RESEARCH ARTICLE

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Value of Shock Index in Predicting No Reflow after Primary Percutaneous Coronary Intervention

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Abstract

Background and Aim: No-reflow (NR) after primary percutaneous coronary intervention (pPCI) remains a frequent and clinically important complication in patients with ST-segment elevation myocardial infarction (STEMI). This study evaluated the predictive value of the shock index (SI) and the modified SI (MSI) for angiographic NR after pPCI.

Materials and Methods: This prospective observational cohort study included 90 patients with STEMI who underwent pPCI at a single university hospital between June 2020 and June 2022. Admission SI was calculated as heart rate divided by systolic blood pressure, and MSI was calculated as heart rate divided by mean arterial pressure. Patients were classified as normal flow (n=65) or NR (n=25) according to post-procedural TIMI flow. Discrimination was evaluated using receiver operating characteristic analysis.

Results: NR occurred in 25 patients (27.8%). Admission SI and MSI were higher in the NR group than in the normal flow group (0.937±0.30 vs. 0.55±0.09 and 1.29±0.45 vs. 0.763±0.167, respectively; both $P < 0.001$). The area under the curve was 0.842 [95% confidence interval (CI): 0.726-0.957] for SI and 0.838 (95% CI: 0.723-0.953) for MSI. At cut-offs of 0.605 and 0.805, sensitivity was 80.0% and 84.0%, respectively; specificity was 75.4% and 70.8%, respectively. These estimates describe apparent performance within the study cohort.

Conclusion: Higher SI and MSI at admission were associated with angiographic NR after pPCI in this small cohort. Neither comparative superiority nor independent or incremental predictive value has been established. The observed discrimination and cut-off values require validation in larger cohorts.

Keywords: Shock index, modified shock index, no-reflow, primary PCI, STEMI

INTRODUCTION

Primary percutaneous coronary intervention (pPCI), along with coronary stenting, dual antiplatelet therapy, and statins, has markedly lowered both early and late mortality in patients with acute myocardial infarction (AMI).[1] Nevertheless, successful restoration of epicardial coronary flow does not invariably result in effective myocardial reperfusion. A considerable proportion of patients still develop substantial microvascular perfusion impairment due to no-reflow (NR) phenomenon despite successful recanalization of infarct-related artery, with previous studies reporting an incidence of up to 20-30% in this setting.[2]

In PCI, angiographic NR is defined as reduced or absent myocardial tissue perfusion despite a patent epicardial coronary artery after PCI. It corresponds to TIMI flow <3 and myocardial blush grade <3 TIMI in the absence of mechanical causes such as residual stenosis, dissection.[3] This phenomenon carries major clinical importance, as it is associated with a higher rate of complications and with greater short- and long-term morbidity and mortality in patients with acute ST-segment

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elevation myocardial infarction (STEMI).[4] Although NR has been reported in only 0.6% to 5% of elective PCI procedures, its frequency is considerably greater in patients undergoing pPCI, underscoring its particular importance in AMI management.[5]

The pathophysiology of NR is intricate and multifactorial. Several mechanisms have been suggested, including distal embolization of material from ruptured atherosclerotic plaques, vasospasm, oxidative stress, microvascular injury, and reperfusion damage.[6] This condition has been reported to complicate nearly 5% to 15% of revascularization procedures.[7] In clinical practice, many studies have explored potential predictors of NR, and factors such as thrombus burden, reperfusion delay, inflammatory biomarkers, and stent-to-vessel diameter ratio have been identified as possible contributors to its occurrence and to clinical outcome in patients with AMI undergoing emergent PCI.[8] Despite the growing volume of evidence, no universally accepted risk stratification tool for predicting this complication has yet been established.[9]

Shock index (SI), calculated as the ratio of heart rate (HR) to systolic blood pressure (SBP), has been investigated as a marker of adverse outcomes in patients with AMI, although its association with NR has been less extensively studied.[10] We hypothesized that higher admission SI and modified SI (MSI) would be associated with angiographic NR after pPCI. The study aimed to evaluate these associations and the discrimination of each index. The analyses reported here address unadjusted associations and discrimination.

