Original Article

Relationship of MAPH Score with Left Ventricular Apical Thrombus and Adverse Events in Patients with Acute Anterior ST-Elevation Myocardial Infarction

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Abstract

Background: **Left ventricular apical thrombus (LVAT) is a clinically crucial complication of anterior ST-elevation myocardial infarction (STEMI). The MAPH score is a newly developed thrombosis-related score. MAPH scores are simple scores that estimate blood viscosity and thrombus sensitivity.**

Objective: **This study aimed to explore the relationship of MAPH score with LVAT and major adverse cardiovascular and cerebrovascular events (MACCE) in patients with acute anterior STEMI.**

Methods: **The study included 185 patients with acute anterior STEMI who underwent percutaneous coronary intervention (PCI). LVAT was detected and divided into two groups in clinical follow-up after PCI. MAPH score obtained before PCI was compared between the groups, and its relationship with MACCE was examined. The MACCE included a combination of all-cause death, non-fatal myocardial infarction, target vessel revascularization, heart failure, and stroke.**

Results: **MAPH score was higher in patients with LVAT than those without LVAT ($p < 0.001$). High MAPH score was determined to be a predictor of LVAT [odds ratio (OR): 1.265; 95% confidence interval (CI): 1.124-1.423; $p < 0.001$] and MACCE [hazard ratio (HR): 1.345; 95% CI: 0.984-1.790; $p = 0.004$] in patients with acute anterior STEMI. A high MAPH score was associated with a higher overall incidence of MACCE in Kaplan-Meier analyses. The cut-off values of the MAPH score for LVAT and MACCE were determined to be 2, based on receiver operating characteristic curve analysis.**

Conclusion: **A high MAPH score may be used for LVAT and MACCE risk assessment in patients diagnosed with acute anterior STEMI.**

Keywords: **Major Cardiovascular and Cerebral Events; Left Ventricular Apical Thrombus; Anterior Myocardial Infarction; MAPH Score.**

Introduction

Myocardial infarction (MI) is still the leading cause of death and morbidity internationally, despite advances in medical treatment and percutaneous coronary interventions.[1] Early revascularization is the preferred reperfusion strategy for patients with ST-segment elevation myocardial infarction (STEMI).[2] A poor prognosis is associated with comorbid conditions, infarct-related arteries (IRA), and a late revascularization time.[3]

Left ventricular apical thrombus (LVAT) is an important issue after acute anterior STEMI and is a risk factor for thromboembolic clinical outcomes.[4] Anticoagulant drugs are used to treat LVAT, and early identification of high-risk individuals is important in reducing thromboembolic events.[5] Previous studies indicate that mean platelet volume (MPV) and age are useful in demonstrating thrombosis.[6,7] The MAPH score, which is composed of MPV (M), age (A), total protein (P), hematocrit (H) components, is an updated score that has been found to be associated with thrombosis such as intracoronary thrombus burden in patients with acute coronary syndrome.[8,9] As far as we know, no study has examined the relationship of MAPH score with LVAT and major adverse cardiovascular and cerebrovascular events (MACCE) after acute anterior STEMI.

We aimed to explore whether there is a relationship between MAPH score and LVAT and MACCE development in patients with acute anterior STEMI.

Methods

Patients diagnosed with the first episode of acute anterior STEMI who underwent coronary angiography (CAG) between January 2015 and January 2023 were

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Central Illustration: Relationship of MAPH Score with Left Ventricular Apical Thrombus and Adverse Events in Patients with Acute Anterior ST-Elevation Myocardial Infarction

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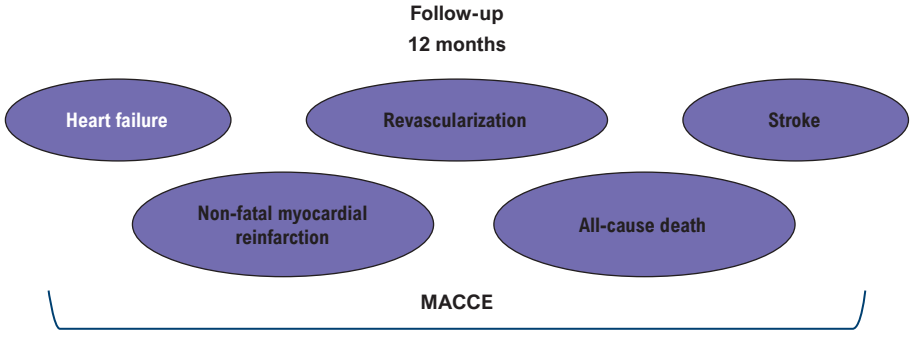
Relationship of MAPH score with left ventricular apical thrombus and adverse events in patients with acute anterior ST elevation myocardial

381 patients with acute anterior STEMI who underwent successful percutaneous coronary intervention were screened. A total of 196 patients were excluded due to one or more exclusion criteria. The study included 185 patients.

