Journal of Advanced in Medicinal, Pharmaceutical and Biomedical Research

Journal homepage: https://www.jampbr.com

DOI: 10.22034/jampbr.2026.572720.1068

Role of the Modified Shock Index in Predicting Long-Term Outcomes of Acute Myocardial Infarction Patients Undergoing Primary PCI

Kamran Mohammadi[1], Ahmad Separham[2*]

[1]Assistant Professor of Cardiology, Department of Cardiology, School of Medicine, Cardiovascular Research Center, Tabriz University of Medical Sciences, Tabriz, Iran

[2]Professor of Cardiology, Department of Cardiology, School of Medicine, Cardiovascular Research Center, Tabriz University of Medical Sciences, Tabriz, Iran

Article info
Received: 29.12.2025
Accepted: 04.02.2026
Available Online: 04.02.2026
Checked for Plagiarism: Yes

Keywords:
Modified Shock Index, Long Term Outcomes, Acute Myocardial Infarction, PCI

ABSTRACT

Introduction: Acute myocardial infarction remains a major cause of long-term morbidity and mortality despite advances in primary percutaneous coronary intervention. Early identification of high-risk patients is therefore essential. The aim of this study was to evaluate the role of the Modified Shock Index as a simple hemodynamic marker for predicting long-term clinical outcomes in patients with acute myocardial infarction undergoing primary PCI.

Material and methods: This cross-sectional study was conducted at Shahid Madani Heart Hospital between 2022 and 2023 using a census sampling method. A total of 234 patients with acute myocardial infarction undergoing primary percutaneous coronary intervention were included. Clinical, hemodynamic, laboratory, echocardiographic, and angiographic data were collected, and the Modified Shock Index was calculated before and after PCI for analysis.

Results: Patients with a higher Modified Shock Index (P≥0.897) were older and exhibited more hypertension (P=0.008), diabetes (P=0.002), and dyslipidemia (P=0.009), along with longer chest pain duration (P=0.004) and higher leukocyte count, troponin, and creatinine (all P≤0.002). In-hospital mortality was markedly greater in both high pre- and post-PPCI MSI groups (each P<0.001).

Conclusion: The present study demonstrates that the Modified Shock Index is a strong, readily obtainable prognostic indicator in patients with acute myocardial infarction undergoing primary percutaneous coronary intervention. An elevated MSI, reflecting the interplay between tachycardia and hypotension, accurately identified subjects with greater cardiovascular risk, prolonged ischemic burden, and biochemical evidence of systemic inflammation and myocardial damage.

Introduction

Acute myocardial infarction remains one of the leading causes of morbidity and mortality worldwide despite substantial advances in reperfusion strategies, pharmacological therapy, and secondary prevention. Primary percutaneous coronary intervention has become the preferred reperfusion modality for patients presenting with acute myocardial infarction, particularly ST-elevation myocardial infarction, due to its superiority in restoring coronary blood flow and reducing short-term mortality. Nevertheless, long-term outcomes following successful primary PCI vary considerably among patients, even in the absence of procedural complications or residual coronary obstruction. This heterogeneity highlights the importance of early risk stratification tools that extend beyond angiographic findings and procedural

Corresponding Author: **Ahmad Separham (A_separham@gmail.com - ORCID: 0000-0001-7011-9507)*
1 Email: Mohammadikamran706@gmail.com - ORCID: 0000-0002-1589-5359)

success to capture the underlying physiological and systemic response to acute ischemic injury (1).

Risk stratification in acute myocardial infarction has traditionally relied on a combination of clinical variables, electrocardiographic findings, laboratory markers, and imaging parameters. Established scoring systems such as the TIMI and GRACE scores have demonstrated utility in predicting short- and intermediate-term outcomes; however, their complexity and reliance on multiple variables may limit rapid bedside application in emergency settings. Furthermore, these scores were primarily developed to estimate early mortality and recurrent ischemic events rather than long-term outcomes following contemporary primary PCI. As a result, there has been growing interest in simpler, readily available indices that reflect hemodynamic instability and systemic compromise, which may provide incremental prognostic value throughout the disease course (2).

Hemodynamic deterioration during acute myocardial infarction reflects the interplay between myocardial dysfunction, autonomic imbalance, inflammatory activation, and end-organ hypo perfusion. Heart rate and blood pressure are among the earliest and most accessible clinical indicators of this pathophysiological cascade. The shock index, defined as the ratio of heart rate to systolic blood pressure, has been proposed as a marker of circulatory failure and has shown prognostic relevance in various critical care and cardiovascular settings. However, systolic blood pressure alone may not fully capture the perfusion pressure experienced by vital organs, particularly in patients with increased arterial stiffness or altered pulse pressure, prompting exploration of alternative hemodynamic indices (3).

The Modified Shock Index, calculated as the ratio of heart rate to mean arterial pressure, has emerged as a refined indicator of hemodynamic status that integrates both cardiac output and vascular tone more comprehensively than the traditional shock index. By incorporating mean arterial pressure, MSI may better reflect tissue perfusion and systemic circulatory adequacy during acute ischemic events. Prior studies have suggested that MSI is associated with short-term mortality, cardiogenic shock, and in-hospital adverse events in patients with acute coronary syndromes. These findings support the hypothesis that MSI captures a broader spectrum of physiological derangements than isolated vital signs or conventional risk factors (4).

