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Original Research Article

Prevalence of Non-Alcoholic Fatty Liver Disease in Type 2 Diabetes Mellitus: A Cross-Sectional Study at Swami Dayanand Hospital, Delhi

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Conflict of interest: Nil

Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) is a common metabolic complication of Type 2 Diabetes Mellitus (T2DM) and is associated with increased risk of hepatic fibrosis and cardiovascular complications. This study aimed to determine the prevalence of NAFLD among patients with T2DM and evaluate its association with metabolic and biochemical parameters.

Methods: A hospital-based cross-sectional study was conducted among 80 patients with T2DM attending Swami Dayanand Hospital, Delhi. Clinical assessment, anthropometric measurements, glycemic parameters, liver function tests, lipid profile, and viral hepatitis screening were performed. All participants underwent transient elastography (FibroScan®) assessment, with Controlled Attenuation Parameter (CAP) used for steatosis assessment and Liver Stiffness Measurement (LSM) for fibrosis evaluation.

Results: NAFLD was detected in 45 (57%) patients, while 35 (43%) had no evidence of NAFLD. The mean age of participants was 54.3 ± 10.5 years, and the mean BMI was 29.2 ± 4.6 kg/m². The study population showed poor glycemic control with a mean HbA1c of 8.4 ± 1.5%. Patients with NAFLD demonstrated significantly higher lipid abnormalities, including increased total cholesterol, triglycerides, and LDL-C, along with lower HDL-C levels. Liver enzymes were also significantly elevated among NAFLD patients. The mean CAP and LSM values were 259.8 ± 25.4 dB/m and 8.4 ± 3.2 kPa, respectively, indicating hepatic steatosis with early fibrosis changes.

Conclusion: NAFLD is highly prevalent among patients with T2DM and is associated with obesity, poor glycemic control, dyslipidemia, and hepatic injury markers. Routine screening using non-invasive tools such as FibroScan may facilitate early detection and management of diabetic patients at risk of progressive liver disease.

Keywords: Non-alcoholic fatty liver disease (NAFLD), Type 2 Diabetes Mellitus (T2DM), Non-alcoholic steatohepatitis (NASH).

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Introduction

Type 2 Diabetes Mellitus (T2DM) and non-alcoholic fatty liver disease (NAFLD) are two major metabolic disorders that have emerged as significant global public health concerns. NAFLD is characterized by excessive accumulation of fat in hepatocytes in the absence of significant alcohol consumption and represents a spectrum of liver disease ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), progressive fibrosis, cirrhosis, and hepatocellular carcinoma (HCC) [1]. With the increasing prevalence of obesity, insulin resistance, and metabolic syndrome, NAFLD has become the most common chronic liver disease worldwide [2].The global prevalence of NAFLD is estimated to be approximately 25%, although substantial variation exists between different geographical regions and populations [2]. The burden of NAFLD is considerably higher among individuals with T2DM, with prevalence estimates ranging from 34% to more than 70%, depending on diagnostic criteria, population characteristics, and assessment methods used [3,4]. Patients with diabetes represent a particularly high-risk group because of shared metabolic abnormalities, including insulin resistance, dyslipidemia, and chronic low-grade inflammation

