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ORIGINAL RESEARCH

Predictive Value of Systemic Immune-Inflammation Index Combined with D-Dimer Levels and Injury Severity Score for the Prognosis of Patients with Multiple Injuries: A Retrospective Analysis

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Background: The prognosis of patients with multiple injuries (MI) remains challenging to predict accurately due to the complexity and heterogeneity of trauma. Traditional scoring systems or single biomarkers often have limitations in terms of sensitivity or specificity.

Purpose: The objective of the present study was to investigate the predictive value of combining the systemic immune-inflammation index (SII), D-dimer (D-D) and injury severity score (ISS) for evaluating the prognosis of patients with MI.

Patients and Methods: A total of 142 patients with MI admitted to the Emergency Department of Shenzhen Longhua District Central Hospital (Shenzhen, China) from January 2019 to December 2023 were retrospectively analyzed. Patients were then divided into survival (n=102) and non-survival (n=40) groups according to their survival status on postoperative day 28. Logistic regression analysis was performed on indicators with significant differences between the two groups to identify prognostic factors in severe MI. Data were retrospectively analyzed using SPSS 26.0 software. In addition, receiver operating characteristic (ROC) curves were plotted to assess the predictive value of the combined SII, D-D and ISS.

Results: The median SII was found to be 340.11 (182.72–578.31) in the survival group and 849.93 (300.83–1034.14) in the non-survival group, yielding a significant difference (P<0.001). Logistic regression indicated that SII, D-D and ISS at admission were independent prognostic factors for mortality. Furthermore, SII, D-D, ISS and their combination showed predictive value [area under the curve (AUC) >0.5] for 28-day mortality, with the combination exhibiting the highest predictive accuracy. Based on the optimal ROC cut-off values (308.8), patients were divided into groups with SII ≤308.8 (n=58) and SII >308.8 (n=84), with significantly lower mortality in the former group (P<0.05). Similarly, patients were categorized into D-D ≤2.35 (n=117) and D-D >2.35 (n=25) groups, with significantly lower mortality in the former group (P<0.05). In conclusion, each indicator demonstrated prognostic significance (AUC >0.5), with the combined model achieving the highest predictive accuracy (AUC =0.969).

Conclusion: The integrated indicators offered improved predictive performance compared with individual measures for MI, providing a clinically valuable reference for patient management.

Keywords: multiple injuries, systemic immune-inflammation index, D-dimer, injury severity score, prognosis

Introduction

Trauma is recognized as a major public health issue worldwide,[1] with traffic accidents being the leading cause of mortality among individuals aged 15–29 years globally, accounting for ~1.3 million cases of mortality and 50 million

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injuries annually.[2] Multiple injuries (MI), defined as serious injuries involving two or more anatomical locations or organs caused by a single event,[3] is characterized by rapid clinical deterioration, high fatality rates and increased susceptibility to infection compared with single-system injuries. MI can trigger systemic inflammatory response (SIR) syndrome (SIRS) due to the marked release of inflammatory factors following the activation of the SIR.[4] Prognostic assessment using simple and effective clinical indicators during early hospitalization, combined with timely interventions, can notably improve survival rates in patients with severe MI.[5]

