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Research Article

Acid-Dependent Diseases of the Gastrointestinal Tract: The Role of Disorders of the Motor Function of the Gastrointestinal Tract

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Abstract

Introduction: Acid-dependent diseases, including gastroesophageal reflux disease (GERD), are the most common pathologies of the gastrointestinal tract.

Purpose: To reveal the role of motor disorders of the gastrointestinal tract in the development of acid-dependent conditions (Barrett's esophagus - PB. YAZH, PID and pancreatitis - P).

Materials and Methods: The study included 16 patients suffering from comorbid pathology - PB, IU, UC and P. The diagnoses were verified using objective studies (endoscopic, ultrasound, X-ray). The comparison group consisted of patients with gastritis C. The motor function of the gastrointestinal tract was recorded electromyographically.

Results: Motility disorders in CCZ include hypermotor gastric dyskinesia, hypomotor duodenal dyskinesia, and spastic activity of the right and left colons. The results obtained indicate the development of SIBO in the small intestine and dysbiosis in the ascending colon. The identified disorders of the motor function of the gastrointestinal tract are important components of the pathogenesis of comorbid acid-dependent conditions.

***Keywords*:** *Acid-Dependent Diseases; Motor Function; Hypo- and Hypermotor Dyskinesia*

Introduction

Currently, acid-dependent diseases, including gastroesophageal reflux disease (GERD), are the most common pathologies of the gastrointestinal tract (GIT). GERD has a chronic relapsing character and is caused by a violation of the motor-evacuation function of the upper digestive tract [1-3].

It has been shown that duodenogastroesophageal reflux is accompanied by exposure of the mucosa of the esophagus to hydrochloric acid, bile acids, lysolecithin, bilirubin [4,5] and contributes to a pronounced damage to the mucous membrane (CO) of the esophagus and the gradual displacement of cells of the stratified squamous non-squamous epithelium to the metaplastic columnar epithelium. Barrett's esophagus (BE) is established in the presence of an area of coverage of the esophagus with metaplastic epithelium of more than 3 cm.

However, the problem of diagnosing BE was complicated by the detection of short segments of cylindrical epithelium in the esophagus, which were determined in 42% of cases in patients with adenocarcinoma of the gastric cardia.

Risk factors that predispose to the development of BE include obesity, hiatal hernia, male gender, and Caucasian ethnicity. The factors of increased risk of developing BE include: prolonged (more than 10 years) existence of reflux esophagitis, the occurrence of reflux at night (horizontal position of the patient), the occurrence of complications - peptic ulcers of the esophagus, strictures, bleeding. Patients with BE are often diagnosed with hiatal hernia (HH), a decrease in the level of epidermal growth factor in saliva and the mucosa of the esophagus. The primary source for specialized columnar epithelium in PB are pluripotent stem cells. They migrate to the lesions of the esophageal mucosa and differentiate into cells of the columnar epithelium, replacing the stratified squamous epithelium.

Peptic ulcer (PU) is a chronic acid-dependent relapsing disease prone to progression with a polycyclic course, the characteristic features of which are seasonal exacerbations, accompanied by the appearance of a peptic ulcer in the mucous membrane of the stomach (G) or duodenum (DK) and the development of life-threatening complications.

PU occurs as a result of a disorder of the general and local mechanisms of the nervous and humoral regulation of the functions of the gastroduodenal system, trophic disorders and activation of proteolysis in the mucous membrane of the stomach and DC. The relationship between the factors of protection and the factors of aggression with the predominance of the latter is violated. Aggression factors include: an increased concentration of hydrogen ions and active pepsin (proteolytic activity), Helicobacter pylori infection, the presence of lysolecithin and bile acids [6,7].

The protective factors include: the amount of protective mucus proteins, especially insoluble and premucosal, secretion of bicarbonates ("alkaline flush"), mucosal resistance: proliferative index of gastroduodenal CO, local immunity (the amount of secretory immunoglobulin A), the state of microcirculation and the level of prostaglandins.

The main factor of the disease includes: Disruption of humoral and neurohormonal mechanisms that regulate digestion and tissue regeneration, disorders of local digestive mechanisms, changes in the structure of SM and DC [8-10].

Violation of nervous trophism is a prerequisite for ulcer formation: SO is most susceptible to dystrophies of neurogenic origin. Active protein-synthetic function is easily disturbed and may be an early sign of dystrophic processes aggravated by aggressive acid-peptic factors of gastric juice.

As shown by electron microscopy data, the number of cholinergic vesicles in the nerve endings of the gastric mucosa increases, indicating the activation of the parasympathetic system, and the number of adrenergic granules decreases, which indicates a decrease in the activity of the sympathetic system. Considering that the parasympathetic vagus nerve provides cell differentiation, and gastrin - their proliferation, it becomes clear that vagal impulses cause accelerated cell maturation, premature aging and death of young cells, which leads to a decrease in the resistance of SM and DC.

