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The Effect of COVID-19 on Long-Term Cardiac Function in Patients With Chronic Heart Failure

Aim	To evaluate functional changes in the heart in the long-term following COVID-19 in patients with chronic heart failure (CHF).
Material and Methods	Case reports of 54 patients aged 69.1±9.7 years who had COVID-19 from January 2021 through January 2022 and had been previously diagnosed with NYHA functional class II–III CHF were studied. Two comparison groups were isolated: HF with LV EF >50% (n=39) and <50% (n=15). Echocardiography was used to evaluate changes in LV EF and pulmonary artery systolic pressure (PASP) 5–6 months following COVID-19.
Results	In all CHF patients after COVID-19 at 5.8 months on average, LV EF decreased (median difference, 2.5%; 95% confidence interval (CI): 6.99×10–5–4.99) and PASP increased (median difference, 8 mm Hg; 95% CI: 4.5–12.9). In the HF group with LV EF <50%, the decrease in EF was greater than in the group with LV EF >50% (6.9 and 0.7%, respectively; p=0.037); furthermore, the CHF phenotype did not influence the change in PASP (p=0.4). The one-factor regression analysis showed that the dynamics of LV EF decrease was significantly influenced by the baseline decrease in LV EF, whereas the change in PASP was influenced by the dynamics of LV EF decrease, presence of dyslipidemia, and statin treatment. Furthermore, the multifactorial analysis showed that prognostically significant factors for long-term changes in LV EF following COVID-19 were male gender (odds ratio (OR), 5.92; 95% CI: 1.31–26.75; p=0.014), LV EF at baseline <50% (OR, 0.88; 95% CI: 0.8–0.96; p<0.001); changes in PASP depended on the presence of dyslipidemia (OR, 0.08; 95% CI: 0.01–0.84; p=0.018).
Conclusion	This study showed that COVID-19 in the long term can influence the course of CHF; in this process, HF patients with EF <50% have progression of systolic dysfunction and PASP, whereas patients with EF >50% have an isolated increase in PASP.

Keywords	SARS-CoV-2; COVID-19; chronic heart failure; echocardiography; left ventricular ejection fraction; pulmonary artery systolic pressure; pulmonary hypertension
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Introduction

The World Health Organization (WHO) declared the outbreak of the disease caused by the SARS-CoV-2 coronavirus (COVID-19) a pandemic on March 11, 2020 [1]. Since that time and to the present day, more than 6.3 million people have died of COVID-19 worldwide, and more than 373 thousand people died in Russia. These figures continue to increase [2]. In more than 25% of cases, death is caused by the complications of cardiovascular diseases (CVDs): acute coronary syndrome (ACS), decompensated chronic heart failure (CHF), pulmonary embolism, cardiac arrest, stroke [3, 4]. It is worth noting that cardiovascular complications remain one of the main causes of high mortality of cardiac patients during COVID-19, in particular, due to the development of acute infectious myocarditis, myocardial infarction, coronary artery vasculitis, life-threatening arrhythmias [5, 6]. These pathologies are possible during interstitial pneumonia, acute respiratory distress syndrome, cytokine storm, and/or septic shock, which are the stigmas of severe COVID-19. However, not all patients, including the cardiac profile, suffered the severe form of the disease. Moreover, most cases were mild and accompanied by fever, dry cough, asthenia, muscle ache, and sometimes anosmia

[5, 7]. Patients with CVDs with such complaints may not have sought medical attention for COVID-19.

However, the long-term follow-up of patients with a history of COVID-19 showed that they continued to have cardiovascular complications after recovery as post-COVID-19 syndrome, which is as a reason for hospitalization. These complications are caused by the lesion of cardiomyocytes, lung tissue, and vascular endothelium during the acute period, which lead to the formation of areas of myocardial fibrosis and autonomous dysfunction, as well as the damage of interstitial tissue and lung parenchyma [8]. Against this background, transient and later, as pulmonary fibrosis develops, permanent pulmonary hypertension (PH) is formed, which, in combination with chronic myocardial damage, significantly increases the risk of death in patients with CVDs. Concomitant lung diseases can also lead to the gradual formation of PH, and COVID-19 will only aggravate it through the mechanisms described above. The combined effect of infectious and non-infectious factors result in a gradual increase of the postload, and the right heart dysfunction and dilatation develop over time, which leads to the onset or progression of CHF [9–13].

