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FACTORS ASSOCIATED WITH RESISTANCE TO RESOLUTION OF LEFT ATRIAL APPENDAGE THROMBUS IN PATIENTS WITH ATRIAL FIBRILLATION: 12-MONTH FOLLOW-UP RESULTS

A.V.Belokurova, A.V.Mamarina, N.Yu.Khorkova, T.P.Gizatulina

Tyumen Cardiology Research Center, Tomsk National Research Medical Center, Russia, Tyumen, 111 Melnikaite str.

Aim. To study the dynamics of left atrial appendage (LAA) thrombosis and to determine the factors associated with resistant LAA thrombus in patients with non-valvular atrial fibrillation (AF) during 12 months of follow-up.

Methods. A prospective study included 83 patients with LAA thrombosis detected by transesophageal echocardiography (TEE). The end point was resolution or stability of the thrombus. All patients underwent clinical examination, complete blood count and biochemical blood test, coagulation testing, transthoracic echocardiography (TTE) and TEE.

Results. According to the results of TEE, the patients were divided into two groups: group 1 (n=45) with resolution LAA thrombus and group 2 (n=38) with resistant LAA thrombus. Group 2 patients were more likely to take beta-adrenoblockers (57.9% and 31.1%, p=0.014), diuretics (60.5% and 35.6%, p=0.023) and rivaroxaban (39,5% и 13,3%, p=0,010). According to TTE data, group 2 had a higher right atrial volume index (30.7 [24.7; 34.7] vs 24.5 [21.0; 32.2] ml/m^2, respectively, p=0.034). Laboratory data analysis showed that group 2 had higher mean platelet volume (MPV) levels (9.1 [8.3; 9.8] vs 8.4 [7.9; 9.4] fl, p=0.035), platelet distribution width (PDW) (15.9 [15.7; 16.2] vs 15.7[15.5; 15.9] %, p=0.007) and platelet large cell ratio (P-LCR) (30.0±9.2 vs 25.3±7.4%, p=0.014).

There were significant direct correlations of MPV and P-LCR with the following parameters: right atrial volume, left atrial volumes, pulmonary artery systolic pressure, red blood cell level, hemoglobin level and hematocrit. The inverse association of MPV and P-LCR was with platelet count.

Conclusions. Resistance of LAA thrombus to resolution in patients with non-valvular AF is associated with morphofunctional parameters of platelets, which correlate with atrial structural remodeling. The results obtained indicate the need to continue research aimed at studying the contribution of the platelet activity to resistance to LAA thrombus, despite taking oral antocoagulants.

Key words: mean platelet volume; platelet distribution width; platelet large cell ratio; left atrial appendage thrombus; atrial fibrillation; transesophageal echocardiography

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Corresponding author: Belokurova Alfira, E-mail: alfira_m@inbox.ru

A.V.Belokurova - ORCID 0000-0002-6049-8985, A.V.Mamarina - ORCID 0000-0002-8160-7060, N.Yu.Khorkova - ORCID 0000-0002-7083-3214, T.P.Gizatulina - ORCID 0000-0003-4472-8821

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Atrial Fibrillation (AF) is a heart rhythm disorder associated with the risk of thrombus formation in the left atrial appendage (LAA). The development of LAA thrombosis occurs with the involvement of the factors of Virchow's triad: reduced blood flow velocity in the LAA, endothelial injury, and hypercoagulability. According to current literature, the prevalence of LAA thrombosis varies from 1.1% to 8% [1-4]. The presence of a thrombus in the LAA is an obstacle to restoring sinus rhythm in patients with persistent AF and performing catheter ablation (CA). To date, clear guidelines for managing such patients have not been developed. However, considering the need for restoring sinus rhythm and performing CA to prevent the progression of chronic heart failure (CHF), attempts are made to perform thrombolysis using various regimens of anticoagulant therapy: the use of oral anticoagulants (OACs) if none were previously taken, switching from one OAC to another, changing from OAC to parenteral anticoagulants, prescribing rivaroxaban 30 mg/day in a twice-daily regimen, or increasing the dose of warfarin to achieve an INR in the range of 2.5-3.5 [5-9]. Despite this, literature reports indicate that, in some cases, LAA thrombosis remains resistant to thrombolysis even with anticoagulant therapy [10-12]. This explains the relevance of identifying factors that impede LAA thrombus resolution in patients with non-valvular AF.

