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The use of systemic immune-inflammation index to predict new onset atrial fibrillation in the context of acute coronary syndrome

Aim	The objectives of this study were to determine the relationship between the systemic immune-inflammation index (SII) and new onset atrial fibrillation (NOAF) in patients with acute coronary syndrome (ACS), and to assess the use of this relation, if any, to predict NOAF in the context of ACS
Material and Methods	A total of 622 patients diagnosed with ACS and followed up between September 2019 and September 2021 were included in this study. 35 (5.6%) of these patients, suffering from NOAF, were designated as the patient group, and the remaining 577 (94.4%) patients were designated as the control group. SII was calculated with the formula [(platelet count x neutrophil count)/lymphocyte count] in all patients.
Results	SII was significantly increased in the NOAF group [1641 (778–4506) vs. 660 (54–2835); p<0.001. The multivariable logistic regression analysis revealed that SII [OR: 1.002, 95%CI: 1.001–1.002, p<0.001] is one of the independent predictors for NOAF, in addition to age (p=0.003) and left atrium size (p=0.005).
Conclusion	The SII index is an independent predictor of NOAF in ACS patients. This index can be used as an easily accessible value in the clinic. Assessment of risk factors for NOAF may permit early treatment and close follow-up of patients with poor prognosis who may develop AF.

Keywords	Acute coronary syndromes; atrial fibrillation; systemic immune-inflammation index
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Introduction

Cardiovascular disease (CVD) is the leading cause of world-wide mortality [1]. A significant portion of the mortality associated with CVD is attributed to acute coronary syndrome (ACS) [2], and atrial fibrillation (AF) is a common complication observed in ACS [3]. Despite that ACS, as a primary disease, is a cause of mortality and morbidity, coexistence of ACS and AF worsens the prognosis and increases major adverse events, including death [4]. As is the case with all CVDs, inflammation plays a major role in the pathogenesis of AF [5]. Various studies have demonstrated that the development of new onset atrial fibrillation (NOAF) following ACS was predicted by advanced age, high C-reactive protein (CRP), highN-terminal proB-type natriuretic peptide (NT-proBNP), D-dimer concentrations, coronary flow disorders, and impaired cardiac function [3, 6, 7].

The systemic immune-inflammation index (SII) is calculated using neutrophil, platelet, and lymphocyte counts. SII has been used to evaluate the prognosis and response to treatment in cases of many malignancies, such as urinary system cancers, glioblastoma, and hepatocellular carcinoma [8–10]. Recently, SII has been used also in clinical prediction and evaluation of prognosis in CVDs, such as coronary artery disease and carotid artery disease [11, 12].

In this study, considering the role of inflammation in atrial fibrillation, we investigated the relationship of SII, an inflammation index, with NOAF in ACS patients.

Material and methods

Research Design and the Study Population

This study was carried out as a single-center, cross-sectional, retrospective study. A total of 955 patients with a diagnosis of ACSand admission to the coronary intensive

care unit between September 2019 and September 2021were evaluated for inclusion in the study. Exclusion criteria were cardiogenic shock, pulmonary edema, active infection, malignancies, history of AF, or history of hematological or chronic inflammatory diseases. Consequentially, 622 patients were included in the study, and the demographic data of these patients were recorded. All patients were followed with in-hospital continuous ECG monitoring for at least 48 hr, and a 12-lead ECG was obtained twice daily during the hospital stay. Additionally, when patients had symptoms suggesting arrhythmia, such as palpitations or dyspnea, the rhythm was checked with a 12-lead ECG. The patients were divided into two groups in terms of NOAF development. The study protocol was approved by the local ethics committee in accordance with the Declaration of Helsinki and good clinical practices.

