BMC Cardiovascular Disorders

RESEARCH

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Usefulness of platelet mass index in the prediction of angiographic no-reflow in patients with acute ST-segment elevation myocardial infarction

Erdoğan Sökmen[1*] and Muhammet Salih Ateş[1]

Abstract

Background and objective No-reflow phenomenon (NR) is a serious complication with increased morbidity and mortality in percutaneous coronary interventions in patients with acute ST-segment elevation myocardial infarction (STEMI). Studies on the relationship between the NR and mean platelet volume (MPV) and platelet count (PLT) are controversial. Platelet mass index (PMI) is a novel inflammation and platelet index, calculated as PLT multiplied by MPV. So, it would be prudent to assume that a high PMI is likely to be associated with NR. PMI's low cost and rapid availability may aid NR risk stratification. Our aim was to assess the relationship between PMI and no-reflow in acute STEMI patients.

Methods A total of 212 acute STEMI patients were enrolled in this retrospective study and the patients were stratified into two subgroups as no-reflow group ($n=45$) and reflow group ($n=167$). Patient data regarding demographics, clinical, angiographic and laboratory parameters were retrieved from the digital hospital archives. No-reflow was defined angiographic thrombolysis in myocardial infarction (TIMI) flow grade ≤ 2. PMI was calculated as platelet count multiplied by mean platelet volume (MPV).

Results Mean age of the no-reflow and reflow groups was 59.3 ∓ 8.6 and 59.1 ∓ 12.6 years, respectively ($p>0.05$). PMI was greater in the no-reflow group [2585(2278–3000) vs. 2054(1594–2344), respectively, $p<0.001$]. PMI was correlated with WBC count ($r=0.290$, $p<0.001$), Hemoglobin (r=-0.281, $p<0.001$), neutrophil count ($p=0.303$, $p<0.001$), platelet count ($r=303$, $p<0.001$), MPV ($r=0.195$, $p=0.006$), platelet distribution width ($p=0.215$, $r=0.002$), and PCT ($r=0.970$, $p<0.001$), and Syntax score-2 ($r=0.162$, $p=0.024$). In mulvariate logistic regression analysis, PMI [OR: 1.008(1.003–1.012), $p=0.001$], age [OR: 1.111 (1.036–1.253), $p=0.007$], and WBC count [OR: 0.018(0.001–0.581), $p=0.024$] were independently associated with NR.

Conclusion PMI has been a simple and readily available parameter that could be a promising indicator to estimate NR in patients with acute STEMI.

Keywords Acute myocardial infarction, No-reflow phenomenon, Platelet mass index

*Correspondence:
Erdoğan Sökmen
erdoganmen@gmail.com
[1]Department of Cardiology, Kırsehir Ahi Evran Education and Research Hospital, Kırsehir, Turkey

Introduction

The most contemporary treatment modality in acute ST-segment elevation myocardial infarction (STEMI) is percutaneous intervention of the infarct-related coronary artery (PCI). However, no-reflow (NR) phenomenon still stands as an important obstacle in the very front of the success of a PCI.

NR has been a phenomenon implying a non- or underachievement of a normal coronary reperfusion in spite of recanalization of an occluded coronary artery and caused by coronary dysfunctional microcirculation due to endothelial swelling, spasm in the microvasculature, and distal embolization of the thrombus debrides [1–3]. Moreover, NR may complicate in varying ratios up to 60% of PCI procedures in patients with acute STEMI, thereby leading to a long line of risks including greater mortality due to inadequate ventricular healing and remodeling [2].

Previous studies showed that some existing parameters such as high blood glucose and fibrinogen levels, increased serum uric acid/albumin ratio, increased neutrophil/lymphocyte ratio (NLR), increased serum D-dimer level, increased C-Reactive Protein/Albumin ratio, elevated high-sensitive C-reactive protein and fibrinogen levels were associated with NR [4–8]. Current approaches to predict NR have important limitations. Inflammatory indices like the NLR show moderate predictive value (AUC 0.65–0.72) but lack standardized cutoffs [4, 5]. Thrombotic markers such as D-dimer exhibit good specificity but poor sensitivity [6]. Complex angiographic scores like SYNTAX, while valuable, cannot be calculated before PCI. These limitations highlight the need for a more reliable, readily available predictor that integrates both the thrombotic and inflammatory pathways central to NR pathophysiology. However, little is known about the relationship between the hematological parameters, especially the platelet functionality, and NR phenomenon in patients with acute STEMI.