METHODS

Design and Population

This prospective observational cohort study included 90 patients with STEMI who underwent pPCI at the cardiology department of the participating university hospital between June 2020 and June 2022. The study protocol was approved by the institutional Research Ethics Committee of Mansoura University (approval code: MD.20.02.285, date: 26.04.2020). Verbal informed consent was obtained from all participants in accordance with the consent procedure approved by the Research Ethics Committee. Enrollment was based on the predefined study period; no formal a priori sample size calculation was performed.

Eligibility Criteria

Patients with STEMI who underwent pPCI during the study period were considered eligible for inclusion. Exclusion criteria were: a retrospective diagnosis of myocardial infarction (MI) with non-obstructive coronary arteries; absence of stent implantation because of unsuitable coronary anatomy, non-significant lesions, or referral for CABG; prior fibrinolytic therapy within a pharmaco-invasive approach; trauma or recent surgery within the previous month; and malignancy, hematologic disorders, severe renal impairment, febrile illness, hepatic dysfunction, or chronic or acute inflammatory conditions at enrollment. Patients presenting ≥24 hours after AMI onset were also excluded.

Clinical Assessment

After enrollment, all patients underwent detailed history- taking and clinical evaluation. Demographic and clinical data included age, sex, diabetes mellitus (DM), hypertension (HTN), smoking history, family history of coronary artery disease, prior transient ischemic attack, prior stroke, prior MI, renal impairment, and dyslipidemia. General examination included assessment of vital signs on admission, including HR, BP, temperature, and RR, and examination for jugular venous distension and lower limb edema. A local cardiac examination was also performed to detect pathological murmurs or abnormal heart sounds suggestive of HF.

SI and MSI

SI was calculated for each patient as HR divided by SBP.[11] MSI was calculated as HR divided by mean arterial pressure (MAP); MAP was calculated as diastolic blood pressure plus one-third of the pulse pressure.[12] Both indices were assessed on admission as bedside hemodynamic markers.

SI and MSI share HR as the numerator and incorporate related blood pressure measurements in their denominators. Their correlations with HR and blood pressure, therefore, partly reflect their mathematical construction. The indices were evaluated as individual markers, and no inference of independent association was made from the simultaneous inclusion of SI, MSI, and their component variables.

Laboratory and Non-invasive Investigations

Venous blood samples were obtained on the day of admission and sent to the laboratory for routine investigations using standard laboratory methods including CBC, lipid profile, renal and liver function tests, random blood glucose (RBS), and cardiac biomarkers. Serum creatinine was used as part of the assessment of renal function.

All patients underwent a standard 12-lead electrocardiography (ECG) at a paper speed of 25 mm/s and a calibration of 1 mV=10 mm. ECG evaluation included assessment of HR, rhythm, ST-segment elevation and depression, T-wave inversion, and pathological Q-waves. Transthoracic echocardiography was performed in accordance with institutional practice using a General Electric Vivid S5 system to assess systolic and diastolic cardiac function.

Coronary Angiography and Procedural Assessment

All patients underwent coronary angiography in multiple projections. The culprit lesion was defined as a lesion showing complex angiographic features consistent with acute plaque rupture, such as an intraluminal filling defect or thrombus, a dissection or an intraluminal flap, extraluminal contrast leakage, an ulcer with overhanging edges, multiple irregular contours, or an acute vessel occlusion. Based on its anatomical location, the culprit lesion was categorized as proximal, mid, or distal.

TIMI flow[13] was angiographically evaluated in all patients. TIMI grade 0 denoted the absence of antegrade flow distal to the culprit lesion; grade 1 indicated the passage of contrast beyond the occlusion without full opacification of the distal vessel; grade 2 indicated complete opacification of the distal vessel with delayed antegrade flow; and grade 3 represented normal coronary perfusion. Angiographic NR during PCI was defined as TIMI flow grade ≤2 in the absence of mechanical causes such as residual stenosis, dissection, distal embolization, or vasospasm.

Outcome Measures

The primary outcome was angiographic NR after pPCI in patients with STEMI. The analysis evaluated the association of admission SI and MSI with this outcome and the discriminatory performance of each index.