Among the regulatory amines, histamine, serotonin and kinins play a significant role in ulcer formation, increasing the permeability of cells and blood vessels, activating the activity of parietal cells through histamine, which produce hydrochloric acid and, through gastrin, increase acid formation in the stomach.

The development of peptic ulcer is associated with secretin deficiency. The hormone secretin inhibits the acid-forming function of the stomach, stimulates the production of bicarbonates and water by the pancreas. With secretin insufficiency and hypersecretion of hydrochloric acid, acidic contents enter the stomach into the duodenum, which is not sufficiently neutralized with secretin insufficiency by bicarbonates, as a result of which the DC resistance is impaired and an ulcer is formed.

In case of peptic ulcer disease, there is also a deficiency of neurotensin, which, in turn, inhibits the secretion of hydrochloric acid. In a certain part of patients, it can be considered as a primary immunodeficiency, since the content of secretory immunoglobulin A in the gastric juice decreases [4].

Motor-evacuation disorders are of great importance in the development of Barrett's esophagus (BE) and peptic ulcer. Duodenogastric reflux of bile contributes to the formation of antral gastritis and gastric ulcers as a result of the cytolytic action of bile acids and lysolecithin, and the accelerated passage of acidic contents into the DC contributes to ulceration of its mucous membrane.

Movement disorders of the DC create conditions for acid-peptic damage to the SD of the DC and reflux of duodenal contents into the pancreatic duct with the development of premature activation of pancreatic enzymes, which ultimately leads to the development of pancreatitis.

Purpose of the Study

To reveal the role of motor disorders of the gastrointestinal tract in the development of acid-dependent conditions (Barrett's esophagus - BE. GI, UC and pancreatitis - P).

Materials and Methods

The study included 16 patients suffering from comorbid pathology - PB, YA. YADK and P. The diagnoses were verified using objective studies (endoscopic, ultrasound, X-ray). The comparison group consisted of patients with gastritis C. Exclusion criteria: patients with pathology of the mental sphere; patients with oncological diseases; patients with acid-dependent diseases not included in the study: reflux esophagitis, reflux gastritis; pregnant women; patients who refused to conduct electromyography (EMG).

The motor function of the gastrointestinal tract was recorded electromyographically using bipolar silver electrodes placed on the anterior abdominal wall in the area of the projection of the recorded organ (stomach, duodenum, right and left sections of the colon).

Statistical analysis was performed by the Mann-Whitney small sample method at p 0.05.

Research Results and Discussion

The results of studying the motor activity of the stomach in acid-dependent diseases are presented in table 1.

Research Group	Slow waves			Spike activity			Propulsive activity
	Frequency	Wave amplitude	Power of tonic contractions	Frequency	Wave amplitude	Power of phase contractions	
Acid dependent diseases	8,2 ± 0,9	0,36 ± 0,004	2,952 ± 0 0,0112	1,6 ± 0,3	0,19 ± 0,002	0,304± 0.015	9,7± 0,6
Control	5,5 ± 0,4	0,15 ± 0,002	0,825 ± 0,032	1,0 ± 0,04	0,1 ± 0,002	0,1 ± 0,004	8,25 ± 0,13

Table 1: *Parameters of electromyography of smooth muscles of the stomach in patients with acid-dependent diseases in various conditions.*

From table 1 it follows that in acid-dependent diseases, the propulsive activity of the stomach in patients with acid-dependent diseases is increased by 17.6% ($p < 0.05$), the power of tonic contractions of the longitudinal muscle layer is increased by 49% ($p < 0.05$), the power phase contractions of the circular muscle layer increased by 204% ($p < 0.001$). That is, in acid-dependent comorbid diseases (CCD), hypermotor dyskinesia of the stomach is observed, which, with retrograde propagation of an electrical wave, contributes to the

regurgitation of gastric contents into the esophagus with the development of reflux esophagitis and BE. In addition, there is a pronounced hypersensitivity of the circular muscle layer, which determines the pain syndrome in the epigastric region observed in patients with CC.

The results of the study of the motor activity of the duodenum under various conditions are presented in table 2.

Research Group	Slow waves			Spike activity			Propulsive activity
	Frequency	Wave amplitude	Power of tonic contractions	Frequency	Wave amplitude	Power of phase contractions	
Acid dependent diseases	8,82 ± 0,9	0,3 ± 0,05	2,646 ± 0,183	1,22 ± 0,2	0,14 ± 0,006	0,1708 ± 0.0021	15,4 ± 1,3
Control	22,0 ± 0,9	0,1 ± 0,003	2,2 ± 0,13	1,0 ± 0,04	0,1 ± 0,002	0,1 ± 0,003	22,0 ± 1,7

Table 2: *Parameters of electromyography of smooth muscles of the duodenum in patients with acid-dependent diseases in various conditions.*

From table 2 it follows that the propulsive activity of the duodenum in patients with CC is reduced by 30% ($p < 0.05$), the power of tonic contractions of the longitudinal muscles in CC is increased by 20.3% compared with the control ($p < 0.05$), the power of phase contractions - by 70.8% ($p < 0.05$). That is, hypomotor dyskinesia of the duodenum was revealed in CC, indicating the development of duodenostasis with an increase in intraluminal pressure, contributing to the regurgitation of duodenal contents into the stomach and esophagus and increasing pressure in the pancreatic duct with the development of pancreatitis.

