Clinical evaluation of peptic ulcer disease

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Release Date: June 2013
Expiration Date: June 2014
Estimated time to complete the educational activity: 0.50 hours

Program Description: From the June 2013 issue of The Clinical Advisor: Clinical evaluation of peptic ulcer disease. Most often caused by use of nonsteroidal anti-inflammatory drugs or bacterial infection, peptic ulcer disease usually presents as epigastric pain.

Target Audience: This activity has been designed to meet the educational needs of nurses, nurse practitioners and physician assistants.

Activity Objectives: After completing the activity, the participant should be better able to:

- Explain the factors associated with the development of peptic ulcer disease.
- Describe in which patients a “test and treat” approach can be used.
- Name the medication that should not be given for up to two weeks prior to performing a rapid urease test.
- Identify the medication used in first-line triple therapy in non-penicillin-allergic patients with Helicobacter pylori-related ulcers.

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NPACE designates this educational activity for a maximum of 0.50 contact hours of credit. Participants should only claim credit commensurate with the extent of their participation in the activity.

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The authors have no relationships to disclose relating to the content of this article.

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**Method of Participation:** There are no fees for participating in and receiving CME credit for this activity. During the period June 2013 through June 2014, participants must: 1) read the learning objectives and faculty disclosures, 2) study the educational activity, 3) complete the posttest and submit it online.

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**HOW TO TAKE THE POST-TEST:** Click here after reading the article to take the post-test on myCME.com.

Peptic ulcer disease (PUD) is a common disorder in the United States, with approximately 500,000 new cases diagnosed each year and 4 million cases of ulcer recurrence.¹ Complications related to PUD cause nearly 15,000 deaths annually.²

Men are affected slightly more often than women, and although peptic ulcers can occur at any age, individuals aged 30 years...
to 55 years are more likely to have duodenal ulcers, whereas gastric ulcers occur most often between age 55 years and age 70 years.1

A peptic ulcer is defined as a disruption in the mucosa of the stomach or duodenum >5 mm in diameter and extending to the submucosa.1-3 Peptic ulcers occur when there is an imbalance between the protective factors of the mucosa and such aggressive factors as acid and pepsin.1-3 The majority of ulcers are located in the duodenum, and approximately 90% occur within 3 cm of the pylorus.1,2

Etiology

Approximately 90% of PUD cases are caused by Helicobacter pylori infection or nonsteroidal anti-inflammatory drug (NSAID) use.1,4,5 H. pylori, a gram-negative bacteria, is able to withstand the acidic conditions in the stomach by using urease to produce ammonia.2,3 This bacteria causes several changes in its host that lead to ulcer development, including activation of the inflammatory response, increase in gastric-acid secretion, and impairment in the mucosal defense system.5

H. pylori infection is relatively common, with more than 50% of people worldwide being infected; ulcer development occurs in 5% to 10% of these cases.3 H. pylori is present in 75% to 90% of individuals with duodenal ulcers.1 Although H. pylori is only able to colonize gastric epithelial cells, excess secretion of gastric acid causes gastric metaplasia in the duodenal bulb, which enables duodenal colonization.3

The prevalence of H. pylori tends to be higher in developing countries; the United States has a prevalence of approximately 30%.2 Predisposing factors for H. pylori infection include poor socioeconomic status, less education, crowded or unclean living conditions, unsanitary food or water, and exposure to gastric contents of an individual infected with H. pylori.2

NSAIDs, including aspirin, are associated with an increased risk of gastric and duodenal mucosal injury (e.g., erosions, ulcers, and ulcer complications). Studies have shown that 15% to 30% of patients using NSAIDs have ulcers, and clinically significant ulcers and ulcer complications are present in 3% to 4.5% of NSAID users.5

Furthermore, NSAID users have been found to have a fourfold increased risk of ulcer complications, and patients who take low-dose aspirin have a twofold to threefold increased risk of ulcer bleeding.3 It has been shown that NSAID users who are also infected with H. pylori are at increased risk for PUD.3,4

