ECRONICON OPEN ACCESS

EC GASTROENTEROLOGY AND DIGESTIVE SYSTEM

Research Article

Chronic Pancreatitis in the Practice of a Gastroenterologist and Dentist

Dyakonov Yu M[1], Lychkova AE[2]*, Terentyev AA[3] and Puzikov AM[4]

[1]Moscow Regional Research and Clinical Institute ("MONIKI"), Russia

[2]MOSCOW Clinical Scientific and Practical Center Named After A. S. Loginov of the Moscow Department of Health, Russia

[3]FSAOU HE "Russian National Research Medical University Named After N.I. Pirogov" Ministry of Health of the Russian Federation, Russia

[4]AMG-ESTETIC, Russia

***Corresponding Author:** Lychkova AE, MOSCOW Clinical Scientific and Practical Center Named After A. S. Loginov of the Moscow Department of Health, Russia.

Received: April 23, 2024; **Published:** May 03, 2024

Abstract

Introduction: The incidence of CP in developed countries ranges from 5 - 10 cases per 100,000 population; in the world as a whole - 1.6-23 cases per 100,000 population per year (19-22). There is a tendency in the world to increase the incidence of acute and chronic pancreatitis, over the past 30 years - by more than 2 times.

Aim of the Study: The aim is to identify the possibility of changing the motor function of the gastrointestinal tract and lesions of the oral cavity in chronic pancreatitis.

Materials and Methods: The motor function of the gastrointestinal tract was recorded electromyographically. The electromyogram curve was used to evaluate the amplitude-frequency characteristics of slow waves and spikes, the power of phase and tonic contractions, and propulsive activity. The study was conducted in a group of patients with chronic pancreatitis (48 patients), the comparison group consisted of 39 patients with gastritis C. Statistical analysis was carried out using the Statistika -16 software package at $p < 0.05$.

Results: In CP, pronounced hypermotor dyskinesia of all muscle groups of the stomach was observed with a predominance of spasm of the circular and partially oblique muscle layers, which determines a decrease in the propulsive activity of the stomach.

Moderate hypermotor dyskinesia of the small intestine in CP was revealed due to a marked decrease in the tone of the circular muscles, which may be due to secretory insufficiency in pancreatic pathology.

Hypermotor dyskinesia of the descending colon and colon as a whole was determined, which may be associated with the development of intestinal dysbiosis due to secretory insufficiency of the pancreas in CP.

Patients with pancreatic pathology often have various immune disorders. The close connection of the pancreas and salivary glands is demonstrated by the presence of antibodies to the tissue antigen of the salivary glands in 1/3 of patients with chronic pancreatitis with the presence of antibodies to the tissue antigen of the pancreas.

Conclusion: Electromyography can be used to study the motor function of the gastrointestinal tract in chronic pancreatitis.

The pathology of the pancreas in CP determines the nature of the violation of the motor function of the gastrointestinal tract - hypomotor dyskinesia of the stomach and hypermotor dyskinesia of the small and large intestine due to secretory insufficiency of the pancreas and developing intestinal dysbiosis.

Keywords: *Motor Activity of the Gastrointestinal Tract; Secretory Insufficiency of the Pancreas; Intestinal Dysbiosis*

Introduction

In the system of relationships between a general practitioner and other specialists, the aspects of their interaction with a dentist are the least studied. This is due to the mutual underestimation of changes on the part of organs and tissues of the oral cavity in various diseases of internal organs, including the digestive system, and under the influence of drug therapy of these diseases [1,2]. The presence of certain changes on the part of organs and tissues of the oral cavity and appropriate dental advice can help a general practitioner in diagnosis and adequate treatment of the patient [1,2].

