



Frequency of Fatty Liver in Patients Presenting with Acute Pancreatitis and Its Association with Disease Severity

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) may worsen the course of acute pancreatitis, yet evidence from regional settings is limited. **Objective:** To estimate the frequency of NAFLD among patients with acute pancreatitis and evaluate its association with radiological severity. **Methods:** A cross-sectional study was conducted in the department of Radiology, CMH Multan (June–December 2024). Consecutive adults (18–65 years) presenting within 24 hours and fulfilling clinical and biochemical criteria for acute pancreatitis underwent non-contrast abdominal computed tomography. NAFLD was defined by a liver-to-spleen attenuation ratio <1 with ≥ 10 Hounsfield Unit difference in individuals without alcohol use. Severity was graded using the CT Severity Index (CTSI). **Results:** Ninety-five patients were analyzed (61.1% male; mean age 42.3 ± 13.7 years); NAFLD was present in 41/95 (43.2%). Compared with non-NAFLD, NAFLD cases had higher body mass index (29.2 ± 4.1 vs 24.7 ± 3.8 kg/m²; $p < 0.001$) and more diabetes (41.5% vs 18.5%; $p = 0.014$) and hypertension (46.3% vs 22.2%; $p = 0.016$). Median amylase and lipase were higher in NAFLD (both $p < 0.001$). Severity shifted toward higher CTSI categories with NAFLD (mild 26.8% vs 68.5%, moderate 48.8% vs 27.8%, severe 24.4% vs 3.7%; $p < 0.001$); mean CTSI was 4.2 ± 2.8 vs 2.1 ± 1.9 ($p < 0.001$). NAFLD was associated with moderate-to-severe pancreatitis (OR 4.83; 95% CI 2.14–10.91) and severe pancreatitis (OR 8.37; 95% CI 1.79–39.14). Radiological complications were more frequent with NAFLD: necrosis 31.7% vs 11.1% ($p = 0.015$), peripancreatic fluid 58.5% vs 33.3% ($p = 0.018$), pseudocyst 19.5% vs 5.6% ($p = 0.037$). Findings were consistent across strata. **Conclusions:** NAFLD was common and associated with higher CTSI severity and local complications; routine CT assessment of hepatic steatosis may aid early risk stratification.

INTRODUCTION

The prevalence of fatty liver disease (FLD), a condition characterized by the accumulation of fat within the hepatic parenchyma, has seen a marked increase in recent decades [1]. Concurrently, acute pancreatitis (AP), an inflammatory condition of the pancreas, has also gained attention due to its rising incidence and potential for severe outcomes. While both diseases represent considerable burdens on healthcare systems globally, the relationship between them remains poorly understood. Specifically, the frequency of fatty liver disease in patients presenting with acute pancreatitis and its association with the severity of pancreatitis has not been adequately explored [2].

Acute pancreatitis is often classified based on its severity, which ranges from mild to moderate and severe.

The severity of the condition is determined by various factors such as systemic inflammation, organ failure, and local complications [3]. Fatty liver disease, on the other hand, is primarily diagnosed through imaging studies and can exist in various stages, from simple steatosis to the more severe form of non-alcoholic steatohepatitis (NASH) [4].

Evidence on the association between non-alcoholic fatty liver disease (NAFLD) and acute pancreatitis (AP) severity is mixed. In 199 AP cases (62.8% women; mean age 41 years), hepatic steatosis occurred in 36.5% and one quarter of these had severe AP [5]. In 128 cases, NAFLD prevalence was 43.8% and rose stepwise with severity: 25.0% in mild, 54.2% in moderately severe, and 71.4% in severe AP [6]. A larger series of 2,671 patients reported NAFLD in 17.97% and no significant APACHE II difference

by fatty liver status [7]. A 200-patient study found higher CRP and greater severity with NAFLD (33.5% prevalence) [8]. The literature offers conflicting findings regarding the relationship between non-alcoholic fatty liver disease (NAFLD) and acute pancreatitis (AP) severity. Some research indicates higher AP severity in patients with NAFLD, while others find no such association [6–8]. This divergence in findings underscores a significant research gap in understanding NAFLD's frequency and its potential impact on AP severity. To address these inconsistencies, the proposed study aims to explore NAFLD frequency in AP patients and assess its influence on the disease's severity. The research seeks to fill existing knowledge gaps, thereby aiding in prognostic evaluation.

MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Radiology, Combined Military Hospital (CMH) Multan. Data were collected over six months (June 2024 to December 2024). Consecutive non-probability sampling was used to enroll eligible patients as they presented. Ethical approval was obtained from the Institutional Ethics Review Committee (ERC No. 98/2023).

