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The left atrial appendage thrombosis of in patients with persistent atrial fibrillation after the novel coronavirus infection

Aim	To evaluate the incidence and features of left atrial appendage (LAA) thrombosis in patients with persis tent atrial fibrillation (AF) after novel coronavirus infection (COVID-19).
Material and methods	Percutaneous echocardiography (pcEchoCG) was performed for 128 patients with persistent AF prepared for cardioversion, 36 (28.1%) of whom had had COVID-19. In 3 (8.3%) patients, the lung lesion area was 50–75%; in 31 (86.1%) patients, 25–50%; in 1 (2.8%) patient, less than 25%. One patient had no lung lesion. Median time from the onset of COVID-19 to the patient enrollment in the study was 76.5 days. At the time of enrollment, the polymerase chain reaction test for SARS-CoV-2 was negative in all patients.
Results	Patients after COVID-19 and those who had not had COVID-19 were comparable by age (62.5±9.2 and 62.4±9.1 years, respectively; p=0.956), gender (men 52.8 and 59.8%, respectively; p=0.471), and risk of stroke (score 2.19±1.28 and score 1.95±1.35, respectively; p=0.350). Duration of the last arrhythmia episode was longer for patients after COVID-19 than for the comparison group (76.5 and 45.0 days, respectively; p=0.011). All patients received oral anticoagulants. 55.6% of COVID-19 patients received rivaroxaban, whereas 62.0% of patients who had not had COVID-19 were treated with apixaban. Median duration of the anticoagulant treatment was longer for COVID-19 patients than for the comparison group (61.5 and 32.0 days; p=0.051). LAA thrombus was detected in 7 (19.4%) patients after COVID-19 and in 6 (6.5%) patients of the comparison group (p=0.030). In COVID-19 patients, the thrombus adhered to LAA wall over the entire thrombus length whereas in patients who had not have COVID-19, the thrombus had a free part that formed a sharp angle with LAA walls. In the presence of LAA thrombus, the LAA blood flow velocity was considerably higher for COVID-19 patients than for the comparison group (31.0±8.9 and 18.8±4.9 cm/sec, respectively; p=0.010). At the follow-up examination performed at 24.0 days on the average, the thrombus was found to be dissolved in 80 and 50% of patients after and without COVID-19, respectively (p=0.343).
Conclusion	In patients with persistent AF after the novel coronavirus infection, LAA thrombosis was detected more frequently than in patients who had never had COVID-19; it was characterized by mural localization and was not associated with a decrease in LAA blood flow velocity.

Keywords	Novel coronavirus infection COVID-19; atrial fibrillation; percutaneous echocardiography; left atrial appendage thrombosis
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Introduction

At the time of writing, according to the Federal Service for Surveillance on Consumer Rights Protection and Human Wellbeing, 5.49 million Russian citizens already had a history of the novel coronavirus disease (COVID-19). Many of those patients also suffer from cardiovascular diseases, including atrial fibrillation (AF). The lack of a correction of anticoagulant therapy (ACT) in AF patients with a history of COVID-19 in the current guidelines [1–3] may be due to a lack of data on the prevalence of atrial thrombosis and the incidence of thromboembolic adverse events in such patients. In this regard, we considered it reasonable to analyze the findings of transesophageal echocardiography (TEE) in patients with persistent AF during the COVID-19 pandemic.

Objective

To estimate the incidence and characteristics of left atrial appendage (LAA) thrombosis in patients with persistent AF and a history of COVID-19.

Material and Methods

This observational study was carried out following the Good Clinical Practice guidelines, in accordance with the Declaration of Helsinki, and approved by the Ethics Committee of Tver State Medical University. All patients signed an informed consent for the scientific use of examination results.

All patients with persistent AF who underwent outpatient TEE prior to being admitted to the Tver State Medical University Hospital for scheduled cardioversion were successively included in the study, beginning with the examination of the first patient with a history of COVID-19 on 09/07/2020. Since then, 128 patients were included in the study, of whom 36 (28.1%) patients had a history of COVID-19.

