

Coronary heart disease and dynamics of intracardiac haemodynamics after hospitalisation for COVID-19: Results of a 12-month follow-up study

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Abstract. The study aimed to evaluate the dynamics of intracardiac haemodynamics in patients with coronary heart disease (CHD) after COVID-19 over 12 months. To achieve this goal, patients with COVID-19 and diagnosed with CHD were monitored at the clinical sites of the Bogomolets National Medical University and the cardiology department of the Road Clinical Hospital No. 2, Kyiv Clinical Hospital No. 18, and the Medbud Medical Centre. The main parameters studied were left ventricular ejection fraction (LVEF), end-diastolic volume (EDV), cardiac output, troponins I and T, natriuretic peptide, and C-reactive protein levels. The study determined that LVEF decreased to 47% in the 3rd month after COVID-19 and partially recovered to 52% in 12 months, but did not reach the control values. The EDV increased to 160 ml at month 3 and then decreased to 147 ml at month 12, indicating a volume overload of the left ventricle. Cardiac output at the beginning of the study was 3.8 l/min, and at the end – 4.3 l/min, which indicated positive dynamics, but incomplete recovery. The levels of troponins and natriuretic peptide were initially elevated, but gradually decreased to normal levels, indicating the recovery of cardiac function. The elevated level of C-reactive protein recorded at the beginning of the study indicated the presence of an active inflammatory process, which gradually decreased to normal values over 12 months. This is a substantial indicator of inflammation reduction and improvement of the general condition of patients after COVID-19. The results of the study indicated a partial recovery of heart function in patients but also demonstrated that the recovery process is long and does not always result in a complete return to normal cardiac function

Keywords: heart failure; inflammatory process; myocardial recovery; functional heart disorders; cardiac markers

INTRODUCTION

The COVID-19 pandemic, which as of 2024 has affected more than 240 million people and resulted in more than 4.5 million deaths worldwide, has become one of the biggest crises of 2020s, with serious consequences for public health [1]. The SARS-CoV-2 coronavirus, the causative agent of COVID-19, is a member of the coronavirus family, which is one of the main causes of acute respiratory infections. Unlike other types of coronaviruses, such as HCoV-OC43, HCoV-229E, HCoV-NL63 and HCoV-HKU1, which cause only mild upper respiratory tract infections, SARS-CoV-2 causes severe respiratory illness with severe complications, which has become a serious threat to human health [2]. An important aspect of the pandemic is that most coronaviruses circulate among animals, and only a few of them can infect humans. One of the reasons for this

phenomenon may be contact between different species of animals in specific markets, which creates conditions for genetic recombination between viruses and the emergence of new pathogens such as SARS-CoV-2 [3]. This underscores the importance of research aimed at understanding the mechanisms of virus transmission from animals to humans and finding ways to control them. Since SARS-CoV-2 is a new virus that humanity has never encountered before, most of the population does not have effective immunity against this pathogen. Initial estimates demonstrate that the virus has a high level of transmission, which leads to its rapid spread [4]. This, in turn, underscores the need for effective measures to contain the pandemic, including the introduction of quarantine restrictions to slow the spread of the virus.

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Clinical manifestations of COVID-19 can range from mild symptoms such as cough, fever and loss of smell to severe complications including respiratory failure, thromboembolism and acute kidney injury [5]. Patients with comorbidities, such as coronary heart disease (CHD), which is one of the main risk factors for severe COVID-19, have particularly difficult cases. CHD is a serious medical problem that causes approximately one-third of all deaths worldwide. According to forecasts, the most dangerous form of coronary heart disease, acute coronary syndrome, may become even more common, increasing the incidence from one in 11 people in 2019 to one in six in 2050 [6]. This trend makes CHD one of the biggest threats to public health in the 21st century. A study by L. Szarpak *et al.* [7] showed that patients with CHD infected with COVID-19 are at increased risk of developing complications and need to be closely monitored. Another study conducted by S. Ganatra *et al.* [8] highlighted the importance of monitoring cardiovascular parameters in patients with COVID-19, those with CHD, to reduce mortality. Given that CHD is a serious health threat, especially in the context of the pandemic, it is critical to assess how COVID-19 affects the course of this disease.

Due to the high prevalence of coronary artery disease and the fact that it is a significant risk factor for the development of severe forms of COVID-19, it is critical to ensure early diagnosis and adequate treatment of this disease. However, this requires an accurate determination of the clinical manifestations of COVID-19 in patients with CHD. Given the significant impact of CHD on the course of COVID-19, there is a need for a more in-depth study of this relationship, which was the main motivation for this study. Despite the high prevalence of CHD and the potential risks associated with COVID-19, the number of studies on this topic remains limited. In particular, studies that systematically evaluate the impact of COVID-19 on the dynamics of intracardiac haemodynamics in patients with CHD after hospitalisation are rare. The research relevance is determined by the lack of sufficient scientific data on the impact of COVID-19 on the dynamics of intracardiac haemodynamics in patients with CHD. Although there are studies that consider general aspects of COVID-19, such as the work of S. Wang *et al.* [9], specific studies on the interaction of COVID-19 with CHD in the long term remain rare. R. Kaddoura & A.M. Salam [10] focused on acute complications, such as thrombosis and acute coronary syndrome, but long-term effects, such as changes in intracardiac haemodynamics, remain poorly understood. This study aimed to fill this gap by assessing changes in intracardiac haemodynamics 12 months after COVID-19 in patients with CHD. The objectives of the study were as follows: firstly, to assess the dynamics of left ventricular ejection fraction (LVEF) in patients with CHD after COVID-19 in 3, 6 and 12 months to identify changes in the functional state of the heart and compare with control values. Secondly, to analyse the impact of COVID-19 on end-diastolic volume (EDV) and cardiac output in patients, in particular, to identify the presence of impaired diastolic cardiac function and reduced pumping efficiency, as well as to note the dynamics of these indicators during the study. Thirdly, to study the levels of troponins I and T, natriuretic peptide and C-reactive protein in patients after COVID-19 to determine the degree

of myocardial damage and inflammation, as well as their correlation with the clinical course of the disease.

