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ASPECTS OF THE TREATMENT OF CHRONIC HEART FAILURE

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Abstract
For several decades, chronic heart failure (CHF) has remained one of the most pressing problems in the developed countries of the world. Despite the great successes and achievements of 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%. CHF is characterized by a high level of disability and mortality of the population. 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].

Introduction

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

An important role in the pathogenesis of heart failure (HF) is played by an imbalance of neurohumoral systems, consisting in the predominance of the effects of vasoconstrictor, antidiuretic, proliferative systems, of which the most important role is played by RAAS, and in the weakening of vasodilating systems: nitric oxide, bradykinin, prostacyclin, natriuretic peptide. This eventually leads to the development of myocardial hypertrophy, remodeling of the heart and blood vessels, systolic and diastolic dysfunction [5].

Despite the common pathophysiological processes underlying HF of any etiology, the mechanisms of CHF development in patients with coronary heart disease have their own specifics, consisting in such irreversible changes as postinfarction scar, persistent ischemia, stunned and hibernating myocardium [2,8]. To date, much less is known about the features of the development of CHF in DCMP. DCMP is based on changes at the molecular and gene levels, leading to impaired synthesis of contractile proteins, activation of cardiomyocyte apoptosis and autoimmune processes. DCMP is characterized by the development of pronounced systolic myocardial dysfunction and the formation of maladaptive LV remodeling. Activation of RAAS, especially tissue activation, is one of the key links underlying the progression of HF of any etiology [5].

ACE inhibitors are able to act on all links in the pathogenesis of HF, primarily due to their blocking effect on the RAAS (circulating and tissue). The effectiveness of ace inhibitors has been studied

in various clinical groups, including in patients who have suffered a myocardial infarction. However, in most cases, the contingent included in the study consisted of patients with acute myocardial infarction (AMI) and systolic dysfunction, while the effectiveness of this group of drugs in relation to late postinfarction remodeling with diastolic dysfunction has been little studied [7,1].

The aim of the study was to study the effect of the ACE inhibitor zofenopril 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. 52 [men – 23 (44.2%), women – 29 (55.7%)] patients with CHF in combination with other concomitant diseases were examined. All patients were examined: a survey and examination; general clinical and biochemical studies, ECG, echocardiography (EchoCG). The study included patients with a LV ejection fraction of less than 50%. The patients were divided into 2 groups. The first group (control group) included 24 patients who received only basic therapy (beta-blockers, metabolic drugs, anticoagulants, antianginal drugs). The second group included 28 patients who, along with basic therapy, additionally received zocardis at a dose of 30 mg 2 times a day for 3 months.

The average age of the patients was 58±1.72 and 61±1.85 years, respectively. The control group included 24 patients (men – 11 (%), women - 13 (%). Of these, patients with coronary heart disease were 11 (46%), with GB -5 (21%), with HRBS - 3 (13%), with pneumonia -2 (8%), with DCMP - 1 (4.1%), with chronic bronchitis-1 (4.1%) and with congenital heart defects-1(4.1%). The second group included 28 patients (men – 16 (57%), women - 12 (42%)) who, along with basic therapy, received zocardis at a dose of 30 mg 2 times a day for 3 months. Of these, 12 (43%) with coronary heart disease, 5 (18%) with GB, 4 (14.2%) with CFS, 2 (7.1%) with pneumonia, -2 (7.1%) with DCMP, 1 (3.5%) with chronic bronchitis, and 1 with congenital heart defects (3.5%) and with diabetes mellitus- 1 (3.5%). These basic clinical indicators did not differ significantly in both groups. The effectiveness of the therapy was evaluated by LV remodeling in patients with CHF in combination with other diseases. The structural and functional state of the left ventricle was studied using a MindrayDC-7 echocardiograph using a standard technique. Echocardiography was performed before and after treatment. The following parameters were evaluated: final systolic size (CSR), final diastolic size (CDR), final diastolic volume (CDR), final systolic volume (CSR), stroke volume (UO), left ventricular ejection fraction (LVEF).

Results

When analyzing the dynamics of HF symptoms against the background of ongoing therapy, it was revealed that in both groups there was a significant decrease in the functional class in the studied patients. Thus, in patients of group 1, FC decreased by 25% after treatment (out of 24 patients, in 6 cases, FC III switched to FC II) and in patients of group 2, FC decreased by 43% (out of 28 patients, in 12 cases, FC III switched to FC II). When comparing the average FC values of patients between groups 1 and 2 after three months of therapy, it was found that in group 2 of patients, the average FC was 21.7% lower (p=0.005). None of the 52 patients included in the study showed any

deterioration in their general condition during the three-month therapy, and all patients successfully completed the study program.

In the analysis of biochemical parameters in the control group, cholesterol was 6.8±0.5 mmol/l before treatment, and 6.3±0.5 mmol/l after treatment. In the second group (who received additional zocardis before treatment, cholesterol was 6.6±0.7 mmol/l, and after treatment - 6.0± 0.2 mmol/L. When analyzing echocardiography parameters in the control group before treatment, CD was 159 ± 1.84 mm/m2, and after treatment - 154.4± 1.58 mm/m2 l; CSR before treatment – 86.84 ±5.11 mm/m2, and after treatment - 78.67±2.28 mm/m2; LVL before treatment -45±0.62%, after treatment – 50.2 ± 1.26%. The dose before treatment was 63.57 ± 4.33 , and after treatment – 72.72 ± 2.48 ml . In the 2nd group before treatment. The BWF was - 146 ± 2.35mm/m2, and after treatment - 114.03± 5.32 mm/m2; the CVD of treatment – 84.65 ±1.64 mm/m2, after – 52.82± 2.18 mm/m2; LVL - before treatment – 43.6± 1.45%, after treatment – 56 ±2.25%. after treatment –58.74 ± 1.43 , and after treatment - 71.88 ± 3.18 ml .

Thus, after treatment, in both groups of patients with CHF, there was a tendency to normalize cholesterol, decrease in FC, echocardiography, and clinical improvement in the general condition of patients. These indicators were most pronounced in the second group of patients who additionally included zocardis in therapy ($p<0.05$).

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