Nonendoscopic Management Strategies for Acute Esophagogastric Variceal Bleeding

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KEYWORDS

• Portal hypertension • Variceal hemorrhage • Variceal bleeding • Varices • TIPS
• Portosytemic shunt • BRTO

KEY POINTS

- Initial stabilization and resuscitation is imperative in the management of acute variceal bleeding along with attention at prevention of associated complications such as hepatic encephalopathy, acute renal injury, spontaneous bacterial peritonitis, and sepsis.
- Urgent attention at achieving hemostasis through endoscopic means remains the key and should be supported by pharmacotherapy aiming at reducing portal venous pressure.
- Patients failing initial treatment should be rescued with transjugular intrahepatic portosystemic shunt, balloon-occluded retrograde transvenous obliteration, or rarely surgical shunts.

Acute variceal bleeding is a potentially life-threatening complication of portal hypertension defined as elevation of hepatic venous pressure gradient (HVPG) to greater than 5 mm Hg. Portal hypertension is classified as prehepatic, intrahepatic, or posthepatic, intrahepatic being the form most often caused by cirrhosis, irrespective of the cause.[1] Portal hypertension results in redistribution and increased blood flow through the coronary veins and the short gastric veins, resulting in esophageal and gastric varices. Gastroesophageal varices begin to form at a pressure gradient of 8 to 10 mm Hg, with bleeding risk increased at a gradient of 12 mm Hg.[1] In patients without varices, esophageal varices develop and grow in size at a rate of about 7%

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per year as a result of ongoing portal hypertension.[2,3] Variceal rupture could potentially occur in about one-third of patients, with the highest rates observed in patients with HVPG greater than 20 mm Hg[4] and/or Child C patients with large varices with red wale markings.[5,6] Acute variceal bleeding occurs in 25% to 40% of cirrhotic patients and carries a mortality of 25% to 30%, making it one of the most dreaded complications of portal hypertension.[7] Bleeding usually occurs at the gastroesophageal junction because varices are most superficial and have the thinnest wall at this anatomic location. Approximately 50% of the acute variceal bleeding ceases spontaneously.[8] After an index bleeding episode, most of the episodes of rebleeding occurs in the first 6 weeks[9,10] and more than 50% of such rebleeding episodes occur within 3 to 4 days from the time of the initial bleeding episode.[9,11–14] The risk factors for early rebleeding are severe initial bleeding as defined by a hemoglobin less than 8 g/dL, gastric variceal bleeding, thrombocytopenia, encephalopathy, alcohol-related cirrhosis, large varices, active bleeding during endoscopy, and a high HVPG.[11–16] In the long term, approximately 70% of subjects experience further variceal bleeding and have a similar risk of mortality within the first year.[17,18] Age greater than 60 years, large esophageal varices, severity of liver disease, continued alcoholism, renal failure, and presence of a hepatoma increase the risk of rebleeding.[12,19]

Before the advent of pharmacotherapy, endoscopic therapy, and shunt procedures for control of variceal bleeding, almost 40% of patients with acute variceal hemorrhage died within 6 weeks, one-third rebled at 6 weeks, and only about one-third survived beyond 1 year.[9]

Significant advances have been observed in the last 2 decades in the management of acute variceal bleeding by both endoscopic and nonendoscopic means and have resulted in significant reductions in both morbidity and mortality from this potentially life-threatening condition. Endoscopic treatment is important and remains the cornerstone in the management of acute variceal bleeding, and newer techniques are continuing to evolve. The current article, however, intends to highlight only the current nonendoscopic treatment approaches for control of acute variceal bleeding and recent developments.

MANAGEMENT OF ACUTE VARICEAL BLEEDING

General Management

Acute variceal bleeding is a potentially life-threatening event. Most patients vomit blood but hematochezia and melena might be the only initial symptoms. Hemodynamic stability depends on the amount of blood lost; presentation could include symptoms of orthostatic hypotension to hemorrhagic shock. Despite advances in therapy, up to 40% of patients still die from exsanguinating bleeding. Of note, most deaths are unrelated to bleeding per se and are rather caused by complications of bleeding such as liver failure, infections, and hepatorenal syndrome.[20,21] The degree of liver dysfunction, creatinine level, hypovolemic shock, active bleeding on endoscopy, and presence of hepatocellular carcinoma are important determinants of adverse outcome.[20–24] Thus, the management of patients with acute variceal bleeding includes not only treatment and control of active bleeding but also the prevention of rebleeding, hepatic encephalopathy, infections, and renal failure.[25] Available therapeutic options to control bleeding include medical and endoscopic treatment, balloon tamponade, placement of fully covered self-expandable metallic stents, transjugular intrahepatic portosystemic shunt (TIPS), and surgical shunts. Nowadays, the initial approach is a combination of vasoactive drugs, antibiotics, and endoscopic therapy,[26] followed by a more aggressive approach in patients failing first-line treatment.