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Review Article

Motor Function of the Gastrointestinal Tract and Biliary Tract in Primary Sclerosing Cholangitis Associated with Inflammatory Bowel Diseases

Lychkova AE*, Ashrafov RA, Ashrafova SR, Puzikov AM

Department of Health, Moscow Clinical Research and Practice Center, Named after A. S. Loginov of Moscow, Moscow, Russia

***Corresponding author:** Lychkova AE, Department of Health, Moscow Clinical Research and Practice Center, Named after A. S. Loginov of Moscow, Moscow, Russia

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Summary

The Aim: Is to identify the features of the motor activity of the gastrointestinal tract and the gastrointestinal tract in combined IBD - PSC. **Materials and Methods:** 10 patients with PSC - IBD were under observation, and the ratio of men: women is 2:3. Clinically, patients (PSC - CD) had ileum lesion to varying degrees, total damage to the colon, stool from 1 to 3 times a day without blood. The motor function of the gastrointestinal tract and the gastrointestinal tract in the studied patients was recorded electromyographically using the Nihon Kohden hardware and software complex (Japan). Statistical analysis was performed by the Mann-Whitney small sample method at $p<0.05$. **Results:** With PSC - IBD, hypermotor gastric dyskinesia of a weak degree is observed with a proportional increase in the motor function of the longitudinal, circular and oblique muscles. Hypomotor dyskinesia was detected in the duodenum. High hypermotor dyskinesia was detected in the jejunum, and moderately pronounced in the iliac. Signs of dysbiosis were revealed in the cecum and in the ascending colon. Hypermotor dyskinesia was detected in the descending colon and sigmoid. The lack of propulsive activity of choledochus in PSC - IBD is compensated by hypermotor dyskinesia of the gallbladder, which supports the motility of the duodenum and jejunum due to the intake of concentrated bile into the intestine.

Keywords: Inflammatory bowel diseases; Primary sclerosing cholangitis

Among the extra-intestinal manifestations of Inflammatory Bowel Diseases (IBD) granulomatous hepatitis, cholestatic hepatitis and primary sclerosing cholangitis have been described in patients primarily with Crohn's disease. H.H. Rasmussen, et al. (1997) studied the prevalence of Primary Sclerosing Cholangitis (PSC) in 262 patients with Crohn's disease for 15 years and concluded that in Crohn's disease PSC occurs with the same frequency as in Ulcerative Colitis (UC). The study of the nature of the relationship between PSC and IBD showed that the associated forms of PSC - IBD may represent a special phenotype in addition to previously established and defined phenotypes. Three loci of UC susceptibility have been identified that may be associated with PSC and were associated with the putative candidate genes REL, IL 2, CARD9 [1,2] moreover, there are indications of overlapping but different genetic mechanisms of PSC and IBD. The model of pathogenesis of PSC and IBD is based on intestinal translocation of microbial flora with subsequent activation of the immune system and inflammation of the biliary tract. Activated intestinal lymphocytes enter the enterohepatic circulation and are stored as memory cells capable of initiating an inflammatory process in the liver. Chemokines and adhesion molecules can interact with immune complexes both in the liver and in the intestine.

The clinical course of colitis in patients with and without PSC has a number of differences. Colitis associated with PSC is characterized by a milder course, a more significant lesion of the proximal colon and less pronounced changes (60-70%) from the rectum [3]. There are indications of a higher incidence of colorectal cancer in PSC - IBD. However, in general, clinical observations indicate that IBD - UC is an independent disease, different from the classical form of UC [3]. The age of manifestation of IBD in

PSC -IBD, according to various authors, is estimated ambiguously: the period of appearance of the first symptoms of IBD in patients with PSC was 19-24 years, according to V. Zoo et al., in patients with PSC - UC manifests itself at the age of 24.5 years. whereas UC manifests itself at the age of 33.8 years [4]. The course of PSC associated with IBD differs from PSC without IBD in several aspects: PSC - IBD is characterized more often than the isolated form of IBD by pancolitis, absence of rectal lesions, retrograde ileitis, mild course of the disease and malignant lesions of the colon [5]. Intestinal inflammation in UC - IBD is milder than in the classical version of the course of UC without PSC. Morphological examination showed marked cellular infiltration in the cecum and ascending colon in patients with PSC-UC, and the degree of inflammation in the rectum and/or descending colon was minimal.

