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THE ANATOLIAN JOURNAL OF CARDIOLOGY

THE EXECUTIVE BOARD OF TURKISH SOCIETY OF CARDIOLOGY

Dear Colleagues,

In addition to organizing various training programs and events throughout the year, the Turkish Society of Cardiology plans to hold the National Cardiology Congress in November, as always, at a level worthy of its 58th year.

The Society is planning to present the rich content of our congress, which is one of the leading scientific congresses at national and international level with both the number of participants and high quality scientific content, with a wide range of satisfying scientific programs and various social activities that will appeal to all our participants.

Our goal of making our congress the leading meeting of the region in the field of Cardiology continues.

Our colleagues from other continents, as well as our colleagues from the European Society of Cardiology, member countries, will attend our congress this year. We expect the participation in our congress, which we held online last year, to increase even more this year.

In our congress, we tried to prepare the best program for you. We will update and discuss our latest knowledge on cardiovascular diseases through "Symposiums", "Contrasting View" and "How To" sessions. We expanded our "Cardiology in Daily Practice" sessions under the title of "Young Cardiologists Sessions" to cover the entire cardiology practice.

We will improve our skills as well as our knowledge with the certified "Interactive Courses", the number of which we have increased due to the intense interest in the past years. In each of our sessions, there are valuable speakers and panelists from Turkey and the world who are prominent scientists of their areas.

We believe that our joint sessions with ESC, ACC, Turkish World Society of Cardiology, EACVI, EHRA and EAPCI will be watched carefully.

Our congress, whose international dimension has become stronger this year, will again be credited by the TSD.

We will be pleased to see you among us at our congress.

Looking forward to meeting with you all within the 37th Turkish Cardiology Congress with International Participation on 18-21 November 2021, to share our knowledge,

With our best wishes and best regards,

Prof. Dr. Vedat Aytekin
President of TSC

Prof. Dr. Muzaffer Değertekin
President Elect
Chair Scientific Committee

THE ANATOLIAN JOURNAL OF CARDIOLOGY

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Cardiac imaging / Echocardiography

OP-001

Left ventricular blood flow energetics after acute ST-segment elevation myocardial infarction associate with left ventricular remodeling

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Background and Aim: ST-segment elevation myocardial infarction (STEMI) leads to complex changes in left ventricular (LV) haemodynamics. It remains unknown how four-dimensional (4D) acute changes in LV blood flow kinetic energy (KE) affect LV remodeling. This study aimed to investigate how LV blood flow energetics early after MI affects adverse LV remodeling.

Methods: In total, 69 revascularised STEMI patients were enrolled. All patients underwent cardiovascular magnetic resonance (CMR) examination within 2 days of the index event and at 3-month. CMR examination included cine, late gadolinium enhancement, and whole-heart 4D flow acquisitions. LV volume-function, infarct size (indexed to body surface area), microvascular obstruction, mitral inflow, and 4D blood flow KEi (indexed to end-diastolic volume) characteristics were obtained. Adverse LV-remodeling was categorized according to increase in LV end-diastolic volume: 10% (mild), 15% (moderate), and 20% (severe).

Results: Twenty-four patients (35%) developed mild, 17 patients (25%) moderate, 11 patients (16%) severe LV remodeling. Demographics and clinical history were comparable between patients with/without LV remodeling. A-wave KE was significantly lower in MI patients with adverse LV remodeling than MI patients without adverse LV remodeling (p=0.02) (Fig. 1). A-wave KEi was associated with mild, moderate, severe LV remodeling (p=0.03, p=0.02, p=0.02, respectively), whereas infarct size only with mild LV remodeling (p=0.02). In multivariable analysis, A-wave KEi was identified as independent marker for mild, moderate, severe LV remodeling (p=0.09, p<0.01, p<0.01, respectively), whilst infarct size only for mild LV remodeling (p=0.03). In ROC analysis for A-wave KEi to predict LV remodeling, the area under the curve was 0.67 for mild (p=0.02), 0.70 for moderate (p=0.01), 0.71 for severe (p=0.03) LV remodeling.

Conclusions: In patients with STEMI, LV hemodynamics assessment by LV blood flow KE demonstrates a significant association with adverse LV remodeling. A-wave KE early after acute MI had an independent effect on adverse LV remodeling.

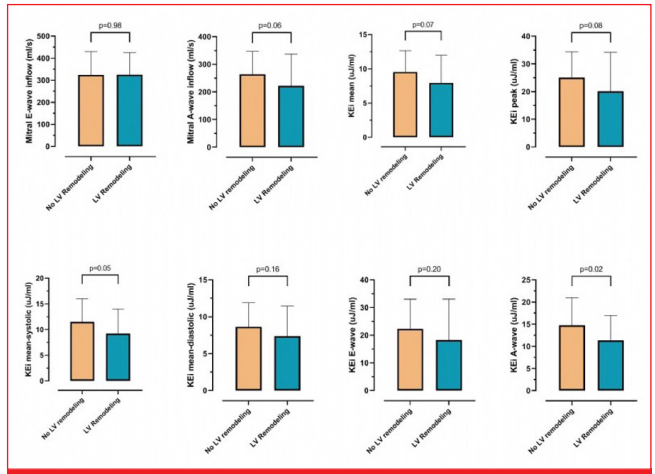

Figure 1. Comparison of flow hemodynamics between patients with/without LV remodeling

Cardiac imaging / Echocardiography

OP-002

Evaluation of clinical, echocardiographic, and laboratory parameters in patients with intracardiac device and prothesis valve endocarditis

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Background and Aim: Prosthetic Valve Endocarditis (PVE) and Cardiac Device-Related Infective Endocarditis (CDRIE) are the most difficult to diagnose group among infective endocarditis (IE). Vegetation-like structures detected in transesophageal echocardiography (TEE) can sometimes cause false positive. Modified Duke criteria are in the routinely diagnosis of IE lower diagnostic accuracy in PVE and CDRIE patients. In this study, it was aimed to

evaluate and compare the diagnostic performance of clinical, laboratory, echocardiographic parameters, and nuclear medicine examinations in patients with a diagnosis of CDRIE and PVE, to examine the use of Modified Duke Criteria used in the diagnosis of IE in this selected patient group and to investigate how many of the patients examined with suspicion of IE were actually IE and what parameters contributed to predicting IE.

Methods: In the study, the records of the patients who had been diagnosed as IE and who had undergone TEE, were reviewed retrospectively. 94 patients with a preliminary diagnosis of PVE and CDRIE were included in the study. Demographic features, additional diseases, clinical features, echocardiographic parameters, laboratory parameters, blood culture results, if any, nucleer imaging results, treatment method, and clinical course applied to the patients were investigated from the records. The data of these patients who were hospitalized with a preliminary diagnosis of IE were evaluated according to the Modified Duke Criteria. The existence of two major or one major and three minor criteria or five minor criteria were defined as "Definitive IE". In the presence of a major and a minor or three minor criteria, it was evaluated as "Possible IE". Patients who did not meet these criteria or had an alternative diagnosis were accepted as "not IE".

Results: 94 patients (34 female, 60 male; mean age 62.06±13.12 years) were included in the study. According to the Modified Duke criteria, 32.98% of the patients, who underwent TEE with a preliminary diagnosis of PVE and CDRIE and who had an image compatible with vegetation, were found to be compatible with the diagnosis of definitive IE, 38.3% of them were coherent with the diagnosis of possible IE, and 28.72% did not comply with the criteria of IE. While fever is observed in the majority of patients (80%) with PVE, however, most patients (57.9%) with CDRIE have not been shown to have fever. Considering the TEE findings, which is one of the most important criteria used in the diagnosis of IE, patients with vegetation below 5 mm in size or seen as a linear mobile structure are unlikely to be definitive IE as a final diagnosis. On the contrary, half of these patients did not have IE. Vegetation of 6mm and above in TEE has been shown to be significant in definitive IE diagnosis (p=0.013).

Conclusions: In the definitive diagnosis of PVE and CDRIE, 5 mm and smaller vegetation on TEE, especially when the vegetations are linear, being more careful for the diagnosis may prevent overdiagnosis.

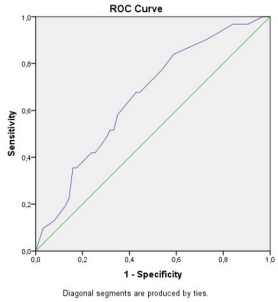

Figure 1. ROC curve showing the correlation of vegetation size above 6 mm with definitive IE diagnosis

Table 1. Comparison of vegetation size on TEE in patients with definitive IE, possible IE, and not IE

Vegetation size in TEE (mm)	Definitive IE (n=31)	Possible IE (n=36)	Not IE (n=27)
Linear mobile structure	3 (9.68%)	2 (5.56%)	5 (18.52%)
0-5 mm	3 (9.68%)	9 (25.00%)	9 (33.33%)
6-10 mm	10 (32.26%)	16 (44.44%)	7 (25.93%)
11-15 mm	5 (16.13%)	5 (13.89%)	1 (3.7%)
16-20 mm	6 (19.35%)	3 (8.83%)	4 (14.81%)
>21 mm	1 (3.23%)	0 (0.00%)	1 (3.70%)
Apse	3 (9.68%)	1 (2.78%)	0 (0.00%)

Table 2. Demographic data of the patients

	Patient (n=94)
Gender (Female/Male)	34/60
Age (years)	62.06±13.12
Height (m)	1.66±0.88
Weight (kg)	78.15±12.83
BSA (m^2)	1.87±0.87
DM	28 (29%)
ASCVD	42 (44%)
PVE	25 (26.6%)
CDRIE	69 (73.4%)

Table 3. Patients diagnosed with definitive and possible IE according to Modified Duke criteria

	Modified Duke Criteria	Patient (n=94)
Definitive IE	2 Major	31 (32.98%)
	1 Major+3 Minor	0
	5 Minor	0
Possible IE	1 Major + 1 Minor	36 (38.3%)
	3 Minor	0
Not IE		27 (28.72%)

Cardiac imaging / Echocardiography

OP-003

The role of coronary CT angiography and person's lifestyle habits in risk assessment for primary protection from cardiovascular diseases

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Background and Aim: Cardiovascular disease (CVD) risk factors are; age, gender, family history, hyperlipidemia (HL), hypertension (HT), smoking, diabetes mellitus (DM), alcohol, obesity, physical inactivity, unhealthy diet. Recent studies have shown that the individual's socioeconomic level, educational status, social support, cognitive functions and Agat-

ston score [which measures coronary artery calcium (CAC) in cardiac computed tomography (CT)] also pose a risk for CVD. The Systematic Coronary Risk Evaluation (SCORE) risk model includes classical atherosclerotic risk factors. There is a need to add new parameters that will increase the power of risk assessment in addition to SCORE system. The aim of this study is to investigate the importance of coronary CTA findings and lifestyle characteristics of patients in primary cardiovascular prevention in patients admitted to cardiology outpatient.

Methods: 565 patients who applied to the İzmir Medical Park Hospital cardiology outpatient clinic between 15.01.2019-20.01.2020 and underwent CCTA for any reason were screened. Patients with known CAD were excluded, 497 people were included in the study and the questionnaire was applied. Age, body mass index (BMI), gender, cardiovascular risk factors, socioeconomic, lifestyle characteristics of the person were learned. Predicted cardiovascular mortality for 10 years was calculated in accordance with the SCORE risk scale. Agatston CCS, hepatosteatosis, paracardial adipose tissue thickness (PATT) were measured From the CCTA. Parameters that were significant in univariate analysis were included in multivariate analysis, and independent predictors of atherosclerosis and agate scores were investigated.

Results: The average age was 54±13 and 63% were male. Patients who were divided into patients with an CCS=0 and CCS>0 were compared. Age, male gender, BMI, SCORE, HT, DM, working life, PATT were statistically significant. As a result of multivariate analysis, SCORE significantly predicted PATT and working life Agatston CCS=0. The threshold value for PATT was found to be 5 mm by Roc analysis (p=0.000 Area under the curve: 0.597, 95% CI: 0.547-0.648). According to the results of this analysis; Agatston CCS=0; SCORE <5 was 9.1 times (p=0.000 OR: 9.128, 95% CI: 3.341-29.943), PATT <5 mm was 2.6 times (p=0.018 OR: 2.677, 95% CI: 1.185-6.048) predicted. Also It was observed that 7 of 100 patients with low-medium SCORE risk have Agatston score above 100. In 56 of 85 patients with medium SCORE risk have CCS=0.

Conclusions: As a result of this study, PATT may be useful as a parameter predicting atherosclerosis of the person besides the SCORE system. We can add PATT measurement practically to our routine and make the primary prevention of people more effective. Also, we saw that Agatston CACS changed the cardiovascular risk category of people; This practice will prevent the use of unnecessary drugs when it reduces the risk, and will direct people to receive effective medical treatment in cases when the risk increases.

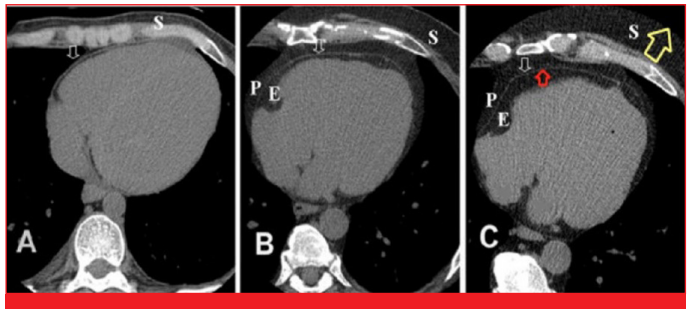

Figure 1. Adipose tissue measurement

Independent Predictors of Being Agatston CCS=0, Multivariate analysis

			%95 CI	
	Anlamlılık (p)	Exp (B)	Min	Max
PATT	**0,018**	0,854	0,750	0,974
BMI	0,589	0,968	0,860	1,089
Creatinin (mg/dL)	0,379	0,186	0,004	7,897
DM	0,121	0,331	0,082	1,339
SVD	0,635	2,059	0,104	40,762
Working life	**0,022**	0,269	0,087	0,828
SCORE	**0,000**	0,656	0,533	0,807

Figure 2. Independent predictors of being Agatston CCS=0, multivariate analysis

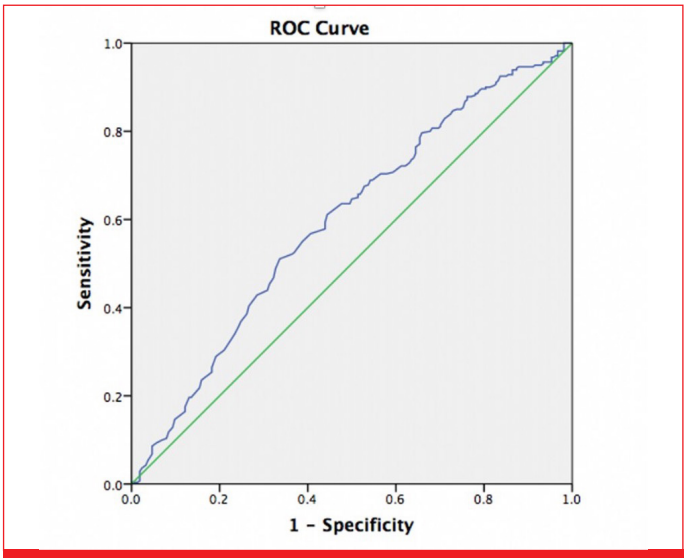

Figure 3. ROC

Independent Predictors of Being Agatston CCS=0, Multivariate analysis

			%95 CI	
	Anlamlılık (p)	Exp (B)	Min	Max
PATT<5	**0,018**	**2,677**	1,185	6,048
SCORE<5	**0,000**	**9,128**	3,341	29,943
Working life	0,076	0,459	0,194	1,085

Figure 4. Independent predictors of being Agatston CCS=0, multivariate analysis-2

Comparison of Intermediate SCORE Risk Based on Agatston KKS 0 Threshold

Agastson CCS	Medium SCORE risk n=85
CCS=0 - n (%)	**56 (%65,9)**
CCS>0- n (%)	29 (%34,1)

Figure 5. Comparison of intermediate SCORE risk based on Agatston KKS 0 threshold

Comparison of Low-Moderate/High-Very High SCORE Risk Based on Agatston KKS 100 Threshold Value

	SCORE	
Agastson CCS	<5 n=100	≥5 n=37
<100- n (%)	93 (%93)	25 (%67,6)
≥100- n (%)	**7 (%7)**	12 (%32,4)

Figure 6. Comparison of low-moderate/high-very high SCORE risk based on Agatston KKS 100 threshold value

Cardiac imaging / Echocardiography

OP-004

The relationship between arterial stiffness and disease severity in patients with COVID-19

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Background and Aim: It is known that COVID-19 infection causes cardiovascular complications. It is thought that this may be related to endothelial dysfunction. The aim of this study is to reveal the relationship between arterial stiffness, which may be an indicator of endothelial dysfunction, and disease severity.

Methods: Patients with COVID-19 infection and age, sex and cardiac risk factors matched control group were included in the study. Arterial stiffness was measured in the early period (within the first month) of COVID-19 infection. Echocardiographic measurements were also performed at the same time. The patients were divided into two groups according to the severity of covid, as mild and severe. In order to determine the severity of the disease, oxygen or intensive care need, biochemical parameters such as C-reactive protein (CRP), hs-troponin, complete blood count (CBC) were used in line with the recommendations. Arterial stiffness measurements were made by the gold standard applanation tonometry method, from the radial, carotid and femoral arteries.

Results: A total of 84 patients were included in the study, 27 of which were in the control, 32 in the mild disease and 25 in the severe disease groups. The mean age of the population was 48.6±12.9 years, and 69% (n=58) were male. The whole group had 30% hypertension (HT), 10% Diabetes Mellitus (DM), 11% coronary artery disease (CAD), 11% were smoking and 2% with hyperlipidemia (HL). There was no significant difference between the groups in terms of these risk factors. The mean pulse wave velocity (PWV) value obtained in arterial stiffness measurements was 8.02, 8.07 and 8.75 in the control group, mild disease group and severe disease group, respectively. These values were found to be statistically significant when the control group and mild disease groups were compared with the severe disease group. (p=0.007, p=0.008, respectively).

Conclusions: The results of this study revealed that the deterioration in arterial stiffnes, which is a good indicator of endothelial dysfunction, is more significant in patients with severe COVID-19 infection. These patients should be followed more closely for subsequent cardiovascular complications.

Table 1. Basal characteristic features and findings

	Control Group	Mild Disease Group	Severe Disease Group	*P*-value
Age (years)	52.4	42.9	52	0.005
Gender (Male, %)	66.7	59.4	84	0.13
BMI	28.2	26.3	27.1	0.268
HT (%)	40.7	21.9	48	0.1
DM (%)	11.1	15.3	24	0.08
CAD (%)	22.2	3.1	16	0.09
Smoking (%)	25.9	9.4	4	0.07
CRP (mg/dL)	2.43	13.7	139.3	0.000
Troponin(ng/mL)	3	4.03	14.7	0.000
PWV	8.02	8.07	8.75	0.009

BMI - body mass index, CAD - coronary artery disease, CRP - C-reactrive protein, DM - diabetes mellitus, HT - hypertension, PWV - pulse wave velocity

Cardiac imaging / Echocardiography

OP-005

Study of transesophageal echocardiography in young patients with cryptogenic stroke: Prevalence of patent foramen ovale and interpretation of the RoPe score

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Background and Aim: The incidence of ischemic stroke in young patients is increasing and associated with unfavorable prognosis. In many young patients the cause of stroke remains unknown, referred to as cryptogenic stroke (CS). Patent foramen ovale (PFO) is seen in approximately 25% of adults and generally is considered a normal anatomic variant. A consistent association between PFO and CS has been observed. The Risk of Paradoxical Embolism (RoPE) score (Table 1), a tool that stratifies patients with CS according to the probability of finding a PFO. Aim of our retrospective study is to investigate the role of transesophageal echocardiography (TEE) to detect the etiology of CS in young patients, determine the prevalence of PFO and determine the role of RoPe score in PFO-related strokes.

Methods: We reviewed the medical records of patients with acute arterial ischemic stroke we applied TEE between 2016 and 2020. We included patients aged 18-55 years without any valvular heart disease, prosthetic valve, or previously diagnosed atrial fibrillation (AF). Transesophageal echocardiography was performed in all eligible patients preferably within the first week of the onset of ischemic arterial stroke. All patients with normal TEE underwent holter to rule out paroxysmal AF.

Results: Totally, 50 CS patients were included in the study (mean age: 39.6±9.4 years, 68% female). Arterial hypertension (20%), smoking (14%), hyperlipidemia (8%), and diabetes (8%) were the most frequent vascular risk factors. Patent foramen ovale was detected in 19 (38%) patients and it was the most common cardiac abnormality in CS patients. Atrial septal aneurysm was found in 7 (14%) patients. In 5 patients (10%), atrial septal aneurysm was found along with a PFO. Atrial fibrillation on holter was seen in 2 patients (4%) with normal TEE. Comparison of patient characteristics with and without PFO was shown in Table 2. Patient with PFO was younger than patients without PFO (38.6±7.2 years versus 40.2±10.6 years, p=0.55) but not statistically significant. The mean RoPe score in patients with PFO was higher than patients without PFO, although it did not reach statistical significance (7.68±1.1 versus 6.77±1.9 p=0.07). 18 of 19 patients with PFO have RoPe score equal or above 7. We could not find any relation between migraine and PFO. Patients with PFO have mostly single infarct during radiological examination (14, 42.4% versus 11, 33.3%, p=0.04).

Conclusions: In our study, PFO prevalence in the CS patients was 38% as much higher than the widely accepted 25% in normal population. The RoPE score cutoff was ⩾7 for PFO in patients with CS. The RoPE score may calculate the probability that PFO is causally related to stroke. Transesophageal echocardiograhy is the gold standard to detect PFO and RoPe score may help to decide which patients underwent TEE.

Table 1. RopE Score Calculator

Characteristic	Points
No history of hypertension	1
No history of diabetes	1
No history of stroke or TIA	1
Nonsmoker	1
Cortical infarct on imaging	1
Age, year	
18–29	5
30–39	4
40–49	3
50–59	2
60–69	1
>=70	0
Total score (sum of individual points)	10 RoPE - Risk of Paradoxical Embolism

Table 2. Comparison of patient characteristics with and without PFO

Patient characteristics	PFO (n=19)	non-PFO (n=31)	*P*-value
Age	38.6±7.2	40.2±10.6	0.55
Female	14 (28%)	20 (40%)	0.71
Hypertension	2 (4%)	8 (16%)	0.28
Diabetes	1 (2%)	3 (6%)	1.0
Smoking	3 (9.7%)	4 (12.9%)	1.0
Dyslipidemia	1 (2%)	3 (6%)	1.0
RoPe Score	7.68±1.1	6.77±1.9	0.07
Migraine	4 (8%)	6 (12%)	1
Single lesion*	14 (42.4%)	11 (33.3%)	0.04
Multiple lesion*	2 (5.7%)	7 (20%)	1.13
Superficial lesion*	8 (22.9%)	8 (22.9%)	0.9
Large lesion*	1 (3%)	4 (12.1%)	0.34

*Radiologic variables of lesions Data are n (%), mean ± SD PFO - patent foramen ovale

Cardiac imaging / Echocardiography

OP-006

Importance of troponin-T levels and two-dimensional speckle tracking echocardiography in the assessment of cardiac injury during immune checkpoint inhibitor therapy for non-small cell lung cancer

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Background and Aim: Cancer immunotherapy with immune checkpoint blockers (ICI) has revolutionised the management of a wide variety of malignancies endowed with poor prognosis. Interestingly, early animal studies have demonstrated that CTLA-4 and PD-1 deletion can cause cardiac damage. The left ventricular ejection fraction (LVEF) assesses cardiac function and troponin-T (TnT) is widely recognized as a diagnostic marker of cardiotoxicity from cytotoxic chemotherapy treatment. However these tests sometimes are unable to identify cardiac damage. Developments in echocardiographic imaging, such as strain rate imaging (SRI), have increased the accuracy of myocardial functional assessment. In this study we aimed to investigate the role of cardiac biomarker (TnT) and SRI to assess early cardiac involvement during ICI therapy for non-small cell lung cancer (NSCLC).

Methods: This study included 30 NSCLC patients treated with ICI (monoclonal antibodies targeting PD-1 (e.g., nivolumab, pembrolizumab) and PD-L1 (e.g., atezolizumab) between June 2019 and June 2021. Patients with a history of ischemic or valvular heart disease, abnormal renal tests (creatinine >1.5 mg/dL), and LVEF below 55% were excluded from the study. TnT levels, conventional echocardiographic and left and right ventricular global longitudinal strain and strain rate (GLS and GLSR) parameters were recorded before, at the end of first and third months of ICI treatment.

Results: The average age of the patients was 55.0±9 years. 25 of the patients (83.3%) were male. We did not observe significant difference between TnT levels (before the treatment, first and third month follow up) (p1: 0.169, p2: 0.161, p3: 0.184, respectively) (Table 1). Additionally, LVEF, E/e', E/A, TAPSE, sPAB and other echocardiographic parameters did not differ significantly within before the treatment, first month and third month following ICI therapy (LVEF p1: 0.18, p2: 0.13, p3: 0.57), (E/e' p1: 0.10, p2: 0.11, p3: 0.51), (E/A p1: 0.25, p2: 0.20, p3: 0.25), (TAPSE p1: 0.26, p2: 0.28, p3: 0.11), (sPAB p1: 0.06, p2: 0.61, p3: 0.34). (All echocardiographic parameters are shown in Table 2). Although the strain values were within normal limits, GLS and GLSR values for RV and LV were reduced within the first month and third month following ICI therapy (LV GLS p1: 0.01, p2: 0.001 p3: 0.001), (LV GLSR p1: 0.01, p2<0.001, p3: 0.005), (RV GLS p1: 0.03, p2<0.001, p3: 0.001), (RV GLSR

p1: 0.02, p2: 0.003, p3: 0.01) (Table 2). These results showed that; LV GLS, LV GLSR, RV GLS and RV GLSR values were decreased by 1.0%, 2.5%, 1.1% and 2.9% in the first month, 5.3%, 10.2%, 6.3% and 8.8% in the third month sequentially.

Conclusions: In this study; we showed that SRI is a sensitive and valuable method to detect ICI-induced changes in cardiac function in NSCLC patients that cannot be detected by conventional echocardiographic measures and cardiac biomarkers. These findings should be investigated in long-term follow-up studies with larger series of patients.

Variables (N:30)	Before ICI therapy	One month after ICI therapy	Three month after ICI therapy	P1	P2	P3
Tn T (ng/dl)	5.6±1.4	5.7±1.3	5.8±1.3	0.169	0.161	0.184

Variables (N:30)	Before ICI therapy	One month after ICI therapy	Three month after ICI therapy	P1	P2	P3
EF %	61.3± 1.4	61.1± 1.3	61.0±.1.1	0.18	0.13	0.57
LVEDD (mm)	4.61±0.30	4.63±0.29	4.63±0.29	0.31	0.13	0.76
LVESD (mm)	3.02±0.28	3.03±0.28	3.04±0.29	0.08	0.26	0.50
IVS (mm)	1.16±1.48	1.17±1.48	1.17±1.4	0.57	0.10	0.78
PW (mm)	0.88 ±0.09	0.89±0.09	0.89±0.10	0.26	0.10	0.74
LA (mm)	3.63±0.17	3.64±0.18	3.65±0.19	0.43	0.08	0.48
RA (mm)	3.50±0.18	3.51±0.19	3.51±0.18	0.08	0.35	0.82
RV (mm)	3.18±0.23	3.25±0.26	3.18±0.22	0.07	0.71	0.09
sPAB (mm/hg)	23.8±3.0	24.8±3.1	24.1±3.3	0.06	0.61	0.34
E/e'	6.6±1.6	7.0±1.6	7.3±2.6	0.10	0.11	0.51
E/A	1.24±0.30	1.26±0.28	1.24±0.30	0.25	0.20	0.25
Tapse	2.33±0.23	2.34±0.22	2.30±1.95	0.26	0.28	0.11
LV GLS	-18.7±1.4	-18.5±1.2	-17.7±1.0	0.01	0.001	0.001
LV GLSR	0.78±0.13	0.76±0.11	0.70±.0.15	0.01	<0.001	0.005
RV GLS	-17.3±1.6	-17.1±1.4	-16.2±1.3	0.03	<0.001	0.001
RV GLSR	0.68±0.16	0.66±0.14	0.62±0.12	0.02	0.003	0.01

P1: Before ICI vs. one month after ICI therapy; P2 : Before ICI vs. three month after ICI therapy; P3 : One month after ICI therapy vs. three month after ICI therapy

Tables 1-2.

Hypertension

OP-007

The relationship between circulating serum omentin-1 levels and nascent metabolic syndrome in hypertensive patients

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Background and Aim: The prevalence of metabolic syndrome (MetS) is more common in hypertensive patients and is associated with an increased risk of target organ damage and/or cardiovascular diseases (CVD). Omentin-1 is a beneficial adipokine considered to play a role in metabolic syndrome (MetS) and MetS-related states such as obesity, diabetes and coronary artery disease. The goal of this study was to reveale the relationship between circulating omentin-1 levels and MetS uncomplicated by diabetes or CVD, defined as nascent MetS, in hypertensive patients.

Methods: 110 patients (average age 49.72±11.32 years, male gender 49%) under treatment for hypertension but without overt diabetes and/or CVD were enrolled in this study. According to American Heart Association/National Heart, Lung and Blood Institute criteria, 66 patients were stratified as MetS (+) (group I), 44 patients as MetS (-) (group II). The triglyceride glucose (TyG) index was used to evaluate insulin resistance (IR). Circulating omentin-1 levels were measured by enzyme-linked immunosorbent assay (ELISA) kit in venous blood samples.

Results: Circulating omentin-1 levels were significantly lower in patients with MetS compared with those without [46.35 (42.7-57.7) vs. 130.95 (62.83-236.48), p<0.001]. Omentin-1 was inversely correlated with TyG index (r=-0.204, p=0.033). In multivariate logistic regression analysis using backward stepwise method, omentin-1, TyG index and body mass index (BMI) were independent predictors for MetS presence. The ROC curve analysis determined the best cut off value for omentin-1 as 62.20 ng/ml in predicting MetS presence, and the area under the curve (AUC) was 0.880 (95% CI=0.817-0.942, p<0.001).

Conclusions: In this study, circulating omentin-1 levels were obviously decreased in hypertensive patients with nascent MetS. According to the study findings, it can be thought that omentin-1 plays a role in the development of MetS in this population. It may also be a useful marker for identifying hypertensive patients at high risk for MetS.

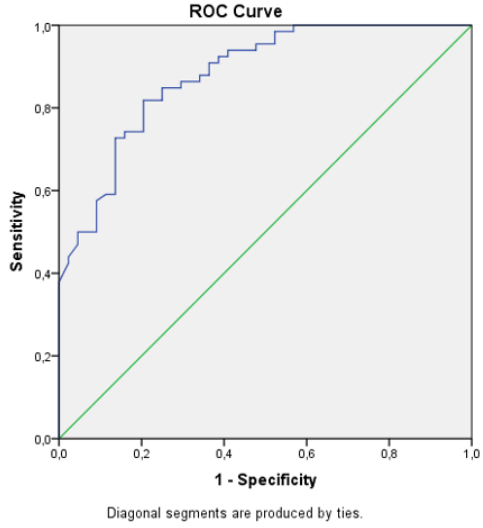

Figure 1. The receiver operating characteristic (ROC) curve of omentin-1 in predicting metabolic syndrome presence

Table 1. Baseline demographic and clinical characteristics of study population

Variables	All population (n=110)	Group I (n=66)	Group II (n=44)	*P*-value
Age (years)	49.72±11.32	49.08±10.56	50.68±12.45	0.469
Male, n (%)	54 (49)	33 (50)	21 (47.7)	0.815
BMI, kg/m^2	28.40±4.82	29.18±5.20	27.24±3.96	0.049
WC (cm) (IQR)	92 (84-102)	93.5 (84.75-104.25)	90 (84-99.5)	0.153
WHtR	0.56±0.07	0.57±0.08	0.54±0.06	0.132
Smoking, n (%)	37 (33.6)	22 (33.3)	15 (34)	0.934
EF (%) (IQR)	65 (60-65)	65 (60-65)	65 (60,25-65)	0.374
SBP (mmhg)	124.265±5.02	128.98±5.39	115.95±4.92	0.032
DBP (mmHg)	76.96±3.89	80.48±5.52	74.50±3.43	0.046

BMI - body mass index; WC - waist circumference; WHtR - waist circumference to height ratio; EF - ejection fraction; SBP - systolic blood pressure; DBP - diastolic blood pressure; IQR - interquartile range

Table 2. Laboratory findings of study population

Variables	All population (n=110)	Group I (n=66)	Group II (n=44)	*P*-value
Glucose, mg/dL	96.16±10.12	97.17±10.65	94.66±9.19	0.234
GFR, mL/min	94.46±14.95	93.27±15.10	96.25±14.71	0.306
TG, mg/dL	160.02±85.01	179.09±92.74	131.41±62.62	0.002
Tchol, mg/dL	190.56±33.67	192.98±28.62	186.54±40.84	0.271
LDL-C, mg/dL	111.30±29.90	111.72±27.14	110.63±34.34	0.637
HDL-C, mg/dL	48.10±10.38	45.85±9.48	51.48±10.87	0.006
CRP, mg/dL	0.32±0.37	0.34±0.36	0.31±0.38	0.107
Hemoglobin, g/dL	14.68±1.86	14.75±1.77	14.57±2.01	0.621
WBC, cells/µL	8.12±2.04	8.22±1.94	7.97±2.19	0.618
Omentin-1, ng/ml (IQR)	55.05 (44.2-121.53)	46.35 (42.7-57.7)	130.95 (62.83-236.48)	<0.001
TyG index	8.82±0.53	8.96±0.53	8.62±0.45	0.001
TG/HDL-C	3.70±2.60	4.26±2.86	2.85±1.87	0.001
LDL-C/HDL-C (IQR)	2.34 (1.95-2.81)	2.36 (1.94-3.04)	2.29 (1.91-2.67)	0.333

GFR, glomerular filtration rate; TG, triglycerides; TChol; total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; CRP, C-reactive protein; WBC, white blood cells; TyG index, triglyceride glucose index; IQR, Interquartile range

Table 3. Multivariate logistic regression analysis for metabolic syndrome presence

Variables	*P*-value	OR	95% CI
Constant	0.004	0.000	-
Omentin-1	0.000	0.962	0.945-0.979
TyG index	0.005	6.813	1.783-26.040
BMI	0.020	1.161	1.023-1.318

a. Variable(s) entered on step 1: Omentin-1, TyG index, BMI, TG/HDL, CRP Abbreviations: TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; TyG index, triglyceride glucose index; BMI, body mass index; CRP, C-reactive protein; OR, odd ratio; CI, confidence intervale

Epidemiology

OP-008

Association of *ITLN1* rs2274907 gene polymorphism and serum intelectin-1 level with hypertension in Turkish population: In cross-sectional study

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Background and Aim: Intelectin-1 also known as an endothelial lectin HL-1, is highly and selectively expressed in visceral adipose tissue. It has vasodilation and anti-inflmamation effects on isolated blood vessels through endothelium-derived nitric oxide. High blood pressure causes increased inflammatory activity and endothelial dysfunction. In this study, it was aimed to investigate the relationship between *ITLN1* rs2274907 A>T polymorphism and serum intelectin-1 leves with hypertension.

Methods: The genotype analysis was performed using LightSNiP assay in a group of 1260 hypertensive and 1003 non-hypertensive individuals participating in the "Turkish Adults Risk Factors (TARF)" cohort study.

Results: The genotype distributions of rs2274907 A>T polymorphism in the hypertensive individuals were 53.7% (n=677) for AA, 38.8% (n=488) for AT and 7.6% (n=95) for TT genotypes, while 49.9% (n=501) for AA, 41.9% (n=420) for AT and 8.2% (n=82) for TT in non-hypertensive individuals. There were no significant differences in genotype and allele distributions between two groups (p>0.05). However, serum intelectin-1 levels were higher in hypertensive men than non-hypertensive men (p=0.004). In addition, TT genotype was found to be associated with increased total cholesterol (p=0.010) in hypertensive male individuals.

Conclusions: Our results suggest that the *ITLN1* rs2274907 A>T gene polymorphism may not be associated with the risk of hypertension in the Turkish population, but may have an effect on serum intelectin-1 levels.

Lipid / Preventive cardiology

OP-010

Awareness level of patients about the significance of statin treatment and the effect of misinformations in communication networks on their medication compliance

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Background and Aim: In spite of many evidences supported by literature, unfortunately it is possible to encounter a lot of news on television or social media against statin treatment. Although patients were informed well by physicians; we know that these kind of misinformations on media may effect their medication compliance negatively. Our aim was to achieve the real life data about the effect of misleading news on statin therapy adherence of patients.

Methods: This was a cross-sectional study in a tertiary center (Tepecik Training and Research Hospital, İzmir, Turkey) which included 480 patients under statin treatment. We filled out a questionnaire for patients who admitted to our cardiology clinic. The questionnaire contained patients' demographic characteristics and their awareness level about the significance of statin therapy; indication, dose, advers events and type of statin treatment; level of lipid parameters in last six months (with any dose adjustment); and also medication compliance of patients despite misleading informations they encountered on television or internet.

Results: Mean age of participants was 54.2 (+18.4) and 57% (n=274) of study population were female. Thirty-three patients were taking statin for primary prevention (e.g. diabetes mellitus) and others were taking for secondary prevention (e.g. coronary heart disease). Awareness level of the importance of statin therapy was 82.3% (n=395) and this was not correlated with education level, age and sex of participants. While total of 302 patients' (62.9%) LDL levels were in target range according to current guidelines; other participants' LDL levels were above the target levels and the main reason of this finding was poor medication adherence because of two factors: (1) adverse events of the drugs such as myopathy (4.5%, n=8), (2) the news they encountered on television or social media and people's suggestions from their social life (49.4%, n=88). Medication compliance was worse in patients who were taking statin for primary prevention compared to patients who were taking the drug for secondary prevention (74.6% vs. 82.8%, p<0.05, respectively). Some participants had decreased the dose of the drugs by themselves or took them every other day; and some of them had given up the treatment completely. We enlightened patients about the importance of statin therapy and misleading news; medication compliance rate significantly increased from 80.0% (384 of 480) to 98.3% (472 of 480) (p<0.05) and patients with target level of LDL also significantly increased from 62.9% (302 of 480) to 93.3% (448 of 480 participants) (p<0.05) at control visit 1 month later.

Conclusions: We found in our study that 18.3% of patients whether decreased their statin dose or took the medicine every other day, or qive up the therapy completely because of misleading news. We also observed a remarkable increase in medication compliance and effectiveness after we informed participants about misinformations and the importance of the therapy.

Lipid / Preventive cardiology

OP-011

Predictive role of protein convertase subtilisin kexin type-9 levels for fatty plaque morphology in asymptomatic intermediate carotid stenosis – A cross sectional study

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Background and Aim: Carotid plaques are divided into calcified, mixed and fatty types according to the morphological ultrastructure. Increased risk of rupture, thromboembolism and stroke are more pronounced in fatty carotid plaques due to composition and instability. The present study aimed to assess the role of protein convertase subtilisin kexin type-9 (PCSK9) to predict fatty plaques in patients with intermediate carotid artery stenosis.

Methods: A total of 213 asymptomatic patients who had 50-70% stenosis in the carotid artery were included in this cross-sectional study. Patients were assigned into three groups based on plaque morphology: 90 with calcified, 68 with mixed and 55 with fatty plaques. Groups were compared in terms of PCSK9. Predictive role of PCSK9 for fatty plaque was investigated.

Results: PCSK9 was significantly higher in fatty plaque group. It with a cut off of 197.4, had 81.8% sensitivity and 61.4% specificity for prediction of fatty plaque (AUC: 0.760, 95% CI: 0.694-0.827, p<0.001). Multivariate regression analysis showed age (OR: 0.907, p=0.001), PCSK9 (OR: 1.045, p<0.001) and neutrophile count (OR: 3.932, p<0.001) were significant independent predictors of fatty plaques in intermediate carotid artery stenosis, after adjusting for other risk factors.

Conclusions: PCSK9 plays a favourable role for prediction of fatty plaques in asymptomatic intermediate carotid artery stenosis.

Cardiovascular nursing / Technician

OP-012

Correlation between acceptance of illness and comfort level in patients with heart failure

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Background and Aim: Acceptance of illness in chronic diseases like heart failure which continue for a life time, is highly significant in adaptation to treatment and lifestyle changes.

The connection between acceptance of illness and quality of life in patients with heart failure is known, whereas the connection with comfort level is not. The aim of this study was to determine the correlation between acceptance of illness and comfort level in patients with heart failure.

Methods: The descriptive study was conducted with 106 patients diagnosed with heart failure in the cardiology service in a university hospital. The data of the study was collected using the personal information form, the Acceptance of Illness Scale and the General Comfort Questionnaire between November 2019 and February 2020. In the descriptive statistics for data analysis, the Student's t, One-way Anova, Welch's test for group variances, Bonferroni, Games-Howell test and Pearson's correlation coefficient for pairwise comparisons were used.

Results: 55.7% of the patients included in the study were male and their mean age was 67.90±11.6 years. In the study the Acceptance of Illness Scale and the General Comfort Questionnaire mean scores of the patients were found to be 18.79±7.75 and 2.65±0.40, respectively. The patients who were non-literate, were low-income and high-income and whose duration of HF diagnosis was 4 years or above, had a lower Acceptance of Illness Scale mean score than other groups ($p<0.05$). A moderate positive correlation was identified between the Acceptance of Illness Scale and the General Comfort Questionnaire total scores ($r: 0.517$, $p<0.001$).

Conclusions: According to the results of the study, the acceptance of illness level in HF patients was lower, while the comfort level was medium. As the illness acceptance level of the heart failure patients increased, their comfort level increased.

Interventional cardiology / Valve and structural heart diseases

OP-013

Naples prognostic score as a novel prognostic prediction tool in patients undergoing transcatheter aortic valve implantation

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Background and Aim: Transcatheter aortic valve implantation (TAVI) is well established as a treatment for intermediate-high risk patients with aortic stenosis (AS). Predicting postoperative mortality among patients suitable for TAVI is crucial to identify patients who need close follow-up and treatment. The Naples prognostic score (NPS) calculated from serum albumin, total cholesterol concentrations and hematological indices is an index assessing both systemic inflammatory response and malnutrition. NPS is an independent prognostic indicator for the outcomes of patients with different types of cancer. We aimed to investigate the prognostic impact of NPS in patients undergoing TAVI in our institute.

Methods: We retrospectively reviewed 430 consecutive patients with severe AS who underwent TAVI at the Bezmialem Foundation University School of Medicine Hospital between March 2012 and December 2020. The NPS was calculated based on the following 4 parameters: serum albumin, total cholesterol, the neutrophil:lymphocyte ratio (NLR) and the lymphocyte:monocyte ratio (LMR) as described previously (Fig. 1). All patients were regularly followed up after TAVI. Patients were contacted mainly via outpatient examination or phone call according to our institutional regulations. The time intervals for follow-up were every 3 months for the first 3 years, and then every 6 months in the following years. Quantitative variables were expressed as mean ± standard deviation and qualitative variables as numbers and percentages. Long-term survival analysis was performed using Kaplan–Meier curves and subgroups were compared using a log rank test. Univariate predictors of overall survival were entered into Cox proportional hazards model to identify independent predictors of mortality. SPSS statistical software pack (SPSS 27.0 for Windows, Inc., Chicago, IL, USA) was used for data analysis. Significance was assessed at a p level of <0.05.

Results: The mean age of the patients was 79.5±8.1 years. Female predominance was observed (n=248, 57.7%). We divided the patients into 3 groups according to NPS: patients with NPS 0 (n=24, 5.6%) were defined as group 0, patients with a score of 1 to 2 (n=213, 49.5%) were defined as group 1, and patients with a score of 3 to 4 (n=193, 44.9%) were defined as group 2. 183 patients (42.6%) died during 175.6±125.6 weeks follow up. Mortality rates of the groups were found as: 2/24 (8.3%) in group 0, 66/213 (31%) in group 1 and 115/193 (59.6%) in group 2 ($p<0.001$) (Fig. 2). Univariate analysis was performed to determine the potential predictors of mortality including age, gender, co-morbidity, left ventricular ejection fraction <50%, pacemaker implantation and cerebrovascular accident. Multivariate analysis identified NPS (OR: 3.38, 95% CI 2.08–5.50, $p<0.001$) as a significant independent predictor of mortality.

Conclusions: Preoperative NPS is a simple, easily obtainable scoring system strongly associated with outcome in patients with AS who are undergoing TAVI.

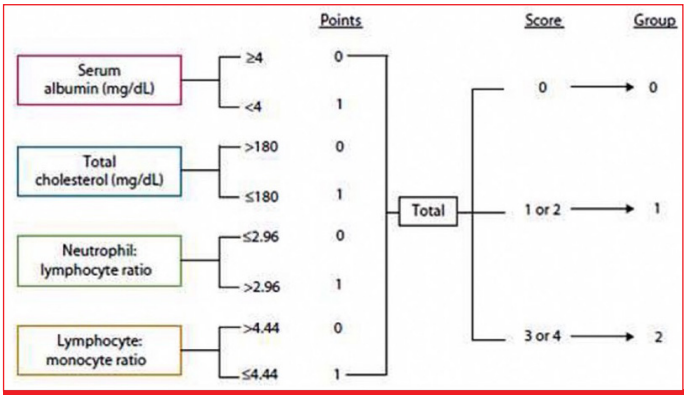

Figure 1. Calculation of the Naples prognostic score

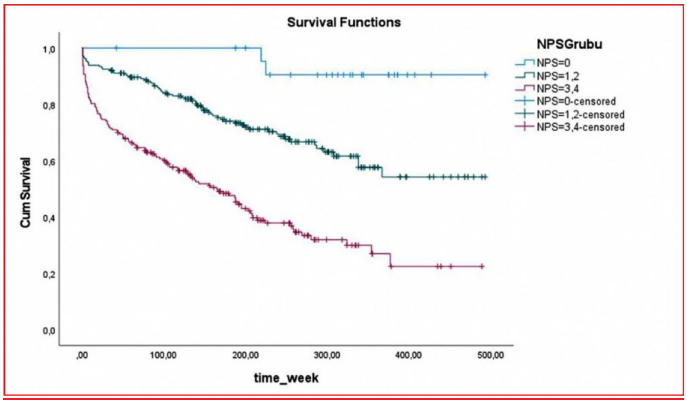

Figure 2. Kaplan-Meier survival curves for NPS groups

Interventional cardiology / Valve and structural heart diseases

OP-014

Predictive role of the InsCor score for the mortality in patients undergoing transcatheter aortic valve implantation

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Background and Aim: Transcatheter aortic valve implantation (TAVI) is the accepted treatment option in patients with severe aortic stenosis who are under intermediate to high surgical risk. Risk scores are important tools for predicting adverse events in TAVI, but their accuracy varies when applied to different populations. The objective of this study is to evaluate the performance of the Brazilian score InsCor as a predictor of mortality after TAVI compared to the European System for Cardiac Operative Risk Evaluation (EuroSCORE) and Society of Thoracic Surgeons (STS) scores.

Methods: A total of 161 patients (81 male, 80 female) with severe symptomatic aortic stenosis undergoing TAVI were included. We retrospectively investigated patients. The one-year mortality of the patients was evaluated. Baseline demographic and clinical variables were recorded as well as InsCor Score; Age over 70 years=3 point, female gender=2 point, recent infarction<90 day=2 point, reoperation= 3 point, creatinine greater than 2 mg/dl=5 point, LV ejection fraction less than 30%=3 point preoperative events (use of preoperative inotropic support, cardiogenic shock, cardiac resuscitation, use of intra-aortic balloon, acute renal failure, cardiac massage, tracheal intubation, tachycardia or ventricular fibrillation)=5 point. Patients were divided into two groups as patients without mortality (140 patients) and patients with mortality (21 patients).

Results: During the follow up period, one-year total mortality rate was 13% (21/161). A higher InsCor Score (p=0.013) and STS/TAVI Score (p=0.001) was foıund to be associated with the mortality. Deceased patients had higher frequencies of cerebrovascular accident (CVA, p<0.001) and NYHA class III-IV (p=0.015) than survivors. In addition, left ventricular ejection fraction (p=0.041) and estimated glomerular filtration rate (p=0.038) was lower in patients who died. Multivariate regression analysis showed that high STS/TAVI score (HR=1.493, 95% CI=1.168-1.908, p=0.001), low left ventricular ejection fraction (HR=0.956, 95% CI=0.914-0.999, p=0.047) and total cholesterol (HR=0.992, 95% CI=0.986-0.998, p=0.014) were independently predictor of one-year mortality but InsCor score was not.

Conclusions: Although a Higher InsCor score has been observed in TAVI-caused deaths, it did not predict the one-year mortality. Further large-scale prospective studies are needed to clarify this issue.

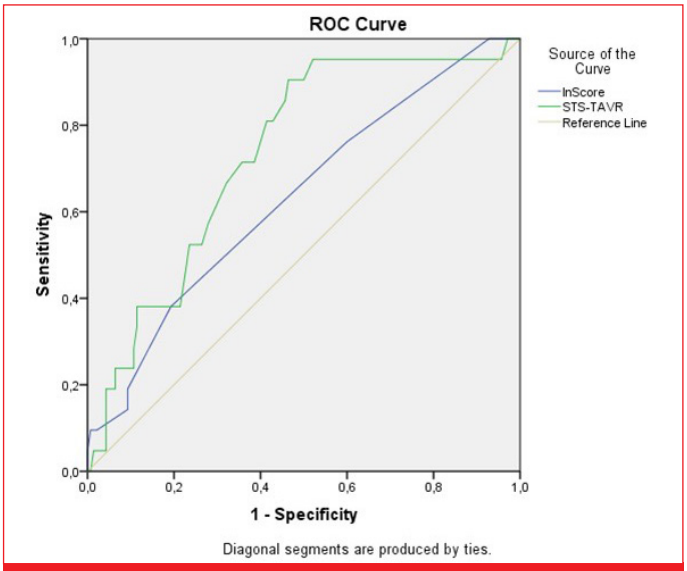

Figure 1. Receiver-operating characteristic curves indicating the discriminative ability of the STS/TAVR and InsCor score
InsCor score AUC: 0.632 95% CI 0.506-0.759, p=0.051 STS/TAVI AUC: 0.733 95% CI 0.629- 0.838 p=0.001

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

Variables	All population	Alive n=140 (87%)	Dead n= 21 (13%)	*P*-value
Male gender, n %	81 (50.3)	71 (50.7)	10 (47.6)	0.791
Age	78.2± 6.8	78.2±6.7	77.4±8.1	0.648
BMI	26.7±4.1	26.7±4.1	27.4±4.7	0.452
Hypertension, n (%)	103 (64)	89 (63.6)	14 (66.7)	0.783
Vascular disease history, n (%)	87 (54)	74 (52.9)	13 (61.9)	0.438
CVA history, n (%)	17 (10.6)	10 (7.1)	7 (33.3)	<0.001
NYHA Class III-IV, n (%)	68 (42.2)	54 (38.6)	14 (66.7)	0.015
Atrial fibrillation, n (%)	27 (16.8)	23 (16.4)	4 (19)	0.765
Presence of BBB, n (%)	21 (15)	21 (15)	4 (19)	0.633
Aortic valve area, cm^2	0.71±0.15	0.71±0.15	0.70±0.19	0.695
Mean Aortiv valve gradient, mm Hg	48.2±9	47.9±8.5	50.6±11.6	0.199
Left ventricular ejection fraction, %	51.7±9.4	52.2±9.1	47.8±10.7	0.041
sPAP, mm Hg, [IQR]	39.8±13.2	39.6±13.1	41.3±14.3	0.588
STS/TAVR score, [IQR]	3.8 [2.6-5.2]	3.5 [2.5-4.9]	5.0 [4.0-7.1]	0.001
EuroScore, [IQR]	4.6 [3.3-8.2]	4.4 [3.1-7.9]	6.6 [4.1-11.9]	0.127
InsCor score, [IQR]	5.0 [3.0-5.0]	5.0 [3.0-5.0]	5.0 [4.0-6.0]	0.013
FBG, mg/dL	135±44	136±44	128±42	0.446
Hematocrit, %	36.2±5.5	36.4±5.3	35.2±6.8	0.388
WBC, $10^3/\mu L$	7.4±2.1	7.4±2.1	6.9±1.9	0.264
Platelet, $10^3/\mu L$	231±80	234±84	216±43	0.333
CRP, mg/L, [IQR]	5.9 [3.2-15.9]	5.9 [3.0-15]	6 [3.1-16.5]	0.647
eGFR, mL/min/1.73 m^2	64±19	65±18	56±22	0.038
Diabetes mellitus, n (%)	76 (47.2)	68 (48.6)	6 (38.1)	0.370
COPD, n (%)	71 (44.1)	60 (42.9)	11 (52.4)	0.412

BBB - bundle branch block; BMI - body mass index; COPD - chronic obstructive pulmonary disease; CRP - C-reactive protein; CVA - cerebrovascular accident; eGFR - estimated glomerular filtration rate; EuroSCORE II -European System for Cardiac Operative Risk Evaluation II; FBG - fasting blood glucose; IQR - interquartile range; NYHA - New York Heart Association; sPAP - pulmonary artery systolic pressure; TAVR - transcatheter aortic valve replacement; WBC - white blood count

Table 2. Procedural and postprocedural parameters of study population during the follow-up period

Variables	All population	Alive n=140 (87%)	Dead n=21 (13%)	*P*-value
Type of Valve, n (%)				0.204
EVOLUT R	19 (11.8)	18 (12.9)	1 (4.8)	
SAPIEN XT	52 (32.3)	41 (29.3)	11 (52.4)	
PORTICO	68 (42.2)	60 (42.9)	8 (38.1)	
ACURATE	11 (6.8)	10 (7.1)	1 (4.8)	
Direct flow	11 (6.8)	11 (7.9)	0 (0)	
Predilatation, n (%)	92 (57.1)	85 (60.7)	7 (33.3)	0.018
Post dilatation, n (%)	24 (14.9)	19 (13.6)	5 (23.8)	0.219
Implantation depth, mm	5.27±0.75	5.27±0.75	5.29±0.77	0.910
Paravalvular leakage (> 2+), n (%)	13 (8.1)	12 (8.6)	1 (4.8)	0.550
Major vascular complications, n (%)	25 (15.5)	23 (16.4)	2 (9.5)	0.415
Bleeding complications, n (%)	40 (24.8)	35 (25)	5 (23.8)	0.906
Pericardial Tamponade, n (%)	4 (2.5)	3 (2.1)	1 (4.8)	0.472
Acute kidney injury, n (%)	38 (23.6)	30 (21.4)	8 (38.1)	0.093
Permanent pacemaker, n (%)	19 (11.8)	18 (12.9)	1 (4.8)	0.284
Rehopsitalization, n (%) (cardiovascular-caused)	32 (19.9)	23 (16.4)	9 (42.9)	0.005
Postprocedural IS or TIA, n (%)	6 (3.7)	5 (3.6)	1(4.8)	0.788
Myocardial infarction, n (%)	2 (1.2)	2 (1.4)	0 (0)	0.582

IS - ischemic stroke; TIA - transient ischemic attack

Table 3. Two different univariable and multivariable cox proportional-hazards regression analysis models for determining the predictors of the mortality of 1-year

Parameters	Univariable OR (95% CI)	*P*-value	Multivariable OR (95% CI)	*P*-value
CVA history	6.318 (2.538-15.730)	<0.001	7.126 (2.468-20.573	<0.001
LVEF	0.955 (0.918-0.994)	0.024	0.956 (0.914-0.999)	0.047
GFR	0.976 (0.956-0.997)	0.026	1.021 (0.988-1.054)	0.215
STS/TAVR score	1.447 (1.231-1.702)	<0.001	1.493 (1.168-1.908)	0.001
InsCor score	1.338 (1.118-1.600)	0.001	1.074 (0.866-1.332)	0.515

Interventional cardiology / Valve and structural heart diseases

OP-015

Transcatheter tricuspid valve-in-valve and valve-in-ring ımplantation with a novel balloon expandable Myval® transcatheter heart valve

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Background and Aim: Tricuspid valve surgery is usually performed with a bioprosthetic valve (BPV) or ring method and valve durability is less longevity than other valvular prostheses. The transcatheter tricuspid valve-in-valve (TVIV) or valve-in-ring (TVIR) implantation is on the way to an important alternative to high-risk redo surgery in patients with degenerated tricuspid BPV or repair. Unlike common valve-in-valve therapy in the aortic position, there are only a few results for the tricuspid position. We report our experience of transcatheter TVIV and TVIR with novel novel balloon expandable Myval transcatheter heart valve (THV) system in patients with degenerated BPV and failed ring annuloplasty.

Methods: Transcatheter TVIV: A 59-year-old male patient was referred to our clinic from the center where he presented with recently increased edema in the legs, abdomen, and NYHA Class III dyspnea. Eleven years ago, he underwent a TV operation with a 31 mm bioprosthesis St. Jude Medical (SJM) Epic (St. Jude Medical, Inc, St. Paul, MN, USA) due to the Ebstein anomaly. Transthoracic echocardiography (TTE) revealed TV bioprosthesis stenosis or degenerated with a mean gradient of 15 mm Hg with moderate mitral regurgitation, mild impaired right ventricular dysfunction, massive right atrial dilatation

and a preserved LVEF (60%). The 29 mm Myval THV balloon-expandable valve was slowly implanted in the proper position on fluoroscopy. In the first month follow-up, improvement in functional capacity was observed, and 4 mm Hg mean gradient was detected in the tricuspid valve in echocardiography.

Results: Transcatheter TVIR: A 59-year-old female patient, firstly in 1989, closed mitral commissurotomy was first applied to the patient for mitral stenosis. Afterward, mitral valve replacement in 1991 and finally aortic valve replacement and a 34 mm Edwards MC3 rigid partial ring implantation together with tricuspid commissurotomy and patch augmentation were performed in 2016. Multi-slice computed tomography (MSCT) showed the shape of the annuloplasty ring very well, and the ring area, perimeter (933 mm), and diameter measurements (30 mm) were executed in detail without thrombus and vegetation. The 32 mm Myval THV balloon-expandable valve was slowly implanted under the rapid pacing (180 beats/min) in the precise position on fluoroscopy. After it was seen that Myval's functions were good with hemodynamic, echocardiographic, and fluoroscopic controls the procedure was terminated by applying a figure of eight sutures to the femoral vein. She was discharged with a mild valvular and mild paravalvular tricuspid regurgitation with mean gradient of 3.5 mm Hg after one day of uneventful hospitalization postoperatively.

Conclusions: In contemporary, to overcome this dilemma, case series concerning transcatheter TVIV or TVIR have emerged due to the risk of redo surgery. In the literature on transcatheter TVIR and TVIV, there are successful and unsuccessful cases with Edwards Sapien XT, SAPIEN 3 and Melody valves, but these Myval cases are the first.

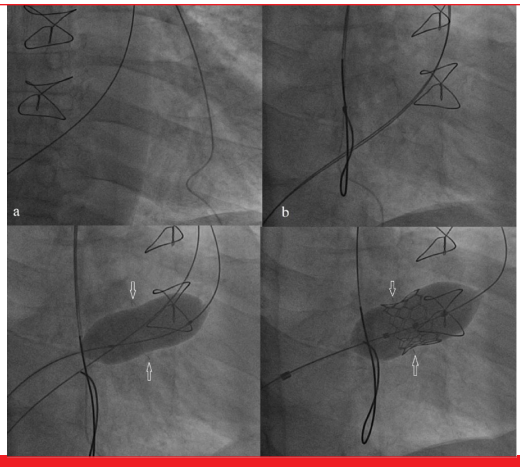

Figure 1.

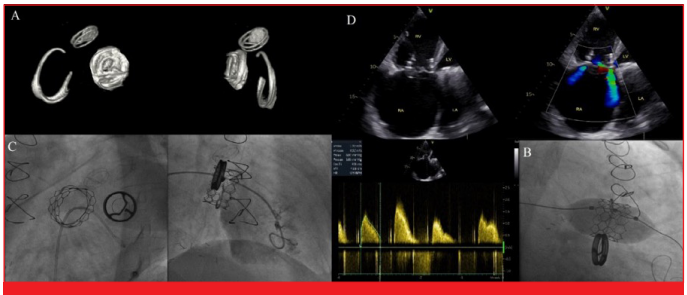

Figure 2.

Interventional cardiology / Valve and structural heart diseases

OP-016

Transcarotid transcatheter aortic valve implantation with a novel balloon expandable Myval® THV under the local anesthesia

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Background and Aim: Although many studies have shown that transfemoral transcatheter aortic valve implantation (TF-TAVI) is better than other alternative approaches in terms of morbidity and mortality, it has been shown that alternative routes, transapical, transsubclavian-axillary, or transcarotid routes, are used successfully in patients in whom transfemoral access is not possible. We presented transcarotid TAVI (TC-TAVI) with a novel balloon-expandable Myval transcatheter heart valve (THV) system in patients with a prohibitive abdominal aortic disease, which is the first in the literature.

Methods: A 77-year-old female patient with a history of coronary artery bypass grafting presented with exertional dyspnea (New York Heart Association Class III). She referred to our center due to the not suitable for TF-TAVI. Transthoracic echocardiography confirmed severe AS with a mean gradient of 54 mm Hg and an aortic valve area 0.57 cm2 with 50% left ventricular ejection fraction. Multi-slice computed tomography (MSCT) was revealed bilateral circumferential calcification with 5.5 mm luminal diameter in abdominal aorta below the renal arteries. A heavily calcified left subclavian artery was noted with stenotic proximal segment. The right common carotid artery (CCA) has no critical stenosis, but the left CCA originating from the arcus aorta (bovine arch) is 8.7 mm, and the internal carotid artery (ICA) is occluded. The patient was evaluated with the heart team, and it was decided to perform TAVI as a cut-down from the left CCA since the right carotid is essential for the brain and the left ICA is occluded.

Results: Under local anesthesia, right femoral access was achieved following an uneasy crossing through the tight abdominal aorta with a hydrophilic 0.35 guidewire, a pigtail catheter positioned into the aortic arch. Afterward, a 6F sheath was placed to the below carotid bifurcation surgically exposed left CCA using the Seldinger technique. An intravenous bolus of 5000 IU of heparin was given to reach an activated clotting time of 250-300 s. After the 6F AL2 catheter was passed through the aortic valve with a 0.38 flat-tipped guidewire, the Safari stiff guidewire was placed in the left ventricle and the 14Fr Expandable Introducer Sheath was in-

serted from the CCA to the ascending aorta over Safari wire. The 23 mm Myval THV system (Meril Life Sciences Pvt. Ltd., Vapi, Gujarat, India) was implanted under rapid ventricular pacing. Final aortography revealed mild paravalvular leak, mean gradient 11 mm Hg and the procedure was finished. No neurological, bleeding, vascular, and rhythm complications were observed in the coronary intensive care follow-up after the procedure. The patient was discharged home after 48 h on Clopidogrel 75 mg daily.

Conclusions: We present the first successful implantation of Myval THV under local anesthesia with no short-term complications via carotid artery access.

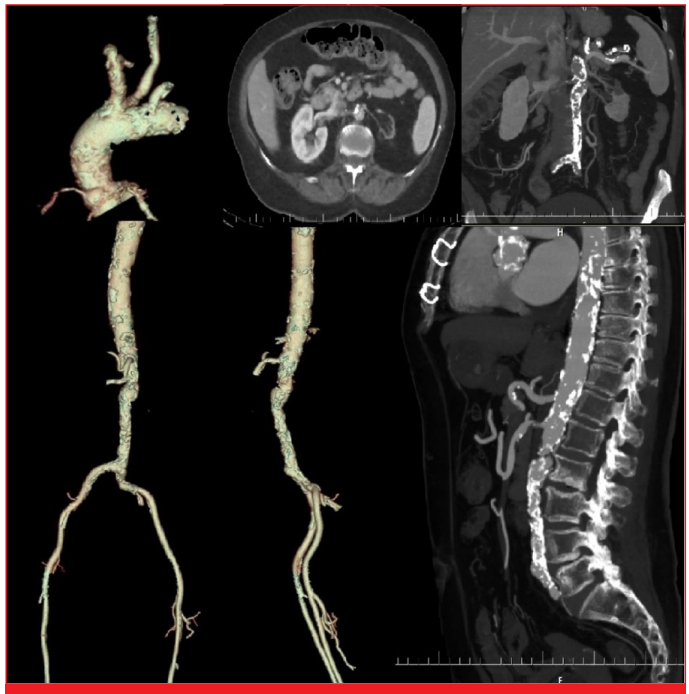

Figure 1.

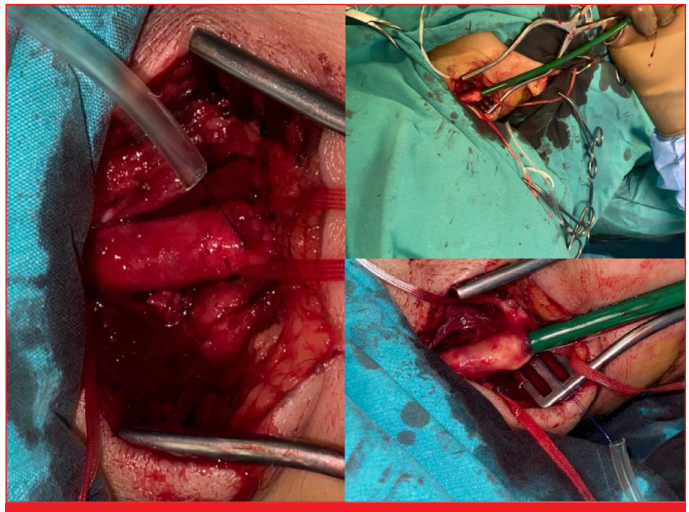

Figure 2.

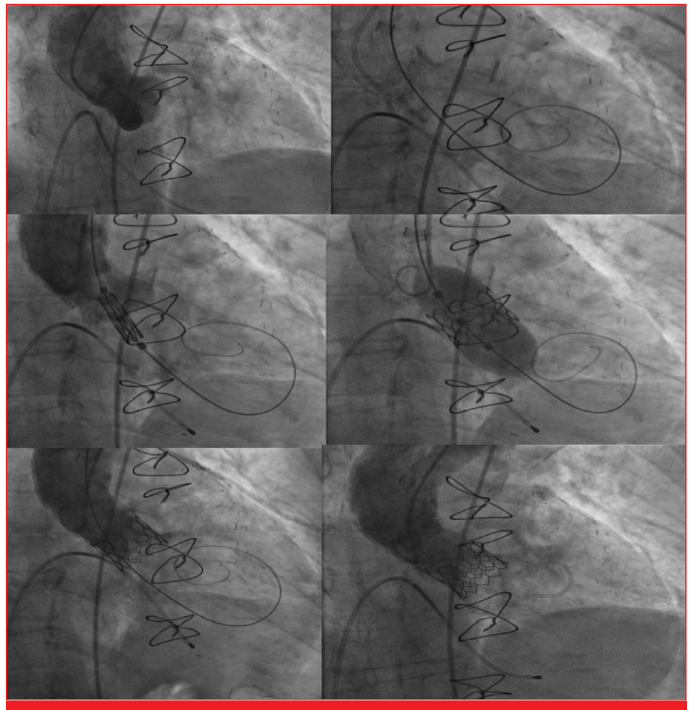

Figure 3.

Interventional cardiology / Valve and structural heart diseases

OP-017

Effects of Anemia on TAVR Outcomes

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Background and Aim: Transcatheter aortic valve replacement (TAVR) is a standard of care treatment option in patients with severe symptomatic aortic valvular disease who have intermediate-high surgical risk. Anemia is frequent in patients with aortic stenosis due to increased bleeding episodes as a result of decreased von Willebrand factor levels and presence of additional comorbidities such as iron deficiency, malignancies and myelodysplastic syndrome. We aimed to evalaute the effects of anemia and hemoglobin level on TAVR outcomes.

Methods: All of the patients who underwent TAVR procedure consecutively between January 2010 and June 2020 in Hacettepe University Hospital were enrolled. Anemia was graded according to WHO classification and patients' characteristics and mortality datas were obtained from hospital electronic database.

Results: A total of 308 patients were enrolled in the study. Mean age of the study population was 76.0±10.4 years and 39% of the patients (n=120) were male. Median follow up

was 28.64 (13.1-49.8) months. Milde anemia was detected in 81 patients, moderate to severe anemia was detected in 100 patients while 127 patients had normal hemoglobin levels. Baseline characteristics were similar between patients with mild anemia, moderate to severe anemia and without anemia except male gender, beta blocker use and sPAP value. Baseline characteristics were presented in Table-1. Male gender ratio was significantly lower while beta blocker use and sPAP value were significantly higher in patients with moderate-severe anemia than other cases (Table 1). Rate of TAVR related complications including insertion site complications, stroke and permanent pacemaker implantation were similar between patients with and without anemia (Table 2). Contrast nephropathy, in hospital and all cause mortality rates were significantly higher in patients with moderate-to severe anemia than mild anemia or without anemia (Table 2).

Conclusions: Anemia is a frequent finding in patients who underwent TAVR. Both the presence and severity of anemia is related with increased rates of contrast nephropathy, in hospital mortality and all cause mortality. Although the frequency of favorable parameters for cardiovascular mortality such as beta blocker use and female gender were higher in patients with moderate to severe anemia than remaining cases; both in hospital and all cause mortality rates were higher in the same group.

Table-1. Baseline characteristics

	Normal Hemoglobin (n=127)	Mild Anemia (n=81)	Moderate-Severe Anemia (n=100)	p value
Age, years, mean ± sd	74,5 ± 10,8	76,6 ± 9,6	77,5 ± 10,4	0,075
Gender, male, n (%)	51 (40,2 %)	39 (48,1 %)	30 (30 %)	0,041*
Comorbidities, n (%):				
*Hypertension	95 (74,8 %)	59 (72,8 %)	75 (75 %)	0,936
*Diabetes	33 (26 %)	33 (40,7 %)	33 (33 %)	0,084
*COPD	22 (17,3 %)	20 (24,7 %)	23 (23 %)	0,376
*Coronary artery disease	51 (40,2 %)	38 (46,9 %)	43 (43 %)	0,631
*Atrial fibrillation	27 (21,2 %)	11 (13,6 %)	24 (24 %)	0,450
*Heart failure	31 (24,4 %)	17 (20,9 %)	21 (21 %)	0,831
Drugs, n (%):				
*Beta blockers	82 (64,4 %)	42 (51,9 %)	75 (75 %)	0,005*
*RAAS inhibitor	74 (58,3 %)	39 (48,1 %)	51 (51 %)	0,310
*Statin	46 (32,2 %)	39 (48,1 %)	40 (40 %)	0,232
Echocardiographic parameters:				
*LV EDD, mm,	49,1 ± 6,6	49,2 ± 6,3	49,3 ± 6,5	0,986
*LA, mm,	42,7 ± 6,3	43,5 ± 6,5	43,8 ± 6,5	0,366
*Interventricular septum, mm,	12,5 ± 2,1	12,3 ± 1,8	12,6 ± 1,9	0,618
*AVA, cm^2	0,78 ± 0,25	0,75 ± 0,16	0,80 ± 0,27	0,553
*Mean aortic gradient, mmHg	44,9 ± 13,6	45,8 ± 16,4	46,4 ± 15,3	0,732
*Moderate- severe MR, n (%)	76 (59,8 %)	48 (59,2 %)	62 (62 %)	0,779
*sPAP, mmHg	45,2 ± 13,8	49,5 ± 18,1	50,5 ± 16,7	0,030*
Laboratory parameters:				
*Hemoglobin, g/dL	13,5 ± 1,06	11,7 ± 0,49	9,9 ± 0,7	<0,001*
*MCV	87,2 ± 5,1	85,4 ± 6,9	85,0 ± 7,1	0,024*
*RDW	14,8 ± 1,5	15,6 ± 2,5	16,8 ± 3,0	<0,001*
*Creatinine, mg/dL	0,88 (0,72-10,8)	0,93 (0,77-1,11)	1,01 (0,77-1,35)	0,123
Follow-up, months	32,1 (16,7 - 54,6)	21,8 (9,6-44,4)	24,3 (9,4-50,0)	0,051

Table-2. TAVR outcomes

	Normal Hemoglobin (n=127)	Mild Anemia (n=81)	Moderate-Severe Anemia (n=100)	p value
Insertion site complications, n (%)	18 (14,1 %)	16 (19,7 %)	19 (19 %)	0,386
Stroke, n (%)	3 (2,4 %)	6 (7,4 %)	8 (8 %)	0,095
Permanent pacemaker implantation, n (%)	33 (26 %)	18 (22,2 %)	17 (17 %)	0,341
Contrast nephropathy, n (%)	13 (10,2 %)	11 (13,6 %)	23 (23 %)	0,029*
In hospital mortality, n (%)	2 (1,6 %)	4 (4,9 %)	9 (9 %)	0,031*
All cause mortality, n (%)	22 (17,3 %)	28 (34,6 %)	52 (52 %)	<0,001*

Table 1, Table 2.

Interventional cardiology / Valve and structural heart diseases

OP-018

The impact of moderate to severe mitral regurgitation on mortality in patients undergoing transcatheter aortic valve implantation

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Background and Aim: Transcatheter aortic valve implantation (TAVI) is an effective treatment modality in severe aortic stenosis. However, concomitant heart valve diseases are still important clinical conditions in this population. In this study, our aim was to investigate the impact of moderate to severe mitral regurgitation (MR) in patients undergoing TAVI.

Methods: A total of 243 consecutive patients undergoing TAVI were evaluated in this study. Patients were divided into two groups as group 1 with none to mild MR (122 patients) and group 2 with moderate to severe MR (121 patients). Baseline clinical and demographic variables were compared in both groups besides 30-day and long-term mortality.

Results: The mean age of the whole group was 78±7.8 years and 156 patients (64.2%) were female. The mean follow-up time was 33±28.2 months. Long-term mortality was significantly higher in group 2 [32 (26.2); 47 (38.8), p=0.036] compared to group 1. Additionally, creatinine level [OR: 1.478, 95%CI: 1.002-2.182, p=0.049] and moderate to high MR [OR: 1.770, 95%CI: 1.008-3.109, p=0.047] were found to be independent predictors of long-term mortality.

Conclusions: Moderate to severe MR had worse prognostic value on long-term mortality in patients with severe aortic stenosis who underwent TAVI.

Table 1. Baseline clinical and demographic variables of study population

	None to mild MR (n=122)	Moderate to severe MR (n=121)	*P*-value
Age (years)	77±8	80±7	0.003
Gender (Female), n (%)	80 (65.6)	76 (62.8)	0.653
CAD, n (%)	84 (68.9)	76 (62.8)	0.321
COPD, n (%)	70 (57.4)	69 (57.0)	0.956
DM, n (%)	57 (46.7)	50 (41.3)	0.397
CRF, n (%)	36 (29.5)	33 (27.3)	0.699
HT, n (%)	91 (74.6)	80 (66.1)	0.148
CABG, n (%)	29 (23.8)	26 (21.5)	0.671
PVD, n (%)	39 (32.0)	41 (33.9)	0.751
CVD, n (%)	4 (3.3)	2 (1.7)	0.346
AF, n (%)	22 (18.0)	16 (13.2)	0.302
Creatinine (mg/dL)	0.95 (0.8-1.2)	1.0 (0.8-1.2)	0.463
Hemoglobine (g/dL)	11±2	11±2	0.554
Maximum gradient (mm Hg)	76 (68-92)	81.5 (68-93)	0.426
Mean gradient (mm Hg)	48 (43-58)	51 (42-57)	0.844
AVA (cm^2)	0.7 (0.6-0.9)	0.75 (0.6-0.8)	0.865
EF (%)	60 (50-60)	60 (45-60)	0.123
Heart rate (bpm)	75 (67.5-82)	75.5 (65-85)	0.893
STS score	10.3 (8.5-11.8)	10.3 (9.0-15.3)	0.237

AF: atrial fibrillation, AVA: aortic valve area, bpm: beat per minute, CABG: coronary artery bypass grafting, CAD: coronary artery disease, COPD: chronic obstructive pulmonary disease, CRF: chronic renal failure, CVD: cerebrovascular disease, DM: diabetes mellitus, EF: ejection fraction, HT: hypertension, MR: mitral regurgitation, PVD: peripheral vascular disease, STS: the society of thoracic surgeons

Table 2. Echocardiographical and clinical evaluation of the whole group

	None to mild MR (n=122)	Moderate to severe MR (n=121)	*P*-value
Valve type, n (%)			
Balloon exp	87 (71.3)	83 (68.6)	
Self exp	25 (20.5)	25 (20.7)	
Mechanically exp	10 (8.2)	13 (10.7)	0.786
Valve size, n (%)			
23 mm	31 (25.4)	33 (27.3)	
25 mm	7 (5.7)	10 (8.3)	
26 mm	36 (29.5)	40 (33.1)	
27 mm	13 (10.7)	9 (7.4)	
29 mm	35 (28.7)	28 (23.1)	
34 mm	0 (0)	1 (0.8)	0.653
Second valve implantation, n (%)	3 (5.8)	0 (0)	0.160
Postoperative AR, n (%)			
none	97 (79.5)	90 (74.4)	
mild		22 (18.0)	25 (20.7)
moderate	3 (1.2)	6 (5.0)	0.484
Coronary occlusion, n (%)	0 (0)	2 (1.7)	0.247
Stroke, n (%)	4 (3.3)	1 (0.8)	0.187
Major vascular complication, n (%)	10 (8.2)	6 (5.0)	0.309
Minor vascular complication, n (%)	13 (10.7)	10 (8.3)	0.524
Tamponade, n (%)	4 (3.3)	2 (1.7)	0.346
Permanent pacemaker, n (%)	17 (13.9)	16 (13.2)	0.871
30-day mortality, n (%)	12 (9.8)	18 (14.9)	0.232
Long term mortality, n (%)	32 (26.2)	47 (38.8)	0.036
Follow-up time (months)	33±27	34±29	0.731

AR: aortic regurgitation, MR: mitral regurgitation

Table 3. Multivariate logistic regression analysis giving information about independent predictors of long-term mortality

	Multivariate analysis		
	Odds ratio	95% CI (Lower-Upper)	*P*-value
Age	1.020	0.982-1.058	0.307
DM	1.433	0.822-2.497	0.205
Moderate to severe MR	1.770	1.008-3.109	0.047
Creatinine	1.478	1.002-2.182	0.049
AF	1.021	0.477-2.187	0.956

AF: atrial fibrillation, DM: diabetes mellitus, MR: mitral regurgitation

Interventional cardiology / Carotid and peripheral vascular

OP-019

Comparison between PeRcutanEous and surgical femoral aCcess for endovascuLar aOrtic repair in patientS with typE III aortic Dissection (PRECLOSE Trial)

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Background and Aim: Aortic dissections are cardiovascular events with high mortality and morbidity rates. Management might be either with medical or interventional approach. Recently, thoracic endovascular intervention (TEVAR) becomes the first treatment of choice because of its better results and lower rates of complications in patients with type III aortic dissections. The intervention might be performed via femoral artery either with percutaneous or with surgical approach. Because of large sheath insertion to femoral artery, Pre-close technique is described in literature. The aim of this study was to investigate and compare the outcomes and safety of 'Pre-close technique' to surgical approach in patients with type III aortic dissections who underwent TEVAR with femoral access 22 F.

Methods: A total of 96 patients whom had type III aortic dissection and was performed TEVAR were retrospectively included in the study. Fifty-six patients had TEVAR with percutaneous approach and these patients are named as P-TEVAR group, and 40 patients had TEVAR with surgical approach and these patients are named as S-TEVAR group. Pre- and post-procedural data with complications and procedural data during TEVAR were evaluated for both groups and compared in between.

Results: The main finding was that there was no significant difference between S-TEVAR and P-TEVAR groups in terms of complications and technical success. Operating room time was significantly decreased in P-TEVAR group (p<0.001). Overall success rate for femoral approach in patients with Pre-close technique was 94.6% and was 100% for surgical approach. P-TEVAR group had post-operative complications in three patients and S-TEVAR group had in four patients.

Conclusions: Total percutaneous approach with Pre-close technique using Pro-Glide device is a safe and feasible method of femoral access in patients with type III aortic dissections.

Table 1. Antomical characteristics of CFA detected in computurized tomography and characteristics of sheath and device used for patients

	P-TEVAR	S-TEVAR	*P*-value
CFA diameter	10.48±1.66	9.91±2.06	0.139
sheath size diameter	23.95±1.10	23.80±0.91	0.493
22	11 (19.6%)	7 (17.5%)	0.791
24	28 (50.0%)	27 (67.5%)	0.087
25	15 (26.8%)	6 (15.0%)	0.168
26	2 (3.6%)	0 (0.0%)	0.509
sheath size diameter(mm)	7.90±0.36	7.85± 0.30	0.545
Skin to CFA distance (mm)	34.13 ±13.31	30.78±12.34	0.214
calcification grade of femoral artery	0 (0-1)	1(0-2)	0.120
Device size used for endovascular repair	38.52±5.08	36.85±5.47	0.128

Table 2. Baseline characteristics and symptoms of admission for patients

	P-TEVAR (n=56)	S-TEVAR	*P*-value
age (years)	58.7±9.9	57.2±11.8	0.506
male (n %)	45 (80.4%)	29 (72.5%)	0.367
BMI (kg/m^2)	26.4±5.9	26.8±6.1	0.468
diabetes mellitus (n %)	16 (28.5%)	6 (15.0%)	0.119
Hypertension (n %)	54 (96.4%)	36 (90.0%)	0.200
hyperlipidemia(n %)	8 (14.3%)	5 (12.5%)	0.231
smoking(n %)	29 (51.8%)	17 (42.5%)	0.369
glomerular filtration rate (ml/min)	93	88 (65-97)	0.330
coronary artery disease (n %)	14 (25.0%)	6 (15%)	0.234
peripheral artery disease (n %)	5 (8.9%)	2 (5.0%)	0.696
symptoms of admission			
persistent pain or resistant hypertension	36 (64.3%)	25 (62.5%)	0.233
malperfusion	18 (32.1%)	11 (27.5%)	
rupture	2 (3.6%)	4 (10.0%)	

Table 3. Details of reasons for pre-close failure

Number of patient	sheath (F)	Reasons for failure	Calcification	Total pro glide usage	treatment
First patient	25	persisten bleeding	grade 1	3	open repair
Second patient	25	persisten bleeding	grade 1	3	open repair
Third patient	26	persisten bleeding	grade 1	3	open repair

Table 4. Procedural data, clinical outcomes and procedural complications

	P-TEVAR	S-TEVAR	*P*-value
Operating room time (min)	104.3±13.8	160±25.4	<0.001
Hospital lenght of stay (days)	9.09±4.74	10.06±4.96	0.253
Manual compression time (min)	8.16±2.41		
Technical success	53 (94.6%)	40 (100.0%)	0.263
Complications	3 (5.4 %)	4 (10.0%)	0.446
Complications			
Groin infection	0	1 (2.5%)	0.669
Pseudoaneursym	1 (1.8%)	0	
Haematoma/seroma	2 (3.6%)	3 (7.5%)	

Interventional cardiology / Coronary

OP-020

Comparison of systemic immune-inflammation index levels in patients with isolated coronary artery ectasia versus patients with obstructive coronary artery disease and normal coronary angiogram

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Background and Aim: Coronary artery ectasia (CAE) is associated with increased risk of mortality, equivalent to that of patients with obstructive coronary artery disease (CAD). Considering the role of inflammation in the pathogenesis of CAE, we aimed to investigate whether there is an association between systemic immune-inflammation index (SII) and isolated CAE.

Methods: The study population included 510 patients of which 170 patients with isolated CAE, 170 patients with obstructive CAD and 170 patients with normal coronary angiograms (NCA). The severity of CAE was determined according to the Markis classification. Multivariate stepwise logistic regression, including covariates found to have a significant association with CAE in univariate analysis, was used to identify independent predictors of isolated CAE.

Results: Patients with isolated CAE had significantly higher SII values compared to those with obstructive CAD and NCA [median 550 IQR (404-821), median 526 IQR (383-661), and median 433 IQR (330-555), respectively, p<0.001]. In multivariate analysis, SII (OR 1.032, 95% CI 1.020-1.044, p=0.003), male gender (OR 2.083, p=0.008), eGFR (OR 0.979, p=0.016), and CRP (OR 1.105, p=0.005) were independent factors of isolated CAE. Moreover, in the Spearman correlation analyze, there was a moderate but significant positive correlation between SII and CRP (r=0.379, p<0.001).

Conclusions: Higher SII levels were independently associated with the presence of isolated CAE. This result suggests that a more severe inflammatory process may play a role in the development of this variant of CAD.

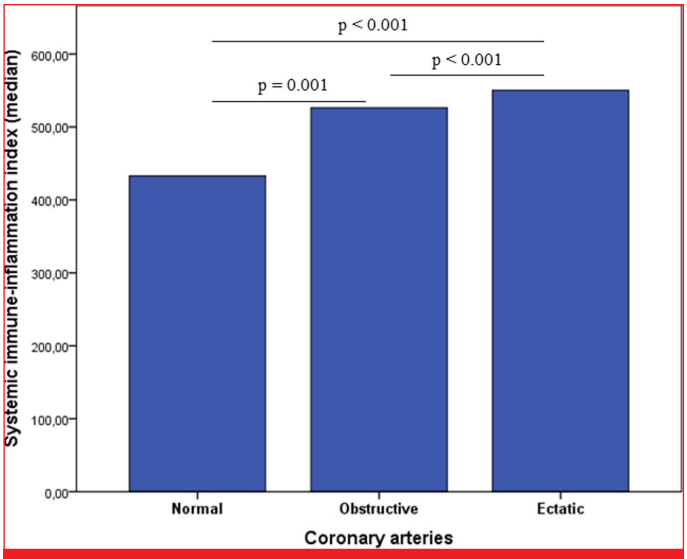

Figure 1. Systemic immune-inflammation index levels of patients with angiographically normal coronary arteries, patients with obstructive coronary artery disease, and patients with isolated coronary artery ectasia

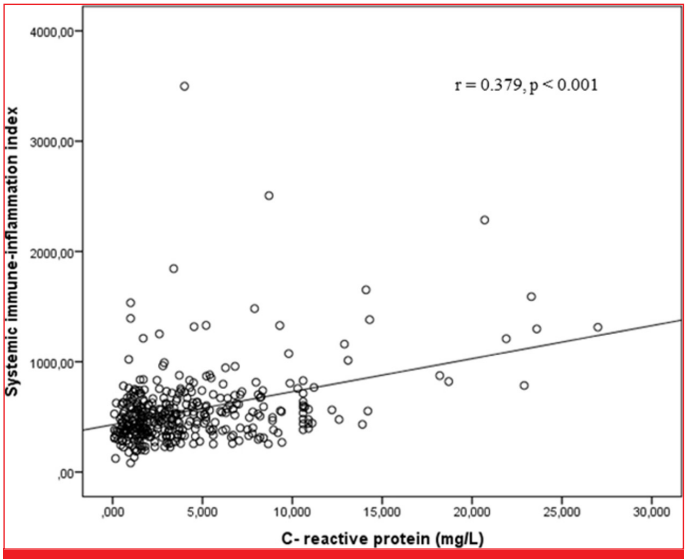

Figure 2. Correlation analyze between systemic immune-inflammation index and c-reactive protein in Spearman rank test

Table 1. Baseline clinical characteristics of the study population

Variable	Coronary Arteries			p
	Normal (n=170)	Obstructive (n=170)	Ectatic (n=170)	Value
Age (years)	58.85±11.54	63.14±10.77	61.23±11.11	0.002
Men	63 (37.1%)	104 (61.2%)	115 (67.6%)	<0.001
Body mass index (kg/m^2)	29.24±4.80	28.28±4.53	29.15±4.01	0.103
Hypertension	87 (51.2%)	104 (61.2%)	101 (59.4%)	0.138
Diabetes mellitus	44 (25.9%)	72 (42.4%)	59 (34.7%)	0.006
Active smoker	33 (19.4%)	52 (30.6%)	42 (24.7%)	0.058
Hypercholesterolemia	72 (42.4%)	73 (42.9%)	76 (44.7%)	0.901
Family history of coronary artery disease	45 (26.5%)	51 (30%)	51 (30%)	0.709
Left ventricular ejection fraction (%)	58.82±8.73	56.41±8.38	55.62±11.06	0.145
Previous medications				
Aspirin	61 (36.1%)	75 (44%)	75 (44.1%)	0.310
Angiotensin-aldosteron system blockers	74 (43.5%)	71 (41.8%)	87 (51.2%)	0.180
Beta blockers	68 (40.2%)	68 (40.2%)	84 (49.4%)	0.174
Statins	67 (39.8%)	60 (35.8%)	80 (47.6%)	0.122

Table 1. Baseline clinical characteristics of the study population

Table 2. Laboratory and angiographic characteristics of the study population

Variable	Coronary Arteries			p
	Normal (n=170)	Obstructive (n=170)	Ectatic (n=170)	Value
Creatinine (mg/dL)	0.82±0.19	0.96±0.23	0.88±0.21	<0.001
Estimated glomerular filtration rate (ml/min)	83±13	77±17	76±14	<0.001
C- reactive protein (mg/L)	2.2 (1.2-4.5)	2.76 (1.4-5.2)	3.9 (1.6-7.8)	0.001
Total cholesterol (mg/dL)	201.02±38.09	194.30±48.62	196.42±48.90	0.389
Low density lipoprotein cholesterol (mg/dL)	124.86±35.0	124.48±39.75	123.68±41.86	0.960
High density lipoprotein cholesterol (mg/dL)	48.05±11.74	40.56±9.82	43.06±10.31	<0.001
Triglyceride (mg/dL)	134 (97-196)	123 (85-172)	132 (93-180)	0.170
Hemoglobin (g/dL)	13.6±1.44	13.40±1.57	13.96±2.1	0.344
Platelet count (10^9/mL)	246.31±58.22	225.42±60.6	258.1±77.45	<0.001
Neutrophil count (10^9/mL)	3.94±1.25	4.67±1.28	4.99±1.91	<0.001
Lymphocyte count (10^9/mL)	2.29±0.85	2.05±0.67	2.05±0.82	0.006
Systemic immune-inflammation index	433 (330-555)	526 (383-661)	550 (404-821)	<0.001
Markis classification				
Type 1			23 (13.5%)	
Type 2			18 (10.6%)	
Type 3			6 (3.5%)	
Type 4			123 (72.4%)	
Distribution of ectatic coronary arteries				
Left anterior descending			132 (77.6%)	
Left circumflex			89 (52.4%)	
Right coronary artery			88 (51.8%)	

Table 2. Laboratory and angiographic characteristics of the study population

Table 3. Logistic Regression Analyzes Presenting Independent Predictors of Isolated Coronary Artery Ectasia

Variable	Univariate Analysis		Multivariate Analysis	
	Odds ratio (95% Confidence interval)	p Value	Odds ratio (95% Confidence interval)	p Value
Age	1.002 (0.986-1.019)	0.792		
Male gender	2.166 (1.473-3.184)	<0.001	2.083 (1.212-3.571)	0.008
Diabetes mellitus	1.529 (1.013-2.309)	0.043	1.539 (0.891-2.658)	0.122
Hypertension	1.141 (0.786-1.658)	0.486		
Smoking	1.016 (0.663-1.556)	0.942		
Platelet count	1.005 (1.002-1.008)	0.001	1.003 (0.999-1.008)	0.128
Neutrophil count	1.324 (1.169-1.501)	<0.001	1.041 (0.840-1.291)	0.709
Lymphocyte count	0.822 (0.645-1.048)	0.113		
C-reactive protein	1.149 (1.083-1.219)	<0.001	1.105 (1.031-1.185)	0.005
Systemic immune-inflammation index	1.003 (1.002-1.004)	<0.001	1.032 (1.020-1.044)	0.003
Creatinine	1.177 (0.508-2.724)	0.702		
Estimated glomerular filtration rate	0.969 (0.956-0.983)	<0.001	0.979 (0.962-0.996)	0.016
High density lipoprotein cholesterol	0.990 (0.973-1.007)	0.232		
Triglyceride	1.000 (0.998-1.002)	0.841		

Table 3. Logistic regression analyzes presenting independent predictors of isolated coronary artery ectasia

Interventional cardiology / Coronary

OP-021

Recurrent acute coronary syndrome, how successful are we in risk modification and guideline recommendations?

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Background and Aim: Despite the marked decline in the rate of recurrent AMI over recent decades, recurrence remains a significant threat to ACS survivors. 21% of men and women, will have a recurrent AMI or fatal CHD event within 5 years of their first AMI. The importance optimal drug therapy and modification of risk factors for the prevention of recurrent cardiovascular events are increasing. 2019 ESC dyslipidemia guidelines sugest that LDL-C goal of <40 mg/dL for patients with ASCVD who experience a second vascular event within 2 years while taking maximally tolerated statin-based therapy (class IIb). In the current study we compared risk factor of first and repetitive ACS patients.

Methods: This cross-sectional observational study enrolled consecutive adult patients (>18 years) who were diagnosed with ACS and admitted hospital between September 2019 and December 2020. Patients were categorized by the diagnosis of first or repetitive ACS. During the hospitalization period, face-to-face interviews and physical examinations were performed, and laboratory findings and CV risk factors were determined.

Results: A total of 710 patients (359 patients in first ACS and 351 patients in repetitive ACS group) were included. Mean age was 58±12 years and 83% patients were male. Table 1 lists the demographic characteristics of patients. Risk factor modification was found to be insufficient in patients with recurrent ACS (Table 1). Although a slight decrease was observed in LDL cholesterol values, it was determined that the achieved LDL cholesterol value was far behind the guideline recommendations (Fig. 2, 3). It was determined that the patients could not achieve success in lifestyle changes such as regular physical activity, weight control, quitting smoking, and controlling psychosocial stress.

Conclusions: This study showed that risk factor modification is insufficient in patients with recurrent ACS. In particular, the achieved LDL-C values are far behind the recommended targets. It is important to give more importance to risk factor modification for the prevention of recurrent events and to initiate the necessary drug treatments to achieve the goals in the guideline recommendation.

Figure 1. Comparison of LDL-C values
LDL-C: Low density lipoprotein cholesterol

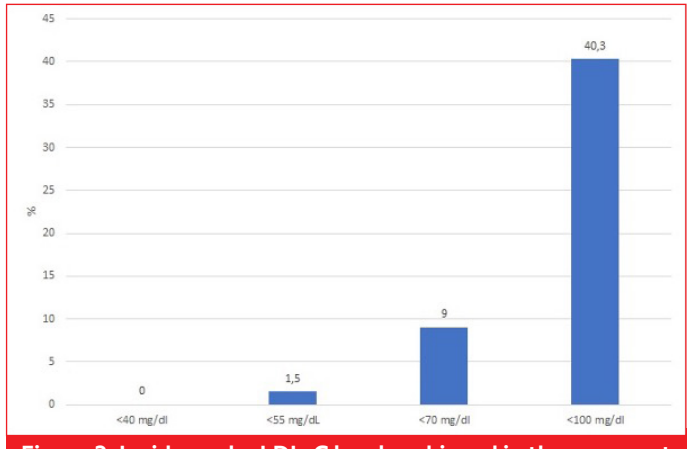

Figure 2. Incidence by LDL-C levels achieved in the recurrent ACS group
LDL-C: Low density lipoprotein cholesterol

Table 1. General characteristics

	First ACS	Recurrent ACS	*P*-value
Age (mean ± SD)	57.41±12.03	61.38±11.53	0.012
BMI (mean ± SD)	28.13±4.47	27.92±4.63	0.739
Systolic BP (mean ± SD)	137.69±25.54	139.7±28.20	0.582
Diastolic BP (mean ± SD)	83.46±15.29	84.81±19.06	0.587
Total Cholesterol (mean ± SD)	210.68±55.36	194.49±51.19	0.026
LDL Cholesterol (mean ± SD)	136.21±41.41	121.97±43.67	0.011
HDL Cholesterol (mean ± SD)	44.05±17.33	40.57±11.67	0,042
Non HDL Cholesterol (mean ± SD)	164.77±52.08	154.07±48.37	0.102
Triygliseride (mean ± SD)	169.7±158.53	182.12±191.14	0.570
VLDL (mean ± SD)	37.45±36.80	37.37±38.37	0.987
Current smoke (%)	75.8	72.5	0.553
DM (%)	27.7	36.2	0.155
HTN (%)	31.6	58.0	<0.001
Psikososyal stress (%)	75.8	80.9	0.367
Active lifestyle (%)	46.5	34.3	0.021
Regular Exercise (%)	14.0	14.5	0.781

ACS: Acute coronary syndrome, BMI: Body mass index, BP: Blood pressure, HDL: High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein, SD: Standart deviation

Interventional cardiology / Coronary

OP-022

Characteristics of the patients with atrioventricular conduction block after ST segment elevation myocardial infarction and its clinical importance

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Background and Aim: ST elevation myocardial infarction (STEMI) is a life-threatening health problem. STEMI increases morbidity and mortality rates. Thrombolytic and invasive methods are available for STEMI treatment. After invasive methods developments, complication rates had been decreased. Despite this decrease, atrial and ventricular arrhythmias after STEMI can still be encountered at substantial rates today. Atrioventricular (av) conduction may be impaired due to ischemic and metabolic causes after STEMI. The development of the atrioventricular conduction block (AVB) after STEMI increases morbidity and mortality rates. In the prior studies investigators generally investigated the HG-AVB predictors and its clinical effects. However, type-I AVB is also observed in a significant portion of patients with av conduction defects after STEMI. The differences between type-1 AVB patients, those with normal av conduction duration, and the patients with HG-AVB have not been adequately investigated. In this study we aimed to evaluate the differences between the patients who had normal av conduction intervals and the patients who had type-I AVB and HG-AVB after STEMI.

Methods: We included 600 consecutive STEMI (Anterior-STEMI, n=261, 43.5%; Inferior-STEMI n=339, 56.5%) patients who had undergone coronary angiography in our center. Patients were divided into two groups; normal sinus rhythm group (NSR group, n=464, 77.3% and AVB group, n=136, 22.7%). After these evaluations, the patients with AVB were analyzed among themselves. The patients who have AVB rhythm divided into two groups [Type-I AVB group (n=78, 13%) and High Grade AVB (HG-AVB group, n=58, 9.7%)] for this purpose. As a result of these evaluations, variables that would predict the development of HG-AVB were determined.

Results: The incidence of HG-AVB after STEMI was 9.7%. The independent predictors of AVB development were determined after regression analysis. These were; inferior-STEMI, right coronary artery (RCA) occlusion, right dominance, age >75, SBP <90 mm Hg, total cholesterol, and hemoglobine level (Table 1). There were 33 (5.5%) patients were died in hospital period during study. Mortality rates were significantly higher in HG-AVB group than others (n=12, 20.7% vs. n=21, 3.9%, p<0.001). There were 6 (1.0% of all patients, 10.4% of HG-AVB patients) permanent pacemaker (PPM) implanted to the patients and all of them were HG-AVB group (p<0.001). Diabetes mellitus, hypertension and smoking rates were similar between groups. There were some differences between Type-I and HG-AVB groups. HG-AVB predictors are

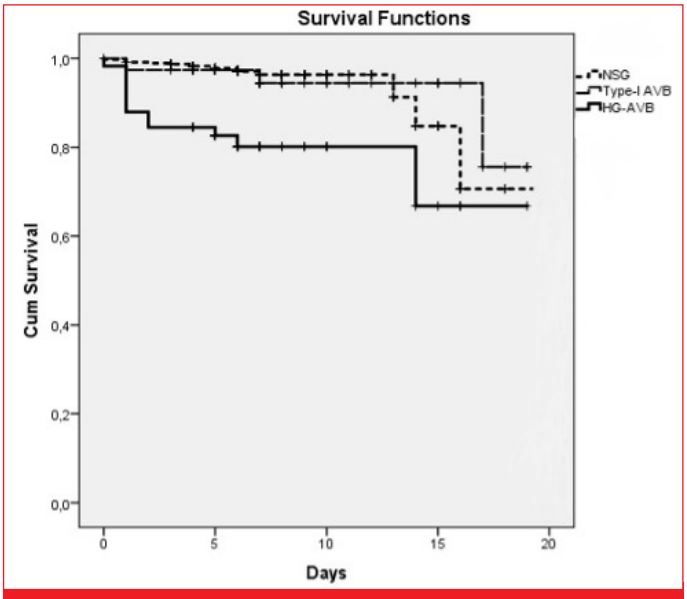

Figure 1. When the Kaplan-Meier graph was examined, it was determined that life expectancy was lower in the high-grade atrioventricular block (HG-AVB) group (log-rank p<0.0001).

RCA occlusion, presence of retrograde filling, smoking, SBP<90 mm Hg and total cholesterol level (Table 2).

Conclusions: AVB after STEMI is usually reversible. HG-AVB development after STEMI increases morbidity and mortality rates (Fig. 1). Early revascularization decreases PPM implantation rates. Despite this, a small number of patients may still need PPM insertion.

Table 1.

Variables	Univariate Odds Ratio (95%)	Univariate *P*-value	Multivariate Odds ratio (95%)	Multivariate *P*-value
Age>75 years	2.8 (0.8-5.7)	0,002	4.2 (0.1-0.5)	0.039
Inferior STEMI	43.6 (17.1-84.1)	<0.001	11.1 (4.6-20.9)	<0.001
SBP<90 mm Hg	75.8 (0.7-3.1)	<0.001	26.7 (9.7-42.5)	<0.001
Total cholesterol	15.6 (6.4-25.1)	0.080	4.4 (1.4-9.1)	0.042
Hemoglobine	9.4 (3.2-17.6)	0.002	3.8 (1.5-6.7)	0.050
Right dominance	9.0 (1.0-3.7)	0.004	4.9 (1.7-8.8)	0.027
RCA occlusion	38.9 (13.8-61.3)	<0.001	13.1 (6.2-21.8)	<0.001

Univariate and multivariate regresssion analyzes of atrioventricular conduction block development

Table 2.

Variables	Univarite Analyzes Odds ratio (95%)	Univarite Analyzes *P*-value	Multivariate Analyzes Odds ratio (95%)	Multivariate Analyzes *P*-value
SBP<90 mm Hg	12.4 (4.8-21.7)	0.001	5.5 (1.8-8.4)	0.018
Total cholesterole	8.6 (3.4-15.1)	0.004	4.1 (1.5-7.1)	0.049
Smoking	17.9 (10.1-34.5)	<0.001	5.5 (2.1-9.5)	0.023
RCA occlusion	19.6 (8.8-33.3)	<0.001	5.2 (2.2-9.8)	0.026
No retrograde filling	6.9 (2.1-9.1)	0.009	3.8 (0.9-3.7)	0.047

Univariate and multivariate regression analysis of high-grade atrioventricular block (HG-AVB) predictors. Hg-AVB predictors were found to be; SBP<90mmHg, total cholesterol, smoking, RCA occlusion and no retrograde filling.
SBP; systolic blood pressure, RCA; right coronary artery

Interventional cardiology / Coronary

OP-023

Let's not deprive the COVID-19 patient from percutaneous coronary intervention

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Background and Aim: In this study, the relationship of revascularization via percutaneous coronary intervention (PCI) with 1-month mortality in non-ST-elevation myocardial infarction (NSTEMI) patients with active COVID-19 infection, coronary angiography (CAG) and PCI application preferences of cardiologists to COVID-19 patients, and the infection rates of the personel working in the angio of the active COVID-19 patient will be investigated.

Methods: The study included 101 consecutive patients who were admitted to our clinic with the diagnosis of NSTEMI while they had active COVID-19 infection between 03/2020 and 02/2021. Patients were divided into two groups as those who were revascularized by PCI and those who were not. The angio records of the patients who underwent CAG but did not undergo PCI were re-evaluated by two cardiologists blinded to the study, and the patients who were deemed to need PCI by both cardiologists were determined. On the other hand, cardiologists, nurses and angio technicians who entered the procedure of patients who underwent CAG were identified from the hospital records and the results of those who gave the COVID-19 test within 14days after the procedure were recorded.

Results: CAG was performed in 62.4% of the patients included in the study. The rates of performing CAG were significantly lower in patients in the severe-critical COVID-19 category. Only 46.0% of the patients who underwent CAG were revascularized with PCI. The 1-month mortality rate in our study was 24.8%. In the regression analysis, revascularization with PCI was associated with 1-month mortality, independent of age and COVID-19 category, while the absence of revascularization increased mortality by approximately 5times. When 34 patients who underwent CAG and did not undergo PCI were evaluated retrospectively, 5 (14.7%) patients were found to be suitable for PCI. A total of 48 personnel took part in 63CAG operations. 13 COVID-19 tests were performed by the personnel in charge within 14 days after the procedure, but none of the tests were positive.

Conclusions: When compared to the studies in the literature in the similar period, it was seen that CAG was applied to patients at a higher rate. Despite this, most of the patients, especially in the advanced COVID-19 category, were deprived of this opportunity. In addition, although they did not have a large rate, it was observed that some of the patients who underwent CAG could be treated with PCI, but not. We think that the fear of prolonged contact time with a COVID-19 patient may play a role in this situation.

In our study, it was determined that revascularization with PCI was associated with 1-month mortality regardless of age and COVID-19 category. On the other hand, no healthcare personnel were infected due to being in the process of the COVID-19 patient. Considering these two results together, we think that the approach to COVID-19 patients diagnosed with NSTEMI should not be different from other patients, provided that necessary precautions are taken.

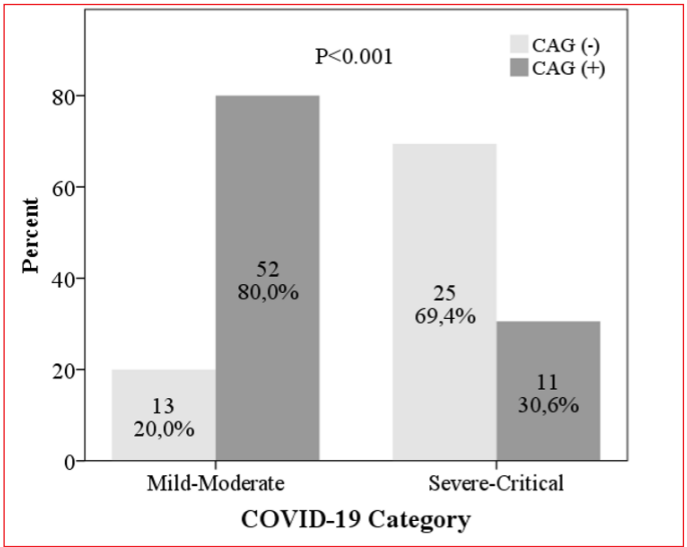

Figure 1. Rates of CAG by COVID-19 category

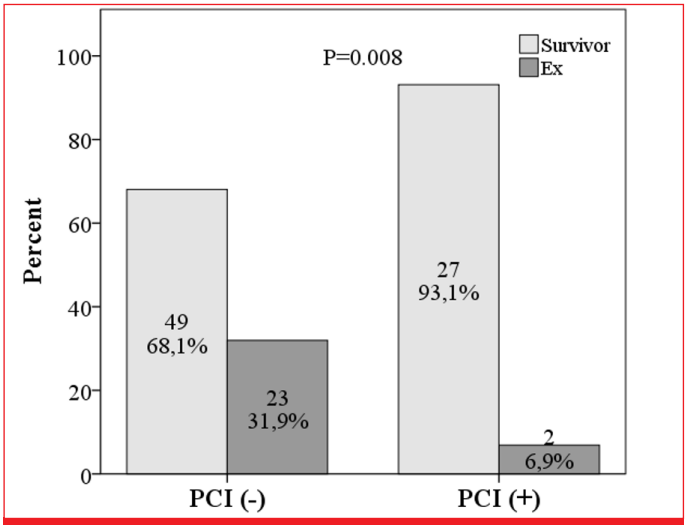

Figure 2. Mortality rates in patients with and without percutaneous coronary intervention

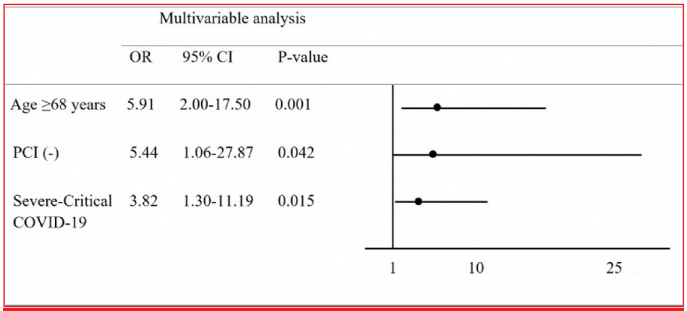

Figure 3. Multivariable logistic regression analysis for 1-month mortality

Table 1. Baseline characteristics of COVID-19 patients by presence of percutaneous coronary intervention.

Variables	Total n=101	PCI (-) n=72	PCI (+) n=29	*P*-value
Age, years	62.7±13.9	63.9±14.7	59.9±11.4	0.190
Male, n (%)	72 (71.3%)	51 (70.8%)	21 (72.4%)	0.874
Body mass index, kg/m^2	28.4±4.5	28.2±4.2	28.8±5.1	0.503
Current smoking, n (%)	18 (17.8%)	14 (19.4%)	4 (13.8%)	0.502
Hypertension, n (%)	66 (65.3%)	46 (63.9%)	20 (69.0%)	0.628
Diabetes mellitus, n (%)	34 (33.7%)	25 (34.7%)	9 (31.0%)	0.723
Coronary artery disease, n (%)	46 (45.5%)	32 (44.4%)	14 (48.3%)	0.726
Congestive heart failure, n (%)	3 (3.0%)	3 (4.2%)	0 (0.0%)	0.555
Chronic kidney disease, n (%)	4 (4.0%)	4 (5.6%)	0 (0.0%)	0.322
COPD, n (%)	2 (2.0%)	2 (2.8%)	0 (0.0%)	1.0
CVA history, n (%)	6 (5.9%)	5 (6.9%)	1 (3.4%)	0.670
LV ejection fraction, %	49.5±10.3	49.7±10.0	49.1±11.3	0.770
Severe-Critical COVID-19	36 (35.6%)	28 (38.9%)	8 (27.6%)	0.283
White Blood Cells, 10^3/uL	8.20 (5.49-11.16)	7.91 (5.25-11.56)	8.87 (7.06-10.91)	0.290
Hemoglobin, g/dL	13.1±1.9	13.0±1.9	13.4±1.8	0.377
Neutrophil, 10^3/uL	6.08 (3.96-8.51)	5.97 (3.51-9.04)	6.24 (4.82-8.32)	0.401
Lymphocyte, 10^3/uL	1.20 (0.78-2.04)	1.15 (0.78-1.71)	1.54 (0.84-2.44)	0.146
NLR	3.95 (2.67-8.62)	4.01 (2.74-8.82)	3.65 (2.63-7.71)	0.553
Glucose, mg/dL	142 (108-254)	137 (102-264)	178 (117-251)	0.344
Creatinine, mg/dL	0.93 (0.75-1.31)	0.96 (0.77-1.32)	0.93 (0.69-1.29)	0.194
ALT, U/L	25 (15-50)	25 (14-49)	28 (18-52)	0.471
AST, U/L	36 (22-59)	36 (21-59)	38 (23-58)	0.910
Lactate dehydrogenase, U/L	444 (256-658)	373 (253-646)	537 (279-1141)	0.169
C-reactive protein, mg/dL	33.0 (10.2-99.6)	40.5 (12.7-119.7)	21.0 (5.7-63.3)	0.028
Ferritin, ng/mL	489 (255-763)	406 (218-783)	551 (317-641)	0.633
D-dimer, ug/mL	1.24 (0.66-2.38)	1.24 (0.77-2.60)	1.23 (0.57-2.26)	0.883
Creatine kinase, U/L	192 (76-328)	186 (73-307)	205 (150-654)	0.361
CK-MB, ng/mL	3.47 (2.35-12.9)	3.06 (2.25-10.8)	6.05 (2.35-23.90)	0.183
Troponin T, ng/L	110 (34-375)	87 (31-309)	177 (69-555)	0.074

Data are presented as percentage, mean ± standard deviation or median (interquartile range).
PCI, percutaneous coronary intervention; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; LV, left ventricular; NLR, neutrophil lymphocyte ratio; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK-MB, creatine kinase muscle brain.

Interventional cardiology / Coronary

OP-024

The association between invasive microvascular function and CMR-derived microvascular injury indicators and left ventricular function and infarct size at 1-month after reperfused STEMI

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Background and Aim: The restoration of the coronary microcirculation in ST-segment elevation myocardial infarction (STEMI) patients remains hampered in up to 50% of the STEMI patients after successful primary percutaneous coronary intervention (PPCI). The association between the coronary microvascular function and injury indicators and functional outcome remains debated. This study aims to investigate the relation between PPCI invasive microvascular function and cardiovascular magnetic resonance (CMR)-derived microvascular injury indicators, and left ventricular (LV) function and infarct size (IS) at 1-month after STEMI.

Methods: In total, 110 patients underwent angiography for PPCI and at 1-month follow-up. Invasive assessment of coronary microcirculation physiology in the culprit artery was performed during both procedures and included coronary flow reserve (CFR) and the index of microcirculatory resistance (IMR). A ratio of ≥2 for CFR and a value of <25 U for IMR were considered normal. CMR was performed during the acute phase (2 to 7 days after PCI) and at 1-month, and provided assessment of LV function, IS and non-invasive information of microvascular injury by microvascular obstruction (MVO) and intra-myocardial hemorrhage (IMH).

Results: Over 1-month, CFR, IMR, LV function, and IS all significantly improved (p<0.001) (Fig. 1). The post-PPCI IMR (p=0.001), IMR normal (p<0.01), MVO presence, MVO size, IMH presence, IMH size (all, p<0.001) were significantly associated with LV ejection fraction at 1-month. The post-PPCI CFR normal (p=0.02), IMR (p<0.01), MVO presence, MVO size, IMH presence, IMH size (all, p<0.001) were all associated with 1-month IS. In multivariable linear regression analyses, post-PPCI IMR and MVO presence were identified as invasive and non-invasive independent markers respectively, related to both 1-month LV ejection fraction and IS.

Conclusions: In STEMI patients, CFR and IMR significantly improve one month after PPCI. Post-PPCI IMR and MVO presence are independently associated with 1-month LV function and IS.

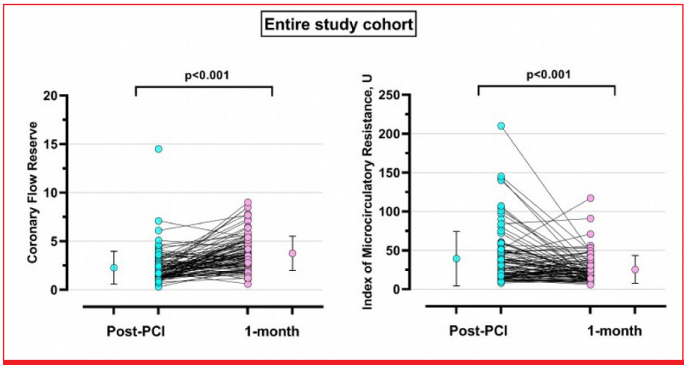

Figure 1. Temporal evolution of CFR and IMR

Interventional cardiology / Coronary

OP-025

A novel method: Use of guideliner in the treatment of no-reflow

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Background and Aim: The treatment of the No-Reflow phenomenon, which is the "nightmare" of invasive cardiologists, is not promising. Particularly in the case of TIMI0 flow, the treatment methods used to restore the distal flow are not satisfactory. Various balloon and catheter methods were tried previously for this condition. New treatment methods are needed in this respect. We presented this study which we successfully achieved distal flow by using GuideLiner in the treatment of the No-Reflow phenomenon, which is not presented in the literature.

