

ETIOPATHOGENESIS, PREVENTION AND TREATMENT OF ACUTE GASTRITIS AND DUODENITIS

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Abstract: This article is devoted to the analysis of the current state of medicine in matters of etiopathogenesis, prevention and treatment of acute inflammatory diseases of the stomach and duodenum. Acute and chronic focal lesions of the mucous membrane of the stomach and duodenum are symptomatic ulcers, and in etiology and pathogenesis they differ from peptic ulcer disease.

Key words: acute inflammation, ulcer, bleeding, antacids.

INTRODUCTION: It should be noted that acute injuries to the mucous membrane of the stomach and duodenum include ulcerative lesions of the mucous membrane that developed in patients in critical condition.

Thus, in the 19th century, acute ulcers and gastric bleeding were discovered in patients who had suffered a body burn (Curling), stress, damage to the hypothalamus (Billroth), sepsis (Eyselsberg), and cerebral hemorrhage (Cushing).

Currently, clinical cases of the presence of acute ulcers in patients in shock, frostbite, with severe forms of pulmonary, cardiac, renal and liver failure, with tetanus, poliomyelitis, typhoid fever, general peritonitis have been described [7].

The pathogenesis of these diseases is ischemia and acid-peptic damage to the mucous membrane, which results in inhibition of all gastroprotective functions and anemia. This disease can lead to hypotension, ischemia of the gastroduodenal mucosa, and subsequently reverse diffusion of H⁺, acidosis, depletion of buffer systems, death of epithelial cells and damage to the mucosa.

Against the background of mucosal ischemia, incomplete neutralization of hydrochloric acid occurs due to insufficient production of protective factors (bicarbonates, mucin, etc.).

Also, dysfunction of the mucous barrier occurs with bile reflux, uremia, and NSAID use, which is accompanied by an increase in the reverse diffusion of hydrogen ions and their accumulation inside cells [8]. Bile salts entering the stomach during duodenogastric reflux can, in small concentrations, disrupt the secretion of bicarbonate, reduce the viscosity and elasticity of the mucous gel, reduce the pH gradient and cause disruption of the integrity of surface epithelial cells in the antrum of the stomach.

Acute inflammatory processes are usually multiple, located mainly on the lesser curvature, less often - on the fundus and antrum of the stomach.

In this case, the damage can be in the form of superficial erosions and deep ulcers of the mucous membrane of the stomach and duodenum, which can be numerous and occupy most of the surface of the mucous membrane. A distinctive feature of acute ulcers is the absence of an inflammatory shaft surrounding the ulcer.

Acute lesions of the mucous membrane of the stomach and duodenum have a polymorphic course and symptoms (manifest by abdominal pain and/or dyspeptic disorders of an unspecified nature), and also be asymptomatic, masked by the clinical manifestations of the underlying disease (myocardial infarction, trauma, burn disease, etc.). Erosive and ulcerative lesions sometimes manifest themselves with typical clinical symptoms of peptic ulcer disease. Of all the complications of acute erosions and gastric ulcers, the most common is bleeding (in 15–78% of cases), which is

manifested by vomiting of blood or coffee grounds, melena [5,6,7,8], and less commonly, perforation of the ulcer (6–14%). The mortality rate for these complications ranges from 6 to 14% [4].

Endoscopic examination is the main method for diagnosing acute lesions of the mucous membrane of the stomach and duodenum, which makes it possible to assess the extent, depth of the lesion, the risk of recurrent bleeding, and to carry out endoscopic hemostasis. Erosive-ulcerative lesions are most often localized in the stomach - 72%, in the duodenum (duodenum) - 54% and in the esophagus - 20%. In 38% of cases, erosions and ulcers are found simultaneously in various organs, while in 12% of cases, examination reveals synchronous damage to the esophagus, stomach and duodenum [3]. The frequency of detection of acute erosions and acute ulcers is almost the same, while in 23% of studies a combination of them is observed.

It should be noted that acute erosions are multiple in nature, and single ulcers with equal frequency are detected in 43% of patients and are localized in the stomach and duodenum; multiple ulcers are predominantly duodenal and occur in 57% of cases [10].

Prevention of acute gastritis and acute bulbitis should be aimed at timely and effective treatment of the underlying disease, maintaining normal blood volume, ensuring the normal functioning of the respiratory and circulatory organs, correcting the acid-base and electrolyte balance, and preventing infectious complications.

Preventive and therapeutic measures for acute erosive and ulcerative lesions of the mucous membrane of the stomach and duodenum include antacid, antisecretory and gastroprotective therapy aimed at maintaining pH above 3.5 (up to 6.0), normalizing gastric motility, increasing the stability of the mucous barrier [1,2,3,4]. For these purposes use:

- antacids and gastroprotectors;
- histamine H₂ receptor blockers (H₂ blockers);
- proton pump inhibitors.

Antacids reduce acidity in the stomach by chemically reacting with hydrochloric acid without affecting the secretion of hydrochloric acid.

However, antacids can cause diarrhea and slow down the absorption of medications. Sodium bicarbonate and calcium carbonate, when interacting with hydrochloric acid, form carbon dioxide, which, increasing pressure in the stomach, leads to distension of the stomach and can cause regurgitation and aspiration of gastric contents into the trachea, and can also cause a secondary increase in the secretion of hydrochloric acid.

The prophylactic use of H₂ blockers prevents the development of acute erosive and ulcerative lesions of the gastroduodenal zone. However, drugs in this group have a short-term effect and can aggravate ischemia of the walls of the stomach and duodenum. The use of H₂-blockers in large doses, especially in stressful situations, has an extremely negative effect on the detoxification function of the liver (inhibition of the cytochrome P450 system), which leads to aggravation of existing encephalopathy.

Proton pump inhibitors (PPIs) effectively suppress basal and stimulated production of hydrochloric acid by inhibiting H⁺/K⁺-ATPase, the proton pump of the parietal cell [9].

The advantage of PPIs is the absence of the ability to cause tachyphylaxis and a more predictable effect, which allows them to be used both for the prevention and treatment of stress erosive and ulcerative injuries of the gastrointestinal tract, as well as to stop gastroduodenal bleeding [11].

In general, based on the analysis of literature data regarding the problems of stress erosive and ulcerative damage to the mucous membrane of the stomach and duodenum, we can conclude:

1. Stress conditions that often occur during critical conditions have a negative effect on the mucous membrane of the gastrointestinal tract, and the increasing frequency of various ulcerogenic medications taken by the population creates favorable conditions for an increase in the number of erosive and ulcerative lesions of the upper digestive tract.

2. The clinical significance of acute injuries to the mucous membrane of the stomach and duodenum is that they occur in 11-78% of cases under various stressful conditions and pose a serious threat to people's lives, as they are associated with a high risk of bleeding, the fatal outcome of which reaches 64 %.

3. Treatment of acute injuries to the mucous membrane of the stomach and duodenum is currently carried out with a wide range of drugs with different mechanisms of action. However, in critical situations accompanied by the development of bleeding from stress ulcers, it is more advisable to use drugs that have different routes of administration into the body and have a quick, long-lasting, safe effect.

4. In the selection of drugs, a differentiated approach is necessary, taking into account their mechanism of action, which will allow obtaining a rapid clinical effect on the course of acute lesions of the mucous membrane of the stomach and duodenum.

5. To prevent stress ulcers of the stomach and duodenum and their complications, one should primarily focus on identifying risk groups, timely and thorough treatment of the underlying disease, maintaining normal blood volume, ensuring respiratory and circulatory functions, correcting the acid-base state and electrolyte balance, fighting with infectious complications.

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