Aim: to study the dynamics of LAA thrombosis and identify factors associated with resistance to LAA thrombus resolution over a 12-month period.

METHODS

A prospective study included 83 patients from a total of consecutively enrolled patients with AF from 2018 to 2024, in whom LAA thrombosis was detected by transesophageal echocardiography (TEE) prior to planned CA or

electrical cardioversion. 76 patients (91.6%) were OAC. During the first 3 months, the OAC regimen was not changed. However, in cases of recurrent LAA thrombus detection on follow-up TEE, a switch between OACs was performed. Patients who had not previously received anticoagulant therapy were prescribed OACs upon admission to the hospital.

Patients with LAA thrombosis were included in a prospective follow-up, the duration of which depended on the thrombus resolution time (ranging from 3 to 12 months) or was limited to 12 months. The end point of the follow-up was either thrombus resolution or stability of the LAA thrombus. In the case of thrombus resolution, the patient was removed from further follow-up.

Table 1.

Clinical and demographic characteristics of the patients included in the study

Indicator	Value
Age, years	62 [55.5; 65.5]
Male sex, n (%)	49 (59)
Female sex, n (%)	34 (41)
AH, n (%)	79 (95.2):
Stage 1 AH, n (%)	8 (10.1)
Stage 2 AH, n (%)	30 (38)
Stage 3 AH, n (%)	41 (51.9)
CAD, n (%)	53 (63.9)
History of MI, n (%)	2 (2.4)
DM, n (%)	14 (16.9)
Obesity, n (%)	55 (66.3):
Stage 1 obesity, n (%)	32 (58.2)
Stage 2 obesity, n (%)	19 (34.5)
Stage 3 obesity, n (%)	4 (7.3)
CKD, n (%)	14 (16.9)
CHF IIA stage and above, n (%)	6 (7.2)
Paroxysmal AF, n (%)	39 (47)
Persistent AF, n (%)	44 (53)
Non-smokers, n (%)	56 (67.5)
Former smokers, n (%)	14 (16.9)
Current smokers, n (%)	13 (15.7)
Average CHA_2DS_2-VASc score	2 [2; 3]
Low risk, n (%)	1 (1.2)
Moderate risk, n (%)	27 (32.5)
High risk, n (%)	55 (66.3)
HAS-BLED	1 [0; 1]
HAS-BLED 0 points, n (%)	41 (49.4)
HAS-BLED 1 point, n (%)	37 (44.6)
HAS-BLED 2 points, n (%)	5 (6)

Note: AH - Arterial Hypertension; CAD - Coronary Artery Disease; MI - Myocardial Infarction; DM - Diabetes Mellitus; CKD - Chronic Kidney Disease (eGFR less than 60 ml/min using the EPI formula); CHF - Chronic Heart Failure. Thromboembolic Risk: Low risk according to the CHA2DS2-VASc scale: 0 points for men and 1 point for women; Moderate risk: 1 point for men and 2 points for women; High risk: 2 or more points for men and 3 or more points for women.

Inclusion Criteria

- Presence of AF (paroxysmal or persistent, non-valvular etiology) lasting more than 30 seconds, confirmed by ECG or 24-hour Holter ECG monitoring, in patients of any gender and age.
- First detected LAA thrombosis by TEE.
- Signed informed consent to participate in the study.

Exclusion Criteria

- Permanent AF.
- Refusal to participate in the study.

All patients underwent general clinical examinations, including complete blood count, biochemical blood tests, coagulation testing, ECG, transthoracic echocardiography (TTE), and TEE. TTE was performed using General Electric "Vivid E9" and General Electric "Vivid S70" machines with a multi-frequency sector probe (2.5-5.0 MHz). TEE was performed using General Electric "Vivid E9" and General Electric "Vivid S70" machines with a transesophageal probe initially and during follow-up at intervals of 3 months until LAA thrombus resolution or up to 12 months. During TEE, the presence of LAA thrombosis, spontaneous echo contrast, and blood flow velocity in the LAA were assessed. The size of the thrombus was not considered at this stage of the study.

Complete blood count was performed using the Mindray BC-5800 automatic analyzer (China), biochemical blood tests using the Mindray BC-480 automatic analyzer (China), and coagulation testing using the Destiny Plus coagulometer (Ireland). ECG was conducted using the Poli-spectr device (Neurosoft).

