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Important Aspects of the Treatment of Chronic Heart Failure

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Relevance of the topic. Chronic HF is a disease with a complex of characteristic symptoms (shortness of breath, fatigue and decreased physical activity, edema, etc.) associated with inadequate perfusion of organs and tissues at rest or during exercise and often with fluid retention in the body. Decompensation of CHF often leads to death, which becomes a socio-economic problem for the state. The problem of preventing decompensation and creating the basis for a stable course of CHF is one of the main tasks of the cardiological medical community [4]. Although there have been many advances and achievements in medicine in the treatment of cardiovascular diseases, the prevalence of CHF is steadily increasing, ranging from 1.5 to 2.0% in the general population, and among people over 65 years of age it reaches 6-17%. Among the causes of disability and mortality of the population, CHF occupies a high level. More than 70% of men and 63% of women with CHF die within 6 years after the first clinical manifestations of the disease. According to some researchers, CHF will become the main problem of cardiology that society will have to face in the next 50 years [1, 7]. CHF is a pathophysiological syndrome in which, as a result of one or another disease of the cardiovascular system or under the influence of other etiological causes, there is a violation of the ability of the heart to fill or empty, accompanied by an imbalance of neurohumoral systems (RAAS, sympatho-adrenal system, natriuretic peptide system, kinin -kallikrein system) with the development of vasoconstriction and fluid retention, which leads to further dysfunction of the heart (remodeling) and other target organs (proliferation), as well as to a mismatch between the provision of organs and tissues of the body with blood and oxygen with their metabolic needs.

Keywords: Heart, Treatment, symptoms.

Currently, the main etiological cause of the development of CHF is considered to be coronary artery disease. According to the Framingham study (USA), 54% of patients with CHF have coronary artery disease. However, epidemiological studies of recent years indicate a significant contribution of dilated cardiomyopathy (DCM) to the development of CHF. According to the results of the international study Euro Heart Survey Study (Cleland, 2001), DCM is the cause of CHF in 11% of patients and ranks 3rd after IHD and valvular heart disease [4,3].

The imbalance of neurohumoral systems in the pathogenesis of heart failure (HF) plays an important role, including in the prevalence of vasoconstrictor and antidiuretic effects, as well as proliferative systems, of which the RAAS plays the most important role, and in the relaxation of vasodilating systems: nitric oxide, bradykinin, prostacyclin, natriuretic peptide. As a result, myocardial hypertrophy, remodeling of the heart and blood vessels, systolic and diastolic dysfunction develop [5]. Despite the unity of the pathophysiological processes that underlie HF of any etiology, the mechanisms of CHF development in IHD patients have their own specifics, consisting in such irreversible changes as postinfarction scar,

persistent ischemia, stunned and hibernating myocardium [2,8]. Activation of the RAAS, especially tissue, is one of the key links underlying the progression of HF of any etiology [5].

Due to the low frequency of use of a combination of essential drugs in the presence of CHF, their insufficient dose, frequent interruption of basic treatment leads to a very high overall mortality in patients of any functional class of CHF. ACE inhibitors are able to act on all links in the pathogenesis of heart failure, primarily due to their blocking effect on the RAAS (circulating and tissue). ACE inhibitors in the maximum tolerated doses are used in all patients with CHF I–IV FC and with an LV EF of 85 mmHg. Art. and leads to an increased risk of death in patients with CHF (class recommendations Ia level of evidence A). The effectiveness of ACE inhibitors was studied in a variety of clinical groups, including in patients with myocardial infarction. However, in most cases, the study population included patients with acute myocardial infarction (AMI) and systolic dysfunction, while the effectiveness of this group of drugs in relation to late post-infarction remodeling with diastolic dysfunction has been little studied [7,1].

The aim of the study was to study the effect of the ACE inhibitor perindopril on general hemodynamics in patients with CHF.