Although traditional tools, such as the injury severity score (ISS), a scoring system that quantifies overall trauma severity by summing the squares of the three highest Abbreviated Injury Scale (AIS) grades from three different body regions,[6] can be used to assess anatomical injury severity, they lack the capability to dynamically monitor immune-inflammatory reactions, which represent a key pathological link influencing prognosis.[7] Given the strong association between complex immune-inflammatory responses and patient outcomes,[8] identifying reliable indices that can quantify this pathophysiological process has become a notable research objective. Recently, the systemic immune-inflammation index (SII), integrating neutrophil, lymphocyte and platelet counts, has demonstrated unique value in injury-related immune monitoring.[9] Proposed by Hu et al,[10] the SII is calculated based on peripheral neutrophil (N), platelet (P) and lymphocyte (L) counts, using the formula (P x N)/L The SII serves as a comprehensive indicator of systemic immune-inflammatory status and can be utilized to reflect the clinical progression or deterioration through the collective action of multiple immune-inflammatory cells.[11] It demonstrates superior predictive capability for trauma-related mortality and complications compared with the neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio, since it encompasses three key immune response pathways.[12] Higher levels of SII in patients with trauma are an indication of the complex interaction between pro- and anti-inflammatory responses, which forms the key issue in the pathophysiology of secondary organ injury. In particular, higher SII values may indicate greater severity of inflammation and poorer prognosis.[13,14] Due to its accessibility and cost-effectiveness, the SII offers valuable guidance for therapeutic strategies and long-term outcomes.

ISS has been recognized as the gold standard for trauma assessment, whereas SII and D-dimer levels (D-D) have been extensively studied as markers of systemic inflammation and coagulation activation,[15,16] respectively. However, to the best of our knowledge, studies dynamically integrating these three parameters into a comprehensive predictive model for the early and accurate prediction of 28-day mortality remain limited. The 28-day mortality is widely adopted in critical care and trauma research as it captures the peak period of post-traumatic complications (such as sepsis and multiple organ dysfunction) while ensuring a stable outcome assessment with minimal loss to follow-up, thereby allowing for meaningful comparisons with existing literature.[17] Therefore, the present study aims to systematically assess the predictive efficacy of a combined SII, D-D and ISS model for 28-day mortality in patients with MI. Through receiver operating characteristic (ROC) curve analysis, optimal cut-off values for SII and D-D in predicting mortality were calculated, thereby providing clinicians with a quantitative tool for rapidly identifying patients at high-risk of 28-day mortality. Specifically, the clinical data of 142 patients were analyzed, aiming to obtain a prognostic indicator that is more convenient compared with traditional assessment indicators.

Materials and Methods

Patients

Clinical data from 142 patients with MI [mean age, 43.29±16.39 years; 116 male patients (81.7%) and 26 female patients (18.3%)] admitted to the Emergency Department of Shenzhen Longhua District Central Hospital (Shenzhen, China) between January 2019 and December 2023 were retrospectively analyzed.

The inclusion criteria were the following: i) Primary injury; ii) injury-to-admission time <24 h; iii) >14 years of age; iv) clinical confirmation of MI; and v) complete clinical records. The exclusion criteria were the following: i) Patients ≤14 years of age; ii) death upon admission; and iii) history of chronic diseases (including cardiac, cerebral and renal diseases, in addition to malignant tumors). The study protocol was approved by the Ethics Committee of Shenzhen Longhua District Central Hospital (Shenzhen, China; approval no. 2025–062-01).

Methods Upon Admission to the Emergency Resuscitation Room

Appropriate resuscitative measures were immediately performed, which included tracheal intubation, cardiopulmonary resuscitation, defibrillation, volume expansion, hemostasis, bandaging and immobilization to maintain vital signs. Patient information was collected from electronic medical records.

Routine blood tests, biochemical tests and coagulation tests were conducted. Blood samples were drawn at the time of initial presentation to the emergency department, prior to any major surgical intervention or blood transfusion. Routine blood tests were performed using a Mindray BC-7500 (Shenzhen Mindray Bio-Medical Electronics Co., Ltd.) fully automatic hematology analyzer. Biochemical analyses were conducted using a Johnson & Johnson VITROS-5600 automated biochemical analyzer and coagulation tests were performed using a Sysmex CICO (Sysmex America, Inc.) automated coagulation analyzer.

The 142 patients were categorized into survival (n=102) and death (n=40) groups based on their survival status on postoperative day 28. Differences in clinical data between groups were retrospectively analyzed, with indicators yielding statistically significant differences undergoing further evaluation.

