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A comprehensive analysis on “peptic ulcer: causes and treatment"

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ABSTRACT :

Peptic ulcer disease (PUD) is a common gastrointestinal disorder characterized by mucosal erosion in stomach or upper duodenum due to imbalance in aggressive factors like gastric acid, Pepsin, Helicobacter pylori, and NSAID use and the protective mechanism of the gastric mucosa. This review explores the etiology, pathophysiology, clinical presentation, diagnostic methods, and evidence-based treatment options for peptic ulcers. H. pylori infection and NSAID use remain the predominant causes, with lifestyle factors such as smoking and alcohol further contributing to disease progression. Diagnosis relies heavily on endoscopic evaluation and testing for H. pylori. Treatment focuses on acid suppression using proton pump inhibitors and eradication of H. pylori through antibiotic regimens, with triple and quadruple therapy protocols commonly employed. NSAID-related ulcers require drug discontinuation or gastroprotective co-therapy.

Keywords: Peptic ulcer, acid suppression, NSAID, mucosa,

Introduction

A peptic ulcer is a mucosal erosion equal to or greater than 0.5 cm in the gastrointestinal tract, typically found in the stomach (gastric ulcer) or the upper part of the small intestine (duodenal ulcer). These ulcers arise due to the corrosive effects of gastric acid and pepsin, often exacerbated by infection with Helicobacter pylori or the prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs) [1].
Peptic ulcer affect millions globally and is a significant cause of death. With advancements in diagnostics and therapy, outcomes have improved but recurrence and complications remain still concerns. This article delves into the causes, symptoms, diagnosis, and treatment of peptic ulcers, supported by recent evidence and guidelines.

Etiology and Risk Factors

1. Helicobacter pylori Infection
H. pylori infection is the most common cause of peptic ulcers. This gram-negative bacterium colonizes the gastric mucosa, leading to inflammation, mucosal damage, and ulceration. It affects over 50% of the global population, although only a subset develops ulcers [2].

2. NSAID Use
NSAIDs such as aspirin, ibuprofen, and naproxen inhibit cyclooxygenase (COX) enzymes, reducing prostaglandin synthesis. This leads to diminished gastric mucosal protection and increased susceptibility to acid-related injury [3].

3. Other Risk Factors
Other contributing factors include smoking, excessive alcohol intake, corticosteroid use, stress (especially in critically ill patients), Zollinger-Ellison syndrome (gastrinoma producing excessive acid), and a family history of peptic ulcers [4].

Pathophysiology

Peptic ulcers result from an imbalance between aggressive factors (acid, pepsin, H. pylori, NSAIDs) and defensive mechanisms (mucus, bicarbonate, mucosal blood flow, prostaglandins). H. pylori disrupts the mucosal barrier and elicits an inflammatory response, while NSAIDs directly damage the epithelium and reduce protective prostaglandins [5].

Clinical Presentation

Common symptoms of peptic ulcer include:

Epigastric pain: Described as burning or gnawing, often relieved by food (duodenal) or worsened postprandially (gastric) [6].
Nausea and bloating
Early satiety
Weight loss
Alarm symptoms (which may suggest complications or malignancy) include:
Gastrointestinal bleeding (hematemesis or melena)
Anemia
Vomiting
Unintended weight loss
Dysphagia

Diagnosis

1. Endoscopy
Upper gastrointestinal endoscopy is the gold standard for diagnosing peptic ulcers. It allows for direct visualization, biopsy, and exclusion of malignancy, especially in gastric ulcers [7].

2. Testing for H. pylori
Non-invasive: Urea breath test, stool antigen test, and serology.
Invasive (via endoscopy): Rapid urease test, histology, and culture.
The urea breath test is highly sensitive and specific and is preferred for initial diagnosis and confirmation of eradication [8].

Treatment

The primary goals of treatment are:
Relieving symptoms
Promoting ulcer healing
Preventing complications
Eradicating H. pylori (if present)

Preventing recurrence

1. Eradication of H. pylori
Triple Therapy (for 14 days):
Proton Pump Inhibitor (PPI) twice daily
Clarithromycin 500 mg twice daily
Amoxicillin 1 g twice daily or Metronidazole 500 mg twice daily

This regimen has a success rate of 70–85% but is limited by rising clarithromycin resistance [9].

Bismuth-based Quadruple Therapy:

PPI twice daily
Bismuth subsalicylate 525 mg four times daily
Tetracycline 500 mg four times daily
Metronidazole 500 mg three to four times daily
Preferred in areas with high antibiotic resistance or treatment failure [10].

2. Acid Suppression
PPIs (e.g., omeprazole, lansoprazole) are the mainstay for acid suppression. They inhibit the H+/K+ ATPase in gastric parietal cells, reducing acid secretion and promoting ulcer healing [11].
H2-receptor antagonists (e.g., ranitidine) are less effective but still used in some cases.
Antacids and sucralfate: These provide symptomatic relief and mucosal protection but are adjunctive.

3. NSAID-Associated Ulcers

For patients who must continue NSAIDs:

Co-prescription of PPIs

Use of selective COX-2 inhibitors (e.g., celecoxib) which are less ulcerogenic [12]

Discontinuation of NSAIDs if possible.

Conclusion

Peptic ulcer disease, although it is common ans severe, butit is highly manageable with appropriate therapy. Eradication of H. pylori, rational use of NSAIDs, and acid suppression therapy remain the cornerstones of treatment. With the growing issue of antibiotic resistance, personalized therapy and strict adherence to treatment protocols are essential for long-term success and prevention of complications.

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