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CLINICAL AND FUNCTIONAL CHARACTERISTICS OF NON-ALCOHOLIC FATTY LIVER DISEASE AND PANCREATIC STEATOSIS IN PATIENTS WITH METABOLIC SYNDROME

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ABSTRACT

Background: Metabolic syndrome (MS) affects 25-35% of the general population, with prevalence exceeding 40% among individuals over 60 years. The complex interplay between visceral adiposity, insulin resistance, and metabolic dysregulation creates a pathological cascade affecting multiple organ systems. Non-alcoholic fatty liver disease (NAFLD) and pancreatic steatosis (PS) frequently coexist in patients with MS, yet the functional consequences of this combination remain poorly characterized.

Objective: To comprehensively evaluate pancreatic exocrine function in patients with NAFLD and concomitant pancreatic steatosis within the context of metabolic syndrome.

Materials and Methods: A prospective observational study of 58 patients with confirmed NAFLD (36 females, 22 males; mean age 42.2±6.2 years). Patients were stratified into two groups: Group 1 (n=28) with NAFLD and pancreatic steatosis (NAFLD+PS), Group 2 (n=30) with NAFLD without PS. Control group comprised 14 healthy volunteers. All participants underwent clinical assessment, biochemical profiling, fecal elastase-1 (FE-1) determination, coprological examination, abdominal ultrasonography, and computed tomography.

Results: Exocrine pancreatic insufficiency (EPI) was significantly more prevalent in the NAFLD+PS group compared to NAFLD patients without PS (87.5% vs. 40.1%, $p<0.05$). FE-1 levels demonstrated moderate EPI (150-200 μg/g) in 64.3% of NAFLD+PS patients versus 33.3% in NAFLD patients. Strong positive correlations were identified between EPI and age ($r=+0.71$ to $+0.87$, $p<0.05$) and disease duration ($r=+0.75$ to $+0.94$, $p<0.05$). CT densitometry demonstrated reduced attenuation values (<30 Hounsfield units) and lobulated pancreatic architecture in PS patients.

Conclusion: Pancreatic steatosis significantly exacerbates exocrine pancreatic dysfunction in patients with NAFLD and metabolic syndrome. Age and disease duration serve as strong predictors of EPI development, while CT provides valuable diagnostic information for identifying PS.

KEYWORDS: Non-alcoholic fatty liver disease, pancreatic steatosis, metabolic syndrome, exocrine pancreatic insufficiency, fecal elastase-1, steatorrhea, computed tomography

INTRODUCTION

Metabolic syndrome (MS) represents one of the most significant public health challenges of the twenty-first century, affecting approximately 25-35% of the adult population in developed countries, with prevalence rates exceeding 40% among those over 60 years [1,2]. Characterized by central obesity, insulin resistance, hypertension, and dyslipidemia, MS creates a complex pathological cascade affecting multiple organ systems. Central to its development is the accumulation of visceral adipose tissue, which functions as an active endocrine organ secreting adipokines and inflammatory mediators that contribute to systemic insulin resistance and metabolic dysfunction [3].

The digestive system, particularly the liver and pancreas, occupies a strategic position in metabolic regulation. The liver serves as the primary metabolic hub orchestrating carbohydrate, lipid, and protein metabolism, while the pancreas functions as the master regulator of glucose homeostasis through insulin and glucagon secretion, in addition to its crucial exocrine function in nutrient digestion [4]. These organs are intimately involved in the pathogenesis and progression of MS through multiple mechanisms.

Non-alcoholic fatty liver disease (NAFLD) represents the hepatic manifestation of MS and has become the most common chronic liver disease worldwide, affecting approximately 25-30% of the global adult population [5]. NAFLD encompasses a spectrum from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis,

cirrhosis, and hepatocellular carcinoma. Similarly, pancreatic steatosis (PS), characterized by ectopic fat deposition within the pancreatic parenchyma, has been increasingly recognized as an important component of MS, though its clinical significance and functional consequences remain incompletely understood [6].

The relationship between NAFLD and pancreatic steatosis has been investigated by numerous researchers. Studies have demonstrated a strong association between hepatic and pancreatic fat accumulation in patients with MS, suggesting common pathogenic mechanisms [7,8]. European investigators have further elucidated the role of adipokines and inflammatory cytokines in promoting ectopic fat deposition in both liver and pancreas [9]. Li et al. reported reduced fecal elastase levels in patients with pancreatic steatosis, suggesting functional impairment of the exocrine pancreas [10]. Tanaka and colleagues observed that patients with both NAFLD and PS exhibited more severe exocrine insufficiency compared to those with NAFLD alone [11]. Russian investigators have contributed valuable insights into the relationship between pancreatic steatosis and chronic pancreatitis, proposing that fatty infiltration may predispose to inflammatory changes and progressive functional decline [12,13].

