STM JOURNALS

Research & Reviews: A Journal of
Pharmacology

https://journals.stmjournals.com/rrjop

ISSN: 2230-9861 (Online)
ISSN: 2349-1299 (Print)
Volume 16, Issue 1, 2026
January–April
DOI (Journal): 10.37591/RRJoP

Review RRJoP

A Systematic Review on Peptic Ulcer Disease and Its Pharmacological Treatment

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Abstract

Introduction: *A chronic gastrointestinal condition known as Peptic Ulcer Disease (PUD) is typified by the formation of ulcers or mucosal erosions in the proximal duodenum and stomach. In the pathophysiology of PUD, the equilibrium between defensive processes, like mucus and bicarbonate secretion, mucosal blood flow, and prostaglandin production, and aggressive factors, such as stomach acid secretion, pepsin activity, and Helicobacter pylori infection, is upset. Although stress and food have historically been blamed, recent research shows that H. pylori infection and long-term use of nonsteroidal anti-inflammatory medicines (NSAIDs) are the main causes. When PUD is clinically diagnosed, symptoms include nausea, epigastric pain, and, if left untreated, bleeding or perforation. The mainstays of pharmacological therapy include proton pump inhibitors, antibiotics, and cytoprotective drugs, which are used to control acid, eradicate H. pylori, and protect mucosa. This review attempts to give a current summary of the pathophysiology, etiology, clinical characteristics, and current pharmacological treatment options of PUD.* ***Background:*** *Helicobacter pylori infection and gastric acid are examples of aggressive forces that interfere with the mucosa's defences, causing damage to the stomach or duodenal lining and Peptic Ulcer Disease (PUD). NSAID use is a significant contributor to ulcer development as well.* ***Methodology:*** *Examining the origins, pathophysiology, and pharmacological therapy of PUD, this review is based on a thorough search of recent scientific publications from sources including PubMed and Google Scholar.* ***Results:*** *The main treatment for ulcer healing and lowering acid secretion is proton pump inhibitors. Antibiotic treatment successfully removes the H. pylori infection, reducing the chance of recurrence. Drugs known as cytoprotective help to improve healing and shield the mucosa. When taken as a whole, these therapies have enhanced patient results.* ***Conclusion:*** *Targeted acid suppression and infection eradication have improved patient outcomes in the pharmaceutical management of PUD, which has advanced significantly. To further lessen the prevalence of PUD globally, future research must concentrate on innovative treatments and individualized treatment strategies.*

Keywords: Duodenal ulcer, *H. pylori* bacteria, NSAIDs, peptic ulcer, PUD

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Received Date: September 14, 2026
Accepted Date: September 29, 2026
Published Date: October 01, 2026

Citation: Priya Haldar, Ashish Kushwaha, Arun Kr. Maurya, Lalit Bisht. A Systematic Review on Peptic Ulcer Disease and Its Pharmacological Treatment. Research & Reviews: A Journal of Pharmacology. 2026; 16 (1): 76–84p.

INTRODUCTION

Peptic Ulcer Disease (PUD) is the term used to describe the development of open sores or ulcers in the first segment of the small intestine (duodenal ulcers) or the mucosal lining of the stomach (gastric ulcers). When the body's defence mechanisms, such as mucus secretion and bicarbonate synthesis, are out of balance with detrimental elements including stomach acid, pepsin, Helicobacter pylori infection, and NSAID use, the disease results. At first, stress, and spicy meals were frequently blamed for PUD. However, more recent studies have shown that H. pylori infection and NSAID use play crucial roles in the pathophysiology of PUD. Burning epigastric pain, nausea, and in extreme situations, gastrointestinal bleeding are some of the symptoms that the illness presents with [1–3].

Effective PUD treatment is a top issue in clinical practice because of its widespread occurrence and influence on patient quality of life. The mainstay of PUD treatment consists of pharmacological treatments that lower the production of stomach acid, eliminate *H. pylori* infection, and shield the mucosal lining. With a focus on recent developments and clinical recommendations that guide treatment choices, this review offers a thorough summary of the etiology, pathophysiology, clinical characteristics, and pharmacological therapies currently employed in PUD treatment (Figure 1).