Hypermotor dyskinesia of the stomach leads to disruption of antro-duodenal coordination and acceleration of the flow of acidic gastric contents into the duodenum with the development of peptic ulcer.

The results of the study of the motor activity of the right sections of the colon in comorbid acid-dependent diseases are presented in table 3.

Research Group	Slow waves			Spike activity			Propulsive activity
	Frequency	Wave amplitude	Power of tonic contractions	Frequency	Wave amplitude	Power of phase contractions	
Acid dependent diseases	5,1 ± 0,7	0,13 ± 0,002	0,663 ± 0,032	1,1 ± 0,09	0,16 ± 0,003	0,176 ± 0,012	3,8 ± 0,4
Control	11,0 ± 1,2	0,1 ± 0,004	1,1 ± 0,05	1,0 ± 0,02	0,1 ± 0,003	0,1 ± 0,004	11,0 ± 1,0

Table 3: *Parameters of electromyography of smooth muscles of the right sections of the colon in patients with comorbid acid-dependent diseases in various conditions.*

From table 3 it follows that the propulsive activity of the right sections of the colon in patients with CC is reduced by 65.4% ($p < 0.05$), the power of tonic contractions of the longitudinal muscles is reduced compared to the control by 39.7% ($p < 0. 05$), the power of phase contractions increased by 76% ($p < 0.05$). That is, with CRC, hypomotor dyskinesia of the right sections of the colon was revealed, which may indicate the development of intestinal dysbiosis.

The results of the study of motor activity of the left sections of the colon in CCZ are presented in table 4.

Research Group	Slow waves			Spike activity			Propulsive activity
	Frequency	Wave amplitude	Power of tonic contractions	Frequency	Wave amplitude	Power of phase contractions	
Acid dependent diseases	6,1 ± 0,9	0,16 ± 0,002	0,976 ± 0,004	0,9 ± 0,03	0,1 ± 0,02	0,09 ± 0.003	10,8 ± 1,2
Control	6,0 ± 0,5	0,1 ± 0,003	0,6 ± 0,05	1,0 ± 0,02	0,1 ± 0,002	0,1 ± 0,004	6,0 ± 0,2

Table 4: *Parameters of electromyography of smooth muscles of the left sections of the colon in patients with co-morbid acid-dependent conditions in various conditions.*

From table 4 it follows that the propulsive activity of the left sections of the colon in patients with acid-dependent diseases is increased by 80% ($p < 0.001$), the power of tonic contractions of the longitudinal muscles in CCZ is increased compared to the control by 62.7% ($p < 0.05$), the power of phase contractions of the circular muscle layer was reduced by 10% ($p < 0.05$). The presence of spastic contractions of the longitudinal muscles of the left sections and the circular muscles of the right sections of the colon determine the development of constipation. That is, with CC, a violation of the motor function of the digestive tract, expressed to varying degrees, was revealed: Motility disorders in CCZ include hypermotor gastric dyskinesia and hypomotor intestinal dyskinesia and spastic activity of the right and left colons. The results obtained indicate the development of SIBO in the small intestine and dysbiosis in the ascending colon.

Attention is drawn to the presence of duodenostasis in CC. As is known, with duodenostasis, intracavitary pressure in the duodenum increases, which leads to reflux of duodenal contents into the bile duct, which changes the flow of bile components, in particular bile acids, into the duodenum with a decrease in intestinal motility and the development of SIBO due to a violation of the bactericidal functions of duodenal contents.

Reflux of the contents of the duodenum and pancreatic duct leads to premature activation of proteolytic enzymes of the pancreas, the intake of lysolecithin, bile acids and emulsified fats and bacteria, which generally leads to the development of pancreatitis. The entry into the pancreatic duct of bile containing active pancreatic enterokinase leads to the development of destructive pancreatitis with subsequent chronification of the process. Thus, impaired motility of the upper digestive tract is an important component of CCZ - reflux esophagitis and BE, frank gastric and duodenal disease, and chronic pancreatitis.

Conclusion

1. Comorbid acid-dependent conditions of the upper digestive tract are characterized by hypermotor dyskinesia of the stomach, which, along with other factors, determines the development of Barrett's esophagus.
2. In acid-dependent diseases, hypomotor dyskinesia of the duodenum was revealed, which indicates the development of a syndrome of excessive bacterial growth and duodeno-pancreatic reflux, leading to the development of pancreatitis.
3. Hypermotor dyskinesia of the stomach leads to disruption of antro-duodenal coordination and acceleration of the flow of acidic gastric contents into the duodenum with the development of peptic ulcer.
4. Spastic activity of the right and left sections of the colon was revealed, which indicates the development of dysbiosis in the ascending section of the colon and constipation.

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