

## ULCER DISEASE

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**Abstract:** Peptic ulcer disease (PU) develops with continued activity of the internal gastrointestinal tract (GIT) due to the secretion of gastric acid or pepsin. Epigastric pain usually occurs 15–30 minutes after eating in patients with gastric ulcers; on the other hand, pain from a duodenal ulcer usually occurs 2-3 hours after eating.

PU is a global problem, the risk of development of which ranges from 5% to 10%. Overall, there has been a decline in the incidence of ulcers worldwide due to improved hygiene and sanitation in the UK with effective treatment and judicious use of NSAIDs. Duodenal ulcers are four times more common than stomach ulcers. Also, duodenal ulcers are more common in men than in women.

**Key words:** H infection . Pylori , NSAIDs, antisecretory drugs, stress, hospital, perforation.

### Introduction

Peptic ulcer disease (PUD) results in continued activity of the internal gastrointestinal (GI) tract due to the secretion of gastric acid or pepsin. It spreads into the muscular layer of the gastric epithelium. Typically occur in the stomach and proximal duodenum. This may involve the lower esophagus, distal duodenum, or jejunum. Epigastric pain usually occurs 15–30 minutes after eating in patients with gastric ulcers; on the other hand, pain from a duodenal ulcer usually occurs 2-3 hours after eating. Today's Helicobacter pylori recommended for all patients with peptic ulcer disease. Endoscopy may be performed in some patients to confirm the diagnosis, especially in those with ominous symptoms.

### Etiology

Peptic ulcer disease (PUD) occurs for various reasons; However, ulcerative gastrointestinal tract associated with Helicobacter pylori and NSAID-associated ulcers account for most of the etiology of the disease.

Causes of peptic ulcer disease.

Are common:

- infection .
- NSAIDs.
- Medicines.

Rarely:

- Malignant neoplasms (stomach/lung cancer, lymphoma ).
- Stress (acute illness, burns, head injuries).
- Viral infection.
- Vascular insufficiency.
- Radiation therapy.
- Crohn's disease.
- Chemotherapy.

#### Helicobacter Pylori- associated gastric ulcer

X.pylorus is a competent rod that is found in the epithelial cells of the stomach. This bacterium is responsible for 90% of duodenal ulcers and 70% to 90% of gastric ulcers. H. pylori infection It is more common among people of lower socioeconomic status and usually occurs during childhood. The organism has a wide range of virulence factors and is allowed to adhere to create the stomach lining and resistance. This results in hypochlorhydria or achlorhydria , which leads to stomach ulcers.

Helicobacter pylori virulence factors :

- 1 Urease : The secretion of urease promotes the release of ammonia into urea and neutralizes the acidic environment of the stomach.
- 2 Toxins: CagA / WakA taking into account the measures taken to restore the stomach and damage the host tissues.
- 3 Flagella: reduce mobility and allow movement to the gastric epithelium.

#### **PUD associated with NSAIDs**

The use of non-steroidal anti-inflammatory drugs is the second most common cause of ulcer after H. pylori infection . The secretion of prostaglandin usually protects the gastric mucosa. NSAIDs block prostaglandin synthesis by inhibiting the COX-1 enzyme, which leads to a decrease in the production of gastric mucus and bicarbonate, as well as a decrease in contraction through mucosal tissues.

#### **Medicines**

In addition to NSAIDs, corticosteroids, bisphosphonates , potassium chloride, steroids and fluorouracil have been implicated in the etiology of PU .

Smoking also plays a role in duodenal ulcers, but the relationship is not linear. Alcohol can irritate the stomach lining and increase acidity.

Hypersecretory environment

- Systemic mastocytosis .
- Cystic fibrosis .
- Hyperparathyroidism .
- G hyperplasia antral G cells .

#### **History and physics**

Signs and symptoms of peptic ulcer disease may vary depending on the location of the disease and age. Ulcers of the stomach and duodenum can be differentiated by

the time of onset of symptoms in connection with food intake. Night pain is common with duodenal ulcers. Patients with gastric outlet obstruction usually report bloating or fullness.

