Peptic Ulcer Disease

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Peptic ulcer disease usually occurs in the stomach and proximal duodenum. The predominant causes in the United States are infection with *Helicobacter pylori* and use of nonsteroidal anti-inflammatory drugs. Symptoms of peptic ulcer disease include epigastric discomfort (specifically, pain relieved by food intake or antacids and pain that causes awakening at night or that occurs between meals), loss of appetite, and weight loss. Older patients and patients with alarm symptoms indicating a complication or malignancy should have prompt endoscopy. Patients taking nonsteroidal anti-inflammatory drugs should discontinue their use. For younger patients with no alarm symptoms, a test-and-treat strategy based on the results of *H. pylori* testing is recommended. If *H. pylori* infection is diagnosed, the infection should be eradicated and antisecretory therapy (preferably with a proton pump inhibitor) given for four weeks. Patients with persistent symptoms should be referred for endoscopy. Surgery is indicated if complications develop or if the ulcer is unresponsive to medications. Bleeding is the most common indication for surgery. Administration of proton pump inhibitors and endoscopic therapy control most bleeds. Perforation and gastric outlet obstruction are rare but serious complications. Peritonitis is a surgical emergency requiring patient resuscitation; laparotomy and peritoneal toilet; omental patch placement; and, in selected patients, surgery for ulcer control. (Am Fam Physician 2007;76:1005-12, 1013. Copyright © 2007 American Academy of Family Physicians.)

▶ **Patient information:** A handout on peptic ulcers, written by the authors of this article, is provided on page 1013.

Peptic ulcer disease is a problem of the gastrointestinal tract characterized by mucosal damage secondary to pepsin and gastric acid secretion. It usually occurs in the stomach and proximal duodenum; less commonly, it occurs in the lower esophagus, the distal duodenum, or the jejunum, as in unopposed hypersecretory states such as Zollinger-Ellison syndrome, in hiatal hernias (Cameron ulcers), or in ectopic gastric mucosa (e.g., in Meckel's diverticulum).

Approximately 500,000 persons develop peptic ulcer disease in the United States each year.[1] In 70 percent of patients it occurs between the ages of 25 and 64 years.[2] The annual direct and indirect health care costs of the disease are estimated at about $10 billion.[1] However, the incidence of peptic ulcers is declining, possibly as a result of the increasing use of proton pump inhibitors and decreasing rates of *Helicobacter pylori* infection.[3]

Causes of Peptic Ulcer Disease

H. pylori infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs) are the predominant causes of peptic ulcer disease in the United States, accounting for 48 and 24 percent of cases, respectively *(Table 1)*.[4] A variety of other infections and comorbidities are associated with a greater risk of peptic ulcer disease (e.g., cytomegalovirus, tuberculosis, Crohn's disease, hepatic cirrhosis, chronic renal failure, sarcoidosis, myeloproliferative disorder). Critical illness, surgery, or hypovolemia leading to splanchnic hypoperfusion may result in gastroduodenal erosions or ulcers (stress ulcers); these may be silent or manifest with bleeding or perforation.[5] Smoking increases the risk of ulcer recurrence and slows healing.

H. PYLORI

Although *H. pylori* is present in the gastroduodenal mucosa in most patients with duodenal ulcers, only a minority (10 to 15 percent) of patients with *H. pylori* infection develop peptic ulcer disease.[6] *H. pylori* bacteria adhere to the gastric mucosa; the presence of an outer inflammatory protein and a functional cytotoxin-associated gene island in the bacterial chromosome increases virulence and probably ulcerogenic potential.[7] Patients with *H. pylori* infection have increased resting and meal-stimulated gastrin levels and decreased gastric mucus production and duodenal mucosal bicarbonate secretion, all of which favor ulcer formation. Eradication of *H. pylori* greatly reduces the incidence of ulcer recurrence—from 67 to 6 percent in

