

Peptic Ulcer Disease: Review Questions

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QUESTIONS

Choose the single best answer for each question.

- A 34-year-old man presents to the emergency department (ED) with intermittent melena of 3 days' duration. He is mildly fatigued but hemodynamically stable and denies any hematemesis or coffee ground emesis. His serum hemoglobin level is 8.2 g/dL. Intravenous (IV) fluids are started. Physical examination is essentially unremarkable. What is the next best step in this patient's evaluation?

 - Check serum *Helicobacter pylori* antibody levels
 - Perform a colonoscopy
 - Perform an esophagogastroduodenoscopy (EGD)
 - Start a histamine₂ receptor antagonist (H₂ blocker)
 - Transfuse 2 U of packed red blood cells
- A 75-year-old man presents to his primary care physician with a painful right great toe. Past medical history is remarkable for recent deep venous thrombosis currently treated with warfarin and hypertension treated with an angiotensin-converting enzyme (ACE) inhibitor and a calcium channel blocker. On examination, he appears to be having an attack of gout. The patient is prescribed indomethacin 50 mg orally 3 times daily and colchicine 0.6 mg orally 3 times daily. The patient calls the office the next day to report that he is feeling better, and his physician advises him to continue with the medication regimen. Two days later, the patient is admitted to the intensive care unit with a life-threatening upper gastrointestinal (GI) bleed. Endoscopy reveals several deep and actively bleeding gastric ulcers, which are treated to good effect. What is the most likely cause of this patient's bleeding event?

 - Failure to stop the ACE inhibitor prior to starting therapy for gout
 - Failure to stop the calcium channel blocker prior to starting therapy for gout
 - Failure to stop warfarin prior to starting therapy for gout
 - The addition of colchicine to his regimen
 - The addition of indomethacin to his regimen
- A 45-year-old man with a history of bleeding duodenal ulcers presents to his primary care physician with abdominal pain. Two years ago, the patient was found to have *H. pylori* infection and was treated with lansoprazole 30 mg twice daily, amoxicillin 1 g twice daily, and clarithromycin 500 mg twice daily for 14 days. He states that he was compliant with the medications. At this visit, he reports that his abdominal pain is similar to what he experienced during the prior episode. A gastroenterology consult is obtained, and upper endoscopy reveals 2 small ulcers in the duodenal bulb that do not require endoscopic treatment. Gastric biopsies obtained at the time of the procedure reveal the presence of *H. pylori*. What is the most likely explanation for the presence of *H. pylori* in this patient's stomach?

 - Failure of the original treatment regimen 2 years ago
 - False-positive result on the gastric biopsy
 - Incorrect drug choices 2 years ago
 - Incorrect drug doses 2 years ago
 - Repeat infection following eradication of prior infection
- A 68-year-old man presents to the ED with coffee ground emesis, abdominal pain, weakness, and nausea. He states that he has had 3 episodes of coffee ground emesis over the past 2 hours. After appropriate resuscitation, an upper endoscopy is performed, which reveals a deep ulcer along the lesser curvature of the stomach (**Figure**). There appears to be a visible vessel in the center of the ulcer. The remainder of the endoscopic examination is normal. What is the next best step in this patient's management?

 - Begin IV octreotide
 - Endoscopic therapy
 - Insert a nasogastric tube
 - Observation, nothing by mouth status, and IV proton pump inhibitor (PPI)
 - Surgical consultation

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Figure. Deep ulcer along the lesser curvature of the stomach with a visible vessel in the center of the ulcer found during upper endoscopy in the patient described in question 4.

5. A 21-year-old college football player presents to the ED for copious hematemesis. He recently started taking ibuprofen 800 mg 3 times a day for muscle and joint aches and pains. After IV fluids are started, an upper endoscopy is performed; however, the stomach and proximal intestine is full of fresh and clotted blood, and the gastroenterologist is unable to adequately visualize the gastric or duodenal mucosa. An IV PPI is started and a nasogastric tube is placed, but the thick blood and clots are difficult to aspirate through the tube. What is the next best step in this patient's management?
- Administer IV erythromycin 250 mg and repeat endoscopy in 1 hour
 - Administer IV levofloxacin 500 mg and repeat endoscopy in 1 hour
 - Induce vomiting to clear the stomach
 - Repeat endoscopy in 24 hours after the blood has had time to pass into the distal small bowel
 - Surgical evaluation
6. A 56-year-old woman presents to the ED with dehydration, abdominal pain, nausea, and vomiting. The patient states that her vomit appears to contain flecks of blood. She is borderline hypotensive but otherwise hemodynamically stable. The abdominal examination is remarkable for mild epigastric tenderness to deep palpation, but there are no peritoneal signs. IV fluids are administered and the patient's blood pressure normalizes. EGD is performed, which reveals what appears to be partial gastric outlet obstruction at the level of the pylorus
- due to several benign-appearing, clean-based ulcers in the pyloric channel. A rapid urease test to assess for the presence of *H. pylori* is negative. The ulcers do not warrant endoscopic therapy. What is the next best step in this patient's management?
- Endoscopic dilation of the stenosed pylorus
 - Endoscopic placement of a transpyloric stent
 - High-dose H₂ blocker
 - High-dose PPI therapy
 - Surgical evaluation

