iJRASET
International Journal For Research in Applied Science and Engineering Technology

INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 9 **Issue:** XII **Month of publication:** December 2021

DOI: https://doi.org/10.22214/ijraset.2021.39492

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International Journal for Research in Applied Science & Engineering Technology (IJRASET)
ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.429
Volume 9 Issue XII Dec 2021- Available at www.ijraset.com

Significance of Helicobacter Pylori in Iron Deficiency

Maxmonov Lutfulla Saydullaevich[1], Mamatkulova Feruza Khaydarovna[2], Shomurodov Qodir Ergashevich[3], Temirov Nuriddin Najmiddinovich[4]
[1]*Head of the Department of Hematology, Samarkand Medical Institute*
[2, 3,] *4Assistant of the Department of Hematology, Samarkand Medical Institute*

Abstract: This article discusses the importance of helicobacter pylori in iron deficiency. It is well known that iron deficiency is one of the major problems in medicine today. The following article examines the complications of this disease and the effective methods used to treat it.
Keywords: anemia, Helicobacter pylori, blood, pathology, gastrointestinal tract, microorganism.

I. INTRODUCTION

Iron deficiency (IR) is the leading cause of anemia in the world. JJ negatively affects the health and performance of both adults and adolescents, increasing the risk of developing various diseases. One of the microorganisms that can provoke the development and aggravate the manifestation of ID is the bacterium Helicobacter pylori (Hp). Helicobacter is the only currently known microorganism capable of not only survive in the acidic and anaerobic stomach contents, but also alter the absorption of iron. The first publications on the relationship between Hp and ID infection appeared in the 19906s. Initially, these reports were regarded as paradoxical and improbable, but a number of descriptions of cases of curing anemia in patients infected with Hp and did not have any pathology of the gastrointestinal tract forced scientists to study this problem more closely.

II. MAIN PART

Initially, the effect of Hp infection on the development of ID was studied in children of different ages. The results of the study by J. Seo (2002), carried out with the participation of 753 Korean schoolchildren aged 6-12 years, are widely known. It turned out that children infected with Hp more often suffered from ID than uninfected children (13.9 and 2.8%, respectively), while the concentration of ferritin in the blood serum of Hp seropositive patients significantly exceeded that of Hp sero negative ($p < 0.001$). After adjusting for the age and socioeconomic status of children, J. Seo et al. confirmed the previously obtained result (odds ratio – OR – 5.6; 95% confidence interval (CI) 1.0 30.6) and concluded that Hp infection provokes a decrease in the level of ferritin in the blood serum; the possibility of subsequently developing jelly-deficiency anemia (IDA).

Similar data were obtained from a survey of 700 children aged 7 to 10 years living in rural areas in Alaska (N. Baggett, 2006). In the surveyed cohort, 86% of patients were infected with Hp, 38% of respondents suffered from ID, 7.8% of participants had signs of IDA. Scientists recorded a high Hp infection in children with ID - 91%, while the latter was more often diagnosed in schoolchildren living in private homes with □6 family members, and Hp positive children were more likely to develop IDA than Hp negative ones.

The relationship between HP infection and the development of anemia was recorded not only in children of different ages, but also in pregnant women. German scientists analyzed the results of observation of 898 pregnant women and compared the hemoglobin (Hb) level in those infected with Hp and in patients without antibodies to Hp. M. Weyermann et al. (2005) found a negative effect of Hp infection on Hb concentration: pregnant women infected with Hp had lower Hb levels than Hp negative women (0.25 g / dL; 95% CI 0.49 to 0.003); Hp seropositive pregnant women showed a tendency to a significant decrease in Hb concentration during pregnancy (0.14 g / dL; 95% CI 0.38 to 0.10). The main risk factor for the infection of the infant with Hp was recognized as the low socioeconomic status of the parents, and not the infection of the mother (O. Suoglu, 2007). The best protection against infection by O. Suoglu et al. consider exclusive breastfeeding of an infant during the first 4 months of life. Some studies have failed to confirm the relationship between development DZh and infection with Nr. For example, M. Akcam (2007) did not record a significant decrease in the level of folic acid, zinc in children infected with Hp, but there was a tendency to an increase in serum ferritin concentration ($p = 0.09$) and a significant decrease in vitamin B12 ($p = 0.04$). After the publication of the results of a large-scale epidemiological study conducted by American scientists under the guidance of V. Cardenas (2006), the relationship between the

development of ID and Hp infection was recognized as obvious. Observation of 7,462 respondents made it possible to establish that Hp infection provokes a decrease in serum ferritin concentration (percentage of change: 13.9; 95% CI from 19.5 to 8.0), but does not affect the percentage of saturation of trans ferrin and the content of Hb (percent change: 2.8 and 1.1, respectively). Researchers recorded a relationship between the presence of Hp infection and the prevalence of IDA (OR 2.5; 95% CI 1.5 4.6), the development of anemia itself (OR 1.3; 95% CI 1.0 1.7) and 40% an increase in the prevalence of ID (OR 1.4; 95% CI 0.9 2.0). The work of V. Cardenas (2006) was the first large study that convincingly proved the development of ID / IDA in persons infected with Hp, regardless of the presence or absence of peptic ulcer disease.