[3].The pathophysiological association between NAFLD and T2DM is complex and bidirectional. Insulin resistance plays a central role in disease development by promoting increased adipose tissue lipolysis, elevated free fatty acid delivery to the liver, enhanced hepatic triglyceride synthesis, and impaired lipid oxidation, resulting in hepatic steatosis [6,7]. Persistent metabolic stress leads to oxidative stress, mitochondrial dysfunction, hepatocellular injury, and inflammatory activation, which may progress from simple steatosis to NASH and advanced hepatic fibrosis [6]. Conversely, NAFLD itself contributes to systemic insulin resistance and increases the future risk of developing T2DM, further strengthening the metabolic link between these two conditions [7].The coexistence of NAFLD and T2DM has important clinical implications. Individuals with both conditions have an increased risk of cardiovascular disease, progressive liver fibrosis, cirrhosis, and liver-related mortality compared with individuals without metabolic liver disease [3,6]. Although NAFLD is frequently asymptomatic and incidentally detected, a proportion of patients with NASH may progress to advanced fibrosis and cirrhosis, resulting in significant morbidity and mortality [1]. Therefore, early identification of NAFLD among patients with T2DM is essential for appropriate risk stratification and preventive management. Ultrasonography remains the most commonly used imaging modality for detection of hepatic steatosis because of its non-invasive nature, wide availability, and cost-effectiveness. It demonstrates good diagnostic accuracy for moderate-to-severe hepatic steatosis and is particularly useful for screening high-risk populations such as patients with diabetes mellitus [9]. Although newer techniques such as controlled attenuation parameter (CAP) and vibration-controlled transient elastography (VCTE/FibroScan®) provide improved assessment of steatosis and fibrosis, their availability remains limited in many healthcare settings [9].The understanding of metabolic fatty liver disease has evolved in recent years. An international expert consensus proposed the term metabolic dysfunction-associated fatty liver disease (MAFLD) to emphasize the central role of metabolic dysfunction in the development of fatty liver disease [8]. This concept highlights that fatty liver disease is not merely a liver-specific disorder but a systemic metabolic condition associated with diabetes, obesity, and other cardiometabolic risk factors.In India, the prevalence of NAFLD has increased substantially due to the rising burden of diabetes, obesity, and sedentary lifestyles. Indian patients with T2DM are considered a high-risk population for NAFLD; however, available epidemiological data remain variable due to differences in geographical distribution, dietary patterns, ethnicity, and healthcare access [5]. Most published studies have originated from selected regions, and data from northern urban healthcare settings remain relatively limited.Although biochemical parameters such as alanine aminotransferase (ALT) are commonly evaluated in diabetic patients, liver enzyme abnormalities alone may underestimate the actual burden of NAFLD, as many patients with hepatic steatosis may have normal liver function tests. Taseer et al. reported the presence of NAFLD and associated biochemical abnormalities among patients with T2DM, highlighting the importance of imaging-based evaluation for accurate detection [10]. Considering the increasing prevalence of metabolic disorders and the limited regional data regarding NAFLD among diabetic individuals, this study was undertaken to determine the prevalence of non-alcoholic fatty liver disease among patients with Type 2 Diabetes Mellitus attending Swami Dayanand Hospital, Delhi.

Materials and Methods

This hospital-based cross-sectional study was conducted in the Department of Medicine, Swami Dayanand Hospital, Delhi, over a period of 18 months. The study will include patients with Type 2 Diabetes Mellitus (T2DM) attending the diabetes clinic. The study aims to assess the prevalence of non-alcoholic fatty liver disease (NAFLD) and its association with metabolic and biochemical parameters. A total of 80 patients with T2DM fulfilling the eligibility criteria will be enrolled after obtaining written informed consent. The diagnosis of T2DM were established according to standard diagnostic criteria.

Data Collection and Clinical Assessment: After enrollment, detailed history and clinical examination will be performed. Demographic and clinical data including age, sex, and duration of diabetes, BMI, and relevant medical history will be recorded.

Laboratory investigations will include:

- Fasting and postprandial blood glucose
- HbA1c
- Liver function tests (AST, ALT, alkaline phosphatase)
- Lipid profile (total cholesterol, triglycerides, HDL-C, LDL-C)
- Screening for hepatitis B surface antigen (HBsAg) and hepatitis C virus (HCV)

FibroScan Assessment: All eligible patients will undergo transient elastography (FibroScan®) examination using the TOUCH 502 FibroScan system (France) with appropriate M or XL probe selection based on automated skin-to-liver distance assessment.

Two parameters will be assessed:

Controlled Attenuation Parameter (CAP): CAP values will be used for grading hepatic steatosis and expressed as dB/m:

- S0: <237 dB/m (No steatosis)
- S1: 237–259 dB/m (Mild steatosis)
- S2: 259–291 dB/m (Moderate steatosis)
- S3: 291–400 dB/m (Severe steatosis)

Liver Stiffness Measurement (LSM): LSM values will be expressed in kPa and used for fibrosis staging:

- F0: <5.5 kPa (No fibrosis)
- F1: 5.5–8.0 kPa (Mild fibrosis)
- F2: 8.0–10.0 kPa (Moderate fibrosis)
- F3: 11–16 kPa (Severe fibrosis)
- F4: >16 kPa (Cirrhosis)

Inclusion Criteria

- Patients aged 22–75 years
- Confirmed diagnosis of Type 2 Diabetes Mellitus
- Patients with fatty liver on ultrasonography
- BMI >23 kg/m² (Asian Indian criteria)
- Patients providing informed consent

Exclusion Criteria

Patients with the following conditions will be excluded:

- Type 1 diabetes mellitus, secondary diabetes, or MODY
- Significant alcohol consumption
- Pregnancy
- Viral hepatitis infection
- Total parenteral nutrition
- Untreated thyroid disorders
- History of bariatric surgery
- Inflammatory bowel disease
- Recent starvation or rapid weight loss
- Inherited metabolic disorders affecting hepatic lipid metabolism
- Use of medications associated with hepatic steatosis (e.g., corticosteroids, methotrexate, amiodarone, tamoxifen, valproate, HAART, and hepatotoxic herbal preparations)

Statistical Analysis: Statistical analysis will be performed using SPSS version 25.0. Continuous variables will be expressed as mean ± standard deviation or median (IQR), while categorical variables will be presented as frequency and percentage. Comparison between groups will be performed using the independent t-test or appropriate non-parametric tests for continuous variables and Chi-square/Fisher's exact test for categorical variables. A p-value <0.05 will be considered statistically significant.