The sample size was calculated using the OpenEpi sample size calculator, assuming an anticipated frequency of fatty liver among patients with acute pancreatitis of 43.8%, a 95% confidence level, and a 10% margin of error; the required sample size was 95. All eligible patients aged 18–65 years, of either sex, who presented within 24 hours of symptom onset and met the diagnostic criteria for acute pancreatitis were considered for inclusion. Patients were excluded if medical history or records indicated other liver diseases (cirrhosis, alcoholic liver disease, or viral hepatitis), severe cardiac or pulmonary disease likely to influence acute pancreatitis severity, current pregnancy, or a documented history of chronic pancreatitis.

Operational definitions were prespecified. Non-alcoholic fatty liver disease (NAFLD; hepatic steatosis) was diagnosed on non-contrast computed tomography (CT) by a consultant radiologist in individuals with no history of alcohol consumption. Hepatic steatosis was defined as liver attenuation at least 10 Hounsfield Units (HU) lower than splenic attenuation, yielding a liver-to-spleen attenuation ratio <1 . Acute pancreatitis was defined by the presence of acute abdominal pain radiating to the back with a Visual Analog Scale (VAS) score ≥ 4 , together with serum amylase ≥ 3 times the upper limit of normal (≥ 140 U/L) and serum lipase ≥ 3 times the upper limit of normal (≥ 140 U/L). Disease severity was graded using the CT Severity Index (CTSI): mild (score 0–2, no pancreatic necrosis), moderately severe (score 3–6, peripancreatic inflammation or fluid collections), and severe (score 7–10, extensive inflammation and/or necrosis). Hypertension was defined as systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg on two separate measurements under controlled conditions. Type 2 diabetes mellitus was diagnosed if any of the following were present: fasting plasma glucose ≥ 126 mg/dL (7.0 mmol/L), glycated hemoglobin (HbA1c) $\geq 6.5\%$, 2-hour oral glucose tolerance test value ≥ 200 mg/dL (11.1 mmol/L), or random plasma glucose ≥ 200 mg/dL (11.1 mmol/L) in the presence of classic hyperglycemic

symptoms. Obesity was defined as body mass index (BMI) ≥ 30 kg/m². A smoker was defined as an individual using tobacco daily (cigarettes, cigars, or pipe) for at least six months.

Data collection followed a standardized pathway. Patients arriving at the emergency department with suspected acute pancreatitis were assessed by the attending team and referred to radiology for imaging. Written informed consent was obtained prior to enrollment. Missing essential clinical information (symptom history, VAS score) or laboratory results (serum amylase and lipase) were obtained at this stage according to the hospital's standard protocol. Non-contrast abdominal CT was performed as per institutional practice. A consultant radiologist, blinded to study objectives, reviewed CT scans to document liver attenuation, spleen attenuation, liver-to-spleen ratio, and pancreatic findings. The presence or absence of NAFLD and the CTSI score were recorded on a predesigned proforma along with demographics, BMI, blood pressure, diabetes status, hypertension status, and smoking history. All records were kept confidential and stored in restricted-access files.

Statistical analysis was performed using IBM SPSS Statistics, version 26. Continuous variables (e.g., age, serum amylase, serum lipase) were assessed for normality using the Shapiro–Wilk test. Normally distributed variables were summarized as mean \pm standard deviation, and non-normally distributed variables as median with interquartile range. Categorical variables (e.g., sex, NAFLD status, CTSI category, obesity, diabetes, hypertension, smoking) were presented as frequencies and percentages. The association between NAFLD and acute pancreatitis severity (CTSI categories) was evaluated using the Chi-square test with a two-sided significance level of $p < 0.05$. Stratified analyses were conducted by age, sex, obesity, diabetes, hypertension, and smoking status to examine effect consistency; post-stratification Chi-square tests were applied to assess the relationship between NAFLD and severity within strata.

