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Management of *helicobacter pylori* infection-petic ulcer disease

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Abstract

Peptic ulcer disease typically affects the stomach and proximal duodenum the primary causes in the United States are *Helicobacter pylori* infection and the use of nonsteroidal anti-inflammatory drugs. Weight loss, appetite loss, and epigastric discomfort particularly pain that can be alleviated by eating or taking antacids are the hallmarks of peptic ulcer disease makes you wake up in the middle of the night or in between meals. Antisecretory therapy (ideally with a proton pump inhibitor) should be administered for four weeks after the infection has been completely eradicated. The most frequent reason for surgery is bleeding, Perforation and obstruction of the gastric outlet are rare but dangerous side effects. If problems arise or the ulcer is not improving with medication, surgery is recommended as a surgical emergency, peritonitis necessitates peritoneal toilet, laparotomy, patient resuscitation, the placement of an abdominal patch, and in certain cases, surgery to control ulcers.

Endoscopy should be recommended for patients who have ongoing symptoms. Surgery is recommended. The most frequent reason for surgery is bleeding the majority of bleeding is controlled by endoscopic therapy and proton pump inhibitor administration. Rare but dangerous side effects include gastric outlet obstruction and perforation. Peritonitis is a surgical emergency that necessitates peritoneal toilet, laparotomy, patient resuscitation, the implantation of an abdominal patch, and, in certain cases, surgery to control ulcers.

Keywords: Peptic ulcer disease, *helicobacter pylori*, Nonsteroidal anti-inflammatory drugs (NSAIDs), Gastric outlet obstruction, perforation, peritonitis

Introduction

An ulcer is an open sore. The word "peptic" suggests that the problem stems from acid. The two most common types of peptic ulcer are called “gastric ulcers” and “duodenal ulcers” It refers to the location where the ulcer is found gastric ulcers is found at the beginning of the small intestine [also known as small bowel] known as duodenum [1]. Food is broken down by the stomach's production of acids and enzymes that are resistant to a variety of dangerous chemicals, including alcohol, hydrochloric acid, refluxed bile salts, and other irritants. In addition to preventing damage, the stomach's numerous physiological defence mechanisms also promote rapid mucosal healing and enable the mucosa to retain a high level of resistance to harm. When irritants overwhelm these defence mechanisms, a stomach mucosal lesion, such as an ulcer or gastric erosion, may develop [1].

Definition

A peptic ulcer is a break or sore in the lining of the stomach, lower oesophagus, or duodenum [the first part of the small intestine] that occurs due to the corrosive effects of stomach acid and pepsin.

There are two main types

- **Gastric ulcers:** Occur in the stomach
- **Duodenal ulcers:** Occur in the duodenum [2]

Epidemiology
The lifetime prevalence of peptic ulcer disease in the general population is approximately 7.5%. However, among individuals infected with *Helicobacter pylori*, the lifetime prevalence increases significantly to about 15.0%, with an annual incidence of nearly 1.0% in these infected individuals. The age of onset also varies with ulcer type, as gastric ulcers most commonly peak around the age of 60 years, whereas duodenal ulcers tend to occur earlier, with a peak incidence around the age of 45 years [3, 4].

