



Does Nonalcoholic Pancreatic Steatosis Always Correlate with Nonalcoholic Fatty Liver Disease?

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Purpose: To identify the correlation of nonalcoholic pancreatic steatosis (NAPS) with nonalcoholic fatty liver disease (NAFLD) in an outpatient group. Based on its metabolic and imaging properties, NAPS has been increasingly recognized in recent years; however, its interaction with NAFLD is still not clear.

Patients and Methods: In this cross-sectional observational study, 345 consecutive patients without any chronic illness who were referred to the senior radiologist for abdominal ultrasound (US) were included. The US report showed hepatic and pancreatic echogenicity. The patients' demographic, anthropometric, and laboratory data were collected from medical records.

Results: Overall, NAPS and NAFLD were seen in 227 (65.8%) and 219 (63.5%) patients, respectively. Normal echogenicity was noted in 74 (21.4%) patients. Forty-four patients (12.8%) had steatotic liver without NAPS, 52 (15.1%) had steatotic pancreas without NAFLD, and 175 (50.7%) had steatosis in both organs. The discordance in steatosis grading between NAPS and NAFLD was 55.1%. Insulin resistance was present in 8.7, 26.7, 19, and 61.3% of patients with no steatosis, only NAFLD, only NAPS, and steatosis in both organs, respectively. Evident NAFLD and NAPS having grade 2 and 3 steatosis were present in 15.3% and 29.0% of the study group, respectively. Cholelithiasis was present in 6.8, 13.6, and 28.8% of patients with normal echogenic pancreas, only NAFLD, and only NAPS, respectively ($p=0.01$).

Conclusion: Based on the ultrasonographic, clinical, demographic, and anthropometric features of the included patients, we found that NAPS did not fully accompany nonalcoholic fatty liver. Despite severe pancreatic steatosis, more than a quarter of cases had normal liver echogenicity. Insulin resistance frequency was insignificantly higher in NAFLD than NAPS ($p=0.694$). The significantly higher frequency of cholelithiasis in NAPS needs further large-scale studies. The inconsistency of steatosis degree in NAPS and NAFLD in >50% cases may reflect differences in the pathophysiology of these two clinical entities.

Keywords: nonalcoholic pancreatic steatosis, nonalcoholic fatty liver disease, insulin resistance, ultrasound

Introduction

Nonalcoholic fatty liver (NAFLD) is defined as the intracellular deposition of triglycerides in hepatocytes, and nonalcoholic pancreatic steatosis (NAPS) is identified as the fatty infiltration of the pancreas; both conditions were found to be highly related with metabolic, cardiovascular, and oncological risks, as well as obstructive sleep apnea.¹⁻⁴ Fat replacement of the pancreas is substitution of fat in areas of acinar cell loss, while fat accumulation is parallel to an increase in total body adipocyte load leading to fatty infiltration of the pancreas.^{1,2} Despite being a fairly new disease entity,

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there are approximately 5346 publications on NAPS in PubMed, as revealed by the keyword search on “pancreatic steatosis”, accessed on April 16, 2021; in contrast, NAFLD featured in a total of 91,386 publications. NAPS is generally regarded as a consequence of NAFLD.^{1,6,7} The relationship of NAPS with the severity of acute pancreatitis and development of pancreatic cancer is still debatable.^{1,2} In this study, we aimed to compare the features, correlations, and inconsistencies of NAPS and NAFLD. Fat infiltration of the pancreas was assessed in 35% of medical check-up cases.⁸

Patients and Methods

In this retrospective, observational, analytical case-control study, the cases comprised 345 consecutive dyspeptic patients without any chronic illnesses, who were referred for abdominal ultrasound (US) to the senior radiologist blinded to the clinical and laboratory details. The report included the height and contour of the liver, portal vein wall visualization, gall bladder examination, echogenicity status, and grading of hepatic and pancreatic steatosis. Pancreatic echogenicity was evaluated using the following grading system: Grade 0=pancreatic echogenicity was equal to renal cortical echogenicity, Grade 1=definitely lower than retroperitoneal fat, Grade 2=slightly lower than retroperitoneal fat, and Grade 3=equal to retroperitoneal fat. The grading for NAFLD was as follows: Grade 0=normal echotexture of the liver, Grade 1=slight and diffuse increase of liver echogenicity with normal visualization of the diaphragm and portal vein wall, Grade 2=moderate increase of liver echogenicity with impaired appearance of the portal vein wall but still appreciable diaphragm echogenicity, and Grade 3=marked increase of liver echogenicity with almost no visualization of the portal vein wall, diaphragm, and posterior part of the right liver lobe.^{9–11}

Body mass index (BMI) was calculated as weight (kg)/square of height (m). A BMI between 17 and 25 kg/m² was accepted as the normal range. Body fat percentage (BFP) was estimated using the following formula: $BFP = 1.39 \times BMI + (0.16 \times \text{age}) \times (10.34 \times \text{sex}) - 9$, where sex for female was assigned a 0 and for male was assigned a 1.

