Chronic Heart Failure In Post-Covid Syndrome

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ABSTRACT

In addition to damage to the nervous and respiratory systems, kidneys, liver and other organs, doctors note frequent complications after coronovirus on the heart and blood vessels, which can significantly limit a person's daily and professional life, as well as cause delayed mortality. There is a sufficient number of studies proving that coronavirus causes complications in the human heart and blood vessels, and such complications also affect patients who have not previously suffered from diseases of the cardiovascular system.

Keywords: COVID-19, post-COVID syndrome, cardiovascular complications, heart failure

Despite the fact that COVID-19 poses a threat to the general population, people aged 60 years and over, as well as those aged 18-59 years with comorbid hypertension, malignancy, COPD, DM, obesity and cardiovascular disease (CVD) are at high risk of severe COVID-19. An increased risk was also noted among residents of large cities and metropolitan areas [3] .

In addition to the respiratory system, SARS-CoV -2 can affect various other organs and systems, including the cardiovascular system. Patients with CVD and risk factors (male sex, old age, obesity, hypertension, diabetes) are not only prone to severe infection, but also have a high risk of hospital mortality. According to some data, with concomitant CVD, the risk of death increases by 2.4 times [4, 5] .

Among the various comorbid conditions in deceased patients with COVID-19, the leading positions are occupied by hypertension, diabetes and ischemic heart disease. CHF was noted in history in 17.1% of patients (*Fig*) [3] . According to F. Zhou et al ., CHF was detected in 23% of patients with COVID-19 [6] .

According to researchers (S. Huang et al ., 2020), elderly patients with coronavirus infection, suffering from hypertension and other CVDs, as well as COPD and DM, are often at risk of developing acute respiratory distress syndrome (ARDS), septic shock, difficult to correct metabolic acidosis and coagulation dysfunction [7] .

At the same time, CHF is a predictor of adverse outcome in patients with coronavirus infection. So, according to some reports, CHF occurred as a concomitant disease in 51.9% of deaths, while acute kidney injury was diagnosed in 11.7% of cases [8] . Patients with CHF who have had COVID-19 are readmitted within the first 6 months after discharge in about 50% of cases [9] .

According to the analysis by P. Goyal et al ., out of 8920 people hospitalized with COVID-19, 11% had a history of CHF. The presence of heart failure also increased the risk of death compared with that in persons without a history of CHF by almost 2 times (31.6 vs. 16.9%, respectively) [10] .

It must be emphasized that the period of clinical manifestations of COVID-19 is not limited to the moment of obtaining a negative

laboratory result. According to M. Tempany et al ., out of 139 patients who had this infection, 19% noted that they did not feel fully recovered, and 71% reported persistent persistent symptoms [11] . In this regard, the post-covid syndrome is an urgent problem, in relation to which numerous studies are being carried out.

The purpose of our review is to consider the pathogenetic relationship between COVID-19 and heart failure, the concept of " post-COVID syndrome", and the management features of this group of patients.

Clinical And Pathogenetic Relation Between Heart Failure And New Coronavirus Infection Covid-19

According to some reports, despite active study, the direct mechanisms of damage to the heart and blood vessels when exposed to SARS-CoV-2 have not been fully established [8] . Therefore, understanding the pathogenesis of COVID-19 is the basis of diagnosis and prevention [12] .

Angiotensin-converting enzyme type 2 (ACE-2), which regulates the renin- angiotensin - aldosterone system (RAAS), is believed to play a key role in binding to SARS-CoV-2 viral particles and their entry into the cell . ACE 2 is found in the tissues of the brain, kidneys, heart, lungs, and testicles. In addition, ACE 2 is especially expressed in type 2 alveolocytes , epithelial cells of the ileum and colon, esophagus, and also in cholangiocytes [13] . There is evidence that more than 80% of ACE 2 is present in type 2 alveolocytes and endotheliocytes , and therefore the respiratory and cardiovascular systems are more susceptible to damage [8] .