Statistical Analysis

Data were analyzed using SPSS. Categorical variables were summarized as numbers and percentages, and continuous variables as means ± standard deviations. Between-group comparisons used the chi-square test or the Fisher exact test for categorical variables, and the Student's t-test for continuous variables. Spearman rank correlation was used to assess associations between continuous variables. Receiver operating characteristic analysis was used to evaluate discrimination between SI and MSI, yielding AUCs with 95% confidence intervals (CIs). Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy were reported at the selected cut-offs. All analyses were exploratory, and a two-sided *P*-value ≤0.05 was considered statistically significant. The reported performance estimates are apparent estimates based on the study cohort. No formal paired AUC comparison, verified adjusted model, calibration assessment, or optimism-corrected performance are reported.

RESULTS

Patients with NR were substantially older than those with normal flow (66.80±11.13 vs. 53.63±10.46 years, $P < 0.001$). They also had markedly higher frequencies of HTN (88.0% vs. 26.2%, $P < 0.001$), DM (84.0% vs. 27.7%, $P < 0.001$), and dyslipidemia (96.0% vs. 35.4%, $P < 0.001$) than in patients with normal flow. In contrast, glycoprotein (GP) IIb/IIIa inhibitors and thrombus aspiration were notably more frequently used in the normal flow group than in the NR group (72.3% vs. 40.0%, $P = 0.004$; 61.5% vs. 8.0%, $P < 0.001$, respectively) (Table 1).

Patients with NR had markedly higher HR than those with normal flow (94.32±24.76 vs. 78.40±12.43 beats/min, $P < 0.001$), while SBP was substantially lower in the NR group (103.80±22.19 vs. 141.85±16.97 mmHg, $P < 0.001$). They also had substantially higher serum creatinine levels (1.15±0.37 vs. 0.989±0.21 mg/dL, $P = 0.012$) and markedly higher RBS levels (323.0±58.17 vs. 180.2±45.49 mg/dL, $P < 0.001$) than patients with normal flow. In addition, patients with NR showed notably lower EF (45.16±7.13% vs. 51.20±8.41%, $P = 0.002$), together with substantially higher SI (0.937±0.30 vs. 0.55±0.09, $P < 0.001$) and MSI (1.29±0.45 vs. 0.763±0.167, $P < 0.001$) (Table 2).

For SI, the AUC for angiographic NR was 0.842 (95% CI: 0.726-0.957). At the selected cut-off of 0.605, sensitivity was 80.0%, specificity 75.4%, PPV 55.6%, NPV 90.7%, and accuracy 76.7% (Table 3; Figure 1A).

For MSI, the AUC was 0.838 (95% CI: 0.723-0.953). At the selected cut-off of 0.805, sensitivity was 84.0%, specificity 70.8%, PPV 52.5%, NPV 92.0%, and accuracy 74.4% (Table 3; Figure 1B). No formal paired comparison of the two AUCs is reported; these estimates do not establish the superiority or equivalence between the two indices.

SI showed significant positive correlations with age (r=0.226, $P = 0.032$), HR (r=0.817, $P < 0.001$), and RBS (r=0.248, $P = 0.018$), and significant negative correlations with SBP (r=-0.715, $P < 0.001$) and EF (r=-0.350, $P = 0.001$). Similarly, MSI showed significant positive correlations with HR (r=0.813, $P < 0.001$) and RBS (r=0.227, $P = 0.03$), and significant negative correlations with SBP (r=-0.532, $P < 0.001$) and EF (r=-0.426, $P < 0.001$) (Table 4).

SI and MSI, according to different categorical parameters in the studied patients, are shown in Table 5.

The findings presented above represent unadjusted group comparisons, correlations, and apparent discrimination. They do not establish that SI or MSI predicts NR independently of age, comorbidities, admission hemodynamics, or angiographic and procedural factors.