Statistical analysis

Data were analyzed using the IBM SPSS Statistics software package. The Kolmogorov-Smirnov test was used to assess the normality of data distribution. The results are presented as mean and standard deviation (M±SD) for normally distributed quantitative variables, and as median (Me) and interquartile range (25%; 75%) for non-normally distributed data. The significance of differences between the two groups for normally distributed quantitative data was determined using the Student's t-test, and for non-normally distributed data, the Mann-Whitney U test was used. For comparing categorical variables, the chi-square test or Fisher's exact test was applied. Differences were considered statistically significant when $p < 0.05$.

Correlation analysis between quantitative variables was performed using Pearson's correlation for normally distributed data, and Spearman's rank correlation for non-normally distributed data.

The study was conducted in accordance with the Declaration of Helsinki principles. The study protocol was approved by the local ethics committee (protocol No. 136, 06.04.2018). Informed consent for participation in the study was obtained from all participants.

RESULTS

A total of 83 patients with AF and LAA thrombosis were included in the final analysis. The clinical-de-

mographic characteristics of the overall patient group are shown in Table 1. Among the patients with diagnosed LAA thrombosis, there was a predominance of males, and more than half of the patients had persistent AF. Almost all patients had hypertension, and one in six had chronic kidney disease. Considering that cardiovascular diseases were common in patients with LAA thrombosis, most had a high thromboembolic risk according to the CHA_2DS_2-VASc scale, with no patients having a history of thromboembolic complications or high bleeding risk according to the HAS-BLED scale. It is worth noting that 1 patient had a low thromboembolic risk.

The median thrombus resolution time was 6 [3; 6] months, with the majority of patients (n=24) having thrombus resolution in the first 3 months. During the following 3 months, 14 patients showed resolution, and another 7 patients by the end of the year. Based on TEE results, patients were divided into two groups: Group 1 (n=45), who had thrombus resolution within 12 months, and Group 2 (n=38), who did not have thrombus resolution.

When comparing the clinical-demographic parameters (Table 2), Groups 1 and 2 were comparable in terms of age and sex, with no statistically significant differences in the prevalence of cardiovascular diseases. The analysis showed that patients who did not experience thrombus resolution were more likely to take beta-blockers and diuretics, which is likely related to the need for a rate control strategy and a higher risk of CHF. No significant differences were observed in other drug categories.

A comparative analysis of echocardiographic parameters (Table 3) between groups revealed that patients with persistent thrombus in the LAA had a higher right atrial volume index, while the left atrial volume index did not differ between the groups. Additionally, there was a trend toward a thicker left ventricular posterior wall in Group 2. In Group 1, after thrombus resolution, spontaneous echo contrast persisted in 11 patients (24.4%).

The laboratory data analysis (Table 4) revealed statistically significant differences in the complete blood count parameters related to the platelet component of hemostasis. In patients with persistent LAA thrombus, mean platelet volume (MPV), platelet distribution width (PDW), and platelet large cell ratio (P-LCR) were significantly higher. The total leukocyte count, although within the reference range, was higher in Group 2, primarily due to lymphocytes, with a tendency toward higher neutrophil levels. The target INR was achieved in only 4 patients taking warfarin: 1 (14.3%) in Group 1 and 3 (75%) in Group 2.

Statistically significant correlations of MPV and P-LCR with other laboratory parameters and echocardiographic data are shown in Table 5. Significant positive correlations were found between MPV and P-LCR with indexed volumes of both atria and the left atrial diameter, pulmonary artery systolic pressure, and levels of red blood cells, hemoglobin, and hematocrit. An inverse relationship between MPV and P-LCR and platelet count was observed. Our results indicated that, in patients with LAA thrombosis, platelet morphological features are associated with structural remodeling of both atria.

DISCUSSION

The issue of effective LAA thrombus resolution in patients with non-valvular AF raises many questions,

Table 2.

Clinical and demographic data of patients with resolved and persistent LAA thrombus

