Acid Peptic Disease

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KEYWORDS

• Peptic ulcer disease • Bleeding ulcer • Perforated ulcer • Obstructing ulcer
• *Helicobacter pylori*

KEY POINTS

- The surgical management of peptic ulcer disease has changed drastically due to advances in acid suppression therapy and the discovery and treatment of *Helicobacter pylori*.
- Complications of peptic ulcer disease include bleeding, perforation, and obstruction and are still a significant cause of morbidity and mortality.
- Surgical management is rarely necessary in patients bleeding from peptic ulcer disease.
- Because of advances in medical, endoscopic, and angiographic therapy, surgery is most often used in the emergent setting of a patient with a perforated ulcer.

INTRODUCTION: NATURE OF THE PROBLEM

As the understanding of the pathophysiology of peptic ulcer disease (PUD) developed through the 1970s and 1980s, surgical treatment has become less frequent. The major decline has been in elective surgery for intractable disease, but the number of emergent operations has also decreased.[1] The annual incidence of PUD requiring medical or surgical treatment ranges between 0.10% and 0.19% and is declining.[2,3] Despite this decline, the complications of PUD (which include bleeding, perforation, and obstruction) still account for approximately 150,000 hospital admissions per year in the United States.[4] Although bleeding is the most common complication (ratio of 6:1), perforation carries the highest mortality risk of up to 30%.[4]

PATHOPHYSIOLOGY

Mucosal disruption in patients with acid peptic disease can be due to either infection, barrier disruption, or gastric acid hypersecretion. Risk factors for developing PUD include *Helicobacter pylori* infection, alcohol consumption, tobacco use, cocaine and amphetamine use, nonsteroidal anti-inflammatory drugs (NSAIDs), fasting,

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Zollinger-Ellison syndrome, cancer treatment with angiogenesis inhibitors, and bariatric surgery (Fig. 1).[4]

PUD in most patients is a result of *H pylori* infection or chronic NSAID or aspirin use. *H pylori* infection causes both a direct bacterial effect and a secondary host inflammatory response inflicting damage to the mucosa of the stomach and duodenum. Of the patients infected with *H pylori*, 10% to 15% will have hypersecretion of gastric acid leading to antral or duodenal ulcers secondary to inhibition of somatostatin secretion, thereby stimulating gastrin release. The remaining majority of patients infected with *H pylori* will have gastric ulcers associated with hypochlorhydria and mucosal atrophy. NSAIDs damage the gastric mucosa by inhibiting Cyclooxygenase-1 prostaglandins, which provide a protective effect on the gastric mucosa.[2]

Most peptic ulcers heal with gastric acid suppression, most commonly by administration of a proton pump inhibitor (PPI) alone or with *H pylori* treatment for 6 to 8 weeks. More than 85% of NSAID-induced ulcers will heal within 6 to 8 weeks after cessation of the offending drug along with gastric acid suppression.[2] The effectiveness of this

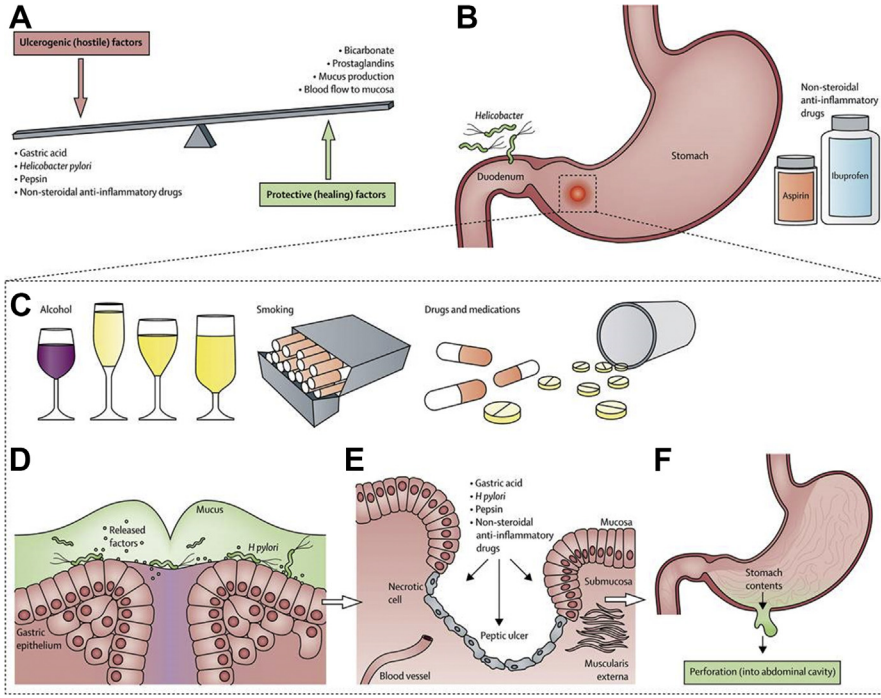

Fig. 1. Mechanisms and factors in pathogenesis of perforated peptic ulcer. (*A*) An imbalance between hostile and protective factors start the ulcerogenic process, and (*B*) although many cotributors are known, *Helicobacter* infection and use of NSAIDs appear of importance in disturbing the protective mucosal layer and (*C*) expose the gastric epithelium to acid. Several additional factors (*D*) may augment the ulcerogenic process (such as smoking, alcohol use, and use of several drugs) that leads to erosion (*E*). Eventually, the serosal lining is breached (*F*), and when perforated, the stomach content, including acidic fluid, will enter the abdominal cavity, giving rise to intense pain, local peritonitis that may become generalized, and eventually lead to a systemic inflammatory response syndrome and sepsis with the risk of multiorgan failure and mortality. (*Adapted from* Søreide K, Thorsen K, Harrision EM, et al. Perforated peptic ulcer. Lancet 2015;386(10000):1291; with permission.)