GALAXY INTERNATIONAL INTERDISCIPLINARY RESEARCH JOURNAL (GIIRJ)
ISSN (E): 2347-6915
Vol. 10, Issue 9, Sep. (2022)

PANCREATIC STEATOSIS IS A NEW THERAPEUTIC PROBLEM IN GASTROENTEROLOGY
(Literature Review)

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ANNOTATION
Currently, pancreatic steatosis is a common accidental finding during ultrasound examination of the abdominal cavity for other reasons and represents a new problem in gastroenterology and pancreatology. In this review article we have made an attempt to give a general idea of pancreatic steatosis, etiopathogenesis, diagnostic methods, as it is of particular importance due to its close connection with type 2 diabetes mellitus, non-alcoholic fatty liver disease and cardiovascular diseases.

Keywords: fatty pancreatic disease, obesity, pancreatic steatosis.

INTRODUCTION
Fatty infiltration in the pancreas (called pancreatic steatosis or pancreatic steatosis. This pathology describes the disease from infiltration of fat in the pancreas to inflammation of the pancreas and the development of pancreatic fibrosis and carcinoma [4.39]. Just as obesity and metabolic syndrome are global problems, pancreatic steatosis, especially in the form of non-alcoholic fatty disease, is a serious problem for pancreatologists, gastroenterologists, diabetologists and nutritionists.

If liver steatosis is currently a more studied pathology, then pancreatic steatosis still remains a little-studied problem, so it is an accidental finding during diagnostic manipulations due to the lack of pronounced symptoms [48,54] however, with the advent of the obesity epidemic and the spread of metabolic syndrome among all age groups of the population, and the expected increased non-alcoholic fatty disease pancreas, he began to receive more and more attention.

For the first time, the phenomenon of fat accumulation in the pancreas has been known for more than 90 years. So, in 1926, J.H. schaefer discovered the relationship between the weight of the pancreas and the whole body. The next research in this area was done by R. Ogilvie in 1933. He described the presence of fat in the pancreas in 17% of obese patients, while in slim patients fat was present in only 7%. The author first used the term "pancreatic lipomatosis" to denote fatty infiltration of the pancreas.

T.S. olsen in 1978 Post-mortem examination of 394 patients revealed that the increase in the amount of pancreatic fat is directly related to age. Similarly, b.h. stamm et al. In 1984, an increase in pancreatic fat associated with older age was proved. They also found a significant association between pancreatic steatosis, when the fat content in the pancreas is 25% or more, and the risk of developing type 2 diabetes and atherosclerosis. In 2010, E.J. van reenen et al.

[58] hypothesized that obesity and its association with insulin resistance play an important role in the infiltration of the pancreas by adipocytes, leading to steatosis of the gland. Insulin resistance also leads to peripheral lipolysis and, subsequently, to the influx of fatty acids into the liver parenchyma and the occurrence of non-alcoholic fatty liver disease non-alcoholic fatty pancreatic disease.

Since the initial description, the deposition of fat in the pancreas has been studied by many researchers in various studies, and many different definitions and terms have been used, such as: fat replacement, fat infiltration, pancreatic lipomatosis, lipomatous pseudohypertrophy, non-alcoholic fatty pancreatic disease, non-alcoholic fatty pancreatic disease and non-alcoholic fatty steatopancreatitis [8,44,55]. R pezzilli and L.calculli suggested that the most appropriate name for the accumulation of fat in the pancreas is the term "pancreatic steatosis" [40].

Recently, the most widely used term in the literature to describe the accumulation of fat in the pancreas associated with obesity, without significant alcohol consumption, has become non-alcoholic fatty pancreatic disease [1,44]. Moreover, this term also describes the possibility that fat accumulation is a reversible process.

According to studies, fat accumulation can be uniform or uneven [6,12]. Four different types of uneven pancreatic lipomatosis are described: (1) type 1a (35% of cases): replacement of the head with preservation of the hook-shaped process and the peribiliary region; (2) type 1b (35%): replacement of the head, neck and body with preservation of the hook-shaped process and the peribiliary region; (3) type 2a (12%): replacement of the head, including the hook-shaped process, and preservation of the peribiliary region; and (4) type 2b (18%): complete replacement of the pancreas with preservation of the peribiliary region.

Data on the prevalence of non-alcoholic fatty pancreatic disease are few due to the lack of standardized screening tests. Epidemiological studies conducted in the period from 2014 to 2016 show that the prevalence of non-alcoholic fatty pancreatic disease ranges from 16 to 35% according to abdominal imaging data [11,33,36,49,59,65]. This is mainly based on the results obtained in the Asian population. Only one epidemiological study published in 2016 [41], concerned the pediatric population and estimates the prevalence of pancreatic steatosis at 10%. However, it was conducted exclusively on hospitalized children, and not on the general pediatric population.

