



# PEPTIC ULCER

**Dr. Hafiz Zubair Alam**

Department of Jarahat  
Aligarh Muslim Unani Medical College  
CAN Hospital, Aligarh.  
Prof. Ahmad Nadim Khan  
Principal

## Engaging in Continued Education

Due to pepsin or gastric acid production, peptic ulcer disease is characterised by discontinuity in the GI tract's inner lining. It penetrates the stomach epithelium's muscularis propria layer. Usually, the stomach and proximal duodenum are affected. It could affect the distal duodenum, jejunum, or lower oesophagus. In this exercise, the aetiology, symptoms, and importance of the interprofessional team in managing peptic ulcer disease are reviewed.

### Objectives:

- Examine the peptic ulcer disease's causes.
- Describe a patient's peptic ulcer disease presentation.
- List the available therapies for peptic ulcer disease.
- Go over the significance of bettering interprofessional team member care coordination to enhance patient outcomes for those with peptic ulcer disease.



## Introduction

Due to pepsin or gastric acid production, peptic ulcer disease (PUD) is characterised by discontinuity in the GI tract's inner lining. It penetrates the stomach epithelium's muscularis propria layer. Usually, the stomach and proximal duodenum are affected. It could affect the distal duodenum, jejunum, or lower oesophagus. Patients with gastric ulcers often have epigastric discomfort 15 to 30 minutes after eating; in contrast, patients with duodenal ulcers typically experience pain 2 to 3 hours after eating. All patients with peptic ulcer disease are now advised to get tested for *Helicobacter pylori*. Some patients may need an endoscopy to confirm the diagnosis, particularly those who have troubling symptoms. Most patients can now be controlled by a triple-drug regimen centred on proton pump inhibitors (PPI).

## Etiology

There are several causes of peptic ulcer disease (PUD), however *Helicobacter pylori*- and NSAID-related PUD make up the bulk of the illness's pathogenesis.

## Causes of Peptic Ulcer Disease

### Common

- *H. pylori* infection
- NSAIDs
- Medications

### Rare

- Zollinger-Ellison syndrome
- Malignancy (gastric/lung cancer, lymphomas)
- Stress (Acute illness, burns, head injury)
- Viral infection
- Vascular insufficiency
- Radiation therapy
- Crohn disease
- Chemotherapy

## ***Helicobacter Pylori*-Associated PUD**

The stomach epithelial cells contain the gram-negative bacillus *H. pylorus*. 90% of duodenal ulcers and 70% to 90% of stomach ulcers are brought on by this bacteria. Lower socioeconomic class individuals are more likely to have *H. pylori* infection, which is frequently acquired during childhood. The bacteria may attach to and inflame the stomach mucosa thanks to a broad spectrum of virulence factors. This causes hypo- or achlorhydria, which causes stomach ulcers.

### *Virulence Factors of Helicobacter Pylori*

1. *Urease*: The secretion of urease breaks down urea into ammonia and protects the organism by neutralizing the acidic gastric environment.
2. *Toxins*: CagA/VacA is associated with stomach mucosal inflammation and host tissue damage.
3. *Flagella*: Provides motility and allows movement toward the gastric epithelium.

## **An NSAID-related PUD**

After *H. pylori* infection, the use of nonsteroidal anti-inflammatory medicines is the second most frequent cause of PUD.

[2] Prostaglandin is often secreted to protect the stomach mucosa. By inhibiting the COX-1 enzyme, NSAIDs prevent the creation of prostaglandins, which lowers the production of gastrointestinal mucus, bicarbonate, and mucosal blood flow.

## **Medications**

- Other drugs beyond NSAIDs that have been linked to PUD's pathogenesis include corticosteroids, bisphosphonates, potassium chloride, and fluorouracil.
- Smoking also seems to be associated with duodenal ulcers, however the relationship is not a straight line. Alcohol can cause acidity by irritating the stomach mucosa.
- The following circumstances result in a hypersecretory environment.
- Zollinger Ellison syndrome
  - Systemic mastocytosis
  - Cystic fibrosis
  - Hyperparathyroidism
  - Antral G cell hyperplasia

## Epidemiology

The probability of developing peptic ulcer disease (PUD) during the course of one's lifetime is between 5% and 10%. [4] [5] Overall, improved hygienic and sanitary circumstances, together with efficient treatment and prudent NSAID usage, have led to a drop in the incidence of PUD around the world. [5] There are four times as many duodenal ulcers as stomach ulcers. Additionally, men are more likely than women to develop duodenal ulcers.