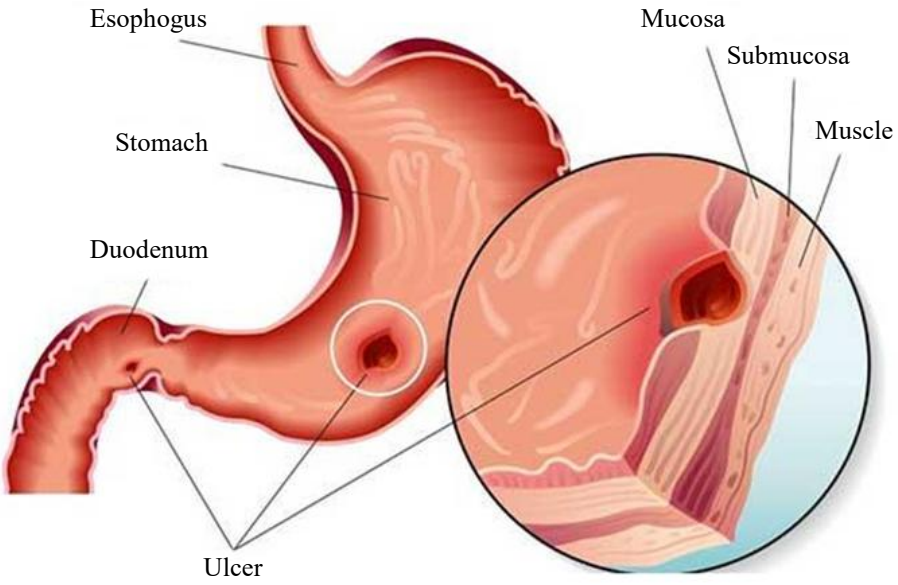

Figure 1. Peptic ulcer disease.

ETIOLOGY AND RISK FACTORS

Peptic ulcer disease (PUD) arises from an imbalance between defensive systems like mucus secretion, bicarbonate synthesis, and mucosal blood flow, and aggressive forces including stomach acid, pepsin, and Helicobacter pylori. One of the main etiological agents is *H. pylori* infection, which damages the mucosal layer, causes inflammation, and encourages the development of ulcers in the lining of the stomach or duodenum. Chronic NSAID use is a major contributing factor as well, as it damages the protective mucosal processes. In addition, alcohol consumption damages the mucosal barrier, smoking slows the healing of ulcers and increases acid production, and psychological stress can make symptoms worse. Additionally, some systemic diseases and genetic predispositions raise the chance of ulcer development, demonstrating the disease's complex character (Figures 2–3).

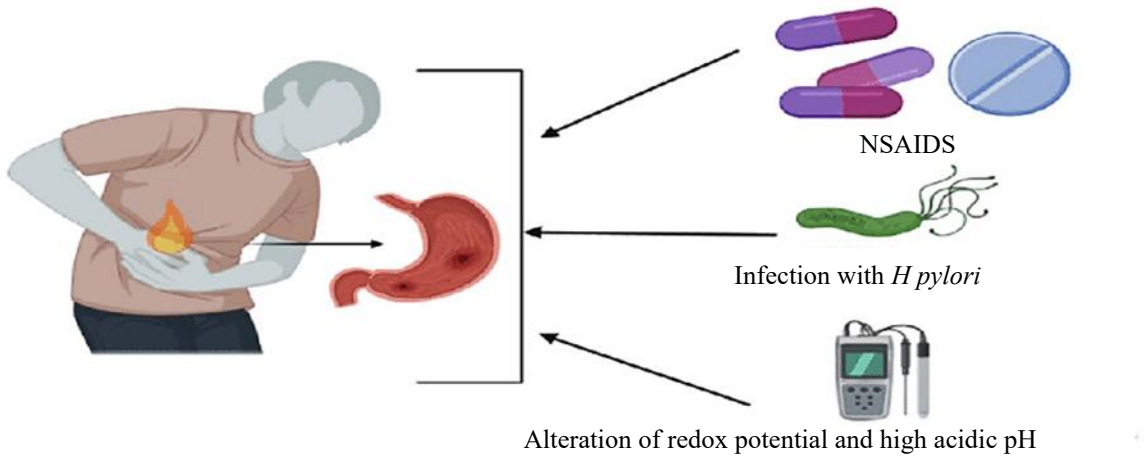

Figure 2. Etiology of peptic ulcer disease.

