eISSN: 2349-5715 pISSN: 2349-5707

Volume: 11, Issue 11, November-2024 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

Badalov Saidikrom

Andijan State Medical Institute

IDENTIFICATION OF CIRRHOTIC CARDIOMYOPATHY

Abstract. Despite the advances made in the study of liver cirrhosis (LC), it remains an extremely important medical and social problem. There is a continuous increase in persistent disability and, especially, mortality from chronic liver disease, which is among the top ten most common causes of death in the population [3]. It is important to improve the methods of early diagnostics of structural and functional changes in the heart, both for predicting the course of the disease and for possible correction of treatment, including therapeutic and surgical approaches. The relevance of further study of the state of cardiohemodynamics in LC is also due to the need to clarify the criteria for the so-called "cirrhotic cardiomyopathy" (CCM) proposed at the World Congress of Gastroenterologists in 2005 [2]. This term refers to the presence of cardiac dysfunction in patients with CP, characterized by suppression of contractile reactivity to stress and/or changes in diastolic relaxation, with typical electrophysiological manifestations in the absence of any other cardiac pathology [4]

Key words: Liver cirrhosis, hepatitis virus, cardiomyopathy, diagnostics

Patients with liver cirrhosis, especially in the terminal stage, have multiple organ failure, including changes in the cardiovascular system [1, 3]. Currently, it is defined as the presence of one or more myocardial disorders: 1) normal or increased LV contractility at rest, but impaired LV systolic or diastolic function under stress; 2) structural or histological changes in the chambers of the heart; 3) electrophysiological abnormalities of the heart, such as a prolonged QT interval on the ECG; 4) increased levels of serum markers of myocardial stress [5]. In 2005, a working group of experts adopted the definition of CCMP and proposed its diagnostic and auxiliary criteria [5].

Pathogenesis. In patients with liver cirrhosis, the hyperkinetic type of hemodynamics is initially caused by reduced vascular resistance and redistribution of plasma volume [2]. Increased intrahepatic resistance due to fibrosis causes portal hypertension, which in turn contributes to an increase in the level of circulating and endothelial vasodilators due to both compensatory release and slowing of their hepatic degradation. As liver function deteriorates, the plasma volume is redistributed to the abdominal bed. Therefore, despite the overall increase in plasma volume, the circulating blood volume in the central bed decreases. This activates the sympathoadrenal and renin-angiotensin-aldosterone systems, allowing compensation for the decrease in arterial pressure and reduction in circulating blood volume [3]. An increase in cardiac output and a decrease in total peripheral resistance correlate with the severity of hepatocellular insufficiency and portal hypertension [4]. Cirrhotic cardiomyopathy, according to experts, is one of the forms of chronic cardiac dysfunction and is characterized by decreased contractility in response to stress and/or changes in diastolic function with electrophysiological features in the absence of other known heart diseases [1]. Thus, general approaches to the diagnosis of this pathological condition have been established in order to clarify the prevalence, systematize the study and develop effective tactics for managing such patients. Due to the lack of clear criteria for cirrhotic cardiomyopathy until recently, its prevalence remains unknown. According to Soon Koo Baik and colleagues, such distinctive features of cirrhotic cardiomyopathy as prolongation of the QT interval and diastolic dysfunction of the left ventricular myocardium determined by echocardioscopy are present in most patients with class B and C liver cirrhosis according to Child-Pugh [1]. According to our data, 18% of patients with liver cirrhosis have ECG signs of cirrhotic cardiomyopathy regardless of the severity class of liver cirrhosis and its etiology.

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The pathogenesis of cirrhotic cardiomyopathy is currently being actively studied. Cardiomyocyte contractility is mainly regulated by stimulation of β-adrenergic receptors. Binding of adrenaline/noradrenaline to β-adrenergic receptors leads to interaction of the receptor and the binding protein known as Gs or stimulating protein. As a result, another membrane-bound protein, adenylate cyclase, is activated. The end result is the production of cyclic AMP from adenosine triphosphate. The Gs protein is also involved in the direct activation of sarcolemmal calcium channels. This promotes the influx of calcium into the cytoplasm of cardiomyocytes and their contraction [3]. In an experiment, Alqahtani SA and colleagues found several abnormalities in the functioning of β-adrenergic signaling pathways in cirrhosis:decreased density of β-adrenergic receptors; decreased concentration of Gs protein; impaired adenylate cyclase activity, which leads to decreased contractility of cardiomyocytes [4]. For early and late repolarization of cardiomyocytes, a necessary condition is the activation of potassium channels, of which there are three types [5]. Ward CA and colleagues showed in an experiment that in liver cirrhosis there is a decrease in current density for all three types of potassium channels, which can contribute to prolongation of the QT interval in patients with liver cirrhosis. In addition, impaired permeability of cell membranes has been shown due to changes in its lipid composition in cirrhosis, which leads to impaired functioning of β-adrenergic receptors [6,7]. Cardiomyopathies caused by the hepatitis C virus are registered mainly in Asian countries such as Japan, since the prevalence of hepatitis C in these countries is significantly higher than in the USA and European countries. Hepatitis C is associated with frequent development of dilated and hypertrophic cardiomyopathy. It is suggested that the hepatitis C virus may have a direct effect on the growth and hypertrophy of myocardial cells [3].Most researchers consider the main diagnostic criteria for CMC to be the presence of signs of systolic dysfunction (SD) and diastolic dysfunction (DD) of the left ventricle [3, 4]. In addition, important additional criteria for CMC include prolongation of the QT interval, a decrease in the expected number of heart contractions (HR) per load, electromechanical dyssynchrony, myocardial hypertrophy, an increase in the size of the left atrium (LA), an increase in the concentration of phosphatase in the blood. The criteria for CMC are ambiguous and debatable, and their identification is necessary to correct the treatment of patients with CMC, which significantly worsens the prognosis of cirrhosis. The true prevalence of CMC has not yet been studied, which is associated with both the lack of clear diagnostic criteria for this pathology and the insufficient awareness of practicing physicians about the nature of changes in the cardiovascular system in cirrhosis. troponin I, brain natriuretic peptide (BNP) [6, 8].

Conclusion. Thus, the analysis of literary data presented in domestic and foreign sources shows that many viruses and their combinations can be etiological factors leading to cardiovascular pathology. The development of acute damage to the heart muscle or conduction system cannot always be diagnosed based on clinical data alone. Untimely verification leads to incorrect patient management tactics, both in the acute period of the disease and during dispensary observation. Early screening diagnostics contributes to the effective preservation of patients' quality of life.

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