MEDICINE AND PHARMACY

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EARLY DIAGNOSTICS OF CARDIOGENIC LIVER DAMAGE IN PATIENTS WITH CHRONIC HEART FAILURE AND ESTIMATION OF THE EFFECTIVENESS OF TREATMENT WITH LIVERIN

Cardiogenic damage to the liver in CHF is associated with decreased perfusion in its blood vessels and venous stasis [1]. During the course of the disease due to systemic hemodynamic disorders and hypoxemia, a number of neurohormones such as renin-angiotensin-aldosterone (RAA) system hormones are activated, in particular aldosterone (Ald), under the influence of which fibroblasts turn into myofibroblasts. Myofibroblasts are highly sensitive to various endo- and exogenous factors, which increase the production of collagen [2]. Aldosterone, as in all organs, intensifies the process of ischemia and hypoxia in the liver, causing the proliferation of fibroblasts, resulting in increased production of collagen. This leads to the development of perivascular and diffuse interstitial fibrosis in the cardiovascular system and target organs [3, 4].

Like aldosterone, lectin is a biologically active substance (lectin) directly involved in the remodeling of systemic hypoxia and inflammation and extracellular

matrix (ETsM) in patients with SYuE, which is directly involved in fibrosis by increasing fibroblast proliferation and collagen production. participates [1]. It is involved in the process of cell adhesion, apoptosis, and angiogenesis in the liver and can stimulate or inhibit cell maturation according to the amount in the ETsM and intracellular area. In addition, aldosterone is involved as an intermediate mediator in the process of induced perivascular fibrosis in combination with G-3 CHF comorbid diseases [2, 4].

The aim of the study: Early diagnosis of liver hemodynamic disorders and fibrosis processes in different hemodynamic phenotypes of CHF and optimization of treatment.

Materials and methods: Changes in serum levels of neurohormones and changes in hepatic hemodynamics under the influence of the medicine Liverin were evaluated on the basis of standard treatment in 123 CHF patients with various degrees of LVEF. The patients were randomly assigned to 2 groups, the 1st consisted of 64 patients and the 2nd group consisted of 59 patients. The patients with the 1st group were prescribed the drug Liverin 600 mg - 2 ml intravenously in addition to the standard treatment of CHF for 7 days, then 2 capsules 3 times a day for 3 months. Patients of the 2nd groups received only standard treatment. Hepatic vascular dopplerography and serum interleukin (IL) -1, -6, and alpha-tumor necrosis factor (TNF-α) and aldosterone and galectin-3 (G-3) were evaluated and compared 3 months after treatment.

Results: In patients of the 1st group, administration of Liverin in addition to standard treatment resulted in a significant decrease in serum levels of IL-1, IL-6 and TNF-α, G-3 and aldosterone ($p < 0.001$), which indicate the activity of systemic hypoxia and fibrosis as well as improvement in hemodynamic parameters, ie an increase in the rate of systolic and diastolic blood flow in the portal vein and hepatic artery, a decrease in hepatic vascular resistance and pulsation index ($p < 0.001$). Although partially positive changes in the clinical and functional status of patients were observed in patients of the 2nd group due to a decrease in the activity of neurohormones on the basis of standard treatment, no statistically significant changes was observed in portal hemodynamics.

Conclusion: In early diagnosis and evaluation of the effectiveness of treatment of hepatic fibrosis in CHF, it is advisable to conduct hepatic dopplerography, to determine and monitor the rate of blood flow in the portal vein and hepatic artery, peripheral resistance and pulsation index. The use of Liverin in addition to standard treatment in the treatment of CHF leads to positive changes in central and portal hemodynamics, significantly reducing the serum levels of IL-1, IL-6 and TNF-α G-3 and aldosterone , indicating the activity of systemic hypoxia and fibrosis processes in the body.

Reference:

1. Ather S, Chan W, Bozkurt B, Aguilar D, Ramasubbu K, Zachariah AA et al. Impact of noncardiac comorbidities on morbidity and mortality in a predominantly male population with heart failure and preserved versus reduced ejection fraction // J Am Coll Cardiol. – 2012. – Vol. 59 (11). P. 998 - 1005. DOI:10.1016/j. jacc. 2011.11.040.
2. Swedberg K, Komajda M, Böhm M, Borer JS, Ford I, Dubost-Brama A et al. Ivabradine and outcomes in chronic heart failure (SHIFT): a randomised placebo-controlled study // The Lancet. - 2010. - Vol. 376 (9744). - P. 875 – 85
3. Torp-Pedersen C, Poole-Wilson PA, Swedberg K, Cleland JGF, Di Lenarda A, Hanrath P et al. Effects of metoprolol and carvedilol on cause-specific mortality and morbidity in patients with chro nic heart failure – COMET // Am Heart J. – 2005. – Vol. 149 (2). – P. 370 - 6.
4. Turakulov R.I., Sayfullayev M.B. Gadayeva N.A. Features of differential diagnosis of anemia of chronic disease and iron deficiency anemia Comorbidities in chronic heart failure // CHALLENGES IN SCIENCE OF NOWADAYS 26-28.11.2020 WASHINGTON, USA P. 1095-1100