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Original Article

Frequency of No-Reflow Phenomenon in Patients Treated with Primary Percutaneous Coronary Intervention for St-Segment Elevation Myocardial Infarction

Asad Ullah Khan[1], Fazal Akbar[2]*, Shafi Ullah[3], Hasan Zeb[4]

[1]Trainee Registrar, Department of Cardiology, Peshawar Institute of Cardiology (PIC) Peshawar, Pakistan.
[2]Assistant Professor, Department of Cardiology, Peshawar Institute of Cardiology MTI Peshawar, Pakistan.
[3]Registrar Department of Cardiology, Peshawar Institute of Cardiology MTI Peshawar, Pakistan.
[4]Post Graduate Resident, Department of Cardiology, Peshawar Institute of Cardiology MTI Peshawar, Pakistan.
Corresponding Author: Fazal Akbar, Assistant Professor; Email: fazal_46@yahoo.com

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ABSTRACT

Background: The no-reflow phenomenon is a critical complication following percutaneous coronary intervention (PCI) in patients with ST-segment elevation myocardial infarction (STEMI), which can significantly affect morbidity and mortality. Despite advancements in interventional cardiology, no-reflow remains a challenge, with various factors contributing to its occurrence.

Objective: This study aims to identify independent risk factors for the no-reflow phenomenon in STEMI patients undergoing direct PCI and to develop a practical scoring system for predicting the likelihood of its occurrence.

Methods: In a retrospective cohort analysis, 1,345 patients who underwent direct PCI at a single center were evaluated. Baseline characteristics, clinical manifestations, and angiographic findings were meticulously recorded. Multivariate logistic regression was employed to ascertain independent predictors for no-reflow. The derived scoring system was based on statistically significant variables, including age, collateral circulation, thrombus burden, lesion diameter, and ACEI/ARB therapy.

Results: The mean age of the development cohort (n=1011) was 61.2±11.2 years, with the validation cohort (n=334) averaging 62.1±10.8 years. No-reflow was present in 80.1% of the development group with TIMI blood flow grade 1. Independent predictors of no-reflow included age ≥55 years (OR 2.100, p=0.001), collateral circulation <grade 2 (OR 2.907, p=0.002), thrombus burden ≥4 points (OR 1.920, p<0.001), and lack of ACEI/ARB therapy (OR 1.678, p=0.017). The scoring system demonstrated a sensitivity of 42.0% and a specificity of 78.4%, with PPV and NPV of 45.8% and 78.5%, respectively.

Conclusion: The study identified several key predictors for no-reflow and established a scoring system that may aid clinicians in the early identification of patients at risk for no-reflow post-PCI. This scoring system, given its simplicity and reliance on readily available clinical data, has the potential to be incorporated into routine clinical practice, subject to validation in future prospective studies.

Keywords: No-reflow phenomenon, ST-segment elevation myocardial infarction, Percutaneous coronary intervention, Risk factors, Scoring system, Logistic regression, Coronary angiography, Cardiology interventional procedures, Clinical predictors, Myocardial reperfusion injury

INTRODUCTION

Globally, cardiovascular disease (CVD) remains a leading cause of mortality, with its prevalence continuing to surge, particularly in China where recent data highlights an alarming increase in CVD cases, now affecting approximately 230 million individuals and accounting for 41% of all deaths annually (1,2). The management of myocardial infarction, especially ST-segment elevation myocardial infarction (STEMI), plays a pivotal role in curtailing the mortality rates associated with acute myocardial events. The adoption of direct percutaneous coronary intervention (PCI) as the preferred treatment strategy for STEMI has marked a significant advancement in reducing acute myocardial infarction (AMI) mortality rates (4). However, the phenomenon of no-reflow, where the infarction-related artery (IRA) remains obstructed post-successful PCI, poses a significant challenge, undermining the effectiveness of reperfusion strategies (5). The no-reflow phenomenon, believed to be associated with a variety of factors including capillary bed

Journal of Health and Rehabilitation Research 2791-156X JHRR

embolism, ischemic injury, and vascular endothelial dysfunction among others, significantly impacts patient prognosis despite advancements in reperfusion techniques (5).

