

Systemic Immune-Inflammation Index as an independent predictor of in-hospital morbidity in diabetic patients with ST-elevation myocardial infarction

Rafid B. Altaweel,¹ Abdulameer A. Abdulhameed,² Karzan M. Abdullah³

¹Department of Medicine, College of Medicine, Al-Nahrain University, Baghdad; ²Department of Medicine, College of Medicine, University of Basra; ³Rizgari Teaching Hospital, Arbil, Iraq

Abstract

Diabetic patients presenting with ST-elevation myocardial infarction (STEMI) are particularly vulnerable to unfavorable in-hospital outcomes. We examined the prognostic value of the systemic immune-inflammation index (SII) for predicting in-hospital morbidity in a prospective cohort of 108 diabetic patients with STEMI; patients were classified by clinical outcome into morbid (n=57) and non-morbid (n=51) groups. Demographic characteristics, clinical findings, laboratory results, and angiographic data were recorded. In-hospital morbidity occurred in 52.8% of patients. Multivariate logistic regression identified prior myocardial infarction [p=0.038, odds ratio (OR)=3.34, 95% confidence interval (CI): 1.07-11.01], three-vessel disease (p=0.019, OR=3.81, 95% CI: 1.26-19.51), and SII (p=0.022, OR=3.16, 95% CI: 1.12-14.5) as independent predictors of morbidity. SII demonstrated good discriminatory ability for predicting morbidity with an area under the receiver operating characteristic curve of 0.718 (95% CI: 0.662-0.844, p<0.001); at a cut-off >604.75, the sensitivity was 77% and specificity 63%. These findings suggest that SII is a useful and readily available inflammatory marker for predicting in-hospital morbidity among diabetic patients with STEMI.

Key words: Systemic Immune-Inflammation Index, ST-elevation myocardial infarction, diabetes mellitus, in-hospital mortality, in-hospital morbidity.

Correspondence to: Rafid B. Altaweel, Department of Medicine, College of Medicine, Al-Nahrain University, Baghdad, Iraq.
E-mail: dr Rafid76@nahrainuniv.edu.iq

Introduction

ST-elevation myocardial infarction (STEMI) is the most severe and potentially fatal manifestation of cardiovascular disease, which is the world's leading cause of death. In comparison to non-diabetic patients, diabetic patients are a particularly vulnerable subgroup because they have more extensive coronary atherosclerosis, microvascular dysfunction, a pro-thrombotic, dyslipidemia, pro-inflammatory milieu, all of which increase the risk of heart failure, arrhythmia, and in-hospital death following STEMI.^{1,2} Early risk stratification in diabetic STEMI is still not ideal, despite advancements in primary percutaneous coronary intervention (PCI) and guideline-directed therapy.

Plaque rupture, thrombus formation, and myocardial injury are all significantly influenced by inflammation and immune activation. The neutrophil-to-lymphocyte ratio and other straightforward blood-based inflammatory indices that are derived from complete blood counts (CBC) have been linked to poorer short-term and long-term outcomes following acute coronary syndromes.³ Nevertheless, the relationship between inflammation, immunological status, and thrombosis is not adequately captured by these markers. Combining these elements into a single metric, the Systemic Immune-

Inflammation Index (SII) has shown promise as a prognostic indicator for a number of cardiovascular diseases, including STEMI.⁴

In a number of malignant tumors, prior research has shown a strong correlation between SII and prognosis.⁵ In solid tumors, hepatocellular carcinoma, lung cancer, and breast cancer, high SII levels are especially associated with a poor prognosis.⁶ In patients with a variety of cardiovascular conditions [such as atrial fibrillation, chronic heart failure, and coronary artery disease (CAD)], diabetes, and related complications, elevated SII has recently been found to be a prognostic indicator associated with unfavorable outcomes.⁷ Nevertheless, data focusing specifically on diabetic patients presenting with STEMI are limited, and it is unclear whether SII can reliably predict in-hospital morbidity in this high-risk group. Therefore, the present study aims to evaluate the SII as a predictor of in-hospital morbidity in diabetic patients with STEMI.

