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Original Research

Investigating the Correlation between Non-Alcoholic Fatty Pancreas and Non-Alcoholic Fatty Liver Disease through Transabdominal Ultrasound Examination

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ABSTRACT:

Background:In contemporary times, the detection of fat infiltration in the pancreas has become a frequent observation during routine abdominal ultrasound examinations. Notably, this occurrence often coincides with fatty infiltration in the liver. Consequently, there is a plausible connection between non-alcoholic fatty pancreas (NAFP) and non-alcoholic fatty liver disease (NAFLD), particularly prevalent in urban populations and developing countries. Numerous studies have delved into this correlation, revealing that several metabolic factors implicated in fatty pancreas are also associated with fatty liver disease, as evidenced by previous research. Today, the clinical implications of non-alcoholic fatty pancreas have captured the attention of clinicians, particularly those specializing in gastroenterology. Our study endeavors to unveil the potential link between non-alcoholic fatty pancreas and non-alcoholic fatty liver disease.**Methods**:In this retrospective analysis, a total of 610 cases were included, encompassing individuals within the age range of 20 to 60 years. The imaging scans were conducted under the scrutiny of two seasoned radiologists, ensuring a comprehensive and expert evaluation.**Results**:Our study reveals a noteworthy finding: a positive correlation exists between the prevalence of non-alcoholic fatty pancreas (NAFP) and non-alcoholic fatty liver disease (NAFLD). This correlation is particularly evident, as fatty pancreas emerges as a frequent observation in abdominal ultrasound examinations.**Conclusion**:While our study has successfully identified an elevated prevalence of fatty pancreas within the normal population and its association with fatty liver, further investigations are warranted to elucidate the intricate relationship between metabolic abnormalities and non-alcoholic fatty pancreas. The need for additional studies arises to deepen our understanding of these connections and unveil potential underlying mechanisms.

Keywords: NAFP (Non-alcoholic fatty pancreas) NAFLD (Non-alcoholic fatty liver disease, Abdominal Ultrasound, Metabolic Diseases.

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INTRODUCTION

The accumulation of fat in visceral organs within the human body is a complex phenomenon, often attributed to a heightened intake of dietary fats. One specific manifestation of this phenomenon is non-alcoholic fatty pancreas (NAFP), where various factors contribute to its development, with obesity standing out as a principal cause, particularly in the absence of alcohol consumption.A myriad of terminologies has been employed to characterize intrapancreatic fat deposition, showcasing the diverse nature of this condition.[1] The terms used include non-alcoholic fatty pancreatic disease, lipomatous atrophy of the pancreas, pancreatic adiposity, fatty replacement of the pancreas, pancreatic steatosis, and fatty infiltration of the pancreas.The term "fatty pancreas," alternatively known as pancreatic steatosis, was first introduced into medical discourse by Ogilvie in 1978. In their pioneering study, they observed a significant association between obesity and fatty pancreas, reporting that 17% of obese patients exhibited this condition compared to 7% in their slim counterparts. Building on this, Olsen et al contributed to the understanding of NAFP by establishing an age-related increase in pancreatic fat deposition. Subsequent research by Stamm further supported these findings, indicating a positive correlation between age and fat content in the pancreas.

Insights from Van Geenen et al's studies shed light on the intricate role of insulin resistance in the development of fatty deposits in the pancreas. This insulin resistance not only contributes to the accumulation of fat within the pancreas but also sets off a cascade of events leading to peripheral steatosis. Furthermore, the influx of fatty acids into the hepatic parenchyma triggered by insulin resistance has been identified as a pivotal factor in the genesis of non-alcoholic fatty liver disease (NAFLD).In summary, the intricate relationship between high-fat intake, obesity, insulin resistance, and the various terms used to describe fatty infiltration in the pancreas underscores the multifaceted nature of this condition.[2] Ongoing research endeavors continue to unravel the complexities surrounding non-alcoholic fatty pancreas and its interconnectedness with broader metabolic abnormalities.

