The Egyptian Journal of Hospital Medicine (April 2023) Vol. 91, Page 4606-4613

Neutrophil-to-Lymphocyte Ratio and Mean Platelet Volume in Predicting Coronary No-Reflow in Patients with ST Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention

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ABSTRACT

Background: Primary percutaneous coronary intervention (PPCI) is the recommended treatment for acute ST-elevation myocardial infarction (STEMI). However, 40% of patients receiving initial PCI may experience the no-reflow phenomenon (NRP), which can enlarge the myocardial infarction region and increase death rate.

Objectives: The aim of the current work was to ascertain the role of the mean platelet volume (MPV) and neutrophil-to-lymphocyte ratio in predicting coronary no-reflow in STEMI cases undergoing PPCI.

Patients and Methods: This prospective study included a total of 100 patients with ST segment elevation myocardial infarction (STEMI) treated with primary PCI at Nasser Institute Hospital and Ain Shams University Hospitals. The patients were divided into 2 groups according to TIMI flow grades after primary PCI. 50 patients with TIMI flow grade 3 formed (reflow group) and 50 patients with TIMI flow grades 0-2 formed (no-reflow group).

Results: Cases which manipulated with post stent dilatation in no-reflow group were significantly higher than in the reflow group (P = 0.028). Ejection fraction (EF) by M mode was significantly lower in the no-reflow group cases compared to those of reflow group (P = 0.040). Absolute lymphocytes were significantly higher in the no-reflow group cases compared to those of reflow group (P = 0.001). When compared to cases in the reflow group, the neutrophils to lymphocytes ratio was significantly higher in the no-reflow group cases (P = 0.011). When compared to the reflow group cases, MPV was significantly higher in the no-reflow group cases (P = 0.001). Hb level was significantly higher in the no-reflow group cases compared to those of reflow group (P = 0.005). Mean platelet volume and Neutrophil to lymphocyte ratio are considered a promising positive predictor of no-reflow phenomenon after primary PCI.

Conclusion: It could be concluded that mean platelet volume is thought to be a potentially favorable predictor of the no-reflow phenomenon. New independent variables for forecasting no-reflow phenomenon in patients having PPCI include post-stent dilatation and EF by M mode. In patients having PPCI, the neutrophils to lymphocytes ratio plays a part in forecasting the no-reflow occurrence.

Keywords: Mean platelet volume, Neutrophil-to-lymphocyte ratio, no-reflow phenomenon, PPCI, STEMI.

INTRODUCTION

Despite the complete removal of the artery-related infarction-related obstruction, no-reflow phenomenon (NRP) refers to the failure of myocardial perfusion. Mojno came up with the word "no-reflow" in 1967 [(1)].

Myocardial infarction (MI), higher mortality, impaired left ventricular ejection fraction, malignant rhythms, and occurrence of cardiac rupture are all associated with the formation of the NRP [(1)].

Macroscopic symptoms of NRP include extensive tissue hemorrhage and myocardial necrosis, while microscopic symptoms include endothelial injury, inflammation, and intracellular swelling [(2,3)].

Leukocyte-erythrocyte plugs, microcirculatory vasospasm, platelet agglutination, neutrophil infiltration, free radical damage, and severe myocardial capillary damage brought on by distal microthrombus embolization all contributed to the development of these symptoms [(2,4, 5)].

The etiology of myocardial reperfusion and inflammation, which are both important contributors to the development of coronary artery disease and its consequences, are closely related [(6)].

The growth of NRP and the creation of thrombi are actively influenced by systemic and local inflammation. The development and rupture of atherosclerotic plaque, as well as artery blockage, can all be caused by inflammation [(7)]. Techniques like angiography, electrocardiography (ECG), contrast ultrasound, scintigraphy, and magnetic resonance imaging (MRI) can be used to identify it [(5)].

A severe slowing or lack of distal coronary flow (TIMI 0-1-2) without any dissection, vasospasm, thrombosis, or remnant stenosis is what it is known angiographically as no-reflow phenomenon [(5)]. The frequency during primary PCI is approximately 11%-41%, despite the fact that it was 0.6-2.0% in all PCIs [(1)].

MBG and TIMI flow scores are commonly used to evaluate coronary perfusion. The term "TIMI flow" describes both the rate of dye flow and intensity of visualization of infarct related epicardial artery.

TIMI ranges from 0 to 3. The myocardial perfusion refill and clearing are assessed using MPG. MBG receives a score of 0-3. Upon filling, cardiac color is visible (or ground glass appearance of the myocardium) [(9)].

