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Correlation Between Modified Shock Index and Number/Type of Involved Vessels in STEMI Patients: A Predictive Approach

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A B S T R A C T

Introduction: ST-segment elevation myocardial infarction (STEMI) demands rapid risk stratification, often limited by the availability of complex scores. The Modified Shock Index (MSI), derived from routine vital signs, reflects acute circulatory stress. This study aims to determine the predictive correlation between admission MSI values and the underlying angiographic coronary anatomy, specifically the number and type of involved vessels, thereby assessing MSI's utility as a simple bedside marker for disease complexity in STEMI patients.

Material and methods: This cross-sectional study analyzed 234 consecutive STEMI patients via census sampling at Shahid Madani Hospital in Tabriz between 2018 and 2019. The core procedure involved calculating the Modified Shock Index (MSI) from vital signs recorded at admission. The study then correlated these pre-treatment MSI values with the detailed coronary anatomy determined by subsequent angiography to assess the extent of vessel involvement.

Results: Patients with high MSI (>0.897) were older (61 vs. 57 years, p=0.002) and had higher rates of hypertension (p=0.008), diabetes mellitus (p=0.002), and dyslipidemia (p=0.009), along with longer chest pain duration (p=0.004) and elevated WBC (p=0.001), troponin (p=0.002), and creatinine levels (p=0.001). MSI did not differ by number or type of involved vessels (all p>0.05) but showed moderate predictive value for MACE (pre-PPCI AUC=0.677; post-PPCI AUC=0.631; both p<0.001).

Conclusion: In conclusion, this study demonstrates that the Modified Shock Index is closely associated with patient-level vulnerability and acute disease severity rather than the anatomical extent or distribution of coronary artery involvement in STEMI.

Introduction

ST-segment elevation myocardial infarction (STEMI) represents one of the most serious and time-sensitive manifestations of coronary artery disease, leading to substantial morbidity and mortality worldwide. It occurs predominantly as a result of sudden occlusion of a major epicardial coronary artery following rupture or erosion of an unstable atherosclerotic plaque, inducing ischemia and necrosis of the myocardium.

The prompt restoration of coronary blood flow through primary percutaneous coronary intervention

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or thrombolytic therapy is the cornerstone of management to limit infarct size and preserve cardiac function. Despite major advances in reperfusion therapy and pharmacological management, early risk stratification remains essential to identify patients at high risk of adverse outcomes and facilitate appropriate triage and treatment decisions (1).

The severity of clinical presentation in STEMI frequently correlates with the underlying coronary anatomy, which has major implications for prognosis and management. Angiography plays a critical role in defining the culprit lesion and evaluating the presence of multi-vessel coronary artery disease (MVD). Patients with MVD tend to have a more extensive atherosclerotic burden, impaired ventricular function, and a higher risk of recurrent ischemic events compared to those with single-vessel disease (SVD). This subset of patients often requires more complex revascularization strategies and experiences poorer long-term outcomes. Moreover, the type and location of the involved coronary vessels such as left main or proximal left anterior descending artery introduce additional prognostic significance due to their larger myocardial territories and hemodynamic importance. Identifying these patterns earlier in the course of STEMI could thus improve prognostic assessment and guide clinical decision-making in the acute setting (2).

Traditional risk stratification tools, including the TIMI and GRACE scores, provide valuable prognostic insights but rely on numerous variables and laboratory parameters that may not be readily available at initial presentation. In emergency or resource-limited environments, simple and rapidly measurable indices derived from basic physiological parameters offer particular appeal. Predictors based on hemodynamic indices can capture the extent of cardio circulatory compromise induced by acute left ventricular failure and systemic stress. Heart rate and blood pressure remain two cornerstone vital signs that integrate the effects of sympathetic activation and myocardial workload. However, their isolated interpretation can underestimate subtle systemic decompensation, hence the need for composite indices like the Shock Index and its modified version (3).