	LVAT (+) (n=35)	LVAT (-) (n=150)	p value
MAPH score	2.34±0.59	1.47±0.60	<0.001
LVEF, %	36.37±6.86	45.44±5.92	<0.001
Apical aneurysm, n (%)	22 (62.9)	12 (8)	<0.001

LVEF: Left ventricle ejection fraction; LVAT: Left ventricular apical thrombus.



	LVAT (+) (n=35)	LVAT (-) (n=150)	p value
MACCE, n (%)	23 (65.7)	37 (24.7)	<0.001

MACCE: Major adverse cardiovascular and cerebrovascular event

LVAT can lead to serious cardiovascular consequences such as systemic embolization, stroke, or even death.

MAPH scores can be calculated easily in routine practice. The test can be used to identify patients with anterior STEMI who are at high risk for LVAT.

High MAPH score my be used for LVAT and MACCE risk assessment in patients diagnosed with acute anterior STEMI.

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enrolled in this study. Due to the potential impact of previous apical aneurysms on the incidence of apical thrombus in the case of acute anterior MI, we only recruited patients with a first episode of acute anterior MI. During the study, 381 patients were screened. One hundred ninety-six patients were excluded due to one or more exclusion criteria. The study included 185 patients with acute anterior STEMI who underwent successful percutaneous coronary intervention (PCI).

Electrocardiography (ECG) samples were taken within 10 minutes of admission to the emergency department (ED) from patients who presented at the ED with chest pain. Before CAG, all patients underwent ECG (12-lead, 25 mm.sec-1 paper speed, and 10 mm.mV-1 calibration). The diagnosis of STEMI was made in patients consulting with chest pain in at least two adjacent derivations and patients with ST-segment elevation measured from the J point in 12-lead ECG (≥ 2.5mm in males under 40 years of age in

V2-V3 derivations, ≥ 2 mm in males over 40 years of age, ≥ 1.5 mm in females over 40 years of age and/or ≥1 mm in other derivations [A left bundle branch block (LBBB) or left ventricular hypertrophy (LVH) is not present]).[10]

The exclusion criteria were a history of coronary artery disease (CAD), coronary artery bypass graft (CABG), known heart failure with reduced ejection fraction [left ventricular ejection fraction (LVEF)≤ 40%], stroke, chronic kidney disease (eGFR < 30 [mL/min/1.73m2]), active infection, known coagulopathy, thrombocytosis, malignancy, moderate heart valve disease, mitral valve repair or a prosthetic valve, uncontrolled thyroid dysfunction, chronic inflammatory disease, hematological diseases, autoimmune diseases, recent blood transfusion, patients with known atrial fibrillation, pathological Q waves, Vaccination or infection history with COVID-19 in the last two months, using oral anticoagulants, patients without follow-up echocardiographic data and under 18 years of age were not included in the study.

The study was approved by the local ethics committee (approval no: 2023/03-05). The Declaration of Helsinki was complied with in all study procedures. Due to the retrospective study design, written informed consent was not obtained from the participants before the study.

Echocardiography evaluation

Blood pressure levels were measured just before starting echocardiographic imaging. Echocardiographic examinations were performed following the patients' blood pressure measurements. All patients underwent echocardiographic examinations within 24-36 hours after hospital admission. The left lateral position was used for echocardiographic measurements. We used modified Simpson's formula to calculate LVEF.[11] LVAT was defined as an echo-dense mass adjacent to an akinetic or dyskinetic myocardial segment seen throughout systole and diastole (usually apical and short axis)[12] (Figure 1). Various transthoracic echocardiography (TTE) gain settings, depth of field, and multiple imaging positions were used to exclude mimicking the LVAT, such as pseudo-tendons trabecular structures.[13] If a diagnosis of LVAT was suspected, patients underwent a second evaluation, such as computed tomography or cardiac magnetic resonance imaging. The same echocardiographic methods were used in clinical follow-up (3rd, 6th, and 12th months).

