JOURNAL OF MULTIDISCIPLINARY SCIENCES AND INNOVATIONS

VOLUME 04, ISSUE 09
MOPNTHLY JOURNALS

ISSN NUMBER: 2751-4390
IMPACT FACTOR: 9,08

MANAGEMENT STRATEGIES FOR OF CIRRHOTIC CARDIOMYOPATHY.

Badalov S.J.

Andijan State Medical Institute.

Abstract: Although considerable progress has been achieved in understanding liver cirrhosis (LC), it continues to pose a significant medical and social challenge. The prevalence of chronic liver disease–related disability and mortality is steadily increasing, making it one of the ten leading causes of death worldwide [3]. Enhancing early diagnostic approaches to identify structural and functional cardiac alterations is essential for predicting disease outcomes and optimizing therapeutic as well as surgical management strategies. The ongoing importance of assessing cardiohemodynamic parameters in LC also stems from the need to further refine the diagnostic criteria for *cirrhotic cardiomyopathy* (CCM), a concept introduced at the 2005 World Congress of Gastroenterologists [2]. CCM refers to cardiac dysfunction in patients with LC, manifested by a blunted contractile response to stress and/or impaired diastolic relaxation, accompanied by characteristic electrophysiological changes in the absence of other underlying heart diseases [4].

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

In patients with liver cirrhosis, the hyperkinetic pattern of circulation initially develops as a result of decreased vascular resistance and redistribution of plasma volume [2]. Progressive fibrosis leads to increased intrahepatic resistance, giving rise to portal hypertension, which in turn promotes the accumulation of circulating and endothelial vasodilators—both through compensatory overproduction and impaired hepatic degradation. As hepatic function declines, plasma volume becomes increasingly redistributed toward the splanchnic circulation. Consequently, despite an overall expansion of plasma volume, the effective circulating blood volume within the central compartment decreases. This reduction triggers activation of the sympathoadrenal and renin–angiotensin–aldosterone systems, which help maintain arterial pressure and partially compensate for reduced central blood volume [3]. The increase in cardiac output and the concomitant decline in total peripheral resistance are closely associated with the severity of hepatocellular failure and portal hypertension [4]. According to expert consensus, cirrhotic cardiomyopathy represents a specific form of chronic cardiac dysfunction characterized by reduced contractile responsiveness to stress and/or impaired diastolic function, accompanied by distinctive electrophysiological abnormalities in the absence of other primary heart diseases [1]. These general diagnostic approaches have been established to better determine the prevalence of the condition, standardize its investigation, and develop effective management strategies for affected patients. However, due to the absence of well-defined diagnostic criteria until recently, the true prevalence of cirrhotic cardiomyopathy remains uncertain. According to Soon Koo Baik and colleagues, characteristic manifestations of this condition—such as QT interval prolongation and left ventricular diastolic dysfunction detected by echocardiography—are observed in the majority of patients with Child-Pugh class B and C liver cirrhosis [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. 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]. Despite considerable advances in the study of liver cirrhosis (LC), it remains a major medical and social concern. The incidence of persistent disability and, particularly, mortality associated with chronic liver disease continues to rise, placing it among the ten leading causes of death worldwide [3]. Improving early diagnostic approaches to detect structural and functional cardiac alterations is essential for predicting disease progression and optimizing therapeutic as well as surgical management strategies. The continued relevance of investigating cardiohemodynamic disturbances in LC is also linked to the need for refining the diagnostic criteria of *cirrhotic cardiomyopathy* (CCM), a concept introduced at the World Congress of Gastroenterologists in 2005 [2]. This condition is defined as cardiac dysfunction in patients with LC, characterized by a blunted contractile response to stress and/or impaired diastolic relaxation, accompanied by specific electrophysiological abnormalities in the absence of other primary heart diseases [4].Patients with liver cirrhosis, particularly at advanced stages, frequently develop multiple organ failure, including cardiovascular involvement [1, 3]. Cirrhotic cardiomyopathy (CCM) is currently described as the presence of one or more myocardial abnormalities, such as: (1) preserved or enhanced left ventricular (LV) contractility at rest with impaired systolic and/or diastolic performance during stress; (2) structural or histological remodeling of the cardiac chambers; (3) electrophysiological disturbances, including QT interval prolongation on electrocardiography; and (4) elevated concentrations of biochemical markers indicating myocardial stress [5]. In 2005, an expert working group formally defined CCM and proposed diagnostic and supportive criteria for its identification [5].In patients with liver cirrhosis, the