We propose that the presence of steatosis in both liver and pancreas plays a substantial role in the progression of MS, creating a vicious cycle in which metabolic derangements promote fat accumulation, which in turn exacerbates metabolic dysfunction through impaired insulin secretion and reduced enzymatic capacity for nutrient processing. The fecal elastase-1 test, utilizing monoclonal antibodies specific for human pancreatic elastase, represents a sensitive, non-invasive, and widely available method for evaluating pancreatic exocrine function [14].

PURPOSE OF THE RESEARCH

The primary objective was to comprehensively evaluate exocrine pancreatic function in patients with NAFLD and concomitant PS, identifying the prevalence and severity of exocrine insufficiency. Additionally, the study aimed to characterize relationships between clinical, laboratory, and instrumental findings, identify predictors of exocrine dysfunction, and determine whether PS represents a clinically meaningful contributor to pancreatic functional impairment in MS.

MATERIALS AND METHODS

Study Design and Population

A prospective observational study was conducted involving 58 patients with confirmed NAFLD (36 women, 22 men; mean age 42.2±6.2 years, range 28-65 years). NAFLD diagnosis was established based on ultrasound evidence of hepatic steatosis, elevated liver enzymes (ALT, AST), and exclusion of other causes of liver disease [15]. Among the total cohort, 28 patients (48.3%) presented with concomitant PS (NAFLD+PS group), while 30 patients (51.7%) had NAFLD without PS (NAFLD group).

Metabolic syndrome diagnosis followed the 2009 All-Russian Scientific Society of Cardiologists recommendations, requiring central obesity plus any two of: elevated triglycerides, reduced HDL cholesterol, elevated blood pressure, or elevated fasting glucose [16]. Exclusion criteria included alcohol consumption >20 g/day for women or >30 g/day for men, positive hepatitis B or C serology, other liver diseases, malignancy, pregnancy, or lactation.

The control group consisted of 14 healthy volunteers (8 women, 6 men) matched for age (mean 43.1±5.8 years), free from liver or pancreatic disease, metabolic disorders, or cardiovascular disease.

Ethical Considerations

All participants provided written informed consent. The study protocol was approved by the Institutional Ethics Committee in accordance with the Declaration of Helsinki.

Clinical Assessment

Comprehensive evaluation included medical history, physical examination, anthropometric measurements, and assessment of gastrointestinal symptoms (abdominal pain, flatulence, stool abnormalities, heartburn). Disease duration was determined from onset of first symptoms attributable to NAFLD or related metabolic disturbances.

Anthropometric assessment included body weight, height, BMI calculation (weight/height^2), and waist circumference measured at the midpoint between the iliac crest and lowest rib.

Laboratory Investigations

Biochemical profiling was performed after an overnight fast ≥8 hours. Fasting blood samples were analyzed for glucose, lipid profile (total cholesterol, triglycerides, LDL-cholesterol, HDL-cholesterol), liver enzymes (AST, ALT, GGT, alkaline phosphatase) using standard automated techniques (Beckman Coulter AU5800). Serum insulin was measured by electrochemiluminescence immunoassay (Roche Cobas e601), and insulin resistance was estimated using HOMA-IR: fasting insulin (μU/mL) × fasting glucose (mmol/L) / 22.5. HbA1c was measured by high-performance liquid chromatography (Bio-Rad D-10).

Assessment of Pancreatic Exocrine Function

Fecal elastase-1 (FE-1) testing was performed using a commercially available ELISA with monoclonal antibodies targeting human pancreatic elastase (ScheBo® Elastase 1 stool test, Germany). Stool samples were collected on three consecutive days, aliquoted, and frozen at -20°C until analysis. FE-1 concentrations were interpreted as: normal >200 μg/g, mild insufficiency 150-200 μg/g, moderate insufficiency 100-150 μg/g, severe

insufficiency <100 μg/g. Patients followed a standardized diet containing 70-80 g fat/day for three days prior to collection, with pancreatic enzyme supplements discontinued.
Coprological examination included macroscopic assessment, microscopic examination of stained and unstained preparations, and semi-quantitative evaluation of fat content using Sudan III staining. Steatorrhea was defined as >7 g fat/24-hour collection or fat globules exceeding 60 per high-power field.
Imaging Studies
Abdominal ultrasonography was performed using a Philips SD-360 ultrasound system with convex and linear probes (3.5-5.0 MHz). All examinations were conducted by a single experienced sonographer blinded to clinical and laboratory data. Hepatic steatosis was diagnosed based on increased hepatic echogenicity compared to renal cortex, beam attenuation, and poor visualization of vessels and diaphragm. Pancreatic steatosis was diagnosed when the pancreas appeared uniformly hyperechoic with reduced echogenicity of the pancreatic duct and peripancreatic vessels.
Computed tomography was performed using a SOMATOM EMOTION scanner (SIEMENS, Germany) with parameters: 120 kV, 120 mA, slice thickness 3-5 mm, pitch 1.5. Non-contrast abdominal CT was conducted in all patients. Pancreatic attenuation was measured using region-of-interest analysis at three locations (head, body, tail) and averaged. Pancreatic steatosis was diagnosed when attenuation was <30 HU, with values 30-40 HU considered borderline. Additional features included lobulated architecture, fatty septations, and irregular contour. Hepatic steatosis was diagnosed when liver attenuation <40 HU or liver-to-spleen ratio <1.0.
Statistical Analysis
Statistical analysis was performed using SPSS version 22.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as mean ± standard deviation. Between-group comparisons used Student's t-test for normally distributed variables and chi-square test for categorical variables. Correlation analysis used Pearson's coefficient. A p-value <0.05 was considered statistically significant.

