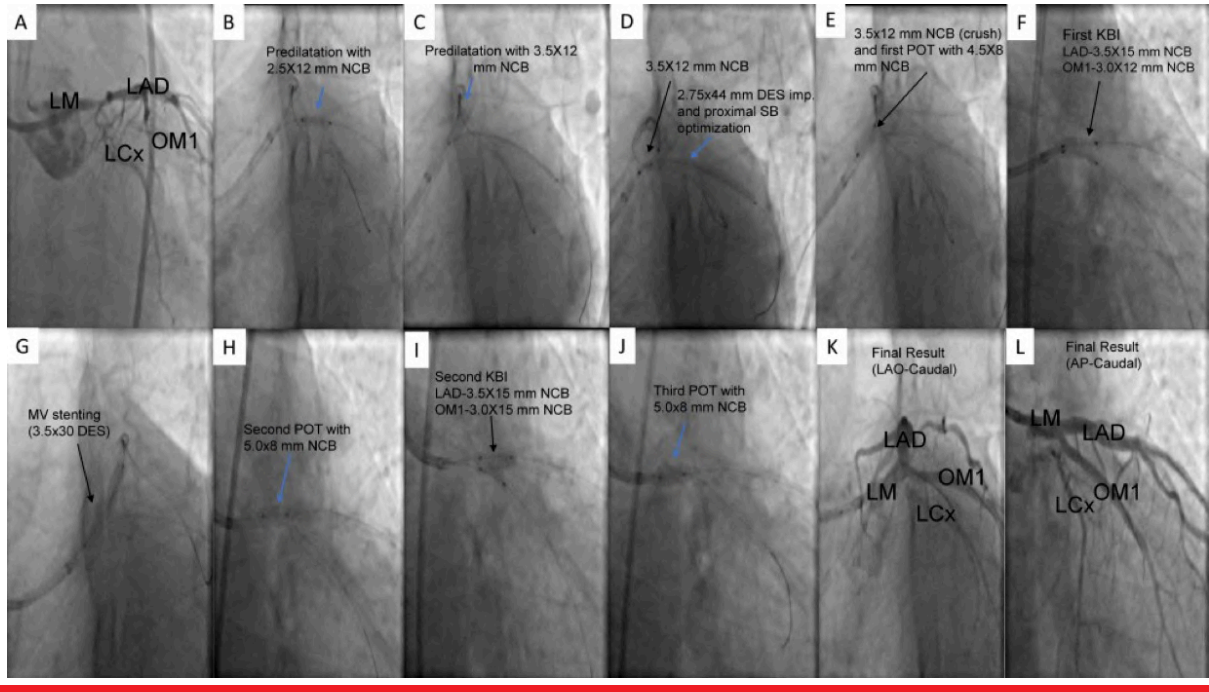

Figure 1. Step-by-step DKC via transfemoral approach. A) Bifurcation localization in the distal LM. B) Wiring and pre-dilatation of LCx-OM1. C) Pre-dilatation of LAD, an uninflated NCB in the distal LM, and LCx-OM1 stent positioning. D) LCx-OM1 stent placement and proximal side-branch optimization with stent balloon. E) Balloon crushing of the LCx-OM1 stent and first POT. F) First KBI with the NCBs. G) Positioning and placement of the LM-LAD stent after removing the stent balloon from the LCx-OM1. H) Second POT with NCB. I) Final KBI with NCBs. J) Third POT with the NCB. K,L) Final result. AP: Antero-posterior view; DES: Drug-eluting stent; DKC: Double kissing crush technique; KBI: Kissing balloon inflation; LAD: Left anterior descending artery; LAO: Left anterior oblique view; LCx: Left circumflex artery; LM: Left main coronary artery; MV: Main vessel; NCB: Non-compliant balloon; OM1: First obtus maginal artery; POT: Proximal optimization technique; SB: Side branch.

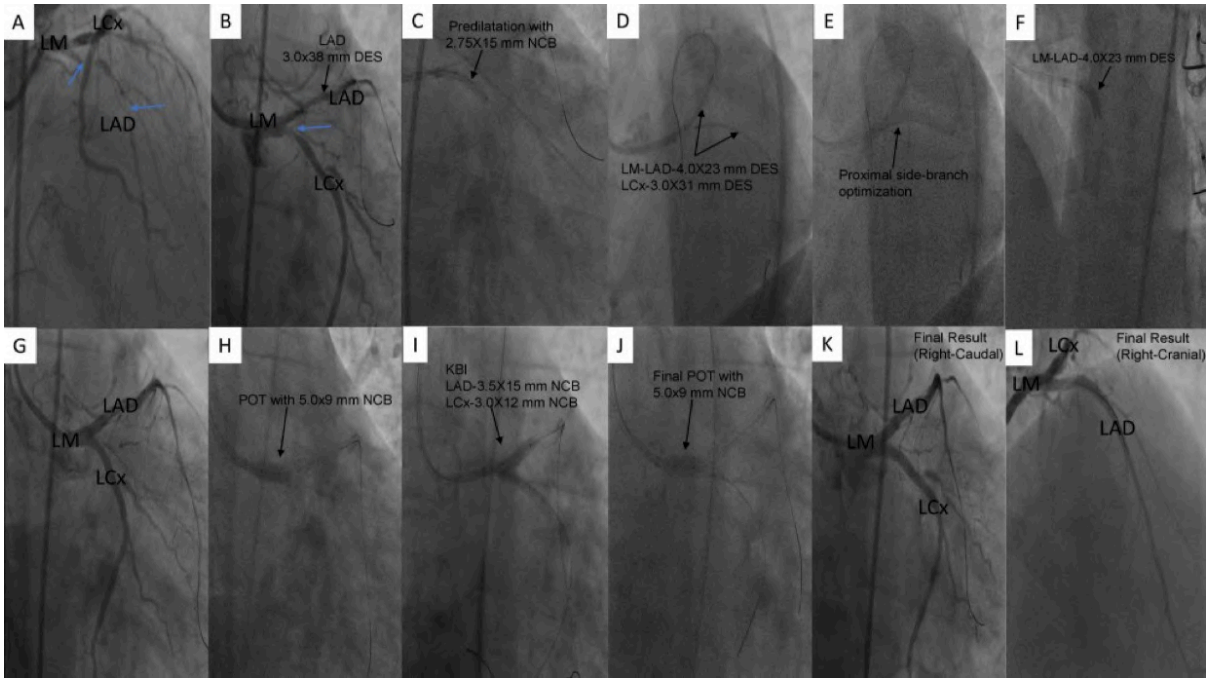

Figure 2. Technical steps of MCT via transfemoral access. A) Bifurcation localization in the distal LM and LAD-CTO lesion. B) Wiring and pre-dilatation of LAD and stenting proximal-mid LAD lesion. C) Pre-dilatation of LCx. D) LM-LAD and LCx stents positioning and placement. E) Proximal side-branch optimization with LCx stent balloon. F) Positioning and placement of the LM-LAD stent. G) Checking LM-LAD-LCx stenting before POT. H) POT with NCB. I) Final KBI with NCBs. J) Final POT with NCB. K, L) Final result. DES: Drug-eluting stent; KBI: Kissing balloon inflation; LAD: Left anterior descending artery; LCx: Left circumflex artery; LM: Left main coronary artery; MCT: Mini-crush technique; NCB: Non- compliant balloon; POT: Proximal optimization technique DES: Drug-eluting stent; KBI: Kissing balloon inflation; LAD: Left anterior descending artery; LCx: Left circumflex artery; LM: Left main coronary artery; MCT: Mini-crush technique; NCB: Non- compliant balloon; POT: Proximal optimization technique.

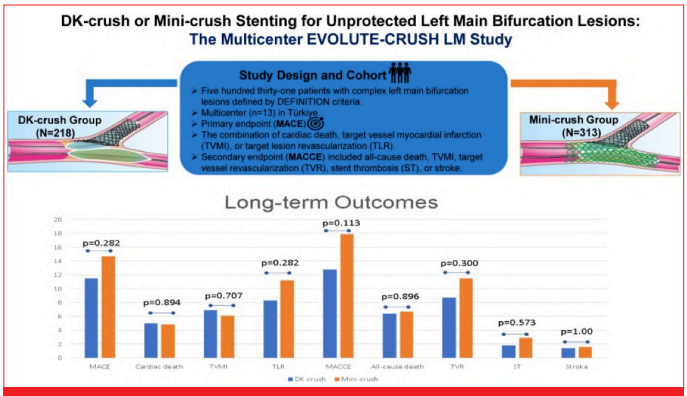

Figure 3. An overview of study design and clinical outcomes. DK: Double kissing; MACE: Major adverse cardiac events; MACCE: Major adverse cardiovascular and cerebral events; ST: Stent thrombosis; TVMI: Target vessel myocardial infarction; TLR: Target lesion revascularization; TVR: Target vessel revascularization.

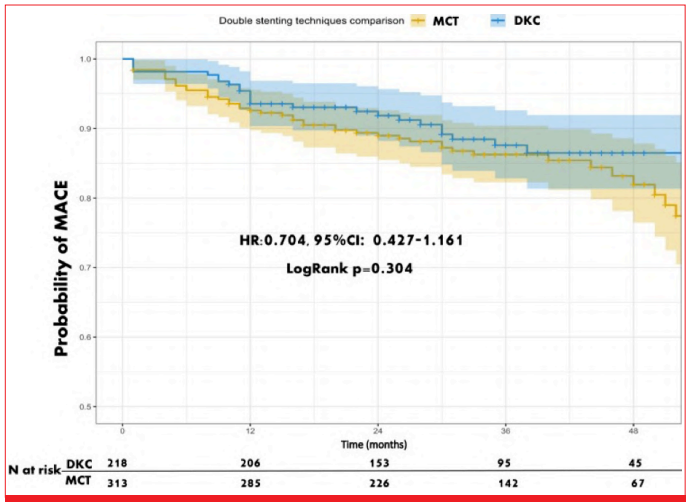

Figure 4. Kaplan-Meier curves for the primary endpoint (MACE) according to the treatment of DKC (blue) or MCT (yellow). DKC: Double kissing crush technique; HR: Hazard ratio; MACE: Major adverse cardiovascular events; MCT: Mini-crush technique; N: Number.

Methods: From 2014 to 2024, patients who underwent percutaneous coronary intervention (PCI) for complex LMB lesions were retrospectively collected. The primary endpoint was major adverse cardiac events (MACE) as the combination of cardiac death, target vessel myocardial infarction (TVMI), or clinically driven target lesion revascularization (TLR) during follow-up. Besides, the secondary endpoint was measured as major adverse cardiovascular and cerebral events (MACCE) including all-cause death, target vessel revascularization, TVMI, stent thrombosis, and stroke.

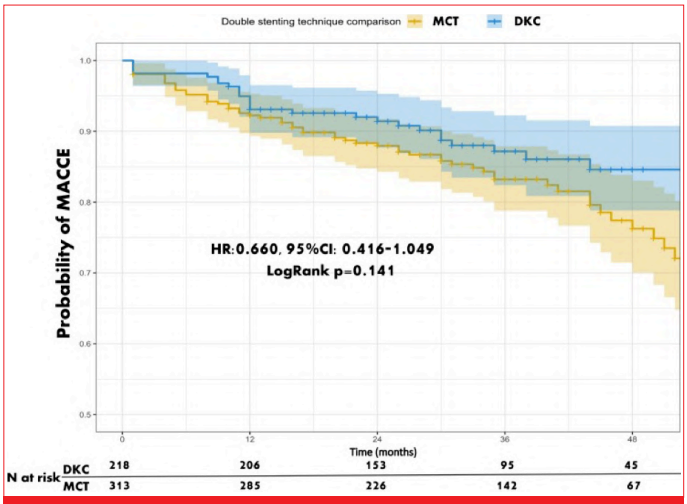

Figure 5. Kaplan-Meier curves for the secondary endpoint (MACCE) according to the treatment of DKC (blue) or MCT (yellow). DKC: Double kissing crush technique; HR: Hazard ratio; MACCE: Major adverse cardiovascular and cerebral events; MCT: Mini-crush technique; N: Number.

Table 1. Baseline demographic and clinical characteristics of per study group

Variables	DKC Group (n=218)	MCT Group (n=313)	P value
Age (years)	63.08±11.12	63.21±11.38	0.895
Male, sex n (%)	176 (80.7)	229 (73.2)	**0.044**
Body mass index, kg/m²	28.26±3.34	28.06±3.42	0.356
Comorbidities, n(%)			
Hypertension	149 (68.3)	222 (70.9)	0.524
Diabetes mellitus	103 (47.2)	151 (48.2)	0.821
Hyperlipidemia	117 (53.7)	170 (53.4)	0.884
Chronic kidney disease	62 (28.4)	92 (29.4)	0.812
Current Smoker	92 (42.2)	131 (41.9)	0.936
History of Stroke	12 (5.5)	15 (4.8)	0.713
Peripheral artery disease	17 (7.8)	25 (8.0)	0.937
Prior PCI	51 (23.4)	61 (19.5)	0.278
Prior MI	57 (26.1)	66 (21.1)	0.174
Heart Failure	34 (15.6)	45 (14.4)	0.698
LV Ejection Fraction (%)	54.19±9.76	54.22±9.64	0.936
Moderate-severe valve disease, n (%)	33 (15.1)	29 (9.3)	**0.038**
Laboratory measurements			
White blood cell count, (10³/L)	8.88±2.78	8.94±2.97	0.835
Hemoglobin, (g/dL)	14.36±12.39	13.05±1.78	0.319
Creatinine, (mg/dL)	1.09±.49	1.14±1.39	0.157
Platelet count, (10³/L)	238.23±59.67	237.26±72.25	0.726
Total cholesterol, (mg/dL)	182.05±53.20	186.25±51.15	0.259
Clinical Presentation, n (%)			
CCS	103 (47.2)	149 (47.6)	0.936
NSTEMI	94 (43.1)	137 (43.8)	0.882
USAP	21 (9.6)	27 (8.6)	0.691
Medications Used, n (%)			
Acetylsalicylic acid	217 (99.5)	311 (99.4)	0.785
Clopidogrel	107 (49.1)	151 (48.2)	0.849
Ticagrelor	77 (35.3)	109 (34.8)	0.906
Prasugrel	34 (15.6)	53 (16.9)	0.682
Beta Blockers	188 (86.2)	272 (86.9)	0.825
CCB	68 (31.2)	85 (27.2)	0.312
ACEI/ARB	165 (75.7)	233 (74.4)	0.744
Statin	199 (91.3)	281 (89.8)	0.562
Diuretics	52 (23.9)	81 (25.9)	0.596
Insulin	40 (18.3)	63 (20.1)	0.610

Bold indicates significance level at P <0.05. Abbreviations: ACEI: Angiotensin-converting enzyme inhibitor, ARB: Angiotensin receptor blocker, CCB: Calcium channel blocker; CCS: Chronic coronary syndrome, DKC: Double kissing-crush technique; LV: Left ventricle; MCT: Mini-crush technique; MI: Myocardial infarction; NSTEMI: Non-ST segment elevation myocardial infarction; PCI: Percutaneous coronary intervention; USAP: Unstable angina pectoris

Table 2. Lesions characteristics per study group

Parameters	DKC Group (n=218)	MCT Group (n=313)	P value
Multi-vessel disease, n(%)	184 (84.4)	271 (86.6)	0.481
SYNTAX score	27.67±6.54	27.94±6.99	0.792
SYNTAX score ≤22, n(%)	48 (22.0)	61 (19.5)	0.478
SYNTAX score 23-32, n(%)	109 (50.0)	164 (52.4)	0.587
SYNTAX score ≥33, n(%)	61 (28.0)	88 (28.1)	0.973
Left main lesion location, n(%)*			
Ostial	12 (5.5)	9 (2.9)	0.173
Mid-shaft	21 (9.6)	49 (15.7)	**0.044**
Distal	218 (100)	313 (100)	-
Medina classification, n(%)			
0.1.1	55 (25.2)	94 (30.0)	0.226
1.1.1	163 (74.8)	219 (70.0)	0.226
Lesion length, mm			
Main vessel	27.72±6.45	26.31±8.26	**0.045**
Side branch	17.63±7.09	17.75±7.15	0.929
Reference vessel diameter, mm			
Main vessel	4.86±0.20	4.98±2.62	0.096
Side branch	3.09±0.43	3.12±1.55	0.814
Assessment of bifurcation complexity, n(%)			
Calcification ≥ moderate	93 (42.7)	132 (42.2)	0.911
Multiple lesions	152 (69.7)	235 (75.1)	0.172
Thrombus identified by angiography	27 (12.4)	38 (12.1)	0.933
Trifurcation	8 (3.7)	21 (6.7)	0.174
Tortuosity ≥ moderate	25 (11.5)	29 (9.3)	0.409
Bifurcation angle between LAD and Cx (°)	77.70±13.58	77.98±10.91	0.930
Main vessel, n(%)			
TIMI flow grade <3	15 (6.9)	23 (7.3)	0.837
Chronic total occlusion	5 (2.3)	10 (3.2)	0.604
Thrombus-containing lesion	20 (9.2)	22 (7.0)	0.367
Side branch, n(%)			
TIMI flow grade <3	20 (9.2)	22 (7.0)	0.367
Chronic total occlusion	3 (1.4)	3 (1.0)	0.693
Thrombus-containing lesion	20 (9.2)	19 (6.1)	0.177

*Footnote: *"n" denotes the number of left main lesion locations rather than the number of patients with left main lesions. Bold indicates significance level at P <0.05. Abbreviations: DKC: Double kissing-crush technique; LAD: Left anterior descending; LCx: Left circumflex; LMCA: Left main coronary artery; MCT: Mini-crush technique; MV: Main vessel; OM: Obtuse marginal artery; PDA: Posterior descending artery; PL: Posterolateral artery; SB: Side branch; TIMI: Thrombolysis in myocardial infarction*

Results: This large-scale multicenter (n=13) observational study included a total of 531 consecutive patients [men: 405 (76.3%), mean age: 63.16 ± 11.26 years] with complex LMB lesions who underwent PCI. The initial revascularization strategy was MCT in 313 (59%) cases and DKC in 218 (41%) patients. The number of balloons (5.91 ± 1.53 vs. 6.72 ± 1.70, p<0.001) used and procedure (66.60 ± 24.20 vs. 72.97 ± 19.97 min, p<0.001) time were notably lower in the MCT group. In the overall population, the long-term MACE (HR=0.704, p=0.169) and MACCE (HR=0.660, p=0.079) did not differ in individuals with complex LMB lesions treated with MCT and the DKC. Other endpoints were also comparable between the two groups.

Conclusions: In complex LMB lesions, risk-adjusted MACE and MACCE rates were comparable between both techniques, with a non-significant trend favouring DKC at long-term follow-up.

Table 3. Procedural characteristics of the study groups

Parameters	DKC Group (n=218)	MCT Group (n=313)	P value
Access site, n(%)			
Femoral	190 (87.2)	268 (85.6)	0.614
Radial	28 (12.8)	45 (14.4)	0.510
Tirofiban use during PCI, n (%)	16 (7.3)	25 (8.0)	0.783
Utilization of IVUS, n(%)	69 (31.7)	96 (30.7)	0.810
Thrombus Aspiration, n(%)	0 (0.0)	3 (1.0)	0.273
Utilization of IABP, n(%)	5 (2.3)	7 (2.2)	1.00
Pre-dilation			
Main vessel	205 (94.0)	290 (92.7)	0.532
Side branch	205 (94.0)	293 (93.6)	0.841
Final kissing balloon inflation	214 (98.2)	306 (97.8)	0.749
Main vessel	3 (1.4)	6 (1.9)	0.743
Side branch	3 (1.4)	1 (0.3)	0.310
Management of calcification, n (%)			
Performed rotablation	4 (1.8)	6 (1.9)	1.00
Balloon-based strategies	89 (40.8)	126 (40.3)	0.852
Main vessel			
Stent number, n	1.14±0.39	1.16±0.39	0.497
Stent diameter, mm	3.92±0.29	3.86±0.28	**0.007**
Stent length, mm	31.39±8.30	31.20±10.38	0.185
Side branch			
Stent number, n	1.03±0.20	1.02±0.14	0.520
Stent diameter, mm	3.06±0.38	3.03±0.27	0.499
Stent length, mm	22.25±7.38	22.12±6.50	0.836
Drug-eluting stent brand, n(%)			
Xince	51 (23.4)	142 (45.4)	**<0.001**
Promus	47 (21.6)	50 (16.0)	0.101
Firehawk	92 (42.2)	81 (25.9)	**<0.001**
Resolute Onyx	10 (4.6)	15 (4.8)	0.913
Ultimaster Nagomi	6 (2.8)	6 (1.9)	0.563
Biomime	8 (3.7)	10 (3.2)	0.810
Cre8	4 (1.8)	3 (1.0)	0.453
Supraflex	0 (0.0)	3 (1.0)	0.273
Synergy Megatron	0 (0.0)	3 (1.0)	0.273
Proximal side-branch optimization, n (%)	188 (86.2)	272 (86.9)	0.825
Final POT, n (%)	215 (98.6)	310 (99.0)	0.654
Resource utilization, n (%)			
Guiding catheter number	1.08±0.29	1.11±0.42	0.996
Guidewire number	2.84±0.90	2.61±0.77	**0.002**
Balloon number	6.72±1.70	5.91±1.53	**<0.001**
Stent number	2.18±0.46	2.16±0.38	0.949
Any type of non-fatal intraprocedural complications, n(%)	28 (12.8)	33 (10.5)	0.413
Intraprocedural complication, n(%)			
Abrupt occlusion			
Main vessel	3 (1.4)	9 (2.9)	0.376
Side branch	5 (2.3)	5 (1.6)	0.747
TIMI<3			
Main vessel	6 (2.8)	9 (2.9)	1.00
Side branch	11 (5.0)	9 (2.9)	0.247
Dissection			
Main vessel	10 (4.6)	12 (3.8)	0.665
Side branch	11 (5.0)	9 (2.9)	0.247
Thrombus formation			
Main vessel	6 (2.8)	7 (2.2)	0.779
Side branch	5 (2.3)	4 (1.3)	0.498
Coronary Perforation			
Main vessel	0(0.0)	0 (0.0)	-
Side branch	0 (0.0)	1 (0.3)	1.00
Procedure time, min	72.97±19.97	66.60±24.20	**<0.001**
Fluoroscopy time, min	27.08±9.05	24.70±8.02	**0.002**
Contrast media volume (mL)	243.46±74.03	235.16±69.58	0.307
Angiographic success, n(%)			
Main vessel	215 (98.6)	305 (97.4)	0.348
Side branch	215 (98.6)	306 (97.8)	0.473

Bold indicates significance level at P <0.05. Abbreviations: AKI: Acute kidney injury; DKC: Double kissing-crush technique; IABP: Intra-aortic balloon pump; IVUS: Intravascular ultrasound; MCT: Mini-crush technique; PCI: Percutaneous coronary intervention; POT: Proximal optimization technique; TIMI: Thrombolysis in myocardial infarction

Table 4. In-hospital and long-term outcomes per study group

Parameters	DKC Group (n=218)	MCT Group (n=313)	Treatment effect HR (95% CI)	P value
In-hospital complications, n (%)				
Death	4 (1.8)	6 (1.9)		1.00
Major bleeding	6 (2.8)	9 (2.9)		1.00
Pseudoaneurysm	5 (2.3)	10 (3.2)		0.605
Fatal arrhythmias	9 (4.1)	11 (3.5)		0.818
Stent thrombosis	2 (0.9)	3 (1.0)		1.00
Spontaneous MI	4 (1.8)	5 (1.6)		1.00
Contrast-induced AKI	27 (12.4)	28 (9.8)		0.201
Stroke	0 (0.0)	2 (0.6)		0.515
Follow-up time, month	35.56±20.06	35.69±20.82		0.905
Long-term Outcomes, n (%)				
MACE	25 (11.5)	46 (14.7)	0.704 (0.427-1.161)	0.169*
Cardiac death	11 (5.0)	15 (4.8)		0.894
TLR	18 (8.3)	35 (11.2)		0.269
TVMI	15 (6.9)	19 (6.1)		0.707
MACCE	28 (12.8)	56 (17.9)	0.660 (0.416-1.049)	0.079*
All-cause death	14 (6.4)	21 (6.7)		0.896
TVMI	15 (6.9)	19 (6.1)		0.707
TVR	19 (8.7)	36 (11.5)		0.300
Stent thrombosis	4 (1.8)	9 (2.9)		0.573
Stroke	3 (1.4)	5 (1.6)		1.00

*Footnote:*Multivariate Cox regression models-hazard ratio.*

Bold indicates significance level at P <0.05. Abbreviations: AKI: Acute kidney injury; DKC: Double kissing crush technique; HR: Hazard ratio; IPW: Inverse probability-weighted; MACCE: Major adverse cardiovascular and cerebral events; MACE: Major adverse cardiac events; MCT: Mini-crush technique; MI: Myocardial infarction; TLR: Target lesion revascularization: TVMI: Target vessel myocardial infarction; TVR: Target vessel revascularization

Table 5. Univariate and multivariate Cox Regression analysis showing independent predictors of primary endpoint (MACE) with propensity matched data

Parameters	Univariate Level		Multivariate Level	
	HR (95% CI)	P value	HR (95% CI)	P value
Treatment (DKC)	0.758 [0.467-1.232]	0.264	0.704 [0.427-1.161]	0.169
Age	1.007 [0.986-1.028]	0.531	0.996 [0.973-1.020]	0.739
Diabetes mellitus	2.673 [1.618-4.414]	<0.001	2.466 [1.483-4.101]	**0.001**
Chronic kidney disease	2.669 [1.700-4.286]	<0.001	2.452 [1.480-4.064]	**0.001**
Heart failure	2.052 [1.214-3.467]	0.007	0.922 [0.514-1.653]	0.784
Clinical presentation (ACS)	1.646 [1.034-2.621]	0.036	1.691 [1.026-2.787]	**0.039**
SYNTAX score	1.132 [1.090-1.176]	<0.001	1.146 [1.101-1.194]	**<0.001**
Bifurcation classification	0.958 [0.572-1.605]	0.871	1.655 [0.939-2.916]	0.081

Table 6. Univariate and multivariate Cox Regression analysis showing independent predictors of MACCE

Parameters	Univariate Level		Multivariate Level	
	HR (95% CI)	P value	HR (95% CI)	P value
Treatment (DKC)	0.701 [0.446-1.102]	0.124	0.660 [0.416-1.049]	0.079
Age	1.016 [0.996-1.036]	0.126	1.007 [0.986-1.028]	0.535
Diabetes mellitus	2.404 [1.529-3.780]	<0.001	2.188 [1.380-3.469]	**0.001**
Chronic kidney disease	2.499 [1.633-3.824]	<0.001	2.129 [1.349-3.360]	**0.001**
Heart failure	2.108 [1.306-3.404]	0.002	1.022 [0.599-1.744]	0.937
Clinical presentation (ACS)	1.542 [1.007-2.363]	0.047	1.523 [0.966-2.402]	0.070
SYNTAX score	1.107 [1.070-1.145]	<0.001	1.123 [1.082-1.166]	**<0.001**
Bifurcation classification	1.094 [0.690-1.736]	0.702	1.800 [1081-2.996]	**0.024**

Bold indicates significance level at P <0.05. Abbreviations: ACS: Acute coronary syndrome; CI: Confidence Interval; DKC: Double kissing crush; IVUS: Intravascular ultrasound; MACE: Major adverse cardiovascular events; HR: Hazard ratio

OP-022 [Interventional Cardiology / Coronary]

Robotic-assisted coronary artery bypass grafting vs. percutaneous coronary intervention strategies for ostial left anterior descending lesions

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Background and Aim: The comparison of outcomes of robotic-assisted coronary artery bypass grafting (RA-CABG) vs. stenting techniques (ostial or crossover stenting) for ostial left anterior descending (LAD) artery lesions is still lacking. This retrospective study sought to determine the mid-term outcomes of RA-CABG, crossover stenting (CS), and ostial stent implantation (OSI) in patients with ostial LAD disease.

Methods: All cases were divided into 3 groups as follows: RA-CABG (group 1) (n=157), CS (group 2) (n=104), and OSI

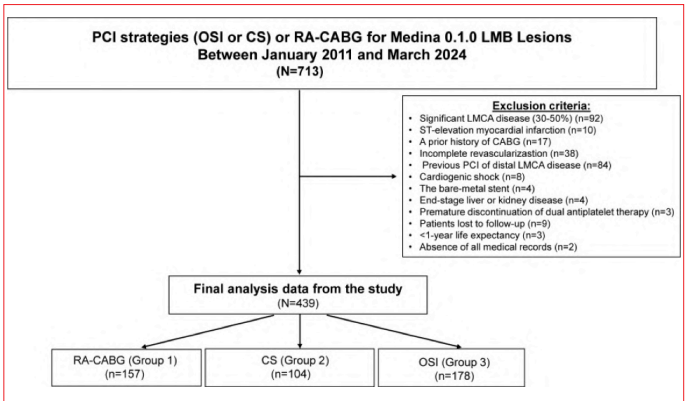

Figure 1. Flow chart for the patient selection. CABG: Coronary artery bypass grafting; CS: Crossover stenting; LAD: Left anterior descendibg artery; LMB: Left main bifurcation; LMCA: Left main coronary artery; OSI: Accurate ostial stent implantation; RA-CABG: Robotic-assisted coronary artery bypass grafting; PCI: Percutaneous coronary intervention.

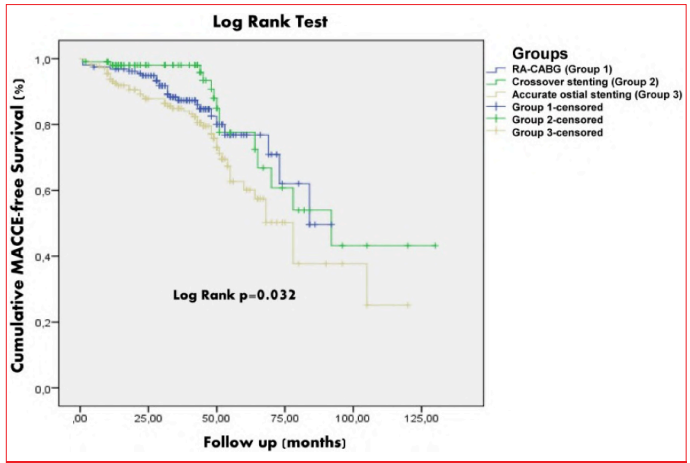

Figure 2. Kaplan-Meier survival analysis for primary endpoint (MACCE) under mid-term follow-up. RA-CABG: Robotic-assisted coronary artery bypass grafting; MACCE: Major adverse cardiovascular and cerebral events.

Table 1. Baseline demographic, clinical, and angiographic characteristics per study group

Variables	RA-CABG (Group1) (n=157)	CS (Group2) (n=104)	OSI (Group3) (n=178)	Pvalue
Age, years	60.50±6.30	59.12±10.87	59.04±10.54	0.108 for 3-group comparison 0.347 for Groups 1 vs 2 **0.023** for Groups 1 vs 3 0.728 for Groups 2 vs 3
Gender, male	122 (77.7)	77 (74.0)	142 (79.8)	0.536 for 3-group comparison 0.495 for Groups 1 vs 2 0.644 for Groups 1 vs 3 0.264 for Groups 2 vs 3
Comorbidities				
Hypertension, n(%)	128 (81.5)	70 (67.3)	112 (62.9)	**0.001** for 3-group comparison **0.009** for Groups 1 vs 2 **0.001** for Groups 1 vs 3 0.458 for Groups 2 vs 3
DM, n(%)	66 (42.0)	47 (45.2)	86 (48.3)	0.515 for 3-group comparison 0.615 for Groups 1 vs 2 0.250 for Groups 1 vs 3 0.612 for Groups 2 vs 3
COPD, n(%)	20 (12.7)	13 (12.5)	19 (10.7)	0.820 for 3-group comparison 0.955 for Groups 1 vs 2 0.557 for Groups 1 vs 3 0.641 for Groups 2 vs 3
CKD, n(%)	32 (20.4)	26 (25.0)	37 (20.8)	0.633 for 3-group comparison 0.380 for Groups 1 vs 2 0.927 for Groups 1 vs 3 0.412 for Groups 2 vs 3
Hyperlipidemia,n(%)	97 (61.8)	65 (62.5)	112 (62.9)	0.977 for 3-group comparison 0.907 for Groups 1 vs 2 0.850 for Groups 1 vs 3 0.944 for Groups 2 vs 3
Smoker, n(%)	69 (43.9)	48 (46.2)	77 (43.3)	0.892 for 3-group comparison 0.726 for Groups 1 vs 2 0.899 for Groups 1 vs 3 0.637 for Groups 2 vs 3
Prior PCI, n(%)	45 (28.7)	23 (22.1)	49 (27.5)	0.475 for 3-group comparison 0.258 for Groups 1 vs 2 0.818 for Groups 1 vs 3 0.315 for Groups 2 vs 3
Prior stroke, n(%)	1 (0.6)	1 (1.0)	4 (2.2)	0.413 for 3-group comparison 0.708 for Groups 1 vs 2 0.225 for Groups 1 vs 3 0.655 for Groups 2 vs 3
Heart failure, n(%)	27 (17.2)	18 (17.3)	35 (19.7)	0.812 for 3-group comparison 0.982 for Groups 1 vs 2 0.562 for Groups 1 vs 3 0.625 for Groups 2 vs 3
LVEF (%)	53.82±8.10	53.75±9.70	53.23±10.97	0.904 for 3-group comparison 0.926 for Groups 1 vs 2 0.947 for Groups 1 vs 3
EuroSCORE II	1.13±.78	1.11±.80	0.99±.61	0.951 for Groups 2 vs 3 **0.005** for 3-group comparison **Bonferroni Group 1=Group 3** 0.756 for Groups 1 vs 2 **0.003** for Groups 1 vs 3 **0.020** for Groups 2 vs 3
Clinical presentation, n(%)				
CCS	86 (54.8)	54 (51.9)	90 (50.6)	0.758 for 3-group comparison 0.651 for Groups 1 vs 2 0.441 for Groups 1 vs 3 0.825 for Groups 2 vs 3
USAP	22 (14.0)	12 (11.5)	21 (11.8)	0.781 for 3-group comparison 0.561 for Groups 1 vs 2 0.543 for Groups 1 vs 3 0.948 for Groups 2 vs 3
NSTEMI	49 (31.2)	38 (36.5)	67 (37.6)	0.440 for 3-group comparison 0.371 for Groups 1 vs 2 0.217 for Groups 1 vs 3 0.851 for Groups 2 vs 3
Medications, n (%)				
Antiplatelet agents	157 (100.0)	104 (100.0)	178 (100.0)	-
Beta Blockers	135 (86.0)	86 (82.7)	154 (86.5)	0.659 for 3-group comparison 0.469 for Groups 1 vs 2 0.888 for Groups 1 vs 3 0.384 for Groups 2 vs 3
CCB	20 (12.7)	14 (13.5)	25 (14.0)	0.941 for 3-group comparison 0.865 for Groups 1 vs 2 0.726 for Groups 1 vs 3 0.891 for Groups 2 vs 3
ACEI/ARB	140 (89.2)	94 (90.4)	161 (90.4)	0.916 for 3-group comparison 0.753 for Groups 1 vs 2 0.699 for Groups 1 vs 3 0.986 for Groups 2 vs 3
Statin	145 (92.4)	95 (89.4)	164 (92.1)	0.653 for 3-group comparison 0.413 for Groups 1 vs 2 0.940 for Groups 1 vs 3 0.440 for Groups 2 vs 3
Diuretics	29 (18.5)	21 (20.2)	40 (22.5)	0.661 for 3-group comparison 0.729 for Groups 1 vs 2 0.366 for Groups 1 vs 3 0.654 for Groups 2 vs 3
Insulin	35 (22.3)	22 (21.2)	42 (23.6)	0.890 for 3-group comparison 0.827 for Groups 1 vs 2 0.777 for Groups 1 vs 3 0.637 for Groups 2 vs 3
Lesion characteristics, n(%)				
Multi-vessel disease	96 (61.1)	63 (60.6)	108 (60.7)	0.994 for 3-group comparison 0.926 for Groups 1 vs 2 0.980 for Groups 1 vs 3 0.987 for Groups 2 vs 3
SYNTAX score	23.54±6.02	23.45±6.24	23.44±6.08	0.968 for 3-group comparison 0.844 for Groups 1 vs 2
SYNTAX score ≤22	77 (49.0)	50 (48.1)	84 (47.2)	0.817 for Groups 1 vs 3 0.903 for Groups 2 vs 3 0.974 for 3-group comparison 0.958 for Groups 1 vs 2 0.824 for Groups 1 vs 3 0.886 for Groups 2 vs 3
SYNTAX score 23-32	62 (39.5)	43 (41.3)	75 (42.1)	0.833 for 3-group comparison 0.765 for Groups 1 vs 2 0.623 for Groups 1 vs 3 0.897 for Groups 2 vs 3
SYNTAX score ≥33	18 (11.5)	11 (10.6)	19 (10.7)	0.998 for 3-group comparison 0.823 for Groups 1 vs 2 0.818 for Groups 1 vs 3 0.980 for Groups 2 vs 3
Severity of ostial LAD stanosis (%)	78.22±8.95	79.29±11.04	78.96±13.20	0.290 for 3-group comparison 0.285 for Groups 1 vs 2 0.802 for Groups 1 vs 3 0.128 for Groups 2 vs 3
LMCA stanosis <30%	28 (17.8)	20 (19.2)	33 (18.5)	0.960 for 3-group comparison 0.776 for Groups 1 vs 2 0.867 for Groups 1 vs 3 0.886 for Groups 2 vs 3

NSTEMI: CCB: Calcium channel blocker; CCS: Chronic coronary syndrome; CKD: Chronic kidney disease; COPD: Chronic obstructive pulmonary disease; CS: Crossover stenting; DM: Diabetes mellitus; LAD: Left anterior descending; LVEF: Left ventricle ejection fraction; LMCA: Left main coronary artery; Non ST-elevation myocardial infarction; OSI: Accurate ostial stent implantation; RA-CABG: Robotic-assisted coronary artery bypass grafting; PCI: Percutaneous coronary intervention

(group 3) (n=178). The primary endpoint was defined as the major adverse cardiac and cerebral events (MACCE), which included cardiac death, target vessel myocardial infarction, target vessel revascularization (TVR), stroke, and stent thrombosisor symptomaticgraftocclusionduringfollow-up.This is thefirst investigation comparing mid-term outcomes of RA-CABG, CS, and OSI as revascularization options for ostial LAD lesions.

Results: A total of 439 consecutive individuals [male: 341 (77.6%), mean age: 59.58 ± 9.35 years] with ostial LAD disease were included in this study. The rates of MACCE (p=0.020 for groups 3 vs. 1; p=0.011 for groups 3 vs. 2) and clinically driven TVR (15.7 vs. 4.5%, p=0.001 for groups 3 vs. 1; 15.7 vs. 5.8%, p=0.014 for groups 3 vs. 2) were notably higher in group 3 than the others. The mid-term MACCE

Table 2. Procedure details of RA-CABG group

Variables	RA-CABG (Group 1) (n=157)
LITA use, n (%)	156 (99.4)
Radial artery graft use, n (%)	9 (5.7)
Single arterial greft use, n (%)	145 (92.4)
Multiarterial CABG, n (%)	11 (7.0)
Saphenous venous grafts, n (%)	72 (45.9)
Number of total arterial greft	1.06 ± 0.27
Number of total greft	2.01 ± 1.02
Conversion to full sternotomy, n (%)	10 (6.4)

CABG: Coronary by-pass grafting; LITA: Left internal thoracic artery; RA-CABG: Robotic-assisted coronary artery bypass grafting

Table 3. Procedural characteristics of the PCI groups

Variables	CS (Group 2) (n=104)	OSI (Group 3) (n=178)	P value
Access site, n (%)			
Femoral	100 (96.2)	169 (94.9)	0.640
Radial	4 (3.8)	9 (5.1)	0.773
IABP support, n (%)	7 (6.7)	4 (2.2)	0.106
Utilization of IVUS, n (%)	32 (30.8)	43 (24.1)	0.613
Stent diameter, mm	3.78 ± 0.26	3.72 ± 0.46	0.514
Stent length, mm	23.75 ± 7.04	23.01 ± 5.14	0.910
Maximum POT balloon diameter, mm	4.70 ± 0.27	3.98 ± 0.29	<0.001
Active SB protection, n (%)	5 (4.8)	0 (0.0)	0.006
SB intervention, n (%)	7 (6.7)	8 (4.5)	0.423
Performed RA, n (%)	3 (2.9)	3 (1.7)	0.673
Performed IVL, n (%)	2 (1.9)	3 (1.7)	1.00
Thrombus Aspiration, n (%)	2 (1.9)	7 (3.9)	0.493
Tirofiban use during PCI, n (%)	8 (7.7)	14 (7.9)	1.00
Contrast media volume (mL)	140.17 ± 67.23	125.78 ± 61.33	0.112
Fluoroscopy time, min	15.04 ± 6.04	15.41 ± 5.07	0.095

Bold indicates significance level at P value < 0.05. CS: Crossover stenting; IABP: Intra-aortic balloon pump; LITA: Left internal thoracic artery; IVL: Intravascular lithotripsy; IVUS: Intravascular ultrasound; LCx: Left circumflex; OSI: Accurate ostial stent implantation; POT: Proximal optimization technique; RA: Rotational atherectomy; SB: Side branch.

Table 4. In-hospital and mid-term clinical outcomes per study group

Variables	RA-CABG (Group 1) (n=157)	CS (Group 2) (n=104)	OSI (Group 3) (n=178)	P-value
In-hospital outcomes, n (%)				
All-cause-death	1 (0.6)	1 (1.0)	2 (1.1)	1.00 for 3-group comparison 1.00 for Groups 1 vs 2 1.00 for Groups 1 vs 3 1.00 for Groups 2 vs 3
ST or SGO	1 (0.6)	1 (1.0)	1 (0.6)	0.872 for 3-group comparison 1.00 for Groups 1 vs 2 1.00 for Groups 1 vs 3 1.00 for Groups 2 vs 3
TVMI	4 (2.5)	1 (1.0)	2 (1.1)	0.490 for 3-group comparison 0.651 for Groups 1 vs 2 0.425 for Groups 1 vs 3 1.00 for Groups 2 vs 3
Stroke	3 (1.9)	0 (0.0)	2 (1.1)	0.363 for 3-group comparison 0.278 for Groups 1 vs 2 0.668 for Groups 1 vs 3 0.533 for Groups 2 vs 3
AKI requiring RRT	3 (1.9)	2 (1.9)	3 (1.7)	0.984 for 3-group comparison 1.00 for Groups 1 vs 2 1.00 for Groups 1 vs 3 1.00 for Groups 2 vs 3
Prolonged endotracheal intubation (>24 h)	1 (0.6)	2 (1.9)	3 (1.7)	0.609 for 3-group comparison 0.565 for Groups 1 vs 2 0.626 for Groups 1 vs 3 1.00 for Groups 2 vs 3
Pneumothorax	7 (4.5)	0 (0.0)	0 (0.0)	-
Superficial wound infections	2 (1.3)	1 (1.0)	0 (0.0)	0.341 for 3-group comparison 1.00 for Groups 1 vs 2 0.219 for Groups 1 vs 3 0.369 for Groups 2 vs 3
TIMI-major bleeding	6 (3.8)	4 (3.8)	5 (2.8)	0.846 for 3-group comparison 1.00 for Groups 1 vs 2 0.761 for Groups 1 vs 3 0.730 for Groups 2 vs 3
Non-fatal MAE	16 (10.2)	7 (6.7)	11 (6.2)	0.354 for 3-group comparison 1.00 for Groups 1 vs 2 1.00 for Groups 1 vs 3 1.00 for Groups 2 vs 3
Hospital LOS, days	5.06±1.36	2.20±2.24	2.27±1.70	**<0.001** for 3-group comparison **Bonferroni: Group 1>Group 2 and Group 1>Group 3** **<0.001** for Groups 1 vs 2 **<0.001** for Groups 1 vs 3 **<0.001** for Groups 2 vs 3
Follow-up time, months	39.88±16.76	38.81±25.17	39.35±21.71	0.677 for 3-group comparison 0.297 for Groups 1 vs 2 0.940 for Groups 1 vs 3 0.573 for Groups 2 vs 3
Mid-term outcomes, n (%)				
MACCE	25 (15.9)	14 (13.5)	47 (26.4)	**0.011** for 3-group comparison 0.585 for Groups 1 vs 2 **0.020** for Groups 1 vs 3 **0.011** for Groups 2 vs 3
Cardiac death	12 (7.6)	4 (3.8)	17 (9.6)	0.215 for 3-group comparison 0.294 for Groups 1 vs 2 0.536 for Groups 1 vs 3 0.100 for Groups 2 vs 3
TVMI	13 (8.3)	5 (4.8)	18 (10.1)	0.293 for 3-group comparison 0.327 for Groups 1 vs 2 0.578 for Groups 1 vs 3 0.175 for Groups 2 vs 3
Clinically driven TVR	7 (4.5)	6 (5.8)	28 (15.7)	**0.001** for 3-group comparison 0.773 for Groups 1 vs 2 **0.001** for Groups 1 vs 3 **0.014** for Groups 2 vs 3
ST or SGO	2 (1.3)	2 (1.9)	6 (3.4)	0.843 for 3-group comparison 0.624 for Groups 1 vs 2 0.878 for Groups 1 vs 3 0.714 for Groups 2 vs 3
Stroke	1 (0.6)	1 (1.0)	4 (2.2)	0.413 for 3-group comparison 1.00 for Groups 1 vs 2 0.376 for Groups 1 vs 3 0.655 for Groups 2 vs 3
All-cause death	14 (8.9)	6 (5.8)	22 (12.4)	0.181 for 3-group comparison 0.477 for Groups 1 vs 2 0.310 for Groups 1 vs 3 0.098 for Groups 2 vs 3

Bold indicates significance level at p value <0.05. AKI: Acute kidney injury; CS: Crossover stenting; LOS: Length of stay; MACCE: Major adverse cardiovascular and cerebral event; MAE: Major adverse events; MI: Myocardial infarction; OSI: Accurate ostial stent implantation; Robotic-assisted coronary artery bypass grafting; RRT: Renal replacement therapy; ST: Stent thrombosis; SGO: Symptomatic graft occlusion; TVMI: Target vessel myocardial infarction; TVR: Target vessel revascularization

[(adjusted hazard ratio=2.129 [95% confidence interval: 1.360-3.334], p=0.001)] in the overall population significantly differed between group 3 and the others.

Conclusions: The findings of the study suggest that OSI for ostial LAD lesions was associated with higher mid-term MACCE and TVR rates than revascularization with RA-CABG or CS.

OP-023 [Interventional Cardiology / Coronary]

Comparison of different side branch ballooning following provisional stenting in coronary bifurcation lesion-related STEMI

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Background and Aim: This investigation aimed to retrospectively assess clinical outcomes of proximal optimization technique (POT)-kissing-POT (PKP) and POT-side-POT (PSP) in ST-segment elevation myocardial infarction (STEMI) patients with culprit coronary bifurcation lesion (CBL) following a provisional stenting (PS).

Methods: This large-scale multicenter (n=10) study included STEMI patients with culprit CBLs who underwent PKP or PSP following PS. The primary endpoint was defined as the major adverse cardiac events (MACE) [cardiac death, target vessel myocardial infarction (TVMI), or clinically driven target lesion revascularization (TLR)].

Table 1. Baseline demographic characteristics of per study group

Variables	PKP Group (n=386)	PSP Group (n=210)	P value
Age (years)	57.64±11.28	59.07±12.51	0.168
Male, sex n (%)	318 (82.4)	173 (82.4)	0.999
Comorbidities, n(%)			
Hypertension	193 (50.0)	123 (58.6)	**0.045**
Diabetes mellitus	117 (30.3)	69 (32.9)	0.522
Hyperlipidemia	115 (29.8)	75 (35.7)	0.138
Chronic kidney disease	17 (4.4)	17 (8.1)	0.063
Current Smoker	223 (57.8)	123 (58.6)	0.386
History of Stroke	10 (2.6)	3 (1.4)	0.558
Peripheral artery disease	18 (4.7)	9 (4.3)	1.00
Prior PCI	87 (22.5)	41 (19.5)	0.392
Heart Failure	97 (25.1)	60 (28.6)	0.362
LV Ejection Fraction (%)	47.23±9.70	46.51±9.58	0.405
Moderate-severe valve disease, n (%)	31 (8.0)	24 (11.4)	0.171
Laboratory measurements			
White blood cell count, (10^9/L)	11.52±4.07	12.83±10.42	0.229
Hemoglobin, (g/dL)	14.22±7.34	14.00±1.74	0.551
Creatinine, (mg/dL)	0.93±.28	1.02±.43	**0.008**
Platelet count, (10^9/L)	266.40±83.66	253.40±70.68	0.129
Total cholesterol, (mg/dL)	178.43±62.08	182.37±51.75	0.251
Troponin Peak (ng/L)	5714.90±3210.02	5777.86±2846.81	0.921
Medications Used, n (%)			
Acetylsalicylic acid	386 (100.0)	210 (100.0)	-
Clopidogrel	138 (35.8)	78 (37.1)	0.736
Ticagrelor	205 (53.1)	116 (55.2)	0.618
Prasugrel	43 (11.1)	16 (7.6)	0.169
Beta Blockers	376 (97.4)	198 (94.3)	0.053
ACEI/ARB	370 (95.9)	200 (95.2)	0.725
Statin	383 (99.2)	208 (99.0)	0.823
Diuretics	98 (25.4)	62 (29.5)	0.276
Insulin	73 (18.9)	50 (23.8)	0.158

Bold indicates significance level at p<0.05. ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, LV: Left ventricle; PCI: Percutaneous coronary intervention; PKP: POT-kissing-POT; PSP: POT-side-POT.

Table 2. Lesions characteristics per study group

Variables	PKP Group (n=386)	PSP Group (n=210)	P value
SYNTAX score	20.48±7.52	20.68±8.27	0.898
SYNTAX score ≤22, n (%)	245 (63.5)	128 (61.0)	0.544
SYNTAX score 23-32, n (%)	109 (28.2)	58 (27.6)	0.872
SYNTAX score ≥33, n (%)	31 (8.0)	24 (11.4)	0.171
Culprit bifurcation localization, n (%)			
LMCA (LAD-LCX)	35 (9.1)	20 (9.5)	0.854
LAD-Diagonal	222 (57.5)	117 (55.7)	0.672
LCx-OM	98 (25.4)	48 (22.9)	0.550
PDA-PL	31 (8.0)	25 (11.9)	0.122
Medina classification, n (%)			
0.0.1	3 (0.8)	0 (0.0)	0.556
1.0.0	37 (9.6)	13 (6.2)	0.153
1.1.0	79 (20.5)	38 (18.1)	0.486
0.1.0	74 (19.2)	32 (15.2)	0.230
1.0.1	23 (6.0)	16 (7.6)	0.434
0.1.1	72 (18.7)	34 (16.2)	0.453
1.1.1	101 (26.2)	67 (31.9)	0.137
True bifurcation, n (%)	194 (50.3)	117 (55.7)	0.203
Moderate or severe calcification, n (%)	22 (5.7)	16 (7.6)	0.360
Multiple lesions, n (%)	144 (37.3)	76 (36.2)	0.788
Main vessel reference diameter < 2.5mm, n (%)	10 (2.6)	5 (2.4)	1.00
High thrombus burden, n (%)	60 (15.5)	34 (16.2)	0.836
Bifurcation angle <45° or >70°, n (%)	170 (44.0)	91 (43.3)	0.868
Lesion length, mm			
Main vessel	20.35±7.46	20.98±7.40	0.272
Side branch	9.70±4.35	8.97±4.13	0.157
Moderate or severe calcification, n (%)	22 (5.7)	16 (7.6)	0.360
Reference vessel diameter, mm			
Main vessel	3.11±0.74	3.07±0.42	0.613
Side branch	2.62±1.66	2.64±2.22	0.457
Preprocedural TIMI flow grade			
Main vessel	0.84±1.15	0.82±1.13	0.995
Side branch	1.29±1.28	1.27±1.29	0.984

LAD: Left anterior descending; LCx: Left circumflex; OM: Obtuse marginal artery; PDA: Posterior descending artery; PL: Posterolateral artery; PKP: POT-kissing-POT; PSP: POT-side-POT; SB: Side branch; TIMI: Thrombolysis in myocardial infarction.

Table 3. Procedural characteristics of the study groups

Variables	PKP Group (n=386)	PSP Group (n=210)	P value
Access site, n (%)			
Femoral	381 (98.7)	208 (99.0)	0.710
Radial	5 (1.3)	2 (1.0)	1.00
Tirofiban use during PCI, n (%)	59 (15.3)	35 (16.7)	0.658
Thrombus Aspiration, n (%)	20 (5.2)	7 (3.3)	0.410
Performed IVUS	34 (8.8)	19 (9.0)	0.922
Side branch dissection (> type B), n (%)	18 (4.7)	12 (5.7)	0.575
Main vessel			
Stent diameter, mm	3.08±1.33	3.03±.34	0.627
Stent length, mm	24.51±7.59	24.98±6.78	0.191
Bail-out 2-stent, n (%)	45 (11.7)	31 (14.8)	0.278
TAP	30 (7.8)	19 (9.0)	0.588
Culotte	15 (3.9)	12 (5.7)	0.305
Postprocedural TIMI<3 flow grade			
Main vessel	10 (2.6)	6 (2.9)	0.798
Side branch	28 (7.3)	15 (7.1)	0.960
Side branch residual stenosis, n (%)			
70%-90%	22 (5.6)	4 (1.9)	**0.035**
50%-70%	50 (13.0)	20 (9.5)	0.214
30%-50%	112 (29.0)	75 (35.7)	0.092
<30%	202 (52.3)	111 (52.9)	0.902
Procedure time, min	44.86±13.17	42.04±13.41	**0.026**
Fluoroscopy time, min	14.54±7.29	13.78±5.94	0.274
Contrast media volume (mL)	176.13±64.63	170.88±59.84	0.616
Angiographic success, n (%)			
Main vessel	376 (97.4)	204 (97.1)	0.848
Side branch	195 (50.5)	111 (52.9)	0.585

Bold indicates significance level at P <0.05. **Abbreviations:** PCI: Percutaneous coronary intervention; PKP: POT-kissing-POT; PSP: POT-side-POT; POT: Proximal optimization technique; TAP: T and minimal protrusion; TIMI: Thrombolysis in myocardial infarction

Table 4. In-hospital and long-term outcomes per study group

Variables	PKP Group (n=386)	PSP Group (n=210)	P value
In-hospital complications, n (%)			
Death	6 (1.6)	4 (1.9)	0.747
Major bleeding	8 (2.1)	5 (2.4)	0.777
Spontaneous MI	6 (1.6)	4 (1.9)	0.747
Stent thrombosis	2 (0.5)	2 (1.0)	0.616
Contrast-induced AKI	23 (6.0)	12 (5.7)	0.904
Follow-up time, months	25 (16.0-39.0)	25 (16.0-40.0)	0.716
Mid-term Outcomes, n (%)			
Primary endpoint (MACE)	47 (12.2)	37 (17.6)	0.068
Cardiac death	16 (4.1)	12 (5.7)	0.387
TLR			
Side branch	20 (5.2)	13 (6.2)	0.607
Main vessel	14 (3.6)	15 (7.1)	0.057
TVMI			
Side branch	19 (4.9)	10 (4.8)	0.931
Main vessel	11 (2.8)	13 (6.2)	**0.047**
Stent thrombosis	6 (1.6)	8 (3.8)	0.094
All-cause death	18 (4.7)	13 (6.2)	0.422

Bold indicates significance level at P <0.05. **Abbreviations:** AKI: Acute kidney injury; MACE: Major adverse cardiac events; MI: Myocardial infarction; PKP: POT-kissing-POT; PSP: POT-side-POT; TLR: Target lesion revascularization: TVMI: Target vessel myocardial infarction

Results: A total of 596 consecutive patients [male: 491 (82.3%), mean age: 58.14 ± 11.71 years] were included. The study cohort was divided into two groups as PKP (n=386) and PSP (n=210). In the overall population, mid-term MACE (hazard ratio [HR]=0.921, p=0.461) did not differ in individuals with CBL-related STEMI treated with either PKP or PSP. The frequency of main vessel-TLR (0 vs. 30%, p=0.001) and main vessel-TVMI (0 vs. 20%, p=0.014) were significantly lower in the PKP group in the left main bifurcation localization. Diabetes mellitus (HR=2.628, p<0.001), high SYNTAX score (HR=1.081, p<0.001), and bifurcation localization (HR=2.109, p=0.014) were found to be independent predictors of MACE.

Conclusions: In the overall population, risk-adjusted MACE rates for culprit CBLs were comparable between both techniques.

Table 5. Univariate and multivariate Cox regression analysis showing independent predictors of MACE

Parameters	Univariate Level		Multivariate Level	
	HR (95% CI)	P value	HR (95% CI)	P value
Treatment (PKP)	0.830 [0.669-1.030]	0.090	0.921 [0.739-1.147]	0.461
Diabetes mellitus	3.911 [2.521-6.067]	<0.001	2.628 [1.643-4.203]	**<0.001**
Chronic kidney disease	2.186 [1.091-4.380]	0.027	1.050 [0.504-2.189]	0.897
LV ejection fraction	0.963 [0.942-0.984]	0.001	0.977 [0.954-1.001]	0.055
Main vessel stent diameter	0.956 [0.727-1.257]	0.747	0.830 [0.528-1.304]	0.419
True bifurcation classification	1.821 [0.964-3.440]	0.065	1.745 [0.906-3.361]	0.096
SYNTAX Score	1.103 [1.079-1.126]	<0.001	1.081 [1.055-1.107]	**<0.001**
Bifurcation Localization (LMCA)	2.470 [1.412-4.321]	0.002	2.109 [1.161-3.830]	**0.014**
Bail-out 2-stent	2.678 [1.655-4.334]	<0.001	1.567 [0.949-2.588]	0.079

Bold indicates significance level at P <0.05. **Abbreviations:** CI: Confidence Interval; LMCA: Left main coronary artery; LV: Left ventricle; MACE: Major adverse cardiovascular events; HR: Hazard ratio

OP-024 [Interventional Cardiology / Coronary]

Active side-branch protection strategy for culprit bifurcation lesion in STEMI patients: The multicenter STEMI-BIF registry

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Background and Aim: To date, the best side branch (SB) protection strategy in patients with coronary bifurcation lesions (CBLs)-related ST-segment elevation myocardial infarction (STEMI) has not yet

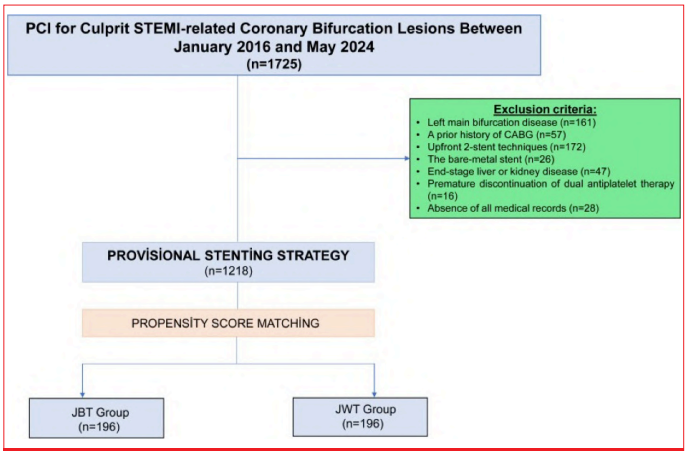

Figure 1. Study flowchart. CABG: Coronary bypass grafting; JBT: Jailed balloon technique; JWT: Jailed wire technique; PCI:Percutaneous coronary intervention; STEMI: ST-segment elevation myocardial infarction.

been settled. This study sought to evaluate the clinical outcome of the jailed balloon (JBT) and jailed wire techniques (JWT) for the SB protection strategy in STEMI patients with culprit CBLs.

Methods: This large-scale multicenter (n=10) observational retrospective study included STEMI patients with culprit CBLs who underwent PCI with provisional stenting. The primary endpoint was major adverse cardiac events (MACE) as the combination of death from cardiac causes, target vessel myocardial infarction, or clinically driven target lesion revascularization (TLR). Propensity score-matching analysis was performed.

Results: A total of 1218 consecutive patients [male: 1020 (83.7%), mean age: 57.68 ± 11.76 years] were included in this study. The study cohort was divided into 2 groups as JBT (n=196) and JWT (n=1022). The incidences of the SB intervention (21.4 vs. 36.2%, p<0.001) and residual stenosis of SB ≥70% (23.0 vs. 45.5%, p<0.001) were significantly lower in the JBT group compared to the JWT group. Whereas procedure time (47.21 ± 17.70 vs. 40.94 ± 13.18 min, p<0.001) and fluoroscopy time (15.92 ± 9.51 vs. 13.39 ± 6.69 min, p=0.001) were notably higher in the JBT group than in the JWT group. The risk-adjusted mid-term MACE (HR=0.688, p=0.200) and clinally driven TLR (HR=0.566, p=0.170) did not differ in individuals with culprit CBLs to protect the SB with JBT and the JWT in the propensity-matched cohort.

Conclusıons: The present study suggests that risk-adjusted MACE and TLR rates were comparable between both techniques at mid-term follow-up.

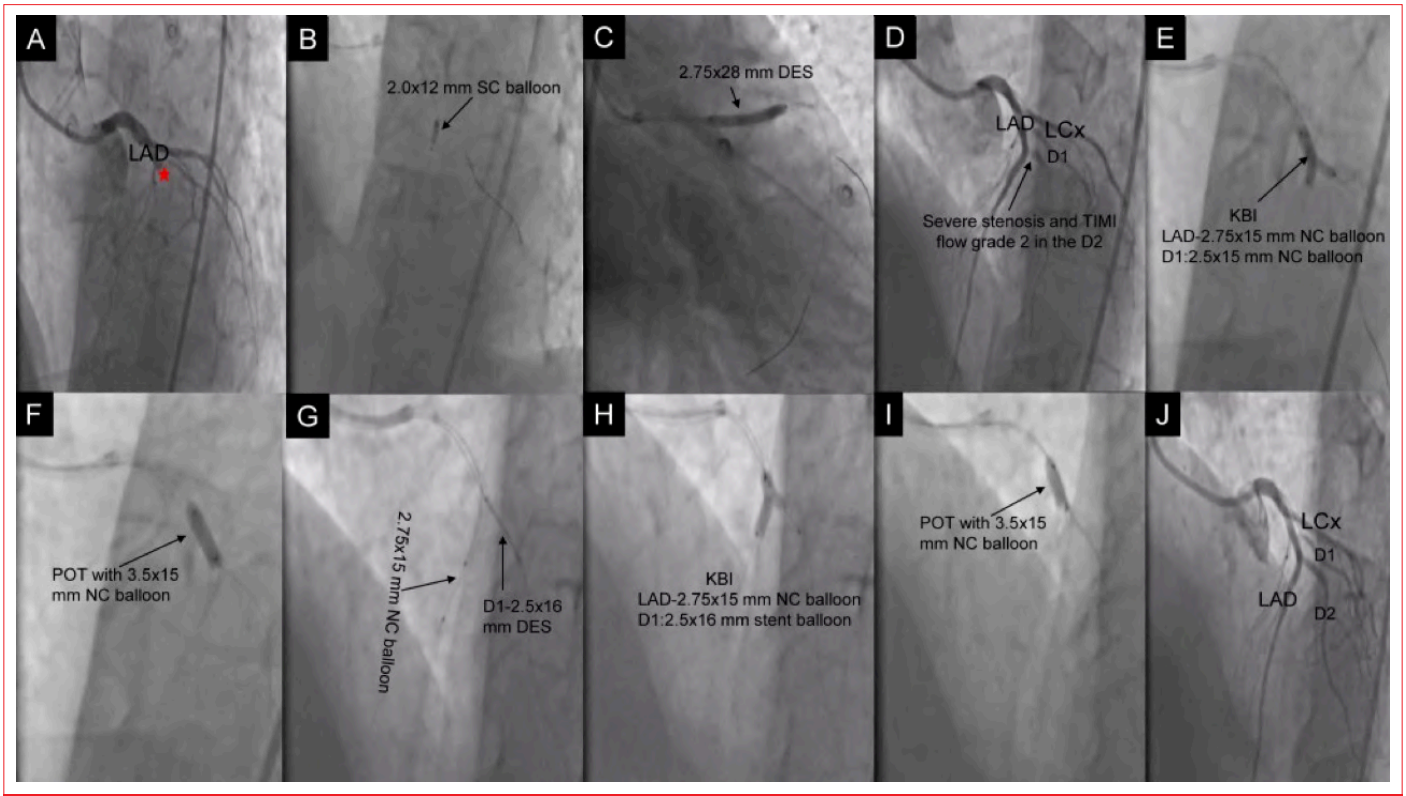

Figure 2. Technical steps of JWT for SB protection via transfemoral access. A) Bifurcation localization in the LAD-D1 arteries with TIMI flow grade 0. B) Wiring and pre-dilatation of LAD and D1 arteries. C) A 2.75x28 mm DES implantation in the LAD and performing POT. D) Critical stenosis (≥90%) and TIMI flow grade 2 in the D1 artery. E, F) KBI with NC balloons and re-POT. G) SB stenting with bail-out TAP technique owing to a major dissection and worsening TIMI flow grade.H, I) KBI and final POT. J) Final angiographic result. DES: Drug-eluting stent; D1: The first diagonal artery; JWT: Jailed wire technique; KBI: Kissing ballon inflation; LAD: Left anterior descending artery; NC: Non-compliant; POT: Proximal optimization technique; SB: Side branch; TAP: T and small protrusion; TIMI: Thrombolysis in myocardial infarction.

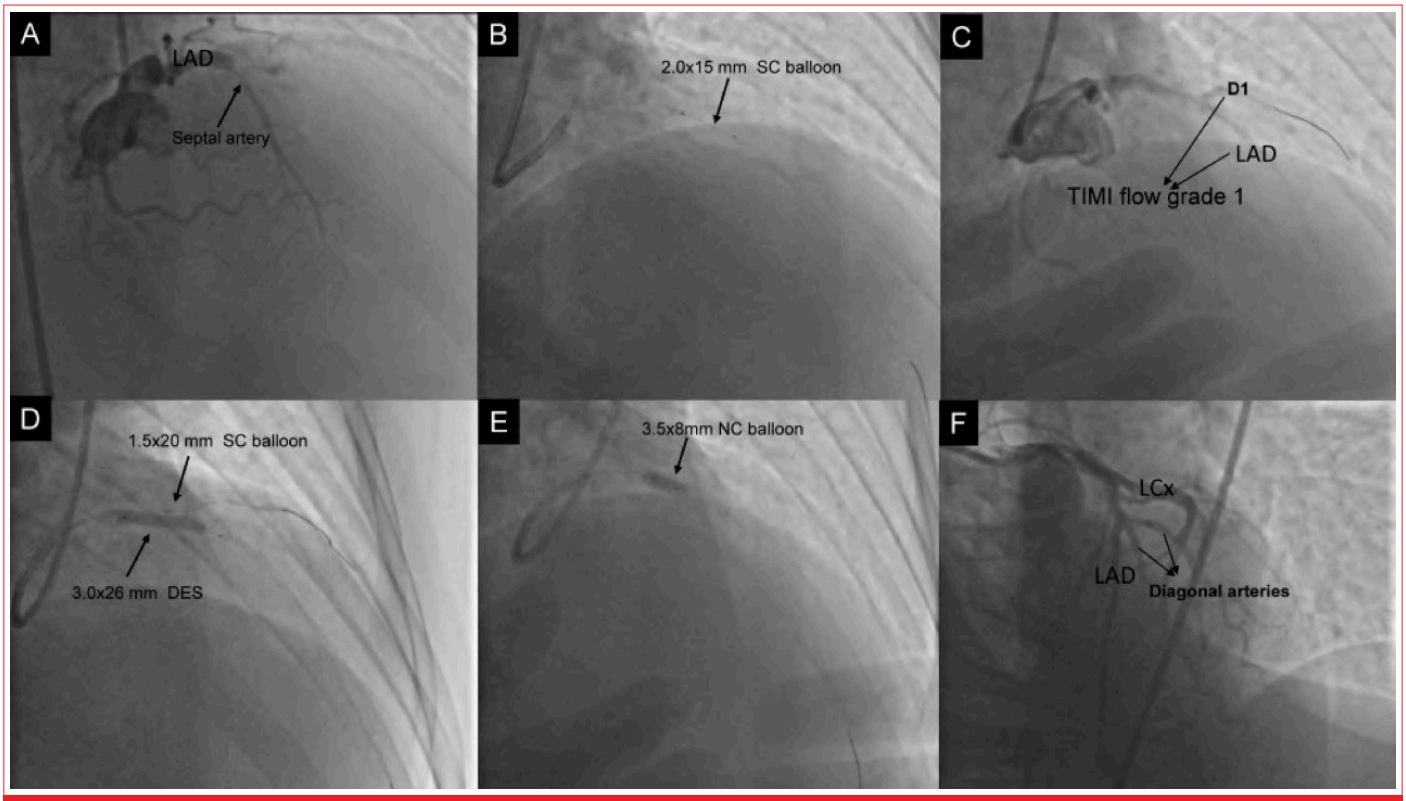

Figure 3. Step-by-step JBT for SB protection via transfemoral approach. A) Bifurcation localization in the LAD-D1 arteries with TIMI flow grade 0. B, C) Wiring and pre-dilatation of LAD and D1 arteries. D) A 1.5x20 mm SC balloon positioned in the LAD bifurcation and placement of the DES with provisional approach in the LAD. F) POT with a 3.5x8 mm NC balloon. Final result. DES: Drug-eluting stent; D1: The first diagonal artery; JBT: Jailed balloon technique; LAD: Left anterior descending artery; NC: Non-compliant; POT: Proximal optimization technique; SB: Side branch; SC: Semi-compliant; TIMI: Thrombolysis in myocardial infarction.

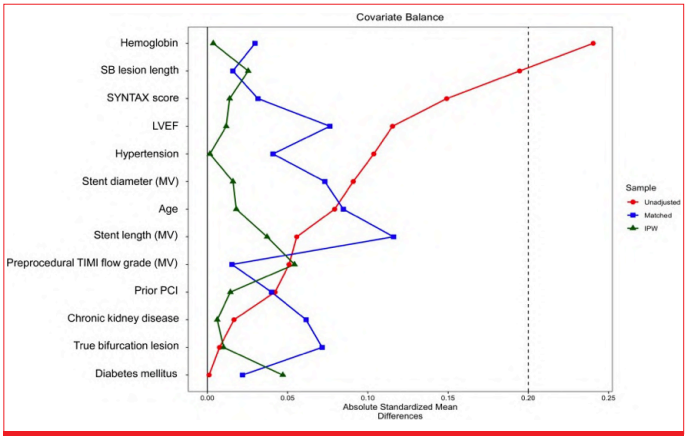

Figure 4. The standardized variable differences Plot (Covariate balance before and after propensity score matching). LVEF: Left ventricle ejection fraction; MV: Main vessel; PCI: Percutaneous coronary intervention; SB: Side branch; TIMI: Thrombolysis in myocardial infarction.

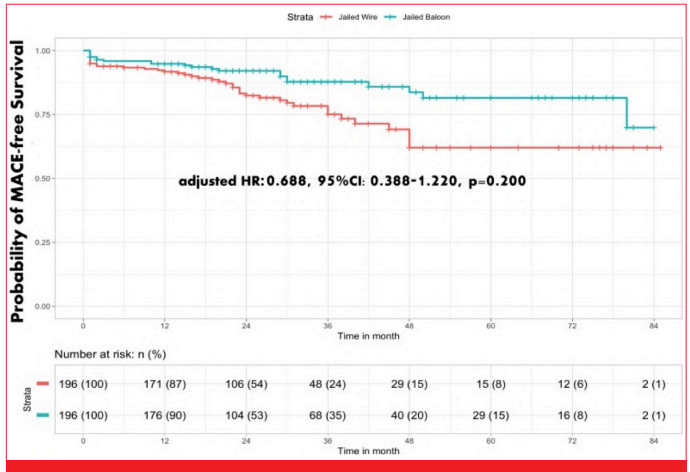

Figure 5. Kaplan-Meier curves after propensity-matched data for the primary endpoint according to the side-branch protection strategy of jailed balloon and jailed wire techniques.

Table 1. Baseline demographic characteristics of before and after PSM per study group

	Overall population			Propensity score-matched population		
Variables	**JBT Group (n=196)**	**JWT Group (n=1022)**	**P**	**JBT Group (n=196)**	**JWT Group (n=196)**	**P**
Age (years)	58.48±12.05	57.52±11.71	0.298	58.48±12.05	59.50±11.69	0.395
Male, sex n (%)	163 (83.2)	857 (83.9)	0.831	163 (83.2)	152 (77.6)	0.162
Comorbidities, n(%)						
Hypertension	103 (52.6)	590 (57.7)	0.180	103 (52.6)	99 (50.5)	0.686
Diabetes mellitus	63 (32.1)	328 (32.1)	0.989	63 (32.1)	65 (33.2)	0.829
Hyperlipidemia	83 (42.3)	448 (43.9)	0.692	83 (42.3)	84 (41.3)	0.838
CKD	13 (6.6)	72 (7.0)	0.836	13 (6.6)	16 (8.2)	0.563
Current Smoker	127 (64.8)	641 (62.7)	0.787	127 (64.8)	119 (60.7)	0.403
History of Stroke	3 (1.5)	18 (1.8)	1.00	3 (1.5)	4 (2.0)	0.703
PAD	8 (4.1)	36 (3.5)	0.678	8 (4.1)	7 (3.6)	0.792
Prior PCI	35 (17.9)	166 (16.2)	0.577	35 (17.9)	38 (19.4)	0.697
≥moderate HVD	20 (10.2)	78 (7.6)	0.225	20 (10.2)	17 (8.7)	0.604
LVEF (%)	46.76±9.22	45.70±9.29	0.100	46.76±9.22	46.06±9.75	0.463
Laboratory measurements						
WBC count, (10^3/L)	11.39±4.12	11.96±3.78	**0.005**	11.39±4.12	11.71±3.84	0.420
Hemoglobin, (g/dL)	13.82±2.03	15.21±10.61	**<0.001**	13.82±2.03	13.89±1.95	0.760
Creatinine, (mg/dL)	0.92±0.40	0.94±0.29	0.436	1.02±0.40	1.06±.98	0.546
Platelet count, (10^3/L)	259.24±67.48	260.49±69.61	0.855	259.24±67.48	260.64±80.38	0.852
Total cholesterol, (mg/dL)	181.61±52.24	181.26±46.34	0.672	181.61±52.24	177.70±49.81	0.449
Troponin Peak (ng/L)	5741.36±3101.56	5849.31±3382.21	0.753	5741.36±3101.56	6011.44±3183.31	0.395
Medications, n (%)						
Acetylsalicylic acid	196 (100.0)	1022 (100.0)	-	196 (100.0)	196 (100)	-
Clopidogrel	58 (29.6)	286 (28.0)	0.647	58 (29.6)	57 (29.1)	0.912
Ticagrelor	128 (65.3)	666 (65.2)	0.970	128 (65.3)	125 (63.8)	0.751
Prasugrel	10 (5.1)	70 (6.8)	0.366	10 (5.1)	14 (7.1)	0.399
Beta Blockers	189 (96.4)	990 (96.9)	0.748	189 (96.4)	192 (98.0)	0.359
ACEI/ARB	190 (96.9)	984 (96.3)	0.652	190 (96.9)	189 (96.4)	0.778
Statin	194 (99.0)	1010 (98.9)	0.943	194 (99.0)	194 (99.0)	1.00
Diuretics	55 (28.1)	325 (31.8)	0.301	55 (28.1)	63 (32.1)	0.378
Insulin	44 (22.4)	225 (22.0)	0.893	44 (22.4)	38 (19.4)	0.456

Bold indicates significance level at P <0.05. Abbreviations: ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, CKD: Chronic kidney disease, JBT: Jailed balloon technique, JWT: Jailed wire technique, HVD: Heart valve disease, LVEF: Left ventricle ejection fraction, PAD: Peripheral artery disease, PCI: Percutaneous coronary intervention, PSM: Propensity score matching, WBC: White blood cell.

Table 2. Lesions characteristics before and after PSM

	Overall population			Propensity score-matched population		
Parameters	**JBT Group (n=196)**	**JWT Group (n=1022)**	**P**	**JBT Group (n=196)**	**JWT Group (n=196)**	**P**
SYNTAX score	19.48±6.33	20.43±7.06	0.125	19.48±6.33	19.28±6.73	0.762
SYNTAX score ≤22, n(%)	136 (69.4)	674 (65.9)	0.350	136 (69.4)	138 (70.4)	0.826
SYNTAX score 23-32, n(%)	53 (27.0)	273 (26.7)	0.924	53 (27.0)	48 (24.5)	0.564
SYNTAX score ≥33, n(%)	7 (3.6)	75 (7.3)	0.061	7 (3.6)	10 (5.1)	0.621
Locations of culpirit bifurcation lesions, n(%)						
LAD-Diagonal	127 (64.8)	677 (66.2)	0.695	127 (64.8)	135 (68.9)	0.391
LCx-OM	47 (24.0)	241 (23.6)	0.904	47 (24.0)	39 (19.9)	0.329
PDA-PL	22 (11.2)	104 (10.2)	0.659	22 (11.2)	22 (11.2)	1.00
Medina classification, n(%)						
0.0.1	2 (1.0)	14 (1.4)	1.000	2 (1.0)	2 (1.0)	1.00
0.1.0	34 (17.3)	163 (15.9)	0.507	34 (17.3)	35 (17.9)	0.894
1.0.0	28 (14.3)	143 (14.0)	0.914	28 (14.3)	21 (10.7)	0.285
1.1.0	38 (19.4)	208 (20.4)	0.885	38 (19.4)	51 (26.0)	0.117
1.0.1	19 (9.7)	132 (12.9)	0.210	19 (9.7)	20 (10.2)	0.866
0.1.1	8 (4.1)	37 (3.7)	0.683	8 (4.1)	9 (4.6)	1.000
1.1.1	67 (34.2)	325 (32.0)	0.549	67 (34.2)	58 (29.6)	0.329
True bifurcation, n(%)	94 (48.0)	494 (48.3)	0.923	94 (48.0)	87 (44.4)	0.478
Lesion length, mm						
MV	19.43±5.46	18.81±5.73	0.168	19.43±5.46	18.74±5.62	0.217
SB	7.50±2.91	6.38±3.04	**0.001**	7.50±2.91	7.15±3.18	0.881
Reference vessel diameter, mm						
MV	3.04±0.33	3.81±12.31	0.657	3.04±0.33	3.10±.33	0.061
SB	2.39±0.31	2.36±0.29	0.141	2.39±0.31	2.40±.32	0.868
Preprocedural TIMI flow grade						
MV	1.18±1.17	0.94±1.10	**0.008**	1.18±1.17	1.08±1.24	0.382
SB	1.44±1.24	1.32±1.25	0.235	1.44±1.24	1.48±1.27	0.747

Bold indicates significance level at P <0.05. Abbreviations: JBT: Jailed balloon technique; JWT: Jailed wire technique; LAD: Left anterior descending; LCx: Left circumflex; MV: Main vessel; OM: Obtuse marginal artery; PDA: Posterior descending artery; PL: Posterolateral artery; PSM: Propensity score matching, SB: Side branch; SYNTAX: Synergy between Percutaneous Coronary Intervention with Taxus and Cardiac Surgery; TIMI: Thrombolysis in myocardial infarction.