Laboratory and Echocardiographic Assessment

Complete blood count (CBC), kidney function, lipid panel, glucose, CRP, albumin, and high-sensitivity troponin-I concentrations were measured during admission to the coronary intensive care unit. CBC variables were automatically measured using the Mindray BC-5800 automated hematology analyzer (Mindray Medical Electronics Co. Shenzhen, China). All patient data were obtained from the hospital database. An echocardiographic examination was performed with the Philips IE33 system (Philips Medical Systems, Andover, MA, USA). Left ventricular (LV) ejection fraction (LVEF) was calculated using the Simpson's method. Mitral early filling inflow (E-wave), obtained using Doppler echocardiography, and the E'-wave, obtained from the mitral lateral annulus using tissue Doppler imaging (TDI), were recorded. The left atrium (LA) was measured from the apical parasternal long-axis view. All echocardiograms were obtained and interpreted by two experienced cardiologists blinded to the patients' enrollment in accordance with the recommendations of the American Society of Echocardiography (ASE) [13].

Calculation of Systemic Immune-Inflammation Index, Syntax Score and CHA2DS2-VASc Score

The SII was calculated using the formula: SII = [(platelet count × neutrophil count)/lymphocyte count]. SII was calculated from the CBCmeasured at at coronary care unit.

The SYNergy between PCI with TAXUS and Cardiac Surgery (SYNTAX) scores (SS) were calculated, as described in the literature [14], by two experienced operators blinded to other parameters using the SXscore online calculator. The CHA2DS2-VASc Score (C, congestive heart failure 1 point; H, hypertension history: 1 point; A2, age≥75: 2 point; D, diabetes history. 1 point;

Table 1. Baseline clinical characteristics of the study population

Parameters	NOAF Group, n=35	Control Group, n=587	p
Age (yrs)	69.14±12.26	62.55±12.28	0.002[a]
Sex (F/M) (n/%)	12/23	140/447	0.163[b]
BMI (kg/m²)	33.2 (22.04–40.6)	29.06 (16.49–46.57)	0.005[c]
STEMI	30 (86)	208 (35)	<0.001[b]
Hypertension	30 (86)	317 (54)	<0.001[b]
Diabetes Mellitus	13 (37)	142 (24)	0.085[b]
Hyperlipidemia	15 (43)	167 (28)	0.069[b]
Smoking	25 (71)	359 (61)	0.225[b]
Discharge time (days)	4 (3-6)	4 (2-6)	0.150[c]
Hemoglobin (g/dl)	13.1 (8.6–16.3)	14 (6.3–18.4)	<0.001[c]
WBC (×10⁹/l)	11.9 (5.34–22.25)	9.2 (4.07–22)	0.001[c]
RDW (%)	14.1 (12.1–16.7)	13.4 (11.1–19.6)	0.116[c]
PLT (×10⁹/l)	225 (97-374)	219 (96-702)	0.468[c]
Lymphocyte	1.22 (0.31–2.50)	2.03 (0.45–14.4)	<0.001[c]
Neutrophil	9.7 (4.22–19.82)	6.1 (1.69–19)	<0.001[c]
MPV (fl)	9.02±1.05	8.61±0.99	0.053[a]
Creatinine (mg/dl)	1 (0.43–3.41)	0.9 (0.32–6.87)	0.020[c]
Glucose (mg/dl)	118 (94-394)	114 (70-471)	0.558[c]
CRP (mg/l)	1.5 (0.2–30)	1.81 (0.1–25.46)	0.852[c]
Albumin (mg/dl)	3.83 (2.25–5.14)	4.2 (2.31–6.33)	<0.001[c]
LDL–C (mg/dl)	144 (64-292)	137 (41-288)	0.353[c]
HDL–C (mg/dl)	39.5 (18-63)	41 (20-288)	0.224[c]
Total Cholesterol (mg/dl)	167.5 (116-323)	199 (43-444)	0.020[c]
Triglyceride (mg/dl)	116 (45-472)	120 (0.87–824)	0.836[c]
Hs-Troponin I (ng/l)	20.36 (0.38–129.87)	8 (0.05–425.09)	0.094[c]

Data are mean±SD, median (minimum – maximum), or as number (percentage). [a] Independent t test. [b] Chi-square test. [c] Mann–Whitney U test. ACS, acute coronary syndrome; BMI, body mass index; CRP, C reactive protein; HDL–C, high density lipoprotein cholesterol; hs-Troponin I, high-sensitivity troponin I; LDL–C, low density lipoprotein cholesterol; MPV, mean platelet volume; PLT, platelet; RDW, red cell distribution width; STEMI, ST elevation myocardial infarction; WBC, white blood cell.