The prevalence of chronic pancreatitis (CP) in Europe is 25.0 - 26.4 cases per 100,000 population, in Russia - 27.4 - 50 cases per 100,000 population [3]. The incidence of CP in developed countries ranges from 5 - 10 cases per 100,000 population; in the world as a whole - 1.6 - 23 cases per 100,000 population per year [19-22]. There is a tendency in the world to increase the incidence of acute and chronic pancreatitis, over the past 30 years - by more than 2 times [3]. CP usually develops in adulthood (35 - 50 years). In developed countries, the average age since diagnosis has decreased from 50 to 39 years, men suffer from CP 2 times more often than women, there is a tendency to increase the proportion of women among the sick (by 30%); primary disability of patients reaches 15% [4,21].

Mortality after the initial diagnosis of CP is up to 20% during the first 10 years, and more than 50% after 20 years, averaging 11.9%. 15 - 20% of patients with CP die from complications arising during exacerbations of pancreatitis, others due to secondary digestive disorders and infectious complications [19,20].

Particular importance is currently attached to hypertriglyceridemia, others to hyperlipidemia in the development of CP.

To diagnose hypertriglyceridemic pancreatitis, an increase in serum triglycerides of more than 1000 mg/dl (11.2 mmol/l) is necessary. However, a recurrence of pancreatitis can occur already with a lower rise in blood triglyceride levels - more than 500 mg/dl (5.6 mmol/l) [4,5].

Mutations of the cationic trypsinogen (PRSS1) gene lead to the synthesis of pathologically active trypsin and the development of hereditary pancreatitis [17]. In patients with hereditary pancreatitis, symptoms usually occur much earlier (before the age of 20), the disease progresses rapidly, and against this background, the risk of developing pancreatic adenocarcinoma increases. Mutations of the cystic fibrosis transmembrane conduction regulator (CFTR) gene [7] and trypsin inhibitor (PSTI or SPINK1) were detected in patients with idiopathic CP [19]. In the mixed Russian population, the N34S mutation in the SPINK1 gene is significantly more common than in the control (14.6 and 2.9%, respectively; $p < 0.05$). The odds ratio of developing idiopathic CP in the presence of the N34S mutation in this study was 4.6 [9]. The N34S mutation determines 10% or more cases of idiopathic CP [10].

Duct obstruction: Conditions associated with the development of obstructive CP are trauma, the presence of concretions, false cysts and tumors. It has not been definitively proven whether CP occurs against the background of pancreas divisum and dysfunction of the Oddi sphincter.

Immunological factors: Autoimmune pancreatitis (AIP) can occur in isolation or in combination with Sjogren's syndrome, inflammatory bowel diseases and many other autoimmune diseases [22]. In recent years, two types of AIP have been distinguished - I and II [12-14].

The proven causes of CP include chronic renal failure and hyperparathyroidism, ischemic lesion in atherosclerosis of mesenteric vessels, while the exact role of drugs and toxic substances remains unproven [3,15-17].

Cystic inflammatory transformation (CVT) of the duodenum is an inflammatory and cystic lesion of its wall with localization in the descending part of the intestine, which occurs in patients with CP. There are three possible mechanisms of development of CVT:

- Inflammation of the dorsal part of the head of the pancreas embedded in the wall of the duodenum, due to a violation of the outflow of secretions through the Santorini duct;
- Secondary spread of the inflammatory process from the head of the pancreas to the paraduodenal fiber and the wall of the duodenum;
- Rarely - primary inflammatory cystic lesion of a truly heterotopic pancreatic tissue with secondary obstructive pancreatitis or with the parallel development of CP with alcohol abuse [18].

The organs of the oral cavity are also affected in chronic pancreatitis. However, this issue is not sufficiently covered in the literature.

Aim of the Study

The aim is to identify the possibility of changing the motor function of the gastrointestinal tract and lesions of the oral cavity in chronic pancreatitis.

Materials and Methods

The diagnosis of chronic pancreatitis is established on the basis of:

- Anamnestic data (characteristic complaints of abdominal pain attacks and/or clinical signs of exocrine and/or endocrine pancreatic insufficiency, detection of chronic pancreatitis before).
- Physical examination (abdominal wall muscle soreness during palpation in the pancreatic projection area)
- Laboratory examination (signs of exocrine and endocrine pancreatic insufficiency according to functional tests).
- Instrumental examination (calcifications in the parenchyma and pancreatic ducts, dilation of the main pancreatic duct and its branches according to CT, MRCPG, ESP).

