Management of gastric varix and portal hypertensive gastropathy

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Gastric varices (GVs) are common cause of gastrointestinal bleeding in patients of cirrhosis. Bleeding from GVs is generally more severe than bleeding from esophageal varices, but is thought to occur less frequently. GVs are classified by Sarin as: GOV1 (extending 2-5 cm below the GE junction and in continuity with the esophageal varices); GOV2 (in the fundus of the stomach and in continuity with the esophageal varices); IGV1 (isolated gastric varices in the fundus of the stomach in the absence of esophageal varices); and IGV2 (in the gastric body, antrum or pylorus). Gastric varices may also be graded based on size: small (<10 mm), medium (10-20 mm), and large (>20 mm). The presence of red signs, large size, and Child class B or C cirrhosis are important risk factors for bleeding from gastric varices. GOV1 comprises 70% of all gastric varices; however, bleeding is most common from GOV2 and IGV1 varices (i.e., gastric fundal varices). Because the blood flow in the GVs is relatively large and the bleeding is rapid and often profuse, endoscopic means of treating bleeding GVs are the treatments of choice. The choice of endoscopic therapy used often depends on local availability and expertise. GOV1 should be treated just like EVs by band ligation. For fundal varices, various endoscopic methods of treatment are available. Tissue adhesives are the first line of therapy for the control of acute GV bleeding and for prevention of GV rebleeding. Endoscopic sclerotherapy is less efficacious in achieving hemostasis, with high rebleeding rate and high incidence of local complications. VBL is an alternative method to tissue adhesives. Thrombin is effective and very safe for the control of GV bleeding and is easy to use; however, controlled trials are required to establish its role. Interventional radiological (such as TIPS or BRTO) procedures may be useful in preventing re-bleeding from GVs.

Portal hypertensive gastropathy is gastric mucosal lesions that can cause chronic gastrointestinal hemorrhage and, consequently, chronic anemia, in patients with cirrhosis. Although chronic anemia is the most common clinical manifestation, it may also lead to acute gastrointestinal bleeding. Up to 65% of patients with portal hypertension from cirrhosis will develop PHG and is often associated with the presence of esophageal and/or GVs. The mechanisms involved in the pathogenesis of PHG have not been fully elucidated; however, regulation of gastric nitric oxide, prostaglandins, tumor necrosis factor alpha, and epidermal growth factor production may be involved. The most important pharmacotherapy for prevention of bleeding from PHG involves the use of beta-blockers. Terlipressin, somatostatin and its analogue octreotide are used in therapy of bleeding PHG. Pharmacological (e.g. propranolol), or interventional radiological (such as TIPS) procedures may be useful in preventing re-bleeding from PHG.