Non-Alcoholic Fatty Pancreas Disease: A Clinical Entity We Should not Ignore any more

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Abstract

Obesity and diabetes are major clinical health problems with their prevalence growing every day, being responsible for high mortality rates and cardiovascular implications. Excessive fat accumulation in multiple organs causes malfunctions. Non-alcoholic fatty liver disease (NAFLD), as a reflection of ectopic fat accumulation at the liver, is easily diagnosed and well-studied. On the other hand, pancreatic fat accumulation or Non-alcoholic fatty liver disease (NAFPD) is until now an unknown and probably underestimated entity. During recent years, contemporary radiological techniques, such as endoscopic ultrasound, computed tomography (CT) and magnetic resonance imaging (MRI), made diagnosis of NAFPD easier to access. It mostly coexists with NAFLD, but it seems that it has its own clinical significance. It correlates with impaired glucose metabolism and cardiometabolic risk and has an interesting relation with other pancreatic diseases, such as acute and chronic pancreatitis and pancreatic cancer. NAFPD is a medical issue that may have a more interesting role in the metabolic syndrome. Still, there is a long scientific way to have in order to clarify its pathogenesis, clinical course and exact clinical impact. Nevertheless, we should not ignore it anymore as it may independently contributes in metabolic dysfunction.

Keywords: Non-Alcoholic Fatty Pancreas Disease; Non-Alcoholic Fatty Liver Disease; Pancreatic Fibrosis; Obesity; Type 2 Diabetes Mellitus

Abbreviations

NAFLD: Non-alcoholic fatty liver disease; NAFPD: Non-alcoholic Fatty Pancreas disease; DM: Diabetes Mellitus; BMI: Body mass index; CT: Computed tomography; MRI: Magnetic resonance imaging; U/S: Ultrasound; IGT Impaired glucose tolerance; IFG: Impaired fasting glucose

Introduction

Obesity and diabetes mellitus (DM) are a growing pandemic issue worldwide with their prevalence reaching very high. Obesity, defined as Body Mass Index (BMI) ≥ 30 kg/m² is a major health problem, not only because of its cardiometabolic implications [1], Recent studies have shown a high prevalence among children and adults [2].

Type 2 DM is the most significant clinical implication of obesity and is about to affect 645 million people by 2045 and will rise the overall global cost to 802 billion USD [3]. Both diseases are responsible for mortality, cardiovascular complications and high percentage of hospital admissions as this pandemic grows, excessive fat accumulates in tissues such as liver, pancreas, skeletal muscles and heart [4].

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Non-alcoholic fatty liver disease (NAFLD) is considered the hepatic manifestation of ectopic fat accumulation caused by abdominal obesity and insulin resistance and is getting a serious, clinical problem, resulting through gradual progression to steatohepatitis, cirrhosis and in some cases in liver failure [5]. In addition, it should be considered a cardiovascular risk factor [6]. On the other hand, Non-Alcoholic Fatty Pancreas disease (NAFPD), is rather an unknown and probably underestimated entity, even if the term pancreatic steatosis was described, nearly, 90 years ago [7]. Nowadays, it is diagnosed incidentally, most often after a Computed tomography (CT) or Magnetic resonance imaging (MRI) is ordered, mainly for other clinical purposes. It reflects the abnormal pancreatic fat accumulation and fat deposition in the pancreatic cells and associates with an obese and or dysmetabolic phenotype [8].

While NAFLD has been well investigated, the impact of NAFPD on clinical practice is not very well established. The aim of this review is to collect all the information regarding the natural history, diagnosis and implications of NAFPD

**Definition-prevalence-pathophysiology**

Many terms could describe the accumulation of fat in the pancreas, such as pancreatic lipomatosis, fatty pancreas, fatty infiltration, non-alcoholic fatty pancreatic disease and pancreatic steatosis [9]. Maybe the term pancreatic steatosis describes the primary step of fat accumulation to pancreas, indicating the possibility that fat accumulation is a reversible process, while the term NAFPD refers to a new clinical entity where there is evidence of significant pancreatic fatty infiltration or pancreatic islet cell steatosis, without any significant alcohol consumption and possibly is related to cardiometabolic and other complications with organs and systems we are going to describe in this review [9].

