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PECULIARITIES OF INCIDENCE OF DIGESTIVE HYDROLASES OF THE STOMACH AND PANCREAS IN CHRONIC VIRAL HEPATITIS WITH C

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ABSTRACT-- *A comprehensive assessment of the state of the pancreas in the group of patients with chronic viral liver lesions revealed a violation of the functional state of the organ in 80% of cases with chronic hepatitis and in 96.3% of cases with cirrhosis [4.6].With combined infection with hepatitis B and C viruses, more pronounced changes in the blood proteinase activity (trypsin and kallikrein activity), a more distinct decrease in antitryptic activity are noted.Exocrine pancreatic function in patients with hepatitis was also more impaired, which was manifested in some studies by a decrease in basal and stimulated secretion, enzyme secretion (especially trypsin and amylase) and bicarbonate production [4,6], in others - by an increase in amylase and lipase activity . Violations of external secretion increase with increasing severity of the main clinical and biochemical syndromes. The content of glucagon and somatostatin in the blood of patients with chronic hepatitis is increased.*

Keywords*--stomach, pancreas, hepatitis, blood hydrolases, pepsinogen, kallikrein*

I. INTRODUCTION

Despite the long period, starting from the sixties of the last century, of studying the effect of chronic liver diseases on the change in the functional state of the stomach and pancreas, there is still no unambiguous idea about the change in their exosecretion and growth. It remains unclear the issue of the multidirectional changes in the

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secretory and incretory activity of the digestive glands of the stomach and pancreas in chronic liver diseases. At the same time, the study of changes in incretory function is of interest for practical diagnostics. both in diseases of the stomach and pancreas, and a violation of their function in chronic liver diseases.

A comprehensive assessment of the state of the pancreas in the group of patients with chronic viral liver lesions revealed a violation of the functional state of the organ in 80% of cases with chronic hepatitis and in 96.3% of cases with cirrhosis [4,6].

With combined infection with hepatitis B and C viruses, more pronounced changes in blood proteinase activity (trypsin and kallikrein activity), a more distinct decrease in antitryptic activity are noted.

Exocrine pancreatic function in patients with hepatitis was also more impaired, which was manifested in some studies by a decrease in basal and stimulated secretion, enzyme secretion (especially trypsin and amylase) and bicarbonate production [4,6], in others - by an increase in amylase and lipase activity .

Violations of external secretion increase with increasing severity of the main clinical and biochemical syndromes. The content of glucagon and somatostatin in the blood of patients with chronic hepatitis is increased [5,6].

In alcoholic liver disease, exocrine pancreatic secretion tends to increase with the severity of liver damage, but does not correlate with the severity of chronic pancreatitis

In alcoholic liver disease, exocrine pancreatic secretion tends to increase with the severity of liver damage, but does not correlate with the severity of chronic pancreatitis It has been suggested that alcohol abuse and the effect of hepatitis virus have an equal pathogenic effect on the liver and pancreas [3].

Pancreatic enzyme levels - serum and pancreatic amylase, and serum lipase levels increase with progression of liver disease in patients diagnosed with viral hepatitis. Pancreatic disease, asymptomatic in most cases, may be an extrahepatic manifestation of chronic viral hepatitis. It has been suggested that the liver metabolism of amylase and lipase from the blood decreases in patients with chronic infectious liver diseases, especially in patients with cirrhosis, which can lead to the accumulation of these enzymes in the blood [12,18].

As for the stomach, it was shown that in patients with cirrhosis of the liver, the average rates of free and totl acidity, as well as pepsinogen 1 in the blood serum, were lower than under normal conditions. Also, a decrease in blood flow was noted in the gastric mucosa, and the gastrin content was significantly lower than in the group of healthy patients. While the concentration of serum gastrin and somatostatin in patients with cirrhosis was significantly higher [7].

In another study in dogs with liver disease, hypergastrinemia and frequent manifestations of gastrointestinal disturbances that could be caused by ulceration were detected. The paper suggests that the liver is important for the inactivation of certain forms of gastrin. Therefore, hypergastrinemia is involved in the pathogenesis of gastrointestinal ulcerations associated with liver dysfunction [15].

Purpose of the study. To study the peculiarities of changes in the content of blood hydrolases added by the stomach and pancreas in chronic viral hepatitis C and give an analysis of the mechanisms of these changes.

Material and methods. 112 men and women aged 20 to 70 years were examined. For comparison, a group of healthy people was formed in the amount of 42 people who did not have markers of HCV infection, and liver tests were normal. Of the examined 70, there were positive serological markers, of which 38 people had markers regarding HCV post-infection, 32 had markers related to chronic HCV infection. For all examined in blood serum

by ELISA (standard kits of CJSC Vector-Best, Russia), the determination was made: pepsinogen-1 (PG1) and pepsinogen-2 (PG2). Pancreatic amylase (standard kits of Vector-Best CJSC, Russia) and pancreatic lipase HUMAN, Germany were determined by biochemical methods. Hepatic tests were studied in all patients: alanine transaminase (ALT), aspartate aminotransferase (AST), total and direct bilirubin.

