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Ulcer and Antiulcer Drugs: An Academic Review

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ABSTRACT :

Peptic ulcer disease (PUD) is a chronic condition characterized by mucosal erosions in the stomach or duodenum resulting from an imbalance between aggressive factors (acid, pepsin, Helicobacter pylori infection) and defensive mechanisms (mucus, bicarbonate, prostaglandins). This review focuses on the pathophysiology, classification, causes, and therapeutic management of ulcers, emphasizing the pharmacological actions of antiulcer drugs. Recent advances in drug therapy and alternative approaches, including herbal and nutraceutical agents, are also discussed. Ulcer is a common gastrointestinal disorder which is seen among many people. It is basically an inflamed break in the skin or the mucus membrane lining the alimentary tract. Ulceration occurs when there is a disturbance of the normal equilibrium caused by either enhanced aggression or diminished mucosal resistance. It may be due to the regular usage of drugs, irregular food habits, stress, and so forth. Peptic ulcers are a broad term that includes ulcers of digestive tract in the stomach or the duodenum. The formation of peptic ulcers depends on the presence of acid and peptic activity in gastric juice plus a breakdown in mucosal defenses.

1. Introduction

An ulcer is a localized defect in the mucosal lining of the gastrointestinal tract, commonly affecting the stomach (gastric ulcer) or duodenum (duodenal ulcer). It results from excessive secretion of gastric acid and pepsin combined with mucosal barrier disruption. Peptic ulcer disease remains a global health concern, with a significant burden in developing countries due to *H. pylori* infection, NSAID use, and lifestyle factors (Sonnenberg & Everhart, 1997).

The discovery of *Helicobacter pylori* revolutionized ulcer management by highlighting the bacterial contribution to mucosal injury, leading to the introduction of antimicrobial and acid-suppressing therapies (Marshall & Warren, 1984).

Ulcers are an open sore of the skin or mucus membrane characterized by sloughing of inflamed dead tissue . Ulcers are lesions on the surface of the skin or a mucous membrane characterized by a superficial loss of tissue. Ulcers are most common on the skin of the lower extremities and in the gastrointestinal tract, although they may be encountered at almost any site. There are many types of ulcer such as mouth ulcer, esophagus ulcer, peptic ulcer, and genital ulcer. Of these peptic ulcer is seen among many people. The peptic ulcers are erosion of lining of stomach or the duodenum

2. Etiology and Pathophysiology:-

The etiology of peptic ulcer disease includes multiple interacting factors:

- **Helicobacter pylori infection:** damages gastric mucosa and increases acid secretion.
- **Non-Steroidal Anti-Inflammatory Drugs (NSAIDs):** inhibit prostaglandin synthesis, reducing mucosal defense.
- **Hyperacidity:** due to excessive gastrin or vagal stimulation.
- **Lifestyle factors:** smoking, alcohol, and stress.
- **Genetic predisposition** and **Zollinger–Ellison syndrome** also contribute.

The ulcer results from an **imbalance between aggressive (acid, pepsin, bile salts, bacteria)** and **defensive (mucus, bicarbonate, prostaglandins, mucosal blood flow)** factors (Konturek et al., 2011).

3. Types of Ulcers:-

1. **Gastric ulcer:** occurs in the stomach, often linked to NSAIDs.
2. **Duodenal ulcer:** common in younger individuals, usually *H. pylori*-related.
3. **Stress ulcer:** caused by severe physiological stress (e.g., burns, trauma).
4. **Esophageal ulcer:** due to gastroesophageal reflux disease (GERD).

1.Gastric ulcer : A peptic ulcer is a sore on the lining of your stomach or duodenum.

Do peptic ulcers have other names?

Peptic ulcers are sometimes called stomach ulcers, duodenal ulcers, or peptic ulcer disease.

How common are peptic ulcers?

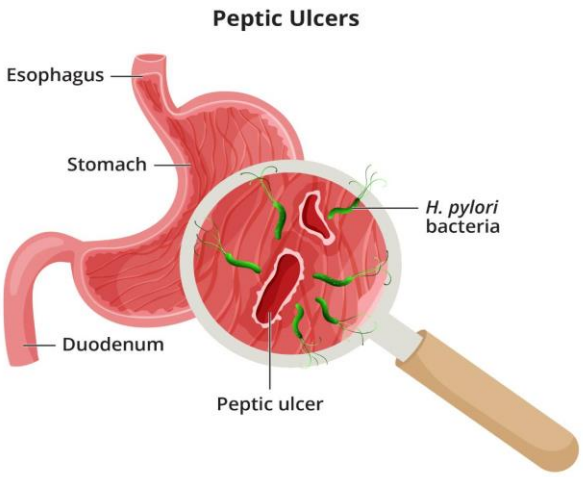
Researchers estimate about 1% to 6% of people in the United States have peptic ulcers.[1]

Who is more likely to develop peptic ulcers?

