

Original article

Reprint

Systemic inflammation biomarker levels in atrial fibrillation patients: A propensity score matching analysis

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Abstract:

Objective: to compare the levels of systemic inflammation biomarkers between groups of patients with paroxysmal and persistent atrial fibrillation (AF).

Materials and methods. The study included 232 patients with AF who underwent primary pulmonary vein ablation. The mean age was 62 (54; 67) years. Patients were distributed between two groups: with paroxysmal AF and with persistent AF.

Results. The analysis of the levels of inflammatory markers did not reveal statistically significant differences between the groups: 0.8 (0.6; 1.1) vs. 0.9 (0.7; 1.1), $p=0.077$ for the systemic inflammation response index; 361 (276; 509) vs. 373 (294; 551), $p=0.505$ for the systemic-immune inflammation index; 170 (122; 255) vs. 197 (139; 259), $p=0.150$ for the aggregate index of systemic inflammation; 1.6 (1.3; 2.2) vs. 1.8 (1.3; 2.3), $p=0.428$ for the neutrophil-to-lymphocyte ratio; 122 (86; 132) vs. 107 (91; 130), $p=0.576$, for the platelet-to-lymphocyte ratio; and 0.228 (0.177; 0.291) vs. 0.241 (0.200; 0.301); $p=0.262$ for the monocyte-to-lymphocyte ratio. No differences were found after statistical correction for baseline clinical characteristics. According to the ROC analysis, no statistically significant threshold values of the levels of the studied markers were found for each type of AF.

Conclusion. We revealed no differences in the levels of systemic inflammation biomarkers between patients with paroxysmal and persistent types of AF. Currently, data on the existence of an association between the levels of systemic inflammation biomarkers and various types of AF are contradictory, and further research in this area is required.

Keywords: atrial fibrillation, inflammation, inflammation biomarkers, systemic immune-inflammation index, systemic inflammation response index, aggregate index of systemic inflammation

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Introduction

Currently, according to published data, atrial fibrillation (AF) is the most common type of heart arrhythmia globally. It has been proven that it is associated with the development of complications that increase disability and mortality in the population [1–3].

Among the pathophysiological mechanisms for the development of AF, inflammation plays an important role, being both a potential trigger and an element of sustaining AF and its further progression [4–6]. Numerous fundamental and clinical studies examined the relationship between the levels of various inflammatory markers and AF. Many of these studies demonstrated a statistically significant association of inflammation with the recurrence of AF after treatment [7]. The levels of inflammatory markers, such as C-reactive protein, interleukins (IL-6 and IL-8), tumor necrosis factor, and others, were significantly higher in patients with AF compared with patients with sinus rhythm (SR) [8, 9].

Recently, more and more published studies on the so-called biomarkers of systemic inflammation and their impact

on the development of cardiovascular diseases appeared [10–12]. These hematological indices are based on the number of leukocytes and their subtypes: systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), aggregate index of systemic inflammation (AISI), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte-to-lymphocyte ratio (MLR). In clinical practice, these hematological biomarkers can be calculated routinely. In addition, they are considered available and easily reproducible indicators of the systemic inflammation response.

It has also been substantiated that the severity of inflammation directly correlates with the duration of AF, and in patients with persistent and permanent arrhythmia types, the levels of inflammation markers are higher than in patients with the paroxysmal type. In the study by S.E. Ömür et al. [13], a significant relationship was discovered between the levels of SII, NLR and PLR, and the duration of AF. The studied biomarkers also exhibited statistically significantly higher levels in the group of patients with permanent AF than in the group with paroxysmal AF. In patients of the SR group,

these markers had significantly lower levels compared with both groups of patients with AF.

However, published data on this matter are scarce; and, anyway, a single study cannot reflect an objective situation. The latter requires new studies and accumulation of data on the association of systemic inflammation biomarkers with AF.

Objective: comparative assessment of the levels of systemic inflammation biomarkers between groups of patients with paroxysmal and persistent AF.