Table 2. Comparison of the echocardiographic and index data

Parameters	NOAF Group, n=35	Control Group, n=587	p
LV-EF (%)	45 (25–70)	58 (20–75)	<0.001[a]
E/E'	16 (10.6–21.40)	11.6 (0.09–45.5)	<0.001[a]
LA size (mm)	37 (32.9–43.7)	33 (20.3–53)	<0.001[a]
SYNTAX score	20.5 (4–34)	11 (0–38.5)	<0.001[a]
CHA_2DS_2-VASc	5 (2–7)	1 (1–2)	<0.001[a]
NLR	7.16 (2.43–34.16)	2.84 (0.25–16.33)	<0.001[a]
SII	1641 (778–4506)	660 (54–2835)	<0.001[a]

Data are median (minimum – maximum). [a] Mann–Whitney U test. AF, atrial fibrillation; E, peak early mitral filling velocity; E', early diastolic myocardial velocity; LV-EF, left ventricular ejection fraction; NLR, neutrophil to lymphocyte ratio; SII, systemic immune-inflammation index.

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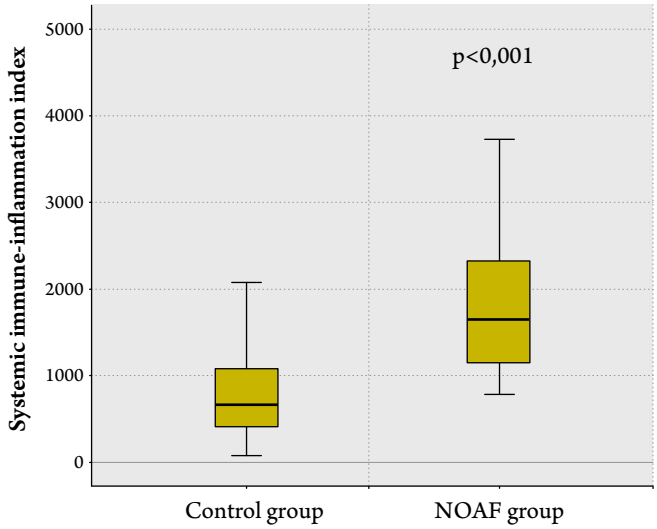
Figure 1. SII was increased in NOAF patients compared to control patients

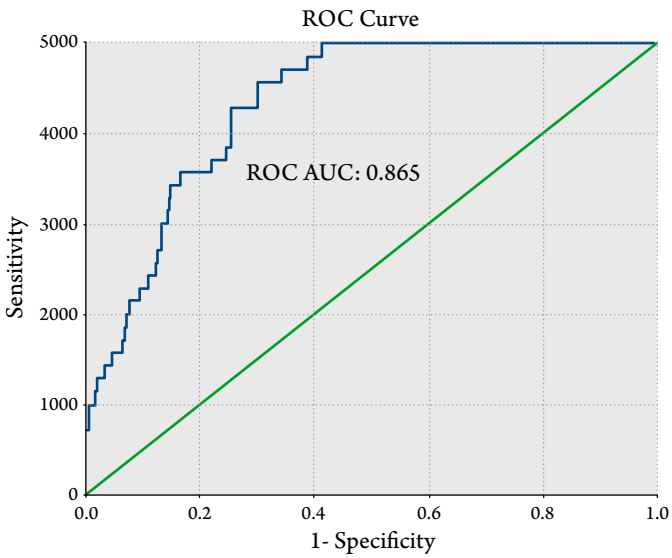


S2, stroke/TIA/thromboembolism history: 2 point; V, vascular disease history:1 point; A, age 65–75: 1 point; Sc, female gender: 1 point) is calculated by questioning during the anamnesis.

