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On the Issue of Helicobacter Pylori–Associated Gastritis of the Gastric Stump and its Treatment Strategy

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Annotation: This article discusses the pathogenesis, clinical features, and treatment approaches of Helicobacter pylori–associated gastric stump gastritis. The persistence of H. pylori infection after gastric surgery plays a significant role in chronic inflammation and mucosal atrophy. Timely diagnosis and the use of targeted anti-Helicobacter therapy are crucial to prevent disease progression and postoperative complications. The study emphasizes the need for individualized therapeutic strategies based on microbial resistance patterns and patient-specific factors.

Keywords: Helicobacter pylori, gastric stump gastritis, anti-Helicobacter therapy.

In recent years, significant progress has been achieved in gastroenterology, accompanied by changes in the understanding of the **etiological factors and pathogenetic mechanisms** of digestive system diseases, leading to substantial advancements in their **diagnosis and treatment.** Despite these achievements, chronic inflammatory diseases of the gastrointestinal tract remain among the most common pathologies in the adult population. This issue is also highly relevant in our country, as the incidence of gastroenterological diseases has increased by **53% over the past decade** [18].

The introduction of experimental research findings into clinical practice and the improvement of diagnostic techniques have contributed to notable progress in addressing many problems of modern gastroenterology. New functional and morphological methods have broadened the understanding of **etiological and pathogenetic aspects** of gastrointestinal diseases. For a long time, these factors were considered to include **dietary, allergic, genetic, neuropsychological, and immunological disorders,** as well as **discoordination of gastric and duodenal motility.**

The discovery of *Helicobacter pylori* (*H. pylori*) and its crucial role in the **pathogenesis of**

chronic gastritis, peptic ulcer disease, and gastric cancer, made by Australian scientists **B. Marshall and I. Warren in 1983,** represented a true revolution in the understanding of the etiology of digestive system diseases. This discovery paved the way for the development of **new and more effective pathogenetic principles of treatment and prevention** in gastroenterology.

Since the discovery of *H. pylori*, more than two decades have passed, during which numerous studies have been published on the problems of **Helicobacter infection** [27]. Nevertheless, despite extensive research, new aspects of *H. pylori*'s pathogenic influence on various mechanisms of **gastroduodenal pathology** continue to emerge. Recently, increasing attention has been paid to the role of *H. pylori* in the development of **post-gastrectomy and post-vagotomy complications** of inflammatory, dystrophic, and dysplastic nature [5,7,8,12,16,28,29,38]. However, the results of published epidemiological studies remain inconsistent. The exact relationship between *H. pylori* and **gastric cancer,** as well as the intermediate pathogenetic stages leading from precancerous mucosal changes to carcinoma, remain unclear [4,12,31,39].

H. pylori infection induces an **acute inflammatory response** in the gastric mucosa of most infected individuals. In a subset of patients with persistent infection, **active chronic gastritis** develops, leading to mucosal atrophy and consequently increasing the risk of **adenoma, carcinoma, and gastric MALT lymphoma** [2,23].

Based on global research, **P. Correa** proposed in 1988 the paradigm of **gastric carcinogenesis,** now known as the **Correa cascade.** According to this model, the progression of chronic gastritis results in the development of **atrophy and intestinal metaplasia** of the gastric mucosa. The metaplastic epithelium then undergoes further **genetic and phenotypic alterations,** resulting in **dysplasia,** which may progress to invasive carcinoma. Subsequently, Correa modified his model to include *H. pylori* as the initiating factor in this specific sequence of pathological processes [5,7,12,39].

Although this model is well-established and widely recognized, many questions remain regarding the **diagnostic identification of the critical stages** in the cascade of gastric epithelial atrophy and dysplasia. Therefore, identifying patients who have undergone **gastric resection** and require **long-term surveillance** for the early detection of preneoplastic changes in the gastric mucosa has become increasingly important. Despite this, the factors contributing to **inflammatory and atrophic processes** in the mucosa of the operated stomach remain **insufficiently studied** to date [1,13].

The history of gastric surgery has evolved in such a way that **gastrectomy** has become virtually the only widely used operative method for gastric pathology. Despite the large number of operations performed, it is now possible to critically evaluate this intervention in light of its long-term consequences. Undoubtedly, gastrectomy performed according to strict indications results in the lowest rate of functional disorders. However, the issue of **post-gastrectomy pathology** remains highly significant today, as a substantial number of patients continue to suffer from various syndromes requiring treatment, despite the decline in the frequency of both elective and emergency surgeries.

Most patients with this pathology undergo **long-term outpatient examinations and treatments** for diverse symptoms and diagnoses, which often leads to missed opportunities for timely diagnosis and adequate therapy. Many authors [1,15,21,25,26] emphasize that an **individualized treatment approach** should be based on an objective analysis of **clinical and pathogenetic features** that determine disease development and prognosis in each patient. Such an approach directs researchers toward studying **individual characteristics** influencing disease progression and developing **methods of differential diagnosis and treatment** in accordance with the modern understanding of the **clinical and pathogenetic heterogeneity** of the condition [12].