As a result, TIMI flow grading measure the epicardial flow, whereas MBG analyze the capillary flow. Angiographical no-reflow is defined by TIMI flow classification and MBG as TIMI flow <3 (with any MBG grade) or TIMI flow 3 with MBG 0-1 whereas effective reperfusion is attained when MBG is (2-3) and TIMI flow is 3 [(10)].

Received: 22/11/2022
Accepted: 24/01/2023

A measure of platelet size known as the mean platelet volume (MPV) has been shown to correspond with platelet reactivity. The (MPV) is typically used in the differential diagnosis of thrombocytopenia. Higher MPV denotes a rise in bone marrow platelet production. Large platelets are thought to be more chemically and enzymatically reactive and hemostatic, and this may be a risk factor for MI [(11)].

A granulocyte, or neutrophil, makes up 99% of polymorphonuclear cells. Their lifespan is typically less than a day, they are actively phagocytic, and neutrophil counts rise particularly in inflammatory conditions. The ratio of neutrophils to lymphocytes (N/L) indicates whether the body's neutrophil and lymphocyte populations are in equilibrium as well as whether there is widespread inflammation [(12)].

This study was aimed to examine the effects of mean platelet volume and neutrophil-to-lymphocyte ratio on coronary no-reflow in ST-elevation myocardial infarction patients undergoing primary percutaneous coronary intervention.

PATIENTS AND METHODS

This prospective study included a total of 100 patients with ST segment elevation myocardial infarction (STEMI) treated with primary PCI at Nasser Institute Hospital and Ain Shams University Hospitals. This study was conducted between March 2020 and October 2021.

The patients were divided into 2 groups according to TIMI flow grades after primary PCI. **Group I (reflow group):** 50 patients with ITMI flow grade 3, and **Group II (no-reflow group):** 50 patients with TIMI flow grade 0-2.

Inclusion criteria: Patients with acute STEMI who received PPCI and presented to the hospital within 24 hours of the start of normal chest pain, and the Surface ECG revealed new left bundle branch block or ST segment elevation of less than ≥1 mm in at least two adjacent leads or more than 2 mm in leads v1 through v3.

Exclusion criteria: Patients who arrived more than 24 hours after a severe event. Patients who had received thrombolytic treatment, those with hematological conditions like leukemia, lymphoma, and thrombocytosis, as well as those who have experienced mechanical problems like dissection or suboptimal stent inflation.

Sampling Method: it was calculated using G*Power software for Windows with a power of 0.8 and alpha error 0.05.

Study tools and Procedures: The patient's medical history, physical test results, and risk factors, with a focus on the duration from pain to injection. To identify patients with NRP and patients with normal coronary flow, the entire study group was examined.

Laboratory Analysis: Immediately after the ECG reading and STEMI diagnosis, venous blood samples were collected from all patients at the emergency department, and the blood routine, including neutrophil count, lymphocyte count, platelet count, MPV and hemoglobin concentration was measured.

Ethical approval:

The study was approved by the Ethics Board of Ain Shams University and an informed written consent was taken from each participant or their parents in the study. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistical analysis:

Software from a social science statistical program was used to conduct the study (SPSS, version 20, SPSS Inc., Chicago, Illinois, USA). Number and percentage were used to characterize qualitative facts. To confirm the distribution's normalcy, the Shapiro-Wilk test was utilized. The range (minimum and maximum), mean, standard deviation, median, and interquartile range were used to characterize quantitative statistics (IQR). The correct data analysis techniques were used. At the 5% level, significance of the findings was determined. P value < 0.05 was considered significant.

RESULTS

Table (1): Distribution of the studied cases according to TIMI (n= 100)

TIMI flow grade	No.	%
0	6	6.0
1	11	11.0
II	33	33.0
III	50	50.0

Table (2) shows the demographic data of the patients. As regard sex, there was no statistically significant difference between two groups. ($P = 0.118^{*}$). The average age for reflow group was (59.36 ± 10.80) ranged from 40 to 80 years and for no-reflow group was (56.94 ± 8.14) ranged from 41 to 75 years with no statistically significant difference between the two groups ($P = 0.209^{*}$).

Table (2): Comparison between the two studied groups according to demographic data

	Reflow group (n = 50)		No-reflow group (n = 50)		Test of Sig.	p
	No.	%	No.	%		
Sex						
Male	38	76.0	44	88.0	X^2=2.439	0.118
Female	12	24.0	6	12.0		
Age (years)						
Min. – Max.	40.0 – 80.0		41.0 – 75.0		t= 1.265	0.209
Mean ± SD.	59.36 ± 10.80		56.94 ± 8.14			

Table (3) shows the type of STEMI, among the two studied groups. There was no significant difference (P = 0.051).