Despite growing evidence supporting the short-term prognostic value of MSI, its role in predicting long-term outcomes following primary PCI remains less clearly defined. Long-term prognosis after acute myocardial infarction is influenced not only by infarct size and left ventricular function but also by systemic responses initiated during the acute phase, including inflammatory activation, neurohormonal dysregulation, and microvascular dysfunction. Hemodynamic instability at presentation may serve as an early surrogate for these processes, potentially linking acute circulatory compromise to chronic myocardial remodeling and progressive heart failure. Accordingly, indices such as MSI assessed at admission or early after reperfusion may hold prognostic significance well beyond the index hospitalization (5).

In the era of primary PCI, anatomical factors such as the extent of coronary artery disease, infarct-related artery location, and angiographic success have traditionally dominated risk assessment. However, emerging data suggest that anatomical severity alone does not fully explain variability in long-term outcomes among patients undergoing timely and technically successful revascularization. Patients with similar angiographic profiles may experience markedly different clinical trajectories, underscoring the influence of patient-specific physiological reserve and systemic vulnerability. MSI, as a composite marker derived from fundamental hemodynamic parameters, may help bridge the gap between anatomical assessment and functional risk evaluation (6).

Long-term adverse outcomes after acute myocardial infarction, including all-cause mortality, recurrent ischemic events, and heart failure hospitalization, impose a substantial burden on healthcare systems and patients alike. Identifying high-risk individuals early in the clinical course enables targeted surveillance, optimization of medical therapy, and closer follow-up after discharge. Importantly, an ideal prognostic marker should be simple, reproducible, inexpensive, and universally applicable across diverse clinical settings. MSI fulfills many of these criteria, as it can be calculated rapidly at the bedside without the need for laboratory testing or advanced imaging, making it particularly attractive for early risk stratification (7).

While several studies have evaluated the prognostic implications of MSI in emergency medicine and critical care populations, data specifically focusing on patients with acute myocardial infarction treated with primary PCI are still evolving. Existing studies often emphasize in-hospital or short-term outcomes, with limited attention to long-term follow-up. Moreover, variations in study design, timing of MSI assessment, and outcome definitions have contributed to inconsistent findings across the literature. These gaps underscore the need for focused investigations examining the relationship between MSI and long-term outcomes in well-characterized PCI-treated myocardial infarction cohorts (8).

Against this background, the present study was designed to evaluate the role of the Modified Shock Index in predicting long-term outcomes in patients with acute myocardial infarction undergoing primary percutaneous coronary intervention. By

examining the association between MSI and subsequent clinical outcomes beyond the acute phase, this study aims to clarify whether a simple hemodynamic index obtained early in the disease course can provide meaningful prognostic information in the contemporary PCI era. Establishing such a relationship may support the integration of MSI into routine clinical assessment and contribute to more individualized risk stratification strategies for patients with acute myocardial infarction.

Material and methods

Study Design and Setting

This cross-sectional study was conducted at Shahid Madani Heart Hospital, a tertiary cardiovascular referral center affiliated with Tabriz University of Medical Sciences, Iran. The study included patients admitted with a diagnosis of acute myocardial infarction who underwent primary percutaneous coronary intervention (PCI) between the beginning of 2018 and the end of 2019. A census sampling approach was employed, whereby all eligible patients treated during the study period were consecutively enrolled, yielding a final sample size of 234 participants.

Study Population

The study population consisted of adult patients (≥18 years) with a confirmed diagnosis of acute myocardial infarction based on clinical symptoms, electrocardiographic findings, and elevated cardiac biomarkers, who were treated with primary PCI as the initial reperfusion strategy. Patients were excluded if they died prior to coronary intervention, received thrombolytic therapy followed by rescue PCI, or had incomplete clinical or procedural data. Additional exclusion criteria included loss to one-year follow-up, pregnancy, advanced systemic diseases with limited survival expectancy (such as active malignancy), end-stage renal disease requiring dialysis, recent coronary artery bypass graft surgery, or a documented myocardial infarction within the previous 30 days.

Data Collection and Baseline Assessment

Baseline demographic information, cardiovascular risk factors, and clinical characteristics were extracted from medical records. Vital signs, including heart rate and systolic and diastolic blood pressure, were recorded at admission before coronary reperfusion using standardized measurement protocols. Mean arterial pressure was calculated accordingly. Laboratory data, including white blood cell count, serum creatinine, and cardiac troponin levels, were obtained during the initial phase of hospitalization. Left ventricular systolic function was evaluated using transthoracic echocardiography performed during the index admission.

Modified Shock Index Calculation

The Modified Shock Index (MSI) was defined as the ratio of heart rate to mean arterial pressure. MSI was calculated at two predefined time points: at hospital admission prior to primary PCI (pre-PCI MSI) and immediately after completion of the PCI procedure following hemodynamic stabilization (post-PCI MSI). These measurements were used to assess the relationship between hemodynamic status and long-term clinical outcomes.

Coronary Angiography and PCI Procedure

Coronary angiography and primary PCI were performed according to institutional protocols and contemporary clinical practice guidelines. Procedural data, including vascular access site, infarct-related artery, number of diseased coronary vessels, type of stent implanted, and post-procedural TIMI flow grade, were documented. Procedural success was defined as restoration of adequate coronary blood flow without the occurrence of major per procedural complications. Any procedural or in-hospital adverse events, including bleeding, vascular complications, or contrast-induced nephropathy, were recorded.

Follow-Up and Outcome Assessment

Patients were followed for a minimum of one year after the index PCI through outpatient clinic visits, structured telephone interviews, and review of hospital and registry records. The primary outcome was all-cause mortality at one year. Secondary outcomes included major adverse cardiac events (MACE), defined as a composite of cardiac death, recurrent myocardial infarction, repeat revascularization, or hospitalization due to heart failure. Outcome assessment was performed by investigators blinded to baseline MSI values to reduce potential bias.