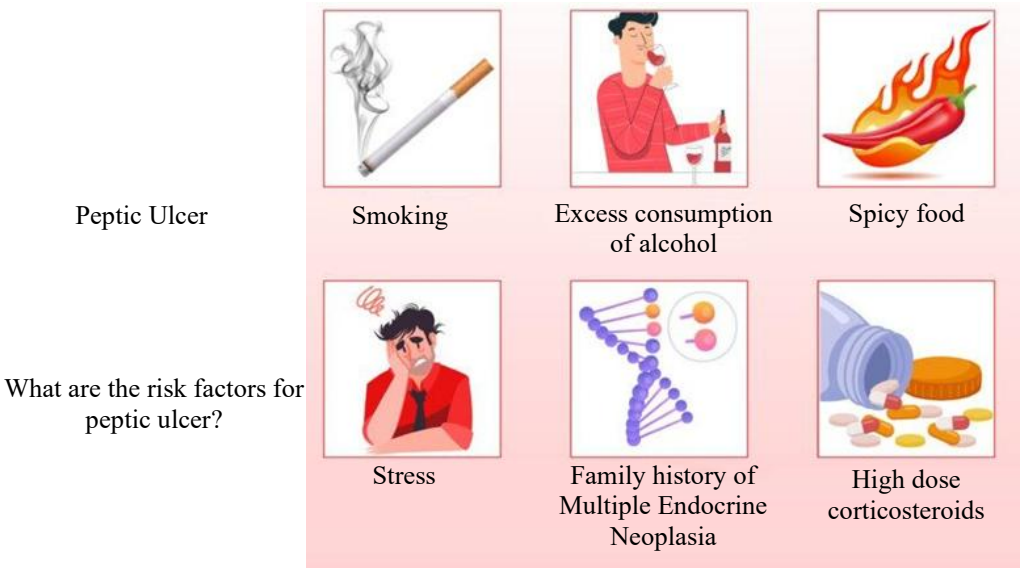

Figure 3. Risk factor of peptic ulcer disease.

Helicobacter Pylori Infection

Helicobacter pylori is a gram-negative, spiral-shaped bacterium that is closely linked to the emergence of peptic ulcers. To ensure they survive in the acidic gastric environment, it produces urease, which transforms urea into ammonia, and colonizes the stomach lining. This bacterium impairs the stomach and duodenum's protective mucosal barrier and causes persistent inflammation, which increases the tissue's vulnerability to acid injury. In certain people, the infection increases gastrin levels and acid secretion by interfering with normal stomach physiology [4–6].

NSAIDs (Non-Steroidal Anti-Inflammatory Drugs)

NSAIDs contribute significantly to the pathogenesis of peptic ulcers, especially in individuals without *H. pylori* infection. These drugs inhibit the cyclooxygenase (COX) enzymes – particularly COX-1 – leading to reduced synthesis of prostaglandins, which play a vital role in maintaining gastric mucosal integrity. Prostaglandins promote mucus and bicarbonate secretion, enhance mucosal blood flow, and support cellular repair. Their depletion weakens the gastric defenses, making the mucosa more susceptible to damage from gastric acid and pepsin. Prolonged NSAID use, especially at high doses or in elderly patients, greatly increases the risk of gastric mucosal injury and ulcer formation [5].

Alcohol Use

While the real cause is still up for discussion, excessive alcohol use can damage and irritate the stomach lining, causing inflammation and making people more susceptible to ulcers, particularly when paired with other risk factors.

Psychological Stress

While stress may not be the direct cause of ulcers, it can worsen pre-existing ulcers or postpone their healing by changing the mucosal defences and acid secretion.

Hereditary Predisposition

A history of peptic ulcers in the family is regarded as a risk factor, indicating a hereditary component to the weakness of the mucosa, acid hypersecretion, or *H. pylori* infection [7–10].

