
Endoscopic Therapy for Gastric Varices

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Abstract

Gastric varices occur in about 20% of patients with portal hypertension. Although bleeding from gastric varices occurs less frequently than from esophageal varices, it is usually more severe and more difficult to treat. There are multiple treatment options for bleeding gastric varices including placement of the Sengstaken-Blakemore tube, endoscopic sclerotherapy, performing a transjugular intrahepatic portosystemic shunt (TIPSS) and balloon-occluded retrograde transvenous obliteration (B-RTO). However, B-RTO is only possible to perform in the presence of splenorenal shunts. The Sengstaken is a measure of last resort, not used by most experts anymore. TIPSS should mainly be used if there is chance for liver transplantation. Of the available endoscopic options, injection of *N*-butyl-2-cyanoacrylate sclerotherapy is highly effective for the obliteration and treatment of active bleeding gastric varices. This article describes the endoscopic approach to gastric varices.

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Gastric varices occur in 20% of patients with portal hypertension [1]. Although bleeding from gastric varices occurs less frequently than from esophageal varices, it is usually more severe and more difficult to treat [1–3]. There are multiple treatment options for bleeding gastric varices including placement of the Sengstaken-Blakemore tube, endoscopic sclerotherapy, performing a transjugular intrahepatic portosystemic shunt (TIPSS) and balloon-occluded retrograde transvenous obliteration (B-RTO). Of note, B-RTO is only possible to perform in the presence of splenorenal shunts [3–7]. Of the available endoscopic options, injection of *N*-butyl-2-cyanoacrylate sclerotherapy is highly effective for the obliteration and treatment of active bleeding gastric varices [6–12].

Procedural Aspects

Patient Preparation

Generally, endoscopic therapy for gastric varices is performed in patients who have significant bleeding and is rarely used on elective basis (fig. 1). It is of paramount importance that the

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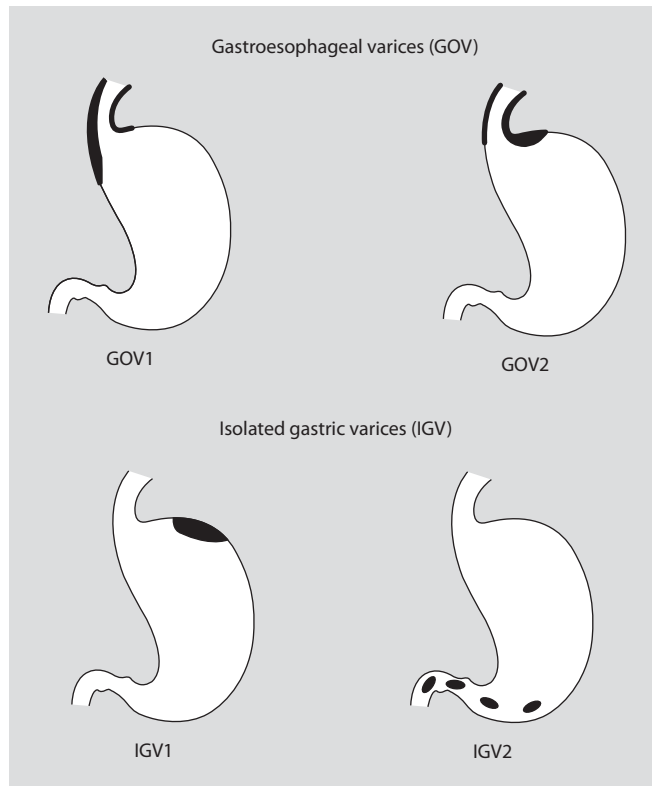
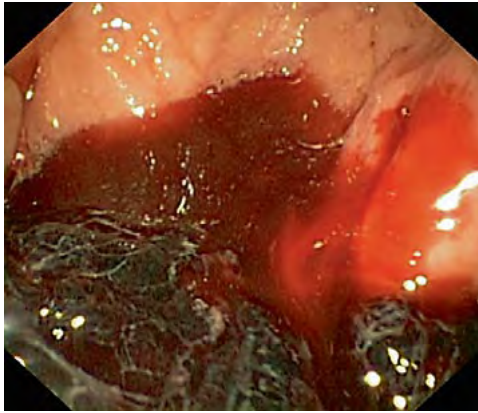


