

Original Research Article

NEUTROPHIL-TO-LYMPHOCYTE RATIO AND PLATELET INDICES AS INDEPENDENT PREDICTORS OF THE NO-REFLOW PHENOMENON IN ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION UNDERGOING PRIMARY PERCUTANEOUS CORONARY INTERVENTION: A CROSS-SECTIONAL STUDY FROM NORTH-WESTERN INDIA

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ABSTRACT

Background: Objective: To determine whether the neutrophil-to-lymphocyte ratio (NLR), mean platelet volume (MPV) and platelet distribution width (PDW) independently predict angiographic no-reflow in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI), and to compare their discriminative performance alone and in combination.

Materials and Methods: In this single-centre, cross-sectional observational study at a tertiary-care cardiology department in Jaipur, India, 240 consecutive adults with STEMI undergoing primary PCI were enrolled. Venous blood was drawn before any antiplatelet loading dose. No-reflow was defined as post-stenting Thrombolysis in Myocardial Infarction (TIMI) flow grade <3 without mechanical obstruction. Independent predictors were identified by multivariable logistic regression, and discrimination was assessed by receiver-operating-characteristic (ROC) analysis.

Results: No-reflow occurred in 70 patients (29.2%). Admission NLR (6.8 ± 2.9 vs 3.2 ± 1.4), MPV (10.9 ± 1.2 vs 9.1 ± 1.0 fL) and PDW (16.8 ± 2.4 vs 13.9 ± 2.0 %) were higher in the no-reflow group (all $p < 0.001$). NLR (adjusted odds ratio [aOR] 1.48 per unit; 95% CI 1.24–1.77), MPV (aOR 1.42 per fL; 1.11–1.82), PDW (aOR 1.28 per %; 1.05–1.56), diabetes mellitus, pain-to-balloon time >6 h and high thrombus grade were independent predictors. NLR had the highest area under the curve (AUC 0.82; cut-off >4.9, sensitivity 78.6%, specificity 74.7%); a combined haematological model improved the AUC to 0.87. No-reflow rose from 8.8% to 52.5% across ascending NLR tertiles (p for trend < 0.001).

Conclusions: Admission NLR, MPV and PDW—available from a routine complete blood count—independently predict no-reflow after primary PCI for STEMI, and their combination improves risk discrimination.

Keywords: Mean platelet volume; Neutrophil-to-lymphocyte ratio; No-reflow phenomenon; Primary percutaneous coronary intervention; ST-segment elevation myocardial infarction.

INTRODUCTION

Cardiovascular disease remains the leading cause of death worldwide, and a growing share of the resulting premature mortality is concentrated in low- and middle-income countries, including India.^[1,2] ST-segment elevation myocardial infarction (STEMI) carries the highest early risk among its clinical presentations, and primary percutaneous coronary intervention (PCI) is the guideline-recommended reperfusion strategy for eligible patients.^[3] Restoration of epicardial patency, however, does not always restore tissue-level perfusion.

Angiographic no-reflow—reduced antegrade coronary flow (Thrombolysis in Myocardial Infarction [TIMI] flow grade <3) despite successful mechanical recanalisation and in the absence of residual obstruction—is a common complication of primary PCI and is associated with larger infarcts, heart failure and short- and long-term mortality.^[4,5] Its pathogenesis is multifactorial, combining distal atherothrombotic embolisation, ischaemia–reperfusion injury, microvascular spasm and an individual predisposition shaped by the inflammatory and metabolic milieu; diabetes mellitus is a recognised amplifier.^[4,6] Because preventive measures are most useful when applied before the procedure, simple tools that identify high-risk patients at presentation are clinically attractive.