Table (3): Comparison between the two studied groups according to the type of STEMI

STEMI	reflow group (n = 50)		No-reflow group (n = 50)		X^2	^{MC}p
	No.	%	No.	%		
ANT	32	64.0	26	52.0	5.78	0.051
INF	15	30.0	24	48.0		
LAT	3	6.0	0	0.0		

Table (4) shows the culprit artery, among the reflow group LAD was 62.0 % (31 patients), LCX was 10 % (5patients) and RCA was 28 % (14 patients). Among no-reflow group LAD was 48 % (24 patients), LCX was 16 % (8 patients) and RCA was 36 % (18 patients). There was no statistically significant difference between the two groups, (P = 0.353).

Table (4): Comparison between the two studied groups according to culprit artery

Culprit artery	reflow rate (n = 50)		No Flow rate (n = 50)		X^2	p
	No.	%	No.	%		
LAD	31	62.0	5.78	48.0	2.083	0.353
LCX	5	10.0	8	16.0		
RCA	14	28.0	18	36.0		

Table (5) shows the evaluation of the post-stent dilatation**:** In no-reflow group about 62% of the cases 31 patients were manipulated with post stent dilatation versus 40% of the cases 20 patients in the reflow group and **significant differences was found between the two categories (P = 0.028).**

Table (5): Evaluation of the post-stent dilatation between the two study groups

Post-stent dilatation	Reflow group (n = 50)		No-reflow group (n = 50)		X^2	p
	No.	%	No.	%		
No	30	60.0	19	38.0	4.842*	0.028*
Yes	20	40.0	31	62.0		

Table (6) shows the evaluation using the EF by M mode**:** EF by M mode in reflow group mean ± SD was (51.68 ± 8.40), and in no-reflow group mean ± SD was (48.34± 7.60), being lower in the no-reflow group and significant differences existed between the two study groups **(p = 0.040)**.

Table (6): Evaluation of the two groups using the EF by M mode.

EF by M mode (%)	Reflow group (n = 50)	No-reflow group (n = 50)	T	p
Min. – Max.	35.0 – 71.0	33.0 – 61.0	2.032*	0.040*
Mean ± SD.	51.68 ± 8.40	48.34± 7.60		

Table (7) shows the comparison between the two studied groups regarding neutrophils, lymphocytes, and neutrophils to lymphocytes ratio. There were no statistically significant differences between the two groups regarding relative and absolute neutrophils (P > 0.05). As regard relative neutrophils in reflow group, mean ± SD (70.94 ± 13.51) and in no-reflow group mean ± SD (75.95 ± 12.80). As regard absolute neutrophils in reflow group mean ± SD (8.05 ± 3.28) and in no-reflow group mean ± SD (8.76 ± 3.97). Regarding relative lymphocytes, there was no statistically significant difference between the two groups (P = 0.497), being (18.68 ± 10.67) in reflow group and (17.41 ± 10.25) in no-reflow group.
Regarding absolute lymphocytes there was higher statistically significant difference in the no-reflow group Mean ± SD (2.29 ± 0.95) versus reflow group Mean ± SD. (1.71 ± 0.92) (p = 0.001*) and regarding neutrophils to lymphocytes ratio, **there was** higher statistically significant difference in no-reflow group mean ± SD. (7.41 ± 5.97) versus reflow group Mean ± SD. (4.79 ± 4.34) (p = 0.011*).

Table (7): Comparison between the two studied groups according to neutrophils, lymphocytes, and neutrophils to lymphocytes ratio

		Reflow group (n = 50)	No-reflow group (n = 50)	Test of sig.	p
Neutrophils	**Relative (%)**				
	Mean ± SD.	70.94 ± 13.51	75.95 ± 12.80	t= 1.905	0.060
	Absolute (10^3/µL)				
	Mean ± SD.	8.05 ± 1.84	8.76 ± 2.11	t= 0.968	0.335
Lymphocytes	**Relative (%)**				
	Mean ± SD.	18.68 ± 4.41	17.41 ± 4.32	U= 1151.50	0.497
	Absolute (10^3/µL)				
	Mean ± SD.	2.29 ± 0.54	1.71 ± 0.41	U= 773.50*	0.001*
Neutrophils / lymphocytes ratio					
Mean ± SD.		4.79 ± 1.13	7.41 ± 1.72	U= 879.50*	0.011*