Statistical Analysis

Statistical analyses were conducted using SPSS software version 23. Continuous variables were presented as mean ± standard deviation or median with interquartile range, depending on data distribution. Categorical variables were expressed as frequencies and percentages. The Kolmogorov Smirnov test was used to assess normality of continuous variables. Comparisons between groups were performed using the independent t-test or Mann–Whitney U test, as appropriate. Categorical variables were compared using the chi-square test. Graphical analyses and one-way analysis of variance were conducted using Graph Pad Prism version 9. A two-sided p-value <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was approved by the Ethics Committee of Tabriz University of Medical Sciences (approval code: IR.TBZMED.REC.1400.583). All procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants, and patient confidentiality was strictly maintained throughout the study. This research was performed as part of an approved academic thesis.

Results

Patients with a higher Modified Shock Index (≥0.897) were significantly older and demonstrated a greater burden of cardiovascular risk factors, including hypertension, diabetes, and dyslipidemia. This group also experienced longer chest pain duration and showed higher inflammatory and myocardial injury markers, as evidenced by elevated leukocyte count, troponin levels, and serum creatinine, suggesting more severe systemic and cardiac involvement at presentation (table 1).

Table 1. Comparison of Demographic Characteristics, Cardiovascular Risk Factors, and Laboratory Findings Between Low and High MSI Groups

Characteristic	MSI < 0.897 (n = 118)	MSI ≥ 0.897 (n = 115)	*P* value
Age (years)	57	61	0.002
Male sex, n (%)	90 (75.0)	86 (76.4)	0.123
Current smoker, n (%)	45 (38.1)	44 (38.0)	0.172
Cardiovascular risk factors			
Hypertension, n (%)	76 (64.0)	63 (56.5)	0.008
Diabetes mellitus, n (%)	23 (19.4)	25 (21.7)	0.002
Dyslipidemia, n (%)	21 (17.7)	12 (10.4)	0.009
Family history of CAD, n (%)	12 (10.1)	10 (8.6)	0.541
Chest pain duration (hours)	7.56	9.10	0.004
Laboratory findings			
Fasting blood glucose (mg/dL)	82	88	0.717
Hemoglobin (g/dL)	14.24	14.10	0.123
White blood cell count (/µL)	6,789	7,552	0.001
Cardiac troponin (ng/L)	17.12	24.83	0.002
Serum creatinine (mmol/L)	1.11	1.30	0.001

Among 102 patients who experienced major adverse cardiovascular events, in-hospital mortality constituted the most frequent outcome (61.7%), followed by out-of-hospital deaths from any cause (16.6%) and cardiovascular deaths (14.7%). Other complications, including coronary restenosis (14.7%), re-myocardial infarction (2.9%), heart failure (1.9%), and cerebrovascular accident (1.9%), occurred less frequently. The overall distribution of event types showed a statistically significant difference (χ^2 test, P=0.001), reflecting that fatal outcomes particularly in-hospital mortality represented the predominant component of adverse events in this cohort (figure 1).

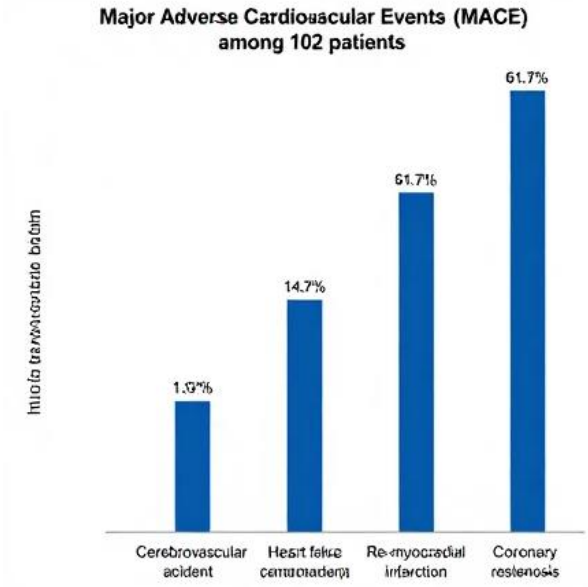

Figure 1. Distribution of Major Adverse Cardiovascular Events (MACE) During One-Year Follow-Up

As shown in Figure 2, the only component of major adverse cardiovascular events (MACE) for which the mean Modified Shock Index (MSI) differed significantly between patients with and without the event was in-hospital mortality from any cause ($p < 0.001$).

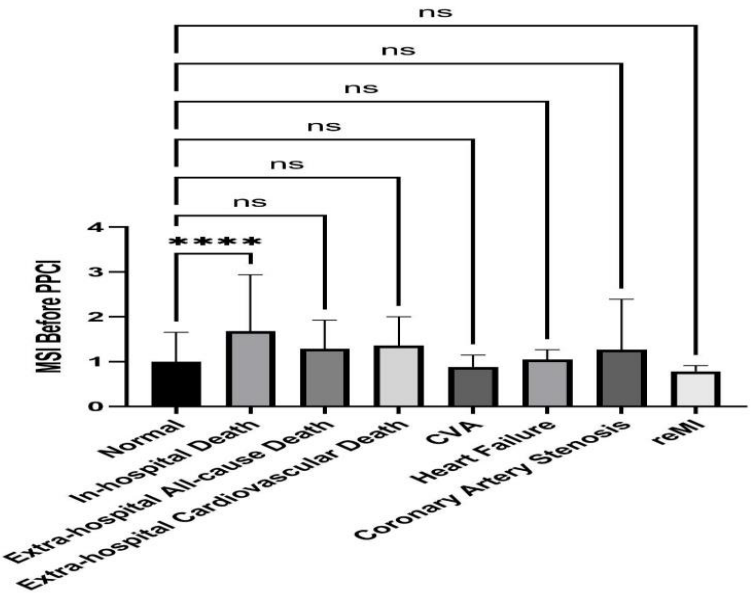

Figure 2. Association Between Mean Modified Shock Index and in-Hospital Mortality

As illustrated in Figure 3, the only MACE outcomes demonstrating a significant difference in mean MSI values between groups with and without the event were in-hospital deaths from any cause, respectively ($p < 0.001$).