Fig. 1. Endoscopic classification of gastric varices (classification of Sarin and Kumar [9]).

patient is hemodynamically stable and adequate airway protection can be warranted. In cases of massive bleeding, patients should undergo endotracheal intubation to protect the airways. If the patient is hemodynamically stable the endoscopy is performed either in the shock room of the emergency room or in the endoscopy suite under constant monitoring of their vital signs. In cases of hemodynamic instability the endoscopy should always be performed in the intensive care unit. If present, correction of a bleeding diathesis is mandatory. We do not transfuse with platelets unless the total count is $<20,000$. In patients with advanced cirrhosis of the liver, we routinely administer intravenous vitamin K for at least 3 days. Antibiotic prophylaxis is mandatory as it associated with less variceal rebleeding and may also prevent spontaneous bacterial peritonitis in patients with ascites. The preferred antibiotics are ceftriaxone or levofloxacin.

Accessories

Endoscopy. Endoscopy is performed using a forward-viewing endoscope. Gastric varices are categorized according to the classification of Sarin and Kumar [9] into two main categories. The first category includes varices which extend from the gastroesophageal junction into the stomach (GOV). The varices which extend from the esophagus into the lesser curvature of the stomach are GOV type 1 (GOV1). GOV1 appear as a continuation of esophageal varices and extend for 2–5 cm below the gastroesophageal junction into the lesser curvature. Frequently such varices can and should be treated using endoscopic band ligation, in the same way as esophageal varices, especially if they extend <3 cm into the lesser curvature. GOV type 2 (GOV2) varices extend beyond the gastroesophageal junction into the fundus of the stomach (fig. 1–4) [9]. The second category,



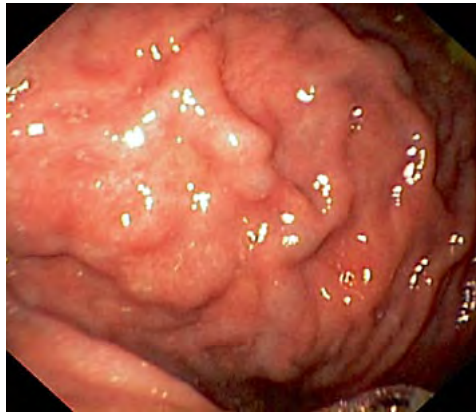
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Fig. 2. Common endoscopic finding in the presence of bleeding gastric varices. Usually the stomach fundus is filled with blood and blood clots.

Fig. 3. Large gastric varices extending from the gastroesophageal junction into the fundus (GOV2). They have the appearance of a bunch of grapes.



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Fig. 4. Isolated fundic varices (IGV1).

isolated gastric varices (IGV), includes two types: fundic (IGV1) or those located anywhere else in the stomach (IGV2). Active bleeding is defined as active blood spurting or oozing from a varix and evidence of recent bleeding was defined as the presence of nipple or red whale signs.

Potential complications from the procedure include aspiration, fever, worsening hemorrhage due to the sclerotherapy and systemic embolization of cyanoacrylate.

Materials Used for Sclerotherapy. There are two major cyanoacrylates (*N*-butyl-2-cyanoacrylate and 2-octyl-cyanoacrylate, Dermabond®). Histoacryl® (*N*-butyl-2-cyanoacrylate (B. Braun, Aesculap AG, Tuttlingen, Germany) is prepared as follows: the injection needle is primed with water, followed by the mixture of Histoacryl (0.5 ml) and lipiodol (1.5 ml) (Lipiodol Ultra-Fluid, Guerbet GmbH, Sulzbach, Germany). Sclerotherapy should always be performed using a disposable sclerotherapy needle (e.g. sclerotherapy needle, 2.3 mm diameter, MTW Endoskopie, Wesel, Germany).

Technique

The catheter is introduced into the working channel of the endoscope and advanced. Once the bleeding varix is localized, the needle is exposed and inserted into the base of the varix and the assistant injects the Histoacryl-lipiodol mixture. The injection is done slowly, lasting about 10 s. The need for further injection is determined by probing the varix with the catheter. In the presence of a 'cushion sign' (i.e. soft varix filled with blood) there is a need for further injections [11].

