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FEATURES OF DIARRHEA IN PATIENTS WITH CHRONIC PANCREATITIS

Abstract

Chronic pancreatitis (CP) is a chronic inflammatory condition of the pancreas that often leads to exocrine insufficiency. A common manifestation is diarrhea, which reduces quality of life and leads to nutritional deficiencies. This paper reviews the main pathogenetic mechanisms, clinical features, diagnostic approach, and treatment options for CP-related diarrhea.

Keywords: chronic pancreatitis, diarrhea, exocrine insufficiency, steatorrhea, enzyme therapy

Introduction.

Chronic pancreatitis (CP) is a chronic inflammatory disease of the pancreas that leads to irreversible morphological changes, progressive fibrosis, and a decline in both exocrine and endocrine function. The global incidence of CP varies from 4 to 10 cases per 100,000 population annually, with increasing prevalence among younger individuals due to alcohol abuse and dietary habits. One of the most debilitating symptoms of CP is chronic diarrhea, which occurs in approximately 30–40% of patients. In most cases, CP develops at the age of 35-50 years. This condition leads to significant weight loss, malnutrition, fat-soluble vitamin deficiencies, and reduced quality of life [8].

Pathogenesis and Mechanisms of Diarrhea.

The primary cause of diarrhea in CP is exocrine pancreatic insufficiency (EPI), resulting from acinar cell destruction and a decrease in digestive enzyme production [4]. Impaired lipid digestion leads to steatorrhea, a hallmark of EPI [6]. Additional mechanisms include bile acid malabsorption, small intestinal bacterial overgrowth (SIBO), and altered intestinal pH and motility [2]. These pathophysiological processes contribute to frequent, greasy, foul-smelling stools and nutritional deficiencies, particularly of vitamins A, D, E, and K [8]. As a result, patients experience diarrhea, steatorrhea, flatulence, loss of appetite, etc. Clinically expressed insufficiency of the exocrine pancreatic function develops only when its functional activity decreases by more than 90%. Clinical manifestations of impaired fat absorption include steatorrhea and flatulence, weight loss (in 30–52% of patients).

Clinical Presentation.

Patients with CP-associated diarrhea commonly report frequent bowel movements (3–6 or more times per day), steatorrhea, bloating, nausea, and unintended weight loss [6]. Appetite may be reduced, and fatigue is common. Chronic nutrient malabsorption can result in anemia, hypoproteinemia, and micronutrient deficiencies manifesting as muscle cramps, dry skin, visual disturbances, and increased susceptibility to infections [6].

Differential diagnosis of diarrhea in CP includes distinguishing it from irritable bowel syndrome (IBS), inflammatory bowel disease (IBD), celiac disease, and parasitic infections. IBS is functional in nature and does not involve steatorrhea or significant weight loss [6]. IBD (e.g., Crohn’s disease, ulcerative colitis) is often associated with bloody stools and systemic inflammation, confirmed by endoscopy. Celiac disease is marked by positive serological markers (e.g., anti-tTG antibodies) and villous atrophy on biopsy. Parasitic causes such as giardiasis or amoebiasis are diagnosed through stool microscopy or PCR. CP-related diarrhea is best distinguished by low fecal elastase-1 levels and imaging findings of pancreatic fibrosis or ductal changes.

Diagnostic evaluation of CP-related diarrhea includes fecal elastase-1 measurement (values below 200 μg/g indicate EPI), 72-hour fecal fat test, breath tests for SIBO, and abdominal imaging such as ultrasound, CT, or MRI [2]. Endoscopic ultrasound (EUS) is useful for detecting subtle structural changes. Nutritional assessment involves biochemical markers and anthropometry.

Calculation of the fat absorption coefficient requires a stool collection for 72 hours after ingestion of a known amount of fat (100 g/day). Excretion of more than 7 g/day of fat in the stool indicates fat malabsorption, while excretion of more than 15 g/day is considered severe fat malabsorption. However, this procedure is difficult for both patients and laboratory personnel and is rarely performed in routine practice. In general, indirect tests are moderately sensitive and specific for the diagnosis of CP [7].

Pancreatic elastase-1 is a human-specific enzyme that does not degrade during intestinal transit, is enriched 5-6 times in feces and, therefore, is a marker of exocrine pancreatic function [7]. Normally, the elastase level is more than 200 μg/g of feces. In mild to moderate exocrine insufficiency, its content fluctuates within 100-200 μg/g of feces. In severe exocrine insufficiency, the elastase content is less than 100 μg/g of feces. This simple, fast and inexpensive method has no limitations in application and allows to determine the state of the exocrine function of the pancreas at earlier stages.

Treatment. The main principles of treatment of SIBO are therapy of the underlying disease, which became the main reason for its development, decontamination of opportunistic microflora and restoration of eubiosis. It would be advisable to prescribe adequate doses of enzyme preparations, which are the basic treatment for CP with SIBO .The mainstay of treatment is pancreatic enzyme replacement therapy (PERT), typically using pancreatin at doses of 25,000–50,000 lipase units with meals. Dietary modification includes low-fat intake and supplementation of fat-soluble vitamins. For SIBO, antibiotics such as

rifaximin are prescribed, along with probiotics [2]. In cases of bile acid malabsorption, bile acid sequestrants like cholestyramine may be used. The effectiveness of treatment is monitored by stool consistency, frequency, and nutritional improvement.

Conclusion.

Diarrhea in chronic pancreatitis is multifactorial, requiring an integrated and personalized approach. Timely diagnosis and effective therapy including PERT and adjunctive treatments significantly improve digestive function, nutritional status, and quality of life in affected patients. The modern list of drugs and the correctness of their use enable the practicing physician to carry out correction and prevention of disorders of the function of the pancreas and the microbiocenosis of the small intestine in each specific case with high efficiency and safety. Future research should focus on standardizing diagnostic criteria and optimizing therapeutic regimens.

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