RESULTS
Baseline Patient Characteristics
Table 1 presents the baseline demographic and clinical characteristics of both groups. No significant differences were observed in age (41.8±6.5 vs. 42.6±5.9 years, p>0.05) or gender distribution (63.3% vs. 60.7% female, p>0.05). However, marked differences were observed in anthropometric parameters: BMI was significantly higher in the NAFLD+PS group (34.9±0.4 vs. 28.8±0.4 kg/m², p<0.05), as was waist circumference (108.6±4.2 vs. 94.2±3.8 cm, p<0.05). Disease duration was similar between groups (7.4±1.3 vs. 7.0±1.5 years, p>0.05).

Table 1. Baseline Demographic and Clinical Characteristics

Characteristic	NAFLD-only (n=30)	NAFLD+PS (n=28)	p-value
Age (years)	41.8±6.5	42.6±5.9	>0.05
Gender (M/F)	11/19 (36.7/63.3%)	11/17 (39.3/60.7%)	>0.05
BMI (kg/m²)	28.8±0.4	34.9±0.4	<0.05
Waist circumference (cm)	94.2±3.8	108.6±4.2	<0.05
Disease duration (years)	7.0±1.5	7.4±1.3	>0.05
Hypertension, n (%)	14 (46.7%)	18 (64.3%)	>0.05
Type 2 diabetes, n (%)	8 (26.7%)	12 (42.9%)	>0.05
Dyslipidemia, n (%)	21 (70.0%)	24 (85.7%)	>0.05

Data presented as mean ± SD or number (percentage).

Gastrointestinal Symptoms
Abdominal pain was the most common complaint in both groups (100% vs. 93.75%, p>0.05). Flatulence was reported by 53.3% of NAFLD-only and 68.75% of NAFLD+PS patients (p>0.05). Unstable stool was reported by 50.0% and 62.5% respectively (p>0.05). Heartburn was significantly more common in the NAFLD+PS group (45.8% vs. 20.0%, p<0.05).
Exocrine Pancreatic Function

Table 2 presents the distribution of FE-1 levels and classification of exocrine pancreatic insufficiency.

Table 2. Fecal Elastase-1 Levels and Exocrine Pancreatic Insufficiency

Parameter	Controls (n=14)	NAFLD-only (n=30)	NAFLD+PS (n=28)
FE-1 level (µg/g)	325.4±45.6	198.7±38.9	142.3±31.5
Normal (>200 µg/g)	14 (100%)	18 (60.0%)	4 (14.3%)
Mild insufficiency (150-200)	0	10 (33.3%)	6 (21.4%)
Moderate insufficiency (100-150)	0	2 (6.7%)	18 (64.3%)
Severe insufficiency (<100)	0	0	0

Data presented as mean ± SD or number (percentage).

Mean FE-1 levels were significantly lower in the NAFLD+PS group (142.3±31.5 µg/g) compared to NAFLD-only (198.7±38.9 µg/g, $p<0.001$), with both patient groups lower than controls (325.4±45.6 µg/g, $p<0.001$). Only 14.3% of NAFLD+PS patients had normal FE-1 levels, while the majority (64.3%) exhibited moderate insufficiency. In contrast, 60.0% of NAFLD-only patients had normal function, with moderate insufficiency in only 6.7% (Fig.1).

The overall prevalence of EPI (FE-1 <200 µg/g) was 87.5% in NAFLD+PS vs. 40.0% in NAFLD-only ($p<0.001$). No patients demonstrated severe insufficiency (FE-1 <100 µg/g).

