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Investigating the Relationship Between No-Reflow and Slow-Flow Occurrence with the Involved Vessel and Shock Index

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Abstract

Background: Impaired myocardial reperfusion (no-reflow or slow-flow) after primary percutaneous coronary intervention (PPCI) is an adverse complication in patients with ST-segment elevation myocardial infarction (STEMI), particularly in those with left anterior descending artery (LAD) involvement. The shock index (SI) may aid early risk stratification in these patients.

Objectives: To investigate the association between admission SI and the occurrence of impaired coronary flow in patients with STEMI undergoing PPCI, with consideration of infarct-related artery involvement.

Methods: In this single-center observational study, 150 consecutive patients with acute STEMI treated with PPCI were enrolled. The admission SI was calculated as heart rate divided by systolic blood pressure at presentation. Coronary flow after PPCI was assessed using the Thrombolysis in Myocardial Infarction flow grade and myocardial blush grade. Slow-flow and no-reflow were analyzed as a combined impaired coronary flow endpoint. Admission SI was compared between patients with normal and impaired flow using the Mann–Whitney U test, and the association between the infarct-related artery and impaired flow was evaluated using the chi-square test.

Results: Of the 150 patients, impaired coronary flow (slow-flow or no-reflow) occurred in 42 (28.0%), and normal flow was achieved in 108 (72.0%). Patients with impaired flow had a higher admission SI than those with normal flow (0.65 ± 0.09 vs 0.60 ± 0.05; P = 0.005). Impaired flow was more frequent when the LAD was the infarct-related artery (21 of 46, 45.7%) than when the left circumflex artery (LCX) (10 of 55, 18.2%) or the right coronary artery (11 of 49, 22.4%) was involved (P = 0.005).

Conclusions: In this single-center study, a higher admission shock index and LAD involvement were associated with impaired coronary flow after PPCI in patients with STEMI. These unadjusted findings suggest that the shock index may be a simple bedside marker related to impaired reperfusion; however, its predictive value and independence from other risk factors require confirmation in larger studies.

Keywords: ST Elevation Myocardial Infarction, No-Reflow Phenomenon, Coronary Artery Disease

1. Background

Acute ST-segment elevation myocardial infarction (STEMI) is one of the most critical cardiovascular emergencies and remains a leading cause of mortality and long-term disability worldwide (1, 2). In appropriately equipped centers, the treatment of choice for STEMI is primary percutaneous coronary intervention (PCI), which aims to achieve rapid reperfusion of the occluded coronary artery and restore epicardial blood flow to Thrombolysis in Myocardial Infarction (TIMI) grade 3 (2). However, despite successful recanalization of the epicardial vessel, adequate myocardial perfusion at the microvascular level is not achieved in a subset of patients. This phenomenon is referred to as no-reflow or, in less severe cases, slow-flow (3).

The no-reflow phenomenon is classically defined as TIMI flow grade ≤ 2 in the absence of mechanical obstruction, whereas slow-flow refers to TIMI grade 3 flow accompanied by a reduced myocardial blush grade (MBG) of ≤ 2 (4). The reported incidence of these phenomena varies across studies, ranging from approximately 10% to

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30% (4-6). Their pathophysiology is multifactorial and includes distal microembolization of thrombotic or atherosclerotic debris, microvascular spasm, endothelial dysfunction, inflammatory responses, and tissue edema resulting from ischemia–reperfusion injury (7).

No-reflow and slow-flow are associated with several adverse clinical outcomes, including larger infarct size, impaired left ventricular function, increased incidence of ventricular arrhythmias, and higher short- and long-term mortality rates (8, 9). Given the limited efficacy of therapeutic interventions once these phenomena have developed, early risk stratification and identification of high-risk patients before or during PCI are of paramount importance.

Both hemodynamic status at presentation and angiographic characteristics have been implicated in the development of impaired coronary flow. The shock index (SI), calculated as heart rate divided by systolic blood pressure, is a simple bedside marker of circulatory status and sympathetic activation (10, 11). In patients with STEMI, an elevated SI has been associated with worse clinical outcomes, including cardiogenic shock and mortality (12, 13). Emerging evidence suggests that SI may also be related to microvascular reperfusion failure (14, 15).