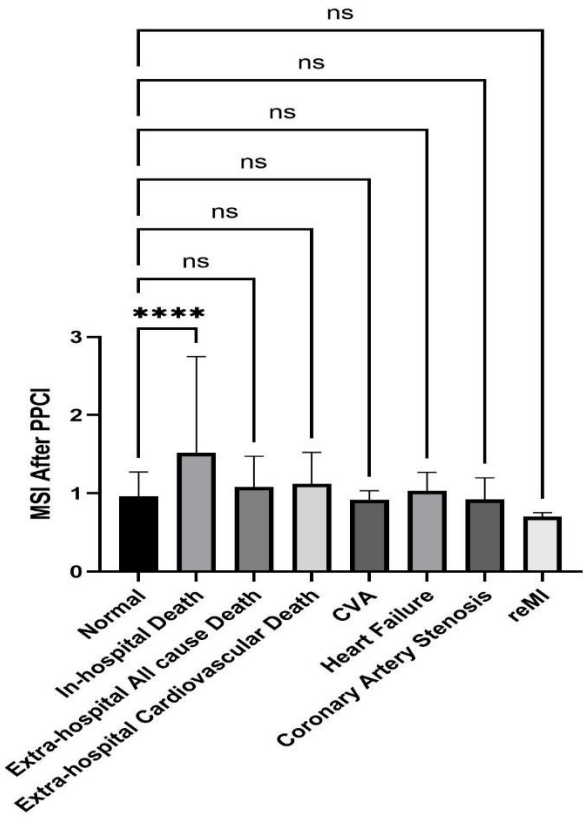

Figure 3. Relationship Between Mean Modified Shock Index and Components of MACE

As shown in Table 7-4, in-hospital mortality from any cause occurred significantly more frequently among patients with a high pre-PPCI Modified Shock Index compared with those with a low MSI ($P < 0.001$). Although out-of-hospital cardiovascular death tended to be more common in

the high-MSI group, this association did not reach statistical significance (P=0.055). No significant differences were observed between MSI groups for other MACE components, including non-cardiovascular death, cerebrovascular accident, heart failure, re-myocardial infarction, or coronary restenosis (P > 0.05 for all), indicating that pre-PPCI MSI was primarily associated with early in-hospital mortality rather than non-fatal or late adverse events (table 2).

Table 2. Frequency of Major Adverse Cardiovascular Events According to Pre-PPCI Modified Shock Index

MACE Component	Pre-PPCI MSI Low (n = 118)	Pre-PPCI MSI High (n = 115)	Total (n = 233)	*P* value
In-hospital death (any cause)	15 (23.8%)	48 (76.2%)	63 (100%)	<0.001
Out-of-hospital cardiovascular death	4 (26.7%)	11 (73.3%)	15 (100%)	0.055
Out-of-hospital non-cardiovascular death	2 (100%)	0 (0%)	2 (100%)	0.498
Cerebrovascular accident (CVA)	1 (50.0%)	1 (50.0%)	2 (100%)	1.000
Heart failure	1 (50.0%)	1 (50.0%)	2 (100%)	1.000
Re-myocardial infarction (re-MI)	3 (100%)	0 (0%)	3 (100%)	0.247
Coronary restenosis	10 (66.7%)	5 (33.3%)	15 (100%)	0.199

As demonstrated in Table 8-4, in-hospital mortality from any cause was significantly more frequent among patients with a high post-PPCI Modified Shock Index compared with those with a low MSI (P < 0.001). Although out-of-hospital cardiovascular deaths occurred more commonly in the high-MSI group, this difference did not reach statistical significance (*P* = 0.166). No significant associations were observed between post-PPCI MSI status and other components of MACE, including non-cardiovascular mortality, cerebrovascular accident, heart failure, re-myocardial infarction, or coronary restenosis (P > 0.05 for all), indicating that post-procedural MSI is predominantly linked to early in-hospital mortality rather than late or non-fatal adverse outcomes (table 3).

Table 3. Frequency of Major Adverse Cardiovascular Events According to Post-PPCI Modified Shock Index

MACE Component	Post-PPCI MSI Low (n = 118)	Post-PPCI MSI High (n = 115)	Total (n = 233)	*P* value
In-hospital death (any cause)	17 (27.0%)	46 (73.0%)	63 (100%)	<0.001
Out-of-hospital cardiovascular death	5 (33.3%)	10 (66.7%)	15 (100%)	0.166
Out-of-hospital non-cardiovascular death	2 (100%)	0 (0%)	2 (100%)	0.498
Cerebrovascular accident (CVA)	1 (50.0%)	1 (50.0%)	2 (100%)	1.000
Heart failure	1 (50.0%)	1 (50.0%)	2 (100%)	1.000
Re-myocardial infarction (re-MI)	3 (100%)	0 (0%)	3 (100%)	0.247
Coronary restenosis	9 (60.0%)	6 (40.0%)	15 (100%)	0.454

Discussion

The findings of the present study highlight the prognostic utility of the Modified Shock Index (MSI) in patients presenting with acute myocardial infarction (AMI) who undergo primary percutaneous coronary intervention (PCI). A higher MSI at the time of presentation and after revascularization clearly identified a subgroup of patients with more adverse clinical profiles and poorer outcomes during long-term follow-up. These individuals were generally older, carried a heavier burden of cardiovascular comorbidities, and demonstrated prolonged ischemic symptoms accompanied by significant laboratory evidence of systemic inflammation and myocardial injury. In addition, a strong association between elevated MSI and in-hospital mortality emphasizes the sensitivity of this index in detecting high-risk hemodynamic states. Overall, these results underscore the value of MSI as a simple yet powerful bedside marker, integrating both circulatory compromise and systemic stress to predict adverse prognosis in AMI patients treated with PCI (9,10).

