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BIOCHEMICAL INDICATORS OF GASTRIC JUICE IN INCOMPETENCE OF THE PHYSIOLOGICAL GASTRIC CARDIA

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Insufficiency of the gastric cardia (incompetence of the physiological cardia — IPC) at the gastroesophageal junction, meaning the inability to close the upper end of the stomach, is significant in hiatal hernia (HH). The cardia, a muscular circular sphincter at the base of the esophagus, prevents acid and food reflux from the stomach. With HH, the upper part of the stomach protrudes through the opening of the diaphragm, leading to the relaxation of the cardia. This worsens reflux prevention, allowing acidic stomach contents to damage the esophageal mucosa. IPC in the HH is pivotal in the pathogenesis of gastroesophageal reflux. Understanding this determines the treatment strategy, including conservative (dietary changes, medications) and surgical interventions for hernia correction and restoration of physiological cardia function [1, 3].

Aim. Assess the peculiarities of biochemical indicators in gastric juice in the case of physiological cardia incompetence at the gastroesophageal junction.

Methods. Biochemical studies of gastric juice were conducted in 42 patients with IPC, corresponding to the diagnosis of hiatal hernia (2024 ICD-10-CM Diagnosis Code K44), who were hospitalized in the surgical department of the State Institution "Institute of Gastroenterology of the National Academy of Medical Sciences of Ukraine." Depending on the characteristics of the anatomical defect, patients with IPC were classified into two types: type I (sliding) hiatal hernia ($n = 26$) and type II (paraesophageal) hiatal hernia ($n = 16$). The control group ($n = 9$) consisted of healthy volunteers without diagnosed cardia pathology. Gastric juice sampling was performed during video esophagogastroduodenoscopy. pH, pepsin concentration, and bile acids were determined [5], as well as calcium content ("Filisit-Diagnostics"). Stable metabolites based on the total level of nitric oxide (NOx) were determined using a method involving the simultaneous reduction of nitrates to nitrites in the presence of vanadium chloride and diazotization reaction. Statistical analysis of the obtained results was performed on a personal computer using the licensed software package "Microsoft Excel" by paired comparison method, with significant differences at a probability error of less than 0.05.

Results and Discussion. In patients with type I, gastric juice was acidic, nearing hypoacidity, with a pH of 3.73±0.49, which was 2.3 times higher ($P < 0.01$) compared to control values (1.6±0.2). However, in type II, the pH level was 2.34±0.72, statistically indistinguishable from the control. It is conceivable that patients with type I experienced decreased acidity of gastric juice, possibly due to suppression of parietal cell function.

The concentration of pepsin in gastric juice in type I was 1.33±0.22 mg/ml, which was 4.4 times higher ($P < 0.05$) compared to the control (0.3±0.05 mg/ml). This indicates hyperfunction of the chief cells of the gastric mucosal glands. In type II, pepsin concentration (0.72±0.27 mg/ml) did not show statistically significant differences compared to the control. The combined changes in gastric juice acidity and pepsin concentration suggest functional disturbances in the secretory

activity of the gastric mucosa as the physiological direct correlation between increased juice acidity and increased pepsin concentration is lost.

Experimental studies revealed that stimulation of gastric chief cells under conditions of excess NO is associated with compensatory increased pepsin production in response to reduced activity under low acidity conditions [5].

To confirm or refute the hypothesis, the concentration of NOx in the gastric juice of patients with HH was determined. In type I, NOx concentration was 83.15±11.03 μmol/L, 32.0% lower ($P < 0.05$) than in the control group (122.8±15.52 μmol/L). This suggests that changes in the composition of gastric juice in type I were not determined by the direct action of the NOergic system on secretory cells. NOx concentration in type II (117.65±72.28 μmol/L) did not reach statistical significance ($P > 0.05$).

It is known that the release of Ca^{2+} and subsequent influx are processes that control the physiological secretion of digestive enzymes in response to stimulation via the vagus nerve [2]. The total calcium concentration in gastric juice in patients with type I was (1.05±0.13 mmol/L) and in type II (1.30±0.20 mmol/L), with no statistically significant differences compared to the control (1.02±0.18 mmol/L).

The observed increase in gastric juice pH could be caused by duodenogastric reflux [4] amid reduced nitric oxide metabolite concentration. However, reflux of duodenal contents into the stomach was not confirmed in patients with both types of HH. The concentration of bile acids in gastric juice in patients with type I was (0.21±0.04 mmol/L), and in type II (0.24±0.08 mmol/L), with no statistically significant differences compared to the control (0.21±0.08 mmol/L).

Therefore, the identification of links influencing the disturbance of gastric secretion regulation in patients with type I requires further investigation.

Conclusions. It was found that in the case of type II, there were no changes in the secretory activity of parietal and chief cells of the gastric mucosal glands, as well as in the concentration of stable metabolites of nitric oxide.

The pH indicators of gastric juice in type I were acidic, tending towards hypoacidity, and increased by 2.3 times ($P < 0.01$) compared to control values. The pepsin level in the sliding type (type I) was increased by 4.4 times ($P < 0.05$) compared to the control. Nitric oxide, based on the level of stable metabolites (NOx), in type I HH was 32% lower than in the control group ($P < 0.05$). Determining the pathophysiological mechanisms leading to the observed changes in the biochemical indicators of gastric juice requires further investigation.

Key words: incompetence of the physiological cardia, gastric juice, hiatal hernia.

Authors' contribution. AMH conducted biochemical studies, processed and analyzed the obtained results, wrote the abstract, OVS edited the text and analyzed the data.

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