

DIAGNOSIS AND TREATMENT OF MYOCARDIAL DAMAGE IN PATIENTS WITH COVID-19

Asila K. Kuzieva¹, Kholmurod S. Akhmedov²

¹Master's degree, Cardiology, Department of Internal Diseases, Faculty of Medicine and Education,
Tashkent Medical Academy, Tashkent, Uzbekistan.

Email: An19nazar@gmail.com,

ORCID: 0000000346411537

²Doctor of Medical Sciences, Head of the Department of Internal Diseases of the Medical and
Pedagogical Faculty, Tashkent Medical Academy, Tashkent, Uzbekistan.

Email: Akhmedov.kholmurod@gmail.com

ABSTRACT

The cardiovascular system's complications in the treatment of COVID-19 are one of the main problems facing doctors today. The greatest danger of the disease is for people with pathological changes in the vascular system. However, it was found that changes in the heart also occur in patients who have not previously experienced this problem. Some patients have hypercoagulation syndrome, accompanied by thrombosis and thromboembolism. According to the latest data, it is established that changes in the blood clotting system and heart muscle occur even when the disease occurs without clinical signs.

Keywords: coronavirus, COVID-19, myocardial damage, arrhythmia, echocardiography, electrocardiography, Angiotensin-converting enzyme 2 (ACE II).

I. INTRODUCTION

With COVID-19 virus infection and the respiratory system, serious life-threatening changes are observed in the central nervous, gastrointestinal, endocrine, immune, and cardiovascular systems. Experts from the United States, Italy, and China claim that the virus can negatively affect the heart's physiological parameters. These changes are caused by an inflammatory reaction or are caused by damage to the heart muscle. The impact of coronavirus on the cardiovascular system is manifested by the development of various complications (myocarditis, arrhythmias, thromboembolism, heart failure).

Diagnosis and treatment of myocarditis arising based on COVID-19 have a complex and completely unsolved problem over the past year. There are no generally accepted scientific views that justify the occurrence and development of myocarditis caused by COVID-19. It makes it difficult to diagnose and treat myocarditis. Also, myocarditis can cause rhythm and conduction disorders, as well as the development of chronic heart failure, and can be observed for a long time. A heart rhythm and conduction disorder are developing in these patients, and a life-threatening situation occurs. The study, conducted by a team of scientists from Germany, Italy and Russia, showed that two months after the onset of symptoms of the coronavirus, 78 out of 100 patients had changes in the heart, and 60 of them had myocarditis. The data mentioned above indicate that the early diagnosis of myocardial damage in COVID-19, the elimination of life-threatening rhythm and conduction disorders, is one of the problems facing doctors. In this regard, there is not enough necessary information. Today, early detection and elimination of observed changes in the cardiovascular system in the treatment and prevention of COVID-19 are important challenges facing doctors working in the healthcare system.

II. MATERIALS AND METHODS

An examination of patients with myocarditis of the etiology of COVID-19 was planned. And all patients underwent the necessary laboratory and revolutionary studies.

1. Collecting complaints and anamnesis
2. Biochemical blood tests (Troponin)
3. Electrocardiogram (ECG)
4. Echocardiography

III. RESULTS

In COVID-19, it is suggested to use two definitions of myocardial damage: expanded and abbreviated. In the first case, myocardial damage is defined as one or more of the following signs [1, 2]:

- cTn content in the blood exceeding the 99th percentile of the upper limit of the reference values;
- new changes on the electrocardiogram (ECG) – supraventricular tachycardia, ventricular tachycardia, atrial fibrillation, ventricular fibrillation, blockage of the legs of the His bundle, elevation/depression of the ST segment, flattening/inversion of the T wave (the repolarization of the ventricles), prolongation of the QT interval (a measurement made on an electrocardiogram used to assess some of the electrical properties of the heart);
- new echocardiographic (EchoCG) changes – a decrease in the left ventricular ejection fraction (LVEF < 50%) or a further decrease in LVEF for patients with LVEF < 50%, violations of general or segmental contractility, pericardial effusion, pulmonary hypertension. Applying the abbreviated definition of myocardial damage, they are limited only to the cTn level statement in the blood exceeding the 99th percentile of the upper limit of the reference values, regardless of EchoCG changes [3]. When using the expanded definition, signs of myocardial damage were detected in 12-17% of all hospitalized COVID-19 patients and in 31% of patients in the Intensive Care Unit (ICU) [21]. According to other data, myocardial damage diagnosed only by the cTn level is typical for 19.7% of COVID-19 patients receiving inpatient treatment [3]. The pathological level of cTn I (> 28 ng/l when using a highly sensitive method of determination) in patients with ICU is detected almost eight times more often than in other clinical observations [1]. In deceased patients, the content of cTn I is on average ten times higher than in discharged patients [4].