PATHOPHYSIOLOGY OF PEPTIC ULCER DISEASE

The delicate balance between the protective systems of the stomach mucosa and aggressive aqueous factors like pepsin and hydrochloric acid is upset, resulting in peptic ulcer disease. The mucus–bicarbonate barrier, prostaglandin synthesis, mucosal blood flow, and epithelial regeneration of cells all

work together to prevent harm to the normal gastric mucosa. An increase in aggressive agents or a modification in these defence mechanisms may cause mucosal damage and ulcer development [11–15].

Helicobacter pylori is a gram-negative spiral bacterium that grows in the gastric mucosa and is a significant cause of peptic ulcers. To neutralize stomach acid and allow the organism to survive in the acidic environment, it creates the urease enzyme, which hydrolyzes urea to ammonia. In addition, *H. pylori* cause local inflammation and secretes cytotoxins (such as Vac A and CagA is), which damages epithelium and causes ulceration, particularly in the duodenum and antrum.

Another important factor that causes the development of ulcers is non-steroidal anti-inflammatory medicines, or NSAIDs. NSAIDs decrease the production of prostaglandins, which are necessary for mucosal protection, by inhibiting cyclooxygenase (COX) enzymes, especially COX-1. Reduced mucus and bicarbonate secretion, poor blood flow, and increased permeability to acid-induced damage result from this.

Mechanism of Peptic Ulcer Formation

Cause/trigger	Mechanism	Effect
H. pylori infection	– Production of urease → Ammonia → Irritation of mucosa – Cytotoxins (VacA, CagA) → Inflammation	Damage to epithelium → Ulcer formation
Use of NSAIDs	– Inhibition of COX → ↓ Prostaglandins	↓ Mucosal protection → Ulcer development
Contributing Factors	– Alcohol, Smoking, Stress → Reduced blood flow and delayed healing	Increased risk of mucosal damage and ulcers

The Sign and Symptoms of Peptic Ulcer Disease

Depending on the ulcer's location (gastric or duodenal), the degree of mucosal damage, and the existence of complications, peptic ulcer disease (PUD) can manifest a wide range of clinical symptoms. For many people, particularly those who are elderly or taking NSAIDs, the problem may last years without showing any symptoms until complications like bleeding or perforation occur.

- *Epigastric Pain:* It is typically described as a dull, searing, or gnawing pain that is situated immediately behind the breastbone in the upper abdomen. The ache usually happens at night when the stomach is empty or in between meals. Eating meals, drinking milk, or using antacids will briefly ease it, but it usually returns. Whether the ulcer is duodenal or stomach may affect the timing and pattern of the discomfort [16–23].
- *Nausea and Vomiting:* A common symptom of peptic ulcers is chronic nausea, which can occasionally be followed by vomiting. Vomiting may become acute and frequent in cases of severe ulceration or obstruction of the stomach outlet. This symptom, which is more prevalent in gastric ulcers, could be a sign of problems or the disease's advancement.
- *Bloating and Abdominal Fullness:* Patients frequently complain of bloating or distension in their abdomens, particularly after eating. Despite eating small meals, there may be a feeling of fullness that can be confused with other gastrointestinal problems such as functional dyspepsia.
- *Indigestion (Dyspepsia):* Often linked to peptic ulcers, dyspepsia is typified by upper abdominal pain or burning, belching, heartburn, and acid reflux. Without adequate research, it might become worse after meals and resemble the symptoms of gastroesophageal reflux disease (GERD), making diagnosis difficult.
- *Loss of Appetite and Weight Loss:* Peptic ulcer illness frequently causes a noticeable decrease in appetite, which frequently leads to inadvertent weight loss. Usually, this sensation is brought on by the pain and discomfort that comes with eating, especially if the ulcer is in the stomach. Fearing that food may exacerbate their symptoms, people may start to avoid it, particularly if the discomfort gets worse soon after eating. Over time, malnutrition, and decreased caloric intake may result from this avoidance. In long-term situations, persistent malnutrition can result in weakness, exhaustion, and considerable weight loss.

