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THE NEGATIVE EFFECT ON THE HEART OF THE COVID-19 CORONAVIRUS INFECTION. CLINICAL CASE

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ABSTRACT

Acute respiratory tract infections are considered to be triggers of cardiovascular diseases. These include influenza, bacterial pneumonia, and respiratory syncytial infection. If at the time of the disease, the patient has concomitant cardiovascular pathology, then the probability of the development and progression of the infectious process increases. The appearance of complications of cardiovascular diseases against the background of COVID-19 has turned into a pandemic. Most of the patients with the detected virus have cardiovascular diseases, which can lead to coronary heart disease, myocarditis, and other heart diseases even for a long time after recovery. Currently, there are no specific therapeutic strategies for the management of patients with post-covid syndrome. From the point of view of understanding the pathophysiological mechanisms and existing therapy algorithms in cardiology, several therapeutic strategies can be formulated: to control risk factors, including blood pressure, lipid levels, glucose, and obesity. In addition, it is necessary to recommend lifestyle changes and quitting smoking and alcohol, modification of physical activity, and nutrition. This article describes a clinical case - a 57-year-old man who has had COVID-19, including negative changes occurring in his body. The data of the conducted research are given.

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Introduction

COVID-19 caused by a new strain of coronavirus SARS-CoV-2 is an acute respiratory disease characterized by lesions of the upper and lower respiratory tract of varying severity. Clinical manifestations of this disease can have both an asymptomatic course and occur in the form of a severe form of viral pneumonia with the development of acute respiratory failure, and can also be complicated by sepsis and septic shock [1, 2].

The main route of transmission of SARS-CoV is carried out by airborne droplets from person to person. It is known that the SARS-CoV coronavirus strain is released into the environment and can be transmitted through the hands of patients and medical personnel [3]. The rapid spread of COVID-19 with the onset of acute respiratory syndrome has acquired the scale of a pandemic. Therefore, in order to prevent the spread of this disease, sanitary and hygienic requirements have been developed aimed at protecting the eyes and upper respiratory tract from the penetration of coronavirus infection [4].

Despite the property of SARS-CoV-2 to affect mainly lung tissue, the virus attacks various human systems and organs, which contributes to the development of cardiovascular, renal, coagulopathic, metabolic, neurocognitive, motor, and mental disorders [5]. A significant proportion of patients affected by the SARS-CoV-2 coronavirus strain have cardiovascular diseases. Heart

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damage is observed in 28% of hospitalized patients with COVID-19. Awareness of the cardiovascular phenomena of COVID-19 is important for providing timely medical care to patients, especially those with cardiovascular diseases [6].

According to the literature, in the 1930s, a link was established between the activity of the influenza virus and increased mortality from concomitant chronic diseases, including diseases of the cardiovascular system. Based on the data obtained, there is an assumption that a viral infection may be a provoking factor in the development of cardiovascular pathology and subsequent death [7].

The combination of a significant systemic inflammatory process and localized inflammation of the vascular wall in coronavirus infection affects the clinical manifestations of existing diseases of the cardiovascular system and contributes to the formation of complications such as the development of acute coronary syndrome, arrhythmia, decompensation of heart failure, thromboembolic complications [8].

In patients with heart diseases after a coronavirus infection, as a result of damage to the heart muscle (myocardium) by an infectious agent, subclinical and cardiovascular disorders may be observed for a long time [9]. The manifestations of the post-covid syndrome among cardiovascular diseases include cardialgia, tachycardia, extrasystoles, angina pectoris, arterial hypertension, and heart failure [7].

The effect of coronavirus infection on the myocardium and its damage can be characterized by several pathological mechanisms: direct and indirect effects [10].

As a result of the interaction of SARS-CoV-2 with myocardial receptors of angiotensin-converting enzyme 2, direct myocardial damage occurs. Indirect damage to the myocardium can be caused by the influence of cytokines and other pro-inflammatory factors on the cardiac muscle, disorders of microcirculation processes, and changes in cardiomyocytes as a result of hypoxia **(Table 1)** [11].

Table 1. Pathological mechanisms of myocardial damage when exposed to coronavirus infection

Direct damage	Interaction of SARS-CoV-2 with myocardial receptors of angiotensin-converting enzyme 2
Indirect damage	The effect of cytokines, the influence of pro-inflammatory factors, violation of the microcirculation process, hypoxic damage to cardiomyocytes

Complications after coronavirus infection include various types of arrhythmias: atrial fibrillation, tachycardia, and ventricular extrasystole [8]. Instrumental studies of patients after COVID-19, for example, magnetic resonance imaging of the heart, can reveal myocardial edema, fibrosis, and dysfunction of both ventricles of the heart. These data may be the basis of morphological features of the course of cardiovascular pathology of the post-covid syndrome [12].