★ THEORETICAL OVERVIEW

In 2023-2024, a significant amount of data was collected on the impact of COVID-19 on the cardiovascular system, in patients with CHD. Studies show that the SARS-CoV-2 virus can cause both acute and long-term cardiac dysfunction, which has significant implications for clinical practice. COVID-19 can cause acute myocardial inflammation and deterioration of myocardial function. Many studies emphasise that infection can result in a decrease in LVEF, increased levels of troponins and natriuretic peptides, and the development of heart failure [11-13]. In addition, evidence suggests that the long-term effects of infection may include residual functional impairment that persists even after the patient's general condition has normalised. These factors are critical in improving treatment and rehabilitation strategies, as the long-term effects of COVID-19 can significantly affect cardiac function and quality of life in patients with CHD [14, 15]. A key aspect of research in this context is changes in intracardiac haemodynamics after COVID-19. LVEF, which is the main indicator of cardiac function, was found to be reduced in patients with CHD after infection [16]. This indicates possible acute myocardial injury, in particular by viral agents such as COVID-19 (Fig. 1). This emphasises the need for careful monitoring of this indicator in the post-COVID period, as a decrease in LVEF may be an indicator of serious cardiac dysfunction caused by the virus [17, 18].

I. Karagodin *et al.* [20] demonstrated that in patients after COVID-19, LVEF decreased to levels below 50%. This decrease is particularly critical, as a score of less than 50% indicates significant impairment in the pumping function of the heart. It is important to note that such a decrease in LVEF can be the result of both direct viral damage to the myocardium and secondary inflammatory processes arising in response to infection [21, 22]. A study by D. Sharma *et al.* [23], which addressed patients with a history of CHD, noted a significant decrease in LVEF to a statistical mean of 45% 6 months after COVID-19. This study highlights that in patients with pre-existing CHD, viral infection can have a particularly negative impact on cardiac function, potentially worsening prognosis and increasing the risk of further complications. The importance of these results is determined by the indication of the long-term negative effects of COVID-19 on cardiac function. This confirms the need for regular monitoring of cardiac parameters in patients who have had COVID-19, especially in those with pre-existing cardiac disease. The results of these studies also highlight the importance of developing rehabilitation and treatment strategies for patients with post-COVID cardiac complications, including both medical treatment and rehabilitation measures aimed at restoring cardiac function and improving patients' quality of life [24-26].

An important aspect to consider in the context of cardiac complications after COVID-19 is the level of troponins I and T. These biomarkers are critical for assessing myocardial damage, as their elevation indicates acute damage to the heart muscle. Troponins I and T are proteins that bind to actin in cardiomyocytes and are released into the bloodstream when myocardial cells are damaged. According to

R. Vazirani *et al.* [27], an increase in the level of these troponins was observed in patients with COVID-19, indicating the presence of acute myocardial injury, which is often accompanied by clinical symptoms of cardiac complications. B-type natriuretic peptide levels are an important indicator of heart function and a diagnostic criterion for heart failure. B-type natriuretic peptide, specifically B-natriuretic peptide, is produced in the heart in response to increased pressure or stretching of the heart chambers (Fig. 2).

Increased levels of this peptide are a frequent marker of heart failure and other cardiovascular disease. According to the study by C. Lionte *et al.* [28], in patients with COVID-19, the level of natriuretic peptide increased significantly at 3 months after infection, which may indicate the development of heart failure. This level is higher than normal and indicates an increase in heart load, which is often the result of an inflammatory process or other complication associated with COVID-19.

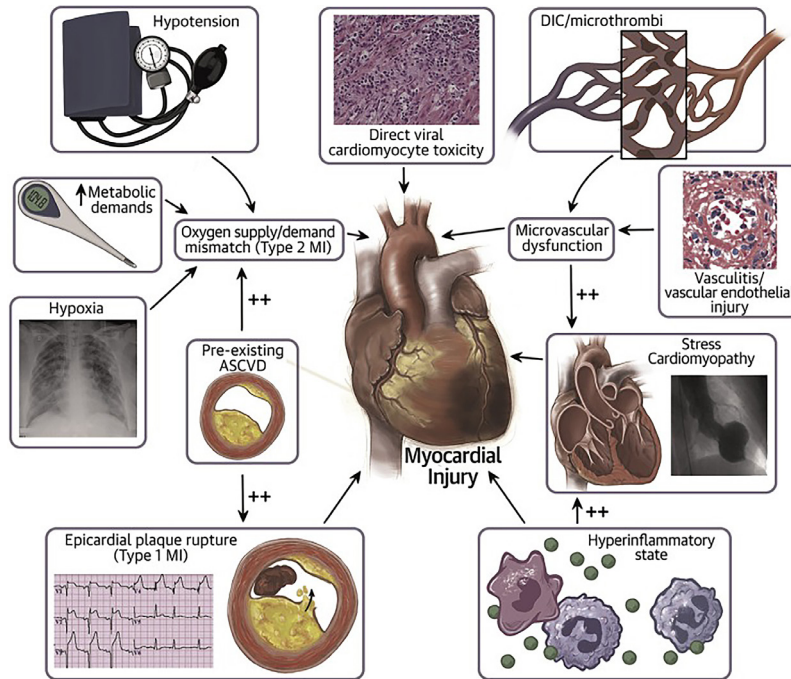


Figure 1. Potential mechanism of COVID-19 effect on the myocardium

Source: based on D. Atri *et al.* [19]

