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GASTROENTEROLOGY BOARD REVIEW MANUAL

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The *Hospital Physician Gastroenterology Board Review Manual* is a study guide for fellows and practicing physicians preparing for board examinations in gastroenterology. Each quarterly manual reviews a topic essential to the current practice of gastroenterology.

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Complications of Portal Hypertension

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Complications of Portal Hypertension

Christian M. Mendez, MD, Luis S. Marsano, MD, and Richard A. Wright, MD

INTRODUCTION

Cirrhosis is a diffuse and reversible pathologic process in which the normal structure of the liver is replaced by regenerative nodules of hepatocytes separated by bands of fibrosis. Cirrhosis may result from various forms of chronic hepatic insult (Table 1). Clinical features of cirrhosis derive from the morphologic alterations and often reflect the severity of hepatic damage rather than the etiology of the underlying liver disease.

The central event leading to hepatic fibrosis is activation of the hepatic stellate cell. The pathologic process should be viewed as a final common pathway of many types of chronic liver injury.¹ Cirrhosis initially may develop without giving rise to signs or symptoms. Compensated cirrhosis may progress, however, and has 2 major pathophysiologic consequences: portal hypertension and hepatic failure. After the diagnosis of cirrhosis, the probability of developing decompensated cirrhosis by 10 years is approximately 60%, and the 10-year survival rate is approximately 50%.²

Proper diagnosis and management of the complications of portal hypertension are vital to improving quality of life and reducing mortality. The complications of portal hypertension reviewed in this article include ascites, spontaneous bacterial peritonitis (SBP), hepatorenal syndrome, gastroesophageal varices, and hepatic encephalopathy.

ASCITES

Ascites, the abnormal accumulation of fluid in the abdominal cavity, is one of the most common complications of advanced liver disease. In general, ascites carries a poor prognosis with high mortality. In the United States, approximately 8 of every 10 patients develop ascites as a consequence of cirrhosis of the liver.³ In patients with compensated cirrhosis, ascites develops at a 5-year cumulative rate of approximately 30%.⁴ Once ascites has developed, the 1-year survival rate is approximately 50% compared with 90% in patients with compensated cirrhosis.⁴ Prognosis is particularly poor in pa-

tients who develop refractory ascites or hepatorenal syndrome.⁵ The treatment of ascites does not appear to prolong life in the cirrhotic patient; it does, however, improve the quality of life and protect the patient from spontaneous infections of the fluid that convey high death rates.⁶

The consequences of ascites are related to the high risk of spontaneous infection in the fluid, the development of abdominal hernias with incarceration or rupture, difficulty breathing due to pressure of the abdomen on the thoracic cavity, decreased food intake with progressive malnutrition, and decreased physical activity.

PATHOGENESIS OF ASCITES RELATED TO PORTAL HYPERTENSION

Portal hypertension leads to ascites formation in patients with liver disease. Three theories of ascites formation have been proposed. The *underfill theory* holds that leakage of intravascular fluid into the peritoneal cavity occurs owing to an alteration in the hydrostatic-oncotic balance. The resultant intravascular volume contraction activates the renin-angiotensin-aldosterone and sympathetic nervous systems, leading to compensatory renal sodium and fluid retention. The *overflow theory* is based on the observation that cirrhotic patients have intravascular hypervolemia as a consequence of primary renal sodium retention, causing overflow of fluid into the peritoneal cavity. According to the *peripheral arterial vasodilation theory*, portal hypertension leads to peripheral vasodilation. The reduced effective arterial blood volume triggers neurohumoral excitation, causing compensatory renal sodium and fluid retention that lead to overflow of fluid into the peritoneal cavity.⁷

EVALUATION OF ASCITES

Patients who develop ascites for the first time, those with ascites who are admitted to the hospital because of illness, those who have ascites that is difficult to control, and those who develop symptoms because of tense ascites all require removal of the fluid for diagnostic evaluation and/or to improve discomfort in the abdomen. The most rapid and cost-effective way to determine the cause of ascites is paracentesis with