Epidemiology, Diagnosis and Early Patient Management of Esophagogastric Hemorrhage

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KEYWORDS

• Acute variceal bleeding • Esophageal varices • Gastric varices
• Endoscopic variceal ligation

KEY POINTS

- Esophageal and gastric varices are common among persons with cirrhosis.
- Short-term antibiotic prophylaxis, early resuscitation, targeting of conservative goals for blood transfusion, and management of complications such as infection and renal failure are important.
- Combination therapy with vasoactive drugs and endoscopic variceal ligation is the first-line treatment in the management of acute variceal bleeding after adequate hemodynamic resuscitation.
- The MELD score is an important predictor of early mortality after variceal bleeding.

INTRODUCTION

Epidemiology

At the time of initial diagnosis of cirrhosis, approximately half of the patients have esophageal varices, and with the progression of cirrhosis approximately 90% of patients develop esophageal varices.[1–3] Varices are present in approximately 40% of patients with compensated cirrhosis and 60% of patients with ascites.[3,4] Large esophageal varices (>5 mm) are seen in 16% of all patients screened for varices by upper endoscopy.[5] The presence of varices correlates with the severity of liver disease: 20% to 40% of Child-A cirrhosis patients have esophagogastric varices (EGV), compared with up to 85% of Child-C cirrhotics. Patients with primary biliary cirrhosis may develop varices and aute variceal bleeding even in the absence of established cirrhosis.[6]

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Esophageal Varices

Patients with cirrhosis who do not have varices at the time of initial upper endoscopy develop varices at a rate of 8% per year.[7] The progression of small (<5 mm) to large varices (>5 mm) occurs at a similar rate of 7% to 8% per year.[8] An elevated hepatic venous pressure gradient (HVPG) of greater than >10 mm Hg is an independent predictor of the development of varices.[7] About one-third of patients who have varices develop AVB.[9,10] Patients who are found to have small varices at the time of initial endoscopy have a 5% per year risk of bleeding, compared with 15% per year in those with medium-sized or large varices at diagnosis.[4] Moreover, 40% of patients with AVB spontaneously stop bleeding without any intervention, compared with 80% of those with nonvariceal causes of upper gastrointestinal bleeding.[9] However, patients with severe liver disease (Child C) are less likely to stop bleeding spontaneously. With the current standard of therapy, 80% to 90% of patients have cessation of hemorrhage.[3] Despite treatment, 1 in 4 patients will still show either a failure to control the bleeding or an early recurrence of the hemorrhage in the first 6 weeks after the initial bleeding.[11,12] The risk of rebleeding is highest in the period immediately after the sentinel bleed: 40% of all rebleeding episodes occur within the first 5 days.[13,14] An elevated HVPG of greater than 20 mm Hg, when measured within 24 hours of variceal hemorrhage, is associated with failure to control bleeding and early rebleeding. A Model for End-Stage Liver Disease (MELD) score of 18 or higher is also an independent predictor of early rebleeding.[15,16]

Gastric Varices

Gastric varices (GV) are present in 20% of patients with portal hypertension, and are the source of 5% to 10% of all upper gastrointestinal bleeding episodes in patients with cirrhosis. GV carry a 10% to 16% risk of bleeding in 1 year and 25% risk of bleeding in 2 years.[17] Although the prevalence and bleeding risk of GV are lower, the bleeding is usually more severe, requires more transfusions, and is associated with higher mortality. Even after endoscopic injection of tissue glue, GV bleeding is still associated with high rebleeding rates, ranging from 22% to 37%. The risk of recurrent bleeding depends on the location of the varix: isolated varices in the gastric fundus (53%) bear the highest risk for recurrent bleeding, followed by varices along the greater gastric curvature (19%) and lesser gastric curvature (6%). The annual incidence of bleeding is 4% in patients with Child class A with small varices without red wale signs, and 65% in patients with Child class C with large varices with red wale signs.[18] Large fundal varices may occasionally bleed despite HVPG values of less than 12 mm Hg.[19]

Mortality

Any death occurring in the first 6 weeks after the index bleed is considered a bleeding-related death. Three decades ago, AVB was associated with a mortality of 60% at 6 weeks.[13] With recent developments in pharmacologic and endoscopic treatments, this figure has significantly improved in the present era to 20% or less.[20] Immediate mortality from uncontrolled bleeding is in the range of 4% to 8%.[21] Child class C, MELD score 18 and higher, and failure to control bleeding or early rebleeding predict 6-week mortality.[16,22] Mortality is 0% among patients with Child class A disease and approximately 30% among patients with Child C. Forty percent of deaths are directly related to bleeding and shock, with the remainder being due to renal failure, hepatic encephalopathy (HE), and sepsis.[19] In a recent analysis, MELD greater than 19 predicted 20% or greater mortality, whereas MELD scores of less than 11 predicted less than 5% mortality.[23]