Table 3. Procedural characteristics before and after PSM

	Overall population			Propensity score-matched population		
Parameters	**JBT Group (n=196)**	**JWT Group (n=1022)**	**P**	**JBT Group (n=196)**	**JWT Group (n=196)**	**P**
Access site, n(%)						
Femoral	192 (98.0)	1002 (98.0)	0.938	192 (98.0)	191 (97.4)	0.736
Radial	4 (2.0)	20 (2.0)	1.00	4 (2.0)	5 (2.6)	0.736
Tirofiban use during PCI, n (%)	44 (22.4)	215 (21.0)	0.658	44 (22.4)	48 (24.5)	0.634
Utilization of IVUS, n (%)	20 (10.2)	90 (8.8)	0.532	20 (10.2)	22 (11.2)	0.744
Thrombus Aspiration, n(%)	7 (3.6)	40 (3.9)	1.00	7 (3.6)	10 (5.1)	0.457
MV						
Stent diameter, mm	2.97±0.30	2.94±0.36	0.133	2.97±0.30	3.00±36	0.512
Stent length, mm	24.67±5.27	24.37±6.31	0.162	24.67±5.27	24.06±6.08	0.288
SB Intervention, n(%)	42 (21.4)	370 (36.2)	**<0.001**	42 (21.4)	85 (43.4)	**<0.001**
POT-side-POT	13 (6.6)	87 (8.5)	0.380	13 (6.6)	17 (8.7)	0.447
POT-kissing-POT	29 (14.8)	290 (28.4)	**<0.001**	29 (14.8)	70 (35.7)	**<0.001**
Bail-out 2-stent	13 (6.6)	133 (13.0)	**0.012**	13 (6.6)	36 (18.4)	**<0.001**
TAP	7 (53.8)	57 (42.8)	0.561	7 (53.8)	11 (5.6)	0.334
Culotte	6 (46.2)	76 (57.2)	0.560	6 (46.2)	24 (12.2)	0.001
Postprocedural TIMI flow grade <3						
MV	5 (2.6)	33 (3.2)	0.823	5 (2.6)	7 (3.6)	0.558
SB	18 (9.2)	129 (12.6)	0.190	18 (9.2)	26 (13.3)	0.201
Residual SB stenosis ≥70%, n(%)	45 (23.0)	465 (45.5)	**<0.001**	45 (23.0)	82 (41.8)	**<0.001**
Procedure time, min	47.21±17.70	40.94±13.18	**<0.001**	47.21±17.70	39.94±11.95	**<0.001**
Fluoroscopy time, min	15.92±9.51	13.39±6.69	**0.001**	15.92±9.51	13.53±6.08	**0.003**
Contrast media volume (mL)	182.96±79.26	171.54±72.17	0.075	182.96±79.26	172.65±67.49	0.167
Angiographic success, n(%)						
MV	191 (97.4)	989 (96.8)	0.617	191 (97.4)	189 (6.4)	0.558
SB	20 (10.2)	152 (14.9)	0.086	20 (10.2)	31(15.8)	0.099

Bold indicates significance level at P <0.05. Abbreviations: JBT: Jailed balloon technique; JWT: Jailed wire technique; MV: Main vessel; PCI: Percutaneous coronary intervention; POT: Proximal optimization technique; PSM: Propensity score matching, SB: Side branch; TAP: T and minimal protrusion; TIMI: Thrombolysis in myocardial infarction

Table 4. In-hospital and mid-term outcomes before and after PSM per study group

	Overall population			Propensity score-matched population		
Parameters	**JBT Group (n=196)**	**JWT Group (n=1022)**	**P**	**JBT Group (n=196)**	**JWT Group (n=196)**	**P**
IH complications, n (%)						
Death	4 (2.0)	27 (2.6)	0.806	4 (2.0)	5 (2.6)	1.00
Major bleeding	3 (1.5)	30 (2.9)	0.342	3 (1.5)	5 (2.6)	1.00
Spontaneous TVMI	1 (0.5)	29 (2.8)	0.073	1 (0.5)	4 (2.0)	0.372
Contrast-induced AKI	11 (5.6)	46 (4.5)	0.462	11 (5.6)	8 (4.1)	0.639
Follow-up time, month	30.87±21.32	31.90±21.89	0.336	30.87±21.32	27.95±19.04	0.153
Mid-term Outcomes, n (%)						
Primary endpoint (MACE)	22 (11.2)	194 (19.0)	**0.009**	22 (11.2)	41 (20.9)	**0.009**
Cardiac death	9 (4.6)	56 (5.5)	0.730	9 (4.6)	12 (6.1)	0.655
TLR	9 (4.6)	106 (10.4)	**0.011**	9 (4.6)	20 (10.2)	0.052
Main vessel	8 (4.1)	89 (8.7)	**0.030**	8 (4.1)	18 (9.2)	0.066
Side branch	3 (1.5)	23 (2.3)	0.787	3 (1.5)	3 (1.5)	1.00
TVMI	12 (6.1)	83 (8.1)	0.339	12 (6.1)	19 (9.7)	0.190
Main vessel	12 (6.1)	76 (7.4)	0.515	12 (6.1)	17 (8.7)	0.335
Side branch	2 (1.0)	12 (1.2)	1.00	2 (1.0)	4 (2.0)	0.411
Stent thrombosis	2 (1.0)	12 (1.2)	1.00	2 (1.0)	4 (2.0)	0.685
All-cause death	10 (5.1)	77 (7.5)	0.288	10 (5.1)	16 (8.2)	0.223

Bold indicates significance level at P <0.05. Abbreviations: AKI: Acute kidney injury; IH: In-hospital; JBT: Jailed balloon technique; JWT: Jailed wire technique; MACE: Major adverse cardiac events; PSM: Propensity score matching, TLR: Target lesion revascularization: TVMI: Target vessel myocardial infarction

Table 5. Univariate and multivariate Cox Regression analysis showing independent predictors of primary endpoint (MACE) with propensity matched data

Parameters	Univariate Level		Multivariate Level	
	HR (95% CI)	P value	HR (95% CI)	P value
SB Protection Strategy (JBT)	0.495 [0.294-0.831]	0.007	0.688 [0.388-1.220]	0.200
Age	1.017 [0.996-1.038]	0.108	1.006 [0.0981-1.032]	0.630
Diabetes mellitus	2.241 [1.366-3.675]	0.001	1.628 [0.959-2.766]	0.071
Chronic kidney disease	1.282 [0.512-3.210]	0.595	0.818 [0.300.2.227]	0.694
LVEF	0.973 [0.948-0.999]	0.042	0.977 [0.501-1.004]	0.101
Hemoglobin	0.855 [0.757-0.965]	0.011	0.858 [0.747-0.986]	**0.031**
WBC	1.027 [0.968-1.019]	0.374	1.044 [0.977-1.114]	0.181
SYNTAX score	1.059 [1.024-1.095]	<0.001	1.041 [1.005-1.078]	**0.022**
Preprocedural TIMI flow grade <3 (MV)	1.745 [0.946-3.215]	0.074	1.818 [0.922-3.584]	0.084
SB lesion length	1.112 [1.040-1.189]	0.001	1.099 [1.023-1.180]	**0.009**
SB intervention	2.874 [1.747-4.728]	<0.001	2.734 [1.334-5.603]	**0.006**
Residual SB stenosis ≥70%	0.865 [0.501-1.496]	0.606	1.441 [0.669-3.106]	0.350

Bold indicates significance level at P <0.05. Abbreviations: CI: Confidence Interval; HR: Hazard ratio; JBT: Jailed balloon technique; LV: Left ventricle ejection fraction; MACE: Major adverse cardiac events; MV: Main vessel; SB: Side branch; SYNTAX: Synergy between Percutaneous Coronary Intervention with Taxus and Cardiac Surgery; TIMI: Thrombolysis in myocardial infarction; WBC: White blood cell

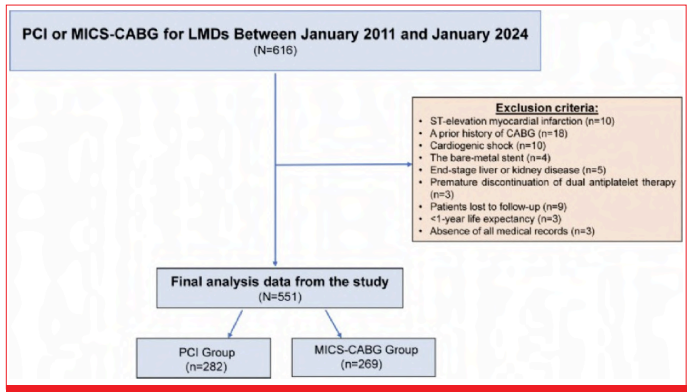

Figure 1. Flow chart for patient selection. CABG: Coronary artery bypass grafting; LMD: Left main coronary artery disease; MICS-CABG: Minimally invasive coronary artery bypass grafting; PCI: Percutaneous coronary intervention.

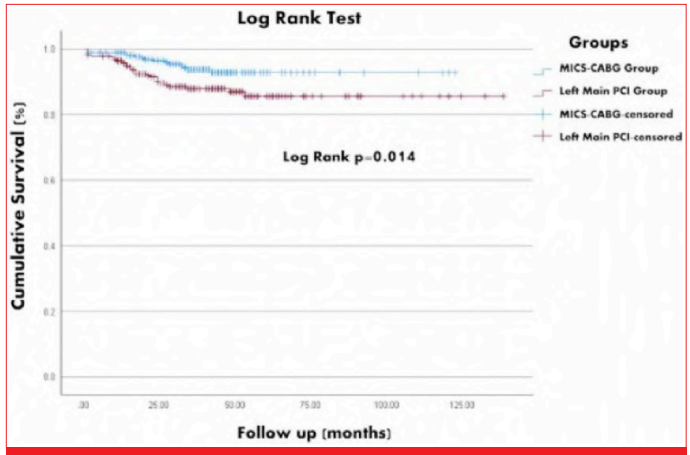

Figure 2. Kaplan-Meier survival analysis for primary endpoint (mortality) under long-term follow-up. MICS-CABG: Minimally invasive coronary artery bypass grafting; PCI: Percutaneous coronary intervention.

OP-025 [Interventional Cardiology / Coronary]

Comparison of long-term oOutcomes of minimally invasive CABG and PCI for left main disease

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Background and Aim: Minimally invasive coronary artery bypass grafting (MICS-CABG) is frequently used for coronary revascularization, but the comparison of long-term clinical results with percutaneous coronary intervention (PCI) in left main disease (LMDs) remains unclear. The present study sought to determine the long-term outcomes of MICS-CABG and PCI in patients with LMDs.

Methods: A total of 551 consecutive patients [man: 457 (82.9%), mean age: 60.70 ± 9.54 years] who underwent PCI or MICS-CABG for LMDs were included. The primary endpoint was defined as the all-cause death during follow-up. The secondary endpoint defined as the major cardiovascular and cerebral events (MACCE) included cardiac death, myocardial infarction, target vessel revascularization, stroke, and stent thrombosis or graft occlusion. Inverse probability weighting (IPW) was performed to reduce treatment selection bias. This is the first report comparing the long-term outcomes of MICS-CABG and PCI in patients with LMDs.

Results: The initial revascularization strategy was MICS-CABG in 269 (48.8%) cases and PCI in 282 (51.2%) patients. The SYNTAX scores [31.25 ± 4.63 vs. 26.05 ± 5.9, p<0.001)] were notably higher in the MICS-CABG group than the PCI group. The incidence of long-term mortality (11 vs. 5.6%, p=0.022) and MACCE (22 vs. 15.2%, p=0.042) were notably higher in the PCI group than in the MICS-CABG group. The long-term mortality (adjusted HR (IPW)=6.38 [95% CI: 3.00-13.57], p<0.001)] and MACCE (adjusted HR (IPW)=4.51 [95% CI: 2.90-7.03], p<0.001)] in the overall population significantly differed between the PCI group and the MICS-CABG group.

Conclusions: The present study suggests that MICS-CABG for LMDs was associated with lower long-term mortality and MACCE rates than PCI.

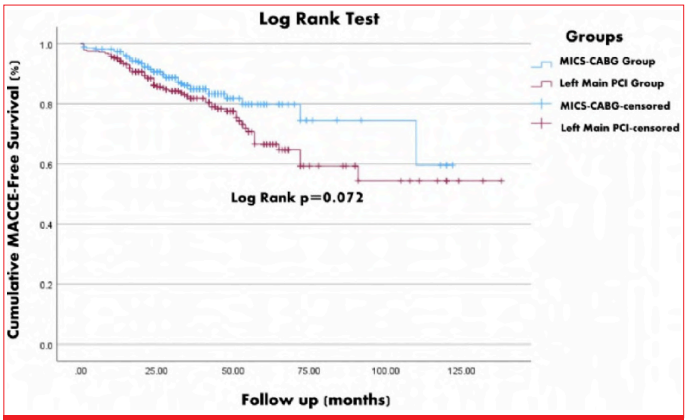

Figure 3. Kaplan-Meier survival analysis for secondary endpoint (MACCE) under long-term follow-up. MACCE: Major cardiovascular and cerebral event; MICS-CABG: Minimally invasive coronary artery bypass grafting; PCI: Percutaneous coronary intervention.

Table 1. Baseline demographic, clinical, and lesion characteristics per study group

Variables	MICS-CABG Group (n=269)	PCI Group (n=282)	P value
Age, years	62.06±7.41	59.41±11.07	**<0.001**
Sex, man	241 (89.6)	216 (76.6)	**<0.001**
Comorbidities			
Hypertension, n(%)	210 (78.1)	216 (76.6)	0.680
Diabetes Mellitus, n(%)	100 (37.2)	93 (33)	0.302
COPD, n(%)	34 (12.6)	32 (11.3)	0.641
Chronic kidney disease, n(%)	62 (22)	73 (25.9)	0.439
Hyperlipidemia, n(%)	170 (63.2)	168 (59.6)	0.383
Smoker, n(%)	134 (49.8)	135 (47.9)	0.649
Previous MI, n(%)	82 (30.5)	83 (29.4)	0.788
Prior valve surgery, n(%)	3 (1.1)	4 (1.4)	1.00
Mediastinal radiation, n(%)	1 (0.4)	2 (0.7)	0.591
History of stroke, n(%)	5 (1.9)	14 (5)	0.060
Heart failure, n(%)	43 (16)	51 (18.1)	0.512
eGFR	84.43±20.7	84.15±21.93	0.323
LVEF(%)	54.31±8.44	54.12±11.26	0.955
EuroSCORE II	1.41±1.12	1.12±0.84	**<0.001**
Clinical presentation			
Chronic coronary syndrome, n(%)	149 (55.4)	133 (47.2)	0.053
USAP nstable angina, n(%)	33 (12.3)	28 (9.9)	0.460
NSTEMI, n(%)	85 (31.6)	121 (42.9)	**0.006**
Lesion characteristics			
Multi-vessel disease, n(%)	237(88.1)	214(75.9)	**<0.001**
SYNTAX score	31.25±4.63	26.05±5.9	**<0.001**
SYNTAX score ≤22, n(%)	26 (10.4)	81(28.7)	**<0.001**
SYNTAX score 23-32, n(%)	130 (51.7)	143 (50.7)	0.821
SYNTAX score ≥33, n(%)	110 (40.9)	58 (20.6)	**<0.001**
RCA disease, n(%)	142 (52.8)	108 (38.2)	**<0.001**
LMCA stenosis, %	76.56±9.81	77.24±13.46	0.207
Lesion location, n(%)*			
Ostial	30 (11.2)	29 (10.3)	0.742
Mid	33 (12.3)	32 (11.3)	0.738
Distal bifurcation	240 (90)	250 (88.7)	0.424
True bifurcation, n(%)	110 (40.9)	100 (35.5)	0.189

Footnote: *"n" donates the number of lesion of LMCA rather than the number of patients LMCA disease.Bold indicates significance level at P value < 0.05.

COPD: Chronic obstructive pulmonary disease; eGFR: Estimated glomerular filtration rate; EuroSCORE: European system for cardiac operative risk evaluation; LMCA: Left main coronary artery; LVEF: Left ventricle ejection fraction; MICS-CABG: Minimally invasive coronary artery bypass grafting; MI: Myocardial infaction; NSTEMI: Non ST-elevation myocardial infarction; PCI: Percutaneous coronary intervention; RCA: Right coronary artery; USAP:Unstable angina pectoris

Table 2. Procedure details of both interventional groups

Variables	MICS-CABG Group (n=269)
Robotic-assisted MICS-CABG, n(%)	161 (59.8)
Procedure, n(%)	
Elective	225 (83.6)
Urgent/emergent	44 (16.4)
Vessels treated	2.01±0.77
ITA use, n(%)	268 (99.6)
Radial artery graft use, n(%)	18 (6.7)
Single arterial greft use, n(%)	240 (89.2)
Multiarterial CABG, n(%)	28 (10.4)
Saphenous venous grafts, n(%)	51 (19)
Hybrid coronary revascularisation, n(%)	89 (31.1)
Number of total arterial greft	1.11±0.33
Number of total greft	1.38±0.65
Conversion to full sternotomy, n(%)	11 (4.1)

CABG: Coronary bypass grafting; ITA: Internal thoracic artery; MICS-CABG: Minimally invasive coronary artery bypass grafting

Table 3. Procedure details of left main PCI

Variables	Left Main PCI (n=282)
Procedure, n(%)	
Elective	232 (82.3)
Urgent/emergent	50 (17.7)
Access site, n(%)	
Femoral	275 (97.5)
Radial	7 (2.5)
Intra-aortic balloon pump support, n(%)	12 (4.3)
Utilization of IVUS, n(%)	30 (10.6)
Performed rotational atherectomy, n(%)	2 (0.7)
Performed intravascular lithotripsy, n(%)	2 (0.7)
Pre-dilation, n(%)	222 (78.7)
Provisional stenting, n(%)	181 (64.2)
2-stenting technique, n(%)	99 (35.1)
DK-Crush	30 (10.6)
Nano-crush	12 (4.3)
Mini-crush	21 (7.4)
Mini-culotte	14 (5)
DK-culotte	3 (1.1)
V/SKS	2 (0.7)
TAP	17 (6)
Main vessel stent diameter, mm	3.74±0.26
Main vessel stent length, mm	23.76±7.22
LCx stent diameter, mm	1.15±1.46
LCx stent length, mm	8.30±11.46
Final kissing balloon inflation, n(%)	116 (41.1)
Final POT, n(%)	272 (96.5)
POT balloon diameter, mm	4.49±0.72
Thrombus Aspiration, n(%)	5 (1.8)
Tirofiban use during PCI, n (%)	30 (10.6)
Contrast media volume (mL)	147.38±74.31

Abbreviations: DK: Double kissing; IVUS: Intravascular ultrasound; LCx: Left circumflex artery; PCI: Percutaneous coronary intervention; POT: Proximal optimization technique; SKS: Simultaneous kissing stents; TAP: T and small protrusion technique.

Table 4. In-hospital and long-term clinical outcomes per study group

Variables	MICS-CABG Group (n=269)	Left Main PCI Group (n=282)	P value
In-hospital outcomes, n (%)			
All-cause-death	3 (1.1)	5 (1.8)	0.725
TVR	4 (1.5)	6 (2.1)	0.752
ST or symptomatic graft occlusion	0 (0)	2 (0.7)	0.499
MI	4 (1.5)	6 (2.1)	0.752
Stroke	1 (0.4)	0 (0)	0.488
MACCE	8 (3)	8 (2.8)	1.00
All-cause-death or MI	6 (2.2)	8 (2.8)	0.789
Contrast induced nefropathy	0 (0)	30 (10.6)	-
AKI requiring RRT	8 (3)	5 (1.8)	0.409
Prolonged endotracheal intubation (>24 h)	10 (3.7)	4 (1.4)	0.107
Pneumothorax	3 (1.1)	0 (0)	-
Superficial wound infections	4 (1.5)	0 (0)	-
TIMI-Major bleeding	10 (3.7)	4 (1.4)	0.107
Hospital length of stay, days	4.77±1.46	2.19±1.31	**<0.001**
Follow-up time, months	38.25±19.43	38.70±25.24	0.107
Long-term outcomes, n (%)			
All-cause death	15 (5.6)	31 (11)	**0.022**
MACCE	41 (15.2)	62 (22)	**0.042**
Cardiac death	12 (4.5)	22 (7.8)	0.103
MI	16 (5.9)	16 (5.7)	0.891
TVR	27 (10)	41 (14.5)	0.228
Stroke	3 (1.1)	4 (1.4)	1.00
ST or symptomatic graft occlusion	8 (3)	5 (1.8)	0.409

Footnote: Bold indicates significance level at P value < 0.05.

Abbreviations: AKI: Acute kidney injury; MACCE: Major cardiovascular and cerebral event; MI: Myocardial infarction; MICS-CABG: Minimally invasive coronary artery bypass grafting; RRT: Renal replacement therapy; ST: Stent thrombosis TVR: Target vessel revascularization

Table 5. IPW-Cox proportional models predicting long-term all-cause death

Parameters	Unadjusted HR	95% CI	P	Adjusted HR (IPW)	95% CI	P
Being in LM-PCI group	2.12	1.14-3.93	0.017	6.38	3.00-13.57	**<0.001**
Age	-	-	-	1.04	1.00-1.09	**0.038**
Gender (male)	-	-	-	0.87	0.39-1.97	0.745
Diabetes Mellitus	-	-	-	0.78	0.38-1.63	0.517
Chronic kidney disease	-	-	-	1.38	0.59-3.24	0.455
Chronic pulmonary disease	-	-	-	1.71	0.82-3.54	0.152
LVEF	-	-	-	0.99	0.96-1.02	0.474
SYNTAX score	-	-	-	1.52	1.34-1.72	**<0.001**
U/E procedure	-	-	-	5.66	2.79-11.48	**<0.001**

Footnote: Bold indicates significance level at P value < 0.05.

Abbreviations: CI: Confidence Interval; HR: Hazard ratio; IPW: Inverse probability-weighted; LV: Left ventricular ejection fraction; PCI: Percutaneous coronary intervention; U/E: Urgent/emergency

Table 6. IPW-Cox proportional models predicting long-term MACCE

Parameters	Unadjusted HR	95% CI	P value	Adjusted HR (IPW)	95% CI	P value
Being in Left Main-PCI group	1.35	0.91-1.99	0.138	4.52	2.90-7.03	**<0.001**
Age	-	-	-	1.02	0.99-1.04	0.188
Sex (man)	-	-	-	0.68	0.41-1.13	0.133
Diabetes Mellitus	-	-	-	0.78	0.50-1.22	0.283
Chronic kidney disease	-	-	-	1.84	1.16-2.93	**0.010**
Chronic pulmonary disease	-	-	-	1.26	0.75-2.13	0.381
LVEF	-	-	-	1.01	0.99-1.03	0.539
SYNTAX score	-	-	-	1.40	1.32-1.49	**<0.001**
U/E procedure	-	-	-	2.47	1.51-4.02	**<0.001**

Footnote: Bold indicates significance level at P value < 0.05.

Abbreviations: CI: Confidence Interval; HR: Hazard ratio; IPW: Inverse probability-weighted; LVEF: Left venticle ejection fraction; MACCE: Major cardiovascular and cerebral event; PCI: Percutaneous coronary intervention; U/E: Urgent/emergency

OP-026 [Hypertension]

Association between circadian blood pressure patterns and retinal nerve fiber layer: An early indicator of microvascular damage in newly diagnosed hypertensive individuals

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Background and Aim: Retinal nerve fiber layer (RNFL) thinning may serve as an early indicator of subclinical target organ damage in hypertension, as the retina is one of the few organs where the microvascular structure can be directly visualized. Non-dipping patterns (including non-dipper and reverse dipper subgroups), characterized by a reduced or absent nocturnal blood pressure fall, have been associated with target organ damage and adverse cardiovascular outcomes. However, the relationship between these abnormal blood pressure patterns and RNFL thickness remains insufficiently clarified.

Methods: A total of 46 newly diagnosed hypertensive individuals without comorbidities or antihypertensive treatment were enrolled. Based on 24-hour ambulatory BP monitoring, patients were categorized as dipper (n=23), non-dipper (n=13), or reverse dipper (n=10). RNFL thickness was measured using optical coherence tomography (OCT), with both eyes of each participant evaluated separately, totaling 92 eyes. Multivariable logistic regression and ROC analysis were performed to identify independent predictors of RNFL thinning.

Results: Mean RNFL thickness was significantly lower in patients with non-dipping BP patterns (i.e., non-dipper and reverse dipper) compared to dippers (p<0.001). In multivariable logistic regression (Table 1), older age (OR: 1.233, 95% CI: 1.096–1.386, p<0.001), higher 24-hour average systolic BP (OR: 1.079, 95% CI: 1.014–1.148, p=0.016), and not having a dipping BP pattern (i.e., non-dipping profile) (OR: 0.106, 95% CI: 0.022–0.509, p=0.005) were independently associated with RNFL thinning. ROC analysis (Figure 1) revealed that the area under the curve (AUC) for 24-hour average systolic BP was 0.720 (p=0.001), with an optimal cut-off value of 126 mmHg (sensitivity: 73%, specificity: 67%) for predicting RNFL thinning.

Conclusions: Non-dipping BP patterns were significantly associated with RNFL thinning. This finding suggests that RNFL thickness may have prognostic value as an early marker of microvascular damage. Even in newly diagnosed, untreated individuals, the presence of RNFL thinning in non-dipping profiles highlights their higher risk for target organ damage. RNFL measurement may represent a valuable biomarker for early detection and prevention strategies in this population.

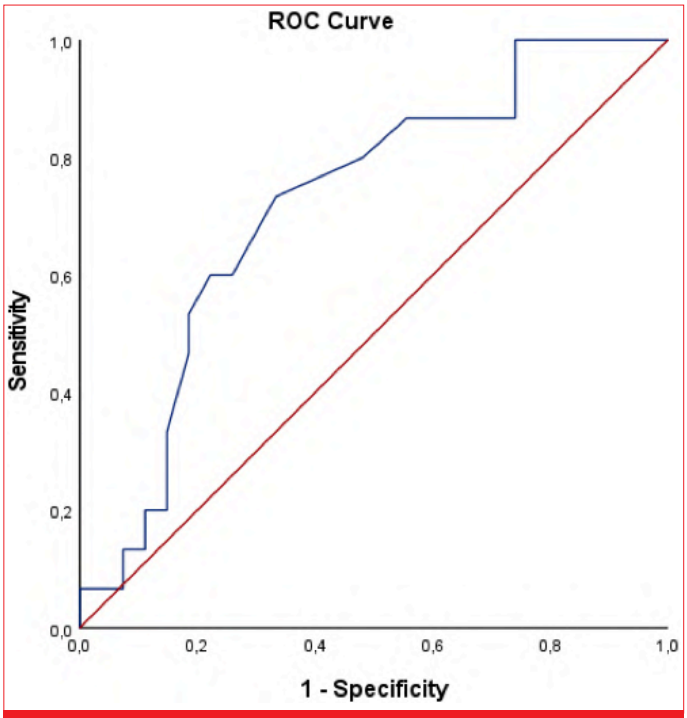

Figure 1. ROC curve of 24-hour average systolic blood pressure for predicting RNFL thinning.

Table 1. Multivariable logistic regression analysis of independent predictors of RNFL thinning

Variables	OR	CI %95	p value
Age	1.233	1.096-1.386	<0.001
24h average SBP	1.079	1.014-1.148	0.016
Dipper blood pressure pattern	0.106	0.022-0.509	0.005

OP-027 [Hypertension]

Artificial intelligence-based prediction of hypertension patterns in patients with newly diagnosed hypertension

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Background and Aim: In patients with hypertension (HT), circadian blood pressure (BP) variation patterns affect the risk of cardiovascular events. Our study aims to contribute to the development of early diagnosis and prevention plans by using artificial intelligence-supported models to identify these patterns in patients newly diagnosed with HT, based on pre-treatment baseline data.

Methods: This retrospective cross-sectional study included 315 patients with newly diagnosed HT. Patients were categorized into three BP circadian rhythm groups based on ambulatory BP monitoring (ABPM): dipper (n=110), non-dipper (n=105), and reverse-dipper (n=100). However, ABPM values were excluded from the input features, and only clinical measurements obtained during the initial outpatient visit were used to evaluate the predictability of circadian patterns. Two supervised machine learning models were trained to classify patients into dipper, non-dipper, and reverse-dipper categories: Multinomial Logistic Regression and Random Forest Classifier. The dataset was randomly split into training (80%) and testing (20%) subsets using stratified sampling. Input features were normalized using Standard Scaler. Model performance was evaluated based on accuracy, F1-score, precision, recall, and confusion matrices.

Results: The mean age of the patients was 52.47 ± 9.42 years, their mean body mass index was 29.67 ± 2.98 kg/m², and 52.4% (n=165) of them were male. Their average office systolic BP was 153.43 ± 10.23 mmHg, diastolic BP was 98.2 ± 7.79 mmHg, 24-hour systolic BP was 145.62 ± 11.3 mmHg and diastolic BP was 95.42 ± 8.99 mmHg. The multinomial logistic regression model achieved a higher overall accuracy (0.87) compared to the random forest model (0.78). Similarly, both macro-averaged and weighted F1-scores were superior in the logistic regression model, indicating better class balance and prediction consistency across all three groups. The dipper and reverse-dipper groups had excellent classification metrics, with F1-scores of 0.91 in both groups. The recall for the reverse-dipper class reached 1.00, indicating that the model correctly identified all reverse-dipper patients in the test set. In contrast, the non-dipper group had a lower recall (0.67), suggesting some degree of misclassification, possibly due to overlapping phenotypic features. To identify the most influential variables in the prediction of circadian BP patterns, feature importance was analyzed using the random forest model. The most influential feature was interventricular septum thickness, followed by body mass index, posterior wall thickness and body roundness index.

Conclusions: In conclusion, circadian BP patterns can be effectively predicted using baseline clinical data and machine learning techniques, without requiring ABPM. This approach may offer a practical decision-support tool in primary care settings or resource-limited environments where ABPM is unavailable. Future validation in external cohorts is warranted.

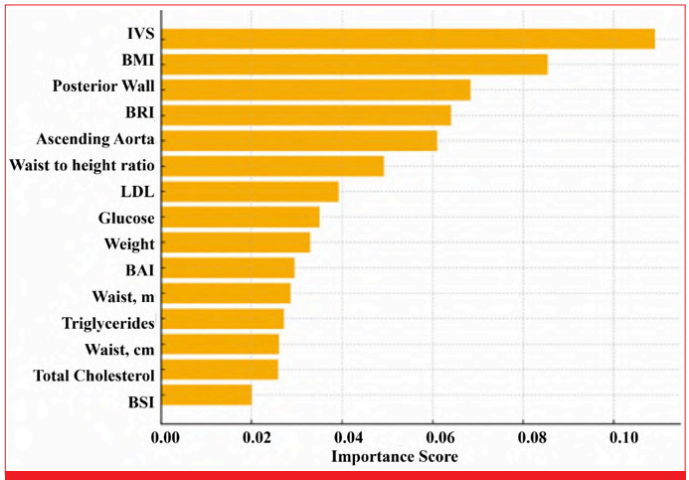

Figure 1. Top 15 most important features (random forest). IVS: Interventricular septum, BMI: Body mass index, BRI: Body roundness index, LDL-C: Low-density lipoprotein cholesterol, BAI: Body adiposity index, BSI: Body shape index.

Table 1. Comparison of model performances

Model	Accuracy	Macro Average F1	Weighted Average F1
Logistic Regression	0.87	0.87	0.87
Random Forest	0.78	0.77	0.78

F1: The F1 score is the harmonic mean of the precision and recall values calculated for each class.

Table 2. Class-wise performance metrics for the logistic regression model

Class	Precision	Recall	F1-Score
Dipper	0.88	0.95	0.91
Non-dipper	0.93	0.67	0.78
Reverse-dipper	0.83	1.00	0.91

F1: The F1 score is the harmonic mean of the precision and recall values calculated for each class.

Table 3. Top 15 most important features (random forest)

Rank	Feature	Importance score
1	IVS, mm	0.109
2	BMI, kg/m^2	0.085
3	Posterior wall, mm	0.068
4	BRI	0.064
5	Ascending aorta, mm	0.061
6	Waist to height ratio	0.049
7	LDL-C, mg/dL	0.039
8	Glucose, mg/dL	0.035
9	Weight, kg	0.033
10	BAI	0.030
11	Waist, m	0.029
12	Triglycerides, mg/dL	0.027
13	Waist, cm	0.026
14	Total cholesterol, mg/dL	0.026
15	BSI	0.020

IVS: Interventricular septum, BMI: Body mass index, BRI: Body roundness index, LDL-C: Low-density lipoprotein cholesterol, BAI: Body adiposity index, BSI: Body shape index

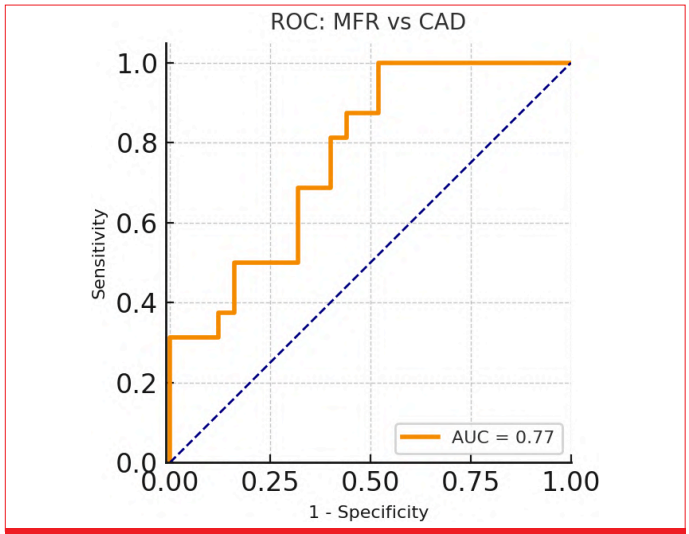

Figure 1. ROC curve for MFR in predicting significant coronary artery stenosis. AUC=0.77.

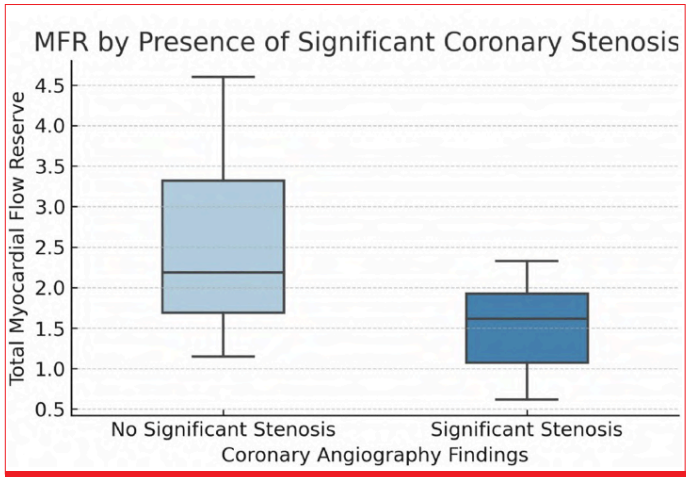

Figure 2. Boxplot of MFR values stratified by angiographic findings. Patients with significant coronary stenosis (>50%) demonstrated lower MFR values compared to those without stenosis.

OP-028 [Nuclear Cardiology]

The relationship between angiographic significant coronary artery stenosis and myocardial flow reserve using dynamic CZT-SPECT imaging

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Background and Aim: Dynamic SPECT imaging with cadmium-zinc-telluride (CZT) detectors enables noninvasive quantification of myocardial flow reserve (MFR), providing functional information into both epicardial stenosis and coronary microvascular dysfunction. This study aimed to investigate the relationship between MFR and significant coronary artery disease (CAD), ischemia burden on myocardial perfusion scintigraphy (MPS), and clinical risk factors in patients with and without obstructive disease.

Methods: We retrospectively evaluated 45 patients (median age: 62 years, 58% male) who had dynamic CZT-SPECT imaging with quantitative MFR calculation, MPS and coronary angiography. MFR was calculated for each major coronary territory and globally averaged. Abnormal MFR was defined as <2.0-2.5 and significant CAD as >50% stenosis on angiography. Clinical data including age, sex, presence of hypertension, diabetes, hyperlipidemia, previous CAD, angina and dyspnea were collected. Patients were classified by the presence or absence of significant stenosis. Statistical analyses included Spearman correlation, Mann–Whitney U test and ROC analysis.

Results: Patients with significant coronary artery stenosis had significantly lower MFR values compared to those without stenosis (median MFR: 1.61 vs. 2.19, p=0.002). ROC analysis was performed to assess the diagnostic performance of MFR in detecting angiographically significant CAD. Using a threshold of MFR<2.3, the test achieved a sensitivity of 94% and specificity of 52%, with fair overall diagnostic accuracy (AUC=0.77). MFR demonstrated an inverse correlation with ischemia percentage on MPS (ρ=–0.37, p=0.022). A trend toward higher ischemia percentages in patients with stenosis was observed but did not reach statistical significance (p=0.056). Among patients without significant stenosis, there was no statistically significant association between cardiovascular risk factors or symptoms and MFR values.

Conclusions: CZT-SPECT derived MFR is significantly associated with both angiographic stenosis and ischemia burden, reinforcing its utility in identifying physiologically relevant obstructive CAD. In patients with angiographically normal coronaries, MFR abnormalities consistent with microvascular dysfunction were frequently observed, yet not reliably predicted by conventional risk factors or symptomatic status. The high sensitivity and moderate specificity observed suggest that MFR is a valuable screening tool for ruling out significant coronary artery disease. Notably, 52% of patients with normal coronary angiograms had reduced MFR, which may explain the test's moderate specificity and suggests the presence of functional abnormalities such as microvascular angina that are not detectable by conventional angiography. These findings highlight the potential value of incorporating MFR quantification alongside conventional MPS into diagnostic algorithms to improve early detection and management of both obstructive and non-obstructive coronary syndromes.

OP-029 [Pulmonary Hypertension / Pulmonary Vascular Disease]

Evaluation of the correlation between right ventricular function parameters and sST2 in patients with idiopathic pulmonary fibrosis

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Background and Aim: To evaluate sST2 as a biomarker for right ventricular dysfunction and its correlation with echocardiographic parameters in IPF patients before and after treatment. This prospective study enrolled 58 patients with newly diagnosed IPF or established IPF initiating therapy. Right heart assessment included TAPSE, FAC, tricuspid S', RVSP, E/A ratio, and E/e' ratio. Serum sST2 levels were measured at baseline and 3-month follow-up

Methods: This prospective cardiovascular study enrolled 58 patients with newly diagnosed IPF or established IPF patients initiating pulmonary vasodilator or antifibrotic therapy. Comprehensive right heart assessment included advanced echocardiographic evaluation: tricuspid annular plane systolic excursion (TAPSE), right ventricular fractional area change (FAC), tricuspid lateral annular systolic velocity (S'), estimated right ventricular systolic pressure (RVSP), tricuspid E/A ratio, and tricuspid E/e' ratio for diastolic function assessment. Serum sST2 levels were measured at baseline and 3-month follow-up. Correlations between sST2 dynamics and right heart remodeling parameters were analyzed.

Results: Mean age was 67.3 ± 8.2 years (62% male). Baseline sST2 levels were elevated (45.2 ± 12.8 ng/mL) and significantly reduced after treatment (32.1 ± 9.4 ng/mL, p<0.001). Baseline sST2 correlated positively with RVSP (r=0.68, p<0.001), E/A ratio (r=0.41, p<0.05), and E/e' ratio (r=0.56, p<0.01), and negatively with TAPSE (r=–0.52, p<0.01), FAC (r=–0.48, p<0.01), and S' velocity (r=–0.44, p<0.05). Baseline E/A ratio was 1.8 ± 0.6 and E/e' ratio was 12.4 ± 4.2, indicating diastolic dysfunction. sST2 reduction correlated with improved TAPSE (r=0.58, p<0.001), RVSP reduction (r=0.61, p<0.001), and diastolic parameter normalization.

Conclusions: sST2 strongly correlates with right ventricular dysfunction parameters in IPF patients. Its reduction following therapy parallels cardiovascular improvement, positioning sST2 as a valuable biomarker for monitoring therapeutic response and cardiovascular risk stratification in IPF management.

OP-030 [Pulmonary Hypertension / Pulmonary Vascular Disease]

Pulmonary hypertension registry in Turkey (TURKPAH)

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Background and Aim: Despite significant advances in the diagnosis and treatment of pulmonary arterial hypertension (PAH), it remains a progressive and fatal disease. National and regional registries provide essential data regarding disease characteristics, treatment patterns, and outcomes.

Methods: As Turkish Society of Cardiology study group, we conducted a nationwide pulmonary hypertension (PH) registry study through a questionnaire survey from 39 PAH centers in 7 regions of Turkey. Demographic data, hemodynamic classification, and treatment methods of patients diagnosed with PH by cardiac catheterization were queried. Ethical committee approval was obtained for the study.

Results: A total of 3682 patients were included in the study. The mean age at diagnosis was 52.2 years, and 69% of the patients were female. Of the registered patients, 2954 were still alive and 728 had died. The distribution of patients by region is shown in Figure 1. In the hemodynamic classification of patients, 61 had postcapillary PH, 82 had combined pre- and postcapillary PH, and the majority had precapillary PH. Among these, Group 1 PAH: 2735 (74.3%), Group 3 (hypoxia-related) PH: 136 (3.7%), Group 4 PH due to pulmonary artery obstruction: 642 (17%), and Group 5 PH: 26 patients (0.7%). Of the patients in Group 1 PAH, 47.4% had IPAH, and 45.9% had associated PH. The distribution is shown in Table 1. Treatment distribution in Group 1 included monotherapy in 32.6%, dual combination therapy in 40.3%, triple oral combination in 20.4%, and parenteral therapy in 0.6%. Ten vasoreactive patients received calcium channel blockers, while 134 patients remained untreated. Among Group 4 patients on medical therapy (n=495), monotherapy was most common (n=428) mostly riociguat, while 56 received dual, 9 triple, and 2 parenteral therapy.

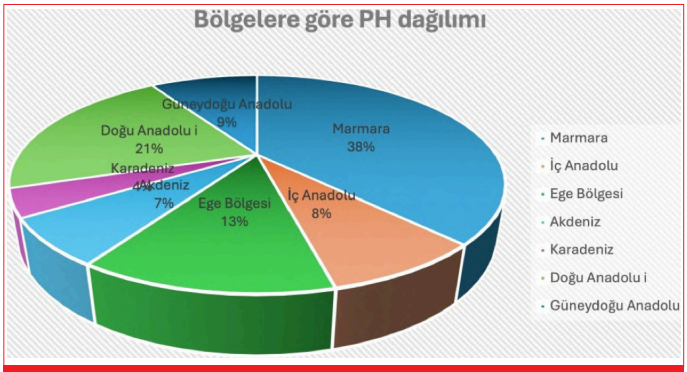

Figure 1. Distribution of patients applying to PAH centers in Turkey by region.

Conclusions: This registry represents the largest multicenter PH database in Turkey, including 39 centers from all 7 regions. Female predominance was consistent with international registries. Compared to other cohorts, connective tissue disease- and portopulmonary hypertension–related PH were less frequent, while congenital heart disease–associated PH was more prevalent. The widespread use of dual and triple oral combination therapy was notable, whereas parenteral therapy rates were lower than in international registries. These findings highlight both similarities and unique treatment patterns in the Turkish PH population.

Table 1. Ratios of all groups and PAH patients according to etiologies

PAH Patient group	N	% (within all group)	% (within PAH patients)
IPAH %	1295	35	47.4
Vasoreactive	43	0.11	0.15
Non vasoreactive	1254		
dug /toxin associated (%)	11	0.2	
PVOD (%)	4	0.1	0.15
Connective tissue disease (%)	363	9.8	13.2
Congenital heart disease (%)	875	23.7	32
Porto-pulmonary hypertansiyon (%)	21	0.6	0.7
Associtaed PAH (total)	1260	34	45.9

OP-031 [Pulmonary Hypertension / Pulmonary Vascular Disease]

A real-life, single-center experience: Retrospective evaluation of prognostic indicators and risk stratification models in pulmonary arterial hypertension

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Background and Aim: Risk stratification plays a critical role in optimizing treatment and predicting outcomes in patients with pulmonary arterial hypertension (PAH). We present real-life data from a single-center cohort to evaluate prognostic indicators and compare performance of risk models in predicting mortality.

Methods: This retrospective, observational cohort study included 405 adult patients (88.1% incident, 11.9% prevalent) who underwent right heart catheterization for pulmonary hypertension (PH) at Ege University PAH Center between June 2008 and April 2021. Data were collected through May 2023. Survival analysis from catheterization to all-cause mortality was conducted. Independent predictors of

mortality were identified through univariate and multivariate Cox and logistic regression analyses. Risk model performance was evaluated via ROC curves, Youden index, and Kaplan-Meier survival analysis.

Results: Of 405 patients, 40.7% had PAH; the largest subgroup was CHD-associated PAH, while CTD-associated PAH had the worst prognosis (Log-Rank p<0.001) (Figure 2, Graphic 4). Key independent predictors of mortality across the PH cohort included TAPSE/sPAP (HR 0.040, p=0.020), SvO_2 (HR 0.947, p=0.007), and RAP/PCWP ratio (HR 4.113, p=0.020) (Table 4). A newly developed real-life risk score based on these predictors demonstrated better sensitivity (73%) compared to the 2022 ESC/ERS three-strata model (33%), albeit with moderate specificity (70% vs. 89%) (Graphic 1). However, in the PAH subgroup, only the ESC/ERS score retained statistical significance (AUC 0.682), while the new score lacked predictive value (AUC 0.523, p=0.422) (Graphic 2). Kaplan-Meier analysis confirmed predictive utility of WHO-FC, 6MWT, and TAPSE/sPAP (Graphic 8-10). NT-proBNP discriminated only low-risk from higher risk strata (Graphic 11). SvO_2 and RAP remained robust survival indicators even within the PAH subgroup, whereas cardiac index and stroke volume index failed to distinguish risk (Graphic 12-19).

Conclusions: This real-world cohort highlights the limitations of generic risk scores in heterogeneous PH populations and underscores the need for subgroup-specific, adaptable models. Integration of real-life data into AI-powered platforms may enhance individualized risk prediction and clinical decision-making in PAH management.

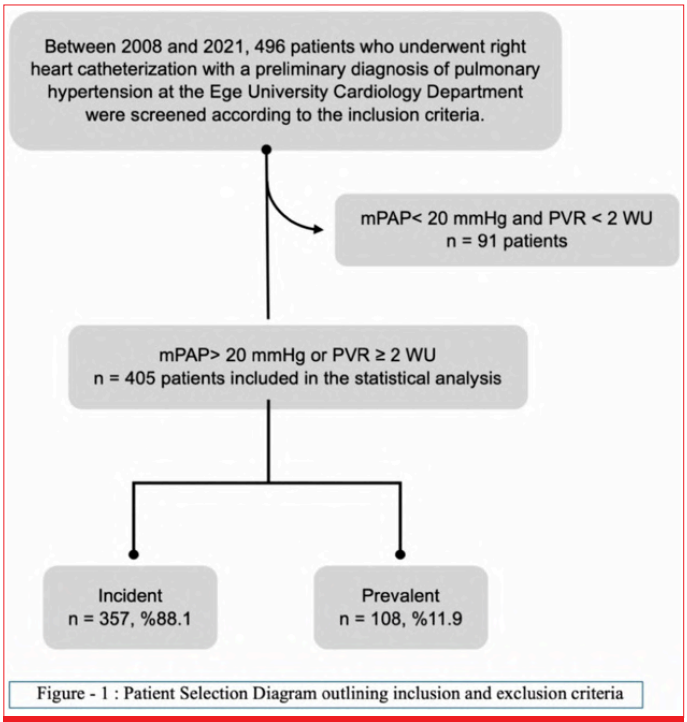
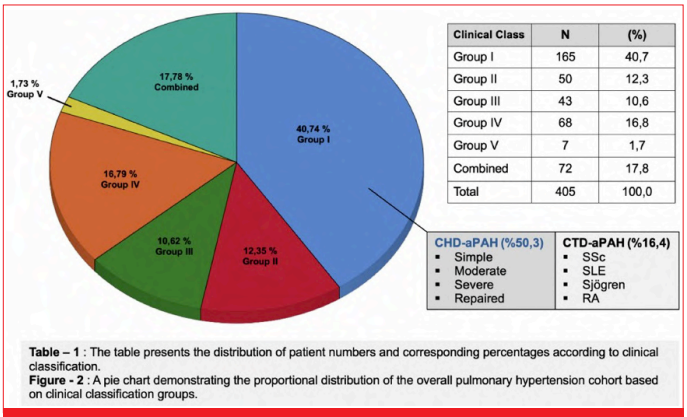


Figure - 1 : Patient Selection Diagram outlining inclusion and exclusion criteria

Figure 1. Flowchart / Patient selection diagram. A. Inclusion Criteria I. Patients who underwent right heart catheterization for hemodynamic and clinical assessment at the Department of Cardiology, Ege University Faculty of Medicine Pulmonary Arterial Hypertension (PAH) Center II. Patients with a mean pulmonary arterial pressure (mPAP) ≥ 20 mmHg and pulmonary vascular resistance (PVR) ≥ 2 Wood units as determined by right heart catheterization III. Patients aged 18 years and older IV. Patients (or, in the case of deceased individuals, their first-degree relatives) who read, understood, and signed the informed consent form B. Exclusion Criteria I. Patients who read the informed consent form and explicitly declined to participate in the study II. Patients who underwent right heart catheterization at institutions other than the Ege University PAH Center. The study was designed as an analytical, retrospective cohort study.



Clinical Class	N	(%)
Group I	165	40,7
Group II	50	12,3
Group III	43	10,6
Group IV	68	16,8
Group V	7	1,7
Combined	72	17,8
Total	405	100,0

Table – 1 : The table presents the distribution of patient numbers and corresponding percentages according to clinical classification.
Figure - 2 : A pie chart demonstrating the proportional distribution of the overall pulmonary hypertension cohort based on clinical classification groups.

Figure 2 & Table 1. Distribution according to clinical classification. The term 'combined group' refers to patients in whom features from multiple clinical classifications are concurrently prominent, and in whom clinical and hemodynamic findings suggest a multifactorial etiology that justifies grouping under more than one PAH classification due to the coexistence of multiple dominant clinical features. Abbreviations: CHD-aPAH: Congenital Heart Disease-associated Pulmonary Arterial Hypertension, CTD-aPAH: Connective Tissue Disease-associated PAH, SSc: Systemic Sclerosis, SLE: Systemic Lupus Erythematosus, RA: Rheumatoid Arthritis.

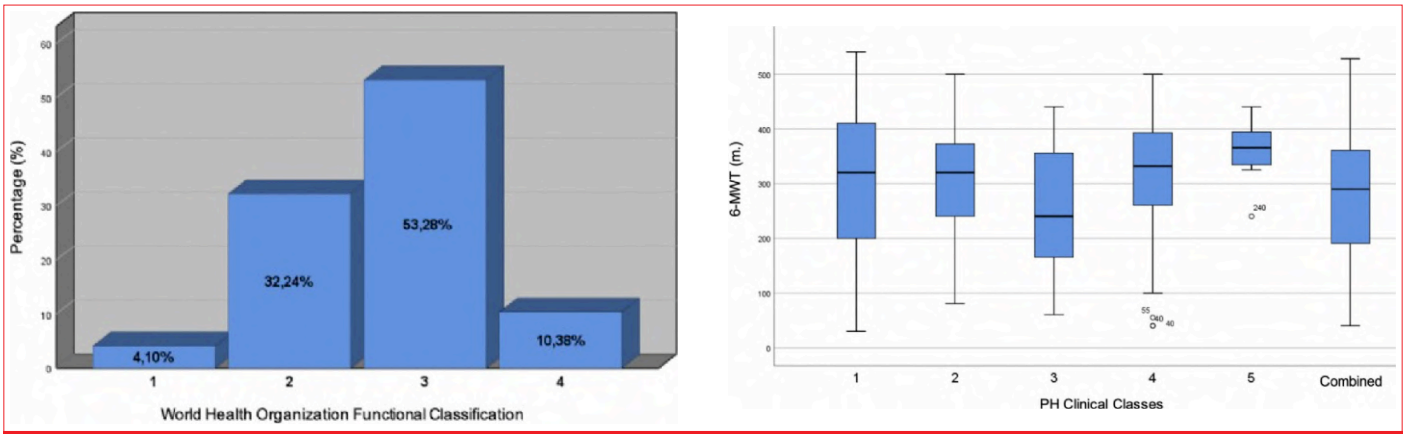


Figure 3, 4. WHO - FC Histogram & 6-MWT Box Plot of PH Cohort. Figure 3: A histogram depicting the distribution of the entire pulmonary hypertension (PH) cohort according to the World Health Organization functional classification (WHO-FC) is presented. The majority of patients (53.28%) were classified as WHO Functional Class III. Figure 4: The box plot illustrates the distribution of 6-minute walk test (6MWT) distances by PH clinical classification, presented as median and interquartile ranges. Group 3 PH is observed to have the lowest median walking distance among the clinical groups. Outlier values beyond the interquartile range are also displayed.

Table 2. Baseline characteristics, medical history, co-morbidities and their distribution across clinical groups

Variable	Total	Group I	Group II	Group III	Group IV	Group V	Combined Group	p value
Age	53.9 ± 15.9	48.1 ± 17.1	58.0 ± 12.0	58.3 ± 12.7	55.9 ± 15.7	53.6 ± 13.3	59.3 ± 13.4	<0.001
Incident	357 (88.1%)	143 (35.3%)	44 (10.9%)	35 (8.6%)	61 (15.1%)	6 (1.5%)	68 (16.8%)	0.397
Female Gender	275 (67.9%)	119 (29.4%)	37 (9.1%)	20 (5%)	43 (10.7%)	7 (1.7%)	49 (12.2%)	0.009
Weight (kg)	71.4 ± 16.3	68.8 ± 16.2	72.38 ± 14.7	72.75 ± 16.0	74.45 ± 14.3	59.43 ± 8.0	74.39 ± 18.8	0.022
Height (cm)	161.9 ± 9.5	161.6 ± 8.8	160.65 ± 11.246	164.70 ± 10.469	164.34 ± 9.472	157.29 ± 1.976	160.36 ± 9.001	0.028
Body Mass Index (kg/m^2)	27.31 ± 6.49	26.34 ± 6.05	28.50 ± 8.24	26.83 ± 5.82	27.63 ± 5.34	24.05 ± 3.52	28.97 ± 7.24	0.032
Body Surface Area (m^2)	1.78 ± 0.22	1.74 ± 0.22	1.78 ± 0.20	1.81 ± 0.23	1.83 ± 0.20	1.60 ± 0.10	1.80 ± 0.23	0.014
Heart Rate (bpm)	82.0 ± 14.5	80.5 ± 13.3	81.2 ± 17.8	88.4 ± 13.5	80.4 ± 12.9	86.8 ± 19.30	83.6 ± 15.3	0.032
Atrial Fibrillation	56 (13 8%)	14 (3 5%)	22 (5.5%)	4 (1%)	5 (1.3%)	1 (0.3%)	10 (2.5%)	<0.001
Arterial Hypertension	135 (33.3%)	51 (12. 7%)	25 (6.2%)	12 (3%)	18 (4. %5)	1 (0.2%)	28 (7%)	0.110
Diabetes Mellitus	81 (20%)	26 (6.5%)	20 (5%)	8 (2%)	9 (2.3%)	2 (0.5%)	16 (4%)	0.013
Dyslipidemia	40 (9.9%)	12 (3%)	12 (3%)	2 (0.5%)	7 (1.8%)	0 (%)	7 (1.8%)	0.023
Smoking	68 (16.9%)	23 (5.7 %)	5 (1.2%)	12 (3%)	16 (4%)	1 (0.2%)	11 (2.7%)	0.083
Cerebrovascular Disease	9 (2.2%)	2 (0.5%)	4 (1%)	1 (0.2%)	1 (0.2%)	0 (0%)	1 (0.2%)	0.137
Lung Disease	163 (40.6%)	27 (6.7%)	6 (1.5%)	37 (9.2%)	48 (12%)	2 (0.5%)	43 (10.7%)	<0.001
Liver Disease	10 (2.5%)	6 (1.5%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	4 (1%)	0.155
Kidney Disease	7 (1.7%)	2 (0.5%)	1 (0.2%)	1 (0.2%)	1 (0.2%)	0 (0%)	2 (0.5%)	0.965
Hematologic Disease	11 (2.7%)	1 (0.2%)	0 (0%)	0 (0%)	5 (1.2%)	3 (0.7%)	2 (0.5%)	<0.001
Rheumatologic Disease	59 (14.7%)	27 (6.7%)	1 (0.2%)	3 (0.7%)	8 (2%)	2 (0.5%)	18 (4.5%)	0.005
Malignancy	15 (3.7%)	6 (1.5%)	2 (0.5%)	1 (0.2%)	2 (0.5%)	0 (0%)	4 (1%)	0.942
All-Cause Death	250 (61.9%)	86 (21.3%)	28 (6.9%)	36 (8.9%)	43 (10.6%)	4 (1%)	53 (13.1%)	0.001

The table presents the baseline characteristics of the study population as mean ± standard deviation, while comorbidities and clinical features are expressed as frequencies (%). Scale parameters were analyzed using one-way ANOVA, and categorical variables were assessed through crosstables. The percentages in parentheses were calculated based on the total study population of 405 patients.

Table 3. Distribution of 6-minute walk distance (6MWD) and WHO functional class (WHO-FC) by clinical classification

	Total	Group I	Group II	Group III	Group IV	Group V	Combined Group	p value
6-Minute Walking Test (m)	299.54 ± 121.5	307.44 ± 129.2	317.00 ± 100.7	253.70 ± 109.9	313.82 ± 111.3	357.43 ± 65.7	279.60 ± 121.4	0.039
World Health Organization Functional Class (n, %)								0.259
WHO – FC 1	15 (4.1%)	7 (1.9%)	0 (0%)	0 (0%)	2 (0.6%)	0 (0%)	6 (1.7%)	
WHO – FC 2	118 (32.2%)	50 (13.8%)	16 (4.4%)	7 (1.9%)	22 (6.1%)	3 (0.8%)	20 (5.5%)	
WHO – FC 3	195 (53.3%)	79 (21.6%)	20 (5.5%)	24 (6. %6)	34 (9.3%)	4 (1.1%)	34 (9.3%)	
WHO – FC 4	38 (10.5%)	13 (3.6%)	2 (0.6%)	8 (2.2%)	7 (1.9%)	0 (0%)	8 (2.2%)	

Distribution of parameters used to assess physical activity and functional capacity across clinical groups: The table presents the number and percentage of patients according to WHO Functional Class (WHO-FC), along with mean ± standard deviation values of the 6-minute walk test (6MWT) results.

Table 4. Distribution of PAH-specific medical therapy modalities according to clinical classification

	None	Monotherapy	Sequential	Upfront	Total	p value
Group I	58 (14.3%)	34 (8.4%)	54 (13.3%)	19 (4.7%)	165 (40.6%)	
Group II	50 (12.3%)	0 (0%)	0 (0.0%)	0 (0.0%)	50 (12.3%)	
Group III	28 (6.9%)	9 (2.2%)	6 (1.5%)	0 (0.0%)	43 (10.6%)	
Group IV	29 (7.2%)	22 (5.4%)	14 (3.5%)	3 (0.7%)	68 (16.8%)	
Group V	0 (0.0%)	4 (1.0%)	3 (0.7%)	0 (0.0%)	7 (1.7%)	
Combined Group	32 (7.9%)	18 (4.4%)	16 (4.0%)	6 (1.5%)	72 (17.8%)	
Total	197 (48.6%)	87 (21.5%)	93 (23%)	28 (6.9%)	405 (100%)	<0.001

The distribution of PAH-specific pharmacological treatment modalities across clinical classification groups is presented as patient numbers and corresponding percentages based on crosstab analysis. The term 'combined group' refers to patients in whom features from multiple clinical classifications are concurrently prominent, and in whom clinical and hemodynamic findings suggest a multifactorial etiology that justifies grouping under more than one PAH classification due to the coexistence of multiple dominant clinical features.

Table 5. Distribution of PH clinical groups according to the ESC/ERS three-strata risk stratification

	Group I	Group II	Group III	Group IV	Group V	Combined Group	Total	p value
Low Risk	22 (5.4%)	4 (1.0%)	8 (2.0%)	7 (1.7%)	2 (0.5%)	8 (2%)	51 (12.6%)	
Intermediate Risk	114 (28.1%)	32 (7.9%)	25 (6.2%)	43 (10.6%)	4 (1.0%)	46 (11.4%)	264 (65.2%)	
High Risk	29 (7.2%)	14 (3.5%)	10 (2.5%)	18 (4.5%)	1 (0.2%)	18 (4.5%)	90 (22.3%)	
Total	165 (40.7%)	51 (12.3%)	43 (10.6%)	68 (16.8%)	7 (1.7%)	72 (17.8%)	405 100.0%	0.609

The number of patients (and corresponding percentages relative to the total population) classified according to pulmonary hypertension (PH) clinical groups and matched with the 2022 ESC/ERS three-strata risk stratification system is presented using cross-tabulation analysis.

Table 6. Directly measured hemodynamic variables obtained via cardiac catheterization and their distribution by clinical groups

	Total	Group I	Group II	Group III	Group IV	Group V	Combined	p value
PCWP	12.4 ± 6.0	10.82 ± 4.2	19.15 ± 6.4	10.44 ± 4.7	10.15 ± 3.7	8.86 ± 2.1	15.15 ± 7.3	<0.001
mPAP	41.61 ± 16.4	45.36 ± 18.9	35.81 ± 11.7	38.24 ± 14.1	41.69 ± 14.7	36.29 ± 10.3	40.13 ± 14.7	0.003
sPAP	66.22 ± 26.1	71.06 ± 28.1	55.71 ± 20.2	60.59 ± 22.5	69.54 ± 26.6	59.00 ± 17.6	64.36 ± 24.2	0.003
dPAP	26.36 ± 12.3	28.84 ± 14.7	23.65 ± 8.1	24.73 ± 10.5	24.88 ± 11.1	23.00 ± 9.4	25.58 ± 10.3	0.041
RV (es)	67.2 ± 26.7	73.15 ± 28.2	56.58 ± 20.2	60.46 ± 22.6	69.60 ± 29.0	58.57 ± 18.0	64.54 ± 24.2	0.001
RV (ed)	9.91 ± 5.4	9.24 ± 4.2	11.85 ± 5.8	8.10 ± 4.5	9.33 ± 4.7	7.14 ± 4.2	11.92 ± 7.3	<0.001
RAP	9.51 ± 5.6	8.55 ± 4.3	11.75 ± 5.8	7.95 ± 4.5	8.67 ± 4.5	6.71 ± 3.9	12.06 ± 7.8	<0.001
SBP	139.75 ± 24.3	137.18 ± 22.5	145.31 ± 29.3	139.31 ± 22.1	139.62 ± 23.6	148.00 ± 35.6	141.10 ± 25.0	0.367
DBP	69.66 ± 10.7	69.99 ± 10.4	68.30 ± 12.0	69.72 ± 11.3	70.02 ± 9.4	66.86 ± 9.8	69.96 ± 11.7	0.909
MAP	93.75 ± 14.1	93.23 ± 13.7	94.04 ± 16.3	93.00 ± 14.0	93.88 ± 12.5	94.14 ± 18.6	95.07 ± 14.9	0.968
LV (es)	139.25 ± 24.3	136.84 ± 21.7	147.50 ± 32.6	137.73 ± 21.2	137.36 ± 20.7	150.00 ± 38.6	141.00 ± 26.2	0.128
LV (ed)	11.79 ± 5.1	10.78 ± 3.5	17.07 ± 6.5	10.61 ± 4.3	10.35 ± 3.9	8.50 ± 2.4	12.92 ± 6.2	<0.001
SaO_2	92.0 ± 6.9	92.2 ± 6.7	95.1 ± 3.4	87.3 ± 10.7	91.5 ± 6.4	93.3 ± 3.2	92.1 ± 5.7	<0.001
SvO_2	65.9 ± 10.9	68.1 ± 10.3	65.8 ± 9.0	64.0 ± 12.4	64.6 ± 10.2	66.9 ± 8.1	62.9 ± 12.5	0.023

The mean ± standard deviation of hemodynamic parameters obtained through cardiac catheterization are shown in Table 6. PCWP: Pulmonary Capillary Wedge Pressure, mPAP: mean Pulmonary Arterial Pressure, sPAP: systolic Pulmonary Arterial Pressure, dPAP: diastolic Pulmonary Arterial Pressure, RV (es): End-systolic right ventricular pressure, RV (ed): End-diastolic right ventricular pressure, RAP: Right atrial pressure, SBP: Systolic Blood Pressure measured via Aorta, DBP: Diastolic Blood Pressure, MAP: Mean Arterial Pressure, LV (es): End-systolic left ventricular pressure, LV (ed): End-diastolic left ventricular pressure, SaO2: Arterial oxygen saturation, SvO2: Mixed venous oxygen saturation. All pressure variables are expressed in "mmHg" while oxygen saturations are presented as percentage, %.

Table 7. Derived hemodynamic variables and their distribution by clinical groups

	Total	Group I	Group II	Group III	Group IV	Group V	Combined	p value
CO (Fick)	5.07 ± 1.91	5.32 ± 2.31	4.70 ± 1.46	5.03 ± 1.48	5.00 ± 1.87	4.61 ± 1.26	4.92 ± 1.45	0.351
CI	2.85 ± 1.03	3.04 ± 1.26	2.62 ± 0.71	2.78 ± 0.76	2.73 ± 0.94	2.86 ± 0.70	2,73 ± 0.79	0.070
SV	63.96 ± 28.45	67.08 ± 30.81	62.46 ± 30.27	58.56 ± 19.98	64.70 ± 30.41	55.43 ± 19.82	61.67 ± 24.37	0.469
SVi	35.89 ± 15.15	38.41 ± 16.67	34.62 ± 15.17	32.26 ± 10.21	35.18 ± 15.78	34.23 ± 11.10	34.27 ± 13.31	0.154
PVR	6.67 ± 5.24	7.72 ± 6.19	3.90 ± 2.38	6.27 ± 3.87	7.48 ± 5.04	6.37 ± 2.98	5.93 ± 4.76	<0.001
PVRi	11.64 ± 8.66	13.13 ± 9.7	6.90 ± 4.09	11.31 ± 7.27	13.51 ± 9.03	10.08 ± 4.38	10.55 ± 8.07	<0.001
SVR	18.42 ± 6.55	18.41 ± 7.36	18.83 ± 6.66	17.90 ± 5.61	18.67 ± 6.11	19.55 ± 4.17	17.98 ± 5.73	0.961
TPG	28.93 ± 16.8	34.09 ± 19.1	16.65 ± 8.8	27.80 ± 13.3	31.54 ± 15.0	27.43 ± 9.8	24.97 ± 14.0	<0.001
RAP / PCWP	0.814 ± 0.42	0.838 ± 0.39	0.628 ± 0.25	0.773 ± 0.32	0.898 ± 0.45	0.796 ± 0.52	0.847 ± 0.53	0.015
PAPP	39.86 ± 18.6	42,22 ± 18.9	32.06 ± 15.8	35.85 ± 14.7	44.66 ± 21.4	36.00 ± 13.4	38.78 ± 17.0	0.002
PACi	1.132 ± 0.80	1.093 ± 0.75	1.408 ± 1.12	1.107 ± 0.70	1.020 ± 0.72	1.007 ± 0.31	1.131 ± 0.79	0.162
PAPi	5.41 ± 3.85	6.11 ± 4.05	3.23 ± 1.90	5.74 ± 3.92	6.32 ± 4.69	6.97 ± 3.98	4.29 ± 2.61	<0.001
RV-SWi	15.52 ± 11.2	19.59 ± 15.2	10.72 ± 6.0	12.89 ± 5.0	15.02 ± 6.8	13.75 ± 6.0	12.40 ± 6.0	<0.001
RV Power	0.459 ± 0.24	0.531 ± 0.32	0.366 ± 0.15	0.419 ± 0.16	0.443 ± 0.18	0.369 ± 0.14	0.418 ± 0.14	<0.001
LV-SWi	40.31 ± 19.1	43.50 ± 20.8	35.99 ± 16.2	36.37 ± 13.7	40.49 ± 20.3	41.31 ± 20.6	38.44 ± 18.0	0.110
LV power	1.070 ± 0.45	1.117 ± 0.53	0.990 ± 0.33	1.052 ± 0.39	1.060 ± 0.45	0.991 ± 0.41	1.053 ± 0.35	0.613

The mean ± standard deviation values of hemodynamic parameters derived from cardiac catheterization are presented in Table 7, stratified by clinical classification. CO: Cardiac Output calculated by Fick Method (L/min), CI: Cardiac Index (L/min·m^2), SV: Stroke Volume (mL/beat), SVi: Stroke Volume index (mL/m^2), PVR: Pulmonary Vasculary Resistance (WU), PVRi: Pulmonary Vasculary Resistance index (WU·m^2), SVR: Systemic Vasculary Resistance (WU), SVRi: Systemic Vasculary Resistance index (WU·m^2), TPG: Transpulmonary Gradient (mmHg), RAP / PCWP: Right Atrial to Left Atrial Pressure ratio, PAPP: Pulmonary Arterial Pulse Pressure (mmHg), PACi: Pulmonary Arterial Compliance index (mL/mmHg·m^2), PAPi: Pulmonary Arterial Pulsatility index (PAPP/RAP ratio), RV-SWi: Right Ventricular Stroke Work index (J·m^2), LV-SWi: Left Ventricular Stroke Work index (J·m^2), RV power: Right Ventricular power (J), LV-power: Left Ventricular power (J).

Table 8. Univariate Cox regression analysis of hemodynamic variables

	HR	%95 CI	p value		HR	%95 CI	p value
PCWP	1.021	0.999-1.043	0.056	CI	0.743	0.649-0.851	<0.001
mPAP	1.006	0.999-1.012	0.099	SV	0.989	0.984-0.994	<0.001
sPAP	1.004	1.000-1.009	0.043	SVi	0.979	0.970-0.988	<0.001
dPAP	1.005	0.996-1.014	0.289	PVR	1.033	1.014-1.053	0.001
RV (es)	1.003	0.999-1.008	0.115	PVRi	1.024	1.012-1.037	<0.001
RV (ed)	1.056	1.033-1.079	<0.001	SVR	1.015	0.997-1.034	0.104
RAP	1.061	1.038-1.084	<0.001	PAC	0.826	0.743-0.919	<0.001
SBP	0.994	0.989-1.000	0.039	PACi	0.714	0.589-0.867	0.001
MAP	0.988	0.979-0.998	0.020	RAP / PCWP	1.742	1.347-2.253	<0.001
LV (es)	0.993	0.987-0.999	0.014	PAPi	0.978	0.944-1.014	0.228
PAPP	1.007	1.001-1.013	0.027	RV-SWi	0.981	0.969-0.994	0,003
TPG	1.005	0.998-1.011	0.168	RV Power	0.573	0.349-0.939	0.027
SaO_2	0.954	0.939-0.970	<0.001	LV-SWi	0.981	0.974-0.989	<0.001
SvO_2	0.955	0.944-0.966	<0.001	LV power	0.499	0.364-0.683	<0.001
CO (Fick)	0.855	0.796-0.919	<0.001				

HR: Hazard ratio; 95% CI: 95% confidence interval indicating the lower and upper bounds of the HR. A p-value less than 0.05 was considered statistically significant. Abbreviations: PCWP: Pulmonary Capillary Wedge Pressure, mPAP: mean Pulmonary Arterial Pressure, sPAP: systolic Pulmonary Arterial Pressure, dPAP: diastolic Pulmonary Arterial Pressure, RV (es): End-systolic right ventricular pressure, RV (ed): End-diastolic right ventricular pressure, RAP: Right atrial pressure, SBP: Systolic Blood Pressure measured via Aorta, DBP: Diastolic Blood Pressure, MAP: Mean Arterial Pressure, LV (es): End-systolic left ventricular pressure, LV (ed): End-diastolic left ventricular pressure, SaO2: Arterial oxygen saturation, SvO_2: Mixed venous oxygen saturation. CO: Cardiac Output calculated by Fick Method (L/min), CI: Cardiac Index (L/min·m^2), SV: Stroke Volume (mL/beat), SVi: Stroke Volume index (mL/m^2), PVR: Pulmonary Vasculary Resistance (WU), PVRi: Pulmonary Vasculary Resistance index (WU·m^2), SVR: Systemic Vasculary Resistance (WU), SVRi: Systemic Vasculary Resistance index (WU·m^2), TPG: Transpulmonary Gradient (mmHg), RAP / PCWP: Right Atrial to Left Atrial Pressure ratio, PAPP: Pulmonary Arterial Pulse Pressure (mmHg), PACi: Pulmonary Arterial Compliance index (mL/mmHg·m^2), PAPi: Pulmonary Arterial Pulsatility index (PAPP/RAP ratio), RV-SWi: Right Ventricular Stroke Work index (J·m^2), LV-SWi: Left Ventricular Stroke Work index (J·m^2), RV power: Right Ventricular power (J), LV-power: Left Ventricular power (J).

Table 9. Univariate and multivariate logistic regression analysis of all variables in entire PH cohort

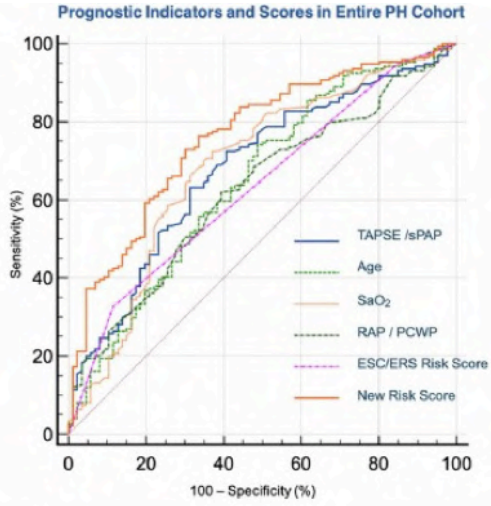
	HR	95% CI	p value	HR	95% CI	p value
2022 ESC/ERS 3-strata Risk Score	6.158	3.468 - 10.936	<0.001			
TAPSE / sPAP	0.103	0.022-0.492	0.004	0.040	0.003-0.608	0.020
Age	1.032	1.017-1.047	<0.001	1.051	1.027-1.077	<0.001
Female Gender	0.416	0.244-0.708	0.001	3.622	1.564-8.389	0.003
BMI	0.985	0.951-1.021	0.410			
Atrial Fibrillation	0.871	0.456-1.662	0.675			
Diabetes Mellitus	1.070	0.600-1.911	0.818			
GFR (CKD – EPI)	0.978	0.969-0.987	<0.001			
NT-proBNP	1.000	1.000-1.000	0.332			
6-MWT	0.995	0.993-0.997	<0.001			
WHO – FC II	1.227	0.349-4.316	0.750			
WHO – FC III	3.048	0.890-10.439	0.076			
WHO – FC IV	38.400	3.784-389.706	0.002			
mPAP	1.008	0.993-1.023	0.288			
MAP	0.984	0.969-1.000	0.048			
LV (ed)	1.022	0.972-1.074	0.396			
RAP	1.079	1.028-1.131	0.002			
PAPP	1.008	0.995-1.021	0.209			
RAP / PCWP	3.332	1.700-6.528	<0.001	4.113	1.246-13.582	0.020
PAPi	0.964	0.911-1.019	0.192			
PCWP	1.001	0.964-1.039	0.961			
PVR	1.056	1.005-1.109	0.032			
CI	0.743	0.587-0.941	0.014			
SvO_2	0.942	0.918-0.967	<0.001	0.947	0.911-0.985	0.007
PACİ	0.711	0.490-1.033	0.073			
RV power	0.473	0.193-1.163	0.103			
LV power	0.496	0.283-0.868	0.014			
RV-SWi	0.979	0.959-0.999	0.037			
LV-SWi	0.985	0.972-0.998	0.021			

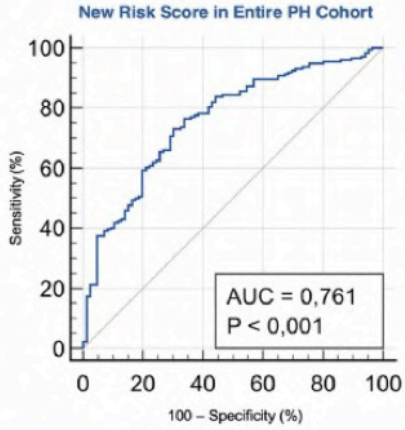
Table 9 presents the univariate and multivariate logistic regression analyses performed to identify independent predictive variables of mortality in the entire PH cohort. The analysis was conducted using a selected dataset derived from baseline characteristics, biochemical laboratory parameters, echocardiographic findings, and catheter-derived hemodynamic measurements. HR: Hazard ratio; 95% CI: 95% confidence interval indicating the lower and upper bounds of the HR. A p-value less than 0.05 was considered statistically significant. Abbreviations: ESC/ERS: European Society of Cardiology / European Respiratory Society, TAPSE / sPAP: Tricuspid Annular Plane Systolic Excursion / systolic Pulmonary Arterial Pressure, BMI: Body Mass Index, GFR (CKD - EPI): Glomerular Filtration Rate (Chronic Kidney Disease Epidemiology Collaboration), NT-proBNP: N-terminal pro b-type natriuretic peptide, 6-MWT: 6-minute walking test, WHO - FC: World Health Organization Functional Classification, mPAP: mean Pulmonary Arterial Pressure, MAP: Mean Arterial Pressure, LV (ed): Left Ventricular end-diastolic pressure, RAP: Right Atrial Pressure, PAPP: Pulmonary Arterial Pulse Pressure, RAP / PCWP: Right Atrial to Left Atrial Pressure ratio, PAPi: Pulmonary Arterial Pulsatility index, PVR: Pulmonary Vascular Resistance, CI: Cardiac Index, SvO_2: Mixed Venous Oxygen Saturation, PACi: Pulmonary Arterial Compliance index, RV power: Right Ventricular power, LV power: Left Ventricular power, RV-SWi: Right Ventricle Stroke Work index, LV-SWi: Left Ventricle Stroke Work index.

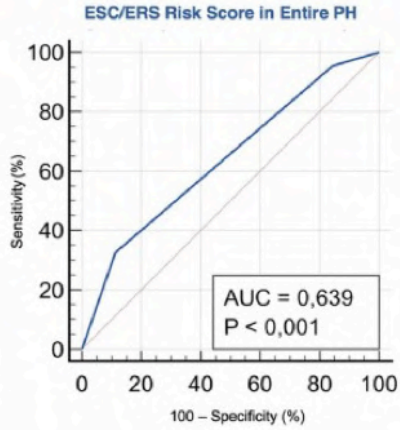
Table 10. Univariate and multivariate logistic regression analysis in PAH cohort

	HR	CI %95	p value	HR	CI 95%	p value
TAPSE/ sPAP	0.001	0.000-0.070	0.001	0.000	0.000-0.040	0.003
SvO_2	0.917	0.874-0.961	<0.001	0.952	0.822-0.884	0.001
LV (ed)	1.178	1.044-1.329	0,008	1.491	1.042-1.246	0.016
RAP / PCWP	2.674	1.029-6.947	0.044			
RAP	1.115	1.021-1.219	0.016			
CO	0.818	0.676-0.989	0.038			
CI	0.725	0.529-0.994	0.046			
SaO_2	0.914	0.852-0.979	0.011			
Age	1.033	1.012-1.055	0.002			
6-MWT	0.995	0.991-0.998	0.001			
WHO – FC IV	2.250	0.111-45.723	0.598			
NT-proBNP	1.000	1.000-1.000	0.072			
GFR (CKD – EPI)	0.981	0.968-0.995	0.007			

Table 10 shows the univariate and multivariate logistic regression analysis of selected parameters hypothesized to be mortality predictors in patients with pulmonary arterial hypertension (PAH). HR: Hazard ratio; 95% CI: 95% confidence interval indicating the lower and upper bounds of the HR. A p-value less than 0.05 was considered statistically significant.



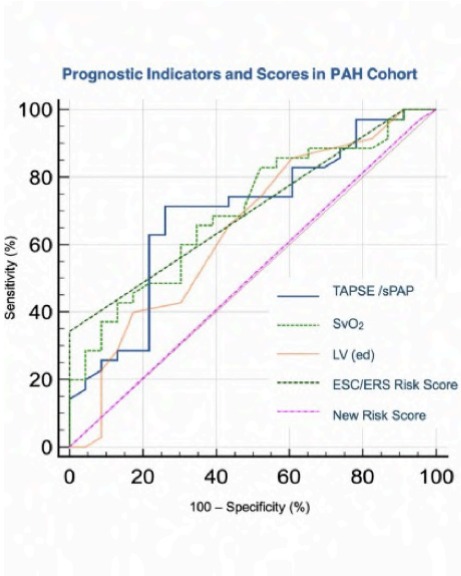


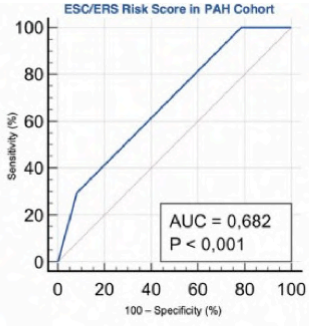


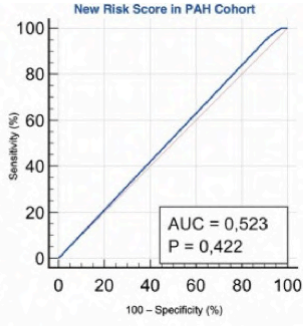
Variables		DBA	95% CI	p value
TAPSE/sPAP	New Risk Score	0,081	0,008 - 0,154	0,028
Age		0,113	0,051 - 0,175	<0,001
SaO_2		0,074	0,012 - 0,137	0,019
RAP /PCWP		0,135	0,059 - 0,212	<0,001
ESC/ERS Risk Score		0,128	0,066 - 0,189	<0,001
	TAPSE/sPAP	0,046	-0,01 - 0,113	0,167
	Age	0,014	-0,06 - 0,09	0,736
	SaO_2	0,053	-0,02 - 0,133	0,186
	RAP /PCWP	0,007	-0,07 - 0,09	0,858
De Long, Binomial Exact				

	Sensitivity (%)	Specificity (%)
New Risk Score	**72,99**	**69,77**
2022 ESC/ERS 3-strata Risk Score	**32,58**	**88,70**
Youden Index		

Graphic 1. ROC curve analysis in entire PH cohort. A new risk score was constructed by multiplying the variables identified as independent predictors in the multivariate logistic regression analysis — including the TAPSE/sPAP ratio, arterial oxygen saturation (SaO_2), RAP/PCWP ratio, and age — by their respective regression coefficients. The discriminative performance of the derived risk score within the entire PH cohort was assessed using receiver operating characteristic (ROC) curve analysis. In the same context, the diagnostic sensitivity and specificity of the ESC/ERS three-strata risk stratification model were also evaluated and comparatively analyzed. The table presents the differences between the areas (DBA) under the curve (ΔAUC) for prognostic indicators, along with their 95% confidence intervals and corresponding p-values.