Several studies on changes in the echocardiographic picture during COVID-19 were published, but they related mainly to the acute period of COVID-19 [14] and included patients with moderate and severe course of the disease and more than 50% damage of lung tissue according to the CT findings. The development of systolic LV dysfunction caused by ACS, small-vessel vasculitis, and myocarditis has been described [15]. There are even fewer works on dysfunction of the right heart caused by the lesion of the interstitial tissue and parenchyma, pulmonary vessel thrombosis and thromboembolism. Finally, single articles were published that assessed the condition of the myocardium of patients with a history of mild COVID-19 in the long-term period [16].

Given the above, it was decided to conduct a retrospective comparative analysis of available echocardiographic parameters in patients with CHF and a history of COVID-19 and assess the contribution of various factors to the development of functional changes in the heart in the long-term follow-up period.

Objective

Determine functional changes in the heart and the effect of COVID-19 on such changes in the long-term period after the infection in patients with CHF.

Material and Methods

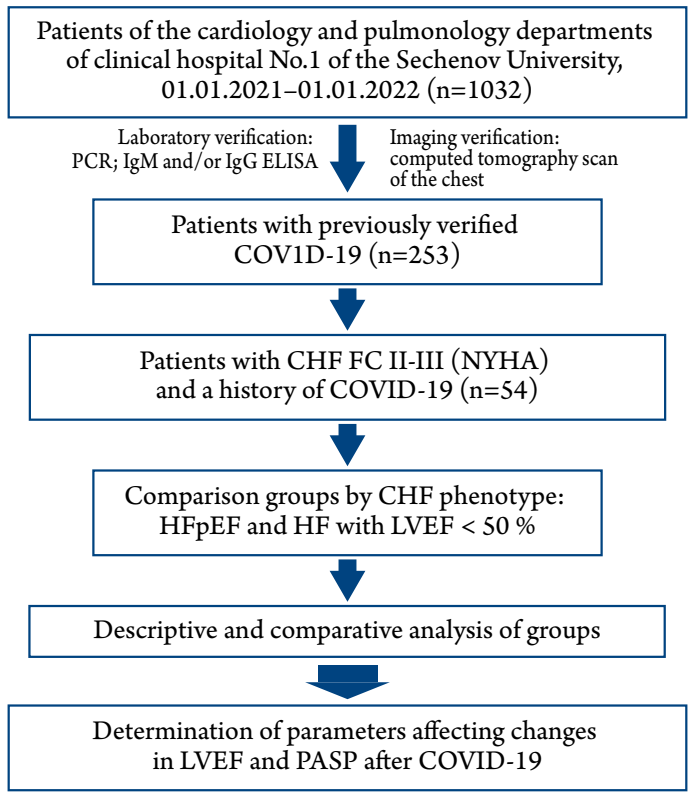
A retrospective cohort study was conducted (Figure 1).

To perform the study, 1032 hospital charts of patients of the cardiology and pulmonology department of clinical hospital No. 1 of Sechenov University from 01.2021 to 01.2022. Among them, 253 patients of ≥ 25 years old with a history of COVID-19 (confirmed by polymerase chain reaction and/or enzyme immunoassay for anti-SARS-CoV-2 antibodies (IgM and IgG) in any form (from asymptomatic to severe course with more than 75% lung lesion according to CT) were selected.

54 patients with CHF functional class (FC) II–III according to the NYHA classification diagnosed before COVID-19 were selected. Patients are distributed according to the heart failure phenotype taking into account left ventricular ejection fraction (LVEF) after COVID-19: heart failure with preserved ejection fraction (HFpEF; n=39), heart failure with reduced ejection fraction (HFrEF), and heart failure with mid-range ejection fraction (HFmrEF). Due to the common treatment approaches, patients with HFrEF and HFmrEF were combined in a single group of HF with LVEF < 50% (n = 15).