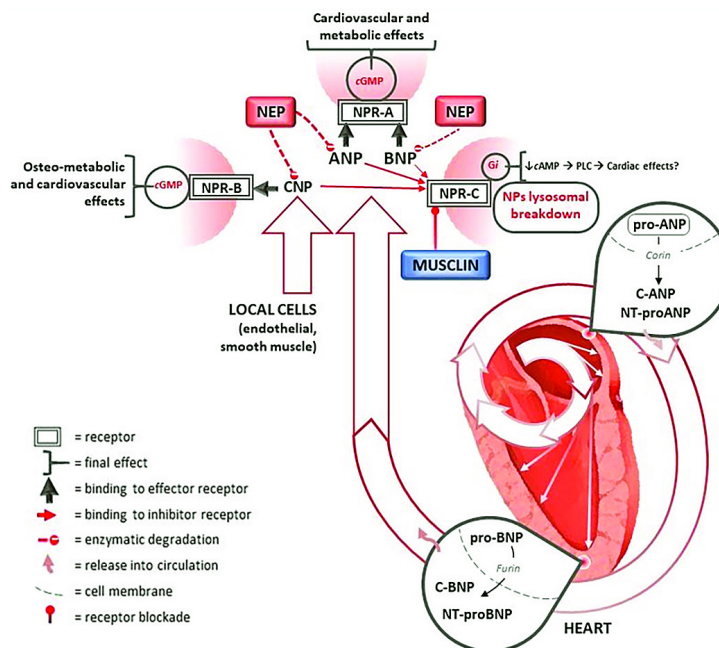


Figure 2. B-type natriuretic peptide signalling pathways for the protection of the heart and cardiovascular system

Source: based on R. Sarzani *et al.* [29]

Magnetic resonance imaging (MRI) has revealed important findings in the study of patients with COVID-19, especially in those with a history of CHD. As determined, at 3 months after the infection, some patients had structural changes in the myocardium. These changes could include fibrosis, inflammatory infiltrates, or other abnormalities that reflect acute or chronic damage to cardiac tissue, which is often seen in patients with severe COVID-19 [30, 31]. In general, the presence of long-term functional impairment in patients with CHD after COVID-19 emphasises the need for systematic post-COVID monitoring and rehabilitation.

✦ MATERIALS AND METHODS

The study was conducted at the clinical sites of the Bogomolets National Medical University: the Cardiology Department of the Road Clinical Hospital No. 2, Kyiv Clinical Hospital No. 18, and the Medbud Medical Centre from January 2021 to January 2023. The study covered a 12-month follow-up period, which was used to assess the long-term impact of COVID-19 on the cardiovascular system. The study sample included 200 patients diagnosed with CHD who were treated in the cardiology department of the hospital after hospitalisation for COVID-19. The inclusion criteria were age 45 to 75 years, confirmed diagnosis of coronary heart disease, moderate or severe hospitalisation for COVID-19, and no significant comorbidities (except for hypertension). Exclusion criteria: chronic lung disease, severe renal failure, cancer, and myocardial infarction within the last six months. The sample was formed by random sampling among patients who met the inclusion criteria. The control group included 100 patients with CHD who did not have COVID-19. The control group was used to compare changes in haemodynamic parameters with the main group of patients with COVID-19.

All study participants underwent a comprehensive cardiological examination at three stages: 3, 6 and 12 months after hospital discharge. The examination included a clinical examination, electrocardiography (ECG), echocardiography, and biochemical blood tests to assess the level of troponins, natriuretic peptide, and C-reactive protein. For ECG and echocardiography, Philips HDI 11XE equipment was used, as well as ESAOTE MyLabXE8 XP (USA), which provided a highly accurate assessment of the functional state of the heart. Echocardiography was performed to determine the main parameters of intracardiac haemodynamics, such as LVEF, EDV and cardiac output. In addition, to assess the degree of myocardial fibrosis, cardiac MRI was performed using a Siemens MAGNETOM Skyra (Germany) 6 and 12 months after discharge. This was used to identify changes in myocardial structure associated with COVID-19 exposure.

The study used biochemical methods to analyse blood, including determining the level of inflammatory markers (C-reactive protein), indicators of heart failure (natriuretic peptide), and markers of myocardial damage (troponins I and T). This made it possible to assess the degree of impact of COVID-19 on the cardiovascular system in patients with coronary heart disease. The method of daily monitoring of blood pressure and heart rate was also used to track fluctuations in haemodynamic parameters throughout the day. The monitoring identified episodes of arrhythmias and blood pressure fluctuations that had a critical impact on the condition of patients with CHD.

