Original Article

Systemic Immune-Inflammation Index as a Predictor of Left Atrial Thrombosis in Nonvalvular Atrial Fibrillation

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Abstract

Background: *The systemic immune-inflammation index (SII) has recently been investigated for cardiovascular diseases. We aimed to evaluate the relationship between SII and left atrial thrombosis (LAT).*

Methods: *This retrospective, case-control study recruited patients with nonvalvular atrial fibrillation (NVAF) who underwent transesophageal echocardiography (TEE) for LAT detection before cardioversion or catheter ablation at a tertiary hospital between 2012 and 2021. Demographic characteristics were obtained from the hospital data system. According to TEE findings, the patients were categorized into LAT (+) and (-) groups. Age, gender, history of chronic diseases, urea, creatinine, albumin, hemogram parameters, the neutrophil-lymphocyte ratio (NLR), the platelet-lymphocyte ratio (PLR), SII, the $CHADS_2$ score, the CHA_2DS_2-VASc score, echocardiographic parameters, antiaggregant-anticoagulant use, and non-paroxysmal atrial fibrillation were included and analyzed.*

Results: *The study population consisted of 403 patients, including 228 men (56.6%), at a mean age of 60.84±12.26 years. A high white blood cell count (WBC) (OR, 1.26; 95% CI, 1.05 to 1.51; P=0.013), a high SII (OR, 1.00, 95% CI, 1.00 to 1.00; P=0.003), and a low ejection fraction (OR, 0.95; 95% CI, 0.90 to 0.99; P=0.018) were independent predictors of LAT (+). A spontaneous echo contrast (OR, 2.43; 95% CI, 1.35 to 4.39; P=0.003) was associated with LAT (+). SII values above 693.6 predicted LAT (+) with 71.6% sensitivity and 71.7% specificity (AUC, 0.77; P<0.001). The predictiveness of SII was similar to that of NLR (0.77 vs 0.74, P=0.093) but higher than PLR (0.77 vs 0.67; P<0.001) and WBC (0.77 vs 0.69; P=0.031).*

Conclusion: *SII is an independent predictor of LAT in patients with NVAF.*

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Keywords: *Thrombus; Atrial fibrillation; Inflammation; Biomarkers; Echocardiography*

Introduction

Atrial fibrillation (AF) is a common arrhythmic disorder in clinical practice, with its incidence and prevalence rising with age.[1] Ischemic strokes and other AF-induced thromboembolic events are related to increased morbidity and mortality, and these events are closely linked with left atrial thrombosis (LA).[2,3]

Transesophageal echocardiography (TEE) is the essential diagnostic tool to detect LAT with 97% sensitivity and

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100% specificity.[4,5] TEE is routinely recommended for LAT exclusion before AF ablation and cardioversion.[6]

Hypercoagulability, inflammation, and stasis due to weak LA contraction are essential to LAT pathogenesis in patients with AF.[7] Inflammatory cells and mediators involved in inflammation may directly result in thrombogenic activity and endothelial damage, triggering thrombogenesis.[8] Abnormal changes in systemic inflammation are associated with prothrombotic indicators in AF, which suggests that inflammation might induce prothrombotic status in AF.[9]

The systemic immune-inflammation index (SII) is an indicator based on the number of neutrophils, platelets, and lymphocytes and may comprehensively reflect the state of inflammation within the body. SII is mostly used in patients with cancer to predict adverse clinical outcomes and has recently been introduced in cardiovascular studies.[10-13] Nonetheless, no data are available on the relationship between SII and LAT. The present investigation aimed to evaluate the relationship between LAT and SII in patients with nonvalvular atrial fibrillation (NVAF).

Methods

Between January 2012 and January 2021, the present retrospective case-control study evaluated 403 patients with NVAF who underwent TEE for LAT detection before cardioversion or catheter ablation. NVAF was defined following the current guidelines.[6] The patients' demographics, medication history, laboratory, and echocardiographic data were evaluated retrospectively from the records. Patients with valvular AF, acute coronary syndrome, acute or chronic pulmonary embolism, venous thrombosis, severe heart failure (the New York Heart Association functional class IV), severe valvular diseases, a history of any systemic inflammatory diseases, severe thrombocytopenia (<50.000/μL), moderate and severe anemia (<10 g/dL), known malignancies, connective tissue diseases, kidney failure, liver failure, evidence of acute or chronic infection, or a history of blood transfusions in the preceding 3 months were excluded. The study complied with the principles of the Declaration of Helsinki and was approved by the local ethics committee (approval date: December 23, 2021; approval number: 0559).

