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Evaluation and management of Non-variceal upper gastrointestinal bleeding

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ABSTRACT

Non-variceal upper gastrointestinal bleeding continues to be an important cause of morbidity and mortality. The most common causes include peptic ulcer disease, Mallory–Weiss syndrome, erosive gastritis, duodenitis, esophagitis, malignancy, angiodysplasias and Dieulafoy's lesion. Initial assessment and early aggressive resuscitation significantly improves outcomes. Upper gastrointestinal endoscopy continues to be the gold standard for diagnosis and treatment. We present a comprehensive review of literature for the evaluation and management of non-variceal upper gastrointestinal bleeding.

Introduction

Upper gastrointestinal bleeding is defined as bleeding proximal to the ligament of Treitz without evidence of esophageal, gastric, and duodenal varices.[1] Gastrointestinal bleeding (GIB) is a

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significant public health issue costing nearly five billion dollars annually to the health system.[2] Although, the incidence rates vary widely based on geographic location, prior studies have consistently shown higher incidence among males and the elderly population.[3] In 2012, approximately 1,80,767 patients were hospitalized with UGIB.[2] Recent studies have shown a decrease in mortality which is likely due to the advent of proton pump inhibitors (PPI), improved detection and treatment of *Helicobacter pylori* (*H. pylori*), and advances in endoscopic and interventional radiologic techniques.[3,4] The most common cause continues to be peptic ulcers, although the incidence has declined.[5] Despite endoscopy being the definitive diagnostic and therapeutic modality, a fundamental knowledge of the different etiologies and appropriate management is needed for emergency medicine physicians, intensivists, surgeons, interventional radiologists, internists, and gastroenterologists.

Etiology

Peptic ulcers are mucosal erosions that extend through the muscularis mucosa into deeper layers of the wall. Peptic ulcers are the most common form of non-variceal upper gastrointestinal bleeding (NVUGIB) accounting for about 31–67% of upper gastrointestinal bleeding.[5–8] The recent decline in incidence of peptic ulcers is thought to be due to increase in PPI use, affective *H. pylori* treatment, and awareness of nonsteroidal anti-inflammatory drugs (NSAIDS) as a cause of peptic ulcers. Peptic ulcers can be present in either the stomach or bulb of duodenum and are most commonly caused by *H. pylori* and NSAID use. *H. pylori* attaches to the gastric and duodenal mucosa inducing tissue injury by several mechanisms.[9] NSAIDS continue to be a common cause for peptic ulcers.[10,11] Injury is caused by the local and systemic effects of prostaglandin inhibition.[12]

Other causes of upper GIB include esophagitis, gastritis, and duodenitis which are inflammatory processes that can further progress to ulcerations and have similar risk factors as peptic ulcers[13] (Table). Mallory–Weiss syndrome is caused by longitudinal mucosal tears at the gastroesophageal junction or gastric cardia due to a rapid increase in intraabdominal pressure which is usually caused by vomiting or retching.

Vascular lesions including angiodysplasia, Dieulafoy's lesions, and gastric antral vascular ectasia (GAVE) cause 2–8% of NVUGIB.[5,6,8] Angiodysplasias, vascular ectasia, or arteriovenous malformations (AVM) can be used synonymously and refer to thin-walled tortuous vessels found throughout the gastrointestinal tract. The pathogenesis of AVMs is not completely understood, but they are frequently found in individuals with aortic stenosis (Heyde's syndrome), Von Willebrand disease, and chronic renal failure.[14] Dieulafoy's lesions are dilated submucosal arteries (1–5 mm) that infiltrate into the mucosa in the absence of an overlying ulceration.[15] The exact mechanism of Dieulafoy's lesions is not completely understood at this time, but several mechanisms have been proposed. It is seen more commonly in men and patients with comorbidities that include DM, HTN, cardiovascular diseases, and chronic kidney disease.[15–18] Neoplasms of the upper gastrointestinal tract can arise from any type of cell line whether local or metastatic, and can also lead to upper GIB due to superficial erosions or via invasion into the vasculature.

Table
Etiologies of non-variceal upper gastrointestinal bleeding.

Etiology	Frequency
Peptic ulcer	26–59%
Mallory–Weiss tear	7–12%
Erosive gastritis/duodenitis	7–28%
Esophagitis	4–12%
Malignancy	4–6%
Angiodysplasia	2–8%
Other	2–11%