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Study of the Etiopathogenesis of Hyperacid Syndrome in Diarrheas of Various Etiologies

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Annotation: The aim of this work is to analyze the pathogenetic relationship between hyperacid syndrome and the development of diarrheal syndrome, to evaluate the role of acid-dependent mechanisms in the formation of the clinical picture, and to substantiate the need to include gastric acid production assessment in the diagnostic algorithm for chronic diarrhea. Hyperacidity is considered a condition characterized by excessive secretion of hydrochloric acid by the parietal cells of the stomach, leading to the inability of the protective mechanisms of the duodenum and pancreas to effectively neutralize acidic chyme. The entry of acidic contents into the upper sections of the small intestine causes inactivation of digestive enzymes, disruption of nutrient hydrolysis processes, and malabsorption, which is accompanied by osmotic and secretory diarrhea. The mechanisms of intestinal mucosal damage, acceleration of motility, and loss of fluid and electrolytes are described. It is emphasized that hyperacidity often remains an underdiagnosed cause of chronic diarrhea and is mistakenly interpreted as colitis or irritable bowel syndrome. It has been proven that therapy aimed at reducing acid production (proton pump inhibitors) leads to the cessation of diarrhea, which confirms the key role of the acid factor[1].

Keywords: hyperacidic syndrome;

hydrochloric acid; acid production; gastrin; malabsorption; secretory diarrhea; parietal cells; gastrointestinal tract.

Introduction. Hyperacid syndrome is a pathological condition characterized by excessive production of hydrochloric acid by the stomach's parietal cells. Increased acidity of gastric secretions disrupts the physiological balance between aggressive factors (hydrochloric acid, pepsin) and protective factors (mucus, bicarbonates, adequate duodenal peristalsis). In clinical practice, hyperacidity is primarily associated with gastritis, peptic ulcer disease, and gastroesophageal reflux disease. However, the connection between acid hypersecretion and the development of diarrheal syndrome is considered much less frequently, leading to an underestimation of the pathogenetic role of the acid factor in the formation of chronic or recurrent diarrhea[2]. Diarrhea is traditionally interpreted as a consequence of infectious processes, malabsorption syndrome, enzymatic insufficiency, inflammatory bowel diseases, or functional disorders. Nevertheless, in some patients, the cause of frequent liquid stools is not intestinal pathology, but rather an excessive acid load entering the duodenum from the stomach, especially after meals. In conditions of hydrochloric acid hypersecretion, the pH of chyme can remain critically low as it passes through the small intestine, leading to the inactivation of digestive enzymes, stimulating the secretion of water and electrolytes into the intestinal lumen, and accelerating intestinal motility[3].

The mechanism of acid-induced diarrhea develops in several stages: acidic chyme enters the duodenum in a volume exceeding the compensatory capacity of pancreatic bicarbonate secretion; low pH inactivates lipase and amylase, leading to impaired nutrient digestion; undigested substances create an osmotic load and cause water and electrolyte secretion; irritation of small intestine receptors enhances peristalsis. [4]

Thus, hyperacidity syndrome is directly linked to the mechanism of diarrhea development, as confirmed by studies conducted by Russian gastroenterologists. Zollinger-Ellison syndrome is a classic example of a condition in which acid hypersecretion leads to persistent diarrhea that is refractory to conventional therapy. Similar mechanisms are observed in gastrin hypersecretion associated with chronic gastritis, G-cell hyperplasia in the antral region, prolonged abuse of caffeine and nicotine, use of NSAIDs, and disorders of neurohumoral regulation [5].

The influence of diet and lifestyle deserves special attention. Consuming large amounts of acid-producing foods increases the secretion of gastrin and acid. In patients with functional digestive disorders, this can manifest as "postprandial diarrhea." Clinically, hyperacidic diarrhea is characterized by watery stools with an acidic odor, pain and burning in the epigastrium, symptoms appearing mainly after eating, and relief after taking antacids or proton pump inhibitors (PPIs). Despite obvious pathogenetic mechanisms, the diagnosis is rarely made and patients often receive ineffective therapy, whereas suppression of acid production leads to rapid improvement [6].

Pathogenesis, mechanism of development, and diagnosis. Hyperacidic syndrome is a state of persistent increase in the secretion of hydrochloric acid by parietal cells of the stomach under the influence of neurohumoral factors. The main regulator of acid production is gastrin. Its overproduction, disruption of neurogenic regulation, and alteration of negative feedback mechanisms contribute to the formation of pathological acid hypersecretion. Acid production disorders play an important role not only in the development of gastroduodenal diseases but also in the formation of diarrheal syndrome, which is caused by the damaging effect of acidic chyme on the mucous membrane of the small intestine and on the processes of nutrient hydrolysis [7].