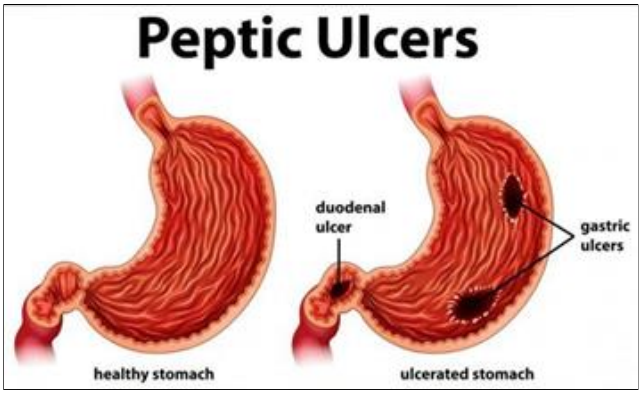

Fig 1: Systematic representation of PUD

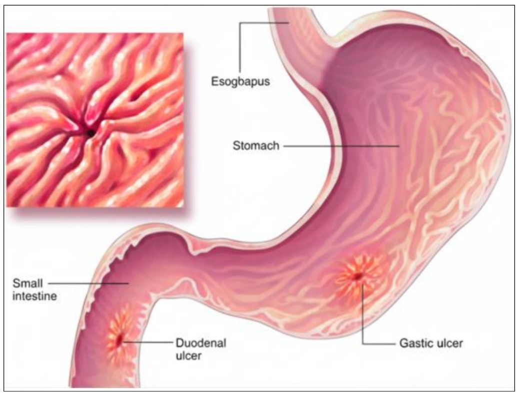

Fig 2: Gastric and duodenal ulcer

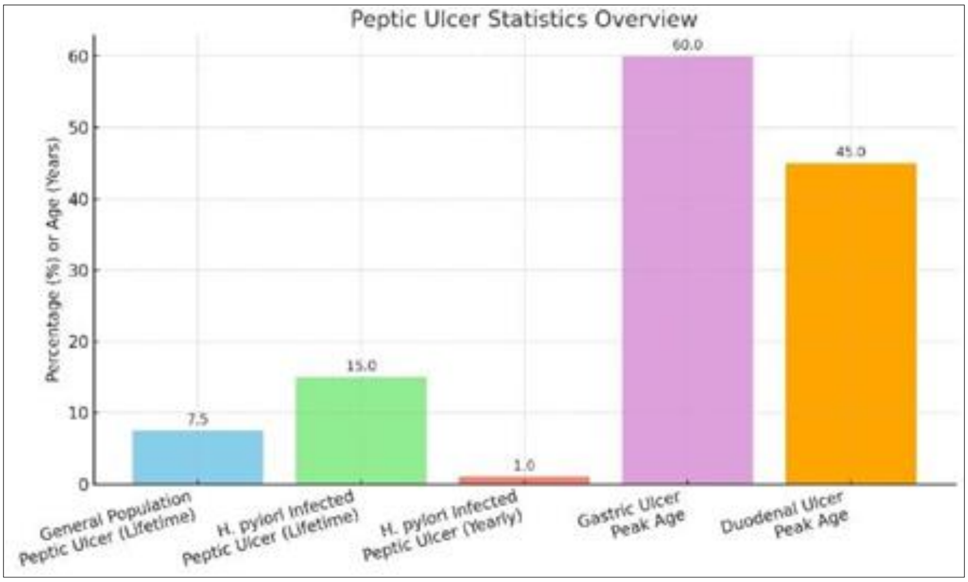

Fig 3: epidemiology of PUD

Etiology
Although there are many causes of PUD, the majority of cases are caused by *Helicobacter pylori*-associated PUD and NSAID-associated PUD. [1] Causes of Peptic Ulcer Disease

Common causes
- H. pylori infection
- NSAIDs
- Rare causes:
- Radiation therapy
- Crohn disease
- Chemotherapy

***Helicobacter pylori*-Associated PUD:** The gram-negative bacillus H is found in the gastric epithelial cells pylorus between 70% and 90% of stomach ulcers and 90% of duodenal ulcers are caused by this bacterium. Infection with H pylori is usually acquired as a child and is more prevalent in those with lower socioeconomic status.

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.

NSAID-associated PUD
Nonsteroidal anti-inflammatory drug use is the second most common cause of PUD after H. pylori infection. The secretion of prostaglandin normally protects the gastric mucosa. Nonsteroidal anti-inflammatory drug use is the second most common cause of PUD after H. pylori infection. The secretion of prostaglandin normally protects the gastric mucosa. NSAIDs inhibit the synthesis of prostaglandins by blocking the COX-1 enzyme. This lowers the production of gastric mucus and bicarbonate and decreases blood flow to the mucosa [5, 6].

Pathophysiology

The muscularis mucosa, which is necessary to preserve the integrity of the stomach mucosa, is disrupted in peptic ulcers.

The stomach mucosa's defensive mechanism:

The mucosal barrier is composed of the following three protective components [7, 8].

Layer of fluid-repelling epithelial cell lining that is connected by tight connections. The entire gastric mucosal surface is covered in a protective gel-like layer of mucus generated by surface epithelial cells and foveolar cells. Acid-neutralizing layer of bicarbonate ions released by surface epithelial cells [9, 10].

Colonization

Surface epithelial cells degenerate when virulent CagA-positive *Helicobacter pylori* strains colonize an area. Surface epithelial cells exfoliate more as a result of this deterioration. The migration of immature cells to the surface and foveolae is caused by compensatory cell growth mediated by exfoliation.

Immature cells result in decreased synthesis of mucin and bicarbonate, which may jeopardize the integrity of the mucosal barrier.

