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The Main Criteria for The Effectiveness of Treatment of Iron Deficiency Anemia Associated with Helicobacter Pylori

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Abstract: ***This article discusses the main criteria for the effectiveness of treatment of iron deficiency anemia associated with Helicobacter pylori. New methods used in its treatment will be discussed, the problems encountered in this process and methods of treatment will be discussed.***
Keywords: treatment, deficiency anemia, Helicobacter pylori, health system, etiological factors.

I. INTRODUCTION

Nowadays no one doubts the fact that the bacterium Helicobacter pylori (H. pylori) is involved in the development of chronic gastritis, peptic ulcer disease, stomach maltoma and adenocarcinoma. In other words, infection with Helicobacter pylori can cause a wide range of various pathological processes - from inflammation and ulcers to malignant tumors. At the same time, in recent years, the number of works on the undoubted role of Helicobacteria in the defeat of organs not related to the digestive system has been increasing. It was found that H. pylori and the damage to the gastroduodenal zone caused by them not only coexist with a number of diseases, but the latter may precede, be pathogenetically related or not dependent on H. pylori. As a result of numerous studies, it has been established that the range of extradigestive manifestations of H. pylori infection is unusually wide - from vascular, autoimmune and skin lesions to such as Parkinson's disease, Bekhcher, migraine, glaucoma and uveitis, celiac disease, food allergy, iron deficiency anemia, and even impaired reproductive function. Despite the differences in the pathogenesis of the above diseases, successful eradication therapy promotes their partial or even complete remission. Many researchers associate the development of extragastric manifestations of infection with the influence of H. pylori virulence factors, mediators of their action, the immune status of the macroorganism, the effect of proinflammatory cytokines, peptides, hemostatic factors, heat shock proteins, lipid peroxidation products, etc.

Let us dwell in more detail on the effect of infection. H. pylori on the development of iron deficiency anemia (IDA). The problem of iron deficiency is so urgent that WHO, together with UNISEF, made a statement on the need for effective comprehensive control of anemia. It called on the heads of national health systems to promote the development and implementation of a set of geographically adapted measures aimed at reducing the prevalence of anemia by at least a third by 2010. Today, the problem of studying the etiological factors of the development of IDA remains relevant. This is due to the fact that in more than 30% of cases, the cause of the development of IDA cannot be identified, and, therefore, it is not possible to choose an adequate therapy. Among the probable reasons for the development of IDA of unclear etiology, H. pylori is increasingly called. There are many reports in the literature that after successful eradication in individuals infected with H. pylori, the symptoms of IDA refractory to iron therapy disappear [7, 12].

First evidence of a possible causal role H. pylori in the onset of IDA was published in 1993 by C. Dufour et al. Described a case of disappearance of symptoms of refractory IDA of unknown etiology in a seven-year-old boy after successful treatment of H. pylori associated gastritis [25]. This report was followed by a number of publications on the development of refractory IDA without any etiological factors in individuals with H. pylori associated gastritis and on the normalization of ferrostatus after successful eradication therapy, without additional intake of iron-containing drugs ... The largest number of such cases is described by B. Annibale et al. [4]. They analyzed the results of observation of 30 patients infected with H. pylori with symptoms of refractory, unexplained IDA. All of them underwent eradication therapy. After 12 months, red blood counts returned to normal in persons with successful eradication (iron preparations were not included in the course of treatment). In patients after unsuccessful eradication therapy, red blood counts changed little.

Epidemiological studies have shown that the level of serum iron in patients infected with H. pylori is significantly lower than in non-infected patients. The largest study of this process was carried out in Denmark (n = 2794), Germany (n = 1806) [9] and the USA (n = 1806). Possible mechanisms by which H. pylori is involved in the onset and development of the iron deficiency state and has not been studied. However, there are a number of theories trying to explain this phenomenon. Here are the most significant new facts on the problem under discussion.