Statistical Analysis

SPSS 20.0 for Windows (Statistical Package for the Social Sciences version 20.0, SPSS Inc., Chicago, IL, US) software package was used for statistical analysis. Kolmogorov-Smirnov tests were used to analyze the parametric and non-parametric distributions of the data. Independent sample t-tests were used to compare groups of parametrically distributed variables, and Mann–Whitney U tests were used to compare groups of the non-parametrically distributed variables. Categorical variables were compared using the chi-squared test. Parametric, continuous variables are expressed as mean±standard deviation (SD), whereas non-parametric variables are expressed as median (minimum–maximum) values. Categorical variables are expressed as numbers and percentages. In order to evaluate the correlation between NOAF and SII, if the parameters are normally distributed, pearson correlation was used evaluated, if not, spearman correlation analysis was used. Multivariable logistic regression analysis was performed to identify the parameters that predict NOAF. Receiver operating characteristic (ROC) curve analysis was performed to determine the cut-off values of SII that could be used to predict NOAF. Probability (p) values of < 0.05 were deemed to indicate statistically significance.

Figure 2. Receiver operating characteristic curve of SII to predict NOAF. The area under the curve was0.865 (0.821-0.908), p<0.001)



State Variable: New onset atrial fibrillation. Test Variable: Systemic immune-inflammation index. The optimal cutoff level of SII was 1 093 with 75.3% specificity and 74.3% sensitivity.

Results

A total of 622 patients were included in this study. NOAF was detected in 35 (5.6%) of the patients (12 female and 23 male, mean age 69.1±12.3yrs). These patients were identified as the patient group.587 patients (140 female and 447 male, mean age 62.6±12.3yrs) who did not develop AF were identified as the control group. The demographic characteristics and blood data of both groups are shown in Table 1. The following variables differed significantly between the two groups: age, body mass index (BMI), type of acute coronary syndrome, hypertension (HT), hemoglobin (Hb), white blood cells (WBC), lymphocytes, neutrophils, creatine, albumin, and total cholesterol. Echocardiographic and index findings are shown in Table 2. SII mean and standard deviations of all patients were calculated as 902±657. The following variables differed significantly between the two groups: LV-EF, the ratio between early mitral inflow velocity and mitral annular early diastolic velocity (E/E'), LA size, SS, and a score computed from the following factors: presence of congestive heart failure, hypertension, age ≥75 yrs, diabetes mellitus, stroke or transient ischemic attack (TIA), vascular disease, age 65 to 74 yrs, and gender (CHA2DS2-VASc). SII values differed significantly as shown in Figure 1 [NOAF 1641 (778-4506) vs Control 660 (54-2835)] (p<0.001). The other variables did not differ significantly between the groups.

The correlation between NOAF and SII was determined using Spearman's rank correlation (correlation coefficient, 0.622; p<0.001). In Model A, the multivariable regression analysis revealed that age (Odds Ratio (OR) 1.085, 95%Confidence Interval (CI) 1.029–1.144, p=0.003), LA (OR 1.155, 95%CI 1.045–1.278, p=0.005), and SII (OR 1.002, 95%CI 1.001–1.002, p<0.001) were independent predictors for NOAF (Table 3). In model B, the multivariable

Table 3. Multivariable analysis showing the association between variables and NOAF

Parameters	Model A				Model B			
	OR	95%Cl		p	OR	95%Cl		p
		Lower	Upper			Lower	Upper	
Age	1.085	1.029	1.144	**0.003**	1.092	1.033	1.154	**0.002**
Gender	0.634	0.157	2.560	0.522	0.844	0.184	3.866	0.827
BMI	1.076	0.966	1.199	0.183	1.095	0.977	1.227	0.120
HT	1.171	0.275	4.992	0.831	0.724	0.157	3.342	0.679
Smoking	1.252	0.0.278	5.647	0.770	0.973	0.217	4.375	0.972
HB	1.271	0.911	1.774	0.158	1.168	0.816	1.672	0.395
Glucose	0.992	0.976	1.007	0.297	0.991	0.975	1.008	0.301
Creatinin	1.258	0.337	4.703	0.733	1.4618	0.401	6.531	0.499
Albumin	0.724	0.246	2.126	0.556	0.858	0.272	2.703	0.793
LDL–C	0.998	0.985	1.012	0.800	1.000	0.985	1.014	0.956
LV-EF	0.951	0.906	0.998	0.041	0.973	0.922	1.026	0.311
E/E’	1.042	0.950	1.143	0.387	1.022	0.925	1.129	0.666
LA	1.155	1.045	1.278	**0.005**	1.122	1.009	1.247	**0.034**
SS	–	–	–	–	1.116	1.031	1.208	**0.006**
SII	1.002	1.001	1.002	**<0.001**	1.002	1.001	1.002	**<0.001**