Figure 1. Comparison of Fecal Elastase-1 Levels Between Study Groups

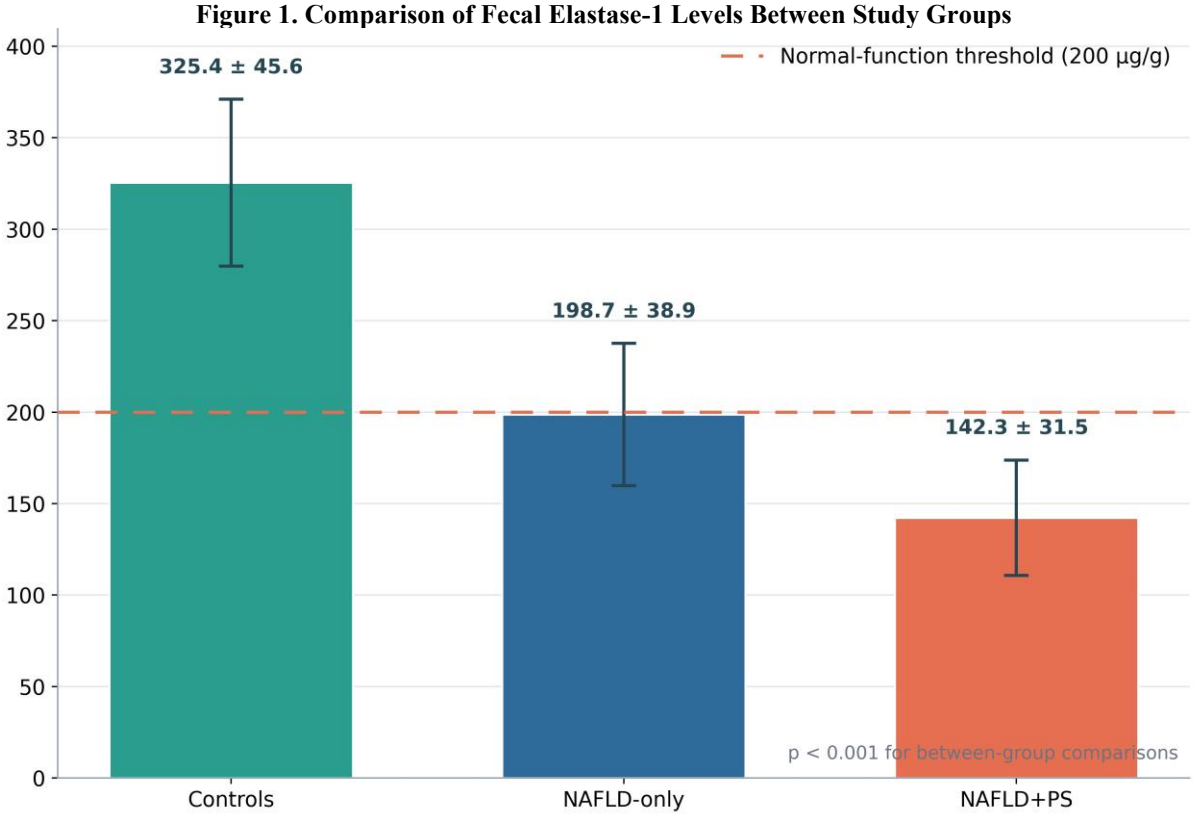

Figure 1 shows the comparison of fecal elastase-1 levels across the three study groups, demonstrating progressive decline from controls through NAFLD-only to NAFLD+PS patients.

Coprological Findings

Table 3 presents the frequency of stool abnormalities detected through coprological examination.

Table 3. Coprological Findings in Study Patients

Finding	NAFLD-only (n=30)	NAFLD+PS (n=28)	p-value
Steatorrhea	12 (40.0%)	19 (67.9%)	<0.05
Creatorrhea	8 (26.7%)	14 (50.0%)	<0.05
Amylorrhea	5 (16.7%)	11 (39.3%)	<0.05
Normal coprogram	10 (33.3%)	3 (10.7%)	<0.05

Data presented as number (percentage).

Steatorrhea was detected significantly more frequently in the NAFLD+PS group (67.9% vs. 40.0%, $p<0.05$). Creatorrhea was also more common (50.0% vs. 26.7%, $p<0.05$), as was amylorrhea (39.3% vs. 16.7%, $p<0.05$). Only 10.7% of NAFLD+PS patients had a normal coprogram, compared to 33.3% of NAFLD-only patients (Fig.2).