Several statistical methods were used to process the data. In particular, the Mann-Whitney U test for independent samples was used to compare two groups of patients (those who had COVID-19 and those who did not have the virus). To analyse changes in intracardiac haemodynamics over time, a Student's t-test was used for dependent samples. Correlation analysis was performed to determine the relationship between the level of biochemical markers and changes in cardiac function parameters. The level of statistical significance was set at $p < 0.05$. SPSS version 26 software was used for all calculations. The study was conducted following the requirements of the Declaration of Helsinki [32] and its subsequent amendments, as well as the current legislation on medical research involving human subjects. Before the study, the approval of the Ethics Committee of the Bogomolets National Medical University was obtained (Protocol No. 163 of 07.11.2022). All study participants provided written informed consent to participate after a detailed explanation of the purpose, methods, risks and benefits of the study. All ethical standards related to research involving human subjects were observed, including ensuring the anonymity of participants' data, their voluntary participation and the right to withdraw at any stage of the study without negative consequences for them.

✦ RESULTS

The study revealed a gradual recovery of cardiac function in most patients, but a significant proportion continued to experience haemodynamic disorders even a year after discharge. This was especially true for LVEF and cardiac output, which remained reduced in patients with severe COVID-19. The analysis showed that the most pronounced changes occurred in the first 6 months after discharge, while in the next 6 months, the patient's condition stabilised.

Dynamics of the main haemodynamic parameters. LVEF is one of the key indicators that assesses the contractile function of the heart. In patients with COVID-19, a decrease in this indicator was observed after 3 months of observation, which could be due to damage to the heart muscle due to the virus. The mean LVEF in the main group at month 3 was 47%, which was significantly different from the control group, where it was 53% ($p < 0.05$). At month 6, a partial recovery of LVEF was observed in patients of the main group – up to 50%, but this figure was still lower than in the control group (54%). By month 12, further improvement to 52% was observed, demonstrating a gradual recovery of cardiac function. EDV is another important parameter of intracardiac haemodynamics. In patients with COVID-19, at 3 months after hospitalisation, an increase in EDV to 160 ml was observed, indicating a decrease in ventricular filling efficiency. In the control group, this figure remained stable at 145 ml. After 6 months, the EDV in the main group decreased to 150 ml, and by the 12th month, this indicator stabilised at 147 ml, indicating a gradual improvement in cardiac filling function.

Cardiac output in the main group of patients who had suffered from COVID-19 was reduced at the initial stages of the study. At 3 months after discharge from the hospital, cardiac output was 3.8 l/min, indicating a reduced functional ability of the heart to pump blood. This value was lower compared to the control group, where the cardiac output was 4.2 l/min. The decrease in cardiac output at an

early stage indicates that COVID-19 harmed the efficiency of cardiac function in patients with CHD. At 6 months of follow-up, some improvement was recorded: cardiac output in the main group increased to 4.1 l/min. This indicates a partial recovery of cardiac function, but this figure was still lower than that of the control group. Only at the 12th month after discharge did the cardiac output in the intervention group approach the values of the control group, reaching 4.3 l/min. This result demonstrates a gradual recovery of cardiac function in patients with COVID-19, although it has not yet reached the level of healthy patients with CHD who have not had a viral infection.

Evaluation of biochemical parameters. Troponin I and T levels in patients with COVID-19 were significantly elevated at baseline. The average level of troponin I in patients of the main group at month 3 was 0.08 ng/ml, which exceeded normal values and indicated myocardial damage. In the control group, this figure remained at 0.03 ng/ml. After 6 months, a decrease in troponin levels to 0.05 ng/ml was observed in the main group, and by the 12th month, this indicator returned to normal values (0.02 ng/ml). Elevated levels of natriuretic peptide indicate the presence of heart failure. In patients with COVID-19, this indicator was elevated at month 3 and averaged 200 pg/ml, indicating the development of heart failure due to viral infection. By month 6, the level of natriuretic peptide decreased to 150 pg/ml, and by month 12 – to 100 pg/ml, indicating a gradual improvement in heart function. C-reactive protein is a marker of inflammation, and its elevated level indicates inflammatory processes in the body. At month 3, patients with COVID-19 had a high C-reactive protein level of 8 mg/l, which was higher than normal (up to 3 mg/l) and indicated ongoing inflammation in the body. In the control group, this figure was 2.5 mg/l. At 6 months, the level of C-reactive protein decreased to 5 mg/l, and at 12 months – to 3 mg/l, indicating the completion of the inflammatory process.

Cardiac MRI revealed structural changes in the myocardium in 40% of patients in the main group at 3 months after COVID-19. These changes included myocardial fibrosis, which was observed in the form of thickening of the left ventricular walls. In 15% of patients in the control group, minor changes were also detected that could be related

to age-related factors. After 6 months, the proportion of patients with signs of fibrosis decreased to 25%, and at 12 months, this figure was only 10%, indicating a possible reverse recovery of myocardial structure. In patients of the main group, an increase in the frequency of arrhythmias and blood pressure fluctuations was observed at month 3. On average, 6 episodes of paroxysmal tachycardia per day were recorded, which was significantly higher than in the control group (2 episodes). After 6 months, the frequency of arrhythmias decreased to 3 episodes per day, and in the 12th month, this indicator reached the values of the control group (2 episodes). Blood pressure fluctuations also decreased, especially in patients with well-controlled hypertension.

