IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

‘‘A Comprehensive Review Of Peptic Ulcer Disease Management Strategies’’

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1. Abstract

Peptic ulcer disease (PUD) is a disorder where the lining of the stomach or duodenum develops open sores due to acid related damage. These ulcers occurred due to an imbalance between the harmful factors such as gastric acid and protective mechanisms of the mucosa. The common etiological included infection with Helicobacter pylori and prolonged use of non-steroidal anti-inflammatory drugs (NSAIDs) [(1).] Peptic ulcer disease which includes both stomach and duodenal ulcers, mainly a significant portion of people seeking surgical advice to the world. A peptic ulcer is a develops on the stomach or duodenal lining. Peptic ulcers are caused by factors like hydrochloric acid (HCL), pepsin, refluxed bile, leukotriene’s (LTs), reactive oxygen species (ROS), and defensive factors like the mucus bicarbonate barrier, prostaglandins (PGs) [(2).] the last one-hundred-year written history on a peptic ulcer disease and the treatment options are most common complications. Perforation The reason for reviewing the literature was evaluating most common ideas on how to treat for peptic ulcers (PPU) in general opinions treatment and surgical treatment. Peptic ulcer disease (PUD) is a mucus secretion, bicarbonate production, mucosal blood flow, and prostaglandins. PUD remains a significant global health contributing to morbidity and health care burden, particularly in developing countries.

The primary etiological factors include h pylori infection and prolonged use of Non-steroidal anti-inflammatory drugs (NSAIDs), inhibit the prostaglandin synthesis and compromise mucosal defense. Additional risk factors include smoking, alcohol consumption, stress, and genetic predisposition.

Clinically, patients with PUD may present with Epigastria pain, bloating, nausea, vomiting, and in severe cases, complications such as gastrointestinal bleeding, perforation, and gastric outlet obstruction. Diagnosis is typically established through endoscopic examination, along with non-invasive tests such as the urea breath test, stool antigen test, and serological assays for H. pylori detection or surgery

Management of PUD focuses on the H. pylori infection, reduction of gastric acid secretion, and prevention of the PUD. Pharmacological therapy includes proton pump inhibitors (PPIs), H2 receptor antagonists, antibiotics and Cytoprotective agents. Eradication therapy, or other therapy of a combination of antibiotics

and reduce rates of PUD. Lifestyle modifications and avoidance of NSAIDs are also required to management.

Advances diagnosis technology and treatment, challenges such as antibiotic are early diagnosis therapy, and patient education is reducing disease burden and improving clinical safety.

Keywords: Etiology, Causes, Symptoms, Pathophysiology, Complication, Diagnosis, Radiology [(3)].

2. History

For a thousand of years healthy people have had acute abdominal pain, nausea, vomiting and diarrhea followed by death in a few hours or days. Often these symptoms were observed to poisoning and people have been sent to prison for this King Charles I's daughters, Henrietta-Anne, died suddenly in 1670 (at 26 years of age) after a day of abdominal pain and tenderness. Since poisoning was suspected autopsy was performed revealing peritonitis and a small hole in the anterior wall of the stomach. John Lykoudis, a general practitioner in Greece, treated patients for peptic ulcer disease with antibiotics, beginning in 1958, long before it was commonly recognized that bacteria were a dominant cause for the disease. The history of peptic ulcer disease (PUD) reflects the evolution of medical understanding from ancient theories to modern scientific discoveries. In ancient times, physicians such as Hippocrates described symptoms like abdominal pain and indigestion, but the exact cause of ulcers remained unknown and was attributed to dietary habits and imbalance of body fluids. In the 19th century, pathological studies advanced the understanding of ulcers when Jean Cruveilhier provided detailed descriptions of gastric ulcers, and Rudolf Virchow suggested inflammation as a key factor. During the mid-20th century, stress and lifestyle factors such as smoking and diet were believed to be the primary causes, while later research identified Non-steroidal anti-inflammatory drugs (NSAIDs) as important contributors to mucosal damage.

The discovery of helicobacter pylori by Barry Marshall and robin warren in 1982 with the discovery, which revolutionized the understanding of PUD by establishing it as an infectious disease rather than solely an acid-related condition. This discovery led to a paradigm shift in treatment NSAID use. Gastric acid secretion, and lifestyle factors. Advances in diagnostic techniques such as endoscopy and non-invasive tests have further improved disease management. Overall, the history of PUD demonstrates a transition from empirical observations to evidenced based medicine, significantly improving patient outcomes [(4).]

3. Introduction

The information about "peptic ulcer disease" (PUD) is the upper gastrointestinal tract mucosal breakage caused by acid peptic digestion, which leads to ulcers that penetrate the sub mucosa and go past the muscular is mucosae. It can also develop in the distal esophagus, distal duodenum, jejunum, and diverticulum of Meckel's with the heterotrophic gastric mucosa. The stomach and initial portion of the duodenum are the most typical places it occurs. An imbalance between the stomach mucosa's endogenous protective factors (mucus and bicarbonate secretion, appropriate blood flow, prostaglandin E2, nitric oxide, sulfhydryl compounds, antioxidant enzymes, and others) and aggressive factors (acid and pepsin secretions) leads to peptic ulcer, a chronic disease. The genesis of stomach ulcers has also been linked to behavioral and environmental factors [(5).]This layer passes through the deeper layer of the intestine wall this is called Peptic ulcer [(4).]The PUD is characterized by a significant loss of substance that affects the mucosa of the stomach and/or duodenum and typically extends across the muscular is mucosa to the muscle layer as a result of ambient gastric acid secretion. Peptic ulcer disease (PUD) is a common illness that affects 5-10% of people globally, with significant racial and regional differences. The use of non-steroidal anti-

inflammatory medicines (NSAIDs) and a chronic Helicobacter pylori (HP) infection are the two most common etiological causes.

Peptic ulcer disease (PUD) is not only a disorder of acid imbalance but a complex, multifactorial condition involving disruption of mucosal integrity and failure of protective mechanisms. The gastric mucosa is normally protected by a well-coordinated system that includes mucus-bicarbonate. When these protective factors are weakened or overwhelmed by aggressive factors like mucosal injury occurs, leading to ulcer formation.

One important advancement in understanding PUD is recognizing that it is not merely due to excess acid secretion, but in gastric ulcers, mucosal defense is primarily impaired, whereas in duodenal ulcers, increased acid secretion plays a more dominant role. The presence of H. pylori significantly alters gastric physiology by increasing gastrin secretion, reducing somatostatin levels, and causing chronic inflammation, which further damages the mucosal lining Environmental and behavioral factors also play a crucial role. Smoking has been shown to reduce bicarbonate secretion and mucosal blood flow, thereby delaying ulcer healing. Alcohol consumption may directly irritate the mucosa and increase acid production. The widespread use of NSAIDs (such as aspirin and ibuprofen) contributes significantly to ulcer formation by inhibiting cyclooxygenase (COX) enzymes, leading to decreased prostaglandin synthesis and reduced mucosal protection.

Epidemiologically with variations depending on geographic region, socio economic conditions, and prevalence of H. pylori infection. In developing countries, infection-related ulcers are more common, whereas in developed countries, NSAID-induced ulcers are increasingly prevalent. The disease can occur at any age but is more common in adults, particularly in individuals with chronic NSAID use or underlying comorbidities.

Another important aspect is the natural history of the disease. Untreated ulcers may heal spontaneously but often recur if the underlying cause is not addressed. Chronic ulcers can lead to complications such as hemorrhage, perforation, penetration into adjacent organs, and gastric outlet

Obstruction. These complications significantly increase morbidity and may become life-threatening if not managed promptly.

Recent advances in molecular biology and microbiology have further expanded the understanding of PUD. Research is now focusing on bacterial virulence factors (such as CagA and VacA proteins of h pylori), host genetic susceptibility, and immune responses. Additionally, increasing antibiotic resistance in H. pylori strains poses a major challenge in effective eradication therapy, requiring tailored treatment.

