

## THE COURSE OF CHRONIC HEART FAILURE IN THE POST-COVID PERIOD IN PATIENTS WITH ISCHEMIC HEART DISEASE

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### **Introduction.**

It is known that viral infections can provoke the development of acute coronary syndrome, arrhythmias, decompensation of heart failure, thromboembolic complications mainly due to a combination of a pronounced systemic inflammatory response and limited inflammation of the vascular wall. COVID-19 is no exception in this regard, which is likely to change the clinical manifestations of current cardiovascular diseases (CVD) and lead to the development of additional life-threatening complications.[1].The severity and extent of clinical manifestations, short-term and long-term cardiovascular changes against the background of COVID-19, long-term complications, the course of the disease in patients with CHF, and possible complications in this group of patients are still poorly understood.

The SARS-CoV-2 virus can cause direct damage to cardiomyocytes, leading to CHF decompensation, shock, and sudden death. The results of recent studies have shown an increased risk of developing various arrhythmias in cardiovascular patients who have undergone COVID-19 [2,3].

**The aim of our study** was to study the features of the course of CHF in patients with ischemic heart disease (IHD) angina pectoris, FC II-III, complicated by CHF FC II-III in the post-COVID period.

### **Material and research methods.**

69 patients were studied with IHD, exertional angina, FC II-III, complicated by CHF FC II-III, who were hospitalized at the Multidisciplinary Clinic of the Tashkent Medical Academy. The first group of patients consisted of 44 patients with IHD who underwent COVID-19 in 2020-2021. The interval of the period of the 1st group after undergoing COVID-19 was  $4 \pm 0.43$  months. Group 2 - 25 patients with the same diagnosis of IHD without undergone COVID-19. All patients were underwent an assessment of complaints, anamnesis taking, examination of the objective status, including heart rate (HR), pulse, pressure, and conventional

examinations of cardiac patients: general blood analysis and urine analysis, coagulogram, lipid spectrum, echocardiography (EchoCG) on the device Toshiba, Holter monitoring (HM) of an electrocardiogram (ECG).

In the patients examined by us, a PCR test for coronavirus and IgG SARS-CoV-2 coronavirus in the blood were evaluated.

The average age of patients in group 1 was  $64.2 \pm 9.3$  years, in group 2 -  $67.0 \pm 11.1$  years. The clinical condition of the examined patients was assessed according to the SHOKS scale (according to V.Yu. Mareev, 2000). We assessed the quality of life of the examined patients using the Minnesota Questionnaire. The survey did not include patients with acute cerebrovascular accident, diabetes mellitus, COPD and complex heart rhythm disturbances.

### Results.

In all 69 patients examined by us, were concomitant hypertension stage III. In the 1st group of patients, the percentage of arterial hypertension (AH) of I degree was 22.7%, II degree 36.4%, III degree 40.9% of patients. And in the second group, patients with 1st degree of AH accounted for 40%, with 2nd degree 36%, and with 3rd degree of AH 24%, respectively. In patients of group 1, with a history of COVID-19, HD III stage and AH of 3 degrees predominated, and in the second group - AH of 1 degree.

The first group consisted of 21 patients (47.7%) with CHF II FC and 23 patients (52.3%) with CHF III FC. The second group consisted of 13 patients (52%) of FC II and 12 patients (48%) with CHF of FC III.

Patients of groups 1 and 2 also suffered from diabetes mellitus 19 (43.5%) and 10 (40%) patients, respectively. The patients were comparable in age and concomitant diseases.

When analyzing the body mass index (BMI) in the patients examined by us, patients with concomitant obesity predominated in the first group and amounted to 26 patients (59.1%) and 10 patients (40%) of the second group, respectively. The percentage of obesity depending on the degree in the 1st group of patients was: I degree of obesity 46.1%, II degree -30.8%, III degree -23.1% of patients. In the second group - I degree of obesity 50%, II degree -30%, III degree -20% patients, respectively. We did not find any difference in the degree of obesity in the examined groups (Table 1).

In patients with a history of COVID-19, when analyzing complaints, 23 (52.3%) patients showed progression of dyspnea, which they associated with a previous coronavirus infection. Palpitations, interruptions in the work of the heart disturbed 5 (11.4%) patients

Table 1.

