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Endoscopic management of acute esophageal variceal bleeding

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ABSTRACT

Esophageal varices develop in the setting of portal hypertension, most commonly caused by cirrhosis. Esophagogastroduodenoscopy is considered the gold standard for both diagnosis and treatment of acute variceal bleeding. In this review, we highlight the management of both acute and refractory bleeding from esophageal varices, with an emphasis on endoscopic therapies, including injection sclerotherapy, band ligation, and esophageal stent placement.

1. Introduction

Esophageal varices develop in the setting of portal hypertension, most commonly caused by cirrhosis, as well as hepatic vein thrombosis, portal vein thrombosis, or infiltrative processes within the liver [1]. Varices are present in up to 50% of patients with cirrhosis, and acute variceal bleeding can occur at a yearly rate of 15% in these patients [2]. The severity of variceal bleeding episodes is directly related to the severity of the patient's underlying cirrhosis, with a 30-day mortality rate of up to 30% per variceal bleeding event in patients with Child-Pugh class C cirrhosis [3]. Bleeding in these patients can be severe and fatal, and is also associated with renal failure and infections, such as spontaneous bacterial peritonitis [4].

Esophagogastroduodenoscopy is considered the gold standard for both diagnosis and treatment of acute variceal bleeding, as it can be performed emergently and provide therapy at the time of assessment [5]. Endoscopic signs of recent variceal bleeding include an adherent clot over a varix, a platelet plug (white nipple sign), or a longitudinal red streak located on a varix (red wale sign) [6]. Treatment failure (refractory bleeding) is defined as hematemesis or >100 mL of blood in the nasogastric aspirate >2 hours after the start of pharmacologic or endoscopic treatment, development of hypovolemic shock, or a drop in hemoglobin of ≥ 3 g/dL within a 24-hour period [7].

In this review, we will discuss the treatment of both acute and refractory bleeding from esophageal varices, which is generally aimed at decreasing portal hypertension and directly treating the varices. Whereas pharmacologic therapy and transjugular intrahepatic portosystemic shunt (TIPS) are methods used to reduce portal hypertension, our emphasis will be on endoscopic modalities, such as band ligation, sclerotherapy, and esophageal stent placement, which directly target esophageal varices.

2. Treatment modalities

2.1. Balloon tamponade

Balloon tamponade can be used to achieve hemostasis in patients with actively bleeding esophageal varices. In general, this is a short-term treatment that is used to provide temporary hemodynamic stabilization until definitive portal decompressive therapy can be offered [5].

Several balloon systems are available to achieve tamponade, with the most commonly used device being the Sengstaken-Blakemore tube, which contains a gastric balloon, an esophageal balloon and a gastric suction port. The Minnesota tube (a modified Sengstaken-Blakemore tube) and the Linton-Nachlas tube (larger gastric balloon used primarily for gastric varices) are other variations of a similar balloon system, which have a traction and pulley apparatus to maintain constant tension on the tube [8]. The inflated balloon can remain in place for up to 48 hours, with deflation of the balloon every 12 hours while it remains in place to evaluate for rebleeding.

Complications associated with the use of balloon tamponade devices range from minor adverse events, such as nasopharyngeal mucosal bleeding, chest pain and discomfort, to much more serious and life-threatening complications. Major adverse events, which can occur in up to 37% of patients, include airway

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obstruction, aspiration pneumonia, and esophageal or gastric rupture [9]. In addition, keeping the balloon inflated can result in pressure necrosis. Although balloon tamponade can be successful in stopping bleeding, approximately 50% of patients experience rebleeding after deflation of the balloon, further highlighting the urgent need for more definitive therapy [10,11]. Because of technical unfamiliarity, procedure-related risks and availability of other effective therapies for variceal bleeding, balloon tamponade is uncommonly performed nowadays. However, when properly placed, it serves as a useful bridge to more definitive therapy, particularly in the torrential variceal bleeder [12].

2.2. Endoscopic variceal sclerotherapy

Endoscopic variceal sclerotherapy (EVS) was among the first therapies introduced for the treatment of bleeding varices. EVS involves the injection of a sclerosing agent within the varices to induce an inflammatory reaction, which results in the development of intraluminal thrombosis, intimal thickening, and eventually perivenous fibrosis [13]. Several sclerosing agents have been used, including absolute alcohol, polidocanol, ethanolamine oleate, and sodium tetradecyl sulfate, although there is no clear evidence that any one sclerosant is superior to another [14].

Initial studies comparing sclerotherapy to sham injections or medical management alone found EVS to be superior in regards to control of active bleeding and prevention of recurrent bleeding [15-17]. In addition, overall survival was improved by EVS, with 1 meta-analysis reporting a reduction in mortality of 25% over a follow-up period of 2-5 years [18]. Compared to balloon tamponade, EVS has been shown to be as effective in achieving immediate hemostasis, and more effective in preventing early rebleeding (within 6 weeks of the initial bleed) [19]. The risk of rebleeding remains relatively high, however, around 15%-50% within the first 24 hours [20]. EVS is typically performed on a weekly basis until varices have been obliterated completely, with an average of approximately 3 sessions to achieve that goal [21].