Routine blood samples were taken before cardioversion or catheter ablation. In addition, levels of blood biochemical parameters, including urea, creatinine, and albumin, were measured using the Beckman Coulter AU 5800 AutoAnalyzer. The Modification of Diet in Renal Disease (MDRD) formula was employed to calculate the glomerular filtration rate.

Blood samples were collected in Monovette tubes (SARSTEDT Monovette, Nümbrecht, Germany) anticoagulated with EDTA for complete blood count analysis, comprising the levels of hemoglobin and the hematocrit; the counts of platelets, white blood cells (WBC), monocytes, lymphocytes, and neutrophils; the mean volumes of platelets and corpuscles; and the red blood cell distribution width (RDW). An automated blood cell counter (Beckman Coulter LH 750; Beckman Coulter, Inc, USA) was utilized to analyze the variables of the complete blood count. SII was calculated using neutrophil, lymphocyte, and platelet values.[14]

All the patients were evaluated with a Philips iE33 transthoracic echocardiography device (Philips Healthcare, Inc, Andover, MA, USA). Routine views were obtained from the parasternal and apical echocardiographic windows. The LA diameter was measured from parasternal long-axis images at the end of the ventricular systole. The left ventricular ejection fraction (LVEF) was evaluated using the modified Simpson method.

All the patients underwent TEE with a Philips iE33 multiplane TEE probe (Philips Healthcare, Inc, Andover, MA, USA). Written informed consent forms were obtained from the patients before each procedure. All TEE procedures were performed by experienced echocardiographers. Before TEE, the oropharynx was anesthetized with a local anesthetic spray; then, the patients were sedated. The procedure was performed in the left lateral decubitus position, and the presence of LAT was investigated in different TEE planes. Dense echo masses with a tissue other than the LA endocardium were defined as a thrombus. The patients were classified as LAT (+) and LAT (-) according to the presence of thrombi in TEE.

The $CHADS_2$ and CHA_2DS_2-VASc scores were calculated to evaluate ischemic stroke risks in patients with NVAF.[15,16] According to the $CHADS_2$ scoring, congestive heart failure, hypertension, a minimum age of 75 years, and diabetes mellitus are scored 1 point each; and a history of transient ischemic attacks, strokes, and thromboembolic events is scored 2 points. According to the CHA_2DS_2-VASc scoring, each of congestive heart failure, hypertension, age between 65 and 74 years, diabetes mellitus, vascular disease, and female gender is scored 1 point; and a minimum age of 75 years and a history of transient ischemic attacks, strokes, and thromboembolic events are each scored 2 points. Congestive heart failure was defined as clinical signs and symptoms of heart failure or objective evidence of LV dysfunction.[17] Patients were defined as having hypertension if they had an office blood pressure exceeding 140/90 mm Hg measured at rest or current antihypertensive regimens.[18] A plasma fasting blood glucose level exceeding 126 mg/dL or a blood glucose level of greater than 200 mg/dL at any time measurement or the use of any antidiabetic treatment was considered diabetes mellitus.[19] Vascular diseases comprised peripheral artery diseases, previous myocardial infarctions, and aortic plaques. Paroxysmal AF was defined as the type of AF self-terminating in less than

7 days from AF.[6] Adequate anticoagulation was defined as the use of direct oral anticoagulants or warfarin therapy with a weekly monitoring of blood clotting time during the 3 weeks before TEE.[6]

The IBM SPSS software statistical package, version 25.0, (SPSS Inc, Chicago, IL, USA) was used to perform the statistical analyses. The normality of the analyzed data was evaluated using the Kolmogorov–Smirnov test. Continuous variables with a normal distribution were introduced as mean±standard deviation, and continuous variables without a normal distribution were introduced as medians and interquartile ranges (1–3 quartiles). Categorical variables were presented as percentages. The continuous variables were analyzed using the Student *t* or Mann–Whitney *U* test, while the χ^2 test was employed to analyze the categorical variables. Univariate and multivariate logistic regression analyses were utilized to determine the independent predictors of LAT (+). A univariate logistic regression analysis was performed to determine the relationship between predictors and LAT (+). Variables with a P value of less than 0.05 in the univariate analysis were entered into a multivariate logistic regression analysis to detect independent predictors of LAT (+). Parameters in SII were not included in the multivariate analysis to avoid multicollinearity. Variables with a P value of less than 0.05 in the multivariate logistic regression analysis were considered independent predictors of LAT (+). The receiver operating characteristic (ROC) analysis was applied to assess the optimum cutoff values of the predictors of LAT (+). The area under the curve (AUC) of the predictors was compared with the results of the DeLong test. A *P* value of less than 0.05 was considered statistically significant.

