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Prevalence and clinical correlates of non-alcoholic fatty liver disease in type 2 diabetes mellitus: A cross-sectional study

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Abstract:
Non-alcoholic fatty liver disease (NAFLD) is highly prevalent among patients with type 2 diabetes mellitus (T2DM), contributing significantly to increased morbidity and metabolic complications. Therefore, it is of interest to determine the prevalence of NAFLD in T2DM patients and compare demographic, clinical and biochemical characteristics between those with and without NAFLD. A cross-sectional study of 100 diabetic patients was conducted using ultrasonography for NAFLD diagnosis, alongside evaluation of clinical parameters and laboratory investigations. NAFLD was present in 42% of patients and was significantly associated with female gender, obesity, poor glycemic control, diabetic complications and abnormal lipid and liver profiles. Thus, data shows that emphasizing the strong clinical association between NAFLD and T2DM, supporting routine screening and integrated management strategies to reduce disease burden.

Keywords: Diabetes mellitus (DM), dyslipidemia, fatty liver, glycemic control, obesity

Background:
The liver, as the largest metabolic organ of the human body, plays a central role in maintaining metabolic homeostasis. It performs multiple functions including nutrient metabolism, synthesis of plasma proteins and clotting factors, glycogen storage and bile production **[1]**. Due to its unique anatomical position, the liver receives most of the blood from the gastrointestinal tract through the portal circulation, placing it at the forefront of nutrient processing and metabolic regulation **[2]**. Liver diseases constitute a significant global health burden. Among liver diseases, non-alcoholic fatty liver disease (NAFLD), recently renamed metabolic dysfunction-associated steatotic liver disease (MASLD), has emerged as the leading cause of chronic liver disease worldwide **[3]**. The prevalence of NAFLD in the general population is estimated at 38%, having increased by 50% over the past three decades. NAFLD encompasses a spectrum of liver conditions ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), which can progress to cirrhosis and hepatocellular carcinoma **[4]**. The global diabetes epidemic has paralleled the rise in NAFLD. The International Diabetes Federation estimated that there were 537 million people with diabetes mellitus globally in 2021, projected to increase to 643 million by 2030 and 783 million by 2045 **[5]**. High body mass index (BMI) is a major driver of this burden, with obesity affecting over 890 million adults worldwide in 2022. The highly accelerated incidence of type 2 diabetes mellitus (T2DM) has been driven by excessive calorie intake and physical inactivity, resulting in obesity and ectopic fat accumulation in organs such as the liver **[6]**. The association between T2DM and NAFLD is well established and bidirectional. Recent meta-analyses report that the prevalence of NAFLD among patients with T2DM ranges from 55% to 70%. Conversely, the pooled global prevalence of type 2 diabetes among NAFLD patients is 28.3%, with an incidence density of 24.6 per 1000 person-years **[7]**. The presence of NAFLD among individuals with T2DM is associated with a 2.2-fold increase in all-cause mortality **[8]** and patients with both conditions face accelerated progression to fibrosis, cirrhosis and hepatocellular carcinoma **[9]**. Despite the well-documented relationship between T2DM and NAFLD, liver disease is often overlooked as a diabetic complication. The American Diabetes Association's 2025 Standards of Care now emphasize screening for metabolic dysfunction-associated steatotic liver disease (MASLD) in adults with type 2 diabetes, particularly those at high risk for liver fibrosis **[10]**. Recent evidence show that even mild deterioration in glycemic control, with HbA1c levels of 6.5-7.4%, contributes to progression of NAFLD activity and fibrosis **[11]**. Therefore, it is of interest to assess the prevalence of NAFLD among patients with T2DM attending a tertiary care hospital and to compare demographic, clinical and laboratory parameters between diabetic patients with and without NAFLD to establish possible correlations.

Materials and Methods:
This observational cross-sectional study was conducted at the Department of Medicine in collaboration with the Department of Gastroenterology, Vivekananda Polyclinic and Institute of Medical Sciences, Lucknow, from July 2013 to June 2014. The study was approved by the Institutional Ethics Committee and informed consent was obtained from all participants. The study population comprised patients with T2DM attending the outpatient departments of Medicine and Gastroenterology. Sample size was calculated based on a predicted NAFLD prevalence of 32% using the formula $n = z^2 [(p (1-p))/e^2]$, where p represents the sample proportion, e is the error allowance (0.1 at 10% allowance) and z is the constant at 95% confidence level (1.96). The calculated sample size was 84 patients; accounting for a 20% dropout rate, we enrolled 100 patients using random sampling. Inclusion criteria were: (i) patients with T2DM, (ii) age >18 years and (iii) either gender. Exclusion criteria included: (i) known chronic liver disease (HBsAg or anti-HCV positive), (ii) pregnancy, (iii) alcohol consumption or use of drugs causing fatty liver or deranged liver function and (iv) history of jejunoileal bypass surgery. After obtaining informed consent, eligible patients were enrolled and assigned unique identification numbers to maintain confidentiality. Comprehensive data were collected including demographic information (age, gender, residence), medical history, diabetes duration, family history of diabetes, presence of diabetic

