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The Role of Helicobacter Infection in the Development of Functional Dyspepsia and Inflammatory Periodontal Diseases

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Abstract: This article analyzes the provisions of the Maastricht Consensus-5 on the study of H. pylori infection. It is shown how the ideas about the diagnosis and treatment of Helicobacter pylori infection have changed in the previous Maastricht consensuses. The issues of the relationship of this infection with the pathology of the gastro duodenal zone, including functional dyspepsia and inflammatory diseases of the oral mucosa and periodontium, are considered separately. Modern approaches to the diagnosis of helicobacteriosis are shown with the determination of the choice of the most optimal diagnostic method in various situations. The importance of H. pylori eradication in terms of preventing functional dyspepsia, inflammatory diseases of the oral mucosa and periodontium has been confirmed.

Key words: H. pylori infection; diagnostics; treatment; prevention of functional dyspepsia and inflammatory periodontal diseases; Maastricht Consensus-5.

Functional dyspepsia (FD) is a widespread functional disease of the gastrointestinal tract (GIT), characterized by a heterogeneous pattern of clinical manifestations and a significant negative impact on the quality of life of patients [1, 2]. According to the Rome criteria of the 3rd revision (2006), the term FD is understood as the presence of one or more symptoms in a patient - pain or burning in the epigastrium, a feeling of fullness in the epigastrium or early satiety in the absence of data on organic pathology that can explain these symptoms [3]. Currently, the etiopathogenesis of FD appears to be a complex multifactorial process, the causal relationships of which continue to be actively studied. It is most likely that a combination of a number of physiological, genetic, environmental and psychological factors in a particular patient leads to the development of certain combinations of sensory-motor disorders of the gastrointestinal tract and, as a result, to the manifestation of symptoms of dyspepsia [2, 8, 10]. Data on the involvement of Helicobacter pylori (H. Pylori) infection in the etiology of FD are ambiguous and contradictory. According to various sources, the microorganism is detected in 39–87% of patients with FD and, as a rule, twice as often as in controls [11, 12]. In the 90s of the last century, specialists dealing with the problem of helicobacteriosis tried to streamline the accumulated knowledge in the field of studying H. pylori infection and formulate general rules, general directions in the diagnosis and treatment of helicobacter pylori infection. The European Helicobacter pylori Study Group, founded in 1987, made great efforts in this direction. called the Maastricht Consensus. In 2000, on the basis of further progress in the study of Helicobacter pylori infection, the Maastricht Consensus-2 was adopted. The third Maastricht consensus (2006) continued the same diagnostic and treatment strategy as in Maastricht II. In 2010, the fourth Maastricht consensus was discussed and adopted. In October 2016, a new Maastricht Consensus - the fifth (Management of Helicobacter pylori infection - the Maastricht V/Florence Consensus Report) was published in the Gut journal [5]. The first statement of the consensus indicates that H. pylori gastritis is an infectious disease regardless of the presence of symptoms and complications. Thus, the provisions expressed in the recent Kyoto Consensus (2015) [6] are confirmed here. H. pylori cause chronic active gastritis in all

infected patients. Maastricht 5 discusses the relationship between H. pylori infection and gastric secretion. With regard to the diagnosis of H. pylori infection, the Maastricht Consensus-5 indicates that the 13C-urease breath test is the most researched and recommended test in the context of the test and treat strategy [9].