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	*Evidence rating*	*References*
Prompt upper endoscopy is recommended for patients with peptic ulcers who are older than 55 years, those who have alarm symptoms, and those with ulcers that do not respond to treatment.	A	1, 19
In patients with peptic ulcer disease, *Helicobacter pylori* should be eradicated to assist in healing and to reduce the risk of gastric and duodenal ulcer recurrence.	A	1, 8
In patients with peptic ulcers, proton pump inhibitors provide acid suppression, healing rates, and symptom relief superior to other antisecretory therapies.	A	23
Patients with bleeding peptic ulcers should be given a proton pump inhibitor to reduce transfusion requirements, need for surgery, and duration of hospitalization. *H. pylori* testing should be performed and eradication therapy prescribed if results are positive.	A	32, 34
In patients with perforated ulcers, coexisting *H. pylori* infection should be eradicated to minimize the need for long-term antisecretory therapy and further surgical intervention.	C	25, 37

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, see page 922 or http://www.aafp.org/afpsort.xml.

patients with duodenal ulcers and from 59 to 4 percent in patients with gastric ulcers.[8]

NSAIDS

NSAIDs are the most common cause of peptic ulcer disease in patients without *H. pylori* infection.[9] Topical effects of NSAIDs cause submucosal erosions. In addition, by inhibiting cyclooxygenase, NSAIDs inhibit the formation of prostaglandins and their protective cyclooxygenase-2–mediated effects (i.e., enhancing gastric mucosal protection by stimulating mucus and bicarbonate secretion and epithelial cell proliferation and increasing mucosal blood flow). Coexisting *H. pylori* infection increases the likelihood and intensity of NSAID-induced damage.[10]

The annual risk of a life-threatening ulcer-related complication is 1 to 4 percent in patients who use NSAIDs long-term, with older patients at the highest risk.[11] NSAID use is responsible for approximately one half of perforated ulcers, which occur most commonly in older patients who are taking aspirin or other NSAIDs for cardiovascular disease or arthropathy.[12,13] Other risk factors for NSAID-related ulcers are listed in *Table 1*.[4] Proton pump inhibitors and misoprostol (Cytotec) minimize the ulcerogenic potential of NSAIDs and reduce NSAID-related ulcer recurrence.

Table 1. Causes of Gastroduodenal Ulcers

Cause	*Comments*
Common	
Helicobacter pylori infection	Gram-negative, motile spiral rod found in 48 percent of patients with peptic ulcer disease[4]
NSAIDs	5 to 20 percent of patients who use NSAIDs over long periods develop peptic ulcer disease
	NSAID-induced ulcers and complications are more common in older patients, patients with a history of ulcer or gastrointestinal bleeding, those who use steroids or anticoagulants, and those with major organ impairment
Other medications	Steroids, bisphosphonates, potassium chloride, chemotherapeutic agents (e.g., intravenous fluorouracil)
Rare	
Acid-hypersecretory states (e.g., Zollinger-Ellison syndrome)	Multiple gastroduodenal, jejunal, or esophageal ulcers
Malignancy	Gastric cancer, lymphomas, lung cancers
Stress	After acute illness, multiorgan failure, ventilator support, extensive burns (Curling's ulcer), or head injury (Cushing's ulcer)

NSAID = nonsteroidal anti-inflammatory drug.

Information from reference 4.

Diagnosis of Peptic Ulcer Disease

The diagnosis of peptic ulcer disease is usually based on clinical features and specific testing, although it is important to be aware that individual signs and symptoms are relatively unreliable.

CLINICAL FEATURES

Typical symptoms of peptic ulcer disease include episodic gnawing or burning epigastric pain; pain occurring two to five hours after meals or on an empty stomach; and nocturnal pain relieved by food intake, antacids, or antisecretory agents. A history of episodic or epigastric pain, relief of pain after food intake, and nighttime awakening because of pain with relief following food intake are the most specific findings for peptic ulcer and help rule in the diagnosis.[14] Less common features include indigestion, vomiting, loss

of appetite, intolerance of fatty foods, heartburn, and a positive family history.[14] The physical examination is unreliable—in one study, tenderness to deep palpation reduced the likelihood of ulcer.[14]

The natural history and clinical presentation of peptic ulcer disease differ in individual populations *(Table 2[6,15-18])*.[15] Abdominal pain is absent in at least 30 percent of older patients with peptic ulcers.[16] Postprandial epigastric pain is more likely to be relieved by food or antacids in patients with duodenal ulcers than in those with gastric ulcers. Weight loss precipitated by fear of food intake is characteristic of gastric ulcers.