A history of COVID-19 was established based on medical records, including positive polymerase chain reaction (PCR) test for SARS-CoV-2 and lung computed tomography (CT) results. The volume of pulmonary involvement was 50–75% (CT-3) in 3 (8.3%) patients, 25–50% (CT-2) and up to 25% (CT-1) in 31 (86.1%) and 1 (2.8%) patient, respectively. Another patient had no lung damage and was treated at home.

AF was registered in all 36 patients with COVID-19; in 17 patients (47.2%), for the first time. The remaining 19 patients (52.8%) already had AF episodes requiring cardioversion prior to COVID-19 diagnosis. Low-molecular-weight heparin ACT was administered to all hospitalized patients, while outpatients received long-term therapy with rivaroxaban 20 mg/day for previously diagnosed AF. Following discharge from the infectious disease hospital, 8 (47.1%) patients with newly diagnosed AF and 13 (68.4%) with previously diagnosed AF continued taking oral anticoagulants. Fifteen (41.7%) patients had a 20-136-day break in taking anticoagulants (median 49 days), including ten patients who required continuous ACT (Cha2DS2 VASc score > 1 for male patients and > 2 in female patients). The time from COVID-19 to the inclusion in this study ranged from 35 days to 1 year (median 76.5 days). All patients had a negative PCR test result for SARS-CoV-2 at the time of inclusion.

TEE was performed on a Vivid S70 (GE, USA) fitted with a transesophageal multiplanar phased array transducer (2D/3D/4D) 6VT-D. LAA was scanned via a mid-esophagus access in sections from 0 to 180° at 10–30° intervals. LAA clots were defined as discrete echo-positive masses that differed from the endocardium and pectineal muscles in density.

LAA clots were detected in 13 (10.2%) of 128 patients included in the study. If a clot was found, the hospitalization for the scheduled cardioversion was canceled and a repeat TEE procedure was scheduled for controlling thrombolysis during outpatient ACT. Repeated TEE was conducted in nine patients in whom a LAA clot detected during the first examination. Three patients failed to visit for repeated TEE, while one patient did not undergo TEE due to the scheduled time not coinciding with the time of study results analysis.

Statistical processing of the results was carried out using IBM Statistics v. 22. Normally distributed variables are expressed as the means and the standard deviations (M±SD); non-normally distributed variables are presented as the medians and interquartile ranges (Me [P25; P75] or absolute values and percentages (n (%)). Depending on the type of variable distribution, the intergroup differences were estimated using Student's t-test or the Mann–Whitney U-test. Frequency analysis was performed using a chi-squared test. Differences were statistically significant when p was less than 0.05.

Results

There were no statistically significant differences between patients with persistent AF with and without a history of COVID-19 in age, sex, and the prevalence of comorbidities taken into consideration to assess the risk of stroke (Table 1). There were no statistically significant differences in the mean CHA2DS2 VASc scores. However, it should be noted that coronary artery disease, diabetes mellitus, and chronic heart failure were approximately 2 times more common in patients with a history of COVID-19 than in the comparison group.

In the groups being compared, the duration of the underlying disease was not statistically significantly different. There were no statistically significant differences in the percentages of patients newly diagnosed AF. The mean duration of the previous paroxysmal event was 31 days longer in patients with a history of COVID-19 than in the comparison group. Although atrial flutter was almost 2 times less common in patients with a history of COVID-19, this difference was not statistically significant.

All patients received ACT, typically consisting of direct oral anticoagulants. Apixaban was administered to 62.0% of patients without a history of COVID-19, while rivaroxaban was administered to 55.6% of patients with a history of COVID-19. The mean duration of anticoagulant therapy in patients with a history of COVID-19 was almost twice as long as in the comparison group.

Although LAA flow velocity was almost the same in the groups being compared, LAA thrombosis in patients with a history of COVID-19 was three times more common than in patients without a history of COVID-19.