DISCUSSION

Slow-flow/NR during pPCI is a frequent complication associated with poor prognosis, with a reported incidence ranging widely from 4% to 44%.[14] Its pathophysiology is complex and multifactorial, with proposed mechanisms including microvascular spasm, endothelial swelling secondary to ischemia-reperfusion injury, and distal microembolization of

Table 1. General characteristics of the study population

Characteristic	Total (n=90)	Normal flow (n=65)	NR (n=25)	*P*-value
Age (years)	57.29±12.14	53.63±10.46	66.80±11.13	<0.001*
Sex				**0.682**
Male	77 (85.6)	55 (84.6)	22 (88.0)	
Female	13 (14.4)	10 (15.4)	3 (12.0)	
HTN	39 (43.3)	17 (26.2)	22 (88.0)	<0.001*
Diabetes	39 (43.3)	18 (27.7)	21 (84.0)	<0.001*
Previous stroke	6 (6.7)	3 (4.6)	3 (12.0)	0.342
Anemia	24 (26.7)	15 (23.1)	9 (36.0)	0.214
Smoking	53 (58.9)	39 (60.0)	14 (56.0)	0.813
Dyslipidemia	47 (52.2)	23 (35.4)	24 (96.0)	<0.001*
MI type				**0.356**
Anterior	46 (51.1)	31 (47.7)	15 (60.0)	
Inferior	37 (41.1)	27 (41.5)	10 (40.0)	
Lateral	6 (6.7)	6 (9.2)	0 (0.0)	
Posterior	1 (1.1)	1 (1.5)	0 (0.0)	
GP IIb/IIIa inhibitors	57 (63.3)	47 (72.3)	10 (40.0)	0.004*
Thrombus aspiration	42 (46.7)	40 (61.5)	2 (8.0)	<0.001*

Values are mean ± SD or n (%). HTN: Hypertension, NR: No-reflow, SD: Standard deviation, MI: Myocardial infarction, GP: Glycoprotein, *$P \leq 0.05$

Table 2. Clinical and laboratory characteristics by angiographic outcome

Variable	Normal flow (n=65)	NR (n=25)	*P*-value
HR (beats/min)	78.40±12.43	94.32±24.76	<0.001*
SBP (mmHg)	141.85±16.97	103.80±22.19	<0.001*
Stent length (mm)	33.26±8.63	33.08±8.75	0.929
Stent diameter (mm)	3.30±0.44	3.31±0.48	0.983
Serum creatinine (mg/dL)	0.989±0.21	1.15±0.37	0.012*
RBS (mg/dL)	180.2±45.49	323.0±58.17	<0.001*
EF (%)	51.20±8.41	45.16±7.13	0.002*
Time to PCI (minutes)	73.94±24.71	81.64±28.81	0.21
Shock index	0.55±0.09	0.937±0.30	<0.001*
Modified shock index	0.763±0.167	1.29±0.45	<0.001*

Values are mean ± SD. HR: Heart rate, SBP: Systolic blood pressure, NR: No-reflow, RBS: Random blood glucose, EF: Ejection fraction, PCI: Percutaneous coronary intervention, *$P \leq 0.05$

Table 3. Apparent discrimination of SI and MSI for angiographic NR

Measure	SI	MSI
AUC (95% CI)	0.842 (0.726-0.957)	0.838 (0.723-0.953)
Selected cut-off	0.605	0.805
Sensitivity (%)	80.0	84.0
Specificity (%)	75.4	70.8
PPV (%)	55.6	52.5
NPV (%)	90.7	92.0
Accuracy (%)	76.7	74.4

SI: Shock index, MSI: Modified shock index, NR: No-reflow, AUC: Area under the receiver operating characteristic curve, CI: Confidence interval, PPV: Positive predictive value, NPV: Negative predictive value. Values describe performance in the study cohort. No paired AUC comparison or superiority claim is reported