Table 2. Peptic Ulcer Disease in Different Populations

Population	*Features*
Children	*Incidence:* Rare; most ulcers occur between eight and 17 years of age; duodenal ulcer up to 30 times more common than gastric ulcer
	Cause: Helicobacter pylori infection contributory
	Presentation: Patients may present with poorly localized abdominal pain
	Testing: EGD should be performed if ulcer suspected; test-and-treat strategy not recommended; *H. pylori* testing and treatment recommended only if ulcer is documented by EGD or contrast studies
	Treatment: Antisecretory agents
	Complications: 25 percent of bleeding duodenal ulcers may be silent; perforation and penetration rare
Older patients	*Presentation:* More likely to have painless ulcers; 50 percent present acutely (e.g., with perforation); may present with nonspecific complaints (e.g., confusion, restlessness, abdominal distention, fall)
	Complications: Perforations associated with mortality three times higher than in younger patients; hemorrhagic complications more likely (20 percent from silent ulcers); more likely to have continued bleeding and to need transfusions and surgery
Patients with stress ulcers	*Cause:* Breakdown of mucosal protectants as a result of stress leads to splanchnic hypoperfusion and ulcer; risk factors include mechanical ventilation longer than 48 hours, burns, coagulopathy, moderate to severe trauma, head or spinal cord injury, liver failure, and organ transplantation
	Presentation: Patients may be asymptomatic or may develop bleeding or perforation
	Treatment: Early institution of PPI prophylaxis with oral or intravenous pantoprazole (Protonix) minimizes ulcer risk; histamine H_2 blockers and sucralfate (Carafate) are other options for prophylaxis
Pregnant women	*Presentation:* Ulcer symptoms milder and may improve during pregnancy; vomiting is nocturnal or postprandial and worse in third trimester
	Testing: Ultrasonography and EGD are safe diagnostic tests
	Treatment: Early, aggressive treatment with PPI recommended; misoprostol (Cytotec) contraindicated; *H. pylori* infection treated as usual; avoid tetracyclines throughout pregnancy and metronidazole (Flagyl) during first trimester
	Complications: Infrequent; hypotension treated vigorously to minimize placental hypoperfusion; risk of miscarriage, abruption, and preterm labor if complications ensue

EGD = esophagogastroduodenoscopy; PPI = proton pump inhibitor.

Information from references 6 and 15 through 18.

EVALUATION

If the initial clinical presentation suggests the diagnosis of peptic ulcer disease, the patient should be evaluated for alarm symptoms. Anemia, hematemesis, melena, or heme-positive stool suggests bleeding; vomiting suggests obstruction; anorexia or weight loss suggests cancer; persisting upper abdominal pain radiating to the back suggests penetration; and severe, spreading upper abdominal pain suggests perforation. Patients older than 55 years and those with alarm symptoms should be referred for prompt upper endoscopy. Esophagogastroduodenoscopy (EGD) is more sensitive and specific for peptic ulcer disease than upper gastrointestinal barium studies and allows biopsy of gastric lesions.[19]

Patients younger than 55 years with no alarm symptoms should be tested for *H. pylori* infection and advised to discontinue the use of NSAIDs, smoking, alcohol, and illicit drug use. Presence of *H. pylori* can be confirmed with a serum enzyme-linked immunosorbent assay (ELISA), urea breath test, stool antigen test, or endoscopic biopsy *(Table 3[1,19,20])*. Serum ELISA is the least accurate test and is useful only for diagnosing the initial infection. The stool antigen test is less convenient but is highly accurate and can also be used to confirm *H. pylori* eradication, as can the urea breath test.[19]

If test results are positive for *H. pylori*, the infection should be eradicated and antisecretory therapy, preferably with a proton pump inhibitor, administered for four weeks[1,19] *(Figure 1)*. Further management is based on the endoscopic or radiologic diagnosis. Patients with persistent symptoms should be referred for endoscopy to rule out refractory ulcer and malignancy.