In the context of a real pandemic, a severe acute respiratory syndrome caused by a coronavirus (SARS-CoV-2) should be considered as a predictor and potential source of myocardial damage and the development of fibrosis/myocardial (FM). According to various authors, in COVID-19, acute myocardial damage occurs from 12% to 28-30%. In patients with myocardial damage associated with coronavirus pneumonia, it is necessary to pay attention to specific clinical manifestations and symptoms and specific tests to determine the damage to the myocardium and treatment choice.(5)

The situation with the COVID-19 outbreak is developing rapidly with uncertain clinical and physiopathological characteristics. Regardless of the underlying cardiovascular disease, abnormally elevated myocardial damage markers are widespread in patients diagnosed with COVID-19. They are closely related to the disease's progression and prognosis. According to Huang et al., among 41 patients with COVID-19, 5 (12%) were diagnosed with acute myocardial injury, mainly manifested by an increase in troponin I [6]. In a retrospective study of 99 patients diagnosed with COVID-19, most patients had elevated myocardial damage markers. In another single-centre retrospective study of 138 patients diagnosed with COVID-19, ten patients (7.2%) were diagnosed with acute myocardial injury, of which two were with a mild course of the disease, which was 2% of the total number, eight patients had a severe course, which was equal to 22% of the total number of patients. Besides, the levels of Creatine kinase MB, Lactate dehydrogenase (LDH), and TnI in severe patients admitted to intensive care units were higher, suggesting that myocardial damage in patients with COVID-19 is associated with disease progression. Among the earliest published deaths, one patient was hospitalized with myocarditis associated with COVID-19. The diagnosis was confirmed based on increased biomarkers of myocardial damage and corresponding abnormal electrocardiographic manifestations [7].

Currently, the exact mechanism of myocardial damage caused by infection is not entirely clear. However, it may be clear from numerous studies that SARS-CoV2 can cause myocardial damage and, thereby, affect the prognosis and course of the disease. From a treatment perspective, first of all, it is necessary to actively treat the COVID-19 infection to control the progression of pneumonia. If the patient also has cardiovascular diseases, they should also be actively monitored and treated. The American College of Cardiology (ACC) has published an

announcement on the effects of SARS-CoV-2 on the heart, recommending that patients with cardiovascular diseases and COVID-19 actively prescribe statins, beta-blockers and renin-angiotensin system (RAS), depending on the indications [8]. Acetylsalicylic acid is also recommended for patients with chronic cardiovascular diseases. If acute myocardial damage occurs in a patient infected with SARS-CoV-2, it is reasonable to use drugs such as coenzyme Q10, Adenosine triphosphate (ATP) preparations, sodium creatine phosphate and vitamin C, fish oil, etc. if myocardial damage in patients with COVID-19 results in rhythm disturbances, the type of arrhythmia should be treated according to the patient's hemodynamic status [9].

With COVID-19, we can suspect two types of myocardial damage: primary or secondary. In the case of primary injury, patients mostly present cardiac rather than respiratory complaints. The cause of primary damage may be acute myocardial infarction (type 1), viral myocarditis, or stress-induced cardiomyopathy. Patients complain of chest pain, and elevated cardiac enzymes are detected in tests. Left ventricle (LV) dysfunction and changes associated with the ST-T segment on the ECG are observed in echocardiography but with normal coronary vessel patency on the angiogram [10]. Secondary damage is associated with direct damage to the cardiomyocyte virus, generalized inflammation. It is expressed by a tendency to increase cTnI in parallel with an increase in inflammatory biomarkers (interleukin-6, D-dimer, ferritin, and LDH).

