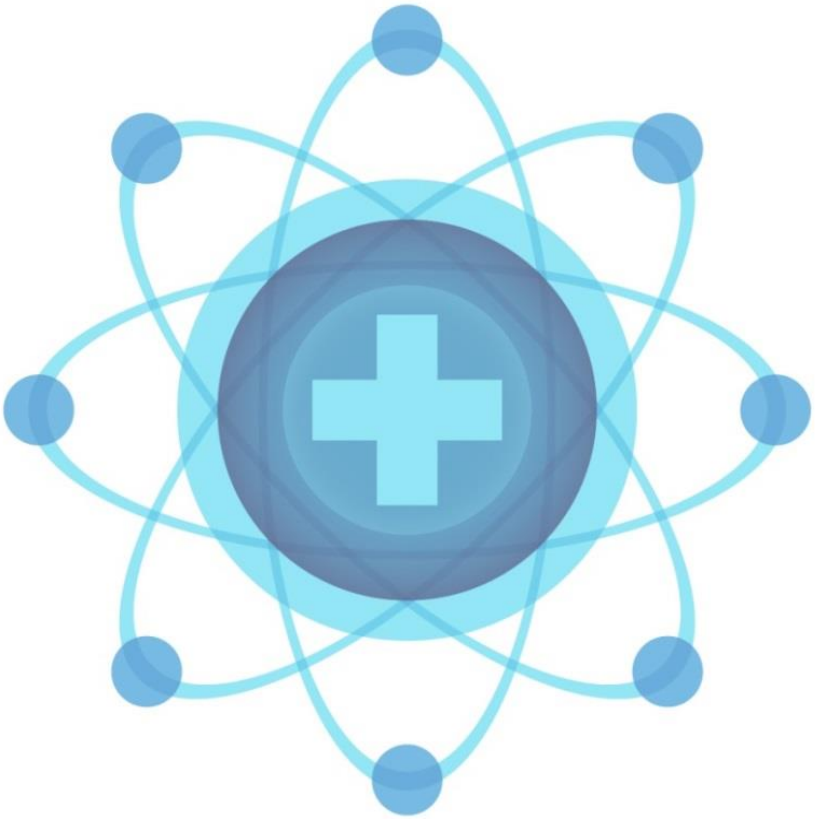

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DETERMINATION OF HEPSIDIN IN PATIENTS WITH CHRONIC HEPATITIS ASSOCIATED WITH CHF AS A PREDICTOR OF THE SEVERITY OF HEPATOCARDIAL SYNDROME

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Abstract. One of the important problems, which is currently receiving much attention, is the combination of heart failure and liver dysfunction, which is currently referred to as hepatocardial or cardiohepatic syndrome. This study is devoted to the study of the relationship between diagnostic parameters of heart and liver failure. Also, the determination of new markers of anemia in hepatocardial syndrome.

Keywords. CHF, chronic hepatitis C, hepcidin, anemia

CHF is one of the most urgent problems of modern health care due to the steady increase in the incidence in all countries of the world, the poor prognosis for patients, and the need for high material costs for their treatment [1].

Over the past few years, small changes have been made to the structure of the etiology of CHF: along with coronary heart disease (CHD) and arterial hypertension (AH), the proportion of postinfarction cardiosclerosis (PICS) has increased to 19.7% and diabetes mellitus (22.7%) [1] . It should be noted that an increase in the proportion of patients with CHF and modification of the leading causes are inextricably linked with an improvement in the quality of medical care, an increase in life expectancy and survival after vascular accidents.

To date, CHF has acquired the character of a pandemic, which has already affected more than 23 million people worldwide. In the United States, the number of patients with CHF is approaching 6 million.

One of the important problems that is currently receiving much attention is the combination of heart failure and liver dysfunction, which is currently referred to as hepatocardial or cardiohepatic syndrome (HPS), defined as an increase in any of the liver parameters with the exclusion of liver damage of another etiology. [3]. It can be diagnosed in 40-85.1% of patients with decompensated heart failure [23].

According to the EFICA study, liver failure develops in 83% of patients with cardiogenic shock and in 51% of patients without cardiogenic shock [24]. The biochemical profile is characterized by a sharp increase in the level of aspartic (AST) and alanine (ALT) transaminases and lactate dehydrogenase (LDH) in serum more than 10-20 norms and the ratio of serum ALT to LDH <1.5 during the first three days after hemodynamic disorders, an increase in the prothrombin index [25].

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Lindvig K. et al. (2012), studying the density of the liver by NEM in 212 patients upon admission to the hospital, concluded that its increase is an independent predictor of the risk of 30-day mortality [4,6].

To date, many studies have been conducted in patients with HF, confirming the association of SPS with a worse prognosis (leads to increased mortality, poor quality of life), a more severe course of the disease (high FC, low LV EF, congestion symptoms, severity of clinical manifestations of HF), impaired renal function, increased brain natriuretic peptide (BNP), impaired intracardiac hemodynamics and remodeling of the right and left parts of the heart, as well as laboratory syndromes of cytolysis, cholestasis and signs of reduced synthetic function (hypoalbuminemia, hypolipidemia).

The purpose of this study is to study the indicators of ferrokinetics, hepcidin levels and indices of liver fibrosis in patients with heart failure.