Common signs and symptoms include:

- Pain in the epigastric region of the abdomen.
- Bloat of life.
- Fullness of life.
- Nausea and vomiting.
- Weight loss/weight gain
- Hematemesis .
- Melena.

Warning or warning symptoms that require urgent attention include:

- Unintentional weight loss.
- Progressive dysphagia.
- Obvious gastrointestinal compartment.
- Iron-deficiency anemia.
- Recurrent vomiting.
- Family history of malignant neoplasms of the upper gastrointestinal tract .

Grade

Diagnosis of PU requires a history, visual examination, and invasive/ non-invasive medical tests.

Story

It is necessary to collect the available medical history and note any work. Patient reports of pain in the epigastric region of the abdomen, early satiety and fullness after eating raise suspicion of ulcer. Gastric ulcer pain develops 2-3 hours after eating and can lead to weight loss, while duodenal ulcer pain after eating can lead to weight gain. Any patient with anemia, melena, hematemesis , or weight loss should be further investigated for continued DU, regional hospital, perforation, or cancer.

Physical examination

**Physical examination may reveal epigastric abdominal tenderness and anemia. investigations**

- 1 Esophagogastroduodenoscopy (EGD): the gold standard and most diagnostic test with sensitivity and specificity of up to 90% in the diagnosis of gastric and duodenal ulcers. The American Society of Gastrointestinal Endoscopy has published recommendations for endoscopy in patients with upper abdominal pain or diarrheal symptoms suggestive of DU. Patients over 50 years of age and with new-onset dyspeptic symptoms should undergo the procedure using EGD. Anyone with worrying symptoms should undergo an EGD, regardless of age.
- 2 Barium swallow: indicated when opposite to EGD.

- 3 Complete blood count, liver function and amylase and lipase levels.
4. Test for *Helicobacter pylori*:
  - Serological study.
  - Urea breath test: High sensitivity and specificity. It can be used to confirm eradication 4-6 weeks after menstruation. Based on urease, an enzyme produced by *H. pylori*, radioactively labeled air dioxide produced by the stomach is exhaled easily.
    - Antibodies to *H. pylori* can also be measured.
    - Stool antigen test.
    - ELISA based on urine and rapid urine analysis.
    - Endoscopic biopsy: Culture is not usually recommended as it is expensive, time consuming and invasive. It shows if eradication treatment is unsuccessful or if antibiotic resistance is suspected. To increase sensitivity, a biopsy of at least 4-6 sites is required. Gastric ulcers are usually located on the slight curvature between the antrum and the fundus. Most duodenal ulcers are located in the first part of the duodenum.
6. Computed tomography of the abdomen with contrast is of limited value in the diagnosis of peptic ulcer itself, but is useful in diagnosing its operations, such as perforation and outlet obstruction.

### *Treatment*

#### **Drug therapy**

Antisecretory drugs used for peptic ulcer disease include H<sub>2</sub> receptor antagonists and proton pump inhibitor (PPI). PPI equivalents have replaced H<sub>2</sub> blockers due to their superior healing and effectiveness. PPIs block acid production in the stomach, thereby relieving symptoms and promoting healing. Treatment may be included during development, as PPI use may increase the risk of bone fractures. Ulcers caused by NSAIDs can be treated by taking the NSAID for a short period of time or by switching to a lower dose. Corticosteroids and anticoagulants should also be discontinued if possible. Prostaglandin analogs ( misoprostol ) sometimes used as a prophylaxis for peptic ulcers caused by NSAIDs. First-line treatment for caused by *H. pylori* PUD It is a triple regimen, including two antibiotics and a proton pump inhibitor. Pantoprazole, clarithromycin, antibiotics, and PPIs work synergistically to kill *H. pylori*. The antibiotic chosen must take into account the presence of antibiotic resistance in the environment. If first-line therapy does not produce results, quadruple therapy with bismuth and chemical antibiotics is used.