RESULTS

The study population comprised 58 males (61.1%) and 37 females (38.9%), with a mean age of 42.3 ± 13.7 years (range 18–65 years). Demographic analysis revealed that 41 patients (43.2%) had non-alcoholic fatty liver disease (NAFLD) as diagnosed by CT abdomen, while 54 patients (56.8%) did not have fatty liver disease. Baseline clinical characteristics demonstrated significant differences between patients with and without fatty liver disease. The mean age was comparable between groups (NAFLD: 44.1 ± 12.8 years vs. non-NAFLD: 40.9 ± 14.2 years, $p = 0.264$). Male gender predominance was more pronounced in the NAFLD group (68.3% vs. 55.6%, $p = 0.223$). Patients with NAFLD exhibited significantly higher body mass index (29.2 ± 4.1 kg/m² vs. 24.7 ± 3.8 kg/m², $p < 0.001$) and increased prevalence of obesity (73.2% vs. 29.6%, $p < 0.001$). Type 2 diabetes mellitus was more frequent in NAFLD patients (41.5% vs. 18.5%, $p = 0.014$), as was hypertension (46.3% vs. 22.2%, $p = 0.016$). Smoking history showed no significant difference between groups (24.4% vs. 20.4%, $p = 0.643$).

Table 1
Baseline Characteristics of Study Population

Characteristic	Total (n=95)	NAFLD (n=41)	Non-NAFLD (n=54)	p-value
Age (years), mean \pm SD	42.3 \pm 13.7	44.1 \pm 12.8	40.9 \pm 14.2	0.264
Male gender, n (%)	58 (61.1)	28 (68.3)	30 (55.6)	0.223
BMI (kg/m ²), mean \pm SD	26.7 \pm 4.6	29.2 \pm 4.1	24.7 \pm 3.8	<0.001*
Obesity (BMI \geq 30), n (%)	46 (48.4)	30 (73.2)	16 (29.6)	<0.001*
Type 2 DM, n (%)	27 (28.4)	17 (41.5)	10 (18.5)	0.014*
Hypertension, n (%)	31 (32.6)	19 (46.3)	12 (22.2)	0.016*
Smoking history, n (%)	21 (22.1)	10 (24.4)	11 (20.4)	0.643

*p < 0.05 considered statistically significant

Laboratory investigations revealed significant differences in biochemical markers between patients with and without fatty liver disease. Serum amylase levels were significantly elevated in NAFLD patients compared to non-NAFLD patients (median 385 U/L [IQR: 312-468] vs. 298 U/L [IQR: 245-356], $p < 0.001$). Similarly, serum lipase levels demonstrated marked elevation in the NAFLD group (median 412 U/L [IQR: 341-495] vs. 321 U/L [IQR: 267-389], $p < 0.001$). Pain severity, as assessed by Visual Analog Scale scores, showed comparable intensity between groups (NAFLD: 6.8 ± 1.4 vs. non-NAFLD: 6.5 ± 1.6 , $p = 0.287$).

Table 2
Laboratory Parameters and Clinical Presentation

Parameter	NAFLD (n=41)	Non-NAFLD (n=54)	p-value
Serum amylase (U/L), median (IQR)	385 (312-468)	298 (245-356)	<0.001*
Serum lipase (U/L), median (IQR)	412 (341-495)	321 (267-389)	<0.001*
VAS pain score, mean \pm SD	6.8 \pm 1.4	6.5 \pm 1.6	0.287

*p < 0.05 considered statistically significant

The overall frequency of non-alcoholic fatty liver disease among patients with acute pancreatitis was 43.2% (41/95 patients), with a 95% confidence interval of 33.1% to 53.7%. Non-alcoholic fatty liver disease (NAFLD) was identified in 43.2% (41/95; 95% CI 33.1–53.7), closely matching the anticipated 43.8%. On CT, the liver-to-spleen attenuation ratio was lower in NAFLD than non-NAFLD (0.87 ± 0.09 vs 1.24 ± 0.15 ; $p < 0.001$), supporting the imaging definition. Severity by CT Severity Index differed significantly (χ^2 , $p < 0.001$), with NAFLD showing fewer mild cases and more advanced disease: mild 26.8% vs 68.5%, moderately severe 48.8% vs 27.8%, and severe 24.4% vs 3.7%.

Table 3
Distribution of Acute Pancreatitis Severity by CTSI Score

Severity Category	Total (n=95)	NAFLD (n=41)	Non-NAFLD (n=54)	Chi-square	p-value
Mild (CTSI 0-2)	48 (50.5%)	11 (26.8%)	37 (68.5%)	21.47	<0.001*
Moderate (CTSI 3-6)	33 (34.7%)	20 (48.8%)	15 (27.8%)		
Severe (CTSI 7-10)	14 (14.7%)	10 (24.4%)	2 (3.7%)		