Materials and methods

Study design. Data collection. Our study was retrospective. All medical records of patients with a diagnosis of AF who underwent primary pulmonary vein ablation from 2021 through 2023 in the Division of X-ray Surgical and Intraoperative Diagnostics and Treatment of Arrhythmias were selected from the archive. The data were collected from the MedWork medical information system and were subsequently analyzed.

Inclusion and exclusion criteria. The study included patients 40 to 80 years of age who underwent primary pulmonary vein ablation. Exclusion criteria were as follows: any concomitant oncological and autoimmune diseases, liver diseases, taking glucocorticosteroids in the last month, previous open-heart surgery, any surgery in the last year, moderate or severe chronic renal failure (glomerular filtration rate <50 mL/min), and a history of infective endocarditis.

Definitions. The studied indices of systemic inflammation were calculated using the following formulas:

- $SIRI = \text{neutrophil count} \times \text{monocyte count/lymphocyte count}$;
- $SII = \text{neutrophil count} \times \text{platelet count/lymphocyte count}$;
- $AISI = \text{neutrophil count} \times \text{monocyte count} \times \text{platelet count/lymphocyte count}$;
- $NLR = \text{neutrophil count/lymphocyte count}$;
- $PLR = \text{platelet count/lymphocyte count}$;
- $MLR = \text{monocyte count/lymphocyte count}$.

Statistical data processing. Quantitative data in our study have a non-normal distribution; hence, they are presented as a median, and lower and upper quartiles: Me (Q1; Q3). Categorical data are presented as a proportion (%) and a count. Comparisons of two groups was performed using the Mann–Whitney U test for quantitative data and the χ^2 test for categorical data. Differences were assumed statistically significant at $p < 0.05$.

Propensity Score Matching (PSM) was used in a 1:1 ratio to statistically adjust for baseline clinical differences between the paroxysmal and persistent AF groups. Propensity scores were calculated for each patient using multivariate logistic regression based on the following covariates: age, gender, weight, and body surface area (BSA). Receiver-operating characteristic (ROC) analysis with calculation of the area under the curve (AUC), as well as assessment of the sensitivity and specificity, were employed to identify diagnostic cutoff points for quantitative parameters (SIRI, SII, AISI, NLR, PLR, and MLR levels). Statistical analyses were performed using Statistica 10 (Statsoft, USA) and

MedCalc (MedCalc Software Ltd, Belgium) software. PSM was conducted using IBM SPSS® Statistics 26.0 (USA).

Results

A total of 1,016 patients underwent primary pulmonary vein ablation for AF from 2021 through 2023. Patients with a repeat procedure and those who did not meet the inclusion/exclusion criteria were excluded. The final analysis included 232 patients (130 men and 102 women) ranging 54 – 67 years of age. The median age of the study cohort was 62 (54; 67) years. The proportion of men in the sample was 56%. Their general clinical characteristics are reviewed in *Table 1*.

The paroxysmal AF group included 186 patients, while the persistent AF group encompassed 46 individuals. The comparison of the two groups yielded no statistically significant differences in the main clinical characteristics, except for the following parameters: gender, weight and BSA (*Table 2*). The proportion of men was higher in the persistent AF group (70% vs. 53%, $p = 0.039$), and body weight was significantly greater in the persistent AF group patients: 93 (80; 105) kg vs. 87 (74; 97) kg, $p = 0.014$. The BSA was statistically significantly higher in the persistent AF group: 2.17 (1.99; 2.3) m² vs. 2.06 (1.86; 2.2) m², $p = 0.013$. However, the studied biomarkers of systemic inflammation (SIRI, SII, AISI, NLR, PLR and MLR) did not differ between the two groups (*Table 2*).