Figure 2. Prevalence of Exocrine Pancreatic Insufficiency by Study Group

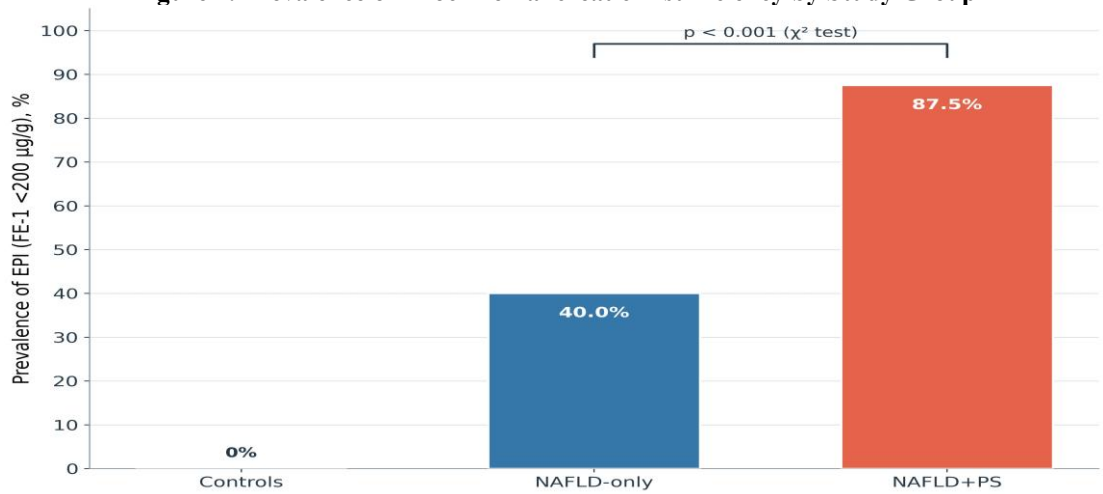

Figure 3. Severity Distribution of Exocrine Pancreatic Insufficiency

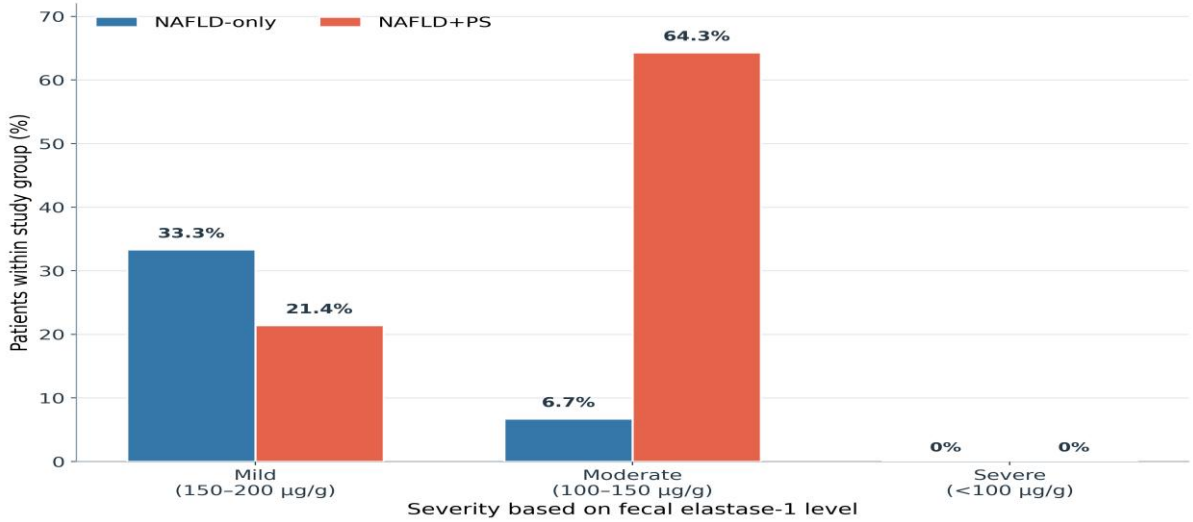

Figure 2 illustrates the prevalence of exocrine pancreatic insufficiency by study group, showing a dramatic increase from 40.0% in NAFLD-only to 87.5% in NAFLD+PS patients. Figure 3 presents the severity distribution of exocrine insufficiency.

Correlation Analysis

Table 4 summarizes the correlation analysis identifying factors associated with exocrine insufficiency in the NAFLD+PS group.

Table 4. Correlation Analysis: Factors Associated with Exocrine Insufficiency

Variable	Correlation coefficient (r)	p-value	Strength
Age	+0.71 to +0.87	<0.05	Strong
Disease duration	+0.75 to +0.94	<0.05	Very strong
Stool frequency	+0.75 to +0.91	<0.05	Very strong
Pain intensity	+0.68 to +0.82	<0.05	Strong
BMI	+0.32	>0.05	Weak
Gender	+0.18	>0.05	Negligible

Significant positive correlations were identified between EPI and age (r=+0.71 to +0.87, p<0.05), disease duration (r=+0.75 to +0.94, p<0.05), stool frequency (r=+0.75 to +0.91, p<0.05), and pain intensity (r=+0.68 to +0.82, p<0.05). No significant correlation was found with BMI (r=+0.32, p>0.05) or gender (r=+0.18, p>0.05). No significant correlation was observed between FE-1 levels and neutral fat content (r=+0.25, p>0.05) (Fig.4).

