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Modern Ideas About the Formation of Helicobacter Pylori Associated Gastroduodenal Pathology in Children

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Annotation. Chronic gastroduodenitis (CGD) in the structure of diseases of the digestive organs belong to the most common pathology of childhood with progressive growth exceeding 100 cases per 1000 children. For comparison, according to A.B. Mazurin in 1989, the prevalence of this pathology was recorded at the level of 79 cases per 1000 children under the age of 14. In addition to a distinct upward trend in detectable HCG, which averages 10% per year, there is a tendency to aggravate the course not only due to an increase in the specific gravity of erosive, subatrophic and atrophic forms, but also due to polymorbid flow with the presence of up to 2 or more concomitant diseases. As the most frequent comorbid conditions of HCG, a number of authors indicate violations of the trophological status (dyslipidemia, changes in the musculoskeletal and cardiovascular systems, a high frequency of autonomic dysfunctions, dysbiotic disorders. The presence of frequent concomitant changes on the part of other organs and systems in children with HCG raises the question of the existence of uniform pathogenetic mechanisms in which HCG is considered as a pathological process involving an integral organism with a violation of metabolic processes, homeostatic parameters, regulatory circuit, intestinal endoecology, and, accordingly, the optimality of the development of a growing organism. Such a complex of changes leads to a decrease in the adaptive capabilities of the child's body with subsequent changes in behavioral reactions and social relations, which goes beyond the scope of gastroenterological pathology proper.

Keywords: gastroduodenoscopy, Helicobacter pylori, children, clinic.

The discovery of the microorganism Helicobacter pylori has radically changed the views of scientists and practitioners on the problem of etiology, pathogenesis, diagnosis and treatment of VOPT diseases. Currently, there is no doubt that Helicobacter pylori in half of patients is the causative agent of chronic gastritis and takes an important part in the pathogenesis of peptic ulcer disease, stomach tumors (14, 21). The Hp microorganism is found in more than 90-95% of patients suffering from duodenal ulcer (duodenal ulcer), in 70-85% of patients with gastric ulcer. In children, the association of gastroduodenal pathology with Helicobacter pylori infection, according to various authors, ranges from 96 to 100% in case of IBD, with erosive lesions of the gastric mucosa (SOJ) and duodenum (SOD) from 58 to 85% and with gastritis / gastroduodenitis without destructive changes. That is why the problem of improving the early diagnosis and adequate treatment of chronic diseases of the upper digestive tract in children acquires not only medical, but also social significance. In modern gastroenterology, highly informative diagnostics of Hp infection of children and parents with symptoms of dyspepsia and the development of acceptable, from a socio-economic point of view, outpatient treatment regimens are of paramount importance. Infection with Helicobacter pylori infection in most cases occurs in childhood. Most often, infection with helicobacteriosis occurs between family members orally or through personal hygiene items. As a rule, all family members are affected by the same strain of microbe, although there are studies proving that two or more strains of Helicobacter pylori microbe can persist in one family at the same time (23). Helicobacter pylori infection is widespread among all segments of the population. More than 50% of people worldwide are infected with Helicobacter pylori. In Africa, Mexico, South America and Central America, the prevalence of infection reaches 70% - 90% among the entire population [22]. In Russia, the prevalence of infection in children reaches 60-70%. The infection rate among children aged 7-11 years with diseases of the upper gastrointestinal tract (2007) exceeds 50% and is almost 80% in children of high school age [10]. Epidemiological studies conducted in various countries and presented in the literature

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indicate that 75-100% of cases of chronic gastritis are caused by Helicobacter pylori infection, as well as 70-80% of cases of gastric ulcer, 80-100% of duodenal ulcer and 30-90% of non-ulcerative dyspepsia [1]. And more and more often we get evidence of the involvement of infection in the development of some other, extra-gastric diseases [17,20]. N.I. Ursova (2009) notes that infection with Helicobacter pylori begins in early childhood, reaches 33.3% by the age of 10 and 56.3% by the age of 17 [6].

Numerous reports in the literature indicate that helicobacter infection, like any other, is not limited only to local effects on the gastric mucosa, but is also capable of showing systemic effects (inflammatory, autoimmune, allergic, etc.)

