ORIGINAL ARTICLE

Eastern Journal of Medicine
East J Med 27(4): 657-663, 2022
DOI: 10.5505/ejm.2022.94824

Systemic Immune-Inflammation Index in Patients with ST-Elevated Myocardial Infarction Undergoing Percutaneous Coronary Intervention

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ABSTRACT

Our aim was to assess value of systemic immune-inflammation index (SII) to predict coronary artery disease severity in patients with ST elevated myocardial infarction (STEMI) who underwent percutaneous coronary intervention (PCI).
Methods: One hundred twenty six patients diagnosed as STEMI who underwent primary PCI were retrospectively analyzed. The severity of coronary stenosis was determined by the SYNTAX score (SxS). Patients with SxS<22 constituted group 0 and ≥22 constituted group 1. SII was obtained by multiplying number of platelets by neutrophil to lymphocyte ratio (SII = p × n/L ratio).
Lymphocyte count, ejection fraction and hematocrit was lower in patients with high SxS. Patients with higher SxS, had higher SIIs than those with lower SxS (1073.85 (147.28-6142.84) vs (778.88 (228.71-3179.78) p=0.044 respectively). SII had positive correlation with SxS, glucose, troponin T, creatinine, CK-MB levels and WBC count. SII was found to be predictor of high SxS (odds ratio: 1.001; 95% CI: 1.000-1.001; P = 0.018). SII with a cutoff value of 1124.7679 predicted the severity of coronary lesion with a sensitivity of 48% and specificity of 73.5% (AUC: 0.606, 95% CI: 0.507–0.705, p=0.46).
Pre-procedural SII might provide an information about atherosclerotic burden in coronary artery in STEMI patients.

Keywords: Myocardial infarction, platelet activation, systemic immune-inflammation index.

Introduction

Coronary artery disease (CAD) is still most important cause of mortality globally. Atherosclerosis, in which inflammatory pathyways participates in the pathophysiology, is a key driver factor for onset, progression and complications of CAD (1). Elevated levels of inflammatory biomarkers have been shown to be associated wih increased cardiac complication rates in patients with CAD (2).

White blood cells (WBC) and platelets secrete variety of inflammatory mediators. Since the central role of inflammation in the formation of CAD is known, WBC and its subtypes have been used as inflammatory markers in cardiovascular diseases (3,4). Increased number of neutrophils is highly predictive of atherosclerotic coronary artery disease severity (4,5). Discovery of role of platelets in atherosclerosis pathophysiology has been raised considerable interest concerning function of platelets in severity of CAD (6). Determination of patients who have higher risk of cardiac complications is very important. Accordingly, In addition to classical risk factors and comorbidities, there is still a need for easily accessible and highly accurate markers in daily practice to predict the severity of CAD.

Systemic immune-inflammation index (SII) is a novel predictor which provides information about patients' inflammatory and immune status simultaneously (7,8). Its calculation is based on neutrophil, platelet and lymphocyte counts and it has been shown to predict adverse events in cancer patients. Hence it could be used as a inflammatory marker in early disease for identification of those with higher risk of complications (8). Even more, it predicted mortality in cardiovascular patients, including CAD and acute coronary syndrome (9,10).

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Received: 25.12.2021, **Accepted:** 23.05.2022

SYNTAX II score (SxS) is an important angiographic practical tool that combines anatomical and clinical factors into a single parameter. It predicts post-procedural complications and also calculates complexity of CAD (11). In other words, high SxS is related to more complex lesions and worse outcomes following PCI.

Severity of atherosclerotic disease has paramount importance for prognostic status of CAD patients (12). In this context, it is important in the clinical practice to have an idea about complexity of the lesion and the atherosclerotic burden in patients with STEMI. Since the SII is an inexpensive, easily obtainable parameter from complete blood count (CBC), its predictive value for CAD severity in STEMI patients was evaluated in the present study.

Materials and Methods

One hundred twenty six STEMI patients who underwent primary percutaneous coronary intervention (PCI) at a tertiary centre were retrospectively analyzed between December 2018 and December 2019. Ethical Committee of our hospital was approved the study and it was directed in conformity with the declaration of Helsinki.

Patients with acute or chronic infection, history of stroke, autoimmune diseases, sepsis, trauma, recent major surgery, active malignancy, liver and kidney failure were exclusion criteria for this study. Patients' clinical and biochemical findings were extracted retrospectively from patients records. As above mentioned the severity of CAD was determined by the SxS. Patients with SxS<22 constituted group 0 and ≥22 constituted group 1.

