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Original Article

Accuracy of Low Haemoglobin Level in Predicting No-Reflow Phenomenon in Primary Percutaneous Coronary Intervention Patients

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ABSTRACT

Objective: To determine the accuracy of low haemoglobin level in predicting the no-reflow phenomenon in patients undergoing primary percutaneous coronary intervention.

Study Design: Cross-sectional analytical study

Place and Duration of Study: Armed Forces Institute of Cardiology and National Institute of Heart Diseases (AFIC/NIHD), Rawalpindi, Pakistan from Dec 2024 to May 2025.

Methodology: A total of 115 patients aged 30-70 years, regardless of gender, with acute STEMI undergoing PPCI were enrolled through consecutive sampling. Post-coronary artery bypass graft (CABG) patients, those who had received glycoprotein IIb/IIIa inhibitors before the procedure, patients presenting beyond 24 hours of symptom onset, with chronic kidney disease, and with a history of major bleeding or haemorrhagic disorders were excluded. Low haemoglobin was defined as <11.5 g/dL. The no-reflow phenomenon was described as TIMI flow grade <3.

Results: Among 115 patients [105(91.3%) male; median age 57.00(51.00–62.00) years], no-reflow was observed in 30(26.1%) patients. Patients with low haemoglobin (<11.5 g/dL) experienced higher no-reflow than with normal haemoglobin (p=0.018). The sensitivity and specificity were 16.7% and 97.6%, respectively, with a positive predictive value (PPV) of 71.4%, a negative predictive value (NPV) of 76.8%, and an overall diagnostic accuracy of 76.5%.

Conclusion: Low haemoglobin level demonstrated high specificity but poor sensitivity in predicting the no-reflow phenomenon following PPCI.

Keywords: Haemoglobin, No-Reflow, Thrombolysis in Myocardial Infarction

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INTRODUCTION

The foremost objective in managing acute ST-segment elevation myocardial infarction (STEMI) is the rapid restoration of coronary blood flow.[1] Among reperfusion methods in use, primary percutaneous coronary intervention (PPCI) remains the gold standard for acute STEMI treatment due to its higher efficacy in developing vessel patency again and reducing myocardial injury.[2] However, despite successful revascularization, many patients experience inadequate myocardial perfusion, known as the no-reflow phenomenon. This condition, marked by impaired microvascular reperfusion in a reopened infarct-related artery, occurs in about 11–41% of PPCI-treated STEMI cases,[3,4] and is a strong predictor of poor cardiovascular outcomes, including recurrent ischemia, complications, and higher mortality.

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The pathogenesis of no-reflow is multifactorial, involving distal embolisation, ischemia-reperfusion injury, microvascular dysfunction, myocardial necrosis, and vasoconstriction mediated by α-adrenergic activity and platelet-derived substances such as thromboxane A_2 and serotonin. Microvascular blockage may worsen if procoagulant tissue factors are released from damaged plaques..[5–7] Predicting whether patients are at danger is still challenging despite advancements in interventional procedures, underscoring the necessity for accurate, straightforward predictors.

Haemoglobin level has gained attention among potential bio-markers, for its influence on myocardial oxygen delivery and perfusion. Low haemoglobin may worsen tissue hypoxia and microcirculatory recovery, thereby increasing susceptibility to no-reflow.[8] Yet, evidence regarding the link between baseline haemoglobin concentration and no-reflow incidence remains inconsistent and underexplored topic in Pakistan.

Given the limited regional data and conflicting findings in past literature, our study aims to evaluate the accuracy of low haemoglobin levels in predicting no-reflow phenomenon in patients of acute STEMI undergoing PPCI. Thus, identifying haemoglobin as an independent predictor could provide a cost-effective, accessible factor for timely risk assessment, facilitating clinicians in on-time interventions to improve reperfusion success and patient health outcomes.

METHODOLOGY

This cross-sectional analytical study was performed at the Cardiology Department of Armed Forces Institute of Cardiology and National Institute of Heart Diseases (AFIC/NIHD), Rawalpindi, Pakistan and spanned over a six-month period (from Dec 2024 to May 2025) following approval of the study synopsis from the Institutional Ethical Review Board (IERB) under letter number (9/2/R&D/2023/263; Dated: 25 May, 2023). The sample size of 87 was calculated via a sensitivity and specificity calculator, 90% confidence level, 49.3% prevalence of no-reflow, 80.4% sensitivity, 96.7% specificity of low haemoglobin,[9] and 10% margin of error were used. However, we gathered 115 study sample and participants were recruited via a non-probability consecutive sampling technique.

