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THE COURSE OF CARDIOVASCULAR COMPLICATIONS IN PATIENTS WITH COVID-19

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ANNOTATION

In a pandemic situation caused by a coronavirus infection, a special risk group is made up of patients with cardiovascular diseases, which are often found in the population. The co-occurrence of these diseases increases the risk of adverse effects. Caring for these patients requires physicians to be aware of the characteristics of the viral infection, its clinical presentation, the events that occur in association with cardiovascular disease, and individual and collective protection measures. The safety of medical personnel and a good prognosis for patients is one of the highest values of the modern healthcare system. Arrhythmias , myocarditis and heart failure are not only typical clinical manifestations of coronavirus infection, but also occupy a prominent place in the structure of mortality. The problem is exacerbated by the potential cardiotoxicity and arrhythmogenicity of a number of drugs prescribed for the treatment of COVID-19. All this requires maximum cardiac vigilance in the treatment of patients with COVID-19, timely use of echocardiography, ECG, control of biomarkers of myocardial damage and stress, as well as pathogenetically justified prescription of cardiotonic and cardioprotective drugs.

Keywords: Cardiovascular disease • Heart failure • ACS • Prognosis • Drug therapy • Arrhythmias • Myocarditis

INTRODUCTION

A number of reviews, cohort studies and case reports on cardiovascular complications (CVC) of SARS-CoV-2 (COVID-19) infection have been published in recent months. For example, Italian colleagues reported the treatment of a 53-year-old patient without concomitant cardiac pathology, whose clinical manifestations of confirmed COVID-19 were not pneumonia, but severe myopericarditis with fever, laboratory changes, and hemodynamic destabilization [8]. Clinicians believe that the development of cardiovascular diseases exacerbates the severity of the patient's condition and increases the risk of death [4]. Indeed, it appears that the risk of cardiovascular disease caused by novel coronavirus infection is higher than the risk observed during epidemics caused by SARS-CoV (severe acute respiratory syndrome) [2] and MERS-CoV (Middle East respiratory syndrome) [9].

Data on the frequency of concomitant arterial hypertension in patients with COVID-19 are directly related to the actively discussed relationship between the risk of infection with the SARS-CoV-2 virus and the use of blockers of the renin-angiotensin-aldosterone system (RAAS) - Angiotensin, converting enzyme inhibitors (ACE) and blockers angiotensin II receptors (ARBs). It is known that the initial stage of SARS-CoV-2 penetration into target cells is the interaction of the peplomer (spike protein, S-protein) of the virus with type II ACE receptors (ACE2), in which the transmembrane serine protease TMPRSS2, which activates the viral peplomer, plays an important role. [7]. Recall that the structures of the ACE2 receptor primarily mediate the formation of angiotensin II from inactive angiotensin I. A number of researchers have suggested that long-term use of ACE inhibitors and/or ARBs for the treatment of hypertension may be accompanied by increased expression of ACE2 receptors in the respiratory tract, which increases the risk of COVID infection. -19 [20]. The basis for such concerns was experimental studies demonstrating that ACE inhibitors and ARBs can increase the number of ACE receptors in tissues and change their functional activity [22]. Chronic use of an ACE inhibitor and/or ARB for the treatment of hypertension does not affect the risk of morbidity, severity, and mortality from COVID-19. Risk factors for CVD in COVID-19 are diverse: CVD and diabetes, advanced senility, comorbidities in 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 [17, 18, 1, 10]. The types of CVD 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 [17, 4, 18, 1 , 10, 11, 23, 7].