*p < 0.05 considered statistically significant

Non-alcoholic fatty liver disease (NAFLD) was associated with substantially greater radiologic severity of acute pancreatitis. Mean CT Severity Index (CTSI) was higher in NAFLD than non-NAFLD (4.2 ± 2.8 vs 2.1 ± 1.9 ; $p < 0.001$). The association between NAFLD presence and CTSI category was significant ($\chi^2 = 21.47$; $df = 2$; $p < 0.001$), with Cramer's $V = 0.475$ indicating a moderate–strong effect size. In risk analyses, NAFLD increased the odds of moderate-to-severe disease (OR=4.83; 95% CI 2.14–10.91; $p < 0.001$) and of severe disease specifically (OR=8.37; 95% CI 1.79–39.14; $p = 0.007$). Findings were robust across strata: age ≤ 45 years ($p = 0.002$) and > 45 years ($p = 0.018$); male sex ($p = 0.001$) and female sex ($p = 0.045$). This pattern indicates a moderate–strong association at the population level and a markedly elevated individual risk for severe presentations among those with hepatic steatosis, supporting its value for risk stratification in clinical practice.

Table 4
Stratified Analysis of NAFLD and AP Severity Association

Stratification Variable	Subgroup	NAFLD with Severe AP (%)	p-value
Age	≤ 45 years (n=52)	8/22 (36.4%)	0.002*
	> 45 years (n=43)	2/19 (10.5%)	0.018*
Gender	Male (n=58)	7/28 (25.0%)	0.001*
	Female (n=37)	3/13 (23.1%)	0.045*
Obesity	BMI ≥ 30 (n=46)	8/30 (26.7%)	0.003*
	BMI < 30 (n=49)	2/11 (18.2%)	0.021*
Diabetes	Present (n=27)	5/17 (29.4%)	0.012*
	Absent (n=68)	5/24 (20.8%)	0.006*
Hypertension	Present (n=31)	4/19 (21.1%)	0.025*
	Absent (n=64)	6/22 (27.3%)	0.002*

*p < 0.05 considered statistically significant

Associations were strongest in comorbid subgroups. Among obese patients (BMI ≥ 30 kg/m²), NAFLD remained linked to severe acute pancreatitis ($p = 0.003$); significance also persisted in diabetes ($p = 0.012$) and hypertension ($p = 0.025$). CT findings showed higher complication burdens with NAFLD: pancreatic necrosis 31.7% vs 11.1% ($p = 0.015$), peripancreatic fluid collections 58.5% vs 33.3% ($p = 0.018$), and pancreatic pseudocysts 19.5% vs 5.6% ($p = 0.037$). The composite endpoint of any radiological complication occurred more frequently with NAFLD than without (73.2% vs 42.6%; $p = 0.003$).

Table 5
Radiological Complications in Acute Pancreatitis

Complication	NAFLD (n=41)	Non-NAFLD (n=54)	OR (95% CI)	p-value
Pancreatic necrosis	13 (31.7%)	6 (11.1%)	3.71 (1.29-10.65)	0.015*
Peripancreatic fluid collections	24 (58.5%)	18 (33.3%)	2.83 (1.26-6.35)	0.018*
Pancreatic pseudocysts	8 (19.5%)	3 (5.6%)	4.11 (1.06-15.92)	0.037*
Splenic vein thrombosis	3 (7.3%)	1 (1.9%)	4.17 (0.42-41.35)	0.225

*p < 0.05 considered statistically significant

DISCUSSION

The present study identified non-alcoholic fatty liver disease (NAFLD) in 43.2% of patients with acute pancreatitis, a frequency closely aligned with reports from

Jia et al. (43.8%), intermediate between Lazcano Becerra et al. (36.5%) and Yoon et al. (33.5%), and lower than the 49.7% reported by Liu et al., supporting external consistency of NAFLD prevalence among acute pancreatitis presentations across settings [5,6,8,9]. The demographic pattern of higher male proportion and greater adiposity in the NAFLD group paralleled prior observations, with higher body mass index and obesity enrichment mirroring pooled effects in meta-analyses and large observational series [10–12].

Disease severity was markedly greater in individuals with NAFLD, with severe acute pancreatitis in 24.4% versus 3.7% of those without steatosis and a two-point higher mean CT Severity Index (4.2 ± 2.8 vs 2.1 ± 1.9), findings concordant with multiple datasets showing a shift from mild to moderate/severe categories when hepatic steatosis is present [6,9,12]. The magnitude of association in the current analysis (odds ratio [OR] 4.83 for moderate-to-severe disease; OR 8.37 for severe disease) is directionally consistent with pooled estimates from meta-analyses showing increased odds for moderately severe and severe acute pancreatitis (e.g., OR 2.66–3.57) and lower odds of mild disease (OR ~ 0.28 – 0.32), although the present effect sizes are larger—likely reflecting case-mix, imaging-based NAFLD definition, and single-center selection factors [10,11]. While most comparative studies report higher severity with steatosis, heterogeneity persists; one large retrospective series noted no significant difference in APACHE II ranges between fatty and non-fatty liver groups, underscoring variation in severity metrics across scoring systems and populations [7].