Statistical Analysis

Statistical analyses were performed using SPSS 26.0 software (IBM, Corp)., Categorical variables (such as sex) are expressed in counts. Continuous variables that conform to the normal distribution are expressed as the mean ± standard deviation [such as hemoglobin (HGB)]. For most continuous variables that do not conform to the normal distribution, the interquartile range is used to describe their central tendency and degree of dispersion. Data with a non-normal distribution are presented as median (first quartile, third quartile) and were compared using the Mann–Whitney *U*-test. The comparison of HGB between the two groups was conducted using an unpaired *t*-test. Categorical data are presented as n (%). Group comparisons were performed using the χ^2-test or Fisher's exact test when expected frequencies were <5 (for example, for the comparison of 28-day survival rates). To construct the cumulative predictive model, multivariate binary logistic regression was conducted using SII, D-D and ISS as continuous independent variables. A linear predictor score derived from the model as regression coefficients were used to predict a 28-day mortality log-odds value of each patient. The combination predictor (combined score) formula was as follows: Combined score = $\beta_0 + \beta_1$ x SII + β_2 x D-D + β_3 x ISS. In this formula, β_0 is the intercept, whereas β_1, β_2 and β_3 are the regression coefficients of the individual variables. ROC analysis was used to evaluate the predictive performance of the combined model, where the area under the curve (AUC) was compared with that of the individual indicators. The results of this ROC analysis are presented in Figure 1. Plots of ROC curves were made to assess the prognostic capabilities of SII, D-D, ISS and the combined model (integrating SII, D-D and ISS), where AUC with the 95% CI was calculated for each indicator. P<0.05 was considered to indicate a statistically significant difference. The DeLong test of paired ROC curves was used to compare the joint model and individual indicators to evaluate the correctness of the prediction of the joint model.

Results

Comparison of General Patient Characteristics Between the Two Groups

A total of 142 patients were included in the present study, which were divided into survivor (n=102) and non-survivor (n=40) groups according to 28-day outcomes. The primary injury causes were traffic accidents (65 cases; 45.77%), high falls (37 cases; 26.05%) and low falls (20 cases; 14.08%). The distribution of injury mechanisms was compared between the survivor and non-survivor groups. There was no significant difference in survival rate among patients with different injury mechanisms (P>0.05; χ^2 test; Table 1). Other injury causes included stab wounds (13 cases; 9.15%) and blunt injuries (7 cases; 4.93%).

No significant differences were found between groups regarding sex, age, white blood cell count, platelet count, total protein, neutrophils, lymphocytes, creatinine, systolic blood pressure, diastolic blood pressure and mean arterial pressure. By contrast, significant differences were observed for HGB, D-D, ISS, heart rate, respiratory rate and SII (all P<0.05; Table 2). The median SII was significantly higher in the non-survivor group [849.93 (300.83, 1034.14)] than in the survivor group [340.11 (182.72, 578.31); P<0.001] The median HGB level was 116.29±29.49 g/l in the non-survivor group and 129.73±23.22 g/l in the survivor group, where the HGB levels were significantly lower in the non-survivor group (P<0.05).

