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ROLE OF H. PYLORI IN CHRONIC GASTRITIS AND ULCER DISEASE

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Abstract

This article discusses in detail the crucial role of Helicobacter pylori infection in the etiology and pathogenesis of chronic gastritis and peptic ulcer disease. The adaptation of the microorganism to the acidic environment of the stomach (in particular, protection by the urease enzyme), the mechanisms of adhesion to epithelial cells, the release of cytotoxic factors and the activation of the local immune-inflammatory response are analyzed on a scientific basis. Also, the increased acid production as a result of increased gastrin secretion by the bacteria, the disruption of the protective barrier systems of the mucous membrane and, as a result, the formation of erosion and ulcer defects are explained step by step. The article reviews the morphological changes associated with H. pylori - lymphocytic and neutrophilic infiltration, glandular atrophy, intestinal metaplasia, destructive lesions of the mucous membrane - and their clinical manifestations in their interrelation. In addition, the practical importance of modern diagnostic methods (breath test, endoscopic biopsy, serological and fecal antigen tests) and the principles of eradication treatment are highlighted. The article substantiates the possibility of reducing the recurrence of chronic gastritis and ulcer disease and preventing complications through early detection and targeted treatment of H. pylori infection

Keywords

chronic gastritis, ulcer disease, Helicobacter pylori, inflammation, mucosa, acid secretion, eradication.

Relevance of the topic: Chronic gastritis and ulcer disease are widespread throughout the world and are important gastroenterological problems that lead to disability and a decrease in the quality of life among the working population. Recent scientific studies have shown Helicobacter pylori infection as one of the main etiological factors of these diseases. This bacterium persists in the gastric mucosa for a long time, provokes a chronic inflammatory process, weakens protective barriers and enhances the effect of acid-aggressive factors.

Pathological processes associated with H. pylori are inextricably linked not only with gastritis and ulcers, but also with glandular atrophy, intestinal metaplasia and even an increased risk of oncology. Therefore, a thorough study of the role of this microorganism in pathogenesis is of

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urgent importance in clinical practice for early diagnosis, development of effective treatment strategies and prevention of complications. Especially in developing countries, sanitary and hygienic conditions, food culture and high prevalence of infection further increase the importance of the topic.

Purpose of the topic: The purpose of this article is to deeply analyze the etiological, pathogenetic and morphological role of Helicobacter pylori infection in the development of chronic gastritis and ulcer disease. Also:

- to shed light on the mechanisms of adaptation and damage to the gastric mucosa by the bacterium,
- to explain the relationship between inflammatory processes and acid secretion,
- to show the relationship between morphological changes and clinical signs,
- to substantiate the importance of modern diagnostics and eradication treatment,
- to scientifically analyze ways to reduce disease recurrence and prevent complications.

Main part: Helicobacter pylori infection plays a central pathogenetic role in the development of chronic gastritis and peptic ulcer disease. This bacterium is spiral-shaped, gram-negative and motile, and is able to survive in the strongly acidic environment of the stomach. The main reason for this is its production of the enzyme urease. Urease breaks down urea into ammonia and carbon dioxide, resulting in a relatively alkaline microenvironment around the bacteria. Thus, microorganisms are protected from the effects of acid and persist on the surface of the mucous membrane for a long time.H. pylori penetrates the mucous layer of the gastric mucosa and adheres to epithelial cells. Bacterial adhesins play an important role in the adhesion process. As a result of this close contact, the bacteria secrete cytotoxic factors, including vacuolizing toxins and proteins that enhance inflammation. As a result, epithelial cells are damaged, the protective barrier properties of the mucous membrane are weakened, and a local inflammatory process begins.

During the inflammatory process, neutrophils, lymphocytes, and macrophages migrate to the mucosa. They secrete inflammatory mediators, further deepening the process. This leads to the formation of chronic gastritis. Over time, atrophic changes are observed in the glandular apparatus, and intestinal metaplasia in the epithelium. These changes reduce the physiological ability of the mucosa to regenerate.H. pylori infection increases the secretion of the hormone gastrin. An increase in gastrin, in turn, increases the production of hydrochloric acid. As a result, the gastric mucosa is constantly exposed to an aggressive acid-peptic environment. The mucosa, whose protective factors are weakened, cannot withstand such effects, and erosions and ulcer defects occur. In particular, it has been established that the majority of duodenal ulcers are associated with H. pylori.

Morphologically, in chronic gastritis, the mucosa thickens, inflammatory infiltration increases, and the number of glands decreases. In ulcer disease, a defect reaching the deep layers of the mucosa, foci of necrosis, and subsequent formation of fibrous tissue are observed. These changes are clinically manifested by pain, dyspeptic symptoms, and sometimes bleeding.

Detection of H. pylori is important in the diagnostic process. For this, urease breath test, endoscopic biopsy, serological tests, and antigen detection methods in feces are used. Early

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diagnosis allows for proper treatment of the disease. Eradication treatment uses a combination of antibiotics and proton pump inhibitors. This treatment, along with the destruction of bacteria, reduces inflammation, creates conditions for the restoration of the mucous membrane, and prevents ulcer recurrence. Thus, the elimination of H. pylori infection directly affects the pathogenesis of chronic gastritis and ulcer disease.

Conclusion: Helicobacter pylori infection is a leading etiological and pathogenetic factor in the occurrence and development of chronic gastritis and ulcer disease. Adaptation of this microorganism to the acidic environment of the stomach, cytotoxic effect by adhesion to epithelial cells, activation of local immuno-inflammatory processes and increased acid secretion disrupt the protective mechanisms of the mucosa. As a result, chronic inflammation, erosion, atrophic changes and ulcer defects occur in the mucosa.

The close relationship between morphological changes and clinical signs confirms the crucial role of H. pylori in the pathogenesis of the disease. Early detection of infection through modern diagnostic methods and the use of targeted eradication therapy slow down the inflammatory process, accelerate the recovery of the mucosa and dramatically reduce the likelihood of ulcer recurrence.

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