Platelets play a crucial role in the thrombus formation during atherosclerotic plaque rupture. Previous studies reported that increased platelet counts or aggregates was related to NR during PCIs in acute STEMI cases [3, 8–10]. In a recent meta-analysis, there appeared an independent relationship between platelet count and NR (OR = 1.002, 95% CI: 1.000–1.005, $p = 0.038$) [8]. Mean platelet volume (MPV) is also an important platelet parameter, since voluminous platelets have been more active and thrombogenic [11]. Also, studies have revealed a significant association between MPV and NR [12–15].

Platelet mass index (PMI) has recently emerged as a new platelet parameter, calculated by multiplying the MPV with platelet count. As a novel index of both platelet activity and inflammation, PMI has been proposed to possess superior predictor ability compared to lone MPV or platelet count [16, 17]. However, studies on the relationship between PMI and coronary artery disease (CAD) is sparse. A study reported an increased PMI in psoriasis and it was concluded that PMI was likely to be a relevant factor of coronary plaque formation due to close interplay between CAD formation and psoriasis [18]. There is a delicate balance where lower platelet count and higher MPV maintain a normal cumulative platelet functionality so that the hemostatic capability of a thrombotic plaque may be dictated more efficiently by PMI compared with the sole platelet count or MPV [18–20].

Potential clinical implication of a higher PMI would be more aggressive thrombogenicity than normally expected in the setting of a plaque rupture leading to an acute STEMI.

In this study, our aim was to assess the relationship between PMI and no-reflow in acute STEMI patients and evaluate its potential clinical utility for early risk stratification. If validated, PMI could serve as a rapid, cost-effective tool to identify high-risk patients who may benefit from adjunctive therapies (e.g., glycoprotein IIb/IIIa inhibitors or thrombus aspiration) to guide personalized treatment decisions before or during PCI, or to complement existing risk scores without requiring additional testing.

Methods

Patient enrollment

Angiographic records of total of 350 patients presented to our hospital with an ASTMI and undergoing percutaneous intervention between March 2022 and May 2023 were assessed retrospectively. The inclusion criteria were the patients greater than 18 years of age and admission to our emergency department with an acute STMI. Among the exclusion criteria was: history of myocardial infarction, and ischemic and dilated cardiomyopathy; history of rheumatic diseases, hematological malignancies, or oncological diseases where increased innate thrombogenicity could be observed; thrombolytic agent administration before percutaneous interventions, presence of chronic inflammatory diseases where reactive increase in the platelet counts could be observed, severe hepatic [ALT/AST > 3× upper limit of normal, total bilirubin > 2.0 mg/dL (except Gilbert's syndrome), albumin < 2.8 g/dL with clinical signs of cirrhosis, or INR > 1.5 without anticoagulant use]

or kidney disease (eGFR < 30 mL/min/1.73 m² or ongoing dialysis) where platelet functions could be deteriorated. After excluding 138 patients on the basis of the exclusion criteria, we enrolled a final 212 patients into the study (Fig. 1). The reports as regards detailed medical history, physical examination, and blood and echocardiographic parameters were retrieved from the hospital's digital records.