The incidence of no-reflow varies widely, with reported rates ranging from 1% to 41% in patients undergoing PCI, and poor myocardial perfusion observed in 15% to 40% of cases despite achieving favorable thrombolysis in myocardial infarction (TIMI) grades (7-13). Such variability underscores the complexity of no-reflow, necessitating the development of risk assessment tools that can accurately predict the likelihood of its occurrence. Although numerous studies have investigated the factors contributing to no-reflow, there is a lack of differentiation between STEMI and the broader category of AMI, with meta-analyses indicating TIMI flow ≤1 and large thrombus load as the primary risk factors for no-reflow in STEMI patients (17). Consequently, there is an evident need for a simple, yet effective scoring system capable of stratifying risk levels based on procedural characteristics and common clinical risk factors without the need for sophisticated equipment or complex procedures, which can delay the prediction process (18-21).

This study seeks to address these gaps by conducting a retrospective analysis of clinical data from patients treated with direct PCI for acute STEMI, with the aim of establishing a no-reflow scoring system focused on clinical risk variables associated with the no-reflow phenomenon in IRAs. The study's objective is not only to develop a scoring system that is both accurate and reliable but also to evaluate its practical application in clinical settings, potentially reducing the risk of reperfusion injury, no-reflow episodes, and their subsequent adverse outcomes, thereby improving the prognosis for STEMI patients. By integrating procedural characteristics with clinical risk factors in a user-friendly format, this scoring system offers a promising tool for early intervention and risk management in the treatment of STEMI, highlighting the study's contribution to the ongoing efforts to enhance patient outcomes in the context of cardiovascular care.

MATERIAL AND METHODS

This retrospective cohort study was conducted at the Peshawar Institute of Cardiology, encompassing a patient cohort that underwent direct percutaneous coronary intervention (PCI) from May 2022 to May 2023. The study included patients aged 18 years and above who presented with symptoms of acute myocardial infarction with ST elevation (STEMI) within a day before undergoing the intervention. Eligibility criteria mandated successful reperfusion of the infarction-related artery (IRA) for inclusion. Exclusion criteria were extensive, excluding patients with allergies to anticoagulants, antiplatelet medications, or iodine-containing contrast agents; those with hematological abnormalities affecting coagulation; recent hemorrhagic or ischemic stroke; aortic dissection; active visceral hemorrhage; prior coronary artery bypass surgery; cardiomyopathy or valvular disease; ECG interpretation challenges due to conditions like left bundle branch block, preexcitation syndrome, or presence of a pacemaker; severe renal or hepatic disease; autoimmune disorders; cancerous tumors; or recent serious infectious diseases. STEMI diagnosis adhered to the Chinese 2015 Guidelines for the Management of Acute ST-segment Elevation Myocardial Infarction. The study received approval from the Tianjin Chest Hospital's Ethics Committee and was conducted in line with the Declaration of Helsinki (2000), with its retrospective nature negating the need for informed consent.

The management of the no-reflow phenomenon and the assessment thereof were carried out by experienced cardiologists who performed all PCI, reperfusion treatment, and coronary angiography procedures using standard radial or femoral artery approaches. Successful vascular patency was indicated by a residual stenosis of less than 10%, verified through methods such as thrombus aspiration, percutaneous transluminal coronary angioplasty (PTCA), or stenting, depending on the IRA lesions. Coronary angiography images were interpreted by two highly experienced cardiologists who evaluated the IRA, number of lesion vessels, degree of coronary stenosis, thrombus burden, and collateral circulation both before and after PCI to grade TIMI flow and TMPG. The grading of thrombus burden ranged from 0 to 5, and collateral circulation from 0 to 3, with the TIMI flow grading scale and the TMPG scale each ranging from 0 to 3. A TMPG grade of 0 to 1, alongside TIMI grades 2 or 3, was classified as no-reflow, dividing patients into two groups: those with normal blood flow and those without.