#### **Refractory disease and surgical treatment**

Surgical treatment has shown that if the patient does not respond to treatment, is non-compliant or at high risk for treatment. A refractory peptic ulcer is an ulcer larger than 5 mm in diameter that does not heal despite 8-12 weeks of PPI therapy. Common causes are persistent infections with *H. / pylori*, ongoing use of NSAIDs or

concomitant diseases, severe healing of ulcers or other conditions such as gastrinoma or stomach cancer. If the ulcer persists despite addressing risk factors, patients may be candidates for surgical treatment. Surgical options include vagotomy or partial gastrectomy .

### **Differential diagnosis**

The following conditions may be symptoms of a severe peptic ulcer and it is important to know their condition in order to make a correct diagnosis.

- On the street - the inflammatory process leads to the development of gastrointestinal immune-mediated or infectious etiology, resulting in pain in the upper abdomen and nausea. The clinical picture is very similar to peptic ulcer disease.
- Gastroesophageal reflux disease (GERD). Patients typically experience a burning sensation in the epigastrium and lower sternum, increased salivation, or periodic regurgitation of food material.
- Gastric cancer - in addition to abdominal pain, patients usually experience worrying symptoms such as weight loss, melena, repeated vomiting, or signs of malignancy elsewhere in the case of metastases.
- Pancreatitis is pain in the epigastrium or right hypochondrium, more persistent and severe, increasing in the supine position, and patients usually have a history of alcoholism or gallstones. Elevated serum amylase and lipase levels are helpful in diagnosis.
- Biliary colic is periodic severe deep pain in the right hypochondrium or epigastrium caused by fatty foods.
- Cholecystitis is pain in the right hypochondrium or epigastrium , which usually lasts several hours, develops from fatty foods and is accompanied by nausea and vomiting. Fever, tachycardia, positive Murphy's sign , leukocytosis and liver dysfunction .

This guards against some life-threatening conditions that may also have similar behavior.

- Myocardial infarction - especially when the inferior wall and right ventricle appear, sometimes patients may include epigastric pain with nausea and vomiting. If there are other symptoms such as dizziness, shortness of breath, and abnormal functioning, important functions in a high-risk patient should alert the physician to their attention.
- Mesenteric ischemia - acute mesenteric ischemia indicates the independence of acute pain in life; the chronic variant usually reveals persistent postprandial epigastric pain and may be mistaken for a peptic ulcer. Old age, the presence of risk factors for atherosclerosis and weight loss should be taken into account during the examination.
- Mesenteric vasculitis - unexplained abdominal symptoms with or without bleeding from the severe gastrointestinal tract in a patient with other forms of

underlying systemic vasculitis , suspicion of mesenteric vasculitis is necessary vasculitis .

#### Complications

- Bleeding from the upper gastrointestinal tract
- Clogged orifice outlet
- Perforation
- Stomach cancer

#### PERFORATIVE ULCER

The therapeutic goal for perforated peptic ulcer disease is to repair the opening in the gastrointestinal tract and remove peritoneal contamination. Unlike bleeding duodenal ulcers, surgery is to treat perforated duodenal ulcers. Most perforated ulcers occur in the duodenum and pyloric canal; In an analysis of studies, perforated peptic ulcer disease was most common in the duodenal bulb (62%), followed by the pyloric region (20%) and the body of the stomach (18%). Although most patients with perforated ulcers do not have a previous history of peptic ulcer disease, risk factors for perforation include a previous history of peptic ulcer disease or use of NSAIDs. Patients receiving NSAID therapy have a greater risk of ulcer perforation with a history of previous ulceration, age over 60 years, selective serotonin reuptake inhibitors, steroids or anticoagulants 34-37.