Of interest and practical significance are the data that H. pylori is more often detected in FD with epigastric pain syndrome and less frequently in FD with postprandial distress syndrome [11, 12]. The evolution of views on the role of helicobacteriosis in the pathogenesis of FD has gone from the idea of bacteria as a commensal to recognizing it as one of the pathogen etic factors of this disease. It has been established that in H. pylori-positive patients with FD, disturbances in the motor function of the stomach and duodenum (in particular, weakening of the motility of the antrum, slowing down evacuation from the stomach) are more pronounced than in H. pylori-negative patients [13]. It is believed that H. pylori infection of the gastric mucosa leads to the development of chronic H. pylori gastritis, which in some patients is accompanied by a dysfunction of the pacemaker of the stomach, insufficient fundic relaxation, and expansion of the antrum of the stomach with a weakening of its postprandial motility. In other words, the development of FD symptoms should be considered as a consequence of long-term chronic inflammation in the gastric mucosa induced by H. pylori infection, which leads to impaired motility and visceral sensitivity of the gastro duodenal zone [14]. The inhibitory effect of H. pylori on gastric motility involves cytokines (IL-1β, IL-6, IL-8) and tumor necrosis factor-alpha (TNF-α), the production of which is enhanced by the presence of Helicobacter pylori infection. . The relationship between the clinical manifestations of FD and the presence of H. pylori in the gastric mucosa in such patients has been studied for a long time. It has been noted that in H. pylori-positive patients, the clinical symptoms of FD are more diverse than in H. pylori-negative individuals [16, 17]. In addition, in patients with FD, a correlation was found between the severity of pain in the epigastric region and the presence of H. pylori in the gastric mucosa. Recent studies have established that the oral cavity can serve as a reservoir of H. pylori infection in the human body, and the persistence of Helicobacter in periodontal pockets, saliva and plaque is associated with the presence of H. pylori in the mucous membrane of the stomach and duodenum in a number of gastro duodenal diseases, including PD [18]. There is a report on the presence of H. pylori in the dental plaque of patients treated for stomach diseases [19], after a course of antibiotic therapy, microorganisms disappeared from the stomach, but were detected in dental plaque. The authors concluded that plaque is the main reservoir of H. pylori infection, especially in developing countries. There are other reports on the detection of H. pylori in plaque in the absence of microorganisms in the stomach [20], which allows us to consider plaque not only as an important reservoir of H. pylori infection, but also as a place of its colonization in the human body. Thus, most researchers are inclined to think about the possibility of the existence in the oral cavity of a permanent source of self-infection and reinfection after successful eradication of H. pylori from the stomach [21]. According to a number of authors, there is a significant correlation between the urease activity of gingival pocket biopsies and stomach biopsies ($r = +0.39$, $p < 0.05$), as well as between the constant carriage of H. pylori infection in the gingival pockets of the oral cavity and the degree the severity of morphological changes in the gastric mucosa ($r = +0.52$, $p = 0.05$) in patients with H. pylori-associated gastrointestinal diseases [22]. At the same time, works devoted to the study of H. pylori status of the oral cavity in patients with FD are extremely few and contradictory. Most likely, this can be explained by the fact that diagnostic methods traditionally used to detect these bacteria in the stomach are used to detect H. pylori infection in the oral cavity, while only some of them are reliable enough to detect helicobacteriosis in the oral cavity. Most often, a biochemical method (rapid urease test) and a polymerase chain reaction (PCR) method using primers specific for fragments of various H. pylori genes (ureA, ureB, ureC, cagA) are used to detect H. pylori infection in the oral cavity. The frequency of occurrence of a microorganism in dental plaque by PCR, according to different authors, ranges from 3 to 84.4% and reaches its maximum value when using a large number (8-12) of samples obtained from one person [20]. A simpler method for diagnosing H. pylori is a biochemical one (rapid urease test). The frequency of occurrence of a positive urease test when plaque is used as a substrate, according to various

researchers [23-26], ranges from 79 to 100%. A number of studies note the presence of a statistically significant correlation between the degree of contamination of H. pylori in dental plaque (cytological method) and the gastric mucosa (histological method), as well as the practical coincidence of the positive results of the urease test (43-100%) and the frequency of detection of H. pylori histological method and PCR (43-88%) in plaque. In this regard, some authors [27] consider the biochemical method as a screening method, which makes it possible to identify not only permanent carriers of infection in the oral cavity, but also patients with H. pylori-associated diseases of the gastrointestinal tract, including patients with FD.