Indicator	Group 1 (n=45)	Group 2 (n=38)	p
Age, years	61.1±8.4	59.9±7.3	0.490
Male sex, n (%)	26 (57.8)	23 (60.5)	0.800
Female sex, n (%)	19 (42.2)	15 (39.5)	
BMI, kg/m²	31.6±5.7	32.7±4.6	0.332
AH, n (%)	43 (95.6)	36 (94.7)	0.617
Stage 1 AH, n (%)	6 (14)	2 (5.5)	
Stage 2 AH, n (%)	15 (34.9)	15 (41.7)	
Stage 3 AH, n (%)	22 (51.1)	19 (52.8)	
CAD, n (%)	30 (66.7)	23 (60.5)	0.562
History of MI, n (%)	1 (2.2)	1 (2.6)	0.904
CHF IIA stage and above, n (%)	3 (6.7)	3 (7.9)	0.688
DM, n (%)	7 (15.6)	7 (18.4)	0.775
CKD, n (%)	7 (15.6)	7 (18.4)	0.775
History of bleeding, n (%)	2 (4.4)	1 (2.6)	0.564
History of anemia, n (%)	2 (4.4)	4 (10.5)	0.405
Paroxysmal AF, n (%)	24 (53.3)	15 (39.5)	0.208
Persistent AF, n (%)	21 (46.7)	23 (60.5)	
Medication Therapy			
Amiodarone, n (%)	6 (13.3)	1 (2.6)	0.118
Propafenone, n (%)	2 (4.4)	4 (10.5)	0.405
Sotalol, n (%)	14 (31.1)	6 (15.8)	0.127
Allapinin, n (%)	4 (8.9)	4(10.5)	0.801
Beta-blockers, n (%)	14 (31.1)	22 (57.9)	0.014
ACE inhibitors / ARBs, n (%)	34 (75.6)	31 (81.6)	0.398
Statins, n (%)	30 (66.7)	26 (70.3)	0.727
Diuretics, n (%)	16 (35.6)	23 (60.5)	0.023
Calcium antagonists, n (%)	7 (15.6)	13 (35.1)	0.069
Oral Anticoagulants			
Warfarin, n (%)	7 (15.6)	4 (10.5)	0.538
Apixaban, n (%)	15 (33.3)	11 (28.9)	0.668
Rivaroxaban, n (%)	6 (13.3)	15 (39.5)	0.010
Dabigatran, n (%)	13 (28.9)	5 (13.2)	0.111

Note: BMI - body mass index; ACE inhibitors - angiotensin-converting enzyme inhibitors; ARBs - angiotensin receptor blockers

as there are no clear recommendations in the contemporary literature regarding treatment methods and timelines for such patients. According to the results of our study, LAA thrombosis persisted in 45.8% of patients during the 12-month follow-up, which is comparable to the findings of P. Bernhardt et al., where thrombus resolution was also absent in 44% of patients during 1 year of follow-up, despite antithrombotic therapy [11]. In the study by E.S. Kropacheva et al., the results were worse: adequate therapy with vitamin K antagonists led to thrombus resolution in only 43.7% of cases over the course of a year [12]. In contrast, a high efficacy of anticoagulant therapy for LAA thrombus resolution was presented in the study by M.C. Saaed et al.: 67 (12.9%) of 520 patients with non-valvular AF had LAA thrombus; after four weeks of warfarin therapy, 18 (90%) patients showed thrombus resolution [13].

Regarding the timing of LAA thrombus resolution, the median in our study was 6 [3; 6] months, with 53% of patients (n=24) experiencing thrombus resolution within the first 3 months. In the study by A.D. Niku et al., 60% of patients had LAA thrombus resolution based on TEE performed at 96±72 days [14]. In E.S. Mazur et al.'s study, the median thrombus resolution time was found to be 30 [22.0-41.0] days, which is significantly shorter than our result [15].

In our study, we found that patients with persistent thrombus were more likely to use rivaroxaban than those in Group 1: 39.5% vs 13.3% respectively (p=0.010). This aligns with the findings of A. Lenart-Migdalska et al., who showed an increase in circulating microparticles produced by platelets and the endothelium, playing a prothrombotic role at the peak concentration of rivaroxaban in plasma [16]. We attribute these results to the high variation in the drug concentration at its peak and at the end of its action due to single-dose administration. No significant differences were found for other types of OACs, which is consistent with previously published works [14, 17, 18].

The higher NT-proBNP levels observed in patients with persistent LAA thrombus suggest more pronounced subclinical left ventricular systolic dysfunction, although there were no significant differences in left ventricular volumes and LVEF. We also hypothesize that Group 2 patients had more pronounced diastolic dysfunction, which is supported by statistically significant differences in the indexed right atrial volume. In the study by A. Watanabe et al., patients with warfarin-resistant LAA thrombus had a lower LVEF compared to patients with thrombus resolution [19]. Similar results were obtained by M. Hautmann et al.: when comparing 450 patients with LAA thrombus and 481 without LAA thrombus, there was a higher frequency of CHF progression and cardiac chamber dilation in the thrombus group [20].