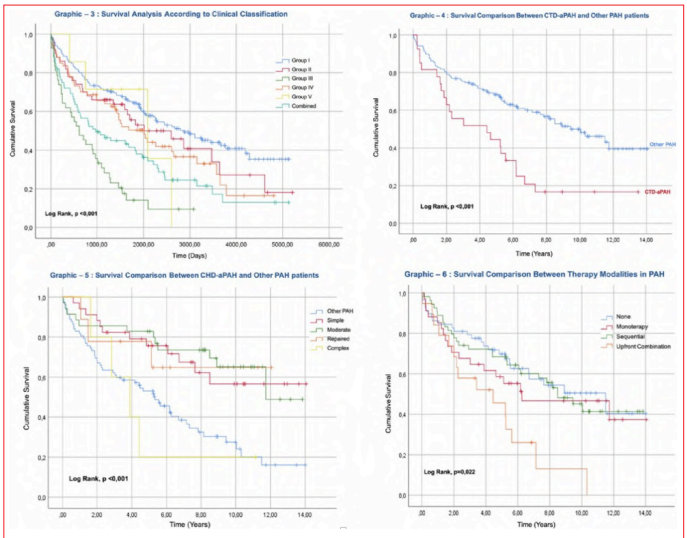




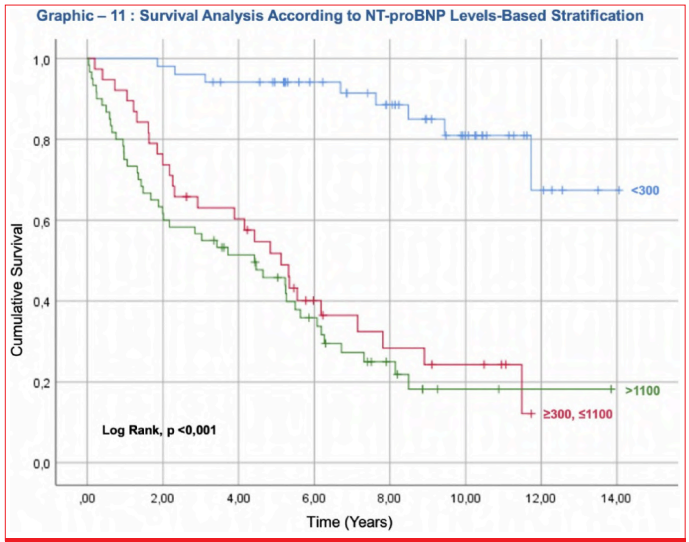


Variable	AUC	CI 95% [a]	p value
TAPSE /sPAP	0,694	0,559 - 0,808	
SvO_2	0,699	0,565 - 0,813	
LV (ed)	0,645	0,508 - 0,766	
ESC/ERS 3-strata Risk Score	0,682	0,597 - 0,758	<0,001
Group I New Risk Score	0,523	0,373 - 0,641	0,422
[a] Binomial exact			

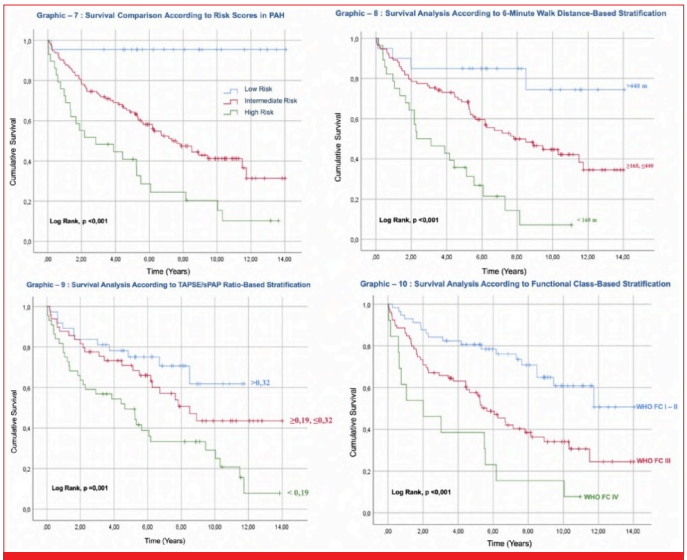
Graphic 2. ROC curve analysis in PAH cohort. Receiver operating characteristic (ROC) curves are provided to assess the diagnostic accuracy of the newly derived risk score—based on prognostic indicators of mortality within the PAH cohort—as well as that of the ESC/ERS three-strata risk stratification model applied to the same patient population. The table presents the areas under the curve (AUC) for predictors of mortality and risk scores, along with their 95% confidence intervals and corresponding p-values.



Graphic 3-4-5-6. Kaplan–Meier survival analysis stratified by clinical classification and PAH-specific treatment modalities. Graphic 3: The mean time to all-cause mortality across clinical groups was, respectively: 2906, 2489, 900, 2170, 1838, and 1721 days, from Group 1 to the combined group. In the overall PH cohort, the mean time to the primary endpoint was calculated as 2361 ± 106 days (log-rank test, p < 0.001), corresponding to approximately 6.5 years. When survival differences among PH clinical subgroups were assessed, Group 3 exhibited the poorest prognosis, with a mean survival of 900 ± 144 days. In contrast, isolated Group 1 patients demonstrated a more favorable course, with a median survival of 7.63 years. Graphic 4: During the follow-up period, the primary endpoint of all-cause mortality occurred in 81.5% of patients with CTD-associated PAH (CTD-aPAH), who exhibited a more unfavorable prognosis within the PAH cohort with a mean survival of 4.9 ± 0.8 years.

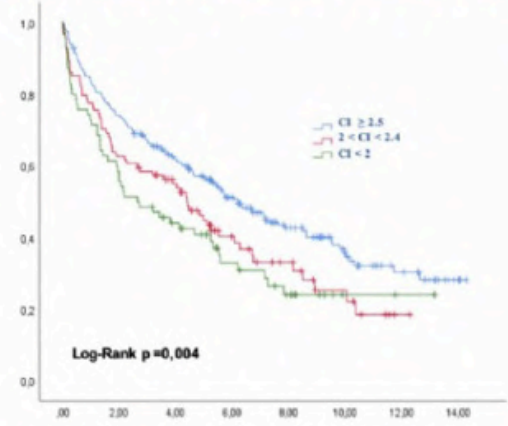


Graphic 11. Survival analysis according to NT-proBNP levels-based stratification. Survival analysis based on NT-proBNP–derived risk stratification revealed that, although intermediate- and high-risk groups had comparable long-term outcomes, patients with NT-proBNP levels below 300 ng/L demonstrated a clear prognostic advantage. The low-risk group exhibited a 1-year mortality rate of only 2%, with a mean survival of 12.2 ± 0.5 years. While first-year mortality reached 8% in the intermediate-risk group and 25% in the high-risk group, the absence of significant differences between these groups over the entire follow-up period highlights the importance of early-phase mortality risk. These findings suggest that NT-proBNP levels <300 ng/L at diagnosis may serve as a key prognostic determinant in patients with PAH.

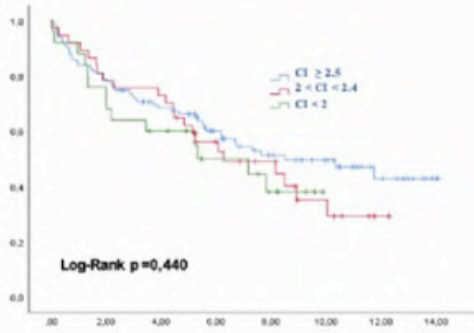


Graphic 7-8-9-10. Survival comparisons based on individual variables comprising the risk stratification model. Within the PAH cohort, patients were stratified into low-, intermediate-, and high-risk groups, and survival analysis based on time to all-cause mortality was performed using Kaplan–Meier curves. According to this analysis, WHO functional class, 6-minute walk test distance (6MWD), and the TAPSE/sPAP ratio were identified as significant determinants in differentiating survival among the groups. In accordance with this, the ESC/ERS three-strata risk stratification demonstrated prognostic utility in predicting survival within the PAH cohort.

Graphic – 12 : Survival Comparison According to CI-Based Stratification in Entire PH

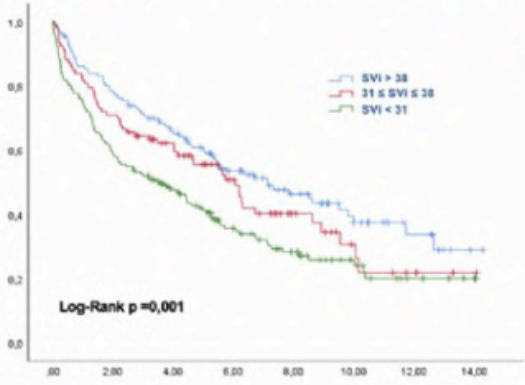


Graphic – 13 : Survival Comparison According to CI-Based Stratification in PAH

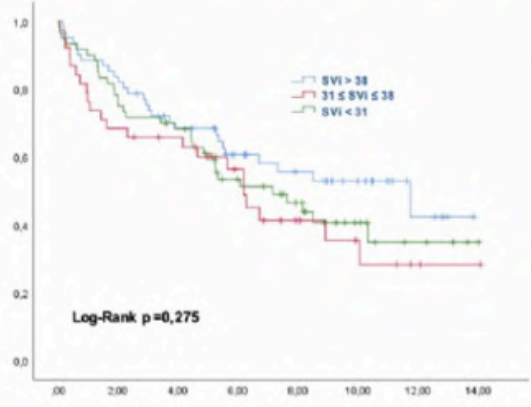


Time (years)	1	3	5	10	Median Survival	p value
Low Risk	%83	%67	%56	%34	6,1 ±0,6	0,004
Intermediate Risk	%77	%58	%44	%25	4,4 ±0,5	
High Risk	%72	%48	%40	%24	2,6 ±0,9	
The risk boxes carry the color of the curve they represent. The colors do not indicate the degree of risk.						

Graphic – 14 : Survival Comparison According to SVi-Based Stratification in Entire PH



Graphic – 15 : Survival Comparison According to SVi-Based Stratification in PAH

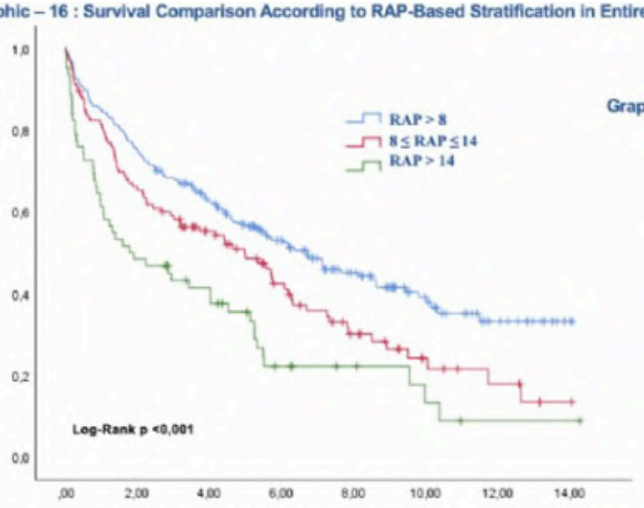


Time (years)	1	3	5	10	Median Survival	p value
Low Risk	%86	%71	%60	%37	7,1 ±1,3	0,001
Intermediate Risk	%81	%64	%55	%30	6,0 ±0,7	
High Risk	%74	%52	%41	%24	3,5 ±0,7	
The risk boxes carry the color of the curve they represent. The colors do not indicate the degree of risk.						

Graphic 12-13-14-15. Survival comparisons according to CI and SVi-based stratification in entire PH and PAH. In both the entire PH cohort and the PAH cohort, survival outcomes were evaluated based on risk stratification using hemodynamic parameters, as illustrated by Kaplan–Meier curves. When classified according to cardiac index into low-, intermediate-, and high-risk categories, the PH cohort demonstrated overall statistical significance; however, the separation between the intermediate- and high-risk groups was not clearly delineated. In contrast, this cardiac index–based stratification did not reach statistical significance within the PAH patients. Similarly, risk stratification based on stroke volume index demonstrated statistical significance within the overall PH cohort; however, it was not consistently effective in predicting outcomes or discriminating between risk groups throughout the entire follow-up period.

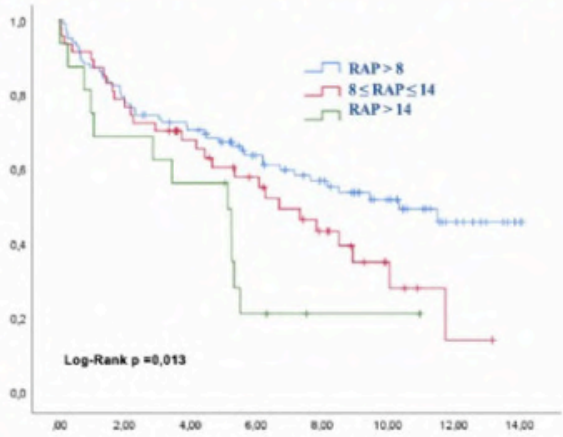
 Anatol J Cardiol 2025; 29 (Suppl 1): S1-S143 / doi: 10.14744/AnatolJCardiol.2025.102025

Graphic – 16 : Survival Comparison According to RAP-Based Stratification in Entire PH

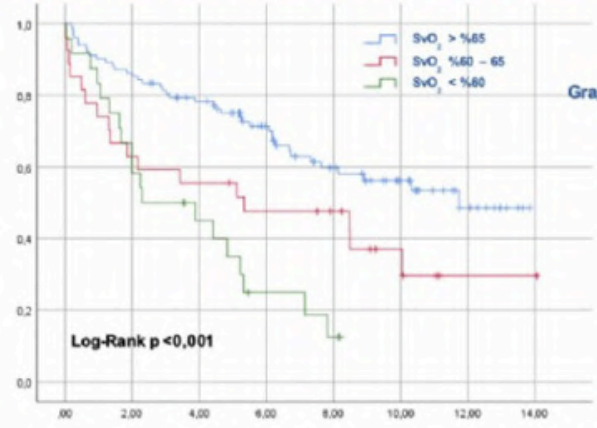


Time (years)	1	3	5	10	Median Survival	p value
Low Risk	%85	%68	%56	%37	6,7 ±0,8	<0,001
Intermediate Risk	%81	%59	%48	%24	4,9 ±0,9	
High Risk	%61	%43	%35	%13	1,7 ±0,8	
The risk boxes carry the color of the curve they represent. The colors do not indicate the degree of risk.						

Graphic – 17 : Survival Comparison According to RAP-Based Stratification in PAH

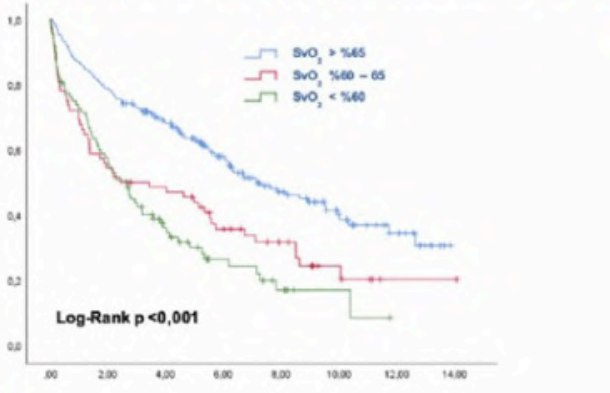


Graphic – 18 : Survival Comparison According to SvO_2-Based Stratification in PAH



Time (years)	1	3	5	10	Median Survival	p value
Low Risk	%90	%81	%75	%56	11,7	<0,001
Intermediate Risk	%74	%59	%55	%37	5,3 ±3,7	
High Risk	%83	%50	%35	%12	2,3 ±1,4	
The risk boxes carry the color of the curve they represent. The colors do not indicate the degree of risk.						

Graphic – 19 : Survival Comparison According to SvO_2-Based Stratification in Entire PH



Graphic 16-17-18-19. Survival comparisons according to RAP and SvO2-based stratification in entire PH and PAH. Within the PAH cohort, mortality rates between the low- and intermediate-risk groups were similar during the first two years following diagnosis, with significant survival differences becoming apparent only with extended follow-up. Conversely, in the entire PH cohort, RAP-based risk stratification exhibited discriminative ability in survival differentiation from the time of diagnosis. Risk stratification based on mixed venous oxygen saturation (SvO_2) revealed that, in both cohorts, patients with SvO_2 values above 65%—classified as low risk—demonstrated better survival outcomes compared to those in the intermediate- and high-risk groups. Accordingly, SvO_2-based risk stratification appeared more effective in distinguishing low-risk patients from those at higher risk within our study population. Furthermore, differences in survival between the intermediate- and high-risk groups became apparent after a follow-up period exceeding two years.

OP-032 [Coronary Artery Disease / Acute Coronary Syndrome]

Comparison of the predictive performance of Mehran and NCDR CathPCI scores for contrast-induced nephropathy in STEMI patients

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Background and Aim: Contrast-induced nephropathy (CIN) is a significant complication in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI), leading to increased morbidity and mortality. Risk stratification tools such as the Mehran and NCDR CathPCI scores have been developed to predict CIN; however, their performance may vary across populations. This study aimed to compare the predictive accuracy of these two scoring systems in STEMI patients from two tertiary-care centers.

Methods: This retrospective cohort study included 851 STEMI patients who underwent primary PCI between 2020 and 2024 at two tertiary-care centers. CIN was defined as a ≥30% increase in serum creatinine at discharge compared to baseline. Mehran and CathPCI scores were calculated for each patient using their original algorithms: ● Mehran Score: Assigned points for hypotension (5), intra-aortic balloon pump use (5), congestive heart failure (5), age >75 years (4), anemia (3), diabetes mellitus (3), contrast media volume (1 per 100 mL), and baseline serum creatinine or estimated glomerular filtration rate (eGFR). ● CathPCI Score: Based on the original NCDR CathPCI Registry algorithm, this score incorporated patient-related variables (age, diabetes mellitus, anemia, chronic kidney disease or reduced eGFR, COPD, hypothension,and left ventricular ejection fraction) and procedural factors (use of intra-aortic balloon pump and emergent PCI). Weighted points were assigned to each variable to generate a composite risk score for CIN prediction. The predictive performance of both scores was assessed using receiver operating characteristic (ROC) curves and area under the ROC (AUROC) analysis with 95% confidence intervals (CIs). Optimal cut-off values were determined through sensitivity and specificity analyses.

Results: CIN occurred in 191 patients (22.4%). The mean Mehran score was significantly higher in the CIN group compared to the non-CIN group (11.4 ± 4.1 vs. 7.5 ± 3.2; p<0.001). Similarly, the CathPCI score was elevated in the CIN group (38.2 ± 9.1) versus the non-CIN group (27.6 ± 8.4; p<0.001) (Table 1). ROC analysis demonstrated that the CathPCI score had superior predictive performance over the Mehran score (AUROC: 0.82 [95% CI, 0.77–0.86] vs. 0.78 [95% CI, 0.73–0.83]; p=0.003). A CathPCI cut-off value of ≥33 achieved 78.9% sensitivity and 70.1% specificity, while a Mehran cut-off of ≥10 provided 75.6% sensitivity and 68.2% specificity (Figure 1).

Conclusions: Both Mehran and CathPCI scores demonstrated strong predictive ability for CIN in STEMI patients undergoing primary PCI. However, the CathPCI score outperformed the Mehran score in this high-risk population. Unlike prior studies favoring Mehran in non-ACS cohorts, these findings suggest that the CathPCI score may better reflect acute hemodynamic instability and renal function variability in STEMI. This highlights its potential clinical utility as an alternative risk stratification tool.

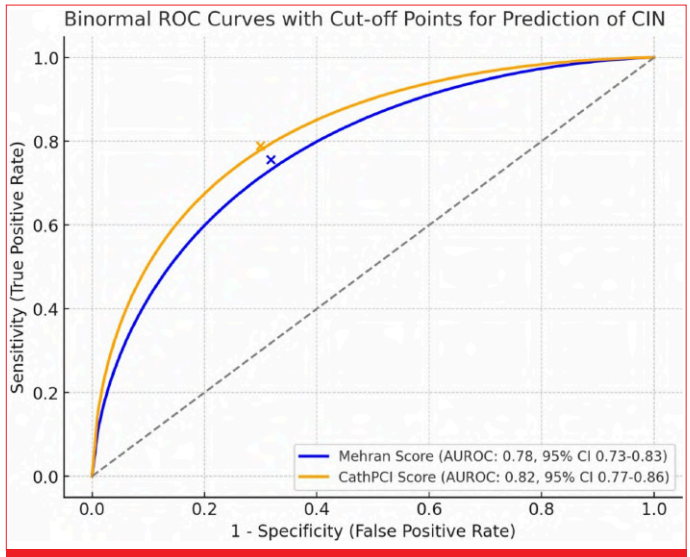

Figure 1. Comparison of binormal ROC curves for Mehran and CathPCI scores in predicting CIN.

Table 1. Comparison of binormal ROC curves for Mehran and CathPCI scores in predicting CIN

Variables	CIN (-) n=660	CIN (+) n=191	p value
Age	63.68 ± 11.1	64.68 ± 9.61	0.227
Sex (male %)	504 (76.4)	143 (74.9)	0.541
Smoking (%)	343 (52.0)	83 (43.5)	0.047
Hypertension (%)	313 (47.4)	93 (48.7)	0.821
Diabetes mellitus (%)	255 (38.6)	78 (40.8)	0.035
Chronic heart failure (%)	157 (23.8)	41 (21.5)	0.0568
CKD (%)	52 (7.9)	21 (11.0)	0.0429
AF (%)	13 (2.0)	6 (3.1)	0.492
COPD (%)	24 (3.6)	16 (8,4)	0.024
Baseline Creatinine (mg/dL)	1.0 (0.91-1.46)	1.06 (0.87-1.52)	0.023
Discharge Creatinine (mg/dL)	0.97 (0.81-1.48)	1.97 (1.48-2.4)	0.001
Glucose (mg/dL)	125.0 (96.0-174.0)	112.0 (91.0-159.05)	0.11
PLT (103/uL)	250.78 ± 84.27	247.34 ± 101.09	0.668
Albumin (g/dL)	40.3 ± 3.8	39.45 ± 4.8	0.025
Hemoglobin (g/dL)	13.61 ± 2.38	13.19 ± 2.15	0.021
Anemia (%)	123(18.70)	70 (36.50)	0.001
Hypotension (%)	853 (12.5)	52 (27.30)	0.001
Hear rate (bpm)	74.36 ± 17.07	76.6 ± 17.46	0.112
LDL (mg/dL)	115.87 ± 35.05	110.64 ± 36.06	0.083
HDL (mg/dL)	41.46 ± 7.68	41.78 ± 7.14	0.598
Mehran score	7.5 ± 3.5	11.4 ± 4.1	0.001
CathPCI score	27.6 ± 4.4	38.2±9.1	0.001
IABP (%)	21 (3.20)	17 (8.90)	0.001
Cardiac arrest (%)	16 (2.4)	7 (6.6)	0.001
Contrast Volume (mL)	180.00 ± 1.2	210.00 ± 1.8	0.001

OP-034 [Coronary Artery Disease / Acute Coronary Syndrome]

Effect of ticagrelor on left ventricular function in patients with mildly reduced ejection fraction after acute myocardial infarction

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Background and Aim: There are limited data about the effect of new P2Y12 inhibitors on left ventricular ejection fraction (LVEF) after acute myocardial infarction (AMI). We aimed to investigate the effect of ticagrelor on left ventricular function, compared to clopidogrel in patients with heart failure with mildly reduced ejection fraction (HFmrEF) after AMI.

Methods: In this cross-sectional, single-center study, we included 251 patients with LVEF between 40% and 50% after AMI before discharge. The patients were divided into 2 groups according to the use of ticagrelor (166 patients) and clopidogrel (85 patients). At the end of the 12-month period, LVEF changes were assessed by echocardiography. P<0.05 was considered statistically significant.

Results: The mean LVEF before discharge was 46.5% ± 3.6%, and no difference was observed between the ticagrelor and clopidogrel groups (p=0.20). At the end of the first year, the mean LVEF of the patients increased to 49.8% ± 7.6% in both groups. The use of ticagrelor (β ± SE=2.05 ± 0.93; p=0.029), low creatinine level (β ± SE=−10.44 ± 2.35; p<0.001), low troponin level (β ± SE=−0.38 ± 0.14; p=0.006), and low heart rate (β ± SE=−0.98 ± 0.33; p=0.003) were found to be independent predictors of the increase in LVEF (β ± SE 2.05 ± 0.93; 95% confidence interval: 0.21 to 3.90; p=0.029).

Conclusions: In our study, ticagrelor improved left ventricular function in 12 months follow-up compared to clopidogrel in patients with HFmrEF after AMI.

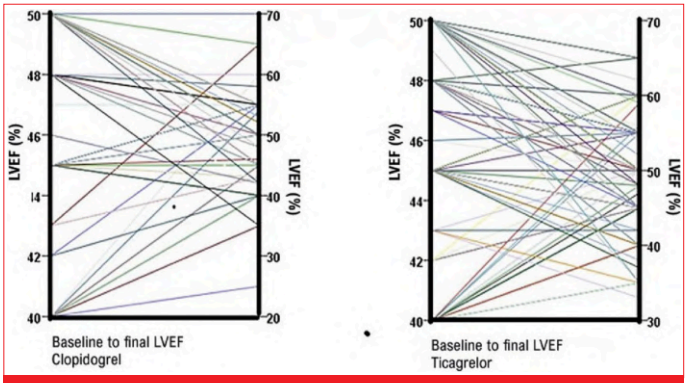

Figure 2. Change from baseline LVEF (%) to final LVEF (%).

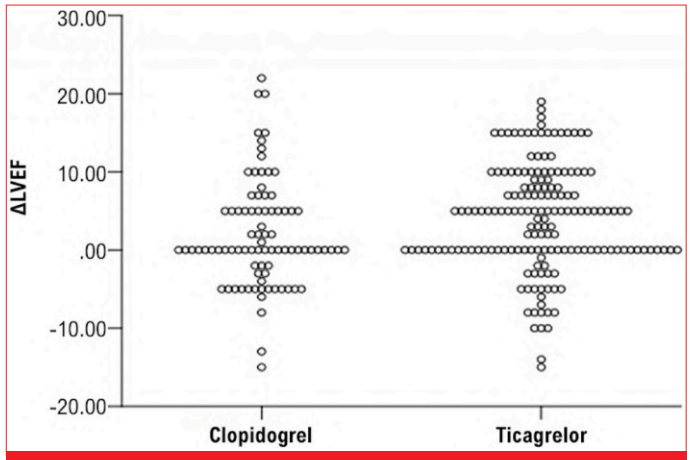

Figure 3. Distribution of LVEF (%) change according to treatment groups.

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

Spontaneous coronary artery dissection in a contemporary cohort of young women: A five-year single-center experience

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Background and Aim: Spontaneous coronary artery dissection (SCAD) is an uncommon but important cause of acute coronary syndrome (ACS) and sudden cardiac death (SCD) particularly in women under 50 years of age without traditional cardiovascular risk factors. It is defined as a non-traumatic, non-iatrogenic, non-atherosclerotic tear in the intimal layer of the coronary artery, formation of an intramural hematoma and compression of the true lumen, resulting in myocardial ischemia. Its clinical presentation ranges from ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) to ventricular arrhythmias, heart failure, and sudden cardiac death. Coronary angiography remains the main diagnostic tool. Most SCAD cases heal spontaneously, and conservative management is preferred in uncomplicated patients. Despite generally favourable long-term outcomes, recurrence remains a concern. The aim of this study was to evaluate the clinical presentation, angiographic features, management strategies, and outcomes of SCAD patients admitted to our institution.

Methods: We retrospectively reviewed female patients <51 years of age who underwent coronary angiography for suspected coronary events at Ankara Bilkent City Hospital between 2019 and 2025. All patients presented with ST-T segment changes and/or elevated high-sensitivity cardiac troponin levels. Angiograms were independently evaluated by three experienced interventional cardiologists. Clinical characteristics, angiographic findings, treatment strategies, and follow-up outcomes were analysed.

Results: Among 352 patients screened, 136 had normal coronary arteries, 165 had atherosclerotic coronary artery disease, 3 had intracoronary thrombus, 4 had coronary spasm, 1 had coronary aneurysm with vasculitis, 1 had anomalous pulmonary venous return, 1 had anomalous coronary artery, 1 had iatrogenic coronary dissection, and 40 were diagnosed with SCAD. SCAD cases were classified into types 1–4 using the Yip–Saw classification. Two patients underwent coronary artery bypass grafting; three patients were treated conservatively while the remainder were managed with percutaneous coronary intervention. During a five-year follow-up, only one patient required repeat revascularization. Two patients died: one due to cardiogenic shock at the index event and another from complications of heart failure.

Conclusions: SCAD is a relevant but underdiagnosed cause of ACS in young women without conventional risk factors. Because its clinical presentation is similar to atherosclerotic ACS, careful angiographic evaluation is crucial for diagnosis. Even though long-term survival is favourable, and mortality rates are low; clinical knowledge about its management is still lacking. To improve the clinical understanding and optimize the management strategies for this infrequent but important cause of mortality and morbidity in young women, close follow-up and randomized trials are highly anticipated.

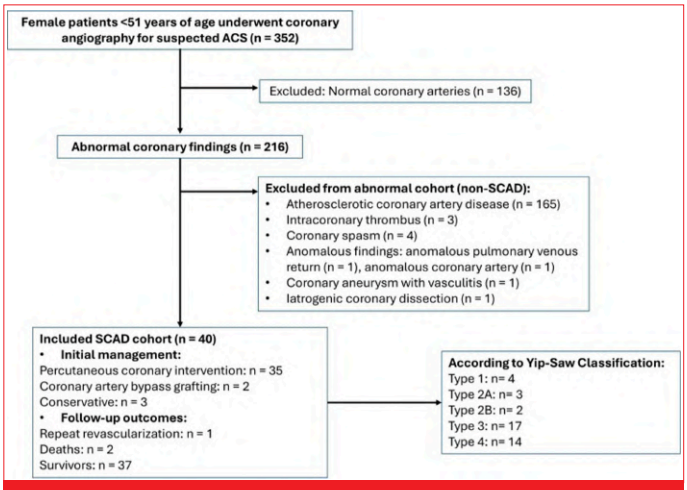

Figure 1. SCAD flowchart.

OP-036 [Coronary Artery Disease / Acute Coronary Syndrome]

The comparison of drug-coated balloons in acute coronary syndromes between younger and older populations

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Background and Aim: Coronary artery disease (CAD) is growing more common among younger people. According to WHO (World Health Organization) young coronary artery disease is defined as coronary illness that develops in patients under 45 years of age. Drug-coated balloon (DCB) has many advantages, including the absence of permanent struts, and non-inferiority clinical outcomes in patients with in-stent restenosis (ISR) and de novo small vessel disease. Furthermore, the increase of prevalence CAD in the younger population encouraged DCB by supporting stent-less PCI and leave-nothing-behind strategy. We aimed to compare the efficacy and safety of DCB in acute coronary syndromes between younger and older populations at early term.

Methods: This prospective study included 42 patients with acute coronary syndromes who underwent percutaneous coronary intervention with DCB) from September 2023 to Jun 2025 in Medicana International Ankara Hospital cardiology clinic. 12 of 42 patients had younger (≤45 years of age), and other patients (30 patients) had >45 years of age. ST-elevation myocardial infarction cases were excluded. Optimal lesion preparation was achieved using balloons with a balloon-to-vessel ratio of 0.8-1:1. DCB were inflated to their nominal pressure for at least 30 seconds.

Results: In younger patient group (12 patients) with acute coronary syndromes, mean age was 38 ± 4.3 years, 91.8% of patients were male, and 52.6% were diabetic. Mean vessels diameters were 2.73 ± 3.4 mm, lesion lengths were 28 ± 11.4 mm. The patients underwent DCB as sirolimus combined with phospholipids in 8 patients, sirolimus biodegradable polymer in the 2 patients), and paclitaxel coated in the 2 patients. One patient was exposed bail-out stenting due to > type C dissection. One patient had an ISR lesion, other patients had de-novo small- diffuse coronary lesions (≤3 mm). We performed DCB interventions in left anterior descending artery (3 patients), diagonal branch (2 patients), circumflex artery (3 patients), right coronary artery (4 patients). Five patients underwent the hybrid approach of combining DCB with drug eluting stents. In elderly group (30 patients) had mean age was 58 ± 4.3 years, 71.8% of patients were male, and 36.6% were diabetic. Mean vessels diameters were 2.71 ± 3.4 mm, lesion lengths were 34 ± 11.4 mm. The patients underwent DCB as sirolimus combined with phospholipids in 10 patients, sirolimus biodegradable polymer in the 4 patients), and paclitaxel coated in the 16 patients. Four patients were exposed bail-out stenting. Two patients had ISR lesions. Younger patients had less bail out stenting procedure ($p<0.05$). At 6-monh following time, cardiac death, MI, TLR, and stroke were not significantly different between the groups.

Conclusions: We observed that DCB interventions appeared effective and safe in younger patients with acute coronary syndromes small-diffuse coronary lesions.

OP-036A [Interventional Cardiology / Valvular and Structural Heart Disease]

Evaluation of the effectiveness of hemoglobin, albumin, lymphocyte and platelet score on in-hospital mortality after transcatheter aortic valve implantation

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Background and Aim: Transcatheter aortic valve implantation (TAVI) has emerged over the past two decades as an alternative treatment for patients with symptomatic and advanced aortic stenosis, particularly those at high surgical risk. Initially developed as an alternative to surgical aortic valve replacement (SAVR), TAVI has now become a treatment option with expanding indications, supported by strong clinical evidence demonstrating its efficacy and safety. Early mortality, generally defined as death occurring within 30 days of the procedure, remains a critical outcome in TAVI research. In this context, biomarkers such as the hemoglobin, albumin, lymphocyte, and platelet (HALP) score may be useful in predicting prognosis. The HALP score has emerged as an immune-nutritional biomarker that uses commonly measured parameters to provide a single composite score reflecting the patient's overall health status. The HALP score is calculated using the formula hemoglobin (g/L) × albumin (g/L) × lymphocytes (/L) / platelets (/L). Recent studies have also shown that the HALP score can predict mortality risk in patients with cardiovascular disease. The aim of this study was to investigate whether there is a relationship between HALP score and 30-day mortality rate in patients undergoing TAVI.

Methods: This retrospective study included 377 patients aged 18 years and older, 55.4% of whom were women, with advanced and symptomatic AS and anatomically suitable for transfemoral intervention who underwent elective percutaneous TAVI at a tertiary cardiac center between 2012 and 2022. Patients' HALP scores were recorded within 24 hours before the procedure. Laboratory values, complications, and mortality rates were recorded during the 30-day follow-up period.

Results: Forty-four (8.5%) of the patients died within 30 days. The HALP score is an independent factor affecting mortality (OR=0.000, CI: 0.000-0.015, p=0.007). ROC curve analysis of the HALP score for mortality prediction showed that a HALP score of 0.215 showed 84.1% sensitivity and 64.3% specificity, with an area under the curve (AUC) value of 0.816 (AUC=0.816, CI: 0.763–0.870, p<0.001).

Conclusions: Our study finds that the HALP score, which is cost-effective and easily obtained from laboratory data, is a significant predictor of increased 30-day mortality after TAVI.

Table 1. Baseline clinical and outcome characteristics by survival status

Variables	Survivors (n=333)	Non-Survivors (n=44)	P value	Variables	Survivors (n=333)	Non-Survivors (n=44)	P value
Gender (Female)	181 (54.4%)	28 (63.6%)	0.244	Any Bleeding	108 (32.4%)	26 (59.1%)	0.001
Age (years)	79.68 ± 7.618	81.68 ± 6.778	0.097	Minor Bleeding			<0.001
Hypertension	274 (82.3%)	39 (88.6%)	0.291	Grade 0	228 (68.5%)	17 (38.6%)	
Diabetes Mellitus	125 (37.5%)	20 (45.5%)	0.310	Grade 1	72 (21.6%)	9 (20.5%)	
Coronary Artery Disease	243 (73.0%)	34 (77.3%)	0.544	Grade 2	33 (9.9%)	18 (40.9%)	
Smoking	42 (12.6%)	2 (4.5%)	0.117	Overall Major Complications			<0.001
Sinus Rhythm	226 (67.9%)	27 (61.4%)	0.229	Grade 0	270 (81.1%)	13 (29.5%)	
STS Score	6.70 ± 5.10	7.90 ± 5.45	0.145	Grade 1	32 (9.6%)	4 (9.1%)	
EuroSCORE Logistic	20.61 ± 13.32	22.53 ± 11.66	0.362	Grade 2	31 (9.3%)	27 (61.4%)	
LVEF (%)	50.38 ± 11.54	49.77 ± 10.78	0.739	Hemoglobin Difference	1.38 ± 1.38	2.09 ± 1.47	0.002
Aortic Valve Area (cm^2)	0.717 ± 0.134	0.721 ± 0.146	0.839	Creatinine (mg/dL)	1.12 ± 0.65	1.72 ± 1.45	<0.001
Post-TAVI Atrial Fibrillation	99 (29.7%)	16 (36.4%)	0.369	Albumin (g/dL)	3.53 ± 0.55	2.51 ± 0.66	<0.001
Post-TAVI AV Block	39 (11.7%)	6 (13.6%)	0.711	Hemoglobin (g/dL)	11.51 ± 1.62	11.10 ± 1.80	0.123
Chronic Kidney Disease	31 (9.3%)	4 (9.1%)	0.963	Hematocrit (%)	34.84 ± 4.69	33.95 ± 5.08	0.243
Rehospitalization	26 (7.8%)	2 (4.5%)	0.438	Neutrophil Count ($\times10^3/\mu L$)	5.01 ± 3.78	8.16 ± 10.42	<0.001
Stroke	3 (0.9%)	3 (6.8%)	0.003	Lymphocyte Count ($\times10^3/\mu L$)	1.59 ± 0.79	1.39 ± 0.52	0.108
Length of Stay (days)	6.20 ± 4.49	5.59 ± 10.62	0.497	Platelet Count ($\times10^3/\mu L$)	220.5 ± 66.9	282.7 ± 77.1	<0.001
HALP Score	0.31 ± 0.18	0.14 ± 0.07	<0.001				

OP-037 [Heart Failure]

Comparative evaluation of the potential cardioprotective effects of finerenone and spironolactone against doxorubicin-induced cardiotoxicity in a rat model

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Background and Aim: Doxorubicin is an anthracycline chemotherapeutic agent whose use is limited by cardiotoxicity, ranging from asymptomatic myocardial injury to irreversible heart failure. Spironolactone, a steroidal mineralocorticoid receptor antagonist (MRA), offers cardioprotective benefits in various cardiomyopathies but is limited by adverse effects such as hyperkalemia. Finerenone, a novel non-steroidal MRA, has shown favorable cardiovascular and renal outcomes with a lower risk of hyperkalemia. However, its role in doxorubicin-induced cardiotoxicity remains uninvestigated. This study aims to compare the potential protective effects of spironolactone and finerenone in a rat model of doxorubicin-induced cardiotoxicity.

Methods: This study was approved by Baskent University Ethical Committee for Experimental Research on Animals (Project no: DA:23/23) and supported by Baskent University Research Fund. Forty-eight adult male Wistar rats were randomly assigned to six groups (n=8 each) and treated for 14 days. Group I received oral saline daily and intraperitoneal (i.p.) saline every other day. Group II received oral saline daily and doxorubicin 2.5 mg/kg i.p. every other day. Group III received oral spironolactone daily (25 mg/kg/day) and i.p. saline every other day. Group IV received oral finerenone daily (10 mg/kg/day) and i.p. saline every other day. Group V received oral spironolactone daily (25 mg/kg/day) and doxorubicin 2.5 mg/kg i.p. every other day. Group VI received oral finerenone daily (10 mg/kg/day) and doxorubicin 2.5 mg/kg i.p. every other day. Body weights were measured at baseline and before sacrification. Echocardiography was performed under ketamine/xylazine anesthesia. Serum cTnI and BNP levels were analyzed. Heart tissues were collected post-sacrification and examined by light microscopy and caspase-3 immunohistochemical staining to assess apoptosis.

Results: All groups exhibited weight loss, most notably in the doxorubicin-only group ($p<0.001$). Serum cTnI and BNP levels showed no clinically meaningful differences among groups. Echocardiographic analysis revealed significant impairment in the doxorubicin-only group ($p<0.001$), while co-treatment with spironolactone or finerenone preserved cardiac function without a clear advantage of one over the other. Histological examination showed mild myocardial changes in the doxorubicin group without statistical significance. Caspase-3 immunohistochemical staining showed significantly increased apoptosis in the doxorubicin-only group ($p=0.018$), and this increase was similarly reduced in the spironolactone and finerenone groups, with no significant difference between the two.

Conclusions: Both spironolactone and finerenone demonstrated protective effects against doxorubicin-induced cardiotoxicity, mitigating cardiac dysfunction and apoptosis similarly. Finerenone, with fewer side effects, may represent a favorable alternative. Further studies are needed to confirm clinical applicability.

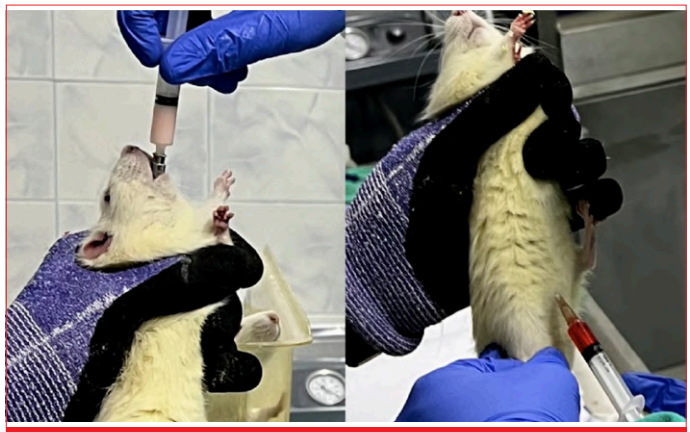


Figure 1. Methods 1, 2. Oral administration via gavage and intraperitoneal (IP) administration.

Figure 2. Methods 3. Echocardiographic evaluation performed using a vivid S60N (general electric) ultrasound device and a 12S-D (general electric) transducer.

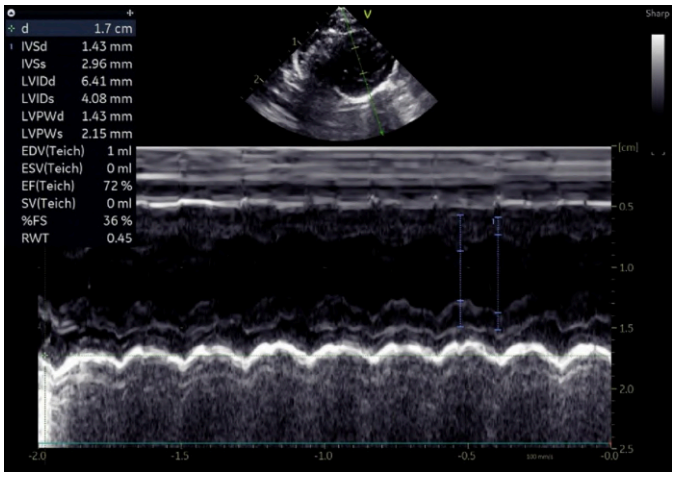


Figure 3. Methods 4. Calculation of left ventricular ejection fraction using the teicholz method via M-Mode echocardiographic evaluation from the parasternal short-axis view.

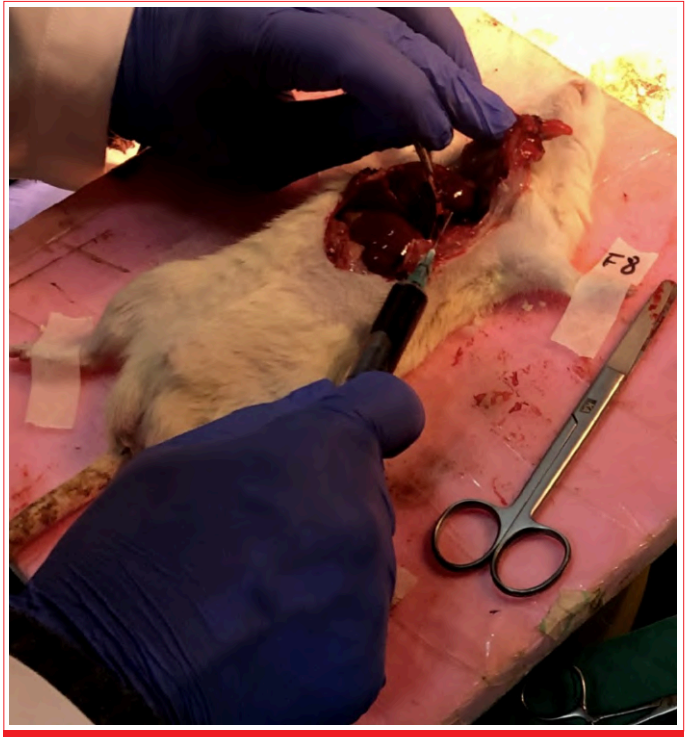


Figure 4. Methods 5. Collection of blood and tissue samples.

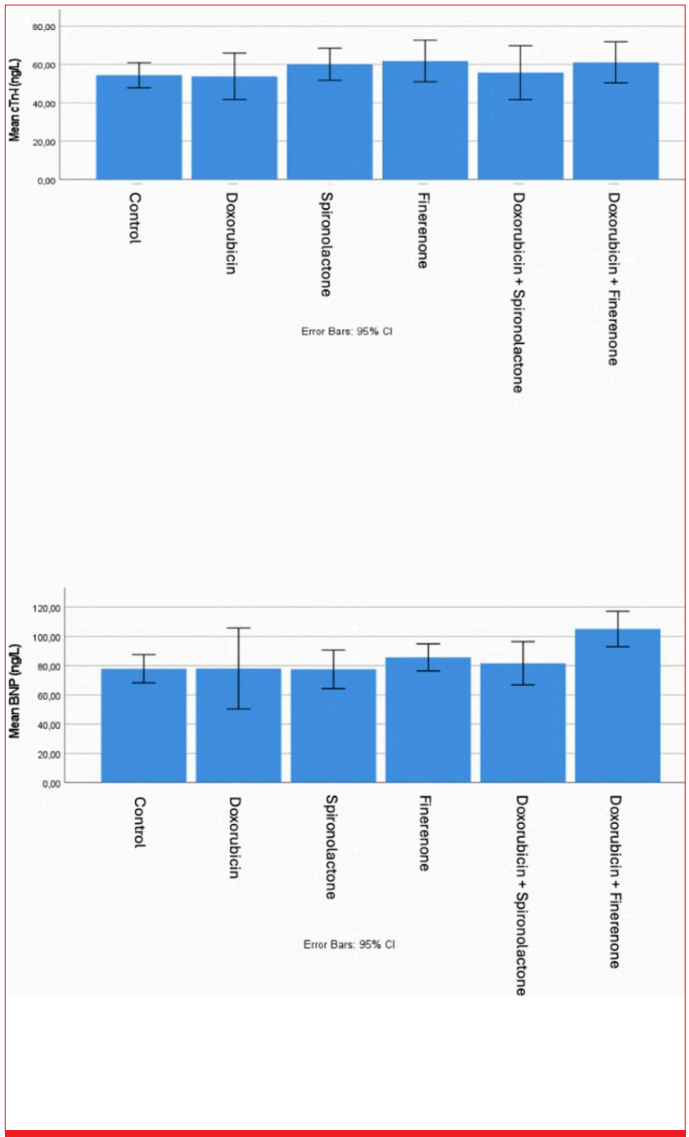


Figure 5. Results 1. Graphs of troponin and BNP results.

Table 1. Results 2. Table of echocardiographic results

	GROUP	Control (n=7)	Dox (n=5)	Sp (n=8)	Fin (n=7)	Dox+Sp (n=7)	Dox+Fin (n=8)	p-value
IVSd	Mean±SD	1,37±,04	1,31±,04	1,32±,03	1,37±,06	1,36±,17	1,35±,04	0,089
(mm)	Med(Min-Max)	1,39(1,3-1,43)	1,3(1,26-1,35)	1,32(1,25-1,35)	1,35(1,3-1,44)	1,39(1,03-1,57)	1,35(1,3-1,4)	
LVPWd	Mean±SD	1,53±,20	1,4±,10	1,46±,12	1,54±,05	1,46±,09	1,48±,04	0,142
(mm)	Med(Min-Max)	1,48(1,39-1,98)	1,44(1,26-1,52)	1,49(1,3-1,62)	1,53(1,48-1,61)	1,44(1,35-1,58)	1,49(1,44-1,53)	
LVIDd	Mean±SD	5,99±,47	5,21±,52	5,77±,25	5,78±,24	6,46±,62	5,68±,72	**0,005**
(mm)	Med(Min-Max)	5,98(5,23-6,58)	5,02(4,63-5,98)	5,84(5,37-6,08)	5,8(5,43-6,06)	6,52(5,56-7,37)	5,99(3,99-6,11)	
LVIDs	Mean±SD	3,72±,34	3,97±,35	3,57±,22	3,55±,25	4,51±,45	3,79±,54	**<0,001**
(mm)	Med(Min-Max)	3,73(3,02-4,08)	3,86(3,51-4,36)	3,57(3,15-3,85)	3,6(3,23-3,94)	4,5(3,95-5,26)	3,94(2,6-4,35)	
LVEF	Mean±SD	74±3,61	53,8±3,42	74,38±2,20	75±2,83	63,57±2,37	68,25±4,62	**<0,001**
(%)	Med(Min-Max)	72(70-79)	53(50-59)	74(72-78)	76(70-77)	63(61-68)	69,5(59-74)	
FS	Mean±SD	37,71±2,75	23,4±2,30	38,13±1,81	38,43±2,51	30,14±1,46	33,25±3,28	**<0,001**
(%)	Med(Min-Max)	36(35-42)	23(21-27)	38(36-41)	39(34-41)	30(29-33)	34(27-38)	
E/A	Mean±SD	1,93±,18	1,14±,42	1,84±,24	1,88±,37	1,44±,27	1,64±,44	**<0,001**
	Med(Min-Max)	1,9(1,74-2,26)	1,38(0,55-1,49)	1,97(1,41-2,1)	1,82(1,54-2,62)	1,38(1,16-1,84)	1,6(0,75-2,2)	
LA Diam.	Mean±SD	3,75±,22	4,54±,21	3,88±,09	3,75±,14	4,56±,39	4,03±,14	**<0,001**
(mm)	Med(Min-Max)	3,83(3,41-3,93)	4,62(4,24-4,77)	3,9(3,67-3,98)	3,75(3,5-3,92)	4,51(4,02-4,98)	4(3,91-4,3)	
RA Diam.	Mean±SD	3,49±,27	4,24±,25	3,63±,27	3,51±,19	4,13±,30	3,42±,31	**<0,001**
(mm)	Med(Min-Max)	3,52(3-3,83)	4,33(3,93-4,5)	3,69(3,26-3,92)	3,5(3,25-3,87)	4,11(3,75-4,74)	3,36(3,02-3,91)	
TAPSE	Mean±SD	2,7±,23	1,88±,03	2,42±,27	2,57±,19	2,34±,25	2,23±,15	**<0,001**
(mm)	Med(Min-Max)	2,83(2,33-2,93)	1,89(1,84-1,93)	2,34(2,16-2,93)	2,52(2,33-2,91)	2,31(2,12-2,83)	2,25(1,98-2,38)	

One-way ANOVA and Dunn's Bonferroni tests were used for data analysis.

n: number of subjects, Mean: average, SD: standard deviation, Med: median, Min: minimum, Max: maximum

Control: control group, Dox: doxorubicin group, Sp: spironolactone group, Fin: finerenone group, Dox+Sp: doxorubicin + spironolactone group, Dox+Fin: doxorubicin + finerenone group

IVSd: end-diastolic interventricular septal thickness, LVPWd: end-diastolic posterior wall thickness of the left ventricle, LVIDd: end-diastolic left ventricular diameter, LVIDs: end-systolic left ventricular diameter, FS: fractional shortening, LVEF: left ventricular ejection fraction, E/A: ratio of E and A waves from mitral inflow measured by pulsed Doppler method, LA Diam.: left atrium diameter, RA Diam.: right atrium diameter, TAPSE: tricuspid annular plane systolic excursion

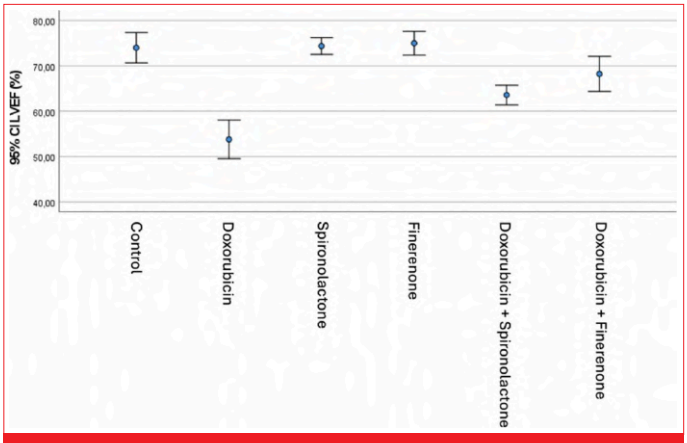
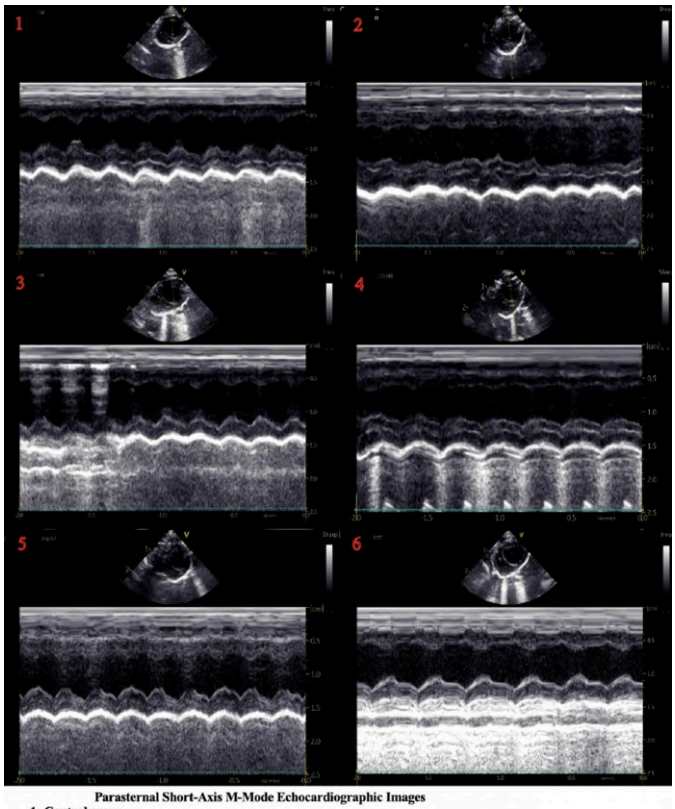
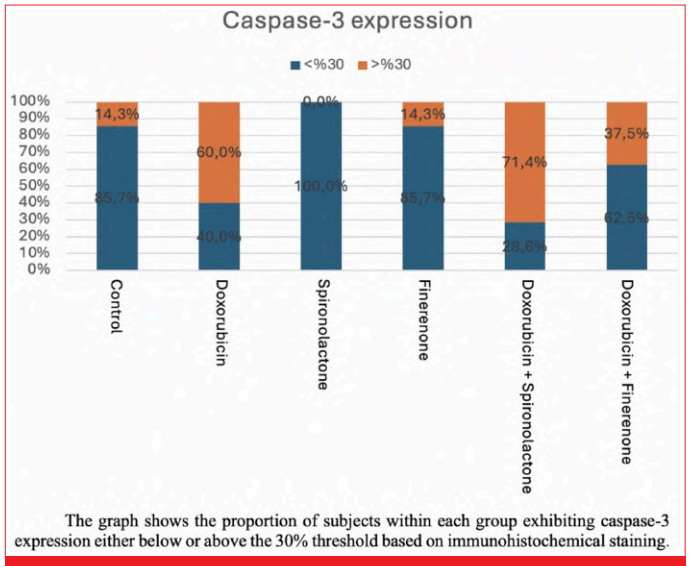


Figure 6. Results 3. Graph of left ventricular ejection fraction measurements by echocardiography.



Parasternal Short-Axis M-Mode Echocardiographic Images
1: Control group
2: Doxorubicin group
3: Spironolactone group
4: Doxorubicin + Spironolactone group
5: Finerenone group
6: Doxorubicin + Finerenone group

Figure 7. Results 4. Representative echocardiographic ımages of the groups.



The graph shows the proportion of subjects within each group exhibiting caspase-3 expression either below or above the 30% threshold based on immunohistochemical staining.

Figure 8. Results 6. Graph of immunohistochemical Caspase-3 expression.

Table 2. Results 5. Table(s) of light microscopy findings

	Congestion						
	Mild		Moderate		Severe		
GROUP	n	%	n	%	n	%	p-value
Control	-	-	7	(100)	-	-	0,132
Dox	-	-	1	(20,00)	4	(80,00)	
Sp	-	-	6	(75,00)	2	(25,00)	
Fin	1	(14,29)	4	(57,14)	2	(28,57)	
Dox+Sp	1	(14,29)	4	(57,14)	2	(28,57)	
Dox+Fin	-	-	5	(62,50)	3	(37,50)	

	Vascular proliferation						
	Mild		Moderate		Severe		
GROUP	n	%	n	%	n	%	p-value
Control	3	(42,86)	4	(57,14)	-	-	0,612
Dox	-	-	3	(60,00)	2	(40,00)	
Sp	2	(25,00)	4	(50,00)	2	(25,00)	
Fin	2	(28,57)	3	(42,86)	2	(28,57)	
Dox+Sp	2	(28,57)	5	(71,43)	-	-	
Dox+Fin	1	(12,50)	4	(50,00)	3	(37,50)	

	Hydropic degeneration						
	Mild		Moderate		Severe		
GROUP	n	%	n	%	n	%	p-value
Control	4	(57,14)	3	(42,86)	-	-	0,034
Dox	-	-	4	(80,00)	1	(20,00)	
Sp	2	(25,00)	6	(75,00)	-	-	
Fin	1	(14,29)	3	(42,86)	3	(42,86)	
Dox+Sp	3	(42,86)	3	(57,14)	-	-	
Dox+Fin	-	-	4	(50,00)	4	(50,00)	

	Hypertrophy						
	Mild		Moderate		Severe		
GROUP	n	%	n	%	n	%	p-value
Control	7	(100,00)	-	-	-	-	0,010
Dox	-	-	2	(40,00)	3	(60,00)	
Sp	3	(37,50)	5	(62,50)	-	-	
Fin	2	(28,57)	4	(57,14)	1	(14,29)	
Dox+Sp	4	(57,14)	3	(42,86)	-	-	
Dox+Fin	3	(37,50)	4	(50,00)	1	(12,50)	

	Capillaritis						
	Mild		Moderate		Severe		
GROUP	n	%	n	%	n	%	p-value
Control	7	(100,00)	-	-	-	-	0,085
Dox	2	(40,00)	3	(60,00)	-	-	
Sp	5	(62,50)	3	(37,50)	-	-	
Fin	2	(28,57)	4	(57,14)	1	(14,29)	
Dox+Sp	2	(28,57)	4	(57,14)	1	(14,29)	
Dox+Fin	2	(25,00)	5	(62,50)	1	(12,50)	

	Capillary dilatation						
	Mild		Moderate		Severe		
GROUP	n	%	n	%	n	%	p-value
Control	4	(57,14)	3	(42,86)	-	-	0,289
Dox	3	(60,00)	1	(20,00)	1	(20,00)	
Sp	1	(12,50)	5	(62,50)	2	(25,00)	
Fin	2	(28,57)	1	(14,29)	4	(57,14)	
Dox+Sp	3	(42,86)	3	(42,86)	1	(14,29)	
Dox+Fin	2	(25,00)	2	(25,00)	4	(50,00)	

The Fisher-Freeman-Halton Exact Test was used for data analysis.

n: number of subjects, %: percentage of subjects within the group

Mild: less than 10% change, Moderate: 10–30% change, Severe: more than 30% change

Control: control group, Dox: doxorubicin group, Sp: spironolactone group, Fin: finerenone group, Dox+Sp: doxorubicin + spironolactone group, Dox+Fin: doxorubicin + finerenone group

OP-038 [Heart Failure]

Impact of low-volume high-intensity interval training on autonomic regulation, exercise capacity, and quality of life in HFpEF

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Background and Aim: Heart failure with preserved ejection fraction (HFpEF) is prevalent among older adults, characterized by impaired exercise tolerance, reduced quality of life, and high morbidity and mortality. While exercise training can improve cardiorespiratory fitness, autonomic regulation, and patient-reported outcomes, the role of low-volume, high-intensity interval training (LV-HIIT) in this population remains poorly defined. Given its potential time efficiency and tolerability, this study aimed to assess the effects of an 8-week LV-HIIT program on heart rate variability (HRV), functional capacity, biochemical markers, and quality of life in HFpEF patients.

Methods: In this prospective, controlled, parallel-group trial, 20 HFpEF patients with comparable baseline characteristics were randomly allocated (computer-generated sequence; sealed opaque envelopes) to LV-HIIT (n=11) or control (n=9) groups. The study was single-blind with blinded outcome assessors. LV-HIIT was performed on a calibrated Monark 881E arm ergometer(Monark 881E, Monark Exercise AB, Vansbro, Sweden) twice weekly for 8 weeks (16 sessions), each comprising eight 1-min bouts at 90% and 40% of peak workload (determined by incremental test; 1:1 work/rest). Controls received guideline-based medical therapy only. Primary outcomes were changes in SDNN and LF/HF ratio, obtained from 5-min supine recordings using the Polar H10 heart rate sensor (Polar Electro, Kempele, Finland) and analyzed with Kubios HRV software (morning sessions; caffeine and strenuous exercise restricted). Secondary outcomes were changes in 6-minute walk test (ATS protocol), NT-proBNP, and Nottingham Health Profile scores. Statistical analyses included the Shapiro–Wilk test, Mann–Whitney U test, unpaired t-test, and Chi-square test, as appropriate. $p<0.05$ was considered significant. This research was conducted by Tarsus University Department of Scientific Research Projects with project number SBF.24.001.

Results: After 8 weeks, LV-HIIT significantly increased SDNN (80→105 ms, p=0.005) and RMSSD (62→80 ms, p=0.004), with no changes in controls. LF/HF ratio decreased from 2 to 1 in LV-HIIT participants (p=0.002) but rose slightly in controls. Functional capacity improved in the LV-HIIT group (361→448 m) and declined in controls (371→336 m; p=0.027), exceeding the minimal clinically important difference. No significant between-group differences were found for NT-proBNP (p=0.879). NHP scores improved substantially in LV-HIIT participants (183→80; p=0.003) but worsened in controls.

Conclusions: An 8-week LV-HIIT program produced significant improvements in autonomic function, exercise capacity, and quality of life in patients with HFpEF. These findings support LV-HIIT as a time-efficient, feasible, and clinically effective training modality, potentially mediated by enhanced autonomic regulation and functional reserve. Larger randomized trials with blinded assessments are warranted to validate these results and assess long-term clinical outcomes.

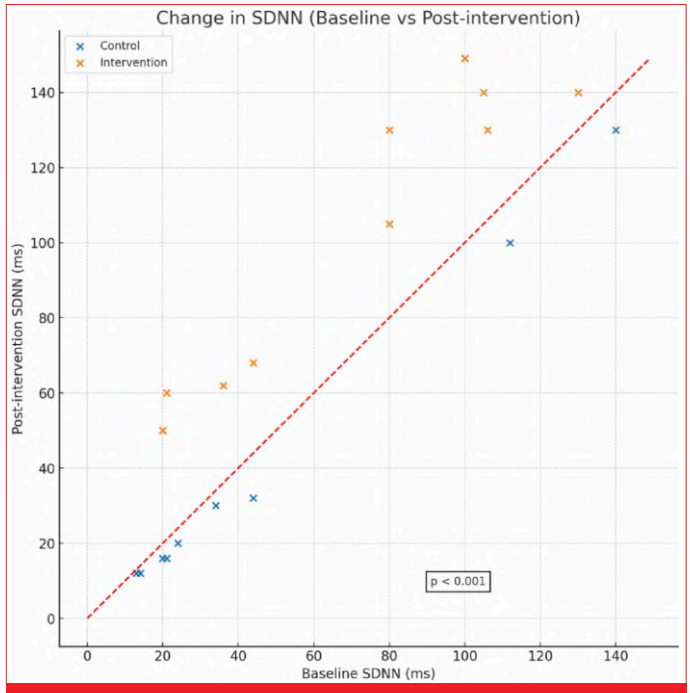

Figure 1. Change in SDNN (baseline vs. post-intervention). SDDNN: Standard deviation of normal-to-normal RR intervals.

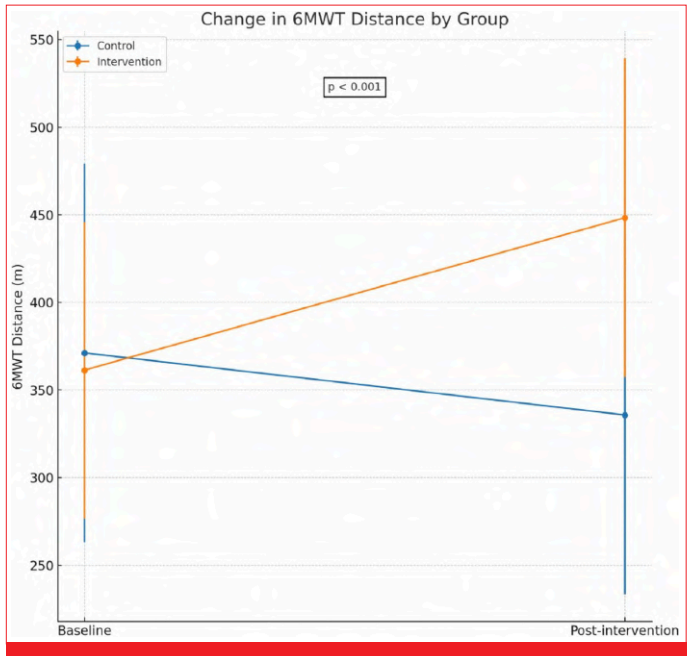

Figure 2. Change in 6MWT distance by group. 6MWT: 6-minute walk test.

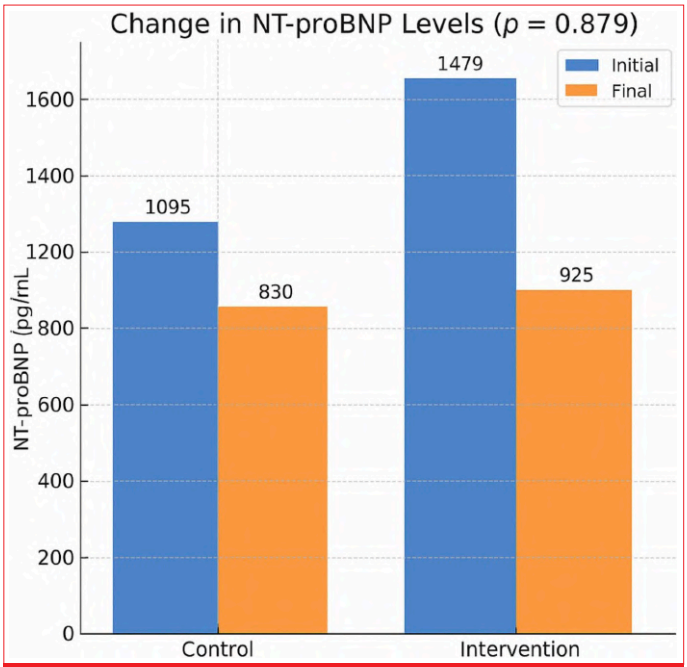

Figure 3. Change in NT-proBNP levels. NT-proBNP:N-terminal pro-B-type natriuretic peptide.

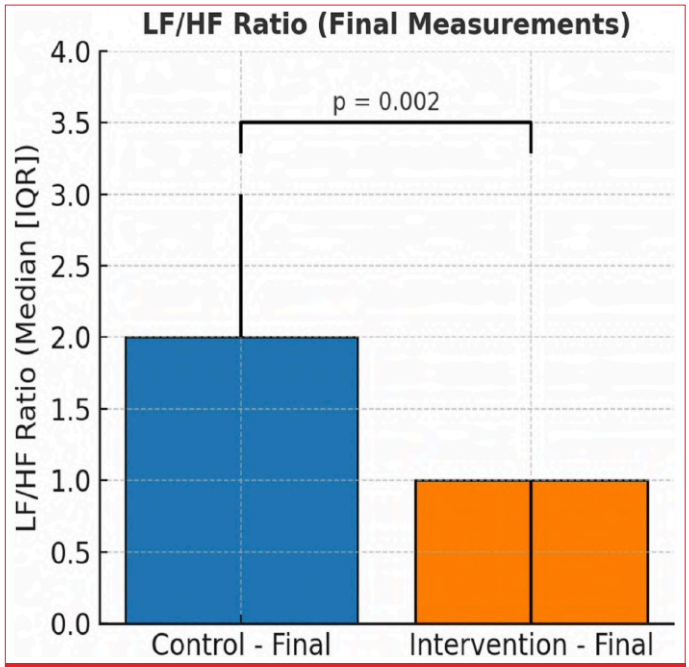

Figure 5. LF/HF ratio (final measurements). LF/HF ratio: Low-frequency to high-frequency power ratio.

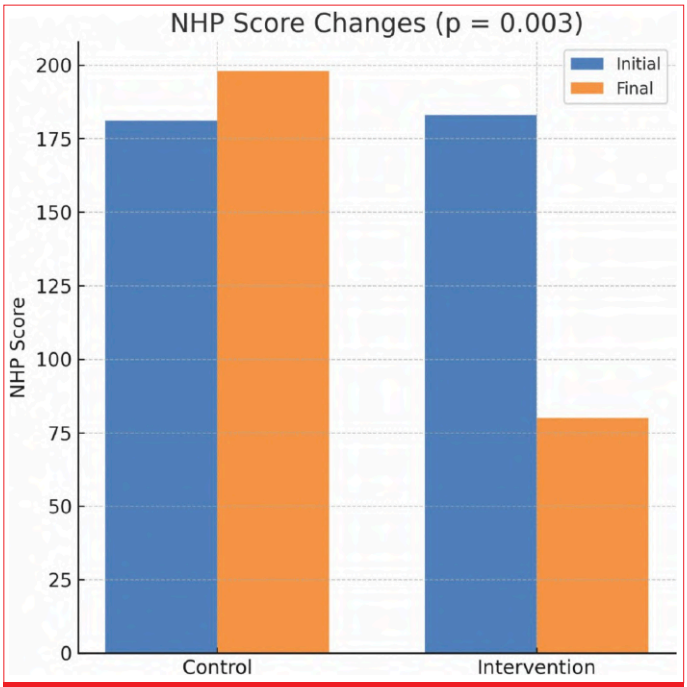

Figure 4. NHP scores changes. NHP:Nottingham Health Profile.

Table 1. Baseline demographic and clinical characteristics

Variable	Control Group (N=9)	Intervention Group (N=11)	p value
Age	70.9 ± 9	67.1 ± 10.2	0.397
Gender (female) n (%)	3 (33.3%)	5 (45.5%)	0.927
BMI	26 (25-31)	26 (25-31)	0.987
EF (%)	55 (50-55)	50 (50-55)	0.536
DM	7 (77.8%)	6 (54.5%)	0.374
HT	9 (100%)	9 (81.8%)	0.479
Charlson Comorbidity Index	3.11 ± 1.69	3.18 ± 1.25	0.582
NT-proBNP (initial)	1095 (617-2388)	1479 (938-2494)	0.884
6MWT (initial test)	371.18 ± 114.61	361.36 ± 88.77	0.836
NYHA Class (baseline)	2.20 (1.93–2.47)	2.10 (1.70–2.50)	0.603
SDNN (initial)	24 (20–44)	80 (28–102)	0.286
RMSSD (initial)	16 (10–30)	62 (21–80)	0.148
LF/HF (initial)	1 (0–3)	2 (1–2)	0.543
NHP Initial Score	181 (179–332)	183 (130–310)	0.790
Diuretic use n (%)	8 (88.9%)	10 (90.9%)	1.000
RAAS blocker use n (%)	9 (100.0%)	9 (81.8%)	0.479
SGLT2i use n (%)	7 (77.8%)	6 (54.5%)	0.374
Beta-blocker use n (%)	8 (88.9%)	9 (81.8%)	1.000

Values are presented as median [interquartile range] or number (percentage). Between-group comparisons were analyzed using the Mann–Whitney U test or independent t-test for continuous variables, and the Chi-square test for categorical variables. A p-value <0.05 was considered statistically significant. BMI: Body Mass Index; EF: Ejection Fraction; DM: Diabetes Mellitus; HT: Hypertension; NT-proBNP: Pro B-type natriuretic peptid; 6MWT: 6-Minute Walk Test; NYHA: New York Heart Association; SDNN: Standard deviation of NN intervals; RMSSD: Root mean square of successive differences; LF/HF: Low frequency/high frequency ratio; NHP: Nottingham Health Profile; RAAS: Renin–angiotensin–aldosterone system; SGLT2i: Sodium–glucose cotransporter-2 inhibitor; MRA: Mineralocorticoid receptor antagonist.

Table 2. Comparison of initial and final measurements

Variable	Control Group	Intervention Group	p value
SDNN (initial)	24 (20–44)	80 (28–102)	0.286
SDNN (final)	20 (16–32)	105 (61–135)	0.005
RMSSD (initial)	16 (10–30)	62 (21–80)	0.148
RMSSD (final)	15 (11–24)	80 (46–98)	0.004
LF/HF (initial)	1 (0–3)	2 (1–2)	0.543
LF/HF (final)	2 (2–3)	1 (0–1)	0.002
NT pro-BNP (initial)	1095 (617–2388)	1479 (938–2494)	0.884
NT pro-BNP (final)	830 (330–1457)	925 (325–1455)	0.879
6MWT (initial test)	371.18 ± 114.61	361.36 ± 88.77	0.836
6MWT (final test)	335.78 ± 108.74	448.36 ± 95.53	0.027
NYHA Class (baseline)	2.20 (1.93–2.47)	2.10 (1.70–2.50)	0.603
NYHA Class (final)	2.20 (1.93–2.47)	1.82 (1.55–2.09)	0.129
NHP Initial Score	181 (179–332)	183 (130–310)	0.790
NHP Final Score	198 (181–360)	80 (46–120)	0.003

Values are presented as median [interquartile range] or number (percentage) unless otherwise indicated. Between-group comparisons were analyzed using the Mann–Whitney U test or independent t-test for continuous variables, and the Chi-square test for categorical variables. A p-value <0.05 was considered statistically significant. SDNN: Standard deviation of NN intervals; RMSSD: Root mean square of successive differences; LF/HF: Low frequency/high frequency ratio; BNP: B-type natriuretic peptide; 6MWT: 6-Minute Walk Test; NYHA: New York Heart Association; NHP: Nottingham Health Profile.

OP-039 [Heart Failure]

SGLT2 inhibitors and cardiovascular event reduction in HFpEF: A systematic review & meta-analysis

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Background and Aim: Heart failure with preserved ejection fraction (HFpEF) is a growing global health burden, characterized by high morbidity, mortality, and frequent hospitalizations. Despite its prevalence, effective therapies remain limited. Sodium–glucose cotransporter 2 (SGLT2) inhibitors, originally designed for glycaemic control in type 2 diabetes, have demonstrated promising cardiovascular and renal benefits in HFpEF populations. This meta-analysis aimed to provide a comprehensive evaluation of their efficacy and safety in reducing adverse cardiovascular outcomes and improving patient-centred metrics.

Methods: A systematic literature search was conducted in PubMed, Cochrane Library, EMBASE, Scopus, and ClinicalTrials.gov for randomized controlled trials (RCTs) published from 2000 to 2024. Eligible studies included adult patients with HFpEF (left ventricular ejection fraction ≥50%) receiving SGLT2 inhibitors (empagliflozin, dapagliflozin, canagliflozin, or ertugliflozin) compared to placebo or standard care. Primary outcomes were major adverse cardiovascular events (MACE) and heart failure-related hospitalizations. Secondary outcomes included all-cause and cardiovascular mortality, quality of life (measured via the Kansas City Cardiomyopathy Questionnaire [KCCQ]), and renal function. A random-effects model was used to pool data, with heterogeneity assessed using the I^2 statistic.

Results: Fifteen RCTs encompassing 94,000 HFpEF patients were analysed. SGLT2 inhibitors significantly reduced the risk of MACE by 21% (risk ratio [RR]: 0.79; 95% confidence interval [CI]: 0.69–0.90; p<0.001) and heart failure hospitalizations by 26% (hazard ratio [HR]: 0.74; 95% CI: 0.65–0.85; p<0.001). Quality of life improved by a mean of 5.5 points on the KCCQ (p<0.05), exceeding the minimal clinically important difference. Although all-cause mortality showed a favourable trend (RR: 0.86; 95% CI: 0.74–0.99), statistical significance was not uniformly observed. Renal function was preserved, with a slower decline in eGFR compared to controls (mean difference: 1.25 mL/min/1.73 m^2/year; p<0.01).

Conclusions: SGLT2 inhibitors represent a positive shift in HFpEF management, with demonstrated reductions in cardiovascular events and hospitalizations, alongside improvements in quality of life and renal preservation. While mortality benefits remain inconclusive, this meta-analysis supports integrating SGLT2 inhibitors into standard HFpEF treatment protocols, particularly for patients with comorbid diabetes or chronic kidney disease. Further long-term trials are needed to confirm mortality benefits.

OP-040 [Heart Failure]

Comparative analysis of implantable cardioverter defibrillator efficacy in ischemic and non-ischemic cardiomyopathy in patients with heart failure

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Background and Aim: The benefit of implantable cardioverter defibrillators (ICDs) in reducing sudden cardiac death (SCD) among patients with heart failure with reduced ejection fraction (HFrEF) is well established in ischemic cardiomyopathy (ICM). However, in non-ischemic cardiomyopathy (NICM), the survival advantage

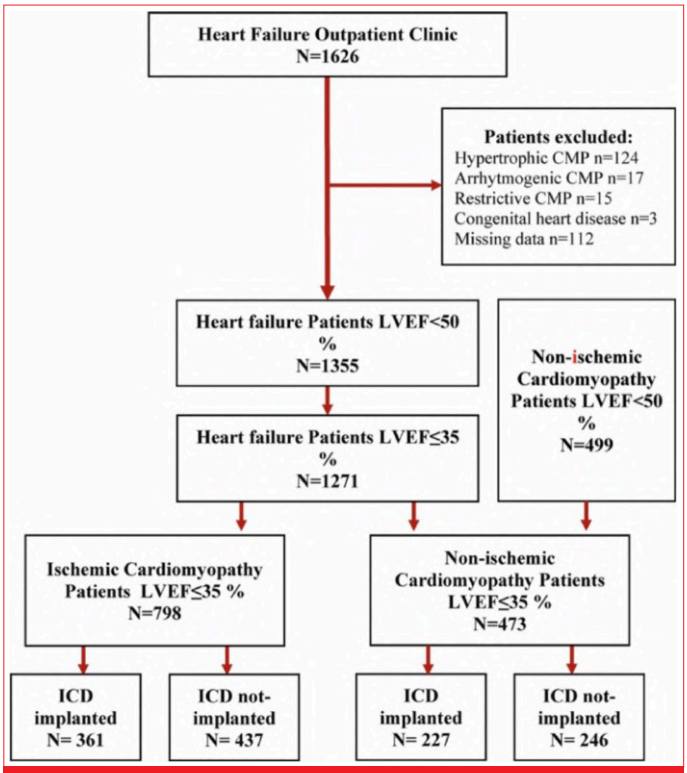


Figure 1. Flow chart of the study design and patient selection process. This flow chart illustrates the stepwise inclusion and exclusion of patients from the Heart Failure Outpatient Clinic cohort. A total of 1626 patients were initially screened. Of these, patients with hypertrophic cardiomyopathy (n=124), arrhythmogenic cardiomyopathy (n=17), restrictive cardiomyopathy (n=15), congenital heart disease (n=3), and those with missing data (n=112) were excluded. The remaining 1355 patients had heart failure with left ventricular ejection fraction (LVEF) < 50%. From this group, 1271 patients had LVEF ≤35% and were further evaluated. Based on etiology, patients were categorized into ischemic cardiomyopathy (ICM, n=798) and non-ischemic cardiomyopathy (NICM, n= 473). Each subgroup was stratified according to implantable cardioverter defibrillator (ICD) implantation status. Among ICM patients, 437 did not receive an ICD, while 361 underwent ICD implantation. Among NICM patients, 246 did not receive an ICD and 227 were implanted with an ICD. This classification enabled comparative evaluation of outcomes based on both etiology and ICD status in a realworld heart failure population.

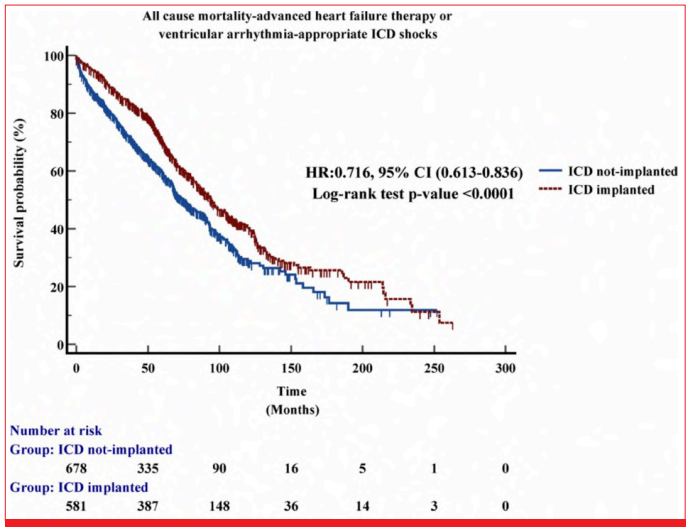


Figure 2. Survival analysis of ICD implanted and not-implanted patients, left ventricle ejection fraction ≤35%.

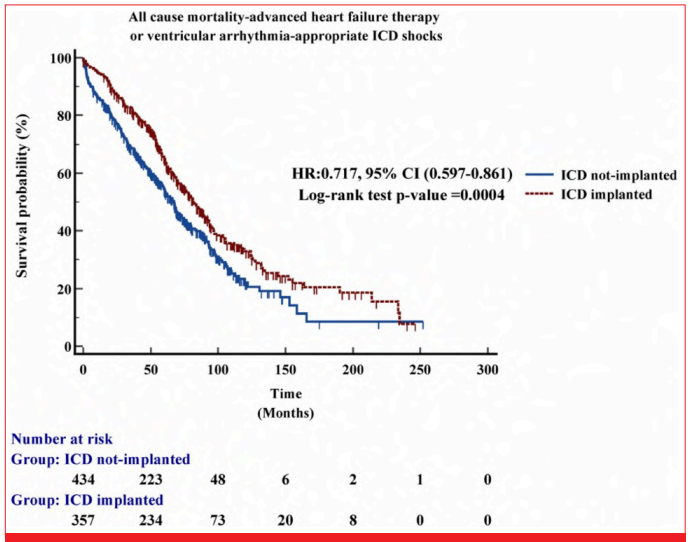


Figure 3. Survival analysis of ICD implanted and not-implanted ischemic heart failure patients, left ventricle ejection fraction ≤35%.

remains uncertain in the context of contemporary medical therapy. This retrospective, observational cohort study aimed to evaluate the real-world efficacy of ICDs in patients with LVEF ≤35%, stratified by underlying etiology.

Methods: This retrospective cohort study included 1,271 patients with LVEF ≤35%, of whom 588 (46.3%) received ICDs. The primary endpoint was a composite of all-cause mortality, advanced heart failure therapy (LVAD or transplantation), or ventricular arrhythmias requiring appropriate ICD shock. Patients were stratified by etiology (ICM vs. NICM), ICD status, and presence of LVEF recovery (>35%).