In addition, the location of the infarct-related artery may influence the risk of impaired myocardial reperfusion. The left anterior descending artery (LAD) supplies the largest myocardial territory, including the anterior wall, interventricular septum, and left ventricular apex (16). Owing to the extensive area at risk, LAD involvement may be associated with larger infarct size (17, 18), greater thrombus burden, and more severe microvascular injury, which could predispose patients to no-reflow or slow-flow. Some observational studies have identified LAD culprit location as a predictor of impaired coronary flow (6, 8, 19).

2. Objectives

Accordingly, this study aimed to evaluate the relationship of admission shock index and infarct-related vessel involvement with the occurrence of no-reflow and slow-flow in patients with STEMI undergoing primary percutaneous coronary intervention (PPCI).

3. Methods

3.1. Study Design and Population

This single-center, prospective, observational study was conducted at Modarres Hospital, Tehran, Iran, a tertiary referral cardiovascular center. The study population consisted of patients diagnosed with acute STEMI who presented to the emergency department and underwent PPCI in 2025. A total of 150 consecutive patients who met the eligibility criteria were included in the final analysis.

Patients aged 18 years or older who were diagnosed with STEMI based on clinical presentation and electrocardiographic criteria and were treated with PPCI were eligible for inclusion. Patients were excluded if they had incomplete admission vital sign data (heart rate or systolic blood pressure), had received thrombolytic therapy before PCI, had a history of coronary artery bypass graft surgery that interfered with angiographic assessment, or presented with severe cardiogenic shock requiring immediate advanced circulatory support.

The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

3.2. Data Collection

Baseline demographic characteristics, cardiovascular risk factors, and clinical data were extracted from medical records. Admission vital signs were recorded before PPCI. The shock index (SI) was calculated as the ratio of heart rate (beats per minute) to systolic blood pressure (mm Hg) measured at presentation.

Coronary angiographic data were reviewed to identify the infarct-related artery, categorized as the LAD, left circumflex artery (LCX), or right coronary artery (RCA). Post-intervention coronary flow was assessed using the TIMI flow grading system and the MBG.

Normal coronary flow was defined as TIMI grade 3 with adequate myocardial blush. Slow-flow was defined as TIMI grade 3 with reduced myocardial blush (MBG ≤ 2), whereas no-reflow was defined as TIMI flow grade ≤ 2 in the absence of mechanical obstruction. For analytical purposes, patients with slow-flow and those with no-reflow were combined into a single impaired coronary flow group, and the two were not analyzed separately.

3.3. Statistical Analysis

Statistical analyses were performed using SPSS software version 24.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation, and categorical variables as frequencies and percentages. Admission shock index was compared between patients with normal and impaired coronary flow using the Mann–Whitney U test because of its non-normal distribution (mean ± SD values are shown for descriptive purposes). The association between the infarct-related artery and impaired coronary flow was evaluated using the chi-square test. All analyses were unadjusted. A P value of less than 0.05 was considered statistically significant.

4. Results

4.1. Baseline Demographic and Clinical Characteristics

A total of 150 patients with ST-segment elevation myocardial infarction who underwent primary percutaneous coronary intervention were included in the analysis. The study population was predominantly male (78.7%), with a mean age of 58.23 ± 12.09 years. Cardiovascular risk factors were common and included hypertension in 42% of patients, diabetes mellitus in 25.3%, hyperlipidemia in 35.3%, and active smoking in 37.3%. Chronic kidney disease was present in 4.7% of the cohort. The mean shock index at admission was 0.62 ± 0.07. Detailed baseline characteristics are presented in Table 1.

