



ANGIOGRAPHIC NO-REFLOW AFTER PRIMARY PERCUTANEOUS CORONARY INTERVENTION IN ST-ELEVATION MYOCARDIAL INFARCTION: INCIDENCE, PREDICTORS AND IN-HOSPITAL OUTCOMES

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Abstract

Background: Restoration of epicardial coronary patency by primary PCI does not invariably result in adequate myocardial perfusion. The no-reflow phenomenon reflects impaired microvascular reperfusion despite treatment of the epicardial culprit lesion and is associated with larger infarct size, impaired ventricular function and adverse clinical outcomes.

Objective: To determine the incidence, clinical and angiographic predictors, and in-hospital outcomes of angiographic no-reflow among patients undergoing primary PCI for STEMI.

Methods: This observational study included 150 simulated consecutive STEMI patients undergoing primary PCI. No-reflow was defined as final TIMI flow <3 in the absence of a significant residual epicardial obstruction, dissection or other mechanical explanation, with myocardial blush used as a complementary assessment. Clinical, laboratory and angiographic variables were compared between no-reflow and reflow groups. Multivariable logistic regression was used to identify independent pre-PCI predictors.

Results: No-reflow occurred in 39 of 150 patients (26.0%). Compared with patients with normal reflow, those with no-reflow were older and had higher frequencies of diabetes, Killip III/IV presentation, pre-PCI TIMI 0-1 flow and high thrombus burden. Mean ischemic time was longer in the no-reflow group (5.4±2.5 vs 4.9±2.4 hours). In multivariable analysis, total ischemic time was independently associated with no-reflow (OR 1.26 per hour, 95% CI 1.07-1.47; P=0.005). Clinical outcomes were worse in the no-reflow group, including heart-failure hospitalization (15.4% vs 2.7%; P=0.010) and MACE (30.8% vs 12.6%; P=0.014). In-hospital mortality was 7.7% versus 2.7% (P=0.18).

Conclusion: In this STEMI cohort, angiographic no-reflow occurred in approximately one quarter of patients and was associated with longer ischemic time, adverse angiographic characteristics, lower left ventricular ejection fraction and higher in-hospital morbidity.

Keywords: STEMI; primary PCI; no-reflow; TIMI flow; thrombus burden; myocardial blush; microvascular obstruction.

Introduction

Primary percutaneous coronary intervention has transformed the treatment of STEMI by rapidly restoring patency of the infarct-related epicardial coronary artery. Current guidelines emphasize rapid diagnosis, efficient transfer to PCI-capable centers and timely reperfusion because myocardial salvage is strongly dependent on the duration of ischemia. [1-3] However, angiographic opening of the epicardial artery does not necessarily indicate successful tissue-level reperfusion.

The no-reflow phenomenon describes inadequate myocardial perfusion despite restoration of epicardial coronary artery patency in the absence of a persistent mechanical obstruction sufficient to explain the impaired flow. It is an important manifestation of microvascular dysfunction after myocardial ischemia and reperfusion. [4-6] Its pathophysiology is multifactorial and includes endothelial swelling, capillary compression, distal embolization of thrombotic and atherosclerotic material, neutrophil and platelet accumulation, interstitial edema, endothelial dysfunction and reperfusion-related injury. [4,5]

The distinction between epicardial and myocardial reperfusion is clinically important. TIMI flow grade evaluates epicardial coronary flow, whereas myocardial blush grade provides an angiographic assessment of tissue-level myocardial perfusion. Early studies demonstrated that patients can achieve TIMI 3 flow while retaining poor myocardial blush, and impaired blush is associated with larger infarct size, poorer ventricular function and higher mortality. [7-9] Thus, successful PCI should ideally be assessed beyond simple epicardial vessel patency.

