International Journal of Research
Available at https://pen2print.org/index.php/ijr/
e-ISSN: 2348-6848
p-ISSN: 2348-795X
Volume 06 Issue 2
February 2019

Peculiarities of Chronic Pancreatitis with External Necessary Failure

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Annotation. *In clinical terms, chronic pancreatitis is a dynamic, poorly identifiable disease, the evolution of which is determined by: the cause caused, the activity of the process, the state of the surrounding organs, functionally affecting the workings of the RV. The main symptom, indicating the development of functional insufficiency of the pancreas, is the emergence of clinical manifestations of digestive disorders. The appearance of the symptom complex on the part of the gastrointestinal tract is the basis for the objectification of exocrine pancreatic insufficiency and the determination of the degree of its manifestation. The article describes the characteristics and the most common diseases.*

Key words: Chronic pancreatitis, glands, pancreatic elastase, pancreas.

Chronic pancreatitis (CP) is a dynamic, difficult to diagnose disease, the evolution of which is due to the progression of the destruction of pancreatic tissue (PC) and the development of complications. Dynamic observation allows you to determine the facts that affect the rate of progression, stage, nature and effectiveness of therapeutic measures. Most researchers consider CP as a progressive disease with recurring exacerbations of chronic inflammation, the development of sclerosis and fibrosis, leading to the replacement of the glandular secretory tissue with connective tissue and a decrease in the external secretion of the pancreas.

The leading symptoms in the clinical picture of CP are pain and signs of insufficiency of the exocrine function of the pancreas.

The main clinical manifestation of CP - pain syndrome, leads to disability of the patient in the physical and labor sphere: pain in the epigastric region in the comparison group was observed in 28 (58.3%) patients, and in the main group - in 46 (88.5%) ($P < 0.001$; $\chi 2 = 11.78$): pain in the hypochondrium in patients of the comparison group - in 20 (41.7%); in the main - in 35 (67.3%) ($P < 0.001$; $\chi 2 = 6.63$).

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Among the clinical manifestations of CP in the presence of exocrine insufficiency, diarrheal syndrome (92.3%) (P <0.001; $\chi 2$ = 39.92) and asthenia syndrome (57.7%) (P <0.001; $\chi 2$ = 10.05) were significantly more common.

Increased saliva production in patients with chronic pancreatitis with exocrine insufficiency is 2.6 times more common than in patients with chronic pancreatitis without it (Table 3.1).

Table 3.1

Complaints of the examined patients with chronic pancreatitis

Complains	Comparison group (n=48)		Main group (n=52)		χ^2	P
	abc	%	abc	%		
Epigastric pain	28	58,3	46	88,5	11,78	<0,001
Pain in hypochondrium	20	41,7	35	67,3	6,63	<0,01
Reduced appetite	17	35,4	38	73,1	14,30	<0,001
Flatulence	36	75,0	48	92,3	5,56	<0,001
Nausea	21	43,8	33	63,5	3,90	<0,05
Vomiting	7	14,6	18	34,6	5,34	<0,05
Belching	16	33,3	38	73,1	15,87	<0,001
Heartburn	12	25,0	26	50,0	6,62	<0,01
Increased saliva production	8	16,7	21	40,4	6,82	<0,01
Diarrhea	15	31,3	48	92,3	39,92	<0,001
The feeling of heaviness in the right hypochondrium	24	50,0	45	86,5	15,58	<0,001
Constipation alternating with diarrhea	26	54,2	42	80,8	8,12	<0,01
Weight loss	12	25,0	30	57,7	10,95	<0,001

The average BMI in the examined patients was 22.3 ± 0.9 kg / m2. Among all patients with CP, insufficient weight was recorded in 3 (6.3%) patients of the

1st group and in 24 (46.2%) - 2 groups (P <0.001; χ2 =), with a normal mass, the number of patients of the 1st group p 1.4 times was more than in group 2.

In 12 (25.0%) patients of the 1st group and in 3 (5.8%), obesity of the first degree was overweight P <0.001; χ2 =), weight loss (from 1 to 10 kg) was observed in 5 (10.4%) patients with CP 1 group and in 25 (48.1%); on average, the mass loss was 5.3 ± 1.1 kg.