Venous blood samples of patients were drawn from the antecubital vein before PCI, within 6 hours after hospital admission. Blood samples were analyzed for fasting blood glucose (FBG), troponin I and T, creatine kinase MB fraction (CK-MB), serum creatinine, total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C). Blood samples were analyzed by laboratory auto analyzer and complete blood count including platelet, neutrophil, and lymphocyte numbers were obtained. SII was obtained by multiplying number of platelets by neutrophil to lymphocyte ratio (SII = $p \times n$/L ratio) (6).

STEMI was described as typical chest pain, an increase in myocardial necrosis biomarkers, and presence of ST elevation in at least two consecutive leads or new left bundle branch block on 12-lead surface electrocardiogram (13).

Body mass index was derived by dividing weight (kg) to squared height (m2). Diagnosis of hypertension was made if patients had systolic and/or diastolic blood pressure greater than 140 mmHg and 90 mmHg, respectively and if they took antihypertensive drugs. TC concentration greater than 220 mg/dL, LDL-C concentration greater than 130 mg/dL, or antilipidemic medication usage was considered as dyslipidemia. Diabetes mellitus was described as HbA1c level greated than 6.5 %, random plasma glucose greater than 220 mg/dL, or antidiabetic medication usage.

All PCI procedures and treatment strategies after PCI were under the authority of the clinician performing the procedure. SxS is an angiographic and clinical tool for the quantification of complexity of CAD (11). SxS of each patient was calculated according to algorithm which includes anatomical and clinical variables including number of lesions, lesion location, presence of bifurcation and/or trifurcation lesions, ostial stenosis, tortuosity, lesion length more than 20 mm, calcification, thrombus, small vessel or diffuse disease, age, sex, renal function, ejection fraction, left main disease, chronic obstructive pulmonary disease and peripheral vascular disease.

Statistical Analysis: Normality of the data was tested by Kolmogorow-Smirnow test. Parameters which had normal and non-normal distribution were expressed as mean±SD and median (min-max), respectively Correlaton of SII with other parameters was done by Spearman rank correlation test. Comparison of groups was made by independent samples t-test and Mann-Whitney U test. ROC curve analysis was used to determine the predictive values of SII and syntax score on mortality of the patients. For identification of predictors of high SxS, logistic regression analysis was used. All statistical calculations were done by IBM SPSS statistics 25 software. P value of less than 0.05 was considered as significant.

Results

The mean age of the study population was 55.13±12.22 years, and 111 (88.1%) patients were male. Patients were divided into two groups according to their SxS score. Patients with SxS of 21 and lower and 22 and upper constituted group

Table 1. Clinical and Biochemical Characteristics of The Study Population

Parameter	
Gender (n. %)	
Female	15 (11.9)
Male	111 (88.1)
Age (years)	55.13±12.22
Troponin I (ng/ml)	0.31 (0.002-92.0)
Troponin T (ng/ml)	0.109 (0.003-182)
Creatinin kinase-MB	6.21 (1.01-388)
WBC (103/μL)	11.57±3.52
Neutrophil (103/μL)	8.36 (2.17-21.99)
Lymphocyte (103/μL)	2.49 (0.57-8.23)
Platelet (×109 /l)	262±59.30
SII	969.46 (147.28-6142.84)
Hct (%)	42.15 (25.6-49.8)
Syntax score 2	24.2±8.08
EF (%)	50 (25-65)
LDL-C (mg/dl)	134.71±40.61
HDL-C (mg/dl)	106 (4-810)
Glucose (mg/dl)	119 (74-374)
Creatinine (mg/dl)	0.9 (0.6-2.2)
CRP (mg/l)	4.22 (0.23-27.5)

CRP: C reactive protein, EF: Ejection fraction, Hct: Hemaotcrit, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, SII: Systemic immune-inflammatory index, TG: Triglyceride, WBC: White blood cell. .Continuous variables are presented as mean ± SD or median (min-max), nominal variables presented as frequency (%)

0 and 1, respectively. Table 1 depicts clinical and demographic features of the study patients.

In terms of laboratory variables, troponin I and T, CK-MB, lipid profiles, WBC, neutrophil and platelet counts were not different between two group of patients ($p<0.001$) (Table 2). However lymphocyte counts, ejection fraction (EF) and hematocrit was lower in patients with high SxS. Remarkably we also observed that patients with higher SxS, had higher SIIs than those with lower SxS. SII showed weak correlation with SxS ($r=0.173$, $p= 0.046$). Correlation analysis of SII with other parameters is shown in Table 3.

According to univariable regression analysis SII, hematocrit, age, EF and creatinine were found to be the predictors of high SxS. Multivariate logistic regression analysis showed that SII, age, EF, creatinin were independent predictors for disease severity (Table 4 and 5 show the univariate and multivariate regression results).