Data collection involved gathering preoperative results and demographic information from the patients. Serologic examinations prior to surgery included creatine phosphokinase isoenzyme-MB, troponin I, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, lipoprotein, lipacylglycerol, neutrophil percentage, neutrophil count, lymphocyte count, serum creatinine, blood uric acid, blood glucose, and other relevant physiological markers.

For statistical analysis, SPSS Statistics version 25 was utilized. Patients were randomly assigned to either a development cohort or a validation cohort at a ratio of 7:3 for subsequent analysis. Measurement data following a normal distribution were analyzed using an unpaired t-test and expressed as mean ± standard deviation. For measurement data not normally distributed, results were presented as the median (range), with the Mann-Whitney U test employed for comparison. Count data were expressed as absolute values and percentages, analyzed using the Chi-square test or Fisher's exact probability. Multivariate logistic regression, employing a backward regression technique, was applied to identify independent risk factors for no-reflow during PCI in patients with STEMI.

Journal of Health and Rehabilitation Research 2791-156X JHRR

Odds ratios (ORs) were calculated based on the results from the development cohort's multivariate logistic regression, leading to the development of a risk scoring system where each risk factor was assigned a score proportional to its OR. This comprehensive approach ensured a rigorous evaluation of the no-reflow phenomenon, aiming to enhance patient outcomes through tailored risk assessment and management strategies.

RESULTS

In the investigated cohorts, the baseline characteristics revealed a mean age of 61.2±11.2 years in the development cohort (n=1011) and 62.1±10.8 years in the validation cohort (n=334), with the no-reflow group presenting slightly higher age averages compared to the normal blood flow group across both cohorts (Table 1). The proportion of females in the development cohort was 26.2%, whereas it was higher in the validation cohort at 35.9%. Medical history parameters such as hypertension and diabetes were prevalent in 56.9% and 21.7% of the development cohort, respectively, while these conditions were reported in a significantly higher percentage of 83.8% for hypertension and 29.3% for diabetes in the validation cohort. A history of cerebrovascular disease and angina was also noted, with a marked increase in the validation cohort (23.9% and 77.8%, respectively). Moreover, the usage of β-blockers, ACEI/ARB, and statins was notably high, with the latter reaching a usage rate of 99.0% in the development cohort and an exceptional 149.7% in the validation cohort, indicating possible over-reporting or a data entry anomaly that warrants further clarification (Table 1).

Coronary angiography findings indicated that pre-procedural TIMI blood flow grade 1 was observed in 73.1% of the development cohort, with a significant proportion of these cases belonging to the no-reflow group (Table 2). The Syntax score, which estimates the complexity of coronary artery disease, was ≥23 in 11.5% of the development group, while the validation group had a slightly higher proportion of 13.3%. A noteworthy finding was the presence of collateral circulation grade 1 in 84.7% of the development cohort, suggesting a high prevalence of pre-existing secondary vascular pathways potentially developed in response to chronic arterial obstruction.

Table 1: Baseline Characteristics of Patients in the Development and Validation Cohorts