Weight loss was observed in patients with all etiological forms of CP, significantly more often in patients with CP of toxic etiology, compared with biliary-dependent CP.

Table 3.2

The characteristic of body weight of the examined patients with CP

Indicators	Comparison group (n=48)		Main group (n=52)		χ^2	P
	abc	%	abc	%		
Underweight	3	6,3	24	46,2	8,55	<0,001
Normal body weight	33	68,8	25	48,1	11,53	<0,05
First degree obesity	12	25,0	3	5,8	0,67	<0,01
Slimming	5	10,4	25	48,1	2,72	<0,001

An analysis of concomitant diseases in patients with chronic pancreatitis showed that the presence of which leads to aggravation of premorbid background, thereby worsening the course of the disease (Table 3.3).

Table 3.3

Concomitant diseases in patients with chronic pancreatitis

Types of diseases	Comparison group (n=48)		Main group (n=52)		χ^2	P
	abc	%	abc	%		
Chronic cholecystitis	28	58,3	44	84,6	8,55	<0,001
Chronic gastritis	16	33,3	35	67,3	11,53	<0,001

Chronic pyelonephritis	4	8,3	7	13,5	0,67	>0,05
Chronic hepatitis	8	16,7	16	30,8	2,72	>0,05
Cholelithiasis	5	10,4	13	25,0	3,60	<0,05
Ischemic heart disease	10	20,8	22	42,3	5,29	<0,05
HD (Hypertonic disease)	7	14,6	10	19,2	0,38	>0,05

Chronic cholecystitis was observed in 58.3% of patients in the comparison group, which was significantly lower, namely, 45.1% (P <0.001; $\chi 2 = 8.55$) than in the main group. Chronic pyelonephritis, chronic hepatitis are less common, their differences in the groups are unreliable (P> 0.05; $\chi 2 = 0.67$ and $\chi 2 = 2.72$).

According to the ultrasound of the abdominal cavity revealed various structural changes of the pancreas, presented in table 3.4

Table 3.4

Ultrasound signs of CP in examined patients

Indicators	Comparison group (n=48)		Main group (n=52)		χ^2	P
	abc	%	abc	%		
Homogeneous diffuse increase in echogenicity of the parenchyma, preservation of the pattern	19	39,6	38	73,1	11,42	<0,001
Painting "cobblestone pavement"	15	31,3	32	61,5	9,19	<0,01
Medium and dense echoes, unevenly distributed on a normal background	17	35,4	35	67,3	10,17	<0,001
Inhomogeneous distribution of echo signals with alternation of dense and cystic areas	9	18,8	27	51,9	11,92	<0,001

Extreme variability of the amplitude and length of echosignals	21	43,8	44	84,6	18,32	<0,01
Increase in pancreas	24	50,0	47	90,4	19,77	<0,001
Calcification of pancreatic tissues	14	29,2	33	63,5	11,78	<0,001
Stone in the pancreatic duct	7	14,6	18	34,6	5,34	<0,05
Cysts	5	10,4	18	34,6	8,25	<0,001
Expansion of pancreatic duct (> 2.5 mm)	14	29,2	29	55,8	7,21	<0,001
Deformation G:	11	22,9	26	50,0	7,85	<0,01
Increased density of the pancreas tissue	8	16,7	17	32,7	3,42	>0,05

Thus, the uneven echogenicity of the pancreatic parenchyma was typical for all patients examined and was found in 100% of cases. The most pronounced structural changes were noted in patients of group 2 with CP and exocrine insufficiency. For patients of group 2, conditional specificity can be identified — the tendency to pancreatofibrosis, calcification, ductal hypertension — that is, the most gross structural changes of the parenchyma and ductal system of the pancreas.

Pancreatic elastase is synthesized in the acinar cells of the pancreas and is produced as an inactive proenzyme along with other enzymes in the duodenum, where it is converted into elastase by the action of trypsin. The enzyme is not affected when passing through the intestinal tract, therefore its content in feces is used as an indicator of the exocrine function of the pancreas.

