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HELICOBACTER PYLORI INFECTION AND PRINCIPLES OF THERAPY IN CHILDREN

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Abstract
The article outlines modern views on the principles of therapy for Helicobacter pylori infection, the resistance of the pathogen, the drugs used, their combinations and effectiveness in achieving remission and reducing the frequency of relapses.

Keywords: Helicobacter pylori infection, treatment, effectiveness.

Introduction

In modern conditions, the course of gastroenterological diseases undergoes certain changes. This is manifested by a gradual and imperceptible onset, “rejuvenation” of the disease, persistent chronic relapsing course, loss of seasonality of exacerbations, and the appearance of severe complications [2]. Currently, there is a high frequency of combined, multiple organ pathologies, an increase in allergic diseases, chronic intoxication syndrome, which results in resistance to traditional methods of treatment and the difficulty of choosing a drug.

The most common among chronic diseases of the digestive system is pathology of the stomach and duodenum (gastritis, gastroduodenitis, peptic ulcer). The leading factors in the development of chronic inflammatory diseases of the gastroduodenal region are nutritional, neurogenic, hereditary, immunological, and allergic. In the last 25 years, most domestic and foreign gastroenterologists have attributed the development of gastritis and peptic ulcers to pyloric helicobacteriosis [1, 2]. As a result of exposure to Helicobacter pylori (Hp), local immunity is disrupted, the microecological structure of the stomach and intestines is destroyed, and a vicious circle of the inflammatory process of the mucous membrane of the digestive organs is closed [5].

Epidemiological studies conducted in various countries and presented in the literature indicate that 75-100% of cases of chronic gastritis, 70-80% of cases of gastric ulcer, 80-100% of duodenal ulcer, 30-90% of non-ulcer dyspepsia is caused by Hp infection [4]. The level of infection with Helicobacter pylori infection among children 7-11 years old with diseases of the upper gastrointestinal tract exceeds 50% and is almost 80% in children of senior school age. A. A. Korsunsky [1] revealed the prevalence of this infection in 60-70% of children. N.I. Ursova [5] notes that Hp infection begins in early childhood, reaching 33.3% by 10 years of age and 56.3% by 17 years of age.

Numerous data indicate that Hp infection, like any other, is not limited only to local effects on the gastric mucosa, but can also exhibit systemic effects (inflammatory, autoimmune, allergic, etc.), causing corresponding reactions from certain organs and systems. HP may be related to the development of extragastric pathological conditions: blood diseases (iron deficiency anemia, thrombocytopenic purpura), various skin diseases (atopic dermatitis, lichen planus,

rosacea, psoriasis, erythroderma). There is also evidence of the role of Hp in delayed physical development in children, food allergies, etc. [2,3].

The proposed mechanism underlying extragastric pathology associated with HP involves the direct effects of bacteria: the inflammatory process is activated along with the release of cytokines and inflammatory mediators and subsequent systemic effects; this ultimately leads to mimicry between bacterial and host antigens. HP triggers a cascade of immune reactions with an increase in the amount of interleukins, lysosomal enzymes, and tumor necrosis factors. Thanks to the production of a number of enzymes, HP is able to have an immunosuppressive effect, influence the differentiation of T-lymphocytes and inhibit phagocytic activity [4]. Thus, the presence of common properties of HP with components of the gastric mucosa makes it possible to identify this microbe also as an inducer of autoimmune reactions. There are three possible mechanisms leading to diseases associated with immune disorders: HP interact with mast cells, initiating the release of mediators. HP, acting as full-fledged antigens, cause allergic reactions in the host's body. HP reduce the barrier function of the intestine, causing the entry of allergens into the blood (incomplete hydrolysis of nutrients).

In allergic diseases (in particular, atopic dermatitis), as a result of sensitization of the body HP, an abnormally high level of Ig E is formed. When the allergen interacts with Ig E, mast cells are activated with the release of allergy mediators (tryptase, histamine, platelet activating factor, arachidonic acid metabolites) , which leads to increased vascular permeability, edema, hypersecretion of mucous glands, stimulation of migration of eosinophils and Th-2 cells into the skin and mucous membranes. A direct correlation has been proven between the degree of HP infection and the severity of dermatitis. With a high degree of Hp contamination, the recurrent course of atopic dermatitis becomes continuous; in the absence of bacteria after Hp eradication, the severity of dermatitis is minimal and patients with a continuous course of the disease are not found.

To maintain its existence, HP requires iron. Helicobacter pylori is a virulent agent that absorbs and uses a significant amount of iron for its vital functions [2, 3, 4, 5]. Helicobacter pylori, like other gram-negative bacteria, enter into complex competitive relationships for iron. For this, a siderophore of the phenolate or hydroxomate type synthesized by it is used, which is subsequently combined with ferrate of siderophiles, followed by extraction of iron from the cell surface. Direct lysis of cells is possible under the influence of HP-produced urease and mucinase with the extraction of iron from the macroorganism (human), digestion of hemoglobin and assimilation (assimilation) of heme with the formation of siderophores, allowing the extraction of iron from the macroorganism.