Methods: Our study includes a total of 91 patients who applied to the cardiology clinic. Balloon angioplasty was performed in all of these cases. As soon as the No-Reflow phenomenon diagnosis was established, a GuideLiner catheter (5.5 F) was immediately advanced to the treated vessel's distal end over the same wire. Angiography was repeated with the GuideLiner in the distal vasculature (Distal angiography). According to the distal angiography images, no reflow treatment was decided. No obstructive lesion was observed in the distal angiography, we administered intracoronary nitrate and adenosine to the distal coronary vasculature through the GuideLiner. If dissection detected in distal angiography, this part of the vessel was treated with additional stent implantation. We administered an intracoronary Gp2b3a inhibitor because of an evident distal thrombus via distal angiography. Thus, a clear treatment was achieved with the distal angiography we performed through the guideliner (Fig. 1).

Results: Considering the TIMI frame currents, which is the most important data of the study, serious differences were detected between the groups. TIMI 0-1 flow was observed as 36% in the conventional group and 4% in the guidlenear group. Although TIMI II flow is seen similar in the two groups (conventional treatment group 30% vs. guideliner group 22%), TIMI III current con-

ventional treatment group was detected as 34%, while the guideliner group was found to be 74%. Considering the rates of medical treatment for flow between the two groups, a difference was found between the use of adenosine and trofiban in the two groups. While adenosine was used at a rate of 48% in the conventional treatment group, it was used at a rate of 24% in the guideliner group. The same is true for tirofiban, it was used less in the guidlinear group and it was statistically significant (conventional treatment group 58% vs. guideliner group 21%)

Conclusions: We believe that this method will provide quite satisfactory outcomes in clinical cases like No-Reflow phenomenon, whose mechanism and treatment are still unclear. With this off-label use, we obtained quite successful results. This method is quite convenient, easy, reliable and effective.

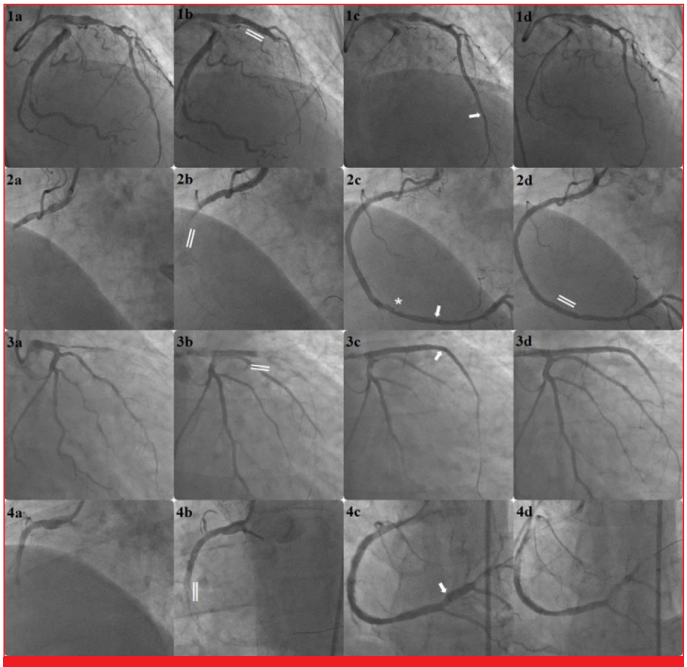

Figure 1

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-026

The relationship between oxidative stress and autophagy - apoptosis in patients with paroxysmal atrial fibrillation

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Background and Aim: Although the pathophysiology of paroxysmal AF (PAF) is not fully elucidated, oxidative stress (OS) and atrial remodeling appear to be important triggers. In our study, we aimed to investigate autophagy and apoptosis in patients with PAF and reveal whether they are related to OS.

Methods: We included 44 patients with PAF between the ages of 18-80 who applied to our clinic between February 2020 and August 2020 and 44 healthy volunteers. Serum total oxidative status (TOS), total antioxidant status (TAS), oxidative stress index (OSI), and serum ATG5 levels for autophagy and serum M30 levels for apoptosis were investigated in two groups.

Results: Both groups were similar in terms of demographic characteristics (Table 1). Serum TOS, OSI, ATG5, M30 levels, and left atrium (LA) diameter were significantly higher in the PAF group than the control group, while serum TAS levels were significantly lower (p<0.001 for all) (Table 2). While there was a significant linear correlation between OSI and ATG5 and M30 levels (p<0.001 for each), there was a significant positive correlation between LA diameter and only ATG5 level (p<0.05) (Table 3). In the stepwise multivariate logistic regression analysis to determine the independent predictors of the presence of PAF, TOS (p=0.002, OR=2.82), ATG5 (p=0.013, OR=16.97) and M30 (p=0.006, OR=13.32) were found to be independent predictors. In the linear regression analysis, the only independent predictor of LA enlargement was the ATG5 level. In the ROC analysis, the values of 2.02 U/L and above of ATG5 with 82% sensitivity and 93% specificity (area under the curve=0.880, 95% confidence interval: 0.798-0.961) and M30 values of 3.54 U/L and above values predicted the presence of PAF with 75% sensitivity and 86% specificity (area under the curve=0.854, 95% confidence interval: 0.776-0.931) (Fig. 1a, 1b).

Conclusions: Our study revealed that the increase in autophagy and apoptosis in patients with PAF was significantly correlated with OS, and ATG5 was the strongest independent predictor for PAF. Our results also revealed that ATG5 is the only independent predictor for LA enlargement.

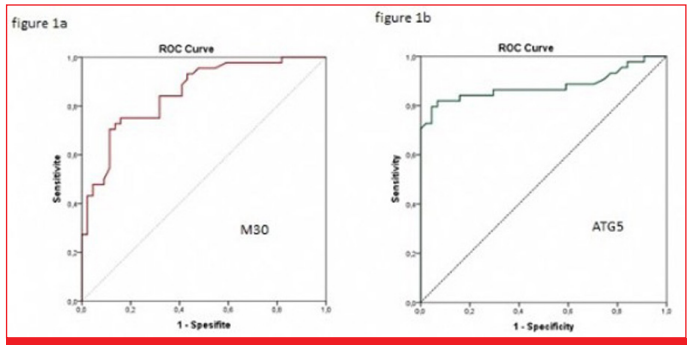

Figure 1. ATG5 and M30 ROC curve in predicting the presence of PAF

Table 1. The relationship of autophagy and apoptosis indicators with oxidative stress parameters and left atrium.

	ATG5		M30	
TAC	R=-0.434	P<0.001	R=-0.344	P=0.001
TOS	R=0.444	P<0.001	R=0.348	P=0.001
OSI	R=0.462	P<0.001	R=0.366	P<0.001
Left atrium	R=0.319	P=0.002	R=0.205	P=0.056

TAS; Total antioxidant capacity, TOS; Total oxidative stress, OSİ; Oxidative stress index, ATG5; Autophagy-associated protein, M30; Apoptosis-associated protein.

Table 2. Comparison of demographic and clinical features of the cases

	Patient (n=44)	Control (n=44)	*P*-value
Age (year)	58.0±12.6	56.8±11.5	0.628
Male, n (%)	18 (40.9)	24 (54.5)	0.200
BMI (kg/m^2)	29.0±3.2	29.6±3.2	0.384
HT, n (%)	24 (54.5)	16 (36.4)	0.087
DM, n (%)	13 (29.5)	8 (18.2)	0.211
HL, n (%)	5 (11.4)	2 (4.5)	0.237
CAD, n (%)	14 (31.8)	17 (38.6)	0.503
Cigarette, n (%)	8 (18.2)	14 (31.8)	0.140
ASA, n (%)	5 (11.4)	21 (47.7)	<0.001
OAC, n (%)	29 (65.9)	0	<0.001
Beta-bloker, n (%)	35 (79.5)	20 (45.5)	0.001
CCB, n (%)	4 (9.1)	2 (4.5)	0.676
Anti-arrhythmic, n (%)	11 (25)	0	<0.001

BMI; Body mass index, HT; Hypertension, DM; Diyabetes mellitus, HL; Hyperlipidemia, CAD; Coronary arteria disease, ASA; Acetyl salicylic acid, OAK; Oral anti coagulant, CCB; Calcium canal blocker

Table 3. Comparison of oxidative stress, autophagy and apoptosis parameters between groups.

	Patient (n=44)	Control (n=44)	*P*-value
TAC (mmol Trolox® equivalents/l)	1.04±0.16	1.35±0.17	<0.001
TOS (micmol H2O2 equivalents/l)	14.04±2.29	10.80±1.53	<0.001
OSI (arbitrary units)	0.82±0.17	1.38±0.36	<0.001
ATG5 (U/L)	2.43±0.53	1.63±0.35	<0.001
M30 (U/L)	4.06±0.90	2.85±0.87	<0.001

TAS; Total antioxidant capacity, TOS; Total oxidative stress, OSİ; Oxidative stress index, ATG5; Autophagy-associated protein, M30; Apoptosis-associated protein.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-027

Long-term Follow-up of Patients with Drug Related Atrioventricular Block without A Need of Permanent Pacemaker During Index Hospitalization: A Retrospective Study

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Background and Aim: Most of the patients hospitalized due to drug related atrioventricular (AV) block do not require permanent pacemaker implantation (PPI) since AV block regresses following cessation of the responsible drug. However, AV block requiring PPI may relapse in long-term follow-up. In this study, we retrospectively evaluated the factors predicting the need for a PPI in the long-term follow-up in patients admitted to our hospital with drug related AV block but did not require PPI in index hospitalization.

Methods: We evaluated 177 patients who had been hospitalized with drug related AV block between January 2012 and July 2020 and who had not required PPI during hospital follow up. The patients were divided into two groups according to whether PPI was performed or not. The independent predictors of long-term PPI were evaluated and the effect of glomerular filtration rate (GFR) of the patients during the index hospitalization on the long-term outcome were compared.

Results: A GFR above 60 ml/min is an independent significant risk factor in predicting long-term permanent pacemaker implantation in drug related AV blocks. It is found that the need for PPI was 2.64 times higher without adjusted and 1.9 times higher with adjusted for all covariates in patients with GFR above 60 ml/min during hospitalization compared to those with GFR below 30 ml/min.

Conclusions: Recurrence of AV block is high in the follow-up of patients whose AV conduction system has improved by discontinuing the responsible drug in drug-related AV blocks. The development of drug-related block in patients with normal GFR can be considered as an indicator of the disease in the cardiac conduction pathways.

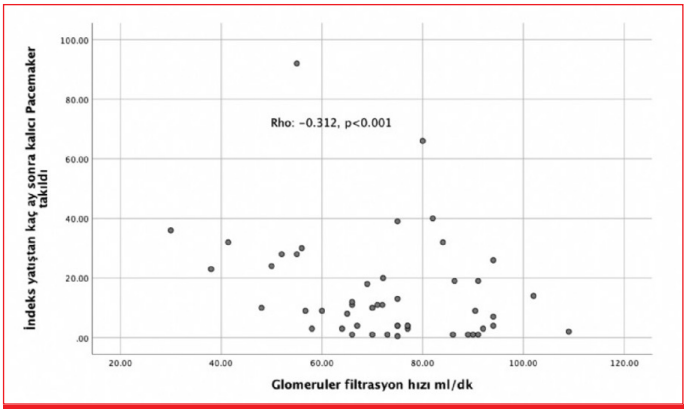

Figure 1. Correlation analysis

Table 1. Demographic characteristics of the cases

	Permanent pacemaker implantation (-) in follow-up n=128	Permanent pacemaker implantation (+) in follow-up n=49	*P*-value
Age, year	76 (66-83)	73 (65-80)	0.286
Male gender	54 (42.2%)	21 (42.9%)	0.936
Hypertension	99 (77.3%)	42 (85.7%)	0.203
Diabetes Mellitus	62 (48.4%)	21 (42.9%)	0.505
Hyperlipidemia	29 (22.7%)	12 (24.5%)	0.797
Smoking	12 (9.4%)	2 (4.1%)	0.355
Chronic renal failure	45 (35.2%)	12 (24.5%)	0.167
Congestive heart failure	18 (14.1%)	8 (16.3%)	0.706
Chronic obstructive pulmonary disease	18 (14.1%)	3 (6.1%)	0.121
Alzheimer	6 (4.7%)	1 (2.0%)	0.675
Coronary artery disease	47 (36.7%)	20 (40.8%)	0.616

Table 1. Demographic characteristics of the cases (Continued)

	Permanent pacemaker implantation (-) in follow-up n=128	Permanent pacemaker implantation (+) in follow-up n=49	*P*-value
Percutaneous coronary intervention	28 (21.9%)	7 (14.3%)	0.245
Coronary artery bypass grafting	23 (18.0%)	8 (16.3%)	0.796
Myocardial infarction	37 (28.9%)	14 (28.6%)	0.965
Cerebrovascular accident	10 (7.8%)	1 (2.0%)	0.294
Atrial fibrillation	37 (28.9%)	3 (6.1%)	<0.001
Hospitalization period, days	7 (5-10)	9 (6-12)	0.186
Follow-up	33 (17-61)	52 (26-89)	
Time to permanent pacemaker implantation, months		10 (3-23)	

Table 2. Classification of cases according to their ECG characteristics

	Permanent PM implantation in the follow up (-) n=128	Permanent PM implantation in the follow up (+) n=49	*P*-value
Second degree AV block	3 (2.3%)	1 (2.0%)	1.000
2:1 AV block	27 (21.1%)	15 (30.6%)	0.190
Third degree AV block	72 (56.3%)	31 (63.3%)	0.395
Atrial fibrillation and bradyarrhythmia	26 (20.3%)	6 (12.2%)	0.198

Table 3. Drug use characteristics of the cases

	Permanent PM implantation in the follow up (-) n=128	Permanent PM implantation in the follow up (-) n=128	*P*-value
1 drug risk factor	94 (73.4%)	44 (89.8%)	0.013
>1 drug risk factor	34 (26.6%)	5 (10.2%)	0.013
Calcium channel blocker	32 (25.0%)	5 (10.2%)	0.022
Digoxin	8 (6.3%)	1 (2%)	0.448
Beta blocker	103 (80.5%)	45 (91.8%)	0.053
Amiodarone	10 (7.8%)	0 (0.0%)	0.064

Table 4. Laboratory and transthoracic echocardiography values of the patients at hospitalization

	Permanent PM implantation in the follow up (-) n=128	Permanent PM implantation in the follow up (-) n=128	*P*-value
Hemoglobin (g/dL)	11.6 (10.0-13.3)	11.9 (11.0-13.5)	0.120
White blood cells (cell/μL)	9.7 (7.7-12.0)	8.4 (7.3-10.7)	0.120
Lymphocyte (cell/μL)	1.7 (1.2-2.4)	2.0 (1.3-2.5)	0.146
Creatinine (mgl/dL)	1.3 (0.9-1.9)	0.9 (0.7-1.4)	0.001
Urea (mg/dL)	28.0 (19.0-40.0)	23.0 (17.0-31.0)	0.059
Glomerular filtration rate (ml/min)	44.0 (28.0-75.0)	69.0 (41.0-89.0)	0.002
Glomerular filtration rate <30 ml/min	38 (29.7%)	6 (12.2%)	0.012
Glomerular filtration rate 30-60 ml/min	46 (35.9%)	12 (24.5%)	0.140
Glomerular filtration rate >60 ml/min	44 (34.4%)	31 (63.3%)	0.001
TSH (mIU/L)	1.3 (0.8-2.2)	1.4 (0.8-2.0)	0.496
T3 (pg/mL)	2.2 (1.8-2.6)	2.4 (2.0-2.7)	0.142
T4 (ng/dL)	1.0 (0.9-1.2)	1.0 (0.9-1.2)	0.851
Troponin I first admission (ng/mL)	0.01 (0.01-0.04)	0.02 (0.01-0.03)	0.584
Troponin I peak value (ng/mL)	0.04 (0.01-0.21)	0.03 (0.01-0.09)	0.072
Glucose (mg/dL)	129 (103-199)	123 (105-207)	0.880
ALT (IU/L)	24 (15-45)	24 (14-51)	0.846
AST (IU/L)	27 (19-55)	25 (17-39)	0.222
Sodium (mEq/L)	139 (135-141)	140 (136-141)	0.260
Calcium (mg/dL)	9.0 (8.5-9.5)	9.1 (8.6-9.5)	0.445
Potassium (mEq/L)	4.9 (4.3-5.6)	4.4 (4.2-5.1)	0.037
Left ventricle ejection fraction, %	60 (50-60)	60 (50-60)	0.296
LVEDD, mm	48 (45-52)	45 (42-54)	0.349
LVESD, mm	30 (26-35)	29 (26-34)	0.885
Left atrium anteroposterior diameter, mm	41 (34-45)	38 (36-42)	0.804

Continuous variables are presented as median (interquartile range).
Nominal variables presented as frequency (%).
LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter

Table 5. Univariable analysis and multivariable model for long-term permanent pacemaker implantation prediction according to admission demographic and clinical characteristics, laboratory parameters and echocardiography variables

Univariable analysis	*P*-value	HR (95% CI)	Multi-variable analysis	*P*-value	HR (95% CI)
Atrial fibrillation	0.039	0.355 (0.141-0.897)			
>1 drug usage	0.009	0.210 (0.065-0.674)	>1 drug usage	0.044	0.453 (0.210-0.978)
CCB	0.023	0.342 (0.135-0.862)	CCB	0.038	0.333 (0.118-0.943)
Creatinine	0.026	0.598 (0.380-0.939)			
Potassium	0.036	0.711 (0.517-0.978)			
GFR> 60	0.001	2.680 (1.497-4.798)	GFR> 60	0.021	2.780 (1.164-6.641)

All clinically relevant parameters were included in the model.
HR, Hazard ratio; CI, confidence interval.

Table 6. Cox proportional analysis and logistic regression models for permanent pacemaker implantation by glomerular filtration rate

	Glomerular filtration rate <30 ml/min (n=44)	Glomerular filtration rate 30-60 ml/min (n=58)	Glomerular filtration rate >60 ml/min (n=75)
Permanent pacemaker implantation			
Number of patients	6	12	31
Case rate, %	13.6	20.7	41.3
Permanent pacemaker implantation HR (95% CI) Model 1: unadjusted	1 [Referans]	1.65 (0.62-4.40)	3.64 (1.51-8.74)
Model 2: adjusted for medications	1 [Referans]	1.53 (0.51-6.52)	3.32 (1.35-9.38)
Model 2: adjusted for all covariates(a)	1 [Referans]	1.22 (0.48-7.24)	2.90 (1.28-10.24)

(a)Adjusted for; age, gender, hypertension, diabetes mellitus, smoking, hyperlipidemia, chronic obstructive pulmonary disease, coronary artery disease, atrial fibrillation, chronic renal failure, medications, first measurement during hospitalization of the following laboratory values (creatinine, urea, peak troponin, potassium) and left ventricle ejection fraction
CI, confidence interval; HR, hazard ratio

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-028

Comparison of transesophageal echocardiography and positron emission tomography lead endocarditis in patients with implantable electronic cardiac device and battery pocket infection

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Background and Aim: Modified Duke criteria and Transesophageal Echocardiography (TEE) are often insufficient to diagnose lead endocarditis in patients with implantable electronic cardiac devices. 18-FDG PET / CT seems to be a encouraging method for the detection of lead endocarditis. We aimed to compare the diagnostic performance of 18-FDG PET / CT and TEE in detecting lead endocarditis.

Methods: The dermographic and characteristic features of 40 patients admitted to our hospital between January 2014 and December 2018 who were hospitalized with the diagnosis of battery pocket infection were evaluated. 18F-FDG PET / CT results, TEE records, treatment methods applied to patients and clinical status of the patients at follow-up were recorded from the database of hospital. All patients had TEE and 18 FDG-PET / CT reports. According to TEE and 18 FDG PET / CT results, patients were classified as 'Lead endocarditis (LE) positive' and 'LE negative'. After at least 3 months of follow-up, the patient's lead cultures, tissue and blood cultures, clinical responses after antibiotic treatment were reviewed by using the modified DUKE criteria. The exact diagnosis was compared with 18FDG-PET / CT and TEE results.

Results: No involvement was observed on 18-FDG PET / CT in 12 patients (30%). While in the remaining 25%of the patients had involvement in the battery pocket, only 2 patients had systemic involvement. In the follow-up of 23 patients diagnosed with lead endocarditis by TOE procedure, 14 were found to be compatible with lead endocarditis. Seventeen of 18 patients who had suspicion of lead endocarditis were diagnosed with definite lead endocarditis by PET/CT (p=0.125). Six of the 22 patients with negative PET / CT scans were false negative and diagnosed as definite IE. PET/CT had a sensitivity of 73.9% and a specificity of 94.1%.

Conclusions: 18-FDG PET/CT is superior to transesophageal echocardiography in diagnosing of IE in suspected patients with implantable electronic cardiac devices.

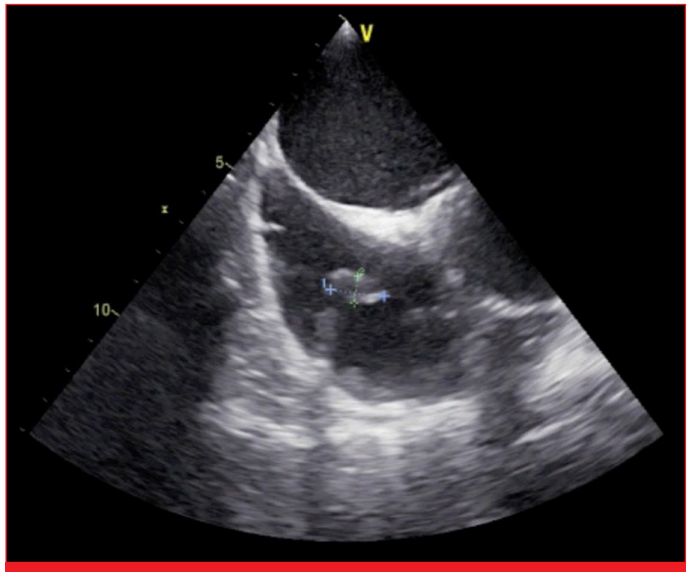

Figure 1. Fixed, immobile mass. Fixed, immobile mass wrapped around the lead on transesophageal echocardiography

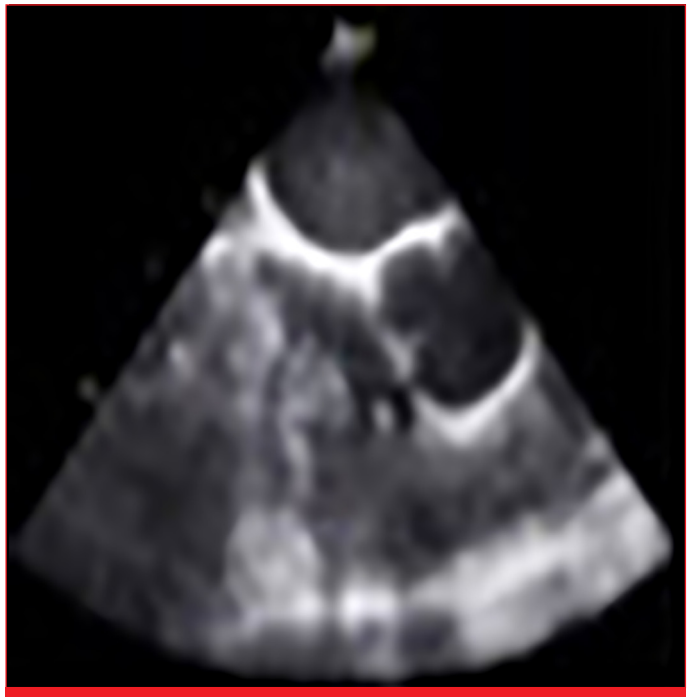

Figure 2. Mobile mass. Mobile mass around the lead on transesophageal echocardiography

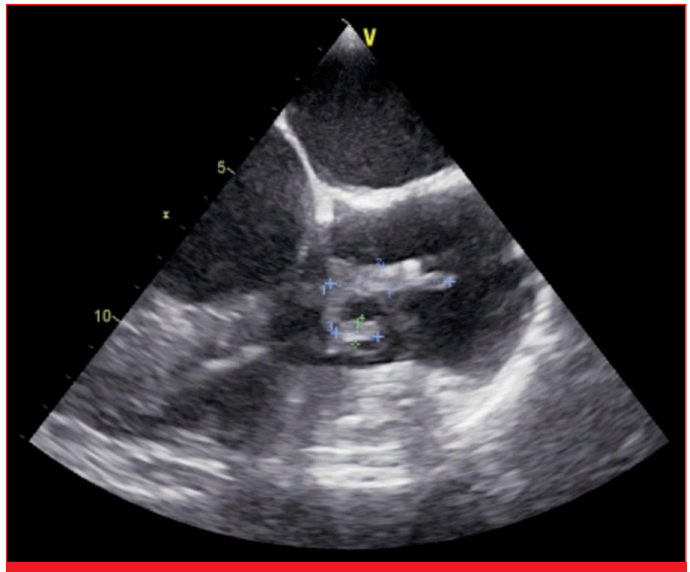

Figure 3. Multiple masses. Multiple masses around the lead on transesophageal echocardiography

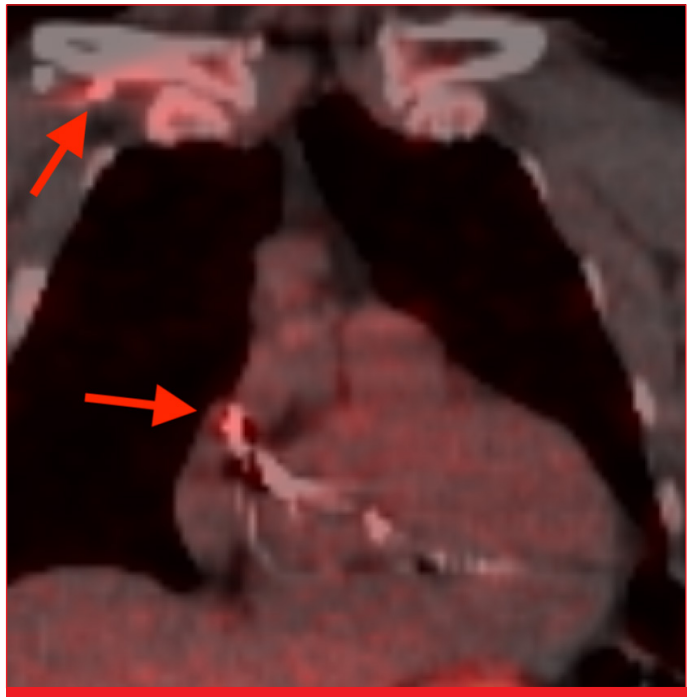

Figure 4. Positive PET/CT. Positive PET/CT examination showing the involvement of the leads with 18-FDG

		Değer
Cinsiyet n (%)	Erkek	26 (65)
	Kadın	14 (35)
Yaş (yıl) ort. ± SS		61,5±13,5
Cihaz n (%)	Tek Boşluk ICD	12 (30)
	Tek Boşluk PM	6 (15)
	Dual Boşluk PM	6 (15)
	BV-ICD	12 (30)
	DDD-ICD	4 (10)
Kan kültürü pozitifliği n (%)	Candida	1 (6,6)
	Enterococcuc Faecalis	1 (6,6)
	MRKoNS	4 (26,6)
	Polimikrobiyal	1 (6,6)
	MSSA	4 (26,6)
	Streptokokus	2 (13,3)
	VRE	2 (13,3)
TEE pozitifliği (n=23)	Mobil Kitle	12 (42,1)
	İmmobil Kitle	4 (17,3)
	Multiple Kitle	7 (30,4)
Doku Üreme poztifliği (n=9)	Candida	1 (11,1)
	MRKoNS	1 (11,1)
	Polimikrobiyal	1 (11,1)
	MSSA	4 (44,4)
	Streptokokus	1 (11,1)
	VRE	1 (11,1)
Lead Kültürü n (%)	Negatif	10 (25)
	Pozitif	2 (5)
	Yok	28 (70)
Başlangıç Modifiye Duke n (%)	Lead Endokarditi Değil	2 (5)
	Olası Lead Endokarditi	21 (52,5)
	Kesin Lead Endokarditi	17 (42,5)
Takip n (%)	Ölüm	9 (22,5)
	Tekrarlayan Enfeksiyon	3 (7,5)
	Tamamen İyileşme	28 (70)
Tedavi n (%)	Medikal Tedavi	12 (30)
	Transvenöz Lead Ekstrasyonu	19 (47,5)
	Cerrahi Tedavi	3 (7,5)
	Cep Revizyonu	6 (15)
PET	Tutulum yok	12 (30)
	Pil Cebi Tutulum var	10 (25)
	Lead ve Pil Cebi tutulum var	16 (40)
	Sistemik Tutulum	2 (5)

Table 1. Main Characteristics of the Working Group
Main Characteristics of the Working Group

			Kesin Tanı		
			Lead endokarditi (-) %(n)	Lead endokarditi (+) %(n)	p değeri
Ön Tanı	TOE	Yok	47,1 (8)	52,9 (9)	1,000*
		Var	39,1 (9)	60,9 (14)	
	PET/BT	Yok	72,7 (16)	27,3 (6)	0,125*
		Var	5,6 (1)	94,4 (17)	

Table 2. Comparison of the diagnostic performance of positron emission tomography and transesophageal echocardiography according to the last diagnosis made at follow-up

	TOE (%)	PET/BT (%)
Sensitivite Değeri	60,8	73,9
Spesifisite Değeri	47,0	94,1
Pozitif Prediktif Değer	60,9	94,4
Negatif Prediktif Değer	47,0	72,8

Table 3. Sensitivity and specificity of positron emission tomography and transesophageal echocardiography in detecting lead infection in patients with suspected lead endocarditis

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-029

The relationship of idiopathic non-sustained ventricular tachycardia with left ventricular strain in patients with preserved ejection fraction

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Background and Aim: Idiopathic non-sustained ventricular tachycardias (NSVT) refers to arrhythmias occurring in patients without structural heart disease, metabolic/electrolyte abnormalities, or channelopathy syndromes. NSVTs are generally thought to be benign, but their effects on mortality are still unclear. In addition, the effects of idiopathic NSVTs on cardiac function in patients with preserved EF are unknown. In our study, we aimed to investigate the effects of idiopathic NSVTs on left ventricular global longitudinal strain.

Methods: In our study, patients with NSVT detected in 24-hour rhythm monitoring in our center between 2019-2021 were analyzed. As a result of the evaluations (electrocardiography, echocardiography, and cardiac MRI), 54 patients with no signs of structural heart disease or channelopathy were included in the study, considering idiopathic NSVT. In addition, 43 healthy people with similar demographic characteristics were included in the study as a control group. Transthoracic echocardiography of all participants included in the study was performed and left ventricular functions were evaluated. In addition, left ventricular global longitudinal strains were evaluated using speckle tracking imaging method.

Results: The mean age of our study was 52.32±11.7 years. 38.1% of the participants in our study were female. In our study, no significant difference was found between the groups in terms of demographic characteristics (Table 1). In our study, no significant difference was found between the groups in terms of LVd, RVd and EF (p: 0.401; p: 0.074; p: 315; respectively). Left atrium(LA) diameter was significantly higher in the NSVT group (p:0.006) and Left ventricular (LV) global longitudinal strain(GLS) was significantly lower in the NSVT group (p<0.001)

Conclusions: In our study, LV GLS values of patients diagnosed with idiopathic NSVT were found to be significantly lower. This situation shows that idiopathic NSVT disrupts strain values even if the EF value is normal. It is thought that LV GLS measurements can guide the follow-up and treatment plans of these patients.

Table 1. Baseline demographic characteristics and echocardiography parameters of study groups

	NSVT Group (n=54)	Control Group (n=43)	*P*-value
Age (years)	49.8±11.8	55.4±11.5	0.400
Female n, %	17 (32.1%)	19 (44.2%)	0.223
Haemoglobin (mg/dL)	13.8±1.5	13.9±1.5	0.216
Leukocytes (/mm^3)	6.8±1.4	7.1±1.6	0.424
Creatinine (mg/dL)	0.78±0.2	0.81±0.2	0.355
Na (mg/dL)	142.6±2.2	141.2±2.4	0.146
K (mg/dL)	4.6±0.2	4.5±0.2	0.188
TSH (mIU/mL)	1.97±1.1	2.01±1.2	0.116
LDL cholesterol (mg/dL)	122.9±34.2	124.2±32.4	0.253
LA (mm)	36.7±4.2	34.4±3.6	0.006
LVd (mm)	46.9±3.7	47.6±4.6	0.401
RVd (mm)	22.1±1.9	21.3±1.5	0.074
EF (mm)	59.3±2.6	58.9±2.8	0.315
GLS	17.8±2.1	21.7±1.5	<0.001

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-030

The comparison of scoring systems in predicting bleeding in patients using novel oral anticoagulants

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[2]*İstanbul University Haseki Institute of Cardiology, İstanbul*

Background and Aim: Despite advances in the diagnosis and treatment of AF, it remains one of the leading causes of cardiovascular mortality, heart failure, stroke, and sudden death. Anticoagulant agents used for treatment significantly decrease stroke and stroke-related mortality in atrial fibrillation patients and increase the risk of bleeding. In our study, we aimed to examine the relationship between the new and old risk scores of ATRIA, HEMORR2HAGES and ORBIT bleeding scoring systems with bleeding in patients using NOAC.

Methods: 447 patients who were initiated with a new generation oral anticoagulant (dabigatran, rivaroxaban, apixaban or edoxaban) due to atrial fibrillation between June 2011 and June 2019 were included in our study. The records of the patients were analyzed retrospectively through the hospital information system and archive records. ATRIA, HEMORR2HAGES and ORBIT scores of the all patients were calculated. The primary endpoint of the study was defined as major and minor bleeding. The relationship of bleeding scores with these endpoints was examined.

Results: 447 patients were included in our study. While 37.8% (n=169) of the patients were male, 62.2% (n=278) were women. The age of the patients included in the study was found to be 75.6±10.1 years. 31.3% (n=140) of the patients included in the study were using apixaban, 57% (n=255) rivaroxaban, 10.7% (n=48) dabigatran and 0.9% (n=4) edoxaban. When the patients included in the study were evaluated according to the scoring systems; According to the HEMORR2HAGES scoring system, 28.6% (n=128), 25.3% (n=113) according to the ATRIA scoring system, and 21.5% (n=96) according to the ORBIT scoring system were in the high risk group. Major bleeding was detected in 30 patients and minor bleeding in 66 patients. Although the predictive value of the HEMORR2HAGES scoring system compared to the ATRIA scoring system was close to the statistical significance limit (p=0.058), no statistically significant difference was found between the predictive values of all three tests. The difference between the areas under the curve when comparing ORBIT with HEMORR2HAGES is 0.0268, CI: 0.0112 - 0.0649, p=0.167; When ORBIT and ATRIA are compared, the difference between the areas under the curve is 0.0320, CI: 0.0132 - 0.0772, p=0.165; When HEMORR2HAGES and ATRIA were compared, the difference between areas under the curve was found as 0.0588, CI: 0.00209 - 0.120, p=0.058.

Conclusions: We found that 3 bleeding risk scores (HEMORR2HAGES, ORBIT, and ATRIA) had similar, albeit moderate, performance in predicting major bleeding and the HEMORR2HAGES score and ORBIT score to be independent predictors for detecting major and minor bleeding. The optimal bleeding score remains a controversial issue.

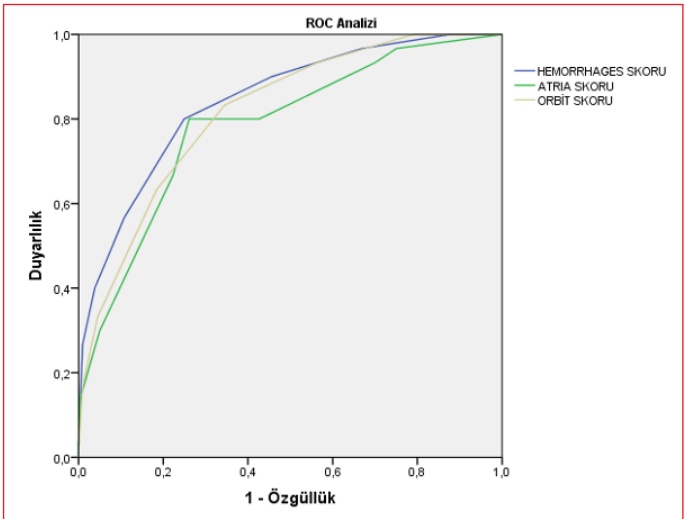

Figure 1. ROC analysis of bleeding scoring systems

Table 1. Evaluation of demographic data in patients with major bleeding

	Major bleeding (+) (n=30, %)	Major bleeding (-) (n=417, %)	*P*-value
Male sex, n (%)	16 (53.3)	153 (36.7)	0.069
DM, n (%)	10 (33.3)	146 (35.0)	0.852
HT, n (%)	24 (80.0)	273 (65.5)	0.103
Ischemic heart disease, n (%)	19 (63.3)	157 (37.6)	0.005
CABG, n (%)	10 (33.3)	52 (12.5)	0.001
ASA+clopidogrel, n (%)	0 (0.0)	7 (1.7)	0.474
ASA, n (%)	5 (16.7)	25 (6.0)	0.024
Clopidogrel, n (%)	4 (13.3)	23 (5.5)	0.083
PLT<150.000, n (%)	3 (10.0)	20 (4.8)	0.213
Heart failure (EF<40%), n (%)	7 (23.3)	55 (13.2)	0.121
Liver disease, n (%)	1 (3.3)	4 (1.0)	0.232
GFR < 60 ml/dk/1.73 m^2, n (%)	17 (56.7)	144 (34.5)	0.015
GFR < 30 ml/dk/1.73 m^2, n (%)	1 (3.3)	3 (0.7)	0.142
Alcohol abuse, n (%)	1 (3.3)	32 (7.7)	0.380
Malignancy, n (%)	1 (3.3)	18 (4.3)	0.797
Anemia, n (%)	21 (70.0)	151 (36.2)	0.000
Stroke, n (%)	11 (36.7)	52 (12.5)	0.000
Bleeding history, n (%)	13 (43.3)	33 (7.9)	0.000
Sistolic blood pressure >160 mm Hg, n (%)	12 (40.0)	53 (12.7)	0.000
Age >75, n (%)	25 (83.3)	231 (55.4)	0.003

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-031

Preliminary study of a novel wireless single lead ECG device

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Background and Aim: Though rhythm monitoring and screening are getting more important, accessibility to an ECG device is still difficult. However, most of the population currently have an easy access to digital blood pressure measuring devices. Our team developed a novel, easy to build (ie, everybody could easily make it by following some instructions and all parts are widely used easy to find circuits), low cost (less than 30$) open source (no patent payment) and wireless (bluetooth connection to an android tablet or phone by a free dedicated application) single lead ECG device (Fig. 1). Recoding capabilities of ECG signals of the novel ECG device were tested in this preliminary study.

Methods: Fifty healthy individuals were included in the study. All of them had novel ECG device recordings from four different lead placement configurations (Position 1-4) and standard 12-lead ECG consecutively (Fig. 2 and 3). All recording were evaluated for visible P, QRS, and T waves and difficulties about rhythm determination or PR, QRS, QT interval measurements. Recordings were classified for recording quality as poor (disability of single interval measurement), acceptable, and good (any difficulties at measurement and no artefact). Interval measurements (PR-QRS-QT) from P1 and P4 recordings were also compared with lead-D1 recordings of standard ECG with Bland-Altman graphs. All recordings were evaluated by two cardiologists in a blinded fashion. Intra and interobserver variabilities of novel device recordings were also tested. Study was approved by Local Ethical Board.

Results: Both sexes were equally distributed in the study. Mean age was 36±8 years, body mass index was 27±5 kg/m^2. All patients were in sinus rhythm (detected by standard ECG device). Sinus rhythm was detected successfully in all patients in all different lead positions by the new device. Also all PR, QRS, and QT intervals were easily measured in all patient s from different lead positions with novel device. However, some difficulties were reported for measurements in some lead positions (Table 1). For recordings 88% of the P1, 96% of the P2, 84% of the P3 and 84% of the P4 were in good quality and rest of them were acceptable. Intra and interobserver variability analyses showed good correlation (all with a >0.85 ICC coefficient). Though, the study was not designed for comparison of the novel device with standard ECG for diagnostic capabilities, PR, QRS, and QT interval measurements were comparable. Both methods showed good agreement with measured intervals in Bland-Altman graphs (Fig. 4).

Conclusions: Present study showed that novel single-lead ECG device has good ECG signal recording capability in 50 healthy individuals and good agreement with standard ECG device for PR, QRS, and QT-interval measurements.

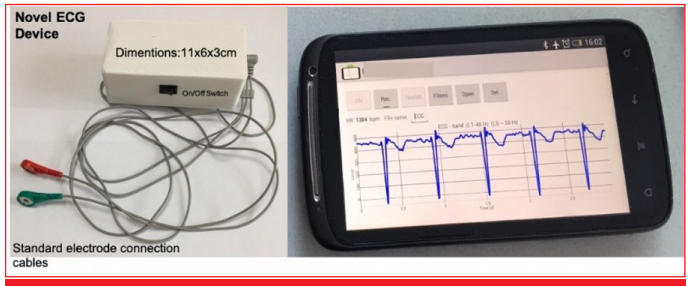

Figure 1. Novel ECG device

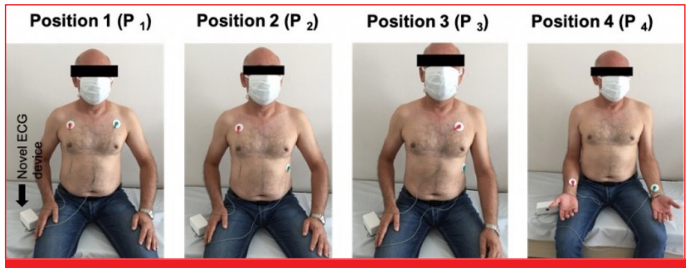

Figure 2. Electrode positions of novel ECG device

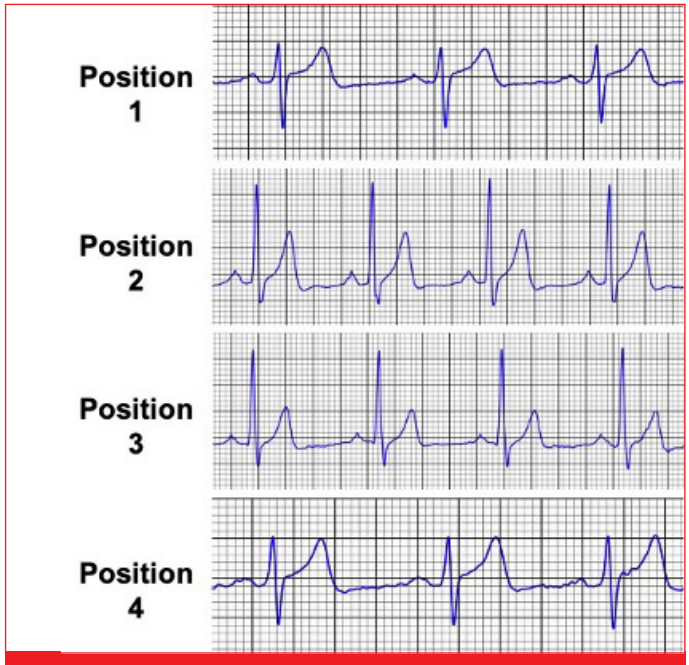

Figure 3. ECG recordings of novel device from different electrode positions

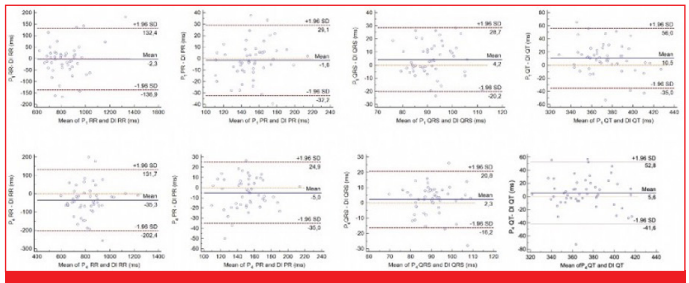

Figure 4. Bland altman graphs for novel ECG device P1 and P4 vs DI of standard ECG device

Table 1. Results of the interval measurements and novel device's ECG recordings quality assessments

	Position 1	Position 2	Position 3	Position 4	D I lead
Difficulties at Rhythm Decision (%)	0	0	0	0	Not evaluated
RR interval (ms)±SD	830± 144	834± 133	843± 139	797± 134	832±127
Difficulties at RR interval Measurement (%)	0	0	0	0	Not evaluated
PR interval (ms)±SD	151± 24	153± 23	153± 19	147± 22	152±21
Difficulties at PR interval Measurement (%)	4	2	12	0	Not evaluated
QRS interval (ms)±SD	93±9	99,2±9	98±10	91±9	89±9
Difficulties at QRS interval Measurement (%)	4	2	0	0	Not evaluated
QT interval (ms)±SD	379± 21	386± 32	389± 25	374± 25	368±27
Difficulties at QT interval Measurement (%)	4	0	4	2	Not evaluated
Isoelectric line issues (%)	2	0	0	10	Not evaluated
Signal artefact (%)	2	2	0	8	Not evaluated
Good Quality (%)	88	96	84	84	Not evaluated
Acceptable Quality (%)	12	4	16	16	Not evaluated
Poor Quality (%)	0	0	0	0	Not evaluated

Other

OP-032

Effects of semaglutide versus comparators on cardiovascular events across a continuum of baseline cardiovascular risk: Combined analysis of the sustain and pioneer trials

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Background and Aim: In patients with type 2 diabetes (T2D) at high risk of CV events, there were fewer major adverse CV events (MACE) with semaglutide vs. placebo [HRs (95% CIs): 0.74 (0.58;0.95) for subcutaneous, 0.79 (0.57;1.11) for oral semaglutide]. However, the effects of semaglutide on MACE in those at low-moderate risk of CV events were untested. We performed a post hoc analysis to examine CV effects of semaglutide by baseline CV risk derived from a large dataset with common adjudicated endpoints.

Methods: Subcutaneous once-weekly (SUSTAIN) and oral once-daily (PIONEER) semaglutide phase 3a trials were combined to assess time to first adjudication-confirmed MACE (CV death, nonfatal stroke, nonfatal myocardial infarction). A prognostic score (MACE risk score) was developed with independent data from LEADER (liraglutide vs. placebo), considering baseline variables common to all datasets. Semaglutide data were analyzed using Cox regression including effects of treatment, MACE risk score, and the interaction between both.

Results: The LEADER-derived MACE risk score predicted CV risk in the semaglutide data well (AUC: 0.77; Fig. 1). There was a reduced risk of MACE for semaglutide vs comparators across risk scores with a nonsignificant (p=0.06) trend to largest relative effects in those with lowest MACE risk score.

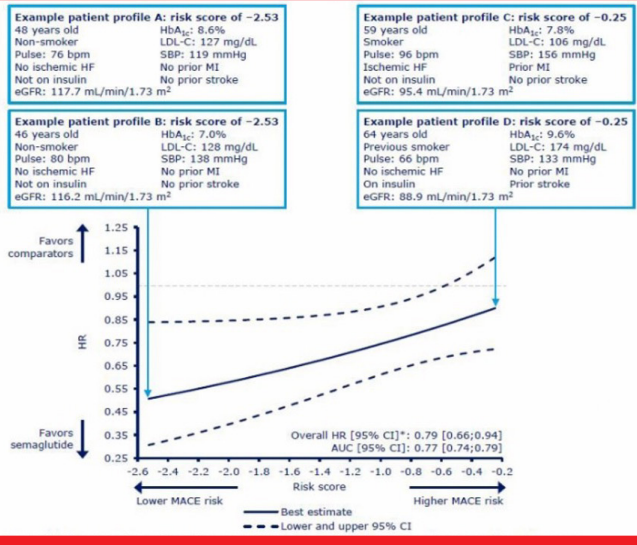

Figure 1. The HR (semaglutide vs comparators) for time to first adjudication-confirmed MACE as a function of baseline CV risk score in the semaglutide phase 3a programs

Overall HR for treatment effect (semaglutide vs comparators). Curves show the HR (solid curve; semaglutide vs comparators) and 95% CI (dased curves) estimated using a Cox regression model including effects of treatment, MACE risk score, and interaction between both. The MACE risk score developed using LEADER data (liraglutide vs placebo) considered all common baseline variables. Real patient profile examples (all are NYHA class I) were chosen at the 5- and 95- percentiles of risk-score distribution. The factors mentioned in relation to the patient profiles (including NYHA class) were identified using LEADER to significantly affect CV risk (no other factors were identified as important). Horizontal dashed line represents an HR of 1.00. AUC, area under the curve; bpm, beats per minute; CI, confidence interval; CV, cardiovascular; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; HF, heart failure; HR, hazard ratio; LDL-C, low-density lipoprotein-cholesterol; NYHA, New York Heart Association; MACE, major adverse cardiovascular events; MI, myocardial infarction; SBP, systolic blood pressure.

Conclusions: Semaglutide reduced the risk of MACE vs comparators in patients with T2D across baseline CV risk, including low CV risk.

Other

OP-033

Assessment and comparison of cardiovascular risk scoring models before noncardiac surgery, in a larger patient population

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Background and Aim: The cardiovascular risk of a noncardiac surgery patient must be evaluated from two aspects: The risk of the planned operation itself and his cardiovascular comorbidities. This would lead the clinician to take precautionary measures prior to the operation to reduce morbidity and mortality. Several risk scoring systems have been developed to evaluate the risk of surgery. In this study, we aimed to compare three cardiovascular risk scoring models ASA (American Society of Anesthesiologists Physical Status Classification System), Lee Index, and Gupta score with each other, in a larger patient population than our preliminary results presented last year.

Methods: 3423 preoperative patients are included in the study. Patients' demographic characteristics, current diseases, blood parameters, functional test results, and pre-and peri-operative medications were recorded. The predictive values of ASA, Lee Index, and Gupta scores are evaluated in terms of total mortality and cardiac endpoints (CEP) (cardiac mortality, acute myocardial infarction (MI), arrhythmia, and heart failure) in 30 days.

Results: 1735 (50.7%) patients were male (62±17 years). Total mortality rate was 1.9% (n=64). Body mass index (BMI) and hematocrit (HCT) values were significantly lower and mean age, systolic pulmonary arterial pressure (sPAB), left ventricular ejection fraction (LVEF) values were significantly higher in patients with mortality (Table 1). Mortality rate was significantly higher patients with coronary artery disease (CAD), chronic obstructive pulmonary disease(COPD), insulin-dependent diabetes mellitus (IDDM), previous MI, previous coronary artery by-pass grafting (CABG), hypertension (HT), cerebrovascular disease (CVD), peripheric arterial disease (PAD), heart failure (HF), atrial fibrillation(AF). Lee index, Gupta and ASA scores of patients with mortality were significantly higher (Table 1). Age (≥75), operation risk, HCT (<30), sPAB, PAH and ASA were identified as independent predictors of 30-day mortality. CEP were observed in 52 (1.5%) patients. Cardiac mortality rate, was %0.2 (n=8). LVEF and HCT values were significantly lower and mean age and sPAB values were significantly higher in patients with CEP. CEP was significantly higher in patients with CAD, hyperlipidemia, COPD, IDDM, previous MI, HF, and AF (Table 2). HF, sPAB, and operation risk were identified as independent predictors of CEP. In ROC Curves, the area under the curves of the Gupta, ASA, and Lee Index were 0.79, 0.75, and 0.68; and were 0.72, 0.71, and 0.65 for CEP, respectively.

Conclusions: When we compared three risk scoring systems: In 30 days Gupta had a higher predictive value mortality, Gupta and ASA outperformed the Lee Index in estimating CEP. Furthermore, all risk scoring systems showed a low correlation with each other. sPAB and HCT which are not found in these risk scoring systems, were found to be additional independent risk factors that significantly affected outcomes in our study.

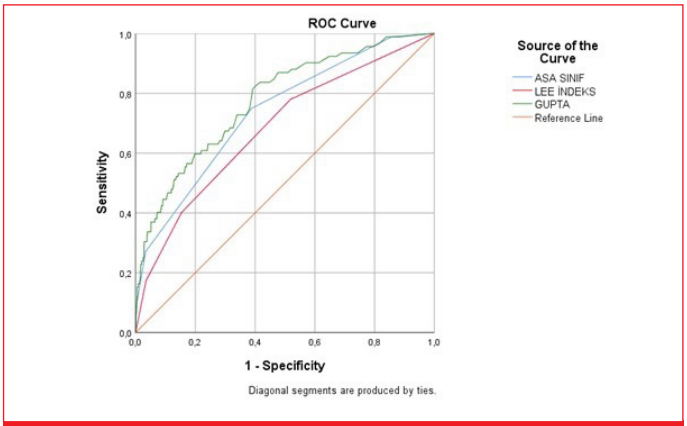

Figure 1. ROC Curve for 30-day mortality

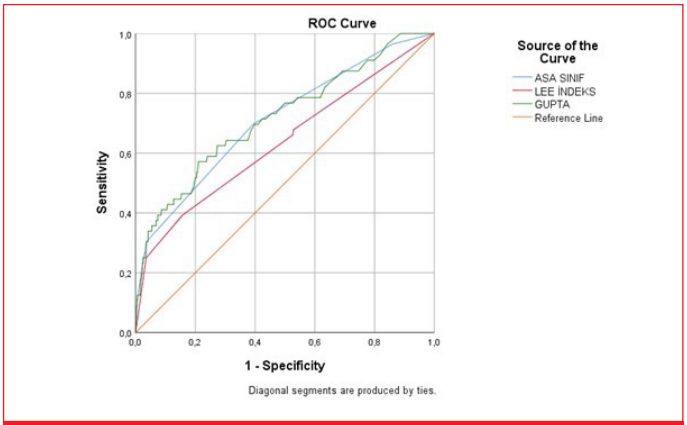

Figure 2. ROC Curve for cardiac endpoints

	30-day mortality n:64 (1.9%)	Survivors n: 3359 (98.1%)	p
Age (year)	71.7 ±13.7	61.8±17.1	**<0.001**
Men (n%)	40(62.5%)	1695(50.5%)	**0.056**
BMI	26.0±4.9	27.7±5.9	**0.03**
HT (n%)	51(79.7%)	2039 (60.7%)	**0.002**
HL (n%)	20(31.3 %)	838(24.9%)	0.249
Smoker (n%)	20 (31.3%)	978(29.1%)	0.710
CAD (n%)	22 (34.4%)	649 (19.3%)	**0.003**
COPD (n%)	11 (17.2 %)	198 (5.9%)	**<0.001**
PASP (mmHg)	36.1±12.4	29.4±9	**<0.001**
PASP (40) (mmHg)	20 (5.1%)	39 (1.5%)	**<0.001**
LVEF(n%)	58.7±5.8	56.4±8.4	**0.038**
DM (N%)	23 (35.9%)	866 (25.8%)	0.066
DM + insulin usage (n%)	14 (21.9%)	300 (8.9%)	**<0.001**
Previous Miyocard infarction (n%)	14 (21.9%)	299 (8.9%)	**<0.001**
Previous CABG (n%)	9 (14.1 %)	231 (6.9%)	**0.026**
CVA (n%)	10 (15.6%)	135(4%)	**<0.001**
PAD (n%)	13 (20.3%)	137 (4.1%)	**<0.001**
CKD (n%)	14 (21.9 %)	505 (15.0%)	0.131
HF (n%)	6 (9.4%)	142 (4.2%)	**0.045**
AF (n%)	14 (21.9%)	303 (9.0%)	**<0.001**
HGB	10.5±2.2	12.2±2.1	**<0.001**
HCT	32.4±7	37.3±6	**<0.001**
HCT27	10 (6.6%)	48 (1.8%)	**<0.001**
HCT30	26 (7.1%)	32 (1.3%)	**<0.001**
Creatinine (mg/dl)	1.24±1.3	1.28±1.6	0.824
ASA Score	3.19±0.96	2.29±0.76	**<0.001**
Gupta Score	3.50±5.53	0.53±1.19	**<0.001**
LEE Index	4.09±4.24	1.70±2.64	**<0.001**

NS: HT: Hypertension, HL: Hyperlipidemia, LVEF: Left ventricular ejection fraction , PASP: Pulmonary artery systolic , CAD: Coronary artery disease, DM: Diabetes Mellitus, HF: Heart failure, COPD: Chronic obstructive pulmonary disease, CVH: Cerebrovasculary disease, CKD: Chronic Kidney Disease,

Table 1. Characteristics of patients with 30-day mortality

	Cardiac endpoints n:52	Without endpoints n: 3371	P
Age (year)	68.73 ±14.35	61.91±17.09	**0.001**
Men (n%)	29(55.8%)	1706(50.6%)	0.460
BMI	28.19±7.18	27.64±5.87	0.520
HT (n%)	37(71.2%)	2053(60.9%)	0.132
HL (n%)	20(38.5 %)	838(24.9%)	**0.025**
Smoker (n%)	15 (28.8%)	983(29.2%)	0.961
CAD (n%)	16 (30.8%)	655 (19.4%)	**0.041**
COPD (n%)	9 (17.3%)	200 (5.9%)	**<0.001**
PASP (mmhg)	39.3±15.3	29.4±9	**<0.001**
PASP (40mmHg)	20 (43.5%)	369 (12.3%)	**<0.001**
LVEF(n%)	52.2±11.2	58.7±5.7	**<0.001**
DM (N%)	19 (36.5%)	870 (25.8%)	0.080
DM on insulin (n%)	11(21.2%)	303 (9%)	**0.003**
Previous Miyocard infarction (n%)	11 (21.2%)	302 (9%)	**0.002**
Previous CABG (n%)	6 (11.5%)	234 (6.9%)	0.198
CVA (n%)	4 (7.7%)	141(4.2%)	0.212
PAD (n%)	5 (9.6%)	145 (4.3%)	**0.063**
CKD (n%)	8 (15.4%)	511 (15.2%)	0.964
HF (n%)	14 (26.9%)	134(4%)	**<0.001**
AF (n%)	11 (21.2%)	306 (9.1%)	**0.003**
HCT30	11 (26.8%)	356 (12.8%)	**0.008**
HCT27	6 (14.6%)	146 (5.3%)	**0.008**
Creatinine (mg/dl)	1.28±0.83	1.28±1.62	0.998
ASA Score	3.06±1.02	2.29±0.76	**<0.001**
Gupta Score	2.42±3.55	0.56±1.39	**<0.001**
LEE Index	4.15±4.51	1.71±2.65	**<0.001**

HT: Hypertension, HL: Hyperlipidemia, LVEF: Left ventricular ejection fraction , PASP: Pulmonary artery systolic , CAD: Coronary artery disease, DM: Diabetes Mellitus, HF: Heart failure, COPD: Chronic obstructive pulmonary disease, CVH: Cerebrovasculary disease, CKD: Chronic Kidney Disease,

Table 2. Characteristics of patients with cardiac endpoints (MI, death, arrhythmia, heart failure)

Classification	Low risk	Intermediate risk	High risk
ASA Score	2062 (60.2%)	1225 (35.8%)	136 (4%)
Gupta Score	2946 (86.1%)	425 (12.4%)	52 (1.5%)
LEE Index	2873 (83.9%)	418 (12.2%)	132 (3.9%)

Table 3. Overall agreement of all risk scores

	LEE Index	Gupta
ASA	0.404	0.366
LEE Index	-	0.520

Table 4. Cronbach's Alpha values of scores

	OR	P	CI
Age (≥75)	2.318	0.004	1.310-4.101
Operation risk	1.872	0.006	1.194-2.936
HCT (<30)	3.233	<0.001	1.803-5.799
sPAB	1.030	0.016	1.010-1.050
PAD	3.370	0.001	1.603-7.087
ASA	3.172	<0.001	2.191-4.593

sPAB: Pulmonary artery systolic, PAD: Peripheric arterial disease, HCT: Hematocrit

Table 5. Independent predictors of 30-day mortality

	OR	P	CI
HF	7.360	<0.001	3.280-16.514
sPAB	1.040	p:0.003	1.013-1.067
Operation risk	1.887	0.017	1.120-3.179

HF: Heart failure, sPAB: Pulmonary artery systolic

Table 6. Independent predictors of cardiac endpoints

Other

OP-034

High risk characteristics in patients with hypertrophic cardiomiyopathy for predicting cardiovascular events

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Background and Aim: Sudden cardiac death is the most feared complication of hypertrophic cardiomyopathy due to its tendency to appear in young asymptomatic patients. Although intracardiac defibrillators proved to be most efficient treatment, selection of the patients for primary prevention is controversial due to lack of randomized trials. Current risk algorithms fail to determine all high-risk patients. In this study, we aimed to evaluate high risk factors to predict cardiovascular events in our hypertrophic cardiomyopathy population.

Methods: A total of 254 patients who fulfill the criteria of hypertrophic cardiomyopathy by echocardiography between 2011 and 2020 were retrospectively included in the study. Data on recent comprehensive vital clinical and survival status were obtained up to 30th January 2020, by hospital visit, telephone contact or from hospital system. All the factors that affected primary end points; all-cause of death, myocardial infarction, cerebrovascular events and appropriate ICD interventions; were examined during the median follow up 54.5 months. Clinical features used to determine in-

creased risk and guide ICD recommendations were based on consensus standards according to the guidelines. Patients risk score was calculated in accordance with ESC mathematically derived quantitative risk score to predict SCD event rates over 5 years.

Results: 254 patients with median age 58 were admitted in the study. During follow up 18 (7.1%) all-cause mortality, 4 (1.6%) myocardial infarction, 12 (4.7%) cerebrovascular events, 19 appropriate ICD interventions, 83 (32.7%) hospitalization due to cardiovascular cause were encountered. Median time of the primary end points was 37.5 months. The incidence of ischemic heart disease, atrial fibrillation, syncope, nonsustained ventricular tachycardia were higher in patients with primary end points. Mean ejection fraction was lower (p=0.000) and mean left atrium diameter was larger (p=0.002) in the group of patients who experienced primary end points. History of syncope and EF lower than 60 were significant (p<0,001, p<0,001) with primary end points in multivariate analysis. Patients with ICD encountered primary end points more frequently that contains appropriate ICD interventions (p=0.000). High risk patients according to risk score algorithm of European Society of Cardiology reached primary end points more frequently, significant amount of low risk patients also had cardiovascular events.

Conclusions: As the current risk algorithms for sudden cardiac death seem to be inadequate in identifying high risk patients, new algorithms with high sensitivity and specificity needs to be developed in our population.

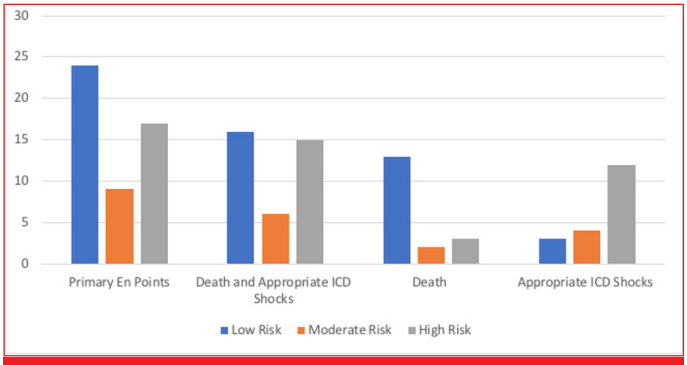

Figure 1. Comparison of end points due to risk groups
Composite end-points 24 (48%) low risk, 9 (18%) moderate risk, 17 (34%) high risk. Death and appropriate ICD shocks; 16 (43.2%) low risk, 6 (16.3%) moderate risk 15 high risk. Death 13 (72.2%) low risk, 2 (11.1%) moderate risk, 3 (16.7%) high risk. Appropriate ICD shocks 3 (15.8%) low risk, 4 (21.1%) moderate risk, 12 (63.2%) high risk.

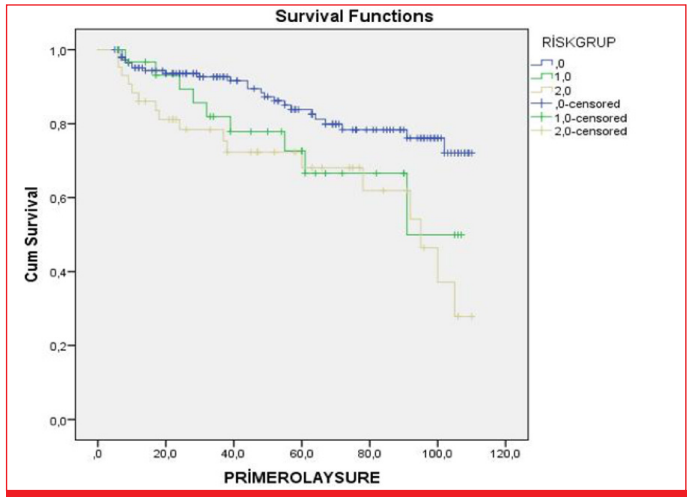

Figure 2. Kaplan Meier curve addressing outcome between risk groups
Kaplan–Meier curves for the cumulative incidence of primary outcomes in patients with different risk score groups. log rank analysis p=0.001

Table 1. The clinical and demographic characteristics of general population and patients with end points

	All Population (n=254)	Patients with End Points (n=50)
Age (years)*	58 (18-90)	60.0 (18-90)
Follow up (month)*	54.5 (6-110)	56 (6-106)
Male, n (%)	162 (63.8%)	34 (68.0%)
SCD history, n (%)	54 (22.6%)	14 (28.0%)
Syncope, n (%)	51 (21.4)	19 (38.0%)
Atrial Fibrilation, n (%)	82 (32.3%)	24 (48.0%)
ICD, n (%)	60 (23.6%)	22 (44.0%)
NSVT, n (%)	62 (37.8)	18 (36.0%)
Septal Myectomy, n (%)	9 (3.5%)	3 (6.0%)
Alcohol Septal Ablation, n (%)	4 (1.6%)	1 (2%)
NYHA III-IV, n (%)	60 (23.6%)	25(50%)
B-blockers, n (%)	199 (88.3%)	46 (92.0%)
Ca Chanel Blockers, n (%)	79 (33.1%)	15 (30.0%)
Amiodarone	27 (11.3%)	12 (24.0%)
Warfarin, n (%)	34 (14.2)	10 (20.0%)
EF %	58.9±5.4	55.3±9.1
Left Atrium (mm)	42.5±6.4	45.2±7
Interventricular Septum (mm)	17.9±3.4	17.6±3.6
LVOT gradient >30 mm Hg, n (%)	78 (30.7%)	14 (28%)
SCD Score 0-4 n (%)	151 (67.1%)	24 (48.0%)
SCD Score 4-6 n (%)	31 (13.8%)	9 (18.0%)
SCD Score >6 n (%)	43 (19.1%)	17 (34%)

*Median (min-max) ICD: intracardiac defibrillator, NSVT: Nonsustained Ventricular Tachycardia, NYHA: New York Hearth Association Functional Classification, LVOT: Left Ventricular Outflow, SCD: Sudden Cardiac Death

Table 2. The comparison of baseline clinical characteristics of patients with and without end points

	Patients without End Points (n=254)	Patients with End Points (n=50)	*P*-value
Age (years)*	57 (18-85)	60.0 (18-90)	0.078
Male, n (%)	128 (62.7%)	34 (68.0%)	0.488
SCD history, n (%)	40 (19.6%)	14 (28.0%)	0.304
Syncope, n (%)	32 (15.7%)	19 (38.0%)	0.001
Atrial Fibrillation, n (%)	58 (28.4%)	24 (48.0%)	0.01
ICD, n (%)	38 (18.6%)	22 (44.0%)	0.000
Ischemic Heart Disease, n (%)	45 (22.1%)	21 (42.0%)	0.006
NSVT, n (%)	44 (21.6)	18 (36.0%)	0.033
B-blockers, n (%)	153 (75.0%)	46 (92.0%)	0.029
Ca Chanel Blockers, n (%)	64 (31.4%)	15 (30.0%)	0.633
Amiodarone, n (%)	15 (7.4%)	12 (24.0%)	0.001
Warfarin, n (%)	24 (11.8%)	10 (20.0%)	0.147
EF %	59.8±3.6	55.3±9.1	0.000
Left Atrium (mm)	41.8±6.1	45.2±7	0.002
Interventricular Septum (mm)	17.9±3.4	17.6±3.6	0.455
LVOT Rest Gradient (mm Hg)	49.6±24.1	53.6±24.2	0.57

SCD: Sudden Cardiac Death, ICD: Intracardiac defibrillator, NSVT: Nonsustained Ventricular Tachycardia, EF: Ejection fraction

Table 3. Comparison of risk groups according to primary end points

	Low Risk Score (n=151)	Moderate Risk Score (n=31)	High Risk Score (n=43)	*P*-value
Primary Endpoints*, n (%)	24 (15.9%)	9 (29.0%)	17 (39.5%)	0.003
Death and Appropriate ICD Shocks, n (%)	16 (10.6%)	6 (19.4%)	15 (34.9%)	0.000
Death, n (%)	13 (8.6%)	2 (6.5%)	3 (7.0%)	0.430
Myocardial Infarction, n (%)	2 (1.3%)	2 (6.5%)	0 (0%)	
Cerebrovascular Events, n (%)	7 (4.6%)	2 (6.5%)	3 (7.0%)	0.798

Death, Myocardial Infarction, Cerebrovascular Events, Appropriate ICD Shocks

Other

OP-035

An evaluation of the fragmentation of occlusive calcifications in the vascular and urinary systems using cryotherapy

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Background and Aim: This study concerns a multi-layer gas carrier system with low thermal conductivity suited to the cryotherapy by fragmentation of calcifications in the vascular and urinary systems.

Methods: Specimens taken from calcified carotid artery plaques from each patient were divided into two groups, control and cold shock application. All specimens were kept for 30 s in a specially designed liquid nitrogen bath (-196 °C) to determine the effect of cold shock application. Next, a computer-controlled copper-constantan thermocouple was used to determine time-dependent temperature changes. Histopathological Analysis Carotid artery tissue specimens were rapidly trimmed to a volume of 1.5 cm^3 and fixed in 10% neutral formalin (Sigma Aldrich, St. Louis, MO, USA) for 24 h for light microscopy examination. Following fixation, the tissues were dehydrated by being passed through increasing ethanol (Merck GmbH, Darmstadt, Germany) series (30%,50%, 70%, 80%, 90%, 96%, and 100%). After dehydration, tissue specimens were cleared with xylol (Merck GmbH, Darmstadt, Germany) and embedded in paraffin blocks (Merck GmbH, Darmstadt, Germany). After staining, the tissue specimens were examined under a light microscope (Olympus BX51, Olympus Corporation, Tokyo, Japan) and photographed with an Olympus DP71 camera (Olympus Corporation, Tokyo, Japan). Scanning Electron Microscope Analysis: Specimens containing vascular and urinary tract system calcific structures were exposed to EDS analysis by examination under a scanning electron microscope (Jeol 59 JSM6610, Akishima, Japan) at an operating voltage of 20 kV.

Results: Histopathological Analysis: Widespread areas of fibrous callus and calcification in the subendothelial regions of the tunica intima layer compatible with Grade VIII of the American Heart Association classification of atheromatous lesions were present under light microscopic examination of the carotid artery tissue specimens of the carotid artery sections (Fig. 4a-4d) (29–31). Diffuse inflammation was also present (Fig. 4a, 4b, Fig. 5a, 5b). In contrast, a marked decrease was observed in areas of fibrous callus and calcification in the cold shock application group, with a low number of infiltration areas (Fig. 5c).

Conclusions: Catheter-based vascular (arterial) interventions and therapies have long been used in the treatment of arterial obstruction. One study involving arterial occlusion and reducing subsequent narrowing reported very good results from cold shock (cryotherapy), leading to stent restenosis in arterial obstruction have been obtained with this property. No studies have addressed the treatment of atheroma plaques using freezing. However, studies have reported that the effects of freezing on fibrin networks and blood plasma result in an irreversible breakdown. In our preliminary study, we observed that the freezing process eliminated bleb-like protrusions in subendothelial stellate cells and ball-like formations in the extracellular matrix

Figure 1. Light microscopy image of the calcified structure formed in the vascular system. H&E staining.

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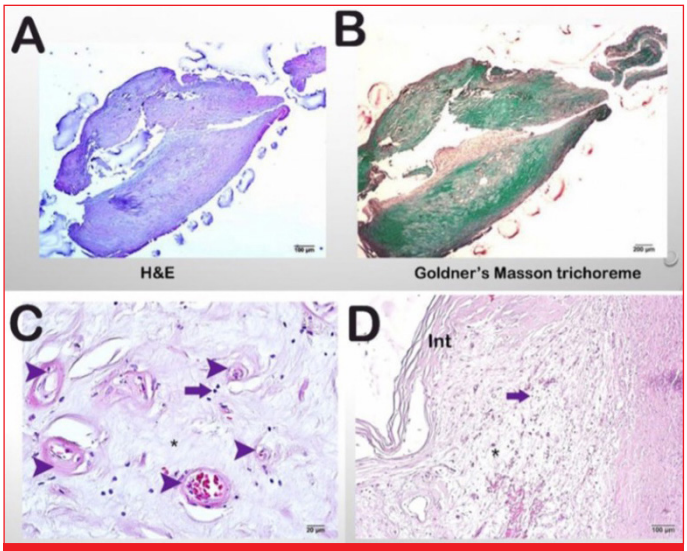


Figure 2. Light microscope image of the calcified structure formed in the vascular system. (A, C, D) H+E staining. (B) Goldner's Masson trichrome.

Grade VIII of the American Heart Association classification of atheromataus lesions; In the carotid artery tunica intima (In) subendothelial area, fibrous callus (asterisk) and calcification areas (arrowhead) are observed. Inflammation (arrow).

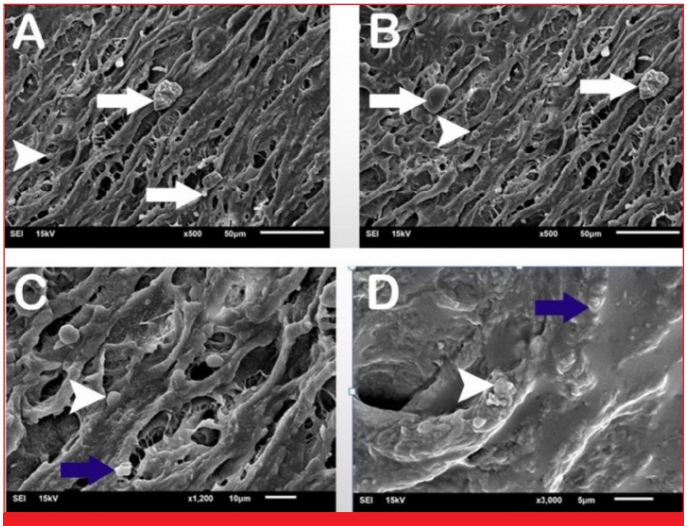
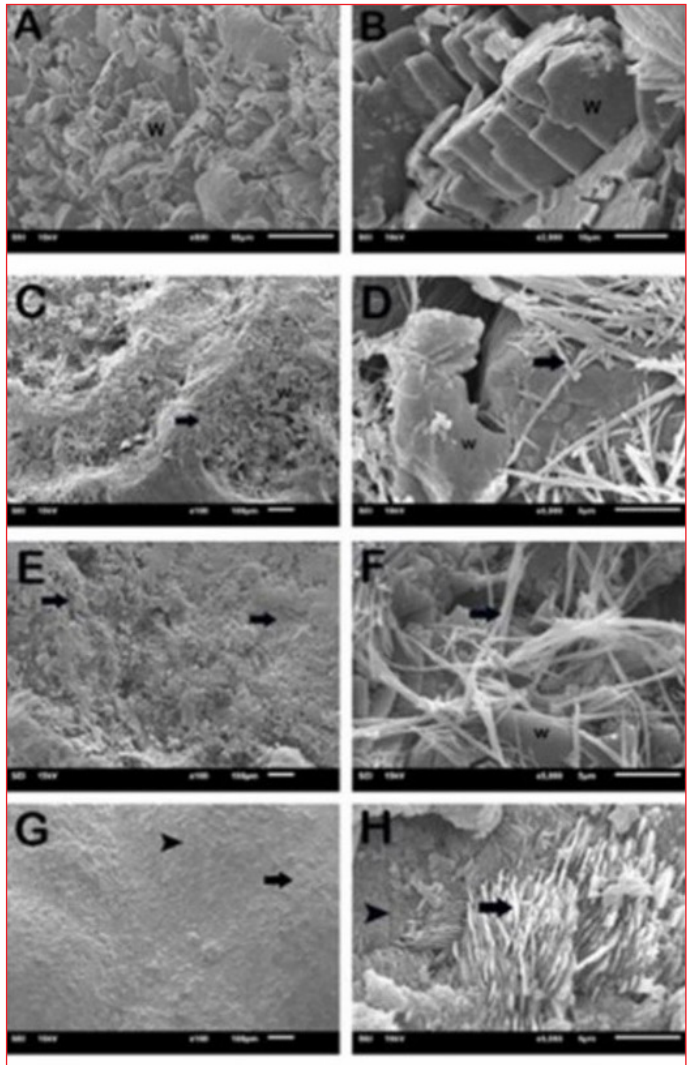


Figure 3. SEM image of atheroma plaque and treatment groups in the carotid artery.



A(x500)-B(x2000): control, calcium bipyramidal Weddellite crystals (CaC_2O_4 $2.25H_2O$; w) monitored. **C(x100)-D(x5000):** After 15 seconds of cold application, shrinkage (w) of bipyramidal Welwellite crystals is observed. It is also observed that the bipyramidal crystal structures are replaced by fibrous protrusions (arrow). **E(x100)-F(x5000):** After 30 seconds of cold application, shrinkage and significant (w) decrease of bipyramidal crystal structures are observed. Also, it is observed that it is replaced by common fibrous appendages (arrow). **G(x100)-H(x5000):** It is observed that the crystals disappear in the bipyramidal crystal structures after 60 seconds of cold application (arrow). Besides, Welwellite crystals are observed in widespread cracks (arrowhead).

Figure 4. SEM image of treatment groups before and after cryotherapy for kidney stones

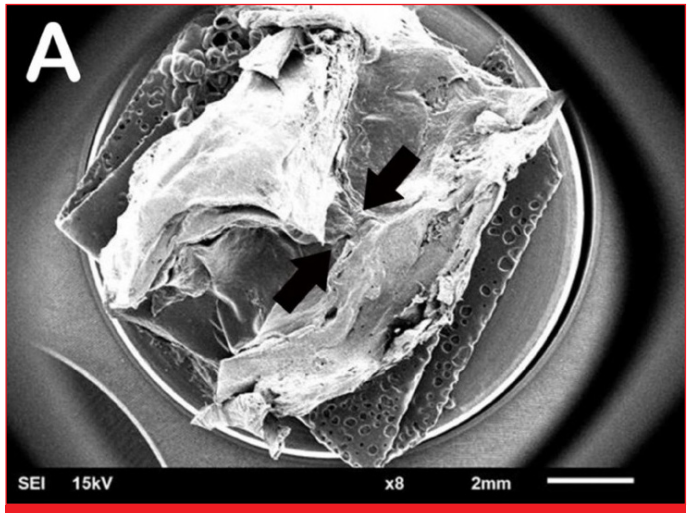


Figure 5. SEM image of atheroma plaque (arrow) in the carotid artery

Other

OP-036

Empagliflozin significantly prevents QTc prolongation due to Amitriptyline intoxication via intracellular calcium regulation

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Background and Aim: Empagliflozin is a SGLT-2 inhibitor used in the treatment of Type 2 diabetes and has positive effects on cardiovascular outcomes. Amitriptyline can be used in many clinical indications but leads to cardiotoxicity by causing QT prolongation. Our aim in the present study is to observe the effect of the concomitant use of amitriptyline and empagliflozin together, which have an effect on sodium and calcium balance in myocytes, on QTc by using ECG.

Methods: Twenty-four male Wistar-Albino rats were randomized into four groups. The control group received only serum physiologic (1 ml) via orogastric gavage (OG). The EMPA group received empagliflozin (10 mg/kg) via OG. The AMT group received amitriptyline (100 mg/kg) via OG. The AMT+EMPA group (n=6) received amitriptyline and empagliflozin at same dose (Fig. 1a). Under anesthesia; QT and QTC intervals were measured at baseline, first, and second hours (Fig. 1b).

Results: The differences in QT and QTc values between the AMT group and control group were observed from the 1nd hour (Table 1, Fig. 2). It was detected that the measurements of the control group were within normal limits. In the control group, QT was 77.33±9.02 ms at the basal, 73.50±2.26 ms at the 1st hour, 78.17±6.18 ms at the 2nd hour. QTc calculation was 165.42±18.34 ±10.5 ms at the basal, 166.63±17.92 msat the 1st hour, 184.65±12.86 ms at the 2nd hour. ECG findings of the EMPA group were within normal limits and similar to the control group (Table 1). The durations of QT interval and QTc calculations were found to be statistically longer in the AMT group than the control group at the 1st and 2nd hour ($p \leq 0.001$). Empagliflozin significantly ameliorated AMT-induced QT and QTc prolongation. The durations of QT interval were significantly lower at first ($p<0.001$) and 2nd hours ($p<0.01$) in the AMT+EMPA group compared to the AMT group. Moreover, QTc calculation was significantly lower in the AMT+EMP group than the AMT group at 1st and 2nd hour ($p<0.01$) (Table 1). Electrocardiographic comparisons of all groups for one second within the second hour can be seen in Figure 1.

Conclusions: In the present study, we have detected that empagliflozin significantly ameliorates amitriptyline induced QT prolongation. This effect is probably due to the opposite effects of these two agents in the intracellular calcium balance. It's known that; toxic dose of amitriptyline increases the amount of Sarcoplasmic Ca and the calcium permeability of Ryanodine channels, decreases SERCA-mediated Ca-reuptake by decreasing the calcium binding capacity of calsequestrin, and these leads QTc prolongation. It has been shown that empagliflozin increases Ca reuptake by causing a significant increase in SERCA activity, and decreases Ca sparks by causing inhibition of Ryanodine activity. With more clinical trials, the routine use of empagliflozin may be suggested to prevent QTc prolongation in the diabetic patients receiving amitriptyline.

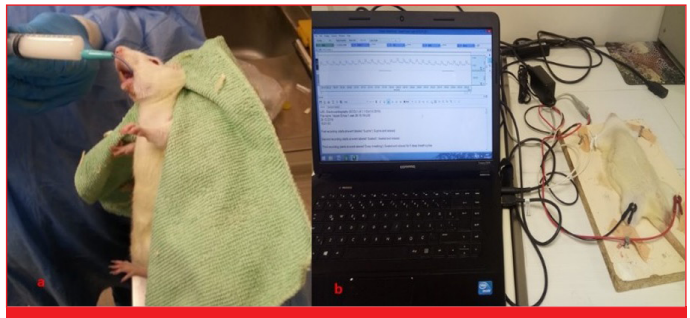

Figure 1. a: Drug adminstration via orogastrik tube b:ECG recording of the rats for the D2 lead at supine position

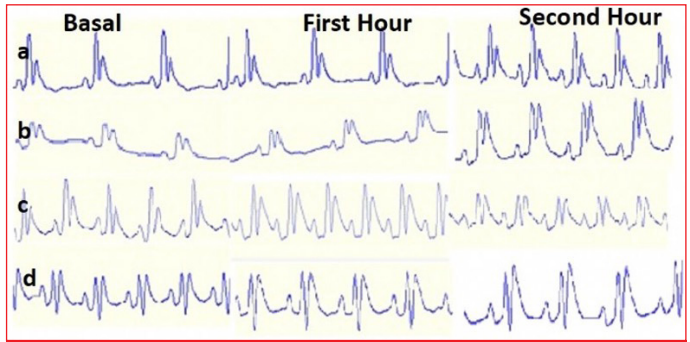

Figure 2. ECG comparisons of all groups at basal,1st and 2nd hour a:control, b: EMPA group, c: AMT group, d: AMT+EMPA group

Table 1.

	Control	Empagliflozin	Amitriptilin	Amitriptilin+ Empagliflozin	*P*-value
Bazal					
Qt ms					
Mean ± SD	77.33±9.02	78.33±6.28	71.50±5.68	73.33±8.02	0.35
HR					
Mean ± SD	263.00±39.38	230.00±10.06	314.83±42.48	287.50±29.99	0.002
QTc ms					
Mean ± SD	165.42±18.34	152.57±11.07	163.11±11.59	159.97±15.18	0.453
First Hour					
Qt ms					
Mean ± SD	73.50±2.26	75.67±4.27	108.67±5.96[a]	90.33±5.39[b]	<0.001
HR					
Mean ± SD	335.50 (75.75)	245.00 (144.25)	248.50 (123.50)	232.50 (107.25)	0.279
QTc ms					
Mean ± SD	166.63±17.92	154.60±20.43	227.45±26.89[a]	179.40 ±17.63[c]	<0.001
Second Hour					
Qt ms					
Mean ± SD	78.17±6.18	77.33±7.31	106.00±12.60[a]	87.83 ±4.54[c]	<0.001
HR					
Mean ± SD	335.83±21.99	295.67±30.54	288.67±53.86	326.30±36.97	0.118
QTc ms					
Mean ± SD	184.65±12.86	171.63±20.36	229.89±19.83[d]	191.66±10.93[c]	<0.001

[a]: amitiriptilin vs control *P*<0.001 [b]: amitriptilin+ empagliflozin vs amitriptilin *P*<0.001 [c]: amitriptilin+ empagliflozin vs amitriptilin *P*<0.01 [d]: amitiriptilin vs control *P*=0.001

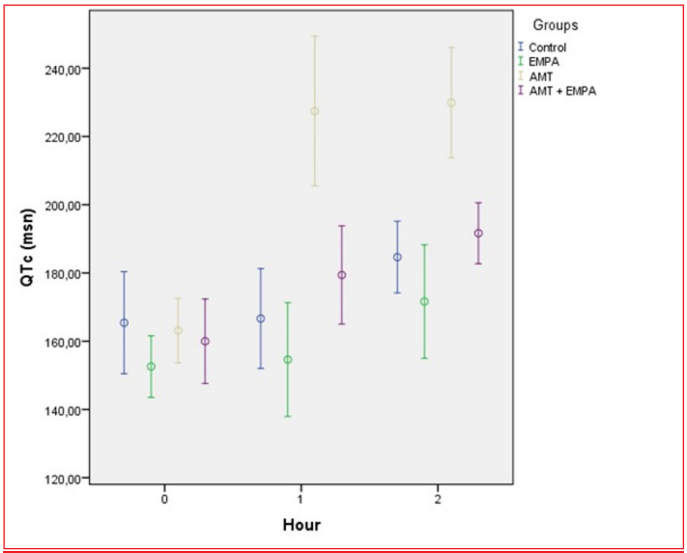

Figure 3. QTc comparisions for all groups basal, 1st and 2nd hour

Heart valve diseases

OP-037

Evaluation of procedural, clinical outcomes and 8-year survivals of TAVI: A single-center experience with different bioprosthetic valves

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Background and Aim: Indications for transcatheter aortic valve implantation (TAVI) have expanded in the last decade due to the number of experienced operators' increase and improved results and longer survival rates from many clinical trials. We aimed to present clinical, electrocardiographic, echocardiographic and laboratory results of TAVI with four different bioprosthetic valves.

Methods: A retrospective, single-center study included 441 patients with symptomatic severe aortic stenosis out of 459 patients who applied to the Cardiology Department of Dokuz Eylül University Hospital between June 2012 and May 2019 and were approved for TAVI by the heart team.

Results: A total of 441 patients with severe aortic stenosis, including 243 women (55%), were included in the study. The average age of the patients was 77.7±7.8 years. The mean logistic EuroSCORE 23.34±11.3% and mean STS score were 3.92±2.05%. TTE were shown baseline LVEF was 51.62±13.67%. 211 (47.8%) of the patients were treated with Medtronic CoreValve (Medtronic, Minneapolis, MN, USA), 176 (39.9%) were treated with Portico valve, 51 (11.6%) were treated with Edwards-SAPIEN valve (Edwards Lifesciences, Irvine, CA) and 3 (0.7%) were treated with Direct Flow Medical (Santa Rosa, CA). The median follow-up of intensive care unit and hospi-

tal stay were four days and 14 days, respectively. After TAVI, LVEF improved in all patients, especially in the patients with LVEF below 40%. The device success rate was 88.4% according to VARC-2 and 84.1% according to VARC-3. Vascular complications were observed in 47 (10.7%) patients. Interestingly, vascular complications were detected less common in patients with 0 blood type. 47 (10.7%) of patients needed permanent pacemaker implantation. Acute cerebrovascular event developed in 2.1% of patients; contrast nephropathy developed in 9.1%, and the need for hemodialysis in 1.4% of patients. In-hospital mortality rate was 7%. The independent predictors of in-hospital mortality were detected pre-TAVI serum albumin level (OR: 0.230; p=0.028), post-TAVI cerebrovascular event development (OR: 15.904; p=0.004), and post-TAVI contrast nephropathy (OR: 6.322; p=0.001). In addition, all-cause first-year mortality was observed in 21.3% of the patients. Pre-TAVI serum albumin level (OR: 0.364; p=0.011), and pre-TAVI serum neutrophil/lymphocyte ratio (OR: 1.144; p=0.019) was detected as the independent predictors of all cause first-year mortality. In the analysis of Kaplan Meier curve, the median survival time was detected 53 months at the 8-year follow-up. According to Kaplan Meier analysis, longer survival time was observed in women of 61 months compared to men of 40 months (p=0.024 Log Rank). Finally, the fifth-year and the eighth-year survival rate were 46.5% and 26.2%, respectively.

Conclusions: Our TAVI experience with four different bioprosthetic valves demonstrated in-hospital and first-year mortality predictors as well as our study has the largest patient population in a single center in Turkey with the 8-year follow-up.

Table 1. Independent predictors of in-hospital mortality according to multivariate regression analysis

	β (SE)	Wald	OR (95% CI)	*P*-value
Pre-TAVI serum albumin level	-1.471 (0.669)	4.836	0.230 (0.062-0.852)	0.028
Post-TAVI cerebrovascular event	2.767 (0.948)	8.518	15.904 (2.481-101.943)	0.004
Post-TAVI contrast nephropathy	1.844 (0.565)	10.637	6.322 (2.087-19.150)	0.001

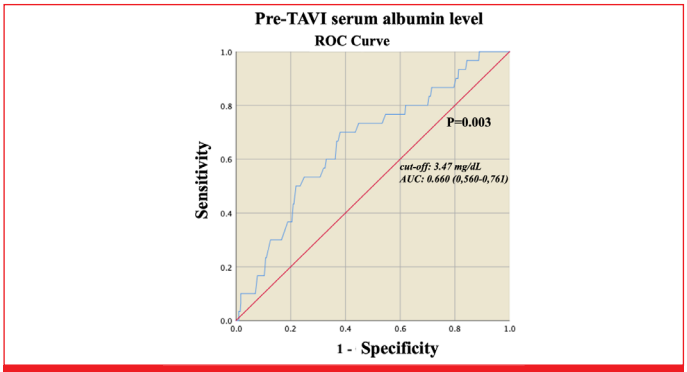

Figure 1. Receiver operating characteristic curve of pre-TAVI serum albumin levels for predicting in-hospital mortality

Table 2. Independent predictors of first-year mortality according to multivariate regression analysis

	β (SE)	Wald	OR (95% CI)	*P*-value
Pre-TAVI serum albumin level	-1.012 (0.399)	6.426	0.364 (0.166-0.795)	0.011
Pre-TAVI neutrophil/lymphocyte ratio	0.135 (0.057)	5.536	1.144 (1.023-1.280)	0.019

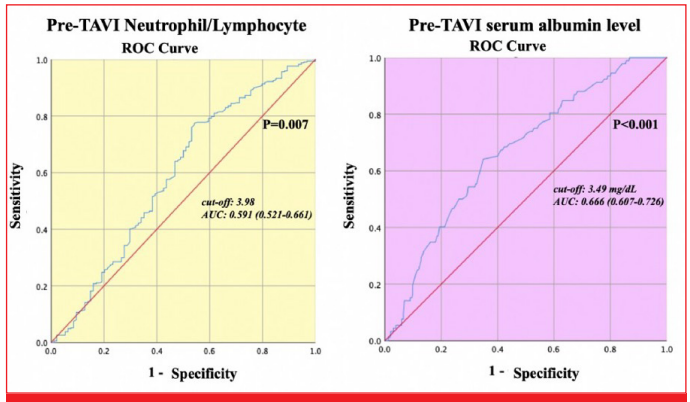

Figure 2. Receiver operating characteristic curve of pre-TAVI neutrophil/lymphocyte ratio and serum albumin levels for predicting first-year mortality

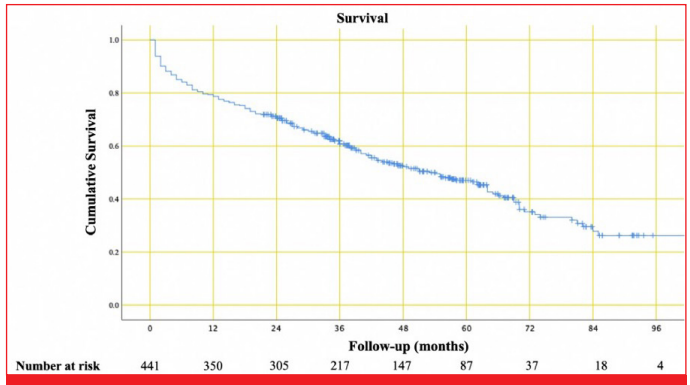

Figure 3. Kaplan Meier survival curve of all patients

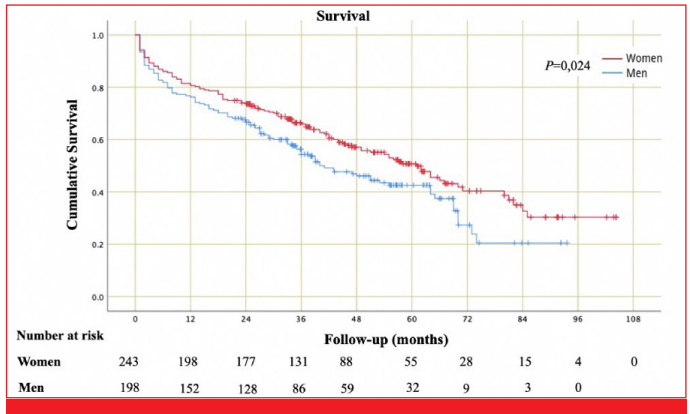

Figure 4. Kaplan Meier survival curve according to gender

Heart valve diseases

OP-040

Head to head compArison of ThrombolyTic therapy and sUrgery in patients with obStructive prosthetic Heart vAlve thrombosis: The multicenter HATTUSHA Trial

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Background and Aim: Prosthetic valve thrombosis (PVT) is one of the life-threatening complications of prosthetic heart valve replacement. Due to the lack of randomized controlled trials, the optimal treatment of PVT remains controversial between thrombolytic therapy (TT) and surgery. This study aimed to prospectively compare the outcomes of TT and surgery as the first-line treatment strategy in patients with obstructive PVT.

Methods: A total of 158 obstructive PVT patients [female: 103 (65.2%), median age: 49 (39-60) years] were enrolled in this multi-center observational prospective study. The selection of therapeutic strategy was based on clinical judgement. TT was performed using slow (6 h) and/or ultra-slow (25 h) infusion of low dose tissue plasminogen activator (tPA) (25 mg) mostly in repeated sessions. All patients were evaluated by serial transthoracic and transesophageal echocardiographic studies and followed up for 3 months.

Results: The initial management strategy was TT in 83 (52.5%) patients and surgery in 75 (47.5%) cases. The success rate of TT was 90.4% with a median tPA dose of 59 (37.5-100) mg. The incidence of minor [29 (38.7%) vs. 7 (8.4%), p<0.001] and major [31 (41.3%) vs. 5 (6%), p<0.001] complications, and the 3-month mortality rate [14 (18.7%) vs. 2 (2.4%), p=0.001] was significantly higher in the Surgery group as compared to TT group. Being in the Surgery group [OR: 20.8 (95% CI: 2.4-184.3), p=0.006] and high systolic pulmonary artery pressure [OR: 1.05 (95% CI: 1.009-1.096, p=0.017] were identified as the independent predictors of mortality.

Conclusions: Prolonged infusions of low dose TT are found to be superior to surgical therapy in providing effective therapy with lower complications and significantly reduced mortality rates in NYHA class I-IV patients with obstructive PVT. Furthermore, the combination of two regimens-slow and ultra-slow infusions of low dose tPA- can be performed in the same index patient during the clinical course of the disease. Consequently, TT should be the first-line treatment in patients with obstructive PVT in the absence of contraindications and reoperation should be left for patients in which TT is contraindicated, or in those where TT has already failed.

Table 1. Comparison of baseline clinical characteristics between groups.

Clinical feature	Thrombolytic therapy (n=83)	Surgery (n=75)	*P*-value
Age (years)	49 (37-58)	49 (39-60)	0.618
Gender, n (%)			
Male	23 (27.7)	32 (42.7)	
Female	60 (72.3)	43 (57.3)	0.049
BMI (kg/m^2)	26.3 (24.3-28.9)	25.5 (24.7-28.7)	0.712
ETSVS (months)	35 (10-76)	60 (20-108)	0.028
Heart rhythm			
Sinus, n (%)	46 (55.4)	46 (61.3)	
Atrial fibrillation, n (%)	37 (44.6)	29 (38.7)	0.452
NYHA, n (%)			
Class I	15 (18.1)	13 (17.3)	
Class II	36 (43.4)	36 (48)	
Class III	19 (22.9)	18 (24)	
Class IV	13 (15.7)	8 (10.7)	0.811
Chief complaint on admission, n (%)			
Asymptomatic	12 (14.5)	13 (17.3)	0.788
Valve obstruction related symptoms	61 (73.5)	55 (73.3)	
Embolism related symptoms	10 (12)	7 (9.3)	
History of, n (%)			
Stroke	11 (13.3)	8 (10.7)	0.618
TIA	6 (7.2)	5 (6.7)	0.890
HT	24 (28.9)	25 (33.3)	0.549
DM	13 (15.7)	9 (12)	0.507
CAD	8 (9.6)	7 (9.3)	0.948
Asthma/COPD	3 (3.6)	3 (4)	1.0
Previous PVT	15 (18.1)	19 (25.3)	0.267
Thyroid dysfunction	3 (3.6)	2 (2.7)	1.0
Smoking	11 (13.3)	10 (13.3)	0.988
Family history of thromboembolism, n (%)	11 (13.4)	3 (4)	0.039
Admission INR	1.65 (1.3-2.5)	1.66 (1.34-2.71)	0.594
Thrombosed valve location, n (%)			
MVR	55 (67.1)	63 (86.3)	
AVR	18 (22)	6 (8.2)	
TVR	9 (11)	4 (5.5)	0.019

Table 2. Comparison of baseline echocardiographic findings between the groups

Echocardiographic feature	Thrombolytic therapy (n=83)	Surgery (n=75)	*P*-value
2D-thrombus area (cm^2)	1.1 (0.9-1.3)	1.1 (0.9-1.4)	0.697
2D-mobile thrombus length (cm)	1.4 (1.2-1.6)	1.1 (0.75-1.45)	0.085
Mitral (n=119)	(n=55)	(n=64)	
Valve area (cm^2)	1.1(0.9-1.4)	0.95 (0.8-1.25)	0.035
Max gradient (mm Hg)	28 (25-35)	30 (25-36)	0.496
Mean gradient (mm Hg)	15 (12-21)	16 (12-23)	0.434
Aortic (n=26)	(n=19)	(n=7)	
Valve area (cm^2)	0.65 (0.6-0.72)	0.8 (0.74-0.83)	0.026
Max gradient (mm Hg)	93 (65-115)	84 (76.5-104.75)	0.975
Mean gradient (mm Hg)	58 (40-70)	49 (39-65.75)	0.726
Tricuspid (n=13)	(n=9)	(n=4)	
Valve area (cm^2)	1.1 (0.77-1.4)	0.78 (0.72-0.88)	0.163
Max gradient (mm Hg)	23 (20-24.5)	25.5 (19.5-30.75)	0.391
Mean gradient (mm Hg)	12 (11-14.5)	11.5 (10.25-15.75)	0.584
Stuck leaflet, n (%)	56 (67.5)	60 (80)	0.075
LV ejection fraction (%)	60 (50-60)	60 (50-60)	0.844
LA diameter (cm)	4.6 (4.1-4.9)	4.7 (4.5-4.9)	0.006
LA spontaneous ECHO contrast, n (%)	43 (51.8)	53 (69.3)	0.025
Estimated sPAP (mm Hg)	35 (30-45)	35 (30-50)	0.104

Table 3. Comparison of main laboratory findings between the study groups

Blood work-up feature	Thrombolytic therapy (n=83)	Surgery (n=75)	*P*-value
Glucose, (mg/dL)	101 (92-133)	104 (94-116)	0.914
Creatinine, (mg/dL)	0.79 (0.62-1.03)	0.84 (0.69-1.09)	0.097
Aspartate aminotransferase, (U/L)	23 (17-28)	25 (19-30)	0.129
Alanine aminotransferase, (U/L)	19 (14-24)	20 (14-26)	0.346
LDH, (U/L)	342 (259-393)	349 (300-410)	0.446
Total bilirubine, (mg/dL)	0.7 (0.5-1.0)	0.7 (0.5-1.16)	0.417
White blood cell count, (109/L)	9.1 (7-11)	9.9 (7.4-12.4)	0.108
Hemoglobin, (g/dL)	12.4 (11-13.9)	12.3 (10.7-13.7)	0.643
Platelet (109/L)	236 (179-306)	242 (192-330)	0.39
CRP, (mg/dL)	1.5 (0.53-3.7)	1.69 (0.56-6.71)	0.452
ESR, (mm/h)	20 (13.5-42.5)	25 (9.75-42)	0.946

Table 4. Comparison of adverse event rates between the study groups

Variables*	Thrombolytic therapy (n=83)	Surgery (n=75)	*P*-value
Hospital stay (days)	5 (4-7)	9 (5-13)	<0.001
Total complications, n (%)	10 (12)	47 (62.7)	<0.001
Major complications, n (%)	5 (6)	31 (41.3)	<0.001
Minor complications, n (%)	7 (8.4)	29 (38.7)	<0.001
Total embolic complications, n (%)	2 (2.4)	4 (5.3)	0.424
Total bleeding, n (%)	7 (8.4)	12 (16)	0.144
Major bleeding, n (%)	2 (2.4)	7 (9.3)	0.086
Cerebral	1 (1.2)	1 (1.3)	1.0
Non-cerebral	1 (1.2)	6 (8)	0.054
Minor bleeding, n (%)	5 (6)	5 (6.7)	1.0
Re-thrombosis requiring therapy, n (%)	2 (2.4)	5 (6.7)	0.258
3 months or in-hospital death, n (%)	2 (2.4)	14 (18.7)	0.001

Table 5. Cox regression model including the covariates for predicting in-hospital or 3-month mortality

Variables	Multivariable analysis OR (95% CI)	*P*-value
Age	1.016 (0.964-1.070)	0.556
Gender	2.391 (0.530-10.790)	0.257
NYHA Functional Class	1.318 (0.656-2.649)	0.438
Baseline sPAP	1.035 (1.004-1.067)	0.026
2D Thrombus Area	0.720 (0.260-1.995)	0.528
Presence of stuck leaflet	3.044 (0.524-17.691)	0.215
Baseline LVEF	0.968 (0.909-1.032)	0.324
Treatment Group (Surgery)	17.155 (2.133-137.977)	0.008

Other

OP-041

Is Automatic CPR more effective than manuel CPR in patients with cardiac arrest in the emergency department: Comparison with ETCO2 values

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Background and Aim: A well-performed cardiopulmonary resuscitation (CPR) among patients suffering from cardiac arrest is predictive of survival. Mechanical CPR devices are able to negate fatigue of healthcare staff delivering CPR and thus to administer highly efficient CPR and ETCO2 is a non-invasive indicator of CPR efficacy by acting as a surrogate marker of effective circulation during CPR effort. Our objective was to compare the effectivenesses of mechanical and manual chest compressions in cardiac arrest by using continuous end-tidal PCO2 (ETCO2) monitoring.

Methods: A prospective observational study of patients who experienced cardiac arrest while being monitored at the emergency department (ED) was conducted. Demographics, ECG rhythm, CPR technique (manual or automatic CPR), laboratory data, the time to return of spontaneous circulation (ROSC), length of stay in the Emergency department, hospitalization and mortality were compared according to CPR type. Patients under the age of 18 and those who had arrest due to trauma or drowning were excluded.

Results: 90 patients meeting the inclusion criteria were included in the study. Demographic and clinical characteristics, the 15th-minute, and 20th-minute SaO2 levels and the 10th-, 15th-, and 20th-minute ETCO2 levels showed a significant difference between the automatic CPR and manual CPR with all three variables being higher in the automatic CPR group (p=0.013, p=0.002, p=0.037, p=0.002, p<0.001, respectively).

Conclusions: According to the results of our study, we found that automatic CPR machine provides high quality CPR than manuel CPR in hospital cardiac arrest and mid-resuscitation ETCO2 values equal to or greater than 20 mm Hg appear to be a good predictor of ROSC, and they may perhaps become a predictor of survival in the upcoming years. Further research is needed to investigate how the predictive performance of ETCO2 for ROSC would be affected by the timing of its measurement during CPR and to determine sensitive and specific cut-off values for determining the efficacy of both manual and mechanical CPR.

Coronary artery disease / Acute coronary syndrome

OP-042

Prognostic value of Systemic Immune-Inflammation Index in hospitalized COVID-19 patients with coronary artery disease

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Background and Aim: Coronavirus disease 2019 (COVID-19) is an ongoing pandemic that has affected millions of people worldwide. COVID-19 patients with multiple comorbidities including coronary artery disease (CAD) show poor clinical outcomes. Inflammation has been associated with many diseases, including cardiovascular disease. The aim of our

study was to evaluate the usefulness of systemic immune-inflammation (SII) index at admission in predicting in-hospital outcomes in hospitalized COVID-19 patients with coronary artery disease.

Methods: In this study, we retrospectively evaluated 403 consecutive patients hospitalized for COVID-19 who had a previous diagnosis of CAD in two centers, between March 15, 2020 and December 01, 2020. We compared clinical features, laboratory findings and in-hospital outcomes according to the SII index. The SII index was calculated as follows: platelet count × (neutrophil/lymphocyte).In-hospital clinical outcomes were defined as death, respiratory failure requiring mechanical ventilation and requiring vasopressor support, length of intensive care unit (ICU) stay and length of hospital stay. The ROC curve was utilized to evaluate SII index and predict in-hospital outcomes (Area under the curve 0.755; p<0.001).

Results: Mean age of the study population was 72.4±9.8 years old and 68.7% were male. According to SII index, 403 patients were divided into SII indexlow group (n=219) with mean SII index of 4658.5±4095 and SII indexhigh group (n=184) with mean SII index of 697.5±382. There was no significant difference between the two groups in terms of comorbidities include hypertension, diabetes mellitus, atrial fibrillation (p>0.05, for all). Inflammatory parameters were significantly higher in patients with SII indexhigh group as compared those with SII indexlow group (Table 1). The in-hospital clinical outcomes occured in 17.4% of into SII indexlow group, 56 % of patients with SII indexhigh group (p<0.001).

Conclusions: We concluded that SII index can predict the in-hospital clinical outcomes in hospitalized COVID-19 patients with coronary artery disease.

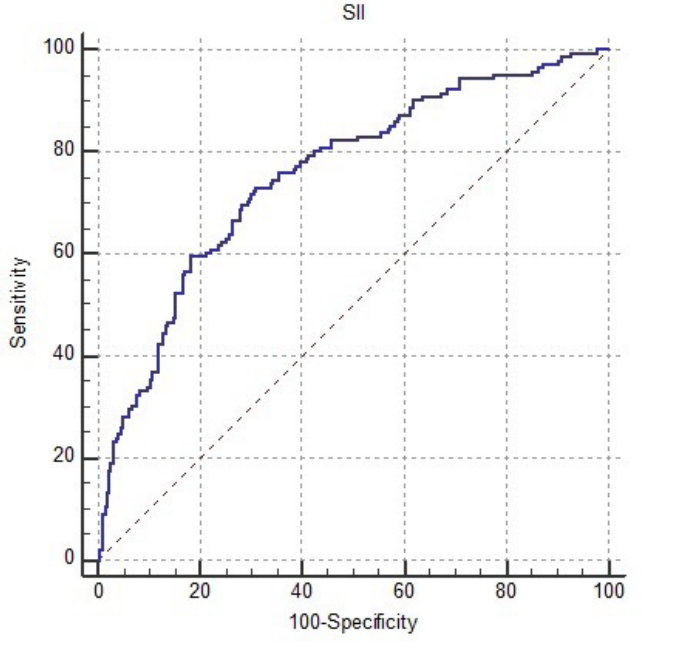

Figure 1. ROC curve for SII index

Table 1. Baseline characteristics and in-hospital outcomes according to SII index

Variables	SII index low <1509 group (n=219)	SII index high ≥ group (n=184)	*P*-value
CRP, mg/L	68.3 (20.5-120.4)	146.85 (84.0-222.2)	<0.001
Procalsitonin, ng/mL	0.13 (0.1-1.64)	1.6 (0.35-11.65)	<0.001
Ferritin, ng/dL	389.0 (174.5-776.0)	729 (394.5-1523.0)	<0.001
D-dimer, ng/mL	1.06 (0.5-3.5)	3.56 (1.65-8.41)	<0.001
Fibrinogen, mg/dL	461.1 (327.3-610.7)	601.4 (415.8-763.1)	<0.001
Length of hospital stay, days	7.0 (5.0-13.0)	11.0 (8.0-19.0)	<0.001
Length of ICU stay, days	0.00 (0.0-0.3)	6.0 (0.0-11.0)	<0.001
Mechanical ventilation, n (%)	29 (13.2%)	85 (46.2%)	<0.001
Vasopressör support, n (%)	33 (15.1%)	89 (48.4%)	<0.001
Death, n (%)	27 (12.3%)	82 (44.6%)	<0.001

Coronary artery disease / Acute coronary syndrome

OP-043

The effects of medical history, electrocardiographic biochemical and coronary angiographic features and applied treatment on in-hospital and long-term survival of patients attending with cardiac arrest

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Background and Aim: We carried out this study with the aim of finding the in-hospital and long-term survival rates in these patients and the factors that predict and affect it, using our experience and our database, on sudden cardiac arrest, which is not widely studied in the medical literature. We designed our study on patients who were hospitalized with a diagnosis of sudden cardiac arrest and underwent interventional coronary angiography

Methods: 171 patients (mean age 57.6±12.8 years; male gender ratio 80.7%) who were admitted to our hospital with a diagnosis of cardiac arrest between 2013 and 2020 and underwent invasive coronary angiography were included in the study we designed as a single center and retrospective. Mean follow-up time in hospital was 10.6±19.8 days and long-term mean follow-up was 409.7±580.4 days.

Results: Factors with statistically significant difference in in-hospital mortality between patients who died and were discharged alive were: Age (60.9±11.6; 54.8±13.2; p=0.002), presence of diabetes (37%; 21.1%; p=0.028), the place where

the cardiac arrest occurred (the rate of out-of-hospital cardiac arrest was 54% in the deceased group, 30% in the survived group; in-hospital cardiac arrest rate was 46% in the deceased group, in the survived group with 70%; p=0.002), unsuccessful percutaneous coronary intervention (16%; 2.2%; p=0.003). Factors with a statistically significant difference in long-term mortality between patients who died and who survived: Age (61.1±11.4; 53.5±13.2; p<0.001), location of cardiac arrest (the rate of out-of-hospital cardiac arrest was 51.1% in the group who died; 29.9% in the survived group; in-hospital cardiac arrest rate 48.9% in the group who died; 70.1% in the survived group; p=0.008), failed percutaneous coronary intervention (16%; 2.2%; p=0.003), making the CABG decision (9.6%; 1.3%; p=0.013) and having a low EF at follow-up (72.7%; 22.7%; p=0.002) and preserved EF (9.1%; 41.3%; p=0.002). The presence of ST elevation, history of coronary artery disease and PCI/CABG, medical treatment decision after coronary angiography and successful PCI application, location of the culprit coronary lesion, and SYNTAX-1 score have not been shown to significantly contribute to in-hospital and long-term mortality. Patients who did not have significant stenosis in their coronary arteries and had single-vessel disease had lower mortality during in-hospital and long-term follow-up compared to patients with two vessels, three vessels and left main coronary (p=0.012 and p=0.001).

		Hastane içi mortalite görülen hastalar (n=81)	Sağ olarak taburcu edilen hastalar (n=90)	p değeri
Yaş (mean±sd)		60,9±11,6	54,8±13,2	**0,002**
Erkek Cinsiyet		62 (%76,5)	76 (%84,4)	0,245
Sigara öyküsü		31 (%38,3)	46 (%51,1)	0,124
Hipertansiyon		35 (%43,2)	41 (%45,5)	0,878
Diyabetes mellitus		30 (%37)	19 (%21,1)	**0,028**
KAH ÖYKÜSÜ	YOK	57[a] (%70,4)	66[a] (%73,3)	
	NONOBSTRUKTİF KAH	7[a] (%8,6)	8[a] (%8,9)	0,549
	PKG ÖYKÜSÜ	17[a] (%21)	14[a] (%15,6)	
	CABG ÖYKÜSÜ	0[a] (%0)	2[a] (%2,2)	
CPA YERİ	HASTANE DIŞI	44[a] (%54)	27[b] (%30)	
	HASTANE İÇİ	37[a] (%46)	63[b] (%70)	**0,002**
MI TİPİ	NON- ST ELEVE MI	27[a] (%33,3)	30[a] (%33,3)	
	ST ELEVE MI	54[a] (%66,7)	60[a] (%66,7)	1,000
İŞLEM SONUCU	FARMAKOLOJİK TEDAVİ	11[a] (13,7)	19[a] (21,2)	
	BAŞARILI PKG	50[a] (61,7)	66[a] (73,3)	**0,003**
	BAŞARISIZ PKG	13[a] (16)	2[b] (2,2)	
	CABG KARARI	7[a] (8,6)	3[a] (3,3)	
KÜLPRİT LEZYON VARLIĞI YERİ	NORMAL KORONER/NONKRİTİK DARLIK	5[a] (6,2)	12[a] (13,3)	
	LMCA	2[a] (2,5)	1[a] (1,1)	
	LAD	33[a] (40,7)	46[a] (51,1)	
	CX	16[a] (19,8)	14[a] (15,6)	0,177
	RCA	21[a] (25,9)	12[a] (13,3)	
	IMA/YÜKSEK OM	4[a] (4,9)	5[a] (5,6)	
SYNTAX-1 SKORU	0-22	54[a] (66,7)	66[a] (73,3)	
	23-32	15[a] (18,5)	19[a] (21,1)	0,143
	≥33	12[a] (14,8)	5[a](5,6)	

Table 1. In-hospital follow up

Conclusions: In cardiac arrest patients, advanced age, diabetes, out-of-hospital occurence of the event, failure to provide the necessary revascularization (unsuccessful PCI application and CABG decision but not being made), presence of left main coronary lesion or multi-vessels disease and low left ventricular ejection fraction after discharge have been shown to be factors that increase mortality.