Peptic ulcer disease is now considered a chronic relapsing condition with infectious, pharmacological, and environmental components, other than a simple acid-related disorder. Modern management strategies emphasize a combination of eradication of infection, acid suppression, mucosal protection, and lifestyle modification to achieve long-term healing and prevent.

❖ Objective of peptic ulcer disease

1. Relieve pain and discomfort.
2. To promote healing of the ulcer disease.
3. Eradicate helicobacter pylori infection.
4. To prevent complications like bleeding and perforation.
5. To prevent recurrence of the ulcer.
6. Improve quality of patient life.
7. To reduce gastric acid secretion and maintain acid balance.
8. To strengthen the gastric mucosal defense system.

9. To avoid risk factors like a NSAID, smoking, and alcohol.
10. To provide effective and safe pharmacological therapy.
11. To monitor and manage associated conditions like stress or chronic illness.
12. Ensure a proper patient education about diet and lifestyle changes.
13. To minimize Hospital and healthcare costs
14. To detect and treat Helicobacter pylori infection early.
15. To prevent progression to severe conditions like gastric cancer (in high-risk cases).
16. To maintain long-term remission of symptoms.

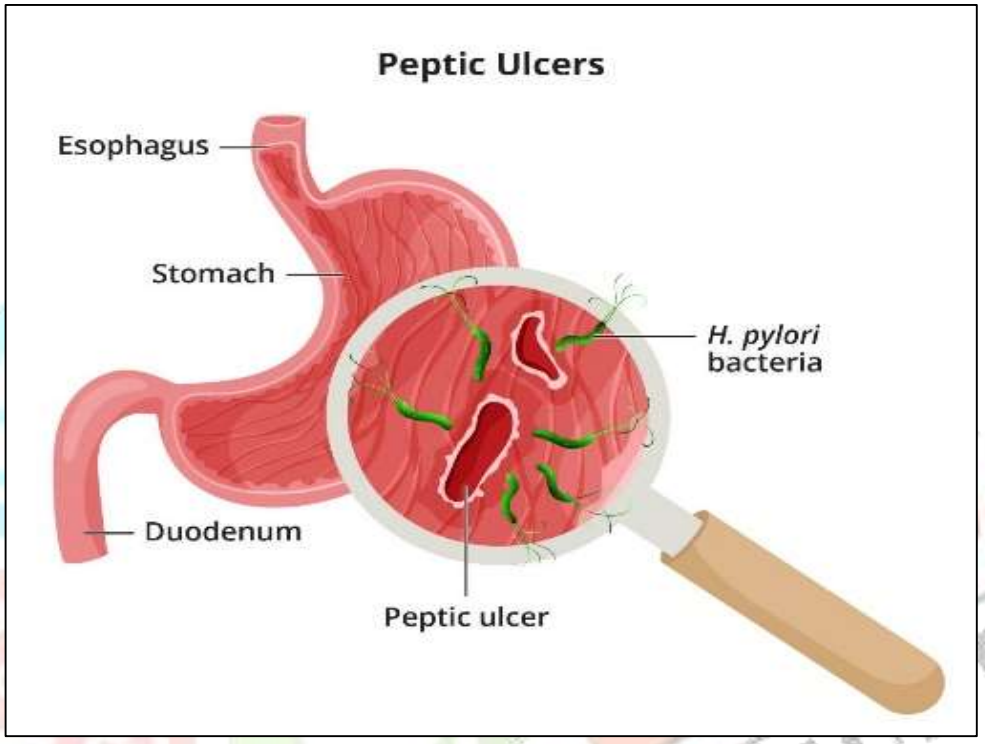


Fig no. 1 Peptic ulcer disease

Definition: -

Peptic ulcer disease can be defined as a condition in which there is a break or erosion in the inner lining of the gastrointestinal tract, particularly in the stomach or duodenum, caused by the action of gastric acid and digestive enzymes. This damage extends beyond the superficial layer and may involve deeper tissue [(6).]

4. Etiology

4.1 Common causes

The two most common etiological causes are the chronic infection with Helicobacter pylori (HP) and the use of Non-steroidal anti-inflammatory drugs (NSAIDs), including of course, the acetylsalicylic acid (ASA). There are other less common causes that can cause a PUD, which are considered together, account for less than 5% of cases. Zollinger-Ellison syndrome or gastrinoma is one among them which is a neuroendocrine tumor, usually located at the head of the pancreas or in the duodenal wall, overactive and gastrin secretory (Table 1).However, we must remember that approximately 5-15% of patients considered Hp negative, despite performing wide comprehensive etiological studies, do not get to find again the precise cause of PU, which are referred to as idiopathic Tobacco abuse and O blood group, are considered as the risk factors for development of ulcer disease. Regarding the existence of genetic factors is unknown, although some cases with familial.

Table no. 1 factor responsible for peptic ulcer disease

Common causes	Frequent causes
• Helicobacter Pylori Infection • NSAIDs and ASA Treatment • Stress Ulcer	• Gastrinoma (Zollinger-Ellison syndrome) • Hyperplasia/hyper function of antral G cells • Systemic mastocytosis • Myeloproliferative Syndromes with basophile • Viral infections (herpes simplex virus tipo I and cytomegalovirus) • Vascular insufficiency (cocaine) • Ischemia caused by stenosis of celiac artery • Radiation • Chemoembolization (via hepatic artery) • Corn's Disease • Type II amyloidosis • Neuhauser syndrome (tremor-nystagmus-ulcer) • Porphyria cutanea tarda • Other drugs (potassium chloride, bisphosphonates Mycophenolate)