NSAIDs decrease inflammation through the inhibition of prostaglandins.4 Nonselective NSAIDs accomplish this by inhibiting COX-1 and COX-2 enzymes.1 Mucosal damage results mainly from COX-1 inhibition, which is involved in mucosal defense, and inhibition of thromboxane A2, which causes bleeding.5 This knowledge led to the development of COX-2-selective NSAIDs, which are associated with a decreased risk of ulcers and ulcer complications.3,5 However, COX-2-selective NSAIDs are associated with an increased risk of cardiovascular complications.1

Although H. pylori and NSAID use are the most common causes of PUD, clinicians must be aware of other potential causes, including acid hypersecretory states, cytomegalovirus, Crohn's disease, lymphoma, medications, and such chronic medical illnesses as cirrhosis and chronic kidney disease.1 The cause of some ulcers remains unknown. Additional contributory factors associated with PUD include stress, cigarette smoking, alcohol use, lower socioeconomic status, and genetics.2,5,6

Clinical presentation

Epigastric pain is the characteristic symptom associated with PUD.1,3 A review of 30 studies found that abdominal pain and epigastric pain were the most common symptoms associated with PUD, with each occurring in 81% of study participants.7 Patients may describe the pain as gnawing, dull, aching, or “hunger-like.”1
Pain relief following the intake of food or antacids and the return of pain during the fasting state and/or pain during the night that awakens the individual is reported in some cases, especially in those with duodenal ulcers.\textsuperscript{3,5} Such symptoms as fullness, bloating, early satiety, and nausea may also be seen in patients with PUD.\textsuperscript{3} Although less common, weight loss and vomiting may be associated with gastric ulcers as well.\textsuperscript{1,5}

The majority of individuals with PUD go through periods of waxing and waning pain, during which time they will be symptomatic for as long as several weeks followed by pain-free periods ranging from months to years.\textsuperscript{1} Chronic ulcers, particularly those caused by NSAID use, may be present without any symptoms.\textsuperscript{3} Such complications as upper GI bleeding or perforation may be the first indication of PUD in these individuals.\textsuperscript{3} Interestingly, age may impact clinical presentation. Several studies have found that younger patients more often reported abdominal pain, whereas bleeding was more common in older individuals.\textsuperscript{7}

In addition to the signs and symptoms noted above, several other components of the patient history may lead to the diagnosis of PUD. For example, the patient may report a history of cigarette smoking: Ulcers and ulcer complications have been found to occur more frequently in smokers, and smoking has been found to have a negative effect on ulcer healing rates and responsiveness to therapy.\textsuperscript{2}

A positive family history of PUD may be present. First-degree relatives of patients with duodenal ulcers have an increased likelihood of ulcer development exists in.\textsuperscript{5} Other contributory factors associated with PUD include alcohol use, psychological stress, decreased prostaglandin levels associated with aging, and use of such medications as bisphosphonates, potassium chloride, and immunosuppressants.\textsuperscript{5}

Such diseases as systemic mastocytosis, chronic pulmonary disease, chronic renal failure, cirrhosis, nephrolithiasis, and alpha–1 antitrypsin deficiency are strongly associated with PUD as well and may be part of the patient's medical history.\textsuperscript{2}

In the absence of ulcer-related complications, the physical exam of an individual with PUD may be completely normal.\textsuperscript{1} The most common exam finding in patients with PUD is epigastric tenderness to palpation.\textsuperscript{2} Some patients will have a positive fecal occult blood test or fecal immunochemical test.\textsuperscript{1}

Bleeding, perforation, penetration, and obstruction are the four major complications associated with PUD, and it is important to be familiar with the clinical presentation of each.\textsuperscript{1,8} The most common complication is GI hemorrhage.\textsuperscript{3,8} Potentially life-threatening bleeding occurs in up to 15% of PUD patients, and ulcer bleeding is associated with a 7% overall mortality rate.\textsuperscript{1,8}