complications (retinopathy, nephropathy, neuropathy, diabetic foot), comorbidities (hypertension, cardiac disease), personal habits (smoking, tobacco use) and dietary preferences. Clinical examination included anthropometric measurements (height, weight, waist circumference) and body mass index (BMI) calculation. Patients were assessed for clinical features suggestive of liver disease including fatigability, anorexia, icterus, right hypochondrial tenderness and abdominal distension. Blood samples were collected after overnight fasting for the following investigations: (i) glycemic control: fasting blood glucose, postprandial blood glucose and glycosylated hemoglobin (HbA1c); (ii) liver function tests: serum bilirubin (total, direct, indirect), aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP); (iii) lipid profile: total cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL); and (iv) renal function: serum creatinine. Abdominal ultrasonography was performed to diagnose and grade NAFLD. Fatty liver was identified as a 'bright liver' with ultrasonographic contrast between hepatic and renal parenchyma, vessel blurring and narrowing of hepatic vein lumen, in the absence of findings suggestive of chronic liver disease. Ultrasonography has showed good diagnostic accuracy for detecting moderate-to-severe hepatic steatosis, with sensitivity of 78-92% and specificity of 85-95%. NAFLD grading was performed based on standard ultrasonography criteria: Grade 1 (mild steatosis) - slightly increased liver echogenicity with normal vessels and absent posterior attenuation; Grade 2 (moderate steatosis) - moderately increased liver echogenicity with partial dimming of vessels and early posterior attenuation; Grade 3 (severe steatosis) - diffusely increased liver echogenicity with absence of visible vessels and heavy posterior attenuation. Data were analyzed using appropriate statistical methods. Categorical variables were compared using chi-square test. Continuous variables were expressed as mean ± standard deviation and compared using Student's t-test (for two groups) or analysis of variance (ANOVA) (for more than two groups). A p-value <0.05 was considered statistically significant. All analyses were performed using standard statistical software.

Results:

Of the 100 patients with T2DM enrolled in the study, 42 (42%) had NAFLD on ultrasonography, while 58 (58%) did not have NAFLD. Among patients with NAFLD, 18 (18%) had Grade I disease, 18 (18%) had Grade II disease and 6 (6%) had Grade III disease (**Table 1).** The study population consisted of 65 males (65%) and 35 females (35%). The majority of subjects (90%) were aged >40 years and 77% were from urban areas. No significant association was found between age or residence and NAFLD prevalence (p=0.137 and p=0.870, respectively). However, female gender showed a significant association with NAFLD. Among 35 female subjects, 20 (57.1%) had NAFLD compared to 22 (33.8%) of 65 male subjects (χ^2=5.069, p=0.024). When analyzing NAFLD severity, female gender was significantly associated with higher grades of NAFLD (χ^2=11.069, p=0.011), with Grade II NAFLD showing the highest female predominance (66.7%) (**Table 2**). Although a higher proportion of NAFLD patients had diabetes duration >5 years (69.05% vs 53.45%) and family history of diabetes (73.81% vs 60.34%), these differences were not statistically significant (p=0.116 and p=0.161, respectively). Patients with NAFLD had significantly higher rates of diabetic complications and comorbidities compared to those without NAFLD (**Table 3**). The severity of NAFLD correlated with the presence of these complications. Personal habits including smoking (22% vs 19%, p=0.428) and tobacco use (35.71% vs 17.24%, p=0.081) showed no significant association with NAFLD prevalence. Similarly, dietary preferences (vegetarian vs non-vegetarian) were not significantly associated with NAFLD (p=0.157). Anthropometric parameters showed strong associations with NAFLD (**Table 4**). All obese patients (BMI >30 kg/m²) had NAFLD (100%). Among the study population, 30.95% of NAFLD patients were obese (BMI 30-35 kg/m²) compared to 0% in the non-NAFLD group. Higher grades of obesity were associated with higher grades of NAFLD (χ^2=47.992, p<0.001). Clinical features suggestive of liver disease were more prevalent in NAFLD patients. Fatigability was the most characteristic finding, present in 61.9% of NAFLD patients compared to 20.69% of non-NAFLD patients (χ^2=17.564, p<0.001). Anorexia (16.67% vs 1.72%, χ^2=7.390, p=0.007) and abdominal distension (9.52% vs 0%, χ^2=5.754, p=0.016) were also significantly more common in NAFLD patients. Fatigability and anorexia increased with higher NAFLD grades (p<0.001 and p=0.015, respectively). Mean HbA1c levels were significantly higher in NAFLD patients compared to non-NAFLD patients (8.22±1.17% vs 7.59±1.70%, t=-2.801, p=0.040). When analyzed by NAFLD severity, mean HbA1c levels progressively increased: Grade I (7.93±1.45%), Grade II (8.42±0.97%) and Grade III (8.50±0.58%), although this trend did not reach statistical significance (F=1.824, p=0.148). Liver function tests and lipid profile showed significant abnormalities in NAFLD patients (**Table 5**). All parameters showed significant associations with NAFLD severity.