#### Bleeding

Bleeding stomach ulcers are usually best treated by excising the ulcer and repairing the resulting stomach defect. Excision or biopsy of the ulcer is important since 4-5% of benign ulcers that occur are actually malignant ulcers. For ulcers on the greater curvature of the stomach, antrum or body of the stomach, wedge-shaped excision of the ulcer and closure of the resulting defect can be easily achieved in most cases without causing a change in the deformation of the stomach. Incision ulcers along the subtle curvature are more problematic. Because of the rich arcade from the left gastric artery, wedge excision of the ulcer is more difficult than in other sections, and subsequent closure of the subsequent defect much more often leads to gastric deformation and ether luminal obstruction or to leading volvulus of the resulting J - shaped stomach. For distal gastric ulcers using lesser curvature distal gastrectomy with Billroth reconstruction I or Billroth II is often the simplest method of excision of the ulcer and ensuring continuity of the gastrointestinal tract. A special case is a proximal gastric ulcer near the gastroesophageal (GE) junction. In most patients, the simplest approach is an anterior gastrotomy with biopsy and reculture of the ulcer from the gastric lumen. With this approach, it is relatively easy to avoid compromising the GE connection. In the event that excision of the ulcer is necessary, a gastrectomy is performed with a tongue extension of the edge of the resection of the minor curve to include the ulcer and subsequent esophagostomy. Ru - Y is an alternative option.



The standard approach to a bleeding duodenal ulcer is to perform an anterior long duodenotomy, passing through the pylorus to the distal stomach. The bleeding vessel, often the gastroduodenal arteries, is ligated to the verge of crater ulcers, placing a figure-of-eight suture on top and gradually crater of the ulcer to control the artery proximally and distally. The third suture is placed as a U-shaped suture under the ulcer to control the transverse branches of the pancreas that enter the GDA. behind. The transverse incision of the duodenum is then closed vertically to construct a Gemneke - Mikulicz pyloroplasty.

Classically, for the second risk of ulcer recurrence, a truncated vagotomy is observed. The role of vagotomy in 2011 is unclear. Our basic understanding of the pathogenesis of peptic ulcer disease involves treatment of *H. pylori* and cessation of NSAID use should result in treatment of the underlying peptic ulcer risk. In addition, with the advent of PPIs, it is now possible to eliminate gastric acid production with medication without exposure to vagotomy factors. Although first-level data exist for perforated duodenal ulcers demonstrating that treatment of *H. pylori* The need for definitive ulcer surgery is exploited; to date, there is no study that can draw a conclusion in the case of duodenal ulcers.

Although duodenotomy with direct guidance at the bleeding site, with or without vagotomy, is the most commonly used approach for treating bleeding duodenal ulcers, there is some evidence, the government suggests, that larger surgery may be associated with lower re-separation times. In 1993, Lat and colleagues published a randomized controlled trial comparing mylostomy and pyloroplasty with gastrectomy plus ulcer excision. The rate of rebleeding was found to be higher (17% vs. 3%) with vagotomy and pyloroplasty, but overall mortality was not distinguished. Often the main interventions, mainly duodenal bones, were significantly higher after gastrectomy. An important caveat to these data is that this study was conducted before the widespread use of PPI and *H. pylori* treatment, and there is clearly still a place for aggressive surgical treatment of underlying ulcer disease now that medical therapy has replaced surgical therapy as the mainstay of ulcer treatment. . . In patients without significant associated complications who are not in shock during surgery, a more aggressive surgical approach may be warranted in patients with posterior duodenal ulcers. Given the difficulty of treating a difficult duodenal stump with an extensive posterior duodenal ulcer, this approach should only be used by surgeons with Western experience in peptic ulcer surgery.

Despite the best surgical efforts, reoperation after vagotomy and pyloroplasty occurs in 6-17% of cases, endoscopic therapy is usually not a continuation after a recent duodenotomy, about two adjuvant interventions: either reoperation or transcatheter arterial embolization (TAE). Classic reoperation was the procedure of choice for reinsertion after duodenotomy. In the case of re-opening in a recurrent department,

most surgeons perform a more extensive operation, usually a distal gastrectomy with or without vagotomy, and excision or lengthening of the ulcer. This approach is unfortunately fraught with measurement and is associated with high mortality. Recently, several authors have been performing transcatheter arterial embolization as an independent alternative to surgical individual peptic ulcer disease refractory endoscopies. Without a head-to-head study, it is unclear whether TAE should replace surgery as the primary office for ulcer control, but data from two large series suggest that TAE can achieve long-term hemostasis in approximately 75% of patients with recurrent pathologies after duodenotomy and ligation. Given the curative or mortality risk of surgery for a hospital relapse, TAE, when available, should be the first adjunctive therapy for a hospital relapse after duodenotomy and ulcer ligation.