Some strains of H. pylori are capable of absorbing alimentary iron, competing with the host and reflecting the struggle of the macro and microorganisms for the availability of the iron source [3]. However, until now, none of the researchers has been able to confirm the role of specific strains of H. pylori in the development of IDA. H. pylori infection increases the body's iron requirements, as some of the nutritional iron is used to “meet the needs” of the infection itself. Unlike other bacteria, which use oxidized iron (Fe3 +) as a source [12], reduced iron is the main source for H. pylori. This fact is the result of the adaptation of H. pylori to a specific habitat. Under the influence of a low pH value, ferric iron in the stomach is oxidized to a bivalent form that is bioavailable for humans. This adaptive mechanism allows H. pylori bacteria to assimilate iron, which is a necessary growth factor for H. pylori, as well as for other bacteria [4]. P. Doig et al suggested that the bacterium H. pylori contains an iron-binding protein that binds to heme iron in erythrocytes, and is similar in structure and function to human ferritin. It is for this reason that it competes with human ferritin in the process of iron metabolism. In an in vitro study, M.O. Husson et al. Found that the outer membrane receptors of the H. pylori bacterium are capable of capturing and using for the growth of human iron lactoferrin and ferum-binding glycoprotein produced by neutrophils of the gastric mucosa [3]. In an in vivo study conducted by Y. Choe et al., It was shown that the level of lactoferrin in the gastric mucosa is significantly higher in H. pylori positive individuals with IDA than in H. pylori positive individuals without anemia, in H. pylori negative with IDA and in H. pylori negative without signs of IDA [15].

Another hypothesis was put forward, somewhat different from the previous ones, according to which it was assumed that the mutation in the pfr gene of H. pylori is associated with the development of IDA. But Y. Choe and co-authors, who put forward this hypothesis, failed to find facts that would confirm it. Here is another possible mechanism of influence H. pylori on iron absorption proposed by R. Pel licano et al. In those infected with H. pylori, the production of hepcidin by hepatocytes decreases in response to an increase in interleukin 6 production [7]. In addition, interleukin increases the concentration of the acute phase protein, α1 antitrypsin, which inhibits erythropoiesis by disrupting the binding of ferritin to trans ferrin receptors and internalizing the transferrin receptor – transferrin complex [13]. Interesting results were obtained by L. Keenan and co-workers when studying the effect of H. pylori on iron metabolism in mice. It was found that with sufficient reserves of iron, Helicobacter pylori infection does not cause iron deficiency, while in the presence of iron deficiency in the body, it aggravates it to the point of anemia.

Another possible and most studied mechanism of the effect of H. pylori on the absorption of alimentary iron is ascorbic acid (AA) of gastric juice. This mechanism, in contrast to those described above, reflects the indirect effect of H. pylori on the bioavailability of iron. AA is released into gastric juice by active secretion from blood plasma. About 80% of iron, which enters the body with food, is in an oxidized state, has a low bioavailability, forms large, easily aggregating complexes with OH–, as well as with other anions and water. The release of iron from these compounds and its reduction to a bivalent, bioavailable form is facilitated by low pH and AA values of gastric juice, which is the most important "enhancer" for the absorption of non-heme iron. This fact even served as the basis for the intriguing hypothesis that iron, like vitamin B12, has its own intrinsic factor, which is AA. According to some studies, H. pylori infection decreases the concentration of AA in gastric juice, and this effect is more pronounced when infected with cytotoxic CagA positive strains of H. pylori [7]. In contrast to mammals, which synthesize AA in the liver from glucose, humans do not have a specific enzyme, halo nolactone oxidase, and the need for AA is satisfied only through the alimentary route.

The mechanisms by which H. pylori affects the level of AA in gastric juice are not fully understood. There are several hypotheses. First, it is assumed that the decrease in the AA level is a consequence of increased AA oxidation under the direct influence of H. pylori . Secondly, this fact is associated with impaired bioavailability of AA in individuals infected with H. pylorii [11], and thirdly, the mechanism is mediated by the influence of hypoacidity of gastric juice, which occurs during the development of atrophic changes in the gastric mucosa. At a gastric pH of more than 4, AA is oxidized to a biologically inactive form [13].