Materials and methods. We have studied 134 patients with chronic viral hepatitis C. Of these, women 42.9% (n=61), men 51.4% (n=73). The mean age of the patients was 58±2.3 years.

ALT, AST, LDH, GGT, platelet count, INR were determined in all patients. To assess the ferrokinetic parameters, transferrin, ferritin, and hepcidin were determined.

Carrying out standard procedures for examining a cardiological patient (general clinical blood and urine tests, a biochemical blood test, a coagulogram, echocardiography (Echo-KG) with an assessment of intracardiac hemodynamic parameters, electrocardiography (ECG) in 12 leads, ultrasound of the abdominal organs and kidneys, radiography of the chest organs).

An increasing number of researchers are paying special attention to the search for informative non-invasive methods for screening hepatic dysfunction with the development of scales that allow indirect assessment of the severity of histological changes in liver tissue.

To date, IFPs have been developed that are calculated on the basis of clinical data and changes in indicators that directly or indirectly indicate damage to the liver tissue with the formation of fibrosis. The APRI index, the simplest and most accessible for calculation, has not yet been studied in patients with CVD. This formula was developed to identify a high risk group for significant fibrosis and cirrhosis in patients with viral hepatitis.

Hepcidin was tested in 75 patients. Serum hepcidin was determined by enzyme-linked immunosorbent assay (ELISA) according to the principle of competitive binding (ELISA kit, USA). The analysis was carried out in the scientific laboratory of the Bukhara State Medical Institute.

Research results and discussion. Calculation of IFP APRI for patients included in the study was carried out on the basis of the results of general and biochemical blood tests taken on the first day after hospitalization.

The results of calculating the risk of severe liver fibrosis according to the APRI index. In 84 (62.8%) patients, the APRI index did not exceed 0.5, which corresponds to a low risk of severe liver fibrosis, and the "gray zone" was 11.2% of patients (n=15) patients. "Gray zone" - the median values of the indices, for which, according to the definition, it is impossible to speak with confidence about a high or low risk of severe fibrosis or liver density.

A high risk of severe liver fibrosis was identified in 35 (26%) patients.

We have studied a comparative analysis of the parameters of liver fibrosis according to the APRI index and the ejection fraction (EF) of the left ventricle.

In the presence of cardiac arrhythmias, there was a significant scatter in the values of the APRI index. There were no significant differences between the groups (p=0.01).

The main objective of our study was to assess changes in the level of hepcidin at different levels of the decrease in left ventricular EF.

Recently, not only new biochemical markers, which are links in the pathogenesis of CHF, have been actively studied, but their influence on the remodeling of internal organs and the development of multiple organ failure, which determines an unfavorable prognosis of patients, has also been assessed. We studied hepcidin as such a marker. Of the 75 patients with defined hepcidin levels, 52.3% of patients had HF with low EF (HFpEF), 28.4% of patients with intermediate EF (HFpEF), and 19.3% of patients with preserved EF (HFpEF).

The groups did not statistically significantly differ in age, severity of CHF. (p>0.05). According to the results of the analyzes, women (66.6%) predominated among patients in the HFpEF group. In all groups there was a positive relationship between hemoglobin and hepcidin. Also, patients with preserved LV EF were less likely to have anemic laboratory syndrome (Table 1).

Table 1

Clinical characteristics of patients depending on the degree of decrease in LV EF

Parameter	HFpEF1 (n=39)	HFrEF2 (n=21)	HFrEF3 (n=15)
Gender M/F	18/11	12/9	5/10
Age, years	55.8±9.4	62.3±7.05	68.15±11.19
III-IV FC, n (%)	19 (92%)	11 (79.1%)	12 (100%)
Anemia, n (%)	28.4 (%)	41.2 (%)	78.4 (%)

Hepcidin, ng/ml	28.2	20.4	15.3

The results of the study showed that there is a positive relationship between the level of hepcidin and EF, and patients belonging to the first group with HFpEF1 showed median values of this indicator, had the most favorable clinical and laboratory profile (higher levels of hemoglobin, platelets, less often had an increase in ALT and AST , $p>0.05$).

In the CHF patients examined by us, with varying degrees of severity of systolic dysfunction of the left ventricle, various factors influencing the level of hepcidin were identified.

In patients with HFrEF, a significant effect of the presence of cardiac arrhythmias on the level of hepcidin was noted. It is known that the presence of rhythm disturbances further worsens organ perfusion in CHF, aggravating hypoxia.

It is noteworthy that the level of hepcidin in patients with severe LV systolic dysfunction was higher (28.4 ng/ml (95% CI: 22.2-69.6)) than in patients with preserved and intermediate LV EF, in the absence of differences by the frequency of occurrence and severity of anemia. In addition, the level of hepcidin showed a negative relationship with LV EF according to the results of our multivariate regression analysis.

The revealed trend towards an increase in the level of hepcidin with a decrease in LV EF less than 40% in the absence of correlations of its level with other clinical and laboratory data does not nevertheless allow us to unambiguously assess its independent role in the progression of CHF, since the regulation of the level of this indicator in patients with CHF is multifactorial and dependent on various metabolic parameters and comorbid conditions, as a hepatocardial syndrome, which complicates its assessment as a diagnostic and prognostic marker.

Conclusion: From the results of our study, it should be noted that the regulation of hepcidin in patients with CHF is multifactorial, which complicates the assessment of hepcidin levels as a prognostic marker.

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