Results: ICD implantation was associated with a significant reduction in the primary endpoint in ICM patients (HR 0.717; 95% CI 0.595–0.861; p=0.0004). In contrast, no statistically significant benefit was observed in NICM patients overall (HR 0.767; 95% CI 0.573–1.026; p=0.074). Notably, in a subgroup of 103 patients whose LVEF improved beyond 35% during follow-up and were excluded from the primary analysis, ICD therapy conferred a survival benefit in NICM patients (HR 0.645; 95% CI 0.478–0.870; p=0.0041). Patients experienced LVEF recovery above 35% during follow-up, 28 NICM patients with ICDs and recovered LVEF, none experienced death or required advanced therapy, though 4 developed significant ventricular arrhythmias. These findings underscore residual arrhythmic risk even with apparent recovery. Among NICM patients, independent predictors of adverse outcomes included NYHA class III–IV (HR 1.934), moderate-to-severe mitral regurgitation (HR 1.956), reduced tricuspid annular plane systolic excursion (TAPSE) (HR 0.945), and elevated NT-proBNP levels (log-transformed) (HR 1.531). A multivariate risk score derived

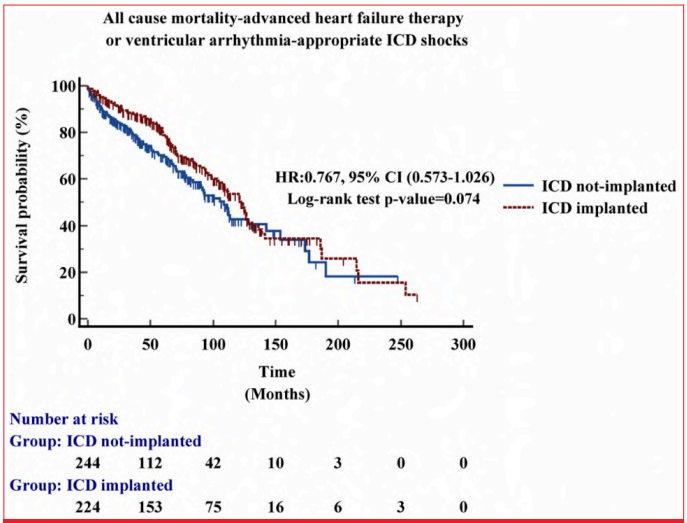


Figure 4. Survival analysis of ICD implanted and not-implanted non-ischemic cardiomyopathy patients, left ventricle ejection fraction ≤35%.

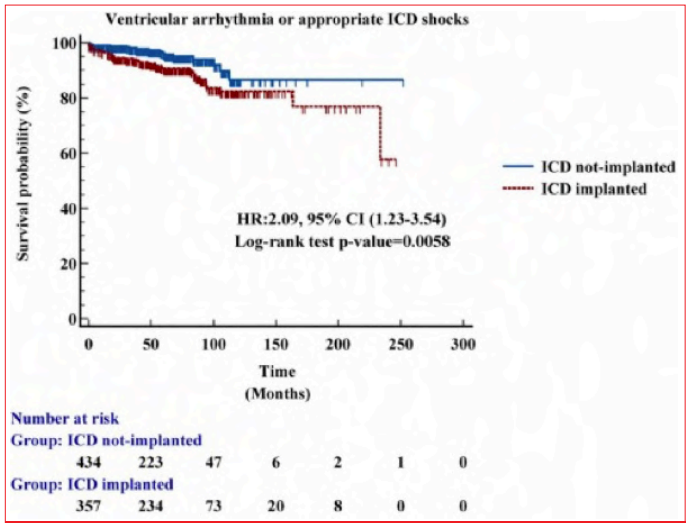


Figure 6. Arrhtyhmia or appropriate ICD shocks free survial of ICD implanted and not-implanted ischemic cardiomyopathy patients, left ventricle ejection fraction ≤35%.

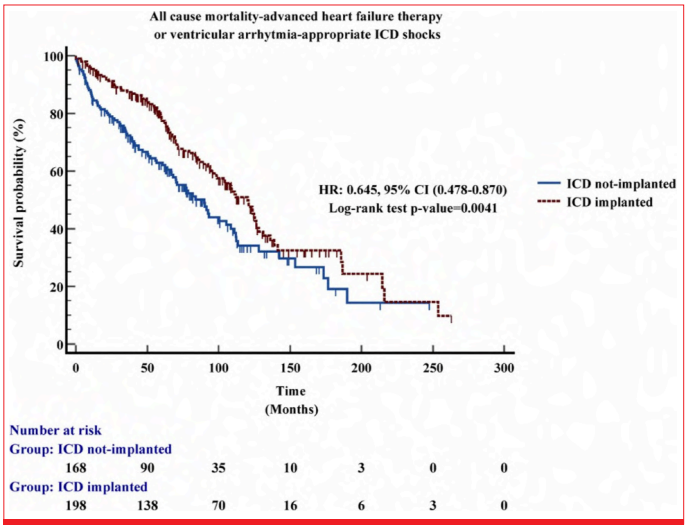


Figure 5. Survival analysis of ICD implanted and not-implanted non-ischemic cardiomyopathy patients, left ventricle ejection fraction ≤35%, heart failure improved patients excluded.

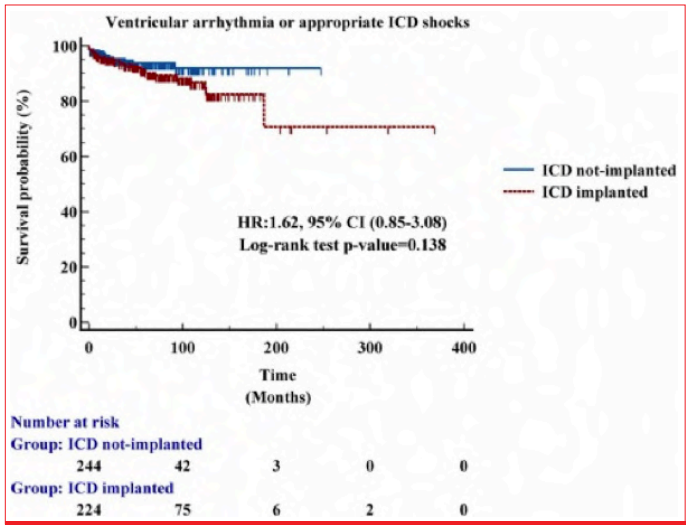
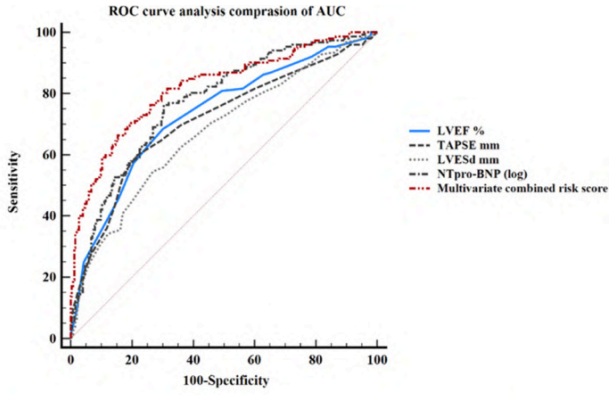


Figure 7. Arrhtyhmia or appropriate ICD shocks free survial of ICD implanted and not-implanted non-ischemic cardiomyopathy patients, left ventricle ejection fraction ≤35%.

from these variables demonstrated superior predictive accuracy compared to LVEF alone (AUC: 0.819 vs. 0.731), suggesting that reliance on LVEF as a solitary criterion may be insufficient in this population.

Conclusions: These findings reinforce the established role of ICDs in ICM while highlighting the complexity of decision-making in NICM. In the modern era of heart failure management, where reverse remodeling and LVEF recovery are increasingly common with optimized medical therapy, risk stratification in NICM requires a more individualized approach. Integration of functional, biochemical, and echocardiographic parameters into decision algorithms may better identify NICM patients at true risk of arrhythmic events and improve therapeutic allocation. As such, this study supports a paradigm shift toward precision-based ICD utilization to enhance patient outcomes and minimize unnecessary interventions in HFrEF care.



Variable	AUC	95% CI
LVEF %	0.732	0.686 to 0.774
TAPSE mm	0.712	0.665 to 0.755
LVESd mm	0.675	0.627 to 0.720
NTpro-BNP (log) pg/ml	0.770	0.726 to 0.810
Multivariate combined risk score	0.819	0.778 to 0.856

Figure 8. ROC-curve analysis comprasion positive predictive probability in patients with non-ischemic cardiomyopathy, LVEF ≤50%.

Table 1. Clinical characteristics of the patients whose left ventricle ejection fraction ≤35% at baseline and comparison according to ICD implantation

	Total 1271	None 683 (53.7%)	ICD 588 (46.3%)	*p* value
Age years	46.9 ± 12.0	45.8 ± 12.7	48.1 ± 11.0	0.001
Male n (%)	968 (76.2)	500 (73.2)	468 (79.6)	0.008
BMI (kg/m²)	26.5 ± 3.9	26.3 ± 4.0	26.7 ± 3.7	0.228
BSA (m²)	1.91 ± 0.18	1.91 ± 0.18	1.92 ± 0.18	0.309
SBP (mmHg)	114 ± 18	116 ± 18	111 ± 17	<0.0001
DBP (mmHg)	69.5 ± 11.3	70 ± 11	68 ± 10	0.009
NYHA FC (%)	–	–	–	<0.0001
I	136 (12.4)	91 (16.9)	45 (8.0)	–
II	539 (49.0)	270 (50.3)	269 (47.8)	–
III	354 (32.2)	150 (27.9)	204 (36.2)	–
IV	71 (6.5)	26 (4.8)	45 (8.0)	–
III-IV	425 (38.6)	176 (32.8)	249 (44.2)	<0.0001
Ischemic etiology n (%)	798 (62.8)	437 (64.0)	361 (61.4)	0.34
Arterial hypertension n (%)	444 (35.0)	220 (32.2)	224 (38.4)	0.022
Diabetes mellitus n (%)	371 (29.2)	184 (26.9)	187 (31.9)	0.055
Dyslipidemia n (%)	227 (17.9)	100 (14.7)	127 (21.7)	0.001
Smoking history n (%)	514 (40.6)	266 (39.2)	248 (42.2)	0.276
Cerebrovascular event n (%)	61 (4.8)	37 (5.4)	24 (4.1)	0.265
COPD n (%)	74 (5.9)	43 (6.4)	31 (5.3)	0.423
Renal disease n (%)	37 (2.9)	23 (3.4)	14 (2.4)	0.297
Chronic liver disease n (%)	5 (0.5)	1 (0.1)	4 (0.7)	0.301
Peripheral arterial disease n (%)	24 (2.5)	11 (2.7)	13 (2.4)	0.900
AF n (%)	191 (16.4)	114 (17.4)	77 (15.1)	0.309
CRT (%)	124 (9.8)	–	124 (21.1)	–
Family history of heart failure n (%)	163 (12.9)	76 (11.1)	87 (14.9)	0.046
NT-proBNP (pg/ml)	1445 (592-4080)	1488 (568-4471)	1421 (611-3582)	0.378
Follow-up time median IQR (months)	56.9 (28.7-88.3)	49.1 (21.7-78.1)	64.5 (38.9-100.7)	<0.0001
Primary end-point n (%)	655 (51.5)	51.7 (353)	51.4 (302)	0.908

AF atrial fibrillation, Body mass index, BSA body surface area, COPD chronic obstructive pulmonary disease, CRT cardiac resynchronization therapy, ICD implanted cardioverter-defibrillator, DBP diastolic blood pressure, IQR interquartile range, LVEF left ventricular ejection fraction, NYHA FC New York Heart Association Functional Class, SBP systolic blood pressure. *Values are mean ± SD, median (IQR) or n (%) p value <0.05.

Table 2. Medical therapy of patients whose left ventricle ejection fraction ≤35% at baseline and comparison according to ICD implantation

	Total 1271	None 683 (53.7%)	ICD 588 (46.3%)	*p* value
ACEi or ARB n (%)	1008 (79.3)	574 (84.2)	433 (73.9)	<0.0001
ACEi or ARB dose	–	–	–	<0.0001
Exact dose n (%)	210 (17.5)	108 (17.2)	102 (17.7)	–
Half dose n (%)	448 (37.3)	259 (41.3)	189 (32.9)	–
Quarter dose n (%)	360 (30.0)	197 (31.4)	163 (28.3)	–
ARNI n (%)	116 (9.1)	61 (8.9)	55 (9.4)	0.794
ARNI dose	–	–	–	0.808
Exact dose n (%)	29 (2.3)	14 (2.0)	15 (2.6)	–
Half dose n (%)	38 (3.0)	19 (2.8)	19 (3.2)	–
Quarter dose n (%)	48 (3.8)	28 (4.1)	20 (3.4)	–
Beta-blocker n (%)	1248 (98.2)	676 (99.0)	572 (97.3)	0.024
Beta-blocker dose	–	–	–	<0.0001
Exact dose n (%)	110 (9.0)	53 (8.3)	57 (9.8)	–
Half dose n (%)	651 (53.4)	385 (60.5)	266 (45.6)	–
Quarter dose n (%)	435 (35.7)	191 (30.0)	244 (41.9)	–
Aldosterone antagonist n (%)	1175 (92.4)	635 (93.0)	540 (91.8)	0.445
Aldosterone antagonist dose	–	–	–	<0.0001
Exact dose n (%)	262 (20.7)	210 (31.1)	52 (8.8)	–
Half dose n (%)	896 (70.9)	410 (60.7)	486 (82.7)	–
Quarter dose n (%)	11 (0.9)	9 (1.3)	2 (0.3)	–
SGLT2-i n (%)	203 (16.0)	100 (14.6)	103 (17.5)	0.163
Loop diuretic n (%)	1069 (84.1)	537 (78.6)	49.8 (90.5)	<0.0001
Anticoagulant n (%)	374 (29.4)	192 (28.1)	182 (31.0)	0.268
Antiplatelet n (%)	746 (58.7)	383 (56.1)	363 (61.7)	0.041
If channel blocker n (%)	430 (33.8)	216 (31.6)	214 (36.4)	0.073
Digoxin n (%)	226 (17.8)	109 (16.0)	117 (19.9)	0.067
Antihyperlipidemic n (%)	492 (38.7)	210 (30.7)	282 (48.0)	<0.0001
Antiarrhythmic n (%)	95 (10.0)	16 (3.9)	79 (14.5)	<0.0001

ACEi angiotensinogen converting enzyme inhibitor, ARB angiotensin receptor blocker, ARNI angiotensin receptor-neprilysin inhibitor, SGLT2-i sodium-glucose transport protein two inhibitors. *Values are mean ± SD, median (IQR) or n (%) p value <0.05.

Table 3. Echocardiography parameters of patients whose left ventricle ejection fraction ≤35% at baseline and comparison according to ICD implantation

	Total 1271	None 683 (53.7%)	ICD 588 (46.3%)	*p* value
LVEDd (mm)	65.1 ± 8.8	64.4 ± 9.1	66.0 ± 8.5	0.003
LVESd (mm)	55.6 ± 10.0	54.8 ± 10.3	56.5 ± 9.7	0.004
LAd (mm)	47.2 ± 7.0	47.1 ± 7.5	47.3 ± 6.3	0.554
LVEF (%)	24.6 ± 5.2	24.9 ± 5.5	24.3 ± 4.9	0.055
Mitral regurgitation n (%)	-	-	-	<0.0001
None n (%)	82 (6.5)	55 (8.1)	27 (4.6)	-
Mild n (%)	319 (25.2)	143 (21.0)	176 (30.1)	-
Moderate n (%)	578 (45.7)	310 (45.5)	268 (45.8)	-
Severe n (%)	287 (22.7)	173 (25.4)	114 (19.5)	-
Moderate or severe n (%)	865 (68.3)	483 (70.9)	382 (65.3)	0.032
Aortic regurgitation n (%)	-	-	-	0.978
None n (%)	977 (77.4)	524 (77.2)	453 (77.7)	-
Mild n (%)	214 (17.0)	115 (16.9)	99 (17.0)	-
Moderate n (%)	62 (4.9)	35 (5.2)	27 (4.6)	-
Severe n (%)	9 (0.7)	5 (0.4)	4 (0.3)	-
Moderate or severe n (%)	71 (5.6)	40 (5.9)	31 (5.3)	0.65
Tricuspid regurgitation n (%)	-	-	-	0.082
None n (%)	186 (14.7)	105 (15.4)	81 (13.9)	-
Mild n (%)	458 (36.2)	226 (33.2)	232 (39.7)	-
Moderate n (%)	431 (34.1)	237 (34.9)	194 (33.2)	-
Severe n (%)	189 (15.0)	112 (16.5)	77 (13.2)	-
Moderate or severe n (%)	620 (49.1)	349 (51.3)	271 (46.4)	0.081
Pulmonary regurgitation n (%)	-	-	-	0.010
None n (%)	1078 (85.6)	560 (82.7)	518 (88.9)	-
Mild n (%)	81 (6.4)	48 (7.1)	33 (5.7)	-
Moderate n (%)	82 (6.5)	57 (8.4)	25 (4.3)	-
Severe n (%)	19 (1.5)	12 (1.8)	7 (1.2)	-
Moderate or severe n (%)	101 (8.0)	69 (10.2)	32 (5.5)	0.002
TAPSE mm	16.8 ± 4.8	16.5 ± 4.9	17.1 ± 4.7	0.046
RVsm (TDI) (m/s)	10.0 ± 2.6	9.8 ± 2.5	10.2 ± 2.75	0.026
TRV (m/s)	2.87 ± 0.53	2.86 ± 0.52	2.89 ± 0.55	0.383
SPAP (mmHg)	37.0 ± 15.5	36.7 ± 15.2	37.3 ± 15.8	0.502

Values are mean ± SD, median (IQR) or n (%) p value <0.05. LVEDd left ventricular end-diastolic diameter, LVESd left ventricular end-systolic diameter, LAd left atrial diameter, LVEF left ventricular ejection fraction, TAPSE tricuspid annular plane systolic excursion, RVsm right ventricular systolic motion tissue Doppler imaging, TRV tricuspid regurgitation velocity, SPAP systolic pulmonary artery pressure.

Table 4. Primary and secondary endpoints in ICD-implanted and not-implanted patients

	Total 1271	None 683 (53.7%)	ICD 588 (46.3%)	*p* value
Primary end-point n (%)	655 (51.5)	51.7 (353)	51.4 (302)	0.908
All-cause mortality n (%)	314 (24.7)	162 (23.7)	152 (25.9)	0.380
Advanced heart failure therapy n (%)	280 (22.1)	170 (24.9)	110 (18.7)	0.008
Heart failure-related hospitalization n (%)	461 (36.3)	174 (25.5)	287 (48.8)	<0.0001
Ventricular arrhythmias or appropriate ICD shocks n (%)	95 (7.5)	30 (4.4)	65 (11.1)	<0.0001

Values are mean ± SD, median (IQR) or n (%) p value <0.05. ICD implanted cardioverter defibrillator.

Table 5. Non-ischemic cardiomyopathy patients, LVEF ≤35% Cox regression univariate and multivariate analysis

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	*p* value	HR (95% CI)	*p* value
Age years	1.008 (0.995-1.021)	0.220	-	-
Gender male	1.159 (0.853-1.570)	0.341	-	-
BMI (kg/m^2)	0.987 (0.932-1.044)	0.642	-	-
BSA (m^2)	0.581 (0.169-2.006)	0.391	-	-
SBP (mmHg)	0.983 (0.973-0.992)	<0.0001	-	-
DBP (mmHg)	0.990 (0.976-1.005)	0.178	-	-
NYHA FC III-IV	3.238 (2.382-4.402)	<0.0001	1.934 (1.302-2.871)	0.001
Arterial hypertension	0.821 (0.577-1.168)	0.272	-	-
Diabetes mellitus	0.801 (0.547-1.172)	0.253	-	-
Dyslipidemia	0.885 (0.556-1.409)	0.607	-	-
Smoking history	0.941 (0.675-2.153)	0.813	-	-
COPD	0.832 (0.390-1.776)	0.635	-	-
Ischemic cerebrovascular event	1.379 (0.727-2.615)	0.326	-	-
Renal disease	0.622 (0.154-2.509)	0.505	-	-
Chronic liver disease	5.275 (1.290-21.563)	0.021	-	-
Atrial fibrilation	1.3352 (0.899-2.032)	0.147	-	-
ICD	0.769 (0.576-1.027)	0.075	-	-
CRT	0.637 (0.410-0.991)	0.045	0.545 (0.327-0.908)	0.020
Family history	0.848 (0.601-1.195)	0.345	-	-
ACEi or ARB	0.962 (0.684-1.352)	0.823	-	-
ARNI	0.663 (0.384-1.143)	0.663	-	-
Beta-blocker	0.363 (0.192-0.687)	0.002	-	-
Aldosterone antagonist	0.705 (0.372-1.336)	0.284	-	-
SGLT2-i	0.698 (0.458-1.064)	0.094	-	-
Loop diuretic	1.980 (1.231-3.186)	0.005	-	-
Anticoagulant	0.999 (0.728-1.369)	0.994	-	-
Antiplatelet	1.069 (0.799-1.430)	0.655	-	-
If channel blocker	0.890 (0.659-1.202)	0.448	-	-
Digoxin	1.205 (0.864-1.680)	0.273	-	-
Antiarrhythmic	1.469 (0.962-2.244)	0.075	-	-
Antihyperlipidemic	0.678 (0.441-1.043)	0.077	-	-
LVEDd (mm)	1.025 (1.009-1.034)	0.002	-	-
LVESd (mm)	1.047 (1.016-1.046)	<0.0001	-	-
LAd (mm)	1.054 (1.032-1.077)	<0.0001	-	-
LVEF (%)	0.908 (0.882-0.934)	<0.0001	-	-
Mitral regurgitation moderate or severe	2.867 (1.973-4.168)	<0.0001	1.956 (1.224-3.126)	0.005
Tricuspid regurgitation moderate or severe	2.647 (1.964-3.567)	<0.0001	-	-
Aortic regurgitation moderate or severe	2.275 (1.264-4.094)	0.006	-	-
TRV (m/s)	1.622 (1.203-2.187)	0.002	-	-
TDI Rvsm (m/s)	0.814 (0.767-0.865)	<0.0001	-	-
TAPSE (mm)	0.879 (0.849-0.910)	<0.0001	0.945 (0.904-0.987)	0.011
SPAP (mmHg)	1.023 (1.014-1.032)	<0.0001	-	-
NTpro-BNP (log) (pg/ml)	2.871 (2.274-3.625)	<0.0001	1.531 (1.074-2.183)	0.019

*Values are HR and 95% CI, p value <0.05. ACEi angiotensin-converting enzyme inhibitor, AF atrial fibrillation, ARB angiotensin receptor blocker, ARNI angiotensin receptor-neprilysin inhibitor, BSA body surface area, BMI body mass index, COPD chronic obstructive pulmonary disease, CRT cardiac resynchronization therapy, DBP diastolic blood pressure, ICD implantable cardioverter-defibrillator, IQR interquartile range, LAd left atrial diameter, LVEDd left ventricular end-diastolic diameter, LVEF left ventricular ejection fraction, LVESd left ventricular end-systolic diameter, NYHA FC New York Heart Association Functional Class, RVsm right ventricular systolic motion tissue Doppler imaging, SBP systolic blood pressure, SGLT2-i sodium-glucose co-transporter 2 inhibitors, SPAP systolic pulmonary artery pressure, TAPSE tricuspid annular plane systolic excursion, TRV tricuspid regurgitation velocity.

Table 6. Non-ischemic cardiomyopathy patients, LVEF <50% binary logistic regression univariate and multivariate analysis

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	*p* value	HR (95% CI)	*p* value
Age years	1.000 (0.985–1.014)	0.949	–	–
Gender male	1.181 (0.809–1.726)	0.389		–
BMI (kg/m^2)	0.997 (0.936–1.061)	0.916	–	–
BSA (m^2)	0.530 (0.139–2.020)	0.352	–	–
SBP (mmHg)	0.969 (0.957–0.981)	<0.0001	–	–
DBP (mmHg)	0.974 (0.957–0.990)	0.002	–	–
NYHA FC III–IV	7.513 (4.746–11.892)	<0.0001	3.486 (1.886–6.441)	<0.0001
Arterial hypertension	0.719 (0.467–1.106)	0.133	–	–
Diabetes mellitus	0.877 (0.545–1.412)	0.590	–	–
Dyslipidemia	0.896 (0.507–1.583)	0.706	–	–
Smoking history	0.985 (0.651–1.488)	0.941	–	–
COPD	1.266 (0.463–3.459)	0.645	–	–
Renal disease	0.349 (0.075–1.631)	0.181	–	–
Ischemic cerebrovascular event	2.747 (0.982–7.686)	0.054	–	–
Chronic liver disease	–	–	–	–
Atrial fibrillation	1.957 (1.105–3.466)	0.021	–	–
ICD	1.326 (0.923–1.904)	0.127		
CRT	0.883 (0.512–1.524)	0.655	–	–
Family history	1.002 (0.652–1.538)	0.994	–	–
ACEi or ARB	0.931 (0.602–1.439)	0.747	–	–
ARNI	0.525 (0.278–0.993)	0.048	–	–
Beta-blocker	0.059 (0.008–0.468)	0.007	–	–
Aldosterone antagonist	1.201 (0.546–2.640)	0.649	–	–
SGLT2-i	0.568 (0.344–0.939)	0.028	–	–
Loop diuretic	4.155 (2.468–6.995)	<0.0001	–	–
Anticoagulant	1.363 (0.910–2.041)	0.133	–	–
Antiplatelet	0.911 (0.631–1.315)	0.618	–	–
If channel blocker	0.824 (0.566–1.199)	0.311	–	–
Digoxin	2.426 (1.516–3.880)	<0.0001	–	–
Antiarrhythmic	3.258 (1.668–6.363)	0.001	–	–
Antihyperlipidemic	0.640 (0.382–1.073)	0.091	–	–
LVEDd (mm)	1.066 (1.042–1.089)	<0.0001	1.050 (1.017–1.084)	0.002
LVESd (mm)	1.071 (1.050–1.092)	<0.0001	–	–
LAd (mm)	1.114 (1.079–1.149)	<0.0001	–	–
LVEF (%)	0.876 (0.835–0.901)	<0.0001	–	–
Mitral regurgitation moderate or severe	3.128 (2.119–4.888)	<0.0001	–	–
Tricuspid regurgitation moderate or severe	3.681 (2.517–5.383)	<0.0001	–	–
Aortic regurgitation moderate or severe	2.835 (1.096–7.333)	0.032	–	–
TRV (m/s)	2407 (1.561–3.713)	<0.0001	–	–
TDI Rvsm (m/s)	0.738 (0.676–0.804)	<0.0001	–	–
TAPSE (mm)	0.846 (0.808–0.887)	<0.0001	0.936 (0.880–0.996)	0.036
SPAP (mmHg)	1.038 (1.024–1.051)	<0.0001	–	–
NTpro-BNP (log) (pg/ml)	6.926 (4.551–10.539)	<0.0001	2.379 (1.357–4.171)	0.002

ACEi angiotensin-converting enzyme inhibitor, AF atrial fibrillation, ARB angiotensin receptor blocker, ARNI angiotensin receptor-neprilysin inhibitor, BSA body surface area, BMI body mass index, COPD chronic obstructive pulmonary disease, CRT cardiac resynchronization therapy, DBP diastolic blood pressure, ICD implantable cardioverter-defibrillator, IQR interquartile range, LAd left atrial diameter, LVEDd left ventricular end-diastolic diameter, LVEF left ventricular ejection fraction, LVESd left ventricular end-systolic diameter, NYHA FC New York Heart Association Functional Class, RVsm right ventricular systolic motion tissue Doppler imaging, SBP systolic blood pressure, SGLT2-i Sodium-glucose co-transporter 2 inhibitors, SPAP systolic pulmonary artery pressure, TAPSE tricuspid annular plane systolic excursion, TRV tricuspid regurgitation velocity. *Values are HR and 95% CI, p value <0.05.

OP-041 [Heart Failure]

Risk stratification of ventricular tachycardia in non-ischemic cardiomyopathy: Integrating cardiac MRI and ICD-derived arrhythmia data

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Background and Aim: Risk stratification for Ventricular Tachycardia (VT) in patients with Non-Ischemic Cardiomyopathy (NICM) remains challenging despite the prognostic utility of Cardiac Magnetic Resonance (CMR). While LVEF is the conventional risk marker, its predictive power is limited. We aimed to identify independent predictors of VT in strictly defined NICM and to evaluate the incremental prognostic performance of a logistic regression–based combined risk score compared with CMR-derived LVEF.

Methods: We retrospectively analyzed 438 patients with angiographically or CT-confirmed NICM who underwent systematic clinical, echocardiographic, and CMR evaluation. VT events were defined by interrogation of Implantable Cardioverter-Defibrillator (ICD) data, including appropriate shocks, Anti-Tachycardia Pacing (ATP), and episodes of Non-Sustained VT (NSVT). In addition, VT episodes documented on 12-lead surface ECG outside ICD recordings were also categorized as VT. Patients were classified as VT (n=32, 7.3%) or non-VT (n=406, 92.7%) during follow-up. Baseline demographics, comorbidities, therapies, imaging parameters, and outcomes were compared. Logistic regression was performed to identify predictors of VT, and a combined risk score was calculated

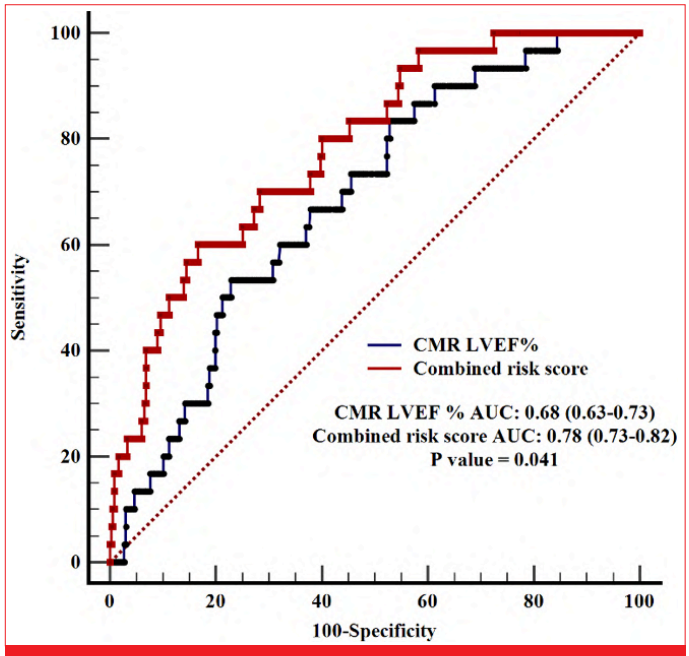

Figure 1. ROC curves comparing CMR-derived LVEF and combined risk score for prediction of ventricular tachycardia.

Table 1. Baseline demographic and clinical characteristics of the study population with a comparative analysis between patients presenting with VT and those without VT

	Total N=438	Non-VT N=406	VT N=32	p value
Age, years	42.0 ± 14.3	42.5 ± 14.3	36.0 ± 13.8	0.014
Male, n (%)	294 (67.1)	273 (67.2)	21 (65.6)	0.851
BMI, kg/m2	27.7 ± 5.6	27.7 ± 5.6	26.4 ± 5.5	0.207
BSA, m2	1.9 ± 0.2	1.9 ± 0.2	1.8 ± 0.2	0.257
NYHA FC III-IV, n (%)	93 (21.2)	84 (22.3)	9 (28.1)	0.449
Arterial Hypertension, n (%)	100 (22.8)	96 (23.6)	4 (12.5)	0.148
Diabetes Mellitus, n (%)	81 (18.5)	77 (19.0)	4 (12.5)	0.364
Dyslipidemia, n (%)	29 (6.6)	27 (6.7)	2 (6.3)	0.643
Smoking, (%)	119 (27.2)	113 (27.8)	6 (18.8)	0.266
Renal Disease, n (%)	23 (5.3)	23 (5.7)	0 (0.0)	0.167
Malignancy, n (%)	-	-	-	-
AF, n (%)	61 (14.2)	57 (14.3)	4 (12.5)	0.516
Intracardiac Device	-	-	-	-
ICD, n (%)	100 (22.8)	68 (16.7)	32 (100)	<0.0001
CRT, n (%)	21 (4.8)	18 (4.4)	3 (9.4)	0.192
Family History of Heart Failure, n (%)	60 (14.0)	51 (12.8)	9 (29.0)	0.019
SBP, mmHg	116 ± 16	117 ± 16	106 ± 13	<0.0001
DBP, mmHg	73 ± 11	73 ± 11	67 ± 10	0.002
NTpro-BNP, pg/mL (IQR)	1166 (313-3636)	1187 (310-3904)	1131 (508-3269)	0.997
Follow-up Time Days (IQR)	33.8 (15.5-63.8)	32.7 (14.2-62.6)	53.5 (32.3-74.1)	0.002
All Cause Mortality, n (%)	69 (15.8)	67 (16.5)	2 (6.3)	0.125
LVAD, n (%)	36 (8.2)	30 (7.4)	6 (18.8)	
Heart Transplantation, n (%)	5 (1.1)	5 (1.2)	0	
HF Related Hospitalisation, n (%)	62 (14.2)	56 (13.8)	6 (18.8)	0.442
Myocardial Recovery, n (%)	93 (21.2)	88 (21.7)	5 (15.6)	0.420

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

Table 2. Baseline medical therapy of the study population with a comparative analysis between patients presenting with VT and those without VT

	Total N=438	Non-VT N=406	VT N=32	p value
ACEi or ARB, n (%)	322 (73.5)	303 (74.6)	19 (59.4)	0.060
Beta-blocker, n (%)	413 (94.3)	381 (93.8)	32 (100)	0.142
Aldosterone Antagonist, n (%)	322 (73.5)	292 (71.9)	30 (93.8)	0.007
ARNI, n (%)	63 (14.4)	51 (12.6)	12 (37.5)	<0.0001
SGLT2-i, n (%)	91 (20.8)	85 (20.9)	6 (18.8)	0.769
Anticoagulant, n (%)	114 (26.0)	105 (25.9)	9 (28.1)	0.779
Antiplatelet, n (%)	178 (40.6)	162 (39.9)	16 (50.0)	0.263
If Channel Blocker, n (%)	89 (20.3)	82 (20.2)	7 (21.9)	0.820
Loop Diuretic	269 (61.4)	246 (60.6)	23 (71.9)	0.207

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

Table 3. Baseline echocardiographic parameters of the study population with a comparative analysis between patients presenting with VT and those without VT

	Total N=438	Non-VT N=406	VT N=32	p value
LVEDd, mm	60.0 ± 9.7	59.6 ± 9.7	64.4 ± 9.2	0.008
LVESd, mm	49.3 ± 12.3	48.8 ± 12.2	55.9 ± 12.2	0.002
LAd, mm	43.4 ± 7.5	43.4 ± 7.5	42.8 ± 8.0	0.640
LVEF, %	31.4 ± 12.3	31.9 ± 12.4	24.5 ± 7.9	<0.0001
Mitral Regurgitation Moderate or Severe, n (%)	173 (41.7)	159 (41.5)	14 (43.8)	0.805
Aortic Regurgitation Moderate or Severe, n (%)	18 (4.3)	18 (4.7)	0 (0)	0.229
Tricuspid Regurgitation Moderate or Severe, n (%)	103 (25.0)	96 (25.1)	7 (23.3)	0.827
TRV, m/sec	2.7 ± 0.7	2.6 ± 0.5	3.2 ± 1.9	0.204
TDI RVsm, m/sec	10.7 ± 2.7	10.7 ± 2.7	10.0 ± 3.2	0.171
TAPSE, mm	18.7 ± 5.2	18.8 ± 5.2	16.9 ± 5.0	0.067
SPAP, mmHg	37.9 ± 15.8	37.1 ± 14.1	47.6 ± 28.8	0.108

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

Table 4. Baseline cardiac magnetic resonance parameters of the study population with a comparative analysis between patients presenting with VT and those without VT

	Total N=438	Non-VT N=406	VT N=32	p value
IVS, mm	9.6 ± 2.1	9.6 ± 2.1	8.8 ± 2.1	0.048
LVEDd, mm	61.5 ± 10.5	61.0 ± 10.3	68.0 ± 10.3	<0.0001
LVESd, mm	52.0 ± 12.4	51.4 ± 12.2	60.4 ± 11.5	<0.0001
LAd, mm	46.6 ± 9.2	46.5 ± 9.2	47.6 ± 9.1	0.510
LVEDV, mL	249.2 ± 100.3	246.7 ± 100.8	282.1 ± 90.1	0.055
LVESV, mL	178.2 ± 92.9	175.2 ± 92.8	217.3 ± 87.6	0.014
LVEF, %	30.5 ± 11.7	31.0 ± 11.7	23.5 ± 8.9	<0.0001
SV, mL	69.4 ± 27.8	69.9 ± 28.3	62.0 ± 18.7	0.120
CO, L.min	5.3 ± 2.0	5.3 ± 2.1	4.6 ± 1.3	0.067
CI, L.min/m2	2.7 ± 1.0	2.7 ± 1.0	2.4 ± 0.6	0.110
LVmass, gr	163.7 ± 57.9	164.6 ± 58.4	153.3 ± 52.6	0.362
Late Gadolinium Enhancement, n (%)	119 (27.2)	106 (26.1)	13 (40.6)	0.076
Subendocardial LGE, n (%)	23 (5.3)	22 (5.4)	1 (3.4)	0.486
Midwall LGE, n (%)	76 (17.4)	69 (17.0)	7 (21.9)	0.483
Subepicardial LGE, n (%)	20 (4.6)	15 (3.7)	5 (15.6)	0.011

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

from predicted probabilities. ROC analysis was used to compare its discriminative ability with that of CMR LVEF.

Results: VT patients were younger (36.0 ± 13.8 vs. 42.5 ± 14.3 years, p=0.014), had lower systolic blood pressure (106 ± 13 vs. 117 ± 16 mmHg, p<0.0001), and more often had a family history of heart failure (29.0% vs. 12.8%, p=0.019). They also showed greater LV dilation and reduced function by echocardiography (LVEF 24.5 ± 7.9% vs. 31.9 ± 12.4%, p<0.0001) and CMR (LVEF 23.5 ± 8.9% vs. 31.0 ± 11.7%, p<0.0001). Subepicardial late gadolinium enhancement (LGE) was significantly more prevalent among VT patients (15.6% vs. 3.7%, p=0.011). In multivariate analysis, independent predictors of VT were: family history of heart failure (OR 3.40, 95% CI 1.10–10.48, p=0.033), lower systolic blood pressure (OR 0.96, 95% CI 0.92–0.998, p=0.037), reduced LVEF on CMR (OR 0.93, 95% CI 0.88–0.99, p=0.024), ARNI therapy (OR 3.16, 95% CI 1.11–9.05, p=0.032), and subepicardial LGE (OR 6.57, 95% CI 1.48–29.19, p=0.013). ROC analysis demonstrated that the combined risk score significantly outperformed LVEF alone in VT prediction (AUC 0.78, 95% CI 0.73–0.82 vs. 0.68, 95% CI 0.63–0.73; p=0.041).

Conclusions: In this large NICM cohort, VT occurred in 7.3% of patients and was independently predicted by clinical (family history, systolic blood pressure, ARNI therapy) and imaging (LVEF, subepicardial LGE) parameters. A combined risk score derived from these predictors provided superior discriminatory ability compared with conventional LVEF, underscoring the incremental value of integrated clinical-imaging risk stratification for guiding preventive strategies such as ICD implantation.

Table 5. Clinical characteristics of patients with VT: Findings from univariate and multivariate regression analyses

	Univariate Analysis	Univariate Analysis	Multivariate Analysis	Multivariate Analysis
	OR (95% CI)	p value	OR (95% CI)	p value
Age, years	0.968 (0.942-0.994)	0.016	-	-
Male, %	0.930 (0.436-1.985)	0.851	-	-
BMI, kg/m2	0.956 (0.892-1.025)	0.207	-	-
BSA, m2	0.402 (0.083-1.938)	0.256	-	-
NYHA FC III-IV	1.365 (0.608-3.062)	0.450	-	-
Arterial Hypertension, n (%)	0.461 (0.158-1.348)	0.157	-	-
Diabetes Mellitus, n (%)	0.610 (0.208-1.791)	0.369	-	-
Dyslipidemia, n (%)	0.936 (0.212-4.126)	0.930	-	-
Smoking History, n (%)	0.598 (0.240-1.492)	0.271	-	-
Renal Disease, n (%)	-	-	-	-
AF, n (%)	0.857 (0.290-2.535)	0.781	-	-
ICD, n (%)	-	-	-	-
CRT, n (%)	2.230 (0.621-8.013)	0.219	-	-
Family History of h Heart Failure, n (%)	2.775 (1.211-6.361)	0.016	3.400 (1.103-10.482)	0.033
SBP, mmHg	0.955 (0.931-0.980)	0.001	0.958 (0.920-0.998)	0.037
DBP, mmHg	0.946 (0.913-0.981)	0.003	-	-
Log-NTpro-BNP	1.009 (0.634-1.606)	0.970	-	-

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

Table 6. Medical therapies of patients with VT: Findings from univariate and multivariate regression analyses

	Univariate Analysis	Univariate Analysis	Multivariate Analysis	Multivariate Analysis
	OR (95% CI)	p value	OR (95% CI)	p value
ACEi or ARB, n (%)	0.497 (0.237-1.041)	0.064	-	-
Beta-Blocker, n (%)	-	-	-	-
Aldosterone Antagonist, n (%)	5.856 (1.377-24.906)	0.017	-	-
Anticoagulant, n (%)	1.122 (0.503-2.501)	0.779	-	-
Antiplatelet, n (%)	1.506 (0.733-3.097)	0.265	-	-
If Channel Blocker, n (%)	1.106 (0.462-2.647)	0.820	-	-
ARNI, n (%)	4.176 (1.927-9.052)	<0.0001	3.163 (1.105-9.052)	0.032
SGLT-2i, n (%)	0.871 (0.348-2.185)	0.769	-	-
Loop diuretic, n (%)	1.662 (0.750-3.684)	0.211	-	-

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

Table 7. Echocardiographic parameters of patients with VT: Findings from univariate and multivariate regression analyses

	Univariate Analysis	Univariate Analysis	Multivariate Analysis	Multivariate Analysis
	OR (95% CI)	p value	OR (95% CI)	p value
LVEDd, mm	1.050 (1.012-1.090)	0.009	-	-
LVESd, mm	1.048 (1.017-1.081)	0.002	-	-
LAd, mm	0.989 (0.942-1.037)	0.639	-	-
LVEF, %	0.929 (0.888-0.972)	0.002	-	-
Mitral Regurgitation Moderate or Severe	1.096 (0.529-2.268)	0.805	-	-
Aortic Regurgitation Moderate or Severe	-	-	-	-
Tricuspid Regurgitation Moderate or Severe	0.907 (0.377-2.180)	0.827	-	-
TRV, m/sec	1.928 (0.985-3.772)	0.055	-	-
RVsm, m/sec	0.903 (0.780-1.045)	0.171	-	-
TAPSE, mm	0.931 (0.862-1.005)	0.068	-	-
SPAP, mmHg	1.031 (1.008-1.056)	0.009	-	-

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

Table 8. Cardiac magnetic resonance parameters of patients with VT: Findings from univariate and multivariate regression analyses

	Univariate Analysis	Univariate Analysis	Multivariate Analysis	Multivariate Analysis
	OR (95% CI)	p value	OR (95% CI)	p value
IVS, mm	0.825 (0.680-1.000)	0.050	-	-
LVEDd, mm	1.062 (1.027-1.098)	<0.0001	-	-
LVESd, mm	1.060 (1.029-1.093)	<0.0001	-	-
LAd, mm	1.013 (0.975-1.052)	0.509	-	-
LVEF, %	0.941 (0.908-0.975)	0.001	0.931 (0.875-0.990)	0.024
LEDV, mL	1.003 (1.000-1.006)	0.057	-	-
LESV, mL	1.004 (1.0001-1.008)	0.016	-	-
SV, mL	0.987 (0.972-1.003)	0.114	-	-
CO, L.min	0.812 (0.652-1.010)	0.062	-	-
CI, L.min/m^2	0.705 (0.461-1.077)	0.106	-	-
LV mass, gr	0.996 (0.989-1.004)	0.362	-	-
Late gadolinium enhancement	1.936 (0.924-4.056)	0.080	-	-
Subendocardial LGE	0.563 (0.073-4.318)	0.581	-	-
Midwall LGE	1.368 (0.569-3.288)	0.484	-	-
Subepicardial LGE	4.827 (1.632-14.281)	0.004	6.573 (1.480-29.190)	0.013

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

OP-042 [Heart Failure]

Rationale and design of the Türkiye Heart Failure (TURK-HF) registry

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Background and Aim: The Türkiye Heart Failure (TURK-HF) registry was designed to address the knowledge gap regarding the identification, management, and long-term prognosis of patients with heart failure (HF) in real-world settings. The registry also aimed to document barriers to externalizing current guidelines for routine patient care in the country.

Methods: The TURK-HF registry is a national, multicenter, prospective, observational study of unselected patients with HF, regardless of ejection fraction, who presented with de novo, chronic, or acute HF. A total of 46 investigators from 38 centers in 22 cities across Türkiye participated in the study (Figure 1). The TURK-HF registry data will be collected using an electronic case report form (e-CRF) integrated into an electronic data capture system. The study investigators will gain access to the e-CRF via the www.turkhf.com website using their username and password. The TURK-HF registry was registered at ClinicalTrials.gov (ID: NCT06707220).

Results: The baseline assessment of patients will include socio-demographic data, frailty assessment, health-related quality of life (HRQoL) questionnaire, HF-related information, medical history and comorbidities, H2FPEF score calculation, and medical and device-based HF therapies (Figure 2-3). To determine the socio-economic status and health inequalities, detailed data will be obtained on age, sex, marital status, urban or rural living, housing conditions and household income, education level,

Figure 1.

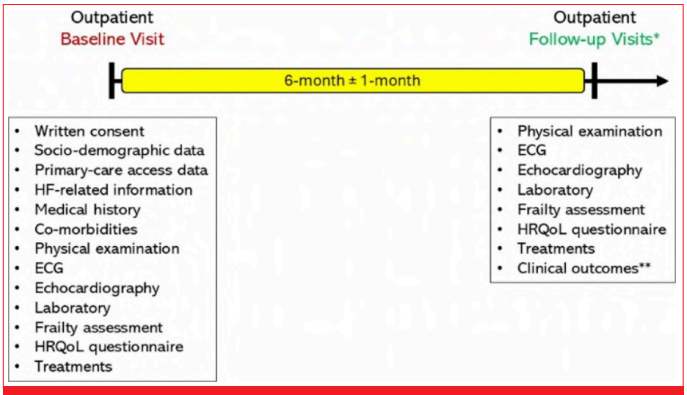
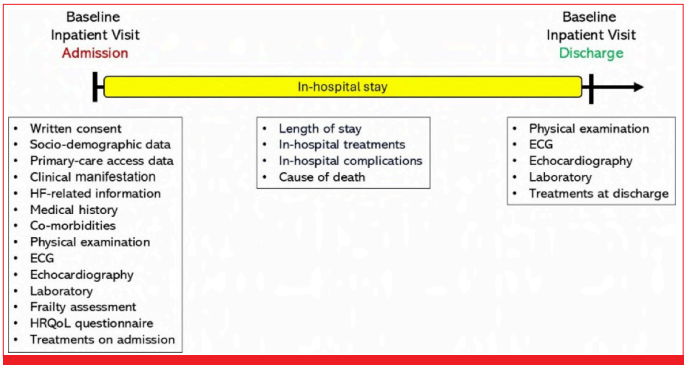


Figure 2.



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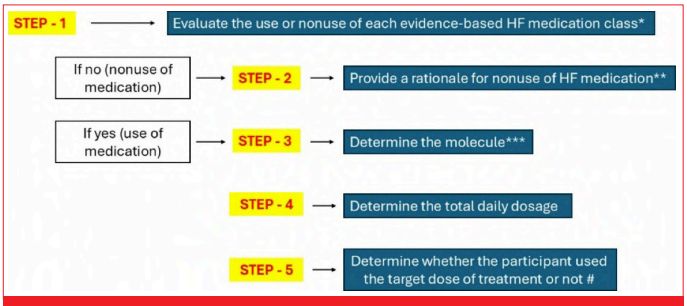

Figure 4.

and employment status. We integrated the Canadian Study of Health and Aging Clinical Frailty Scale into the TURK-HF registry to measure the clinical frailty of patients with HF. The TURK-HF registry was registered with the EuroQol Customer Portal (registration no: 68900), and the EQ-5D-5L questionnaire will be administered to patients enrolled in the TURK-HF registry to determine their HRQoL. We integrated an automatic calculation system into the e-CRF as a "decision support tool" to calculate the H2FPEF score in patients with HF with preserved ejection fraction. We integrated an assessment algorithm into the e-CRF to assess the use or non-use of GDMT by registry participants (Figure 4). Longitudinal data will be collected at regular outpatient visits every six months, with a margin of error of one month, or via telephone interviews if the patient is not eligible for outpatient visits (Figure 2). The clinical endpoints of the TURK-HF registry will reflect clinical "hard" endpoints, including cardiovascular mortality or all-cause mortality, morbidity outcomes (hospitalizations for HF), clinician-interpreted or patient-reported outcomes, and surrogate endpoints, either alone or in combination.

Conclusions: The TURK-HF registry offers comprehensive and distinctive insights into contemporary HF clinical characteristics, diagnostic methods, treatments, and outcomes. This registry has the potential to influence implementation strategies, clinical research, and public policies across Türkiye.

OP-043 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

The relationship between the left atrioventricular coupling index and new-onset atrial fibrillation in patients with obstructive and nonobstructive hypertrophic cardiomyopathy

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Background and Aim: Hypertrophic cardiomyopathy (HCM) is a myocardial disease characterized by an enlarged left ventricle and disorganized cardiac myocytes, which can result in ventricular dysfunction. It affects about 1 in 500 people and may lead to complications such as arrhythmias, systemic thromboembolism, and heart failure. Atrial fibrillation (AF) is the most common persistent arrhythmia in HCM, with both clinical and subclinical episodes happening in almost half of the patients. The left atrioventricular coupling index (LACI) is a new echocardiographic measurement showing promise in predicting patient outcomes across different clinical settings. Evaluating the coupling between the left atrium and left ventricle may provide a more accurate assessment of left atrioventricular dysfunction. This study aimed to examine the relationship between new-onset AF (NOAF) and LACI according to obstructive and non-obstructive HCM.

Methods: This retrospective study examined 171 patients diagnosed with obstructive and non-obstructive HCM patients. Each participant underwent transthoracic echocardiography prior to the procedure, and LACIs were calculated for all patients. LACI was defined by the ratio of left atrial (LA) end-diastolic volume divided by left ventricular (LV) enddiastolic volume (Figure 1). Patients were monitored for 36 months to identify any occurrences of NOAF.

Results: The baseline demographic, clinical, and imaging characteristics of the patients are summarized in Table 1. The study sample consisted of 171 patients with HCM. The patients were monitored for 36 months. The study's primary outcome NOAF in HCM patients was significantly higher in those with higher LACI (%) (47 ± 11 vs. 54 ± 16, p=0.028). Univariate logistic regression analysis revealed significant correlations between NOAF in HCM patients with gender, ICD (implantable cardioverter-defibrillator) and LACI. Further analysis of these variables using the multivariate logistic regression analysis indicated that LACI (OR: 1.46, 95% CI: 1.097-1.944; p=0.01) and ICD were independent predictor for the development of NOAF in HCM patients (Table 2). In the ROC analysis, the LACI optimal cut-off value of >0.57 (57%) predicted NOAF in patients with HCM, showing 43.24% sensitivity and 88.06% specificity (AUC: 0.618 [95% CI: 0.541–0.691, p=0.046) (Figure 2).

Conclusions: In this study, we showed that LACI, a new left atrioventricular coupling index, is significantly linked to NOAF in patients with obstructive and nonobstructive hypertrophic cardiomyopathy. Importantly for clinical use, LACI is easy to calculate from standard echocardiographic images, requiring no additional imaging or postprocessing software. Notably, LACI provided a more powerful risk stratification for NOAF than traditional LA parameters. A higher LACI, which indicates LA-LV uncoupling, was independently linked to the occurrence of AF in HCM patients, showing greater and added predictive value compared to traditional LA parameters.

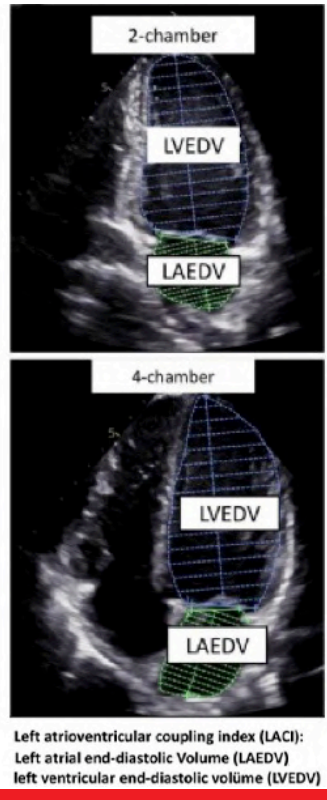

Figure 1. Measurement of left atrioventricular coupling index (LACI) using echocardiography.

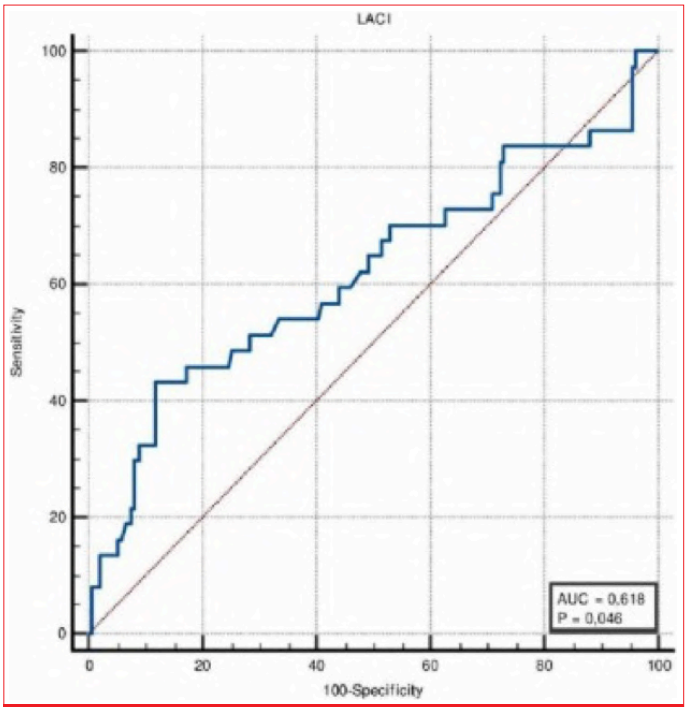

Figure 2. ROC curve analysis of the left atrioventricular coupling index (LACI) in patients with hypertrophic cardiomyopathy who experienced new-onset atrial fibrillation (NOAF). LACI: Left atrioventicular coupling index, ROC: Receiver operating characteristic curve.

Table 1. Demographic, clinical, and echocardiographic characteristics of the study group

	NOAF- (n=134)	NOAF- (n=134)	NOAF+ (n=37)	NOAF+ (n=37)	p value
Age (years)	51.2 ± 12.9		55.5 ± 10.4		0.111
Gender, n (%) (Male)	106	79.1	24	64.9	0.073
Patients with HT, n (%)	66	49.3	25	67.6	0.049
Patients with DM, n (%)	19	14.2	10	27	0.066
Smokig Status, n (%)	68	50.7	13	35.1	0.093
BMI (kg/m2)	27.21 ± 3.95		27.92 ± 3.45		0.19
NYHA class I/II (n, %)	108	80.6	30	91.1	0.968
NYHA class III/IV (n, %)	26	19.4	7	18.9	0.968
ICD (n, %)	10	7.5	9	24.3	0.004
LV mass index (g/m2)	81.32 ± 21.25		85.37 ± 13.58		0.025
Moderate or severe MR (n, %)	22	16.4	6	16.2	0.977
Rest LVOT gradient (mmHg)	25	(22-36)	23	(21-28)	0.055
E/A	0.97 ± 0.37		1.06 ± 0.52		0.674
E/e'	8	(0.88-13)	10.55	(6.98-16.2)	0.128
LAVI (mL/m2)	34.5 ± 4.4		36.6 ± 6.3		0.213
LVEDVI (mL/m2)	76.2 ± 18.7		72.2 ± 17.1		0.117
LACI (%)	47 ± 11		54 ± 16		0.028
LVEF (%)	62.4 ± 7.2		60.7 ± 6.9		0.089

NOAF: New-onset atrial fibrillation, p: Probability statistic, DM: Diabetes mellitus, BMI: Body mass index, NYHA: New York Heart Association, ICD: Implantable cardioverter-defibrillator, MR: Mitral regurgitation, LV: Left ventricle, LVEF: Left ventricular ejection fraction. LAVI: Indexed LA volume, LVEDVI: Indexed LV enddiastolic volume, LACI: Left atrioventricular coupling index.

Table 2. Results of the univariate and multivariate analyses of the variables regarding their prognostic value in predicting atrial fibrillation in patients with hypertrophic cardiomyopathy

	Univariate Analysis	Univariate Analysis	Univariate Analysis	Multivariate Analysis	Multivariate Analysis	Multivariate Analysis
	Univariate OR, 95% CI	Univariate OR, 95% CI	p value	Multivariate OR, 95% CI	Multivariate OR, 95% CI	p value
Gender	2.162	(0.932-5.017)	0.072	-	-	-
ICD	4.62	(1.665-12.824)	0.003	3.986	(1.482-10.721)	0.006
LACI	1.449	(1.075-1.953)	0.015	1.46	(1.097-1.944)	0.01

OR: Odds ratio, CI: Confidence interval, p: Probability statistic, LACI: Left atrioventricular coupling index, ICD: Implantable cardioverter-defibrillator.

OP-044 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Impact of T-AMYLO risk score and red flag findings on cardiovascular outcomes in patients with cardiac conduction defects treated with intracardiac device implantation

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Background and Aim: Cardiac amyloidosis (CA) is more common than previously thought, with an incidence of up to 15% in aortic stenosis and heart failure with preserved ejection fraction (1,2). Pacemaker need in CA patients ranges from 9.5% to 20%, while its prevalence in those with cardiac conduction defects remains unclear (3). Additionally, data on validating risk scores and red flag findings for CA diagnosis in this group are highly limited. This study aims to investigate the effects of the T-AMYLO (4) score and red flag findings on cardiovascular outcomes in patients with cardiac conduction defects requiring intracardiac device implantation.

Methods: This retrospective, single-center cohort study included 1107 patients who underwent CIED implantation for unexplained cardiac conduction defects between 2015 and 2024. Exclusion criteria comprised pacemaker implantation after acute coronary syndrome, pre-existing cardiomyopathy, post-surgical or transcatheter conduction defects, congenital AV block, and metabolic causes. Clinical, laboratory, ECG, and echocardiographic data were collected from hospital records and the national registry. The T-AMYLO score, risk category, and red flag findings were assessed. Their effects on primary endpoints; all cause of mortality, non-fatal myocardial infarction, and non-fatal stroke were analyzed. Independent predictors for primary endpoints were identified using multivariate Cox regression, and survival impact was evaluated via Kaplan-Meier analysis. The study received ethical approval.

Results: Among the 1107 patients evaluated, 460 experienced a primary endpoint, including 346 cases of all cause of mortality. Adverse outcomes were more frequent in older and male patients. When examining CIED indications, patients with AV block and AF with slow ventricular response had higher event rates. Similarly, patients implanted with single ventricular lead devices experienced more adverse outcomes. Patients with adverse outcomes had higher T-AMYLO scores and a greater presence of red flag findings, including AV block, aortic valve disease, peripheral neuropathy, low voltage, and increased septal thickness. T-AMYLO score elevation (HR: 1.06, p=0.012), along with red flag findings such as aortic valve disease (HR: 1.29, p=0.016) and AV blocks (HR: 1.43, p=0.009), were identified as independent predictors of adverse outcomes. Kaplan-Meier analysis demonstrated a significant decline in survival as T-AMYLO risk group increased (p<0.001), with a similar trend observed as the total number of red flag findings increased (p<0.001).

Conclusions: The association of the T-AMYLO risk score and red flag findings with cardiovascular outcomes in patients requiring device

Table 1. Clinical and demographic characteristics of patients according to primary endpoints and all-cause mortality

	Primary Endpoints (-) (n: 647)	Primary Endpoints (+) (n: 470)	P value	All-Cause Mortality (-) (n: 761)	All-Cause Mortality (+) (n:346)	P value
Age, (years)*	74 (24-97)	82 (36-102)	**<0.001**	74 (24-97)	84 (49-102)	**<0.001**
Male, , n (%)	295 (46.3)	276 (58.7)	**<0.001**	370 (48.6)	201 (58.1)	**0.003**
Indications for Implantation, n (%)						
• AV Blocks, n (%)	226 (35.5)	216 (46.0)		270 (35.5)	172 (49.7)	
• Bundle Branch Conduction Defects (non AV block),n (%)	62 (9.7)	39 (8.3)	**<0.001**	71 (9.3)	30 (8.7)	**<0.001**
• Sick Sinus Syndrome and Sinusoidal Bradycardia, n (%)	249 (39.1)	105 (22.3)		295 (38.8)	59 (17.1)	
• AF with slow ventricular rate, n (%)	100 (15.7)	110 (23.4)		125 (16.4)	85 (24.6)	
Types of Implanted CIEDs, n (%)						
• VR pacemaker	128 (20.1)	139 (29.6)		146 (19.2)	121 (35.0)	
• DR pacemaker	389 (61.1)	215 (45.7)		460 (60.4)	144 (41.6)	
• VR-ICD	34 (5.3)	62 (13.2)	**<0.001**	49 (6.4)	47 (13.6)	**<0.001**
• DR-ICD	78 (12.2)	51 (10.9)		98 (8.6)	31 (2.8)	
• CRT-D	8 (1.3)	3 (0.6)		8 (0.7)	3 (0.3)	
Heart Failure: n (%)						
• - Patients with EF <50%	111 (17.4)	163 (37.7)	**<0.001**	154 (20.2)	120 (34.7)	**<0.001**
DM, n (%)	216 (33.9)	221 (47.0)	**<0.001**	266 (35.0)	171 (49.4)	**<0.001**
HT, n (%)	499 (78.3)	423 (90.0)	**<0.001**	604 (79.4)	318 (91.9)	**<0.001**
Stroke, n (%)	127 (19.9)	155 (33.0)	**<0.001**	161 (21.2)	121 (35.0)	**<0.001**
CAD, n (%)	171 (26.8)	228 (48.5)	**<0.001**	250 (32.9)	149 (43.1)	**0.001**
CKD, n (%)	132 (20.7)	191 (40.6)	**<0.001**	159 (20.7)	164 (47.4)	**<0.001**
AF, n (%)	287 (45.1)	256 (54.5)	**0.002**	338 (44.4)	205 (59.2)	**<0.001**
Senkop-presenkop, n (%)	481 (75.5)	334 (71.1)	0.097	569 (74.8)	246 (71.1)	0.199
Smoker, n (%)	156 (24.5)	113 (24.0)	0.860	191 (25.1)	78 (22.5)	0.358
Aortic Valve Disease**, n (%)						
• None	302 (47.4)	155 (33.0)		356 (46.8)	101 (29.2)	
• Mild to Moderate Aortic Valve Disease	274 (43.0)	247 (52.6)	**<0.001**	333 (43.8)	188 (54.3)	**<0.001**
• Severe Aortic Valve Disease	24 (3.8)	20 (4.3)		27 (3.5)	17 (4.9)	
• SAVR - TAVI	37 (5.8)	48 (10.2)		45 (5.9)	40 (11.6)	
Medical Treatments; n (%)						
• Beta Blocker	462 (72.5)	391 (83.2)	**<0.001**	570 (74.9)	283 (81.8)	**0.011**
• Non-DHP Calcium Channel Blocker	96 (15.1)	81 (17.2)	0.330	110 (14.5)	67 (19.4)	**0.039**
• Amiodarone	52 (8.2)	80 (17.0)	**<0.001**	69 (9.1)	63 (18.2)	**<0.001**
• Class 1 Anti-Arythmic Drugs	27 (4.2)	17 (3.6)	0.610	31 (4.1)	13 (3.8)	0.801
• Digoxin	114 (17.9)	116 (24.7)	**0.006**	133 (17.5)	97 (28.0)	**<0.001**
• ACE-i / ARB	491 (77.1)	420 (89.4)	**<0.001**	606 (79.6)	305 (88.2)	**<0.001**
• ARNI	30 (4.7)	20 (4.3)	0.710	37 (4.3)	13 (3.8)	0.412
• Oral Anticoagulant	439 (68.9)	368 (78.3)	**<0.001**	520 (68.3)	287 (82.9)	**<0.001**
• MRA	187 (29.4)	221 (47.0)	**<0.001**	235 (30.9)	173 (50.0)	**<0.001**
• Diuretic	491 (77.1)	445 (94.7)	**<0.001**	601 (79.0)	335 (96.8)	**<0.001**

*median (max-min)
**Aortic Valve Disease is defined as mild to moderate valve disease, mild or moderate aortic regurgitation and/or stenosis, severe aortic valve disease is defined as severe aortic regurgitation and/or stenosis, and SAVR-TAVI is defined as surgical or transcatheter aortic valve replacement.
ACEi: angiotensin-converting enzyme inhibitor, ARB: angiotensin receptor blocker, AF: atrial fibrillation, ARNI: angiotensin receptor/neprilysin inhibitor, AV: atrioventricular, CRT: cardiac resynchronisation therapy, CIED: cardiac implantable electronic device, CTS: carpale tunnel syndrome, DM: diabetes mellitus, DR: atrial and ventricular 2 leads, EF: ejection fraction, HF: heart failure, HT: hypertension, ICD: implantable cardioverter-defibrillator, CAD: coronary artery disease, CKD: chronic kidney disease, MVR: mitral valve replacement, MRA: aldosterone receptor antagonist, Non-DHP: non dihydropyridine calcium channel blocker, NYHA: New York Heart Association Classification, SAVR: surgical aortic valve replacement, SVO: cerebrovascular accident, SGLT-2: sodium-glucose cotransporter-2, TAVI: transcatheter aortic valve replacement, TVR: tricuspid valve replacement, VR: ventricular single lead

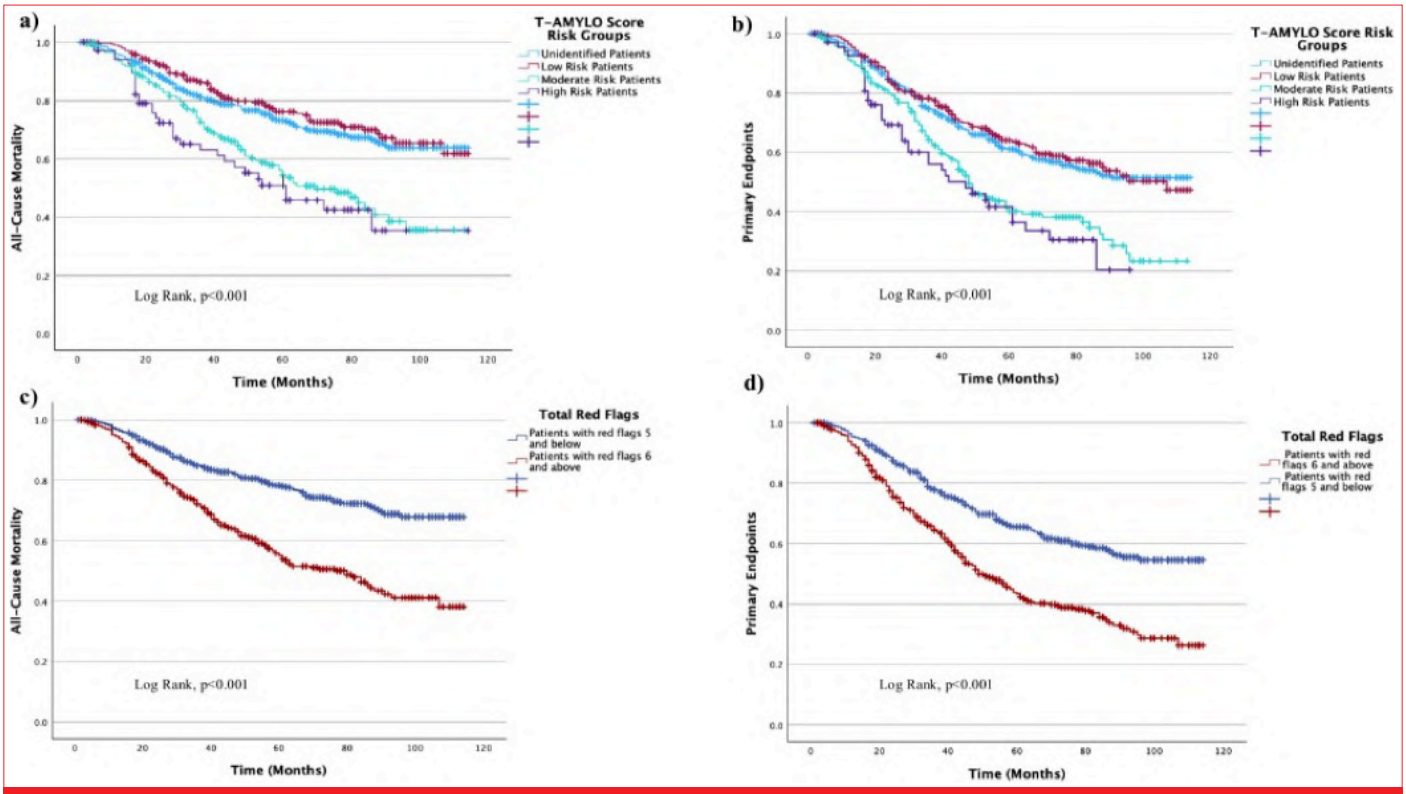

Figure 1. Kaplan–Meier curves demonstrating the impact of T-AMYLO risk score and total red flag findings on survival outcomes.

Table 2. Association of T-AMYLO risk score and red flag findings of cardiac amyloidosis with primary endpoints and all-cause mortality

	Primary Endpoints (-) (n: 647)	Primary Endpoints (+) (n: 470)	P Value	All-Cause Mortality (-) (n: 761)	All-Cause Mortality (+) (n: 346)	P Value
T-AMYLO Score Risk Groups, n (%)*						
• Unidentified patients	352 (55.3)	221 (47.0)		414 (54.4)	159 (46.0)	
• Low risk patients	182 (28.6)	108 (23.0)	<0.001	218 (28.6)	72 (20.8)	<0.001
• Medium risk patients	73 (11.5)	102 (21.7)		93 (12.2)	82 (23.7)	
• High-risk patients	30 (4.7)	39 (8.3)		36 (4.7)	33 (9.5)	
T-AMYLO Score, n**	1.0 (0.0-94.1)	2.9 (0.0-99.1)	<0.001	0.2 (0.0-99.1)	4.8 (1.8-99.1)	<0.001
Aortic Valve Disease, n(%)***	237 (37.2)	246 (52.3)	<0.001	287 (37.7)	196 (56.6)	<0.001
Heart Failure, (LVEF>40%), n (%)	354 (55.0)	301 (64.0)	0.006	423 (55.8)	226 (65.4)	0.004
Age > 65, n (%)	485 (76.1)	434 (92.3)	<0.001	592 (77.8)	327 (94.5)	<0.001
CTS, n (%)	106 (16.6)	83 (17.7)	0.656	123 (16.2)	66 (19.1)	0.233
Peripheral Neuropathy, n (%)	50 (7.8)	91 (19.4)	<0.001	63 (8.3)	78 (22.5)	<0.001
Autonomic Dysfunction, n (%)	115 (18.1)	96 (20.4)	0.321	142 (18.7)	69 (19.9)	0.615
Presence of AV Blocks****, n (%)	413 (64.8)	373 (79.4)	<0.001	495 (65.0)	291 (84.1)	<0.001
Low Voltage (IVS > 12mm) , n (%)	86 (13.5)	86 (18.3)	0.029	107 (14.1)	65 (18.8)	0.044
Low Voltage (IVS < 12mm) , n (%)	93 (14.6)	81 (17.2)	0.234	108 (14.2)	66 (19.1)	0.038
LVDD, n (%)	448 (70.3)	344 (73.2)	0.297	535 (70.3)	257 (74.3)	0.174
Pseudo Q Wave, n (%)	27 (4.2)	33 (7.0)	0.043	31 (4.1)	29 (8.4)	0.003
IVS > 12mm, n (%)	282 (44.3)	246 (52.3)	0.008	345 (45.3)	183 (52.9)	0.020
Total Number of Red Flags**	4.5 (0-9)	5.5 (1-9)	<0.001	5 (0-9)	6 (1-9)	<0.001
Total Follow-up Time, n (Month)**	70 (1-114)	39 (3-114)	<0.001	70 (1-114)	31 (3-107)	<0.001

*T-AMYLO Score risk group distinction was determined by evaluating the risk percentages of the patients according to the T-AMYLO score system. In this scoring system, patients with a risk percentage below 19.9% were considered as low risk, patients with a risk percentage between 20-74.9% were considered as medium risk, and patients with a risk percentage of 75% and above were considered as high risk. Patients to whom this scoring system can be applied are those with an IVS thickness of 12 mm and above. Due to this limiting step in the T-AMYLO scoring system, the risk group cannot be determined in patients with IVS < 12 mm. For this reason, a subgroup called 'patients with undetermined risk group' is specified in the risk group distinction.

**median (maximum-minimum)

***Aortic valve disease was considered as a red flag finding in all patients with echocardiographic evidence of stenosis and/or regurgitation accompanied by thickening and degenerative changes in the aortic leaflets.

****The presence of AV block was considered as a red flag finding in all patients with symptomatic bradycardia in the presence of bifascicular block or trifascicular block, all high-grade AV blocks of Mobitz type 2 and above, AV complete blocks and AF with slow ventricular rate.

AF: atrial fibrillation, AV: atrioventricular, CTS: carpale tunnel syndrome, IVS: interventricular septum, LVEF: left ventricular ejection fraction, LVDD: left ventricular diastolic dysfunction.

implantation underscores the need to validate T-AMYLO in this population. Investigating these risk scores, especially in unexplained cardiac conduction defects, could aid early cardiac amyloidosis diagnosis and help reduce mortality.

OP-045 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Catheter ablation with a zero-fluoroscopy approach without intracardiac imaging: A single-center experience

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Background and Aim: Advancements in catheter ablation techniques and technologies have brought about significant changes in the management of arrhythmias. In particular, the use of three-dimensional electroanatomic mapping has improved treatment success and reduced fluoroscopy time. Despite this, fluoroscopy is still used in the majority of procedures, leading to issues such as cumulative radiation exposure for the operator and orthopedic problems caused by protective lead aprons. Performing procedures without fluoroscopy can reduce radiation exposure and potentially prevent such orthopedic complications. Although intracardiac imaging facilitates a zero-fluoroscopy approach, it is not an absolute requirement. This study investigates the outcomes of catheter ablation performed using a zero-fluoroscopy approach for various arrhythmias in a tertiary care center.

Methods: Catheter ablation procedures performed at the Department of Cardiology, Ankara University Faculty of Medicine,

between January 1, 2021, and June 1, 2025, were retrospectively reviewed. Cases performed with a zero-fluoroscopy approach, based on operator preference, were selected from this population. In patients requiring left atrial access, patent foramen ovale (PFO) was used or transseptal puncture was performed under transesophageal echocardiography (TEE) guidance. Left ventricular access, when necessary was achieved through the retroaortic approach. Patients who underwent coronary angiography were excluded. Patient outcomes were assessed through chart reviews.

Results: A total of 38 procedures in 38 patients were included in the study (Table 1). The median left ventricular ejection fraction (LVEF) was 60% (IQR: 60–58.7). Atrial fibrillation (AF) ablation was performed in 4 patients (10.5%), cavotricuspid isthmus (CTI) flutter ablation in 4 patients (10.5%), premature ventricular contraction (PVC) ablation in 26 patients (68.4%), atrioventricular nodal reentrant tachycardia (AVNRT) ablation in 1 patient (2.6%), and Wolff–Parkinson–White (WPW) syndrome ablation in 1 patient (2.6%). Acute success was not achieved in 1 patient who underwent papillary muscle PVC ablation (Table 2). One patient experienced delayed cardiac tamponade. There was no periprocedural mortality. During follow-up, recurrence was observed in 4 patients with PVCs.

Conclusions: This study demonstrates that catheter ablation of both supraventricular and ventricular arrhythmias using a zero-fluoroscopy approach with three-dimensional electroanatomic mapping is feasible in selected patients.

Table 1. Study population and results

Age, yrs (IQR)	57(65.2-50)
Sex, female , n(%)	19(50)
Hypertension, n(%)	20(52.6)
Diabettes mellitus, n(%)	8(21.1)
Coronary artery disease, n(%)	10(26.3)
Atrial fibrillation, n(%)	10(26.3)
EF	60(60-58.7)
History of stroke, n(%)	1(2.6)
Severe valvular disease, n(%)	1(2.6)
COPD, n(%)	1(2.6)
Ablation, n(%) AF CTI flutter PVC AVNRT WPW	 4(10.5) 6(15.8) 26(68.4) 1(2.6) 1(2.6)

Table 2. Procedural and follow-up data

	AF 4(10.5)	CTI 4(10.5)	PVC 26(68.4)	AVNRT 1(2.6)	WPW 1(2.6)
Access to left sided structres	PFO (3) TEE ile TSP (1)	-	Retroaortik (17)	-	PFO (1)
Tamponade, n(%)	0	0	1(2.6)	0	0
Hospitalization, Days, n(%)	2	1	1	1	1
Acute success, n(%)	4(100)	4(100)	25(96.1)	1(100)	1(100)
Arrhythmia free survival, n(%)	4(100)	4(100)	21(80.7)	1(100)	1(100)

OP-046 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

The interpretation of CHA2DS2-VA score components in clinical practice

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Background and Aim: Atrial fibrillation (AF) is a growing global health burden and the most common cardiac rhythm disorder worldwide. Currently, the most widely used stroke risk stratification tool is the CHA_2DS_2-VA score, introduced in the 2024 ESC guidelines. The present online survey aims to assess variations in the interpretation of the 'C', 'H', 'D', and 'V' components of the CHA_2DS_2-VA score among clinicians in Turkey, and to analyze their attitudes and approaches regarding its clinical application in order to identify educational and informational needs.

Methods: The questionnaire consisted of 10 multiple-choice questions, including both single best answer and multiple-answer formats. The survey link was distributed to physicians via WhatsApp and remained accessible between 2024 and 2025.

Results: The majority of the 178 participants were general cardiologists (49.7%). Overall adherence to the ESC-defined scoring criteria was found to be good. The most pronounced variability was observed in the interpretation of the 'C' component, particularly regarding the relevance of left ventricular ejection fraction (LVEF) and brain natriuretic peptide (BNP) in scoring, as well as in the inclusion of a single elevated blood pressure reading in the 'H' component, and the exclusion of patients with type 1 diabetes mellitus from the 'D' component. 59.4% of the participants expressed greater confidence in scoring the 'H' component, whereas this proportion was lower for the 'C' (21.1%) and 'V' (19.4%) components. The majority of participants (43.4%) relied on their own knowledge when calculating the CHA_2DS_2-VA score.

Conclusions: This survey revealed an overall high level of adherence to guideline-recommended scoring for the 'C', 'H', 'V', and 'D' components of the CHA_2DS_2-VA score. However, the greatest variability among respondents was observed in the interpretation of

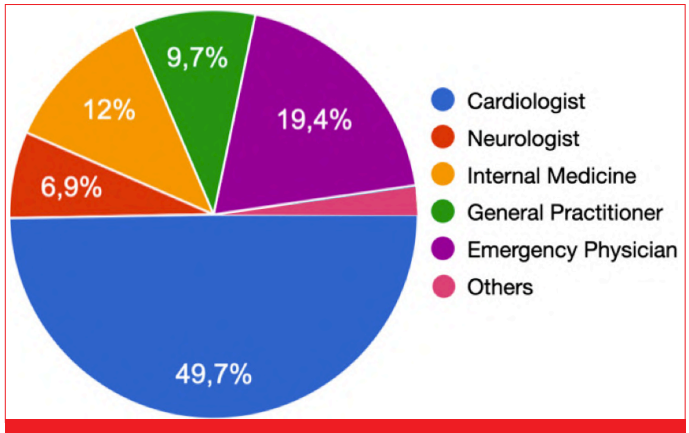

Figure 1. Characteristics of survey respondents.

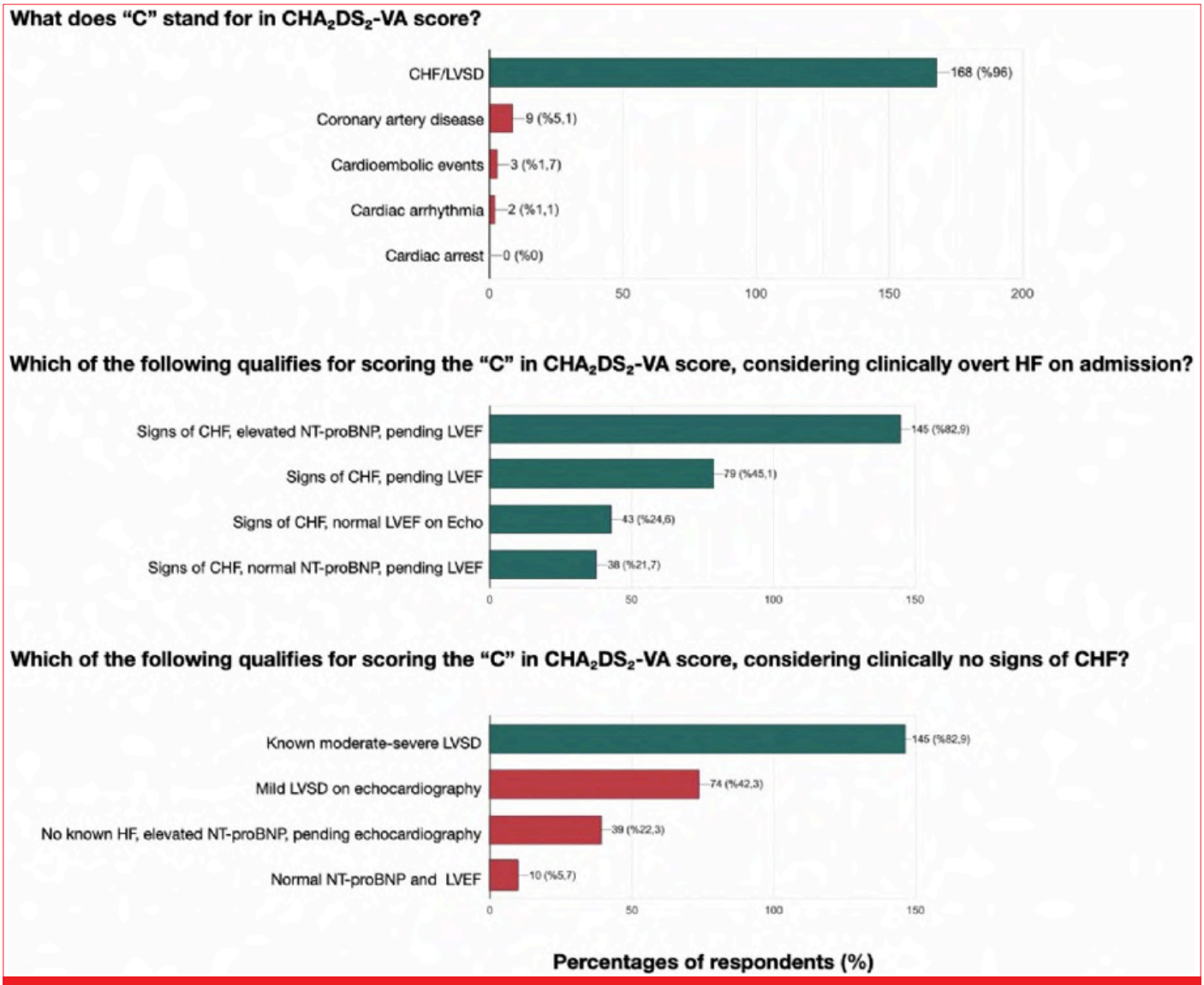

Figure 2. Scoring of the 'C' criterion in CHA2DS2-VA score. The bars in turquoise are correct answers, while those in red are incorrect.

the 'C' and 'H' components, indicating a need for further clarification. The resources used by clinicians to calculate the CHA_2DS_2-VA score varied across different healthcare professionals. This survey highlights the importance of clearly defining each component of the CHA_2DS±-VA score and ensuring access to guideline-based education for healthcare providers.