When exposed to SARS-CoV-2 on cardiomyocytes , direct damage cannot be ruled out. Viral RNA has also been reported in autopsy samples from the hearts of deceased patients with coronavirus infection. In addition to the direct damaging effect, the role of hypoxemia in pneumonia and ARDS in the development of intracellular acidosis and lipid peroxidation with damage to the phospholipids of the cardiomyocyte membrane and apoptosis of the latter is considered. On the other hand, lactic acidosis , according to E.D. Bazdyreva et al ., can lead to diastolic dysfunction and impaired coronary perfusion [8] .

According to E. Nakou et al ., the main putative pathophysiological mechanisms that cause cardiovascular complications associated with COVID-19 include:

- direct cytotoxic myocardial damage;
- suppression of ACE 2, which performs cardioprotective function (antifibrotic , antioxidant and anti-inflammatory factor);
- endotheliocyte damage , thrombosis and inflammation;
- excessive production of pro-inflammatory cytokines, leading to endothelial dysfunction and activation of complement pathways, platelets, von Willebrand factor and tissue factor, which together increase the risk of thrombosis;
- hypoxic injury;
- side effects of drugs (the role of azithromycin , tocilizumab , chloroquine and hydroxychloroquine is discussed) [14] .

Destabilization of atherosclerotic plaques is also considered to be a proposed mechanism [15] .

Recent studies report a possible indirect cardiotoxic effect of antiviral therapy. In particular, antiviral drugs such as lopinavir and ritonavir can lead to prolongation of QT and PR intervals, especially in patients with a prolonged QT interval or at risk of conduction disturbance [8] .

Coronavirus infection, in turn, can provoke decompensation of CHF. According to M.G. Bubnova and D.M. Aronova, CHF occurred in 52% of those who died from COVID-19 and in 12% of survivors. Perhaps, against the background of a viral infection and initial systolic dysfunction, ARDS, there is a high probability of developing acute heart failure. In addition, the mechanism of right ventricular CHF mediated by pulmonary hypertension due to hypoxia or pulmonary embolism is also considered [4] .

However, at present it is not clear whether CHF occurs more often due to an increase in pre-existing left ventricular

dysfunction or due to the developed dysfunction of *de novo* [16, 17] .

Today, the mechanisms of development of the cardiovascular consequences of COVID-19 are of increasing interest. It is believed that the inflammatory response leads to the death of cardiomyocytes and fibro-fatty replacement of desmosomal proteins. Changes in the heart also cause a decrease in myocardial reserve, dysregulation of the RAAS, and glucocorticoid therapy . Developing fibrosis in the myocardium threatens the occurrence of reciprocal arrhythmias. In addition, arrhythmias are supported by hypercatecholaminemia , an increase in the content of pro-inflammatory cytokines [18] .

The Concept Of "Post-Covid Syndrome"

In connection with the detection of persistent symptoms of COVID-19 for a long time after recovery, the UK National Institutes of Health proposed a classification of post-COVID conditions (2020):

- acute COVID-19 (symptoms lasting up to 4 weeks);
- ongoing symptomatic COVID-19 (symptoms lasting 4 to 12 weeks);
- post-covid syndrome (symptoms lasting more than 12 weeks, not explained by an alternative diagnosis, capable of changing over time).

Ongoing symptomatic COVID-19 and post-COVID syndrome are sometimes referred to as long COVID [19] .

According to the WHO definition, post-COVID conditions are symptoms usually occurring 3 months after the onset of COVID-19 that last ≥ 2 months and cannot be explained by an alternative diagnosis [20] . In connection with the introduction of the concept of " post-COVID syndrome" in September 2020, a code was introduced into the ICD-10 to designate this condition: U09.9 - a condition after COVID-19 [21] .

This disorder led to the realization that the absence of SARS-CoV-2 after COVID-19 does not necessarily mean a complete recovery [22] .

According to DV Parums , the median time for onset of post-COVID syndrome after the onset of the first symptoms of infection was 219 days [23] . In a prospective In a cohort study of patients who underwent COVID-19, 77 days after recovery, post-COVID syndrome was detected in 50.9% [24] .

