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Research Article

Autoimmune Hepatitis: The Role of Disorders of the Motor Activity of the Gastrointestinal Tract and Biliary Tract

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Abstract

Introduction: Autoimmune hepatitis (AIG) is a chronic liver disease characterized by immune and autoimmune features with the presence of significant titers of circulating tissue autoantibodies, hypergammaglobulinemia. Periportal inflammation with the development of stepped necrosis.

Aim of the Study: The aim is to show violations of the motor function of the gastrointestinal tract (gastrointestinal tract) and biliary tract (GI) in AIG.

Materials and Methods: 14 patients with autoimmune hepatitis were under observation, among them women aged 56.2 ± 7.9 years, men aged 49.3 ± 12.5 years with a ratio of f:m as 5:2 Clinically, patients with AIG revealed subfebrility, tachycardia, decreased appetite, vascular asterisks, arthralgia. The motor function of the gastrointestinal tract and the gastrointestinal tract was recorded electromyographically using an electromyograph "Nihon Kohden" (Japan) with a bandwidth of 0.1 - 10 mV. Statistical analysis was carried out using the Statistics - 15 software package.

Results: Electromyographically, the propulsive activity was 12.1 ± 0.8 (a decrease of 45%, $p < 0.05$). That is, hypomotor dyskinesia of the duodenum was noted in AIG due to the possible development of excessive bacterial growth syndrome (SIBR). The propulsive activity of the gallbladder was 7.56 ± 0.81 (a decrease of 5.5%, $p < 0.05$). That is, hypomotor dyskinesia of the biliary system was observed in AIG.

Conclusion: Analysis of the propulsive activity of the gastrointestinal tract and the gastrointestinal tract in patients suffering from AIG showed that its values are reduced in the biliary system and small intestine, which may be associated with a decrease in the flow of bile into the duodenum and the possible development of bacterial overgrowth syndrome (SIBR). The tendency to develop hypermotor dyskinesia of the stomach contributes - due to the gastrocolytic reflex - to the development of hypermotor dyskinesia of the left colon and sigmoid colon.

Keywords: *Autoimmune Hepatitis; Electromyography*

Introduction

Autoimmune hepatitis is a chronic liver disease characterized by immune and autoimmune features with the presence of significant titers of circulating tissue autoantibodies, hypergammaglobulinemia. Periportal inflammation with the development of stepped necrosis [1]. The target antigens responsible for the formation of autoimmune hepatitis (AIG) include a liver-specific protein, an antigen of hepatic-renal macrosomes and an asialoglycoprotein receptor. There is a close relationship with the NLA antigens involved in immunoregulatory processes, there is a high frequency of NLA alleles A1, B8, DR3, DR4. NLP3 positive patients are characterized by a severe course of the disease. Autoimmune hepatitis c NLA-DR4 is mainly prevalent in the countries of Southeast Asia, in Japan, the course of the disease is characterized by frequent systemic manifestations and a good response to immunosuppressive therapy [2]. In 85% of cases, there is a type 1 form of AIG associated with seropositive reactions to antibodies to smooth muscles in titers of 1:40 or more and/or homogenous antinuclear antibodies. In addition, anti-neutrophil cytoplasmic antibodies of the p-type (p-ANCA) are detected, which are characteristic, among other things, for primary sclerosing cholangitis. Patients with autoimmune hepatitis type 1 have antibodies to the soluble hepatic antigen SLA, which react with cytokeratins 8 and 18 in the cytoplasm of hepatocytes. Further, it was shown that the markers of type 1 AIG are ANA (antinuclear antibodies), SMA (antibodies to actin) and antiSLA. Autoimmune hepatitis type 2 with antibodies to hepatic-renal microsomes (antiLCM1). It is often combined with other autoimmune diseases - autoimmune thyroiditis, insulin-dependent diabetes mellitus, vitiligo. Autoimmune hepatitis type 2 has a specific autoantigen - hepatic macrosomal enzyme - cytochrome p450-2-D6 - a protein expressed on the surface of hepatocyte membranes and is recognized by anti-LCM1. Concomitant immunological disorders in patients with type 2 AIG are more common than in type 1 AIG. Non-organ-specific antibodies, including antithyroid, antimicrosomal, anti-islet (to pancreatic islets) are detected in 1/3 patients, there is a faster progression to cirrhosis. Considering that the genetic predisposition to AIG is not realized in all patients, the role of additional factors triggering the autoimmune process is being considered. The debut of AIG after infection with viral hepatitis A, B, C, herpes (HCV-6 and HCV-1), Epstein-Barr virus is described. The trigger factors of autoimmune reactions can be reactive metabolites of drugs metabolized by enzymes of the family of SUR and UDP-glucuronyltransferases. The main role in the pathogenesis of AIG belongs to T-lymphocytes. Activation of CD4 T helper cells is the main factor in the development of autoimmune pathology, leading to the appearance of banned clones of lymphocytes sensitized to liver autoantigens and causing damage to it. The revealed imbalance of CD4/CD8 lymphocytes in favor of the CD4 fraction, an increase in their number in the portal tracts, suppressive cytotoxic lymphocytes predominate in the areas of stepped necrosis. However, along with the autoimmune process, the motor-secretory function of the biliary tract (GI) and gastrointestinal tract (gastrointestinal tract) should play a certain role in the pathogenesis of AIG, however, this area of research is insufficiently described in the literature.

