addition, patients with high levels of kinesiophobia had more complications during their hospital treatment, including signs of heart failure ($p<0,05$) and such kind of arrhythmia as atrial fibrillation ($p<0,05$). The presence of kinesiophobia and the fear associated with physical rehabilitation potentially might interfere with successful cardiac rehabilitation. Further research should expand this information and develop optimal treatment interventions for patients with the high level of kinesiophobia and the main goal of increasing physical activity and exercise.

The exercise program is well tolerated and can be used as an alternative to traditional hospital exercise programs. The TSK-SV Heart Scale was assessed as a reliable, valid questionnaire to measure kinesiophobia in patients with coronary heart disease. In patients with cardiovascular disease, kinesiophobia has a multifactorial nature and is much greater in patients with NYHA III, and especially class IV. The impact on kinesiophobia was identified by clinical variables that affected rehabilitation outcomes and prognosis, representing all components of ICF, medical variables, and health-related quality of life in patients with coronary heart disease.

Hulaha O.I.

EPLERENONE USE IN ACUTE MYOCARDIAL INFARCTION

Department of Internal Medicine, Physical Rehabilitation and Sports Medicine
Bukovinian State Medical University

Heart failure (HF) remains an important social problem. The severity of the prognosis of clinically manifest HF is indicated by the fact that approximately half of such patients die within 4 years. An important aspect of research remains the impact on the progression of HF by selecting adequate pathogenetically drug therapy.

In order to identify the influence of markers of HF progression, 121 patients with acute myocardial infarction (AMI), whose average age was 51.5 ± 3.94 years, were examined. All patients received nitrates, β-blockers, angiotensin-converting enzyme inhibitors, anticoagulants, antiplatelets. Patients were divided into two groups: group 1 received basic therapy with the addition of spironolactone at a dose of 25 mg for 25 days; group 2 received basic therapy with the addition of eplerenone at a dose of 25 mg for 28 days. The control group consisted of 15 healthy individuals of the same sex and age. The state of neurohumoral regulation was studied by determining the level of aldosterone and the state of proteolytic activity according to the assessment of azocollagen (by lysis of high molecular weight proteins).

We found that before treatment, the level of aldosterone was 1.6 times higher than in the control (240.58 ± 27.12 vs. 149.36 ± 19.24 pmol / l; $p <0.01$), and the proteolytic activity of azocollagen before treatment was almost 3.5 times lower than in the control (0.010 ± 0.002 vs. 0.035 ± 0.001 E440 / ml / h; $p <0.01$). After treatment, aldosterone levels decreased significantly in both groups with a greater tendency in the second group, azocollagen proteolysis in both groups increased significantly, but most pronounced in the group of patients receiving aldosterone antagonist eplerenone.

According to U.P Jorde [2019], the use of aldosterone antagonists in AMI leads to a decrease in intramyocardial aldosterone production, a decreasing in the level of type III procollagen, as well as a marker of myocardial dysfunction - brain natriuretic peptide. Development RALES studies have for the first time shown a 30% reduction in the risk of death in patients treated with long-term spironolactone treatment. The results of the EPHESUS clinical trial using eplerenone confirmed the success of the tactics of blocking the effects of aldosterone at the receptor level. Thus, the use of eplerenone in patients with AMI leads to a decrease in the stimulation of myocardial fibroblasts, a decrease in the formation of collagen in a cardiac muscle, improvement in the contractile function of the myocardium.

The inclusion of eplerenone in the complex dictation of patients with AMI and HF contributes to the normalization of the processes of the proteolytic activity of blood plasma and leads to the formation of adequate remodeling of postinfarction myocardium, which determines the further course of clinical manifestations of HF.