The standard Shock Index (SI), defined as the ratio of heart rate to systolic blood pressure, reflects the relationship between tachycardia and hypotension and provides a rapid bedside measure of physiological instability. Although simple and easily obtained, the SI may fail to accurately represent tissue perfusion, particularly in cases of altered vascular tone, since it excludes the influence of diastolic pressure. In acute myocardial infarction, sympathetic activation leads to increased heart rate and vasoconstriction, which may maintain systolic pressure despite declining mean perfusion pressure. Consequently, sole reliance on SI can lead to underestimation of circulatory impairment. Studies have demonstrated the prognostic relevance of SI in trauma, sepsis, and cardiac emergencies, but its predictive sensitivity in cardiac contexts remains limited, motivating the refinement of this parameter (4).

The Modified Shock Index (MSI) addresses these limitations by incorporating mean arterial pressure (MAP) into the calculation, thereby reflecting both systolic and diastolic components of blood pressure. The formula MSI = HR/MAP accounts more effectively for systemic perfusion than SI, allowing for a more physiologically comprehensive representation of cardiovascular stress. Because diastolic pressure is directly linked to coronary perfusion, MSI provides insight into both systemic and myocardial oxygen delivery. In acute myocardial infarction, a higher MSI correlates with larger infarct size, poorer ventricular function, and increased mortality. By integrating heart rate elevation and reduced MAP, MSI mirrors the level of sympathetic drive and the degree of circulatory compromise. Recent studies suggest MSI may outperform the traditional SI in predicting clinical deterioration across a wide range of critical illnesses, including cardiac failure and shock (5).

From a pathophysiological standpoint, the Modified Shock Index may indirectly reflect the burden of coronary artery disease. Extensive atherosclerosis and multi-vessel involvement increase the risk of diffuse myocardial ischemia, impairing overall cardiac reserve. When STEMI occurs in a patient with preexisting multi-vessel disease, compensatory mechanisms are quickly overwhelmed, resulting in more pronounced hemodynamic instability observable through elevated heart rate and decreased mean arterial pressure. In contrast, patients with isolated single-vessel involvement often demonstrate more localized impairment and better systemic compensation. Therefore, changes in MSI measured upon presentation might be linked not only to the infarct size or hemodynamic status but also to the complexity and number of coronary vessels affected. This relationship may arise because extensive coronary disease exacerbates left ventricular dysfunction and reduces contractile reserve, magnifying the physiologic stress response even before invasive diagnostic procedures are performed (6).

The number and type of involved vessels carry distinct prognostic implications. Multi-vessel disease has been associated with increased risk of recurrent infarction, arrhythmias, heart failure, and higher mortality, while specific involvement of vessels such as the left main or proximal left anterior descending artery signifies greater myocardial jeopardy. These key anatomical territories contribute substantially to global ventricular function, and their obstruction imposes greater

demand on compensatory tachycardia and systemic vasoconstriction. The MSI, as a reflection of such compensatory balance, could thus indirectly mirror the underlying coronary complexity. This potential association offers a practical avenue for early risk estimation even before the coronary anatomy is delineated by angiography, enabling more informed decisions regarding hemodynamic support, timing of revascularization, and resource prioritization (7).

Despite the theoretical and physiological rationale linking MSI to coronary disease extent, there remains a notable paucity of evidence exploring this correlation specifically within STEMI populations. Existing literature primarily focuses on MSI as a marker of mortality or cardiogenic shock but rarely investigates its relationship with angiographic findings. Establishing a concrete association between MSI and the number or type of coronary vessels involved could transform early triage in cardiac emergencies. Clinicians could utilize a simple bedside measurement to infer disease burden, anticipating procedural complexity and prognostic risk before angiography results are available. Such an approach aligns with the growing emphasis on predictive physiological metrics that provide rapid, cost-effective, and reproducible risk assessment tools in acute coronary syndromes (8).

