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Helicobacter Pylori And its Importance in the Development of Anemia Associated With Iron Deficiency and Vitamin B12

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Abstract: In recent years, there are reports that Helicobacter pylori (H. Pylori), which causes diseases of the gastroduodenal area, can also cause extragastroduodenal diseases. They mention the occurrence of hematological, metabolic, cardiovascular, neurodegenerative and allergic diseases under the influence of H. Pylori. Although some studies confirm the existence of an organic relationship between H. Pylori infection and anemia, there is relatively less information about its association with extragastroduodenal diseases, in particular, anemia. Although infection with H. pylori and iron and vitamin B12 deficiency anemia are widespread among the population in all regions of our Republic, no scientific observations have been made regarding their interaction and similar approaches to treatment. However, it is of great scientific and practical importance to study and create principles of a new approach to the treatment of iron and vitamin B12 anemia associated with H. Pylori, taking into account such situations as the area where the population lives, its sanitary and hygienic conditions, and geographical location.

Keywords: H. Pylori, anemia, iron-deficiency, vitamin B12.

In the world, including in Uzbekistan, gastrointestinal diseases and anemia are common diseases among the population. In recent years, in a number of cases, there is information that H. Pylori, which causes gastroduodenal (chronic gastritis, gastric and duodenal ulcer disease, etc.) diseases, also causes extragastroduodenal diseases. They mention the occurrence of hematological, metabolic, cardiovascular, neurodegenerative and allergic diseases under the influence of H. Pylori. Also, various mechanisms of the development of the pathological process, including induction of molecular and low-activity inflammatory response, have been shown [6, 9]. H. Pylori belongs to the gram-negative bacillus group and is the most common bacterium in the world. Helicobacter pylori is detected in about 50% of citizens in developed countries, and 90% in developing countries. According to scientific observations, this Gram-negative bacillus is most often found among people living on the African continent [11, 16]. The prevalence of this bacterium among the population of Latin America is close to that of African countries [8, 14]. In Western European countries and Australia, the infection is relatively rare, occurring in 30-40% of the population. In the population of the Russian Federation, infection is detected in 50-80% of the population, and its indicators differ somewhat in different regions of the country [7]. Helicobacter pylori is widespread in our country, and 80% of those diagnosed with gastrointestinal diseases are diagnosed with positive strains of Helicobacter pylori. Among the regions, this bacterium was found the most in Khorezm region (79%) and the least in the population of Tashkent city (60%) [3]. H. pylori is not only distributed in different countries of the world, but ethnic, geographical factors, genetic predisposition, sanitary and hygienic conditions in the region and family (taking into account the faecal-oral transmission of infection), confirm that the composition of the food eaten is important. Scientific observations show that high salt content in food increases the oncogenic potential of H. pylori by increasing the expression of SagA protein [12].

