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

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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.

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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.

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 1st of October 2024 to 1st of March 2025. The study included 108 patients who presented with STEMI and had a confirmed diagnosis of 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 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 modified Simpson method. LFEV was defined as follows: normal ≥50%, mildly reduced 41-49%, moderate reduced 30-40%, severely reduced <30%.

Data collection

Demographic, clinical, and laboratory data were collected using a structured case report form. Variables included age, sex, 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 MI, was 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 maintenance dose. Venous blood samples were collected at admission using standardized dipotassium EDTA tubes. CBC analysis was performed to measure platelets, lymphocytes, neutrophils, hemoglobin, and white blood cells. The SII was calculated according to the following formula:

SII = Platelet count × Neutrophil count / Lymphocyte count

In addition, random blood sugar, and 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≤40%) and/or Killip's class ≥II.

Statistical analysis

All data were recorded in Microsoft excel and analyzed with Statistical package for Social Sciences (SPSS) software version 25.0. Continuous data were subjected to normality test (Shapiro Wilk test), Data with normally distribution were presented as mean and standard deviation, and analyzed with Student t-test. Data with non-normal distribution were presented as median and range and analyzed with Mann Whitney U test (for two groups comparison) or Kruskal Wallis (for more than two groups comparison). Categorical variables were expressed as number and percentage and analyzed with Chi-square test. Receiver operating characteristic curve (ROC) was used to evaluate predictive value of SII in morbidity. Spearman's correlation test was used to examin the possible correlation of SII with other continuous variables. A p- value less 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 ≥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 (LAD, LCX, RCA, and 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±1.42×10^9/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 had a p≤0.1 in the univariate analysis were entered the model. The results are shown in Table 3. Each of history of MI (p=0.038, OR=3.34, 95% 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

Receiver operating characteristic (ROC) curve was used to evaluate the predictive valve SII as 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 was 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 Hb (r=-0.256, p=0.007). Otherwise, no signification 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).

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. 480 Chinese patients with acute STEMI were included in Zhang *et al*.'s study.[8] The AUC=0.839 (95% CI: 0.752-0.927; p<0.001), the best cutoff 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 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 MACE linked to higher SII values in a large study on patients who had previously undergone CABG.[11] Even after controlling for conventional risk factors, elevated SII independently doubled the risk of MACE (HR≈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 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 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 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 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 cutoff 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.

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

Variables	Non-morbid (n=51)	Morbid (n=57)	p-value
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, k/m^2			
Mean± SD	25.98±3.34	27.25±3.97	0.075
Range	20.0-34.4	21-35.7	
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	

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

Variables	Non-morbid (n=51)	Morbid (n=57)	p-value
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	

Table 3. Multivariate analysis for factors associated with morbidity in diabetic patients with STEMI.

Variable	Regression coefficient (B)	p-value	Odds ratio	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

Table 4. Spearman's correlation of SII with other variables.

Variable	SII	
	Coefficient	p-value
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

Table 5. Association of SII with categorical variables.

Variable	SII	p-value
Sex		
Male	514.3(128.8-1789.0)	**<0.001**
Female	1009.2(695.7-1520)	
Smoking		
No	557.15(151.3-1789.0)	**0.026**
Yes	888.7(128.8-1789.0)	
Hypertension	514.3(270.0-1291.2)	0.053
	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)	
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)	

Data were expressed as median and range.

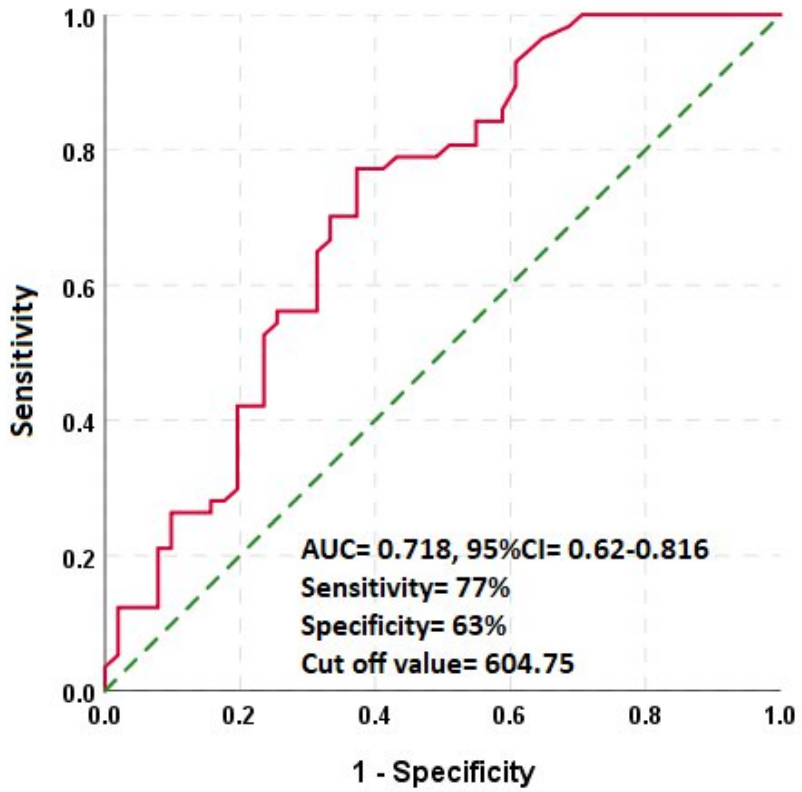

Figure 1. Receiver operating characteristic curve for SII as predictor for morbidity in diabetic patients with STEMI.