Endoscopic Hemostasis in Acute Esophageal Variceal Bleeding

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KEYWORDS

• Endoscopy • Hemostasis • Acute variceal bleeding • Portal hypertension

KEY POINTS

- Acute variceal bleeding (AVB) is a serious complication of patients with portal hypertension.
- Initial management includes appropriate volume replacement, transfusion of blood to keep hemoglobin levels around 7 to 8 g/dL, antibiotic prophylaxis, and endotracheal intubation in selected cases.
- Standard of care mandates early administration of vasoactive drug therapy followed by endoscopic band ligation (EBL) or injection endoscopic sclerotherapy (if EBL cannot be performed) within the first 12 hours of patient presentation.
- Patients who fail endoscopic hemostasis therapy may require the temporary placement of balloon tamponade or an esophageal stent; however, experience with esophageal stents is limited and use of balloon tamponade is associated with potentially lethal complications such as aspiration and perforation of the esophagus.
- Both modalities should be available for potential use and all patients surviving an episode of AVB should undergo secondary prophylaxis in order to prevent variceal rebleeding.

The development of portal hypertension in cirrhosis changes the natural course of patients with chronic liver disease because it has several consequences, including the development of gastroesophageal varices, variceal bleeding, ascites, hepatorenal syndrome, and hepatic encephalopathy. The initial appearance of varices in patients

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with compensated cirrhosis indicates a progression of the disease from a low-risk state to an intermediate state, but once bleeding occurs this indicates decompensation and an increased risk of death.[1] Although mortalities caused by acute variceal bleeding (AVB) have declined from nearly 60% to 15% to 20% at 6 weeks in the past 3 to 4 decades, there is still a significant risk of recurrence and of morbidity and mortality.[2–4] All patients surviving an AVB should therefore receive secondary prophylaxis.[5] This article reviews the current management approach of patients with AVB with particular emphasis on endoscopic hemostatic techniques for bleeding varices.

NATURAL HISTORY AND DIAGNOSIS OF ACUTE VARICEAL BLEEDING

Patients with esophageal varices have an incidence of AVB that ranges from 4% to 15% per year depending on the severity of liver disease, variceal size, presence of red wale markings, and a hepatic venous pressure gradient (HVPG) value greater than 12 mm Hg.[6–8] In most cases (80%), patients with cirrhosis and gastrointestinal bleeding have gastroesophageal varices as the cause of hemorrhage. Thus upper gastrointestinal bleeding in patients with cirrhosis must be presumed to be variceal in origin until proved otherwise. Although the clinical history is highly reliable in assuming the diagnosis of AVB, the gold standard for the diagnosis is upper endoscopy. Endoscopy can show active blood spurting or oozing from a varix (this can be present in 15% of patients) (**Fig. 1**), a white nipple or clot adherent to a varix, or the presence of varices without other potential sources of bleeding in the upper gastrointestinal tract. Although acute bleeding from varices may cease spontaneously in nearly half of patients, rebleeding rates are significantly high (30%–40%) if patients are not treated appropriately.[9,10] The highest risk occurs within the 48 hours following the index bleed and most of the rebleeding episodes occur with the first 14 days. Initial endoscopic failure to control bleeding occurs most commonly in patients with Child class C cirrhosis, concomitant bacterial infection, portal vein thrombosis, active spurting of a varix, and an HVPG greater than 20 mm Hg.[11–15]

Survival from an episode of AVB has improved greatly: from 42% in the 1980s[10] to the current rates of 80% to 85%.[3,4,16–19] This improvement has been caused by overall improvements in intensive care, volume repletion, pharmacologic and endoscopic therapy, implementation of transjugular intrahepatic portosystemic shunts

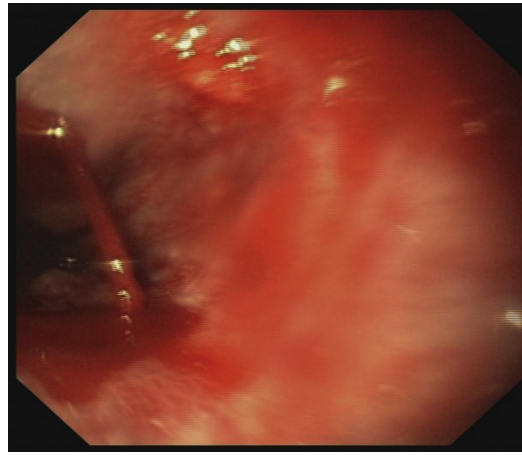

Fig. 1. Endoscopic view of actively bleeding esophageal varix.