Materials and Methods

This prospective cohort study was conducted at Al-Emamain Al-Kadhmain Medical City and Al-Yarmouk Teaching Hospital emergency room and cardiac care unit from the 1st of October 2024 to the 1st of March 2025. The study included 108 patients

who presented with STEMI and had a confirmed diagnosis of type 2 diabetes mellitus (T2DM). The study population comprised adult patients diagnosed with both T2DM and STEMI who were admitted to the hospital during the study period. The diagnosis of STEMI was based on clinical presentation, electrocardiographic findings, and elevated cardiac biomarkers.

Patients with hematological diseases, recent use of immunosuppressive therapy (for example in patients with autoimmune disorders), active infection or inflammatory conditions at the time of admission, history of malignancy, under glucocorticoids therapy, and those with chronic kidney disease requiring dialysis were excluded from the study.

Ethical approval

Ethical and scientific approval for the research was obtained from the Institutional Review Board, College of Medicine, Al-Nahrain University (No. 202512126 in 07/12/2025).

Echocardiography

Transthoracic echocardiography was performed by an experienced echocardiographer (Vivid S6/USA) with the patient in a partial left lateral decubitus position, and all examinations, measurements, and calculations were performed according to recommendations by the American Society of Echocardiography. Left ventricular ejection fraction (LVEF) was measured using the modified Simpson method. LFEV was defined as follows: normal $\geq 50\%$, mildly reduced 41–49%, moderately reduced 30–40%, and severely reduced $<30\%$.

Data collection

Demographic, clinical, and laboratory data were collected using a structured case report form. Variables included age, sex, body mass index (BMI), comorbidities, smoking status, and previous cardiovascular history. Additionally, ejection fraction and Killip's classification, which quantified the severity of heart failure in patients with myocardial infarction (MI), were calculated for the patients on admission. After diagnosis of MI, upon arrival at the emergency department, patients received a loading dose of aspirin and clopidogrel, followed by a maintenance dose. Venous blood samples were collected at admission using standardized dipotassium ethylene diamine tetra-acetic acid tubes. CBC analysis was performed to measure platelets, lymphocytes, neutrophils, hemoglobin, and white blood cells. The SII was calculated according to the following formula [Eq. 1]:

$$\text{SII} = \text{Platelet count} \times \text{Neutrophil count} / \text{Lymphocyte count} \quad [\text{Eq. 1}]$$

In addition, random blood sugar, and glycated hemoglobin (HbA1c) were also documented for the patients as part of the data collection process.

Outcome assessment

The main outcome of the study was in-hospital morbidity (during up to 7 days hospitalization) and morbidity which was defined as reduced LVEF $\leq 40\%$ and/or Killip's class $\geq \text{II}$.

Statistical analysis

All data were recorded in Microsoft Excel and analyzed with Statistical Package for Social Sciences (SPSS) software version 25.0 (IBM, Armonk, NY, USA). Continuous data were subjected to the normality test (Shapiro-Wilk test). Data with normal distribution

were presented as mean and standard deviation and analyzed with the Student t-test. Data with non-normal distribution were presented as median and range and analyzed with the Mann-Whitney U test (for two-group comparison) or Kruskal-Wallis (for more than two-group comparison). Categorical variables were expressed as numbers and percentages and analyzed with the chi-square test. The receiver operating characteristic (ROC) curve was used to evaluate the predictive value of SII in morbidity. Spearman's correlation test was used to examine the possible correlation of SII with other continuous variables. A p-value lower than 0.05 was considered to indicate a statistically significant difference.

Results

Morbidity rate

Out of 108 patients included in the study, 57 (52.78%) presented with moderately reduced EF and/or Killip's classification $\geq \text{II}$.

Association of baseline demographic and clinical characteristics of the patients with morbidity

Patients in the morbid group were significantly older than those in the non-morbid group (60.19 ± 14.13 vs. 54.80 ± 10.52 years, $p=0.027$). Additionally, there was a significant association between sex and morbidity ($p=0.034$: males made up the majority of the non-morbid group (84.31%), while females were comparatively more common among those who experienced complications (33.33%). On the other hand, there was no significant difference in BMI or smoking status between patients who were morbid and those who were not. Similarly, both groups had high rates of hypertension, dyslipidemia, ischemic heart disease, and chronic kidney disease, with no discernible differences.