Helicobacter pylori, causing a chronic inflammatory process in the stomach, accompanied by the production of cytokines, signaling molecules, activation of pro-inflammatory proteins and contributing to intracellular mutations, has not only local, but also some systemic effects and can affect other organs and systems [1, 2]. The results of numerous studies suggest the possible significance of Helicobacter infection pylori in the development of a number of extra-digestive diseases [3, 4]. The list of these diseases is quite extensive, in the genesis of some of them the role of Helicobacter pylori infection can be considered proven, in others it is associated with only a part of cases, sometimes its role is assumed, but the available scientific data are contradictory, therefore require further clarification. It is important to emphasize at the same time that extra-ventricular manifestations, as a rule, develop years after the development of Helicobacter pylori-associated gastritis, they can coexist with it, but sometimes they are a very distant consequence of infection. The severity of extra-ventricular manifestations initiated by Helicobacter pylori does not correspond to the severity of gastroduodenal pathology. In most cases, with extra-gastric manifestations, the activity of chronic gastritis is quite low, but by localization it usually has a widespread character with the involvement of the stomach body.

In this review, we will focus only on those diseases whose connection with Helicobacter pylori infection is most studied.Infection with Helicobacter pylori and iron deficiency anemia. Since the first publication about Helicobacter pylori in 1983, a fairly large number of scientifically substantiated facts have been accumulated, indicating that this infection causes not only stomach diseases, but also has so-called extra-gastric manifestations. In the English-language literature, several collective concepts of such a connection are used - "extradigestive manifestations of Helicobacter pylori", "extra-intestinal clinical manifestations of Helicobacter pylori", "Helicobacter pylori and organ systems outside of the gastrointestinal tract", "extragastric disorders of Helicobacter pylori". [13, 15, 19].

This fact, according to many scientists [20, 22], is due to the fact that H. pylori belongs to the virulent agent that absorbs and uses a significant amount of iron for its vital activity and can compete with the host for the content of iron reserves [23].

Helicobacter pylori, as well as other gram-negative bacteria, enter into complex competitive interactions for iron. For this purpose, a phenolate or hydrosomatic type siderophore synthesized by him is used, later it is combined with siderophile ferrate with subsequent extraction of iron from the cell surface. Direct lysis of cells is possible under the influence of urease and leucinase produced by Helicobacter pylori with the extraction of iron of the macroorganism (human), digestion of hemoglobin under conditions of assimilation of heme with the formation of sideroforms that allow the extraction of iron of the macroorganism (patient). Moreover, the process of absorption and assimilation of iron may be disrupted due to an increase in intragastric pH in common variants of Helicobacter pylori-associated gastritis. With hypochlorhydria, the process of iron oxidation is disrupted, which is necessary for further absorption. This iron absorption disorder in HP-infected patients is also evidenced by experimental studies [5, 15]. The presence of Helicobacter pylori on the mucous membrane contributes to the development of iron deficiency anemia in both adults and children [10, 15]. Moreover, the severity of anemia depends not only on the volume of the affected mucous membrane, but also on the type of persistent Helicobacter pylori. In particular, according to Dhaeneus L., Szczebara F., Van Nieuwenhuyse S. et al (1999) [16], all four types of varieties of strains of helicobacter microorganisms (H. felis, H. acinonyx, H. mustelal), with the exception of Helicobacter pylori, which uses iron from human lactoferrin, receive iron for their vital activity from heme and hemoglobin. Other varieties of Helicobacter are able to use a fairly wide range of iron

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sources for their growth (lactoferrin, transferrin, heme and hemoglobin). The ability found in Helicobacter pylori to use human lactoferrin as a source of iron determines the special virulence of Helicobacter pylori infection [18].

Iron deficiency (J) is the leading cause of anemia in the world, it affects about 2 billion people, among whom children and women of childbearing age make up the largest group [5]. It is accompanied by a decrease in physical strength and performance in adults and adolescents, has a negative impact on the cognitive development of children and increases the risk of infectious diseases [6]. Since the beginning of the 90s of the last century, many works have been published in the world devoted to the study of the relationship between Helicobacter pylori infection and iron deficiency anemia (IDA).

