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Motor Function of the Gastrointestinal Tract in Gilbert's Syndrome

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Abstract

Introduction: Gilbert's syndrome is a genetically determined decrease in the uptake and conjugation of bilirubin.

Goal: The goal is to identify violations of the motor function of the gastrointestinal tract in the development of Gilbert's syndrome.

Materials and Methods: There were 12 patients with GS under observation: 5 men at the age of 24.7 ± 4.3 years and 7 women at the age of 21.4 ± 3.7 years. The motor function was studied electromyographically (EMG), the amplitude-frequency characteristics of slow waves and spikes, the power of phase and tonic contractions, propulsive activity were determined on the EMG curve using a hardware-software complex with a bandwidth of 0.01-10 kW. Statistical analysis was performed using the Mann-Whitney small sample method, M ± m, at $p < 0.05$.

Results: The increase in propulsive activity is due to the simultaneous increase in the frequency of slow waves and the frequency of spikes. a decrease in the motor function of the duodenum as a result of the possible development of bacterial overgrowth syndrome (SIBO), when excess microbial gas-forming flora contributes to the stretching of the intestinal wall and a decrease in motor function as a result of a decrease in the contacts of the actin-myosin complex. In the jejunum, bacterial overgrowth increases and propulsive activity is halved. The decrease in propulsive activity is due not only to the development of SIBO, but also to a decrease in the reabsorption of conjugated bile acids in Gilbert's syndrome. Complex disorders of the motility of the large intestine were revealed. The motility of the biliary system is multidirectional. The identified disorders of the motility of the digestive system are for the impairment of motor function characteristic of Gilbert's syndrome.

Keywords: *Gilbert's Syndrome; Bilirubin; Gastrointestinal Tract*

Introduction

Gilbert's syndrome is a genetically determined decrease in the uptake and conjugation of bilirubin. It is observed in 1 - 5% of the population, inherited in an autosomal dominant manner [1]. At the heart of Gilbert's syndrome (SG) is a genetic defect - the presence of an additional dinucleotide TA on the promoter region of the gene encoding UDP - glucuronyl transferase.

The disease is detected in adolescence and continues almost all life. The average age of patients is 21 - 25 years. The main symptom of the disease is sclera icterus: icteric coloration of the skin of the face, less often of the feet, palms, nasolabial triangle. Clinical symptoms of the disease are: liver mask, eyelid xanthelasma and yellowness of the skin. Provoking the latter symptom contributes to physical or psychoemotional stress, taking medications or alcohol.

The development of Gilbert's syndrome is accompanied by asthenovegetative syndrome with disorders: fatigue, weakness, dizziness, depression. An enlarged liver is observed in 25% of patients: the liver is soft on palpation, painless and protrudes 1 - 2 cm under the edge of the costal arch.

In most patients, the serum bilirubin level increases at the end of the icteric period or during the period of convalescence. Morphological examination reveals clusters of stellate endothelial cells, moderate sclerosis of some portal tracts, collagenization of reticulin fibers and mild periportal fibrosis. Along with the description of the clinical and morphological picture of the SG, it should be noted that the motor function of the gastrointestinal tract (GIT) and biliary tract (BHT) has not been described.

Goal of the Study

The goal is to identify violations of the motor function of the gastrointestinal tract and gastrointestinal tract in the development of Gilbert's syndrome.

Materials and Methods

There were 12 patients with GS under observation: 5 men at the age of 24.7 ± 4.3 years and 7 women at the age of 21.4 ± 3.7 years. Clinically, 44% of patients showed signs of chronic gastritis C, 55.6% - duodeno - gastric reflux and 44.5% - gastroesophageal reflux, 22.3% - signs of chronic cholecystitis and metabolic syndrome proceeding against a background of constipation, diarrhea was noted: in 33.4% of cases, diffuse changes in the liver and pancreas - in 11.1%. The diagnoses were verified based on the Standard for the provision of medical care (2017). The comparison group consisted of 11 patients with chronic gastritis C. The motor function was studied electromyographically (EMG) with the help of Electromyograph NEUROPACK MEB-9400 (Nihon Kohden), the amplitude-frequency characteristics of slow waves and spikes, the power of phase and tonic contractions, propulsive activity were determined on the EMG curve using a hardware-software complex with a bandwidth of 0.01-10 kW. Statistical analysis was performed using the Mann-Whitney small sample method M ± m at $p < 0.05$.