Neutrophils and platelets are central to this process. Activated neutrophils plug capillaries and release proteolytic enzymes and reactive oxygen species, whereas relative lymphopenia reflects the physiological stress response; the neutrophil-to-lymphocyte ratio (NLR) integrates both into a stable, inexpensive index of systemic inflammation.^[7] Elevated admission NLR has been associated with impaired myocardial perfusion and long-term mortality after primary PCI.^[8] Larger platelets are metabolically and enzymatically more active; mean platelet volume (MPV) and platelet distribution width (PDW) therefore serve as surrogates of platelet reactivity, and a high admission MPV independently predicted no-reflow and mortality in STEMI.^[9] The platelet-to-lymphocyte ratio (PLR), which links thrombotic and inflammatory pathways, has also been associated with impaired reflow.^[10]

Studies that have compared these markers head-to-head have produced inconsistent results. In one cohort, NLR, MPV and PDW each predicted no-reflow with only modest discrimination (areas under the curve 0.59–0.65), and combining them offered no significant gain,^[11] while another found no single admission haematological parameter clearly superior.^[12] Data from Indian primary-PCI populations, who differ in age at presentation, risk-factor profile and ischaemic times, are limited. We therefore evaluated whether NLR, MPV and PDW independently predict no-reflow in patients with STEMI undergoing primary PCI, and compared the

discriminative performance of each marker and of their combination.

MATERIALS AND METHODS

Study design and population

This cross-sectional observational study was conducted in the Department of Cardiology, SMS Medical College and Attached Group of Hospitals, Jaipur, Rajasthan, over six months (month year to month year) and is reported according to the STROBE statement.¹³ Consecutive patients aged ≥ 18 years with STEMI who underwent primary PCI were enrolled. Patients were excluded if they had received antiplatelet or fibrinolytic agents before blood sampling, or had known chronic kidney or liver disease, active infection, autoimmune or haematological disorders, or were taking non-steroidal anti-inflammatory drugs, oestrogen-containing oral contraceptives or selective serotonin reuptake inhibitors.

The Institutional Ethics Committee approved the study (approval number and date), and written informed consent was obtained from every participant. The study conformed to the Declaration of Helsinki.

Sample size

Assuming a no-reflow incidence of approximately 30% and six candidate predictors, a sample of 240 patients was required at 95% confidence and 80% power, providing more than 10 outcome events per predictor variable in the final model.

Clinical and laboratory assessment

Demographic data, cardiovascular risk factors, Killip class, haemodynamic parameters, pain-to-balloon and door-to-balloon times were recorded. Five millilitres of antecubital venous blood was drawn as early as possible and before any antiplatelet loading dose into EDTA and plain tubes. Complete blood counts, including differential leucocyte counts, MPV and PDW, were measured on an automated haematology analyser (Sysmex XN-1000, Sysmex Corporation, Kobe, Japan); samples were stored at 4 °C until processing. NLR and PLR were calculated as the ratios of absolute neutrophil and platelet counts, respectively, to the absolute lymphocyte count. Admission glucose, serum creatinine, lipid profile and high-sensitivity C-reactive protein were also measured. Left-ventricular ejection fraction (LVEF) was estimated by two-dimensional echocardiography.

Primary PCI and angiographic analysis

Primary PCI was performed according to standard institutional protocol. Pre-procedural TIMI flow, culprit vessel, extent of disease and stent length were recorded. Thrombus burden was graded 0–5 by the TIMI thrombus grade, with grades 4–5 considered high.¹⁴ Post-stenting TIMI flow grade, corrected TIMI frame count (cTFC) and myocardial blush grade were assessed as described previously.⁵ No-reflow was defined as a post-procedural TIMI flow

grade <3 in the absence of dissection, spasm, residual stenosis or visible thrombus; patients with TIMI 3 flow constituted the normal-reflow group.

Statistical Analysis

Categorical variables are presented as number (percentage) and compared with the chi-square or Fisher exact test. Continuous variables are presented as mean $\pm$ standard deviation or median (interquartile range) according to the Shapiro–Wilk test, and compared with the independent-samples t test or Mann–Whitney U test. Variables with $p < 0.05$ on univariable analysis were entered into a multivariable logistic regression model to identify independent predictors of no-reflow; model fit was assessed with the Hosmer–Lemeshow test and Nagelkerke R². ROC analysis was used to derive the area under the curve (AUC), optimal cut-off (Youden index), sensitivity, specificity and predictive values of each marker; a combined model was built from the predicted probabilities of a logistic model containing NLR, MPV, PDW and PLR. Correlations with cTFC and myocardial blush grade were assessed using Pearson/Spearman — confirm coefficients. Analyses were performed with SPSS version 25.0 (IBM Corp., Armonk, NY, USA), and a two-tailed $p < 0.05$ was considered significant.

