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Mini Review

Heart Changes in Liver Cirrhosis

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Abstract

The article presents data on the pathogenesis, clinical manifestations, main therapeutic approaches and methods of surgical treatment of cirrhotic cardiomyopathy (CCM).

Keywords: *Cirrhotic Cardiomyopathy; Liver Cirrhosis; Heart Failure; Systolic Dysfunction; Diastolic Dysfunction*

Introduction

The relationship between heart failure (HF) and liver disease has been studied for many years. According to various authors, hepatic dysfunction can be detected in 15 - 65% of patients with heart failure, closely correlating with hemodynamic parameters [1-4]. Associated pathology of heart and liver pathology of the liver due to diseases of the cardiovascular system: Cardiogenic ischemic hepatitis.

Congestive hepatopathy (cardiogenic cirrhosis) Vascular malformations liver pathology of heart, formed on the background of liver diseases: Cirrhotic exudative pericarditis CCMP Systolic heart failure due to intrahepatic arteriovenous fistula Diseases occurring simultaneously with damage to the heart and liver: Metabolic and autoimmune pathology (hemochromatosis, glycogenosis, diabetes mellitus, steatohepatitis, sarcoidosis) and parasitic (schistosomiasis, syphilis, tuberculosis, trypanosomiasis, cysticercosis, mycosis) blood diseases (hemolytic anemia, leukemia, lymphoma, DIC) Chronic alcoholism dense of primary localization pathological process. Previously, it was assumed that patients with CP have a low risk of developing cardiac pathology, and the detected HF is due to the cardiotoxic effect of overt or covert alcohol intake. Subsequently, it was revealed that in patients with non-alcoholic LC in orthostatic position, hemodynamics differs from healthy individuals in the form of diastolic dysfunction of the left ventricular myocardium (LV) [5-7]. Wong F., *et al*. revealed a decrease in exercise tolerance among patients with LC due to LV myocardial hypertrophy, disorders of diastolic, inotropic and chronotropic LV functions. Moreau R., *et al*. found an abnormal long- term pressor effect of vasopressin in patients with LC [7]. In 1989, SS Lee suggested that impaired cardiac response in LC is due not so much to the effects of alcohol as to the disease itself [2,4,7]. In 1996, Z. Ma and SS Lee introduced the term "cirrhotic cardiomyopathy": "violation of the contractile response of the myocardium to stress and/or diastolic relaxation with electrophysiological changes in the absence of any known heart disease" [5,6]. Research in recent decades has shown that impaired myocardial response to stress is observed in LC, regardless of its origin. In 2005, at the World Congress of Gastroen-

terologists in Montreal (Canada), diagnostic criteria for CMMC were first proposed. Most researchers consider the main signs of CCMP to be the presence of systolic and diastolic LV dysfunction, additional - lengthening of the QT interval; decrease in heart rate (HR) per load, electromechanical dyssynchrony; myocardial hypertrophy; an increase in the size of the left atrium; increased blood concentration of troponin I and brain natriuretic peptide. The incidence of CMMP remains unknown. It is not excluded that such signs of CCM as prolongation of the QT interval and sonographic indicators of LV diastolic dysfunction are present in most patients with Childe-Pugh class B and C LC.

Diagnostic criteria for MCMP Signs of systolic dysfunction decrease in ejection fraction less than 55% decrease in the increase in cardiac output in response to exercise, pharmacological stimulation Signs of diastolic dysfunction E/A <1 (depending on age) increase in the time of deceleration of blood flow in the early phase diastole more than 200 ms prolongation of isovolumic relaxation more than 80 ms Additional criteria prolongation of the QT interval change in the expected heart rate for exercise (decrease) electromechanical dyssynchrony myocardial hypertrophy increased concentration of troponin I in the blood increased concentration of brain natriuretic peptide in the blood increased size of the left atrium. Development mechanisms.

The pathogenesis of CCMP is not yet fully understood. Theories are put forward related to dysfunction of B-receptors, the release of cardiodepressive substances - cytokines, nitric oxide (NO), endotoxins. In recent years, a hypothesis has emerged about the production of cardiodepressive substances in patients with LC with intestinal dysbiosis. It is believed that one of the mechanisms responsible for the synthesis of cytokines - tumor necrosis factor alpha, interleukins 1, 6, and NO - is bacterial overgrowth in the small intestine and the presence of opportunistic bacteria in the intestine. With CP, the hyperkinetic type of hemodynamics prevails with a compensatory increase in blood flow under conditions of increased arteriovenous shunting in the liver, water and electrolyte retention occurs, which leads to an increase in portal blood flow and progression of portal hypertension in a feedback manner. The redistribution of the circulating blood volume (BCC) is followed by the decentralization of blood flow and the development of hypovolemia. Reducing the effective bcc (central and arterial volume) along with hypotension promotes activation vasoconstrictor systems (sympathetic and renin-angiotensin-aldosterone), force the hyperdynamic circulation, increased pre railway and afterload [7,8]. Activation of the renin-angiotensin-aldosterone system (RAAS) in CP causes an increase in the reabsorption of water and electrolytes in the kidneys, an increase in the BCC. This leads to the maintenance of increased CO and myocardial overload. Reduced afterload and increased compliance of the arterial wall neutralize cardiac dysfunction, predetermine an increase in heart rate, stroke volume, cardiac index, which, in turn, is accompanied by the clinical debut of the CMF. The mechanisms of development of vasodilation and impaired contractility of cardiomyocytes in LC are investigated. Overproduction of NO [9] and endocannabinoids, as well as activation of intracellular signal transduction pathways through cannabinoid receptors, underlie vasodilation. With systolic myocardial function associated violation of heart rate control; myocardial conduction and repolarization; contractility of cardiomyocytes. Among the putative pathophysiological and biochemical mechanisms, the activation of cannabinoid receptors-1, a decrease in the sensitivity of beta-adrenergic receptors and the content of G-proteins, an increase in the inhibitory effects of hemeoxygenase, carbon monoxide, NO, and tumor necrosis factor alpha are discussed [4-6].