BMI, body mass index; E, peak early mitral filling velocity; E’, early diastolic myocardial velocity; HB, hemoglobin; HT, hypertension; LDL–C, low density lipoprotein cholesterol; LV-EF, left ventricular ejection fraction; SII, systemic immune-inflammation index; SS, SYNTAX score.

Table 4. Results of ROC curve analysis

Risk Factor	AUC (95%)	Cutoff	p	Sensitivity (%)	Specificity (%)
SII	0.865 (0.821–0.908)	1093	<0.001	74.3	75.3
SS	0.781 (0.699–0.864)	15.25	<0.001	71.4	72.6
CHA_2DS_2-VASc	0.777 (0.671–0.884)	3.5	<0.001	70.8	69.5
NLR	0.857 (0.810–0.904)	5.63	<0.001	80	79.6

Sensitivity and specificity by the optimized cutoff points for detecting NOAF.AUC,areaunder the ROC curve; NLR, neutrophil to lymphocyte ratio; SII,systemic immune-inflammation index; SS, SYNTAX score.

regression analysis, age (OR 1.092, 95%CI 1.033–1.154, p=0.002), LA (OR 1.122, 95%CI 1.009–1.247, p=0.034), SS (OR 1.116, 95%CI1.031–1.208), p=0.006), and SII (OR 1.002, 95%CI 1.001–1.002, p<0.001) were independent predictors for NOAF (Table 3).

ROC curve analysis revealed the SII cut-off value of 1093, with a sensitivity of 74.3% and a specificity of 75.3%, which can be used to predict NOAFarea under curve (AUC) of 0.865, 95%CI: 0.821–0.908, p<0.001, Figure 2]. ROC curve analysis of SS and CHA2DS2-VASc are presented in Table 4 (p<0.001).

Subgroup analysis of the non-ST segment elevation myocardial infarction (non-STEMI) group revealed significantly higher SII values in those with NOAF (p<0.001). The results of the ROC curve analysis taking only the non-STEMI group into consideration revealed an SII cut-off value of 1416 found with a sensitivity of 100% and a specificity of 94.2%, which can be used to predict NOAF (AUC0.977, p<0.001).

Discussion

In this study, the relationship between NOAF and SII in patients with ACS was investigated. The results revealed that the patients who developed AF had higher SII values. In parallel, high SII values were found to be an independent predictor of the development of AF in ACS patients.

Inflammation plays an important role in the pathophysiology of ACS [15]. Accordingly, the inflammatory parameters and indices are likely to increase during ACS, which explains why the ACS patients in this study had higher SII values as compared to healthy individuals. On the other hand, the results indicated that the difference between the inflammatory parameters of the ACS patients and healthy individuals were even more significant when another inflammatory event, such as atrial fibrillation, was present. As a matter of fact, in 2021Cırakoglu et al. [12] reported a relationship between the SII index and carotid intima-media thickness of patients who did not previously have cardiovascular disease and were studied with respect

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to the development of carotid artery disease. The mean SII value of the patients included in that study was 624 as compared to 902, the mean SII value of all patients included in this study. The difference between the two results is attributed to the impact of an existing coronary event on inflammatory values. Furthermore, in this study, the mean SII value of the NOAF group was 1641.