The precise pathophysiological pathway of NAFPD remains still unclear. Obesity, mainly as augmentation of visceral adipose tissue, is the primal factor that leads to ectopic fat accumulation in pancreatic tissue, along with hepatic fat accumulation, through death of acinar cells and replacement by adipocytes [10].

Fat accumulation in the liver or pancreas might result from disturbance in the balance between supply, formation, consumption and hepatic oxidation or disposal of triglycerides [11]. The potential sources of lipids in the liver or pancreas come from circulating free fatty acids (FFAs), lipogenesis and dietary fat intakes. This lipotoxicity leads to cellular damage and promotes further ectopic hepatopancreatic fat accumulation [12].

Not only pancreatic and hepatic steatosis increase, through visceral adipose tissue and increased fibrogenic markers such as TGF-β1 and proinflammatory cytokines, but it also creates a more atherogenic profile with higher body weight and hypertension [13]. In humans, pancreatic steatosis is closely associated with higher BMI, insulin resistance and hepatic adiposity. It has been stated that that pancreatic fat may lead to a loss of β-cell mass and function, which possibly contributes to the development of diabetes. The coexistence between patients with NAFLD and NAFPD, has also driven interest in lipid accumulation in the pancreas as a cause of impaired beta-cell insulin secretion [14].

There is little evidence about the prevalence of NAFPD in the general population, mainly because of the lack of standard diagnostic techniques. Most studies come from Asian populations. Among 901 Indonesian adults who had routine upper abdominal ultrasound the prevalence was 35% [15], while among 8097 Taiwan individuals the prevalence of NAFPD was 16%, diagnosed again with abdominal U/S [16]. When MRI was the choice of detecting NAFPD, in Taiwan population, the prevalence reached again 16% [17]. Furthermore, data on pancreatic fat percentage from nine studies (1209 healthy individuals who underwent magnetic resonance imaging), reached 4.48% and associated with significantly increased risk of metabolic syndrome [18]. In an American study, among 230 individuals examined through endoscopic U/S, 27.8% were found to have fatty pancreas which in turn had a strong association the metabolic syndrome [19]. NAFPD is not only met in adults but in children, too. In a retrospective chart review study of 232 patients 2 to 18 years old (admitted or hospitalized) who underwent abdominal CT, pancreatic steatosis was identified in 10% of the study population and was associated with obesity and NAFLD [20].

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As it is already mentioned obesity is the major pathogenetic factor associated with pancreatic steatosis [9]. There are differences between sexes. According to a study with obese individuals, men had a higher pancreatic fat content (estimated by MRI), compared with women of comparable BMI. It also seems that men have the highest prevalence of NAFPD at the age of 40 - 49 years, whereas the prevalence of NAFPD in women was very low in early life, but rapidly increased after menopause [17]. Another significant factor is age, as pancreatic fat is increasing as we grow [21]. This linear relation keeps until the age of 50 years, where it seems that the pancreatic steatosis increases irrespective of the volume of pancreatic mass, probably due to the progressive fatty infiltration of the pancreas [22]. Obesity and age are not the only factors related with NFAPD. Hemochromatosis may result through iron overload, in pancreatic steatosis [23]. There are also genetic syndromes related with pancreatic fat replacement, such as cystic fibrosis [24] and Shwachman-Diamond syndrome [25]. Pancreatic fat replacement is related also with viral infections. It is not very clear if hepatic diseases, in general, induce pancreatic fat accumulation, since there are only some case reports, regarding chronic hepatitis B and liver cirrhosis, that support this issue [26].

**Diagnosis**

Undoubtedly, the most accurate diagnosing method is biopsy of the pancreatic tissue. As it is well understood, it has a great limitation due to the retroperitoneal location and difficult access of the pancreas, in contrast to the liver tissue. It would be very interesting to diagnose through biopsy early stages of NAFPD, where it seems that intracellular accumulation in pancreatic acinar and islet cells may occur prior to the infiltration by adipocytes [16]. Besides the only existing score scaling pancreatic fat accumulation was counted on the percentage of infiltration from adipocytes after autopsies [27].

Since the direct access to the pancreatic tissue is almost prohibitive in living individuals, we use imaging techniques for the diagnosis of NAFPD.