The collective body of literature in the medical domain consistently underscores a compelling association between non-alcoholic fatty pancreas (NAFP) and non-alcoholic fatty liver disease (NAFLD). This correlation extends beyond mere coincidence, as both conditions exhibit a noteworthy connection to metabolic syndrome, implying a shared set of underlying metabolic disturbances. The embryological origin of the pancreas and liver from the same embryonic endoderm further reinforces the idea that these two organs might share a predisposition to metabolic abnormalities, providing a solid basis for the observed correlation between NAFPD and NAFLD.

In-depth analyses conducted by various researchers consistently reveal a high prevalence of NAFPD among individuals diagnosed with NAFLD, with estimates suggesting that nearly 70% of patients with NAFLD also exhibit signs of fatty pancreas. This robust association raises intriguing questions about shared etiological factors and pathways contributing to the development of both conditions.Exploring the common risk factors implicated in the deposition of fat in both the pancreas and liver unveils a spectrum of influences. Hypertension, male gender, hypertriglyceridemia, and central obesity emerge as significant contributors, acting as shared catalysts for fat accumulation in these vital organs.[3] This convergence of risk factors further deepens the understanding of the intricate interplay between NAFPD and NAFLD, shedding light on the multifaceted nature of these metabolic disorders.The study by Dite P et al adds granularity to our comprehension of the mechanisms and risk factors associated with fat accumulation specifically in the pancreas. Older age, higher body mass index, dyslipidemia, and the presence of metabolic syndrome with metabolic dysfunction are identified as critical elements contributing to the buildup of fat in the pancreas. This nuanced understanding allows for a more targeted exploration of potential intervention strategies and highlights the need for a comprehensive approach to managing these metabolic conditions.

Against this backdrop of existing evidence and gaps in understanding, our study endeavors to contribute to the scientific discourse by delving into the specifics of the correlation between non-alcoholic fatty pancreas and non-alcoholic fatty liver disease.[4] By addressing this research gap, we aspire to provide nuanced insights that not only enrich our understanding of these interconnected conditions but also pave the way for more effective diagnostic and therapeutic strategies in the context of metabolic syndrome.

MATERIALS AND METHODS

The study, encompassing a robust cohort of 610 participants aged between 20 to 60 years, sought to meticulously investigate the potential association between non-alcoholic fatty pancreas (NAFP) and non-alcoholic fatty liver disease (NAFLD). The research, conducted by two proficient radiologists utilizing abdominal ultrasound examinations, adhered to a well-defined set of inclusion and exclusion criteria to ensure the reliability and validity of its findings.

INCLUSION CRITERIA

The inclusion criteria were carefully crafted to select a representative study population. Patients within the age range of 18 to 60 years were considered, irrespective of gender, to capture a broad demographic. The participants were required to be free from serious illnesses at the time of examination, ensuring that the observed conditions were not confounded by acute health issues. In addition, inclusion mandated the availability of routine laboratory tests encompassing Liver Function Tests (LFT), Fasting Blood Sugar (FBS), and Postprandial Blood Sugar (PPBS). Only patients with a confirmed diagnosis of fatty liver through these tests were included, maintaining specificity and relevance to the study objectives.

EXCLUSION CRITERIA

The exclusion criteria were designed to refine the study cohort and eliminate potential confounding factors. Patients with alcoholic fatty liver disease were excluded to focus specifically on non-alcoholic etiologies. Cases where the pancreas could not be adequately visualized during abdominal ultrasound examinations were excluded to ensure data accuracy. Individuals with a history of renal disease were excluded to prevent the influence of this factor on the observed conditions. Participants with a history of alcohol consumption were omitted to maintain the study's focus on non-alcoholic fatty liver disease and its potential correlation with non-alcoholic fatty pancreas. Cases with a history of pancreatic operations were excluded to avoid the impact of surgical interventions. Patients with pancreatic

diseases other than fatty pancreas were also excluded to enhance the study's clarity and relevance.