Management of Peptic Ulcer Disease

Treatment of peptic ulcer disease should include eradication of *H. pylori* in patients with this infection *(Table 4[19,21-25])*. The recommended duration of therapy for eradication is 10 to 14 days; however, shorter treatment courses (regimens of one, five, and seven days) are

Table 3. Tests Used in the Diagnosis of Peptic Ulcer

Test	*Comments*
EGD	Indicated in patients with evidence of bleeding, weight loss, chronicity, or persistent vomiting; those whose symptoms do not respond to medications; and those older than 55 years
	More than 90 percent sensitivity and specificity in diagnosing gastric and duodenal ulcers and cancers
Barium or diatrizoate meglumine and diatrizoate sodium (Gastrografin) contrast radiography (double-contrast hypotonic duodenography)	Indicated when endoscopy is unsuitable or not feasible, or if complications such as gastric outlet obstruction suspected
	Diagnostic accuracy increases with extent of disease; 80 to 90 percent sensitivity in detecting duodenal ulcers
Helicobacter pylori testing	
Serologic ELISA	Useful only for initial testing (sensitivity, 85 percent; specificity, 79 percent); cannot be used to confirm eradication
Urea breath test	More expensive
	Sensitivity, 95 to 100 percent; specificity, 91 to 98 percent; can be used to confirm eradication
	PPI therapy should be stopped for two weeks before test
Stool antigen test	Inconvenient but accurate (sensitivity, 91 to 98 percent; specificity, 94 to 99 percent)
	Can be used to confirm eradication
Urine-based ELISA and rapid urine test	Sensitivity, 70 to 96 percent; specificity, 77 to 85 percent
	Cannot be used to confirm eradication
Endoscopic biopsy	Culture (sensitivity, 70 to 80 percent; specificity, 100 percent), histology (sensitivity, > 95 percent; specificity, 100 percent), rapid urease (CLO) test (sensitivity, 93 to 97 percent; specificity, 100 percent)

EGD = esophagogastroduodenoscopy; ELISA= enzyme-linked immunosorbent assay; PPI= proton pump inhibitor; CLO= Campylobacter-*like organism.*

Information from references 1, 19, and 20.

being assessed.[21,22] Potential benefits of shorter regimens include better compliance, fewer adverse effects, and lower costs.

Administration of an H_2 blocker or proton pump inhibitor for four weeks *(Table 4[19,21-25])* induces healing in most duodenal ulcers. Proton pump inhibitors provide superior acid suppression, healing rates, and symptom relief and are recommended as initial therapy for most patients. One meta-analysis of randomized controlled trials comparing proton pump inhibitors with H_2 blockers showed earlier pain control and better healing rates at four weeks for proton pump inhibitors (85 versus 75 percent).[23] A recent systematic review of randomized controlled trials showed that proton pump inhibitors healed duodenal ulcers in more than 95 percent of patients at four weeks and gastric ulcers in 80 to 90 percent of patients at eight weeks.[24] Therefore, there is little reason to prescribe proton pump inhibitors for longer than four weeks for duodenal ulcers unless the ulcers are large, fibrosed, or unresponsive to initial treatment.

Eradicating *H. pylori* is often sufficient in patients with small duodenal ulcers. Repeated EGD with biopsy is recommended to confirm healing of gastric ulcers and to rule out malignancy. Maintenance therapy with H_2 blockers or proton pump inhibitors prevents recurrence in high-risk patients (e.g., those with a history of complications, frequent recurrences, ulcers testing negative for *H. pylori*, refractory giant ulcers, or severely fibrosed ulcers), but it is not generally recommended for patients in whom *H. pylori* has been eradicated and who are not taking NSAIDs long-term.

