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Cardiohemodynamic Changes and Cardiac Arrhythmias After Coronavirus Infection

Aim	To study changes in cardiohemodynamic alterations of the myocardium and heart rhythm disorders at 3 and 6 months following the coronavirus infection.
Material and Methods	EchoCG, ECG Holter monitoring, and Doppler ultrasonography of hepatolienal blood vessels were performed for 77 patients (mean age, 35.9 years) at 3 and 6 months after coronavirus infection. The patients were divided into the following groups: group 1, with injury of the upper respiratory tract; group 2, with bilateral pneumonia (CT1, 2), and group 3, with severe pneumonia (CT3, 4). Statistical analysis was performed with a SPSS Statistics Version 25.0 software package.
Results	At 6 months after the disease onset, the patients noted an improvement of their general condition. In patients with moderate pneumonia, early peak diastolic velocity (p=0.09), right ventricular isovolumic diastolic time (p=0.09), and pulmonary artery systolic pressure (p=0.005) where decreased, while tricuspid annular peak systolic velocity was, in contrast, increased (p=0.042). Both segmental systolic velocity of the LV mid-inferior segment (p=0.006) and the mitral annular Em/Am ratio were decreased. In patients with severe disease at 6 months, right atrial indexed volume was reduced (p=0.036), tricuspid annular Em/Am was decreased (p=0.046), portal and splenic vein flow velocities were decreased, and inferior vena cava diameter was reduced. Late diastolic transmitral flow velocity was increased (p=0.027), and LV basal inferolateral segmental systolic velocity was decreased (p=0.046). In all groups, the number of patients with heart rhythm disorders was decreased, and parasympathetic autonomic influences prevailed.
Conclusion	At 6 months after coronavirus infection, practically all patients noted improvement of their general condition; incidence rate of arrhythmia and cases of pericardial effusion were decreased; and autonomic nervous system activity recovered. In patients with moderate and severe disease, morpho-functional parameters of the right heart and the hepatolienal blood flow were normalized, however, occult disorders of LV diastolic function remained, and LV segmental systolic velocity was reduced.

Keywords	Heart; hemodynamics; heart rhythm disorders; COVID-19
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Introduction

World seized the COVID-19 pandemic, which resulted in high morbidity and mortality: the total number of cases exceeded 254.2 million patients, more than 5.5 million people died. In Russia, more than 8.9 million cases of the infection were confirmed, 253 thousand people died [1–3]. The receptors of SARS-CoV-19, which causes coronavirus pneumonia, were found in the gastrointestinal epithelium, liver, kidneys, vascular endothelium, and myocardium with serious cardiac involvement [2–5]. The virus directly affects the myocardium, and pro-inflammatory cytokines directly affect cardiomyocytes, causing damage, remodeling, and decreased cardiac performance [5, 6]. Moreover, the vascular endothelium is damaged, which causes impaired microcirculation and the development of thrombosis [5, 6]. Interleukin-1 is involved in the destabilization of atherosclerotic plaques, which leads to myocardial infarction and pulmonary embolism in those affected [6]. The adverse effect of hypoxia on the myocardium is significant [6]. Aggressive COVID-19 treatments may also cause damage to the myocardium [1, 7].

M.R. Dweck et al. [8] reported the echocardiographic findings obtained in a large sample of patients with COVID-19 (from 69 countries) and showed that 50% of the examined patients had dysfunction of both ventricles. Every seventh patient had serious myocardial injury (myocardial infarction, myocarditis, or takotsubo cardiomyopathy) [8]. In another study,

autopsy of COVID-19 patients showed traces of the virus in the heart of more than 60% of the deceased; 16 patients had clinically significant viral load in the tissues at the time of death [9].

It is well known that this virus also has long-term sequelae with the development of post-COVID-19 syndrome [2]. Cardiovascular involvement was revealed in the German cohort of patients a mean of 71 days after diagnosing COVID-19 [10]. Magnetic resonance imaging (MRI) showed that 78% of patients had higher left ventricular volume, weight, and ejection fraction (LVEF) compared to the control group; active lymphocytic inflammation was detected by myocardial biopsy in patients with the most severe MRI changes [10]. The presence of hyperechoic inclusions buried in the myocardium, diastolic LV dysfunction, and a small amount of free fluid in the pericardium were discovered by echocardiography performed in patients 1–7 months after COVID – 19 (the mean patient age was 53 years) [2].