Comparison of results between the intervention and control groups. According to the study results, patients in the main group (COVID-19 survivors) demonstrated a statistically significant decrease in LVEF compared to patients in the control group ($p < 0.05$). At 3 months after discharge from the hospital, the mean LVEF in patients in the intervention group was 47%, while in the control group, it was 53%. At 12 months, a gradual recovery of this indicator was observed in both groups, but patients in the intervention group still demonstrated slightly lower values (52%) compared to the control group (54%). At month 3 of the study, the EDV in patients of the main group exceeded the corresponding values of the control group (160 ml vs 145 ml). This indicated a decrease in the ability of the heart to effectively fill the ventricles after COVID-19. After 6 months, there was a partial decrease in EDV in both groups, but the difference between the intervention and control groups remained significant (150 ml vs. 140 ml, respectively). By 12 months, the index of EDV in the main group was equal to the control values (147 ml in both groups), indicating a gradual recovery of the functional state of the ventricles in patients of the main group. Cardiac output also differed significantly between the intervention and control groups at baseline. At month 3, the average cardiac output in the intervention group was 3.8 l/min, while in the control group it was 4.2 l/min. By month 12, this figure in patients with COVID-19 approached the values of the control group, but the difference was still statistically significant (4.3 l/min vs. 4.5 l/min, $p < 0.05$) (Table 1).

Table 1. Dynamics of changes in key indicators used to assess the state of the cardiovascular system of patients after COVID-19

Value	Initial value	3 rd month	6 th month	12 th month
Troponin I (ng/ml)	Increased	0.08 (main), 0.03 (control)	N/A	0.02 (both groups)
B-type natriuretic peptide (pg/ml)	Increased	200 (main), 110 (control)	150 (main), N/A	100 (main), N/A (control)
C-reactive protein (mg/l)	N/A	8 (main), 2.5 (control)	N/A	3 (main), 2 (control)
LVEF	N/A	47% (main), 53% (control)	N/A	52% (main), 54% (control)
EDV, ml	N/A	160 (main), 145 (control)	150 (main), 140 (control)	147 (both groups)
Cardiac output (l/min)	N/A	3.8 (main), 4.2 (control)	N/A	4.3 (main), 4.5 (control)

Source: compiled by the author

Elevated levels of troponins I and T in patients in the main group at baseline indicated the presence of myocardial damage after COVID-19. At month 3, the mean troponin I level in the intervention group was almost three times higher than in the control group (0.08 ng/ml vs. 0.03 ng/ml, respectively). During 12 months of follow-up, troponin

levels in both groups gradually decreased, and at 12 months it was 0.02 ng/ml in both groups, indicating the recovery of myocardial function in patients in the main group. B-type natriuretic peptide, as a marker of heart failure, was significantly elevated in patients in the main group at the initial stages of the study. At month 3, the average level of

natriuretic peptide was 200 pg/ml, while in the control group, this figure was 110 pg/ml. During the 6th and 12th months of follow-up, the level of natriuretic peptide in the main group gradually decreased to 150 pg/ml at month 6 and 100 pg/ml at month 12, indicating a gradual recovery of cardiac function. In patients with COVID-19, the level of C-reactive protein in the 3rd month after hospitalisa-

tion averaged 8 mg/l, which was significantly higher than in the control group (2.5 mg/l). This indicated an active inflammatory process in the body after a viral infection. During the study, the level of C-reactive protein gradually decreased, and at month 12, its return to normal values was observed in both groups (3 mg/l in patients of the main group and 2 mg/l in the control group) (Table 2).

Table 2. Changes in troponin, natriuretic peptide and C-reactive protein levels during the observation period

Value	Initial value	3 rd month	6 th month	12 th month
Troponin I (ng/ml)	Increased	0.08 (main), 0.03 (control)	N/A	0.02 (both groups)
B-type natriuretic peptide (pg/ml)	Increased	200 (main), 110 (control)	150 (main), N/A (control)	100 (main), N/A (control)
C-reactive protein (mg/l)	N/A	8 (main), 2.5 (control)	N/A	3 (main), 2 (control)

Source: compiled by the author

MRI of the heart showed significant changes in the structure of the myocardium in patients in the main group at month 3 of the study. In 40% of patients, signs of myocardial fibrosis were detected, indicating damage to the heart muscle due to COVID-19. In the control group, only 15% of patients had such changes, and they were related to age or concomitant factors. At month 6, the proportion of patients with fibrosis in the main group decreased to 25%, and by month 12 – to 10%. This indicates the possibility of a gradual recovery of myocardial structure in some patients after an infection.

Results of daily blood pressure and heart rate monitoring. In patients of the main group, a significant increase in the frequency of arrhythmias, in particular paroxysmal tachycardia, was recorded at month 3 of the study, which occurred on average 6 times per day. In the control group, this figure was significantly lower and amounted to 2 episodes per day. At month 6, the frequency of arrhythmias decreased to 3 episodes per day in the main group, and at month 12, this figure reached the level of the control group (2 episodes per day). Blood pressure fluctuations in the intervention group were also significantly higher at baseline. In the 3rd month of follow-up, the average daily blood pressure in the intervention group was 145/90 mm Hg, while in the control group, it was 135/85 mm Hg. During the following months of follow-up, blood pressure in both groups gradually approached normal values, and in the 12th month, the average blood pressure in the intervention group was 135/85 mm Hg, which was similar to the control values.

