MEDICAL SCIENCES

DIABETES MELLITUS AS A RESULT OF PANCREATIS

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Abstract
Today, the correction of exocrine insufficiency of the pancreas in patients with diabetes mellitus remains one of the most pressing problems of modern gastroenterology, despite sufficient experience in its study. That is why the management of such patients is a difficult task even for experienced doctors and requires an extremely careful approach.

Keywords: pancreatitis, diabetes, causes, symptoms, complication, prognosis, prevention

It is well known that patients with chronic pancreatitis develop a special form of diabetes mellitus - type III, caused by inflammatory and fibrous damage to islet cells. Unlike type I diabetes, type III diabetes is accompanied by diffuse damage to the endocrine part of the pancreas, which is a consequence of damage to both β-cells and α- and PP-cells. In addition, this form of diabetes is characterized by severe disorders of exocrine function, manifested by disorders of absorption of nutrients from the gastrointestinal tract, decreased secretion of incretins and decreased insulin secretion.

Pancreatogenic diabetes mellitus (type III diabetes mellitus) is a secondary disorder of glucose metabolism that develops as a result of damage to the incretory apparatus of the pancreas. The disease occurs in 10-90% of patients with chronic pancreatitis. This variability in data is associated with the difficulty of predicting the development of endocrine pancreatic dysfunction and the difficulty of differential diagnosis of pathology. After suffering from acute pancreatitis, the risk of developing type III diabetes is 15%. The disease affects more often than males who drink excessively alcohol, fatty foods.

Causes of pancreatogenic diabetes

The disease develops in violation of endocrine and exocrine function of the pancreas. There are the following causes of damage to the islet apparatus of the gland:

- *Chronic inflammation of the pancreas*. Frequent exacerbations of pancreatitis increase the risk of developing diabetes. Chronic inflammation causes the gradual destruction and sclerosis of the islets of Langerhans.
- *Operations on the pancreas.* The incidence of postoperative diabetes varies from 10% to 50% depending on the volume of the operation. Most often the disease develops after total pancreatectomy, pancreatoduodenal resection, longitudinal pancreatojejunostomy, resection of the caudal part of the pancreas.
- *Other diseases of the pancreas.* Pancreatic cancer, pancreatic necrosis causes endocrine dysfunction with the formation of persistent hyperglycemia. There are risk factors that provoke pancreatogenic diabetes in patients with pancreatic dysfunction. These include: Alcohol abuse. Systematic alcohol consumption several times increases the risk of alcoholic pancreatitis with the formation of transient or persistent hyperglycemia. Eating disorders. Excessive consumption of foods rich in fats, easily digestible carbohydrates contributes to the development of obesity, hyperlipidemia and impaired glucose tolerance (prediabetes). Prolonged use of medications (corticosteroids) is often accompanied by hyperglycemia.

Of particular note are some studies showing that the development of endocrine insufficiency in this category of patients was directly related to the duration of diabetes, early onset and use of insulin, as well as low body mass index and high HbA1c.

Given these data, we can conclude that the main factors that provoke the development of exocrine insufficiency of the drug among patients with diabetes are:

- considerable duration of the disease;
- significant insulin requirements and poor glycemic control;
- severe diabetes.

For type II diabetes, severe exocrine insufficiency is primarily associated with the development of systemic lesions: autonomic neuropathy and microvascular damage, the development of fibrosis and atrophy of the pancreas, loss of islet-acinar-ductal axis and gastroenteropancreatic system. In contrast, severe exocrine dysfunction of the pancreas in type I diabetes occurs due to autoimmune damage to islet cells, which leads to a decrease in insulin levels and, consequently, a decrease in the trophic effect of the latter on exocrine cells. Given the combination of the above factors, the prevalence of exocrine insufficiency is higher among people with type I diabetes than those with type II diabetes (approximately 60 versus 30% of cases).

Symptoms of pancreatogenic diabetes mellitus

Pathology more often occurs in people of lean or normal physique with hyperexcitability of the nervous system. Pancreatic lesions are accompanied by dyspeptic symptoms (diarrhea, nausea, heartburn, flatulence). Painful sensations with exacerbation of inflammation of the gland are localized in the epigastric region and have different intensities. The formation of hyperglycemia in chronic pancreatitis occurs gradually, on average after 5-7 years. As the duration of the disease and the

frequency of exacerbations increase, the risk of developing diabetes increases. Diabetes can also debut in the manifestation of acute pancreatitis. Postoperative hyperglycemia is formed simultaneously and needs to be corrected by insulin. Pancreatogenic diabetes occurs in mild form with a moderate increase in blood glucose and frequent attacks of hypoglycemia. Patients are satisfactorily adapted to hyperglycemia up to 11 mmol / l. Further increase in blood glucose causes symptoms of diabetes (thirst, polyuria, dry skin). Pancreatogenic diabetes is well treated with diet and sugar-lowering drugs. The course of the disease is accompanied by frequent infectious and skin diseases.

