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PEPTIC ULCER DISEASE AND ITS TREATMENT: A COMPREHENSIVE REVIEW

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ABSTRACT

Peptic Ulcer Disease(PUD) represents a break in the mucosal lining of the stomach or duodenum, extending through the muscularis mucosa. Once considered a chronic condition primarily driven by lifestyle factors and acid hypersecretion, the understanding of PUD has been revolutionized by the discovery of Helicobacter pylori (H. pylori) and the widespread use of non-steroidal anti-inflammatory drugs (NSAIDs). This review provides a comprehensive overview of the epidemiology, pathophysiology, clinical presentation, diagnostic strategies, and contemporary treatment paradigms of PUD, emphasizing the shift from acid suppression to curative antimicrobial therapy and risk mitigation. Peptic ulcer disease (PUD) is a common gastrointestinal disorder characterized by mucosal damage of the stomach or duodenum due to the corrosive effects of gastric acid and pepsin. The major etiological factors include Helicobacter pylori infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs). Despite advances in diagnosis and therapy, PUD remains a significant cause of morbidity worldwide. This review provides a comprehensive overview of the etiology, pathophysiology, clinical manifestations, diagnosis, and current treatment strategies of peptic ulcer disease, with emphasis on pharmacological management and prevention of complications.

1. INTRODUCTION

Peptic ulcer disease,encompassing gastric and duodenal ulcers, is a common gastrointestinal disorder affecting approximately 5-10% of the global population at some point in their lives. Historically managed with antacids and later histamine-2 receptor antagonists (H2RAs), the

disease was marked by high recurrence rates. The landmark discovery of H. pylori by Barry Marshall and Robin Warren in 1982 fundamentally altered the model of PUD pathogenesis, establishing it as an infectious disease in most cases. Today, management focuses on eradicating H. pylori, discontinuing injurious agents, and promoting healing with potent acid suppression.

Peptic ulcer disease refers to a defect in the gastrointestinal mucosa that extends through the muscularis mucosae and occurs primarily in the stomach (gastric ulcer) or the proximal duodenum (duodenal ulcer). PUD has affected millions of people globally and continues to be a major public health concern. The discovery of H. pylori revolutionized the understanding and treatment of this disease, shifting management from symptom control to curative therapy.

2. Etiology and Pathophysiology

The central pathophysiology involves an imbalance between aggressive factors and defensive mucosal mechanisms.

A. Primary Etiologic Factors:

1. Helicobacter pylori Infection: Colonizes the gastric antrum in ~70% of gastric ulcers and >90% of duodenal ulcers. It induces chronic active gastritis, disrupts the mucus layer, promotes interleukin secretion, and increases gastrin release, leading to increased acid production. 2. NSAIDs and Aspirin: Inhibit cyclooxygenase-1 (COX-1), reducing cytoprotective prostaglandins (PGE2) that maintain mucosal blood flow, mucus, and bicarbonate secretion.

This impairs mucosal defense and repair, making it susceptible to acid injury.

3. Stress-Related Mucosal Disease: Seen in critically ill patients (e.g., on mechanical ventilation, coagulopathy). Splancnic hypoperfusion and ischemia lead to mucosal injury.

B. Other Contributing Factors: Includes hypersecretory states (Zollinger-Ellison syndrome), smoking (delays healing), genetic predisposition, and severe physiological stress.

3. Clinical Presentation

- Epigastric Pain: The classic symptom, often described as burning or gnawing. Duodenal ulcer pain may improve with food and recur 2-3 hours postprandially or at night. Gastric ulcer pain may be exacerbated by food.
- Dyspepsia: Bloating, fullness, early satiety.

- Complications: May be the presenting feature:
- Bleeding: Hematemesis, melena, or iron-deficiency anemia. · Perforation: Sudden, severe abdominal pain with peritonitis.
- Penetration: Into adjacent organs (e.g., pancreas).
- Gastric Outlet Obstruction: From recurrent scarring, presenting with vomiting and early satiety.

