Endoscopic Management of Nonvariceal, Nonulcer Upper Gastrointestinal Bleeding

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KEYWORDS

• UGIB • Angiodysplasia • Dieulafoy • GAVE • Gastric cancer • Hemospray • OTSC

KEY POINTS

- Although peptic ulcer and esophagogastric varices remain the most common causes of upper gastrointestinal bleeding, a quarter to half of these events is due to a range of other conditions.
- Endoscopy is the mainstay for diagnosis of these bleeds, as well as the management of the largest proportion of them.
- Endoscopic treatment makes use of the same modalities as used for peptic ulcer and varices.
- Evidence for specific endoscopic therapy per specific cause is mounting.
- The use of novel modalities such as hemostatic powder, self-expandable metal stents, and over-the-scope-clips have expanded and may replace other treatment methods.

INTRODUCTION

The endoscopic management of gastrointestinal (GI) hemorrhage consists of injection therapy (with epinephrine or cyanoacrylate and other sclerosing agents), endoscopic thermal therapy, mechanical modalities such as hemoclips and over-the-scope-clips, and more recently, topical hemostatic sprays.[1,2] Solid evidence exists on how these modalities can be used in peptic ulcer and variceal bleeds, but it is less clear for other causes. This article deals with the endoscopic management of nonvariceal, nonulcer upper gastrointestinal bleeding (UGIB).[3–6]

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EROSIVE CAUSES OF UGIB

Nonsteroidal antiinflammatory drugs are the principal cause for drug-induced erosions. They can be responsible for intramucosal petechial hemorrhage, superficial hemorrhagic erosions, gastroduodenitis, and ulceration.[7] In an endoscopic study of 187 patients using low-dose aspirin for at least 3 months with and without gastroprotective agents, erosions were observed in 34% and 63% of subjects, respectively.[8,9]

Other classes of drugs that can lead to erosions and ulceration are selective serotonin reuptake inhibitors, corticosteroids, nitrogen-containing bisphosphonates, potassium tablets, some antibiotics (eg, erythromycin, nalidixic acid, sulfonamides, and derivatives), and various chemotherapeutic agents.[7]

Larger hiatal hernia can result in linear erosions and ulcers or so-called *Cameron lesions* within the stomach at the impression of the diaphragm.[10] They predominantly occur along the lesser curvature. They most likely occur as a result of the combination of chronic mechanical trauma (eg, rubbing of the mucosal folds at the level of the diaphragm during respiratory excursions) and acid injury. Local ischemia may also play a role. Cameron lesions are found in about 5% of patients with hiatal hernia undergoing upper endoscopy; two-thirds of these patients have multiple lesions.[11] They may occur in 10% to 20% of patients with a hernia greater than or equal to 5 cm.[12]

Erosive esophago-gastro-duodenitis has many possible causes such as excessive alcohol consumption, use of mucosal erosion–causing drugs (see earlier discussion),[13] gastroesophageal reflux, and *Helicobacter pylori* infection.[14–16] Gastroduodenal erosions infrequently cause clinically significant blood loss.[3,5,14,17–20]

Endoscopic Management of Erosive Causes of UGIB

Endoscopy has only a marginal role in the treatment of hemorrhage caused by erosions. The treatment of choice is acid suppression, leading to healing of erosions and normalization of hemoglobin levels. The outcome is generally excellent. Acute bleeds sometimes require endoscopic treatment such as of a visible vessel, for which the treatment is similar to the approach of gastroduodenal ulcers. A different approach may be the control of bleeding by a topical hemostatic spray. Hemospray (Cook Medical, Winston Salem, NC, USA) is a novel proprietary powder specifically developed for the treatment of UGIB.[21,22] It is a nonorganic mineral blended powder that is thought to work via absorption of liquids at the bleeding site, forming an adhesive and cohesive mechanical barrier over the bleeding site. In a large European observational study in 63 patients with UGIB due to various causes, the application of Hemospray as primary monotreatment led to immediate hemostasis in 85% of patients with a 7-day rebleeding rate of 15%. When used as salvage therapy, after failure of conventional endoscopic treatment modalities, initial hemostasis was 100% and rebleeding 25%, reflecting the refractory nature of these lesions. Causes of nonvariceal, nonulcer UGIB included erosive esophagitis, gastritis, and/or duodenitis (n = 7), as well as Dieulafoy lesions, gastric antral vascular ectasia (GAVE), and Mallory-Weiss lesions.[23] Concomitant use of antithrombotics, mostly causative for erosive UGIB, do not appear to influence the success rates of Hemospray.[22]

UGIB CAUSED BY VASCULAR ANOMALIES

Vascular anomalies that most commonly cause UGIB include angiodysplasia, Dieulafoy lesions, and GAVE. Although not a true anomaly, but rather the result of congestion, portal hypertensive gastropathy is also ranked in this same category of vascular lesions.