Table 1. Demographic and Clinical Characteristics of the Study Population [a]

Variables	Values
Gender (male)	118 (78.7)
Diabetes (Yes)	38 (25.3)
Hypertension (Yes)	63 (42)
Hyperlipidemia (Yes)	53 (35.3)
Smoking (Yes)	56 (37.3)
Chronic kidney disease (Yes)	7 (4.7)
Coronary flow	
Normal flow	108 (72.0)
Impaired flow (slow-flow or no-reflow)	42 (28.0)
Infarct-related artery	
LCX	55 (36.7)
RCA	49 (32.7)
LAD	46 (30.7)
Age, y	58.23 ± 12.09
Shock index	0.62 ± 0.07

[a] Values are expressed as No. (%) or mean ± SD. Impaired flow = slow-flow or no-reflow. Abbreviations: LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; SD, standard deviation.

Following PCI, normal coronary flow was achieved in 108 patients (72.0%), whereas impaired flow (slow-flow or no-reflow) occurred in 42 patients (28.0%). The infarct-related artery was the left circumflex artery (LCX) in 55 patients (36.7%), the RCA in 49 (32.7%), and the LAD in 46 (30.7%).

4.2. Association Between Infarct-Related Artery and Slow-Flow/No-Reflow

The infarct-related artery was significantly associated with impaired coronary flow (P = 0.005, chi-square test). Impaired flow occurred in 21 of 46 patients (45.7%) with LAD involvement, compared with 10 of 55 (18.2%) with LCX involvement and 11 of 49 (22.4%) with RCA involvement. Normal flow was more common in patients with LCX and RCA lesions (Table 2).

4.3. Association Between Shock Index and Slow-Flow/No-Reflow

Patients with impaired coronary flow had higher admission shock index values than those with normal flow (0.65 ± 0.09 vs 0.60 ± 0.05; P = 0.005, Mann–Whitney U test). This finding suggests an association between higher SI and impaired coronary flow, although the absolute difference was small (0.05). This comparison is shown in Table 3.

5. Discussion

In this single-center observational study of patients with STEMI undergoing PPCI, we evaluated the association of admission SI and the infarct-related artery (LAD versus non-LAD) with impaired myocardial reperfusion. Patients who developed slow-flow or no-reflow had higher SI values at presentation, and LAD-related infarctions were associated with a higher frequency of impaired coronary flow than non-LAD culprit vessels. These unadjusted findings suggest that both hemodynamic status at presentation and infarct location may be related to postprocedural microvascular dysfunction, but they do not establish either factor as an independent predictor.

The clinical significance of no-reflow and slow-flow is well established. Numerous studies have demonstrated that impaired myocardial reperfusion following PPCI is associated with larger infarct size, reduced left ventricular systolic function, higher rates of malignant ventricular arrhythmias and heart failure, and increased short- and long-term mortality (20-22). Importantly, these adverse outcomes occur even when angiographic epicardial patency is restored, highlighting the critical role of the microcirculation in determining myocardial salvage and prognosis. Therefore, identifying patients at high risk of no-reflow or slow-flow before or during PCI remains a major unmet clinical need.

The mean SI in our cohort (0.62 ± 0.07) was within the range of approximately 0.5 – 0.7 generally considered normal for adults (11), which is expected because patients in severe cardiogenic shock were excluded. Patients who developed slow-flow or no-reflow had higher SI values than those with normal flow, but the absolute difference was small (0.65 vs. 0.60). Whether such a modest difference has clinical value cannot be determined from our data, because we did not assess cutoff values, predictive accuracy, or clinical outcomes.

Our findings are consistent with previous reports on the relationship between SI and impaired coronary flow.

Table 2. Association Between the Involved Coronary Artery and the Occurrence of Slow-Flow/No-Reflow [a]

Variables	Normal Flow	Impaired Flow (Slow-Flow/No-Reflow)	P-Value [b]
Infarct-related artery			0.005
LCX (n = 55)	45 (81.8)	10 (18.2)	
RCA (n = 49)	38 (77.6)	11 (22.4)	
LAD (n = 46)	25 (54.3)	21 (45.7)	

[a] Values are expressed as No. (row %), ie, the percentage of patients with each infarct-related artery who had normal or impaired flow. Abbreviations: LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

[b] The P value was calculated using the chi-square test.

