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ASSESSMENT OF SIGNS OF MYOCARDIAL DYSFUNCTION IN PATIENTS WITH LIVER CYRROSIS

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Annotation
The article describes in detail from etiology to differential diagnosis of portal hypertensive enteropathy. Changes in the ultrastructure of the mucosal epithelium in portal hypertensive bowel disease are mainly characterized by colorectal bleeding. Bleeding is mainly caused by varicose veins of the submucosa, sudden increases in pressure in the portal vein, erosion or ulceration of the mucosal surface, obstruction of blood clotting, and a decrease in the quality or number of platelets.

Keywords: portal hypertensive bowel disease, portal hypertensive enteropathy, bleeding, vascular ectasia, varicose veins.

Introduction

Liver cirrhosis (LC) is characterized by hyperdynamic circulation, which manifests itself as visceral vasodilation and increased cardiac output [1]. These disorders of systemic circulation, combined with high intrahepatic resistance, contribute to the development and progression of portal hypertension (PH) and often represent the main complications of cirrhosis in the form of variceal bleeding, fluid retention and decreased renal blood flow.

However, the possibility of associated specific disorders in the myocardium in cirrhosis has been recognized relatively recently. These include an enlarged left ventricular cavity associated with diastolic dysfunction and systolic exercise incompetence (normal or increased initial ejection fraction without adequate stress gain). The combination of these abnormalities has been termed cirrhotic cardiomyopathy. However, cirrhotic cardiomyopathy syndrome has not yet been finally classified and the mechanisms of development of myocardial dysfunction in cirrhosis are only partially known. However, reports of deaths due to heart failure during liver transplantation, trans jugular intrahepatic portosystemic shunting [3], and surgical portocaval shunts in cirrhotic patients suggest that myocardial dysfunction may progress with sodium retention and increased blood volume. Therefore, early diagnosis and clinical intervention can improve the survival of these patients [4]. Epidemiological studies revealed a relationship between the

severity of heart failure of various etiologies and the content of brain natriuretic peptides (BNP and its precursor, NT-proBNP) in plasma, which made it possible to consider the concentration of these peptides as a "laboratory test" of myocardial dysfunction. Plasma BNP levels can be used as markers of cardiac dysfunction in decompensated cirrhosis, but remain normal or mildly increased in compensated cirrhosis. Carefully designed study by Henriksen JH et al. showed that the increased circulating levels of natriuretic peptides in patients with cirrhosis mainly reflect increased myocardial secretion in the development of dysfunction, rather than decreased hepatic degradation of these peptides. It was found that the level of NT-proBNP better than BNP identified patients with asymptomatic heart disease [6]. Thus, the use of traditional clinical, laboratory and instrumental diagnostic methods for LC does not allow predicting the course of portal hypertension syndrome and the severity of myocardial dysfunction.

In a study conducted with S.A. Pribylov. 45 patients with LC of class B-C according to Child with portal hypertension syndrome (age 49 ± 2, 14 women) were included who were on a diet with restriction of sodium chloride to 1 g / day and liquid to 1.5 l / day for 7 days. Diet restriction was chosen because studies associated with additional volume overload showed the appearance and aggravation of myocardial dysfunction in some patients with compensated cirrhosis without ascites [7]. Alcoholic etiology of CP was found in 12 patients without ascites and 22 with ascites, but they abstained from alcohol for at least 6 months before the study, which was confirmed by questionnaires, interviews with relatives and the level of glutamyl transpeptidase. Cirrhosis was associated with chronic hepatitis C in 7 and hepatitis B in 2 patients , 2 patients had cryptogenic CP.

In accordance with clinical (ascites, esophageal varicose veins, splenomegaly, etc.) and ultrasound (reduction of blood flow in the portal vein and detection of portosystemic anastomoses with color Doppler mapping) criteria for portal hypertension syndrome, patients were divided into 3 groups: 1 group consisted of 16 patients with instrumental confirmation PG syndrome, group 2 - 12 patients with "small" ascites, group 3 - 17 patients with "large" and "medium" ascites, the presence of peripheral edema in 13 patients (76.4%), hepatic encephalopathy grade I-II. characteristics of Doppler sonographic indicators of blood flow by groups showed: in comparison with group 1, an increase in the diameter of the portal vein in group 3 of patients with CP (sectional area 1.1 ± 0.3 cm2 versus 1.5 ± 0.2 cm2, p 350 fmol / ml as a prognostic the criterion for the presence of myocardial dysfunction in CP (n = 45) in the 2x2 contingency table the following values of sensitivity are established - 90.9%, specificity - 41.6%, diagnostic parity - 77% ($\chi 2 = 4.3$; $p = 0.03$).