Materials and methods: The study was conducted in the cardiology department of the clinic of the Samarkand Medical Institute. We examined 75 [men - 43 (57.3%), women - 32 (42.7%)] patients with CHF in combination with other concomitant diseases. All surveyed conducted: questioning and examination; general clinical and biochemical studies, ECG, echocardiography (EchoCG). The study included patients with an LV ejection fraction of less than 50%. The patients were divided into 2 groups. The first group (control group) included 34 patients who received only basic therapy (beta-blockers, metabolic drugs, anticoagulants, antianginal drugs). The second group included 41 patients (men - 26 (63%), women - 15 (37%)), who, along with basic therapy, additionally received perindopril at a dose of 4 mg 2 times a day for 3 months. Of these, 20 (48.7%) had coronary artery disease, 15 (36.6%) had hypertension, 2 (5%) had CRHD, 7 (17%) had myocarditis, and 3 (7.3%) had DCM.), with chronic bronchitis - 5 (12.2%) and with diabetes mellitus - 15 (36.6%). The mean age of the patients was 58 ± 1.72 and 61 ± 1.85 years, respectively. the control group consisted of 34 patients (men - 20 (58.8%), women - 14 (41.2%). Of these, patients with coronary artery disease accounted for 18 (53%), DCM - 1 (2.9%), with chronic bronchitis - 1 (2.9%) and with congenital heart disease - 1 (2.9%) In both groups, these clinical parameters did not differ significantly. the criterion for evaluating the effectiveness of the treatment in patients with CHF. EchoCG study was carried out before and after therapy. EchoCG study studied the following indicators: end systolic size (ESD), end diastolic size (EDD), end diastolic volume (EDV), end systolic volume (ESD), stroke volume (SV), left ventricular ejection fraction (LVEF), left ventricular myocardial mass (LVMI), left ventricular myocardial mass index (iMMLV).

Results. When synthesizing changes in the signs of heart failure against the background of ongoing therapy, it was found that in both groups there was a significant decrease in the functional class in the examined patients. After treatment, in patients of the 1st group, FC decreased by 30% (out of 34 patients in 6 cases from FC III switched to FC II), and in patients of the 2nd group, FC decreased by 53% (out of 28 patients in 12 cases, it switched from FC III FC to II FC). When comparing the average values of the FC of patients between the 1st and 2nd groups after three months of therapy, it was revealed that in the 2nd group of patients the average FC was less by 32%. None of the 75 patients included in the study during the three-month therapy showed a deterioration in the general condition.

When analyzing biochemical parameters, cholesterol in the control group before treatment was 6.8 ± 0.5 mmol/l, and after treatment - 6.3 ± 0.5 mmol/l. In the second group (those receiving additional perindopril cholesterol before treatment was 6.6 ± 0.7 mmol / l, and after treatment - 6.0 ± 0.2 mmol / l. When analyzing EchoCG parameters in the control group before treatment, the EDV was 159 ± 1.84 mm/m2,

and after treatment - 154.4 ± 1.58 mm/m² l; CSD before treatment - 86.84 ± 5.11 mm/m², and after therapy - 78.67 ± 2.28 mm/m² EF at the beginning of treatment – $45 \pm 0.62\%$, after treatment – $50.2 \pm 1.26\%$ VR – before treatment was 63.57 ± 4.33 , and after treatment – 72.72 ± 2.48 ml In group 2 before treatment: EDV was 146 ± 2.35 mm/m², and after treatment - 114.03 ± 5.32 mm/m²; EDV before treatment was 84.65 ± 1.64 mm/m² , after - 52.82 ± 2.18 mm/m², LVEF - before treatment - $43.6 \pm 1.45\%$, after treatment - $56 \pm 2.25\%$. and after treatment - 71.88 ± 3.18 ml.

In conclusion, in both groups of patients with CHF after treatment, there was an improvement in the general condition of patients, a decrease in FC, a decrease in the level of total cholesterol, EchoCG parameters. Especially, these values were most pronounced in the second group of patients who received perindopril in addition to therapy ($p \leq 0.05$).

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