

Features of Diagnostics of Heart Failure and Refractory Anemia Developed on The Background of Chronic Hepatitis C

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Abstract:

Chronic hepatitis C (HCV-infection) is one of the most pressing medical and social problems in the world, which is due to a wide, consistently high incidence and ubiquitous spread, as well as enormous economic costs for diagnostic and treatment processes.

One of the most important problems requiring study at present is cardiohepatic syndrome (CHS).

Keywords: Chronic hepatitis C, cardiohepatic syndrome, refractory anemia, hepcidin, ferrokinetics.

Introduction

According to the World Health Organization, the number of patients infected with viral hepatitis C in the world is about 210 million people, which is about 3% of the world's population. According to estimates, the possible prevalence of this disease in our country is up to 1.5%. This viral infection acquires a chronic course in 80% of cases, which leads to an almost inevitable increase in pathological processes in the liver, the outcome of which in most cases is liver cirrhosis and (or) hepatocellular carcinoma.

Target scientific research is the study of indicators of ferrokinetics, the level of hepcidin and indexes of liver fibrosis in patients with heart failure.

Materials and research 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).

Results and discussions. 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.

Conclusions. 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.

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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