Complication

Ketoacidosis and ketonuria are rare in patients with type III diabetes. Patients with pancreatogenic diabetes are characterized by frequent short attacks of hypoglycemia, accompanied by hunger, cold sweat, pale skin, excessive agitation, tremor. A further drop in blood glucose causes turbidity or loss of consciousness, the development of seizures and hypoglycemic coma. With the long-term course of pancreatogenic diabetes complications from other systems and organs (diabetic neuropathy, nephropathy, retinopathy, angiopathy), hypovitaminosis A, E, disorders of magnesium, copper and zinc metabolism.

Diagnosis

Diagnosis of pancreatogenic diabetes is difficult. This is due to the long absence of symptoms of diabetes, the difficulty of recognizing inflammatory diseases of the pancreas. With the development of the disease often ignore the symptoms of pancreatic lesions, prescribing only hypoglycemic therapy. Diagnosis of carbohydrate metabolism disorders is carried out in the following areas:

1. *Endocrinologist consultation.* Careful study of the history and association of diabetes with chronic pancreatitis, pancreatic surgery, alcoholism, metabolic disorders, and steroid use is important.

2. *Glycemic monitoring.* Involves determining the concentration of glucose on an empty stomach and 2 hours after a meal. With type III diabetes, fasting glucose levels will be within normal limits, and after a meal is elevated.

3. *Assessment of pancreatic function.* Carried out using biochemical analysis to determine the activity of diastase, amylase, trypsin and lipase in the blood. Indicative data of OAM: in pancreatogenic diabetes traces of glucose and acetone in the urine are usually absent.

4. *Instrumental methods of visualization.* Ultrasound of the abdominal cavity, MRI of the pancreas can assess the size, echogenicity, structure of the pancreas, the presence of additional formations and inclusions.

In endocrinology, the differential diagnosis of the disease is made with type 1 and type II diabetes. Type I diabetes is characterized by a sharp and aggressive onset of the disease at a young age and severe symptoms of hyperglycemia. The blood test detects antibodies to beta cells of the pancreas. Distinctive features of type II diabetes will be obesity, insulin resistance, the presence of C-peptide in the blood and the absence of hypoglycemic attacks. The development of both types of diabetes is not associated with inflammatory diseases of the pancreas, as well as surgery on the body.

Treatment of pancreatogenic diabetes mellitus

For best results, concomitant treatment of chronic pancreatitis and diabetes should be performed. You need to give up alcohol and smoking forever, adjust your diet and lifestyle. Complex therapy has the following directions:

1. Diet. Diet in pancreatogenic diabetes includes correction of protein deficiency, hypovitaminosis, and electrolyte disturbances. Patients are advised to limit the consumption of "fast" carbohydrates (pastries, bread, candy, cakes), fried, spicy and fatty foods. The main diet consists of proteins (lean meats and fish), complex carbohydrates (cereals), and vegetables. Food should be taken in small portions 5-6 times a day. It is recommended to exclude fresh apples, legumes, rich meat broths, sauces and mayonnaise.

2. Compensation for enzyme deficiency of software. Medicines that contain enzymes of amylase, protease, and lipase in different ratios are used. Drugs help to regulate the digestive process, eliminate protein and energy deficiency. The European Guidelines for the Management of Chronic Pancreatitis are one of the guidelines widely followed by clinicians in the management of patients with diabetes and concomitant exocrine insufficiency worldwide, according to which the appointment of substitution therapy requires compliance with nutritional status (level of evidence 1A). In particular, factors such as mixing with food and evacuation from the stomach, mixing with duodenal contents and their rapid release in the duodenum should be taken into account when prescribing enzyme preparations. In addition, oral enzyme preparations, which are traditionally used to correct exocrine insufficiency, should be divided between main meals and snacks (level of evidence 1A). Thus, the minimum dose of lipase should be from 40,000 to 50,000 IU per main dose and 50% of this dose - for a snack. It is recommended to take such drugs while eating. It should be emphasized that encapsulated enzyme preparations take the first place when choosing enzyme replacement therapy, and the use of 2.2-2.5 mm mini-tablets is just as effective (level of evidence 1B). It is also worth noting the proven positive effect of enzyme replacement therapy on glycemic compensation. In particular found that the correction of exocrine dysfunction in patients with diabetes reduced the level of glycated hemoglobin by approximately 17%. Importantly, oral enzyme replacement therapy in this category of patients provided better control of diabetes, which was manifested by a significant reduction in postprandial blood glucose and glycated hemoglobin, reduced abdominal pain, diarrhea, steatorrhea and flatulence, and flatulence overall quality of life.