Table 1. General characteristics of patients included in the study

Parameter	Value (n=232)
Age, years	62 (54;67)
Male gender, % (count)	56 (130)
Weight, kg	89 (75;99)
BSA, m ²	2.07 (1.88;2.2)
BMI, score	30 (27;33)
AF type % (count):	
Paroxysmal	80 (186)
Persisting	20 (46)
AF duration, months	4 (2;7)
CHA2DS2VASc, pts	2 (1;3)
HAS-BLED, pts	1 (1;2)
Diabetes, % (count)	12 (27)
CAD, % (count)	19 (44)
COPD, % (count)	2 (5)
HTN, % (count)	75 (175)
Acute CVA, % (count)	4 (9)
Smoking, % (count)	16 (38)

Here and below in Tables 2 and 3: BSA, body surface area; BMI, body mass index; AF, atrial fibrillation; CHA2DS2-VASc, scale for assessing the risk of developing thromboembolic complications in patients with AF; HAS-BLED, bleeding risk score; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; HTN, hypertension; CVA, cerebrovascular accident.

Table 2. Parameter values by group

Parameter	Groups based on AF type		p
	Paroxysmal (n=186)	Persisting (n=46)	
Age, years	62 (54;67)	59 (51;67)	0.157
Male gender, % (count)	53 (98)	70 (32)	0.039*
Weight, kg	87 (74;97)	93 (80;105)	0.014*
BSA, m ²	2.06 (1.86;2.2)	2.17 (1.99;2.3)	0.013*
BMI, score	29.4 (26;33)	30.5 (27;34)	0.164
AF duration, months	4 (2;8)	3 (1;7)	0.088
CHA2DS2VASc, pts	2 (1;3)	2 (1;3)	0.057
HAS-BLED, pts	1 (1;2)	1 (1;1)	0.062
Diabetes, % (count)	12 (23)	9 (4)	0.489
CAD, % (count)	20 (38)	13 (6)	0.248
COPD, % (count)	2 (3)	4 (2)	0.255
HTN, % (count)	77 (144)	67 (31)	0.158
Acute CVA, % (count)	4 (7)	4 (2)	0.857
Smoking, % (count)	15 (27)	24 (11)	0.124
Biomarkers of systemic inflammation			
SIRI	0.8 (0.6; 1.1)	0.9 (0.7; 1.1)	0.077
SII	361 (276; 509)	373 (294; 551)	0.505
AISI	170 (122; 255)	197 (139; 259)	0.150
NLR	1.6 (1.3; 2.2)	1.8 (1.3; 2.3)	0.428
PLR	122 (86; 132)	107 (91; 130)	0.576
MLR	0.228 (0.177; 0.291)	0.241 (0.200; 0.301)	0.262

* statistically significant differences. Here and below in Tables 3 and 4: SIRI, systemic inflammation response index; SII, systemic immune-inflammation index; AISI, aggregate index of systemic inflammation; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; MLR, monocyte-to-lymphocyte ratio.

After statistical correction via PSM, each group included 46 patients, whose clinical characteristics no longer exhibited statistically significant differences. The levels of SIRI, SII, AISI, NLR, PLR and MLR did not differ statistically significantly between the groups, just as before correction (Table 3).

We then performed ROC analysis to identify cutoff values of systemic inflammation biomarkers that would be associated with paroxysmal or persistent types of AF (Table 4).

The results of the ROC analysis also did not reveal statistically significant cutoff values for the SIRI, SII, AISI, NLR, PLR, and MLR levels for the AF type. The values of AUC, sensitivity and specificity were extremely low (Table 4).