Results

Based on LAT, the study population was categorized into 2 groups: 81 LAT (+) patients (male: 45 [55.6%]; age= 61 [53–67.5] y) and 322 LAT (-) patients (male: 183 [56.8%]; age= 62 [53–70] y).

Clinical, demographic, laboratory, and echocardiographic data were compared between the LAT (+) and LAT (-) groups (Table 1). The LAT (+) group had higher rates of congestive heart failure (24 [29.6] vs 47 [14.6] %; *P*=0.002), stroke history (15 [18.5] vs 21 [6.5] %; *P*=0.001), spontaneous echo contrast (SEC) (49 [60.5] vs 87 [27.0] %; *P*<0.001), and non-paroxysmal AF (47 [58.0] vs 131 [40.7] %; *P*=0.005). On the other hand, the median age, gender distribution, and the frequencies of diabetes mellitus, hypertension, chronic obstructive pulmonary disease, and a history of coronary artery disease were similar. The LAT (+) group had a lower LVEF (54 [40–60] vs 60 [50–60] %; *P*<0.001) and a larger LA diameter (44 [39–49] vs 41.5 [38–45] mm; *P*<0.001). The $CHADS_2$ and CHA_2DS_2-VASc scores significantly differed between the groups (1 [1–2] vs 1 [0–2]; *P*<0.001 and 3 [1–4)] vs 2 [1–3]; *P*=0.001).

No statistically significant differences existed between the LAT (+) and LAT (-) groups regarding routine serum biomarkers, such as the levels of urea, creatinine, hemoglobin, and hematocrits; the glomerular filtration rate; and the mean volumes of corpuscles and platelets. On the other hand, serum albumin, WBC (9.39 [7.44–11.3] vs 7.45 [6.22–9.23] $\times 10^3$ /μL; *P*<0.001), neutrophils (6.47 [4.89–8.04] vs 4.48 [3.59–5.74] $\times 10^3$ /μL; *P*<0.001), lymphocytes (1.81 [1.50–2.26] vs 2.10 [1.64–2.60] $\times 10^3$ /μL; *P*=0.007), platelets (259 [218.5–338] vs 239 [197.5–282.2] $\times 10^3$ /μL; *P*=0.006), monocytes (0.67 [0.47–0.78] vs 0.56 [0.43–0.70] $\times 10^3$/μL; P=0.014), and RDW (14.2 [13.3–15.4] vs 13.8 [13.1–14.9] %; *P*=0.036) differed significantly between the groups (Table 1).

Inflammatory parameters, including the neutrophil-lymphocyte ratio (NLR), the platelet-lymphocyte ratio (PLR), and SII, were compared between the LAT (+) and LAT (-) groups. NLR (2.93 [2.35-4.63] vs 2.22 [1.57–2.86]; *P*<0.001), PLR (151.3 [108.1–186.7] vs 113.7 [89.9–148.1]; *P*<0.001), and SII (883.3 [655.2–1291.8] vs 516.7 [360.1–725.4]; *P*<0.001) were significantly higher in the LAT (+) group than in the LAT (-) group (Table 1).

Parameters that were significant in the univariate logistic regression analysis were included in multivariate logistic regression analysis to determine predictors of LAT (+). A low LVEF (OR, 0.95; 95% CI, 0.90 to 0.99; *P*=0.018), SEC (+) (OR, 2.43; 95% CI, 1.35 to 4.39; P=0.003), WBC (OR, 1.26; 95% CI, 1.05 to 1.51; *P*=0.013), and SII (OR, 1.00; 95% CI, 1.00 to 1.00; *P*=0.003) were independent predictors of LAT (+) (Table 2). In the ROC curve analysis (Figure 1), WBC over a cutoff of 8.51×10^3 /μL predicted LAT(+) with 65.4% sensitivity and 65.5% specificity (AUC, 0.69; 95% CI, 0.63 to 0.76; *P*<0.001), NLR over a cutoff of 2.63 predicted LAT(+) with 66.7% sensitivity and 66.8% specificity (AUC, 0.74; 95% CI, 0.68 to 0.80; *P*<0.001), PLR over a cutoff of 131.5 predicted LAT(+) with 64.2% sensitivity and 64.6% specificity (AUC, 0.67; 95% CI, 0.61 to 0.74; *P*<0.001), and SII over a cutoff of 693.6 predicted LAT(+) with 71.6% sensitivity and 71.7% specificity (AUC, 0.77; 95% CI, 0.71 to 0.83; *P*<0.001). Thereafter, the areas under the WBC, NLR, PLR, and SII curves were compared with the DeLong method. The results revealed that the AUC of SII was not statistically larger than that of NLR (0.77 vs 0.74; *P*=0.093) but was statistically larger than those of PLR (0.77 vs 0.67; *P*<0.001) and WBC (0.77 vs 0.69; *P*=0.031).