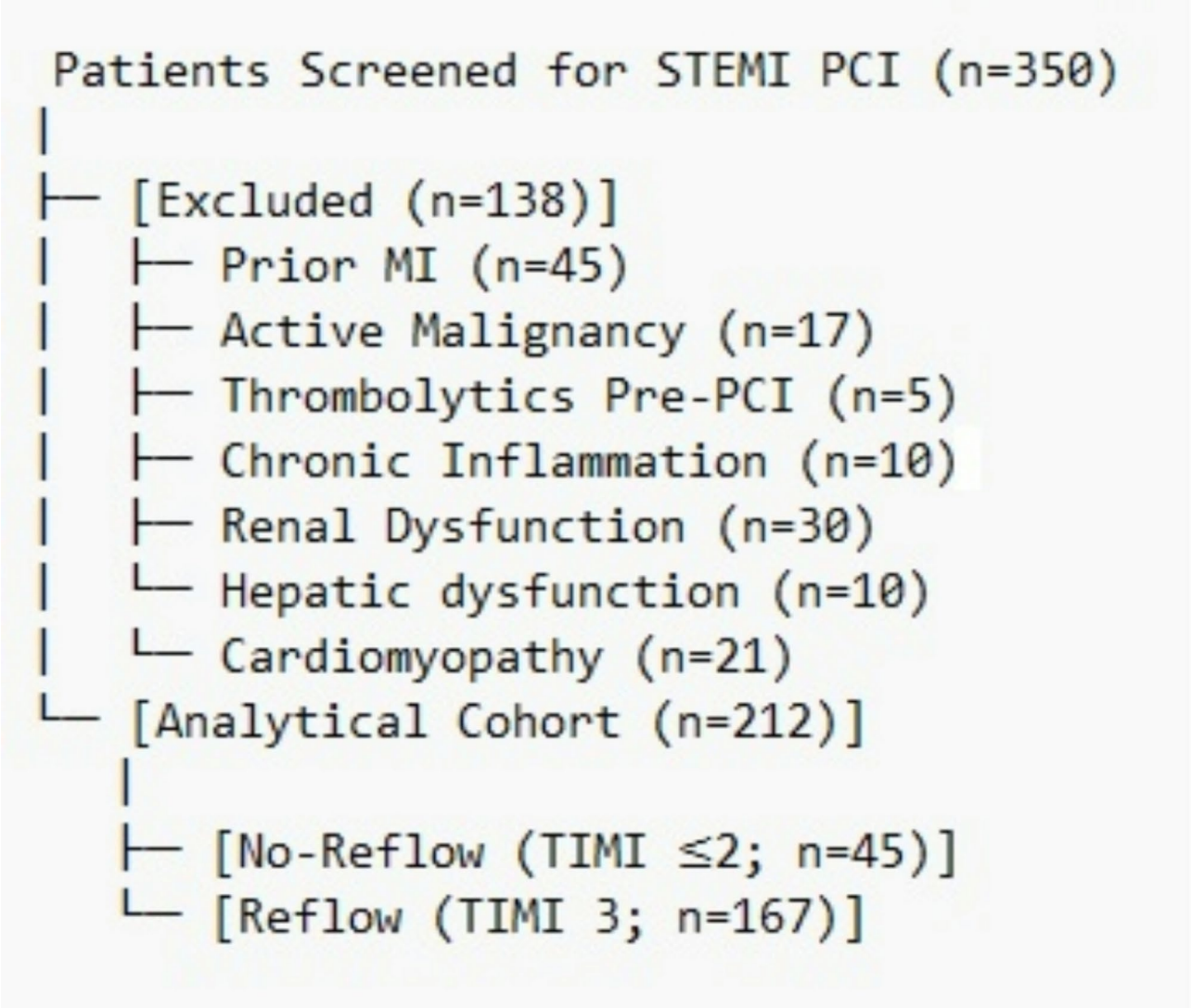

Fig. 1 Flowchart of the patient inclusion and exclusion

A Vivid 7 Pro echocardiography devise (GE, Horten, Norway) was used for the echocardiographic evaluations of the patients. Simpson's rule was utilized in calculating left ventricular ejection fraction. Patients on anti-hypertensive therapy were considered to have hypertension. Diabetes mellitus was defined by a fasting blood glucose > 126 mg/dL, HgA1c > 6.5 or the use of antidiabetic drugs. Hyperlipidemia was defined as: total cholesterol > 200 mg/dL, low density lipoprotein (LDL) > 130 mg/dL, or triglycerides > 150 mg/dL.