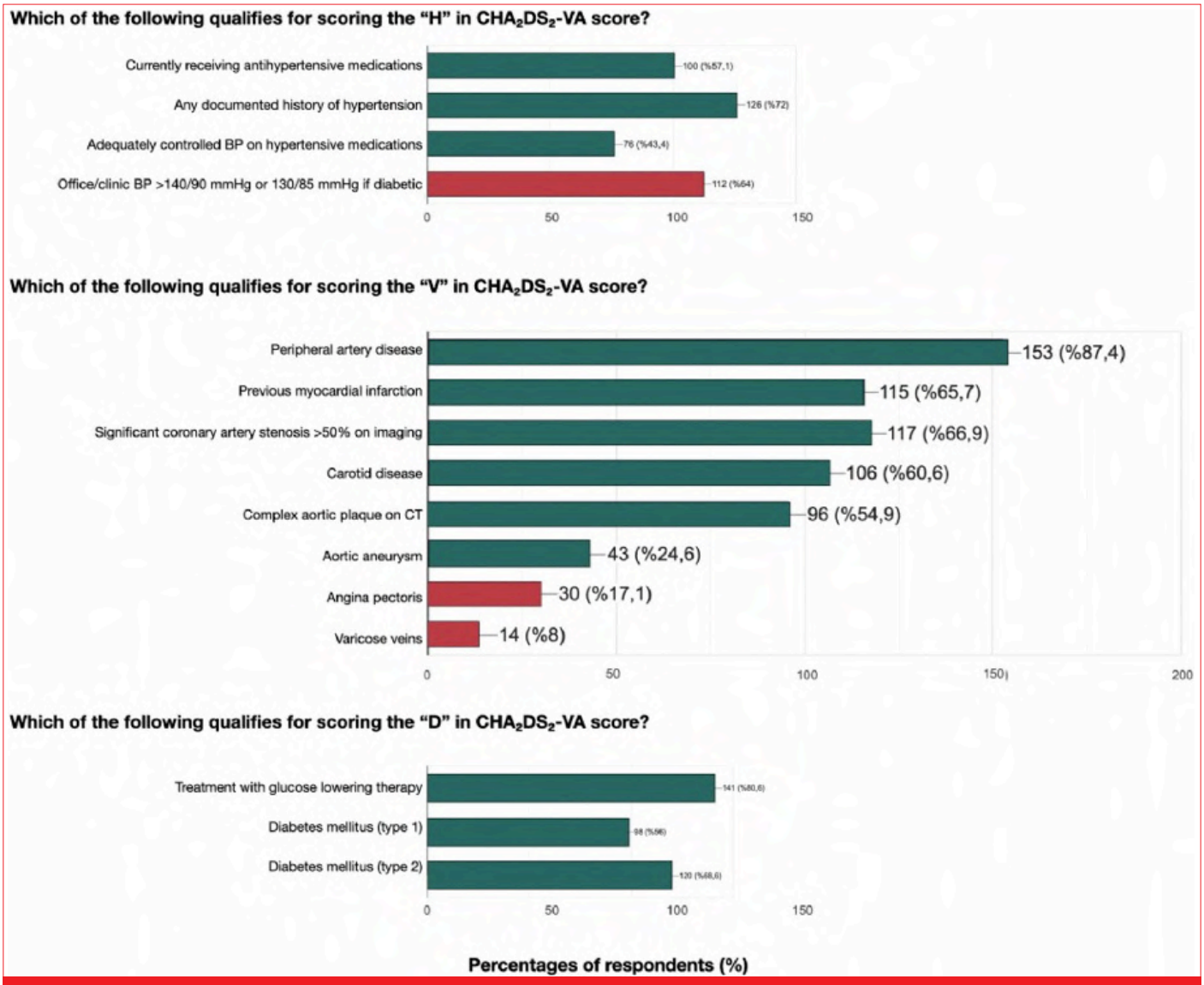

Figure 3. Scoring the 'H', 'V' and 'D' criteria in CHA_2DS_2-VA score. The bars in colour turquoise are correct answers, while those in red are incorrect.

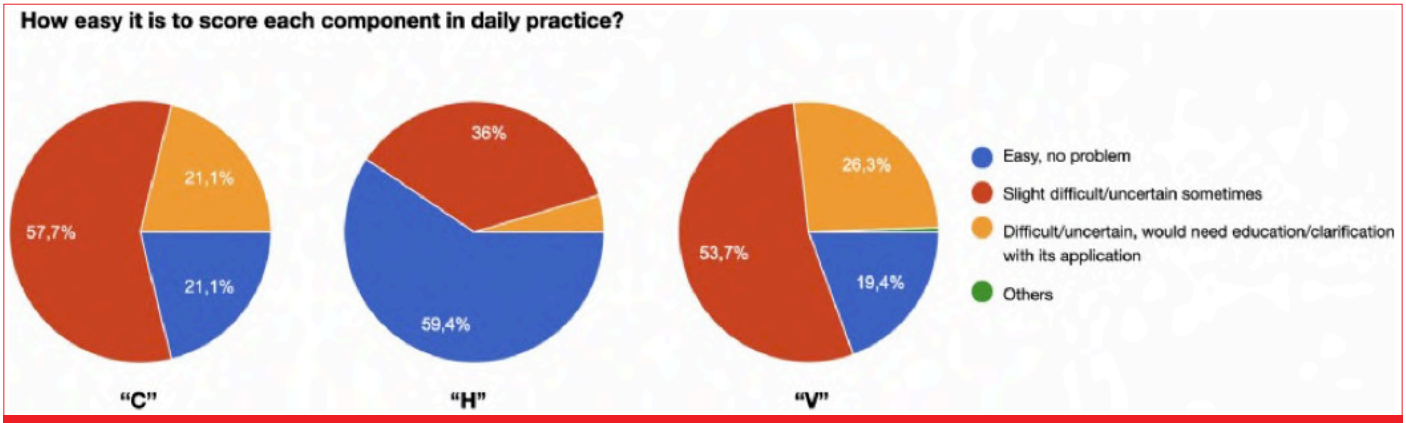

Figure 4. The ease of scoring the 'C', 'H', and 'V' in CHA_2DS_2-VA score.

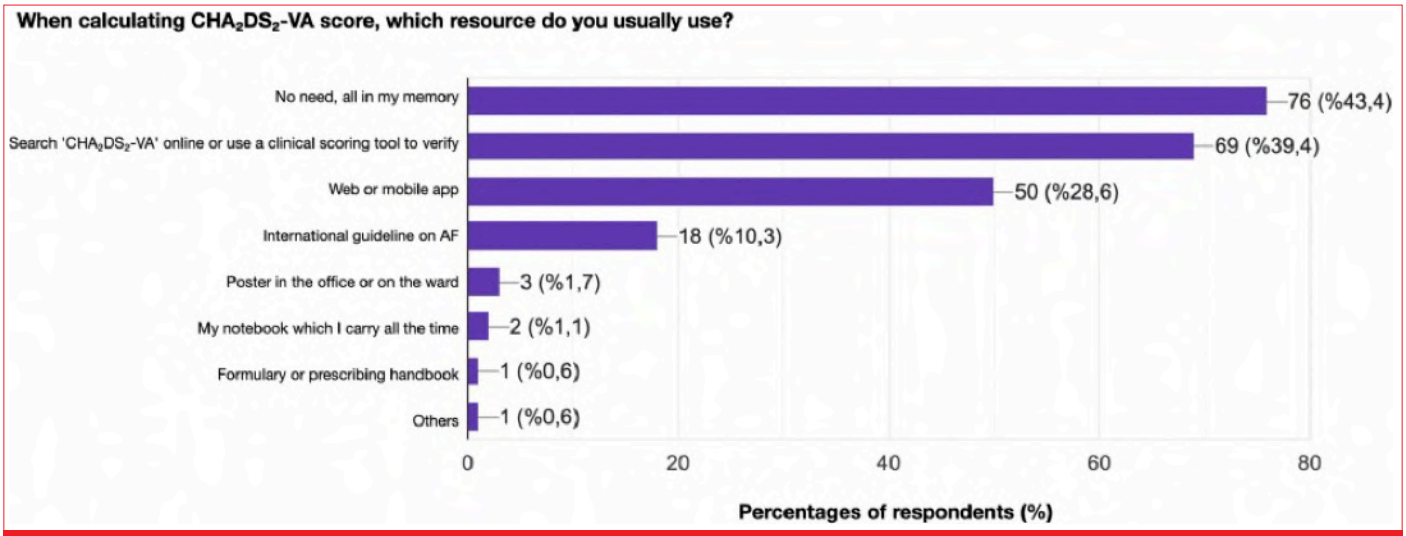

Figure 5. Referencing resources for CHA_2DS_2-VA score in daily practice.

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

Assessment of patients' awareness of anticoagulant use in atrial fibrillation: A cross-sectional survey

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Background and Aim: Atrial fibrillation (AF) increases thromboembolic risk; oral anticoagulants (OACs) mitigate this but require informed use. We assessed AF/OAC awareness among Turkish patients and explored predictors of knowledge.

Methods: Single-centre cross-sectional survey (March–August 2024) of consecutive adults with non-valvular AF on warfarin or direct OACs for ≥3 months. A structured questionnaire captured demographics, comorbidities, medication profile, adherence, and knowledge across six domains (arrhythmia name, indication, dosing schedule, side-effects, drug/food interactions, consequences of dose change). A composite knowledge score (0–6) summed correct items. Multivariable linear regression identified predictors (age, sex, and education). Ethics approval and consent were obtained.

Results: We enrolled 261 patients (median age 67 years; 50.2% women; Table 1). Hypertension and diabetes were present in 64.8% and 32.2%, respectively. DOACs comprised 72.4% of OACs (rivaroxaban 27.6%, apixaban 24.5%, edoxaban 17.2%, dabigatran 3.1%); warfarin 27.6% (Figure 1). Thirty-six percent had AF >6 years, yet only 16.5% could name their arrhythmia. While 92% knew they were taking an anticoagulant, 24% misunderstood or did not know the indication. Side-effect knowledge was limited (68% could not name any). Awareness of interactions was poorer: 88% knew no drug–drug interactions and 71% no food interactions. Most knew dosing schedules (92.3%) and would not double a missed dose (99.6%). Self-reported adherence was high (94% "always," 4% "often"); timing with meals correlated with adherence (χ^2=17.0, p<0.001). Over one-third (34%) recalled no initial education; 86.2% cited physicians as their sole information source. Mean knowledge score was 2.8 ± 1.3 (Cronbach's α=0.50). In adjusted models, higher education (+0.3 points per level, p<0.001) and female sex (+0.6, p<0.001) predicted greater knowledge, whereas age was inversely related (–0.02 per year, p=0.01). Warfarin users better recognized dietary issues, yet misconceptions persisted; some DOAC users imposed unnecessary dietary restrictions.

Conclusions: Patients exhibited excellent self-reported adherence but striking gaps in understanding AF/OACs—especially side-effects and interactions. Knowledge tracked with education, age, and sex, underscoring the need for targeted, literacy-sensitive counselling, teach-back methods, and written/app-based aids, particularly for older, less-educated, and male patients. Limitations include a single-centre design and reliance on self-report. Structured education at initiation and reinforcement at follow-up may enhance safety and outcomes in AF anticoagulation.

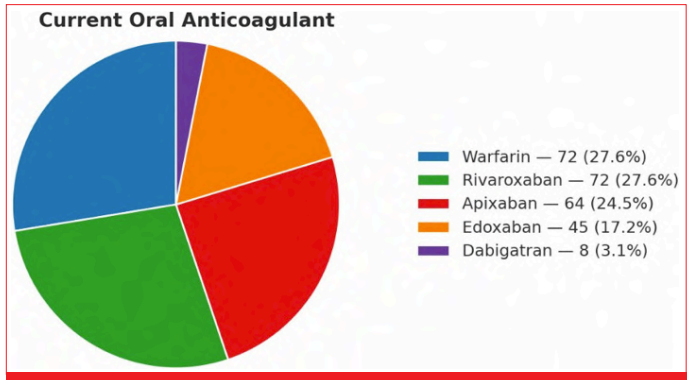

Figure 1. Type of oral anticoagulation.

Table 1. Baseline characteristics

Characteristic	Total n=261 (%)
Age (years; median)	67 (min-max: 30-92)
Female	131 (50.2%)
Education Level	
No formal education	16 (6.1%)
Primary School	120 (46.0%)
Middle School	24 (9.2%)
High School	45 (17.2%)
University	54 (20.7%)
Master's Degree	2 (0.8%)
Occupation	
Unemployed	58 (22.2%)
Retired	181 (69.3%)
Other	22 (8.4%)
Residence	
Urban	234 (89.7%)
Rural	27 (10.3%)
Smoking Status	
Current smoker	28 (10.7%)
Never smoked	170 (65.1%)
Former smoker	63 (24.1%)
Alcohol Use	
Current user	32 (12.3%)
Never used	200 (76.6%)
Former user	29 (11.1%)

Table 1. Clinical, laboratory and medical treatment findings in patients before and after AF ablation

	Before AF ablation n=109	After AF ablation n=109	p
Tobacco use, n (%)	46 (42%)[a]	44 (40%)[a]	0.481
Systolic blood pressure (mmHg)	117 ± 8.2	118 ± 8.8	0.293
Diastolic blood pressure (mmHg)	73 ± 10	72 ± 9.7	0.264
Heart rate (beats/min)	94 ± 17	74 ± 8.9	<0.001
White blood cell (uL)	9.7 ± 2.1	9.4 ± 1.4	0.055
Hemoglobin (gr/dL)	14.2 ± 1.31	13.9 ± 1.49	0.060
Blood urea nitrogen (mg/dL)	32.4 ± 7.9	33.4 ± 6.8	0.051
Creatinine (mg/dL)	0.83 ± 0.20	0.82 ± 0.18	0.781
Uric acid (mg/dL)	4.89 ± 0.54	4.80 ± 0.56	0.023
Total cholesterol (mg/dL)	149 ± 35	146 ± 39	0.004
Low-density lipoprotein cholesterol (mg/dL)	97 ± 26	93 ± 30	0.001
High-density lipoprotein cholesterol (mg/dL)	45 ± 6.8	46 ± 7.5	0.040
Triglycerides (mg/dL)	156 ± 84	140 ± 62	0.001
Ejection fraction (%)	63 ± 3.8	62 ± 3.9	0.082
Left atrial diameter (mm)	33.5 ± 3.6	32.9 ± 3.8	<0.001
Phosphodiesterase 5 inhibitors, n (%)	32 (29%)[a]	5 (4.6%)[b]	<0.05
Propafenone, n (%)	24 (22%)[a]	14 (13%)[b]	<0.05
Flecainide, n (%)	12 (11%)[a]	2 (1.8%)[b]	<0.05
Amiodarone, n (%)	18 (17%)[a]	10 (9.2%)[b]	<0.05
Calcium channel blockers, n (%)	9 (8%)[a]	8 (7.3%)[a]	>0.05
Beta blockers, n (%)	56 (51%)[a]	37 (3.4%)[b]	<0.05

OP-048 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Improvement in erectile dysfunction with ablation therapy in patients with paroxysmal atrial fibrillation

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Background and Aim: Studies have clearly shown that atrial fibrillation (AF) is associated with erectile dysfunction (ED). However, there is no data in the literature on the effect of AF ablation therapy on ED in these patients. The aim of this study was to investigate the effect of cryoballoon ablation on ED in patients with paroxysmal AF.

Methods: In this cross-sectional study, 1420 patients who underwent AF for paroxysmal AF in our clinic were screened. We included 109 male patients (age 37.7 ± 5.2 years) with paroxysmal AF, CHA2DS(2)–VA score 0 and no recurrence at 1-year follow-up. International Index of Erectile Function (IIEF) and Sexual Health Inventory for Men (SHIM) scores were performed in addition to clinical, laboratory and medical treatments before and after AF ablation. Patients with SHIM score ≤21 were defined as having ED, while those with SHIM score >21 were defined as not having ED.

Results: It was found that 21% of AF patients had ED before ablation and 1 year after AF ablation treatment, ED decreased significantly, and its frequency decreased to 8.3% (p<0.05). Heart rate, serum uric acid, total cholesterol, low-density lipoprotein cholesterol, triglycerides and left atrial diastolic diameter decreased significantly after AF ablation (p<0.05 for each). Serum high-density lipoprotein cholesterol level was found to be higher after AF ablation (p<0.05). In addition, the frequency of taking phosphodiesterase-5 inhibitors was significantly lower after AF ablation. SHIM, IIEF average, erectile function, sexual desire and intercourse satisfaction scores were significantly higher after AF ablation (p<0.05 for each).

Conclusions: Our study demonstrated that the presence of ED, which is common in AF patients, was significantly reduced with ablation therapy. We conclude that this may be considered as an additional clinical benefit to the existing symptomatic benefits of AF ablation.

Table 2. SHIM and IIEF scores in subject before and after AF ablation

	Before AF ablation n=109	After AF ablation n=109	p
SHIM score	22 ± 1.68	23 ± 1.37	<0.001
Erectile dysfunction, n (%)	23 (21%)[a]	9 (8.3%)[b]	<0.05
IIEF average score	41 ± 3.6	42 ± 3.1	<0.001
Erectile function	16 ± 1.49	17 ± 1.42	<0.001
Orgasmic function	4.32 ± 0.78	4.39 ± 0.68	0.109
Sexual desire	5.71 ± 0.93	6.17 ± 0.63	<0.001
Intercourse satisfaction	7.61 ± 0.86	7.87 ± 0.80	<0.001
Overall satisfaction	6.22 ± 0.77	6.24 ± 0.77	1.000

OP-049 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Contemporary outcomes of periaortic ventricular tachycardia ablation in non-ischemic cardiomyopathies

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Background and Aim: Periaortic ventricular tachycardias (VTs) represent challenging substrate in non-ischemic cardiomyopathies (NICM). Tissue thickness, proximity to vascular structures and to the conduction are important considerations when ablating this area. Furthermore, limited access to the intramural substrate makes complete VT circuit characterization difficult in many circumstances. Recently developed techniques such as intravascular mapping, ethanol ablation and anatomical ablation may improve outcomes in periaortic VTs. In this study we sought to investigate outcomes of periaortic VT ablation and compare them to the other NICM substrate.

Methods: Patients undergoing NICM VT ablation between 1st February 2022 and 1st April 2025 with one of the authors (EB, OA, TA) being primary operators were screened for the inclusion to the study. Among these patients those with periaortic substrate were included to the study. Clinical and procedural outcomes were analyzed. Recurrence rate of periaortic VTs was compared to non-periaortic substrate in NICM.

Results: 25 patients (30.4%) had periaortic substrate among 82 patients who underwent VT ablation for NICM. Patients were predominantly male (80%) and the median ejection fraction was 40% (IQR: 45-25%) (Table 1). 15 patients (60%) presented with a VT storm. Median number of VTs induced per procedure was 2 (IQR: 3-1). Venous ethanol ablation was performed in 4 patients (16%). Non-inducibility was achieved in all but one patient, who developed cardiogenic shock and succumbed to progressive shock one week later (i.e. early death). There was one instance of cardiac tamponade necessitating surgical correction. During median follow-up of 14 months (IQR: 19-8.5) recurrence was observed in 8 patients (32%). When compared to other NICM VT patients, recurrence rate was similar (p=0.145). Kaplan-Meier survival graph is present at Figure 1.

Conclusions: Late recurrence remains to be a challenge for periaortic VT substrate, yet recurrence rates are not different to other NICM substrates.

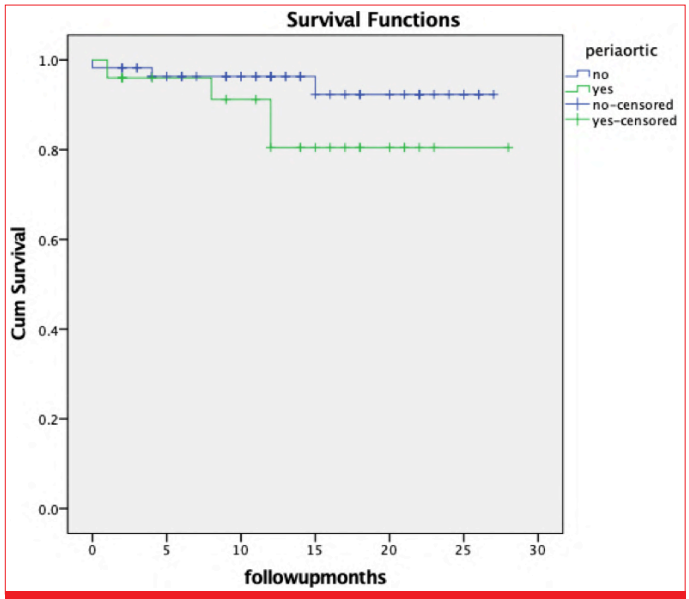

Figure 1. Kaplan-Meier survival graph shows similar recurrence rate among periaortic and non-periaortic NICM VT substrate (p=0.146 by log rank).

Table 1. Clinical characteristics

	Periaortic VT (n=25)
Age	58.5 (70.5-58.5)
Sex, male	20 (80)
EF	40 (45-25)
Diabetes Mellitus	9 (36)
AF	10 (40)
HT	15 (60)
VT storm	15 (60)
Procedure duration	140 (190-100)
Rf duration	28.2 (40-17.7)
Fluoro time	10.9 (20-4.3)
General anesthesia	13 (52)
Number of VTs induced, -n(IQR)	2 (3-1)
Non-inducibility	24 (96)
Early death	1 (4)
Recurrence	8 (32)
Death after discharge	3 (12)
Follow-up duration	14 (19-8.5)

OP-050 [Interventional Cardiology / Valvular and Structural Heart Disease]

Early term results for transcatheter tricuspid interventions

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Background and Aim: Severe symptomatic tricuspid regurgitation (TR) with right heart failure is associated with significant morbidity and mortality. There are three types of TR, each with different etiologies: primary, secondary, and cardiac electric implantable device-related (CEID). Medical therapy is alone unsuccessful, and surgical correction has not been performed due to perioperative risk. Therefore, transcatheter tricuspid valve interventions started to improve severe TR. Currently, there are two main strategies, including transcatheter tricuspid valve repair and transcatheter tricuspid valve replacement. We evaluate totally 7 patients with severe symptomatic tricuspid regurgitation which underwent transcatheter tricuspid valve repair with the TriClip device (Abbott Vascular) and transcatheter tricuspid valve replacement with CAVI - the TricValve system (P&F Products Features Vertrieb, Vienna, Austria).

Methods: Totally 7 patients with severe symptomatic TR undergoing 4 cases with heterotopic tricuspid valve implantation (CAVI) and 3 cases with transcatheter tricuspid valve repair with TriClip between January 2004 and June 2025 were included. Mean age was 69 ± 12.7 years. Transthoracic echocardiography showed torrential TR, Tricuspid Annular Plane Systolic Excursion (TAPSE) of 13 mm, systolic pulmonary artery pressure of 45 mmHg, and left ventricular ejection fraction (LVEF) of 40%. Tricuspid annular diameters were >40 mm. In TriClip patients, the primary end point was mortality, heart failure hospitalizations, and quality of life improvement ≥15 points assessed using the Kansas City Cardiomyopathy Questionnaire (KCCQ). In CAVI patients, the TRI-SCORE was used to predict in-hospital mortality. Her TRI-SCORE was calculated as 14. Computed tomography (CT) was performed to visualize the vena cava, and device sizes were determined. The patient underwent CAVI using the TricValve system (P&F Products Features Vertrieb, Vienna, Austria) under general anesthesia.

Results: Four patients with CAVI procedure discharged after 4 days of hospitalization with significant improvement in functional capacity [from New York Heart Association (NYHA) 3-4 to NYHA 2]. Peripheral edema regressed, and the need for diuretics decreased (furosemide from 120 mg to 20 mg/day). Jugular venous distention disappeared. Three patients with TriClip discharged after 3 days of hospitalization with significant improvement in functional capacity [from New York Heart Association 3-4 to 2). At 6 months following time, the functional capacity stabilized as NYHA class 2. No develop death.

Conclusions: Transcatheter tricuspid valve interventions are effective therapy in the patient with high-risk surgery conditions.

OP-051 [Interventional Cardiology / Valvular and Structural Heart Disease]

Postoperative right ventricular-pulmonary arterial uncoupling predicts long-term mortality after transcatheter aortic valve replacement

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Background and Aim: The tricuspid annular plane systolic excursion (TAPSE) to pulmonary artery systolic pressure (PASP) ratio is an established indicator of right ventricular–pulmonary arterial (RV–PA) coupling. Although the prognostic significance of the preoperative TAPSE/PASP ratio in patients undergoing transcatheter aortic valve replacement (TAVR) has been previously reported, the impact of postoperative echocardiographic TAPSE/PASP measurements needs to be determined. This study aims to evaluate the prognostic value of the TAPSE/PASP ratio in predicting long-term mortality in patients who have undergone TAVR.

Methods: This single-center retrospective study included 786 patients who underwent TAVR between June 1, 2020, and March 1, 2025. The primary outcome of the study was all-cause long-term mortality. Receiver operating characteristic (ROC) curve analysis was used to determine the optimal cut-off value of the TAPSE/PASP ratio for predicting long-term mortality. Kaplan–Meier survival analysis was performed to compare survival probabilities between two groups stratified by the identified TAPSE/PASP cut-off value. Univariate Cox regression analysis was conducted to assess potential predictors, including baseline clinical characteristics, comorbidities, postoperative echocardiographic parameters, laboratory values at admission, and preoperative Agatston score. Variables found to be statistically significant in univariate analysis were subsequently included in the multivariate Cox regression model. A two-tailed p-value <0.05 was considered statistically significant.

Results: Follow-up duration for overall population was 509 (283-847) days. ROC curve identified a TAPSE/PASP ratio cut-off value of 0.052 cm/mmHg (AUC: 0.626) (Figure 1). Low TAPSE/PASP group (n=278) experienced a higher mortality (35 (12.6%) vs. 26 (5.1%), p<0.001) than high TAPSE/PASP group (n=508). Kaplan–Meier survival analysis revealed significantly worse survival in the group with a low TAPSE/PASP ratio (Figure 2, log-rank test p<0.001). In univariate Cox regression, the following variables emerged as significant predictors of long-term mortality: older age, pre-existing chronic obstructive pulmonary disease (COPD), atrial fibrillation, higher creatinine, higher aspartate aminotransferase, lower albumin, elevated C-reactive protein, lower postoperative left ventricular ejection fraction (LVEF), lower TAPSE/PASP ratio, and lower maximal aortic gradient (Table 1). These predictors, along with sex and hemoglobin, were subsequently included in the multivariate model. In the multivariate Cox regression, the following were independently associated with long-term mortality: age (HR: 1.04, p=0.04), pre-existing COPD (HR: 2.26, p=0.01), and postoperative TAPSE/PASP ratio (per 0.01 cm/mmHg increase; HR: 0.85, p=0.03) (Table 2).

Conclusions: A lower postoperative TAPSE/PASP ratio, as an indicator of impaired RV-PA coupling, is associated with higher long-term all-cause mortality in patients who have undergone TAVR.

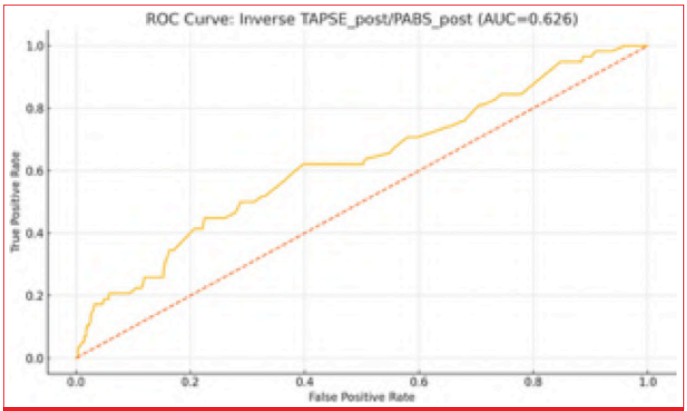

Figure 1. ROC curve analysis for the prediction of long-term mortality (AUC=0.626).

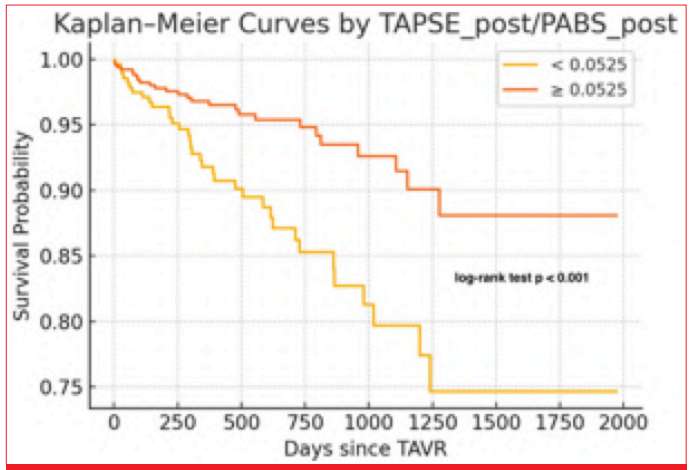

Figure 2. Kaplan–Meier survival analysis to compare the survival probabilities between two groups stratified by the identified TAPSE/PASP cut-off value.

Table 1. Univariate Cox regression analysis for the prediction of long-term mortality

Variables	Hazard Ratio	95% Confidence Interval	p value
Baseline Clinical Characteristics			
Age (per 1 year increase)	1.044	1.002-1.087	0.037
Sex (male)	0.936	0.566-1.548	0.797
Hypertension	0.861	0.466-1.589	0.632
Coronary artery disease	1.573	0.932-2.653	0.089
Diabetes mellitus	1.260	0.751-2.117	0.380
Chronic obstructive pulmonary disease	2.024	1.114-3.680	0.020
History of cerebrovascular accident	1.629	0.739-3.588	0.225
Atrial fibrillation	2.034	1.229-3.366	0.005
Laboratory Parameters at Admission			
Creatinine (per 1 mg/dL increase)	1.274	1.023-1.588	0.030
Aspartate aminotransferase (per 1 IU/L increase)	1.003	1.000-1.006	0.047
Alanine aminotransferase (per 1 IU/L increase)	1.002	0.997-1.006	0.385
Albumin (per 1 g/dL increase)	0.601	0.401-0.902	0.014
C-reactive protein (per 1 mg/L increase)	1.008	1.003-1.014	0.001
Hemoglobin (per 1 g/dL increase)	0.891	0.781-1.016	0.087
Post-operative Echocardiographic Parameters			
Left ventricular ejection fraction (per 1% increase)	0.969	0.950-0.988	0.002
TAPSE/PASP ratio (per 0.01 cm/mmHg increase)	0.808	0.708-0.922	0.002
Maximal aortic gradient (per 1 mmHg increase)	0.954	0.913-0.997	0.036
Moderate to severe paravalvular leak	1.658	0.815-3.374	0.163
Pre-operative Computed Tomography			
Agatston Score	0.745	0.469-1.182	0.211

Table 2. Multivariate Cox regression analysis for the prediction of long-term mortality

Variables	Hazard Ratio	95% Confidence Interval	p-value
Age (per 1 year increase)	1.044	1.001-1.089	0.044
Sex (male)	1.001	0.564-1.776	0.997
Chronic obstructive pulmonary disease	2.261	1.192-4.290	0.012
Atrial fibrillation	1.311	0.752-2.287	0.339
Hemoglobin (per 1 g/dL increase)	0.947	0.817-1.098	0.471
Creatinine (per 1 mg/dL increase)	1.218	0.920-1.614	0.167
Aspartate aminotransferase (per 1 IU/L increase)	1.001	0.997-1.006	0.459
Albumin (per 1 g/dL increase)	0.884	0.447-1.747	0.723
C-reactive protein (per 1 mg/L increase)	1.003	0.995-1.010	0.473
Post-op left ventricular ejection fraction (per 1% increase)	0.983	0.961-1.007	0.162
Post-op TAPSE/PASP ratio (per 0.01 cm/mmHg increase)	0.856	0.743-0.988	0.033
Post-op maximal aortic gradient (per 1 mmHg increase)	0.962	0.917-1.008	0.108

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

Comparison of single versus double suture-based vascular closure devices in femoral access for transcatheter aortic valve ımplantation: Procedural success and complication rates

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Background and Aim: Transcatheter aortic valve implantation (TAVI) has revolutionized the management of severe aortic stenosis, providing a less invasive alternative to surgical aortic valve replacement, especially in high- and intermediate-risk patients. With the expansion of TAVI to lower-risk populations, procedural safety and vascular access optimization have become increasingly important. Vascular closure is a critical step in transfemoral TAVI, traditionally performed via surgical cut-down but now predominantly achieved using percutaneous closure devices. Suture-based closure devices such as ProGlide have gained popularity, but there remains debate regarding the use of a single versus double device strategy. This study aims to compare the procedural success and complication rates of single (SCD) and double (DCD) suture-based closure devices in TAVI patients.

Methods: This prospective observational study included 262 consecutive patients undergoing transfemoral TAVI between 2020 and 2025. Exclusion criteria included puncture site calcification, high femoral bifurcation, anterior wall puncture outside the target site, and surgical cut-down cases. Patients were divided into two groups: single closure device (SCD, n=125) and double closure device (DCD, n=137). The SCD was deployed at the 12 or 1 o'clock position, while DCD was deployed at the 10 and 2 o'clock positions. Procedural failure was defined as the need for a second device in the SCD group, additional device in the DCD group, or the requirement for stent graft, balloon angioplasty, or surgical repair. Demographic,

Table 1. Baseline characteristics of single or double closure devices groups (SCD vs. DCD)

Variable	SCD (%)	DCD (%)	p value
Female	56.3	51.7	0.32
Hypertension	80.9	86.0	0.12
Diabetes	40.9	38.7	0.77
Peripheral arteriopathy	5.5	5.3	0.82

Table 2. Procedural results and complications (SCD vs. DCD)

Variable	SCD (%)	DCD (%)	p value
Device Failure	8.1	14.2	0.002
Femoral Stenting	2.4	2.2	0.88
Femoral Balloon Angioplasty	6.3	7.4	0.09
Femoral Acute Thrombosis/ Stenosis	3.1	5.4	0.012
Minor Vascular Complications	9.2	8.1	0.08
Major Vascular Complications	3.2	3.9	0.66
Femoral Artery Dissection	4.1	4.3	0.86
Pseudoaneurysm	2.2	1.5	0.52
Bleeding VARC Type 1	3.8	5.4	0.002
Bleeding VARC Type 2	2.6	2.2	0.75
Bleeding VARC Type 3	2.6	2.8	0.90
Femoral Hematoma	9.2	8.1	0.08
Retroperitoneal Bleeding	0	0	NS
Surgical Closure	3.1	2.4	0.26
Cardiovascular Death (in-hospital)	2.5	3.7	0.12

laboratory, echocardiographic, vascular imaging, procedural, and post-procedural data were collected and analyzed using appropriate statistical methods.

Results: SCD group had significantly higher procedural success compared to DCD (91.9% vs. 85.8%, p<0.001) and lower post-closure stenosis/occlusion rates (3.1% vs. 5.7%, p=0.012). Major vascular complications, contrast-induced nephropathy, and in-hospital mortality rates were similar between groups.

Conclusions: Our findings suggest that single suture-based closure devices (SCD) may provide higher procedural success rates and lower post-closure stenosis/occlusion rates compared to double devices (DCD) in transfemoral TAVI. This may be related to less arterial wall manipulation and reduced suture tension in the single device approach. Both groups demonstrated comparable rates of major vascular complications, bleeding, and in-hospital mortality. Previous studies have shown mixed results regarding optimal closure strategy, and our data support the selective use of SCD in anatomically favorable patients. Limitations include the single-center design and lack of long-term follow-up data. Future randomized trials are warranted to confirm these results.

OP-053 [Heart Valve Diseases]

Retrospective analysis of therapeutic modalities in prosthetic heart valve thrombosis: A 15-year single-center experience

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Background and Aim: Prosthetic valve thrombosis remains a major cause of morbidity and mortality due to valve thrombogenicity and inadequate anticoagulation. An optimal therapeutic strategy—medical versus surgical—continues to be debated. This study aimed to evaluate patient characteristics, treatment strategies, and clinical outcomes in individuals diagnosed with prosthetic valve thrombosis over a fifteen-year period at a single tertiary care center.

Methods: Adult patients (>18 years) with confirmed prosthetic valve thrombosis (either mechanical or bioprosthetic) were retrospectively reviewed. Diagnosis was established through echocardiographic studies (transthoracic or transesophageal) and clinical findings. Treatment consisted of intravenous heparin alone, low-dose fibrinolytic therapy (tissue plasminogen activator [t-PA]), or redo surgical intervention. Endpoints included treatment success, mortality, and overall complications. A multivariate logistic regression model was used to assess predictors of mortality, including New York Heart Association (NYHA) Functional Classification.

Results: A total of 76 patients were included, the majority of whom had mechanical prostheses. In 54 cases, the thrombosed valve was situated in the mitral position, in 20 cases in the aortic position, and in 2 cases in the tricuspid position. Of the 37 patients treated exclusively with heparin, the success rate was 67.6%, with a 24.3% mortality and a 40.5% overall complication rate. In the 27 patients receiving t-PA infusion, the success rate reached 59.3%, with a 7.4% mortality and a 37% complication rate. Surgical intervention was performed in 20 patients, yielding a 50% success rate, 50% mortality, and 65% complications. Across the entire cohort, patients in NYHA class III–IV demonstrated a significantly higher mortality risk (p<0.001) compared with those in NYHA class I–II. Moreover, multivariate logistic regression indicated that NYHA III–IV conferred a more than tenfold increase in mortality risk relative to lower NYHA classes.

Conclusions: In this study, low-dose t-PA therapy demonstrated reduced mortality compared with surgery, albeit with comparable success rates to heparin. Higher NYHA class significantly impacted survival irrespective of treatment modality. Timely identification and individualized treatment, particularly in NYHA class III–IV patients, appear critical to optimising outcomes in prosthetic valve thrombosis.

OP-054 [Heart Valve Diseases]

Prognostic value of CHADS2VASC, PRECISEDAPT and ARC-HBR scores in infective endocarditis

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Background and Aim: Infective endocarditis (IE) is a severe infectious cardiac disease associated with high mortality and morbidity. Despite advances in diagnosis and treatment, the mortality rate remains between 15% and 30% and complications such as systemic embolism, stroke, and heart failure are common. The heterogeneous nature of patients, comorbidities, and variable clinical courses make risk stratification and prognosis prediction challenging. Therefore, easily applicable and reliable risk models are needed to identify high-risk patients early. CHADS2VASC, PRECISEDAPT, and ARC-HBR scores are widely used in various cardiovascular conditions to predict thromboembolic events, bleeding risk, or long-term prognosis. However, limited evidence exists regarding their prognostic value in infective endocarditis. This study aimed to evaluate the prognostic value of CHADS2VASC, PRECISEDAPT, and ARC-HBR scores in patients with IE and to propose a combined risk model based on these scores.

Methods: This retrospective single-center study included 116 patients diagnosed with infective endocarditis between 2022 and 2025. The diagnosis was based on the 2023 ESC Guidelines for the Management of Infective Endocarditis. $CHADS_2$VASC, PRECISEDAPT, and ARC-HBR scores were calculated for each patient. Demographic, laboratory, and echocardiographic data, as well as surgical indications and outcomes, were recorded. The primary endpoint was in hospital mortality and secondary endpoints included stroke, systemic embolism and major bleeding. Statistical analyses included descriptive statistics, ROC curve analysis, Youden index cut-off determination, and multivariate logistic regression to identify independent predictors.

Results: The mean $CHADS_2$-VASc and PRECISE-DAPT scores were 1.97 ± 1.2 and 31.8 ± 8.6, respectively. ARC-HBR major

Table 1. Patient characteristics

Variable	Value
Number of Patients	116
Mean CHADS2VASC	1.97
Mean PRECISEDAPT	31.8
ARC-HBR Major Criteria (%)	78.4%
In-hospital Mortality (%)	14.2%

Table 2. Logistic regression analysis

Variable	P-value	Odds Ratio	95% CI (Lower)	95% CI (Upper)
const	0.001	0.016	0.001	0.182
CHADS2VASC	0.353	0.820	0.540	1.246
PRECİSEDAPT	0.021	1.062	1.009	1.118
ARC-HBR	0.578	1.889	0.201	17.793

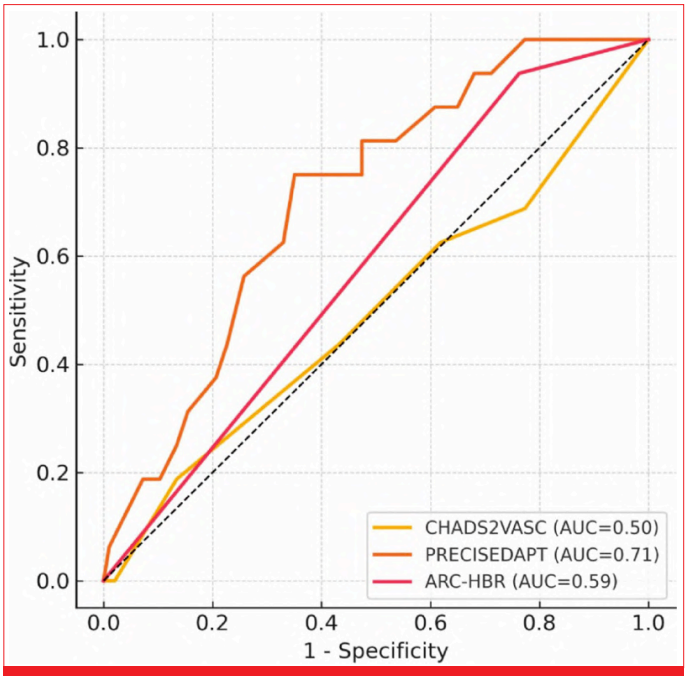

Figure 1. ROC curves for mortality prediction.

criteria were present in 78.4% of patients, reflecting a high baseline bleeding risk in this population. The overall in-hospital mortality rate was 14.2%. ROC curve analysis demonstrated that the PRECISE-DAPT score had the highest predictive value for in-hospital mortality (AUC=0.71), outperforming $CHADS_2$-VASc and ARC-HBR scores. In multivariate logistic regression analysis, the PRECISE-DAPT score remained the only independent predictor of mortality (OR=1.06; 95% CI: 1.01–1.12; p=0.021).

Conclusions: This study shows that $CHADS_2$-VASc, PRECISE-DAPT, and ARC-HBR scores are linked to prognosis in infective endocarditis, with PRECISE-DAPT being the strongest independent predictor of in-hospital mortality. Although $CHADS_2$-VASc and ARC-HBR were associated with mortality in univariate analyses, they were not independent predictors in multivariate models. These findings suggest that these simple, accessible scores may aid early risk stratification and guide management of IE patients in clinical practice. Larger prospective multicenter studies are needed to confirm these results and to assess whether combining these scores could further improve prognostic accuracy and optimize patient outcomes.

OP-055 [Interventional Cardiology / Valvular and Structural Heart Disease]

Transcatheter mitral valve repair using the edge-to-edge MitraClip

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Background and Aim: The MitraClip system is a device for percutaneous edge-to-edge reconstruction of the mitral valve in patients with severe mitral regurgitation (MR). Studies have emphasized the therapeutic benefit of the MitraClip system in the patients with high risk for mitral valve surgery, suffering from either degenerative or functional mitral regurgitation. The data of the EVEREST (Endovascular Valve Edge-to-Edge Repair Study) trials demonstrated that the MitraClip procedure is feasible and safe. Procedural success has been shown to increase with operator experience. We evaluated the mid-term clinical results and the impact of post-repair mitral valves function and outcome composite endpoint after MitraClip implantation in surgical high-risk patients.

Methods: A total of 46 consecutive patients with severe MR were included (from 2016 to 2025 years). The age range were from 44 years to 89 years, (67%, men). These patients who underwent the MitraClip procedure with severe mitral regurgitation had high-risk surgery. Grade ≤1 mitral regurgitation after procedure was accepted as successful. We followed those patients in terms of mortality, cardiac re-hospitalization, re-intervention.

Results: There was no operative mortality. One patient died due to massive pulmonary embolism after 24-hours procedure. In hospital- mortality was %2. Forty of 46 patients had ≤1 grade residual MR after procedure and postoperative mitral valve gradient meant 2 ± 1.3 mmHg. Procedure success was 87%. In one patient, 3rd grade MR was developed due to chordal rupture, which underwent elective valve surgery. Five patients had 2nd grade MR. In follow one year, that 5 patients were exposed to progression from 2nd to 3rd. One of 5 patients underwent re-intervention (MitraClip). Four of 5 patients followed by medical therapy. Follow-up was 290 ± 173 days. Three patients died at following time. The 3 patients re-hospitalized because of congestive heart failure symptoms.

Conclusions: The MitraClip procedure has low peri-procedural complication rates, significant reduction in MR in the severe mitral regurgitation with high-risk surgery.

OP-056 [Interventional Cardiology / Coronary]

Controlled-balloon crush or mini-crush stenting for coronary bifurcation disease: The multicenter EVOLUTE-CRUSH-CBC study

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Background and Aim: The mini-crush technique (MCT) is one of the leading double-stent techniques frequently performed by numerous interventional cardiologists for treating coronary bifurcation lesions (CBLs). It remains popular with several iterations having emerged in the past two decades. However, the two major challenges of MCT are follows: 1) rewiring of the side-branch (SB) after main vessel (MV) stent implantation, 2) advancing a non-compliant balloon into the SB without using a low-profile balloon. Recently, the controlled balloon-crush technique (CBC) has been introduced in the literature, and we have tested whether this novel approach may overcome these disadvantages of the MCT. This multicenter study aimed to prospectively evaluate the procedural and short-term outcomes of contemporary MCT and CBC in patients with true CBLs.

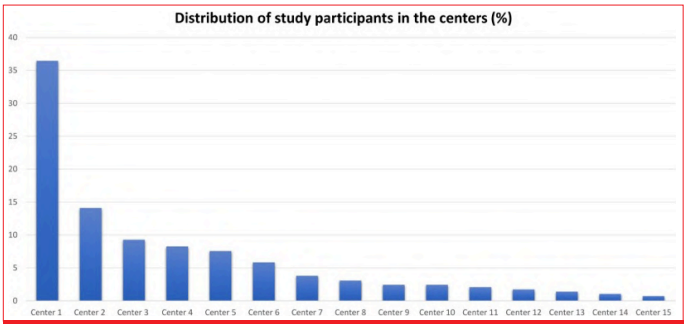

Figure 1. Distribution of study participants in the centers (%).

Table 1. Baseline characteristics of per study group

Variables	Mini-crush Group (n=174)	CBC Group (n=117)	P value
Age (years)	61.64±10.49	61.41±9.17	0.847
Gender, male, n (%)	148 (85.1)	95 (81.2)	0.384
Comorbidities, n(%)			
Hypertension	114 (65.5)	80 (68.4)	0.612
Diabetes Mellitus	70 (40.2)	51 (43.6)	0.569
Hyperlipidemia	114 (65.5)	79 (67.5)	0.723
Chronic kidney disease	37 (21.3)	27 (23.1)	0.714
Current Smoker	86 (49.4)	66 (56.4)	0.242
History of Stroke	4 (2.3)	2 (1.7)	1.000
Peripheral artery disease	13 (7.5)	9 (7.7)	1.000
Prior PCI	57 (32.8)	36 (30.8)	0.721
Prior MI	41 (23.6)	23 (19.7)	0.430
Heart Failure	32 (18.4)	24 (20.5)	0.653
Moderate-severe Valve Disease, n (%)	24 (13.8)	15 (12.8)	0.811
LV Ejection Fraction (%)	53.66±10.12	54.36±9.08	0.535
Laboratory measurements			
White blood cell count, (10^9/L)	8.93±2.55	8.81±2.20	0.689
Hemoglobin, (g/dL)	13.58±2.87	13.50±1.78	0.762
Platelet count, (10^9/L)	253.02±73.43	247.41±73.25	0.523
Creatinine, (mg/dL)	1.01±0.27	1.05±0.76	0.532
Total cholesterol, (mg/dL)	191.67±55.91	188.21±56.14	0.606
Clinical Presentation, n (%)			
CCS	82 (47.1)	57 (48.7)	0.790
NSTEMI	77 (44.3)	51 (43.6)	0.911
USAP	15 (8.6)	9 (7.7)	0.778
Medications Used, n (%)			
Acetylsalicylic acid	174 (100.0)	116 (99.1)	0.222
Clopidogrel	65 (37.4)	49 (41.9)	0.438
Ticagrelor	75 (43.1)	47 (40.2)	0.619
Prasugrel	33 (19.0)	21 (17.9)	0.827
Beta Blockers	156 (89.7)	105 (89.7)	0.981
CCB	40 (23.0)	29 (24.8)	0.724
ACEI/ARB	142 (81.6)	95 (81.2)	0.929
Statin	162 (93.1)	110 (94.0)	0.757
Diuretics	35 (20.1)	25 (21.4)	0.796
Insulin	23 (13.2)	19 (16.2)	0.472

ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker; CBC: Controlled balloon crush; CCB: Calcium channel blocker; CCS: Chronic coronary syndrome, LV: Left ventricle; MI: Myocardial infaction; NSTEMI: Non-ST segment elevation myocardial infarction; PCI: Percutaneous coronary intervention; USAP: Unstable angina pectoris.

Methods: The preliminary analysis comprised 291 patients [male: 243 (83.5%), mean age: 61.54 ± 9.95 years] who underwent bifurcation PCI between January 2025 and August 2025 were included in the study from 15 tertiary centers. The clinical co-primary endpoint was defined as the major adverse cardiovascular events (MACE), which include cardiac death, target vessel myocardial infarction (TVMI), or clinically driven target lesion revascularization (TLR). The co-primary technical endpoint was defined as the need for low-profile balloon use for the SB dilatation after MV stent implantation. Secondary endpoint was defined as the major adverse cardiac and cerebral events (MACCE), which include all-cause death, TVMI, clinically driven target vessel revascularization, stent thrombosis, or stroke.

Results: The initial revascularization strategy was MCT in 174 (59.8%) cases and CBC in 117 (40.2%) patients. Left main bifurcation localization (25.3 vs. 12%, p=0.005), bifurcation angle (61.88 ± 16.76 vs. 56.08 ± 16.470, p=0.004), MV lesion length (28.82 ± 12.37 vs. 25.67 ± 7.09 mm, p=0.006), and MV diameter (3.60 ± 0.51 vs. 3.42 ± 0.51 mm, p=0.004) were significantly

Table 2. Lesions characteristics per study group

Parameters	Mini-crush Group (n=174)	CBC Group (n=117)	P value
Multi-vessel disease, n(%)	106 (60.9)	71 (60.7)	0.968
SYNTAX score	21.95±8.43	21.82±6.54	0.881
SYNTAX score ≤22, n(%)	96 (55.2)	67 (57.3)	0.724
SYNTAX score 23-32, n(%)	51 (29.3)	35 (29.9)	0.912
SYNTAX score ≥33, n(%)	27 (15.5)	15 (12.8)	0.521
Locations of bifurcation lesions, n(%)			
LMCA	44 (25.3)	14 (12.0)	**0.005**
LAD-Diagonal	104 (59.8)	82 (70.1)	0.072
LCx-OM	21 (12.1)	19 (16.2)	0.311
PDA-PL	7 (4.0)	4 (3.4)	1.000
Type of Medina classification, n(%)			
1.0.1	7 (4.0)	6 (5.1)	0.774
0.1.1	31 (17.8)	19 (16.2)	0.727
1.1.1	134 (77.0)	91 (77.8)	0.878
Reference vessel diameter, mm			
MV	3.42±0.51	3.60±0.51	**0.004**
SB	2.78±0.30	2.78±0.28	0.888
SB reference vessel diameter ≥2.5 mm, n(%)	162 (93.1)	109 (93.2)	0.984
Complex bifurcation disease (DEFINITION) , n(%)	152 (87.4)	104 (88.9)	0.694
Assessment of complex bifurcation lesions, n(%)			
Moderate or severe calcification	65 (37.4)	39 (33.3)	0.483
SB stenosis 70% or 90%	167 (96.0)	109 (93.2)	0.287
MV reference diameter < 2.5mm	1 (0.6)	1 (0.9)	1.000
Multiple lesions	107 (61.5)	74 (63.2)	0.762
Bifurcation angle <45° or >70°	92 (52.9)	55 (47.0)	0.327
Thrombus identified by angiography	13 (7.5)	10 (8.5)	0.739
Bifurcation angle (°)	61.88±16.76	56.08±16.47	**0.004**
Lesion length, mm			
MV	28.82±12.37	25.67±7.09	**0.006**
SB	17.60±8.30	16.97±6.19	0.458
MV, n(%)			
TIMI flow grade <3	18 (10.3)	14 (12.0)	0.665
Chronic total occlusion	10 (5.7)	6 (5.1)	1.000
Thrombus-containing lesion	10 (5.7)	6 (5.1)	1.000
SB, n(%)			
TIMI flow grade <3	21 (12.1)	15 (12.8)	0.849
Chronic total occlusion	7 (4.0)	4 (3.4)	1.000
Thrombus-containing lesion	10 (5.7)	6 (5.1)	1.000

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

higher in the MCT group than in the CBC group. After MV stent implantation, usage of low-profile balloon (co-primary technical endpoint) (66.7 vs. 10.3%, p<0.001), requiring the anchor balloon technique (17.8 vs. 3.4%, p<0.001), total balloon number (6.98 ± 1.93 vs. 5.18 ± 0.96, p<0.001), procedure time (63.37 ± 23.77 vs. 50.66 ± 13.04 min, p<0.001), and contrast media volume (268.74 ± 100.96 vs. 234.76 ± 76.60 min, p=0.001) were notably higher in the MCT group compared to the CBC group. Whereas, 1:1 NC pass to SB-first attempt after MV stenting (33.3 vs. 89.7%, p<0.001) was significantly lower in the MCT group. At a mean follow-up of 5.27 ± 2.67 months, the incidence of co-primary clinical (MACE) (4 vs. 2.6%, p=0.745) and secondary endpoints (MACCE) (5.2 vs. 2.6%, p=0.373) was similar in both groups.

Conclusions: This non-randomized, prospective, multicenter study demonstrates that both techniques have comparable short-term clinical outcomes in patients with CBLs. Whereas, the CBC technique has better procedural and resource utilization data.

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

Parameters	Mini-crush Group (n=174)	CBC Group (n=117)	P value
Access site, n(%)			
Femoral	131 (75.3)	92 (78.6)	0.508
Radial	43 (24.7)	25 (21.4)	0.508
Pre-dilation, n(%)			
MV	165 (94.8)	112 (95.7)	0.725
SB	169 (97.1)	114 (97.4)	0.874
Utilization Scoring/cutting balloon, n(%)	21 (12.1)	5 (4.3)	**0.022**
Performed rotablation, n(%)	2 (1.1)	3 (2.6)	0.394
Performed IVL, n(%)	4 (2.3)	0 (0)	0.151
After MV stent implantation, SB opening			
1:1 NC pass to SB-first attempt, n(%)	58 (33.3)	105 (89.7)	**<0.001**
Requiring anchor balloon, n(%)	31 (17.8)	4 (3.4)	**<0.001**
Usage low profil balloon, n(%)	116 (66.7)	12 (10.3)	**<0.001**
1.0	17 (9.8)	4 (3.4)	0.062
1.25	26 (14.9)	1 (0.9)	**<0.001**
1.5	75 (43.1)	7 (6.0)	**<0.001**
2.0	19 (10.9)	4 (3.4)	**0.025**
Final kissing balloon inflation, n(%)	171 (98.3)	117 (100.0)	0.153
MV NC diameter, mm	3.23±0.36	3.20±0.37	0.421
SB NC diameter, mm	2.80±0.28	2.78±0.30	0.689
MV			
Stent number, n	1.25±0.47	1.23±0.48	0.699
Stent diameter, mm	3.31±0.42	3.35±0.43	0.423
Stent length, mm	32.05±9.46	28.70±8.02	**0.001**
SB			
Stent number, n	1.07±0.28	1.04±0.20	0.265
Stent diameter, mm	2.73±0.30	2.73±0.27	0.849
Stent length, mm	22.17±7.82	19.55±6.04	**0.001**
Proximal side-branch optimization, n **(%)**	162 (93.1)	117 (100)	**0.037**
Final POT, n (%)	171 (98.3)	117 (100)	0.153
Final POT balloon diameter, mm	4.06±0.53	4.01±0.47	0.365
Thrombus Aspiration, n (%)	3 (1.7)	0 (0)	0.276
Tirofiban use during PCI, n (%)	11 (6.3)	8 (6.8)	1.000
Resource utilization			
Guiding catheter, n (%)			
JL	15 (8.6)	10 (8.5)	0.982
EBU	150 (86.2)	98 (83.8)	0.564
AL	5 (2.9)	6 (5.1)	0.323
JR	4 (2.3)	3 (2.6)	1.000
Other	0 (0.0)	0 (0.0)	-
Guiding catheter number	1.03±0.18	1.03±0.18	0.989
Guidewire number	3.10±0.84	2.42±0.59	**<0.001**
Requiring stiff guidewire (CTO), n (%)	29 (16.7)	17 (14.5)	0.624
Balloon number	6.98±1.93	5.18±0.96	**<0.001**
Total stent number	2.34±.61	2.29±.58	0.449
Stent brands, n (%)			
Firehawk	115 (66.1)	97 (82.9)	**0.002**
Evermine	1 (0.6)	0 (0)	1.000
Promus	5 (2.9)	5 (4.3)	0.530
Xience	19 (10.9)	14 (12.0)	0.783
Onyx	24 (13.8)	2 (1.7)	**<0.001**
Intraprocedural complication, n(%)			
Abrupt occlusion			
MV	3 (1.7)	0 (0)	0.276
SB	2 (1.1)	1 (0.9)	1.000
TIMI<3			
MV	6 (3.4)	2 (1.7)	0.482
SB	4 (2.3)	2 (1.7)	1.000
Dissection			
MV	12 (6.9)	12 (10.3)	0.307
SB	9 (5.2)	2 (1.7)	0.209
Thrombus formation			
MV	3 (1.7)	1 (0.9)	0.651
SB	2 (1.1)	0 (0)	0.518
Coronary Perforation			
MV	0 (0)	1 (0.9)	0.402
SB	0 (0)	0 (0)	-
Procedure time, min	63.37±23.77	50.66±13.04	**<0.001**
Fluoroscopy time, min	26.32±8.29	20.99±6.60	**<0.001**
Contrast media volume (mL)	268.74±100.96	234.76±76.60	**0.001**
Angiographic success, n(%)			
MV	173 (99.4)	115 (98.3)	0.347
SB	174 (100.0)	116 (99.1)	0.222
In-hospital complications, n (%)			
Death	1 (0.6)	1 (0.9)	1.000
Major bleeding	2 (1.1)	3 (2.6)	0.394
Pseudoaneurysm	1 (0.6)	0 (0)	1.000
Fatal arrhythmias	2 (1.1)	0 (0)	0.518
Stent thrombosis	1 (0.6)	0 (0)	1.000
Spontaneous myocardial infarction	1 (0.6)	0 (0)	1.000
Contrast-induced AKI	12 (6.9)	4 (3.4)	0.295

AKI: Acute kidney injury; CBC: Controlled balloon crush; CTO: Chronic total occlusion; IVUS: Intravascular ultrasound; NC: Non-complian balloon; PCI: Percutaneous coronary intervention; MV: Main vessel; POT: Proximal optimization technique; SB: Side branch; TIMI: Thrombolysis in myocardial infarction.

Table 4. Clinical and technical endpoints per study group

Parameters	Mini-crush Group (n=174)	CBC Group (n=117)	P value
Follow-up time, month	5.48±2.72	4.93±2.61	0.086
Co-primary clinical end-point (MACE), n (%)	7 (4.0)	3 (2.6)	0.745
Cardiac death	2 (1.1)	1 (0.9)	1.000
Myocardial infarction	6 (3.4)	2 (1.7)	0.482
Target lesion revascularization	5 (2.9)	2 (1.7)	0.706
Co-primary technical end-point, n (%)	116 (66.7)	12 (10.3)	**<0.001**
Secondary clinical end-point (MACCE), n (%)	9 (5.2)	3 (2.6)	0.373
All-cause death	2 (1.1)	1 (0.9)	1.000
Myocardial infarction	6 (3.4)	2 (1.7)	0.482
Target vessel revascularization	6 (3.4)	2 (1.7)	0.482
Stent thrombosis	4 (2.3)	1 (0.9)	0.651
Stroke	1 (0.6)	0 (0)	1.000

CBC: Controlled balloon crush; MACE: Major adverse cardiovascular events; MACCE: Major adverse cardiovascular and cerebral events.

OP-057 [Interventional Cardiology / Coronary]

Double kissing culotte or nano-crush stenting for true coronary bifurcation lesions: The multicenter COLLECT-BIF-II registry

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Background and Aim: Double kissing culotte technique (DCT) and nano-crush (NCT) techniques have recently gained popularity, however, the comparison of the clinical results of both techniques remains unclear. This non-randomized multicenter study aimed to retrospectively assess the mid-term clinical outcomes of DCT and NCT in individuals with true coronary bifurcation lesions (CBLs).

Methods: This large-scale multicenter (n=10) observational study included a total of 384 consecutive patients with true CBLs who underwent DCT or NCT. The primary endpoint was major adverse adverse cardiac events (MACE) as the combination of cardiac death, target vessel myocardial infarction (TVMI), or clinically driven target lesion revascularization (TLR) during follow-up. Besides, the secondary endpoint was measured as majör adverse cardiovascular and cerebral events (MACCE) including all-cause death, clinically driven TLR, TVMI, stent thrombosis, or stroke. Inverse probability weighting (IPW)-Cox analysis was performed to reduce selection bias caused by non-random selection. This is the first study to compare the clinical outcomes of DCT and NCT in patients with true CBLs.

Results: The initial revascularization strategy was NCT in 132 (34.4%) cases and DCT in 252 (65.6%) patients. Prevelance of priop PCI (40.9 vs. 29.4%, p=0.023), moderate or severe valve disease (12.9 vs. 6.7%, p=0.045), were significantly higher NCT group than in the DCT group. Whereas, side branch diamater (2.73 ± 0.29 vs. 2.62 ± 0.27 mm, p<0.001), side branch stent diameter (2.68 ± 0.31 vs. 2.64 ± 0.27 mm, p=0.005), number of balloons (6.30 ± 1.84 vs. 5.97 ± 1.81, p=0.041), procedure time (65.10 ± 20.34 vs. 59.66 ± 21.48 min, p=0.001), and contrast media volume (236.11 ± 86.98 vs. 203.37 ± 68.99, p<0.001) were notably higher DCT group than in the NCT group. In the overall population, the mid-term MACE (adjusted HR (IPW): 1.341, p=0.352) and MACCE (adjusted HR (IPW): 0.775, p=0.346) did not differ in patients with true CBLs treated with DCT and the NCT. Other endpoints were also comparable between the two groups. High SYNTAX score (adjusted HR (IPW): 1.134, p<0.001), proximal side branch optimization (adjusted HR (IPW): 0.378, p=0.002), and non-fatal intra-procedural complications (HR: 2.263, p=0.007) were found to be independent predictors of MACE. Besides, proximal side branch optimization (adjusted HR (IPW): 0.421, p=0.003), non-fatal intra-procedural complications (adjusted HR (IPW): 2.691, p=0.001), and localization of bifurcation disease (left main) were found to be independent predictors of MACCE.

Conclusions: This trial demonstrates that both bifurcation stenting techniques have comparable ischemia-driven combined outcomes. However, resource utilization, procedure time, and contrast medium volume were significantly higher in the DCT group. Nevertheless, a multicenter randomized trial comparing the DCT with the NCT with clearly defined inclusion and exclusion criteria would raise the evidence available to guide the management of patients with CBLs.

Table 1. Baseline characteristics of per study group

Variables	Nano-crush Group (n=132)	DK-culotte Group (n=252)	P value
Age (years)	61.48±8.79	59.98±10.44	0.140
Gender, male, n (%)	93 (70.5)	208 (82.5)	**0.006**
Comorbidities, n(%)			
Hypertension	87 (65.9)	138 (54.8)	**0.035**
Diabetes Mellitus	54 (40.9)	92 (36.5)	0.399
Hyperlipidemia	81 (61.4)	116 (46.0)	**0.004**
Chronic kidney disease	25 (18.9)	35 (13.9)	0.195
Current Smoker	66 (50.0)	144 (57.4)	0.168
History of Stroke	3 (2.3)	4 (1.6)	0.696
Prior PCI	54 (40.9)	74 (29.4)	**0.023**
Prior MI	42 (31.8)	54 (21.4)	**0.026**
Heart Failure	22 (16.7)	36 (14.3)	0.536
LV Ejection Fraction (%)	54.71±9.41	53.40±9.28	0.093
Moderate-severe Valve Disease, n (%)	17 (12.9)	17 (6.7)	**0.045**
Laboratory measurements			
White blood cell count, (10^9/L)	9.37±2.78	9.32±2.86	0.699
Hemoglobin, (g/dL)	13.37±2.06	13.37±2.07	0.878
Platelet count, (10^9/L)	244.26±74.36	250.09±71.40	0.494
Creatinine, (mg/dL)	1.06±.71	.96±.45	0.376
Total cholesterol, (mg/dL)	180.01±55.90	184.33±48.55	0.449
Clinical Presentation, n (%)			
CCS	59 (44.7)	92 (36.5)	0.119
NSTEMI	61 (46.2)	122 (48.4)	0.682
USAP	12 (9.1)	38 (15.1)	0.098
Medications Used, n (%)			
Acetylsalicylic acid	132 (100.0)	252 (100.0)	-
Clopidogrel	62 (47.0)	131 (52.0)	0.351
Ticagrelor	59 (44.7)	108 (42.9)	0.730
Prasugrel	11 (8.3)	13 (5.2)	0.222
Beta Blockers	126 (95.5)	231 (91.7)	0.168
CCB	20 (15.2)	41 (16.3)	0.776
ACEI/ARB	104 (78.8)	207 (82.1)	0.426
Statin	126 (95.5)	243 (96.4)	0.640
Diuretics	24 (18.2)	25 (9.9)	**0.021**
Insulin	33 (25.0)	34 (13.5)	**0.005**

ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, CCB: Calcium channel blocker; CCS: Chronic coronary syndrome, DK: Double kissing; LV: Left ventricle; MI: Myocardial infaction; NSTEMI: Non-ST segment elevation myocardial infarction; PCI: Percutaneous coronary intervention; USAP: Unstable angina pectoris.

Table 2. Lesions characteristics per study group

Parameters	Nano-crush Group (n=132)	DK-culotte Group (n=252)	P value
Multi-vessel disease, n(%)	90 (68.2)	120 (47.6)	**<0.001**
SYNTAX score	23.99±6.85	19.20±6.63	**<0.001**
SYNTAX score ≤22, n(%)	57 (43.2)	172 (68.3)	**<0.001**
SYNTAX score 23-32, n(%)	51 (38.6)	57 (22.6)	**0.001**
SYNTAX score ≥33, n(%)	24 (18.2)	20 (7.9)	**0.003**
Locations of bifurcation lesions, n(%)			
LMCA	13 (9.8)	18 (7.1)	0.355
LAD-Diagonal	80 (60.6)	153 (60.7)	0.984
LCx-OM	33 (25.0)	70 (27.8)	0.560
PDA-PL	6 (4.5)	11 (4.4)	1.00
Type of Medina classification, n(%)			
1.0.1	3 (2.3)	46 (18.3)	**<0.001**
0.1.1	39 (29.5)	70 (27.8)	0.715
1.1.1	91 (68.9)	131 (52.0)	**0.001**
Reference vessel diameter, mm			
MV	3.16±.45	3.17±.40	0.575
SB	2.62±.27	2.73±.29	**<0.001**
SB reference vessel diameter ≥2.5 mm, n(%)	110 (83.3)	231 (91.7)	**0.014**
Complexity of bifurcation lesions, n(%)			
SB diameter stenosis 70 or 90%	132 (100.0)	199 (79.0)	**<0.001**
Moderate or severe calcification	44 (33.3)	48 (19.0)	**0.002**
MV reference diameter < 2.5mm	1 (0.8)	11 (4.4)	0.065
Multiple lesions	96 (72.7)	121 (48.0)	**<0.001**
Bifurcation angle <45° or >70°	68 (51.5)	109 (43.3)	0.123
Thrombus identified by angiography	16 (12.1)	20 (7.9)	0.181
Lesion length, mm			
MV	26.89±8.47	22.30±8.77	**<0.001**
SB	16.89±5.27	15.21±7.67	**<0.001**
MV, n(%)			
TIMI flow grade <3	15 (11.4)	27 (10.7)	0.846
Chronic total occlusion	4 (3.0)	3 (1.2)	0.239
Thrombus-containing lesion	7 (5.3)	14 (5.6)	1.00
SB, n(%)			
TIMI flow grade <3	9 (6.8)	18 (7.1)	1.00
Chronic total occlusion	3 (2.3)	2 (0.8)	0.344
Thrombus-containing lesion	7 (5.3)	13 (5.2)	1.00

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

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

Parameters	Nano-crush Group (n=132)	DK-culotte Group (n=252)	P value
Access site, n(%)			
Femoral	128 (97.0)	249 (98.8)	0.201
Radial	4 (3.0)	3 (1.2)	0.239
Tirofiban use during PCI, n (%)	11 (8.3)	15 (6.0)	0.378
Performed IVUS, n(%)	10 (7.6)	19 (7.5)	0.990
Thrombus Aspiration, n(%)	1 (0.8)	5 (2.0)	0.669
Pre-dilation			
MV	112 (84.8)	209 (82.9)	0.631
SB	111 (84.1)	209 (82.9)	0.773
Proximal side-branch optimization, n **(%)**	109 (82.6)	218 (86.5)	0.303
Final kissing balloon inflation	129 (97.7)	252 (100.0)	**0.016**
Final POT, n (%)	129 (97.7)	247 (98.0)	0.851
MV			
Stent number, n	1.18±0.39	1.12±0.33	0.119
Stent diameter, mm	3.12±0.42	3.01±0.34	**0.013**
Stent length, mm	31.52±7.89	27.98±9.28	**<0.001**
SB			
Stent number, n	1.02±0.15	1.04±0.19	0.488
Stent diameter, mm	2.64±0.27	2.68±0.31	**0.005**
Stent length, mm	21.15±6.17	21.31±7.09	0.820
Resource utilization, n (%)			
Guiding catheter number, n	2.16±.37	2.16±.39	**0.026**
Guidewire number, n	2.47±.73	2.71±.88	**0.009**
Balloon number, n	5.97±1.81	6.30±1.84	**0.041**
Any type of intraprocedural complication, n(%)	14 (10.6)	42 (16.7)	0.110
Abrupt occlusion			
MV	1 (0.8)	10 (4.0)	0.106
SB	2 (1.5)	5 (2.0)	1.00
TIMI<3			
MV	3 (2.3)	18 (7.1)	0.058
SB	4 (3.0)	10 (4.0)	0.779
Dissection			
MV	4 (3.0)	17 (6.7)	0.159
SB	3 (2.3)	6 (2.4)	1.00
Thrombus formation			
MV	2 (1.5)	3 (1.2)	1.00
SB	4 (3.0)	4 (1.6)	0.454
Coronary Perforation			
MV	0 (0.0)	2 (0.8)	0.548
SB	0 (0.0)	1 (0.4)	1.00
Procedure time, min	59.66±21.48	65.10±20.34	**0.001**
Fluoroscopy time, min	20.71±6.27	23.80±8.19	**<0.001**
Contrast media volume (mL)	203.37±68.99	236.11±86.98	**<0.001**
Angiographic success, n(%)			
MV	130 (98.5)	248 (98.4)	0.957
SB	129 (97.7)	248 (98.4)	0.633

*Chi-squared test **Fisher's exact test ***Mann–Whitney U test

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

Table 4. In hospital and mid-term clinical outcomes per study group

Parameters	Nano-crush Group (n=132)	DK-culotte Group (n=252)	P value
In-hospital complications, n (%)			
Death	2 (1.5)	2 (0.8)	0.610
Major bleeding	5 (3.8)	3 (1.2)	0.130
Pseudoaneurysm	3 (2.3)	6 (2.4)	1.00
Fatal arrhythmias	2 (1.5)	4 (1.6)	1.00
Stent thrombosis	2 (1.5)	2 (0.8)	0.610
Myocardial infarction	2 (1.5)	3 (1.2)	1.00
Contrast-induced AKI	12 (9.1)	21 (8.3)	0.801
Follow-up time, month	29.58±10.82	27.75±11.62	0.074
Primary end-point (MACE), n (%)	24 (18.2)	29 (11.5)	0.072
Cardiac death	6 (4.5)	7 (2.8)	0.383
Target vessel myocardial infarction	15 (11.4)	17 (6.7)	0.120
Target lesion revascularization	18 (13.6)	24 (9.5)	0.220
Secondary end-point (MACCE), n (%)	26 (19.7)	34 (13.5)	0.112
All-cause death	7 (5.3)	13 (5.2)	1.00
Target vessel myocardial infarction	15 (11.4)	17 (6.7)	0.120
Target lesion revascularization	18 (13.6)	24 (9.5)	0.220
Stent thrombosis	7 (5.3)	9 (3.6)	0.430
Stroke	1 (0.8)	4 (1.6)	0.664

*Fisher's exact test **Chi-squared test ***Mann–Whitney U test

DK: Double kissing; MACE: Major adverse cardiovascular events; MACCE: Major adverse cardiovascular and cerebral events.

Table 5. IPW-Cox proportional hazard analysis showing independent predictors of primary endpoint (MACE)

Parameters	Unadjusted HR	95% CI	P	Adjusted R (IPW)	95% CI	P
DK-culotte technique	0.666	[0.388-1.145]	0.141	1.341	[0.723-2.487]	0.352
Hyperntension	2.120	[1.134-3.965]	0.019	1.530	[0.793-2.954]	0.205
Chronic kidney disease	1.881	[1.021-3.465]	0.043	1.046	[0.545-2.010]	0.891
Prior PCI	1.876	[1.093-3.217]	0.022	1.246	[0.697-2.226]	0.458
SYNTAX score	1.153	[1.109-1.199]	<0.001	1.134	[1.086-1.184]	**<0.001**
Proximal SB optimization	0.252	[0.145-0.438]	<0.001	0.378	[0.207-0.691]	**0.002**
Intraprocedural complication	4.239	[2.440-7.361]	<0.001	2.263	[1.253-4.089]	**0.007**

CI: Confidence interval; DK: Double kissing; HR: Hazard ratio; IPW: Inverse probability-weighted; MACE: Major adverse cardiovascular events; SB: Side branch.

Table 6. IPW-Cox proportional hazard analysis showing independent predictors of secondary endpoint (MACCE)

Parameters	Unadjusted HR	95% CI	P	Adjusted HR (IPW)	95% CI	P
DK-culotte technique	0.728	[0.437-1.214]	0.224	0.775	[0.456-1.316]	0.346
Diabetes mellitus	1.658	[0.999-2.751]	0.050	1.180	[0.677-2.056]	0.559
Hypertension	1.891	[1.067-3.352]	0.029	1.446	[0.795-2.631]	0.227
Chronic kidney disease	2.248	[1.294-3.096]	0.004	1.794	[0.999-3.221]	0.050
Prior PCI	1.969	[1.186-3.268]	0.009	1.521	[0.880-2.630]	0.133
Proximal SB optimization	0.330	[0.193-0.564]	<0.001	0.421	[0.237-0.745]	**0.003**
LMCA localization	2.689	[1.362-5.310]	0.004	2.791	[1.386-5.623]	**0.004**
Intraprocedural complication	3.272	[1.910-5.605]	<0.001	2.691	[1.493-4.847]	**0.001**

CI: Confidence interval; DK: Double kissing; HR: Hazard ratio; IPW: Inverse probability-weighted; MACCE: Major adverse cardiovascular and cerabral events; SB: Side branch.

OP-058 [Interventional Cardiology / Coronary]

Crossover vs. ostial stent implantation for ostial left circumflex artery lesions: The multicenter CROSS-LCX registry

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Background and Aim: To date, the optimal revascularization strategy for patients with ostial left circumflex artery (LCx) lesions has not been established. This study sought to assess the cardiovascular outcomes of the crossover stenting (CSI) and ostial stent implantation (OSI) for the ostial LCx lesions under long-term follow-up.

Methods: This large-scale multicenter (n=12) observational retrospective study included 414 patients [men: 290 (70%), mean

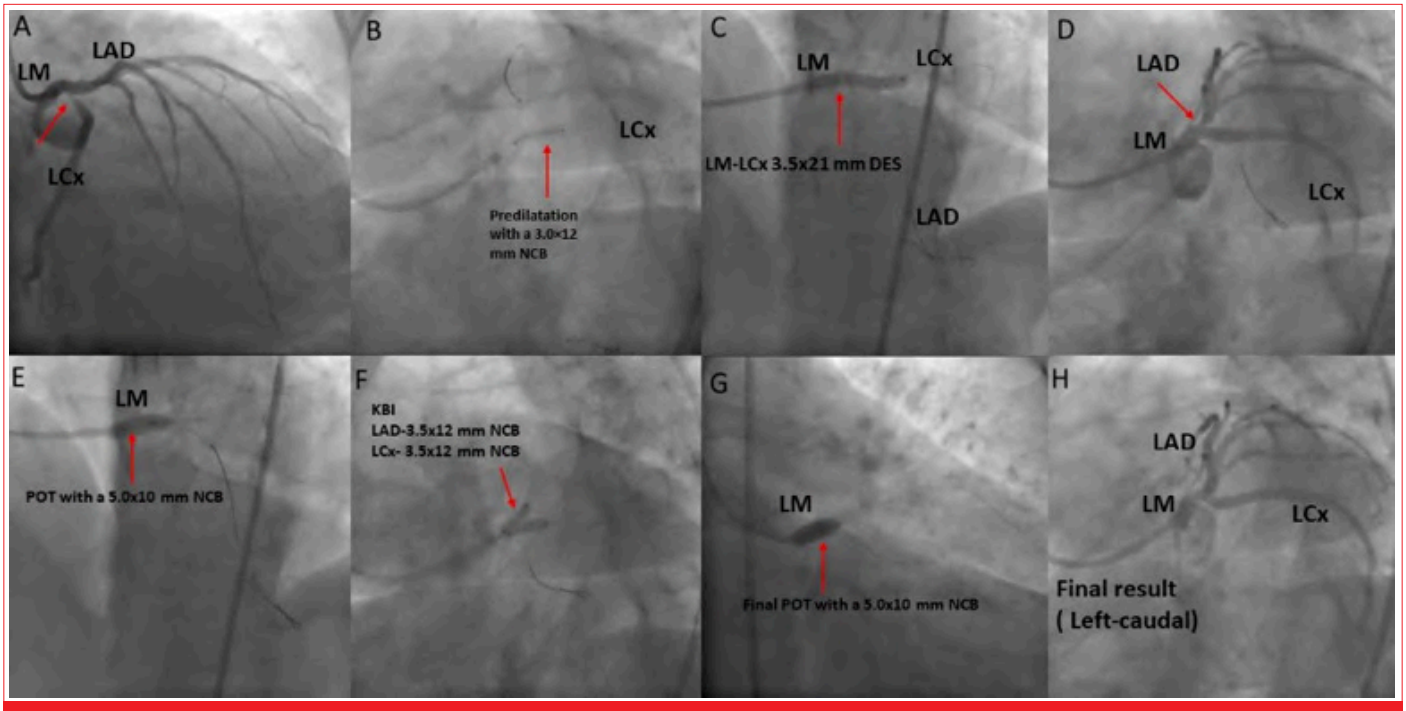


Figure 1. A: Baseline angiographic view of the ostial LCx lesion in the right caudal projection, B: Pre-dilatation with a non-compliant (NC) balloon at the lesion site, C: Crossover stent implantation from the left main coronary artery (LM) to the LCx, D: Post-stenting angiographic result in the right caudal projection, E: First proximal optimization technique (POT) performed with a short NC balloon, F: Final kissing balloon inflation between the LAD and LCx, G: Second POT (final POT) following kissing balloon inflation, H: Final angiographic result in the left caudal projection. DES: Drug-eluting stent, KBI: Kissing balloon inflation, LAD: Left anterior descending artery, LCx: Left circumflex artery, LM: Left main coronary artery, NCB: Non-Compliant balloon, POT: Proximal optimization technique.