This stringent selection process was undertaken to create a homogeneous study population, facilitating a more focused investigation into the nuanced relationship between non-alcoholic fatty pancreas and non-alcoholic fatty liver disease. By carefully defining and adhering to these criteria, the study aimed to provide precise and insightful findings, advancing our understanding of the intricate interplay between these two conditions within a carefully delineated patient cohort.The clinical examination of patients commenced after obtaining informed consent, adhering to a meticulous protocol for abdominal ultrasound examination. Prior to the procedure, all necessary preparations were conducted to ensure the smooth implementation of the study. Patients were positioned in the supine posture, and the examination began with a focus on pancreatic echogenicity, comparing it to the echogenicity of the renal cortex and liver. In instances where the pancreas was not adequately visualized, alternative approaches, such as examining the patient in the right lateral decubitus position with a waterbed in the stomach, were employed to enhance visibility.Fatty pancreas was defined through a careful assessment of echogenicity, specifically as an image displaying increased echogenicity of the pancreatic parenchyma in comparison to the renal and liver echogenicity. This evaluation was conducted systematically, with the pancreas examined following the assessment of the liver or both kidneys to ensure a comprehensive understanding of echogenicity patterns. A diagnosis of fatty pancreas was assigned when a comparative hyperechoic pancreas, in contrast to the renal cortex, was identified. Following the primary assessment, the presence of elevated echogenicity in the pancreas was correlated with the presence of hepatic steatosis.To enrich the analysis, ultrasound findings were then correlated with pertinent laboratory parameters, including liver function tests, lipid profile, and gluco-insulinemic profile (including Random Blood Sugar - RBS, and Hemoglobin A1c - HbA1c). In instances where laboratory data was not initially available, patients were recommended for further laboratory investigations as deemed necessary to augment the comprehensive evaluation.

RESULTS

In our comprehensive study involving 610 patients, abdominal ultrasound examinations revealed that the pancreas was successfully visualized in 490 individuals, constituting 80.32% of the total cohort. Conversely, non-visualization of the pancreas was noted in 120 patients, accounting for 19.67% of the study population.Within the subset of 490 patients where the pancreas was visualized, further analysis demonstrated that 188 patients, or 38.36%, exhibited signs of fatty pancreas, while the remaining 302 patients (61.63%) showed no evidence of pancreatic steatosis.

Delving deeper into the correlation between fatty pancreas and fatty liver, among the 188 patients with fatty pancreas, a substantial 141 individuals (75%) were concurrently diagnosed with fatty liver, indicating a notable association between these two conditions. In contrast, 47 patients (25%) with fatty pancreas presented with normal liver status.Remarkably, among the 302 patients without evidence of fatty pancreas, 153 individuals were found to have fatty liver, highlighting that the co-occurrence of fatty liver in the absence of fatty pancreas was also noteworthy. Collectively, our study underscored the intricate interplay between fatty liver and fatty pancreas, revealing that a significant proportion of patients (75%) demonstrated the coexistence of both conditions. These findings contribute valuable insights into the concurrent presence and potential association between non-alcoholic fatty pancreas and non-alcoholic fatty liver disease within our study population.

Table1: Depiction of cases

Examination Outcome	Number of Patients	Percentage
Pancreas Visualized	490	80.32%
Non-visualization of Pancreas	120	19.67%
Fatty Pancreas	188	38.36%
Non-fatty Pancreas	302	61.63%
Fatty Pancreas + Fatty Liver	141	23.11%
Fatty Pancreas + Normal Liver	47	7.70%
Normal Pancreas + Fatty Liver	153	25.08%