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Persistence of bacteria in the gastric mucosa for a long time, in addition to inflammatory processes, participates in the formation of atrophic changes, causes intestinal metaplasia, and eventually develops cancer. Among the diseases caused by extragastroduodenal effects of H. Pylori, anemia has a special place. Because they, especially iron deficiency anemia, are common in our country as well as in the rest of the world. In the case of deficiency of macro- and microelements in the human body or their increase, resistance to adverse environmental factors decreases, immune deficiency states are formed, the activity of antioxidant defense systems is disturbed, diseases become chronic, and the risk of development of common ones increases, the quality of life deteriorates, and the effectiveness of treatment measures [4]. One of such vital microelements in the body is iron. Its deficiency is an important medical and social problem, and, as mentioned above, it is the cause of the development of a common disease such as iron deficiency anemia. In such cases, protein and mineral metabolism is disturbed, which leads to functional and morphological changes in all organs. Iron is considered an irreplaceable trace element in the human body, it is part of hemoglobin, myoglobin, cytochromes, participates in a number of oxidation-reduction reactions, plays an important role in blood formation processes. An adult human body contains on average about 3-4 g of iron (about 40 mg Fe/kg body weight in women and about 50 mg Fe/kg body weight in men). A large part of this trace element (60%, or more than 2 g) is in hemoglobin, about 9% - in myoglobin, about 1% - in heme and non-heme enzymes. 25-30% of iron is deposited in the reserve in association with ferritin protein and hemosiderin [1, 13, 10]. This trace element is taken into the human body with food and is mainly absorbed in the duodenum. Cases of iron deficiency in the body are observed in about half of the world's population. According to the estimates of the World Health Organization, about 4 billion people suffer from various degrees of iron deficiency, which is more than 60% of the population of the Earth. About 2 billion of them account for iron deficiency anemia. This shows that it takes the leading place (90%) among anemias, which are considered one of the most common diseases in the world [20]. As mentioned above, existing H. Pylori infection in the body and the inflammatory processes caused by it lead to iron absorption disorders. It is known that this bacterium activates pro-inflammatory cytokines (interleukin-6, α-tumor necrosis factor). They, in turn, increase the synthesis of hepcidin, which controls iron absorption. As a result, iron absorption is blocked at the level of enterocytes and the reserve in macrophages increases. H. pylori uses some iron for its activity as a virulent agent in addition to this mechanism that causes iron deficiency anemia. Like other Gram-negative bacteria, this microorganism enters into a complex competition for iron. For this purpose, a synthetic siderophore of the phenolate or hydroxomate type is used, which then combines with the ferrate siderophile, causing iron to migrate from the cell surface. Perhaps, due to the effect of urease and mucinase produced by H. Pylori, cell dissolution (lysis) is observed and provides an opportunity to extract iron from the human body. The combination of iron deficiency anemia and H. Pylori in most cases leads to the damage of the entire surface of the gastric mucosa, that is, pangastritis. As a result of oral ferrotherapy, the amount of iron in the body is restored, but after it is stopped, anemia occurs again. About 20 mg of iron enters the body when a person eats normally, and 1-2 mg of it is absorbed. The association between iron deficiency anemia and H. pylori was first published in 1991 by Blecker et al. They reported that a 15-year-old girl with hemorrhagic gastritis caused by H. Pylori resolved anemia due to anemia with the help of anti-infective eradication therapy without the use of iron preparations. The relationship between vitamin B12 deficiency and H. Pylori infection was determined in 1984 by O. Connor and co-authors. The authors found Campylobacter-like organisms in type A gastritis and in patients with pernicious anemia. A number of other studies have also found a link between H. Pylori and anemia. But there is relatively little information on the association of this infection with extragastroduodenal diseases, in particular with anemia. Results of a meta-analysis of 15 studies on the association between H. pylori and iron deficiency anemia were published by Qu and co-authors [17]. In it, as a result of endoscopic and histological examination, 5 cases without gastric peptic ulcer and cancer were selected. In another 10 cases, the presence of H. Pylori was confirmed using a serological test and a urease breath test. The obtained data showed a high risk of iron deficiency anemia in patients with H. Pylori infection (odds ratio 2.2; confidence interval 1.5-3.2). Also, Yuan and co-authors performed a meta-analysis of 16 randomized controlled trials (n=956) [19]. It investigated the effectiveness of H. Pylori eradication therapy in iron deficiency anemia. Eight experiments involved

patients without peptic ulcer and H. pylori was detected by rapid urease test or histological examination. In the remaining patients, the presence of infection was recorded using a breath urease test. The duration of follow-up ranged from 1 to 3 months. Hemoglobin, iron in blood serum, ferritin were statistically significantly different from each other in the dynamics of treatment in patients who received antihelicobacter and iron preparations. Based on a series of observations, the Maastricht-4 recommendation states that all patients with a positive H. Pylori test and iron deficiency should undergo antibacterial eradication procedures [14]. Sarari and co-authors observed that 67.4% of patients with H. Pylori had symptoms of vitamin B12 deficiency [18]. Shuval-Sudai and co-authors reported a higher prevalence of H. Pylori in patients with low serum vitamin B12 levels. In addition, no similar international observations have been conducted confirming the effectiveness of antihelicobacter therapy in the treatment of vitamin B12 deficiency anemia.

Analysis of the studies confirms that there is a link between H. Pylori infection and anemia. In addition, the mechanisms of interaction between iron deficiency anemia and H. Pylori remain unknown in most cases. In particular, why do not all patients with H. Pylori infection develop iron deficiency anemia? According to some researchers, H. Pylori needs iron in the host for its proliferation and growth. Some strains of bacteria have the ability to disrupt iron metabolism and add it to their outer membrane. They show that the CagA strain, as well as its mutation, can be highly virulent in terms of iron uptake. It is in patients infected with the CagA strain that obvious atrophic changes of the gastric mucosa are observed, accompanied by a decrease in the secretion of its juice, an increase in intragastric pH and gastrin in the serum. Some of the controversy in the treatment of iron deficiency anemia associated with H. pylori may be due to the different distribution of bacterial strains in different geographic areas. In one of the recent meta-analyses, eradication therapy followed by iron supplementation was found to be more effective in Asia-Pacific regions than in North America [19]. Although infection with H. pylori and iron and vitamin B12 deficiency anemia are widespread among the population in all regions of our Republic, no scientific observations have been made regarding their interaction and similar approaches to treatment. However, it is not only scientific, but also of great practical importance to create new principles of study and treatment of iron and vitamin B12 anemia associated with H. Pylori, taking into account such situations as the area where the population lives, its sanitary and hygienic conditions, and geographical location.

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