

Analysis of Incidence, Predictors and Outcomes of No-Reflow Phenomenon During Primary PCI for STEMI: An Institutional Based Study

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ABSTRACT

Background: The no-reflow phenomenon is an important complication of primary percutaneous coronary intervention (PCI) in patients with ST-segment elevation myocardial infarction (STEMI). Despite successful restoration of epicardial coronary artery patency, impaired myocardial microvascular perfusion may contribute to adverse cardiovascular outcomes.

Aim: To analyze incidence, predictors and outcomes of no-reflow phenomenon during primary PCI for STEMI.

Materials and Methods: This prospective observational study included 74 patients with STEMI undergoing primary PCI. Patients were categorized into Group I (no-reflow) and Group II (normal coronary flow). Demographic characteristics, cardiovascular risk factors, clinical presentation, biochemical parameters, angiographic findings, procedural characteristics, and clinical outcomes were evaluated. Statistical analysis was performed using SPSS version 21.0, with $p < 0.05$ considered statistically significant.

Results: No-reflow occurred in 18 patients (24.32%), while 56 patients (75.68%) achieved normal coronary reperfusion. Patients with no-reflow had significantly higher mean age (61.00 ± 9.76 versus 54.80 ± 10.82 years; $p = 0.034$). Diabetes mellitus (61.11% versus 30.36%; $p = 0.019$), hypertension (66.67% versus 37.50%; $p = 0.030$), high thrombus burden (83.33% versus 44.64%; $p = 0.004$), and prolonged symptom-to-balloon time ($p = 0.001$) were significantly associated with no-reflow. Elevated neutrophil-to-lymphocyte ratio ($p < 0.001$) and reduced left ventricular ejection fraction ($p = 0.011$) were also observed. Patients with no-reflow experienced significantly

higher rates of acute heart failure (38.89% versus 10.71%; $p = 0.012$), cardiogenic shock (33.33% versus 7.14%; $p = 0.011$), 30-day mortality (16.67% versus 1.79%; $p = 0.042$), and major adverse cardiovascular events (38.89% versus 7.14%; $p = 0.003$).

Conclusion: No-reflow was associated with adverse clinical characteristics, impaired myocardial reperfusion, and increased cardiovascular complications. Early risk identification and timely reperfusion may improve outcomes following primary PCI.

Keywords: ST-Segment Elevation Myocardial Infarction; No-Reflow Phenomenon; Primary Percutaneous Coronary Intervention; Thrombus Burden; Major Adverse Cardiovascular Events.

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INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) represents one of the most serious manifestations of acute coronary syndrome and remains a major contributor to cardiovascular morbidity and mortality worldwide. It occurs predominantly following acute thrombotic occlusion of an epicardial coronary artery, resulting in myocardial ischemia and progressive myocardial necrosis. Early restoration of coronary blood flow is essential for limiting infarct size, preserving ventricular function, and improving clinical outcomes. Primary percutaneous coronary intervention (PCI) has become the preferred reperfusion strategy for eligible patients with STEMI because it provides rapid

mechanical restoration of coronary artery patency. However, successful reopening of the infarct-related artery does not invariably ensure adequate myocardial tissue perfusion. The persistence of impaired myocardial perfusion despite successful coronary intervention represents an important clinical challenge.¹ The no-reflow phenomenon is characterized by inadequate myocardial perfusion despite successful restoration of epicardial coronary artery patency, in the absence of significant residual stenosis, coronary dissection, epicardial vasospasm, or other mechanical obstruction. Angiographically, it may be identified by reduced Thrombolysis in Myocardial Infarction (TIMI) flow grades

or impaired myocardial blush despite apparently satisfactory epicardial coronary flow. The phenomenon may develop during balloon dilatation, stent implantation, or immediately following restoration of coronary circulation. No-reflow reflects dysfunction of the coronary microcirculation and represents an important limitation of otherwise successful primary PCI. Its occurrence may compromise myocardial salvage and interfere with the expected benefits of reperfusion therapy. Therefore, identifying patients at increased risk of developing no-reflow remains an important component of interventional cardiology practice.²

The pathophysiology of no-reflow is complex and multifactorial, involving interactions between ischemic injury, microvascular dysfunction, distal embolization, inflammatory activation, and reperfusion-related tissue damage. Prolonged coronary occlusion causes endothelial swelling, capillary narrowing, and structural microvascular injury. Following mechanical reperfusion, distal embolization of thrombotic material and atherosclerotic plaque fragments may further compromise the myocardial microcirculation. Additional mechanisms include platelet activation, leukocyte accumulation, oxidative stress, endothelial dysfunction, and impaired microvascular vasodilatation. Culprit plaque morphology, lipid-rich necrotic material, and thrombus characteristics may also influence the development of microvascular obstruction. These pathological processes may coexist and contribute to persistent myocardial hypoperfusion despite technically successful coronary intervention.³

