Diagnosing pancreatic steatosis in obese patients

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In recent decades, there has been a steady increase in the prevalence of obesity. In obesity, especially in its visceral form, ectopic deposition of adipose tissue is often observed in various organs and tissues: in the heart, muscles, kidneys, liver (non-alcoholic fatty liver disease), as well as in the pancreas. According to various studies, the frequency of pancreatic steatosis in the adult population in the general population can be from 16 to 35% [1, 2, 4, 5].

Etiology of pancreatic steatosis

Possible causes of triglyceride accumulation in the pancreas include age, obesity, visceral obesity, type 2 diabetes. Additional reasons include factors such as male gender, Caucasoid and Mongoloid race, hypertriglyceridemia, and signs of metabolic syndrome (MS).

For the diagnosis of pancreatic steatosis associated with the presence of obesity (non-alcoholic fatty pancreatic disease), it is necessary to exclude other causes of steatosis, for example, taking toxic substances, alcohol, drugs, and hereditary diseases.

Nomenclature, terminology, definition

Currently, there are no unified approaches to the terminology and criteria for diagnosing conditions characterized by an increased content of adipose tissue in the pancreas. The most widely cited nomenclature classification is made by M.M. Smits, E.J.M. van Geenen [12].

- Pancreatic steatosis (pancreatic lipomatosis, fatty pancreatic disease) — the accumulation of adipose tissue in the pancreas due to various reasons.

- Lipomatous pseudohypertrophy — an “extreme” variant of the accumulation of adipose tissue in the pancreas; total or local increase in pancreas; replacement of exocrine cells with adipocytes in the absence of an association with obesity.
- Fat replacement — adipocyte replacement of dead acinar cells (for example, with viral infections, hemochromatosis, obstruction of pancreatic ducts), an irreversible process.
- Fatty infiltration — adipocyte infiltration due to obesity.
- Non-alcoholic fatty pancreatic disease — the accumulation of fatty tissue in the pancreas associated with obesity or MS.
- Non-alcoholic pancreatic steatosis — the accumulation of adipose tissue in the pancreas associated with obesity or MS, without signs of inflammation.
- Non-alcoholic fat steatopancreatitis — pancreatitis, which developed against the background of the accumulation of adipose tissue in the pancreas associated with obesity or MS, in the absence of other etiological factors.

According to the authors of the presented nomenclature, it is necessary to develop a differentiated approach to the diagnosis of conditions accompanied by the accumulation of triglycerides in secretory (acinous) cells, P-cells or in intrapancreatic adipose tissue. The lack of criteria for such a diagnosis limits the use of the existing classification [9].

“Steatosis” is a universal term that reflects the accumulation of intracellular fat in the organ parenchyma, and this process is considered as potentially reversible. Along with it, the terms “lipomatosis”, “fatty pancreatic disease” are also used, in the English language literature — “fatty pancreas”; these terms can be regarded as synonyms [10]. It was found that with a decrease in body weight, a decrease in pancreatic steatosis is observed in the case of the use of troglitazone, a combination of telmisartan and sitagliptin and some other drugs [1, 7].

Diagnosis

With pancreatic steatosis, as a rule, there are no clinical manifestations, it is characterized by an asymptomatic course. Diagnosis is by imaging methods. For the diagnosis of pancreatic steatosis, general clinical, laboratory and instrumental examination methods can be used.

Complaints are uncharacteristic and non-specific. Pancreatalgia with pancreatic steatosis — abdominal pain of low or moderate intensity, in the epigastric region or in the left hypochondrium, aggravated after eating or occurring 30–40 minutes after eating, sometimes radiating to the back. Dyspeptic symptoms in the form of vomiting, nausea, bloating are found in half of patients. Sometimes there is an increase in stool more than 2 times a day, its liquid consistency.

Exocrine insufficiency (the content of pancreatic elastase in the stool is less than 200 μg/g) is uncharacteristic for non-alcoholic fatty pancreatic disease. Steatorrhea is rare, it occurs in patients with severe pancreatic steatosis or steatopancreatitis with exocrine insufficiency [10, 13].

A feature of pancreatic damage in obesity is the absence of changes in biochemical parameters or their slight changes. RV steatosis is characterized by an increase in fasting glucose, triglycerides, total cholesterol, low density lipoproteins, alanine aminotransferase, γ-glutamyl transpeptidase and leptin, as well as a decrease in high density lipoproteins and serum lipase; the presence of insulin resistance, signs of MS and an increased level of both systolic and diastolic blood pressure. Amylase levels are usually low, most often due to exacerbation of chronic pancreatitis. At the same time, with chronic pancreatitis, higher values of inflammation indicators are observed, such as leukocyte count, erythrocyte sedimentation rate and amylase activity.

One of the methods of radiation diagnosis of pancreatic steatosis is ultrasound (ultrasound). The sensitivity of diagnosis of pancreatic steatosis using ultrasound varies from 37 to 94%, specificity — from 48 to 100% [11]. Several approaches have been described to determine the degree of pancreatic steatosis.