REFRACTORY ULCERS

Refractory peptic ulcer disease (i.e., disease that fails to heal after eight to 12 weeks of therapy) may be caused by persistent or resistant *H. pylori* infection, continued NSAID use, giant ulcers requiring longer healing time, cancer, tolerance of or resistance to medications, or hypersecretory states.[26] Therapy for refractory peptic ulcer disease involves treatment of the underlying cause and prolonged administration of standard doses of a proton pump inhibitor *(Figure 1)*. Up to 25 percent of patients with gastric ulcers who continue to take NSAIDs may require proton pump inhibitor therapy for longer than eight weeks.[27]

SURGERY

Surgery is indicated in patients who are intolerant of medications or do not comply with medication regimes, and those at high risk of complications (e.g., transplant recipients, patients dependent on steroids or NSAIDs, those with giant gastric or duodenal ulcer, those with ulcers that fail to heal with adequate treatment). Surgery should also be considered for patients who have a relapse during maintenance treatment or who have had multiple courses of medications.[28]

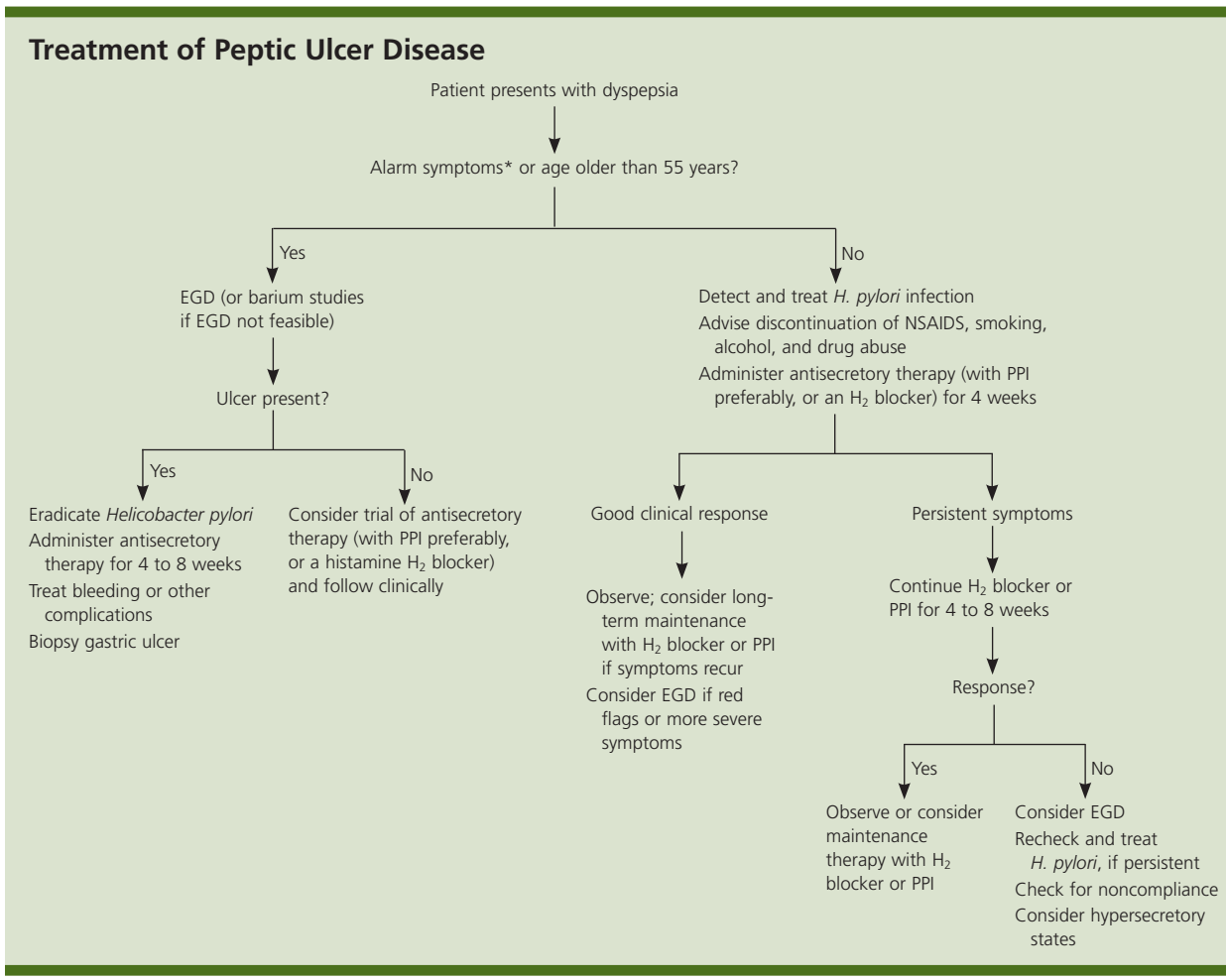

Figure 1. Algorithm for the treatment of peptic ulcer disease. (EGD = esophagogastroduodenoscopy; PPI = proton pump inhibitor; NSAID = nonsteroidal anti-inflammatory drug.)

**—Alarm symptoms include evidence of bleeding (e.g., anemia, heme-positive stool, melena), perforation (e.g., severe pain), obstruction (e.g., vomiting), and malignancy (e.g., weight loss, anorexia).