Coronary angiography

CAG was performed by expert operators using the standard Judkins technique. All patients were treated following the current guidelines of the European Society of Cardiology.[14,15] The patients were administered clopidogrel (600 mg), ticagrelor (180 mg), or prasugrel (60 mg) in addition to 300 mg aspirin for preprocedural antiplatelet therapy. Various image planes were considered while identifying the lesions responsible for infarction. After administering heparin (70 U/kg bolus) into the IRA, coronary revascularizations were performed with stents, while balloon predilatation was performed before coronary stenting for some lesions. In all patients without contraindications, isosorbide dinitrate was administered by an interventional cardiologist before the first angiographic images to exclude the coronary slow flow phenomenon. The intervention was terminated after obtaining images following the administration of isosorbide dinitrate in all patients without contraindications. All patients received guideline-recommended long-term drug therapy, including statins, beta-blockers, angiotensin-converting enzyme inhibitors, or angiotensin receptor blockers, as necessary.

MAPH score

Antecubital vein blood samples were obtained at admission to analyze biochemical and haemogram parameters. Laboratory tests were performed and analyzed

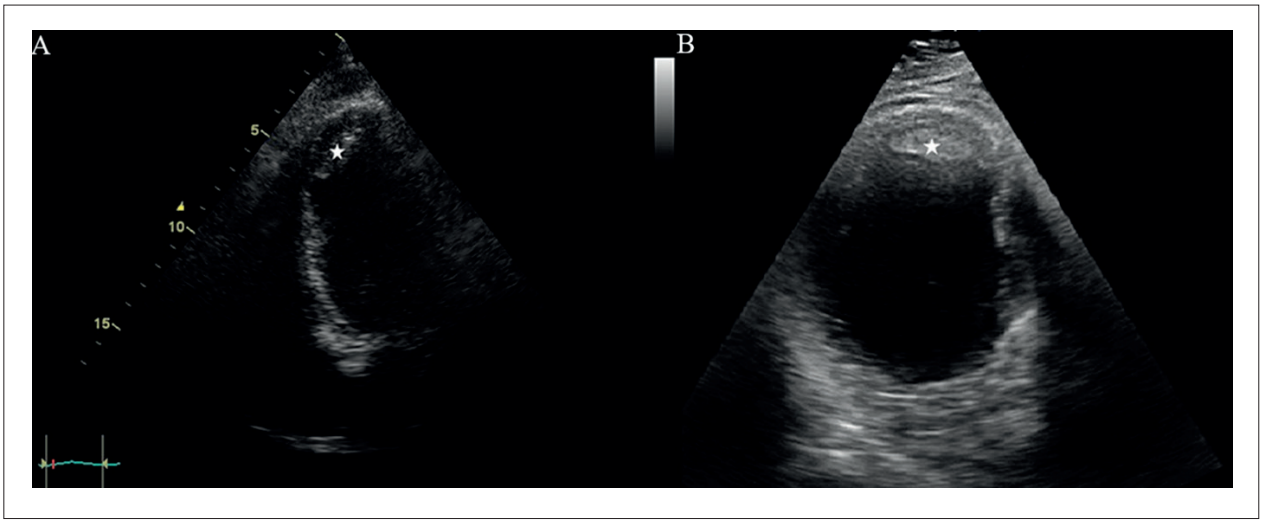

Figure 1 – *Transthoracic echocardiographic view of a thrombus (asterisk) in the apex of the left ventricle. A: 45-year-old male patient, B: 56-year-old female patient.*

using appropriate kits for measuring serum values of HCT, total protein, MPV, and other tests. Using the Youden index, we determined predictive cut-offs for MPV, age, total protein, and HCT for LVAT. Levels above the cut-off point were assigned 1 point, and levels below the cut-off point were given 0 points. The total MAPH score was obtained by summing the scores of these 4 variables (MPV, age, total protein, and HCT).[9]

Clinical follow-up and end points of the study

A regular record of clinical visits of patients was used to determine clinical outcomes. An occurrence of any of the following components during 12 months of follow-up was defined as MACCE: all-cause death, heart failure (HF), non-fatal myocardial reinfarction, revascularization, and stroke. In-hospital all-cause mortality refers to cardiovascular (including cardiac arrest, pulmonary edema, and cardiogenic shock) and non-cardiovascular death. HF was defined as new-onset HF in patients with no history of HF (LVEF ≤40%). Non-fatal myocardial reinfarction was defined as any subsequent hospitalization with a discharge diagnosis of myocardial reinfarction. Revascularization was defined as the need to restore lumen patency after lumen loss in the lesion responsible for the index infarct (re-intervention to address an acute re-occlusion within the previous stent). Neurological dysfunction caused by a focal cerebral, spinal, or retinal infarction was defined as a stroke.