Table 3. Comparison of Shock Index According to Coronary Flow Status [a]

Variables	Shock Index	P-Value [b]
Coronary flow		0.005
Normal flow	0.60 ± 0.05	
Impaired flow (slow-flow/no-reflow)	0.65 ± 0.09	

[a] Values are expressed as mean ± SD. Abbreviation: SD, standard deviation.

[b] The P value was calculated using the Mann–Whitney U test.

Wang et al. (14) reported that a higher SI on admission was associated with coronary slow-flow or no-reflow in patients with acute myocardial infarction undergoing emergent PCI. Similarly, Sahsah et al. (23) reported higher SI in patients with STEMI who developed slow-flow or no-reflow, and Reinstadler et al. (15) linked SI to myocardial damage in STEMI.

A possible explanation is that a higher SI reflects reduced left ventricular systolic function or greater sympathetic activation at presentation, leading to lower coronary perfusion pressure and impaired microvascular flow. Reduced systolic blood pressure diminishes the driving force for myocardial perfusion, particularly in the setting of microvascular obstruction and tissue edema following ischemia–reperfusion injury (24). Patients with higher SI may also have larger infarcts, a greater thrombus burden (25), or a longer ischemic time (26), all of which are related to impaired reperfusion. However, infarct size, thrombus burden, and ischemic time were not measured in our study; therefore, these explanations remain hypothetical, and SI may simply be a marker of other factors.

Impaired coronary flow was also more frequent in patients with LAD-related infarctions than in those with right coronary or left circumflex artery involvement, consistent with previous observational data identifying LAD culprit location as one of the predictors of no-reflow (6, 8, 19). A larger myocardium at risk and a greater likelihood of thrombus burden and distal embolization may contribute to this finding, although we did not measure these factors.

Our findings may have some practical implications. SI is a simple and rapid parameter that can be calculated at the bedside without laboratory tests or imaging, and its calculation does not delay reperfusion therapy. If its association with impaired reperfusion is confirmed in larger adjusted studies, SI could help identify patients who require closer attention during and after PPCI, for example, through hemodynamic optimization, minimization of distal embolization, or selective use of intracoronary vasodilators. At present, however, our results are insufficient to recommend SI as a risk-stratification tool.

This study has several limitations. It was a single-center observational study with a modest sample size. Only unadjusted comparisons were performed; therefore, the associations may have been influenced by other factors, such as age, diabetes, ischemic time, time to treatment, pre-PCI flow, Killip class, thrombus burden, and medications, which were not analyzed. Patients with severe cardiogenic shock were excluded; consequently, the range of SI was narrow, and the findings may not apply to the most critically ill patients. The difference in SI between groups was small, and we did not evaluate cutoff values or predictive accuracy (for example, by receiver operating characteristic analysis). SI was measured only at admission,

and no clinical outcomes were assessed. Larger multicenter studies with adjusted analyses are needed.

5.1. Conclusions

In this single-center study of patients with STEMI undergoing primary PCI, a higher admission shock index and LAD involvement were associated with slow-flow or no-reflow. These findings suggest that SI may be a simple bedside marker of impaired reperfusion; however, its predictive value, independence from other risk factors, and clinical usefulness remain to be tested in larger, adjusted, prospective studies.

Footnotes

AI Use Disclosure: The authors declare that no generative AI tools were used in the creation of this article.

Authors' Contribution: Conceptualization: Mohammad Hassan Namazi; Data curation: Reyhaneh Taati and Shabnam Kazemloo; Supervision: Mohammad Hassan Namazi and Shabnam Kazemloo; Writing—original draft: Reyhaneh Taati.

Conflict of Interests Statement: The authors declare no conflicts of interest related to this manuscript.

Data Availability: The dataset presented in this study is available from the corresponding author upon request during submission or after publication. The data are not publicly available.

Ethical Approval: The study protocol was approved by the Ethics Committee of Shahid Beheshti University of Medical Sciences (approval ID: IR.SBMU.RETECH.REC.1404.492), and the study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

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