Results, Discussion and Conclusion

The frequency of slow waves of the stomach was 8.5 ± 0.8/min (5,5 ± 0,1/min, an increase of 54.5%, $p < 0.05$), the amplitude was 0.1 ± 0.03 mV (0,15 ± 0,002 mV, a decrease of 33.3%, $p < 0, 05$), the power of tonic contractions was 0.85 ± 0.0 3 (0.825 ± 0.0 3 increase by 4%, $p < 0.05$). The spike frequency was 2.6 ± 0.05\1.0 ± 0.05 an increase of 160%, $p < 0.001$), the amplitude was 0.03 ± 0.004 mV (0,1 ± 0,01 a mV, decrease of 69.8%, $p < 0.05$), the power of phase contractions was 0.078 ± 0.0031 (0.1 ± 0,002 decrease by 22.7%, $p < 0.05$), propulsive activity - 10.13 ± 0.93 (8.25 ± 0.3 increase by 23.4%, $p < 0.05$). The increase in propulsive activity is due to the simultaneous increase in the frequency of slow waves and the frequency of spikes.

Electromyographically, the frequency of slow waves of the duodenum is 13.3 ± 0.7/min (22.0 ± 2.5 decrease by 39%, $p < 0.05$), amplitude - 0.11 ± 0.003 mV (0.1 ±0.00 mV 2 increase by 10%, $p < 0.05$), power of tonic contractions - 1.463 ± 0.052 (2.2 ± 0.3, decrease by 33.5%, $p < 0.05$). The spike frequency was 2.5 ± 0.04 (1.0 ± 0.02, an increase of 150%, $p < 0.001$), the amplitude was 0.04 ± 0.004 mV (1.0 ± 0.05, a decrease of 59.8%, $p < 0.05$), the power of phase contractions was 0, 1 ± 0.03 (within the reference values), propulsive activity - 14.63 ± 1.3 (22 ± 1,5, decrease by 33.7%, $p < 0.05$). This indicates a decrease in the motor function of the duodenum as a result of the

possible development of bacterial overgrowth syndrome (SIBO), when excess microbial gas-forming flora contributes to the stretching of the intestinal wall and a decrease in motor function as a result of a decrease in contacts of the actin-myosin complex. What could be judged by the clinical picture of the disease.

In the jejunum, the frequency of slow waves is 19.0 ± 0.5/min (20 ± 1.7, decrease by 5%, $p < 0.05$), the amplitude is 0.08 ± 0.003 mV (0.1 ± 0,03, decrease by 20%, $p < 0.05$), power of tonic contractions - 1.52 ± 0.0046 (2. ± 0.2, decrease by 24%, $p < 0.05$). The spike frequency is 2.2 ± 0.03\1.0 ± 0.08, increase by 120%, $p < 0.001$), the amplitude is 0.095 ± 0.003 mV (0.1 ± 0.01, decrease by 5%, $p < 0.05$), the power of phase contractions is 0.21 ± 0, 0033 (increase by 110%, $p < 0.003$), propulsive activity - 7.24 ± 0.90 (20 ± 1.6, decrease by 63.9%, $p < 0.05$). That is, in the jejunum, the processes of bacterial overgrowth increase (in comparison with the duodenum), and the propulsive activity is halved.

The frequency of slow waves of the ileum is 15.1 ± 0.8/min (14 ± 1.1, increase by 5.6%, $p < 0.05$), amplitude - 0.08 ± 0.002 mV (0.1 ± 0.03, decrease by 20%, $p < 0.05$), power of tonic contractions - 1.208 ± 0.004 (1.4 ± 0.2, decrease by 34.5%, $p < 0.05$). The spike frequency is 2.0 ± 0.3 (1.0 ± 0.1, an increase of 99.8%, $p < 0.02$), the amplitude is 0.01 ± 0.002 mV (0.1 ± 0.02, a decrease of 90%, p is 0.04), the power of phase contractions is 0, 2 ± 0.032 (0.1 ± 0.03, increase by 98%, p 0.03), propulsive activity - 6.0 ± 0.15 (14 ± 1.3, decrease by 37.5%, p 0.05). The decrease in propulsive activity is due not only to the development of SIBO, but also to a decrease in the reabsorption of conjugated bile acids in Gilbert's syndrome (through the processes of absorption of conjugated bile acids in Gilbert's syndrome by increased hydrogen evolution, according to the hydrogen breath test).