RESULTS

Baseline characteristics

Of 240 patients (mean age 57.6 ± 11.2 years; 74.2% men), no-reflow developed in 70 (29.2%) and normal reflow (TIMI 3) was achieved in 170 (70.8%). Hypertension (50.8%), smoking (45.8%) and diabetes (40.0%) were the commonest risk factors, and the left anterior descending artery was the culprit vessel in half the cohort (Table 1). Patients with no-reflow were older and more often diabetic, presented

later (pain-to-balloon time 6.8 ± 2.9 vs 4.9 ± 2.2 h) and more frequently in Killip class >I, and had lower LVEF (all $p \leq 0.002$). They also had higher admission glucose and hs-CRP (both $p < 0.001$), and more often had an anterior culprit lesion, high thrombus grade, multivessel disease and pre-procedural TIMI 0 flow (Table 1).

Haematological indices

Total leucocyte and neutrophil counts were higher, and lymphocyte counts lower, in the no-reflow group, so that admission NLR was more than twice as high (6.8 ± 2.9 vs 3.2 ± 1.4 ; $p < 0.001$). MPV, PDW and PLR were likewise significantly elevated (all $p < 0.001$), whereas haemoglobin did not differ (Table 2). All four indices correlated positively with cTFC and inversely with myocardial blush grade, the strongest correlation being observed for NLR ($r = 0.52$ and -0.48 , respectively; both $p < 0.001$). The incidence of no-reflow increased steeply across ascending NLR tertiles, from 8.8% to 26.2% and 52.5% (p for trend < 0.001 ; Fig. 2A).

Discriminative performance

NLR showed the largest AUC (0.82; 95% CI 0.76–0.88), followed by MPV (0.74), PDW (0.71) and PLR (0.70) (Table 3, Fig. 1). At a cut-off of >4.9 , NLR had a sensitivity of 78.6%, specificity of 74.7% and negative predictive value of 89.4%. The combined model yielded an AUC of 0.87 (95% CI 0.82–0.92), with sensitivity of 83.0% and specificity of 79.0%.

Independent predictors

On multivariable analysis, NLR (aOR 1.48 per unit), MPV (aOR 1.42 per fL), PDW (aOR 1.28 per %), diabetes mellitus (aOR 2.14), pain-to-balloon time >6 h (aOR 2.26) and high thrombus grade (aOR 2.58) remained independent predictors of no-reflow, whereas PLR, age >60 years, LVEF $<40\%$ and Killip class $>I$ did not (Table 4, Fig. 2B). The model showed good calibration (Hosmer–Lemeshow $p = 0.62$; Nagelkerke R² = 0.41).

Table 1: Baseline clinical, laboratory and angiographic characteristics according to reflow status

Variable	Overall (n = 240)	No-reflow (n = 70)	Normal reflow (n = 170)	p
Demographics and risk factors				
Age, years	57.6 ± 11.2	62.3 ± 11.2	55.7 ± 10.6	<0.001
Age >60 years	106 (44.2)	42 (60.0)	64 (37.6)	0.001
Male sex	178 (74.2)	54 (77.1)	124 (72.9)	0.49
Diabetes mellitus	96 (40.0)	40 (57.1)	56 (32.9)	<0.001
Hypertension	122 (50.8)	42 (60.0)	80 (47.1)	0.068
Current smoking	110 (45.8)	38 (54.3)	72 (42.4)	0.093
Dyslipidaemia	88 (36.7)	30 (42.9)	58 (34.1)	0.20
Clinical presentation				
Heart rate, bpm	82 ± 15	88 ± 16	80 ± 14	<0.001
Systolic BP, mmHg	124 ± 21	118 ± 22	126 ± 20	0.006
Killip class $>I$	72 (30.0)	31 (44.3)	41 (24.1)	0.002
Pain-to-balloon time, h	5.5 ± 2.6	6.8 ± 2.9	4.9 ± 2.2	<0.001
LVEF, %	46.4 ± 7.9	41.8 ± 7.9	48.3 ± 7.1	<0.001
Biochemistry				
Admission glucose, mg/dL	155 ± 52	186 ± 58	142 ± 44	<0.001
Serum creatinine, mg/dL	0.99 ± 0.26	1.05 ± 0.28	0.96 ± 0.24	0.012
LDL cholesterol, mg/dL	108 ± 32	118 ± 34	104 ± 30	0.002
hs-CRP, mg/L	5.4 ± 4.1	9.4 ± 4.6	3.8 ± 2.5	<0.001
Angiographic				
LAD as culprit artery	120 (50.0)	45 (64.3)	75 (44.1)	0.004
Pre-procedural TIMI 0 flow	151 (62.9)	51 (72.9)	100 (58.8)	0.038