		Uzun dönem takipte mortalite görülen hastalar (n=94)	Uzun dönem takipte sağ olan hastalar (n=77)	p değeri
Yaş (mean±sd)		61,1±11,4	53,5±13,2	**<0,001**
Erkek Cinsiyet		73 (77,7)	65 (84,4)	0,331
Sigara öyküsü		37 (39,4)	40 (51,9)	0,123
Hipertansiyon		40 (%42,6)	36 (%46,8)	0,644
Diyabetes mellitus		31 (%33)	18 (%23,4)	0,178
KAH ÖYKÜSÜ	YOK	66[a] (70,2)	57[a] (74)	
	NONOBSTRUKTİF KAH	8[a] (8,5)	7[a] (9,1)	0,294
	PKG ÖYKÜSÜ	20[a] (21,3)	11[a] (14,3)	
	CABG ÖYKÜSÜ	0[a] (0)	2[a] (2,6)	
CPA YERİ	HASTANE DIŞI	48[a] (51,1)	23[b] (29,9)	**0,008**
	HASTANE İÇİ	46[a] (48,9)	54[b] (70,1)	
MI TİPİ	NON- ST ELEVE MI	35[a] (%37,2)	22[a] (%28,6)	1,00
	ST ELEVE MI	59[a] (%62,8)	55[a] (%71,4)	
İŞLEM SONUCU	FARMAKOLOJİK TEDAVİ	14[a] (14,9)	16[a] (20,8)	
	BAŞARILI PKG	59[a](62,8)	57[a] (74)	**0,013**
	BAŞARISIZ PKG	12[a] (12,8)	3[b] (3,9)	
	CABG KARARI	9[a] (9,6)	1[b] (1,3)	
KÜLPRİT LEZYON VARLIĞI VE YERİ	NORMAL KORONER/NONKRİTİK DARLIK	6[a] (6,4)	11[a] (14,3)	
	LMCA	2[a] (2,1)	1[a] (1,3)	
	LAD	40[a] (42,6)	39[a] (50,6)	0,343
	CX	19[a] (20,2)	11[a] (14,3)	
	RCA	21[a] (22,3)	12[a] (15,6)	
	IMA/YÜKSEK OM	6[a] (6,4)	3[a] (3,9)	
SYNTAX-1 SKORU	0-22	62[a] (66)	58[a] (75,3)	
	23-32	19[a] (20,2)	15[a] (19,5)	0,154
	≥33	13[a] (13,8)	4[a] (5,2)	
		n=11	n=75	
UZUN DÖNEM TAKİPTE EF (%)	<40	8[a] (72,7)	17[b] (22,7)	**0,002**
	40-49	2[a] (18,2)	27[a] (36)	
	≥50	1[a] (9,1)	31[b] (41,3)	

Table 2. Long term follow up after hospitalization

		Anlamlı darlık yok	Tek damar hastalığı	İki damar hastalığı	Üç damar hastalığı	LMCA hastalığı	Toplam	p değeri
HASTANE İÇİ MORTALİTE	YOK n, (%)	12[a](70,6)	39[a] (63,9)	16[b] (40,0)	20[a,b] (50,0)	3[b] (23,1)	90 (52,6)	
	VAR n, (%)	5[a] (29,4)	22[a] (36,1)	24[b] (60,0)	20[a,b] (50,0)	10[b] (76,9)	81 (47,4)	**0,012**
TOPLAM	n, (%)	17 (100)	61 (100)	40 (100)	40 (100)	13 (100)	171 (100)	

Table 3. The relationship between the extension of coronary artery disease and in-hospital mortality

		Anlamlı darlık yok	Tek damar hastalığı	İki damar hastalığı	Üç damar hastalığı	LMCA hastalığı	Toplam	p değeri
UZUN DÖNEM TAKİPTE MORTALİTE	YOK n, (%)	11[a,b](64,7)	37[b](60,7)	12[a](30,0)	15[a,b] (37,5)	2[a](15,4)	77 (45,0)	
	VAR n, (%)	6[a,b](35,3)	24[b](39,3)	28[a](70,0)	25[a,b] (62,5)	11[a](84,6)	94 (55,0)	**0,001**
TOPLAM	n, (%)	17 (100)	61 (100)	40 (100)	40 (100)	13 (100)	171 (100)	

Table 4. The relationship between the extension of coronary artery disease and long term mortality

TEK DAMAR HASTALARI			LAD	CX	RCA	Toplam	P değeri
HASTANE İÇİ MORTALİTE	YOK	n, (%)	27ª(69,2)	6ª (66,7)	6ª(46,2)	39 (63,9)	
	VAR	n, (%)	12ª (30,8)	3ª (33,3)	7ª(53,8)	22 (36,1)	0,266
TOPLAM		n, (%)	39 (100)	9 (100)	13 (100)	61 (100)	

Table 5. Correlation of coronary artery with culprit lesion and in-hospital mortality in single vessel patients

TEK DAMAR HASTALARI			LAD	CX	RCA	Toplam	P değeri
UZUN DÖNEM TAKİPTE MORTALİTE	YOK	n, (%)	26ª(66,7)	5ª(55,6)	6ª(46,2)	37(60,7)	
	VAR	n, (%)	13ª (33,3)	4ª(44,4)	7ª(53,8)	24 (39,3)	0,304
TOPLAM		n, (%)	39 (100)	9 (100)	13 (100)	61 (100)	

Table 6. Correlation of coronary artery with culprit lesion and long term mortality in single vessel patients

Coronary artery disease / Acute coronary syndrome

OP-044

Outcomes in Coronary No-Reflow Phenomenon Patients and the Relationship between Kidney Injury Molecule-1 and Coronary No-Reflow Phenomenon

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Background and Aim: Coronary no-reflow phenomenon (CNP) is associated with an increased risk of major cardiovascular adverse events (MACE). This study aimed to evaluate the relationship between serum Kidney Injury Molecule-1 (KIM-1) levels and CNP in patients with acute ST-segment elevation myocardial infarction (STEMI).

Methods: This study included a total of 160 patients (113 males and 47 females; mean age: 61.65±12.14 years) who were diagnosed with STEMI. The patients were divided into two groups, the reflow group (RG) (n=140) and the no-reflow group (NRG) (n=20). Patients were followed during one year. A p-value of <0.05 was considered significant. This study included a total of 160 patients (113 males and 47 females; mean age: 61.65±12.14 years) who were diagnosed with STEMI. The patients were divided into two groups, the reflow group (RG) (n=140) and the no-reflow group (NRG) (n=20). Patients were followed during one year. A p-value of <0.05 was considered significant.

Results: CNP was observed in 12.50% of the patients. Serum KIM-1 was significantly higher in the NRG than in the RG (20.26±7.32 vs. 13.45±6.40, p<0.001). Body mass index (BMI) was significantly higher in the NRG than in the RG (29.41 (28.48-31.23) vs. 27.56 (25.44-31.03), p=0.047). Heart rate (HR) was significantly lower in the NRG than in the RG (61.6±8.04 vs. 80.37±14.61, p<0.001). The European System for Cardiac Operative Risk Evaluation II (EuroSCORE II) was significantly higher in the NRG than in the RG (3.06±2.22 vs. 2.36±2.85, p=0.016). The incidence of stroke was significantly higher in the NRG than in the RG (15% vs. 2.90%, p=0.013). The baseline KIM-1 level (OR=1.19, 95% CI:1.07 to 1.34, p=0.002) and HR (OR=0.784, 95% CI:0.69 to 0.88, p<0.001) were the independent predictors of CNP.

Conclusions: In conclusion, baseline serum KIM-1 concentrations and lower HR are independently associated with CNP in STEMI patients and the incidence of stroke was significantly higher in the NRG in the one-year follow-up.

Coronary artery disease / Acute coronary syndrome

OP-045

Can we use triglyceride glucose index to predict the presence of coronary slow flow in patients without diabetes?

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Background and Aim: Coronary slow flow (CSF) is characterized as the delay of distal vessel opacity due to an abnormal decrease in coronary flow velocity during angiography without significant epicardial coronary stenosis. CSF has been considered as a result of the complex process including microvascular dysfunction, inflammation, increased oxidative stress, small vessel disease, and diffuse atherosclerosis. In addition, there is growing evidence that insulin resistance (IR) may impair the microvascular circulation of coronary vessels even if in absence of overt diabetes. Triglyceride glucose (TyG) index derived from fasting glucose and triglycerides, is a novel reliable marker used in evaluation of IR. Recent studies reported the relationship between TyG index with hypertension, diabetes, MetS, arterial stifness, carotid atherosclerosis, CAD and adverse cardiovascular outcomes. However there is no scant data the role of the TyG index on CSF development. Hence, in this study, we aimed to reveale the relationship between TyG index and CSF and to evaluate whether the TyG index may be a useful marker for predicting CSF in patients without diabetes.

Methods: In this study, we retrospectively analyzed 94 patients with CSF and 109 patients with normal coronary flow (CNF), who underwent coronary angiography because of angina pectoris or equivalent symptoms or/ and abnormal non-invasive stress test. Demographic and clinical characteristics of the study population were obtained from the hospital registry system. Insulin resistance (IR) was evaluated via TyG index using by the formula ln[fasting triglycerides (mg/dL)×fasting blood glucose (mg/dL)/2]. The thrombolysis in myocardial infarction (TIMI) frame count was calculated and the patients were divided into two groups according to CSF presence.

Results: Patients with CSF had a higher TyG index compared to those without (9.01±0.43 vs. 8.53±0.48, p<0.001). TyG index was significantly correlated with mean TIMI frame count (r=0.682, p<0.001). In multivariate logistic regression analysis, TyG index was an independent predictor for CSF presence (OR= 3.364, 95% CI= 3.231-11.650, p<0.001). The ROC curve analysis revealed

the best cut off value of the TyG index as 8.57 with 87% sensitivity and 58% specificity in predicting of CSF and the area under the curve (AUC) was 0.769. (95% CI= 0.705-0.833, p<0.001).

Conclusions: This study showed that CSF patients had a significantly higher TyG index regardless of all causes, and the use of the TyG index can be considered in predicting the presence of CSF.

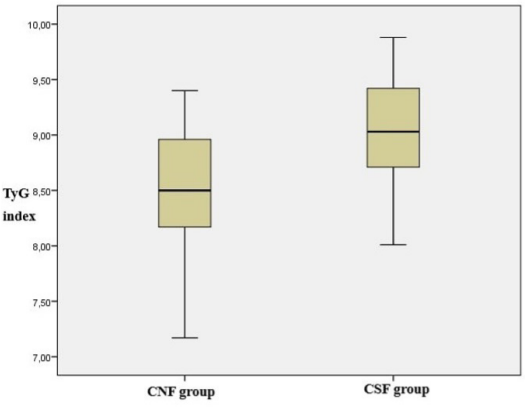

Figure 1. The comparison of TyG index between the groups
TyG index, triglyceride glucose index; CNF, coronary normal flow; CSF, coronary slow flow

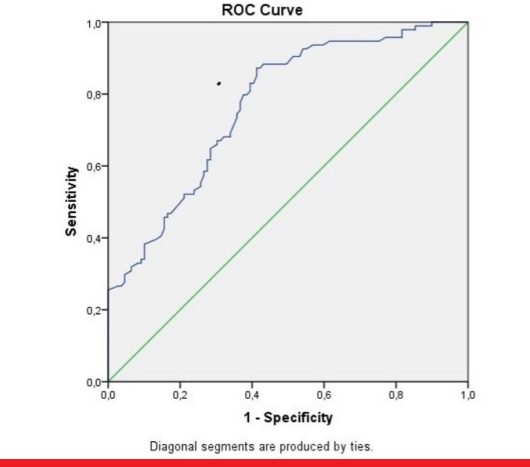

Figure 2. The receiver operating characteristic (ROC) curve of TyG index for detecting coronary slow flow

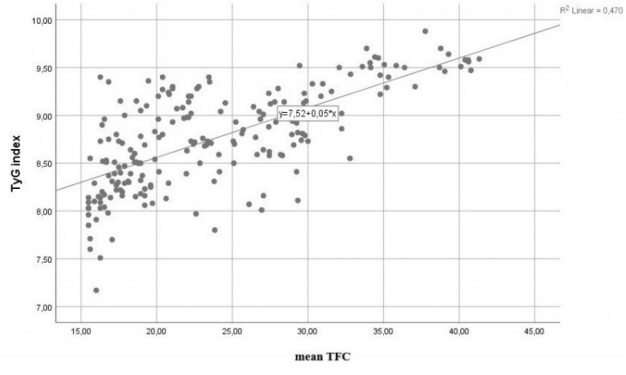

Figure 3. The relationship between TyG index and mean TFC

Variables	CNF group (n=109)	CSF group (n=94)	*p*-value
Demographic parameters			
Age (years)	56.92±10.94	54.88±11.83	0.205
Male gender, n (%)	65 (59.6)	60 (63.8)	0.540
Hypertension, n (%)	57 (52.2)	43 (45.7)	0.352
Hyperlipidemia, n (%)	23 (21.1)	16 (17)	0.462
Smoking, n (%)	39 (35.7)	37 (39.3)	0.599
Ejection fraction (%)	58.71±3.13	58.72±3.78	0.969
Laboratory parameters			
Fasting glucose, mg/dL	84.24±9.76	96.38±9.49	<0.001
HbA1c (%)	5.34±0.46	5.94±0.41	<0.001
Creatinine, mg/dL	0.79±0.13	0.85±0.15	0.007
TG, mg/dL	137.04±56.60	178.85±56.05	<0.001
Tchol, mg/dL	185.61±42.82	190.02±37.63	0.439
LDL-C, mg/dL	112.28±34.85	113.51±34.29	0.800
HDL-C, mg/dL	47.02±10.56	37.47±6.58	<0.001
Hemoglobin, g/ dL (IQR)	14 (13.05-15.15)	14 (12.55-15.12)	0.739
WBC, cells/μL (IQR)	8.04 (6.78-9.71)	9.03 (8.09-10.72)	<0.001
CRP, mg/dL (IQR)	0.4 (0.25-0.61)	0.63 (0.39-0.9)	<0.001
TG/HDL-C	3.14±1.65	4.81±1.81	<0.001
TyG index	8.53±0.48	9.01±0.43	<0.001
Medications			
RAS blocker, n (%)	41(37.6)	31(32.9)	0.491
CCB, n (%)	27 (24.7)	11(11.7)	0.057
Diuretics, n (%)	9 (8.2)	11 (11.7)	0.411
Statin, n (%)	18(16.5)	12 (12.7)	0.451

Table 1. Basic demographic and clinical data of the study population
CNF, coronary normal flow; CSF, coronary slow flow;TG, triglycerides; TChol; total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density, lipoprotein cholesterol; WBC, white blood cells; CRP, C-reactive protein; TyG index, triglyceride glucose index; LAD, left anterior descending artery; LCx, left circumflex artery; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction; TFC, "thrombolysis in myocardial infarction" frame count; RAS, Renin-angiotensin system; CCB, calcium channel blockers; IQR, Interquartile range

Variables	CNF group (n=109)	CSF group (n=94)	*p*-value
Corrected TIMI frame count			
LAD	19.44±2.07	30.58±0.72	<0.001
LCx	18.33±3.56	28.25±8.66	<0.001
RCA	18.18±3.40	30.23±8.58	<0.001
Mean TFC	18.61±2.31	29.79±5.30	<0.001
Slow flow related to coronary artery, n (%)			
LAD	-	65 (69.1)	-
LCx	-	42 (44.7)	-
RCA	-	57 (60.6)	-
Number of vessels with CSF, n (%)			
Single vessel, n (%)	-	43 (45.7)	-
Two or three vessels, n (%)	-	51 (54.3)	-

Table 2. Angiographic data of the study population
CNF, coronary normal flow; CSF, coronary slow flow; LAD, left anterior descending artery; LCx, left circumflex artery; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction; TFC: "thrombolysis in myocardial infarction" frame count

Table 3. Univariate and multivariate logistic regression analysis to determine the independent predictors of coronary slow flow

Variables	OR	95% CI	P-value	OR	95% CI	*P*-value
Men	1.195	0.676-2.110	0.540	-	-	-
Age	0.984	0.960-1.009	0.204	-	-	-
Hypertension	0.769	0.442-1.337	0.352	-	-	-
Hyperlipidemia	0.767	0.378-1.557	0.463	-	-	-
Smoking	1.165	0.659-2.060	0.599	-	-	-
Ejection fraction	1.001	0.923-1.084	0.987	-	-	-
Creatinine	9.296	1.966- 63.528	0.009	1.246	0.100- 15.567	0.864
WBC	1.345	1.160-1.561	<0.001	1.097	0.945-1.274	0.222
LDL-C	1.001	0.993-1.009	0.799	-	-	-
CRP	5.627	2.413-13.123	<0.001	1.231	0.428-3.539	0.699
TG/HDL-C	1.711	0.850-0.925	<0.001	0.919	0.875-0.964	<0.001
TyG index	4.255	2.131-12.901	<0.001	3.364	3.231-11.650	<0.001

OR, odd ratio; CI, confidence interval; WBC, white blood cells; LDL-C, low-density lipoprotein cholesterol; CRP, C-reactive protein; HDL-C, high-density lipoprotein cholesterol; TyG index, triglyceride glucose index

Coronary artery disease / Acute coronary syndrome

OP-046

The lipid ratios and lipid levels are associated with the combination of *NOS3* and *APOE* gene polymorphisms in coronary artery disease

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Background and Aim: Coronary artery disease (CAD) is a multifactorial disease and CAD along with its complications is the leading cause of death worldwide. In this study, we aimed to examine whether *APOE* and *NOS3* gene polymorphisms have contributions to CAD and its major risk factors.

Methods: The blood samples of the recruited 461 CAD patients [≥50 stenosis ≥1 vessel(s)] and 342 non-CAD controls (≤30 stenosis) who underwent coronary angiography were collected before the procedure and the levels of serum lipids were assessed. The DNA samples isolated from leukocytes and individuals were genotyped for *NOS3* (rs1799983 G/T, also known as G894T) and *APOE* (rs429358 and rs7412) gene polymorphisms using hydrolysis probes and the Real-Time PCR method. Individuals were divided into four groups according to allele carriage status of *NOS3* and *APOE* gene polymorphisms. The results were statistically evaluated.

Results: In the analysis in which the combinational effect of *APOE* and *NOS3* polymorphisms was assessed, it was observed that carriers of minor (T) allele of *NOS3* in addition to ε4 allele had lower LDL-C/HDL-C ratios compared with ε4 allele carrier [ε4(+)] *NOS3* G homozygotes (p=0.050) in the non-CAD group. In the CAD group, carriers of the *NOS3* T allele combined with the ε4 allele also had lower total cholesterol (p=0.049), and LDL-C (p=0.041) levels compared with the ε4(+) *NOS3* G homozygotes. In addition, ε4(-) *NOS3* G homozygotes had a higher body mass index than ε4(-) *NOS3* T allele carriers (p=0.039). Besides, in the non-CAD group, ε2(+) *NOS3* T allele carriers had lower LDL-C (p=0.031), total cholesterol/HDL-C (p=0.033), and LDL-C/HDL-C ratios (p=0.008) when compared with ε2(-) *NOS3* G homozygotes.

Conclusions: The *NOS3* G894T gene polymorphism was associated with serum lipid levels, ratios of the lipids, and body mass index in CAD and non-CAD groups when combined with both ε2 and ε4 alleles.

Coronary artery disease / Acute coronary syndrome

OP-047

Effect of logistics clinical syntax score on long-term clinical results in coronary bifurcation lesions with double stent applied

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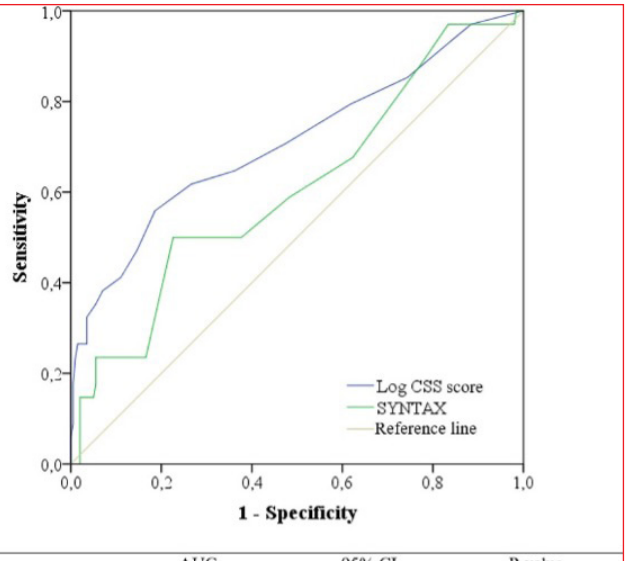
Background and Aim: The logistics Clinical SYNTAX score (log CSS), developed by combining clinical variables with the anatomical SYNTAX score (SS), has been shown to perform better than the SS alone in predicting 1-year outcomes after percutaneous coronary intervention (PCI). However, the long-term survival and ability of this score to predict significant adverse cardiovascular events are unknown. The aim of this study is to evaluate whether log CSS is associated with the development of major adverse cardiovascular events (MACE) in patients undergoing PCI in coronary bifurcation lesions.

Methods: A total of 233 patients with acute coronary syndrome who underwent PCI between May 2011 and October 2019 were retrospectively enrolled. Anatomical SS and log CSS score were calculated. The patients were divided into two groups according to the cut-off value determined by ROC analysis. Patients with log CSS value> 6 were defined as high log CSS group (n=56) and those with log CSS value ≤6 were defined as low log CSS group (n=177). Log CSS's ability to predict 2-year MACE was evaluated.

Results: MACE occured in 33.9% of patients with high log CSS, 8.5% of patients with low log CSS, and 14.4% of the whole patient group (p<0.001). In the Kaplan - Meier survival analysis, the long-term survival of patients with high log CSS was significantly lower than those in the low log CSS group (log rank p<0.001). In multivariate COX analyzes, high Log CCS (OR, 3.781; 95% CI, 1.706 - 8.377; p=0.001) was the strongest independent predictor of MACE. Log CSS was significantly better at predicting 2-year MACE development compared to the anatomical SS (log CSS, AUC: 0.710 (95% CI, 0.603 - 0.817), p<0.001 and SS, AUC: 0.610 (95% CI). 0.503-0.717), p=0.040).

Conclusions: Log CSS combining clinical characteristics with the anatomic SS substantially better predict 2-year mortality than the SS alone. Log CSS has emerged as the strongest independent predictor of MACE and should be used for long-term risk stratification of patients undergoing PCI.



	AUC	95% CI	P-value
Log CSS	0.710	0.603-0.817	<0.001
SYNTAX score	0.610	0.503-0.717	0.040

Figure 1. Long-term risk prediction with ROC curve of Log CCS and SS

Table 1. Comparison of characteristics of study patients with Log CSS

	Total n=233	Log CSS ≤ 6 n=177	Log CSS > 6 n=56	*P*-value
Age, years	59.5±11.1	56.0±8.9	70.3±10.4	<0.001
Male, n (%)	181 (77.7)	143 (80.8)	38 (67.9)	0.043
Diabetes mellitus, n (%)	90 (38.6)	57 (32.2)	33 (58.9)	<0.001
Hypertension, n (%)	109 (47.0)	74 (42.0)	35 (62.5)	0.008
Hyperlipidemia, n (%)	102 (43.8)	82 (46.3)	20 (35.7)	0.163
Prior MI, n (%)	77 (33.0)	54 (30.5)	23 (41.1)	0.143
Prior PCI, n (%)	72 (30.9)	52 (29.4)	20 (35.7)	0.371
Peripheral artery disease, n (%)	16 (6.9)	8 (4.5)	8 (14.3)	0.028

Table 1. Comparison of characteristics of study patients with Log CSS (Continued)

	Total n=233	Log CSS ≤ 6 n=177	Log CSS > 6 n=56	*P*-value
Carotid artery disease, n (%)	13 (5.6)	5 (2.8)	8 (14.3)	0.003
Cerebrovascular disease, n (%)	1 (0.4)	0 (0.0)	1 (1.8)	0.240
Chronic kidney disease, n (%)	2 (0.9)	2 (1.1)	0 (0.0)	1.0
Atrial fibrillation, n (%)	8 (3.4)	4 (2.3)	4 (7.1)	0.097
LVEF, %	52.8±10.1	56.5±6.8	40.9±9.7	<0.001
Laboratory data				
Hematocrit (%)	40.6±5.3	41.2±4.9	38.6±5.8	0.002
White blood cells (10^3/uL)	9.55±2.89	9.64±2.89	9.29±2.90	0.432
Platelet (10^3/uL)	260±62	263±58	250±73	0.173
Creatinine (mg/dL)	0.86 (0.72-0.97)	0.85 (0.71-0.95)	0.89 (0.79-1.01)	0.027
GFR, ml/min	95.7±25.4	99.3±24.3	84.3±25.8	<0.001
Total cholesterol (mg/dL)	189±50	191±49	182±51	0.216
LDL-C (mg/dL)	107 (84-133)	109 (89-134)	107 (67-128)	0.311
HDL-C (mg/dL)	44±11	43±10	46±12	0.193
Triglyceride (mg/dL)	152 (100-230)	162 (102-246)	145 (96-183)	0.182
SYNTAX score	15.7±4.6	14.6±3.6	18.9±5.8	<0.001
Bifurcation location				
LAD-CX, n (%)	16 (6.9)	3 (1.7)	13 (23.2)	
LAD-Diagonal, n (%)	135 (57.9)	103 (58.2)	32 (57.1)	
CX-OM, n (%)	74 (31.8)	63 (35.6)	11 (19.6)	
PDA-PLA, n (%)	8 (7.0)	8 (4.5)	0 (0.0)	

Table 2. Comparison of characteristics of study patients with Log CSS

	Total N = 233	Log CSS ≤ 6 n = 177	Log CSS > 6 n = 56	*P*-value
Stenting strategy				0.185
T-stenting, n (%)	93 (39.9)	76 (42.9)	17 (30.4)	
Culotte stenting, n (%)	57 (24.5)	43 (24.3)	14 (25.0)	
Crush stenting, n (%)	83 (35.6)	58 (32.8)	25 (44.6)	
Main branch lesion length (mm)	19.9±6.5	20.0±6.7	19.7±5.7	0.813
Side branch lesion length (mm)	12.6±5.6	12.5±5.4	12.9±6.2	0.715
Main branch vessel diameter (mm)	2.88±0.29	2.86±0.27	2.95±0.33	0.043
Side branch vessel diameter (mm)	2.61±0.23	2.59±0.22	2.66±0.25	0.044

Table 2. Comparison of characteristics of study patients with Log CSS (Continued)

	Total N = 233	Log CSS ≤ 6 n = 177	Log CSS > 6 n = 56	*P*-value
Main branch stent length (mm)	25.3±6.5	25.4±6.7	25.0±6.0	0.631
Side branch stent length (mm)	19.3±5.0	19.4±5.1	19.0±4.8	0.674
Main branch stent size (mm)	2.88±0.29	2.86±0.27	2.95±0.33	0.040
Side branch stent size (mm)	2.61±0.23	2.60±0.23	2.66±0.25	0.066
Predilation, n (%)	188 (80.7)	140 (79.1)	48 (85.7)	0.274
POT, n (%)	197 (84.5)	152 (85.9)	45 (80.4)	0.319
Final kissing, n (%)	225 (96.6)	171 (96.6)	54 (96.4)	1.0
Procedural time (min)	70 (55-93)	68 (55-90)	75 (55-104)	0.363
Fluoroscopy time (min)	23 (18-35)	23 (17-33)	28 (18-39)	0.116
Contrast volume (mL)	329±109	323±106	347±119	0.154

Data are presented as percentage, mean ± standard deviation or median (interquartile range). CSS: Clinical syntax score, MI: Myocardial infarction, PCI: Percutaneous coronary intervention, LVEF: Left ventricular ejection fraction, GFR: Glomerular filtration rate, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, LAD: Left anterior descending artery, LCx: Left circumflex artery, OM: Obtuse marginal artery, PDA: Posterior descending artery, PLA: Posterolateral artery, POT: Proximal optimisation technique.

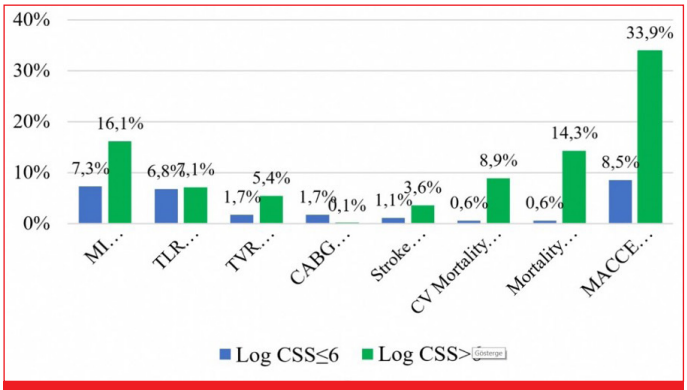


Figure 2. Comparison of 2-year clinical outcomes of high and low log CSS groups.

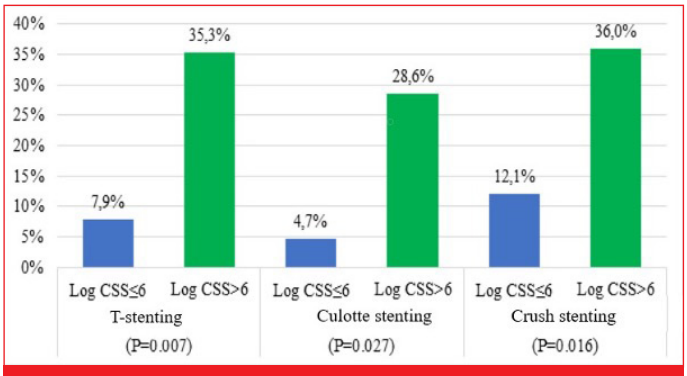


Figure 3. Comparison of 2-year clinical outcomes of high and low log CSS groups in different stent strategies

Table 3. Univariable and multivariable logistic regression analyzes for the development of MACE

	Univariate analiz	Uni-variate analiz	Multivariate analiz	Multi-variate analiz
	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
Prior PCI	1.864 (0.946-3.676)	0.072	1.966 (0.954-4.050)	0.067
Cerebro-vascular disease	13.650 (1.810-103.0)	0.011	7.401 (0.771-71.041)	0.083
Triglyceride	1.002 (1.000-1.004)	0.095	1.002 (1.000-1.004)	0.131
Log CSS>6	5.102 (2.582-10.079)	<0.001	3.781 (1.706-8.377)	0.001
Bifurcation location		0.019		0.144
LAD-Diagonal*	0.386 (0.156-0.952)	0.039	1.826 (0.564-5.912)	0.315
CX-OM*	0.148 (0.045-0.485)	0.002	0.575 (0.138-2.390)	0.446
PDA-PLA*	<0.001 (<0.001->1000)	0.974	<0.001 (<0.001->1000)	0.976
POT	0.534 (0.254-1.120)	0.097	0.586 (0.256-1.343)	0.207
Final kissing	0.314 (0.096-1.032)	0.056	0.241 (0.059-0.991)	0.049
Contrast volume	1.003 (1.000-1.006)	0.034	1.003 (1.000-1.006)	0.054

HR: hazard oranı, CI: güven aralığı, CSS: Clinical syntax score, PCI: Percutaneous coronary intervention, LAD: Left anterior descending artery, LCx: Left circumflex artery, OM: Obtuse marginal artery, PDA: Posterior descending artery, PLA: Posterolateral artery, POT: Proximal optimisation technique. * Compared to LAD-LCx. * Compared to T-stenting.

Coronary artery disease / Acute coronary syndrome

OP-048

In-hospital outcomes and 1-year mortality after myocardial infarction in diabetic patients: Nationwide data from TURKMI registry

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On behalf of the TURKMI registry investigators

Background and Aim: Diabetes mellitus (DM) is an important prognostic factor for patients with myocardial infarction (MI). However, there is no up-to-date information in Turkey regarding the outcomes in diabetic patients after MI. We present the results of the TURKMI registry.

Methods: TURKMI registry enrolled 1930 patients with acute MI (61.7% with NSTEMI and 37.8% with STEMI) in a 15-day time window from 50 centres representing the Turkeys' adult age distribution. In this study, we compared in hospital mortality, in-hospital major

adverse cardiovascular and cerebrovascular events (MACCE), and 1-year mortality in diabetic and non-diabetic patients. MACCE was defined as a composite of in-hospital death, stroke, stent thrombosis, and heart failure or cardiogenic shock. Multivariable logistic regression and Cox-regression analyses were used for analysis of in-hospital and 1-year outcome, respectively.

Results: There were 654 (33.9%) patients with DM and 1276 (66.1%) without DM. Baseline characteristics are presented in the Table. In-hospital mortality and MACCE was significantly higher in diabetic patients (in-hospital mortality 35 [5.4%] vs. 40 [3.1%], p=0.017; and MACCE 52 [8.0%] vs. 67 [5.3%], p=0.020). Age, sex, and diagnosis (STEMI and NSTEMI) adjusted risk of in-hospital mortality (Odds ratio and 95% CI 1.74 [1.07-2.83], p=0.025) and MACCE (Odds ratio 1.59 [1.07-2.37], p=0.021) was significantly higher in diabetics. At 1-year, 81 (12.4%) diabetic and 93 (7.3%) non-diabetic patients died (log-rank p<0.001, Figure). The risk of mortality at 1 year (adjusted for age, gender, history of hypertension, smoking, MI/CABG/PCI, renal failure, aspirin, and statin use) was significantly higher in diabetics (Hazard ratio 1.57 [1.14-2.17], p=0.006). No significant heterogeneity in the risk between STEMI and NSTEMI patients was observed for in-hospital mortality, MACCE and mortality at 1 year (p-values for interaction 0.167, 0.286, and 0.877, respectively).

Conclusions: The risk of MACCE and mortality after MI is increased by 60-70% in diabetics, therefore strict measures for primary and secondary prevention should be taken.

Table 1. Baseline characteristics

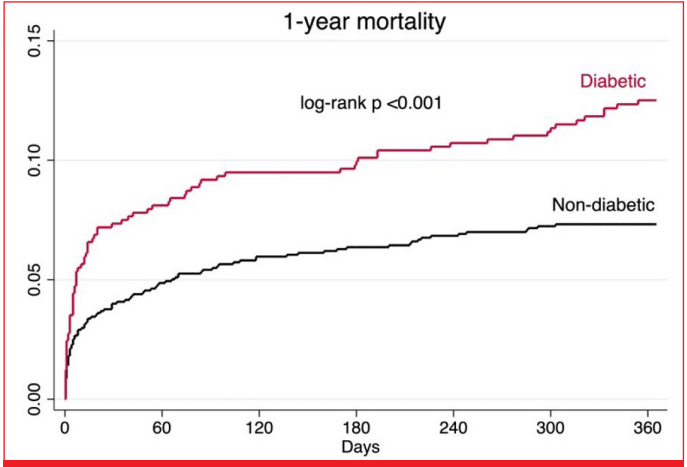

Figure 1. Kaplan-Meier analysis for 1-year mortality

	Non-Diabetics n= 1276	Diabetics n=654	*P*-value
Diagnosis, n (%)			
NSTEMI	747 (58.5)	448 (68.5)	
STEMI	529 (41.5)	206 (31.5)	<0.001
Age, mean ± SD	60.5±14.0	64.8±11.1	<0.001
Female, n (%)	246 (19.3)	258 (39.4)	<0.001
History of MI/CABG/PCI, n (%)	292 (22.9)	258 (39.5)	<0.001
Hypertension, n (%)	491 (38.5)	464 (70.9)	<0.001
Smoking, n(%)	711 (55.7)	231 (35.3)	<0.001
Hyperlipidemia (self report), n (%)	103 (8.1)	130 (19.9)	<0.001
Renal failure, n (%)	50 (3.9)	53 (8.1)	<0.001
Acetylsalicylic acid use, n (%)	284 (24.2)	250 (40.5)	<0.001
Statin use, n (%)	95 (7.4)	109 (16.7)	<0.001
LDL cholesterol, mg/dl, median (IQR)	122 (98-152)	115 (86-142)	<0.001
HDL cholesterol, mg/dl, mean ± SD	41.5±10.0	40.8±11.2	0.270
Triglyceride cholesterol, mg/dl, median (IQR)	126 (84-190)	144 (100-219)	<0.001
Glucose mg/dl, mean ± SD	113.6±41.4	169.4±73.6	<0.001

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-049

Effect of SGLT-2 inhibitors on P wave indices and atrial electromechanics in type 2 diabetes mellitus patients

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Background and Aim: Atrial fibrillation (AF) is one of the leading causes of thromboembolic complications. Increase in P wave indices and atrial electromechanical coupling intervals are precursors of AF. Patients with diabetes mellitus (DM) tend to have an increased risk for the development of AF. Although the precise mechanism is not clear, sodium-glucose co-transporter 2 (SGLT-2) inhibitors have been shown to reduce the risk of AF occurrence in patients with DM. In this study, we analysed the effect of SGLT-2 inhibitors on P wave indices and atrial electromechanics in type 2 DM patients

Methods: 157 metformin monotherapy and 135 metformin+SGLT-2 inhibitor combination therapy patients included. Electrocardiographic recordings and standard transthoracic echocardiographic data of all patients were obtained. P wave dispersion (PWD), P terminal force in V1 (PTFV1), atrial electromechanical coupling intervals were measured and compared (Fig. 1).

Results: All demographical, clinical, and biochemical characteristics of the 2 groups are presented in detail in Table 1. Both groups were similar in terms of age, gender, BMI, BSA, smoking history, dyslipidemia, metformin dosage, and time on DM treatment. P wave indices, echocardiographic and atrial electromechanical data of both groups are given in Table 2. Althoguh PWD (61.28±12.79 vs. 54.80±11.87, p<0.0001), PTFV1 (32.01±6.74 vs. 25.81±5.58, p<0.0001), LAVI (32.33±7.31 vs. 26.84±5.51, p<0.0001), LEMD (27.61±8.50 vs. 22.64±8.41, p<0.0001), REMD (30.65±8.05 vs. 26.85±7.91, p<0.0001), IEMD (35.96±6.30 vs.

33.03±6.46, p<0.0001) were significantly lower in combination therapy group (Fig. 2), there were no statistically significant difference between Empagliflozin and Dapagliflozin subgroups interms of mentioned parameters (Fig. 3). There were positive correlation between PWD and LAVI (r=0.402, p<0.0001), LAVI and PTFV1 (r=0.685, p<0.0001), LEMD, REMD, IEMD and LAVI (r=0.308, p<0.0001; r=0.310, p<0.001; r=0.189, p<0.001 respectively), LA diameter and LAVI (r=0.152, p=0.025)

Conclusions: SGLT-2 inhibitors significantly improves P wave indices and atrial electromechanics in type 2 DM patients. This effect seems to be a group effect rather than molecule effect.

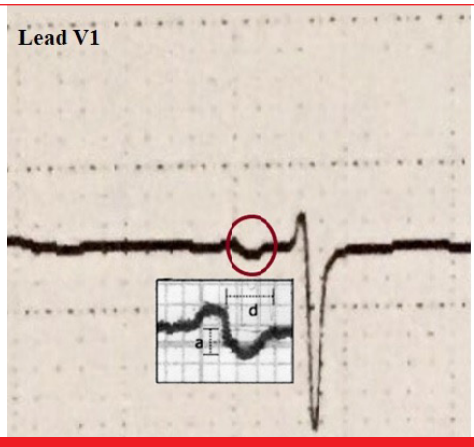

Figure 1. P terminal force defined as the duration (in miliseconds) of the terminal part (negative) of the P wave in lead V1 multiplied for its depth (in milimeters)

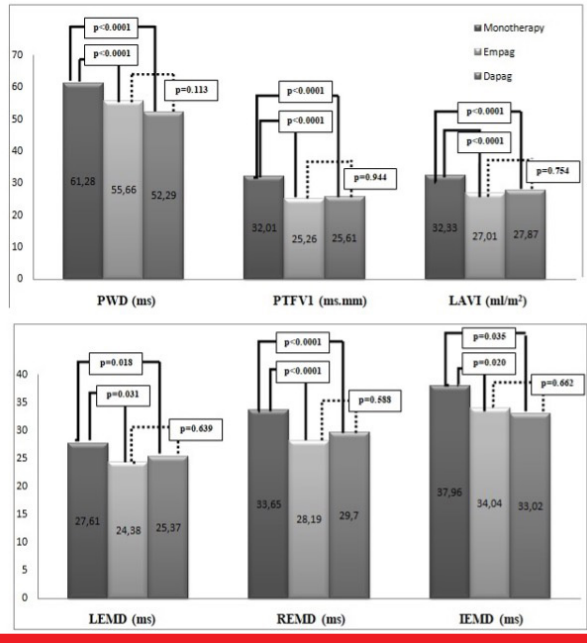

Figure 2. Comparison of monotherapy, Empag and Dapag groups interms of P wave indices and atrial electromechanics (PTFV1: P terminal force in V1, LAVI: Left atrial volume index, PWD: P wave dispersion, LEMD: Left sided intra atrial electromechanical delay, REMD: Right sided intra atrial electromechanical delay, IEMD: Interatrial electromechanical delay)

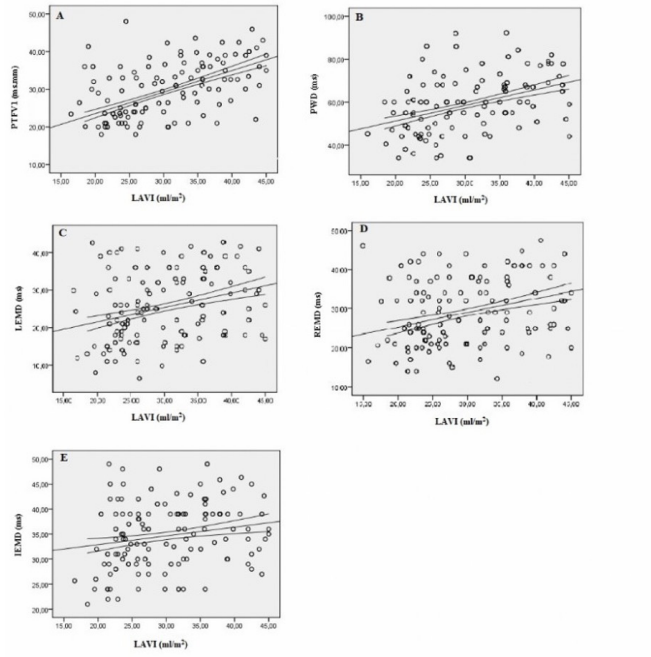

Figure 3. Graphs A-Showing the correlation between PTFV1 and LAVI, B-Showing the correlation between PWD and LAVI. C-Showing the correlation between LEMD and LAVI. D-Showing the correlation between REMD and LAVI. E- Showing the correlation between IEMD and LAVI. (PTFV1: P terminal force in V1, LAVI: Left atrial volume index, PWD: P wave dispersion, LEMD: Left sided intra atrial electromechanical delay, REMD: Right sided intra atrial electromechanical delay, IEMD: Interatrial electromechanical delay)

Table 1.

Variables	Monotherapy (Metformin) group; n=157	Combination therapy (Metformin+SGLT-2 inhibitor) group; n=135	*P*-value
Age (years)	62.05±5.78	61.35±6.28	0.322
Gender (male n/%)	85/54.14	82/60.74	0.271
BMI (kg/m²)	29.15±2.57	27.91±2.83	0.067
BSA (m²)	1.91±0.14	1.88±0.12	0,078
Metformin dosage (mg/day)	1774.74±373.39	1835.46±274.97	0.427
Time on diabetes mellitus treatment (months)	58.96±16.44	55.01±11.76	0.121
Hypertension (n/%)	90/57.32	95/70.37	0.028
Smoking (n/%)	53/33.75	58/42.96	0.118
Dyslipidemia (n/%)	97/61.78	91/67.40	0.069
Fasting blood Glucose (mg/dL)	136.76±37.26	144.10±32.04	0.032
HBA1C (%)	6.75±1.03	7.10±1.36	0.016
Total Cholesterol (mg/dL)	206.00±41.42	189.92±38.03	<0.001
LDL (mg/dL)	120.80±35.18	111.84±33.29	0.031
HDL (mg/dL)	40.27±8.22	45.83±11.74	<0.001

Table 1. (Continued)

Variables	Monotherapy (Metformin) group; n=157	Combination therapy (Metformin+SGLT-2 inhibitor) group; n=135	*P*-value
Triglyceride (mg/dL)	189.78±83.70	165.84±93.66	<0.001
BUN (mg/dL)	19.10±5.34	19.84±6.37	0.300
Creatinine (mg/dL)	0.98±0.18	0.96±0.20	0.495
Sodium (mEq/L)	139.46±3.12	140.20±2.31	0.712
Potasium (mEq/L)	4.33±0.39	4.46±0.42	0.837
AST (mg/dL)	27.62±10.94	29.08±7.37	0.546
ALT (mg/dL)	27.74±7.98	26.77±8.98	0.098
Hemoglobin (g/dL)	12.84±1.22	12.44±1.88	0.341
WBC (109/l)	7.18±1.32	7.68±1.89	0.592
PLT (109/l)	238.87±34,98	241.76±21.87	0.084

Demographical, clinical and biochemical characteristics of monotherapy and combination therapy groups. (BMI: Body mass index, BSA: Body surface area, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, BUN: Blood urea nitrogen, AST: Aspartate amino transferase, ALT: Alanine amino transferase, WBC: White blood cell, PLT: Platelet)

Table 2.

Variables	Monotherapy (Metformin) group; n=157	Combination therapy (Metformin+SGLT-2 inhibitor) group; n=135	*P*-value
Heart rate (bpm)	78.34±7.63	79.54±7.83	0.187
P maximum (ms)	105.62±11.60	100.13±10.73	0.029
P minimum (ms)	43.38±11.15	46.15±10.29	0.060
PWD (ms)	61.28±12.79	54.80±11.87	<0.0001
PTFV1 (ms.mm)	32.01±6.74	25.81±5.58	<0.0001
LAVI (ml/m^2)	32.33±7.31	26.84±5.51	<0.0001
LA diameter (mm)	37.51±3.99	36.99±4.12	0.145
LA volume (ml)	46.18±5.91	44.59±8.95	0.042
LVEF (%)	56.34±5.51	55.67±7.83	0.792
Left PA	75.14±12.19	67.90±13.17	<0.0001
Septal PA	41.31±8.46	35.96±8.14	<0.0001
Right PA	30.51±4.76	27.90±6.24	<0.001
LEMD (ms)	27.61±8.50	22.64±7.51	<0.0001
REMD (ms)	33.65±8.05	26.85±7.91	<0.0001
IEMD (ms)	37.96±6.30	33.03±6.46	<0.0001

P wave indices and atrial electromechanics' data of both groups (PWD: P wave dispersion, PTFV1: P terminal force in V1, LAVI: Left atrial volume index, LA: Left atrium, LVEF: Left ventricular ejection fraction, LEMD: Left sided intra atrial electromechanical delay, REMD: Right-sided intra atrial electromechanical delay, IEMD: Interatrial electromechanical delay)

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-050

Increased sympathetic activity might be associeted with increased tendency for ventricular tachycardia progression in patients with frequent outflow tract ventricular premature complexes

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Background and Aim: The aim of this study is to evaluate the predictive value of sympathovagal balance for idiopathic monomorfic outflow tract ventricular tachycardia (VT) occurrence in patients with frequent idiopathic outflow tract premature ventricular complexes (PVCs).

Methods: A total of 125 patients who had had undergone outflow tract PVC ablation between 01 august 2019 and 01 august 2021 constituted our study population. Patients with isolated outflow tract PVC without any inducible VT at electrophysiologic study and without any VT on Holter telemetry were classified as isolated PVC group. Patients with any episode of VT that has same morphology with outflow tract PVC were classified as VT group. Heart rate variability parameters calculated and compared between groups.

Results: There were 56 patients with isolated PVC and 69 patients with VT. PNN50 and HF values were significantly lower and LF/HF ratio values were significantly higher in patients with VT episodes compared to patients with isolated PVC.

Conclusions: These findings show that patients with frequent PVC with VT epizodes had decreased heart rate variability parameters and increased LF/HF ratio compared to patients with isolated frequent PVC. increased LF/HF ratio is assume to be a marker of impaired symphatovagal balance in faver of increased symphatetic activity hence symphatetic overactivity mihgt be involved in progression to VT in patients with frequent idiopathic outflow tract PVCs.

Table 1. Demographic and clinical characteristics of patients who had frequent idiopathic isolated PVC and PVC with VT episodes

Variables	Isolated PVC (n=56)	PVC with VT episodes (n=69)	*P*-value
Age	48±14	52±14	0.129
EF	65 (56-65)	65 (53-65)	0.172
Male sex	31 (54.4%)	37(45.6%)	0.510
Max HR	124±19	124±21	0.945
Mean HR	74±9	76±8	0.109
Min HR	47±7	46±8	0.649
SDNN24	118 (103-149)	115 (91-148)	0.127
SDANN index	112 (82-133)	102 (89-149)	0.171
SDNN index	54 (46-63)	47 (39-61)	0.085
RMSSD	33 (27-45)	27 (21-45)	0.083
PNN50	11 (5-17)	5 (2-15)	0.025
VLF	1748 (1251-2813)	1959 (1068-2676)	0.515
LF	517 (286-714)	400 (201-792)	0.388
HF	150 (91-221)	83 (42-224)	0.008
LF/HF ratio	3.0 (2.2-4.4)	4.6 (2.8-6.7)	0.004

PVC; premature ventricular complex, VT; ventricular tachycardia, EF;left ventricle ejection fraction, HR; heart rate, SDNN24; standard deviation of NN intervals, SDANN; standard deviation of the average NN intervals, RMSSD; square root of the mean of the squares of the successive differences between adjacent NNs, pNN50; proportion of NN50 divided by total number of NNs, VLF; very low frequency, LF; low frequency, HF; high frequency, Data are presented as mean ± SD, median (1st - 3rd quartiles), or number of patients (%)

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-051

Serum Elabela level is associated with left atrial function in non-cardioembolic stroke patients

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Background and Aim: Apelin is known to play a critical role in cardiac development and function during the embryonic period. Elabela is a recently found endogenous peptide ligand of the Apelin-APJ receptor pathway. Cardiovascular causes occupy a considerable place in patients with ischemic stroke. The etiology of many patients with stoke can not be clarified and they are called non-cardioembolic stroke. It is known that patients with non-cardioembolic stroke have impaired atrial functions. Elabela was found to be increased in patients with complete atrioventricular block. In this study, we aimed to evaluate the relationship between Elabela and electrical atrial-atrial conduction in non-cardioembolic stroke patients.

Methods: Our prospective cross-sectional observational study included 75 consecutive patients [age 72.24 (58-90); female gender (38.7%)] who had ischemic stroke. Atrial fibrillation (according to medication report, electrocardiogram and 48-hour rhythm holter monitoring), intracardiac thrombus, hemodynamic instability, uncontrolled hypertension, posterior circulation system stroke, heart failure with decreased ejection fraction (EF ≤39%), severe valvular disease, end stage liver and renal failure, existing infectious or autoimmune disease, malignancy and a history of previous coronary artery disease and intracranial events with all subjects were excluded from the study. All participants were divided into two groups as with and without interatrial block (IAB) according to 12-lead electrocardiogram. 19 participants had IAB (p wave duration >120 ms) and 56 participants had no IAB. The demographic, echocardiography parameters, routine blood tests, and serum Elabela levels of the groups were compared.

Results: There was no significant difference between the groups in basic characteristics and biochemical blood parameters. Among the echocardiographic parameters, left atrial diameter (LAD), left atrial area (LAA), left atrial volume (LAV), and left atrial volume index (LAVI) were significantly higher in the group with IAB (p<0.001 for all parameters). Serum Elabela level was significantly higher in the group with IAB (6.8±1.7) than in the group without IAB (5.5±1.8) (p=0.006). Furthermore, a statistically significant, moderate and positive linear correlation was observed between p wave duration and Elabela (r=0.534, p<0.01).

Conclusions: In the present study, we showed that the level of Elabela, which was found to be a new endogenous ligand, provides information on the impaired left atrial function in non-cardiogenic cerebral ischemia. Elabela may have a role in left atrial electromechanics, which has an important place in the pathogenesis of acute non-cardiogenic stroke patients, but more detailed and large-scale studies are needed for its use as a therapeutic agent.

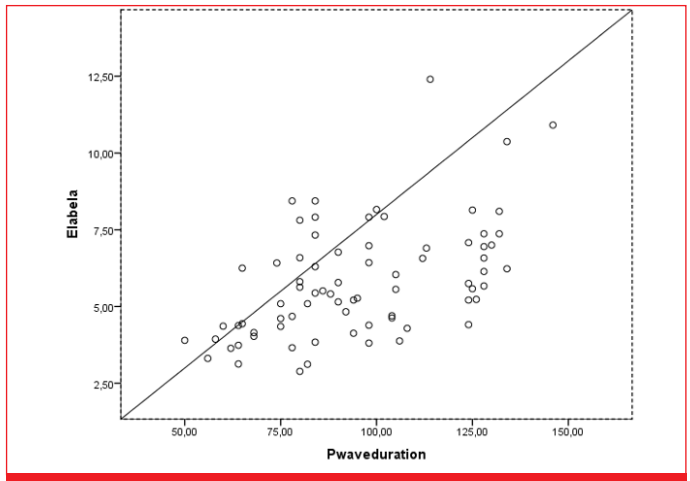

Figure 1. Correlation between Elabela and P wave duration (r=0.534, p<0.01).

Table 1. Demographic, electrocardiographic, routine blood and, echocardiographic parameters of the study groups

	IAB + (n=19)	IAB - (n=56)	*P*-value
Age, years	74.9±7.4	71.3±7.9	0.083
Famele gender, n(%)	6 (24)	23 (41)	0.644
BMI, (kg/m^2)	28.3±3.6	28.2±3.9	0.938
Diabetes mellitus,n(%)	6 (24)	20 (36)	0.961
Hypertension, n(%)	7 (37)	21(38)	0.959
Smoking habit, n(%)	8 (42)	11 (20)	0.069
Albumin, mg/dL	38.0±3.0	38.4±5.2	0.700
Creatinine, mg/dL	0.9±0.1	0.9±0.2	0.360
Sodium, mEq/L	139.7±3.9	140.6±2.9	0.375
Potassium, mEq/L	4.2±0.4	4.3±0.4	0.364
Hemoglobin, g/dL	14.4±2.0	13.5±1.6	0.069
C-reactive protein, mg/L	6.7 (0.1–16.8)	6.9 (0.1-29.6)	0.912
Elabela, ng/ml	6.8±1.7	5.5±1.8	0.006
Ejection fraction,%	56.2±4.8	55.2±4.0	0.376
LVDD, mm	43.7±3.3	43.0±5.0	0.604
LVSD, mm	35.2±2.7	34.5±4.6	0.542
IVSD, mm	11.4±1.2	11.3±1.6	0.828
PWD, mm	9.9±1.0	10.2±0.9	0.422
RVD, mm	27.3±3.5	27.6±2.8	0.861
E/A ratio	0.8±0.2	0.9±0.4	0.125
VTI E, cm	11.9±2.2	11.4±2.2	0.516
LAD, mm	50.2±4.3	43.4±4.1	<0.01
LAA1, cm^2	18.7±3.6	13.8±2.3	<0.01
LAA2, cm^2	17.0±2.0	13.2±2.0	<0.01
LAV, ml	52.7±10.9	35.4±8.7	<0.01
LAVI, ml/m^2	27.3±4.7	19.0±4.7	<0.01
P wave duration, ms	126.1±13.4	84.9±15.9	<0.01

BMI; Body mass index; IVSD, Interventricular septal diameter; LAD, Left atrial diameter; LAA, Left atrial area; LAV, Left atrial volume; LAVI, Left atrial volume index; LVDD, Left ventricular end-diastolic diameter; LVSD, Left ventricular end- systolic diameter; RVD, Right ventricular diameter; PWD, Posterior wall diameter; VTI, Velocity-time integral.

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-052

Postoperative atrial fibrillation after coronary artery bypass grafting surgery: A two-dimensional speckle tracking echocardiography study

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Background and Aim: Postoperative atrial fibrillation (POAF) may develop after coronary artery bypass grafting (CABG). The aim of the study was to explore the relationship between preoperative left atrial function and atrial fibrosis and POAF after CABG.

Methods: Forty-eight consecutive patients undergoing CABG (mean age: 61.6 ±8.9 years, 39 male) were included. All patients were in sinus rhythm during surgery. Patients were followed by continuous electrocardiography monitoring and daily electrocardiogram. Left atrial function was assessed by both conventional and speckle tracking echocardiography. Atrial fibrosis was determined by samples taken from right atrium.

Results: Postoperative atrial fibrillation was detected in 13 patients. Female sex and number of bypassed vessels were significantly higher and cardiopulmonary bypass time was significantly longer in patients with POAF. Left atrial volume index (LAVI) was significantly higher while left atrial reservoir strain was significantly lower in POAF patients. The percentage of patients with severe fibrosis was higher in the POAF group. Regression analysis revealed fibrosis and LAVI as independent predictors of POAF. Left atrial volume index ≥36 mL/m^2 predicted POAF with a sensitivity of 84.6% and specificity of 68.6% in our cohort.

Conclusions: Patients who developed POAF after CABG had more fibrosis, increased LAVI and lower left atrial reservoir strain. Preoperative echocardiography might be helpful in discriminating these patients.

Table 1. Characteristics of the patient

	POAF (+) Patients (n=13)	POAF (-) Patients (n=35)	*P*-value
Age(years)	64.5±7.7	60.5±9.2	0.094
Male (n)	8(61.5%)	31(88.6)	0.033
Body Surface Area (m^2)	1.82±0.09	1.85±0.15	0.403
Diabetes (n%)	8 (61.5.%)	17 (48.6%)	0.424
Hypertension (n%)	13 (100%)	34 (97.1%)	1
Hyperlipidaemia (n%)	6 (46.2%)	17 (48.6%)	0.882
Peripheral artery disease (n%)	4 (30.8)	5 (14.3%)	0.228

Table 1. Characteristics of the patients (Continued)

	POAF (+) Patients (n=13)	POAF (-) Patients (n=35)	*P*-value
Chronic renal faiure (n%)	1 (7.7%)	1 (2.9%)	0.473
Smoking (n%)	9 (69.2%)	25 (71.4%)	1
Previous stroke			
Ischemic (n%)	3 (23.1%)	5 (14.3%)	0.664
Haemorrhagic (n%)	0	1 (2.9%)	1
CPB time(min)	94.1±29.7	73.5±24.3	0.040
Aortic cross-clamp time (min)	48.6±17.4	41.3±14.9	0.214
Number of CABG vessels (n)	3.3±0.7	2.7±0.9	0.044
1 vessel	0	4	
2 vessels	1	8	
3 vessels	8	18	
4 vessels	3	4	
5 vessels	1	1	
beta blockers (n%)	12 (92.3%)	34 (97.1%)	0.473
calcium blocker (n%)	1 (7.7%)	2 (5.7)	1
ACE inhibitors (n%)	10 (76.9%)	33 (94.3%)	0.115
Statins (n%)	6 (46.2%)	17 (48.6%)	0.882

Table 2. Conventional echocardiographic measures of the patients

	POAF(+) patients (n=13)	POAF(-) patients (n=35)	*P*-value
LVED diameter(mm)	51.5±6.8	50.5±5.1	0.798
LVES diameters (mm)	34.6±7.8	33.2±5.7	0.710
LVED volume (mL)	168.4±47.6	161.4±33.4	0.816
LVES volume (mL)	76.1±30.5	74.9±22.1	0.790
LVEF(%)	54.5±5.8	56.6±5.9	0.273
LA diameter(mm)	40.0±5.2	36.4±4.7	0.007
LAVI (mL/m^2)	41.1±9.2	32.6±9.0	0.006
E/A	0.91±0.47	0.90±0.29	0.451
E wave deceleration time (ms)	201±23	189±28	0.108
E/e´	10.2±1.7	8.6±2.7	0.027
e´(cm/s)	7.5±1.6	8.7±2.8	0.222

Table 3. Speckle Tracking echocardiographic measures of the patients

	POAF(+) patients (n=13)	POAF(-) patients (n=35)	*P*-value
LV global longitudinal strain (%)	16.9±4.3	16.8±4.1	0.871
LV global circumferential strain (%)	19.7±9.7	18.0±7.0	0.945
LV global radial strain (%)	27.3±11.6	33.2±19.2	0.213
LA reservoir function(%)	20.8±6.9	30.0±12.8	0.038
LA conduit function(%)	11.1±3.8	14.6±7.0	0.132

Table 4. Comparison of echocardiographic parameters between the patients according to severity of fibrosis

	Severe fibrosis (n=23)	Mild fibrosis (n=25)	*P*-value
LA diameter (mm)	39.0±5.6	35.9±4.0	0.066
LAVI (mL/m^2)	36.4±10.6	34.5±9.0	0.959
LVEF(%)	55.7±6.1	56.3±5.8	0.967
E/e´	9.9±2.4	8.2±2.4	0.031
LV global longitudinal strain (%)	17.2±3.7	16.5±4.5	0.380
LV global circumferntial strain (%)	20.2±8.0	16.9±7.3	0.132
LV global radial strain (%)	24.7±15.4	34.6±19.8	0.060
LA reservour function /%)	23.1±9.3	31.6±13.2	0.025
LA conduit function(%)	12.5±5.6	14.7±7.0	0.261
POAF(n %)	11(47.8%)	2(8%)	0.003

Table 5. Multivariate analysis to determine the independent predictors of POAF

	Odds ratio	95% Confidence interval	*P*-value
LAVI	1.18	1.02-1.36	0.024
LVEF	1.15	0.92-1.45	0.220
E/e´	0.90	0.54-1.52	0.694
LA reservoir function (%)	0.94	0.83-1.08	0.943
Fibrosis	18.38	1.80-187.82	0.014
Age	1.05	0.92-1.21	0.457
Female sex	2.26	0.22-22.98	0.491

Arrhythmia / Electrophysiology / Pacemaker / CRT- ICD

OP-053

Percutaneous extraction of transvenous permanent pacemaker/defibrillator leads; A single center experience

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Background and Aim: The growing problem with endocardial lead infections and lead malfunctions has increased the need for percutaneous lead extraction approaches. The centers' experiences and data are growing in terms of lead dysfunction type, profile, lead extraction indications, using methods, complications, follow-up data, effectiveness, and safety. In this report, we present our initial experience in percutaneous lead extraction.

Methods: We present an observational retrospective cohort study of 93 patients admitted between January 2014 and December 2019 for the transvenous removal of total 163 endocardial leads. Patient data, device details, indications, lead types, lead characteristics, complications, re-implantation procedure, and follow-up data were collected from electronic medical records.

Results: Sixty-seven patients out of ninety-three were male (72%), and the mean age was 68.6±11.6 years. The indications for lead extraction were device pocket infection in 33 (35.5%) cases, lead dysfunction in 33 (35.5%) cases, device (system) upgrade in 21 (23%) cases and lead endocarditis in 6 (6%) cases. The duration from implantation to extraction time was detected median 43 (12-356) months or 3 (1-29) years. The retracted number of leads per patient was detected mean 1.75±0.84 and, total of 163 leads. The most common retracted lead type was the RV defibrillator lead (62%), and one hundred (61%) lead fixation type was active. After lead extraction, the new device was inserted in seventy-four (80%) patients and at the same session was detected in forty-nine patients (66%). The most common re-implanted device type was CRT-D (61%). After lead extraction, the patients were followed up mean 17 (1-72) months or 1 (0-6) years and eighteen patients (19%) died at follow-up. The complications were observed in eleven patients (12%), and the most common was pocket hematoma (in six patients). Complete procedural success was achieved in 83 (89%) patients, radiological failure was present in 8 (9%) patients and clinical failure was present in 16 (17%) patients.

Conclusions: Our experience suggested that transvenous lead extraction has a high success rate with an acceptable risk of procedural complications. The simple manual traction method has a high rate of procedural success, despite a high dwell time of lead.

Heart failure

OP-054

Exhaled air humudity in rats with pulmonary edema

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Background and Aim: Acute pulmonary edema is a condition that requires emergency treatment and is characterized by impaired gas exchange as a result of increased fluid in the lung interstitial and alveolar space. After acute pulmonary edema, the amount of fluid in the intra-alveoli, interstitial space, and pleural space will vary considerably. With the effect of intra-alveolar heat of the change in the amount of fluid in these spaces, it will evaporate in different amounts compared to the

fluid at physiological limits. Thus, the rate of intra-alveolar humidity will change. With the expiratory air, the air of altered intra-alveolar humidity will be expelled. The aim of this study is to compare the humidity rate of expiratory air between healthy and pulmonary edema developing rats.

Methods: 12 healthy adult rats were included in the study (Group 1, before ANTU). Acute pulmonary edema was induced by intraperitoneal administration of ANTU (alpha-naphthylythiourea) to healthy rats whose expiratory air humidity was measured (Group 2, after ANTU). 4 hours after ANTU, humidity was measured in the expiratory air again. Humidity rate in expiratory air was compared between the two groups using the Wilcoxon signed-rank test. Histopathological changes were evaluated using hematoxylin-eosin staining.

Results: Humidity measurement average in expiratory air was 71.22±3.59 before ANTU and 56.28±3.94 after acute pulmonary edema was created with ANTU, while the average humidity difference between the two groups was -14.94±5.96. It was determined that the humidity measurement average between the two groups was statistically different.

Conclusions: Humidity in the expiratory air was found to be significantly lower in rats with acute pulmonary edema than in healthy rats. This result supports the hypothesis that monitoring the humidity in the expiratory air can be used as an important parameter in patients who are followed up clinically for the development of pulmonary edema.

Figure 1. Humidity and temperature measurement in expiratory air in rats

Table 1. Comparisons of rat weights, average exhaled air temperature, and humidity measurements between groups

Rat weight before ANTU	195.92±18.31	0.01*
Rat weight after ANTU	190.83±17.68	0.01*
Average temperature before ANTU	24.13±0.49	0.04*
Average temperature after ANTU	23.65±0.38	0.04*
Average humidity before ANTU	71.22±3.59	0.01*
Average humidity after ANTU	56.28±3.94	0.01*

ANTU: Alpha-naphthylythiourea

Heart failure

OP-055

Comparison of inflammatory parameters of patients hospitalized with acute heart failure and classifield phenotypically

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Background and Aim: Heart failure (HF) is the inability of the heart to pump oxygen-rich blood to meet the metabolic needs of the tissues during rest and exercise despite adequate venous return. The clinics of patients with acute heart failure (AHF) are categorized by the presence of congestion (wet or dry) and the state of peripheral perfusion (cold or warm). In our study, we aimed to compare AFR values between groups in terms of mortality and prognosis.

Methods: Between December 2020 and August 2021, 240 patients who were newly diagnosed with AHF as a result of clinical evaluation or who had a diagnosis of HF and developed decompensation were included in the study. The patients were divided into 4 groups according to the phenotypic class of AHF (hot-wet, hot-dry, cold-wet, cold-dry). Congestion findings; pulmonary edema, orthopnea, paroxysmal nocturnal dyspnea, peripheral edema, neck venous engorgement, hepatomegaly, acid and hepatojugular reflux. Hypoperfusion findings; cold and sweaty extremities, oliguria, mental confusion, narrowed pulse pressure and increased lactate levels

In the patient groups, acute phase reactants (AFR) (CRP, Sedimentation, Albumin) were measured at hospitalization, discharge and 30±7 days after discharge.

Results: A significant difference was found between the exitus distributions between classes(p=0.019).There was no significant difference between the classes in terms of rehospitalization (p=0.604) (Table 1). A significant difference was found between the albumin hospitalization and discharge median values between classes (p<0.001). There was a significant difference in albumin control median values between classes (p=0.038) (Table 2). A significant difference was found between the median values of CRP hospitalization and discharge between classes (p<0.001). There was no significant difference between the CRP control median values between classes (p>0.050) (Table 3). There is no significant difference between the sedimentation median values at different times between classes(p>0.050) (Table 4). In the univariate model, the mortality risk decreases 0.794 times for each unit increase in albumin hospitalization value (p<0.001). The risk of mortality increases 1.013 times with each unit increase in the CRP hospitalization value (p=0.003). Mortality risk increases 1.026 times with each unit increase in sedimentation lying value(p=0.002). For each unit increase in albumin discharge value, the risk of mortality decreases 0.85 times (p=0.043). Other risk factors were not statistically sig-

nificant (p>0.050). In the multivariate model, the mortality risk decreases 0.803 times with each unit increase in the hospitalization albumin value (p<0.001). Mortality risk increases 1.021 times with each unit increase in sedimentation value (p=0.049). Risk factors measured at discharge were not statistically significant (p>0.05) (Table 5).

Conclusions: According to our study, it was understood that keeping albumin values high and CRP and sedimentation values low in hospitalized patients would contribute positively to mortality.

Table 1. Comparison of the groups in terms of mortality and rehospitalization

	Hot Wet	Hot Dry	Cold Wet	Cold Dry	Total	Test statistic	*P*-value
Rehospitalization							
No	53 (88.3)	55 (91.7)	52 (86.7)	56 (93.3)	216 (90)	χ^2=1.852	0.604
Yes	7 (11.7)	5 (8.3)	8 (13.3)	4 (6.7)	24 (10)		
Mortality							
No	56 (93.3)	59 (98.3)	51 (85)	50 (83.3)	216 (90)	χ^2=10.000	0.019
Yes	4 (6.7)^{ab}	1 (1.7)^{a}	9 (15)^{b}	10 (16.7)^{b}	24 (10)		

χ^2: Chi-square test statistic, $^{a-c}$: There is no difference between groups with the same letter

Table 2 Comparison of Albumin value between and within groups

	Hot Wet	Hot Dry	Cold Wet	Cold Dry	Total	Test statistic	*P*-value
Albumin hospitalization	36.59 ± 5.20	38.88 ± 4.27	33.87 ± 4.43	37.06 ± 3.99	36.60 ± 4.81	χ^2=35.386	0.001
	37.1 (20.3-48.8)^{bA}	38.95 (26.8-50.0)^{bA}	34.65 (21.5-43.0)^{aA}	37.10 (30.3-46.9)^{bA}	36.8 (20.3-50.0)		
Albumin discharge	35.11 ± 4.45	36.52 ± 4.4	33.23 ± 4.58	35.51 ± 3.87	35.14 ± 4.47	χ^2=15.943	0.001
	34.8 (22.7 - 46.8)^{abB}	37.0 (25.8 - 45.6)^{bB}	33.1 (19.8 - 42.1)^{aB}	35.7 (25.3 - 41.7)^{bB}	35.2 (19.8 - 46.8)		
Albumin control	37.65 ± 4.8	38.84 ± 4.45	36.37 ± 4.33	38.52 ± 5.12	37.91 ± 4.74	χ^2=8.397	0.038
	39.3 (20.7 - 44.3)^{abA}	39.5 (27.3 - 47.6)^{bA}	36.3 (24.2 - 44.6)^{aA}	39.4 (25.7 - 47.3)^{abA}	38.6 (20.7 - 47.6)		
Test statistic*	χ^2=17.015	χ^2=27.701	χ^2=29.129	χ^2=17.753			
p	<0.001	<0.001	<0.001	<0.001			

F: One-way analysis of variance test statistic,**F: Tertiary analysis of variance test statistic,χ^2: Kruskal Wallis test statistic, χ^2: Friedman test statistic, a-c: There is no difference between groups with the same letter, A-B: There is no difference between the times with the same letter within the classes ,mean ± standarddeviation, median (minimum – maximum)

Table 3. Comparison of CRP value between and within groups

	Hot Wet	Hot Dry	Cold Wet	Cold Dry	Total	Test statistic	*P*-value
CRP hospitalization	24.93 ± 52.37	11.73 ± 19.3	36.17 ± 37.42	20.69 ± 35.44	23.38 ± 38.76	χ^2=37.140	0.001
	10.25 (1.2-350.8)^{cAB}	5.2 (0.6 - 102.2)^{a}	22.50 (1.4-166.7)^{bB}	7.55 (0.6-213.8)^{acAB}	9.8 (0.6-350.8)		
CRP discharge	20.47 ± 28.71	12.79 ± 15.66	32.69 ± 40.12	17.66 ± 22.99	20.64 ± 28.76	χ^2=16.034	0.001
	11.4 (1.3-162.4)^{abB}	7.7 (1.2 - 80.2)^{a}	17.0 (2.1- 2 08.4)^{bB}	11.6 (1.7-141.1)^{abB}	10.9 (1.2-208.4)		
CRP control	13.77 ± 22.65	12.01 ± 23.52	18.67 ± 34.6	9.59 ± 12.98	13.35 ± 24.36	χ^2=4.933	0.177
	5.9 (0.8 - 110.3)^{A}	4.35 (0.5 - 149.0)	7.9 (0.5 - 158.2)^{A}	4.9 (0.9 - 51.1)^{A}	5.7 (0.5 - 158.2)		
Test statistic*	χ^2=8.133	χ^2=3.402	χ^2=21.907	χ^2=8.995			
p	0.017	0.183	<0.001	0.011			

F: One-way analysis of variance test statistic,**F: Tertiary analysis of variance test statistic,χ^2: Kruskal Wallis test statistic,χ^2: Friedman test statistic, a-c: There is no difference between groups with the same letter, A-B: There is no difference between the times with the same letter within the classes ,mean ± standarddeviation, median (minimum – maximum)

Table 4. Comparison of Sedimentation value between and within groups

	Hot Wet	Hot Dry	Cold Wet	ColdDry	Total	Test statistic	*P*-value
Sedimentation hospitalization	24.14 ± 20.87	22.98 ± 18.85	27.7 ± 26.0	26.55 ± 23.28	25.35 ± 22.35	χ^2=0.401	0.940
	16.0 (4.0 - 87.0)^{A}	15.5 (3.0 - 81.0)^{B}	20.0 (3.0-100.0)^{B}	19.5 (2.00-90.0)	18.0 (2.0-100.0)		
Sedimentation discharge	29.56 ± 23.83	27.08 ± 20.93	33.42 ± 26.43	26.45 ± 21.75	29.05 ± 23.25	χ^2=1.900	0.594
	24.0 (3.0 - 121.0)^{B}	19.5 (3.0 - 84.0)^{AB}	26.5 (4.0-103.0)^{A}	24.0 (3.0-97.0)	24.0 (3.0-121.0)		
Sedimentation control	29.15 ± 22.89	27.7 ± 21.59	27.86 ± 24.01	26.06 ± 25.23	27.7 ± 23.23	χ^2=1.817	0.611
	19.5 (3.0 - 112.0)^{AB}	21.0 (5.0 - 88.0)^{A}	19.0 (3.0-84.0)^{B}	18.0 (3.0-91.0)	19.0 (3.0-112.0)		
Test statistic*	χ^2=6.437	χ^2=14.462	χ^2= 1.593	χ^2= 1.144			
p	0.040	0.001	0.003	0.564			

F: One-way analysis of variance test statistic,**F: Tertiary analysis of variance test statistic, χ^2: Kruskal Wallis test statistic, χ^2: Friedman test statistic, a-c: There is no difference between groups with the same letter, A-B: There is no difference between the times with the same letter within the classes ,mean ± standarddeviation, median (minimum – maximum)

Table 5. Investigation of the effect of acute phase reactants on mortality by binary logistic regression

	Univariate		Multivariate 1		Multivariate 2	
	OR (%95 CI)	*P*-value	OR (%95 CI)	*P*-value	OR (%95 CI)	*P*-value
Albumin hospitalization	0.794 (0.719 - 0.876)	<0.001	0.803 (0.723 - 0.891)	<0.001		
CRP hospitalization	1.013 (1.005 - 1.022)	0.003	1.005 (0.996 - 1.014)	0.244		
Sedimentation hospitalization	1.026 (1.009 - 1.042)	0.002	1.021 (1 - 1.043)	0.049		
Albumin discharge	0.85 (0.726 - 0.995)	0.043			0.849 (0.702 - 1.027)	0.091
CRP discharge	1.011 (0.994 - 1.028)	0.200			1.005 (0.983 - 1.027)	0.673
Sedimentation discharge	1.003 (0.973 - 1.035)	0.835			0.997 (0.961 - 1.033)	0.852

Heart failure

OP-056

Differences in clinical characteristics and outcome of patients admitted to emergency department with de novo heart failure compared to chronic decompensated heart failure

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Background and Aim: Acute heart failure (AHF) is a syndrome defined as the new onset de-novo heart failure (HF)] or worsening [chronic decompensated heart failure (CDHF)] of symptoms and signs of HF, mostly related to systemic con-

gestion. Recent evidence shows differences in clinical characteristics and outcomes between de novo HF and ADHF. In this study, we aimed to evaluate the differences between clinical characteristics and outcome of patients with de-novo HF and CDHF.

Methods: This prospective, single-center study included 719 consecutive AHF patients who admitted to the Emergency department (ED) for new onset or worsening HF, between October 01, 2015 and September 30, 2016. We compared clinical characteristics between these two groups. Additionally, in-hospital outcome and short-term outcome of death from any cause, heart failure related hospitalization and admission to the ED at 90-day has been examined.

Results: Median age of the study population was 73.0 [66.0-80.0] years old and 54.9% were male. Of 719 patients, 373 (51.9%) had de-novo-HF, 346 (48.1%) had CDHF. Chronic decompensated HF suffered more frequently from hypertension (76.3% vs. 67.6%, p=0.009), coronary artery disease (78.6% vs. 38.1%, p<0.001) and chronic kidney disease (19.4% vs. 13.9%, p=0.051) compared to the de-novo HF group. Presence of atrial fibrillation/ atrial flutter was similar in both groups (20.8% vs. 17.4%, p=0.248).The most common symptoms were dyspnea without differences between the groups (99.3%). Furthermore, CDHF group exhibited more pronounced signs and symptoms of congestion, including rales (92.8% vs. 86.3%, p=0.005) and peripheral edema (76.6% vs. 63.0%, p<0.001). Orthopnea (96.0% vs. 95.4%, p=0.736) and body weight change (25.4% vs. 21.4%, p=0.207) were similar in both groups. CDHF group also had lower left ventricular ejection fraction 25.0% [16.0-35.0 IQR] vs 45.0% [30.0-55.0 IQR] (p<0.001) by bedside echo in ED. There was no significant difference between the two groups in terms of in-hospital clinical outcomes requiring mechanical ventilation, length of ED stay, length of intensive care unit (ICU) stay, length of total hospital stay and in-hospital death (p>0.05, for all). The 90-day admission to the ED were significantly higher in the CDHF group (p=0.001). However, there were no significant differences in 90-day mortality and re-hospitalization rate between both groups (p>0.05, for all) (Table 1).

Conclusions: This study demonstrated that patients admitted to ED for de-novo HF show a different clinical profile from patients with CDHF but there were no differences between the 2 groups regarding in-hospital mortality, 90-day re-hospitalization and mortality.

Table 1. In-hospital and 90-day outcomes of the groups

	CDHF group (n=346)	De-novo HF group (n=373)	*P*-value
Length of ED stay, min, [IQR]	248.0 [157.8-327]	259.0 [165.0-350]	0.480
Length of hospital stay, days, [IQR]	6 [4-10]	6 [4-11]	0.680
In-hospital death, n%	32 (9.2%)	28 (7.5%)	0.399
90-day ED readmissions, n (%)	157 (50.8%)	130 (38.0%)	0.001
90-day HF related hospitalization, n (%)	148 (47.9%)	141 (41.5%)	0.100
90-day death, n (%)	62 (17.9%)	51 (13.7%)	0.118

Heart failure

OP-057

The prognostic role of the Systemic Inflammatory Index (SII) in heart failure patients

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Background and Aim: The prevalence of heart failure (HF) is increasing worldwide with new treatment methods (percutaneous, medical) that increase survival in heart diseases. Inflammation plays a central role in the development of HF. Inflammation-related biomarkers have gained special interest in the cardiovascular era. Systemic immun-inflamatuar index (SII) is well-defined inflammation related biomarker that can be easily attained from a complete blood count examination. In addition, SII was demonstrated to predict mortality in infective endocarditis and coronary artery disease. In recent years, it has been shown that SII is associated with mortality in coronary artery disease, valve disease, myocardial infarction. In this study, we aimed to relationship between SII and survival in patients with HF.

Methods: A total of 672 HF were included in this retrospective and observational study. SII was determined as absolute platelet count × absolute neutrophil count/absolute lymphocyte count. SII was divided into two as low and high SII values according to the median (666x103) value. The primary endpoint of the study was all-cause mortality. The median follow-up duration of the study patients was 21 (8-42) months. During the follow-up period, the patients were divided into two groups as those who developed all-cause mortality or those who did not. Cumulative survival rates were determined by the Kaplan-Meier analysis and compared between the two groups using the log-rank test. Cox proportional hazard model was applied to clinical and laboratory features which are thought to be related with mortality for heart failure.

Results: After the application of exclusion criteria, finally 672 patients were analysed and all-cause mortality developed in 278 of this patient groups during follow-up period (Table 1). The number of all-cause mortality was 113 (34%) in the low SII group, the number of all-cause mortality was was 165 (49%) in the high SII group group. Median survival time in the whole study population was 59 (42-77, 95% CI) months, whereas median survival time in the low and high SII groups were 85 months (54-117, 95% CI), 42 months (29-55, 95% CI) respectively (p<0.001) (Fig. 1). While the male gender was statistically significantly less in the mortality group, left ventricular ejection fraction, systolic pulmonary artery pressure, functional capacity were higher (p<0.001 in all parameters). Urea nitrogen, NT-proBNP, SII were significantly higher in the mortality group (p<0.001 in all parameters). Time dependent Cox regression analysis revealed that high SII (≥666x103) level was found to be independent predictor of mortality (Table 2).

Conclusions: To the best of our knowledge, this is the first study to evaluate the relationship between SII and survival in patients with heart failure. High SII level is an independent predictor of mortality in patients with HF. Full blood count is a readily accessible, inexpensive and routine examination that provides correct and reproducible information.

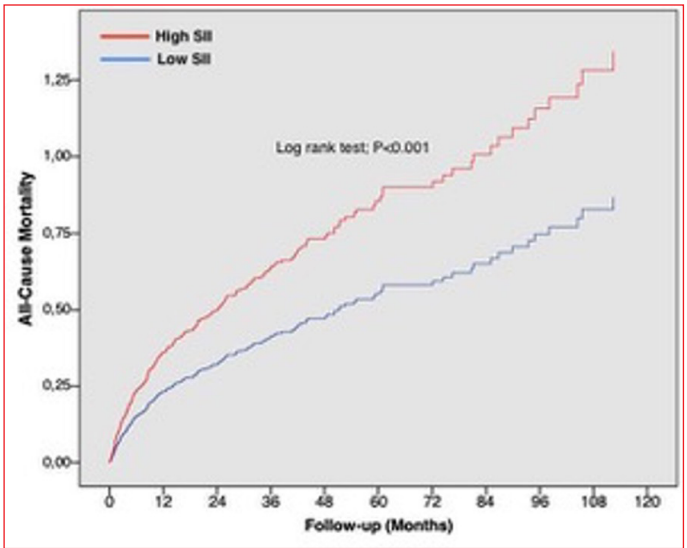

Figure 1.

Table 1. Baseline characteristics of study populations

Variables (n=672)	All-Cause Mortality		
	Without event (n= 394)	With event (n= 278)	P value
Demographic and clinical features			
Age (years)	49 (39-54)	50 (41-57)	.014
Gender (male)	288 (53%)	254 (91%)	<.001
Diabetes mellitus (n=600)	92 (27%)	71 (27%)	.815
Hypertension (n=651)	56 (15%)	36 (13%)	.535
Atrial fibrillation (n=657)	129 (34%)	80 (29%)	.355
Implantable cardiac defibrillator (n=670)	306 (78%)	196 (70%)	.026
Etiology (ischemic) (n=661)	141 (37%)	97 (35%)	.611
Left ventricular ejection fraction (%)	20 (18-26)	20 (15-22)	<.001
Systolic pulmonary artery pressure (mmHg)	42 (35-54)	48 (40-58)	<.001
NYHA (New York Heart Association) functional class			
Class I	63 (16%)	18 (6%)	
Class II	157 (40%)	65 (23%)	<.001
Class III	140 (36%)	110 (40%)	
Class IV	34 (8%)	85 (31%)	
Laboratory findings			
Glucose (mg/dl)	102 (90-123)	99 (88-116)	.027
Urea nitrogen (mg/dl)	40 (31-51)	48 (37-66)	<.001
Creatinin (mg/dl)	1.06 (0.87-1.25)	1.03 (0.86-1.16)	.066
Aspartate transaminase (SGOT) (U/L)	23 (18-30)	27 (20-35)	<.001
Alanine transaminase (SGPT) (U/L)	21 (16-32)	25 (16-41)	.009
Total bilirubin (mg/dl)	0.92 (0.59-1.54)	1.59 (0.92-2.40)	<.001
NT-proBNP (pg/mL) (n= 387)	903 (284-2467)	1605 (722-4090)	<.001
Hemoglobin (g/dL)	13.9±1.9	13.1±1.9	<.001
Hematocrit (%)	43.1±5.5	40.9±5.5	<.001
White cell count (10^3/mm^3)	8.20 (6.88-9.40)	7.82 (6.69-9.25)	.111
Neutrophil count (10^3/mm^3)	5.17 (4.01-6.30)	5.20 (4.37-6.40)	.138
Lymphocyte count (10^3/mm^3)	1.90 (1.44-2.40)	1.60 (1.26-2.10)	<.001
Platelet count (10^3/mm^3)	229 (190-280)	220 (180-272)	.111
Systemic immune-inflammation index (x10^3)	607 (420-919)	732 (499-1068)	<.001

Table 1.

Table 2. Time dependent Cox-regression analysis for all-cause mortality

Variables	All-cause mortality		
	Hazard ratio	95% CI	P value
Age	1.02	1.01-1.03	.002
Gender	1.39	0.85-2.29	.414
Left ventricular ejection fraction, %	0.95	0.92-0.97	<.001
Ischemic cardiomyopathy	0.88	0.67-1.16	.362
Diabetes mellitus	0.81	0.61-1.08	.156
Hypertension	1.01	0.69-1.47	.972
Implantable cardiac defibrillator	0.73	0.55-0.97	.028
Aspartat transaminase	1.01	1.00-1.01	<.001
High SII (≥666x10^3)	1.51	1.19-1.92	.001

SII= Systemic immun-inflammtion index

Table 2.

Heart failure

OP-058

The single center experience of Atrial Flow Regulator (AFR) implantation in patients with heart failure with either reduced or preserved ejection fraction

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Background and Aim: The left atrial decompression has emerged a new option to decrease left ventricular filling pressure in patients with heart failure. This article aimed to report the in-hospital and 3-month follow-up results following Atrial Flow Regulator (AFR) implantation in patients with heart failure with either reduced or preserved ejection fraction.

Methods: AFR-PRELIEVE trial is the multicenter, prospective, open-label and non-randomized study that enrolled symptomatic heart failure patients with either preserved or reduced ejection fraction. Patients who had left ventricular end-diastolic pressure > 15 mm Hg at rest or > 25 mm Hg during exercise with EF> 15% were enrolled in this study. We report here procedural and 3 months of follow-up data of our center. Echocardiographic features, 6-minute walking distance, Kansas City Cardiomyopathy Questionnaire (KCCQ), PCWP/LVEDP, BNP were assessed before and 3 months after implantation.

Results: Twenty-seven (69.2%) patients with reduced ejection fraction (HFrEF) and twelve (30.8%) patients with preserved ejection fraction (HFpEF) were enrolled in this study. Implantation success was 100% in both groups. Acute arterial deoxygenation was not reported. All patients were discharged from the hospital successfully. One patient died after hospital discharge due to pneumonia in HFrEF group. Three patients (11.1%) in HFrEF group were hospitalized for worsening heart failure. No worsening HF was observed in HFpEF group. Procedural related SAE was observed in 2 patients (5.1%), all

were in HFrEF group. No stroke/TIA, myocardial infarction or complications required device removal were noted in both groups. Device patency was 100% at 3-month follow-up.

Conclusions: Implantation of the AFR device in HFrEF and HFpEF seems safe and feasible. It led to improve symptoms and parameters of heart failure in some patients but not all. To determine that which patients benefit from AFR is the subject of another study.

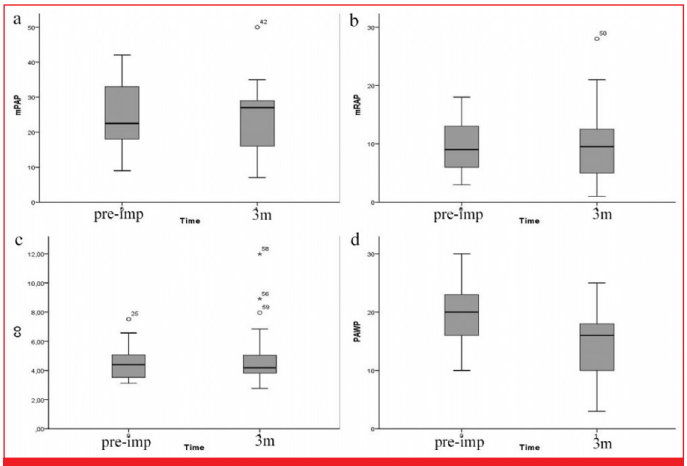

Figure 1. Temporal changes in CO (a), mean PAP (b), PAWP (c) and mean RAP (d) in HFrEF patients.

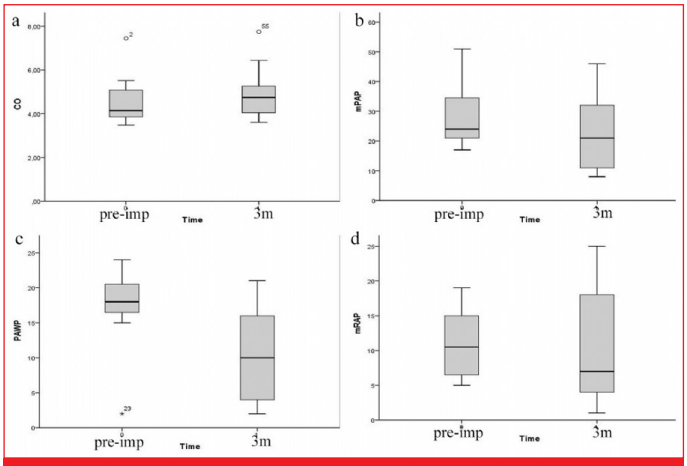

Figure 2. Temporal changes in CO (a), mean PAP (b), PAWP (c) and mean RAP (d) in HFpEF patients

Table 1. Pracedural characteristics of the study population.

	HFrEF patients	HFpEF patients
Implantation success, n (%)		
Device fenestration diameter		
8 mm, n (%)	22 (82)	11 (92)
10 mm, n (%)	5 (19)	1 (8)
Device waist height		
5 mm, n (%)	27 (100)	10 (83)
10 mm, n (%)	0 (0)	2 (17)
Balloon atrial septostomy duration, min	10 (4- 14)	9 (4- 14)
Device implantation duration, min	4 (3- 5)	4 (3- 6)
Overall catheterization duration, min	86 (70 103)	89 (71-105)
Fluoroscopy time, min	23 (18-27)	21 (16-27)
Qp/Qs ratio immediately after implantation of AFR	1.23±0.29 n=22	1.18±0.37 n=10
Qp/Qs ratio at 3 months	1.32±0.39 n=22	1.12±0.32 n=10
Left-to-right shunt flow at 3 months, n (%)	22 (100)	10 (100)

AFR- atrial flow regulator; TEE- transesophageal echocardiography

Table 2. Adverse events during 3-month follow-up

	HFrEF patients n=27	HFpEF patients n=12	All patients n=39
Hospitalization for worsening HF, n (%)	3 (11.1)	0 (0)	3 (7.7)
Death, n (%)	1(3.7)	0	1(2.6)
Stroke or TIA, n (%)	0	0	0
Myocardial infarction, n (%)	0	0	0
Device removal, n (%)	0	0	0
Procedure-related SAE, n (%)	2	0	2
SADE, n (%)	0	0	0
SAE rate, total numbers of events, n	30	10	1
Patients with SAE, n (%)	12 (44.4)	4 (33.3)	16 (41.0)
AE rates, total number of events, n	23	43	66
Patients with AE, n (%)	4(14.8)	11 (91.7)	15(38.5)
ADE total number, n	2	0	2
Patients with ADE, n (%)	2(7.4)	0	2(5.1)

ADE- adverse device event(s); AE- adverse event(s); HF- heart failure; SADE- serious adverse device event(s); SAE- serious adverse event(s); Transient ischemic attack

Table 3. Invasive measurements

Parameters	HFrEF patients, baseline n=24	HFrEF patients, at 3 months n=24	*P*-value	HFpEF patients, Baseline n=10	HFpEF patients, at 3 months n=10	*P*-value
Mean RAP, mm Hg, IQR	9.33 (5.25-12.75)	10.00 (4.50- 12.75)	0.70	10.90 (7.25- 14.25)	74 (56- 88)	0.80
Mean RAP, mm Hg, IQR	29 (18-33)	24 (16-29)	0.70	28 (20-36)	23 (11-33)	0.39
Systolic PAP, mm Hg	43 (29-54)	41 (35-47)	0.91	45 (30-56)	43 (32-56)	0.39
CO, L/min	4.45 (3.48- 4.79)	4.91 (3.80 -5.05)	0.45	4.67 (3.86- 5.44)	4.99 (4.01- 5.55)	0.65
PCWP, mm Hg	19 (16-24)	14 (8-18)	0.007	18 (17-21)	10 (4-17)	0.037
LVEDP, mm Hg	18 (14-22)	14 (8- 18)	0.01	16 (14- 21)	11 (4- 17)	0.10
Systolic aortic pressure, mm Hg	136 (124-151)	140 (119 – 155)	0.88	154 (135- 173)	152 (132- 173)	0.88
Diastolic aortic pressure, mm Hg	74 (64-82)	76 (64 – 88)	0.91	74 (56- 88)	75 (59- 93)	0.88

CO- cardiac output; LVEDP- left ventricular end-diastolic pressure; PAP- pulmonary artery pressure; PCWP- pulmonary capillary wedge pressure; RAP- right atrial pressure

Pulmonary hypertension / Pulmonary vascular diseases

OP-059

Predictors of health related quality of life in patients with pulmonary hypertension

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Background and Aim: There are various factors contributing to differences in severity of symptoms and decreased health-related quality of life (HRQoL) in patients with pulmonary hypertension (PH). Among the most evident determinants of well being, WHO (World Health Organization) functional class, along with some other clinical findings, plays one of the major roles in predicting HRQOL and estimating clinical outcomes in these patients. We aimed to investigate whether self-reported quality of life (QoL) is affected by or exhibits any changes in relation to etiology and severity of disease.

Methods: A total of 80 patients aged 18-80 admitted to Marmara University Hospital Pulmonary Vascular Diseases Outpatient Clinic consecutively were asked to complete PAH-SYMPACT® questionnaire. Respondents were interviewed in face-to-face fashion, whereas some took the digital version of the same questionnaire. The clinical properties e.g. WHO functional class, 6MWD (6 minute walk distance), NT-pro-BNP (N-terminal-pro-brain natriuretic peptide) and oxygen saturation levels were also evaluated. Obtained scores were evaluated for association with aforementioned clinical properties.

Results: The characteristics of study population was given in Table 1. As seen, 58 patients were (72.5%) female and 22 (27.5%) were male. Based on etiological classification, majority of the participants belong to Group 4 PH (43.8%) and Group 1 PH (37.5%), whereas minority of the patients belong to Group 2 (13.8%) and Group 3 (5%) PH. Reported mean value for 6MWD test is 341.4±125.4 meters, and mean values for pro-BNP and oxygen saturation levels are 1372.4±4199.2 pg/mL and 94%±8, respectively. Correlation between 6MWD, pro-BNP, oxygen saturation levels and the respective PAH-SYMPACT® scores for each of the mentioned variables was demonstrated in Table 2 using Pearson correlation coefficient. According to this results, 6MWD can be said to be highly correlated with PAH-SYMPACT® impact scores (r=-0.569, p<0.001), and moderately correlated with the total scores (r=-0.452, p<0.001). Interestingly, oxygen saturation have demonstrated lower correlation with impact scoring (r=-0.027, p=0.018). Both of the continuous variables exhibit inverse correlation in the above-mentioned categories. However, none of the Pearson's correlation coefficients was significant for pro-BNP levels. 6MWD remains to be one of the best predictors of HRQOL in PH patients, showing inverse correlation with PAH-SYMPACT® scores. Total PAH-SYMPACT® scores vary in accordance with certain, yet not all, PH groups.

Conclusions: HRQoL scoring shows variation with respect to the functional class, etiology and clinical outcomes in PH patients. It is therefore crucial to discover tools that will enable better monitoring of PH patients and maintain high QOL in the context of healthcare.

Table 1. General characteristics of the study population

Patients	n (%)
Female	58 (72.5)
Male	22 (37.5)
Etiology	
Idiopathic or associated PAH	30 (37.5)
PH due to left heart disease	11 (13.8)
PH due to lung diseases	4 (5)
PH due to CTEPH	35 (43.8)
Other Clinical Findings	
6MWD	341.4±125.4
NT-proBNP	1372.4±4199.2
Oxygen saturation %	98±8

Correlations

		Impact Score	Total Score	6 MWD Test	pro-BNP	Symptoms Score	Oxygen Saturation
Impact Score	Pearson Correlation	1	.925**	-.569**	-.035	.671**	-.270*
	Sig. (2-tailed)		.000	.000	.769	.000	.018
	N	80	80	62	73	80	77
Total Score	Pearson Correlation	.925**	1	-.452**	.034	.902**	-.177
	Sig. (2-tailed)	.000		.000	.773	.000	.123
	N	80	80	62	73	80	77
6 MWD Test	Pearson Correlation	-.569**	-.452**	1	-.026	-.227	.481**
	Sig. (2-tailed)	.000	.000		.841	.076	.000
	N	62	62	62	61	62	62
pro-BNP	Pearson Correlation	-.035	.034	-.026	1	.113	-.060
	Sig. (2-tailed)	.769	.773	.841		.341	.616
	N	73	73	61	73	73	71
Symptoms Score	Pearson Correlation	.671**	.902**	-.227	.113	1	-.042
	Sig. (2-tailed)	.000	.000	.076	.341		.719
	N	80	80	62	73	80	77
Oxygen Saturation	Pearson Correlation	-.270*	-.177	.481**	-.060	-.042	1
	Sig. (2-tailed)	.018	.123	.000	.616	.719	
	N	77	77	62	71	77	77

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

Table 2. Correlation between PAH-SYMPACT® scores and the clinical variables

Pulmonary hypertension / Pulmonary vascular diseases

OP-060

Effects of thyroid functions on long term mortality in patient with pulmonary arterial hypertension

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Background and Aim: PAH is a rare disease with high long-term mortality and morbidity, and it is also called Group 1 pulmonary hypertension. This disease has subgroups such as IPAH, congenital heart disease, connective tissue-related PAH, portopulmonary hypertension, pulmonary veno-occlusive disease. Although many echocardiographic and hemodynamic factors such as 6MWT, RV S', PVR, TAPSE and CI have been shown to have an effect on mortality, data on metabolic disorders are limited. Our aim in this study is to investigate the effect of thyroid dysfunction on PAH mortality.

Methods: Patients who were diagnosed with pulmonary arterial hypertension (PAH) between 2012-2020 years were included into this retrospective study. Their all-cause mortality information was extracted by hospital database search. The predictors of mortality were tested with logistic regression analysis including variables like thyroid function tests, pro-BNP, six minute walking test (6MWT), tricuspid annular plane systolic excursion (TAPSE), right ventricle pulsed Doppler S wave (RV S'), right atrial pressure (RAP) and cardiac index (CI).

Results: One hundred and twenty one PAH patients were included into the retrospective analysis. The mean age of 121 patients was 48.1±15.6 and 84% (n=6) patients was female gender. Median follow up duration was 74 months. Twenty one deaths occurred during the follow up. Female gender, hypothyroidism and portopulmonary hypertension frequency was higher in patient who died compared to patient who were alive. Hemoglobin levels (12.7±3.7 g/dL), T3 levels (2.76±0.75), 6MWT (214.2±97.7), TAPSE (14.4±2.8), RV S' (9.19±3.1), RV FAC (33.2±8.6) and CI (2.49±0.69) was lower in patient who died compared to patients who were alive. T4 levels (11.01±1.27), TSH (7.48±2.61), LDL (127.1±36.9), NT pro-BNP (2466±1641), RVED diameter (41.1±6.2), mPAB (49.2±13.3), RAP (11.9±5.5) and pulmonary vascular resistance(PVR) (8.81±4.48) was higher in patient who died compared to patients who were alive. Left ventricular ejection fraction (LVEF), sPAP and PCWP were similar between the groups. Logistic regression analysis revealed that hemoglobin levels, T4, TSH, 6MWT, RV S', RAP an CI were the independet predictor of long term mortality.

Conclusions: Thyroid stimulating hormones and T4 levels can be used as an independent predictors of long term mortality in PAH patients.

Table 1. Demographic and clinical characteristics of the overall study population and study group with regard to mortality

	Overall study population (n=121)	Without mortality (n=100)	With mortality (n=21)	*P*-value
Age, years	48.1±15.6	47.7±15.6	50.1±15.7	0.526
Female gender n (%)	84 (69.4%)	76 (76%)	9 (42.9%)	0.008
BMI	26.1±5.2	26.1±5.3	26±4.7	0.954
Subgroup of PAH				
IPAH n (%)	32 (26.4%)	23 (23%)	9 (42.9%)	0.084
CHD n (%)	46 (38%)	43 (43%)	3 (14.3%)	0.001
CTD n (%)	36 (29.8%)	30 (30%)	6 (28.6%)	0.476
PVOD n (%)	2 (1.7%)	2 (2%)	0	-
Portal HT n (%)	5 (4.1%)	2 (2%)	3 (14.3%)	0.006
Risk factors				
HT n (%)	30 (24.7%)	22 (22%)	8 (38.1%)	0.178
DM n (%)	8 (6.6%)	4 (4%)	4 (19.1%)	0.109
HL n (%)	7 (5.7%)	4 (4%)	3 (14.3%)	0.215
AF n (%)	5 (4.1%)	3 (3%)	2 (9.5%)	0.346
Hypotiroidism	46 (38.1%)	33 (33%)	13 (61.9%)	0.022

BMI: Body mass index, IPAH: idiopathic pulmonary arterial hypertension, CHD: Congenital heart disease, PVOD: Pulmonary veno-occlusive disease, HT: Hypertension, DM: Diabetes mellitus, HL: Hyperlipidemia, AF: atrial fibrillation

Table 2. Laboratory and echocardiographic findings of study population

	Overall study population (n=121)	Without mortality (n=100)	With mortality (n=21)	*P*-value
Hemoglobin (g/dL)	14.03±2.62	14.3±2.26	12.72±3.71	0.012
T3 (ng/mL)	3.28±0.91	3.39±0.91	2.76±0.75	0.004
T4 (µg/dL)	11.69±1.69	11.83±1.74	11.01±1.27	0.043
TSH (mIU/L)	5.52±2.74	5.11±2.59	7.48±2.61	<0.001
LDL(mg/dL)	111.1±33.6	107.8±32.1	127±36.9	0.017
Creatinine (mg/dL)	0.82±0.24	0.79±0.21	0.95±0.36	0.007
NT pro-BNP (pg/mL)	1420±1952	1200±1947	2466±1641	0.006
6MWT(meters)	358.7±312.8	389±333.7	214±97.7	0.019
LVEF(%)	61.7±2.48	61.7±2.4	61.6±2.8	0.866
sPAP(mm Hg)	68.6±27.6	66.7±28.6	77.5±20.8	0.104

Table 2. Laboratory and echocardiographic findings of study population (Continued)

	Overall study population (n=121)	Without mortality (n=100)	With mortality (n=21)	*P*-value
TAPSE (mm)	16.6±4.2	17.1±4.2	14.4±2.8	0.006
RV S' (cm/sec)	11.1±3.5	11.6±3.5	9.2±3.1	0.005
RVED d (mm)	38.9±8.1	37.3±8.3	41.1±6.2	0.054
RV FAC (%)	38.1±10.8	39.4±10.9	32.2±8.6	0.006

TSH: tyroid stimulate hormone, LDL: low density lipoprotein, NT-pro BNP: N-terminal pro b-type natriuretic peptit, 6MWT: six mşinute walking test, LVEF: Left ventricular ejection fraction, sPAB: systolic pulmonar artery pressure,TAPSE: tricuspid annular plane systolic excursion, RV S': right ventricle pulsed Doppler S wave,RVEDd: right ventricular end diastolic diameter, RV FAC: right ventricular fractional area change

Table 3. Hemodynamic findings of study population

	Overall study population (n=121)	Without mortality (n=100)	With mortality (n=21)	*P*-value
sPAP (mm Hg)	64.1±26.2	61.9±26.6	74.2±22.1	0.051
mPAP (mm Hg)	41.1±17.2	39.3±17.5	49.2±13.3	0.016
RAP (mm Hg)	9.2±5.2	8.64±4.9	11.9±5.5	0.008
PCWP (mm Hg)	10.1±2.5	9.95±2.52	10.3±2.7	0.484
PVR (woods)	6.37±4.46	5.86±4.31	8.81±4.48	0.005
CO (L/min)	5.13±1.72	5.3±1.75	4.3±1.3	0.015
CI (L/min/m^2)	3.1±1.1	3.34±1.1	2.49±.0.7	<0.001

sPAP: systolic pulmonary artert pressure, mPAP: mean pulmonary artery pressure, PCWP: pulmonary capillary wedge pressure, PVR: pulmonary vascular resistance, CO: cardiac output, CI: Cardiac index

Table 4. Univariate and multivariate analyses showing the predictors of mortality

	Univariate logistic regression				Multivariate logistic regression			
	OR	95% Confidence interval		*P*-value	OR	95% Confidence interval		*P*-value
Hgb (g/dL)	0.802	0.662	0.972	0.025	0.746	0.572	0.974	0.032
T4 (µg/dL)	0.778	0.593	1.020	0.069	1.866	0.880	3.954	0.104
TSH (mIU/L)	1.339	1.129	1.589	0.001	1.592	1.089	2.328	0.017
6MWT (meters)	0.991	0.986	0.995	<0.001	0.990	0.983	0.997	0.004
RVS' (cm/sec)	0.815	0.702	0.946	0.007	1.370	1.012	1.855	0.042
RAP (mm Hg)	1.111	1.023	1.207	0.013	1.205	1.039	1.398	0.014
CI (L/min/m^2)	0.303	0.144	0.637	0.002	0.264	0.103	0.677	0.006

Hgb: hemoglobin, TSH: tyroid stimulate hormone, 6MWT: six minute wlking test, RV S': right ventricle pulsed Doppler S wave, RAP: right atrial pressure, CI: cardiac index

Pulmonary hypertension / Pulmonary vascular diseases

OP-061

Artificial intelligence for prediction of chronic thromboembolic pulmonary hypertension using electrocardiography

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Background and Aim: Screening and true diagnosis of chronic thromboembolic pulmonary hypertension(CTEPH) are critical for managing the progression and preventing associated mortality; however, there are no tools for this purpose. We developed and validated an artificial intelligence (AI) algorithm for predicting CTEPH using electrocardiography (ECG).

Methods: ECG signals were obtained from 54 healthy and 23 CTEPH patients to test the method. A dataset was created by converting the ECG results to digital. The size of the 12 channel ECG signal received from 77 individuals is 924x1300 in size. The end-point was the diagnosis of CTEPH. By applying the suggested Nigerian motif pattern method on this data set, we obtained a feature matrix of 924x15010. FSC-mRMR algorithm was determined the most influential 947 characteristics among 15010 features and obtained a matrix of 924x947. We used a decision tree, SVM, and used KNN algorithms to classify the most weighted features selected.

Results: We achieved 98.05% success with the decision tree algorithm, 99.89% with the SVM algorithm, and 99.67% with the KNN algorithm. AI algorithm focused on the S-wave, P-wave, and T-wave for each patient by QRS complex characteristics.

Conclusions: The AI algorithm demonstrated high accuracy for CTEPH prediction using 12-lead and single-lead ECGs.

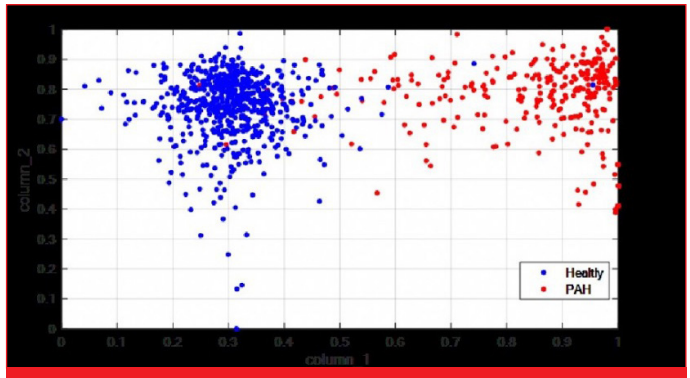

Figure 1. Scatter plots of the selected features

Pulmonary hypertension / Pulmonary vascular diseases

OP-062

Artificial Intelligence for Prediction of Pulmonary Arterial Hypertension Detection Using Computer Tomography

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Background and Aim: Artificial intelligence based on deep learning is developing rapidly in the field of pulmonary arterial hypertension(PAH), and verifying its efficacy is a prerequisite for promoting its clinical application. This study evaluates the value of artificial intelligence software based on an automated learning method to detect PAH in detecting pulmonary arterial hypertension on computed tomography (CT) of the chest.

Methods: The chest CT database built by the Firat University Hospital selected 491 (284 healthy and 207 PAH CT images) chest CT data. These images were acquired from 106 patients and 58 of them male and 48 of them female. There are two categories, and these are healthy and PAH. The proposed automated PAH detection method has exemplar pyramid feature generation, neighborhood component analysis-based feature selection, and classification phases(local binary pattern (LBP) and histogram oriented gradients (HOG) methods). The exemplar pyramid's generated features employing LBP and HOG are forwarded to the neighborhood component analysis (NCA) selector. The selected most valuable features are utilized as an input of the support vector machine.

Results: The proposed exemplar pyramid LBP and HOG based method yielded a 99.59% detection rate on the collected CT dataset.

Conclusions: According to findings and results, the recommended exemplar pyramid feature generation architecture and NCA selector based automated PAH method are very successful. This research also denotes the success of machine learning on a rarely seen disease.

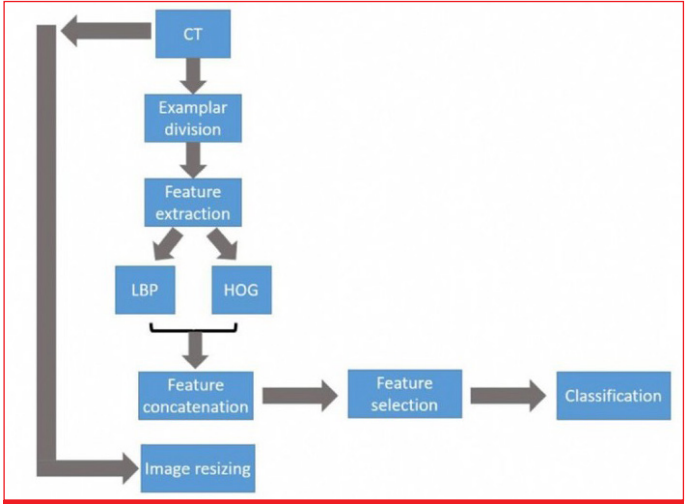

Figure 1. The block diagram of the presented exemplar pyramid LBP and HOG feature generation and NCA selector based method

Table 1. The confusion matrix of the proposed exemplar LBP and HOG based method

	PAH	HEALTHY	RECALL(%)
PAH	207	0	100
HEALTHY	2	282	99,29
PRECİSİON(%)	99,4	100	99,59

Pulmonary hypertension / Pulmonary vascular diseases

OP-063

Effects of balloon pulmonary angioplasty procedure on electrocardiographic parameters in patients with chronic thromboembolic pulmonary hypertension

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Background and Aim: To investigate the electrocardiography (ECG) changes that can predict the improvement in hemodynamics and right heart functions with ECG follow-ups in patients who are being followed up with the diagnosis of chronic thromboembolic pulmonary hypertension (CTEPH) and underwent balloon pulmonary angioplasty (BPA)

Methods: Data of 32 patients who underwent BPA in our hospital between 01.10.2017 and 31.12.2020 were analyzed in our study. During ECG analysis, IR, IS, IIp, IIIR, IIIS, aVR R, V1 R, V1 S, V5 R, V5 S, V6 R, V6 S, V1 t, V2 t, V3 t and QRS axis were measured in all ECGs. And the data obtained before BPA and at the 6th month control visit were compared with each other. In addition to ECG analysis, all patients' right heart catheterization (CCT) data, echocardiography data, B-type natriuretic peptide (BNP) levels and world health organization (WHO) functional classifications were recorded before the procedure and at the 6th month control visit. All parameters obtained before and after the BPA procedure were compared within themselves. The relationship of possible changes in electrocardiographic parameters with changes in hemodynamic parameters was investigated.

Results: Daniel scoring, which was previously seen to show a poor prognosis in acute pulmonary embolism, decreased from 8.22±5.68 to 6.56±5.55 after the BPA procedure (p=0.035). In addition, it was observed that the T wave height (V2 t) in V2 derivation increased from-0.77±2.39 to 1.27±2.58. The amount of change in V2 t was found to correlate with the amount of change in systolic right ventricular pressure, mean pulmonary artery pressure, pulmonary vascular resistance, and systemic vascular resistance.

Conclusions: It was observed that BPA significantly decreased the Daniel score and also significantly increased V2 t in CTEPH patients. According to these results, it may be suggested that by following the change in V2 t wave after BPA in CTEPH patients, it might be possible to have an idea about the change in systolic right ventricular pressure, mean pulmonary artery pressure, pulmonary vascular resistance and systemic vascular resistance.

Table 1. Demographic characteristics of the study population

Age (years)	51.7 (±14.3)
Male	14 (43.8%)
Female	18 (56.2%)
BMI (kg/m²)	27.6±6.6
Anticoagulation	25 (78.1%)
VKA	14 (43.8%)
NOAC	11 (34.4%)
Pulmonary Endarterectomy	15 (46.9%)
DM	2 (6.3%)
HT	3 (9.4%)

BMI: Body mass index, DM: Diabetes Mellitus, HT: Hypertension VKA: Vitamin K antagonist, NOAC: New oral anticoagulants.

Table 2. Echo, RHC, BNP and WHO classification changes, change amounts, change intervals and significance values before and after the procedure

Variable	Pre-BPA	Post-BPA	Change Amount %95	Change interval	*P*-value
Systolic RVP (mm Hg)	82.0±18.6	65.6±17.0	16.4±19	8.3 / 24.4	<0.001
RA pressure (mm Hg)	10.1±5.1	9.3±2.8	1.3±5.0	-1.5 / 3.3	0.453
Mean PAP (mm Hg)	50.3±12.9	41.3±9.47	9.1±11.4	4.65/13.49	<0.001
PAWP (mm Hg)	10.1±2.3	10.3±1.6	-1.1±2.0	-0.89/0.67	0.782
CO (L/min.)	4.37±1.34	4.81±0.88	-4.4±1.6	-1.06/0.18	0.160
CI (L/min./m^2)	2.43±0.64	2.73±0.62	-0.31±0.93	-0.67/0.05	0.089
PVR (woods)	10.69±5.18	7.36±3.44	3.3±4.89	1.43/5.22	0.001
SVR (woods)	19.29±4.94	15.86±4.03	3.43±6.81	0.56/6.31	0.021
WHO	2.87±0.63	2.43±0.82	0.43±0.73	0.16/0.71	0.003
BNP (pg/ml)	2 251±4 190	1 407±3 420	844±4262	-720/2407	0.279
RV basal diameter (mm)	46.7±7.6	45.2±7.1	1.47±4.84	-0.37/3.31	0.112
RA area (cm^2)	24.7±8.6	21.6±7.8	3.1±6.7	0.62/5.62	0.016
TAPSE (mm)	14.6±4.3	15.0±4.7	-0.43±2.83	-1.49/0.62	0.408
RVS (cm/s)	9.9±2.6	10.6±3.4	0.67±2.46	-1.61/0.27	0.154
Peak TR jet (m/s)	4.17±0.71	3.89±0.78	0.27±0.58	0.05/0.49	0.018
EF (%)	60.6±8.5	60.3±8.2	0.31±10.7	-3.5/4.1	0.867
LVEDD (mm)	43.3±6.8	43.3±5.8	0.03±3.71	-1.4/1.4	0.961
LVESD (mm)	27.5±5.6	27.6±5.1	-0.1±4.36	-1.7/1.5	0.901
Septum (mm)	9.80±1.35	9.90±1.75	-0.1±1.34	-0.62/0.42	0.698
Posterior wall (mm)	9.40±1.13	9.37±1.43	0.03±1.40	-0.49/0.56	0.897

BNP: B-type natriuretic peptide, CI: Cardiac index, CO: Cardiac output, EF: Ejection fraction, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, PAP: Pulmonary artery pressure, PAWP: Pulmonary artery wedge pressure, PVR: Pulmonary vascular resistance, RA: Right atrium, RHC: Right heart catheterization, RV: Right ventricle, RVP: Right ventricular pressure, RVS: Tricuspid annular systolic velocity, SVR: Systemic vascular resistance, TAPSE: Systolic movement of the tricuspid valve annular plane towards the apex, TR: Tricuspid regurgitation, WHO: World Health Organization.