Table 1. Comparison of baseline characteristics and laboratory findings between the groups

Variable	LAT(+) (n=81)	LAT(-) (n=322)	P
Baseline Characteristic			
Age (y)	61 (53-67.5)	62 (53-70)	0.626
Gender (male), n (%)	45 (55.6)	183 (56.8)	0.836
Diabetes mellitus, n (%)	20 (24.7)	53 (16.5)	0.086
Hypertension, n (%)	41 (50.6)	152 (47.2)	0.583
Congestive heart failure, n (%)	24 (29.6)	47 (14.6)	0.002
Stroke, n (%)	15 (18.5)	21 (6.5)	0.001
Chronic obstructive pulmonary disease, n (%)	7 (8.6)	24 (7.5)	0.720
History of CAD. n(%)	23 (28.4)	63 (19.6)	0.083
Urea (mg/dL)	17 (13-21.5)	16 (13-20)	0.232
Creatinine (mg/dL)	0.87 (0.78-1.07)	0.85 (0.77-1)	0.213
GFR (mL/dk/1.73 m^2)	80.05 (68.4-98.2)	85.87 (73-98)	0.330
Albumin (mg/dL)	39.8 (39-39.9)	39.8 (39.8-41)	0.047
WBC ($\times10^3$ /μL)	9.39 (7.44-11,3)	7.45 (6.22-9.23)	<0.001
Neutrophils ($\times10^3$ /μL)	6.47 (4.89-8.04)	4.48 (3.59-5.74)	<0.001
Lymphocytes ($\times10^3$ /μL)	1.81 (1.50-2.26)	2.10 (1.64-2.60)	0.007
Monocytes ($\times10^3$ /μL)	0.67 (0.47-0.78)	0.56 (0.43-0.70)	0.014
Hemoglobin (g/dL)	14.1 (13-14.9)	13.9 (12.7-14.8)	0.250
HTC (%)	42.60±4.55	41.60±4.48	0.071
MCV (fl)	86.8 (82.5-89.1)	86.5 (83.5-90.5)	0.601
RDW (%)	14.2 (13.3-15.4)	13.8 (13.1-14.9)	0.036
Platelets ($\times10^3$ /μL)	259 (218.5-338)	239 (197.5-282.2)	0.006
MPV (fl)	10.50±1.13	10.40±1.13	0.534
NLR	2.93 (2.35-4.63)	2.22 (1.57-2.86)	<0.001
PLR	151.3 (108.1-186.7)	113.7 (89.9-148.1)	<0.001
SII	883.3 (655.2-1291.8)	516.7 (360.1-725.4)	<0.001
$CHADS_2$ score	1 (1-2)	1 (0-2)	<0.001
CHA_2DS_2-VASc score	3 (1-4)	2 (1-3)	0.001
LVEF (%)	54 (40-60)	60 (50-60)	<0.001
Left atrial diameter (mm)	44 (39-49)	41.5 (38-45)	<0.001
IVS (mm)	11 (10-12)	11 (10-12)	0.214
SEC, n (%)	49 (60.5)	87 (27.0)	<0.001
ASA, n (%)	13 (16.0)	65 (20.2)	0.400
Clopidogrel, n (%)	3 (3.7)	20 (6.2)	0.591
Effective use of oral anticoagulant, n (%)	18 (22.2)	81 (25.2)	0.584
Non-paroxysmal atrial fibrillation, n (%)	47 (58.0)	131 (40.7)	0.005

LAT, Left atrial thrombus; CAD, Coronary artery disease; GFR, Glomerular filtration rate; WBC, White blood cell count; HTC, Hematocrit; MCV, Mean corpuscular volume; RDW, Red cell distribution width; NLR, Neutrophil-lymphocyte ratio; PLR, Platelet-lymphocyte ratio; SII: Systemic immune-inflammation index; LVEF, Left ventricular ejection fraction; IVS, Interventricular septum; SEC, Spontaneous echo contrast; ASA, Acetylsalicylic acid

