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Helicobacter Pylorosis in Children: Features of Diagnosis and Treatment

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Abstract:

The article presents literature data on the mechanisms and role of Helicobacter pylori infection in the development of diseases of the gastroduodenal zone, features of diagnosis and therapy.

Keywords: Helicobacter pylori, pathogenicity factors, eradication therapy.

Introduction

Diseases of the digestive tract occupy one of the leading places in the pathology of childhood. In the structure of diseases of the digestive system, the leading place is occupied by pathology of the stomach and duodenum. The discovery in 1983 by Australian scientists R. Warren and B. Marshall of the microorganism Helicobacter pylori radically changed the scientific view of the pathogenesis of inflammatory diseases of the human gastrointestinal tract. Researchers Barry James Marshall and Robin Warren were able to isolate and cultivate spiral-shaped microorganisms found on the gastric mucosa of a patient suffering from gastritis. The described microorganism was included in the international taxonomy of bacteria in 1985 as Campylobacter pyloridis. In 1987, it was renamed Campylobacter pylori, and in 1989, C.S. Goodwin and a team of scientists finally identified the bacterium as Helicobacter pylori, proving that it does not genetically belong to the genus Campylobacter [8,9,14]. The latter term reflects 2 morphological features: in vivo — helical microorganism, in vitro — rod-shaped (bacter). In 1998, the genome of Helicobacter pylori was completely deciphered. In 2005, Robin Warren and Berry Marshall, the discoverers of the medical significance of bacteria, were awarded the Nobel Prize in Medicine. Pathologist Robin Warren and his young 28-year-old colleague internist Berry Marshall made their discovery while working in Perth, Western Australia, where there was only one hospital. The significance of the discovery of R. Warren and B. Marshall can hardly be overestimated: it required a revision of traditional views on the origin, development and therapy of a wide range of diseases. Subsequently, active study of Helicobacter pylori revealed numerous facts of its involvement in the development and progression of a number of diseases not only of the gastroduodenal zone, but also of extragastric

manifestations [1,5,6], and epidemiological studies indicate a high prevalence of Helicobacter pylori infection, primarily in children.

Helicobacter pylori is a clinically significant pathogen responsible for a substantial portion of total morbidity and mortality worldwide. In 1987, the European Pyloric Helicobacter Pylori Group was established, a non-governmental structure designed to coordinate the efforts of various research groups.

Helicobacter pylori infection is one of the most common chronic human infections. The incidence of Helicobacter pylori infection in adults varies from 40% to 90% in different regions of the world.

The prevalence of Helicobacter pylori in different regions of the world is determined by both the level of economic development of the country and the social level of individual groups of the population within the country. In many patients, Helicobacter pylori can be defined as a symbiotic and non-pathogenic microorganism, which complicates the question of whether to prescribe specific therapy. Helicobacter pylori is a special pathogenic agent that causes asymptomatic course in most infected people. However, patients with an asymptomatic course are a risk group in which chronic gastritis, gastric ulcer, and duodenal ulcer develop over time [7,10,11].

A person acquires Helicobacter pylori in early childhood, usually after the first year of life. Mother-to-child and child-to-child transmission is most likely, the risk of infection is highly correlated with the infectious status of the mother and siblings, and is also associated with living conditions in overcrowded families [2,3,4].

The transmission mechanism is contact and household. However, Helicobacter pylori quickly dies outside the human stomach due to exposure to higher concentrations of oxygen and even light. The most likely routes of transmission are gastro-oral (possible in gastroenteritis with vomiting); oral-oral when the saliva of an infected person comes into contact with a healthy person (when kissing, since Helicobacter pylori can survive in saliva and plaque; licking the nipples, using common cutlery, etc.); rarely iatrogenic (it has been proven that Helicobacter pylori can be transferred from one person to another through poorly treated instruments during various probe procedures, through insufficiently disinfected endoscopes and biopsy forceps). Fecal-oral transmission is possible in conditions in which Helicobacter pylori transits through the lower gastrointestinal tract, which is unlikely. The possibility of transmission of Helicobacter pylori with microaerosols formed by talking or coughing is not excluded [12,13].