II. RESULTS AND ITS DISCUSSION.

It was found that in the examined healthy individuals the indicators of amylase, lipase, pepsinogen-1 and pepsinogen-2 in the blood were within normal limits (Table 1). In individuals with HCV post-infection (Table 1), liver function tests were within normal limits, but higher than in healthy subjects. In these individuals, despite the absence of an active HCV process, the levels of amylase and lipase in the blood were significantly higher than normal and compared to healthy ones. At the same time, the indicators of pepsinogen-1 were within normal limits, but were lower than in healthy ones, while the indicators of pepsinogen-2 were slightly higher than in healthy ones, but within normal limits.

Table 1: Changes in the content of stomach and pancreas hydrolases in the blood of healthy and sick with viral hepatitis C

Serum markers	Healthy	HCV post-infection	Chronic HCV infection
Liver tests			
AST (mmol / h * L) Norm 0,1-0,68	0,36±0,04	0,47±0,05	1,26±0,13
ALT (mmol / h * L) Norm 0,1-0,68	0,21±0,02	0,38±0,04	0,89±0,09
Total bilirubin (μmol / L) Norm 8,5-20,5	13,6±1,2	18,3±1,9	61,5±6,7
Direct bilirubin (μmol / L) Norm 0-5.0	2,0±0,1	3,9±0, 4	34,2±4,27
Blood hydrolases			
Amylase Pancreatic Norm 0-60 U / L	41,6±5,8	85,2±11,4	129,7±15,2
Pancreatic lipase Norm 0-53 U / L	26,8±3,7	69,5±8,1	94,4±12,6

Pepsinogen-I (μg / L) Norm 40–130	98,6±12,5	48,9±6,3	19,5±2,3
Pepsinogen- II (μg / L) Norm 4–22 (мкг/л)	16,9±1,8	18,4±2,1	11,2±1,4

In patients with chronic HCV infection, the indicators of all hepatic samples taken were above normal. In these same patients, a marked increase was noted above the norm of amylase and lipase compared with patients with HCV post-infection. At the same time, the indicators of pepsinogen-1 were less than the lower limit of the norm and significantly lower than in individuals with HCV post-infection. At the same time, the indicators of pepsinogen-2 were within normal limits, but were slightly lower than in individuals with HCV post-infection.The data presented demonstrate that in healthy individuals all the indicators taken into account were within normal limits. At the same time, in patients with HCV post-infection, the blood amylase and lipase levels are higher than normal, and pepsinogen-1 and pepsinogen-2 are normal, indicating the absence of significant changes in the function of the digestive glands of the stomach and a slight increase in the functional activity of the pancreas, which is possibly associated with latent form of pancreatitis.

In patients with chronic HCV infection, a marked increase in blood above the norm of amylase and lipase indicates an increase in the functional activity of the pancreas, and possibly a latent form of pancreatitis. The observed values below the norm of pepsinogen-1, which is produced by the main cells of the glands of the bottom and body of the stomach, indicate a decrease in the enzyme-excreting activity of the stomach, while a decrease in the concentration of serum pepsinogen-1 (PG1) to values less than 40 μg / l is observed with a noticeable decrease hydrochloric acid secretion and the development of atrophic gastritis.

[10]. Available indicators within the normal pepsinogen-2 (PG2), which is produced by mucin-forming cells of the glands of all parts of the stomach, indicates the absence of a change in the mucin-forming function of the stomach. At the same time, a change in the PG1 / PG2 ratio (19.5 / 11.3) below coefficient 3 is an additional indicator of the development of atrophic gastritis. Thus, in patients with chronic HCV infection, a marked increase in the functional activity of the pancreas is noted, which may be a manifestation of the latent form of pancreatitis and a decrease in the functional activity of the digestive glands of the stomach, which may be a manifestation of the latent form of atrophic gastritis. However, the mechanisms of these changes in the literature are not covered.

III. CONCLUSION

In our opinion, this is due to the physiological metabolism by the liver of low molecular weight peptides, in particular, CCK-8, which was shown by us in previous publications [1,2] and which is confirmed by a number of other researchers [8,9]. It has been shown that the liver affects the metabolism of CCK-8 and this metabolic effect can vary significantly with liver diseases. It was found that CCK-8 is metabolized to a large extent in healthy individuals and to a lesser extent in patients with cirrhosis. Due to this, the content of CCK-8 in the blood of patients with cirrhosis increases [17].

The physiological role of CCK-8 is known as a stimulator of pancreatic secretion [11,16]. At the same time, the results of a study of the physiological role of cholecystokinin as a regulator of gastrin secretion show that CCK-8 can play a decisive role in inhibiting the stimulated secretion of gastric acid and controls the production of stomach acid, plasma gastrin content and somatostatin secretion [13]. It was found that cholecystokinin inhibits acid secretion by activation of type A CCK receptors and a mechanism involving somatostatin production [14].

Thus, it can be assumed that, normally, CCK-8 is more utilized by the liver, and in chronic hepatitis C, its utilization in the liver is impaired and the concentration in the blood rises. Due to which, the mechanisms described above stimulate the secretion of the pancreas and the development of pancreatitis, while inhibiting the secretion of the stomach, and the development of atrophic gastritis.

Output. In patients with chronic HCV infection by incretion, an increase in the functional activity of the pancreas and the development of a latent form of pancreatitis, with a simultaneous decrease in the functional activity of the digestive glands of the stomach, which is a sign of the latent form of atrophic gastritis, have been established. We suggest that CCK-8 is the main factor contributing to the development of these disorders.

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