Table 2. Logistic regression analysis of potential predictor factors for left atrial thrombi

Variable	Univariate Analysis			Multivariate Analysis		
	OR	95% CI	P	OR	95% CI	P
$CHADS_2$ score	1.43	1.17-1.75	0.001	0.79	0.43-1.46	0.449
CHA_2DS_2-VASc score	1.25	1.08-1.45	0.003	1.07	0.74-1.56	0.711
WBC	1.39	1.24-1.57	<0.001	1.26	1.05-1.51	0.013
Neutrophils	1.64	1.42-1.89	<0.001			
Lymphocytes	0.65	0.46-0.92	0.015			
Monocytes	3.87	1.36-11.00	0.011	0.33	0.07-1.64	0.176
RDW	1.10	0.97-1.24	0.139			
Platelets	1.01	1.00-1.01	0.002			
Albumin	0.93	0.87-0.99	0.025	0.94	0.87-1.02	0.138
LVEF	0.95	0.93-097	<0.001	0.95	0.90-0.99	0.018
Left atrial diameter	1.09	1.05-1.13	<0.001	1.03	0.98-1.08	0.262
NLR	1.59	1.36-1,85	<0.001			
PLR	1.01	1.00-1.01	<0.001			
SII	1.00	1.00-1.00	<0.001	1.00	1.00-1.00	0.003
Congestive heart failure	2.46	1.40-4.35	0.002	0.50	0.14-1.77	0.280
Stroke	3.26	1.60-6.65	0.001	2.81	0.85-9.30	0.091
Non-paroxysmal atrial fibrillation	2.02	1.23-3.30	0.005	1.47	0.78-2.77	0.237
SEC	4.14	2.49-6.88	<0.001	2.43	1.35-4.39	0.003

WBC, White blood cell count; RDW, Red cell distribution width; LVEF, Left ventricular ejection fraction; NLR, Neutrophil-lymphocyte ratio; PLR, Platelet-lymphocyte ratio; SII: Systemic immune-inflammation index; SEC, Spontaneous echo contrast

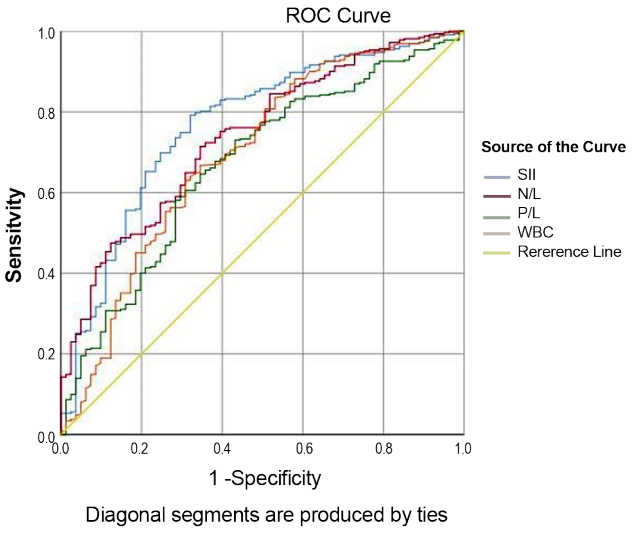

Figure 1. The image depicts the receiver operating characteristic (ROC) curves for the systemic immune-inflammation index (SII), the neutrophil-to-lymphocyte ratio (NLR), the platelet-to-lymphocyte ratio (PLR), and the white blood cell count (WBC) for predicting left atrial thrombosis.

Discussion

This research suggests that SII is an independent and essential predictor of LAT in patients with NVAF. To the best of our knowledge, this is the first study to investigate the relationship between SII and LAT in patients with NVAF. We found an association between LAT and NLR, PLR, WBC, SEC, and a low EF.

Patients with AF are at an increased risk of cardioembolic events due to LAT formation. The relationship between some parameters with LAT has been demonstrated. SEC is a known precursor of thrombus formation.[20,21] A low EF is also a proven risk factor for LAT(+).[22,23] Our results support previous data on these relationships. In addition, there are studies investigating the relationship between LAT formation and many markers. Zhang et al[24] evaluated 2.246 patients and identified an independent relationship between abnormal uric acid metabolism and LAT. Another study evaluated the association between LAT and RDW and N-terminal (NT)-prohormone BNP (NT-proBNP) in the blood and reported that higher levels of RDW (cutoff ≥12.95%) and NT-proBNP (cutoff ≥368.9 ng/L) were independent predictors of LAT.[25] A study on patients with NVAF who underwent radiofrequency ablation revealed that the $CHADS_2$ and CHA_2DS_2-VASc scores were associated with LAT.[26] Contrary to the above results, we found no significant correlations between LAT and the $CHADS_2$ and CHA_2DS_2-VASc scores in the present study.

