RESEARCH ARTICLE

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The Prognostic Value of the Systemic Immune-inflammation Index in ST-segment Elevation Myocardial Infarction Patients and Its Correlation with Syntax II Score and TIMI Risk Score

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Abstract

Background and Aim: The systemic immune-inflammation index (SII) has been identified as a novel prognostic marker in various illnesses. We investigated the relationship between SII and mortality in patients undergoing primary percutaneous coronary intervention (pPCI). In addition, we planned to examine how SII correlated with SYNTAX II and thrombolysis in myocardial infarction (TIMI) risk scores in this population.

Materials and Methods: This retrospective observational study included patients with ST-segment elevation myocardial infarction who underwent pPCI. The endpoint was 1 year all-cause mortality. SII [(neutrophil x platelet)/lymphocyte] was calculated from admission blood samples. Besides clinical and laboratory findings, SII, Syntax II and TIMI risk scores were compared between survivors and non-survivors. The correlation between SII and Syntax II and TIMI risk scores was also evaluated.

Results: The study included 334 patients (82.3% male). In the 1 year follow-up, 18 patients (5.4%) died. The SII, Syntax II, and TIMI risk scores were significantly higher in non-survivors than in survivors [mean (standard deviation: SD), 2423 (2005) vs 1686 (998), $P = 0.005$; median (interquartile range) 43 (35-53) vs 30 (25-37), $P < 0.001$; and 4 (2-5) vs 2 (1-3), $P = 0.005$, respectively]. Furthermore, the Syntax II score, TIMI risk score, and SII was independent predictors of 1 year all-cause mortality. SII showed a significant correlation with Syntax II and TIMI risk scores ($R^2 = 0.28$, $P = 0.001$ and $R^2 = 0.37$, $P < 0.001$, respectively).

Conclusion: SII might provide additional prognostic data alongside Syntax II and TIMI risk scores in patients undergoing pPCI.

Keywords: Systemic immune-inflammation index, STEMI, PCI, Syntax II score, TIMI risk score

INTRODUCTION

Cardiovascular diseases (CVDs) are the leading cause of mortality worldwide.[1] Around 18 million people die from ischemic heart disease (IHD) yearly, and atherosclerosis is a key contributing factor.[2,3] In the context of IHD, ST-segment elevation myocardial infarction (STEMI) is more common than non-ST-segment elevation myocardial infarction.[2] The primary cause of STEMI is the rupture of the coronary atherosclerotic plaque with subsequent thrombus development.[4]

According to studies, immunological and inflammatory responses play a significant role in all stages of STEMI development, including the progression of atherosclerosis,

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plaque rupture, and intraluminal thrombosis.[4-6] As part of the immune system, white blood cells, platelets, neutrophils, and lymphocytes play various roles in atherosclerosis and acute coronary syndrome.[7,8] Increased platelet counts indicate a prothrombotic state and harmful inflammatory activity.[7] Neutrophils can accelerate tissue damage by activating cytotoxicity, while lymphocytes regulate the inflammatory response and have a protective effect.[7,8]

The systemic immune-inflammation index (SII) was developed using neutrophil, platelet, and lymphocyte counts to evaluate the inflammatory and immunological states.[9] SII is considered an accurate prognostic indicator in many conditions, including cancer and CVDs.[9-11] However, limited scientific publications are available investigating SII association with long-term prognosis in patients with STEMI. Our objective was to investigate the relationship between SII and 1 year all-cause mortality in patients undergoing primary percutaneous coronary intervention (pPCI). We also planned to look at how SII correlated with conventional scoring systems, the Syntax II score, and the thrombolysis in myocardial infarction (TIMI) risk score in this population.

MATERIALS AND METHODS

Patients with STEMI undergoing pPCI between September 01, 2019 and June 30, 2021 were included retrospectively in this observational study. Patients with severe valvular heart disease, cardiogenic shock, active infection, history of coronary revascularization, oncological illness, and liver or kidney disorders were excluded. In addition, patients whose follow-up data could not be retrieved or who had incomplete data were not included. The study endpoint was 1 year all-cause mortality. The study was performed according to the 2008 revision of the Declaration of Helsinki. Kafkas University Ethics Committee approved the study (decision no: 80576354-050-99/260, date: 24.02.2023).

STEMI was diagnosed based on a recently accepted definition.[12] The hospital database was used to obtain the patients' demographics, comorbidities, admission laboratory results, and angiographic views. The formula used to determine SII was (neutrophil count x platelet count)/lymphocyte count.