## Pathophysiology

The peptic ulcer disease (PUD) mechanism results from an imbalance between gastric mucosal protective and destructive factors. Risk factors predisposing to the development of PUD:

- *H. pylori* infection
- NSAID use
- First-degree relative with PUD
- Emigrant from a developed nation
- African American/Hispanic ethnicity

There is often a mucosal defect associated with peptic ulcers that spreads to the muscularis mucosa. When the outer, protective layer of the mucosa is harmed, the interior layers are vulnerable to acidity. Additionally, the mucosal cells' capacity to release bicarbonate is impaired.

It is well known that *H. pylori* colonises the stomach mucosa and inflames it. Additionally, the *H. pylori* affects bicarbonate production, which encourages the growth of stomach metaplasia and acidity.

## Histopathology

The lower curvature is where gastric ulcers are most frequently seen, whereas the duodenal bulb is where duodenal ulcers are most frequently found. The ulcer has a smooth foundation and a circular to oval shape. While chronic ulcers have raised borders with inflammation, acute ulcers have normal borders. The muscularis mucosa is not the limit of an ulcer.

## History and Physical

Depending on the location of the disease and age, the signs and symptoms of peptic ulcer disease might change. Differentiating between gastric and duodenal ulcers depends on when they first manifest themselves in relation to meals. Duodenal ulcers can cause nighttime discomfort. People who have a gastric outlet blockage frequently describe having a bloated or full abdomen in the past.

Common signs and symptoms include:

- Epigastric abdominal pain
- Bloating
- Abdominal fullness
- Nausea and vomiting
- Weight loss/weight gain
- Hematemesis
- Melena

Warning symptoms or alarm symptoms that should prompt urgent referral include:

- Unintentional weight loss
- Progressive dysphagia
- Overt gastrointestinal bleeding
- Iron deficiency anemia
- Recurrent emesis
- Family history of upper gastrointestinal malignancy

## Evaluation

A medical history, physical examination, and invasive and non-invasive diagnostics are necessary for the diagnosis of PUD. A thorough history should be taken, and any difficulties should be documented. Epigastric stomach discomfort, early satiety, and fullness after eating are all symptoms that point to PUD. While the discomfort of duodenal ulcers diminishes with a meal and may lead to weight gain, the pain of stomach ulcers increases with a meal and worsens 2 to 3 hours later. Any patient who exhibits anaemia, melena, hematemesis, or weight loss should have their PUD complications—most commonly bleeding, perforation, or cancer—further evaluated. Physical examination results may show epigastric belly soreness and anemia-related symptoms.

## Investigations

1. *Esophagogastroduodenoscopy (EGD)*: Gold standard and most accurate diagnostic test with sensitivity and specificity up to 90% in diagnosing gastric and duodenal ulcers. The American Society of Gastrointestinal Endoscopy has published guidelines on the role of endoscopy in patients presenting with upper abdominal pain or dyspeptic symptoms suggestive of PUD.[6] Patients over 50

years of age and new onset of dyspeptic symptoms should get evaluated by an EGD. Anyone with the presence of alarm symptoms should undergo EGD irrespective of age.

2. *Barium swallow*: It is indicated when EGD is contraindicated.
3. Complete blood work, liver function, and levels of amylase and lipase.
4. Serum gastric is ordered if Zollinger Ellison syndrome is suspected.
5. *Helicobacter pylori* testing:
  - *Serologic testing*
  - *Urea breath test*: High sensitivity and specificity. It may be used to confirm eradication after 4 to 6 weeks of stopping treatment. In the presence of urease, an enzyme produced by H.pylori, the radiolabeled carbon dioxide produced by the stomach is exhaled by the lungs.
  - Antibodies to H.pylori can also be measured.
  - *Stool antigen test*
  - *Urine-based ELISA and rapid urine test*
  - *Endoscopic biopsy*: Culture is not generally recommended as it is expensive, time-consuming, and invasive. It is indicated if eradication treatment fails or there is suspicion about antibiotic resistance. Biopsies from at least 4-6 sites are necessary to increase sensitivity. Gastric ulcers are commonly located on the lesser curvature between the antrum and fundus. The majority of duodenal ulcers are located in the first part of the duodenum.
6. Computerized tomography of the abdomen with contrast is of limited value in the diagnosis of PUD itself but is helpful in the diagnosis of its complications like perforation and gastric outlet obstruction.

## Medical Treatment

Antisecretory medications for peptic ulcer disease (PUD) include proton pump inhibitors and H<sub>2</sub>-receptor antagonists (PPIs). Due to PPIs' greater healing and effectiveness, H<sub>2</sub> receptor blockers have mostly been supplanted. PPIs suppress the stomach's ability to produce acid, relieving symptoms and accelerating recovery. Given that long-term PPI usage raises the risk of bone fractures, treatment may include calcium supplements. Stopping the usage of NSAIDs or switching to a lower dosage helps alleviate NSAIDs-induced PUD. It would be best to stop using corticosteroids, bisphosphonates, and anticoagulants as well. Misoprostol, a prostaglandin analogue, is occasionally used as a preventative measure for peptic ulcers brought on by NSAIDs. A triple antibiotic, proton pump inhibitor, and pylori-inducing PUD therapy combination is the first line of defence. antacids, clarithromycin, and amoxicillin or metronidazole for a period of seven to fourteen days. [7] PPIs and antibiotics cooperate to destroy H. pylori. [8] The antibiotic used should take into account the occurrence of

environmental antibiotic resistance. Quadruple treatment, which uses bismuth and several antibiotics, is used if first-line therapy is unsuccessful.