Thus, the present study aims to evaluate the correlation between the Modified Shock Index calculated at initial presentation and the angiographic characteristics of coronary involvement, including the number and the anatomical type of affected vessels in patients diagnosed with ST-segment elevation myocardial infarction. The goal is to determine whether MSI can function as a predictive marker for coronary complexity, thereby contributing to faster risk stratification and optimized patient management. Exploring this relationship could reinforce MSI's utility not only as a prognostic measure but also as a practical, non-invasive predictive tool reflecting the underlying extent of coronary pathology in acute myocardial infarction.

Material and methods

Study Design: This cross-sectional study was conducted at Shahid Madani Hospital, affiliated with Tabriz University of Medical Sciences, from the beginning of 2018 to the end of 2019. The investigation formed part of an approved thesis project (Thesis No. 63653) and was extracted from its specific objective number 4. The study aimed to evaluate the relationship between the Modified Shock Index (MSI) and the number and type of coronary vessels involved in patients with ST-segment elevation myocardial infarction (STEMI).

Sampling Method: A census sampling method was employed, including all eligible patients who met the inclusion criteria during the study period. Based on this approach, a total of 234 consecutive patients admitted with confirmed STEMI diagnosis were enrolled. All data were collected prospectively from hospital records and direct patient assessments following standardized protocols.

Inclusion and Exclusion Criteria: Participants were included if they were adults aged 18 years or older, admitted with a definitive diagnosis of ST-segment elevation myocardial infarction according to standard electrocardiographic and enzymatic criteria, and had undergone coronary angiography during the index hospitalization. Patients were required to have complete hemodynamic data at admission, including heart rate, systolic and diastolic blood pressure, from which the Modified Shock Index (MSI = HR/MAP) could be calculated. Exclusion criteria encompassed patients with non-ST elevation myocardial infarction (NSTEMI), unstable angina, cardiogenic shock prior to admission, significant valvular heart disease, history of previous revascularization (PCI or CABG), severe arrhythmias affecting hemodynamic stability, chronic renal or hepatic failure, and incomplete medical records. Patients with concurrent severe systemic infections or those receiving medications that could significantly alter blood pressure or heart rate at presentation were also excluded to minimize confounding physiological effects.

Study Procedure: Upon admission, all patients underwent comprehensive clinical evaluation, including detailed history-taking, physical examination, and baseline vital sign assessment. The heart rate and blood pressure were recorded using calibrated monitoring equipment immediately at the time of emergency department presentation before administration of any intravenous medications or fluids to accurately calculate the Modified Shock Index (MSI). Routine laboratory tests, including cardiac biomarkers, complete blood count, serum creatinine, and lipid profile, were performed according to standard hospital protocols. Electrocardiographic evaluation was done to confirm the presence and location of ST-segment elevation, and echocardiography was used when necessary to determine left ventricular function.

All patients underwent diagnostic coronary angiography within the recommended timeframe following admission, according to clinical stability and institutional protocols. The angiographic data were interpreted by two experienced interventional cardiologists blinded to the patients' MSI values. The number of significantly stenosed coronary arteries (defined as luminal narrowing ≥70%) and the type of involved vessels (LAD, LCx, RCA, or multi-vessel disease) were systematically documented. Based on angiographic findings, participants were categorized into groups according to single-, double-, or triple-vessel involvement. The relationship between the admission MSI and angiographic patterns was then analyzed to

determine the potential predictive capacity of MSI for the extent and complexity of coronary artery disease.

Statistical Analysis: All statistical analyses were performed using SPSS software version 23 (IBM Corp., Armonk, NY, USA). Quantitative data were expressed as mean ± standard deviation (SD) or median and interquartile range (IQR), depending on the distribution pattern. Categorical variables were presented as frequencies and percentages. The normality of data distribution was assessed using the Kolmogorov–Smirnov test. For comparison of quantitative variables between groups, either the independent t-test or the Mann–Whitney U test was employed as appropriate. Categorical variables were compared using the Chi-square test. Additionally, to illustrate group differences and perform One-way ANOVA, GraphPad Prism software version 9 (GraphPad Software, San Diego, CA, USA) was utilized. A two-tailed P-value <0.05 was considered statistically significant.