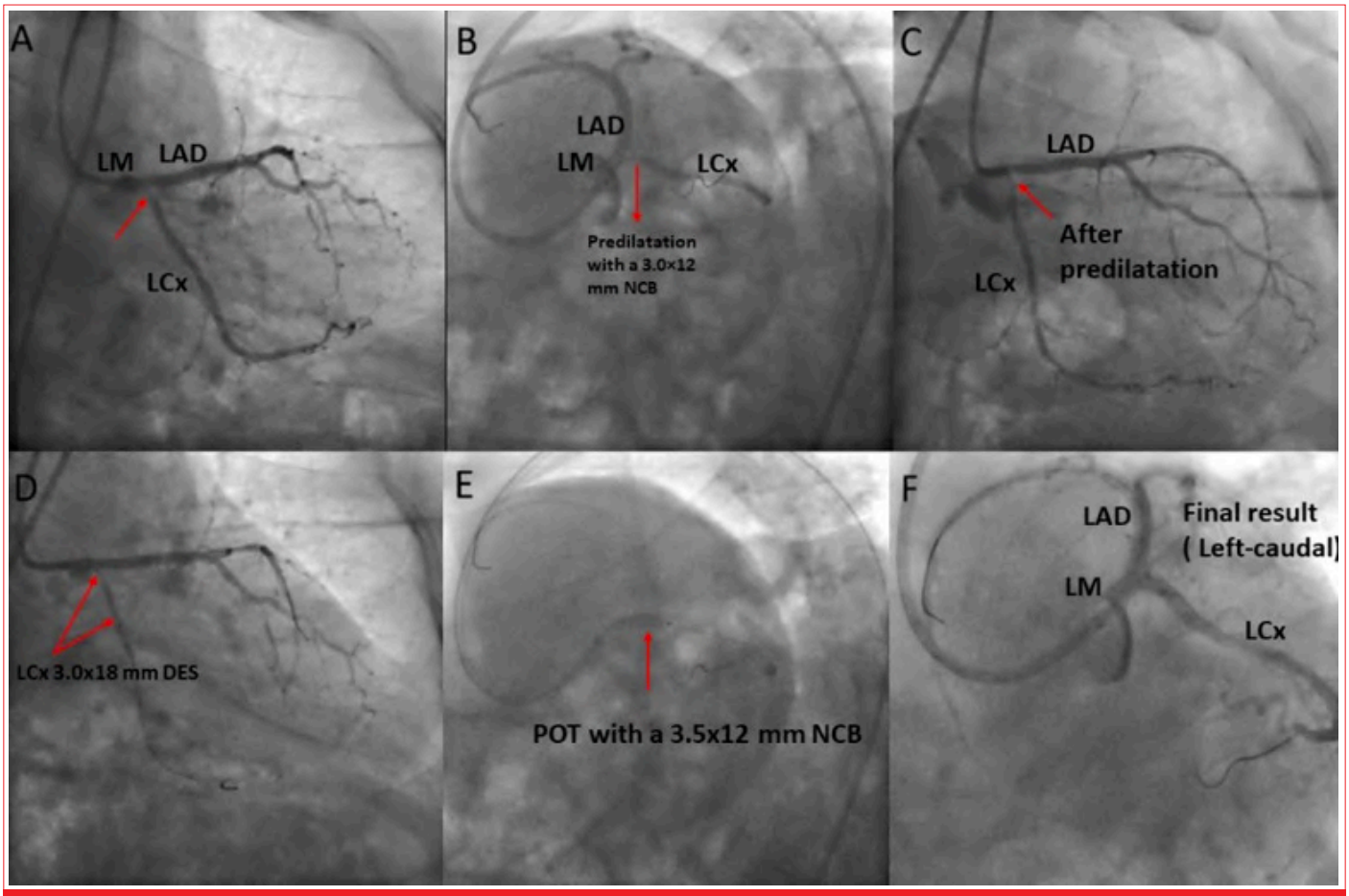


Figure 2. A: Baseline angiographic view of the ostial LCx lesion in the right caudal projection, B: Pre-dilatation with a non-compliant (NC) balloon at the lesion site, C: Post-dilatation angiographic view in the right caudal projection, D: Ostial stent implantation in the right caudal view, E: Proximal optimization technique (POT) following ostial stent implantation, F: Final angiographic result in the spider view. DES: Drug-eluting stent, LAD: Left anterior descending (artery), LCx: Left circumflex artery, LM: Left main coronary Artery, NC: Non-Compliant balloon, POT: Proximal optimization technique.

age: 64.95 ± 11.73 years] who underwent PCI with CSI or OSI for ostial LCx lesions between 2014 and 2025. The primary outcome was major adverse cardiac events (MACE), including cardiac death, target lesion revascularization, and target vessel myocardial infarction.

Results: The study cohort was divided into two groups as OSI (n=212) and CSI (n=202). SYNTAX scores and the utilization of intravascular imaging rates were similar in both groups. Stent diameter (3.27 ± 0.34 vs. 2.92 ± 0.27 mm, p<0.001), total stent length (25.26 ± 6.95 vs. 22.83 ± 7.46 mm, p<0.001), and incidences of the LAD narrowing >50% (25.7 vs. 6.1%, p<0.001), and bail-out 2 stent technique (13.9 vs. 5.7%, p=0.005) were notably higher in the CSI group than in the OSI group. The risk-adjusted long-term MACE (HR: 0.361, p<0.001), major adverse cardiac and cerebral events (HR: 0.393, p=0.001) significantly differed in individuals with ostial LCx lesions to revascularize with CSI and OSI. Additionally, diabetes mellitus, chronic kidney disease, intravascular imaging, reduced left ventricle ejection fraction, high SYNTAX score, and direct stenting were found to be independent predictors of MACE.

Conclusions: The present study suggests that CSI was associated with lower risk-adjusted MACE and MACCE rates at long-term follow-up.

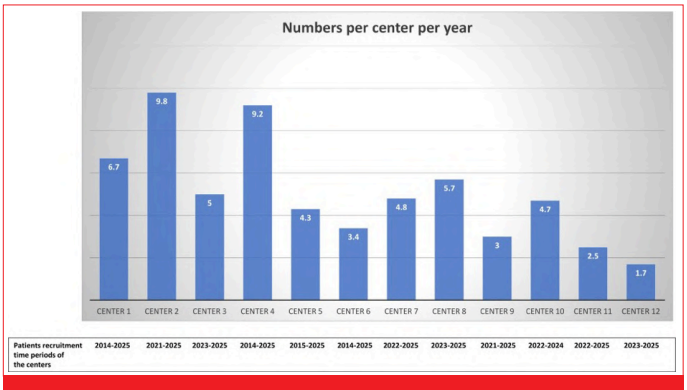


Figure 3. Distribution of the total number of enrolled patients per center per year.

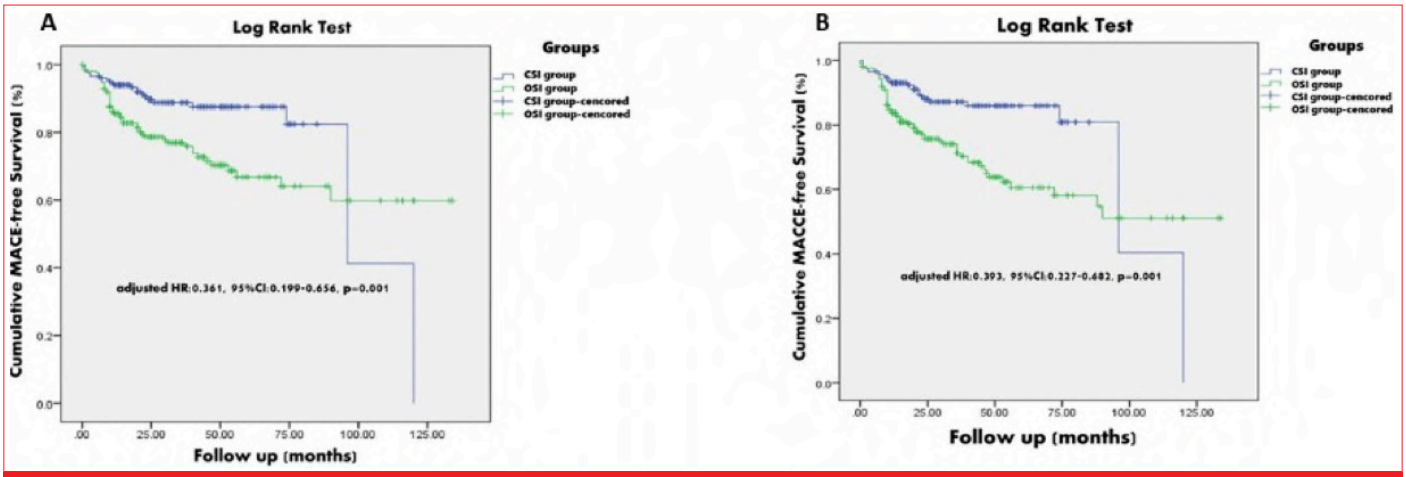
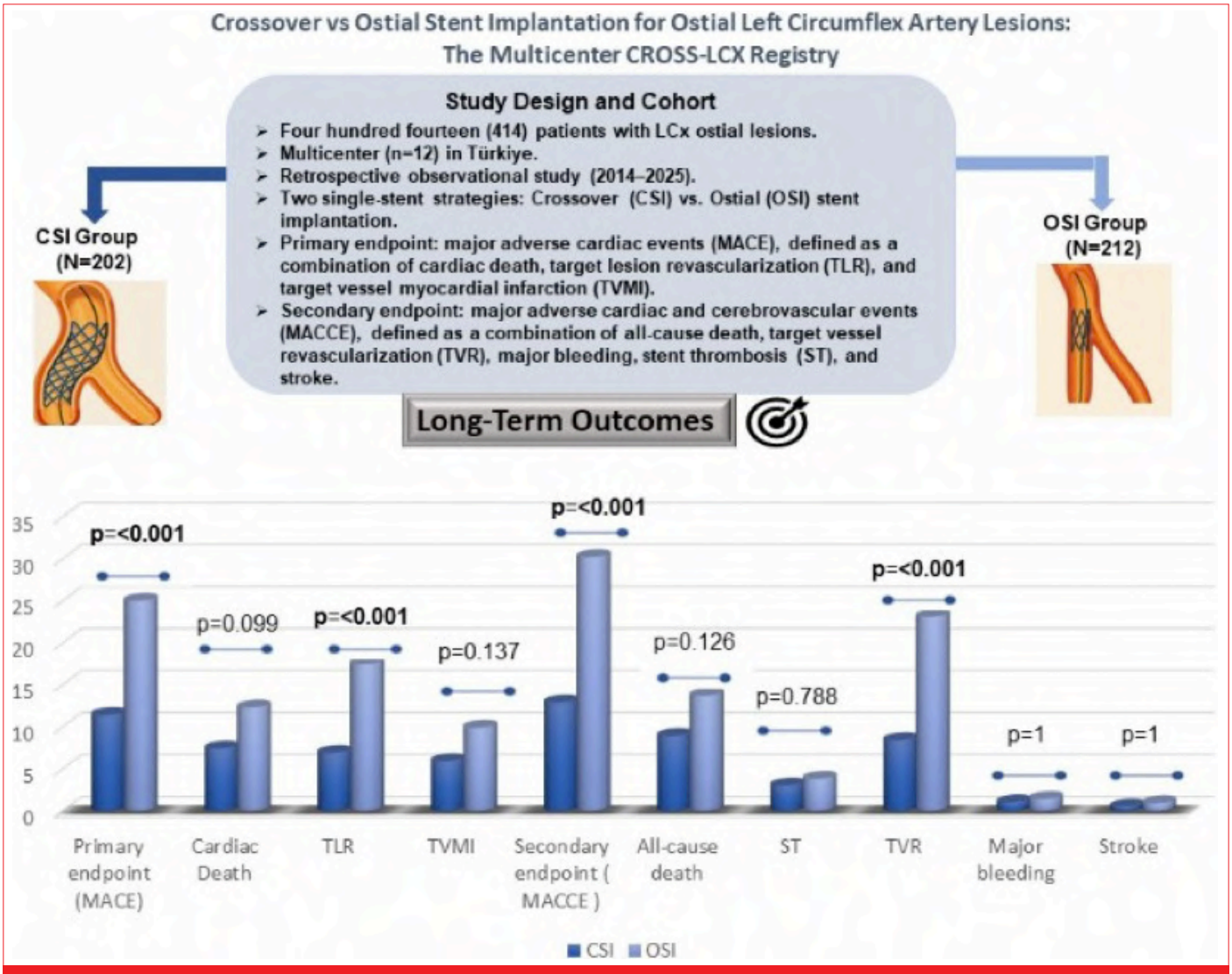


Figure 4. Kaplan–Meier curves for MACE (A) and MACCE (B) according to the stenting strategy of CSI (blue) versus OSI (green). CI: Confidence intervals; CSI: Crossover stent implantation; HR: Hazard ratio; MACE: Major adverse cardiac events; MACCE: Major adverse cardiac and cerebrovascular events; OSI: Ostial stent implantation.



Graphical abstract

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

Variables	CSI (n= 202)	OSI (n= 212)	P value
Age (years)	66.44±11.18	63.52±12.08	**0.011**
Gender, male, n (%)	143 (70.8)	147 (69.3)	0.747
Diabetes Mellitus, n (%)	88 (43.6)	90 (42.5)	0.819
Hypertension, n (%)	144 (71.3)	152 (71.7)	0.926
Current Smoker, n (%)	97 (48.0)	116 (54.7)	**0.009**
Hyperlipidemia, n (%)	101 (50.2)	109 (51.4)	0.813
History of Stroke, n (%)	8 (4.0)	6 (2.8)	0.594
Chronic Kidney Disease, n (%)	40 (19.8)	49 (23.1)	0.412
Previous PCI, n (%)	95 (47.0)	101 (47.6)	0.901
Peripheral Artery Disease, n (%)	19 (9.4)	18 (8.5)	0.744
Multi-vessel Disease, n (%)	137 (67.8)	134 (63.2)	0.324
Clinical Presentation, n (%)			
STEMI	39 (19.3)	36 (17.0)	0.539
NSTEMI	75 (37.1)	83 (39.2)	0.672
USAP	13 (6.4)	17 (8.0)	0.535
CCS	75 (37.1)	76 (35.8)	0.787
LV Ejection Fraction (%)	52.00±9.27	52.19±10.00	0.843
Moderate-severe Valve Disease, n (%)	24 (11.9)	29 (13.7)	0.584
Medication used, n (%)			
Acetylsalicylic acid	198 (98.0)	211 (99.5)	0.160
Clopidogrel	124 (61.4)	117 (55.2)	0.201
Ticagrelor	68 (33.7)	85 (40.1)	0.175
Prasugrel	10 (5.0)	10 (4.7)	0.912
Beta-Blockers	183 (90.6)	194 (91.5)	0.744
CCB	33 (16.3)	32 (15.1)	0.728
ACEI/ARB	163 (80.7)	169 (79.7)	0.803
Statin	195 (96.5)	203 (95.8)	0.681
Diuretics	41 (20.3)	47 (22.2)	0.642
Nitrate	21 (10.4)	22 (10.4)	0.995
Insulin	37 (18.3)	39 (18.4)	0.983

ACEI: Angiotensin-converting inhibitor, ARB: Angiotensin receptor blocker, CCB: Calcium channel blocker, CCS: Chronic coronary syndrome, CSI: Crossover stent implantation, LV: Left ventricle, NSTEMI: Non-ST segment elevation myocardial infarction, OSI: Ostial stent implantation, PCI: Percutaneous coronary intervention, STEMI: ST segment elevation myocardial infarction, USAP: Unstable angina pectoris.

Table 2. Angiographic and procedural characteristics of the study population

Variables	CSI (n= 202)	OSI (n= 212)	P value
Access site, n (%)			
Femoral	184 (91.1)	188 (88.7)	0.417
Radial	18 (8.9)	24 (11.3)	0.417
Guiding catheter size, n (%)			
6 Fr	22 (10.9)	70 (33.0)	**<0.001**
7 Fr	180 (89.1)	142 (67.0)	**<0.001**
LM diameter, mm	4.69±0.40	4.68±0.41	0.730
Proximal LCx diameter, mm	3.42±0.39	3.29±0.37	**<0.001**
Proximal LAD diameter, mm	3.37±0.38	3.49±0.44	**0.002**
Percentage of ostial LCx stenosis, %	86.67±8.88	86.80±9.67	0.888
Percentage of ostial LAD stenosis, %	6.88±11.62	5.21±10.23	0.121
Thrombus identified by angiography, n (%)	33 (16.3)	38 (17.9)	0.668
LM stenosis <30%, n (%)	45 (22.3)	48 (22.6)	0.929
SYNTAX Score	20.23±8.91	18.95±9.82	0.167
Intra-aortic balloon pump support, n (%)	11 (5.4)	10 (4.7)	0.736
Performed IVUS, n (%)	56 (27.7)	55 (25.9)	0.683
Rotablator use, n (%)	4 (2.0)	0 (0)	0.056
Thrombus Aspiration, n (%)	2 (1.0)	3 (1.4)	1.00
Direct stenting, n (%)	12 (5.9)	13 (6.1)	0.935
Second or third generation stent, n (%)			
Everolimus-eluting	64 (31.7)	72 (34.0)	0.622
Zotarolimus-eluting	29 (14.4)	28 (13.2)	0.735
Sirolimus-eluting	109 (54.0)	112 (52.8)	0.818
Stent diameter, mm	3.27±0.34	2.92±0.27	**<0.001**
Total stent length, mm	25.26±6.95	22.83±7.46	**<0.001**
Maximum post-dilatation balloon diameter, mm	4.45±0.49	3.33±0.45	**<0.001**
SB protection, n (%)			
Jailed wire technique	168 (83.2)	62 (29.2)	**<0.001**
Jailed balloon technique	8 (4.0)	0 (0)	**0.003**
LAD narrowing >50%, n (%)	52 (25.7)	13 (6.1)	**<0.001**
Side branch intervention, n (%)			
POT-kissing-POT	73 (36.1)	6 (2.8)	**<0.001**
POT-side-POT	4 (2.0)	0 (0)	0.056
Bailout 2-stent technique	28 (13.9)	12 (5.7)	**0.005**
Tirofiban use during PCI, n (%)	15 (7.4)	18 (8.5)	0.689
Contrast media volume (mL)	204.65±68.07	204.50±68.08	0.982
Procedure time, min	54.35±21.04	51.34±20.03	0.138
Fluoroscopy time, min	19.60±7.84	18.85±8.49	0.353

Fr: French, IVUS: Intravascular ultrasound, LAD: Left anterior descending, LCx: Left circumflex, LM: Left main coronary artery, PCI: Percutaneous coronary intervention, POT: Proximal optimization technique, SB: Side-branch.

Table 4. Univariate and multivariate Cox regression analysis showing independent predictors of MACE in patients with ostial LCx stenosis

Variables	Univariate Level		Multivariate Level	
	HR (95% CI)	P	HR (95% CI)	P
Treatment (COS)	0.453 [0.277-0.741]	0.002	0.361 [0.199-0.656]	**0.001**
Diabetes mellitus	3.389 [2.060-5.576]	<0.001	2.467 [1.413-4.308]	**0.002**
Chronic kidney disease	4.171 [2.639-6.591]	<0.001	2.426 [1.429-4.119]	**0.001**
Clinical presentation (CCS)	0.647 [0.388-1.078]	0.095	0.879 [0.505-1.529]	0.648
LV ejection fraction	0.944 [0.926-0.964]	<0.001	0.964 [0.942-0.986]	**0.002**
SYNTAX score	1.077 [1.051-1.102]	<.001	1.082 [1.054-1.112]	**<0.001**
Performed IVUS	0.431 [0.227-0.817]	0.010	0.487 [0.250-0.950]	**0.030**
Direct stenting	4.731 [2.637-8.486]	<0.001	4.216 [2.195-8.099]	**<0.001**
Total stent length	0.972 [0.940-1.005]	0.091	0.999 [0.964-1.036]	0.966
Gender (male)	0.738 [0.463-1.177]	0.202	0.863 [0.515-1.446]	0.577
Age	1.005 [0.986-1.025]	0.589	0.996 [0.974-1.019]	0.755
Stent diameter	0.745 [0.379-1.464]	0.393	0.849 [0.401-1.797]	0.668
Current smoker	1.094 [0.713-1.681]	0.681	1.512 [0.923-2.478]	0.100

CCS: Chronic coronary syndrome, CI: Confidence Interval, CSI: Crossover stent implantation, HR: Hazard ratio, IVUS: Intravascular ultrasound, LCx: Left circumflex artery, LV: Left ventricle, MACE: Major adverse cardiac events.

Table 5. Univariate and multivariate Cox regression analysis showing independent predictors of MACCE in patients with ostial LCx stenosis

Variables	Univariate Level		Multivariate Level	
	HR (95% CI)	P	HR (95% CI)	P
Treatment (COS)	0.426 [0.269-0.674]	<0.001	0.393 [0.227-0.682]	**0.001**
Diabetes mellitus	2.918 [1.866-4.564]	<0.001	2.116 [1.292-3.466]	**0.003**
Chronic kidneydisease	3.267 [2.132-5.007]	<0.001	2.078 [1.279-3.378]	**0.003**
Clinical presentation (CCS)	0.814 [0.521-1.273]	0.367	1.037 [0.641-1.678]	0.882
LV ejection fraction	0.952 [0.935-0.970]	<0.001	0.968 [0.948-0.988]	**0.002**
SYNTAX score	1.062 [1.040-1.086]	<0.001	1.067 [1.042-1.093]	**<0.001**
Performed IVUS	0.549 [0.320-0.944]	0.030	0.584 [0.333-1.024]	0.060
Direct stenting	3.892 [2.193-6.910]	<0.001	3.218 [1.703-6.079]	**<0.001**
Total stent length	0.971 [0.942-1.001]	0.062	0.994 [0.963-1.027]	0.738
Gender (male)	0.680 [0.445-1.039]	0.075	0.777 [0.491-1.228]	0.279
Age	1.004 [0.987-1.023]	0.622	0.998 [0.978-1.018]	0.843
Stent diameter	0.594 [0.314-1.125]	0.110	0.741 [0.367-1.496]	0.403
Current smoker	0.963 [0.648-1.430]	0.851	1.310 [0.841-2.043]	0.233

CCS: Chronic coronary syndrome, CI: Confidence Interval, CSI: Crossover stenting, HR: Hazard ratio, IVUS: Intravascular ultrasound, LCx: Left circumflex artery, LV: Left ventricle, MACCE: Major adverse cardiac and cerebral events.

Table 6. Clinical outcomes of per study group

Variables	CSI (n=202)	OSI (n=212)	p value
In-hospital complications, n (%)			
Death	4 (2.0)	3 (1.4)	0.718
Fatal ventricular arrhythmias	4 (2.0)	4 (1.9)	1.000
Contrast-induced AKI	16 (7.9)	22 (10.4)	0.387
Pseudoaneurysm	6 (3.0)	8 (3.8)	0.788
Follow-up time, months	33.73 ± 22.59	34.25 ± 28.62	0.839
Long-term Outcomes, n (%)			
Primary end-point (MACE), n (%)	23 (11.4)	53 (25.0)	<0.001
Cardiac death	15 (7.4)	26 (12.3)	0.099
TLR	14 (6.9)	37 (17.5)	<0.001
TVMI	12 (5.9)	21 (9.9)	0.137
Secondary end-point (MACCE), n (%)	26 (12.9)	64 (30.2)	<0.001
All-cause death	18 (8.9)	29 (13.7)	0.126
TVR	17 (8.4)	49 (23.1)	<0.001
Major bleeding	2 (1.0)	3 (1.4)	1.00
Stent thrombosis	6 (3.0)	8 (3.8)	0.788
Stroke	1 (0.5)	2 (0.9)	1.00

AKI: Acute kidney injury, CSI: Crossover stent implantation, MACE: Major adverse cardiac events, MACCE: Major adverse cardiovascular and cerebral events, TLR: Target lesion revascularization, OSI: Ostial stent implantation, TIMI: Thrombolysis in myocardial infarction, TVMI: Target vessel myocardial infarction, TVR: Target vessel revascularization.

OP-059 [Interventional Cardiology / Coronary]

Ostial stent ımplantation or crossover stenting for ostial LAD lesions: The multicenter CROSS-ANATOLIA registry

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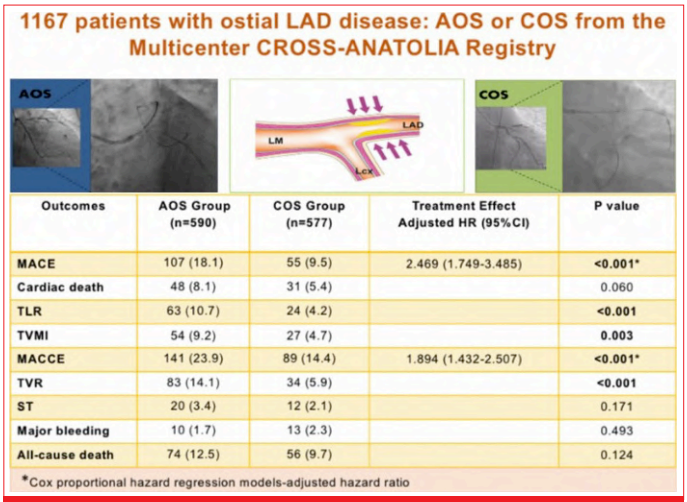
Background and Aim: Percutaneous coronary intervention (PCI) for isolated ostial left anterior descending artery (LAD) lesions remains technically difficult. Accurate ostial stenting (AOS) aims to prevent involvement of the left main coronary artery (LMCA), while crossover stenting (COS) ensures complete ostial coverage but may increase procedural complexity. This study aimed to evaluate the long-term outcomes of patients who underwent AOS or COS for ostial LAD disease.

Methods: From 2014 to 2025, patients who underwent PCI for ostial LAD lesions were retrospectively collected. The primary outcome was major adverse cardiac events (MACE), including cardiac death, target lesion revascularization (TLR), and target vessel myocardial infarction (TVMI).

Results: This large-scale multicenter (n=12) observational study included a total of 1,167 consecutive patients [men: 859 (73.6%), mean age: 61.70 ± 12.73 years] with ostial LAD lesions who underwent PCI; 590 (50.6%) of the cases were revascularized with AOS, and 577 (49.4%) were treated with COS. The incidences of



Outcomes	AOS Group (n=590)	COS Group (n=577)	Treatment Effect Adjusted HR (95%CI)	P value
MACE	107 (18.1)	55 (9.5)	2.469 (1.749-3.485)	<0.001*
Cardiac death	48 (8.1)	31 (5.4)		0.060
TLR	63 (10.7)	24 (4.2)		<0.001
TVMI	54 (9.2)	27 (4.7)		0.003
MACCE	141 (23.9)	89 (14.4)	1.894 (1.432-2.507)	<0.001*
TVR	83 (14.1)	34 (5.9)		<0.001
ST	20 (3.4)	12 (2.1)		0.171
Major bleeding	10 (1.7)	13 (2.3)		0.493
All-cause death	74 (12.5)	56 (9.7)		0.124

*Cox proportional hazard regression models-adjusted hazard ratio

Graphical abstract

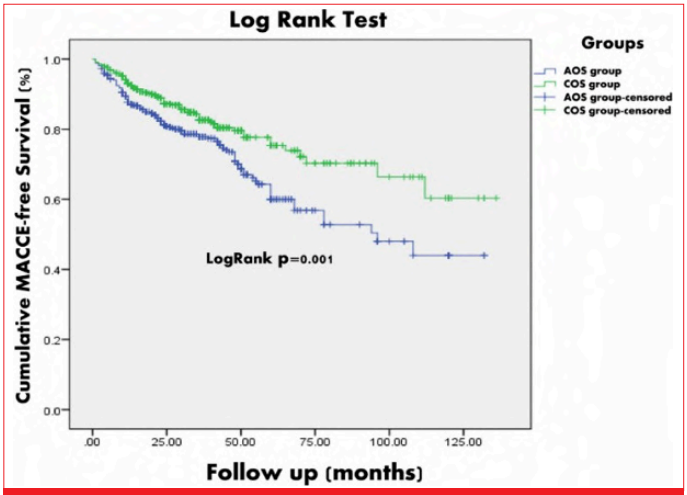


Figure 1. Kaplan-Meier analaysis of MACCE.

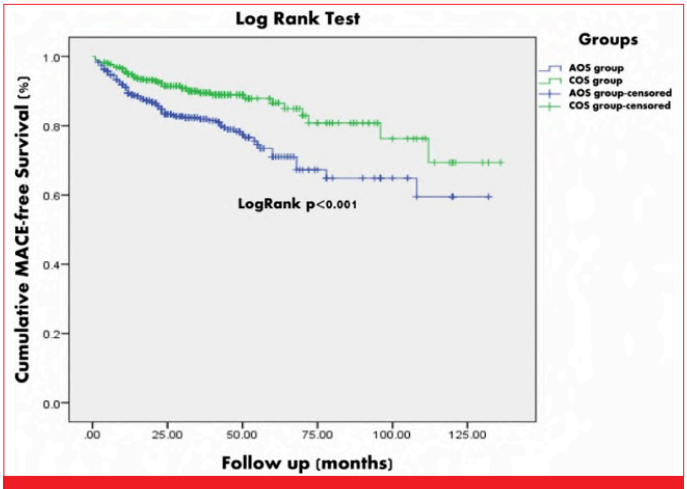


Figure 2. Kaplan-Meier analaysis of MACE.

MACE (18.1 vs. 9.5%, p<0.001), TVMI (9.2 vs. 4.7%, p=0.003), and clinically driven TLR (10.7 vs. 4.2%, p<0.001) were notably higher in the AOS group compared to the COS group. Treatment (AOS) (HR: 2.469, p<0.001), chronic kidney disease (HR: 1.832, p=0.003), left ventricle ejection fraction (HR: 0.984, p=0.042), SYNTAX score (HR: 1.089, p<0.001), utilization of intravascular imaging (HR: 0.689, p=0.049), direct stenting (HR: 2.171, p=0.001), stent length (HR: 1.036, p=0.001) were found to be independent predictors of MACE.

Conclusions: This non-randomized study suggests that COS was associated with better long-term MACE, TVMI, and clinically driven TLR rates compared with AOS in patients with ostial LAD disease.

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

Variables	AOS (n=590)	COS (n=577)	P value
Age (years)	60.82±12.16	62.60±13.47	**0.018**
Gender, male, n (%)	438 (74.5)	421 (73.0)	0.554
Body Mass Index, kg/m^2	26.95±3.05	26.98±2.95	0.865
Diabetes Mellitus, n (%)	268 (45.4)	225 (39.0)	**0.026**
Hypertension, n (%)	354 (60.0)	374 (64.8)	0.089
Current Smoker, n (%)	280 (47.5)	272 (47.1)	**0.010**
Hyperlipidemia, n (%)	285 (48.3)	281 (48.7)	0.893
History of Stroke, n (%)	18 (3.1)	20 (3.5)	0.689
Chronic Kidney Disease, n (%)	74 (12.5)	77 (13.3)	0.683
Previous PCI, n (%)	173 (29.3)	187 (32.4)	0.254
Peripheral Artery Disease, n (%)	37 (6.3)	45 (7.8)	0.311
Multi-vessel Disease, n (%)	319 (54.1)	304 (52.7)	0.636
Clinical Presentation, n (%)			
STEMI	156 (26.4)	138 (23.9)	0.321
NSTEMI	200 (33.9)	213 (36.9)	0.281
USAP	53 (9.0)	43 (7.5)	0.341
CCS	181 (30.7)	183 (31.7)	0.702
LV Ejection Fraction (%)	47.97±11.88	47.88±11.18	0.889
Moderate-severe Valve Disease, n (%)	62 (10.5)	59 (10.2)	0.874
Medication used, n (%)			
Acetylsalicylic acid	585 (99.2)	575 (99.7)	0.447
Clopidogrel	258 (43.7)	250 (43.3)	0.890
Ticagrelor	302 (51.2)	271 (47.0)	0.149
Prasugrel	30 (5.1)	56 (9.7)	**0.003**
Beta-Blockers	513 (86.9)	521 (90.3)	0.072
CCB	67 (11.4)	89 (15.4)	**0.041**
ACEI/ARB	479 (81.2)	480 (83.2)	0.372
Statin	549 (93)	545 (94.5)	0.451
Diuretics	200 (33.9)	186 (32.2)	0.546
Nitrate	45 (7.6)	59 (10.2)	0.119
Insulin	83 (14.1)	76 (13.1)	0.573

Abbreviations: ACEI: Angiotensin-converting inhibitor, AOS: Accurate Ostial Stenting, ARB: Angiotensin receptor blocker, CCB: Calcium channel blocker, CCS: Chronic coronary syndrome, COS: Crossover Stenting, LV: Left ventricle, NSTEMI: Non-ST segment elevation myocardial infarction, PCI: Percutaneous coronary intervention, STEMI: ST segment elevation myocardial infarction, USAP: Unstable angina pectoris

Table 2. Angiographic and procedural characteristics of the study population

Variables	AOS (n=590)	COS (n=577)	P
Access site, n (%)			
Femoral	524 (88.8)	492 (85.3)	0.071
Radial	66 (11.2)	85 (14.7)	0.071
Guiding catheter size, n (%)			
6 Fr	203 (34.4)	58 (10.1)	**<0.001**
7 Fr	387 (65.6)	518 (89.8)	**<0.001**
LM diameter, mm	4.66±.39	4.66±.36	0.864
Proximal LAD diameter, mm	3.69±.37	3.64±.35	**0.024**
Proximal LCx diameter, mm	3.08±.40	3.02±.40	**0.022**
Percentage of ostial LAD stenosis, %	85.38±12.63	86.09±13.55	0.358
Thrombus identified by angiography, n (%)	77 (13.1)	82 (14.2)	0.563
LM stenosis <30%, n (%)	91 (15.4)	119 (20.6)	**0.021**
SYNTAX Score	19.25±8.25	20.69±7.85	**0.002**
Intra-aortic balloon pump support, n (%)	10 (1.7)	9 (1.6)	1.000
Performed IVUS, n (%)	139 (23.6)	145 (25.1)	0.532
Rotablator use, n (%)	10 (1.7)	16 (2.8)	0.212
Thrombus Aspiration, n (%)	26 (4.4)	30 (5.2)	0.526
Direct stenting, n (%)	62 (10.5)	52 (9.0)	0.389
Second or third generation stent			
Everolimus-eluting	186 (31.5)	189 (32.8)	0.653
Zotarolimus-eluting	60 (10.2)	76 (13.2)	0.110
Sirolimus-eluting	344 (58.3)	312 (54.1)	0.145
Stent diameter, mm	3.31±.35	3.53±.64	**<0.001**
Total stent length, mm	24.60±7.57	25.81±7.22	**0.005**
Maximum post-dilatation balloon diameter, mm	3.67±.31	4.57±.39	**<0.001**
SB protection, n(%)			
Jailed wire technique	36 (6.1)	529 (91.7)	**<0.001**
Jailed balloon technique	6 (1.0)	18 (3.1)	**0.013**
SB narrowing >50%, n (%)	59 (10.0)	126 (21.8)	**<0.001**
Side branch intervention, n (%)			
POT-kissing-POT	29 (4.9)	97 (16.8)	**<0.001**
POT-side-POT	2 (0.3)	16 (2.8)	**<0.001**
Bailout 2-stent technique	16 (2.7)	59 (10.2)	**<0.001**
TAP	13 (81.3)	38 (65.5)	0.229
Culotte	3 (18.8)	20 (34.5)	0.361
Tirofiban use during PCI, n (%)	65 (11.0)	54 (9.4)	0.349
Contrast media volume (mL)	179.40±77.96	192.53±84.80	**0.006**
Procedure time, min	44.23±15.34	46.58±18.53	**0.018**
Fluoroscopy time, min	15.98±7.32	18.74±7.49	**<0.001**

Abbreviations: Fr: French, IVUS: Intravascular ultrasound, LAD: Left anterior descending, LCx: Left circumflex, LM: Left main coronary artery, PCI: Percutaneous coronary intervention, POT: Proximal optimization technique, SB: Side-branch

Table 3. Clinical outcomes of per study group

Variables	AOS (n=590)	COS (n=577)	P value
In-hospital complications, n(%)			
Death	5 (0.8)	3 (0.5)	0.726
Fatal ventricular arrhythmias (Sustained VT/VF)	25 (4.2)	22 (3.8)	0.712
Contrast-induced AKI	74 (12.5)	57 (9.9)	0.150
Psodoaneurysm	12 (2.0)	14 (2.4)	0.650
Follow-up time, months	30.70±23.78	31.59±24.10	0.524
Mid-term Outcomes, n(%)			
Primary end-point (MACE)	107 (18.1)	55 (9.5)	**<0.001**
Cardiac death	48 (8.1)	31 (5.4)	0.060
TLR	63 (10.7)	24 (4.2)	**<0.001**
TVMI	54 (9.2)	27 (4.7)	**0.003**
Secondary end-point (MACCE), n (%)	141 (23.9)	89 (14.4)	**<0.001**
All-cause death	74 (12.5)	56 (9.7)	0.124
TVR	83 (14.1)	34 (5.9)	**<0.001**
TVMI	54 (9.2)	27 (4.7)	**0.003**
TIMI-major bleeding	10 (1.7)	13 (2.3)	0.493
Stent thrombosis	20 (3.4)	12 (2.1)	0.171
Stroke	6 (1.0)	4 (0.7)	0.753

Abbreviations: AKI: Acute kidney injury, AOS: Accurate ostial stenting, COS: Crossover stenting, MACE: Major adverse cardiac events, MACCE: Major adverse cardiovascular and cerebral events, TLR: Target lesion revascularization, TIMI: Thrombolysis in myocardial infarction, TVMI: Target vessel myocardial infarction, TVR: Target vessel revascularization

Table 4. Univariate and multivariate Cox regression analysis showing independent predictors of MACE in patients with ostial LAD stenosis

Variables	Univariate Level		Multivariate Level	
	HR (95% CI)	P	HR (95% CI)	P
Treatment (AOS)	1.967 [1.420-2.723]	<0.001	2.469 [1.749-3.485]	**<0.001**
Age	1.019 [1.007-1.032]	0.002	1.012 [0.998-1.025]	0.098
Diabetes mellitus	1.011 [0.739-1.381]	0.946	1.234 [0.890-1.712]	0.206
Chronic kidney disease	2.035 [1.410-2.937]	<0.001	1.832 [1.236-2.715]	**0.003**
Current smoker	1.001 [0.744-1.347]	0.994	1.140 [.825-1.574]	0.427
Clinical presentation (STEMI)	2.030 [1.479-2.788]	<0.001	1.283 [0.856-1.921]	0.227
LV ejection fraction	0.962 [0.949-0.975]	<0.001	0.984 [0.969-0.999]	**0.042**
Potent P2Y12 receptor inhibitors	0.887 [0.651-1.208]	0.446	0.933 [0.672-1.297]	0.680
SYNTAX score	1.090 [1.070-1.111]	<0.001	1.089 [1.067-1.112]	**<0.001**
Performed IVUS	0.633 [0.410-0.977]	0.039	0.689 [0.441-1.077]	**0.049**
Direct stenting	1.547 [.994-2.406]	0.049	2.171 [1.358-3.470]	**0.001**
Total stent length	1.044 [1.024-1.064]	<0.001	1.036 [1.015-1.058]	**0.001**
Stent diameter	0.799 [0.634-1.007]	0.057	0.845 [0.605-1.180]	0.324
Percentage of ostial LAD stenosis	1.021 [1.007-1.036]	0.003	1.016 [1.001-1.031]	**0.042**
LM stenosis <30%	0.998 [0.668-1.492]	0.994	1.385 [.912-2.103]	0.127
Access site (Radial)	0.828 [0.527-1.300]	0.413	0.685 [0.431-1.090]	0.111

Abbreviations: AOS: Accurate ostial stenting, CI: Confidence Interval, HR: Hazard ratio, IVUS: Intravascular ultrasound, LAD: Left anterior descending artery, LM: Left main coronary artery, LV: Left ventricle, MACE: Major adverse cardiac eventsn, STEMI: ST-segment elevation myocardial infarction

Table 5. Univariate and multivariate Cox regression analysis showing independent predictors of MACCE in patients with ostial LAD stenosis

Variables	Univariate Level		Multivariate Level	
	HR (95% CI)	P	HR (95% CI)	P
Treatment (AOS)	1.586 [1.216-2.068]	0.001	1.894 [1.432-2.507]	**<0.001**
Age	1.021 [1.010-1.031]	<0.001	1.012 [1.000-1.023]	**0.046**
Diabetes mellitus	0.908 [0.699-1.177]	0.469	1.118 [0.853-1.468]	0.417
Chronic kidney disease	1.782 [1.293-2.455]	<0.001	1.522 [1.080-2.144]	**0.016**
Current smoker	0.843 [0.654-1.085]	0.184	0.958 [0.731-1.256]	0.758
Clinical presentation (STEMI)	1.592 [1.208-2.097]	0.001	1.129 [0.805-1.585]	0.482
LV ejection fraction	0.966 [0.955-0.976]	<0.001	0.979 [0.967-0.992]	**0.001**
Potent P2Y12 receptor inhibitors	0.774 [0.597-1.002]	0.052	0.816 [0.620-1.073]	0.145
SYNTAX score	1.073 [1.057-1.091]	<0.001	1.070 [1.052-1.089]	**<0.001**
Performed IVUS	0.866 [0.621-1.208]	0.048	0.949 [0.674-1.337]	**0.042**
Direct stenting	1.492 [1.027-2.169]	0.036	1.921 [1.296-2.846]	**0.001**
Total stent length	1.034 [1.017-1.052]	<0.001	1.027 [1.009-1.045]	**0.004**
Stent diameter	0.836 [0.681-1.027]	0.088	0.861 [0.658-1.126]	0.275
Percentage of ostial LAD stenosis	1.012 [1.001-1.024]	0.028	1.009 [0.997-1.021]	0.154
LM stenosis <30%	0.964 [0.685-1.356]	0.832	1.233 [0.866-1.755]	0.245
Access site (Radial)	0.869 [0.588-1.285]	0.485	0.780 [0.522-1.165]	0.225

Abbreviations: AOS: Accurate ostial stenting, CI: Confidence Interval, HR: Hazard ratio, IVUS: Intravascular ultrasound, LAD: Left anterior descending artery, LM: Left main coronary artery, LV: Left ventricle, MACCE: Major adverse cardiac and cerebral events, STEMI: ST-segment elevation myocardial infarction

OP-060 [Interventional Cardiology / Coronary]

Mini-crush or nano-crush stenting technique for complex coronary bifurcation lesions: The multicenter MINANO registry

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Background and Aim: This multicenter study aimed to retrospectively evaluate the mid-term clinical outcomes of mini-crush (MCT) and nano-crush (NCT) techniques in patients with complex bifurcation lesions (CBLs).

Methods: The study comprised 431 patients [male: 318 (73.8%), mean age: 60.96 ± 10.34 years] who underwent bifurcation PCI between January 2018 and December 2023 were included in the study from six tertiary centers. The primary endpoint was defined as the major cardiovascular events (MACE), which include cardiac death, target vessel myocardial infarction (TVMI), or clinically driven target lesion revascularization (TLR). This is the first study to compare the clinical outcomes of MCT and NCT in patients with CBL.

Results: The initial revascularization strategy was MCT in 302 (70%) cases and NCT in 129 (30%) patients. SYNTAX scores [24.33 ± 6.54 vs. 24.43 ± 5.45, p=0.707] were comparable in both groups. The incidence of MACE (18.6 vs. 10.9%, p=0.031), TVMI (11.6 vs. 5.6%, p=0.030), and clinically driven TLR (14 vs. 6%, p=0.006) were significantly higher in the NCT group compared to the MCT group. Being in the MCT group (HR: 0.549, p=0.035), high SYNTAX score (HR: 1.105, p<0.001), non-fatal intra-procedural complications (HR:

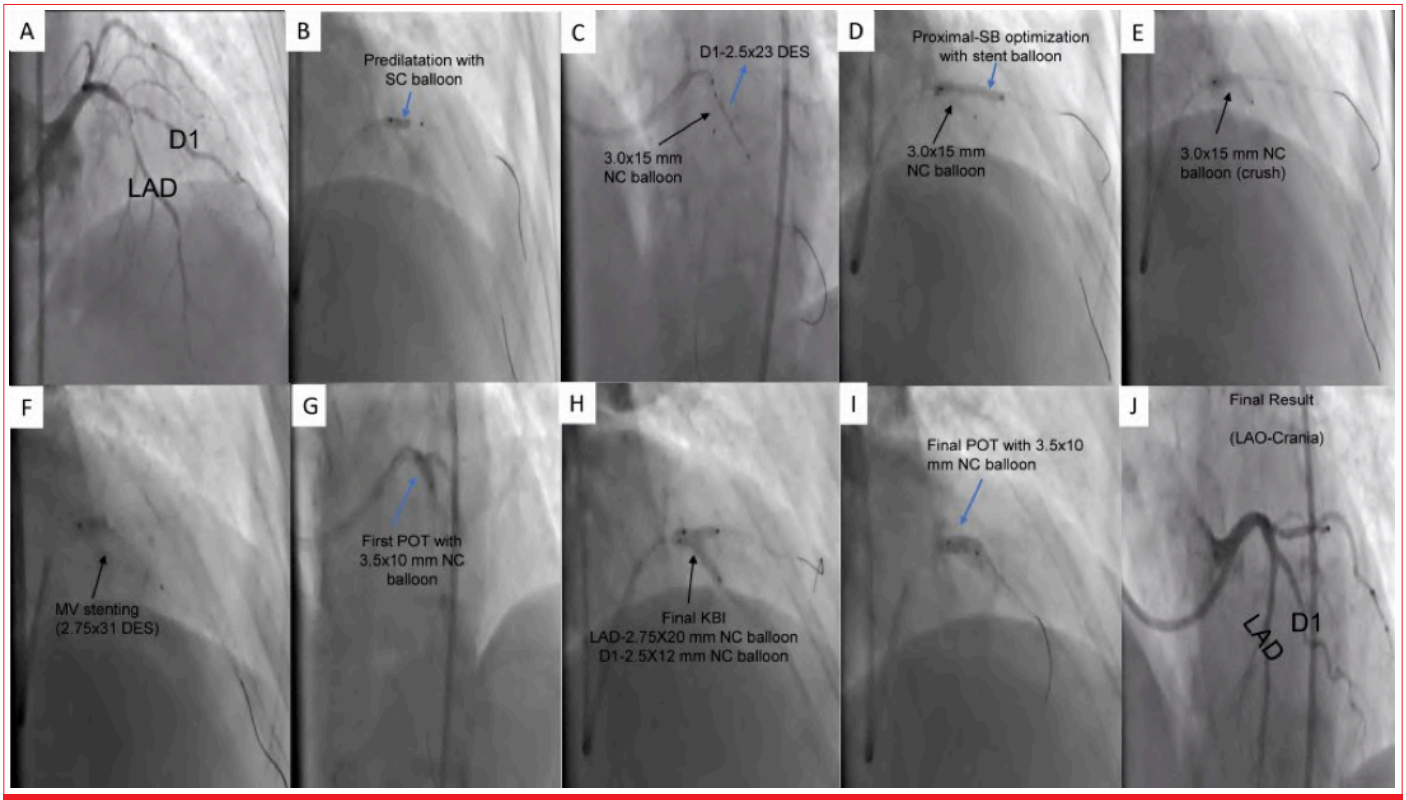

Figure 1. Step-by-step mini-crush technique. A: Bifurcation localization in the LAD-D1, B: Wiring and pre-dilatation of both branch and stenting distal LAD lesion, C: SB stent positioning and placement with an uninflated NC balloon in the MV, D: SB proximal optimization with stent balloon. E: Balloon crushing of the SB stent, F: Positioning and placement of the MV stent after removing the balloon and stent from the SB, G: First POT with NC balloon, H, I: Final KBI and POT with the NC balloons, J: Final result. DES: Drug-eluting stent; D1: First diagonal artery; KBI: Kissing balloon inflation; LAD: Left anterior descending artery; LAO: Left anterior oblique view; MV: Main vessel; NC: Non-Compliant; POT: Proximal optimization technique; SB: Side branch; SC: Semi-compliant.

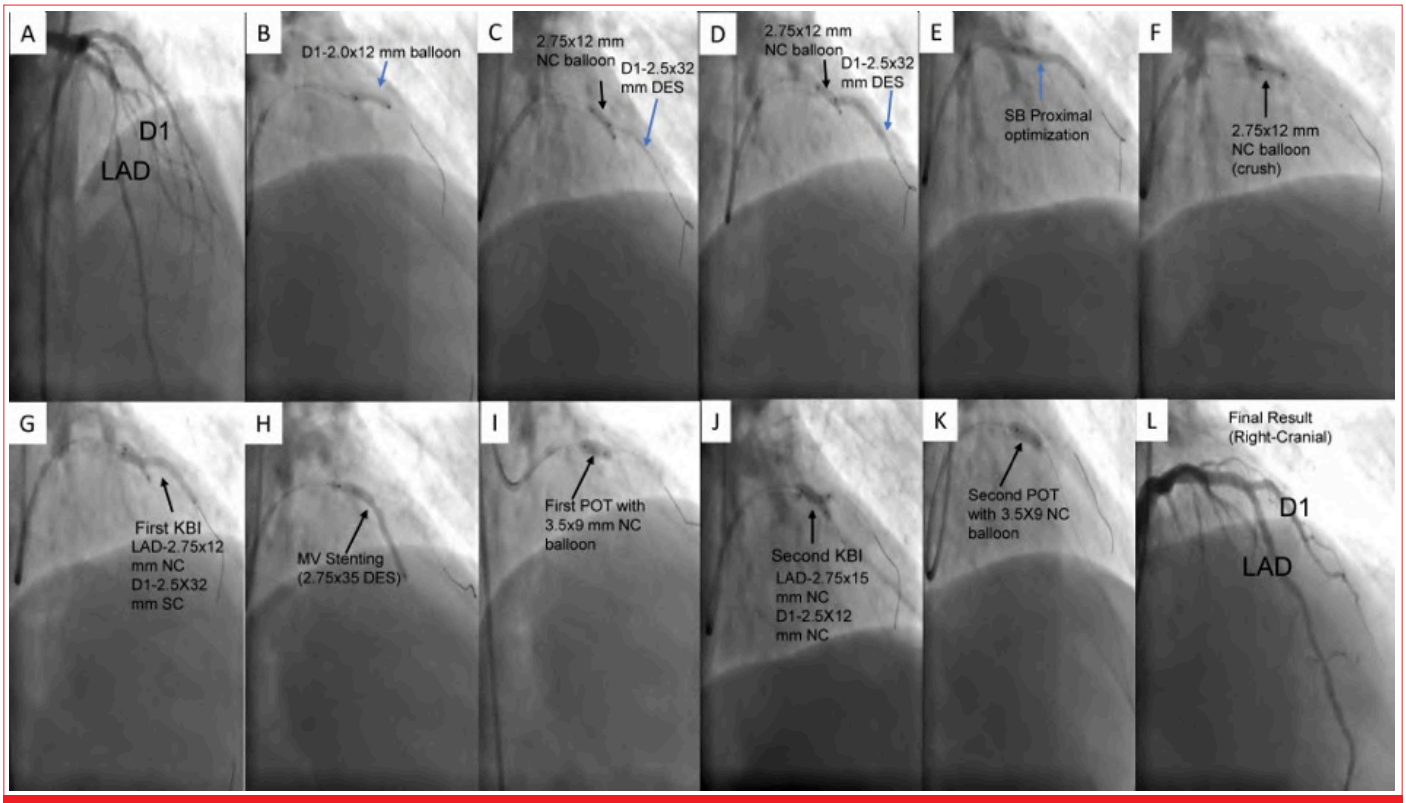

Figure 2. Step-by-step nano-cush technique. A: Vessel anatomy showing bifurcation lesion in the LAD-D1, B: Wiring and pre-dilatation of both branches, C,D: SB stent positioning with an inflated NC balloon in the MV, E: SB proximal optimization with stent balloon, F: Balloon crushing of the SB stent, G: First KBI with the stent balloon and an NC balloon, H: Positioning of the MV stent after removing the balloon and stent from the SB, I: First POT with NC balloon, J, K: Second KBI and POT with the NC balloons, L: Final result. DES: Drug-eluting stent; D1: First diagonal artery; KBI: Kissing balloon inflation; LAD: Left anterior descending artery; MV: Main vessel; NC: Non-compliant; POT: Proximal optimization technique; SB: Side branch; SC: Semi-compliant.

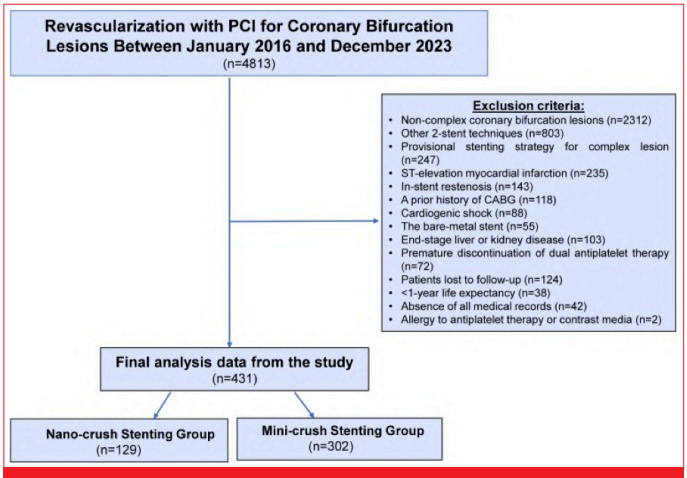

Figure 3. Flow chart for patient selection.

3.269, p<0.001), proximal side-branch optimization (HR: 0.451, p=0.013), diabetes mellitus (HR: 2.263, p=0.009), and chronic kidney disease (HR: 1.948, p=0.024) were found to be independent predictors of MACE.

Conclusions: This non-randomized study suggests that MCT was associated with better mid-term MACE, TVMI, and clinically driven TLR rates compared with NCT in patients with CBLs.

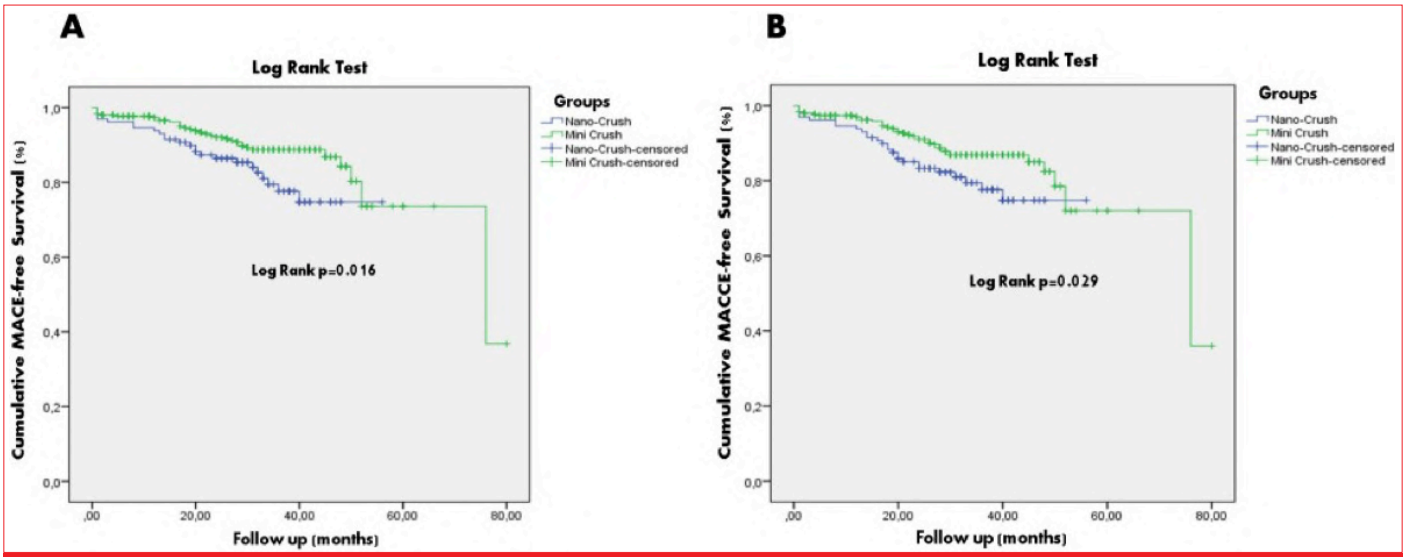

Figure 4. Kaplan-Meier survival analysis for primary endpoint (MACE) (A) and secondary endpoint (MACCE) (B) during follow-up. MACE: Major adverse cardiovascular events; MACCE: Major adverse cardiovascular and cerebral events.

Table 1. The key differences between mini-crush and nano-crush techniques.

	Mini-crush (Contemporary)	Nano-crush (Ray S, et al) (Contemporary)
Guiding catheter (Fr)	6 or 7	6
MV and SB stent positioning	No/MV balloon-SB stent	No/MV balloon is smaller than the reference vessel diameter and is inflated before SB stent deployment to assist in the positioning of the SB stent.
Protrusion length of SB stent into the MV	1-3 mm	0.5-1 mm
Removal of SB wire before crushing	No	No
Proximal SB optimization	Strong recommendation	Strong recommendation
Removal of SB stent balloon before crushing	Yes	No/ Slightly pullback of the deflated SB stent balloon after proximal SB optimization
Crushing	MV-NC balloon	MV-NC balloon
Rewiring before first KBI	No	No
First KBI	No	Yes (SB with stent balloon)
Rewiring before final KBI	Yes	Yes
Final KBI	Yes (both branches with NC balloons)	Yes (both branches with NC balloons)
Final POT	Strong recommendation	Strong recommendation
Bifurcation angle	Easy rewiring at narrow angles Excessive main vessel protrusion risk at narrow-angles	Risk of a geographic miss at narrow-angles Optimum coverage of ostial of SB stent at wide-angles
Resource utilization	$	$

Table 2. Baseline characteristics of per study group

Variables	Nano-crush Group (n=129)	Mini-crush Group (n=302)	P value
Age (years)	61.5±8.89	60.73±10.91	0.438
Gender, male, n (%)	91 (70.5)	227 (75.2)	0.318
Comorbidities, n(%)			
Hypertension	87 (67.4)	201 (66.6)	0.858
Diabetes Mellitus	53 (41.1)	112 (37.1)	0.434
Hyperlipidemia	80 (62.0)	174 (57.6)	0.395
Chronic kidney disease	25 (19.4)	62 (20.5)	0.785
Current Smoker	65 (50.4)	154 (51.0)	0.908
History of Stroke	3 (2.3)	5 (1.7)	0.701
Prior PCI	53 (41.1)	109 (36.1)	0.327
Prior MI	41 (31.8)	100 (33.1)	0.788
Heart Failure	22 (17.1)	44 (14.6)	0.512
LV Ejection Fraction (%)	54.82±9.43	53.44±10.08	0.174
Moderate-severe Valve Disease, n (%)	17 (13.2)	27 (8.9)	0.183
Laboratory measurements			
White blood cell count, (10^3/L)	9.41±2.8	9.37±2.93	0.673
Hemoglobin, (g/dL)	13.33±2.07	13.52±1.93	0.411
Platelet count, (10^3/L)	243.84±74.29	246.18±66.33	0.675
Creatinine, (mg/dL)	1.06±0.72	1±0.62	0.741
Total cholesterol, (mg/dL)	179.14±56.16	187.15±54.09	0.106
Clinical Presentation, n (%)			
CCS	58 (45.0)	122 (40.4)	0.379
NSTEMI	59 (45.7)	154 (51.0)	0.317
USAP	12 (9.3)	26 (8.6)	0.816
Medications Used, n (%)			
Acetylsalicylic acid	129 (100)	302 (100)	-
Clopidogrel	61 (47.3)	135 (44.7)	0.622
Ticagrelor	57 (44.2)	145 (48.0)	0.466
Prasugrel	11 (8.5)	22 (7.3)	0.657
Beta Blockers	123 (95.3)	283 (93.7)	0.505
CCB	20 (15.5)	34 (11.3)	0.223
ACEI/ARB	101 (78.3)	250 (82.8)	0.273
Statin	123 (95.3)	290 (96.0)	0.747
Diuretics	23 (17.8)	59 (19.5)	0.679
Insulin	33 (25.6)	69 (22.8)	0.541

ACEI: Angiotensin-converting enzyme inhibitor; ARB: Angiotensin receptor blocker; CCB: Calcium channel blocker; CCS: Chronic coronary syndrome, LV: Left ventricle; MI: Myocardial infarction; NSTEMI: Non-ST segment elevation myocardial infarction; PCI: Percutaneous coronary intervention; USAP: Unstable angina pectoris.

Table 3. Lesions characteristics per study group

Parameters	Nano-crush Group (n=129)	Mini-crush Group (n=302)	P value
Multi-vessel disease, n(%)	89 (69.0)	211 (69.9)	0.856
SYNTAX score	24.33±6.54	24.43±5.45	0.707
SYNTAX score ≤22, n(%)	54 (41.9)	133 (44.0)	0.676
SYNTAX score 23-32, n(%)	51 (39.5)	126 (41.7)	0.673
SYNTAX score ≥33, n(%)	24 (18.6)	43 (14.2)	0.252
Locations of bifurcation lesions, n(%)			
LMCA	13 (10.1)	24 (7.9)	0.470
LAD-Diagonal	78 (60.5)	203 (67.2)	0.178
LCx-OM	32 (24.8)	65 (21.5)	0.455
PDA-PL	6 (4.7)	10 (3.3)	0.579
Type of Medina classification, n(%)			
0.1.1	39 (30.2)	84 (27.8)	0.611
1.1.1	91 (70.5)	218 (72.2)	0.729
Reference vessel diameter, mm			
MV	3.16±0.45	2.97±0.48	0.417
SB	2.62±0.27	2.59±0.23	0.397
SB reference vessel diameter ≥2.5 mm, n(%)	108 (83.7)	264 (87.4)	0.307
Bifurcation angle (°)	66.41±48.25	63.36±45.08	0.076
Assessment of complex bifurcation lesions, n(%)			
Moderate or severe calcification	43 (33.3)	98 (32.5)	0.858
MV reference diameter < 2.5mm	1 (0.8)	4 (1.3)	1.00
Multiple lesions	95 (73.6)	210 (69.5)	0.391
Bifurcation angle <45° or >70°	66 (51.2)	149 (49.3)	0.729
Thrombus identified by angiography	15 (11.6)	38 (12.6)	0.782
Lesion length, mm			
MV	26.85±8.54	25.24±8.8	**0.033**
SB	16.89±5.27	16.78±6.44	0.388
MV, n(%)			
TIMI flow grade <3	14 (10.9)	32 (10.6)	0.937
Chronic total occlusion	4 (3.1)	9 (3.0)	1.00
Thrombus-containing lesion	6 (4.7)	20 (6.6)	0.513
SB, n(%)			
TIMI flow grade <3	9 (7.0)	38 (12.6)	0.094
Chronic total occlusion	3 (2.3)	2 (0.7)	0.161
Thrombus-containing lesion	7 (5.4)	23 (7.6)	0.536

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

Table 4. Procedural characteristics and in-hospital complications of the study groups

Parameters	Nano-crush group (n=129)	Mini-crush group (n=302)	P value
Access site, n(%)			
Femoral	125 (96.9)	291 (96.4)	0.779
Radial	4 (3.1)	11 (3.6)	1.00
Tirofiban use during PCI, n (%)	11 (8.5)	40 (13.2)	0.165
Performed IVUS, n(%)	10 (7.8)	18 (6.0)	0.489
Thrombus Aspiration, n(%)	1 (0.8)	2 (0.7)	1.000
Pre-dilation			
MV	109 (84.5)	253 (83.8)	0.852
SB	108 (83.7)	263 (87.1)	0.355
Final kissing balloon inflation	126 (97.7)	295 (97.7)	0.996
MV			
Stent number, n	1.19±0.39	1.18±0.39	0.923
Stent diameter, mm	3.12±0.42	2.97±0.45	**0.015**
Stent length, mm	31.49±7.97	30.64±9.65	0.113
SB			
Stent number, n	1.02±0.15	1.05±0.21	0.260
Stent diameter, mm	2.64±0.28	2.45±0.64	**0.021**
Stent length, mm	21.15±6.14	21.24±6.83	0.781
Proximal side-branch optimization, n (%)	106 (82.2)	253 (83.8)	0.683
Final POT, n (%)	126 (97.7)	287 (95.0)	0.209
Resource utilization, n (%)			
Guiding catheter number	1.12±0.51	1.07±0.25	0.472
Guidewire number	2.47±0.73	2.73±0.74	**0.001**
Balloon number	5.98±1.81	5.82±2.15	0.096
Intraprocedural complication, n(%)	14 (10.9)	45 (14.9)	0.263
Abrupt occlusion			
MV	1 (0.8)	6 (2.0)	0.680
SB	2 (1.6)	8 (2.6)	0.730
TIMI<3			
MV	3 (2.3)	11 (3.6)	0.568
SB	4 (3.1)	9 (3.0)	1.00
Dissection			
MV	4 (3.1)	11 (3.6)	1.00
SB	3 (2.3)	7 (2.3)	1.00
Thrombus formation			
MV	2 (1.6)	3 (1.0)	0.638
SB	4 (3.1)	10 (3.3)	1.00
Coronary Perforation			
MV	0 (0.0)	1 (0.3)	1.00
SB	0 (0.0)	1 (0.3)	1.00
Geographic miss of the SB ostium, n(%)*	1 (10)	0 (0)	0.333
Procedure time, min	59.84±21.69	62.46±18.72	0.053
Fluoroscopy time, min	20.74±6.33	20.50±6.70	0.509
Contrast media volume (mL)	200.08±63.88	205.41±69.26	0.524
Angiographic success, n(%)			
MV	127 (98.4)	294 (97.4)	0.488
SB	126 (97.7)	294 (97.4)	0.845
In-hospital complications, n (%)			
Death	2 (1.6)	5 (1.7)	1.00
Major bleeding	4 (3.1)	6 (2.0)	0.495
Pseudoaneurysm	3 (2.3)	11 (3.6)	0.568
Malignant ventricular arrhythmias	2 (1.6)	7 (2.3)	0.731
Stent thrombosis	2 (1.6)	2 (0.7)	0.586
Target vessel myocardial infarction	2 (1.6)	3 (1.0)	0.638
Contrast-induced AKI	11 (8.5)	31 (10.3)	0.577

* "n" indicates the number of patients with bifurcation lesions who underwent intravascular imaging. AKI: Acute kidney injury; IVUS: Intravascular ultrasound; PCI: Percutaneous coronary intervention; MV: Main vessel; POT: Proximal optimization technique; SB: Side branch; TIMI: Thrombolysis in myocardial infarction.

Table 5. Clinical outcomes per study group

Parameters	Nano-crush Group (n=129)	Mini-crush Group (n=302)	P value
Follow-up time, month	29.67±10.93	30.86±14.73	0.351
Primary end-point (MACE), n (%)	24 (18.6)	33 (10.9)	**0.031**
Cardiac death	6 (4.7)	15 (5.0)	1.00
Target vessel myocardial infarction	15 (11.6)	17 (5.6)	**0.030**
Target lesion revascularization	18 (14.0)	18 (6.0)	**0.006**
Secondary end-point (MACCE), n (%)	26 (20.6)	38 (12.6)	**0.043**
All-cause death	7 (5.4)	17 (5.6)	1.00
Target vessel myocardial infarction	15 (11.6)	17 (5.6)	**0.030**
Target lesion revascularization	18 (14.0)	18 (6.0)	**0.006**
Stent thrombosis	7 (5.4)	6 (2.0)	0.068
Stroke	1 (0.8)	3 (1.0)	1.00

MACE: Major adverse cardiovascular events; MACCE: Major adverse cardiovascular and cerebral events.

OP-061 [Interventional Cardiology / Coronary]

Double kissing culotte or mini-crush stenting for true coronary bifurcation lesions: The multicenter COLLECT-BIF registry

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Background and Aim: This multicenter observational study aimed to retrospectively evaluate the mid-term clinical outcomes of the mini-crush technique (MCT) and double kissing culotte technique (DKCT) in patients with coronary bifurcation lesions (CBLs).

Methods: This large-scale, multicenter study (n=8) included patients with CBLs who underwent percutaneous coronary intervention with either MCT or DKCT. The primary endpoint was defined as major adverse cardiac events (MACE), which include cardiac death, target vessel myocardial infarction, or clinically driven target lesion revascularization.

Results: A total of 728 patients [male: 584 (80.2%), mean age: 60.93 ± 10.46 years] were included. The initial revascularization strategy was MCT in 476 (65.4%) cases and DKCT in 252 (34.6%) cases. The number of balloons used (6.30 ± 1.84 vs. 5.43 ± 1.99, p<0.001) and procedure time (65.10 ± 20.34 vs. 61.30 ± 18.48 min, p=0.020) were significantly higher in the DKCT group. In multivariate Cox regression analysis, risk-adjusted mid-term MACE (hazard ratio (HR): 0.645, [95%CI: 0.395-1.053], p=0.079) did not differ in the MCT group compared to the DKCT group. Additionally, chronic kidney disease (HR=2.434, p<0.001), high SYNTAX score (HR=1.085, p<0.001), final KBI (HR=0.110, p<0.001), presence of non-fatal intraprocedural complications (HR: 5.818, p<0.001), and high total cholesterol level (HR: 1.007, p=0.005) were found to be independent predictors of MACE.

Conclusions: This multicenter registry demonstrates that in patients with CBLs, the risk-adjusted MACE rate was similar between both techniques with a non-significant trend favoring MCT at mid-term follow-up.

Table 1. Baseline characteristics of per study group (n=728)

Variables	Mini-crush group (*n* = 476)	DK-culotte group (*n* = 252)	*P* value
Age, mean ± SD (years)	61.43 ± 10.46	59.98 ± 10.44	0.077
Gender, male, *n* (%)	376 (79.0)	208 (82.5)	0.253
Comorbidities, *n* (%)			
Hypertension	270 (56.7)	138 (54.8)	0.612
Diabetes	182 (38.2)	92 (36.5)	0.647
Hyperlipidemia	224 (47.1)	116 (46.0)	0.792
Chronic kidney disease	82 (17.2)	35 (13.9)	0.243
Current smoker	260 (54.6)	144 (57.4)	0.478
History of stroke	26 (5.5)	4 (1.6)	**0.011**
Prior PCI	155 (32.6)	74 (29.4)	0.377
Heart failure	79 (16.6)	36 (14.3)	0.416
LV ejection fraction (%)	53.23 ± 10.12	53.40 ± 9.28	0.725
Moderate-severe valve disease, *n* (%)	42 (8.8)	17 (6.7)	0.328
Laboratory measurements, mean ± SD			
White blood cell count, (10^9/l)	9.33 ± 2.69	9.32 ± 2.86	0.789
Hemoglobin, (g/dl)	13.03 ± 1.81	13.37 ± 2.07	**0.002**
Platelet count, (10^9/l)	245.33 ± 57.40	250.09 ± 71.40	0.737
Creatinine, (mg/dl)	0.98 ± 0.54	0.96 ± 0.45	0.705
Total cholesterol, (mg/dl)	191.06 ± 50.12	184.33 ± 48.55	**0.018**
Clinical presentation, *n* (%)			0.460
CCS	167 (35.1)	92 (36.5)	
NSTEMI	249 (52.3)	122 (48.4)	
USAP	60 (12.6)	38 (15.1)	
Medications used, *n* (%)			
Acetylsalicylic acid	476 (100.0)	252 (100.0)	–
P2Y12 receptor inhibitors			0.499
Clopidogrel	232 (48.7)	131 (52.0)	
Ticagrelor	213 (44.7)	108 (42.9)	
Prasugrel	31 (6.5)	13 (5.2)	
Beta blockers	441 (92.6)	231 (91.7)	0.637
CCB	76 (16.0)	41 (16.3)	0.916
ACEI/ARB	397 (83.4)	207 (82.1)	0.667
Statin	453 (95.2)	243 (96.4)	0.430
Diuretics	70 (14.7)	25 (9.9)	0.068
Insulin	78 (16.4)	34 (13.5)	0.303

Bold indicates significance level at p<0.05. ACEI: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker; CCB: Calcium channel blocker; CCS: Chronic coronary syndrome; DK: Double kissing; LV: Left ventricle; MI: Myocardial infarction; NSTEMI: Non-ST segment elevation myocardial infarction; PCI: Percutaneous coronary intervention; USAP: Unstable angina pectoris.

Table 2. Lesions characteristics per study group (n=728)

Parameters	Mini-crush group (n = 476)	DK-culotte group (n = 252)	P value
Multivessel disease, n (%)	246 (51.7)	120 (47.6)	0.297
SYNTAX score, mean ± SD	20.40 ± 7.43	19.20 ± 6.63	**0.032**
SYNTAX score, n (%)			0.248
≤22	298 (62.6)	172 (68.3)	
23–32	133 (27.9)	57 (22.6)	
≥33	45 (9.5)	20 (7.9)	
Locations of bifurcation lesions, n (%)			0.696
LMCA	37 (7.8)	18 (7.1)	
LAD-Diagonal	287 (60.3)	153 (60.7)	
LCx-OM	125 (26.3)	70 (27.8)	
PDA-PL	27 (5.7)	11 (4.4)	
Type of medina classification, n (%)			0.420
1.0.1	89 (18.7)	46 (18.3)	
0.1.1	160 (33.6)	70 (27.8)	
1.1.1	227 (47.7)	131 (52.0)	
Reference vessel diameter, mean ± SD (mm)			
MV	3.07 ± 0.49	3.17 ± 0.40	**0.008**
SB	2.64 ± 0.26	2.73 ± 0.29	**<0.001**
SB reference vessel diameter ≥2.5 mm, n (%)	423 (88.9)	231 (91.7)	0.234
Complexity of bifurcation lesions, n (%)			
SB diameter stenosis 70 or 90%	386 (81.1)	199 (79.0)	0.493
Moderate or severe calcification	109 (22.9)	48 (19.0)	0.229
MV reference diameter <2.5 mm	12 (2.5)	11 (4.4)	0.176
Multiple lesions	249 (52.3)	121 (48.0)	0.270
Bifurcation angle <45° or >70°	232 (48.7)	109 (43.3)	0.158
Thrombus identified by angiography	52 (10.9)	20 (7.9)	0.199
Lesion length, mean ± SD (mm)			
MV	22.01 ± 8.95	22.30 ± 8.77	0.732
SB	14.62 ± 5.90	15.21 ± 7.67	0.746
MV, n (%)			
TIMI flow grade <3	59 (12.4)	27 (10.7)	0.504
Chronic total occlusion	19 (4.0)	3 (1.2)	**0.040**
Thrombus-containing lesion	31 (6.5)	14 (5.6)	0.610
SB, n (%)			
TIMI flow grade <3	50 (10.5)	18 (7.1)	0.138
Chronic total occlusion	2 (0.4)	2 (0.8)	0.612
Thrombus-containing lesion	28 (5.9)	13 (5.2)	0.687

Bold indicates significance level at p<0.05. DK: Double kissing; LAD: Left anterior descending; LCx: Left circumflex; LMCA: Left main coronary artery; MV: Main vessel; OM: Obtuse marginal artery; PDA: Posterior descending artery; PL: Posterolateral artery; SB: Side branch; TIMI: Thrombolysis in myocardial infarction.

Table 3. Procedural characteristics and in-hospital complications of the study groups (n=728)

Parameters	Mini-crush group (n = 476)	DK-culotte group (n = 252)	P value
Access site, n (%)			0.347
Femoral	465 (97.7)	249 (98.8)	
Radial	11 (2.3)	3 (1.2)	
Tirofiban use during PCI, n (%)	41 (8.6)	15 (6.0)	0.200
Performed IVUS, n (%)	34 (7.1)	19 (7.5)	0.845
Thrombus aspiration, n (%)	11 (2.3)	5 (2.0)	1.00
Predilation			
MV	399 (83.8)	209 (82.9)	0.759
SB	413 (86.8)	209 (82.9)	0.164
Proximal side-branch optimization, n (%)	412 (86.6)	218 (86.5)	0.986
Final kissing balloon inflation	467 (98.1)	252 (100.0)	**0.028**
Final POT, n (%)	458 (96.2)	247 (98.0)	0.187
MV, mean ± SD			
Stent number, n	1.14 ± 0.35	1.12 ± 0.33	0.458
Stent diameter, mm	3.01 ± 0.34	3.06 ± 0.47	**0.024**
Stent length, mm	28.31 ± 9.03	27.98 ± 9.28	0.550
SB, mean ± SD			
Stent number, n	1.03 ± 0.17	1.04 ± 0.19	0.644
Stent diameter, mm	2.51 ± 0.61	2.68 ± 0.31	**<0.001**
Stent length, mm	19.94 ± 5.59	21.31 ± 7.09	0.125
Resource utilization, mean ± SD			
Guidewire number, n	2.69 ± 0.71	2.71 ± 0.88	0.376
Balloon number, n	5.43 ± 1.99	6.30 ± 1.84	**<0.001**
Intraprocedural complication, n (%)	75 (15.8)	42 (16.7)	0.750
Abrupt occlusion			
MV	14 (2.9)	10 (4.0)	0.460
SB	11 (2.3)	5 (2.0)	1.00
TIMI < 3			
MV	23 (4.8)	18 (7.1)	0.198
SB	23 (4.8)	10 (4.0)	0.594
Dissection			
MV	21 (4.4)	17 (6.7)	0.178
SB	15 (3.2)	6 (2.4)	0.647
Thrombus formation			
MV	7 (1.5)	3 (1.2)	1.00
SB	10 (2.1)	4 (1.6)	0.781
Coronary perforation			
MV	1 (0.2)	2 (0.8)	0.276
SB	1 (0.2)	1 (0.4)	1.00
Procedure time, mean ± SD (min)	61.30 ± 18.48	65.10 ± 20.34	**0.020**
Fluoroscopy time, mean ± SD (min)	21.12 ± 6.32	23.80 ± 8.19	**<0.001**
Contrast media volume mean ± SD (ml)	217.24 ± 87.40	236.11 ± 86.98	**<0.001**
Angiographic success, n (%)			
MV	468 (98.3)	248 (98.4)	0.925
SB	468 (98.3)	248 (98.4)	0.925

Bold indicates significance level at p<0.05. AKI: Acute kidney injury; DK: Double kissing; IVUS: Intravascular ultrasound; PCI: Percutaneous coronary intervention; MV: Main vessel; POT: Proximal optimization technique; SB: Side branch; TIMI: Thrombolysis in myocardial infarction.

Table 4. In hospital and mid-term clinical outcomes per study group (n=728)

Parameters	Mini-crush group ($n=476$)	DK-culotte group ($n=252$)	Treatment effect adjusted HR (95% CI)	*P* value
In-hospital complications, *n* (%)				
Death	5 (1.1)	2 (0.8)		1.00
Major bleeding	9 (1.9)	3 (1.2)		0.558
Pseudoaneurysm	18 (3.8)	6 (2.4)		0.387
Fatal arrhythmias	9 (1.9)	4 (1.6)		1.00
Stent thrombosis	2 (0.4)	2 (0.8)		0.612
Myocardial infarction	5 (1.1)	3 (1.2)		1.00
Contrast-induced AKI	37 (7.8)	21 (8.3)		0.791
Follow-up time, mean ± SD (months)	28.91 ± 15.12	27.75 ± 11.62		0.378
Primary endpoint (MACE), *n* (%)	57 (12.0)	29 (11.5)	0.645 (0.395–1.053)	0.079*
Cardiac death	17 (3.6)	7 (2.8)		0.666
Target vessel myocardial infarction	34 (7.1)	17 (6.7)		0.842
Target lesion revascularization	31 (6.5)	24 (9.5)		0.144
Secondary endpoint (MACCE), *n* (%)	73 (15.3)	34 (13.5)	0.955 (0.621–1.469)	0.834*
All-cause death	26 (5.5)	13 (5.2)		0.863
Target vessel myocardial infarction	34 (7.1)	17 (6.7)		0.842
Target lesion revascularization	31 (6.5)	24 (9.5)		0.144
Stent thrombosis	12 (2.5)	9 (3.6)		0.420
Stroke	6 (1.3)	4 (1.6)		0.744

*Cox proportional hazard regression models risk-adjusted hazard ratio. AKI: Acute kidney injury; DK: Double kissing; MACE: Major adverse cardiac events; MACCE: Major adverse cardiac and cerebral events.

Table 5. Univariate and multivariate Cox regression analysis showing independent predictors of primary endpoint (MACE)

	Univariate level		Multivariate level	
Parameters	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Being in the MCT group	1.002 (0.638–1.574)	0.992	0.645 (0.395–1.053)	0.079
Chronic kidney disease	4.251 (2.763–6.541)	<0.001	2.434 (1.478–4.007)	**<0.001**
Prior PCI	1.861 (1.216–2.847)	0.004	1.481 (0.933–2.352)	0.096
Presentation (NSTEMI)	1.699 (1.100–2.625)	0.017	1.349 (0.857–2.125)	0.196
Total cholesterol	1.005 (1.001–1.009)	0.027	1.007 (1.002–1.011)	**0.005**
SYNTAX score	1.125 (1.093–1.157)	<0.001	1.085 (1.050–1.120)	**<0.001**
Final KBI	0.236 (0.075–0.750)	0.014	0.110 (0.033–0.371)	**<0.001**
Proximal SB optimization	0.355 (0.224–0.564)	<0.001	0.950 (0.568–1.587)	0.844
Intraprocedural complications	8.786 (5.712–13.514)	<0.001	5.818 (3.637–9.306)	**<0.001**

Bold indicates significance level at p<0.05. CI: Confidence Interval; HR: Hazard ratio; KBI: Kissing balloon inflation; MACE: Major adverse cardiovascular events; MCT: Mini-crush technique; MV: Main vessel; NSTEMI: Non ST-segment elevation myocardial infarction; PCI: Percutaneous coronary intervention; SB: Side branch.