The aforementioned variables cause complement to be triggered through the alternative pathway, and mast cells release chemical mediators. Triggered polymorphs may cause focal ischemia damage to the surface epithelium and micro vascular disruptions. Disease caused by *Helicobacter pylori* alters the mucosa's resilience to acid damage by down regulating the expression of E-cadherin in gastric epithelial cells. Mucosal injury from decreased mucus production, chemical mediator release, and down-regulation of E-cadherin results in ulcer development. Reaction of the immune system [11-12].

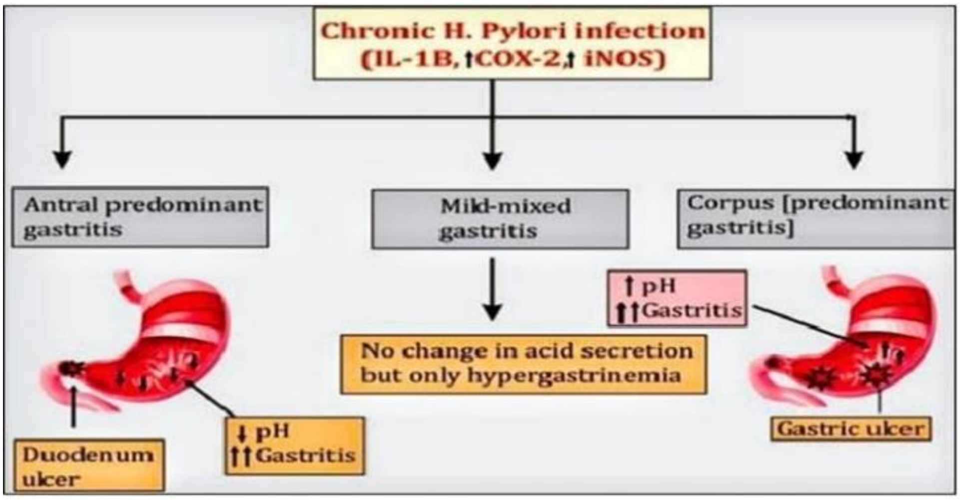

Fig 4: Depicting colonization of H. pylori in different regions of the stomach leading to various gastroduodenal diseases

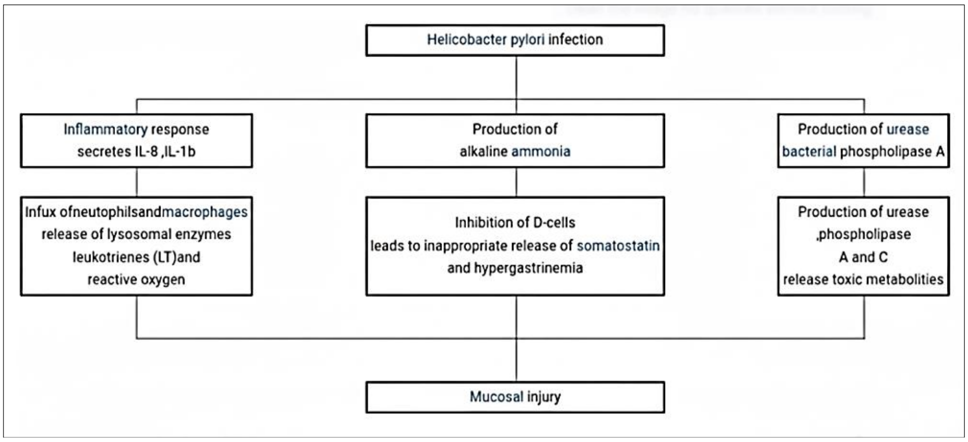

Fig 5: The following elements are in charge of *Helicobacter pylori*'s immune response: [13, 14].

Table 1: Category drug adverse effects

Category	drug	Adverse effects
Proton pump inhibitors	Omeprazole, lansoprazole, esomeprazole, pantoprazole	Abdominal pain, diarrhoea, constipation, flatulence, osteoporosis
H2 receptor blockers	Cimetidine, famotidine, rantidine	Cardiovascular events, thrombocytopenia anxiety, depression
Potassium competitive acid blockers	vonoprazan	Nasopharyngitis, eczema, diarrhoea, upper respiratory tract, inflammation
Antacids	Aluminium hydroxide, magnesium hydroxide	Hypophosphatemia, electrolyte imbalance, cramping, chalky taste

Classification of peptic ulcers

Table: classification of PUD

Mechanism of action

Omeprazole

A proton pump inhibitor omeprazole inhibits the production of gastric acid by selectively inhibiting the H/K ATPase enzyme system found in the stomach parietal cells.

