UTC: 616.009/33/34-002.44

# VEGETATIVE DYSTONIA SYNDROME AS ETIOLOGY ULCER DISEASE (LITERATURE REVIEW)

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**Resume.** The article discusses the nosology of gastric ulcer and duodenal ulcer, pathogenetic mechanisms of their occurrence, pathomechanisms of their connection with the syndrome of vegetative dystonia, the main cause of which is the endogenous factor of the disease development - dysfunction of the autonomic nervous system. The main causative agent of the disease, Helicobacter pylori, was not left without attention..

**Keywords:** gastric ulcer and duodenal ulcer, autonomic nervous system, autonomic dystonia syndrome, helicobacter pylori, vagotonia.

Gastric ulcer disease (GUD) and duodenal ulcer disease (DUD) are chronic polyetiological recurrent diseases, which are associated with complex changes in the nervous, hypothalamic-pituitary, hypothalamic-pituitary-adrenal and local gastroduodenal processes, leading to changes in trophic processes in the mucous membrane of the stomach and duodenum. In turn, the resulting ulcer defect is a source of interoreceptor stimulation, which supports the disruption of neuro-hormonal control. Thus, the resulting "vicious circle" leads to the chronic course of gastric ulcer [15].

One of the ideas about changes in the mucous membrane of the stomach and duodenum in pathophysiology is based on the doctrine of the role of the nervous system in the regulation of metabolic and trophic processes, which ensure the structural integrity and physiological (functional) state of cells, tissues and organs [6]. The emergence of this view was based on the concept of I.P. Pavlov "on the trophic function of the nervous system", the teachings of L.A. Orbeli "On the adaptive-trophic role of the sympathetic nervous system" [10]. and P.K. Anokhin's studies "on functional systems" [15].

The role of the hypothalamus, hypothalamus-pituitary-adrenal system in the pathogenesis of gastric and duodenal ulcers has been studied by a number of scientists [6]. Therefore, many scientists believe that the hypothalamus-pituitary-adrenal system plays an important role in the development of dysregulation of the stomach

and duodenum and the appearance of dystrophic lesions, but in our opinion, the mechanisms of such effects have not been fully explored.

In the pathogenesis of peptic ulcer disease, the syndrome of vegetative dystonia always occurs with an increase in the parasympathetic system [12]. Increased activity of the gastric mucosa, accompanied by hypersecretion, leads to hyperplasia of the gastric glands and simultaneous disruption of trophic activity, as a result of which the mechanism of development of peptic ulcer disease is based not only on an increase in the tone of the parasympathetic nervous system, but also on a decrease in the sympathetic nervous system [11]. When assessing the nature of autonomic tone changes in patients with peptic ulcer disease, relying on autonomic indicators, electrocoagulogram, gastric secretion data, and electrogastrogram (EGG) data gave conflicting results [12]. According to the Curdo index, vagotonia was often detected in patients with DUD, and according to the minute volume index, sympathotonia was detected. Both of these tests are common in patients with peptic ulcer disease. In DUD, vagotonia was detected by this examination. At the same time, EGG and electrocoagulogram data showed the importance of sympathetic influence.

It is known that the onset of an emotional state is mainly carried out through the nervous system, that is, through nerve stimuli transmitted from the cerebral hemispheres to the hypothalamus, reticular formation and limbic systems. The first mediator responsible for this reaction is believed to be acetylcholine, and the subsequent implementation of the stress response occurs through neurogenic and hormonal pathways [5]. The sympathetic and parasympathetic subzones of the reticular formation are located at different levels of the CNS [12]. Through each of them, its own group of adaptive loops is closed, directed to a group of effector devices. Each type of receptor has its own adaptive reflex loop. Reticular neurons capable of autonomic activity are sources of a constant background signal in both parts of the autonomic nervous system. Each lower zone of the reticular formation and its microzones have effector loops aimed at activating their own group with a specific spectrum of warning afferent signals. Based on this, it is advisable to assess not the general vegetative tone, but its components in different organs [1].