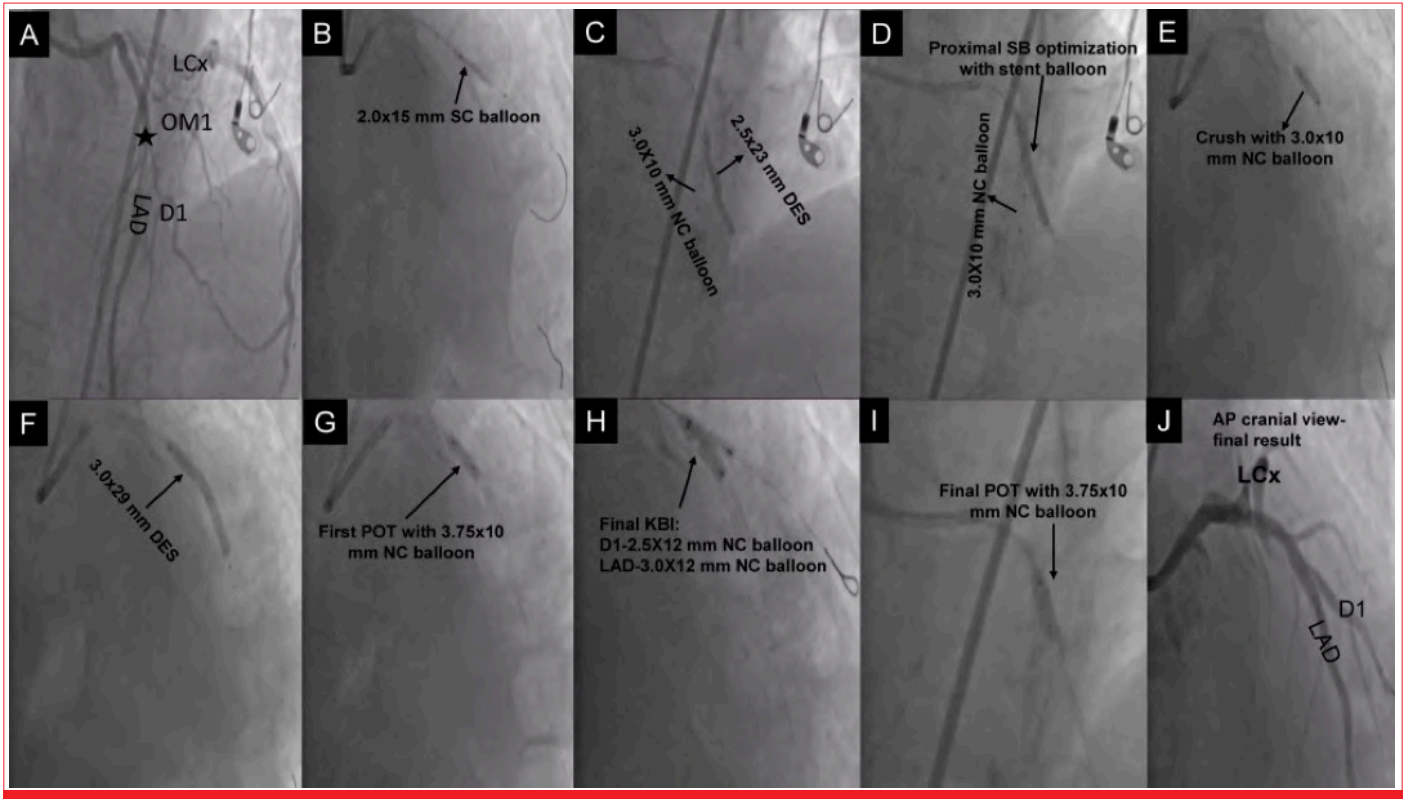

Figure 1. Step-by-step mini-crush technique. A: Bifurcation localization in the LAD-D1 arteries (black star), B: Wiring and pre-dilatation of both branch and stenting distal LAD lesion, C: SB stent positioning and placement with an uninflated NC balloon in the MV, D: SB proximal optimization with stent balloon, E: Balloon crushing of the SB stent, F: Positioning and placement of the MV stent 16 after removing the balloon and stent from the SB, G: First POT with NC balloon, H, I: Final KBI and final POT with the NC balloons, J: Final angiographic result. AP: Anteroposterior view; DES: Drug-eluting stent; D1: First diagonal artery; KBI: Kissing balloon inflation; LAD: Left anterior descending artery; LCx: Left circumflex artery; MV: Main vessel; NC: Non-compliant; OM1: First obtus marginal artery; POT: Proximal optimization technique; SB: Side branch; SC: Semi-compliant.

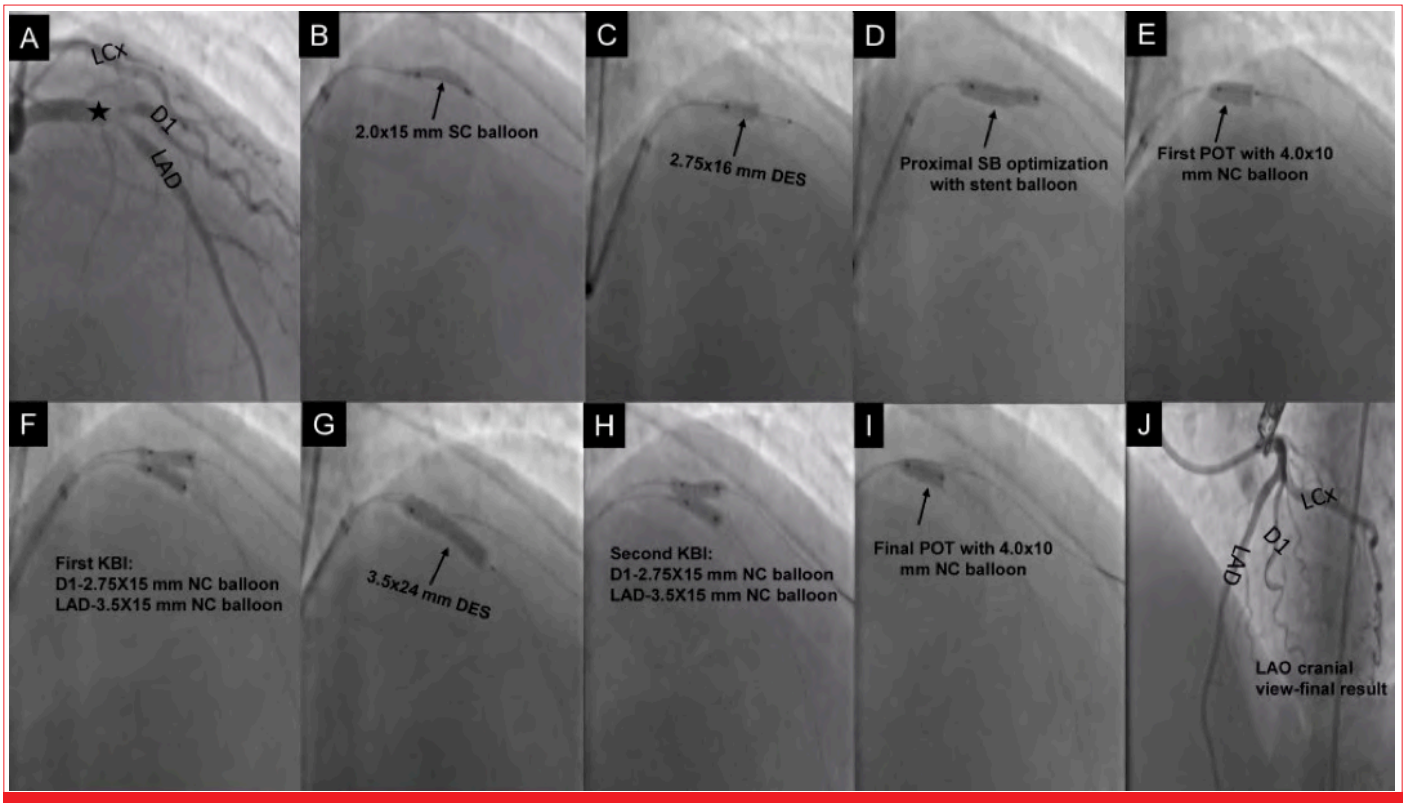

Figure 2. Step-by-step DKCT technique. A: Vessel anatomy showing bifurcation lesion in the LAD-D1 (black star), B: Wiring and predilatation may be performed for both vessels, C: The first stent is implanted from MV to SB with a minimal protrusion (2-3 mm) to the proximal MV, D, E: Proximal SB optimization and first POT, F: First KBI with NC balloons (distal cell), G: MV stent placement according to the 1:1 distal MV size, H: After second POT and rewiring to distal MV from distal cell second KBI with NC balloons, I, J: Third POT and final angiographic view. DES: Drug-eluting stent; DKCT: Double kissing culotte technique; D1: First diagonal artery; KBI: Kissing balloon inflation; LAD: Left anterior descending artery; LAO: Left anterior oblique view; LCx: Left circumflex artery; MV: Main vessel; NC: Non-compliant; POT: Proximal optimization technique; SB: Side branch; SC: Semi-compliant.

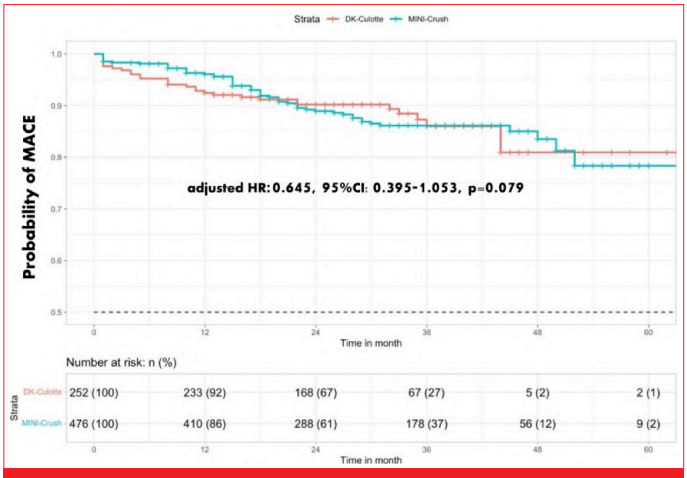

Figure 3. Kaplan-Meier survival analysis for primary endpoint (MACE) during follow-up. DK: Double kissing; MACCE: Major adverse cardiovascular and cerebral events.

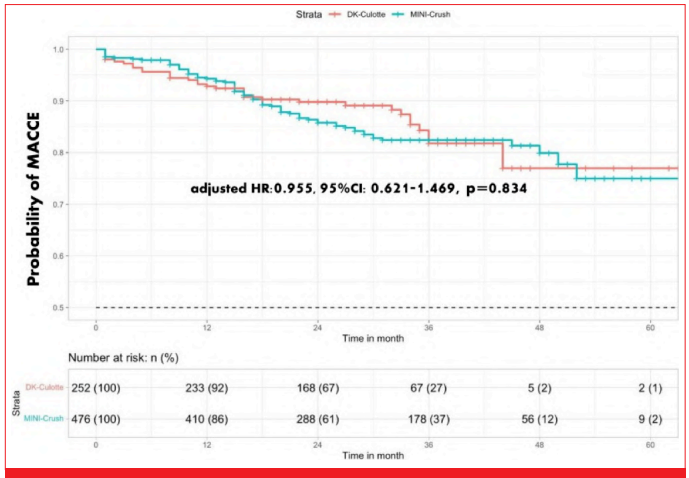

Figure 4. Kaplan-Meier survival analysis for secondary endpoint (MACCE) during follow-up. DK: Double kissing; MACCE: Major adverse cardiovascular and cerebral events.

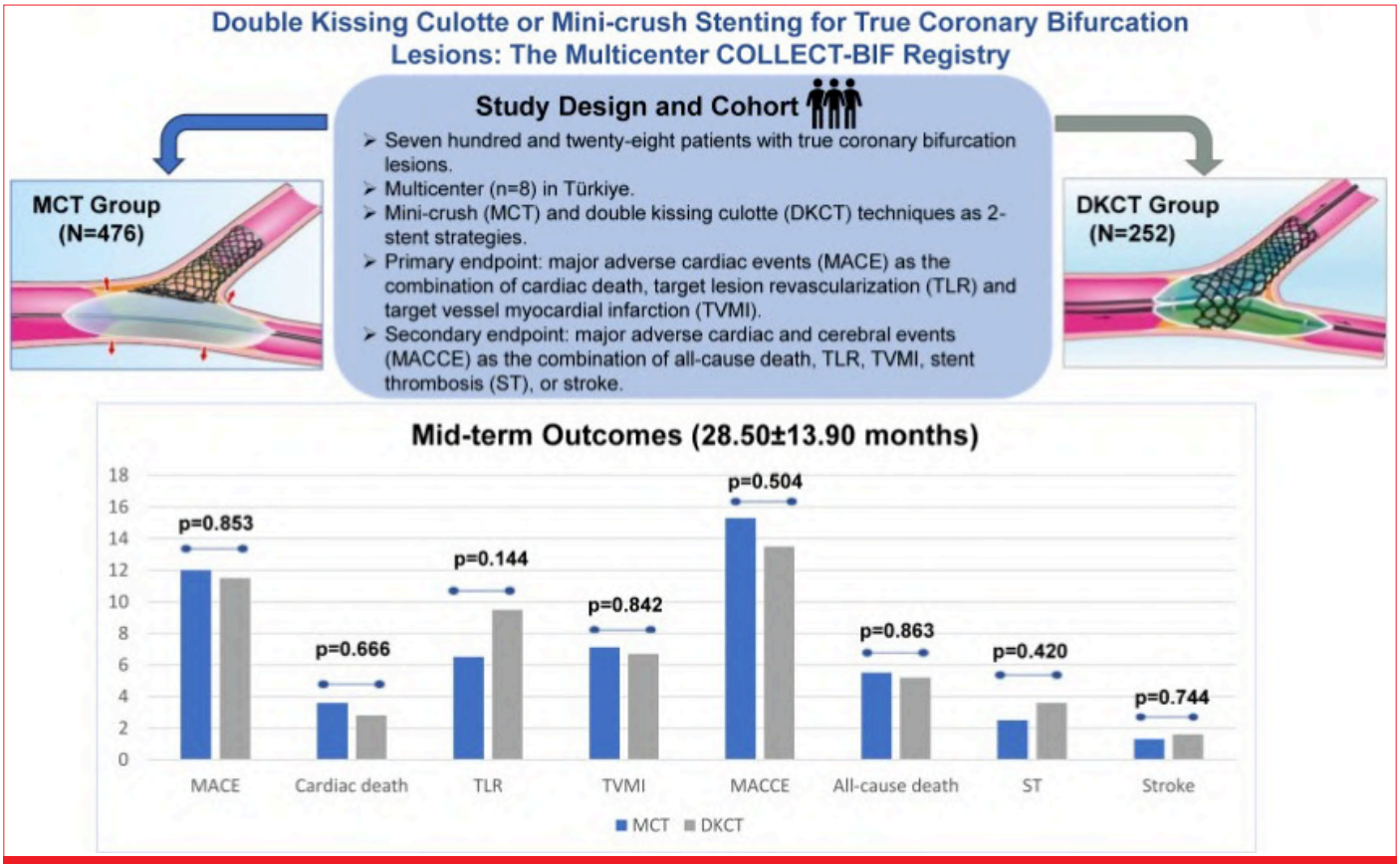

Figure 5. An overview of study design and clinical outcomes. DKCT: Double kissing culotte technique; MACE: Major adverse cardiac events; MACCE: Major adverse cardiovascular and cerebral events; MCT: Mini-crush technique; ST: Stent thrombosis; TVMI: Target vessel myocardial infarction; TLR: Target lesion revascularization.

OP-062 [Interventional Cardiology / Coronary]

Hybrid approach (drug-coated balloon plus stent) in patients with chronic total occlusions

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Background and Aim: Chronic total occlusion (CTO) has commonly long lesions. CTO percutaneous coronary intervention (PCI) has some technical challenges, including underestimated stent dimensions, risk of late stent malapposition, and risk of full-metal jacket. Drug-coated balloons (DC) have many advantages, including preserving physiological properties (vasomotion), induce positive vessel remodeling, avoiding metallic complication (malapposition, ISR), and shorter dual antiplatelet therapy. Therefore, drug-coated balloons provide patients with an alternative or complementary agent to drug-eluting stent (DES) using. Recent study showed that using a DCB-based treatment strategy (either alone or combined with DES) could be effective promising opportunities for CTO lesions. We assessed patients treated hybrid approach (DCB combined DES) for de novo CTO lesions in our clinic at 6 months following time.

Methods: A total of 12 of 136 patients treated with DCB had de novo CTO lesions. We used hybrid technique in 11 patients, the combination DCB with DES (DCB-based PCI). Only one patient underwent DCB treatment alone. Eight patients had LAD lesions, 3 patients had right coronary artery lesions, and 2 patients had circumflex lesions. DCBs were coated with sirolimus combined with either phospholipid (Magic Touch; Concept Medical, Gujarat, India) in 4 patients, biodegradable polymer (Selution; Med Alliance, Nyon, Switzerland) in 5 patients, and paclitaxel coated (Agent; Boston Scientific, Marlborough, Massachusetts) as a carrier for the drug in 3 patients. The primary end point was target lesion failure at 6-months, defined as the composite of target lesion revascularization, cardiac death, and target vessel myocardial infarction.

Results: A total of 11 patients underwent the hybrid approach combining DCB with DES (Figure 1, 2). After 6 months, we performed coronary angiography of those patients (Figure 3). Target lesion failure was not available at 6- months. In 9 of 12 patients, we used dual catheter access. All patients underwent antegrade CTO approach. DCB length was greater than stent length in all hybrid patients. The patients had mean 23 ± 3.6 mm stent lengths. DCB length was 34 ± 8.2 mm. The procedure time were 78 ± 24 min. The fluoroscopy time were 54.6 ± 14 min.

Conclusions: The DCB-based hybrid PCI approach provides excellent results and significantly reduces the stent burden on CTO lesions. This approach seems effective for CTO lesion.

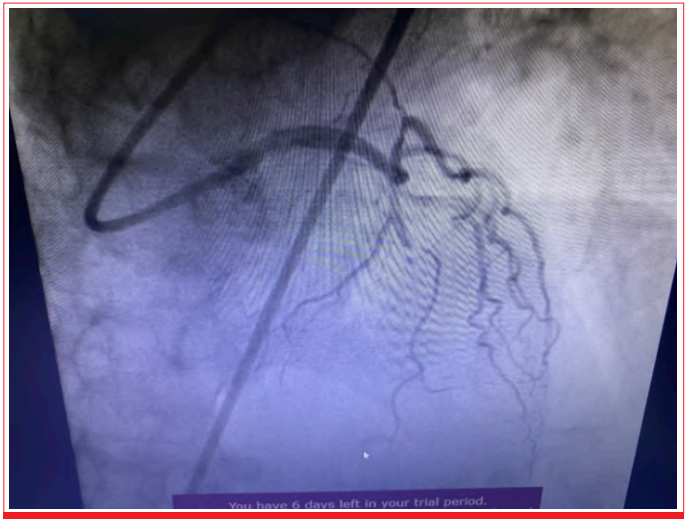

Figure 1. LAD chronic total occlusion.

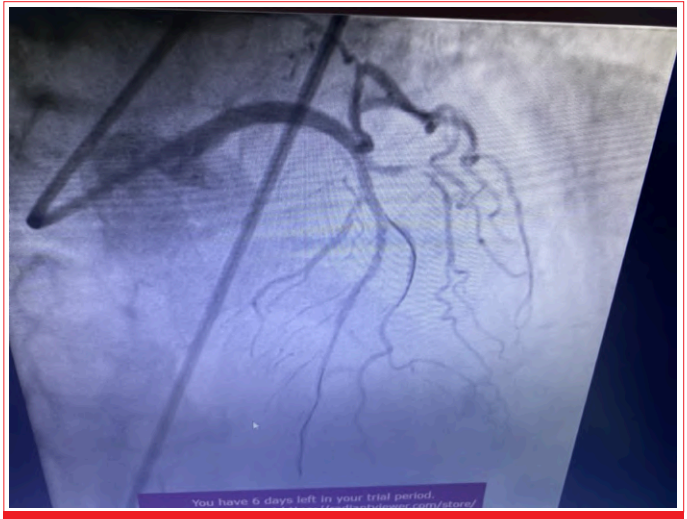

Figure 2. LAD angiographic appearance, after hybrid approach (DCB plus DES).

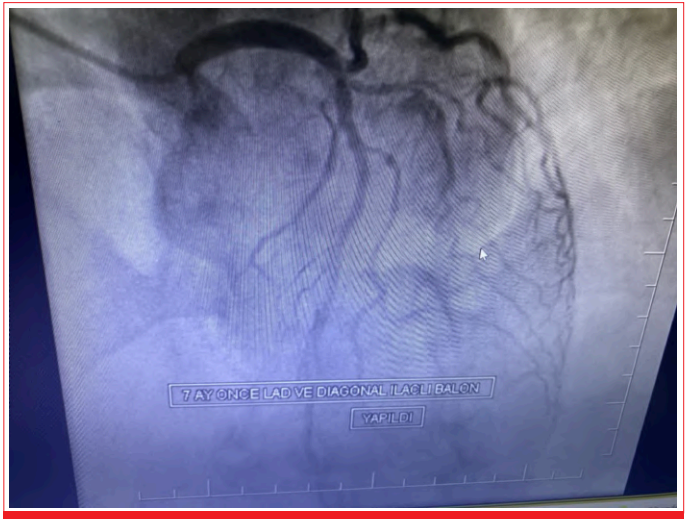

Figure 3. After 6-month, LAD angiographic appearance.

OP-063 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Artificial intelligence vs. human expertise in PVC localization: A prospective validation study

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Background and Aim: Estimating the anatomical localization of ventricular extrasystoles (VES) based on surface electrocardiographic morphology can assist in pre-procedural planning by enabling the appropriate selection of the intervention site. This may help reduce unnecessary arterial punctures and related vascular complications, and can contribute to the optimization of procedure duration and fluoroscopy exposure. In this study, the localization prediction performance of artificial intelligence in outflow tract-origin VES was evaluated.

Methods: Thirty patients who successfully underwent VES ablation were included in the study. For each patient, 12-lead ECG recordings acquired in the electrophysiology lab at a speed of 200 mm/s were used. The origin of each VES was determined as the anatomical region where the earliest intracardiac activity was recorded via ablation catheter, using a three-dimensional electroanatomical mapping system (Figure1). Anatomical classification was based on the ventricular arrhythmias chapter of Clinical Arrhythmology and Electrophysiology: A Companion to Braunwald's Heart Disease, 4th edition (RV free wall, RV posterolateral, RV posteromedial, LCC, RCC, LCC-RCC interleaflet, NCC/parahisian, AMC, Summit). In some patients, adjacent regions were accepted as jointly correct due to overlapping activation zones. Four groups were formed: Two experienced electrophysiologists (EPs), non-trained AI (nAI), and trained AI (tAI). The training involved a brief, text-based exposure outlining morphological ECG features associated with common outflow tract-orogon VES. Each group selected the three most likely regions per case and distributed a total of 10 points among them. Prediction accuracy was calculated as the proportion of the score assigned to the true source region. In cases where the earliest signal was located at two adjacent regions and these were among the predicted zones, combined scores were used. Ventricle prediction accuracy was also analyzed.

Results: 30 outflow tract VES cases were analyzed, most of which originated from the LVOT (Figure 2). In terms of regional localization accuracy, EPs performed better than both tAI and nAI (EPs vs. tAI, p=0.01, EPs vs. nAI, p<0.001, tAI vs. nAI, p=0.06). In ventricle prediction, both EP and tAI achieved higher accuracy compared to nAI (EPs vs. tAI, p=0.09, EPs vs. nAI, p<0.001, tAI vs. nAI, p<0.001) (Table 1-3). The prediction scores and distribution patterns for each group are shown in Figure 3-4 EPs and tAI demonstrated comparable performance in ventricle classification, while nAI had lower accuracy in both region and ventricle prediction tasks.

Conclusions: Although tAI was not as accurate as EPs in regional localization, its ventricle prediction was comparable to EPs. Even limited algorithm-based input improved the predictive accuracy of a large language model in VES localization. Properly trained artificial intelligence models have the potential to assist clinicians in diagnostic decision-making within the field of electrophysiology.

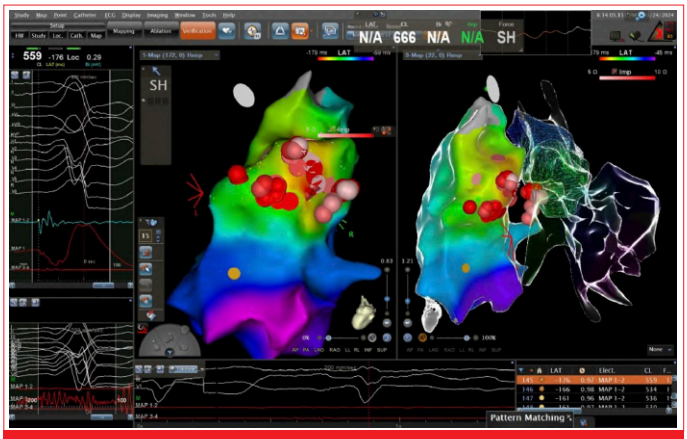

Figure 1. The origin of each VES was determined as the anatomical region where the earliest intracardiac activity was recorded via ablation catheter, using a three-dimensional electroanatomical mapping system.

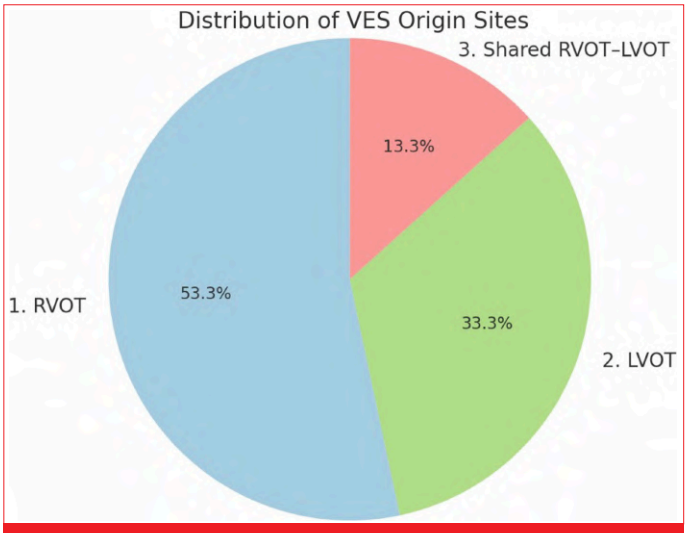

Figure 2. Pie chart illustrating the anatomical distribution of VES origin sites as determined by electroanatomic mapping.

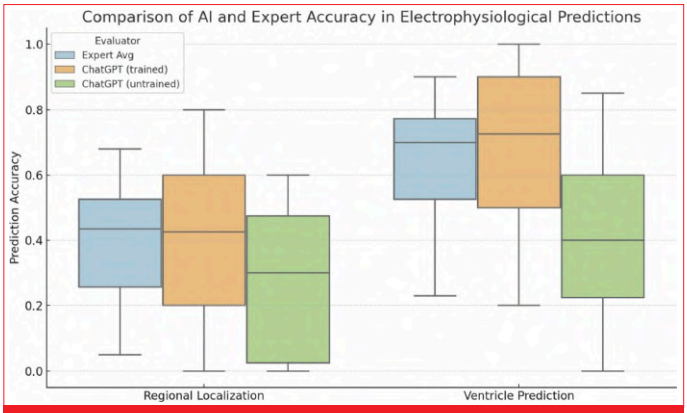

Figure 3. Box plots comparing the prediction accuracy of expert electrophysiologists and AI models (trained and untrained) for VES localization. Two accuracy metrics are shown: regional localization (specific site) and ventricle prediction (right vs. left outflow tract).

Table 1. Regional and ventricle prediction accuracy

Group	Regional Prediction Accuracy	Standard Deviation	Ventricle Prediction Accuracy	Standard Deviation
Experienced Electrophysiologists	0.49	0.25	0.75	0.27
Trained AI	0.38	0.24	0.68	0.26
Non-trained AI	0.29	0.21	0.40	0.23

Table 2. Region-based accuracy comparison

Comparison	Results
Expert Avg vs. AI (trained)	t=1.72, p=0.0953
Expert Avg vs. AI (untrained)	t=8.35, p<0.0001
AI (trained) vs. AI (untrained)	t=5.52, p<0.0001

Statistical comparison of specific anatomical localization accuracy between different evaluators. The trained AI model was compared to the expert average and to the untrained AI model. Statistical significance was observed between the expert average and both AI models, with the trained version showing better alignment with expert performance.

Table 3. Ventricle prediction accuracy comparisons

Compare	Results
Expert Avg vs. AI (trained)	t=2.72, p=0.0110
Expert Avg vs. AI (untrained)	t=4.88, p<0.0001
AI (trained) vs. AI (untrained)	t=1.91, p=0.0655

Statistical comparison of diagnostic accuracy between the trained AI model and the averaged performance of electrophysiology experts. The upper section compares specific anatomical localization, while the lower section assesses broader region-level classification (RVOT vs. LVOT).

OP-064 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Early changes in depolarization and repolarization after catheter ablation of ventricular tachycardia in ischemic cardiomyopathy

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Background and Aim: The development of ventricular tachycardia (VT) has been shown to be associated with depolarization and repolarization parameters. However, the effect of radiofrequency ablation (RFA) applied for VT on depolarization and repolarization parameters remains unknown. This study aimed to investigate changes in measurable depolarization and repolarization parameters following successful RFA in patients with ischemia-induced VT.

Methods: Ninety-six patients with ischemic cardiomyopathy who underwent successful RFA for VT between 2018 and 2025 were included in this study. Twelve-lead electrocardiograms (ECGs) were recorded before and after (72 hours) the RFA procedure. From

both ECGs, PR and QRS durations, QT, corrected QT interval (QTc), Tp–Te intervals, Tp–Te/QT ratio, and QRS–T angle were measured. In addition, left ventricular ejection fraction (LVEF) was measured before and after RFA. Early changes in ECG parameters and LVEF due to RFA were evaluated.

Results: The mean age of the study population was 67.1 ± 8.8 years, and 81% of the patients were male. Analysis of ECG parameters showed significant differences before and after VT ablation in QT, QTc, Tp–Te intervals, Tp–Te/QT ratio, and QRS–T angle ($p<0.001$ for all). QT interval (426 ± 33 vs. 409 ± 31 ms), QTc interval (441 ± 18 vs. 424 ± 17 ms), Tp–Te interval (89 ± 16 vs. 80 ± 16 ms), Tp–Te/QT ratio (0.21 ± 0.04 vs. 0.20 ± 0.04), and QRS–T angle (93 ± 51° vs. 72 ± 45°) were all reduced after ablation. There was no significant change in LVEF before and after RFA (33.6 ± 9.8 vs. 34.1 ± 8.5, Z=-1.356, p=0.195).

Conclusions: Our findings demonstrated that successful RFA therapy in patients with ischemia-induced VT has favorable early effects on ventricular depolarization and repolarization parameters. This information provides a novel and important contribution to the literature on VT ablation.

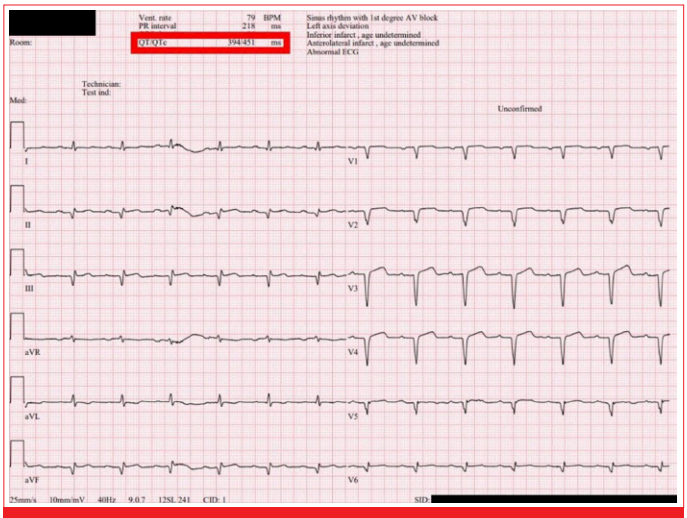

Figure 1. Measurement of electrocardiographic parameters before RFA procedure (increased ventricular depolarization and repolarization parameters).

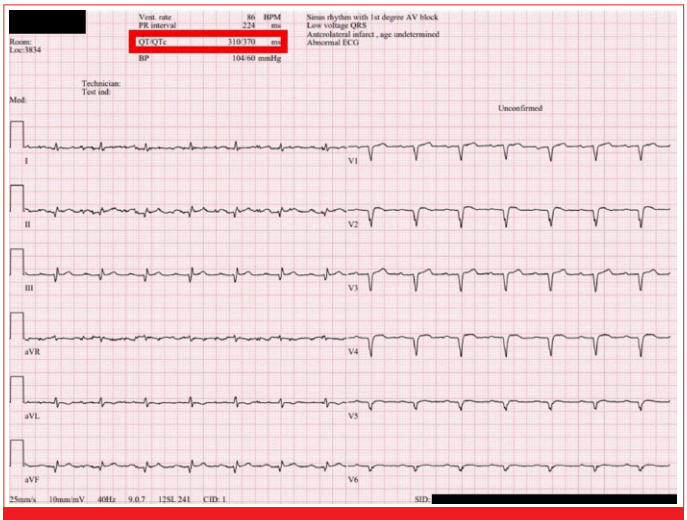

Figure 2. Measurement of electrocardiographic parameters after RFA procedures (normalized ventricular depolarization and repolarization parameters).

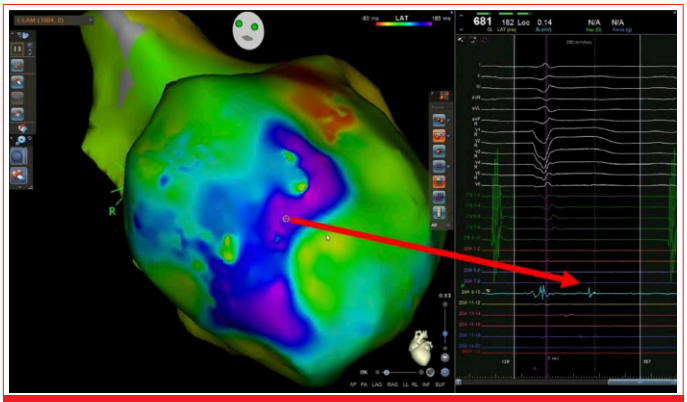

Figure 3. ILAM mapping were performed during sinus rhythm before RFA.

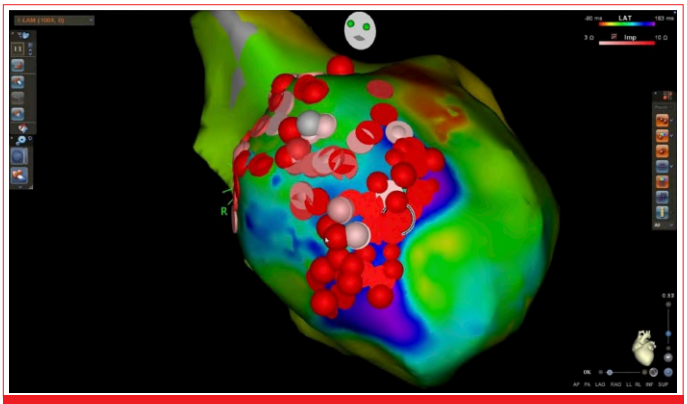

Figure 4. Complete elimination of late potentials during VT ablation.

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

Variables	n=96
Age (year)	65.8 ± 10.6
Gender (Male), n	80 (81%)
Hypertension, n (%)	83 (84%)
Diabetes mellitus, n (%)	38 (39%)
Smoking, n (%)	22 (23%)
Hypercholesterolemia, n (%)	63 (64%)
Systolic blood pressure (mmHg)	123 ± 21
Diastolic blood pressure (mmHg)	78 ± 21
Heart rate (beat/min)	82 ± 15
White blood cell (10^3/ µL)	10.2 ± 6.6
Platelet count (10^3/ µL)	206 ± 59
Hemoglobin (g/dL)	13.1 ± 1.9
Creatinine (mg/dL)	0.97 ± 0.28
Blood Urea Nitrogen (mg/dL)	49.9 ± 13.4
Total cholesterol (mg/dL)	162 ± 19
High-density lipoprotein (mg/dL)	42 ± 10
Low-density lipoprotein (mg/dL)	97 ± 31
Triglycerides (mg/dL)	142 ± 45

Table 2. Comparison of 12 lead electrocardiographic measurements before and after radiofrequency ablation

Variables	Basal measurements n=96	Measurements after RFA n=96	t and Z value	p
PR duration (msec)	161 ± 19	167 ± 22	-1.775 t	0.079[a]
QRS duration (msec)	116 ± 28	119 ± 34	-1.060 Z	0.292[b]
QT interval (msec)	426 ± 33	409 ± 31	10.734 t	<0.001[a]
QTc interval (msec)	441 ± 18	424 ± 17	15.439 t	<0.001[a]
JT interval (msec)	310 ± 34	290 ± 33	6.128 t	<0.001[a]
Tp-Te interval (msec)	89 ± 16	80 ± 16	17.758 t	<0.001[a]
Tp-Te/QT ratio	0.21 ± 0.04	0.020 ± 0.04	8.268 t	<0.001[a]
QRS-T angle (°)	93 ± 51	72 ± 45	5.916 Z	<0.001[b]

a: Paired t-test; b: Wilcoxon test, RFA: radiofrequency ablation.

OP-065 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Flecainide may be used for rhythm control therapy in patients with failed cryoballoon ablation for paroxysmal atrial fibrillation

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Background and Aim: Class IC anti-arrhythmic drugs are primarily preferred for rhythm control of atrial fibrillation (AF). In cases where ablation fails, the rhythm control strategy approach is still unclear. In our study, we aimed to determine the efficacy of flecainide, propafenone and radiofrequency ablation (RFA) as rhythm control strategies in paroxysmal AF patients with failed cryoballoon ablation (CBA).

Methods: In this cross-sectional study, 1120 patients who underwent CBA for paroxysmal AF between 2017 and 2024 were screened. In this patient group, 230 patients with recurrent AF (≥ 3 months) after CBA were identified. A total of 120 patients (40 cases per treatment) who received rhythm control decision and who received flecainide or propafenone or RFA were included in the study. Patients were divided into 3 groups: flecainide (Group I), propafenone (Group II) and RFA (Group III). All patients were followed for at least 1 year for AF recurrence.

Results: F recurrence was found in 52 (43.2%) patients included in the study. The AF recurrence rates in Groups I, II and III were 35%, 75% and 20%, respectively. Although the frequency of AF recurrence in Group I and III was statistically similar (p>0.05), the frequency of AF recurrence in Group I-III was significantly lower than

Table 1. Demographic, clinical and medical treatment findings of the study groups

Variables	Group I n=40	Group II n=40	Group III n=40	p
Age (year)	61.5 ± 12.4	58.9 ± 13.1	56.8 ± 12.5	0.178
Gender (female), n	20 (50)	14 (35)	16 (40)	0.339
Heart failure, n (%)	4 (10)	4 (10)	2 (5)	0.404
Hypertension, n (%)	16 (40)	18 (45)	20 (50)	0.645
Diabetes mellitus, n (%)	7 (18)	6 (15)	8 (20)	0.467
Coronary artery disease history, n (%)	6 (15)	6 (15)	10 (25)	0.256
Previous cerebrovascular accident, n (%)	5 (13)	4 (10)	4 (10)	0.545
Systolic blood pressure (mmHg)	129 ± 12	128 ± 13	124 ± 13	0.145
Diastolic blood pressure (mmHg)	80 ± 7.8	81 ± 9.8	79 ± 9.5	0.285
Heart rate (beat/min)	74 ± 10	78 ± 13	72 ± 16	0.165
CHA2DS2-VA score	1.6 ± 1.5	1.4 ± 1.6	1.4 ± 1.7	0.345
ACEI or ARB use, n (%)	20 (50)	16 (40)	18 (45)	0.374
Beta blocker use, n (%)	40 (100)[α,β]	32 (80)	28 (70)	0.001
Oral anti-coagulant use, n (%)	36 (90)	36 (90)	32 (80)	0.378
Maintenance sinus rhythm ≥1 year, n (%)	26 (65)[α]	10 (25)¥	32 (80)	0.032

The values were shown as mean ± standard deviation or n (%), ACEI: Angiotensin converting enzyme inhibitors, ARB: Angiotensin receptor blocker. Group I: Flecainide group, Group II: Propafenone group and Group III: 3D radiofrequency catheter ablation group. α: The significant association between the Group I and Group II (p<0.05), β: The significant association between the Group I and Group III (p<0.05), ¥: The significant association between the Group II and Group III (p<0.05).

Table 2. Laboratory findings of the study groups

Variables	Group I n=40	Group II n=40	Group III n=40	p
White blood cell (μL)	8.5 ± 2.2	7.4 ± 1.6	8.2 ± 2.9	0.172
Hemoglobin (g/dL)	13.2 ± 1.8	13.9 ± 2.7	13.3 ± 1.7	0.467
Platelet count (10^3/μL)	248 ± 69	229 ± 76	264 ± 60	0.138
Blood urea nitrogen (mg/dL)	32 ± 8.1	29 ± 7.2	34 ± 9.2	0.146
Creatinine (mg/dL)	0.86 ± 0.27	0.81 ± 0.14	0.82 ± 0.23	0.178
Total cholesterol (mg/dL)	181 ± 44	183 ± 43	193 ± 54	0.511
Low density lipoprotein cholesterol (mg/dL)	122 ± 25	116 ± 26	125 ± 38	0.481
High density lipoprotein cholesterol (mg/dL)	47 ± 15	46 ± 12	45 ± 13	0.752
Triglyceride (mg/dL)	140 ± 52	192 ± 78	182 ± 88	0.183
hs-CRP (mg/L)	0.41 ± 0.10	0.43 ± 0.34	0.47 ± 0.38	0.566
LVEF (%)	57 ± 4.6	56 ± 6.8	58 ± 4.3	0.676
Left atrial dimension (mm)	43 ± 3.4	43 ± 2.8	41 ± 5.9	0.072

The values were shown as mean ± standard deviation. hs-CRP: High sensitive C reactive protein, LVEF: Left ventricular ejection fraction. Group I: Flecainide group, Group II: Propafenone group and Group III: 3D radiofrequency catheter ablation group.

Table 3. Parameters found to be different in in patients with and without AF recurrence

Variables	AF recurrence (-) n=68	AF recurrence (+) n=52	p
Left atrial dimension (mm)	42 ± 4.5	45 ± 4.5	<0.001
Flecainide use, n (%)	26 (38)	14 (27)	0.034
Propafenone use, n (%)	10 (15)	30 (58)	0.001
RF ablation, n (%)	32 (47)	8 (15)	<0.001

AF: Atrial fibrillation, RF: Radiofrequency catheter ablation.

in Group II (p<0.05). Group I patients were significantly more likely to use beta-blockers than Group I-II patients (p<0.05). Patients with AF recurrence had higher left atrial (LA) diameter and propafenone use. The number of patients who used flecainide and underwent RFA was lower in the AF recurrence group. In logistic regression analysis, LA diameter was found to be an independent predictor of patients with AF recurrence (p=0.002).

Conclusions: According to the results of our study, flecainide treatment can be used with an acceptable success rate in patients with recurrent AF after CBA.

OP-066 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Apelin is decreased in HFrEF and closely related with ventricular tachycardia

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Background and Aim: The serum apelin level in patients with heart failure with reduced ejection fraction (HFrEF) and its relationship with ventricular tachycardia (VT) are not clearly known. This study aimed to investigate changes in serum apelin levels in patients with HFrEF and their relationship with VT.

Methods: This cross-sectional study included 60 patients with and without VT and HFrEF, and 30 controls without coronary artery disease. In addition to routine medical history, physical examination, laboratory tests, and echocardiography, serum apelin levels were measured. Patients were divided into three groups: HFrEF with VT (Group I), HFrEF without VT (Group II), and the control group (Group III). All parameters were compared between groups. Parameters associated with VT and apelin levels were identified.

Results: Serum apelin levels were found to be significantly higher in Group I-II than in Group III. Serum glucose, creatinine, and left atrial diameter were shown to be significantly higher in Group I-II than in

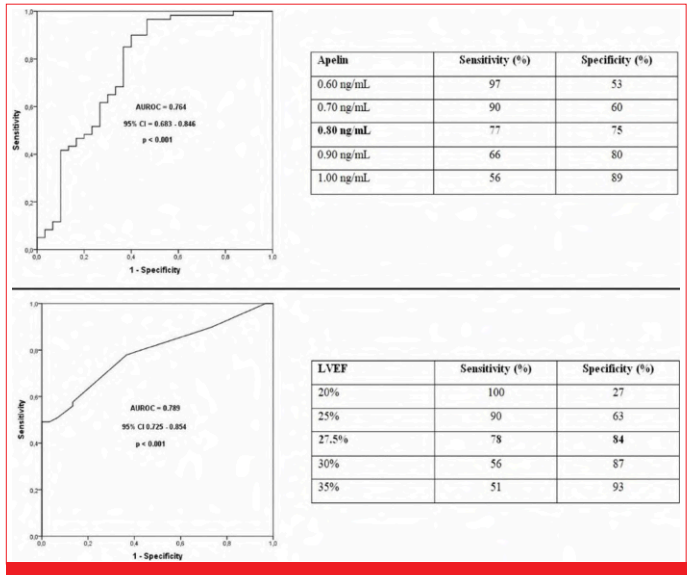

Figure 1. Receiver operating characteristic curves and cut-off levels for plasma apelin and left ventricular ejection fraction in the predicting of ventricular tachycardia.

Table 1. Demographic, clinical and laboratory findings of the study groups

Variables	Group I n=40	Group II n=40	Group III n=40	p
Age (year)	65.8 ± 10.6	64.3 ± 9.1	63.4 ± 10.9	0.278
Gender (female), n	4 (10)	6 (15)	5 (13)	0.567
Hypertension, n (%)	24 (60)	27 (68)	19 (48)	0.123
Diabetes mellitus, n (%)	13 (33)	16 (40)	7 (18)	0.116
Smoking, n (%)	21	20	19	0.435
Coronary artery disease history, n (%)	6 (15)	6 (15)	10 (25)	0.256
Previous cerebrovascular accident, n (%)	5 (13)	4 (10)	4 (10)	0.545
NYHA class I-II-III-IV	6-29-22-3	6-30-22-2	–	0.799
Heart rate (beat/min)	76 ± 7.8	77 ± 11	76 ± 11	0.167
Systolic blood pressure (mmHg)	122 ± 13	123 ± 11	121 ± 12	0.345
Diastolic blood pressure (mmHg)	74 ± 7.7	76 ± 6.8	76 ± 8.8	0.485
Body mass index (kg/m2)	27.8 ± 4.6	27.7 ± 3.8	28.1 ± 3.4	0.762
White blood cell (μL)	9.1 ± 2.7	8.6 ± 2.9	7.9 ± 2.5	0.256
Hemoglobin (g/dL)	13.2 ± 2.2	13.7 ± 1.7	13.1 ± 2.3	0.474
Glucose (mg/dL)	130 ± 42α	132 ± 37β	103 ± 21	0.006
Blood urea nitrogen (mg/dL)	48.1 ± 30.1	42.7 ± 29.8	31.9 ± 17.5	0.068
Creatinine (mg/dL)	1.04 ± 0.41α	1.03 ± 0.46β	0.76 ± 0.26	0.009
Total cholesterol (mg/dL)	186 ± 55	196 ± 58	202 ± 27	0.490
Low density lipoprotein cholesterol (mg/dL)	120 ± 44	132 ± 41	132 ± 19	0.427
High density lipoprotein cholesterol (mg/dL)	42.6 ± 8.6α	40.8 ± 9.4β	49.4 ± 9.9	0.015
Triglyceride (mg/dL)	176 ± 592	182 ± 102	173 ± 104	0.945
High sensitive C reactive protein (mg/L)	20.4 ± 24.8	17.2 ± 28.5	5.9 ± 6.9	0.171
Apelin (ng/mL)	0.83 ± 0.55α	1.02 ± 0.46β	1.77 ± 0.95	0.001
Left ventricular ejection fraction (%)	26.7 ± 6.8α	27.8 ± 5.3β	59.2 ± 3.3	<0.001
Left atrial dimension (mm)	42.6 ± 3.2α	41.1 ± 3.6β	34.3 ± 3.1	<0.001

The values were shown as mean ± standard deviation or n (%), Group I: Heart failure with ventricular tachycardia, Group II: Heart failure without ventricular tachycardia and Group III: Control group. ¥: The significant association between the Group I and Group II ($p<0.05$), α: The significant association between the Group I and Group III ($p<0.05$), β: The significant association between the Group II and Group III ($p<0.05$).

Table 2. Multivariate logistic regression analysis for identifying patients with ventricular tachycardia

Variable	Odds ratio	95% CI	p
Apelin (ng/mL)	0.313	0.124-0.788	0.014
Left ventricular ejection fraction (%)	0.912	0.877-0.968	<0.001

Group III. HDL cholesterol and left ventricular ejection fraction (LVEF) levels were significantly lower in Group I-II compared to Group III. A positive and negative correlation was found between plasma apelin levels and LVEF and age, respectively. In logistic regression analysis, plasma apelin levels and LVEF values were found to independently determine VT in patients and 1 ng/mL decrease in apelin levels was found to increase the risk of VT by 69%. In the ROC analysis, the area under the ROC curve for apelin was found to be 0.764. When the apelin cutoff value was set at 0.80 ng/mL, it was found to predict the presence of VT with 77% sensitivity and 75% specificity.

Conclusions: According to the results of our study, serum apelin levels are significantly reduced in patients with HFrEF, and reduced apelin levels are closely associated with the development of VT in these patients.

OP-067 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Evaluation of long term outcomes of implantable cardioverter defibrillator therapy for primary and secondary prophylaxis

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Background and Aim: The aim was to compare the long-term follow-up results of patients with implantable cardioverter defibrillators (ICD) for primary and secondary prophylaxis indications, to determine the time to ICD therapy, and to evaluate the clinical factors affecting this time.

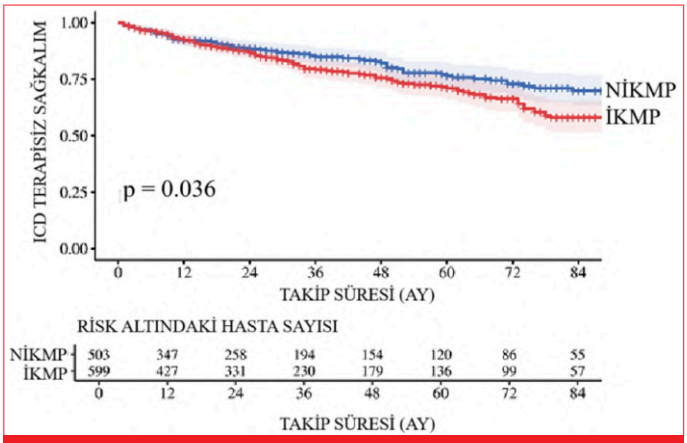

Figure 1. Survival curve ICD therapy-free in primary prophylaxis in ICMP and NICMP groups.

Methods: Patients aged ≥18 years who underwent ICD implantation between January 1, 2015, and July 1, 2024, were retrospectively analyzed. Patients were grouped as primary (n=1102) and secondary (n=227) prophylaxis. Ischemic (ICMP) and nonischemic cardiomyopathy (NICMP) subgroups were identified based on etiology. Time to ICD therapy, appropriate/inappropriate shock rates, and survival analyses were performed.

Results: The mean follow-up period was 38.9 ± 31.9 months. A total of 1329 patients, 960 males (72.2%) and 369 females (27.8%), were included in the study. The mean age of the patients was 61 ± 14 years. The mean ICD therapy-free follow-up period for patients with ICMP was 34 ± 29 months, and for patients with NICMP was 34 ± 31 months. During the follow-up period, ICD therapy occurred in 161 (23.61%) patients with ICMP and 139 (21.67%) patients with NICMP. No significant difference was observed in ICD therapy-free survival during the follow-up period in either group (p=0.44). However, the survival rate of patients in the ICMP group was significantly lower than that of patients in the NICMP group throughout the entire follow-up period (p<0.001). The rate of appropriate shocks for VT/VF was significantly higher in NICMP patients in the secondary prophylaxis group compared to the primary prophylaxis group (78.85% vs. 32.12%; p<0.001). ICD therapy occurred in 130 (21.70%) patients with NICMP in the primary prophylaxis group and in 83 (16.50%) patients with NICMP. ICD therapy-free survival was significantly higher in the NICMP group among primary prophylaxis patients (p=0.036) (Figure 1). In the multivariate analysis examining all groups, VT ablation (HR: 0.41; 95% CI: 0.27–0.62; p<0.001) and ischemic cardiomyopathy (HR: 0.62; 95% CI: 0.39–0.97; p=0.037) were found to be significant predictors reducing VT and ICD therapy recurrence.

Conclusions: Patients with an ICD implanted for secondary prophylaxis, particularly in the NICMP subgroup, require earlier and more frequent ICD therapy. VT ablation is effective in reducing ICD therapy relapses, and individualized patient selection may improve long-term outcomes.

OP-068 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Silent cerebral embolism following left ventricular endocardial ablation: The SILVER study

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Background and Aim: Endocardial ventricular ablation is frequently used to manage premature ventricular complexes (PVCs) and idiopathic ventricular tachycardias (VTs); however, data regarding new silent cerebral embolism (SCE) during this procedure are quite limited. This prospective study sought to determine the incidence and potential risk factors of SCE in patients who underwent left ventricular (LV) ablation for PVCs or idiopathic VTs.

Methods: A total of 37 [male: 15 (40.5%), mean age: 52.27 ± 14.31 years] patients who underwent treatment of endocardial ventricular ablation at our institution between August 2022 and January 2024 were included. Baseline data, arrhythmia characteristics (PVC/VT), and procedural details were prospectively evaluated. The primary endpoint was defined as a new SCE after the procedure. All patients were evaluated with diffusion-weighted magnetic resonance imaging (DW-MRI) before and after the procedure within 24 hours.

Results: Right ventricular (RV) and LV endocardial ablation was performed in 10 (VT, n=1, PVC, N=9) and 27 patients (VT, n=3, PVC, n=24), respectively. At least 1 new SCE detected by DW-MRI was observed in 6 patients (22.2%) in the LV ablation group, while no SCE was detected in the RV ablation group (p=0.162). Older age (64.67 ± 7.42 vs. 51.33 ± 14.13 years, p=0.022), higher CHA_2DS_2-VASc scores (3.33 ± 1.21 vs. 1.52 ± 1.12, p=0.002), and elevated NT-proBNP levels (625.00 ± 477.78 vs. 119.48 ± 112.99, p=0.001) were associated with SCE among LV ablation patients.

Conclusions: Endocardial LV ablation for PVC/idiopathic VT is at significant risk for SCE, with postprocedural SCE detected in 22.2% of the patients. Additionally, older age, high CHA_2DS_2-VASc score, and elevated NT-pro BNP levels are associated with SCE.

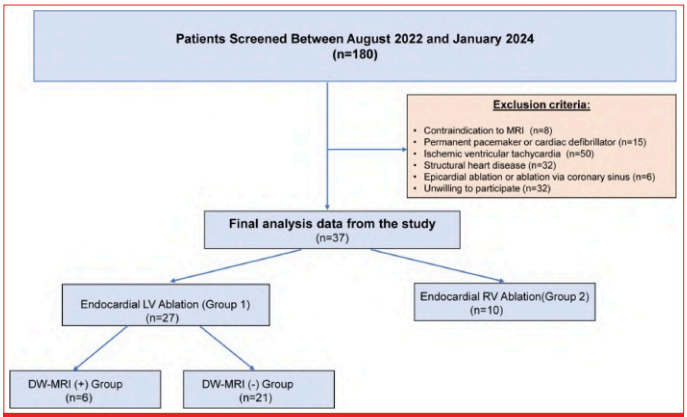


Figure 1. Flow chart and patient selection of study. LV: Left ventricle; MRI: Magnetic resonance imaging; RV: Right ventricle SCE: Silent cerebral embolism.

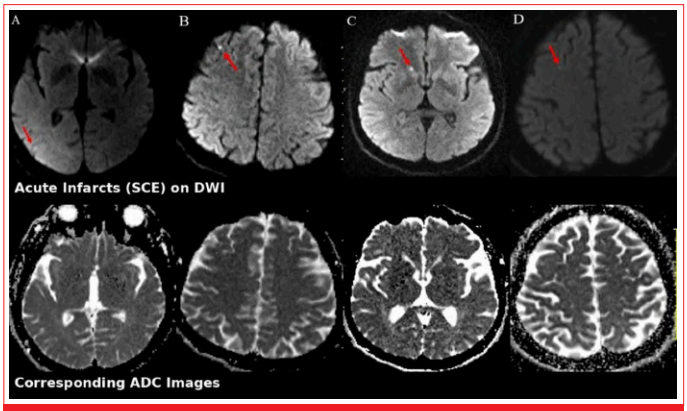


Figure 2. Diffusion-weighted imaging (DWI) and corresponding apparent diffusion coefficient (ADC) images of patients with a single silent cerebral embolism (SCE) following ablation. Silent cerebral embolic (SCE) lesions are shown with red arrows. A: Patient LV-1; Right parieto-occipital lobe B: Patient LV-8; Right frontal lobe C: Patient LV-10; Right caudate nucleus D: Patient LV-20; Right corona radiata.

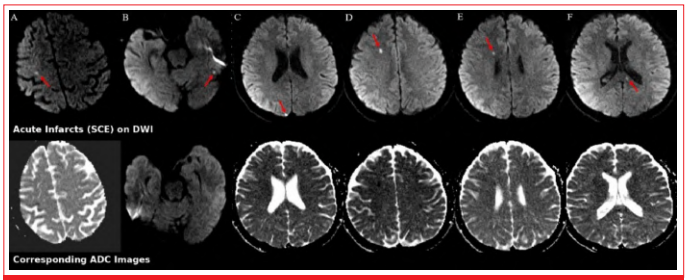


Figure 3. Diffusion-weighted imaging (DWI) and corresponding apparent diffusion coefficient (ADC) images of patients with multiple silent cerebral embolism(SCE) following ablation. Silent cerebral embolic (SCE) lesions are shown with red arrows. Patient LV-4: (A) Right parietal cortex; (B) Left cerebellar hemisphere. Patient LV-12: (C) Right occipital cortex; (D) Right corona radiata; (E) Right caudate nucleus; (F) Splenium of the left corpus callosum.

Table 1. Baseline demographic, clinical, and echocardiographic characteristics per study group

Variables	RV Ablation Group (n=10)	LV Ablation Group (n=27)	P value
Age, years	46.8±15.06	54.3±14	0.191
Gender, male	3 (30)	12 (44.4)	0.481
Comorbidities			
Body mass index, kg/m^2	29.92±4.97	28.64±3.25	0.366
Hypertension, n (%)	4 (40)	12 (44.4)	1.00
Diabetes mellitus, n (%)	2 (20)	12 (44.4)	0.260
Atrial fibrillation, n (%)	0 (0)	1 (3.7)	1.00
CHA_2DS_2-VASc	1.80±1.32	1.93±1.36	0.933
PVC induced cardiomyopathy,n (%)	3 (30)	5 (18.5)	0.655
Hyperlipidemia, n (%)	4 (40)	19 (70.4)	0.132
Current smoker, n (%)	2 (20)	3 (11.1)	0.597
Previous PCI, n (%)	0 (0)	4 (14.8)	0.557
History of stroke/TIA, n (%)	0 (0)	0 (0)	-
Peripheral artery disease, n (%)	0 (0)	1 (3.7)	1.00
Previous PVC ablation, n (%)	0 (0)	3 (11.1)	0.548
Previous VT ablation, n (%)	0 (0)	1 (3.7)	1.00
Clinical symptoms n (%)			
Syncope	0 (0)	1 (3.7)	1.00
Palpitation	10 (100)	26 (96.3)	1.00
Dyspnea	3 (30)	8 (29.6)	1.00
Medications Used, n (%)			
Propafenone	2 (20)	4 (14.8)	0.653
CCB	1 (10)	3 (11.1)	1.00
Beta blockers	9 (90)	22 (81.5)	1.00
ACEi / ARB	4 (40)	11 (40.7)	1.00
Antiplatelets	3 (30)	11 (40.7)	0.710
Statin	4 (40)	3 (11.1)	0.069
Diuretics	4 (40)	3 (11.1)	0.069
Echocardiography			
LV ejection fraction (%)	52.40±10.86	56.15±8.81	0.345
Aortic valve sclerosis, n(%)	2 (20)	3 (11.1)	0.597
Mitral valve sclerosis, n(%)	1 (10)	6 (22.2)	0.647
Moderate-severe valve disease, n (%)	3 (30)	6 (22.2)	0.679
LAD, (mm)	36.4±4.77	37.1±4.47	0.679
LVEDD, (mm)	50.4±6.69	49.59±4.61	0.880
LVESD, (mm)	36.6±6.64	33.59±6	0.158
IVST, (mm)	9.8±0.63	10.07±1.46	0.371

ACEI: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker; CCB: Calcium channel blocker; IVST: Interventricular septal thickness; LAD: Left atrial diameter; LVEDD: Left ventricular end diastolic diameter; PVC: Premature ventricular complex.

Table 2. The main laboratory findings and procedural data of ablation patients

Variables	RV Ablation Group (n=10)	LV Ablation Group (n=27)	P value
Blood Work-up			
White blood cell count, (10^9/L)	7.09±1.16	6.95±1.99	0.229
Hemoglobin, (g/dL)	13.04±1.34	13.58±1.47	0.319
Platelet (10^9/L)	255.40±76.50	223.48±62.51	0.180
Creatinine, (mg/dL)	0.72±0.19	0.79±0.2	0.332
eGFR,mL/min/1.73 m^2	101.6±14.9	92.25±18.13	0.154
INR	1.06±0.07	1.09±0.14	0.533
LDL-cholesterol, mg/dL	103±39.82	111.07±31.29	0.389
CRP, mg/dL	4.79±6.82	2.59±2.07	0.906
NT-pro BNP, pg/mL	156.9±166.97	231.81±315.58	0.578
Troponin T, ng/mL	6.4±3.89	16.96±38.96	0.555
Neutrophil cell count, (10^9/L)	3.95±0.92	4.23±1.38	0.933
Lymphocyte cell count, (10^9/L)	2.4±0.62	2.12±0.73	0.335
Procedural characteristics			
Arrhythmia target-idiopathic VT, n (%)	1 (10)	3 (11.1)	1.00
Arrhythmia target-PVC, n (%)	9 (90)	24 (88.9)	1.00
PVC percentage	24.4±10.82	27.81±14.5	0.440
Monomorphic	8 (80)	22 (81.5)	1.00
Polymorphic	2 (20)	5 (18.5)	1.00
EnSite mapping, n (%)	1 (10)	18 (66.7)	**0.003**
CARTO-Mapping, n (%)	9 (90)	9 (33.3)	**0.003**
LV ablation locations, n (%) *			
LV-Summit	0 (0)	8 (29.6)	-
LV-AMC	0 (0)	2 (7.4)	-
LCC	0 (0)	8 (29.6)	-
RCC	0 (0)	8 (29.6)	-
Purkinje	0 (0)	1 (3.7)	-
Left posterior fascicle	0 (0)	2 (7.4)	-
Mitral anulus anterior	0 (0)	1 (3.7)	-
Mitral anulus posterior	0 (0)	1 (3.7)	-
RV ablation locations, n(%)*			
RVOT posterolateral	1 (10)	0 (0)	-
RVOT anterolateral	6 (60)	0 (0)	-
RVOT septal	4 (40)	0 (0))	-
Parahisian	1 (10)	0 (0)	-
Procedure time,min	53.5±14.73	57.3±19.66	0.749

* indicates number of LV ablation locations rather than patients with endocardial ablation. Abbreviations: CRP: C-reactive protein; eGFR: Estimated glomerular filtration rate; INR: International normalized ratio; LDL=low-density lipoprotein; LV: Left ventricle; LCC: Left coronary cusp; LVOT-AMC: Left venticular outflow tract-aorto mitral continuity; NT-pro BNP: N-terminal prohormone of brain natriuretic peptide; PVC: Premature ventricular complex; RCC: Right coronary cusp; RVOT: Right venticular outflow tract; VT: Ventricular tachycardia.

Table 3. Peri- and postprocedural characteristics per study group

Outcomes	RV Ablation Group (n=10)	LV Ablation Group (n=27)	p
Periprocedurel new-onset AF, n (%)	0 (0)	0 (0)	-
Periprocedural CVE/TIA, n (%)	0 (0)	0 (0)	-
Silent new cerebral emboli, n (%)	0 (0)	6 (22.2)	0.162
<2 mm	0 (0)	1 (3.7)	1.00
2-5 mm	0 (0)	4 (14.8)	0.557
>5 mm	0 (0)	1 (3.7)	1.00
New emboli count	0 ± 0	0.7 ± 1.54	0.319
Death	0 (0)	0 (0)	-

AF: Atrial fibrillation; CVE: Cerebrovascular event; TIA: Transient ischemic attack.

Table 4. Demographic and procedural characteristics of silent cerebral embolism patients who underwent left ventricular endocardial ablation

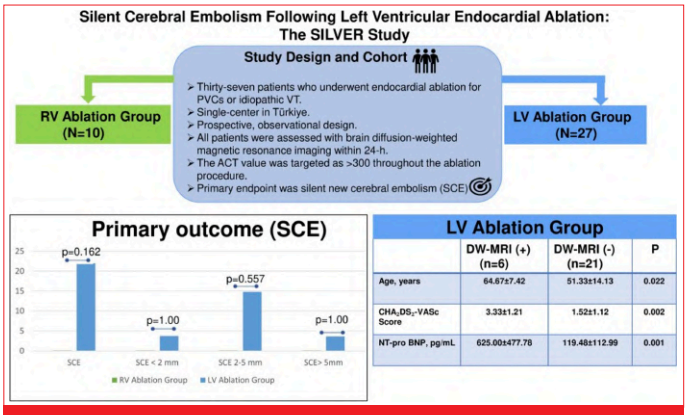
Variables	DW-MRI (+) Group (n=6)	DW-MRI (-) Group (n=21)	P value
Age, years	64.67±7.42	51.33±14.13	**0.022**
Gender, male	1 (16.7)	11 (52.4)	0.182
Comorbidities			
Body mass index, kg/m^2	30.06±2.58	28.24±3.37	0.235
Hypertension, n (%)	4 (66.7)	8 (38.1)	0.357
Diabetes mellitus, n (%)	4 (66.7)	8 (38.1)	0.357
Atrial fibrillation, n (%)	0 (0)	1 (4.8)	1.00
CHA_2DS_2-VASc	3.33±1.21	1.52±1.12	**0.002**
PVC induced cardiomyopathy, n (%)	1 (16.7)	4 (19.0)	1.00
Hyperlipidemia, n (%)	5 (83.3)	14 (66.7)	0.633
Current smoker, n (%)	0 (0)	3 (14.3)	1.00
Previous PCI, n (%)	1 (16.7)	3 (14.3)	1.00
History of stroke, n (%)	1 (16.7)	0 (0)	1.00
History of PVC ablation, n (%)	0 (0)	3 (14.3)	1.00
History of VT ablation, n (%)	0 (0)	1 (4.8)	1.00
Clinical symptoms, n (%)			
Syncope	0 (0)	1 (4.8)	1.00
Palpitation	6 (100)	20 (95.2)	1.00
Dyspnea	2 (33.3)	6 (28.6)	1.00
Medications Used, n (%)			
Propafenone	1 (16.7)	3 (14.3)	1.00
CCB	1 (16.7)	2 (9.5)	0.545
Beta blockers	5 (83.3)	17 (81.0)	1.00
ACEi / ARB	5 (83.3)	6 (28.6)	**0.027**
Antiplatelets	2 (33.3)	9 (42.9)	1.00
Statin	1 (16.7)	2 (9.5)	0.545
Diuretics	1 (16.7)	2 (9.5)	0.545
Echocardiographic Features			
LV ejection fraction (%)	54.33±9.75	56.67±8.71	0.577
Aortic valve sclerosis, n (%)	1 (16.7)	2 (9.5)	0.545
Mitral valve sclerosis, n (%)	3 (50.0)	3 (14.3)	0.101
Moderate-severe valve disease, n (%)	2 (33.3)	4 (19.0)	0.588
LAD, (mm)	39.2±3.07	36.5±4.69	0.199
LVEDD, (mm)	50.5±4.42	49.33±4.74	0.595
LVESD, (mm)	35.83±6.94	32.95±5.73	0.239
IVST, (mm)	10.22±1.33	10.02±1.52	0.781
Blood Work-up			
White blood cell count, (10^9/L)	7.57±3.05	6.78±1.63	0.629
Neutrophil count, (10^9/L)	4.89±1.88	4.04±1.2	0.798
Lymphocyte count, (10^9/L)	2.14±1.1	2.12±0.62	0.957
Hemoglobin, (g/dL)	13.5±1.09	13.6±1.58	0.886
Platelet count, (10^9/L)	219.83±92.1	224.52±54.36	0.875
Creatinine, (mg/dL)	0.82±0.24	0.78±0.19	0.669
eGFR, mL/min/1.73 m^2	82±16.07	95.18±17.94	0.118
INR	1.19±0.23	1.06±0.1	0.175
ACT (seconds)	319.1±21.4	322.5±22.8	0.954
LDL-cholesterol, mg/dL	117.33±36.29	109.29±30.47	0.588
CRP, mg/dL	3.83±3.82	2.23±1.14	0.512
NT-pro BNP, pg/mL	625.00±477.78	119.48±112.99	**0.001**
Troponin T, ng/mL	10.50±8.92	18.81±44.01	0.755
Procedural characteristics			
Arrhythmia target-idiopathic VT, n (%)	0 (0)	3 (14.3)	1.00
Procedure time, min	59.00±15.62	56.81±20.98	0.815
Total fluoroscopy time, min	6.8±1.9	6.7±2.5	0.483
Total RF duration, sec	400.2 ± 102.3	459.0 ± 100.2	0.307
Arrhythmia target-PVC, n (%)	6 (100)	18 (85.7)	1.00
PVC percentage	29.83±9.33	27.24±15.81	0.621
Monomorphic	6 (100)	16 (76.2)	0.555
Polymorphic	0 (0)	2 (9.5)	1.00
EnSite mapping, n (%)	4 (66.7)	14 (66.7)	1.00
CARTO-Mapping, n (%)	2 (33.3)	7 (33.3)	1.00
Approach to LV			
Retrograde only	6 (100)	20 (95.2)	0.983
Retrograde + transseptal	0 (0)	1 (4.8)	1.00
Ablator sheath used			
8 Fr short	13 (61.9)	4 (66.7)	0.648
8 Fr long non-deflectable	5 (23.8)	1 (16.7)	0.587
11 Fr deflectable	1 (4.8)	0 (0)	1.00
11.5 Fr deflectable	2 (9.5)	1 (16.7)	1.00
LV ablation locations, n (%)			
LV-Summit	2 (33.3)	6 (28.6)	1.00
LV-AMC	0 (0)	2 (9.5)	1.00
LCC	2 (33.3)	6 (28.6)	1.00
RCC	0 (0)	7 (33.3)	0.172
Purkinje	0 (0)	1 (4.8)	1.00
Posterior fascicle	0 (0)	2 (9.5)	1.00
Mitral anulus anterior	1 (16.7)	0 (0)	0.222
Mitral anulus posterior	1 (16.7)	0 (0)	0.222
Procedural BP, mm Hg			
Max systolic	151.67±11.25	142.62±8.46	0.075
Max diastolic	91.67±5.16	88.10±6.80	0.316
Min systolic	96.67±5.16	91.90±5.80	0.082
Min diastolic	63.33±4.08	59.52±5.46	0.127

ACEI: Angiotensin converting enzyme inhibitor; ACT: Activated clotting time; ARB: Angiotensin receptor blocker; CCB: Calcium channel blocker; CRP: C-reactive protein; EF: Ejection fraction; eGFR: Estimated glomerular filtration rate; Fr: French; INR: International normalized ratio; LDL: Low-density lipoprotein; LAD: Left atrial diameter; LVEDD: Left ventricular end diastolic diameter; LV: Left ventricle; LCC: Left coronary cusp; LV-AMC: Left venticular outflow tract-aorto mitral continuity; IVST: Interventricular septal thickness; NT-pro BNP: N-terminal prohormone of brain natriuretic peptide; PVC: Premature ventricular complex; RCC: Right coronary cusp; RF: Radiofrequency; RVOT: Right venticular outflow tract; VT: Ventricular tachycardia.

Table S1. Procedural and demographic characteristics of patients with SCE: +

	Patient LV- 1	Patient LV-4	Patient LV-8	Patient LV-10	Patient LV-12	Patient LV-20
Gender	Female	Female	Female	Female	Male	Female
CHA_2DS_2-VASc	4	5	3	2	2	4
NT-pro BNP, pg/mL	1380	195	280	940	306	1100
Atrial fibrillation	-	-	-	-	-	-
Aortic valve sclerosis	-	+	-	-	-	-
LV EF (%)	50	55	65	55	60	65
Atherosclerotic aortic Disease	-	+	-	-	-	-
LV ablation locations	LV-Summit	LCC	LCC	Mitral anulus anterior	Mitral anulus posterior	LCC
Mapping	EnSite	EnSite	EnSite	EnSite	CARTO	CARTO
Procedure time, min	75	80	55	65	80	45
Approach to LV	Retrograde	Retrograde	Retrograde	Retrograde	Retrograde and transseptal	Retrograde
Total fluoroscopy time, min	10,2	7,2	5,5	6,2	9,2	4,3
Total RF duration, sec	440	320	350	440	490	345

EF: Ejection fraction; LV: Left ventricle; NT-pro BNP: N-terminal prohormone of brain natriuretic peptide; RF: Radiofrequency.



Graphical abstract

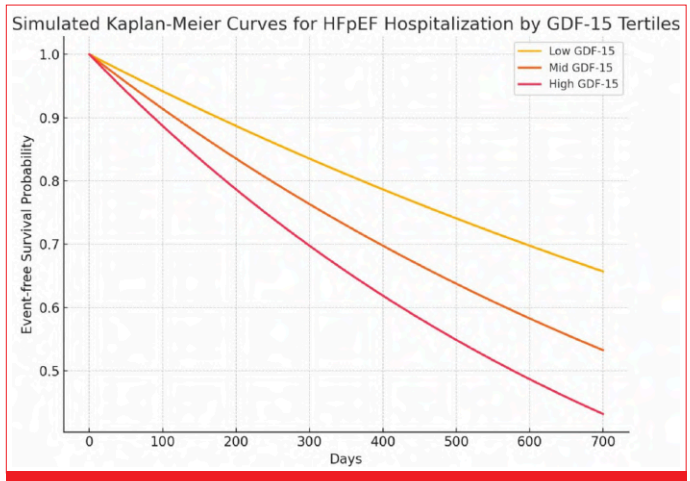


Figure 1. Kaplan-Meier Survival Curves for Heart Failure Hospitalization Stratified by GDF-15 Tertiles Patients in the high GDF-15 tertile exhibited significantly lower event-free survival over time compared to those in the low and mid tertiles, suggesting the prognostic value of GDF-15 in HFpEF.

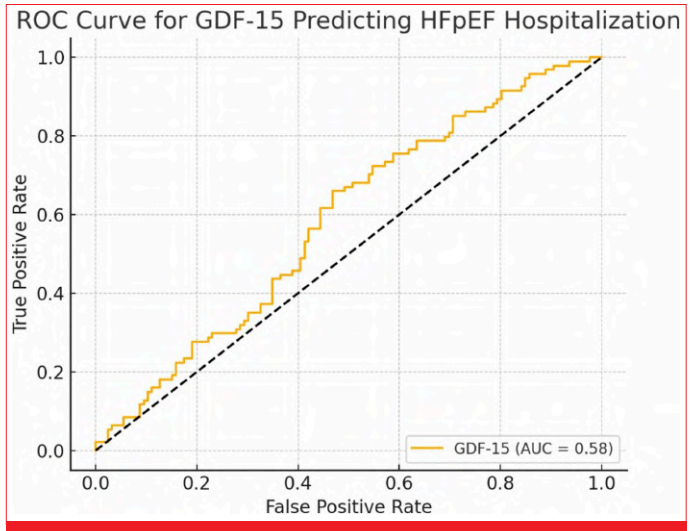


Figure 2. Receiver Operating Characteristic (ROC) Curve for GDF-15 in Predicting Hospitalization Due to HFpEF The ROC curve demonstrates the predictive performance of baseline GDF-15 levels for heart failure-related hospitalization. The area under the curve (AUC) was 0.78.

OP-069 [Heart Failure]

Prognostic value of growth differentiation factor-15 in predicting hospitalization due to heart failure with preserved ejection fraction

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Background and Aim: Heart failure with preserved ejection fraction (HFpEF) accounts for nearly half of all heart failure cases and is associated with high morbidity and frequent hospitalizations. Growth Differentiation Factor-15 (GDF-15), a biomarker of oxidative stress and inflammation, has emerged as a potential predictor of adverse cardiovascular outcomes. This study aimed to evaluate the prognostic value of baseline GDF-15 levels in predicting heart failure-related hospitalization in patients with HFpEF.

Methods: In this observational cohort study, 220 patients diagnosed with HFpEF (LVEF ≥50%) were enrolled and followed for a median of 18 months. Baseline serum GDF-15 levels were measured using an enzyme-linked immunosorbent assay (ELISA). The primary endpoint was hospitalization due to worsening heart failure. Cox proportional hazards regression was used to assess the predictive value of GDF-15, and receiver operating characteristic (ROC) analysis was conducted to determine optimal cut-off values.

Results: During follow-up, 72 patients (32.7%) were hospitalized due to HF exacerbation. Median baseline GDF-15 levels were significantly higher in hospitalized patients compared to non-hospitalized counterparts (1.783 pg/mL [IQR: 1.412-2.371] vs. 1.142 pg/mL [IQR: 896-1,508]; p<0.001). In multivariable analysis adjusting for age, NT-proBNP, eGFR, and NYHA class, GDF-15 remained independently associated with heart failure hospitalization

(HR: 1.68 per log unit increase; 95% CI: 1.31–2.15; p<0.001). The area under the ROC curve for GDF-15 predicting hospitalization was 0.78 (95% CI: 0.71–0.84) (Figure 2).

Conclusions: Elevated baseline GDF-15 levels are independently associated with increased risk of hospitalization in patients with HFpEF. Incorporation of GDF-15 into clinical risk models may improve early identification of high-risk patients and guide management strategies.

OP-070 [Heart Failure]

Effect of stem-cell therapy in heart failure with reduced ejection fraction

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Background and Aim: Heart failure with reduced ejection fraction (HFrEF) is a chronic and debilitating condition affecting multiple systems. Despite contemporary approaches, there is an unmet need for alternative medical approaches. Given the emerging potential of stem cell therapy in treating heart failure, this study focused on evaluating the efficacy and safety of umbilical cord mesenchymal stem cells (MSCs) in patients with HFrEF.

Methods: This study had a prospective group design which consisted of 10 consecutive HFrEF outpatients with a LVEF ≤40% at optimal medical treatment at the time of inclusion. Patients were referred to our cardiology clinic in order to receive MSCs upon the conventional treatments. During the first session, intracoronary 20 x 10^9 exosome particles were given. For the second and third sessions, 1 x 10^6 MSCs per kilogram were administered intravenously. The sessions were scheduled one weekly intervals. Kansas City Cardiomyopathy Questionnaire (KCCQ-12) score, left ventricular ejection fraction (LVEF), NT-proBNP leves, and general satisfaction with life (graded as unsatisfied, neutral or satisfied) were studied at basal and at three- months follow-up

Results: The mean age of study group was 50 ± 16.3 years. Whole patient cohort were male. Given the patients' general satisfaction with life, seven patients (70%) reported increased quality of life after the treatment, whereas three patients (30%) reported therapy neutrally influenced their quality of life (p=0.008). KCCQ-12 scores (42.3 [IQR 18.5, 42.4]; vs. 53.5 [IQR 32.5, 90.3] p=0.29), LVEF (27.4 ± 9 vs. 27.4 ± 9.9; p=1) and NT-proBNP levels (1264 [IQR 696, 4031]; vs. (937 [IQR 372, 3161]; p=0.51) showed no statistical difference at three months follow-up compared to baseline.

Conclusions: Treatment of HFrEF patients with stem cells could be effecient in terms of improved quality of life with no change in KCCQ-12 scores, LVEF and NT-proBNP levels.

Table 1. Results

Pt	Aetiology	Age	Sex	HT	DM	DL	Smoker	EF Basal	EF at 3 month	ARNI	Beta blocker	SGLT2I	MRA	AF	Device therapy	Basal NT-proBNP	NT-proBNP at 3 months	Basal KCCQ12 Score	KCCQ12 Score at 3 months	Quality of life
1	Ischemic	30	M	NO	NO	NO	YES	38	41	YES	YES	YES	YES	NO	No	1216	411	57	97	Satisfied
2	Ischemic	67	M	NO	NO	NO	YES	19	18	YES	YES	YES	YES	YES	CRT-D	986	1054	14	52	Satisfied
3	Ischemic	70	M	NO	NO	YES	NO	32	22	YES	YES	YES	YES	NO	ICD	821	820	69	55	Satisfied
4	Nonischemic	58	M	NO	NO	NO	NO	22	24	YES	YES	YES	YES	YES	CRT-D	7085	1682	0	31	Satisfied
5	Ischemic	54	M	NO	NO	YES	YES	44	41	YES	YES	YES	NO	NO	No	34	32	93	88	Satisfied
6	Ischemic	59	M	YES	YES	YES	NO	34	41	YES	YES	YES	YES	NO	No	1312	755	91	99	Satisfied
7	Nonischemic	28	M	NO	NO	NO	NO	25	15	YES	YES	YES	YES	YES	ICD	3013	7245	46	35	Neutral
8	Ischemic	36	M	YES	YES	YES	NO	20	29	YES	YES	YES	YES	NO	ICD	321	255	39	33	Neutral
9	Nonischemic	34	M	NO	NO	NO	NO	20	21	YES	YES	YES	YES	NO	CRT-D	12823	16755	30	69	Satisfied
10	Ischemic	64	M	YES	NO	NO	NO	20	23	YES	YES	YES	YES	NO	CRT-D	2389	1800	20	16	Neutral

OP-072 [Heart Failure]

Protective effects of Sacubitril/Valsartan against radiation-induced cardiotoxicity

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Background and Aim: With the increasing incidence of cancer among adults, radiotherapy (RT) has emerged as a cornerstone in the treatment of various malignancies. Nevertheless, the use of ionizing radiation is frequently associated with collateral damage to adjacent healthy tissues, including those surrounding the primary tumor. Sacubitril/valsartan (Sac/Val), a therapeutic agent commonly employed in the management of heart failure with reduced ejection fraction (HFrEF), has been shown to exert notable cardioprotective effects in several studies. The present study was designed to investigate the potential cardioprotective role of Sac/Val in mitigating RT-induced cardiotoxicity.