First, of course, comes the conventional transabdominal ultrasound (U/S). It has several limitations, with the greater to be the lack of suspicion from the radiologist’s point of view. NAFLD is much more easier to diagnose (comparing liver echogenicity with the kidney’s) and more recognizable as an entity. Besides transabdominal U/S was never the choice examination for pancreatic diseases, in general, mostly because of overlying stomach/small intestine gas [12]. That’s why endoscopic U/S (EUS) is a much better choice and gives even a way of classification pancreatic steatosis (comparing its echogenicity with spleen), associating it with metabolic syndrome [28]. EUS provides more detailed images of the pancreas but has the major disadvantage of being invasive.

Transient elastography is not a technique that is used for assessing pancreatic fat, as it is for liver tissue. Studies are conflicting, with one study finding reduced pancreatic stiffness in a group of patients with pancreatic insufficiency in cystic fibrosis [29] and another finding no difference between normal pancreatic tissue and NAFPD [30]. Nonetheless, the deep intra-abdominal location of the pancreas precludes the use of transient elastography, but acoustic radiation force impulse (ARFI) can be used to deliver localized stress under standard B-mode ultrasound imaging guidance [31].

CT is an easy imaging technique that can detect pancreatic fat deposition using Hounsfield units. Pancreatic fat can be quantified by using CT by measuring the difference between pancreatic and splenic attenuation and the areas of normal parenchyma between the fat deposits can portray enhancement [32]. Maybe the main limitation is the high radiation one could absorb, especially if the examination takes place only for diagnosing NAFPD. An additional limitation is the intravenous contrast administration, especially in diabetic-nephropathy population, not to mention that CT is unable to distinguish between fat in adipocytes and intracellular fat in parenchymal cells [31]. Furthermore, there is conflicting data about the accuracy of CT for pancreatic fat estimation. There are studies claiming there is no relationship between pancreatic adipose tissue infiltration and beta cell function, regardless of glucose tolerance status [33], while others correlate pancreatic fat by using CT with clinical assessment of impaired glucose metabolism [32].
Less radioactive but much more cost-effective techniques are magnetic resonance imaging (MRI) and magnetic resonance spectroscopy (MRS). As technology improves, we have alternative approaches to MR series. Magnetic Resonance Spectroscopy (MRS) is often considered as a quantitative and reproducible non-invasive clinical research tool and the gold-standard for non-invasive pancreatic fat quantification. It quantifies pancreatic triglyceride (TG) content and probably will enable systematic studies of the relationship between ectopic fat accumulation in the pancreas and development of type 2 diabetes [34].

MRS studies have also confirmed that pancreatic fat is increased in individuals with IFG and/or IGT and obesity [35]. One of the newly advanced techniques called IDEAL (Iterative Decomposition with Echo Asymmetry and Least squares estimation) compared to conventional MRS proved to have a stronger relationship mainly to hepatic fat and secondly to pancreatic fat, as well [36]. Another MRI technique is chemical-shift imaging (CSI). CSI imaging is an extension of MR spectroscopy, allowing metabolite information to be measured in an extended region and to add the chemical analysis of body tissues to the potential clinical utility of magnetic resonance [37].

Studies using CSI have reported correlations between pancreatic and visceral fat, but no association with insulin resistance, BMI, subcutaneous adiposity or other metabolic syndrome features [38]. Finally, proton density fat fraction mapping (PDFF), that gives quantitative information of fat deposition in liver and has a potential for quantifying the fat fraction in other tissues. In a study where liver and pancreatic fat were quantified by MRI-PDFF, among other tissues pancreatic fat was well correlated in NAFLD patients with diabetes [39].

**NAFPD and NAFLD**

Liver and pancreas steatosis have a strong association, since the two organs have a common embryogenic origin. NAFPD was associated with more than 2.5 times higher co-prevalence of NAFLD [40]. In a study in obese, but otherwise healthy, individuals, there were significant associations between pancreatic fat accumulation and liver fat content as well as insulin resistance and other metabolic abnormalities, mediated by the amount of visceral adipose tissue, assessed with MRI [41].

In addition, there are studies that show a significant relationship between fatty liver and fatty pancreas through endoscopic U/S [42]. The same relationship was proven when NAFLD and NAFPD were assessed through MRI. Probably, more interesting results in everyday clinical practice, where EUS and MRI are not an easy option, pointed that fatty pancreas was related, among others, with higher insulin resistance, visceral fat area, tri-glyceride, AST, ALT, and γGT levels than nonfatty pancreases [10].