Early childhood is a critical period for Helicobacter pylori infection. With age, the likelihood of infection with Helicobacter pylori decreases. Helicobacter pylori is the only known microorganism that is able to colonize in the aggressive acidic environment of the human stomach (but quickly dies at a low pH value in the lumen of the stomach). Helicobacter pylori is a special microorganism that has been infecting humans for thousands of years, has a specific habitat Adhesion of Helicobacter pylori occurs only on the epithelium of the stomach and in areas of gastric metaplasia of the duodenum or heterotopia in the large intestine [4,5]. The

bacterium Helicobacter pylori has developed complex defense mechanisms that allow it to avoid the aggressive effects of gastric juice, survive in the gastric mucosa, attach to the gastric epithelium and interact with both it and the immune cells of the host [1, 7, 8, 12]. Helicobacter pylori is a microaerophilic gram-negative bacterium that has a curved S-shaped or slightly spiral shape with polarly arranged flagella. Another form of Helicobacter pylori is coccoides. The peptidoglycan of the wall of the coccoid form of the bacterium is a weak activator of the natural immune response. The transition to the coccoid form may be a consequence of the transformation of the cell wall in order to modulate immunity. Helicobacter pylori converts to the coccoid form in the anaerobic space of the small intestine, is phagocytosed by dendritic cells, and stimulates host immunity by targeting Peyer's plaques. Helicobacter pylori positive individuals have a population of immune cells, including regulatory T cells, that is absent in Helicobacter pylori negatives. Helicobacter pylori has the ability to form biofilms, which contributes to the bacteria's resistance to antibiotic therapy and protects against the host's immune response, thereby increasing its survival in the acidic and aggressive environment of the stomach [6,9, 20].

Helicobacter pylori has pronounced enzyme systems. Urease, the main enzyme of Helicobacter pylori, breaks down urea (including foodstuffs, urea from the bloodstream) into ammonia and carbon dioxide. Urease activity is expressed in almost all strains of Helicobacter pylori, and therefore it is used to diagnose helicobacteriosis. The presence of a "cloud" of alkaline products in its microenvironment protects the pathogen from the effects of an acidic environment (buffering effect of ammonia) and promotes the reproduction of Helicobacter pylori (pH 6.0-8.0). There is a kind of "deception" of the secretory apparatus: gastrin stimulation, a decrease in somatostatin secretion and constant stimulation of hydrochloric acid.

The presence of ammonia in gastric juice disrupts mitochondrial and cellular respiration, causing necrotic damage to the gastric mucosa and contributing to the development of antral gastritis and hypergastrinemia. Helicobacter pylori secretes mucinase, which causes depolymerization and dissolution of the protective mucus of the stomach, contributing to increased permeability to hydrogen ions. Helicobacter pylori also produces such coolant-damaging enzymes as catalase (prevents phagocytosis of Helicobacter pylori), phospholipase (destroys the surfactan-like phospholipid protective layer of coolant), hemolysin, lipase, protease, oxidase, and numerous adhesins.

The most studied virulence factors of Helicobacter pylori are vacuolizing cytotoxin A (VacA), the "pathogenicity island" of Cag-PAI genes embedded in the genome of the most virulent strains of Helicobacter pylori, and its marker, cytotoxin-associated gene A (cytotoxic-associated gene-CagA) [8, 10, 14].