Often, non-alcoholic fatty pancreatic disease occurs in combination with non-alcoholic fatty pancreatic disease [2,59,65]. Approximately 50-80% of patients with non-alcoholic steatohepatitis also have fat deposition in the pancreas, which is confirmed by imaging studies of the abdominal cavity [9,57].

P.s. sepe at al. It is claimed that in metabolic syndrome, non-alcoholic fatty pancreatic disease occurs in 100% of cases [49]. The authors found that patients aged 41-70 years were more predisposed to fat deposition in the pancreas, and the presence of any component of metabolic syndrome was the reason for an increase in prevalence by 37%.

O.N. Korneeva et al. (2011) noted this combination in 67% of patients.

Thus, the results of studies have shown that the presence of non-alcoholic fatty pancreatic disease may be a risk factor for the development of non-alcoholic fatty pancreatic disease. Therefore, patients suffering from pancreatosteatosis should be examined for both non-alcoholic fatty pancreatic disease and non-alcoholic fatty pancreatic disease. However, additional

epidemiological studies are needed to better understand the mechanisms of occurrence of non-alcoholic fatty pancreatic disease.

Pathophysiology and risk factors non-alcoholic fatty pancreatic disease

The pathophysiology of non-alcoholic fatty pancreatic disease has not been fully studied, mainly two mechanisms leading to the accumulation of fat in the pancreas are described. The first is the death of acinar cells and their replacement by adipocytes, as well as fat infiltration, which is characterized by the accumulation of fat in the pancreas and association with metabolic syndrome and/or obesity, which determines non–alcoholic fatty pancreatic disease [5,30,53]. In this case, the condition is called "fat substitution".

The second is the accumulation of fat, called "fat infiltration". Obesity is the main contributing factor to this disease, which leads to non-alcoholic fatty pancreatic disease [37,38,49]. Adipose tissue is considered an endocrine organ because it sends signals to other organs. During weight gain, the reserves of adipose tissue are exceeded, causing the redistribution of fat in non-fat tissues such as the liver, skeletal muscles and pancreas. Infiltration of pancreatic fat initially causes hypertrophy and hyperplasia of pancreatic cells, which leads to such clinical conditions as insulin resistance, beta-cell dysfunction and type 2 diabetes mellitus [24,42,48,57].

Summarizing the information from literary sources, the most well-known factors provoking steatopancreatitis [22,26,45,50,58] include:

- Congenital diseases (schwachman-diamond syndrome, johanson-blizzard syndrome, cystic fibrosis, heterozygous mutation of carboxyester lipase);
- Alcohol abuse;
- Infections (viral infection with reovirus);
- Hemochromatosis;
- Medications (rosiglitazone, corticosteroids, octreotide, gemcitabine, etc.);
- Malnutrition;
- Non-Alcoholic fatty pancreatic disease and chronic hepatitis b;
- Necrotizing pancreatitis, recurrent acute pancreatitis, hereditary chronic pancreatitis;
- Metabolic syndrome.

Experimental studies by R. Carter et al. It is shown that maternal obesity and a postpartum diet that promotes obesity lead to the occurrence of non-alcoholic fatty pancreatic disease. Inducers are endoplasmic reticulum, imbalance and changes in circadian metabolic processes [17].

In a clinical study by M. Bojkova et al. It has been proved that metabolic syndrome and its components (obesity, hypertension, hypertriglyceridemia, changes in HDL cholesterol and cd2) are significant factors in the development of non-alcoholic fatty pancreatic disease [15].

M. Heni et al. It is claimed that the correlation between the detection of non-alcoholic fatty pancreatic disease and non-alcoholic fatty pancreatic disease is not absolutely reliable [26]. Although liver fat is localized mainly intracellularly, pancreatic fat is associated with the presence of adipocytes infiltrating its parenchyma. Therefore, for example, during bariatric surgery, the mentioned liver fat and pancreatic fat change and disappear completely independently of each other [23]. However, it cannot be excluded that non-alcoholic fatty

pancreatic disease and non-alcoholic fatty pancreatic disease affect each other with respect to the onset and progression of the disease.

A study by C.D. corte et al. [20] showed that non-alcoholic fatty pancreatic disease is a common disease in obese children. At the same time, pancreatic fat should not be considered as an inert accumulation of fat, but as an additional factor that can affect glucose metabolism and the severity of liver disease, increasing the risk of developing metabolic syndrome.