Numerous clinical studies indicate various non-specific manifestations of the post-COVID syndrome. Thus, according to the materials of JH Becker et al ., a high frequency of cognitive impairment was found several months after infection with COVID-19 [25] . A.P. Kazarin and V.M. Selikhanov emphasize the emergence of psychiatric problems not only in patients, but also in their physicians [26] . A study presented by M. Taquet et al . showed that almost 20% of people diagnosed with COVID-19 are diagnosed with mental disorders such as anxiety, depression or insomnia within 3 months [27] .

According to M. Luo et al ., the prevalence of anxiety and depression was highest among patients with pre-existing illnesses and COVID-19 infection (56 and 55%, respectively). Studies in China, Italy, Turkey, Spain, and Iran suggest that the incidence of these conditions is higher in healthcare workers in the aggregate than in the general population [28] .

In an outpatient follow-up in Italy, on average 2 months after the first manifestations of COVID-19, 87.4% of patients discharged from the hospital reported persistence of symptoms. The most common symptoms were fatigue (53.1%), shortness of breath (43.4%), arthralgias (27.3%), and chest pain (21.7%). More than 50% of patients experienced 3 or more symptoms [29] .

According to prospective In a cohort study conducted in Wuhan, China, 76% of patients reported at least one persistent symptom 6 months after experiencing COVID-19 . The most common manifestations were fatigue/muscle weakness (63%), dyssomnia (26%), and anxiety/depression (23%) [30] .

According to the results of a Swiss study, 39% of patients noted persistent symptoms 7-9 months after the diagnosis of a new coronavirus infection COVID-19. Fatigue

(20.7%), ageusia or anosmia (16.8%), dyspnea (11.7%), and headache (10.0%) were the most frequently reported [31] .

YM Zhao et al . describe the presence of fibrotic changes in the lungs, according to CT data, 3 months after mild and moderate COVID-19 (25 and 65% of cases, respectively) [32] . In another prospective In a cohort study, 62% of patients had residual CT changes at 6 months, such as parenchymal streaks, irregular interfaces (bronchovascular , pleural, or mediastinal), traction bronchiectasis , pulmonary honeycombs, ground glass, and thickening of the interstitial tissue [33] . In addition, according to some reports, micro- and macrothrombosis of the pulmonary vessels occurs in 20–30% of patients [34] .

EL Graham et al . describe post-COVID-19 neurological symptoms: brain fog (81%), headache (68%), paresthesia (60%), dysgeusia (59%), anosmia (55%), myalgia (55%) , dizziness (47%), pain (43%), blurred vision (30%), tinnitus (29%) [35] .

The literature also describes the manifestations of post-covid syndrome from the gastrointestinal tract: loss of appetite (24%), nausea (18%), acid reflux (18%), diarrhea (15%), bloating (14%), belching (10%), vomiting (9%), abdominal pain (7%) and bloody stools (2%) [36] .

On the part of the cardiovascular system, the consequences of COVID-19 are manifested by arrhythmia, myo- and pericarditis, as well as cardiomyopathy (CMP). Heart failure may appear several weeks after discharge from the hospital. According to KS Bhatia , 4% of patients after suffering COVID-19 are diagnosed with atrial fibrillation and flutter for the first time. 0.5% developed for the first time a high-degree atrioventricular block: II (Mobitz II) and III degree. In 2% of cases, cardiomyopathy and heart failure are newly diagnosed [37] .

COVID-19 infection can have long-term effects on the heart. It is possible that an acute cardiac event contributes to the transition of previously asymptomatic heart failure to clinically significant . In addition, persistent myocardial damage caused by infection can increase the risk of CHF decompensation. Many survivors of severe COVID-19 may be at risk for right ventricular heart failure due to pulmonary hypertension [38] .

Patients with ECG changes, cardiomegaly , arrhythmias or CHF should undergo echocardiography , as well as routine vaccination against influenza and pneumococcal infection [3] .

A complete clinical examination is recommended for patients with post-COVID syndrome and concomitant CHF. The 12-lead ECG should always be repeated and compared with the acute phase ECG, if available. Elevated BNP/NT- proBNP levels associated with post-COVID-19 dyspnea are useful to evaluate systolic and diastolic myocardial function and perform transthoracic echocardiography .