In pancreatitis, the clinical picture may contain Sjogren's syndrome (Sjogren), or "dry syndrome", which is characterized by autoimmune damage to the salivary glands that determine the development of pathology of the oral cavity. To recognize Sjogren's syndrome, a triad of signs is used: dry keratoconjunctivitis; xerostomia and/or parenchymal mumps - diseases in the pathogenesis of which autoimmune reactions are involved. The presence of the first two signs makes it possible to diagnose the "dry" syndrome, Sjogren's disease, the presence of all three signs - Sjogren's syndrome (secondary Sjogren's syndrome). Sjogren's disease (primary Sjogren's syndrome) is a chronic inflammatory disease of the exocrine glands, primarily salivary and lacrimal glands, with the gradual development of their secretory insufficiency in combination with various systemic manifestations [10,11], which significantly alter the processes of primary processing of food contents in the oral cavity.

Various immune disorders are often detected in patients with pancreatic pathology [12,13]. The close connection of the pancreas and salivary glands is demonstrated by the presence of antibodies to the tissue antigen of the salivary glands in 1/3 of patients with chronic pancreatitis with the presence of antibodies to the tissue antigen of the pancreas.

The motor function of the gastrointestinal tract was recorded electromyographically by placing cutaneous silver electrodes on the anterior abdominal wall in the projection area of the registered organ. The electromyogram curve was used to evaluate the amplitude-frequency characteristics of slow waves and spikes, the power of phase and tonic contractions, and propulsive activity. The study was

conducted in a group of patients with chronic pancreatitis (48 patients), the comparison group consisted of 39 patients with gastritis C. Statistical analysis was carried out using the Statistika -16 software package at $p < 0.05$.

Results and their Discussion

In patients suffering from pancreatitis, the frequency of slow waves of gastric EMA was 8.0 ± 1.3 per minute (45.6% higher), the amplitude was 0.18 ± 0.03 mV (20% higher, $p < 0.05$). The spike frequency was 1.53 ± 0.12, the spike amplitude was 0.18 ± 0.02 mV, which were observed in all cases. The power of tonic contractions was 1.44 ± 0.13 (an increase of 74.5%, $p < 0.05$), the power of phase contractions was 0.270 ±0.08 (an increase of 170%, $p < 0.001$), propulsive activity was 5.3 ± 0.4 (a decrease of 35.7%, $p < 0.05$). That is, in CP, pronounced hypomotor dyskinesia of the stomach was observed with a predominance of spasm of the circular and partially oblique muscle layers, which determines a decrease in the propulsive activity of the stomach.

Electromyographically, in patients with chronic pancreatitis, the frequency of slow waves of small intestine EMA was 9.9 ± 1.0 per minute (100.5% lower than normal), the amplitude was 0.14 ± 0.02 mV (decrease by 40%, $p < 0.05$). The spike frequency was 0.97 ± 0.08, the spike amplitude was 0.06 ± 0.01 mV, spikes were observed in 84% of cases. The power of tonic contractions was 1,386 ± 0.15 (decrease by 47%, $p < 0.05$), the power of phase contractions was 0.0582 ± 0.001 (decrease by 41.8%, $p < 0.05$), propulsive activity was 23.8 ± 1.4 (increase by 19%, $p < 0.05$). That is, moderate hypermotor dyskinesia of the small intestine was observed in CP due to a marked decrease in the tone of the circular muscles, which may be due to secretory insufficiency in pancreatic pathology.