The increase in acid-peptic activity is also influenced by the participation of endocrine hormones from the parasympathetic nervous system (ACTH, cortisol, thyroxine, insulin, etc.) and hormones of the gastrointestinal system (histamine, gastrin, bombesin), as well as factors that physiologically activate "substance P" - gastrin [2]. The aggressive action of the above hormones, as a rule, is manifested in a decrease in hormonal activity, inhibition of gastric secretion. Such hormones include somatostatin, glucagon, calcitonin, sex hormones, secretin, cholecystokinin, urogastrone, serotonin, prostaglandins, as well as endogenous opioids, enkephalins and endorphins.

In 1983, the discovery of campylobacter pylori, now known as Helicobacter pylori (HP), led to the development of a large-scale study of the infectious theory of the etiology of peptic ulcer disease [7]. It is known that the presence of metaplasia in the gastric field in the duodenum is a cellular marker for HP. According to research, the formation of foci of gastric metaplasia in the duodenum occurs as a result of prolonged damage to its mucous membrane with hydrochloric acid. Increased acid production in the stomach and prolonged acidification of the duodenum lead to the development of foci of gastric metaplasia in it, creating conditions for HP colonization.

Despite the large number of theories and theories of the development of peptic ulcer disease, there is no explanation for the origin of peptic ulcer disease in half of HP-negative patients [4]. The possibility of Helicobacter pylori infection in peptic ulcer disease can be diagnosed in no more than 2/3 of patients with duodenal ulcer, and in 2/5 of all patients with gastric ulcer. Thus, the author concludes that there is no data on the 100% bacterial infection of patients with gastric ulcer, HP infection acts as a local factor that affects the mucosa from the outside and acts as an activating factor of aggression. The authors of this article agree with this point of view, namely, due to a decrease in local immunity and other variable factors in the gastroduodenal system, HP is considered only a pathogenetic link [13].

ШУ нарса аниққи, ошқозон-ичак тизимига Helicobacter pylori инвазияси does not lead to ulcerative processes, and, for example, the presence of HP in a large proportion of gastritis cases without gastric ulcer is reliable evidence that HP is not an etiological factor in the development of gastric ulcer or duodenal ulcer. This is confirmed by a number of studies [3]. Currently, there is no experimental model of gastric ulceration by Helicobacter pylori introduction [9].

According to Soshina A.A. et al., gastric ulcer associated with Helicobacter pylori occurs in 22.7% of patients, and OBJECTIVES - in 80.9% of patients. Zhernakova N.I. [4] studied the clinic, pathogenesis and treatment of the disease in elderly patients with gastric ulcer, and out of 178 patients, 147 were diagnosed with gastric ulcer associated with Helicobacter pylori, and 31 patients did not have infection. At the same time, the author noted that the infection increases during the course of gastric ulcer. The leading indicators are ensuring a favorable course of the disease by eliminating Helicobacter pylori and normalizing the neuroendocrine state.

According to our data [8], in patients with peptic ulcer disease, cortisol concentration during the exacerbation period was higher than in patients in the control group ($p<0.05$).

According to some literature data [3], the association of peptic ulcers with Helicobacter pylori accounts for 70-80% of duodenal ulcers and 50-60% of gastric ulcers. Therefore, we must understand that association does not imply causation.

Currently, none of the theories of the pathogenesis of peptic ulcer disease covers the diversity of functions involved and the changes that lead to their development [12].

Ярали касаллик билан оғриган беморларнинг физиологик жараёнларини It is urgent to study the complex in the context of various methods of treatment at different stages of the disease. Therapy, the author continues [14], “Diagnosis and treatment of diseases of the digestive system taking into account quality standards” (Order of the Ministry of Health of the Russian Federation of April 17, 1998 No. 125) from a modern point of view is considered to be able to really affect only the local factors of ulcer pathogenesis - HP and peptic factor.

**Conclusion.** Thus, we should consider the existence of very diverse mechanisms of development of gastric and duodenal ulcers. Perhaps none of the listed mechanisms plays an independent role in determining the formation and chronicity of gastric and duodenal ulcers, and only their combination can determine the nosological form of gastric and duodenal ulcers.

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