Morbid patients were significantly more likely than non-morbid patients to have had a prior history of MI (35.09% vs. 13.73% , $p=0.010$). On the other hand, there were no statistically significant differences between the two groups' patterns of coronary vessel involvement [left anterior descending artery (LAD), left circumflex artery (LCX), right coronary artery (RCA) and left main coronary artery (LMCA)]. Troponin, admission random blood sugar, and HbA1c are examples of biomarkers of infarct size and glycemic status that did not significantly differ between non-morbid and morbid patients (Table 1).

Association of hematologic indices of the patients with morbidity

Patients in the morbid group had significantly lower hemoglobin levels than non-morbid patients (12.65 ± 2.22 vs. 13.89 ± 2.3 g/dL, $p=0.005$). Although total WBC and neutrophil counts were slightly higher in the morbid group, these differences did not reach statistical significance ($p=0.197$ and 0.051 , respectively). In contrast, lymphocyte counts were significantly lower among morbid patients (2.41 ± 0.92 vs. $2.95 \pm 1.42 \times 10^9/\text{L}$, $p=0.010$). The SII was markedly elevated in the morbid group (median 905.4 vs. 501.6, $p<0.001$), suggesting that a higher inflammatory and pro-thrombotic state is strongly associated with in-hospital morbidity in this cohort (Table 2).

Multivariate analysis

In order to find independent factors associated with morbidity, multivariate logistic regression was performed. For this test, all factors that had a $p \leq 0.1$ in the univariate analysis were entered into the model. The results are shown in Table 3. Each of the history of MI [$p=0.038$, odds ratio (OR)=3.34, 95% confidence interval (CI): 1.07–

11.01], three affected vessels ($p=0.019$, $OR=3.81$, 95% CI: 1.26-19.51), and SII ($p=0.022$, $OR=3.16$, 95% CI: 1.12-14.5) were independent predictors of morbidity in diabetic patients with STEMI.

Systemic Immune-Inflammation Index as predictor for morbidity

The ROC curve was used to evaluate the predictive value of SII as a predictor for morbidity in T2DM patients with STEMI. The area under the curve (AUC) was 0.718, 95% CI: 0.62-0.816, $p<0.001$. The sensitivity and specificity of the test at cut-off value of $SSI>604.75$ were 77% and 63%, respectively (Figure 1).

Correlation of Systemic Immune-Inflammation Index with other variables

Spearman's correlation test was used to examine the correlation of SII with other variables. SII displayed a significant positive correlation with each of age ($r=0.253$, $p=0.008$). On the other hand, SII

had a significant negative correlation with each of LVEF ($r=-0.507$, $p<0.001$) and hemoglobin ($r=-0.256$, $p=0.007$). Otherwise, no significant correlation was found with other parameters (Table 4).

Association of Systemic Immune-Inflammation Index with categorical variables

The relationship between the SII and different categorical variables is displayed in Table 5. SII was significantly higher in smokers compared to non-smokers (888.7 vs. 557.15, $p=0.026$) and in females compared to males (median 1009.2 vs. 514.3, $p<0.001$). Additionally, SII was significantly higher in patients with dyslipidemia than in those without (846.45 vs. 581.2, $p=0.039$), whereas hypertension had a marginally significant association ($p=0.053$). SII gradually increased from Killip I to III (501.6, 851.0, and 1110, respectively; $p<0.001$). Conversely, CKD, IHD, MI history, and the particular culprit vessel (LAD, LCX, RCA, LMCA) did not significantly affect SII (all $p>0.05$).

Table 1. Association of demographic characteristics of the patients with morbidity.