Slow waves of EMA of the ascending colon of patients with chronic pancreatitis were characterized by a frequency of 10.7 ± 1.4 per minute (decrease by 2.7%, $p > 0.1$), amplitude - 0.13 ± 0.05 mV (increase by 30% $p < 0.05$). Spikes with an amplitude of 0.07±0.001 mV followed with a frequency of 0.92 ± 0.09 and were observed in 85.6% of cases. The power of tonic contractions was 1,391 ±0.14 (an increase of 26.4%, $p < 0.001$), the power of phase contractions was 0.0644 ± 0.11 (a decrease of 35.6%, $p < 0.05$), propulsive activity was 14.8 ± 1.2 (an increase of 21.6%, $p < 0.05$). That is, in CP, hypermotor dyskinesia of the smooth muscles of the ascending colon was observed due to a decrease in the power of contractions of the circular muscle layer.

Slow waves of EMA of the descending colon of patients with chronic pancreatitis were characterized by a frequency of 8.5 ± 0.9 per minute (higher by 54.5%, $p < 0.05$), an amplitude of 0.16 ± 0.02 mV (higher by 60%, $p < 0.05$). Spikes with an amplitude of 0.09 ± 0.005 mV followed with a frequency of 1.0 ± 0.08 and were observed in all observations.

The power of tonic contractions was 1.36 ± 0.12 (an increase of 23.6%, $p < 0.001$), the power of phase contractions was 0.09 ± 0.11 (a decrease of 10%, $p < 0.05$), propulsive activity was 15.1 ± 1.2 (an increase of 15.2%, $p < 0.05$). That is, hypermotor dyskinesia of the descending colon and colon as a whole was observed, which may be associated with the development of intestinal dysbiosis due to secretory insufficiency of the pancreas in CP.

The study showed that the motor function of the gastrointestinal tract in chronic pancreatitis is characterized by a decrease in the propulsive activity of the stomach, which is clinically accompanied by a feeling of rapid saturation, hypermotor dyskinesia of the ascending and descending sections of the colon, caused, apparently, by secretory insufficiency of the pancreas in CP.

In pancreatitis, the clinical picture may contain a dry syndrome of Sjogren's syndrome, which is characterized by autoimmune damage to the salivary glands. To recognize Sjogren's syndrome, a triad of signs is used: dry keratoconjunctivitis; xerostomia and/or parenchymal mumps - diseases in the pathogenesis of which autoimmune reactions are involved. Sjogren's disease is a chronic inflammatory disease of the exocrine glands, primarily salivary and lacrimal, but also exocrinocytes of the pancreas with the gradual development of their secretory insufficiency in combination with various systemic manifestations that significantly change the processes of primary processing

of food contents in the oral cavity and, due to secretory insufficiency of the pancreas, a violation of the digestive processes in the small intestine and the microbial landscape in the small and large intestine.

Patients with pancreatic pathology often have various immune disorders. The close connection of the pancreas and salivary glands is demonstrated by the presence of antibodies to the tissue antigen of the salivary glands in 1/3 of patients with chronic pancreatitis with the presence of antibodies to the tissue antigen of the pancreas, therefore, the condition of the salivary glands may be an early harbinger of the development of CP.

Conclusion

Electromyography can be used to study the motor function of the gastrointestinal tract in chronic pancreatitis.

The pathology of the pancreas in CP determines the nature of the violation of the motor function of the gastrointestinal tract - hypomotor dyskinesia of the stomach and hypermotor dyskinesia of the small and large intestine due to secretory insufficiency of the pancreas and developing intestinal dysbiosis.