Table 3. Parameter values by group after correction

Parameter	Groups based on AF type		p
	Paroxysmal (n=186)	Persisting (n=46)	
Age, years	61.5 (53;66)	59 (51;67)	0.681
Male gender, % (count)	59 (27)	70 (32)	0.281
Weight, kg	90 (77;104)	93 (80;105)	0.496
BSA, m ²	2.13 (1.96;2.3)	2.17 (1.99;2.3)	0.775
BMI, score	31 (26;33)	30.5 (27;34)	0.678
AF duration, months	5 (2;7)	3 (1;7)	0.201
CHA2DS2VASc, pts	2 (1;3)	2 (1;3)	0.278
HAS-BLED, pts	1 (1;2)	1 (1;1)	0.294
Diabetes, % (count)	4 (2)	9 (4)	0.276
CAD, % (count)	11 (5)	13 (6)	0.754
COPD, % (count)	0 (0)	4 (2)	0.159
HTN, % (count)	80 (37)	67 (31)	0.094
Acute CVA, % (count)	4 (2)	4 (2)	0.991
Smoking, % (count)	13 (6)	24 (11)	0.183
Biomarkers of systemic inflammation			
SIRI	0.7 (0.6; 1.1)	0.9 (0.7; 1.1)	0.123
SII	347 (284; 522)	373 (294; 551)	0.561
AISI	160 (124; 285)	197 (139; 259)	0.180
NLR	1.6 (1.3; 2.1)	1.8 (1.3; 2.3)	0.540
PLR	104 (86; 125)	107 (91; 130)	0.959
MLR	0.227 (0.163; 0.294)	0.241 (0.200; 0.301)	0.254

Table 4. ROC analysis results

Parameter	Cutoff point	AUC (95% confidence interval)	Se	Sp	p
SIRI	≤0.72	0.584 (0.518–0.648)	44.6	76.1	0.061
SII	≤293	0.532 (0.465–0.597)	32.8	76.1	0.503
AISI	≤167	0.568 (0.502–0.633)	47.3	69.6	0.130
NLR	≤2.1	0.538 (0.471–0.603)	74.2	37.0	0.428
PLR	>97.1	0.527 (0.460–0.592)	65.6	45.7	0.563
MLR	≤0.158	0.553 (0.487–0.619)	20.4	93.5	0.246

ROC, receiver-operating characteristic; AUC, area under the curve; Se, sensitivity; Sp, specificity.

Discussion

Our study did not reveal any differences in the levels of systemic inflammation biomarkers between patients with paroxysmal AF and persistent AF. The values of SIRI, SII, AISI, NLR, PLR, and MLR, both before and after statistical correction for clinical differences, did not differ significantly between the two groups of patients with different AF types. We found only two studies (published in 2023 and 2024) that compared the levels of SIRI, SII, AISI, NLR, PLR, and MLR between patients with paroxysmal and persistent AF. The results of these studies contradicted our results.

S.E. Ömür et al. [13] compared the levels of inflammatory biomarkers between patients with paroxysmal (n=145) and persistent AF (n=206), and also with normal SR (n=140). A total of 752 patients were included in the study. All groups differed statistically significantly in age, laboratory and instrumental data, and the entire cohort was characterized by pronounced comorbidity: for example, approximately 30–35% of patients in all groups suffered from diabetes and coronary artery disease. In our study, the proportion of these comorbidities was significantly lower (12% and 19%, respectively.) We selected clear inclusion/exclusion criteria, which made our patient cohort more homogeneous. Besides that, we used the propensity score matching method to match the compared groups.

S.E. Ömür et al. did not make any adjustments for clinical and laboratory heterogeneity between the groups. The mean value of the NLR parameter was 4.53 (0.27–17.94) for the permanent AF, 3.09 (0.40–11.0) for the paroxysmal AF, and 2.34 (0.61–13.51) for the normal SR group; $p < 0.05$. Significant differences were also obtained for the PLR parameter: the means were 209.71 (40.73–604) for the permanent AF, 188.51 (53.95–617.46) for the paroxysmal AF, and 130.40 (26.42–680.39) for the normal SR group; $p < 0.05$. Mean value of SII in the permanent AF group was 1,569.54 (139–6,069), while in paroxysmal AF group it amounted to 1,035.09 (133–4,013); in the group with normal SR, SII was 629.47 (104–4,695); $p < 0.05$. Hence, the study [13] revealed a dependence of all chronic inflammation markers on the duration and the type of AF, and the lowest values of chronic inflammation markers were observed in patients with SR.