The observation that patients with higher MSI values tended to be older and had a greater prevalence of hypertension, diabetes mellitus, and dyslipidemia aligns with the understanding that chronic exposure to cardiovascular risk factors leads to diminished vascular elasticity and impaired autonomic control. Age-related alterations in

baroreceptor sensitivity and arterial compliance result in exaggerated increases in heart rate and disproportionate falls in blood pressure during episodes of acute ischemia. Hypertensive patients often exhibit myocardial hypertrophy and reduced diastolic function, while those with diabetes and dyslipidemia experience endothelial dysfunction and microvascular disease, further compromising cardiac perfusion during stress. Because MSI incorporates both systolic blood pressure and heart rate, it integrates these physiological deteriorations into a single numerical expression, effectively capturing the cumulative hemodynamic burden of years of vascular injury and metabolic imbalance (11,12).

Longer chest pain duration among patients with elevated MSI points to a delay in seeking medical assistance or slower recognition of ischemic symptoms. A prolonged ischemic interval before reperfusion typically results in larger infarct size, more extensive necrosis, and a greater degree of myocardial stunning. These pathophysiological processes increase sympathetic output and trigger inflammatory cascades, leading to tachycardia, hypotension, and dynamic fluctuations in systemic vascular resistance the very parameters reflected by MSI. A high MSI under these conditions therefore functions as a marker of both delayed intervention and advanced ischemic injury. From a path biological perspective, extended oxygen deprivation promotes cytokine release and excess circulating catecholamines, which intensify metabolic strain and further destabilize hemodynamic equilibrium. This link between ischemic time and systemic disturbance helps explain why patients with high MSI experienced longer symptom duration and more pronounced metabolic derangements (13,14).

The elevation of leukocyte count, troponin, and serum creatinine in the high-MSI group further supports the hypothesis that MSI represents a broad index of systemic and myocardial stress. Leukocytosis mirrors an acute inflammatory reaction, consistent with endothelial activation and tissue injury following infarction. Troponin, as a direct biomarker of cardiac cell death, reflects the extent of myocardial necrosis, while an increase in creatinine may indicate renal hypo perfusion secondary to systemic hypotension. Together, these findings reveal that patients with high MSI possess a greater degree of multiorgan involvement, combining cardiac, renal, and inflammatory components into a common spectrum of physiological compromise. This multisystem derangement is a plausible explanation for their worse outcomes, as organ cross-talk between the failing heart and peripheral organs amplifies mortality risk even after mechanical reperfusion (15,16).

The overall composition of major adverse cardiovascular events demonstrated that fatal outcomes dominate the clinical course of these patients. In-hospital death constituted the majority of MACE incidents, followed by out-of-hospital deaths, whereas non-fatal events such as heart failure, recurrent infarction, or cerebrovascular accident were relatively uncommon. This distribution underscores that the primary determinant of poor prognosis in AMI is acute hemodynamic instability rather than delayed mechanical complications. It is plausible that the initial systemic insult, captured by a high MSI, exceeds the threshold of physiological compensation even after successful PCI. Severe myocardial dysfunction or persistent systemic inflammation may drive rapid deterioration despite revascularization. The emphasis on fatal rather than non-fatal events reinforces the clinical importance of early identification of high-risk patients based on MSI prior to intervention (17,18).

The finding that only in-hospital mortality exhibited a statistically significant relationship with MSI both before and after PCI highlights the index's sensitivity to short-term, acute phase risk. In-hospital deaths generally arise from cardiogenic shock, arrhythmias, or acute mechanical complications, all of which reflect profound circulatory failure. MSI, calculated from the simple ratio of heart rate to systolic blood pressure, dynamically captures these conditions by indexing the relative dominance of sympathetic stimulation over vascular tone. Conversely, late complications such as restenosis or recurrent infarction are more influenced by procedural factors, medication adherence, or biological healing rather than acute hemodynamic stress. Therefore, the absence of a clear link between MSI and long-term non-fatal outcomes confirms that this index is a robust predictor of immediate critical deterioration rather than subsequent chronic problems (19,20).

When comparing pre-procedural and post-procedural MSI, both phases demonstrated the same pattern: patients with higher MSI experienced substantially more frequent fatal outcomes. This consistency implies that the physiological information encapsulated by MSI is persistent across the clinical timeline of AMI. Pre-PCI values likely reflect the extent of hemodynamic collapse upon hospital arrival, whereas post-PCI values may represent residual instability or incomplete recovery following reperfusion. A persistently high MSI after PCI could indicate inadequate myocardial salvage, residual ischemic dysfunction, or ongoing systemic inflammatory activation. The pathophysiological mechanisms linking post-intervention MSI to mortality might involve reperfusion injury, arrhythmic storms, or persistent left ventricular dysfunction, each contributing to sustained low cardiac output and hypotension. Hence, serial MSI assessment can help clinicians identify patients who

remain critically unstable even after mechanical reperfusion (21,22).