Analysis of data on the interaction of H. pylori infection with the development of IDA indicates that not everything is known about the true nature of the interaction between H. pylori and the development of an iron deficiency state. It is necessary to decipher the specific mechanisms of this process, as well as to develop tactics for the treatment of iron deficiency in those infected with H. pylori. And in this sense, it is difficult to disagree with the conclusion of M. Blaser, who, summarizing all the ambiguity of the problem of H. pylori infection, said: “... anyone who is looking for simple answers to the question of how H. pylori causes the development of various H. pylori associated diseases will undoubtedly be disappointed. The complexity of this problem is probably even older than humanity itself ”[10].

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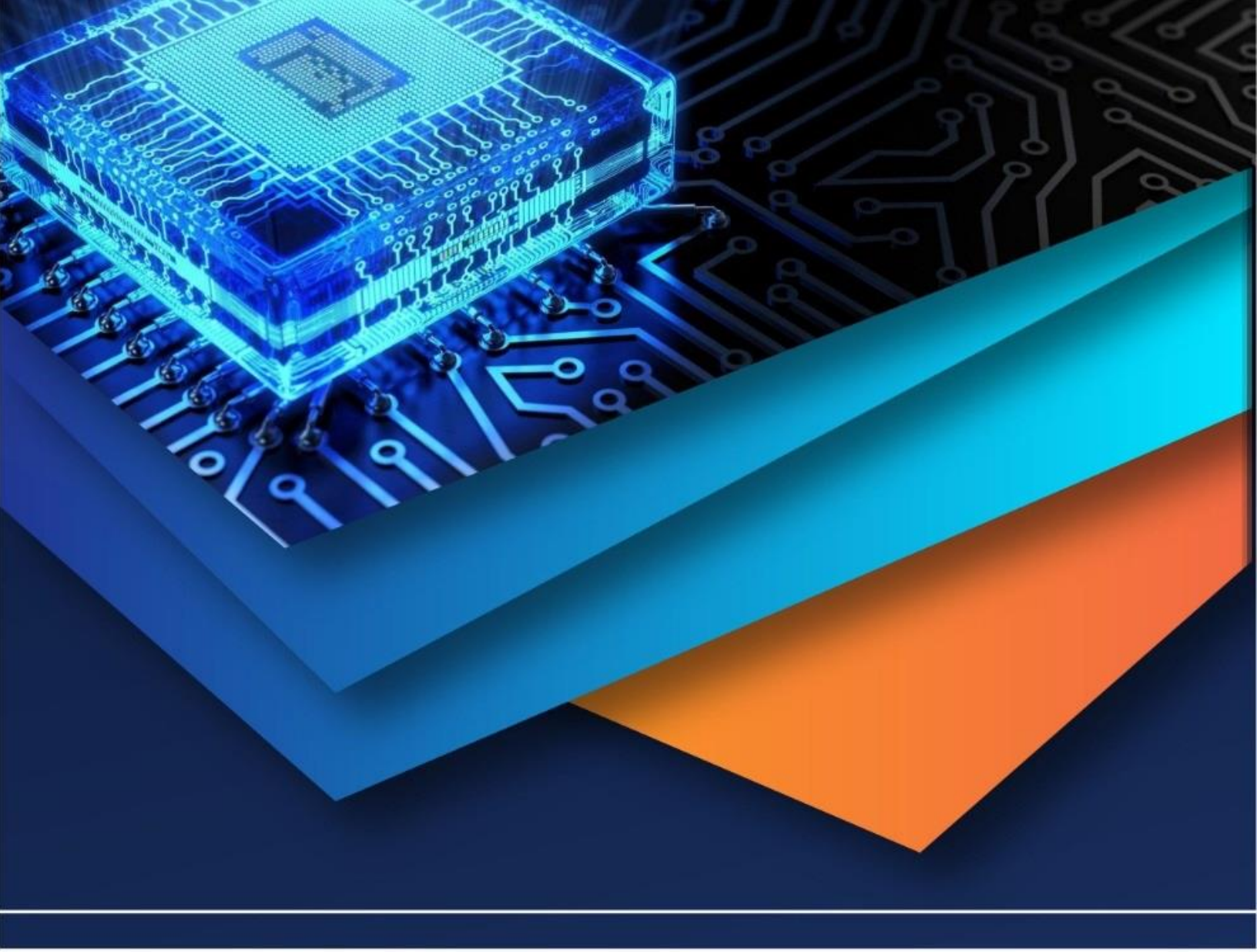

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