Acute STEMI was defined according to the relevant guideline [21]. On brief, change in the cardiac troponins together with at least one of the following criteria such as ischemia-related symptoms; new or presumably new ST segment elevation in ≥ 2 contiguous derivations or new left bundle branch block; pathological Q waves in the ECG; surrogates of viable myocardium loss or new regional wall motion anomaly during imaging; and depiction of coronary thrombus during angiography.

We further stratified the enrolled patients into two subgroups as no-reflow group ($n = 45$, mean age 59.3 ∓ 8.6 years) and normal flow group ($n = 167$, mean age 59.1 ∓ 12.6 years).

The principles of the Declaration of Helsinki were conformed during the performance of this study and local ethics committee of Ahi Evran University Medical Faculty approved our protocol.

Coronary angiographic procedures and definition of NR

The patients enrolled in the study were treated according to the recommendations by pertinent acute STEMI management guideline [22]. As soon as the written informed consent for cardiac catheterization was taken, an emergency coronary angiography was performed

using standard techniques in all the subjects. After the culprit artery was wired, administration of glycoprotein IIb/IIIa inhibitor (tirofiban) or implementation of thrombus aspiration in the cases in the catheterization laboratory were dictated by the choice of the operator. Direct stent implantation to the culprit lesion was intended where appropriate, whereas pre-dilatation using a coronary balloon was preferred in the other patients. Standard clinical practice was used during primary PCI of the culprit artery and all the patients were treated with drug-eluting stents. Thrombolysis in myocardial infarction (TIMI) flow grading of the culprit artery in each patient was assessed by a single cardiologist before and after PCI who was blinded to the study data to obviate the inter- and intra-observer variability. A grade 3 TIMI flow in the culprit artery with residual stenosis < 20% was considered normal flow [23]. Grade 0 TIMI flow was considered upon no further passage of the contrast agent beyond the point of intervention. Grade 1 TIMI flow was considered when contrast passage is observed beyond the point of intervention but not till the end of the vessel. Grade 2 TIMI flow was considered when contrast passage occurs until the very distal end of the vessel but in a sluggish manner. NR was termed as a TIMI flow grade ≤ 2 after stenting of the culprit artery without any evidence of coronary dissection, vasospasm or high visible thrombus burden [23]. In the current study, myocardial blush grading (MBG) was not used in the definition of angiographic NR due to the study's retrospective character, since, for an adequate assessment, MBG requires sufficiently long angiographic runs for both of the culprit and non-culprit arteries, left lateral angiographic view if left coronary artery is involved, and right oblique view if the right coronary artery is involved [24]. Only then does the MBG possess a significantly high degree of reproducibility and lowest inter- and intra-observer variability [24].

Computing the SYNTAX score-1 and SYNTAX score-2

Coronary angiography was conducted using standard methods. The catheterization images were reviewed to evaluate the complexity of CAD based on the SYNTAX Scores-1 and 2 (SS-1 and SS-2). The SYNTAX scores were calculated through the website "http://www.syntaxscore.com" by one independent cardiologist blinded to the study data to avoid interobserver variability. SS-1 and SS-2 were calculated in every case.

Biochemical and hematological parameters

An automated analyzer (Roche Hitachi Cobas c8000 autoanalyzer, Roche Diagnostic Corp., Mannheim, Germany) was used to measure serum biochemistry. Moreover, a Beckman Coulter LH 780 Analyzer (Miami, FL, USA) was utilized for complete blood count. MPI was computed as platelet count multiplied by MPV [18]. This combination reflects total platelet activity because: [1] MPV represents platelet size (larger platelets are more prothrombotic) [2], platelet count indicates available thrombotic mass, and [3] their product estimates functional platelet burden more comprehensively than either parameter alone [11, 16, 18].

Statistical analysis

The study parameters were analyzed using the Statistical Package for Social Sciences (SPSS) for Windows, version 29.0 (SPSS Inc., Chicago, IL, USA). The parameters possessing normal and nonnormal distributions were defined by use of The Kolmogorov-Smirnov or Shapiro-Wilk test. Normally-distributed parametric variables were expressed as mean ± standard deviation, while those that did not follow a normal distribution were presented as median (25th-75th percentiles). Categorical parameters were expressed as counts and percentages. The bivariate analysis of the study parameters was implemented using respective Chi Square test, Mann-Whitney-U test and independent t test where appropriate. For missing laboratory/clinical values (< 5% of cases), we performed median imputation (for non-normally distributed variables) or mean imputation (for normally distributed variables). The correlations between the variables were assessed by using Pearson or Spearman correlation analysis, as appropriate. A multinominal logistic regression analysis was performed to define the parameters independently associated with NR. A p value was accepted statistically significant when it is < 0.05.