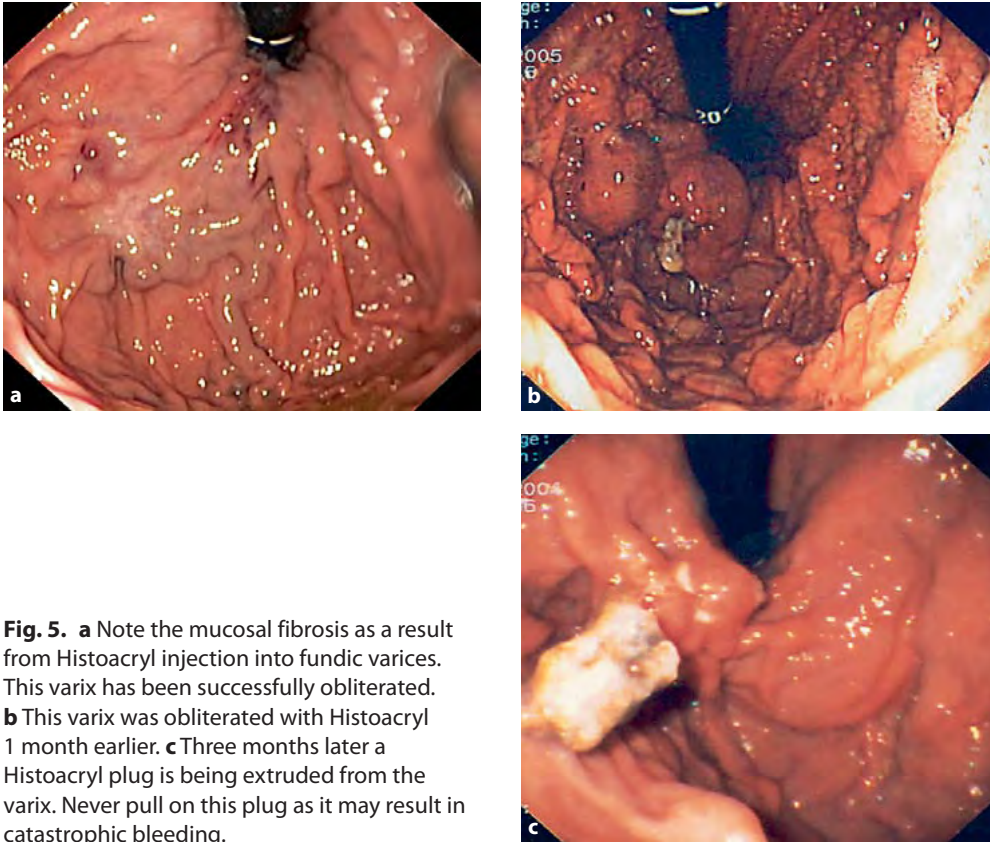


Fig. 5. **a** Note the mucosal fibrosis as a result from Histoacryl injection into fundic varices. This varix has been successfully obliterated. **b** This varix was obliterated with Histoacryl 1 month earlier. **c** Three months later a Histoacryl plug is being extruded from the varix. Never pull on this plug as it may result in catastrophic bleeding.

However, we recommended not to inject more than 1 ml Histoacryl per session as this may be associated with a higher risk of thromboembolism. Upon removing the needle from the varix, the assistant injects normal saline to flush the remaining glue from the catheter. In order to prevent damage to the endoscope, the needle catheter is not retrieved out of the working channel. Instead, the endoscope and the catheter are removed in toto. Both the endoscopist and endoscopy assistant should use goggles as part of the universal precautions guidelines, but also to decrease the likelihood of accidental sprinkling and glue-induced damage to the eyes. Eradication of gastric varices can be accomplished in two thirds of treated patients (fig. 5). Please remember that GOV1 can often be treated using band ligation, as they behave more like esophageal varices.

Post-Procedure Care

Patients with bleeding gastric varices should be managed in the intensive care unit. The patient should remain NPO and receive adequate intravenous fluid volume and electrolyte substitution. Transfusion of fresh-frozen plasma is recommended when the INR remains > 1.6 , despite adequate vitamin K supplementation. All patients should also receive either intravenous octreotide or terlipressin. Unless there is no further bleeding, a repeat or second-look endoscopy is not necessary.