According to ROC curve analysis, SII detected the severity of coronary lesion with a cut-off point of 1124.7679 with sensitivity and specificity of 48% and 73.5% respectively (AUC: 0.606, 95% CI: 0.507–0.705, p=0,46, Fig. 1).

Discussion

In this study we have shown that patients with increased SxS had higher SII values compared to patients with lower SxS. Moreover SII had a predictive value for the severity of coronary artery disease. Our study was the first one conducted to assess the value of SII in predicting coronary lesion severity in this group of patients.

WBC and its subtypes have been shown to be useful in acute myocardial infarction (AMI) patients for prognostic risk stratification, but risk models based on one or two inflammatory parameters might have relatively low predictive value in prognosis in AMI (14,15). Therefore, we evaluated predictive abilities of SII and each parameter through univariate and multivariate logistic regression analyses. After adjustment of confounding factors, variables including SII, age, creatinine, and EF had predicted the disease severity.

Many studies proven that platelets have a pivotal role in the pathogenesis of CAD (16). Persistent inflammation due to thrombocytosis predisposes to a prothrombotic state (17). After endothelial dysfunction, aggregated platelets adhere to endothelial

Table 2. Comparision of Clinical and Biochemical Variables of Two Groups

Parameter	Group 0 (n=49) (SxS<22)	Group 1 (n=77) (SxS ≥22)	p
Gender			0.021*
Female (n,%)	2 (4.1)	13 (16.9)	
Male (n,%)	47(95.9)	64 (83.1)	
Age (years)	47.51±8.9	60.40±11.44	<0.001*
Troponın I (ng/ml)	0.256 (0.003-92.0)	0.325 (0.002-63)	0.989
Troponin T (ng/ml)	0.055 (0.003-51)	0.124 (0.004-182)	0.182
CK-MB (U/l)	6 (1.17-388)	7.37 (1.01-313)	0.343
WBC (103/μL)	12.08 (4.55-27.67)	11.33 (5.32-22.9)	0.481
Neutrophil (103/μL)	8.52±3.62	8.69±3.32	0.782
Lymphocyte (103/μL)	2.75 (1.21-5.86)	1.98 (0.57-8.23)	0.003*
Platelet (×109 /l)	270.06±48.61	271.00±68.02	0.934
SII	778.88 (228.71-3179.78)	1073.85 (147.28-6142.84)	0.044*
Hct (%)	43.49±3.21	41.03±4.56	0.001*
EF (%)	53 (40-65)	47 (25-65)	0.018*
LDL-C (mg/dl)	141.00±33.44	130.12±44.88	0.172
HDL-C(mg/dl)	39.88±7.03	39.98±8.79	0.950
TG (mg/dl)	132 (44-810)	110 (4-272)	0.010*
Glucose (mg/dl)	107 (84-319)	124 (74-374)	0.024*
Creatinine (mg/dl)	0.87±0.131	0.907±0.259	0.025*
CRP (mg/l)	4.03(0.23-19.88)	4.83 (0.34-27.5)	0.154

CRP: C reactive protein, EF: Ejection fraction, Hct: Hematocrit, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, SII: Systemic immune-inflammatory index, TG: Triglyceride, WBC: White blood cell. .*p<0,05 Continuous variables are presented as mean ± SD or median (min-max), nominal variables presented as frequency (%)

Table 3. Correlation of SII With Other Parameters

	r	p
Syntax score	0.173	0.046
Glucose	0.223	0.015
Age	0.073	0.418
Troponin T	0.247	0.008
EF	0.004	0.969
Creatinine	0.218	0.015
CRP	0.161	0.095
HDL -C	0.036	0.708
LDL *C	0.035	0.717
CK-MB	0.284	0.002
WBC	0.455	<0.001
Hct	0.162	0.072

CRP: C reactive protein, EF: Ejection fraction, Hct: Hematocrit, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, WBC: White blood cell

cell surfaces, causing monocytes to recruit to the arterial wall. Activated platelets produces proinflammatory cytokines, matrix metalloproteinases, and reactive oxidative species all of which play an essential role in pathogenesis and rupture of atherosclerotic plaque (18).