*

Surgical options for duodenal ulcers include truncal vagotomy and drainage (pyloroplasty or gastrojejunostomy), selective vagotomy (preserving the hepatic and celiac branches of the vagus) and drainage, highly selective vagotomy (division of only the gastric branches of the vagus, preserving Latarjet's nerve to the pylorus), or partial gastrectomy. Surgery for gastric ulcers usually involves a partial gastrectomy. Procedures other than highly selective vagotomy may be complicated by postprocedure dumping and diarrhea.

Associated Complications

About 25 percent of patients with peptic ulcer disease have a serious complication such as hemorrhage, perforation, or gastric outlet obstruction. Silent ulcers and complications are more common in older patients and in patients taking NSAIDs.[16,17] The incidence of serious upper gastrointestinal complications among persons in the general population who do not take NSAIDs is extremely low (less than one per 1,000 person-years).[29]

BLEEDING

Upper gastrointestinal bleeding occurs in 15 to 20 percent of patients with peptic ulcer disease. It is the most common cause of death and the most common indication for surgery in the disease. In older persons, 20 percent of bleeding episodes result from asymptomatic ulcers.[17] Patients may present with hematemesis (bright red or "coffee ground"), melena, fatigue caused by anemia, orthostasis, or syncope.

There are several risk-stratification schemes that can help physicians determine the need for urgent intervention and predict continued or recurrent bleeding after endoscopic therapy. The Rockall risk scoring system is useful in stratifying patients at higher risk of rebleeding and death and has been prospectively validated in different populations *(Table 5*[30,31]*)*.[30]

In stable patients with gastrointestinal bleeding, potentially ulcerogenic medications should be discontinued. A proton pump inhibitor should be administered intravenously; this reduces transfusion requirements, need for surgery, and duration of hospitalization,