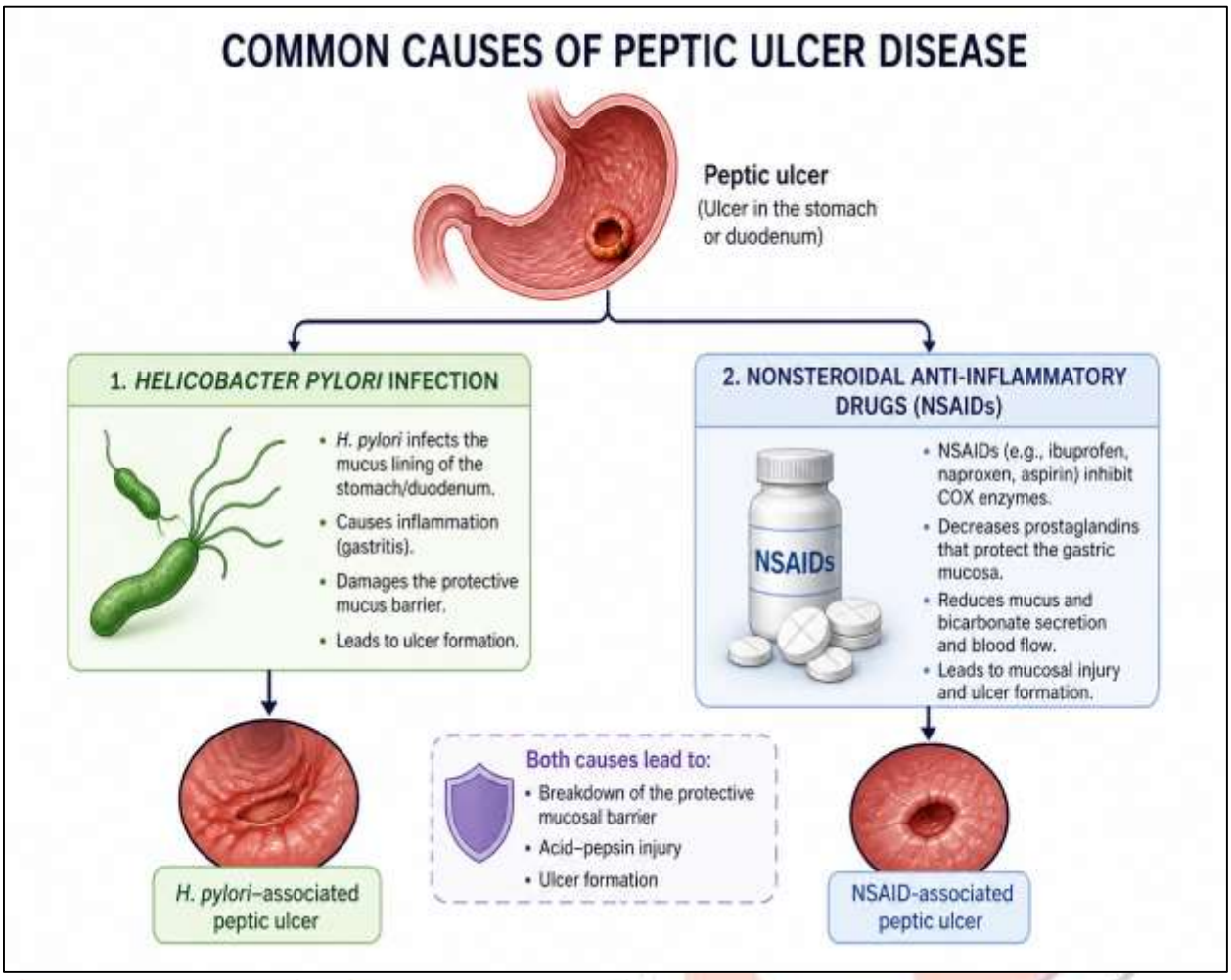


Fig no. 2 Etiological factors of peptic ulcer disease

4.2 Risk factors for NSAIDs related ulcers

1. Smoking
2. Alcohol consumption
3. Stress
4. Genetic predisposition [7].

5. Symptoms

1. Burning or gnawing Epigastric pain
2. Gastric ulcer → worse with food
3. Duodenal ulcer → relieved by food, worse later
4. Nausea or vomiting
5. Bloating
6. Pain related to meals:
7. Duodena
8. Weight loss (more common in a gastric ulcer)

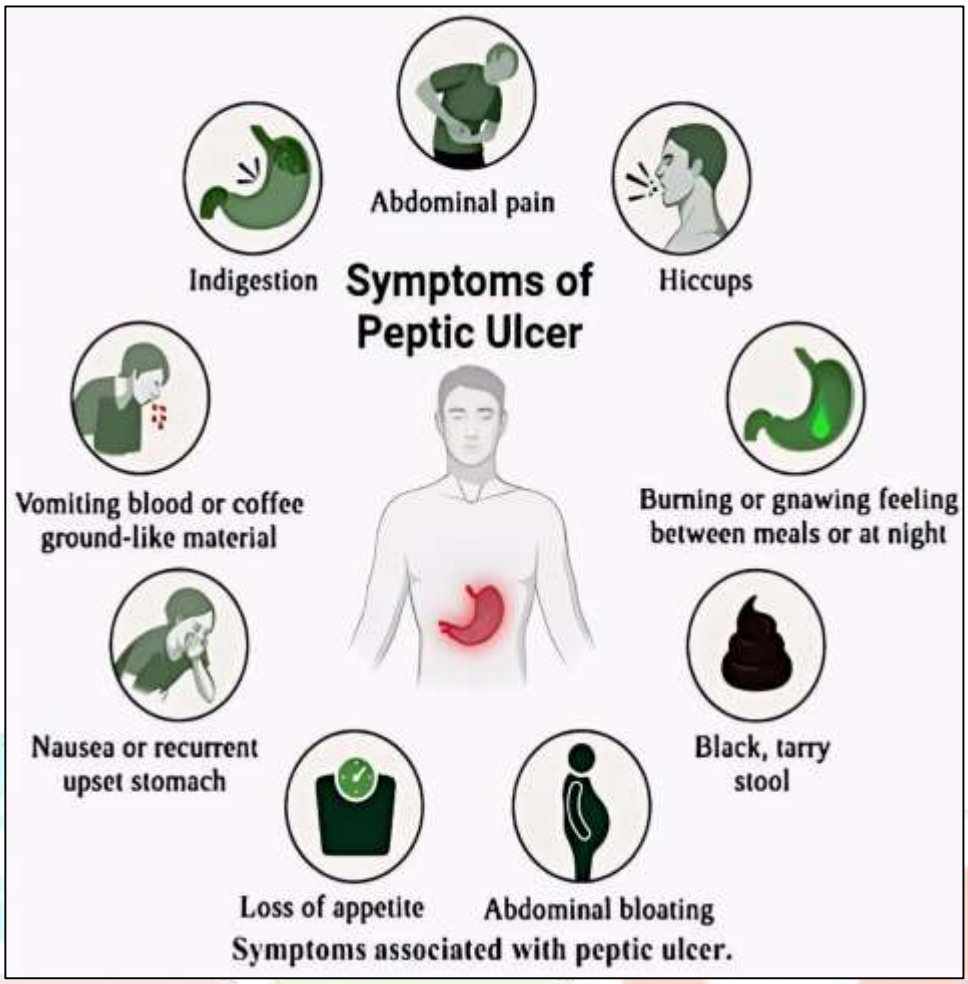


Fig no. 3 Clinical manifestations of peptic ulcer disease

6. Pathophysiology

Historically our understanding of the pathophysiology of peptic ulcer disease focused on abnormalities in the secretion of gastric acid and pepsin and on the suppression of acid as a treatment strategy. gastric hyper secretion associated with gastrinoma in Zollinger Ellison syndrome, antral G-cell hyperplasia, an increase in parietal cell mass, and a physiological imbalance between the antagonistic gastric Hormones gastrin and somatostatinis still an important issue in peptic ulcer disease.it is known that cholinergic hypersensitivity and parasympathetic dominance are related to the stimulation not only of hydrochloric acid but also pepsin, which is often neglected as a cofactor in the development of erosive injury to the gastric mucosa [(2).] there are three clinical phases

6.1 Phase 1: Chemical peritonitis/contamination. The perforation causes a chemical peritonitis. Acid sterilizes the gastro duodenal content; it is only when gastric acid is reduced by treatment or disease (gastric cancer) that bacteria and fungi are present in the stomach and duodenum.

6.2 Phase 2: Intermediate stage. After 6–12 h many patients obtain some relief of pain. This is probably due to the dilution of the irritating gastro duodenal contents by ensuing peritoneal exudates.

6.3 Phase 3: Intra-abdominal infection. After 12–24 h intra-abdominal infection supervenes [(8).]

7. Complications

7.1 Bleeding ulcer

The role of endoscopy in bleeding PUD has been discussed in detail in a previous ASGE guideline. Endoscopy is an effective tool in the diagnosis, prognostication and therapy of bleeding PUD and has been shown in randomized studies to lead to a reduction in blood transfusion requirements, to shortened intensive care unit and hospital stays, to a decreased need for surgery, and a lower mortality rate. Early endoscopy (within 24 hours of admission) has been shown to reduce blood transfusion requirements and length of hospital stay. Patients who are hemodynamic ally stable with endoscopy revealing ulcer without high-risk stigmata may be safely discharge home after endoscopy. Patients with endoscopic stigmata indicating a high risk of bleeding, including adher-ent clots, visible vessels, and active arterial bleeding should all undergo endoscopic therapy to achieve hemostasis and reduce the risk of rebreeding Recurrent bleeding may occur in as many as 10% of patients despite end therapy and the use of high-dose proton pump inhibitors. In patients who rebreeding after initial endoscopic therapy, repeat endoscopic therapy is suggested before considering surgical or radiologic intervention [(9).]

7.2 Perforation

Perforation occurs in approximately 2 to 10 % of peptic ulcers it usually involves the anterior wall of the duodenum (60 %), although it may also occur in antral (20 %) and lesser curve (20 %) gastric ulcers. Perforation of ulcers in children is rare free peritoneal perforation and resulting chemical and bacterial peritonitis is a surgical emergency causing sudden, rapidly spreading, severe upper abdominal pain exacerbated by movement [(3).]