Variables	Non-morbid (n=51)	Morbid (n=57)	p
Age, years			
Mean± SD	54.76±9.95	59.93±14.37	0.034
Range	40-87	22-87	
Sex			
Male	43 (84.31%)	38 (66.67%)	0.034
Female	8 (15.69%)	19 (33.33%)	
BMI, kg/m ²			
Mean± SD	25.98±3.34	27.25±3.97	0.075
Range	20.0-34.4	22.1-35.7	
Smoking			
Never	18 (35.29%)	28 (49.12%)	0.147
Ex/current	33 (64.71%)	29 (50.88%)	
Comorbidities			
Hypertension	41 (80.39%)	48 (84.21%)	0.603
Dyslipidemia	26 (50.98%)	26 (45.61%)	0.577
IHD	20 (39.22%)	25 (43.86%)	0.625
CKD	10 (19.61%)	13 (22.81%)	0.685
History of MI			
No	44 (86.27%)	37 (64.91%)	0.010
Yes	7 (13.73%)	20 (35.09%)	
Vessels involved			
LAD	36 (70.59%)	34 (59.65%)	0.235
LCX	31 (60.78%)	38 (66.67%)	0.525
RCA	10 (19.61%)	15 (26.32%)	0.409
LMCA	4 (7.84%)	5 (8.77%)	0.862
No. of vessels			
One	22 (43.14%)	26 (45.61%)	0.083
Two	27 (52.94%)	22 (38.6%)	
Three	2 (3.91%)	9 (15.79%)	
Troponin, ng/mL			
Median	501.6	902.4	0.965
Range	50-1000	50-4000	
RBS, mg/dL			
Mean± SD	230.76±91.34	223.84±66.44	0.651
Range	100-600	150-600	
HbA1c			
Mean± SD	8.82±1.6	9.18±1.29	0.211
Range	6.0-12.0	7.0-12.0	

SD, standard deviation; BMI, body mass index; IHD, ischemic heart disease; CKD, chronic kidney disease; MI, myocardial infarction; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; LMCA, left main coronary artery; RBS, random blood sugar; HbA1c, glycated hemoglobin.

Discussion

The present study aimed to evaluate the SII as a predictor of in-hospital morbidity in diabetic patients with STEMI. According to the result of the study, The AUC for SII as predictor for morbidity was 0.718. The sensitivity and specificity of the test at cut-off value of $SSI > 604.75$ was 77% and 63%, respectively.

These findings are consistent with a number of earlier research projects conducted globally. A total of 480 Chinese patients with acute STEMI were included in the study by Zhang *et al.*⁸ The AUC=0.839 (95% CI: 0.752-0.927; $p < 0.001$), the best cut-off value was 941, the sensitivity was 63.8%, and the specificity was 88% in the ROC curve results for predicting adverse outcome. These results suggest a close relationship between inflammation, thrombosis, and immune dysregulation in post-infarction remodeling, which is especially pertinent in diabetic patients who already have elevated inflammatory and pro-thrombotic states. The authors also verified that SII maintained prognostic power across the majority of clinical

subgroups and performed better than conventional inflammatory markers alone.

Orhan *et al.* proved that the higher the level of SII, the higher the in-hospital mortality in diabetic patients with acute coronary syndrome (ACS).⁹ Li *et al.* included 1701 patients with ACS who underwent PCI and demonstrated that SII is an independent predictor of MI, nonfatal ischemic stroke, and all-cause death.¹⁰ Zhao *et al.* found a higher incidence of major adverse cardiovascular events (MACE) linked to higher SII values in a large study on patients who had previously undergone coronary artery bypass grafting.¹¹ Even after controlling for conventional risk factors, elevated SII independently doubled the risk of MACE (hazard ratio ≈ 2.0 , $p = 0.023$). According to ROC analysis, the SII's AUC for predicting MACE following PCI was 0.63 (95% CI: 0.52-0.71, $p < 0.001$). A meta-analysis suggests that SII may be a potential biomarker for the development of CAD, and elevated SII is associated with an increased risk of CAD.¹²

In patients who received primary PCI for STEMI, Esenboga *et al.* showed an independent correlation between SII levels and the

Table 2. Association of hematologic indices of the patients with morbidity.

Variables	Non-morbid (n=51)	Morbid (n=57)	p
Hb, g/dL			
Mean± SD	13.89±2.3	12.65±2.22	0.005
Range	9.0-17.4	9.0-17.0	
WBC×10 ⁹ /L			
Mean± SD	10.5±4.48	11.62±4.5	0.197
Range	5±23.0	3.2-24.0	
Lymphocyte ×10 ⁹ /L			
Mean± SD	2.95±1.42	2.41±0.92	0.020
Range	0.8-6.8	0.5-5.0	
Neutrophil ×10 ⁹ /L			
Mean± SD	6.4±3.67	7.73±3.67	0.051
Range	2.50-19.00	2.10-19.00	
PLT ×10 ⁹ /L			
Mean± SD	253.02±61.23	272.12±78.12	0.699
Range	170-442	103-442	
SII			
Median	501.6	905.4	<0.001
Range	128.8-1732.5	340-1789	

SD, standard deviation; Hb, hemoglobin; WBC, white blood cell; PLT, platelets; SII, Systemic Immune-Inflammation Index.