hyperkinetic pattern of circulation initially develops as a result of decreased vascular resistance and redistribution of plasma volume [2]. Progressive fibrosis increases intrahepatic resistance, giving rise to portal hypertension, which promotes the accumulation of circulating and endothelial vasodilators—both through compensatory overproduction and reduced hepatic degradation. As hepatic function declines, plasma volume becomes increasingly redistributed toward the splanchnic circulation. Consequently, despite overall plasma expansion, the effective central blood volume decreases. This reduction activates the sympathoadrenal and renin–angiotensin–aldosterone systems, which help maintain arterial pressure and compensate for reduced circulating volume [3]. The resulting increase in cardiac output and decrease in total peripheral resistance correlate with the severity of hepatocellular failure and portal hypertension [4]. According to expert consensus, cirrhotic cardiomyopathy represents a distinct form of chronic cardiac dysfunction characterized by decreased contractility in response to stress and/or impaired diastolic function, together with specific electrophysiological alterations in the absence of other cardiac diseases [1]. These diagnostic principles have been formulated to improve understanding of its prevalence, standardize research approaches, and optimize patient management. However, due to the lack of clearly established criteria until recently, the true prevalence of CCM remains uncertain. Soon Koo Baik and colleagues reported that characteristic manifestations—such as QT interval prolongation and left ventricular diastolic dysfunction identified by echocardiography—are observed in most patients with Child-Pugh class B and C cirrhosis [1].

Conclusion.

A review of domestic and international literature also suggests that various viral agents and their combinations may serve as etiological factors contributing to cardiovascular pathology. Acute myocardial or conduction system damage cannot always be identified solely on clinical grounds. Delayed or inaccurate verification may lead to suboptimal therapeutic decisions during both the acute phase and long-term follow-up. Therefore, early screening and diagnostic evaluation are crucial for preserving cardiac function and maintaining patients' quality of life.

References

1. Alqahtani, S. A. Cirrhotic cardiomyopathy / S. A. Alqahtani, T. R. Fouad, S. S. Lee // Semin. Liver Dis. – 2008. – Vol. 28. – P. 59–69.

2. American Heart Association Council on the Kidney in Cardiovascular Disease and Council on Peripheral Vascular Disease, American Heart Association, American College of Cardiology Foundation. Cardiac disease evaluation and management among kidney and liver transplantation candidates: a scientifi c statement from the American Heart Association and the American College of Cardiology Foundation: endorsed by the American Society of Transplant Surgeons, American Society of Transplantation, and National Kidney Foundation / K. L. Lentine [et al.] // Circulation. – 2012. – Vol. 126. – P. 617–663.

3. Bayley, T. J. The circulatory changes in patients with cirrhosis of the liver at rest and during exersize / T. J. Bayley, N. Segel, J. M. Bishop // Clin. Sci. – 1964. – Vol. 26. – P. 227–235.

4. Cardiac alterations in cirrhosis: reversibility after liver transplantation / M. Torregrosa [et al.] // J. Hepatol. – 2005. – Vol. 42. – P. 68–74.

5. Cardiac and proinfl ammatory markers predict prognosis in cirrhosis / S. Wiese [et al.] // Liver Int. – 2014. – Vol. 34. – P. e19–e30.

6. Cardiac dysfunction during liver transplantation: incidence and preoperative predictors / C. Ripoll [et al.] // Transplantation. – 2008. – Vol. 85. – P. 1766–1772. 12. Cardiac dysfunction in cirrhosis is not associated with the severity of liver disease / M. Merli [et al.] // Eur. J. Int. Med. – 2013. – Vol. 24. – P. 172–176.

7.Matsumori A. Hepatitis C virus infection and cardiomyopathies. Circ Res 96: 2005; 96: 144.
8.Поражения сердца при инфекционных болезнях / Ю.П. Финогеев [и др.]. – СПб: Фолиант, 2003. – 251 с.