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Mini Review

Pancreatic Lipomatosis: Focus on the Motor Function of the Gastrointestinal Tract and Biliary Tract

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Abstract

The aim is to identify the role of disorders of the motor function of the gastrointestinal tract and the gastrointestinal tract in pancreatic lipomatosis.

There were 9 patients with pancreatic lipomatosis under observation, women aged 69.3 ± 7.3 years and men aged 57.0 ± 6.4 years. In 66.7% of patients, obesity of 2 - 3 degrees was noted. The diagnosis of pancreatic lipomatosis was established endosographically. The comparison group consisted of 10 patients with chronic gastritis C.

The motor function of the gastrointestinal tract and the gastrointestinal tract was recorded electromyographically by installing bipolar silver electrodes with a diameter of 0.3 - 0.5 mm on the anterior abdominal wall in the projection area of the organ. The amplitude-frequency characteristics of slow waves and spikes, the power of phase and tonic contractions, and the propulsive activity were analyzed using a Nihon Kohen electromyograph (Japan).

Hypomotor dyskinesia of the stomach, which determines the development of functional dyspepsia, was revealed.

Hypermotor dyskinesia of the duodenum and jejunum, the left colon indicates the possible development of dysbiosis of the small and large intestine.

Keywords: *Electromyography; Pancreas; Hypermotor Dyskinesia*

Introduction

Chronic pancreatitis is characterized by the versatility and complexity of its etiology and pathogenesis. Depending on the etiological factor, primary pancreatitis is isolated, in which the pathological process is localized directly in the pancreas, and secondary, resulting from diseases of other digestive organs.

Alcoholic, alimentary, medicinal, post-traumatic, operational factors play a role in the etiology of primary pancreatitis. Secondary inflammatory lesion of the pancreas occurs due to pathology of the biliary tract, liver, gastrointestinal tract (gastrointestinal tract), inflammatory and sclerotic changes of the large duodenal papilla. A Y-shaped dependence on daily fat intake is shown: in highly developed Western countries, this variant of pancreatitis affects mainly men from affluent segments of the population aged 30 - 40 years [1,2].

According to the etiological classification of chronic pancreatitis TIGAR - 0 distinguish:

- Toxic-metabolic
- Alcoholic
- Nicotine
- Hypercalcemic
- Hyperlipidemic
- Medical
- Toxic (including organic components)
- With chronic renal failure.

Chronic pancreatitis is characterized by an inhomogeneous structure of the gland, the density of which may vary, uneven contours may be noted, but no pronounced deformation is observed.

There are numerous methods of pancreatic examination [3]: Ultrasound with the determination of contours, sizes and density of pancreatic tissue.

X-ray with the determination of calcinates in the pancreatic tissue and duodenal X-ray to determine duodenoastric reflux or transient duodenostasis.

Endoscopic- To study the area of the large duodenal papilla and identify possible pathology of the stomach and duodenum.

Computed tomography is the detection of an inhomogeneous structure of the gland, the gland is often uniformly enlarged, less often atrophied, and pseudocysts can be determined by it.

ERCP- Detection of pathology of the large duodenal papilla, calculosis of the biliary tract.

Morphological examination to detect inflammatory cell infiltration in combination with fibrous changes, the presence of lipid inclusions in exocrinocytes.

Laboratory diagnostics.

However, one of the manifestations of chronic pancreatitis, including pancreatic lipomatosis, is an external secretory insufficiency that causes the development of diarrhea. At the same time, there is not enough data on the study of the motor function of the gastrointestinal tract and biliary tract (VVP).

The aim is to identify the role of disorders of the motor function of the gastrointestinal tract and the gastrointestinal tract in pancreatic lipomatosis.

Materials and Methods

There were 9 patients with pancreatic lipomatosis under observation, women aged 69.3 ± 7.3 years made up two thirds of the patients and men aged 57.0 ± 6.4 years made up one third of the patients. Diverticular disease was observed in 66.7% of patients, esophagitis, duodenogastric reflux, obesity of 2-3 degrees, stool disorders in 33.4% of cases. The diagnosis of pancreatic lipomatosis was established endosographically. The comparison group consisted of 10 patients with chronic gastritis.

The motor function of the gastrointestinal tract and the gastrointestinal tract was recorded electromyographically by installing bipolar silver electrodes with a diameter of 0.3 - 0.5 mm on the anterior abdominal wall in the projection area of the organ. The amplitude-frequency characteristics of slow waves and spikes, the power of phase and tonic contractions, and the propulsive activity were analyzed using a Nihon Kohen electromyograph (Japan). Statistical analysis was carried out using the Statistics - 15 software package.