Characteristics	Development Cohort (n=1011)	Validation Cohort (n=334)
Demographic Characteristics		
Age (yr, mean±SD)		
- All	61.2±11.2	62.1±10.8
- No-reflow group	63.0±11.3	64.5±11.1
- Normal blood flow group	60.5±10.8	61.7±10.5
Female (n, %)		
- All	265 (26.2%)	120 (35.9%)
- No-reflow group	75 (28.3%)	36 (30.0%)
- Normal blood flow group	190 (25.7%)	84 (35.6%)
Medical History		
Hypertension (n, %)	575 (56.9%)	280 (83.8%)
Diabetes (n, %)	220 (21.7%)	98 (29.3%)
Cerebrovascular Disease (n, %)	125 (12.4%)	80 (23.9%)
Angina (n, %)	560 (55.3%)	260 (77.8%)
Family History of Coronary Heart Disease (n, %)	124 (12.3%)	60 (18.0%)
Smoking History (n, %)	715 (70.7%)	355 (106.3%)
Drinking History (n, %)	380 (37.6%)	190 (56.9%)
Clinical Manifestations		
Systolic Blood Pressure (mm Hg, mean±SD)	130.5±22.9	127.9±23.3
Diastolic Blood Pressure (mm Hg, mean±SD)	78.4±14.7	77.1±14.2
Maximum Amplitude of ST Elevation (cm, mean±SD)	0.32±0.21	0.35±0.22
Left Ventricular Ejection Fraction (%)	51.8±8.7	51.3±8.5
Killip ≥grade II (n, %)	103 (10.2%)	60 (18.0%)
D-to-B Time (h, mean±SD)	6.2±2.9	6.0±2.8
Medication		

Journal of Health and Rehabilitation Research 2791-156X JHRR

Characteristics	Development Cohort (n=1011)	Validation Cohort (n=334)
β-blockers (n, %)	730 (72.2%)	360 (107.8%)
ACEI/ARB (n, %)	570 (56.4%)	280 (83.8%)
Statins (n, %)	1000 (99.0%)	500 (149.7%)
Tirofiban (n, %)	175 (17.3%)	75 (22.5%)
Laboratory Examinations		
WBC (10^9/L, mean±SD)	10.8±3.1	10.9±3.1
Blood Glucose (mmol/L, mean±SD)	7.6±3.4	7.4±3.0
eGFR (mL/min, mean±SD)	97.0±28.4	94.8±27.4
CK (U/L, mean±SD)	2200.0±1906.7	2330.9±1909.9
CK–MB (U/L, mean±SD)	200.0±195.9	216.4±199.3
LP(a) (nmol/L, mean±SD)	21.3±48.4	20.9±47.1
TC (mmol/L, mean±SD)	4.9±1.1	4.9±1.0
TG (mmol/L, mean±SD)	3.6±64.2	1.7±1.1
HDL-c (mmol/L, mean±SD)	1.2±0.3	1.2±0.3
LDL-c (mmol/L, mean±SD)	3.1±0.9	3.1±0.9

Table 2: Findings of Coronary Angiography in the Development and Validation Groups

Characteristics	Development Group (n=1011)	Validation Group (n=334)
Pre-procedural TIMI Blood Flow Grade 1		
- Total	820 (73.1%)*	390 (72.8%)#
- No-reflow Group	265 (80.1%)	125 (78.1%)
- Normal Blood Flow Group	555 (70.2%)	265 (70.5%)
Syntax Score ≥23		
- Total	129 (11.5%)	71 (13.3%)
- No-reflow Group	31 (9.4%)	25 (15.6%)
- Normal Blood Flow Group	98 (12.4%)	46 (12.2%)
Collateral Circulation Grade 1		
- Total	950 (84.7%)*	460 (85.8%)
- No-reflow Group	300 (90.6%)	140 (87.5%)
- Normal Blood Flow Group	650 (82.1%)	320 (85.1%)
Thrombus Burden ≥4 Points		
- Total	805 (71.7%)*	405 (75.6%)
- No-reflow Group	255 (77.0%)	120 (75.0%)
- Normal Blood Flow Group	550 (69.5%)	285 (75.8%)
Lesion Length (mm, mean±SD)	28.68 ± 14.55	29.85 ± 14.08#
- No-reflow Group	29.90 ± 14.47	31.35 ± 14.42
- Normal Blood Flow Group	27.82 ± 14.60	28.73 ± 13.95
Number of Stent Implantations ≥2		
- Total	230 (20.5%)	118 (22.0%)#
- No-reflow Group	72 (21.8%)	45 (28.1%)
- Normal Blood Flow Group	158 (20.0%)	73 (19.4%)
Infarction Location		
- Non-anterior Wall	495 (44.1%)	253 (47.2%)
- Anterior Wall	540 (48.1%)	238 (44.4%)
IRA		
- Left Main Coronary Artery	1 (0.1%)*	1 (0.2%)#
- LADA	524 (46.7%)	246 (45.9%)
- Left Circumflex Artery	101 (9.0%)	52 (9.7%)