Methods: A total of 40 male Wistar albino rats were subjected to a 10-day acclimatization period to adapt to the laboratory environment. Subsequently, the animals were randomly allocated into four experimental groups, each consisting of 10 rats (Control=10, Sac/Val=10, RT=10, RT+Sac/Val=10). The RT and RT+Sac/Val groups received a single dose of 20 Gray (Gy) x-ray irradiation, administered to a 4 x 4 cm area at a rate of 0.60 Gy/min. Concurrently, rats in the Sac/Val and RT+Sac/Val groups were treated with a daily oral gavage of sacubitril/valsartan at a dose of 60 mg/kg. During the second week of the experiment, all animals underwent echocardiographic and electrocardiographic evaluations. Cardiotoxic effects were further assessed through histopathological examination (Figure 1).

Results: No significant changes in body weight were observed in the Sac/Val and control groups; however, a statistically significant reduction in body weight was detected in both the RT and RT+Sac/Val groups ($p<0.001$). Electrocardiographic (ECG) parameters showed no significant baseline differences among groups. By week 2, ST-segment elevation occurred in 40% of the RT group versus 10% of the RT+Sac/Val group ($p=0.022$), with the RT group also exhibiting significantly elevated heart rate, prolonged QTc, PR, and QRS intervals ($p<0.001$). Echocardiographic analysis revealed greater reductions in ejection fraction (EF) and fractional shortening (FS) in the RT group compared to RT+Sac/Val (EF: 59.40 ± 5.91% vs. 73.90 ± 7.37%; FS: 37.60 ± 5.68% vs. 44.70 ± 4.49%; $p<0.001$), alongside significant increases in left ventricular end-diastolic diameters (LVEDD) and left ventricular end-systolic diameters (LVESD) ($p=0.013$) (Table 1, Figure 2,3). Histopathological evaluation showed severe myocardial damage in the RT group, including necrosis and inflammation, which was markedly reduced in the RT+Sac/Val group ($p<0.001$) (Table 2, Figure 4-7). These findings suggest Sac/Val attenuates RT-induced cardiotoxicity at functional, electrical, and histological levels.

Conclusions: This study demonstrated the positive remodeling effects of Sac/Val against left ventricular remodeling and development of left ventricular dysfunction in an animal model of RT-induced cardiomyopathy.

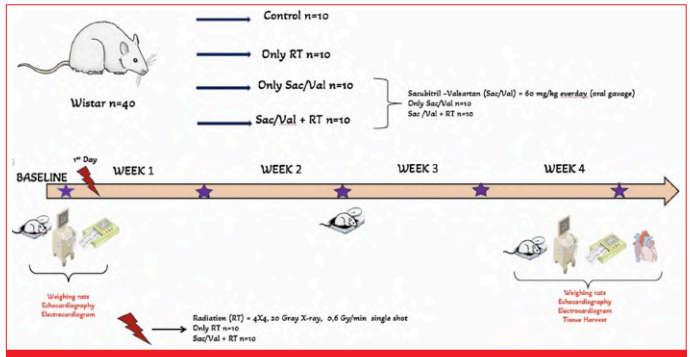

Figure 1. Study design.

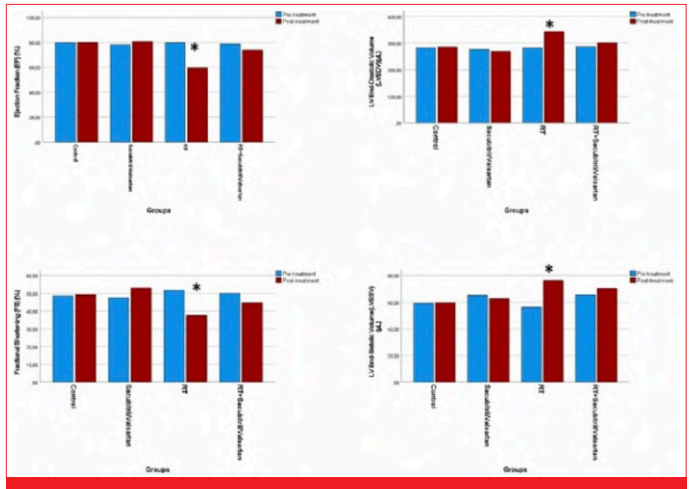

Figure 2. Echocardiographic parameters. Control, sacubitril-valsartan, radiotherapy, radiotherapy + sacubitril-valsartan. *Significant at 0.05 level according to Two-way ANOVA.

Table 1. Weight, echocardiographic and electrocardiographic measurements of the study groups

	Control (a)	Sacubitril/ Valsartan (b)	RT (c)	RT + Sacubitril/ Valsartan (d)	Pgroups	Pmeasures	Pgroups* measures
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD			
Baseline weight	320.60 ± 43.88	302.80 ± 27.45	310.20 ± 23.26	298.80 ± 30.48	0.461		
Final Weight	323.64 ± 44.56	305.80 ± 31.23	265.61 ± 43.11	281.71 ± 39.57	<0.001	<0.001	a>c, b>c, b>d, d>c, a>d
Baseline EF (%)	79.60 ± 3.50	78.10 ± 2.51	79.70 ± 2.71	78.60 ± 2.45	0.525		
Final EF (%)	80.00 ± 3.09	80.70 ± 2.86	59.40 ± 5.91	73.90 ± 7.37	<0.001	<0.001	a<c, b<c, b<d, d<c
Baseline FS (%)	45.50 ± 3.77	47.40 ± 2.59	51.70 ± 4.85	50.10 ± 4.38	0.106		
Final FS (%)	49.30 ± 3.74	53.10 ± 9.63	37.60 ± 5.68	44.70 ± 4.49	<0.001	<0.001	a<c, b<c, b<d
Baseline End-diastolic Volume (mL)	281.90 ± 12.08	276.30 ± 13.98	281.80 ± 13.48	286.30 ± 17.73	0.503		
Final End-diastolic Volume (mL)	285.60 ± 9.93	268.50 ± 14.46	343.10 ± 27.57	300.80 ± 22.03	<0.001	<0.001	a<c, b<c, b<d, d<c
Baseline End-systolic Volume (mL)	59.20 ± 9.78	65.10 ± 7.60	56.20 ± 6.28	65.50 ± 9.36	0.047		
Final End-systolic Volume (mL)	59.50 ± 9.19	62.50 ± 10.35	76.30 ± 12.93	70.40 ± 9.16	0.013	<0.001	a<c, b<c
ST elevation in week 2 ECG (%)	0 (0%)	0 (0%)	4 (40%)	1 (10%)	0.022		
Baseline Heart Rate (bpm)	315.20 ± 22.00	325.70 ± 32.43	316.40 ± 17.53	316.00 ± 15.83	0.708		
Final Heart Rate (bpm)	313.90 ± 21.34	309.40 ± 35.26	370.90 ± 22.20	331.80 ± 13.55	<0.001	<0.001	a<c, b<c, d<c
Basal QTc (msn)	168.50 ± 16.32	161.90 ± 14.51	166.20 ± 7.75	169.90 ± 13.16	0.565		
Final QTc (msn)	169.10 ± 16.96	157.20 ± 12.32	197.60 ± 20.79	176.60 ± 15.02	<0.001	<0.001	a<c, b<c, d<c
Basal PR (msn)	44.00 ± 6.71	40.70 ± 3.52	44.30 ± 3.71	45.20 ± 4.39	0.189		
Final PR (msn)	44.60 ± 6.94	40.60 ± 3.97	58.50 ± 11.35	48.00 ± 4.42	<0.001	<0.001	a<c, b<c, d<c
Basal QRS (msn)	13.95 ± 2.03	13.50 ± 0.97	13.70 ± 1.25	13.70 ± 1.49	0.927		
Final QRS (msn)	14.38 ± 2.38	12.70 ± 1.33	18.20 ± 2.61	15.00 ± 1.82	<0.001	<0.001	a<c, b<c, d<c

a,b,c,d: Lower case letters denote the significant pairwise comparisons between study groups according to Tukey HSD post-hoc test. bpm: Beats per minute; mL: Mililiters; EF: Ejection fraction; FS: Fractional shortening; QTc: Calculated QT interval

Table 2. Histopathologic results of animal study groups

Parameters	Control (a)	Sacubitril/Valsartan (b)	RT (c)	RT + Sacubitril/Valsartan (d)
Eosinophilia in Myofibrils	0 ± 0	1.20 ± 0.42	2.90 ± 0.31	2.10 ± 0.31
Fragmanted Fibrilles	0 ± 0	0 ± 0	2.70 ± 0.48	2.10 ± 0.56
Interstitial Edema	0 ± 0	1.20 ± 0.42	2.90 ± 0.31	2.10 ± 0.31
Myofiber Waviness	0 ± 0	0 ± 0	2.9 ± 0.31	1.30 ± 0.67

Values are expressed as mean ± SD. RT: Radiotherapy. Statistically significant differences at 0.05 level across pairwise groups according to Tukey's HSD post hoc test. * Statistically significant differences between control and other groups (bp<0.001, cp<0.001, and dp<0.001). ** Statistically significant differences between RT and other groups (dp<0.001). *** Statistically significant differences between RT and other groups (bp<0.001, and dp<0.001). **** Statistically significant differences between RT and other groups (p<0.001).

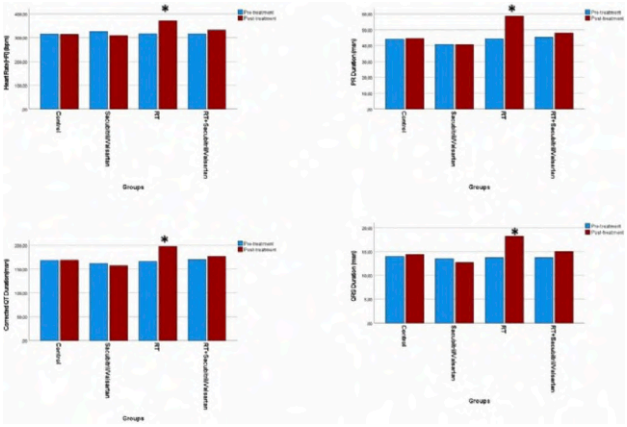

Figure 3. Electrocardiogram parameters. Control, sacubitril-valsartan, radiotherapy, radiotherapy+sacubitril-valsartan. *Significant at 0.05 level according to Two-way ANOVA.

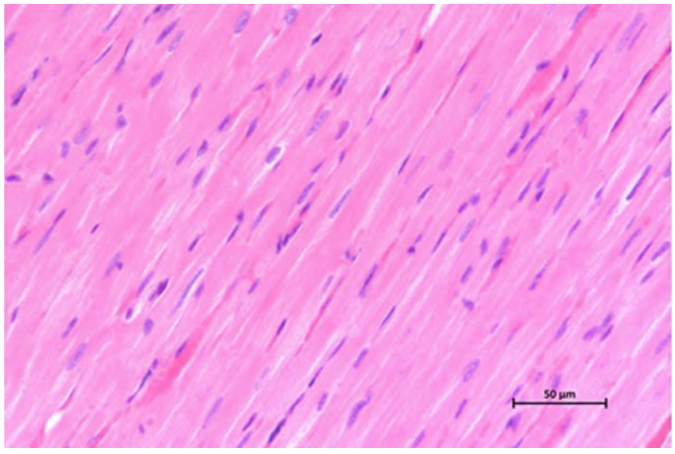

Figure 4. Control group. Myocardial layer. Cardiac muscle fibers, nuclei (arrow) and arrangement of fibers were observed in near-normal histology (HEX40).

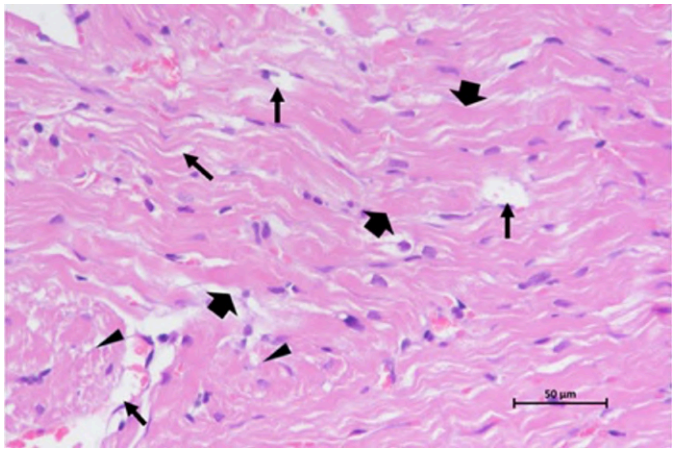

Figure 5. Radiotherapy group. Significant myofiber waviness due to myocardial injury, intense interstitial edema (arrow), dense fragmented fibrin deposition in multiple regions (arrowhead), and areas of intense eosinophilic staining within the myofibrils (thick arrow) were observed (HEX40).

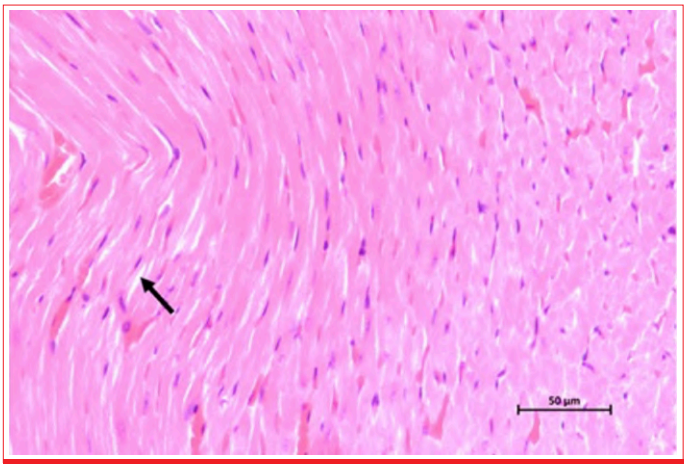

Figure 6. Sacubitril-Valsartan group. In most regions, cardiac histology appeared nearly normal, with only subtle interstitial edema observed (HEX40).

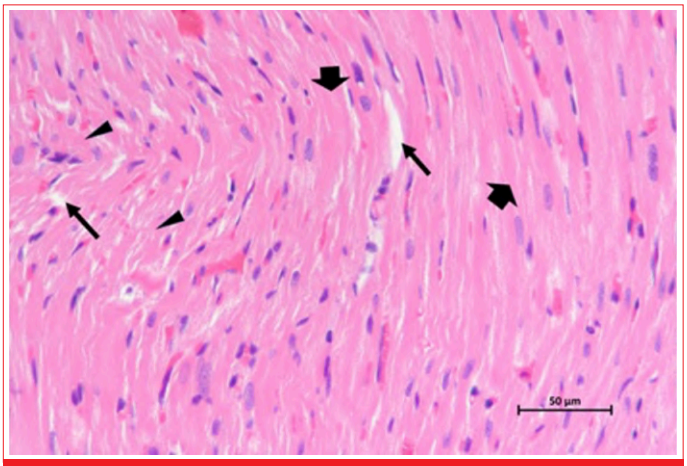

Figure 7. Radiotherapy+Sacubitril-Valsartan group. Compared to the injured group, reduced interstitial edema (arrow), decreased fragmented fibrin deposition (arrowhead), and fewer eosinophilic areas within the myofibrils were observed (HEX40).

OP-073 [Heart Failure]

Clinical approaches in heart failure: Perspectives of cardiologists (HF-PERSPECTIVE)

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Background and Aim: Heart failure (HF), is an increasingly significant health problem due to its high rates of hospitalizations and mortality, for which guideline-directed medical therapy is recommended to improve outcomes. This study aims to evaluate the approaches of cardiologists in the diagnosis, treatment, and follow-up processes of patients with HF through a survey-based methodology.

Methods: A survey study consisting of 28 questions, was prepared online using Google Forms to reflect cardiologists' approaches to patients with HF (particularly HF with reduced ejection fraction, HFrEF). The survey was distributed via social media and WhatsApp groups. The survey questions were structured to cover the following domains. Demographic information, Adherence to Guidelines, Diagnostic and Therapeutic Strategies, Patient Follow-Up, Challenges, Individual-Institutional Practice Variations. The collected data were analyzed.

Results: Participants were represented from all seven geographical regions of Türkiye: Marmara (29.6%), Central Anatolia (23.7%), and Aegean (17.3%) regions. A total of 493 cardiology residents (37.5%), specialists (27%), and faculty members (35.5%) participated in the survey. ARNI emerged as the first-choice drug class that most cardiologists consider initiating in patients with HFrEF. When asked which medication they would prefer if they could use only one, 64% of participants responded that they would choose ARNI. The majority of participants reported that they do not use urine sodium measurements or biomarkers in decongestion management. Among participants, 62.9% preferred initiating ARNI at or before discharge in newly diagnosed HF patients if affordable, and 94.9% reported willingness to start SGLT2i during hospitalization or at discharge. Metoprolol was identified as the most commonly used beta-blocker in patients with HFrEF. Among patients already receiving ACEi/ARB therapy, 54.8% of participants preferred a starting ARNI dose of 49/51 mg, while in ACEi/ARB-naive patients, 83.8% of participants preferred a starting dose of 24/26 mg. In decongestion therapy, 62.7% of participants reported using acetazolamide. The majority of participants reported recommending sodium restriction in patients with HFrEF. Within the first three months, 63.5% of participants stated that they could achieve target therapy doses, while in daily practice, participants reported that they could reach target doses in 45.4% of their patients.

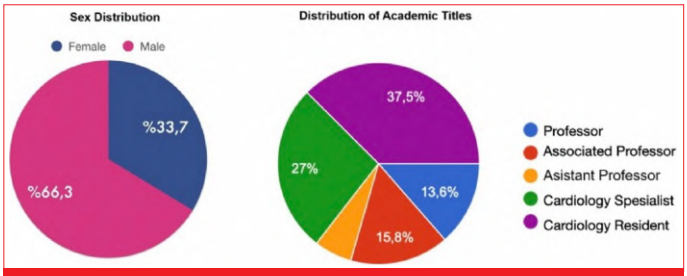

Figure 1. Sex and academic title distributions.

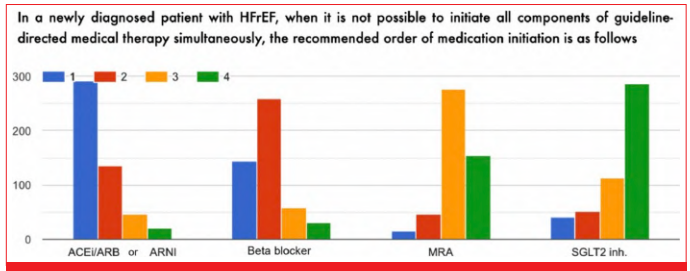

Figure 2. Physicians' preferred sequence of initiating guideline-directed medical therapy in newly diagnosed HFrEF when simultaneous initiation is not feasible.

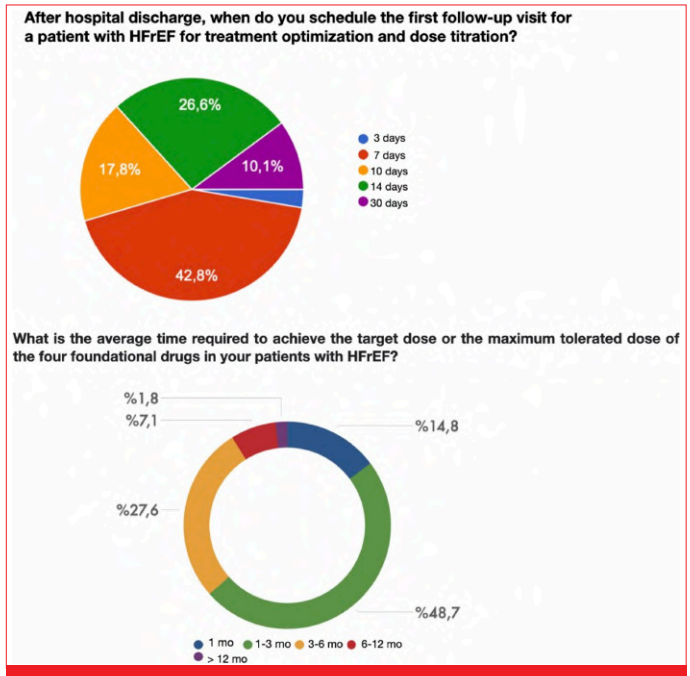

Figure 3. Timing of first follow-up visit and titration period for foundational therapies in patients with HFrEF.

Conclusions: This survey was conducted with a high participation rate, including 493 cardiologists, providing a broad and representative perspective on physician practices in the management of HFrEF in Türkiye. The findings of this survey revealed significant gaps between real-world clinical practices of cardiologists in Türkiye and the recommendations of clinical guidelines. In conclusion, this large-scale survey provides valuable insights into the adherence to guidelines and the practical challenges faced by cardiologists in Türkiye in the management of HFrEF.

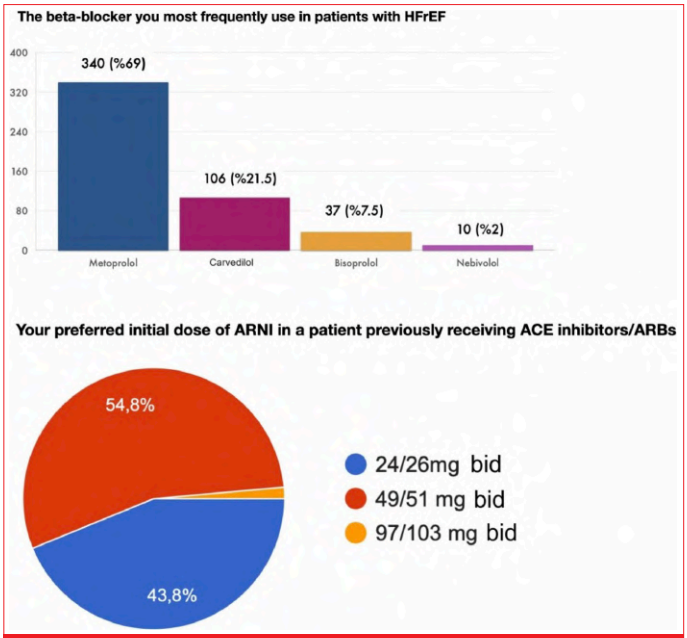

Figure 4. Physicians' preferences regarding the choice of beta-blocker and initial ARNI dosing in patients with HFrEF previously treated with ACE inhibitors/ARBs.

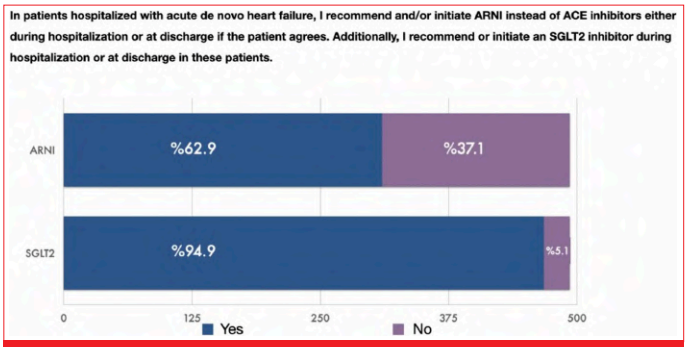

Figure 5. Physicians' preferences for initiating ARNI and SGLT2 inhibitors during hospitalization or at discharge in patients with acute de novo heart failure.

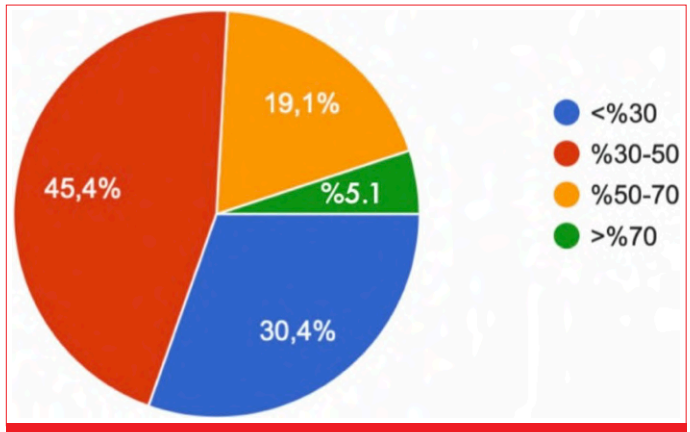

Figure 6. Percentage of HFrEF patients achieving target doses in daily practice.

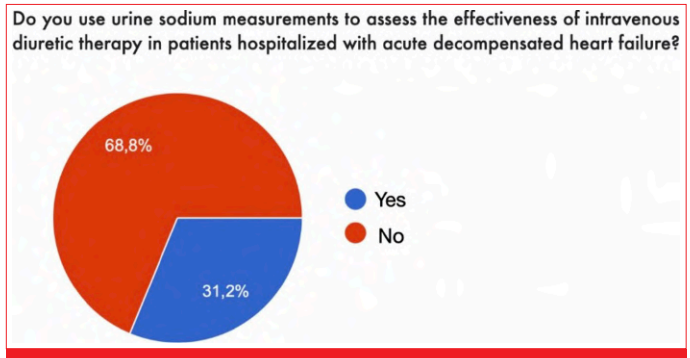

Figure 7. Physician preferences regarding the use of urinary sodium measurements to assess diuretic response in acute decompensated heart failure.

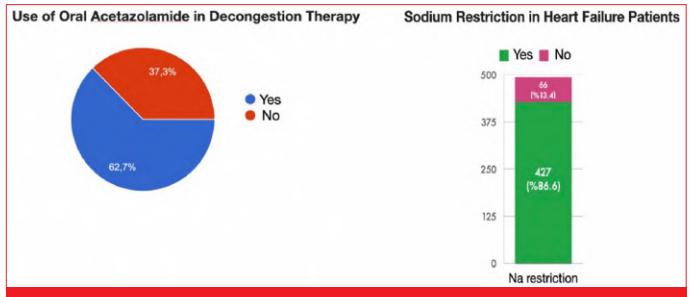

Figure 8. Physician preferences regarding the use of oral acetazolamide in decongestion therapy and sodium restriction in patients with heart failure.

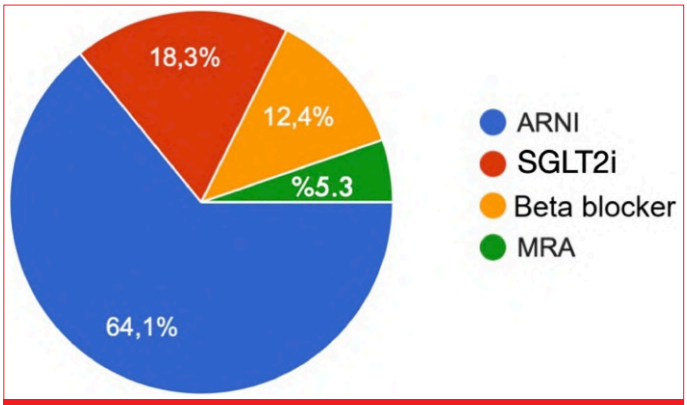

Figure 9. The drug preferred by physicians when given the option to choose only one treatment for patients with HFrEF.

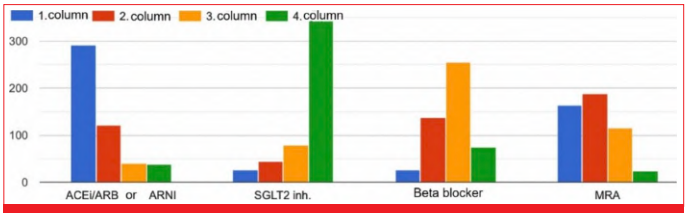

Figure 10. First foundational drug to discontinue or reduce in hypotensive patients.

OP-074 [Heart Failure]

Prevalence, predictors, and clinical outcomes of heart failure in patients with atrial fibrillation treated with factor Xa inhibitors

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Background and Aim: Atrial fibrillation (AF) and heart failure (HF) often coexist in patients. We aimed to determine the prevalence, clinical characteristics, predictors, and outcomes of HF among a nationwide real-world AF population treated with factor Xa inhibitors.

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 in patients with AF with and without HF.

Results: The present study included a cohort of 1,158 patients diagnosed with AF and treated with factor Xa inhibitors. Overall, 40.0% (463 patients) of the study population had HF. Patients with HF were older, with a higher proportion of patients aged ≥75 years than those without HF. Compared with those without HF, patients with HF were more often male, had a higher burden of cardiovascular risk factors such as diabetes and dyslipidemia, and were more likely to have a history of coronary artery disease, myocardial infarction, chronic kidney disease, and anemia. Patients with HF were more likely to have permanent AF, whereas those without HF were more likely to experience paroxysmal AF. The prescription rates of the different factor Xa inhibitors were similar in patients with and without HF. However,

the prescription of the appropriate standard dose of factor Xa inhibitors was significantly lower in patients with HF than in those without (57.5% vs. 77.6%, p<0.001, Figure 1). The concomitant use of antiplatelet drugs with factor Xa inhibitors was markedly higher in patients with HF. In the logistic regression model, advanced age, history of myocardial infarction, presence of diabetes, chronic kidney disease, anemia, and a one unit increase in CHA_2DS_2-VASc score were significantly associated with the presence of HF in patients with AF. Conversely, the presence of paroxysmal AF (reference group: permanent AF) was associated with the absence of HF (Figure 2). During a median follow-up of 13 months (IQR 3 months), 140 (12.1%) patients died. Patients with AF and HF exhibited a higher rate of all-cause and cardiovascular mortality than those without HF (18.4% vs. 7.9%, p<0.001; and (9.0% vs. 3.2%, p<0.001, respectively). The rates of ischemic events, including myocardial infarction, stroke, and systemic embolism, were comparable between the groups during the follow-up period. The overall bleeding rate was significantly higher in patients with AF and HF than in those with AF without HF (18.8% vs. 14.1%, p=0.033, Figure 3).

Conclusions: In a large, real-world cohort of patients with AF, we found that HF was associated with an increased risk of cardiovascular and all-cause mortality. Our results highlight the unmet need for establishing novel and enhanced therapeutic options and algorithms to address the challenges faced by patients with AF and HF.

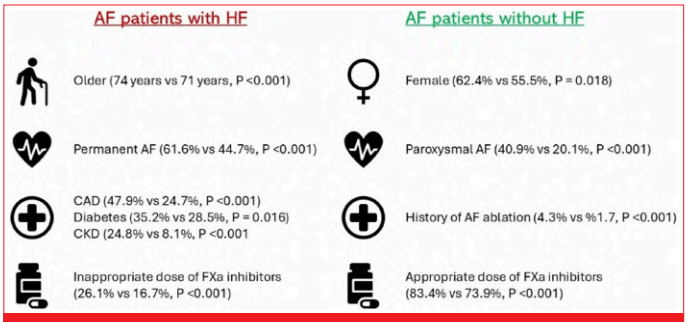

Figure 1.

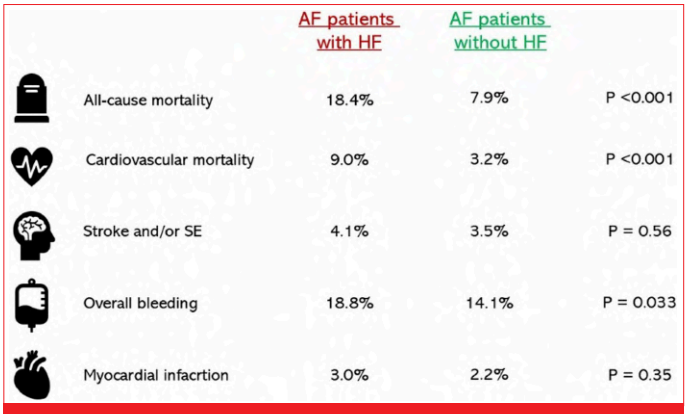

Figure 2.

Table 2. Logistic regression model for clinical variables associated with HF among patients with AF

Variable	Model*		
	Odds ratio	95% CI	P-value
Male sex	1.12	0.86–1.45	0.41
Age group			
• <65 years *(reference)*			
• 65–75 years	1.69	1.19–2.41	0.003
• ≥75 years	3.10	1.86–5.19	<0.001
AF type			
• Permanent AF *(reference)*			
• New-onset AF	0.51	0.21–1.22	0.12
• Persistent AF	0.97	0.64–1.47	0.89
• Paroxysmal AF	0.34	0.25–0.48	<0.001
CHA_2DS_2-VASc score (per 1 unit increase)	2.11	1.81–2.45	<0.001
Myocardial infarction	3.66	2.54–5.27	<0.001
Diabetes	1.94	1.38–2.72	<0.001
Chronic kidney disease	2.51	1.59–3.95	<0.001
Anemia	1.53	1.06–2.20	0.022

OP-076 [Lipid / Preventive Cardiology]

Comparison of hospital anxiety and depression scale scores among cardiac pathologies: A questionnaire-based study

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Background and Aim: While the physiological burden of cardiovascular diseases is well established, their psychological consequences are often underappreciated in clinical practice. However, anxiety and depression are known to influence both the quality of life and clinical outcomes of cardiac patients. In this context, our study aimed to evaluate the levels of anxiety and depression in patients with various cardiac pathologies using the Hospital Anxiety and Depression Scale (HADS) and to explore how these scores relate to sociodemographic and clinical parameters.

Methods: This was a cross-sectional, observational study involving 442 adult patients followed in the cardiology outpatient clinic of our institution. All participants completed the HADS questionnaire, which includes two subscales—HADS-A for anxiety and HADS-D for depression. Patients were stratified into diagnostic subgroups according to their primary and secondary cardiac conditions: Coronary artery disease (CAD), heart failure (HF), atrial fibrillation (AF),valvular heart disease, hyperlipidemia, hypertension (HT) healthy control group. HADS scores were calculated separately within each diagnostic category, and statistical comparisons were performed among these groups. Additionally, demographic factors such as age, sex, education level, marital status, and income were included in the analysis.

Results: The mean age was 60.1 ± 14.6 years; 55.2% were male. The mean depression score was 7.0 ± 3.6, and the mean anxiety score was 7.2 ± 3.8. Key findings by diagnosis: Patients with atrial fibrillation (AF) had significantly higher depression scores compared to those without AF (p=0.001). Patients with hypertension (HT) had significantly higher anxiety scores (p=0.039). Heart failure, CAD, and valvular disease were not associated with significantly different HADS scores. The presence of multiple diagnoses was associated with higher depression and anxiety scores compared to patients with

a single diagnosis or healthy controls (p<0.01). Sociodemographic findings: Women had significantly higher HADS scores than men (p<0.05). Lower education and older age were associated with increased scores. Married participants had unexpectedly higher anxiety and depression scores compared to singles. A positive but weak correlation was observed between age and both HADS-A and HADS-D scores.

Conclusions: These findings highlight that specific cardiac diagnoses—particularly atrial fibrillation and hypertension—are associated with a greater psychological burden. This may relate to the chronic and unpredictable nature of these conditions, leading to emotional distress. Moreover, demographic variables such as female gender, lower education level, and advanced age were significantly linked with higher HADS scores, suggesting a multifactorial origin of emotional symptoms in cardiology patients.Patients with AF and HT are at higher risk for emotional distress. Routine psychological screening in cardiology practice is essential for holistic patient care.

Table 1. HADS scores by cardiac diagnosis

Diagnosis	Depression Score (Mean ± SD)	p-value (Depression)	Anxiety Score (Mean ± SD)	p-value (Anxiety)
Healthy Control	4.71 ± 3.54	0.001	5.37 ± 3.59	0.010
AF	8.39 ± 3.71	0.001	8.00 ± 3.77	0.101
HT	7.17 ± 3.46	0.442	7.72 ± 3.81	0.039
CAD	7.24 ± 3.71	0.399	6.93 ± 3.84	0.302
HF	6.66 ± 3.32	0.558	6.46 ± 4.25	0.211
Valvular Disease	6.37 ± 3.95	0.260	7.26 ± 4.14	0.975

AF: Atrial fibrillation; CAD: Coronary artery disease; HF: Heart failure.

Table 2. HADS scores by demographic variables

Variable	Depression Score (Mean ± SD)	p-value (Depression)	Anxiety Score (Mean ± SD)	p-value (Anxiety)
Gender - Male	6.65 ± 3.72	0.024	6.77 ± 3.99	0.004
Gender - Female	7.43 ± 3.45	0.024	7.83 ± 3.64	0.004
Primary school	7.89 ± 3.68	0.001	7.80 ± 4.11	0.007
University	5.14 ± 3.06	0.001	6.18 ± 3.94	0.007
Single	5.55 ± 4.03	0.004	5.64 ± 3.64	0.003
Married	7.17 ± 3.54	0.004	7.44 ± 3.87	0.003

OP-077 [Coronary Artery Disease / Acute Coronary Syndrome]

Inflammatory biomarkers predicting contrast-induced acute kidney injury in elderly patients with ST-segment elevation myocardial infarction

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Background and Aim: The inflammatory response is critically important in ST-segment elevation myocardial infarction (STEMI). The systemic immune-inflammation index (SII) and systemic inflammation response index (SIRI), novel inflammatory biomarkers, have been linked to the determination of outcomes in various diseases. The aim of the current study was to examine the relation of the SII and SIRI with contrast-induced acute kidney injury (CI-AKI) in elderly subjects with STEMI undergoing primary percutaneous coronary intervention (pPCI).

Methods: All patients diagnosed with STEMI between November 2020 and September 2024 were screened, and patients aged over 70 were retrospectively analyzed in the present study. The patients were divided into two groups according to CI-AKI development. The SII and SIRI were calculated based on the peripheral blood counts. A receiver operating characteristic (ROC) curve analysis was performed to determine the sensitivity and specificity of the SII and SIRI in predicting CI-AKI. Additionally, multivariable logistic regression models were employed to investigate the associations between inflammatory indices and the incidence of CI-AKI in elderly patients with STEMI.

Results: A total of 263 participants were included (mean age 77.67 ± 6.20, 56% women). Both the SII and SIRI were higher in the CI-AKI group than in the non-CI-AKI group (3252 ± 2257, 1097 ± 991 p<0.001 for SII; 12.1 ± 4.54, 4.86 ± 2.42 p<0.006 for SIRI). In the receiver operating characteristic analysis, the SII and SIRI showed the highest area under curve (AUC) compared with other inflammatory parameters. The AUC of the SII and SIRI were 0.903

Table 1. The demographic and clinical features of elderly patients with STEMI

	All (*n* = 263)
Variables	***n* (%)**
Female	150 (57%)
Age(years)	77.5 ± 6.2
Smoking	44 (16.7%)
Diabetes	75 (28.5%)
Dyslipidemia	52 (19.8%)
Hypertension	178 (67.7%)
AF	28 (10.6%)
Killip class > 1	74 (28.1%)
Previous history of CAD	92 (34.9%)
COPD	33 (12.6%)
Periferal artery disease	12 (4.6%)
Malignancy	20 (7.6%)
CHF	25 (9.5%)
Anterior STEMI	98 (37.2%)
Bleeding	11 (4.2%)
Vasopressor and inotrope Use	59 (22.4%)
Contrast media (ml)	166.7 ± 41.8
ACEI/ARB	90 (34.2%)
Beta blockers	145 (55.1%)
Statins	215 (81.7%)
Ticagrelor	107 (40.7%)
Clopidogrel	156 (59.3%)

Table 2. The demographic and clinical features of patients with and without CI-AKI in elderly patients with STEMI

	CI-AKI (*n* = 113)	Non-CI-AKI (*n* = 150)	*p* Value
Variables *n* (%)			
Female	64 (56.6%)	86 (57.3%)	0.726
Age (years)	78.7 ± 6.3	76.6 ± 5.9	0.005
Smoking	32 (21%)	12 (11%)	0.02
Diabetes	34 (30.1%)	41 (27.3%)	0.624
Dyslipidemia	27 (24%)	25 (17%)	0.145
Hypertension	96 (64%)	82 (73%)	0.141
AF	12 (10.6%)	16 (10.7%)	0.975
Killip class > 1	41 (36.3%)	33 (22%)	0.011
Previous history of CAD	42 (38.7%)	50 (33%)	0.149
COPD	12 (10.6%)	21 (14%)	0.413
Periferal artery disease	6 (5.3%)	6 (4%)	0.614
Malignancy	9 (8%)	11 (7%)	0.853
CHF	16 (14.2%)	9 (6%)	0.026
Anterior STEMI	50 (44%)	48 (32%)	0.046
Bleeding	8 (7.1%)	3 (2%)	0.042
Vasopressor and inotrope Use	34 (30%)	25 (17%)	0.01
Contrast media (ml)	167.8 ± 38.4	163.3 ± 43.6	0.667
ACEI/ARB	40 (35.4%)	50 (33%)	0.223
Beta blockers	62 (54.9%)	83 (55.3%)	0.824
Statins	90 (79.6%)	125 (83.3%)	0.882
Ticagrelor	45 (39.8%)	62 (41.3%)	0.726
Clopidogrel	68 (60.2%)	88 (58.7%)	0.741

Table 3. The laboratory features of elderly patients with STEMI

Laboratory Data	All (*n* = 263)
White blood cell count, $\times10^3$/μL	11.93 ± 4.24
Hemoglobin, g/dL	12.71 ± 2.1
Platelet count, $\times10^3$/μL	260 ± 30.38
Neutrophil count, $\times10^3$/μL	8.99 ± 3.78
Lymphocyte count, $\times10^3$/μL	1.76 ± 1.19
Monocyte count, $\times10^3$/μL	1.23 ± 0.84
Serum creatinine at admission, mg/dL	1.31 ± 0.83
Maximum serum creatinine, mg/dL	1.76 ± 1.26
CRP at admission, mg/L	25.9 ± 6.3
Maximum CRP, mg/L	70.1 ± 58.6
Glucose at admission, mg/dL	125.8 ± 49.8
Troponin I at admission	2527 ± 1074
Peak troponin I	7553 ± 4933
LVEF	46.7 ± 10.3
SII	2023.61 ± 196.92
SIRI	6.83 ± 2.69

Abbreviations: CI-AKI, contrast-induced acute kidney injury; CRP, C-reactive protein; SII, systemic immune-inflammation index; SIRI, systemic inflammation response index; LVEF, left ventricular ejection fraction.

and 0.867 (p<0.001). In multivariate logistic regression analysis, the SII and SIRI were found as independent predictors of CI-AKI.

Conclusions: The SII and SIRI were found to be important markers for predicting post-procedural CI-AKI in elderly patients with STEMI.

Table 4. The laboratory features of patients with and without CI-AKI in elderly patients with STEMI

	CI-AKI (*n* = 113)	Non-CI-AKI (*n* = 150)	*p* Value
Laboratory Data			
White blood cell count, $\times10^3$/μL	13.22 ± 4.52	10.4 ± 3.26	<0.001
Hemoglobin, g/dL	12.49 ± 2.1	12.85 ± 2.01	0.174
Platelet count, $\times10^3$/μL	286.93 ± 81.8	239.65 ± 73	<0.001
Neutrophil count, $\times10^3$/μL	10.81 ± 4.14	7.62 ± 2.8	<0.001
Lymphocyte count, $\times10^3$/μL	1.07 ± 0.38	2.27 ± 1.0	<0.001
Monocyte count, $\times10^3$/μL	1.34 ± 1.0	1.17 ± 0.82	0.134
Serum creatinine at admission, mg/dL	1.29 ± 0.58	1.32 ± 0.68	0.765
Maximum serum creatinine, mg/dL	2.23 ± 1.32	1.39 ± 1.1	<0.001
CRP at admission, mg/L	25.53 ± 6.8	26.29 ± 5.0	0.905
Maximum CRP, mg/L	90.37 ± 69.3	66.94 ± 50	<0.001
Glucose at admission, mg/dL	125.5 ± 48.9	126.5 ± 50.1	0.892
Troponin I at admission	2673 ± 1123	2420 ± 1038	0.790
Peak troponin I	10100 ± 6827	4529 ± 3562	0.003
LVEF	44.10 ± 10.8	48.60 ± 9.80	<0.003
SII	3252.35 ± 225.7	1097.95 ± 99.1	<0.001
SIRI	12.1 ± 4.54	2.86 ± 1.48	<0.006

Abbreviations: CI-AKI, contrast-induced acute kidney injury; CRP, C-reactive protein; SII, systemic immune-inflammation index; SIRI, systemic inflammation response index; LVEF, left ventricular ejection fraction.

Table 5. Univariate and multivariate logistic regression analyses showing the independent predictors of CI-AKI after pPCI in elderly patients with STEMI

	Univariate		Multivariate	
	OR (95%CI)	*p* Value	OR (95%CI)	*p* Value
Age	1.058 (1.016–1.101)	0.006	1.030 (0.967–1.097)	0.356
Gender (female)	1.627 (1.008–2.699)	0.05		
Max. CRP	1.006 (1.003–1.009)	<0.001	1.004 (0.998–1.010)	0.222
Max. hsTnI	1.006 (1.002–1.010)	0.003	1.005 (0.997–1.016)	0.227
Hemoglobin	0.922 (0.819–1.037)	0.175		
Killip > 1	2.019 (1.171–3.480)	0.011	0.940 (0.355–2.488)	0.901
Glucose at admission	1.005 (1.001–1.010)	0.022	1.002 (0.996–1.008)	0.462
Creatinine at admission	0.955 (0.707–10290)	0.765		
Anterior STEMI	1.670 (1.007–2.769)	0.047	1.282 (0.564–2.916)	0.553
SII	1.007 (1.001–1.011)	<0.001	1.008 (1.003–1.020)	<0.001
SIRI	1.569 (1.401–1.757)	<0.001	1.231 (1.057–1.433)	0.008

Abbreviations: Max-CRP, maximum high-sensitivity C-reactive protein; Max hsTnI, maximum high-sensitivity troponin I; OR, odds ratio; SII, systemic immune-inflammation index; SIRI, systemic inflammation response index.

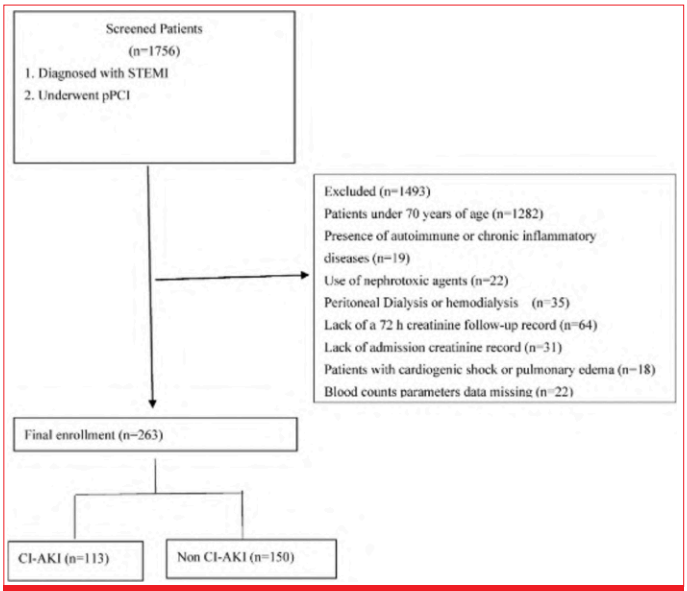

Figure 1. Flow chart of patients enrolment. pPCI, primary percutaneous coronary intervention; CI-AKI, contrast-induced acute kidney injury; STEMI, ST elevation myocardial infarction.

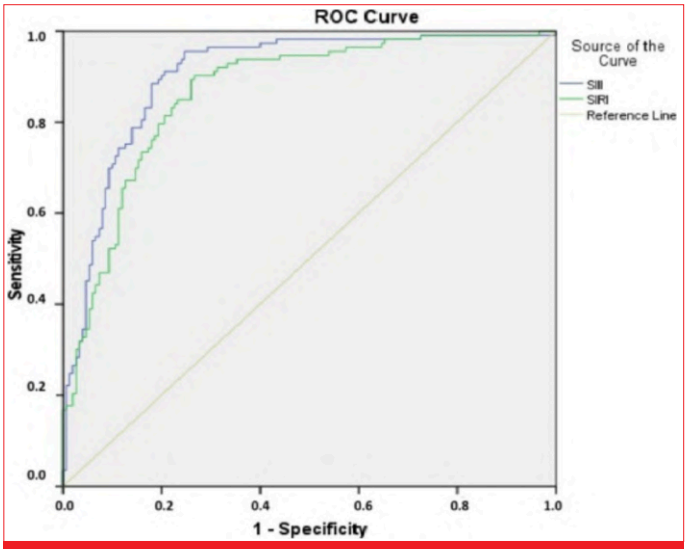

Figure 2. Receiver operating characteristic (ROC) curve with calculated area under the curve and optimal cut-off point for the SII and SIRI to identify the presence of CI-AKI after pPCI in elderly patients with STEMI.

OP-078 [Interventional Cardiology / Carotid and Peripheral Vascular]

Prognostic value of glucose lymphocyte ratio for occurrence of stroke in patients with carotid artery stenosis

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Background and Aim: Carotid atherosclerotic disease is one of the leading causes of stroke. Previous studies have investigated the effects of metabolic dysregulation and inflammation on the progression and complications of the atherosclerotic process. Our goal was to determine the prognostic value of the glucose-lymphocyte ratio (GLR) in predicting stroke occurrence.

Methods: A total of 333 patients who applied to the cardiology outpatient clinic between 2019 and 2021 were enrolled in the study. Patients were divided into two groups based on whether their carotid artery stenosis was greater than or less than 50%. Additionally, patients who had experienced a stroke were compared to those who had not. GLR was obtained by dividing glucose levels by lymphocyte count.

Results: The mean age of the study population was 65.92 ± 10.17 years. Patients with stenosis of at least 50% were older and had higher levels of glucose, total cholesterol, low-density lipoprotein cholesterol, and GLR. These patients also had lower monocyte counts. Patients who experienced a stroke were older and had higher neutrophil, glucose, creatinine, and GLR values. These patients also had a higher degree of carotid artery stenosis and lower lymphocyte values. A GLR value of 52.75 exhibited 69.9% sensitivity and 55.7% specificity. The results of the multivariable logistic regression analysis showed that the degree of carotid artery stenosis, neutrophil count, GLR, and clopidogrel use were independent predictors of stroke.

Table 1. Characteristics of the study group and comparison of patients who had carotid artery disease <50% and ≥50%

	All patients (n=333)	Group 0 Carotid artey disease <50% (n=79)	Group 1 carotid artery disease ≥ 50% (n=254)	p
Age (years)	65.92±10.17	65.00 (56.00 – 71.00)	67.50 (61.00 – 75.00)	**0.003**
Gender (n, %)				0.838
Female	117 (35.1)	27 (34.2)	90 (35.4)	
Male	216 (64.9)	52 (65.8)	164 (64.6)	
Carotid artery stenosis (%)	80.00 (50.00 – 90.00)	20.00 (00 – 40.00)	90.00 (80.00 – 95.75)	**<0.001**
Stroke during follow-up	183 (55.0)	16 (20.3)	167 (65.7)	**<0.001**
Death during follow-up	151 (45.3)	25 (31.6)	126 (49.6)	**0.005**
Total cholesterol (mg/dL)	189.00 (155.00 – 223.50)	177.00 (135.00 –212.00)	190.00 (159.75 – 226.00)	**0.006**
LDL-C (mg/dL)	118.00 (92.00 – 144.00)	100.00 (85.00 –133.00)	120.50 (94.00 – 149.00)	**0.025**
HDL-C (mg/dL)	38.00 (32.00 – 45.00)	38.00 (31.00 –46.00)	38.00 (32.00 – 45.00)	0.918
Triglyceride (mg/dL)	144.00 (107.00 – 202.00)	142.00 (97.00 –197.00)	144.50 (114.50 – 208.87)	0.216
Hemoglobin (g/dL)	13.30 (11.90 – 14.85)	13.20 (11.70 –15.00)	13.35 (11.96 – 14.78)	0.609
Neutrophil count (10^9/L)	4.86 (4.21 – 5.80)	4.78 (4.34 – 5.78)	4.87 (4.16-5.82)	0.830
Lymphocyte count (10^9/L)	2.54 (1.90 – 2.87)	2.64 (2.09-2.87)	2.49 (1.88-2.87)	0.161
Monocyte count (10^9/L)	0.67 (0.53 – 0.81)	0.73 (0.58-0.88)	0.66 (0.51-0.79)	**0.038**
Platelet count (10^9/L)	250.00 (196.00 – 290.00)	238.00 (195.00 –279.00)	254.00 (196.00 – 295.00)	0.304
Glucose (mg/dL)	125.00 (100.00 – 204.50)	116.00 (92.00 –160.00)	130.00 (103.00 – 208.25)	**0.008**
Creatinine (mg/dL)	0.90 (0.78 – 1.15)	0.85 (0.75-1.07)	0.93 (0.79-1.18)	0.085
GLR	59.03 (40.03-97.00)	47.05 (33.69-85.55)	61.24 (42.00-99.75)	**0.004**
Medical treatment (n, %)				
Acetylsalicylic acid	307 (92.2)	71 (89.9)	236 (92.9)	0.392
Clopidogrel	252 (75.7)	43 (54.4)	209 (82.3)	**<0.001**
Statin	180 (54.1)	38 (48.1)	142 (55.9)	0.224
B-blocker	168 (50.5)	38 (48.1)	130 (51.2)	0.633
ACEI/ARB	171 (51.4)	39 (49.4)	132 (52)	0.686
Risk factors (n, %)				
DM	142 (57.4)	30 (38)	112 (44.1)	0.337
HT	242 (72.7)	55 (69.6)	187 (73.6)	0.486
CAD	294 (88.3)	65 (82.3)	229 (90.2)	0.068
Smoking	112 (33.6)	31 (39.2)	81 (31.9)	0.227
HL	105 (31.5)	22 (27.8)	83 (32.7)	0.658

ACEI: angiotensin converting enzyme inhibitor, ARB: angiotensin receptor blocker, CAD: coronary artery disease, DM: diabetes mellitus, GLR: glucose lymphocyte ratio, HDL-C: high density lipoprotein cholesterol, HL: hyperlipidemia, HT: hypertension, LDL-C: low density lipoprotein cholesterol.

Conclusions: Compared to glucose levels and lymphocyte counts, the GLR was predictive of stroke occurrence in patients with carotid artery stenosis. Our results provide new evidence of the importance of inflammation in the prognosis of atherosclerotic disease.

Table 2. Comparison of the patients with and without stroke

	STROKE (-) (n=150)	STROKE (+) (n=183)	p
Age (years)	66.00 (58.00 – 71.00)	67.00 (62.00 – 75.00)	**0.015**
Gender (n, %)			0.108
Female	59 (50.9)	57 (49.1)	
Male	90 (41.7)	126 (58.3)	
Carotid artery stenosis (%)	60.00 (30.00 – 90.00)	80.00 (80.00 – 95.00)	**<0.001**
Total cholesterol (mg/dL)	191.16±46.87	190.55±49.91	0.941
LDL-C (mg/dL)	112.00 (93.20 –142.00)	120.00 (91.00 – 149.00)	0.795
HDL-C (mg/dL)	39.00 (32.50 –48.00)	37.00 (32.00 – 45.00)	0.109
Triglyceride (mg/dL)	142.00 (100.00 –198.00)	145.00 (115.00 – 208.50)	0.410
Hemoglobin (g/dL)	13.30 (12.00 –14.77)	13.30 (11.90 – 14.90)	0.757
Neutrophil count (10^9/L)	4.61(4.14 – 5.58)	5.05 (4.33-6.03)	**0.012**
Lymphocyte count /10^9/L)	2.61 (2.14-2.92)	2.40 (1.72-2.87)	**0.002**
Monocyte count /10^9/L)	0.68 (0.55-0.80)	0.66 (0.52-0.84)	0.717
Platelet count /10^9/L)	246.00 (196.00 –279.00)	254.00 (195.00 – 295.00)	0.365
Glucose (mg/dL)	116.00 (98.00 –185.00)	137.00 (104.00 – 215.00)	**0.008**
Creatinine (mg/dL)	0.87 (0.75-1.10)	0.95 (0.80-1.20)	**0.038**
GLR	48.08 (35.69-78.67)	65.78 (44.31-108.51)	**<0.001**
Medical treatment (n, %)			
Acetylsalicylic acid	135 (44.1)	171 (55.9)	0.340
Clopidogrel	99 (39.3)	153 (60.7)	**<0.001**
Statin	83 (46.1)	97 (53.9)	0.623
B-blocker	71 (42.5)	96 (57.5)	0.383
ACEI/ARB	72 (42.1)	99 (57.9)	0.295
Risk factors (n, %)			
DM	67 (47.5)	74 (52.5)	0.406
HT	104 (43.2)	137 (56.8)	0.303
CAD	130 (44.4)	163 (55.6)	0.608
Smoking	51 (45.5)	61 (54.5)	0.864
HL	44 (41.9)	61 (58.1)	0.459

ACEI: angiotensin converting enzyme inhibitor, ARB: angiotensin receptor blocker, CAD: coronary artery disease, DM: diabetes mellitus, GLR: glucose lymphocyte ratio, HDL-C: high density lipoprotein cholesterol, HL: hyperlipidemia, HT: hypertension, LDL-C: low density lipoprotein cholesterol.

Table 3. ROC curve analysis results of glucose, lymphocyte and GLR for prediction of stroke

	AUC	P	95% CI
Glucose	0.585	0.008	0.523-0.647
Lymphocyte	0.400	0.002	0.339-0.460
GLR	0.637	<0.001	0.577-0.696

GLR: glucose lymphocyte ratio.

Table 4. Multivariable logistic regression for prediction of stroke

	p	OR	95% CI
Age (years)	0.073	1.024	0.998-1.051
Carotid artery stenosis	<0.001	1.029	1.019-1.039
Neutrophil count	0.030	1.198	1.018-1.410
GLR	0.001	1.010	1.005-1.015
Clopidogrel use	0.038	1.895	1.036-3.466

GLR: glucose lymphocyte ratio.

Table 5. Univariable logistic regression for prediction of stroke

	p	OR	95% CI
Age (years)	0.024	1.026	1.003-1.048
Carotid artery stenosis	<0.001	1.031	1.022-1.041
Neutrophil count	0.024	1.172	1.021-1.345
Lymphocyte count	0.064	0.802	0.635-1.013
Glucose	0.069	1.002	1.000-1.005
Creatinine	0.955	0.991	0.712-1.379
GLR	<0.001	1.010	1.005-1.015
Clopidogrel use	<0.001	2.576	1.534-4.326

GLR: glucose lymphocyte ratio.

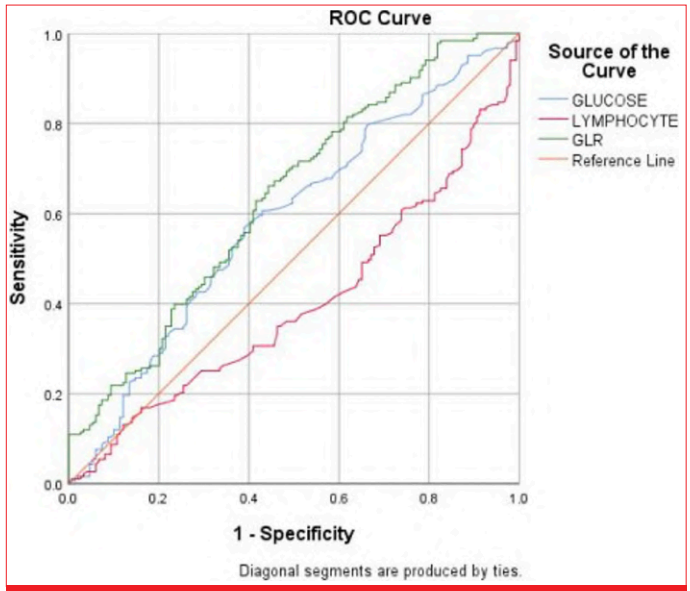

Figure 1. ROC curve analysis for prediction of stroke.

OP-079 [Heart Failure]

Clinical relevance of HALP score in predicting hospital stay duration and outcomes in acute heart failure

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Background and Aim: Heart failure (HF) remains a global health burden characterized by frequent hospitalizations and high mortality. The Hemoglobin, Albumin, Lymphocyte, and Platelet (HALP) score has emerged as a composite marker reflecting nutritional and inflammatory status, but its prognostic utility in acute HF settings remains underexplored.

Methods: This retrospective study included 222 patients hospitalized with acute heart failure at a tertiary care center in Somalia between January 2022 and December 2023. Patients were stratified by hospital stay duration: short (≤7 days) vs. long (>7 days). The HALP score was calculated using routine laboratory values, and a previously validated cut-off was used for stratification. Logistic regression analysis was used to identify predictors of long hospital stay. Model performance was assessed using Hosmer–Lemeshow test, Nagelkerke R^2, classification statistics, and ROC curve analysis.

Results: A total of 222 patients were analyzed; 86 (38.7%) had prolonged hospitalization. Patients with HALP scores above the prognostic threshold were significantly more likely to experience long hospital stay (p=0.002). In multivariable analysis, a HALP score ≥ cut-off (OR: 10.19, 95% CI: 2.49–41.63, p=0.002) and prior stroke (OR: 8.44, 95% CI: 1.15–61.88, p=0.035) independently predicted prolonged hospital stay. Model fit was

adequate (Hosmer–Lemeshow p=0.105), and explanatory power was moderate (Nagelkerke R^2=0.31). However, the HALP score's standalone discriminative ability was poor (AUC=0.511).

Conclusions: The HALP score is an independent predictor of prolonged hospitalization in acute heart failure patients. While its individual discriminative power is limited, its role within multivariable risk stratification models appears promising. Further prospective validation is warranted.

Table 1. Comparison of demographic, clinical, and laboratory parameters according to hospital stay duration in acute heart failure patients

Variable	Total (n = 222)	Short hospital stay (n = 136)	Long hospital stay (n = 86)	p-value
Demographics				
Age ≥65 years, n (%)	42 (19.1%)	13 (9.6%)	29 (34.5%)	**<0.001**
Male sex, n (%)	138 (62.7%)	90 (66.2%)	48 (57.1%)	0.229
Comorbidities				
Chronic Kidney Disease, n (%)	29 (13.2%)	14 (10.3%)	15 (17.9%)	0.160
Hypertension, n (%)	118 (53.6%)	75 (55.1%)	43 (51.2%)	0.665
Diabetes, n (%)	123 (55.9%)	74 (54.4%)	49 (58.3%)	0.668
Laboratory values				
Albumin low, n (%)	148 (67.3%)	98 (72.1%)	50 (59.5%)	0.076
CRP elevated, n (%)	198 (90.0%)	120 (88.2%)	78 (92.9%)	0.379
Procalcitonin elevated, n (%)	31 (14.1%)	17 (12.5%)	14 (16.7%)	0.507
ProBNP elevated, n (%)	216 (98.2%)	132 (97.1%)	84 (100.0%)	0.286
Outcomes				
HALP Score ≥ cut-off, n (%)	23 (10.5%)	7 (5.1%)	16 (19.0%)	**0.002**
In-hospital mortality, n (%)	21 (9.5%)	4 (2.1%)	17 (65.4%)	**<0.001**

CRP = C-reactive protein; ProBNP = Pro-B-type natriuretic peptide; HALLP = Hemoglobin, Albumin, Lymphocyte, LDH, and Platelet score.

Table 2. Multivariable logistic regression analysis of predictors of long hospital stay in acute heart failure patients

Variable	Odds Ratio (95% CI)	p-value
HALLP score ≥ cut-off	10.19 (2.49 – 41.63)	0.002
Stroke	8.44 (1.15 – 61.88)	0.035
Platelet count low	0.18 (0.04 – 0.79)	0.028
Age ≥65 years	2.30 (0.84 – 6.27)	0.110
Male sex	1.98 (0.94 – 4.15)	0.077

CI – Confidence Interval;
OR – Odds Ratio;
HALP – Hemoglobin, Albumin, Lymphocyte, and Platelet;
HF – Heart Failure.

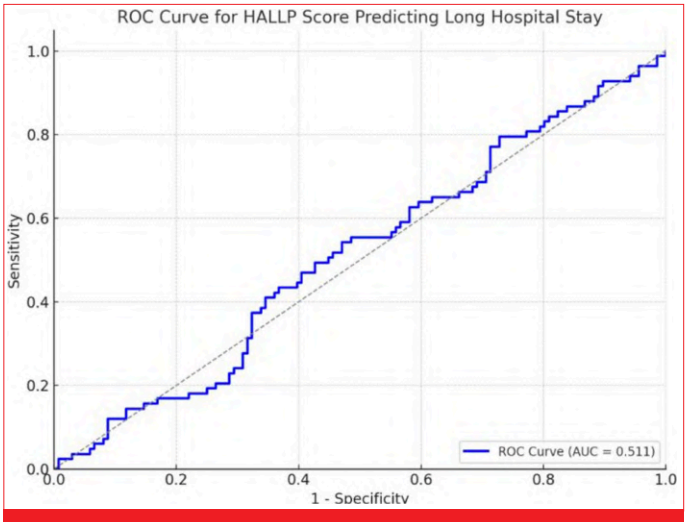

Figure 1. ROC curve evaluating the diagnostic performance of the HALLP score in predicting long hospital stay among acute heart failure patients.

OP-080 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Atrial fibrillation pulsed field ablation (PFA), one-month experience at a single centre

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Background and Aim: Catheter ablation is an effective treatment for symptomatic atrial fibrillation (AF) patients. Major complications such as atrioesophageal fistula, phrenic nerve palsy, and pulmonary vein stenosis have been observed in traditional thermal (radiofrequency and cryoballoon) ablation. Pulsed field ablation (PFA) creates non-thermal lesions in cardiac tissue within milliseconds through an irreversible electroporation mechanism. The aim of this study is to share our experiences with AF PFA procedures performed in our clinic over the past month.

Methods: This retrospective, single-centre study enrolled patients diagnosed with AF who underwent PFA within the last month. All interventions were performed under general anaesthesia and with uninterrupted oral anticoagulation. Transoesophageal echocardiography was performed on patients with AF on the day of the procedure, in order to rule out the presence of a thrombus. The procedures were performed under fluoroscopic guidance. Following femoral vein puncture, 5,000 units of intravenous heparin were administered. Transseptal puncture was performed and the remaining heparin dose was administered according to the patient's weight. Measurements were taken every 15 minutes throughout the procedure to ensure an active clotting time of at least 350 seconds. Additional heparin applications were performed as required. A 13F Faradrive sheath (Boston Scientific) was advanced into the left atrium over a guidewire. Under fluoroscopic guidance, a PFA catheter (Farawave, Boston Scientific) was placed into each pulmonary vein ostium through the sheath. Repeated PFA applications were performed on each pulmonary vein, with at least four basket and four flower-shaped applications. After each application, the catheter was rotated to achieve complete circumferential isolation. Following transthoracic echocardiography, all patients were discharged the day after the procedure.

Results: We included ten patients (age 63.3 ± 12.17 years, 20% female) with AF (7 paroxysmal and 3 persistent). In one patient, the left veins were connected to the atrium through a common ostium. Baseline characteristics and laboratory findings of the patients are summarized in Table 1. An average of 42.60 ± 3.35 PFA procedures were performed on patients. Isolation of the pulmonary veins was achieved in all patients. Two patients underwent additional cavotricuspid isthmus (CTI) ablation using a radiofrequency ablation catheter due to episodes of atrial flutter. One of the patient underwent CTI catheter ablation using the CARTO 3 mapping system. This catheter enabled the creation of a scar map of the patient's left atrium and pulmonary veins, demonstrating that the pulmonary veins were isolated. No patient experienced early AF recurrence, and no procedure-related complications were observed. The AF PFA procedure is illustrated in Figures 1 and 2.

Conclusions: PFA is an increasingly popular and effective treatment method for patients with symptomatic AF.

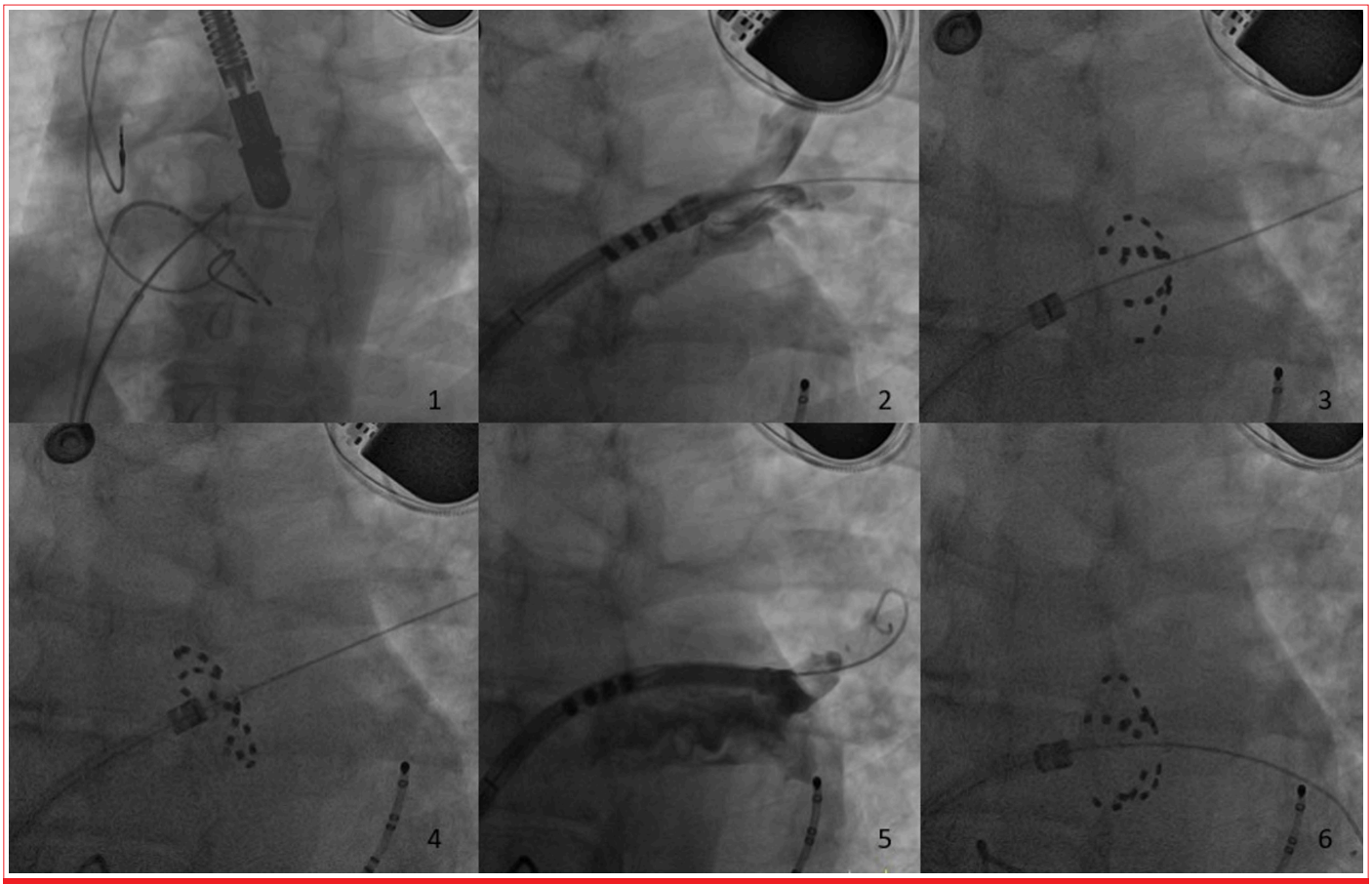

Figure 1. The AF PFA procedure is illustrated in Figures 1 and 2.

Table 1. Baseline characteristics and laboratory findings of patients

	(n=10)
Baseline characteristics	
Age (years), mean (SD)	63.3±12.17
Gender (female), n (%)	2 (%20)
Diabetes Mellitus, n (%)	3 (%30)
Hypertension, n (%)	7 (%70)
Body Mass Index (kg/m2), mean (SD)	27.89±4.69
Atrial Fibrillation Type (paroxysmal), n (%)	7 (%70)
Left Ventricular Ejection Fraction (%), mean (SD)	61.50±2.29
Left Atrial Diameter (mm), mean (SD)	45.80±5.13
CHADVA Score, mean (SD)	1.80±1.78
HASBLED Score, mean (SD)	1.0±0.77
Laboratory Findings	
Creatinine (mg/dl; SD)	0.96±0.22
WBC (x10^3 /µL; SD)	8.43±1.96
Neutrophil (x10^3 /µL; SD)	5.36±2.44
Lymphocyte (x10^3 /µL; SD)	2.19±0.95
Haemoglobin (g/dL; SD)	13.66±1.14
Platelets (x10^3 /µL; SD)	182.50±32.49
ALT (U/L; SD)	18.60±4.90
AST (U/L; SD)	22.90±7.73
TSH (uIU/mL; SD)	2.87±1.0
LDL (mg/dl; SD)	128.50±32.89
Medications	
NOAC, n (%)	10 (100%)
RAAS Blockers, n (%)	5 (50%)
β Blocker, n (%)	9 (90%)
Statins, n (%)	6 (60%)

Table 2. Clinical parameters of the patients who underwent AF PFA

No	Gender	Age	Atrial Fibrillation Type	Number of Applications	Left Atrial Diameter (mm)	Body Mass Index	CHADVA Score	Complication
1	F	75	Paroxysmal	44	51	18,82	5	None
2	M	51	Persistent	40	48	37,55	2	None
3	M	69	Paroxysmal	36	43	28,65	0	None
4	M	47	Persistent	40	44	30,75	2	None
5	M	58	Paroxysmal	44	42	28,70	1	None
6	M	39	Paroxysmal	40	38	29,07	0	None
7	M	52	Paroxysmal	48	46	27,74	1	None
8	M	64	Paroxysmal	46	55	25,95	0	None
9	M	72	Paroxysmal	44	40	29,07	2	None
10	F	76	Persistent	44	51	22,60	5	None

OP-55A [Interventional Cardiology / Coronary]

Relationship between prognostic nutritional index score and in-hospital mortality in patients with NSTEMI

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Background and Aim: Non-ST segment elevation myocardial infarction (NSTEMI) is among the main causes of death in

Table 1. The baseline demographic, laboratory and angiographic characteristics of the study population

Variables	Survived group, n=1302	Non-Survived group, n=90	Total Patients, n=1392	p
Age, years (SD)	55 ± 12	64 ± 13	56 ± 12	<0.001
Female, n (%)	293 (23%)	25 (28%)	318 (23%)	0.249
Diabetes mellitus, n (%)	289 (22.2%)	32 (35.6%)	321 (23.1%)	0.004
Hypertension, n (%)	515 (39.6%)	47 (52.2%)	562 (40.4%)	0.018
Smokers, n (%)	477 (36.6%)	32 (35.6%)	509 (36.6%)	0.837
LVEF, % (SD)	48 ± 8	42 ± 9	47 ± 8	<0.001
Systolic BP (SD)	125 ± 19	119 ± 46	124 ± 22	0.683
Diastolic BP (SD)	75 ± 13	68 ± 27	74 ± 14	0.318
IRA, n (%) LAD	690 (53%)	56 (62.2%)	746 (53.6%)	0.375
IRA, n (%) CX	290 (22.3%)	10 (11.1%)	300 (21.6%)	0.375
IRA, n (%) RCA	234 (18%)	14 (15.6%)	248 (17.8%)	0.375
IRA, n (%) Others	88 (6.8%)	10 (11.1%)	98 (7%)	0.375
Beta-blocker, n (%)	917 (70.4%)	50 (55.6%)	967 (69.5%)	0.003
ACEI, n (%)	878 (67.4%)	64 (71.1%)	942 (67.7%)	0.471
ASA, n (%)	851 (65.4%)	51 (56.7%)	902 (64.8%)	0.095
Statin, n (%)	829 (63.7%)	50 (55.6%)	879 (63.3%)	0.123
WBC, x10^3/μL (SD)	9.6 ± 4	10 ± 4	9.6 ± 4	0.228
Hemoglobin, g/dL (SD)	13.8 ± 1.68	13.5 ± 1.78	13.84 ± 1.69	0.131
Glucose (mg/dL) (IQR)	125 (103-156)	130 (95-173)	125 (102-157)	0.640
Platelet count, x10^3/μL (SD)	258 ± 67	262 ± 65	258 ± 67	0.462
eGFR, mL/min/1.7^3 m^2 (SD)	80 ± 15	74 ± 22	79 ± 15	0.024
Baseline Troponin I, (ng/mL) (IQR)	2070 (910-5500)	1685 (900-7000)	2000 (900-5580)	0.336
Total cholesterol, mg/dL (IQR)	175 ± 48	174 ± 53	175 ± 48	0.463
Triglycerides, mg/dL (IQR)	114 (81-166)	113 (81-164)	114 (81-165)	0.844
LDL-C, mg/dL (SD)	130 ± 46	133 ± 54	130 ± 47	0.804
HDL-C, mg/dL (SD)	38 ± 12	38 ± 15	38 ± 12	0.482
PNI Score (SD)	47.3 ± 7.4	39.1 ± 7.1	46.8 ± 7.6	<0.001
Albumin, mg/L (SD)	38 ± 5	35 ± 6	38 ± 5	<0.001
Lymphocyte (10^3/μL) (IQR)	1.90 (1.10-2.56)	1.34 (0.60-2.0)	1.85 (1.06-2.54)	<0.001

LVEF: Left ventricular ejection fraction; BP: Blood pressure; IRA: Infarct-related artery; LAD: Left anterior descending; CX: Circumflex artery; RCA: Right coronary artery; ACEI: Angiotensin-converting-enzyme inhibitor; ASA: Acetyl salicylic acid; WBC: White blood cell; eGFR: Estimated glomerular filtration rate; LDL-C: Low-density lipoprotein cholesterol; HDL-C: High-density lipoprotein-cholesterol; PNI: Prognostic nutritional index.

patients with acute coronary syndrome. When individuals have both cardiovascular disease and malnutrition, their risk of death increases. The prognostic nutritional index (PNI) is a straightforward measure that reflects nutritional and immune status. This study aimed to evaluate the effectiveness of the PNI score in predicting in-hospital mortality among patients with NSTEMI who underwent percutaneous coronary intervention (PCI).

Methods: NSTEMI patients who underwent primary PCI and were hospitalized in the cardiac intensive care unit. The following patients were included in our study: In accordance with the 2018 global diagnostic criteria for NSTEMI and Age ≥18 years. Exclusion criteria included: patients whose serum albumin and/or lymphocyte counts were not measured; tumor patients; severe liver and kidney dysfunction; patients during the acute infection period. Ultimately, the study population consisted of 1392 patients, divided into two groups based on mortality: The survived group (n=1302) and the non-survived group (n=90).

Results: The current study involved 1074 males (77%) and 318 females (23%), with a mean age of 56 ± 12 years. The non-survived group was older and had a higher prevalence of hypertension and diabetes mellitus. However, the non-survived group had significantly lower levels of albumin, lymphocytes, and eGFR. These patients also had a lower left ventricular ejection fraction (LVEF) compared to the survived group. Additionally, these patients had a lower PNI score (Table 1). A multivariate logistic regression analysis revealed that age, LVEF, and PNI score were independently associated with in-hospital mortality in patients with NSTEMI (Table 2). The ROC curve analysis identified optimal cut-off values for the PNI score, which were independent predictors of in-hospital mortality in the multivariate analysis (AUC: 0.787, Cut-off: <40.95, Sensivity: 62.2%, Specificity: 80.8%, p<0.001) (Figure 1).

Conclusions: Our study demonstrates that the PNI score independently predicts in-hospital mortality in NSTEMI patients undergoing PCI. Mortality is higher in the early days of ACS.

Table 2. Univariate and multivariate analyses of variables predicting in-hospital mortality in patients with NSTEMI treated with percutaneous coronary intervention

	Univariate			Multivariate		
Variables	**OR**	**95% CI**	**p value**	**OR**	**95% CI**	**p value**
PNI Score	0.834	0.803-0.866	<0.001	0.851	0.819-0.885	<0.001
Age	1.056	1.038-1.074	<0.001	1.035	1.016-1.054	<0.001
Diabetes mellitus	1.934	1.232-3.036	0.004			
Hypertension	1.670	1.088-2.563	0.019			
LVEF	0.924	0.900-0.948	<0.001	0.938	0.911-0.965	<0.001
Beta-blocker	0.525	0.341-0.809	0.003			
eGFR	0.979	0.967-0.992	0.001			

LVEF: Left ventricular ejection fraction; eGFR: Estimated glomerular filtration rate.

Therefore, it's critical to forecast mortality and implement the necessary precautions for patients at high risk. The calculation of the PNI requires only the serum albumin level and the lymphocyte count of the patient. Malnutrition is a common and significant problem, especially among hospitalized elderly patients. Previous studies suggest that malnutrition is closely linked to poor prognosis and higher mortality in cases of terminal kidney failure, malignancy, and hematological diseases. Additionally, low PNI values have been shown to predict long-term mortality in NSTEMI patients. However, our study is the first to predict in-hospital mortality specifically in the NSTEMI group. The PNI score, a simple and easy-to-calculate index, may help predict in-hospital mortality in NSTEMI patients.

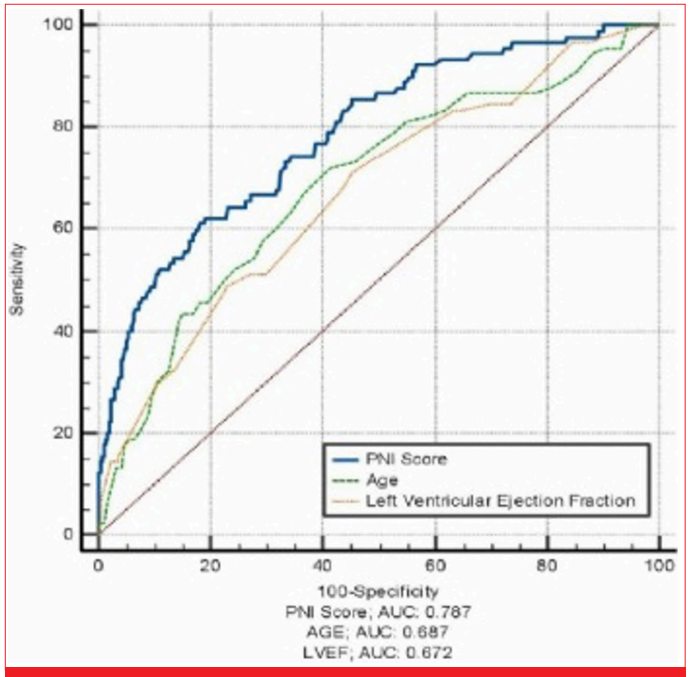

Figure 1. ROC curve analysis of PNI score, age, and LVEF in predicting in-hospital mortality among NSTEMI patients treated with percutaneous coronary intervention.