Hypertension was described as a systolic blood pressure of ≥140 mmHg or a diastolic blood pressure of ≥90 mmHg in two measurements or antihypertensive medication. Smokers were defined as patients who had smoked continuously for at least six months in the previous year.

Patients with fasting glucose levels of ≥126 mg/dL or postmeal glucose levels of ≥200 mg/dL or using antidiabetic drugs were diagnosed with diabetes mellitus. Patients with dyslipidemia were identified as those whose serum low-density lipoprotein cholesterol was ≥140 mg/dL, triglyceride levels were ≥150 mg/dL, or high-density lipoprotein cholesterol was less than 40 mg/dL. The estimated glomerular filtration rate (eGFR) was determined using the Cockcroft-Gault formula. We used the modified Simpson technique to quantify the left ventricular ejection fraction (LVEF).[13]

Coronary artery angiography (CAG) was conducted through the femoral artery using the Seldinger technique. Before CAG, patients received 300 mg of acetylsalicylic acid, a loading dose of P2Y12 inhibitors, and 70-100 U/kg of infractionated heparin. Two experienced cardiologists blinded to the data thoroughly examined the angiographic views of the patients. Lesions in coronary arteries with stenosis of ≥50% and ≥1.5 mm in diameter were recorded using the online Syntax Score calculator (https://syntaxscore.org/calculator/start.htm, accessed in March-April 2023). An online calculator was used to obtain the Syntax II score[14] (https://syntaxscore.org/calculator/start.htm, accessed in March-April 2023). Variables for the Syntax II score were age, gender, chronic obstructive pulmonary disease, peripheral arterial disease, creatinine clearance, and LVEF. The TIMI risk score for STEMI was calculated using an online calculator (https://www.mdcalc.com/calc/99/timi-risk-score-stemi, accessed in March-April 2023).

Clinical follow-up information was acquired using the hospital and pharmacy databases or by calling patients or their relatives on the phone. Death certificates from the governmental database were used to confirm the death.

Statistical Analysis

SPSS software, version 21.0, was used for the statistical analysis. The normality test was maintained by using the Kolmogorov-Smirnov test. Continuous variables that showed a normal distribution were represented as mean [standard deviation (SD)] and those that did not show a normal distribution were expressed as median [interquartile range (IQR)]. Categorical data were represented as numbers (percentages) and analyzed using Pearson chi-square or Fisher's exact tests. The independent Student's t-test or Mann-Whitney U test was used to analyze continuous variables. Univariate regression analysis was performed for variables that differed significantly across the groups. A multivariate logistic regression analysis, including age, eGFR, Syntax II score, TIMI risk score, and SII, was used to describe the independent predictors of mortality. Data are displayed as odd ratios [95% confidence intervals (CI)]. Receiver operating characteristic (ROC) curve analysis was also used to indicate the performance of SII for predicting mortality. Pearson correlation analysis was performed to show the correlation between SII and Syntax II and TIMI risk scores. A *P*-value of 0.05 was used as the statistical significance threshold.

RESULTS

Our study included 334 patients (82.3% male). Table 1 represents the demographic characteristics and laboratory findings. The mean age of the patients was 56.6 ± 11 years. A total of 18 patients (5.4%) died during the 1-year follow-up period.

Non-survivors were older than survivors [age, years mean, standart deviation (SD), 62.8 (10.8) vs 56.2 (11), P =0.013]. Creatinine levels were significantly higher, whereas hemoglobin levels were significantly lower in non-survivors than in survivors [median (IQR), 1.04 (0.86-1.6) vs 0.89 (0.77-1), P = 0.019 and 12.4 (11.25-14.1) vs 13.9 (13-15), P = 0.015, respectively]. In non-survivors, eGFR was also significantly lower [mean (SD) 69 (28) vs 86 (23), P = 0.003]. Furthermore, non-survivors had significantly higher SII, TIMI risk score, and Syntax II score [mean (SD), median (IQR), 2423 (2005) vs 1686 (998), P = 0.005;43 (35-53) vs 30 (25-37), P < 0.001; and 4 (2-5) vs 2 (1-3)], P = 0.005, respectively]. In univariate analysis, age, eGFR, Syntax II score, TIMI risk score, and SII were associated with mortality [odds ratio (OR) (95% CI), 1.054 (1.010-1.100), P = 0.015; 0.968 (0.946-0.989), P = 0.003; 1.089 (1.048-1.132), P < 0.001; 1.325 (1.069-1.642), P = 0.009; and 1.030 (1.028-1.062), P = 0.011, respectively]. According to multivariate analysis, the Syntax II score, TIMI risk score, and SII were independent predictors of mortality [OR (95% CI), 1.084 (1.031-1.139), P = 0.002; 1.068 (1.014-1.361), P = 0.012; and 1.016 (1.008-1.068), P = 0.048, respectively] (Table 2). The results of univariate and multivariate analyzes are presented in Table 2. In ROC curve analysis (Figure 1), a cutoff value of 1820 for SII predicted mortality with a sensitivity of 61% and specificity of 63% [area under the curve (AUC) was 0.628; P = 0.047)], AUC for TIMI risk and Syntax II scores was 0.689 (P = 0.007) and 0.826 (P < 0.001), respectively. In the Pearson correlation test,