Outcomes

Therapy of bleeding gastric varices is a therapeutic challenge. Thus, it is important to have methods that can be helpful in such situations [3]. Most patients with bleeding gastric varices receive medical therapy with octreotide or terlipressin with the aim of reducing portal pressure by inducing splanchnic vasodilation [13–15]. Whereas this approach has been validated for bleeding esophageal varices, its use has not been confirmed for gastric varices. Nevertheless, we always use any of these agents in the treatment algorithm of patients with bleeding gastric varices with the hope of reducing the pressure of the varix. Similar to patients with esophageal varices, we also recommend using β -blockers in patients with IGTV.

Many endoscopists, especially outside Europe, are wary of using tissue adhesives such as cyanoacrylate to treat gastric varices because of the reported complications including embolization, increased bleeding and bacteremia [16–18]. This fear is not unfounded. Nevertheless, endoscopic therapy of gastric varices with band ligation is also associated with complications [19–22]. Furthermore, when treating patients with a catastrophic event such as gastric variceal bleeding, the endoscopist needs to take into consideration the higher benefit of stopping the bleeding in relation to the lower risk of complications. The hemostatic success of rates Histoacryl ranges from 55 to 90% [7–19]. Furthermore, it is important to emphasize that there are currently very few other options to treat patients with gastric variceal bleeding [23, 24]. The use of TIPSS is limited if there is portal vein thrombosis, a small portal vein or awkward anatomy [25, 26]. The use of endoloops to obliterate gastric varices appears promising, but there are only few data documenting its efficacy [27]. The use of the Sengstaken-Blakemore tube is considered by many a futile exercise and is generally used as a last resort [28]. However, if a patient is a good candidate to receive a TIPSS, then temporary use of the Sengstaken-Blakemore tube may be beneficial.

To summarize, *N*-butyl-2-cyanoacrylate sclerotherapy is highly effective for the treatment of active bleeding gastric varices, with 15% complications occurring both acutely and long term.

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Portal Hypertensive Gastropathy

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Abstract

Portal hypertensive gastropathy (PHG) and gastric antral vascular ectasia (GAVE), frequently combined, are responsible for acute and chronic bleeding in patients with liver cirrhosis and portal hypertension. Endoscopy is basic for detection of typical features and histological examination can enhance diagnostic accuracy. PHG is frequently responsible for chronic bleeding/anemia and the first treatment option is medical therapy; otherwise, failures can be managed by shunting procedures, such as transjugular intrahepatic portosystemic shunt or surgery. Vasoactive drugs (terlipressin or somatostatin and analogues) can be effectively adopted in less common acute bleeding episodes and additional endoscopic therapy can be performed. Endoscopic hemostatic techniques, like argon plasma coagulation, heater probe and laser, are the first-line treatment for both acute and chronic bleeding from GAVE; preliminary data supported the effectiveness of cryotherapy, radiofrequency and banding ligation as appropriate management options. Copyright © 2010 S. Karger AG, Basel

Portal hypertensive gastropathy (PHG) can be considered as a portal hypertension-related syndrome which affects patients with liver cirrhosis (32.7%), liver fibrosis (23.4%) or extrahepatic portal obstruction (43%) and is frequently associated with esophageal and/or gastric varices [1]. PHG, uncommon in cirrhotic patients, can however be considered as a predictor of bleeding from esophageal varices when associated with gastric varices [2], and accounts for 8% of obscure non-variceal hemorrhages in chronic liver disease, along with chronic iron deficiency anemia and high blood transfusion, with an overall mortality rate of 12.5% [3].

Pathophysiology. The mechanisms involved in the pathogenesis of PHG have not been fully elucidated, however increased gastric total blood flow, with reduction in the mucosal component and enhancement in the submucosal and muscular layers, has been demonstrated [4]. Moreover, reduction in the mucus secretion, abnormalities in gastric nitric oxide regulation as well as anomalous production of prostaglandins [4, 5], tumor necrosis factor (TNF- α), and epidermal growth factor may be involved [6]. All these elements can explain the high sensitivity of the gastric mucosa to NSAIDs and the likelihood of developing non-*Helicobacter coli*-related peptic disease [7].

Furthermore, esophageal and associated gastric varices, as well as previous endoscopic treatments (sclerotherapy 20.5%, ligation 2.3%), have been recognized as risk factors for the development of PHG [1, 8]. Anyway, the natural history of PHG, precisely defined in two prospective trials (cirrhotic patients followed for 3 years at 6-month intervals) correlates well with the severity and duration of liver disease [9]; PHG affects 80% of patients, with different evolution patterns