Numerous reports in the literature indicate that helicobacter infection, like any other, is not limited only to local effects on the gastric mucosa, but is also capable of showing systemic effects (inflammatory, autoimmune, allergic, etc.) [12].

This leads to the development of reactions from other organs and systems, can contribute to the occurrence of extra-gastric pathological conditions, including the formation of vascular and autoimmune diseases, as well as blood diseases, among which iron deficiency anemia most often occurs [7,9].

This fact, according to many scientists [9, 22, 9] is due to the fact that Helicobacter pylori refers to the virulent agent that absorbs and uses a significant amount of iron for its vital activity and can compete with the host for the content of iron reserves. [13].

Helicobacter pylori, like other gram-negative bacteria, enter into complex competitive interactions for iron. For this purpose, a phenolate or hydrosomatic type siderophore synthesized by him is used, later it is combined with siderophile ferrate with subsequent extraction of iron from the cell surface. Direct lysis of cells is possible under the influence of urease and leucinase produced by Helicobacter pylori with the extraction of iron of the macroorganism (human), digestion of hemoglobin under conditions of assimilation of heme with the formation of sideroforms that allow the extraction of iron of the macroorganism (patient). Moreover, the process of absorption and assimilation of iron may be disrupted due to an increase in intragastric pH in common variants of Helicobacter pylori-associated gastritis. With hypochlorhydria, the process of iron oxidation is disrupted, which is necessary for further absorption. This iron absorption disorder in Helicobacter pylori-infected patients is also evidenced by experimental studies [11].

The first example of an association between Helicobacter pylori infection and J was the description of a 15-year-old girl admitted to the hospital for the treatment of anemia, whose examination revealed Helicobacter pylori-associated chronic active gastritis, after which, without the additional use of iron preparations, normalization of hematological parameters was achieved [6, 7]. Later, a number of descriptions of cases of anemia that are poorly treatable with iron preparations appeared in patients who do not have signs of gastrointestinal blood loss and do not complain about the pathology of the gastrointestinal tract (gastrointestinal tract). Nevertheless, Helicobacter pylori-associated gastritis was detected in all the described patients during the examination, and after eradication of Helicobacter pylori, a cure for IDA was achieved. The first data were obtained in children and adolescents [8-12], later similar results were described in adults [13]. [2]. Numerous data indicate that H. pylori infection, like any other, is not limited only to local effects on the gastric mucosa, but is also capable of showing systemic effects (inflammatory, autoimmune, allergic, etc.), causing appropriate reactions from some organs and systems. Helicobacter pylori may be related to the development of such extra-gastric pathological conditions as vascular diseases (atherosclerosis, coronary heart disease, Raynaud's syndrome), autoimmune diseases and syndromes (autoimmune thyroiditis, rheumatoid arthritis, Sjogren's syndrome), blood diseases (anemia, thrombocytopenic purpura), various skin diseases. There is also evidence of the role of Helicobacter pylori in infertility, Parkinson's disease, bronchial asthma, bronchitis, glaucoma, headache, delayed physical development in children, food allergies, etc. [7, 8, 9, 21].

Of particular interest is the involvement of Helicobacter pylori infection in the development of iron deficiency conditions and iron deficiency anemia (IDA), which are more characteristic of the child population. Anemia, according to the World Health Organization (WHO), affects about 2 billion people in the world. Approximately 50% of all cases of anemia are iron deficiency anemia [18, 23]. This is the most common nutritional deficiency in both developing and developed countries of the world.

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It has been proven that IDA has a negative impact on the performance and health of patients, leads to disorders in the immune system, cognitive and reproductive functions, and also affects the overall mortality rate from IDA in most developing and developed countries [21].

In childhood, IDA accounts for 90% of all anemia [8], and in adults up to 80%. IDA can be called a socially significant disease. The prevalence of IDA in children aged 2.5 years in Nigeria is 56%, in Russia - 24.7%, in Sweden - 7%. According to WHO experts, if the prevalence of IDA exceeds 40%, then this problem ceases to be medical and requires decision-making at the state level. [3]. According to the Ministry of Health of the Russian Federation, the incidence of anemia among the children's population of our country remains high, without any downward trend, despite all the measures taken. In 2005, the rate of anemia patients per 100,000 children was 2971.5, and in 2009 [4].