Figure1: Depiction of cases

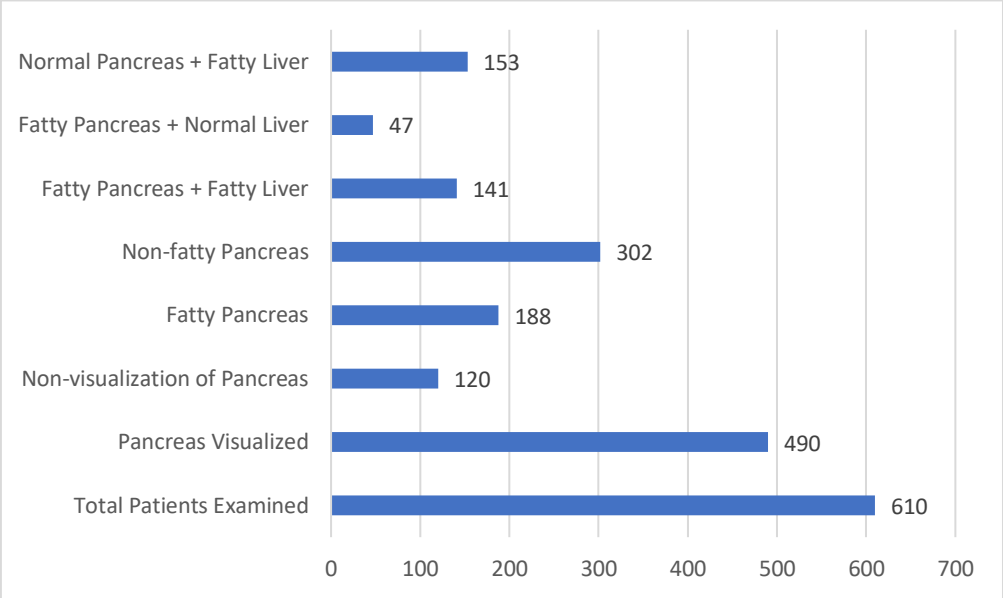
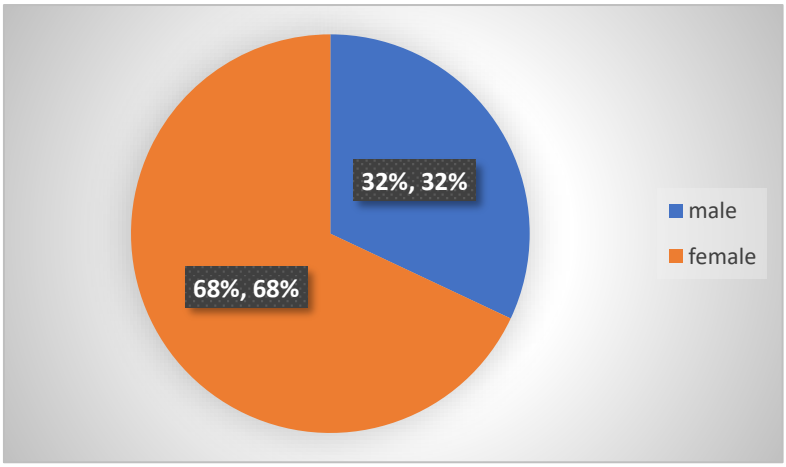


Figure2: Gender distribution



Intriguingly, our study uncovered a notable gender disparity in the prevalence of fatty pancreas. Among the individuals diagnosed with this condition, a higher proportion—68%—was observed in males, while females constituted the remaining 32%. This finding adds a nuanced dimension to our understanding of the distribution of fatty pancreas within the studied population, suggesting that gender may play a role in influencing the occurrence of this condition. Further exploration and analysis of potential contributing factors could shed light on the underlying mechanisms driving this observed gender difference in the prevalence of fatty pancreas.

