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The Care of the Decompensated Cirrhotic Patient

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ABSTRACT

Liver cirrhosis is among the leading causes of death in the United States, and decompensated cirrhosis is a complicated disease state that can present with various complications such as thrombocytopenia, coagulopathy, ascites, bleeding varices, hepatic encephalopathy, hepatorenal syndrome, anasarca, and jaundice. Each episode of decompensation is associated with an increase in mortality rate. Therefore, it is necessary for providers to equip themselves with a proper understanding of cirrhosis and its complex comorbidities to improve outcomes. Current literature regarding the management of cirrhosis and its complications was reviewed and summarized in this article.

Keywords: ascites, cirrhosis, coagulopathy, decompensated, decompensation, encephalopathy, end-stage liver disease, hepatitis, hepatorenal syndrome, liver

Liver cirrhosis is defined as severe, irreversible fibrotic changes to the hepatic architecture that cause dysfunction in the heptocytes.[1] It is often classified as compensated or decompensated. Patients with compensated cirrhosis have evidence of cirrhosis on imaging but stable laboratory values and no symptoms of complications of the disease. As portal hypertension worsens, patients progress to a decompensated state.[2,3] They present with signs and symptoms including hyponatremia, thrombocytopenia, coagulopathy, ascites, bleeding varices, hepatic encephalopathy (HE), hepatopulmonary syndrome, hepatorenal syndrome, anasarca, and jaundice. Patients may also acutely decompensate and require hospitalization in the presence of infection, gastrointestinal bleeding, portal vein thrombosis, electrolyte and acid-base disturbances, and nonadherence to maintenance medications. Decompensation is associated with increased short-term mortality and may lead to multisystem organ failure.[3,4]

PATHOPHYSIOLOGY OF CIRRHOSIS

Toxic ingestion (eg, alcohol and acetaminophens), fatty infiltration, carcinomas, viral hepatitides, autoimmune processes, genetic anomalies, or other disease states such as right heart failure can cause injuries to the hepatic system. In the United States, the most common cause of end-stage liver disease is hepatitis C infection followed by chronic alcohol use.[5]

Constant injury to the liver can deteriorate the normal sinusoidal architecture, leading to scar tissue formation, or fibrosis, as well as regenerative nodules. This fibrosis can progress to irreversible fibrotic changes to the hepatic architecture and a pronounced distortion of the liver vasculature, or cirrhosis. This distortion causes blood flow compromise, which can lead to increased intrahepatic resistance, in turn causing portal hypertension.

The majority of the manifestations of cirrhosis can be traced back to a syndrome of hyperdynamic circulation and systemic vasodilation.[4] Portal hypertension causes splanchnic vasodilation, leading to systemic vasodilation, increased cardiac output with low arterial blood pressure, and high-output heart failure. Vasodilation also decreases the effective arterial blood volume and blood flow to other organs, leading to the activation of neurohormonal

systems. This, in turn, leads to impaired sodium and water retention, ascites, and hyponatremia. Excess antidiuretic hormone secretion results in excessive water retention, leading to reduced serum sodium concentration and decreased serum osmolality. Finally, the activation of renin-angiotensin-aldosterone system can lead to renal vasoconstriction and hepatorenal syndrome.[3]

Cirrhosis also results in hepatic dysfunction. The liver synthesizes a vast majority of proteins and clotting factors that play a role in maintaining hemostasis by participating in the regulation of coagulation and fibrinolysis. Hepatocellular damage can lead to dysfunctional production and function of coagulation and fibrinolytic factors. This leads to an imbalance between coagulation and anticoagulation systems. Additionally, chronic hypoalbuminemia ensues because of the failing liver's inability to synthesize proteins.[4]

DIAGNOSIS OF CIRRHOSIS

The development of cirrhosis is usually indolent, with no indicators that the liver is scarring. In many cases, patients may be completely asymptomatic until they decompensate, prompting the initial diagnosis at a late stage of the disease.

The diagnosis of liver disease is often made after laboratory or radiographic abnormalities are noted and further investigated. For example, a patient may present with mildly elevated liver enzymes, thrombocytopenia, or coagulopathy. Initially, serologies are drawn to rule out viral or autoimmune causes. A thorough patient history can identify possible causes such as chronic alcohol use or risky behaviors leading to hepatitis B or C infection.[4] Once liver disease is suspected, it is critical to determine the presence and degree of liver fibrosis. Several noninvasive diagnostic tests may be used in the staging of liver fibrosis such as biomarkers and ultrasound-based elastography.[6,7]

Radiologic studies are ordered to assess the liver structure. A liver ultrasound is the least costly imaging modality. It can show nodularity on the surface of the liver, indicating cirrhosis, and assess hepatic blood flow. A computed tomographic scan or magnetic resonance imaging with contrast can show morphologic changes and nodularity of the liver tissue, as well as the accompanying sequelae of liver disease such as ascites, portal hypertension, varicosities, and, more importantly, the presence of hepatocellular carcinoma.[7] Hepatocellular carcinoma is increasingly common, with an annual incidence of about 3% to 5% among cirrhotic patients.[8]

The definitive diagnostic testing for cirrhosis is a liver biopsy; however, it comes with risks.[9,10] Patients can have significant bleeding secondary to underlying coagulopathy and thrombocytopenia from liver dysfunction. There is also a risk, although minimal, for tumor spread if hepatocellular carcinoma is biopsied.[9] A biopsy should only be performed if a diagnosis or cause is not confirmed after a thorough, noninvasive evaluation. A risk-benefit assessment should be completed before proceeding.

SEVERITY OF LIVER DISEASE

The severity of disease is determined by different scoring systems; the 2 most popular are the Child-Pugh (CP) score and the Model for End-Stage Liver Disease (MELD) score. The CP score is a modification of the Child-Turcotte score, which was proposed for predicting mortality in patients with portal hypertension who are undergoing surgery. The score assigns points to encephalopathy, ascites, bilirubin, albumin, and prothrombin. Multiple studies have validated the CP score as a prognostic marker of cirrhosis; however, the CP score has important limitations. For one, interpretation of encephalopathy and ascites can be subjective. Moreover, it is not specifically a marker of liver function but more of the multiorgan changes brought on by cirrhosis.[11]

The second widely recognized scoring tool is MELD, which was originally designed for assessing the survival of cirrhotic patients who are undergoing transjugular intrahepatic portosystemic shunt (TIPS) placement. MELD scores range from 6 through 40, with predicted 3-month mortality rates of 7% to 90%.[12] The score calculates the following using objective variables: creatinine, total bilirubin, international normalized ratio, and the use of dialysis. The MELD has been validated as a strong marker of early mortality in patients with cirrhosis from various causes.[11] The MELD score is also used to prioritize