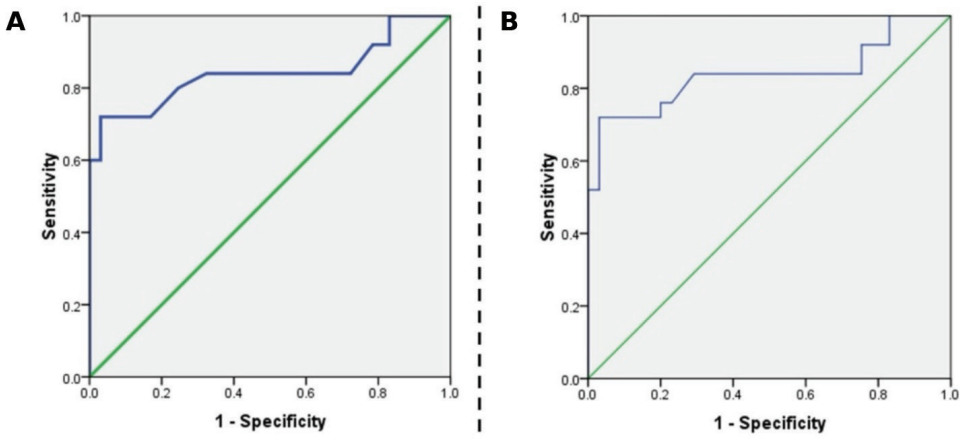


Figure 1. ROC curves for admission SI (A) and MSI (B) in relation to angiographic NR after pPCI

ROC: Receiver operating characteristic, SI: Shock index, MSI: Modified shock index, NR: No-reflow, pPCI: Primary percutaneous coronary intervention

Table 4. Correlations of SI and MSI with continuous variables

Variable	SI r	SI *P*-value	MSI r	MSI *P*-value
Age (years)	0.226	0.032*	0.051	0.636
SBP (mmHg)	-0.715	<0.001*	-0.532	<0.001*
HR (beats/min)	0.817	<0.001*	0.813	<0.001*
Stent length (mm)	0.158	0.137	0.175	0.098
Stent diameter (mm)	-0.044	0.679	0.008	0.94
Serum creatinine (mg/dL)	0.065	0.541	0.093	0.386
RBS (mg/dL)	0.248	0.018*	0.227	0.03*
EF (%)	-0.35	0.001*	-0.426	<0.001*
Time to PCI (minutes)	0.098	0.359	-0.026	0.807

Correlations with HR and SBP partly reflect the mathematical definitions of SI and MSI. r: Spearman rank correlation coefficient, SI: Shock index, MSI: Modified shock index, EF: Ejection fraction, PCI: Percutaneous coronary intervention, *$P \leq 0.05$

thrombotic debris.[15] Several patient- and procedure-related factors have been linked to its occurrence, most consistently advanced age, high thrombus burden, delayed reperfusion, low preprocedural TIMI flow grade, longer target lesion length, and elevated C-reactive protein.[16]

SI is a simple marker that combines HR and SBP and has been studied in acute coronary syndromes and STEMI. Its association with angiographic NR should be distinguished from its association with other outcomes, including cardiogenic shock.[11,17]

Patients who developed NR were older and had higher prevalence of DM, HTN, and dyslipidemia. These differences are potential sources of confounding, rather than evidence that SI or MSI contributes independent prognostic information. GP IIb/IIIa inhibitor use and thrombus aspiration were more frequent in the normal flow group, but these unadjusted observations cannot establish a protective treatment effect because procedural treatment selection may depend on patient and lesion characteristics.

Consistent with our results, Mazhar et al.[18] reviewed the PCI database of Canberra Hospital and identified 781 patients who underwent pPCI between 2008 and 2012. Compared with patients without NR, those with NR were older (64±12 vs. 61±13 years) and tended to have a lower prevalence of smoking (22% vs. 29%). In addition, they more commonly presented with an initial TIMI flow grade of 0-1 (77% vs. 64%) and a thrombus score ≥4 (83% vs. 69%).

Patients with NR also had higher HR, serum creatinine, RBS, SI, and MSI, and lower SBP and EF. Stent length, stent diameter, and the reported time-to-PCI variable did not show statistically significant differences between groups. With only 25 NR events, non-significant results cannot exclude clinically relevant associations.