Results

A total of 212 patients was included in our final cohort. Mean age of the NR and reflow groups was 59.3∓8.6 years and 59.1∓12.6 years, respectively ($p > 0.05$). Table 1 demonstrates the demographic, clinical and angiographic features of the study groups. Both groups were similar as regards age, gender, DM, HT, HL, chronic obstructive pulmonary disease, peripheral artery disease and smoking ($p > 0.05$), except for previous history of CAD which was greater in the NR group (17.7% vs. 10.1%, $p = 0.024$). NR patients have significantly higher SS-1 and SS-2 compared with the reflow group [SS-1: 18.2 ± 60.3 vs. 15.8 ± 7.2, respectively, p=0.033; SS-2: 35.38 ± 70.9 vs. 29.70 ± 9.44, respectively, p=0.012]. Admission Killip classification, admission blood pressures and heart rate were similar between the two groups. As for blood biochemistry, glomerular filtration rate was significantly lower in the NR group compared with the reflow group (75.6∓24.8 vs. 84.0∓20.6, $p = 0.039$). Otherwise, both groups were similar in terms of glucose, total cholesterol, high- and low-density lipoprotein, triglyceride and admission hs-Troponin I levels (p>0.05). In hematological parameters, white blood cell, neutrophil, lymphocyte

Table 1 Demographic, clinical and angiographic features of the study groups

Variable	No-reflow (*n* = 45)	Re-flow (*n* = 167)	*p*
Age, years	59.3∓8.6	59.1∓12.6	0.880
Gender, female, n (%)	10 (22.2)	40 (24)	0.342
DM, n (%)	14 (30)	53 (31.7)	0.956
HT, n (%)	18 (40)	72 (43.1)	0.120
CAD, n (%)	8 (17.7)	17 (10.1)	**0.024**
COPD, n (%)	3 (6.6)	12 (7.2)	0.456
PAD, n (%)	4 (8.8)	16 (9.5)	0.632
Smoking, n (%)	14 (30)	54 (32.3)	0.245
LVEF, %	47.2 ± 11.4	50.4 ± 12.5	0.287
Syntax score-1	18.2 ± 60.3	15.8 ± 7.2	**0.033**
Syntax score-2	35.38 ± 70.9	29.70 ± 9.44	**0.012**
Systolic BP, mmHg	131.5∓19.6	128.8∓17.4	0.168
Diastolic BP, mmHg	76.3∓12.5	74.4∓11.3	0.321
HR, beats/min	79.2∓13.2	74.6∓12.2	0.245
Killip > 1	6 (16.6)	26 (18.9)	0.678
Killip 1	30 (83.3)	112 (81.1)	
Glucose, mg/dL	117.1∓50.3	120.4∓53.7	0.728
GFR, mL/min/1,73 m^2	75.6∓24.8	84.0∓20.6	**0.039**
Total Cholesterol, mg/dL	158.8∓51.7	171.9∓47.7	0.206
HDL Cholesterol, mg/dL	39∓7.9	40.1∓11.3	0.527
LDL Cholesterol, mg/dL	88∓37.7	96.8∓40.7	0.259
Triglyceride, mg/dL	152.1∓92.1	175.7∓99.5	0.204
hs-Troponin I, ng/L	1969.5(158.6–5709)	1062(69.3-3721.5)	0.198
WBC, x10^9/L	9223∓4234	10,507∓3555	0.101
Hg, g/dL	13.7∓2.1	14.2∓1.7	0.195
Neutrophil, x10^9/L	7006∓2976	6091∓4421	0.247
Lymphocyte, x10^9/L	2162∓955	2480∓1166	0.095
Monocyte, x10^9/L	803∓278	748∓225	0.216
Platelet, x10^9/L	267.3∓72.8	230.2∓105.3	**0.015**
MPV, fL	10.5∓0.85	10.2∓0.07	0.083
PDW, %	12.4∓1.8	12.2∓2.0	0.382
PCT, %	0.260(0.230–0.297)	0.210(0.190–0.270)	**0.002**
PMI	2585(2278–3000)	2054(1594–2344)	**< 0.001**
Angiographic Features			
Pain to balloon time, hour	5.1∓3.6	5.4∓2.1	0.235
Infarct related artery			
LAD	20 (55.5)	76 (55)	0.824
LCX	4 (11.1)	17 (12.3)	
RCA	12 (33)	43 (31.1)	
Stent count, n	1.56∓0.81	1.47∓0.72	0.243
Stent length, mm	41.8∓20.4	39.1∓19.2	0.221
Stent diameter, mm	3.2∓0.5	3.2∓0.4	0.886