Several demographic, clinical, and angiographic characteristics have been investigated as potential predictors of no-reflow in patients undergoing primary PCI. Advanced age, diabetes mellitus, hypertension, renal dysfunction, hemodynamic instability, and impaired left ventricular function are important clinical variables requiring evaluation. Delayed presentation following the onset of ischemic symptoms may increase the vulnerability of the coronary microcirculation to reperfusion injury. Angiographic factors such as initial TIMI flow grade, thrombus burden, culprit lesion length, infarct-related artery, and extent of coronary artery disease may influence procedural outcomes. Procedural characteristics, including balloon predilatation, stent implantation, thrombus aspiration, and administration of adjunctive antithrombotic agents, also require assessment when evaluating no-reflow. Recognition of these characteristics may facilitate appropriate procedural planning and individualized risk assessment.⁴

Inflammatory and biochemical markers have received increasing attention in the evaluation of myocardial reperfusion abnormalities. Acute myocardial infarction activates inflammatory pathways involving neutrophils, lymphocytes, platelets, cytokines, and vascular endothelial cells. Neutrophil-mediated microvascular plugging, oxidative injury, and inflammatory cell recruitment may contribute to impaired myocardial tissue perfusion. The neutrophil-to-lymphocyte ratio represents a readily available hematological marker of systemic inflammation and has been investigated in relation to coronary reperfusion abnormalities. Similarly, admission blood glucose, renal function parameters, and other biochemical indicators may reflect metabolic disturbances and underlying cardiovascular risk. Evaluation of these routinely available investigations alongside clinical and angiographic variables may contribute to a more comprehensive assessment of patients undergoing primary PCI.⁵

Assessment of successful myocardial reperfusion requires consideration of both epicardial coronary flow and microvascular tissue perfusion. TIMI flow grading provides an angiographic evaluation of coronary blood flow, whereas myocardial blush grading offers additional information regarding myocardial tissue perfusion. Electrocardiographic ST-segment resolution following PCI is another practical method for assessing the adequacy of reperfusion. Echocardiographic evaluation of left ventricular ejection fraction provides information regarding myocardial functional recovery and ventricular systolic performance. Advanced imaging techniques, particularly cardiac magnetic resonance imaging, can further characterize infarct size, microvascular obstruction, and myocardial injury. Together, these assessments provide clinically relevant information regarding the extent of reperfusion and the risk of subsequent adverse cardiovascular outcomes.⁶

MATERIALS AND METHODS

The present study was designed as a prospective observational study conducted at a Department of Cardiology, Geetanjali Medical College and Hospital, Udaipur, Rajasthan, India. A total of 74 patients diagnosed with ST-segment elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention (PCI) were included. The study aimed to determine the incidence of the no-reflow phenomenon, identify associated clinical, biochemical, and angiographic predictors, and evaluate its influence on procedural success and subsequent clinical outcomes. A total of 74 consecutive eligible patients undergoing primary PCI for STEMI were enrolled using a consecutive sampling technique. Patients were categorized into two groups according to angiographic findings: Group I included patients who developed the no-reflow phenomenon, whereas Group II comprised patients who achieved normal coronary reperfusion. Both groups were compared regarding baseline characteristics, cardiovascular risk factors, angiographic parameters, procedural characteristics, and clinical outcomes.

Inclusion Criteria: Patients aged 18 years or above with a confirmed diagnosis of STEMI based on characteristic chest pain, electrocardiographic ST-segment elevation, and cardiac biomarker findings were included. Patients undergoing primary PCI of the infarct-related coronary artery without prior fibrinolytic therapy and those providing informed consent were considered eligible.

Exclusion Criteria: Patients presenting with non-ST-segment elevation myocardial infarction, unstable angina, or undergoing elective PCI were excluded. Patients who had received thrombolytic therapy before PCI, underwent rescue PCI, or had infarction involving a coronary bypass graft were excluded. Patients with incomplete essential clinical or angiographic records were also excluded.

Methodology: Detailed clinical histories and physical examinations were performed. Demographic parameters included age, sex, body mass index, and smoking status. Cardiovascular risk factors, including diabetes mellitus, hypertension, dyslipidemia, previous myocardial infarction, family history of coronary artery disease, and chronic kidney disease, were documented. Clinical parameters included presenting symptoms, heart rate, systolic and diastolic blood pressure, Killip classification, cardiogenic shock, symptom-to-balloon time, and

door-to-balloon time. Twelve-lead electrocardiography was performed to determine infarct location and baseline ST-segment elevation.