Ethical Considerations: This research strictly adhered to the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Tabriz University of Medical Sciences under the code IR.TBZMED.REC.1400.583. Written informed consent was obtained from all participants or their legal guardians prior to inclusion in the study. Confidentiality of all patient data was fully preserved throughout data collection, analysis, and reporting. Importantly, this investigation represents the fourth specific objective (Objective No. 4) of the postgraduate thesis project registered under Thesis No. 63653. The study was conducted under academic and ethical supervision as part of the requirements for the fulfillment of the Master's degree program at Tabriz University of Medical Sciences, ensuring complete compliance with institutional research standards.

Results

The comparative analysis between patients with low and high Modified Shock Index (MSI) values (<0.897 vs. >0.897) revealed significant differences in several demographic and risk-related characteristics. Patients in the high MSI group were slightly older on average (61 vs. 57 years, p=0.002), suggesting that elevated MSI values may parallel the hemodynamic vulnerability often seen with increasing age. The distribution of male gender, smoking habits, and family history of coronary artery disease showed no significant differences between groups, indicating these factors were evenly balanced. However, the prevalence of hypertension (p=0.008), diabetes mellitus (p=0.002), and dyslipidemia (p = 0.009) was notably higher among those with elevated MSI, reflecting a clustering of metabolic and vascular risk factors that may contribute to greater cardiovascular strain. Furthermore, the duration of chest pain before hospital presentation was longer in the high MSI cohort (9.1 vs. 7.56 hours, p=0.004), suggesting delayed care-seeking behavior or more gradual hemodynamic deterioration in these patients.

Regarding laboratory findings, no significant differences were observed in fasting blood glucose (p=0.717) or hemoglobin concentrations (p=0.123) between the two groups, implying that acute metabolic and oxygen-carrying parameters were comparable. In contrast, patients with higher MSI exhibited markedly elevated white blood cell counts (p=0.001), troponin levels (p=0.002), and serum creatinine (p=0.001), all pointing toward more pronounced systemic stress, myocardial injury, and transient renal dysfunction associated with heightened circulatory compromise. Taken together, these findings suggest that elevated MSI at admission is associated not only with greater cardiovascular risk factor burden but also with laboratory indicators of more severe acute myocardial and inflammatory responses, underscoring its potential prognostic value in stratifying disease severity among STEMI patients.

The present analysis sought to evaluate the relationship between the anatomical complexity of coronary artery disease, stratified by the number of diseased vessels (1VD, 2VD, and 3VD), and the Modified Shock Index (MSI) measured both before and after primary percutaneous coronary intervention (PPCI). The results indicated a distinct lack of statistical significance when comparing the baseline (Pre-PPCI) MSI values across the three groups (P=0.334). Furthermore, post-PPCI follow-up measurements similarly failed to establish a significant difference among the 1VD, 2VD, and 3VD cohorts (P=0.717). Consequently, these findings strongly suggest that, in this study population, the Modified Shock Index despite its utility in assessing broad hemodynamic status does not possess sufficient discriminatory power to serve as a sensitive, independent predictor of anatomical severity based purely on the number of involved coronary vessels (figure 1).

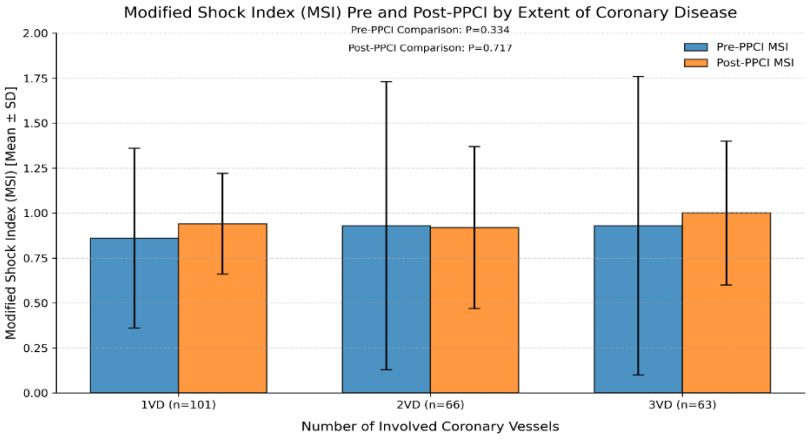

Figure 1. Mean Modified Shock Index (MSI) Distribution Across Coronary Vessel Involvement Groups (1VD, 2VD, and 3VD) Before and After Primary PCI.