Table 1: Demographic and laboratory findings

	Total (n=334)	Survivors (n=316)	Non-survivors (n=18)	P-value
Male, n (%)	275 (82.3)	262 (82.9)	13 (72.2)	0.247
Age (years), mean (SD)	56.6 (11)	56.2 (11)	62.8 (10.8)	0.013
SBP (mmHg), median (IQR)	135 (120-1447)	135 (120-148)	131 (90-139)	0.316
DBP (mmHg), median (IQR)	80 (70-90)	80 (70-90)	76 (56-90)	0.278
Heart rate, median (IQR)	80 (70-88)	80 (70-88)	81 (65-98)	0.588
SII, mean (SD)	1725 (1084)	1686 (998)	2423 (2005)	0.005
Syntax II score, median (IQR)	31 (25-39)	30 (25-37)	43 (35-53)	<0.001
TIMI risk score, median (IQR)	2 (1-4)	2 (1-3)	4 (2-5)	0.005
Laboratory				
Hemoglobin (g/dL) median (IQR)	13.9 (13-15.1)	13.9 (13-15)	12.4 (11.25-14.1)	0.015
WBC ($\times10^3$/µL), median (IQR)	12.83 (11.18-14.59)	12.8 (11.2-14.35)	13 (10.22-17.57)	0.762
Neutrophil ($\times10^3$/µL), median (IQR)	10.2 (8-12)	10 (8-11.7)	12.05 (8.8-14.7)	0.066
Lymphocyte($\times10^3$/µL), median (IQR)	1.8 (1.29-2.5)	1.8 (1.3-2.5)	1.35 (1.07-2.17)	0.078
Platelet ($\times10^3$/µL), median (IQR)	260 (222-298)	260 (222-298)	227 (214-297)	0.988
Troponin I (ng/mL) median (IQR)	2.66 (0.81-5.78)	2.4 (0.75-5.6)	3.83 (2.25-8.13)	0.091
CK-MB (ng/mL) median (IQR)	35 (25-47)	35 (25-46)	42.5 (29.2-61.7)	0.181
Creatinine (mg/dL) median (IQR)	0.9 (0.78-1.03)	0.89 (0.77-1)	1.04 (0.86-1.6)	0.019
eGFR, mean (SD)	85 (23)	86 (23)	69 (28)	0.003
Glucose (mg/dL), mediyan (IQR)	129 (108-172)	129 (108-172)	138 (100-293)	0.477
Comorbities				
Hypertension, n (%)	153 (45.8)	144 (45.6)	9 (50)	0.714
Diabetes, n (%)	77 (23.1)	70 (22.2)	7 (38.9)	0.101
Smoking, n (%)	190 (56.9)	180 (57)	10 (55.6)	0.907
COPD, n (%)	19 (5.7)	17 (5.4)	2 (11.1)	0.307
PAD, n (%)	51 (15.3)	46 (14.6)	5 (27.8)	0.168
Dyslipidemia, n (%)	138 (41.3)	134 (42.4)	4 (22.2)	0.091

CK-MB: Creatine kinase-MB, COPD: Chronic obstructive pulmonary disease, DBP: Diastolic blood pressure, eGFR: Estimated glomerular filtration rate, IQR: Interquartile range, SBP: Systolic blood pressure, SII: Systemic immune-inflammation index, SD: Standard deviation, PAD: Peripheral artery disease, TIMI: Thrombolysis in myocardial infarction, WBC: White blood cell count

SII was significantly correlated with the TIMI risk score and the Syntax II score ($R^2 = 0.37$, $P < 0.001$ and $R^2 = 0.28$, $P = 0.001$, respectively) (Figure 2).