More than 40 pathogenicity (virulence) genes of Helicobacter pylori are not scattered along the chromosome, but are collected in one of its segments, called the "inslet of pathogenicity" - CagPAI. The "pathogenicity island" is embedded in the genome of the most virulent strains of Helicobacter pylori. Its marker-protein CagA with mol. with a mass of 120-140 kD, encoded

by the gene citotoxin associated gene A CagA. An obligatory attribute of virulence is the presence in Sag-PAI of genes of the type IV secretion system (TFSS), which encode macromolecular structures that function as small needles for the transfer of bacterial agents from Helicobacter pylori to host cells, inject plasmid DNA, which allows Helicobacter pylori to modulate the metabolism of gastric mucosal epithelial cells, including the expression of proto-oncogenes. There is a hypothesis that CagA PAI Helicobacter pylori may serve as a new transport system for the secretion of virulence factors [6,7,16].

VacA is a pore-forming cytotoxin. VacA plays an important role in the vital activity of Helicobacter pylori in the human body. To have a toxic effect, VacA must be secreted by the bacterium and delivered in active form to the host cell membrane, where it forms pores that release chloride ions. VacA has several toxigenic properties that may affect the outcome of Helicobacter pylori infection and colonization. The most studied in VacA is the effect of endosomal mutation, leading to vacuolation of epithelial cells. It is assumed that VacA embeds itself in the membranes of endosomal vesicles, forms pores with active channels of chloride ions, and changes the composition of anions inside the endosomes, which subsequently leads to osmotic swelling. VacA is also able to cause the leakage of ions of small molecules such as iron, nickel, sugars and amino acids, disrupting the barrier function of tight compounds without major damage to their integrity. This may be the mechanism by which Helicobacter pylori receives nutrients through the intact epithelial barrier [13,17,18,19].

Another important feature of the pathogenicity of Helicobacter pylori is its ability to block the proton pumps of parietal cells, resulting in transient hypochlorhydria contributing to other infections, such as helminthiases. Under unfavorable conditions, Helicobacter pylori can transform into an atypical coccal form, i.e. less vulnerable to antibiotics, then back into a full-fledged S-shaped form.

Among all the methods of diagnosing Helicobacter pylori, there are two large groups – invasive and non-invasive methods. Invasive ones are based on the study of biopsies during FEGDS, non-invasive ones do not require endoscopic examination. To date, there is no universal method for diagnosing Helicobacter pylori that would achieve 100% sensitivity and specificity. Each method has its own advantages and disadvantages, which determine its indications for use in clinical practice [2,4].

The most widely used is a seven-day, three-drug regimen with a proton pump inhibitor (PPI) and two antibiotics. The action of these drugs is aimed at both reducing the acid-forming function of the stomach and destroying Helicobacter pylori on the surface of the mucous membrane. PPIs have no anti-Helicobacter pylori activity, but they do increase the pH of gastric secretions. At the same time, vegetative forms of Helicobacter pylori existing on the surface of the mucous membrane of the antral part of the stomach, protecting themselves from the effects of acid by the ammonia membrane, die under alkaline conditions under the influence of ammonia formed by them. A kind of "suicide" of Helicobacter pylori occurs. Those bacteria

that have survived in the fundal region in the form of cocci, when the pH in the stomach increases, turn into a vegetative form and become available to antibiotics.

More than 25 years of experience in the treatment of Helicobacter pylori infection has shown that eradication is becoming increasingly difficult as the microorganism rapidly acquires resistance to antibacterial drugs.

