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RESULTS OF THE STUDY OF THE ACID-PRODUCING FUNCTION OF H. PYLORI-ASSOCIATED DISEASES OF THE STOMACH.

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Abstract:

The authors studied the acid-producing function of the stomach in H. pylori-associated pathologies. According to the study, the studied pathology was characterized by the presence of a hyperacid state of gastric juice in chronic non-atrophic gastritis according to the pH values. Also, in patients with atrophic gastritis in gastric juice, sub- and decompensated violations of the acid neutralizing function of the stomach are noted. At the same time, the arch and antrum of the stomach are the most informative parameters for measuring pH in the stomach.

Keywords: H. pylori-associated diseases of the stomach, pH in the stomach, acid neutralizing function, fornix and antrum.

THE URGENCY OF THE PROBLEM. With the development of medical technologies, to date, it has been established that the main biomarker of diseases of the upper floor of the digestive tract is the presence of H. pylori on the gastric mucosa [9]. H. pylori-associated pathologies are "new" infections that affect the organs of the upper gastrointestinal tract [2].

According to various authors [5], H. pylori is one of the most common infections in the world and correlates with age. A group of researchers led by R.N. Nunt from the World Organization of Gastroenterology (OMGE), in the course of many years of comprehensive study of the problem, came to the conclusion that H. pylori-associated diseases have a global scale, directly proportional to the level of development of countries [3, 7].

Thus, H. pylori has a much higher prevalence in developing countries and less in developed ones. In general, the global distribution of H. pylori in the world exceeds 50%. Factors influencing the number and prevalence of H. pylori infection are age, ethnicity, gender, geographic location, and social and economic characteristics of different segments of the population.

In chronic gastritis, lymphoid follicles increase plasma cell populations. With H. pylori invasion, the amount of Th-1 in the lymphoid infiltrate increases, while the number of Th-2 cells remains unchanged [2, 5]. In response to H. pylori infection, both a local inflammatory reaction of the gastric mucosa and systemic immune and autoimmune responses of the body are traced in the form of activation of cellular and humoral immunity, an increase in autoantibody titers to the pathogen and glandular cells of the gastric mucosa, leading to its atrophy [1 , 3]. In H. pylori-associated atrophic gastritis, the surface epithelium produces less mucus, which increases IgA secretion and often leads to the development of intestinal metaplasia [4]. Morphologically, H. pylori is not detected in areas of intestinal metaplasia, but it is present in large numbers in the border mucosa [6]. Long-term antigenic exposure and autoimmune inflammation lead to disruption of immunological homeostasis with increased levels of pro-inflammatory cytokines (PUI-6, IL-8, IL-12, TNF-a) responsible for the development of intestinal metaplasia in patients with H. pylori-associated gastric diseases [5,7].

H. pylori infection triggers a number of lymphoproliferative responses in the gastric mucosa in the body: lymphoepithelial damage, the formation of lymphoid follicles, diffuse infiltration, and the formation of low-grade lymphoma - MALT-lymphoma [2, 8].

In the process of life, H. pylori form ammonia from urea, which is involved in the alkalization of the antrum of the stomach. As a result, the process leads to increased secretion of gastrin and hyperproduction of hydrochloric acid HCl, which are based on constant stimulation of parietal cells in the stomach [8]. The acid-forming function of the stomach is characterized by the amount and nature of the secretion of hydrochloric acid, determined in the gastric juice, in which it can be in the form of free or bound hydrochloric acid. Also, some strains of H. pylori secrete cytotoxins that damage the mucosa. These reasons lead to the development of antral gastritis, gastric metaplasia, H. pylori migration [9].

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The purpose of this work was to study the acid-producing function of H. pylori-associated diseases of the stomach.

MATERIAL AND METHODS. Determination of the acidity of gastric juice was performed using a microprocessor acidogastrometer "AGM-03" (Istok system, Russia) in 232 patients with H. pylori-associated diseases of the stomach, verified by PCR diagnostics. Nosological groups included 84 patients with chronic non-atrophic gastritis, chronic atrophic gastritis-41, gastric ulcers-31, gastric cancer-26 and 42 patients with MALT-lymphoma.

Their acidity was determined using antimony-platinum probes during diagnostic gastroscopy. The method of simultaneous pH-metry was based on the determination of the concentration of free hydrogen ions in the stomach, which made it possible to draw a conclusion about the acid-forming function of the stomach.

As a result, it was found that in all nosological groups there were changes in the acid-forming and acid-neutralizing functions of the stomach (Table 1). Thus, pH values in the zone of active acid formation, corresponding to the zones of the anterior and posterior sections of the body of the stomach and the "lake", were in the range from 0.9 to 1.8 units. In the zone of acid neutralization corresponding to the antrum of the stomach, these values were within the range of acidity pH from 4.1 to 3.9 units.