The demographic, anthropometric, and laboratory data from medical records of patients were also collected. Study approval was obtained from the ethical committee at Medeniyet University (2019–015). The tenets of the Declaration of Helsinki and guidelines of Good Clinical Practice were adhered to. The need for informed consent was waived because of the retrospective nature of the study. The authors preserved the privacy and confidentiality of study cases.

Statistical Analysis

All statistics were performed in SPSS 20 (IBM Corporation, Armonk, NY, USA). Numerical data are given as mean±standard deviation (mean±SD). The power analysis was estimated with effect size 0.2, alpha 0.05, power 0.80, and dF as 5, which gave an output of 321 cases. The normality of distribution was assessed by Kolmogorov–Smirnov and Shapiro–Wilk tests depending on case number. Categorical data were analyzed by chi-square or Fisher’s exact tests. Student’s *t*-test or Mann–Whitney *U* test were used for comparison of two groups, and ANOVA or Kruskal–Wallis were used for >2 groups depending on normality of distribution. The cut-off point was calculated based on a receiver operator characteristic (ROC) curve analysis. A Spearman correlation test with a binary logistic regression test was performed to analyze the correlation between parametric data. The results were given as mean±SD. *P*<0.05 was considered to indicate statistical significance.

Results

The study group consisted of 345 patients (220 female [mean age: 50±15 years, range: 19–84] and 125 male [mean age: 52±15 years, range: 20–84]). Normal liver and pancreatic echogenicity was present in only 74 (21.4%), NAPS with normal liver was present in 52 (15.1%), NAFLD with normal pancreas was detected in 44 (12.8%), and steatosis in both liver and pancreas was seen in 175 (50.7%) patients. The discordance in steatosis grading between NAPS and NAFLD was 55.1%. Isolated steatosis of liver or pancreas was assessed in 96 (35.4) of 271 cases with NAPS and NAFLD, whereas 175 (64.6%) of 271 patients had steatosis in both organs (Table 1). Insulin resistance (IR) was present in 8.7, 26.7, 19, and 61.3% of cases with no steatosis, only NAFLD, only NAPS, and both steatotic organs, respectively. Cholecystolithiasis, sludge, or history of cholecystectomy were noted in 6.8, 13.6, 28.8, and 28.3% of cases with no steatosis, only NAFLD, only NAPS, and both steatotic organs steatotic, respectively (*p*=0.01). Evident NAFLD and NAPS with grade 2 or grade 3 steatosis was present in 15.3% and 29.0% of patients, respectively. Cases with only NAFLD and only NAPS had insignificant differences in older age, IR, total cholesterol, LDL-cholesterol, and BFP. NAFLD and NAPS cases had significant differences in age (44±13 vs 56±15, *p*<0.001), BMI (28.1±4.2 vs 26.5±2.7, *p*=0.04), and HDL-cholesterol (46.0±9.1 vs 54±13, *p*=0.01), respectively (Table 2).

Table 1 Demographic and Anthropometric Features of Included Patients

Status of Steatosis	Normal	NAFLD	NAPS	NAPS+NAFLD	p**
Cases (n=345)	74(21.4%)	44(12.8%)	52(15.1%)	175(50.7%)	—
Sex (F/M)	54/20	29/15	37/15	100/75	0.661
Age (years)	41±14	44±13	56.4±15	55±13	0.000*
BMI (kg/m ²)	23±3	28±4	26±3	31±5	0.040*
BFP (%)	28±8	34±9	34±7	39±9	0.930
Diabetes mellitus	5 (6.8%)	7(15.9%)	9(17.3%)	84(36%)	1.000
Hepatomegaly	24.3%	47.7%	30.8%	70.3%	0.098
Cholelithiasis	6.8%	13.6%	28.8%	26.3%	0.087

Notes: *Significant (p<0.05); **Comparison between NAFLD and NAPS.

Abbreviations: NAFLD, nonalcoholic fatty liver disease; NAPS, nonalcoholic pancreatic steatosis; F, female; M, male; BMI, body mass index; BFP, body fat percentage.