Key differences in patients with resistant LAA thrombus were identified in the comparison of laboratory data. A higher leukocyte count - due to lymphocytes and to a lesser extent neutrophils - was observed in Group 2, which generally corresponded to reference values and was not associated with an increase in C-reactive protein or neutrophil-lymphocyte ratio. Therefore, we cannot consider these features as manifestations of chronic inflammation in patients with resistant LAA thrombus. However, previous studies by Y. Deng et al. showed that neutrophil-lymphocyte ratio acted as an independent predictor of LAA thrombus presence or spontaneous echo contrast in non-valvular AF patients [21]. Furthermore, research by Y. Feng et al. using Mendelian randomization demonstrated that genetically predicted increases in CD4+ T-lymphocyte count were associated with an increased risk of AF development [22].

Table 3.

Echocardiographic parameters of patients with resolved and persistent LAA thrombus

	Group 1 (n=45)	Group 2 (n=38)	p
RA volume index, ml/m²	24.5 [21.0; 32.2]	30.7 [24.7; 34.7]	0.034
LA volume index, ml/m²	36.6 [30.9; 46.4]	38.5 [31.3; 45.1]	0.942
LV ESD index, mm/m²	17.0±3.0	17.0±2.0	0.997
LV EDD index, mm/m²	24.5±2.8	24.3±3.1	0.869
LV EDV index, ml/m²	49.9±11.0	52.8±16.1	0.334
LV ESV index, ml/m²	18.1 [15.7; 24.6]	19.8 [15.4; 25.7]	0.759
IVS, mm	12 [11; 12]	12 [11; 13]	0.233
PW LV, mm	10 [10; 11]	11 [10; 12]	0.060
MM LV, g	207 [181; 227]	218 [185; 249]	0.271
MM LV index, g/m²	100.0±17.6	107.4±28.2	0.146
SV LV, ml	60.1±13.7	65.4±20.8	0.173
LVEF, %	60 [57; 64]	59.5 [55; 64]	0.787
PASP, mmHg	27 [25; 30]	28 [25; 35]	0.173
Flow velocity in LAA, cm/s	35 [30; 42]	32 [30; 36]	0.113

Note: RA - right atrium; LA - left atrium; EDD, ESD, EDV, and ESV - end-diastolic and end-systolic dimensions and volumes; LV - left ventricle; IVS - interventricular septum; PW - posterior wall; MM - myocardial mass; SV - stroke volume; LVEF - left ventricular ejection fraction; PASP - pulmonary artery systolic pressure; LAA - left atrial appendage.

In our study, patients with persistent LAA thrombus had higher platelet morphological parameters, such as MPV, PDW, and P-LCR. As is known, MPV reflects the degree of platelet maturity circulating in the blood, PDW indicates the degree of anisocytosis and the presence of platelet aggregates or fragments, and P-LCR reflects the number of cells with high thrombus formation activity [23].

High MPV levels were previously found by N. Bayar et al. in patients who had suffered a stroke or transient ischemic attack [24]. Similar results were obtained by S.W. Choi et al.: in their study of 352 AF patients, MPV was identified as a predictor of stroke or the presence of LAA thrombus [25]. Additionally, it has been reported that patients with a low INR level (less than 2.0) had higher MPV, PDW, and P-LCR compared to patients with therapeutic INR levels [26].

Table 4.

Comparison of laboratory parameters in patients with resolved and persistent LAA thrombus