According to the classification of J.S. Lee et a1. and A. Smereczynski, K. Kolaczyk, there are three degrees of severity of pancreatic steatosis [8, 11]:

- I degree — echogenicity of the pancreas is equal to the echogenicity of adipose tissue in the region of the superior mesenteric artery. The dimensions of the pancreas are not increased, the echogenicity is evenly increased, the contour is smooth, the splenic vein, superior mesenteric artery and pancreatic duct are well visualized;
- II degree — increased echogenicity against the background of a weakened signal in the distal, dorsal pancreas (decreased conductivity of the acoustic signal, attenuation of the ultrasound signal behind the posterior surface of the pancreas), fuzzy edges of the splenic vein and pancreatic duct with a practically non-visualized region of the superior mesenteric artery;
- III degree — a decrease in the ultrasound conductivity of the pancreas, wave-like (convoluted), fuzzy contours, the splenic vein, the region of the superior mesenteric artery and the pancreatic duct are not visualized.

The hyperdiagnosis of pancreatic steatosis with ultrasound is most likely due to the fact that the density of the pancreas is compared with the density of the parenchyma of the kidney, liver and/or spleen, and not with the density of retroperitoneal fiber. Our studies and comparison of the results of computed tomography (CT) and ultrasound of the pancreas allowed us to offer the following method for diagnosing the degree of pancreatic steatosis [3].

Ultrasound diagnostic criteria for pancreatic steatosis:

- norm: pancreatic density corresponds to the density of the cortical layer of the kidney;
- mild: pancreatic density is higher than the echogenicity density of the cortical layer of the kidney, but lower than the density of retroperitoneal fiber;
- moderate: pancreatic density corresponds to the density of retroperitoneal fiber;
- severe: pancreatic density is higher than retroperitoneal fiber density.

Computed tomography allows you to determine the structure, quantify pancreatic tissue density (in Hounsfield units), so that you can track the dynamics of changes, as well as compare the results of various studies, develop quantitative criteria for the diagnosis of steatosis. Signs of pancreatic steatosis according to direct densitometric analysis for CT include a decrease in attenuation coefficients in Hounsfield units (pancreatic density less than 30 Hounsfield units, lower than spleen density, with severe steatosis — comparable to the density of nearby retroperitoneal tissue or below it), as well as a uniform or heterogeneous change pancreatic structures: lobular structure of the gland with pronounced fatty layers.

To assess the condition of the pancreas, magnetic resonance imaging (MRI), proton magnetic resonance spectroscopy are used. The advantages of various MRI modes are the high sensitivity and specificity of the diagnosis of pancreatic steatosis, non-invasive nature, the absence of ionizing radiation. The results of MRI are comparable with the results of CT of the pancreas. Modern MRI techniques can significantly increase the sensitivity and specificity of the diagnosis of pancreatic steatosis. According to MRI, pancreatic steatosis is diagnosed with a fatty tissue content in the pancreas of more than 10.4%.

Conclusion

For the early diagnosis of pancreatic steatosis, it is recommended to examine individuals with obesity, type 2 diabetes mellitus and signs of MS. The examination plan must include a biochemical blood test with the determination of the lipid profile, alanine aminotransferase, γ-glutamyl transpeptidase, lipase, glucose and abdominal ultrasound.

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Frequency of pancreatic steatosis in adults of general population is up to 35%. Causes of triglyceride accumulation in the pancreas include aging, obesity, type 2 diabetes, metabolic syndrome, hypertriglyceridemia (genetically determined or secondary one). Nomenclature by M.M. Smits is used for diagnosing, which sets out pancreatic steatosis, lipomatous pseudohypertrophy of the pancreas, fatty replacement of the pancreas, fatty infiltration of the pancreas, non-alcoholic fatty pancreatic disease, non-alcoholic pancreatic steatosis, non-alcoholic steatopancreatitis.

Pancreatic steatosis usually does not have any clinical manifestations, as it is characterized by an asymptomatic course. It is diagnosed on the basis of results of imaging methods (ultrasound, computed or magnetic resonance imaging). According to the ultrasound, there are 3 degrees of pancreatic steatosis:

- Degree I — pancreatic echogenicity is equal to the echogenicity of adipose tissue in area of superior mesenteric artery. Pancreas is not enlarged, echogenicity is uniformly increased, contour is smooth, splenic vein, superior mesenteric artery and pancreatic duct are well-visualized;
- Degree II — increased echogenicity on the background of weak signal in the remote, dorsal part of the pancreas (reduced acoustic signal conductivity, attenuation of the ultrasonic signal behind the posterior surface of the pancreas), indistinct edges of splenic vein and pancreatic duct with almost non-visualized area of superior mesenteric artery;

- Degree III — reduction of ultrasound conductivity of the pancreas, undulating (convoluted), indistinct contours, splenic vein, area of superior mesenteric artery and pancreatic duct are not visualized.

Main feature of the pancreas lesion in obesity is the absence of changes in biochemical indices or their minor changes. Exocrine and endocrine pancreatic insufficiency, hyperlipidemia may develop.