Table 3. ECG changes, change amounts, change intervals and significance values before and after the procedure

Variable	Pre-BPA	Post-BPA	Change Amount 95%	Confidence interval	*P*-value
IR (mm)	4.5±2.1	4.6±2.2	-0.75±1.73	-0.70/0.55	0.806
IS (mm)	3.9±3.0	4.0±2.9	-0.54±1.92	-0.75/0.64	0.875
IIp (mm)	1.3±0.5	1.4±0.5	-0.11±0.52	-0.30/0.08	0.244
IIIR (mm)	6.4±6.0	6.4±4.9	0.03±3.85	-1.38/1.45	0.960
IIIS (mm)	2.2±1.8	2.1±1.9	0.05±2.00	-0.67/0.77	0.883
avR R (mm)	2.8±1.8	2.6±1.6	0.16±1.30	-0.31/0.63	0.504
V1R (mm)	5.3±4.5	5.8±7.0	-0.46±6.57	-2.83/1.91	0.693
V1S (mm)	3.1±2.66	3.7±3.1	-0.59±2.24	-1.40/0.21	0.143
V5R (mm)	10.5±6.9	5.1±3.2	0.87±5.69	-1.18/2.93	0.392
V5S (mm)	5.7±4.5	5.1±3.2	0.58±4.35	-0.99/2.15	0.458
V6R (mm)	8.2±4.3	8.5±4.0	-0.35±4.59	-2.00/1.31	0.673
V6S (mm)	3.8±2.5	3.75±2.5	0.08±2.65	-0.87/1.04	0.858
V1 t (mm)	-1.41±1.19	-1.11±1.06	-0.30±1.02	-0.67/0.07	0.111
V2 t (mm)	-0.77±2.39	1.27±2.58	-0.89±2.30	-1.72/-0.06	0.036
V3 t (mm)	-1.10±3.53	-0.39±2.95	-0.71±2.48	-1.60/0.19	0.116
QRS aks	63±60	74±50	-11.7±43.4	-27.3/4.0	0.138
Velocity (pulse/min)	81.4±13.6	85.3±15.3	-3.9±17.0	-10.1/2.2	0.201
V1R + V5S	11.1±7.5	10.9±8.3	0.12±6.76	-2.32/2.55	0.924
V1R/V1S	2.20±2.55	2.91±4.76	-0.71±3.68	-2.16/0.75	0.326
V5R/V5S	2.91±3.26	2.53±2.24	0.37±3.08	-0.78/1.53	0.511
V6R/V6S	2.76±2.22	2.82±2.49	-0.06±2.46	-1.02/0.89	0.891
Daniel Score	8.22±5.68	6.56±5.55	1.66±4.24	0.13/3.18	0.035

Table 4. Correlation analysis between the amount of change in Daniel score and the amount of change in Echo/RHC parameters

ΔDaniel	R	*P*-value
ΔSystolic RVP	0.306	0.145
ΔMean PAP	0.156	0.429
ΔPVR	0.295	0.128
ΔRA area	-0.135	0.476
ΔWHO	0.303	0.104
ΔSVR	0.302	0.152

PAP: Pulmonary arterial pressure, PVR: Pulmonary vascular resistance, RA: Right atrium, RHC: Right heart catheterization RVP: Right ventricular pressure, SVR: Systemic vascular resistance, WHO: World Health Organization.

Table 5. Correlation analysis between the amount of change in V2 t and the amount of change in Echo/RHC parameters

ΔV2 t	R	*P*-value
ΔSystolic RVP	-0,514	0,010
ΔMean PAP	-0,398	0,036
ΔPVR	-0,486	0,009
ΔRA area	-0,338	0,068
ΔWHO	-0,254	0,176
ΔSVR	-0,508	0,011

PAP: Pulmonary arterial pressure, PVR: Pulmonary vascular resistance, RA: Right atrium, RHC: Right heart catheterization, RVP: Right ventricular pressure, SVR: Systemic vascular resistance, WHO: World Health Organization.

Congenital heart diseases

OP-064

Early repolarization pattern may predict poor cardiovascular outcomes in patients with potentially serious coronary artery anomalies

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Background and Aim: Congenital coronary artery anomalies (CCAAs) have serious life-threatening complications such as fatal ventricular arrhythmias, syncope, and sudden cardiac death (SCD). Omreover, early repolarization pattern (ERP) has been associated with an increased risk of ventricular fibrillation and SCD. In this study, we aimed to evaluate the relationship between ERP and poor cardiovascular clinical outcomes in patients with potentially serious CCAAs.

Methods: This retrospective study included 106 potentially serious CCAA patients (mean age: 55±13 years; male:54) who were diagnosed with conventional and coronary computed tomography angiography (CCTA). All patients underwent transthoracic echocardiography and 12-lead surface electrocardiography. Cardiac events were defined as sustained ventricular tachycardia or fibrillation, syncope, cardiac arrest, and SCD.

Results: The presence of interarterial course (IAC) was confirmed by CCTA in 48 (44.3%) patients. During a median follow-up time of the 24 (18-50) months, a total of 12 (11.3%) patients experienced cardiac events. The presence of IAC was significantly more frequent and ERP were significantly greater in patients with poor clinical outcomes. Moreover, the presence of IAC and ERP were found to be independent predictors of poor clinical outcomes and decreased long term cardiac event free survival in these patients.

Conclusions: ERP and IAC may be associated with poor cardiovascular clinical outcomes in potentially serious CCAA patients.

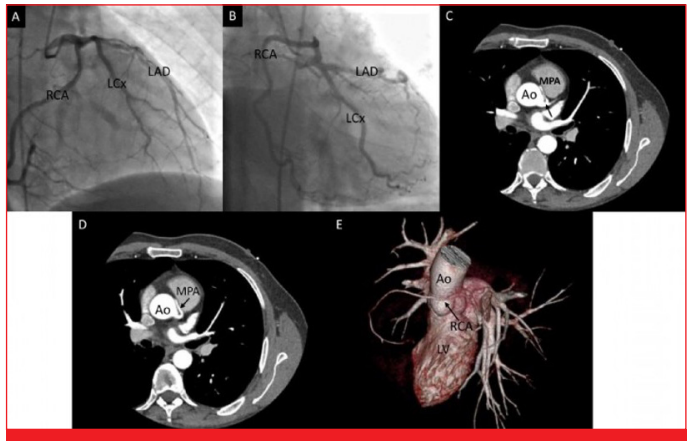

Figure 1. Coronary angiography shows a single coronary artery (SCA) without significant stenosis in any coronary artery segment (A, B). Contrast-enhanced cardiac computed tomography indicates that the right coronary artery is between the aorta and pulmonary artery (arrowheads) and confirms the SCA originating from the left sinus of the Valsalva (arrowhead) (C–E).

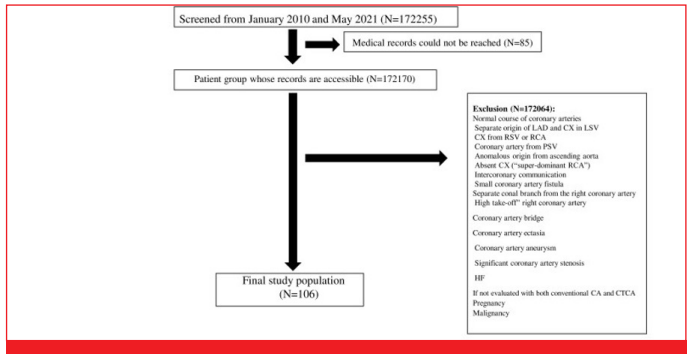

Figure 2. Flowchart for patient selection

Table 1.

	All Patients (n=106)	Patients with poor CV clinical outcomes (n=12)	Patients without poor CV clinical outcomes (n=94)	*P*-value
Coronary artery anomalies				
LMCA from RSV, n (%)	20 (18.9)	3 (25.0)	17 (18.1)	0.403
LAD from RSV, n (%)	9 (8.5)	0 (0)	9 (9.6)	0.324
RCA from LSV, n (%)	32 (30.2)	4 (33.3)	28 (29.8)	0.518
LMCA from PA, n (%)	2 (1.9)	0 (0)	2 (2.1)	0.785
SCA from RSV, n (%)	15 (14.2)	1 (8.3)	14 (14.9)	0.466
SCA from LSV, n (%)	16 (15.1)	4 (33.3)	12 (12.8)	0.081
Large/Multiple CAF, n (%)	7 (6.6)	0 (0)	7 (7.4)	0.420
Interarterial Course, n (%)	48 (45.3)	37 (91.7)	11 (39.4)	0.001

The distribution of coronary artery anomalies among study population. (CAF: Coronary artery fistulae, CV: Cardiovascular, LAD: Left anterior descending coronary artery, LMCA: Left main coronary artery, LSV: Left sinüs of Valsalva, PA: Pulmonary artery, RCA: Right coronary artery, RSV: Right sinus of Valsalva, SCA: Single coronary artery)

Table 2.

	All Patients (n=106)	Patients without poor CV clinical outcomes (n=94)	Patients with poor CV clinical outcomes (n=12)	*P*-value
Age, (years)	55±13	56±14	52±9	0.342
Gender, male	54 (50.9)	47 (50.0)	7 (58.3)	0.587
Diabetes Mellitus, n(%)	15 (14.2)	14 (14.9)	1 (8.3)	0.466
Hypertension, n (%)	39 (36.8)	37 (39.4)	2 (16.7)	0.109
Dyslipidemia, n (%)	26 (24.5)	25 (26.6)	1 (8.3)	0.151
Smoking, n (%)	22 (20.8)	20 (21.3)	2 (16.7)	0.527
Chronic Renal Disease, n (%)	25 (23.6)	25 (26.6)	0 (0)	0.032
Non-critical CAD, n (%)	41 (38.7)	36 (38.3)	5 (41.7)	0.528
LVEF, (%)	60 (55-65)	60 (55-65)	65 (60-65)	0.008
LAD, (mm)	37 (34-40)	37 (34-40)	35 (34-38)	0.314
LVEDD, (mm)	46 (44-49)	46 (44-50)	45 (44-45)	0.103
LVESD, (mm)	29 (26-32)	30 (26-32)	27 (26-29)	0.070
PWT, (mm)	10 (9-11)	10 (9-11)	10 (9-10)	0.081
IVST, (mm)	11 (11-12)	11 (11-12)	11 (11-12)	0.449
Diastolic Dysfunction, n (%)	21 (19.8)	20 (21.3)	1 (8.3)	0.263
P wave duration, (msec)	100 (93.5-108.5)	100 (94-108)	100 (93-109)	0.753
QRS duration, (msec)	86 (80-92)	88 (80-92)	86 (81-87)	0.349
T wave duration, (msec)	158 (148-166.25)	158 (148-165)	160 (143-166)	0.944
PR interval, (msec)	166 (144-182.5)	166 (144-184)	166 (148-178)	0.881
Pathological Q wave, n (%)	6 (5.7)	6 (6.4)	0 (0)	0.477
Early repolarization, n (%)	19 (17.9)	11 (11.7)	8 (66.7)	<0.001
T wave inversion, n (%)	11 (10.4)	7 (7.4)	4 (33.3)	0.021
ST depression> 1mm, n (%)	5 (4.7)	5 (5.3)	0 (0)	0.542
Antiplatelet agents, n (%)	33 (31.1)	31 (33.0)	2 (16.7)	0.211
B-blockers, n (%)	23 (21.7)	22 (23.4)	1 (8.3)	0.213
CCB, n (%)	20 (18.9)	18 (19.1)	2 (16.7)	0.597

Comparison of demographic, echocardiographic and electrocardiographic parameters between patients with and without poor cardiovascular clinical outcomes (CAD: Coronary artery disease, CCB: Calcium channel blocker, CV: Cardiovascular, IVST: Interventricular septal thickness, LAD: Left atrial diameter, LVEDD: Left ventricular end diastolic diameter, LVEF: Left ventricular ejection fraction, LVESD: Left ventricular end systolic diameter, PWT: Posterior Wall thickness)

Table 3.

Parameters	OR	95% CI	*P*-value
Early repolarization	14.971	2.402-93.316	0.004
Interarterial course	10.056	1.059-95.510	0.044

Multivariate logistic regerssion analysis showing independent predictors of poor cardiovascular clinical outcomes in patients with potentially serious coronary artery anomalies Abbreviations: CI: Confidence Interval; OR: Odds ratio

Cardiac imaging / Echocardiography

OP-065

Evaluation of right atrial function in patients with COVID-19 pneumonia using speckle tracking echocardiography

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Background and Aim: Subclinical biventricular systolic dysfunction in patients with COVID-19 has been recently shown. The aim of this study was to evaluate right atrial reservoir strain (RA Sr) in these patients, to compare it with age and risk-factor matched controls and to determine if it correlates with laboratory and echocardiographic parameters.

Methods: In this prospective, single-center study, data were gathered from patients treated for COVID-19 between January 1 and February 28, 2021. Two-dimensional echocardiography (2-DE) and speckle tracking echocardiography (STE) images were obtained for all patients. Patients were divided in two groups: those with COVID-19 patients and those without COVID-19 (control group). COVID-19 patients were further divided in two subgroups: severe pneumonia and non-severe pneumonia group. Data regarding clinical characteristics and laboratory findings were obtained from electronic medical records.

Results: A total of 55 patients hospitalized for COVID-19 were included in this study. The mean age of the COVID-19 group was 52 (10), 56% of whom were male. The mean age of the control group (n=30) was 51 (10), 57% of whom were male (Table 1, 2). RA Sr were decreased compared to control group (RA Sr: 36.8 [30–40] vs.42.5 [29.6-48.3], respectively [p = 0.007]) (Table 3; Figure 1). In addition, patients with severe pneumonia (n=37) had a lower RA Sr compared to patients with non-severe pneumonia (n=18) (RA Sr: 31.2 [27.0–39.0] vs 37.4 [32.8–42.3], respectively [p=0.033]) (Table 4; Fig. 1). Furthermore, right atrial strain negatively correlated with high-sensitivity troponin T (rho=-0.430; p=0.001), but positively correlated with right ventricular fractional area change and tricuspid annulus planar systolic excursion (rho=0.298, p=0.029; rho = 0.350, p=0.009) (Table 5).

Conclusions: Our study demonstrated that patients with COVID-19 pneumonia had impaired right atrial reservoir function compared to age and risk-factor matched controls.

Table 1. Baseline characteristics, clinical features, treatment and complications of patients

	COVID-19 Patients (n=55)	Control Group (n=30)	*P*-value
Age	52 (10)	51(10)	0.626
Male gender	31 (56%)	17 (57%)	0.979
BMI	29.4 (3.9)	28.2 (4.1)	0.724
Hypertension	12 (22%)	10 (33%)	0.247
Diabetes mellitus	10 (18%)	6 (20%)	0.838
Fever (°C)	36.5 [36.2–36.8]	36.4 [36.2–36.6]	0.265
SpO_2 (%)	95 [93–97]	97 [96–97]	<0.001
Respiratory rate per minute	20 [18–21]	16 [16–16]	0.002
Bilateral involvement on CT or DR	32 (58%)	-	-
Severe pneumonia	17 (31%)	-	-
Treatment			
Nasal cannula	33 (60%)	-	-
NIMV	4 (7%)	-	-
High-flow oxygen therapy	11 (20%)	-	-
Antiviral	55 (100%)	-	
Antibiotics	29 (53%)	-	-
Anticoagulant	54 (98%)	-	-
Steroid	40 (73%)	-	
Biologic agents	11 (20%)	-	-
Complications			
Admission to the intensive care unit	8 (15%)	-	-
Invasive mechanical ventilaton	2 (4%)	-	-
Acute cardiac injury	6 (11%)	-	-

Values are mean (SD) or % or median values and interquartile range, as indicated. BMI = body-mass index; NIMV = non-invasive mechanical ventilation

Table 2. Baseline laboratory findings of patients on admission

	COVID-19 Patients (n=55)	Control Group (n=30)	*P*-value
Leucocytes/μL	6980 [4360–9625]	7538 [6512–7915]	0.591
Lymphocytes/μL	1040 [663–1602]	1501 [1498–1643]	<0.001
Neutrophils/μL	5550 [2370–7700]	3512 [3199–3998]	0.011
Hemoglobin, g/dL	13 [12–13.9]	14.5 [13–15.6]	<0.001
Platelets x1000/μL	242 [181–363]	255 [171–316]	0.326
C-reactive protein, mg/dL	42 [8-90]	1 [0-2]	<0.001
Procalcitonin, ng/mL	0.06 [0.05–0.1]	-	-
High-sensitivity troponin T, pg/mL	4 [3 -9]	-	-
N-terminal pro-B-type natriuretic peptide, pg/mL	125 [54 – 312]	-	-
Creatinine, mg/dL	0.7 [0.6–0.8]	0.9 [0.8–1.0]	0.9 [0.8–1.0] <0.001
ALT, U/L	29 [22–56]	-	-
AST, U/L	31 [20–53]	-	-
Lactate dehydrogenase, U/L	278 [240–384]	-	-
D-dimer, μg/L	0.4 [0.3–1.0]	-	-
Ferritin, ng/mL	442 [210–676]	-	-

Values are mean (SD) or % or median values and interquartile range, as indicated

Table 3. Comparison of Echocardiographic Parameters among Groups

	COVID-19 Patients (n=55)	Control Group (n=30)	*P*-value
LVEF (%)	60 [60 – 64]	60 [60 – 61]	0.410
LVEDD (mm)	47 [44–51]	47 [47–49]	0.506
LVESD (mm)	31 [28–35]	31 [31–33]	0.266
IVSd (mm)	10 [9–11]	10 [9–10]	0.692
PWd (mm)	10 [9–10]	9 [9–10]	0.301
LAVI (mL/m^2)	27 [20–33]	24 [22–29]	0.073
RA area (cm^2)	14.4 [12.5–16.5]	14.2 [13.6–17.5]	0.815
RV diameter (mm)	32 [30–35]	31 [30–35]	0.296
RV FAC (%)	46 (5)	47 (5)	0.555
TAPSE (mm)	22 [20– 4]	24 [23–24]	<0.001
RVGLS (-%)	20.4 [18–23.8	25 [24– 27.8]	<0.001
RVFWS (-%)	24.0 [21–26.7]	29.5 [24–34]	<0.001
RA reservoir strain (%)	36.8 [30–40]	42.5 [29.6– 48.3]	0.007

Values are mean (SD) or % or median values and interquartile range, as indicated. IVSd = interventricular septum in diastole; LAVI = left atrial volume index; LVEDD = left ventricular enddiastolic diameter; LVESD = left ventricular endsystolic diameter; PWd = posterior wall in diastole; RVFWS = right ventricular free wall strain; RVGLS = right ventricular global strain; RV FAC= right ventricular fractional area change; TAPSE = tricuspid annular plane systolic excursion

Table 4. Comparison of Echocardiographic Parameters between Severe and Non-Severe COVID-19 Patients

	Non-severe Pneumonia Group (n=38)	Severe Pneumonia Group (n=17)	*P*-value
LVEF (%)	60 [60–64]	60 [60–63]	0.773
LVEDD (mm)	47 (5)	48 (6)	0.419
LVESD (mm)	31 (4)	32 (5)	0.893
IVSd (mm)	10 [9–11]	10 [9–11]	0.640
PWd (mm)	9 [9–10]	10 [9–10]	0.396
LAVI (mL/m2)	26 [20–33]	27 [20–31]	0.820
RA area (cm2)	14.7 [12.6– 17.8]	14.8 [11.8– 16.4]	0.591
RV diameter (mm)	33 [30–36]	30 [30 – 33]	0.096
RV FAC (%)	47 (6)	46 (7)	0.525
TAPSE (mm)	23 [21–24]	21 [20–24]	0.206
RVGLS (-%)	22.6 (4.4)	19.7 (3.4)	0.012
RVFWS(-%)	25.2 (4.4)	22.4 (4.4)	0.035
RA reservoir strain (%)	38.0 (6.1)	33.2 (7.5)	0.033

Values are mean (SD) or % or median values and interquartile range, as indicated. IVSd = interventricular septum in diastole; LAVI = left atrial volume index; LVEDD = left ventricular enddiastolic diameter; LVESD = left ventricular endsystolic diameter; PWd = posterior wall in diastole; RVFWS = right ventricular free wall strain; RVGLS = right ventricular global strain; RV FAC= right ventricular fractional area change; TAPSE = tricuspid annular plane systolic excursion

Table 5. Spearman correlation coefficients of right atrial strain, high-sensitivity troponin T and echocardiographic parameters in COVID-19 pneumonia

	RA Reservoir Strain	
	rho Value	*P*-value
High-sensitivity troponin T	-0.430	0.001
TAPSE	0.298	0.029
RV FAC	0.350	0.009
RVGLS	0.153	0.270
RVFWS	0.188	0.174

RVFWS = right ventricular free wall strain; RVGLS = right ventricular global strain; RV FAC= right ventricular fractional area change; TAPSE = tricuspid annular plane systolic excursion

Figure 1. Comparison of right atrial reservoir strain between control, non-severe and severe pneumonia

Cardiac imaging / Echocardiography

OP-066

Evaluation of patients with transcateter aortic valve implantation with 3D speckle tracking ecocardiography before and after the procedure

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Background and Aim: In patients with symptomatic severe aortic stenosis, conventional echocardiographic examinations after TAVI have been reported to improve left ventricular systolic and diastolic functions. However, apart from the traditional methods, strain echocardiographic examinations which are used for the more accurate and early detection of left ventricular functions are prominent in recent years. In our study, it was aimed to determine the early changes of the speckle tracking echocardiographic parameters beyond the conventional echocardiographic examination of these functions.

Methods: In our study we enrolled 40 patients who underwent TAVI with symptomatic severe aortic stenosis. The pa-

tients were evaluated with conventional echocardiographic and 3D speckle tracking echocardiography before and after third month of the procedure.

Results: There were no significant changes in left ventricular end-diastolic volume, left ventricular end-systolic volume, left ventricular ejection fraction, 3 months before and after the procedure (p>0.05). The left ventricular 4-space longitudinal strain (-16.5±3.5 vs. -18.7±5 p=0.04), left ventricular 3-space longitudinal strain (-16±4.2 vs. -20.2±4.8, p=0.001), left ventricular 2-space longitudinal strain (-16.9±3.8 vs. -19.4±4.2 p=0.01), left ventricular global longitudinal strain (-16.5±3.1 vs. -19.5±4.7 p=0.02) significant improvement was found. This improvement reflected in NYHA functional capacity (2.75±0.4 vs. 2.1±0.3 p<0.001) was found to be reflected in the level of Pro BNP from laboratory parameters (4602±6885 vs. 1808±2086; p=0.04).

Conclusions: Speckle tracking echocardiography showed statistically significant improvement at the end of the third month. These parameters were correlated with an increase in the functional capacity of patients and a decrease in Pro BNP levels.

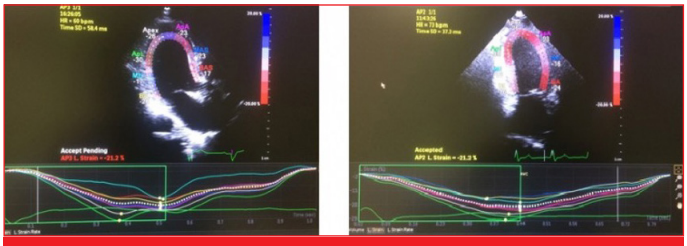

Figure 1. Left ventricular 3-chamber and 2-chamber longitudinal strain values

Table 1. Clinical characteristics (n=40)	
Age—years	76.3 ± 8.1
Female, n(%)	24(60)
Body Mass Index	26.8 ± 5.9
STS PROM Score	14.32 ± 1.3
NYHA II n(%)	10 (25)
NYHA III n(%)	30 (75)
Atrial Fibrillation	4 (10)
Chronic Obstructive Pulmonary Disease n (%)	2 (5)
Smoke n (%)	10 (25)
Diabetes mellitus n (%)	8 (20)
Hypertension n (%)	28 (70)
CABG n (%)	9 (22.5)
Previous PCI n (%)	6 (15)
Prior Stroke or TIA n (%)	0 (0)
Glomerular Filtration Rate (ml/min)	68.2 ±25.01
GFR <50 ml / min n(%)	8 (20)
Use of ACE inh /ARB n (%)	18 (45)
Use of Beta Blocker n (%)	13 (32.5)

Table 2. Conventional echocardiography data of the patients before the procedure	
EF (Simpson method)	57.4% ±7
Septum Thickness (mm)	13.6±1.8
Left Ventricular End-Systolic Diameter (cm)	3.21±0.55
Left Ventricular End-Diastolic Diameter (cm)	4.77±0.72
Posterior Wall Thickness(mm)	12.7±1.6
Left Atrium (mm)	41.7±3.5
Right Ventricle(mm)	39.03±2.67
Right Atrium (mm)	38.95±3.16

Table 3. Left ventricular strain parameters before TAVI and after 3 months

	Before TAVI		After TAVI		
Variable	Mean	Standard Deviation	Mean	Standard Deviation	*P*-value
LV 4B STRAIN	-16.5	3.5	-18.7	5	0.04
LV 3B STRAIN	-16	4.2	-20.2	4.8	<0.001
LV 2B STRAIN	-16.9	3.8	-19.5	4.7	0.01
LV GLS STRAIN	-16.5	3.1	-19.4	4.2	0.02

Table 4. Pro-BNP Levels Before TAVI and After 3 Months

	Before TAVI		After TAVI		
Variable	Mean	Standard Deviation	Mean	Standard Deviation	*P*-value
PRO-BNP	4602	6885	1808	2086	0.04

Cardiac imaging / Echocardiography

OP-067

The Frequency of Pericardial Effusion in COVID-19 Patients and Its Relationship with Mortality

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Background and Aim: Coronavirus disease-2019 (COVID-19) can impair all systems, particularly the cardiovascular and respiratory systems. Cardiovascular involvement, including myocardial damage, pericarditis, and pericardial effusion, is widespread in hospitalized COVID-19 patients. However, there is no enough data on the frequency of pericardial effusion in COVID-19 patients and its relationship with mortality; in the present study, we aimed to search the issue.

Methods: Thoracic computed tomography (CT) images of 989 consecutive COVID-19 patients, who remained after assessing the exclusion criteria, were evaluated. The presence of patients' non-physiological pericardial effusion was examined by a radiologist blinded to the study data, and the pa-

tients were divided into two groups as those with and without pericardial effusion. The presence of myocardial injury was defined according to hs-TnI levels (≥34 ng/dl). In addition, patients were analyzed in two groups according to their survival status, and mortality predictors were investigated.

Results: Pericardial effusion was observed in 125 patients (12.6%). Sixty-three (50.4%) of the patients with pericardial effusion were female, and there was no difference in terms of pericardial effusion between the genders (p=0.315). the mean age of patients with pericardial effusion was 72.8±14.3, while it was 64.8±14.9 in patients without effusion (p<0.001). Hypertension and atrial fibrillation were more frequent in patients with pericardial effusion (p<0.05), and myocardial injury was common in these patients (33.6% vs. 21.4%, p=0.023). Mortality was 36% in patients with pericardial effusion, while it was 25% in patients without effusion (p=0.009). Pericardial effusion thickness was 1.3±3.3 in the mortality group and 0.8±2.6 in the survival group (p<0.001). In the mortality group, age >65 years, male gender, hypertension, chronic obstructive pulmonary disease, chronic kidney failure, coronary artery disease, heart failure, atrial fibrillation, advanced lung parenchyma involvement in computed tomography, and myocardial injury were more common (all p values <0.05). As a result of multivariate regression analysis performed to predict mortality rate, advanced lung parenchyma involvement in computed tomography (OR=2.358, 95% CI 1.099-5.059, p=0.028), neutrophil/lymphocyte ratio (OR=1.073, 95% CI 1.028-1.120, p=0.001), being of age >65 (OR=1.829, 95% CI 1.061-3.152, p=0.030), and presence of myocardial injury (OR=2.677, 95% CI 1.551-4.623, p<0.001) were independently associated with mortality.

Conclusions: The presence of pericardial effusion in hospitalized COVID-19 patients may develop due to either a systemic inflammatory response or direct myocardial injury and increases mortality. Pericardial effusion can be used as a mortality risk predictor in COVID-19 patents, so its routine evaluation in tomography is recommended.

Table 1. Demographic and laboratory data of patient groups with and without mortality

Variables	Death (n=261)	Alive (n=728)	*P*-value
Age	74.1±12.3	62.9±145.3	<0.001
>65 y/o, n %	215 (82.4)	361 (49.6)	<0.001
Male gender, n %	157 (60.2)	375(51.5)	0.004
Hypertension n %	218 (83.5)	503 (69.1)	<0.001
Diabetes mellitus, n %	114 (43.7)	284 (39)	0.187
COPD, n %	70 (26.8)	114 (15.7)	<0.001
CKD, n %	25 (21.1)	61 (8.4)	<0.001
CAD, n %	92 (35.2)	163 (22.4)	<0.001
HF, n %	40 (15.3)	33 (4.5)	<0.001
AF, n %	41 (15.7)	63 (8.7)	0.001
Glucose, mg/dL	168.1±94.3	144.7±69.7	<0.001
GFR	53.6±29.9	71.8±23.8	<0.001
CRP, mg/L	118.2±75.9	67.5±62.1	<0.001
Troponin, ng/L	166.5±740.6	37.7±217.3	<0.001
D-dimer, mg/dL	2580.2±5167.7	1336.1±4017.1	<0.001
WBC, x10^3/µL	8.7±5.2	6.9±3.1	<0.001
Hb, g/dL	13.2±2.2	13.8±5.1	0.058
Plt, x10^3/µL	184.1±69.8	205.1±87	<0.001
Plt<100, x10^3/µL	25 (8.4)	34 (4)	0.003
Neutrophyl, x10^3/µL	7±4.5	4.8±2.8	<0.001
Lymphocyte, x10^3/µL	1.2±2.5	1.4±0.9	0.020
NLR	10.4±11.7	4.6±4.3	<0.001
Pericardial effusion, mm	1.3±3.3	0.8±2.6	<0.001
MI, n %	129 (49.4)	121 (16.7)	<0.001
CT Involvement, no, n %	15 (5.7)	70 (9.6)	0.056
CT Involvement, light, n %	51 (19.5)	239 (32.8)	0.123
CT Involvement, middle, n %	62 (23.8)	178 (24.5)	0.127
CT Involvement, high, n %	133 (51)	241 (33.1)	<0.001

Table 2. Mortality Predictors

Variables	95% CI	B	Odds ratio	*P*-value
Male gender, n %	0.971-2.597	0.463	1.588	0.065
CAD, n %	0.450-1.423	-0.223	0.800	0.448
CKD, n %	0.938-4.525	0.723	2.060	0.072
COPD, n %	0.657-2.271	0.200	1.221	0.527
HF, n %	0.443-2.228	-0.006	0.994	0.988
HF, n %	0.565-3.384	0.324	1.383	0.478
Hypertension, n %	0.485-1.680	-0.103	0.902	0.746
GFR	0.978-1.004	-0.009	0.991	0.194
D-dimer, mg/dL	1.000-1.000	0.000	1.000	0.269
WBC, x10^3/µL	0.923-1.056	-0.013	0.987	0.700
Pericardial effusion	0.626-2.334	0.190	1.209	0.572
High CT Involvement	1.099-5.059	0.858	2.358	0.028
NLR	1.028-1.120	0.071	1.073	0.001
Age>65 y/o	1.061-3.152	0.604	1.829	0.030
MI, n %	1.551-4.623	0.985	2.677	0.001
CRP, mg/L	1.551-4.623	0.003	1.003	0.097
Glucose, mg/dL	1.000-1.006	0.003	1.003	0.075
Plt<100, x10^3/µL	0.275-2.287	-0.232	0.793	0.668

Cardiac imaging / Echocardiography

OP-068

RV free wall longitudinal strain as an independent predictor of survival in wtATTR-CA patients

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Background and Aim: Wild type transthyretin cardiac amyloidosis (wtATTR-CA) is increasingly recognized as a cause

of heart failure with preserved ejection fraction (HFpEF) but prognosis is often limited due to late or misdiagnosis. Longitudinal left ventricular strain and biomarkers are established as markers of disease severity, but the role of RV free wall strain, reflecting RV contractility, is less well understood.

We sought to determine whether RV free wall strain might add incremental prognostic value in wtATTR-CA.

Methods: Consecutive patients diagnosed with wtATTR-CA with tissue confirmation at Mayo clinic between 2013 and 2015 were included. Patients with TTR gene mutations were excluded. Baseline characteristics and transthoracic echocardiography measurements were obtained from the medical records. Speckle tracking RV free wall longitudinal 2D strain and peak LA longitudinal 2D strain were measured using Tom Tec Imaging System. Survival was determined using Kaplan Meier estimates and using the cox proportional hazard ratio, univariate and multivariable analysis were performed to identify predictors of mortality in patients with wtATTR.

Results: The study group comprised 139 patients (mean age 74.9±8.6, 92.8% male), of which 102 had adequate image quality for RV strain, and 99 for LA strain. Both mean RV and LA strain were impaired at baseline: RV free wall strain was -14.7±4.9, and peak atrial longitudinal strain (PALS) was 13.2±8.8%. Using ROC analysis, RV strain of -16.8% was an independent predictor of all-cause mortality. In univariate modeling, higher levels of NT-proBNP (HR: 1.1 per 1000 pg.ml; 95%, CI 1.05-1.15, p<0.001) and Troponin T (HR: 2.0 per 0.1ng/ml; 95% CI 1.49-2.61, p<0.001) were associated with increased all-cause mortality. In addition, LV GLS (HR: 1.13 per 1%;95%CI1.04-1.24,p=0.003), RV free wall LS (HR:2.16 per 5%;95%, CI 1.57-3.03, p<0.0001), and PALS (HR:0.91 per1%; 95% CI 0.85-0.96, p<0.0001) were univariate predictors of all-cause mortality. In multivariate analysis using a stepwise regression model, RV free wall longitudinal strain (HR: 1.81; 95% CI 1.29-2.62, p< 0.001) and Troponin T (HR: 1.7; 95% CI 1.25-2.26, p=0.001) remained independent predictors. Kaplan-Meier survival analysis demonstrated a higher mortality rate above -16.8 RV strain cut-off (long-rank<0.0001, Wilcoxon<0.0001). Furthermore, all stages were divided into two groups by -16.8 % RV strain, and survival in individual stages analyzed using Kaplan-Meier survival analysis. Stage 1 and 2 with <-16.8 RV free wall strain value had higher mortality than ≤-16.8% RV strain (Stage 1: long-rank=0.0057, Wilcoxon=0.0041 and Stage 2: long-rank=0.043, Wilcoxon=0.023). However, there was not a survival difference between two RV strain groups in stage3 (long- rank=0.26, Wilcoxon=0.34)

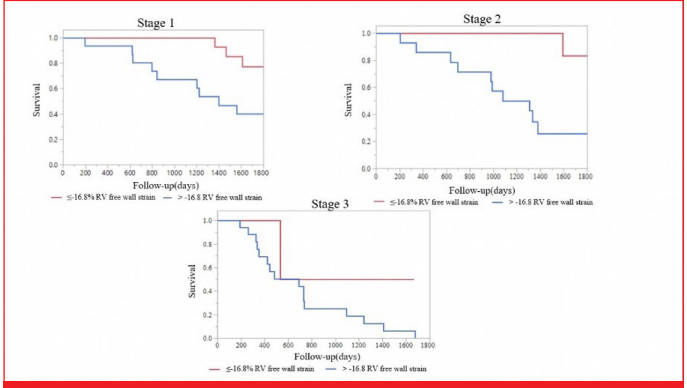

Figure 1. Kaplan Meier survival analysis in stages by -16.8% RV strain

Conclusions: RV free wall strain is an independent predictor of survival in wtATTR patients and may add incremental prognostic value to NT-proBNP and Troponin.

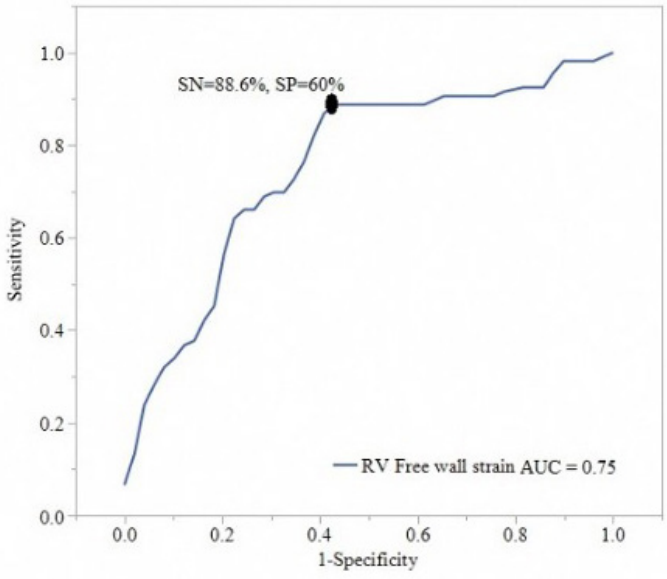

Figure 2. RV free wall strain ROC

Cardiac imaging / Echocardiography

OP-069

Left atrial abnormalities in patients with heart failure and preserved ejection fraction who have transthyretin cardiac amyloidosis

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Background and Aim: Heart failure with preserved ejection fraction (HFpEF) is associated with morphological and functional left atrial (LA) abnormalities. Transthyretin cardiac amyloidosis (TTR-CA) is reported to be present in 13-16% of HFpEF patients. In this study, we aimed to compare the left atrial abnormalities in HFpEF patients who have TTR-CA diagnosed with 99mTechnetium-pyrophosphate (99mTc-PYP) scintigraphy.

Methods: This single-center, prospective study included 95 patients who had a diagnosis of HFpEF according to 2016 ESC HF guidelines. Patients with severe primary valvular diseases, previous myocardial infarction, sarcomeric hypertrophic car-

diomyopathy or myocardial storage diseases were excluded from the study. Comprehensive echocardiography was performed in all patients. 99mTc-PYP cardiac scintigraphy was performed in 75 patients who have ≥2 red flags for TTR-CA. In the absence of monoclonal protein in the serum free light chain assay, serum and urine protein electrophoresis with immunofixation, Grade 2 to 3 myocardial uptake at 3 hours post injection of 99mTc-PYP on planar and SPECT images was considered positive for TTR-CA and Grade 0-1 uptake was considered negative. Echocardiographic findings and left atrial abnormalities were compared between both groups.

Results: Mean age of study population was 68.64±9.9 years. 99mTc-PYP cardiac scintigraphy showed that 19 patients (20% of all HFpEF) have had a grade 2 or 3 cardiac uptake and were diagnosed with TTR-CA. There was no significant difference in left atrial (LA) diameter, LA area, LA volume, LA volume index (LAVI), E/e', left ventricular global longitudinal strain (LV GLS) and LV ejection fraction (LVEF) between TTR-CA positive and negative patients (Table 1). Apical sparing was more common in TTR-CA patients. Despite similar LA diameter, area and volume; LA reservoir strain (LASr) was found to be significantly lower in patients with TTR-CA as compared to those without TTR-CA (Table 1).

Conclusions: The results of this study suggested that in HF-pEF patients with TTR-CA, LASr and apical sparing were the most important echocardiographic parameters in the prediction of TTR-CA.

Table 1.

Variables	TTR-CA negative (n=56)	TTR-CA positive (n=19)	*P*-value
Age, years	66.05±10.1	70.4±6.7	0.081
Male, (%)	23 (41.1%)	6 (31.6%)	0.326
Hypertension, n (%)	45 (80.4%)	15 (78.9%)	0.565
Prior atrial fibrillation, n (%)	17 (30.4%)	10 (52.6%)	0.072
LVEF, %	62 (57.2-63)	59.0 (54.9-62.0)	0.085
LV GLS, %	15.7 (13.9-17.3)	14.3 (11.0-16.0)	0.075
Apical sparing, n (%)	5 (9.1%)	12 (66.7%)	<0.001
LVH (≥12 mm), n (%)	38 (67.9%)	13 (68.4%)	0.600
LA diameter, mm	43.23±5.8	46.1±5.2	0.063
LA area 4C, cm^2	20.2±4.98	22.2±4.24	0.137
LA volume, mL	60.0 (50.0-83.5)	71.0 (60.0-93.0)	0.172
LAVI, (mL/m^2)	32.0 (26.0-43.0)	37.0 (33.0-46.0)	0.134
LASr strain, (%)	21.95 (9.6-28.37)	11.5 (9.8-18.3)	0.048
TPLS*, (ms)	411.2±80.5	450.6±79.5	0.096
E/e'> 15 cm/s, n (%)	12 (21.4%)	5 (26.3%)	0.519
SPAB, mm Hg	35.0 (28.0-50.0)	35.0 (30.0-52.0)	0.661
NTproBNP	940.5 (403-1837)	1008 (768-1581)	0.342

Baseline characteristics and echocardiographic features *time to peak longitudinal strain

Cardiac imaging / Echocardiography

OP-070

Left ventricular global longitudinal strain in low cardiac risk outpatients who recently recovered from Coronavirus disease 2019

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Background and Aim: The cardiac sequelae of coronavirus disease 2019 (COVID-19), a worldwide global pandemic, are still uncertain, particularly in the asymptomatic, low cardiac risk outpatient population. This study aims to evaluate the asymptomatic, low cardiac risk out-patient population who recently recovered from COVID-19, using 2-D left ventricular-global longitudinal strain (LV-GLS) proven to capable of detecting subclinical myocardial injury.

Methods: Out of 305 COVID-19 positive patients, 70 asymptomatic out-patients were determined as the study group and 70 age and sex-matched healthy adults as the control group. The echocardiographic examination did perform with the Philips IE33 system, and LV-GLS was measured using commercially available software QLAB 9 (cardiac motion quantification; Philips Medical Systems). The absolute value of LV-GLS ≤ 18 did deem to be impaired LV-GLS.

Results: The absolute values of LV-GLS were statistically significantly lower in the COVID-19 group than in healthy controls (19.17±2.65 vs. 20.07±2.19, p=0.03). The correlation between having recovered from COVID-19 and impaired LV-GLS (≤18) did detect with the Pearson correlation test (p=0.02). Having recovered from COVID-19 was found as a predictor for detecting impaired LV-GLS (≤18) in the multivariable logistic regression analysis (odds ratio, 0.133 (0.038-0.461); 95% CI, p=0.001).

Conclusions: This study suggests that COVID-19 can cause subclinical LV dysfunction detected by LV-GLS during early recovery even in a population of patients at low cardiac risk, asymptomatic, and recovered with home quarantine. The study findings indicate the long-term cardiovascular follow-up of these patients may be more important than thought.

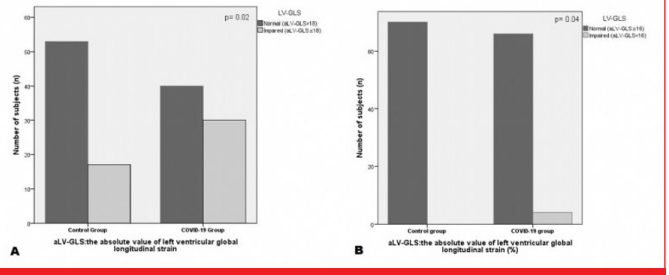

Figure 1. Compare the number of patients with impaired left ventricular global longitudinal strain between groups
A:Included the gray region of the LV-GLS cut-off value (absolute value ≤18); B: Not included the gray region of the LV-GLS cut-off value (absolute value<16).

Table 1. Multivariate analysis for detecting independent predictors of impaired left ventricular global longitudinal strain(absolute LV-GLS ≤18)

Variables	β	SE	Wald	OR (95% CI)	*P*-value
Age	-0.072	0.036	4.025	0.931 (0.868- 0.998)	0.045
Gender	1.265	0.728	3.022	3.543 (0.851-14.748)	0.082
HT	-0.846	0.764	1.225	0.429 (0.096- 1.92)	0.268
Smoking	-0.24	0.654	0.134	0.787 (0.218- 2.835)	0.714
Asthma	-1.278	0.949	1.813	0.279 (0.043- 1.79)	0.178
BMI	-0.053	0.059	0.792	0.949 (0.845-1.065)	0.373
GFR	-0.002	0.025	0.006	0.998 (0.95- 1.048)	0.937
LDL	-0.006	0.008	0.577	0.994 (0.979- 1.01)	0.447
CRP	0.057	0.045	1.553	1.058 (0.968- 1.157)	0.213
Hg	-0.15	0.237	0.402	0.86 (0.541- 1.369)	0.526
E/E'	0.133	0.16	0.689	1.142 (0.834- 1.563)	0.407
DD	-0.106	0.624	0.029	0.899 (0.265- 3.053)	0.865
Recently recovering from COVID-19	-2.121	0.625	11.51	0.12 (0.035- 0.408)	0.001

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PB-001

Mehran risk score model for predicting contrast-induced nephropathy after cardiac resynchronization therapy in patients with heart failure

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Background and Aim: We aimed to investigate the value of Mehran score (MS) to predict CIN development following cardiac resynchronization therapy (CRT) implantation.

Methods: One hundred forty-four heart failure patients who recieved CRT were enrolled to the study. Contrast induced nephropathy (CIN) is challenging conditions after cardiac procedures, which contrast media used. Mehran risk score (MS) is simple tool for predicting CIN.

Results: In this study, we showed that CIN + patients had significantly higher Mehran risk score than CIN-patients (10.4 ± 3.3, 7.6 ± 2.7, p<0.001). After multivariate logistic regression analyses, contrast volume and Mehran risk score were independently asscociated with development of CIN (OR: 1.02; (95% CI): 1.00-1.04; p=0.029, and OR: 1.34; (95% CI): 1.10-1.63; p=0.004, respectively) (Figure 1). Mehran risk score cut-off value at admission for predicting CIN in the entire study population based on receiver-operating characteristic curve (ROC) analysis was determined as 8.5, with a sensitivity of 69%, and a specificity of 71% (AUC: 0.743, 95% CI: 0.645-0.841, p<0.001) (Figure 2).

Conclusions: This study revealed that higher MS is predicting risk factor for CIN. Because of that the operator should be aware about preprocedural, intraprocedural, and postprocedural conditions for preventing CIN.

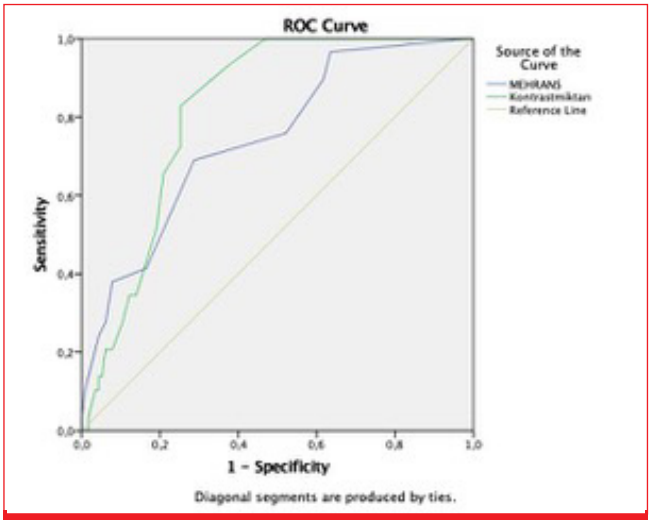

Figure 1. Mehran risk score cut-off value at admission for predicting contrast induced nephropathy based on receiver-operating characteristic curve (ROC) analysis.

Left-bundle branch block	139 (96)	28 (97)	111 (96)	0.994
Length of hospitalization, days, mean±SD	4 (2-11)	8 (4.5-17.5)	3 (1-8)	**0.001**
Contrast volume, ml, mean±SD	36 ± 24	55 ± 24	31 ± 23	**<0.001**
LVEF, %, mean±SD	25 ± 7	22 ± 6	26 ± 7	**0.005**
NYHA, n (%)				
Class I-II	112 (78)	18 (62)	94 (82)	**0.023**
Class III-IV	32 (22)	11 (38)	21 (18)	
Mehran risk score, mean±SD	8.2 ± 3.0	10.4 ± 3.3	7.6 ± 2.7	**<0.001**

Table 1. Clinical features of patients

	Univariate analysis		Multivariate analysis	
Variables	OR (95% CI)	p value	OR (95% CI)	p value
Age	1.06 (1.01-1.11)	**.021**	1.03 (0.98-1.09)	.236
Gender	1.80 (0.74-4.35)	.191	-	-
Diabetesmellitus	1.91 (0.84-4.36)	.125	-	-
Hypertension	0.62 (0.27-1.43)	.264	-	-
Coronaryarterydisease	1.71 (0.72-4.07)	.226	-	-
Dyslipidemia	1.21 (0.53-2.74)	.649	-	-
Ischemicheartfailure	1.57 (0.67-3.66)	.299	-	-
MRA	0.77 (0.30-1.93)	.573	-	-
RAAS blockers	0.87 (0.26-2.86)	.814	-	-
Contrastvolume, ml	1.03 (1.02-1.05)	**<.001**	1.02 (1.00-1.04)	**.029**
Basalcreatinin	2.85 (1.12-7.22)	**.027**	0.63 (0.17-2.31)	.490
Leftventricularejectionfraction	0.92 (0.86-0.98)	**.008**	0.94 (0.86-1.02)	.132
Mehran risk score	1.35 (1.17-1.56)	**<.001**	1.34 (1.10-1.63)	**.004**

Table 2. Independent predictors of development of contrast-induced nephropathy by logistic regression analyses

	Cut off value	AUC	Sensitivity, %	Specificity, %
Mehran risk score	8.5	0.743 (0.645-0.841)	69	71
Contrast volume, ml	43	0.819 (0.751-0.886)	65	80

AUC: Area under the curve.

Table 3. ROC curve analysis for the prediction of contrast-induced nephropathy

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PB-003

Atrial Fibrillation Predicts Long-Term Mortality in Elderly Patients Free from Heart Failure and Undergoing Hip Fracture Surgery

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Background and Aim: Previous studies have evaluated in-hospital and 1-year mortality, but there is a lack of data on long-term mortality of patients with AF who were free from heart failure.

Methods: This observational, retrospective study was done in a single research and training hospital setting. Hospital electronic health record data and National Death Registry System data for 233 consecutive patients who were above 65 years of age and were planned to undergo surgery for hip fracture were retrieved and analyzed. An experienced cardiologist evaluated the patients prior to surgery. Each member of the research cohort was categorised into 1 of 2 groups based on their survival status (survivor and non-survivor groups).

Results: 89 of 233 cases (38.2%) who were included into the investigation died during the follow-up period. The median long-term follow-up period was 34 (12-42) months. The frequency of AF was significantly higher in the non-survivor group. In multivariable COX regression analysis, AF (HR: 2.195, 95% CI: 1.365-3.415, p<0.001), advanced age and blood urea level were determined as independent predictors for all-cause long-term mortality.

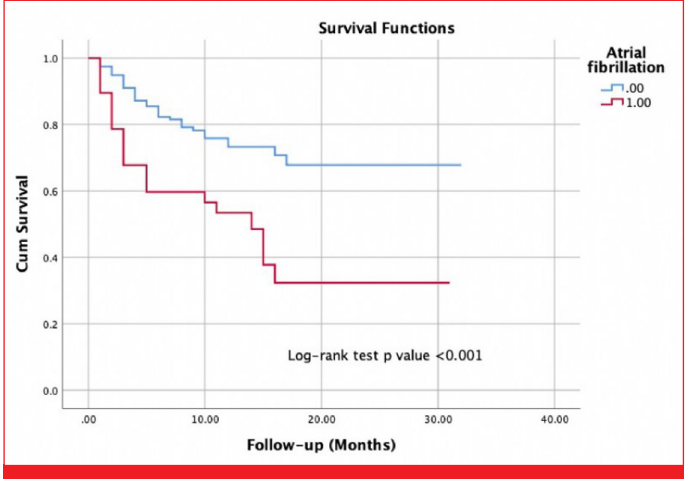

Figure 1. A Kaplan Meier curve analysis illustrating the cumulative long-term survival function of hip fracture cases based on the presence of atrial fibrillation.

Conclusions: AF is an independent predictor for long-term death in hip fracture cases over 65 years of age who were free from heart failure.

Table 1. Univariable and multivariable Cox proportional regression analyses for long-term mortality in patients operated with hip fracture a, b

	Univariable analysis		Multivariable analysis	
	p	HR (95% CI)	p	HR (95% CI)
Age	<0.001	1.079 (1.038 – 1.122)	0.011	1.044 (1.010 – 1.079)
CVA	0.032	2.929 (1.162 – 7.382)	-	-
Atrial fibrillation	<0.001	2.159 (1.365 – 3.415)	0.003	1.882 (1.220 – 2.322)
Urea	<0.001	1.014 (1.008 – 1.020)	0.027	1.008 (1.001 – 1.014)
Albumin	<0.001	0.887 (0.843 – 0.934)	-	-
LAAP diameter	<0.001	1.099 (1.051 – 1.149)	-	-

(a) All clinically relevant parameters were included in the model. (b) Only parameters that reached statistical significance at univariate analysis (P < .05) were given in the leftmost columns. HR, hazard ratio; CI, confidence interval; CVA, cerebrovascular accident; LAAP, left atrium antero-posterior.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PB-005

High grade atrioventricular block development after acute inferior ST segment elevation myocardial infarction and the importance of retrograde filling grade

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Background and Aim: Cardiovascular diseases are the leading causes of morbidity and mortality all over the world. Acute Myocardial Infarction (AMI) is the most common pathology among

these diseases. Thanks to the improvements made in recent years, AMI induced complication rates have decreased significantly. One of these complications is high-grade atrioventricular blocks (HG-AVB) that develop after AMI. In this study, we evaluated an issue that we thought had not been adequately investigated before. We examined the relationship between the presence of retrograde filling in the coronary artery, which is the cause of AMI, and the development of HG-AVB.

Methods: Our study included 339 Inferior-STEMI patients. Av conduction status was evaluated from ECG and monitor recordings of these patients. Patients with Type-II and Type-III AVB were included in the HG-AVB group. Other patients were included in the sinus rhythm group (SRG). Retrograde circulation was evaluated according to the Reentrop classification (RC). Coronary collateral circulation was graded according to the RC. Grading was performed as no filling (grade 0); filling of side branches via collateral channels without visualization of epicardial segment (grade 1); partial filling of the epicardial major coronary artery via collateral channels (grade 2); and complete filling of the epicardial major coronary artery (grade 3).

Results: In our study, the mean age was 64.1 (± 12.2) years and 230 (76.7%) of the patients were male. The dominant artery was RCA in 281 of the patients (82.9%). RCA was detected as the infarct related artery (IRA) in 198 patients (58.4%). The rate of HG-AVB in our study was 15.9% (n = 54). The number of patients implanted permanent pacemaker (PPM) was 5 (1.5%, p<0.001). The rate of retrograde filling at any stage was found to be significantly lower in the HG-AVB group (p=0.022). In addition, Reentrop 2nd and 3rd grade retrograde flow was significantly higher in the sinus rhythm group (SRG). Patients in the HG-AVB group the patients had lower systolic blood pressures (SBP), diastolic BP, LV-EF, and glomerular filtration rates. Syntax score (STXs), total cholesterol, postprandial blood glucose and creatinine levels were found to be higher in HG-AVB group. In the ROC analysis, the STXs value predicting the development of HG-AVB was determined as 26 (AUC; 0.682, Sensitivity; 62%, Specificity; 68%). The variables predicting the development of HG-AVB after regression analysis are; Age >75, STXs >26, absence of retrograde filling, RCA occlusion and hyperlipidemia were determined.

Conclusions: HG-AVB is a complication with significant consequences in patients with inferior STEMI. Permanent HG-AVB rates decreased after PCI era. Many factors are effective on the development of HG-AVB. One of them is the retrograde circulation level. A relationship was found between the absence of retrograde circulation in IRA and the development of HG-AVB. Presence of retrograde circulation was evaluated as a protective feature for Hg-AVB.

Table 1.

Variables	Odds Ratio	p
Age >75 years	2.6 (1.4-5.3)	0.048
Syntax score >26	2.9 (1.2-5.9)	0.047
Absence of Retrograde Filling	5.5 (2.2-9.1)	0.019
RCA occlusion	11.6 (4.1-18.3)	<0.001
Hyperlipidemia	3.4 (1.4-6.5)	0.034

The variables predicting the development of HG-AVB after regression analysis are; Age>75, STXs >26, absence of retrograde filling, RCA occlusion and hyperlipidemia were determined.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PB-006

The mitral inflow E/A ratio before the procedure may predict long-term recurrence after catheter ablation for atrial fibrillation

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Background and Aim: Catheter ablation is a widely used and effective method in patients with symptomatic paroxysmal or persistent atrial fibrillation (AF). Despite technological advances, arrhythmia recurrence can be seen at rates of up to 40% after a single procedure. One of the factors associated with recurrence is left ventricular (LV) diastolic dysfunction. It increases left atrial diastolic pressure and causes atrial remodelling. Its limitations, pulsed Doppler echocardiographic examination of mitral inflow is a simple and practical method commonly used to evaluate LV diastolic dysfunction. We aimed to determine whether the mitral inflow E/A ratio before the procedure was a predictor for long-term recurrence in patients undergoing catheter ablation for AF.

Methods: Patients who underwent AF ablation with second-generation cryoballoon catheter in our center were included in the study. In order to evaluate mitral inflow E and A velocities, only patients with paroxysmal AF and sinus rhythm before the procedure were included. Patients with restrictive or pseudonormal filling pattern were excluded. The patients were followed up for long-term recurrence after the procedure. Recurrence was defined as any atrial tachyarrhythmia episode (such as AF, atrial flutter, or atrial tachycardia) that lasting at least 30 seconds. Recurrences within the three-month blanking period after ablation were defined as early recurrences.

Results: The age of the patients was 58 (50-62) years and 53 (53.5%) were female. The patients were followed for 45.0 (18.0-63.0) months. AF recurrence developed in 25 patients (25.2%). In the long-term follow-up, female gender frequency (72.0% vs 47.3%), the prevalence of hypertension (72.0% vs 47.3%), and the frequency of early recurrence (20.0% vs 4.1%) were higher in those who developed recurrences than those who did not (p=0.032, 0.032 and 0.023, respectively). The CHADS2-VASC score was higher [2.0 (1.0-3.0) vs 1.0 (0-2.0)], the duration of AF was longer [27.8 ± 9.4 vs 22.3 ± 8.3], and the mitral inflow E/A ratio was lower [0.5 (0.4-1.2) vs 1.4 (0.6-1.7)] in patients with long-term recurrence than in those without it (p=0.013, 0.007 and 0.001, respectively) (Table 1). Both groups were similar in terms of procedural characteristics (procedure time, fluoroscopy time, freezing number and freezing time for each pulmonary vein) (p>0.05 for all). In multivariate analysis, female gender (HR: 4.44, 95% CI: 1.31-15.05, p=0.017), presence of early recurrence (HR: 7.35, 95% CI: 2.28-23.71, p = .001), and mitral inflow E/A ratio (HR: 0.28, 95% CI: 0.11-0.68, p=0.005) were independent predictors for long-term recurrence after ablation.

Conclusions: Female gender, early recurrence and mitral inflow E/A ratio before the procedure predict long-term recurrence after cryoballoon ablation for AF. In patients who were in sinus rhythm before catheter ablation for AF, measuring the mitral inflow E/A ratio before the procedure may help to determine the risk of recurrence in long-term follow-up.

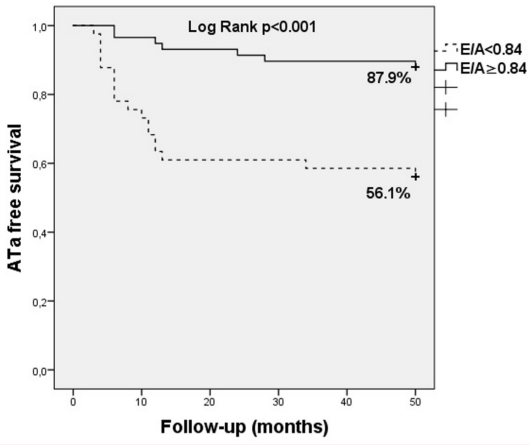

Figure 1. Kaplan-Meier survival curve showing the comparison of ATa free survival rates according to cut off mitral inflow E/A ratio of 0.84 after AF ablation. AF, atrial fibrillation; ATa, atrial tachyarrhythmia.

Table 1. Basal characteristics of patients according to the presence of long-term recurrence after cryoballoon ablation for AF

	Late recurrence (-) (n = 74)	Late recurrence (+) (n = 25)	p
Age (years)	58.0 (47.7 - 62.0)	58.0 (54.0 - 66.0)	0.329
Female gender (n, %)	35 (47.3)	18 (72.0)	0.032
Body mass index (kg/m^2)	27.2 (25.7 - 29.5)	28.6 (27.3 - 31.0)	0.149
Hypertension (n, %)	35 (47.3)	18 (72.0)	0.032
Diabetes mellitus (n, %)	15 (20.3)	8 (32.0)	0.230
HF with reduced EF (n, %)	1 (1.4)	1 (4.0)	0.443
CHA2DS2-VASC score	1.0 (0 -2.0)	2.0 (1.0 - 3.0)	0.013
AF duration (months)	22.3 ± 8.3	27.8 ± 9.4	0.007
Hemoglobin (g/dL)	13.6 (12.6 -14.7)	13.2 (11.9 - 14.2)	0.085
eGFR (mL/dk/1.73m^2)	90.2 ± 19.6	85.3 ± 18.4	0.272
Left atrial diameter (mm)	37.5 ± 3.8	38.6 ± 4.0	0.231
LV EF (%)	60.0 (60.0 - 65.0)	60.0 (60.0 - 65.0)	0.861
LV hypertrophy	10 (13.5)	5 (20.0)	0.520
Mitral inflow E/A ratio	1.4 (0.6 - 1.7)	0.5 (0.4 - 1.2)	0.001
Early recurrence (n, %)	3 (4.1)	5 (20.0)	0.023

AF, atrial fibrillation; eGFR, estimated glomerular filtration rate; EF, ejection fraction; HF, heart failure; LV, left ventricle.

Table 2. Multivariate Cox regression analysis to identify predictors of long-term recurrence after catheter ablation for AF

Parameter	Beta	HR, 95% CI	p
Female gender	1.491	4.44 (1.31-15.05)	0.017
AF duration (month)	0.036	1.03 (0.97-1.10)	0.287
Early recurrence	1.995	7.35 (2.28-23.71)	0.001
Hypertension	0.895	2.44 (0.77-7.77)	0.129
CHADS2-VASC score	-0.16	0.85 (0.55-1.30)	0.459
Mitral inflow E/A ratio	-1.26	0.28 (0.11-0.68)	0.005

AF, atrial fibrillation.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PB-007

Comparison of long-term follow-up of bipolar and quadripolar left ventricular leads in Cardiac Resynchronization Therapy implantation

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Background and Aim: Quadripolar left ventricular leads (QdP LVL) were be associated with more favourable clinical and procedural outcomes in recent years. We aimed to investigate the long-term effects of unipolar and bipolar leads in patients undergoing cardiac resynchronization therapy (CRT) implantation due to heart failure.

Methods: In this retrospective study, we enrolled 84 patients receiving CRT with bipolar (BiP, n=48) and QdP (n=36) LVL. Patients were followed for 12 months, and outcomes were compared concerning CRT response (defined as ≥10% absolute increase in left ventricle ejection fraction [LVEF]), electrocardiographic, echocardiographic parameters, NYHA functional class improvement.

Results: The mean age of patients with QdP was 63.2 ± 8.7, BiP was 63.0 ± 10.1 years. CRT with QdP was associated with a better response rate as compared to bipolar pacing. Patients with ≥1 NYHA improvement was 86.1% vs 58.3%, p=0.006 and LVEF ≥10% was 80.6% vs 58.3%, p=0.031.QRS duration (140.4 ± 11.7 vs 151.7 ± 19.4 ms, p=0.018) and cQT interval (461.1 ± 57.2 vs 482.7 ± 54.6 ms, p=0.037) were narrower in those with QdP leads. The univariable regression analysis revealed that LVEF (OR: 1.082 95% CI [1.005-1.165], p=0.037), QRS duration (OR: 0.980 95% CI [0.961–0.999], p=0.038), NYHA class (OR: 1.107 95% CI [0.075–0.682], p=0.008) and LVEF improvement (OR: 2.959 95% CI [1.083-8.086], p=0.034) were a potential risk factors for favourable response to CRT with QdP lead.

Conclusions: It was observed that LVEF and NYHA class improvement and QRS and cQT interval narrowing were better in those who were applied QdP lead. QdP leads were found to be associated with more favourable clinical outcomes than bipolar leads.

Table 1. Demographic, clinical, and electrocardiographic characteristics of patients receiving CRT with bipolar and quadripolar left ventricular leads

Variables	Quadripolar lead (n = 36)	Bipolar lead (n = 48)	p
Age, years	63.2 ± 8.7	63.0 ± 10.1	0.896
Hypertension, n %	13 (36.1)	17 (35.4)	0.278
Diabetes mellitus, n %	11 (30.5)	13 (27.0)	0.278
ischemic CMP, n %	12 (33.3)	15(31.2)	0.432
After 12 months follow-up			
NYHA class improvement ≥1	16 (44.4)	19 (39.6)	0.036
LVEF improvement	39.9 ± 5.5	36.5 ± 7.5	0.028
QRS duration, ms	140.4 ± 11.7	151.7 ± 19.4	0.018
cQT interval, ms	461.1 ± 57.2	482.7 ± 54.6	0.037

Table 2. The association between quadripolar left ventricular lead and improvement of LVEF, NYHA, electrocardiographic parameters with logistic regression analyses

After 12 months follow-up	Univariable analyses			Multivariable analyses		
	OR	95% CI	p	OR	95% CI	p
LVEF	1.82	1.005-1.165	0.037	1.031	0.937-1.132	0.528
QRS duration	0.980	0.961-0.999	0.038	0.984	0.959-1.010	0.217
cQT interval	0.991	0.982-1.000	0.062	0.992	0.981-1.003	0.155
NYHA improvement	1.107	0.075 - 0.682	0.008	0.989	0.751-1.084	0.765
LVEF improvement	2.959	1.083-8.086	0.034	0.994	0.984-1.067	0.846

PB-008 [Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD]

Assessment of novel atrial fibrillation predictors in obese patients performed bariatric surgery

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Background and Aim: Bariatric surgery has been associated with reduced cardiovascular mortality and morbidity in obese patients. In this study, we aimed to evaluate the risk of developing atrial arrhythmia, especially atrial fibrillation (AF), in patients who achieved effective weight loss with bariatric surgery.

Methods: The study included 58 patients who underwent bariatric surgery. We measured heart rate, PR, P max, P min, Average P axis, PWPTD2, PWPTV1 and V1TF, and we estimated PWdis interval both pre-operation and six months after operation.

Results: Heart rate, PR, P max, P min, PWdis, Average P axis, PWPTD2, PWPTV1 and V1TF values, which were close to the upper limit in the pre-op period, showed statistically significant decreases at six months after operation.

Conclusions: The results of our study indicated that bariatric surgery has positive effects on the regression of ECG parameters which are predictors of atrial arrhytmias such as AF.

Baseline characteristics (n=58) Age: 42.38 ± 12.68 Male sex: 31 (53.4%) Smoker: 18 (31%)	Pre-op	Post-op (6.month)	p value
Weight; kilogram	131.14 ± 23.301	92.67 ± 17.965	p<0.001*
Body mass index	47.08 ± 6.617	33.42 ± 5,696	p<0.001*
Diabetes Mellitus; n (%)	14 (24.1 %)	3 (5.2 %)	0.002*
Hypertension; n (%)	22 (37.9 %)	11(19 %)	p<0.001*

Table 1. Demographic characteristics of the study groups

(n = 58)	Pre-op	Post-op (6. month)	p value
P wave parameters			
PR; ms	154.69 ± 23.67	151.36 ±19.48	0.033*
PW max; ms	120.54 ± 11.35	110.28 ± 9.00	<0.001*
PW min; ms	69.39 ± 9.21	61.49 ± 8.66	<0.001*
PW dis; ms	51.15 ± 9.70	48.79 ± 9.50	0.010*
PWPTD2; ms	55.75 ± 6.91	50.59 ± 7.67	<0.001*
PWPTV1; ms	54.10 ± 7.06	48.05 ± 7.64	<0.001*
Abnormal P wave axis; n(%)	10 (17.2%)	3 (5.2%)	0.016*
Biphasic P wave (+/-); n(%)	7 (12.1%)	5 (8.6%)	0.500
Average P wave axis^0	39.35 ± 23.05	39.10 ± 21.86	0.825
V1TF > 40; n(%)	25 (43.1%)	12 (20.7%)	<0.001*
Other ECG parameters			
Heart rate, bpm	81 ± 13.19	68.71 ± 10.55	<0.001*
QRS; ms	92.86 ± 13.39	92.36 ± 12.09	0.452
QTc max; ms	446.01 ± 30.89	407.64 ± 28.76	<0.001*
cQTd; ms	50.56 ± 7.12	32.28 ± 6.92	<0.001*

bpm: beat per minute, ms: millisecond, PW max: maximum P wave, PW min: minimum P wave, P wave dispersion (PW dis) was determined as the difference between the maximum and minimum P wave, PWPTD2: from the beginning of P wave to peak in lead D2. PWPTV1 from the beginning of P wave to peak in lead V1, V1TF: P wave terminal force in lead 1, QTc max: corrected QT max, cQTd: cQT dispersion (QTd) was determined as the difference between the maximum and minimum QTc interval.

Table 2. Electrocardiographic findings of the study population

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PB-010

Effect of cardiac rehabilitation on atrial conductions following isolated coronary artery bypass surgery

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Background and Aim: Cardiac rehabilitation (CR) is associated with improved clinical outcomes in a broad spectrum of cardiac disease. The beneficial effect of CR has been proven in patients with ischemic heart diseases. Post-operative atrial fibrillation (AF) and atrial arrhythmias are common complications following coronary artery bypass grafting (CABG) surgery. The aim of this study was to evaluate the effect of CR on atrial conductions in patients who underwent isolated CABG surgery.

Methods: After the exclusion criterion was applied 545 patients were included in the study, retrospectively. One group (Rehab +) included patients who participated in CR program and the other group (Rehab -) included patients who did not want to or could not be participated in CR program after isolated CABG surgery. ECG parameters of Pmax, Pmin and P wave dispersion (PWD) of both groups were compared.

Results: All parameters including heart rate, PR interval, Pmax, Pmin and PWD were significantly changed in rehab (+) group after CABG surgery. There was significant change in rehab (+) group when compared to rehab (-) group in terms of parameters of Pmax (p<0.001), Pmin (p<0.001), P wave dispersion (p<0.001).

Conclusions: Our findings referred that CR program significantly improved parameters of atrial conduction including Pmax, Pmin and PWD following isolated CABG surgery. Thereby improvement in atrial conduction by CR program following CABG surgery could reduce atrial arrhythmias.

	Patients who did not undergo Cardiac Rehabilitation (n=255)	Patients who underwent Cardiac Rehabilitation (n=290)	P value
Age (years)	59.2 ± 9.5	59.9 ± 8.4	0.332
Gender (male -%)	196 (76.9)	225 (77.6)	0.841
HFrEF (n-%)	19 (7.5)	16 (5.5)	0.358
PAD (n-%)	14 (5.5)	34 (11.7)	**0.010**
Smoking (n-%)	93 (36.5)	93 (32.1)	0.280
DM (n-%)	144 (56.5)	163 (56.2)	0.951
Hypertension (n-%)	189 (74.1)	240 (82.8)	**0.014**
Previous MI (n-%)	33 (12.9)	52 (17.9)	0.109
LVEF (%)	59 (50-60)	60 (50-60)	0.059
Stroke (n-%)	21 (8.2)	17 (5.9)	0.278
Creatinine (mg/dL)	0.87 (0.74-1.04)	0.90 (0.77-1.05)	0.350
Heart Rate (/min)	81 (71-89)	78 (71-87)	0.132
PR interval (ms)	144 (130-160)	148 (132-164)	0.136
P wave max (ms)	96 (90-102)	94 (88-102)	0.104
P wave min (ms)	72 (66-76)	71 (65-77)	0.205
P wave dispersion (ms)	24 (20-28)	23 (19-27)	0.079

Table 1.

Rehab (+) group (n=290)	Before CABG surgery	After Cardiac Rehabilitation following surgery	P value
Heart Rate (/min)	78 (71-87)	71 (65-79)	**<0.001**
PR interval (ms)	148 (132-164)	154 (140-170)	**<0.001**
P max (ms)	94 (88-102)	94 (86-100)	**0.033**
P min (ms)	71 (65-77)	78 (74-86)	**<0.001**
P wave dispersion (ms)	23 (19-27)	12 (10-16)	**<0.001**

Table 2.

Rehab (-) group (n=255)	Preoperative	Postoperative	P value
Heart Rate (/min)	81 (71-89)	72 (65-83)	**<0.001**
PR interval (ms)	144 (130-160)	148 (134-167)	**<0.001**
P max (ms)	96 (90-102)	96 (90-102)	0.214
P min (ms)	72 (66-76)	72 (66-76)	0.970
P wave dispersion (ms)	24 (20-28)	24 (20-30)	0.428

Table 3.

	Rehab (-) group	Rehab (+) group	P value
Heart Rate (/min)	72 (65-83)	71 (65-79)	0.305
PR interval (ms)	148 (134-167)	154 (140-170)	0.058
P max (ms)	96 (90-102)	94 (86-100)	**<0.001**
P min (ms)	72 (66-76)	78 (74-86)	**<0.001**
P wave dispersion (ms)	24 (20-30)	12 (10-16)	**<0.001**

Table 4.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PB-011

Assessment of Tp-e interval, Tp-e/QT and Tp-e/QTc ratios in patients with acromegaly

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Background and Aim: Cardiovascular complications, including ventricular arrhythmias associated with abnormalities of

ventricular repolarization are the leading cause of morbidity and mortality in patients with acromegaly. Herein, we aimed to investigate ventricular repolarization using Tp-e interval, Tp-e interval/QT and Tp-e interval/QTc ratios in acromegalic patients compared to healthy subjects.

Methods: A total of 29 patients (aged 51.9 ± 11.2, 65.5% women) with acromegaly and 30 control subjects (aged 47.3 ± 14.4, 63.3% women) were enrolled into the study. Tp-e and QT interval, corrected QT (QTc), Tp-e/QT and Tp-e/QTc ratios were calculated from 12-lead electrocardiogram.

Results: Tp-e interval (89.28 ± 12.16 vs 75.97 ± 9.92 ms; p<0.001), Tp-e/QT ratio (0.237 ± 0.045 vs 0.212 ± 0.029; p=0.019) and Tp-e/QTc ratio (0.218 ± 0.031 vs 0.195 ± 0.026; p=0.003) were significantly higher in patients with acromegaly compared to control group. A positive correlation was determined between LAVI and Tp-e interval (r = 0.272, p=0.039).

Conclusions: The current study is the first to have shown significantly increased Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio were increased in acromegalic patients. These results may be important for screening malignant arrhythmic events in acromegaly.

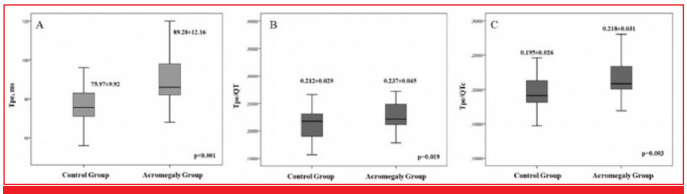

Figure 1.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PB-012

Prognostic value of serum albumin for long-term mortality in patients with dual chamber permanent pacemakers

Mert İlker Hayıroğlu[1], Tufan Çınar[2], Göksel Çinier[1], Gizem Yüksel[1], Levent Pay[1], Kıvanç Keskin[1], Cahit Çoşkun[1], Gökçem Ayan[1], Vedat Çiçek[2], Kazım Serhan Özcan[1], Ceyhan Türkkan[1], Nazmiye Özbilgin[1], Ahmet İlker Tekkeşin[1], Ahmet Taha Alper[1], Kadir Gürkan[1]

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Background and Aim: This investigation aims to examine the prognostic utility of albumin concentrations for long-term all-cause mortality in patients undergoing permanent pacemaker (PPM) implantation.

Methods: In this observational, retrospective study, PPM implanted 1798 patients were divided into quartiles according to serum albumin concentrations. The significance of albumin to predict long-term mortality was evaluated comparing these quartiles. The independent predictors of all-cause mortality were also identified.

Results: Multivariable cox-regression analysis revealed that age, complete atrioventricular block, urea, albumin (HR: 0.356, 95% CI: 0.286-0.443, p<0.001), left ventricle ejection fraction, and left atrium anteroposterior were independent predictors of the long-term all-cause mortality in cases with PPM. The long-term mortality had the higher rates in the Q4 group compared to the Q1-3 groups (n = 224 cases (49.9%) vs n = 213 cases (15.8%), respectively). The risk of the long-term mortality in the Q4 group was 3.6 times higher than Q1-3 groups and after the adjustment for the confounders, Q4 group had 1.7 times higher rates of the long-term mortality compared to Q1-3 groups.

Conclusions: Serum albumin level at the time of device implantation has a great value for the long-term mortality in patients with PPM.

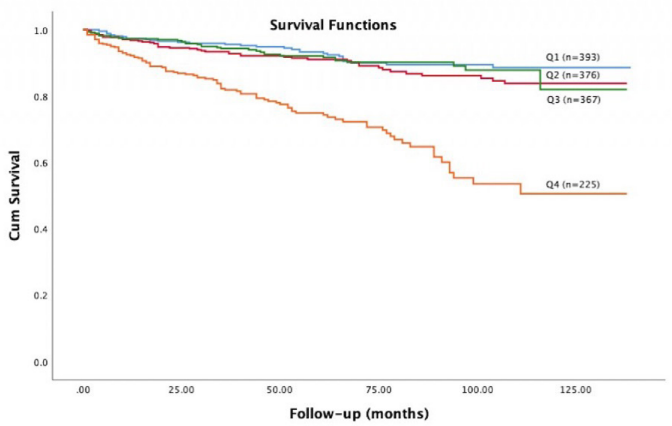

Figure 1. The probability of the long-term survival function was significantly lower in the Q4 groups compared to the Q1-3 groups (the log-rank test = 101.91, p<0.001).

Table 1. Cox-regression models for long-term mortality by quartiles stratified by albumin

	Q1-3 (n = 1349)	Q4 (n = 449)
Long-term mortality		
Number of patients	213	224
Case rate, %	15.8	49.9
Long-term mortality, HR (95% CI)		
Model 1: unadjusted	1 [Reference]	4.6 (3.8–5.6)
Model 2: adjusted for all covariates*	1 [Reference]	2.7 (2.1–3.4)

*Adjusted for; age, gender, complete AV block, urea, ejection fraction and left atrium anteroposterior.
CI, confidence interval; OR, odds ratio.

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PB-013

The effect of the severity of COVID-19 infection on electrocardiographic findings

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Background and Aim: Acute myocardial damage is detected in a significant portion of patients with coronavirus 2019 disease (COVID-19) infection, with a reported prevalence of between 7% and 28%. The aim of this study was to investigate the relationship between electrocardiographic findings and the indicators of the severity of COVID-19 detected on electrocardiography (ECG).

Methods: A total of 219 patients that were hospitalized due to COVID-19 between April 15 and May 5, 2020 were enrolled in this study. Patients were divided into two groups according to the severity of COVID-19 infection: severe (n = 95) and non-severe (n = 124). ECG findings at the time of admission were recorded for each patient. Clinical characteristics and laboratory findings were retrieved from electronic medical records.

Results: Mean age was 65.2 ± 13.8 years in the severe group and was 57.9 ± 16.0 years in the non-severe group. ST depression (28% vs 14%), T-wave inversion (29% vs 16%), ST-T changes (36% vs 21%), and the presence of fragmented QRS (fQRS) (17% vs 7%) were more frequent in the severe group compared to the non-severe group. Multivariate analysis revealed that hypertension (odds ratio [OR]: 2.42, 95% confidence interval [CI]:1.03-5.67; *P* = .041), the severity of COVID-19 infection (OR: 1.87, 95% CI: 1.09-2.65; p=0.026), presence of cardiac injury (OR: 3.32, 95% CI: 1.45-7.60; p=0.004), and d-dimer (OR: 3.60, 95% CI: 1.29-10.06; p=0.014) were independent predictors of ST-T changes on ECG.

Conclusions: ST depression, T-wave inversion, ST-T changes, and the presence of fQRS on admission ECG are closely associated with the severity of COVID-19 infection.

	Severe (n = 95)	Non-severe (n = 124)	*p*
Age (years)	65.2 ± 13.8	57.9 ± 16.0	0.001
Male, n (%)	61 (64%)	69 (56%)	0.226
BMI (kg/m^2)	27.3 ±6.3	26.8 ±5.4	0.654
Chronic medical illness			
HT, n (%)	51 (53%)	59 (47%)	0.371
DM, n (%)	27 (28%)	39 (31%)	0.628
HLD, n (%)	19 (20%)	17 (13%)	0.213
Laboratory findings at admission			
Hemoglobin (g/dl)	11.5 ±2.3	12.3 ±1.6	0.002
WBC (10^3 /µl)	10.0± 4.4	6.7± 2.8	<0.001
Creatinine (mg/dl)	0.9 (0.7-1.4)	0.7 (0.6-0.9)	<0.001
Sodium (mmol/L)	136.4 ± 5.8	137.8 ± 3.5	0.031
Potassium (mmol/L)	4.2 ± 0.6	4.3 ± 0.7	0.328
Glucose (mg/dL)	163.4±78.3	121.2±47.8	<0.001
CRP (mg/dL)	106 (57-175)	40 (11-97)	<0.001
Prokalsitonin (ng/mL)	0.38 (0.13-1.44)	0.12 (0.12-0.17)	<0.001
hs-TnI(pg/ml)	63 (16-225)	6(3-11)	<0.001
D-dimer (ng/mL)	690 (400-2800)	658 (440-992)	<0.001
CK-MB (ng/mL)	3.2 (1.7-7.0)	1.0 (0.5-1.5)	<0.001
Clinical outcome			
ICU, n (%)	89 (94%)	-	
Non-ICU, n (%)	6 (6%)	124(100%)	<0.001
MV, n (%)	86 (90%)	-	

Abbreviations: BMI, Body mass index; HT, hypertension; DM, diabetes mellitus; HLD, hyperlipidemia; WBC, white blood cell, CRP, C-reactive protein; hs-TnI, high sensitive Troponin I; CK, creatinine kinase; ICU, intensive care unit; MV, mechanical ventilation.

Table 1. Demographic and clinical characteristics

	All patients (n=219)	Severe (n=95)	Non-severe (n=124)	p
Heart rate, (bpm)	91.9± 17.9	95.3±20.0	89.3±15.8	0.019
HR >100 bpm, n(%)	69 (31%)	39 (41%)	30 (24%)	0.008
PVC, n (%)	10 (5%)	3 (3%)	7 (6%)	0.382
PAC, n (%)	9 (4%)	6 (9%)	3 (2%)	0.150
PR interval, (ms)	155.0 ± 23.0	155.2±23.0	154.8±23.0	0.907
PR<120 ms, n (%)	10 (4%)	4 (%5)	6 (5%)	
PR 120-200 ms, n (%)	184 (84%)	79 (94%)	105 (91%)	
PR>200 ms, n (%)	5 (2%)	1 (1%)	4 (3%)	
PR depression, n (%)	6 (3%)	3 (3%)	3 (2%)	
QRS duration, (ms)	94.5±19.8	94.8±21.9	94.2±18.2	0.844
QRS>120ms, n (%)	32 (14%)	18 (19%)	14 (11%)	0.112
QTc interval (ms)	433.5±27.5	435.6±33.0	431.8±22.8	0.322
QTc>500ms, n (%)	12 (5%)	9 (9%)	3 (2%)	0.034
RBBB, n (%)	11 (5%)	3 (3%)	8 (6%)	0.357
IVCD, n (%)	11 (5%)	6 (6%)	5 (4)	0.538
fQRS, n (%)	26 (12%)	17 (17%)	9 (7%)	0.016
fQRS inferior, n (%)	20 (9%)	14 (14%)	6 (5%)	0.022
fQRS lateral, n (%)	6 (3%)	3 (3%)	3 (2%)	0.981
ST elevation, n (%)	10 (4%)	6 (6%)	4 (3%)	0.150
ST depression, n (%)	45 (20%)	27 (28%)	18 (14%)	0.012
ST depression anterior, n (%)	4 (3%)	0 (0%)	4 (3%)	
ST depression inferior, n (%)	18 (8%)	13 (13%)	5 (4%)	
ST depression lateral, n (%)	20 (9%)	11 (11%)	9 (7%)	
ST depression common, n (%)	3 (2%)	3 (3%)	0 (0%)	
T inversion, n (%)	48 (22%)	28 (29%)	20 (16%)	0.018
T inversion anterior, n (%)	16 (8%)	9 (9%)	7 (6%)	
T inversion inferior, n (%)	12 (5%)	10 (11%)	2 (1%)	
T inversion lateral , n (%)	20 (9%)	9 (9%)	11 (9%)	
ST-T change, n (%)	62 (28%)	35 (36%)	27 (21%)	0.014

Abbreviations: HR, heart rate; PVC, premature ventricular contractions; PAC, premature atrial contraction; QTc, corrected QT; RBBB, right bundle branch block; IVCD, intraventricular conduction delay; fQRS, fragmented QRS.

Table 2. Electrocardiographic findings

Arrhythmia / Electrophysiology / Pacemaker / CRT-ICD

PB-014

Evaluation of ventricular electrophysiological balance index in COVID-19 patients with SIRS

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Background and Aim: Malignant ventricular arrhythmia is an important cause of mortality in COVID-19 patients (1-3). In our study, we aimed to investigate the cardiac electrophysiological balance index (ICEB), which predicts the risk of malignant ventricular arrhythmia in patients with COVID-19 who developed SIRS (systemic inflammatory response syndrome).

Methods: After exclusion criteria (atrial fibrillation, left bundle branch block, pre-excitation), a total of 533 COVID-19 patients, of whom 197 (37%) were SIRS, were included in the study.

Results: The average age in the study population was 62 (49-72), and the gender distribution was 49% (261) female, 51% (272) male. The patients were divided into two groups as the control group with SIRS and the control group without SIRS. The clinical, laboratory and demographic characteristics of the patients were compared in Table 1. The QTc/QRS ratio (ICEBc) in the SIRS group was 5.1 (4.64-5.1) and was significantly higher than 4.98 (4.5-5.45) in the control group (p=0.004). The QTc interval was 450 (422-474) and 427 (407-447) significantly longer in the SIRS group than the control group (p=0.001). As a result of multivariable linear regression analysis, a significant correlation was found between ICEBc and SIRS, age, gender and CRP.

Conclusions: Malign ventricular arrhythmias developing in COVID-19 patients are an important cause of mortality. ICEBc and QTc were significantly higher in the SIRS group than in the control group. It was thought that ICEBc could be used to predict malignant ventricular arrhythmias in the patient group developing SIRS.

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

	Total (n = 533)	SIRS (n = 197)	Control (n = 336)	p
Age, years	62 (49-72)	65 (53-73	46 (60-71)	0.16
Gender (female), n (%)	261 (49%)	84 (43%)	178 (53%)	0.21
Hypertension, n (%)	197 (39%)	84 (43%)	123 (37%)	0.168
Diabetes mellitus, n (%)	140 (26%)	59 (30%)	81 (124%)	0.139
Chronic renal failure, n (%)	23 (4%)	17 (9%)	6 (2%)	<0.001
Congestive heart failure, n (%)	18 (3%)	9 (5%)	9 (3%)	0.359
Coronary artery disease, n (%)	81 (15%)	34 (17%)	47 (14%)	0.310
Chronic respiratory disease, n (%)	24 (5%)	8 (4%)	16 (5%)	0.465
Myocardial injury, n (%)	33 (6%)	24 (12%)	9 (3%)	<0.001
mortality	110 (21%)	90 (46%)	6 (27%)	<0.001
Beta blocker use, n (%)	69 (13%)	34 (17%)	35 (10%)	0.024
Intensive care unit admission, n(%)	140 (26%)	111 (56%)	29 (9%)	<0.001
Loop diuretic use, n (%)	11 (2%)	3 (2%)	8 (2%)	0.754
Temperature, °C	36.7 (36.4-36.9)	36.8 (36.4-37.3)	36.6 (36.4-36.8)	<0.001
Systolic blood pressure, mm Hg	120 (110-130)	120 (110-130)	120 (110-125)	0.151
Diastolic blood pressure, mm Hg	70 (65-80)	70 (67-80)	70 (60-80)	0.053
White blood cell, 10^3/uL	6.94 (5.19-9.51)	7.96 (4.86-12.7)	6.59 (5.25-8.43)	<0.001
Hemoglobin, g/dL	13.3 (12.2-14.4)	13.1 (12-14.35)	13.4 (12.4-14.4)	0.703
C-reactive protein, mg/L	80 (35.9-129.4)	105 (48.5-168.7)	65.1 (28.3-113.4)	<0.001
Procalsitonin, ng/mL	0.11 (0.05-0.27)	0.18 (0.07-0.59)	0.1 (0.04-0.18)	0.98
D-dimer, ng/mL	274 (168-443)	352 (204-680)	240 (162-351)	0.246
Creatinine, mg/dL	0.91 (0.76-1.18)	0.97 (0.77-1.4)	0.9 (0.75-1.11)	0.063
Albumin, g/L	33 (29--36)	31 (28-35)	34 (31-37)	<0.001
Aspartate aminotransferase, IU/L	32 (23.5-47)	37 (24-50)	29 (23-45)	0.03
Alanine aminotransferase, IU/L	24 (17-39)	28 (18-43)	22 (16-36)	0.07
Corrected calcium, mg/dL	8.74 (8.4-9.1)	8.78 (8.36-9.12)	8.71 (8.4-9.04)	0.717
Potassium, mmol/L	4.14 (3.82-4.49)	4.13 (3.79-4.52)	4.14 (3.84-4.45)	0.832

Table 2. Electrocardiographic findings of patients

	Total (n = 533)	SIRS (n = 197)	Control (n = 336)	p
Heart rate, beat/min	92 (82-100)	98 (93-106)	86 (78-96)	<0.001
QRS duration, ms	85 (80-96)	86 (80-100)	85 (80-95)	0.46
QT interval, ms	360 (330-380)	346 (320-380)	360 (340-380)	0.02
QTc interval, ms	435 (413-458)	450 (422-474)	427 (407-447)	<0.001
QTc/QRS ratio	5.02 (4.56-5.5)	5.1 (4.64 – 5.1)	4.98 (4.5-5.45)	0.004
Tp-e interval	80 (80-94)	80 (80-95)	80 (80-93)	0.963
QT/QRS ratio	4 (3.65-4.45)	4 (3.6-4.46)	4.1 (3.75-4.5)	0.022
Tp-e/QT ratio	0.235 (0.211-0.264)	0.236 (0.213-0.267)	0.234 (0.210-0.263)	0.162
Tp-e/QTc ratio	0.192 (0.171-0.215)	0.186 (0.164-0.208)	0.194 (0.175-0.220)	<0.001

Table 3. Multivariable linear regression analysis between the İCEBc (QTc/QRS) and clinical variable

	β-coefficient	CI 95%	p
Age (year)	2.265	0.001-0.009	0.024
Gender (female)	2.079	0.008-0.27	0.018
CHF	-0.718	-0.52-0.24	0.473
CRP	2.077	0.001-0.002	0.038
SIRS	2.372	0.028-0.302	0.018
Myocardial injury	0.560	-0.193-0.347	0.575
CAD	1.446	-0.55-0.364	0.149
Beta blocker	-0.220	-0.243-0.194	0.83
Corected Calcium(mg/dL)	-1.345	-0.192-0.36	0.179
Potassium (mmol/L)	0.811	-0.073-0.175	0.418
CRF	0.723	-0.205-0.444	0.47

Epidemiology

PB-015

The impact of daily Troponin-I and D-dimer serum levels on mortality of moderate-to-severe COVID-19 pneumonia patients

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Background and Aim:The aim of this study is to analyse the daily Troponin-I and D-dimer levels and their impact on the need for intensive care and mortality of the COVID-19 infected patients.

Methods: 206 patients who were hospitalized between March 20, 2020-May 5, 2020 with a diagnosis of moderate-to-severe COVID-19 pneumonia were analysed retrospectively. Serum Troponin-I and D-dimer levels were recorded at least 10 days.

Results: Average age was higher in mortality group compared to non-mortality group (respectively 67.79 ± 14.9, 56.87 ± 18.15, p<0.001). Presence of hypertension, diabetes mellitus, previous coronary bypass surgery, heart failure, chronic renal failure and chronic obstructive pulmonary disease were statistically significant affecting mortality (respectively p:0.003, p:0.004, p:0.045, p:0.02, p:0.003, p:0.007). First 10 days measurements of Troponin-I and D-dimer values was associated with mortality and intensive care requirement (p<0.001). Both Troponin-I and D-dimer were higher in mortality group compared to the patients requiring intensive care. Troponin-I value on the 7th day ≥16.05 pg/mL was related with need for intensive care (AUC: 0.896, sensitivity: %78.6, specificity: %78.3, p<0.001). Troponin-I value ≥30.25 pg/mL on the 9th day was related with mortality (AUC: 0.920, sensitivity: %89.5, specificity: %89.3, p<0.001). D-dimer value ≥878 hg/mL on the 2nd day was associated with intensive care need (AUC: 0.896, sensitivity: %78.6, specificity: %78.3, p<0.001). D-dimer value ≥1106 hg/mL on the 10th day was associated with mortality (AUC: 0.817, sensitivity: %68.4, specificity: %65.2, p<0.001). It was observed that hospitalization periods ≥9.5 days were associated with mortality (AUC: 0.738, sensitivity: %68.4, specificity: %65.9, p<0.001).

Conclusions: We showed that hospitalizations ≥9.5 days increased mortality. Troponin-I and D-dimer follow-ups in serum are more effective than other inflammatory markers to show the need for intensive care and mortality. A high Troponin-I value should alert the clinician in terms of clinical deterioration.

Table 1. Relation between demographic characteristics and mortality

Demographic characteristics	All patients (n=205)	Mortality (-) (n=167, %)	Mortality (+) (n=38, %)	p
Age (years)	58.81 ± 18.07	56.87 ± 18.15	67.79 ± 14.9	<0.001
Gender (female)	109 (52.9)	88 (52.1)	21 (55.3)	0.857
Hypertension	65	45 (26.9)	20 (52.6)	0.003
Diabetes mellitus	40	28 (16.8)	12 (31.6)	0.04
PCI*	17	12 (7.2)	5 (13.2)	0.323
CABG#	5	2 (1.2)	3 (7.9)	0.045
Heart failure	12	5 (3.0)	7 (18.4)	0.02
Chronic renal failure	7	2 (1.2)	5 (13.2)	0.003
Atrial fibrillation	15	12 (7.2)	3 (7.9)	1
COPD†/Asthma	18	10 (6)	8 (21.1)	0.07
Cerebrovascular disease	3	2 (1.2)	1 (2.6)	0.46

Table 2. Relationship between daily Troponin-I values with mortality and intensive care needs

Daily Troponin-I (pg/mL)	Mortality (-)	Mortality (+)	P value	Intensive care (-)	Intensive care (+)	p
Troponin-I 1	115 ± 703	744 ± 3211	<0.001	50 ± 293	696 ± 2585	<0.001
Troponin-I 2	999 ± 4665	2315 ± 5660	.001	1143 ± 565	1655 ± 4556	<0.001
Troponin-I 3	560 ± 2268	1600 ± 4165	.006	496 ± 2515	1217 ± 3317	<0.001
Troponin-I 4	181 ± 935	666 ± 1760	<.001	180 ± 1046	481 ± 1419	<0.001
Troponin-I 5	194 ± 745	484 ± 1017	<.001	99 ± 559	499 ± 1053	<0.001
Troponin-I 6	37 ± 103	420 ± 888	<.001	21 ± 58	288 ± 719	<0.001
Troponin-I 7	31 ± 103	458 ± 1146	<.001	10 ± 14	369 ± 1004	<0.001
Troponin-I 8	43 ± 124	649 ± 1823	<.001	40 ± 134	455 ± 1517	<0.001
Troponin-I 9	20 ± 51	227 ± 322	<.001	14 ± 24	165 ± 283	<0.001
Troponin-I 10	13 ± 16	162 ± 226	<.001	16 ± 18	135 ± 213	0.006

Table 3. Relationship between daily D-dimer values with mortality and intensive care needs

Daily D-dimer (ng/mL)	Mortality (-)	Mortality (+)	p	Intensive care (-)	Intensive care (+)	p
D-dimer 1	1178 ± 1834	3082 ± 3470	0.008	897 ± 1394	2941 ± 3186	<0.001
D-dimer 2	1265 ± 1415	4401 ± 3512	<0.001	835 ± 832	3456 ± 3145	<0.001
D-dimer 3	1272 ± 1626	2761 ± 2552	0.002	1034 ± 1588	2448 ± 2191	<0.001
D-dimer 4	1050 ± 1140	2749 ± 2363	<0.001	964 ± 1044	2100 ± 2092	0.002
D-dimer 5	940 ± 1023	2261 ± 1822	0.001	861 ± 1009	1982 ± 1668	<0.001
D-dimer 6	1119 ± 1048	2114 ± 1556	0.004	898 ± 849	2005 ± 1475	<0.001
D-dimer 7	999 ± 875	2118 ± 1712	0.008	873 ± 788	1910 ± 1575	0.001
D-dimer 8	1033 ± 918	2311 ± 2465	0.01	904 ± 932	2031 ± 2168	0.02
D-dimer 9	1106 ± 1048	2084 ± 2030	0.005	1014 ± 1083	1825 ± 1759	0.02
D-dimer 10	1038 ± 8220	2599 ± 2082	<0.001	938 ± 848	2411 ± 1938	<0.001

Table 4. Sensitivity, specificity, AUC, cut-off of daily Troponin-I and D-dimer values with intensive care needs

Intensive care	Sensitivity (%)	Specificity (%)	AUC	Cut-off	p
Troponin-I 1 (pg/mL)	75.9	73.9	0.843	9.1	<0.001
Troponin-I 2 (pg/mL)	78.9	78.6	0.832	20	<0.001
Troponin-I 3 (pg/mL)	77.1	76.50	0.819	14	<0.001
Troponin-I 4 (pg/mL)	73.5	73.2	0.827	18.8	<0.001
Troponin-I 5 (pg/mL)	78.8	76.9	0.863	19.5	<0.001
Troponin-I 6 (pg/mL)	70.6	70	0.798	17.30	<0.001
Troponin-I 7 (pg/mL)	78.6	78.3	0.896	16.05	<0.001
Troponin-I 8 (pg/mL)	80.6	77.8	0.846	13.45	<0.001
Troponin-I 9 (pg/mL)	78.6	78.9	0.837	13.15	<0.001
Troponin-I 10 (pg/mL)	73.9	70	0.804	23.55	0.006
D-dimer 1 (ng/mL)	76.3	63.1	0.773	717	<0.001
D-dimer 2 (ng/mL)	76.9	74.2	0.858	878	<0.001
D-dimer 3 (ng/mL)	72.7	72.7	0.780	933	<0.001
D-dimer 4 (ng/mL)	65	63	0.685	854	0.002
D-dimer 5 (ng/mL)	69.7	68.6	0.747	897	<0.001
D-dimer 6 (ng/mL)	67.6	66.7	0.758	907	<0.001
D-dimer 7 (ng/mL)	68.8	66.7	0.746	818	0.001
D-dimer 8 (ng/mL)	76.7	75	0.742	880	0.002
D-dimer 9 (ng/mL)	71.9	70.4	0.741	832	0.002
D-dimer 10 (ng/mL)	78.3	73.7	0.844	1028	<0.001

Table 5. Sensitivity, specificity, AUC, cut-off of daily Troponin-I, D-dimer and other laboratory values with mortality

Mortality	Sensitivity (%)	Specificity (%)	AUC	Cut-off	p
Troponin-I 1 (pg/mL)	76.7	76.1	0.804	16.95	<0.001
Troponin-I 2 (pg/mL)	77.3	65.9	0.757	27.15	0.001
Troponin-I 3 (pg/mL)	75	71.4	0.710	21.85	0.006
Troponin-I 4 (pg/mL)	71.4	70.4	0.761	22.4	<0.001
Troponin-I 5 (pg/mL)	77.3	76	0.787	29.75	<0.001
Troponin-I 6 (pg/mL)	81	79.1	0.810	28.2	<0.001
Troponin-I 7 (pg/mL)	76.2	73.3	0.857	22.9	<0.001
Troponin-I 8 (pg/mL)	90.5	85.7	0.878	21.75	<0.001
Troponin-I 9 (pg/mL)	89.5	89.3	0.920	30.25	<0.001
Troponin-I 10 (pg/mL)	84.2	78.6	0.883	26.35	<0.001
D-dimer 1 (ng/mL)	63.2	61.2	0.688	753	0.008
D-dimer 2 (ng/mL)	73.9	72.3	0.807	1172	<0.001
D-dimer 3 (ng/mL)	68.4	67.2	0.742	1125	0.002
D-dimer 4 (ng/mL)	68.2	68.1	0.770	1012	<0.001
D-dimer 5 (ng/mL)	69.6	68.9	0.740	949	0.001
D-dimer 6 (ng/mL)	69.6	65.9	0.718	997	0.004
D-dimer 7 (ng/mL)	65.2	63.9	0.707	938	0.008
D-dimer 8 (ng/mL)	71.4	69.7	0.710	1053	0.01
D-dimer 9 (ng/mL)	66.7	65.8	0.721	968	0.005
D-dimer 10 (ng/mL)	68.4	65.2	0.817	1106	<0.001
CK* (U/L)	61.8	59.7	0.668	79.5	0.003
CRP# (mg/L)	62.9	62.3	0.676	51.55	0.01
Ferritin (ng/mL)	66.7	65.6	0.758	333	<0.001
Urea (ng/mL)	67.6	66.3	0.693	39.5	0.001
Creatinine (mg/dL)	65.7	64.5	0.640	0.9	0.01
LDH† (U/L)	76.3	75.3	0.823	317	<0.001

Epidemiology

PB-016

Fibrinogen-to-albumin ratio may be a predictor for ascending aortic aneurysm

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Background and Aim: The predictive value of the fibrinogen to albümin ratio has been evidenced in coronary artery disease. Available data demonstrated that inflammation and oxidative stress, are relevant mechanisms of ascending aortic aneurysm (AAA) formation and dilatation. The fibrinogen-to-albumin ratio (FAR), reflecting oxidative stressand inflammation. This study investigated the correlation between FAR and AAA.

Methods: 250 consecutive patients with AAA and 250 consecutive patients with normal ascending aort diameter were included into the study by comprehensive transthoracic echocardiography. All data and FAR was compered between two groups.

Results: FAR levels were significantly higher in AAA group compared to normal ascending aortic diamater group (p<0.001). Also there was significantly positive correlation beetween the diamater of the ascending aorta and the FAR (p<0.001).

Conclusions: FAR is associated with AAA and may serve as blood marker for identifying high-risk patients.

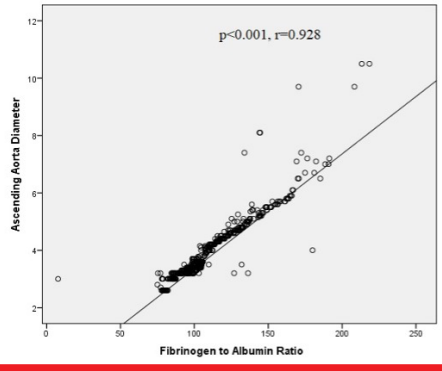

Figure 1. Correlation between fibrinogen to Albumin ratio andascending aortic diameter.

Table 1. Clinical and demographic characteristics of the study population

Variables	Control group (n = 250)	Case group (n = 250)	p
Age, years	59.10 ± 14.15	57.50 ± 15.21	0.222
Female, n (%)	80 (32.0%)	63 (25.2%)	0.092
Body mass index, kg/m^2	29.12 ± 2.39	28.29 ± 2.89	0.541
Hyperlipidemia, n (%)	53 (21.2%)	73 (29.2%)	0.097
Hypertension, n (%)	95 (38.0%)	122 (48.8%)	0.015
Diabetes Mellitus, n (%)	42 (16.8%)	52 (20.8%)	0.252
Smoking, n (%)	94 (37.6%)	97 (38.8%)	0.782
Coronary artery disease, n (%)	32 (12.8%)	31 (12.4%)	0.893

Data are given as mean ± SD, n or median (interquartile range).

Table 2. Echocardiographic characteristics of the study population

Variables	Control group (n = 250)	Case group (n = 250)	p
LVEF (%)	63.1 ± 2.2	61.0 ± 2.2	0.523
ARVC (mm)	0.8 ± 1.0	2.4 ± 1.0	<0.001
Aortic annulus diameter (mm)	2.19 ± 0.21	2.30 ± 0.37	<0.001
Sinus valsalva diameter (mm)	3.47 ± 0.71	4.07 ± 0.78	<0.001
Ascending aorta diameter (mm)	3.27 ± 0.24	4.60 ± 1.47	<0.001
Bicuspid aortic valve, n (%)	45 (18.0%)	8 (3.2%)	<0.001

Data are given as mean ± SD, n or median (interquartile range). ARVC, vena contracta width of aortic regurgitation; LVEF, left ventricular ejection fraction.

Table 3. Blood parameters of the study population

Variables	Control group (n = 250)	Case group (n = 250)	p
Glucose, mg/dL	113.9 ± 42.3	109.3 ± 37.1	0.481
Creatinine, mg/dL	1.07 ± 0.25	1.07 ± 0.32	0.749
Uric Acid, mg/dl	5.51 ± 2.37	6.71 ± 2.67	0.027
Hemoglobin, g/dL	13.8 ± 1.4	14.0 ± 1.7	0.410
WBC, 10^3/mm^3	7.8 ± 2.4	8.1 ± 2.4	0.343
Hs-CRP, mg/L	4.5 ± 2.5	8.4 ± 3.7	<0.001
Total cholesterol, mg/dL	188.9 ± 42.8	181.2 ± 45.3	0.056
LDL-C, mg/dL	118.8 ± 33.8	112.7 ± 45.3	0.291
HDL-C, mg/dL	47.3 ± 10.7	47.7 ± 12.7	0.847
Albumin (g/dL)	3.83 ± 0.09	3.68 ± 0.14	0.041
Fibrinogen (µg/ml)	364 ± 36	467 ± 73	0.004
Fibrinogen to Albumin ratio	95 ± 10	127 ± 24	<0.001

Data are given as mean ± SD, n or median (interquartile range). WBC, white blood cell; Hs-CRP, high-sensitivity C-reactive protein; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MHR, Monocyte-high-density lipoprotein ratio.

Table 4. Multivariate linear regression analysis showing the predictors for the Ascending aortic dilatation

Variables	Univariable Beta (95% CI)	P value	Multivariable Beta (95% CI)	p
Hypertension	1.518 (1.351-1.706)	0.014	1.477 (0.998-2.187)	0.057
Uric Acid	1.045 (0.996-1.099)	0.086		
Hs-CRP	1.041 (1.022-1.061)	<0.001	1.032 (1.013-1.052)	0.002
Albumin	1.051 (1.008-1.087)	0.015	1.066 (1.020-1.123)	0.076
Fibrinogen	1.048 (1.030-1.073)	0.010	1.051 (1.033-1.074)	0.055
Fibrinogen to Albumin ratio	1.201 (1.158-1.246)	<0.001	1.224 (1.165-1.281)	0.001

Hs-CRP, high-sensitivity C-reactive protein.

Epidemiology

PB-017

Impact of the COVID-19 pandemic on acute coronary syndrome hospitalizations

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Background and Aim: A noticeable decrease in the rate of hospitalizations for acute coronary syndrome (ACS) during the coronavirus disease 2019 (COVID-19) pandemic has been reported. We aim to compare data on hospitalizations for ACS for an 11-week date range during the COVID-19 pandemic and compare these data to data for the same date range in 2019.

Methods: In this retrospective analysis, we examine the weekly incidence rates of hospitalization for ACS between March 15, 2019 and May 31, 2019 (2019 period) and between March 15, 2020 and May 31, 2020 (pandemic period). A total of 636 ACS patients were screened for this study. The length of hospital stay was recorded for all of the included patients. The incidence rate ratio (IRR) was calculated for overall ACS and for the subtypes of ACS.

Results: A total of 511 patients were included in the final analysis. There was a decrease in the mean number of weekly hospitalizations for overall ACS by 23.7% in pandemic period compared to the 2019 period (IRR: 0.76 [95% confidence interval (CI): 0.64-0.90], p=0.002). This decrease in the mean number of weekly hospitalizations for ACS from the 2019 period to the pandemic period is shown in Fig. 1. There was a statistically nonsignificant decrease in total ST-elevation myocardial infarction (STEMI) cases of 8.6% from the 2019 period to the pandemic period (IRR: 1.02 (95% CI: 0.76-1.36), p=0.883). There was a statistically significant decrease in total non-STEMI (NSTEMI) cases of 28.2% from the 2019 period to the pandemic period [IRR: 0.71 (95% CI: 0.56-0.90), p=0.005]. In-hospital mortality and percutaneous coronary intervention (PCI) rates were similar between the two periods (p=0.300 and p=0.398, respectively).The median length of hospital stay was found to be significantly lower during the pandemic period than during the 2019 period [85 (95% CI: 63-112) h vs 117 (95% CI: 93-181) h, p<0.00001]. The duration of transfer between the emergency department and cardiac care unit for NSTEMI patients decreased from the 2019 period to the pandemic period by 50%.

Conclusions: The COVID-19 pandemic has affected ACS hospitalizations across the world, and this is most likely explained by several factors. At our center, we observed adecrease in the mean number of weekly hospitalizations from ACS by 23.7% from the 2019 period to the pandemic period, which is similar to the decrease reported in other recently performed studies. We discharged stable patients early during the pandemic period. There was no increase in in-hospital mortality in our patient population from the 2019 period to the pandemic period. In cases where the need for intensive care beds increases, such as during a pandemic, early discharge can be considered a precautionary measure.

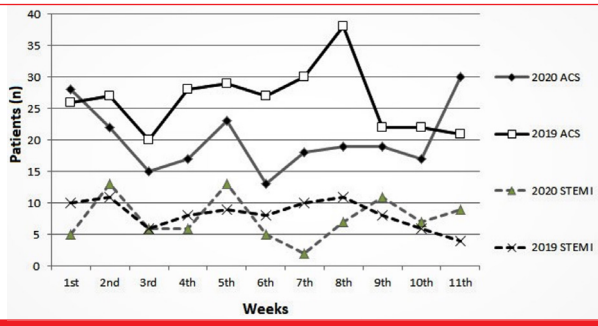

Figure 1. Weekly acute coronary syndrome (ACS) and ST elevation myocardial infarction (STEMI) hospitalizations in the study population from March 15 to May 31, 2019 and from March 15 to May 31, 2020. Black line: ACS hospitalizations during the time of from March 15 to May 31, 2019 (2019 period). Grey line: ACShospitalizations during the time of from March 15 to May 31, 2020 (pandemic period). Black dashed line: STEMI hospitalizations during the 2019 period. Grey dashed line: STEMI hospitalizations during the pandemic period.

Table 1. Incidence-rate ratios of hospitalizations for acute coronary syndrome ACS during pandemic period as compared with the 2019 period

ACS subtype	Pandemic period	2019 period	IRR (95% CI)	p
Overall (n)	221	290		
Number of weekly admission	20.09	26.36	0.76 (0.64-0.90)	0.002
STEMI (n, %)	84 (38)	92 (32)		
Number of weekly admission	7.63	8.36	1.02 (0.76-1.36)	0.883
NSTEMI(n, %)	122 (55)	170 (59)		
Number of weekly admission	11.09	15.45	0.71 (0.56-0.90)	0.005
UAP (n, %)	15 (7)	28 (9)		
Number of weekly admission	1.36	2.54	0.53 (0.28-1.00)	0.05

95% CI: 95% confidence intervals.
IRR, incidence-rate ratios; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction; UAP, unstable angina pectoris.

Epidemiology

PB-018

Clinical features and outcomes of COVID-19 patients with cardiac disease: Single center experience

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Background and Aim: Comorbidities accompany the majority of COVID-19 patients. Studies have shown that between 15% and 40% of them have a history of heart disease, and it has been reported that the course of the disease is more severe in those with heart disease.The aim of our study is to compare the clinical presentations and outcomes of patients

with and without cardiac disease hospitalized for COVID-19 infection and to share our single center experience.

Methods: 184 COVID-19 patients who were admitted to Antalya Kepez State Hospital between 15 March and 01 June and whose diagnosis was confirmed by a positive PCR test were included in the study. The data of the patients were obtained retrospectively from patient files and hospital information management system. Patients were divided into two groups as those with and without cardiac disease. Demographic characteristics, clinical presentations, laboratory tests, radiological imaging results and in-hospital outcomes of the patients were recorded. The data of 30 patients with cardiac disease and 154 patients without cardiac disease were compared.

Results: 176 of 184 patients were hospitalized. Coronary artery disease was present in 66.7%, atrial fibrillation in 46%, and heart failure in 40% of COVID-19 patients with accompanying cardiac disease. The mean age of patients with cardiac disease was higher than those without cardiac disease (72.5 ± 15.8 vs 45.4 ± 15.4, p<0.001). There was no significant difference in presentation symptoms between the two groups. Oxygen saturation at admission was lower and respiratory rate was higher in patients with cardiac disease. Serum creatinine, Hs troponin, D-Dimer, C-reactive protein levels were significantly higher in patients with cardiac disease. When the findings of 174 patients evaluated with thorax computed tomography at the time of admission were compared, no significant difference was found between the groups. The rate of death (20% vs 0% p<0.004), thromboembolic events (13.3% vs 0% p=0.025), acute respiratory distress syndrome (26.7% vs 1.3% p=0.002) and septic shock (33.3% vs 1.3% p<0.001) during hospitalization was higher in patients with cardiac disease.

Conclusions: Patients with cardiac disease with COVID-19 have higher rates of mortality, thromboembolic events, ARDS and septic shock than those without a history of cardiac disease, and the prognosis of these patients is quite poor.