Thus, the cardiovascular involvement is diagnosed relatively often in patients with COVID-19 during hospital treatment [2–10], but there are only few studies on long-term cardiovascular sequelae.

Objective

Study cardiac and hemodynamic changes, frequency and nature of cardiac arrhythmias 3 and 6 months after COVID-19.

Material and methods

The study included 77 patients after COVID-19 treatment (mean age 35.9 years). The following examinations were conducted 3 and 6 months (a median of 98 and 189 days, respectively) after diagnosis: Echocardiogram and Doppler ultrasound of hepatolienal blood flow, Holter monitoring. The viral origin of the lesion was confirmed by polymerase chain reaction for SARS-CoV-2 RNA. All patients with a history of COVID-19 were apparently healthy before the disease and denied chronic and cardiovascular diseases.

Patients were divided into three groups: Group 1 (n=31) patients with upper respiratory tract infections, without complications, treated outside of hospital,without lung involvement according to computed tomography (CT); Group 2 (n=27) patients with bilateral, multisegmental, viral bacterial pneumonia CT1 (up to 25% involvement, n=16) and CT2 (25–50%; n=11); Group 3 (n=19) with severe CT3 (n=11) and CT4 (n=8) lung involvement. The mean age of patients was 35.5 [23; 46], 36 [27; 43.5] years, and 36.9 [35.2; 48] years, respectively, in Group 1, Group^2, and Group 3.

Umifenovir (Arbidol) was the most commonly used antiviral agent in all groups (62.3%). Vitamin C and diazoline were also administered in Group 1. In Group 2 and Group 3, patients received macrolide antibiotics, third-generation cephalosporins (oral/intravenous), anticoagulants, and expectorants. Detoxification agents were also administered in Group 3. Hydroxychloroquine regiment was additionally administrated to 17 (58%) patients in Group 2 and all patients in Group 3 (earlier version of the clinical guideline was in effect).

The control group comprised 22 healthy volunteers of the corresponding age and without signs of cardiac diseases. The study did not include individuals older than 48 years, patients with cardiac diseases and severe comorbidities.

Echocardiography and Doppler scanning were conducted following the standard procedure on a VIVID E95 device. Regional longitudinal strain and LV strain rate were measured by non-Doppler two-dimensional grey scale imaging. The examination was conducted from the apical long axis view, LV myocardium was studied with optimal visualization of all segments, at the rate of 50 to 80 fpm, and stable ECG registration. The endocardium was clearly traced, the epicardial surface was traced automatically. The program automatically calculated the displacement of the spot pattern from frame to frame, within the area of interest, throughout the cardiac cycle. After the optimization of the area of interest, strain curves were generated by the software for each segment.

Blood flow velocity in the portal, splenic and inferior vena cava veins, the probe was held perpendicular to the costal arch and moved from the metasternum to the portal fissure image and in the opposite direction. The splenic vein was also located in the area of the splenic hilum.

Holter monitoring was performed using an Astrocard complex for 23.0 [22.3; 23.9] hours. The nature and frequency of cardiac arrhythmias and cardiac rhythm variability parameters were evaluated. The protocol was approved by the ethics committee of Chita State Academy of Medicine. All patients signed the informed consent before being included in the study.

There are no potential restrictions of the study. The statistical analysis was based on the International Committee of Medical Journal Editors (ICMJE) and the Statistical Analysis and Methods in the Published Literature (SAMPL) guidelines [11].

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The normality of the sign distribution, given that the study groups comprised less than 50 patients, was analyzed using the Shapiro-Wilk test. Given the non-normal distribution of signs, the data obtained are presented as the medians and the first and third quartiles (Me [Q1; Q3]) in all study groups. Two dependent groups was compared using the Wilcoxon test. Given a sample size of more than 25 elements, the Wilcoxon test was converted to a Z-score. Differences were statistically significant in all cases of p value being less than 0.05. The data obtained were statistically processed using IBM SPSS Statistics 25.0 [12].

Results

The vast majority of patients with mild, moderate, and severe COVID-19 complained of fatigue, decreased performance, and sleep disturbance when they were examined 3 months (98 [92; 103] days after the diagnosis of COVID-19). In 6 months (189 [174; 207] days from the onset of the disease), those complaints persisted in 39% of patients in Group 1, 43% in Group 2, and 42% in Group 3 (Table 1). Complaints of heart pains, mixed dyspnea, palpitations, intermissions were less common 6 months after COVID-19 (Table 1). Blood pressure normalized in 6 months only in one patient in Group 1, all other patients continued their antihypertensive treatment.