As is known, the cause of IDA is insufficient iron intake, chronic blood loss, malabsorption, hemolysis or a combination of these conditions [21, 22, 23]. Recently, among the possible causes of IDA, the involvement of Helicobacter pylori infection has been discussed [14,19,20]. The presence of an association between Helicobacter pylori infection and a decrease in iron content in the blood is confirmed by many studies [16, 17]. In Germany, it was found that Helicobacter pylor infection is associated with a decrease in serum ferritin concentration by 17% (according to age and gender) [13]. In Denmark, low ferritin in persons seropositive to Helicobacter pylori was less in 40% of cases. The association between low ferritin levels and Helicobacter pylori infection in children has been established regardless of the type of strain, age, gender, or their iron intake [8]. The dependence of Helicobacter pylori infection with iron deficiency has differences in different age groups. Thus, low ferritin levels in infected, the most common [21]. Low iron content is more pronounced in girls compared to boys [22]. The existing relationship is confirmed by the normalization of iron metabolism after eradication without additional administration of iron-containing drugs [15].

The negative impact of Helicobacter pylori infection on the development of iron deficiency is of particular relevance in childhood and adolescence. And if at an early age the main cause of IDA is nutritional iron deficiency, then in schoolchildren and adolescents, the spectrum of risk factors becomes much wider [11]. Such causes that contribute to the development of iron deficiency in adults as colorectal cancer, gastroectomy or copious menstruation are not typical for children, therefore, Helicobacter pylori infection is considered as the main cause of iron deficiency, especially in cases of anemia refractory to therapy. Infection with Helicobacter pylori among children increases with age, adolescence is critical, and especially girls [16, 21]. There is evidence of a close association of Helicobacter pylori with low serum ferritin levels and erythropoiesis deficiency in adolescent girls. [5]. It has been shown that by the age of 9, the infection rate in children with anemia is 4 times higher. There is no such close relationship among the adult population, suggesting the presence of other causes in the development of their iron deficiency. At the same time, there are isolated publications in the literature where the association of Helicobacter pylori infection and iron deficiency is not confirmed [41]. In a study conducted in schoolchildren aged 10 to 15 years [15], there was a 2 times higher infection rate of Helicobacter pylori (31.1%) among children with IDA compared with children with normal hematological indicators (15.5%). Similar results were obtained in adolescents, and the prevalence of Helicobacter pylori-associated IDA was 2 times higher in athletes (5.5%) than in other adolescents (2.3%) [16], which is associated with a higher. On the other hand, the duration of Helicobacter pylori infection probably matters for the formation of J, so the longer the child is infected, which means the older he is, the more likely the depletion of iron reserves and the development of IDA.

Studies devoted to evaluating the results of IDA treatment in Helicobacter pylori-infected children [18, 19] have shown that eradication of Helicobacter pylori leads to both an increase in ferritin levels, restoration of iron reserves, and normalization of hemoglobin levels. Moreover, eradication of Helicobacter pylori is effective both in combination with taking iron preparations and without them [20], in contrast, prescribing only iron preparations turned out to be ineffective.

How does Helicobacter pylori infection affect iron metabolism and its reserves in the body? The process of absorption and assimilation of iron can be disrupted due to a decrease in the content of ascorbic acid in the stomach, as well as an increase in intragastric pH in common variants of Helicobacter pylori. In hypochlorhydria, the process of iron oxidation is disrupted, which is necessary for its further absorption [21]. Oxidation is facilitated in the presence of ascorbic acid, the content of which is significantly reduced against the background of chronic inflammation in the coolant, it depends on the activity of inflammation.