DISCUSSION

The detection rate of fatty pancreas in our study, at 38.36%, aligns with findings from a study by Lesmana et al, where a similar diagnosis was made in 35% of the cases.[5] Interestingly, a study conducted in Korea reported a higher detection rate of 67.9%, potentially attributed to a focus on obese patients in their research, in contrast to the more generalized approach in our study.Remarkably, our study revealed a substantial overlap between fatty liver and fatty pancreas, with 76% of patients diagnosed with fatty pancreas also presenting with fatty liver. This robust association underscores a significant link between these two conditions and suggests a commonality in the underlying metabolic risk factors contributing to their coexistence.[6]In conclusion, our study contributes valuable insights into the prevalence of fatty pancreas, aligning with and adding to existing literature. The observed association between fatty liver and fatty pancreas emphasizes the need for a comprehensive understanding of metabolic risk factors and their implications in the manifestation of these conditions. The nuanced comparison with studies from different regions further enriches our understanding, highlighting potential variations in detection rates influenced by demographic or methodological factors. The accumulation of fat in the pancreas can occur through two primary mechanisms, each contributing

to what is commonly referred to as non-alcoholic fatty pancreatic disease (NAFPD). The first mechanism involves the death of acinar cells, the cells responsible for producing digestive enzymes in the pancreas.[7] When these acinar cells die, they are replaced by adipose tissue, leading to what is known as "fatty replacement." This process fundamentally alters the composition of the pancreas, substituting functional tissue with fat.

The second mechanism involves the direct accumulation of fat within the pancreatic tissue, referred to as "fatty infiltration." In this scenario, excess fat is deposited within the pancreatic parenchyma, impacting the organ's normal structure and function. Both mechanisms contribute to the development of fatty pancreas, a condition associated with various metabolic abnormalities.Numerous studies have investigated the factors contributing to the onset of NAFPD, and maternal obesity or exposure to an obesogenic diet during the postnatal period has emerged as a significant risk factor. This suggests a potential developmental origin for fatty pancreas, implicating early-life influences in its pathogenesis.

Dite et al have specifically identified several metabolic factors that play a crucial role in the development of non-alcoholic fatty pancreatic disease.[8] These factors include obesity, arterial hypertension, hyperglycemia (elevated blood sugar levels), and alterations in high-density lipoprotein (HDL), cholesterol, or the presence of diabetes mellitus. This underscores the complex and multifactorial nature of NAFPD, involving a combination of metabolic and lifestyle factors.Adding to this understanding, your study observed a higher detection of fatty pancreas in advanced age. This aligns with findings from previous research and suggests that age could be a contributing factor in the development or detection of fatty pancreas. The intricate interplay between aging and metabolic factors underscores the need for a holistic approach to studying and managing NAFPD.In summary, the development of fatty pancreas involves intricate processes such as fatty replacement and infiltration, influenced by a range of factors including maternal obesity, obesogenic diets, and various metabolic abnormalities. These insights contribute to a more comprehensive understanding of the pathogenesis of NAFPD, offering potential avenues for early intervention and management strategies.

CONCLUSION

The transabdominal ultrasound may not be the optimal choice for diagnosing fatty pancreas and its correlation with fatty liver compared to other modalities. However, it can still be considered a viable option if performed by an experienced radiologist due to its accessibility and cost-effectiveness. Our study indicates a higher prevalence of fatty pancreas in urban populations, with a significant number of cases exhibiting both fatty pancreas and fatty liver. The changing lifestyle and dietary habits in the modern era contribute to conditions like obesity and metabolic diseases, leading to the emergence of Non-Alcoholic Fatty Pancreatic Disease (NAFPD) as a concern for gastroenterologists and clinicians.Given the potential impact on prognosis, our study suggests prioritizing the diagnosis of fatty pancreas alongside fatty liver using transabdominal ultrasound. This approach aims to mitigate future adverse outcomes related to pancreatic diseases. We emphasize the need for further research and in-depth studies to identify risk factors associated with NAFPD, which poses a potential threat to human life. Intrapancreatic Fat (IPF) is highlighted as a factor that might play a role in causing or exacerbating various pancreatic diseases, including metabolic, inflammatory, fistula, and neoplastic conditions.

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