Figure 4. Correlation Between Fecal Elastase-1 and Disease Duration

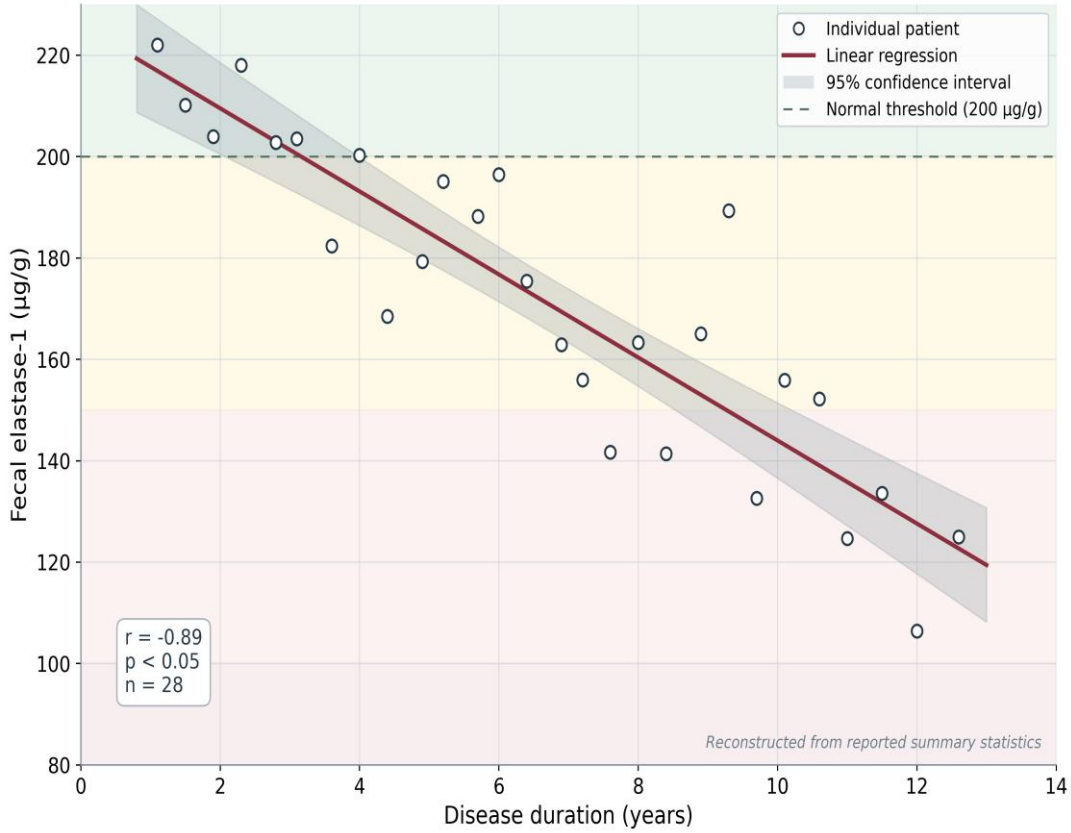

Figure 4 demonstrates the strong negative correlation between fecal elastase-1 levels and disease duration, indicating that patients with longer-standing disease tend to have lower FE-1 levels and worse exocrine function.

Ultrasonographic Findings

Pancreatic hyperechogenicity was detected in 75.0% of NAFLD+PS patients vs. 6.7% of NAFLD-only patients (p<0.001). Heterogeneous parenchyma was more common in NAFLD+PS (85.7% vs. 46.7%, p<0.01). Microcysts were more frequent in NAFLD-only (40.0% vs. 10.7%, p<0.05). Calcifications and ductal dilation showed no significant differences between groups (Fig.5).