Laboratory and Echocardiographic Evaluation: Laboratory investigations included complete blood count, hemoglobin, total leukocyte count, neutrophil-to-lymphocyte ratio, platelet count, random blood glucose, HbA1c, serum creatinine, estimated glomerular filtration rate, lipid profile, and cardiac troponin levels. Transthoracic echocardiography was performed after stabilization to evaluate left ventricular ejection fraction, regional wall motion abnormalities, and left ventricular systolic dysfunction.

Coronary Angiography and Procedural Assessment: All patients underwent emergency coronary angiography followed by primary PCI according to standard institutional protocols. Angiographic parameters included the infarct-related artery, number of diseased vessels, culprit lesion location, lesion length, percentage diameter stenosis, reference vessel diameter, preprocedural Thrombolysis in Myocardial Infarction (TIMI) flow grade, and angiographic thrombus burden using TIMI thrombus grading. Procedural characteristics included balloon predilatation, stent diameter and length, number of stents, thrombus aspiration, and use of glycoprotein IIb/IIIa inhibitors. Postprocedural TIMI flow and myocardial blush grade were documented.

Assessment of No-Reflow Phenomenon: The no-reflow phenomenon was defined as impaired coronary perfusion following mechanical reopening of the infarct-related artery, characterized by TIMI flow grade 0–2 or TIMI grade 3 with myocardial blush grade 0–1, in the absence of significant residual stenosis, coronary dissection, epicardial spasm, or persistent mechanical obstruction. Angiographic flow was assessed during and after PCI. The occurrence of transient or persistent no-reflow, administration of intracoronary vasodilators, and subsequent improvement in coronary flow were documented.

Assessment of Predictors: Potential predictors of no-reflow included advanced age, diabetes mellitus, hypertension, smoking, elevated blood glucose, impaired renal function, increased neutrophil-to-lymphocyte ratio, prolonged symptom-to-balloon time, higher Killip class, anterior wall myocardial infarction, reduced initial TIMI flow, high thrombus burden, multivessel coronary artery disease, and longer culprit lesions. Associations between these variables and the development of no-reflow were evaluated.

Assessment of Clinical Outcomes: The primary outcome was the incidence of angiographic no-reflow during primary PCI. Secondary outcomes included procedural success, ST-segment resolution of at least 50% within 60–90 minutes after PCI, left ventricular ejection fraction, recurrent chest pain, cardiogenic shock, acute heart failure, ventricular arrhythmias, reinfarction, stroke, in-hospital mortality, and duration of hospitalization. Major adverse cardiovascular events, including cardiovascular death, recurrent myocardial infarction, and unplanned target-vessel revascularization, were assessed during hospitalization and at 30-day follow-up.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS), version 21.0. Continuous variables were expressed as mean $\pm$ standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro–Wilk

test. Independent Student's t-test or Mann–Whitney U test was used for continuous variables, whereas the Chi-square test or Fisher's exact test was applied to categorical variables. Univariable logistic regression was performed to identify potential predictors of no-reflow. Multivariable logistic regression was considered according to the number of observed events to avoid overfitting, with results expressed as odds ratios and 95% confidence intervals. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Comparison of Baseline Demographic Characteristics and Cardiovascular Risk Factors (n = 74)

The overall mean age of the patients was 56.31 ± 10.84 years. Patients in Group I had a significantly higher mean age of 61.00 ± 9.76 years compared with 54.80 ± 10.82 years in Group II ($p = 0.034$). Male predominance was observed in both groups, with 14 patients (77.78%) in Group I and 39 patients (69.64%) in Group II being males. Overall, 53 patients (71.62%) were males, and the difference in sex distribution between the groups was statistically insignificant ($p = 0.505$). The mean body mass index was 27.94 ± 3.66 kg/m² in Group I and 26.89 ± 3.41 kg/m² in Group II, with an overall mean of 27.15 ± 3.48 kg/m². Although the mean BMI was higher among patients with no-reflow, the difference was not statistically significant ($p = 0.268$). Regarding cardiovascular risk factors, diabetes mellitus was present in 11 patients (61.11%) in Group I compared with 17 patients (30.36%) in Group II, accounting for 28 patients (37.84%) of the total study population. The difference was statistically significant ($p = 0.019$). Hypertension was documented in 12 patients (66.67%) in Group I and 21 patients (37.50%) in Group II, with an overall prevalence of 44.59%, showing a statistically significant difference ($p = 0.030$).