Pathogenetic mechanisms Having confirmed the connection between Hp infection and the development of ID, scientists attempted to establish the mechanisms of the influence of Hp on iron metabolism. and its reserves in the human body. Currently, there are several theories explaining this phenomenon. The most widespread hypothesis is the competitive absorption of alimentary iron Hp, according to which this microorganism is able to assimilate iron supplied from food, and thereby significantly increase the need of the macroorganism for iron. This theory is supported by the results of a study by P. Doig, who proved the presence of a special iron-binding protein in the HP bacterium, which has a structural similarity to human ferritin and is capable of binding to the heme gland of erythrocytes. In addition, Hp has another interesting ability: the receptors of the outer membrane of these microorganisms are able to capture and use iron contained in human lactoferrin and iron-binding glycoprotein (M. Husson, 2007). Another theory explaining the development of ID upon Hp infection is the hypothesis of the mediated blockade of hepcidin synthesis (a protein that plays a dominant role in the process of iron absorption in the duodenum) in hepatocytes (R. Pellicano, 2004). An opinion was expressed about the role of ascorbic acid deficiency in gastric juice that develops upon infection with Hp. Researchers recorded a decrease in the concentration of ascorbic acid in gastric juice in patients infected with Hp, while the greatest drop in this indicator was recorded in persons who were carriers of cytotoxic Hp strains with the CagA gene (A. Barabino, 2002). The mechanisms by which Hp affects the level of ascorbic acid in gastric juice are not fully understood. Scientists explain this fact by increased oxidation of ascorbic acid or a violation of its bioavailability in the presence of Hp (Nahon, 2003; Yoshimura, 2003); others associate it with the onset of hypoacidity of gastric juice on condition of the development of Hp associated chronic atrophic gastritis (Byrd, 2002). Proponents of the genetic theory of ID formation upon Hp infection argue that Hp are carriers of specific genes (in particular, pfr and feoB genes responsible for ferritin consumption and iron transport) that allow microorganisms to assimilate alimentary iron. The followers of this hypothesis have established that the appearance in Hp of a specific mutation in the fur gene regulating iron consumption leads to a progressive uptake of iron, even in spite of its excess in Hp tissues.

After studying the possible pathogenic mechanisms of the appearance of ID, scientists tried to reveal the relationship between the degree of Hp contamination of the gastric mucosa and the activity of the inflammatory process. It was suggested that the presence of asymptomatic Hp carriage does not affect iron absorption and cannot provoke ID, let alone IDA (D. Mahalanabis, 2005). However, the implementation of eradication therapy, regardless of the degree of infection activity, promoted the restoration of the Hb level and the concentration of serum iron even without additional administration of iron-containing drugs (A. Kurekci, 2005).

Modern research data Over the past three years, a large number of trials have been carried out that continued to study the effect of Hp on iron metabolism and began to study the features of the course and treatment of ID or IDA in patients infected with Hp. In some of these works, the role of Hp in the development of JJ was confirmed, in others it was completely refuted. The prevalence of Hp infection (70.7%) and anemia (20.6%) in the surveyed cohort of respondents (I. Santos, 2009). After adjusting for the patient's sex and age, skin color, smoking habits, the Brazilian scientists also did not find the effect of Hp infection on the Hb concentration (mean difference 0.07 g / dl; p = 0.4). Similar results were obtained in another study conducted in Brazil with the participation of 194 adolescents aged 10-16 years (L. Araf, 2010).

The median serum ferritin concentration and mean Hb values in individuals infected with Hp did not differ from those in Hp negative patients (33.6 and 35.1 ng / ml, respectively; 13.83 ± 1.02 and 14.0 ± 1.06 g / dl). The lack of a relationship between Hp infection and the development of ID was recorded by Iranian scientists in a study of primary school children. The normal content of ferritin in blood serum was recorded in 296 (25%) of the examined patients, despite the presence of antibodies to Hp, while a low concentration of ferritin was found in 29% of schoolchildren infected with Hp (A. Zamani, 2011). In contrast to the above data, the work of A. Fraser, K. Muhsen, H. Duque confirmed the relationship between HP infection and the development of ID. Investigating the prevalence of Hp infection in ethnic groups living in New Zealand, A. Fraser et al. (2010) registered a significant decrease in the saturation of transferrin with iron (p = 0.013) in individuals infected with Hp. After adjusting for age and ethnicity, scientists found a high probability of developing ID (RR 1.20; 95% CI 1.08 1.34), but not IDA (RR 1.01; 95% CI 0.87 1, eighteen) in Hp seropositive patients.