	Group 1 (n=45)	Group 2 (n=38)	p
Complete blood count			
Leukocytes, 10^9/L	5.7±1.7	6.7±1.8	0.014
Erythrocytes, 10^9/L	4.8±0.5	5.0±0.5	0.097
Hemoglobin, g/L	140.6±15.7	146.5±14.0	0.083
Hematocrit, %	43.8±5.3	45.3±4.4	0.160
Platelets, 10^9/L	211.0±45.4	224.3±64.3	0.276
Thrombocrit, %	0.17 [0.15; 0.19]	0.20 [0.15; 0.22]	0.266
MPV, fl	8.4 [7.9; 9.4]	9.1 [8.3; 9.8]	0.035
PDW, %	15.7 [15.5; 15.9]	15.9 [15.7; 16.2]	0.007
P-LCR, %	25.3±7.4	30.0±9.2	0.014
Neutrophils, 10^9/L	3.0 [2.4; 4.0]	3.6 [3.0; 4.3]	0.065
Lymphocytes, 10^9/L	1.7 [1.4; 2.2]	2.1 [1.6; 2.6]	0.033
NLR	1.8 [1.2; 2.2]	1.7 [1.4; 2.0]	0.981
Biochemical blood test			
Glucose, mmol/L	5.7 [5.2; 6.2]	5.6 [5.3; 6.4]	0.955
Creatinine, μmol/L	86 [79; 91]	85.4 [77; 101]	0.531
eGFR, ml/min/$^{1.73}m^2$	75.8±15.2	73.7±12.7	0.507
AST, U/L	23.4 [19.8; 27.5]	22.2 [17.6; 27.4]	0.635
ALT, U/L	24.5 [20.2; 38.5]	26.1 [19.4; 37.1]	0.680
TC, mmol/L	4.3±1.0	4.5±1.0	0.294
HDL-C, mmol/L	1.3 [1.1; 1.5]	1.2 [1.0; 1.4]	0.512
LDL-C, mmol/L	2.4 [1.9; 2.9]	2.6 [2.1; 3.1]	0.162
TG, mmol/L	1.2 [1.0; 1.5]	1.3 [1.1; 1.8]	0.457
CRP, mg/L	1.9 [1.1; 4.4]	1.9 [0.9; 3.7]	0.563
NT-proBNP, pg/ml	280 [78; 639]	599 [128; 1656]	0.058
Coagulation test			
APTT	33.7 [30.8; 38.7]	34.5 [30.8; 40.5]	0.625
Fibrinogen	3.1±0.5	3.0±0.6	0.652
Thrombin Time	18.6 [17.0; 26.6]	18.6 [17.1; 26.0]	0.731
D-dimer	0.29 [0.18; 0.37]	0.29 [0.23; 0.40]	0.374
Antithrombin III, %	93.2±20.8	93.4±21.4	0.974
PT	82.9 [72.6; 91.6]	78.6 [67.5; 87.0]	0.235

Note: MPV - mean platelet volume; PDW - platelet distribution width; NLR - neutrophil-lymphocyte ratio; P-LCR - platelet large cell ratio; eGFR - estimated glomerular filtration rate; AST - aspartate aminotransferase; ALT - alanine aminotransferase; TC - total cholesterol; HDL-C - high-density lipoproteins; LDL-C - low-density lipoproteins; TG - triglycerides; CRP - c-reactive protein; NT-proBNP - n-terminal pro b-type natriuretic peptide; APTT - activated partial thromboplastin time; PT - prothrombin time; INR - international normalized ratio.

Correlation Analysis

Correlation analysis showed that platelet morphological parameters positively correlated with erythrocyte count, hemoglobin levels, and both atrial volumes. In the study by X. Zhou et al., a higher erythrocyte count was observed in patients with LAA thrombus compared to those without it, with RDW (red cell distribution width) being a predictor of LAA thrombus presence in non-valvular AF patients [27].

The direct correlation between MPV and P-LCR with the atrial volume indices confirms the relationship between platelet activation and structural remodeling of the atria. Our research group previously noted an association between LAA thrombosis and polymorphisms in platelet receptor genes for collagen (integrin A2, ITGA2) and fibrinogen (integrin B3, ITGB3). The presence of polymorphisms in both genes was associated with the most pronounced structural remodeling of the left atrium [28].

Table 5.

Results of correlation analysis

	MPV		P-LCR	
	CC r	p	CC r	p
Hemoglobin	0,268	0,017	0,245	0,032
Hematocrit	0,280	0,012	0,300	0,008
Erythrocytes	0,281	0,012	0,248	0,029
Platelets	-0,313	0,005	-0,430	<0,001
RA VI	0,262	0,022	0,282	0,015
LA VI	0,225	0,046	0,270	0,018
PASP	0,235	0,040	0,267	0,021

Note: CC - correlation coefficient; VI - volume index.

Further research is needed to study the development of resistance to LAA thrombus resolution and to develop methods for overcoming this resistance.

Study Limitation

The main limitations of the study are the small sample size and the lack of a unified approach to prescribing antithrombotic therapy.

CONCLUSION

Resistance of LAA Thrombus to Resolution in patients with non-valvular AF is associated with changes in peripheral blood parameters, primarily with morphofunctional platelet characteristics, which correlate with structural remodeling of the atria. The obtained results highlight the need for further research aimed at studying the role of the platelet component of hemostasis in the resistance to LAA thrombus resolution, despite the use of oral anticoagulants.

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