

## Changes Observed in Functional Classes of Chronic Heart Failure in Renal Dysfunction

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**Abstract:** Renal fibrosis markers were evaluated in dynamics to study specific changes in the kidneys of patients with different hemodynamic types and functional classes of chronic heart failure with anemia and to evaluate the effectiveness of complex treatment. The renal fibrosis marker TGF- $\beta$ 1 in the blood was  $2591.0 \pm 108.4$  and  $755.0 \pm 18.87$  pg / ml, respectively, in chronic heart failure with anemia and without anemia ( $p < 0.01$ ). This was indicative of a fibrosis process occurring in the kidney. After complex treatments with the addition of iron, the TGF- $\beta$ 1 index decreased by 2.25 times ( $p < 0.01$ ), the clinical condition, quality of life and resistance to physical exertion changed significantly positively.

**Keywords:** chronic heart failure, chronic kidney disease, renal dysfunction, fibrosis markers, cystatin-C, TGF- $\beta$ 1, ferrokinetic indicators, galectin-3, hemodynamic types

**Introduction.** CHF is a disease that is among the leading causes of morbidity and mortality in the world and has significant social and economic significance. Despite advances in the treatment of cardiovascular disease over the past 20 years, this serious complication remains an unresolved clinical problem. According to the epidemiological survey, the prevalence of CHF in the U.S. and European countries ranged from 0.4% to 2%, a significant increase with age, reaching 10% in those over 60 years of age. At the same time, the incidence of syphilis on the planet has been steadily increasing, reaching a level comparable to that of the most dangerous infectious epidemics in terms of scale and speed of spread (1). About 5.8 million people in the United States and 23 million people worldwide are infected with CHF(7). It is known that the development of systemic organ damage in CHF plays an important role in the remodeling of the left ventricle of the heart from its earliest period (16). According to the recommendations of the European Society of Cardiologists (ESC 2016), from 2016, patients with CHF are divided into 3 groups, taking into account hemodynamic disorders. According to the indicators of the left ventricular ejection fraction, its decreased ( $< 40\%$ ), intermediate (40-49%) and preserved ( $\geq 50\%$ ) types are distinguished. The standard composition of pharmacological treatment gives a relatively positive result in patients with a decrease in blood drive fraction. In contrast, almost no positive effect is observed in standard pharmacological treatments, with the exception of nitrates in the CHF where the driving fraction is preserved. Therefore, the phase of the disease leads to negative consequences in almost all cases. Indeed, a number of authors have suggested that left ventricular diastolic filling disorders play a more important role in the pathogenesis of syphilis than systolic dysfunction, depending on the severity of the disease and its consequences. The process of diagnosing diastolic CHF is complex and its pathophysiology has not been fully studied (12; 6; 3; 15; 10).

As noted above, in addition to the prevalence of CHF, it is distinguished from a number of other diseases by its adverse effects and high disability rate [13]. The mean 5-year mortality rate in the population of patients with CHF (IIV FC) was 59% in men and 45% in women, 6–7 times higher than in the general population of the same age (5;8; 18; 37). Because the degree of damage to the myocardium, along with other organs and systems, iecomorbidity, is important in this complication, which determines the fate of patients and the consequences of the disease (15). Among them, anemia

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has a special place and in most cases is accompanied by CHF (11). Anemia not only exacerbates CHF symptoms, but also worsens quality of life by prolonging hospitalization, reduces endurance to physical exertion, and increases the risk of death by 2 or more times (14). It should be noted that there is a weak feedback between hemoglobin and the left ventricular blood drive fraction (28; 35). A number of observations have shown that anemia is an independent risk factor in patients with CHF, in which myocardial oxygen supply is significantly reduced (17; 27). It is known that in addition to anemia, a number of other polymorbid diseases are also detected in patients with CHF. Among them, renal dysfunction plays a leading role not only in the pathogenesis and development of CHF, but also in the development of anemia (26; 29). However, at the same time, such comorbid cases remain poorly understood from a scientific point of view. (36.) The purpose of the study. Evaluation of the effectiveness of antianemic therapy based on standard therapy in renal and cardiac fibrosis processes in patients with different hemodynamic types of chronic heart failure (preserved intermediate and low) with anemia.

**Material and methods.** The 120 patients with CHF involved in the study were divided into 2 groups (75 of whom were anemic and 45 were anemic) and underwent excellent clinical and laboratory examinations. In order to carry out the tasks set before us, 75 patients with CHF anemia were divided into 3 groups (in each group there were 25 left ventricular hemorrhage fractions, intermediate and low ones). They ranged in age from 50 to 70 years and averaged  $64.0 \pm 5.0$ . All patients were followed up in an outpatient setting after treatment in a hospital setting. The clinical classification of the follow-up patients is given in Table 1.