Viral infections and virus-induced immune reactions are of great importance in the occurrence of the inflammatory process in myocarditis.

Some literature sources believe that the leading mechanisms of myocardial damage in the acute phase of the disease are the following pathological processes:

- invasion into the target cell of a viral particle with a tropicity to the myocardium;
- direct cytopathogenic effect of the virus and the inclusion of non-specific mechanisms of antiviral protection implemented by macrophages and NK cells;
- activated macrophages and other cells of the immune system attract T- and B-lymphocytes to the focus of inflammation, which implement the mechanisms of cell-mediated cytolysis and ensure the production of antiviral antibodies;
- this triggers the mechanism of cardiomyocyte apoptosis with further systolic myocardial dysfunction [13].

Sporadic cases of autopsy and reports of cases of severe myocarditis with systolic dysfunction of the left ventricle after COVID-19 suggest the possibility of infiltration of the myocardium by interstitial mononuclear inflammatory cells [14].

According to various studies, among hospitalized patients who had previously suffered a coronavirus infection, there is a high prevalence of myocardial damage, which is a prognostically unfavorable factor of coronavirus infection [15].

The diagnostic algorithm for patients after COVID-19 is aimed at preventing the exacerbation of cardiovascular diseases, the development of complications, and life-threatening conditions **(Table 2)** [16].

Table 2. Methods of Examination of Patients after COVID-19

Research methods	Main	Additional
Laboratory	• C- reactive protein; • D-dimer; • Clinical blood test with leukocyte formula; • Basic metabolic profile: fasting plasma glucose, lipid profile, uric acid; • General urine analysis; • Biochemical blood analysis: alanine aminotransferase, aspartate aminotransferase, bilirubin, total protein, creatinine, glomerular filtration rate, potassium, sodium; • Natriuretic peptides; • Troponin;	• The Willebrand factor; • Interleukin 6; • Glycated hemoglobin; • Albuminuria; • Antibodies to cardiolipin; • Ferritin; • Antibodies to cardiomyocytes; • Thyroid hormones TSH, T4 free;

Instrumental	• Saturation; • 6-minute walk test; • Echocardiogram;; • Echocardiography; • Holter ECG monitoring; • Chest X-ray/computed tomography of the lungs;	• Daily pulse oximetry; • Spirometry; • Magnetic resonance imaging of the heart with gadolinium; • Coronarography; • Positron emission tomography of the heart; • Computed tomography angiography of the lungs; • Duplex scanning of the veins of the lower extremities;

Currently, there are no specific therapeutic strategies for the management of patients with post-covid syndrome. From the point of view of understanding the pathophysiological mechanisms and existing therapy algorithms in cardiology, a number of therapeutic strategies can be formulated: to control risk factors, including blood pressure, lipid levels, glucose, and obesity. In addition, it is necessary to recommend lifestyle changes and quitting smoking and alcohol, modification of physical activity, and nutrition.

According to international and Russian clinical guidelines for the treatment of hypertension, most patients are recommended to start combination therapy. Following the recommendations, drugs of metabolic action that can be used for myocardial ischemia include trimetazidine and ranolazine [17].

Effective recovery after a coronavirus infection requires drugs that can directly restore energy consumption and the subsequent cascade of recovery processes in relation to membrane repair, synthesis processes, and electrolyte balance not only at the level of cardiomyocytes but also skeletal muscles. It seems appropriate to use phosphocreatine in patients with myocardial and coronary microvascular dysfunction, as well as with post-covid syndrome to optimize energy exchange and accelerate rehabilitation processes.

Clinical Case

A man aged 57 years is a patient of the city clinical polyclinic of Vladikavkaz (Republic of North Ossetia-Alania, Russia). Complaints of shortness of breath during exercise, episodes of palpitations, dizziness, staggering when walking; heaviness in the lower extremities when walking; weakness in the right arm and right leg.

Considers himself ill since 2020. Within two years, there has been an increase in blood pressure to 210/130 mmHg (adapted to the figures of blood pressure 155/95 mmHg).

In March 2020, he suffered a coronavirus infection COVID-19 (laboratory confirmed), complicated by bilateral poly-segmental pneumonia. From the protocol of spiral computed tomography of the chest organs (March 2020): "The area of lesion of the parenchyma of the right lung is 23%, the left lung is 8%. Conclusion: signs of bilateral poly-segmental pneumonia, high probability of COVID-19 viral pneumonia. The degree of lung damage is CT–1."

Outpatient treatment was prescribed: Oseltamivir 75 mg 2 times a day, nasal Interferon alpha–2b, then Levofloxacin 500 mg + Ceftriaxone 2 g / day for 10 days, Acetylcysteine, Vitamin D.

