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# IMPACT OF CORONAVIRUS INFECTION (COVID-19) ON CARDIOVASCULAR SYSTEM IMPACT OF CORONAVIRUS INFECTION ON CARDIOVASCULAR SYSTEM

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**Abstract** Acute viral infections of the respiratory tract can increase the likelihood of progression of existing comorbidities, including those of cardiovascular origin. The emergence of life-threatening complications on the background of coronavirus 2 (severe acute respiratory syndrome coronavirus 2, or SARS-CoV-2), which causes coronavirus disease 2019 (Coronavirus disease 2019, or COVID-19), necessitates studying the cardiovascular effects of COVID-19 in order to provide a rational care for patients, especially the elderly. The article presents a review of the literature data on the analysis of the clinical and functional characteristics of patients with COVID-19, including those who had an unfavorable prognosis. Attention is paid to the pathophysiological features that occur against the background of an infectious process in the cardiovascular system, risk factors and predictors of mortality in COVID-19.

**Keywords:** coronavirus, cardiovascular disease, infection, severe acute respiratory syndrome, coronavirus disease 2019, SARS-CoV-2, COVID-19

Coronaviruses, which got their name because of the characteristic structural features (crown-like spikes on the surface of the virus), belong to the subfamily Coronaviridae, which has four groups: α, β, γ and δ CoVs according to phylogenetic clustering, of which α and β cause infection in humans. Coronaviruses contain four main structural proteins: the spike protein (S) (provides attachment to the host cell receptor and subsequent fusion of the virus with the cell membrane), protein nucleocapsid (N), membrane protein (M), and envelope protein (E). The first human coronavirus (HCoV) was identified in 1965 in cultured human fetal tracheal tissues, and until 2003 only two types of HCoV were recognized: HCoV-229E and HCoV-OC43. The problem of cardiac comorbidity in COVID-19 has several aspects: the impact of concomitant CVD on the incidence of a new viral infection, the severity of its course and the risk of mortality, as well as the possible side effects of a number of drugs traditionally prescribed for the treatment of certain types of CVD With COVID-19, various etiopathogenetic mechanisms of CVC formation can be combined: the presence of pre-existing cardiovascular diseases (CVD), direct and indirect damage to the myocardium and blood vessels, and, finally, the cardiotoxic effects of drugs from different pharmacological groups that are prescribed to treat this infection. Cardiovascular disorders due to COVID-19 Today there is evidence of the negative impact of COVID-19 on the development of de novo cardiovascular disease [10]. N.S. Hendren et al. proposed to designate the cardiac manifestations of COVID-19 to introduce a new concept: acute COVID-19-associated cardiovascular syndrome (acute COVID-19 cardiovascular syndrome, ACovCS), which describes a wide range

of cardiovascular and thrombotic complications of coronavirus infection [8. Acute COVID-19-associated cardiovascular syndrome is represented by arrhythmias (atrial fibrillation, ventricular tachycardia and ventricular fibrillation), acute myocardial injury, fulminant myocarditis (which is significant for the development of HF), effusion pericarditis, cardiac tamponade, arterial and venous thrombotic disorders in the form acute coronary syndrome (ACS), stroke, pulmonary embolism (PE), deep vein thrombosis (see table). Cardiac manifestations may be primary phenomenon in COVID-19 (according to some researchers, this is the "cardiac phenotype" of the disease), but they can also be secondary to to pulmonary damage (mixed pulmonary phenotype) [7]. The risk factors for cardiovascular events in COVID-19 are diverse: CVD and diabetes, advanced and senile age, concomitant diseases of the lungs and kidneys, systemic inflammation and immune responses, coagulopathy and metabolic disorders, multiple organ dysfunction, prolonged immobilization, and, finally, adverse cardiotropic effects of drugs [ 4, 10, 11.20]. Types of CVEs also vary widely: arrhythmias, myocardial injury and myocarditis, heart failure (HF) and cardiomyopathy, acute coronary syndrome (ACS) and myocardial infarction (MI), cardiogenic shock and cardiac arrest, venous thromboembolism [10, 3, 4, 11 , 12, 14, 17, 18]. An important arrhythmogenic factor is myocardial damage, accompanied by an increase in the content of cardiospecific troponin in the blood. In patients with normal levels of the biomarker, the frequency of life-threatening ventricular arrhythmias (VA) is 5.2%, and with hypertroponinemia it reaches 11.5% [5]. **A**ccording to recently published data from an extensive international study, antimalarial drugs and macrolide antibiotics prescribed for the treatment of COVID-19 contribute to the development of VA. It is possible that other drugs used to treat COVID-19 may adversely affect the conduction system of the heart and stimulate ectopic foci of excitation [4, 12]. The hypothesis that influenza can act as provoking factor of acute cardiovascular events and death was proposed in the 1930s. Then for the first time noted the relationship between seasonal activity of the influenza virus and higher mortality from all causes, including bronchopulmonary pathology, pulmonary tuberculosis, diabetes mellitus, organic heart disease and hemorrhagic stroke [4]. In 2004, a wide range of life-threatening clinical manifestations of coronavirus infection was shown, including death due to myocardial infarction, which was the cause of two out of five deaths, which indicates the need to take urgent measures to treat patients with CVD during an epidemic of viral infections [13]. Viral infection and virus-induced immune reactions in most cases underlie the inflammatory process in myocarditis. Invasion of a viral particle with tropism for the myocardium into the target cell, direct cytopathogenic effect of the virus and the inclusion of non-specific mechanisms antiviral protection (implemented by macrophages and NK cells) are the leading mechanisms of myocardial damage in the acute phase of the disease. Among the main symptoms of COVID-19 are fever, cough, feeling short of breath (shortness of breath, rapid breathing). Less common are