Table 4. Treatment of Peptic Ulcers

Treatment	*Comment*	*Options*
Eradication of *Helicobacter pylori*	Treatment duration is 10 to 14 days (although courses lasting one to seven days have been reported to have comparable effectiveness[21,22])	Omeprazole (Prilosec) 20 mg two times daily or lansoprazole (Prevacid) 30 mg two times daily
		plus amoxicillin 1 g two times daily or metronidazole (Flagyl) 500 mg two times daily (if allergic to penicillin)
		plus clarithromycin (Biaxin) 500 mg two times daily
	Eradication rates 80 to 90 percent or higher	Ranitidine bismuth citrate (Tritec)* 400 mg two times daily
		plus clarithromycin 500 mg two times daily or metronidazole 500 mg two times daily
		plus tetracycline 500 mg two times daily or amoxicillin 1 g two times daily
		Levofloxacin (Levaquin) 500 mg daily
		plus amoxicillin 1 g two times daily
		plus pantoprazole (Protonix) 40 mg two times daily
		Bismuth subsalicylate (Pepto-Bismol) 525 mg (two tablets) four times daily
		plus metronidazole 250 mg four times daily
		plus tetracycline 500 mg four times daily
		plus H_2 blocker for 28 days or proton pump inhibitor for 14 days
Histamine H_2 blockers	70 to 80 percent healing in duodenal ulcer after four weeks, 87 to 94 percent after eight weeks	Ranitidine (Zantac) 150 mg two times daily or 300 mg at night
		Famotidine (Pepcid) 20 mg two times daily or 40 mg at night
		Cimetidine (Tagamet) 400 mg two times daily or 800 mg at night
Proton pump inhibitors	Treatment duration is four weeks for duodenal ulcer and eight weeks for gastric ulcer	Omeprazole 20 mg daily
		Lansoprazole 15 mg daily
	80 to 100 percent healing	Rabeprazole (Aciphex) 20 mg daily
		Pantoprazole 40 mg daily
Sucralfate (Carafate)	Treatment duration is four weeks	1 g four times daily
	Effectiveness similar to H_2 blockers	
Surgery	Rarely needed	Duodenal ulcer: truncal vagotomy, selective vagotomy, highly selective vagotomy, partial gastrectomy
		Gastric ulcer: partial gastrectomy with gastroduodenal or gastrojejunal anastomosis

**—Not available in the United States.*

Information from references 19 and 21 through 25.

although it does not reduce mortality.[32] EGD should be performed to find characteristics that suggest a high rate of bleeding recurrence (e.g., ulcer larger than 1 cm, visible or actively bleeding vessel).[30]

Patients whose condition is unstable should undergo fluid or packed-cell resuscitation followed by emergent EGD and coagulation of bleeding sites through endoscopic ligation; placement of hemoclips; injection of epinephrine, alcohol, or a sclerosant; or a combination of methods.[33]

Oral antisecretory therapy should be initiated as soon as patients resume oral intake. Treatment of *H. pylori* infection is more effective than antisecretory therapy without eradication of *H. pylori* for preventing recurrent bleeding. Therefore, patients with bleeding peptic ulcers should be tested for *H. pylori* infection and should be prescribed eradication therapy if results are positive.[34] If continued administration of aspirin or NSAIDs is required, concurrent administration of misoprostol or a proton pump inhibitor should be considered.[35,36] Patients with nonhealing gastric ulcers should have biopsy to rule out cancer.

Angiographic embolization of bleeding vessels or surgery is indicated if a patient's vital signs or laboratory studies suggest continued or recurrent bleeding.[33] Surgical options include gastroduodenotomy and oversewing of the blood vessel with or without vagotomy and drainage in duodenal ulcer; and excision of the ulcer with vagotomy and drainage or partial gastrectomy in bleeding gastric ulcers.

PERFORATION

Perforation occurs in approximately 2 to 10 percent of peptic ulcers.[25] It usually involves the anterior wall of the duodenum (60 percent), although it may also occur in antral (20 percent) and lesser-curve (20 percent) gastric ulcers. Perforation of ulcers in children is rare.