7.3 Obstruction from edema:

It occurs in 2% patients It is most often occurs due to pyloric channel ulcers. It causes crampy abdominal pain [(10).]

8. Types of peptic ulcer disease

8.1 Gastric ulcer

The lower esophagus, distal duodenum, and jejunum can also be affected, to a lesser degree, gastric ulcer disease is more commonly known as peptic ulcer disease (PUD), which is to ulcers that can occur in the stomach and the proximal duodenum (the two organs most frequently impacted by the secretion of pepsin and gastric acid.

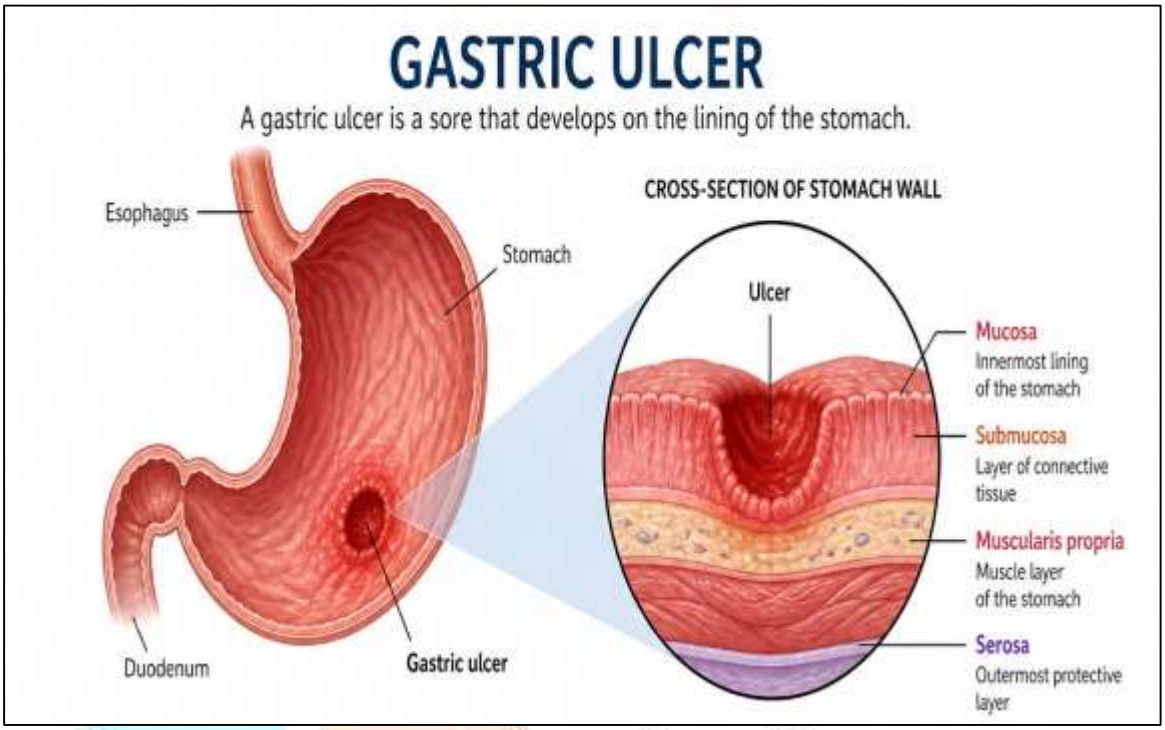


Fig no. 4 Structure of gastric ulcer

8.1.1 Causes of gastric ulcer

1) H. pylori infection
2) NSAIDs use
3) Excess acid secretion
4) Smoking & alcohol
5) Stress and other medical conditions

8.1.2 Symptoms of gastric ulcer disease

1. Burning or gnawing stomach pain (often in the upper abdomen)
2. May improve or worsen after eating
3. Can wake you up at night
4. Indigestion (dyspepsia)
5. Bloating or a feeling of fullness
6. Belching (burping) [11].

8.1.3 Treatment of gastric ulcer

Gastric ulcer management focuses on relieving symptoms, promoting ulcer healing, eradicating Helicobacter pylori infection (if present), and preventing recurrence or complications. Treatment depends on the underlying cause, especially H. pylori infection and NSAID use [12].

8.2. Duodenal Ulcer:

A duodenal ulcer is a type of Peptic Ulcer Disease in which an open sore develops in the duodenum (the first part of the small intestine), usually due to damage from gastric acid and pepsin.

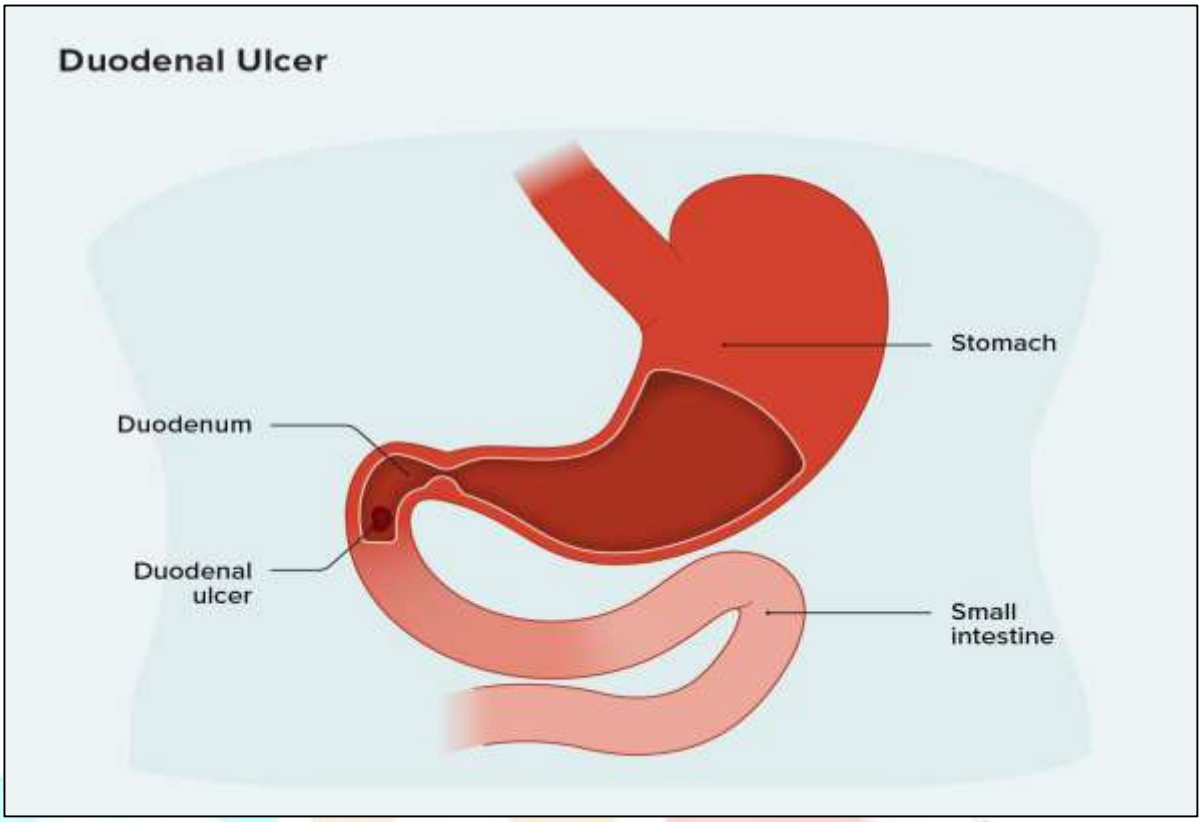


Fig no. 5 Duodenal ulcer

8.2.1 Symptoms of duodenal ulcer

Pain increases when the stomach is empty (especially at night)

1. Relief of pain after eating food
2. Heartburn or acidity
3. feeling of fullness
4. Increased appetite but discomfort after

8.2.2 Cause

1. Infection with Helicobacter pylori (most common cause)
2. Long-term use of medicine like (e.g., Aspirin, Ibuprofen)
3. Excess acid production in the stomach
4. Smoking (increases risk and delays healing)
5. Alcohol consumption
6. Stress
7. Genetic factors (family history of ulcers)
8. Irregular eating habits or unhealthy diet [13].