High thrombus grade (4–5)	128 (53.3)	52 (74.3)	76 (44.7)	<0.001
Multivessel disease	108 (45.0)	40 (57.1)	68 (40.0)	0.015

Values are mean $\pm$ SD or n (%). BP, blood pressure; hs-CRP, high-sensitivity C-reactive protein; LAD, left anterior descending artery; LDL, low-density lipoprotein; LVEF, left-ventricular ejection fraction; TIMI, Thrombolysis in Myocardial Infarction. Overall values for continuous variables were pooled from group summaries. Mean stent length was 26.4 ± 6.8 mm and door-to-balloon time 48 ± 14 min.

Table 2: Haematological indices according to reflow status and their correlation with microvascular perfusion

Parameter	No-reflow (n = 70)	Normal reflow (n = 170)	p	r vscTFC	r vs MBG
Haemoglobin, g/dL	13.1 $\pm$ 1.8	13.4 $\pm$ 1.7	0.22	–	–
Total leucocyte count, $\times 10^3/\mu\text{L}$	12.8 $\pm$ 3.6	9.6 $\pm$ 2.8	<0.001	–	–
Neutrophils, $\times 10^3/\mu\text{L}$	10.4 $\pm$ 3.1	6.5 $\pm$ 2.3	<0.001	–	–
Lymphocytes, $\times 10^3/\mu\text{L}$	1.7 $\pm$ 0.6	2.4 $\pm$ 0.8	<0.001	–	–
Platelets, $\times 10^3/\mu\text{L}$	262 $\pm$ 68	244 $\pm$ 62	0.049	–	–
NLR, mean $\pm$ SD	6.8 $\pm$ 2.9	3.2 $\pm$ 1.4	<0.001	+0.52*	–0.48*
NLR, median (IQR)	6.3 (4.7–8.4)	3.0 (2.2–3.9)	<0.001	–	–
MPV, fL	10.9 $\pm$ 1.2	9.1 $\pm$ 1.0	<0.001	+0.41*	–0.37*
PDW, %	16.8 $\pm$ 2.4	13.9 $\pm$ 2.0	<0.001	+0.36*	–0.33*
PLR	178 $\pm$ 62	118 $\pm$ 48	<0.001	+0.34*	–0.29*

*p < 0.001. cTFC, corrected TIMI frame count; IQR, interquartile range; MBG, myocardial blush grade; MPV, mean platelet volume; NLR, neutrophil-to-lymphocyte ratio; PDW, platelet distribution width; PLR, platelet-to-lymphocyte ratio.

Table 3: Receiver-operating-characteristic analysis of haematological indices for predicting no-reflow

Marker	AUC	95% CI	Cut-off	Sens. (%)	Spec. (%)	PPV (%)	NPV (%)	Accuracy (%)
NLR	0.82	0.76–0.88	>4.9	78.6	74.7	56.1	89.4	75.8
MPV	0.74	0.67–0.81	>9.9 fL	71.4	68.8	48.5	85.4	69.6
PDW	0.71	0.64–0.78	>15.2%	68.6	65.9	45.3	83.6	66.7
PLR	0.70	0.63–0.77	>145	67.1	64.7	44.0	82.9	65.4
Combined model	0.87	0.82–0.92	–	83.0	79.0	–	–	–

All AUCs p < 0.001. Predictive values calculated at the observed no-reflow prevalence of 29.2%. The combined model included NLR, MPV, PDW and PLR. AUC, area under the curve; NPV, negative predictive value; PPV, positive predictive value; Sens., sensitivity; Spec., specificity.