Several lines of reasoning explain why MSI remains predictive despite successful PCI. First, reperfusion therapy corrects the mechanical obstruction of coronary flow but does not immediately restore microvascular integrity or correct systemic inflammatory and neurohumoral activation. Patients with higher MSI likely experience a multidimensional stress response that extends beyond coronary occlusion alone. Second, recovery of blood pressure and heart rate after PCI depends on myocardial contractility and autonomic equilibrium; both may remain impaired in severe infarctions. Third, high MSI values can indicate diminished cardiac reserve or secondary organ hypo perfusion, independent of angiographic success. These complex physiologic interactions highlight the index's utility in bridging procedural assessment with systemic clinical reality. MSI, thus, serves as an integrative physiological marker rather than a mere surrogate for successful intervention (23,24).

Clinically, the practicality of MSI lies in its simplicity and accessibility. Computation requires only basic vital signs parameters easily obtained in any emergency setting yet provides insight comparable to more complex scoring systems. Its correlation with mortality risk offers emergency physicians and interventional cardiologists an immediate gauge of physiological severity without relying on imaging or laboratory results. In resource-limited environments, this simplicity becomes particularly beneficial, allowing rapid risk stratification and prioritization for intensive monitoring. Integrating MSI into triage and early management protocols could improve the identification of high-risk STEMI patients and optimize the allocation of critical care resources (25,26).

From a pathophysiological standpoint, the Modified Shock Index embodies the ratio between compensatory cardiac acceleration and systemic circulatory failure. When the heart rate increases disproportionately to systolic pressure, the body is failing to maintain adequate perfusion. This imbalance predicts poor tissue oxygenation, acidosis, and subsequent multiorgan dysfunction all hallmarks of impending shock. The observation that MSI correlated strongly with both inflammatory markers and renal function further supports this interpretation. High MSI at presentation can thus be considered an early warning sign of cardiovascular exhaustion, signaling the need for aggressive hemodynamic support and precise reperfusion strategies. In this regard, MSI complements traditional biomarkers, adding a real-time physiological dimension to risk assessment (27,28).

Explanations for why non-fatal MACE components did not differ between groups may involve the multifactorial nature of complications such as restenosis or recurrent infarction. These outcomes depend largely on stent characteristics, pharmacologic management, and metabolic factors rather than early hemodynamic status. While MSI effectively captures acute hemodynamic collapse, it does not directly reflect endothelial regeneration or antiplatelet response, which govern these longer-term events. Therefore, lack of association does not undermine MSI's prognostic value; rather, it delineates the temporal specificity of its predictive capacity, pinpointing the critical window in which physiological instability most affects survival (29,30).

The study's implications extend to the follow-up phase of AMI management. Patients showing persistently high MSI after PCI may require tailored interventions beyond standard reperfusion protocols, including closer monitoring for arrhythmias, reassessment of ventricular function, and optimization of fluid balance. Incorporating MSI trends into discharge planning could guide post-hospital risk stratification and inform the intensity of outpatient surveillance. Future investigations might explore integrating MSI with other dynamic indices such as lactate levels or non-invasive cardiac output monitoring to refine prognostic modeling and enhance predictive accuracy (31,32).

In interpreting these findings, it is essential to recognize that MSI quantifies a purely hemodynamic facet of cardiac dysfunction. Its elevation results from a combination of tachycardia and hypotension, which may stem from autonomic perturbation, myocardial pump failure, or systemic inflammation. The ability of MSI to detect high-risk states lies in its responsiveness to these mechanisms, each contributing to circulatory insufficiency. Therefore, MSI may perform optimally when interpreted alongside clinical context patient age, comorbidities, infarct location, and biochemical markers. Its simplicity should not obscure the complexity it reflects: a dynamic interplay between cardiac output, peripheral resistance, and neurohormonal activation (33,34).

Finally, the consistent association between MSI and mortality reinforces its potential as an independent prognostic tool in acute coronary care. Despite its simplicity, MSI encapsulates the essence of cardiovascular instability in real time. Using MSI during initial evaluation and post-PCI assessment can refine treatment priorities and inform post-procedural management strategies. By enabling clinicians to rapidly identify patients at heightened risk of death, the Modified Shock Index bridges the gap between physiological insight and practical bedside decision-making. Its prognostic power, validated across diverse parameters, underlines its importance not only as a predictive measure but also as a guiding principle for early intervention in modern cardiac emergency practice (35,36).

Conclusion

The present study demonstrates that the Modified Shock Index is a strong, readily obtainable prognostic indicator in patients with acute myocardial infarction undergoing primary percutaneous coronary intervention. An elevated MSI, reflecting the interplay between tachycardia and hypotension, accurately identified subjects with greater cardiovascular risk, prolonged ischemic burden, and biochemical evidence of systemic inflammation and myocardial damage. Notably, both pre- and post-procedural MSI were independently associated with in-hospital mortality, underscoring its value as a marker of early hemodynamic instability and systemic compromise. The fact that MSI retained prognostic significance despite angiographic reperfusion success supports its interpretation as a global physiological index rather than a purely cardiac measure. Because its calculation requires only basic vital signs, MSI provides clinicians with a rapid, cost-free method to stratify risk and to guide immediate management decisions in the acute phase of myocardial infarction. Incorporating serial MSI assessment into routine PCI workflows could enhance the early identification of high-risk patients, optimize allocation of critical care resources, and inform post-discharge surveillance strategies. Future multicenter studies with larger cohorts and longer follow-up are warranted to validate optimal MSI thresholds and to explore its integration with established prognostic scoring systems for a more comprehensive assessment of cardiovascular risk.

Disclosure Statement

No potential conflict of interest reported by the authors.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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.