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Date of Publication: 20-03-2023

There may be several mechanisms that explain the pathogenic connection of infection and a decrease in iron metabolism. One of these potential mechanisms explaining the loss of iron in Helicobacter pylori infection is secondary to erosive gastritis blood loss, including erosive gastritis. But in the overwhelming number of publications, this hypothesis does not find its confirmation based on the data of endoscopic examination and examination of feces for hidden blood [19]. Another explanation is a decrease in iron absorption due to reduced acidity of gastric juice. Absorption of iron in the gastrointestinal tract occurs at the level of the duodenum and jejunum. Iron must be in a certain form (heme) in order to be available for absorption by epithelial cells. 80% of iron in the diet consists of (Nonheme) nonheme forms (vegetables, cereals, rice.), requiring certain conditions for absorption. This is the presence of both hydrochloric and important ascorbic acid [19]. It was found that the concentration of ascorbic acid decreases against the background of chronic inflammation in the gastric mucosa (SOJ), depends on the activity of inflammation, and on the presence of CagA (+) Helicobacter pylori. Its level is restored after successful eradication of Helicobacter pylori [7,8,11]. The mechanism of competitive absorption of iron, which is necessary for the vital activity of the bacterium itself, deserves attention. The microorganism's need for iron is explained by its use for growth and vital activity, and, therefore, Helicobacter pylori is able to compete with the host. A study with labeled iron showed that in the presence of Helicobacter pylori, iron deviates from the bone marrow for the needs of the microorganism [12]. The regulation of iron intake of Helicobacter pylori is partially different from other bacteria. In Helicobacter pylori, some iron-consuming systems are genetically more active, which improves adaptation in the human stomach, where there is often both an excess and a lack of iron. Iron consumption is normally regulated by a special protein (ferric uptake regulator - Fur), but in Helicobacter pylori this protein is mutant, so the consumption of iron by the microorganism does not decrease even with its excess. The proteome of Helicobacter pylori strains isolated from patients with and without IDA differs. This indicates that the polymorphism of Helicobacter pylori strains may be one of the determinants affecting the consumption of iron reserves in the host organism. The main method of consumption of Helicobacter pylori iron is lactoferrin COOLANT, the level of which in COOLANT is increased in patients with Helicobacter pylori infection with IDA, but not increased in the absence of bacteria. Even with a lack of iron in the stomach, the growth of Helicobacter pylori is supported by the consumption of lactoferrin. After eradication of Helicobacter pylori, the level of lactoferrin in the COOLANT normalizes. It is assumed that the presence of the napA gene in the bacterium allows it to sequester lactoferrinization of COOLANT. It is also assumed that the pfr gene of Helicobacter pylori, which determines the consumption of ferritin, and the feoB gene, which plays the role of an iron transporter, are involved [1]. In 2008, K.Muhsen and D.Cohen [14] published the results of a meta-analysis of 19 epidemiological studies, 6 experimental papers and 12 series of clinical observations conducted in different regions of the world devoted to the study of the relationship of Helicobacter pylori infection, J and IDA. The meta-analysis of the association of Helicobacter pylori and IDA included 7 studies; the calculation of the total indicators showed a twofold increase in the risk of IDA in Helicobacter pylori-positive patients compared with Helicobacter pylori–negative (relative risk - HR =2.0; $p<0.001$), statistical differences between the results of the studies were not revealed. The association of Helicobacter pylori and J was analyzed in a set of 9 studies, a decrease in ferritin levels (<15 mcg/l) was significantly more common (HR=1.6; $p<0.01$) in Hp-infected patients. In a study conducted in schoolchildren aged 10 to 15 years [15], there was a twofold greater HP infection (31.1%) among children with IDA compared with children with normal hematological indicators (15.5%). Similar results were obtained in adolescents, and the prevalence of Helicobacter pylori-associated IDA was 2 times higher in athletes (5.5%) than in other adolescents (2.3%) [16], which is associated with a higher need for iron in athletes. J.Seo et al. [17] showed that in In children aged 6-12 years, J (ferritin <15 mcg/l) is 5.6 times more common in Helicobacter pylori-infected (13.9%) than in uninfected (2.8%). In children under 10 years of age, the average hemoglobin level in the presence of Helicobacter pylori was significantly lower (124 g/l) than in its absence (131 g/l) [18]. On the one hand, this can be explained by the fact that a number of other negative factors contributing to the development of IDA are possible at an early age. On the other hand, the duration of Helicobacter pylori infection probably matters for the formation of J. The longer the child is infected, which means that the older he is, the more likely the depletion of iron reserves and the development of IDA.