Interventional cardiology / Valve and structural heart diseases

PB-019

Relationship between prealbumin levels and TAVR outcomes

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Background and Aim: Transcatheter aortic valve replacement (TAVR) is a standard of care treatment option in patients with severe symptomatic aortic valvular disease who have intermediate-high surgical risk. Prealbumine is an indicator of malnutrition in geriatric population. In this study; we aimed to evalaute the association between prealbumin levels and TAVR outcomes.

Methods: All of the patients who underwent TAVR procedure between January 2010 and June 2020 in our center were screened and patients in whom prealbumin level was checked were enrolled.

Results: A total of 55 patients were enrolled in the study. Mean age of the study population was 75.7 ± 9.3 years and 30.9% of the patients (n = 17) were male. Median follow up was 21.8 (5.7-46.1) months. Peralbumin level was low in 24 (43.6%) patients. Baseline characteristics were similar between patients with low prealbumin and counterparts except LV end-diastolic diameter. Rate of TAVR related complications including insertion site complications, stroke, contrast nephropathy and permanent pacemaker implantation were similar between the two groups. In hospital and all cause mortality rates were significantly higher in patients with low prealbumin than patients with normal prealbumin levels.

Conclusions: Prealbumin is an indicator of malnutrition and associated with poor prognosis in geriatric patients. This study shows that; both all cause and in hospital mortality rates after TAVR are increased in patients with low prealbumin levels.

	Normal Prealbumin (n=31)	Low Prealbumin (n=24)	p value
Age, years, mean ± sd	75,0 ± 9,8	76,5 ± 8,8	0,567
Gender, male, n (%)	11 (35,5 %)	6 (25 %)	0,589
Comorbidities, n (%):			
*Hypertension	25 (80,6 %)	20 (83,3 %)	1,000
*Diabetes	10 (32,3 %)	5 (20,8 %)	0,380
*COPD	6 (19,4 %)	4 (16,7 %)	1,000
*Coronary artery disease	12 (38,7 %)	8 (33,3 %)	0,898
*Atrial fibrillation	6 (19,3 %)	7 (29,2 %)	0,694
*Heart failure	8 (25,8 %)	7 (29,1 %)	0,710
Drugs, n (%):			
*Beta blockers	18 (58,1 %)	16 (66,7 %)	0,710
*RAAS inhibitor	15 (48,4 %)	12 (50 %)	1,000
*Statin	12 (38,7 %)	8 (33,3 %)	0,898
Echocardiographic parameters:			
*LV EDD, mm	50,9 ± 7,1	46,8 ± 5,3	0,025*
*LV EF, %	56,3 ± 8,2	54,2 ± 12,1	0,433
*LA, mm	42,4 ± 6,4	44,3 ± 7,5	0,315
*Interventricular septum, mm	12,3 ± 1,7	12,7 ± 2,4	0,573
*AVA, cm^2	0,89 ± 0,31	0,82 ± 0,26	0,444
*Mean aortic gradient, mmHg	40,5 ± 11,9	42,2 ± 17,5	0,674
*Moderate- severe MR, n (%)	16 (51,6 %)	14 (58,4 %)	0,807
*sPAP, mmHg	47,2 ± 13,5	51,0 ± 16,5	0,354
Laboratory parameters:			
*Hemoglobin, g/dL	12,0 ± 1,7	12,0 ± 1,3	0,971
*Creatinine, mg/dL	0,95 (0,67-1,23)	1,01 (0,77-1,16)	0,696
* Prealbumin, mg/dL	24,8 ± 4,7	8,7 ± 3,3	<0,001*
* Albumin, mg/dL	3,8 ± 0,5	3,4 ± 0,5	0,004*
Follow-up, months	31,0 (15,8-62,4)	11,6 (0,4-28,7)	0,215

Table-2. TAVR Outcomes

	Normal Prealbumin (n=31)	Low Prealbumin (n=24)	p value
Insertion site complications, n (%)	8 (25,8 %)	6 (25 %)	0,181
Stroke, n (%)	2 (6,5 %)	5 (20,8 %)	0,220
Permanent pacemaker implantation, n (%)	7 (22,6 %)	8 (33,3 %)	0,560
Contrast nephropathy, n (%)	4 (12,9 %)	6 (25 %)	0,304
In hospital mortality, n (%)	2 (6,5 %)	9 (37,5 %)	0,006*
All cause mortality, n (%)	6 (19,4 %)	18 (75 %)	<0,001*

Table 1 and 2. Baseline characteristics

Interventional cardiology / Valve and structural heart diseases

PB-020

Association of the novel inflammatory marker systemic immune inflammation index and contrast-induced nephropathy in patients undergoing TAVR for severe aortic stenosis

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Background and Aim: This study aims to investigate whether the systemic immune inflammation index (SII) is an independent risk factor to predict the development of contrast-induced nephropathy (CIN) in patients undergoing TAVR for severe aortic stenosis.

Methods: One hundred forty-four patients were included in the study. Fourteen patients were excluded from the study due to various reasons. The remaining 130 patients were included in the analysis. Patients who had a 25% or greater increase in creatinine level from baseline serum creatinine; or an increase in creatinine of 0.5 mg/dL or more at 48 hours after contrast exposure were considered CIN. The patients were divided into 2 groups as those who developed CIN (+) and those who did not develop CIN (-). The SII index was calculated as the ratio of the product of the total neutrophil count and the total platelet count to the lymphocyte count.

Results: Demographic characteristics, clinical characteristics, laboratory parameters, echocardiographic findings, and SII score of the patients were compared between the 2 groups. Contrast-induced nephropathy developed in 20 (15.3%) patients after the TAVR procedure. WBC (7.66 ± 1.75 vs 6.78 ± 1.71, p=0.038), neutrophil (5.1 (3.9-6.7) vs 4.2 (3.5-5.1), p=0.024), neutrophil-lymphocyte ratio (4.20 (2.39-7.00) 2.75 (2.06-3.88), p=0.010) and SII index (1069 (616-1514) vs 598 (426-955), p=0.003) were detected at higher levels in patient with CIN than the group without CIN. The receiver operating characteristic curve (ROC) analysis showed that the SII index had the best cut-off value of 743 to predict the development of CIN, with 65% sensitivity and 60% specificity (area under ROC curve = 0.709 [95% CI: 0.594-0.823]). In addition, the SII index was found to be an independent predictor for the development of CIN.

Conclusions: The SII index is an independent predictor for the development of contrast nephropathy in patients undergoing TAVR for severe aortic stenosis, which can be easily calculated from a complete blood test.

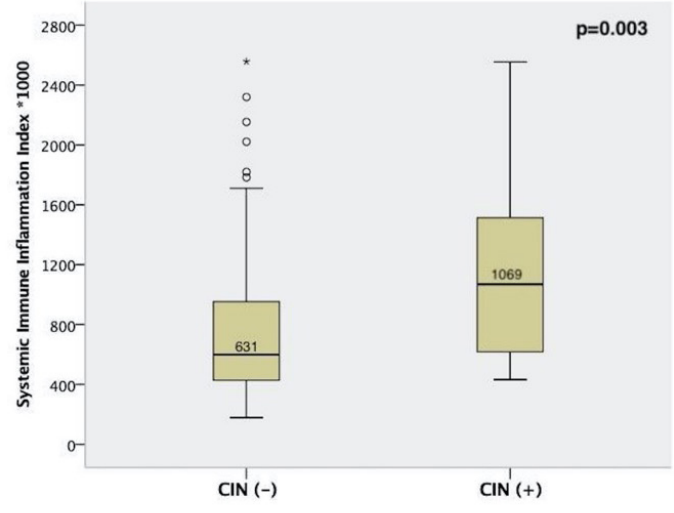

Figure 1.

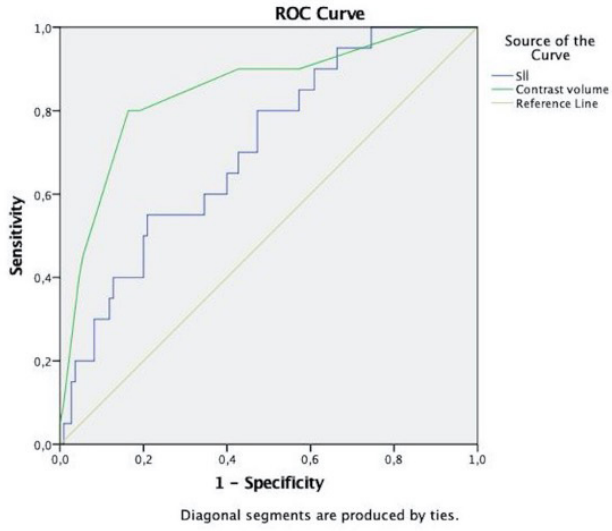

Figure 2.

Demographic and clinical features	CIN (+) (n = 20)	CIN (–) (n = 110)	P
Age, years	79 ± 8	76 ± 8	.315
Gender			.030
Male	14 (70%)	48 (44%)	
Female	6 (30%)	62 (56%)	
Diabetes mellitus	12 (60%)	55 (50%)	.410
Hypertension	13 (65%)	76 (69%)	.717
Coronary artery disease	4 (20%)	29 (26%)	.781
Heart failure	6 (30%)	27 (25%)	.587
Cerebrovascular disease	2 (10%)	4 (4%)	.230
CHADSVASC score	3.7 ± 1.1	3.8 ± 1.3	.499
Treatments			
MRA	3 (15%)	15 (14%)	.871
ACEI/ARBs	11 (55%)	76 (69%)	.218
Thiazide diuretics	6 (30%)	21 (19%)	.367
Laboratory parameters			
Basal eGFR, mL/min/1.73 m^2	64 ± 19	64 ± 18	.874
48th hour eGFR, mL/min/1.73 m^2	41 ± 16	69 ± 18	**<.001**
Glucose, mg/DL	125 (100–157)	107 (89–136)	.084
HbA1c, %	6.7 ± 1.1	6.5 ± 1.1	.431
Baseline creatinine, mg/dL	1.08 (.80–1.27)	.97 (.80–1.20)	.494
48th hour creatinine, mg/dL	1.49 (1.22–1.87)	.91 (.74–1.07)	**<.001**
Potassium, mEq/L	4.5 ± 0.4	4.4 ± 0.5	.179
Albumin, g/dL	38 ± 5	39 ± 4	.373
HDL-cholesterol(mg/dL)	37 (32–50)	40 (33–51)	.572
LDL-cholesterol (mg/dL)	98 ± 29	96 ± 38	.778
NT-proBNP (pg/mL)	912 (302–3471)	876 (302–3721)	.826
CRP (mg/L)	3 (3–10)	6 (3–15)	.581
CRP/albumin ratio	.09 (.07–.27)	.13 (.07–.41)	.730
Triglycerides (mg/dL)	111 (87–172)	96 (81–142)	.257
WBC (10^3/mm^3)	7.66 ± 1.75	6.78 ± 1.71	**.038**
Neutrophil (10^3/mm^3)	5.1 (3.9–6.7)	4.2 (3.5–5.1)	.024
Lymphocyte (10^3/mm^3)	1.4±0.7	1.6±0.6	.374
Platelets (10^3/mm^3)	232 ± 78	252 ± 47	.123
Hemoglobin (g/dL)	11.7 ± 1.2	11.9 ± 1.8	.469
Hematocrit, %	36 ± 3	37 ± 5	.431
NLR	4.20 (2.39–7.00)	2.75 (2.06–3.88)	**.010**
SII×10^3	1069 (616–1514)	598 (426–955)	**.003**
Echocardiographic parameters			
AVA, cm^2	.74 ± .17	.70 ± .17	.495
Maximum gradient, mmHg	78 ± 19	76±20	.677
Mean gradient, mmHg	50 ± 13	48±13	.592
LVEF, %	50 ± 14	48 ± 14	.740
Contrast volume, mL	153 ± 12	137 ± 10	**<.001**
Mehran risk score	10.6± 3.5	8.8±4.0	**.042**
	11 (8.2–12)	9.0 (5.7–11.2)	**.042**

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blockers; AVA, aortic valve area; CIN, contrast-induced nephropathy; CRP, C-reactive protein; eGFR, glomerular filtration rate; HDL, high density lipoprotein; LDL, low density lipoprotein; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonists; NLR, neutrophil to lymphocyte ratio; NT-ProBNP, N-terminal pro brain natriuretic peptide; SII, systemic immune-inflammation index; WBC, white blood cell.

Table 1. Demographic and clinical features

Variables	Univariate Analysis OR (95% CI)	P	Multivariate Analysis[a] OR (95% CI)	p
Age	1.03 (.97–1.10)	.313		
Gender	.33 (.12–.93)	.035	.22 (.06–.84)	.027
Diabetes mellitus	1.50 (.57–3.95)	.412		
CHADSVASC score	.89 (.60–1.30)	.537		
Contrast volume	1.14 (1.08–1.20)	<.001	1.15 (1.08–1.22)	<.001
Mehran risk score	1.13 (.99–1.28)	.059		
Basal eGFR	.99 (.97–1.02)	.867		
ACEI/ARBs treatment	.55 (.21–1.44)	.222		
LVEF, %	1.01 (.97–1.04)	.742		
SII	1.001 (1.001–1.002)	.004	1.002 (1.001–1.003)	.005

[a]Multivariate Model's Nagelkerke R^2 = .516, −2 Log Likelihood = 66, P < .001.
Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blockers; CI, confidence interval; eGFR, glomerular filtration rate; LVEF, left ventricular ejection fraction; OR, odds ratio; SII, systemic immune-inflammation index. A P < .05 was considered significant for statistical analyses.

Table 2. Independent predictors of development of contrast-induced nephropathy by logistic regression analyses

	Cut-Off Value	AUC	Sensitivity, %	Specificity, %
SIIx10^3	743	.709 (.594–.823)	65	60
Contrast volume, mL	147	.848 (.748–.947)	80	83

Abbreviations: AUC, area under the curve; SII, systemic immune-inflammation index.

Table 3. ROC curve analysis for the prediction of contrast-induced nephropathy by risk factors that were significant in multivariate analysis

Interventional cardiology / Cover and structural heart diseases

PB-021

First experiences with the transfemoral ACURATE-neo TM self-expanding transcatheter aortic bioprosthesis in our center

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Background and Aim: In present article, we aimed to present our short-term follow-up results of ACURATE-neoTM Aortic Valve System and its ACURATE-neo delivery system (Boston scientific).

Methods: Twenty-four consecutive patients who underwent transfemoral TAVI from June 2020 to August 2021 were included in the study. Clinical and echocardiographic assessment was performed at baseline, postprocedure and at least 30 days. Outcomes were assessed according to valvular academic research consortium (VARC-3) criteria.

Results: Laboratory and clinical characteristics of the patients are shown in Table 1. The mean age of the whole population was 80.54 ± 6.11 years, 19 (79.2%) were female, and 24 (100%) were hypertensive. STS score of the group was 3.35 ± 1.26. Procedural details are described in Table 2. TAVI procedure was performed by transfemoral route to all patients. Device success was achieved in 22 (91.7%) patients. We used cut-down 1 (4.2%) patients and ProGlide in 23 (95.8%) patients for vascular closure. One (4.2%) periprocedural deaths occurred in our study due to stroke. Vascular complications were observed in 2 (8.3%) patient. None of the patients in our study had severe PVL, and one (4.2%) patient had moderate PVL. PPM was required in three (12.5%) patients, and indications were complete AV block. The mean hospital stay in the whole group was 3.83 ± 1.89 days.

Conclusions: Based on our experiences, the ACURATE-neoTM aortic bioprosthesis was safely and successfully implanted by transfemoral approach. The special design of this new generation device ensures a stable and predictable implantation while providing optimum hemodynamic performance.

Table 1.

Parameters	All patients (n = 24)
Age (years)	80.54 ± 6.11
Female gender (%)	19 (79.2)
Diabetes, n (%)	4 (16.7)
Hypertension, n (%)	100
Dyslipidemia, n (%)	18 (75)
Smoking, n (%)	5 (20.8)
Coronary Artery Disease, n (%)	21 (87.5)
Previous MI, n (%)	7 (29.2)
Previous PCI, n (%)	7 (29.2)
Previous CABG, n (%)	1 (4.2)
Previous Stroke, n (%)	3 (12.5)
3 (12.5)	7 (29.2)
Moderate-to-severe COPD, n (%)	1 (4.2)
NYHA, n (%)	
- 3	15 (62.5)
- 4	9 (37.5)
AF, n (%)	6 (25)
Preprocedural LBBB, n (%)	7 (29.2)
STS score (%)	3.35 ± 1.26
Logistic EuroSCORE (%)	17.80 ± 7.99
BMI	27.55 ± 5.05
Chronic kidney disease, n (%)	4 (16.7)
Aortic stenosis classification, n (%)	
- HG-AS	19 (79.17)
- Paradoxical LFLG-AS	5 (20.83)
Aortic max gradient (mmHg)	74.67 ± 23.59
Aortic mean gradient (mmHg)	48.17 ± 14.88
AVA (cm^2)	0.63 ± 0.16
Aortic valve calcium score	2449.83 ± 1573.33

Clinical and demographic characteristics of study subjects.
AF, atrial fibrillation; AS, aortic stenosis; AVA, aortic valve area; BMI, body-mass index; BSA,body surface area; CABG, coronary artery bypass grafting; COPD, chronic obstructive pulmonary disease; HG-AS, high gradient aortic stenosis.

Table 2.

Access site, n (%)	
- Transaxillary	-
- Transfemoral	24 (100)
Cut-down	1 (4.2)
ProGlide	23 (95.8)
Valve size, mm, n (%)	
- 23	4 (16.7)
- 25	7 (29.2)
- 27	13 (54.2)
Pre-dilatation, n (%)	24(100)
Post-dilatation, n (%)	4 (16.7)
Device success, n (%)	22 (91.7)

Procedural details of subjects.
BAV,balloon aortic valvuloplasty

Interventional cardiology / Valve and structural heart diseases

PB-022

The relation between HAS-BLED score and procedural outcomes in patients undergo TAVI

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Background and Aim: Patients that candidate for transcatheter aortic valve implantation (TAVI) are frail and prone to complications during the procedure. The effect of HAS-BLED score to procedure related complications has not been widely investigated before. In this study, we sought to detect the relation between HAS-BLED score and clinical outcomes following the TAVI procedure.

Methods: Study population consisted of 153 consecutive patients that had undergone TAVI between 2013-2020 in a single center. Access site complication was defined as the occurrence of inguinal hematoma and/or iliac artery injury requiring surgical intervention. Post-procedural data, including mortality, access site complication, need for blood transfusion, occurrence of acute kidney injury were noted. The relation of the HAS-BLED score and procedural outcome was assessed by univariate analysis.

Results: A total of 153 patients were included in the study, with an average age of 78, 14 ± 7, 17 and 81 (52.9%) of them being female. Mean HAS-BLED scores was 2.67 ± 0.90. Patients with a high HAS-BLED score had a greater one-year mortality rate (3.04 ± 1.02 vs 2.60 ± 0.84 p=0.035). In addition, patients that received blood transfusion during index hospitalization exhibited higher HAS-BLAD score (2.89 ± 0.86 vs 2.55 ± 0.901 p=0.025). HAS-BLED scores were not different among to patients that acute kidney injury occurred and patients with access site complication.

Conclusions: TAVI patients with one-year mortality and those who underwent blood transfusion during the index hospitalization have a higher HAS-BLED score.

PB-023 [Interventional cardiology / Cover and structural heart diseases]

Vascular complications following transcatheter transfemoral aortic valve implantation: Modified sheath-to femoral artery ratio as a new predictor

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Background and Aim: Vascular complications (VCs) remain a significant issue in transcatheter aortic valve implantation (TAVI) patients increased morbidity and mortality; however, their incidence and predictors are conflicting between studies. This research sought to assess the incidence, impact, and predictors of VCs in transfemoral (TF) TAVI and also investigated the predictive role of manufacturer's size charts and a new predictor modified sheath-to-femoral artery diameter (md-SFAR).

Methods: A total of 223 patients undergoing TF-TAVI were categorized into two groups. Patients were divided as eligible and ineligible according to manufacturer's guidelines (MG) and same patient cohort was dichotomized into eligible and ineligible based on SFAR value of less than or greater than or equal to md-SFAR. Vascular complications (VC) (defined according to VARC-2 criteria) were retrospectively compared.

Results: According to manufacturer's size charts 65 patients were unsuitable however 35 patients based on md-SFAR criteria were ineligible for TF-TAVI. While VCs occurred in 42 cases (18.8%), of those 17 (27.7%) was classified as ineligible according to MG whereas 14 (41.2%) was classified as ineligible in md-SFAR group. In a multiple logistic regression analysis that included md-SFAR, MG, SFAR ≥ 1.05, peripheral artery disease, and minimum iliofemoral artery diameter, only md-SFAR was the sole independent predictor of VCs [OR = 3.71, 95% CI: 1.13-12.53, p=0.031].

Conclusions: According to our results, md-SFAR may provide better patient selection to prevent VCs and improve outcomes in TF-TAVI procedures.

Valve Name	Valve Size (mm)	Sheath Outer Diameter (mm)	Minimum vessel Diameter (mm)	Sheath Size (F)	Sheath Size (mm)	Sheath outer diameter Minimum vessel diameter (md-SFAR)	Sheath French Size Minimum vessel diameter
Sapien XT (Edwards Lifesciences)	23	6.7	6	16	5.33	1.12	2.67
	26	7.2	6.5	18	6	1.11	2.77
	29	8	7	20	6.67	1.14	2.86
Sapien 3 (Edwards Lifesciences)	20	6	5	14	4.67	1.2	2.8
	23	6	5.5	14	4.67	1.09	2.55
	26	6	5.5	14	4.67	1.09	2.55
	29	6.7	6	16	5.33	1.12	2.67
Evolute R (Medtronic)	23	6	5	14	4.67	1.2	2.8
	26	6	5	14	4.67	1.2	2.8
	29	6	5	14	4.67	1.2	2.8
	34	6.7	5.5	16	5.33	1.22	2.9
Portico (Abbot)	23	6	6	18	6	1	3
	25	6	6	18	6	1	3
	27	6.4	6.5	19	6.33	0.98	2.92
	29	6.4	6.5	19	6.33	0.98	2.92

Table 1. Outer diameter of sheaths and minimum vessel diameters required for the TAVI systems

	Manufacturer's Guidelines			md-SFAR value		
Variables	TF-TAVI eligible (n=158)	TF-TAVI ineligible (n=65)	P	TF-TAVI eligible (n=188)	TF-TAVI ineligible (n=35)	P
Age (years)	78.9±7.08	78.95±8.79	0.974	79.1±7.34	78.1±8.98	0.475
Men, n (%)	85(53.8)	31(47.7)	0.407	100(53.2)	16(45.7)	0.416
logEuroScore	26.02±4.07	25.68±3.57	0.556	25.79 ±3.85	26.62±4.23	0.227
NYHA III-IV, n (%)	85(53.8)	32(49.2)	0.534	100(52.9)	17(50)	0.756
LVEF %	49.63±13.8	53±10.03	0.076	50.5±13.2	51.2±11.01	0.737
BMI, kg/m²	26.47±3.63	25.68±3.49	0.134	26.4±3.66	25.35±3.13	0.147
Hypertension, n (%)	143(90.5)	57(87.7)	0.530	172(91)	28(80)	0.063
Diabetes, n (%)	38(24.1)	10(15.4)	0.152	45(23.8)	3(8.8)	0.045
CAD, n (%)	87(55.1)	44(67.7)	0.082	107(56.9)	24(68.6)	0.198
PAD, n (%)	26(16.5)	18(27.7)	0.055	33(17.5)	11(31.4)	0.056
GFR(ml/min/1.73 m2)	68.7±25.9	70.05±25.47	0.761	69.6±25.6	67.1±27.18	0.752
Previous MI, n (%)	41(25.9)	18(27.7)	0.789	48(25.5)	11(31.4)	0.468
Previous CVO, n (%)	8(5.1)	4(6.2)	0.743	9(4.8)	3(8.6)	0.408
Previous PCI, n (%)	47(29.7)	13(20)	0.136	53(28)	7(20)	0.316
CABG, n (%)	29(18.4)	13(20)	0.775	34(18)	8(22.8)	0.507
AF, n (%)	43(27.4)	13(20)	0.249	46(24.5)	10(28.6)	0.619
Anticoagulation, n (%)	53(33.8)	12(18.5)	0.024	56(29.8)	9(25.7)	0.626
Edwards Sapien XT	69(43.7)	44(67.7)	0.001	97(51.6)	16(45.7)	0.016
Edwards Sapien 3	22(13.9)	8(12.3)		24(12.8)	6(17.1)	
Evolute R	54(34.2)	6(9.2)		55(29.3)	5(14.3)	
Portico	13(8.2)	7(10.8)		12(6.4)	8(22.9)	
Iliofemoral calcium Score	1.24±0.74	1.69±1.03	0.002	1.29±0.78	1.82±1.1	0.011
Tortuosity Score	1.34±0.97	1.38±0.97	0.732	1.4±0.98	1.1±0.87	0.072
Minimal IFLD, mm	7.66±1.1	6.32±0.99	<0.001	7.56±1.05	5.66±0.8	<0.001
SFAR	0.89±0.11	1.13±0.15	<0.001	0.91±0.13	1.21±0.17	<0.001
Sheath ≥18F, n (%)	67(42.4)	41(63.1)	0.005	90(47.9)	18(51.4)	0.699
Sheath outer diameter (mm)	6.71±0.67	7.02±0.71	0.002	6.82±0.71	6.68±0.61	0.218

Table 2. Baseline clinical and procedural characteristics of the study population

Variables	Patients without VC (n=181)	Patients with VC (n=42)	P
Age (years)	79.2±7.69	77.98±7.2	0.353
Female, n (%)	83(45.9)	24(57.1)	0.187
logEuroScore	25.7±3.81	26.8±4.33	0.103
LVEF %	50.8±12.78	49.6±13.6	0.591
BMI, kg/m²	26.3±3.5	26.1±3.9	0.809
Hypertension, n (%)	164(90.6)	36(85.7)	0.397
Diabetes, n (%)	40(22.1)	8(19)	0.665
CAD, n (%)	107(59.1)	24(57.1)	0.815
PAD, n (%)	32(17.7)	13(28.6)	0.053
GFR(ml/min/1.73 m2)	69.43±25.3	68.3±27.9	0.803
Previous MI, n (%)	51(28.2)	8(19)	0.227
Previous CVO, n (%)	8(4.4)	4(9.5)	0.246
Previous PCI, n (%)	51(28.2)	9(21.4)	0.374
CABG, n (%)	33(18.2)	9(21.4)	0.633
AF, n (%)	46(25.6)	10(23.8)	0.815
Ineligibility (md-SFAR)	21(11.6)	14(33.3)	<0.001
Ineligibility (CG)	47(26)	18(42.9)	0.030
Calcification ≥moderate	18(9.9)	7(16.7)	0.274
Tortuosity ≥ moderate	73(81.2)	17(39.5)	0.586
Minimal IFLD, mm	7.31±1.16	6.87±1.41	0.055
Sheath ≥18F, n (%)	87(48)	21(50)	0.821
Sheath outer diameter (mm)	6.8±0.7	6.79±0.70	0.986
Number of Proglide per patient	1.86±0.49	1.98±0.71	0.199
Single Proglide,n (%)	37(20.4)	8(19)	0.839
Double Proglide, n (%)	133(73.5)	29(69)	0.561
Number of Proglides ≥3, n (%)	11(6.1)	5(11.9)	0.192
Valve Types			0.333
Sapien XT	88(48.6)	25(59.5)	
Sapien 3	27(14.9)	3 (7.1)	
Evolute R	51(28.2)	9(21.4)	
Portico	15(8.3)	5(11.9)	
SFAR	0.95±0.15	1.02±0.21	0.009
SFAR≥1.05	43(23.8)	16(38.1)	0.058
In-hospital death, n (%)	10(5.5)	5(11.9)	0.167
30-day mortality, n (%)	11(6.1)	8(19)	0.012

Table 3. Baseline characteristics between patients with and without vascular complications

		Manufacturer's Guidelines			md-SFAR Threshold		
	All Patients (n=223)	TF Eligible (n=158)	TF Ineligible (n=65)	P	TF Eligible (n=188)	TF Ineligible (n=35)	P
Overall VARC-II complication, n (%)	42(18.8)	24(15.2)	18(27.7)	0.030	28(14.8)	14(41.2)	<0.001
Major VARC-II complication, n (%)	17(7.6)	9(5.3)	8(12.3)	0.091	10(5.3)	7(20.6)	0.007
Minor VARC-II complication, n (%)	25(11.2)	15(9.5)	10(15.4)	0.205	18(9.5)	7(20.6)	0.083
Major Bleeding, n (%)	21(9.4)	12(7.6)	9(13.8)	0.146	14(7.4)	7(20.6)	0.029
Minor Bleeding, n (%)	16(7.2)	8(5.1)	8(12.3)	0.083	10(5.3)	6(17.6)	0.024
Haematoma, n (%)	12(5.4)	8(5.1)	4(6.2)	0.749	11(5.9)	1(2.9)	0.697
Aortic Dissection, n (%)	1	1	0	-	1	0	-
Rupture, n (%)	5(2.2)	1(0.6)	4(6.2)	0.026	1(0.5)	4(11.4)	0.002
Stenosis/occlusion	16(7.2)	10(6.3)	6(9.2)	0.568	11(5.9)	5(14.3)	0.143
Pseudoaneurysm, n (%)	2	1(0.6)	1(1.5)	0.499	1(0.5)	1(2.9)	0.290
Closure device failure, n (%)	4(1.8)	2(1.3)	2(3.1)	0.582	2(1.1)	2(5.7)	0.127
Sheath Fracture, n (%)	1	-	1	-	-	1	-
Annular rupture, n (%)	1	1	-		1	-	-
In-Hospital ex, n (%)	15(6.7)	11(7)	4(6.2)	1	12(6.4)	3(8.6)	0.711
30-day mortality	19(8.1)	14(8.9)	5(7.7)	0.776	16(8.5)	3(8.6)	0.991

Table 4. Complication types

	Univariate	Multiple	
	p value	HR (95%CI)	p value
Ineligibility (md-SFAR)	0.001	3.7(1.13-12.53)	0.031
PAD	0.114	1.6(0.72-3.6)	0.246
Ineligibility (MG)	0.032	1.1(0.38-3.28)	0.849
Minimum IFLD	0.036	1.03(0.71-1.52)	0.855
SFAR ≥1.05	0.061	0.92(0.29-2.96)	0.895
Calcification ≥ moderate	0.219		

PAD, peripheral artery disease; md-SFAR, modified sheath-to-femoral artery ratio; MG, manufacturer's guidelines; IFLD, Iliofemoral artery lumen diameter; SFAR, sheath-to-femoral artery ratio

Table 5. Predictors of overall vascular complications

Interventional cardiology / Valve and structural heart diseases

PB-024

Incidence and predictors of access-related vascular complications after transfemoral transcatheter aortic valve implantation with the Portico device

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Background and Aim: Access-related vascular complications (VCs) in transfemoral transcatheter aortic valve implantation (TAVI) are associated with increased morbidity and unfavorable outcomes. Studies addressing the incidence and predictors of VCs are limited owing to baseline differences in the study population, qualitative measurement of vessel characteristics, and the use of different valve characteristics and delivery systems. There is a need to adequately characterize VCs to reduce adverse outcomes for different valve types. This study sought to evaluate the incidence, predictors, and impact of access-related VCs after transfemoral TAVI with the self-expanding Portico valve.

Methods: From 2017 to 2021, 104 patients underwent transfemoral TAVI at a tertiary cardiac center. All patients received a Portico valve using an 18- or 19-F Ultimum introducer and percutaneous closure with a Perclose ProGlide system. Postoperative evaluation of the vascular access site was performed by ultrasound imaging. Technical, procedural, and clinical outcomes were compared between patients with and without access-related VCs. Access site-related VCs were defined according to the Valvular Academic Research Consortium-3 criteria. Multivariable logistic regressions were used to identify predictors of VCs.

Results: The average patient age was 80.5±6.0 years, and 36.5% of the patients were male. There was no significant difference in patient baseline characteristics (Table 1). Twenty-seven patients presented access site VCs (6.7% major, 19.3% minor), most of which were hematoma (11.5%), pseudoaneurysm (9.6%), bleeding (5.8%) or iliofemoral dissection (5.8%) (Figure 1). Patients with VCs had a higher rates of femoral artery calcification (55.6 vs. 15.6%, p<0.001), greater femoral artery depth (56.8±8.8 vs. 49.8±7.1 mm, *P* < 0.001), larger SFAR (1.30±0.22 vs. 1.15±0.17, p<0.001), and longer hospital stay (Table 2). The sheath to femoral artery ratio (SFAR) threshold with the highest sum of sensitivity (70.4%) and specificity (63.6%) for predicting access site VCs was 1.18 (area under the curve 0.707, 95% CI, 0.597 - 0.817, p=0.001) (Figure 2). On multivariable regression analysis, SFAR (odds ratio [OD]: 4.25 [95% CI: 1.38-13.10], p=0.012), calcification anywhere along common femoral artery (OD: 5.80 [95% CI: 1.86-18.10], p=0.002), and 90° depth to common femoral artery (OD: 1.10 [95% CI: 1.03- 1.17], p=0.005) were identified as independent predictors of VCs (Table 3).

Conclusion: The findings of this study suggest that access-related VCs remain an issue of transfemoral TAVI with the Portico, and access-related VCs are associated with a statistically significantly longer hospital stay and procedure time. The presence of femoral artery calcification, larger SFAR, and greater femoral artery depth were the independent predictors of VCs and therefore should be routinely evaluated during pre-procedural screening and imaging techniques.

Table 1. Baseline characteristics of the study population

Parameters	Total (n = 104)	Access-related vascular complications (n = 27)	No Access-related vascular complications (n = 77)	p
Demographic and clinical characteristics				
Age (years)	80.5±6.0	79.8±6.0	80.7±6.1	0.480
Sex (male)	38 (36.5)	12 (44.4)	26 (33.8)	0.321
Coronary artery disease	77 (74.0)	21 (77.8)	56 (72.7)	0.607
COPD	32 (30.8)	8 (29.6)	24 (31.2)	0.881
Diabetes	43 (41.3)	11 (40.7)	32 (41.6)	0.941
Chronic kidney disease	32 (30.8)	12 (44.4)	20 (26.0)	0.074
Hypertension	67 (64.4)	17 (63.0)	50 (64.9)	0.854
Previous CABG	24 (23.1)	5 (18.5)	19 (24.7)	0.514
Peripheral artery disease	25 (24.0)	9 (33.3)	16 (20.8)	0.189
Cerebrovascular disease	3 (2.9)	2 (7.4)	1 (1.3)	0.164
STS score	6.5 (4.1-9.0)	6.3 (4.5-8.0)	6.5 (4.0-9.0)	0.665
Hemoglobin (g/dL)	11.1±1.6	10.8±1.3	11.3±1.7	0.165
Creatinine (g/dL)	1.2±0.8	1.4±1.1	1.1±0.7	0.064
Echocardiographic characteristics				
LVEF (%)	54.5±9.4	53.8±10.1	54.7±9.2	0.680
sPAP (mm Hg)	41.4±11.6	40.0±12.8	41.9±11.3	0.497
Aortic valve area (cm^2)	0.72±0.13	0.69±0.13	0.73±0.13	0.160
Peak pressure gradient (mmHg)	76.1±16.7	81.8±20.6	76.1±14.8	0.062
Mean pressure gradient (mm Hg)	47.7±11.9	51.2±14.6	46.6±10.7	0.085

Values represent mean±SD, n (%) or median (interquartile range). CABG, coronary artery bypass grafting; COPD, Chronic obstructive pulmonary disease; LVEF, left ventricular ejection fraction; sPAP, systolic pulmonary artery pressure; STS, Society of Thoracic Surgeons.

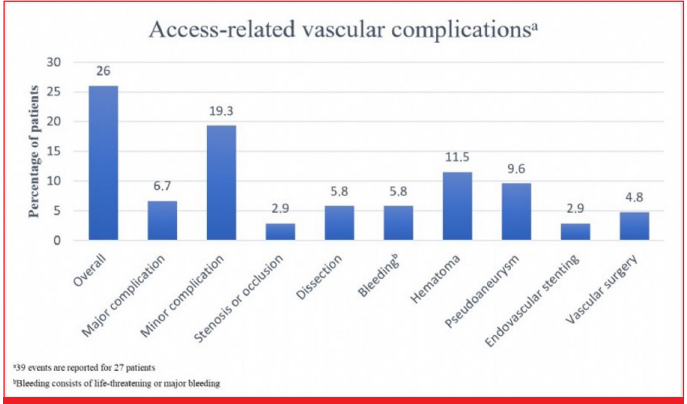

Figure 1. Distribution of access-related vascular complications according to the VARC-3 definitions
VARC, Valvular Academic Research Consortium.

Table 2. CT and procedural characteristics of patients

Parameters	Total (n = 104)	Access-related vascular complications (n = 27)	No Access-related vascular complications (n = 77)	p
CT characteristics				
External iliac artery minimum lumen diameter (mm)	6.3±1.0	5.8±0.9	6.5±1.0	0.003
External iliac artery maximum lumen diameter (mm)	7.3±1.1	6.8±0.9	7.5±1.0	0.003
Calcification anywhere along external iliac artery	42.0 (40.4)	18 (66.7)	24 (31.2)	0.001
Common femoral artery minimum lumen diameter (mm)	6.9±1.2	6.3±1.2	7.1±1.1	0.006
Common femoral artery maximum lumen diameter (mm)	8.0±1.2	7.3±1.2	8.2±1.2	0.003
Calcification anywhere along common femoral artery	27 (26.0)	15 (55.6)	12 (15.6)	< 0.001
Anterior calcification of common femoral artery access site	6 (5.8)	5 (18.5)	1 (1.3)	0.004
90° depth to common femoral artery (mm)	51.6±8.1	56.8±8.8	49.8±7.1	<0.001
45° approach distance to common femoral artery (mm)	54.1±7.3	58.2±9.0	52.7±6.1	0.001
Sheath to femoral artery ratio	1.19±0.20	1.30±0.22	1.15±0.17	0.001
Sheath to external iliac artery ratio	1.10±0.18	1.21±0.21	1.06±0.15	0.001
Procedural characteristics				<0.001
Conscious sedation	98 (94.2)	26 (96.3)	72 (93.5)	1.0
Procedural time, min	126.5±30.5	137.6±38.8	122.2±26.2	0.028
Skin to skin, min	82.0±24.9	92.6±34.0	78.1±19.6	0.012
Contrast medium volume, ml	150 (120- 200)	150 (110-170)	150 (120-205)	0.122
ICU stay, days	2 (1- 3)	2 (1-3)	1 (1-3)	0.869
Hospital stay, days	6.5 (4.1-9.0)	7.5 (5-10.7)	6 (3.2-9)	0.016
Sheath outer diameter (mm)	7.4±0.3	7.5±0.3	7.4±0.3	0.518
18F sheath	24 (23.1)	5 (18.5)	19 (24.7)	0.514
19F sheath	80 (76.9)	22 (81.5)	58 (75.3)	0.514
Proglide, number	2.0±0.2	2.2±0.5	2.0±0.0	<0.001

Values represent mean±SD, n (%) or median (interquartile range). CT, computed tomography; ICU, intensive care unit.

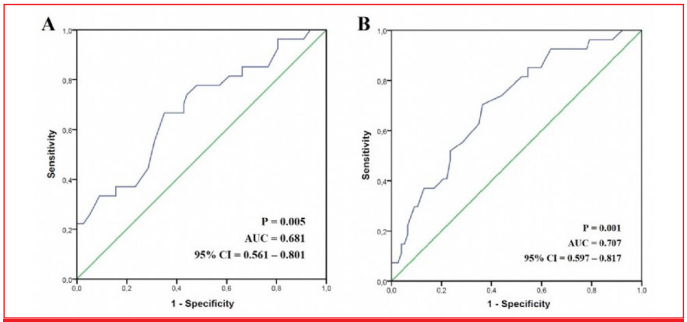

Figure 2. Receiver-operating characteristic (ROC) curves of the SEIAR and SFAR for the prediction of access-related vascular complications.
(A) The AUC value is 0.681 for the SEIAR (cutoff value = 1.14; sensitivity, 66.7%; specificity, 64.9%; negative predictive value, 84.7%; P = 0.005). (B) The AUC value is 0.707 for the SFAR (cutoff value, 1.18; sensitivity, 70.4%; specificity, 63.6%; negative predictive value, 86.0%; P = 0.001). AUC, area under the ROC curve; SEIAR, sheath to external iliac artery ratio; SFAR, sheath to femoral artery ratio.

Table 3. Predictors of access-related vascular complications on univariable and multivariable analysis

Variables	Univariable analysis		Multivariable analysis	
	OR (95% CI)	p	OR (95% CI)	p
Age (years)	0.97 (0.90-1.04)	0.476		
Sex (female)	0.63 (0.26-1.55)	0.323		
Diabetes	0.96 (0.39-2.35)	0.941		
Coronary artery disease	1.31 (0.46-3.70)	0.607		
Chronic kidney disease	2.28 (0.91-5.68)	0.077	2.83 (0.92-8.68)	0.069
Peripheral artery disease	1.90 (0.72-5.03)	0.193		
STS score	0.97 (0.85-1.10)	0.661		
Calcification anywhere along common femoral artery	6.77 (2.54-17.99)	<0.001	5.80 (1.86-18.10)	0.002
90° depth to common femoral artery	1.11 (1.05-1.18)	<0.001	1.10 (1.03-1.17)	0.005
Sheath to femoral artery ratio	4.15 (1.61-10.72)	0.003	4.25 (1.38-13.10)	0.012

CI, confidence interval; OD, odds ratio; STS, Society of Thoracic Surgeons.

Interventional Cardiology / *Heart Valve & Structural Diseases*

PB-025

First experiences with a new balloon-expandable Myval® transcatheter aortic valve, in our center; A preliminary study

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Background and Aim: In present article, we aimed to present our short-term follow-up results of a new type of ballon expandable Myval valve (Meril Life Sciences, India).

Methods: Sixty-eight consecutive patients who underwent transfemoral TAVI from June 2020 to August 2021 were included in the study.

Results: The mean age of the whole population was 78±7.81 years, 39 (57%) were female, and 38 (55.9%) were hypertensive. STS score of the group was 3.7%±1.97%. TAVI procedure was performed by transfemoral route to all patients. In 47 (69.1%) cases, we started the procedure without pre-dilatation. In six (12,8%) cases, the prosthesis did not pass the native valve we had to take the valve back this cases. We succesfully got back the valve in ten cases. In one case, while removing the valve, it was completely detached from the balloon (Figure 1A) and remained in the sheath (Figure 1B). After getting the sheat back completely, we were able to remove the valve by cutting the sheat (Figure 1C), After removing the valve, we implanted the same valve again. But in other case, while retrieving the valve, distal part of the sheat that cannot be seen clearly on Xray stripped the valve from the balloon. Then the valve was implanted into the descending aorta. After that, the another valve was implanted succesfully. We used cut-down 13 (19.1%) patients, Prostar XL in 6 (8.8 %) patients and ProGlide in 49 (72.1 %) patients for vascular closure. Three (4.4%) periprocedural deaths occurred in our study. Vascular complications were observed in 3(4.4%) patient. None of the patients in our study had severe PVL, and three (4.4%) patient had moderate PVL. PPM was

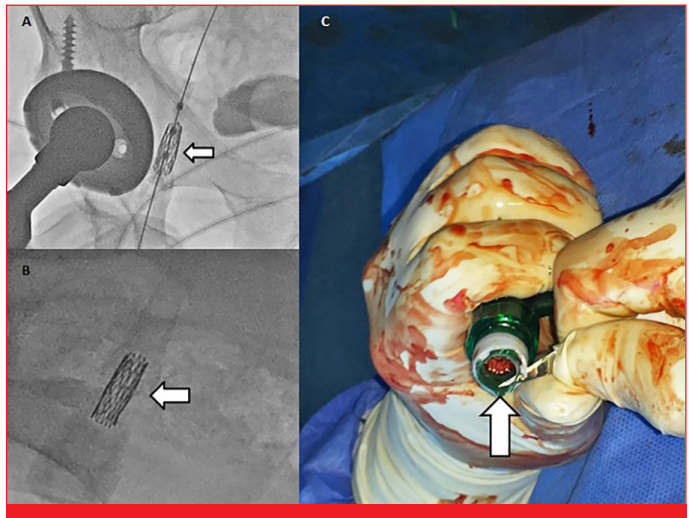

Figure 1. (A) The valve slipped backwards from the balloon (arrow). (B) The valve was completely detached from the balloon and remained in the sheath (arrow). (C) After getting the sheat back completely, we were able to remove the valve by cutting the sheat (White arrows indicate the valve tucked into the sheath)

required in seven (10.3%) patients, and indications were complete AV block. The mean hospital stay in the whole group was 3(2-12) days.

Conclusion: Based on our experiences, a new type of ballon expandable valve is easy to use, efficient and have a few negligible drawbacks such as a need of predilation of sheath. While shaft flexibility may have advantages in some situations including very tortuoses arteries, it may cause some difficulties in alignment of the valves.

Interventional Cardiology / Carotid and Peripheral Vascular

PB-026

Clinical efficacy and safety of the PROglide device as a suture-mediated closure in thorasic endovascular aortic repair in patients with previous groin intervention

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Background and Aim: While the percutaneous approach is increasingly preferred, suture-mediated closure devices have been put into clinical practice to close the femoral artery during procedures requiring a large-sized introducer. However, scar in the groin is considered a contraindication or an exclusion criterion for percutaneous procedures. The aim of our study was to investigate the outcomes and safety of Pro-Glide device as suture-mediated closure device in patients who underwent thoracic endovascular aortic repair with percutaneous femoral access 22 F who had previous groin intervention

Methods: A total of 73 patients who underwent endovascular repair with percutaneous femoral access were retrospectively included in the study. Previous groin intervention was defined as history of open surgical access or large sheath insertion (>18 F) to femoral artery because of endovascular or valvular intervention. Patients were divided into two groups as who had previous groin intervention PGI (þ) and had not PGI ().

Results: A total of 73 patients [60 male (82.2%)] were included in the study. Seventeen patients had PGI, and 56 did not. When groups were compared in terms of sheath sizes, a significantly higher sheath sizes were used in PGI (þ) patients (24.5 1.1 F vs. 23.8 0.9 F, p 1/4 0.005). The overall success rate in the femoral approach with pre-close technique was statistically insignificant between two groups (94.1% vs. 96.4%, p 1/4 0.55). One patient in PGI (þ) group and two patients in PGI () had technical failure for percutaneous femoral approach. One patient (5.9%) in PGI (þ) group and one patient (1.8%) in PGI () group had femoral complications after the procedures; however, there was no significant difference between the groups in terms of complications (5.9% vs. 1.8%, p 1/4 0.13).

Conclusion: A total of 73 patients [60 male (82.2%)] were included in the study. Seventeen patients had PGI, and 56 did not. When groups were compared in terms of sheath sizes, a significantly higher sheath sizes were used in PGI (þ) patients (24.5-1.1 F vs. 23.8-0.9 F, p ¼ 0.005). The overall success rate in the femoral approach with pre-close technique was statistically insignificant between two groups (94.1% vs. 96.4%, p ¼ 0.55). One patient in PGI (þ) group and two patients in PGI () had technical failure for percutaneous femoral approach. One patient (5.9%) in PGI (þ) group and one patient (1.8%) in PGI () group had femoral complications after the procedures; however, there was no significant difference between the groups in terms of complications (5.9% vs. 1.8%, p ¼ 0.13).

Table 1. Anatomical characteristics of CFA detected in computerized tomography and characteristics of sheaths and devices used for patients

	Previous Groin Intervention (+) (n=179)	Previous Groin Intervention (-) (n=56)	*P*
CFA diameter	10.5±1.5	10.6±1.9	0.958
Sheath size diameter, F	24.5±1.1	23.8±0.9	0.005
22	2 (11.8%)	10 (17.9%)	0.720
24	5 (29.4)	36 (64.3%)	0.011
25	8(47.1%)	10 (17.9%)	0.024
26	2 (11.8%)	0 (0.0%)	0.052
Skin to CFA distance,mm	37.1±12.3	33.1±12.8	0.194
Calcificaiton grade of femoral artery	0 (0-1)	1 (0-1)	0.516
Device size used for endovascular repair	41.8±4.2	37.3±4.7	<0.001

Table 2. Demographic and clinical characteristics of patient

	Previous Groin Intervention (+) (n=17)	Previous Groin Intervention (-) (n=56)	*P*
Age, years	58.4±9.4	60.4±10.1	0.497
Male (n, %)	15 (88.2%)	45 (80.4%)	0.719
Body mass index kg/m^2	26.2±5.6	26.9±6.7	0.312
Diabetes mellitus (n, %)	5 (29.4%)	15 (26.8%)	1.0
Hypertension (n, %)	17 (100.0%)	54 (96.4%)	1.0
Hyperlipidemia (n, %)	2 (11.7%)	6 (10.7%)	0.244
Smoking(n-%)	8 (47.1%)	21 (37.5%)	0.481
Glomerular filtration rate (mL / min)	97 (77-106)	86 (40-103)	0.151
Coronary artery disease (n, %)	6 (35.3%)	8 (14.3%)	0.078
Peripheral artery disease	1 (5.9%)	4 (7.1%9)	1.0

Table 3. Procedural data, clinical outcomes and procedural complications

	Previous groin intervention (+) (n=17)	Previous groin intervention (-) (n=56)	*P*
Operating room time,min	87.7±6.9	88.9±10.0	0.898
Manual compression time, min	8.6±1.4	8.2±1.3	0.232
Number of Pro-Glides used			
2 devices	16 (94.1%)	54 (96.4%)	0.456
3 devices	1 (5.9%)	2 (3.6%)	0.367
Technical success for femoral artery closure	16 (94.1%)	54 (96.4%)	0.554
Complications	1 (5.9%)	1 (1.8%)	0.133
Groin infection	0 (0.0%)	0 (0.0%)	0.121
Pseudoaneurysm	0 (0.0%)	0 (0.0%)	
Hematoma/seroma	1 (5.9%)	1 (1.8%)	

Interventional Cardiology / Carotid and Peripheral Vascular

PB-027

Is retinal nerve fiber thickness affected in patients with internal carotid artery stenosis?

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Background and Aim: To evaluate the retinal nerve fiber layer (RNF) of the eye using spectral-domain optical coherence tomography in patients with asymptomatic internal carotid artery (ICA) stenosis.

Methods: A total of 32 patients (20 men, 10 women; mean age: 66.73±6.95 years) with a diagnosis of unilateral ICA stenosis (the percentage of stenosis was between 60 and 90%), and 31 age- and sex-matched healthy subjects (control group) (23 men, 9 women; mean age: 67.03±10.11 years) were included in the study. Patients with ICA stenosis included in the study had no lesions in the external carotid artery and common carotid artery. Using transorbital Doppler and carotid duplex ultrasound examination, we assessed presence and degree of an internal arterial stenosis (ICAS) and we measured the RNFL thickness by spectral-domain optical coherence tomography.

Results: Ophthalmic arterial blood flow in the group with ICA stenosis was statistically significantly decreased compared to the control group (Peak systolic velocity, relatively, 32.70±14.02 vs. 45.50±14.6 cm/s; 0.001*) (End-diastolic velocity, relatively, 8.59±3.01 vs 13.39±5.01 cm/s; <0.001*). There was no statistically significant difference in average RNFL thickness in the group with ICA stenosis compared to the control group (relatively, 102.34±13.73 vs. 103.55±8.01; 0.68).

Conclusion: Although ophthalmic artery systolic and diastolic flows were statistically significantly decreased, The mean RNFL thickness of patients with asymptomatic internal carotid artery (ICA) stenosis did not differ from the control group. The RNFL may be resistant to ischemia in patients with ICA stenosis without lesions in the ipsilateral external carotid artery and common carotid artery.

Interventional Cardiology / Carotid and Peripheral Vascular

PB-028

Impact of vascular access site on procedure success and long-term restenosis among patients undergoing endovascular intervention

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Background and Aim: Favorable results have been obtained with endovascular interventions for iliac artery lesions, compared with the femoral artery lesions. Lesion characteristics and the accepted therapeutic strategy is generally selected based on the Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC-II) classification. However, there is a strong association between long-term restenosis and adverse clinical events in patients who underwent endovascular intervention. Therefore, there have been several reports which revealed the factors associated with long-term restenosis following the endovascular intervention. Yet, a paucity of data exists in the literature that investigates the association between vascular access site and clinical outcomes in this patient population. In this study, we aimed to investigate the impact of vascular access site on procedure success and long-term restenosis among patients undergoing endovascular intervention.

Methods: The records of 406 consecutive patients who underwent endovascular intervention due to symptomatic peripheral artery disease were analyzed. Patients were divided into two groups based on the vascular access sites. For each group, baseline demographic and clinical characteristics, technical aspects of procedures, and long-term restenosis were analyzed.

Results: According to our study, a retrograde access site was chosen in 217 patients (53.4%) with the remaining 189 (46.6%) patients having an antegrade access site. Significantly less fluoroscopy (24 min vs 36 min, p<0.005) and contrast (150 mL vs 195 mL, p <0.05) were used during retrograde access compared with antegrade access. Technical success was achieved in 96.3% of patients with retrograde access vs 96.3%

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	Retrograd Approach (n=217)	Antegrad Approach (n=189)	p
Age, years	60±9	62±8	0.007
Total cholesterol mg/dL	186 (149-226)	169 (127-221)	0.080
LDL mg/dL	112 (67-148)	95 (53-127)	0.127
Some side Abi	0.64±0.17	0.66±0.19	0.595
Opposite side Abi	0.85±0.21	0.81±0.20	0.362
Follow up time month	32±15	31±17	0.965
LVEF %	60 (50-65)	60 (50-65)	0.553
Max balloon diameter mm	6 (5-7)	6 (6-6)	0.880
Max balloon lenght mm	60 (40-80)	65 (60-100)	0.127
Max stent diameter mm	8 (7-9)	8 (7-8)	0.005
Total stent lenght mm	60 (39-98)	69 (40-114)	0.071
Postdilatation max balloon diameter mm	7 (7-8)	7 (6-8)	0.039
Opac cc	180 (100-200)	195 (120-240)	0.006
Intervention time min	24 (15-40)	36 (22-57)	<0.001
Creatinin mg/dL	0.91 (0.78-1.10)	0.91 (0.76-1.16)	0.727
HDL mg/dL	40 (3-48)	40 (33-51)	0.512
Triglicerde mg/dL	165 (106-251)	138 (97-219)	0.027
Gender (male) n (%)	195 (89.9)	168 (88.9)	0.791
DM n (%)	110 (50.7)	104 (55.0)	0.383
HT n (%)	135 (62.2)	137 (72.5)	0.028
COPD n (%)	42 (19.4)	46 (24.3)	0.224
Atrial fibrillation n (%)	10 (4.6)	15 (7.9)	0.164
Coronary artery disease	132 (60.8)	112 (59.2)	0.747
Smoking n (%)	148 (68.2)	128 (67.7)	0.198
Previous PAD n (%)	49 (22.6)	51 (27.0)	0.304
Previous intervention n(%)	45 (20.7)	47 (24.9)	0.321
Rutherford class			
1	0 (0)	1 (0.5)	
2	51 (23.5)	45 (23.8)	
3	127 (58.5)	101 (53.4)	0.272
4	25 (11.5)	25 (13.2)	
5	7 (3.2)	14 (7.4)	
6	7 (3.2)	3 (1.6)	
Fontaine class			
2	0 (0)	1 (0.5)	
2a	55 (25.3)	51 (27.0)	
2b	126 (58.1)	99 (52.4)	0.635
3	24 (11.1)	26 (13.8)	
4	12 (5.5)	12 (6.3)	
Previous CVD n (%)	20 (9.2)	26 (13.8)	0.150
Previous Amputation n(%)	6 (2.8)	6 (3.2)	0.808
Amputation	6 (2.8)	9 (4.8)	0.287
Statin mg/dL	140 (64.5)	122 (64.6)	0.994
Antiaggregant mg/dL	216 (99.5)	189 (100.0)	0.534
Preprocedural distal run-off n(%)			
0	3 (1.5)	2 (1.1)	0.111
1	10 (4.9)	17 (9.0)	
2	40 (19.6)	39 (21.4)	
3	151 (74.0)	116 (66.7)	
Postprocedural distal run-off			
0	3 (1.5)	2 (1.1)	
1	10 (4.9)	16 (9.2)	0.161
2	41 (20.1)	40 (23.0)	
3	150 (73.5)	116 (66.7)	
Chronic total occlusion n(%)	78 (35.9)	85 (45.1)	0.050
TASC n(%)			
A	77 (35.6)	53 (28.2)	
B	80 (37.0)	66 (35.1)	0.198
C	42 (19.4)	50 (26.6)	
D	17 (7.9)	19 (10.1)	
Operation success n (%)	209 (96.2)	182 (96.2)	0.993
Coart technic n (%)	1 (0.5)	4 (2.1)	0.146
Procedürel complication n (%)	30 (13.8)	31 (16.4)	0.468
Residüel stenosis n (%)	29 (13.4)	39 (20.6)	0.050
In hospital trombosis n (%)	0 (0)	2 (1.1)	0.216
Long term restenosis 2-5 year	41 (18.9)	36 (19.0)	0.969
Restenosis time month	15±9	15±11	0.831

Table 1.

of patients with antegrade access (p =0.99). The overall rate of post-procedural complications was comparable for both groups (13.8 vs 16.4, p =0.468) and there were no statistical differences in terms of long-term restenosis (18.9 vs 19.0, p =0.96)

Conclusion: Our data showed, both retrograde and antegrade vascular access can be performed safely in patients undergoing endovascular intervention.

Interventional Cardiology / Carotid ve Peripheral Vascular

PB-030

Role of C-reactive protein/albumin ratio in predicting long-term restenosis after endovascular treatment of the iliac arteries

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Background and Aim: Treatment of iliac artery stenosis with endovascular interventions is one of the routine procedures in our clinical practice. However, as the number of procedures performed increases, the complications increase at that rate. One of the important complications after endovascular interventions is restenosis. As in the atherosclerotic process, one of the important factors in the restenosis process is inflammation. In addition to the classical markers showing inflammation, one of the parameters that has increased in popularity in recent years is the C-reactive protein (CRP)/albumin ratio. This parameter, which has been shown to play a role in the prognostic process of many cardiovascular diseases in previous studies, was also associated with poor outcomes in patients with peripheral artery disease.Our aim in this study is to investigate the role of CRP/albumin ratio in predicting restenosis that may develop after endovascular intervention in patients with iliac artery stenosis.

Methods: Between 2016 and 2019, consecutive 143 patients who underwent endovascular intervention due to iliac artery stenosis in our center were included. Patients with a history of acute coronary syndrome in the last 1 year, heart failure, end-stage renal disease, malignancy were not included in the study. During the follow-up period, the patients were divided into 2 groups as those who developed restenosis and those who did not. Basic demographic and procedural characteristics were compared between the 2 groups. At the same time, the predictive value of the CRP/albumin ratio in patients with restenosis was investigated.

Results: As a result of our study, the number of patients who developed restenosis in the long-term follow-up was found to be 25 (17.4%). The mean follow-up period was 40 months in the group that developed restenosis and 36.5 months in the group that did not develop restenosis. The lesion severity determined by TASC scoring was higher in the restenosis group, and the rate of restenosis was higher in patients who were treated without stent and patients who have residual stenosis after intervention. In the comparison made in terms of CRP, albumin and CRP/albumin values, the rate of restenosis was found to be significantly higher in the long-term follow-up in patients with high CRP and CRP/albumin values and low albumin values (p <0.001). In the multivariate analysis, the CRP/albumin ratio and not using stent in treatment were determined as an independent predictor of long-term restenosis in patients undergoing iliac artery intervention.

Conclusion: Our data showed that, there was a strong association between CRP/albumin ratio and long term restenosis in patients undergoing iliac artery intervention. This simple

	Restenosis (–) (n=118)	Restenosis (+) (n=25)	P
Age, years	60.3±9.2	59.4±8.3	0.632
Gender (male) n (%)	100 (84.7)	19 (76.0)	0.216
DM n (%)	68 (57.6)	11 (44.0)	0.213
HT n (%)	81 (68.6)	16 (64.0)	0.652
CKD n (%)	28 (23.7)	3 (12.0)	0.196
COPD n (%)	28 (23.7)	12 (48.0)	**0.014**
AF, n (%)	7 (5.9)	2 (8.0)	0.489
CAD n (%)	78 (66.1)	19 (76.0)	0.336
CVD n (%)	17 (14.4)	3 (12.0)	0.521
Smoking n (%)	80 (67.8)	21 (84.0)	0.106
Previous PAD n (%)	29 (24.6)	7 (28.0)	0.720
FONTAINE class, n (%)			
1	1 (0.8)	0 (0)	
2a	36 (30.5)	4 (16.0)	
2b	65 (55.1)	15 (60.0)	0.505
3	11 (9.3)	4 (16.0)	
4	5 (4.2)	2 (8.0)	
Previous amputation			
None n (%)	114 (96.6)	23 (92.0)	
Right n (%)	0 (0)	2 (8.0)	0.282
Left n (%)	4 (3.4)	0 (0)	
TASC II			
A	42 (35.6)	5 (20.0)	
B	41 (34.7)	7 (28.0)	**0.065**
C	28 (23.7)	8 (32.0)	
D	7 (5.9)	5 (20.0)	
Contralateral lesion, n (%)	44 (37.3)	8 (32.0)	0.618
Intervention type			
Unilateral, n (%)	91 (77.1)	24 (69.0)	**0.021**
Bilateral, n(%)	27 (22.9)	1 (4.0)	
Stent implantation n			
None, n (%)	9 (7.6)	9 (36.0)	**0.001**
Self expandable n (%)	44 (37.3)	3 (12.0)	
Baloon expandable n (%)	53 (44.9)	8 (32.0)	
Both, n (%)	12 (10.2)	5 (20.0)	
Postdilatation, n (%)	40 (33.9)	5 (20.0)	0.174
Residuel stenosis, n (%)	15 (12.7)	10 (40.0)	**0.003**
Creatinine, mg/dL	0.96 (0.76-1.19)	0.91 (0.79-1.05)	0.560
Neutrophil count, 10^3/mm^3	5.48 (4.38-7.32)	5.22 (3.94-7.51)	0.491
Lymphocyte count, 10^3/mm^3	2230 (1770-2765)	2040 (1810-2600)	0.536
Platelet count, 10^3/mm^3	281.3±117.49	287.4±81.93	0.807
C-Reactive protein, mg/L	5.48 (2.91-15.40)	29.0 (21.64-56.0)	**<0.001**
Albumin, g/dl	4.2 (4.0-4.5)	2.5 (2.3-2.7)	**<0.001**
CRP/Albumin ratio	1.10 (0.55-3.75)	12.61 (7.00-20.0)	**<0.001**
Follow-up time, months	36.5 (23-44)	40 (27-48)	0.117

DM:Diabetes mellitus HT:Hypertension CKD:Chronic kidney disease COPD:Chronic obstructive pulmonary disease, AF:Atrial fibrillation, CAD:Coronary artery disease CVD:Cerebrovascular disease PAD:Peripheric artery disease

Table 1. The demographic, clinical and laboratory characteristics of patients

Variables	OR	95% CI	p
DM	2.441	0.884-6.739	0.085
COPD	1.257	0.414-3.819	0.686
Absence of stent implantation	5.315	1.619-17.453	**0.006**
CRP/Albumin ratio	1.066	1.014-1.121	**0.012**

DM:Diabetes mellitus, COPD:Chronic obstructive pulmonary disease,

Table 2. Multivariable logistic regression analysis of the variables for restenosis

tool may help determine the long-term outcomes of interventions in patients with iliac artery stenosis. Also, based on the results of our study, the use of stents in the endovascular treatment of patients with iliac artery stenosis reduces long-term restenosis.

Interventional Cardiology / Carotid and Peripheral Vascular

PB-031

Serum Sortilin as a Predictor of Stroke in Patients with Intermediate Carotid Artery Stenosis

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Background and Aim: Sortilin was an important molecular protein involved in the pathogenesis of atherosclerosis. Besides, serum sortilin was associated with adverse cerebrovascular events. Atherosclerotic in carotid artery is a major etiology for ischemic stroke. The risk of stroke in patients with intermediate carotid artery stenosis (CAS) was unknown. Hence, the aim of the present study was to evaluate the relationship between serum sortilin levels and stroke in patients with intermediate CAS.

Methods: A total of 195 intermediate CAS patients were included in this crosssectional study. The patients were divided into two groups as symptomatic (N=95) and asymptomatic (N=100) patients. Patients with a transient ischemic attack (TIA), retinal ischemic event, or ischemic stroke resulting from the narrowed carotid artery were considered to be symptomatic. Serum sortilin concentrations were measured using the enzyme-linked immunosorbent assay

Results: Serum sortilin level was significantly higher in the symptomatic group than in the severe asymptomatic group (1.53±0.25 ng/mL vs. 1.34±0.19 ng/mL, p<0.001) (Figure.2, Table.1). Besides, high serum sortilin levels (OR 4.91, 95% CI 1.24-19.51, p=0.023) was identified as independent predictors of symptomatic carotid plaque (Figure.3 Table.2). In the ROC curve analysis, serum sortilin levels higher than 1.34 ng/mL predicted stroke/TIA with a sensitivity of 66.3% and a specificity of 67% (AUC = 0.725, p <0.001) (Figure.1).

Conclusion: Serum sortilin level is increased in the presence of symptomatic intermediate CAS and may have clinical value in the management of patients with carotid artery disease.

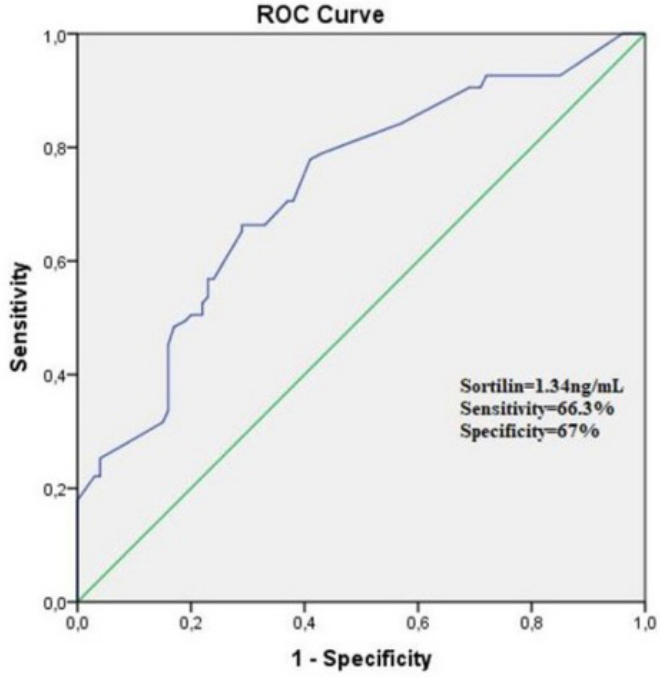

Figure 1. ROC

Parameters	Symptomatic patients (N=95)	Asymptomatic patients (N=100)	P value
Clinical variables			
Age, years	62.35±6.24	62.48±5.62	NS
Gender, male, n (%)	75(79)	76(76)	NS
Hypertension, n (%)	44(46)	49(49)	NS
Diabetes mellitus, n (%)	39(41)	31(31)	NS
Smoking, n (%)	35(37)	50(50)	NS
Systolic blood pressure (mmHg)	123.16±17.08	126.10±16.93	NS
Diastolic blood pressure (mmHg)	75.05±8.33	74.45±9.42	NS
Previous CAD, n (%)	42(44)	47(47)	NS
Pre-hospital medications, n (%)			
Aspirin, n (%)	69(76)	71(71)	NS
Clopidogrel, n (%)	16(17)	15(15)	NS
Beta-blockers, n (%)	53(56)	62(62)	NS
Statin, n (%)	87(91)	89(89)	NS
ACE-inhibitors or ARB, n (%)	26(27)	21(21)	NS
Hemoglobin, g/dL	12.49±1.78	12.42±1.99	NS
Platelet count, 10^3/mm^3	227.37±56.21	222.8±39.08	NS
Lymphocyte count,10^3/mm^3	1005.89±427	1478±830	<0.001
White blood cell count,10^3/mm^3	6439±2006	5537±1898	0.001
Serum glucose, mg/dL	126.05±50.3	135.04±63.5	NS
Serum creatinine, mg/dL	0.85±0.19	0.81±0.18	NS
Uric acid, mg/dL	5.53±0.97	5.52±0.83	NS
BUN, mg/dL	31.53±5.02	32.41±7.67	NS
eGFR, mL/min/1.73 m^2	74.74±15.68	74.92±14.49	NS
hs-CRP, mg/dL	4.63±1.5	4.12±0.96	0.005
Total cholesterol, mg/dL	202.31±40.39	189.81±41.84	0.035
HDL-cholesterol, mg/dL	44.57±7.9	42.83±7.1	NS
LDL-cholesterol, mg/dL	141.62±39.6	121.17±34.2	<0.001
Triglyceride, mg/dL	138.22±65.07	110.42±25.39	<0.001
Sortilin, ng/mL	1.53±0.25	1.34±0.19	<0.001
NLCR	7.33±3.22	4.79±2.58	<0.001

Abbreviation: ACE= angiotensin-converting enzyme; ARB = angiotensin-receptor blocker; HDL=high-density lipoprotein; eGFR: Estimated glomerular filtration rate; LDL=low-density lipoprotein; NS=non-significant; CAD=coronary artery disease; hs-CRP=high sensitive C-reactive protein; NLCR=neutrophil-lymphocyte count ratio; BUN=blood urea nitrogen.

Table 1. Comparison of demographic, clinical and biochemical characteristics of patients with and without transient ischemic attack or ischemic stroke

Variables	Multivariable analysis	
	OR (95% CI)	P value
Age, years	0.94 (0.87-1.02)	0.162
Gender (male)	2.13 (0.82-5.59)	0.121
NLR	1.37 (1.12-1.68)	0.002
Triglyceride, mg/dL	1.001 (0.99-1.02)	0.238
LDL-cholesterol, mg/dL	1.01 (1.00-1.02)	0.040
Sortilin, ng/mL	4.91 (1.24-19.51)	0.023
hs-CRP, mg/dL	0.74 (0.51-1.07)	0.108

Abbreviation: CI: Confidence interval; LDL: Low-density lipoprotein; NLR: neutrophil-lymphocyte count ratio; OR: Odds ratio; hs-CRP: High sensitive C-reactive protein

Table 2. Independent predictors of symptomatic carotid plaque in the multivariate logistic regression analysis

Interventional Cardiology / Coronary

PB-032

The Evaluation of in-hospital and long-term mortality in patients with ST-segment elevation myocardial infarction who underwent primary coronary angiography during on-hours and off-hours

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Background and Aim: There are controversial data on the time of primary coronary angiography (PCA), the success of the procedure, and its effects on clinical outcomes and mortality in patients presenting with ST-segment elevation myocardial infarction (STEMI) during and out of working hours. The aim of this study was to assess the impact of the admission time (on-hours vs off-ours) on in-hospital and long-term all-cause mortality in STEMI.

Methods: A total of 277 patients who were admitted with a diagnosis of STEMI between January 2013 to June 2019 and underwent PCA were included retrospectively. Clinical characteristics, medication, laboratory results, and coronary angiography records of the patients were obtained from the hospital database. Patients or their relatives were called by phone and mortality data were obtained. Patients were classified into two groups as those who underwent PCA on-hours (08:00 AM until 05:00 PM on weekdays) and those who underwent PCA off-hours admission (05:00 PM until 08:00 AM on weekdays and 24 h on weekends and holidays).

Results: The mean age of the patients was 62.5±12.9 and 76.9% were male. One hundred thirty patients were in the on-hours PCA group and one hundred forty-seven patients in the off-hours PCA group. Baseline characteristics, gender, risk factors except for hyperlipidemia, prior medication, MI localization, culprit coronary artery, PCA results, laboratory results except for platelet count, and left ventricular ejection fraction were similar in both groups. Medical contact to PCA duration was longer in the off-hours PCA group (<0.001). Despite this delay, in-hospital and long-term all-cause mortality were not different during the median follow-up period of 42.6±24.5 months.

Conclusion: Our study is in line with studies showing that there is no difference between in-hospital and long-term mortality in STEMI patients whose PCA performed on-hours or off-hours.

Table 1. Clinical characteristics of the patients.

Characteristics	Overall (n = 277)	PCA on-hours (n = 130)	PCA off-ours (n = 147)	*P*
Age (years), mean±SD	62.5±12.9	63.4±12.7	61.8±13.1	0.302
Male, n (%)	213 (76.9)	106 (81.5)	107 (72.8)	0.395
Active smoker, n (%)	131 (47.3)	61 (46.9)	70 (47.6)	0.908
Hypertension, n (%)	100 (36.1)	42 (32.3)	58 (39.5)	0.216
Diabetes mellitus, n (%)	82 (29.6)	39 (30)	43 (29.3)	0.892
Hyperlipidemia, n (%)	27 (9.7)	7 (5.4)	20 (13.6)	0.021
History of CAD, n (%)	50 (18.1)	25 (19.2)	25 (17.1)	0.650
Prior betablocker use, n (%)	27 (11.1)	10 (9.1)	17 (12.8)	0.362
Prior ASA use, n (%)	36 (14.4)	13 (11.4)	23 (16.9)	0.217
Prior ACEI/ARB use, n (%)	40 (17.2)	13 (12.4)	27 (21.1)	0.079
Prior P2Y12 inhibitor use, n (%)	11 (4.4)	4 (3.5)	7 (5.2)	0.521

Table 1. Clinical characteristics of the patients. (Continued)

Characteristics	Overall (n = 277)	PCA on-hours (n = 130)	PCA off-ours (n = 147)	*P*
Anterior MI, n (%)	118 (42.6)	56 (43.1)	62 (42.2)	0.594
Inferior MI, n (%)	129 (46.6)	58 (44.6)	71 (48.3)	0.594
Posterior MI, n (%)	14 (5.1)	9 (6.9)	5 (3.4)	0.594
Lateral MI, n (%)	16 (5.8)	7 (5.4)	9 (6.1)	0.594
LMCA, n (%)	2 (0.7)	1 (0.8)	1 (0.7)	0.930
LAD, n (%)	126 (46.7)	58 (45)	68 (48.2)	0.930
Cx, n (%)	29 (10.7)	14 (10.9)	15 (10.6)	0.930
RCA, n (%)	111 (41.1)	56 (43.4)	55 (39)	0.930
Intermediate, n (%)	1 (0.4)	0 (0)	1 (0.7)	0.930
SVG, n (%)	1 (0.4)	0 (0)	1 (0.7)	0.930
Medical contact to PCA, min	62.3±51.8	49.4±41.7	73.5±57.1	<0.001
Cardiogenic shock on admission, n (%)	19 (6.9)	9 (6.9)	10 (6.8)	0.968
EF (%), mean±SD	44.6±9.9	44.8±9.4	44.5±10.3	0.870
Hemoglobin, g/L	14.2±2	14.1±2.1	14.3±1.9	0.360
Platelets, 10^3/μL	252.1±69	240.4±65	262.3±71	0.008
WBC, 10^3/μL	11.4±4	11.5±4.5	11.4±3.5	0.890
Creatinine, mg/dl	0.96±0.47	1±0.51	0.93±0.43	0.194
Uric acid, mg/dl	5.5±1.7	5.6±1.8	5.4±1.7	0.308

Table 2. Mortality rates of the patients.

	Overall (n = 277)	PCA on-hours (n = 130)	PCA off-ours (n = 147)	*P*
In-hospital mortality, n (%)	29 (10.5)	13 (10)	16 (10.9)	0.810
Long-term mortality, n (%)	38 (13.7)	16 (12.3)	22 (15)	0.601

PB-033 [Interventional Cardiology / Coronary]

Usefulness of the Metabolic Score for Insulin Resistance (METS-IR) for the Prediction of Contrast Induced Acute Kidney Injury

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Background and Aim: Contrast induced acute kidney injury (CI-AKI) that occurs in patients after coronary angiography performed for elective or acute coronary syndrome is a frequently observed complication associated with contrast agent. Previous clinical studies have shown that the development of CI-AKI is associated with poor prognosis in patients undergoing coronary angiography for elective or acute coronary syndrome. The metabolic score for insulin resistance (METS-IR) has emerged as a new non-insulin-based marker to assess insulin resistance and cardiometabolic risk. METS-IR has been reported to be strongly associated with predictive abilities for hypertension and type 2 diabetes. In this study, we aimed to determine the predictive role of METS-IR for CI-AKI in patients who underwent coronary angiography for any reason and were followed up medically or treated with percutaneous coronary intervention.

Methods: In this retrospective study, a total of 238 patients who underwent coronary angiography for any reason and were followed up medically or treated with percutaneous coronary intervention were included. CI-AKI was defined as an increase in serum creatinine (SCr) ≥ 0.5 mg/dL within 48 hours or a percentage increase in SCr ≥ 25% from baseline. METS-IR was calculated as Ln ((2*fasting glucose)+fasting triglycerides)*body mass index)/(Ln(high-density lipoprotein cholesterol)).

Results: The incidence of CI-AKI was 12.6% (n=30 patients). The patients with CI-AKI were older and diabetic. Patients who developed CI-AKI had a significantly lower METS-IR compared to those who did not (2.32±0.19 vs. 2.48±0.23, p =0.001). Multivariate logistic regression analyses revealed that METS-IR, contrast agent volume and hemoglobin were an independent predictor of CI-AKI (OR: 0.060, 95%CI: 0.007-0.498; p =0.009), (OR: 1.016, 95%CI: 1.009-1.023; p <0.001) and (OR: 0.755, 95%CI: 0.601-0.949; p =0.016), respectively. A receiver operating characteristic curves analysis yielded that the optimal cut-off value of METS-IR for CI-AKI was 2.32 with sensitivity 73.6% and specificity 60.7% (AUC: 0.695, 95%CI: 0.602-0.789, p =0.001).

Conclusion: Based on the study findings, we were able to demonstrate that the METS-IR may be an independent predictor of CI-AKI in patients undergoing coronary angiography for elective or acute coronary syndrome. This simple and easily obtained risk score might indicate in whom patients are at a greater risk for the development CI-AKI following coronary angiography.

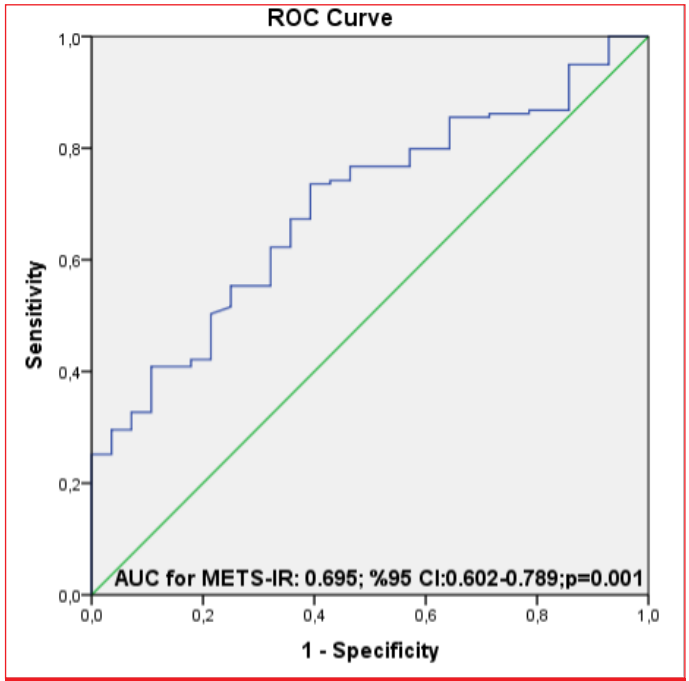

Figure 1. A receiver operating characteristic curves analysis yielded that the optimal cut-off value of METS-IR for CI-AKI

Table 1. Baseline characteristic of all patients

	CI-AKI (-) (n=208)	CI-AKI (+) (n=30)	*P*
Age, years	60.3±11.5	69.4±11.5	<0.001
Female, gender, n (%)	126 (60.6)	17 (56.7)	0.684
Body mass index, kg/m^2	29.5±5.7	28.7±5.9	0.455
Hypertension, n (%)	121 (58.2)	20 (66.7)	0.376
Diabetes mellitus, n (%)	61 (29.3)	15 (50.0)	0.023
Hyperlipidemia, n (%)	35 (16.8)	2 (6.7)	0.151
Current smoking status, n (%)	45 (21.6)	9 (30.0)	0.306
Chronic heart failure, n (%)	34 (16.3)	2 (6.7)	0.167
Admission laboratory variables			
Admission glucose, mg/dL	136.5±59.8	123.0±47.6	0.353
Admission BUN, ng/mL	36.8±17.0	39.8±17.0	0.368
Creatinine, mg/dL	0.78±0.17	0.86±0.19	0.019
Estimated glomerular filtration rate, mL/min	89.0±21.6	86.0±23.1	0.498
White blood cell count, cells/μL	7.45±2.58	6.83±2.08	0.217
Hemoglobin, g/dL	13.02±1.73	11.35±2.67	<0.001
Hematocrit, %	38.5±4.9	36.0±7.2	0.018
Platelet count, cells/μL	238.9±59.9	257.2±81.3	0.012
C-reactive protein, mg/L	5.4±3.8	5.1±2.7	0.642
Sodium, mmol/L	139.0±2.5	139.2±2.8	0.756
Potassium, mmol/L	4.1±0.5	4.2±0.6	0.323
Triglyceride, mg/dL	158.6±106.8	140.9±94.1	0.414
HDL-cholesterol, mg/dL	42.5±10.4	44.8±10.6	0.277
LDL-cholesterol, mg/dL	115.5±37.3	112.0±36.9	0.645
Echocardiographic parameters			
LVEF, %	54.8±9.3	58.6±4.7	0.031
Coronary angiography procedure			
Duration, min	18.2±11.4	30.6±25.4	<0.001
Contrast agent volume, mL	121.9±67.7	219.6±82.7	<0.001
METS-IR	2.48±0.23	2.32±0.19	0.001

Continuous variables are presented as mean±SD, nominal variables presented as frequency (%). BUN indicates blood urea nitrogen; HDL indicates high density lipoprotein; LDL indicates low density lipoprotein; LVEF indicates left ventricle ejection fraction; METS-IR indicates The metabolic score for insulin resistance.

Interventional Cardiology / Coronary

PB-034

The relationship between the success of chronic total occlusion procedure and retrograde coronary artery filling grade

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Background and Aim: The most important cause of myocardial dysfunction is coronary artery disease (CAD). Coronary chronic total occlusion (CTO) is defined as a true total occlusion with complete interruption of antegrade blood flow as assessed by coronary arteriography and with an estimated duration of occlusion of ≥3 months. In this study, it was aimed to evaluate the success of CTO procedure and the importance of retrograde coronary filling grade.

Methods: The patients were divided into four groups according to their retrograde Rentrop circulation levels (n=210). All patients were treated by experienced operators who had been performing CTO procedures. Coronary collateral circulation was graded according to the Rentrop classification. Grading was performed as no filling (0); filling of side branches via collateral channels without visualization of epicardial segment (1); partial filling of the epicardial major coronary artery via collateral channels (2); and complete filling of the epicardial major coronary artery (3). CTO scores of the patients were calculated. Categorical variables were shown as percentage and compared using the χ^2 test and continuous variables as mean ±standard deviation and compared using ANOVA test. Univariate and multivariate analyzes were used to identify the independent predictors of procedural success of CTO-PCI.

Results: The mean age was 60.5 (±12.3) and 176 (83.8%) of the patients were male. Antegrade CTO procedure success rate 64.8%. The technique used was anterior wire escalation (AWE) in 69.1% of patients who underwent successful procedures, and anterior dissection and reentry (ADR) in 30.9% (p =0.001). It was determined that the success of the process increased as the reentrop level increased. It was determined that the highest transaction success was at the reentrop-III level (p =0.012). EBU for the left system and AR for the right system were the most preferred catheters. For the beginning, the ones with good microchannel transition such as Fielder and Pilot 50 were preferred. The process was continued with hydrophilic or Gaia first or second wires at the second level. In patients whose CTO lesion could not be achieved with these two-level wires, wires with high tip pressure and high torque, such as conquest and confienza were used for the procedure. The ADR method was tried in patients who could not achieve lesion transfer with the AWE method. As factors affecting the success of CTO procedure after analyzes were, male gender, J-CTO score, infarct related SYNTAX score, calcification, tortuosity, retrograde filling degree, AWE technique, presence of microchannels (Table 2) (Table 3).

Conclusion: There are several variables that affect CTO transaction success. As the retrograde filling quality increases, the success of the CTO procedure also increases. Longer segments and diffuse lesions are detected in coronary arteries without retrograde filling. It will be useful to evaluate the retrograde filling level when planning for CTO success before the procedure.

Table 1. Retrograde filling grade (Rentrop classification) ant CTO success rate

Rentrop Grade	Unsucessfull procedure	Successfull procedure	Total
Grade 0	26 (12.4%)	16 (7.6%)	42 (20%)
Grade 1	24 (11.4%)	38 (18.1%)	62 (29.5%)
Grade 2	20 (9.5%)	38 (18.1%)	58 (27.6%)
Grade 3	4 (1.9%)	44 (21.0%)	48 (22.9%)
Total	74 (35.2%)	136 (64.8%)	210 (100%)

Table 2. As factors affecting the success of CTO procedure after analyzes were, male gender, J-CTO score, infarct related SYNTAX score, calcification, tortuosity, retrograde filling degree, AWE technique, presence of microchannels

Variables	Multivariate Analysis Odds ratio (95% CI)	Multivariate Analysis *P*
Male	3.8 (1.3-8.3)	0.010
J-CTO score	6.2 (1.6-12.9)	<0.001
Infarct related artery SYNTAX score	5.9 (2.4-10.8)	0.001
Anterior wire escalation technigue	1.8 (0.8-3.4)	0.048
Microchannel	1.7 (0.9-3.5)	0.050
Retrograde filling grade	4.1 (1.5-7.9)	0.007
Tortuosity	2.3 (1.4-3.8)	0.032
Calcification	3.2 (1.3-6.2)	0.016
CTO blunt	2.1 (1.0-4.2)	0.039

Interventional Cardiology / Cornary

PB-036

Pharmacoinvasive Strategy Versus Primary Percutaneous Coronary Intervention In St-Elevation Myocardial Infarction (From An Experience Of A Tertiary Center)

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Background and Aim: It has been shown that the superiority of primary percutaneous coronary intervention (PCI) over fibrinolysis by numerous studies in patients with ST-elevation myocardial infarction (STEMI). Primary PCI is the preferred reperfusion strategy for STEMI after first medical contact. However, timely access to primary PCI remains a major problem due to logistics restrictions and system delays. Therefore, a pharmacoinvasive strategy of fibrinolysis followed by coronary angiography and PCI may be superior to delayed primary PCI in these patients. There are currently limited data regarding the long-term outcomes of pharmacoinvasive strategy compared with primary PCI in STEMI patients. In this retrospective analysis, we aimed to compare the long-term outcomes of patients treated with a pharmacoinvasive or a primary PCI strategy in an experience of a tertiary center.

Methods: 869 patients with STEMI were enrolled in this study. The patients were divided into 2 groups according to the reperfusion strategy: pharmacoinvasive or primary PCI. The primary end point of the study was the occurrence of all-cause mortality. The secondary end points included recurrent MI, target vessel revascularization, heart failure admission, major bleeding, and stroke at follow-up.

Results: Median follow-up was 7.6 (IQR 5.9 to 8.3) and 6.4 (IQR 4.3 to 7.9) years in the pharmacoinvasive and primary PCI groups, respectively. Among patients undergoing reperfusion therapy for STEMI, 71.1% (n = 618) received primary PCI and 28.9% (n = 251) received a pharmacoinvasive agent. Of these, all patients underwent subsequent coronary angiography and PCI. Among patients undergoing a pharmacoinvasive strategy, 19.1% (n = 48) required a rescue/urgent procedure and 80.9% received a scheduled procedure (n = 203). At follow-up, the incidence of all-cause mortality was 24.7% in pharmacoinvasive patients and 29.6% in patients with primary PCI (p = 0.145).

Conclusion: A pharmacoinvasive strategy in patients with STEMI can be a reasonable alternative to primary PCI when primary PCI delay is expected, or PCI is not available.

Variables	Pharmacoinvasive strategy (n = 251)	Primary PCI (n = 618)	p-value
Age, years	58.5 ± 11.5	61.2 ± 12.6	0.004
Men n (%)	45(17.9)	163(26.4)	0.008
Smoking n (%)	124 (54.1)	224 (38.8)	<0.001
Hypertension n (%)	107 (46.7)	249 (42.9)	0.318
Diabetes mellitus n (%)	56 (24.6)	142(24:5)	0.991
History of CAD n (%)	38(16.6)	114 (19.8)	0.300
Dyslipidemia n (%)	54 (3.6)	142(18:4)	0.094
Stroke/TIA n (%)	2 (0.9)	26 (4.5)	0.011
Fibrinolytic agent			-
Tenecteplase n (%)	133 (53)		-
Alteplase n (%)	94 (37.5)		-
Retaplaz n (%)	2 (0.8)		-
STK n (%)	22(8.8)		-
Rescue PCI after fibrinolysis n (%)	48 (19.1)		-
Scheduled PCI after fibrinolysis n(%)	203 (80.9)		-
Death from any cause n (%)	62(24.7)	183 (29.6)	0.276
Myocardial re-infarction n (%)	32 (12.7)	72 (11.7)	0.651
Target-vessel revascularization n (%)	50 (19.9)	84 (13.6)	0.019
Heart failure admission n(%)	17 (6.8)	33 (5.3)	0.411
Stroke at follow-up n (%)	7 (2.8)	26 (4.2)	0.321
Bleeding			0.921
Major n (%)	11 (4.4)	26 (4.2)	
Minor n(%)	10 (4)	18 (2.9)	

Table 1. Demographic data of patients and outcomes

Interventional Cardiology / Coronary

PB-037

Modified glasgow prognostic score estimates high grade intracoronary thrombus burden in patients with acute anterior myocardial infarction

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Background and Aim: High-grade intracoronary thrombus (ICT) burden is critical due to leading to larger myocardial injury following anterior myocardial infarction (MI). The modified Glasgow prognostic score (mGPS) is novel immune-inflammatory tool consisting of C-reactive protein (CRP) and albumin concentration and was evaluated in malignancy predominantly, and in heart disease recently. It was aimed to investigate the role of mGPS in predicting high-grade ICT in anterior MI patients.

Methods: This prospective trial was performed between February 2017 and March 2020. The mGPS was obtained from admission blood parameters. The ICT burden was evaluated angiographically. Except mGPS, other immune-inflammatory indices were also calculated through the gained parameters. Patients were separated into two groups according to the ICT burden and obtained data was compared between groups.

Results: A total of 1132 patients with mean age of 61±12.4 years, consisting of 370 males (32.7%) were enrolled. Prognostic nutritional index (PNI), albumin, and total serum protein were higher in low-grade ICT group, whereas mGPS, CRP, and CRP to albumin ratio (CAR), were higher in high-grade ICT group. Multivariable regression analysis showed that mGPS [OR:4.514; 95%CI: 2.709-7.524; p <0.001], dichotomized mGPS (≥1) [OR:7.391; 95%CI: 3.910-13.972; p <0.001], CAR [OR:1.147; 95%CI: 1.116-1.178; p <0.001], and PNI [OR:0.942; 95%CI: 0.901-0.984; p =0.008] were independent predictor of high-grade ICT burden.

Conclusion: The mGPS is novel predictor of high-grade ICT burden in anterior STEMI patients. Compared to other immuno-inflammatory based grading indices, mGPS is superior scoring method in showing ICT burden and may be used for risk grouping in various coronary artery diseases.

Interventional Cardiology / Coronary

PB-038

Characteristics of Patients with Acute Myocardial Infarction In The Covid-19 Pandemic: A Single Center Experience

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Background and Aim: This study aims to determine the demographic, clinical and angiographic characteristics of patients with acute miyocardial infarction (AMI) and coronavirus (COVID-19) and compare these findings with AMI patients without COVID-19.

Methods: The study was designed as a single center retrospective study. The patients who had been hospitalized due to acute myocardial infarction to our hospital were included. Patients with COVID-19 and AMI were included in Group 1 (n=33) and Group 2 consisted of the rest of the patients (n= 100). In these two groups, demographic, clinical presentation, angiography findings and prognosis were compared.

Results: The average age of Group 1 and Group 2 were 65.9 years and 59.75 respectively(p>0.05).Five patients (15.2%) in Group 1 and 26 patients (26%) in Group 2 were presented with STEMI (p > 0.05).The heart rate and Tp-e / QTc ratio were higher and corrected QT and Tpeak-end were longer in Group 1 (p = 0.000; 0.014; 0.000, respectively).There was no statistical difference between the groups in terms of baseline Thrombolisis in myocardial infarctus (TIMI) flow, thrombus stage, final myocardial blush, frequency of using thrombus aspiration catheter, stent thrombosis, and final TIMI flow (p > 0.05).These two groups were also similar in terms of hospital mortality and the composite end point in the first month (p > 0.05).

Conclusion: The main findings of our study suggest that AMI patients with COVID-19 do not have a significantly increased risk of mortality compared to those with AMI without COVID-19.In addition to that, this study shows that clinical and angiographic characteristics are similar.

Table 1. Characteristics of Both Groups

	COVID-MI (Group 1, n=33)	Controls (Group 2, n=100)	P
Age (year)	65.9 (49.5-80)	59.75 (48-65)	0.47
Sex	Male; 21 (63.6%)	Male; 74 (74%)	0.253
COVID-19 test results	11 (33.3%)	46 (%46)	0.229
Entubation	4 (12.1%)	5 (5%)	0.158
Lenght of stay in intensive care unit	4 (2.5-5)	2 (2-3)	0.000
Lenght of stay in hospital	6 (4.5-11)	4 (3-5)	0.001
Ischemic heart disease	PCI:17 (24.8%)	PCI:21 (21%)	0.001
	CABG:5 (15.2%)	CABG:6 (6%)	0.098
Diabetes mellitus	16 (48.5%)	33 (3%)	0.11
Hypertension	22 (24.8%)	44 (44%)	0.024
Chronic kidney disease	0 (0%)	3 (3%)	0.574
Cerebrovascular Event	1 (3)	1 (1%)	0.436

CABG: Coronary artery bypass graft; CT: Computed tomography; PCI: Percutaneous coronary intervention

Table 2. Coronary Angiography Results

Parameters	Group 1	Group 2	*P*
Type of MI	NSTEMI:28 (84.8%)	NSTEMI:74 (74%)	0.201
	STEMI:5 (15.2%)	STEMI:26 (26%)	
LMCA	3 (9.1%)	5 (5%)	0.391
LAD	20 (60.6%)	60 (60%)	0.951
CX	18 (54.5%)	46 (46%)	0.394
RCA	13 (39.4%)	47 (7%)	0.446
IM	1 (3.1%)	5 (5%)	1
Medical	10 (30.3%)	24 (24%)	0.094
CABG	6 (18.2%)	7 (7%)	0.094
PCI	17 (51.5)	69 (69%)	0.094
Multivessel thrombosis	1 (3%)	0 (0%)	0.248

CABG: Coronary artery bypass grafting; CX: Circumflex artery; IM: Intermedial artery; MI: Myocardial infarctus; NSTEMI: non ST elevation myocardial infarctus; LAD: Left anterior descending artery; LMCA: Left mean coronary artery; PCI: Percutaneous coronary intervention; RCA: Right coronary artery; STEMI: ST elevation myocardial infarctus

Table 3. Characteristics of Percutaneouse Coronary Intervention

Parameters	Group 1 (n=17)	Group 2 (n=68)	*P*
COVID PCR	(+): 13 (76.4%)		
(-): 4 (23.6%)			
Baseline TIMI			0.772
0	9 (52.9%)	27 (39.7%)	
1	0 (0%)	1 (1.5%)	
2	1(5.9%)	6 (8.8)	
3	7 (41.2%)	34 (50%)	
Baseline thrombus grade			0.682
0	8 (47.1%)	24 (35.3%)	
1	0 (0%)	9 (1.2%)	
2	1 (5.9%)	5 (7.4%)	
3	2 (11.8%)	8 (11.8%) %	
4	2 (11.8%)	5 (7.4%) %	
5	4(23.5%)	17(25%)	
Post-PCI TIMI			1
0	1 (5.9%)	3 (4.4%)	
1	0 (0%)	0 (0%)	
2	1 (5.9%)	4 (5.9%)	
3	15 (88.2%)	61 (89.7%)	
Myocardial Blush grade (end)			0.829
0	1 (5.9%)	5(7.4%)	
1	0 (0) %	0 (0) %	
2	0 (0) %	4 (5.9) %	
3	16 (94.1) %	59 (86.8) %	
Multivessel PCI	0 (0) %	5 (7.2) %	0.578
Thrombus aspirating catheter using	1 (5.9) %	2 (2.9) %	0.488
Stent Thrombosis	1(5.9%)	2(2.9%)	0.488

CABG: Coronary artery bypass grafting; CX: Circumflex artery; IM: Intermedial artery; MI: Myocardial infarctus; LAD: Left anterior descending artery; LMCA: Left mean coronary artery; PCI: Percutaneous coronary intervention; RCA: Right coronary artery; TIMI: Thrombolysis in Myocardial Infarction

Table 4. Characteristics of Both Groups

	COVID-MI (Group 1, n=33)	Controls (Group 2, n=100)	*P*
Age(year)	65.9 (49.5-80)	59.75 (48-65)	0.47
Sex	Male; 21 (63.6%)	Male; 74 (74%)	0.253
COVID-19 test results	11 (33.3%)	46 (46%)	0.229
Entubation	4 (12.1%)	5 (5%)	0.158
Lenght of stay in intensive care unit	4 (2.5-5)	2 (2-3)	0.000
Lenght of stay in hospital	6 (4.5-11)	4 (3-5)	0.001
Ischemic heart disease	PCI:17 (24.8%)	PCI: 21 (21%)	0.001
	CABG:5 (15.2%)	CABG: 6 (6%)	0.098
Diabetes mellitus	16 (48.5%)	33 (33%)	0.11
Hypertension	22 (24.8%)	44 (44%)	0.024
Chronic kidney disease	0 (0%)	3 (3%)	0.574
Cerebrovascular Event	1(3%)	1 (1%)	0.436

CABG: Coronary artery bypass graft; CT: Computed tomography; PCI: Percutaneous coronary intervention

Table 5. Electrocardiography and Transthoracic Echocardiography Test Results

Parameters	Group 1	Group 2	P
Ejection Fraction (%)	49.18 (45-55)	51.53 (45-60)	0.158
Spab (mmHg)	25.5 (25-26.5)	36 (26.5-47)	0.05
Heart rate (beat/minute)	92.42±18.64	74.38±13.45	0.000
Sinus Rhythm	30 (90.9%)	96 (96%)	0.256
Atrial fibrillation	3 (9.1%)	4 (4%)	0.256
LBBB	0	0	
RBBB	0 (0%)	1 (1%)	
PR duration (msn)	147.27±27.56	157.45±25.18	0.096
QT duration (msn)	370.64±39.37	392.25±38.3	0.014
cQT duration (msn)	453.76±34.61	434.67±35.87	0.014
QRS-T angle (degree)	22 (19-22)	41.5 (38-45)	0.887
Tpeak-Tend duration (msn)	85 (82.5-87.5)	75 (60-90)	0.000
Tpe/QTc	0.2 (0.17-0.25)	0.17(13-17)	0.000
In hospital mortality	3 (9.1%)	2 (2%)	0.097
First Month of composite endpoint	5 (15.2%)	6 (6%)	0.098

cQT: corrected QT duration; LBBB: Left bundle branch block; mmHg: milimeter mercury; msn: milisecond; RBBB: Right bundle branch block sPAB: systolic pulmonary artery pressure;

Interventional Cardiology / Coronary

PB-039

The Effect of Loading Dose of Rosuvastatin on Peri-procedural Myocardial Infarction

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Background and Aim: High-dose statin therapy before percutaneous coronary intervention (PCI) is thought to reduce the occurrence of peri-procedural myocardial infarction (PPMI), which is known to be associated with increased mortality and prolonged hospitalization, especially in statin naïve patients. The aim of this study is to investigate the effect of rosuvastatin loading dose on PPMI and major adverse cardiac and cerebrovascular events (MACCE) in patients undergoing elective PCI according to their statin use.

Methods: One hundred sixty-five patients were included in the study and divided into two groups as patients already on statin treatment (n:126) and statin naive patients (n:39). Both groups were randomly assigned to high-dose (40 mg) rosuvastatin (n:86) or control group (n:79). The primary endpoint was the incidence of periprocedural myocardial infarction, and the secondary endpoint was MACCE.

Results: The mean age of study population was 59±9.4 years. Most of them (n:127, 77%) were male. Median follow-up time was 368 day. There were 30 patients (19 in high dose statin group, and 11 in control group) diagnosed as PPMI after PCI. Loading dose of statin therapy was not effective to prevent occurrence of PPMI. A positive correlation between PPMI and creatinine (r:0.199, *P*:0.011), lesion complexity (r:0.189, p:0.015) and negative correlation between GFR and PPMI (r: -0.158 *P*=0.043) was remarkable.

Conclusion: Pre-procedural administration of high dose rosuvastatin in patients with stable coronary artery disease has failed to decrease PPMI independent of chronic statin use.

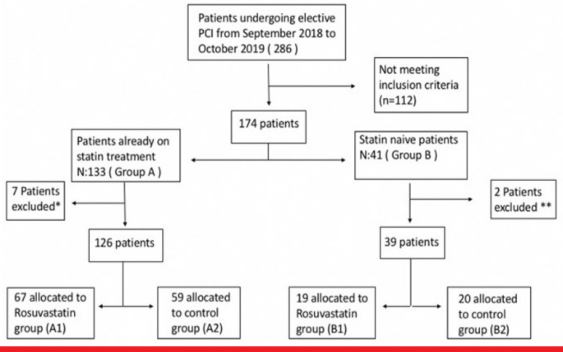

Figure 1. Flow chart of the study.
***:5 had coronary angiography alone, 2 were referred to CABG **:1 had coronary angiography alone, 1 were referred to CABG**

Table 1. Angiographic features of patients with and without periprocedural MI

Variables	All (n=165)	Without Periprocedural MI (n=135)	Periprocedural MI (n=30)	*P*
Lesion Type				
- Simple (A, B1) (n,%)	88 (53.3%)	78 (57.8 %)	10 (33.3 %)	0.015
- Complex (B2, C) (n,%)*	77 (46.7%)	57 (42.2 %)	20 (66.7 %)	
Stent length (mm, median)	22 (5)	22 (4)	22 (4.5)	0.674
Stent diameter (mm, median)	2.8 (0.25)	2.8 (0.3)	2.8 (0.3)	0.879

*ACC/AHA classification of coronary lesions

Table 2. The relationship between rosuvastatin loading and periprocedural MI in statin naive patients and statin users

Variables	All (n=165)	Without Periprocedural MI (n=135)	Periprocedural MI (n=30)	*P*
Treatment Groups				
A1 (n, %)	67 (40.6%)	53 (39.3 %)	14 (46.7 %)	0.436
A2 (n, %)	59 (35.8%)	52 (38.5 %)	7 (23.3 %)	
B1 (n, %)	19 (11.5%)	14 (10.4 %)	5 (16.7 %)	
B2 (n, %)	20 (12.1%)	16 (11.9 %)	4 (13.3 %)	

A1: statin users with rosuvastatin loading, A2: statin users without rosuvastatin loading, B1: statin naïve patients with rosuvastatin loading, B2: statin naïve patients without rosuvastatin loading

Table 3. Correlations

Variables	Correlation	*P*
Periprocedural MI-Creatinine	0.199	0.011*
Periprocedural MI-Gfr	-0.158	0.043*
Periprocedural MI-Lesion (simple/complex)	0.189	0.015*
Creatinine-Gfr	-0.767	<0.001**

p<0.05 is significant

Interventional Cardiology / Coronary

PB-040

The Evaluation of Lesion Localization and Ischemic Myocardial Mass via Arrhythmia Parameters in Stable Coronary Artery Patients with Myocardial Ischemia

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Background and Aim: In this study we aimed to evaluate the arrhythmogenic potential of the critical stenoses in stable coronary artery disease according to the distribution of the involved coronary artery and also whether magnitude of myocardial mass with ischemia or the diseased coronary artery were more closely associated with arrhythmia by means of arrhythmia predictors measured on ECG recordings.

Methods: We scanned 183 patients who underwent percutaneous coronary intervention for single vessel stable coronary artery disease between January 2013 and 2016.Hundred and fifty five patients of 183 patients met study inclusion criteria and were enrolled. This retrospective study was performed by collecting data from hospital records. Assined indicators

of increased arrhythmogenic risk as TpTe interval, QTc interval and TpTe/QT ratio were calculated on ECGs before and after the percutaneous coronary interventions. We calculated the anatomical myocardial area at risk of ischemia as a percentage of the LV myocardium volume for a given site of lesion with APPROACH score (Table 1) by assessing coronary angiography images.

Results: Percutaneous coronary interventions were performed for isolated LAD lesions in 60 patients, for isolated Cx lesions in 54 patients and for isolated RCA lesion in 41 patients. The percentage of left ventricular jeopardized myocardium in LAD lesions (34±10) was significantly higher than those in Cx (17±11) and RCA (21±5) lesions (*P*<0,001). When compared with Cx or RCA lesions group, preoperative TpTe interval, QTc interval and TpTe/QT ratio were significantly higher in LAD lesions group (*P*<0,001) (Table2). The differences between TpTe intervals and TpTe/QT ratios measured before and after the percutaneous coronary intervention (PCI) of were significantly higher in patients with LAD lesions than patients with Cx and RCA lesions (*P*<0,001) (Table3).

Conclusion: In patients with stable coronary artery disease, arrhythmia predictors calculated on ECG recordings were found to be higher in patients with critical LAD lesions and also it was seen that critical LAD lesions could be more arrhythmogenic than other lesions. We have found that the size of the ischemic myocardium was associated with increased arrhythmogenesis. Furthermore, patients with LAD lesions get the greatest benefit from revascularization in terms of arrhythmia predictors calculated on ECG.

Külprit Lezyon	Hedef Arter Yan Dalları ⇨ ⇩		LAD için Diyagonal, Diğerleri için Posterolateral		
			Küçük veya Yok	Orta	Büyük
LAD (Sağ veya Sol Dominant)		Distal	13,75	14,80	15,90
		Mid	27,50	29,70	31,80
		Proksimal	41,25	44,50	47,75
Proksimal Cx (Sağ Dominant)	OM	Küçük veya Yok	9,25	12,50	15,75
		Orta	15,25	18,50	21,75
		Büyük	21,25	24,50	27,75
Proksimal Cx (Sol Dominant)	PDA	Küçük veya Yok	23,50	28,00	32,50
		Orta	29,50	34,00	38,50
		Büyük	35,50	40,00	44,50
Mid Cx (Sol Dominant) RCA (Sağ Dominant)	PDA	Küçük veya Yok	9,25	12,50	15,75
		Orta	15,25	18,50	21,75
		Büyük	21,25	24,50	27,75
Mid Cx (Sağ Dominant)			3,25	6,50	9,75

LAD: Sol ön inen arter, Cx: Sirkumfleks arter, RCA: Sağ koroner arter, PDA: Posterior Desending Arter OM:Obtus Marjinalis

Table 1. Approach Score
Percentage of ischemic myocardium caused by target lesion

		β Katsayısı	p
LAD	Preoperatif TpTe intervali	0,845	<0,001
	Preoperatif QTc intervali	0,456	<0,001
	Preoperatif TpTe/QT oranı	0,751	<0,001
Cx	Preoperatif TpTe intervali	0,135	0,3
	Preoperatif QTc intervali	0,055	0,7
	Preoperatif TpTe/QT oranı	0,073	0,6
RCA	Preoperatif TpTe intervali	0,165	0,3
	Preoperatif QTc intervali	0,236	0,1
	Preoperatif TpTe/QT oranı	0,252	0,1

β:Pearson Korelasyon Katsayısı; LAD: Sol ön inen arter, Cx: Sirkumfleks arter, RCA: Sağ koroner arter

Table 2. Correlation analysis of arrhythmia predictors with percentage of ischemic myocardial tissue

		β Katsayısı	p
LAD	Δ TpTe (sn)	0,81	<0,001
	Δ QTc (sn)	0,07	0,6
	Δ TpTe/QT	0,83	<0,001
Cx	Δ TpTe (sn)	0,08	0,54
	Δ QTc (sn)	0,14	0,31
	Δ TpTe/QT	0,09	0,49
RCA	Δ TpTe (sn)	0,01	0,96
	Δ QTc (sn)	0,03	0,85
	Δ TpTe/QT	0,04	0,83

β: Spearman Korelasyon Katsayısı; LAD: Sol ön inen arter, Cx: Sirkumfleks arter, RCA: Sağ koroner arter,ΔTpTe: Revaskülarizasyon öncesi ve sonrası Tp-Te intervalleri farkı, ΔQT: Revaskülarizasyon öncesi ve sonrası QT intervalleri farkı, ΔTpTe/QT:Revaskülarizasyon öncesi ve sonrası Tp-Te/QT oranları farkı

Table 3. Correlation analysis of percentage of ischemic myocardium and arrhythmia predictors before and after revascularization

Hypertension

PB-041

Blood pressure variability and diastolic function in middle-aged normotensive individuals

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Background and Aim: Association of diastolic dysfunction and hypertension is well established. Blood pressure variability (BPV), a non-conventional blood pressure parameter, has also been shown to contribute hypertensive target organ damage independent of absolute blood pressure levels but it is rarely assessed and its association with diastolic dysfunction is unknown. The present study investigates the association of BPV and diastolic function in middle-aged normotensive individuals.

Methods: 264 normotensive patients aged between 45 and 65 were enrolled into the study. 24-hour ambulatory blood pressure monitoring were performed and BPV was defined

as the standard deviation of systolic blood pressure measurements. Patients were divided into 3 groups according to BPV tertiles. Echocardiographic and tissue doppler diastolic function parameters were compared among the groups.

Results: Mean age of the patients were 50.41 and similar among groups along with gender, hyperlipidemia and blood biochemical values. Left ventricular ejection fraction, wall thicknesses, left ventricular diameters, left ventricular mass index, deceleration time, tricuspid regurgitation velocity, RV diameters and tricuspid annular plane systolic excursion were similar among groups. Mitral inflow E/A (Tertile 1 vs 2 vs 3: 1.10[0.33] vs 1.05 [0.22 vs 1.02 [0.30], p =0.02) and average tissue doppler mitral annular E′ velocity (12 [2] vs 10.5 [1.85] vs 10 [1.55], p =0.02) were highest in the tertile 1 and lowest in the tertile 3. Average E/E′ (Tertile 1 vs 2 vs 3: 7.2 [2.2] vs 8.1 [3.2] vs 9.3 [2.9], p<0.001) was lowest in the tertile 1 and highest in the tertile 3 (Table 1). In addition, there was a positive correlation between BPV and Average E/E′ (Rs =0.401, p<0.001). In contrary, E/A (Rs =- 0.286, p<0.001) and average E′ (Rs =- 0.451, p<0.001) were negatively correlated with BPV (Figure 1).

Conclusion: Blood pressure variability is positively correlated with average E/E′ and negatively correlated with E/A and average E′. Further studies are required to elucidate the relationship of BPV and diastolic functions.

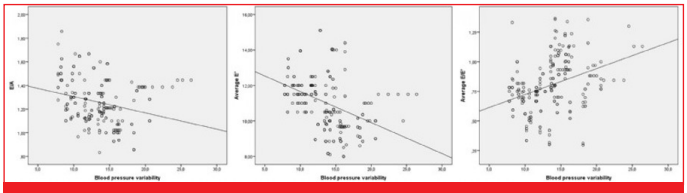

Figure 1. Correlation of blood pressure variability with diastolic parameters

Table 1. Echocardiographic measurements according to blood pressure variability tertiles

	Blood Pressure Variability			
Variables	**Tertile1 (n=88)**	**Tertile2 (n=88)**	**Tertile3 (n=88)**	***P***
Left atrium (mm)	30 (5)	31 (2)	32 (6)	0.422
IV Septum (mm)	9 (2)	9 (2)	9 (2)	0.998
Posterior Wall (mm)	9 (2)	9 (2)	9 (2)	0.986
LVEDD (mm)	42 (4)	43 (3)	43 (5)	0.842
LVESD (mm)	26 (5)	27 (4)	27 (4)	0.874
LVEF (%)	64 (4)	65 (5)	65 (6)	0.752
LVMI	72 (20)	74 (13)	75 (18)	0.685
E/A	1.10 (0.33)	1.05 (0.22)	1.02 (0.30)	0.02
IVRT	94 (12)	97 (10)	99 (10)	0.054
DT	175 (32)	181 (36)	178 (36)	0.453
Average E′	12 (2)	10.5 (1.85)	10 (1.55)	0.02
Average E/E′	7.2 (2.2)	8.1 (3.2)	9.3 (2.9)	<0.001
TRV (m/s)	2.1 (0.6)	2.2 (0.5)	2.2 (0.5)	0.683
RV diameter (mm)	29 (5)	29 (4)	29(7)	0.838
TAPSE	22 (6)	23 (5)	22 (5)	0.888

Hypertension

PB-042

Effect of acute blood pressure reduction on frontal QRS-T angle

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Background and Aim: Hypertension plays an important role in the development of cardiovascular diseases, and an important cause of morbidity. Hypertension causes left ventricular hypertrophy, arterial stiffness, and loss of elasticity leading to diastolic dysfunction as target organ damage. Electrocardiography (ECG) is easy to apply and accessible, inexpensive and practical, and is an indispensable imaging method in cardiological emergency services. The calculation of the frontal QRS-T angle can be measured automatically with digital ECG devices. The frontal QRS-T angle provides information about depolarization and repolarization of the myocardium. An increase in the frontal QRS-T angle is associated with the development of atrial fibrillation and malignant arrhythmia and is a poor prognostic marker. Here we aimed to investigate the effect of acute blood pressure reduction on frontal QRS-T angle beside echocardiographic changes.

Methods: We prospectively enrolled 40 consecutive patients (age: 56.7±8.5 years, 42.5% male) admitted to our emergency department with a hypertensive attack. All patients underwent baseline standard electrocardiographic and echocardiographic evaluation. Patients with systolic dysfunction, left ventricular hypertrophy, acute coronary syndrome and any rhythym other than sinus (pace rhythym or bundle brach block) and patients with poor echogeneicity were excluded. Frontal QRS-T angle could be recorded from automatically analyze digital ECG outputs. This angle calculated as absolute differences of QRS and T angle (QRS axis-T axis). QTc and Tp-e intervals were measured (Figure1). All measurements were repeated after achieving target blood pressure reduction and analyzed.

Results: Of the patients participating in the study, 27.5% had diabetes, 60% had hypertension, and 22.5% had coronary artery disease. In the study, no significant difference was found between baseline and control group after acute blood pressure reduction in terms of frontal QRS-T angle (42 (0-162) vs. 40 (1-154), respectively, p =0.768). With acute blood pressure reduction QTc min and Tp-e interval was significantly decreased (358.5±18.3, p=0.035 and 42.1±2.9, p =0.032, respectively), while QTc dispersion was significantly increased (14.1±7.2, p =0.048). Heart rate and left ventricular ejection fraction was significantly increased from baseline values.

Conclusion: Acute blood pressure reduction had no effect on frontal QRS-T angle which gives information about left ventricular depolarization and repolarization activity.