Table 3. Multivariate analysis for factors associated with morbidity in diabetic patients with ST-elevation myocardial infarction.

Variable	Regression coefficient (B)	p	OR	95%CI
Age	0.023	0.185	1.02	0.90-1.06
Female sex	0.92	0.069	2.5	0.92-6.23
BMI	0.095	0.097	1.1	0.98-1.23
History of MI	1.25	0.038	3.34	1.07-11.01
Number of vessels				
One vessel		0.111	1.0	
Two vessels	-0.37	0.362	0.69	0.31-1.533
Three vessels	1.34	0.019	3.81	1.26-19.51
Hb	-0.18	0.073	0.83	0.68-1.01
Lymphocyte	-0.58	0.078	0.56	0.29-1.07
Neutrophil	0.20	0.057	1.22	0.96-1.64
SII	1.33	0.022	3.16	1.12-14.5

OR, odds ratio; CI, confidence interval; BMI, body mass index; MI, myocardial infarction; Hb, hemoglobin; SII, Systemic Immune-Inflammation Index.

occurrence of the no-reflow phenomenon.¹³ Additionally, a study found that among patients with aortic stenosis undergoing transcatheter aortic valve implantation, elevated SII levels may be a predictor of short-term mortality.¹⁴

A large-scale study revealed that heightened levels of SII correlated with unfavorable clinical prognoses among diabetic patients undergoing PCI.¹⁵ Additionally, Urbanowicz *et al.* identified SII as a predictive marker for long-term prognosis in diabetic patients undergoing off-pump coronary artery bypass grafting.⁷

SII is a novel biomarker that serves as a regular indicator of systemic inflammation and is essentially derived from the count of three important immune cells: neutrophils, platelets, and lymphocytes. Immune cells are known to play a role in both heart damage and repair.¹⁶ Mechanistically, activated neutrophils cause myocardial damage by releasing a range of proteolytic enzymes, including elastase and myeloperoxidase.¹⁷ On the other hand, lymphocytes represent a controlled inflammatory process that limits myocardial damage and suppresses excessive immune responses, making them an early indicator of physiological stress.¹⁸ Platelet aggregation, adhesion, recruitment, and release can all exacerbate systemic inflammation in patients.¹⁹ Numerous proinflammatory and thrombosis-causing cytokines are released by activated platelets, which also interact with other white blood cells to worsen atherosclerosis and plaque instability. This is frequently linked to detrimental cardiovascular consequences.²⁰

In the present study, SII displayed a significant positive correlation with each of the number of vessels ($r=0.235$, $p=0.015$) and Killip class ($r=0.430$, $p<0.001$).

Although studies specifically limited to diabetic STEMI are still relatively scarce, several recent STEMI studies support the same direction of association. A recent study by Su *et al.* in STEMI patients

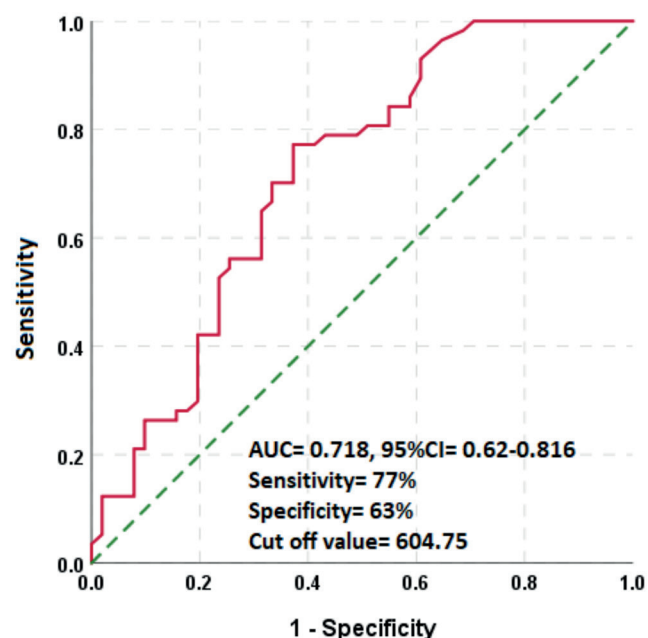


Figure 1. Receiver operating characteristic curve for the Systemic Immune-Inflammation Index as predictor for morbidity in diabetic patients with ST-elevation myocardial infarction. AUC, area under the curve; CI, confidence interval.