Table 2 shows the characteristics of patients with a LAA clot detected by TEE. Patients of the two groups were comparable in age (68.3±7.3 years and 62.0±16.1 years, respectively; p=0.373), sex (33.3% and 57.1% of male patients, respectively; p=0.391), and risk of stroke by the CHA2DS2 VASc score (2.83±0.41 and 3.00±1.29, respectively; p=0.707). All patients received ACT, most

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Table 1. Characteristics of patients examined

Parameter	COVID-19 negative (n=92)	COVID-19 positive (n=36)	p
Male	55 (59.8)	19 (52.8)	0.471
Age, years	62.4±9.1	62.5±9.2	0.956
Arterial hypertension	65 (70.7)	23 (63.9)	0.458
CAD	7 (7.6)	7 (19.4)	0.054
Diabetes mellitus	17 (18.5)	11 (30.6)	0.138
CHF	5 (5.4)	5 (13.9)	0.110
History of stroke	5 (5.4)	3 (8.3)	0.543
CHA2DS2 VASc score	1.95±1.35	2.19±1.28	0.350
Duration of AF, months	12.0 [3.0; 48.0]	7.0 [3.0; 60.0]	0.922
New-onset AF	35 (38.0)	17 (47.2)	0.342
Previous episode, days	45.0 [14.0; 94.0]	76.5 [42.5; 130.5]	0.011
Atrial flutter	22 (23.9)	5 (13.9)	0.212
Apixaban 10 mg/day	57 (62.0)	13 (36.1)	0.015
Warfarin 2.5–5 mg/day	8 (8.7)	3 (8.3)	0.776
Dabigatran 300 mg/day	6 (6.5)	0	0.270
Rivaroxaban 20 mg/day	21 (22.8)	20 (55.6)	<0.001
Duration of ACT, days	32.0 [7.0; 126.5]	61.5 [20.0; 121.5]	0.051
LAA velocity, cm/s	33.2±11.0	32.8±10.0	0.843
LAA clot	6 (6.5)	7 (19.4)	0.030

Data are expressed as the mean and the standard deviation (M±SD), the median and the interquartile range (Me [P25; P75], the absolute and relative values (n (%)). CAD – coronary artery disease; CHF – chronic heart failure; CHA2DS2 VASc – score for estimation of the risk of stroke in atrial fibrillation; AF – atrial fibrillation; ACT – anticoagulant therapy; LAA – left atrial appendage.

taking the form of direct oral anticoagulants. By the time of the first TEE, treatment had lasted between 4 and 350 days (median duration 28.0 days and 22.0 days in patients without and with a history of COVID-19, respectively; p=0.836).

TEE showed large intergroup differences. First, in patients with COVID-19, LAA ejection velocity in the presence of a clot was almost twice as high as in the comparison group: 31.0±8.9 cm/s versus 18.8±4.9 cm/s (p=0.010). By comparison, in the absence of a clot, LAA flow velocity was almost the same in patients without a history of COVID-19: 33.2±10.3 cm/s vs 34.0±10.6 cm/s, respectively (p=0.653). Second, the ultrasound picture of LAA thrombosis differed significantly in the groups being compared. In patients without a history of COVID-19, the clot base occupied the LAA apex, while its free part was located in the LAA cavity to form an acute angle with the walls (Figure 1 A, B). Multislice three-dimensional echocardiography showed that the free part of the clot is not adjacent to the LAA walls (Figure 1, B). This ultrasound picture of the LAA clot can be considered typical of patients with AF.

In patients with a history of COVID-19, the clot filled the LAA apex and extended to the LAA base tightly adjacent to its walls (Figure 2). This variant of LAA thrombosis can be determined as parietal or atypical.