Lymphocytes are also considered as having a potential impact in cardiovascular disease. Lymphocytopenia is commonly related to chronic

Table 4. Univariate Analysis For Prediction of High SxS

	Univariate	
Parameter	Odds Ratio (95% CI)	p
SII	1.001 (1.000 – 1.001)	0.018
Hct	0.853 (0.770– 0.945)	0.002
Troponin T	1.007 (0.985 -1.030)	0.522
Age	1.133 (1.078 – 1.191)	0.000
EF	0.932 (0.887 -0.980)	0.006
LDL-C	0.994 (0.984 – 1.003)	0.190
Creatinine	13.134 (1.455 -118.518)	0.022
CRP	1.033 (0.965 – 1.105)	0.351

EF:Ejection fraction, Hct: Hematocrit, LDL-C; Low density lipoprotein cholesterol, CRP;C-reactive protein, Hct: Hematocrit, LDL-C; Low density lipoprotein cholesterol, CRP;C-reactive protein, SII: Systemic immune-inflammatory index

Table 5. Multivariate Analysis For Prediction of High SxS

	Multivariate	
Parameter	Odds Ratio (95% CI)	p
SII	1.001 (1.000 – 1.001)	0.026
Age	1.184 (1.103 – 1.270)	0.000
EF	0.856 (0.788 -0.930)	0.000
LDL-C	0.994 (0.984 – 1.003)	0.190
Creatinine	44.577(1.405 -1414.430)	0.031
CRP	1.033 (0.965 – 1.105)	0.351

CRP: C-reactive protein, EF: Ejection fraction, LDL-C: Low density lipoprotein cholesterol, SII: Systemic immune-inflammatory index

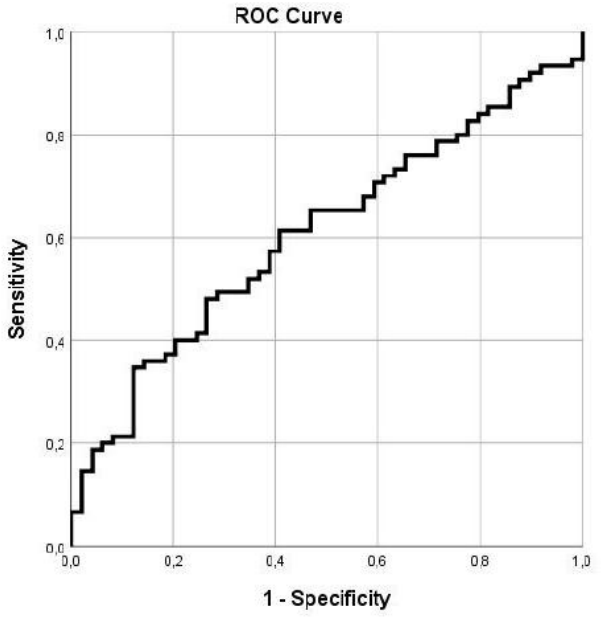

Fig. 1. ROC Curves Analysis Showing The Predictive Cutoff Value of SII in STEMI Patients

inflammatory status and adverse events in patients with acute coronary syndromes (19,20). Nunez et al showed that chest pain patients with a non-diagnostic ECG and normal troponin levels had increased risk of AMI and mortality if they had low lymphocyte count than that of controls (21). Therefore, combining these three risk factors in a simple formula might more comprehensively repesents immune and inflammatory status of the host and coronary lesion severity (22,23). Results from previous studies suggested that patients with elevated SII had greater risk for unfavorable health outcomes (24). As such, an elevated value of SII with increased numbers of neutrophis and platelets and decreased numbers of lymphocytes may be linked to high level of inflammatory activity and poor clinical outcomes. In our study, increased SII was found in STEMI patients. Moreover, our study suggested that an SII 1124.7679 predicted CAD severity with a sensitivity of 48% and specificity of 73.5%.

SxS scoring system, which is an objective and reliable tool for evaluation of coronary lesion severity, was used in our study. Previous studies have reported that high SxS score have been related with worse cardiovascular outcomes (25, 26). SII was an independent predictor for high SxS in multivariate analysis, which suggested that patients with severe coronary artery disease might have relatively higher SII values and this parameter may be helpful to

clinicans to evaluate coronary artery disease severity and risk stratification of the patients. Alvarez et al. showed that higher SxS in STEMI patients who underwent primary PCI added prognostic information related to midterm adverse outcomes (27). Yet other studies also demonstrated that it was a good tool for predicting major adverse cardiac events during follow-up (28,29) Since importance of risk assessment in STEMI is utmost importance for determining future adverse outcomes, this easily obtainable parameter might be performed at hospital admission in STEMI patients.

Limitations: Major limitation of our present study was the limited study population, and a single center study. Besides, other inflammatory markers were not evaluated in the study. Therefore, prospective studies in larger populations are needed to validate our conclusions.

Conclusions: We found that SII which is an indicator of inflammation, could be helpful in determining the severity of coronary artery lesion as a risk factor for atherosclerosis. Therefore, it could be considered that patients with STEMI with higher SII values also might have more atherosclerotic burden than patients with low values. SII which is an easily calculable biomarker, may be used daily by almost every physician for risk stratification of AMI patients.

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