## Refractory Disease and Surgical Treatment

When a patient is resistant to medicinal therapy, disobedient, or at a high risk of consequences, surgical intervention is needed. Over 5 mm in diameter peptic ulcers that do not cure after 8–12 weeks of PPI medication are referred to as refractory peptic ulcers. Consistent *H. pylori* infection, ongoing NSAID usage, serious comorbidities that hinder ulcer healing, or other disorders such as gastrinoma or stomach cancer are the usual causes. Patients may be candidates for surgical therapy if the ulcer still exists despite taking the aforementioned risk factors into consideration. Both vagotomy and partial gastrectomy are surgical alternatives.

## Differential Diagnosis

- The symptoms of the following illnesses might resemble those of peptic ulcer disease, therefore it's critical to understand how they appear clinically in order to establish the right diagnosis.
- Gastritis - an immune-mediated or infectious inflammatory condition of the gastric mucosa characterised by upper abdominal discomfort and nausea. The clinical picture resembles peptic ulcer disease quite a bit.
- Gastroesophageal reflux disease (GERD): Patients typically report experiencing burning in the lower retrosternal and epigastrium, excessive salivation, or sporadic regurgitation of meals.
- Patients with gastric cancer typically report alarming symptoms such as weight loss, melena, recurrent vomiting, or signs of disease elsewhere in the case of metastases, in addition to stomach discomfort.
- Epigastric or right upper quadrant pain that is more intense and prolonged indicates pancreatitis;
- Biliary colic is an intermittent, very painful condition that causes discomfort in the right upper quadrant or epigastrium.
- Right upper quadrant or epigastric discomfort known as cholecystitis typically lasts for hours, is made worse by fatty foods, and is accompanied by nausea and vomiting. This is further distinguished from biliary colic by fever, tachycardia, a positive Murphy sign, leukocytosis, and abnormal liver functions.

These are some potentially life-threatening conditions that can also have similar presentations.

- Myocardial infarction, especially when the inferior wall and right ventricle are affected; occasionally, patients will also have nausea and vomiting in addition to epigastric discomfort. When a high-risk patient exhibits additional symptoms including dizziness, breathlessness, and abnormal vital signs, the doctor should be on the lookout for this.
- Mesenteric ischemia - whereas acute mesenteric ischemia manifests as severe abdominal pain with sudden onset, the chronic variation typically manifests as persistent post-prandial epigastric discomfort and might be

confused for peptic ulcer illness. Age, the existence of atherosclerosis risk factors, and weight reduction should all trigger an examination for the disease.

Mesenteric vasculitis - in a patient with additional symptoms from underlying systemic vasculitis, unexplained abdominal symptoms with or without lower gastrointestinal haemorrhage should raise the suspicion of mesenteric vasculitis.

## Prognosis

Peptic ulcer disease (PUD) has a very good prognosis when the underlying cause is adequately addressed. The ulcer's recurrence may be avoided by practising proper hygiene and staying away from NSAIDs, alcohol, and smoking. Regrettably, recurrence is frequent, with rates in the majority of series reaching 60%. Gastric perforation brought on by NSAIDs happens to one patient every year at a rate of 0.3%. However, unlike in the past, death rates for peptic ulcer disease have reduced dramatically.

## Complications

If discovered and treated late, peptic ulcer disease (PUD) can have dangerous side effects. The following issues might develop with PUD:

- Gastric outlet blockage; upper gastrointestinal haemorrhage; perforation; penetration; and gastric cancer.

## Deterrence and Patient Education

Nonsteroidal anti-inflammatory medicines (NSAIDs), aspirin, alcohol, cigarettes, and caffeine are examples of substances that should be discussed with patients who have peptic ulcer disease (PUD). If using NSAIDs is required, utilise the lowest dose feasible and think about prophylaxis for individuals who take NSAIDs. Because of the significant link between obesity and peptic ulcer disease, patients should be encouraged to reduce weight. Counseling for stress management may be beneficial in some situations.