In the present analysis, myocardial salvage index (MSI) did not differ meaningfully according to the infarct-related vessel, either before or after primary percutaneous coronary intervention (PPCI). Median pre-PPCI MSI values were broadly comparable across patients with LAD, LCX, LM and RCA involvement, and none of the vessel-specific comparisons reached statistical significance (all p-values >0.05), suggesting that the extent of potentially salvageable myocardium at presentation is not strongly determined by the culprit artery. Similarly, post-PPCI MSI remained relatively homogeneous among the four vessel groups, again with non-significant p-values, indicating that the effectiveness of reperfusion in terms of myocardial salvage appears to be generally consistent regardless of whether the infarction is related to LAD, LCX, LM or RCA lesions. Taken together, these findings imply that, within this cohort, vessel territory per se was not a major determinant of the magnitude of myocardial salvage, and that other factors such as ischemic time, microvascular integrity, collateral circulation, or patient-level clinical characteristics may play a more prominent role in influencing MSI than the anatomical location of the culprit lesion (figure 2).

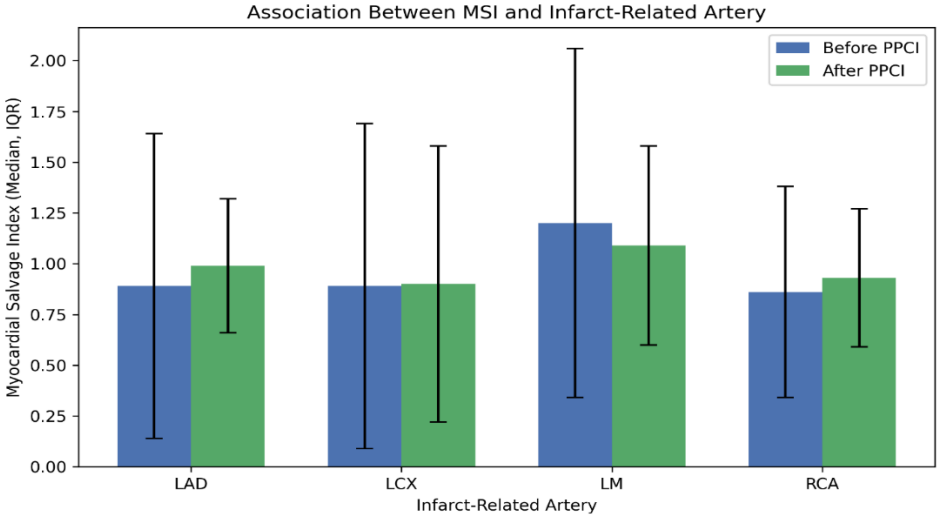

Figure 2. Comparison of Modified Shock Index (MSI) Across Coronary Artery Involvement Types (LAD, LCX, LM, and RCA) Before and After Primary PCI

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive accuracy of the myocardial salvage index (MSI) for major adverse cardiac events (MACE), as illustrated in Figure 3-4. The area under the curve (AUC) for MSI measured before primary percutaneous coronary intervention (PPCI) was 0.677 (95% CI: 0.606-0.747; p<0.001), indicating a fair discriminatory ability for predicting MACE. Similarly, MSI assessed after PPCI demonstrated an AUC of 0.631 (95% CI: 0.559-0.703; p<0.001), reflecting a modest but statistically significant predictive performance. The optimal cut-off value for pre-PPCI MSI was identified as 0.897, yielding a sensitivity of 64.7% and a specificity of 62.6%, while the optimal post-PPCI MSI threshold was 0.969, with a sensitivity of 62.7% and a specificity of 61.1%. Collectively, these findings suggest that

MSI, particularly when measured before PPCI, provides a moderate yet clinically meaningful ability to discriminate patients at higher risk of subsequent MACE, although its predictive performance remains limited and should be interpreted in conjunction with other clinical and imaging parameters (figure 3).