Etiopathogenetic factors of cardiac arrhythmia and conduction disturbances in COVID-19 can be hypoxia, hyperthermia, agitation, hypercatecholaminemia, electrolyte and metabolic disorders, myocardial injury, myocardial ischemia/infarction, and finally drug side effects [17, 18, 1, 10, 23]. Palpitations are the main symptom of COVID-19 in patients without fever and cough. Among hospitalized patients, the frequency of arrhythmias is about 17%, while in patients in the intensive care unit, it rises to 44%. It is emphasized that arterial hypoxemia increases the likelihood of developing atrial fibrillation, especially in the elderly [1, 23]. An important arrhythmogenic factor is myocardial damage, accompanied by an increase in the blood levels of cardiospecific troponin. In patients with normal levels of biomarkers, the frequency of life-threatening ventricular arrhythmias (VA) is 5.2%, and with

hypertroponinemia - 11.5% [24]. In a Wuhan study of 187 patients hospitalized with COVID-19, patients with elevated troponin T levels were more likely to develop malignant arrhythmias such as ventricular tachycardia and fibrillation than those with normal troponin T levels (12% vs. five%). Sudden cardiac arrest in and out of the hospital has also been reported in patients with COVID-19. According to a recently published data from a large international study, antimalarial drugs and macrolide antibiotics prescribed for the treatment of COVID-19 contribute to the development of ventricular arrhythmias (VA) [5]. 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 [18, 10].

In COVID-19, it is proposed to use two definitions of myocardial injury: extended and reduced. In the first case, myocardial damage is determined by one or more of the following signs [25, 16]:

- Blood levels of troponin above the 99th percentile of the upper reference limit;
- New electrocardiogram (ECG) changes - supraventricular tachycardia, ventricular tachycardia, atrial fibrillation, ventricular fibrillation, bundle branch block, ST segment elevation/depression, T wave flattening/inversion, QT prolongation;
- New echocardiographic (EchoCG) changes - a decrease in the left ventricular ejection fraction (LVEF <50%) or a further decrease in LVEF in patients with LVEF <50%, impaired general or segmental contractility, pericardial effusion, pulmonary hypertension.

Using the abbreviated definition of myocardial injury, they are limited to finding a blood troponin level that exceeds the 99th percentile of the upper reference limit, regardless of changes in ECG and echocardiography [11]. Using the detailed definition, evidence of myocardial injury was found in 12–17% of all hospitalized patients with COVID-19 and in 31% of patients in the intensive care unit [25]. According to other data, myocardial damage, diagnosed only by the level of troponin, is typical for 19.7% of patients with COVID-19 who are hospitalized [11]. Pathological levels of troponin I (>28 ng/l when using a highly sensitive method of determination) in patients with AMI are detected almost 8 times more often than in other clinical cases [25]. Inflammatory changes and fibrosis have been described in the myocardium of patients who died from COVID-19 [14], but there is still no direct evidence for the presence of viral ribonucleic acid (RNA) in cardiomyocytes. At the same time, in a similar clinical situation in patients who died from severe acute respiratory syndrome, evidence was obtained of the interaction of the SARS-CoV virus with myocardial ACE2 receptors, and RNA of this virus was found in the myocardium [19]. If the myocardium was "SARS-CoV-positive", the morphological signs of its damage were much more pronounced, and the life expectancy of patients in the hospital was shorter than when biopsied with "SARS-CoV-negative" biopsies [19]. The high probability and severity of myocarditis in COVID-19 is beyond doubt [4]. Moreover, the main symptoms of COVID-19 may be HF and pericarditis [8]. Myocarditis and heart failure have been reported to account for up to 7% of total COVID-19 mortality [4].

Data on the incidence, severity, and clinical significance of HF in COVID-19 are rather limited. The overall incidence of heart failure reaches 23%, and in survivors - 12%, in the dead it increases to 57% ($p < 0.0001$) [16]. Elevated levels of the N-terminal B-type natriuretic peptide precursor (NT-proBNP) have been considered in a number of studies as a laboratory sign of HF; indicate that the assessment of the biomarker of myocardial tension in combination with

echocardiography allows diagnosing heart failure [18, 8]. Normal levels of NT-proBNP have been described in patients without signs of myocardial injury (139–141 pg/mL) and significantly increased in hypertroponinemia (817–1689 pg/mL) [24,8]. The development of a severe form of heart failure is accompanied by an increase in the content of NT-proBNP in the blood up to 8000-8500 pg/ml [8]. In patients with COVID-19, a direct correlation was shown between NT-proBNP and troponin T values [24]. In patients who died from COVID-19, the level of the biomarker before death in the presence of morphological signs of myocardial damage was 12 times higher than in observations where such signs were not detected [14]. There is reason to believe that the unfavorable prognosis of COVID-19 is indicated not so much by the degree of increase in NT-proBNP, but by its dynamics during treatment. An increase in biomarker values is characteristic of an unfavorable outcome of the disease [24].