Table (8) shows the comparison between the two studied groups regarding CBC data (platelets counts, MPV and Hb). Regarding the platelets counts, in reflow group mean ± SD. (283.92 ± 54.36), and in no-reflow group mean ± SD. (258.38 ± 64.12). There was no statistically significant difference between two groups (P = 0.055). Regarding the MPV, there was higher statistically significant difference in the no-reflow group versus the reflow group as in (no-reflow group) mean ± SD. (9.20 ± 0.85) and (reflow group) mean ± SD.(8.63 ± 0.79) (p=0.001) and regarding Hb level, there was statistically significant difference between two groups in no-reflow group mean ± SD. (13.72 ± 1.76) versus reflow group mean ± SD. (12.73 ± 1.68), **(P =0.005).**

Table (8): Comparison of the two study groups based on CBC data (platelets counts, MPV and Hb)

	Reflow group (n = 50)	No-reflow group (n = 50)	T	p
Platelets (10^3/µL)				
Mean ± SD.	283.92 ± 54.36	258.38 ± 64.12	1.940	0.055
MPV (fL)				
Mean ± SD.	8.63 ± 0.79	9.20 ± 0.85	3.446*	0.001*
Hb (g/dl)				
Mean ± SD.	12.73 ± 1.68	13.72 ± 1.76	2.867*	0.005*

Table (9) shows the univariate and multivariate Logistic regression analysis for the parameters affecting no-reflow group. It was proved that **post-stent dilatation, EF by M mode, absolute lymphocytes, neutrophil/lymphocyte ratio (NLR), MPV and Hb** were significantly associated with no reflow. After multivariate analysis for the significant variables it was found that only **post stent dilatation, EF by M mode and MPV** have a significant independent effect on no reflow.

Table (9): Univariate and multivariate Logistic regression analysis for the parameters affecting no-reflow group

	Univariate		#Multivariate	
	p	**OR (95%C.I)**	**P**	**OR (95%C.I)**
Sex (male)	**0.125**	2.316(0.793 – 6.764)		
Age (years)	**0.208**	0.973(0.934 – 1.015)		
STEMI				
INF	**0.109**	1.969(0.861 – 4.503)		
LAT	**0.999**	–		
Culprit artery				
LAD	**0.296**	0.643(0.281 – 1.472)		
LCX	**0.376**	1.714(0.520 – 5.657)		
RCA	**0.392**	1.446(0.621 – 3.368)		
Number of stents	**0.185**	1.584(0.803 – 3.127)		
Number of balloons	**0.107**	1.767(0.885 – 3.530)		
Post-stent dilatation	**0.029***	2.447(1.095 – 5.468)	0.008*	3.846(1.421–10.409)
EF by M mode (%	**0.043***	0.949(0.901 – 0.998)	0.011*	0.914(0.852–0.979)
Neutrophils Relative	**0.063**	1.030(0.998 – 1.062)		
Neutrophils Absolute	**0.333**	1.056(0.946 – 1.179)		
lymphocytes Relative	**0.541**	0.988(0.951 – 1.026)		
lymphocytes Absolute	**0.004***	0.504(0.315 – 0.806)	0.074	0.507(0.240–1.069)
Neutrophils /lymphocytes ratio	**0.020***	1.112(1.017 – 1.215)	0.704	1.025(0.904–1.161)
Platelets	**0.060**	0.994(0.988 – 1.0)		
MPV	**0.002***	2.359(1.372 – 4.055)	0.001*	2.989(1.534–5.825)
Hb	**0.007***	1.400(1.096 – 1.790)	0.186	1.216(0.910–1.623)

DISCUSSION

The purpose of this research was to investigate the role of neutrophil-to-lymphocyte ratio and mean platelet volume in determining coronary no-reflow in patients with ST elevation myocardial infarction undergoing primary percutaneous coronary intervention.

The patients in the current study were divided into 2 groups according to TIMI flow grades after primary PCI. **Group I (reflow group):** 50 patients with ITMI flow grade 3, and **Group II (no-reflow group):** 50 patients with TIMI flow grade 0-2.

The present research analyzed angiographic and echocardiographic data and found no significant variations between the two groups as regard the type of STEMI and the causative artery.

There was a significant difference between the two groups, as determined by post-stent dilatation. No-reflow group had instances that were post-stent dilated more than the reflow group did.