Echocardiograms from the electronic hospital charts of the subjects were used to assess functional changes in the heart. Echocardiography was conducted using a commercial-type fixed equipment (Aplio 500) during the scheduled hospital treatment of patients before and after COVID-19 (in a mean of 5.8 months and not associated with the acute period of the disease).

Figure 1. Cohort study flow chart



HFpEF, heart failure with preserved ejection fraction; PASP, pulmonary artery systolic pressure.

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The following echocardiographic parameters available in the hospital charts were evaluated: LVEF, pulmonary artery systolic pressure (PASP).

Changes in the functional parameters of the heart were selected as the endpoints of the study: LVEF as a key marker of the heart's pumping function and PASP, which reflects the degree of PH in the long-term period after COVID-19.

The data analysis was performed using the R 3.6.3 software environment and language. The normality of distribution was evaluated using the Shapiro-Wilk test. Normally distributed quantitative indicators were presented as the mean values and standard deviations (M±SD), and non-normally distributed quantitative indicators were expressed as the medians and interquartile ranges (Me [Q1; Q3]). The percentages and absolute numbers were determined for the categorical and qualitative variables. LVEF and PASP before and after COVID-19 were compared by calculating the one-tailed Wilcoxon test for paired variables. The comparative analysis for normally distributed quantitative variables was performed using the t-test in the normal distribution and the Mann-Whitney U-test in the non-normal distribution. Regression analysis (univariate and multivariate linear regressions) was used to assess the effects of factors on outcomes. Significant factors were included in a single multivariate regression equation. The level of statistical significance was 0.05 in all cases. The multiplicity adjustment was not used, since the analysis was descriptive.

Results

Characteristics of the subjects with CHF and a history of COVID-19 and the degree of lung lesion according to CT during the acute period of the disease are presented in Table 1.

The structure of diseases of the subjects with CHF is provided in Table 2.

Table 3 contains the numerical values of LVEF and PASP of patients with CHF.

The Wilcoxon test showed a statistically significant decrease in LVEF across the study population with a median difference of 2.5% (95% confidence interval (CI) 6.99×10–5–4.99). PASP increased statistically significantly in the population of interest after COVID-19. The median difference was 8 mm Hg (95% CI 4.5–12.99).

The comparison of changes in LVEF depending on the CHF phenotype showed a more pronounced post-COVID-19 decrease in the group of HF with LVEF < 50% (6.9% versus 0.7% in the HFpEF group; p = 0.037). CHF phenotype was not considered in the assessment of changes in PASP (p = 0.4).

Further, we conducted sequential univariate and multivariate regression analyses of these endpoints in order to determine the factors that can affect changes in LVEF and PASP after COVID-19. We used the following parameters: echocardiographic findings, general patient characteristics (sex, age, body mass index), comorbidities, including dyslipidemia, and statin therapy.

The univariate regression analysis showed that a decrease in LVEF over time is significantly influenced by the initial phenotype of CHF with LVEF < 50% (odds ratio 0.91 (95% CI 0.83–0.98); p=0.006), and changes in LVEF are significantly affected by the degree of a decrease in LVEF, the presence of dyslipidemia and statin therapy (Table 4).

After the multivariate regression analysis, it was determined that initially reduced LVEF and male sex increase the risk of a decrease in LVEF after COVID-19 (Table 5). According to the multivariate regression analysis, only the presence of dyslipidemia affect the increase in PASP in patients with CHF after COVID-19.

Table 1. Characteristics of patients with chronic heart failure and a history of COVID-19

Parameter	Value
Age, years	69.1 ± 9.7
Sex	
• Male	44.4 %
• Female	55.6 %
Body mass index, kg/m²	30.6 ± 7.1
Degree of lung lesion according to CT (% of damaged lung tissue relative to total lung volume)	
0	32 (59.2)
CT1 (1–25 %)	10 (18.5)
CT2 (26–50 %)	7 (13)
CT3 (51–75 %)	3 (5.6)
CT4 (76–100 %)	2 (3.7)

Table 2. Structure of diseases in patients with chronic heart failure and a history of COVID-19

Diseases	Number of patients	% of the total number
Interstitial lung damage as an outcome of COVID-19	8	14.8
Bronchial asthma	6	11.1
Hypertensive heart disease	43	79.6
Coronary artery disease	25	46.3
Atrial fibrillation	29	53.7
Acute cerebrovascular accident/ transient ischemic attack	5	9.3
Diabetes mellitus/impaired glucose tolerance	26	48.1
Chronic kidney disease with GFR ≤ 60 mL/min/1.73 m²	17	31.5
Dyslipidemia	41	75.9

GFR, glomerular filtration rate calculated using the MDRD formula.