The specificity of the test in the study of feces is 93.9%, sensitivity - 93.2%, which makes this method an almost perfect test, the "gold standard" for diagnosing disorders of the exocrine pancreas function.

Clinical manifestations of exocrine pancreatic insufficiency: feeling of heaviness in the stomach after eating, bloating, various stool disorders (constipation, diarrhea, polyfecalia - more than 1 kg), discoloration, smell and consistency of feces (greasy, gray, mushy, viscous), the presence of undigested food in the feces, weight loss. The content of pancreatic elastase, low in newborns, reaches the level of

adults by 2 weeks of age. Pancreatic elastase can enter the blood in increased amounts in case of inflammation of the pancreas (pancreatitis).

Only early diagnosis can prevent the death of this disease. The determination of pancreatic elastase in the stool is valuable to confirm or eliminate the presence of exocrine pancreatic insufficiency due to chronic pancreatitis.

The advantages of detecting pancreatic elastase in feces are that pancreatic elastase is absolutely specific for the pancreas, stable in the process of intestinal transit, the concentration in the feces reflects the secretory function of the pancreas, enzyme replacement therapy does not affect its concentration enzyme insufficiency of the pancreas, but also to evaluate the dynamics of the exocrine function.

The level of concentration of pancreatic elastase in the feces is almost not affected by the presence of any concomitant diseases in the patient. Orally taken enzyme preparations also do not affect the concentration of FE (fecal elastase), since only endogenous human elastase is detected (Table 3.5).

Table 3.5

The results of the feces to determine the level of pancreatic elastase in examined patients

Indicators	Comparison group (n=48)		Main group (n=52)		χ^2	P
	abc	%	abc	%		
> 200 pg / g - normal	48	100,0	0	0,0		-
moderate failure	0	0,0	29	55,8		<0,001
<100pg / g - severe exocrine insufficiency	0	0,0	27	51,9		<0,001

Table 3.5 presents our data, analyzes the frequency of occurrence of the analyzed outcomes of CP, depending on the etiology of the disease, and obtained original data. Thus, endocrine insufficiency of the pancreas occurred in 17.1% of the included patients, significantly more often in patients with toxic and idiopathic CP relative to the subgroup with biliary-dependent CP (P <0.05). The reasons for the lower incidence of endocrine insufficiency in patients with CP of mixed etiology remained unclear to us. This is probably

due to the potential heterogeneity of the group, the frequency of occurrence of endocrine insufficiency of the pancreas in which it occupied an intermediate position relative to other identified etiological forms, while comparative analysis did not reveal statistically significant differences with any of the selected groups.

Primary exocrine pancreatic insufficiency (due to a decrease in the secretion of elastase-1 feces) was registered in 32.5% of cases, i.e. almost 3 times less than when analyzing the frequency of steatorrhea (80%) and the presence of neutral fat in the stool during microscopy (90.7%). This can be explained by the fact of the dominance of the secondary mechanisms of pancreatic insufficiency (violation of segregation, SIBR, intestinal hyperthermia, etc.). These facts are confirmed by the intragroup analysis carried out by us, indicating a small frequency (2.5%) of primary pancreatic insufficiency in patients with biliary-dependent CP, who have less pronounced structural changes in the parenchyma and pancreatic duct system. Moreover, in this group only a slight degree of insufficiency is registered. In the rest of the comparison groups, primary pancreatic insufficiency was found more reliably (more than an order of magnitude). Patients with toxic and idiopathic CP were characterized by the highest frequency of low values of fecal elastase, significantly exceeding that in subgroups of patients with biliary-dependent and mixed CP ($P < 0.05$ in both cases). Analysis of frequencies depending on the severity of primary pancreatic insufficiency showed that in patients with toxic and mixed CP, mild degrees of insufficiency prevailed, while in patients with idiopathic CP all 3 degrees met with a comparable frequency.

Summary. Violation of the exocrine activity of the pancreas (PC) leads to both deep digestive disorders, affecting gastrointestinal secretion, absorption and motility, and metabolic changes in the body.

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e-ISSN: 2348-6848
p-ISSN: 2348-795X
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