Aim of the Study

The aim is to show violations of the motor function of the gastrointestinal tract and the gastrointestinal tract in AIG.

Materials and Methods

14 patients with autoimmune hepatitis were under observation, among them women aged 56.2 ± 7.9 years, men aged 49.3 ± 12.5 years with a ratio of f:m as 5:2. The diagnosis was made on the basis of immunological examination and morphological examination of the biopsy material. Concomitant nosologies are autoimmune thyroiditis, cirrhosis of the liver, detected in 14.3% of patients, chronic cholecystitis - in 57.1%, cholelithiasis - in 42.9%, biliary sludge - in 14.3%, chronic pancreatitis - in 28.5% of cases, hepatosplenomegaly - in 14.3 and 28.6%, respectively, Sjogren's syndrome - in 14.3%, constipation or diarrhea - in 28.6% of cases, itching - in 14.3%, joint syndrome in 28.6%. The comparison group consisted of 10 patients with chronic gastritis. Clinically, patients with AIG have subfebrility, tachycardia, decreased appetite, vascular asterisks, arthralgia. The motor function of the gastrointestinal tract and biliary tract was recorded electromyographically by installing bipolar silver electrodes on the anterior abdominal wall in the projection area of the registered organ. The

analysis of the amplitude-frequency parameters of slow waves and spikes, the power of phase and tonic contractions, and the propulsive activity was carried out using an electromyograph "Nihon Kohden" (Japan) with a bandwidth of 0.1-10 mV. Statistical analysis was carried out using the Statistics - 15 software package.

Results and Discussion

The frequency of slow stomach waves was 9.8 ± 0.7/min (an increase of 78.1%, $p < 0.05$), the amplitude was 0.12 ± 0.002 mV (a decrease of 20%, $p < 0.005$), the power of tonic contractions was 1.176 ± 0.125 (an increase of 42.5%, $p < 0.05$). The frequency of spikes was 4.3 ± 0.5 (an increase of 330%, $p < 0.001$), the amplitude was 0.03 ± 0.004 mV (a decrease of 69.8%, $p < 0.05$), the power of phase contractions was 0.129 ± 0.014 (an increase of 29%, $p < 0.05$), the propulsive activity was 8.3 ± 0.65 (an increase of 0.6%, $p > 0.1$). That is, with AIG, the motility of the stomach is within the reference values.