Table 4: Univariable and multivariable logistic regression for predictors of no-reflow

Variable	Crude OR (95% CI)	p	Adjusted OR (95% CI)	p
NLR (per unit)	1.62 (1.40–1.88)	<0.001	1.48 (1.24–1.77)	<0.001
MPV (per fL)	1.71 (1.38–2.12)	<0.001	1.42 (1.11–1.82)	0.005
PDW (per %)	1.44 (1.22–1.70)	<0.001	1.28 (1.05–1.56)	0.014
PLR (per 10 units)	1.19 (1.09–1.30)	<0.001	NS	–
Diabetes mellitus	2.71 (1.52–4.83)	0.001	2.14 (1.14–4.02)	0.018
Pain-to-balloon time >6 h	2.48 (1.39–4.42)	0.002	2.26 (1.21–4.22)	0.011
High thrombus grade (4–5)	3.55 (1.98–6.36)	<0.001	2.58 (1.34–4.97)	0.005
Age >60 years	2.05 (1.16–3.62)	0.013	NS	–
LVEF <40%	2.30 (1.28–4.14)	0.005	NS	–
Killip class >I	2.36 (1.30–4.28)	0.005	NS	–

Hosmer–Lemeshow p = 0.62; Nagelkerke R² = 0.41. NS, not significant in the final model; OR, odds ratio. Other abbreviations as in Tables 1 and 2.

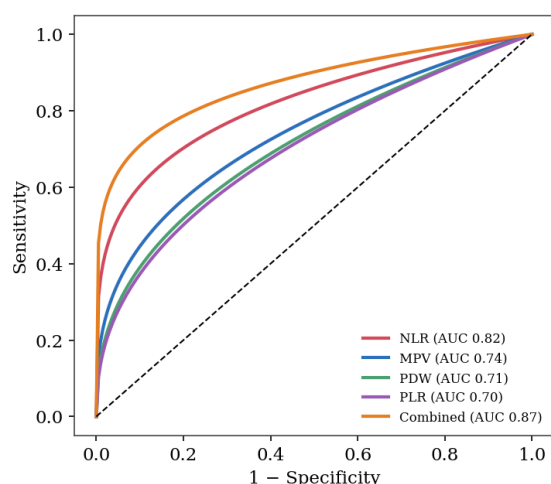


Figure 1: Receiver-operating-characteristic curves of the neutrophil-to-lymphocyte ratio (NLR), mean platelet volume (MPV), platelet distribution width (PDW), platelet-to-lymphocyte ratio (PLR) and the combined model for predicting no-reflow. AUC, area under the curve

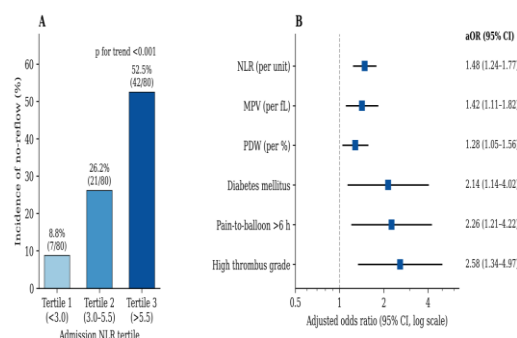


Figure 2: (A) Incidence of no-reflow across tertiles of admission neutrophil-to-lymphocyte ratio (NLR). (B) Independent predictors of no-reflow on multivariable logistic regression; squares denote adjusted odds ratios (aOR) and horizontal lines 95% confidence intervals. MPV, mean platelet volume; PDW, platelet distribution width

DISCUSSION

In this cohort of 240 patients undergoing primary PCI for STEMI, no-reflow occurred in 29.2%. Admission NLR, MPV and PDW each predicted no-reflow independently of diabetes, ischaemic time and thrombus burden; NLR was the best single discriminator, and combining inflammatory and platelet indices improved discrimination further. The risk of no-reflow rose almost six-fold from the lowest to the highest NLR tertile.