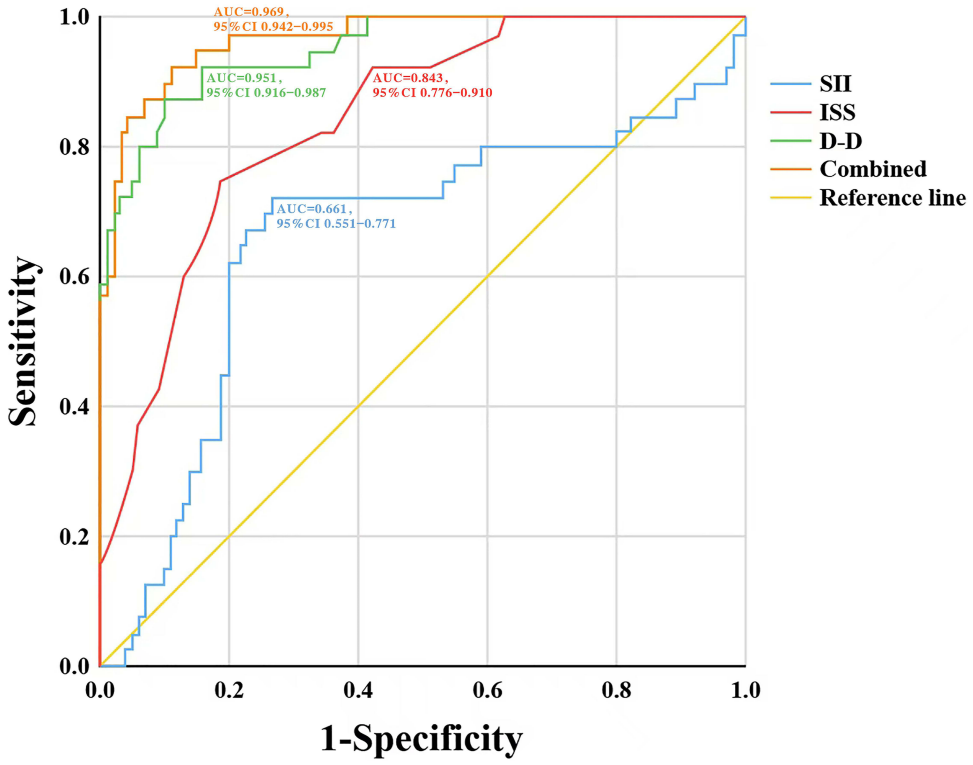

Figure 1 Predictive ability of SII, D-D, ISS and their combination for survival in patients with multiple injuries.
Abbreviations: SII, systemic immune-inflammation index; D-D, D-dimer; ISS, injury severity score.

The median D-D level was 28.07 μg/mL (18.03, 34.44 μg/mL) in the non-survivor group and 6.9 μg/mL (3.88, 10.98 μg/mL) in the survivor group, where the D-D levels were significantly lower in survivors (P<0.001). The median ISS score was 41.00 (34.25, 50.00) in the non-survivor group and 26.00 (20.00, 34.00) in the survivor group, where the ISS score was significantly lower in survivors (P<0.001). The median heart rate was 132.00 bpm (124.25, 137.00 bpm) in the non-survivor group and 114.00 bpm (104.50, 124.25 bpm) in the survivor group, where the heart rate was significantly lower in survivors (P<0.001). The median respiration was 29.00 bpm (25.00, 31.00 bpm) in the non-survivor group and 22.00 bpm (20.00–24.25 bpm) in the survivor group, where the respiration was significantly lower in survivors (P<0.001).

Factors Influencing Prognosis in Patients with Severe MI (ISS ≥16)

Logistic regression analysis was performed on variables with significant differences between groups that are pathophysiological relevant. The results indicated that admission levels of SII (P=0.036), D-D (P<0.001) and ISS (P=0.008) were independent predictors of 28-day mortality in these patients (Table 3).

Table 1 Comparison of Patients with Different Injury Mechanisms

Mechanisms	Non-Survivor (n=40)	Survivor (n=102)	Total (n=142)	P-value
Traffic accidents	25 (62.5)	40 (39.2)	65 (45.8)	0.058
High falls	9 (22.5)	28 (27.5)	37 (26.1)	
Low falls	4 (10.0)	16 (15.7)	20 (14.1)	
Other	2 (5.0)	18 (17.6)	20 (14.1)	

Notes: The data are presented as the number of patients (percentage). The P-value was derived from the χ^2 test (minimum expected frequency >5) comparing the distribution of injury mechanisms between survivors and non-survivors.

