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Mini Review

Helicobacter pylori infection and its therapeutic approaches

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Abstract

Helicobacter pylori (H. pylori) is a gram-negative, spiral-shaped microaerophilic bacterium that colonizes in the human stomach and is related to various gastrointestinal diseases, including dyspepsia, chronic gastritis, peptic ulcers, and gastric cancer. Eradication of H. pylori significantly reduces the risk of ulcer recurrence and gastric malignancy. This mini-review provides an overview of the biology of H. pylori, its pathogenic mechanisms, diagnostic strategies, and current treatment approaches, with a focus on emerging therapies and antibiotic resistance challenges.

Keywords: Helicobacter pylori, review, chronic gastritis, gastric malignancy, peptic ulcers

Introduction

H. pylori infection has declined from 58.2% between 1980 and 1990 to 43.1% in 2011 and 2020 but remains above 60%-70% in many developing countries.[1] It was first isolated in 1983 by Barry Marshall and Robin Warren,[2] who demonstrated its critical role in gastritis and peptic ulcer disease. Since then, eradication strategies have evolved substantially, yet treatment failure due to antimicrobial resistance have become increasingly problem.

Pathogenesis and clinical impact

H. pylori colonizes the gastric mucosa using flagella and urease, which neutralizes gastric acid. Key virulence factors[3] include: CagA (cytotoxin-associated gene A): Linked to increased inflammation and gastric cancer risk.VacA (vacuolating cytotoxin A): Disrupts epithelial cell integrity. Urease enzyme: Converts urea to ammonia, aiding survival in acidic environments. Clinically, infection may be asymptomatic or lead to chronic gastritis, peptic ulcer disease (PUD), gastric mucosa-associated lymphoid tissue (MALT) lymphoma, gastric adenocarcinoma.

Diagnostic methods

Invasive tests includeendoscopic biopsy with histology, rapid urease test (RUT), or culture. Non-invasive tests include Urea breath test (UBT);stool antigen test, serology.

Treatment strategies

First-Line regimens for treatment-naïve patients with H.pylori infection[4]: a. Penicillin allergy: Optimized bismuth quadruple therapy(BQT) (10–14 days): Proton pump inhibitor (PPI)+Bismuth subcitrate or subsalicylate+Tetracycline+Metronidazole;b. No penicillin allergy:

1. Optimized BQT.
2. Rifabutin triple: PPI+Amoxicillin+Rifabutin.
3. Potassium-competitive acid blocker(PCAB) dual: Vonoprazan+Amoxicillin.
4. PCAB- Clarithromycin triple: Vonoprazan+Amoxicillin+ Clarithromycin.

Salvage regimens for treatment-experienced patients with persistent H.pylori infection[4]: a.Clarithromycin and levofloxacin sensitive:

1. Clarithromycin triple.
2. Optimized BQT.
3. Rifabutin triple.
4. Levofloxacin triple:PPI+Levofloxacin+Amoxicillin+Metronidazole;b. Clarithromycin and levofloxacin resistant:

a. Optimized BQT.

b. Rifabutin triple: PPI+Amoxicillin+Rifabutin.

c. High dose PPI or PCAB dual: Vonoprazan+Amoxicillin.

Challenges and emerging therapies

Rising resistance, especially to clarithromycin, metronidazole, and levofloxacin, has reduced eradication success. Resistance-guided therapy using culture or PCR-based assays is increasingly recommended. On the other side, novel approaches include P-CABs: vonoprazan offers superior acid suppression compared to PPIs; Probiotics: Adjunctive use may improve eradication rates and reduce side effects; Vaccines: Still under investigation, none available clinically.[4]

Conclusions

Effective H. pylori eradication remains a cornerstone in preventing gastrointestinal complications, including ulcers and gastric cancer. However, antibiotic resistance necessitates personalized, region-specific treatment strategies. Advances such as P-CABs and molecular diagnostics represent promising developments in this evolving field.

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Conflicts of interest

Authors declare that there is no conflict of interest.

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