Despite in-depth genetic and clinical-morphological studies, no detailed description of the motor activity of the gastrointestinal tract (gastrointestinal tract) and biliary tract (biliary tract) in combined forms of PSC - IBD has been found in the available literature. The aim is to identify the features of the motor activity of the gastrointestinal tract and the gastrointestinal tract in combined IBD - PSC.

Materials and Methods

10 patients with PSC - IBD were under observation, and 6 of them at the age of 35.7 ± 4.6 years suffered from PSC - Ulcerative Colitis (PSC - UC), 4 - at the age of 34.5 ± 4.5 years - PSC - Crohn's Disease (CD) (PSC - CD), and the ratio of men: women is 2:3. Clinically, patients (PSC - CD) had ileum lesion to varying degrees, total damage to the colon, stool from 1 to 3 times a day without blood. In patients with PSK - CD, damage to the gallbladder was also noted, an increase in the frequency of stool up to 5-6 times with an admixture of blood. The comparison group consisted of 8 patients with chronic gastritis C, who had pain syndrome after eating without pronounced changes in the stool. The motor function of the gastrointestinal tract and the gastrointestinal tract in the studied patients was recorded by electromyographic. The amplitude-frequency characteristics of slow waves and spikes, the power of phase and tonic contractions, and propulsive activity were recorded using the Nihon Kohden hardware and software complex (Japan). Statistical analysis was performed by the Mann-Whitney small sample method (M±m, $p < 0.05$).

Results

The frequency of slow stomach waves in patients with PSC-IBD was 12.5 ± 0.2 \min (an increase of 127.3%, $p < 0.001$), the amplitude was 0.15 ± 0.006 mV (within the reference values), the power of tonic contractions was 1.875 ± 0.153 (an increase of 127.3%, $p < 0.001$). The frequency of spikes was 2.9 ± 0.3 (an increase of 190%, $p < 0.001$), the amplitude was 0.07 ± 0.004 Mv (a decrease of 30%, $p < 0.05$), the power of phase contractions was 0.203 ± 0.018 (an increase of 103%, $p < 0.002$), the propulsive activity was 9.24 ± 0.62 (an increase of 12.1%, $p < 0.05$). That is, with PSC - IBD, hypermotor gastric dyskinesia of a weak degree is observed with a proportional increase in the motor function of the longitudinal, circular and oblique muscles. In the duodenum, the frequency of slow waves was 21.2 ± 0.8 \min (decrease by 3.6%, $p < 0.05$), the amplitude was 0.1 ± 0.03 mV (within the reference values), the power of tonic contractions was 2.12 ± 0.15 (decrease by 3.6%, $p < 0.05$). The frequency of spikes was 3.3 ± 0.4 (an increase of 230%, $p < 0.001$), the amplitude was 0.04 ± 0.002 mV (a decrease of 60.1%, $p < 0.05$), the power of phase contractions was 0.132 ± 0.011 (an increase of 32%, $p < 0.05$), the propulsive activity was 16.1 ± 0.9 (a decrease of 26.8%. $p < 0.05$). That is, with IBD-PSC, hypomotor dyskinesia of the duodenum was detected.