LV systolic function in patients when measuring the ejection fraction at rest was preserved in patients with compensated CP. However, diastolic parameters of LV filling in most patients were pathological and characterized by slow ventricular relaxation and worsening ventricular filling in the early phase (type I diastolic dysfunction). More obvious findings were a borderline decrease in LVEF and a moderate increase in pulmonary artery pressure in patients with decompensated ascites, along with diastolic myocardial dysfunction. The design of the present study minimized the influence of some of the pathophysiological factors of volume overload, since patients did not receive diuretics and the water-salt load remained constant during the observation period. However, abnormal fluid distribution may contribute to masking overt heart failure in patients with cirrhosis. An earlier study of patients with LC found a direct relationship between LV EDV and circulating blood volume, had moderate LV dilatation and increased concentrations of natriuretic peptides were not caused only by central volume overload. There is evidence that chronic alcoholism can cause congestive dilated cardiomyopathy [3] with dilatation LV cavity, increased LV EDV and decreased ejection fraction. However, alcoholic cardiomyopathy is reversible with abstinence from alcohol, and myocardial function is comparable at 6 months to the group of non-alcoholic cirrhosis [4]. Huonker et al. Reported systemic and pulmonary hemodynamics in cirrhotic patients after transjugular intrahepatic shunting. Shunting of visceral blood into the systemic circulation led not only to an increase in the volume of circulating blood, cardiac output, generalized systemic vasodilation, a decrease in systemic vascular resistance, but also to an increase in the mean pressure in the pulmonary artery [6]. Thus, patients showed subclinical left ventricular diastolic function due to cardiomyopathy, including the inability of the heart to fully cope with the increased volume load. Therefore, the detected increased pulmonary artery pressure in patients with cirrhosis may also be a marker of the presence of subclinical cardiac dysfunction [1]. In several studies, the authors found in patients with ascites a significant reduction in the early / late diastolic flow rate (E / A ratio), which is a measure of the atrial contribution to end-diastolic volume. The appearance of diastolic dysfunction, together with dilatation of the LV cavity, suggests that patients do indeed have a structural and functional myocardial anomaly. A careful analysis of patients in the compensated LC groups showed that some patients without ascites were able to increase plasma levels of NT-proBNP. This may explain the contradiction in the literature regarding the levels of natriuretic peptides in LC [7] since not all patients with cirrhosis have manifestations of myocardial dysfunction. Volume overload leading to the activation of neurohormones, including norepinephrine, angiotensin II, and aldosterone, results in myocardial hypertrophy

and fibrosis, i.e. induces structural remodeling with increased collagen accumulation in the interstitium, significantly increases myocardial stiffness and provides the release of natriuretic peptides. The correlations between plasma NT-proBNP concentration and markers of cardiac dysfunction, but not markers of hyperdynamic circulation, suggest that an increase in NT-proBNP levels may be a highly sensitive indicator of preclinical myocardial dysfunction in cirrhosis. Conclusion. Since myocardial dysfunction is predominantly asymptomatic in LC and the clinical study has low sensitivity, plasma NT-proBNP levels can be used as a screening marker for cirrhotic cardiomyopathy [8-12] . Elevated NT-proBNP levels correlate best with diastolic myocardial dysfunction in these patients. A high level of NT-proBNP in plasma suggests the need for more expensive studies (echoDCG, ventriculography, etc.), while a normal or low level of BNP has a high negative predictive value. Thus, an increased plasma concentration of NT-proBNP is a reliable non-invasive marker of early myocardial dysfunction against the background of volume overload of the heart chambers during the progression of portal hypertension in patients with LC [13-15].

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