Table 2 Comparison of General Characteristics Between Survival and Non-Survivor Groups in Patients with Multiple Injuries

Testing Index	Normal Reference Range	Non-Survivor Group (n=40)	Survival Group (n=102)	χ^2/t/Z	P-value
Sex (male/female)	–	32/8	84/18	0.106[c]	0.744
Age, years	–	42.50 (30.00–51.00)	36.00 (27.75–50.00)	−1.016	0.309
WBC, 10^9/liter	3.5–9.5	12.18 (6.19–15.51)	9.27 (5.17–14.09)	−1.619	0.105
HGB, g/l	115-150	116.29±29.49	129.73±23.22	2.585	0.012[a]
PLT, 10^9/liter	125-350	220.5.00 (115.50–296.75)	237.00 (174.50–313.50)	−1.442	0.149
Total protein, g/l	65-85	61.00 (48.25–75.00)	70.00 (57.00–79.00)	−1.661	0.097
Neutrophilic granulocyte, 10^9/liter	1.8–6.3	10.15 (5.69–15.27)	9.57 (5.10–13.73)	−0.726	0.468
Lymphocyte, 10^9/liter	1.1–3.2	2.24 (1.48–3.09)	2.77 (1.75–3.20)	−1.347	0.178
Glutamic-pyruvic transaminase, U/l	9-50	151.0 (99.5–214.5)	148.5 (100.0–205.0)	−0.213	0.831
Glutamic oxaloacetic transaminase, U/l	15-40	48.50 (31.75–86.75)	69.00 (29.75–101.00)	−1.134	0.257
Creatinine, U/l	57-97	98.50 (72.25–140.75)	105.50 (80.00–143.25)	−0.728	0.467
D-D, μg/mL	0-0.55	28.07 (18.03–34.44)	6.9 (3.88–10.98)	−8.352	<0.001[a]
ISS	–	41.00 (34.25–50.00)	26.00 (20.00–34.00)	−6.352	<0.001[a]
Heart rate, bpm	60-100	132.00 (124.25–137.00)	114.00 (104.50–124.25)	−6.317	<0.001[a]
Respiration, bpm	12-20	29.00 (25.00–31.00)	22.00 (20.00–24.25)	−4.337	<0.001[a]
Systolic pressure, mmHg	90-140	102.00 (84.00–152.75)	128.50 (105.00–139.25)	−1.495	0.135
Diastolic pressure, mmHg	60-90	78.00 (52.00–108.50)	79.00 (67.25–91.00)	−0.225	0.822
Mean arterial pressure, mmHg	70-105	86.33 (62.17–121.25)	93.50 (82.33–104.42)	−0.708	0.479
SII	–	849.93 (300.83–1034.14)	340.11 (182.72–578.31)	−2.984	0.003[a]

Notes: [a]$P<0.05$. Data presented as the median (interquartile range) were compared using the Mann–Whitney *U*-test. Data presented as the mean ± standard deviation were compared using Student's unpaired *t*-test. Data presented as counts were compared using the χ^2 test.
Abbreviations: WBC, white blood cell; HGB, hemoglobin; PLT, platelet; D-D, D-dimer; ISS, injury severity score; SII, systemic immune-inflammation index.

Table 3 Multivariate Logistic Regression Analysis of Factors Influencing Mortality Outcomes

Index	Regression Coefficient	Standard Error	Wald	Odds Ratio (95% CI)	P-value
D-D	0.294	0.063	21.858	1.342 (1.186–1.519)	<0.001
ISS (≥16)	0.097	0.036	7.112	1.102 (1.026–1.183)	0.008
SII	0.001	0.001	4.392	1.001 (1.000–1.003)	0.036
Constant[a]	−9.609	1.956	24.127	<0.001	<0.001

Notes: [a]Constant refers to the intercept term of the logistic regression model. Univariate analysis in Table 2 revealed that D-D, ISS and SII were significantly associated with mortality ($P<0.05$), and these were therefore included in the multivariate model.
Abbreviations: D-D, D-dimer; ISS, injury severity score; SII, systemic immune-inflammation index.