Bleeding is more common in NSAID-related ulcers, and elderly patients and those with other comorbidities are most at risk.\textsuperscript{1,8} Patients may present with melena, hematemesis, or hematochezia, all of which should be treated as an emergency situation.\textsuperscript{1,8} A complete blood count (CBC) may reveal anemia due to blood loss.\textsuperscript{5}

Perforation of the GI wall can cause contents to spill into the abdominal cavity, possibly leading to acute peritonitis.\textsuperscript{8} Patients may present with severe abdominal pain of sudden onset. Physical exam may reveal a rigid abdomen and rebound tenderness, and lab results are typically positive for leukocytosis.\textsuperscript{1}

Another complication of PUD is ulcer penetration into such nearby structures as the pancreas, liver, and biliary tree. The patient history may include a change in the typical pattern and intensity of symptoms. The clinician should consider ulcer penetration if severe, constant pain is reported in conjunction with radiating pain to the back.\textsuperscript{1}

Gastric-outlet obstruction can occur when swelling and scarring from peptic ulcers causes narrowing of the duodenum.\textsuperscript{1,8} The patient may report early satiety, vomiting, and weight loss, and a succussion splash may be audible in the epigastrium on
physical examination.¹

**Diagnostic testing**

Alarm features of dyspepsia include age greater than 55 years with new-onset disease, family history of upper-GI cancer, weight loss, GI bleeding, dysphagia, odynophagia, iron-deficiency anemia, persistent vomiting, palpable mass or lymphadenopathy, and jaundice (Table 1).⁹

**Table 1. Alarm features requiring endoscopy**

<table>
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<td>Age &gt;55 years with new-onset dyspepsia</td>
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<td>Family history of upper-GI cancer</td>
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<td>Weight loss</td>
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<td>GI bleeding</td>
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<td>Dysphagia</td>
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<td>Odynophagia</td>
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<td>Iron-deficiency anemia</td>
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<td>Persistent vomiting</td>
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<tr>
<td>Palpable mass or lymphadenopathy</td>
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<td>Jaundice</td>
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If the patient is aged 55 years or younger and does not exhibit any alarm features, the American Gastroenterological Association (AGA) recommends a “test and treat” approach (download Figure 1),¹⁰ which involves using such noninvasive tests as the urea breath test, stool antigen test, and serologic testing to detect possible *H. pylori* infection.⁵ If one of these tests is positive, the patient is treated for *H. pylori* infection without having to undergo endoscopy, which proves to be both cost-effective and less invasive for the patient.⁵

A study found the AGA’s recommended approach to be just as effective and safe as prompt endoscopy in the management of dyspeptic patients in the primary-care setting, with only 33% of patients requiring endoscopy following the test-and-treat method.¹¹

The decision as to which noninvasive test is most appropriate depends on the prevalence of *H. pylori* in the area, the clinical setting, and the individual patient.⁵ Since serology testing for anti-*H. pylori* antibody cannot make the distinction between an active and past infection, the urea breath test or stool antigen test are recommended as initial diagnostic studies.⁵,¹⁰
While commonly used, the test-and-treat method is not always necessary. If the prevalence of *H. pylori* infection in a particular area is <5% and the patient is aged 55 years or younger with no alarm features, it is recommended that empiric proton pump inhibitor (PPI) therapy be started first, as testing for *H. pylori* is unlikely to be beneficial.\(^{10}\) However, the decision as to whether to test for *H. pylori* first or treat empirically with PPI therapy depends on the prevalence of *H. pylori* in the area as well as patient and provider preferences.\(^{12}\)

Patients older than age 55 years or those of any age with alarm features should undergo upper GI endoscopy first.\(^9\) Endoscopy enables direct visualization of the mucosa and is the most sensitive and specific test.\(^2\) Endoscopy also allows a tissue sample to be obtained for biopsy to rule out malignancy in cases of gastric ulcers or to detect *H. pylori* infection using the rapid urease test.\(^1,2,5\) In addition, endoscopy can determine whether blood loss is attributable to a bleeding ulcer.\(^2\)