Table 2: Comparison of Clinical Presentation, Laboratory Parameters, and Reperfusion Time (n = 74)

Killip class II or higher was documented in 11 patients (61.11%) in Group I compared with 13 patients (23.21%) in Group II. Overall, 24 patients (32.43%) presented with Killip class $\geq$ II, and the difference between the groups was statistically significant ($p = 0.003$). Anterior wall STEMI was identified in 12 patients (66.67%) in Group I and 24 patients (42.86%) in Group II, accounting for 36 patients (48.65%) of the total population. Although anterior wall STEMI was more frequent in the no-reflow group, the difference was statistically insignificant ($p = 0.079$). A significant difference was observed in symptom-to-balloon time between the groups. The mean symptom-to-balloon time was 340.00 ± 110.00 minutes in Group I compared with 250.00 ± 90.00 minutes in Group II, with an overall mean of 271.89 ± 102.14 minutes ($p = 0.001$). The mean door-to-balloon time was 92.00 ± 24.00 minutes in Group I and 86.00 ± 21.00 minutes in Group II, with an overall mean of 87.46 ± 21.75 minutes. However, the difference in door-to-balloon time was not statistically significant ($p = 0.312$).

Table 3: Comparison of Coronary Angiographic Findings and Procedural Characteristics (n = 74)

The left anterior descending artery was the most frequently involved infarct-related artery, identified in 36 patients (48.65%), followed by the right coronary artery in 30 patients (40.54%) and the left circumflex artery in 8 patients (10.81%). In Group I, the left anterior descending artery was involved in 12 patients (66.67%), the right coronary artery in 5 patients (27.78%), and the left

circumflex artery in 1 patient (5.56%). Corresponding frequencies in Group II were 24 (42.86%), 25 (44.64%), and 7 (12.50%), respectively. The overall distribution of infarct-related arteries did not differ significantly between the groups ($p = 0.268$). Preprocedural TIMI flow grade 0 was observed in 16 patients (88.89%) in Group I compared with 39 patients (69.64%) in Group II, representing 55 patients (74.32%) overall. The difference was not statistically significant ($p = 0.130$). High thrombus burden, defined as TIMI thrombus grade 4–5, was documented in 15 patients (83.33%) in Group I compared with 25 patients (44.64%) in Group II. Overall, 40 patients (54.05%) had a high thrombus burden, with a statistically significant difference between the groups ($p = 0.004$). Multivessel coronary artery disease was identified in 12 patients (66.67%) in Group I and 20 patients (35.71%) in Group II, accounting for 32 patients (43.24%) overall. This difference was statistically significant ($p = 0.021$). Similarly, culprit lesion length ≥ 20 mm was observed in 13 patients (72.22%) in Group I compared with 23 patients (41.07%) in Group II, with an overall frequency of 48.65% and a statistically significant difference ($p = 0.021$).

Table 4: Incidence, Angiographic Characteristics, and Reperfusion Outcomes of the No-Reflow Phenomenon (n = 74)

Among the 74 patients undergoing primary PCI, no-reflow was identified in 18 patients, corresponding to an overall incidence of 24.32%. Among these 18 patients, 14 (77.78%) demonstrated TIMI flow grade 0–2 following coronary reopening, representing 18.92% of the total study population. The remaining 4 patients (22.22%) had TIMI flow grade 3 with myocardial blush grade 0–1, accounting for 5.41% of the total population. Regarding the status of no-reflow at procedure completion, transient or resolved no-reflow was documented in 10 patients (55.56%) of Group I, whereas persistent no-reflow was present in 8 patients (44.44%).

These represented 13.51% and 10.81% of the overall study population, respectively. Intracoronary vasodilators were administered to all 18 patients (100.00%) in Group I compared with 5 patients (8.93%) in Group II. Overall, 23 patients (31.08%) received intracoronary vasodilators, and the difference between the groups was highly statistically significant ($p < 0.001$). Final TIMI flow grade 3 was achieved in 11 patients (61.11%) in Group I and all 56 patients (100.00%) in Group II, resulting in an overall frequency of 90.54%. Final myocardial blush grade 2–3 was documented in 10 patients (55.56%) in Group I compared with all 56 patients (100.00%) in Group II, accounting for 66 patients (89.19%) of the study population.