**Table 1 Classification of patients involved in the study**

No	Indicators	I group n = 75	Group II n = 45
Absolutely	%	Absolutely	%
1 .	Male	32	42,7
2 .	Female	43	57,3
3 .	Ischemic heart disease	45	60.0
4 .	Ischemic heart disease post infarction cardiosclerosis	25	33,4

Group I patients were given 200 mg of iron III hydroxide sucrose complex (venofer) intravenously as an antianemic treatment based on the standard treatment of CHF during hospital treatment. The total dose of the drug administered for the treatment of iron deficiency, using a special formula adopted for the treatment of venofer [total iron deficiency = body weight, kg x (150 - patient hemoglobin index Hb, g / l) x 0.24 + 500 mg] calculated. Group II patients were prescribed the generally accepted CHF antitreatment. Patients in both groups received angiotensin-converting enzyme inhibitors or angiotensin receptor antagonists, --Adreno blockers, and mineralocorticoid receptor antagonists (as eplerenone-antifibrosis drugs) as standard treatment. In the patients involved in the study, the diagnosis of CHF and its functional classes were determined on the basis of their complaints, anamnesis, objective examination and laboratory instrumental examinations, as well as criteria of the New York Heart In recent years, galectin-3 has been proven to be a reliable marker of fibrosis in pathological processes in the body and primarily in the heart. However, although this marker has been studied in the CHF, there is no data in the available literature on its change in anemia. In the left ventricular hemorrhage fraction in which we observed, in the intermediate and decreased groups, when they were anemic and without anemia, the galectin-3 values were  $22.5 \pm 1.1$  and  $19.23 \pm 1.1$ ,  $19.55 \pm 1.3$  and  $18.5 \pm 1.5$ ,  $19.02 \pm 1.2$ , and  $13.2 \pm 1.4$ , respectively, in all cases. ng / ml was equal to ( $p < 0.05$ ). At the same time, its indicators were 1.2, 1.1 and 1.4 times higher in different hemodynamic types, respectively, than in those without anemia. It is known that aldosterone is actively involved not only in water-salt metabolism in the body, but also in fibrous processes, which has been proven in numerous studies. In recent years, there have been reports that this hormone is produced not only in the adrenal glands, but also in other internal organs, including the kidneys and heart. Numerous studies have been conducted on its modification under the influence of various FC and a number of drugs. However, data



on aldosterone levels in the blood are insufficient when this severe complication occurs in comorbidity with anemia. In this context, we studied its efficacy in patients with CHF anemia and non-anemia. At the same time, aldosterone was 1.1, 1.1, and 1.2 times ( $p < 0.05$ ) higher reliably in patients with left ventricular hemorrhage, interstitial, decreased, and anemia, respectively, than in those without anemia. The indicators confirm that aldosterone in the blood increases not only due to the presence of hemodynamic types of CHF, but also the presence of anemia, and therefore the increase in fibrous processes.

TGF- $\beta$ 1 plays a leading role in the development of fibrous processes in the body and primarily in kidney tissue. However, there is insufficient data in scientific sources on the alteration of this cytokine when CHF passes with anemia. In patients with left ventricular hemorrhage fraction, intermediate, decreased, and anemia and anemia without follow-up, TGF- $\beta$ 1 values were  $2554.7 \pm 125.4$  and  $2209.4 \pm 122.2$  ( $p < 0.05$ ),  $2832.7 \pm 176.0$ , and  $2194.3 \pm 75.8$  ( $p < 0.05$ ), respectively.  $2332.8 \pm 167.8$  and  $1994.2 \pm 73.1$  pg/ml ( $p < 0.05$ ).

Cytokine levels were 13.5%, 22.5%, and 14.5%, respectively, in the presence of anemia and in the absence of anemia. It is known that in recent years, special attention has been paid to cystatin-C in the assessment of renal function. It has a number of advantages over creatinine. Therefore, we determined cystatin-C levels in the blood of patients in our follow-up and assessed glomerular filtration using it. Cystatin-C levels were 10.1%, 24.6%, and 4.54% higher, respectively, in patients with left ventricular hemorrhage fraction, interstitial, decreased, and anemic groups. It has been shown that the early development of fibrous processes in the kidneys of patients with anemia and the process adversely affects the functional state of the kidneys. Changes, i.e., when anemia as detected in all hemodynamic types, it was reliably reduced by 4.3%, 7.4%, and 20.2%, respectively, ( $p < 0.05$ ) compared to non-anemia.

**Conclusion.** 1. An increase in galectin-3 levels in the blood led to a reliable decrease in the six-minute walking testing all hemodynamic types (preserved, intermediate, decreased) with chronic heart failure anemia.

2. A positive reliable correlation between aldosterone and galectin-3 in anemia in all hemodynamic types of chronic heart failure confirmed the synchronization of fibrous processes in the body.

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