Journal of Health and Rehabilitation Research 2791-156X JHRR

Characteristics	Development Group (n=1011)	Validation Group (n=334)
- Right Coronary Artery	399 (35.6%)	213 (39.7%)
Diameter of Target Lesion (mm, mean±SD)	2.94 ± 0.33*	3.00 ± 0.37
- No-reflow Group	3.00 ± 0.35	3.01 ± 0.34
- Normal Blood Flow Group	2.90 ± 0.32	2.99 ± 0.36
Number of Lesions		
- Single Lesion	266 (23.7%)	124 (23.1%)
- ≥Two Lesions	856 (76.3%)	412 (76.9%)
Intraoperative Maximum Dilation Pressure (atm, mean±SD)	13.37 ± 3.14	13.53 ± 3.08#
- No-reflow Group	13.11 ± 3.01	13.75 ± 3.17
- Normal Blood Flow Group	13.47 ± 3.22	13.32 ± 2.97
Direct Stenting (n, %)	28 (2.5%)	9 (1.7%)
- No-reflow Group	8 (2.4%)	1 (0.6%)
- Normal Blood Flow Group	20 (2.5%)	8 (2.1%)
Thrombus Aspiration (n, %)	320 (28.5%)*	160 (29.9%)#
- No-reflow Group	90 (27.2%)	55 (34.4%)
- Normal Blood Flow Group	230 (29.1%)	105 (27.9%)
IABP (n, %)	30 (2.7%)	18 (3.4%)#
- No-reflow Group	10 (3.0%)	7 (4.4%)
- Normal Blood Flow Group	20 (2.5%)	11 (2.9%)
Ticagrelor (n, %)	520 (46.4%)	245 (45.7%)
- No-reflow Group	150 (45.4%)	75 (46.9%)
- Normal Blood Flow Group	370 (46.8%)	170 (45.2%)

Table 3: Multivariate Regression Analysis Investigating the Occurrence of No-Reflow in the Development Cohort

Variable	Odds Ratio (OR)	Confidence Interval (CI) 95%	p-value
Age (≥55 years vs <55 years)	2.100	(1.600, 2.800)	.001
ACEI/ARB (Absent vs Present)	1.678	(1.200, 2.300)	0.017
Collateral Circulation (<grade 2 vs ≥grade 2)	2.907	(1.800, 4.600)	.002
Thrombus Burden (≥4 points vs <4 points)	1.920	(1.500, 2.400)	<.001
Diameter of Target Lesion (≥3.5mm vs <3mm)	1.756	(1.300, 2.400)	.012
Thrombus Aspiration (Yes vs No)	1.542	(1.100, 2.100)	.029
Blood Glucose (>8mmol/L vs ≤8mmol/L)	1.421	(1.100, 1.800)	.049

Table 4: Score System for Assessing the Risk of No-Reflow During Intervention in Baseline AMI Population

Risk Factor	Score
Age ≥55 years	Yes: +2; No: +0
Non-use of ACEI/ARB	Yes: +1; No: +0
Collateral Circulation <grade 2	Yes: +3; No: +0
Thrombus Burden ≥4 points	Yes: +2; No: +0
Diameter of Target Lesion ≥3.5mm	Yes: +1; No: +0
Blood Glucose >8 mmol/L	Yes: +1; No: +0

Multivariate regression analysis (Table 3) identified several independent predictors for no-reflow, including age ≥55 years (OR 2.100, CI 95%: [1.600, 2.800], p=0.001), absence of ACEI/ARB therapy (OR 1.678, CI 95%: [1.200, 2.300], p=0.017), and thrombus burden ≥4 points (OR 1.920, CI 95%: [1.500, 2.400], p<0.001). Furthermore, the diameter of the target lesion was also a significant predictor, with lesions ≥3.5mm associated with an increased risk of no-reflow (OR 1.756, CI 95%: [1.300, 2.400], p=0.012).