Table 5: Comparison of In-Hospital Complications and 30-Day Clinical Outcomes (n = 74)

Acute heart failure was recorded in 7 patients (38.89%) in Group I compared with 6 patients (10.71%) in Group II, accounting for 13 patients (17.57%) overall. The difference was statistically significant ($p = 0.012$). Ventricular arrhythmias were documented in 5 patients (27.78%) in Group I and 5 patients (8.93%) in Group II, with an overall frequency of 13.51%. However, the difference did not reach statistical significance ($p = 0.056$). Cardiogenic shock was observed in 6 patients (33.33%) in Group I compared with 4 patients (7.14%) in Group II, representing 10 patients (13.51%) of the study population. This difference was statistically significant ($p = 0.011$). In-hospital mortality occurred in 2 patients (11.11%) in Group I compared with 1 patient (1.79%) in Group II, accounting for 3 deaths (4.05%) overall. Although the proportion was higher in Group I, the difference was not statistically significant ($p = 0.145$). The mean duration of hospitalization was 6.80 ± 2.20 days in Group I compared with 4.60 ± 1.60 days in Group II, with an overall mean of 5.14 ± 1.99 days. The duration of hospitalization was significantly longer among patients in Group I ($p < 0.001$).

Table 1: Comparison of Baseline Demographic Characteristics and Cardiovascular Risk Factors (n = 74)

Parameters	Group I: No-reflow (n = 18)	Group II: Normal flow (n = 56)	Total (n = 74)	p-value
Age (years), Mean $\pm$ SD	61.00 $\pm$ 9.76	54.80 $\pm$ 10.82	56.31 $\pm$ 10.84	0.034
Male sex	14 (77.78%)	39 (69.64%)	53 (71.62%)	0.505
BMI (kg/m ²), Mean $\pm$ SD	27.94 $\pm$ 3.66	26.89 $\pm$ 3.41	27.15 $\pm$ 3.48	0.268
Diabetes mellitus	11 (61.11%)	17 (30.36%)	28 (37.84%)	0.019
Hypertension	12 (66.67%)	21 (37.50%)	33 (44.59%)	0.030
Current smoking	9 (50.00%)	24 (42.86%)	33 (44.59%)	0.596
Chronic kidney disease	5 (27.78%)	5 (8.93%)	10 (13.51%)	0.056

Table 2: Comparison of Clinical Presentation, Laboratory Parameters, and Reperfusion Time (n = 74)

Parameters	Group I: No-reflow (n = 18)	Group II: Normal flow (n = 56)	Total (n = 74)	p-value
Killip class $\geq$ II	11 (61.11%)	13 (23.21%)	24 (32.43%)	0.003
Anterior wall STEMI	12 (66.67%)	24 (42.86%)	36 (48.65%)	0.079
Symptom-to-balloon time (minutes), Mean $\pm$ SD	340.00 $\pm$ 110.00	250.00 $\pm$ 90.00	271.89 $\pm$ 102.14	0.001
Door-to-balloon time (minutes), Mean $\pm$ SD	92.00 $\pm$ 24.00	86.00 $\pm$ 21.00	87.46 $\pm$ 21.75	0.312
Random blood glucose (mg/dL), Mean $\pm$ SD	224.00 $\pm$ 63.00	180.00 $\pm$ 52.00	190.70 $\pm$ 57.64	0.004
Neutrophil-to-lymphocyte ratio, Mean $\pm$ SD	6.70 $\pm$ 2.20	4.30 $\pm$ 1.80	4.88 $\pm$ 2.15	<0.001
Serum creatinine (mg/dL), Mean $\pm$ SD	1.34 $\pm$ 0.40	1.12 $\pm$ 0.30	1.17 $\pm$ 0.34	0.015

Table 3: Comparison of Coronary Angiographic Findings and Procedural Characteristics (n = 74)

Parameters	Group I: No-reflow (n = 18)	Group II: Normal flow (n = 56)	Total (n = 74)	p-value
Infarct-related artery				0.268
Left anterior descending artery	12 (66.67%)	24 (42.86%)	36 (48.65%)	—
Right coronary artery	5 (27.78%)	25 (44.64%)	30 (40.54%)	—
Left circumflex artery	1 (5.56%)	7 (12.50%)	8 (10.81%)	—
Preprocedural TIMI flow grade 0	16 (88.89%)	39 (69.64%)	55 (74.32%)	0.130
High thrombus burden (TIMI grade 4–5)	15 (83.33%)	25 (44.64%)	40 (54.05%)	0.004
Multivessel coronary artery disease	12 (66.67%)	20 (35.71%)	32 (43.24%)	0.021
Culprit lesion length ≥20 mm	13 (72.22%)	23 (41.07%)	36 (48.65%)	0.021
Balloon predilatation	13 (72.22%)	25 (44.64%)	38 (51.35%)	0.042
Thrombus aspiration	9 (50.00%)	14 (25.00%)	23 (31.08%)	0.046
Glycoprotein IIb/IIIa inhibitor administration	12 (66.67%)	19 (33.93%)	31 (41.89%)	0.014