In the jejunum, the frequency of slow waves was 19.3 ± 0.7 \min (decrease by 3.5%, $p < 0.05$), the amplitude was 0.14 ± 0.003 mV (decrease by 40%, $p < 0.05$), the power of tonic contractions was 2.702 ± 0.12 (increase by 25%, $p < 0.05$). The frequency of spikes was 2.9 ± 0.25 (an increase of 190%, $p < 0.001$), the amplitude was 0.02 ± 0.0015 mV (a decrease of 79.8%, $p < 0.05$), the power of phase contractions was 0.058 ± 0.0037 (a decrease of 42%, $p < 0.05$), the propulsive activity was 46.6 ± 2.4 (an increase of 133%, $p < 0.001$).That is, with PSC - IBD, high hypermotor dyskinesia of the jejunum was detected. In the ileum, the frequency of slow waves was 15.7 ± 1.3 \min (an increase of 12.2%, $p < 0.05$), the amplitude was 0.06 ± 0.003 mV (a decrease of 39.9%, $p < 0.05$), the power of tonic contractions was 0.942 ± 0.081 (a decrease of 32.9%, $p < 0.05$). The frequency of spikes was 2.6 ± 0.2 (an increase of 160%, $p < 0.001$), the amplitude was 0.02 ± 0.003 mV (a decrease of 79.8%, $p < 0.05$), the power of phase contractions was 0.052 ± 0.004 (a decrease of 48%, $p < 0.05$), the propulsive activity was 18.1 ± 1.2 (an increase of 29.3%, $p < 0.05$). That is, with PSC - IBD, moderate hypermotor dyskinesia of the ileum was detected.

The frequency of slow waves of the cecum was 18.5 ± 1.3 \min (an increase of 59.1%, $p < 0.05$), the amplitude was 0.09 ± 0.006 mV (a decrease of 10%, $p < 0.05$), the power of tonic contractions was 1.665 ± 0.13 (an increase of 51.4%, $p < 0.05$). The frequency of spikes was 3.3 ± 0.4 (an increase of 230%, $p < 0.001$), the amplitude was 0.04 ± 0.002 mV (a decrease of 59.8%, $p < 0.05$), the power of phase contractions was 0.132 ± 0.003 (an increase of 32%, $p < 0.05$), the propulsive activity was 12.6 ± 1.4 (an increase of 14.5%, $p < 0.05$). That is, with PSC - IBD, weakly expressed hypermotor dyskinesia of the cecum was detected, which may indicate the presence of dysbiosis of the cecum. In the ascending colon, the frequency of slow waves was 12.0 ± 0.2 \ min (an increase of 9.1%, $p < 0.05$), the amplitude was 0.15 ± 0.003 mV (an increase of 49.8%, $p < 0.05$), the power of tonic contractions was 1.8 ± 0.02 (an increase of 83.6%, $p < 0.05$). The

frequency of spikes was 2.0 ± 0.04 (an increase of 99.8%, $p < 0.01$), the amplitude was 0.01 ± 0.002 mV (a decrease of 89.9%, $p < 0.04$), the power of phase contractions was 0.02 ± 0.004 (a decrease of 79.8%, $p < 0.05$), the propulsive activity was 9.0 ± 0.7 (a decrease of 18.2%, $p < 0.05$). That is, with PSC - IBD, moderate hypomotor dyskinesia of the right colon was revealed, indicating the presence of dysbiosis of the ascending colon.

In the descending colon, the frequency of slow waves was 8.3 ± 0.6 \min (an increase of 38.3%, $p < 0.05$), the amplitude was 0.11 ± 0.02 mV (an increase of 10%, $p < 0.05$), the power of tonic contractions was 0.913 ± 0.062 (an increase of 52.2%, $p < 0.05$). The frequency of spikes was 2.5 ± 0.3 (an increase of 150%, $p < 0.001$), the amplitude was 0.04 ± 0.005 mV (a decrease of 59.8%, $p < 0.05$), the power of phase contractions was 0.10 ± 0.0011 (within the reference values), the propulsive activity was 0.091 ± 0.007 (an increase of 52%, $p < 0.05$). That is, with PSC -IBD, hypermotor dyskinesia of the descending colon was detected. In the sigmoid colon, the frequency of slow waves was 6.3 ± 0.4 \min (an increase of 5%. $p < 0.05$), the amplitude was 0.12 ± 0.013 mV (an increase of 20%, $p < 0.05$), the power of tonic contractions was 0.756 ± 0.051 (an increase of 26%, $p < 0.05$). The frequency of spikes was 4.3 ± 0.2 (an increase of 330%, $p < 0.001$), the amplitude was 0.03 ± 0.002 mV (a decrease of 69.8%, $p < 0.05$), the power of phase contractions was 0.129 ± 0.014 (an increase of 29%, $p < 0.05$). That is, hypermotor dyskinesia of the sigmoid colon, descending colon and sigmoid colon was detected in PSC - IBD.