- *Bleeding in the Gastrointestinal Tract:* If ulcers are left untreated or progressed, they may erode into blood vessels. This can manifest as melena (black, tarry stools) from digested blood and hematemesis (blood vomiting), which might look bright red or like coffee grounds. This is a severe illness that must be treated right now.
- *Peptic Ulcer Disease Diagnosis:* A combination of clinical assessment, laboratory testing, and diagnostic imaging or endoscopic procedures are used to diagnose Peptic Ulcer Disease (PUD). Confirming the existence of an ulcer, locating it, identifying its underlying cause (particularly Helicobacter pylori infection), and ruling out consequences like bleeding, perforation, or cancer are the objectives of diagnostic examination. Clinical.
- *History and Physical Examination:* As part of the diagnosis process, the patient is asked about symptoms such as bloating, nausea, searing epigastric pain, and any aggravating or alleviating variables. Risk factors such the use of NSAIDs, alcohol consumption, smoking, and a history of ulcers are also assessed. Upon physical examination, upper abdominal discomfort may be found, but it is frequently nonspecific.

DIAGNOSIS OF PEPTIC ULCER DISEASE

Lab Examinations

- *Complete Blood Count (CBC):* In situations when blood loss is ongoing, this test may show anemia's test called the Fecal Occult Blood Test (FOBT) is used to find blood in feces that may indicate gastrointestinal bleeding. An extremely accurate and non-invasive method of identifying *H. pylori* infection is the urea breath test. The stool antigen test is helpful for both initial diagnosis and follow-up after therapy since it may identify *H. pylori* antigens in feces.

Endoscopy (Esophagogastroduodenoscopy)

Is the most reliable method of diagnosing peptic ulcers. It makes the stomach or duodenal ulcer directly visible and aids in the detection of complications like bleeding or perforation. To check for Helicobacter pylori or rule out stomach cancer, biopsies can also be collected during endoscopy.

Testing for Helicobacter Pylori Infection

Reliable non-invasive techniques to identify an active *H. pylori* infection include the stool antigen test and the urea breath test. These are better than serology, which can also reveal previous infections. Endoscopy can also be used for invasive biopsy-based testing.

Histological Analysis and Biopsy

Histological evaluation by biopsy is essential for the diagnosis and treatment of peptic ulcer disease (PUD), especially for confirming Helicobacter pylori infection and differentiating benign ulcers from malignant ones. Small tissue samples are taken from the duodenum or stomach mucosa during an upper gastrointestinal endoscopy, particularly from the edges of the ulcer and its environs. To identify *H. pylori* germs, these samples are further examined under a microscope using stains like hematoxylin and eosin or specialized stains like Giemsa or Warthin-Starry. Additionally, biopsy aids in the detection of histological alterations such as cancer, intestinal metaplasia, dysplasia, or chronic inflammation.

Imaging Procedures

When endoscopy is not accessible or is not recommended, imaging procedures can help diagnose peptic ulcer disease. Barium meal radiography is a popular technique that highlights the stomach and duodenum on X-rays by having the patient consume a barium contrast agent. This aids in the detection of mucosal abnormalities, big or persistent ulcers, and indications of problems such as blockage. But compared to endoscopy, it is less sensitive. Abdominal X-rays or CT scans are recommended in emergency situations, such as suspected perforations, to identify any problems or free air in the belly. Complications from ulcers, such as wall thickening, perforation, or penetration into adjacent organs, can be seen in detail with CT imaging. Imaging is still essential in cases that are acute or complex, even though it is not the primary option for diagnosis.

Radiological Imaging (Barium Meal X-ray)

A barium swallow X-ray can be utilized in situations where endoscopy is not an option. Although it is less sensitive and specific than endoscopy, it can reveal ulcer craters and obstruction of the stomach outlet.

PEPTIC ULCER DISEASE AND ITS PHARMACOLOGICAL TREATMENT

The development of ulcers or open sores on the stomach lining or the duodenum, the first segment of the small intestine, is a defining feature of peptic ulcer disease (PUD). These ulcers arise from an imbalance between the gastrointestinal mucosa's protective elements and harmful elements like stomach acid and digestive enzymes. PUD is most often caused by Helicobacter pylori infection and long-term nonsteroidal anti-inflammatory drug (NSAID) use. To control symptoms, encourage healing, and avoid complications, effective pharmaceutical treatment is crucial. Usually, this entails the use of drugs that protect the stomach mucosa, eliminate *H. pylori* infection, and decrease the production of gastric acid.