In agreement with our findings, Wang et al.[19] showed that SI was substantially different between patients with slow/NR

Table 5. SI and MSI according to categorical characteristics

Characteristic	SI	*P*-value	MSI	*P*-value
Sex		**0.259**		**0.18**
Male	0.673±0.264		0.932±0.380	
Female	0.588±0.110		0.785±0.170	
HTN		**0.002***		**0.008***
No	0.591±0.133		0.822±0.232	
Yes	0.753±0.327		1.030±0.460	
Diabetes		**0.051**		**0.109**
No	0.616±0.190		0.857±0.290	
Yes	0.719±0.290		0.981±0.430	
Stroke		**0.368**		**0.42**
No	0.655±0.247		0.902±0.360	
Yes	0.750±0.274		1.030±0.370	
Smoking history		**0.024***		**0.009***
No	0.591±0.192		0.793±0.270	
Yes	0.710±0.273		0.993±0.390	
Anemia		**0.2**		**0.106**
No	0.641±0.229		0.873±0.330	
Yes	0.717±0.296		1.010±0.430	
Dyslipidemia		**0.002***		**0.015***
No	0.576±0.129		0.814±0.230	
Yes	0.739±0.300		0.999±0.430	
MI type		**0.029***		**0.029***
Anterior	0.737±0.280		1.010±0.400	
Inferior	0.581±0.190		0.782±0.270	
Lateral	0.600±0.120		0.995±0.300	
Posterior	0.500±0.000		0.640±0.000	
GP IIb/IIIa inhibitors		**0.414**		**0.497**
No	0.689±0.281		0.945±0.405	
Yes	0.645±0.229		0.891±0.330	
Thrombus aspiration		**0.281**		**0.652**
No	0.634±0.253		0.984±0.370	
Yes	0.691±0.244		0.929±0.350	

Values are mean ± SD. SI: Shock index, MSI: Modified shock index, GP: Glycoprotein, HTN: Hypertension. *P-values* are shown once for each categorical comparison. *$P \leq 0.05$

and those with normal reflow. They further reported that SI ≥0.66 was markedly more frequent in the slow/NR group than in the normal reflow group. In addition, post-PCI LVEF was lower among patients with slow/NR compared with those who achieved normal reflow.

In contrast to our findings, Wang et al.[16] observed no notable variations between the two groups regarding infarct-related coronary artery or cardiovascular risk factors, including DM, HTN, hyperlipidemia, and in-hospital medications.

The observed correlations of SI and MSI with HR and SBP must be interpreted in light of the indices' mathematical definitions. These correlations do not independently validate the indices as prognostic markers. Associations with age, RBS, EF, and categorical clinical characteristics are exploratory and may reflect confounding.

Hyperglycemia has been linked to microvascular dysfunction and may promote angiographic slow/NR. Several mechanisms may account for this association. In particular, hyperglycemia enhances platelet-mediated thrombosis, impairs endothelium-dependent vasodilation, and diminishes collateral perfusion through reduced nitric oxide bioavailability.[20]

Somuncu et al.[21] reported that lower SBP was associated with NR alongside other clinical and angiographic factors. This supports consideration of the underlying hemodynamic

measurements when evaluating the additional information provided by SI or MSI.

SI summarizes the relationship between HR and SBP in a single ratio. Whether that ratio provides more useful information than the original measurements requires a direct comparison of appropriately specified models. The present unadjusted analyses do not answer that question.

The SI threshold reported by Wang et al.[19] in relation to slow/NR was 0.66, whereas the selected SI cut-off in our cohort was 0.605. Differences in patient populations, outcome definitions, and cut-off selection limit the transferability of a threshold from one cohort to another.

The findings support an association between admission hemodynamic status and subsequent angiographic NR. However, the limited number of events and the large differences in baseline risk factors constrain the interpretation. The original combined model cannot support claims of independent predictors because SI, MSI, and SBP encode overlapping hemodynamic information and because the number of predictors was large relative to the number of events. Valid, parsimonious models that separate the indices from their constituent measurements are needed to evaluate adjusted associations.

Wei et al. [17] evaluated SI for cardiogenic shock during pPCI. Because that outcome differs from angiographic NR, their results provide contextual evidence for hemodynamic risk assessment rather than direct validation of the present NR estimates.

The apparent AUCs of 0.842 for SI and 0.838 for MSI were numerically close. In the absence of a formal paired comparison, no conclusion about superiority or equivalence is justified. The cut-offs were assessed in the same small cohort and may perform differently in new patients. Their sensitivity, specificity, and predictive values should therefore not be treated as validated clinical decision thresholds.

The incremental value of SI or MSI beyond established clinical and angiographic factors remains unresolved. The study does not establish improved calibration, risk classification, or clinical outcomes from adding either index to routine assessment. SI and MSI should therefore be interpreted as exploratory markers in this setting.

