Cardiovascular complications in cirrhosis

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Hyperdynamic circulation and cirrhotic cardiomyopathy are the two major cardiovascular complications in cirrhosis. Hyperdynamic circulation is characterized as increased cardiac output and decreased systemic vascular resistance with low arterial pressure. Basically, these hemodynamic alternations arise from portal hypertension. Portosystemic collaterals develop for counterbalance the increased intrahepatic vascular resistance to portal blood flow, and induce an increase in venous return to heart. Increased shear stress in vascular endothelial cell related high blood flow by portosystemic shunting contributes to this upregulation of eNOS resulting in NO overproduction. Additionally, bypassing through portosystemic collaterals and escaping degradation of over-produced circulating vasodilators in the diseased liver can promote the peripheral arterial vasodilation. Vasodilation of the systemic and splanchnic circulations lead to a reduced systemic vascular resistance, increased cardiac output and splanchnic blood flow. Furthermore, neurohumoralvasoconstrictive systems including systemic nervous system, rennin angiotensin aldosterone system and vasopressin were intensively activated secondary to vasodilation.

Despite the increased basal cardiac output, the ventricular contractile response to physiologic or pharmacologic stimuli is attenuated, a condition termed ‘cirrhotic cardiomyopathy’ (CCM).CCM is generally latent but unmasked when the patient is subjected to stress in the form of infection, exercise, drugs, hemorrhage, and surgery, such as liver transplantation or insertion of transjugular intrahepatic portosystemic stent-shunts. Clinical features include structural, histological, electrophysiological, systolic and diastolic functional abnormalities. Studies in experimental animal models of cirrhosis indicate that multiple factors are responsible, including abnormal membrane biophysical characteristics, impaired β-adrenergic receptor signal transduction and increased activity of negative-inotropic pathways mediated by cGMP.

A complex interplay of multifactorial pathogenic mechanisms probably underlies these phenomena, including some factors common to both hyperdynamic circulation and cirrhotic cardiomyopathy such as NO and neurohumoral activation. Extensive research has improved our understanding of pathogenic mechanisms, thus allowing the possibility of novel treatment modalities. Future studies should focus on pharmacologic and genetic approaches to modulate cardiovascular-regulatory systems, and thereby ameliorate complications related to hyperdynamic circulation and cirrhotic cardiomyopathy.

Key words: Hyperdynamic circulation, Cirrhotic cardiomyopathy, Portal hypertension, Cirrhosis.