ISSN NO: 2770-0003
Date of Publication: 20-03-2023

Studies on the evaluation of the results of IDA treatment in Helicobacter pylori-infected children [18, 19] have shown that eradication of Helicobacter pylori leads both to an increase in ferritin levels, i.e., the restoration of iron reserves, and to normalization of hemoglobin levels. Moreover, eradication of Helicobacter pylori was effective both in combination with taking iron preparations and without them [20], however, the administration of iron preparations alone proved ineffective. How does Helicobacter pylori infection affect iron metabolism and its reserves in the body? The process of absorption and assimilation of iron can be disrupted due to a decrease in the content of ascorbic acid in the stomach, as well as an increase in intragastric pH in common variants of Helicobacter pylori-associated gastritis. With hypochlorhydria, the process of iron oxidation is disrupted, which is necessary for its further absorption [21]. Oxidation is facilitated in the presence of ascorbic acid, the content of which is significantly reduced against the background of chronic inflammation in the gastric mucosa (SOJ), depends on the activity of inflammation and is more pronounced in CagA (+) Helicobacter pylori [22]. The level of ascorbic acid is restored after successful eradication of Helicobacter pylori [23]. C. Ciacci et al. [24] revealed a violation of iron absorption in an experiment in Helicobacter pylori-infected patients.

A decrease in iron reserves in the body during Helicobacter pylori infection may be a consequence of increased consumption of it by Helicobacter pylori itself, since iron is necessary for the microorganism to grow and it is able to compete with the host for the creation of iron reserves [15]. A study with labeled iron conducted by A.Barabino et al. [10] showed that in the presence of Helicobacter pylori, iron deviates from the bone marrow for the needs of the microorganism. The regulation of iron intake of Helicobacter pylori is partially different from other bacteria. In Helicobacter pylori, some iron-consuming systems are genetically more active, which improves adaptation in the human stomach, where there is often both an excess and a lack of iron. Iron consumption is normally regulated by a special protein (ferricup take regulator – Fur), but in Helicobacter pylori this protein is mutant, so the consumption of iron by the microorganism does not decrease even with its excess [16]. The proteome of Helicobacter pylori strains isolated from patients with and without IDA differs [17]. This indicates that the polymorphism of Helicobacter pylori strains may be one of the determinants affecting the consumption of iron reserves in the host body. The main method of consumption of Helicobacter pylori iron is lactoferrin COOLANT, the level of which in COOLANT is increased in patients with Helicobacter pylori infection with IDA, but not increased in the absence of Helicobacter pylori. Even with a lack of iron in the stomach, the growth of Helicobacter pylori is supported by the consumption of lactoferrin [18]. After eradication of Helicobacter pylori, the level of lactoferrin in the COOLANT normalizes. One of the possible explanations for the development of J in patients with Helicobacter pylori-associated gastroduodenal diseases is latent blood loss. The cause of it may be erosive and ulcerative lesions. Thus, latent blood loss in Helicobacter pylori-associated gastritis has been described in Alaska Eskimos [14]. However, in most of the above studies, the sources of bleeding were excluded based on the data of endoscopic examination and examination of feces for hidden blood.

Summing up the analysis of the results of studies on the relationship between Helicobacter pylori infection and IDA, there is every reason to conclude that due to the peculiarities of the metabolism of Helicobacter pylori, even in the absence of causes of blood loss, leads to impaired absorption and a decrease in iron reserves in the body. At the same time, Helicobacter pylori-associated gastritis is not always clinically manifest, the patient may not make any gastroenterological complaints. Therefore, in accordance with the recommendations of the Maastricht-3 consensus, in the presence of IDA of unclear etiology, the patient should be examined to exclude Helicobacter pylori infection. Convincing data confirming the effectiveness of eradication therapy in the treatment of IDA allow us to recommend the eradication of Helicobacter pylori even in the absence of gastroenterological complaints and low activity of chronic Helicobacter pylori-associated gastritis in a patient with IDA.