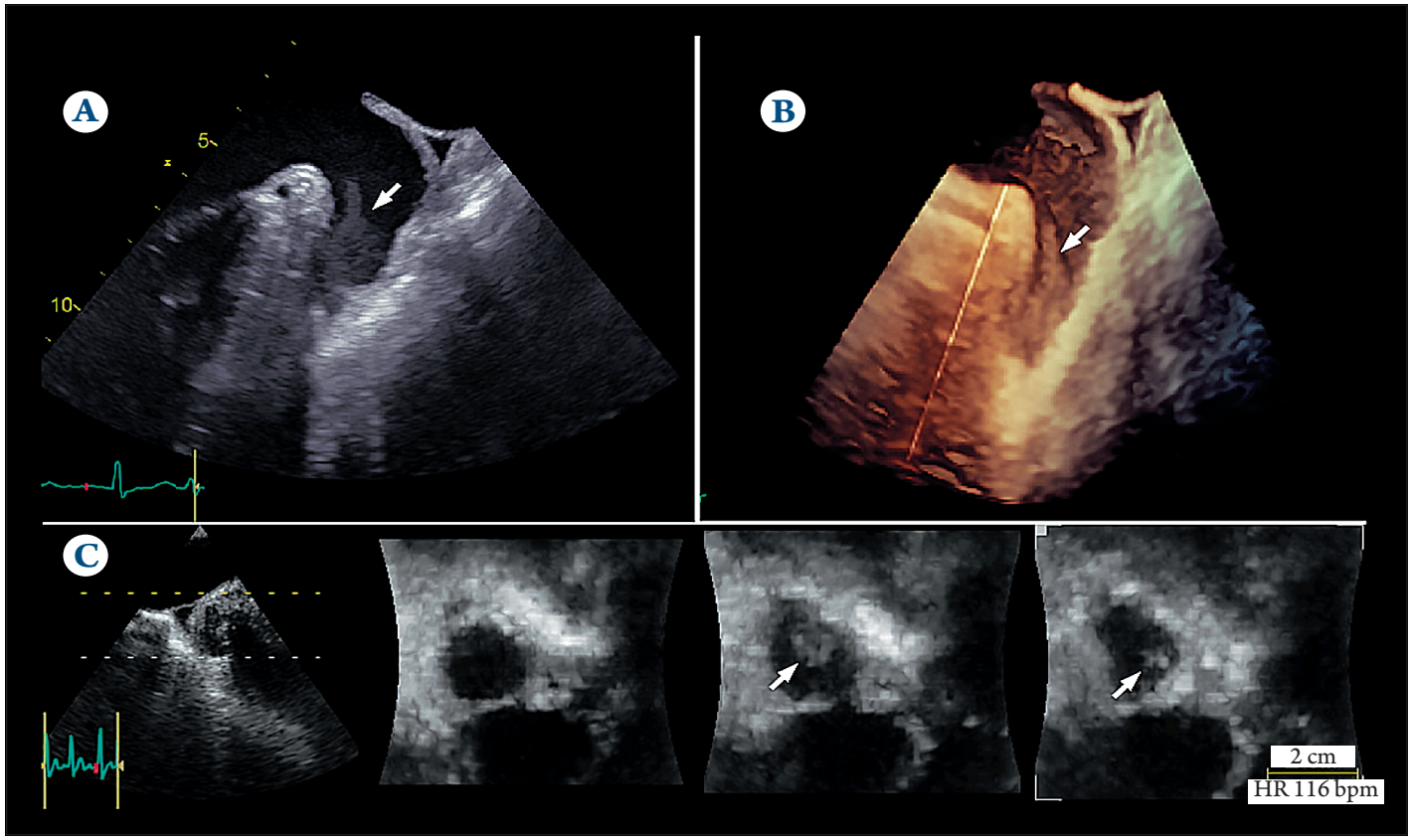
The examination was repeated in 9 patients. ACTS modified or corrected when the clot was detected in 6 of them. The interval between the examinations in patients with and without a history of COVID-19 was 24.0 and 26.0

