



Research Article

Motor Function of the Gastrointestinal Tract and Biliary Tract in Karoli's Disease

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Summary

Focal intrahepatic biliary dilation (Karoli's disease) is characterized by non-obstructive sac-like dilatation of the intrahepatic bile ducts.

The aim: To identify gastrointestinal and ZHVP disorders in Karoli's disease.

Materials and Methods: There were 16 patients under observation with signs of dilation of the bile ducts, of which 75% were women aged 47.9 ± 6.5 years, 25% were men aged 56.0 ± 7.1 years. Additionally, 75% of cases had chronic (25% - biliary) pancreatitis, gastroesophageal reflux disease, liver fibrosis - in half of the patients, the remaining syndromes - diverticulosis, liver cysts, sphincter dysfunction Oddi - in every eighth patient. The presence of Karoli's disease was verified by ultrasound examination during visualization of dilatation of the bile ducts.

Results: Karoli's disease revealed a decrease in the propulsive activity of the common bile duct and possibly a compensatory increase in the motility of the gallbladder. The decrease in the motility of the biliary tract is caused by sac-like extensions of their walls. That is, in Karoli's disease, hypermotor dyskinesia of the colon progressing distally is observed.

Conclusion: The presence of a turbulent flow of bile through choledochus in Karoli's disease causes the development of intestinal dysbiosis. Pronounced changes in the motor function of the colon are due to the development of dysfunction of the Oddi sphincter and liver fibrosis observed in the studied patients.

Focal intrahepatic biliary dilation (Karoli's disease) is characterized by non-obstructive sac-like dilatation of the intrahepatic bile ducts [1-3]. These expanded passages are more sensitive to infection, stones are more often found in them. Histological changes similar to congenital fibrosis appear in the liver, but there is no reason to believe that this condition is genetically determined. In particular, kidney lesions associated with fibrosis are not observed in Karoli's disease. The clinical picture shows abdominal pain, fever, sometimes infectious complications - septicemia, jaundice appear as a syndrome accompanying the development of cholangitis. There is a syndrome of impaired absorption. The diagnosis of Karoli's disease is made on the basis of ultrasound data, the results of percutaneous transhepatic cholangiography, however, the motor function of the gastrointestinal tract (gastrointestinal tract) and biliary tract (biliary tract) has not been sufficiently investigated.

The aim is to identify gastrointestinal and ZHVP disorders in Karoli's disease.

Materials and Methods

There were 16 patients under observation with signs of dilation of the bile ducts, of which 75% were women aged 47.9 ± 6.5 years, 25% were men aged 56.0 ± 7.1 years. Additionally, 75% of cases had chronic (25% - biliary) pancreatitis, gastroesophageal reflux disease, liver fibrosis - in half of the patients, the remaining syndromes - diverticulosis, liver cysts, sphincter dysfunction Oddi - in every eighth patient. The presence of Karoli's disease was verified by ultrasound examination during visualization of dilatation of the bile ducts. The motor function of the gastrointestinal tract and the gastrointestinal tract was examined electromyographically by installing bipolar silver electrodes on the anterior abdominal wall in the projection area of the organ under study. Exclusion

criteria: pregnancy; presence of oncological pathology and pathology of the mental sphere; senile age. The analysis of the amplitude-frequency characteristics of slow waves and spikes, the power of phase and tonic contractions, and the propulsive activity was carried out using a hardware and software complex. Statistical analysis was carried out using the Mann–Whitney small sample method at $p < 0.05$.

Research Results

The frequency of slow choledoch waves was 7.0 ± 0.5 min (decrease by 20.2%, $p < 0.05$), the amplitude was 0.12 ± 0.003 mV (increase by 20%, $p < 0.05$), the power of tonic contractions was 0.84 ± 0.006 (decrease by 6.7%, $p < 0.05$). The frequency of spikes was 3.3 ± 0.4 (an increase of 230%, $p < 0.0001$), the amplitude was 0.05 ± 0.002 mV (a decrease of 50.1%, $p < 0.05$), the power