Results

The frequency of slow stomach waves was 6.7 ± 0.5\min (an increase of 21.8%. $p < 0.05$), the amplitude was 0.15 ± 0.003 mV (within the reference values), the power of tonic contractions was 1.005 ± 0.11 (an increase of 21.8%, $p < 0.05$). The frequency of spikes was 3.0 ± 0.4 (an increase of 199.8%, $p < 0.001$), the amplitude was 0.07 ± 0.002 mV (a decrease of 29.8%, $p < 0.05$), the power of phase contractions was 0.21 ± 0.013 (an increase of 110%, $p < 0.003$), the propulsive activity was 4.8 ± 0.4 (a decrease of 41.8%, $p < 0.05$). That is, hypomotor dyskinesia of the stomach was detected with pancreatic lipomatosis.

Electromyographically, the frequency of slow duodenal waves was 24.5 ± 2.6\min (an increase of 11.4%, $p < 0.05$), the amplitude was 0.12 ± 0.003 mV (an increase of 20%, $p < 0.05$), the power of tonic contractions was 2.94 ± 0.18 (an increase of 33.6%, $p < 0.05$). The frequency of spikes was 1.9 ± 0.2 (increase by 90.1%, $p < 0.04$), amplitude - 0.03 ± 0.001 mV (decrease by 69.8%, $p < 0.05$), power of phase contractions - 0.057 ± 0.004 (decrease by 43%, $p < 0.05$), propulsive activity - 51.5 ± 2.8 (increase by 161%, $p < 0.001$) (Figure 1). That is, with pancreatic lipomatosis, hypermotor dyskinesia of the duodenum was revealed due to a decrease in the tone of the circular muscles.

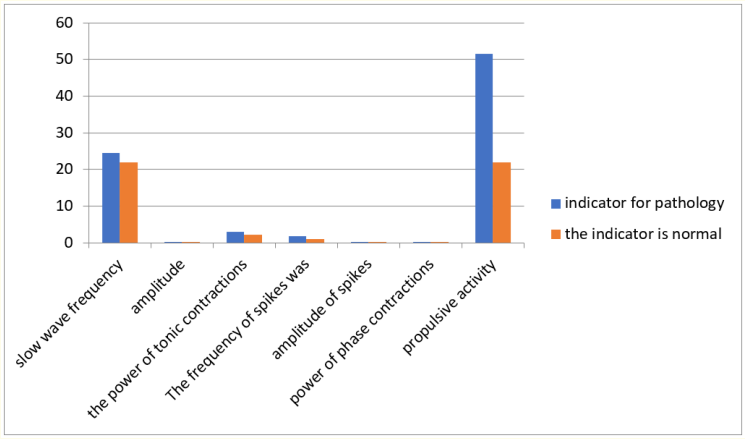

Figure 1: *Electromyographic parameters of duodenal waves in pancreatic lipomatosis.*

In the jejunum, the frequency of slow waves was 18.0 ± 2.5\min (decrease by 10%, $p < 0.05$), the amplitude was 0.14 ± 0.003 mV (increase by 40.1%, $p < 0.05$), the power of tonic contractions was 2.52 ± 0.17 (increase by 26%, $p < 0.05$). The frequency of spikes was 1.9 ± 0.5 (an increase of 89.8%, $p < 0.05$), the amplitude was 0.03 ± 0.002 mV (a decrease of 70.1%, $p < 0.05$), the power of phase contractions was 0.057 ± 0.003 (a decrease of 43%, $p < 0.05$), the propulsive activity was 44.2 ± 1.8 (an increase of 121%, $p < 0.001$). That is, with pancreatic lipomatosis, hypermotor dyskinesia of the jejunum is observed due to a decrease in the tone of the circular muscle layer.

In the right parts of the colon, the frequency of slow waves was 13.3 ± 1.5\min (an increase of 20.9%, $p < 0.05$), the amplitude was 0.18 ± 0.004 mV (an increase of 79.9%, $p < 0.05$), the power of tonic contractions was 2.394 ± 0.19 (an increase of 117.6%, $p < 0.001$). The frequency of spikes was 2.7 ± 0.3 (increase by 170%, $p < 0.001$), amplitude - 0.09 ± 0.003 mV (decrease by 10%, $p < 0.05$), power of phase contractions - 0.243 ± 0.031 (increase by 143%, $p < 0.001$), propulsive activity - 9.8 ± 0.7 (decrease by 10.9%, $p < 0.05$). That is, with pancreatic lipomatosis, hypomotor dyskinesia of the right colon was observed due to pronounced spasm of the circular muscles.

