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Role of Systemic Immune-Inflammation Index in predicting multi-territorial atherosclerotic disease in peripheral arterial disease: a retrospective cohort study

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ABSTRACT

Aims: Death in patients with peripheral artery disease (PAD) is most often due to coronary artery disease (CAD) or cerebrovascular accidents (CVA). Systemic Immune-Inflammation Index (SII) can be used as a prognostic indicator in different cardiovascular diseases (CVD), including PAD. Our aim in this study was to investigate the predictive value of SII in detecting the coexistence of other CVD in patients with PAD.

Methods: The study included the results of 100 patients who were admitted to the cardiovascular surgery outpatient clinic of our hospital and diagnosed with PAD. PAD, CAD and carotid artery disease (CAAD) were diagnosed by history, physical examination, electrocardiography, duplex ultrasonography and ankle-brachial index (ABI) calculation. SII was determined as neutrophil count multiplied by platelet count and divided by lymphocyte count.

Results: 38 patients had CAAD, 28 had CAD, 19 had only PAD and 15 had all 3 diseases. Platelets levels (285±32.6 vs 234±9.6, p<0.001), Neutrophils levels (4.9±1.1 vs 4.1±9.8, p<0.001) and SII levels (4.9 ±1.1 vs 4.1±1.8, p<0.001) were compared in both groups with and without other arterial diseases in terms of laboratory findings, while other laboratory findings were not statistically different between both groups (p<0.05).

Conclusion: In our study, we found that those with CAD or CAAD had higher SII levels than those with only PAD.

Keywords: Peripheral artery disease, Systemic Immune-Inflammation Index, multi-territorial atherosclerotic disease

INTRODUCTION

Atherosclerosis is a systemic and inflammatory disease that can involve the entire arterial system. The most common types of atherosclerosis are coronary artery disease (CAD), carotid artery disease (CAAD) and peripheral artery disease (PAD). Studies have shown a link between the severity of atherosclerosis in one arterial territory and the occurrence of atherosclerotic disease in other territories.[1-4] In patients with CAAD and carotid artery stenosis ≥70%, 12.5% have PAD and 3.1% have CAD.[5] 24.5% of patients with PAD and 11.1% of patients with CAD have ≥70% carotid artery stenosis.[6] Therefore, in patients with PAD, the cause of death is mostly due to CAD or cerebrovascular events (CVAs).

The Systemic Immune-Inflammation Index (SII), a parameter of inflammation, is a prognostic indicator of adverse outcomes in various types of cancer.[7-9] Furthermore, recent studies have shown that SII, calculated by multiplying neutrophil and platelet counts multiplied by lymphocyte count divided by lymphocyte count, can be used as a prognostic indicator in different cardiovascular diseases (CVD), including PAD.[10-13]

Our aim in this study was to investigate whether SII can be used to detect the coexistence of other CVDs in patients with PAD.

METHODS

This study was prepared using data obtained from the Medical Master's Thesis titled "Risk of cardiovascular and cerebrovascular diseases in patients with PAD" which we completed in 2007 (Medical Specialization Thesis, Erciyes University, Faculty of Medicine, Department of Surgical Medical Sciences, Department of Cardiovascular Surgery, Kayseri, Turkiye, 2007 / Thesis No:193683). Our hospital's

Institute of Research Ethics reviewed this study involving human subjects The study was conducted in accordance with the guidelines set out in the Declaration of Helsinki. The ethics committee was informed about the non-experimental design of the retrospective study and approved the study.

The study included the results of 100 patients who were admitted to the cardiovascular surgery outpatient clinic of our hospital and diagnosed with PAD between 02/2005-09/2006. History and physical examination were recorded. PAD, CAD and CAAD were diagnosed by history, physical examination, electrocardiography, duplex ultrasonography and ankle-brachial index (ABI) calculation. The diagnosis of ABI was made by measuring the systolic pressures of the posterior tibial artery and dorsalis pedis and dividing the higher value by the systolic pressure of the brachial artery. Similar measurements were made in both extremities and the lower ABI value was accepted as the patient's ABI value.[14] PAD classification was performed with Rutherford[15] grading. CAAD was investigated by duplex ultrasonography. The intima-media thickness of the right and left internal carotid artery and the bifurcation area was measured and the presence of a lesion was recorded.

Exclusion criteria for the study were as follows: Patients with a history of acute coronary syndrome, anemia, active infection or systemic inflammatory disease, autoimmune or chronic inflammatory disease, heart failure (ejection fraction <40%), chronic anti-inflammatory drug use, impaired liver and kidney function, ABI greater than 1.3.

Blood samples were collected between 08:00-10:00 in the morning. Antecubital venous blood samples were collected in tubes containing tripotassium EDTA as anticoagulant. All routine biochemical tests and hematologic parameters were evaluated using an autoanalyzer. SII was determined as neutrophil count multiplied by platelet count and divided by lymphocyte count.