4. Diagnosis

A structured approach is required:

1. Clinical History: Focus on NSAID/aspirin use, prior ulcer history, and alarm symptoms (weight loss, vomiting, bleeding).
2. Endoscopy (Esophagogastroduodenoscopy - EGD): The gold standard. Allows direct visualization, biopsy, and therapeutic intervention (e.g., hemostasis). Biopsies are taken for:

- H. pylori detection (rapid urease test, histology, culture).
- Exclusion of malignancy (for gastric ulcers, mandatory).

3. Non-Invasive Testing for H. pylori: Used in uncomplicated dyspepsia without alarm features.

- Urea Breath Test: Highly accurate for active infection and confirmation of eradication.
- Stool Antigen Test: Excellent sensitivity and specificity.
- Serology: Indicates past exposure but not active infection; useful in settings of low prevalence or prior investigation.

5. Treatment Strategies

Modern treatment is etiology-driven and focuses on cure.

A. Eradication of Helicobacter pylori:

· First-Line Therapy: Standard triple therapy (Proton Pump Inhibitor (PPI) + Clarithromycin + Amoxicillin/Metronidazole) faces high resistance. Preferred regimens now include:

· Quadruple Therapy: PPI + Bismuth + Metronidazole + Tetracycline for 10-14 days.

· Concomitant Therapy: PPI + Clarithromycin + Amoxicillin + Metronidazole for 10-14 days.

· Sequential Therapy: Less commonly used now.

· Confirmation of Eradication: Recommended using a urea breath test or stool antigen test at least 4 weeks after completion of therapy, especially after complicated ulcers.

B. NSAID-Induced Ulcer Management:

1. Discontinue NSAID if possible.
2. Initiate PPI Therapy: High-dose PPIs (e.g., omeprazole 40mg daily) are the most effective for healing.
3. Ulcer Prevention in Required NSAID Users:

- Use the lowest effective dose of NSAID.
- Co-therapy with a PPI is superior to H2RAs or misoprostol (which has side effects). · Consider switching to a selective COX-2 inhibitor (celecoxib), though cardiovascular risks must be assessed.
- Test for and eradicate H. pylori before starting long-term NSAIDs.

C. Pharmacologic Agents:

- Proton Pump Inhibitors (PPIs): (e.g., Omeprazole, Pantoprazole, Esomeprazole). The cornerstone of therapy, providing profound acid suppression, promoting rapid healing, and reducing complications.
- Histamine-2 Receptor Antagonists (H2RAs): (e.g., Famotidine). Effective for healing but inferior to PPIs; useful for maintenance in select cases.
- Mucosal Protectants: Sucralfate (binds to ulcer base) and Antacids (symptomatic relief).
- Prostaglandin Analogue: Misoprostol, used for NSAID ulcer prophylaxis.

D. Treatment of Complications:

- Bleeding: Endoscopic therapy (injection, thermal coagulation, clipping) combined with high-dose IV PPI infusion.
- Perforation: Surgical repair or conservative management in selected cases.
- Obstruction: Endoscopic balloon dilation or surgery.

6. Prevention and Long-Term Management

- H. pylori Eradication: Effectively prevents recurrence in >90% of cases, changing the natural history of the disease.

- Judicious Use of NSAIDs: Prescribe only when necessary, at the lowest dose and shortest duration.
- PPI Maintenance Therapy: Reserved for high-risk patients who cannot discontinue NSAIDs/aspirin, those with recurrent idiopathic ulcers, or a history of complicated PUD.

7. CONCLUSION

The management of peptic ulcer disease has evolved from long-term symptomatic acid suppression to a strategy focused on identifying and removing the underlying cause.The dual pillars of modern treatment are the eradication of H. pylori infection and the careful management of NSAID use. With accurate diagnosis, targeted therapy, and confirmation of cure, PUD is now a largely curable condition, and its complications are preventable. Future challenges include addressing antibiotic resistance in H. pylori regimens and optimizing gastroprotective strategies for patients requiring antiplatelet and anticoagulant therapy

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