Shortcomings of EVS include local and systemic complications, such as esophageal ulceration, stricture formation, perforation, and bacteremia [22,23]. Clinically significant ulcers can develop in up to 30%-50% of patients undergoing EVS, a risk that cannot be mitigated by proton pump inhibitor or H_2-antagonist therapy as this is not an acid-mediated phenomenon [24,25]. In addition, approximately 15% of patients subsequently develop symptomatic esophageal strictures, and treatment-related deaths have been estimated at 1%-2% [21,26]. Currently, EVS is a rarely performed procedure since it has been largely replaced by endoscopic band ligation (EBL). However, in cases where EBL is technically not feasible or fails at controlling hemorrhage, consideration may be given to the use of EVS as rescue therapy.

2.3. Endoscopic band ligation

EBL was introduced in the 1990s as an alternative to EVS, and has quickly developed into the cornerstone for the management of acute variceal bleeding. Current multiband devices enable deployment of 6-10 rubber bands in 1 session [27]. The principal goal of band ligation is to strangulate and, ultimately, obliterate the perforating veins connecting varices to extraesophageal collaterals. Hemostasis is achieved by suctioning the target varix into the band ligation cap affixed to the tip of the endoscope, then pulling a tripwire to deploy the band at the base of the varix [28]. Band ligation facilitates necrosis of the entrapped tissue (which includes the underlying varix) and leads to thrombosis of the varices within 24-48 hours (Figure 1) [29]. The bands eventually fall off, leaving an ulcer, which heals and scars down to produce lasting blood flow disruption in the varix. EBL can be repeated every 2-6 weeks until all varices are eradicated. EBL is used not only for acute bleeding (Figure 2), but for primary and secondary prophylaxis of varices as well [30].

Steigmann et al were the first to study EBL compared to EVS for acute variceal bleeding in a randomized prospective fashion. Patients were followed for a mean of 10 months, and initial treatment was followed by elective retreatment by the same modality to eradicate the varices. Compared to EVS, EBL resulted in greater initial control of active bleeding (86% vs 77%) and lower rate of recurrent hemorrhage (36% vs 48%), although neither of these outcomes was statistically significant. However, EBL achieved eradication of varices with fewer endoscopic procedures, with a statistically significant lower mortality and complication rates compared with EVS [31]. Laine et al found similar results in their prospective study of 77 patients with bleeding varices who were randomly assigned to receive EVS or EBL at the initial endoscopic examination. Although rates of initial control of bleeding were similar in both groups at about 90%, the rebleeding rate was lower in patients treated with EBL (26% vs 44%). Furthermore, the EBL group had fewer complications at follow-up, with a lower rate of esophageal strictures (0% vs 33%) and complicated esophageal ulcers (2.6% vs 15%) [32]. Meta-analyses of 7 randomized trials provide further evidence that EBL is associated with lower rates of rebleeding, fewer endoscopic sessions to achieve eradication, reduced mortality, and fewer complications [33]. As such, EBL is preferred to EVS and is considered the endoscopic treatment of choice for patients with esophageal variceal bleeding.

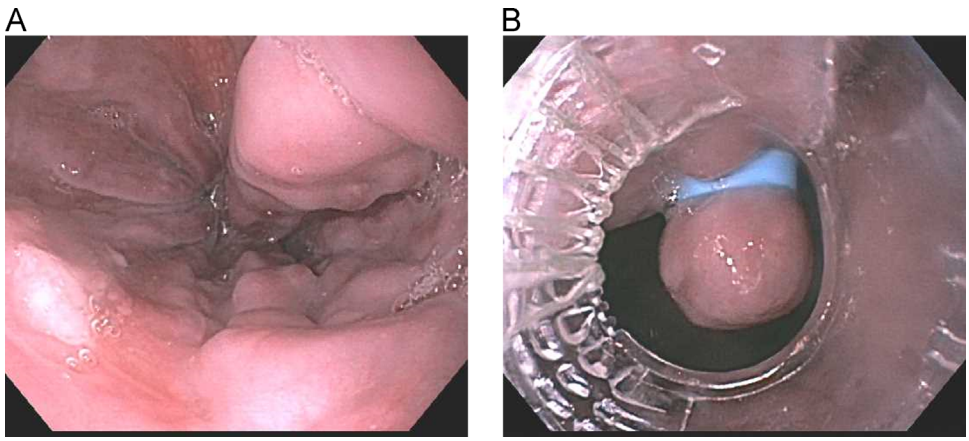

Fig. 1. (A) Endoscopic image of large varices in a patient who had experienced hematemesis. No active bleeding was seen but the bleeding was presumed to originate from his varices. (B) Strangulation of varices after endoscopic band ligation. No rebleeding occurred after band ligation.