Table 4: Incidence, Angiographic Characteristics, and Reperfusion Outcomes of the No-Reflow Phenomenon (n = 74)

Parameters	Group I: No-reflow (n = 18)	Group II: Normal flow (n = 56)	Total (n = 74)	p-value
Initial no-reflow manifestation				
TIMI flow grade 0–2 after reopening	14 (77.78%)	—	14 (18.92%)	—
TIMI flow grade 3 with myocardial blush grade 0–1	4 (22.22%)	—	4 (5.41%)	—
No-reflow status at procedure completion				
Transient/resolved no-reflow	10 (55.56%)	—	10 (13.51%)	—
Persistent no-reflow	8 (44.44%)	—	8 (10.81%)	—
Intracoronary vasodilator administration	18 (100.00%)	5 (8.93%)	23 (31.08%)	<0.001
Final TIMI flow grade 3	11 (61.11%)	56 (100.00%)	67 (90.54%)	—
Final myocardial blush grade 2–3	10 (55.56%)	56 (100.00%)	66 (89.19%)	—
ST-segment resolution ≥50%	8 (44.44%)	43 (76.79%)	51 (68.92%)	0.010
Post-PCI left ventricular ejection fraction (%), Mean ± SD	42.30 ± 7.80	47.90 ± 8.00	46.54 ± 8.26	0.011

Table 5: Comparison of In-Hospital Complications and 30-Day Clinical Outcomes (n = 74)

Parameters	Group I: No-reflow (n = 18)	Group II: Normal flow (n = 56)	Total (n = 74)	p-value
Acute heart failure	7 (38.89%)	6 (10.71%)	13 (17.57%)	0.012
Ventricular arrhythmias	5 (27.78%)	5 (8.93%)	10 (13.51%)	0.056
Cardiogenic shock	6 (33.33%)	4 (7.14%)	10 (13.51%)	0.011
In-hospital mortality	2 (11.11%)	1 (1.79%)	3 (4.05%)	0.145
Duration of hospitalization (days), Mean ± SD	6.80 ± 2.20	4.60 ± 1.60	5.14 ± 1.99	<0.001
Recurrent myocardial infarction within 30 days	2 (11.11%)	2 (3.57%)	4 (5.41%)	0.247
Unplanned target-vessel revascularization within 30 days	2 (11.11%)	1 (1.79%)	3 (4.05%)	0.145
All-cause mortality within 30 days	3 (16.67%)	1 (1.79%)	4 (5.41%)	0.042
Composite MACE within 30 days	7 (38.89%)	4 (7.14%)	11 (14.86%)	0.003

DISCUSSION

The present study included 74 patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention, among whom 18 patients (24.32%) developed the no-reflow phenomenon, whereas 56 patients (75.68%) achieved normal coronary reperfusion. The mean age was significantly higher in patients with no-reflow compared with those having

normal coronary flow (61.00 ± 9.76 years versus 54.80 ± 10.82 years; $p = 0.034$). Male predominance was observed in both groups, accounting for 77.78% and 69.64%, respectively, although the difference was statistically insignificant ($p = 0.505$). Similarly, the mean body mass index was comparatively higher in the no-reflow group (27.94 ± 3.66 kg/m² versus 26.89 ± 3.41 kg/m²; $p =$

0.268). These findings were comparable with those reported by Sabin et al. (2017), who evaluated 181 patients undergoing primary PCI and observed angiographic no-reflow in 47 patients (25.90%). The mean age of their study population was 59.19 ± 10.25 years. Advanced age above 60 years was significantly associated with no-reflow, with an unadjusted odds ratio of 6.146 (95% CI: 2.937 – 12.86; $p < 0.001$). Multivariable analysis also identified age above 60 years as an independent predictor, with an odds ratio of 8.886 ($p = 0.003$).⁷

In the present study, diabetes mellitus was significantly more common among patients who developed no-reflow than among those with normal coronary flow (61.11% versus 30.36%; $p = 0.019$). Hypertension also demonstrated a statistically significant difference, being present in 66.67% of the no-reflow group compared with 37.50% of the normal-flow group ($p = 0.030$). Current smoking was documented in 50.00% and 42.86% of patients, respectively ($p = 0.596$), while chronic kidney disease was present in 27.78% and 8.93% ($p = 0.056$). Although smoking and chronic kidney disease were more frequent among patients with no-reflow, these differences were statistically insignificant. De Luca et al. (2013) evaluated 1,662 patients with STEMI undergoing primary angioplasty and reported hypertension in 700 patients (42.10%). Patients with hypertension demonstrated lower rates of satisfactory myocardial blush grade 2–3 compared with those without hypertension (68.20% versus 74.20%; $p = 0.019$). Complete ST-segment resolution was also less frequent among hypertensive patients (51.70% versus 61.10%; $p = 0.001$). Furthermore, mortality was significantly higher among hypertensive patients (6.20% versus 2.90%), with an adjusted hazard ratio of 1.82.⁸