95% CI 13.38 to 0.011; p = 0.04) between Hp positive and Hp negative schoolchildren, and such changes were not observed in young children. Another study showed a positive effect of Hp eradication on the iron content and Hb concentration. Examining a cohort of children with ID or anemia (n = 72) and conducting eradication therapy in patients infected with Hp (n = 38), Mexican scientists found that effective eradication of Hp was accompanied by an increase in Hb level by 0.37 g / dL (95% CI 0.02 to 0.75). H. Duque et al. (2010) found that the addition of ferrous sulfate to anti-Helicobacter pylori therapy increased the Hb concentration by 0.47 g / dL (95% CI 0.01 0.93) in comparison with children uninfected with Hp and taking only drugs. leza. The administration of ferrous sulfate to patients uninfected with Hp led to an increase in serum ferritin concentration by 11.26 ng / ml (95% CI 1.86 20.65) compared to with children taking placebo.

An interesting feature should be noted: the overwhelming majority of trials, which did not record the effect of Hp infection on the formation of ID, were carried out in Latin America (Argentina, Bolivia, Mexico, Brazil, Cuba, Venezuela), while studies in which this dependence was confirmed, were carried out in the countries of Europe, Asia, or the United States. Some recent studies were devoted to the analysis of the level of soluble transferrin receptor (sTfR) - the only serum marker reflecting the need for iron in tissues participating in erythropoiesis (the diagnostic value of sTfR is also due to the fact that its concentration in blood serum does not depend on the active inflammation). Estonian scientists have investigated the effect of Hp on iron metabolism in schoolchildren, using the determination of the sTfR level to diagnose ID (sTfR> 5.7 mg / L in children aged 7-12 years, sTfR> 4.5 mg / L in adolescents) and setting the Hb concentration for diagnostics of anemia (Hb <115g / l in children 7-12 years old, Hb <130g / l in boys, Hb <120g / l in girls). After examining 363 participants, N. Vendt et al. (2011) revealed ID in 17%, anemia in 5% of schoolchildren, while antibodies to Hp were found in 27%, IgA antibodies to tissue transglutaminase (specific for celiac disease) - in 0.6% of children. Teenagers were more likely to suffer ID (29 vs 23%, respectively) and were infected with Hp than younger children (22 and 9%, respectively), the risk of developing ID was also higher in older schoolchildren (relative risk — RR — 1.1; 95% CI 1.0 1.3; p = 0.03). Proa Having analyzed the obtained data, the scientists came to the conclusion that the true risk factor for developing ID is the child's age, and not Hp infection. At the same time, the work of Irish specialists, carried out under the guidance of M. Hoseinzadeh (2010), demonstrated completely opposite results. In the surveyed In a cohort of 100 newborns, researchers recorded a significant relationship between sTfR concentration, iron content, ferritin level, and the presence of high titers of IgG to Hp (p <0.001); an association was also recorded between a burdened family history of gastrointestinal tract diseases and the level of IgG antibodies to Hp (p <0.001). The results of a recently published trial by Korean scientists, led by S. Lee (2010), confirmed R. Pellica no's hypothesis on the effect of Hp on hepcidin levels. Determination of the dynamics of the serum concentration of prohepcidin (hepcidin precursor protein) before and after the eradication of Hp made it possible to establish a significant decrease in its indicator in patients taking anti-Helicobacter pylori therapy and iron preparations (p = 0.01); at the same time, the rate of decrease in the pro-hepcidin concentration in patients receiving combined treatment (anti-Helicobacter drugs and iron) did not differ from that in patients receiving standard anti-Helicobacter therapy (p = 0.894). Eradication therapy with the simultaneous intake of iron contributed not only to a decrease in the level of prohepcidin, but also to the leveling of IDA phenomena (S. Lee, 2010).

Treatment The unambiguous results of all 5 meta-analyzes were so convincing that certain changes were made to the latest recommendations for the treatment of IDA, published in the online version of the journal Gut on July 6, 2011. At present, the following item is included in the standard of examination of IDA patients: "With the development of refractory IDA and the absence of pathological changes in the gastrointestinal tract, according to the data of upper and lower endoscopy, patients infected with Hp are shown eradication therapy."