It has been suggested that the Virchow triad, consisting of stasis, endothelial damage, and hypercoagulability, accounts for thrombus formation; nevertheless, the pathogenesis of LAT formation has yet to be elucidated.[27] Inflammation has an essential role in thrombus formation, especially by inducing endothelial damage and increasing thrombogenesis. Therefore, the role of inflammatory markers in LAT formation is the subject of ongoing research. Consistent with the present study results, another study on 309 patients suggested an independent relationship between LAT and NLR, an inflammatory marker.[28] Maehama et al[29] investigated the relationship between systemic inflammation and LAT and revealed that a higher C-reactive protein (CRP) level, a significant systemic inflammation marker, could predict LAT. They also suggested that CRP had a high estimator capacity in excluding the likelihood of LAT.

Neutrophils, lymphocytes, and platelets are closely associated with the inflammatory process in thrombus formation. Neutrophil extracellular traps (NETs), released by neutrophils, can act as a skeleton for the adhesion of platelets and specific platelet-adhesion molecules, such as von Willebrand factor, fibronectin, and fibrinogen. This formation also triggers platelet activation and coagulation cascades.30 Relevant studies in the literature have shown that NETs constitute a part of arterial and venous thrombi. Laridan et al31 found that patients with a cardioembolic stroke history had a higher NET formation level than those with a history of non-cardioembolic stroke. They also reported higher levels of neutrophils in old thrombus material (>1 d) than in new thrombus material (<1 d). These observations are suggestive of the fact that NETs are associated with thrombus formation due to stasis, as is the case in AF. Platelets are acute-phase reactants and are elevated in inflammatory conditions. They secrete mediators, which induce proinflammatory and procoagulant effects. Although inconsistent results have been reported regarding platelet activation, some studies have found that platelet activation increases in AF patients with LAT.[32] Unlike these 2 groups of cells, the number of lymphocytes is expected to decrease due to lymphocyte apoptosis in cases associated with increased inflammation.[33] In this context, the results of our study are also compatible with this pathogenesis of the inflammatory process insofar as neutrophil and platelet levels were significantly higher in the LAT(+) group than in the LAT(-) group, while lymphocyte levels were low.

SII is an indicator reflecting the balance between inflammatory and immune status and is based on neutrophil, platelet, and lymphocyte counts. The predictive ability of SII for clinical terminations has been evaluated in various oncological patient groups. SII has also been the focus of some recent cardiovascular studies. Furthermore, some studies have demonstrated the association between SII and thrombus formation. A prior investigation reported that SII was associated with poor termination in patients with acute or subacute cerebral vein thrombosis.[34] Gök et al[35] investigated the relationship between SII and pulmonary thromboembolism severity and concluded that SII could

predict massive pulmonary thromboembolism with high sensitivity and specificity. Another study on 52 patients aged over 60 years with hip fractures compared patient groups with and without venous thromboembolism and concluded that SII was an independent predictor for venous thromboembolism in that patient group.[36] Considering these data, we think that SII can play a significant role in predicting LAT formation. Indeed, our results revealed that SII predicted LAT (+). Many studies have compared the predictive value of SII with NLR and PLR and shown that SII is superior to these other 2 markers.[37-39] In this study, all 3 markers predicted LAT (+), while SII was statistically similar to NLR and superior to PLR in predicting LAT (+).

Our study has several limitations. Firstly, the study was designed with a relatively small sample of patients with the retrospective method. Secondly, the study was single-center and may not reflect the general patient population. Thirdly, the relationships between LAT and other inflammation-related markers, such as CRP, procalcitonin, and interleukin-6, were not evaluated. Fourthly, only the anteroposterior LA diameter measurement was related to the LA in the echocardiographic assessment. Fifthly, the LA appendage emptying velocity, the left atrial volume index value, and the LA appendage morphology, which are associated with the risk of LAT formation, were not assessed. Whether SII predicts LAT can be evaluated in well-designed randomized controlled clinical trials.

Conclusion

SII is an independent and significant blood biomarker for LAT prediction in patients with NVAF. SII can be calculated using data obtained by simple blood testing; it is, thus, a useful predictor to determine LAT in patients with NVAF.

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