Recent studies show that non-alcoholic fatty disease of the pancreas is the initial and even earlier manifestation of the metabolic syndrome [1,10,18,46,60]. Perhaps it is the exo- and endocrine imbalance in the pancreas that is the trigger for metabolic disorders, which then develop into the metabolic syndrome [3,19,49,51].

Y. Bi et al. We analyzed 13 studies involving 49,329 subjects [13]. The data obtained indicate that non-alcoholic fatty pancreatic disease is largely associated with an increased risk of developing the metabolic syndrome and its components, which correlates with the findings of other authors [14,21]. However, well-designed prospective cohort studies between pancreatic steatosis and metabolic syndrome are needed to elucidate a future causal relationship.

Diagnosis of Pancreatic Steatosis

To date, there is no clear algorithm regarding the methods of diagnosing the presence of fat in the pancreas. Opinions unanimously agree only that the optimal method should include simultaneous determination of the presence of fat deposits in the pancreas and its amount in a non-invasive way, since pancreatic steatosis is considered a possible risk factor for pancreatic cancer.

Despite the fact that biopsy is the gold standard in the assessment of steatosis, due to the location of the pancreas, the risk of complications and systematic sampling error, radiological methods still represent the best non-invasive alternatives [7,16,53,55]. Ultrasound, endoscopic ultrasound, computed tomography, magnetic resonance imaging and magnetic resonance spectroscopy are considered to be the main methods that play an important role in the diagnosis of pancreatic steatosis.

Transabdominal ultrasound is a non-invasive, inexpensive and widely available method, but at the same time has its drawbacks. Pancreatic steatosis is defined as an image of increased echogenicity in the pancreatic parenchyma compared to renal echogenicity or liver echogenicity, where the possible presence of liver steatosis is a limitation of the assessment [7,19]. Therefore, it is recommended to first compare the echogenicity of the liver and kidneys, and then, using the same acoustic window, compare the echogenicity of the pancreas with the echogenicity of the kidneys or liver. However, it is known that the normal echogenicity of the pancreas is equal to the echogenicity of the liver or slightly exceeds it [6,37]. Thus, the diagnostic criteria are vague, and complications may arise when diagnosing fatty pancreas, if at the same time there is a non-alcoholic fatty pancreatic disease [53,55].

Another misleading factor in the diagnosis may be pancreatic fibrosis, since it shows a hyperechoic appearance similar to the accumulation of fat in the pancreatic tissue on ultrasound.

In addition, the retroperitoneal location of the pancreas makes it difficult to visualize it, especially in obese patients. Therefore, quantitative analysis or stratification of echogenicity in the pancreas is necessary [28].

Endoscopic ultrasound examination provides very good visualization and evaluation of the examined gland, since the proximity of the ultrasound sensor to the pancreas allows you to obtain an image of pancreatic tissue with a higher resolution compared to computed tomography and magnetic resonance imaging.

In their study, c.r.a. lesman et al. [34] concluded that endoscopic ultrasound can be considered as a screening tool for early detection of pancreatic cancer in pancreatic steatosis.

Various studies have shown a relationship between increased echogenicity of the pancreas and the presence of fatty liver dystrophy, obesity with a body mass index (bmi>30.0), as well as usually with hypertension and even age over 60 years [19,49]. However, the increased echogenicity of the pancreatic parenchyma is not always a reflection of the increased fat content in the pancreas, but may be caused by the presence of pancreatic fibrosis, which is considered a limitation of the method [56]. In addition, endoscopic ultrasound is an invasive procedure and has some disadvantages, such as a high risk of complications and the need for sedation. The fact that both transabdominal ultrasound and endoscopic ultrasound are operation-dependent methods can lead to various diagnostic errors. All these factors make the reliability of studies using methods based on ultrasound controversial.

Ultrasound elastography allows you to assess the stiffness of the organ. In pancreatology, it has proved useful in the diagnosis of diseases of the pancreas, that is, elastography using endoscopic ultrasound allows predicting external secretory insufficiency of the pancreas in chronic pancreatitis [43]. However, with this method, there are also some limitations in the diagnosis of steatopancreatitis, especially with the retroperitoneal location of the pancreas and its small size, which can reduce the accuracy of diagnosis.

Computed tomography is considered one of the most effective methods of detecting a typical fatty pancreas [18,31,64]. It is hypodensive in Hounsfield units compared to the spleen [18,29,32,47]. The method also depends on the operator conducting it, and the assessment of its diagnostic benefit is heterogeneous.

Nevertheless, it is believed that computed tomography using the assessment of the fat/parenchyma ratio is a more reliable method compared to histological diagnosis, as a practical and easily accessible method that provides qualitative and quantitative assessment in a short time without the use of contrast agents.