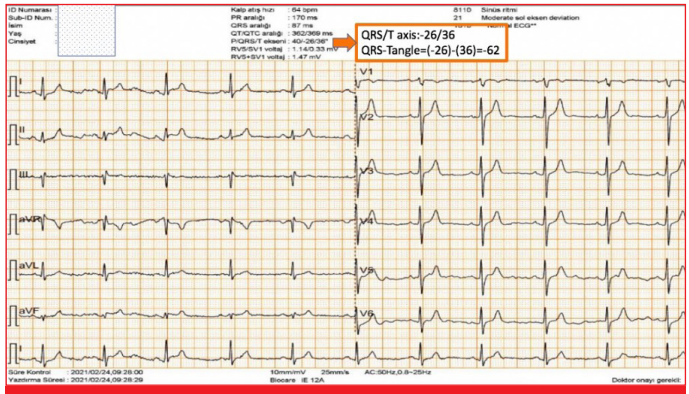

Figure 1. Calculation of QRS-T angle from automatically analyzed surface electrocardiography

Table 1. Characteristics of study participants

Age (year) (mean±SD)	56.7±8.5
Male gender (n, %)	17 (42.5)
Height (cm) (mean±SD)	165.1±7.3
Weight (kg) (mean±SD)	79.0±10.3
BMI (kg/m2) (mean±SD)	28.4±3.5
BSA (m2) (mean±SD)	1.86±0.9
Serum Creatinine (mg/dl)	0.84±0.11
Hypertension (n, %)	24 (60.0)
Diabetes Mellitus (n, %)	11(27.5)
Coronary artery disease (n, %)	9 (22.5)
Smoker (n, %)	6 (15.0)
Duration of Hypertension (year), median (min-max)	1.0 (0.0-23.0)
CHADSVASC score, median (min-max)	2.0 (0.0-4.0)
Antihypertensive drug usage (n, %)	23 (57.5)
Number of drug (median) (min-max)	1(0-3)
None	17 (42.5)
1	7 (17.5)
2	5 (12.5)
3	5 (12.5)

SD, Standard Deviation; BM, Body Mass Index; BSA, Body Surface Area; CHA2DS2-VASc: Congestive heart failure, Hypertension: Age ≥75 years, Diabetes mellitus, Prior Stroke or TIA or Thromboembolism, Vascular disease, Age 65-74 years, Sex; AF: Atrial fibrillation.

Table 2. Acute effects of blood pressure reduction on echocardiographic parameters

	Pre-BP reduction	Post-BP reduction	P
SBP (mm Hg)	181.2±14.9	151.8±13.7	<0.001
DBP (mm Hg)	98.4±17.1	84.5±15.3	<0.001
Pulse Pressure (mm Hg)	82.8±15.0	67.3±10.2	<0.001
LV EDD (cm)	44.3±3.4	43.6±3.6	0.065
LV ESD (cm)	25.8±4.4	23.1±4.1	0.029
Septum (mm)	10.2±1.2	10.4±1.1	0.054
Posterior wall (mm)	10.0±1.2	10.0±1.1	0.558
LV mass	153.6±32.6	152.5±36.2	0.088
LVEF (%)	62.3±8.5 <0.001	69.7±7.6	<0.001
E (cm/s)	80.5±19.9 <0.001	68.5±17.7	<0.001
A (cm/s)	88.8±18.6	90.0 ±18.6	0.418
E/A ratio	0.95±0.33	0.71±0.25	<0.001
E/e' average	9.2±2.3	8.5±1.9	0.028
Heart rate (bpm)	88.5±14.23	92.5±16.3	<0.001

SBP, systolic blood pressure; DBP, diastolic blood pressure; LV EDD, Left ventricular end-diastolic diameter; LV ESD, Left ventricular end-systolic diameter; bpm, beat per minute

Table 3. Effect of acute blood pressure reduction on electrocardiographic parameters

	Pre-BP reduction	Post-BP reduction	P
QTc max (ms)	382 (354-448)	380 (344-434)	0.245
QTc min (ms)	368.4±22.1	358.5±18.3	0.035
QTc dispertion (ms)	12.2±6.6	14.1±7.2	0.048
Tp-e interval (ms)	44.3±3.4	42.1±2.9	0.032
QRS duration (ms)	92 (78-122)	93 (74-120)	0.234
Tp-e/QTc	0.19± 0.02	0.18±0.02	0.353
Frontal QRS-T angle	42 (0-162)	40 (1-154)	0.768

BP, blood pressure

Hypertension

PB-043

COVID-19 in Patients Using RAAS Inhibitors for Hypertension: Evaluating Factors Associated with Lung Involvement, Intensive Care Unit Admission and Mortality

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Background and Aim: We investigated the possible relationships between RAAS inhibitor use (for hypertension) and COVID-19-related characteristics such as lung involvement on CT, admission to intensive care unit, and mortality.

Methods: This case-control study was conducted in a single-center with a retrospective and observational design. We included 220 patients from a total of 654 patients admitted with a confirmed diagnosis of COVID-19 infection (by RT-PCR) and who underwent simultaneous Thoracic CT scan. We divided the patients into three groups: patients without a history of hypertension or drug use as the non-HT group (n=74), patients with HT using RAAS inhibitors (ACEIs and ARBs) in the RAAS group (n=72), and patients with HT using CCBs in the CCB group.

Results: Of the 220 patients, 117 (53.2%) were males and no difference was observed between the groups in term of sex. The overall median age of patients was 63 (21-93) years and the RAAS group was found to have significantly higher mean age, while the non-HT and RAAS groups were similar (66 [24-93] vs. 61 [32-89], 62 [21-88], p= 0.040). No significant difference was observed between the groups in terms of in-hospital mortality, 1-month mortality, mechanical ventilation need, lung involvement and other radiological findings. Albumin (OR=0.250, 95%CI: 0.091-0.690, p = 0.007), WBC (OR=1.000, 95%CI: 1.000-1.000, p = 0.004), platelet count (OR=0.987, 95%CI: 0.979-0.995, p = 0.002), CRP (OR=1.007, 95%CI: 1.001-1.014, p = 0.025) and male gender (OR=3.893, 95%CI: 1.598-9.480, p = 0.003) were determined as factors that were independently associated with ICU admission among hospitalized patients. Also, age (OR=1.095, 95%CI: 1.005–1.194, p = 0.038) and length of stay in ward (OR=0.809, 95%CI: 0.694–0.943, p = 0.007) were found to be independently associated with mortality in patients admitted to the ICU.

Conclusion: In our study, we showed that patients using RAAS inhibitors for HT, those using CCB, and individuals without a history of HT or other chronic diseases did not differ in terms of lung involvement on CT. We also found that HT presence was not associated with ICU admission and death. Albumin level, CRP level, platelet count, WBC count and male gender are independent predictors of ICU admission; also found that age and length of ward stay days were independent predictors of death in patients hospitalized in the ICU.

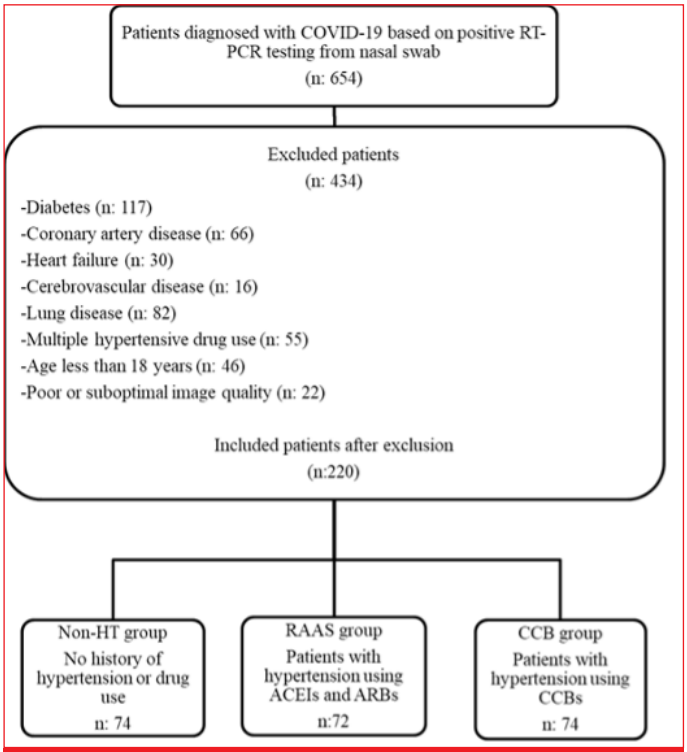

Figure 1. Flow chart of this study.
RT-PCR: Reverse Transcriptase-Polymerase Chain Reaction, ACEIs: Angiotensin-converting enzyme inhibitors, ARBs: Angiotensin-receptor blockers, CCBs: Calcium channel blockers.

Table 1. Baseline demographic and clinical characteristics

	All patients n= 220	Non-HT group n= 74	RAAS group n= 72	CCB group n= 74	*P* value
Male gender	117 (53.2%)	39 (52.7%)	37 (51.4%)	41 (55.4%)	0.884
Age, years	63 (21-93)	61 (32-89)	66 (24-93)	62 (21-88)	0.040**
Smoking status (using)	21 (9.5%)	11 (14.9%)	8 (11.1%)	2 (2.7%)	0.036***
Hospitalization	183 (83.2%)	61 (82.4%)	62 (86.1%)	60 (81.1%)	0.703
Length of stay in ward (days)	6 (0-36)	7 (0-20)	6 (0-36)	7 (0-33)	0.950
ICU admission	48 (21.8%)	9 (12.2%)	18 (25%)	21 (28.4%)	0.042*
Length of stay in ICU (days)	0 (0-20)	0 (0-15)	0 (0-16)	0 (0-20)	0.052
Mechanical ventilation	22 (10%)	3 (4.1%)	11 (15.3%)	8 (10.8%)	0.075
Length of stay in mechanical ventilation (days)	0 (0-8)	0 (0-7)	0 (0-4)	0 (0-8)	0.103
Length of symptomatic days	8 (1-40)	6.5 (1-22)	9 (2-40)	7.5 (1-38)	0.065
In-hospital mortality	30 (13.6%)	5 (6.8%)	13 (18.1%)	12 (16.2%)	0.101
1-month mortality	6 (2.7%)	2 (2.7%)	3 (4.2%)	1 (1.4%)	0.580
Hydroxychloroquine	158 (71.8%)	55 (74.3%)	49 (68.1%)	54 (73%)	0.676
Favipiravir	135 (61.4%)	42 (56.8%)	43 (59.7%)	50 (67.6%)	0.378
Tocilizumab	33 (15%)	8 (10.8%)	10 (13.9%)	15 (20.3%)	0.259
Convalescent plasma treatment	11 (5%)	0 (0%)	4 (5.6%)	7 (9.5%)	0.030*
Pulse steroid therapy	50 (22.7%)	12 (16.2%)	16 (22.2%)	22 (29.7%)	0.145
Low dose steroid therapy	59 (26.8%)	16 (21.6%)	27 (37.5%)	16 (21.6%)	0.045**

Medians are presented as minimum-maximum. ICU: intensive care unit *: The difference between non-HT group and others **: The difference between RAAS group and others ***: The difference between CCB group and others

Table 2. Laboratory findings

	All patients n= 220	Non-HT group n= 74	RAAS group n= 72	CCB group n= 74	P value
Glucose (mg/dL)	116 (64-306)	109 (64-306)	118 (82-266)	126 (77-226)	0.001*
Urea (mg/dL)	35 (14-250)	32.5 (14-185)	40 (16-250)	35 (15-208)	0.038**
Creatinine (mg/dL)	0.9 (0.3-8.0)	0.8 (0.3-8.0)	0.9 (0.5-5.2)	0.9 (0.3-5.5)	0.017***
Albumin (g/dL)	3.9 (2.2-4.9)	4.1 (2.9-4.7)	3.8 (2.7-4.9)	4 (2.2-4.9)	0.001***
ALT (U/L)	22 (4-125)	23 (6-122)	20 (6-125)	23 (4-114)	0.553
AST (U/L)	29 (3-216)	28 (11-99)	28 (9-215)	30 (3-216)	0.370
Direct Bilirubin (mg/dL)	0.1 (0-0.8)	0.1 (0-0.5)	0.1 (0.1-0.7)	0.1 (0.1-0.8)	0.300
Sodium (mmol/L)	137 (125-149)	137 (127-144)	137 (125-148)	135 (126-149)	0.246
Potassium (mmol/L)	4.0 (2.6-5.7)	4.1 (3.2-5.5)	4.0 (2.8-5.7)	4.0 (2.6-5.6)	0.925
WBC (x10^9/L)	5.7 (1.8-23.0)	5.2 (2.4-12.4)	6.1 (1.8-23.0)	6.3 (2.7-19.2)	0.001*
Neutrophils (x10^9/L)	3.6 (1.0-20.9)	3.1 (1.1-10.5)	4.1 (1.0-20.9)	4.1 (1.2-17.8)	<0.001*
Lymphocytes (x10^9/L)	1.2 (0.1-5.4)	1.2 (0.1-5.4)	1.2 (0,2-3.4)	1.2 (0.2-3.6)	0.847
Monocytes (x10^9/L)	0.4 (0,0-1.6)	0.4 (0.1-1.1)	0.4 (0.1-1.1)	0.5 (0.0-1.6)	0.742
Eosinophils (x10^9/L)	0.0 (0.0-0.5)	0.0 (0-0.5)	0.0 (0-0.2)	0.0 (0.0-0.2)	0.112
Hemoglobin (g/dl)	13.5 (8.4-19)	13.6 (10.3-17)	13.3 (8.4-19)	13.5 (8.6-16.8)	0.157
MCV (fL)	84 (63-105)	84 (63-97)	84 (68-105)	83 (64-98)	0.405
Platelet count (x10^9/L)	196 (86-556)	196 (86-459)	188 (91-556)	198 (106-387)	0.827
CRP (mg/L)	30.3 (0.4-350)	18.1 (0.7-184)	46.1 (0.4-350)	46.2 (1.4-322)	0.001*
Troponin I (ng/L)	10 (0-590)	10 (0-590)	10 (0-430)	10 (0-280)	0.710
D-dimer (µg/mL)	0.7 (0.1-10.5)	0.6 (0.1-5.3)	0.7 (0.1-8.4)	0.7 (0.1-10.5)	0.090

Medians are presented as minimum-maximum. ALT: alanine transaminase, AST: aspartate transaminase, WBC: white blood cells, MCV: mean corpuscular volume, CRP: C-reactive protein. *: The difference between non-HT group and others **: The difference between RAAS group and others ***: The difference between non-HT group and RAAS group

Table 3. Radiological findings

	All patients n= 220	Non-HT group n=74	RAAS group n= 72	CCB group n= 74	P
Lung involvement	153 (69.5%)	49 (66.2%)	55 (76.4%)	49 (66.2%)	0.306
Number of involved lung lobes	4.5 (0-5)	2 (0-5)	5 (0-5)	5 (0-5)	0.197
Right upper lobe	131 (59.5%)	38 (51.4%)	48 (66.7%)	45 (60.8%)	0.163
Right medial lobe	120 (54.5%)	33 (44.6%)	46 (63.9%)	41 (55.4%)	0,064
Right lower lobe	126 (57.3%)	38 (51.4%)	48 (66.7%)	40 (54.1%)	0.137
Left upper lobe	129 (58.6%)	40 (54.1%)	45 (62.5%)	44 (59.5%)	0.576
Left lower lobe	131 (59.5%)	39 (52.7%)	49 (68.1%)	43 (58.1%)	0.160
Ground glass opacification	155 (70.5%)	49 (66.2%)	56 (77.8%)	50 (67.6%)	0.248
Consolidation	62 (28.2%)	20 (27%)	25 (34.7%)	17 (23%)	0.278
Reticular	27 (12.3%)	8 (10.8%)	9 (12.5%)	10 (13.5%)	0.880
Honeycomb	10 (4.5%)	6 (8.1%)	2 (2.8%)	2 (2.7%)	0.196
Subpleural	155 (70.5%)	49 (66.2%)	56 (77.8%)	50 (67.6%)	0.248
Central	47 (21.4%)	15 (20.3%)	18 (25%)	14 (18.9%)	0.643
Diffuse	14 (6.4%)	4 (5.4%)	5 (6.9%)	5 (6.8%)	0.917

Medians are presented as minimum-maximum.

Table 4. Demographic, clinical and laboratory characteristics of patients according to lung involvement

	Patients with lung involvement n:153	Patients without lung involvement n: 67	*P* value
Male gender	88 (57.5%)	29 (43.3%)	0.052
Age, years	64 (32-93)	61 (21-84)	0.051
Hypertension	104 (68%)	42 (62.7%)	0.445
ACEIs/ARBs use	55 (35.9%)	17 (25.4%)	0.124
CCBs use	49 (32%)	25 (37.3%)	0.445
Smoking status (using)	14 (9.2%)	7 (10.4%)	0.763
Glucose (mg/dL)	121 (64-306)	109 (82-266)	<0.001
Urea (mg/dL)	36 (15-250)	31 (14-115)	0.018
Creatinine (mg/dL)	0.9 (0.3-8)	0.8 (0.5-4.4)	0.016
Albumin (g/dL)	3.9 (2.2-4.9)	4.1 (2.9-4.9)	<0.001
ALT (U/L)	24 (4-125)	20 (6-64)	0.025
AST (U/L)	31 (3-216)	25 (9-95)	<0.001
Direct Bilirubin (mg/dL)	0.1 (0.1-0.7)	0.1 (0-0.8)	0.012
Sodium (mmol/L)	136 (125-149)	137 (128-142)	0.197
Potassium (mmol/L)	4.0 (2.6-5.7)	4.1 (3.1-5.6)	0.067
WBC (x10^9/L)	5.6 (1.8-23.0)	6.1 (2.4-13.4)	0.698
Neutrophils (x10^9/L)	3.5 (1.0-20.9)	3.6 (1.2-10.7)	0.339
Lymphocytes (x10^9/L)	1.1 (0.1-5.4)	1.4 (0.3-3.4)	0.005
Monocytes (x10^9/L)	0.4 (0.1-1.1)	0.5 (0.0-1.6)	0.001
Eosinophils (x10^9/L)	0.0 (0-0.5)	0 (0-0.3)	<0.001
Hemoglobin (g/dL)	13.4 (8.4-17.3)	13.5 (9.9-19)	0.240
MCV (fL)	84 (63-105)	84 (64-97)	0.259
Platelet count (x10^9/L)	188 (86-556)	207 (98-349)	0.035

Medians are presented as minimum-maximum. ACEIs: Angiotensin-converting enzyme inhibitors, ARBs: Angiotensin-receptor blockers, CCB: calcium channel blocker, ALT: alanine transaminase, AST: aspartate transaminase, WBC: white blood cells, MCV: mean corpuscular volume.

Table 5. Variables independently associated with ICU admittance in hospitalized patients (n = 183) (multivariable logistic regression)

	Odds Ratio	95% CI for Odds Ratio	*P* value
Albumin (g/dL)	0.250	0.091-0.690	0.007
WBC (x10^9/L)	1.000	1.000-1.000	0.004
Platelet count (x10^9/L)	0.987	0.979-0.995	0.002
CRP (mg/L)	1.007	1.001-1.014	0.025
Male gender	3.893	1.598-9.480	0.003

ICU: intensive care unit, CI: Confidence interval, WBC: white blood cells, CRP: C-reactive protein.

Table 6. Variables independently associated with death in patients admitted to the ICU (n = 48) (multivariable logistic regression)

	Odds Ratio	95% CI for Odds Ratio	*P* value
Age, years	1.095	1.005-1.194	0.038
Length of stay in ward (days)	0.809	0.694-0.943	0.007

ICU: intensive care unit, CI: Confidence interval.

Hypertension

PB-044

Determinants of reverse dipping blood pressure in normotensive, non-diabetic population with an office measurement below 130/85 mm Hg

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Background and Aim: The role of dipping blood pressure pattern in normotensives is unclear. The study aims to search the circadian blood pressure rhythm and the clinical determinants related to reverse dipping pattern in a strictly selected, normotensive population.

Methods: The study population was divided into three groups depending on the nocturnal dipping pattern as dipping, non-dipping, and reverse dipping. Basal clinical characteristics, anthropometric measurements, and spot urine samples from the first-morning void were collected. Clinical determinants related to the presence of reverse dipping pattern were tested by the Multiple Binary Logistic Regression analysis.

Results: A total of 233 participants were involved in the study population (median age 45 years [40-50]). Dipping pattern was detected in 55.4%, non-dipping pattern in 33.0%, and reverse dipping pattern in 11.6% of the study population. There was no difference between the groups in terms of basal clinical features. Albumin-to-creatinine ratio (ACR) (p<0.001) and hs-CRP levels (p=0.006) were also statistically significant across the groups. ACR (HR: 1.195, 95% CI: 1.067-1.338, p=0.002) and hs-CRP (HR: 2.438, 95% CI: 1.023-5.808, p=0.044) were found to be related to the presence of reverse dipping blood pressure pattern.

Conclusions: The absence of nocturnal physiological dipping is seen at a remarkable rate in the normotensive Turkish population. ACR and hs-CRP are the clinical determinants related to the presence of reverse dipping blood pressure pattern.

Table 1.

	Hazard ratio	95% Confidence interval	*P* value
ACR (mg/g)	1.195	1.067-1.338	0.002
hs-CRP (mg/L)	2.438	1.023-5.808	0.044
Age (years)	1.029	0.967-1.095	0.363
Waist circumference (cm)	1.032	0.965-1.104	0.352
BMI (kg/m^2)	0.932	0.790-1.100	0.407
Office systolic BP (mm Hg)	0.989	0.933-1.048	0.699

Multiple binary logistic regression analysis for the presence of reverse dipping blood pressure pattern.

Table 2.

	Hazard ratio	95% Confidence interval	*P* value
ACR (mg/g)	1.195	1.067-1.338	0.002
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Multiple binary logistic regression analysis for the presence of reverse dipping blood pressure pattern.

Hypertension

PB-045

Relationship of adverse childhood experiences with essential hypertension

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Background and Aim: It is known that the mental and physical effects of adverse experiences in childhood continue not only in childhood but also in adulthood. It is thought that the physical effects are caused by the psychobiological effects of symptoms such as anxiety, depression, stress, anger, sleep disorders on inflammatory processes. Therefore, it is clear that research is needed to understand the physical and mental impact of adverse childhood experiences. However, there are limited studies investigating the relationship between psychosocial processes and diseases that are very common in clinical practice, such as hypertension. In the present study, it was aimed to compare the adverse childhood experiences of essential hypertension (HT) patients with healthy controls.

Methods: The sample of the current study consists of 200 participants, including 100 HT patients who applied to Kırıkkale Yüksek İhtisas Hospital Cardiology outpatient clinic and 100 healthy controls who applied to the health committee. HT was defined as office systolic blood pressure values ≥140 mmHg and/or diastolic blood pressure values ≥ 90 mmHg in accordance with the European Society of Cardiology guideline. Participants over the age of 65 with increased risk of physical and psychiatric diseases and those with additional comorbidities were excluded from the study. The sociodemographic data form and Adverse Childhood Experiences Scale (ACEs) were applied to the participants who volunteered to participate in the study after obtaining their consent. After the obtained data were scored according to the scale directive, they were subjected to appropriate statistical processing.

Results: The mean age of the participants included in the study was 51.55±8.90 years and 78.5% were women. There was no statistically significant difference in age, gender, marital status, income level, height and weight variables between the healthy control group and HT patients (p>0.05, Table 1). Body Mass Index (t=-2.558, df=198, p=0.011) and ACEs (t=-2.609, df=198, 0.010, Figure 1) scores of HT group were found to be statistically significantly higher than healthy controls (Table 1).

Conclusions: The present study shows that patients with HT are exposed to more adverse childhood experiences compared to healthy volunteers. This result supports studies showing that psychosocial factors may have an effect on the pathogenesis of HT. When evaluated together with the literature, it is noteworthy that adverse childhood experiences and related psychological consequences play a potential role in HT. In this context, referral of patients for psychiatric evaluation in HT patient visits may be effective in elucidating and treating the etiology. Randomized studies with larger numbers of patients are needed to prove this. In addition, it should not be forgotten that preventing adverse experiences in childhood and early intervention can prevent future physical problems.

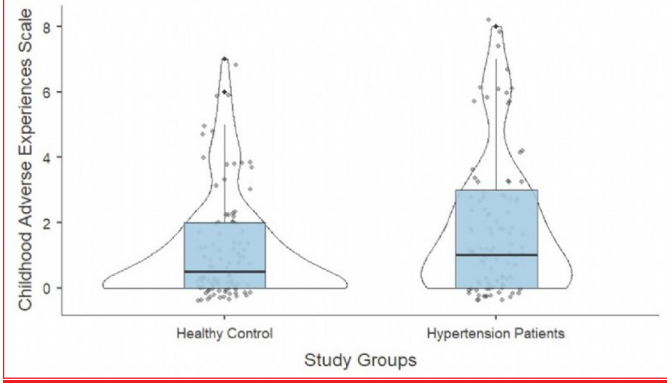

Figure 1. Comparison of Study Groups' Childhood Adverse Experiences Scale.

Table 1. Comparison of the participants' sociodemographic characteristics and childhood adverse experiences among the study groups

Variable	Healthy Control (n=100)	Hypertension Patients (n=100)	t/χ^2	df	p
Age; year, Mean±SD	51.72±8.43	51.38±9.38	0.270	198	0.788
Gender; n			0.267	1	0.606
Female	77	80			
Male	23	20			
Marital Status; n			0.112	2	0.945
Single	8	9			
Married	73	71			
Other	19	20			
Income Status; n			2.929	2	0.231
Low	6	7			
Middle	35	46			
High	59	47			
Length; cm, Mean±SD	168.32±7.49	168.32±8.09	1.568	198	0.118
Weight; kg, Mean±SD	74.00±16.29	76.84±14.67	-1.295	198	0.197
Body Mass Index, kg/m^2, Mean±SD	25.96±4.55	27.57±4.37	-2.558	198	0.011*
Childhood Adverse Experiences; Mean±SD	1.15±1.62	1.84±2.08	-2.609	198	0.010*

*: p≤0.05

Hypertension

PB-046

Morning blood pressure surge is associated with elevated TNF-α levels in normotensive subjects

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Background and Aim: Hypertension is the leading cause of death worldwide. Blood pressure has a circadian rhythm in healthy individuals with a decrease during sleep and elevation in the early hours of the morning. The magnitude of morning blood pressure surge (MBPS) is associated with end-organ damage and cerebrovascular events. We aimed to investigate the relationship between MBPS and inflammatory cytokine tumor necrosis factor alpha (TNF-α) in normotensive subjects.

Methods: We enrolled one hundred thirty-seven patients admitted cardiology policlinic with elevated blood pressure levels outside the hospital. These subjects were also normotensive in three blood pressure recordings. Ambulatory blood pressure monitoring was ordered for suspicion of grade 1 hypertension without target organ damage. Ambulatory blood pressure recordings were also normotensive. Sleep-trough MBPS was calculated for all subjects; the average of three recorded values centered on the lowest nocturnal systolic blood pressure is subtracted from the average systolic blood pressure over 2 hours after awakening. Routine blood chemistry and TNF-α levels were measured early in the morning. Subjects were divided into two groups according to mean sleep-trough MBPS. Subjects with an MBPS equal to or lower than 18.55 mmHg were classified in the low MBPS group and higher than were classified as high MBPS group. Low MBPS group was comprised of 80 subjects (mean age: 37.65±10.75 years, 46 females (57.5%), high MBPS group was comprised of 57 subjects (mean age: 43.19±11.68 years, 31 females (54.4%)).

Results: Mean age body mass index and diastolic blood pressure were higher in the high MBPS group than the low group (43.19±11.68 vs. 37.65±10.75 years, p=0.005, 27.47±4.18 vs. 25.59±4.01 kg/m2, p=0.009 and 82.24±9.13 vs. 78.84±7.7, p= 0.02). All other variables were similar. Tumor necrosis factor alpha levels of the high MBPS group were higher than the low MBPS group. Tumor necrosis factor alpha levels were correlated with the MBPS levels (r= 0.279, p=0.002). In linear regression analysis, MBPS was independently associated with the TNF-α levels (β=0.243 95%CI: 0.105–0.732, p=0.009).

Conclusions: In our study, we have shown that sleep-trough MBPS was correlated with TNF-α levels. Our finding emphasizes the relationship of inflammation with MBPS. This finding may add information on the role of MBPS on target organ damage and vascular events.

	MBPS low group, n=80	MBPS high group, n=57	p
Age, years	37.65 ± 10.75	43.19 ± 11.67	0.005
Gender, female, n, (%)	46 (57.5%)	31 (54.4%)	0.730
Body mass index, kg/m^2	25.58 ± 4.01	27.46 ± 4.17	0.009
24 hours systolic BP, mmHg	120.49 ± 9.69	122.88 ± 12.45	0.228
24 hours diastolic BP, mmHg	78.84 ± 7.7	82.24 ± 9.13	0.02
Total cholesterol, mg/dl	191.48 ± 36.94	200.29 ± 39.56	0.188
HDL-cholesterol, mg/dl	47.6 ± 11.78	50.82 ± 12.56	0.132
LDL-cholesterol, mg/dl	116.83 ± 32.19	124.75 ± 35.95	0.178
Triglycerides, mg/dl	115 (37 - 477)	110 (32 - 429)	0.917
Left ventricular mass, gr	155.29 ± 38.08	164.96 ± 42.19	0.163
Left ventricular mass index, gr/m^2	82.72 ± 23.24	87.64 ± 24.81	0.237
Tumor necrosis factor -α, pg/ml	5.20 (0- 26.55)	7 (0 – 165)	0.05

BP: Blood pressure, MBPS: Morning blood pressure surge. Students' t test, Mann Whitney U tests were used.

Table 1. Comparison of MBPS groups

	r	p
Age, years	0.168	0.05
Body mass index, kg/m2	0.094	0.272
24 hours systolic blood pressure, mmHg	0.308	<0.001
24 hours diastolic blood pressure, mmHg	0.212	0.013
Total cholesterol, mg/dl	0.078	0.371
HDL-cholesterol, mg/dl	-0.044	0.617
LDL-cholesterol, mg/dl	0.093	0.282
Triglycerides, mg/dl	-0.107	0.213
Left ventricular mass index, gr/m2	0.193	0.240
Sleep-trough MBPS, mmHg	0.279	0.002

MBPS: Morning blood pressure surge.Pearson and Spearman correlation tests were used.

Table 2. Correlation analysis of TNF-α

	B	β	95%CI for B	p
Age, years	0.074	0.052	(-0.194- 0.342)	0.586
Body mass index, kg/m^2	0.014	0.004	(-0.778 - 0.806)	0.972
24 hours systolic BP, mmHg	0.418	0.269	(-0.137- 0.973)	0.139
24 hours diastolic BP, mmHg	-0.177	-0.09	(-0.88- 0.526)	0.619
Sleep-trough MBPS, mmHg	0.418	0.243	(0.105- 0.732)	0.009

BP: Blood pressure. R^2=0.123. Linear regression analysis was used

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Table 3. Linear regreesion analysis of TNF-α

Cardiovascular Surgery

PB-047

Effects of surgical atrial septal defect closure operation on the frontal QRS-T angle

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Background and Aim: A new marker has been found for ventricular repolarization and depolarization heterogeneity, which is the frontal plane QRS-T [f(QRS-T)] angle between the directions of ventricular depolarization (QRS axis) and repolarization (T axis). Adverse cardiac outcomes can be detected based on observation of the f(QRS-T) angle. In atrial septal defect (ASD) patients, surgical closure of ASD is safer and more effective than its percutaneous closure with higher popularity in the last two decades. Nevertheless, we have scarce information about the impact of ASD closure on cardiac autonomic function and heart repolarization. The aim of the study is to investigate the potential effect of ASD operation on the f(QRS-T) angle and cardiac repolarization parameters.

Methods: In our retrospective study design we sampled a total of 24 patients who underwent ASD closure surgery operation between December 2011 and January 2019. Preoperative and postoperative 6-week ECG parameters including the f(QRS-T) of the patients were compared.

Results: When pre- and postoperative ECG parameters were compared, no statistically significant difference was found in QT interval (p=0.079), adjusted QT interval (p=0.079), Tpe interval (p=0.150) and Tpe/QTc ratio (p=0.696). An improvement was found in QRS duration (p=0.035), p wave duration (p<0.001) and f(QRS)-T angle (p<0.001).

Conclusions: In our study, it was observed that surgical ASD closure operation was associated with heart repolarization parameters and improvement in f(QRS-T).

Table 1. Demographic, echocardiographic and surgical characteristics of the patients

Variables	(n=30)
Age	35.98±8.07
Female n (%)	15 (50.0%)
Body Mass Index, kg/m^2	26.91±3.71
Diabetes Mellitus, n (%)	3 (10.0%)
Hypertension, n (%)	3 (10.0%)
Smoking, n (%)	10 (33.3%)
Left ventricular ejection fraction (%)	63.35±3.75
Left ventricular end diastolic diameter (cm)	4.2±0.4
Left ventricular end systolic diameter (cm)	2.9±0.2
Interventricular septum thickness (mm)	9.0±0.4
Posterior wall thickness (mm)	8.4±0.3
Left atrium diameter (cm)	3.9±1.0
Systolic pulmonary artery pressure, mmHg	29.8±7.1
Atrial septal defect diameter	1.7±0.9
Primary repair	20 (66.6%)
Repair by patch	10 (33.3%)

Data are given as mean ± standard deviation or percentage [n (%)].

Table 2. Electrocardiographic and echocardiographic features of the groups

Variables	Before surgery	6 weeks after surgery	*p* value
QRS, ms	110.3±22.3	100.1±15.3	0.035
QT, ms	367.5±31.2	356.3±30.6	0.079
QTc, ms	407.2±23.9	394.9±29.0	0.077
Tpe, ms	81.1±17.7	80.2±9.2	0.150
Tpe/QTc	0.20±0.04	0.20±0.04	0.696
f(QRS)-T (°)	76.1±30.5	65.1±14.3	<0.001
P wave, ms	134±24.7	121±21.3	<0.001
Left ventricular ejection fraction (%)	63.35±3.75	62.21±5.33	0.786
Left ventricular end diastolic diameter (cm)	4.2±0.4	4.2±0.5	0.931
Left ventricular end systolic diameter (cm)	2.9±0.2	2.9±0.6	0.876
Systolic pulmonary artery pressure, mmHg	29.8±7.1	27.6±5.1	0.652

Data are given as mean ± SD, n or median (interquartile range). QTc - corrected QT interval; f(QRS)-T; frontal QRS-T angle

Heart Valve Diseases

PB-048

Impact of Tp-e interval, Tp-e/QT and Tp-e/QTc ratios on mortality after surgical aortic valve replacement for severe aortic valve stenosis

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Background and Aim: Aortic stenosis (AS) is the most common degenerative valvular heart disease that can affect left ventricular functions. Tp-e interval and Tp-e/QT ratio is a novel repolarization marker which is associated with adverse cardiovascular events in several cardiovascular diseases. In our study, our aim is to investigate the prognostic effect of Tp-e interval, Tp-e/QT and Tp-e/QTc ratios on mortality in patients who underwent successful surgical aortic valve replacement (AVR)

Methods: A total of three hundred seventy-five patients undergoing successful surgical AVR were included in this study. Then, patients were divided into two groups according to mortality as group 1 without mortality (342 patients) and group 2 with mortality (33 patients). Tp-e interval, Tp-e/QT and Tp-e/QTc ratios were calculated for both groups.

Results: Tp-e interval [71 (63.7-77); 86 (84-88), p<0.001], Tp-e/QT ratio [0.19 (0.17-0.20); 0.23 (0.22-0.23), p<0.001] and Tp-e/QTc ratio (0.17±0.02; 0.21±0.01, p<0.001) were higher in group 2 compared to group 1. In multivariate logistic regression analyses Tp-e interval [OR: 1.315, 95%CI: 1.203-1.437, p<0.001], Tp-e/QT ratio [OR: 7.334, 95%CI: 3.274-11.643, p<0.001] and Tp-e/QTc ratio [OR: 2.567, 95%CI: 1.605-4.106, p<0.001] were found to be independent predictors of mortality. Additionally, a Kaplan-Meier survival analysis also revealed that long term survival was found to be significantly decreased in patients with higher Tp-e/QT ratio (Log-Rank p<0.001) and Tp-e/QTc ratio (Log-Rank p<0.001).

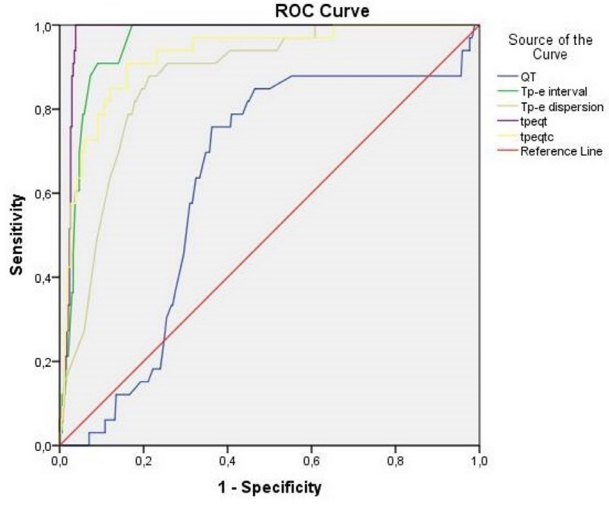

Figure 1: Receiver-operating characteristic curves indicating the discriminative ability of the QT interval, Tp-e interval, Tp-e dispersion, Tp-e/QT ratio and Tp-e/QTc ratio for mortality.

Conclusions: Tp-e interval, Tp-e dispersion, Tp-e/QT and Tp-e/QTc ratios are associated with worse prognosis after surgical AVR in patients with severe AS. All of them are also independent predictors of mortality.

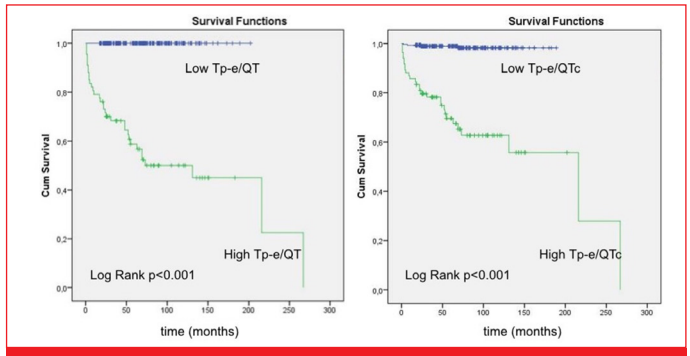

Figure 2: Kaplan-Meier survival curves for low and high Tp-e/QT groups. B- Kaplan-Meier survival curves for low and high Tp-e/QTc groups.

Table 1. Demographic and clinical variables of study population

	Mortality (-) (n=342)	Mortality (+) (n=33)	*P*-value
Age, years	62±12	71±11	<0.001
Gender (female), n (%)	91 (26.6)	15 (45.5)	0.022
Dyspnea (NYHA III-IV), n (%)	95 (27.8)	9 (27.3)	0.951
Angina, n (%)	175 (51.2)	20 (60.6)	0.300
Syncope, n (%)	34 (9.9)	5 (15.2)	0.249
Smoking, n (%)	95 (25.3)	7 (21.2)	0.418
Diabetes mellitus, n (%)	73 (21.3)	8 (24.2)	0.699
Hypertension, n (%)	166 (48.5)	13 (39.4)	0.315
Coronary artery disease, n (%)	1 (0.3)	1 (3.0)	0.168
Atrial fibrillation, n (%)	27 (7.9)	4 (12.1)	0.285
Congestive heart failure, n (%)	53 (15.5)	9 (27.3)	0.082
Medication			
Beta blocker, n (%)	272 (79.5)	22 (66.7)	0.086
ACEi or ARB, n (%)	149 (43.6)	6 (18.2)	0.005
CCB, n (%)	47 (13.7)	4 (12.1)	0.524
Statin, n (%)	75 (21.9)	6 (1.6)	0.617
Hemoglobin, g/dL	13.4 (11.9-14.8)	11.9 (10.4-13.6)	0.004
Creatinine, mg/dL	0.87 (0.7-1.0)	1.0 (0.8-1.23)	0.004
Total cholesterol, mg/dL	178 (150-213)	172 (155-220)	0.922
LDL-C, mg/dL	108.5 (81-133)	101 (81-121)	0.692
HDL-C, mg/dL	2 (35-50)	48 (33-58)	0.227
Triglyceride, mg/dL	124 (92-179)	140 (86-178)	0.560
Valve type			
Mechanical prosthesis, n (%)	249 (72.8)	14 (42.4)	<0.001
Bioprosthesis, n (%)	93 (27.2)	19 (57.6)	
Follow-up duration, months	69±40	41±60	<0.001

Data are presented as percentage, mean standard deviation or median (interquartile range). NYHA, New York Heart Association; ACEi, angiotensin converting enzyme inhibitors; ARB, angiotensin receptor blockers; CCB, calcium channel blockers; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol.

Table 2. Echocardiographic and electrocardiographic variables of whole study group

	Mortality (-) (n=342)	Mortality (+) (n=33)	*P*-value
Ejection fraction, %	60 (55-65)	55 (50-60)	0.002
LVEDD, mm	50 (47-57)	50 (44-57)	0.319
LVESD, mm	32.5 (28-39)	33 (27-42)	0.826
LA diameter, mm	40 (36-43)	42 (40-45)	0.028
Maximum aortic gradient, mm Hg	76 (65-90)	74 (65-89)	0.718
Mean aortic gradient, mm Hg	13 (10-18)	15 (10-21)	0.164
AVA, cm^2	0.70 (0.60-0.80)	0.70 (0.60-0.70)	0.234
Heart rate, bpm	72 (68-77)	71 (66-75)	0.843
QRS duration, ms	85.92±6.29	85.91±6.53	0.996
QT interval, ms	368 (362-384)	379 (376-394)	0.013
QTc interval, ms	409 (390-429)	413 (398-438	0.138
Tp-e interval, ms	71 (63.7-77)	86 (84-88)	<0.001
Tp-e dispersion, ms	15.3 (12.2-19.0)	27 (25-29)	<0.001
Tp-e/QT	0.19 (0.17-0.20)	0.23 (0.22-0.23)	<0.001
Tp-e/QTc	0.17±0.02	0.21±0.01	<0.001

Data are presented as percentage, mean standard deviation or median (interquartile range). LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LA, left atrium; AVA, aortic valve area.

Table 3. Multivariate logistic regression analyses giving information about independent predictors of mortality

	OR	95% CI	*P*-value
Model 1			
Age	1.050	1.002-1.102	0.043
Ejection fraction	0.938	0.890-0.989	0.018
Tp-e	1.315	1.203-1.437	<0.001
Model 2			
Age	1.057	1.016-1.100	0.006
Gender (female)	2.750	1.038-7.283	0.042
Creatinine	2.378	1.244-4.543	0.009
Tp-e dispersion	1.273	1.173-1.383	<0.001
Model 3			
Tp-e/QT	7.334	3.274-11.643	<0.001
Model 4			
Hemoglobin	0.751	0.586-0.963	0.024
Ejection fraction	0.950	0.905-0.997	0.038
Tp-e/QTc	2.567	1.605-4.106	<0.001

OR: odds ratio; CI: confidence interval.

Heart Failure

PB-050

Clinical characteristics and short-term clinical outcomes in patients with acute heart failure and anemia who admitted to the emergency department

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Background and Aim: Anemia is a highly prevalent comorbidity in heart failure (HF) and its etiology is usually multifactorial. Most evidence of prognostic impact and treatment of anemia are from chronic heart failure. However, little is known about prevalence, clinical characteristics and outcomes of anemia in acute heart failure (AHF). This study aimed to describe differences of demographic and clinical features and outcomes of patients admitted to emergency department (ED) for AHF between anemic and non-anemic groups.

Methods: This prospective, single-center study included 719 consecutive AHF patients who admitted to the ED for AHF, between October 01, 2015 and September 30, 2016. Patients were classified by hemoglobin (Hb) level. Anemic group were defined by Hb level <13 g/dl in male and <12 g/dl in female on admission. We compared clinical characteristics and outcomes between these two groups. Short-term outcome was defined as death from any cause, heart failure related hospitalization and admission to the ED at 90-day and 180-day for HF.

Results: Of 719 enrolled patients (mean age 72±10 years, 54.9% male, mean Hb level 12.09±2.17 g/dl) mean Htc level 37.01±6.53%), 415 patients (57.7%) had anemia (mean Hb level 10.63±1.33 g/dl, mean Hct 32.84±4.10%). There was no difference between those with and without anemia in terms of gender and age (for all, p>0.05). The rate of presence of peripheral edema was higher in the anemic group (73.3% vs. 64.5%; p=0.012). Patients with anemia had more prevalence of atrial fibrillation/flutter (21.9% vs. 15.1%; p=0.022), diabetes (39.5% vs. 31.6%; p=0.029), chronic kidney disease stage 3-5 (23.1% vs. 7.6%; p<0.001), compared to those without anemia. There was no difference between the two groups in presence of hypertension (74.0% vs. 68.8%; p=0.124) and coronary artery disease (42.4% vs. 42.4%; p=0.995). There was no significant difference between the two groups in terms of in-hospital clinical outcomes including length of ED stay, length of intensive care unit (ICU) stay, length of total hospital stay and in-hospital death (p>0.05, for all). The 90-day HF related hospitalization were significantly higher in the anemic group (p=0.019). However, there were no significant differences in 90 and 180-day mortality rate between both groups (p>0.05, for all). (Table 1)

Conclusions: Anemia is very common in patients with AHF and associated with worse clinical picture, atrial fibrillation, diabetes mellitus and chronic kidney disease. Although there is no significant difference in terms of in-hospital clinical outcomes, short-term HF re-hospitalization is higher in AHF patients with anemia.

Table 1

	AHF with anemia (n=415)	AHF without anemia (n=304)	*p* value
Length of ED stay, min, [IQR]	260.0 [165.0-344.3]	242.5 [155.0-321.3]	0.177
Length of hospital stay, days, [IQR]	6 [4-11]	6 [4-9]	0.164
In-hospital death, n (%)	31 (7.5)	29 (9.5)	0.322
90-day ED readmissions, n (%)	169 (40.7)	118 (38.8)	0.606
90-day HF related hospitalization, n (%)	182 (43.9)	107 (35.2)	0.019
90-day death, n (%)	69 (16.6)	44 (14.5)	0.433
NT-proBNP, pg/ml	11658 [4718-23351]	6833 [3500-17292]	0.025
LVEF, %	30 [19-45]	30 [18-45]	0.882
180-day ED readmissions, n (%)	232 (61.1)	157 (57.7)	0.392
180-day HF related hospitalization, n (%)	230 (60.8)	151 (55.5)	0.173
180-day death, n (%)	90 (21.7)	55 (18.1)	0235

Heart Failure

PB-051

The prevalence of hyperkalemia in patients with acute heart failure admitted to emergency department and its relationship with in-hospital outcomes

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Background and Aim: Hyperkalemia is increasingly prevalent and life-threatening condition in the heart failure (HF). Furthermore, little data are yet available on the interaction between dyskalemia and in-hospital outcome in patients with acute heart failure (AHF). In this study, we analyzed the prevalence of hyperkalemia and its effect on in-hospital outcomes in real life clinical practice in patients with AHF admitted to emergency department (ED).

Methods: This prospective, single-center study included 719 consecutive AHF patients who presented to the ED with AHF symptoms and signs. Patients' characteristics, potassium level, eGFR data and outcomes (length of hospital stay, length of ED stay, in-hospital death) has been examined. Hyperkalemia was defined as a serum potassium level >5 mEq/L and renal dysfunction was defined as an eGFR level <60 mL/dk.

Results: A total of 719 patients [median age 73(66.0-80.0) years, 54.9% male] were enrolled. In study population, median potassium level was 4.79 mEq/L [4.30-5.20 IQR], median creatinine level was 1.26 mg/dL [0.95-1.69 IQR] and median eGFR was 50.34 [33.86-71.07] mL/min. 287 (39.9%) were receiving RAAS inhibitors. Overall, 62.7% of patients (n=451) had normal serum potassium levels (≥3.5–5 mEq/l) and 33.7% of patients (n=242) were found to have hyperkalemia (>5 mEq/L) and, hypokalemia (<3.5 mEq/L) was found in 3.6% of patients (n=26). In patients with eGFR <60 ml/min, hyperkalemia was much more prevalent than those with eGFR ≥60 ml/min (37.5% and 26.7% respectively, p=0.003). In-hospital outcomes are summarized in Table 1.

Conclusions: Hyperkalemia was present in one third of the patients who presented to the ED due to AHF, and patients with renal dysfunction were particularly at high risk for hyperkalemia. Interestingly, the potassium value at admission to the ED was not associated with in-hospital outcomes.

Table 1. In-hospital outcome of patients with AHF according to potassium value

	AHF with hyperkalemia (n=242)	AHF with hypo/normokalemia (n=477)	*p* value
Length of ED stay, min	261.5 [175.0-350.3]	249.5 [157.8-330.0]	0.261
Length of hospital stay, days	6 [4-9]	6 [4-11]	0.433
In-hospital death, n (%)	23 (9.5%)	37 (7.8%)	0.423
90-day ED readmissions, n (%)	87 (40.7%)	200 (45.8%)	0.217
90-day HF related hospitalization, n (%)	78 (36.4%)	211 (48.5%)	0.004
90-day death, n (%)	40 (16.5%)	73 (15.3%)	0.670

Heart Failure

PB-052

Systemic Immune-Inflammation Index Predicting All-Cause Mortality in Patients with Advanced Heart Failure

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Background and Aim: Systemic inflammation plays a critical role in the prognosis of patients with chronic heart failure. Platelet counts and NLR ratio (SII, platelet count × neutrophil/lymphocyte ratio) were used to calculate the systemic immune-inflammation index (SII), so the patients' inflammatory and immune status were simultaneously considered. Yet, there is no information in the literature on the prognostic value of SII in patients with advanced heart failure.

Methods: This is a retrospective, single-center study. In our study, we included a total of 183 patients who were being followed up in our hospital between October 2015 and December 2020 with a diagnosis of advanced heart failure. The patients were divided into two groups according to their

baseline SII scores (high SII vs low SII) and they were compared according to their demographical and clinical characteristics. All-cause mortality was considered as an endpoint.

Results: SII cut-off value for long-term all-cause mortality was found to be 751 in patients with advanced heart failure (AUC 0.747 [95% CI 0.651–0.843]) (Figure 1). The body mass index of patients with low SII (≤751) was found to be higher [29.3±4.8; 27.2±5.2 p=0.006] when compared to patients with high SII (>751), and anemia was found to be significantly higher in the high SII (>751) group than in the low SII (≤751) group [27 (23.3%); 29 (43.3%) p=0.005]. No significant difference was found between the groups regarding demographical and clinical characteristics (Table 1). The all-cause mortality rate was reported to be significantly higher in the high SII score (>751) group than in the low SII score (≤751) group [8(6.9%), 25 (37.3%) p=<0.001] (Table 1). Multivariate analyses showed that New York Heart Association (NYHA) class IV (HR: 2.36, %95 CI: 1.15-4.81 p=0.02), anemia (HR: 2.11, %95 CI: 1.05-4.23 p=0.03), and SII>751 (HR: 7.31 %95 CI: 3.24-16.48 p<0.001) were predictors for long-term all-cause mortality (Table 2).

Conclusions: Inflammation has been linked to a variety of diseases, such as chronic heart failure, cancer, metabolic disorders, and cardiovascular diseases. In observational clinical studies, SII was primarily indicated to be a significant tool that can be used in predicting the survival rates in multiple solid cancers. Seo et al. demonstrated the predictive value of SII in chronic heart failure patients. In their study, patients with high SII (>551) score was found to have a significantly increased risk of cardiac events. In our study, we examined the long-term prognostic value of SII in patients with advanced heart failure. As a result, a high SII (>751) score was found to be the independent predictor of long-term mortality in patients with advanced heart failure. As far as we know, our study is the first study in the literature investigating the prognostic value of SII in patients with advanced heart failure.

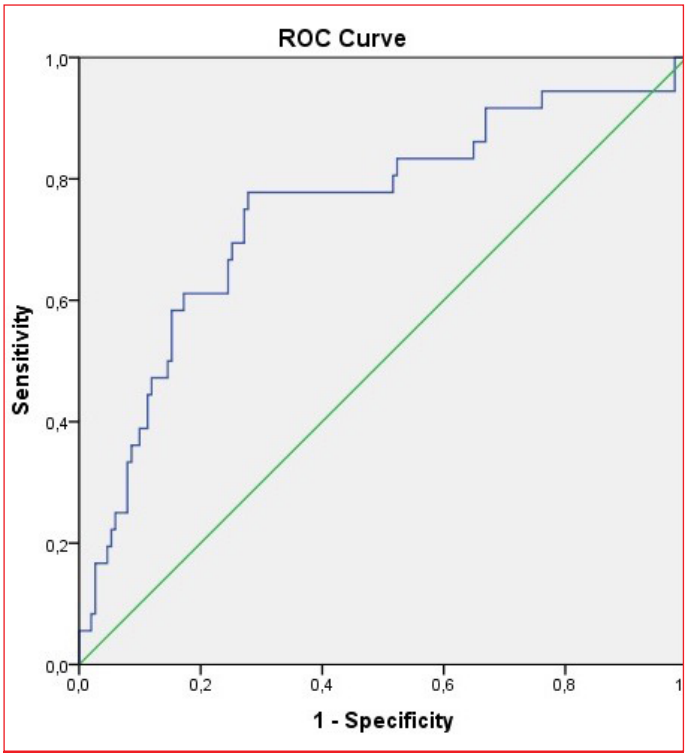

Figure 1. The ROC curve of Systemic Immune-Inflammation Index for long-term all-cause mortality in patients with advanced heart failure

Table 1

	SII≤751 n=116 (63,4%)	SII>751 n=67 (36,6%)	*p*
Age (years), $\overline{X}$ ±(SD)	49.7±9.1	48.7±9.6	0.47
Female, n (%)	25 (21.6%)	7 (10.4%)	0.06
BMI, $\overline{X}$ ±(SD)	29.3±4.8	27.2±5.2	0.006
Ischemic cardiomyopathy, n (%)	78 (67.2%)	48 (71.6%)	0.53
Atrial fibrillation, n (%)	16 (12%)	12 (18.8%)	0.46
DM, n (%)	43 (37.1%)	24 (35.8%)	0.86
Hypertension, n (%)	56 (43.8%)	24 (35.8%)	0.10
COPD, n (%)	8 (6.9%)	8 (11.9%)	0.25
Current Smoking, n (%)	14 (12.1%)	7 (10.4%)	0.74
CRF, n (%)	14 (12.1%)	11 (16.4%)	0.40
Anemia, n (%)	27 (23.3%)	29 (43.3%)	0.005
ICD, n (%)	50 (43.1%)	27 (40.3%)	0.71
LVEF, $\overline{X}$ ±(SD)	25.3±5.9	25.2±6.8	0.93
NYHA, Class IV, n (%)	46 (39.7%)	33 (49.3%)	0.20
Beta bloker, n (%)	106 (91.4%)	59 (88%)	0.82
ACEi/ARBi, n (%)	102 (87.9%)	61 (91%)	0.63
Aldosterone antagonists, n (%)	94 (81%)	55 (82.1%)	0.49
Aspirin, n (%)	58 (50%)	40 (59.7%)	0.20
Statins, n (%)	52 (44.8%)	31 (46.3%)	0.68
LDL (mg/gL), $\overline{X}$ ±(SD)	106.9±43.5	94.2±46.9	0.09
Hemoglobin, g/dL $\overline{X}$ ±(SD)	13.9±1.8	13.2±2.1	0.01
Follow-up time (months), X±(SD)	29.5±17.9	21.4±15.7	0.002
All cause mortality, n (%)	8 (6.9%)	25(37.3%)	<0.001

Comparison of demographic, clinical and laboratory characteristics of patients according to Systemic Immune-Inflammation Index ACE: Angiotensin-converting enzyme, ARB: Angiotensin II receptor blockers, BMI: Body mass index, COPD: Chronic obstructive pulmonary disease, CRF: Chronic renal failure, DM: Diabetes mellitus, ICD: Implantable cardioverter defibrillator, LDL: low-density lipoprotein, LV EF: left ventricle ejection fraction, NYHA: New York Heart Association, SII: Systemic Immune-Inflammation Index, SD: standard deviation, $\overline{X}$: mean

Table 2

	Univariate analysis			Multivariate analysis		
	HR	95% CI	*p*	HR	95% CI	*p*
NYHA Class IV	2.50	1.23–5.10	0.011	2.36	1.15–4.81	0.02
Anemia	2.66	1.34–5.28	0.005	2.11	1.05–4.23	0.03
SII> 751	8.20	3.67–18.30	<0.001	7.31	3.24–16.48	<0.001

Multivariate lojistic regression analysis givinig independent predictors of mortality. CI: confidence interval, HR: Hazard ratio, SII: Systemic Immune-Inflammation Index, NYHA: New York Heart Association

Heart Failure

PB-053

Diurnal Blood Pressure Change in Patients with Ischemic and Non-Ischemic Cardiomyopathy

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Background and Aim: Arterial blood pressure shows a circadian rhythm during the day and at night. A decrease of 10-20% in blood pressure at night compared to daytime is defined as dipper, a decrease of more than 20% as extreme dipper, a decrease of less than 10% as non-dipper. An increase in blood pressure compared to the daytime is defined as a reverse dipper. The non-dipper condition in blood pressure has been mostly examined in hypertensive patients and it was found to be an independent predictor of poor prognosis. Heart failure is a chronic process characterized by neuroendocrine system maladaptation. In this process, observing an increase in sympathetic activation, a decrease in baroreceptor sensitivity, and an increase in peripheral resistance. It is thought that this situation may change the diurnal rhythm of blood pressure. Studies show that mostly non-dipper and reverse dipper conditions are prevalent in heart failure patients. We aimed to investigate the circadian rhythm of blood pressure in patients with ischemic and non-ischemic cardiomyopathy.

Methods: Our study included 44 patients who followed in our cardiology outpatient clinic, with ejection fraction (EF) was less than 40%, left heart enlarged, and sinus rhythm. Patients with severe valvular disease, a recent history of myocardial infarction, atrial fibrillation, chronic renal failure, and biventricular pacemaker were excluded. Twenty-two patients were ischemic cardiomyopathy (CMP) and 22 patients were non-ischemic cardiomyopathy. In addition, 22 patients were identified as a control group. Laboratory tests, echocardiograms, and 24-hour ambulatory blood pressure monitoring (ABPM) were performed for all patients.

Results: The mean age of the study population was 57.8±10.5 and 72.7% (n=48) of patients were male. Body mass index (BMI), glomerular filtration rate (GFR), EF, high density lipoprotein, and low density lipoprotein were higher in the control group. There was no significant difference between ischemic CMP and non-ischemic CMP groups in terms of EF, age, diabetes mellitus, hypertension, BMI, GFR, laboratory parameters, and drugs used (except statin). Dipper was higher in the control group (59.0%, p<0.001), non-dipper was higher in the non-ischemic CMP group (54.0%, p<0.001), and reverse dipper was higher in the ischemic CMP group (63.6%, p<0.001).

Conclusions: In patients with systolic heart failure, blood pressure diurnal rhythm changes. As a result of our study, reverse dipper was significantly higher in the ischemic CMP group. Therefore, it can be predicted that adverse clinical conditions such as mortality and/or hospitalization may be more than in these patients compared to the non-ischemic CMP group. Although the importance of ABPM and non-dipper in defining the prognosis in heart failure patients is known, the role of ABPM should be considered in regulating the hours of taking antihypertensive drugs in these patients.

Table A: Comparison of demographic, laboratory, and ambulatory blood pressure monitoring results of ischemic CMP, non-ischemic CMP, and control groups. Table B: Comparison of drug-using of ischemic and non-ischemic CMP groups

A	İschemic CMP (n=22)	Non-ischemic CMP (n=22)	Control (n=22)	
Age, year	58.5±7.0	57.8±15.8	57.1±8.8	0.667
Male gender, n (%)	21(95.4)	14(63.6)	13(59.0)	<0.001
Diabetes mellitus, n (%)	5(22.7)	4(18.2)	-	<0.001
Hypertension, n (%)	6(27.2)	10(45.4)	2(9.1)	<0.001
Body mass index, kg/ m^2	24.1±3.9	23.6±6.4	27.6±3.4	0.010
GFR, ml/dk	72.0±25.1	62.9±26.3	89.6±15.5	0.001
Ejection fraction, %	25.2±6.2	26.9±6.4	61.9±2.8	<0.001
Triglycerid, mmol/L	144.4±79.0	144.4±79.0	168.3±76.0	0.327
LDL, mmol/L	92.7±27.0	89.2±35.0	123.3±23.0	<0.001
HDL, mmol/L	33.4±7.9	34.1±12.0	42.2±11.0	0.010
Dipper, n (%)	2(9.1)	4(18.2)	13(59.0)	<0.001
Non-dipper, n (%)	6(27.3)	12(54.5)	5(22.7)	<0.001
Reverse dipper, n (%)	14(63.6)	6(27.3)	4(18.2)	<0.001
B				
Drug-using (Ischemic vs non-ischemic CMP), n (%)				
ACE-İ	20(90.9)	18(81.8)	1	>0.005
ARB	1(4.5)	3(13.6)	-	>0.005
Beta-blocker	18(81.8)	20(90.9)	-	>0.005
MRA	19(86.3)	19(86.3)	-	>0.005
Diuretic	21(95.4)	19(86.3)	-	>0.005
Acetylsalicilic acid	21(95.4)	19(86.3)	-	>0.005
Statin	21(95.4)	2(9.1)	-	<0.001

ACE-I: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, CMP: Cardiomyopathy, GFR: Glomerular filtration rate, HDL: High density lipoprotein, LDL: Low density lipoprotein, MRA: Mineralocorticoid receptor antagonist.

Heart Failure

PB-054

The importance of autophagy, microtubules, and microtubule inhibition in patients with HFrEF

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Background and Aim: In this study, we aimed to compare the serum beclin-1 levels which is one of markers and moderators of autophagic activity and the β1 -tubulin level, which is one of the cardiomyocyte structure proteins in the serum of patients with heart failure with reduced ejection fraction (HFrEF), and those healthy subjects. Also, we investigated serum beclin-1 and β1 -tubulin levels according to etiological classifications (ischemic/non-ischemic subgroups). Additionally, the subgroup of patients using colchicine as a microtubule inhibitor for at least three months due to HFrEF was included

Methods: This study included 50 patients with HFrEF (25 with ischemic etiology, 25 with non-ischemic etiology) and 30 healthy subjects between January 2018 and December 2019 in Istanbul University Cardiology Institute. Serum beclin-1 and β1 -tubulin levels were determined by using ELISA method by the ELISA Kit.

Results: Although serum beclin-1 and β1-tubulin levels of all HFrEF group did not reach statistical significance compared to the control group, serum beclin-1 and β1-tubulin levels were increased (p=0.64) [(p=0.6) respectively]. However, NT-proBNP levels were found significantly higher (p=0.01). Serum beclin-1 and serum β1-Tubulin levels correlated with ejection fraction in 50 patient with HFrEF (p=0.018, R^2=0.088). (p=0.018, R^2=0.086). In the non-ischemic etiology subgroup with HFrEF especially had higher serum beclin-1 levels (p=0.01There was also no significant correlation between creatinine and eGFR levels and autophagic activity (p=0.482). Also, we found lower levels of NT-proBNP that did not reach statistical significance and higher beclin-1 levels to reach statistical significance (p=0.015) in the colchicine using patient subgroup.

However,β1- tubulin levels had increased in ischemic HFrEF patients according to the non-ischemic subgroup (p=0.26). Besides, in the subgroup analysis of non-ischemic HFrEF patients used colchicine (n=13) was detected increased levels of β1- tubulin (p=0.29) and decreased levels of NT*proBNP (p=0.69). But, for at least three months low dose colchicine used patient subgroup had better EF (p=0.009) and smaller diastolic left ventricular diameters (p=0.002) respectively.

Conclusions: Autophagy especially increased in the HFrEF with non-ischemic etiology and patients used colchicine subgroup. However, β1- tubulin levels had increased in ischemic HFrEF patients according to the non-ischemic and patients used colchicine subgroup.Therefore low dose colchicine probably regulates autophagy, microtubules inhibition, and vesicle trafficking in HFrEF.

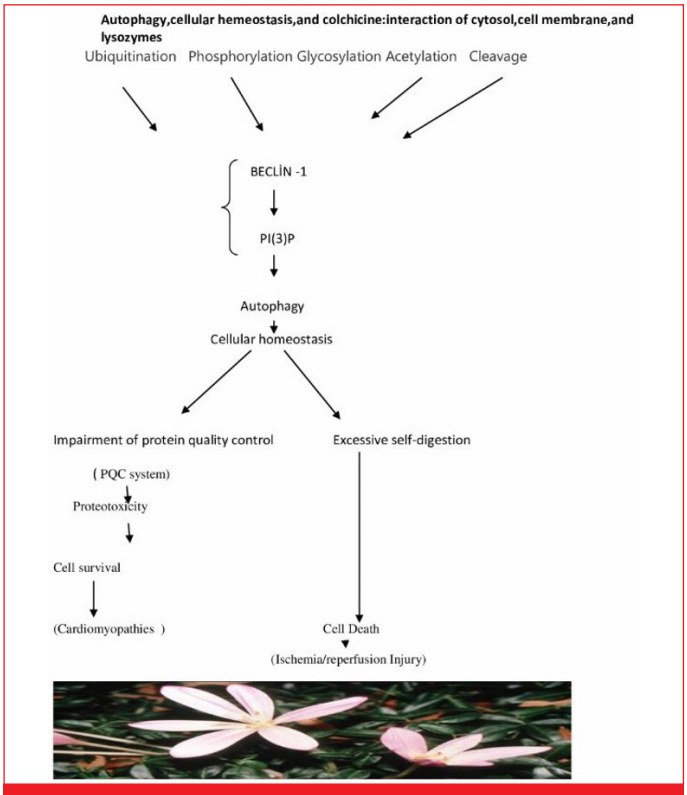

Figure 1. Post-translational modifications of Beclin 1 affect protein stability, confirmation, activity, and its interactome and can be used as a molecular rheostat to fine-tune autophagic activity. â€¢Targeting Beclin 1 modifiers to regulate Beclin 1 post-translational modifications could provide a possible therapeutic intervention for upregulatin autophagy. â€¢Colchicine accelerates the physiological clearance of misfolded proteins from cells through the protein quality control (PQC) system and cytoprotective autophagy.

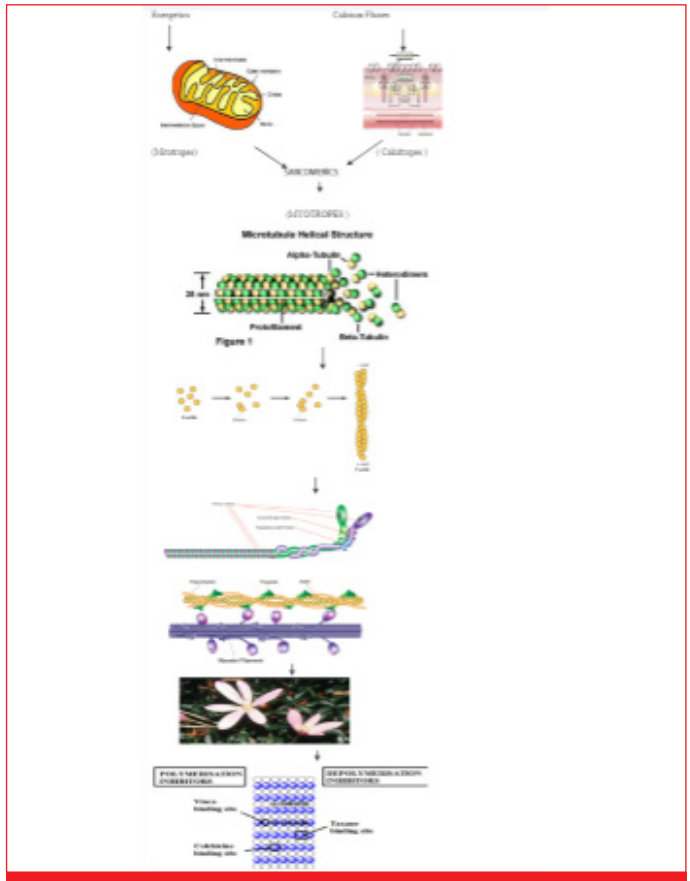

Figure 2. Actin microfilaments are more effective in the microenvironment, and in case of inhibition of microtubules such as the use of colchicine, they may be more bound to myosin heads and increase the number of myosin heads able to bind to actin to undergo a Powerstroke (myotrope effect) such as omecamtiv mecarbil.

Parameters A	Patient(N=50)	Control(N=30)	*P -Value*
NT-proBNP(pg/dl)	2230.4±2079.7	53.6±21.5	*0.01*
Beta-Tubulin(pg/dl)	97.4±174.8	81.3±172.7	*0.6*
Parameters B	Ischemic (N: 25)	Non-ischemic (N: 25)	*P -Value*
NT-proBNP(pg/dl)	2647±2182	1813±1924	0.01
Beta-Tubulin(pg/dl)	108±222	53±95	0,26
Parameters C	Colchicine + (N: 13) (values after treatment with colchicine for at least three months)	Colchicine – (N:37)	*P -Value*
NT-proBNP(pg/dl)	*2029.9±2344*	*2300±2000*	*0.69*
BETA-Tubulin(pg/dl)	*37±81*	*97±194*	*0.29*
EF(%)	*32.6±6.4*	*27.5±3.2*	*0.009*
LVDD(mm)	*58.1±8.2*	*66.1±9.0*	*0.002*
TAPSE(mm)	*18.4±3.0*	*19.8±2.7*	*0.33*
Parameters of D Group	All Patients (N: 50)	Controls (N: 30)	P-value*
EF (%)	31.3±6.2	60	0.001
NT-proBNP (pg/dl)	2230.4±2079,7	53.6±21,5	0.01
Beclin-1 (ng/ml)	6.1±10.4	2.7±6.3	0.64
Parameters of E Group	İschemic HFrEF subgroup(N:25)	Non-ischemic HFrEF subgroup (N: 25)	P- value
Age	64.4±10,2	53.9±13.3	0.004
Gender (male %)	23 (%92)	20 (80%)	0.2
Diabetes Mellitus (%)	14 (%56)	8 (32%)	0.08
Hypertension (%)	19 (76%)	10 (40%)	0.01
Hyperlipidemia (%)	19 (76%)	6 (24%)	0.0001
Cigaret (%)	18 (72%)	6 (24%)	0.001
Alcohol (%)	1 (4%)	2 (8%)	0.5
NT-proBNP (pg/dl)	2647±2182	1813±1924	0.01
Beclin-1 (ng/ml)	2.07±4.7	12.7±16.1	0.01
Parameters of F Group	Colchicine + HFrEF subgroup (N: 13)	Colchicine – HFrEF subgroup (N:37)	P Value
EF (%)(Initial)	27.5±3.2	32.6±6.4	0.009
LVD (mm) 69±7.6(Initial)		59.7±8.9	0.002
LA (mm) (Initial)	46.8±9.4	46.0±8.8	0.59
RVD (mm)	25.0±1.8	25.5±3.3	0.54
TAPSE (mm) (Initial)	18.4±3.0	19.8±2.7	0.33
NT-proBNP (pg/dl) (3 months later)	2029.9±2344	2300±2000	0.69
Beclin-1 (ng/ml) (3 months later)	12.44±10	3.4±8.4	0.015

Table 1.The importance of autophagy, microtubules and microtubule inhibition in patients with heart failure with reduced ejection fraction

A) Average of ejection fraction (EF) values, NT-ProBNP and Beta-tubulin levels of patients with total Heart Failure reduced EF(HFrEF) (N:50) and Control Group (N:30) Participating in the Study (mean±SD) (P:0.01;P:0.29 respectively).. B) NT-proBNP and Beta-tubulin values of HFrEF patients according to ischemic (N:25) and non-ischemic (N:25) etiology patient groups included in the study (mean±SD) (0.01;0.26 respectively) C) Differences in NT-ProBNP and Beta-tubulin levels and echocardiographic parameters of for at least three months colchicine treated non-ischemic subgroup(N:13) and not using colchicine patient group in the HFrEF patient group (N: 50) included in the study (mean±SD)(0.69 ;0.29; 0.009; 0.002 respectively). *Continuous variables are presented as mean±SD and dichotomous variables as percentages. NS, not significant. A two-tailed t-test was used for comparison of means, and x2-test for percentages. D) Average of Echocardiographic EF values, NT-ProBNP and Beclin-1 Levels of Patients with Heart Failure (N: 50) and Control Group (N:30) participating in the study (mean±SD). E) Differences in NT-proBNP and Beclin-1 values of patients with heart failure according to ischemic (N:25) and non-ischemic (N:25) etiology included in the study (mean±SD). F) Differences in NT-ProBNP and Beclin-1 Levels and echocardiographic parameters of colchicine used (N:13) and non-colchicine used patients in the HFrEF patient group (N:37) included in the study (mean±SD). *Continuous variables are presented as mean±SD and dichotomous variables as percentages. A two-tailed t-test was used for comparison of means, and x2-test for percentages.

Heart Failure

PB-055

Prognostic importance of low albumin levels in chronic systolic heart failure

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Background and Aim: Heart failure is a complex disease that interacts with other organ systems, such as the kidneys, and the liver, in a complex manner. Abdominal congestion manifests in a significant number of patients with advanced congestive heart failure. Increased intra-abdominal pressure, as an extreme marker of abdominal congestion, is correlated with renal dysfunction in advanced congestive heart failure. Interesting findings provide evidence that alterations in the liver contribute to systemic congestion in heart failure.

The aims of this study were to investigate the liver enzymes and albumin levels and to evaluate their associations with subsequent cardiovascular mortality in a group of patients with chronic systolic heart failure.

Methods: 650 consecutive patients (423 men and 227 women) with chronic heart failure of both ischemic and non-ischemic etiology were followed up for a mean period of 78 months. The mean age was 65±12 years, the mean left ventricular ejection fraction was 26.0±9%. The mean brain natriuretic peptide (BNP) level was 1862±3194 pg/mL and the mean hs-CRP level was 2.5±4.0 mg/L. The primary endpoint was cardiovascular mortality.

Results: During the follow-up period, 268 (41.2%) patients died of cardiovascular causes. Patients who died during follow up period were older than patients who survived, p<0.001. Left ventricular ejection fractions of patients who died were lower, (22.7±8.1% vs. 27.8±8.1% p<0.001). AST and ALT levels were significantly higher in patients who died during follow up, p<0.001 and p<0.001. Albumin (3.3±0.6 g/dL vs 3.7±0.5 g/dL, p<0.001) and total protein (6.6.±0.7 g/dL vs 6.9±0.5 g/dL, p<0.001) levels were significantly lower in this patient group with cardiovascular mortality. Creatinine levels (1.6±1.0 mg/dL vs 1.3±0.6 mg/dL, <0.001) and urea levels (80.0±48.8 mg/dl vs 60.7±33.2 mg/dl, <0.001) were increased in patients who died during this period (Table). Brain natriuretic peptide and hs-CRP levels did not show significant difference in patients who died and compared to patients who survived. Using stepwise multivariate Cox proportional hazards regression analyses, age, left ventricular ejection fraction and albumin levels proved to be significant independent predictors of cardiovascular mortality. An albumin level of 2.82 g/ dL had a 78% sensitivity and 97% specificity to predict cardiovascular mortality (AUC=0.29, 95% CI 0.25-0.34, p<0.001).

Conclusions: Impaired liver and kidney functions are important prognostic factors in patients with chronic systolic heart failure. Low albumin levels will help to identify patients who have poor prognosis.

Table 1. Clinical and biochemical parameters in patients with chronic systolic heart failure

	Patients who died during follow up n=268	Patients who survived during follow up n=382	*p*
Age (years)	69±11	62±11	<0.001
Male/Female	66%/44%	64%/46%	0.665
AST (U/L)	132.1±347.4	31.7±43.7	<0.001
ALT (U/L)	130.5±328.1	29.1±35.2	<0.001
Albumin (g/dL)	3.3 ±0.6	3.7±0.5	<0.001
Total Protein (g/dL)	6.6.±0.7	6.9±0.5	<0.001
Creatinine (mg/dL)	1.6±1.0	1.3±0.6	<0.001
Urea (mg/dL)	80.0±48.8	60.7±33.2	<0.001
Left ventricular ejection fraction (%)	22.7±8.1	27.8±8.1	<0.001

PB-056 [Heart Failure]

The Effect of Enhanced External Counterpulsation (EECP) Treatment on Peripheral Venous Symptoms

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Background and Aim: Chronic venous diseases (CVD) encompass a large spectrum of morphologic and functional abnormalities in the venous system with complaints such as aching, burning, muscle cramps, swelling. Enhanced external counterpulsation (EECP) which is proven safe and effective in patients with coronary artery disease (CAD) and chronic heart failure is a technique that increase venous return and augment diastolic blood pressure by using inflatable cuffs wrapped around the lower extremities. So it has peripheral training effect which may influence the venous symptoms. This study assessed the effect of EECP on symptoms of CVD by using the Venous Insufficiency Epidemiological and Economic Study-Quality of Life/Symptoms (VEINES-QoL/Sym) questionnaire.

Methods: This study was designed as prospectively for evaluating venous symptoms before and after EECP treatment. Patients with symptomatic CAD and chronic heart failure referred to EECP therapy were recruited. The study population consisted of 30 consecutive patients who admitted to the cardiology clinics fulfilled the inclusion and exclusion criteria. All participants were examined for the presence of CVD. VEINES-Sym questionnaire, which consisting of ten items, was applied to assess venous symptoms one day before treatment and at the completion of EECP treatment. All patients' demographic parameters, cardiovascular risk factors, other co-morbid diseases and drug usage were noted.

Results: The mean age of the patients was 64.62±9.67 years. Patients characteristics, demographic and clinical data are presented in Table 1. After 35 hours of EECP, 28 patients (93%) had at least one New York Heart Association (NYHA) functional class reduction compared with baseline. The mean of the NYHA class significantly decreased from 3.17±0.53 to 1.77±0.57 after EECP treatment (p<0.0001). Evaluation of venous disease symptoms and VEINES-Sym scores of the study patients are demonstrated in Table 2. There was a significant improvement in their swelling and night cramps symptoms (p<000.1; p= 0.05, respectively). The other symptoms such as heavy legs, itching and burning were decreased whereas aching, throbbing and tingling were increased with EECP treatment even both not statistically significant. Also, The LVEF was significantly increased from 33.50±10.59 to 35.57±10.26 (p=0.018) after EECP treatment.

Conclusions: The results of present study implicated that patients treated with EECP showed a significant reduction in swelling and night cramps symptoms. Although total VEIN score did not change after EECP procedure, improvement in swelling and night cramps underlines beneficial effects of EECP through the venous vascular territory.

Table 1. Demographic and clinical characteristics of the patients*

Age (year)	64.62±9.67
Sex, male	23 (76.7%)
Diabetes mellitus	19 (63.3%)
Hypertension	21 (70%)
Dyslipidemia	17 (56.7%)
Smoking	6 (20%)
Prior CABG	11 (36.7%)
PCI	25 (83%)
PCI+CABG	10 (33.3%)
ICD	12 (40%)
Left ventricular ejection fraction ≤ 40%	22 (73.3%)
Drugs	
Antiaggregants (ASA and/or clopidogrel)	30 (100%)
Beta-blockers	28 (93.3%)
ACE inhibitors /ARB	19 (63.3%)
Statins	10 (33.3%)
Diuretics	23 (76.7%)
Ivabradine	6 (20%)

* Data are presented as mean ± SD or n (%). BMI, Body mass index; CABG, Coronary artery bypass graft surgery; PCI, Percutaneous coronary intervention; ICD, Implantable cardioverter defibrillator; ASA, Acetylsalicylic acid; ACE, Angiotensin-II converting enzyme; ARB, Angiotensin II receptor blockers.

Table 2. Comparison of venous leg symptoms and VEINES-Sym scores pre- and post-treatment of EECP

Symptoms	Pre-treatment (n=30)	Post-treatment (n=30)	*p* value
Heavy legs	4.27±1.36	4.53±1.00	0.23
Aching legs	4.03±1.35	3.67±1.44	0.20
Swelling	3.53±1.54	4.57±0.82	<0.0001
Night cramps	3.83±1.42	4.30±1.02	0.05
Heat/burning	4.47±1.11	4.53±1.00	0.69
Restless legs	4.30±1.15	4.30±1.24	1.00
Throbbing	4.63±0.89	4.40±1.16	0.15
Itching	4.63±0.96	4.77±0.90	0.29
Tingling	4.30±1.26	4.07±1.20	0.35
VEINES-Sym score	38.00±7.75	39.17±6.93	0.27

Heart Failure

PB-057

Predictors of length of stay in heart failure patients hospitalized with COVID-19

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Background and Aim: The current pandemic coronavirus disease (COVID-19) is an ongoing health crisis and affects millions of people worldwide. COVID-19 patients with multiple comorbidities, including heart failure (HF), show poor clinical outcomes and require long-term hospitalization. In this study we aimed to assess predictors of length of stay (LoS) in patients with HF who were hospitalized for COVID-19.

Methods: In this study, we retrospectively evaluated 240 consecutive patients hospitalized for COVID-19 who had a previous diagnosis of HF with reduced or preserved ejection fraction in two centers, between March 15, 2020 and December 01, 2020. We compared clinical characteristics, laboratory findings and in-hospital outcomes (death, requiring mechanical ventilation and vasopressor therapy) according to LoS. The median LoS was 9.0 (interquartile range [IQR], 7-16) days. Those hospitalized for less than 9 days were defined as shorter LoS group, those hospitalized for more than 9 days as longer LoS group. Multivariate regression analysis was used to determine the longer LoS predictors.

Results: Mean age of the study population was 73.2±10 years old and 67.5% were male. One hundred nineteen patients (49.5%) had been hospitalized for more than 9 days. There was no significant difference between the two groups in terms of comorbidities include hypertension, prior atrial fibrillation, coronary artery disease (p>0.05, for all). Laboratory parameters relevant in infection were significantly higher in the longer LoS group than in the shorter LoS group and lymphocytes (p=0.004) and albumin (p<0.001) were significantly lower. In univariable linear regression analyses, systolic blood pressure, lymphocyte count, creatinin, albumin, C-reactive protein, fibrinogen and lactate dehydrogenase were found to be associated with longer LoS. These seven significant variables tested in the univariable model were included from the subsequent multivariable model. Finally, in the multivariable model, albumin was found to show a significant association with a longer LoS. (Table 1)

Conclusions: This retrospective study of hospitalized patients with COVID-19 with HF showed that patients with longer LoS had worse clinical outcomes and that the most important predictor of longer LoS was albumin.

Variables	Univariable linear regression				Multivariable linear regression			
	β	CI		p-value	β	CI		p-value
		Lower bound	Upper bound			Lower bound	Upper bound	
Age, years, mean ± SD	0.100	-0.001	0.010	0.122				
SBP, mmHg, mean ± SD	-0.129	-0.005	0.000	**0.046**	-0.034	-0.003	0.002	0.611
Heart rate, bpm, mean ± SD	0.076	-0.001	0.004	0.242				
First oxygen saturation, %, mean ± SD	-0.107	-0.012	0.001	0.098				
Lymphocyte, $10^{+3}/\mu L$	-0.155	-0.146	-0.015	**0.016**	-0.090	-0.116	0.022	0.178
Creatinine, mg/dL	0.127	0.000	0.073	**0.049**	0.029	-0.029	0.046	0.671
Sodium, mmol/L	0.118	-0.001	0.016	0.068				
Albumin, g/dL	-0.244	0.258	-0.083	**0.000**	-0.163	-0.208	-0.019	**0.019**
CRP, mg/L	0.238	0.001	0.002	**0.000**	0.114	0.000	0.001	0.157
Ferritin, ng/dL	-0.030	0.000	0.000	0.647				
Procalsitonin, ng/mL	0.054	-0.003	0.008	0.425				
Fibrinogen, mg/dL	0.224	0.000	0.001	**0.001**	0.087	0.000	0.000	0.261
LDH, U/L	0.159	0.000	0.000	**0.014**	0.058	0.000	0.000	0.406
Lactate, mmol/L	-0.034	-0.030	0.021	0.712				

Table 1. Linear regression model testing associations of predictors with length of stay (n=240)

Heart Failure

PB-058

Pro brain natriuretic peptide level and echocardiographic evidence of systolic or diastolic dysfunction in non-obese patients with non-acute dyspnea

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Background and Aim: In daily practice, we easily and frequently request serum pro brain natriuretic peptide (pro-BNP) level and transthoracic echocardiography (TTE) from most patients with non-acute onset dyspnea. The aim of the study was to evaluate serum pro-BNP levels and evidence of left ventricular (LV) systolic dysfunction (SD) or diastolic dysfunction (DD) in non-obese patients with non-acute dyspnea.

Methods: Patients with non-acute dyspnea who underwent transthoracic echocardiography (TTE) between October 2020 and May 2021 were examined. Patients with moderate to severe valvular heart disease, congenital heart disease, cardiomyopathy, infiltrative storage disease, BMI>35 kg/m^2, GFR<60 ml/dk/m^2, as well as emergency presentations for acute dyspnea were excluded. Pro-BNP≥125 pg/ml was considered as elevated which is the kit's cut-off value. SD included patients with LV ejection fraction (EF) <50%. The presence of DD was decided according to the guidelines.

Results: Ultimately, 435 patients were included in the study. DD was found in 108 subjects (25%) and SD was present in 51(12%). Pro-BNP levels were 2512.64±5243.4 and 3793.72±4413.58 pg/ml respectively in DD and SD, and 382.79±1022 in patients without LV dysfunction. Pro-BNP levels were elevated in 61% (n=264) of the patients (≥125 pg/ml). Fifty patients (19%) had an EF <50% and 98 patients (37%) had DD with pro-BNP≥125. There was no evidence of SD or DD with TTE in 160 (94%) of the patients with pro-BNP<125 and 149 (52%) of those with ≥125. Patients without SD or DD but with pro-BNP≥125 had a significantly higher frequency of H2FPEF Score ≥5, AF, malignancy, and hospitalization for heart failure (HF) than those with pro-BNP<125. In patients without SD or DD but with pro-BNP≥125 interventricular septal (IVS) thickness, LV mass index (LVMI), right ventricular, right atrium, left atrial volume index (LAVI), pulmonary artery pressure (sPAP), and E/e' ratio were higher than those with pro-BNP<125, while E/A ratio was lower. In the multivariate logistic regression analysis, the presence of AF, pro-

BNP and hs-troponin-T levels were independent predictors of hospitalization for HF. A Pro-BNP<752.1 pg/ml excludes SD with 72.5% sensitivity and 83.1% specificity (AUC=0.862, 95% CI 0.812-0.912, p<0.001).752.1 pg/ml cut-off had a negative predictive value (NPV) of 95.8% (95% CI 93.57%-97.27%) for SD. Pro-BNP<350.3 pg/ml excludes DD with 71.3% sensitivity and 75.5% specificity (AUC=0.809, 95% CI 0.763-0.855, p<0.001).350.3 pg/ml cut-off for pro-BNP had a NPV of 88.85% (95% CI 85.47%-91.52%) for DD.

Conclusions: SD or DD was not detected in a significant proportion of patients with elevated pro-BNP in non-obese patients with non-acute dyspnea. It seems appropriate to use higher cut-off values, especially for SD. Pro-BNP was found to be a significant predictor of hospitalization for HF in the absence of SD or DD with TTE. This indicates that the diagnosis of diastolic HF far beyond the TTE guideline criteria.

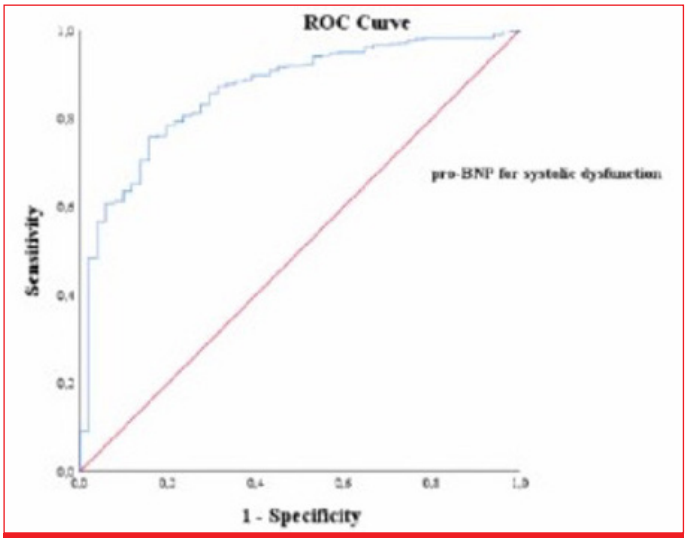

Figure 1. ROC curve analysis showing the specifcity and sensitivity of the pro-BNP in excluding systolic dysfunction

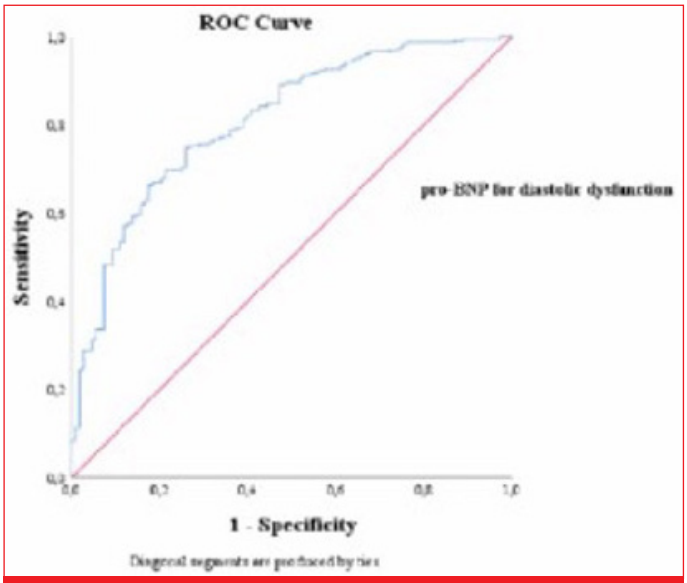

Figure 2. ROC curve analysis showing the specifcity and sensitivity of the pro-BNP in excluding diastolic dysfunction

	Total Patients (n=435)	Pro-BNP<125 (n=171)	Pro-BNP ≥125 (n=264)	p-value
Age (year)	48,02 ± 9,7	44,18 ± 12,3	49,13 ± 7,8	0,083
Gender, Male, n(%)	220 (50,6 %)	83 (48,5 %)	137 (51,9 %)	0,494
Female, n(%)	215 (49,4 %)	88 (51,5 %)	127 (48,1 %)	
HR (bpm)	76,24 ± 13,9	76,33 ± 14,4	76,31 ± 14,4	0,722
BMI (kg/m²)	28 ± 4,8	27,32 ± 4,3	27,63 ± 4,7	0,963
LV EF ≥ 50%	384 (88,3 %)	170 (99,4 %)	214 (81,1 %)	<0,001*
LV diastolic dys + (n,%)	108 (24,8 %)	10 (5,8 %)	98 (37,1 %)	<0,001*
diastolic dys – (n,%)	327 (75,2 %)	161 (94,2 %)	166 (62,9 %)	
H₂FPEF Score ≥ 5, n(%)	64 (16,7 %)	8 (4,7 %)	56 (21,2 %)	<0,001*
H₂FPEF Score	2 (0-9)	1 (0-5)	3 (0-9)	<0,001*
HT, n(%)	98 (22,5 %)	31 (18,1 %)	67 (25,4 %)	0,077
DM, n(%)	32 (7,4 %)	9 (5,3 %)	23 (8,7 %)	0,178
CAD, n(%)	73 (16,8 %)	17 (9,9 %)	56 (21,2 %)	0,002*
COPD, n(%)	19 (4,4 %)	5 (2,9 %)	14 (5,3 %)	0,236
AF, n(%)	33 (7,6 %)	1 (0,6 %)	32 (12,1 %)	<0,001*
Malignancy, n(%)	50 (11,5 %)	14 (8,2 %)	36 (13,6 %)	0,082
Previous COVID-19, n(%)	44 (10,1 %)	34 (19,9 %)	10 (3,8 %)	<0,001*
Hospitalization, n(%)	100 (22,9 %)	19 (11,1 %)	81 (30,7 %)	<0,001*
Laboratory Findings				
Hgb (gr/dl)	12,67 ± 2,1	13,29 ± 1,6	12,19 ± 2,2	<0,001*
Creatinine (mg/dl)	0,8 ± 0,2	0,77 ± 0,2	0,82 ± 0,2	0,174
Hs-troponin-T (pg/ml)	4,47 (3-588)	3 (3-104)	7,13 (3-588)	<0,001*
Pro-BNP (pg/ml)	133,5 (6,95-14364)	65,63 (6,95-123,8)	383 (127,5-14364)	<0,001*
Treatment				
Antiaggregant, n(%)	152 (34,9 %)	45 (26,3 %)	107 (40,5 %)	0,002*
Beta-blocker, n(%)	197 (45,3 %)	45 (26,3 %)	152 (57,6 %)	<0,001*
CCB, n(%)	110 (25,3 %)	36 (21,1 %)	74 (28 %)	0,102
ACE inh/ ARB, n(%)	182 (41,8 %)	52 (30,4 %)	130 (49,2 %)	<0,001*
Diuretic, n(%)	107 (24,6 %)	15 (8,8 %)	92 (34,8 %)	<0,001*
Statin, n(%)	125 (28,7 %)	39 (22,8 %)	86 (32,6 %)	0,028*
Chemotherapy, n(%)	50 (11,5 %)	14 (8,2 %)	36 (13,6 %)	0,082
Echocardiographic Findings				
LVEDV (ml)	97,54 ± 21,9	97,1 ± 19,7	97,91 ± 23,9	<0,001*
LVESV (ml)	35,82 ± 16,7	33,69 ± 12	37,65 ± 19,7	<0,001*
EF (%)	62,56 ± 9	64,67 ± 6,1	60,75 ± 10,7	<0,001*
LVEDD (mm)	45,8 ± 4,2	45,75 ± 3,9	45,84 ± 4,6	<0,001*
IVS (mm)	11,3 ± 2,1	10,9 ± 1,6	11,7 ± 2	<0,001*
LV mass index (gr/m²)	124,76 ± 39,5	111,96 ± 28,23	135,73 ± 44,5	<0,001*
LA (mm)	36,9 ± 4,9	35,5 ± 4,4	38,2 ± 5,1	<0,001*
RV (mm)	26,8 ± 2,6	26,6 ± 2,7	26,9 ± 2,5	<0,001*
RA (mm)	32,5 ± 3,7	31,6 ± 3	33,3 ± 0,4	<0,001*
E/A ratio	0,94 ± 0,4	1,04 ± 0,4	0,86 ± 0,4	0,008*
E/e' ratio	9,62 ± 3,2	8,59 ± 2,3	10,51 ± 3,5	<0,001*
LAVI (ml/m²)	24,54 ± 10,6	21,75 ± 9	26,93 ± 11,3	<0,001*
CO (L/min)	5,02 ± 1,5	4,85 ± 1,6	5,17 ± 1,4	0,802
sPAP (mmHg)	28,46 ± 7,7	26,5 ± 6,7	30,14 ± 8,1	<0,001*
TAPSE (mm)	21,83 ± 5	21,6 ± 5,4	22,02 ± 4,6	0,006*
MAPSE (mm)	14,95 ± 1,8	15,27 ± 1,6	14,68 ± 1,9	<0,001*

Table 1. Demographic, clinical and echocardiographic characteristics of patients with pro-BNP<125 and ≥125 pg/mL

	Total Patients (n=309)	Pro-BNP<125 (n=160)	Pro-BNP ≥125 (n=149)	p-value
Age (year)	46,21 ± 9,1	45,77 ± 12	46,69 ± 4,4	0,990
Gender, Male, n(%)	145 (46,9 %)	77 (48,1 %)	68 (45,6 %)	0,662
Female, n(%)	164 (53,1 %)	83 (51,9 %)	81 (54,4 %)	
HR (bpm)	76,09 ± 14,1	75,33 ± 13,7	77,14 ± 14,8	0,963
BMI (kg/m²)	27,72 ± 4,8	27,26 ± 4	27,74 ± 4,9	0,502
H₂FPEF Score ≥ 5, n(%)	25 (8,1 %)	6 (3,8 %)	19 (12,8 %)	0,004*
H₂FPEF Score	1 (0-8)	1 (0-7)	2 (0-8)	<0,001*
HT, n(%)	59 (19,1 %)	31 (19,4 %)	28 (18,8 %)	0,896
DM, n(%)	20 (6,5 %)	9 (5,6 %)	11 (7,4 %)	0,530
CAD, n(%)	37 (12 %)	15 (9,4 %)	22 (14,8 %)	0,145
COPD, n(%)	12 (3,9 %)	4 (2,5 %)	8 (5,4 %)	0,192
AF, n(%)	9 (2,9 %)	1 (0,6 %)	8 (5,4 %)	0,013*
Malignancy, n(%)	38 (12,3 %)	14 (8,8 %)	24 (16,1 %)	0,049*
Previous COVID-19, n(%)	42 (13,6 %)	32 (20 %)	10 (6,7 %)	0,001*
Hospitalization, n(%)	55 (17,8 %)	18 (11,3 %)	37 (24,8 %)	0,002*
Laboratory Findings				
Hgb (gr/dl)	12,72 ± 2	13,18 ± 1,8	12,17 ± 2,1	<0,001*
Creatinine (mg/dl)	0,79 ± 0,2	0,78 ± 0,2	0,80 ± 0,2	0,373
Hs-troponin-T (pg/ml)	3,62 (3-588)	3 (3-406)	5,64 (3-588)	<0,001*
Pro-BNP (pg/ml)	118,45 (6,95-4178,6)	71,53 (6,95-124,70)	271,3 (127,5-4178,6)	<0,001*
Treatment				
Antiaggregant, n(%)	96 (31,1 %)	42 (26,3 %)	54 (36,2 %)	0,058
Beta-blocker, n(%)	109 (35,3 %)	39 (24,4 %)	70 (47 %)	<0,001*
CCB, n(%)	73 (23,6 %)	34 (21,3 %)	39 (26,2 %)	0,309
ACE inh/ ARB, n(%)	111 (35,9 %)	48 (30 %)	63 (42,3 %)	0,025*
Diuretic, n(%)	43 (13,9 %)	12 (7,5 %)	31 (20,8 %)	0,001*
Statin, n(%)	74 (23,9 %)	30 (18,8 %)	44 (29,5 %)	0,027*
Chemotherapy, n(%)	38 (12,3 %)	14 (8,8 %)	24 (16,1 %)	0,049*
Echocardiographic Findings				
LVEDV (ml)	94,31 ± 17,7	95,74 ± 17,9	92,62 ± 17,5	0,324
LVESV (ml)	32,47 ± 8,8	32,63 ± 7,7	32,28 ± 10	0,091
EF (%)	64,75 ± 4,8	65,09 ± 4,6	64,35 ± 5,1	0,021*
LVEDD (mm)	45,21 ± 3,6	45,5 ± 3,6	44,86 ± 3,6	0,324
IVS (mm)	11,1 ± 2	10,8 ± 2	11,3 ± 2	<0,001*
LV mass index (gr/m²)	116 ± 30	110,42 ± 27,9	122,65 ± 31,5	<0,001*
LA (mm)	36,1 ± 0,5	35,5 ± 0,5	36,8 ± 0,5	<0,001*
RV (mm)	26,6 ± 0,2	26,4 ± 0,3	26,8 ± 0,2	0,023*
RA (mm)	32,1 ± 0,3	31,5 ± 0,3	32,8 ± 0,3	0,023*
E/A ratio	0,98 ± 0,4	1,06 ± 0,4	0,89 ± 0,4	<0,001*
E/e' ratio	8,79 ± 2,2	8,31 ± 2	9,36 ± 2,2	0,017*
LAVI (ml/m²)	23,29 ± 10,2	21,89 ± 9,1	24,96 ± 11,2	<0,003*
CO (L/min)	5,1 ± 1,5	4,94 ± 1,5	5,3 ± 1,5	0,707
sPAP (mmHg)	26,67 ± 6	25,84 ± 5,8	27,65 ± 6,2	<0,001*
TAPSE (mm)	22,11 ± 4,8	21,93 ± 4,6	22,32 ± 5,2	0,302
MAPSE (mm)	15,02 ± 1,6	15,23 ± 1,4	14,78 ± 1,8	0,467

Table 2. Demographic, clinical, and echocardiographic characteristics of patients with pro-BNP<125 and ≥125 pg/mL without systolic or diastolic dysfunction

	Variable	r	p
Pro-BNP	Age	0,200	<0,001*
	Hs-troponin-T	0,649	<0,001*
	MAPSE	-0,317	<0,001*
	sPAP	0,475	<0,001*
	H₂FPEF Score	0,405	<0,001*
	EF	-0,320	<0,001*
	LAVI	0,440	<0,001*
	LV mass index	0,360	<0,001*
	E/A	-0,122	0,018*
	E/e'	0,414	<0,001*
	IVS thickness	0,306	<0,001*

Table 3. Correlation analysis of pro-BNP with clinical, laboratory and echocardiographic parameters

Variable	OR	95 % Confidence Interval	p-value
AF	3,247	1,127-9,352	0,029*
Pro-BNP	1,000	1,000-1,000	0,006*
Hs-troponin-T	1,012	1,004-1,020	0,004*
EF	1,015	0,981-1,051	0,392
sPAP	0,987	0,953-1,022	0,453
TAPSE	0,958	0,914-1,004	0,070

Table 4. Multivariate logistic regression analysis of clinical and echocardiographic parameters predicting hospitalization for heart failure

Heart Failure

PB-059

Evaluation of the relationship between plasma osmolality levels and neutrophil/lymphocyte ratio in heart failure with reduced ejection fraction

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Background and Aim: Heart Failure (HF) is a progressive disease that goes with chronic inflammation and osmolality changes. Neutrophil-to-lymphocyte ratio (NLR) reflects a systemic inflammatory response in most diseases. The relationship between plasma osmolality levels and systemic inflammatory response in patients with HF is not well known. We aimed to evaluate the possible associations of systemic inflammation depicted by NLR with the osmolality of body fluids and N-terminal prohormone B-type natriuretic peptide (NT-proBNP) levels in patients with HF.

Methods: This cross-sectional and prospective study included 189 consecutive patients with HF with an ejection fraction (EF) of <40%. The demographic, clinical data, laboratory parameters and current medications of patients were obtained on admission. Plasma osmolality (mOsm/kg) was calculated as (2 × Na) + (BUN/2.8) + (Glucose/18), which is known as Worthley equation [1]. Patients were classified into 4 groups based on quartiles of admission plasma osmolality as hypo-osmolar (1st quartile, n=53), normo-hypo-osmolar (2nd quartile, n=32), normo-hyperosmolar (3rd quartile, n=54), and hyperosmolar (4th quartile, n=50) (Fig 1). The values between 275 and 295 mOsm/kg were accepted as normal [2]. Patients with acute decompensated HF, lactic acidosis or ketoacidosis and other pathological conditions which may affect osmolal gap or NLR were excluded from the study. We analyzed the relationship between NLR, plasma osmolality and NT-proBNP levels in HF patients.

Results: The demographic and clinical data, medications, and laboratory findings of the study population are presented in Table 1. The mean age of the study population was 68±10.7 years (138 male; 51 female). The EF and NT-proBNP levels were similar in the four subgroups. NLR was significantly different between the subgroups (Table 1, Fig.2). The hyperosmolar group had an increased NLR (p=0.007). Osmolality was negatively correlated with lymphocyte count (r=-0.244, p=0.002) but was significantly positively correlated with NLR (r=0.201, p=0.011) (Table 2A). NT-proBNP levels were negatively correlated with lymphocytes (r=-0.247; p<0.001), and LVEF (%) (r=-0.281; p<0.001) and positively correlated with NLR (r=0.276; p<0.001) (Table 2B). Multivariate logistic regression analysis revealed that the presence of high osmolality was an independent predictor of the increased NLR (Table 3).

Conclusions: The inflammatory activity in HF may be related to plasma osmolality. The present study suggests that systemic inflammatory response depicted by NLR is affected by plasma osmolality levels in patients with HF. NLR and plasma osmolality are inexpensive and easily obtainable parameters. Clinicians should take into account the association between them in clinical practice. As a result, plasma osmolality should be considered as an important clinical variable that affects the inflammatory status in the HF phenomenon. To the best of our knowledge, no available studies have assessed this relationship so far.

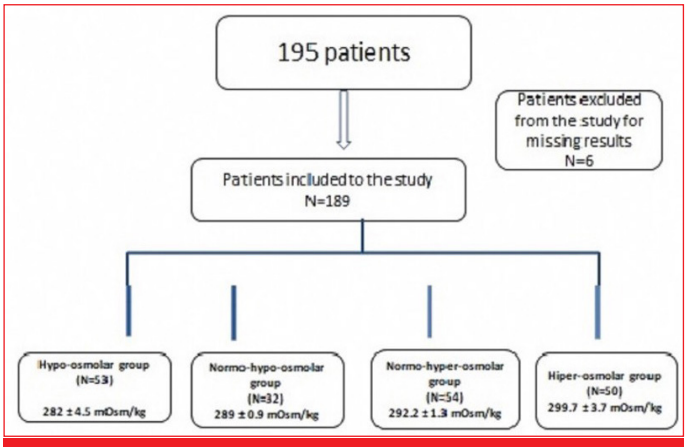

Figure 1. Flow chart of the study design

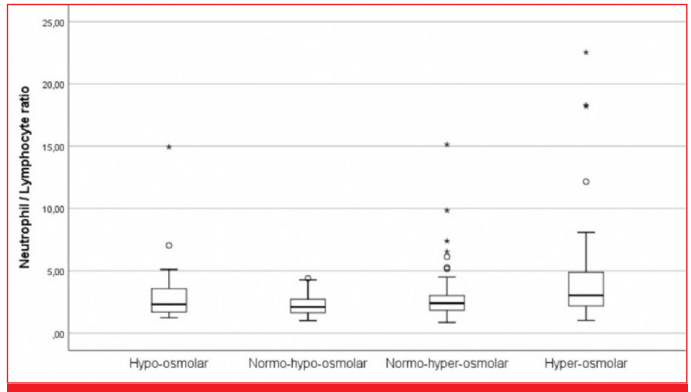

Figure 2. Relationship between neutrophil-to-lymphocyte ratio and plasma osmolality.

Variables	Hypo-osmolar	Normo-osmolar		Hyper-osmolor	P value
	(n=53)	Normo-hypo-osmolar (n=32)	Normo-hyper-osmolar (n=54)	(n=50)	
Mean age, (years)	65.3±12.6	65.7±11.2	69.8±9.5	69.5±8.8	0.065
Male, (%)	39(73.5)	24(75)	39(72.2)	36(72)	0.989
Hypertension, n (%)	27(50.9)	22(68.7)	36(66.6)	30(60)	0.283
Hyperlipidemia , n (%)	14(26.4)	8(25)	17(31.4)	11(22)	0.031
Diabetes mellitus, n (%)	14(26.4)	11(34.3)	17(31.4)	22(44)	0.290
CAD, n (%)	34(64.1)	23(71.8)	37(68.5)	36(72)	0.822
Disease duration, (months)	5.75(0-18)	4.67(0-20)	4.77(0-20)	6.61(0-18)	0.416
Resting heart rate, (bpm)	84.7(56-126)	80(48-118)	81(51-140)	86.5(56-168)	0.439
Systolic BP, (mm Hg)	118.8±13.3	115.6±17	117±11.5	118±16.8	0.743
Diastolic BP, (mm Hg)	62.2±6.4	60.6±7.2	61±7.3	63.8±8.5	0.160
AF, n (%)	24(45.2)	13(40.6)	20(37)	24(48)	0.687
LVEF, (%)	30.6±4.1	30.5±4.3	30.5±4.8	29.8±4.5	0.763
Osmolality, (mOsm/kg)	282±4.5	289±0.9	292.2±1.3	299.7±3.7	<0.001 a,b,c,d,e,f*
Fasting glucose, (mg/dL)	102(57-194)	119(81-215)	115(70-307)	130(51-325)	0.008 a,e*
BUN, (mg/dL)	17.1(7-44)	19.5(11-34.5)	22.5(11.7-52)	29.7±14.9	<0.001 b,c,f*
Creatinine, (mg/dL)	1.03±0.2	1.11±0.3	1.20±0.5	1.36±0.4	<0.001 b,c,f*
Sodium, (mEq/L)	135±2.8	137.5±1.43	138±1.95	141±3.1	<0.001 b,c,f*
Potassium, (mEq/L)	4.3±0.5	4.4±0.5	4.4±0.5	4.3±0.6	0.799
hs-CRP, (mg/dL)	7.14(3.5-8)	6.5(0.1-9)	7.3(0-8)	9.5(3.9-9)	0.638
NT-proBNP, (pg/mL)	3329(136-19600)	2503(153-8870)	4735(147-26700)	6642(170-35000)	0.107
WBC, x 10^3/L	7.1±2.1	7.7±2.1	7.7±2.1	7.3±2.6	0.401
Neutrophils, x 10^3/L	4.5(2.1-8.8)	4.6(1.9-8.9)	4.9(2.4-11.5)	5.0(2.0-11.8)	0.452
Lymphocytes, x 10^3/L	1.8±0.6	2.2±0.95	1.95±0.75	1.50±0.72	<0.001 a,c,d*
NLR	2.86(1.2-15)	2.26(1-4.4)	3.04(0.85-15.1)	4.44(1-22.5)	0.007 c*
Hemoglobin, (g/dL)	13.2±1.8	13.3±2.2	13.1±1.8	12.3±1.7	0.126 c*
Platelet, x 10^3/L	235(94-619)	239(118-520)	236(116-432)	209(94-399)	0.146
Antiplatelet agents, n(%)	50(94.3)	32(100)	51 (94.4)	47(94)	0.585
Beta-blockers, n (%)	45(84.9)	28(87.5)	47(87)	44(88)	0.853
ACEi /ARB, n (%)	34(64.1)	21(65.6)	39(72.2)	32(64)	0.738
Digoxin, n (%)	14(26.4)	5(15.6)	6(11.1)	9(18)	0.303
Diuretics, n (%)	34(64.1)	25(78.1)	34(62.9)	40 (80)	0.253
MRA, n (%)	34(66)	19 (59.3)	28(51.8)	28(56)	0.500

Abbreviations: ACEi, angiotensin- converting enzyme inhibitor; AF, atrial fibrillation; ARB, angiotensin receptor blocker; BP, blood pressure; BUN, blood urea nitrogen; CAD, coronary artery disease; hs-CRP, high-sensitivity C-reactive protein ; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; NLR, neutrophil to lymphocyte ratio; NT-proBNP, N-terminal prohormone brain natriuretic peptide; WBC, white blood cell.

*If there is p<0.05 as the significance level, P^a: Hypo-osmolar vs Normo-hypo-osmolar, P^b: Hypo-osmolar vs Normo-hyper-osmolar, P^c: Hypo-osmolar vs hyper-osmolar, P^d: Normo-hypo-osmolar vs normo-hyper-osmolor, P^e: Normo-hypo-osmolar vs Hyper-osmolor, P^f: Normo-hyper-osmolor vs Hyper-osmolor

Table 1. Baseline characteristics, medications and laboratory findings of study patients

	Correlation coefficients (r)	P value
Age, (years)	0.186	0.010
NT-proBNP, (pg/mL)	0.075	0.348
WBC, x 10³/L	0.007	0.924
Neutrophils, x10³/L	0.098	0.179
Lymphocytes, x10³/L	-0.244	0.002
NLR	0.201	0.011
Hemoglobin, (g/dL)	-0.163	0.025
Platelet , x 10³/L	-0.107	0.142

Abbreviations: NLR, neutrophil to lymphocyte ratio; NT-proBNP, N-terminal prohormone brain natriuretic peptide; WBC, white blood cell.

Table 2A. Correlations between plasma osmolality and laboratory parameters

	r	P value
LVEF, (%)	-0.281	<0.001
hs-CRP, (mg/dL)	0.435	<0.001
Osmolality, (mOsm/kg)	0.130	0.078
WBC, x 10³/L	0.006	0.939
Neutrophils, x 10³/L	0.105	0.155
Lymphocytes, x 10³/L	-0.247	<0.001
NLR	0.276	<0.001
Hemoglobin, (g/dL)	-0.356	<0.001
Platelet , x 10³/L	-0.038	0.610

Abbreviations: hs-CRP, high-sensitivity C-reactive protein; LVEF, left ventricular ejection fraction; NLR, neutrophil to lymphocyte ratio; NT-proBNP, N-terminal prohormone brain natriuretic peptide; WBC, white blood cell.

Table 2B. Correlations between B-type natriuretic peptide and other variables.

	Univariate Analysis			Multivariate Analysis		
Variables	OR	95% CI	P	OR	95% CI	P
Age	1.028	0.986-1.072	0.200			
High Osmolality presence	**1.106**	**1.020-1.199**	**0.015**	**1.581**	**1.301-1.792**	**0.028**
hs-CRP	**1.052**	**1.020-1.084**	**0.001**	**2.581**	**2.127-2.891**	**<0.001**
NT-proBNP	0.997	0.989-1.001	0.091			

Abbreviations: 95% CI, 95% confidence interval; hs-CRP, high-sensitivity C-reactive protein; N/L, neutrophil to lymphocyte ; NT-proBNP, N-terminal prohormone brain natriuretic peptide; OR, odds ratio.

Table 3. Factors predicting increased neutrophil to lymphocyte ratio on logistic regression analysis.

Heart Failure

PB-060

Clinical, Echocardiographic and Hemodynamic Predictors of Major Cardiac Events in Patients with Advanced Heart Failure

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Background and Aim: Advanced heart failure (AHF) accounts for approximately 6% to 25% of heart failure (HF) patients and causes frequent hospitalization and death. Many studies have been conducted to determine mortality and morbidity parameters in patients with AHF. We aimed to investigate the factors affecting major cardiac events (MACE) in patients with AHF.

Methods: The patients with AHF referred for evaluation for heart transplantation (HT) or left ventricular assist device (LVAD) implantation were retrospectively analyzed. The AHF was defined as stage D or NYHA class III-IV as previously described. We obtained demographic features, echocardiographic findings, right heart catheterization findings and blood test results of the patients from the hospital database. A total 470 of patients were stratified into two groups; namely, those with MACE (334 patients) and without MACE (136 patients). MACE was defined as composite of rehospitalization due to decompensation of HF, cardiac-related mortality, the need for LVAD, and need for HT.

Results: Over a median follow-up time of 174.5days (IQR: 36.8-688.0 days) MACE was observed in 334 (71.1%) patients. There were 215 (45.7%) cases for hospitalization 95 (20.2%) LVAD implantation cases, 24 (4.2%) urgent HT cases and 99 (21.0%) death cases. Demographic features, echocardiographic findings, right heart catheterization findings and blood test results of the patients were summarized in the Table 1. Compared to the patients without MACE, the patients with MACE had lower body mass index, hemoglobin level, left ventricular ejection fraction, cardiac output, mean arterial pressure, and more NYHA IV (13% vs. 8%), smoking, severe mitral regurgitation and pulmonary hypertension (PH) (89% vs.71%), higher pulmonary artery mean pressure (PAPm), pulmonary vascular resistance and right atrial pressure. Univariate analysis revealed that smoking (HR:1.31, 95%CI:1.14-1.45, p<0.001), severe tricuspid regurgitation (HR: 1.70, 95%CI:1.16-2.49, p=0.006), combined pre- and postcapillary PH (Cpc-PH) compared to isolated- postcapillary PH (Ipc-PH) (HR: 1.57, 95%CI:1.29-1.91, p<0.001), decrease in hemoglobin level (HR: 1.27, 95%CI:1.1-1.31, p<0.001), increase in PAPm (HR: 1.17, 95%CI:1.01-1.35, p:0.034), cardiac index (HR: 1.21, 95%CI:1.11-1.31, p=<0.001), increase in uric acid (HR:1.28, 95% CI: 1.06-1.54, p<0.008, decrease in TAPSE (HR:1.2, 95%CI:1.1-1.35, p=0.036) were associated with MACE. Multivariate analysis revealed that, decrease in serum Na level (HR:1.29, 95%CI:1.11-1.43, p=0.003), increase in PAPm (HR:1.34, 95%:1.07-1.61, p=0.022), Cpc-PH compared to Ipc-PH (HR: 1.42, 95%CI:1.11-1.81, p=0.005) were independent predictors of MACE.