A similar study by A. Naser et al. [14] involved 453 patients (252 women and 201 men aged 44 to 94 years) with AF (138 patients with paroxysmal AF and 315 study subjects with permanent AF). The mean age of individuals with permanent AF was statistically significantly higher than that of individuals with paroxysmal AF (73.44±8.98 years vs. 70.32±8.26 years; $p = 0.001$). No differences were found between the groups in gender, anthropometric data, or comorbidities, except for diabetes mellitus. The frequency of the latter was significantly higher in patients with permanent AF than in patients with paroxysmal AF (41.6% vs. 26.1%). The levels of NLR, PLR, SII, C-reactive protein, and red blood cell distribution width were significantly larger in patients with permanent AF than in patients with paroxysmal AF. In contrast, the mean lymphocyte count in patients with permanent AF was significantly lower than in patients with paroxysmal AF.

The PLR level was 109.75 (90.46–139.90) for individuals with paroxysmal AF and 133.94 (98.15–180.95) for individuals with permanent AF; $p < 0.001$. NLR level was 1.86 (1.36–2.57) for paroxysmal AF and 2.39 (1.85–3.47) for permanent AF; $p < 0.001$. SII amounted to 444.11 (309.25–601.12) for paroxysmal AF and 562.50 (386.41–897.66) for permanent AF; $p < 0.001$.

In addition to the fact that the previous two studies, the design and objectives of which were similar to ours, dealt with heterogeneous comparison groups, their authors did not employ any mathematical techniques to balance these groups. We would also like to point to the absolute values of biomarker levels in all three studies. For example, the SII level in the study by S.E. Ömür et al. [13] was 1,569.54 (139–

6,069) for persistent AF, and 1,035.09 (133–4013) for paroxysmal AF. Same parameters in the study by A. Naser et al. [14] amounted to 562.50 (386.41–897.66) for persistent AF and 444.11 (309.25–601.12) for paroxysmal AF. In our study, the SII level was 373 (294–551) for persistent AF and 347 (284–522) for paroxysmal AF. This implies marked heterogeneity of patient cohorts in all three studies.

Indeed, the results of our study differ from the data obtained by the authors of the two previously described studies. This difference can be accounted for by a higher proportion of patients in previous studies suffering from coronary artery disease and other concomitant pathology, their initial severity and comorbidity. Probably, higher values of biomarker levels are due to a high proportion of coronary artery disease and the prevalence of atherosclerosis. The role of chronic inflammation biomarkers was investigated in several studies on patients with coronary artery atherosclerosis, and it was shown that their level was higher in patients with atherosclerosis than without it [15, 16].

Currently, the pathophysiological mechanisms of AF are actively studied. A growing volume of evidence indicates a direct relationship between systemic inflammation and the development of this arrhythmia type [17–19]. Data have also accumulated demonstrating that the burden of AF is directly associated with the level of inflammation. The role of inflammation biomarkers in the pathogenesis of AF requires further elucidation and accumulation of relevant evidence.

Conclusion

We did not discover any differences in the level of systemic inflammation biomarkers between patients with paroxysmal and persistent types of AF. For higher reliability, we used two methods of statistical verification: comparison of two independent groups by quantitative variables (Mann-Whitney U test) and ROC analysis with plotting the area under the curve and assessing sensitivity and specificity.

Because of currently existing conflicting results, it is premature to draw any conclusions about the presence or absence of associations of biomarker levels with different AF types. Further data accumulation, along with studies with larger sample sizes and more homogenous patient cohorts, are required.

Author contributions. All authors made contributed equally to the preparation of the manuscript.

Conflict of interest. The study has no commercial interest, nor any interest of other legal entities or individuals. No conflicts of interest are declared by the authors.

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