In the choledoch, the frequency of slow waves was 11.8 ± 0.14 \min (an increase of 31.1% $p < 0.05$), the amplitude was 0.15 ± 0.004 mV (an increase of 49.9%, $p < 0.05$), the power of tonic contractions was 1,770 ± 0.142 (an increase of 96.7%, $p < 0.001$). The frequency of spikes was 3.8 ± 0.35 (an increase of 280%, $p < 0.001$), the amplitude was 0.07 ± 0.003 mV (a decrease of 30%, $p < 0.05$), the power of phase contractions was 0.266 ± 0.014 (an increase of 166%, $p < 0.001$), the propulsive activity was 6.65 ± 0.72 (a decrease of 23.8%, $p < 0.05$) (Figure 1). In the gallbladder, the frequency of slow waves was 10.4 ± 0.6 \ min (an increase of 30%, $p < 0.05$), the amplitude was 0.12 ± 0.007 mV (an increase of 20%, $p < 0.05$), the power of tonic contractions was 1.248 ± 0.113 (an increase of 56%, $p < 0.05$). The frequency of spikes was 2.8 ± 0.03 mV (increase by 180%, $p < 0.001$), amplitude - 0.04 ± 0.005 (decrease by 59.8%, $p < 0.05$), power of phase contractions - 0.112 ± 0.0098 (increase by 12%, $p < 0.05$), propulsive activity - 11.3 ± 1.9 (increase by 41.3%, $p < 0.05$). Thus, the lack of propulsive activity of choledochus in PSC - IBD is compensated by hypermotor dyskinesia of the gallbladder, which supports the motility of the duodenum and jejunum due to the intake of concentrated bile into the intestine.

Figure for the article: motor function of the gastrointestinal tract and biliary tract in primary sclerosing cholangitis associated with inflammatory bowel diseases.

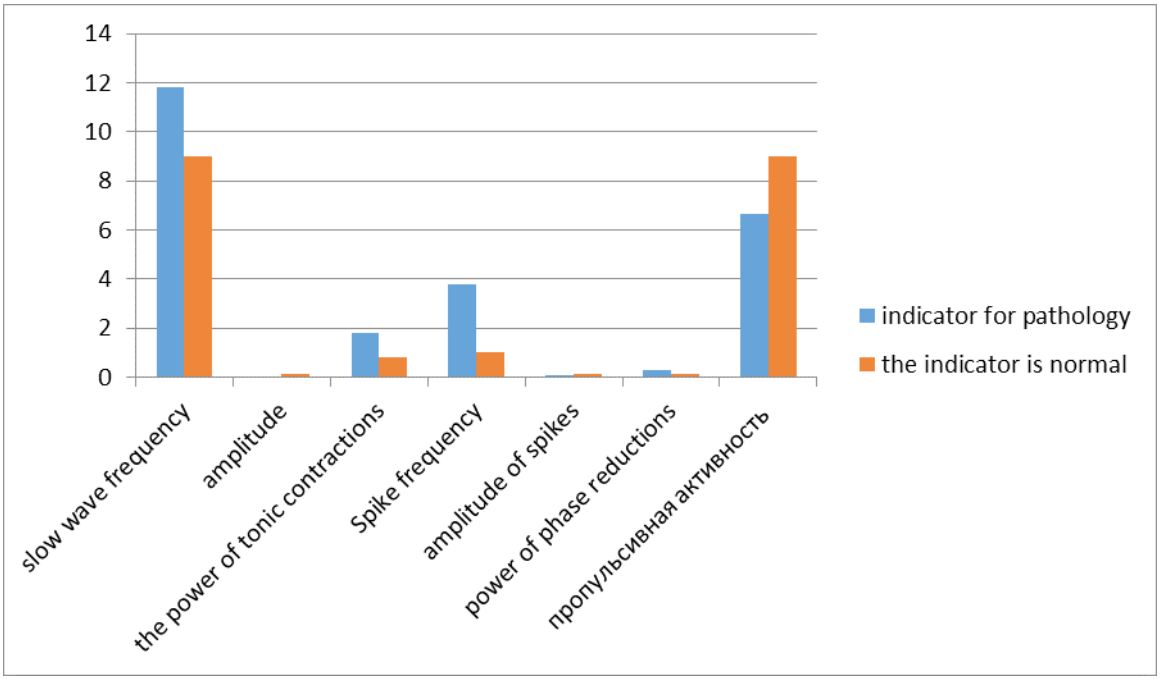

Figure 1: Indicators of electromyography of choledochus in various conditions.