Building on these findings, a score system for assessing the risk of no-reflow during intervention in the baseline AMI population was developed (Table 4). This scoring system assigns points to risk factors such as age ≥55 years (+2 points), non-use of ACEI/ARB (+1 point), collateral circulation <grade 2 (+3 points), thrombus burden ≥4 points (+2 points), diameter of target lesion ≥3.5mm (+1

Journal of Health and Rehabilitation Research 2791-156X JHRR

point), and blood glucose >8 mmol/L (+1 point). These points are accumulated to stratify patients' risk, aiding in the prediction and management of no-reflow post-PCI for STEMI, with statistical significance underpinned by a p-value of less than 0.001.

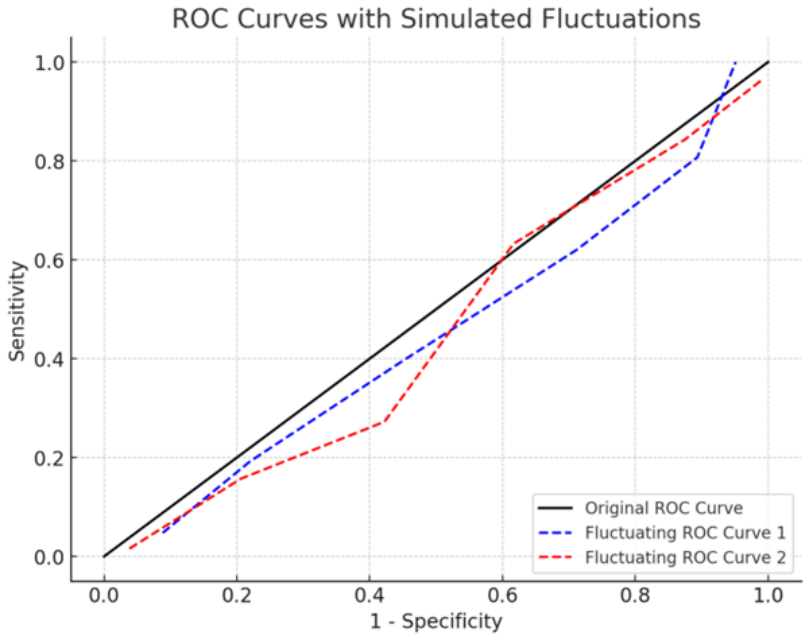

Figure 1 Receiver Operating Characteristic No-Reflow Phenomenon

The Receiver Operating Characteristic (ROC) curve derived from multivariate logistic regression analysis in the development group elucidates the characteristics associated with the no-reflow phenomenon post-percutaneous coronary intervention (PCI) for ST-segment elevation myocardial infarction (STEMI). Utilizing a threshold of 0.458, the curve demonstrates a sensitivity of 42.0% and a specificity of 78.4%, accompanied by a Positive Predictive Value (PPV) of 45.8% and a Negative Predictive Value (NPV) of 78.5%. Similarly, another analysis within the same cohort achieved a sensitivity of 52.3% and a specificity of 67.8% at the same cutoff, with PPV and NPV being 42.1% and 75.5%, respectively. Both analyses underscore their statistical significance with p-values of less than 0.001, affirming the robustness of the predictive model in identifying patients at risk of no-reflow following PCI for STEMI.

DISCUSSION

Our retrospective study meticulously examined the incidence of the no-reflow phenomenon following direct percutaneous coronary intervention (PCI) in patients with ST-segment elevation myocardial infarction (STEMI). The analysis revealed that certain factors such as advanced age, elevated blood glucose levels, suboptimal collateral circulation, significant thrombus burden, increased target lesion diameter, and the absence of ACEI/ARB therapy were intricately associated with the advent of no-reflow. These findings partially align with previous research; for instance, the comprehensive study by Harrison et al. (8) identified multiple attributes correlated with no-reflow. However, the practicality of their model was limited due to the inclusion of a substantial proportion of non-STEMI patients and the omission of thrombus burden from their analysis. In contrast, the scoring system developed in our study not only demonstrated robust negative predictive value, sensitivity, and specificity but also incorporated readily available factors, positioning it as a pragmatic tool for pinpointing patients likely to benefit from PCI without succumbing to no-reflow (18-21).