References

1. Khudaiberganova, N. Kh., Yuldosheva, D. Sh., Abzalova, D. A., and Rashidova M. A. (2019). Study of the nutritional status of overweight school-age children. Volume II, 361
2. Xudayberganova, N.X., Rahmatullayeva, G. K. (2023). To assess the incidence of iron deficiency anemia and Helicobacter pylori infection among school-age children with chronic gastroduodenal pathology. European Scientific and Methodological Journal 1(8), 10-18.
3. Khudayberganova, N. Kh., & Akhmedova, I. M. Clinical and biochemical features of extragastric manifestations of helicobacter pylori associated gastroduodenal pathology in children. Yangi Uzbekiston: Innovation, fan va ta'lim 18-KISM, 26..
4. Khudaiberganova, N. Kh., Akhmedova, I. M. (2023). The course of chronic gastroduodenal pathology in children and helicobacter pylor infection. Academic research in educational sciences, (1), 196-205.
5. Akhmedova, I. M., & Khudaiberganova, N. Kh. (2022). Extragastric manifestations of chronic gastroduodenitis in children.
6. Khudaiberganova N. Kh., (May 2022). Clinical characteristics of Helicobacter pylori associated gastroduodenal pathology in children. Journal of new centure innovations. Volume -4
7. Khudayberganova, N. Kh., Rakhmatullaeva, G. K., & Alikulov, I. T. (2023). Helicobacter pylori infection and principles of therapy in children. Best Intellectual Research, 9(3), 272-277.
8. Khudayberganova, N. Kh., Rakhmatullaeva, G. K. (2023). Prevalence of helicobacter pylori infection in children with gastroduodenal pathology. Best Intellectual Research, 9(3), 278-281.
9. Khudayberganova, N. Kh., Azimova, M. M., & Abzalova, D. A. (2022). Clinical efficacy of Ferlatum for the treatment of iron deficiency anemia (Doctoral dissertation).
10. Khudayberganova, N. Kh. (2023). To study helicobacter pylori infections in school-aged children with chronic associated gastroduodenal pathology. Best Intellectual Research, 9(3), 282-289.
11. Khudayberganova N. K., & Rahmatullayeva, G. K. (2023). To Assess the Frequency of Iron Deficiency Anemia and Helicobacter Pylori Infection Among School-Age Children with Chronic Gastroduodenal Pathology. European Science Methodical Journal, 1(8), 10-18.

12. Khudayberganova, N. Kh., Rakhmatullaeva, G. K., & Tagaeva, M. Kh. (2023). Features of the course of Helicobacteriosis in children with chronic gastroduodenitis.
13. Akhmedova, I. M., Tuhsanova, R. S., & Khudaibergenova, N. Kh. (2017). The role of helicobacter pylori infection in the formation of food allergies in children. Pediatrician, 8(S1), M36-M36.
14. Salaeva, M. S., & Khudaiberganova, N. Kh. (2015). Correlation of social factors with parameters of quality of life in patients with chronic obstructive pulmonary disease. Bulletin of KSMA named after IK Akhunbaev, (3), 74-79.
15. Khudayberganova, N. Kh., Rakhmatullaeva, G. K., & Alikulov, I. T. (2023). Helicobacter pylori infection and principles of therapy in children. *Best Intellectual Research*, *9*(3), 272-277.
16. Талипов, Р. М., Тулабаева, Г. М., Сагатова, Х. М., Нурметов, Х. Т., & Худайберганова, Н. Х. (2021). Peculiarities of comorbidity in elderly patients with myocardial infarction. *Узбекский медицинский журнал*, *2*(3).
17. Azadaeva, K. E., Tukhtaeva, N. Kh., Azimova, M. M., & Khudaiberganova, N. Kh. Retrospective analysis of the clinical state of patients with reactive arthritis taking into account diseases of the digestive system. *Uzbekistan Respublikasi soǵliqni saqlash vazirligi Toshkent tibbiyot akademiyasi*, Bet:71.2023
18. Shukurova, F. N., & Saidova, M. T. (2015). Risk factors for early development of carotid atherosclerosis in patients with rheumatoid arthritis. *Medicus*, (6), 107-110.
19. Shukurova, F.N., Karimov, M.Sh. (2022). *Joint Involvement Associated with Chronic Hepatitis C: Modern Diagnostic Methods* (Doctoral dissertation).
20. Karimov, M. Sh., Shukurova, F. N., & Parpiboeva, D. A. (2020). Features of the treatment of arthritis associated with chronic viral hepatitis: a comparative analysis of the clinical efficacy of nonsteroidal anti-inflammatory drugs.