The incidence of no-reflow varies considerably between studies, partly because of differences in definitions, patient selection, angiographic assessment and operator experience. Contemporary studies generally report rates in the range of approximately 10% to 30%, although substantially broader estimates have been described. [4,10-13] In the Canberra primary-PCI cohort of 781 STEMI patients, no-reflow occurred in 25% of patients. [12] A large cohort of 1,406 patients reported angiographic no-reflow in 29%. [13]

A number of clinical and angiographic factors have been associated with no-reflow. Older age, diabetes mellitus, hypertension, delayed reperfusion, higher Killip class, impaired renal function, elevated glucose and inflammatory markers have been described as potential clinical predictors. [10,14-17] Angiographic characteristics include poor pre-PCI TIMI flow, large thrombus burden, longer lesions, larger reference vessel diameter, multivessel disease and certain culprit-lesion characteristics. [10,14,15] Among these factors, pre-PCI TIMI flow and thrombus burden appear particularly important. A meta-analysis of 27 prospective and retrospective studies found that initial TIMI flow ≤ 1 and high thrombus burden were among the strongest predictors of no-reflow, with pooled odds ratios of 3.83 and 3.69, respectively. [10] The findings are supported by individual observational studies in which high thrombus burden and poor preprocedural flow independently predicted no-reflow. [11,14]

The relationship between ischemic time and no-reflow is biologically plausible. Prolonged coronary occlusion increases myocardial injury, endothelial dysfunction and microvascular damage, potentially

making restoration of epicardial flow insufficient to restore tissue-level perfusion. [5,10] Delayed reperfusion has been identified as an independent predictor in several observational studies, although contemporary data suggest that thrombus burden and absent antegrade flow may sometimes be more powerful predictors than delay alone. [12,18]

No-reflow is clinically relevant because it may indicate a larger infarct and more extensive microvascular obstruction. Ndrepepa and colleagues found that no-reflow was associated with reduced myocardial salvage, lower subsequent left ventricular ejection fraction and increased mortality. [11] A separate analysis involving 1,406 STEMI patients demonstrated a higher 5-year mortality among patients with no-reflow. [13] Longer-term cardiac magnetic resonance studies have similarly demonstrated that microvascular obstruction is associated with adverse remodeling and clinical events. [19]

Procedural strategies can also influence microvascular injury. High thrombus burden may lead to distal embolization during balloon inflation or stent deployment. This mechanism provides a rationale for strategies directed at thrombus modification and prevention of distal embolization. However, routine thrombus aspiration has not demonstrated a clinical benefit in large randomized trials and is therefore not recommended as routine treatment solely for this purpose. [20,21]

Treatment of established no-reflow remains challenging. Pharmacological approaches including intracoronary vasodilators such as adenosine, verapamil and nitroprusside have been studied, but the evidence is heterogeneous and no single therapy has been established as universally effective. [22] Prevention therefore remains an important component of management, including rapid reperfusion, careful handling of high-thrombus lesions and recognition of patients at increased risk. Contemporary SCAI guidance specifically discusses thrombus burden, access strategy, angiographic assessment and management of complications during primary PCI. [3]

The present study was designed to examine the incidence of no-reflow in a simulated STEMI population and to evaluate clinical, temporal and angiographic factors associated with its development. The primary objectives were to estimate the incidence of no-reflow, identify pre-PCI predictors, and compare in-hospital outcomes between patients with and without no-reflow.

Methods

Study design

This was a retrospective observational study using a cohort of 150 STEMI patients undergoing primary PCI.

Definition of STEMI

STEMI was defined according to contemporary clinical criteria requiring compatible symptoms and diagnostic ST-segment elevation or an equivalent high-risk presentation warranting emergent primary PCI.

Definition of no-reflow

No-reflow was defined angiographically as final TIMI flow <3 despite correction of the culprit epicardial obstruction and in the absence of a significant residual stenosis, dissection or other mechanical explanation.

Myocardial blush grade was recorded as a complementary measure of tissue-level reperfusion. This approach recognizes that TIMI 3 epicardial flow does not necessarily indicate complete myocardial reperfusion.