Table 2 Laboratory Values of the Study Group

Status of Steatosis	Normal	NAFLD	NAPS	NAPS+NAFLD	p**
Insulin level	6.6±2.3	8.3±4.0	9.6±8.0	13.5±8.9	0.000*
Insulin resistance	1.5±0.6	1.9±0.8	3.2±5.7	3.9±3.5	0.017
ALT (IU/mL)	22±19	29±18	23±21	31±23	0.130
Triglyceride (mg/dL)	93±47	137±65	119±76	164±83	0.218
HDL-cholesterol	53±13	46±9	54±13	48±15	0.001*
Total cholesterol	189±47	205±46	202±42	207±45	0.816

Notes: *Significant (p<0.05); **Comparison between NAFLD and NAPS.

Abbreviations: NAFLD, nonalcoholic fatty liver disease; NAPS, nonalcoholic pancreatic steatosis; ALT, alanine aminotransferase.

Discussion

NAFLD and fatty infiltration of the pancreas are associated with abdominal obesity, insulin resistance, dyslipidemia, diabetes mellitus, and hypertension and are significant causes of global ill health and economic burden.^{3,5} The adipose tissue, as ectopic fat, may accumulate in the pancreas, muscles, renal sinus of the kidneys, cardiovascular system, internal organs, etc.^{12–14} Nonalcoholic pancreatic steatosis is also identified as fatty pancreas, nonalcoholic fatty pancreas disease, lipomatous pseudohypertrophy, and pancreatic lipomatosis.^{15,16} In this paper, we chose NAPS for clarity. Abdominal US was used in diagnosing and grading of NAPS and NAFLD. A previous study reported that US could better detect NAPS than NAFLD.¹⁷ Although noninvasive methods for detection and quantification with imaging techniques are being evaluated, there is still a lack of standardization.^{18,19}

The role of NAPS is still debatable in acute and chronic pancreatitis, pancreatic cancer, and pancreatic exocrine insufficiency.²⁰ Older age, high BMI, and insulin resistance are reportedly associated with NAPS,^{9,12,21,22} although some studies have found no relation between NAPS and beta-cell function.²³ Fat infiltration of the pancreas may lead to more bleeding and post-operative fistula

formation in pancreatic surgery.^{15,24} NAPS was reported to be associated with prediabetes, especially in male subjects.²⁵ In our study, compared to NAFLD, older age, higher HDL-cholesterol, and higher IR were more associated with NAPS, whereas triglycerides (Figure 1), diabetes mellitus, and BMI were prominent in NAFLD cases, consistent with some reports.^{5,25–27}

The proposed mechanisms of fatty infiltration in the pancreas were the substitution of acinar cells by adipose tissue and the adipocyte effect on exocrine function. In NAFLD, triglycerides and fatty acids are stored within the hepatocytes, whereas adipocytes and pancreatic fat accumulate partly in acinar cells and mainly in the intralobular and interlobular area, especially in septae which are horizontally parallel to the main duct and primarily in the pancreatic head.^{2,12,28} Both ongoing fibrosis and replacement of functional cells of the pancreas with fat and adipocytes may result in insufficiencies in endocrine and exocrine functions.^{29,30} Other data have shown that NAPS affects exocrine functions more than endocrine cells and is associated with age, BMI, and decreased serum lipase.³¹ Our results for amylase and lipase were not different between NAPS patients with normal and steatotic livers (p=0.793).

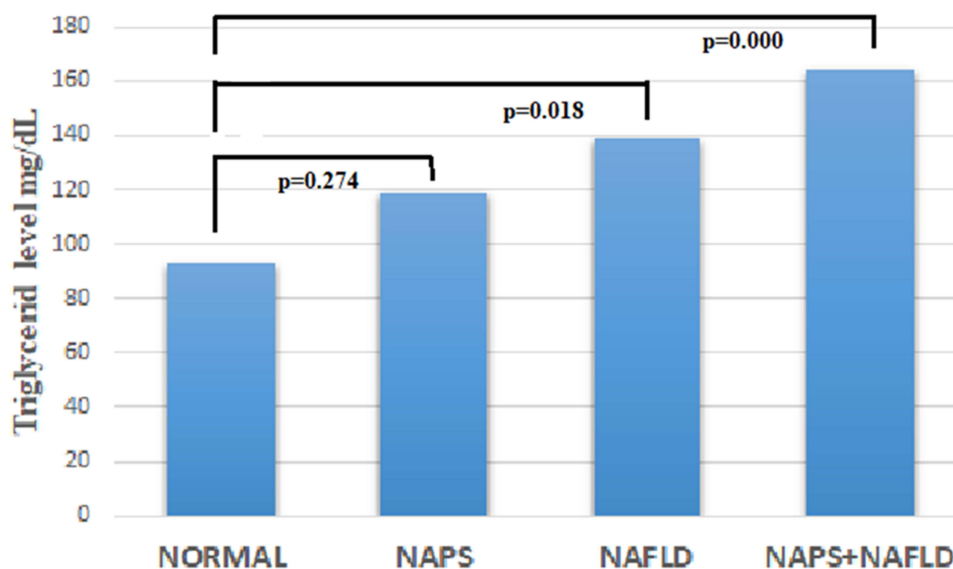


Figure 1 Serum Triglyceride levels of subgroups.