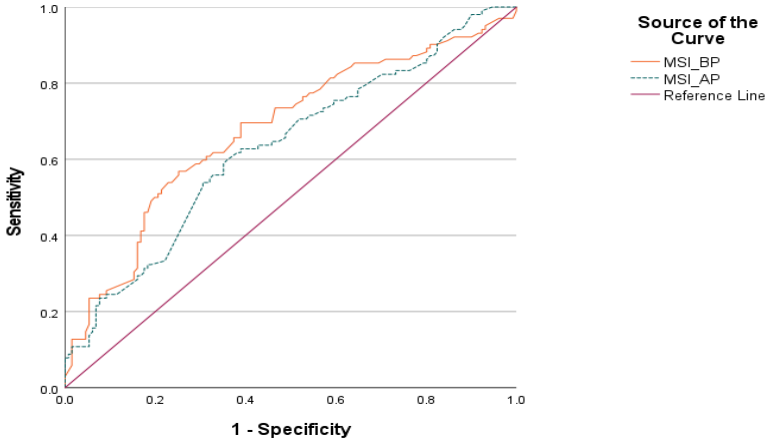

Figure 3. Receiver Operating Characteristic (ROC) Curves of Myocardial Salvage Index for Prediction of Major Adverse Cardiac Events

Discussion

The present study comprehensively evaluated the relationship between the Modified Shock Index and coronary anatomy, vessel involvement, and clinical outcomes in patients with STEMI undergoing primary PCI. Overall, MSI demonstrated meaningful associations with patient risk profiles and adverse outcomes but showed limited correlation with the anatomical extent or type of coronary vessel involvement. Higher MSI values were associated with older age, a greater burden of cardio metabolic risk factors, longer ischemic time, and laboratory markers of heightened systemic stress and myocardial injury. In contrast, MSI did not differ substantially across categories defined by the number of diseased vessels or the infarct-related artery. Importantly, MSI exhibited a moderate ability to predict major adverse cardiac events, particularly when assessed prior to reperfusion, supporting its role as a clinically useful marker of global hemodynamic compromise rather than anatomical disease severity (9,10).

The association between elevated MSI and adverse baseline characteristics observed in this study underscores the physiological relevance of MSI as an integrative marker of circulatory stress. MSI reflects the balance between heart rate and arterial pressure, thereby capturing early hemodynamic instability that may not yet manifest as overt shock. Older patients and those with hypertension, diabetes, and dyslipidemia are more likely to exhibit impaired vascular compliance, autonomic dysregulation, and reduced cardiovascular reserve, all of which may amplify MSI values during acute ischemic events. The clustering of these risk factors among patients with higher MSI suggests that MSI may serve as a surrogate marker for cumulative cardiovascular vulnerability rather than isolated hemodynamic derangement. This aligns with prior studies demonstrating that shock indices often correlate more strongly with systemic illness severity than with single disease characteristics (11,12).

The prolonged duration of chest pain prior to hospital presentation in patients with higher MSI provides further insight into the observed findings. Delayed reperfusion is known to exacerbate myocardial ischemia, promote infarct expansion, and intensify neurohormonal and inflammatory activation. As ischemic time increases, compensatory tachycardia and declining arterial pressure may progressively elevate MSI, even in the absence of frank cardiogenic shock. This temporal dimension suggests that MSI integrates both the intensity and duration of ischemic stress, offering an explanation for its association with worse laboratory profiles and clinical outcomes. Consequently, MSI may act as a dynamic reflection of ischemic burden rather than a static indicator of coronary anatomy (13,14).