Statistical analysis

Data was analyzed using SPSS version 19.0 (SPSS Inc., Chicago, IL, USA). The conformity of continuous variables to normal distribution was analyzed using the Kolmogorov–Smirnov test. Data were expressed as mean ± SD or median (interquartile range) values. Categorical variables were expressed as numbers (n) and percentages (%). Parameters showing normal distribution were compared using the Student's t-test, and those not showing normal distribution with the Mann-Whitney U-test. The Chi-squared test or Fisher's Exact test was used to compare the probability ratios of categorical variables.

The sensitivity and specificity of MPV, age, total protein, and HCT levels were demonstrated by receiver operating characteristic (ROC) curve analysis and the Youden index. To test the discriminative power of variables for LVAT, pairwise comparisons of ROC curves were used. To estimate LVAT and MACCE, possible confounding independent variables [e.g., age, current smoking, sex, LVEF, apical aneurysm, hypertension (HT), diabetes mellitus (DM), cardiac troponin (Tn), MPV, total protein, HCT, MAPH score] were included in the univariate analysis. Variables with a non-adjusted *P*-value less than 0.1 in the univariate analysis were determined as potential risk factors of LVAT and included in multivariate logistic regression analysis. Variables found to be statistically significant in univariate analysis were used in a multivariate Cox proportional hazards regression to determine the independent predictors of MACCE. The hazard ratio (HR) and its 95% CI were calculated. MACCE was analyzed separately using Kaplan-Meier curves. A value of $p < 0.05$ was considered statistically significant.

Results

We screened 381 patients within the study period. One hundred ninety-six patients were excluded because of the presence of one or more of the exclusion criteria. Patients with CAD (n=58), CKD (n=8), stroke (n=5), CABG (n=35), LVEF≤ 40% (n=8), permanent pacemaker (n=5), any cardiomyopathy (n=4), pathological Q waves (n=4), known AF (n=14), pathological Q waves (n=3) moderate heart valve disease, mitral valve repair or a prosthetic valve (n= 8), uncontrolled thyroid dysfunction (n=3), known coagulopathy and thrombocytosis (n=3), chronic inflammatory disease (n=2), hematological diseases (n=1), autoimmune diseases (n=2), active infection and/or malignant disease (n=9), recent blood transfusion (n=4), COVID-19 infection or vaccination history in the last 2 months (n=3), patients without follow-up echocardiographic data (n=7) and using oral anticoagulants (n=10) were excluded from the study. The final study population was comprised of 185 patients. The study included 185 patients with LVAT (n = 35) and without LVAT (n = 150). A comparison of the demographic characteristics of the patients found no differences in terms of DM, HT, chronic obstructive pulmonary disease (COPD), dyslipidemia, or smoking. Laboratory values such as hemoglobin ($p = 0.030$), HCT ($p < 0.001$), MPV ($p = 0.0002$), and total protein ($p = 0.039$) were numerically and statistically higher in the LVAT group. A higher incidence of apical aneurysms was observed in the group with LVAT ($p < 0.001$), in addition to a lower value of LVEF ($p < 0.001$). MAPH scores were higher in LVAD patients than in those without (2.34±0.59 and 1.47±0.60, $p < 0.001$) (Table 1). Patients with MAPH≥2 had more apical aneurysms than patients with MAPH scores <2 ($p = 0.001$) (Table 1).

The cut-off values determined were as follows: for MPV >8.5 fL, sensitivity 45%, and specificity 57%; for age >52 years, sensitivity 67%, and specificity 63%; for HCT >40%, sensitivity 62%, and specificity 60%; for serum total protein levels >60.9 g/L, sensitivity 59%, and specificity 65%; for MAPH score ≥ 2, sensitivity 74.1%, specificity 87.3% were determined as cut-off values (Figure 2). Left ventricular apical thrombus can be predicted better using the MAPH score than self-contained parameters (Table 2).

The MAPH score (Odds ratio [OR]= 1.265, 95% confidence interval [CI]= 1.124–1.423; $p < 0.001$), HCT (OR= 1.005, 95% CI= 0.813–1.030; $p = 0.040$), MPV (OR= 1.084, 95% CI= 0.622–1.206; $p = 0.034$), and LVEF (OR= 0.106, 95% CI= 0.040–0.280; $p < 0.001$)were found to be independent predictors of an LVAT (Table 3).

