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Evaluation and management of variceal bleeding

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ABSTRACT

Acute variceal bleeding is one of the most fatal complications of cirrhosis and is responsible for about one-third of cirrhosis-related deaths. Therefore, every effort should be made to emergently resuscitate the patients, start pharmacotherapy as soon as possible and do endoscopic therapy in a timely manner. Despite the recent advances in treatment, mortality rate is still high. We provide a comprehensive review of evaluation and management of variceal bleeding.

Introduction

Acute variceal bleeding is one of the most fatal complications of cirrhosis and is responsible for about one-third of cirrhosis-related deaths.[1] The development of portal hypertension in cirrhosis is associated with a multitude of complications, most commonly ascites, gastroesophageal varices and hepatic encephalopathy.[2] Nearly half of the patients with cirrhosis have gastroesophageal varices at the time of diagnosis and 5–15% of the patients develop varices each year.[2–4] An important step in decreasing the incidence and mortality of variceal bleeding is screening upper gastrointestinal endoscopy and prophylactic therapy for patients with non-bleeding varices.

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In this report, we discuss the epidemiology, pathophysiology, prevention, and management of acute variceal bleeding.

Epidemiology

Gastroesophageal varices (GOV) are a common complication of cirrhosis affecting up to 50% of patients.[2–4] The development of varices correlates with the severity of liver disease, thus they are more commonly seen in Child-Turcotte-Pugh (CTP) class C patients (85%) than CTP class A patients (40%).[2,5] The annual risk of development of gastroesophageal varices in patients with cirrhosis ranges from 5% to 15%,[3,6,7] and development of varices is seen with a hepatic venous pressure gradient (HVPG) more than 10 mmHg.[3,7]

Esophageal varices (EV) are most frequently seen in the distal 2–5 cm of the esophagus.[4] Gastric varices are classified into four types depending on their location and relation to esophageal varices. In GOV-1, esophageal varices extend along the lesser curvature of the stomach, and this represents the most common type of gastric varices (70%).[3,8–10] In GOV-2, esophageal varices extend into the greater curvature and represent 21% of gastric varices.[3,8,10] Isolated gastric varices (IGV) are less commonly seen and are divided into type 1 IGV which occur in the fundus only (7%) and type 2 IGV which are seen anywhere in the stomach or in the duodenum (2%).[3,8,11] The highest incidence of bleeding is seen in fundal varices (78% in IGV-1, 55% in GOV-2) while GOV-1 and IGV-2 are associated with 10% risk of bleeding.[3]

Gastric varices are seen in about 15–20% of patients with portal hypertension and they are responsible for only 10–30% of cases of variceal hemorrhage, however, they are associated with higher re-bleeding rates, transfusion requirements and mortality rates.[8,9,12]

Acute variceal bleeding is the most common cause of upper gastrointestinal bleeding in cirrhosis and can be fatal.[2,13,14] The annual risk of bleeding from EV is 5–15%.[3,7,15] Several factors increase the risk of bleeding, but the most important is the wall tension of the varix which is directly related to its diameter and pressure.[2,3] The risk of bleeding in varices less than 5 mm in diameter is 7% in 2 years, while it increases to 30% if the diameter is greater than 5 mm.[16] Other important factors include decompensated cirrhosis (CTP B and C) red wale sign and alcoholic liver disease.[2,3,5,13,15]

Acute variceal bleeding represents one of the most severe complications of cirrhosis as it is responsible for one-third of deaths in these patients.[1] Despite advancements in treatment, mortality rate during initial hospitalization is as high as 30%.[16] Mortality rate is highest during the first few days of bleeding and decreases slowly over the following 6 weeks.[3,16]

Pathophysiology

Portal hypertension is the main pathophysiological mechanism that is responsible for the development of gastroesophageal varices. The portal pressure is estimated by the difference between the wedged hepatic venous pressure and the free hepatic venous pressure; the normal gradient is less than 5 mmHg.[17,18] Clinically significant portal hypertension occurs when the hepatic venous pressure gradient (HVPG) is above 10 mmHg, and that can result in multiple complications including GOV, ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, hepatorenal syndrome, splenomegaly, hepatic hydrothorax, portopulmonary hypertension, and hepatocellular carcinoma.[2,3,5,19] Portal hypertension occurs secondary to both an increase in the intrahepatic vascular resistance and increased portal blood flow.[6,17,19,20] Increased intrahepatic vascular resistance occurs through mechanical and dynamic components. The mechanical component is related to the massive structural changes associated with intrahepatic fibrosis and regenerative nodules that lead to sinusoidal portal hypertension.[17,21,22] An additional response to liver injury is the hepatic stellate cells activation and transformation into myofibroblasts which contract around the newly formed sinusoidal vessels thereby increase the intrahepatic vascular resistance.[22]

The dynamic component is related to an imbalance between vasoconstrictors and vasodilators leading to increased intrahepatic vascular tone. In the cirrhotic liver, there is an increased activity of