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COVID-19 AND HEART FAILURE

Madazimova D. H.[1], Zhabborov O. O.[2], Buvamuhammedova N.T.[3], Anand Ahudja[4]

[1,2,3]Tashkent Medical Academy, Tashkent, Uzbekistan
[4]Rhythm Heart Institute, India
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ABSTRACT

Infectious disease caused by the SARS-CoV-2 virus (COVID-19) often occurs in patients with various cardiovascular risk factors and cardiovascular diseases (CVD), which can affect the course of the infectious process. On the other hand, with COVID-19, additional damage to the heart and blood vessels is possible, contributing to the occurrence of cardiovascular complications and aggravation of the course of CVD. This review examines the main facts concerning these interactions, as well as some practical approaches to the management of patients with the simultaneous presence of COVID-19 and cardiovascular pathology..

The pandemic of coronavirus infection COVID-19 (Coronavirus Disease-2019), which is caused by a new strain of coronavirus – SARS-CoV-2 (severe acute respiratory syndrome coronavirus-2), has caused a rapid increase in the number of cases and high mortality worldwide [1]. Despite the tropism of SARS-CoV-2 to the lungs, COVID-19 has a high risk of developing multiple organ failure, including due to damage to the cardiovascular system (CVS).

Viral infection can destabilize the CVD, which significantly increases the risk of mortality in concomitant CVD. The study showed that the risk of death in CVD increased by 2.4 times (p = 0.019) [12]. An analysis of 44,672 cases with a confirmed diagnosis of COVID-19 from the *Chinese Center for Disease Control and Prevention* found a high mortality rate in patients with CVD (10.5 %), hypertension (6.0 %) and DM (7.3 %), while the overall mortality rate was 2.3 % [13].

The increasing incidence of myocardial damage, vascular dysfunction, and thrombosis in COVID-19 patients, including those with asymptomatic or minimal early-stage manifestations of the infection, raises important questions about potential long-term cardiovascular events, which may include heart failure, life-threatening cardiac arrhythmias and conduction disorders, sudden cardiac death, impaired myocardial blood flow due to microvascular damage, coronary artery and aortic aneurysms, and arterial hypertension. hypertension, lability of heart rate and blood pressure response to physical activity, accelerated development of atherosclerosis, as well as venous and arterial thromboembolism [6,7].

Patients with CHF are at risk for severe COVID-19 and its complications; the risk of serious complications requiring hospitalization increases significantly with age. People over 65-70 years of age who have heart failure and concomitant diseases, such as diabetes, obesity, and bronchopulmonary diseases, are particularly at risk. A viral infection can cause decompensation of heart failure, i.e., an increase in shortness of breath, the appearance of edema, weight gain due to fluid retention. Fever increases the load on the heart.

A viral infection can provoke decompensation of chronic HF in the presence of CVD with the subsequent development of a shock state. The frequency of HF in those who died from COVID-19 was 52 %, and among survivors-12 % ($p < 0.0001$) [10], the frequency of new cases of HF in those hospitalized with COVID-19 was on average 23 % [10].

COVID-19 was found to cause heart failure in 23% of the 191 inpatient patients in Wuhan, China [13]. Cases of severe myocarditis with reduced systolic function after a coronavirus infection have also been reported [10]. Among 68 deaths out of 150 patients with COVID-19, 7% were associated with myocarditis and circulatory insufficiency [2]. But it remains unclear whether the heart failure is due to an exacerbation of a pre-existing ventricular dysfunction or a new cardiomyopathy [1].

In research, right ventricular dilatation detected by echocardiography (EchoCG) was found to be associated with a high risk of hospital death in COVID-19 patients [53]. The development of pulmonary hypertension in COVID-19 due to lung damage and hypoxia or due to pulmonary embolism increases the load on the right ventricle, leading to damage to cardiomyocytes. In patients with COVID-19 with decompensation of chronic HF, it is important to monitor diuresis daily, observe a rational drinking regime, and continue taking planned basic therapy, including ACE inhibitors[8].

Thus, patients with chronic heart failure (CHF) constitute a special risk group for severe COVID-19 and a very high risk of complications. It is possible to aggravate the course of CHF against the background of a previous coronavirus infection due to the addition of respiratory failure typical for this disease, fibrous changes in the lung tissue and exacerbation of cardiopulmonary failure. In this regard, it is optimal to organize dispensary follow-up of patients with CHF in high-risk offices.

In the convalescent period after COVID-19, symptoms may persist for a long time, including subfebrility, dyspeptic phenomena, so it is necessary to continue monitoring the volume of fluid consumed and excreted, and the patient's body weight, bearing in mind the need to increase fluid intake with an increase in its losses (significant sweating, fever, diarrhea, etc.). If there are appropriate indications, it is necessary to monitor blood electrolytes and kidney function. It should be remembered that increased shortness of breath can be associated with both decompensation of CHF, and PE, or with the development of respiratory failure.

Compared to the healthy control group and the control group corresponding to risk factors, patients who were recently cured of COVID-19 had a lower ejection fraction, higher volumes and higher left ventricular mass, and overestimated T1 and

T2 scores on MRI. But none of these indicators correlated with the time of the COVID-19 diagnosis. [4]

In terms of X-ray imaging, in addition to "frosted glass" syndrome and thickening of the interlobular septum, the ratio of central and gradient distribution was higher in patients with heart failure than in patients with COVID-19. In patients with heart failure, the degree of expansion of small pulmonary veins was higher, and the regression of lung damage was significantly accelerated after effective treatment of heart failure [12].

So, with concomitant CVD, severe forms of COVID-19 often develop, requiring hospitalization of the patient in the ICU and worsening his/her prognosis. In this regard, in all patients, including those with moderate and even mild COVID-19, it is necessary to assess the state of the CCC initially and monitor it in the future. Exposure to the SARS-CoV-2 virus and other pathogenic factors with toxic, pro-inflammatory, and procoagulant effects can lead to decompensation of concomitant CVD and increase hospital mortality.

Since a number of drugs used in COVID-19 have a cardiotoxic effect, constant monitoring of hemodynamic parameters, ECG and echocardiography (as indicated) is necessary. It is advisable to involve COVID-19 survivors in medical rehabilitation programs for faster and better recovery of functions of various systems (primarily respiratory and cardiovascular), improving the quality of life and reducing the risk of disability.

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