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HELICOBACTER PYLORI AND GASTRIC CANCER: PROSPECTS OF A PERSONALIZED APPROACH

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Abstract: This scientific thesis thoroughly examines the pathological changes induced by Helicobacter pylori infection in the gastric mucosa, including their clinical manifestations, morphological stages, and its role in the development of gastric cancer. Modern diagnostic methods—both invasive and non-invasive—are discussed in detail, alongside effective treatment strategies such as bismuth-based quadruple therapy, combination antibiotic regimens, and adjunctive probiotic use. The scientific and practical relevance of this work lies in the fact that early detection and treatment of H. pylori infection play a crucial role in preventing gastric cancer among the population.

Keywords: Helicobacter pylori, chronic gastritis, intestinal metaplasia, glandular atrophy, gastric cancer, CagA, VacA, p53 mutation, dysplasia, epigenetic changes, modern therapy, urease enzyme, probiotics, inflammatory mediators, non-invasive diagnostics.

Relevance of the Topic: Helicobacter pylori is a microaerophilic, Gram-negative, spiral-shaped bacterium that colonizes the human gastric mucosa. Due to long-term colonization, it leads to various degrees of gastrointestinal pathologies, especially chronic gastritis, peptic ulcer disease, metaplasia, and gastric cancer. According to the World Health Organization (WHO), more than 50% of the global population is infected with H. pylori [5]. Each year, nearly 1 million new cases of gastric cancer are diagnosed worldwide, with approximately 700,000 related deaths. Gastric cancer ranks third among cancers in terms of mortality. H. pylori predominantly localizes in the antral and corpus regions of the stomach and initiates inflammation of the mucosal layer [2]. The virulence factors of the bacterium—CagA (cytotoxin-associated gene A), VacA (vacuolating cytotoxin A), oipA, and babA—directly damage epithelial cells. Furthermore, the urease enzyme produced by H. pylori hydrolyzes urea into ammonia, altering the gastric juice pH and weakening the mucosal defense, ultimately triggering chronic inflammation. Morphologically, chronic gastritis is characterized by lymphocytic infiltration, epithelial dystrophy, lamina propria infiltration with granulocytes, and finally glandular atrophy [1]. Clinically, the infection presents with dyspeptic symptoms such as nausea, bloating, abdominal pain, chronic fatigue, and sometimes anemia.

Precancerous Stages: Atrophy and Intestinal Metaplasia

Prolonged H. pylori infection results in glandular atrophy of the gastric mucosa, which paves the way for intestinal metaplasia—a transformation of the gastric epithelium into intestinal-type epithelium, often marked by the appearance of Goblet cells. This condition is regarded as premalignant. Intestinal metaplasia is typically accompanied by genetic and epigenetic alterations, including mutations of the p53 gene, reduced E-cadherin expression, and changes in DNA methylation, all of which contribute to the initiation of carcinogenesis [3].

Pathogenesis of Gastric Cancer

Gastric adenocarcinoma associated with H. pylori infection predominantly belongs to the intestinal type according to the Lauren classification [4]. The classical sequence in this model follows:

Gastritis → Atrophy → Intestinal Metaplasia → Dysplasia → Carcinoma.

Several pathological mechanisms are involved in this process: Cytokines and Inflammatory Mediators: Activation of Interleukin-1β, TNF-α, and NF-κB increases epithelial cell proliferation. Genetic Alterations: Silencing or methylation of the CDH1 gene, inactivation of the APC gene, and mutations in p53. Mitochondrial Stress: Reactive oxygen species (ROS) produced by H. pylori damage cellular DNA. Epidemiological studies have confirmed that H. pylori infection is a major risk factor for the development of gastric cancer. Consequently, the WHO has classified this bacterium as a Class I carcinogen.

Modern Diagnostic and Treatment Approaches

Currently, H. pylori infection is diagnosed using the following methods: Invasive Methods: Endoscopic biopsy + histology, rapid urease test (CLO test), bacterial culture, and urea breath test. Non-Invasive Methods: Serological testing, urea breath test, and stool antigen detection. Treatment protocols are continually updated to improve eradication rates. First-line therapy typically includes: Bismuth-based Quadruple Therapy: Bismuth subcitrate + metronidazole + tetracycline + proton pump inhibitor (PPI) [6]. Combination with New-Generation Antibiotics: Regimens based on clarithromycin, levofloxacin, or rifabutin. Probiotics: Adjunctive use of probiotics has been shown to improve the effectiveness of H. pylori eradication.

Conclusion

Helicobacter pylori is a microorganism with a strong pathogenic link between chronic gastritis and gastric cancer. Early detection and effective eradication of this infection constitute critical components in the prevention of gastric cancer. In clinical practice, emphasis should be placed on accurate diagnosis, treatment, and post-eradication monitoring of H. pylori infection. Furthermore, combining standard treatment with probiotics may significantly enhance therapeutic outcomes.

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