According to echocardiography performed 6 months after the disease, free fluid in the pericardium, mainly along the posterior wall of the LV (a mean of 3.5–5 mm) and leaf compaction were less frequent in all groups (see Table 1). According to the literature, mild hydropericardium is detected by echocardiography in patients with a history of COVID-19 [2].

There were no significant differences in Group 1 between the echocardiographic parameters of interest 3 and 6 months after diagnosis. Early peak diastolic tricuspid velocity E and right ventricular isovolumic relaxation time decreased over time by 13% and 14%, respectively, as well as peak tricuspid regurgitation pressure gradient and LA systolic pressure (9%), in patients with moderate COVID-19. Peak systolic tricuspid annular velocity, which characterizes RV systolic function, on the contrary, increased by 7%. The study of LV myocardial indicators showed a trend to a decrease in the segmental systolic velocity of the mid-inferior septal segment and a decrease in the basal LV segmental velocity by 15%. Moreover, a medial mitral annular Em/Am ratio decreased to 0.92 (Table 2).

Thus, parameters of the RV diastolic and systolic functions improved and pulmonary artery systolic pressure (PASP) decreased in patients with a history of moderate COVID-19 in 3 and 6 months. Segmental systolic velocity of the mid-basal LV segment and silent LV diastolic dysfunction were also observed.

The analysis of the echocardiographic parameters in patients with a history of severe COVID-19 showed a decrease in the right atrial volume index and an increase in the annular tricuspid Em/Am ratio by 25% in 3 and 6 months (Table 3). The portal and splenic flow velocities decreased, the inferior vena cava diameter decreased, which probably indicates a decrease in pulmonary circulation load. The assessment of the LV diastolic function showed a 18% increase in the late peak diastolic transmitral velocity, possibly due to a decrease in the LV sucking effect and an increase in the left atrial contribution to ventricular filling. Moreover, there was a 13% decrease in the segmental systolic velocity of the LV basal inferolateral segment.

Thus, diastolic function worsened and segmental velocity basal inferiolateral LV segment decreased 3 and 6 months after COVID-19 in Group 3.

Holter monitoring showed that fewer patients with cardiac arrhythmias decreased in all groups 3 and 6 months after COVID-19. Cardiac arrhythmias, such as single supraventricular extrasystoles, preserved in 1 (12.5%) patient in Group 1, 2 (16.6%) patients

Table 1. Main complaints and examination results 3 and 6 months after COVID-19, n (%)

Parameter	Group 1 (n=31)		Group 2 (n=27)		Group 3 (n=19)	
	Month 3, n (%)	Month 6, n (%)	Month 3, n (%)	Month 6, n (%)	Month 3, n (%)	Month 6, n (%)
Asthenia	26 (83.8)	12 (39)	24 (89)	18 (67)	18 (95)	11 (58)
Heart pains	4 (13)	1 (3.2)	9 (33.3)	7 (26)	11 (57)	9 (47.3)
Mixed dyspnea	7 (22.5)	2 (6.4)	14 (51.8)	9 (33)	12 (63)	10 (53)
Palpitations, intermissions	3 (9.6)	1 (3.2)	10 (37)	4 (14.8)	15 (78)	8 (42)
Arterial hypertension	4 (12.9)	3 (9.6)	4 (14.8)	4 (14.8)	9 (47)	9 (47)
Mild pericardial effusion	4 (12.9)	2 (6.4)	7 (25.9)	4 (14.8)	5 (26.3)	3 (15.7)

Table 2. Ultrasound and Holter monitoring findings 3 and 6 months after moderate COVID-19

Parameter	Month 3 (n=27)	Month 6 (n=27)	Z-score test statistics; p
ETV, cm/s	0.58 [0.54; 1.36]	0.51 [0.41; 0.55]	Z= –2.23; p=0.09
TRPG, mm Hg	27.1 [23.5; 31.2]	26.5 [24.2; 29.5]	Z= –2.2; p=0.0028
SmTV, cm/s	0.14 [0.13; 0.18]	0.15 [0.14; 0.15]	Z= –2.03; p=0.042
RC IVRT, ms	74 [72.4; 88.3]	64 [58.5; 69.2]	Z= –2.62; p=0.09
Em/AmMV	1.34 [1.05; 1.49]	0.92 [0.9; 1.14]	Z= –2.23; p=0.026
PASP, mm Hg	29.5 [27; 35.5]	27 [26.5; 27.55]	Z= –2.84; p=0.005
LV segment 9	–22.0 [–21.2; –23.2]	–19.0 [–15.2; –19.6]	Z= –2.5; p=0.012
LV segment 10	–20.0 [–19.4; –22.6]	–17.0 [–16.7; –17.7]	Z= –2.74; p=0.006
PNN50, %	9.05 [4.72; 11.7]	16.4 [16; 17.4]	Z= –2.63; p=0.007