Electromyographically, the frequency of slow waves of the duodenum was 18.1 ± 2.0/min (a decrease of 17%, $p < 0.05$), the amplitude was 0.11 - 0.003 mV (an increase of 10%, $p < 0.05$), the power of tonic contractions was 1.991 ± 0.13 (an increase of 95%, $p < 0.05$). The frequency of spikes was 3.7 ± 0.4 (an increase of 270%, $p < 0.001$), the amplitude was 0.05 ± 0.002 mV (a decrease of 49.9%, $p < 0.05$), the power of phase contractions was 0.165 ± 0.014 (an increase of 65%, $p < 0.05$), the propulsive activity was 12.1 ± 0.8 (a decrease of 45%, $p < 0.05$) (Figure 1). That is, hypomotor dyskinesia of the duodenum was noted in AIG due to the possible development of excessive bacterial growth syndrome (SIBR).

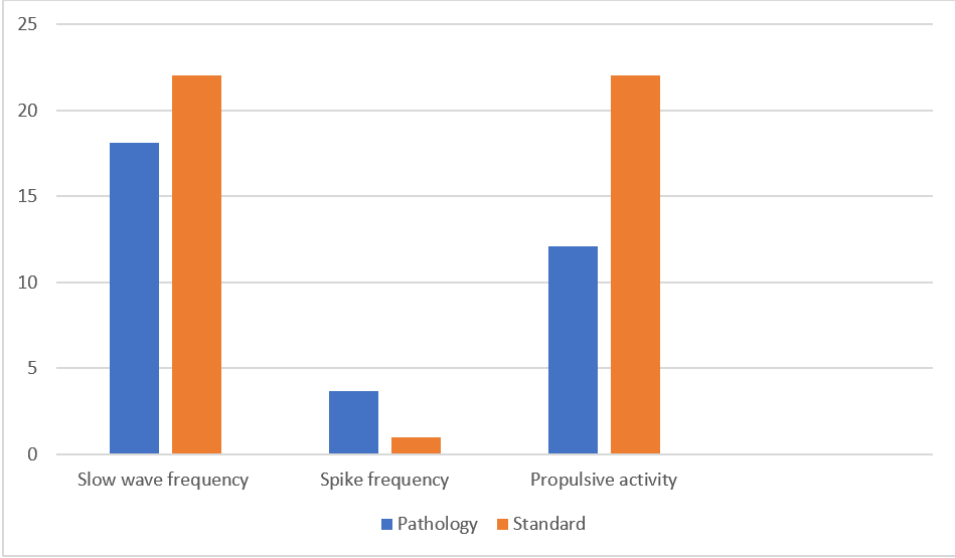

Figure 1: *The frequency of slow waves and spikes and the propulsive activity of the duodenum.*

In the jejunum, the frequency of slow waves was 18.1 ± 0.1/min (decrease by 10.5%, $p < 0.05$), the amplitude was 0.1 ± 0.004 mV (within the reference values), the power of tonic contractions was 1.81 ± 0.21 (decrease by 10.5%, $p < 0.05$). The frequency of spikes was 4.6 ± 0.3 (360% increase, $p < 0.001$), amplitude - 0.04 ± 0.002 mV (60.1% decrease, $p < 0.05$), power of phase contractions - 0.184 ± 0.013

(84% increase, $p < 0.05$), propulsive activity - 9.8 ± 0.7 (decrease by 51%, $p < 0.05$). That is, hypomotor jejunal dyskinesia was observed in AIG, apparently due to the development of SIBR.

In the right parts of the colon, the frequency of slow waves was 11.7 ± 0.6/min (an increase of 6.4%, $p < 0.05$), the amplitude was 0.12 ± 0.005 mV (an increase of 20%, $p < 0.05$), the power of tonic contractions was 1.404 ± 0.12 (an increase of 27.6%, $p < 0.05$). The frequency of spikes was 3.8 ± 0.4 (increase by 280%, $p < 0.001$), amplitude - 0.04 ± 0.003 (decrease by 59.9%, $p < 0.05$), power of phase contractions - 0.152 ± 0.013 (increase by 52%, $p < 0.05$), propulsive activity - 9.23 ± 0.72 (decrease by 26.1%, $p < 0.05$). That is, hypomotor dyskinesia of the right colon was observed in AIG.