A benign-appearing ulcer on endoscopy requires no further endoscopic testing as long as the biopsy results are negative for malignancy, dysplasia, and atypical cells. If biopsy results are positive, however, a second endoscopy should be performed 12 weeks after initiation of treatment to ensure proper healing. If the ulcer is not healing as it should, malignancy should be considered.\(^1\)

Patients who are suspected of having such ulcer-related complications as perforation, penetration, or obstruction should undergo an abdominal CT scan. Although laboratory testing is typically normal in uncomplicated PUD, a CBC is useful in detecting anemia or leukocytosis, which may indicate the presence of ulcer complications.\(^1\)

When testing for *H. pylori*, it is important to keep in mind that PPIs impede urease activity and can affect the results of the rapid urease test, the stool antigen test, and the urea breath test.\(^1,5\) PPIs should be withheld at least two weeks prior to performing these tests.\(^12\) Because bleeding reduces the sensitivity of invasive tests, an endoscopic rapid urease test and histologic testing should be performed in conjunction with the urea breath test in patients with actively bleeding ulcers.\(^5\)

**Differential diagnosis**

Dyspepsia is the most common symptom in PUD, occurring in 80% to 90% of patients.\(^1,3,12\) However, dyspepsia can occur in other diseases as well, causing these conditions to present similarly to PUD.

**Gastroesophageal reflux disease (GERD).** Epigastric pain or discomfort may occur in individuals with GERD.\(^12\) Although 20% of dyspeptic patients have GERD, other symptoms are much more common in the presentation of this disorder, including heartburn and regurgitation.\(^1,12\)

GERD is associated with two patterns of reflux: upright and supine. Upright reflux occurs during the daytime, is commonly characterized by postprandial heartburn, and may be accompanied by regurgitation. Supine reflux typically occurs at night when the individual is lying down.\(^12\)

In most cases, GERD is clinically diagnosed using the patient history. Although it can present similarly to PUD with such symptoms as dyspepsia, GERD is the most likely diagnosis if the predominant symptom is heartburn.\(^1,12\)

**Functional dyspepsia.** Functional (or nonulcer) dyspepsia is defined as dyspepsia lasting for at least three months without any organic, systemic, or metabolic cause.\(^10,12,13\) Up to 60% of patients with dyspepsia have functional dyspepsia, making this condition more common than dyspepsia attributable to organic causes.\(^10,13\)

Since functional dyspepsia is a diagnosis of exclusion, a definitive diagnosis should not be made until an endoscopy is performed and at least six months have passed since the initial onset of symptoms.\(^13\) Although the exact pathophysiologic mechanisms involved in functional dyspepsia remain unclear, possible contributory factors include genetics, psychosocial
distress, and alterations in GI motor and sensory function.\textsuperscript{10,13}

Some patients with dyspepsia may be infected with \textit{H. pylori} in the absence of peptic ulcers or other endoscopic findings and are therefore considered to have functional dyspepsia.\textsuperscript{10} It is recommended that these patients undergo anti-\textit{H. pylori} therapy.\textsuperscript{10,13}

\textbf{Gastric cancer.} Gastric cancer is found in only 1\% of individuals with dyspepsia and is uncommon in patients with uncomplicated dyspepsia who are younger than age 55 years.\textsuperscript{1,12} Most patients with gastric cancer will present with such symptoms as anorexia, early satiety, and weight loss.\textsuperscript{14}

Anorexia may be present in patients with gastric ulcers, but individuals with uncomplicated PUD typically do not experience significant weight loss.\textsuperscript{1} An upper GI endoscopy can be performed to rule out malignancy and should be ordered if any alarm features are present.\textsuperscript{1,2,12}

\textbf{Food and medications.} Eating too fast or overeating can cause indigestion that can manifest as dyspepsia.\textsuperscript{1} High-fat foods, alcohol, and coffee also can cause indigestion.\textsuperscript{2} A number of medications may also lead to dyspepsia, including NSAIDs, calcium antagonists, bisphosphonates, steroids, theophyllines, and nitrates.\textsuperscript{1,12,13} Food or medication intolerances can usually be diagnosed with a thorough patient history.