Particular attention during echocardiography deserves the determination of the size and function of the right ventricle, including a comprehensive assessment of the likelihood of pulmonary hypertension. Impaired pulmonary hemodynamics should be considered and interpreted in the context of left ventricular dysfunction and valvular anomalies, as well as in terms of lung disease (both parenchymal and vascular).

Chest CT can be used as a comprehensive non-invasive imaging modality that evaluates the lung parenchyma as well as the pulmonary and coronary arteries. Numerous studies have divided CT findings into several stages depending on the time since the onset of symptoms: early, intermediate, late, and recovery stage.

Indications for MRI in assessing the late clinical phase of COVID-19 include accurate determination of chamber size and function, detection of ischemia, myocardial infarction, myocarditis, and stress cardiomyopathy.

Wearable devices should be considered in patients with CHF for routine monitoring of vital signs such as oxygen saturation, respiratory rate, blood pressure, body temperature, ECG (for example, to measure the QT interval), as well as position and movement assessment, lung auscultation, and cough monitoring .

According to D. Richter et al ., in the late phase of COVID-19, treatment of diseases of the

heart and vessels diagnosed before the acute phase or occurring in the acute phase should be continued, as in the treatment without infection [38] . However, it is worth emphasizing some features.

It is now known that discontinuation of RAAS blockers in patients with COVID-19 may increase the risk of cardiovascular complications, especially in the presence of hypertension, chronic heart failure, and myocardial infarction [4] . Abrupt discontinuation of indicated classes of drugs may be associated with higher mortality in the acute and subacute phases of COVID-19 [39] . Therefore, it is necessary to continue taking ACE inhibitors or sartans [4] . It has also been established that ACE inhibitors play an important anti-inflammatory role, participating in the work of the RAAS by converting angiotensin II to angiotensin 1-7, which has anti-inflammatory properties [3] .

In addition, with decompensation of CHF, daily control of diuresis and adherence to a rational drinking regimen are important [4] .

To date, the question of the use of corticosteroids remains controversial. CHF has been reported as a potential risk factor for worse outcomes in patients hospitalized with COVID-19. High doses of corticosteroids can cause sodium and water retention in patients with CHF, which can potentially lead to decompensation of the disease. Therefore, high-quality randomized clinical studies of the efficacy and safety of these drugs in patients with a high risk of complications.

According to the results of the work of LM Pérez-Belmonte et al ., the treatment of CHF in patients hospitalized with COVID-19 was carried out with RAAS blockers [40] . A recent observational study in hospitalized patients with COVID-19 showed that the use of RAAS inhibitors and β- blockers is associated with lower mortality [41] .

According to V.I. Podzolkov and A.I. Tarzimanova , in the presence of hypertension in patients with heart failure who underwent COVID-19, in order to prevent cardiovascular complications, it is recommended to take antihypertensive drugs in accordance with the previously administered prescription. The effect of statins on the course of infection is also being actively studied . In addition to the known pleiotropic effect on the cardiovascular system, statins reduce the ability of SARS-CoV-2 to replicate through direct inhibition of the main viral protease [42] .

Conclusion

Studying the data of numerous modern studies on the new coronavirus infection and post-covid syndrome, one can only once again be convinced that the problem of COVID-19 is extremely relevant and its further research has global prospects.

Given the importance of heart failure, the prevalence of which is steadily increasing, and manifestations are one of the leading causes of disability and mortality, it can be concluded that patients with a combination of CHF and post-COVID syndrome represent a special group with a high risk of cardiovascular complications and death.

Thus, careful development of management tactics for patients with CHF in combination with post-COVID syndrome and effective rehabilitation measures is required to prevent long-term adverse effects in them.