References

[1] GBD 2017 Causes of Death Collaborators. (**2018**). Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: A systematic analysis for the Global Burden of Disease Study 2017. *The Lancet, 392*(10159), 1736–1788.

[2] Liu, S., Li, Y., Zeng, X., Wang, H., Yin, P., Wang, L., Zhou, M. (**2019**). Burden of cardiovascular diseases in China, 1990–2016: Findings from the 2016 Global Burden of Disease study. *JAMA Cardiology, 4*(4), 342–352.

[3] Ibanez, B., James, S., Agewall, S., Antunes, M. J., Bucciarelli-Ducci, C., Bueno, H., … Widimský, P. (**2018**). 2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *European Heart Journal, 39*(2), 119–177.

[4] Jneid, H., Addison, D., Bhatt, D. L., Fonarow, G. C., Gokak, S., Grady, K. L., … Anderson, J. L. (**2017**). 2017 AHA/ACC clinical performance and quality measures for adults with ST-elevation and non–ST-elevation myocardial infarction. *Journal of the American College of Cardiology, 70*(16), 2048–2090.

[5] Szummer, K., Wallentin, L., Lindhagen, L., Alfredsson, J., Erlinge, D., Held, C., … Jernberg, T. (**2017**). Improved outcomes in patients with ST-elevation myocardial infarction during the last 20 years are related to implementation of evidence-based treatments. *European Heart Journal, 38*(41), 3056–3065.

[6] Fan, F., Li, Y., Zhang, Y., Zhao, D., Liu, J., Liu, J., … Wang, Y. (**2019**). Chest pain center accreditation is associated with improved in-hospital outcomes of acute myocardial infarction patients in China. *Journal of the American Heart Association, 8*(21), e013384.

[7] Kostis, W. J., Deng, Y., Pantazopoulos, J. S., Moreyra, A. E., & Kostis, J. B. (**2010**). Trends in mortality of acute myocardial infarction after discharge from the hospital. *Circulation: Cardiovascular Quality and Outcomes, 3*(6), 581–589.

[8] Huckfeldt, P., Escarce, J. J., Sood, N., Popescu, I., & Nuckols, T. K. (**2019**). Thirty-day postdischarge mortality among Black and White patients 65 years and older in the Medicare Hospital Readmissions Reduction Program. *JAMA Network Open, 2*(3), e190634.

[9] Khera, R., Dharmarajan, K., Wang, Y., Lin, Z., Ross, J. S., Horwitz, L. I., … Krumholz, H. M. (**2018**). Association of the Hospital Readmissions Reduction Program with mortality during and after hospitalization for acute myocardial infarction, heart failure, and pneumonia. *JAMA Network Open, 1*(5), e182777.

[10] Rosselló, X., Huo, Y., Pocock, S., Van de Werf, F., Chin, C. T., Danchin, N., … Fox, K. A. A. (**2017**). Global geographical variations in ST-segment elevation myocardial infarction management and post-discharge mortality. *International Journal of Cardiology, 245*, 27–34.

[11] Fox, K. A. A., Dabbous, O. H., Goldberg, R. J., Pieper, K. S., Eagle, K. A., Van de Werf, F., … Anderson, F. A. (**2006**). Prediction of risk of death and myocardial infarction in the six months after presentation with acute coronary syndrome: Prospective multinational observational study (GRACE). *BMJ, 333*(7578), 1091.

[12] Morrow, D. A., Antman, E. M., Charlesworth, A., Cairns, R., Murphy, S. A., de Lemos, J. A., … Braunwald, E. (**2000**). TIMI risk score for ST-elevation myocardial infarction: A convenient, bedside, clinical score for risk assessment at presentation. *Circulation, 102*(17), 2031–2037.

[13] Chow, C. K., Teo, K. K., Rangarajan, S., Islam, S., Gupta, R., Avezum, A., … Yusuf, S. (**2013**). Prevalence, awareness, treatment, and control of hypertension in rural and urban communities in high-, middle-, and low-income countries. *JAMA, 310*(9), 959–968.

[14] Yu, T., Tian, C., Song, J., He, D., Sun, Z., & Sun, Y. (**2017**). Age shock index is superior to shock index and modified shock index for predicting long-term prognosis in acute myocardial infarction. *Shock, 48*(5), 545–550.

[15] Zhou, J., Shan, P. R., Xie, Q. L., Wang, J., Zhang, X., & Xu, G. (**2019**). Age shock index and age-modified shock index are strong predictors of outcomes in ST-segment elevation myocardial infarction patients undergoing emergency percutaneous coronary intervention. *Coronary Artery Disease, 30*(6), 398–405.

[16] Zhao, L., Du, Z., Wu, T., Chen, Y., Zhang, Y., & Jin, Q. (**2021**). Association of the age shock index with coronary plaque characteristics in ST-segment elevation myocardial infarction: A three-vessel optical coherence tomography study. *Catheterization and Cardiovascular Interventions, 97*(Suppl. 2), 1080–1088.

[17] Li, J., Li, X., Wang, Q., Hu, S., Wang, Y., Masoudi, F. A., … Krumholz, H. M. (**2015**). ST-segment elevation myocardial infarction in China from 2001 to 2011 (the China PEACE–Retrospective Acute Myocardial Infarction study): A retrospective analysis of hospital data. *The Lancet, 385*(9966), 441–451.

[18] Zhang, Y., Wang, S., Yang, S., Wang, Z., Wang, X., & Zhang, X. (**2019**). Rationale and design of the Henan ST elevation myocardial infarction (STEMI) registry: A regional STEMI project in predominantly rural central China. *BMC Cardiovascular Disorders, 19*(1), 271.