**General characteristics of the examined patients**

indicator	1-group n=44 (M±m)	2-group n=25 (M±m)
Age, years	64.2±9.3	67.0±11.1
Men/Women	24/20 (54.5%/45.5%)	13/12 (52%/48%)
IHD. Stable exertional angina	44 (100%)	25 (100%)
Essencial hypertension	44 (100%)	25 (100%)
Arterial hypertension		
I degree	10 (22.73%)	10 (40%)
II degree	16 (36.36%)	9 (36%)
III degree	18 (40.91%)	6 (24%)
Type 2 diabetes	19 (43.5%)	10 (40%)
BMI, kg/m <sup>2</sup>	32.1±0.78	28.1±1.94
Overweight	16 (36.7)	10 (40%)
Obesity	26 (59.1%)	10 (40%)
I degree	12 (46.1%)	5 (50%)
II degree	8(30.8%)	3 (30%)
III degree	6(23.1)	2 (20%)
CHF		
FC I by NYHA	-	-
FC II by NYHA	21 (47.73%)	13(52%)
FC III by NYHA	23(52.23%)	12(48%)
FC IV by NYHA	-	-
Total patients	44	25

who had a mild disease, 18 (41%) patients with an average degree. After suffering a coronavirus infection, 38 (86.3%) patients began to feel worse. In addition, patients had complaints that did not bother before COVID-19: unreasonable sweating (19 patients, 43.2%), increased hair loss (9 patients, 20.4%), a sharp deterioration in memory (27 patients, 61, 4%). During pulse oximetry, saturation indicators were on average within 96.6%. The frequency of respiratory movements per minute averaged 20 per minute.

Of particular interest was the study of tolerance to physical activity, depending on the undergone COVID-19. 6MWT indicators significantly differed both in patients with CHF FC II and FC III, depending on the undergone COVID-19. So, in patients of the 1st group with CHF FC II, the 6MWT was 321.6 ± 5.7 meters, while in patients with FC III, the 6MWT indicators were reduced and

amounted to  $177 \pm 5.4$  meters. In patients of the 2nd group with CHF FC II, the TNR was  $402.1 \pm 2.5$  m, in patients with FC III, the 6MWT indicators were reduced and amounted to  $232 \pm 5.2$  m..

When studying the clinical condition of patients according to SHOKS, modified by Mareev V.Yu. (2000) revealed a significant difference in the severity of the clinical condition, depending on the history of COVID-19.

In patients with a history of COVID-19 with CHF FC II, SHOKS exceeded by 21.1% ( $p < 0.001$ ) the values of patients in group 2 (Table 2). In patients with FC III, this difference was 16.3% ( $p < 0.001$ ), compared with patients who did not have a history of coronavirus infection, which indicates more severe clinical symptoms in patients with post-COVID syndrome.

Table 2.

The dynamics of changes in exercise tolerance, clinical condition and quality of life in patients with CHF FC II and III (M $\pm$ m)

Indicators	Patient groups	1 group	2 group	R
TSHH (in meters)	FC II	n=21; 321.6 $\pm$ 5.7	n=13; 402.1 $\pm$ 2.5	<0.001
	FC III	n=23; 177 $\pm$ 5.4	n=12; 232 $\pm$ 5.2	<0.001
SHOKS (in points)	FC II	n=21; 5.7 $\pm$ 0.13	n=13; 4.5 $\pm$ 0.19	<0.001
	FC III	n=23; 8.6 $\pm$ 0.16	n=12; 7.2 $\pm$ 0.14	<0.001
Quality of life (in points)	FC II	n=21; 67.9 $\pm$ 0.27	n=13; 61.8 $\pm$ 0.33	<0.001
	FC III	n=23; 74.2 $\pm$ 0.97	n=12; 65.3 $\pm$ 0.94	<0.001

A difference was also found in the indicators of the total index of quality of life. In patients of group 1 with CHF FC II, the QOL index was worse by 9% ( $p < 0.001$ ) from that of patients in group 2. In the group with III FC, the total index of quality of life exceeded by 12% ( $p < 0.001$ ), respectively.