Abbreviations: NAPS, Nonalcoholic pancreatic steatosis; NAFLD, Nonalcoholic fatty liver disease.

The strong correlation of cholecystolithiasis with NAFLD and NAPS in cholecystectomy has been reported in a previous study.³² In our study, gall bladder diseases were highly associated with NAPS rather than NAFLD. The pancreas and gallbladder originate from the same endodermal pouch and are bound via the common bile duct and Wirsung channel. As in biliary pancreatitis, the inflammatory storm or low-level ongoing inflammation in obesity may simultaneously affect both organs. Moreover, age and obesity as the shared risk factors for both may explain the simultaneous increase in fatty pancreas and cholecystolithiasis.

The mechanism of excess fat accumulation may be pathophysiologically different between alcoholic and metabolic fatty pancreas. In a study, alcohol caused intra-pancreatic esterification of cholesterol and thereby accumulation of cholesteryl ester but not other lipids in three-week alcohol- or isocaloric carbohydrate-fed rats.³³ NAPS may progress to nonalcoholic steatopancreatitis (NASP), and analyzing these two consequential steps separately, as in the relationship between NAFLD and nonalcoholic steatohepatitis (NASH), may give more uniform results. The effect of adipocyte-derived adipokines, macrophages, metabolites, inflammatory cytokines, and pro-oncogenic integrals of fatty pancreas infiltration have not been fully explained.^{13,34–36} In our study, 16.1% of NAFLD had NASH, roughly based on elevated ALT, but no additional risk on presence of NAPS ($p=0.921$). In cases with steatosis, 19.2% had NAPS without NAFLD (Figure 2). As a multifactorial clinical benign entity, the co-existence of NAPS and NAFLD may also be the result of them

sharing similar risk factors. The main message of this study is the necessity of subgrouping the fatty pancreas as isolated NAPS and NAFLD+NAPS in future studies, since distinct differences in demographic, anthropometric, and biochemical features exist. Recent data suggest subgroups in NAFLD, too. NAFLD and metabolic associated fatty liver (MAFLD) cover different descriptions; for the latter, the presence of at least two metabolic abnormal components is mandatory.³⁹

This study has some limitations. First, US is highly operator-dependent, although the same radiologist evaluated the cases. Ultrasound has limitations in differentiating pancreatic steatosis and fibrosis, as both may reflect echogenic pancreas.¹⁵ Computed tomography, endoscopic US, magnetic resonance imaging, and MR-based proton density fat fraction are some of the preferred imaging modalities for more standardized data, according to some published reports on prospective studies.^{22,34,37,38} The included cases are patients admitted to the hospital; thus, they may not represent the overall population. Furthermore, ethnic, geographic, and nutritional comparisons were not performed. The study's retrospective design is another limitation, resulting in missing details about anthropometric, past history and viral serology data.

Conclusion

In this study, nonalcoholic pancreatic steatosis did not fully correlate with non-alcoholic fatty liver based on ultrasound, clinical, demographic, and anthropometric features. Despite severe pancreatic steatosis, more than a quarter of cases had completely normal liver echogenicity. In NAPS, compared