The clinical picture. The clinical signs of CMMP are not specific. Among the most common complaints should be noted cardialgia, feeling short of breath, shortness of breath, palpitations, edema, heart rhythm and conduction disturbances. Rapid fatigue, reduced performance, low exercise tolerance without clear symptoms of LV failure at rest can be regarded as manifestations of the underlying disease. The state of affairs is exacerbated by the lack of evidence. During an objective examination of a patient with this pathology, most authors indicate the need to identify the “stigmas” of hyperdynamic circulation, which include palmar erythema and telangiectasia. High heart rate, poorly responsive to reflex tests (skin cold test, Valsalva test), indicate the development of sympathetic dysfunction [8].

Treatment: It is believed that peripheral vasodilation in LC “protects” these patients from severe HF, and minor systolic and diastolic dysfunction in the absence of any stress does not require treatment. However, factors such as sepsis, surgery, and TIPS can lead to the development of heart failure. In such a situation, general therapeutic approaches are similar to those used for non-cirrhotic heart failure (bed rest, diuretics). Significant peripheral vasodilation in CMF requires limiting the use of drugs that can reduce afterload. First of all, this

concerns angiotensin - converting enzyme (ACE) inhibitors, which in some patients can lead to a significant decrease in blood pressure (BP), acute renal dysfunction. However, there are interesting data regarding the use of lisinopril. Its purpose can not only normalize the daily blood pressure profile, but also somewhat improve the functional state of the liver, reduce the level of pro-inflammatory cytokines, increase insulin sensitivity, and significantly reduce the activity of aminotransferases and bilirubin. There are new data on the direct positive effect of ACE inhibitors on the hepatic parenchyma. In the group of patients with liver pathology who received ACE inhibitors, histological examination revealed a lower degree of fibrosis. These differences may be associated with the direct action of an ACE inhibitor on the local RAAS, which is involved in the regulation of fibrinogenesis and the development of portal hypertension [8,9]. There is no data on the advisability of using cardiac glycosides. It was shown that these drugs do not affect the contractile function of the myocardium in patients with alcoholic LC [2]. For the prevention of bleeding from varicose veins of the esophagus, the use of non - selective beta-blockers (BAB) is shown, which can modify the visceral blood flow in patients with LC; selective BABs are less effective. There are reports of clinical efficacy "Carvedilol" providing sympathetic blockade-b lokiruyuschego effect that, in the end, is a clinical regression of the disease. The dose of carvedilol is recommended to be titrated under the control of AD and electrocardiographic data of the α link in HF and vasodilation due to Prescription of aldosterone receptor antagonists for 24 weeks significantly reduced the thickness of the myocardial walls, peripheral sympathetic activation; the effect on diastolic dysfunction is less significant, which may be due to the duration of the therapy and the insufficient daily dose of the drug. M Pozzi., *et al*. showed that after 6 months of therapy with the aldosterone antagonist K-canrenoate in patients with LC and no signs of severe heart failure, there was a decrease in the size and thickness of the LV wall, a decrease in the end diastolic volume and an improvement in diastolic myocardial function [10]. The pathogenetic treatment of CCM has not been developed at present. The main areas of treatment that can affect cardiovascular changes in LC include peritoneovenous shunting, paracentesis, transjugular intrahepatic port bypass (TIPS), and orthotopic liver transplantation. Organ transplantation has proven its ability to reduce systolic and diastolic myocardial dysfunction, to lengthen the QT interval. However, there is always a risk of a sharp deterioration in the course of CMF, its transition from the latent course to the stage of severe clinical manifestations of heart failure after surgical treatment, TIPS therapy, and terlipressin administration in hepatorenal syndrome [1].

Conclusion

CKMP is an urgent problem of clinical medicine. The following signs suggest the presence of CCM in a patient with LC: prolongation of the QT interval; the appearance of symptoms of heart failure in persons who have undergone surgery; change in indicators of transmitral blood flow (peak A, peak E, ratio E/A); an increase in the concentration of troponin I and brain natriuretic peptide in the blood; left atrial hypertrophy and others. The expediency of using certain representatives of antihypertensive drugs (non-selective BAB, lisinopril, aldosterone receptor antagonists) in the treatment of CMMP is not excluded. TIPS, paracentesis, peritoneovenous bypass grafting, and liver transplantation can be considered among the surgical treatments that have a beneficial effect on the cardiovascular prognosis. A prerequisite for the provision of high-quality medical care to patients with CKMP should be the development of clinical guidelines for this nosological unit.

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