

Pancreatic Steatosis - An Innocent Radiological Finding or a Real Disease?

Zoltán Berger*

Department of Medicine, Gastroenterology Section, Hospital Clinico Universidad de Chile, Santiago, Chile

***Corresponding Author:** Zoltán Berger, Department of Medicine, Gastroenterology Section, Hospital Clinico Universidad de Chile, Santiago, Chile.

Received: July 27, 2020; **Published:** August 05, 2020

Keywords: *Non Alcoholic Fatty Pancreas Disease (NAFLD); Obesity; Metabolic Syndrome*

Some decades ago, we began to receive patients in our gastroenterology consultations who asked us: Doctor, I have a fatty liver described by abdominal ultrasound. What is it? Is it dangerous? In general, the gastroenterologists and even the hepatologist told them: take it easy... But the number of patients with this finding of uncertain importance became rapidly growing and nowadays non-alcoholic fatty liver disease (NAFLD) and non-alcoholic steatohepatitis (NASH) transformed in one of the most important etiologies of chronic liver disease and even a leading cause of liver transplantation in several countries.

Now in the last years we begin to see in the radiological reports of abdominal images: fatty infiltration, fatty replacement of the pancreas, or pancreatic steatosis in patients without clinically evident or even suspected pancreatic disease. And again, we tell to our patients: take it easy... But is it correct? Or should we prepare to face an increasing importance of this phenomenon?

In effect, the literature dedicated to fatty infiltration of pancreas is growing, the nomenclature and the significance of different descriptive diagnosis is more and more determined and accepted [1,2]. The terminology "fatty infiltration of pancreas", "pancreatic steatosis" (PS) is used simply to describe the radiological finding. The expression "Non-alcoholic fatty pancreatic disease" (NAFPD) not only signals the analogy with the liver, but already gives a clear clinical importance to the presence of increased amount of fat in the pancreas. Apart of the fatty infiltration of the pancreas, these patients have an increased body mass index (BMI > 30) and metabolic syndrome, but the exocrine pancreatic morphology and function continue to be well preserved.

Pancreatic steatosis has been described as a frequent finding in autopsy, mainly in elderly people and diabetics. It is easily detected by computed tomography, magnetic resonance, endoscopic and even abdominal ultrasound. It is found in more than 30% of abdominal images, excluding patients suffering pancreatic diseases. Its frequency increases with the age, obesity, with diabetes and metabolic syndrome. Several authors found the PS associated to fatty infiltration of the liver, while others not.

There are some differences when PS is compared to NAFLD. Lipid droplets are intracellular in NAFLD; they are seen inside of the hepatocytes. In contrast, the fat accumulation is mainly inter- and intralobular in PS but not intracellular, acinar cells conserve their normal, habitual aspect in the immense majority of cases. However, endocrine pancreas is also affected; several authors described altered morphology of Langerhans islets. Association of PS and metabolic syndrome raises the question of hen or egg: accumulation of the fat favors the inflammation and islet cell damage, but the contrary is also true, islet cell damage favors the increased accumulation of the fat in the whole body, including the pancreas. Thus, a vicious circle is complete, which includes also the major risk of NAFLD - NASH.

The other principal difference is that the clinical significance of PS is not clear till now, it is certainly incomparable to the liver. Its known importance in the pancreatology that fatty infiltration of pancreas increases the risk of fistula after pancreatic surgery [3]. However, already 40 years ago Bordalo's group documented the progression of fatty pancreas to chronic pancreatitis (CP) with nicely illustrated sequential histologic images [4]. This way of development of CP has been considered to be exceptional and the fatty infiltration - "steatopancreatitis" - pancreatic fibrosis/ "cirrhosis" (CP) sequence is not a widely accepted pathomechanism by the majority of experts. Nonetheless, these findings showed a clear progression of fatty pancreas to chronic pancreatitis in some alcoholics. While exceptional, the existence of this sequence was demonstrated. Without underestimating the importance of alcohol consumption, the obesity and the aging represent serious problem for the public health and epidemiology in the whole world. These are the two principal risk factors for the development of PS, thus we can wait for a markedly increased prevalence of NAFPD in the next years. It is estimated that 1.5 to 15% (!) of the "innocent" fatty liver progresses to liver cirrhosis during the years, through steatohepatitis. We have not identified the factors which can transform the "innocent" pancreatic steatosis into a progressive disease, steatopancreatitis and chronic pancreatitis. The obesity and the metabolic syndrome certainly maintain a permanent, while low-grade inflammation [5] in the pancreatic tissue and the alcohol consumption is an additional risk factor. If we suppose that no more than 0.1% of PS progresses to Non-alcoholic Steatopancreatitis (NASP) and consecutive chronic pancreatitis i.e. 10 to 100 times less than in the case of the liver, we can predict a very significant increase in the incidence of inflammatory diseases of the pancreas in the next future. It is time to be prepared.

Bibliography

1. Smits MM and Van Geenen EJM. "The clinical significance of pancreatic steatosis". *Nature Reviews Gastroenterology and Hepatology* 8 (2011): 169-177.
2. Ramkissoon R and Gardner TB. "Pancreatic Steatosis: An Emerging Clinical Entity". *The American Journal of Gastroenterology* 114 (2019): 1726-1734.
3. Gaujoux S., *et al.* "Fatty pancreas and increased body mass index are risk factors of pancreatic fistula after pancreaticoduodenectomy". *Surgery* 148 (2010): 15-23.
4. Noronha M., *et al.* "Sequential changes from minimal pancreatic inflammation to advanced chronic pancreatitis". *Zeitschrift für Gastroenterologie* 21 (1983): 666-673.
5. Liu L., *et al.* "Roles of Chronic Low-Grade Inflammation in the Development of Ectopic Fat Deposition". *Mediators of Inflammation* (2014): 418185.

Volume 7 Issue 9 September 2020

©All rights reserved by Amjad M AlRashed and Mohamed Almousa.