Bibliography

1. Trukhan DI., *et al*. "Changes in organs and tissues of the oral cavity in diseases of internal organs". M.: Practical medicine (2012): 208.
2. Trukhan L Yu., *et al*. "Changes in organs and tissues of the oral cavity in diseases of internal organs". *Handbook of the Polyclinic Doctor* 7 (2011): 8-14.
3. Mayev IV and Kucheryavy Yu A. "Diseases of the pancreas". In 2 volumes. Moscow: JSC "Publishing house "Medicine", publishing house "Niko" (2008): 976.
4. Xiao AI., *et al*. "Global morbidity and mortality from pancreatic diseases: a systematic review, meta-analysis and meta-regression of population-based cohort studies". *The Lancet Journal of Gastroenterology and Hepatology* 1.1 (2016): 45-55.
5. Wu BU., *et al*. "The effect of outpatient triglyceride levels on the risk of recurrence in patients with hypertriglyceridemic pancreatitis". *Diseases of the Digestive System and Scientific Research* 64.3 (2019): 890-897.
6. Whitcomb DK., *et al*. "Hereditary pancreatitis is caused by a mutation in the cationic trypsinogen gene". *Natural Genetics* 14.2 (1996): 141-145.
7. Scherer N., *et al*. "Mutations of the cystic fibrosis gene in patients with chronic pancreatitis". *New England Journal of Medicine* 339.10 (1998): 645-652.
8. Okhmuraya M and Yamamura K. "The role of the serine protease inhibitor Kazal type 1 (SPINK1) in pancreatic diseases". *Experimental Animals/Japan Association for Laboratory Animal Research* 60.5 (2011): 433-444.
9. Curly YU., *et al*. "Mutations of the pancreatic trypsin N34S secretion gene with global idiopathic pancreatitis". 7 (2011): 7-12.
10. Di Leo M., *et al*. "A meta-analysis of the effect of the variability of the SPINK1 p gene was carried out.N34S for Caucasian patients with chronic pancreatitis". *Diseases of the Digestive System and Liver* 49.8 (2017): 847-853.
11. Curly Yu A and Oganesyan TS. "Autoimmune pancreatitis: diagnostic algorithms and treatment approaches". *Clinical Perspectives of Gastroenterology, Hepatology* 6 (2010): 3-10.

12. Okhlobystin AV. "Autoimmune pancreatitis: new ideas about pathogenesis, diagnosis and treatment". *Proven Gastroenterol.* 1 (2013): 22-27.

13. Okazaki K., *et al*. "Modern concept and diagnosis of IgG4-related diseases in the hepatobiliary-pancreatic system". *Journal of Gastroenterology* 48.3 (2013): 303-314.

14. Okazaki K and Uchida K. "The modern concept of autoimmune pancreatitis and diseases associated with IgG4". *American Journal of Gastroenterology* 113.10 (2018): 1412-1416.

15. Funkhauser GT., *et al*. "Minimally invasive treatment of visceral artery aneurysms and pseudoaneurysms". *Journal of Vascular Surgery* 53.4 (2011): 966-970.

16. Naito I., *et al*. "Pancreatic hemosuccus associated with segmental arterial mediolysis was successfully treated with transarterial embolization". *IOP: Journal of the Pancreas* 11.6 (2010): 625-629.

17. Mayev IV., *et al*. "Autoimmune pancreatitis". Textbook for the system of postgraduate education of doctors specializing in gastroenterology: Moscow: Federal State University "FIRO" of the Ministry of Education and Science of the Russian Federation (2011).

18. Kager LM., *et al*. "The results of conservative, endoscopic and surgical treatment of acute pancreatitis: a systematic review". *Journal of Clinical Gastroenterology* 51.8 (2017): 749-754.

19. DiMagno MU and Dimagno ER. "Chronic pancreatitis". *Current Opinion in Gastroenterology* 26.5 (2010): 490-498.

20. Jupp J., *et al*. "Epidemiology and socio-economic consequences of chronic pancreatitis". *Best Practices and Research in the Field of Clinical Gastroenterology* 24.3 (2010): 219-231.

21. Shimizu K and Shiratori K. "Chronic pancreatitis. 1. Its epidemiology and symptoms". *The Journal of the Japanese Society of Internal Medicine "Nihon Naika Gakkai Zasi"* 99.1 (2010): 36-40.

22. Spanier BV., *et al*. "Epidemiology, etiology and outcomes of acute and chronic pancreatitis: updated information". *Best Practices and Scientific Research in the Field of Clinical Gastroenterology* 22.1 (2008): 45-63.

Volume 11 Issue 5 May 2024