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# CLINICAL AND PATHOGENETIC CHARACTERISTICS OF PEPTIC ULCER OF THE STOMACH AND DUODENUM

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**Abstract:** In order to assess the clinical, pathogenetic, functional variant of the course of peptic ulcer disease, I conducted a study of 112 patients with gastric ulcer (34), duodenal ulcer (78). The diagnosis was verified in accordance with the requirements of the modern standard, with an assessment of histomorphological secretory motor, hormonal disorders, as well as the degree of N. rulori contamination, eradication and its effectiveness. Psychosomatic status was assessed using complex methods: multilateral personality studies, the Hamilton scale for assessing depression, the Spielberg scale of situational and personal anxiety, the Beck depression scale, the "unfinished sentences" method for assessing violations in the relationship system.

**Keywords:** peptic ulcer, motor evacuation function of the stomach and duodenum, duodenostasis.



## Introduction

Peptic ulcer disease is a chronic recurrent disease that occurs with alternating periods of exacerbation and remission, in which, as a result of violations of the nervous and humoral mechanisms regulating secretory trophic processes, under the influence of

hydrochloric acid and pepsin ulcers form in the stomach and duodenum [5]. YAB is distinguished as an independent disease and symptomatic ulcers of the stomach and duodenum (medicinal, "stressful", in endocrine pathology, and other chronic diseases of the internal organs). Esophagogastroduodenoscopy is recommended for all patients with suspected YAB, in the absence of contraindications, in order to confirm the diagnosis [1]. In order to determine the indications for eradication therapy, testing for the presence of H. infection is recommended for all patients with YAB. pylori using a 13C breath test or the determination of H. Pylori antigen in feces, and with simultaneous EGDS - using a rapid urease test. [11]All patients with YAB with positive test results for H. Pylori infection are recommended to undergo eradication therapy in order to prevent subsequent relapses of YAB, and antisecretory therapy with proton pump inhibitors is recommended for 4-6 weeks in order to achieve ulcer healing [19]. Clinical recommendations contain criteria for evaluating the quality of medical care, the algorithm of the doctor's action, as well as information for the patient.[6] The etiology of peptic ulcer disease has not been sufficiently studied [8]. Previously, hydrochloric acid and pepsin were considered the main factors in the development of the disease, but recent studies have shown the crucial role of Helicobacter pylori [25]. The infectious process caused by these microorganisms is also a risk factor for stomach cancer and certain types of gastric lymphomas.[30] Normally, these aggressive factors are countered by the protective mechanisms of the gastric and duodenal mucosa, such as mucus, bicarbonates, prostaglandins, in violation of which an ulcer occurs [2].

## Methods

The research employed a combination of clinical and diagnostic methods to investigate the comorbid course of peptic ulcer disease (PUD) with cardiovascular pathology. Esophagogastroduodenoscopy (EGDS) was utilized to diagnose PUD and confirm the presence of ulcers, while tests such as the 13C breath test and H. pylori antigen detection in feces were used to identify Helicobacter pylori infection. Additionally, patients underwent clinical assessments to monitor symptoms such as pain, dyspepsia, and appetite changes. Laboratory tests were conducted to analyze changes in gastric mucus composition, including the concentration of sialic acids and other biochemical markers. The study focused on the relationship between PUD, cardiovascular comorbidities, and the biochemical activity in the gastric mucosa, providing insight into the pathophysiology of the disease and its interactions with cardiovascular disorders.

## Result and Discussion

In order to understand the pathogenesis of peptic ulcer disease, it is necessary to know the physiology of the stomach well [30]. The main stimulant of hydrochloric acid secretion is food intake.[31] It is customary to distinguish three phases of gastric secretion: cerebral, gastric and intestinal. In the cerebral phase, secretion changes in response to the appearance, smell, taste and thoughts of food, in the gastric phase – when food enters the stomach, in the intestinal phase - when it enters the small intestine.[23] The cerebral phase, which involves the cerebral cortex and hypothalamus, is mediated by activation of the vagus nerve, which stimulates enterochromaffin-like and lining cells and to a lesser extent affects the release of gastrin. The gastric phase is caused by irritation of the chemoreceptors and mechanoreceptors of the stomach with food that has entered it.[16] Stretching the stomach with food increases the secretion of hydrochloric acid, but has almost no effect on gastrin levels.[32] The stretching effect is probably mediated by the vagus nerve and is suppressed by atropine. Some components of food, especially proteins and products of their digestion, stimulate the release of gastrin and, thereby, increase the acidity of gastric contents [35]. The intestinal phase of gastric secretion