Predictive Value of SII, D-D, ISS and Their Combination in MI Prognosis

ROC curves and AUC values were calculated to compare the predictive abilities of SII, D-D, ISS and their combination for 28-day mortality. Based on optimal ROC cut-off values, patients were classified into groups: SII ≤308.8 or SII >308.8; and D-D ≤2.35 or D-D >2.35. The 28-day mortality rates were compared between these groups.

The results showed that the AUC of D-D levels was 0.951, the AUC of ISS was 0.843, the AUC of SII was 0.661 and the AUC of the combination was 0.969, and all indicators had prognostic predictive value (AUC>0.5), with their combination demonstrating the highest predictive accuracy (Figure 1; Table 4).

Comparison of Mortality Among Patients with MI Grouped by SII Levels

ROC analysis revealed an optimal SII cut-off value of 308.8. Therefore, patients were divided into two groups (SII ≤308.8 and SII >308.8). The group with SII ≤308.8 had significantly lower D-D levels (P=0.0244) and a lower 28-day mortality rate (P=0.0267) compared with the group with an SII >308.8 (Table 5).

Comparison of Mortality Among Patients with MI Grouped by D-D Levels

ROC curve analysis indicated an optimal D-D cut-off value of 2.35, resulting in the division of patients into the D-D ≤2.35 (n=117) and D-D >2.35 (n=25) groups. The group with D-D ≤2.35 had a significantly lower 28-day mortality rate (P<0.001). Similarly, SII values in the D-D ≤2.35 group were lower (P<0.001; Table 6).

Table 4 Predictive Value of SII, D-D and ISS for Mortality in Patients with Multiple Injuries

Testing Index	Area Under the Curve	95% CI	Critical Value	Sensitivity	Specificity	Youden Index	Z-value	P-value
D-D	0.951	0.916–0.987	15.8	0.875	0.902	0.777	1.976	0.048
ISS	0.843	0.776–0.910	34.5	0.75	0.814	0.564	4.003	<0.001
SII	0.661	0.551–0.771	539.176	0.725	0.735	0.46	5.306	<0.001
Combination	0.969	0.942–0.995	0.232	0.925	0.892	0.817	34.74	<0.001

Notes: The P-value reported in receiver operating characteristic curve analysis tests whether the area under the curve of each indicator or model is significantly greater than 0.5, evaluating if the model performs better than random prediction. The Z-value and P-value for each row correspond to the test of the null hypothesis that the area under the curve for that specific indicator/model is equal to 0.5 (no discriminative ability).
Abbreviations: D-D, D-dimer; ISS, injury severity score; SII, systemic immune-inflammation index.

Table 5 Mortality Comparison Between Groups with Different SII Levels

Parameter	Group		Statistic	P-value
	SII ≤308.8 (n=58)	SII >308.8 (n=84)		
Median D-D, mg/l (IQR)	7.77 (3.95–15.85)	11.24 (5.68–23.70)	W=1,893	0.02435
28-day survival rate, N (%)	48 (82.76)	54 (64.29)	χ^2=4.9094	0.02671

Abbreviations: D-D, D-dimer; IQR, interquartile range; SII, systemic immune-inflammation index.

Table 6 Mortality Comparison Between Groups with Different D-D Levels

Parameter	Group		P-value
	D-D ≤2.35 (n=117)	D-D >2.35 (n=25)	
SII[a]	345.97 (180.76–763.57)	853.98 (651.18–998.59)	<0.001[b]
28-day survival rate, N (%)	101 (86.32)	1 (4)	<0.001[c]

Notes: [a]Data are presented as the median (interquartile range). [b]mann–Whitney *U*-test. [c]Fisher's exact test.
Abbreviations: D-D, D-dimer; SII, systemic immune-inflammation index.