There are even more conclusions to discuss from this same interesting study. Specifically, the positive predictive value of fatty liver in fatty pancreas was approximately 70%, but the negative predictive value of fatty liver in normal pancreas was up to 96%. In addition subjects with fatty pancreas had at the same time fatty liver in a percentage up to 68%, when at the same time almost all subjects with fatty liver (97%), had fatty pancreas, as well. Maybe, we could assume that NAFPD is an independent marker of ectopic fat accumulation of and an earlier one of metabolic syndrome than fatty liver [10].

Eventually, the pathophysiology underlying both conditions might be a coincidence rather than causative. Both fatty pancreas and fatty liver are significantly associated with metabolic risk factors due to excessive energy intake, playing probably a synergetic role even in risk of malignancy [25].

**NAFPD and hyperglycemia**

Maybe the most interesting clinical implication of NAFPD, that must be clarified, is diabetes mellitus (DM). The malfunction of β-cell is the key point for this clarification. The term that could explain their relationship is glucolipotoxicity referring to the harmful effects of elevated glucose and fatty acid levels on pancreatic beta-cell function and survival [43]. The combination of excessive levels of fatty acids and glucose therefore leads to decreased insulin secretion, impaired insulin gene expression, and beta-cell death by apoptosis [44], through several pathways, such as, the extracellular-regulated kinase (ERK1/2) pathway, the metabolic sensor Per-Arnt-Sim kinase (PASK), and the ATF6 branch of the unfolded protein response [45].
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In animals, chronic high-fat diet may lead to pancreatic free fatty acid accumulation and in turn, to the damage of acinar cells and islets, as well as fatty infiltration in the pancreas [46]. Human studies have not yet clarified the relationship between DM and pancreatic steatosis. There is a very interesting study that describes pancreatic atrophy and lipomatosis in diabetic subjects of two Norwegian families with a novel syndrome of DM and exocrine pancreatic dysfunction caused by heterozygous carboxyl-ester lipase (CEL) mutations [47], while others did not find association between pancreatic fat content and b-cell function [48].

The relation between NAFPD and insulin resistance is still unclear. Studies showed that NAFPD had a higher predictive value for insulin resistance along with central obesity and hypertriglyceridemia [17]. It seems that pancreatic fat and HOMA-IR have a linear relation and fatty pancreas has higher insulin resistance, visceral fat area, triglyceride, ALT levels and stronger correlation with metabolic syndrome [10]. There are studies instead that correlate insulin resistance more likely with NAFLD than NAFPD in obese patients [41].

Some studies have shown a correlation between pancreatic fat and prediabetic states and the amount of pancreatic fat increases before type 2 DM occurs [48]. In addition, pancreatic fat is negatively associated with insulin secretion in subjects with IGT/IFG and, therefore, might represent an additional pathogenetic factor leading to beta-cell dysfunction [49].

It is well understood until now that pancreatic fat infiltration might lead to a decrease in b-cell number and function, leading to more rapid progression to DM and that prolonged exposure to fatty acids impairs insulin gene expression in the presence of high glucose [50]. It remains unclear, still, if NAFPD itself leads to the progression and presence of type 2 DM. It is more likely that the presence of NAFPD in addition with NAFLD has a more positive value in the appearance of DM. This is shown in adults, where the coexistence of both non-alcoholic steatohepatitis and fatty pancreas has a further impact on glucose metabolism and DM frequency [51].

Studies showed that prevalence of DM was higher in individuals with NFAPD than normoglycemic ones and related with increased pancreatic fat [16]. Maybe the most interesting and only, so far, five-year longitudinal study to evaluate whether fatty pancreas increases the incidence of type 2 DM comes from Japan. Fatty pancreas was estimated with CT series and attenuation in three regions of the pancreas seen on an unenhanced CT scan was measured, and the mean pancreatic attenuation was calculated to evaluate fatty pancreas at baseline. Univariate analysis resulted in positive correlation between DM and fatty pancreas but after adjustment for potential confounders age, sex, BMI, liver attenuation and alcohol intake fatty pancreas was not independently associated with future type 2 DM [52].

In any case, even if the association between NAFPD and DM is not yet clarified, it is very possible that pancreatic fat could be an independent factor for glucose regulation.