The MAPH score (Hazard ratio [HR]= 1.345, 95% CI= 0.984–1.790; $p = 0.004$), hematocrit (HR= 1.042, 95% CI= 0.764–1.469; $p = 0.037$), LVEF (HR= 0.685, 95% CI= 0.471–1.025; $p = 0.013$), apical aneurysm (HR= 1.179, 95% CI= 0.971–8.794; $p = 0.021$) and

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Table 1 – Baseline clinical, laboratory, and angiographic data of the study patients

	Left Ventricular Apical Thrombus			MAPH score		
	With (n = 35)	Without (n = 150)	p value	≥2(n = 98)	<2 (n = 87)	p value
Age (years)	64.0±11.3	60.4±13.3	0.111	62.5±12.7	59.4±13.1	0.109
Gender (M/F)	22/13	113/37	0.144	71/27	64/23	0.865
Smoking, n (%)	7 (20)	41 (27.3)	0.521	25 (25.5)	23 (26.4)	0.886
Diabetes mellitus, n (%)	6 (17.1)	21 (14)	0.603	16 (16.3)	11 (12.6)	0.479
Hypertension, n (%)	7 (20)	15 (8.1)	0.142	15 (15.3)	7 (8)	0.172
Dyslipidemia, n (%)	10 (28.6)	23 (15.3)	0.085	18 (18.4)	15 (17.2)	0.994
COPD, n (%)	4 (11.4)	9 (6)	0.273	8 (8.2)	5 (5.7)	0.576
Laboratory data						
Glucose, mg/dl	111.20±26.75	105.11±25.97	0.228	105.26±25.10	107.39±27.39	0.584
Creatinine, mg/dl	0.91±0.18	0.89±0.18	0.569	0.91±0.20	0.87±0.16	0.121
Hemoglobin (g/dl)	13.86±1.27	13.04±2.09	0.030	13.40±1.92	12.97±2.05	0.143
Hematocrit, %	42.24±4.43	36.58±6.02	<0.001	38.76±6.69	36.40±5.26	0.009
WBC count 109/ L	8.64±2.88	8.77±3.20	0.818	8.41±2.79	91.3±3.45	0.122
Platelet count, 109/ L	227.43±54.90	226.95±55.04	0.963	220.90±54.20	233.95±55.09	0.107
LDL-cholesterol, mg/dl	141 (93-146)	111 (93-134)	0.108	121 (92-143)	106 (93-134)	0.211
HDL-cholesterol, mg/dl	39.70±8.98	40.77±8.05	0.523	40.21±8.27	40.97±8.20	0.535
Triglyceride, mg/dl	158 (109-238)	155 (126-212)	0.631	154 (108-212)	155 (131-203)	0.723
Cardiac Tn, ng/L	300 (159-467)	197 (81-624)	0.716	321 (93-467)	187 (100-684)	0.707
LVEF, %	36.37±6.86	45.44±5.92	<0.001	42.17±7.53	45.47±6.05	0.001
Apical aneurysm, n (%)	22 (62.9)	12 (8)	<0.001	27 (27.6)	7 (8)	0.001
MPV, fL	8.37±0.64	7.97±0.76	0.002	8.14±0.68	7.94±0.82	0.077
Total protein, g/dL	6.71±0.82	6.39±0.70	0.039	6.39±0.77	6.52±0.68	0.244
Albumin, g/dL	3.86±0.42	3.85±0.43	0.943	3.87±0.42	3.83±0.43	0.697
MAPH score	2.34±0.59	1.47±0.60	<0.001			
Angiographic data						
Only IRA, n (%)	25 (71.4)	110 (73.3)	0.974	70 (71.4)	65 (74.7)	0.956
IRA + critical lesion Non-IRA, n (%)	8 (22.9)	32 (21.3)		24 (24.5)	16 (18.4)	
IRA + noncritical lesion Non-IRA, n (%)	2 (5.7)	8 (5.3)		4 (4.1)	6 (6.9)	

COPD: Chronic obstructive pulmonary disease; LWBC: White blood cell count; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; Tn: Troponin; LVEF: Left ventricle ejection fraction; MPV: Mean platelet volume; MAPH: MPV + age + total protein + haematocrit.

current smoking (HR= 1.002, 95% CI= 0.856–1.593; p = 0.042) were found to be independent predictors of MACCE (Table 3).