In the current study, there was a significant variation in EF by M mode between the two groups that were investigated. The EF was decreased by M mode in the no-reflow group.

Our findings agreed with the analysis of the angiographic in acute myocardial infarction patients receiving percutaneous coronary intervention by **Sadek *et al.*** [13]. According to the results, there was a considerably greater rate of ejection fraction in the reflow group than in the no-reflow group. The findings of **Refaat *et al.*** [14] were consistent with those of our investigation, namely that the normal flow group's left ventricular ejection fraction (LVEF) was considerably greater than the no-reflow group's. They also investigated the origins of the no-reflow phenomena and its detrimental long-term consequences.

Despite the fact that the pathophysiology of the no-reflow phenomenon is not fully understood, prior research has shown that inflammation and excessive thrombotic activity play significant roles, which may ultimately lead to distal microvascular obstruction and endothelial dysfunction in the process of no-reflow phenomenon. Before PPCI, the patient typically has access to their whole blood count without charge, which contains a variety of inflammatory indicators. Previous research reveals that greater neutrophil numbers, lower lymphocyte counts, MPV, and NLR are associated to the genesis of the no-reflow phenomena and its prognostic significance in STEMI [15].

The current study's analysis of the hematological data in the two groups revealed that both groups' neutrophil counts were comparable. Neutrophils, both relative and absolute, did not significantly vary between the two groups ($P > 0.05$).

Our findings corroborated those of **Hanan *et al.*** [16], who examined the clinical and surgical signs of the no-reflow phenomena in 145 STEMI patients following primary PCI and found no statistically significant difference between the two groups in terms of total WBCs or neutrophils.

Wang *et al.* [17] investigated the pathogenesis of angiographic no-reflow following PPCI in patients with ST-segment elevation myocardial infarction and discovered that the no-reflow group had significantly

higher WBC and neutrophil counts than the flow group. Our results were in contrast to their findings.

Acute myocardial infarction causes a significant number of neutrophils to congregate in the ischemic heart, where they release oxygen free radicals and proteolytic enzymes that can harm endothelial cells and activate both internal and exterior coagulation pathways to facilitate the synthesis of fibrin. No-reflow regions' microvasculature showed significant leucocyte plugging [(18)].

According to our findings, the no-reflow group had statistically greater overall lymphocytes than the reflow group did.

Our findings were in contrast to those of **Badran *et al.*** [(19)], who investigated no-reflow in acute STEMI patients treated with PPCI and found a statistically significant difference between the two groups based on the no-reflow group's lower lymphocyte count compared to the reflow group's higher count.

According to research, people with coronary artery disease who have reduced blood lymphocyte counts will experience worse cardiovascular outcomes. Poor clinical results in acute coronary syndrome (ACS) patients have been linked to lower lymphocyte counts brought on by elevated cortisol in reaction to physiological stress [(20)].

According to our study, the neutrophil to lymphocyte ratio was significantly higher in the no-reflow group than it was in the reflow group.

Turkmen *et al.* [(21)] investigated the correlation between the neutrophil/lymphocyte ratio and the TIMI flow grade in STEMI patients having primary PCI. When compared to patients with normal TIMI flow, they found that the N/L ratio was considerably greater in the No-reflow group. This outcome is consistent with what we found.

The findings found were in line with those reported by **Wagdy *et al.*** [(22)], who examined the predictive value of the N/L ratio in STEMI patients who did not receive reflow and showed that patients in the no-reflow group had a statistically higher N/L ratio than patients in the reflow group.

This greater N/L ratio in the no-reflow group supports the idea that elastases produced by neutrophils after PPCI, which induce microvascular injury, are the root of no-reflow. Early after cardiac reperfusion, leukocytes may become stuck in coronary capillaries and venules, and plugging of increased leukocytes in the microcirculation may result in the no-reflow syndrome [(10)].

According to our research, there was no statistically significant difference between the reflow group and the no-reflow group in terms of platelet count.

Contrary to our results, **Badran *et al.*** [(19)] looked into the connection between post-intervention TIMI flow in STEMI patients who had done PPCI and the platelet/lymphocyte ratio (PLR) in those patients. The results showed that TIMI III STEMI reflow patients' platelet counts were significantly lower than those of no-flow patients. Likewise, **Panc *et al.*** [(23)] reported that the platelet count in the no-reflow group was greater than that in the reflow group

It was unclear exactly how elevated PLR in subjects with ACS led to unfavorable results. Megakaryocytic proliferation and relative thrombocytosis have been shown to raise inflammatory activity and exacerbate pro-thrombotic state, both of which constitute a high risk for ACS patients. Therefore, it has been demonstrated that elevated platelet numbers indicate platelet activation and help to cause no-reflow by causing microvascular blockage, thrombus formation, and vasoconstriction [(20)].