Conclusions: Sodium, increase in PAPm and Cpc-PH were independent risk factors for MACE in patients with AHF.

Table 1. Clinical, Laboratory, Echocardiographic and Hemodynamic Characteristics of the Patients

	Without MACE (n=136)	With MACE (n=334)	*P* value
Age	47.8±10.2	48.8±10.5	0.0335
Sex (male, %)	85	88	0.543
Body mass index (%)	27.2±5.1	26.2±4.6	0,037
Hypertension (%)	23	18	0.262
Diabetes mellitus (%)	26	19	0.082
Smoking (%)	51	37	0.005
Coronary artery disease (%)	46	48	0.913
Percutanous coronary intervention (%)	34	37	0.572
Implantable cardiac defibrillator (%)	37	39	0.707
NYHA class (%)			
III	92	87	
IV	8	13	<0.001
Atrial fibrillation (%)	14	16	0.404
WBC	8.7±2.1	8.6±2.6	0,899
Hemoglobin	13.7±1.9	12.9±2.1	<0,001
AST	26.1±14.3	36.8±54.1	0,027
ALT	27.1±27.1	39.7±59.3	0,021
Total Billuribin	1.0±0.9	1.5±1.2	<0,001
Albumine	7.6±10.7	6.0±8.3	0,109
Sodium	137.7±3.9	136.4±16.9	0,224
Glucose	129±57.1	134.9±69.6	0,407
Creatinine	1.1±0.7	1.2±0.7	0.470
Uric acid	7.7±2.3	8.6±2.7	0,001
Ejection fraction (%)	21.2±3.8	20.1±4.0	0,007
LVEDD (cm)	6.8±0.9	6.9±1.0	0,289
LVESD (cm)	5.9±0.9	6.3±3.1	0,119
TAPSE (mm)	16.0±5.5	14.7±5.2	0,031
Pulmonary arterial wedge pressure (mm Hg)	23.5±8.3	22.0±7.5	0,065
Pulmonary artery systolic pressure (mm Hg)	53.5±17.6	49.8±21.7	0,061
Pulmonary artery mean pressure (mm Hg)	34.8±12.0	31.3±14.4	0,008
Cardiac output			
L/dk	3.3±0.9	3.9±2.0	0,001
Cardiac index			
L/dk/m^2	1.8±0.5	2.1±1.4	0,025
Right atrial pressure			
(mm Hg)	11.5±6.5	9.3±5.4	0,001
Pulmonary vascular resistance (WU)	3.8±3.1	3.1±3.4	0,032

Values were presented as mean ± SD, % of cohort, median (25th – 75th percentile). Comparisons of variables were evaluated by the chi-square

ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, LVEDD: Left ventricular end-diastolic dimension, LVESD: Left ventricular end-systolic dimension, MACE: Major cardiac event, NYHA:New York Heart Association, TAPSE: Tricuspid annular plane systolic excursion,WBC: White blood cell

Heart Failure

PB-061

The relationship of Fetuin-A, Omentin-1, and Chemerin with clinical classification in Heart Failure

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Background and Aim: Heart failure (HF) is a clinical syndrome in which the heart cannot pump enough blood for the needs of the human body in terms of life functions. Some biochemical diagnostic tests as well as echocardiography play a role in the early diagnosis of this syndrome. The complex pathophysiology of HF suggests that many other markers may be useful in diagnosis and follow-up.

After many recent studies, it has been suggested that adipokines fetuin-A, omentin-1 and chemerin may be suitable biomarkers for the diagnosis of HF. Our main aim in this study is to determine the relationship between fetuin-A, omentin-1 and chemerin levels with HF clinical classification.

Methods: The patients admitted to the cardiology service with symptomatic HF with heart failure with preserved ejection fraction (HF-pEF, n=62), heart failure with reduced EF (HF-rEF, n=61) and heart failure with mid-range EF (HF-mrEF, n=63) were included in the study. A total of 246 participants were evaluated by taking the control group (n=60) for comparison. The main characteristics of all groups were recorded and serum levels of fetuin-A, omentin-1 and chemerin were evaluated.

Results: When compared with the control group, there was a significant difference for fetuin-A with the HF-rEF group [452.3 (441.4-528.9); 555.3 (453.7-615.6) p<0.001, respectively]. When evaluating for omentin-1, there was a significant difference between the control group and HF-rEF [19.3 (16.9-22.7); 22.9 (16.8-29.7) p<0.001, respectively]. However, there was no significant difference for chemerin between the HF groups and the control group. Significant cut-off value for fetuin-A was found to be 485 in ROC analysis (AUC:0.74 sens:0.72 (95% CI:0.57-0.82), spec:0.69 (95% CI:0.59-0.83), p<0.001).

Conclusions: Serum fetuin-A levels were found to be increased in HF, especially in the HF-rEF group and it can be used in the diagnosis of this patient group.

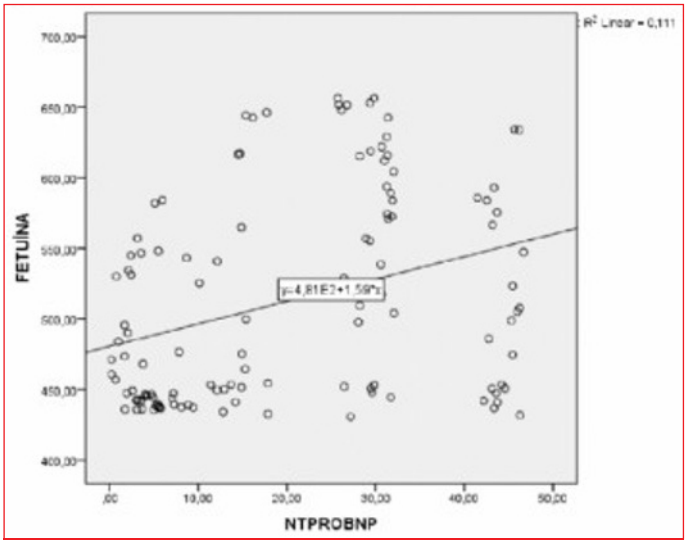

Figure 1. Correlation between fetuin-A and NT-proBNP

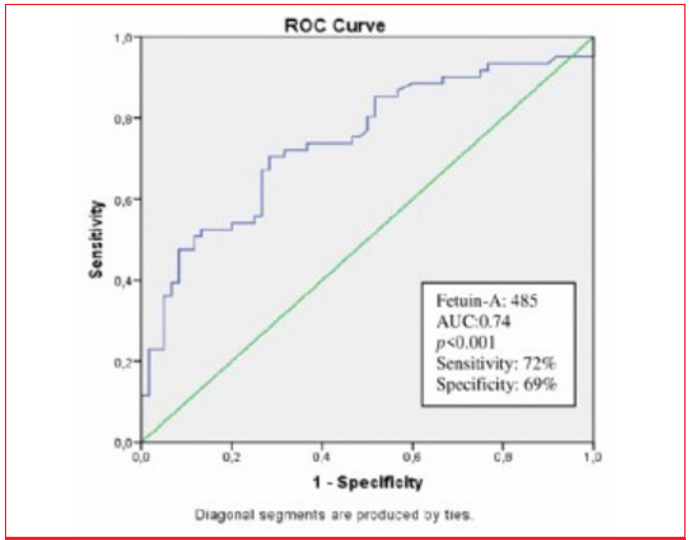

Figure 2. ROC analysis to detect HF-rEF

Table 1. Distribution of the basic characteristics of the groups

Variables	HF-rEF (n=61)	HF-mrEF (n=63)	HF-pEF (n=62)	Control (n=60)	p value
Age (mean±SD)	67.62±9,89	69,93±11,59	71,53±11,65	67,35±6,65	0.29
BMI (mean±SD)	30.21±6.20[a]	32.72±8.08[a]	33.46±7.05[b]	29.27±4.33	0.01
Gender (female, n%)	25(41%)[a]	32(50.8%)[a,b]	37(59.7%)[b]	28(46.7%)[a]	<0.01
Diabetes Mellitus (n%)	33(54.09%)	33(47.61%)	30(48.38%)	22(36.66%)	0.18
Hypertension (n%)	37(60.65%)	41(65.07%)	46(74.19%)	35(58.33%)	
COPD (n%)	19(31.14%)	14(22.22%)	23(37.09%)	-	
Asthma (n%)	1(1.69%)	4(6.34%)	4(6.45%)	-	
CAD (n%)	52(85.24%)[a]	50(79.36%)[a]	37(59.67%)[b]	32(53.33%)[b]	<0.01
Atrial Fibrillation (n%)	25(40.98%)	22(34.92%)	26(41.93%)	1(1.66%)	
Pace Rhythm (n%)	3(4.91%)	1(1.58%)	1(1.61%)	-	
LBBB (n%)	10(16.39%)	5(7.93%)	2(3.22%)	-	
Smoking (n%)	10(16.39%)	3(4.76%)	3(4.83%)	5(8.33%)	
Furosemid Dosage (mg mean±SD)	93.77±46.69	92.69±52.70	118.70±50.68	-	
Digoxin (n%)	7(11.47%)	2(3.17%)	3(4.83%)	-	
MRA (n%)	47(77.04%)	34(53.96%)	23(37.09%)	-	
Ivabradin (n%)	7(11.47%)	4(6.34%)	-	-	
ACE/ARB (n%)	56(91.80%)	55(87.30%)	42(67.74%)	16(26.66%)	
Betablocker (n%)	60(98,369%)	57(90.47%)	46(74.19%)	1(1.66%)	
Antiaggregan (n%)	40(65.57%)	40(63.49%)	28(45.16%)	24(40%)	
Warfarin (n%)	4(6.65%)	5(7.93%)	9(14.51%)	1(1.66%)	
NOAK (n%)	20(32.78%)	7(11.11%)	11(17.74%)	-	

(LBBB: Left Bundle Branch Block, BMI: Body Mass Index, CAD: Coronary Arteries Disease, NOAK: New Oral Anticoagulant, MRA: Mineralocorticoid Receptor Antagonist, COPD: Chronic Obstructive Pulmonary Disease, ACE: Angiotensin Converting Enzyme Blocker, ARB: Angiotensin Receptor Blocker, HF-rEF: Heart Failure with Reduced Ejection Fraction, HF-mrEF: Midrange Heart Failure, HF-pEF: Heart Failure with Protected Ejection Fraction)

Table 2. Evaluation of NYHA classification of heart failure patient groups

	HF-rEF (n=61)	HF-mrEF (n=63)	HF-pEF (n=62)	Control (n=60)	Total
NYHA 1 (n, %)	-	-	-	60 (100%)	60 (24.4%)
NYHA 2 (n, %)	48 (78.7%)	36 (57.1%)	32 (51.6%)	-	116 (47.2%)
NYHA 3 (n, %)	13 (21.3%)	25 (39.7%)	29 (46.8%)	-	67 (27.2%)
NYHA 4 (n, %)	-	2 (3.2%)	1 (1.6%)	-	3 (1.2%)

NYHA: New York Heart Association HF-rEF: Heart Failure with Reduced Ejection Fraction HF-mrEF: Midrange Heart Failure HF-pEF: Heart Failure with Protected Ejection Fraction

Table 3. Comparison of the laboratory and echocardiographic characteristics of the groups

Variables	HF-rEF (n=61)	HF-mrEF (n=63)	HF-pEF (n=62)	Control (n=60)	*p*
Hemoglobin (g/dL)	13.99±1.53	13.37±1.53	13.46±1.20	13.83±1.11	0.053
Hematocride (%)	42.28±4.61	40.89±4.47	41.26±3.97	40.81±3.20	0.178
Platelet (10^3/u/L)	212 (169-240)	216 (181-275)	212 (172-264)	242 (208-283)	0.343
MCV (f/L)	88.31±5.19	87.74±7.57	85.62±7.48	87.05±3.81	0.096
WBC (10^3/u/L)	8.42±2.25	7.83±1.96	7.75±1.81	7.81±1.88	0.208
Glucose (mg/dL)	117 (90-144)[a]	112 (89-130)[a]	109 (96-132)[a]	95 (90-112)[b]	0.008
Creatinine (mg/dL)	1.19±0.36[a]	1.13±0.51[a]	1.27±0.57[a]	0.83±0.18[b]	<0.001
BUN (mg/dL)	28.04±14.29[a]	27.83±13.46[a]	33.34±15.10[a]	15.96±5.41[b]	<0.001
Sodium (mmol/L)	139.00±3.11	139.38±4.00	139.85±4.41	141.28±6.12	0.054
Potassium (mmol/L)	4.58±0.61	4.43±0.57	4.44±0.66	4.36±0.36	0.220
CRP (mg/L)	14.35±14.87[a]	10.93±13.73[a,b]	17.49±22.15[a]	6.00±6.44[b]	<0.001
Uric Acid (mg/dL)	7.38±2.61[a,b]	6.58±2.12[a]	7.66±2.08[b]	5.14±1.34[c]	<0.001
ALT(U/L)	22.57±24.88	38.71±88.08	17.51±10.82	25.81±16.64	0.079
AST(U/L)	24.06±17.95	35.01±84.0	21.20±10.46	22.85±9.55	0.287
GGT(U/L)	67.78±62.52[a]	60.55±53.79[a]	51.85±52.97[a,b]	36.73±30.56[b]	0.007
TSH (ng/dL)	2.52±4.89	1.38±0.86	2.10±4.20	1.72±1.19	0.254
LDL (mg/dL)	89.83±38.71[a]	87.25±0.87[a]	91.12±34.50[a]	108.63±35.23[b]	0.006
TG (mg/dL)	103(76-162)	90(69-147)	87(65-133)	123(92-163)	0.059
Cholesterol (mg/dL)	154.95±53.64[a,b]	148.07±52.74[a]	152.04±43.04[a]	176.06±40.05[b]	0.007
EF (%)	30(26-35)	44(40-45)	54(52-55)	55(54-58)	
EDC (cm)	5.84±0.82	5.08±0.47	4.70±0.34	4.60±0.29	
LA Size (cm)	4.5(4.1-4.8)	4.4(4.2-4.7)	4.5(4.2-4.8)	3.6(3.4-3.8)	
Omentin-1 (ng/mL)	22.9(16.8-29.7)[a]	18.1(16.3-21.6)[b]	18.1(16.5-22.8)[b]	19.3(16.9-22.7)[b]	<0.001
Chemerin (ng/mL)	6.42(6.16-8.53)[a]	6.16(5.91-6.36)[b]	6.3(5.9-8.6)[a,b]	6.2(5.9-6.4)[a,b]	0.003
Fetuin-A (ng/mL)	555.3(453.7-615.6)[a]	443.2(436.2-453.2)[b]	512.7(452.6-571.6)[a]	452.3(441.4-528.9)[b]	<0.001
NT-proBNP (ng/mL)	31.3(29.1-43.3)[a]	30.6(28.3-32)[a]	29.2(25.5-29.6)[b]	5.3(3-11)[c]	<0.001

a, b, c In superscript explanations, the same letters show that there is no statistically significant difference between them, and different letters show that there is a statistically significant difference. (MCV: Mean Corpuscular Volume, WBC: White Blood Cell, BUN: Blood Urea Nitrogen, CRP: C-reactive protein, ALT: Alanine Aminotransferase, AST: Aspartate aminotransferase, GGT: Gamma Glutamyl Transferase, TSH: Thyrotrophin-Stimulating Hormone, T4: Tetraiodothyronine, LDL: Low Density Lipoprotein, TG: Triglyceride, EF: Ejection Fraction, EDC: End Diastolic Diameter, LA: Left Atrium)

Cardiac Imaging / Echocardiography

PB-063

The pulmonary annular motion velocity assessed using tissue Doppler imaging could predict the proximal right coronary artery occlusion in patients with inferior myocardial infarction

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Background and Aim: The right ventricle myocardial infarction (RVMI) is one of the leading reasons for right ventricle (RV) dysfunction. RVMI occurs in 20-50% of inferior infarctions. Echocardiography was performed to forecast the RV participation and the proximal right coronary artery (RCA) occlusion in patients with acute inferior MI. This study sought to answer whether pulmonary annulus motion velocity assessed using tissue Doppler imaging (PAMVUT) values correlated with proximal RCA lesions in patients with acute inferior myocardial infarction in which RCA is the culprit's vessel.

Methods: In the presented study, a total of 50 patients who were diagnosed with acute inferior myocard infarction whose culprit lesions were in the right coronary artery were comprised. Patients with an RCA occluded proximal to the right ventricular branch were allocated to Group A, and those with an RCA occlusion distal to the RV branch were appointed to Group B. Echocardiographic parameters, including the PAMVUT, were measured.

Results: There were significant differences between the groups in terms of parameters showing right ventricular functions. In the univariate correlation analysis; a positive correlation was found between PAMVUT and RV TAPSE, with FAC, with St. In the multivariate logistic regression test, PAMVUT was defined as an independent predictive parameter for proximal RCA occlusion. In the ROC analysis, PAMVUT <8,5 cm/s predicted proximal RCA occlusion with 85% sensitivity and 69% specificity (AUC=0.80, p<0.001).

Conclusions: In this study, we found PAMVUT values to be an important predictor of proximal RCA occlusions.

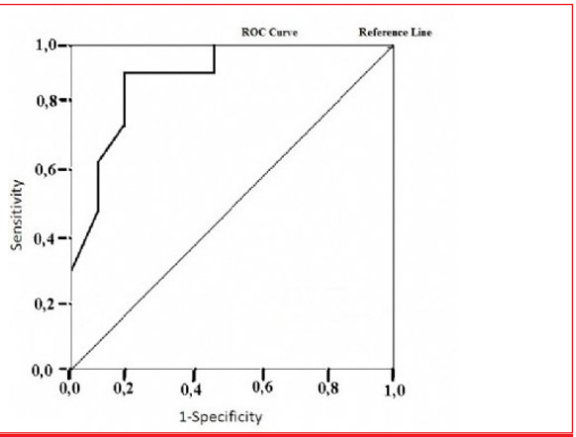

Figure 1.

Variables	Group A(n:26, 52%)	Group B(n:24, 48%)	*P* value
Sex male(n, %)	18(80.7%)	20(83.3%)	0,412
Age(mean±SD)	57.9±10.6	58.1±11.4	0,542
HT(n, %)	9(34.6%)	9(37.5%)	0,309
DM(n, %)	16(61.5%)	13(54.1%)	0,219
Smoking(n, %)	9(34.6%)	7(29.1%)	0,268
Dyslipidemia(n, %)	12(46.1%)	10(41.6%)	0,387
BMI(kg/m2)	25.9±4.2	26.3±4.4	0,426
Glucose(mg/dl)	147(100-182)	149(100-195)	0,981
Creatinine(mg/dl)	0.84±0.21	0.85±0.25	0,621
Hemoglobin(mg/dl)	13.5±1.4	13.5±1.5	0,854
Troponin(ng/ml)	2.8(0.3-9.8)	2.0(0.5-7.2)	0,713
Systolic blood pressure(mmHg	137.8±25.3	135.6±24.8	0,654
Heart rate(bpm)	83.6±13.5	77.9±15.2	0,247
Door to reperfusion(min)	30(30-30)	30(30-30)	0,255

Table 1: Clinical characteristics, demographic and laboratory finding of the study population. BMI, body mass index; DM, diabetes mellitus; HTN, hypertension

Table 1.

Variables	Group A (n=26)	Group B (n=24)	*P* value
EDD(cm)	4.8(4.6-5.1)	4.8(4.7-5.2)	0,625
ESD(cm)	3.0(2.8-3.3)	3.1(2.9-3.2)	0,245
LAD(cm)	3.6±0.2	3.7±0.2	0,842
EF(%)	56(50-58)	55(51-58)	0,568
LV E/A	0.8(0.6-0.9)	0.7(0.6-0.8)	0,861
LV E/Em	8(7-9)	8(7-9)	0,596
TAPSE(cm)	1.6±0.3	1.9±0.3	**<0,001**
St(cm/s)	10(8-12)	13(9-14)	**<0,001**
RV MPI	0.5(0.4-0.6)	0.5(0.4-0.7)	0,233
FAC(%)	32(28-44)	44(40-47)	**<0,001**
PAMVUT(cm/s)	8(7-10)	13(8-14)	**<0,001**

Table 2: Echocardiographic characteristic of the study population. EDD, end diastolic diameter; ESD, end systolic diameter; LAD left atrium diameter, EF, ejection fraction; LV, left ventricle; E, early ventricular filling velocity; A, late ventricular filling velocity; Em ventricular tissue doppler early diastolic velocity, RV right ventricle, TAPSE tricuspid annular plane systolic excursion; PAMVUT, pulmonary annular motion velocity using tissue Doppler imaging; St, tissue Doppler imaging derived tricuspid annulus systolic velocity; MPI myocardial performance index, FAC fractional area change

Table 2.

PAMVUT		
Variables	r	*p*
TAPSE(cm)	0,421	0,001
FAC(%)	0,341	<0,001
St	0,259	0,001

Table 3: Correlation of RV free wall strain with echocardiographic measurements. FAC, fractional area change; PAMVUT, pulmonary annular motion velocity using tissue Doppler imaging; St, tissue Doppler imaging derived tricuspid annulus systolic velocity; TAPSE, tricuspid annular plane systolic excursion

Table 3.

Variable	OR	CI	*p*
PAMVUT(cm/s)	0,541	0,311-0,768	<0,001

Table 4: The result of multivariate logistic regression analysis for the prediction of proximal RCA lesion. PAMVUT, pulmonary annular motion velocity using tissue Doppler imaging ; RCA, right coronary artery

Table 4.

Cardiac imaging / Echocardiography

PB-064

Evaluation of the relationship between paraaortic adipose tissue and ascending aortic aneurysm with a new method

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Background and Aim: Ascending aortic aneurysm is the dilatation of ascending aorta and associated with increased mortality. Para-aortic adipose tissue (PAT) is the local adipose tissue surrounding the aorta externally. In addition to protecting the aorta against trauma, it significantly contributes to aortic atherosclerosis and enlargement with bioactive molecules such as adiponectin and growth factors it secretes. In support of these data, studies conducted with computed tomography and magnetic resonance showed that individuals with aortic aneurysms had more PAT than healthy individuals. However, there is no study on its measurement with transthoracic echocardiography (TTE). In this study, we measured PAT for the first time with TTE. This study aims to investigate the possible relationship of echocardiographically measured PAT with ascending aortic width and cardiovascular risk factors.

Methods: All patients over the age of 18 who applied to the cardiology outpatient clinic and underwent TTE were included in the study. For this purpose, we used Roman's classification. PAT was defined as the hypoechoic space in front of ascending aortic 2 cm above the sinotubular junction at the end of the systole. The values were measured in three cardiac cycles and were averaged. According to Roman's classification (Aortic size index, ASI) patients were divided into 2 groups: Group 1, no aortic dilatation (ASI < 21 mm/m^2), and Group 2, aortic dilatation (ASI ≥ 21mm/m^2).

Results: A total of 321 unselected patients were divided into the following two groups: patients with ascending aortic dilatation (AAD) (96 patients) and patients with normal ascending aorta diameter (225 patients). PAT was significantly higher in the patients with AAD compared with the patients with normal ascending aorta diameter (0.9 (0.48) vs 0.7 (0.91) mm, *P* < .0001). Univariate and multivariate logistic regression analysis revealed that age (OR: 1.028, 95% CI [1.002-1.054]), BMI (OR: 0.885, 95% CI [0.827-0.947]), gender (OR: 0.381, 95% CI [0.207-0.700]), left ventricular mass index (OR: 1.026, 95% CI [1.008-1.044]) and PAT (OR: 3.005, 95% CI [1.445-6.251]) were associated with AAD.

Conclusions: We measured PAT with TTE for the first time and found a correlation between ascending aorta width and PAT. PAT may be an important follow-up parameter in patients at risk of aortic aneurysm development and it can be measured simply and successfully with TTE.

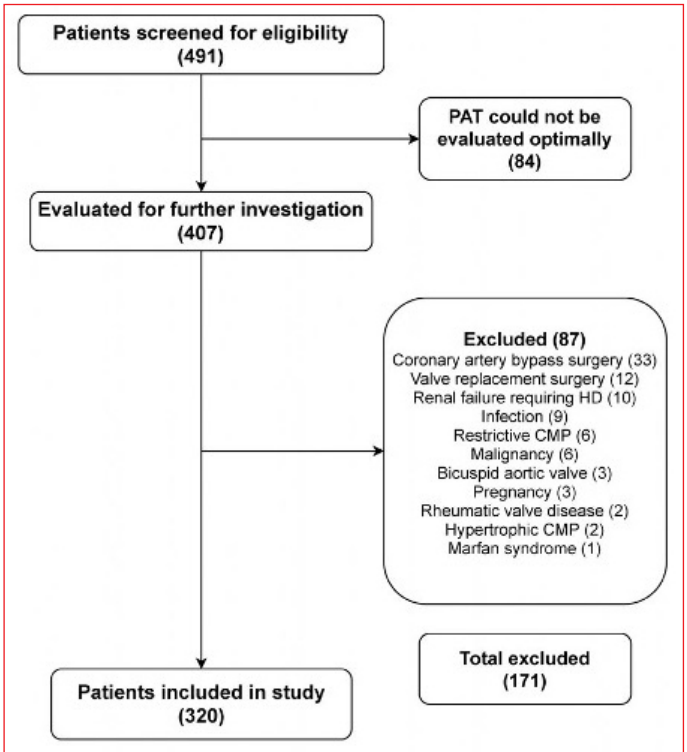

Figure 1. Inclusion flowchart.

Table 1. Baseline characteristics of the study groups with and without aortic dilatation

	Aortic Dilatation No (n = 225)	Aortic Dilatation Yes (n = 96)	*P*
Age (year)	57.3 ± 13.1	64.7 ± 11.7	<0.000
Weight (kg)	80 (17)	67 (17)	<0.000
Height (cm)	165 (11)	159 (12)	<0.000
Body surface area (m^2)	1.9 (0.24)	1.7 (0.22)	<0.000
Obesity, (n, %)	102 (45.3)	28 (29.2)	0.006
Paraaortic adipose tissue, (mm)	0.7 (0.91)	0.91 (0.48)	<0.000
Female gender (n, %)	122 (54.2)	66 (68.8)	0.019
Hypertension (n, %)	148 (65.8)	66 (68.8)	0.698
Diabetes mellitus (n, %)	66 (29.3)	28 (29.2)	1.000
Coronary artery disease (n, %)	29 (12.9)	14 (14.6)	0.721
Hyperlipidemia (n, %)	26 (11.6)	9 (9.4)	0.697
Smoking (n, %)	46 (20.4)	13 (13.5)	0.159
RAS inhibitors (n, %)	112 (49.8)	51 (53.1)	0.627
Statins (n, %)	27 (12)	14 (14.6)	0.584
Calcium channel blockers (n, %)	19 (8.4)	11 (11.5)	0.407
Beta blockers (n, %)	49 (21.8)	24 (25)	0.562
Acetylsalicylic acid (n, %)	52 (23.1)	27 (28.1)	0.396
Left ventricular Hypertrophy (n, %)	36 (16)	38 (39.6)	0.000

Continuous variables are normally distributed showed Mean ± standard deviation; continuous variables are not normally distributed showed as median (interquartile range); categorical variables are presented as number (percentage).

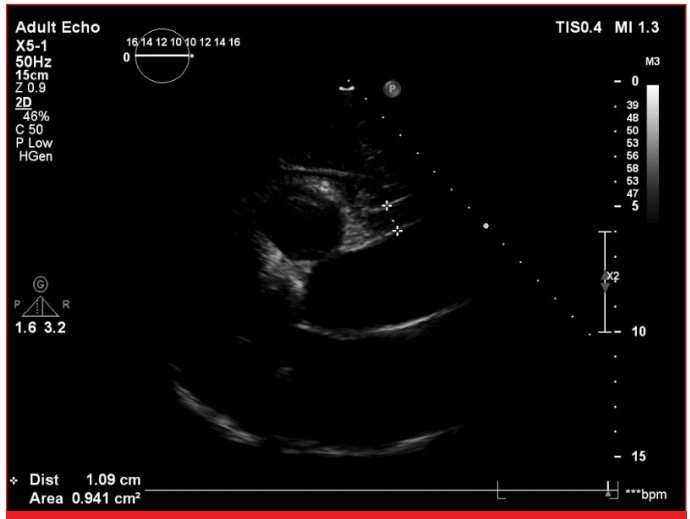

Figure 2. Paraaortic adipose tissue evaluation by transthoracic echocardiography.

Table 2. Laboratory and echocardiographic findings

	Aortic Dilatation No (n = 225)	Aortic Dilatation Yes (n = 96)	*P*
Urea (mg/dL)	30 (14)	32 (16)	0.115
Creatinine (mg/dL)	0.8 (0.3)	0.84 (0.3)	0.232
Aspartate Aminotransferase (U/L)	19 (8)	19 (7)	0.740
Alanine Aminotransferase (U/L)	17.5 (10)	16 (8)	0.112
Glomerular filtration rate (mL/min/1.73m^2)	95 (20)	89 (15.3)	<0.000
Total cholesterol (mg/dL)	188 (50)	190 (47)	0.522
Triglyceride (mg/dL)	135 (81)	132 (84)	0.821
Low-density lipoprotein (mg/dL)	110 (47)	114 (45)	0.465
High-density lipoprotein (mg/dL)	45 (15)	43 (11)	0.571
Glucose (mg/dL)	103 (29)	106 (26)	0.294
Left atrial diameter (mm)	36 (1)	36 (1)	0.030
Left ventricular end-diastolic diameter (mm)	46 (6)	47 (4)	0.630
Left ventricular end-systolic diameter (mm)	28 (5)	28 (5)	0.481
Interventricular septal thickness (mm)	10 (0)	10 (1)	0.017
Posterior wall thickness (mm)	10 (0)	10 (0)	0.166
E (cm/sn)	70 (10)	70 (10)	0.726
A (cm/sn)	80 (15)	83 (10)	0.063
Left ventricular ejection fraction (%)	65 (7.6)	64 (7.2)	0.512
Left ventricular mass index (gr/m^2)	82 (20)	93.5 (22)	<0.000
Left ventricular hypertrophy (n, %)	36 (16)	38 (39.6)	<0.000
Normal left ventricular geometry (n, %)	68 (30.2)	16 (16.7)	0.012
Concentric remodeling (n, %)	121 (53.8)	42 (43.8)	0.113
Concentric hypertrophy (n, %)	13 (5.8)	17 (17.7)	0.001
Eccentric hypertrophy (n, %)	23 (10.2)	21 (21.9)	0.008

Continuous variables are presented as median (interquartile range).

Table 3. Univariate analysis for aortic dilatation

	β	*P*
Age (year)	0.046	<0.0001
Body mass index (kg/m^2)	-0.098	<0.0001
Female gender (%)	-0.619	0.016
Obesity (%)	-0.700	0.007
Para aortic adipose tissue, (mm)	1.020	0.001
Glomerular filtration rate (mL/min/1.73m^2)	-0.011	0.030
Left atrial diameter (mm)	0.081	0.025
LV mass index (gr/m^2)	0.040	<0.000
A (cm/sn)	0.014	0.078
Left ventricle hypertrophy (%)	1.235	<0.000
LV geometry		
Normal	-0.773	0.013
LV geometry		
Eccentric hypertrophy	0.900	0.007
LV geometry		
Concentric hypertrophy	1.255	0.001

LV, left ventricle; β, Regression coefficient.

Table 4. Multivariate analysis for aortic dilatation

	β	OR	95% CI
Age	0.027	1.028	1.002 - 1.054
A	0.013	1.013	0.996 - 1.031
BMI	-0.122	0.885	0.827 - 0.947
Gender	-0.965	0.381	0.207 - 0.700
Para aortic adipose tissue	1.100	3.005	1.445 - 6.251
Left atrial diameter	0.033	1.034	0.931 - 1.148
Left Ventricular Mass Index	0.026	1.026	1.008 - 1.044
Glomerular filtration rate	-0.003	0.997	0.986 - 1.007

CI indicates confidence interval. OR, odds ratio; β, regression coefficient.

Cardiac imaging / Echocardiography

PB-065

Evaluation of the effect of coronary slow flow phenomenon on cardiac functions with cardiac magnetic resonance imaging based deformation imaging

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Background and Aim: Although coronary slow flow phenomenon (CSFP) is seen in 2% of patients undergoing coronary angiography, its clinical significance and impact on ventricular function remain controversial. Cardiac magnetic resonance imaging (CMR) is the gold standard for evaluating ventricular function and volumes. We aimed to assess the impact of CSF on ventricular function by CMR based deformation imaging.

Methods: This is a cross sectional study. 22 people were included in the study. Patients with structural heart disease and secondary coronary slow flow were excluded. 12 subjects with CSFP and 10 subjects with normal flow and normal cardiac function were compared by CMR and CMR strain.

Results: Left ventricle (LV) and right ventricle (RV9) functions and volumes were similar. There was no difference between CMR strains in both groups. Furthermore, there was no correlation between age and heart function in patients with CSF.

Conclusions: CSF has no or limited impact on cardiac functions. Further long-term prospective studies should be carried out to establish the impact and significance of CSF in patients with CSF.

Table 1. General characteristics of study population

Variables	CSF group (n = 12)	Control group (n = 10)	*p*
Age (years)	52.50 ± 9.11	50.60 ± 5.08	0.564
Gender/Male (n, %)	66.7 (8)	70.0 (7)	1.000
BMI (kg/m^2)	30.00 ± 4.84	30.32 ± 3.57	0.865
BSA (m^2)	2.02 ± 0.21	2.06 ± 0.12	0.635
Systolic Blood Pressure (mm Hg)	127.92 ± 13.56	130.50 ± 11.41	0.638
Diastolic Blood Pressure (mm Hg)	79.58 ± 6.89	79.50 ± 7.25	0.978
Heart rate (beats/mn)	72.33± 7.74	69.70 ± 9.14	0.472
Smoking (%, n)	58.3 (7)	50.0 (5)	1.000
Alcohol (%, n)	41.7 (5)	40.0 (4)	1.000
Hypertension (%, n)	41.7 (5)	50.0 (5)	1.000
Diabetes Mellitus (%, n)	25.0 (3)	30.0 (3)	1.000
Hyperlipidemia (%, n)	50.0 (6)	50.0 (5)	1.000

CSF, coronary slow flow; BMI, body mass index; BSA, body surface area.

Table 2. Ventricular volumes and function parameters of study population

Parameters	CSF group (n = 12)	Control group (n = 10)	*P*
LV EDV (mL)	142.17 ± 31.97	157.00 ± 34.40	0.308
LV ESV (mL)	57.67 ± 16.95	60.40 ± 25.48	0.767
LV SV (mL)	83.83 ± 18.83	91.80 ± 23.84	0.391
LV EF (%)	59.58 ± 5.93	60.90 ± 8.29	0.669
LV Mass (gr)	89.25 ± 16.19	101.40 ± 17.77	0.109
RV EDV (mL)	139.17 ± 34.78	156.20 ± 32.00	0.250
RV ESV (mL)	62.50 ± 16.27	65.40 ± 14.61	0.668
RV SV (mL)	76.67 ± 21.01	88.70 ± 26.15	0.245
RV EF (%)	54.92 ± 5.62	57.00 ± 9.08	0.537
LV EDV indexed (mL/m^2)	71.55 ± 13.19	74.26 ± 15.52	0.662
LV ESV indexed (mL/m^2)	28.90 ± 6.81	28.48 ± 10.94	0.915
LV SV indexed (mL/m^2)	42.34 ± 8.97	43.92 ± 12.65	0.429
LV Mass indexed (gr/m^2)	44.84 ± 6.00	48.66 ± 9.44	0.263
RV EDV indexed (mL/m^2)	69.98 ± 14.80	74.01 ± 14.43	0.527
RV ESV indexed (mL/m^2)	31.43 ± 7.05	31.86 ± 7.04	0.887
RV SV indexed (mL/m^2)	38.54 ± 9.20	42.45 ± 13.47	0.429

LV, left ventricle; EDV, end diastolic volume; ESC, end systolic volume; SV, stroke volume; EF, ejection fraction; RV, right ventricle.

Table 3. Cardiac MRI strain parameters

Variables	CSF group (n = 12)	Control group (n = 10)	*p*
Global longitudinal strain	-12.65 ± 1.95	-13.14 ± 2.65	0.631
4 CH longitudinal strain	-12.78 ± 2.53	-13.19 ± 3.02	0.729
3 CH longitudinal strain	-12.73 ± 2.20	-12.43 ± 3.33	0.812
2 CH longitudinal strain	-11.82 ± 1.61	-13.64 ± 3.64	0.138
Global Radial Strain	19.40 ± 4.21	19.92 ± 5.88	0.819

MRI, magnetic resonance imaging; CSF, coronary slow flow; CH, chamber.

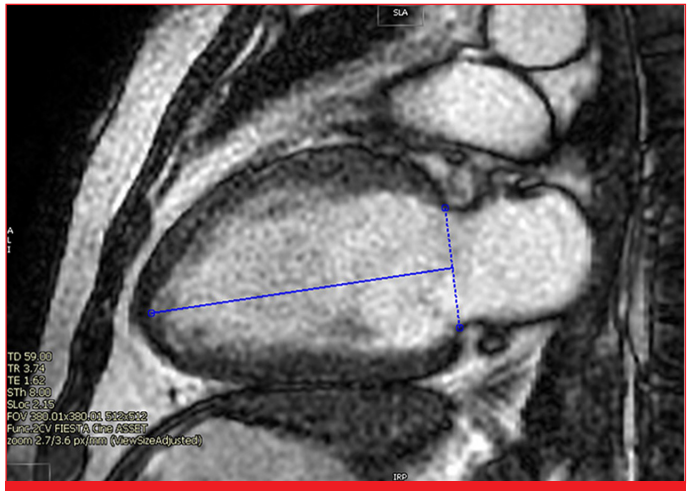

Figure 1. Tissue tracking in CMR.

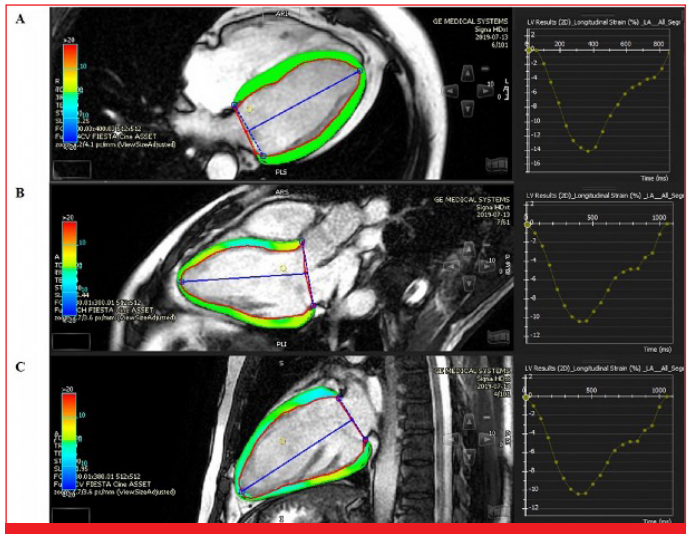

Figure 2. CMR-based deformation imaging to evaluate wall movements.

Cardiac imaging / Echocardiography

PB-066

Assessment of subclinical biventricular myocardial systolic function in COVID-19 patients: A tissue doppler imaging echocardiography study

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Background and Aim: The 2019 novel coronavirus disease (COVID-19) has been reported as pandemy and the number of patients continues to rise. Based on recent data, cardiac injury is a prominent feature of the disease, leading to increased morbidity and mortality.In the present study we aimed to evaluate myocardial dysfunction using transthoracic echocardiography (TTE) and tissue Doppler imaging (TDI) in hospitalized COVID-19 patients.

Methods: We recruited 30 patients (56.7% male, 55.80 ± 14.949 years) who were hospitalized with the diagnosis COVID-19 infection. We analyzed left ventricular (LV) and right ventricular (RV) conventional and TDI parameters at the time of hospitalization and during the course of the disease. Patients without any cardiac disease and with preserved LV ejection fraction (EF) were included. TTE examination was performed and all the variables were recorded and analyzed retrospectively.

Results: We observed that both LV and RV conventional echocardiographic parameters were similar when the day of admission to the hospital was compared to the 5th day of

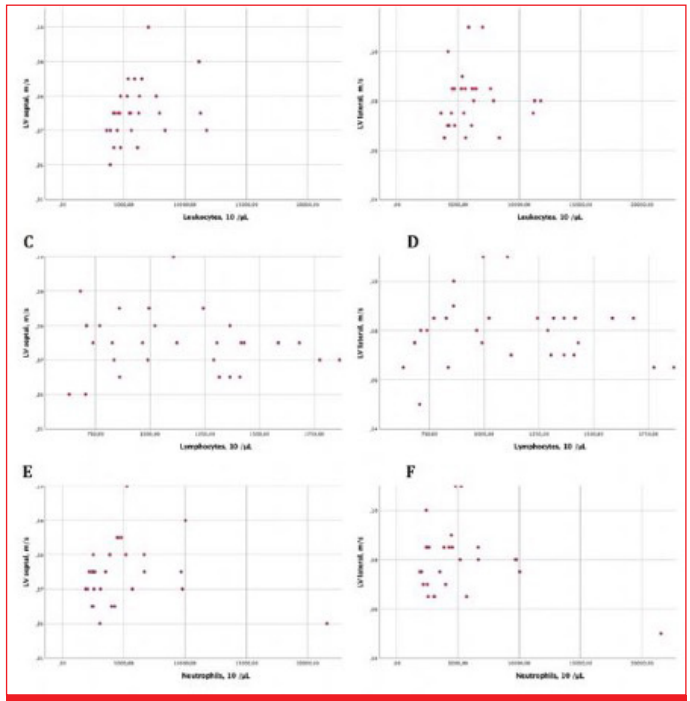

Figure 1. Correlations between some blood cell parameters and TDI measurements. (A) Correlation between LV septal and leukocytes in Covid-19 patients (r=0.226, p=0.23). (B) Correlation between LV lateral and leukocytes in Covid-19 patients (r=0.080, p=0.68). (C) Correlation between LV septal and lymphocytes in Covid-19 patients (r=-0.150, p=0.43). (D) Correlation between LV lateral and lymphocytes in Covid-19 patients (r=-0.032, p=0.87). (E) Correlation between LV septal and neutrophils in Covid-19 patients (r=0.240, p=0.20). (F) Correlation between LV lateral and neutrophils in Covid-19 patients (r=-0.009, p=0.96).

the disease. Regarding TDI analysis, we demonstrated significant impairment in LV septal and lateral deformation (p<0.001). In the correlation analysis no marked correlation was observed between impairment in LV deformation and inflammation biomarkers.

Conclusions: Cardiac involvement is an important feature of the COVID-19 infection but the exact mechanism is stil undefined. Echocardiography is an essential technique to describe myocardial injury and provide new concepts for the possible definitions of cardiac dysfunction.

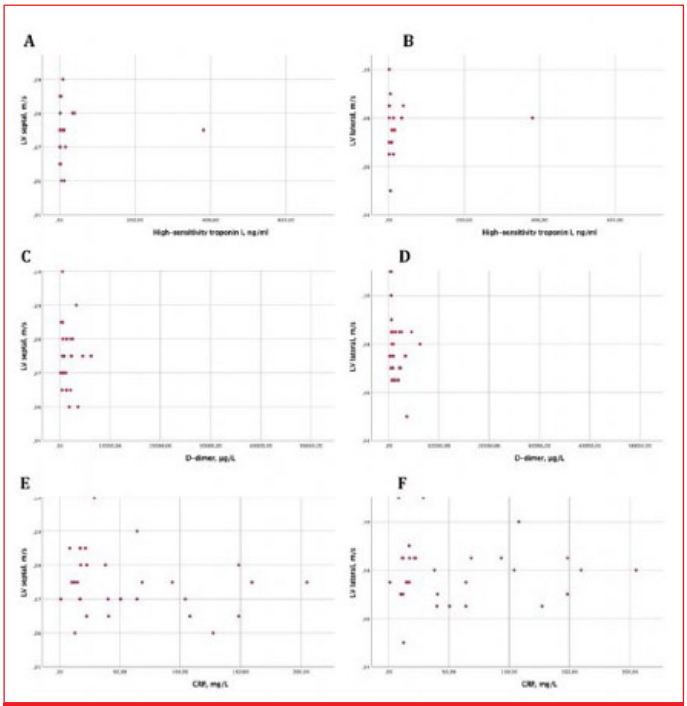

Figure 2. Correlations between some biochemical parameters and TDI measurements. (A) Correlation between LV septal and high-sensitivity troponin in Covid-19 patients (r=-0.027, p=0.91). (B) Correlation between LV lateral and high-sensitivity troponin in Covid-19 patients (r=-0.209, p=0.35). (C) Correlation between LV septal and D-dimer in Covid-19 patients (r=0.005, p=0.98). (D) Correlation between LV lateral and D-dimer in Covid-19 patients (r=0.254, p=0.18). (E) Correlation between LV septal and CRP in Covid-19 patients (r=-0.178, p=0.35). (F) Correlation between LV lateral and CRP in Covid-19 patients (r=-0.065, p=0.73).

Characteristic	Measurement Date		*P* value
	1st day	5th day	
Blood cell count			
Hemoglobin, g	13.18 ± 1.691	12,51 ± 1.596	**.003**
Hct, %	39.47 ± 5.349	38.73 ± 4.182	.41
Leukocytes, 10^3/µL	6233 ± 3520.914	7079 ± 4409.732	.10
Thrombocytes, 10^3/µL	204.43 ± 81.131	252 ± 87.348	**.004**
Lymphocytes, 10^3/µL	1137 ± 475.571	1180.67 ± 478.031	.43
Neutrophils, 10^3/µL	4529.33 ± 3554.711	5108.63 ± 4648.774	.64
Biochemical parameters			
High-sensitivity troponin I, ng/ml	41.30 ± 131.556	97.62 ± 347.356	.49
Ferritin, ng/ml	1513.44 ± 2810.766	1912.09 ± 4466.777	.47
D-dimer, µg/L	3216.50 ± 10266.033	3587.93 ± 10177.555	.76
CRP, mg/L	61.64 ± 65.621	50.86 ± 75.892	.41
Blood pressure			
SBP, mmHg	127.33 ± 4.866	127.33 ± 5.371	1.000
DBP, mmHg	80.33 ± 4.536	79.37 ± 3.846	.14
LVEDD,cm	4.77 ± 0.109	4.76 ± 0.138	.76
LVESD, cm	3.19 ± 0.210	3.18 ± 0.211	.18
IVS thickness, cm	0.99 ± 0.064	0.99 ± 0.066	.32
PW thickness, cm	0.92 ± 0.050	0.93 ± 0.052	.32
LV EF, %	61.33 ± 3.772	61.23 ± 3.720	.08
LA diameter, cm	3.78 ± 0.182	3.78 ± 0.185	.16
RA diameter, cm	3.59 ± 0.241	3.59 ± 0.243	.32
RV EDD, cm	3.40 ± 0.257	3.38 ± 0.258	.16
TAPSE , cm	2.17 ± 0.130	2.15 ± 0.143	.14
Mitral early diastolic velocity (E), m/s	0.81 ± 0.052	0.81 ± 0.058	.11
Mitral late diastolic velocity (A), m/s	0.64 ± 0.079	0.64 ± 0.077	.08
E/A ratio	1.28 ± 0.120	1.27 ± 0.123	.64
TDI early diastolic velocity (é), m/s	7.00 ± 0.571	6.97 ± 0.564	.20
E/é	0.12 ± 0.013	0.11 ± 0.013	.20
Tissue Doppler Imaging parameters			
LV septal systolic velocity, m/s	0.080 ± 0.0100	0.069 ± 0.0088	**<.001**
LV lateral systolic velocity, m/s	0.085 ± 0.0141	0.074 ± 0.0125	**<.001**
RV lateral systolic velocity, m/s	0.142 ± 0.0112	0.140 ± 0.0114	.10

HCT: Hematocrit, CRP: C-reactive protein, LV EDD: Left ventricle end diastolic diameter, LV ESD: Left ventricle end systolic diameter, IVS: Interventricular septum, PW: Posterior wall, LV EF: Left ventricle ejection fraction,

Table 1. Clinical, biochemical and echocardiographic characteristics of patients with COVID-19 in the follow-up (n = 30)
HCT, hematocrit; CRP, c-reactive protein; LV EDD, left ventricle end diastolic diameter; LV ESD, left ventricle end systolic diameter; IVS, interventricular septum; PW, posterior wall; LV EF, left ventricle ejection fraction; LA, left atrium; RA, right atrium; RV EDD, right ventricle end diastolic diameter; TAPSE, tricuspid annular plane systolic excursion; TDI, tissue doppler imaging.

	LV septal		LV lateral	
	r_s	*p*	r_s	*p*
Leukocytes	.226	.23	.080	.68
Lymphocytes	-.150	.43	-.032	.87
Neutrophils	.240	.20	-.009	.96
High-sensitivity troponin	-.027	.91	-.209	.35
D-dimer	-.005	.98	-.254	.18
CRP	-.178	.35	-.065	.73

r_s:Spearman's correlation coefficient

Table 2. Correlations between some biochemical parameters and TDI measurements (n = 30)

Cardiac imaging / Echocardiography

PB-067

Epicardial adipose tissue thickness is higher in right ventricular outflow tract tacchycardia

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Background and Aim: Idiopathic ventricular arrytmias, which occur in the absence of stuctural heart disease, are commonly originated from the outflow tract and 80% of the them arised from the right ventricule. Epicardial adipose tissue (EAT) which, originates from the splanchnopleuric mesoderm, has been shown to be an important source of inflammatory mediators and plays an important role in cardiac outonomic function by epicardial ganglionated plexus (GP). EAT may potentially contribute to the pathophysiology of idiopathic RVOT tachycardia by different mechanisms. In this study, we aimed to investigate the relationship between EAT thickness and RVOT tachycardia.

Methods: This study consisted of 55 patients (32 male, 23 female) with RVOT tachycardia and 60 control subjects (38 male, 22 female). Patients who had more than three consecutive ventricular beats over 100 bpm with specific morphological features (wide range QRS complex, a left bundle branch block, an inferior QRS axis and transition of R dominance after lead V3) in the electrocardiography (ECG) were diagnosed RVOT tachicardia. EAT thickness was measured by transthorasic echocardiography.

Results: EAT thickness was significantly higher in RVOT thachycardia group than in control group respectively 0.914±0.19 cm vs 0.488±0.16 cm (p<0.001). Ejection fraction, posterior wall of left ventricule, interventricular septum were significantly lower and left ventricule end-diastolic diameter, left ventricule end-systolic diameter, and left atrial diameter were significantly higher in patients who had RVOT tachycardia compared to normal subjects (p<0.001).

Conclusions: Patients who were diagnosed with RVOT tachycardia had increased EAT thickness compared to normal subjects. The underlying mechanism of the condition could be mechanical, metabolic, infiltrative or outonomic effects of the EAT.

Table 1. Clinical and demographic findings.

		VT Group (n=55)	Control Group (n=60)	*P*
Sex	Male, n (%)	32 (58%)	38 (63%)	0.57
Sex	Female, n (%)	23 (42%)	22 (37%)	0.57
Age (years)		40.58±12.11	37.57±9.93	0.15
BMI		26.63±4.09	25.09±4.55	0.05

BMI: body mass index

Table 2. Echocardiographic findings in both groups.

	VT Group (n=55)	Control Group (n=60)	*P*
EAT (cm)	0.914±0.19	0.488±0.16	<0.001
EF (%)	55.16±7.85	64.58±3.82	<0.001
LVEDD (cm)	5.01±0.32	4.65±0.28	<0.001
LVESD (cm)	3.4±0.45	2.89±0.25	<0.001
IVS (cm)	0.91±0.08	0.96±0.09	<0.001
PW (cm)	0.82±0.08	0.88±0.09	<0.001
LA (cm)	3.77±0.35	3.39±0.35	<0.001

EAT: epicardial adipose tissue, EF: ejection fraction, IVS: interventricular septum, LA: left atrium; LVEDD: left ventricular end diastolic diameter, LVESD: left ventricular end systolic diameter, PW: posterior wall

Table 3. Complete blood counts measurement for two groups

	VT group (n=55)	Control group (n=60)	*P*
WBC (1/µl)	7.978±1.485	8.923±2.387	0.13
Hg (g/dl)	14.18±1.48	14.08±1.41	0.73
PLT (1/µl)	248.600±63.043	254.066±53.224	0.62
MPV (fL)	8.691±1.08	8.337±0.98	0.70
RDW (%)	14.267±2.35	13.623±0.93	0.06
NEU (1/µl)	4.458±1.101	5.315±1.452	0.01
LYM (1/µl)	2.642±6.10	2.652±1.125	0.95

WBC: white blood cell, Hg: hemoglobin, MPV: mean platelet volume, PLT: platelet RDW: red blood cell distribution width, NEU: neutrophil, LYM: lymphocyte.

Table 4. Biochemical parameters and thyroid stimulating hormone measurements for two groups

	VT group (n=55)	Control group (n=60)	*P*
Urea (mg/dL)	13.32±5.08	12.93±4.24	0.66
Creatinine (mg/dL)	0.84±0.18	0.759±0.15	<0.001
AST (U/L)	24.04±10.36	22.83±4.54	0.43
ALT (U/L)	23.78±14.55	24.63±12.62	0.74
TSH (mIU/l)	1.7023±1.08	1.5631±1.06	0.49
Sodium (mEq/L)	139.509±1.84	141±1.94	<0.001
Potassium (mEq/L)	4.475±0.38	4.153±0.37	<0.001
Glucose (mg/dL)	88.31±11.5	91.5±10.05	0.12

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, TSH: Thyroid stimulating hormone

Cardiac imaging / Echocardiography

PB-068

Comparison of aortic elasticity parameters with transthoracic echocardiography measurements and treadmill exercise test data in healthy adults

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Background and Aim: The elastic properties of the aorta are affected by many conditions, such as age, hypertension (HT), diabetes mellitus (DM), dyslipidemia, and smoking. Studies have shown that increased stiffness in the aortic vessel wall

is associated with increased cardiovascular (CV) mortality and morbidity in various patient groups. In this study; It was aimed to compare aortic elasticity parameters with transthoracic echocardiography (TTE) measurements and treadmill exercise test (TET) data in individuals without known CV disease.

Methods: 105 (mean age 40.19±9.06, 49.5% male) participants who applied to the cardiology outpatient clinic with various complaints or for check-up, had no known CV disease, were scheduled for TTE and TET were included. Patients with known DM, HT, moderate and higher degree of heart valve disease, congenital aortic valve or vascular anomaly, aortic dilatation, rhythms other than sinus and conduction defects on electrocardiography and patients with positive TET were excluded from the study. Aortic elasticity parameters (aortic strain, stiffness, and distensibility) were calculated with the relevant formulas from the M-mode images taken from the ascending aorta, in addition to the traditional measurements of cardiac structure and functions with TTE. These parameters were compared with the data obtained from TET.

Results: There was a significant negative correlation between age and aortic strain and distensibility (r=-0.561, p<0.001; r=-0.553, p<0.001, respectively), and a significant positive correlation with aortic stiffness (r=0.555, p<0.001)). A significant positive correlation was observed between aortic strain and metabolic equivalents (METs), which reflects effort capacity, maximum heart rate reached during exercise test (MHR) and exercise test duration (ETD) (r=0.238, p=0.016; r=0.373, p<0.001; r=0.227, p=0.22, respectively). A significant negative correlation was found between aortic stiffness and METs and MHR (r=-0.201, p=0.043; r=-0.396, p<0.001, respectively). In addition, a significant negative correlation was observed between aortic strain and distensibility and septum thickness, left atrial diameter and E/A values, and a significant positive correlation was observed between aortic stiffness and the same parameters. In the multivariate linear regression analysis, it was determined that all three parameters showed an independent correlation with age.

Conclusions: In our study, a significant correlation was observed between aortic stiffness and strain values and age, exercise capacity and left ventricular diastolic parameters in healthy adults. The data we obtained showed that age is the most important factor affecting the aortic elasticity parameters, and that there is a relationship between these parameters and diastolic functions and cardiac structural changes. Low exercise capacity may also increase CV risk by affecting aortic elasticity.

Parameter	Study group
Age	40.19±9.06
Male (%)	49.5 (n=52)
BSA (m^2)	1.89±0.21
Heart rate (bpm)	74.07±9.11
SBP (mmHg)	109.14±10.03
DBP (mmHg)	68.42±7.14
LDL (mg/dl)	135.39±36.09
HDL (mg/dl)	52.58± 13.97
Total cholesterol (mg/dl)	198.72±38.95
Hiperlipidemi (%)	40 (n=42)
Smoking (%)	15.2 (n=16)

BSA, body surface area; bpm, beat per minute; SBP, systolic blood pressure; DBP, diastolic blood pressure; LDL, low density lipoprotein; HDL, high density lipoprotein.

Table 1. Demographic and laboratory features of the study group

Parameter	Study group
LVEF (%)	60.6±1.77
LVEDD (mm)	45.73±4.52
LVESD (mm)	29.74±3.22
LA diameter (mm)	35.83±1.61
RA diameter (mm)	34.88±1.73
RV diameter (mm)	32.85±2.26
sPAB (mmHg)	22.09±3.13
E wave velocity (cm/s)	80.69±12.92
A wave velocity (cm/s)	65.53±11.38
E/A ratio	1.24±0.20
IVRT (ms)	87.82±7.92
DT (ms)	177.98±17.55
E' (cm/s)	14.36±2.89
E/E' ratio	6.54±7.31
AoS (mm)	29.89±2.84
AoD (mm)	26.1±3.1
Aortic strain (%)	14.62±3.56
Aortic stiffness	3.38±0.91
Aortic distensibility (cm^2.dyne-1.10^3)	7.32±2.09
Treadmill exercise testing data	
METs	9.73±1.34
Percentage of predicted MHR	96.4±13.26
MHR with treadmill testing (bpm)	169.18±16.14
Maximal SBP	144.66±13.66
Maximal DBP	86.85±9.93
Exercise test duration (s)	520.74±93.02
Recovery time (s)	187.14±57.63

LVEF, left ventricular ejection fraction; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end systolic diameter; LA, left atrium; RA, right atrium; RV, right ventricle; sPAB, systolic pulmonary artery pressure; IVRT, isovolumic relaxation time; DT, deceleration time; AoS, systolic aortic diameter; AoD, diastolic aortic diameter; METs, metabolic equivalents; MHR, maximal heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 2. Echocardiographic measurements and treadmill exercise testing data

Parameter		r value	P value
Age			
Aortic strain		-0.561	<0.001
Aortic stiffness		0.555	<0.001
Aortic distensibility		-0.553	<0.001
LDL		0.247	0.021
Total cholesterol		0.252	0.019
E/A ratio		-0.462	<0.001
A velocity		-0.380	<0.001
e' velocity		0.388	<0.001
IVRT		0.213	0.029
LA diameter		0.217	0.026
SBP		0.296	0.002
IVS		-0.323	0.001
MHR with exercise			
Aortic strain			
Age		-0.561	<0.001
BSA		-0.279	0.028
E/A ratio		0.357	<0.001
A velocity		-0.277	0.006
E' velocity		0.279	0.008
IVRT		-0.338	0.001
IVS		-0.394	<0.001
LA diameter		-0.274	0.005
METs		0.238	0.016
MHR with exercise		0.373	<0.001
Exercise test duration		0.227	0.022
Aortic stiffness			
Age		0.555	<0.001
E/A ratio		-0.200	0.048
E' velocity		-0.264	0.013
IVRT		0.281	0.05
IVS		0.239	0.016
LA diameter		0.211	0.034
METs		-0.201	0.043
MHR with exercise		-0.396	<0.001
Aortic distensibility			
Age		-0.553	<0.001
BSA		-0.250	0.012
E' velocity		0.263	0.013
A velocity		-0.265	0.008
E/A ratio		0.273	0.006
IVRT		-0.320	0.001
IVS		-0.319	0.001
LA diameter		-0.329	0.001
MHR with exercise		0.298	0.002

BSA, body surface area; LDL, low density lipoprotein; IVRT, isovolumic relaxation; interventricular septum thickness; LA, left atrium; MHR, maximal heart rate; METs, metabolic equivalents.

Table 3. Statistically significant correlations between the parameters

Cardiac imaging / Echocardiography

PB-069

Echocardiographic features of patients with COVID-19 infection

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Background and Aim: COVID-19 patients with cardiac involvement have a high mortality rate. The aim of this study was to investigate the echocardiographic features in COVID-19 patients between severe and non-severe groups.

Methods: For this single-center study, data from patients who were treated for COVID-19 between March 25, 2020 and April 15, 2020 were collected. Two-dimensional echocardiography (2DE) images were obtained for all patients. Patients were divided into two groups based on the severity of their COVID-19 infections. 2DE parameters indicating right ventricular (RV) and left ventricular (LV) functions were compared between the two groups.

Results: A total of 90 patients hospitalized for COVID-19 were included in this study. The mean age of the severe group (n=44) was 63.3±15.7 years, and 54% were male. The mean age of non-severe group (n=46) was 49.7±21.4 years, and 47% were male. In the severe group, RV and LV diameters were larger (RV, 36.6±5.9 mm vs 33.1±4.8 mm, p=0.003; LV 47.3±5.8 mm vs. 44.9±3.8 mm, p=0.023), the LE ejection fraction (LVEF) and the RV fractional area change (RV-FAC) were lower (LVEF, 54.0±9.8% vs 61.9±4.8%, p<0.001; RV-FAC, 41.4±4.1% vs 45.5±4.5%, p<0.001), and pericardial effusions were more frequent (23% vs 0%) compared to patients in the non-severe group. A multiple linear regression analysis determined that LVEF, right atrial diameter, high-sensitivity troponin I, d-dimer, and systolic pulmonary artery pressure, were independent predictors of RV dilatation.

Conclusions: The results demonstrate that both right and left ventricular functions decreased due to COVID-19 infection in the severe group. 2DE is a valuable bedside tool and may yield valuable information about the clinical status of patients and their prognoses.

	Severe (n = 44)	Non-severe (n = 46)	*p*
Age (years)	63.3 ± 15.7	49.7 ± 21.4	0.001
Male, n(%)	24 (54%)	22 (47%)	0.524
BMI (kg/m^2)	30.1 ± 5.6	28.1 ± 5.3	0.224
HT, n(%)	23(52%)	9(19%)	0.001
DM, n(%)	10(22%)	4(8%)	0.066
HLD, n(%)	7(15%)	3(6%)	0.196
Smoking, n(%)	26(%59)	25(%55)	0.754
HR, beats/min	78.1±13.8	72.9±12.6	0.072
SBP (mmHg)	104.4±10.9	111.3±11.0	0.093
DBP (mmHg)	70.9±6.4	69.7±8.4	0.464
Laboratory findings on admission			
Hemoglobin(g/dl)	11.3 ± 2.3	13.5 ± 1.8	<0.001
WBC (10^3 /µl)	7.0(5.2-12.0)	5.4(3.6-7.3)	0.005
Creatinine (mg/dl)	0.8(0.6-1.2)	0.8(0.6-0.9)	0.563
Sodium (mmol/L)	139.3 ± 6.4	137.3 ± 2.7	0.070
Potassium (mmol/L)	4.0 ± 0.6	4.2 ± 0.4	0.239
Glucose (mg/dL)	147.9± 60.8	117.8± 40.2	0.008
CRP (mg/dL)	102(40-188)	20(10-81)	<0.001
Hs-TnI (pg/ml)	20(5-86)	9(3-16)	0.004
D-dimer (ng/mL)	1170(330-2840)	255(27-510)	<0.001
CK-MB (ng/mL)	2.2(1.1-3.8)	1.3(0.9-2.3)	0.083
O_2 saturation, %*	87.5±3.6	95.5±1.9	<0.001
Clinical parameters			
ICU, n (%)	29(65%)	-	-
MV, n (%)	24(54%)	-	-
Hospital stay**(days)	12.2±4.3	8.0±4.3	<0.001
Pneumonia on CT, n(%)	43(97%)	37(80%)	0.009

Abbreviations: BMI, Body mass index; HT, hypertension; DM, diabetes mellitus; HLD, hyperlipidemia; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; WBC, white blood cell, CRP, C-reactive protein; hs-TnI, high sensitive troponin I; CK, creatinine kinase; ICU, intensive care unit; MV, mechanical ventilation; CT, computed tomography.

Table 1. Demographic and clinical characteristics of patients severe and non-severe

Table 2: Comparison of 2-D transthoracic echocardiographic parameters in the study population

	Non-severe (n=46)	Severe (n=44)	Severe exc. MV (n=20)	p[1]	p[2]
Left heart findings					
LVEF (%)	61.9 ± 4.8	54.0 ± 9.8	58.7 ± 7.4	<0.001	0.041
LVEDD (mm)	44.9 ± 3.8	47.3 ± 5.8	48.3 ± 5.2	0.023	0.005
LVESD (mm)	28.8± 4.1	33.1 ± 6.7	32.8 ± 6.5	0.001	0.004
LV mass, g/m^2	92.9±4.5	95.0±6.2	94.2±5.4	0.076	0.333
LA (mm)	34.6±5.5	39.4±5.5	38.8±5.2	<0.001	0.005
E(cm/s)	90.6±25.4	67.8±13.6	66.5±13.6	<0.001	<0.001
A(cm/s)	69.7±16.2	74.8±14.6	69.5±13.6	0.130	0.965
E/A ratio	1.2±0.5	0.9±0.3	1.0±0.4	<0.001	0.041
Right heart findings					
RV (mm)	33.1±4.8	36.6±5.9	36.7±5.2	0.003	0.010
RV ≥42 mm, n(%)	2(4%)	11(25%)	3(15%)	0.007	0.159
RA (mm)	36.8±6.6	39.9±7.3	41.7±5.9	0.023	0.007
TAPSE (mm)	21.4±3.6	20.1±4.3	20.3±4.3	0.126	0.288
TAPSE ≤ 16 mm, n(%)	4(8%)	11(25%)	3(15%)	0.016	0.425
RV-FAC, %	45.5±4.5	41.4±4.1	41.5±3.4	<0.001	0.001
TDI S', cm/s	13.8±3.0	13.1±3.0	13.3±3.6	0.324	0.585
PA, mm	21.3±3.0	21.3±3.0	21.4±4.9	0.413	0.976
sPAP, mmHg	28.5±7.3	35.5±8.6	32.0±9.3	0.039	0.117
sPAP ≥35 mmHg, n(%)	6(14%)	17(38%)	8(40%)	0.013	0.027
IVC (mm)	12.5±2.6	16.8±5.0	12.8±3.5	<0.001	0.689
Pericardial effusion, n(%)	0(0%)	10(23%)	1(5%)	-	-

Abbreviations: MV, mechanical ventilation, LVEF, left ventricular ejection fraction; LVEDD, left ventricular end diastolic diameter; LVESV, left ventricular end systolic diameter; LA, left atrial; MR, mitral regurgitation; RV, right ventricular; RA, right atrial; TAPSE, tricuspid annular plane systolic excursion; RV-FAC, right ventricular fractional area change; TDI S', tissue Doppler imaging systolic wave S' velocity; PA, pulmonary artery; TR, tricuspid regurgitation; sPAP, systolic pulmonary artery pressure; IVC, inferior vena cava.

Table 2. Comparison of 2-D transthoracic echocardiographic parameters in the study population

Coronary Artery Disease / Acute Coronary Syndrome

PB-070

Vasoactive-Inotropic Score as a Predictor of Mortality in Acute Myocardial Infarction complicated with Cardiogenic Shock

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Background and Aim: The vasoactive-inotropic score (VIS) represents the weighted sum of the inotropic and vasoconstrictor agents administered, reflecting the medical support of the cardiovascular system. We aimed to assess if VIS has a predictive value for mortality in cardiogenic shock.

Methods: 45 episodes of 44 patients diagnosed as a cardiogenic shock secondary to acute myocardial infarction were included in the study. VIS values were calculated for each episode in the initiation and at the first 24 th hours after inotrope commencement. The highest VIS value was also recorded. The primary outcome was mortality.

Results: Cardiogenic shock episodes were divided into two groups according to the outcome as a survivor (n=20) and non-survivor (n: 24). The area under the curve of VIS values for the prediction of mortality VIS 24th hour (0.762; p<0.001), VIS0 (0.762; p=0.001). At the cut-off value of 45, the sensitivity, specificity, positive and negative predictive values of VIS 24th for mortality were 81.4%, 71.8%, 81.4% and 71.8%, respectively. Patients with VIS greater than 45 at 24th had a significantly higher risk of mortality (p<0.001, OR: 11.8, 95% CI (5.9 - 30.6)).

Conclusions: VIS value calculated anytime after initiation of inotropy treatment may be useful for mortality prediction in cardiogenic shock. VIS values after initiation of inotropic agents could help clinicians to identify poor prognosis of acute myocardial infarction complicated with cardiogenic shock.

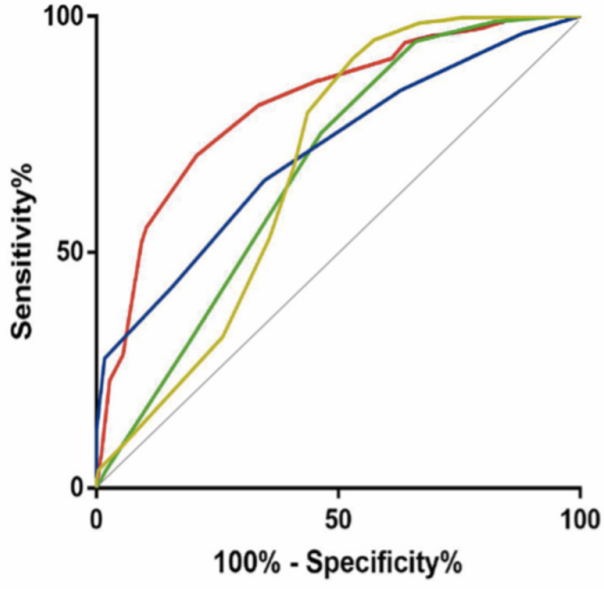

Figure 1. Ability to predict mortality at the cut â€“ off value of 45

Coronary Artery Disease / Acute Coronary Syndrome

PB-071

Blood pressure index predicts right ventricular dysfunction in anterior myocardial infarction

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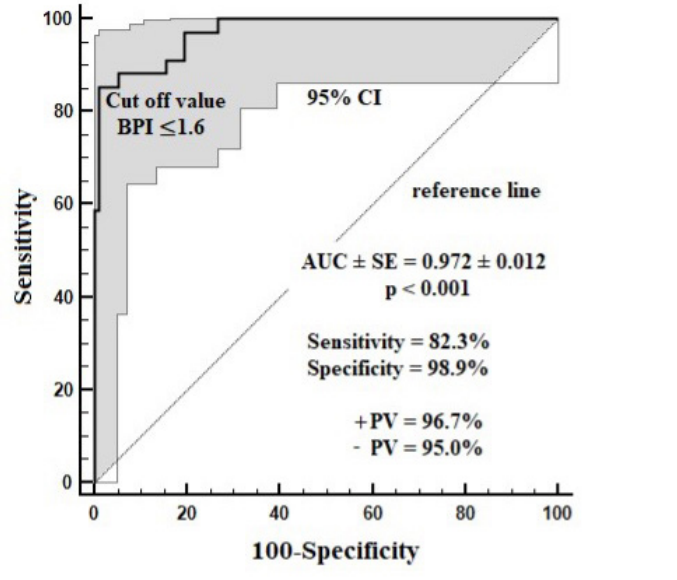
Background and Aim: Right ventricular dysfunction (RVD), which is often neglected in anterior ST-segment elevation myocardial infarction (STEMI), has an important role in the pathology of heart failure in the post-STEMI period. This study aims to investigate the role of blood pressure index (BPI) in detecting RVD in acute anterior STEMI.

Methods: A total of 131 patients without known coronary artery disease with first anterior STEMI and treated with primary percutaneous coronary intervention were included

in this study. Tricuspid annular plane systolic excursion was used to screen for RVD and ≤17 mm was considered RVD (n=34). The BPI was obtained by dividing systolic blood pressure (SBP) by diastolic blood pressure (DBP).

Results: Mean DBP (73±13 mm Hg vs 66±11 mm Hg, p=0.006, respectively) was found to be higher in RVD patients compared to those without RVD, whereas mean SBP and mean BPI (116±18 mm Hg vs 127±19 mm Hg, p=0.004; 1.5±0.1 vs 1.9±0.2, p<0.001, respectively) was lower. The diagnostic performance of BPI in predicting RVD was evaluated by ROC Curve analysis. According to this, the cut-off value of BPI was found to be ≤1.6, with 82.3% sensitivity and 98.9% specificity in predicting RVD (AUC ± SE= 0.972±0.012; p< 0.001; positive predictive value of 96.7%; negative predictive value of 95.0%. In-hospital mortality was significantly higher in the RV dysfunction group (26.5% vs. 5.1%, p<0.001). BPI ≤1.6 was found in 66.6% (n=7) of the patients who died with RV dysfunction.

Conclusions: We found that BPI was an index with high positive predictive value and negative predictive value in detection of RVD in acute anterior STEMI. As RVD plays an important role in the prognosis of post-STEMI, it shows that the functions of the right ventricle should be considered as in the left ventricle. Therefore, BPI can be easily used to identify patients at risk for RVD and to predict prognosis.



Abbreviations: AUC: Area Under the Curve, BPI: Blood pressure index, CI: Confidence interval, SE: Standart error, +PV: Positive predictive value, -PV: Negative predictive value

Figure 1. Diagnostic performance evaluation of BPI in predicting RVD in anterior STEMI patients

Variables	Right ventricular dysfunction		p
	No	Yes	
	n = 97	n = 34	
Male gender, n (%)	82 (84.5)	30 (88.2)	0.600
Age, years	57.6 ± 18.2	59.3 ± 16.7	0.633
Active smoking, n (%)	46 (47.4)	18 (52.9)	0.582
History, n (%)			
Hypertension	31 (31.9)	12 (35.3)	0.717
Diabetes mellitus	20 (20.6)	8 (23.5)	0.724
Hyperlipidemia	7 (7.2)	3 (8.8)	0.763
At admission			
cTn-I, ng/l	12.5 (5.9-40.6)	15.4 (6.1-37.3)	0.677
Creatinine, mg/dl	1.0 ± 0.3	1.0 ± 0.2	0.999
Total cholesterol, mg/dl	188.2 ± 27.3	190.3 ± 32.1	0.713
LDL, mg/dl	108.1 ± 25.5	113.5 ± 29.2	0.308
Hemoglobin, gr/dl	13.3 (2.3)	13.8 (2.3)	0.463
SBP (mmHg)	130 ± 28	116 ± 23	0.010*
DBP (mmHg)	65 ± 16	73 ± 15	0.012*
BPI	1.9 ± 0.3	1.6 ± 0.3	<0.001*
Pre-infarction angina, %	28 (28.9)	11 (32.4)	0.702
Door-to-balloon time, min	44.5 ± 10.5	46.0 ± 11.3	0.484
Symptom-to-balloon time, min	330.3 ± 80.2	356.0 ± 90.7	0.123
Heart rate, beats/min	80.2 ± 15.6	83.4 ± 17.2	0.318
Medication, n (%)			
ACE	92 (94.8)	31 (91.2)	0.453
Beta-blockers	95 (98.0)	32 (94.1)	0.254
Statins	91 (93.8)	32 (94.1)	0.950
In hospital mortality, n (%)	5 (5.1)	9 (26.5)	<0.001*

Table 1. Baseline characteristics of patients with and without right ventricular dysfunction after first anterior STEMI
***p<0.05 is considered significant for statistical analyses. Categorical variables were shown in numbers and percentage, numerical variables were shown as mean ± standard deviation. Abbreviations: SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BPI: Blood pressure index, ACE: Angiotensin-converting enzyme inhibitors**

Coronary Artery Disease / Acute Coronary Syndrome

PB-072

Prognostic Value of SYNTAX and Culprit-SYNTAX Score in MI Patients Presenting with Cardiogenic Shock and Cardiac Arrest

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Background and Aim: The SYNTAX score (SS) is an anatomical scoring system developed to show the extent of coronary artery disease (CAD) angiographically (CAG) and to decide on the choice of rvascularization method. Parameters such as the vessel in which the lesion is located, the number of lesions, the presence of the lesion in the proximal or distal regions of the vessel, and whether it is a bifurcation or chronic total occlusion lesion are used to calculate the SS.It has also been shown that SS can predict cardiac death, myocardial infarction (MI), and target lesion revascularization prognostically in both stable CAD and MI patients. It has been shown that culprit-only PCI has better and safer results than multivessel percutaneous coronary intervention (PCI) in patients presenting with cardiogenic shock. Based on these data, in our study, the SS and SYNTAX scores of culprit lesions (cul-SS) were examined in terms of in-hospital mortality in patients who had MI and were admitted to the hospital with cardiogenic shock or cardiac arrest.

Methods: Patients with cardiogenic shock due to MI or cardiac arrest who underwent successful cardiopulmonary resuscitation at the hospital were included in the study. Patients with a history of coronary artery bypass grafting (CABG)

were not included in the study. SS and cul-SS were calculated from the CAGs of the patients. In-hospital mortality of the patients was recorded and comparisons were made according to both scoring systems.

Results: The median age of 110 patients included in the study was 60 (interquartile range (IQR) 53.25-69) years and the majority were male patients (74.5%). The majority of the patients had no known history of CAD (83.6%) and presented with a diagnosis of STEMI (87.3%). When the CAG data of the patients were examined, it was observed that culprit LMCA was 7.3%, LAD was 53.6%, CX was 10.9%, RCA was 28.2%, and bifurcation lesion was 10.9%. The median SS of the patients was calculated as 19.25 (IQR 11.37-22.50) and cul-SS median 12.25 (IQR 7-19.5). DES stents (90%) were used for treatment in the vast majority of patients. In-hospital death was observed in 43 (39.1%) patients. The data are summarized in Table 1. The Roc-curve analysis performed is summarized in Figure 1.

Conclusions: Our results of this study show that the cul-SS score has more potent predictive power than the SS in demonstrating in-hospital deaths in patients admitted to hospital with cardiogenic shock or cardiac arrest due to MI.

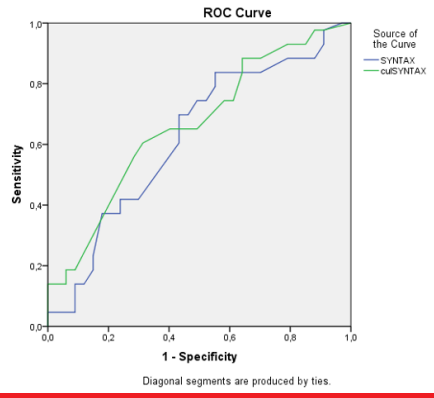

Figure 1. Roc-Curve Analysis of Scoring Systems
SS: AUC: 0.623, %95 CI: 0.516-0.730, p: 0.030 Cul-SS: AUC: 0.662, 595 CI: 0.558-0.766, p:0.004

Table 1. Clinical and Angiographic Parameters

Parameters	Value
Age (Median 25-75% IQR)	60 (53.25-69)
Gender (Male, %)	82 (74.5)
Clinic on admission (STEMI)	96 (87.3%)
Cardiac Arrest on admission	72 (65.5%)
Cardiogenic shock on admission	38 (34.55)
Coronary Artery Disease history	18 (16.4%)
Stent type (DES, %)	99 (90)
Bifurcation lesions (%)	12 (10.9)
Culprit LAD (%)	59 (53.6)
Culprit LMCA (%)	8 (7.3)
Culprit CX (%)	12 (10.9)
Culprit RCA (%)	31 (28.2)
SYNTAX score (Median 25-75% IQR)	19.25 (11.37-22.50)
Cul-SYNTAX score (Median 25-75% IQR)	12.25 (7-19.50)
In-hospital mortality (%)	43 (39.1)

Coronary Artery Disease / Acute Coronary Syndrome

PB-073

The Effect of Total Occlusion Pattern on in Hospital and Long Term Clinical Outcomes in Acute ST Segment Elevated Myocardial Infarction

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Background and Aim: The aim of our study is to determine the relationship between the total occlusion pattern and in hospital no-reflow development and long-term mortality in patient admitted to our hospital with STEMI.

Methods: This study was designed as a single center cross-sectional study. Study population was chosen from patients older than 18 years old who admitted to emergency department of Dr Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital between March 2017 and February 2019 with symptoms suggesting myocardial ischemia who diagnosed as STEMI 1541 patients met these criteria and were enrolled to the study. Primary end points include no-reflow development after PCI and long term death.

Results: In the study, it was found that the no-reflow rate in the hospital was 10% (153 patients), and the mortality rate in the long-term follow up (median 19 months) was 7% (98 patients). In order to evaluate the relationship between no-reflow and subtotal occlusion in logistic regression analysis (OR;0.175, 95% CI;0,060–0,505, p=0.0016). In addition, there was a significant relationship between no-reflow and age and total ischemia time (OR;1.903, 95% CI;1.18-3.046 and OR;1.091, 95% CI;1.029-1.156)

To evaluate the relationship between long-term mortality and candidate predictors, there was no significant relationship between total occlusion types and long-term mortality in Cox proportional hazard regression analysis. (HR;0.881, 95% CI;0.368–2.106 p=0.5711). Among the independent predictors of long-term mortality, a significant relationship was observed between age, duration of ischemia and previous MI..(HR;2.47, 95% CI;1.606-4.699, HR;1.082, 95% CI;1.026-1.140 and HR;2.752 95% CI;1.366-5.545)

Conclusions: We found that in patients presenting with STEMI, the total occlusion pattern was associated with in hospital no-reflow phenomenon, but not with long-term mortality.

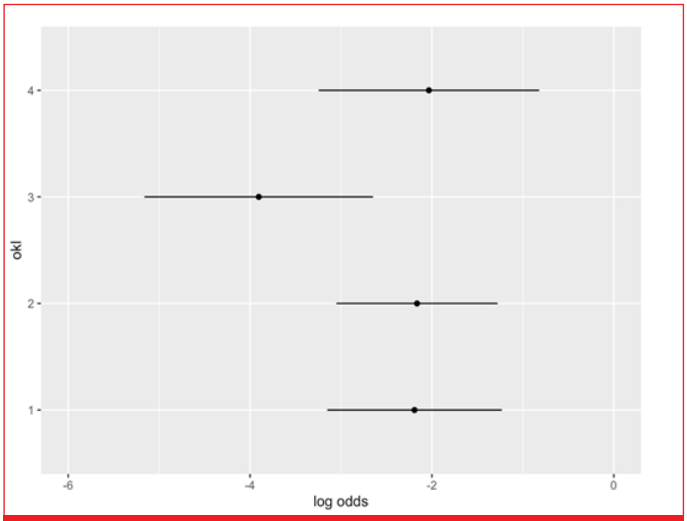

Figure 1. Log Odds Graph Between Total Occlusion Types and No-reflow
A significant correlation was found between subtotal occlusion and no-reflow, and the no-reflow decreased significantly in the subtotal occlusion group.

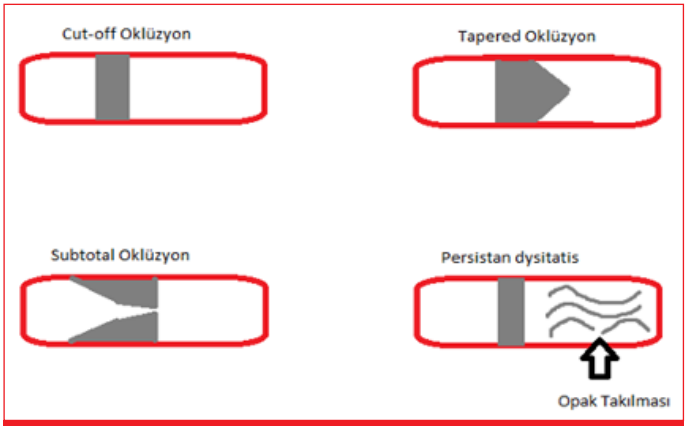

Figure 2. Total Occlusion Patterns
Total occlusions of the patients who were found to have total occlusion in their coronary angiography were divided into 4 classes.

Table 1. Independent Predictors Of No-reflow and Logistic Regression Analysis Table

Variable	Odds Ratio (OR)	%95 confidence interval	*P*
Age	1.903	(1.18 – 3.046)	0.0073
Gender	0.813	(0.37 – 1.769)	0.6033
HT	0.740	(0.379 – 1.444)	0.3779
DM	1.319	(0.679 – 2.563)	0.4132
Smoke	0.785	(0.375 – 1.641)	0.5206
Total ischemia time	1.091	(1.029 – 1.156)	0.0035
MI history	1.656	(0.823 – 3.331)	0.1569
Total occlusion-typ 1:2	0.972	(0.496 – 1.906)	0.9364
Total occlusion-typ 3:2	0.175	(0.060 – 0.505)	0.0016
Total occlusion-typ 4:2	1.139	(0.420 – 3.089)	0.7605

type 1: cut-off occlusion, type 2: tapered occlusion, type 3: subtotal occlusion, type 4: persistan dyasitasis occlusion, HT: hypertension, DM: diabetest, MI: myocardial infarction

Coronary Artery Disease / Acute Coronary Syndrome

PB-074

Evaluation of frontal plane QRS-T angle in patients with slow coronary flow

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Background and Aim: Frontal plane QRS-T angle is a novel marker of myocardial repolarization, and an increased frontal plane QRS-T angle is associated with adverse cardiac outcomes. Slow coronary flow may cause fatal cardiac arrhythmias by causing electrical abnormalities and altering ventricular repolarization. We aimed to evaluate the frontal plane QRS-T angle in patients with slow coronary flow.

Methods: A total of 60 consecutive patients with slow coronary flow and 60 consecutive patients with normal coronary flow were enrolled into the study. Laboratory and some electrocardiography parameters including frontal plane QRS-T angle were compared between the two groups

Results: We have found that the angle of frontal QRS-T was higher in the group with slow coronary flow (p<0.001). In addition, there was a negative correlation between thrombolysis in myocardial infarction frame count and frontal QRS-T angle (r=-0.496, p<0.001).

Conclusions: In conclusion, increased frontal plane QRS-T angle might be an important indicator of slow coronary flow

Table 1. Baseline characteristics of the study groups (n=120)

Parameters	Patients with NCF (n=60	Patients with SCF (n=60)	*P*
Age, years	54.9 ± 9.5	55.8 ± 8.9	0.566
BMI, kg/m^2	27.5 ± 3.3	27.3 ± 4.0	0.752
Female, n, %	24 (40.0)	25 (41.7)	0.853
Diabetes Mellitus, n, %	9 (15.0)	10 (16.7)	0.803
Hypertension, n, %	20 (33.3)	19 (31.7)	0.845
Dyslipidemia, n, %	19 (31.7)	22 (36.7)	0.564
Family history, n, %	7 (11.7)	11 (18.3)	0.306
Smoking, n, %	22 (36.6)	33 (55.0)	0.044

Data are given as mean ± SD, n or median (interquartile range). BMI, Body mass index; LVEF, left ventricle ejection fraction; NCF, normal coronary flow; SCF, slow coronary flow.

Table 2. Comparisons of laboratory findings, TIMI frame counts and ECG parameters.

Parameters	Patients with NCF (n=60)	Patients with SCF (n=60)	*P*
Glucose, mmol/L	6.40 ± 2.45	6.78 ± 3.31	0.490
Creatinine, µmol/L	86.6 ± 17.7	92.8 ± 35.3	0.266
Uric acid, mmol/L	0.35 ± 0.12	0.33 ± 0.10	0.580
WBC count, 10⁹/ L	9.82 ± 2.43	10.29 ± 2.57	0.269
Hemoglobin, g/dL	13.4 ± 1.8	13.7 ± 1.5	0.255
Platelet count, 10⁹/ L	236.4 ± 62.4	231.2 ±56.8	0.671
Total cholesterol, mmol/L	4.77 ± 2.06	4.95 ± 2.00	0.615
Triglyceride, mmol/L	1.40 (0.90-2.15)	1.39 (0.88-1.82)	0.683
LDL-cholesterol, mmol/L	2.93 ± 1.48	3.01 ± 1.52	0.790
HDL-cholesterol, mmol/L	1.06 (0.87-1.24)	1.13 (0.91-1.27)	0.820
Hs-CRP, mg/L	3.1 (1.2- 4.6)	4.9 (2.5- 6.5)	0.030
LVEF, %	58 ± 5	58 ± 5	0.599
TFC-LAD	39 ± 10	17 ± 4	<0.001
TFC-Cx	28 ± 7	12 ± 5	<0.001
TFC-RCA	29 ± 7	12 ± 4	<0.001
TFC-mean	32 ± 6	14 ± 4	<0.001

Data are given as mean ± SD, n or median (interquartile range). HDL, high density lipoprotein; Hs-CRP, high-sensitivity C-reactive protein; LDL, low-density lipoprotein; LVEF, left ventricle ejection fraction; NCF, normal coronary flow; SCF, slow coronary flow; TFC, TIMI, thrombolysis in myocardial infarction frame count; WBC, white blood cells.

Table 3. Comparisons of ECG parameters.

Parameters	Patients with NCF (n=60)	Patients with SCF (n=60)	*P*
QT, ms	376 ± 25	368 ± 30	0.223
QTc, ms	400 ± 15	405 ± 47	0.497
Tpe, ms	72 ± 6	86 ± 10	<0.001
Tpe/QT	0.23 ± 0.11	0.19 ± 0.11	<0.001
Tpe/QTc	0.21 ± 0.99	0.18 ± 0.11	<0.001
f(QRS)-T (°)	46 ± 36	69 ± 51	<0.001

QTc - corrected QT interval; SCF - slow coronary flow; f(QRS)-T; frontal QRS-T angl

Coronary Artery Disease / Acute Coronary Syndrome

PB-075

The predictive value of MELD-XI score for short- and long-term mortality in elderly patients with Non-ST elevation myocardial infarction

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Background and Aim: The goal of this study was to investigate the utility of the Model for End-stage Liver Disease excluding INR (MELD-XI) score in predicting short- and long-term mortality in older patients with non-ST elevation myocardial infarction (NSTEMI) who underwent coronary angiography (CAG).

Methods: In total, 228 consecutive elderly NSTEMI patients who were above 75 years old were analyzed in the study. The modified 5-item frailty index and the Charlson Comorbidity Index were used to assess frailty and comorbidities. The MELD-XI score was calculated using the logarithmic relationship between the serum creatinine and total bilirubin.

Results: The median long-term follow-up was 530 (interquartile range (IQR)=303-817) days and the short- and long-term mortality rates were 11.8% (n=27) and 16.4% (n=33), respectively. Patients who did not survive had a substantially higher MELD-XI score than those who did [10.1 (IQR = 7.8-15.1) vs 4.5 (IQR=1.9-6.9), p<0.001, respectively]. Multivariable Cox regression analyses indicated that MELD-XI score predicted both short- and long-term mortality independently (HR: 1.076, 95%CI: 1.028-1.126, p=0.002 and HR: 1.053, 95%; CI: 1.006-1.103, p=0.025). When the MELD-XI score, serum creatinine, and total bilirubin AUC values were compared to predict long-term mortality, the MELD-XI score had the highest value (AUC: 0.833), followed by serum creatinine (AUC: 0.741) and total bilirubin (AUC: 0.743). (AUC: 0.723).

Conclusions: This was the first investigation to indicate that elderly NSTEMI patients with a high MELD-XI score had poor prognosis in the short- and long-term period.

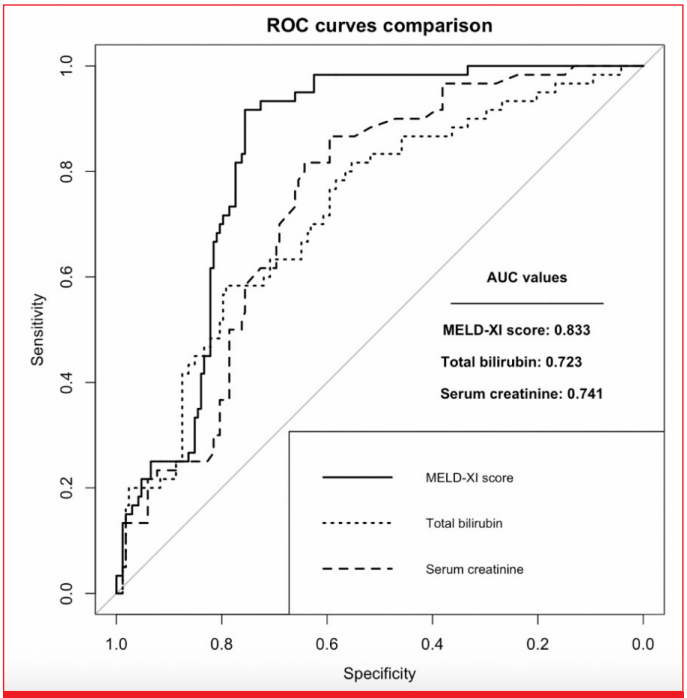

Figure 1. Receiver operating characteristic (ROC) curve analyses of the Model for End-stage Liver Disease excluding INR (MELD-XI) score, serum creatinine, and total bilirubin to predict long-term mortality

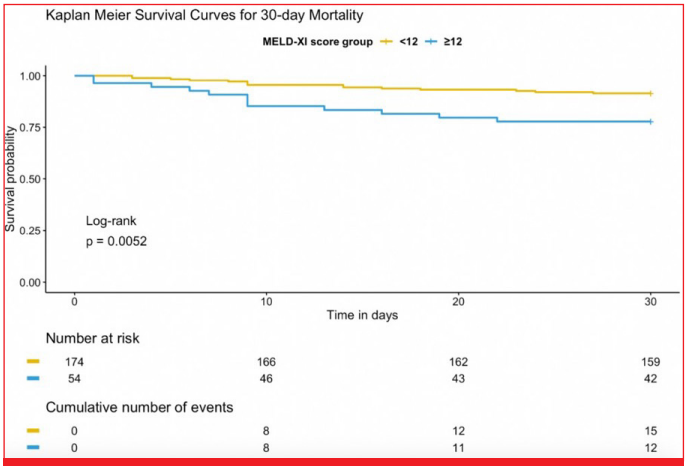

Figure 2. Short-term Kaplan Meir survival analysis of elderly non-ST elevation myocardial infarction (NSTEMI) cases according to the Model for End-stage Liver Disease excluding INR (MELD-XI) score

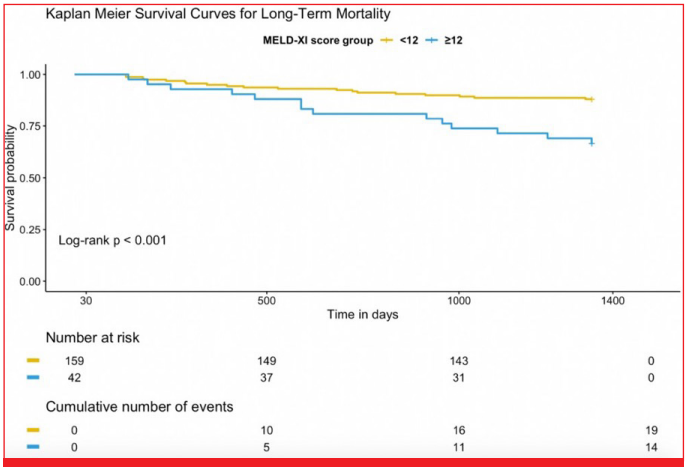

Figure 3. Long-term Kaplan Meir survival analysis of elderly non-ST elevation myocardial infarction (NSTEMI) cases according to the Model for End-stage Liver Disease excluding INR (MELD-XI) score

Coronary Artery Disease / Acute Coronary Syndrome

PB-076

Non-alcoholic fatty pancreas disease is associated with SYNTAX score in acute coronary syndrome

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Background and Aim: The main aim of this study was to investigate the relation between nonalcoholic fatty pancreas disease (NAFPD) and coronary artery lesion complexity and severity assessed using the SYNTAX score (SXscore) in acute coronary syndrome (ACS) patients.

Methods: A total of 99 patients with a first-time diagnosis of acute coronary syndrome (ACS) were consecutively enrolled. Non-alcoholic fatty pancreas disease (NAFPD) was evaluated by using transabdominal ultrasonography (TUS). SYNTAX score (SXscore) was calculated using the SXscore algorithm.

Results: The SXscore was higher in the NAFPD presence group than in the Non-alcoholic fatty pancreas disease (NAFPD) absence group (12.3±6.4 and 8.2±4.3, p<0.001). Univariate analysis showed that hypertension (p=0.033) and presence of NAFPD (p=0.001) were associated with increased SXscore. In addition, multivariate analysis demonstrated that presence of Non-alcoholic fatty pancreas disease (NAFPD) (p=0.002) was associated with increased SYNTAX score (SXscore).

Conclusions: Presence of NAFPD detected by transabdominal ultrasonography (TUS) in acute coronary syndrome (ACS) patients may be a warning signal of complexity and severity of coronary artery disease (CAD).

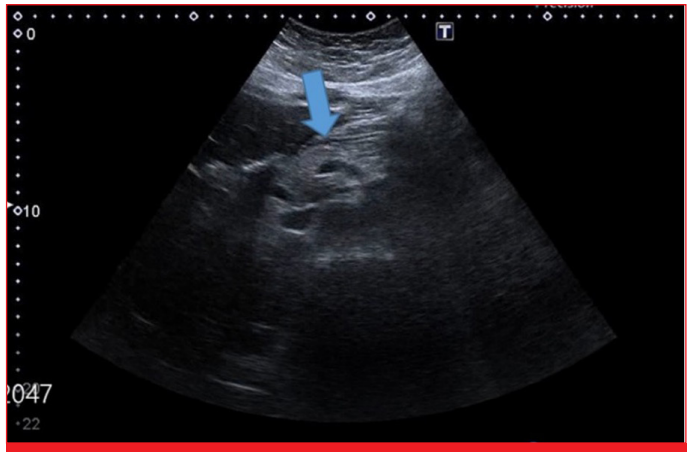

Figure 1. Arrow shows NAFPD

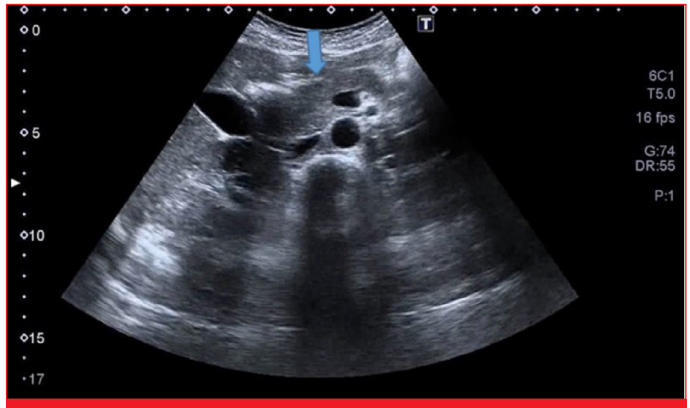

Figure 2. Arrow shows normal pancreas.

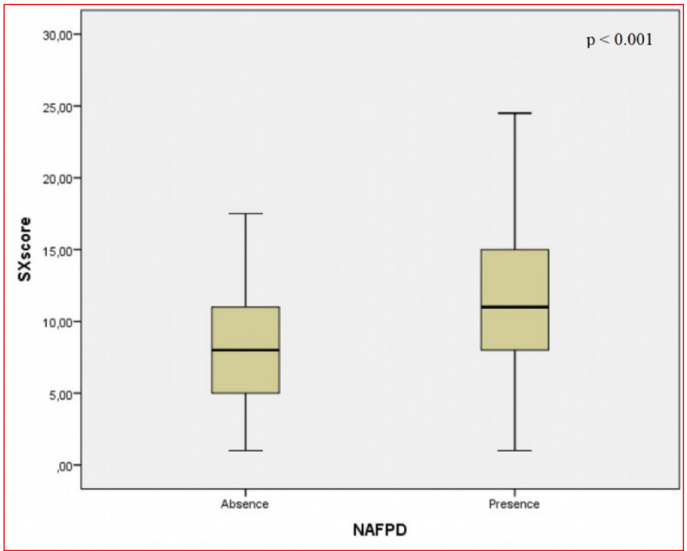

Figure 3. Univariate analysis for increased SXscore

Table 1. Clinical and demographic charactheristics with and without NAFPD subjects

Variables	NAFPD Absence n = 50	NAFPD Presence n= 49	*P*
Echocardiographic parameters			
LVEF, %	55 (50-60)	60 (45-60)	0.329
Aortic root, mm	22 (21-25)	24 (22-26)	0.066
LVEDd, mm	46.3±4.8	45.1±5.7	0.236
LVESd, mm	31.3±5.3	31.7±5.8	0.752
IVST, mm	11.5(11.0-12.0)	12 (11-13)	0.230
PWT, mm	11 (11-12)	12 (11-12)	0.312
E, cm/sec	70.3±19.8	69.4±15.7	0.814
A, cm/sec	73.8±18.9	81.6±27.7	0.118

Echocardiographic parameters LVEF, left ventricular ejection fraction; LVEDd, left ventricle end-diastolic diameter; LVESd, left ventricle end systolic diameter; IVST, interventricular septal thickness; PWT, posterior wall thickness,NAFPD, non-alcoholic fatty pancreas disease.

Table 2. Clinical and demographic characteristics with and without NAFPD subjects

Variables	NAFPD Absence n = 50	NAFPD Presence n= 49	*P*
Clinical and demographic characteristics			
Age, year	61 ± 10	64 ± 11	0.164
Gender, male, n (%)	42 (84)	32 (65)	0.032
Hypertension, n (%)	34 (68)	39 (79)	0.190
DM, n (%)	6(12)	7 (14)	0.736
Dyslipidemia, n (%)	21 (42)	22 (44)	0.771
Current Smoking, n (%)	28 (56)	27 (55)	0.928
Family history of CAD, n (%)	16 (32)	20 (40)	0.285
BMI (kg/m^2)	26.7 ± 3.2	28.7 ± 4.0	0.007
HR (bpm)	74 ± 13	75 ± 12	0.773
SBP, mm HG	126.9±18.4	130.8 ± 16.7	0.276
DBP, mm HG	75.6 ± 11.8	75.4 ± 11.9	0.952
SXscore	8.2±4.3	12.3±6.4	<0.001

Table 2. Clinical and demographic characteristics with and without NAFPD subjects (Continued)

Variables	NAFPD Absence n = 50	NAFPD Presence n= 49	*P*
Biochemical and hematological parameters			
Serum glucose (mg/dL)	125 (98-145)	122 (101-140)	0.823
Serum creatinine (mg/dL)	0.89 ± 0.28	0.84 ± 0.19	0.304
Serum urea (mg/dL)	33 (30-38)	34 (30-40)	0.725
Total cholesterol (mg/dL)	202.4±58.4	210.7±36.2	0.402
LDL-c (mg/dL)	136 ± 43	143 ± 34	0.943
HDL-c (mg/dL)	42±10	43±9	0.871
Triglyceride (mg/dL)	94 (63-170)	124 (93-211)	0.051
WBC (x10^9/L)	8.6 (7.5-11.8)	8.5 (7.7-11.4)	0.817
Hb (g/dL)	14.3 ± 1.3	14.0 ± 1,6	0.242
PLT (x10^9/L)	235 ± 44	254 ± 85	0.179
MPV (fL)	8.4±0.9	8.6±0.7	0.191
Troponin	1821 (149- 8643)	2500 (190-9741)	0.513

DM, diabetes mellitus; BMI, body mass index; CAD, coronary artery disease; WBC, white blood cell; PLT, platelet; HGB, hemoglobin; MPV, mean platelet volume; HDL-c, high-density lipoprotein cholesterol; LDL- c, low-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; SXscore, syntax score;NAFPD, non-alcoholic fatty pancreas disease.

Table 3. Multivariate analysis increased SXscore

Variables	Adjusted Odds ratios (% 95 CI)	*P*
Hypertension	2,459 (0.929 – 6.508)	0.070
Presence of NAFPD	3,934 (1.657 – 9.339)	0.002

NAFPD, non-alcoholic fatty pancreas disease.

Table 4. Univariate analysis for increased SXscore

Variables	Odds ratios (%95 CI)	*P*
SBP	1.005 (0.982 – 1.028)	0.688
DBP	1.004 (0.970 – 1.038)	0.833
HR	0.992 (0.962 – 1.023)	0.609
LVEF	0.967 (0.923 – 1.013)	0.159
LVED[d]	0.999 (0.978 – 1.077)	0.986
LVES[d]	1.001 (0.987 – 1.082)	0.970
IVST	1.038 (0.823 – 1.310)	0.750
PWT	1.127 (0.837 – 1.503)	0.441
E	0.990 (0.967 – 1.014)	0.420
A	1.016(0.997 – 1.035)	0.098
Aortic root diameter	1.017 (0.918 – 1.127)	0.747
Presence of NAFPD	4.154 (1.776 – 9.718)	0.001

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; LVEF, left ventricular ejection fraction; LVEDd, left ventricle end-diastolic diameter; LVESd, left ventricle end systolic diameter; IVST, interventricular septal thickness; PWT, posterior wall thickness; NAFPD, nonalcoholic fatty pancreas disease

Table 5. Univariate analysis for increased SXscore

Variables	Odds ratios (%95 CI)	*P*
Age	1.026 (0.988 – 1.064)	0.180
Gender	1.511 (0.593 – 3.853)	0.387
BMI	0.976 (0.877 – 1.085)	0.653
Family History of CAD	0.658 (0.288 – 1.502)	0.320
Hypertension	2.726 (1.084 – 6.854)	0.033
Diabetes mellitus	0.881 (0.273 – 2.841)	0.832
Dyslipidemia	1.120 (0.502 – 2.500)	0.782
Current smokers	0.499 (0.220 – 1.129)	0.095
WBC	0.907 (0.788 – 1.044)	0.175
PLT	0.996 (0.989 – 1.002)	0.173
HGB	0.759 (0.566 – 1.019)	0.067
MPV	1.140 (0.694 – 1.872)	0.60
Serum glucose	1.002 (0.995 – 1.010)	0.543
Serum urea	1.001 (0.962 – 1.042)	0.950
Serum creatinine	0.318 (0.057 – 1.758)	0.189
Total cholesterol	1.003 (0.994 – 1.011)	0.524
HDL-c	0.999 (0.991 – 1.006)	0.777
LDL-c	1.001 (0.990 – 1.011)	0.878
Triglyceride	1.000 (0.997 – 1.002)	0.949

BMI, body mass index; CAD, coronary artery disease; WBC, white blood cell; PLT, platelet; HGB, hemoglobin; MPV, mean platelet volume; HDL-c, high-density lipoprotein cholesterol; LDL- c, low-density lipoprotein cholesterol

Coronary Artery Disease / Acute Coronary Syndrome

PB-077

Systemic immune inflammation index may predict the development of contrast-induced nephropathy in patients with ST segment elevation myocardial infarction

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Background and Aim: To investigate the predictive capacity of a systemic immune-inflammation index (SII) in detecting contrast-induced nephropathy (CIN) following ST segment elevation myocardial infarction (STEMI).

Methods: This retrospective observational study consisted of 477 patients who were followed-up in our cardiology department between March 2018 and March 2021 for STEMI. Medical and demographic data of the patients such as age, gender, body mass index (BMI), hypertension (HT), diabetes mellitus (DM), hyperlipidemia and smoking were collected. 12-lead electrocardiography (ECG) was used to determine the rhythm. SII was calculated using the formula platelet count x neutrophil count / lymphocyte count. CIN was defined as a 25% increase or 0.5 mg/dl increase in absolute levels of creatinine 72 h after the patient's admission without any other etiology.

Results: A total of 347 patients were included in the study; of these, 85 patients developed CIN. The baseline demographic characteristics, routine laboratory and echocardiographic findings as well as medication history of the study population with and without CIN are listed in Table 1. A multivariate regression analysis was performed for the factors that showed statistical significance with the univariate regression analysis and factors such as left atrial diameter, CK-MB at peak, glucose, Hs-CRP and SII were found to be significantly associated with the development of CIN (Table 2). ROC curve analysis showed that Hs-CRP (C-statistic: 0.783; 95% CI: 0.716-0.850, p<0.001), SII (C-statistic: 0.732; 95% CI: 0.662-0.802, p<0.001), NLR (C-statistic: 0.701; 95% CI: 0.628-0.774, p<0.001) and PLR (C-statistic: 0.646; 95% CI: 0.569-0.723, p<0.001) were significant predictors of CIN following STEMI (Figure 1 and Table 3). A cut-off point of 5.91 for logarithm-transformed SII was identified with 72.97% sensitivity and 65.62% specificity to predict CIN following STEMI. According to pairwise analysis of ROC curve analysis, the predictive power of SII in detecting CIN following STEMI was similar to high-sensitive C reactive protein, and better than neutrophil/lymphocyte ratio or platelet/lymphocyte ratio.

Conclusions: SII can be used as one of the independent predictors of CIN following STEMI.

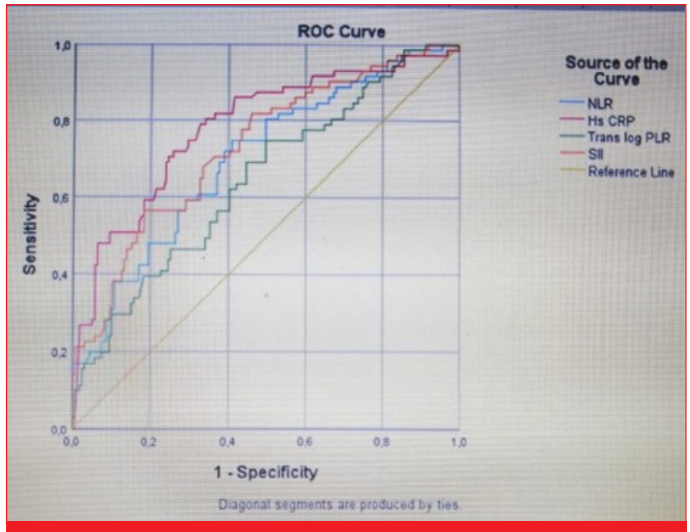

Figure 1. Receiver operating characteristic (ROC) curve with calculated area under the curve and optimal cut-off point for the Hs-CRP, SII, NLR, PLR to identify the presence of CIN. HsCRP, high sensitivity C- reactive protein; PLR, platelet-to-lympho

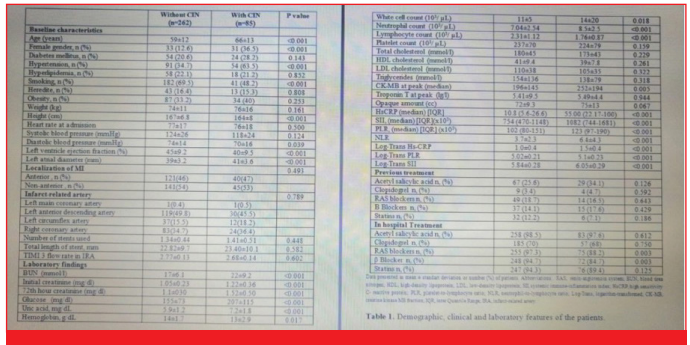

Table 1. Demographic, clinical and laboratory features of the patients

	Univariate regression analysis			Multivariate regression analysis		
	Odds Ratio	%95 Confidence Interval	P value	Odds Ratio	%95 Confidence Interval	P value
Age	1.03	1.01-1.06	<0.001			
Female gender	3.98	2.24-7.06	<0.001	4.65	1.28-16.88	0.019
Hypertension	3.27	1.96-5.44	<0.001			
Unc Acid	1.78	1.48-2.15	<0.001	1.46	1.13-1.90	0.004
Height	0.94	0.91-0.97	<0.001			
Ejection fraction	0.94	0.91-0.97	<0.001			
Left atrial diameter	1.22	1.12-1.33	<0.001			
CK-MB at peak	1.002	1.002-1.004	0.006	1.003	1.001-1.005	0.017
Hemoglobin	0.85	0.75-0.97	0.016			
Glucose	1.006	1.003-1.009	<0.001	1.005	1.001 - 1.009	0.019
Smoking	0.41	0.24-0.67	<0.001			
HsCRP	1.03	1.02-1.04	<0.001	1.032	1.019-1.046	<0.001
Opaque amount	1.02	0.99-1.04	0.007			
SII	14	4.71-42.10	<0.001	6.034	1.433 – 25.482	0.006

Abbreviations: SII, systemic immune-inflammation index; BUN, blood urea nitrogen; HsCRP, high sensitive C- reactive protein; CK-MB, creatine kinase-MB fraction.

Table 2. Univariate and multivariate regression analysis of predictors of contrast-induced nephropathy in the study population

	C-statistic	95%Confidence interval	P value	Cut-off value	sensitivity	specificity
Hs-CRP	0.783	0.716 - 0.850	<0,001	16.4	83.75%	65.62%
Log-trans SII	0.732	0.662 - 0.802	<0,001	5.9133	72.97%	57.53%
NLR	0.701	0.628 - 0.774	<0,001	5.625	55.41%	81.18%
Log-trans PLR	0.646	0.569 - 0.723	<0,001	5.0093	74.32%	49.46%

Abbreviations: SII, systemic immune-inflammationindex; Hs-CRP, high sensitive C- reactive protein; Log-Trans, logarithm-transformed; PLR, platelet-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio.

Table 3. The ROC analysis of inflammatory parameters

Coronary Artery Disease / Acute Coronary Syndrome

PB-078

Lipid profile indices and prediction of in-hospital mortality in patient with STEMI undergoing primer PCI

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Background and Aim: Dyslipidemia is an established risk factors for cardiovascular disease. Increased triglyceride (TG), low-density lipoprotein cholesterol (LDL-C) levels, and decreased HDL-C levels were associated with increased cardiovascular risk. Recently, comprehensive lipid profile indices derived from these conventional parameters have attracted to attention.

Although various studies demonstarted that their relationship with these lipid indices and clinical outcomes in patients with acute coronary syndrome, this situation is not yet clear in acute ST-elevation myocardial infarction (STEMI) patients undergoing primer percutaneous coronary intervention (pPCI).The aim of this study was to focus on the relationship traditional lipid levels and non-traditional comprehensive lipid indices and in hospital-mortality in patients with STEMI after pPCI.

Methods: In this retrospective, single center, hospital-based study, between January 2019 and April 2021, a total of 873 consecutive STEMI patients (540 men; median age 59 ±12 years), whose undergoing pPCI, are enrolled. The patients were divided into two groups according to in-hospital mortality status namely survivor and deceased. Conventional lipid values were measured and non-traditional lipid indexes including non-HDL-C [Total cholesterol minus HDL-C], Total cholesterol/HDL-C, LDL-C/HDL-C, atherogenic index (AI) [non-HDL-C/HDL-C], lipoprotein combine index (LCI) [Total cholesterol*TG*LDL-C/HDL-C] and atherogenic index of plasma (AIP) [Log(TG/HDL-C)] were calculated. All the data were analyzed by using the IBM SPSS Statistics for Macintosh, Version 24.0 (IBM Corp., Armonk, New York, USA) software. For all tests, p value <0.05 was considered to be statistically significant.