Table 3. Echocardiographic parameters of patients with different CHF phenotypes and a history of COVID-19

Parameter	HFpEF, LVEF > 50 % (n = 39)		HF with LVEF < 50 % (n = 15)	
	Before COVID-19	After COVID-19	Before COVID-19	After COVID-19
LVEF, %	59 ± 7.5	58.3 ± 4.8	45.4 ± 8.6	38.5 ± 7.1
PASP < 30 mm Hg	24 [20; 34.5]	35 [24.5; 45]	31.27 ± 12.8	43.6 ± 15.4

Medians and interquartile ranges (Me [Q1; Q3]) are used to represent non-normally distributed data (the Shapiro-Wilk test) of PASP in the group of HFpEF with LVEF >50%; the means and standard deviations (M ± SD) are used to present all other (normally distributed) data.
PASP, pulmonary artery systolic pressure; HFpEF, heart failure with preserved ejection fraction.

Table 4. Factors of increase in PASP in patients with CHF after COVID-19 according to the univariate regression analysis.

Factor	Odds ratio (95 % CI)	p
Changes in LVEF	0.92 (0.84–1)	0.037
Presence of dyslipidemia	0.21 (0.06–0.79)	0.018
Statin therapy	0.24 (0.05–0.93)	0.046

PASP, pulmonary artery systolic pressure; CI, confidence interval.

Table 5. Results of multivariate logistic regression analysis

Comparison parameter	Odds ratio (95 % CI)		p
	excluding all covariates	including all covariates	
Indicators affecting changes in LVEF			
LVEF before COVID-19	0.91 (0.83–0.98)	0.88 (0.8–0.96)	< 0.001
Sex, male	2.4 (0.71–8.11)	5.92 (1.31–26.75)	0.014
Indicators affecting changes in PASP			
Dyslipidemia	0.21 (0.06–0.79)	0.08 (0.01–0.84)	0.018

CI, confidence interval; PASP, pulmonary artery systolic pressure.

Discussion

Our study showed a significant deterioration of the cardiovascular system in patients with CHF 5–6 months after COVID-19. A statistically significant decrease in LVEF was observed only in the HFrEF and HFmrEF group, and PASP increased regardless of the HF phenotype.

Our findings showed that the degree of lung lesion in the acute period of the disease did not affect changes in PASP in the long-term period (about 6 months). Moreover, interstitial lung damage due to COVID-19 in 14.8% of patients was also not associated with a subsequent increase in PASP and a decrease in LVEF. It should be noted that most of the subjects had a history of COVID-19 without lung lesions according to CT or with a less than 50% lesion of the lung tissue.

We also evaluated the contribution of bronchopulmonary pathology to the worsening of CHF. Chronic obstructive pulmonary disease (COPD) increases the risk of hospitalization of patients with COVID-19 [17], however, only 2 patients of the study cohort had this pathology. It was not therefore included in our retrospective analysis, and we could not make an unambiguous conclusion about the contribution of this disease to the course of CHF. Among the concomitant bronchopulmonary diseases, 11.1% of our patients had asthma of varying severity, but its presence also did not affect changes in LVEF and PASP, which corresponds to the results of an epidemiological survey conducted by Ueda et al. in 2021 [18].

Thus, the reasons for the aggravation in subjects with CHF due to reduced LVEF and increased PASP should probably be looked for in a different area.