8.2.3 Treatment of duodenal ulcer

Duodenal ulcer is a common form of peptic ulcer disease, most often associated with Helicobacter pylori infection and, less commonly, with NSAID use. The goals of treatment are rapid symptom relief, ulcer healing, eradication of H. pylori, and prevention of recurrence and complications [14]

8.3 Chronic peptic ulcer

A chronic peptic ulcer is a long standing, recurrent ulcerative lesion occurring in the stomach or duodenum that penetrates beyond the mucosa into the sub mucosa and persists due to an imbalance between factors (gastric acid, pepsin, Helicobacter pylori) and mucosal defense mechanism

8.3.1 Causes of chronic peptic ulcer

1. Long-term use of NSAIDs (e.g., ibuprofen)
2. Excess acid production (sometimes due to stress or certain diseases)
3. Smoking and alcohol use

8.3.2 Symptoms

1. Burning or gnawing abdominal pain
2. Usually in the upper abdomen (epigastric region)
3. Pain may come and go (chronic pattern)
4. Often occurs between meals or at night
5. Pain related to food
6. In gastric ulcer → pain increases after eating
7. In duodenal ulcer → pain improves after eating but returns later
8. Acidity and heartburn
9. Frequent burning sensation in chest or upper abdomen
10. Feeling heavy even after small meals [15].

8.3.3 Treatment of Chronic peptic ulcer

Chronic peptic ulcer disease (PUD) is a long standing or recurrent ulcer condition involving the stomach or duodenum. Management requires not only healing of the active ulcer but also elimination of the cause and prevention. The main etiological factors are Helicobacter pylori infection and long-term NSAID use [16].

9. Diagnosis

A wide variety of methods is available to detect H. pylori infection. Because the organism is trophic for gastric epithelium, it is found primarily in the stomach, where it causes characteristic and easily recognized histologic pattern of acute on chronic inflammation. Typically organisms are plentiful and can be detected using special stains, the most accurate of which is immunohistochemistry with H. pylori specific antibodies A wide variety of other tests is available, ranging from serologic tests for anti H. pylori IgG antibodies to molecular testing using next-generation sequencing Some tests are noninvasive, while others require endoscopy to sample gastric contents (table no:2) Generally noninvasive testing is preferred The diagnostic strategy utilized should reflect not only the clinical indication but also the local availability and costs of the different tests, as well as patient preferences. The presence of the infection elicits a serum immune response, and a number of tests for anti h-pylori IgG are commercially available. Until recently, serology was the most commonly used diagnostic test Currently, serology is generally neither recommended nor reimbursed by Medicare. IgA and IgM anti–H. pylori tests are also available from some laboratories but are generally not approved by the US Food and Drug Administration (FDA) and are not recommended, or to be trusted, because of their low specificity and sensitivity. When using large commercial laboratories in the United States, it is important to request only FDA-approved tests because commercial laboratories also offer, and often preferentially use, in-house derived tests of unknown specificity and sensitivity. Panels of IgG, IgA, and IgM tests provide no added benefit over IgG tests and generally consist of non-FDA-approved tests of unclear diagnostic value. Serologic tests remain positive long after the infection has been eradicated (a serologic "scar"), and therapeutic decisions should not Entirelyrely.

9.1 Test for h-pylori infection

Table no :2 Diagnosis test for peptic ulcer disease

TEST	STRENGTH	WEAKNESSES
NONINVASIVE		
• Serology	• Easily available • Least expensive to patient use • not require medication modification prior to testing	• Does not reliably delineate between active and previous infection. • Cannot be used to confirm eradication.
• Stool antigen test	• High sensitivity and specificity • Can be used to test for active infection and evaluate for eradication	• Stool sample needed, patient aversion. • Requires prior cessation of antibiotics, bismuth products, or proton pump inhibitors to reduce • Risk of false negative result.
• Urea breath test	• High sensitivity and specificity • This test used for active infection and of evaluate for eradication	• Trained personnel are needed to diagnosis patient.

• Endoscopic		
• culture	• Allows testing antibiotic susceptibilities	• Poor availability in some countries In the United States, now available from some major laboratories
• Molecular-based testing	• Detects infection and can assess susceptibility/resistance for all six commonly used • antibiotics Stool can be used Rapid results (days)	• may not be covered by insurance Available only as a "send out," e.g., American Molecular Laboratories, Inc.tp://amlaboratories.com)
• Histology	• Can be used to test for infection and evaluate of eradication • Provide additional information such as inflammation and associated pathology (e.g. intestinal metaplasia atrophic gastric)	• Accurate result required for the pathologist and of special stain preferably immunohistochemical
• Rapid urease tests	• Rapid result • Inexpensive • Good sensitivity and specificity	• Not suitable for all patient

9.2 Non endoscopic tests

9.2.1 Antibody test:

Antibody tests both under diagnose (false negative results) and over diagnose (false positive results) H. pylori infection with some frequency [(3).] Antibody testing offers numerous advantages: it is non-invasive, relatively inexpensive, and overcomes some of the limitations that identify patients with active infection such as urea breath test or stool antigen test. Ingested bismuth compounds, PPIs, or antibiotics do not cause false negative serologic test result [(4).]

9.2.2 X-ray

In this, the patient is made to consume barium, a white, chalky substance that shows up on an X-ray, and is then made to lie down on an inclined examination. The barium is dispersed uniformly throughout the upper digestive tract by tilting, and the X-ray can take pictures from various perspectives. This makes it possible for the physician to find the ulcer and assess its kind and severity [(15).]

9.2.3 Urea breath test

The Urea Breath Test is a non-invasive diagnostic test used to detect infection with Helicobacter pylori, the main etiological agent of peptic ulcer disease [(11).]

9.2.4 Stool antigen test

The stool antigen test (HPSA test) is a non-invasive diagnostic test used to detect active infection with Helicobacter pylori, a major cause of peptic ulcer disease (PUD).

Procedure

1. Collect a fresh stool sample in a sterile container.
2. Sample is processed in the lab.
3. Antigen detection done using immunoassay.
4. Result is reported as:
5. Positive → Infection present
6. Negative → No active infection [10].

10. Radiology

When a patient has upper stomach pain that may be indicative of a perforation, an erect chest X-ray is frequently taken. Nonetheless, with a 98% accuracy rate, contrast enhanced computed tomography is the most effective diagnostic technique. Additionally, CT scans can rule out additional acute abdominal diseases, including acute mesenteric ischemia, acute appendicitis, acute pancreatitis and acute cholestasis.