The same guidelines regulate the specifics of iron replacement therapy: "To correct anemia and replenish iron stores in the body, all patients are shown the appointment of iron preparations." A. Goddard et al. (2011) note that the simplest and most accessible way to stop IDA is to administer 200 mg of ferrous sulfate 2 r / day; a decrease in the daily dosage of ferrous sulfate or the appointment of other ferrous compounds (ferrous fumarate, ferrous gluconate) or dosage forms (suspensions) is possible only with poor patient tolerance of standard doses of ferrous sulfate. For the treatment of JJ in patients infected with Hp, as a rule, the administration of oral iron is sufficient, however, if the latter are intolerant, the use of parenteral forms is possible. The intake of iron-containing drugs should be continued for another 3 months after the level of the clinical manifestations of ID. The provisions of the new manual provide for the possibility of prescribing ascorbic acid (250 500 mg 2 r / day) to improve iron absorption, however, A. Goddard et al. note the lack of convincing data on the effectiveness of ascorbic acid in the treatment of IDA. However, taking into account the pathogenetic features of ID development during Hp infection (lack of ascorbic acid), it is very likely that the appointment of ascorbic acid to patients with Hp associated IDA is justified.

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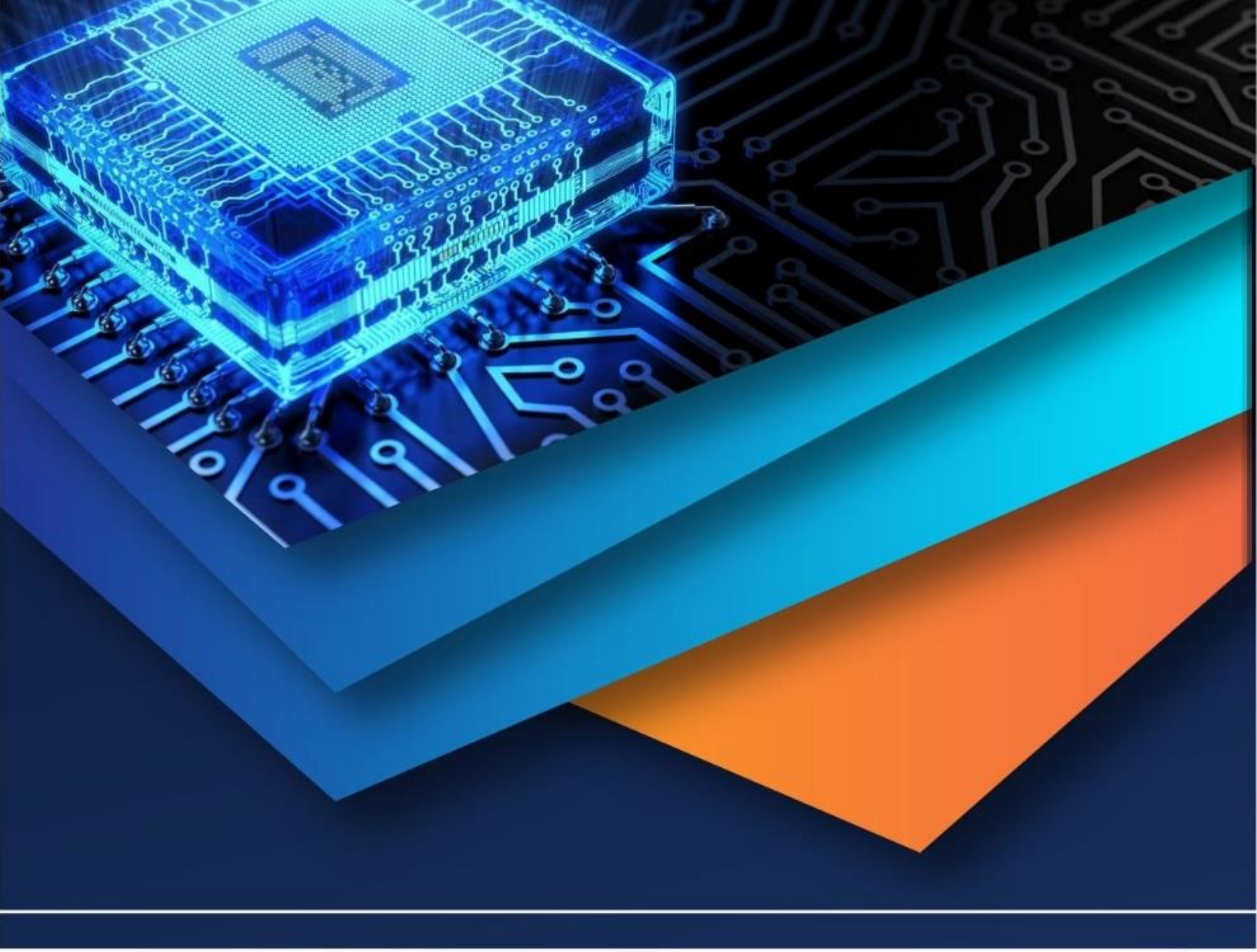

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