

HELICOBACTER PYLORI–ASSOCIATED CHRONIC GASTRITIS: CURRENT DIAGNOSTIC APPROACHES AND THERAPEUTIC STRATEGIES

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Chronic gastritis remains one of the most prevalent gastrointestinal disorders worldwide, with Helicobacter pylori (H. pylori) recognized as its primary etiological agent. Persistent infection induces chronic inflammation of the gastric mucosa and may progress to peptic ulcer disease, gastric atrophy, intestinal metaplasia, and gastric cancer. Accurate diagnosis and timely eradication therapy are therefore essential for preventing disease progression and improving patient outcomes.

This review summarizes current diagnostic approaches and therapeutic strategies for H. pylori-associated chronic gastritis. Recent evidence demonstrates that combining non-invasive and invasive diagnostic techniques with evidence-based eradication regimens improves treatment success. Furthermore, antimicrobial resistance has become an increasing challenge, emphasizing the importance of individualized therapy and antimicrobial stewardship..

Introduction

Chronic gastritis is characterized by persistent inflammation of the gastric mucosa resulting from infectious, autoimmune, environmental, or chemical factors. Among these, *Helicobacter pylori* infection remains the leading cause globally. More than half of the world's population is estimated to be infected with *H. pylori*, although only a proportion develop clinically significant disease. The bacterium survives within the acidic gastric environment by producing urease, which converts urea into ammonia and carbon dioxide, creating a protective alkaline microenvironment. Persistent colonization stimulates chronic inflammatory responses involving neutrophils, macrophages, and lymphocytes, leading to progressive mucosal injury.

If untreated, chronic gastritis may progress to gastric atrophy, intestinal metaplasia, dysplasia, and gastric adenocarcinoma. Consequently, early diagnosis and effective eradication therapy represent important public health priorities.

Aim of the Study

To review current evidence regarding the diagnosis and pharmacological management of *Helicobacter pylori*-associated chronic gastritis and evaluate recent advances in eradication therapy.

Materials and Methods

Scientific publications were identified through searches of PubMed, Scopus, Web of Science, and Google Scholar. Articles published between 2020 and 2026 concerning *Helicobacter pylori*, chronic gastritis, diagnosis, eradication therapy, antimicrobial resistance, and proton pump inhibitors were reviewed. Original clinical studies, systematic reviews, meta-analyses, and international clinical guidelines published in English were included. Duplicate publications, conference abstracts, and studies lacking adequate methodological quality were excluded. Data extraction focused on diagnostic methods, eradication regimens, treatment outcomes, adverse events, and resistance patterns.

Results

Current evidence indicates that non-invasive diagnostic tests, including the urea breath test and stool antigen test, provide excellent diagnostic accuracy for active *H. pylori* infection. Serological testing remains useful in selected circumstances but cannot reliably distinguish active from previous infection. Upper gastrointestinal endoscopy with gastric biopsy remains the reference investigation in patients with alarm symptoms or suspected complications. Histopathology allows direct assessment of mucosal inflammation, atrophy, intestinal metaplasia, and dysplasia. Standard triple therapy has shown declining eradication rates in many regions because of increasing resistance to clarithromycin and metronidazole. Consequently, bismuth-containing quadruple therapy and concomitant therapy have become preferred first-line treatment options in areas with high antibiotic resistance. Potassium-competitive acid blockers and newer acid suppression strategies have demonstrated improved eradication rates in several clinical trials. Adjunctive probiotic supplementation has also been associated with reduced gastrointestinal adverse effects and modest improvements in treatment adherence.

Discussion

The management of *H. pylori*-associated chronic gastritis has evolved considerably over the past decade. International clinical guidelines increasingly recommend susceptibility-guided therapy whenever antimicrobial resistance testing is available. The growing prevalence of antibiotic-resistant *H. pylori* strains represents one of the greatest challenges facing clinicians. Inappropriate antibiotic use has reduced the effectiveness of traditional triple therapy, making individualized treatment strategies increasingly important.

Proton pump inhibitors remain essential components of eradication therapy by increasing gastric pH and enhancing antibiotic activity. Recently introduced potassium-competitive acid blockers may provide stronger acid suppression and higher eradication rates in selected patient populations. Long-term management should include confirmation of eradication using validated non-invasive tests after completion of therapy. Patients with advanced gastric atrophy or intestinal metaplasia require continued endoscopic surveillance because of their increased risk of gastric malignancy. Future research should focus on novel antimicrobial agents, vaccine development, microbiome-based therapies, precision medicine approaches, and artificial intelligence-assisted prediction of treatment response.

Conclusion

Helicobacter pylori remains the principal cause of chronic gastritis worldwide and continues to represent an important risk factor for peptic ulcer disease and gastric cancer. Early diagnosis, evidence-based eradication therapy, confirmation of treatment success, and rational antibiotic use remain fundamental components of effective disease management. Continued research into antimicrobial resistance and individualized therapeutic approaches will further improve patient outcomes.

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