Table 1
Indicators of pH-metry in patients with H. pylori-associated diseases of the stomach, n=232.

Nosology	Localization of points in the stomach					
	"lake"	vault	body, back wall	body anterior wall	antrum small curvature	antrum large curvature
CHNAG, n =84	0.6	0.5	0.6	0.3	3.2	3.5
CHAG, n =49	2.5	5.0	2.9	2.9	7.2	6.9
JABZH, n =31	0.7	0.6	0.4	0.5	3.3	3.0
MALT-lymphoma, n =42	1.3	0.8	0.6	0.4	4.0	3.5
HR, n =26	3.9	0.4	0.6	0.7	2.9	3.1
Total, n =232	1.8	1.4	1.0	0.9	4.1	3.9

Note: n is the number of patients, CNAG - chronic non-atrophic gastritis , CAH - chronic atrophic gastritis, PUD - gastric ulcer, RJ - gastric cancer, MALT-lymphoma - mucosal -associated lymphoid tumor of the stomach.

During the examination, in patients with chronic non-atrophic gastritis, an increase in pH values was observed compared to similar indicators of acidity in other nosological groups towards hyperacidity. This resulted in a decrease in the rate of acid formation in the anterior and posterior walls of the body of the stomach and the "lake", so the pH values were below 0.6 units, and in the zone of acid neutralization of the antrum, where the pH values averaged 3.2-3.5 units According to the difference in pH values in the zone of active acid formation and acid neutralization in patients with chronic non-atrophic gastritis, a compensated violation of the acid-neutralizing function of the stomach was observed.

Comparative analysis of gastric acidity indices in patients with chronic atrophic gastritis showed some differences. This resulted in a more reduced hyperacidity of gastric juice in patients with atrophy of the gastric mucosa than in patients with chronic non-atrophic gastritis. In the anterior and posterior walls of the antrum of the stomach, the average pH values in patients with chronic non-atrophic gastritis were almost twice as high as in patients with chronic atrophic gastritis. Accordingly, the rate of impaired acid neutralization, obtained from the difference in pH values of the gastric corpus and gastric antrum, appeared to be significantly greater in chronic non-atrophic gastritis. In chronic atrophic gastritis, a hypoacid state was observed, its average value was 2.5-2.9 units, and the acid neutralizing function was preserved.

With gastric ulcer, patients showed hyperacidity (0.4-0.7 units), a decrease in acid neutralizing function, which amounted to 3.0-3.3 units. Moderate

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disturbances in acid formation were observed in patients with MALT-lymphoma, since they were found to have normacidity and a moderate increase in acid formation, on average, acidity varied from 0.6 to 1.3 units, acid neutralizing function was moderately reduced according to compared with chronic atrophic gastritis, gastric ulcer and gastric cancer.

In patients with gastric cancer, we observed disturbances in the function of the stomach in acid formation and acid neutralization. The average indicator of acid formation in the stomach varied from 0.4 to 3.9 units, that is, some showed increased acidity, while others showed hypoacidity. The acid-neutralizing function of the stomach in gastric cancer was reduced compared to other nosological groups.

Since the arch of the stomach is considered to be the active zone of acid formation and the antrum of the stomach is considered to be the zone of active production of alkaline secretion, we, in further studies, focused specifically on the pH values in these topographies. At the same time, in assessing the level of acidity, we were guided by generally accepted data on pH in the body and proximal stomach, where its values corresponded to:

- pH >5.0 – anacidic state,
- pH 5.0-2.1 - hypoacid state,
- pH 1.2-2.0 - normative state,
- pH <1.2 - hyperacid state,

And at pH >5.0 in the antrum of the stomach, the alkalizing function was considered preserved. Indicators of pH-metry in the body of the stomach amounted to 1.3-1.7 units. - normative state; pH within 1.7-3.0 units. indicated a hypoacid state; pH over 3.0 units indicated an anacid state, pH values < 1.3 units. characteristic of a hyperacid state of acidity in the stomach. In the pyloric section, with normal acid-forming function of the stomach, pH < 2.5 units.

FINDINGS. Thus, the conducted scientific studies have shown that in chronic non-atrophic gastritis, a hyperacid state of gastric juice is noted according to the pH values. Also, in patients with atrophic gastritis, sub- and decompensated violations of the acid-neutralizing function of the stomach are noted in the gastric juice. At the same time, the arch and antrum of the stomach are the most informative parameters for measuring pH in the stomach.

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