Table 4. Spearman's correlation of the Systemic Immune-Inflammation Index with other variables.

Variable	SII	
	Coefficient	p
Age	0.253	0.008
BMI	0.026	0.787
LVEF	-0.507	<0.001
Hb	-0.256	0.007
Troponin	0.1092	0.344
RBS	0.133	0.170
HbA1c	0.086	0.377

SII, Systemic Immune-Inflammation Index; BMI, body mass index; LVEF, left ventricular ejection fraction; Hb, hemoglobin; RBS, random blood sugar; HbA1c, glycated hemoglobin.

Table 5. Association of the Systemic Immune-Inflammation Index with categorical variables.

Variable	SII	p
Sex		
Male	514.3 (128.8-1789.0)	<0.001
Female	1009.2 (695.7-1520.0)	
Smoking		
No	557.15 (151.3-1789.0)	0.026
Yes	888.7 (128.8-1789.0)	
Hypertension		
No	514.3 (270.0-1291.2)	0.053
Yes	781.2 (128.8-1789.0)	
Dyslipidemia		
No	581.2 (151.3-1732.5)	0.039
Yes	846.45 (128.8-1789.0)	
CKD		
No	695.7 (128.8-1789.0)	0.327
Yes	875.0 (151.3-1732.5)	
IHD		
No	695.7 (128.8-1789.0)	0.430
Yes	851.7 (128.8-1732.5)	
History of MI		
No	781.2 (128.8-1789.0)	0.870
Yes	633.0 (128.8-1732.5)	
Killip's class		
I	501.6 (128.8-1732.5)	<0.001
II	851.0 (340.8-1789.0)	
III	1110 (448.8-1732.5)	
LAD		
No	730.6 (270.0-1789.0)	0.396
Yes	633.0 (128.8-1520.0)	
LCX		
No	905.3 (270.0-1789.0)	0.102
Yes	633.0 (128.8-1520.0)	
RCA		
No	781.2 (128.8-1732.5)	0.372
Yes	493.3 (270.0-1789.0)	
LMCA		
No	695.7 (128.8-1789.0)	0.306
Yes	907.2 (427.2-1732.5)	

SII, Systemic Immune-Inflammation Index; CKD, chronic kidney disease; IHD, ischemic heart disease; MI, myocardial infarction; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; LMCA, left main coronary artery. Data were expressed as median and range.

after PCI found that higher SII levels were independently associated with mortality and MACEs.²¹ Another study by Liu *et al.* showed that inflammatory indices in STEMI were linked to the severity markers such as Killip class and angiographic disease burden.²²

In the present study, SII increased progressively with worsening Killip class among diabetic patients with MI, rising from 501.6 in Killip I to 851.0 in Killip II and 1110 in Killip III ($p < 0.001$). This finding is very close to that reported by Wei *et al.* in AMI patients, where SII had a significantly higher proportion of Killip II-IV status, lower LVEF, and more in-hospital MACE;²³ the optimal SII cut-off for predicting MACE was 1085.55, almost identical to the Killip III mean observed in the present study. Luo *et al.* demonstrated that in diabetic patients with MI, higher SII was independently associated with worse survival, supporting a relationship between SII, myocardial injury, and cardiac dysfunction.²⁴

SII provides a more dynamic and accurate assessment of inflammation with higher sensitivity and predictive ability than single inflammatory markers. SII is also affordable and easy to compute, which makes it appropriate for standard clinical practice, particularly in environments with limited resources.