Discussion

In the present retrospective study, it was demonstrated that the independent variables of 28-day mortality in patients with MI were the admission level of SII >308.8, D-D >2.35 μg/mL and the ISS score (analyzed as a continuous variable). The model that combined these three parameters showed improved predictive accuracy compared with each of the individual variables, supporting the concept of synergetic importance for assessing inflammatory, coagulative and anatomical injury in the initial prognostic model.[18,19]

Early assessment of injury severity, prompt transport and appropriate interventions effectively can reduce mortality and disability rates in patients with MI, improving prognosis.[20] Although medical advances and trauma center systems have enhanced MI management, mortality and disability rates remain high due to injury complexity and rapid progression.[21] Treatment outcomes for MI reflect the comprehensive capabilities of trauma centers. Given the rapidly changing condition of patients with MI, real-time risk assessment using accessible tools is essential.

Immune-inflammatory dysregulation is a central mechanism of secondary injury and markedly influences prognosis in patients with MI, necessitating accurate evaluation.[22] Among the inflammatory markers, SII has garnered attention due to its simple calculation method, low cost and ability to reflect inflammatory activation and immunosuppressive states simultaneously.[23] A previous study has indicated that SII is associated with surgical trauma severity in elderly patients with hip-fracture, as evidenced by its significant positive association with increased risks of 30-day and 1-year all-cause mortality (adjusted hazard ratio, 1.065; 95% CI, 1.044–1.087; P<0.001).[24] Logistic regression analysis in the present study suggested that SII is an independent risk factor for predicting 28-day mortality in patients with MI, with higher values indicating increased mortality risk. The prognostic ability of SII may involve the following mechanisms: i) SII integrates multiple immune processes involving neutrophils, lymphocytes and platelets. Neutrophils, the first inflammatory cell types to activate after injury, release reactive oxygen species and proteases, causing direct tissue damage, where prolonged elevation of neutrophils indicates an excessive inflammatory response.[25] Lymphocytes reflect compensatory anti-inflammatory responses associated with immunosuppression and secondary infection risk,[26] where increased lymphocyte apoptosis after injury reduces lymphocyte counts. Platelets serve as coagulation carriers and inflammation modulators[27] by releasing mediators, such as platelet factor 4 and TGF-β,[28] influencing the organ injury-repair balance.[29] ii) SII dynamically reflects the transition from excessive inflammation to immunosuppression following MI.[30] During the early post-injury stages (24–72 h), increased neutrophils and decreased lymphocytes markedly elevate SII, indicating SIRS risk. In the latter stages (3–7 days), persistently elevated SII reflects an immunosuppressive state associated with sepsis and multiple organ dysfunction syndrome. iii) SII is associated with molecular mediators of organ damage, such that higher SII values are positively associated with endothelial cell injury[31] and mitochondrial DNA release,[32] reflecting microcirculatory disturbances.

Accumulating evidence increasingly supports the association between elevated plasma D-D levels and injury severity.[33] A recent study involving patients with MI demonstrated notably higher D-D levels upon admission in patients who were severely injured compared with patients who were mildly injured. Furthermore, non-survivors had markedly higher D-D levels compared with those in survivors, likely reflecting coagulopathy induced by inflammatory stress.[34] Lee et al[35] previously reported that, compared with patients with D-D levels ≤34.53 mg/l, those with D-D levels >34.53 mg/l within 24 h after injury (trauma mechanisms such as drowning or hanging) had a 1.033-fold increased risk of 28-day mortality. Results from the present study similarly indicated increased mortality risk when admission D-D levels were >2.35 μg/mL, consistent with previous findings. Notably, the optimal D-dimer cut-off value identified in the present study (2.35 μg/mL) was markedly lower than the threshold of 34.53 mg/l associated with mortality in the study by Lee et al.[35]