In the 2009article by Schmitt et al., the probability of developing atrial fibrillation after ACS was reported as 6–22%, yet in the same article it was also predicted that this rate would decrease with the transition from the thrombolytic period to the the time of percutaneous coronary intervention [7]. On the other hand, in the 2005 CArvedilol Post-infaRct survival COntRolledevaluatioN (CAPRICORN) study of McMurray et al., the development of AF was reported to have decreased to 2.3%after ACS [16]. In comparison, in the current study, 35 (5.6%) of the 622 ACS patients developed AF. This ratio is consistent with the respective results reported in the literature.

In 2021, Bagcı et al. concluded that the SII index predicted NOAF in ST elevation myocardial infarction (STEMI) patients [17]. In comparison, in a study of Gholoobi et al, non-STEMI patients that were closely affected by the inflammatory process were also taken into account in addition to the STEMI patients [18]. Additionally, in the study of Bagcı et al., the cut-off value of the SII index that can be used to predict NOAF was 1228, which is similar to the respective result found in this study.

Furthermore, in the current study, a higher ratio of STEMI patients developed AF. Nevertheless, results of the subgroup analysis, taking only the non-STEMI group into account, revealed a significant relationship between SII and NOAF ($p<0.001$). The results of the ROC curve analysistaking only the non-STEMI group into consideration revealed an SII cut-off value of 1416, with a sensitivity of 100% and a specificity of 94.2%, which can be used to predict NOAF (AUC0.977, $p< 0.001$). Therefore, we concluded that SII is a more successful index for predicting AF in the non-STEMI group, regardless of the fact that AF is more common in STEMI patients.

SS has been shown to be associated with AF in a previous study [19]. Similarly, in the current study, SS was found to be an independent predictor of NOAF. Since the relationship between SS and NOAF was strongly demonstrated in the multicenter study of Eren et al. [19], we performed the regression analysis without SS (model A) and with SS (model B) separately in our study; and we demonstrated the importance of SII in predicting NOAF. However, SS has limited use in daily practice as a predictor of NOAF due to the complexity of its calculation. On the other hand, the CHA2DS2-VASc score has been shown to be closely associated with complications of AF [20]. Additionally, the CHA2DS2-VASc score has been shown to be an independent predictor of NOAF [21]. İn our study, the cut-off value of the CHA2DS2-VASc score for predicting NOAF was 3.5 (AUC 0.777, $p<0.001$) with 70.8% sensitivity and 69.5% specificity.

The study of Raczkowska-Golanko et al. [22] found that the neutrophil/lymphocyte ratio (NLR), a parameter of the SII index, was significantly different between groups ofACS patients with and without NOAF. Many clinical studies have been conducted to determine the predictors of NOAF utilizing many different laboratory parameters and clinical findings [23, 24], some of which were addressed in the 2017 meta-analysis of Weymann et al. of laboratory parameters [25] and in the 2017 meta-analysisof Zeng et al. ofechocardiographic parameters [26]. The results of the current study are consistent with the results of thesemeta-analyses.

The AF co-morbidity in ACS patients has been reported to increase in-hospital and in 1-yr mortality rates [27]. In this context, use of SII values to predictin advance AF may allow administering of a more rigorous medical treatment to prevent an increase in mortality rates. Additionally, treatment of AF risk factors and close follow-up of cardiac rhythm would also be pertinent in patients with high SII values.

Limitations of the Study

Apart from the strengths mentioned throughout the text, there were also some limitations to this study. First, according to the procedures of the hospital where this study was performed, patients with unstable angina were treated in the intermediate intensive care unit, so they were not followed up in the coronary intensive care unit. Given that this study was restricted to patients in the coronary intensive care unit, unstable angina patients were not included. Secondly, even though the results of this study demonstrate a relationship between SII and NOAF, as a retrospective cross-sectional study, it reveals the underlying pathophysiological mechanisms only to a limited extent.

Conclusion

The findings of this study indicate that SII could be helpful in predicting the development of NOAF in ACS patients. Predicting the development of AF in advance would provide an opportunity to eliminate associated risk factors and, if necessary, to administer early treatment. Additionally, since the development of AF in ACS patients has been commonly associated with a poor prognosis, close follow-up of patients with high SII values may be recommended.

No conflict of interest is reported.

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