The lack of a significant relationship between MSI and the number of diseased coronary vessels represents an important finding of this study. Although multivessel disease is traditionally associated with worse prognosis, the acute hemodynamic response during STEMI is often driven by the functional impact of the culprit lesion rather than the cumulative anatomical burden. Well-developed collateral circulation, lesion chronicity, and pre-existing myocardial adaptation may mitigate the immediate hemodynamic consequences of multivessel disease. Therefore, MSI appears insufficiently sensitive to discriminate anatomical complexity when assessed solely by vessel count, reinforcing the concept that

physiological indices and anatomical scores capture distinct dimensions of disease severity (15,16).

Similarly, the absence of significant differences in MSI across infarct-related arteries suggests that the anatomical territory of infarction alone does not dictate early hemodynamic status. While LAD or left main involvement is classically associated with larger infarct size, the acute circulatory response depends on multiple modifying factors, including infarct transmurality, myocardial preconditioning, and microvascular function. The homogeneity of MSI across LAD, LCX, LM, and RCA involvement observed in this study implies that MSI reflects global cardiovascular stress rather than regional myocardial damage. This finding supports emerging evidence that simple hemodynamic indices may not reliably mirror infarct localization or coronary anatomy (17,18).

In contrast to its limited anatomical discriminatory capacity, MSI demonstrated clinically relevant prognostic performance for predicting major adverse cardiac events. The moderate predictive accuracy observed, particularly for pre-PCI MSI, highlights the importance of early hemodynamic assessment prior to reperfusion. Before PCI, MSI likely captures the combined effects of ischemia, sympathetic activation, inflammatory response, and early ventricular dysfunction. Following reperfusion, partial normalization of hemodynamics and therapeutic interventions may attenuate these signals, explaining the comparatively reduced predictive strength of post-PCI MSI. These findings reinforce the value of admission MSI as an early risk stratification tool rather than a post-procedural marker (19,20).

The laboratory abnormalities associated with elevated MSI further substantiate its pathophysiological significance. Higher white blood cell counts and troponin levels suggest intensified inflammatory activation and greater myocardial injury, respectively, while elevated creatinine reflects transient renal hypo perfusion or cardio renal interaction. Notably, the absence of differences in hemoglobin and fasting glucose indicates that MSI is more closely linked to acute stress responses than baseline metabolic or oxygen-carrying capacity. This pattern supports the interpretation of MSI as a composite marker of systemic insult during STEMI rather than a reflection of chronic physiological status (21,22).

Taken together, the findings of this study suggest that while MSI does not correlate strongly with the number or type of involved coronary vessels, it provides meaningful prognostic information by capturing the global hemodynamic and inflammatory burden of acute myocardial infarction. Its simplicity, availability, and moderate predictive value make it an attractive adjunct to traditional risk stratification tools, particularly in resource-limited or time-sensitive settings. However, MSI should not be interpreted in isolation but rather integrated with clinical judgment, imaging findings, and established risk scores. Future studies incorporating larger populations and multimodal assessments may further clarify the role of MSI within comprehensive STEMI risk stratification frameworks (23,24).

Conclusion

In conclusion, this study demonstrates that the Modified Shock Index is closely associated with patient-level vulnerability and acute disease severity rather than the anatomical extent or distribution of coronary artery involvement in STEMI. Elevated MSI values reflect a convergence of advanced age, cardio metabolic risk burden, delayed presentation, and heightened inflammatory, myocardial, and renal stress, underscoring its role as an integrative marker of systemic and hemodynamic compromise. Although MSI did not discriminate between the number of diseased vessels or the infarct-related artery, its moderate ability to predict major adverse cardiac events particularly when assessed before reperfusion highlights its clinical utility in early risk stratification. These findings support the use of MSI as a simple, readily available adjunct to conventional clinical and imaging parameters for identifying higher-risk STEMI patients, while emphasizing that anatomical coronary complexity and physiological instability represent distinct and complementary dimensions of cardiovascular risk.

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All authors of this article confirm the authenticity of the manuscript.

Conflicts of interest

The authors declare that they have no competing interests.

Disclosure Statement

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Authors' Contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

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