THERAPY FOR ACID SUPPRESSION

PPIs, or Proton Pump Inhibitors

- *Examples* include pantoprazole, omeprazole, and esomeprazole.
- *Mechanism of Action:* PPIs cause a significant and long-lasting decrease in the release of stomach acid by irreversibly inhibiting the H+/K+ ATPase enzyme (proton pump) in the gastric parietal cells. This facilitates mucosal repair and lowers acidity, which aids in ulcer healing.
- *Use:* First-line treatments for duodenal and stomach ulcers, as well as in conjunction with antibiotics to eradicate *H. pylori*.

H2-Receptor Antagonist

- *Examples* include nizatidine, ranitidine, and famotidine.
- *Mechanism of Action:* By competitively blocking gastric parietal cells' histamine H2 receptors, these medications lower the generation of cyclic AMP, which in turn lowers acid secretion.
- *Use:* A good substitute for PPIs for maintenance treatment for mild to moderate ulcers.

Antacids

Neutralize stomach acid to quickly alleviate symptoms. Calcium carbonate, magnesium hydroxide, and aluminum hydroxide are a few examples. Not as a monotherapy, but as an adjunct.

Agents That are Cytoprotective

Strengthen mucosal defences to aid in healing.

- *Sucralfate:* creates a barrier that protects the ulcer site. It is best taken without food.
- *Misoprostol:* A derivative of prostaglandin E1, it promotes the release of mucus and bicarbonate. particularly helpful for ulcers brought on by NSAIDs. Not recommended during pregnancy.
- *Compounds of Bismuth: H. pylori*-fighting antibacterial, ulcer-protective coating. frequently incorporated with quadruple therapy.
- *Ulcers Caused by NSAIDs:* If at all feasible, stop using NSAIDs. In the event that anti-inflammatory treatment is necessary, switch to COX-2 selective inhibitors (such as celecoxib). PPI co-administration is advised.
- *Therapy for Maintenance:* For high-risk people, long-term PPI or H2RA may be necessary to prevent recurrence. Patient education and relapse monitoring are crucial.

NON-PHARMACOLOGICAL TREATMENT

Although medication therapy is still the mainstay of treatment for peptic ulcer disease (PUD), several food and lifestyle changes can aid in healing, symptom management, and recurrence prevention. Peptic ulcer disease non-pharmacological treatment is essential for bolstering medication therapy and averting ulcer recurrence. Avoiding irritants, like alcohol, coffee, and spicy foods (which can worsen damage to the stomach mucosa), is one of the crucial lifestyle changes. It is extremely advised to stop smoking because it increases acid secretion and hinders mucosal repair. Relaxation exercises and getting enough

sleep are two stress-reduction strategies that assist lower the production of stomach acid brought on by stress. Acid secretion can be reduced, and symptoms can be improved by dietary adjustments including eating smaller, more frequent meals rather than larger ones. It is also recommended that patients seek safer alternatives under medical supervision or refrain from using NSAIDs for an extended period of time. These non-pharmacological methods improve overall patient outcomes and promote ulcer healing when used in conjunction with medical treatment.

Change in Diet

To prevent irritation of the stomach mucosa, a balanced diet is necessary. As these foods can worsen ulcer symptoms, patients are recommended to stay away from hot, acidic, and fatty foods. Small quantities of regular meals assist lower acid production and stop ulcers from getting worse.

Quit Smocking

It has been demonstrated that smoking weakens mucosal defences and slows the healing of ulcers. Giving up smoking is essential since it speeds up healing by lowering the production of stomach acid.

Avoid Alcohol

Drinking alcohol causes irritation to the stomach lining and raises the production of acid. Alcohol consumption can be avoided or reduced to lessen ulcer recurrence and mucosal damage.

Stress Management

Prolonged stress has been associated with decreased mucosal blood flow and increased stomach acid output, both of which can exacerbate ulcers. Stress-reduction methods include yoga, meditation, and counseling.

