FACTORS IN BLEEDING FROM GASTRIC AND DUODENAL ULCERS.

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Summary. Currently, bleeding due to gastric and duodenal ulcers has doubled, especially in the elderly and the elderly. The mortality rate from acute bleeding from ulcers in the gastrointestinal tract is 5-20%, while after emergency operations it is 4-73%, and in the elderly this figure is more than 80%.

Keywords: ulcer diseases; NVPS, bleeding.

Ulcer disease (UD) is a chronic, seasonally recurring, i.e. relapsing and remitting disease, characterized by a limited deep defect in the mucous membrane of the stomach or duodenum.

It is estimated that 11–14% of men and 8–11% of women worldwide will develop peptic ulcer disease during their lifetime. In the United States, 500,000 people develop a new ulcer each year, and more than 4 million people develop a new ulcer [12; 17;, 20;]. Duodenal ulcers are 4–5 times more common than gastric ulcers. Although duodenal ulcers are more common in men than in women, gastric ulcers occur equally in both sexes.

Recently, according to data from several foreign authors, not only in Central Asia, but also in Russia and abroad, the number of hospitalizations of patients with uncomplicated ulcers has significantly decreased. According to the statistical data of the Ministry of Health of the Russian Federation, in terms of the analysis of the prevalence of ulcers, in 2006-2017 the incidence of ulcers decreased from 128.7 to 79.5 per 100,000 people. However, at the same time, an increase in the incidence of ulcers, especially bleeding and perforation, has been noted worldwide, the main reason for this is the use of NSAIDs [2; 6;].

The origin and pathogenesis of peptic ulcer disease is the imbalance, that is, the imbalance between the aggressive factor (acid, pepsin) in the gastric contents and the protective factor in the mucous membrane.

The aggressive environment in the formation of ulcers includes factors such as hyperproduction of gastrin due to the proliferation of connective tissue in this area, impaired nervous and humoral control of acid secretion in the stomach, increased production of pepsin and pepsinogen, impaired gastroduodenal motility (increased or decreased evacuation in the stomach and duodenum), and infection of the mucous membrane with Helicobacter pylori microorganisms.

A decrease in the protective ability of the gastric and duodenal mucosa can be caused by a decrease in gastric mucus or a change in its composition and quality, a decrease in bicarbonate secretion, a decrease in the regenerative activity of epithelial cells, impaired blood circulation in the gastric mucosa, and a decrease in the amount of prostaglandins in the gastric wall (for example, when using nonsteroidal anti-inflammatory drugs).

Currently, the bacterium Helicobacter pylori, discovered in 1983 by Australian scientists Marshall B. and Warren J., which is widespread in the mucous membrane of the stomach and duodenum, is considered to play an important role in the etiology of peptic ulcer disease.

Various mechanisms of the harmful effect of Helicobacter pylori on the mucous membrane of the stomach and duodenum have been identified. During their life, these microorganisms produce cytotoxins that disrupt the protective properties and integrity of the mucous membrane, as well as a number of enzymes, such as urease, protease, phospholipase. Among microorganisms, the Vac A strain of H. Pylori, which secretes a vacuolating cytotoxin, leads to necrosis of epithelial cells. H. Pylori causes inflammation in the gastric mucosa by secreting interleukins and lysosomal enzymes that are factors of necrosis and tumor formation.

The spread of Helicobacter pylori (H. Pylori) in the gastric mucosa is accompanied by superficial antral gastritis and duodenitis, which leads to an increase in the amount of gastrin, and subsequently to an increase in the secretion of hydrochloric acid. A large amount of hydrochloric acid in the duodenal cavity in the absence of pancreatic bicarbonates provokes the development of duodenitis. In addition, this process manifests itself in the duodenum as focal gastric metaplasia, i.e., the reconstruction of the duodenal mucosa epithelium to the gastric type, which ensures the spread of Helicobacter pylori (H. Pylori) in this area. Subsequently, as a result of the continued unfavorable environment and the addition of additional factors, i.e., hereditary predisposition, bad habits, mental depression, etc., an ulcer defect is formed in the area of metaplasia of the mucosa [1; 3; 4].

The role of Helicobacter pylori (H. Pylori) in the etiology and progression of peptic ulcer disease is considered in the description of this disease. Depending on the presence or absence of H. Pylori, peptic ulcer disease is divided into H. Pylori-infected and non-infected types. Also, independent essential ulcers (EEU), symptomatic ulcers of the stomach and duodenum (due to drugs, mental disorders, endocrine disorders and a number of other diseases) are distinguished.

What is the reason for the appearance of morphological changes in a specific limited area of the stomach wall? The question remains as to the role and cause of factors in the development of various forms of chronic gastritis: Helicobacter pylori (H.P.), biliary gastroduodenal reflux gastritis, lymphocytic gastritis, autoimmune

gastritis. For example, Helicobacter pylori is found in 60% of healthy people, while peptic ulcer disease is 0.20% [7; 9]. This situation encourages work on theories about the relationship between peptic ulcer disease and N.p. [2; 4]. Currently, scientists pay great attention to changes in the microcirculation of the gastric mucosa. At one time, Virchow and Aschoff proved that hemodynamic changes in the stomach wall play a major role in the development of ulcers. Their theory can now provide an understanding of the shape and location of ulcers in the gastric wall [8; 10]. Their scientific research in this area has revealed that functional impairment of the submucosal vascular network results in the formation of small, circumscribed areas of infarction in the gastric wall and the formation of ulceration and fibrinoid necrosis in this area.

Stress ulcers are often numerous, erosive, and localized to a specific area of the gastric wall. They do not extend beyond the mucosa. Stress ulcers occur after biological or psychological stress, acute organ failure, or various types of shock. Perforation of acute ulcers is rare, with the diameter of the ulcer being less than 1 cm. The ulcer margins are soft, elastic, and free of infiltration, and there is no evidence of inflammation. The incidence of acute stress ulcers has now been reduced by 50% as a result of preventive measures [5; 9].

Essential ulcers are divided into stages in the diagnosis and its course: period of excitement, healing, scarring, quiescence, and also scar deformation of the stomach and duodenum is noted [4; 5]. Thus, the analysis of the literature showed that, despite extensive scientific and practical work on bleeding from the gastrointestinal system and its timely diagnosis and stopping, conservative, mini-invasive treatment methods and postoperative complications, as well as the mortality rate, remain high. To a large extent, the number of these complications depends on the patient's existing additional diseases, risk factors, the duration of bleeding, and errors in the timely assessment of the patient's condition and the selection of the treatment method.

Conclusion: At the same time, the results of studies conducted in recent years have shown that the authors pay attention to the informational value of risk factors for bleeding as criteria for predicting the course of the pathological process. An analysis of domestic and foreign literature known to us has shown that there is a lack of information on the prognosis and assessment of the outcome of acute bleeding from the upper gastrointestinal tract, especially in cases of concomitant diseases and risk factors, depending on their timely diagnosis and treatment methods. Taking into account the above, there are still unresolved problems of timely diagnosis and cessation of bleeding from the gastrointestinal tract, which is one of the urgent problems of medicine, and the development of indications for the use of anticoagulants in patients, the procedure for their use, contraindications, duration of use, precautions, etc. requires

considerable scientific research. The scientific research we are engaged in is aimed at this very problem of surgery.

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