Table 2. Characteristics of patients with a left atrial appendage clot

No.	COVID-19	Sex	Age	CHA2DS2-VASc, score	First episode	First examination				Second examination			
						ACT	Duration of ACT, days	atypical clot	Velocity, cm/s	ACT	Duration of ACT, days	atypical clot	Velocity, cm/s
1	No	Fem.	62	3	Yes	Apixaban 10 mg	60	No	20	Apixaban 10 mg	30	21	No
2	No	Fem.	63	3	Yes	Apixaban 10 mg	4	No	19	Rivaroxaban 20 mg	183	25	Yes
3	Yes	Male	67	3	Yes	Rivaroxaban 15 mg	36	Yes	38	Rivaroxaban 20 mg	29	40	Yes
4	No	Male	67	3	Yes	Dabigatran 300 mg	36	No	10	Rivaroxaban 20 mg	19	20	Yes
5	Yes	Male	69	4	Yes	Apixaban 10 mg	14	Yes	44	Rivaroxaban 20 mg	22	40	Yes
6	Yes	Male	73	2	Yes	Warfarin 2.5 mg	14	Да	30	Warfarin 3.75 mg	37	50	Yes
7	Yes	Fem.	64	1	Yes	Rivaroxaban 20 mg	22	Да	35	Apixaban 10 mg	24	44	Yes
8	No	Fem.	66	2	No	Apixaban 10 mg	20	Yes	20	Apixaban 10 mg	22	25	No
9	Нет	Male	82	3	No	Apixaban 10 mg	5	No	25	–	–	–	–
10	Yes	Fem.	73	3	No	Apixaban 10 mg	90	Yes	20	–	–	–	–
11	Yes	Fem.	61	5	No	Apixaban 10 mg	6	Yes	30	Apixaban 10 mg	22	20	No
12	Yes	Male	27	3	No	Warfarin 5 mg	105	Yes	20	–	–	–	–
13	No	Fem.	70	3	No	Warfarin 2.5 mg	350	No	19	–	–	–	–

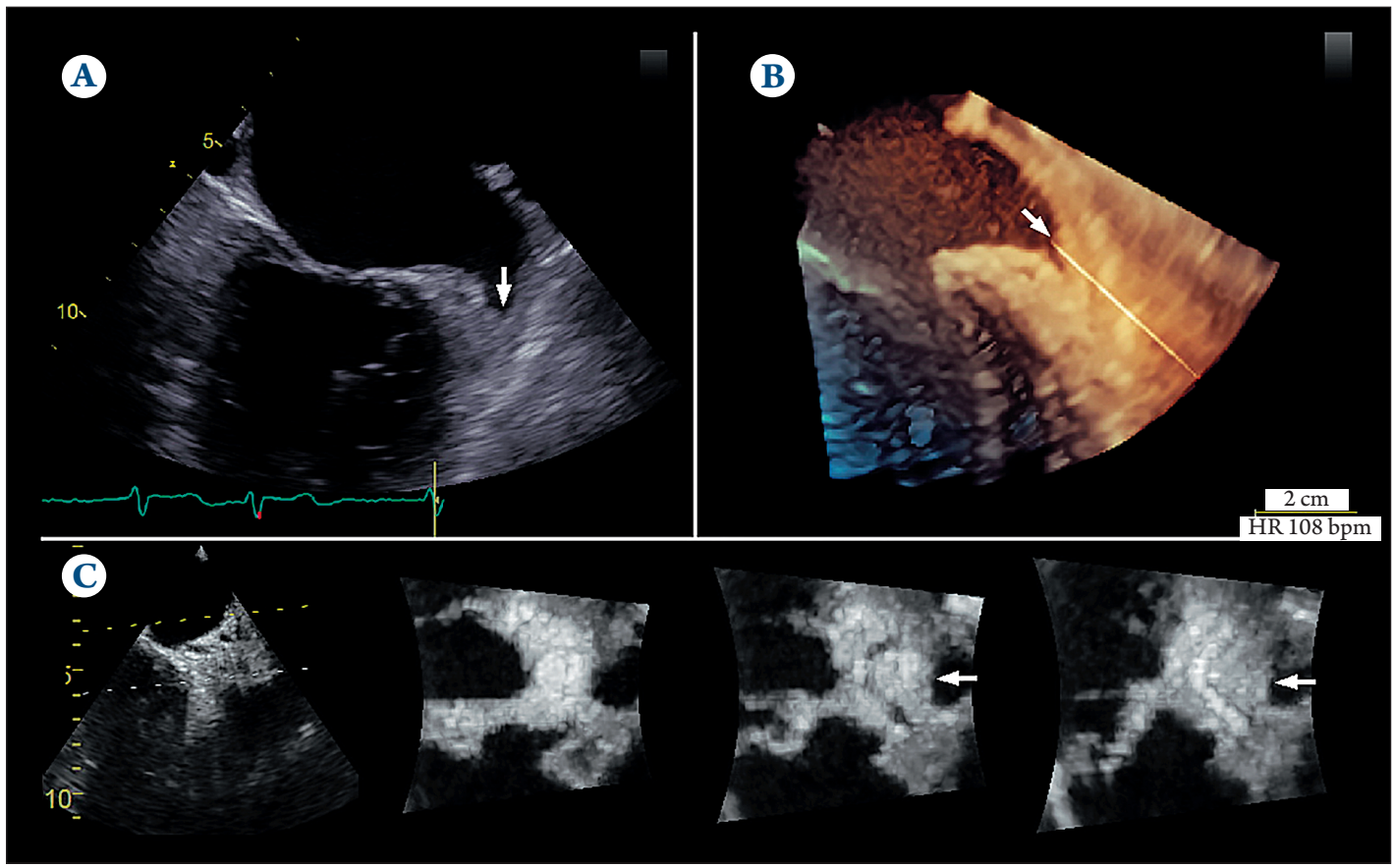
COVID-19 – novel coronavirus disease – CHA2DS2 VASc – score for estimation of the risk of stroke in atrial fibrillation; ACT – anticoagulant therapy (drug and daily dose in milligrams).