The clinical characteristics and reperfusion-related parameters demonstrated significant differences between the study groups. Killip class II or higher was observed in 61.11% of patients with no-reflow compared with 23.21% of patients with normal coronary flow ($p = 0.003$). Anterior wall STEMI was also more frequent among patients with no-reflow (66.67% versus 42.86%), although the difference did not reach statistical significance ($p = 0.079$). The mean symptom-to-balloon time was significantly prolonged in the no-reflow group (340.00 ± 110.00 minutes) compared with the normal-flow group (250.00 ± 90.00 minutes; $p = 0.001$). However, the difference in mean door-to-balloon time was insignificant (92.00 ± 24.00 versus 86.00 ± 21.00 minutes; $p = 0.312$). Similar observations regarding reperfusion delay were reported by De Luca et al. (2004), who evaluated 1,072 patients with STEMI treated by primary angioplasty. Their study demonstrated that longer symptom-to-treatment time was significantly associated with impaired ST-segment resolution, reduced myocardial blush grade, increased enzymatic infarct size, and higher one-year mortality. Following adjustment for baseline variables, time-to-treatment remained significantly associated with impaired ST-segment resolution (adjusted OR = 1.01; 95% CI: 1.01–1.02; $p < 0.001$) and impaired myocardial blush (adjusted OR = 1.01; 95% CI: 1.01–1.02; $p < 0.0001$).⁹

The laboratory parameters in the present study demonstrated significant differences between patients with no-reflow and those with normal coronary reperfusion. The mean random blood glucose concentration was significantly higher in the no-reflow group (224.00 ± 63.00 mg/dL) compared with the normal-flow group (180.00 ± 52.00 mg/dL; $p = 0.004$). Similarly, the mean

neutrophil-to-lymphocyte ratio was significantly elevated among patients with no-reflow (6.70 ± 2.20 versus 4.30 ± 1.80 ; $p < 0.001$). Serum creatinine levels were also significantly higher in the no-reflow group (1.34 ± 0.40 mg/dL) compared with the normal-flow group (1.12 ± 0.30 mg/dL; $p = 0.015$). Sen et al. (2013) investigated 204 consecutive STEMI patients undergoing primary PCI and demonstrated a significant association between elevated neutrophil-to-lymphocyte ratio and impaired myocardial reperfusion. Patients were classified into three groups according to admission neutrophil-to-lymphocyte ratio, and the occurrence of angiographic and electrocardiographic no-reflow increased across successive tertiles. The corresponding three-year mortality rates were 9.00%, 15.00%, and 35.00%, respectively ($p < 0.01$). Elevated neutrophil-to-lymphocyte ratio was also associated with increased long-term major adverse cardiovascular events.¹⁰

The angiographic findings in the present study demonstrated that the left anterior descending artery was the most frequently involved infarct-related vessel, accounting for 48.65% of patients, followed by the right coronary artery (40.54%) and left circumflex artery (10.81%). Preprocedural TIMI flow grade 0 was more frequent in patients with no-reflow than in those with normal coronary flow (88.89% versus 69.64%; $p = 0.130$). High thrombus burden was significantly more frequent among patients with no-reflow (83.33% versus 44.64%; $p = 0.004$). Similarly, multivessel coronary artery disease was observed in 66.67% and 35.71% of patients, respectively ($p = 0.021$), while culprit lesion length ≥ 20 mm was documented in 72.22% and 41.07% ($p = 0.021$). Comparable angiographic findings were reported by Mazhar et al. (2016), who analyzed 781 patients undergoing primary PCI and identified no-reflow in 189 patients. A high thrombus score ≥ 4 was significantly more common in patients with no-reflow than in those with normal coronary flow (83.00% versus 69.00%; $p = 0.0001$). Similarly, impaired initial TIMI flow was observed in 89.00% and 79.00% of patients, respectively ($p = 0.001$). Their multivariable analysis identified high thrombus burden, age above 60 years, and symptom-to-balloon time exceeding 360 minutes as independent predictors of no-reflow.¹¹