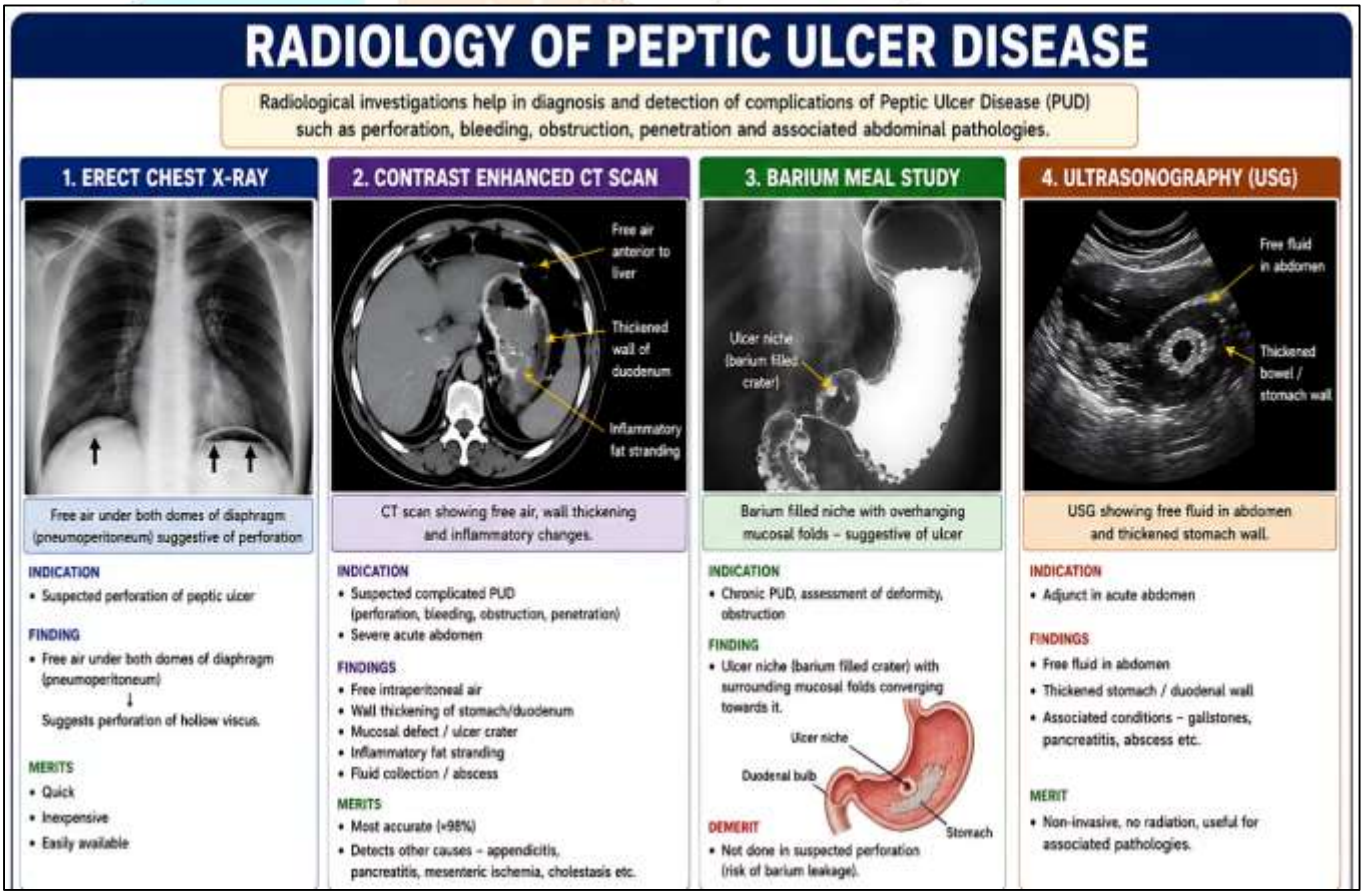


Fig no. 6 Radiology of peptic ulcer disease

11. Non pharmacological management

Non-pharmacological management play important role in controlling and preventing symptoms of peptic ulcer disease.

11.1 Lifestyle Modification

1. Proper Diet
2. Eat a balanced diet (fruits, vegetables, whole grains)
3. Reduce salt, sugar, and fatty foods
4. Avoids a junk food
5. Daily Exercise to maintain healthy diet
6. Helps maintain fitness and overall health
7. Weight Control according to height
8. Maintain normal body weight
9. Prevent obesity
10. Eat meals at regular intervals.

11.2 Stress Management

1. Practice meditation like a yoga.
2. Maintain a healthy work life balance

11.3 Properly rest and sleep

1. Ensure adequate sleep (7–8 hours daily)
2. follow a regular sleep schedule

11.4 Behavioral Changes

1. Stop smoking and alcohol consumption
2. Develop healthy daily habits
3. Group discussion

11.5 Patient Education

1. Inform patients about the disease and symptoms
2. Teach self-care and preventive measures
3. Promote regular health check-ups

11.6 Physical and Supportive Therapy

1. Physiotherapy and exercise therapy
2. Use of hot or cold therapy for pain relief
3. Rehabilitation for recovery

11.7 Environmental Control

1. Maintain cleanliness and hygiene
2. Avoid pollution, dust, and infections

11.8 Social Support

1. Support from family and friends
2. Emotional support improves recovery [(3).]

11.9 Avoid Ulcer-Triggering Substances

1. Avoid spicy foods, caffeine (coffee, tea), and carbonated drinks
2. Limited chocolate and citrus foods if they worsen symptoms
3. Avoid very hot or very cold foods

11.10 Meal Pattern Modification

1. Eat small, frequent meals instead of large meals
2. Do not skip meals
3. Avoid eating late at night (at least 2–3 hours before sleep)

11.11 Posture and Eating Habits

1. Sit upright while eating and for 30–45 minutes after meals
2. Avoid lying down immediately after food
3. Eat slowly and chew food properly

11.12 Hydration

1. Drink adequate water throughout the day (3-4 liter)
2. Avoid excess fluid intake during meals (can increase gastric pressure)

11.13 Avoid NSAIDs

Limit use of pain killers like ibuprofen, aspirin without any medical advice

11.14 Probiotic Intake

1. Include cow milk, buttermilk
2. Helps improve gut health and may reduce H. pylori effects

11.15 Weight Management (Advanced)

1. Avoid abdominal obesity
2. Reduces pressure on stomach and acid reflux risk

11.16. Relaxation Techniques

1. In addition to yoga:
2. Progressive muscle relaxation
3. Mindfulness therapy
4. Listening to calming music
5. Play games

11.17 Avoid Tight Clothing

Tight clothes increase abdominal pressure and worsen symptoms

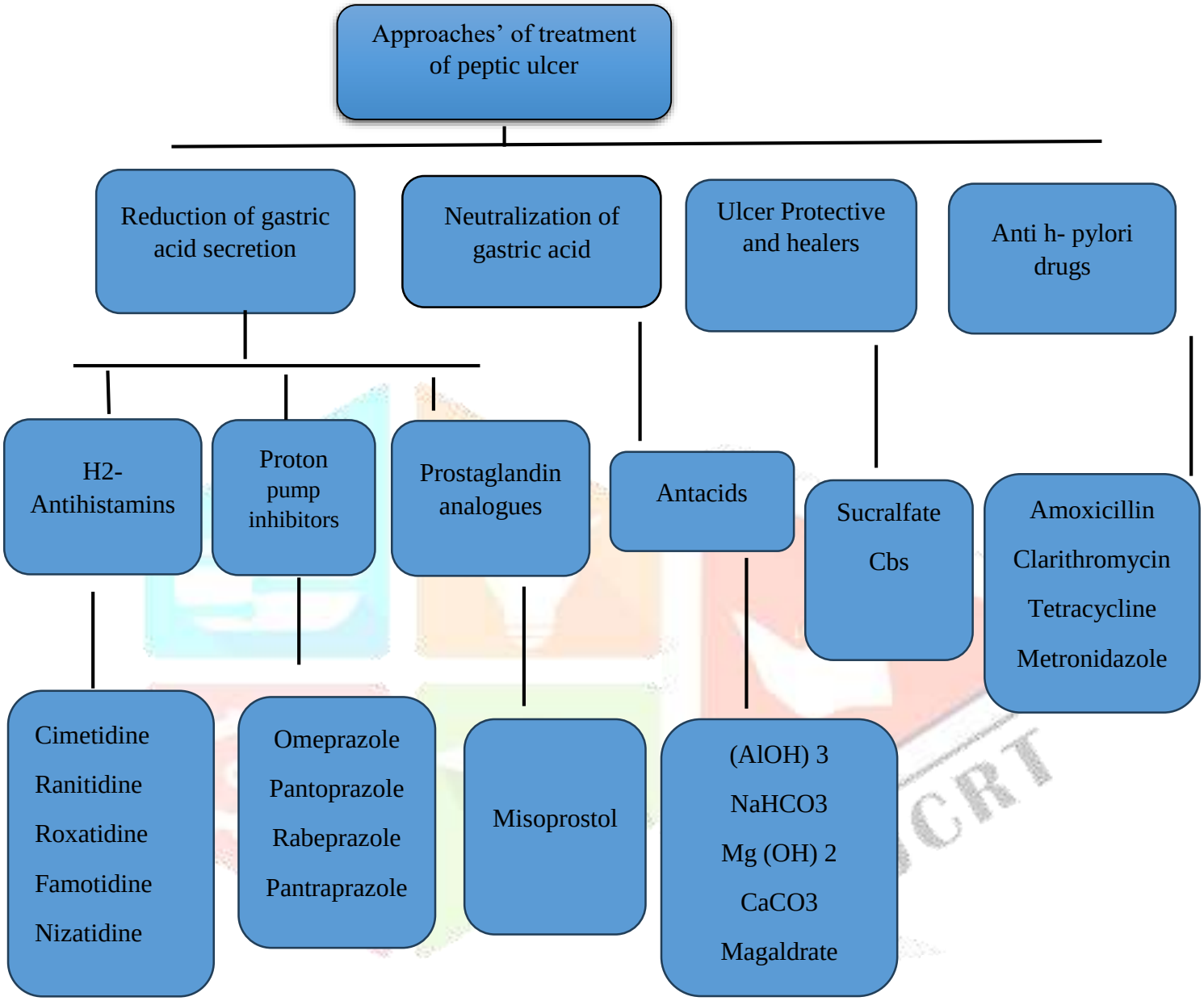
11.18 Patient Education:

