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CONTEMPORARY CONCEPTS OF CHRONIC PANCRYATITIS

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Chronic pancreatitis (CP) is a prolonged inflammatory disease of the pancreas, manifested by irreversible morphological changes in the parenchyma and organs, causing pain and persistent decline in function [3,4].

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The prevalence of CP in Europe is 25.0-26.4 cases per 100,000 population. The incidence of CP in developed countries ranges from 5-10 cases per 100,000 population; globally, it ranges from 1.6-23 cases per 100,000 population per year. Globally, there is a trend towards an increase in the incidence of acute and chronic pancreatitis, which has more than doubled over the past 30 years. CP usually develops in adulthood (35-50 years) [4,5,6].

In developed countries, the average age since the diagnosis was established has decreased from 50 to 39 years, men are 2 times more likely to suffer from CP than women, there is a trend towards an increase in the proportion of women among those infected (30%); primary disability of patients reaches 15%. The mortality rate after the initial diagnosis of CP is up to 20% within 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 from secondary digestive disorders and infectious complications [7,8,9].

The most typical clinical manifestations of CP are abdominal pain and symptoms of gastric insufficiency, however, in some cases, the clinical picture of the disease can manifest as a clinical picture of complications [7,8,9].

Abdominal pain is the main symptom of CP. The pain is usually localized in the epigastrium and radiates to the back, intensifying after eating and decreasing in the sitting or forward position. Pain is observed in 80-90% of patients, "painless pancreatitis" is observed in 10-20%. Pain attacks may recur (type A: short-term pain episodes lasting up to 10 days against the backdrop of long periods of painlessness), sometimes patients experience constant pain (type B: more severe and prolonged episodes with periods of painlessness lasting 1-2 months, more often observed in alcoholic CP) [4,5,8].

Clinically manifested pancreatic insufficiency occurs only when the functional activity of the pancreas decreases by more than 90%. Clinical manifestations of fat absorption disorders include steatoresis and meteorism, and weight loss (30-52% of patients). Alcoholic pancreatitis often leads to exocrine insufficiency than pancreatitis of other etiologies. In patients with alcoholic pancreatitis, maldigestive symptoms arise on average 10 years after the first clinical symptoms appear. Endocrine pancreatic insufficiency develops over time in 70% of CP patients in the form of impaired glucose tolerance [9,10,11].

Diabetes mellitus occurs with prolonged CP, the likelihood of developing diabetes mellitus gradually increases 10 years after the onset of CP manifestations. Pancreatogenic diabetes mellitus differs from type 1 and 2 diabetes mellitus in that it carries a higher risk of developing hypoglycemia and a lower incidence of ketoacidosis [7,8].

To date, pancreatogenic diabetes mellitus as a result of CP development should be attributed to type 3c. Complications such as macro/microangiopathy, nephropathy, neuropathy, and retinopathy are as common as type 1 diabetes [9,12,13].

Approximately 1/3 of patients with CP have pseudocysts of the pancreas, they can be of varying sizes, often asymptomatic, or provoke a clinical picture of compression of adjacent organs, causing pain in the upper abdomen. Spontaneous

regression of pseudocysts in CP occurs less often than in acute pancreatitis (43); spontaneous regression in patients with alcoholic CP is described in 25.7% of cases, and persistence without clinical manifestations - in 23% (44). The risk of developing serious complications in the asymptomatic course of chronic pseudocyst is <10% [10,11,12].

The exacerbation of CP and repeated attacks of acute pancreatitis against the background of CP can lead to pancreatitis with the development of infectious complications (inflammatory infiltrates, abscesses, purulent cholangitis, septic conditions). Duodenal stenosis develops in less than 5% of cases. The main cause of duodenal obstruction is the spread of inflammation to the paroduodenal tissue and the wall of the duodenal wall. The development of a decompensated form of disruption of evacuation from the stomach occurs rarely. The swelling and development of gastric fibrosis can cause compression of the common bile duct with the development of mechanical jaundice (16-35% of patients) [1,2,3].

In some cases, jaundice may be persistent or may be of a recurrent nature, with a slight risk of developing secondary cirrhosis of the liver. The onset of jaundice is preceded by pain in the upper abdomen, which is characteristic of exacerbation. Portal hypertension, caused by compression or thrombosis of the portal, upper mesenteric or splenic veins, as a result of inflammation and fibrosis of the peripancreatic tissue, is observed in 7-18% of patients. Bleeding from phlebetacia of the digestive system or stomach is not a frequent complication of CP. Intestinal bleeding in patients with CP is a pathognomonic symptom of false abnormalities (LA) in the branches of the uterine trunk and upper biliary artery [4,8,9,10].

Meanwhile, gastric duodenoscopy does not reveal any ulcers in the stomach and duodenum, or phlebema of the esophagus. Malabsorption syndrome with micronutrient deficiency is a consequence of progressive pancreatic fibrosis and pancreatic maldigestion, which is not controlled by substitute enzyme therapy. Therefore, signs of irreplaceable macro-and micronutrient deficiency are also attributed to complications of CP with external secretory insufficiency of the pancreas. Patients with CP are at risk of developing vitamin B deficiency (A, D, E, and K) and osteoporosis is a confirmed complication of CP. With prolonged CP,

the risk of developing prostate cancer increases. For patients with a five-year history of CP, the risk of developing cancer increases 8 times [7,10,11,12].

A precursor to prostate cancer can be pancreatic intraepithelial neoplasia (Pancreatic intraepithelial neoplasms - PanIN) - specific morphological changes in the walls of the ducts [12,13].

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