DM, diabetes mellitus; HT, hypertension; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; PAD, peripheric artery disease; BP, blood pressure; HR, heart rate; GFR, glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; WBC, white blood cell count; Hg, hemoglobin; MPV, mean platelet volume; PDW, platelet distribution width; PCT, platecrit; PMI, platelet mass index; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery

and monocyte counts were similar between the groups, while platelet count was significantly higher in the NR group when compared with the reflow group (267.3∓72.8 vs. 230.2∓105.3, respectively, p=0.015). When the platelet indices were interrogated, PMI and platecrit (PCT) were seen to be significantly greater in the NR group than the reflow groups [for PMI: 2585(2278–3000) vs. 2054(1594–2344), respectively, p<0.001; for PCT: 0.260(0.230–0.297) vs. 0.210(0.190–0.270), respectively, $p = 0.002$]. Comparison of the groups regarding angiographic features such as pain-to-balloon time, the distribution of infarct-related arteries, stent count, stent length and stent diameter were similar between the groups ($p > 0.05$).

Table 2 Correlation analysis of PMI with various study parameters

Variable	Rho	*p*
Age	0.18	0.218
WBC	0.290**	**< 0.001**
Hg	-0.281**	**< 0.001**
Neutrophil	0.303**	**< 0.001**
Lymphocyte	0.032	0.648
Platelet	0.303**	**< 0.001**
Monocyte	0.045	0.526
MPV	0.195*	**0.006**
PDW	0.215**	**0.002**
PCT	0.970**	**< 0.001**
hs-CRP	0.100	0.214
Troponin-I	0.098	0.214
Syntax-1 score	0.040	0.644
Syntax-2 score	0.162*	**0.022**

WBC, white blood cell count; Hg, hemoglobin level; MPV, mean platelet volume, PDW, platelet distribution width; PCT, platecrit; hs-CPR, high-sensitive C-reactive protein

Table 3 Logistic regression analysis in the prediction of no-reflow

Variable	Univariate		Multivariate	
	OR (95% CI)	***p***	**OR (95% CI)**	***p***
Age	1.123 (1.045–1.243)	**0.001**	1.111 (1.036–1.253)	**0.007**
Smoking	0.500(0.224–1.116)	0.091		
DM	0.956(0.194–4.710)	0.956		
WBC	0.034(0.001–0.764	**0.005**	0.018(0.001–0.581)	**0.024**
Hg	0.877(0.719–1.070)	0.195		
Platelet	0.993(0.987–0.999)	**0.016**	0.865(0.947–1.014)	0.124
MPV	1.42(0.94–2.15)	0.091		
PDW	1.080(0.899–1.296)	0.411		
PCT	1.094(1.078–1.131)	**0.010**	0.985(0.968–1.034)	0.102
PMI	0.998(0.998–0.999)	**< 0.001**	1.008(1.003–1.012)	**0.001**

DM, diabetes mellitus; WBC, white blood cell count; Hg, hemoglobin level; MPV, mean platelet volume, PDW, platelet distribution width; PCT, platecrit; PMI, platelet mass index

Table 2 demonstrates the correlation of PMI with some other study variables. PMI was found to be correlated with WBC count ($r = 0.290$, $p < 0.001$), Hg (r=-0.281, $p < 0.001$), neutrophil count ($p = 0.303$, $p < 0.001$), platelet count ($r = 303$, $p < 0.001$), MPV ($r = 0.195$, $p = 0.006$), platelet distribution width ($p = 0.215$, $r = 0.002$), PCT ($r = 0.970$, $p < 0.001$), and SS-2 ($r = 0.162$, $p = 0.024$).