ANSWERS AND EXPLANATIONS

- (C) Perform an EGD.** The patient has melena, which suggests GI bleeding from a source proximal to the ligament of Treitz. Based on the patient's serum hemoglobin level, he has likely lost significant blood over the past several days and needs to be evaluated for the presence of a bleeding ulcer or other causes of upper GI bleeding. EGD allows for definitive endoscopic evaluation of the esophagus, stomach, and proximal duodenum. Any ulcers identified that warrant endoscopic therapy (eg, cauterization, clipping) could be treated at that time. *H. pylori* infection could also be assessed via gastric biopsy during the EGD. Serum *H. pylori* testing would be helpful, but this takes time and the patient needs endoscopy first and foremost to identify the source of the bleeding. The patient does not need blood transfusion at this time given that he is hemodynamically stable. H₂ blockers would be inadequate for a patient with a suspected bleeding ulcer. Colonoscopy is unlikely to identify the source of bleeding in a patient with melena.
- (E) The addition of indomethacin to his regimen.** Indomethacin is a potent nonsteroidal anti-inflammatory medication, and the ulcerogenic effects of such medications are well established. Given that the patient was also taking an oral anticoagulant (warfarin), it is not surprising that he developed a significant episode of GI bleeding from gastric ulcers. The addition of an oral PPI to the patient's regimen as GI prophylaxis would likely have avoided this situation. Given the patient's medical history, it would have been inappropriate to stop warfarin, the ACE inhibitor, or calcium channel blocker before starting therapy for gout. Colchicine is not typically associated with the development of gastric ulcers.
- (A) Failure of the original treatment regimen 2 years ago.** The patient was treated with an appropriate (and perhaps the most common) regimen for *H. pylori*

infection 2 years ago with regards to both medications and dosages. The most likely cause of the patient's current presentation is failure of the regimen to eradicate the organism. Failure of initial treatment of *H. pylori* can occur in up to 20% of patients.¹ Re-infection is possible but far less likely than persistence of the original infection. A false-positive result on the gastric biopsy is also possible but is very unlikely given that gastric biopsy showing the presence of the organism is the gold standard for diagnosis of *H. pylori* infection.

4. **(B) Endoscopic therapy.** The patient has a high-risk lesion—an ulcer with a visible vessel—and the risk of rebleeding over the next 24 hours is very high. Endoscopic therapy in the form of mechanical therapy (eg, clips), thermal therapy (eg, electrocautery), or injection (eg, epinephrine) is successful for treating ulcers in the majority of cases. Surgical consultation would be warranted if endoscopic therapy failed or if the ulcer had perforated the stomach. A nasogastric tube would add little benefit at this point in the patient's course given that there is no active bleeding and the tube could traumatize the ulcer and produce further bleeding. IV octreotide, which reduces splanchnic blood flow, is routinely used in patients with variceal bleeding, but its use in patients with peptic ulcer bleeding is controversial. Given the high-risk nature of the lesion, observation combined with nothing by mouth status and IV PPI therapy would be inadequate.
5. **(A) Administer IV erythromycin 250 mg and repeat endoscopy in 1 hour.** The blood in this patient's stomach is precluding endoscopic visualization. Erythromycin is a macrolide antibiotic with potent gastric promotility effects. One dose of IV erythromycin has

been shown to promote gastric emptying,² which can clear gastric contents including blood and improve endoscopic visualization in many patients. This therapy is inexpensive and can be performed quickly. Levofloxacin is a fluoroquinolone antibiotic and does not have promotility properties comparable with those of erythromycin. The patient is experiencing hematemesis and cannot wait 24 hours to repeat the examination. Inducing vomiting could lead to aspiration and should be avoided. Surgical evaluation should be deferred at this time but could be considered if IV erythromycin and the repeat endoscopic procedure are unsuccessful.²

6. **(D) High-dose PPI therapy.** The patient has developed gastric outlet obstruction as a consequence of peptic ulcers. This is a rare but serious complication. First-line therapy is high-dose PPI therapy, preferably administered intravenously, to treat the gastric inflammation and the ulcers themselves. H₂ blockers would not provide adequate acid suppression. Dilation could be considered as a second-line approach if the patient does not respond to PPI therapy. Dilation in the acute phase may precipitate bleeding of the ulcers. Surgical evaluation could be considered if both medical and endoscopic therapy fail to relieve the patient's gastric outlet obstruction. Gastroduodenal stenting is generally contraindicated for benign disease and is reserved for malignant obstruction and should thus be avoided.³

REFERENCES

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3. Boylan JJ, Gradzka MI. Long-term results of endoscopic balloon dilatation for gastric outlet obstruction. *Dig Dis Sci* 1999;44:1883–6.

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