Results: AIP was significantly higher in hospital mortality patients compared to survivor group (0.59, 0.47, p=0.006, respectively). AI, non-HDL-C, Total cholesterol/HDL-C ratio, LDL-C/HDL-C ratio and LCI measurements were similar between two groups. The cut-off value of the AIP (0.50) was associated with 70% sensitivity and 52% specificity for predicts in-hospital mortality. Multivariate logistic regression model yielded AIP (OR: 3.77, 95% CI: 1.34–10.6, p<0.012) as independent predictor of in-hospital mortality.

Conclusions: AIP predicts in-hospital mortality in patients with STEMI undergoing pPCI. AIP, which can be calculated easily from complete blood, can be beneficial in the prognostication of these patients.

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

Variables	Deceased (n= 53)	Survivors (n= 820)	*p*
Age, years	69 (61-76)	58 (51-66)	<0.001
Gender, male	35 (66%)	505 (62%)	0.829
Diabetes mellitus	21 (40%)	203 (25%)	0.016
Hypertension	27 (51%)	302 (37%)	0.040
Prior CABG	4 (8%)	32 (4%)	0.196
Prior PCI	11 (21%)	124 (15%)	0.272
Prior CVD	7 (13%)	20 (2%)	<0.001
Creatinin, mg/dL	1.29 (0.83-1.71)	0.83 (0.72-0.99)	<0.001
Albumin, mg/dL	37 (33-42)	41 (38-43)	<0.001
Aspartate aminotransferase, U/L	150 (52-525)	97 (43-237)	0.007
Hemoglobin, g/L	13.2 (11.5-15.1)	14.2 (13.0-15.3)	0.011
Total cholesterol, mg/dL	162 (135-180)	177 (154-207)	0.005
Triglyceride, mg/dL	144 (83-165)	103 (70-164)	0.033
HDL-C, mg/dL	31 (25-38)	34 (29-40)	0.007
LDL-C, mg/dL	104 (81-135)	118 (93-143)	0.123
VLDL-C, mg/dL	24 (15-32)	21 (14-33)	0.300
AIP	0.59 (0.46-0.83)	0.47 (0.26-0.72)	0.006
AI	4.71 (3.11-5.84)	4.13 (3.19-5.22)	0.168
Non-HDL-C, mg/dL	130 (109-173)	143 (119-170)	0.125
Total cholesterol / HDL-C	5.5 (4.1-6.5)	5.1 (4.2-6.2)	0.533
LDL-C/HDL-C	3.59 (2.60-4.67)	3.42 (2.68-4.21)	0.519
Lipoprotein combine index, x1000, mg/dL	649 (430-1121)	632 (335-1139)	0.567
Infarct location, anterior	26 (49%)	372 (45%)	0.341
Total stent length, mm	33 (28-48)	29 (23-41)	0.057
Stent diamater, mm	3.0 (2.75-3.0)	3.0 (2.75-3.0)	0.576

Coronary Artery Disease / Acute Coronary Syndrome

PB-079

Pretty much factored syndrome-Kounis

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Background and Aim: Kounis syndrome also known as allergic angina syndrome, can be defined as togetherness of an allergic reaction and acute coronary syndrome. The most important point of pathogenesis is degranulation of vasospastic mediators from mast cells. This syndrome can occur by various factors especially drugs (most commonly antibiotics), insect bites, bee stings. Recently, myocardial infarction with non-obstructive coronary arteries becomes more popular in scientific area. Kounis syndrome, similar pathogenesis at some points with this phenomenon is more often than estimated. In our case serial we aim to present six patients of Kounis syndrome that occur with various factors. We also intend to raise awareness about this quite interesting syndrome with different clinical presentations.

Methods: Case serial - Kounis syndrome.

Results: CASE 1: 27 years old female patient. Applied to emergency service with chest pain and allergic symptoms after an analgesic drug (metamizole) injection. CASE 2: 32 years old female patient. Allergic symptoms and chest pain after an analgesic drug (dexketoprofen) injection. CASE 3: 48 years old male patient. Applied to emergency service with allergic symptoms and sweating, after multiple bee stings. CASE 4: 43 years old male patient. Applied to emergency service with allergic symptoms and retrosternal chest pain after unknown insect bite. CASE 5: 43 years old male patient. Applied to emergency service with allergic symptoms and chest burn after a single bee sting. CASE 6: 57 years old male patient. Applied to emergency service with allergic symptoms and chest burn twelve hours after vaccinated for Covid 19.

	CASE 1	CASE 2	CASE 3	CASE 4	CASE 5	CASE 6
AGE	27	32	48	43	51	57
GENDER	Female	Female	Male	Male	Female	Male
SYMPTOMS	Allergic symptoms and chest pain	Allergic symptoms and chest pain	Allergic symptoms and sweating	Allergic symptoms and chest pain	Allergic symptoms and chest pain	Allergic symptoms and chest pain
RISK FACTORS	Smoking	None	Smoking and family history	Smoking	Diabetes and family history	None
ALLERGEN	Analgesic drug	Analgesic drug	Bee sting	Unknown insect	Bee sting	Vaccine
hs-Troponin	0,257	0,012	0,612	0,243	1,113	0,479
ECG	NSR	V2-3-4 T wave negativity	NSR	D1-aVL-V5-V6 ST segment elevation	NSR	V2-3-4-5 ST segment elevation
ECHO	Normal	Mitral Regurgitation Septal hypokinesia	Normal	Anterior and lateral wall hypokinesia	Normal	Apex hypokinesia
CAG	Normal			Circumflex artery - LCX	Circumflex artery - LCX	Left anterior descending - LAD
CT ANGİOGRAPY	-	Normal	Normal	-	-	-
TYPE	1	1	1	2	2	2 and 3

Table 1. Kounis Syndrome
Demographic, clinical and laboratory features

Conclusions: Kounis syndrome may contain allergic symptoms, findings and anamnesis if comes to mind in patients presenting with typical acute coronary syndrome clinical appereance. And this syndrome should be considered in patients with allergic symptoms and chest pain. Sometimes this point of view can clarify complicated clinical presentations.

Coronary Artery Disease / Acute Coronary Syndrome

PB-080

The Relationship of Salusin Beta Level with Coronary Artery Disease in Patients with Non-ST Elevation Myocardial Infarction (NSTEMI)

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Background and Aim: Myocardial infarction is clinically defined as acute myocardial injury detected by abnormal cardiac biomarkers in the presence of evidence of acute myocardial ischemia. Peripheral blood biomarkers are seen as important tools for the detection of subclinical disease, diagnosis and risk stratification of acute or chronic coronary syndromes. Salusins are potential biomarkers in the peptide hormone structure found in human plasma as alpha and beta forms. Serum salusin alpha levels were found to be inversely proportional to the presence and severity of coronary artery disease. It is known that the level of salusin beta in the vascular circulation increases in atherosclerosis and related cardiovascular diseases. However, little information is available about salusin beta levels in patients with coronary artery disease. In this study, for the first time, we aimed to investigate the relationship between salusin beta, a potential biomarker in the diagnosis, follow-up and treatment of coronary artery disease, and coronary artery disease in patients diagnosed with NSTEMI.

Methods: In this study, those who did not have a previous history of cardiovascular disease, peripheral vascular disease, cerebrovascular disease, malignancy, severe liver failure and renal failure, over 18 years of age and under 75 years of age; volunteers who signed an informed consent form were included. Blood samples were taken from the volunteers in the NSTEMI group during the first hospitalization and on the day of discharge for salusin beta level measurement. Blood samples were taken from healthy volunteers in the control group at the outpatient clinic application.

Results: In the analysis of blood samples taken at the time of admission from NSTEMI study group patients and control group individuals, although the salusin beta value was higher in the NSTEMI group, no significant difference was found between the two groups in terms of salusin beta levels (p=0.833) (Figure 1). When the NSTEMI study group was evaluated within itself and the blood samples taken on the day of hospitalization and discharge were analyzed, it was observed that there was a decrease in the salusin beta level measured on the

day of discharge compared to the day of hospitalization, but no statistically significant difference was found between the two groups in terms of salusin beta levels (p=0.059) (Figure 2).

Conclusions: In our prospective study, although salusin beta level increased in patients who had NSTEMI; no statistically significant correlation was found between salusin beta level and the presence, prevalence and severity of coronary artery disease. However, it should be noted that there are few studies on the role of salusin beta level in coronary artery disease in patients with acute coronary syndrome. Our findings form the basis for future studies of salusin, and more studies are needed in order for salusin beta molecule to guide the diagnosis and treatment of coronary artery disease.

	N	Min-Max	Medyan[Q1-Q3]	p
Salusin Beta 1 (NSTEMI hospitalization day)	25	140,68-5262,72	293,97[214,95-370,55]	0,833
Salusin Beta 3 (healthy population control group)	27	169,03-916,03	286,35[258,71-337,09]	

Figure 1. Comparison of salusin beta level measured on hospitalization day in NSTEMI patient group and salusin beta level measured in healthy population
When the median values of salusin beta level(Salusin Beta 1) measured during hospitalization in the patient group with NSTEMI and salusin beta level(Salusin Beta 3) measured in the healthy population control group were compared, although salusin beta value was higher in the NSTEMI group, no statistically significant difference was found between the two groups in terms of salusin beta levels.(p=0.833)

	N	Min-Max	Medyan[Q1-Q3]	p
Salusin Beta 1 (NSTEMI hospitalization day)	25	140,68-5262,72	293,97[214,95-370,55]	0,059
Salusin Beta 2 (NSTEMI discharge day)	24	135,24-1203,70	282,09[243,92-324,50]	

Figure 2. Comparison of salusin beta level on hospitalization and discharge day in NSTEMI patient group
When the salusin beta median value (Salusin Beta 1) measured during hospitalization was compared with the salusin beta median value (Salusin Beta 2) measured on the day of discharge; although it was observed that there was a decrease in the level of salusin beta measured on the day of discharge compared to the day of hospitalization, the difference was not statistically significant.(p=0.059)

Coronary Artery Disease / Acute Coronary Syndrome

PB-081

The impact of serum triglyceride-glucose index in young non-diabetic patients presenting for the first time with acute coronary syndrome

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Background and Aim: In recent years, Triglyceride-glucose (TyG) index has taken its place as a new parameter predicting mortality in cardiovascular diseases. In this study, we aimed to investigate the importance of the TyG index and other cardiovascular risk factors who diagnosed Non-ST elevation myocardial infarctions (STEMI) and non-diabetics patients.

Methods: The data of patients without diabetes and diagnosed with first-time non-STEMI, <65 years, and underwent coronary angiography were analyzed retrospectively from the patient files. TyG index was measured as [fasting Triglyceride (TGs, mg/dL) x fasting serum glucose (mg/dL)/2]. The patients were divided into two groups as young adults (under 45 years, n=46) and middle-aged adults (45-65 years, n=60). Demographic, clinical, and laboratory characteristics of both patient groups were compared.

Results: The serum TyG index (9.6±12.3 vs 5.6±3.1, p=0.024), TG levels (259.2±235.0 vs 161.8±81.7, p=0.006), and smoking (73.9% vs 55.0%, p=0.035) rate were higher in the young patient group, serum high-density lipoprotein (HDL) levels (34.1±8.8 vs 40.2±9.7, p=0.003) were lower. In the univariable regression analysis, smoking (p=0.048), TyG index (p=0.049), and serum TG (p=0.014), and HDL (p=0.005) levels were found as possible independent predictors in young patients with NSTEMI. However, in multivariable regression analyzes, no significant difference was found between risk factors (p>0.5).

Conclusions: In young patients without diabetes who presented with acute coronary syndrome for the first time, in addition to smoking and low HDL levels which are among the known risk factors, high serum TG and TyG indexes were found to be possible independent risk factors associated with NSTEMI.

Table 1. Evaluation of smoking, serum TyGi, HDL, and TG levels with univariate and multivariate regression analysis in patients presenting with non-ST elevation myocardial infarction.

Variables	Univariable Analysis		Multivariable Analysis	
	OR 95 % CI	*P*	OR %95 CI	*P*
Smoking	2.318 (1.009-5.326)	0.048	1.959 (0.712-5.390)	0.193
TyG index	0.873 (0.761-0.987)	0.049	1.005 (0.789-1.280)	0.968
Triglyceride	0.994 (0.990-0.999)	0.014	0.996 (0.986-1.007)	0.501
High-density lipoprotein	1.079 (1.023-1.138)	0.005	1.043 (0.978-1.007)	0.501

Table 2. Comparison of demographic, clinical and laboratory data of young and middle-aged adults

Variables	Age < 45 years (n=46)	Age > 45 years (n=60)	*P*
Sex, male (%)	36 (78.3)	39 (65.0)	0.101
Smoking, (%)	34 (73.9)	33 (55.0)	0.035
Left ventricular ejection fraction, %	54.9±5.8	52.4±8.1	0.074
Triglycerid Glucose index	9.6±12.3	5.6±3.1	0.024
Triglyceride, mg/dL	259.2±235.0	161.8±81.7	0.006
High-density lipoprotein, mg/dL	34.1±8.8	40.2±9.7	0.003

Coronary Artery Disease / Acute Coronary Syndrome

PB-082

Effect of cardiac rehabilitation on heart rate recovery in patients with coronary artery disease

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Background and Aim: Cardiovascular disease (CVD) is one of the leading causes of mortality and morbidity in the world. Although the exact pathophysiology of CVD remain unclear; emerging evidence indicates that the function of the autonomic nervous system has an important role in the development of CVD. Exercise-based cardiac rehabilitation programmes (CR) improve cardiovascular mortality and quality of life in patients with coronary artery disease (CAD). It's postulated that effective exercise-based CR improves the post-exercise parasympathetic function. Vagally mediated heart rate recovery (HRR) is defined as the difference in the heart rate (HR) between the peak of exercise and one or more minutes after exercise cessation. Reduced HRR has been shown to be related with an increased risk of cardiovascular and all-cause mortality. In this study we evaluated the effect of change in HRR after CR on clinical outcomes in CAD patients.

Methods: This was a single center, retrospective cohort study. Patients with CAD who were directed to the CR unit and completed all 30 and 60 sessions were included in the study. A special CR program was performed for each patient according to their cycle ergometer test peak workload. 6-minute walk test (6MWT) was performed for assessing functional capacity.Patients were asked to walk back and forth as much as possible around cones in six minutes on a 30-foot path. Patients were visited by nutritionist for dietary recommendations, received psychosocial management. In order to calculate HRR, the maximum heart rate during the exercise test was recorded. HR was recorded again after the ending of the test at 1, 2, 3 minutes. The difference between maximum HR and at the end of the 1st and 2nd and 3rd minutes after exercise measurements were considered as HRR1, HRR2, HRR3 respectively.

Results: There were a total of 104 patients enrolled in this study. 28.8% of patients were female and 71.2% were male. Mean age was 58.31 ± 9.28. 71.2% of the patients were completed 60 sessions. Body mass index of patients is decreased after training program (p=0.044). Functional capacity (6MWT) and endurance (Cycle Ergometer Test Maximum Watts) of the patients statistically significantly increased after CR sessions (p=0.001, p<0.001 respectively). CR resulted in a significant increase in HRR1, HRR2, HRR3. Mean HRR significantly improved (from 26.24 ± 10.59 to 29.49 ± 8.61; p=0.006). There is no difference in HRR values betweeen 30 sessions (10 week) and 60 sessions (20 week).

Conclusions: Resting HR in healthy individuals is predominantly determined by the vagus nerve. It is known that those with CVD show autonomic dysfunction. The increase in HRR with cardiac rehabilitation suggests that there is improvement in autonomic dysfunction in patients with coronary artery disease. As a simple and noninvasive marker, HRR can be used to monitor patients receiving cardiac rehabilitation.

Table 1. Baseline characteristics of the study population

		30 sessions (n=30)	60 sessions (n=74)	*P*
Age	58.31 ± 9.28	57.63 ± 12.27	58.58 ± 7.85	0.640
Gender (male)	74 (71.2%)	22	52	0.755
HT	68 (%65.4)	20	48	0.861
DM	22 (%21.2)	7	15	0.729
HF	15 (%14.4)	4	11	0.840

Table 2. Evaluation of BMI, endurance and functional capacity, and heart rate recovery before and after cardiac rehabilitaion.

	Before CR	After CR	*P*
Value			
BMI (kg/m^2)	28.81 ± 4.66	28.55 ± 4.37	0.044
Cycle ergometer test max (WATT)	63.22 ± 22.29	77.38 ± 19.87	<0.001
6MWT (meters)	367.83 ± 56.58	381.61 ± 53.76	0.001
HRR1	21.61 ± 11.03	24.30 ± 8.67	0.036
HRR2	28.11 ± 11.72	31.23 ± 8.84	0.015
HRR3	29.01 ± 10.97	32.94 ± 10.38	0.002
Mean HRR	26.24 ± 10.59	29.49 ± 8.61	0.006

Values are given as means ± SD

Table 3. Cardiovascular responses to CR program at baseline and after rehabilitation program

	30 Sessions	60 Sessions	*P*
ΔHRR1	0.43 ± 14.61	3.60 ± 12 16	0.258
ΔHRR2	0.67 ± 13.83	4.12 ± 12.49	0.218
ΔHRR3	0.67 ± 13.14	5.26 ± 12.28	0.094
Δ Mean HRR	0.59 ± 12.67	4.33 ± 11.50	0.148
ΔWATT	10.00 ±19.25	15.83 ±22.10	0.209
Δ6MWT	4.00 ± 35.56	17.74 ± 40.96	0.111

Coronary Artery Disease / Acute Coronary Syndrome

PB-083

Cardiac arrest in a patient with congenital cytomegalovirus infection sequela and heterozygous variation of c.5944C>T p.R1982C in filamin C (FLNC) gene

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Background and Aim: Congenital cytomegalovirus (CCMV) sequela is common cause of sensorineural hearing loss (SNHL). CCMV causes neurodevelopmental disabilities, including cerebral palsy, intellectual disability, vision impair-

ment, and seizures. However, cardiac pathologies are not commonly associated with CCMV. Establishing a diagnosis of congenital CMV infection beyond the first year of life is generally based on clinical suspicion and neuroimaging. Although neurological manifestations of CCMV are well recognized, it has rarely been reported in cardiac pathologies.

Methods: We report a 35 year old male from Uzbekistan with congenital cytomegalovirus infection sequela (CCMV) who was admitted to the hospital after out of hospital cardiac arrest (OHCA).

Results: ECG displayed normal sinus rhythm, normal QT interval, and diffuse STT wave changes in the inferolateral leads (Figure 1). On brain MRI: Right hemisphere is pachygyric, multifocal/confluent subcortical white matter lesions(a,b), with involvement of anterior temporal lobes (c,d) were seen (Figure 2). MRI findings were compatible with the diagnosis of with congenital cytomegalovirus infection sequela. The work-up with coronary angiography, and echocardiography were normal. Genetic testing for arrhythmia related foci displayed filamin C mutation (FLNC, OMIM accession number 102,565). Mutations in FLNC are associated with skeletal and cardiac myopathies, which mainly manifest skeletal muscle weakness and cardiomyopathy. Yet the associaton between FLNC gene and arrhythmia has not been reported before. The identified variation (c.5944C>T (p.Arg1982Cys) has not been reported with any cardiac pathology in the literature. FLNC mutation is reported in 45/140,000 exomes or genomes, and in south Asians it is ~0.1%.

Conclusions: Genetic testing for arrhythmogenic genetic foci displayed FLNC mutation (OMIM accession number 102,565). FLNC gene encodes for filamin C, an actin cross-linking protein, widely expressed in cardiac and skeletal muscles. Mutations in FLNC are associated with skeletal and cardiac myopathies. Yet the associaton between FLNC gene and arrhythmia has not been reported before. Future studies are needed to assess the association of CCMV, ventricular arrhythmia, and FLNC gene variations. Novel bioinformatic programs such as silico prediction based on inferred function, and evolutionary conservation can help us understand the genetic variations and the designated pathologies in future studies.

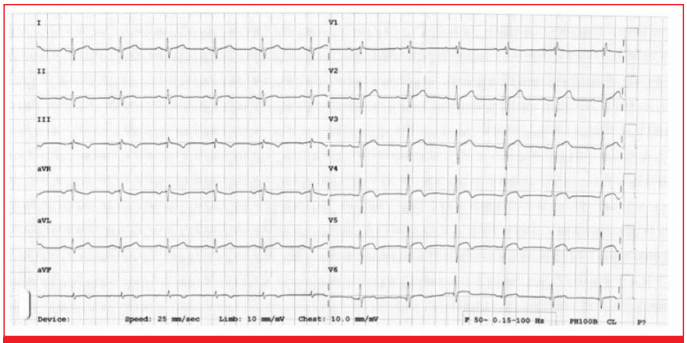

Figure 1: ECG displayed normal sinüs rhythm, normal QT interval, and diffuse STT wave changes in the inferolateral leads.

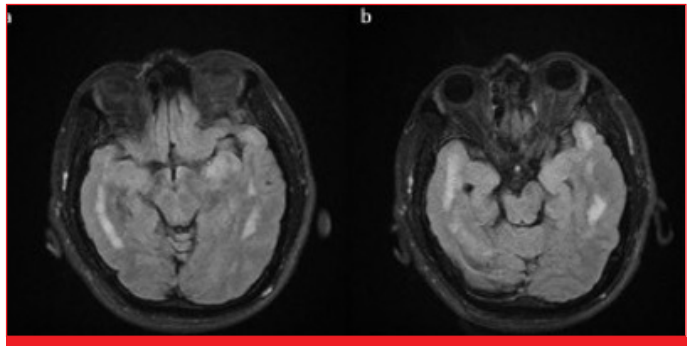

Figure 2: Right hemisphere is pachygyric, multifocal/confluent subcortical white matter lesions (a,b), with involvement of anterior temporal lobes (c,d) were seen

Coronary Artery Disease / Acute Coronary Syndrome

PB-084

The Association Between Fibrinogen -to- Albumin Ratio and Apical Thrombus Formation in Patients with Acute Anterior Myocardial Infarction

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Background and Aim: Left ventricular apical thrombus formation is a clinically important complication of acute myocardial infarction (MI) with increased rate of thromboembolism. Because LV thrombus can be the major source of systemic embolic events, the characterization and elucidation of the underlying mechanisms and identification of early predictors for LV apical thrombus formation is vital for risk stratification and decisions as to management strategies. Recently, fibrinogen-to-albumin ratio (FAR) has emerged as a feasible and valuable serological marker that may reflect haemorheological and inflammatory status. Therefore, we investigated the predictive value of fibrinogen to albumin ratio (FAR) to identify high risk patients who will develop an apical thrombus during the acute phase of anterior transmural infarction.

Methods: Consecutive 1726 patients (mean age: 61.6 ± 9.7 years; male: 65.1%) with first acute anterior MI who underwent primary percutaneous coronary intervention (PCI) were evaluated. Patients meeting the following criteria were excluded from the study: past medication history with warfarin for chronic oral anticoagulation, patients who were treated conservatively or with thrombolysis, severe valvular disease, active infection, renal or hepatic insufficiency, confirmed coagulopathy, thrombocytosis, hematological proliferative diseases, oncological or inflammatory disorders. As the presence of apical aneurysm from the past acute MI can affect the formation of apical thrombus, we included only patients with a first episode of acute transmural anterior MI. Initial echocardiogram performed within 7 days following

admission. Patients were divided into 2 groups based on the presence of apical thrombus. Baseline FAR was calculated using the statistical program (SPSS) by dividing the fibrinogen value with the albumin value.

Results: LV apical thrombus was detected on echocardiogram in 102 patients (5.9%). Patients with an apical thrombus had higher rate of smoking, prolonged pain to balloon time, higher rate of post-PCI TIMI flow ≤1, lower LV ejection fraction (LVEF) and significantly higher FAR values than those without an apical thrombus. In multivariate analysis adjusted with smoking, LVEF, pain to balloon time and the presence of TIMI flow ≤1; having high FAR values was found as independent predictor of apical thrombus formation (OR: 4.024, 95% CI:1.747–9.273, p: 0.001).

Conclusions: In conclusion, FAR can be quickly and inexpensively calculated bedside with simple blood count analysis. In our study, admission FAR values were found to be significant and independent predictor for early LV apical thrombus formation complicating a first-ever anterior wall MI. This easily accessible and costless parameter may be utilised to identify high-risk patients for apical thrombosis and individualization of targeted therapy.

Coronary Artery Disease / Acute Coronary Syndrome

PB-085

Male and female differences in health benefits derived from a structured education and follow-up program in coronary artery disease

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Background and Aim: Primary prevention programs for coronary artery disease (CAD) may be effective in improving health-related behavioral outcomes. However, successful implementation and maintenance of these programs can vary amongst genders. The primary aim of this study is to assess the gender differences in a structured, enhanced education and follow-up program for CAD prevention in an area where the diverse population and economy are major problems.

Methods: SANKO CAD Prevention Project (SCAD-PPI) was designed as a longitudinal study and utilized medical school students to conduct the project under the supervision of professors. It began in 2014 and had two different education and training phases. In the first phase, every school year for second year Medical students underwent a year-long, especially designed training program on primary prevention for CAD. In the second phase, which took place in the second year of the study, a series of conferences regarding the primary prevention for CAD were organized by the University and local municipalities for underserved populations. Participants were prospectively assigned to an intervention where pre- and post-conference knowledge was collected and assessed. Every intervention was conducted by specially trained third year Medical students and an education booklet, which was specially designed for the study, was given to the participants. Every other month thereafter, for 6 months, each participant was followed-up via phone calls. At the 6 months follow -up, data was collected to assess the impact of the program on behavioral outcomes. Every year a new class assigned to undergo the same training program and serve to the local population at different locations within the same city and suburbs.

Results: A total of 172 participant were enrolled; 61% were women, mean age was 40±11.9 years with no significant difference in between the groups.; 67% of women were not working (p<0.001). While BMI rates were higher in women (27.9±5.5 kg/m^2, p<0.016), smoking rates were higher in men (p<0.001). Overall, knowledge on CAD risk factors, primary prevention measures, diet, and daily exercise habits were poor in both groups. After the enhanced education and follow-up program there was a significant improvement on the knowledge of CAD risk factors and primary prevention measures in both groups (p<0.001). Importantly, the follow-up program led both groups to implement those positive changes into their lives and maintain a healthy lifestyle.

Conclusions: This is the first study which showed that a longitudinally structured training program of medical students could be utilized to implement an enhanced education and follow–up program for primary prevention of CAD in an economically challenged, underserved population with successful outcomes in both genders. This model program is not only beneficial for public interest, but also enhances active interaction of medical students with patients at a very early stage of their career.

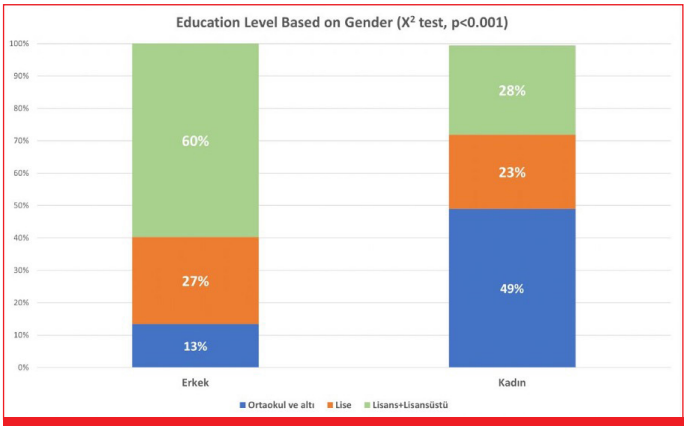

Figure 1. Education Level by Gender

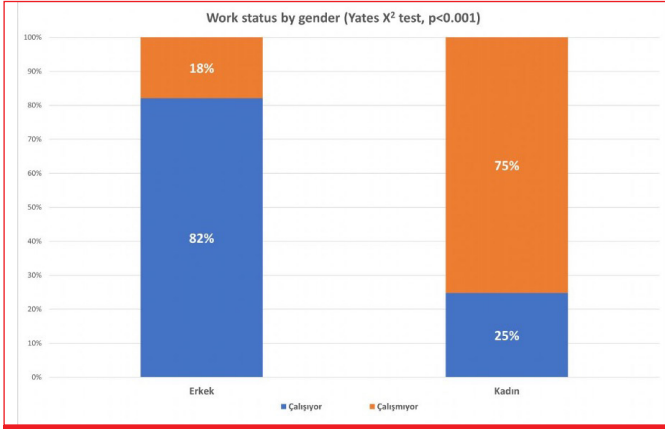

Figure 2. Work Status by Gender

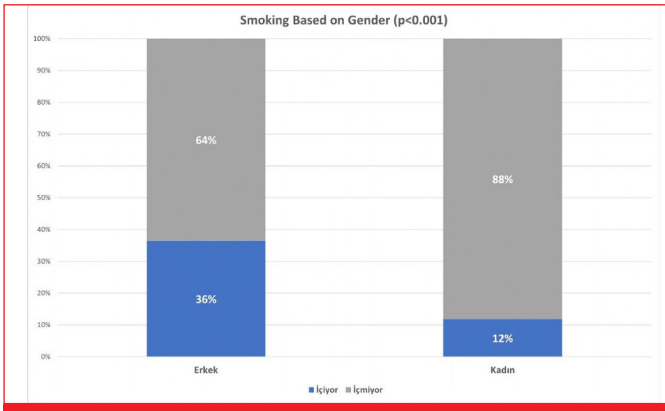

Figure 3. Smoking by Gender

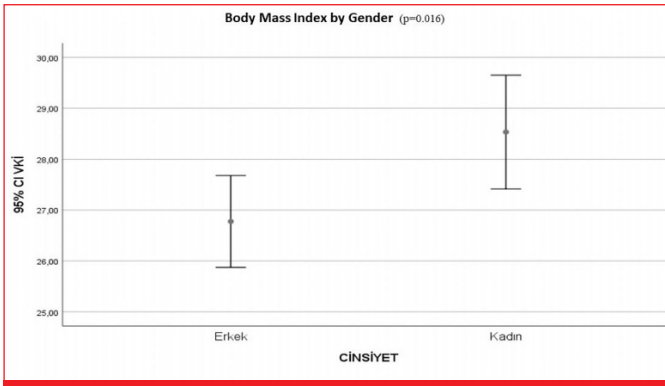

Figure 4. Body Mass Index by Gender

Lipid / Protective Cardiology

PB-086

Assessment of Statin Adherence in Patients Who Underwent Percutaneous Coronary Intervention

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Background And Aim: Statin therapy is one of the cornerstone part of coronary artery disease treatment as Its importance in reduction of cardiovascular (CV) events is proven by several large-scale randomized controlled trials. Current guidelines recommends high intensity statin treatment for primary and secondary prevention of CV events in specific patient populations according to determined targets of cholesterol levels and promotes statin adherence. In daily clinical pratice, discontuniation of statins is a frequent problem. Several reasons contribute statin non adherence including lack of self motivation, misunderstanding of treatment, compelling and non compelling drug side effects. We aimed to evaluate the status and associated factors of statin adherence in patients underwent percutaneous coronary intervention (PCI).

Methods: All cases who underwent index percutaneous coronary intervention (PCI) between January and July 2019 in Hacettepe University Hospital were screened. Patients with previous PCI history and patients who died during follow-up were excluded. Datas were obtained from electronic database of the hospital.

Results: A total of 187 patients were included. Mean age was 63.3±11.0 years and 27.8 % of the patients (n=52) were female. Hypertension was present in 102 (54.5%) cases and diabetes was present in 60 (32.1%) cases. Baseline characteristics were presented in Table 1. Median follow-up after PCI was 14 (6-22) months. Baseline LDL-C level was 135 (101-160) mg/dL and LDL-C level after follow-up was 100 (85-124) mg/dL. Most of the cases (53.5%) were under treament with low intensity statin. During follow-up; 49 (26.2%) patients stopped using statin. Staying at statin treatment at the end of 1 year was 84.2%. The most common cause of statin discontinuation was patient's own request, however doctor recommendation due to low levels of LDL-C under treatment, elevation of liver enzymes, myalgia and pruritus were the following reasons. Predictors of statin discontinuation was assessed after excluding patients who discontinued statin according to doctor recommendation. Multivariate Cox regression analysis showed that presence of hypertension (HR:2,82; p=0.010) and diabetes (HR:0,27; p=0.006) were associated with statin discontinuation (parameters with a p value<0.200 in univariate analysis were included) (Table 2).

Conclusions: Despite statin adherence is crucial in very high risk patients; still an important proportion of patients don't adhere to statin treatment. Patient's own request is the most common cause of statin discontinuation and treatment approach of primary care physicians is still insufficient. Presence of diabetes is associated with statin adherence and it must be due to strict follow-up of patients with both cardiologists and endocrinologists. On the contrary, presence of hypertension is associated with statin discontinuation and this could be explained with possible polypharmacy as many patients with hypertension uses more than one drug for blood pressure control.

Age, years	63,3 ± 11,0
Female, n (%)	52 (27,8 %)
Current smoking, n (%)	84 (44,9 %)
Comorbidities, n (%):	
Hypertension	102 (54,5%)
Diabetes mellitus	60 (32,1 %)
CKD	11 (5,9 %)
Atrial fibrillation	6 (3,2 %)
Cerebrovascular disease	5 (2,7 %)
Follow-up, months, median	14 (6-22)
LDL, mg/dL, median (25-75 percentile)	
Baseline	135 (101-160)
Last visit	100 (85-124)
Statin type, n (%):	
Atorvastatin	153 (81,8 %)
Rosuvastatin	34 (18,2 %)
Statin dose intensity, n (%):	
Low	100 (53,5 %)
Moderate	72 (38,5 %)
High	15 (8 %)
Statin discontiuation, n (%)	49 (26,2 %)
Cause of statin discontinuation, n (%):	
Doctor recommendation (Due to low LDL level)	12 (6,4 %)
Elevated liver enzymes	5 (2,6 %)
Myalgia	7 (3,7 %)
Pruritus	1 (0,5 %)
Own request	24 (12,8 %)
Coronary angiography datas:	
Indication, (Acute coronary syndrome)	61 (32,6 %)
Number of stents, median	1 (1-2)
Percent maximum stenosis	80 (70-95)
PCI performed vessel;	
-LMCA	3 (1,6 %)
-LAD	101 (54,0 %)
-Cx	33 (17,6 %)
-RCA	64 (34,2 %)
Abbreviations; CKD: Chronic kdiney disease; LDL: Low density lipoprotein; PCI: Percuatneous coronary intervention; LMCA: Left main coronary artery; LAD: Left anterior descending artery; Cx: Circumflex artery; RCA: Right coronary artery	

Table 1. Baseline characteristics of the patients (n=187)

	Univariate Analysis		Multivariate Analysis	
	HR (95% CI)	p value	HR (95% CI)	p value
Age	1,02 (0,99-1,05)	0,129	1,02 (0,99-1,05)	0,179
Hypertension	2,49 (1,17-5,27)	0,017	2,82 (1,27-6,2)	0,010*
Diabetes mellitus	0,39 (0,16-0,96)	0,041	0,27 (0,11-0,69)	0,006*
Abbreviations: CI: Confidence interval; * p values < 0.05 were defined as statistically significant				

Table 2. Factors associated with stain discontinuation

Lipid / Protective Cardiology

PB-087

Cardiovascular health attitudes of the geriatric population during "Stay at Home" period of COVID-19 pandemic

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Background and Aim: COVID-19 pandemic may cause major attitude and behavioral changes, especially in the elderly population. We aimed to examine the attitude changes of the geriatric population who previously had received training on cardiovascular health in the first three ("stay-at-home") months of COVID-19 pandemic in Turkey.

Methods: This cross-sectional study was a questionnaire conducted with ≥65-year-old attendees of daycare centers/nursing homes in certain municipalities in Istanbul city, via telephone interviews during COVID-19 pandemic's "stay-at-home" period. The participants had previously attended a structured awareness and training program on atrial fibrillation risk factors and a heart healthy life style project (WebsAFIs; know your heart rhythm) that was held just before the pandemic. The nurses of the project were in touch with attendees during the pandemic and kept on giving advices on heart heathy living. The questionnairewas conducted during the pandemic and health-related attitudes of the participants were described and compared based on their age group and clinical conditions.

Results: Women constituted 66.3% of the participants and the mean age was 71.6±4.4 years. We detected that 60.6% of the respondents had hypertension, 26.0% diabetes, and 21.2% vascular disease. The mean body mass indices in March (28.2±3.8 kg/m2) and June (28.5±4.0 kg/m^2) were similar (p>0.05). Those who would/did not do exercise increased from pre-pandemic 12.5% to 46.2% (p<0.01) during stay-at-home period, which did not differ based on the age group or examined medical conditions. More diabetic patients stated to have unhealthier diet than their non-diabetic counterparts during stay-at-home period (25.9% and 9.1%, respectively; p<0.02). Half of the respondents (50.5%) stated that they were very much worried about the health of their relatives. Near third-quarter (73.1%) of participants were using cardiovascular system drugs whereas 34.6% were using vitamin/mineral/food supplements.

Conclusions: Though the exercise habits of the geriatric population appear to change negatively, no serious problems tend to exist regarding nutrition, cardiovascular health, and healthcare utilization. Body weight was also maintained in spite of decreased activity. The results of the questionnaire in this select group with a previous training on cardiovascular awareness just before the pandemic underlines the importance of patient empowerment in cardiovascular prevention.

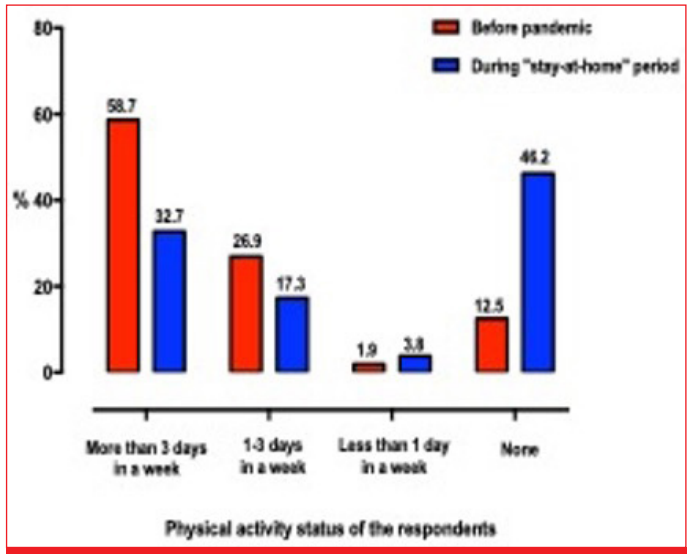

Figure 1. Distribution of changing in exercise routine during "stay-at-home" period.

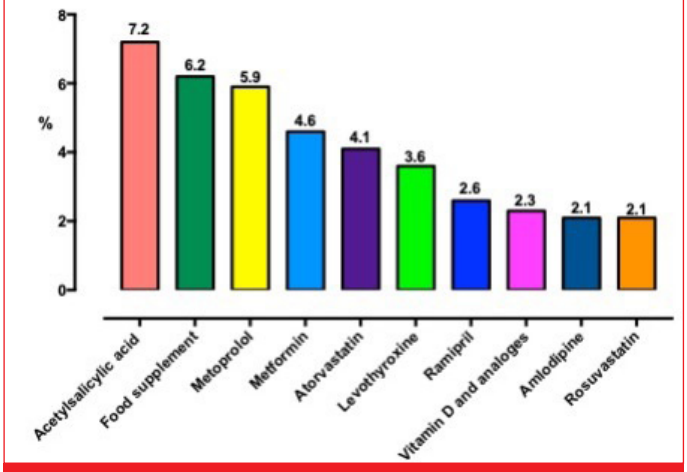

Figure 2. Distribution of the first ten participant-reported drugs used during the "stay-at-home" period.

Lipid / Protective Cardiology

PB-088

The relationship between endothelial functions with abdominal fat accumulation in obese individuals without overt heart disease

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Background and Aim: Percentage flow-mediated dilation (FMD%) has been defined as measure the ability of the arteries to respond with endothelial nitric oxide release during reactive hyperemia (flow mediated) after a 5-minute occlusion of the brachial artery with a blood pressure cuff. In this study we aimed to assess whether endothelial functions were impaired in patients with obesity without overt atherosclerosis and their relationship with abdominal fat accumulation.

Methods: 97 individuals with body mass indices higher than 26 m/kg^2 without overt atherosclerosis (80 F, 17 M; mean age 45.3±10.9 years) and 49 controls wtihout anmy cardiovascular disease and/or systemic disease (31 F, 18 M; 42.4±10.2 years) were included to the study. Body mass indices were calculated according to the formula BMI = kg/m^2 where kg is a person's weight in kilograms and m2 is their height in metres squared. The brachial artery diameter was measured between the arterial lumen-wall interface of the front and posterior walls at the end of diastole. The mean diameter was calculated in four cardiac cycles identified using the R wave of the electrocardiogram. Percentage changes in the brachial artery diameter were calculated compared to the first baseline diameter according to the formula: FMD=[(diameter after decompression–baseline diameter) / baseline diameter]x100. Ultrasonographic measurements were scanned with the L5–12 MHz linear transducer. Perirenal fat thickness was the maximal distance between the posterior wall of the kidney and the inner limit of the abdominal wall on a slice passing through the renal vein. All three aforementioned measurements were made on both sides for each patient. Pre-peritoneal fat thickness were measured in longitudinal and transverse sections in the midline of the abdomen, between the xiphoid process and umbilicus. The maximum thickness of the preperitoneal fat was measured three times, and the mean value was taken. laboratory tests were obtained using blood drawn from a vein after a minimum 8-hour fasting.

Results: Age, diastolic blood pressure and smoking status were similar between the groups (Table 1). Systolic blood pressure, heart rate, glucose, BMI, waist circumference and HbA1C levels were higher in group 1 compared to group 2 (Table 1). Perirenal and pre-peritoneal fat thickness were higher in group 1 compared to group 2. FMD were impaired in group 1 compared to group 2. FMD percentages were significantly and negatively correlated with perirenal and pre-peritoneal fat thickness (r=-0.44; p<0.001, r=-0.41; p<0.001, respectively). FMD was also significantly and negatively correlated with BMI and waist circumference (r=-0.43; p<0.001, r=-0.50; p<0.001, respectively).

Conclusions: In obese individuals endothelial functions were impaired before overt atherosclerotic disease occur. FMD levels were significantly correlated with perirenal and pre-peritoneal fat thickness. These parameters may be important in monitoring and treatment before overt atherosclerotic diseases occur.

Table 1. Demographic, laboratory features, flow mediated dilatation percentages, perirenal and pre-peritoneal fat thickness and aortic elastic properties of the groups.

	Group 1 (n=97)	Group 2 (n=49)	*P*
Age (years)	45.3±10.9	42.4±10.2	0.117
Gender (F,n)	80	31	0.011
Smoking (n)	35	24	0.135
Systolic blood pressure (mm Hg)	128.7±18.0	113.9±10.6	<0.001
Diastolic blood presure (mm Hg)	78.8±10.5	78.712.8	0.843
Heart rate (beat/min)	81.9±11.9	77.3±6.5	0.013
BMI (kg/m^2)	33.7±3.6	22.1±1.7	<0.001
WC (cm)	105.1±10.6	84.6±7.6	<0.001
Glucose (mg/dL)	103.3±29.8	91.0±8.7	0.006
Total cholesterol (mg/dL)	209.5±46.2	198.3±40.9	0.156
Triglyceride (mg/dL)	144.8±108.8	88.3±50.7	0.001
LDL cholesterol (mg/dL)	130.2±37.6	125.2±35.1	0.435
HDL cholesterol (mg/dL)	51.8±15.5	55.6±13.0	0.144
HbA1C (%)	5.8±0.8	5.3±0.3	<0.001
FMD (%)	8.7±5.9	18.7±10.1	<0.001
Perirenal fat thickness (cm)	5.7±1.5	2.3±0.9	<0.001
Pre-peritoneal fat thickness (cm)	5.2±3.1	2.9±0.4	<0.001

Abbrevations: BMI; body mass index, WC; waist circumference, LDL; low density lipoprotein, HDL; high density liporotein; HbA1C; glycated haemoglobin, FMD; flow mediated dilatation

Lipid / Protective Cardiology

PB-089

The relationship of aortic elastic functions with new body composition parameters in individuals with overweight metabolic syndrome

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Background and Aim: Metabolic syndrome (MetS) is a complex disorder defined by a cluster of interconnected factors that increase the risk of cardiovascular atherosclerotic diseases and diabetes mellitus type 2. Metabolic syndrome is closely linked to overweight or obesity and inactivity, and cardiovascular diseases can be seen in a wide spectrum in individuals with MS. The aim of this study is to investigate aortic elastic functions in individuals with obese and non-obes MS individuals and to examine which new body composition index is more closely related to these functions.

Methods: 112 overweight individuals (60 F, 52 M, mean age; 42.8±12.4 years) newly diagnosed with metabolic syndrome according to IDF diagnostic criteria were included in the study. None of the patients were using antihypertensive and/or oral antidiabetic drugs. Systolic and diastolic blood pressures of all patients were measured in the sitting position. Height was measured with a stadiometer to the nearest 0.1 without shoes and hair accessories. Weight was measured to the nearest 0.1 kg, in light clothing and without shoes, using a digital scales. Waist circumference was measured using a standard tape measure. Body mass index (BMI), body roundness index (BRI), body shape index (BSI), waist to hip ratio (WtHR), body adiposity index (BAI), ciscer-al adiposity index (VAI), conicity index (CI), abdominal The volume index (AVI) parameters were calculated according to their formulas. All individuals were divided into 2 groups as group 1; obes MS (52 pts; 31 F, 21 M, mean age: 46.1±14.1 years) and group 2; non-obes MS (60 pts; 34 F, 26 M, mean age: 41.7±13.2 years). In addition to standard echocardiographic parameters, aortic diameters were measured at the end of systole and diastole with transthoracic echocardiography. Aortic elastic functions (aortic strain, aortic distensibility, aortic stiffness index) were calculated from these parameters.

Results: There was no difference between the groups in terms of age and gender. Systolic, diastolic blood pressures, fasting glucose, triglyceride and HbA1C values were higher in group 1. High density lipoprotein was lower in group 1 than in group 2 (Table 1). Aortic elastic functions were more impaired in group 1 than in group 2. In the correlation analysis, all BCI parameters were correlated with aortic elastic functions (Table 2). The highest correlation was found with WtHR and AVI.

Conclusions: Aortic elastic functions in individuals with metabolic syndrome are more impaired in obese individuals than in non-obese individuals. Among the BCIs most paremeter associated with aortic elastic functions are WtHR and AVI. New BCIs should be considered more prominent than conventional parameters (such as BMI, WC) in follow-up and treatment.

Table 1.	**rho**	**p**
Aortic strain		
BMI (kg/m^2)	-0.32	<0.001
WC	-0.39	<0.001
WTHR	-0.42	<0.001
BRI	-0.39	<0.001
AVI	-0.43	<0.001
Aortic distensibility		
BMI (kg/m^2)	-0.39	<0.001
WC	-0.45	<0.001
WtHR	-0.50	<0.001
BRI	-0.46	<0.001
AVI	-0.51	<0.001
Aortic stiffness index		
BMI (kg/m^2)	0.36	<0.001
WC	0.43	<0.001
WtHR	0.46	<0.001
BRI	0.43	<0.001
AVI	0.46	<0.001

Table 2.

	Group 1 (n=52)	**Group 2 (n=60)**	**p**
Age (years)	46.1±14.1	41.7±13.2	0.082
Gender (F,n)	31	34	0.104
Smoking (n)	16	20	0.480
Systolic blood pressure (mm Hg)	138.7±19.4	123.2±18.6	<0.001
Diastolic blood pressure (mm Hg)	82.4±11.5	75.7±10.4	0.002
Heart rate (beat/min)	81.4±11.4	80.8±11.3	0.760
BMI (kg/m^2)	37.2 ±6.4	25.6±2.9	<0.001
WC (cm)	111.8±12.4	88.1±11.7	<0.001
Waist to Hip ratio	0.69±0.09	0.52±0.07	<0.001
BSI	0.079±0.005	0.078±0.006	0.476
BRI	7.83±2.51	3.95±1.35	<0.001
VAI	2.87±1.65	1.58±1.01	<0.001
BAI	40.1±9.0	28.1±4.1	<0.001
CI	1.32±0.09	1.23±0.11	<0.001
Glucose (mg/dL)	133.6±63.6	99.1±32.2	0.001
Total cholesterol (mg/dL)	221.6±41.0	207.9±46.9	0.115
Triglyceride (mg/dL)	172.1±19.4	114.7±61.1	<0.001
LDL cholesterol (mg/dL)	142.1±38.7	133.3±38.2	0.241
HDL cholesterol (mg/dL)	44.4±9.3	51.9±12.3	<0.001
HbA1C (%)	7.2±2.0	6.0±1.2	0.003
Aortic strain (%)	6.61±5.11	10.86±8.52	0.002
Aortic stiffness index	3.51±0.81	2.99±0.89	0.002
Aortic distensibility (cm−2dyn−110−6)	0.25±0.20	0.48±0.37	<0.001

Demographic, laboratory features and aortic elastic properties of the groups

Table 3. Demographic, laboratory features and aortic elastic properties of the groups.

	Group 1 (n=52)	Group 2 (n=60)	p
Age (years)	46.1±14.1	41.7±13.2	0.082
Gender (F,n)	31	34	0.104
Smoking (n)	16	20	0.480
Systolic blood pressure (mm Hg)	138.7±19.4	123.2±18.6	<0.001
Diastolic blood presure (mm Hg)	82.4±11.5	75.7±10.4	0.002
Heart rate (beat/min)	81.4±11.4	80.8±11.3	0.76
BMI (kg/m^2)	37.2 ±6.4	25.6±2.9	<0.001
WC (cm)	111.8±12.4	88.1±11.7	<0.001
Waist to Hip ratio	0.69±0.09	0.52±0.07	<0.001
BSI	0.079±0.005	0.078±0.006	0.476
BRI	7.83±2.51	3.95±1.35	<0.001
AVI	25.4±5.8	15.9±3.9	<0.001
BAI	40.1±9.0	28.1±4.1	<0.001
CI	1.32±0.09	1.23±0.11	<0.001
Glucose (mg/dL)	133.6±63.6	99.1±32.2	0.001
Total cholesterol (mg/dL)	221.6±41.0	207.9±46.9	0.115
Triglyceride (mg/dL)	172.1±19.4	114.7±61.1	<0.001
LDL cholesterol (mg/dL)	142.1±38.7	133.3±38.2	0.241
HDL cholesterol (mg/dL)	44.4±9.3	51.9±12.3	<0.001
HbA1C (%)	7.2±2.0	6.0±1.2	0.003
Aortic strain (%)	6.61±5.11	10.86±8.52	0.002
Aortic stiffness index	3.51±0.81	2.99±0.89	0.002
Aortic distensibility (cm−2dyn−110−6)	0.25±0.20	0.48±0.37	<0.001

Abbrevations: BMI; body mass index, WC; waist circumference, BSI; body surface index, BRI; body roundness index, AVI; abdominal volume index, BAI; body adiposity index, CI; conicity index, LDL; low density lipoprotein, HDL; high density liporotein; HbA1C; glycated haemoglobin

Table 4. Spearman's correlation of aortic strain, aortic stiffnes index and aortic distensibility

	rho	p
Aortic strain		
BMI	-0.32	<0.001
WC	-0.39	<0.001
WtHR	-0.42	<0.001
BRI	-0.39	<0.001
AVI	-0.43	<0.001
Aortic distensibility		
BMI	-0.39	<0.001
WC	-0.45	<0.001
WtHR	-0.50	<0.001
BRI	-0.46	<0.001
AVI	-0.51	<0.001
Aortic stiffness index		
BMI	0.36	<0.001
WC	0.43	<0.001
WtHR	0.46	<0.001
BRI	0.43	<0.001
AVI	0.46	<0.001

Abbrevations:BMI; body mass index, WC; waist circumference, BRI; body roundness index, WtHR; waist to height ratio, AVI; abdominal volume index

Pulmonary Hypertension / Pulmonary Vascular Dis.

PB-090

Iron deficiency in pulmonary arterial hypertension

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Background and Aim: Iron deficiency is a commonly reported condition in Pulmonary Arterial Hypertension (PAH) patients. However, the effects of iron deficiency on cardiac performance and functional capacity in this patient group are not clearly known. The aim of this study was mainly to evaluate the effects of iron deficiency on functional capacity and symptoms in a population of patients with pulmonary arterial hypertension.

Methods: A total of 38 PAH patients were retrospectively examined from hospital records. Iron deficiency in PAH is defined as serum ferritin levels <100 µg/L or transferrin saturation (TSAT) <20% if the ferritin level is 100-299 µg/L.

Results: Mean age of patients was 62±14 years (14% males, 86% females). The mean ferritin values of the patients included in the study were determined as 100,6 µg/L. Iron deficiency was present in 29 patients (76.3%). 6 minutes walking distance is lower than those who are not in the group with iron deficiency [246 m vs 358 m, p:0.021]. In addition, BORG dyspnea score were higher in the iron deficient group [13.7±3.8 vs 10.6±3.0, p:0.028]. Table 1 presents all of the other baseline clinical characteristics of the patients. In the correlation analysis, a statistically significant positive correlation was found between serum ferritin levels with 6-minutes walking distance and hematocrit levels. In addition, a statistically significant negative correlation was found between serum ferritin levels with BORG dyspnea score and NT-proBNP levels (Table 2).

Conclusions: Iron deficiency is highly prevalent in patients with PAH and is associated with worse clinical conditions. The exact mechanism causing iron deficiency is still unclear, iron uptake seems to be impaired in PAH patients, however further research is needed to confirm and clarify this. In this study, the negative effect of iron deficiency on functional status and disease severity was shown in PAH patients. Iron may have an important role in PAH patients with its anti-inflammatory and cardioprotective effect. There is a need for more comprehensive studies to explain this situation.

Table 1. Baseline characteristics of study patients

	Iron Deficient (n= 29)	Non-Iron Deficient (n=9)	*P*
Age (years)	60.1±14.3	66.7±15.2	0.246
Diabetes mellitus, n (%)	7 (24%)	4 (44%)	0.401
Vasoreactivity, n (%)	1 (3%)	1 (11%)	0.368
NYHA 3 or 4, n (%)	14 (48%)	2 (22%)	0.254
SPAP (mm Hg)	66.6±22.7	53.3±8.0	0.012
TAPSE (mm)	17.3±4.1	18.1±2.8	0.587
Pulmonary Artery Pressure (mm Hg)	44.4±17.9	41.8±16.8	0.701
Creatinine (mg/dL)	0.9±0.4	1.0±0.2	0.547
Na (mmol/L)	139.6±3.7	138.3±4.2	0.425
K (mmol/L)	4.7±0.6	4.7±0.5	0.860
6 Minutes Walking Distance (m)	246±128	358±82	0.021
BORG Dyspnea Score	13.7±3.8	10.6±3.0	0.028
NT-proBNP (pg/mL)	1725.4 (45 - 6645)	559.4 (112 – 1236)	0.127
Hemoglobin (g/dL)	11.5±1.6	13.9±2.0	<0.01
RDW	17.2±4.8	17.0±2.9	0.961
Iron (µg/dL)	37.2±23.4	63.9±35.8	0.061
Ferritin (ng/mL)	45.6 (7.2-240.5)	277.6 (113-592.3)	<0.01
MCV	85.6±7.5	86.5±11.1	0.771
Hematocrit	43.5±6.3	35.9±4.6	<0.01
TSAT (%)	10.4±5.5	23.7±9.4	0.038

Table 2. Spearman and Pearson Correlation coeffecients for ferritin

	Serum Ferritin Levels	p
Hematocrit	0.326	0.046
6 Minutes Walking Distance (m)	0.414	0.010
BORG Dyspnea Score	-0.417	0.028
NT-proBNP (pg/mL)	-0.315	0.040

Pulmonary Hypertension / Pulmonary Vascular Dis.

PB-091

Investigation of the effect of PAH specific therapy on blood pressure levels in patients receiving monotherapy or dual combination therapy

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Background and Aim: Pulmonary hypertension (PH) is a progressive disease which could be seen as a detrimental haemodynamic sequelae of various different underlying pathologies. Even though the clinical classification of PH has been updated at the 6th World Symposium on Pulmonary Hypertension, traditional five-group structure of PH classification has been preserved. Two of these five groups of PH, Group 1 and 4, differ from the other 3 groups in terms of available treatment options which are commonly known as pulmonary arterial hypertension (PAH)-specific therapies. Approved PAH-specific therapies have provided morbidity and mortality benefit in patients with Group 1 and 4 PH. However, these treatment options also have systemic side effects such as systemic hypotension, hepatotoxicity and reduction in hemoglobin concentration. In the light of the above-mentioned context, the aim of this study was to assess the potential effect of PAH-specific therapies on systemic blood pressure levels in patients receiving monotherapy or dual combination therapy.

Methods: Patients previously diagnosed with PAH or Group 4 PH who were receiving monotherapy or dual combination therapy were included in the study. Patients were classified as monotherapy if they received only one PAH-specific medical therapy. Patients were stratified as dual combination therapy if they received two PAH-specific drugs targeting different pathways implicated in disease progression. All patients underwent 24-hour ambulatory blood pressure monitoring (ABPM). 24-hour, daytime and nighttime blood pressures were calculated from ABPM recordings.

Results: A total of 44 patients (52.90 ± 14.29 years of age, 18.18% male) were included in the study. 52.27% of the patients were on monotherapy while the other 47.73% of the patients were on dual combination therapy (Table 1). Mean 24-hour, daytime and nighttime systolic blood pressure were significantly lower in patients receiving dual combination therapy compared to patients receiving monotherapy whereas mean 24-hour, daytime and nighttime diastolic blood pressure did not differ significantly among study groups (Table 2).

Conclusions: The findings of our study demonstrated that patients receiving dual combination therapy had reduced 24-hour, daytime and nighttime systolic blood pressure levels. These findings suggest that dual combination PAH-specific therapy could be associated with reduced systolic blood pressure levels and indicate that appropriate surveillance for hazardous systemic effects of PAH-specific therapies is warranted in patients receiving dual combination therapy.

Table 1. Baseline characteristics of the study population

Variable		Monotherapy mean ± SD or n (%)	Dual combination therapy mean ± SD or n (%)	p
Demographics	Age (years)	52.35 ± 12.68	53.58 ± 16.37	0.79
	Female n (%)	20 (86.96)	16 (76.19)	0.43
	Male n (%)	3 (13.04)	5 (23.81)	
Diagnosis	idiopathic PAH n (%)	6 (26.09)	5 (23.81)	0.78
	CTD-PAH n (%)	8 (34.78)	7 (33.33)	
	CHDPAH n (%)	4 (17.39)	5 (23.81)	
	CTEPH n (%)	5 (21.74)	4 (19.05)	
PAH-specific therapies	ERA n (%)	11 (47.82)	-	
	PDE-5i n (%)	6 (26.09)	-	
	SGCs n (%)	6 (26.09)	-	
	ERA+ PDE-5i n (%)	-	12 (57.14)	
	ERA+ SGCs n (%)	-	9 (42.86)	
Laboratory	BNP (pg/ml)	68.67 ± 102.96	97.66 ± 92.64	0.36

BNP = Brain natriuretic peptide, CHDPAH: Congenital heart disease associated pulmonary arterial hypertension, CTD-PAH: Connective tissue disease associated pulmonary arterial hypertension, CTEPH: Chronic thromboembolic pulmonary hypertension, ERA: Endothelin-receptor antagonists, PDE-5i: Phosphodiesterase type-5 inhibitors, SGCs: soluble guanylate cyclase stimulators

Table 2. The 24-hour ambulatory blood pressure parameters of the study groups

Variable	Monotherapy mean ± SD	Dual combination therapy mean ± SD	P
24-hour systolic BP (mm Hg)	114.17 ± 3.20	105.68 ± 7.45	<0.001
24-hour diastolic BP (mm Hg)	66.39 ± 5.48	63.37 ± 6.34	0.110
Daytime systolic BP (mm Hg)	115.57 ± 3.37	106.74 ± 7.94	<0.001
Daytime diastolic BP (mm Hg)	67.22 ± 5.76	64.21 ± 7.15	0.148
Nighttime systolic BP (mm Hg)	111.00 ± 5.04	103.63 ± 9.04	0.004
Nighttime diastolic BP (mm Hg)	64.30 ± 7.18	62.21 ± 7.82	0.376

BP: Blood pressure

Pulmonary Hypertension / Pulmonary Vascular Dis.

PB-092

A new Pulmonary Arterial Hypertension detection model using X-ray images

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Background and Aim: One of the hot-topic research areas is machine learning and it has been used in cardiology to help cardiologists and medical professionals. Moreover, machine learning has been used to develop wearable health assistants for monitoring the heart. Pulmonary Arterial Hypertension (PAH) is the hard cardiologic ailment for diagnosis. Therefore, artificial intelligence and machine learning models can be used to help cardiologist for diagnosis of PAH.

Methods: This research presents a new hand-modeled computer vision model to detect PAH accurately. We collected a new PAH X-ray dataset from Firat University Hospital retrospectively. In our collected dataset, there are 1178 images with two categories and these categories are PAH and healthy. The presented hand-modeled learning model is inspired by vision transformer (ViT). Local binary pattern is utilized as feature generator in our model and we used 16 x 16 sized patches to extract features. Therefore, this model is named Vision Local Binary Pattern (ViLBP). The extracted features from each patch are concatenated and final feature vector is created. To select the best 256 features are selected using Relief F feature selector and support vector machine is deployed to obtain classification results.

Results: ViLBP is applied to the collected X-ray image dataset and SVM classifier reached 98.98% classification accuracy on the collected dataset using 10-fold cross-validation.

Conclusions: Results denoted that the proposed ViLBP can detect PAH using chest X-ray images accurately.

Pulmonary Hypertension / Pulmonary Vascular Dis.

PB-093

How do we manage pulmonary hypertension? Assessment of knowledge, attitude and practice patterns among cardiologists and pulmonologists

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Background and Aim: Pulmonary hypertension (PH) is a heterogeneous, life-limiting condition defined by an elevated pulmonary artery pressure and, if untreated, results in right ventricular failure and death. There are limited data on how clinicians perceive the diagnosis and management of PH. Therefore, This study aimed to evaluate the doctors' knowledge, attitude and practice patterns for pulmonary hypertension in Turkey.

Methods: An internet-based survey, consisting of 31 questions in Turkish, was emailed to all members of the Turkish Society of Cardiology(TSC) and Turkish Thoracic Society (TTS) on 1 June 2021. The survey was shared again four weeks later via social media (Whatsapp) and was closed at the end of the eighth week.

Results: Of the 168 doctors who responded to the survey, were consisted of mainly pulmonologists (60.1%), practiced in education and training hospitals (41.1%). According to responses on the the questionnaires 50.6% of physicians had right cateterization laboratuary, 33.9% of physicians could consultate their PAH patients to lung transplantation center and 52.4% of physicians could consultate them to pulmonary rehabilitation center in their working city. Usually, doctors adviced not to become pregnant for women patients (61.3%). According to the participants ' responses, patients diagnosed with pulmonary arterial hypertension (PAH) often used monotherapy (71.4%). Additionally, Most of doctors primarily preferred endohelin receptor antagonists as monotherapy for vasoreactivity negative PAH. Endothelin receptor antagonists plus PDE-5 inhibors were mostly prefered as combined therapy. Guideline recommendation was the most important factor determining your specific drug choice in the treatment of PAH (98.1%). In patients receiving PAH treatment, adherence to treatment was mostly evaluated as moderate (62.7%). According to the participants, the main causes of non-compliance with treatment were assessed as lack of improvement in symptoms, drug side effects and difficulty in accessing drugs, associated tests, and a doctor.

Conclusions: Interest, knowledge, and awareness of the guidelines appear to influence physician attitude, and thus it is important to lay out educational materials and learning opportunities regarding PH for specialists. Availability of diagnostic and treatment facilities, poor medication adherence, and high cost of medications are seems to be important barriers to good practice. Addressing barriers to good practice is essential for the translation of knowledge into practice.

PB-094 [Pulmonary Hypertension / Pulmonary Vascular Dis.]

Risk assessment in chronic thromboembolic pulmonary hypertension

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Background and Aim: There is currently no recommendation regarding how to assess risk in patients with inoperable chronic thromboembolic pulmonary hypertension (CTEPH). Our objective was to investigate an abbreviated version of the risk assessment strategy proposed by the current European PH guidelines, noninvasive French model, and an abridged version of the REVEAL 2.0 risk score calculator, REVEAL Lite 2 for patients with inoperable CTEPH.

Methods: We enrolled inoperable CTEPH patients from 8 PAH centers from December 2009 to June 2020 (n=123). 70% of patients were treated with PAH therapies after initial evaluation at the discretion of treating physician. Noninvasive French model comprising WHO functional class (FC), 6-minute walk distance (6 MWD), and N-terminal pro–brain natriuretic peptide (NT-pro BNP) or BNP was used for 63 patients. REVEAL Lite 2 including FC, systolic blood pressure, heart rate, 6MWD, BNP/NT-pro BNP, and estimated glomerular filtration rate was used for 71 patients. Patients were grouped into three categories according to the number of non-invasive low-risk criteria (French model) and REVEAL Lite 2 scores. Risk was calculated based on the last available assessment at 12 months' follow-up. Kaplan-Meier (KM) survival was assessed in patients in each risk group with all-cause mortality as the end point. Log-rank test was used to compare estimates.

Results: The mean age was 64 ± 13 years at diagnosis. The median follow up was 22.7 months. 26 patients had died. None of the patients had balloon pulmonary angioplasty. Most patients had received monotherapy (55%). 23.8% of patients were WHO FC I-II, 65.6% III, and 10.7% IV at initial evaluation (Table). Both models discriminated risk in our cohort. Patients having none of low-risk criteria or high risk profile by REVEAL Lite 2 at follow-up had the worst survival. Figure demonstrates KM survival curves for noninvasive French model (A) and REVEAL Lite 2 (B). 37.3% of patients achieved 2 or more low risk criteria at follow-up. 44.8% of patients were in low risk at follow-up (REVEAL lite 2). The estimated survival rate at 5 years of patients meeting 2 and more low-risk criteria was 86% vs. 55% for patients meeting 0 low-risk criterion (p=0.04 by log-rank test). The corresponding survival rate was 45% for high-risk patient, 79% for intermediate-risk patient, and 82% for low-risk patient (REVEAL lite 2) (p=0.029 by log-rank test; figure 1). Survival was similar between treated and untreated patients (p=0.735 by log-rank test).

Conclusions: Both noninvasive French model and REVEAL Lite 2 may provide a simplified method of risk assessment for inoperable CTEPH. This analysis also supports the value of goal-oriented treatment in CTEPH. Patients who have not achieved low risk category at follow-up may benefit from escalation of their treatment regimen.

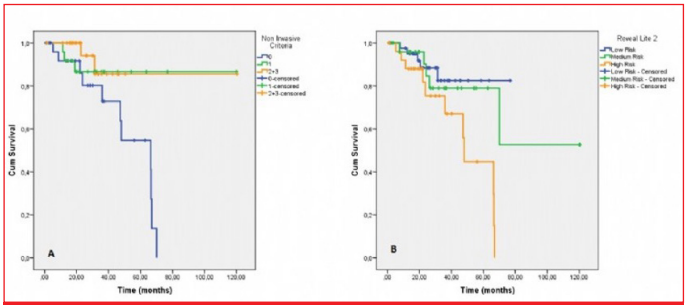

Figure 1. A, B, Kaplan-Meier survival curves obtained at follow-up for noninvasive French model (A) and Registry to Evaluate Early and Long-Term PAH Disease Management (REVEAL) Lite 2 (B).

Characteristic		n	%
Age, y	(mean ±S.D; Med (min - max)	64.08 ± 12.89	66 (25 - 88)
6MWD, m (n = 100)	(mean ±S.D; Med (min - max)	297.52 ± 138.49	307 (0 - 660)
NT-proBNP, pg/mL (n = 69)	(mean ±S.D; Med (min - max)	650.48 ± 784.43	343 (25 - 3563)
PAH Treatment	No therapy	37	30.1
	Mono therapy	67	54.5
	Combination therapy	19	15.4
Sex	Female	42	34.1
	Male	81	65.9
NYHA/WHO FC	I	3	2.5
	II	26	21.3
	III	80	65.6
	IV	13	10.7
Risk Categories at follow-up,			
The number of low-risk criteria	0	27	32.5
	1	25	30.1
	≥2	31	37.3
REVEAL Lite 2	Low	43	44.8
	Intermediate	27	28.1
	High	26	27.1

Values are No. (%) or mean SD. FC= functional class; NT-proBNP = N-terminal prohormone of brain natriuretic peptide; NYHA = New York Heart Association; PAH = pulmonary arterial hypertension; REVEAL = The Registry to Evaluate Early and Long-Term PAH Disease Management; 6MWD = 6-min walk distance; WHO = World Health Organization.
[a]Data at 1 y follow-up.

Table 1. Demographics and Clinical Characteristics of the Patients (n = 123) (Data at 1 y follow-up)

Other

PB-095

Electrocardiographic QRS axis shift, rotation and COVID-19

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Background and Aim: In patients with coronavirus disease (COVID-19), severe dyspnea is the most dramatic complication.Severe respiratory difficulties may include electrocardiographic frontal QRS axis rightward shift (Rws) and clockwise rotation (Cwr). This study investigated the predictability of advanced lung tomography findings with QRS axis shift and rotation.

Methods: This was a retrospective analysis of 160 patients. The patients were divided into two groups: normal oxygen saturation (SpO_2) (NS; n = 80) and low SpO_2 (LS; n = 80).They were then divided into NS Rws (n = 37), NS leftward shift (Lws; n = 43), LS Rws (n = 40), and LS Lws (n = 40) according to electrocardiographic follow-up findings. These groups were compared in terms of electrocardiographic rotation (Cwr, counterclockwise rotation, or normal transition), tomographic stage (CO-RADS5(advanced)/CO-RADS1–4), electrocardiographic intervals, and laboratory findings.

Results: In patients with LS, the amount of QRS axis shift (36.5° [23.2-50°] vs. 16.5° [12.2-24.2°]; p<0.001), Cwr (26 [65%] vs. 7 [17.5%]; p<0.001), and CO-RADS5/CO-RADS1–4 (30/10 [75%/25%] vs. 12/28 [30%/70%]; p<0.001) were significantly higher in the Rws group than in the Lws group. There were no differences in the above parameters between the Rws and Lws groups in patients with NS.Logistic regression analysis revealed that the presence of Cwr and Rws independently increased the risk of CO-RADS5 by 18.9 and 4.6 fold, respectively, in patients with LS.

Conclusions: In patients with COVID-19 who have dyspnea with LS, Cwr with QRS axis Rws demonstrated good sensitivity (80% [0.657–0.943]) and specificity (80%[0.552–>1])for predicting advanced lung tomographic findings.

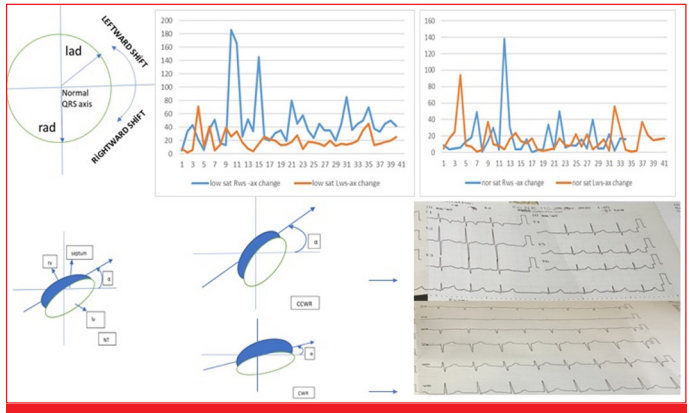

Figure1. Top row: Rightward axis shift (Rws), leftward axis shift (Lws). Axis shift rates in patients with low oxygen saturation (first figure) and normal oxygen saturation (second figure). Blue = Rws, orange = Lws. Bottom row: Rotation groups. Septal angle (α) between interventricular septum and horizontal body axis revealed significant differences among rotation groups. NT = normal transition, Ccwr = counterclockwise rotation, Cwr = clockwise rotation, Rv = right ventricle, Lv = left ventricle, top electrocardiogram = Ccwr, bottom electrocardiogram = Cwr

Table 1. Comparison of baseline electrocardiographic and laboratory characteristics according to QRS axis shift in the overall patient cohort.

	Rws (n=77)	Lws (n=83)	p
Age (year)	60 (50.5 - 67.5)	64.0 (56.0 - 71)	0.197†
Male factor	41 (53.2%)	51 (61.4%)	0.295‡
BMI (kg/m^2)	29 (27- 31)	29(28- 31)	0.597†
DM n (%)	20 (26.0%)	19 (22.9%)	0.788¶
HT n (%)	25 (32.5%)	26 (31.3%)	>0.999¶
HF n (%)	12 (15.6%)	18 (21.7%)	0.432¶
SpO_2 (%)	88 (83.5 - 95)	90 (82 - 95)	0.649†
Sbp (mm Hg)	114 (110 - 120)	125 (110 - 130)	0.002†
Dbp (mm Hg)	74 (69 - 80)	78 (70 - 85)	0.004†
Pulse (bpm)	87.3 (72.3 - 98)	89 (78.3 - 95.5)	0.437†
ECG .....PR (ms)	156.8 (137.4 - 167.3)	160.0 (146.8 - 176.8)	0.125†
QRS (ms)	94 (86 - 98)	94 (85 - 108)	0.645†
QTc (ms)	413 (397.0 - 430.5)	413 (401.3 - 425.3)	0.806†
DeltaQTc (ms)	11 (-0.5 - 24)	9 (-13 - 24)	0.229†
QTdisp (ms)	44 (42.0 - 44.3)	42.0 (40 - 44)	<0.001†
Tpe (ms)	83.3 (81.2 - 85)	80.2 (79.3 - 82.7)	<0.001†
Tpe/QTc	0.203±0.013	0.196±0.012	<0.001¥
Lab.....Glucose (mg/dL)	125 (104.3 - 175.5)	120.5 (96.8 - 155.8)	0.161†
Creatinine (mg/dL)	1.0 (0.8 - 1.2)	1.1 (0.9 - 1.3)	0.186†
Alt (lu/L)	41 (17.5 - 100.5)	27 (17.3 - 41.5)	0.003†
Ast (lu/L)	35(20.5 - 120)	27 (21 - 41.5)	0.006†
Crp (mg/L)	53.6 (10 - 86.8)	56 (10 - 78)	0.813†
hs-cTnl (ng/ml)	26.4 (3.4 - 890)	15 (4.3 - 32)	0.037†
Hemoglobin (g/dL)	12.8±2.2	11.9±2.7	0.018¥
Fibrinogen (mg/dL)	464.3±134.7	422.9±154.1	0.118¥
D-Dimer (ng/mL)	1028.5 (440. - 1888.8)	900 (463.5 - 1259)	0.360

† Rws: Rightward shift, Lws: Leftward shift, BMI: Body Mass İndex(kg/m^2), DM: Diabetes Mellitus, HT: Hypertension, HF: Heart failure, SpO_2: oxygen saturation, Sbp: Systolic blood pressure, Dbp: Diastolic blood Pressure, bpm: beat per minute, Alt: Alanine Aminotransferase, Ast: Aspartate Aminotransferase, Ggt: Gamma-glutamyl transferase, Alp: Alkaline phosphatase, Crp:C-reactive protein, hs-cTnl: High sensitivity-troponin I, WBC: White blood cells, † Mann Whitney U test, ‡ Pearson's χ^2 test, ¶ Continuity corrected χ^2 test, ¥ Student's t test.

Table 2. Comparison of mean axes, axes changes, rotation, and CO-RADS rates in the overall patient cohort, as well as in patients with normal and low oxygen saturation

All patients	Rws n=77	Lws n=83	p
Mean axis °	17.4±43.6	23.4±36.0	0.339†
QRS axis shift °	22.0 (6.5 - 44.0)	15.0 (7.0 - 21.0)	0.003‡
Rotation n % Normal	26 (33.8%)	45 (54.2%)	0.003¶
Clockwise	36 (46.8%)	18 (21.7%)	
Counter-clockwise	15 (19.5%)	20 (24.1%)	
Co-rads n % Non-advance	41 (53.2%)	65 (78.3%)	<0.001¶
Advance	36 (46.8%)	18 (21.7%)	
Normal saturation	n=37	n=43	

Table 2. Comparison of mean axes, axes changes, rotation, and CO-RADS rates in the overall patient cohort, as well as in patients with normal and low oxygen saturation (Continued)

All patients	Rws n=77	Lws n=83	p
Mean axis °	23.3±48.7	33.9±35.7	0.262†
QRS axis shift °	8.0 (4.0 - 17.5)	10.0 (4.0 - 17.0)	0.787‡
Rotation n % Normal	18 (48.6%)	22 (51.2%)	>0.999¶
Clockwise	10 (27.0%)	11 (25.6%)	
Counter-clockwise	9 (24.3%)	10 (23.3%)	
Co-rads n % Non-advance	31 (83.8%)	41 (95.3%)	0.135¥
Advance	6 (16.2%)	2 (4.7%)	
Low saturation	n=40	n=40	
Mean axis °	11.9±38.1	12.1±33.2	0.983†
QRS axis shift °	36.5 (23.2 - 50.0)	16.5 (12.2 - 24.2)	<0.001‡
Rotation n% Normal	8 (20.0%)	23 (57.5%)	<0.001¶
Clockwise	26 (65.0%)	7 (17.5%)	
Counter-clockwise	6 (15.0%)	10 (25.0%)	
Co-rads n % Non-advance	10 (25.0%)	28 (70.0%)	<0.001£
Advance	30 (75.0%)	12 (30.0%)	

Rws: Rightward shift, Lws: Leftward shift, † Student's t test, ‡ Mann Whitney U test, ¶ Pearson's χ^2 test, ¥ Fisher's exact test, £ Continuity corrected χ^2 test.

Table 3. The results of multiple logistic regression analysis determining the best predictors which discirimante the cases with advance CO-RADS from non-advance CO-RADS each other

All patients	Odds ratio	95% Confidence Interval	p
Clockwise rotation	7.621	2.345-24.765	<0.001
DM	4.437	1.243-15.843	0.022
Pulse	1.055	1.011-1.101	0.014
QRS axis shift	1.037	1.004-1.071	0.028
Ast	1.018	1.005-1.030	0.006
Normal saturation			
Tpe	1.830	1.238-2.703	0.002
Ast	1.050	1.012-1.090	0.010
Hemoglobine	0.542	0.329-0.892	0.016
Low saturation			
Rightward shift	4.665	1.172-18.565	0.029
Clockwise rotation	18.998	3.540-101.943	<0.001
DM	4.464	1.047-19.031	0.043

DM:Diabetes Mellitus Ast: Aspartate aminotransferase(lu/L) Tpe: T-peak to T-end interval(ms)

Other

PB-096

Machine learning algorithms for detecting risk of atrial fibrillation using baseline Holter ECG and echocardiography data

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Background and Aim: The use of artificial intelligence (AI) and related methods in cardiology over the past years has been increasing. Prediction of future arrhythmia and especially atrial fibrillation risk with AI as a decision support system may have great impact on clinical practice.

In this study our aim was to evaluate various machine learning (ML) algorithms and create a possible model for detecting future patients with atrial fibrillation (AF) using baseline echocardiographic and 24-h Holter sinus rhythm ECG data.

Methods: Holter ECG data of 1652 patients recorded 2 years ago (age: 53.2 ± 16.8 years), (female n: 994, 60.2%) were analyzed retrospectively and patients with echocardiographic examination within the similar time frame were invited to the hospital for 12 lead ECG recording to find out if there was clinical AF. 110 eligible patients (age: 55.6 ± 1 years), (female n: 69) were found. The ML model was created using 19 patients with AF and 19 without AF (n=38, 19 AF+, 19 AF-). Various ML algorithms were applied to determine the best performing model and different data sets were used for this purpose. Set 1, Set 2 and Set 3 were created with the echocardiographic and Holter data of the subjects. Set 3 contained both echocardiographic parameters and Holter data.

Results: Developed Bag ensemble model contributed the best performance with accuracy 87.9%. Seven subjects with AF could not be detected correctly by the model (Accuracy = 0.879, Specificity=1.000, F-measure=0.8677) (Fig 1 and Fig 2).

Model 8 and Model 9 was formed by Set 3 parameters like SDNN, SDNN index, rMSSD, pNN50, triangle, HF, LF, VLF, QT, QTc, maximum HR, mean HR were found to have the most relevant accuracy.

Conclusions: These findings propose a new method to identify patients with higher risk to develop AF using previous Holter ECG and echocardiography data. Our preliminary findings indicate that ML models using various baseline data could identify patients with the risk of AF development and could be used as decision support system in clinical practice if confirmed by large scale prospective studies.

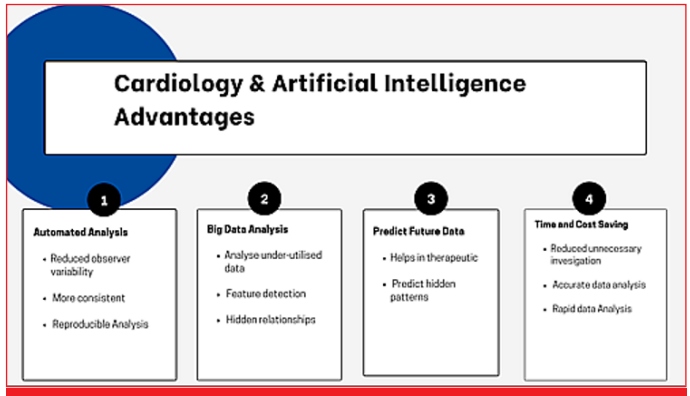

Figure 1. Possible advantages of AI and cardiologic interpretations
Possible advantages of AI and cardiologic interpretations can be grouped as: Automated Analysis, Big Data Analysis, Predict Future Data, Time and Cost Saving

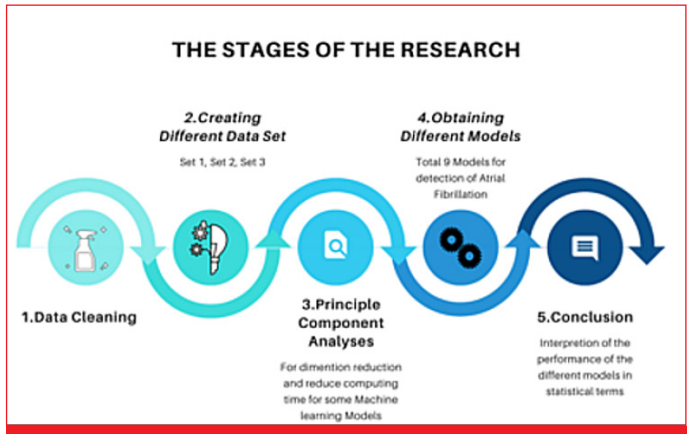

Figure 2. Model Creation and Evaluation Steps
5 creation steps of the proposed model: 1. Cleaning, 2. Creation of datasets, 3. Principle Component Analyses, 4. Creation of models 5. Conclusion of the proposed models

Model Number	Data Set	PCA Applied YES/NO	Machine Learning Algorithm	Accuracy
Model 1	Set 1	NO	Adaboost Ensemble Decision Tree	84.4%
Model 2	Set 1	NO	Support Vector Machine (Linear Kernel)	81,3%
Model 3	Set 1	NO	Support Vector Machine (Quadratic Kernel)	80,2%
Model 4	Set 1	NO	Medium Gaussian Kernel Support Vector Machine	80,2%
Model 5	Set 1	YES	Logi-Boost Ensemble	69.5%
Model 6	Set 2	NO	Logi-Boost Ensemble	83.3%
Model 7	Set 2	YES	Bag Ensemble	91.3%
Model 8	Set 3	NO	Bag Ensemble	84.8%
Model 9	Set 3	YES	Bag Ensemble	87.9%
Model 10	Set 4	NO	Gaussian Naive Bayes	83.3%
Model 11	Set 4	NO	Support Vector Machine (Linear Kernel)	84.4%
Model 12	Set 5	NO	RUS Boost Ensemble	79.2%
Model 13	Set 5	NO	Support Vector Machine (Linear Kernel)	81.3%

Table 1. Created ML models and related data sets.
Created ML models that had the best performance values were represented. Moreover, which data sets were used to create this machine learning models were represented, too.

Table 2. Information about data sets and numbers of AF and Non-AF measurements

Data Set Name	Data Contained	Number of Measurements for male Participants	Number of Measurements for female participants	Number of AF Observed Measurement	Number of Non-AF Observed Measurement	Total Measurement Number	Total Number of Variables in Dataset
Set 1	Holter	48	48	48	48	96	33
Set 2	ECHO	34	14	24	24	48	22
Set 3	Holter + ECHO	22	11	13	20	33	55

Information about data sets and numbers of AF and Non-AF measurements were listed in this table.There were mainly three datasets containing different information

	True Positive	False Positive	True Negative	False Negative	Accuracy	Precision	Sensitivity	F-Measure	AUC
Model 1	44	4	37	11	84.4%	90.2%	0,8	0,854	0,82
Model 2	45	3	33	15	85.4%	93.7%	0,75	0,833	0,81
Model 3	44	4	33	15	80,2%	91,6%	0,745	0,812	0,74
Model 4	43	5	34	14	80,2%	89,5%	0,754	0,801	0,82
Model 5	45	3	36	12	84.4%	93,7%	0,789	0,856	0,88
Model 6	42	6	38	10	83.3%	87.5%	0,807	0,839	0,87
Model 7	23	4	19	0	91.3%	85.19%	1.00	0.920	0.93
Model 8	13	4	15	1	84.85%	76.47%	0,986	0.838	0.88
Model 9	13	4	16	0	87.9%	76.47%	1	0.867	0.93
Model 10	35	4	44	13	82,3%	89.74%	0.729	0.805	0.82
Model 11	36	3	45	12	84.4%	93.75%	0.75	0.827	0.88
Model 12	14	0	24	10	79.2%	100%	0.583	0.736	0.69
Model 13	21	3	18	6	81.3%	85.71%	0.777	0.823	0.84

Table 3. Best performed models in terms of statistical measurement methods
Best performed models in terms of statistical measurement methods.

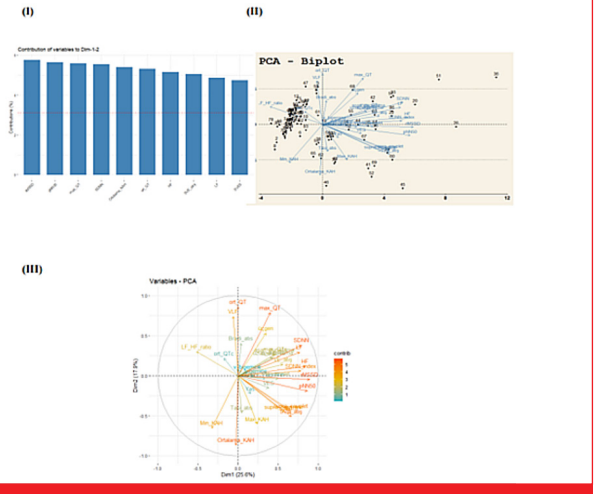

Figure 3. PCA analysis and most contributers based on HOLTER datasets
PCA analysis and most contributors based on HOLTER datasets. (I) Top 10 contributor in PCA. (II) The variables obtained from the whole sampling set and the analysis results of the sampling set. (III) The PCA additive states of all the variables used contributed more to warm colors.
Figure 3: PCA analysis and most contributors based on HOLTER datasets

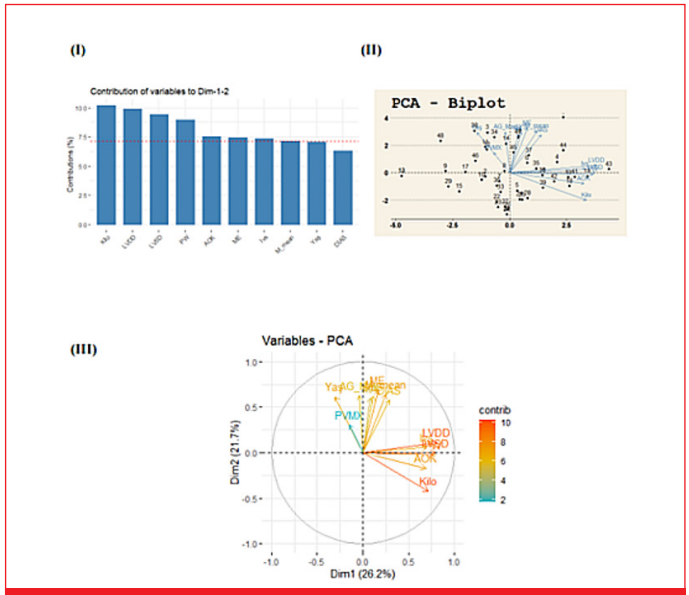

Figure 4. PCA analysis and most contributers based on ECHO datasets
PCA analysis and most contributors based on ECHO datasets. (I) Top 10 contributor in PCA. (II) The variables obtained from the whole sampling set and the analysis results of the sampling set. (III) The PCA additive states of all the variables used contributed more to warm colors.

Other

PB-097

Evaluation of diastolic ECG index in patients with subclinical hypothyroidism

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Background and Aim: Subclinical hypothyroidism (SH) is defined as abnormal thyroid-stimulating hormone (TSH) values with thyroxine and triiodothyronine levels within the normal range. Even these minor changes in the thyroid function in SH are associated with various kinds of cardiovascular disorders, such as heart failure, diastolic dysfunction, atrial fibrillation (AF), and coronary artery disease. In previous studies, patients with both overt and subclinical thyroid dysfunctions were associated with significant changes in several electrocardiographic (ECG) parameters including heart rate, QTc duration, P-wave duration, PR interval, low voltage. Besides these parameters, the relationship between diastolic ECG index and SH has not been evaluated before, so in this study, we aimed to investigate and compare the new ECG parameter, diastolic ECG index between SH and the control groups and to reveal the relationship between this parameter and the TSH levels in subclinical hypothyroidism patients.

Methods: This prospective study included 41 patients with SH and 39 healthy controls. The heart rate, PR interval, QRS interval, QT interval, QTc interval, Tend-P interval, and PQ interval were measured in 12-lead ECGs. Diastolic ECG index was defined as Tend-P/(PQ x age) (Figure 1).

Results: Baseline demographic, laboratory and electrocardiographic characteristics of the study groups are presented in Table 1. In electrocardiographic parameters, the Tend-P interval was significantly shorter in the patient group compared to the control group. The diastolic ECG index values were found to be significantly shorter in patients with SH compared to the control group. In the univariate correlation analysis, a negative correlation was found between serum TSH and diastolic ECG index (Figure 2).

Conclusions: In previous studies, impaired diastolic ECG index has been associated with atrial fibrillation and diastolic dysfunction. In our study, it was found to be significantly different in SH patients compared to the control group, and it was found to be correlated with TSH levels. The abnormal diastolic ECG index may be an indicator of electromechanical changes in the atria in SH patients and may contribute to understanding the pathogenesis of pathologies such as increased AF and diastolic dysfunction in SH patients.

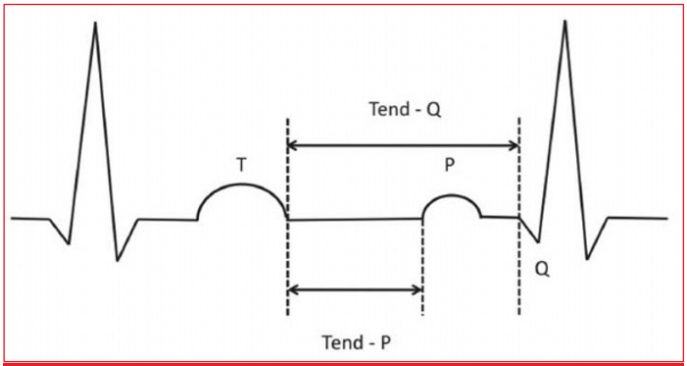

Figure 1.

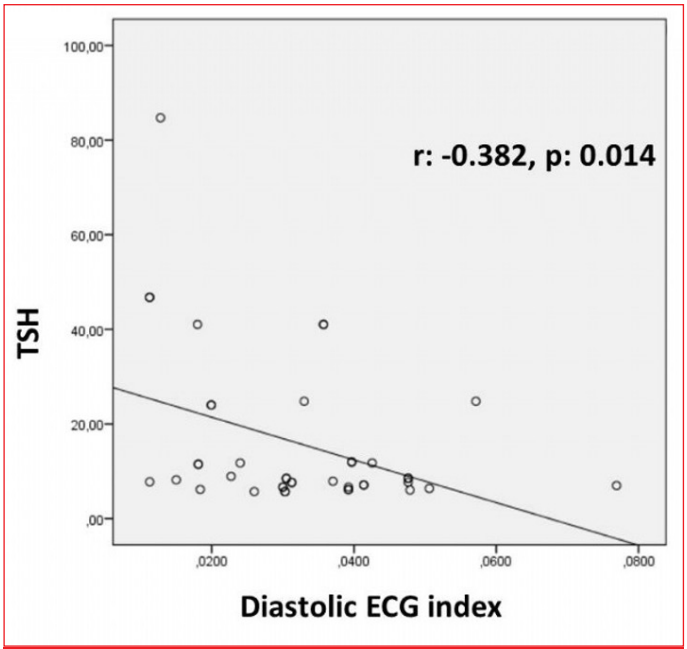

Figure 2.

Table 1

	Subclinical hypothyroidism patients	Control subjects	p
Age	46.3±13.7	44.1±12.1	0.214
Body mass index	28.2±5.1	29.2±6.6	0.525
Hemoglobin	13.6±1.4	13.1±1.8	0.405
White blood cell	8.9±3.1	8.0±2.4	0.673
Platelet	299.4±67.3	313.3±59.3	0.913
Creatinine	0.8 [0.7-1.2]	0.8 [0.6-1]	0.544
Aspartate aminotransferase	25.1±6.7	24.7±6.1	0.789
alanine aminotransferase	24.4±9.7	22.2±7.9	0.108
Total cholesterol	190.7±34.4	176.1±44.6	0.216
Triglyceride	145 [135-216]	125 [120-155]	0.079
High-density lipoprotein	45.9±10.7	46.1±5.1	0.208
Low-density lipoprotein	85.6±29.5	91.7±26.2	0.344
Glucose	111.3±46.8	104.7±29.3	0.634
Thyroid-stimulating hormone	8.5 [6.7-17.9]	2.3 [1,4-4,1]	<0.001
Heart rate	82.0±14.3	79.7±13.1	0.198
PR interval	152.0±18.9	150.1±15.8	0.407
QRS duration	83.1±10.6	81.3±9.6	0.156
QT interval	367.8±32.5	365.7±38.3	0.605
QTc interval	418.6±20.9	415.6±18.5	0.233
Tend-P interval	226.8±88.1	349.4±152.1	<0.001
P-Q interval	155.1±23.1	146.1±22.4	0.06
Diastolic ECG index	0.033±0.014	0.046±0.016	<0.001

PB-098 [Other]

The prognostic value of the TyG index in non-diabetic COVID 19 patients with myocardial injury

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Background and Aim: Insulin resistance (IR) is strongly associated with endothelial dysfunction. There is also evidence that endothelial dysfunction is associated with COVID 19 infection. Triglyceride glucose (TyG) index is newly defined promising surrogate index for IR as a cardiometabolic risk marker. It has been found to be associated with coronary artery calcification and high risk of cardiovascular disease. No data are currently available taking into account the effects of the TyG index on mortality in non-diabetic COVID 19 patients with myocardial injury. We aimed to investigate whether TyG predicts the in hospital mortality in non-diabetic COVID 19 patients with myocardial injury

Methods: This was a retrospective study. 218 non-diabetic patients who have myocardial injury due to COVID 19 infection were included in the study. Blood samples including

high-sensitivity (hs) cardiac troponin (cTn), triglycerides, eGFR, haemoglobin, platelet, D-Dimer, CRP, albumine, uric acid, ferritin and plasma fasting glucose (PFG) concentrations, were collected from the antecubital vein from each patient after at least 8 h of fasting. The TyG index was calculated as follows: log [serum triglycerides (mg/dL) × plasma glucose (mg/dL)/2]. We defined myocardial injury as cTn concentrations >99th percentile upper reference limit. The study cohort was divided into 2 groups as those survivors and non-survivors. Triglyceride and fasting blood glucose were evaluated in a separate multivariate analysis model (model 1). The receiver operating characteristics (ROC) curve analysis was used to evaluate the sensitivity and specificity of the TyG and it's cut-off value in determining the in-hospital mortality. Survival evaluations for the low- and high TyG groups were determined by using Kaplan–Meier and long-rank test

Results: 169 patients were survivors and 49 patients were non-survivors. D-Dimer and CRP levels were more higher in non-survivors group (p<0.01 and p<0.01 respectively). Non survivor patients had also higher TyG index than the others (p<0.01) (Table 1). Age, CHF, uric asid, TG, TyG were found to be independently associated with in-hospital mortality in univariate anaylsis. We used AUC value for diagnostic accuracies and discriminatory performances of the TyG (AUC:0.786, CI 95% 0.721-0.852, p<0.001), TG (AUC:0.738, CI 95% 0.656-0.820, p<0.001), and PFG (AUC:0.660, CI 95% 0.573-0.748, p=0.001) for detecting the in-hospital mortality. The receiver operating characteristics curve analysis revealed a cut off value of TyG index greater than 4.97 predicts the development of in hospital mortality in non-diabetic COVID 19 patients with myocardial injury with a 82% sensitivity, and a 66% specificity.

Variables	All population (n= 218)	Survivors (n= 169)	Non-survivors (n= 49)	p
Male gender, n (%)	142 (65.1)	110 (65.1)	32 (65.3)	0.978
Age, year, median, [IQR]	62 [54-74]	60 [51-73]	71 [61-82]	< 0.001
BMI	26.2±2.7	25.9±2.6	27.1±2.9	0.005
CAD, n (%)	50 (22.9)	31 (18.3)	19 (38.8)	0.003
CHF, n (%)	34 (15.6)	20 (11.8)	14 (28.6)	0.004
Hypertension, n (%)	89 (40.8)	66 (39.1)	23 (46.9)	0.323
CRF, n (%)	43 (19.7)	28 (16.6)	15 (30.6)	0.030
Current Smoking, n (%)	63 (28.6)	49 (29)	14 (28.6)	0.954
COPD, n (%)	48 (22)	31 (18.3)	17 (34.7)	0.015
Cancer, n (%)	25 (5.9)	15 (8.9)	10 (20.4)	0.026
CVA, n (%)	13 (6)	10 (5.9)	3 (6.1)	0.957
ACEI/ARB use history, n (%)	63 (28.9)	49 (29)	14 (28.6)	0.954
Needing ICU, n (%)	41 (18.8)	16 (9.5)	25 (51)	<0.001
Invasive mechanical ventilation n (%)	27 (12.4)	6 (3.6)	21 (42.9)	<0.001
ARDS, n (%)	33 (15.1)	15(8.9)	18 (36.7)	<0.001
MODS, n (%)	10 (4.6)	3 (1.8)	7 (14.3)	< 0.001
Acute kidney injury n (%)	10 (4.6)	4 (2.4)	6 (12.2)	0.004
Fatal ventricular arrhythmia	6 (2.8)	1 (0.6)	5 (10.2)	< 0.001
High grade AV Block	2 (0.9)	2 (1.2)	0 (0)	0.444
Hospitalization period, days, median, [IQR]	8 [5-14]	7 [5-13]	13 [9-16]	< 0.001
FBG, mg/dL , median, [IQR]	129.5 [95-167.5]	118 [93.5-153]	151 [107.5-223]	< 0.001
Triglyceride, mg/dL , median, [IQR]	163 [109-213]	144 [103-200]	216 [165-344]	< 0.001
TyG	4.94 [4.72 -5.25]	4.86 [4.66-5.10]	5.25 [5.0-5.52]	< 0.001
Uric acid, mg/dL	6.0±2.6	5.6±2.3	7.2±3.2	<0.001
eGFR, mL/min/1.73m², median, [IQR]	80 [60-96]	82 [63-100]	69 [42-83]	0.014
WBC, 10^3/uL, median, [IQR]	7.9 [5.4-11.1]	7.8 [5.1-10.1]	9.2 [6.5-13.5]	<0.001
Neutrophil, 10^3/uL, median, [IQR]	6.4 [3.8 -8.9]	5.9 [3.6-8.2]	7.3 [5.2-12.4]	< 0.001
Lymphocyte, 10^3/uL, median, [IQR]	0.9 [0.7-1.2]	1.0 [0.7-1.3]	0.7 [0.5-1.0]	<0.001
Hemoglobin, g/L	12.2±2.2	12.3±2.1	11.7±2.6	0.085
Platelet, 10^3/uL, median, [IQR]	212 [159-291]	211 [159-281]	219 [171-333]	0.537
D-Dimer, µg FEU/mL, median, [IQR]	0.8 [0.4-1.8]	0.7 [0.3-1.5]	1.5 [0.8-2.4]	<0.001
Ferritin, ng/mL, median, [IQR]	415 [156-778]	414 [158-757]	429 [138-1009]	0.364
CRP, mg/L, median, [IQR]	90 [36-243]	74 [32-155]	138 [64-243]	<0.001
Albumin, g/L	32.9±5.2	33.6±5.2	30.4±4.7	<0.001
hs-TnI, pg/mL, median, [IQR]	46.2 [27.8 -98]	45.8 [28 -91]	46.4 [24-118]	0.329

Figure 1. Demographic, admission clinical and laboratory parameters of the study cohort

Conclusions: A TyG above 4.97 was found as a risk factor for in hospital mortality in non-diabetic COVID 19 patients with myocardial injury. TyG may be a part of cardiovascular mortality to identify individuals at high risk for nondiabetic COVID 19 patients with myocardial injury

Variables	Univariate HR (95% CI)	p	Multivariate 1* HR (95% CI)	p	Multivariate 2* HR (95% CI)	p
Age	1.042 (1.017-1.067)	0.001	1.031 (1.005-1.057)	0.018	1.029 (1.004-1.054)	0.020
CAD	1.952 (1.088-3.502)	0.025	1.140 (0.587-2.214)	0.699	1.035 (0.536-1.999)	0.917
CHF	3.890 (2.029-7.461)	<0.001	2.273 (1.026-5.036)	0.043	2.316 (1.060-5.058)	0.035
Cancer	2.269 (1.126-4.570)	0.022	2.328 (1.066-5.086)	0.034	2.340 (1.112-4.924)	0.025
Uric acid	1.177 (1.076-1.288)	<0.001	1.139 (1.031-1.259)	0.010	1.126 (1.021-1.241)	0.018
FBG	1.004 (1.002-1.007)	0.001	1.003 (1.000-1.006)	0.090	-	-
TG	1.001 (1.000-1.003)	0.008	1.001 (1.000-1.003)	0.028	-	-
TyG	3.711 (2.174-6.336)	<0.001			3.780 (2.086-6.852)	<0.001

Figure 2. Factors that were found to be independently associated with in-hospital mortality in univariate anaylsis and in model 1 multivariate cox regression analysis model that does not include inflammation- based scoring systems

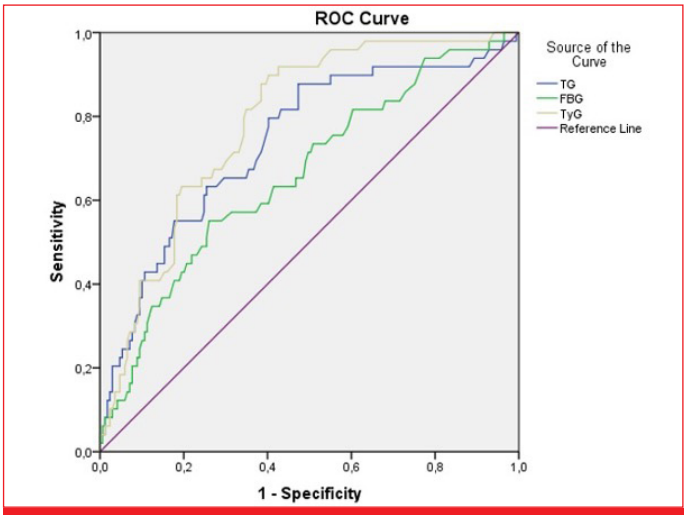

Figure 3. Diagnostic accuracies and discriminatory performances of the TyG (AUC:0.786, CI 95% 0.721-0.852, p<0.001), TG (AUC:0.738, CI 95% 0.656-0.820, p<0.001), and FBG (AUC:0.660, CI 95% 0.573-0.748, p=0.001) for detecting the in-hospital mortality by using AUC value. Cut off value =>4.97 with a 82% sensitivity, and a 66% specificity

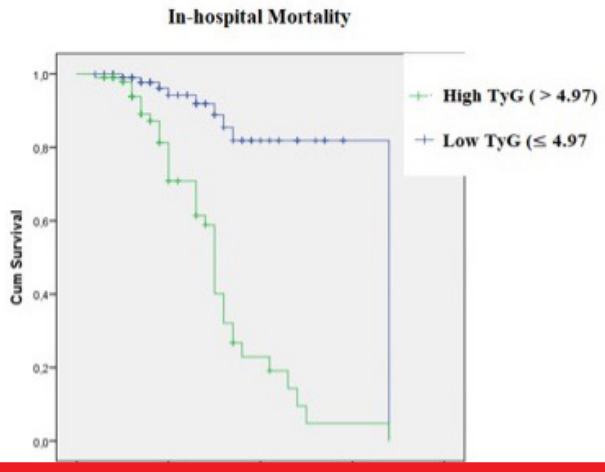

Figure 4. Kaplan–Meier plots of survival curves of patients with low (blue line) and high TyG (green line).

Other

PB-099

Evaluation of the relationship between serum Galectin-3 levels on admission and pneumonia severity in COVID-19 patients

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Background and Aim: A novel coronavirus disease 2019 (COVID-19) which was declared a pandemic in March 2020, has spread rapidly around the world and it is still threatening global health. COVID-19 infection may exhibit several clinical manifestations varying from mild respiratory illness to severe pneumonia and acute respiratory distress syndrome (ARDS). Many studies have shown that inflammatory responses play a pivotal role in the severity and prognosis of COVID-19 disease. Galectin-3, a b-galactoside-binding lectin, is a new important player in the pathophysiological processes of inflammation and fibrosis. In this study, we aimed to investigate the relationship between serum Galectin-3 levels at admission and pneumonia severity and inflammatory parameters in COVID-19 patients.

Methods: A total of 68 patients with laboratory, clinical and radiological confirmed COVID-19 were prospectively recruited to the study. The study population was classified into 2 groups as those with severe pneumonia (n=48) and those with mild pneumonia (n=20) based on chest computed tomography images at admission. Ten milliliter of peripheral venous blood were drawn within 24 hours of admission to estimate serum Galectin-3 levels. Patients with chronic renal failure, other inflammatory or rheumatic diseases and/or malignancies, cardiovascular diseases and diabetes mellitus were excluded from the study. We evaluated the relationship between Galectin-3 levels, pneumonia severity and laboratory parameters in COVID-19 patients.

Results: The demographic and clinical data and laboratory findings of the study population are presented in Table 1. The mean age of the study population was 61.68±14.4 years (45 male; 23 female). The mean level of Galectin-3 was 29.67±16.7 ng/mL. Serum Galectin-3, lactate dehydrogenase (LDH), C-reactive protein (CRP), prohormone B-type natriuretic peptide (pro-BNP), troponin-T, D-dimer and procalcitonin levels, white blood cell (WBC) counts, neutrophil and lymphocyte counts and percentages were significantly different between the groups (Table 1). Serum Galectin-3 levels were found to be higher in severe pneumonia group (p=0.04). In patients with mild pneumonia; Galectin-3 was negatively correlated with neutrophil percentage (r =-0.483 p=0.031) but was positively correlated with lymphocyte percentage and lymphocyte counts (r=0.428, p=0.05; r=0.554, p=0.011, respectively) (Table 2A). However, Galectin-3 was negatively correlated with WBC counts, neutrophil counts and neutrophil percentage (r=-0.317 p=0.028; r=-0.379 p=0.008; r=-0.609 p<0.001, respectively) and positively correlated with lymphocyte percentage (r=0.307; p=0.034) in COVID-19 patients with severe pneumonia (Table 2B).

Conclusions: In this study, we found a significant relationship between serum Galectin-3 levels and pneumonia severity in COVID-19 patients. Therefore, Galectin-3, a new biomarker of inflammation, may be useful as a prognostic factor in patients with COVID-19.

TABLE 1.	Total patients (n=68)	Severe Pneumonia (n=48)	Mild Pneumonia (n=20)	p-value
Clinical characteristics				
Age, (years)	61,68 ± 14,4	63,08 ± 15,2	58,3 ± 11,9	0,091
Gender Male, n (%)	45 (66,2%)	29 (60,4%)	16 (80%)	0,120
Female, n (%)	23 (33,8%)	19 (39,6%)	4 (20%)	
HR, (bpm)	87,86 ± 19,3	86,59 ± 19,1	90,8 ± 19,8	0,298
Temperature, (°C)	36,76 ± 0,6	36,68 ± 0,5	36,95 ± 0,8	0,091
SBP, (mmHg)	120,15 ± 18,7	119,41 ± 18,9	121,85 ± 18,6	0,823
sPO_2 (%)	89 ± 10,2	86,13 ± 10,9	95,45 ± 2,8	<0,001*
Laboratory findings at admission				
Fasting Plasma Glucose (mg/dl)	137,35 (76-710)	145,85 (81,8-710,4)	128 (76-321,9)	0,273
Bun (mg/dl)	17,9 (5,42-155,28)	20,35 (5,42-155,28)	16,17 (6,45-27,43)	0,021*
Creatinine (mg/dl)	1,1 ± 0,9	1,18 ± 1,1	0,92 ± 0,2	0,706
CKD-EPI (mL/min/1,73 m²)	78,77 ± 31,5	71,68 ± 36,5	86,15 ± 17,28	0,030*
Urea (mg/dl)	37,4 (11,6-332,3)	42,1 (11,6-332,3)	32,4 (13,8-54,3)	0,047*
Uric acid (mg/dl)	5,37 ± 2,2	5,62 ± 2,4	5,04 ± 1,2	0,510
Sodium (mmol/L)	136,6 ± 5,9	136,52 ± 6,2	137 ± 3,78	0,337
Potassium (mmol/L)	4,48 ± 0,6	4,46 ± 0,7	4,39 ± 0,4	0,649
AST (U/l)	32,8 (8-181)	37 (8-181)	27,85 (13-155,4)	0,106
ALT (U/l)	25 (5-194)	27,3 (5-194)	24,55 (12-139,5)	0,540
LDH (U/l)	338 (138-925)	372 (142-925)	266 (138-489)	0,001*
CRP (mg/L)	90,04 (2,63-404,64)	104,63 (19,73-404,65)	36,75 (0,75-206,98)	0,001*
D-dimer (µg/L)	990 (330-5460)	1326,55 (392,9-7810)	642,6 (330-2500)	0,002*
Ferritin (ng/ml)	678,5 (20,99-6162)	700,9 (20,99-3116)	383 (23,23-6162)	0,062
Hs-troponin-T (pg/ml)	5,75 (3-366,2)	9,04 (3-394,2)	4,07 (3-23,18)	0,014*
Pro-BNP (pg/ml)	287,5 (16,93-35000)	507,65 (36,41-35000)	128,9 (16,93-2418)	0,001*
Procalcitonin (ng/mL)	0,16 (0,02-2,38)	0,19 (0,03-2,38)	0,10 (0,02-1,73)	0,026*
Hgb (gr/dl)	11,56 ± 2,3	11,63 ± 2	11,87 ± 3	0,408
Hematocrit (%)	34,81 ± 6,4	35,09 ± 5,9	35,39 ± 7,8	0,567
WBC (10^3/µl)	9,17 ± 6,2	10,03 ± 6,1	7,18 ± 5,9	0,027*
RBC(10/µl)	4,08 ± 0,7	4,12 ± 0,7	4,13 ± 0,9	0,956
Neutrophil (10^3/µl)	5,9 (0,5-26,7)	6,75 (0,5-22)	3,5 (0,5-26,7)	0,002*
Lymphocyte (10^3/µl)	0,7 (0,1-2,2)	0,6 (0,2-2)	0,9 (0,1-2,9)	0,002*
Lymphocyte (%)	11,95 ± 8,4	9,61 ± 7,6	19,68 ± 10,1	<0,001*
Neutrophil (%)	79,32 ± 13,5	82,62 ± 12,9	69,34 ± 12,8	<0,001*
Platelet (10^3/µl)	260 (31-590)	266,5 (91-590)	225 (31-582)	0,086
Galectin-3 (ng/ml)	29,67 ± 16,7	31,46 ± 18	24,02 ± 12,3	0,040*

Table 1. Baseline demographic, clinical data and laboratory findings of study population at admission

	Variable	r	P value
TABLE 2A	Hgb (at admission)	0,431	0,058
	WBC (at admission)	0,077	0,745
	Neutrophil (at admission)	0,046	0,848
	Lymphocyte (at admission)	0,554	0,011*
Galectin-3 (mild pneumonia)	Neutrophil percentage (at admission)	-0,483	0,031*
	Lymphocyte percentage (at admission)	0,428	0,050*
	Albumin (at admission)	0,532	0,019

	Variable	r	P value
TABLE 2B	Hgb (at admission)	-0,184	0,210
	WBC (at admission)	-0,317	0,028*
	Neutrophil (at admission)	-0,379	0,008*
	Lymphocyte (at admission)	0,080	0,590
Galectin-3 (severe pneumonia)	Neutrophil percentage (at admission)	-0,609	<0,001*
	Lymphocyte percentage (at admission)	0,307	0,034*
	Albumin (at admission)	0,521	<0,001*

Table 2. Correlation of Galectin-3 with laboratory parameters according to pneumonia severity

Cardiovascular Nursing / Technician

PB-102

An effective educational tool in heart failure: mobile game

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Background and Aim: Heart failure (HF) is a complex clinical syndrome that is difficult to control and manage because of the physical and psychosocial problems it causes. As the disease progresses, the patient's quality of life is deteriorated due to an increase in the burden of symptoms, repeated hospitalizations, side effects due to the treatments used, and difficulties in adapting to the changes in lifestyle. Current guidelines emphasize the importance of patient education and post-discharge follow-up to ensure positive lifestyle changes in HF patients. The aim of the research planned in this direction is to evaluate the effectiveness of the mobile game applied to HF patients in the light of the literature.

Methods: This research was prepared by examining the articles scanned with related keywords.

Results: With the widespread use of mobile devices such as smartphones and tablets, new educational techniques in the field of health have gained popularity and game-based learning has begun to attract attention. In the field of health, play-based learning is used to create health information, disease control, management and positive behavior change. In addition, mobile health games are accepted as a creative and effective strategy in increasing access to health services, strengthening communication with the health care team, increasing adherence to treatment, and gaining health-related knowledge and skills.

There are limited studies in the literature examining the effectiveness of mobile gaming in patients with HF. In the study of Radhakrishnan et al., it was determined that the digital game structured in the Gagne Teaching Situations Model significantly increased the knowledge of the disease in elderly patients with HF, there were improvements in self-management behaviors, but there was no significant difference. In the same study, it was reported that the participants described playing digital games as easy, enjoyable and a useful tool for learning about HF. Klompstra et al., on the other hand, investigated the effect of the Nintendo Wii console game, which is a kind of exercise game and includes a motion-sensitive sensor, on exercise capacity and daily physical activity in patients with HF. At the end of the study, it was determined that there was no difference in daily physical activity, while an increase in exercise capacity was observed in the intervention group when compared with standard care.

Conclusions: With mobile games, health education can be provided to HF patients who need health care in a fun and motivating way, different from traditional learning processes and creatively. A limited number of studies show that mobile games, which emerged as an alternative educational approach, are an effective educational tool in HF patients.

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