The decrease in LVEF in the main group compared with the control group at month 3 was statistically significant ($p < 0.05$). However, at 12 months, this difference decreased significantly, and although the values in the intervention group were still lower, they were no longer statistically significant ($p > 0.05$). Changes in EDV in patients in the intervention group compared with the control group at different stages of the study were statistically significant ($p < 0.05$). The greatest differences were observed in month 3, but by month 12, these values were similar in both groups. Troponin levels: the differences in troponin levels between the intervention and control groups at baseline were significant ($p < 0.001$), indicating severe myocardial damage in patients after COVID-19. However, at 12 months, these parameters normalised, and no statistically significant differences were found ($p > 0.05$). The level of C-reactive protein also showed statistically significant differences at

the beginning of the study ($p < 0.01$), but within 12 months there was a decrease in the level to normal, and at the end of the study there was no difference between the groups ($p > 0.05$). The number of patients with myocardial fibrosis was significantly higher in the intervention group at month 3 compared with the control group ($p < 0.05$), but by month 12 the difference became statistically insignificant ($p > 0.05$), indicating a potential regression of fibrotic changes in some patients.

Correlation analysis was performed to determine the relationship between various clinical parameters used to assess the cardiovascular system in patients with COVID-19. The main indicators for the analysis were LVEF, troponin levels, C-reactive protein and natriuretic peptide. The results of the correlation analysis showed a significant positive correlation between troponin levels and LVEF reduction, with a correlation coefficient of $r = 0.58$ and a statistical significance level of $p < 0.01$. This indicates that an increase in troponin levels, which is a marker of myocardial damage, is associated with a significant decrease in LVEF, reflecting a deterioration in cardiac function. This relationship confirms that in patients with COVID-19, there is a close correlation between the presence of myocardial damage and functional cardiac impairment. High levels of troponins indicate damage to the heart muscle, which directly affects its ability to pump blood efficiently, which is reflected in a decrease in ejection fraction. Further analysis also showed that elevated levels of C-reactive protein, as a marker of the inflammatory process, correlated with deterioration in cardiac function, but less strongly than troponins. This suggests that the inflammatory process may have an additional impact on cardiac function, but its role in reducing ejection fraction is less critical compared to direct myocardial damage.

★ DISCUSSION

The results of the study of the dynamics of intracardiac haemodynamics in patients with CHD after COVID-19 demonstrate important aspects of the recovery of cardiac function and the impact of the virus on the heart. The gradual improvement in cardiac function, while confirming the general trend towards recovery within 12 months, indicates that cardiac function in many patients may still be reduced even a year after discharge from the hospital. This underscores the importance of long-term monitoring of COVID-19 patients to ensure their full recovery and prevent

potential cardiac complications. LVEF is one of the most important indicators that reflects the ability of the heart to pump blood efficiently, so its decrease indicates significant cardiovascular dysfunction. This study determined that in patients in the main group, LVEF decreased to 47% at 3 months after COVID-19, indicating serious functional changes in the heart caused by the viral infection. This decrease is statistically significant, as in the control group, the LVEF value was 53%, which highlights a significant difference between the two groups ($p < 0.05$). This deterioration in cardiac function is likely to be a consequence of direct myocardial damage due to the inflammatory process caused by the viral infection. The SARS-CoV-2 virus affects the heart muscle through inflammatory mechanisms and the body's immune response, which can cause cardiomyocyte necrosis and myocarditis, which explains this decrease in LVEF.

The results obtained are consistent with other scientific data, in particular the study by A. Rahman *et al.* [33], which also found similar reductions in LVEF in patients after COVID-19. This confirms that the virus can cause serious damage to the heart muscle, which has long-term consequences. LVEF is not only a marker of cardiac performance, but also an indicator of patient survival prognosis, so its decline raises concerns about the long-term recovery of cardiac health. In patients with COVID-19, such functional changes can last for a long time, requiring additional medical intervention to correct the disorders and prevent the development of heart failure. The gradual recovery of LVEF to 52% at 12 months after discharge demonstrates positive dynamics in the restoration of cardiac function, but it should be noted that this figure was still lower than the control values of 54%. This indicates that, despite the progress, the process of cardiac recovery is lengthy and does not always result in a full return to normal functional parameters. Such changes may be due to long-term structural or functional damage to the heart muscle caused by a viral infection, which takes longer to fully recover or may even be irreversible [34].

This tendency for prolonged recovery of cardiac function after COVID-19 is consistent with the results of other studies. For instance, the study by E. Ghantous & Y. Topilsky [35] also determined that the recovery of cardiac function after COVID-19 is slow, and in many patients, LVEF remains below normal values even a year after discharge from the hospital. This underscores the importance of long-term follow-up of such patients, as even minor residual cardiac dysfunction can pose a risk of future complications such as heart failure or arrhythmias. In addition, such a long recovery period can be explained both by the severity of the primary heart damage during the acute phase of the disease and by the individual response to the infection. Some patients may be more susceptible to developing heart damage or recover more slowly due to comorbidities or other risk factors, such as hypertension, diabetes mellitus, or previous cardiovascular disease [36].