Electromyographically, the frequency of slow waves of the right colon was 11.0 ± 0.33/min (within the reference values), the amplitude was 0.15 ± 0.005 mV (0.1 ± 0.02, an increase of 50.1%, $p < 0.05$), the power of tonic contractions -1.65 ± 0.035 (1.0 ± 0.01, increase by 50.1%, $p < 0.05$). The spike frequency was 2.8 ± 0.03 (1.0 ± 0.03, an increase of 180%, $p < 0.001$), the amplitude was 0.03 ± 0.004 mV (0,1 ± 0.01, a decrease of 69.8%, $p < 0.05$), the power of phase contractions was 0.084 ± 0.002 (0.1 ± 0.02, decrease by 16%, $p < 0.05$), propulsive activity - 19.6 ± 1.5 (11.0 ± 1.0, increase by 78.1%, $p < 0.05$).

The frequency of slow waves of the left colon was 8.6 ± 0.4/min (6.0 ± 0.4, increase by 43.3%, $p < 0.05$), the amplitude was 0.13 ± 0.015 mV (0.1 ± 0.03, increase by 30%, p < 0, 05), the power of tonic contractions - 1.118 ± 0.013 (0.6 ± 0.03, increase by 18%, $p > 0.1$). The spike frequency is 3.5 ± 0.07 (1.0 ± 0.1, an increase of 250%, $p < 0.001$), the amplitude is 0.03 ± 0.004 mV (0.1 ± 0.02, a decrease of 70.1%, $p < 0.05$), the power of phase contractions is 0.105 ± 0.012 (0.1 ± 0.01, increase by 5%, $p < 0.05$), propulsive activity - 10.65 ± 0.97 (6.0 ± 0.4, increase by 77.5%, $p < 0.05$). Complex disorders of the motility of the colon sections were revealed:

An increase in the propulsive activity of the right sections is mainly due to high-amplitude slow waves and a decrease in the propulsive activity of the left sections - due to the high frequency of adhesions that impede intestinal transit. These disturbances in the rhythm of the colon and determine the differences in stool in the observed patients - diarrhea in 33.4%, constipation - 22.4%, the rest - mixed stool.

The frequency of slow waves of the common bile duct is 8.8 ± 0.6/min (9.0 ± 0.5, decrease by 2.2% $p > 0.1$), the amplitude is 0.12 ± 0.004 mV (0.1 ± 0.03, increase by 20%, $p < 0.05$), power tonic contractions - 1.056 ± 0.0032 (0.9 ± 0.03, increase by 17.3% $p < 0.05$). The frequency of spikes was 3.5 ± 0.06 (1.0 ± 0.02, increase by 249% $p < 0.001$), the amplitude was 0.04 ± 0.005 (0.1 ± 0.02, decrease by 60%, $p < 0.05$). The power of phasic contractions was 0.14 ± 0.012 (0.1 ± 0.04, increase by 40% $p < 0.05$). Propulsive activity was 7.9 ± 0.5 (9.0 ± 0.2, decrease by 12.2% $p < 0.05$).

Electromyographically, the frequency of slow waves of the gallbladder is 8.0 ± 0.7/min (within the reference values), the amplitude is 0.11 ± 0.007 mV (0.1 ± 0.02, an increase of 10%, $p < 0.05$), the power of tonic contractions is 0, 88 ± 0.05 (0.8 ± 0.04, increase by 10% $p < 0.05$). The spike frequency was 2.9 ± 0.3\ 1.0 ± 0.03, increase by 190% $p < 0.01$), the amplitude was 0.03 ± 0.005 mV (0.1 ± 0.02, decrease

by 70% $p < 0.05$). The power of phasic contractions was 0.87 ± 0.025 (0,1 ± 0,03, increase by 77% $p < 0.05$). Propulsive activity was 10.12 ± 1.1 (8.0 ± 0.4, increase by 26.5% $p < 0.05$).

Thus, the propulsive activity of the stomach is increased by increasing the frequency of slow-wave and spike activity, that is, the function of the pacemaker of the stomach improves [2] and the excitability of myocytes increases. The motility of the biliary system is of a multidirectional nature - hypomotor dyskinesia of the common bile duct and hypermotor dyskinesia of the gallbladder, as a result of which a number of patients experience pain, described in the literature, possibly due to the function of the pacemaker of the biliary tract [3].

The study carried out that the severity of hypermotor dyskinesia mainly in the small intestine is due to a decrease in the sequence of conversion of cholesterol into bile acids with subsequent conjugation of bile acids and their participation in the regulation of intestinal motility.

Colon pacemakers restore reduced small bowel motility. In general, the motility of the gastrointestinal tract has a wavy character, increases in the proximal and decreases in the distal digestive tract, which is characteristic of Gilbert's syndrome.

The identified disorders of the motility of the digestive system are algorithms for the impairment of motor function characteristic of Gilbert's syndrome.

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