The Circulatory System in Liver Disease

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KEYWORDS

• Circulation • Liver disease • Cirrhosis • Vascular resistance

KEY POINTS

- Liver failure is a hyperdynamic state, characterized by a decrease in systemic vascular resistance and increase in cardiac output and heart rate.
- This hyperdynamic state results from splanchnic arterial vasodilatation and opening of portosystemic collaterals, which is mostly mediated by overproduction of nitric oxide and other vasoactive substances.
- Cardiac cirrhosis (congestive hepatopathy) is liver dysfunction consequent to right-sided heart failure.
- Despite a hyperdynamic state, latent cardiac dysfunction, termed cirrhotic cardiomyopathy, may develop and may be manifested by decreased cardiac reserve.
- Latent cardiomyopathy may cause heart failure or hemodynamic decompensation in conditions of stress such as infection, postoperatively, or after transjugular intrahepatic portosystemic shunt placement. Cirrhotic cardiomyopathy may be reversible following liver transplant.

In the cirrhotic liver, distortion of the normal liver architecture occurs as a result of both structural and vascular changes. Because of the arterial vasodilatation that occurs mainly in the systemic and splanchnic circulation, portal hypertension is often associated with a hyperdynamic circulatory syndrome in which cardiac output (CO) and heart rate are increased and systemic vascular resistance (SVR) is decreased. The release of several vasoactive substances, most notably nitric oxide (NO), is considered to be the primary factor involved in the reduction of mesenteric arterial resistance. This decrease in effective circulatory volume triggers baroreceptor-mediated activation of the sympathetic nervous system (SNS) and renin-angiotensin-aldosterone system (RAAS), resulting in sodium and water retention with eventual formation of ascites. The hyperdynamic circulatory state also contributes to numerous cardiovascular abnormalities, including diastolic dysfunction, blunted systolic response to stress, and electrophysiologic abnormalities, which together have been termed cirrhotic cardiomyopathy.

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Echocardiography is a useful imaging modality for evaluation of latent cardiac disease in cirrhotic patients as well as for assessment of comorbidities such as pulmonary hypertension and hepatopulmonary syndrome. The clinical manifestations of cirrhotic cardiomyopathy may become apparent during certain procedures and surgeries, as well as infection, when added stress is placed on the cardiovascular system. Management of these patients with acute cardiac dysfunction can be challenging and often requires invasive hemodynamic monitoring in an intensive care unit (ICU) setting to tailor decisions regarding use of fluids and vasopressors.

PATHOPHYSIOLOGY OF PORTAL HYPERTENSION

The liver normally has high compliance and low resistance that accommodates large volumes of blood, as may occur after eating, without a significant increase in portal pressure. Portal hypertension develops as a result of increases in both portal resistance and portal inflow. Cirrhosis increases resistance to portal flow as a result of collagen deposition in the hepatic acinus, which markedly reduces the total cross-sectional area of the hepatic sinusoids by narrowing the sinusoidal lumen.[1] Intrahepatic portal resistance is further increased by compression of the central veins by portal inflammation and regenerating nodules. This increase in resistance to portal outflow leads to the opening of a portosystemic collateral circulation that occurs through dilatation of preexisting vessels and also by angiogenesis in an attempt to reduce portal pressures. Ultimately, these portosystemic shunts are insufficient at normalizing portal pressures, and complications of portal hypertension, such as gastroesophageal varices and hepatic encephalopathy, can ensue.[1,2]

HYPERDYNAMIC CIRCULATORY SYNDROME

Liver failure is a hyperdynamic state, with increased CO, decreased SVR, and normal or decreased blood pressure (**Box 1**). The chief cause of the hyperdynamic circulation is vasodilatation, which occurs in both the systemic and splanchnic circulations. The consequent reduction in SVR leads to an increase in CO, and blood pressure can be reduced as well. Although extracellular fluid volume is increased, circulating blood volume is decreased. In order to maintain hemodynamic homeostasis, CO and heart rate are increased.

Portal hypertension plays a pivotal role in the development of hyperdynamic circulatory syndrome in patients with advanced liver disease. The increased resistance to portal inflow and outflow contributes to systemic and splanchnic vasodilatation. Portal hypertension may also allow intestinal vasoactive substances to bypass the liver and reach the systemic circulation.

According to the peripheral arterial vasodilatation hypothesis, the decrease in SVR that occurs is a result of peripheral vasodilatation that triggers activation of

Box 1
Hyperdynamic circulatory syndrome

- Increased plasma volume
- Increased CO
- Increased heart rate
- Decreased SVR
- Normal or decreased mean arterial blood pressure