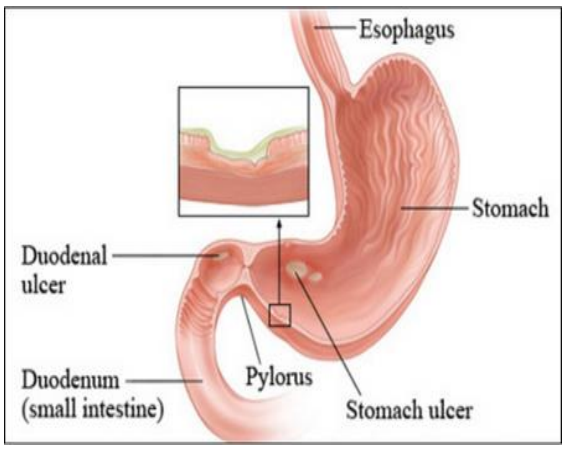
People are more likely to develop peptic ulcers if they are

- infected with the bacteria *Helicobacter pylori (H. pylori)*
- taking nonsteroidal anti-inflammatory drugs (NSAIDs), such as aspirin, ibuprofen, and naproxen

H. pylori infection and taking NSAIDs are the two most common causes of peptic ulcers.



2.Duodenal ulcer: A duodenal ulcer is a sore that forms in the lining of the duodenum. Your duodenum is the first part of your small intestine, just beyond the stomach. These ulcers often cause pain in the upper tummy, just below the breastbone.
You can also get an ulcer in your stomach. Stomach ulcers and duodenal ulcers are both types of peptic ulcers. If you have either of these, you have what's called 'peptic ulcer disease'. Once treated, they usually get better within a few weeks.



4. Clinical Manifestations:-

Common symptoms include:

- Epigastric pain (burning or gnawing)
- Nausea and vomiting
- Loss of appetite
- Gastrointestinal bleeding (in severe cases)

Pain typically occurs **1–3 hours after eating** and may be relieved by food or antacids (Sung et al., 2009).

5. Diagnosis:-

Diagnosis is confirmed through:

- **Endoscopy:** direct visualization of ulcers.
- **Urea breath test / stool antigen test:** for *H. pylori*.
- **Barium meal X-ray.**
- **Histopathological biopsy.**

6. Antiulcer Drugs and Mechanisms:-

6.1. Proton Pump Inhibitors (PPIs)

- **Examples:** Omeprazole, Lansoprazole, Pantoprazole.
- **Mechanism:** Irreversibly inhibit H^+/K^+-ATPase enzyme in parietal cells, reducing gastric acid secretion.
- **Clinical Use:** Effective in *H. pylori*-associated and NSAID-induced ulcers.
  (Shin & Sachs, 2008).

6.2. H_2-Receptor Antagonists:-

- **Examples:** Ranitidine, Famotidine, Cimetidine.
- **Mechanism:** Block histamine H_2 receptors, reducing acid secretion.
- **Note:** Largely replaced by PPIs but still useful for mild cases.
  (Wallace & Sharkey, 2011).

6.3. Antacids:-

- **Examples:** Magnesium hydroxide, Aluminum hydroxide, Calcium carbonate.
- **Mechanism:** Neutralize gastric acid, providing symptomatic relief.
- **Limitation:** Do not promote healing or address underlying cause.

6.4. Mucosal Protective Agents:-

- **Sucralfate:** Forms a protective barrier over ulcers.
- **Bismuth compounds:** Have antibacterial action against *H. pylori*.
- **Prostaglandin analogs (Misoprostol):** Increase mucus and bicarbonate secretion.

6.5. Antimicrobial Therapy:-

- Used for *H. pylori* eradication in combination with PPIs.
- **Triple therapy:** PPI + Clarithromycin + Amoxicillin or Metronidazole.
  (Malfertheiner et al., 2012).

6.6. Anticholinergics:-

- **Examples:** Pirenzepine, Propantheline.
- **Mechanism:** Block vagal stimulation, reducing gastric secretion.
- **Use:** Limited due to side effects (dry mouth, blurred vision).

7. Recent Advances:-

Recent therapies focus on **proton pump-independent acid suppression** (e.g., potassium-competitive acid blockers like Vonoprazan) and **probiotic adjunct therapy** for *H. pylori* eradication.
Natural compounds such as **flavonoids, curcumin, and glycyrrhizin** have shown promising gastroprotective effects (Borrelli & Izzo, 2000).

8. Alternative and Herbal Antiulcer Agents:-

Many herbal extracts (e.g., *Aloe vera*, *Glycyrrhiza glabra*, *Ocimum sanctum*, *Terminalia arjuna*) have demonstrated antiulcer properties by reducing acid secretion and enhancing mucosal defense. Nutraceutical formulations are being investigated for long-term management and minimal side effects (Alqasoumi et al., 2011).

9. Conclusion:-

Peptic ulcer disease remains a major global health concern despite significant advancements in pharmacotherapy. The combination of acid-suppressing agents, antimicrobial therapy, and lifestyle modification remains the cornerstone of treatment. Continued research into novel drug targets, herbal therapies, and probiotic interventions offers potential for safer and more effective management.

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