Inclusion Criteria: Patients aged 30-70 years of either gender who presented with STEMI and underwent PPCI were included.

Exclusion Criteria: Exclusion criteria comprised post-coronary artery bypass graft (CABG) patients, those who had received glycoprotein IIb/IIIa inhibitors before the procedure, patients presenting beyond 24 hours of symptom onset, individuals with chronic kidney disease, and those with a history of major bleeding or haemorrhagic disorders.

Following approval from the institutional ethics review board (IERB) and written informed consent, patients who presented to the emergency department and met the inclusion criteria were enrolled. Demographic and clinical variables such as age, gender, smoking, hypertension (BP ≥140/90 mmHg), diabetes mellitus (random blood sugar >200 mg/dL), dyslipidaemia (cholesterol >200 mg/dL), family history of STEMI were documented.

At admission, a 3cc venous blood sample was taken for blood complete picture test. Haemoglobin level of <11.5 g/dL was considered low and labelled as an indicator of no-reflow.[1] All patients underwent PPCI by administering local anaesthesia, performed by an experienced cardiology team with the assistance of the research scholar. The Thrombolysis in Myocardial Infarction (TIMI) flow grade was observed following vessel recanalization. The no-reflow phenomenon was defined as a TIMI flow grade <3 without any mechanical obstruction (e.g., spasm, dissection, or residual stenosis).[9]

Data was recorded using a predesigned proforma and entered in excel sheet which was imported to Statistical Package for the Social Sciences (SPSS) version 23. Normality was checked with Shapiro–Wilk test. Quantitative variables, such as age and haemoglobin level, were reported as means & standard deviations as they were normally distributed. Categorical variables (gender, smoking, alcoholism, diabetes, hypertension, dyslipidaemia, family history of STEMI, and no-reflow status) were presented as frequency & percentage. A 2×2 contingency table was developed to find the diagnostic accuracy, sensitivity, specificity, negative predictive value (NPV), positive predictive value (PPV) of low haemoglobin (<11.5 g/dL) in predicting the no-reflow phenomenon (TIMI<3) as compared to normal flow (TIMI=3).

RESULTS

Our study enrolled total 115 participants and their mean age was 56.50±8.39 years. The median symptom duration was 4.79(2.00–8.71) hours. The mean haemoglobin level was 14.16±1.75 mg/dL.

Table-I showed that majority of patients were male [105(91.3%)]. Among comorbidities, dyslipidaemia was the most prevalent, presented in 69(60.0%) participants, followed by smoking 64(55.7%), hypertension 61(53.0%), and diabetes mellitus 56(48.7%).

Table-I: Baseline characteristics of study participants (n=115)

Variables		Frequency (%)
Gender	Male	105(91.3%)
	Female	10(8.7%)
		Mean±SD
Age (years)		56.50±8.39
Haemoglobin (mg/dL)		14.16±1.75
		Median(IQR)
Symptom duration (hours)		4.79(2.00–8.71)
Comorbids Frequency (%)		
Smoker		64(55.7%)
Dyslipidaemia		69(60.0%)
Hypertension		61(53.0%)
Diabetes Mellitus		56(48.7%)
Family History of STEMI		50(43.5%)

STEMI=ST-Elevation Myocardial Infarction

Table-II illustrated the association between haemoglobin levels and the no-reflow phenomenon after PPCI. Among patients with low haemoglobin, 5 (16.7%) experienced no-reflow, whereas only 2 (2.4%) had normal flow. In comparison, patients with normal haemoglobin levels, 25(83.3%) had no-reflow, and 83(97.6%) maintained normal flow, and the association between low haemoglobin levels and no-reflow phenomenon was significant (p<0.05).

Table-II: Association between Haemoglobin and No-reflow phenomenon in Primary Percutaneous Coronary Intervention Patients (n=115)

Haemoglobin Level (mg/dL)	No Reflow on Primary Percutaneous Coronary Intervention		*p*-Value
	Yes (n=30)	No (n=85)	
<11.5	5(16.7%)	2(2.4%)	0.02
≥11.5	25(83.3%)	83(97.6%)	
Mean Haemoglobin Level (mg/dL)	13.72±2.20	14.32±1.54	<0.001

Findings from Table-III showed that low haemoglobin level (<11.5 g/dl) demonstrated high specificity (97.6%) but low sensitivity (16.7%) in predicting the no-reflow phenomenon among PPCI patients. This indicates that, while a low haemoglobin level is uncommon, its presence strongly suggests the presence of no-reflow. The positive predictive value was 71.4%, which shows that patients with low haemoglobin have a moderate probability of developing no-reflow. Conversely, the negative predictive value was 76.8%, indicating that normal haemoglobin levels do not completely exclude the possibility of no-reflow. The overall diagnostic accuracy was 76.5%, showing that haemoglobin level alone provides a fairly reliable but not highly sensitive indicator of no-reflow events after PPCI.