IV. DISCUSSION

This clinical example confirms the data of numerous studies that COVID-19 aggravates the course of existing and leads to the development of new cardiovascular diseases. The addition of acute myocardial damage leads to a pronounced aggravation of the disease's course and contributes to the occurrence of multiple organ dysfunctions.

V. CONCLUSION

In the context of the existing threat of a new coronavirus disease pandemic, patients with initial cardiovascular pathology represent a special risk group with high rates of adverse outcomes. In conclusion, it can be stated that the SARS-Cov-2 virus pronounced cardiotoxicity due to both the mechanism of infection mediated by the Angiotensin-converting enzyme two receptors. And the ability to damage the myocardium due to systemic inflammation, hypercytokinemia, hypercoagulation and an imbalance of oxygen delivery/consumption. These pathological processes are particularly significant in patients with cardiovascular diseases, which increases both the risk of severe COVID-19 and death. Myocarditis and Heart Failure (HF) are typical clinical manifestations of coronavirus infection and occupy a prominent place in mortality. The problem is compounded by the potential cardiotoxicity and arrhythmogenicity of several drugs prescribed for the treatment of COVID-19.

All these require maximum cardiological alertness in treating patients with COVID-19, timely use of EchoCG, ECG, control of biomarkers of myocardial damage and tension, and pathogenetically justified prescribing of cardiostimulant and cardioprotective drugs. The greatest danger is the rhythm and conduction disturbances that occur when the heart is damaged, which can be a harbinger of sudden death cases.

CONFLICT OF INTERESTS AND CONTRIBUTION OF AUTHORS

The authors declare the absence of apparent and potential conflicts of interest related to this article's publication and report on each author's contribution.

SOURCE OF FINANCING

No funding was required for this research.

REFERENCES

1. Huang C., Wang Y., Li X. et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *Lancet*, 2020, vol. 395, pp. 497–506.
2. Zhou F., Yu T., Du R. et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. *Lancet*, 2020, vol. 395 (10229), pp. 1054–1062.
3. Shi S., Qin M., Shen B. et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. *JAMA Cardiol.*, 2020, vol. 25, pp. e200950.
4. Ruan Q., Yang K., Wang W. et al. Correction to Clinical predictors of mortality due to COVID-19 based on an analysis of data of 150 patients from Wuhan, China. *Intense. Care Med.*, 2020, vol. 6, pp. 1–4.
5. Tersalvi G. et al. Elevated troponin in patients with coronavirus disease 2019: possible mechanism // *J. Card. Fail.* 2020. Vol. 26, N 6. P. 470–475.
6. Согласованная экспертная позиция по диагностике и лечению фульминантного миокардита в условиях пандемии COVID-19, Ойноткинова О.Ш., Масленникова О. М., Ларина В. Н. // *Академия медицины и спорта.* -2020. -Т. 1, № 2. -С. 28–40.
7. Zhou F. et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study // *Lancet*. 2020. Vol. 395, N 10 229. P. 1054–1062.

8. Kapoor A., Pandurangi U., Arora V. et al. Cardiovascular risks of hydroxychloroquine in treatment and prophylaxis of COVID-19 patients: A scientific statement from the Indian Heart Rhythm Society // Indian Pacing Electrophysiol J. — 2020. — S0972- 6292(20)30038
9. Huang C., Wang Y., Li X. et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China // The Lancet Published Online. — 2020.
10. Bansal M. Cardiovascular Disease and COVID-19 // Diabetes & Metabolic Syndrom

Список сокращений

Covid - 19 - COronaVirus Disease 2019

КФК- МВ - изоферментов креатинкиназы

АПФ II - ангиотензин-превращающего фермента II

АТФ - аденозинтрифосфата

АГ — артериальная гипертония

NT-proBNP - натрийуретического пептида

ФМ - фульминантного миокардита

БИТ — блок интенсивной терапии

РААС — ренин-ангиотензин-альдостероновой система

ЭКГ — электрокардиография