Magnetic resonance imaging is currently the most preferred method. The advantage of magnetic resonance imaging is its non-invasiveness, safety and high sensitivity.

Various tests have shown that its accuracy in determining the presence of fat is comparable to histological examination, and thus it is the preferred method for diagnosing pancreatic lipomatosis [23,25]. However, in different studies based on magnetic resonance imaging in patients with non-alcoholic fatty pancreatic disease and non-alcoholic fatty pancreatic disease, different results were obtained. In some studies based on magnetic resonance imaging, there was a significant positive correlation between hepatosteatosis and pancreatosteatosis [26], whereas in other studies there was no correlation [25]. Because of these different results in studies, the power and adequacy of magnetic resonance imaging to identify the link between

non-alcoholic fatty pancreatic disease and non-alcoholic fatty pancreatic disease are controversial. Another disadvantage that makes magnetic resonance imaging images not quite accurate is the visceral fat tissue around the pancreas, its small size and irregular parenchymal structure.

Proton magnetic resonance spectroscopy or magnetic resonance imaging of the proton density of the fat fraction (magnetic resonance imaging "fat-water"). This method provides a highly accurate quantitative assessment of the amount of fat present in the pancreatic parenchyma [27,63]. Currently, this method is also indicated for the quantitative determination of fat in adjacent parenchymal organs, and not only in the pancreas [61].

Measurement of the proportion of fat with proton density (PDFF) is a method based on magnetic resonance imaging to quantify the steatosis of the area of interest [29,60] although it is usually used to quantify liver steatosis, it can also be used to determine pancreatic steatosis. Diagnostic data in this case can vary significantly depending on the location and due to the heterogeneous nature of the accumulation of fat in the pancreas.

Modern magnetic resonance imaging technology is able not only to ascertain the size of the organ and homogeneous changes in the structure in steatosis, but also to assess the content of lipids, and with proton magnetic resonance spectroscopy, to quantify the content of triglycerides [6,35,62]. I. Lingway, v. Esse, j.l. Legendre et al. Magnetic resonance spectroscopy, a non-invasive and quantitative method for assessing triglyceride accumulation at ectopic sites, has been demonstrated to be an accurate and reproducible clinical research tool for studying pancreatic steatosis in humans [35]. At the same time, there are very few studies on this issue and there are no generally accepted criteria for the diagnosis of pancreatic steatosis.

Treatment of Pancreatic Steatosis

Based on the analysis of the literature data, there is no treatment of pancreatic steatosis as such. The main task of therapy is considered to be the reduction of excess body weight. Thus, the data of the study a.p. rossi, f. Fantin, g.a. zamboni et al. It is shown that even moderate weight loss can reduce ectopic fat deposition along with a decrease in the lipid content in the pancreas, as well as significantly improve insulin resistance, estimated by the homa index [46]. Improvement of metabolic parameters after moderate weight loss is also confirmed in other studies [52].

If we assume that pancreatic steatosis develops due to fat accumulation and infiltration in the pancreas, lifestyle may be one of the determining factors in the development or regression of this disease. Therefore, with pancreatic steatosis, reduction measures are recommended, including rationalization of lifestyle (physical activity) and nutrition. It was found that a decrease in body weight even by 8.9% leads to a statistically significant decrease in the fat content in the pancreas [45]. Thus, reducing the fat content in the pancreas due to lifestyle changes can improve the health and clinical profile of patients with pancreatic steatosis.

However, there is still no consensus in the literature on specific physical training for this disease.

S.I. pimanov reports a decrease in pancreatic steatosis and body weight in the case of troglitazone [6].

In other studies, the therapeutic tactics of managing patients with pancreatic steatosis within the framework of metabolic syndrome is aimed at correcting lipid metabolism disorders, not only in the pancreas, but also, first of all, in the liver [4]. Recommendations in this situation contain the appointment of drugs containing essential phospholipids and lowering cholesterol levels. Patients with pancreatic steatosis with pancreatic insufficiency are prescribed therapy with enzyme preparations.

In the literature available to us, we have not found any data on therapeutic measures specific to pancreatic steatosis, and the available information on therapeutic tactics has been developed for the treatment of metabolic syndrome and its components, as well as liver steatosis.

Thus, based on the analysis of literature data, it can be concluded that pancreatic steatosis is a relatively new disease, as it has attracted the attention of clinicians recently due to the significant occurrence of fatty infiltration of the pancreas, especially in obese patients, metabolic syndrome and its components. The clinical consequences of non-alcoholic fatty pancreatic disease remain largely unknown, despite clinical associations, there are no algorithms for early diagnosis and treatment, since pancreatic steatosis is detected in most cases when examining patients for other diseases, therefore this fact determines the relevance of the problem and requires further scientific research.

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