Several studies demonstrated an aggravation of PH in the acute period of COVID-19. The estimated prevalence of PH in patients with acute COVID-19 is about 13% and it is due to impaired pulmonary circulation resulting from damage to the lung parenchyma [19], which leads to dilatation of the right heart and the subsequent development and aggravation of HF [9, 11, 16]. The pathophysiology of this type of PH is complex and multifactorial, and the mechanisms such as oxidative stress, mitochondrial dysfunction and DNA damage, inflammation, hypoxia associated with endothelial dysfunction, and microembolism of pulmonary vessels are potential factors causing changes in the pulmonary circulation [20, 21]. In addition, previous studies analyzed the association of COVID-19 with various comorbidities, and they reported predominantly severe or critical cases, and less attention was paid to patients with mild-to-moderate COVID-19 (80% of all cases) as in our study. It remains unclear how COVID-19 affects PH in patients with CHF and initially increased PASP in the long term.

Summing up, we can conclude that the probable causes of aggravation of PH may include previous remodeling of the heart chambers and pulmonary hypervolemia [8].

At the same time, the relationship of changes in PASP after COVID-19 with dyslipidemia is of interest. Cholesterol plays an important role in the penetration of SARS-CoV-2 into the cell. In vitro studies showed that a decrease in membrane-bound cholesterol in cells expressing angiotensin-converting enzyme type 2 (ACE2) led to a decrease in the infection of cells with SARS-CoV-2, since spike protein binding was reduced by 50%

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[22]. Patients with dyslipidemia have high levels of total cholesterol, which probably can lead to an increase in the number of ACE-2 molecules in lipid rafts of the cell and contribute to the penetration of the virus [23].

The meta-analysis by Atmosudigdo et al. [24] showed that dyslipidemia was associated with a 39% increase in the risk of death of COVID-19, a 39% increase in the risk of severe course of the disease, especially in elderly patients or patients with arterial hypertension. Several studies also demonstrated a comparable correlation between dyslipidemia and the severity of COVID-19 [25–29]. However, other studies had opposite results. They showed that lipid metabolism disorders were not associated with the severity of COVID-19 and an increased risk of death in hospital [29, 30]. However, significant changes in lipid metabolism occur in COVID-19, and therefore it is difficult to interpret the findings, since it is not known when exactly dyslipidemia developed [31, 32].

The results of the described studies suggest that dyslipidemia does not increase the risk of severe course but, being a risk factor for CVDs, serves as a reflection of concomitant cardiovascular pathology and, therefore, an indicator of worse prognosis [23]. For example, Choi et al. [31] showed in a systematic review that CVDs potentiated by dyslipidemia, rather than dyslipidemia itself, worsen the prognosis in COVID-19. Especially at non-target levels of low-density lipoprotein (LDL) cholesterol, as in the case of our patients (LDL cholesterol levels were determined during hospitalization after COVID-19). Several meta-analyses investigated the effects of statins on the severity and prognosis of COVID-19. Some studies found that statin therapy could improve prognosis and reduce the risk of death in COVID-19 [33–36], but other studies indicated that statins did not improve outcome of infected patients [37].

It is likely that elevated PASP in patients with CHF and a history of COVID-19 is caused by damage to the vascular endothelium resulting from systemic inflammation, to which both lipid metabolism disorders and COVID-19 can lead.

Thus, the retrospective analysis showed that systolic dysfunction and the progression of PH in patients with CHF and a history COVID-19 are more characteristic of HFrEF and HFmrEF. However, given inconsistent data on all the discussed points, further prospective studies are needed to make a final conclusion.

The study was limited by a small sample; the absence of a comparison group of patients with CHF and without a history of COVID-19; the retrospective design of the study; the inability to fully analyze treatment of patients at the time of hospitalization; a limited list of comorbidities and echocardiographic parameters during hospital stay before and after COVID-19 according to the electronic hospital charts.

Conclusion

In this retrospective study, we analyzed the functional changes in the heart after COVID-19 in patients with chronic heart failure and tried to establish the long-term effects of COVID-19 on changes in key functional parameters, left ventricular ejection fraction and pulmonary artery systolic pressure. We determined that there left ventricular ejection fraction decreased and pulmonary artery systolic pressure increase after COVID-19. Patients with initially reduced left ventricular ejection fraction experienced further aggravation of left ventricular systolic dysfunction and the progression of pulmonary hypertension, and patients with preserved left ventricular ejection fraction had only an isolated increase in pulmonary artery systolic pressure. However, we could not determine the reliable relationship of these changes with COVID-19.

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