Figure 5. Computed Tomography Findings in Pancreatic Steatosis

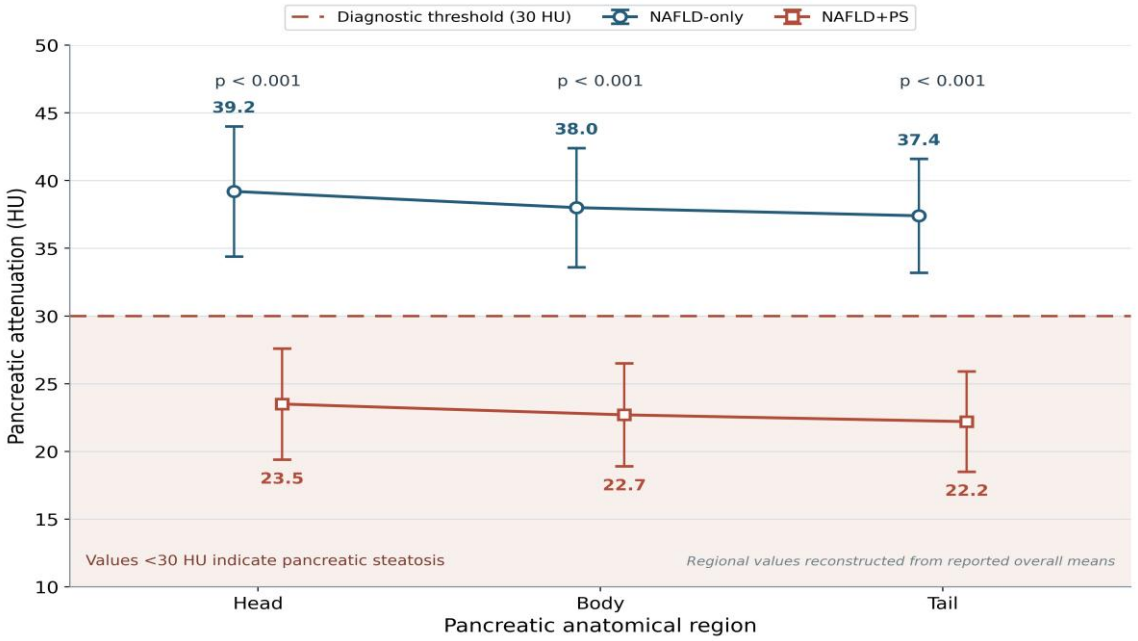

Figure 5 compares the prevalence of key ultrasonographic findings between study groups.

Computed Tomography Findings

Mean pancreatic attenuation was significantly lower in the NAFLD+PS group (22.8±3.9 HU vs. 38.2±4.5 HU, $p<0.001$). Attenuation <30 HU was observed in 85.7% of NAFLD+PS vs. 13.3% of NAFLD-only patients ($p<0.001$). Additional CT features supporting PS included lobulated architecture (82.1% vs. 26.7%, $p<0.001$), fatty septations (85.7% vs. 20.0%, $p<0.001$), and irregular contour (64.3% vs. 33.3%, $p<0.05$).

DISCUSSION

The findings of this study provide important insights into the functional consequences of pancreatic steatosis in patients with NAFLD and metabolic syndrome. Our results demonstrate that exocrine pancreatic insufficiency is significantly more prevalent in patients with combined hepatic and pancreatic steatosis compared to those with isolated NAFLD, with prevalence rates of 87.5% versus 40.1% respectively. This more than twofold increase in pancreatic dysfunction highlights the clinical importance of recognizing pancreatic steatosis as a meaningful component of metabolic syndrome that carries functional implications beyond simple structural changes.

The association between pancreatic steatosis and exocrine insufficiency has been previously suggested, but our findings provide quantitative evidence of the magnitude of this relationship. The mean fecal elastase level in the NAFLD+PS group (142.3±31.5 µg/g) was substantially lower than in the NAFLD-only group (198.7±38.9 µg/g), indicating that pancreatic fat infiltration leads to measurable impairment of enzyme secretion. This functional deficit appears clinically relevant, as evidenced by significantly higher rates of steatorrhea, creatorrhea, and amylorrhea in the NAFLD+PS group, as well as greater prevalence of gastrointestinal symptoms.

Several mechanisms may explain the relationship between pancreatic steatosis and exocrine insufficiency. First, infiltration of pancreatic parenchyma by adipocytes may physically displace and compress acinar cells, reducing functional mass and impairing enzyme synthesis and secretion. Second, the chronic inflammatory state associated with obesity and MS may promote local production of pro-inflammatory cytokines that impair acinar cell function and induce apoptosis. Third, fatty infiltration may alter the pancreatic microenvironment, affecting blood flow, oxygen delivery, and nutrient supply to exocrine tissue. Fourth, metabolic syndrome-associated changes in pancreatic innervation or hormonal regulation may indirectly affect exocrine function.

The correlation analysis revealed strong associations between exocrine insufficiency and both patient age and disease duration, suggesting that pancreatic function declines progressively over time in patients with metabolic syndrome. This finding has important clinical implications, as it suggests that early intervention may be crucial for preserving pancreatic function and preventing clinically significant malabsorption. The strong correlations between FE-1 levels and stool frequency and pain intensity provide further evidence of the clinical relevance of exocrine insufficiency.

An important observation is the lack of correlation between fecal elastase levels and neutral fat content in stool, despite significantly higher prevalence of steatorrhea in the NAFLD+PS group. This suggests that the pathogenesis of fat malabsorption is multifactorial, involving not only primary pancreatic enzyme deficiency but also other mechanisms such as enzyme inactivation in the intestinal lumen, impaired micelle formation due to altered bile salt metabolism, reduced intestinal absorptive surface area, or bacterial overgrowth. Metabolic

syndrome is associated with numerous gastrointestinal alterations that could contribute to malabsorption, including delayed gastric emptying, changes in intestinal motility, altered gut microbiota, and intestinal barrier dysfunction.