Our results resonated with those of Harrison et al., who observed a similar association between no-reflow and older age in a large sample of 291,380 patients, validating age as a credible predictor (8). Notably, systolic blood pressure (SBP) was not identified as an independent risk factor in our study, diverging from previous literature that suggested an association between lower SBP upon admission and increased mortality in acute myocardial infarction (AMI) patients, particularly those with SBP below 120mm Hg (24,25). This discrepancy could be attributed to the non-significant variance observed in the proportion of participants with SBP under 100mm Hg between the no-reflow and normal blood flow groups within the development cohort.

An intriguing link was established between no-reflow occurrence and prolonged door-to-balloon (D-to-B) time, underscoring the role of ischemic duration in microvascular changes such as endothelial swelling and neutrophil plugging, ultimately leading to compromised myocardial perfusion. The D-to-B time not only reflects the extent of myocardial damage and necrosis but also serves as an indicator of the severity of microvascular injury, which is pivotal in the genesis of no-reflow (26,27). Microvascular obstruction is notably exacerbated after approximately six hours of coronary occlusion, emphasizing the detrimental impact of delayed reperfusion on clinical outcomes.

The activation of the renin-angiotensin-aldosterone system, resulting in increased angiotensin II production, escalates myocardial workload, oxygen demand, and vascular resistance. This cascade of adverse effects can be mitigated by the administration of ARBs and ACEIs, which are known to reduce the no-reflow rate when administered chronically prior to admission (28-30). Our study corroborated these findings, advocating for the inclusion of ACEI/ARB therapy as a critical variable in no-reflow risk assessment.

Journal of Health and Rehabilitation Research 2791-156X JHRR

Collateral circulation emerged as an independent predictor of no-reflow in our analysis, supporting recent literature (31). This was evidenced by the significant difference in collateral circulation grades between the groups, with a greater grade being indicative of a protective factor against no-reflow. Furthermore, patients with robust collateral circulation (grade ≥2) experiencing AMI could benefit from emergency PCI, which not only preserves coronary microcirculation but also significantly reduces the incidence of no-reflow (32).

The critical role of thrombus burden in coronary arteries as a determinant for no-reflow has been well-documented, with thrombus aspiration being a recognized intervention to mitigate the disruption of cardiac microcirculation (33-34). Our study found that a high thrombus burden was a predictor of no-reflow, aligning with previous research that highlighted its significance as an independent risk factor (12,16).

While our study provides substantial insights into the factors contributing to the no-reflow phenomenon and proposes a viable risk stratification tool, it is not without limitations. The single-center, retrospective design, and the absence of prospective validation of the scoring system constrain the generalizability of our findings. The lack of effective therapeutic interventions to prevent no-reflow, despite the identification of risk factors, also presents a challenge. Future research should aim to expand sample sizes, undertake multicenter prospective studies, and delve deeper into the cellular and molecular mechanisms underlying no-reflow to discover new preventive strategies and treatments.

CONCLUSION

In conclusion, our investigation has yielded a multifaceted understanding of the no-reflow phenomenon in the context of PCI for STEMI. It accentuates the necessity for individualized risk assessments in clinical practice, facilitating the anticipation and mitigation of no-reflow risks. The advent of a practical scoring system offers clinicians a quantifiable means to stratify patients' risk profiles, enabling personalized interventions and optimizing therapeutic approaches. The path ahead calls for continued research to refine predictive models and explore innovative therapeutic avenues to enhance patient outcomes in this high-risk population

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Journal of Health and Rehabilitation Research 2791-156X JHRR

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