1. Early symptom recognition
2. Avoiding self-medication
3. Importance of follow-up

11.19 Avoid harmful habits

1. Stop smoking
2. Limit or avoid alcohol consumption

12. Pharmacological management



13. Mechanism of action

13.1 H2 receptor antagonists

They exhibit competitive inhibition at the parietal cell H2 receptor. The volume of gastric secretion and concentration of pepsin are also reduced. H2 antihistamines reduce acid secretion stimulated by histamine as well as by gastrin and cholinomimetic agents through two mechanisms:

1) Histamine release from ECL cells by gastrin or vagal stimulation is blocked by binding to the parietal cell H2 receptor.

2) Direct stimulation of the parietal cells by gastrin or Ach results in diminished acid secretion in the presence of H2 receptor blockade. It appears that reduced parietal cell CAMP levels attenuate the intracellular activation of protein kinases by gastrin and Ach.

Medication like ranitidine and famotidine decrease acid production and provide symptomatic relief [16].

13.2 Proton Pump Inhibitors:

PPI is better action produce than H2RAs in reducing the risk of Non selective NSAID-related gastric and duodenal ulcer. PPIs are also as effective as misoprostol but better tolerated. All PPIs are effective when used such drug as omeprazole pantoprazole are commonly used to reduce gastric acid secretion [17].

13.3 Antacids

Antacids are the first such agents were antacids postprandial acid output after antacid ingestion wassigni ficantly increased by an average of 16% the level of gastric juice also rose. Other side effects were increase in gastrin release, gastric secretion, gastric emptying rate and duodenal acid load. 25% of patients suffered from diarrhea, some from duodeno gastric reflux Multiple receptors showed interactions: H2 receptor, acetylcholine receptor and carbamyl-choline interact and seem to sensitize cells like gastrin cells The goal of antacid treatment is to raise the pH to 3.0 as gastric content end-point, to reduce the load of acid delivered into the duodenum, and to reduce pepsin activity by lowering conversion of pepsinogen into pepsin. The bile acid binding properties in the duodenum increase gastric reflux and play a pathogenic role in GU. The four types of antacids: calcium, carbonate, and magnesium and aluminum hydroxide all require evaluation of the speed with which an antacid combines with acid. Alteration in bowel movement it is a frequently combined with constipation [4].

13.4 The Peptic ulcer disease (pud) Current possible medication-based management

The majority of H. pylori eradication treatments over the previous 20 years have included antibacterial medicines in combination with anti-secretory medications. The current global agreement states that triple therapy, consisting of two PPIs per day, 500 mg of clarithromycin per day, and either 1 g of amoxicillin per day (PPI-CA) or 500 mg of metronidazole per day (PPI-CM) for a duration of 7–14 days, should be the first line of treatment. PPI therapy administered twice a day is more effective than once a day First-line treatments have a 70% to 95% success rate in eradication, and 10 and14 day regimens are typically 7–9% more effective than the most popular 7day regimens. The type of antibiotics used depends on treatment failure caused by noncompliance and, depending on the region, bacterial resistance. Since bacteria are more resistant to metronidazole and hardly resistant to amoxicillin, amoxicillin is preferred over metronidazole in first line therapy the use of PPI-CA as a first-line treatment is also advocated over PPI-CM due to worries that PPI-CM treatment may result in subsequent resistance to the most effective forms of treatment, metronidazole and clarithromycin. As a first-line treatment, quadruple therapy-200 mg of bismuth four times a day, 500 mg of metronidazole three times a day, 500 mg of tetracycline four times a day, and a PPI twice a day for at least seven days-has also gained recognition. A global panel of experts summed up this guiding principle in the Maastricht Consensus Report: since re-treatment with the original regimen is not advised, second-line treatment is carried out using a different selection of antimicrobial medications than those used in first-line treatment. Even with this cautious approach, eradication may not always succeed, necessitating further pharmaceutical intervention. Following this, bacterial culture is used to inform the selection of antibiotic therapy, and the prescription of a third-line treatment is based on the microbial sensitivity to antibiotics [9].

13.5 Surgery

Surgery is indicated in patients who are intolerant of medications or do not comply with medication regimes, and those at high risk of complications (e.g., transplant recipients, patients dependent on steroids or NSAIDs, those with giant gastric or duodenal ulcer, those with ulcers that fail to heal with adequate treatment). Surgery should also be considered for patients who have a relapse during maintenance treatment or who have had multiple Surgical options for duodenal ulcers include truncal vagotomy and drainage (pyloroplasty or gastrojejunos-tomy), selective vagotomy (preserving the hepatic and celiac branches of the vagus) and drainage, highly selective vagotomy (division of only the gastric branches of the vagus, preserving Learjet's nerve to the pylorus), or partial gastrectomy. Surgery for gastric ulcers usually

involves a partial gastrectomy. Procedures other than highly selective vagotomy may be complicated by post-procedure dumping and diarrhea [16].

13.6 Proton Pump Inhibitors:

PPIs is more effective than H2RAs in reducing the risk of Non selective NSAID-related gastric and duodenal ulceration. PPIs are also as effective as misoprostol but better tolerated. All PPIs are effective when used in standard doses

13.7 Cox-2 Selective Inhibitors:

Selective COX-2 inhibitors are no more effective than the combination of a PPI and a nonselective NSAID in reducing the incidence of ulcers and are associated with a greater incidence of CV events (e.g., ischemic stroke).Celecoxib is the only agent in this class that remains on the market; its postulated improved GI safety when compared to Non selective NSAIDs. Longer-term studies evaluating the CV risks associated with the use of COX-2 inhibitors have found a higher incidence of CV mortality with these agents compared to traditional NSAID [3].

13.8 Ulcer protective:

Sucralfate is a complex sucrose salt in which hydroxyl groups have been substituted by aluminum hydroxide and sulphate. Sucralfate may act by mechanisms:

- In the gastric environment, aluminum hydroxide dissociates, leaving the polar sulphate anion, which can bind to positively charged tissue proteins found within the ulcer bed, and providing a physicochemical barrier impeding further tissue injury by acid and pepsin.

- It may induce a trophic effect by binding growth factors such as EGF, enhance prostaglandin synthesis, stimulate mucous and bicarbonate secretion, and mucosal defence and repair [1].