[19] Zhang, Y., Wang, S., Cheng, Q., Wang, Z., Wang, X., & Zhang, X. (**2021**). Reperfusion strategy and in-hospital outcomes for ST elevation myocardial infarction in secondary and tertiary hospitals in predominantly rural central China. *BMJ Open, 11*(12), e053510.

[20] Chinese Society of Cardiology of Chinese Medical Association, & Editorial Board of Chinese Journal of Cardiology. (**2019**). 2019 Chinese Society of Cardiology (CSC) guidelines for the diagnosis and management of patients with ST-segment elevation myocardial infarction. *Zhonghua Xin Xue Guan Bing Za Zhi, 47*, 766–783.

[21] DeLong, E. R., DeLong, D. M., & Clarke-Pearson, D. L. (**1988**). Comparing the areas under two or more correlated receiver operating characteristic curves: A nonparametric approach. *Biometrics, 44*(3), 837–845.

[22] Pencina, M. J., D'Agostino, R. B., Sr., D'Agostino, R. B., Jr., & Vasan, R. S. (**2008**). Evaluating the added predictive ability of a new marker: From area under the ROC curve to reclassification and beyond. *Statistics in Medicine, 27*(2), 157–172.

[23] Van Calster, B., Wynants, L., Verbeek, J. F. M., Verbakel, J. Y., Christodoulou, E., Vickers, A. J., & Steyerberg, E. W. (**2018**). Reporting and interpreting decision curve analysis: A guide for investigators. *European Urology, 74*(6), 796–804.

[24] Chaudhry, S. I., Khan, R. F., Chen, J., Dharmarajan, K., Dodson, J. A., Masoudi, F. A., … Krumholz, H. M. (**2014**). National trends in recurrent AMI hospitalizations 1 year after acute myocardial infarction in Medicare beneficiaries, 1999–2010. *Journal of the American Heart Association, 3*(5), e001197.

[25] Yamamoto, T., Yoshida, N., Takayama, M., Tokyo CCU Network Scientific Committee, & Investigators. (**2019**). Temporal trends in acute myocardial infarction incidence and mortality between 2006 and 2016 in Tokyo: Report from the Tokyo CCU Network. *Circulation Journal, 83*(6), 1405–1409.

[26] Song, J., Mariyah, K., Hu, S., Wang, Y., Liu, J., Li, J., Zhao, D. (**2021**). Incidence, predictors, and prognostic impact of recurrent acute myocardial infarction in China. *Heart, 107*(4), 313–318.

[27] Shang, P., Liu, G. G., Zheng, X., Ho, P. M., Hu, S., Li, J., Zhao, D. (**2019**). Association between medication adherence and 1-year major cardiovascular adverse events after acute myocardial infarction in China. *Journal of the American Heart Association, 8*(9), e011793.

[28] Chow, C. K., Brieger, D., Ryan, M., Kangaharan, N., Gulati, M., & Briffa, T. G. (**2019**). Secondary prevention therapies in acute coronary syndrome and relation to outcomes: Observational study. *Heart Asia, 11*(1), e011122.

[29] Bordejevic, D. A., Caruntu, F., Mornos, C., Tomescu, M. C., & Andercou, O. (**2017**). Prognostic impact of blood pressure and heart rate at admission on in-hospital mortality after primary percutaneous intervention for acute myocardial infarction with ST-segment elevation in Western Romania. *Therapeutics and Clinical Risk Management, 13*, 1061–1068.

[30] Shen, J., Liu, G., Yang, Y., Li, J., Wang, Y., & Hu, S. (**2021**). Prognostic impact of mean heart rate by Holter monitoring on long-term outcome in patients with ST-segment elevation myocardial infarction undergoing percutaneous coronary intervention. *Clinical Research in Cardiology, 110*(9), 1439–1449.

[31] Schmitz, T., Harmel, E., Linseisen, J., Heuschmann, P. U., & Kääb, S. (**2022**). Shock index

and modified shock index are predictors of long-term mortality not only in STEMI but also in NSTEMI patients. *Annals of Medicine, 54*(1), 900–908.

[32] Chiang, C. Y., Lin, C. F., Liu, P. H., Huang, C. C., Chan, W. L., & Chen, J. Y. (**2022**). Clinical validation of the shock index, modified shock index, delta shock index, and shock index-C for emergency department ST-segment elevation myocardial infarction. *Journal of Clinical Medicine, 11*(19), 5839.

[33] Pramudyo, M., Marindani, V., Achmad, C., Panggabean, M. M., & Raharjo, S. B. (**2022**). Modified shock index as simple clinical independent predictor of in-hospital mortality in acute coronary syndrome patients: A retrospective cohort study. *Frontiers in Cardiovascular Medicine, 9*, 915881.

[34] Swaminathan, R. V., Rao, S. V., McCoy, L. A., Kim, L. K., Minutello, R. M., Wong, S. C., … Feldman, D. N. (**2015**). Hospital length of stay and clinical outcomes in older STEMI patients after primary PCI. *Journal of the American College of Cardiology, 65*(12), 1161–1171.

[35] Lv, J., Wang, C., Gao, X., Li, J., Wang, Y., & Hu, S. (**2023**). Development and validation of dynamic models to predict post-discharge mortality risk in patients with acute myocardial infarction: Results from China Acute Myocardial Infarction Registry. *BMJ Open, 13*(3), e069505.

[36] Shiraishi, J., Kohno, Y., Nakamura, T., Arihara, M., Hadase, M., Hyogo, M., & Matsubara, H. (**2014**). Predictors of in-hospital outcomes after primary percutaneous coronary intervention for acute myocardial infarction in patients with a high Killip class. *Internal Medicine, 53*(9), 933–939.