Free peritoneal perforation and resulting chemical and bacterial peritonitis is a surgical emergency causing sudden, rapidly spreading, severe upper abdominal

Table 5. Rockall Risk Scoring System for Patients with Peptic Ulcer Disease

Feature	*Points*
Age (years)	
< 60	0
60 to 79	1
> 79	2
Shock	
No shock (SBP ≥ 100, pulse < 100 bpm)	0
Tachycardia (SBP ≥ 100, pulse ≥ 100 bpm)	1
Hypotension (SBP < 100)	2
Comorbid illness	
No major comorbid illness	0
CHF, ischemic heart disease, other major comorbidity	2
Liver or renal failure, disseminated cancer	3
Diagnosis	
Mallory-Weiss tear, no other lesion identified and no stigmata of recent hemorrhage	0
All other pathology causing bleeding (except cancer)	1
Upper gastrointestinal tract cancer	2
Major stigmata of recent hemorrhage	
None or dark spot only	0
Blood in upper gastrointestinal tract, adherent clot, visible or spurting vessel	2
Total:	____

	*Risk (%)**	
Score	*Rebleeding*	*Mortality*
< 3 points	6.2	0.2
3 or 4 points	13	6.8
> 4 points	25	20

SBP = systolic blood pressure (mm Hg); bpm = beats per minute; CHF = congestive heart failure.

**—Interpretation using data from two independent validation studies.[31]*

Adapted with permission from Rockall TA, Logan RF, Devlin HB, Northfield TC. Risk assessment after acute upper gastrointestinal haemorrhage. Gut 1996;38:318, with additional information from reference 31.

pain exacerbated by movement; the pain may radiate to the right lower abdomen or to both shoulders. Fever, hypotension, and oliguria suggest sepsis and circulatory compromise. Generalized abdominal tenderness, rebound tenderness, board-like abdominal wall rigidity, and hypoactive bowel sounds (clinical signs of peritonitis) may be masked in older patients and those taking steroids, immunosuppressants, or narcotic analgesics. Upright or lateral decubitus abdominal radiography or erect chest radiography may demonstrate pneumoperitoneum; however, the absence of this finding does not rule out perforation.[17] Sonography, computed tomography, and gastroduodenography are confirmatory.

Initial resuscitation with large-volume crystalloids; nasogastric suction; and administration of intravenous broad-spectrum antibiotics against gram-negative rods, anaerobes, and oral flora are usually followed by laparotomy and placement of an omental patch (Graham patch plication) in patients with perforated duodenal ulcers. In otherwise healthy patients with a history of chronic ulcer and minimal peritoneal contamination, a concurrent, definitive, anti-ulcer procedure (e.g., vagotomy and drainage, highly selective vagotomy) may also be considered. Perforated gastric ulcers are treated with an omental patch, wedge resection of the ulcer, or a partial gastrectomy and reanastomosis. Coexisting *H. pylori* infection should be eradicated to reduce recurrence and minimize the need for long-term antisecretory therapy and further surgical intervention.[25,37] In older patients, mortality rates from perforation and its management may be as high as 30 to 50 percent.[1]

GASTRIC OUTLET OBSTRUCTION

Peptic ulcer disease is the underlying cause in less than 5 to 8 percent of patients presenting with gastric outlet obstruction. Patients with recurrent duodenal or pyloric channel ulcers may develop pyloric stenosis as a result of acute inflammation, spasm, edema, or scarring and fibrosis.

Symptoms suggesting obstruction include recurrent episodes of emesis with large volumes of vomit containing undigested food; persistent bloating or fullness after eating; and early satiety. Weight loss, dehydration, and a hypochloremic, hypokalemic metabolic alkalosis may result; a tympanitic epigastric mass representing the dilated stomach with visible gastric peristalsis also may be observed.

EGD or gastroduodenography (using diatrizoate meglumine and diatrizoate sodium [Gastrografin] or barium) is recommended to determine the site, cause, and degree of obstruction. Malignancy, a more common cause of obstruction (responsible for more than 50 percent of cases), should be ruled out.[38] Obstruction resulting from acute inflammation or edema responds well to nasogastric decompression, administration of H_2 blockers or proton pump inhibitors, and eradication of *H. pylori*. Prokinetic agents should be avoided. Endoscopic pyloric balloon dilatation or surgery (vagotomy and pyloroplasty, antrectomy, or gastroenterostomy) are options to relieve chronic obstruction.[25]

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