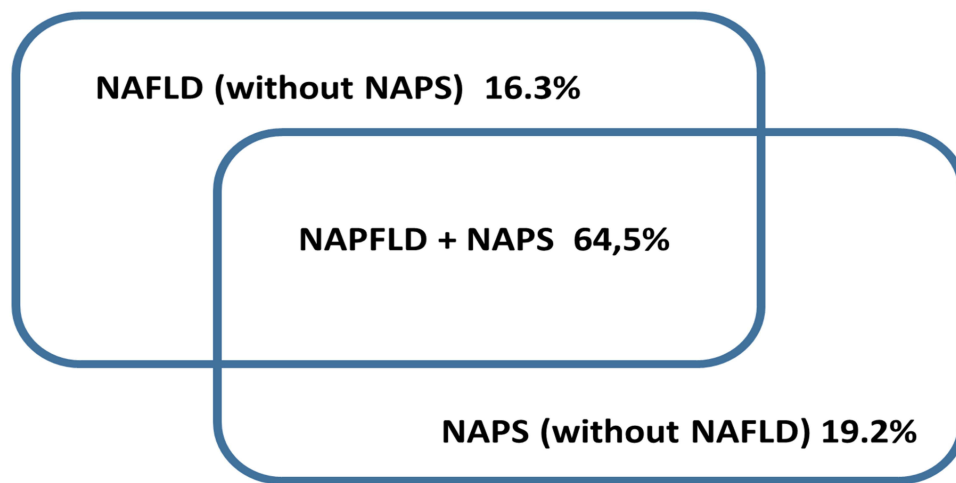


Figure 2 Venn diagram of NAPS and NAFLD.

Abbreviations: NAPS, Nonalcoholic pancreatic steatosis; NAFLD, Nonalcoholic fatty liver disease.

to NAFLD, insulin resistance and HDL-cholesterol were insignificantly higher, whereas BMI, total cholesterol, BFP, LDL-cholesterol, and triglycerides were lower (Figure 3).

The presence of a higher frequency of cholecystolithiasis in NAPS needs further investigation in a larger study. This inconsistency in >50% grading of liver and pancreatic

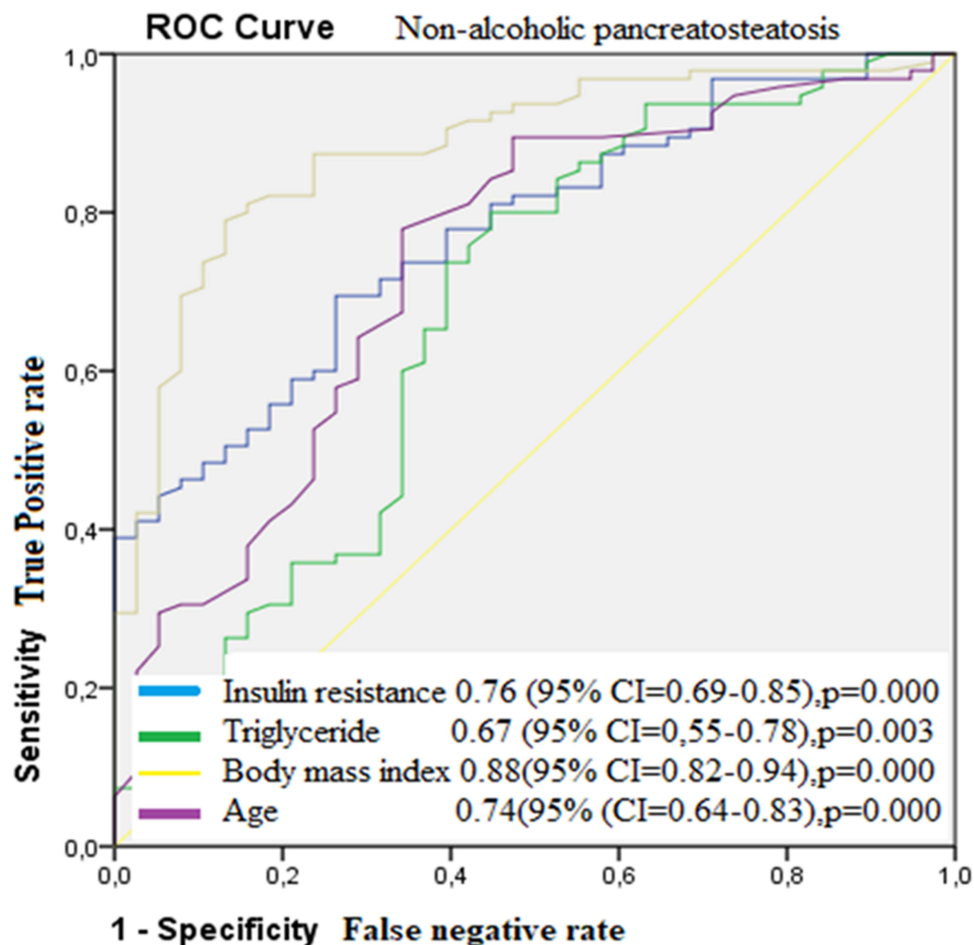


Figure 3 Receiver operating characteristic (ROC) curve for insulin resistance, triglyceride, body mass index, and age in nonalcoholic pancreatic steatosis area under the ROC curve.

steatosis may reflect different pathophysiological components of these clinical entities.

Disclosure

The authors reported no conflicts of interest in this work.

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