Materials and methods. In the clinic of therapeutic dentistry of the Bukhara State Medical Institute in 2020, we conducted a study that assessed the H. pylori status of the oral cavity in 78 patients with FD and 30 healthy individuals in 2 stages. Initially, all patients were tested using a rapid urease test with Christensen's medium. At the second stage, if the result of the biochemical diagnosis of H. pylori was positive, all patients were tested by PCR

Results and discussion. The results of the study showed that, according to the rapid urease test, H. pylori in the oral cavity was found in 78.53% of patients with FD and 46.67% of healthy individuals. At the same time, verification of these results by PCR diagnostics showed a significantly lower frequency of detection of H. pylori infection. In patients with H. pylori FD, PCR was detected in 42.42% of cases, in the control group - in 30.4% of the examined. Statistical analysis showed that the difference between the frequency of detection of H. pylori- in the oral cavity of patients with FD and healthy individuals, according to PCR diagnostics, was statistically significant. In addition, the study assessed the frequency of detection of extra-gastritic symptoms of FD (fibromyalgic syndrome) depending on the H. pylori infection of the oral cavity. An analysis of the incidence of fibromyalgia symptoms in patients with FD depending on the infection of the oral cavity with Helicobacter pylori using a nonparametric chi-square test showed that none of the obligate or additional symptoms of fibromyalgia syndrome was associated with a positive H. pylori status of the oral cavity. At the same time, the frequency of the leading clinical symptoms of FD correlated with oral helicobacteriosis, which indicates the need to study the H. pylori status of the oral cavity in this category of patients.

In view of the above circumstances, most likely, antihelicobacteria can be useful in patients with FD, provided that H. pylori infection is detected. Indeed, a number of studies have shown that successful eradication of H. pylori in most patients with FD leads to a significant improvement and even complete disappearance of dyspeptic complaints, normalization of the secretory and motor functions of the stomach. It was also noted that this therapy did not lead to the normalization of the motor function of the stomach. [7]. In this regard, among practitioners in recent years there has been a strong opinion that the first line of treatment in patients with FD, regardless of H. pylori infection, should be acid-lowering drugs or prokinetics. This approach did not provide for mandatory testing of patients for the presence of H. pylori infection. Only in case of failure of empirical therapy with ant secretory drugs or prokinetics, H. pylori testing were performed, and in case of H. pylori infection, such patients were recommended eradication of H. pylori infection as a second line of treatment. As the third line of treatment, antidepressants, numerous herbal remedies, various psychological effects and non-traditional methods of treatment were offered. . As a result, a small but statistically significant efficacy in the eradication of H. pylori infection was recorded (in 36% of patients) compared with placebo (in 30% of patients), while the relative risk reduction was 9% at a 95% confidence interval. There was no statistically significant heterogeneity or asymmetry between studies. The data obtained led to the conclusion that H. pylori infection eradication is clinically effective in H. pylori-positive patients with FD [4].

Conclusions

1. Data from recent systematic reviews indicate that the eradication of H. pylori infection in FD also has a moderate but significant clinical effect on symptoms. According to the decision of the Maastricht Agreement-5, in establishing the diagnosis of FD, in all cases, the “test and treat” strategy (examine and treat) with the help of a respiratory urease test should be used -

and if it is detected, a course of anti-Helicobacter pylori therapy with eradication control should be carried out.

2. The currently accumulated data give grounds to consider H. pylori, detected in most patients with FD, as a possible etiological factor in the occurrence of this pathology, causing dysmotility of the stomach and duodenum, as well as changes in visceral sensitivity.
3. Eradication of H. pylori infection may be useful in individuals with a reliable diagnosis of FD and is a mandatory medical intervention, provided that bacteria are detected, since it not only leads to a stable regression of the symptoms of the disease, but also reduces the risk of developing inflammatory diseases of the oral cavity and periodontium.

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