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Review Article

PEPTIC ULCER DISEASE & IT'S TREATMENT: A REVIEW

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Peptic ulcer disease (PUD) is one of the most common gastrointestinal disorders worldwide, characterized by a mucosal break in the gastric or duodenal lining due to an imbalance between aggressive (acid, pepsin, Helicobacter pylori, NSAIDs) and defensive (mucus-bicarbonate barrier, prostaglandins, blood flow) factors. The management of PUD has evolved from symptomatic relief to targeted eradication of H. pylori and prevention of NSAID-related damage, with proton pump inhibitors (PPIs) forming the cornerstone of modern anti-secretory therapy. This review summarizes the current understanding of pathophysiology, risk factors, diagnostic approaches, ((and evidence-based treatment strategies for PUD, with emphasis on H. pylori eradication regimens, role of NSAIDs, and emerging therapeutic options

Keywords: pathophysiology, risk factors, diagnostic approaches, ((and evidence-based treatment strategies for PUD, with emphasis on H. pylori eradication regimens, role of NSAIDs.

INTRODUCTION

Peptic ulcer disease refers to the presence of open sores or erosions in the mucosa of the stomach (gastric ulcer) or proximal duodenum (duodenal ulcer), which may extend into the submucosa. It affects up to 10% of the global population at some point in life and is associated with complications such as bleeding, perforation, and gastric outlet obstruction. The paradigm shift in the 1980s, with the discovery of Helicobacter pylori and its causal role in the majority of duodenal and many gastric ulcers, revolutionized both diagnosis and treatment. Alongside infection, chronic use of non-steroidal anti-inflammatory drugs (NSAIDs), lifestyle factors (smoking, alcohol), and stress contribute significantly to ulcerogenesis.[1]

Literature Review

Epidemiology and risk factors

Globally, duodenal ulcers are more common than gastric ulcers, with a higher prevalence in older adults and those with socioeconomic risk factors. Major risk factors include chronic H. pylori infection, regular NSAID or low-dose aspirin (LDA) intake, smoking, alcohol abuse, and a personal history of ulcer disease. The incidence of PUD has declined in many high-income countries due to improved sanitation, reduced smoking, and better H. pylori eradication, but it remains a significant health burden in developing regions. [2]

Pathophysiology

The pathogenesis of PUD is classically explained by an imbalance between aggressive factors (gastric acid, pepsin, H. pylori virulence factors such as CagA and VacA, NSAIDs) and

mucosal defence mechanisms (mucus-bicarbonate barrier, prostaglandins, mucosal blood flow, and epithelial restitution). H. pylori colonizes the gastric antrum, induces chronic gastritis, and disrupts acid-regulatory feedback, leading to increased acid secretion in duodenal ulcer patients and gastric atrophy in some gastric ulcer cases. NSAIDs inhibit cyclooxygenase-1 (COX-1), reducing mucosal prostaglandins and weakening protective defences, thereby facilitating ulcer formation.[3]

Diagnostic approaches

Diagnosis typically combines clinical features (epigastric pain, dyspepsia, nocturnal or meal-related pain patterns) with endoscopic evaluation, which remains the gold standard for confirming ulcer presence, location, size, and complications. Non-invasive tests for H. pylori include urea breath test, stool antigen test, and serology, whereas histology and rapid urease test are performed on endoscopic biopsy specimens. Imaging and laboratory tests are used to detect complications such as bleeding (haemoglobin, endoscopy) or perforation (X-ray/CT for free air)[4]

Current treatment strategies

Modern management of PUD focuses on three pillars: (1) eradication of H. pylori when present, (2) optimal acid suppression, and (3) avoidance or modification of NSAID use. First-line H. pylori eradication commonly involves triple or quadruple therapy combining a PPI with two or three antibiotics (e.g., clarithromycin-based triple therapy or bismuth-containing quadruple regimen), with 7–14 days duration and eradication rates of approximately 70–90% in many settings. In areas with high clarithromycin resistance, bismuth-based quadruple, concomitant, or vonoprazan-based regimens are recommended as first-line. For NSAID-induced ulcers, discontinuation of the offending drug is preferred; if NSAIDs must continue, PPIs or newer acid suppressants such as vonoprazan are used prophylactically.[5]