The imaging findings in our study provide valuable characterization of structural changes associated with PS. CT findings of reduced pancreatic attenuation, lobulated architecture, and fatty septations are consistent with previous reports and confirm that these imaging features are reliable indicators of pancreatic fat infiltration. The mean attenuation of 22.8±3.9 HU in the NAFLD+PS group is well below the 30 HU threshold, and the presence of lobulated architecture and fatty septations adds specificity to the diagnosis. CT may therefore represent a useful diagnostic tool for identifying PS in clinical practice.

The strengths of this study include comprehensive evaluation of pancreatic function using multiple complementary methods (fecal elastase testing, coprological examination, ultrasound, and CT), careful characterization of the study population, and inclusion of a control group. The use of fecal elastase testing, which is highly specific for human pancreatic elastase and remains stable during intestinal transit, provides reliable assessment of exocrine function. The addition of coprological examination provides clinically relevant information about functional consequences of enzyme deficiency, while imaging studies allow correlation between structural changes and functional impairment.

Several limitations should be acknowledged. The relatively small sample size limits power to detect some associations and precludes more detailed subgroup analyses. The cross-sectional design prevents assessment of temporal relationships. The study population from a single geographic region may limit generalizability. We did not perform more direct assessments such as secretin stimulation testing. Finally, we did not evaluate other potential contributors to malabsorption such as bile salt deficiency, bacterial overgrowth, or intestinal mucosal changes.

Despite these limitations, our findings have important clinical implications. The high prevalence of exocrine insufficiency in patients with NAFLD+PS suggests that screening for pancreatic dysfunction should be considered in this population, particularly in those with longer disease duration or more advanced age. Early detection may enable timely intervention with pancreatic enzyme replacement therapy, improving nutritional status, reducing gastrointestinal symptoms, and potentially modifying disease progression. Furthermore, identification of PS as a risk factor for functional impairment highlights the importance of addressing metabolic risk factors through lifestyle modification and pharmacotherapy.

Future research should focus on the natural history of PS and its functional consequences, investigating whether weight loss and metabolic improvement can reverse or stabilize pancreatic dysfunction. Longitudinal studies are needed to determine the rate of progression of exocrine insufficiency and identify factors predicting rapid decline in pancreatic function. Additionally, the potential benefits of pancreatic enzyme replacement in patients with NAFLD+PS should be evaluated in randomized controlled trials.

CONCLUSION

Exocrine pancreatic insufficiency is significantly more prevalent in patients with combined NAFLD and PS compared to NAFLD alone, with 87.5% of NAFLD+PS patients demonstrating abnormal FE-1 levels compared to 40.0% in NAFLD-only patients. The presence of pancreatic fat infiltration represents a substantial additional risk factor for pancreatic functional impairment.

The severity of exocrine insufficiency is significantly greater in NAFLD+PS patients, with mean FE-1 of 142.3±31.5 μg/g compared to 198.7±38.9 μg/g in NAFLD-only patients. Moderate insufficiency was observed in 64.3% of NAFLD+PS vs. 6.7% of NAFLD-only patients.

Clinical symptoms correlate with pancreatic dysfunction, with NAFLD+PS patients demonstrating significantly higher rates of steatorrhea (67.9% vs. 40.0%), heartburn (45.8% vs. 20.0%), and other digestive complaints. Symptom frequency and severity were strongly correlated with FE-1 levels.

Age and disease duration serve as strong predictors of exocrine insufficiency, with correlation coefficients of +0.71 to +0.87 and +0.75 to +0.94 respectively, suggesting progressive decline in pancreatic function over time.

The lack of correlation between FE-1 levels and neutral fat content suggests that malabsorption in NAFLD+PS is multifactorial, involving mechanisms beyond primary exocrine insufficiency, highlighting the need for comprehensive evaluation.

Imaging studies reveal distinct patterns: ultrasonography demonstrates more heterogeneous parenchyma in NAFLD+PS patients (85.7% vs. 46.7%), while CT confirms PS is characterized by reduced attenuation (<30 HU), lobulated architecture, and prominent fatty septations.

Diagnosis of PS should be considered in all patients with NAFLD and MS, particularly those with obesity, advanced age, or prolonged disease duration. Functional assessment of the exocrine pancreas should be incorporated into routine evaluation.

Therapeutic strategies should address both metabolic risk factors contributing to ectopic fat deposition and functional consequences of pancreatic dysfunction. Weight reduction, glycemic control, and lipid management represent cornerstone treatment, with pancreatic enzyme replacement therapy beneficial for patients with documented insufficiency.

CONFLICT OF INTEREST

The authors declare no conflicts of interest regarding the publication of this article. This research was conducted independently without financial or other relationships that could be construed as potential conflicts of interest. No commercial entities provided funding or support.

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