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ETIOLOGY, PATHOGENESIS, DIAGNOSIS, AND PRINCIPLES OF
TREATMENT OF GASTRIC AND DUODENAL PEPTIC ULCER DISEASE

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Abstract: Peptic ulcer disease of the stomach and duodenum is a common gastrointestinal disorder characterized by a mucosal defect extending through the muscularis mucosae. Its development is associated primarily with *Helicobacter pylori* infection, nonsteroidal anti-inflammatory drug use, excessive gastric acid and pepsin activity, and impairment of mucosal defense mechanisms. This article examines the major etiological factors, key pathogenetic mechanisms, diagnostic approaches, and modern principles of treatment of gastric and duodenal peptic ulcers. Particular attention is given to the role of *H. pylori* infection, disruption of the gastric mucosal barrier, acid-peptic injury, and the effects of nonsteroidal anti-inflammatory drugs. The diagnostic value of upper gastrointestinal endoscopy, *H. pylori* testing, and laboratory investigations is discussed. Modern treatment principles include eradication therapy for *H. pylori*, suppression of gastric acid secretion, elimination of ulcerogenic factors, and prevention of recurrence and complications. Early diagnosis and individualized treatment are essential for reducing the risk of gastrointestinal bleeding, perforation, penetration, and other serious complications.

Keywords: peptic ulcer disease, gastric ulcer, duodenal ulcer, *Helicobacter pylori*, pathogenesis, endoscopy, gastric acid, proton pump inhibitors, eradication therapy, gastrointestinal complications.

INTRODUCTION

Peptic ulcer disease (PUD) of the stomach and duodenum remains an important gastrointestinal disorder because of its potential to cause bleeding, perforation, penetration, and recurrence. The disease develops when aggressive factors, particularly gastric acid and pepsin, overcome the protective mechanisms of the gastroduodenal mucosa. The two major etiological factors are *Helicobacter pylori* infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs). *H. pylori* contributes to mucosal inflammation and disruption of normal protective processes, whereas NSAIDs impair prostaglandin-dependent mucosal protection and increase susceptibility to acid-mediated injury. Clinical manifestations vary according to ulcer localization and severity and may include epigastric pain, dyspeptic symptoms, nausea, vomiting, or signs of gastrointestinal bleeding. Therefore, accurate identification of the underlying cause, timely diagnosis, and selection of appropriate therapy are essential components of modern management.

MATERIALS AND METHODS

The study was conducted through a structured analysis of scientific literature concerning the etiology, pathogenesis, diagnosis, and treatment of gastric and duodenal peptic ulcer



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disease. Particular attention was given to the pathogenic role of *Helicobacter pylori*, NSAID-associated mucosal injury, gastric acid secretion, and impairment of mucosal defense mechanisms. Diagnostic approaches were reviewed according to their clinical significance, including upper gastrointestinal endoscopy, *H. pylori* detection methods, and relevant laboratory investigations. Therapeutic approaches were analyzed with consideration of the underlying etiological factor, ulcer localization, disease severity, and the presence or absence of complications. The comparative assessment focused on eradication therapy for *H. pylori*, acid-suppressive treatment, elimination of ulcerogenic factors, and strategies for preventing recurrence. The obtained information was systematized to identify the relationship between etiological factors, pathogenetic mechanisms, diagnostic findings, and therapeutic decisions in contemporary clinical management.

RESULTS

The analysis showed that peptic ulcer disease develops through a multifactorial interaction between mucosal damaging factors and impaired protective mechanisms. *Helicobacter pylori* infection was identified as a major etiological factor because persistent colonization promotes chronic gastric inflammation and weakens mucosal resistance. NSAID use represents another important cause, mainly through inhibition of prostaglandin synthesis, which reduces mucus and bicarbonate secretion and compromises mucosal blood flow. Increased acid-peptic activity further contributes to ulcer formation, particularly when protective mechanisms are insufficient. Upper gastrointestinal endoscopy remains central to diagnosis because it directly visualizes the ulcer, determines its localization and morphology, and allows biopsy when clinically indicated. Testing for *H. pylori* is essential for identifying an underlying infectious cause. Therapeutically, successful management requires elimination of the causative factor, appropriate acid suppression, eradication of *H. pylori* when detected, and monitoring for complications such as bleeding, perforation, and penetration.

DISCUSSION

The findings indicate that successful management of peptic ulcer disease requires treatment directed at both the underlying cause and the mechanisms responsible for mucosal injury. In patients with *H. pylori* infection, eradication therapy is a fundamental component because persistent infection can maintain inflammation and increase the risk of ulcer recurrence. Acid suppression with proton pump inhibitors promotes ulcer healing by reducing gastric acid exposure and allowing restoration of the damaged mucosa. In NSAID-associated ulcers, discontinuation or appropriate modification of the offending drug is particularly important, while gastroprotective therapy may be required when continued NSAID treatment is unavoidable. Endoscopic assessment is also important for evaluating ulcer characteristics and identifying signs of bleeding or other complications. Severe bleeding may require endoscopic hemostatic intervention, whereas perforation, penetration, or uncontrolled hemorrhage can necessitate surgical management. Thus, treatment should be individualized according to etiology, clinical severity, ulcer characteristics, and complications.



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An important aspect of peptic ulcer management is the prevention of recurrence after the initial ulcer has healed. Clinical improvement alone does not always indicate complete elimination of the underlying cause, particularly in patients with persistent *Helicobacter pylori* infection or continued exposure to ulcerogenic medications. Therefore, follow-up should focus not only on symptom resolution but also on confirmation of successful *H. pylori* eradication when infection has been identified. The increasing problem of antimicrobial resistance also makes treatment selection more important, as previous antibiotic exposure and local resistance patterns may influence therapeutic effectiveness. In patients with recurrent or refractory ulcers, clinicians should reconsider the initial diagnosis, treatment adherence, continued NSAID use, persistent *H. pylori* infection, and less common causes of ulceration. Lifestyle-related factors, including smoking and excessive alcohol consumption, may additionally interfere with mucosal healing and should be addressed as part of preventive management. Appropriate patient education is therefore essential, particularly regarding medication adherence, avoidance of unnecessary NSAID use, recognition of alarm symptoms, and the importance of follow-up evaluation. A structured long-term approach can reduce recurrence and facilitate earlier recognition of potentially serious complications.

CONCLUSION

Peptic ulcer disease of the stomach and duodenum is a multifactorial disorder in which *Helicobacter pylori* infection, NSAID use, excessive acid-peptic activity, and impaired mucosal protection play major roles. The analysis demonstrates that effective management depends on accurate identification of the etiological factor and timely assessment of ulcer severity. Upper gastrointestinal endoscopy provides important information about ulcer localization, morphology, and possible complications, while *H. pylori* testing is essential for selecting appropriate eradication therapy. Modern treatment should combine elimination of the causative factor, adequate acid suppression, mucosal healing, and prevention of recurrence. Particular attention should be given to potentially life-threatening complications, including gastrointestinal bleeding and perforation, which require urgent therapeutic intervention. Therefore, an individualized and comprehensive approach integrating etiological treatment, pharmacological therapy, endoscopic monitoring, and prevention of risk factors represents an important principle of contemporary management of gastric and duodenal peptic ulcer disease.

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