The study showed that hypermotor dyskinesia was observed only in the choledochus and in the ascending colon. In the first case, this condition may occur due to the gradual formation of morphological changes (development of periductal fibrosis) in the biliary tract in PSC. In the second case, it is a consequence of the development of intestinal dysbiosis. In addition, there is a theory of the toxic effects of bile acids, which are released in the fraction of the gallbladder during its hypermotor dyskinesia. Cholesterol, cytokines, altered lipids and proteins that play the role of triggers have a generally recognized potentiating damaging effect [6]. A decrease in membrane resistance under the influence of immuno-mediated influences or the destruction of the connective tissue plate that unites epithelial cells,. it can increase sensitivity to the damaging effects of even normal unchanged bile. Based on the theory of the trigger action of bile acids in the development of PSC, genetically determined or acquired defects of bile transport components are of great importance, for example, the intestinal bile acid transporter - IBAT \ideal bile acid transporter\, which is also called the apical sodium-dependent bile transporter. It is a protein mainly localized in the terminal part of the small intestine, which serves as the main intermediary for the capture of conjugated LC in the small intestine, ensures their return to the liver through the portal vein, plays a key role in the enterohepatic circulation of LC [7].

The data obtained indicate the interest of the muscular wall of the jejunum and ileum in the enterohepatic circulation of bile acids and their possible role in the development of PSC - IBD. Despite the fact that, according to the study, there are no pronounced manifestations of SIBR and intestinal dysbiosis, however, it should be noted the presence of hypermotor liskinesia of the cecum and hypomotor dysfunction of the ascending colon, which may indicate the development of mild intestinal dysbiosis. Factors affecting the cholangiocyte of molecules of microbial origin can play a significant role in the pathogenesis of PSC. Due to the increased exposure of microbial derivatives as a result of impaired permeability of the colon, the role of changes in the microbial spectrum in dysbiosis, as well as the aberrant or excessive response of cholangiocytes or other liver cells to the effects of microbial molecules, Primary sclerosing cholangitis: a view of therapists and surgeons. The morphological criterion indicating the development of PSC is periductular fibrosis, portal inflammation, disappearance of the bile ducts, the appearance of copper deposits in the lumen of the bile ducts. One of the phenomena of PSC is IBD . there is a predominant lesion of large ducts. Phenotypic variants of PSC associated with ulcerative colitis, as a rule, proceed as pancolitis, are characterized by a smoothed, mildly pronounced course with nonspecific involvement of the ileum, which was also noted in this study. With a combination of PSC - IBD, the outcomes of the disease are more favorable.

The conducted study showed that the course of PSC in PSC - IBD is milder (propulsive motility of choledochus is reduced by 22.5%) as well as the course of IBD - hypermotor dyskinesia of the cecum - an increase of only 14.5%, the left colon - by 52%, sigmoid colon - by 29%, which were accompanied by an increase in stool frequency up to 5-6 times, whereas in severe ulcerative colitis and Crohn's disease - up to 15-22 times.

Conclusions

1. The revealed profile of deviations in the motor activity of the gastrointestinal tract and gastrointestinal tract is characteristic of PSC associated with IBD.
2. Changes in the propulsive activity of the organs of the digestive tract and gastrointestinal tract in combined pathology are milder than in the isolated course of these pathologies and are accompanied by less pronounced stool disorders.
3. With PSC - IBD, less pronounced disorders of the motor function of the small and large intestine were revealed (compared with the isolated course of pathologies), indicating moderate development of SIBR and intestinal dysbiosis, which may determine a milder course of PSC-IBD.

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