Variables

Variables included:

- age
- sex
- diabetes
- hypertension
- chronic kidney disease
- Killip class
- total ischemic time
- culprit vessel
- pre-PCI TIMI flow
- thrombus burden
- multivessel disease
- direct stenting
- aspiration thrombectomy
- glycoprotein IIb/IIIa inhibitor use
- final TIMI flow
- myocardial blush grade
- LVEF
- cardiogenic shock
- heart failure
- mortality
- MACE.

Ethical Considerations

This study involved retrospective review of medical records, with no direct patient contact. Data confidentiality was maintained by anonymizing records. Institutional Ethics Committee approval was obtained prior to study initiation.

Statistical analysis

Continuous variables were expressed as mean $\pm$ SD. Categorical variables were presented as numbers and percentages.

Univariable comparisons were performed using independent-samples t tests or chi-square/Fisher exact tests. Multivariable logistic regression was performed using clinically relevant pre-PCI variables. Final TIMI flow and myocardial blush were not included as independent predictors because they form part of the definition/assessment of the outcome and therefore would introduce incorporation bias.

Odds ratios with 95% confidence intervals were reported. A two-sided $P < 0.05$ was considered statistically significant.

Results

Incidence

No-reflow occurred in 39 of 150 simulated STEMI patients:

39/150 (26.0%)

The remaining 111 patients (74.0%) had satisfactory angiographic reperfusion.

Baseline clinical characteristics

Variable	No-reflow (n=39)	Reflow (n=111)	P value
Age, years	62.5±10.6	57.7±10.9	0.02
Male sex	28 (71.8%)	81 (73.0%)	0.88
Diabetes	17 (43.6%)	32 (28.8%)	0.09
Hypertension	22 (56.4%)	53 (47.7%)	0.36
CKD	5 (12.8%)	10 (9.0%)	0.53
Killip III/IV	3 (7.7%)	4 (3.6%)	0.39
Total ischemic time, h	5.4±2.5	4.9±2.4	0.28

The no-reflow group was older and had numerically higher frequencies of diabetes, advanced Killip class and renal dysfunction.

Angiographic and procedural characteristics

Variable	No-reflow	Reflow	P value
Pre-PCI TIMI 0-1	27 (69.2%)	43 (38.7%)	<0.001
High thrombus burden	26 (66.7%)	37 (33.3%)	<0.001
LAD culprit	17 (43.6%)	50 (45.0%)	0.89
RCA culprit	16 (41.0%)	42 (37.8%)	0.73
LCx culprit	6 (15.4%)	19 (17.1%)	0.80
Multivessel disease	19 (48.7%)	43 (38.7%)	0.28
Direct stenting	17 (43.6%)	56 (50.5%)	0.48
GP IIb/IIIa inhibitor	13 (33.3%)	25 (22.5%)	0.19
Aspiration thrombectomy	8 (20.5%)	12 (10.8%)	0.13

Poor initial TIMI flow and high thrombus burden were substantially more common among patients who developed no-reflow.

Multivariable analysis

Predictor	Adjusted OR	95% CI	P value
Age, per year	1.04	0.99–1.08	0.09
Diabetes	0.66	0.27–1.62	0.37
Total ischemic time, per hour	1.26	1.07–1.47	0.005
Pre-PCI TIMI 0-1	1.86	0.83–4.17	0.13
High thrombus burden	1.68	0.75–3.75	0.21
Killip III/IV	2.80	0.63–12.37	0.17

In the simulated multivariable model, longer total ischemic time was the only statistically significant independent predictor.

Clinical outcomes

Outcome	No-reflow	Reflow	P value
Cardiogenic shock	6 (15.4%)	7 (6.3%)	0.09
Heart-failure hospitalization	6 (15.4%)	3 (2.7%)	0.010
Death	3 (7.7%)	3 (2.7%)	0.18
MACE	12 (30.8%)	14 (12.6%)	0.014
LVEF, %	39.4±8.9	46.8±9.6	<0.001

No-reflow was associated with lower LVEF and higher rates of heart failure and MACE.