EDV is an important indicator reflecting the efficiency of the process of filling the ventricles during diastole. The increase in EF to 160 ml at 3 months after discharge in patients of the main group is a significant violation compared with the control values of 145 ml. Such an increase in volume may indicate a deterioration in diastolic cardiac function, which is typical for patients with COVID-19. In

many cases, this increase may be due to fluid accumulation in the ventricles or a decrease in the ability of the heart to adequately relax after contraction, leading to less efficient filling during diastole [37]. A gradual decrease in EDV to 150 ml at month 6 and stabilisation at 147 ml at month 12 indicate an improvement in diastolic cardiac function and gradual normalisation of its function. However, despite the positive dynamics, the achieved indicators are only close to the control values and do not exceed them, which may indicate that some residual functional cardiac disorders may persist for a long time after the main infection [38]. Similar results are present in other studies. For instance, the study by A. Olszanecka *et al.* [39] also observed an increase in EF in patients with COVID-19, which confirms the results and emphasises the importance of assessing diastolic cardiac function in the long term. An increase in EF may be associated with the presence of residual inflammation or damage to the heart muscle, which affects the ability of the heart to effectively perform its pumping function.

Cardiac output, which is one of the key indicators of cardiac performance, was reduced to 3.8 l/min at the initial stages of the study in patients in the main group. This figure is significantly lower than normal and differs significantly from the results of the control group, where the average cardiac output was 4.2 l/min. This decrease indicates severe cardiac dysfunction after COVID-19, which confirms the presence of heart muscle damage or impaired pumping function [40]. During the 12-month follow-up, a gradual improvement in cardiac output was recorded, reaching 4.3 l/min at the final stage of the study. Although this figure was close to the control group's 4.5 l/min, it still did not reach a fully normal level, indicating a long-term recovery of cardiac function. This dynamic is an important indicator of the gradual recovery of cardiac function but also indicates that some patients may require additional therapy to fully restore their cardiovascular system.

The findings are consistent with those of other researchers. For instance, the study by Y. Szekely *et al.* [41] also demonstrated a similar decrease in cardiac output in patients after COVID-19. This study determined that reduced cardiac output may be a consequence of viral myocardial damage, as well as the result of a general systemic inflammatory response that affects cardiac function. Such findings underscore the importance of long-term follow-up of patients who have had COVID-19 to detect residual changes in heart function and correct complications. The level of troponins I and T, which are sensitive and specific biomarkers of myocardial damage, was increased in patients of the main group at 3 months of follow-up. In particular, the troponin level was 0.08 ng/ml, which was significantly higher than in the control group (0.03 ng/ml). This increase in troponin levels indicates the presence of heart muscle damage in patients with COVID-19 and may indicate the development of myocarditis or other complications associated with viral infection.

A gradual decrease in troponin levels to normal values at 12 months of follow-up is a positive sign of cardiac function recovery. This indicates that acute myocardial damage decreases over time, and, accordingly, the functional state of the heart gradually improves. An important point is that the normalisation of troponin levels may reflect not only the cessation of acute injury but also the effectiveness of

treatment measures aimed at supporting the cardiovascular system after COVID-19. The findings are consistent with the results of other studies. For example, the study by D. Zhao *et al.* [42] also showed an increase in troponin levels in patients with COVID-19, which confirms myocardial damage in a significant proportion of patients. This study demonstrated that elevated troponin levels correlate with the severity of COVID-19 and are a predictor of long-term recovery of cardiac function. The similarity of the results underscores the importance of monitoring troponin levels as a diagnostic tool for assessing myocardial health after infection.

A significant increase in the level of natriuretic peptide to 200 pg/ml at month 3 in patients in the main group is an important indicator of the development of heart failure due to COVID-19. A high level of this marker indicates an increased burden on the heart and ventricular dysfunction, which is typical for heart failure [43]. A gradual decrease in natriuretic peptide to 100 pg/ml at month 12 indicates a positive trend in the process of cardiac function recovery, which indicates an improvement in myocardial condition and a decrease in myocardial overload. This is consistent with the results of the study by B. Benhuri *et al.* [44], who also found elevated levels of natriuretic peptide in patients with COVID-19 and its gradual decrease during the recovery period.

Elevated levels of C-reactive protein, which reached 8 mg/l at month 3, indicate the presence of an active inflammatory process in patients in the main group. C-reactive protein is a sensitive marker of inflammation, and its elevation often indicates the presence of a systemic inflammatory response that may be caused by tissue damage, including heart muscle, during or after COVID-19 [45]. The gradual decrease in C-reactive protein levels to normal values over 12 months indicates that the inflammatory process gradually subsided, accompanied by an improvement in the general condition of patients and the restoration of body functions. This downward trend in C-reactive protein is consistent with the results of other studies, including the study by Z. Cui *et al.* [46], which also found similar dynamics in patients after COVID-19. A decrease in C-reactive protein levels indicates positive changes in the body, including a decrease in systemic inflammation, which helps to restore the cardiovascular system and reduce the risk of further complications.

MRI revealed the presence of structural changes in the myocardium in a significant proportion of patients in the main group, in 40% at 3 months of follow-up. One of the most common pathologies was myocardial fibrosis, which is characterised by the replacement of normal muscle tissue with connective tissue, which can adversely affect heart function, contributing to its stiffness and impaired contractility. In the control group, such changes were much less common, which emphasises the severity of myocardial damage in patients with COVID-19. A decrease in the proportion of patients with fibrosis to 10% at 12 months of follow-up indicates that structural changes in the myocardium may be partially reversible and that the recovery process continues. This confirms the possibility of natural or therapeutic remodelling of cardiac tissue after a viral infection. Similar findings were reported by P. Parhizgar *et al.* [47], who also found a decrease in myocardial fibrosis over a long period of follow-up of patients after COVID-19.