\section*{Treatment}

The goals of PUD treatment and the treatment regimen depend on the etiologic agent involved in the disease process. Ulcers that are not caused by \textit{H. pylori} infection can be treated with four to eight weeks of PPI therapy (\textbf{Table 2}).\textsuperscript{5,9} Uncomplicated duodenal ulcers can be treated with a PPI for four weeks, while uncomplicated gastric ulcers require eight weeks of therapy.\textsuperscript{1,5}

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\textbf{Uncomplicated ulcers:} & \textbf{Complicated ulcers} \\
\hline
- PPI therapy & - PPI therapy \\
- Duodenal ulcers: four weeks & \\
- Gastric ulcers: eight weeks & \\
OR & \\
- \text{H}\textsubscript{2}-receptor antagonist therapy & \\
- Duodenal ulcers: six weeks & \\
- Gastric ulcers: eight weeks & \\
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\textbf{Discontinue NSAID therapy, if possible}

- If patient must continue NSAID therapy:
  - PPI once daily with NSAID therapy
  OR
  - Substitute COX-2 inhibitor for nonselective NSAID
  OR
  - Misoprostol (Cytotec) with NSAID therapy
Refractory ulcers confirmed on endoscopy:

- If negative for *H. pylori*, NSAID use, or other conditions, consider an additional six to eight weeks b.i.d. PPI therapy.


The six PPIs currently on the market are omeprazole (Prilosec), rabeprazole (AcipHex), esomeprazole (Nexium), lansoprazole (Prevacid), dexlansoprazole (Dexilant), and pantoprazole (Protonix), and each is equally effective in treating PUD. As stated previously, individuals in areas where *H. pylori* prevalence is <5% may be started on empiric PPI therapy without undergoing *H. pylori* testing. If the patient remains symptomatic after four to eight weeks of PPI therapy, an endoscopy and biopsy should be ordered. The patient should be treated based on the endoscopic findings, including *H. pylori* eradication therapy if positive for infection. If the patient is *H. pylori*-negative, has a negative endoscopy, and continues to be symptomatic despite PPI therapy, consider other disorders that may present similarly to PUD.

H₂-receptor antagonists inhibit nocturnal acid secretion and can also be used in the treatment of PUD but require more time to provide pain relief and ulcer healing than do PPIs. Four H₂-receptor antagonists are currently on the market: cimetidine (Tagamet), ranitidine (Tritec, Zantac), famotidine (Fluxid, Pepcid), and nizatidine (Axid). Uncomplicated duodenal ulcers can be treated with an H₂-receptor antagonist for six weeks, and uncomplicated gastric ulcers require eight weeks of treatment. In cases of complicated ulcers, PPIs are preferred over H₂-receptor antagonists.

NSAID use should be discontinued in patients with NSAID-related ulcers whenever possible. If NSAID therapy cannot be discontinued, the addition of a once-daily PPI is recommended. This concomitant treatment results in healing of duodenal ulcers after four weeks and of gastric ulcers after six to eight weeks.

Another option is to replace a nonselective NSAID with a COX-2 inhibitor, a selective NSAID that is associated with a decreased incidence of ulcers. The mucosal protective agent misoprostol (Cytotec) can also be administered simultaneously with NSAID treatment to reduce the risk of ulcer complications.

In the case of a refractory ulcer, in which the patient has a confirmed ulcer on endoscopy and remains symptomatic after treatment with a PPI or H₂-receptor antagonist, *H. pylori* infection or surreptitious NSAID or aspirin use must be ruled out. Since the sensitivity of *H. pylori* tests are less than 100%, it is possible that the infection was originally missed due to a false-negative result.

Such other ulcer-causing conditions as Zollinger-Ellison syndrome should be ruled out as well. If the patient is confirmed to be negative for *H. pylori* infection and other conditions, an additional six to eight weeks of b.i.d. PPI therapy may be needed.

In cases of *H. pylori*-related PUD, the goal of treatment is to eradicate the bacteria. The standard first-line treatment is triple therapy consisting of a PPI b.i.d., clarithromycin (Biaxin) 500 mg b.i.d., and amoxicillin 1 g b.i.d. for seven to 14 days (Table 3).