Our present research showed a statistically significant difference in MPV between the two groups, with the no-reflow group showing a greater degree of difference than the reflow group.

Our results supported those of **Zhang *et al.*** [(24)], who evaluated MPV's predictive value for the incidence of no-reflow in STEMI patients and discovered that MPV was higher in the no-reflow group compared to the reflow group.

Higher MPV seems to have prognostic relevance in STEMI and to be related to the etiology of the no-reflow syndrome. Additionally, it might indicate chronic inflammation, platelet reactivity, and a greater propensity to form thrombi [(15)].

No-reflow group had a higher Hb level than those in the reflow group, according to our findings, which revealed that there were statistically significant differences in the Hb levels between the examined groups.

Opposite to our study, **Wang *et al.*** [(17)] reported that patients with no-reflow had significantly lower hemoglobin levels than patients with normal-reflow.

Numerous factors, including platelet aggregation and an inflammatory response, have been proposed as the primary pathological causes of the no-reflow anomaly in previous research[(15)]. Neutrophils and lymphocytes play a major role in the progression of cardiac ischemia. Other earlier studies have also demonstrated a connection between contrast nephropathy, no-reflow, and prognosis in STEMI patients and the neutrophil-to-lymphocyte ratio (NLR), which represents both neutrophil and lymphocyte alterations as a novel predictor of inflammation [(22,25)].

In our study, we demonstrated that total lymphocytes, post-stent dilatation, EF by M mode, NLR, MPV, and Hb had a significant impact on the chance of no reflow. However, only MPV, EF in M mode, and post-stent dilatation are accurate independent indicators of no-reflow in individuals with PPCI.

Our results support the assessment by **Zhang *et al.*** [(26)] who found that the NLR, MPV, and PDW have predictive value for the no-reflow syndrome in patients with ST-segment elevation myocardial infarction. They discovered that NLR, MPV, and PDW were independent

markers of the no-reflow after adjusting for eGFR, cTnI, and PDW.

Machado *et al.* [27] concurred with our findings in the same study. In a univariate analysis, the research discovered that NLR and MPV greatly increased the chance of no-reflow. NLR and MPV stayed separate predictors of no-reflow in multivariate analysis.

Additionally, a significant correlation between the no-reflow phenomenon and haemoglobin was discovered by **Wang *et al.* [28]** who also conducted univariate and multivariate logistic regression analyses of the relationship between the no-reflow phenomenon and a number of parameters. This result demonstrated that low haemoglobin concentration was an independent predictor of the no-reflow phenomenon. Similar findings were made by **Badran *et al.* [19],** who found that lower EF are significant, independent predictors of no-reflow in STEMI patients having PCI.

While lymphocytes represent the regulating or defensive part of the immune system's physiological stress reaction, neutrophils represent non-specific systemic inflammation that activates the first line of defense. This combination of two significant and opposing immune pathways may account for NLR's predictive value. This may help to explain why NLR performs better in our research than just the total neutrophil count. Additionally, NLR is more stable than neutrophil count, which can change due to a variety of metabolic, pathological, and somatic variables **[24].**

It has been demonstrated that circulating MPV increased quickly after MI due to spleen thrombocyte release. Spleen releases larger, freshly formed PLTs to increase inflammation. It has been established that PLTs play a significant part in inflammation. The interaction between large leukocytes and platelets mediates the inflammatory characteristics of these cells. The probability of stent thrombosis, no-reflow via microthrombi, or microvascular damage during PCI is connected with the increased MPV as a measure of both inflammation and pro-coagulant activity **[29]**.

CONCLUSION

It could be concluded that after initial PCI, mean platelet volume is thought to be a potential favorable predictor of the no-reflow phenomenon. New independent variables for forecasting no-reflow phenomenon in patients having PPCI include post-stent dilatation and EF by M mode. In patients having PPCI, the neutrophils to lymphocytes ratio plays a part in forecasting the no-reflow occurrence.

Study limitation

- Only STEMI patients from two institutions were included in the research.
- Only 100 individuals were enrolled in our research.
- Short-term follow-up.
- Absence of myocardial blush evaluation for microvascular flow.
- Only M mode EF assessment; no 2Devaluation.

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