Stay Clear of NSAIDs and Other Irritants

NSAIDs have the potential to directly harm mucosal tissue and suppress protective prostaglandins. NSAIDs should be avoided by patients or used with gastroprotective medications under a doctor's care. It is recommended to co-administer PPIs or switch to COX-2 inhibitors if NSAIDs must be administered.

Consistent Monitoring

In complex or non-healing situations, endoscopy is frequently advised to monitor ulcer healing. Patients should receive education on how to prevent risk factors and stick to their treatment plan.

COMPLICATION OF PEPTIC ULCER DISEASE

Peptic ulcer disease (PUD) can lead to major consequences if left untreated or treated insufficiently, even though it is usually treatable with the right care. Serious morbidity and, in certain conditions, potentially fatal circumstances can result from these consequences. Prompt identification and suitable action are necessary to stop the progression of the disease and its negative effects. The main issues that PUD causes are as follows:

Gastrointestinal Bleeding

In the case of PUD, upper gastrointestinal bleeding is one of the most frequent and potentially deadly side effects. It happens when an ulcer erodes into a blood artery, causing bleeding. Hematemesis (vomiting fresh or coffee-ground blood), melena (black, tarry stools), lethargy, pallor, and dizziness related to anemia are among the clinical manifestations that vary based on the extent of bleeding. Large-scale bleeding can cause hypovolemic shock in extreme situations, necessitating immediate endoscopic procedures, fluid resuscitation, and blood transfusions. In situations that are recalcitrant, surgery may be necessary, however endoscopic therapy employing thermal coagulation, hemoclips, or injectable methods is frequently successful.

Perforation

A full-thickness breach of the stomach or duodenal wall, known as an ulcer perforation, permits the release of intestinal or gastric contents into the peritoneal space. This results in bacterial and chemical peritonitis, which presents with fever, sepsis symptoms, and abrupt, severe, and widespread abdominal

pain. Surgery must be performed right away because it is a medical emergency. Treatment delays dramatically raise the risk of complications and death, particularly in patients who are elderly or immunocompromised.

Penetration

In other instances, an ulcer may enter nearby organs like the pancreas, liver, or biliary system rather than directly through the peritoneal cavity. We refer to this as ulcer penetration. The patient may have excruciating pain that radiates and is unresponsive to standard ulcer treatments. Acute pancreatitis can be caused by pancreatic invasion in particular. Advanced imaging methods, like endoscopic ultrasonography or CT scans, are frequently used in diagnosis. High-dose proton pump inhibitors (PPIs) and, in certain cases, surgical repair are part of the management if problems continue.

Stomach Outlet Obstruction (GOO)

Inflammation and scarring from chronic ulcers, especially those close to the pyloric region, can cause the stomach outlet to narrow or become obstructed. Early satiety, postprandial bloating, persistent vomiting (often of undigested food), dehydration, and weight loss are some of the signs of this disorder, which prevents food from moving from the stomach into the duodenum. Upon physical examination, a succussion splash might be discovered. Typically, barium meal tests or upper GI endoscopy are used to diagnose GOO. Correcting fluid and electrolyte imbalances, nasogastric decompression, PPI medication, and in certain situations, endoscopic balloon dilatation or surgical pyloroplasty are all part of the treatment.

Malignancy Transformation

However, persistent stomach ulcers can progress to gastric adenocarcinoma, even though most peptic ulcers, especially duodenal ones, are benign. For ulcers associated with Helicobacter pylori infection and persistent mucosal inflammation, this is particularly true. Clinical warning indications of cancer include weight loss, increased dysphagia, persistent vomiting, and gastrointestinal bleeding.

CONCLUSION

A common but dangerous gastrointestinal ailment, peptic ulcer disease is brought on by things like an *H. pylori* infection and prolonged use of NSAIDs. Better knowledge of its pathophysiology and causes has led to more focused and efficient therapy. These days, a mix of antibiotics, protecting medicines, and acid suppression medication is essential to treatment. Timely diagnosis, attention to treatment, and lifestyle changes are necessary to prevent problems. Current treatments work well, but new issues, like antibiotic resistance, show that more research and patient education are needed to guarantee greater results along the road.

Acknowledgments

The authors extend their sincere gratitude to the department and the CV Raman Research Wing of JBIT College of Pharmacy for their valuable support in conducting this study. Appreciation is also due to the anonymous reviewer for their insightful and constructive feedback.