NAFPD and cardiometabolic risk

Since the recognition and diagnosis of NAFPD as an autonomous entity is growing, it is very interesting to see the overall relation not only with diabetes solely, but with metabolic syndrome and other cardiometabolic parameters as well. Starting from animal studies, it was documented that obese mice have heavier pancreas and more pancreatic fat, especially triglycerides, and concluded that obesity leads to fat infiltration of the pancreas [53]. NAFPD and its relation to age, BMI, atherosclerosis and diabetes was first described in post-mortem studies [54].

In a large systematic review, the presence of NAFPD is associated with a 67% higher risk of hypertension, 108% higher risk of DM, and 137% higher risk of metabolic syndrome [18]. In a another case-control retrospective study NAFPD was associated with higher values in the following parameters: BMI, abdominal girth/height, abdominal girth (both genders), fasting and postprandial blood glucose, HbA1c, total cholesterol, triglycerides, LDL-cholesterol, systolic blood pressure, and platelet count [55]. Pancreatic fat is strongly associated with carotid atherosclerosis in non-obese subjects with type 2 DM [56]. This relation was confirmed again when fatty pancreas was associated with carotid intima-media thickness (CIMT) and carotid femoral pulse wave velocity (cf-PWV).
Ethnicity could make a difference in metabolic risk. Certain groups may also have a greater prevalence of NAFPD. For example, it was shown that Hispanics had greater pancreatic fat content than African Americans [48]. Inside the equation of increased metabolic risk in patients with NAFPD, we could insert obstructive sleep apnea syndrome (OSAS), since OSAS is strongly associated with cardiovascular and metabolic diseases may be an independent risk factor for their occurrence [57].

It is well understood, from the previous studies that, from a clinical point of view, it would be useful to be more careful with diagnosing NAFPD in subjects with the metabolic syndrome. The initial definition concluded central obesity, glucose intolerance or diabetes, hypertension and dyslipidemia. NAFLD and NAFPD could be considered as important manifestations of metabolic syndrome [9].

**NAFPD and pancreatic diseases**

One of the most interesting association of NAFPD is the one with other pancreatic diseases such as acute and chronic pancreatitis and carcinoma of the pancreas.

First, we should have in mind that when we get more obese (BMI>30), more fat accumulates in various areas in our body including within the abdominal viscera such as the pancreas [58]. Obesity itself, through visceral fat is linked with poorer prognosis of acute pancreatitis and is recognizable as a risk factor for severe acute pancreatitis [59]. It seems that in obese individuals an increase in the volume of intrapancreatic adipocytes was associated with more extensive pancreatic necrosis during acute pancreatitis and that acute pancreatitis was associated with multisystem organ failure in obese individuals [59]. The worst damage was observed in necrosed adipocytes, which were surrounded by a zone of necrosed pancreatic parenchyma, the so-called peri-fat acinar necrosis, indicating a direct link between the severity of necrosis and the location of infiltrative fat [60].

Having in mind that trypsinogen is activated during pathogenesis of acute pancreatitis in mice, and Intra-acinar trypsinogen activation leads to acinar death during early stages of pancreatitis, maybe the measurement of trypsin could be an extra prognostic factor of severe acute pancreatitis in obese patients [61]. There are animal studies suggesting that NAFPD may lead to non-alcoholic steato pancreatitis (NASP), taking under consideration the fact that obese mice (increased total fat, triglycerides, cholesterol, free fatty acids, and saturated free fatty acids) had more severe pancreatitis histologically than the lean mice, and the hyperleptinemic obese mice had the most severe pancreatitis [62]. To conclude, from a clinical point of view, we should be of high clinical suspicion in humans with NAFPD for having acute pancreatitis.

Chronic pancreatitis is thought to be a result of relapsing episodes of acute pancreatitis. The main pathophysiological feature that indicates the progression from acute to chronic pancreatitis is the reduction in parenchymal mass and its replacement with adipose tissue and fibrosis. It seems that this gradual progressing fibrosis limits the existence of pancreatic fat, which in turn, reduces the severity of acute exacerbations of chronic pancreatitis by reducing lipolytic flux between adipocytes and acinar cells turn [63].

In an animal study, where long-term high-fat diet led to a significant increase in body weight and serum concentrations of triglycerides and cholesterol and significant visceral and subcutaneous fat accumulations, chronic pancreatic injuries were induced, probably through microcirculatory disturbances and oxidative stress [64]. After all, these permanent occult alterations may lead to exocrine and endocrine dysfunction and pancreatic fibrosis as permanent and irreversible damages and develop to typical chronic pancreatitis [64].