At the same time, among **general practitioners and local physicians,** attitudes toward

previously operated patients remain ambiguous [13,22,30]. It is well known that in the vast majority of patients after gastrectomy, **chronic gastritis of the gastric stump** develops relatively quickly [1,13,23,26,29]. It is generally accepted that this results from the **removal of the antral part,** leading to a sharp decrease in adequate stimulation of the **fundic glands** [24]. The **atrophy of the gastric stump mucosa** progresses more intensively in middle-aged and elderly patients and is directly proportional to the time elapsed since surgery [10,17].

According to the literature [5], **90% of chronic gastritis cases** are caused by *Helicobacter pylori* (*H. pylori*) infection, and **atrophic gastritis** is not an inevitable outcome of mucosal aging but is determined specifically by bacterial infection. The activity of the **inflammatory process** in the distal stomach is closely associated with the bacterial load in the gastric mucosa [20,36]. Based on these findings, a reasonable assumption arises regarding the association between *H. pylori* infection and **inflammatory, atrophic, and dysregenerative changes** in the mucosa of the operated gastric stump [13].

Several authors [7,22] have identified a **synergistic effect** between *Helicobacter* infection and **bile reflux,** which induces **reflux gastritis,** suggesting an **increased risk of neoplastic transformation** in the gastric stump mucosa. In this regard, it is becoming essential to **identify patients after gastrectomy** who require **regular dynamic monitoring** for the early detection of such mucosal changes [3,5,7,15,31,32].

In **1994,** experts from the **International Agency for Research on Cancer (IARC, WHO)** classified *Helicobacter pylori* infection as a **Group I carcinogen** for humans. It was established that the **risk of gastric cancer (adenocarcinoma)** in patients infected with this bacterium increases **threefold** [3,4,11,12,31]. This is particularly important considering that, according to statistical data, **around 1 million people die annually** from gastric cancer worldwide [31]. Moreover, data regarding the relationship between *H. pylori* infection rate, the degree of mucosal colonization, and the **morphological changes** in the gastric stump mucosa remain **limited and contradictory** [13,21,22,32].

The recurrent course of post-gastrectomy syndromes, the presence of severe complications, and the resulting temporary disability and early invalidity of patients highlight not only the medical but also the socio-economic significance of this problem [14]. A systematic examination of patients who have undergone gastric surgery, along with efforts aimed at early prevention and detection, can lead to significant improvements in treatment outcomes [1,26].

Modern therapy for Helicobacter infection in the unoperated stomach consists of oral combinations of various antibacterial drugs. Despite the undeniable success of H. pylori eradication therapy, the currently used triple or quadruple regimens cannot be considered fully optimal [6,9,10,18,19,35,37]. The results of many years of research indicate that adequate antibacterial therapy, by eradicating H. pylori, leads to a reduction or complete elimination of chronic inflammation in the gastric mucosa, as well as a decrease in dystrophic, atrophic, and dysregenerative changes [10,37,40].

Therefore, the question arises regarding the use of anti-Helicobacter therapy for the gastric stump mucosa to reduce dysregenerative changes, which in turn may improve the clinical course of the disease and alter the clinico-morphological characteristics of the gastric stump mucosa [10,37]. This approach would allow the development of a prognostic algorithm, determine indications for eradication therapy in patients after gastrectomy for peptic ulcer disease, and enable individualized rehabilitation programs, thereby improving the treatment outcomes of patients with post-gastrectomy syndromes.

Currently, available data indicate that adequate antibacterial therapy, by eradicating H. pylori, results in a reduction of chronic inflammation in the gastric mucosa [38,22]. This provides a strong rationale for recommending anti-Helicobacter therapy in patients after surgical treatment of peptic ulcer disease. The presence of H. pylori infection and the detection of inflammatory

and dystrophic changes in the mucosa of the resected stomach make it reasonable to propose eradication therapy for this category of patients. However, this recommendation often remains underutilized [1,14], as the role of H. pylori in the development of dysregenerative changes in the mucosa of the resected stomach is still insufficiently studied [13,19].

Conclusion: Helicobacter pylori infection plays a crucial role in the development and progression of chronic gastritis, including gastritis of the gastric stump after gastrectomy. Despite significant progress in gastroenterology, the mechanisms underlying post-gastrectomy inflammatory and atrophic processes remain insufficiently studied. The persistent presence of H. pylori, along with factors such as bile reflux and mucosal atrophy, contributes to chronic inflammation, dysregeneration, and an increased risk of neoplastic transformation in the operated stomach. The implementation of anti-Helicobacter eradication therapy in patients after gastric resection can significantly reduce mucosal inflammation and degenerative changes, improving both clinical outcomes and long-term prognosis. Therefore, early detection of H. pylori infection, regular endoscopic and histological monitoring, and the application of individualized eradication and rehabilitation programs should be considered essential components in the management of patients with post-gastrectomy syndromes. Continued clinical and experimental research is necessary to further clarify the pathogenetic mechanisms and optimize therapeutic strategies for this category of patients.

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