L. Dhaenens et al. [2] compared the iron requirement among 4 Helicobacter species that persist in the gastric mucosa (H. pylori, H. felis, H. acinonyx, H. mustelae) and 5 Helicobacter species that colonize the intestinal tract (H. fennelliae, H. cinaedi , H. muridarum, H. bilis, H. hepaticus). It was revealed that gastric Helicobacter species, with the exception of Hp, which uses iron from human lactoferrin, obtain iron for their vital functions from heme and hemoglobin. Other Helicobacter species found in the intestine are able to use a fairly wide range of iron sources for their growth (bovine and human lactoferrin, transferrin, heme and hemoglobin). The ability of HP to use human lactoferrin as a source of iron, discovered in HP, determines the special virulence of Helicobacter infection [3].

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HP positive patients have lower levels of serum ferritin and iron compared to HP negative patients [2, 3], and in patients with atrophic gastritis associated with HP infection, these indicators of "iron" status were the lowest. The presence of HP on the gastric mucosa contributes to the development of iron deficiency anemia in children. The presence of HP infection in combination with iron deficiency anemia is often accompanied by damage to the entire gastric mucosa and the development of pangastritis. Oral ferrotherapy restores and maintains normal iron levels, but after its cessation the anemia returns. M. Konno et al. [8] proved that the traditionally used correction of iron deficiency and resulting iron deficiency anemia in Helicobacter pylori infection is unjustified. C. Hershko, A. Lahad, D. Kereth [3] believe that iron deficiency anemia has a beneficial effect on the destruction of HP.

The introduction of excess amounts of iron (both with food and with medications) affects the severity of the infectious process and reduces the overall resistance of the macroorganism. The presence of exogenous iron leads to increased reproduction of HP. Therefore, in the treatment of patients with iron deficiency anemia associated with HP, it is recommended to use eradication therapy with the inclusion of a proton pump inhibitor and 2 antibacterial drugs for 2 weeks [3] without additional intake of iron supplements.

A control study of patients who received a proton pump inhibitor in combination with two antibacterial drugs at long-term follow-up (between 27 and 50 months after eradication therapy) did not reveal signs of anemia [3]. M. Kostaki, S. Fessatou, T. Karpathios [4] showed the effectiveness of ferrotherapy in children with chronic gastritis associated with HP without signs of esophagogastrointestinal bleeding, only after anti-Helicobacter therapy.

Taking this into account, the Maastricht Consensus-3 (2005) recommended that in cases of unexplained iron deficiency anemia in persons infected with HP, anti-Helicobacter therapy aimed at the complete destruction of HP.

Thus, the current stage of studying HP associated diseases dictates the need for mandatory eradication therapy, since the use of only symptomatic therapy with the help of antatsides, cytoprotectors, prokinetics leads to elimination of HP in only 37%. Complete eradication of this infection leads to the cure of HP-associated gastritis.

Previously, colloidal bismuth tri-potassium dicitrate ("De-nol") was used as a drug that ensures the destruction of HP, isolated as monotherapy for 14-28 days with an eradication effect of no more than 14% or in combination with ampicillin (dual therapy) (efficiency reached 70-80%) [6].

Bismuth tripotassium dicitrate leads to a change in the structure and destruction of the microbe, weakens the action of Hp enzymes, which helps to increase the effectiveness of the body's defenses against the bacterium. The anti-Helicobacter properties of bismuth tripotassium dicitrate are ensured by reducing the adhesion of HP to epithelial cells; weakening the action of HP enzymes, such as urease, catalase, lipase, destruction of the bacterial wall due to the formation of deposit complexes on the bacterial wall and in the periplasmic space [7]. In the acidic environment of the stomach, colloidal bismuth subcitrate forms a protective film, protecting the mucous membrane from the aggressive action of gastric juice. Due to its colloidal structure, bismuth tripotassium dicitrate directly connects with both epithelial cells and protein molecules in areas of necrosis, isolating the bottom of the ulcer from the digestive acid-pepsin factor [6, 8]. Bismuth preparations stimulate the secretion of bicarbonates, form complexes

with mucus, creating a barrier to the diffusion of hydrochloric acid. Bismuth salts increase the synthesis of prostaglandins in the stomach wall, increase the secretion of mucus and bicarbonate ions, thereby providing an antisecretory effect. In addition, under the influence of bismuth subcitrate, the blood supply to the stomach and duodenum is improved, the production of cytokines by cells of the inflammatory infiltrate is suppressed, the reconstruction of the extracellular matrix and complete angiogenesis is ensured, and there is a reparative effect on the mucous membrane.

Proton pump inhibitors (PPIs) are used in all eradication therapy regimens (triple or quadruple therapy). This is due to the fact that under conditions of low acidity of gastric juice, the activity of antibacterial drugs increases, and the environment for HP life deteriorates. In addition, PPIs themselves have anti-Helicobacter activity in vitro. However, in pediatric gastroenterology there are certain limitations in the range of possible antibacterial drugs for successful eradication of HP.

Thus, for effective control of Helicobacter pylori, including resistant strains, adequate therapy to which this microorganism is sensitive is indicated. It is necessary to take into account a family history of diseases of the upper digestive tract, the fact of earlier use of drugs included in eradication therapy regimens for other diseases. For the treatment of diseases associated with helicobacteriosis and overcoming the resistance of this microorganism to antibiotics, such therapy is indicated, including highly active drugs that allow achieving eradication in no less than 80-90% of patients.

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