SPECIFIC COURSE OF CORONAVIRUS INFECTION IN DIFFERENT HEMODYNAMIC PHENOTYPES OF CHRONIC HEART FAILURE

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Relevance. Despite the numerous scientific researches devoted to cardiovascular diseases, the implementation of new methods of their prevention, the implementation of new methods of diagnosis and treatment, the death and disability caused by these diseases is not decreasing and it remains a medical and social problem today (Ageeva L.I. 2017). Chronic heart failure is not only a medical problem, but also a social problem due to the wide spread, high mortality rate and high costs of treatment of patients with chronic heart failure (Gardner R.S. 2014). Epidemiological studies worldwide have shown an increase in the number of patients with chronic heart failure in recent decades. In the United States, approximately 5.7 million patients are diagnosed with congestive heart failure, and according to disappointing forecasts, this figure will increase to 8 million (Sanchis L. 2018). In the Russian Federation, 880,000 to 986,000 patients with chronic heart failure die in one year (Fomin IV 2010). It is known from numerous studies and published literature in a number of countries that the scientific work of many scientists is dedicated to the study of the pathogenic mechanisms of CHF and the optimization of treatment measures (Maggioni A.P. et al., 2014; Ruppar T.M. et al., 2016; Belenkov Yu.N., 2015; Arutyunov G.P., 2016; Mareev Yu.V. et al., 2017; Tereshchenko S.P. et al. .,2017). In the classifications given in the clinical recommendations of 2016, patients with congestive heart failure were also divided into the group of patients with congestive heart failure with intermediate ejection volume according to the size of the left ventricular ejection fraction. In this case, the pathogenesis of chronic heart failure in patients with reduced left ventricular ejection volume and preserved ejection volume differs and has its own characteristics (Savarese, G., 2018). Based on the above information, it is important in modern medicine to improve the methods of studying, evaluating and treating chronic heart failure not only in cardiovascular diseases, but also in patients with Covid-19 in pandemic conditions.

Aim. It consists in evaluating the specific course of coronovirus infection in patients with different hemodynamic phenotypes of chronic heart failure

Materials and methods. 80 patients with chronic heart failure were selected, who are being treated at the Central Clinical Hospital No. 2 under the Administration of the President of the Republic of Uzbekistan. Fibrosis markers in venous blood - aldosterone, c-reactive protein, d-dimer, Echocardiography, clinical status assessment scale and quality of life questionnaire.

Results. From the information given above, it is known that Covid-19 causes functional and organic changes in many organs, including cardiovascular organs. These changes do not affect the general condition of the body. Based on the retrospective examination of patients with chronic heart failure and acute period of coronavirus infection (n=80), it was found that the main group of patients with severe disease this CHF consisted of patients with reduced CHFrEF (n=15) and patients with preserved form of CHF (n=10). The group of patients who had a mildly -reduced CHFmrEF (n=20), and the group with mild coronavirus infection was made up of the patients who had the CHF. We analyzed C-reactive protein, which is considered a mediator of inflammation, in all patients. From the obtained results, it was known that the highest value of inflammatory mediator C-reactive protein was recorded in patients with CHFrEF and CHFmrEF (C-reactive protein-15.5± 4.13 mg/l, n=18) and (C reactive protein-12.6 ± 3.2 mg/l, n=35) respectively, and in the CHFpEF, C reactive protein -7.4 ± 1.3 mg/l (n=270) was obtained (Fig. 11). From these results, it became clear that the CHFrEF and the inflammatory process of the coronavirus infection in the CHFmrEF was determined to be strong.

We examined the results of analysis of D dimer protein, which is considered important for us (to assess the patient's susceptibility to thromboembolic complications) in all patients (n=80). From the obtained results, it was found out that patients with CHFrEF and CHFmrEF of coronavirus infection have a high tendency to thromboembolic state, D dimer - 0.71± 0.045 μg FEU/ml and 0.57± 0.065 μg FEU/ ml respectively

CHF types by LVEF	**CHFprEF n= 27**	**CHFmrEF n=35**	**CHFrEF n=18**
C reactiv protein(мг/л)	7,4 ± 1.3	12,6± 3,2	15,5± 4,13
D-dimer (мкг FEU/мл)	0,42± 0,053	0,57± 0,065	0,71± 0,045

Figure-1. **C-reactive protein and d-dimer mean values in patients with coronavirus infection in different hemodynamic phenotypes of chronic heart failure.**

Therefore, it was found that it is very necessary to analyze the anticoagulant therapy and monitor the dynamics in the treatment process in such patients. Aldosterone is considered the main proinflammatory mediator in the development of CHF. It is known that this mediator causes cardiac remodeling by increasing fibrosis in the heart. When we analyzed the results of aldosterone analysis in all patients, it became clear that the CHFrEF and the amount of aldosterone increased by 22.5% (n=18) and 36.25% (n=35) compared to the norm when infected with the CHFmrEF of

coronavirus. An increase in aldosterone was observed in 8.75% (n=27) of patients who are CHFprE

Figure-2. **Indicators of increased aldosterone level in patients with coronavirus infection in different hemodynamic phenotypes of chronic heart failure**

CHFprEF n=27	CHFmrEF n=35	CHFrEF n=18
8,75% ↑	36,25% ↑	22,5% ↑

Conclusions.

1. The study of the course of coronavirus infection in patients with different hemodynamic phenotypes of chronic heart failure showed that the severity of coronavirus infection is different in different hemodynamic phenotypes of CHF and the infection is severe in the reduced and mildly-reduced forms of CHF n= 15 and n = 20, respectively, it was found that 43.75% of patients had severe coronavirus infection. This indicator is almost half of the patients taken for examination. Therefore, it is known that the passing of this infection is not only due to the infection, but also the causes of the patient's history of CHFrEF and CHFmrEF of pulmonary hypertension and decreased perfusion in the periphery make the passing of these two diseases difficult.

2. Based on the obtained laboratory analysis results, the inflammatory process is clearly expressed and the thromboembolism prone group is observed in the reduced and mildly-reduced form of CHF. As a result, we can observe a proportional and parallel increase in general blood analysis, biochemical and aldosterone levels in patients. This is also a proof of the different course of coronavirus infection in different hemodynamic phenotypes of CHF

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