At the time of hospitalization, he does not take medications. Previously, inpatient treatment was not carried out. There were no operations for cardiovascular pathology.

During the examination: the general condition of moderate severity, clear consciousness, activity within the department, and constitutional features overweight (obesity). Height - 185 cm, weight - 95 kg. Body mass index = 27.8 kg / m2. The body structure is correct. The development of subcutaneous tissue is moderately elevated. There is no swelling. The color of the skin is normal. The color of the mucous membranes is not changed, clean. The language is clean. The lymph nodes are not enlarged. The muscles are painless.

Respiratory system: the chest is of the correct shape, the frequency of respiratory movements is 18 per minute, breathing is hard, carried out in all departments, there is no wheezing.

Cardiovascular system: heart tones are muted, rhythmic, there are no heart murmurs, heart rate = 68 beats/min. Blood pressure: on the left hand - 185/110 mm Hg, on the right hand - 175/110 mm Hg. Pulse of satisfactory filling = 68 beats/min.

Pulsation in the lower extremities is reduced. Appetite is preserved. The liver is not enlarged. The spleen is not palpable. The belly is soft and painless. The chair is regular. Urination is free, painless. The symptom of pounding is negative on both sides.

ECG: the rhythm is sinus, correct. Heart rate is 70 beats/min, there is no data for acute coronary insufficiency.

Daily monitoring of the echocardiogram: the main rhythm is sinus. The average heart rate is 70 beats/min, the maximum heart rate is 136 beats/min, the minimum heart rate is 59 beats/min. The circadian index is 1.5. The duration of tachycardia with a heart rate of 95-100 beats /min is 3 hours 10 minutes, with a heart rate of 110-120 beats /min – 63 minutes, with a heart rate of 130-150 beats /min – 5 minutes (all episodes of tachycardia during the day). 2880 single ventricular extrasystoles were registered (mainly during daytime hours), and 6 single supraventricular extrasystoles. The maximum duration of the QTc is 0.46 seconds (at night). Daily blood pressure monitoring: labile systolic arterial hypertension of the 3rd degree was registered during daytime hours. The degree of nocturnal decrease in systolic blood pressure is 13% (dipper), nocturnal decrease in diastolic blood pressure is 14% (dipper).

Ultrasound duplex scanning: atherosclerosis of brachiocephalic arteries with maximum stenosis up to 38%. Tortuosity of both sides with an increase in the linear velocity of blood flows up to 135 cm / s.
Laboratory methods of research: blood for IgG to SARS-CoV-2 (ELISA) is positive, and the coefficient of positivity is 15.7.
Upon discharge of the patient, the diagnosis was established: "Atherosclerosis. Ischemic heart disease: Grade II angina pectoris. Atherosclerosis of the coronary arteries. Arterial hypertension of the III degree., high risk of cardiovascular complications. The condition after suffering a coronavirus infection in March 2020. Postcovid syndrome. Complications: ventricular extrasystole."
Positive dynamics were noted against the background of drug therapy.

Results and Discussion

In the presented clinical case, a patient who had not previously been observed for cardiovascular diseases, after suffering from COVID-19, had complaints of palpitations, heart failure, and increased blood pressure. The examination revealed frequent ventricular extrasystole. In the analyzed case, a course of antibiotic therapy, including cephalosporins and fluoroquinolones, was used to treat bilateral pneumonia.
Beta-blockers in combination with magnesium preparations are effective means of treating ventricular arrhythmias in patients with acquired forms of QT prolongation syndrome [18]. In addition, in the described case, the multifactorial influence of the transferred infection on the pathogenesis of the observed rhythm disturbances is not excluded. So, in the study of S. T. Lau *et al*. It was described that patients recovering from infection caused by SARS group viruses had palpitations in the form of tachycardia at rest or with light physical exertion. Possible causes, according to the authors, were impaired lung and heart function, thyroid dysfunction, anemia, autonomic dysfunction, and anxiety [19].

Conclusion

Data from various studies show that among middle-aged and older patients who have had a coronavirus infection, there is a high risk of developing post-covid complications. In patients with concomitant cardiovascular pathology, the most common complications are cardiac arrhythmias such as atrial fibrillation or ventricular extrasystole. Life-threatening complications include the development of severe acute respiratory syndrome, and septic shock, in the absence of adequate drug therapy fatal outcome. The occurrence of the above pathologies is a prognostically unfavorable condition for the course of coronavirus infection.
Understanding the pathophysiological mechanisms of post-covid complications contributes to the appointment of optimal treatment of comorbid pathology of cardiovascular diseases and coronavirus infection.

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