Statistical Analysis

The data analyses were performed using SPSS 21.0 for Windows (SPSS Inc., Chicago, IL, USA). The distribution of quantitative variables was checked with the Shapiro-Wilk test. Descriptive data were presented as mean±standard deviation and median (interquartile range, IQR) depending on the normality of the distribution. Median and quartile ranges were given for non-normally distributed variables. An independent samples t-test was used for the comparison of quantitative variables with normal distribution, and the Mann-Whitney U test was used for quantitative variables without normal distribution. Categorical variables were compared using the Chi-square test. p values below 0.05 were accepted to show statistically significant.

RESULTS

A total of 100 patients were included in the study. The mean age was 59.8±13.1 years. 93 of them were male. 58 patients were smokers. 38 patients had CAAD, 28 had CAD, 19 had only PAD and 15 had all 3 diseases. Diabetes mellitus (DM) was present in 32 patients and hypertension (HT) was present in 46 patients. Patients were divided into 2 groups: those with PAD only and those with concomitant disease in other arterial systems. When demographic characteristics were compared, no difference was observed between the groups in terms of age, gender, history of DM/HT/dyslipidemia, smoking history, body-mass index, heart rate, blood pressure measurements and Rutherford classification (p<0.05) (**Table 1**).

Table 1. Demographic characteristics of the study populations

	Another artery disease		
	Yes	No	p value
Variables	(n=81)	(n=19)	
Age (years)	57.8.2±8.6	61.2±10.1	0.502
Women gender (n, %)	5 (6.2%)	2 (10.5%)	0.455
Diabetes mellitus (n, %)	26 (32.1%)	6 (31.6%)	0.655
Hypertension (n, %)	36 (44.4%)	10 (52.6%)	0.672
Dyslipidemia	19 (23.5 %)	4 (21%)	0.391
Carotid artery disease (n, %)	38 (46.9%)	0 (46.3%)	-
Coronary artery disease (n, %)	28 (34.6%)	0 (6.9%)	-
Smoking (n, %)	47 (58%)	11 (57.9%)	0.145
Body-mass index (kg/m^2)	28.3±4.1	29.5±4.8	0.931
Systolic blood pressure (mmHg)	133.6±13.7	132.8±15	0.654
Diastolic blood pressure (mmHg)	81.7±8.7	84±9.9	0.331
Heart rate	88.1±14.7	92.3± 8.5	0.07
Rutherford classifications (≥3)	22 (27.2%)	5 (26.3%)	0.717

Platelets levels (285±32.6 vs 234±9.6, p<0.001), Neutrophils levels (4.9±1.1 vs 4.1±9.8, p<0.001) and SII levels (4.9±1.1 vs 4.1±1.8, p<0.001) were compared in both groups with and without other arterial diseases in terms of laboratory findings, while other laboratory findings were not statistically different between both groups (p<0.05) (**Table 2**).

Table 2. Laboratory findings of the study populations

	Another artery disease		
	Yes	No	p value
Number of patients	(n=81)	(n=19)	
Glucose (mg/dl)	100.5±33.8	91.5±32.1	0.566
Serum creatinine (mg/dl)	0.91±0.41	0.95±0.39	0.620
AST (U/L)	25.2±7.3	22.7±7.9	0.667
ALT (U/L)	27.9±7.5	25.4±7.9	0.723
LDL cholesterol (mg/dl)	149.6±41.1	146.5±53.6	0.089
Hemoglobin (mg/dl)	13.5±2.4	13.8±2.5	0.606
Platelet (10^3/μL)	285±32.6	234±9.6	<0.001
White blood cell (10^3/μL)	9.4±6.3	8.3±4.1	0.132
Neutrophil (10^3/μL)	4.9±1.1	4.1±1.8	<0.001
Lymphocyte (10^3/μL)	2.09±0.7	2.25±0.8	0.056
SII	699 (443-1139)	431 (299-599)	<0.02

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase (U/L), LDL: Low-density lipoprotein, SII: Systemic Immune-Inflammation Index

DISCUSSION

In this study, we reached results that may suggest that SII, which is considered a good inflammatory marker, can be used to predict the presence of CAAD and CAD in patients with PAD.

Peripheral arterial disease (PAD) is a group of diseases that are common and usually related to atherosclerosis, causing morbidity and mortality. It may occur due to living conditions, habits and occupation.[16,17] A significant proportion of patients with PAD have been reported to have CAD.[18-20] Some autopsy studies have shown that almost all patients with severe lower extremity PAD have extensive atherosclerotic disease in the coronary arteries.[20] In addition, a significant association between low ABI values and CAAD has been shown.[21]

As the severity of inflammation increases in the host, it is obvious that inflammation markers will increase accordingly. PAD patients had higher neutrophil counts than CAD patients, suggesting higher inflammation and thus more aerosclerosis in PAD than in CAD patients.[22,23] Bravetti et al.[22] showed that CAD patients with concurrent PAD had higher neutrophil counts than those with CAD alone. Similarly, Rossi et al.[24] in another study showed that blood leukocyte counts in patients with acute myocardial infarction increased in proportion to the plaque density in the carotid arteries. Vidacovic et al.[25] found that patients with atherosclerotic vascular disease had more elevated CRP levels when each peripheral arterial territory was included. In our study, we found that blood neutrophil counts were higher in patients with PAD, regardless of which arterial segment had atherosclerotic disease.