Table-III: Diagnostic Accuracy of Low Haemoglobin Level (Hb <11.5 g/dl) for Predicting No-Reflow Phenomenon in Primary Percutaneous Coronary Intervention Patients (n=115)

	No Reflow on Primary Percutaneous Coronary Intervention		Total
No Reflow based on Haemoglobin Level	Positive (TIMI <3)	Negative (TIMI= 3)	
Positive (Hb<11.5)	5 (TP)	2 (FP)	7
Negative (Hb≥11.5)	25(FN)	83(TN)	108
Total	30	85	115

Sensitivity = 16.7%; Specificity = 97.6%; Positive Predictive Value (PPV) = 0.714 = 71.4%; Negative Predictive Value (NPV) = 0.768 = 76.8%; Accuracy = 0.765 = 76.5%

Table-IV shows that, among all variables analysed, low haemoglobin (<11.5 g/dL) was the only statistically significant predictor. In the unadjusted model, low haemoglobin was associated with 8.30-fold increased odds of no-reflow (95% CI: 1.15–45.42, p=0.01). Haemoglobin remained a strong independent predictor with an adjusted OR of 8.30 (1.15–45.42), p=0.01.

Table-IV: Predictors of No-Reflow in PPCI Patients (n=115)

Variables	Unadjusted OR (95% CI)	*p*-value	Adjusted OR (95% CI)	*p*-value
Age (years)	0.98 (0.93–1.03)	0.52	-	-
Gender	1.80 (0.19–3.34)	0.76	-	-
Duration of symptoms	1.06 (0.98–1.15)	0.13	-	-
Smoking	1.53 (0.65–3.61)	0.32	-	-
Diabetes	0.62 (0.26–1.44)	0.27	-	-
Hypertension	1.46 (0.62–3.41)	0.37	-	-
Dyslipidemia	0.83 (0.35–1.92)	0.66	-	-
Hemoglobin (<11.5)	8.30 (1.15–45.42)	0.01*	8.30 (1.15–45.42)	0.01*

*Note. CI = confidence interval; OR = odds ratio; * = significant variables (p < 0.05)*

DISCUSSION

The present study evaluated the diagnostic accuracy of low haemoglobin levels in predicting the no-reflow phenomenon among patients with acute STEMI undergoing PPCI. In this analysis, a significant association was observed between reduced haemoglobin concentration and no-reflow, highlighting the potential role of baseline haemoglobin as a simple yet crucial prognostic indicator. The findings shown that even though low haemoglobin demonstrated high specificity, its sensitivity persisted limited, verified that anaemia may serve as a strong confirmatory but weak screening marker for no-reflow phenomenon. These results indicate the multifactorial nature of impaired myocardial reperfusion and support the hypothesis that reduced oxygen-carrying capacity may contribute to suboptimal microvascular recovery after PPCI.

In line with our study findings, numerous past studies have explored a significant association between low haemoglobin levels and the occurrence of the no-reflow phenomenon following PPCI. Sepehram et al. examined 1200 STEMI patients treated with primary PCI and reported that those with impaired post-procedural TIMI flow had significantly lower haemoglobin concentrations compared to those with normal flow (13.66±1.69 g/dL vs. 14.15±1.49 g/dL; p<0.001). Additionally, the low haemoglobin was an independent predictor of no-reflow (OR = 0.747; 95% CI: 0.618–0.888; p < 0.001), with excellent diagnostic performance (AUC = 0.872; sensitivity 80.4%; specificity 96.7%).9 Similarly, Yaylak et al. evaluated 3,804 PPCI patients and found that individuals who

developed no-reflow (12.4%) had significantly reduced haemoglobin levels (12.1±1.9 g/dL vs. 13.8 ± 1.8 g/dL; p < 0.001). Their results also identified low haemoglobin as an independent predictor (OR = 0.564; 95% CI: 0.526–0.605; p < 0.001), with a diagnostic cut-off of 11.5 g/dL (AUC = 0.844; sensitivity 83.0%; specificity 80.0%).[1] Compared with these studies, our results revealed no-reflow in 26.1% of patients and a similar, statistically significant association between low haemoglobin and no-reflow (p=0.018), reinforcing the predictive relevance of haemoglobin in STEMI patients undergoing PPCI. However, the lower sensitivity (16.7%) observed in our study subjects indicates that, although reduced haemoglobin is highly specific (97.6% specificity) for identifying patients at high risk, it may not be a sensitive marker alone. This difference could be attributed to variations in sample size, population characteristics, haemoglobin cut-off values, and inclusion criteria for participants in the study. However, the same direction of association across studies represents the physiological plausibility that anaemia compromises myocardial oxygen delivery, prompting patients to impaired microvascular reperfusion and no-reflow.[10,11]