There are a number of limitations to this study that should be noted. First, the results may not be as broadly applicable as they could be due to the single-center design and relatively small sample size. Second, dynamic changes in inflammatory status during hospitalization were not evaluated, which could offer more prognostic information. Instead, SII was measured at a single time point on admission. Third, it was impossible to completely control the possible confounding effects of acute infections, drugs, and reperfusion techniques on inflammatory indices. Finally, the impact of SII on long-term morbidity and survival in diabetic STEMI patients is still unknown because the study only assessed short-term (in-hospital) outcomes.

Conclusions

The current study shows that in diabetic patients with STEMI, the SII is a powerful and independent predictor of in-hospital morbidity. The critical role of the inflammatory and thrombotic burden in determining clinical outcomes in this high-risk population is demonstrated by the significant correlation found between higher SII values and higher rates of in-hospital complications. Additionally, SII demonstrated acceptable sensitivity and specificity at the designated cut-off values, as well as good diagnostic accuracy in predicting unfavorable outcomes. These results suggest that SII is a straightforward, affordable, and easily accessible biomarker that can be used successfully for early risk stratification in patients with diabetic STEMI. By identifying patients who are more likely to experience complications and die, routine clinical assessment using SII may enable more aggressive treatment approaches and closer monitoring.

References

- Chen S, Chen Y, Liu B, et al. Does diabetes mellitus increase the short- and long-term mortality in patients with critical acute myocardial infarction? *Front Endocrinol* 2021;12:797049.
- Mohammedi MKM, Altaweel RB, Gburi AKKA. Exploring the role of non-HDL cholesterol in acute coronary syndrome: a case-control study. *Pak Heart J* 2024;57:13-7.
- Yoon GS, Park HC, Choi JH, et al. Association between on-admission neutrophil-to-lymphocyte ratio and mechanical complications and mortality in patients with STEMI. *J Korean Med Sci* 2021;36:e131.
- Babes EE, Radu AF, Cretu NA, et al. Risk stratification in acute coronary syndromes: the systemic immune-inflammation index as prognostic marker. *Med Sci* 2025;13:116.
- Bruno M, Bizzarri N, Teodorico E, et al. The potential role of systemic inflammatory markers in predicting recurrence in early-stage cervical cancer. *Eur J Surg Oncol* 2024;50:107311.
- Wenpei G, Yuan L, Liangbo L, et al. Predictive value of preoperative inflammatory indexes for postoperative early recurrence of hepatitis B-related hepatocellular carcinoma. *Front Oncol* 2023;13:1142168.
- Urbanowicz T, Michalak M, Al-Imam A, et al. The significance of systemic immune-inflammation index for mortality prediction in diabetic patients treated with off-pump coronary artery bypass surgery. *Diagnostics* 2022;12:634.
- Zhang W, Jia H, Zhao X, et al. Predictive value of cumulative SII for MACE in STEMI patients after PCI. *Medicine* 2025;104:e41983.
- Orhan AL, Saylik F, Cicek V, et al. Evaluating the systemic immune-inflammation index for in-hospital and long-term mortality in elderly non-ST-elevation myocardial infarction patients. *Aging Clin Exp Res* 2022;34:1687-95.
- Li Q, Ma X, Shao Q, et al. Prognostic impact of multiple lymphocyte-based inflammatory indices in acute coronary syndrome patients. *Front Cardiovasc Med* 2022;9:811790.
- Zhao Y, Zhao S, Shi Y, et al. The Predictive Value of the Systemic Immune-Inflammation Index for Cardiovascular Events in Chronic Total Occlusion Patients Who Prior Coronary Artery Bypass Grafting. *J Inflamm Res* 2024;17:8611-23.
- Ye Z, Hu T, Wang J, et al. Systemic immune-inflammation index as a potential biomarker of cardiovascular diseases: a systematic review and meta-analysis. *Front Cardiovasc Med* 2022;9:933913.
- Esenboga K, Kurtul A, Yamantürk YY, et al. Systemic immune-inflammation index predicts no-reflow phenomenon after primary percutaneous coronary intervention. *Acta Cardiol* 2022;77:59-65.
- Tosu AR, Kalyoncuoglu M, Biter H, et al. Prognostic value of systemic immune-inflammation index for major adverse cardiac events and mortality in severe aortic stenosis patients after TAVI. *Medicina* 2021;57:588.
- Bian X, He J, Zhang R, et al. The combined effect of systemic immune-inflammation index and type 2 diabetes mellitus on the prognosis of patients undergoing percutaneous coronary intervention: a large-scale cohort study. *J Inflamm Res* 2023;16:6415-29.
- Anzai A, Ko S, Fukuda K. Immune and inflammatory networks in myocardial infarction: current research and its potential implications for the clinic. *Int J Mol Sci* 2022;23:5214.
- Mauler M, Herr N, Schoenichen C, et al. Platelet serotonin aggravates myocardial ischemia/reperfusion injury via neutrophil degranulation. *Circulation* 2019;139:918-31.
- Zhao WM, Tao SM, Liu GL. Neutrophil-to-lymphocyte ratio in relation to the risk of all-cause mortality and cardiovascular events in patients with chronic kidney disease: a systematic review and meta-analysis. *Ren Fail* 2020;42:1059-66.
- Broijersens A, Hamsten A, Eriksson M, et al. Platelet activity in