Bibliography

1. Зокиров М.М. & Касымова, С. А., & Рустамова, И. К. (2019). Нейропсихологическое исследование пациентов с длительной посттравматической эпилепсией. *Молодой ученый*, (4), 116-118.
2. Sarvinoz, T., & Muzaffar, Z. (2022). Rehabilitation aspects of water therapy in modern medicine. *Uzbek Scholar Journal*, *6*, 102-106.
3. Sarvinoz, T., & Muzaffar, Z. (2022). Rehabilitation for childhood cerebral palsy. *Uzbek Scholar Journal*, *6*, 97-101.
4. Nabievna, M. Y., & Muzaffar, Z. (2022). Literatural review of the relevance of the problem of neurosaids. *Modern Journal of Social Sciences and Humanities*, *4*, 558-561.
5. Nabievna, M. Y., & Muzaffar, Z. (2022). Modern View on the Pathogenesis of Hiv

Encephalopathy. *Spanish Journal of Innovation and Integrity*, *6*, 478-481.
6. Muzaffar, Z., & Okilbeck, M. (2022). Dementia and arterial hypertension. *Modern Journal of Social Sciences and Humanities*, *4*, 19-23.
7. Muzaffar, Z., (2022). Chronic Obstructive Pulmonary Disease in Combination with Cardiovascular Diseases. *European Multidisciplinary Journal of Modern Science*, *6*, 150-155.
8. Зокиров, М., & Мухаммаджонов, О. (2022). Особенности развития тревожных и депрессивных расстройств при заболеваниях, сопровождающихся хроническим болевым синдромом. *Barqarorlik va yetakchi tadqiqotlar onlayn ilmiy jurnali*, 841-844.
9. Зокиров, М., & Мухаммаджонов, О. (2022). Вич энцефалопатия и его патогенетические аспекты. *Barqarorlik va yetakchi tadqiqotlar onlayn ilmiy jurnali*, 855-858.
10. Muzaffar, Z. (2022). HIV Encephalopathy and its Pathogenetic Aspects. *European Multidisciplinary Journal of Modern Science*, *4*, 843-846.
11. Зокиров, М. М., Рустамова, И. К., Касимова, С. А., & Кучкарова, О. Б. (2019). Жарохатдан кейинги талвасада кечки нейровизуализацион ўзгаришлар. In *Современная медицина: новые подходы и актуальные исследования* (pp. 56-60).
12. Zokirov M., Mukhammadjonov, O. (2022). Cognitive Impairments in Patients with HIV-Associated Encephalopathy. *Central asian journal of medical and natural sciences*, *3*(2), 401-405.
13. Zokirov, M. M., & Mukhammadjonov, O. (2022). Cognitive impairment in patients with Parkinson's disease and optimization of its treatment. *Eurasian Scientific Herald*, *7*, 177-180.
14. Зокиров, М., & Туланбоева, С. (2022). Когнитивные нарушений у пациентов с ВИЧ–ассоциированной энцефалопатией. *Barqarorlik va yetakchi tadqiqotlar onlayn ilmiy jurnali*, 68-73.
15. Muzaffar, Z. (2022). Literature reviews on nervous system damage during hiv infection. *Barqarorlik va yetakchi tadqiqotlar onlayn ilmiy jurnali*, *2*(9), 141-147.
16. Muzaffar, Z. (2022). Correction of cognitive disorders in patients with hiv encephalopathy. *Web of Scientist: International Scientific Research Journal*, *3*(12), 402-411.
17. Muzaffar, Z. (2022). Psychological State in Patients with HIV Infection. *Amaliy va tibbiyot fanlari ilmiy jurnali*, *1*(6), 52-56.
18. Зокиров, М., & Мадмаров, Д. (2022). Корреляция ээг картины головного мозга и когнтитивного статуса у пациентов с эпилепсией. *Theoretical aspects in the formation of pedagogical sciences*, *1*(5), 227-230.
19. Зокиров, М. (2021). Medical sciences. *scientific ideas of young scientists*, 21
20. Зокиров, М. (2022). Анализ когнитивных нарушений у пациентов с вич–энцефалопатией. *Barqarorlik va yetakchi tadqiqotlar onlayn ilmiy jurnali*, *2*(10), 251-260.
21. Muhammadjonov, O., & Zokirov, M. Anemiya kasalligida bemorlarning surunkali buyrak etishmovchiligida epokrin preparatini qo'llash. *студенческий вестник Учредители: Общество с ограниченной ответственностью" Интернаука"*