ETV, early diastolic tricuspid velocity; TRPG, peak tricuspid regurgitation pressure gradient; SmTV, tricuspid annular peak systolic velocity; RC IVRT, right ventricular isovolumic relaxation time; Em/AmMV, ratio of diastolic medial mitral annular velocities; PASP, pulmonary artery systolic pressure; LV segment 9, segmental systolic velocity of the left ventricular mid-inferior septal segment; LV segment 10, segmental systolic velocity of the left ventricular mid-inferior segment; PNN50, Holter monitoring parameter characterizing the predominance of the parasympathetic component of the regulation.

Table 3. Ultrasound and Holter monitoring findings 3 and 6 months after severe COVID-19

Parameter	Month 3 (n=19)	Month 6 (n=19)	Z-score test statistics; p
RAVI, mL/m^2	26.5 [21.2; 27.7]	25.1 [23.4; 25.5]	Z= –2.09; p=0.036
Em/AmTV	0.9 [0.8; 1.5]	1.2 [1.1; 2.6]	Z= –2.0; p=0.046
AMV, cm/s	0.58 [0.52; 0.61]	0.7 [0.63; 0.77]	Z= –2.21; p=0.027
IVCD, mm	21 [19.5; 24.1]	15 [13; 17.2]	Z= –2.21; p=0.027
Segment 5	–15.0 [–14.5; –19.2]	–13.0 [–11.9; –13.4]	Z= –2.0; p=0.046
VSV, cm/s	13 [10; 15.5]	12 [11.5; 17.5]	Z= –2.2; p=0.028
VPV, cm/s	22 [19.5; 22.3]	17 [16.9; 20.6]	Z= –2.0; p=0.045
PNN50, %	10.4 [4.2; 18.2]	14.9 [11.6; 18.1]	Z= –2.7; p=0.007

RAVI, right atrial volume index; Em/AmTV, ratio of diastolic lateral tricuspid annular velocities; AMV, late diastolic mitral velocity; IVCD, inferior vena cava diameter; Segment 5, segmental systolic velocity of the left ventricular basal inferior lateral segment; VSV=peak systolic splenic vein velocity; VPV, peak portal vein velocity; PNN50, Holter monitoring parameter characterizing the predominance of the parasympathetic component of the regulation.

in Group 2, and 4 (21%) patients in Group 3. Single ventricular extrasystoles were detected in only 3 (11%) patients in Group 2 and 2 (10.5%) patients in Group 3. One patient with a history of severe COVID-19 still had persistent atrial fibrillation first onset during the disease. QT prolongation was established in only 2 patients in Group 3. Holter monitoring parameter PNN50, which reflects the predominance of parasympathetic component of the nervous system regulation, increased in Group 1, Group 2, and Group 3 by 6%, 45%, and 31%, respectively (see Table 2 and Table 3).

Discussion

Thus, patients with a history of COVID-19 were less likely experience heart pains, mixed dyspnea, palpitations, intermissions, and asthenia over time (in 3 and 6 months). According to Holter monitoring, there were fewer patients with cardiac arrhythmias in all groups. There were fewer patients with mild pericardial effusion in all groups. There were no significant differences between the echocardiographic parameters of interest in patients with a history of mild COVID-193 and 6 months after the disease. The RV diastolic and systolic functions improved and PASP decreased in patients with a history of moderate COVID-19. Segmental systolic velocity of the mid-basal LV segment and silent LV diastolic dysfunction were also observed.

In 6 months, patients with a history of severe COVID-19 had lower RV volume index, inferior vena cava diameter; portal vein velocity decreased and splenic vein velocity increased as well as the ratio of the tricuspid annular velocities. The assessment of the LV parameters showed silent diastolic dysfunction and reduced segmental systolic velocity of the basal inferiolateral segment.