DISCUSSION

The main findings of our study are that; 1) age, eGFR, Syntax II score, TIMI risk score, and SII were significantly associated with 1 year all-cause mortality in patients undergoing pPCI, 2) the Syntax II score, TIMI risk score, and SII were independent predictors of mortality, and 3) SII, albeit weakly, showed a positive correlation with the TIMI risk score and Syntax II score.

Despite technological and therapeutic advances, mortality remains high in patients with STEMI.[1,2] In this sense, the investigation of factors related to clinical outcomes is critical in terms of preventing mortality. Age showed a positive association with mortality in our study. Similarly, previous reports have established that age is associated with both short- and long-term mortality after pPCI.[15,16] Coronary artery characteristics also play a critical role in the prognosis of CVD. Several scoring systems have been established previously in this respect.[14,17] Syntax II and TIMI risk scores are the most studied.[18] In agreement with the literature, our study demonstrated that Syntax II and TIMI risk scores were associated with mortality and were independent predictors of mortality. The Syntax II score calculated from clinical and angiographic variables, accurately predicted 1-year mortality in patients with STEMI.[19] Besides, early reports showed that the TIMI risk score also predicts in-hospital and 1-year death in this population.[20,21] Inflammation plays a key role in all stages of STEMI, including the formation, evolution, and dissection of the plaque and thrombus.[4-6,22,23] SII has recently been proposed as a possible marker based on inflammatory cells associated with poorer outcomes in several disorders, including CVD.[9-11,24] In patients with CVD, the elevation of standard inflammatory markers, e.g., white blood cell count or C-reactive protein, was not only observed, but also associated with atherosclerotic plaque instability and mortality due to CAD.[25,26] Nevertheless, these counts are susceptible to various factors, such as dehydration and fluid overload.[27] SII appears to be more stable and better predicts adverse cardiovascular outcomes than the standard blood counts.[28] Many studies have investigated the relationship between SII and adverse outcomes in CVD. Erdogan et al.[29] showed a significant association between SII and CAD severity. Dey et al.[10] found a relationship between SII and poor postoperative results following off-pump coronary artery bypass surgery. Agus et al.[30] reported that patients with infective endocarditis had an independent relationship between SII and in-hospital mortality. According to Yang et al.,[9] SII was an independent predictor of unfavorable outcomes in patients with STEMI, non-STEMI, and stable angina pectoris. The latter included a heterogeneous coronary artery disease group, but our study included a homogeneous group (only STEMI). A recent report by Saylik and Akbulut[24] showed the role of SII in predicting major adverse cardiovascular events in 843 patients undergoing pPCI. This study included a larger population with a more extended follow-up period. However, it differed in methodology from our study. Furthermore, they did not study the correlation of SII with traditional risk scores. Overall, similar to these publications,

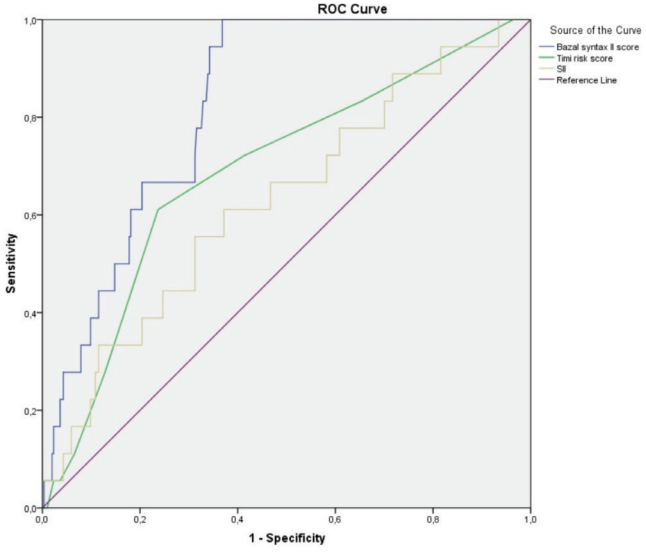

Figure 1: The receiver operating characteristic (ROC) curve for predicting 1 year all-cause mortality using the systemic immune-inflammation index (SII). The area under the curve is 0.628 (cut-off value: 1820, sensitivity: 61%, specificity: 63%).