MACCE was more frequent in patients with LVAT than those without LVAT (p < 0.001). HF (p = 0.004), stroke (p = 0.005), and all-cause mortality (p = 0.007) were more frequent in patients with LVAT compared to patients without LVAT (Table 4). A high MAPH score was associated with a higher overall incidence of MACCE (Figure 3). The main data of the article is presented as a central figure.

Discussion

In the present study, patients diagnosed with the first episode of acute anterior STEMI having LVAT on transthoracic echocardiography had a higher MAPH score. Furthermore, MAPH performed better than the other four parameters (MPV, age, total protein, and HCT) for predicting LVAT. MAPH score is an independent predictor for LVAT and MACCE in

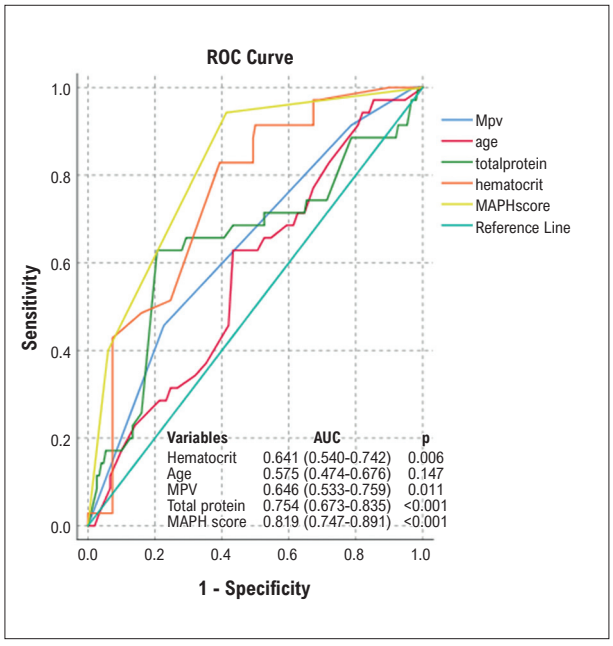

Figure 2 – *ROC curve analysis of variables.*

Table 2 – Pairwise comparison of ROC curve analysis

	Difference between AUC	CI 95%	Z statistics-p value
Age-hematocrit	0.179	0.041-0.317	2.549 - 0.011
Age-MPV	0.066	0.075-0.208	0.921 - 0.357
Age-total protein	0.071	0.085-0.228	0.892 - 0.372
Age-MAPH score	0.295	0.187-0.403	5.338 - <0.001
Hematocrit-MPV	0.113	0.012-0.238	1.769 - 0.077
Hematocrit-total protein	0.108	0.003-0.219	1.905 - 0.057
Hematocrit-MAPH score	0.116	0.027-0.204	2.570 - 0.010
MPV-MAPH score	0.228	0.132-0.325	4.645 - <0.001
MPV-total protein	0.005	0.145-0.323	0.068 - 0.946
MAPH score-total protein	0.224	0.107-0.341	3.746 - <0.001

MPV: Mean platelet volume; MAPH: MPV + age + total protein + haematocrit.

patients diagnosed with first-episode acute anterior STEMI.

LVAT formation after acute anterior STEMI and left ventricular remodeling warrants close monitoring of patients.[16] The prevalence of LVAT was 27% to 46% in the pre-PCI era; after PCI became available 24 hours a day, seven days a week, it has been reduced to 2% to 7%.[17,18] Echocardiography is a widely used diagnostic imaging technique for LVAT. However, the true incidence of LVAT may be low due to the sensitivity of echocardiography. Although, it is an imaging modality with a sensitivity and specificity of 90% in patients with good-quality echocardiography windows.[19-21] As an alternative, computed tomography has similar sensitivity and specificity as TTE, but the additional radiation makes it unfavorable.[22] In practice, MRI is not practical for all patients with pre-diagnosed LVAT. In our study, most patients were diagnosed with TTE, but CT or MR imaging modalities were used in cases of suspicion. The size of the infarction, the decrease in LVEF, and the delay between pain onset and revascularization are all risk factors for LVAT. In addition, left ventricular apical aneurysms are risk factors for LVAT.[23] In our study results, apical aneurysm and reduced LVEF are risk factors for LVAT, and our results are similar to the literature.