ISS is a commonly used injury scoring system, with higher scores indicating greater injury severity and increased mortality risk.[36] Liu et al[37] reported that ISS effectively predicted mortality in elderly patients with trauma (AUC=0.74; 95% CI, 0.71–0.79). Another previous study demonstrated that Trauma and Injury Severity Score and ISS reliably predicted outcomes in pediatric patients with traumatic brain injury, assisting clinicians in risk-adjusted decision-making.[38] In the present study, ISS was found to be an independent predictor of 28-day mortality in patients with MI. Higher ISS scores are associated with increased mortality, likely due to the objective quantification of anatomical injury

severity and systemic pathophysiological disturbances. However, ISS has limitations when predicting prognosis. It neglects physiological factors, such as shock or low Glasgow Coma Scale scores, which markedly impacts patient outcomes.[39] Additionally, ISS underestimates isolated severe injuries (such as high AIS-score injuries confined to a single body region, such as the head, chest or abdomen), despite higher actual mortality compared with patients with multiple-region injuries and identical ISS scores.[40] Future studies should address these limitations by combining ISS with physiological scoring systems to improve prediction of coagulopathy and shock or by using alternative metrics, such as the New Injury Severity Score, which, despite greater sensitivity for single-region injuries,[41] is less frequently used clinically, primarily due to the entrenched historical use and global standardization of the ISS for trauma triage, registry reporting and outcomes research, which creates a high barrier for the adoption of modified scales.

The association of SII and D-D with mortality in the present study supports the close interaction of inflammatory and coagulation pathways in multiple trauma.[42] It is worth noting that the ISS was not included in the comparative analysis as the established cutoff of ISS ≥16 serves as a well-validated threshold for identifying patients with elevated mortality risk in trauma populations.[43] Thus, further comparative analysis of ISS was not performed in this context. Tissue injuries generated due to trauma trigger both innate immunity and the coagulation cascade, causing a pro-thrombotic state, which is marked by increased levels of D-D.[44] In addition, the stimulation of immune cells and endothelial dysfunction is mediated by systemic inflammation that is indicated by elevated SII. This nexus of inflammation-coagulation helps to advance trauma-induced coagulopathy and dysfunction of multiple organs, which eventually affects the survival of the patient.[45] The results of the present study have been consistent with this new evidence that combined measurement of inflammatory and coagulation biomarkers reveals improved prognostic data compared with individual measurements of each of the two pathways.[46–48]

Due to the limited sample size in each injury cause subgroup, a comparative analysis of mortality rates among these specific mechanisms was not performed. Future studies with larger cohorts are warranted to explore potential prognostic differences associated with different injury etiologies.

In summary, data from the present study suggest that SII and D-D are valuable early prognostic predictors for patients with MI. Mortality significantly increases when D-D is >2.35 μg/mL or SII is >308.8 upon admission. Combined assessment using SII, D-D and ISS provides the most accurate prognostic evaluation, indicating that early intervention based on these predictive indicators may reduce mortality in patients with MI. However, the present study has several limitations. Its retrospective, single-center design and relatively small sample size (142 cases) limit result generalizability and introduce potential selection bias. Additionally, the present study lacks an external validation cohort to verify the combined generalizability of the model. Only baseline SII and D-D values were assessed at admission without dynamic monitoring over time, potentially overlooking important prognostic information. Future studies should conduct large-scale, multicenter, prospective cohort studies for external model validation and explore whether changes in SII and D-D over time have greater predictive value. Additionally, investigating the molecular mechanisms underlying immune-coagulation interactions associated with these indicators may offer theoretical foundations for developing novel therapeutic targets.

Data Sharing Statement

The data generated in the present study may be requested from the corresponding author.

Ethics Approval and Consent to Participate

The present study was approved by the Medical Ethics Committee of Shenzhen Longhua District Central Hospital (approval no. 2025-062-01). All patients provided written informed consent for participation, which included the use of their clinical data and samples for research purposes.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically

reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no competing interests in this work.

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