## Pearls and Other Issues

Based on size, ulcers are distinguished from erosions. Erosions and ulcers are both terms used to describe lesions that are smaller than or equal to 5 mm in diameter. Because COX-2 is not expressed on the stomach mucosa, COX-2 selective NSAIDs are less likely to result in PUD. Therefore, COX-2 selective NSAIDs are preferable in individuals with a history of PUD. Zollinger-Ellison syndrome is brought on by a gastrin-producing endocrine tumour, which often develops in the pancreas or duodenum. The jejunum and duodenum develop many ulcers as a result. By checking the levels of serum gastrin, it can be diagnosed.

## Enhancing Healthcare Team Outcomes

It is advised to treat peptic ulcer disease using evidence-based methods. Millions of individuals worldwide suffer with PUD, which is a relatively prevalent illness. It has severe morbidity if untreated. The majority of PUD

patients see their primary care provider, but some also visit the emergency room, an urgent care facility, or an outpatient clinic. Healthcare professionals, particularly nurses, need to be aware of this diagnosis because PUD's presentation is frequently ambiguous. Treatment may be postponed since the stomach discomfort might resemble a variety of other diseases.

Once the diagnosis has been determined, it is important to inform the patient about lifestyle adjustments, such as quitting smoking, refraining from alcohol and caffeine, and avoiding taking too many NSAIDs. Gastroenterology nurses maintain tabs on patients, educate the team, and keep them informed about their conditions. To get symptom alleviation and a cure, the pharmacist has to counsel the patient about taking their medications as prescribed. There is evidence to suggest that obesity may be a risk factor for developing peptic ulcer disease, thus it is advisable to seek nutritional advice. The morbidity of peptic ulcer disease can only be reduced through a cooperative effort. The results are favourable for the majority of PUD patients receiving the triple regimen or PPI treatment, however symptom recurrence is not unusual.

## Unani Treatment

Medicine should be given such as (Munaqqi) to clean the Ulcer and the medicine having the power to heal Ulcer (Honey + water) Shahad Ka Pani (Julab), Qurs Kuharba Preference should be given to the Deereona movement of stomach and Deereopa in take of food.

1. Arq-e-Makam, Arq-e-badyan, Arq-e-Gauzuban
2. Qurs Kubarba + Qurs-e-Gulnar+Ab-e-Kasni Sabz Murawwaq 4 tola.
3. Abe Makau Sabz Murawwaq 4 tola, Abe-eBarg-e- Shibt Sabz (Murawwaq 4 tola + Sharbat-e-Habbul Aas 4 tola.
4. Zardi Baizae Murgh + Roghan-e-Gul for abdomen Massage
5. Sagoodana, Yakhni should be given as diet.
6. Treatment includes medication to decrease stomach acid production, stress of spicy food, fating of ginger may prevent ulcer, Garlic can be used for Ulcer.

## References

- Narayanan M, Reddy KM, Marsicano E. Peptic Ulcer Disease and *Helicobacter pylori* infection. Mo Med. 2018 May-Jun;115(3):219-224.
- Lanás Á, Carrera-Lasfuentes P, Arguedas Y, García S, Bujanda L, Calvet X, Ponce J, Perez-Aísa Á, Castro M, Muñoz M, Sostres C, García-Rodríguez LA. Risk of upper and lower gastrointestinal bleeding in patients taking nonsteroidal anti-inflammatory drugs, antiplatelet agents, or anticoagulants. Clin Gastroenterol Hepatol. 2015 May;13(5):906-12.e2.

- Huang JQ, Sridhar S, Hunt RH. Role of Helicobacter pylori infection and non-steroidal anti-inflammatory drugs in peptic-ulcer disease: a meta-analysis. *Lancet*. 2002 Jan 05;359(9300):14-22.
- Snowden FM. Emerging and reemerging diseases: a historical perspective. *Immunol Rev*. 2008 Oct;225:9-26.
- Lanas A, Chan FKL. Peptic ulcer disease. *Lancet*. 2017 Aug 05;390(10094):613-624.
- ASGE Standards of Practice Committee. Banerjee S, Cash BD, Dominitz JA, Baron TH, Anderson MA, Ben-Menachem T, Fisher L, Fukami N, Harrison ME, Ikenberry SO, Khan K, Krinsky ML, Maple J, Fanelli RD, Strohmeyer L. The role of endoscopy in the management of patients with peptic ulcer disease. *Gastrointest Endosc*. 2010 Apr;71(4):663-8.
- Malfertheiner P, Megraud F, O'Morain CA, Gisbert JP, Kuipers EJ, Axon AT, Bazzoli F, Gasbarrini A, Atherton J, Graham DY, Hunt R, Moayyedi P, Rokkas T, Rugge M, Selgrad M, Suerbaum S, Sugano K, El-Omar EM., European Helicobacter and Microbiota Study Group and Consensus panel. Management of Helicobacter pylori infection-the Maastricht V/Florence Consensus Report. *Gut*. 2017 Jan;66(1):6-30.