Figure 1. Left atrial appendage clot (arrow) in a patient without a history of COVID-19



A – two-dimensional TEE; B – three-dimensional TEE; C – three-dimensional multi-slice TEE.

Figure 2. Left atrial appendage clot (arrow) in a patient with a history of COVID-19



A – two-dimensional TEE; B – three-dimensional TEE; C – three-dimensional multi-slice TEE.

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days, respectively (p=1.000). Thrombolysis was reported in 2 (50.0%) patients without a history of COVID-19 and 4 (80.0%) patients with a history of COVID-19 (p=0.343).

Discussion

Of the 128 patients with persistent AF included in this study, 36 (28.1%) patients had a history of COVID-19, however, the number of patients with a history of COVID-19 was almost 4.1% in our region at the time of writing this article [1]. Given the literature data, such marked differences can be explained by the high incidence of COVID-19 among patients with AF, as well as the high incidence of new cases of arrhythmia during this disease [4].

While the prevalence of AF in the adult population is 2–4% [5], 24.5% of patients who died of COVID-19 had a history of AF [6]. The high prevalence of AF in patients who died of COVID-19 may be because the same factors (old age and somatic diseases) contribute to the development of AF and severe forms of COVID-19 [4, 7]. Here it should also be noted that 85.9% of the patients examined were older than 65 years and/or had somatic comorbidities. At the same time, 34 (94.4%) of 36 patients with a history of COVID-19 had severe or moderate-to-severe disease.

According to the literature, AF is newly diagnosed in 3.6–6.7% of COVID-19 patients generally, but in 10% in severe cases [8]. COVID-19 is not considered as a cause of AF, but as a trigger of arrhythmia in susceptible patients [8, 9]. In our study, AF was first diagnosed in 42.2% of cases; moreover, this type of arrhythmia was observed relatively more often in patients with a history of COVID-19 than in those without a history of COVID-19: 47.2% versus 38.0%, respectively (p=0.342). These differences may reflect the contribution of COVID-19 to the development of arrhythmia.

COVID-19 is associated with severe hemostasis disorders leading to clotting and thromboembolic AEs. Deep vein thrombosis in patients with severe COVID-19 is detected in 17.3–25.0% of cases [10, 11], and small pulmonary artery thromboembolism in 40–81% of cases [12, 13]. Although the incidence of LAA thrombosis in COVID-19 patients is not described in the literature, ischemic stroke in the critical course of the disease is observed in 5.7% of cases [14]. However, all these cases are associated with the acute disease; in the present study, a high incidence of atrial thrombosis was detected in patients with persistent AF and a history of COVID-19 not less than a month earlier. Thus, the question arises: is it possible to correlate the development of LAA thrombosis with the coronavirus infection?