1. **Prodrug Activation:** Omeprazole is a prodrug that is activated in the parietal cell canaliculi's acidic environment. It transforms into an active sulphonamide form at acidic pH values.
2. **Binding to Proton Pump:** Sulfhydryl groups on the H^+/K^+-ATPase enzyme are bound by the active sulphonamide in an irreversible covalent bond.
3. **Inhibition of Acid release:** Regardless of the stimulus (food, gastrin, histamine, etc.), this efficiently prevents the release of hydrogen ions into the stomach lumen by inhibiting the last stage of acid formation.
4. **Duration of Action:** Since the inhibition is permanent, it usually takes 24 to 48hours for the synthesis of new proton pumps before acid secretion to restart.

MOA of Omeprazole

Omeprazole (Oral Intake)

↓

Absorbed in the Small Intestine → Enters Systemic Circulation

↓

Reaches Parietal Cells of Stomach

↓

Activated in Acidic Canaliculi (Low pH)

↓

Converted to Active Sulphonamide Form

↓

Irreversibly Binds to H^+/K^+-ATPase (Proton Pump)

↓

Blocks Final Step of Gastric Acid Secretion

↓

↓ Acid Production in Stomach

↓

Effect Lasts Until New Proton Pumps Are Synthesized (24-48 hrs) [15, 16]

MOA of misoprostol

Misoprostol (Prodrug - Oral or Vaginal Administration)

↓

Converted to Active Form: Misoprostol Acid (in liver)

↓

Binds to Prostaglandin E1 (PGE1) Receptors on Gastric Parietal & Epithelial Cells

↓

Inhibits Gastric Acid Secretion

↓

↓ cAMP in Parietal Cells → ↓ H^+/K^+-ATPase Activity

↓

↓ Secretion of Gastric Acid

↓

Stimulates Mucus & Bicarbonate Secretion from Mucosal Cells

↓

Enhances Gastric Mucosal Defence & Promotes Healing [17]

Diagnosis

The diagnosis of peptic ulcer is based on two main factors. In order to identify the precise cause of the ulcer, the first step is to determine whether the symptoms that have been referred are unrelated to functional dyspepsia. The most precise diagnostic for identifying peptic ulcer disease (PUD) is upper GI endoscopy, which also provides information on the location and size of the ulcer. It makes mucosal biopsies possible for therapy and differential diagnosis, particularly in cases with bleeding ulcers [18].

Radiology

In routine diagnosis, endoscopy has largely superseded barium gastroduodenal tests, which are now rarely utilized. They might still be helpful, nevertheless, in cases where esophageal constriction makes access impossible or in individuals who refuse endoscopy. Malignant ulcers frequently have uneven folds, unequal or non-existent contrast filling, and a bigger appearance.

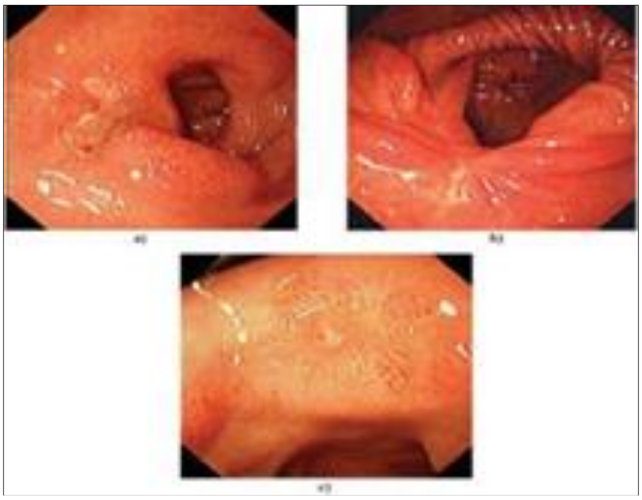

Fig 6: Diagnosis of PUD

Laboratory investigation

1. Esophagogastroduodenoscopy (EGD)
2. Barium swallow
3. Serologic testing Urea breath test
4. Endoscopic biopsy
5. Computerized tomography
6. Stool antigen test
7. *Helicobacter pylori* testing
8. Urine based ELISA and rapid urine test [6]

Conclusion

Peptic ulcer disease is less common now than it was a century ago in high-income countries. It is usually associated with prolonged use of NSAIDs or H. pylori infection. Treatment for an Triple or quadruple therapy is typically used to treat pylori infections. This comprises bismuth sulphate, two antibiotics, and PPI. PPI can be taken either in addition to or instead of NSAIDs while taking them. Complications from peptic ulcer illness are quite prevalent and fatal; in order to save the patient's life, immediate medical or surgical intervention is necessa

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