Mazhar et al.[18] reported that thrombus score ≥4, symptom-to-balloon time >360 minutes, and age >60 years were significant predictors of NR in patients with STEMI.

Differences across published studies may reflect variation in populations, endpoint definitions, reperfusion delays, treatment selection, and sample size. These factors support the need for larger cohorts with consistent definitions and prespecified analytic methods.

Study Limitations

This single-center study included 90 patients, among whom only 25 NR events occurred, and no formal a priori sample-size calculation was performed. The small number of events limits precision and precludes reliable multivariable modeling. The original combined regression estimates are not retained because overlapping hemodynamic predictors increase the risk of unstable estimation and overfitting. Therefore, independent and incremental predictive value remains unestablished. Non-significant findings should not be interpreted as evidence of no association.

Potentially important confounders, including baseline TIMI flow, quantitative thrombus burden, Killip class, lesion complexity, collateral flow, SYNTAX score, contrast volume, detailed reperfusion-delay measures, and procedural pharmacotherapy, were not comprehensively evaluated or adjusted for. Residual confounding cannot be excluded. Exclusion of patients presenting ≥24 hours after symptom onset limits applicability to late-presenting STEMI, and reliance on clinical history obtained during acute presentation may introduce information bias.

Angiographic NR was assessed using the TIMI flow grade, without assessment by an independent angiographic core laboratory or corroboration with complementary measures such as myocardial blush grade or corrected TIMI frame count. TIMI flow is operator-dependent and does not directly quantify microvascular perfusion at the tissue level.

Distributional checks for continuous variables and handling of variable-level missing data are not documented in the reported analyses. In addition, no verified calibration assessment, internal validation, or external validation is reported. The apparent AUCs and selected cut-offs may, therefore, be optimistic and require confirmation in larger, independent cohorts before use in clinical risk stratification.

CONCLUSION

In this small prospective cohort, higher admission SI and MSI were associated with angiographic NR after pPCI. The reported AUCs describe apparent within-cohort discrimination and do not establish the superiority of either index, independent predictive ability, or incremental clinical value. Further analyses using appropriately specified models and validation in larger cohorts are required.

Ethics

Ethics Committee Approval: The study protocol was approved by the institutional Research Ethics Committee of Mansoura University (approval code: MD.20.02.285, date: 26.04.2020).

Informed Consent: Verbal informed consent was obtained from all participants in accordance with the consent procedure approved by the Research Ethics Committee

Footnotes

Authorship Contributions

Data Collection or Processing: A.I.B., A.A.W.S., M.M.A.Y., Analysis or Interpretation: M.M.A.Y., Writing: A.I.B., E.M.E.H.S., A.A.W.S., S.M.T.