A component of the MAPH score is MPV, which provides information about platelet activity and function. High MPV values are associated with a higher risk of thrombosis and poor prognosis of thrombotic diseases.[24] Similarly, advanced age is associated with thrombotic events.[25] Three important serum elements regulate total protein plasma viscosity: albumin, fibrinogen, and globulin.[26] Non-albumin protein levels were higher in patients with spontaneous echo contrast, and TIMI frame count and beta 2 microglobulin levels were positively correlated.[27] A recent study found higher HCT values in patients with an increased intracoronary thrombus load.[28] All these data indicate that MPV, total protein, advanced age, and HCT values are associated with thrombosis. Our study found that these parameters were higher in patients with LVAT compared to the control group. Based on all these parameters, our study found that the MAPH score better predicted LVAT and MACCE in patients with anterior STEMI.

Anticoagulant therapies are used to treat LVAT and may reduce the risk of embolic events associated with LVAT. Anticoagulants are recommended for patients with LVAT.[29] An early diagnosis of LVAT is important to initiate anticoagulation therapy to prevent thromboembolic disease. The MAPH score may be the new score for predicting LVAT in patients with anterior STEMI. As a result, MAPH scores can be used to take early precautions for thromboembolic complications related to LVAT.

Limitations

Our study has several limitations. The LVAT was performed using TTE, but contrast agent imaging was impossible since it is not commonly used in our country. To perform LVAT using TTE without contrast agents, the healthcare provider may use a combination of different imaging techniques, such as M-mode, 2D, and Doppler ultrasound. These techniques can help visualize the LVAT without the use of contrast agents. It is important to note that using contrast agents can improve the accuracy of LVAT assessment, especially in cases where the imaging quality is poor or when there is limited acoustic access. However, it is not always necessary, and alternative techniques can be used to obtain accurate and reliable information. In our study, cardiac magnetic resonance imaging was not routinely performed on patients diagnosed

Table 3 – Predictors of left ventricular apical thrombus in patients with ST-elevation myocardial infarction

	Left ventricular apical thrombus					
	Univariate analysis			Multivariate analysis		
Variables	OR	95% CI	p	OR	95% CI	p
Age	0.978	0.950-1.008	0.127			
Current smoking	1.505	0.610-3.711	0.375			
Gender	0.554	0.254-1.208	0.138			
LVEF	0.087	0.040-1.456	<0.001	0.106	0.040-0.280	<0.001
Apical aneurysm	1.251	0.921-2.127	<0.001	1.165	1.045-.1613	0.007
Hypertension	0.444	0.166-1.190	0.107			
Diabetes mellitus	0.787	0.292-2.123	0.636			
Cardiac Tn	1.000	0.999-1.001	0.897			
MPV	1.036	0.742-1.785	0.006	1.084	0.622-1.206	0.034
Total protein	1.004	0.403-1.909	0.021	0.564	0.234-1357	0.342
Hematocrit	1.081	0.750-1.804	<0.001	1.005	0.813-1.030	0.040
MAPH score <2	1.178	1.029-1.408	<0.001	1.011	1.003-1.060	<0.001
MAPH score ≥2	1.323	1.201-1.456	<0.001	1.265	1.124-1.423	<0.001
	Adverse events					
	Univariate analysis			Multivariate analysis		
Variables	OR	95% CI	p	OR	95% CI	p
Age	0.988	0.965-1.012	0.329			
Current smoking	1.083	0.913-1.647	0.003	1.002	0.856-1.593	0.042
Gender	1.102	0.554-2.193	0.782			
LVEF	0.790	0.727-0.858	<0.001	0.685	0.471-1.025	0.013
Apical aneurysm	1.375	0.617-5.517	<0.001	1.179	0.971-8.794	0.021
Hypertension	0.578	0.202-1.648	0.305			
Diabetes mellitus	1.317	1.104-1.962	0.043	0.784	0.164-3.737	0.760
Cardiac Tn	1.000	0.999-1.001	0.742			
MPV	2.235	1.376-3.629	0.001	1.124	0.957-1.481	0.068
Total protein	1.029	0.676-1.567	0.894			
Hematocrit	1.286	0.528-2.647	0.003	1.042	0.764-1.469	0.037
MAPH score <2	1.317	1.050-2.186	<0.001	1.026	1.004-1.529	0.025
MAPH score ≥2	1.591	1.036-2.441	<0.001	1.345	0.984-1.790	0.004

LVEF: Left ventricle ejection fraction; Tn: Troponin; MPV: Mean platelet volume; MAPH: MPV + age + total protein + hematocrit; CI: Confidence interval; OR:Odds ratio; HR: Hazard ratio.

with LVAT. MAPH may perform better if the number of imaging modalities increases, resulting in differences in LVAT incidence.