Table 3 depicts the results of the logistic regression analysis regarding the parameters associated with NR. Logistic regression analysis revealed that PMI [OR: 1.008(1.003–1.012), $p = 0.001$], age [OR: 1.111 (1.036–1.253), $p = 0.007$], and WBC count [OR: 0.018(0.001–0.581), $p = 0.024$] were independently associated with NR. ROC-curve analysis did not reveal a significant cut-off value for PMI in the prediction of NR ($p > 0.05$).

Discussion

In the current study, PMI, age and WBC count were found to be independently associated with NF phenomenon. To our knowledge, PMI has been studied for the first time in our study in NR patients with acute STEMI. Early risk assessment is of paramount importance in acute STEMI patients in order for the physicians' to be able to gauge better the invasive and/or pharmacological therapy, and PMI may prove to be a simple and useful hematologic parameter in this regard.

The observed correlations between PMI and various hematological and clinical parameters may reflect underlying pathophysiological processes. The positive correlation between PMI and WBC count likely indicates an inflammatory milieu, as both elevated WBC and platelet activation are hallmarks of systemic inflammation. This connection aligns with the role of inflammatory cytokines, such as interleukin-6, in stimulating both megakaryopoiesis and leukopoiesis. The association between PMI and the SYNTAX II score suggests that higher platelet mass may be linked to more advanced coronary artery disease, reflecting the pro-thrombotic and pro-inflammatory state often present in severe atherosclerosis. Conversely, the negative correlation between PMI and hemoglobin levels may signify the impact of chronic inflammation or disease burden, as anemia is frequently associated with increased platelet production through mechanisms such as iron-restricted erythropoiesis and elevated thrombopoietin levels. These findings underscore the interconnected roles of inflammation, thrombosis, and hematologic alterations in cardiovascular pathology.

Inflammation and increased thrombogenicity assume critical roles in the pathogenesis of NR. Re-establishment of normal coronary flow following a certain ischemia period traumatizes coronary microcirculation, thereby leading to platelet and neutrophil infiltration and formation of aggregates which in turn are likely to block microcirculation [25]. Moreover, neutrophil activation promotes the secretion of inflammatory mediators, thus further damaging the endothelial cells which is likely to cause vasospasm in the microvasculature and exacerbates neutrophil and platelet migration to the reperfusion-damaged area in a vicious cycle [26]. Increase platelet count and MPV have been known to be associated with NR in patients with acute STEMI. Danesh Sani et al. [15] reported an increased MPV and WBC count in patients with acute STEMI and they also reported a significant association between these parameters and NR. Although studies on the effect of WBC count on NR yielded conflicting results, a quite recent meta-analysis showed a significant relationship between NR and increased WBC count [8]. Likewise, we also found a significant association between WBC count and NR in our study. In

another study, Wang et al. [5] showed an independent association between MPV and NR in chronic total coronary occlusion cases undergoing PCI. Results of the studies on the effect of platelet count on NR are controversial, and a meta-analysis on this issue showed a significant association between platelet count and NR with a combined odd ratio: 1.002 (95% CI: 1.000–1.005, $p = 0.038$) [8]. Therefore, it is prudent to assume that a high PMI is likely to be associated with NR. We found, in our study, an independent association between PMI and NR.

Previous studies showed that age was an important predictor of NR [6, 27]. In the study by Namazi et al. [6], ROC cut-off for age was 62.5 years with 93% sensitivity and 77% specificity (AUC:0.636) to predict NR. In our study, age was also found to be a associated with NR, and the mean age of the NR group was 59.1 ∓ 12.6 years, which is very close to this cut-off value.