Methodology

This review was conducted by systematically analyzing recent peer-reviewed literature on peptic ulcer disease published between 2019 and 2026. Relevant articles were identified using databases such as PubMed, PMC, and journal websites (e.g., StatPearls, JAPTR, and specialty gastroenterology journals) with MeSH terms and keywords including “peptic ulcer disease,” “Helicobacter pylori,” “NSAID-induced ulcer,” and “peptic ulcer treatment guidelines.” Only studies providing epidemiological data, mechanistic insights, and clinical guidelines were included, while very old or non-English articles with limited relevance were excluded.Data on treatment regimens, eradication rates, and outcomes were extracted and synthesized into descriptive summary sections.[6]

Results and Discussion

Clinical features and outcomes

Peptic ulcer disease typically presents with epigastric burning or gnawing pain, often relieved or worsened by meals, and may be associated with nausea, bloating, or weight loss. Many ulcers are asymptomatic until complications arise, such as hematemesis, melena, or acute abdominal pain suggesting perforation. Most uncomplicated ulcers heal within 4–8 weeks with appropriate medical therapy, though larger or complicated ulcers may require longer treatment and endoscopic or surgical intervention.[7]

Role of Helicobacter pylori eradication

Eradication of H. pylori significantly reduces ulcer recurrence and complications compared with acid-suppressive therapy alone. First-line regimens (PPI plus clarithromycin plus amoxicillin or metronidazole for 7–14 days) achieve eradication in about 70–85% of patients, but resistance-driven treatment failure has led to increasing use of bismuth-based quadruple and levofloxacin-based salvage regimens. Vonoprazan-based therapies, especially in Asian populations, show higher eradication rates and better acid control than conventional PPI-based regimens.[8]

NSAID- and drug-induced ulcers

NSAIDs are a major cause of gastric and duodenal ulcers, particularly in elderly patients and those on long-term low-dose aspirin for cardiovascular protection. Discontinuation or dose reduction of NSAIDs, when feasible, leads to healing in the majority of cases. When NSAID use is mandatory, PPIs or vonoprazan are recommended for prophylaxis, often combined with selective COX-2 inhibitors such as celecoxib to further reduce ulcer risk.[9]

Acid-suppressive therapy: PPIs vs. H2-blockers

Proton pump inhibitors (PPIs) are superior to histamine H2-receptor antagonists (H2-RAs) in accelerating ulcer healing, controlling symptoms, and preventing re-bleeding in haemorrhagic PUD.PPIs irreversibly inhibit H^+/K^+-ATPase in parietal cells, providing prolonged acid suppression and promoting mucosal repair. Oral and intravenous PPIs have comparable efficacy in most patients, although high-dose intravenous PPIs are preferred in acute bleeding until endoscopic therapy is performed.[10]

Emerging trends and future directions

Recent research emphasizes molecular mechanisms (genetic and epigenetic factors), gut microbiome interactions, and oxidative stress in ulcer pathogenesis.Experimental models using ethanol, NSAIDs, or H. pylori-induced ulcers in rodents help elucidate signaling pathways and evaluate novel therapeutics.Emerging approaches include phytochemicals (flavonoids, polyphenols), probiotics, and mucosal-protective agents that

may complement conventional therapy and reduce antibiotic dependence.(11-15)

CONCLUSION

Peptic ulcer disease remains an important gastrointestinal condition whose management has improved dramatically with the advent of H. pylori detection and eradication, along with more effective acid-suppressive drugs. A rational therapeutic approach includes confirmation of H. pylori status, tailored eradication regimens based on local resistance patterns, appropriate PPI or vonoprazan use, and avoidance or careful management of NSAIDs. Future research should focus on personalized strategies, microbiome-based interventions, and novel agents that enhance mucosal defence while minimizing drug-related adverse effects.

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Conflict of Interest

The authors declare that they have no conflict of interest