of phase contractions was 0.165 ± 0.013 (an increase of 65%, $p < 0.05$), the propulsive activity was 5.1 ± 0.3 (a decrease of 43.3%, $p < 0.05$) (Figure 1). Electromyographically, the frequency of slow gallbladder waves was 8.7 ± 0.6 \min (an increase of 9%, $p < 0.05$), the amplitude was 0.08 ± 0.002 mV (a decrease of 20%, $p < 0.05$), the power of tonic contractions was 0.696 ± 0.012 (a decrease of 13%, $p < 0.05$). The frequency of spikes was 2.2 ± 0.4 (an increase of 120%, $p < 0.001$), the amplitude was 0.03 ± 0.003 mV (a decrease of 70.1%, $p < 0.05$), the power of phase contractions was 0.066 ± 0.0031 (a decrease of 34%, $p < 0.05$), the propulsive activity was 10.54 ± 1.13 (an increase of 31.8%, $p < 0.05$). That is, Karoli's disease revealed a decrease in the propulsive activity of the common bile duct and possibly a compensatory increase in the motility of the gallbladder. The decrease in the motility of the biliary tract is caused by sac-like extensions of their walls.

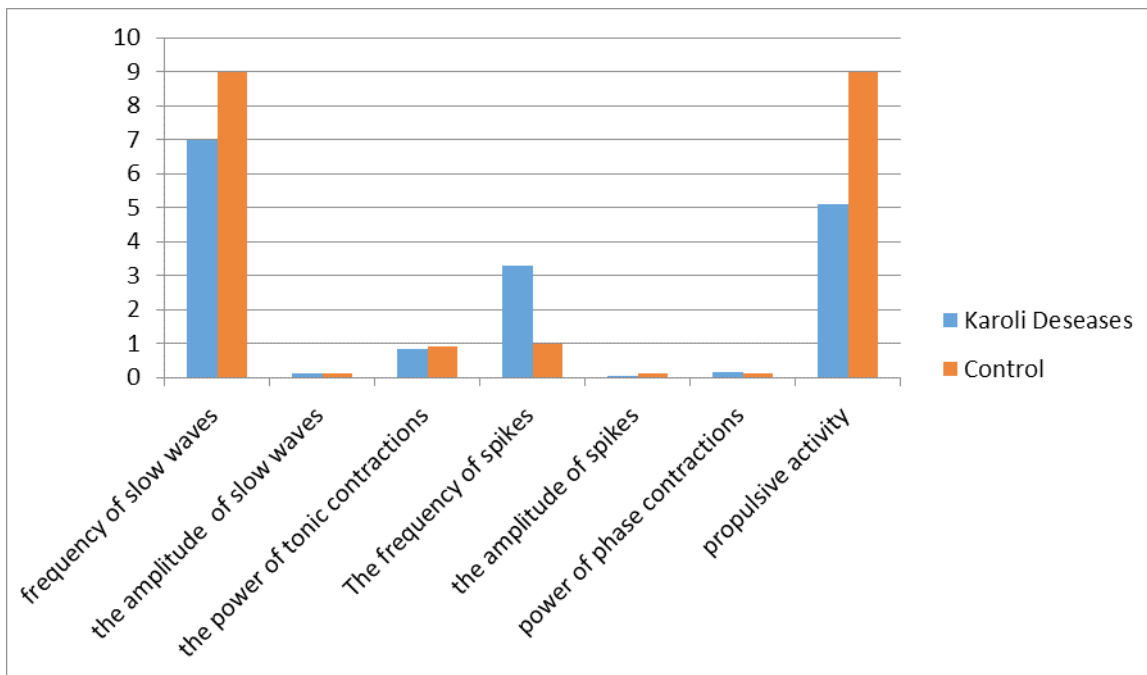


Figure 1: Indicators of the electromyogram of the common bile duct in Karoli's disease and in control.

The frequency of slow stomach waves was 8.2 ± 0.7 \min (an increase of 49%, $p < 0.05$), the amplitude was 0.12 ± 0.004 mV (a decrease of 20%, $p < 0.05$), the power of tonic contractions was 0.974 ± 0.052 (an increase of 19.3%, $p < 0.05$). The frequency of spikes was 3.7 ± 0.3 (an increase of 270%, $p < 0.001$), the amplitude was 0.04 ± 0.002 mV (a decrease of 59.9%, $p < 0.05$), the power of phase contractions was 0.148 ± 0.012 (an increase of 48%, $p < 0.05$), the propulsive activity was 6.7 ± 0.3 (a decrease of 18.8%, $p < 0.05$). That is, in Karoli's disease, the propulsive activity of the stomach is reduced mainly due to hypertonicity of the circular and to a lesser extent due to oblique muscles. Electromyographically, the frequency of slow duodenal waves was 21.7 ± 2.0 \min (decrease by 1.4%, $p > 0.1$), the amplitude was 0.13 ± 0.003 mV (increase by 30%, $p < 0.05$), the power of tonic contractions was $2,821 \pm 0.172$ (increase by 28.2%, $p < 0.05$). The frequency of spikes was 2.7 ± 0.3 (an increase of 170% $p < 0.001$), the amplitude was 0.06 ± 0.002 mV (a decrease of 40.1%, $p < 0.05$), the power of phase contractions was 0.162 ± 0.002 (an increase of 62%, $p < 0.05$), the propulsive activity was 17.4 ± 0.9 (a decrease of 20.9%, $p < 0.05$). That is, in Karoli's disease, the propulsive activity of the duodenum is reduced due to a decrease in the passage of bile through the biliary tract and possible development of SIBR. Electromyographically, the frequency of slow waves in