PMI is a relatively novel platelet parameter on which sparse number of studies are present. Preliminary studies on PMI were conducted on the need of platelet transfusion in pediatric patients in neonatal intensive care unit [19, 20]. There seems to be a balance in the PMI, as the rapidity at which new platelets with larger megakaryocyte cytoplasm forms upon decrease in platelet number for various reasons such as inflammation, intravascular coagulation or platelet al.loimmunization is directly related to increase MPV [19, 28]. Sole platelet count may be a poor clinical predictor in NR due to conflicting results of the previous studies; therefore, it would be more logical to assume that PMI as addressed by platelet count X MPV might be more predictive compared with platelet count or MPV alone. Absolute range of PMI is yet to be defined in the clinical practice. However, greater PMI seems to affect the disease-prediction ability. In this regard, Demir et al. [29] showed that PMI could be a more useful predictor of patent ductus arteriosus closure in preterm neonates compared with the platelet count alone. Günday et al. [16] reported that PMI was a more valuable indicator than MPV alone in coronary bypass operations. Unal [18]. showed in his study encompassing psoriatic patients an increased PMI and MPV, and concluded that the increase in these parameters may put the psoriasis patients to a more vulnerable state as regards atherosclerotic plaque complications.

Limitations

Our study must be viewed with a number of limitations. Relatively small number of our cohort puts our study in need of validation by future prospective studies with greater number of patient enrollment. As a single-center study, our results may not be generalizable to other populations due to potential regional variations in patient characteristics and treatment protocols. The retrospective design limited our ability to account for all potential confounders, particularly the influence of anti-inflammatory therapies on both PMI values and no-reflow outcomes. Moreover, retrospective studies are inherently subject to various biases and limitations that may impact the validity of their findings. Selection bias is a common issue, as the study population may not accurately represent the broader patient group. Recall bias can also arise, particularly when relying on historical records or patient memory, leading to inaccurate data collection. Additionally, confounding factors may not be fully accounted for, as retrospective designs often lack control over variables. Furthermore, we only measured PMI at a single timepoint (admission), which may not fully reflect the dynamic changes in platelet activity during PCI. While we demonstrated PMI's independent predictive value, our study did not compare its performance against other established no-reflow predictors in external validation cohorts. Missing or incomplete data in medical records further limits the robustness of the analysis. Despite these challenges, careful methodological design and statistical adjustments can help mitigate some of these biases and improve the reliability of retrospective study results. In this regard we took special attention to the conduction of a careful methodology and not to have any missing data which would otherwise affect our statistical analysis. As we mentioned in the methods section, we did not assess the MBG due to the retrospective nature of our study, which could have affected our reflow group to a certain extent, since some of the patients with an apparent TIMI 3 flow grade may have had MBG 1 or 2 which would put them in NR category. As MBG requires additional and longer angiographic recordings from more specific angles, prospective studies would be more appropriate for its utilization. Lastly, we did not seek to associate PMI with in-hospital prognosis of the study patients.

Conclusion

PMI is independently associated with NR in patients admitted with acute STEMI. This simple and readily available parameter could be a promising indicator to estimate NR in these patients. However, future prospective studies with larger cohorts are needed to justify our results.

Acknowledgements

We would like to express our gratitude to Dr. Fikret Keles for his generous contribution in the statistical analyses in the article.

Author contributions

Conceptualization, E.S and M.S.A; methodology, E.S; software, M.S.A; validation, E.S and M.S.A; formal analysis, M.S.A; investigation, E.S; resources, M.S.A and E.S; data curation, E.S and M.S.A; writing—original draft preparation, E.S; writing—review and editing, E.S and M.S.A; All authors reviewed the manuscript. All authors have read and agreed to the published version of the manuscript.

Funding

None to declare.

Data availability
No datasets were generated or analysed during the current study.

Declarations

Human ethics and consent to participate
Written informed consent was waived due to the retrospective nature of the study and approved by the Ethic Committee of Ahi Evran University Medical Faculty.

Consent for publication
Not applicable.

Clinical trial number
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 8 October 2024 / Accepted: 30 April 2025
Published online: 19 May 2025

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