it is triggered by the entry of chyme into the proximal part of the small intestine: gastrin and peptides similar in action are produced in its wall, and the absorbed amino acids act directly on the lining cells.[29] Currently, peptic ulcer disease is considered to be a multifactorial disease considered by the theory of scales, on which aggressive (risk factors) and protective mechanisms are located [30]. A special place among aggressive factors is occupied by N.B.R., as well as neuro-endocrine factors in terms of psychosomatic disorders. Psychosomatic medicine today examines the relationship and relationship between a person's emotional life and the causes (etiological) factors of somatic disorders, i.e. evaluates the role of individual mental environmental factors in the formation of diseases.[22] During a special examination, various psychopathological disorders are determined in patients with YAB, including psychovegetative syndrome, and astheno-depressive symptoms prevail [34]. Psychogenic factors (family and labor conflicts, sudden material problems, etc.) play the role of a "triggering" mechanism of psychosomatic disease, and the choice of a target organ (stomach, duodenum) is explained by the presence of hereditary YAB burden and ready-made biological determinants. In the realization of a psychosomatic disease, the participation of a personal factor is necessary.[15] In the personality structure of a YAB patient, we noted the predominance of cycloid and epileptoid traits; emotive and demonstrative (emotionally unbalanced) types were less common. Psychosomatic disease usually develops in that organ or in that system of organs that, in the patient's mind, are the most important in life

the body [9]. In recent years, attitudes towards the alimentary (nutritional) factor have changed due to the lack of direct evidence of the influence of dietary disorders on the development of diabetes. However, prolonged consumption of spicy, coarse, cold or hot food can contribute to the development of chronic gastritis, which is a pre-ulcerative condition.[11] Our daily life experience indicates that errors in diet often cause an exacerbation of YAB [14]. The role of bad habits (smoking, alcohol abuse) in the development of diabetes is quite modest, but it is at least unwise to completely underestimate their impact on the course of the disease. Nicotine and alcohol stimulate the formation

of gastric juice, disrupt the protective functions of the mucous membrane of the gastrointestinal tract (GI tract) and can provoke the development and exacerbation of UD [33]. The clinical picture of the disease depends on the location of the ulcer, the stage of the disease (exacerbation, remission), the characteristics of the secretory and motor evacuation functions of the gastroduodenal zone, and the age of the patient. [28] Patients seek medical help, as a rule, with an exacerbation of the disease, accompanied by the appearance of a number of nonspecific symptoms that occur in other gastrointestinal diseases.[31] An asymptomatic exacerbation of YAB is possible. Pain and dyspeptic syndromes are leading in the clinical picture of the disease [26]. Pain is the most typical and frequent sign of IAB. As a rule, the pain is localized in the epigastric region to the left or right of the median line, sometimes in the xiphoid process of the sternum. Its intensity varies; Subjectively, pain is perceived as aching, pressing, burning, cutting. It is important to find out the time of occurrence and disappearance of pain, their connection with food intake. Early pains occur within 1 hour after eating, gradually increase and persist for 1.5-2 hours. With ulcers of the cardiac, subcardial and fundal parts of the stomach, pain appears immediately after eating, with ulcers of the stomach body – 0.5–1 hour after eating. Late pain appears 1.5–2 hours after eating and is characteristic of ulcers of the pyloric part of the stomach and duodenal bulb. Hungry (nocturnal) pains are observed 2-4 hours after eating, disappear after the next meal and are characteristic of duodenal ulcers. A combination of early and late pain is possible with combined or multiple ulcers [14]. Dyspeptic disorders – heartburn, vomiting, nausea, belching, decreased appetite – are less permanent, specific and less affecting the psychological state of the patient signs of YAB than pain. Heartburn can precede the formation of ulcers and be the only symptom of the disease. Nausea is often combined with vomiting, which more often occurs "at the height" of pain and brings relief. [17]. Appetite is usually preserved, and its decrease may be due to fear of eating due to the possibility of pain or increased pain. Constipation with a peculiar dense, fragmented stool ("sheep feces") is a frequent companion of YAB [19].

## Conclusion

1. The comorbid course of IB with concomitant pathology of the cardiovascular system is formed with ulcers of F in 36.2% and ulcers of DC in 25.5% of cases. The leading clinical manifestations are atypical pain syndrome, severe and prolonged dyspeptic manifestations with asthenovegetative syndrome. 2. When combined with the pathology of the cardiovascular system, there is a pronounced change in the composition of gastric mucus, manifested by an increase in the concentration of free and oligocoated sialic acids and a decrease in BSSC, which indicates the activity of catabolic processes in the mucosa of the gastroduodenal zone and a decrease in its resistance to aggressive factors.

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