In the left colon, the frequency of slow waves was 9.0 ± 0.7/min (an increase of 50%, $p < 0.05$), the amplitude was 0.11 ± 0.003 mV (an increase of 10%, $p < 0.05$), the power of tonic contractions was 0.99 ± 0.06 (an increase of 65%, $p < 0.05$). The frequency of spikes is 4.1 ± 0.5 (increase by 310%, $p < 0.001$), the amplitude is 0.02 ± 0.003 mV (decrease by 80%, $p < 0.05$), the power of phase contractions is 0.082 ± 0.003 (decrease by 18%, $p < 0.05$), the propulsive activity is 12.1 ± 0.3 (increase by 101.3%, $p < 0.001$). That is, hypermotor dyskinesia of the left colon was observed in AIG.

In the sigmoid colon, the frequency of slow waves was 8.7 ± 0.9/min (an increase of 74%, $p < 0.05$), the amplitude was 0.12 ± 0.004 mV (an increase of 20%, $p < 0.05$), the power of tonic contractions was 1.044 ± 0.13 (an increase of 108.8%, $p < 0.003$). The frequency of spikes was 3.9 ± 0.4 (an increase of 290.1%, $p < 0.001$), the amplitude was 0.03 ± 0.002 mV (a decrease of 69.9%, $p < 0.05$), the power of phase contractions was 0.117 ± 0.016 (a decrease of 17%, $p < 0.05$), the propulsive activity was 8.9 ± 0.7 (an increase in by 78%, $p < 0.05$). That is, in the course of the colon, phase changes in the motility of the colon were noted: hypermotor dyskinesia in the right flank, hypermotor dyskinesia in the left flank and sigmoid colon.

The biliary system, the frequency of slow choledoch waves was 9.7 ± 0.8/min (an increase of 7.8, $p < 0.05$), the amplitude was 0.12 ± 0.003 (an increase of 20%, $p < 0.05$), the power of tonic contractions was 1,164 ± 0.152 (an increase of 29.3%, $p < 0.05$). The frequency of spikes was 4.3 ± 0.4 (an increase of 330%, $p < 0.001$), the amplitude was 0.04 ± 0.002 (a decrease of 59.9%, $p < 0.05$), the power of phase contractions was 0.172 ± 0.012 (an increase of 72%, $p < 0.05$), the propulsive activity was 6.8 ± 0.3 (a decrease of 24.4%. $p < 0.05$).

The frequency of slow gallbladder waves was 7.4 ± 0.3/min (decrease by 7.5%, $p < 0.05$), the amplitude was 0.11 ± 0.004 mV (increase by 10%, $p < 0.05$), the power of tonic contractions was 0.884 ± 0.007 (increase by 15%, $p < 0.05$). The frequency of spikes is 3.9 ± 0.22 (increase by 290%, $p < 0.001$), the amplitude is 0.03 ± 0.001 mV (decrease by 69.9%, $p < 0.05$), the power of phase contractions is 0.117 ± 0.009 (increase by 17%, $p < 0.05$), the propulsive activity is 7.56 ± 0.81 (decrease by 5.5%, $p < 0.05$). That is, hypomotor dyskinesia of the biliary system was observed in AIG.

Conclusion

Analysis of the propulsive activity of the gastrointestinal tract and the gastrointestinal tract in patients suffering from AIG showed that its values are reduced in the biliary system and small intestine, which may be associated with a decrease in the flow of bile into the duodenum and the possible development of the syndrome of excessive bacterial growth. The tendency to develop hypermotor dyskinesia of the stomach contributes - due to the gastrocolytic reflex - to the development of hypermotor dyskinesia of the left colon and sigmoid colon. Thus, the development of AIG is accompanied by hypomotor dyskinesia of the biliary system, small intestine and right parts of the colon and due to a decrease in bile secretion. The revealed profile of disorders of the motor function of the gastrointestinal tract and the gastrointestinal tract is characteristic only of autoimmune liver damage.

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