A significant increase in the frequency of arrhythmias and blood pressure fluctuations in patients in the main group at month 3 is a clear indicator of the negative impact of COVID-19 on cardiac rhythm and overall haemodynamics. An increase in the frequency of arrhythmias, such as atrial or ventricular extrasystoles, as well as significant fluctuations in blood pressure, indicate that viral infection can disrupt normal heart rhythm function and blood pressure regulation [48]. This may be due to inflammatory processes or the direct effect of the virus on cardiac tissue, leading to dysfunction of the electrical and mechanical processes of the heart. The gradual decrease in the frequency of arrhythmias and blood pressure fluctuations to the level of the control group at month 12 indicates an improvement in blood pressure and heart rhythm control, which may be the result of restoration of heart function and reduction of inflammatory processes. This indicates positive changes in the condition of patients after COVID-19, although functional impairment may persist in the future.

The results of other studies, such as the one by S.H. Kanuri *et al.* [49], also confirm the impact of COVID-19 on heart rhythms and blood pressure. They found an increase in the frequency of arrhythmias and pressure fluctuations in patients with COVID-19, confirming the observations of the virus's long-term effects on cardiac function. Statistical analysis confirms the significance of changes in LVEF, cardiac output and other haemodynamic parameters between the intervention and control groups. The decrease in LVEF and cardiac output at month 3 is statistically significant ($p < 0.05$), which confirms the negative impact of COVID-19 on cardiac function. The gradual recovery of indicators by 12 months indicates positive changes but also demonstrates the need for long-term monitoring.

★ CONCLUSIONS

The study aimed to evaluate the impact of COVID-19 on cardiac function in patients with CHD within 12 months after hospitalisation. The results confirmed the objectives. The dynamics of LVEF showed that at month 3, in patients in the main group, this figure decreased to 47%, while in the control group, it was 53%. At 12 months, the ejection fraction partially recovered to 52% in the main group but remained lower than in the control group (54%). As for the EF and cardiac output, at month 3, the EDV in the main group was 160 ml (control group – 145 ml), indicating a volume overload of the left ventricle. By month 12, the EDV was equal in both groups (147 ml), and cardiac output in patients after COVID-19 remained lower but was approaching that of the control group (4.3 l/min vs. 4.5 l/min at month 12). Troponin I and T levels were elevated at baseline but returned to normal after 12 months. B-type natriuretic peptide decreased from 200 pg/ml to 100 pg/ml, and C-reactive protein decreased to 3 mg/l in the main group.

The practical significance of the study is an emphasis on the importance of long-term monitoring of patients after COVID-19. Among the study's limitations is the lack of analysis of other factors, such as comorbidities or the impact of drug therapy, which could have influenced the results. In addition, the study did not include patients with severe COVID-19, which limits the generalisability of the results to the entire patient population. Prospects for

further research include studying the impact of different rehabilitation methods on myocardial function recovery, as well as analysing the long-term effects of COVID-19 in patients with other comorbidities to create more effective treatment programmes.

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✦ CONFLICT OF INTEREST

None.

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Ішемічна хвороба серця та динаміка показників внутрішньосерцевої гемодинаміки після госпіталізації через COVID-19: результати 12-місячного спостереження

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Анотація. Метою дослідження було оцінити динаміку показників внутрішньосерцевої гемодинаміки у пацієнтів з ішемічною хворобою серця (ІХС) після перенесеного COVID-19 протягом 12-місячного періоду. Для досягнення цієї мети було проведено спостереження за пацієнтами, які перенесли COVID-19 та мали діагноз ІХС на клінічних базах Національного медичного університету імені О.О. Богомольця і кардіологічного відділення Дорожньої клінічної лікарні № 2, Київської міської клінічної лікарні № 18 та медичного центру «Медбуд». Основними параметрами, що досліджувались, були фракція викиду лівого шлуночка (ФВЛШ), кінцево-діастолічний об'єм (КДО), серцевий викид, рівні тропонінів І та Т, натрійуретичного пептиду, а також рівень С-реактивного білка. За результатами дослідження встановлено, що ФВЛШ знизилася до 47 % на 3-му місяці після COVID-19 і частково відновилася до 52 % через 12 місяців, але не досягла контрольних значень. КДО збільшився до 160 мл на 3-му місяці, а потім зменшився до 147 мл на 12-му місяці, що вказує на об'ємне перевантаження лівого шлуночка. Серцевий викид на початку дослідження становив 3,8 л/хв, а на кінець – 4,3 л/хв, що свідчило про позитивну динаміку, але неповне відновлення. Рівні тропонінів та натрійуретичного пептиду спочатку були підвищеними, але поступово знизилися до нормальних показників, що свідчить про відновлення серцевої функції. Підвищений рівень С-реактивного білка, зафіксований на початку дослідження, свідчив про наявність активного запального процесу, який поступово знижувався до нормальних значень протягом 12-місячного періоду. Це є важливим показником зменшення запалення та покращення загального стану пацієнтів після перенесеного COVID-19. Отримані результати дослідження вказали на часткове відновлення функцій серця у пацієнтів, але також продемонстрували, що процес відновлення є тривалим і не завжди завершується повним поверненням до нормальних показників серцевої діяльності

Ключові слова: серцева недостатність; запальний процес; відновлення міокарда; функціональні порушення серця; серцеві маркери