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REFERENCES

1. Kim YH, Her AY, Jeong MH, Kim BK, Hong SJ, Kim S, *et al.* Effect of statin treatment in patients with acute myocardial infarction with prediabetes and type 2 diabetes mellitus: a retrospective observational registry study. Medicine (Baltimore). 2021;100:e24733.
2. Rezkalla SH, Dharmashankar KC, Abdalrahman IB, Kloner RA. No-reflow phenomenon following percutaneous coronary intervention for acute myocardial infarction: incidence, outcome, and effect of pharmacologic therapy. J Interv Cardiol. 2010;23:429-36.
3. Yue J, He S. Letter by Yue J, et al. regarding article "The relationship between atherogenic index of plasma and no-reflow in patients with acute ST-segment elevation myocardial infarction who underwent primary percutaneous coronary intervention". Int J Cardiovasc Imaging. 2020;36:1187-8.
4. Chen X, Meng Y, Shao M, Zhang T, Han L, Zhang W, *et al*. Prognostic value of pre-infarction angina combined with mean platelet volume to lymphocyte count ratio for no-reflow and short-term mortality in patients with ST-segment elevation myocardial infarction undergoing percutaneous coronary intervention. Med Sci Monit. 2020;26:e919300.
5. Durante A, Camici PG. Novel insights into an "old" phenomenon: the no reflow. Int J Cardiol. 2015;187:273-80.
6. Jaffe R, Charron T, Puley G, Dick A, Strauss BH. Microvascular obstruction and the no-reflow phenomenon after percutaneous coronary intervention. Circulation. 2008;117:3152-6.
7. Ünal S, Yayla Ç, Yayla KG, Karanfil M, Ertem AG, Erdöl MA, *et al.* Anticoagulants and no-reflow. Angiology. 2020;71:192.
8. Zhou H, He XY, Zhuang SW, Wang J, Lai Y, Qi WG, *et al.* Clinical and procedural predictors of no-reflow in patients with acute myocardial infarction after primary percutaneous coronary intervention. World J Emerg Med. 2014;5:96-102.
9. Mirbolouk F, Gholipour M, Salari A, Shakiba M, Kheyrkhah J, Nikseresht V, *et al.* CHA2DS2-VASc score predict no-reflow phenomenon in primary percutaneous coronary intervention in primary percutaneous coronary intervention. J Cardiovasc Thorac Res. 2018;10:46-52.
10. Abe N, Miura T, Miyashita Y, Hashizume N, Ebisawa S, Motoki H, *et al.* Long-term prognostic implications of the admission shock index in patients with acute myocardial infarction who received percutaneous coronary intervention. Angiology. 2017;68:339-45.
11. Koch E, Lovett S, Nghiem T, Riggs RA, Rech MA. Shock index in the emergency department: utility and limitations. Open Access Emerg Med. 2019;11:179-99.
12. Qiang J, Sun C, Ye F, Wang D, Lv Y, Zheng X. Shock index, modified shock index, and age-adjusted shock index for the prediction of impaired myocardial work in patients with ST-segment elevation myocardial infarction. Quant Imaging Med Surg. 2025;15:12290-302.
13. Gibson CM, Cannon CP, Daley WL, Dodge JT Jr, Alexander B Jr, Marble SJ, *et al.* TIMI frame count: a quantitative method of assessing coronary artery flow. Circulation. 1996;93:879-88.
14. Kumar R, Ammar A, Saghir T, Sial JA, Khan KA, Shah JA, *et al.* Development and validation of a novel risk stratification model for slow-flow/no-reflow during primary percutaneous coronary intervention (the RK-SF/NR score). Am J Cardiol. 2022;171:32-9.
15. Liu Y, Ye T, Chen K, Wu G, Xia Y, Wang X, *et al.* A nomogram risk prediction model for no-reflow after primary percutaneous coronary intervention based on rapidly accessible patient data among patients with ST-segment elevation myocardial infarction and its relationship with prognosis. Front Cardiovasc Med. 2022;9:966299. Erratum in: Front Cardiovasc Med. 2023;10:1229889.
16. Wang JW, Chen YD, Wang CH, Yang XC, Zhu XL, Zhou ZQ. Development and validation of a clinical risk score predicting the no-reflow phenomenon in patients treated with primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. Cardiology. 2013;124:153-60.
17. Wei Z, Bai J, Dai Q, Wu H, Qiao S, Xu B, *et al.* The value of shock index in prediction of cardiogenic shock developed during primary percutaneous coronary intervention. BMC Cardiovasc Disord. 2018;18:188.
18. Mazhar J, Mashicharan M, Farshid A. Predictors and outcome of no-reflow post primary percutaneous coronary intervention for ST elevation myocardial infarction. Int J Cardiol Heart Vasc. 2015;10:8-12.
19. Wang Q, Shen H, Mao H, Yu F, Wang H, Zheng J. Shock index on admission is associated with coronary slow/no reflow in patients with acute myocardial infarction undergoing emergent percutaneous coronary intervention. J Interv Cardiol. 2019;2019:7873468.
20. Meza CA, La Favor JD, Kim DH, Hickner RC. Endothelial dysfunction: is there a hyperglycemia-induced imbalance of NOX and NOS? Int J Mol Sci. 2019;20:3775.
21. Somuncu MU, Akgun T, Cakır MO, Akgul F, Serbest NG, Karakurt H, *et al.* The elevated soluble ST2 predicts no-reflow phenomenon in ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. J Atheroscler Thromb. 2019;26:970-8.