Structural and functional impairment and cardiac arrhythmias in patients with a history of COVID-19 may be directly caused by the effect of

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В исследованиях первичной профилактики доза аторвастатина составляла 10 мг в сутки. Может понадобиться повышение дозы с целью достижения значений ХС-ЛПНП, соответствующих современным рекомендациям. Применение у детей с 10 до 18 лет при гетерозиготной семейной гиперхолестеринемии: рекомендуемая начальная доза – 10 мг 1 раз в сутки. Доза может быть увеличена до 80 мг в сутки в зависимости от клинического эффекта и переносимости. Дозу препарата необходимо титровать в зависимости от цели гиполипидемической терапии. Коррекция дозы должна проводиться с интервалами 1 раз в 4 недели или больше. У пациентов с недостаточностью функции печени дозу необходимо снижать, при регулярном контроле активности «печеночных» трансаминаз: аспартатаминотрансферазы (АСТ) и аланинаминотрансферазы (АЛТ); у пациентов с недостаточностью функции почек и у пожилых пациентов коррекции дозы не требуется. Побочное действие: Липримар® обычно хорошо переносится; побочные реакции, как правило, легкие и преходящие: головная боль, боль в горле, носовое кровотечение, запор, метеоризм, диспепсия, тошнота, диарея, миалгия, артралгии, боль в конечностях, судороги мышц, припухлость суставов, боль в спине, мышечно-скелетные боли, отклонение от нормы результатов «печеночных» тестов (АСТ и АЛТ), повышение активности сывороточной креатинфосфокиназы (КФК), аллергические реакции, гипергликемия, назофарингит. Форма выпуска: таблетки, покрытые пленочной оболочкой, 10 мг, 20 мг, 40 мг и 80 мг. 2,4,5 или 8 блистеров по 7 таблеток; 3,5 или 10 блистеров по 10 таблеток в картонную пачку с контролем первого вскрытия (только для производства «Пфайзер Фармасьютикалз ЭлЭлСи») вместе с инструкцией по применению. Срок годности: 3 года. Условия отпуска: по рецепту. Перед назначением препарата ознакомьтесь с полной инструкцией по медицинскому применению препарата Липримар® П N014014/01.

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viruses on cardiomyocytes, endothelial dysfunction, with aggravates tissue swelling and contributes to the development of a prethrombotic state with the possible development of microthrombosis including in the myocardium [2, 5, 6]. Existing hypercoagulation, complement activity, and the effects of pro-inflammatory cytokines on cardiomyocytes can contribute to the development of systemic inflammation with metabolic disturbance and inotropic myocardial function with gradual remodeling of the heart cavities [2, 5, 6, 10]. The effect of hypoxia on the myocardium, which further disturbs the condition of intracardiac hemodynamics, is also very significant [5, 6]. Moreover, lung tissue damage, when a large number of alveoli are shut down from the gas exchange, pulmonary hemodynamics is disturbed, pulmonary artery pressure increases, and RV load increases and its function is impaired. Patients with severe COVID-19 have remodeling of the right heart and veins of the hepatolienal system due to extensive lung tissue damage [5, 6]. LV dysfunction may be caused by the interventricular interaction as a result of a sharp increase in the RV load. The side effect of drugs used to treat COVID-19 on the myocardium, such as hydroxychloroquine, antiviral drugs, antibiotics that cause the development of ventricular arrhythmias, is not excluded [1, 7]. In 6 months, patients reported general well-being mend, fewer cardiac arrhythmias and cases of pericardial effusion were registered, and the activity of the autonomic nervous system recovered. Normalization of morphological and functional parameters of the right heart and hepatolienal blood flow was faster than that of the LV function, possibly due to the restoration of lung tissue, the pulmonary vascular bed, and a decrease in the pulmonary circulation pressure. Meanwhile, silent LV diastolic dysfunction persisted and LV segmental systolic velocity decreased. Given our findings and the literature data, we can assume that COVID-19 can cause low-level chronic systemic inflammation with vascular involvement, impaired microcirculation, and thrombosis, in some patients, even those with the asymptomatic disease [2, 8, 9]. In some patients, a pronounced immune response may result in fulminant myocarditis, heart failure, and cardiogenic shock [2–7].

Conclusion

Thus, it is necessary to conduct additional research on the long-term cardiovascular sequelae in all patients with a history of COVID-19 in order to earlier detect myocardial involvement and consequently prevent heart failure.

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