Due to the production of a number of enzymes, Helicobacter pylori is able to have an immunosuppressive effect, affect the differentiation of T-lymphocytes and inhibit phagocytic activity [7, 17, 19]. Thus, the presence of the common properties of Helicobacter pylori with the components of the gastric mucosa makes it possible to identify this microbe also as an inducer of autoimmune reactions. Three possible mechanisms leading to diseases associated with immune disorders are suggested: Helicobacter pylori interact with mast cells, initiating the release of mediators;

— Helicobacter pylori, acting as full-fledged antigens, cause allergic reactions in the host body;

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Date of Publication: 20-03-2023

—Helicobacter pylori 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 of Helicobacter pylori, an abnormally high level of Ig E occurs. When the allergen interacts with Ig E, mast cells are activated with the release of allergy mediators (tryptase, histamine, platelet activation 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 between the degree of infection with Helicobacter pylori and the severity of dermatitis has been proven. With a high degree of Helicobacter pylori contamination, the recurrent course of atopic dermatitis becomes continuous, in the absence of bacteria after the eradication of Helicobacter pylori, the severity of dermatitis is minimal and patients with a continuous course of the disease are not detected.

To maintain its existence, Helicobacter pylori needs iron. Helicobacter pylori refers to the virulent agent that absorbs and uses a significant amount of iron for its vital activity [17, 19, 22]. Helicobacter pylori, like other gram-negative bacteria, enter into complex competitive relationships for iron. For this purpose, the siderophenolate or hydroxomate type synthesized by him is used, then it is combined with siderophile ferrate with subsequent 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 of the macroorganism (human), digestion of hemoglobin and assimilation (assimilation) of heme with the formation of siderophores that allow the extraction of iron of the macroorganism.

L. Dhaenensetal. [12] compared the need for iron among 4 varieties of Helicobacter persisting in the gastric mucosa (H. pylori, H. felis, H. acinonyx, H. mustelae) and 5 varieties of Helicobacter colonizing the intestinal tract (H. fennelliae, H. cinaedi, H. muridarum, H. bilis, H. hepaticus). It was revealed that gastric species of Helicobacter, with the exception of Helicobacter pylori, which uses iron from human lactoferrin, receive iron for their vital activity from heme and hemoglobin. Other varieties of Helicobacter pylori detected 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 found in Helicobacter pylori to use human lactoferrin as a source of iron determines the special virulence of Helicobacter infection [11].

Helicobacter pylori-positive patients have lower levels of serum ferritin and iron compared to Helicobacter pylori-negative patients [9, 10], and in patients with atrophic gastritis associated with Helicobacter pylori infection, these indicators of the "iron" status were the lowest [2].

The presence of Helicobacter pylori on the gastric mucosa contributes to the development of iron deficiency anemia in adults and children [4-6]. The presence of Helicobacter pylori infection in combination with iron deficiency anemia is more often accompanied by damage to the entire gastric mucosa and the development of pangastritis [7].

Oral ferrotherapy restores and maintains normal iron levels, but after its termination, anemia returns again. M. Konnoetal. [9] proved that the traditionally used correction of iron deficiency and the resulting iron deficiency anemia in helicobacter infection is unjustified. C. Hershko, A. Lahad, D. Kereth [6] believe that iron deficiency anemia favorably affects the destruction of Helicobacter pylori.

The introduction of an excessive amount of iron (both with food and with medications) affects the severity of the infectious process, reduces the overall resistance of the macroorganism. The presence of exogenous iron leads to increased reproduction of Helicobacter pylori. Therefore, in the treatment of patients with iron deficiency anemia associated with Helicobacter pylori, it is recommended to use eradication therapy with the inclusion of a proton pump inhibitor and 2 antibacterial drugs for 2 weeks [5, 6] without additional administration of jelly preparations

Conclusions:

1. The problem of etiopathogenesis, diagnosis, clinic, treatment of children with chronic gastritis, gastroduodenitis, gastric ulcer, duodenal ulcer is relevant, as the number of children with chronic diseases of the gastroduodenal zone is steadily increasing.
2. The course of chronic disease of the gastroduodenal zone has its own characteristics. This is important when choosing further treatment tactics.
3. Chronic gastroduodenitis is rarely a monozabolism.

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Date of Publication: 20-03-2023

4. In children with pathology of the gastroduodenal zone, the features of the clinic, vegetative status and frequency of family burden were revealed.

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