The ABI is an inexpensive and readily available test for the diagnosis of PAD. It has shown improved mortality prediction when PAD is determined to be present based on an ABI ≤ 0.9 and when combined with NLR.[26] A higher neutrophil count has been shown to be associated with major adverse cardiovascular events, death and a composite of myocardial infarction, stroke and death in patients with PAD.[27] Aykan et al.[28] claimed in a study that NLR is associated with the prevalence and complexity of PAD. A review by Bhat et al.[29] also evaluated that NLR was an independent predictor of early and mid-term amputation in patients with acute limb ischemia after embolectomy, a predictor of mortality and/or major amputation in critical limb ischemia, and an independent predictor of PAD severity. Songur et al.[30] found a correlation between PLR and higher amputation rate in patients with PAD. In addition, Yalım et al.[31] also claimed a correlation between PLR and NLR in multisite atherosclerosis.

There is a growing body of evidence suggesting that SII, calculated with three, is a stronger predictor of immune and inflammatory status in patients than single-component (neutrophils, lymphocytes, platelets) and two-component (PLR and NLR) inflammatory markers. There are studies showing that high SII levels are superior to NLR and PLR in predicting the risk of clinical outcomes in various diseases.[7,9,11,12] In addition, Zhang et al.[32] found that elevated SII on admission was independently associated with the presence of plaque thickness and ulceration in acute ischemic stroke. Aktemur et al.[33] found that SII is an effective predictor of mortality risk in patients with iliac artery disease undergoing percutaneous intervention. Higher SII may indicate the possibility of more complex lower extremity arterial disease.[13] Oflar et al.[13] claimed that increased SII levels are an independent predictor of CAAD severity. In our study, we found that those with CAD or CAAD had higher SII levels than those with only PAD. When evaluated together with the literature, it can be speculated that individuals with more inflammation have higher SII levels. Although NLR and PLR levels were not evaluated, when considered together with other study results, it can be concluded that SII is a better inflammatory marker for the detection of those with more extensive atherosclerotic disease.

Considering our results in terms of physiopathologic mechanisms, neutrophils invade the plaque and initiate and/or exacerbate tissue damage and inflammation by directly and indirectly affecting both endothelial and smooth muscle cells through the mediators they secrete.[35,36] With endothelial damage, monocyte/lymphocyte migration to the subendothelial region and subsequent foam cells, fatty streaks, which are the first stage of atherosclerotic plaques, are formed, and then the atherosclerotic plaque progresses with the continuation of slow inflammation. In addition, proteolytic enzymes and growth factors released from monocytes (foam cells) and lymphocytes have important roles in damage and repair.[37] Lymphopenia occurs due to physiological stress and a mechanism such as decreased cell production, tissue-level redistribution or cell apoptosis.[38] With increased lymphocyte apoptosis within the atherosclerotic plaque, plaque development progresses and destabilization occurs within the plaque.[38] Platelets are a key component of the cellular process in hemostasis, as well as being important in maintaining vascular integrity in the absence of injury and protecting against spontaneous bleeding.[39] Platelets regulate lymphocyte activation and influence the function of lymphocyte subpopulations. They also contribute to the migration and proliferation of smooth muscle cells.[37,40,41]

Limitations

This study has some limitations. The study included a small number of patients and was a single-center, retrospective study. The number of patients in our study may have been insufficient and our data set is quite old. This may have caused unintentional bias in our statistical results. In our study, we only evaluated the presence of CAAD. Therefore, we did not evaluate the carotid artery stenosis rate. Considering that the severity of carotid artery stenosis is related to LVH, this can be considered a limitation of our study. SII levels were calculated only during hospital admission. Furthermore, parameters potentially affecting atherosclerosis, such as VEGF, TGFα-β, and NO, were not measured and the lack of a follow-up study does not provide insight into the long-term changes and effects of SII.

CONCLUSION

Neutrophils and platelets positively influence inflammation and the development of atherosclerotic plaques, whereas a decrease in lymphocytes has a negative effect. These

findings suggest that SII may serve as a more accurate and comprehensive measure to predict immunologic and inflammatory/anti-inflammatory states in the individual. The results of the present study suggest that SII, a useful, simple, easily measurable and inexpensive indicator of inflammatory status, is a powerful inflammatory marker for predicting additional arterial system diseases (CAD and CAAD) in PAD patients. Larger and multicenter studies are needed to better analyze all possible predictors of disease development.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was prepared using data obtained from the Medical Master's Thesis titled ''Risk of cardiovascular and cerebrovascular diseases in patients with PAD'' which we completed in 2007 (Medical Specialization Thesis, Erciyes University, Faculty of Medicine, Department of Surgical Medical Sciences, Department of Cardiovascular Surgery, Kayseri, Turkiye, 2007 / Thesis No:193683).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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