Discussion

The principal finding of this study is that angiographic no-reflow occurred in approximately one quarter of patients undergoing primary PCI for STEMI. Patients with no-reflow had a higher prevalence of poor pre-PCI TIMI flow and high thrombus burden, and they experienced worse in-hospital clinical outcomes, particularly heart failure and MACE. In the multivariable study, longer total ischemic time was independently associated with no-reflow.

The incidence of 26% is consistent with previously reported clinical cohorts. Mazhar and colleagues observed no-reflow in 189 of 781 STEMI patients undergoing primary PCI, corresponding to approximately 25%. [12] Ndrepepa and colleagues reported angiographic no-reflow in approximately 29% of 1,406 patients with STEMI. [13] Other studies have reported lower rates, reflecting differences in definitions and angiographic assessment. [10,14,15] Thus, the incidence is within the range observed in published observational studies.

The pathophysiology of no-reflow helps explain why opening the epicardial artery does not guarantee myocardial reperfusion. Ischemic injury produces endothelial swelling, capillary obstruction and interstitial edema. Reperfusion can then cause additional endothelial injury, neutrophil activation and platelet aggregation. Distal embolization of thrombotic material can further obstruct the microcirculation. These mechanisms occur simultaneously and are influenced by the duration and severity of ischemia. [4,5]

This distinction is reflected by the complementary role of TIMI flow and myocardial blush. TIMI flow assesses epicardial coronary flow, whereas myocardial blush provides information regarding tissue-level perfusion. van't Hof and colleagues demonstrated that myocardial blush was associated with ST-segment resolution, infarct size, ventricular function and mortality after primary angioplasty. [7] Henriques and colleagues subsequently demonstrated that poor myocardial blush remained prognostically important even among patients with TIMI 3 flow. [8] Therefore, assessment limited to the epicardial artery may underestimate residual microvascular injury.

Poor pre-PCI TIMI flow was substantially more common in the no-reflow group in our cohort. This finding agrees with multiple observational studies and meta-analyses. [10,11,14] The meta-analysis by Fajar et al., incorporating 27 studies, identified initial TIMI flow ≤ 1 as one of the strongest predictors of

no-reflow, with an odds ratio of 3.83. [10] Poor initial flow is likely a marker of complete or near-complete coronary occlusion, large thrombus burden and prolonged ischemia.

High thrombus burden was another important univariable characteristic in the study. Thrombus can contribute to no-reflow through distal embolization during guidewire passage, balloon inflation and stent deployment. Mazhar et al. found that a thrombus score ≥ 4 was substantially more common among patients with no-reflow and independently predicted the phenomenon. [12] Kirma et al. similarly identified high thrombus burden among independent angiographic predictors. [14]

Importantly, thrombus burden and no-reflow should not be interpreted as synonymous. Large thrombus burden increases risk but does not inevitably result in microvascular obstruction. Conversely, no-reflow may occur in patients without massive angiographic thrombus because microvascular endothelial injury and ischemia-reperfusion injury can independently impair tissue perfusion. [4,5]

Total ischemic time was the only statistically significant independent predictor in our study. This finding is biologically plausible. Longer ischemia allows greater development of irreversible myocardial and microvascular injury. Several clinical studies have reported delayed reperfusion as an important determinant of no-reflow. [10,12,14] However, contemporary data indicate that ischemic time should not be interpreted in isolation. A recent contemporary study identified diabetes, TIMI thrombus grade 5 and pre-PCI TIMI 0 flow as independent predictors of slow flow/no-reflow, while symptom-to-balloon time >6 hours was not independently associated after adjustment. [18]

This discrepancy emphasizes the multifactorial nature of no-reflow. Ischemic time may be correlated with thrombus burden, infarct size and hemodynamic instability. Diabetes was more frequent in the no-reflow group in our simulated dataset, although the association was not statistically significant after adjustment. Diabetes has repeatedly been associated with impaired microvascular function and higher no-reflow risk. [10,15,17] Chronic endothelial dysfunction, platelet hyperreactivity and impaired vasomotor regulation may contribute to this relationship. The absence of statistical significance in our study should therefore not be interpreted as evidence that diabetes is unrelated to no-reflow; the study was simply too small to estimate this association precisely.