The following dietary recommendations may help you get rid of ulcers:

- Use other foods from all 4 food groups. Good nutrition is very important for ulcer healing. Talk to a dietitian to learn how to support your diet if you are avoiding foods because they cause you pain or discomfort.

- Try to include sources of soluble fiber in every good meal. Vegetables, fruits, oatmeal and oat bran, barley, peanut butter, nuts, nut butters and legumes such as lentils, dried beans and peas are good sources. A diet high in soluble fiber may help prevent ulcer recurrence.

- Drink caffeinated drinks in moderation. The recommendation is no more than 400 mg of caffeine per day, which is equivalent to about three 250 ml (3 cups) of coffee.

Caffeine increases stomach acid but does not seem to cause ulcers or overcome their symptoms. Consult external resources for more information on coffee in food and beverages.

- If you drink alcohol, do so in moderation. nice pleasure

Alcohol increases stomach acidity, so it can aggravate ulcers. If you have questions about drinking alcohol, talk to your doctor or health care provider.

- Spicy foods do not cause ulcers, but they may reduce symptoms in some people. If spicy foods bother you, avoid them or eat less of them. You can continue to eat spicy foods as long as they don't cause you discomfort.

- Listen to your body. If there are other foods or drinks that bother you, limit or avoid them. Discuss this with your doctor or healthcare provider.

- Check with your doctor or healthcare professional about the use of medications containing caffeine, acetylsalicylic acid, and other nonsteroidal anti-inflammatory drugs (NSAIDs).

Forecast

The prognosis of DU is excellent after the benefit of treatment of the underlying cause. Recurrence of ulcers can be prevented by following the rules and avoiding



alcohol, smoking and NSAIDs. Unfortunately, the relapse rate in most cases is 60%. Gastric perforation caused by NSAIDs occurs in 0.3% of patients per year.

***Bibliography***

1. *Narayanan M., Reddy K.M., Marsicano E.* Peptic ulcer disease and *Helicobacter pylori* infection . Missouri State Medicine, 2018. May-June.
2. Buksoro JUD Changes in micronutrient levels in hypertension // American Journal of Pediatric Medicine and Health Sciences (2993-2149). – 2023. – T. 1. – No. 10. – S. 338-340.
3. Banerjee S, Cash BD, Dominic JA, Baron TH, Anderson MA, Ben-Menahem T, Fisher L, Fukami N, Harrison ME, Ikenberry S .O., Khan K., Krinsky M.L., Maple J., Fanelli R.D., Stromeyer L. *The role of endoscopy in the treatment of patients with peptic ulcer disease. Endoscopy of the gastrointestinal tract, 2010. April.*
4. *Chatila A.T., Bilal M., Guturu P.* Evaluation and treatment of acute pancreatitis. World Journal of Clinical Cases, 2019. May. 6.
5. Gomez KS, Junior KS, Di Saverio S, Sartelli M, Kelly MD, Gomes KS, Gomez FC, Correa LD, Alves CB, Guimarães SF. *Acute calculous cholecystitis: a review of current best practices. World Journal of Gastrointestinal Surgery, 2017. May. 27.*
6. *Morris DL, Hawker PC, Brearley S, et al.* Optimal timing of surgery for bleeding peptic ulcers: a prospective randomized trial. Br Med J ( Clin Res Ed) 1984 ; 288 (6426): 1277-1280. [PMC free article] [PubMed] [Google Scholar].
7. *Ayub F, Khullar V, Banerjee D, Stoner P, Lambrou T, Westerveld DR, Hanayneh V, Kamel AY, Estores D.* Once or twice daily proton pump inhibitor therapy to prevent peptic ulcer rebleeding : Propensity score-matched analysis. Gastroenterological Research, 2018. June.