Complication profiles reinforced the severity signal. Pancreatic necrosis occurred in 31.7% of NAFLD cases versus 11.1% in non-NAFLD, echoing Liu et al., where necrosis was more frequent with steatosis (23.4% vs 10.5%), and aligning with pooled odds for necrosis (OR ~ 3.05) and for local complications such as peripancreatic fluid collections and pseudocysts that were likewise elevated in the present series [9,11]. The significantly higher composite of any radiological complication among NAFLD patients (73.2% vs 42.6%) is concordant with meta-analytic evidence showing greater local and systemic complications, persistent systemic inflammatory response syndrome, and organ failure in steatotic liver states [10,11].

Biochemical indices demonstrated higher serum amylase and lipase in NAFLD, suggesting greater pancreatic injury at presentation; although most comparative literature emphasizes inflammatory biomarkers (e.g., higher C-reactive protein at admission and peak values in steatosis), the direction of effect is consistent with a more intense inflammatory burden reported by Jia et al. and at meta-analysis level [6,11]. Length of stay and mortality could not be assessed here, but prior prospective and pooled analyses indicate prolonged hospitalization and higher mortality in steatosis, with prospective data identifying fatty liver as an independent predictor of moderate–severe disease, local complications, and organ failure after adjustment for age and sex [10,13,14].

Subgroup and stratified analyses in this study—demonstrating consistent associations across age, sex, obesity, diabetes mellitus, and hypertension—support an effect of NAFLD beyond simple confounding by metabolic comorbidity, mirroring meta-analytic subgroup results that showed the association persisting in both obese and non-obese strata (Jiang et al., 2022). Mechanistically, hepatic steatosis may amplify systemic inflammation and alter lipid metabolism, predisposing to hypertriglyceridemia-related injury, sustained systemic inflammatory response syndrome, and organ dysfunction—an inference compatible with elevated rates of persistent systemic inflammatory response syndrome and organ failure reported in steatosis [4,12,14].

Critical appraisal highlights several considerations. First, imaging criteria were standardized via liver-to-spleen attenuation ratio differences on computed tomography (0.87 ± 0.09 vs 1.24 ± 0.15), enhancing internal validity and comparability to studies using similar thresholds; however, variable diagnostic modalities across the literature (ultrasonography vs computed tomography, non-contrast vs contrast protocols) may contribute to effect-size heterogeneity [6,9,11]. Second, the cross-sectional design precludes causal inference and cannot exclude residual confounding by etiological factors (e.g., gallstones, alcohol, hypertriglyceridemia), which have shown differential associations with steatosis and disease course; studies stratified by etiology and employing multivariable adjustment strengthen inference and should be emulated in future work [10–12]. Third, severity grading was based on CT Severity Index here, whereas several comparators used the Revised Atlanta classification, Modified CT Severity Index, or APACHE II; although all show a consistent directional association, differences in grading frameworks limit direct numeric comparisons [9,13]. Finally, single-center sampling may restrict generalizability compared with large multicenter or registry-based analyses that reported broader etiological spectra and outcome capture, including mortality [3,15].

Overall, convergence across this analysis and prior prospective, retrospective, and pooled evidence supports hepatic steatosis as a clinically relevant marker of increased acute pancreatitis severity and complications, with consistency across demographic strata and imaging-based definitions, while acknowledging inter-study heterogeneity and the need for etiologically stratified, multivariable designs to refine risk estimates and causal pathways.

CONCLUSION

Non-alcoholic fatty liver disease was common among patients presenting with acute pancreatitis and was linked to greater radiological severity and more local complications, including necrosis, peripancreatic fluid collections, and pseudocyst formation. The association remained evident across age, sex, and key metabolic comorbidities, indicating a risk signal beyond confounding by obesity, diabetes, or hypertension. Routine assessment of hepatic steatosis on computed tomography can aid early risk stratification and clinical monitoring. The cross-sectional design and single-center setting limit causal

inference and generalizability. Prospective, etiologically stratified studies are warranted to clarify mechanisms and

to test whether targeted metabolic optimization mitigates adverse outcomes clinically.

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