3. Taking antidiabetic drugs. To normalize carbohydrate metabolism, a good result is given by the appointment of drugs based on sulfonylureas.

4. Postoperative replacement therapy. After surgery on the pancreas with full or partial resection of the tail of the gland is shown fractional administration of insulin not more than 30 IU per day. The recommended blood glucose level is not less than 4.5 mmol / l due to the risk of hypoglycemia. At stabilization of glycemia

it is necessary to pass to appointment of oral sugar-lowering drugs. Autotransplantation of islet cells is carried out in specialized endocrinological medical centers. After successful transplantation, patients undergo pancreatotomy or resection of the pancreas.

Prognosis and prevention

In the complex treatment of pancreatic lesions and correction of hyperglycemia, the prognosis is positive. In most cases, it is possible to achieve a satisfactory condition of the patient and normal blood sugar levels. In severe cancer, radical surgery on the gland, the prognosis will depend on the intervention and rehabilitation period. The course of the disease is complicated by obesity, alcoholism, abuse of fatty, sweet and spicy foods. To prevent pancreatogenic diabetes mellitus should lead a healthy lifestyle, abstain from alcohol, in the presence of pancreatitis in a timely manner to be examined by a gastroenterologist.

Conclusion. Therefore, the treatment of pancreatogenic diabetes is not very difficult if the patient responded in a timely manner to the development of the disease and sought medical help. After doctors are able to normalize carbohydrate metabolism and pancreatic function, therapy is used directly to stabilize blood sugar levels. Only a doctor decides which drugs will be used for this purpose.

REFERENCES:

1. Babinets, L.S. (2015). Aktualni aspekty formuvannia khronichnoho biliarnoho pankreatytu [Actual aspects of formation of chronic biliary pancreatitis]. Vestnik kluba pankreatologov – Bulletin of the Club of Pancreatologists, 4 (32), 46-50 [in Ukrainian].

2. Babinets, L.S. (2017). Koreliatsiia mizh trofolohichnym statusom ta osnovnymy kharakterystykamy zakhvoriuvannia u patsiientiv iz khronichnym pankreatytom biliarnoho genezu [Correlation between the trophological status and the main characteristics of the disease in patients with chronic bronchial pancreatitis]. Vestnik kluba pankreatologov –Bulletin of the Club of Pancreatologists, 1 (34), 38-40 [in Ukrainian].

3. Hontsariuk, D.O., & Khrystych, T.N. (2017). Rol mikrobioty u perebihu ta rozvytku metabolichnykh protsesiv pry khronichnomu pankreatyti [The role of microbiota in the course and development of metabolic processes in chronic pancreatitis]. Vestnik kluba pankreatologov – Bulletin of the Club of Pancreatologists, 4 (37), 22-26 [in Ukrainian].

4. Khrystych, T.M. (2007). Rol pidshlunkovoi zalozy v rehuliatsii systemnoho metabolizmu ta adaptyvnykh funktsii [The role of pancreas in the regulation of systemic metabolism and adaptive functions]. Novosti meditsyny i farmatsii. Hastroenterolohiia – News of Medicine and Pharmacy.– Gastroenterology, 226,7-8 [in Ukrainian].

5. Khristich, T.N. (2007). Parazitarnyy genez khronicheskikh pankreatitov. Gastroenterologiya [Parasitic genesis of chronic pancreatitis. Gastroenterology]. Mizhvidomchyi zbirnyk – Interdepartmental Compilation, 39, 140-145 [in Ukrainian].

6. Rosendahi, J., & Chymotrypsin, C. (2008). (CTRC) variants that diminish activity or secretion are associated with chronic pancreatitis. Nat. Genet., 40 (1), 78-82.

7. Venneman, N.G., Renooij, W., & Rehfeld, J.F. (2005). Small gallstones preserved gall bledermotilite and fast crystallization are associated with pancreatitis Hepatology, 41, 738-746.