In the left colon, the frequency of slow waves was 12.0 ± 1.7\min (an increase of 199.8%, $p < 0.001$), the amplitude was 0.18 ± 0.004 mV (an increase of 79.9%, $p < 0.05$), the power of tonic contractions was 2.16 ± 0.14 (an increase of 260%, $p < 0.001$). The frequency of spikes was 2.4 ± 0.08 (increase by 140.2%, $p < 0.001$), amplitude - 0.08 ± 0.002 mV (decrease by 20%, $p < 0.05$), power of phase contractions - 0.192 ± 0.016 (increase by 92%, $p < 0.04$), propulsive activity - 11.2 ± 1.4 (increase by 86.7%, $p < 0.05$). That is, with pancreatic lipomatosis, hypermotor dyskinesia of the left colon was detected, which is clinically accompanied by the development of diarrhea.

Electromyographically, in the sigmoid colon, the frequency of slow waves was 10.0 ± 0.9\min (an increase of 99.8%, $p < 0.04$), the amplitude was 0.18 ± 0.002 mV (an increase of 79.9%, $p < 0.05$), the power of tonic contractions was 1.8 ± 0.03 (an increase of 260%, $p < 0.001$). The frequency of spikes was 4.0 ± 0.3 (increase by 299.8%, $p < 0.001$), amplitude - 0.03 ± 0.004 mV (decrease by 70%, $p < 0.05$), power of phase contractions - 0.12 ± 0.03 (increase by 20%, $p < 0.05$), propulsive activity - 15.0 ± 1.3 (increase by 199.8%, $p < 0.001$). That is, with pancreatic lipomatosis, hypermotor dyskinesia of the sigmoid colon was observed, which determines the development of diarrhea.

The frequency of slow choledoch waves was 8.7 ± 0.6\min (decrease by 3.3%, $p > 0.1$), the amplitude was 0.1 ± 0.002 mV (within the reference values), the power of tonic contractions was 0.87 ± 0.05 (decrease by 3.3%, $p > 0.1$). The frequency of spikes was 2.4 ± 0.3 (increase by 140.1%, $p < 0.001$), amplitude - 0.02 ± 0.001 mV (decrease by 80.1%, $p < 0.05$), power of phase contractions - 0.048 ± 0.005 (decrease by 52%, $p < 0.05$), propulsive activity - 18.1 ± 2.1 (increase by 101%, $p < 0.001$).

Electromyographically, the frequency of slow gallbladder waves was 7.4 ± 0.7\min (decrease by 7.5%, $p < 0.05$), the amplitude was 0.1 ± 0.003 mV (within the reference values), the power of tonic contractions was 0.74 ± 0.06 (decrease by 7.5%, $p < 0.05$). The frequency of spikes was 1.9 ± 0.13 (increase by 89.9%, $p < 0.04$), amplitude - 0.04 ± 0.002 mV (decrease by 59.9%, $p < 0.05$), power of phase contractions - 0.076 ± 0.008 (decrease by 24%, $p < 0.05$), propulsive activity - 9.86 ± 0.9 (increase by 23.2%, $p < 0.05$). That is, hypermotor dyskinesia of the biliary system was observed in pancreatic lipomatosis.

Discussion

The study showed that the nature of changes in the motor activity of the gastrointestinal tract is determined by the position of the organ in the digestive transport conveyor: hypomotor dyskinesia of the stomach, accompanied by dyspeptic syndrome in the clinic, was revealed. The external secretory insufficiency developing with pancreatic lipomatosis with an insufficient number of enzymes and bicarbonates disrupt intracavitary digestion in the small intestine and further in the colon, which is electromyographically manifested by an increase in the propulsive activity of the left colon and sigmoid colon.

A decrease in bicarbonate secretion by the pancreatic duct epithelium leads to acidification of the contents of the duodenum, resulting in denaturation of pancreatic enzymes and precipitation of bile acids. The mixing of enzymes with food chyme is disrupted, due to motor disorders of the duodenum and jejunum, inactivation of enzymes due to excessive growth of microflora. Electromyographically, this is manifested by pronounced hypermotor dyskinesia of the duodenum and jejunum.

Conclusion:

1. Electromyography can be used to analyze the motor activity of the gastrointestinal tract and the gastrointestinal tract in pancreatic lipomatosis.
2. Revealed hypomotor dyskinesia of the stomach, which determines the development of functional dyspepsia.
3. Hypermotor dyskinesia of the duodenum and jejunum, the left colon indicates the possible development of dysbiosis of the small and large intestine.

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