To date, there are no published results from targeted studies of ACS or MI in patients with COVID-19. Nevertheless, many experts point out the possibility of an increased risk of these complications [18,10]. There is no doubt that pathogenetic factors of type 1 and type 2 MI are present in COVID-19 [29]. Systemic inflammation can contribute to the destabilization and rupture of unstable atherosclerotic plaques, and an increase in the procoagulant potential of the blood can contribute to coronary artery thrombosis, leading to type 1 MI. Type 2 MI risk factors are: on the one hand, an increase in the level of cytokines, hypercatecholaminemia, hyperthermia and tachycardia, an increase in myocardial oxygen demand, on the other hand, hypoxemia, a reduction in the period of diastolic myocardial perfusion during tachycardia, a decrease in contractility with an increase in end-diastolic pressure in the ventricles, reduced oxygen delivery to cardiomyocytes [18, 10]. Taking into account the possible difficulties in transporting patients in serious condition with critical hypoxemia or the lack of X-ray operating rooms equipped with anti-epidemic agents, the possibility of more active use of systemic fibrinolysis is being considered [15].

The high risk of cardiovascular diseases in COVID-19 determines the interest of clinicians in the correct tactics of prescribing sympathomimetic cardiotonics and vasopressors, as well as in the advisability of using cardioprotective drugs. The possibility of myocardial damage and heart failure determined the increased attention to the timely administration of dobutamine. The recommendations "Anesthesia and resuscitation of patients with a new coronavirus infection COVID-19" [21] indicate that patients with arterial hypotension, despite taking norepinephrine and signs of myocardial dysfunction, should be prescribed dobutamine, and not increase the dose of norepinephrine. This recommendation is extremely important, since dobutamine is the only sympathomimetic cardiotonic agent capable of causing pulmonary vasodilation [26]. Pulmonary hypertension is highly likely in community-acquired pneumonia and ARDS [12], including coronavirus infection [6, 25], and can lead to severe right ventricular dysfunction/failure [13]. Dobutamine is the sympathomimetic of choice for the treatment of this variant of acute heart failure. It has been shown to be effective in community-acquired pneumonia [12] as well as in the treatment of heart failure resulting from myocarditis caused by COVID-19 [8]. Another Russian recommendation [21] for cardiotropic therapy is the appointment of phosphocreatine in the complex therapy of myocarditis and / or myocardial damage associated with COVID-19. The effectiveness of the latter in viral myocarditis has been demonstrated in a number of works by Chinese researchers [3, 28]. The mechanism of the

complex cardioprotective action of exogenous phosphocreatine is described in sufficient detail [3]. This effect is realized in patients of different age groups, including children [27, 28]. It is important that phosphocreatine has practically no side effects and drug interactions with drugs for the treatment of COVID-19 - lopinavir / ritonavir, hydroxychloroquine, ribavirin and tocilizumab [21]

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 imbalance. These pathological processes are especially important in patients with comorbid cardiovascular diseases, which increase the risk of both severe COVID-19 and death. Arrhythmias, myocarditis and heart failure are not only typical clinical manifestations of coronavirus infection, but also occupy a prominent place in the structure of mortality. The problem is exacerbated by the potential cardiotoxicity and arrhythmogenicity of a number of drugs prescribed for the treatment of COVID-19. All this requires maximum cardiac vigilance in the treatment of patients with COVID-19, timely use of echocardiography, ECG, control of biomarkers of myocardial damage and stress, as well as pathogenetically justified prescription of cardiotonic and cardioprotective drugs.

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