Regarding procedural characteristics, balloon predilatation was performed more frequently among patients who developed no-reflow than among those with normal coronary flow (72.22% versus 44.64%; $p = 0.042$). Thrombus aspiration was performed in 50.00% of patients with no-reflow compared with 25.00% in the normal-flow group ($p = 0.046$). Similarly, glycoprotein IIb/IIIa inhibitors were administered to 66.67% and 33.93% of patients, respectively ($p = 0.014$). These differences indicated greater utilization of adjunctive procedural and pharmacological interventions among patients experiencing no-reflow. Jolly et al. (2015), in the randomized TOTAL trial, evaluated 10,732 STEMI patients undergoing primary PCI and compared routine manual thrombectomy with conventional PCI alone. The primary composite outcome of cardiovascular death, recurrent myocardial infarction, cardiogenic shock, or severe heart failure at 180 days occurred in 6.90% of patients receiving routine thrombectomy compared with 7.00% undergoing PCI alone (hazard ratio = 0.99; 95% CI: 0.85–1.15; $p = 0.86$). The trial therefore demonstrated no significant improvement in the primary clinical outcome with routine thrombectomy. In contrast, the present observational study documented a higher frequency of thrombus aspiration among patients with no-reflow.¹²

The angiographic and myocardial reperfusion outcomes demonstrated substantial differences between the two groups. Among the 18 patients developing no-reflow, 14 (77.78%) showed TIMI flow grade 0–2 following coronary reopening, whereas 4 patients (22.22%) demonstrated TIMI flow grade 3 with myocardial blush grade 0–1. Transient or resolved no-reflow was documented in 55.56% of affected patients, while persistent no-reflow was observed in 44.44%. Final TIMI flow grade 3 was achieved in 61.11% of patients with no-reflow compared with 100.00% of patients in the normal-flow group. Similarly, final myocardial blush grade 2–3 was documented in 55.56% and 100.00% of patients, respectively. ST-segment resolution $\geq 50\%$ occurred significantly less frequently among patients with no-reflow (44.44% versus 76.79%; $p = 0.010$), while mean post-PCI left ventricular ejection fraction was also significantly lower ($42.30 \pm 7.80\%$ versus $47.90 \pm 8.00\%$; $p = 0.011$). Brener et al. (2013), in an analysis of 2,367 patients from the HORIZONS-AMI trial, reported myocardial blush grade 2–3 in 77.70% and ST-segment resolution $\geq 50\%$ in 75.10% of patients following primary PCI. Discordance between myocardial blush grade and ST-segment resolution was identified in 30.90% of patients. Furthermore, satisfactory myocardial blush grade was associated with lower three-year mortality than impaired blush grade (4.40% versus 8.40%; adjusted HR = 0.57; $p = 0.003$).¹³

The present study demonstrated significantly poorer clinical outcomes among patients developing no-reflow following primary PCI. Acute heart failure occurred in 38.89% of patients with no-reflow compared with 10.71% of patients with normal coronary reperfusion ($p = 0.012$). Cardiogenic shock was documented in 33.33% and 7.14% of patients, respectively ($p = 0.011$). Ventricular arrhythmias were also more frequent in the no-reflow group (27.78% versus 8.93%), although the difference was statistically insignificant ($p = 0.056$). In-hospital mortality was 11.11% in the no-reflow group compared with 1.79% in the normal-flow group ($p = 0.145$). The mean hospitalization period was significantly longer in patients with no-reflow (6.80 ± 2.20 versus 4.60 ± 1.60 days; $p < 0.001$). During 30-day follow-up, all-cause mortality was 16.67% among patients with no-reflow compared with 1.79% among those with normal flow ($p = 0.042$), while composite major adverse cardiovascular events occurred in 38.89% and 7.14%, respectively ($p = 0.003$). Similar adverse prognostic findings were reported by Choo et al. (2014), who evaluated 2,017 STEMI patients undergoing primary PCI and identified no-reflow in 262 patients (13.00%). During a median follow-up of 4.1 years, mortality was significantly higher among patients with no-reflow compared with those with normal reperfusion (30.20% versus 18.30%; $p < 0.001$). The association was particularly pronounced during the first 30 days, with an adjusted hazard ratio for mortality of 3.11 (95% CI: 1.91–5.05; $p < 0.001$).¹⁴

CONCLUSION

The present study demonstrated that the no-reflow phenomenon occurred in 24.32% of patients with STEMI undergoing primary PCI. Advanced age, diabetes mellitus, hypertension, prolonged symptom-to-balloon time, elevated neutrophil-to-lymphocyte ratio, and high thrombus burden were significantly associated with no-reflow. Patients developing no-reflow exhibited impaired myocardial reperfusion, reduced left ventricular ejection fraction,

and increased cardiovascular complications. Higher rates of acute heart failure, cardiogenic shock, 30-day mortality, and major adverse cardiovascular events were observed in these patients. Early identification of high-risk patients and timely reperfusion may help improve clinical outcomes following primary PCI.

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