**Table 3. Treatment of *H. pylori* related ulcers**
**First-line triple therapy:**
- PPI b.i.d., clarithromycin (Biaxin) 500 mg b.i.d., and amoxicillin 1 g b.i.d. for seven to 14 days
- Penicillin allergy: substitute metronidazole (Flagyl) 500 mg b.i.d.

**Second-line quadruple therapy:**
- PPI b.i.d.; bismuth subsalicylate 120 mg four times daily; tetracycline 500 mg four times daily; and metronidazole 250 mg four times daily or 500 mg three times daily for at least seven days

Confirm successful eradication with H. pylori testing four to eight weeks after therapy and at least two weeks after PPI therapy.

**After H. pylori is eradicated, additional PPI therapy may be needed:**
- Ulcers >1 cm or ulcer complications: PPI once daily for two to four weeks (duodenal ulcers) or four to six weeks (gastric ulcers)
- If patient continues to be symptomatic: four-week course of PPI therapy

Amoxicillin is preferred over metronidazole (Flagyl) because more bacterial strains are resistant to metronidazole.1,5 In cases of penicillin allergy, however, metronidazole 500 mg b.i.d. can be substituted.1,5 H. pylori infection is successfully eliminated in 70% to 95% of patients using the triple therapy regimen.5 Although seven, 10-, and 14-day regimens are all effective, 10-day and 14-day regimens have been found to be 7% to 9% more effective in the eradication of H. pylori and are therefore preferred.5,10

In cases of treatment failure, commonly due to poor patient compliance or bacterial resistance, second-line quadruple therapy consisting of a PPI b.i.d., bismuth subsalicylate 120 mg four times daily, tetracycline (Sumycin) 500 mg four times daily, and metronidazole 250 mg four times daily or 500 mg three times daily for at least seven days can be used.5 Following successful treatment and eradication of H. pylori, ulcers are usually adequately healed and recurrence rates decreased, particularly in the absence of NSAID or aspirin use.5

All patients should undergo H. pylori testing four to eight weeks after therapy to confirm successful eradication.1,5 While confirmation can be done using the urea breath test, stool antigen test, or endoscopy with biopsy, the urea breath test is the preferred method.1,5 Make sure the patient has not taken PPIs within two weeks of H. pylori testing.12

Some cases may require additional PPI therapy after completion of eradication therapy. Patients with ulcers >1 cm or those with ulcer complications should remain on a once-daily PPI for two to four weeks for duodenal ulcers or four to six weeks for gastric ulcers.1,5

If symptoms continue after the infection is eradicated, a four-week course of PPI therapy should be prescribed.9,10 An endoscopy should be ordered if symptoms persist after H. pylori eradication therapy and PPI therapy, and endoscopic findings should determine subsequent treatment.12

**Summary**

PUD is a common disorder most often caused by H. pylori infection and NSAID use. Contributory factors associated with PUD include stress, cigarette smoking, alcohol use, lower socioeconomic status, and genetics.
The clinical presentation of PUD most commonly includes epigastric pain with such symptoms as fullness, bloating, early satiety, and nausea also occurring in some patients. Other patients may be asymptomatic, especially those with chronic ulcers. Although the physical exam is usually normal in an individual with PUD, epigastric tenderness may be present.

Other diseases can present similarly to PUD, and a thorough history, a physical exam, and appropriate diagnostic testing can aid in ruling these out. Diagnostic testing is determined by the patient's age and the presence or absence of alarm features.

Treatment is dependent on the cause of PUD. Non-\textit{H. pylori}-related cases should be treated with either PPI or H$_2$–receptor antagonist therapy. \textit{H. pylori}–related PUD requires triple therapy that includes antibiotics and a PPI.

Additional PPI therapy may be needed in some cases. Such complications as GI bleeding, perforation of the GI wall, organ penetration, and gastric outlet obstruction have been found to occur in some cases of PUD.

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References


\textit{All electronic documents accessed June 10, 2013.}

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