**Discussion.** Long-term COVID, or PCS, is a multisystem disease in survivors of COVID-19 who develop symptoms 12 weeks or more after diagnosis. PCS develops regardless of the initial severity of the disease and age and lasts from several weeks to months. PCS is accompanied by a wide range of recurrent symptoms that vary in intensity and duration and do not necessarily appear in parallel or sequentially [4,5]. The UK National Institute for Health and Care Excellence (NICE), the Scottish Intercollegiate Recommendation Network and the Royal College of General Practitioners have developed the COVID-19 Quick Guide: Managing the Long-term Consequences, which defines PCS as a set of signs and

symptoms that developed during or after COVID-19, 19, and lasting more than 12 weeks, which cannot be associated with alternative diagnoses [6]. Myocarditis, pericarditis, HF, MI, arrhythmias, and pulmonary embolism may develop several weeks after acute COVID-19 and are more common in patients with pre-existing CVD [7]. According to magnetic resonance imaging of the heart, 60% of recovered patients, regardless of the severity of COVID-19, were diagnosed with myocardial inflammation [8]. Mechanisms that determine cardiovascular complications in PCD include direct viral invasion, dysregulation in the ACE/ACE2 system, and a chronic inflammatory response affecting the structural integrity of the myocardium, pericardium, and cardiac conduction system. In recovered patients, cardiometabolic demand may increase, as noted in long-term follow-up of SARS survivors. This may be due to a decrease in the energy reserve of cardiomyocytes, the use of corticosteroids, and dysregulation of the RAAS. Fibrosis or scarring of the myocardium, as well as cardiomyopathy caused by a viral infection, can lead to recurrent arrhythmias. COVID-19 may also provoke the development of arrhythmias due to an increased catecholaminergic state, as well as increased blood levels of pro-inflammatory cytokines such as IL-6, IL-1 and TNF- $\alpha$ , which can alter the action potentials of cardiomyocytes by modulating the expression of ion channels of cardiomyocytes [9]. A decrease in vagal control, a disturbance of myocardial metabolism, its increased rigidity contribute to the occurrence of arrhythmia and cardiac conduction disturbances, in the origin of which the use of toxic drugs for the treatment can play an important role, such as hydroxychloroquine, antiviral agents, some antibiotics that cause prolongation of the QT interval with the development of ventricular arrhythmias [10, 11, 12]. In our study, according to HM ECG data in patients who underwent COVID-19, cardiac arrhythmias and conduction disorders were most often represented by supraventricular extrasystole - in 38.7% of patients, ventricular extrasystole in 34.1% of patients, atrial fibrillation - in 9.1% of patients. Also in this group of patients, 16% of patients had sinus tachycardia. In addition, in group 1, transient atrioventricular blockade of the 1st degree was noted in 13% of patients. In 11.4% of patients, there was a decrease in the circadian index.

Coronavirus infection can also 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% survivors [13].

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, RAAS dysregulation, and glucocorticoid therapy. [14].

According to a systematic review by E. Yonas, I. Alwi et al. (2021), patients with HF have an increased risk of hospitalizations for any reason and adverse outcomes and death from COVID-19 [15]. At the same time, even those who recovered from COVID-19 and did not have HF subsequently demonstrated a higher risk of its development [16].

In our study, when studying the clinical condition of patients according to SHOKS, modified by Mareev V.Yu. (2000) revealed a significant difference in the severity of the clinical condition, depending on the past history of COVID-19.

In patients with a history of COVID-19 with CHF FC II SHOKS exceeded by 21.1% ( $p < 0.001$ ) from the values of patients of the 2nd group. In patients with FC III, this difference was 16.3% ( $p < 0.001$ ), compared with patients who did not have a history of coronavirus infection, which indicates more severe clinical symptoms in patients with post-COVID syndrome.

A difference was also found in the indicators of the total index of quality of life, in patients of the 1st group with CHF FC II, the QOL index was worse by 9% ( $p < 0.001$ ) from that of the patients of the 2nd group. In the group with III FC, the total index of quality of life exceeded by 12% ( $p < 0.001$ ), respectively, which indicates a more severe course of CHF in patients who underwent COVID-19.

### **Conclusion**

In patients with IHD, angina pectoris, FC II-III, complicated by CHF FC II-III in the post-COVID period, various rhythm and conduction disturbances of the heart are more often observed compared to the control group. Also in this group, there is a more severe clinical condition of patients, as well as lower indicators of quality of life. Taking into account these data, for the timely diagnosis of post-COVID syndrome, all patients after suffering COVID-19 are recommended to undergo EchoCG, HM ECG, assessment of the clinical condition and quality of life for the timely treatment and rehabilitation of patients with CHF.

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