Our findings on NLR are concordant with previous reports. Sen et al. observed a progressive increase in impaired myocardial perfusion and three-year mortality across NLR tertiles,⁸ Wagdy et al. reported higher admission NLR in patients who developed no-reflow, new-onset atrial fibrillation or in-hospital adverse events,¹⁵ and Vakili et al. found a positive correlation between NLR and TIMI frame count.¹⁶ The graded tertile relationship and the correlation of

NLR with cTFC and blush grade in our study support a dose-dependent link between systemic inflammation and microvascular obstruction. Biologically, neutrophils adhere to injured endothelium, promote platelet-leucocyte aggregates and release myeloperoxidase, elastase and oxygen radicals that injure the microcirculation.^{4,7}

The platelet indices provided complementary information. After plaque rupture, rapid platelet consumption stimulates release of larger, granule-rich, more reactive platelets, raising MPV and PDW. Huczek et al. showed that a high admission MPV independently predicted no-reflow (odds ratio 4.7) and six-month mortality,⁹ and Kurtul et al. and Şenöz et al. identified pre-procedural PLR as an independent predictor of impaired reflow.^{10,17} In our model, PLR lost significance after adjustment, probably because it shares its lymphocyte denominator with NLR.

The AUC of NLR in our cohort (0.82) was higher than that reported by Zhang et al. (0.65), who also found no significant incremental value from combining NLR with platelet parameters.¹¹ Differences in timing of blood sampling (strictly before any antiplatelet loading dose in our study), longer ischaemic times and a higher prevalence of diabetes and high thrombus burden in our population may explain the stronger association. Our finding that a combined model outperformed individual markers is in keeping with Çiçek et al., who showed that combining NLR and PLR improved prognostic discrimination,¹⁸ and with recent work in which composite inflammatory-metabolic indices, such as the triglyceride-glucose index combined with NLR, achieved better prediction of no-reflow than either marker alone.^{19,20}

Diabetes, delayed reperfusion and high thrombus burden were independent predictors, consistent with the adverse effect of diabetes on myocardial perfusion described by Prasad et al.,⁶ and with the central role of distal embolisation and prolonged ischaemia in no-reflow.⁴ The persistence of NLR, MPV and PDW after adjustment for these factors indicates that haematological indices add information beyond conventional clinical and angiographic predictors. Because they are available from a single routine blood count within minutes of arrival, they could help flag patients for pre-emptive strategies—such as thrombus management, intracoronary vasodilators or glycoprotein IIb/IIIa inhibitors—in resource-constrained catheterisation laboratories. The high negative predictive value of NLR (89.4%) may be particularly useful for identifying low-risk patients.

Limitations

This was a single-centre study with a modest sample and a cross-sectional design, which limits generalisability and precludes causal inference. No-reflow was defined by epicardial TIMI flow, which does not capture all microvascular injury, and advanced imaging such as cardiac magnetic resonance was not available. Clinical outcomes

beyond the index hospitalisation were not assessed, the cut-off values were derived and tested in the same cohort without internal validation, and residual confounding cannot be excluded. Prospective, multicentre validation is required before these thresholds are adopted in practice.

CONCLUSION

In patients with STEMI undergoing primary PCI, admission NLR, MPV and PDW independently predict angiographic no-reflow. NLR is the most discriminating single marker, and combining it with platelet indices improves risk stratification. These inexpensive, universally available parameters may help identify high-risk patients before primary PCI.

Author contributions (CRediT)

Gulshan Kumar: Conceptualisation, Investigation, Data curation, Formal analysis, Writing – original draft. Deepak Maheshwari: Conceptualisation, Supervision, Methodology, Writing – review & editing. Confirm/edit for all authors.

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Declaration of competing interest

The authors have no conflicts of interest to declare.

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