Similar to our study outputs, Shakiba and colleagues reported that 20.4% of patients in their sample experienced no-reflow phenomenon, with significant difference in mean haemoglobin levels between groups with and without no-reflow groups (12.96±1.83 g/dL vs. 13.97±3.72 g/dL; p=0.021).[12] Similarly, our study demonstrated a 26.1% prevalence of no-reflow, with notably lower haemoglobin values among affected patients (13.72±2.20 vs 14.32±1.54), reemphasizing the premise that deficient oxygen-carrying capacity contributes to impaired myocardial perfusion.[13,14]

Unlike scarce local research directly addressing the aforementioned association, Pakistani research remains limited on the predictive role of haemoglobin in no-reflow. Nevertheless, Khan *et al.,* stated another hematological marker, the red cell distribution width (RDW), for predicting high thrombus burden, and reported 86.1% sensitivity and 66.5% specificity at a 14.5% cut-off.[15] Although their study focused on thrombus load instead of microvascular flow, it chains the emerging relevance of simple blood indices in predicting adverse procedural outcomes. Our study extended this perspective by evaluating that haemoglobin levels exhibit strong specificity (97.6%) for predicting the no-reflow phenomenon. This indicates that a basic hematological evaluation can provide meaningful clinical insight prior to PPCI.

Moreover, Haq and co-researchers from the National Institute of Cardiovascular Diseases (NICVD), Karachi, studied the prognostic importance of haemoglobin in treating the PPCI patients and observed a significant association between lower haemoglobin levels (7.0–11.0 g/dL) and an increased in-hospital death rate, which reached 8% in patients having haemoglobin <8.0 g/dL.16 Although our study did not assess mortality or long-term outcomes, the shared evidence presents that anemia not only predisposes to no-reflow but may also play role in poor prognosis.[17,18] Shah *et al.,* recruited cardiac patients from Hayatabad Medical Complex, Peshawar, and explained that patients with low hemoglobin level experienced higher mortality [20.0%, 10.0% and 4.0% severely anaemic, mildly anaemic, and without anaemia, respectively p<0.05]. Adverse events occurred in 30.0%, 17.5%, and 6.0% of these study subjects (p<0.05).[19] This features the clinical significance of careful pre-procedural haemoglobin assessment and timely corrective action to mitigate downstream complications such as no-reflow and its potential sequelae.

Our findings, supported by prior research, show the constitutive role of low haemoglobin levels in impaired myocardial reperfusion after PPCI by reducing oxygen delivery, exacerbating tissue hypoxia, and promoting microvascular dysfunction. These mechanisms explain the higher risk of no-reflow among anaemic patients despite successful epicardial recanalization. Clinically, this underscores the importance of measuring haemoglobin at admission as a simple and cost-effective method to identifying high-risk STEMI patients. Early correction of anaemia and careful peri-procedural management may help improve perfusion outcomes. However, because of its low sensitivity, haemoglobin alone cannot serve as a standalone predictor. A multifactorial model that includes clinical, angiographic, and biochemical factors is necessary for more accurate risk assessment. Overall, haemoglobin remains a useful but limited prognostic tool, underscoring the need for individualized management strategies to reduce no-reflow and improve the efficacy of reperfusion therapy in acute STEMI.

LIMITATIONS OF STUDY

This study was conducted at a single hospital and had a relatively small sample size, which may limit its

generalizability. Potential confounders such as inflammatory markers, renal function, and procedural variables were not fully evaluated, which may have influenced the predictive accuracy of haemoglobin levels.

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CONCLUSION

Low haemoglobin levels are strongly associated with the occurrence of the no-reflow phenomenon in patients undergoing PPCI for acute STEMI. Its high specificity highlights that it can serve as a useful indicator for identifying high-risk individuals, while its low sensitivity limits its usefulness as a standalone predictor.

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Authors' Contribution

Following authors have made substantial contributions to the manuscript as under:

MN & AAC: Data acquisition, data analysis, critical review, approval of the final version to be published.

UI & QUZB: Study design, data interpretation, drafting the manuscript, critical review, approval of the final version to be published.

GA & SP: Conception, data acquisition, drafting the manuscript, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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