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Review

Helicobacter pylori - Impact on Gastritis and Peptic Ulcer: A Brief Review

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ABSTRACT

Helicobacter pylori (*H. pylori*) is a spiral-shaped, Gram-negative bacterium that colonizes the gastric epithelium and represents one of the main etiological factors for chronic gastritis and peptic ulcer disease. This globally prevalent infection transmitted chiefly via the oral-oral or fecal-oral route is linked to unfavorable socioeconomic conditions and poor hygiene and may be found as a commensal organism in up to 50 % of individuals. A hallmark of *H. pylori* is its ability to survive in an acidic environment through production of urease, which converts urea into ammonia and carbon dioxide, thereby reducing local acidity. Persistent bacterial presence triggers a chronic inflammatory response, destroys the stomach's protective barrier and may lead to ulcer formation. Beyond gastrointestinal manifestations, *H. pylori* infection increases the risk of gastric neoplasms such as adenocarcinoma and mucosa-associated lymphoid tissue (MALT) lymphoma. Standard treatment aims at bacterial eradication with a proton-pump inhibitor combined with antibiotics; however, rising antimicrobial resistance is undermining these regimens and driving the search for alternative protocols, including new antibiotic combinations and probiotic supplementation. Early detection and treatment are pivotal to minimize complications such as bleeding or gastric perforation and to reduce gastric cancer risk. Diagnosis relies on invasive tests (endoscopy with biopsy) or non-invasive tests (urea breath test, stool antigen test and serology), each with specific sensitivity and specificity profiles. Given its clinical and epidemiological relevance, *H. pylori* infection remains a major focus in gastroenterology, necessitating ongoing research into pathophysiology, therapeutic optimization and public-health policies for prevention and control.

Keywords: *Helicobacter pylori*; Gastritis; Peptic ulcer; Bacterial resistance; Gastric cancer

Introduction

Helicobacter pylori (*H. pylori*) is one of the most significant infectious agents in upper-gastrointestinal disease, serving as a key etiological factor for chronic gastritis, peptic ulcer disease and, in some cases, gastric neoplasia[1,2]. Since its discovery by Barry Marshall and Robin Warren in 1982, knowledge of the bacterium's pathophysiology and clinical consequences has expanded dramatically, reshaping many aspects of gastroenterology. *H. pylori* is a spiral, microaerophilic, Gram-negative microorganism that produces urease, an enzyme essential for neutralizing the stomach's acidic pH[3]. Global prevalence exceeds one-half of the world's population. Rates are even higher in developing countries due to socioeconomic factors such as inadequate sanitation, high population density and limited access to clean water. Transmission occurs mainly via oral-oral and fecal-oral routes and is associated with hygiene practices and food handling. Although many infected individuals remain asymptomatic for life, persistent colonization elicits chronic inflammation that can progress to atrophic gastritis, peptic ulcer disease or even gastric cancer[4].

The bacterium's pathogenic core rests on its ability to adhere to gastric epithelium and buffer its micro-environment through urease-mediated ammonia production. *H. pylori* also secretes virulence factors most notably VacA cytotoxin and CagA protein that damage tissue, provoke inflammation and perturb immune responses. Local inflammation characterized by pro-inflammatory cytokine release attracts innate and adaptive immune cells, resulting in mucosal injury and structural changes that may advance to atrophy and intestinal metaplasia[5].

Disease outcome is multifactorial, involving both host and microbial characteristics. Host genetics, diet pattern, smoking and stress influence clinical progression, while bacterial strains carrying particular virulence genes (e.g., *cagA*, *vacA*) provoke more intense inflammation and thus more severe disease[6]. Dysregulated acid production in response to infection further undermines mucosal integrity, predisposing to ulcers. Peptic ulcer disease-defined by deeper mucosal loss in stomach or duodenum may also be influenced by chronic NSAID use and lifestyle factors, yet *H. pylori* remain the primary cause in many populations[7]. Medically, *H. pylori* garners attention for its strong association with gastric neoplasia, chiefly adenocarcinoma and MALT lymphoma. In certain patients, chronic inflammation fosters cellular alterations that progress through pre-malignant stages to cancer. Accordingly, detecting and eradicating *H. pylori* are crucial preventive strategies[8].