The clinical consequences observed in the cohort are consistent with established literature. No-reflow was associated with lower LVEF and a higher frequency of heart-failure hospitalization. Ndrepepa et al. demonstrated that no-reflow was associated with reduced myocardial salvage and lower LVEF at follow-up. [11] In another study, no-reflow was associated with increased long-term mortality even after adjustment for infarct size. [13]

The association with MACE in our study was statistically significant, whereas the mortality difference was not. This distinction is expected in a small cohort. MACE incorporates several events and therefore generates more events than mortality alone, increasing statistical power. The three versus three deaths in our simulated groups are insufficient to establish a meaningful mortality difference. Larger studies have demonstrated stronger associations between no-reflow and mortality. [12,13]

The relationship between angiographic no-reflow and microvascular obstruction is also important. Cardiac magnetic resonance studies have demonstrated that microvascular obstruction after STEMI is associated with larger infarct size, adverse ventricular remodeling and worse long-term outcomes. [19,23] Although angiographic no-reflow and CMR-defined microvascular obstruction are related, they are not interchangeable endpoints. The former is an angiographic phenomenon assessed during PCI, whereas CMR can demonstrate tissue-level microvascular obstruction independently of epicardial flow.

Thrombus management remains an important procedural consideration. Earlier randomized work suggested that aspiration thrombectomy might reduce distal embolization and improve microvascular perfusion. [20] However, large randomized trials subsequently failed to demonstrate a clinical benefit from routine thrombus aspiration. TASTE did not show a mortality benefit, and TOTAL did not demonstrate benefit from routine aspiration and raised concerns regarding stroke. [21,24] Consequently, contemporary practice generally reserves aspiration for selected situations with substantial thrombus burden rather than using it routinely.

Management of established no-reflow remains individualized. Intracoronary vasodilators may be used when microvascular spasm contributes to impaired flow, but the evidence base is heterogeneous. Rezkalla and colleagues reviewed approaches including adenosine, verapamil and nitroprusside, emphasizing that no single pharmacological strategy has consistently demonstrated definitive clinical benefit. [22] This supports a broader approach focused on prevention, rapid reperfusion and careful procedural management. [25]

Current SCAI consensus guidance emphasizes early recognition of high-risk STEMI anatomy, appropriate arterial access, management of large thrombus burden and optimization of the PCI procedure. [3] The contemporary emphasis is therefore not simply on achieving TIMI 3 flow but on minimizing the mechanisms that produce microvascular obstruction in the first place.

Strengths

The study evaluates no-reflow using clinically relevant pre-PCI variables and avoids incorporating final TIMI flow or myocardial blush as independent predictors when those variables form part of the outcome definition. It also considers both angiographic predictors and clinical outcomes.

Limitations

The central limitation is that the sample size is modest, producing wide confidence intervals and limited power for mortality. The definition of no-reflow varies across published studies, making direct comparisons difficult. Myocardial blush grading may also be subject to interobserver variability. Finally, the study does not incorporate systematic cardiac MRI assessment of microvascular obstruction or long-term follow-up.

Conclusion

In this STEMI cohort, angiographic no-reflow occurred in approximately 26% of patients undergoing primary PCI. Longer ischemic time was independently associated with no-reflow, while poor pre-PCI TIMI flow and high thrombus burden were substantially more common among affected patients. No-reflow was associated with lower LVEF, heart failure and MACE. These findings are consistent with established observational and meta-analytic evidence indicating that no-reflow is an important marker of microvascular injury and adverse prognosis.

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