Conclusion

LVAT can lead to serious cardiovascular consequences such as systemic embolization, stroke, or even death. Therefore, patients who experience a STEMI and are at high risk for LVAT should be closely monitored and considered for anticoagulant or thrombolytic therapy to prevent or treat thrombus formation. MAPH scores can be calculated easily in routine practice. The test can identify patients with anterior

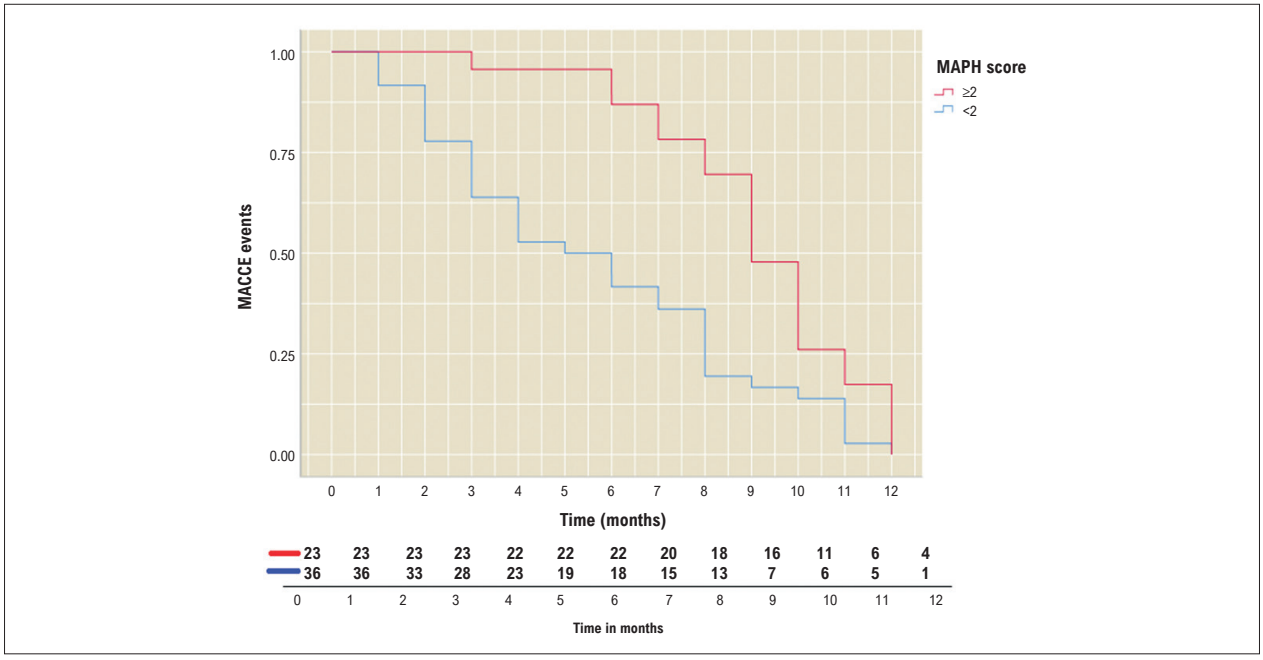

Figure 3 – *Cumulative incidence of MACCE in patients admitted for STEMI and treated by PCI during 12 months of follow-up.*

Table 4 – Comparison of adverse events

	Left ventricular apical thrombus		
	With (n = 35)	Without (n = 150)	p
MACCE	23 (65.7)	37 (24.7)	<0.001
Outcomes			
Heart failure, n (%)	16 (45.7)	30 (20)	0.004
Non-fatal myocardial reinfarction, n (%)	1 (2.9)	3 (2)	0.571
Revascularization, n (%)	0 (0)	4 (2.7)	0.329
Stroke, n (%)	4 (11.4)	1 (0.7)	0.005
All-cause death, n (%)	5 (14.3)	3 (2)	0.007

MACCE: Major adverse cardiovascular and cerebrovascular event.

STEMI at high risk for LVAT. It may also help reduce adverse outcomes by identifying patients who will benefit from future treatment, such as anticoagulant therapy.

Author Contributions

Conception and design of the research; Acquisition of data; Analysis and interpretation of the data; Statistical analysis; Writing of the manuscript and Critical revision of the manuscript for important intellectual content: Küçük U, Altınsoy M.

Potential conflict of interest

No potential conflict of interest relevant to this article was reported.

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Study association

This study is not associated with any thesis or dissertation work.

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Çanakkale Onsekiz Mart University under the protocol number 2023/03-05. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

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