the jejunum was 21.0 ± 2.4 /min (an increase of 5%, $p < 0.05$), the amplitude was 0.09 ± 0.003 mV (a decrease of 10%, $p < 0.05$), the power of tonic contractions was 1.89 ± 0.13 (a decrease of 5.5%, $p < 0.05$). The frequency of spikes was 1.5 ± 0.2 (an increase of 50.1%, $p < 0.05$), the amplitude was 0.02 ± 0.0015 mV (a decrease of 79.9%, $p < 0.05$), the power of phase contractions was 0.03 ± 0.002 (a decrease of 69.8%, $p < 0.05$), the propulsive activity was 63.0 ± 4.9 (an increase of 215%, $p < 0.001$). That is, the propulsive activity of the jejunum is increased due to the development of dysbiosis of the middle parts of the small intestine.

In the right parts of the colon, the frequency of slow waves was 13.0 ± 0.8 /min (an increase of 18.1%, $p < 0.05$), the amplitude was 0.1 ± 0.02 mv (within the reference values), the power of tonic contractions was 1.3 ± 0.12 (an increase of 18.1%, $p < 0.05$). The frequency of spikes was 2.6 ± 0.3 (an increase of 160%, $p < 0.001$), the amplitude was 0.04 ± 0.002 mV (a decrease of 60.1%, $p < 0.05$), the power of phase contractions was 0.104 ± 0.013 (an increase of 4%, $p < 0.05$), the propulsive activity was 12.5 ± 1.4 (an increase of 13.6%, $p < 0.05$). That is, hypermotor dyskinesia of the right colon was detected in Karoli's disease. In Karolyi's disease in the left colon, the frequency of slow waves was 11.4 ± 0.4 /min (an increase of 90%, $p < 0.05$), the amplitude was 0.13 ± 0.003 mV (an increase of 30%, $p < 0.05$), the power of tonic contractions was 1.482 ± 0.12 (an increase of 147%, $p < 0.0001$). The frequency of spikes was 3.6 ± 0.4 (an increase of 260%, $p < 0.001$), the amplitude was 0.04 ± 0.002 mV (a decrease of 60%, $p < 0.05$), the power of phase contractions was 0.144 ± 0.011 (an increase of 44%, $p < 0.05$), the propulsive activity was 10.2 ± 0.8 (an increase of 70.1%, $p < 0.05$).

In the sigmoid colon, the frequency of slow waves was 10.0 ± 0.5 /min (an increase of 99.8%, $p < 0.03$), the amplitude was 0.13 ± 0.002 mV (an increase of 30%, $p < 0.05$), the power of tonic

contractions was 1.3 ± 0.11 (an increase of 160%, $p < 0.001$). The frequency of spikes was 2.0 ± 0.3 (increase by 100.3%, $p < 0.002$), amplitude - 0.03 ± 0.002 (decrease by 69.9%, $p < 0.05$), power of phase contractions - 0.06 ± 0.004 (decrease by 40%, $p < 0.05$), propulsive activity - 21.7 ± 1.5 (increase by 334%, $p < 0.001$). That is, in Karoli's disease, hypermotor dyskinesia of the colon progressing distally is observed.

Discussion and Conclusions

The development of Karoli's disease is accompanied by hypomotor dyskinesia of the stomach, choledochus and duodenum, antro-duodenal coordination is 1:2.6 (normally 1:4), which indicates an acceleration of the evacuation of gastric contents into the small intestine. The presence of a turbulent flow of bile through choledochus in Karoli's disease causes the development of intestinal dysbiosis. Pronounced changes in the motor function of the colon are due to the development of dysfunction of the Oddi sphincter and liver fibrosis observed in the studied patients.

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