It would be very helpful, if we had an easy access for having human pancreatic biopsies, in order to study thoroughly, the gradual progression of fibrosis in pancreas and have more information for a possible NAFPD - NASP model, as we already mentioned. Studies on pathogenic mechanism of fibrosis in human chronic pancreatitis are restricted by limited availability of tissues obtained from surgery. Therefore, animal models, despite their limitation in recapitulating all aspects of human disease, have been useful to investigate the initiation and progression of chronic pancreatitis. There is a very interesting study supporting the fact that mouse and human pancreatic stellate cells are a source of IL-4/IL-13 and their pharmacologic inhibition in human *ex vivo* studies as well as in established mouse chronic pancreatitis, decreases pancreatic activated macrophages and consequently, fibrosis [65].
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It is already known that obesity is associated with increased risk of common and less common malignancies in both men and women, such as gastrointestinal adenocarcinomas, thyroid, renal and endometrial cancers, even with weaker associations with haematological malignancies [66]. Hyperinsulinemia and possibly hypoestrogenism secondary to a metabolic syndrome, and independently from diabetes status, appear to be the key elements of the pathogenesis in pancreatic cancer secondary to excess body fat [67].

Fatty infiltration is a significant risk factor for pancreatic ductal adenocarcinoma and this relation remained significant even after adjustment for BMI, prevalence of DM and other confounding factors [68]. Obese individuals were also shown to have an approximately 20% greater risk of developing pancreatic adenocarcinoma compared to normal weight controls and centralized fat distribution may increase pancreatic cancer risk, especially in women [69].

As we already mentioned, most patients with pancreatic cancer have significant pancreatic fibrosis and there is evidence that NAFPD itself is a prognostic factor for pancreatic cancer [70]. However, whether this pancreatic fibrosis has been caused by non-alcoholic SteatoPancreatitis (NASP) a concept analogous to non-Alcoholic Steatohepatitis (NASH), after which pancreatic cancer develops, or whether pancreatic cancer causes the fibrosis owing to duct obstruction is unknown [9].

In contrast to the endocrine function, which is damaged by fat accumulation, exocrine pancreatic function is not affected until over 90% of its tissue is replaced by fat [71]. There is little evidence regarding the association between pancreatic steatosis and exocrine dysfunction, since diffuse and primitive fat replacement of the exocrine pancreas is a rare cause of exocrine pancreatic insufficiency in adults. Although, theoretically, fat accumulation could cause exocrine insufficiency, that is not confirmed yet by studies, and only case reports reported association with exocrine insufficiency [72]. The most prominent clinical implication of exocrine dysfunction is malabsorption syndrome including chronic diarrhoea, fatty stools, and weight loss [15].

Treatment

It is very clear until now that there is no targeted treatment for improving NAFPD. The only treatment we have available is lifestyle modification, weight loss and low-calorie diet that could lead to normalization of both beta cell function and hepatic insulin sensitivity in type 2 DM. We already know that type 2 DM is clearly reversible following bariatric surgery and the normalisation of plasma glucose concentration follows within days of surgery [73].

In addition, during a follow up eight-week period after bariatric surgery in diabetic patients a fall in intrapancreatic triacylglycerol occurred [74]. A very interesting study demonstrated the time course of a return of normal beta cell function and hepatic glucose output by acute restriction of dietary energy intake in individuals with type 2 DM. The changes occurred in association with decreases in pancreatic and liver triacylglycerol concentrations [75]. In vitro studies examine the potential beneficial effect of drug treatment such as troglitazone or berberine and cinnamic acid in pancreatic steatosis [27].

Conclusion

NAFPD is a new upcoming clinical entity as the pandemic of obesity and DM grows. It reflects the fat accumulation in the pancreatic tissue which is more and more recognizable due to modern radiology techniques. More often, it coexists with NAFLD, but it seems that NAFPD has strong relations with the metabolic syndrome and the overall damage of the pancreatic tissue. We suggest that medical society should be aware of its existence because it may have a greater clinical impact than we believed until now. Further studies should clarify its pathogenesis and clinical course in order to understand it better and even find targeted treatments.

Bibliography


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