myalgia, anorexia, nausea, weakness, sore throat, nasal congestion, headache. Symptoms may appear after 2 days or by the 14th days after contact with the sick person. The severity and extent of clinical manifestations, short-term and long-term cardiovascular changes in the context of COVID-19, along with the effects of specific treatment, are currently unknown and are subject to careful study. It should be noted that during influenza epidemics, most patients are more likely to dies from cardiovascular problems, and not from pneumonia caused by a virus. A study of 1,099 hospitalized and outpatients with laboratory-confirmed COVID-19 (median age 47 years, 42% women) found that.

The most common comorbidities in patients were hypertension (14.9%), diabetes mellitus (7.4%), and coronary artery disease (2.5%). Similar data are presented in another study [7], according to which, out of 187 patients with a confirmed diagnosis of COVID-19, 27.8% developed acute cardiovascular complications leading to cardiac dysfunction and arrhythmias, and the combination of cardiovascular complications with an increase in highly sensitive troponin has been associated with high mortality. Although the exact pathophysiological mechanisms underlying myocardial injury due to COVID-19 are not well understood, current data suggest the presence of the SARS-CoV genome in the myocardium in 35% of patients with SARS. These data increase the likelihood of possible direct damage to cardiomyocytes by viruses. The pathogenesis of myocardial injury in COVID-19 is complex. Several mechanisms are discussed: direct myocardial damage mediated by the interaction of the SARS-CoV-2 virus with myocardial ACE2 receptors, and viral myocarditis, damage to the heart muscle by cytokines and other pro-inflammatory factors, microcirculation disorders and endothelial dysfunction in the coronary bed, and finally, hypoxic changes in cardiomyocytes [4, 5, 10 11, 12, 18]. Myocarditis often manifests as arrhythmias with progressive heart failure and sudden cardiac death, which can occur at any stage of the disease. The first manifestations of myocarditis include weakness, fatigue, myalgia, and occasionally low-grade fever, which are not caused by myocardial damage, but by manifestations of an infectious-inflammatory process.

Heart failure. Data on the frequency, severity and clinical significance of HF in COVID-19 are rather limited. The overall frequency of HF reaches 23%, and if it is 12% in survivors, it increases to 57% in the dead ($p < 0.0001$) [8]. There is no doubt that pathogenetic factors of type 1 and type 2 MI are present in COVID-19 [9]. ystemic inflammation can contribute to the destabilization and rupture of unstable atherosclerotic plaques, and an increase in the procoagulative potential of the blood can lead to thrombosis of the coronary artery, resulting in the development of type 1 MI. Risk factors for type 2 MI are: on the one hand, an increase in the level cytokines, hypercatecholaminemia, hyperthermia and tachycardia, which increase myocardial oxygen demand, on the other hand, hypoxemia, shortening of the period of diastolic myocardial perfusion during tachycardia and a decrease in contractility

with an increase in end-diastolic pressure in the ventricles, reducing oxygen delivery to cardiomyocytes [4, 12]. These considerations have prompted clinicians to develop protocols for the intensive treatment of MI in patients with COVID-19.

Described detailed algorithms are designed to provide myocardial revascularization in combination with minimal risks for both patients and medical personnel [16,19, 22-30]. Taking into account the possible difficulties of transporting patients in serious condition with critical hypoxemia or the absence of anti-epidemic equipped X-ray operating rooms, the possibility of more active use of systemic fibrinolysis is being considered [19, 31-42]. Patients who have had COVID-19, especially in moderate and severe forms, with complications from the cardiovascular system, need medical rehabilitation. Considering that COVID-19 is a multisystem disease, when creating rehabilitation programs, it is better to proceed from the syndromic-pathogenetic approach. The goal of the rehabilitation of people who have undergone COVID-19 is to restore the functions of external respiration **,**transport and utilization of oxygen by working tissues / organs, reducing the severity of dyspnea, maintaining CVS and reducing the risk of cardiovascular complications, improving the quality of life, normalizing psychological status, restoring daily activity and returning a person to an active life [2, 43-55].

**Conclusion.** In conclusion, it can be stated that the SARS-Cov-2 virus has a pronounced cardiotropism due to both the infection mechanism mediated by ACE2 receptors and

the ability to damage the myocardium due to systemic inflammation, hypercytokinemia, hypercoagulability, and oxygen delivery / consumption imbalance. These pathological processes are especially significant in patients with concomitant CVD that increase the risk of both severe COVID-19 and death. All this requires maximum cardiological vigilance in the treatment of patients with COVID-19, the timely use of EchoCG, ECG, control of biomarkers of myocardial damage and stress, as well as pathogenetically justified prescription of cardiotonic and cardioprotective drugs.

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