Fan et al. [15] described four cases of arterial thrombosis in young (mean age – 38.5 years) healthy men occurring 78 days on average following asymptomatic coronavirus disease. Having analyzed the results of the examinations and the literature data, the authors concluded that the cause of thrombosis could be persistent endothelial dysfunction caused by its damage during the acute infection.

Blagova et al. [16] detected SARS-CoV-2 RNA in the myocardium in 5 of 6 patients with morphologically verified post-Covid-19 myocarditis (mean age 49.0±9.2 years), whose symptoms appeared on average 5.5±2.4 months after COVID-19. The maximum time following COVID-19 diagnosis to detection of the virus in the myocardium of patients with active myocarditis was 9 months. Endocardial biopsy detected the signs of lymphocytic endocarditis with endocardial thickening and sclerosis in two patients; here, individual biopsies were completely represented by thrombotic masses composed of fibrin and erythrocytes that were also permeated with neutrophils. Biopsy data showed mural thrombosis without endocarditis in another patient.

Our observations indicate that COVID-19 can cause thrombosis not only during the acute phase of the disease, but also after a long period of time after infection. Moreover, endothelial or endocardial damage may be the main factors of clotting in patients with COVID-19. This is also confirmed by the TEE findings.

Reduced LAA ejection velocity associated with atrial systolic dysfunction is the main cause of LAA thrombosis in AF. According to Cresti et al. [17], mean LAA flow velocity in AF is 38±17 cm/s without a clot and 23±13 cm/s in the presence of the clot (p<0.001). Khorkova et al. [18] published similar values of 42.5±5.3 cm/s and 26.4±7.5 cm/s, respectively (p<0.001). In this study, LAA flow velocity in patients without a history of COVID-19 was 18.8±4.9 cm/s and 34.2±10.6 cm/s with and with the clot, respectively (p<0.001). Thus, LAA flow velocity with the clot was on average 15 cm/s lower than without the clot in all three cases. However, LAA flow velocity was nearly similar in AF patients with a history of COVID-19 in the absence and in the presence of the clot (33.2±10.3 cm/s and 31.0±8.9 cm/s, respectively; p=0.573), which is why low LAA flow velocity is not the main reason for clotting in this category of patients.

The second characteristic of LAA clotting in patients with a history of COVID-19 is its parietal nature (see Figure 2), which is characteristic of clotting caused by vascular wall or endocardial damage [19, 20]. According to Blagova et al. [16], patients with post-COVID-19 myocarditis had parietal thrombosis. Two patients with a history of COVID-19 described by Ueda et al. [15] had parietal aortic thrombosis.

Thus, a comparison of the literature data and our findings suggests that the persistent presence of SARS-CoV-2 in the myocardium and/or LAA endocardium may be the cause of LAA thrombosis in AF patients with a history of COVID-19. This may help to explain the high incidence

of LAA thrombosis in patients with AF and a history of COVID-19. However, the possible influence of other factors affecting the likelihood of atrial thrombosis on the incidence of thrombosis in the comparison groups cannot be excluded. Coronary artery disease, heart failure, and diabetes mellitus were more common in the examined AF patients with a history of COVID-19; here, the mean duration of previous episodes of arrhythmia was longer. Atrial flutter with higher LAA flow velocity and less common LAA thrombosis than in AF was less frequent [17]. However, these factors cannot explain the echocardiographic features of LAA thrombosis identified in our patients with persistent AF and a history of COVID-19.

Given the epidemiological situation, it is expected that more patients with a history of COVID-19 will present with AF, thus justifying the further study the incidence and causes of LAA thrombosis in this category of patients.

Limitations

The small number of cases of LAA thrombosis in patients with persistent AF and a history of COVID-19 does not allow our findings to be extrapolated to the entire patient population. Thus, it is necessary to either confirm or refute the results of this study with the findings of other independent studies.

Conclusion

Left atrial appendage thrombosis is more common in patients with persistent atrial fibrillation and a history of COVID-19 than in patients who did not have COVID-19. It has a parietal nature and is not associated with reduced flow velocity in the left atrial appendage.

No conflict of interest is reported.

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