Table 1: Distribution of NAFLD grades in study population

NAFLD Status	Number (n)	Percentage (%)
No NAFLD (Grade 0)	58	58
Grade I NAFLD (Mild)	18	18
Grade II NAFLD (Moderate)	18	18
Grade III NAFLD (Severe)	6	6

Table 2: Demographic characteristics of study participants

Variable	No NAFLD (n=58)	NAFLD (n=42)	p-value
Age >40 years, n (%)	50 (86.2)	40 (95.2)	0.137
Female gender, n (%)	15 (25.9)	20 (47.6)	0.024*
Urban residence, n (%)	45 (77.6)	32 (76.2)	0.87

**p<0.05 considered statistically significant*

Table 3: Diabetic complications and comorbidities in study participants

Complication	No NAFLD n (%)	NAFLD n (%)	p-value
Hypertension	19 (32.8)	34 (81.0)	<0.001*
Cardiac morbidity	11 (19.0)	21 (50.0)	0.001*
Neuropathy	6 (10.3)	25 (59.5)	<0.001*
Retinopathy	5 (8.6)	12 (28.6)	0.009*
Nephropathy	6 (10.3)	12 (28.6)	0.019*
Diabetic foot	2 (3.4)	12 (28.6)	<0.001*

**p<0.05 considered statistically significant*

Table 4: Anthropometric measurements in study participants

Parameter	No NAFLD (n=58)	NAFLD (n=42)	p-value
Body weight (kg)	61.2 ± 10.5	72.5 ± 9.4	<0.001*
Waist circumference (cm)	86.4 ± 9.2	98.7 ± 8.5	<0.001*
BMI (kg/m²)	23.8 ± 3.9	28.2 ± 4.1	<0.001*
Obesity (BMI >30), n (%)	0 (0)	13 (31.0)	<0.001*

Values presented as mean ± SD or n (%).
**p<0.05 considered statistically significant*
BMI: Body mass index

Table 5: Biochemical parameters in study participants

Parameter	No NAFLD (n=58)	NAFLD (n=42)	p-value
Liver Function Tests			
Total bilirubin (mg/dL)	0.67 ± 0.19	0.82 ± 0.24	0.001*
AST (IU/L)	28.3 ± 8.9	42.5 ± 16.8	<0.001*
ALT (IU/L)	29.1 ± 9.2	45.2 ± 18.3	<0.001*
ALP (IU/L)	98.7 ± 24.3	128.4 ± 32.5	<0.001*
Lipid Profile			
Total cholesterol (mg/dL)	178.4 ± 32.6	215.3 ± 38.2	<0.001*
Triglycerides (mg/dL)	138.2 ± 38.4	182.4 ± 52.3	<0.001*
LDL cholesterol (mg/dL)	108.2 ± 24.3	132.5 ± 28.4	<0.001*
VLDL cholesterol (mg/dL)	27.6 ± 7.7	36.5 ± 10.5	<0.001*
HbA1c (%)	7.59 ± 1.70	8.22 ± 1.17	0.040*
Serum creatinine (mg/dL)	0.94 ± 0.28	1.12 ± 0.34	0.005*

*Values presented as mean ± SD. *p<0.05 considered statistically significant. AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase; LDL: Low-density lipoprotein; VLDL: Very low-density lipoprotein; HbA1c: Glycosylated hemoglobin*

Discussion:

This cross-sectional study examined the prevalence and clinical correlates of NAFLD in patients with T2DM at a tertiary care hospital. The observed NAFLD prevalence of 42% falls within the range of recent estimates. A systematic review by Younossi *et al.* (2024) **[12]** reported that the prevalence of NAFLD among patients with T2DM ranges from 55% to 70%, while a 2023 meta-analysis by Cao *et al.* (2024) **[13]** of over 6.8 million participants found a global prevalence of 28.3% of type 2 diabetes among NAFLD patients. Regional variations in NAFLD prevalence among diabetic populations reflect differences in genetic susceptibility, dietary patterns and body composition characteristics. The strong association between obesity and NAFLD observed in our study aligns with current understanding of disease pathophysiology. Recent evidence demonstrates that the risk of metabolic syndrome in patients with obesity is 4.85 times greater than in those with BMI <25 kg/m². A 2022 meta-analysis reported that NAFLD affects approximately 75% of individuals with obesity **[14]**. Our finding that all obese patients (BMI >30 kg/m²) had NAFLD emphasizes the critical role of adiposity in disease pathogenesis. The mechanisms involve insulin resistance, increased hepatic de novo lipogenesis and enhanced fatty acid influx from peripheral adipose tissue. The significant association between poor glycemic control (higher HbA1c) and NAFLD represent a key finding with clinical implications. Recent research shows that glycemic control status is associated with exacerbation of NAFLD activity score and fibrosis stage **[15]**. Furthermore, a 2025 study utilizing NHANES data found that each 1% increase in the HbA1c/HDL-C ratio was associated with a 20% higher risk of NAFLD **[16]**. The bidirectional relationship between insulin resistance, hyperglycemia and hepatic steatosis creates a vicious cycle that promotes disease progression. The significant female predominance in NAFLD prevalence and severity observed in our study warrants discussion. While some Asian studies have reported higher NAFLD prevalence in females, this finding contrasts with Western populations where males typically show higher prevalence. Hormonal factors, body fat distribution patterns and metabolic differences may contribute to these gender-specific variations. The mechanisms underlying sex differences in NAFLD susceptibility require further investigation in Asian populations **[17]**. The strong association between diabetic complications and NAFLD observed in our study has important clinical implications. Recent evidence confirms that NAFLD progression is modulated by the same cardiometabolic risk factors that drive diabetic complications **[18]**. The 2025 ADA Standards of Care now recommend screening for MASLD in adults with type 2 diabetes, particularly those at high risk for liver fibrosis using th Fibrosis-4 index (FIB-4). This suggests that NAFLD may be part of a broader pattern of diabetic end-organ damage or that common pathophysiological mechanisms underlie both NAFLD and other diabetic complications **[19]**.

Ultrasonography served as the primary diagnostic modality in this study. Recent validation studies confirm that B-mode ultrasound is an excellent method for detecting moderate and severe steatosis in NAFLD patients, with sensitivity ranging from 78-92% and specificity from 85-95%. The dyslipidemia observed in our NAFLD patients aligns with contemporary understanding of metabolic dysfunction. Recent studies have identified novel obesity and metabolic indices that show superior performance in identifying NAFLD risk, with the lipid accumulation product and cardiometabolic index showing AUCs exceeding 0.72 **[20]**. Cho *et al.* (2023) **[21]** showed that NAFLD is associated with a significantly increased risk of liver-related complications, cardiovascular disease, and all-cause mortality, emphasizing the importance of early detection and risk stratification among patients with metabolic disorders. These findings further support the need for routine screening of patients with T2DM for hepatic steatosis. Kumar *et al.* (2022) **[22]** reported a high prevalence of NAFLD among patients with type 2 diabetes mellitus and identified obesity, dyslipidemia, and poor glycemic control as significant risk factors. Their findings are consistent with the present study, which also demonstrated significant associations between NAFLD, increased BMI, and elevated HbA1c levels. Nagal (2024) **[23]** observed that NAFLD is highly prevalent among individuals with T2DM and recommended routine ultrasonographic screening for early diagnosis, particularly in patients with metabolic risk factors. This supports the present findings and highlights the importance of timely identification of hepatic steatosis in diabetic populations. Hariharan *et al.* (2021) **[24]** found a significant association between NAFLD and obesity, poor glycemic control, and dyslipidemia in patients with T2DM. Their results closely parallel those of the present study, reinforcing the role of metabolic abnormalities in the development and progression of NAFLD. Karunakar *et al.* (2025) **[25]** reported that obesity-related metabolic indices and adverse lipid profiles were significantly

associated with NAFLD severity in patients with T2DM. The authors concluded that comprehensive metabolic assessment can improve early identification of high-risk individuals, which is in agreement with the findings of the present study. The integration of these indices with clinical parameters may enhance risk stratification in diabetic populations.

Conclusion:

NAFLD is highly prevalent in patients with T2DM and is closely linked to poor glycemic control, obesity, dyslipidemia and increased diabetic complications. Routine screening is essential, especially in individuals with multiple metabolic risk factors. Thus, early detection and integrated management focusing on glycemic control, weight reduction and cardiovascular risk can significantly improve long-term outcomes.

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