13.9 Ulcer prevention

The realization in recent years that selective cyclooxygenase (COX)-2 inhibitors were not as 'GI safe' as their promotional materials suggested led to several evaluations of alternative methods for preventing gastric ulceration induced by non-steroidal anti-inflammatory drugs (NSAIDs). Proton pump inhibitors (PPIs) have long been suggested to reduce the incidence of serious gastrointestinal complications during NSAID use. Further support to this notion by reporting that the use of PPIs was associated with a significant reduction in the risk of ulcer in both acute and chronic users of NSAIDs. Moreover, the number needed to treat' to avoid one peptic ulcer in the elderly was low: three for both acute and chronic NSAID users. Chan et al. examined the utility of PPIs for prevention of recurrent ulcer bleeding in patients taking aspirin to prevent vascular diseases. Patients who presented with ulcer bleeding were randomized, after their ulcers had healed, to receive aspirin plus a PPI (esomeprazole), or another antiplatelet drug (Clopidogrel). Clopidogrel is an ADP receptor antagonist that inhibits platelet aggregation and is recommended for patients who have major gastrointestinal intolerance of aspirin [4].

13.10 Dietary Adjustments

The addition of omega-3 poly unsaturated fatty acids is recommended because they reduce inflammation and guard against a stomach ulcer. Eat nothing hot. Avoid eating late at night. Eat a well-balanced food, low-cholesterol diet

13.11 Lifestyle Adjustments:

Refrain from going straight to bed after eating. Raise the bed's head. Stay calm [7].

13.12 Cytoprotective agents

Drugs like sucralfate and misoprostol help protect the gastric mucosa and enhance healing.

14. Future direction

In the past, H. pylori infection and the use of NSAIDs have dominated research into peptic ulcer disease and have shaped its diagnosis and treatment. Even though H. pylori infection can be successfully controlled with currently available pharmacologic approaches, there is still a serious need for novel eradication mono therapies that will simplify treatment regimens while improving eradication rates Molecular techniques will continue to help us identify genetic factors that predict the development of idiopathic ulcers. The identification of an H. pylori gene that promotes the development of duodenal ulcers has introduced a novel marker that can identify patients at increased risk of duodenal ulcer development and reduced risk for gastric atrophy and cancer The array of predisposing factors is, however, predominantly host oriented; that is, based on the genetic characteristics of the patient. The exist once of host-related differences in the physiology of acid secretion might lead to the identification of genetic markers associated with peptic ulcer disease. Such markers might, in the future, help to identify patients at high risk of or with susceptibility to peptic ulcer disease [(10).]

14.1 Artificial Intelligence in diagnosis

1. AI-assisted endoscopy
2. Detect ulcers early
3. Differentiate benign vs. malignant lesions
4. Improves diagnostic accuracy and reduces human error.

14.2 Other prevention strategies

1. Focus on reducing risk factors
2. Rational use of NSAIDs
3. Co-therapy with proton pump inhibitors
4. Public health education and screening programs

14.3 Personalized Medicine

1. Future treatment may be customized according to:
2. genetic profile,
3. antibiotic resistance pattern,
4. Patient lifestyle and Micro biome.
5. This can improve treatment success and reduce recurrence.

14.4 Use of Herbal and Natural Therapies

1. Many medicinal plants and natural compounds are being investigated for anti-ulcer activity.
2. Future research may identify safer and cost-effective alternatives to synthetic drugs.

15. Conclusion

Peptic ulcer disease is a condition that requires to management. Understanding the risk factors, pathophysiology, and treatment options is essential for healthcare providers to provide effective care. The role of H. pylori infection and NSAID use in the development of PUD highlights the importance of prevention and early diagnosis. Endoscopy and radiology play an important role in diagnosis, while medications and surgery offer effective treatment options. By adopting a multi-faceted approach to PUD

management, healthcare providers can improve patient outcomes and reduce the risk of complications. Further research is needed to develop more effective prevention and treatment strategies for this prevalent gastrointestinal disorder [(1).] Peptic ulcer is help in common clinical concern in our society, affecting people of all ages. Peptic ulcer disease is predicted to continue to have a large global influence on health care delivery, health economics, and patient quality of life as the prevalence of the illness rises with age Peptic ulcer illness continues to be a problem in medical visits. The majority of people who present with dyspepsia should be examined for peptic ulcer disease. Gastric acid secretion must be understood in order to determine which portion of the stomach is most impacted by the etiologic agent of peptic ulcer disease. Pylori has remained a risk factor for the development of peptic ulcer disease until now. This bacteria's predilection location determines its clinical prognosis. Understanding the pathophysiology of peptic ulcers can help doctors be more aware of potential complications, such as stomach cancer.

15. Reference

1. Jaiswal, F., Rai, A. K., Wa, P., Wal, A., & Singh, S. P. (2021). Peptic ulcer disease: A review. Asian Journal of Pharmaceutical Education and Research (AJPER), 10(4), 1–17. https://doi.org/10.38164/AJPER/9.4.2021.01-17

2. Bertleff, A., & Lange, J. F. (2010). Perforated peptic ulcer disease: A review of history and treatment. Digestive Surgery, 27, 161–169. https://doi.org/10.1159/000264653

3.Gulia, Y., & Chaudhary, M. (2011). Pharmacological approaches in peptic ulcer disease. Pharmacologyonline, 3, 48–70.

4. Srivastava, Y., Kumar, V., Srivastava, Y., & Kumar, M. (2023). Recent advance in peptic ulcer treatment. Journal of Advances in Medical and Pharmaceutical Sciences (JAMPS), 25(11), 14–27. https://doi.org/10.9734/JAMPS/2023/v25i11651

5. Sadiq, K., Rizwan, B., Noreen, S., Fatima, A., Sheraz, M., Shafqat, M., & Rashid, H. M. (2020). Peptic ulcer disease: Etiology and management. EAS Journal of Pharmaceutical Science, 2(5). https://doi.org/10.36349/easjnfs.2020.v02i05.003

6. Lauret, M. E., Rodriguez-Pelaez, M., Perez, I., & Rodrigo, L. (2015). Helicobacter pylori and peptic ulcer disease. Journal of Gastrointestinal and Liver Diseases. https://doi.org/10.19104/jgld.2015.10

7. Yuan, Y., Padol, I. T., & Hunt, R. H. (2006). Peptic ulcer disease today. Nature Clinical Practice Gastroenterology & Hepatology, 3(2). https://www.nature.com/clinical practice/gasthep

8. American Society for Gastrointestinal Endoscopy. (2010). Role of endoscopy in the management of peptic ulcer disease. Gastrointestinal Endoscopy, 71(4). https://doi.org/10.1016/j.gie.2009.11.026

9. Ramakrishnan, K., & Salinas, R. C. (2007). Peptic ulcer disease. American Family Physician. https://www.aafp.org/afp

10. Ghonge, P. G., Hatwar, P. R., Shelke, P. G., Bakal, R. L., & Khandare, S. S. (2025). GSC Advanced Research and Reviews, 23(3). https://doi.org/10.30574/gscarr.2025.23.3.0156

11. American College of Gastroenterology. (2017). Clinical guideline for the management of peptic ulcer disease.

12. Ford, A. C., et al. (2016). Helicobacter pylori eradication therapy for peptic ulcer disease: A systematic review. The Lancet.

13. Malfertheiner P et al. Maastricht V/Florence Consensus Report (Gut, 2017

14.Davidson's Principles and Practice of Medicine, 2021

15. Harrison's principles of internal medicine (21st Ed.). McGraw-Hill.

16. Jaswanth, K., Kumar, C., & Venkatesh, J. (2022). Peptic ulcer disease: A review. UPI Journal of Pharmaceutical and Medical Health Sciences, 5(1), 19–26. https://doi.org/10.37022/jpmhs.v5i1.73

17. Holle, G. E. (2010). Peptic ulcer disease overview. International Journal of Molecular Medicine, 25, 483–491. https://doi.org/10.3892/ijmm_00000368