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CARBOHYDRATE METABOLISM DISORDERS IN PATIENTS WITH CHRONIC PANCREATITIS DUE TO COMORBIDITY WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Chronic pancreatitis (CP) and chronic obstructive pulmonary disease (COPD) have many mechanisms of mutual severity. Each nosology of this pathological combination can lead to glucose metabolism disorders. With the long-term course of CP, the development of secondary diabetes mellitus (DM) is possible due to a significant reduction in the area or number of functioning β-cells of the islets of Langerhans and absolute insulin deficiency.

The study's objective is to determine indicators of carbohydrate metabolism in patients with chronic pancreatitis with its isolated course and with comorbid COPD and diabetes mellitus. 100 patients with chronic pancreatitis of a mixed etiology in the exacerbation stage of moderate severity were examined. The first group of patients included 36 individuals with an isolated course of chronic pancreatitis (1 group), 2nd group included 33 patients with chronic pancreatitis and COPD, 3rd group included 35 patients with chronic pancreatitis and COPD and T3cDM. The control group (CCOPD) included 30 individuals with isolated COPD, the control group (CDM) includes 32 individuals with isolated type 2 DM. The group of comparison included 30 practically healthy individuals (PHI).

The state of glycaemia and regulation of carbohydrate metabolism in patients with CP were assessed depending on comorbid pathology of COPD and DM. Analysis of the obtained results showed a reliable increase of glucose content on an empty stomach in patients suffering from CP with an isolated course 1.4 times ($p<0.05$) in comparison with PHI, and the level of glycaemia increased when comorbid COPD joined CP: 1.5 times in comparison with PHI ($p<0.05$). At the same time, patients suffering from CP with two comorbid diseases – COPD and DM – developed 3.2 times increased glucose concentration on an empty stomach in comparison with PHI ($p<0.05$). This parameter in the 1st and 2nd groups 2.1 and 2.2 times increased respectively ($p<0.05$). The state of glycaemia on an empty stomach in patients from the 3rd group is similar to that with DM. Comparison of glucose content in the blood on an empty stomach in patients with type 2 diabetes mellitus with the parameter of this group found reliable 1.3 times difference ($p<0.05$), that appeared to be 2.4 times higher than that of PHI ($p<0.05$).

A reliably higher level of postprandial (after meals) glycaemia was found in patients with CP from the 1st group, that was 1.2 times higher than that of the control ($p<0.05$). At the same time, 1.4 times increase of postprandial glycaemia was registered in patients from the 2nd group ($p<0.05$), in the 3rd group –2.6 times as compared to the parameter of PHI ($p_{2,3}<0.05$), which is indicative of the dependence of a degree of tolerance disorder to glucose on comorbid COPD and manifested DM. Postprandial hyperglycemia was found in patients from the 5th group, that was 2.3 times higher than that of PHI ($p<0.05$), and that was 11.9 % lower than the parameters in the 3rd group ($p>0.05$).

Analysis of the laboratory findings concerning HbA1c content in the blood serum as a marker of persistence and intensity of hyperglycemia showed its reliable increase in patients from the 1, 2, 3 and 5 groups 1.2, 1.3, 1.4 and 1.4 times respectively in comparison with PHI ($p_{1,2,3,5}<0.05$), which confirms the role of chronic pancreatitis in the development of chronic postprandial hyperglycemia, advanced disorder to glucose tolerance, intensified glycosylation of transport proteins (hemoglobin), and further formation of DM.

Chronic pancreatitis in its exacerbation stage without comorbid pathology is associated with reliable postprandial hyperglycemia (1.2 times), an increased content of glycated hemoglobin (1.2 times), which is indicative of initial signs of carbohydrate metabolism dysfunction. These disorders are exacerbated under the conditions of accession of background COPD, and under conditions of three-component comorbidity with diabetes, decompensation of carbohydrate metabolism occurs.