Objectives

To provide a critical and comprehensive review of the scientific literature on *Helicobacter pylori*, emphasizing its impact on the pathophysiology, diagnosis, therapeutic management and prevention of chronic gastritis and peptic ulcer disease, as well as its role in gastric carcinogenesis.

Materials and Methods

A literature review was conducted using the PubMed, SciELO, Google Scholar and ScienceDirect databases.

Discussion

Lesion formation in *H. pylori*–associated gastric and duodenal disease is driven by a complex host–pathogen interplay. Colonization begins with bacterial movement through gastric mucus, aided by flagella and urease-mediated pH buffering[9]. Adherence to epithelial cells is mediated by specific adhesins, while bacterial proteins such as CagA and VacA initiate inflammatory cascades and cellular damage (VacA induces gastric-cell apoptosis; CagA disrupts cellular structures and intensifies inflammation). From the host perspective, infection elicits an innate immune response characterized by pro-inflammatory cytokines (interleukins-1, -6, -8 and tumor necrosis factor-alpha), chemokines and activation of neutrophils and macrophages. Notably, IL-8 production is triggered by CagA. Adaptive mechanisms follow, recruiting T and B lymphocytes and sustaining inflammation. An imbalance between protective factors (bicarbonate, mucus, mucosal blood flow) and aggressive factors (hydrochloric acid, pepsin, bacterial toxins) culminates in mucosal damage and potential ulceration[10,11].

Clinical outcomes ranging from asymptomatic infection to chronic gastritis, peptic ulcer or neoplasia depend on bacterial strain, host genetics and environmental factors (diet, smoking, alcohol consumption, medications). *cagA*-positive strains are linked to stronger inflammation and higher complication risk. Exaggerated immune responses can produce extensive tissue destruction, facilitating ulcer development[12]. Ulcers manifest clinically as epigastric pain, burning and postprandial discomfort; severe cases may bleed or perforate, necessitating urgent care. Long-standing infection may drive proliferative mucosal changes leading to atrophic gastritis, intestinal metaplasia and elevated cancer risk[13]. Therapeutic strategies for *H. pylori* eradication have evolved. Initial triple therapy clarithromycin, amoxicillin (or metronidazole) and a proton-pump inhibitor has lost efficacy due to rising resistance, particularly to clarithromycin and metronidazole. Current guidelines favor quadruple regimens, antibiotic substitutions for rescue therapy (e.g., levofloxacin or bismuth) and longer treatment courses (10-14 days) to overcome resistance[14,15].

Conclusion

Infection with *Helicobacter pylori* stands as a landmark discovery in gastroenterology, fundamentally altering perceptions of gastric-disease etiology and management. Before Marshall and Warren's findings, chronic gastritis and peptic ulcer disease were often attributed to stress, diet or excess acid. Proving that a microorganism could survive in such a hostile environment and provoke chronic inflammation revolutionized clinical practice and research. *H. pylori* colonization triggers sustained inflammatory responses that can progress to chronic gastritis, peptic ulcer disease and, in some cases, gastric neoplasia. Disease heterogeneity reflects bacterial virulence, environmental conditions and individual susceptibility. Oral-oral and fecal-oral transmission routes, coupled with socioeconomic and hygiene factors, confer global prevalence, especially in developing regions.

Early diagnosis is critical. Endoscopic biopsies enable direct detection via histology, culture or rapid urease testing, whereas non-invasive methods-urea breath testing, stool antigen assays, serology offer alternatives for patients unwilling or unable to undergo endoscopy. Confirmation of infection is the first step in preventing serious complications. Therapeutic progress demonstrates ongoing efforts to circumvent resistance. While clarithromycin-based triple therapy once achieved high

eradication rates, widespread antibiotic use fostered resistant strains, reducing efficacy. Quadruple regimens incorporating bismuth or alternative antibiotics and extended treatment durations now improve success and susceptibility-guided approaches may further enhance outcomes.

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