Under normal conditions, hydrochloric acid performs key physiological functions: it activates pepsinogen, provides a bactericidal effect, regulates gastric emptying, and stimulates the

secretion of secretin and cholecystokinin in the duodenum. Acid secretion is controlled by three main mechanisms: neurogenic (acetylcholine via M3 receptors), hormonal (gastrin via CCK-2 receptors), and paracrine (histamine via H2 receptors). In cases of hyperacidity, all three pathways are activated, leading to constant stimulation of H+/K+-ATPase - the final effector of acid production[8].

Factors causing hyperacidity include: hypergastrinemia (Zollinger-Ellison syndrome, G-cell hyperplasia), chronic gastritis, prolonged nicotine and caffeine stimulation, stress-induced vagal activity, long-term use of NSAIDs, and impaired gastric emptying. In some patients, hyperacidity is functional in nature and is not associated with evident organic pathology[9]. Diarrhea in hydrochloric acid hypersecretion results from the combined effects of chemical damage to the mucosa, osmotic disturbances, secretory effects, and intestinal motor reactions. Inactivation of pancreatic enzymes at low pH (lipase, trypsin, amylase) leads to steatorrhea and malabsorption, while undigested substrates create an osmotic load. The acid activates enterochromaffin cells, stimulating serotonin secretion and increasing motility. Furthermore, the acid destroys protective mucin, increases mucosal permeability, and promotes inflammation[10].

Diagnosis includes confirming hyperacidity and ruling out other causes of diarrhea. Main methods: intragastric pH monitoring (24-hour or nocturnal), fasting blood gastrin level measurement, esophagogastroduodenoscopy with biopsy if necessary, laboratory tests for pancreatic insufficiency and infections, and stool analysis for steatorrhea. If gastrinoma is suspected, imaging (CT, MRI, endoscopic ultrasonography) and functional peptide tests are performed. Clinically, the causal relationship is confirmed by experimental suppression of acid production: if pH normalizes and diarrhea resolves, the connection is considered proven[11].

Of particular importance is the assessment of accompanying factors: diet, medication use (especially NSAIDs and prokinetics), gastric and duodenal motility, functional disorders, and psychogenic factors. Recent studies emphasize the role of the duodenal microenvironment and its interaction with pancreatic secretions in the development of symptoms[12].

Treatment. Treatment of hyperacid syndrome aims to suppress hydrochloric acid secretion, neutralize already secreted acid, reduce acid effects on the gastric and duodenal mucosa, correct digestive disorders, and prevent diarrheal syndrome[13].

Proton pump inhibitors (PPIs) form the cornerstone of therapy: omeprazole, pantoprazole, rabeprazole, esomeprazole. They block the H+/K+-ATPase of parietal cells, effectively reducing secretory activity. With adequate dosage and adherence to the treatment regimen, pH normalization contributes to the restoration of pancreatic enzyme activity and the cessation of osmotic diarrhea[14].

Antacids and aluminum-magnesium preparations are used to neutralize already secreted acid and rapidly alleviate symptoms. H2-receptor blockers (famotidine, ranitidine) can be used in cases of PPI intolerance, but their effectiveness is lower[15].

Dietary therapy includes reducing the consumption of products that stimulate acid secretion (strong coffee, alcohol, spicy and fried foods), increasing the intake of dietary fiber, and adopting a fractional eating pattern. Dietary adjustments help reduce the frequency of relapses and decrease the severity of symptoms[16].

In confirmed cases of gastrinoma (Zollinger-Ellison syndrome), surgical intervention and adjuvant oncological approaches are considered alongside drug therapy, depending on the stage of the disease[17,18]. Supportive therapy includes correction of electrolyte imbalances, replenishment of fat-soluble vitamin deficiencies in cases of steatorrhea, and, when necessary, enzyme replacement therapy[19,20].

Conclusions. Hyperacid syndrome is an important, but often underestimated cause of diarrhea. Mechanistically, excessive acidity leads to the inactivation of pancreatic enzymes, osmotic and

secretory components of diarrhea, damage to the mucous membrane, and accelerated motility. Comprehensive diagnosis and correction of acid production lead to the restoration of digestive functions and the elimination of diarrhea. Including acid production assessment in the diagnostic algorithm for patients with chronic diarrhea can increase diagnostic accuracy and treatment effectiveness.

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