Funding

This research was conducted without any external financial support.

Informed Consent Statement

Not applicable.

Data Availability Statement

All relevant data are included within the manuscript.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

REFERENCES

1. Sung JJY, Kuipers EJ, El-Serag HB. Systematic review: The global incidence and prevalence of peptic ulcer disease. Aliment Pharmacol Ther. 2009;29:938–946.
2. Sonnenberg A, Everhart JE. The prevalence of self-reported peptic ulcer in the United States. Am J Public Health. 1996;86:201–205.
3. Vomero ND, Colpo E. Nutritional care in peptic ulcer. ABCD Arq Bras Cir Dig. 2014;27:298–302.
4. Williams MP, Pounder RE. Review article: The pharmacology of rabeprazole. Aliment Pharmacol Ther. 1999;13:3–10.
5. Wroblewski LE, Peek RM, Wilson KT. Helicobacter pylori and gastric cancer: Factors that modulate disease risk. Clin Microbiol Rev. 2010;23:714–730.
6. Wang FW, Tu MS, Mar GY. Prevalence and risk factors of asymptomatic peptic ulcer disease in Taiwan. World J Gastroenterol. 2011;17:1199–1203.
7. Lemos LMS, Martins T, Tanajura GH. Evaluation of antiulcer activity of chromanone fraction from Calophyllum brasiliense Camb. J Ethnopharmacol. 2012;143:432–439.
8. Severed E, Agréus L, Dunn JM. Gastroenterologist practice. Pract. 2019;2:1–8.
9. Goli S. Peptic ulcer disease: Maintenance treatment with H2 blockers. Gastroenterol Pancreat Hepatobiliary Disord. 2017;1(1):01–4.
10. Lanas A, Chan FKL. Peptic ulcer disease. Lancet. 2017;390(10094):613–624.
11. Sung JJY, Kuipers EJ, El-Serag HB. Systematic review: The global incidence and prevalence of peptic ulcer disease. Aliment Pharmacol Ther. 2009;29(9):938–946.
12. Malfertheiner P, Chan FKL, McColl KE. Peptic ulcer disease. Lancet. 2009;374(9699):1449–1461.
13. Chey WD, Wong BCY, Practice Parameters Committee of the American College of Gastroenterology. American College of Gastroenterology guideline on the management of Helicobacter pylori infection. Am J Gastroenterol. 2007;102(8):1808–1825.
14. Laine L, Jensen DM. Management of patients with ulcer bleeding. Am J Gastroenterol. 2012;107(3):345–360.
15. Lanza FL, Chan FKL, Quigley EMM. Guidelines for prevention of NSAID-related ulcer complications. Am J Gastroenterol. 2009;104(3):728–738.
16. Sachs G, Shin JM, Howden CW. Review article: The clinical pharmacology of proton pump inhibitors. Aliment Pharmacol Ther. 2006;23(Suppl 2):2–8.
17. Graham DY, Fischbach L. Helicobacter pylori treatment in the era of increasing antibiotic resistance. Gut. 2010;59(8):1143–1153.
18. Bandyopadhyay D, Chakraborty A. Peptic ulcer: A brief overview of conventional and herbal therapy. Int J Complement Alt Med. 2016;3(5):00099.
19. Hunt RH, Yuan Y. Helicobacter pylori and gastric cancer: The best advice is to eradicate. CMAJ. 2011;183(4):411–416.
20. Konturek PC, Brzozowski T, Konturek SJ. Stress and the gut: Pathophysiology, clinical consequences, diagnostic approach and treatment options. J Physiol Pharmacol. 2011;62(6):591–599.
21. Takeuchi K, Satoh H. Gastric ulcerogenesis by NSAIDs and the roles of anti-ulcer drugs in its prevention. J Physiol Pharmacol. 2015;66(1):3–14.
22. Fashner J, Gitu AC. Diagnosis and treatment of peptic ulcer disease and H. pylori infection. Am Fam Physician. 2015;91(4):236–242.
23. Suerbaum S, Michetti P. Helicobacter pylori infection. N Engl J Med. 2002;347(15):1175–1186.

