Peptic Ulcer Disease: Fire in the Hole! **Twitter/Instagram: @emboardbombs**

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Author: Chris Musselwhite, MD
Peer Reviewer: Blake Briggs, MD; Travis Smith DO

Introduction

While peptic ulcer disease (PUD) cases are overall decreasing, with an estimated prevalence of 5-10% and incidence 0.1 - 0.3% per year, mortality for PUD complications has remained around 5-10%.[1]

Chronic non-steroidal anti-inflammatories (NSAIDs) and *Helicobacter pylori* (*H. pylori*) infection are the main two risk factors for PUD. Only 20% of PUD cases are *H. pylori*/NSAID negative. However, it's not *just H. pylori* and NSAIDs, because only small percentage of patients with *H. pylori* and NSAID use develop ulcers. Other factors such as genetics, smoking, alcohol use, and even stress contribute to ulcer formation.

Quick pathophysiology (and super nerdy stuff)

H. pylori is a gram-negative, spiral-shaped bacteria with special adaptations to live in the acidic environment of our stomachs. *H. pylori* is a common and ancient infection, found in nearly 50% of all human stomachs, including the 5,300-year-old ice mummy Otzi.[2] In fact, a closely related species, *H. acinonychis*, is found in the stomachs of large African cats. The large cats were likely infected from snacking on ancient humans (yeah, good job ancient humans for giving ulcers to lions, that'll teach 'em!).[3] The hypothesized pathophysiology of ulcer formation by *H. pylori* is complicated, involving a complex interplay of gastric acid secretion, impaired mucosal defenses, and inflammation.

NSAIDs, on the other hand, cause ulceration by suppressing prostaglandin via cyclooxygenase inhibition. Prostaglandins contribute to gastric mucosal protections. Patients taking chronic NSAIDs have a reduced risk of developing PUD and upper gastrointestinal bleeding (UGIB) if they take a selective COX inhibitor, like celecoxib. This risk is even less if it's combined with a proton pump inhibitor. There are also other medications that increase the risk of UGIB, such as selective serotonin inhibitors, corticosteroids, and aldosterone antagonists.[1]

Signs and Symptoms

Classic symptoms include pain in the epigastric region around two to five hours after eating when acid is secreted in the absence of a food buffer. Most studies note around 80% of patients with PUD having epigastric pain. The pain may radiate to the back and is usually worse at night, roughly 11 pm to 2 am) when the circadian pattern of acid secretion is maximal. Patients also report associated belching, fullness, early satiety, nausea and/or vomiting. They tend to avoid fatty foods as this worsens their symptoms. Additionally, gastroesophageal reflux disease (GERD) can occur concomitantly with one study showing 46% of PUD patients having GERD symptoms.[4]

However, patients can also present with NONE of the classic symptoms, but with horrendous upper gastrointestinal bleeding, especially in the elderly or those taking NSAIDs.

In fact, bleeding is the most common complication, accounting for 40-60% of all acute UGIBs, but there are other complications.[5]

Ulcers love to burrow their way into things, such as the stomach wall, causing a perforation.

Perforation, occurring between 4 to 14 cases per 100,000 individuals,[6] is usually associated with severe, diffuse abdominal pain, and peritoneal signs such as a rigid abdomen with rebound and guarding. Mortality can be as high as 20%![1]

Ulcers can also burrow between organs creating fistulous tracts connecting the stomach to the duodenum, or stomach/duodenum to the colon. The ulcer can even burrow and turn into a walled-off abscess, or to vasculature resulting in a truly massive UGIB, to the biliary tree resulting in biliary obstruction, or even the pancreatic duct resulting in pancreatitis. As you can see, ulcers can tear their way through to any adjacent structure.

Workup, Treatment, and Disposition

Patients with peptic ulcer disease can present from stable to critically ill.

Any patient with a massive UGIB with either brisk hematochezia or massive hemoptysis will need large bore IV access. Remember, this is not the time for a triple lumen catheter given its long length and smaller diameter impeding rapid fluid flow). These patients will also likely need emergency blood transfusions. The patient should be started on high-dose PPI (usually 40 mg of pantoprazole IV, with studies now favoring intermittent bolus therapy being equally as efficacious as continuous infusion.[7]

Ultimately, these patients will need an emergent GI consult and endoscopy.

Patients with suspected perforation presenting with peritoneal signs and/or septic shock will require emergent surgical consult. This critical subset of patients will need large-bore IV access, intravenous fluids, and broad-spectrum antibiotics. The antibiotic choices may vary according to your specialist preference, but the choice(s) should cover gram-negative and anaerobic organisms (i.e., third or fourth-generation cephalosporin plus metronidazole or piperacillin/tazobactam). Advanced imaging with computed tomography with contrast could be considered depending on patient stability, but an upright chest x-ray to evaluate for air under the diaphragm can quickly and easily be obtained, with sensitivities up to 90% in some studies. Forget the abdominal x-ray. An upright chest x-ray is much more accurate.

Stable patients without vital sign abnormalities, but with risk factors for peptic ulcer disease and classic signs or symptoms should be worked up as needed to exclude other emergent causes of abdominal pain. *There is no definitive test in the ED to diagnose peptic ulcer disease.*

For patients with likely PUD, they will need urgent GI consult or close GI follow-up depending on the circumstances. If you find yourself on the fence regarding admission vs discharge for these patients, one validated scoring system available is the AIMS65 Score, with a score of zero associated with 0.3% in-hospital mortality and a score of 5 (maximum) associated with 24.5% in-hospital mortality for patients with UGIB.

If you are discharging from the ED, patients should be started on a PPI, with most cases healing in 6 to 8 weeks of treatment.[1] The treatment length and dosing depend on the ulcer type (uncomplicated vs complicated) and the location (gastric vs duodenal) as some complicated gastric ulcers need 8-12 weeks of therapy vs only 4-8 weeks of a comparable duodenal ulcer.
Histamine-2 blockers are less effective at ulcer healing than PPIs.

Further testing and treatment will be needed as an outpatient to determine the presence of H. pylori infection (such as urea breath test, stool antigen testing, or other tests from a gastric mucosa tissue sample obtained via endoscopy) and initiation of triple antibiotic therapy (clarithromycin, amoxicillin, and PPI) vs bismuth quadruple therapy (bismuth, metronidazole, tetracycline, PPI).

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