- vivo in hyperlipoproteinemia – importance of combined hyperlipidemia. *Thromb Haemost* 1998;79:268-75.
20. Totani L, Evangelista V. Platelet-leukocyte interactions in cardiovascular disease and beyond. *Arterioscler Thromb Vasc Biol* 2010;30:2357-61.
 21. Su G, Zhang Y, Xiao R, et al. Systemic immune-inflammation index as a promising predictor of mortality in patients with acute coronary syndrome: a real-world study. *J Int Med Res* 2021;49:3000605211016274.
 22. Liu Y, Liu J, Liu L, et al. Association of systemic inflammatory response index and pan-immune-inflammation-value with long-term adverse cardiovascular events in st-segment elevation myocardial infarction patients after primary percutaneous coronary intervention. *J Inflamm Res* 2023;16:3437-54.
 23. Wei X, Zhang Z, Wei J, Luo C. Association of systemic immune inflammation index and system inflammation response index with clinical risk of acute myocardial infarction. *Front Cardiovasc Med* 2023;10:1248655.
 24. Luo J, Qin X, Zhang X, et al. Prognostic implications of systemic immune-inflammation index in myocardial infarction patients with and without diabetes: insights from the NOAFCA-MI-SH registry. *Cardiovasc Diabetol* 2024;23:41.

Received: 4 May 2026; Accepted: 12 July 2026; Early view: 31 July 2026.

Contributions: all the authors have directly participated in the preparation of this manuscript and have approved the final version submitted. Rafid B. Altaweel: data collection, laboratory works. Abdulameer A. Abdulhameed: statistical analysis and draft of the manuscript. Karzan M. Abdullah: study design and interpretation of the result. All the authors have read and approved the final manuscript.

Conflict of interest: the authors declare that they have no conflict of interest.

Ethics approval and consent to participate: the study design was approved by the Institutional Review Board (Medical Ethics Committee) of the College of Medicine/ Al-Nahrain University (No. 202512126).

Informed consent: written informed consent to participate in the study was obtained from all participants before enrolment and prior to the commencement of data collection. Participants received a comprehensive explanation of the study objectives, procedures, confidentiality measures, and the intended use of their data for research purposes.

Patient consent for publication: prior to the commencement of data collection, a written informed consent was procured from each participant following a comprehensive explanation of the study's objectives. The confidentiality of the data throughout the research was assured, and the participants were informed that the data would be utilized solely for research purposes.

Availability of data and materials: all data generated or analyzed during this study are included in this manuscript. The datasets and materials used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Acknowledgments: the authors highly appreciate the great help of the medical staff at Al-Emamain Al-Kadhmain Medical City and Al-Yarmouk Teaching Hospital especially those in cardiac imaging and echography departments.

Declaration of generative artificial intelligence and artificial intelligence-assisted technologies in the writing process: during the preparation of this manuscript, the authors used ChatGPT, developed by OpenAI, solely to improve the English language, readability, and clarity of the text. After using this tool, the authors carefully reviewed and edited the manuscript and take full responsibility for its content.

Publisher's note: all claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article or claim that may be made by its manufacturer is not guaranteed or endorsed by the publisher.

This work is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License (CC BY-NC 4.0).