Table 2: Univariate and multivariate regression analyzes for predicting 1 year all-cause mortality

	Univariate		Multivariate	
	OR (95% CI)	***P*-value**	**OR (95% CI)**	***P*-value**
Age	1.054 (1.010-1.100)	0.015	1.001 (0.946-1.059)	0.972
eGFR	0.968 (0.946-0.989)	0.003	0.996 (0.969-1.023)	0.749
Syntax II score	1.089 (1.048-1.132)	<0.001	1.084 (1.031-1.139)	0.002
TIMI risk score	1.325 (1.069-1.642)	0.009	1.068 (1.014-1.361)	0.012
SII	1.030 (1.028-1.062)	0.011	1.016 (1.008-1.068)	0.048

CI: Confidence interval, eGFR: Estimated glomerular filtration rate, OR: Odds ratio, SII: Systemic immune-inflammation index, TIMI: Thrombolysis in myocardial infarction

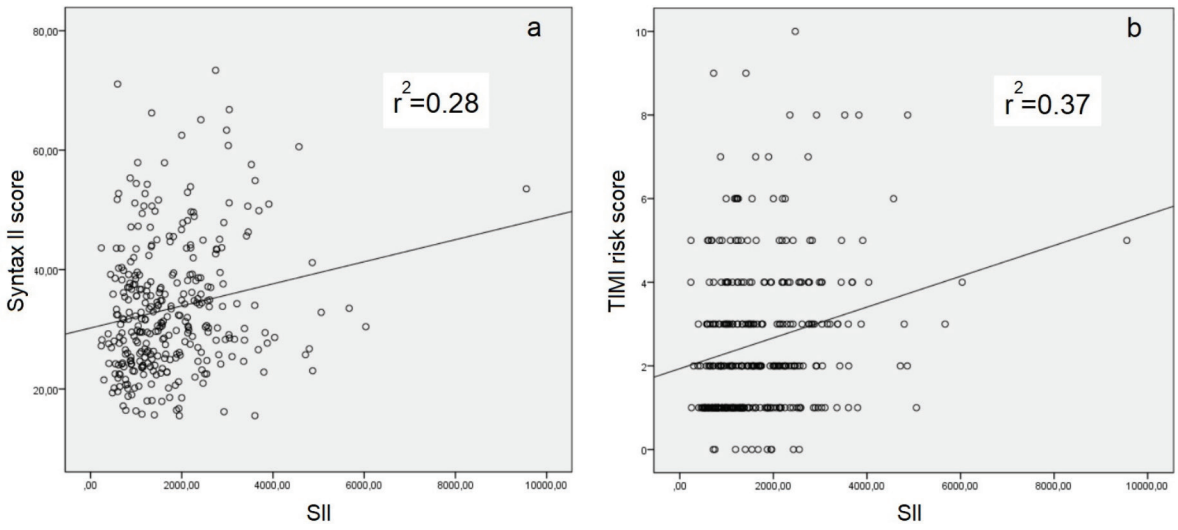

Figure 2: Correlation graphics between the systemic immune-inflammation index (SII) and Syntax II score (**a**) and TIMI risk score (**b**)

our study revealed a relationship between SII and 1 year all-cause mortality. Moreover, we demonstrated a 1 year predictive value of SII in a particular patient group (STEMI). Furthermore, we showed a positive correlation between SII and TIMI risk and Syntax II scores.

Consequently, predicting adverse outcomes early in risky STEMI patients who undergo pPCI is crucial for prioritized treatment. In this context, SII may play an essential prognostic role in risk stratification for these patients, alongside the Syntax II score and TIMI risk score.

Study limitations

Our study has main limitations were the relatively small size of the study population and the single-center and retrospective design. In addition, our endpoint was all-cause mortality, and we could not provide the exact cause of death. Large randomized controlled studies are needed to confirm the predictive value of SII in STEMI patients receiving p-PCI.

CONCLUSION

Our study found a positive correlation between SII and Syntax II and TIMI risk scores in patients undergoing pPCI. SII and Syntax II and TIMI risk scores were associated with 1 year all-cause mortality. Thus, SII, as an easily calculable marker, might provide additional prognostic data alongside Syntax II and TIMI risk scores in patients undergoing pPCI.

Ethics

Ethics Committee Approval: Kafkas University Ethics Committee approved the study (decision no: 80576354-050-99/260, date: 24.02.2023).

Informed Consent: Retrospective study.

Peer-review: Externally and internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: M.K., İ.A., Y.K., İ.R., Concept: M.K., Design: T.O., Data Collection or Processing: B.A., Analysis or Interpretation: C.D., İ.R., Literature Search: A.A., Writing: T.O.

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