Results

A hospital-based cross-sectional study was conducted among 80 patients with Type 2 Diabetes Mellitus (T2DM) to assess the prevalence of non-alcoholic fatty liver disease (NAFLD) and its association with demographic, metabolic, biochemical, and FibroScan parameters. Among the 80 participants, NAFLD was present in 45 (57.0%) patients, while 35 (43.0%) had no evidence of NAFLD (Table 2). The mean age of the study population was 54.3 ± 10.5 years (median: 53 years; IQR: 48–60). The most common age group was 50–59 years (37.5%), followed by 40–49 years (25.0%). The study population included 40 (50.0%) males and 40 (50.0%) females. The mean BMI was 29.2 ± 4.6 kg/m², with 37.5% participants being overweight and 37.5% obese (Table 1).The mean fasting blood glucose, postprandial blood glucose, and HbA1c levels were 180.5 ± 45.0 mg/dL, 225.8 ± 50.5 mg/dL, and 8.4 ± 1.5%, respectively. The lipid profile showed mean total cholesterol of 185.5 ± 22.4 mg/dL, triglycerides of 190.2 ± 30.5 mg/dL, HDL-C of 45.7 ± 10.2 mg/dL, and LDL-C of 125.3 ± 20.5 mg/dL (Table 2).The mean AST, ALT, and alkaline phosphatase levels were 55.3 ± 10.2 U/L, 62.7 ± 12.8 U/L, and 120.2 ± 20.5 U/L, respectively. FibroScan analysis showed a mean CAP value of 259.8 ± 25.4 dB/m and LSM value of 8.4 ± 3.2 kPa, suggesting hepatic steatosis with mild-to-moderate fibrosis (Table 3; Figure 2).Patients with NAFLD had significantly higher total cholesterol (190.0 ± 24.5 vs 180.0 ± 18.0 mg/dL; p=0.045), triglycerides (195.0 ± 35.0 vs 180.0 ± 28.0 mg/dL; p=0.042), and LDL-C levels (130.0 ± 25.0 vs 120.0 ± 18.0 mg/dL; p=0.015), with significantly lower HDL-C levels (43.5 ± 9.0 vs 48.0 ± 10.2 mg/dL; p=0.022) compared with non-NAFLD patients (Table 4).

Among NAFLD patients, those with steatosis demonstrated significantly higher CAP (280.0 ± 30.0 vs 240.0 ± 20.0 dB/m; p=0.001), LSM (9.5 ± 3.5 vs 7.0 ± 1.5 kPa; p=0.023), BMI (32.0 ± 5.0 vs 28.0 ± 4.0 kg/m²; p=0.035), and HbA1c levels (8.9 ± 1.3 vs 8.0 ± 1.5%; p=0.047) (Table 5).Liver enzymes were significantly elevated in patients with NAFLD, including AST (58.0 ± 12.0 vs 50.0 ± 8.5 U/L; p=0.031), ALT (65.0 ± 15.0 vs 55.0 ± 10.0 U/L; p=0.039), and alkaline phosphatase (125.0 ± 23.0 vs 110.0 ± 20.0 U/L; p=0.022) (Table 6; Figure 3).

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants (n=80)

Variable	Mean ± SD / Number (%)
Age (years)	54.3 ± 10.5
Age group <40 years	15 (18.75%)
Age group 40–49 years	20 (25.0%)
Age group 50–59 years	30 (37.5%)
Age group ≥60 years	15 (18.75%)
Male	40 (50%)
Female	40 (50%)
BMI (kg/m²)	29.2 ± 4.6
Normal weight (BMI <24.9)	20 (25%)
Overweight (BMI 25–29.9)	30 (37.5%)
Obese (BMI ≥30)	30 (37.5%)

Table 2: Prevalence of NAFLD and Metabolic Parameters among Study Participants

Parameter	Value
Total participants	80
NAFLD present	45 (57%)
NAFLD absent	35 (43%)
Fasting blood glucose (mg/dL)	180.5 ± 45.0
Postprandial blood glucose (mg/dL)	225.8 ± 50.5
HbA1c (%)	8.4 ± 1.5
Total cholesterol (mg/dL)	185.5 ± 22.4
Triglycerides (mg/dL)	190.2 ± 30.5
HDL-C (mg/dL)	45.7 ± 10.2
LDL-C (mg/dL)	125.3 ± 20.5

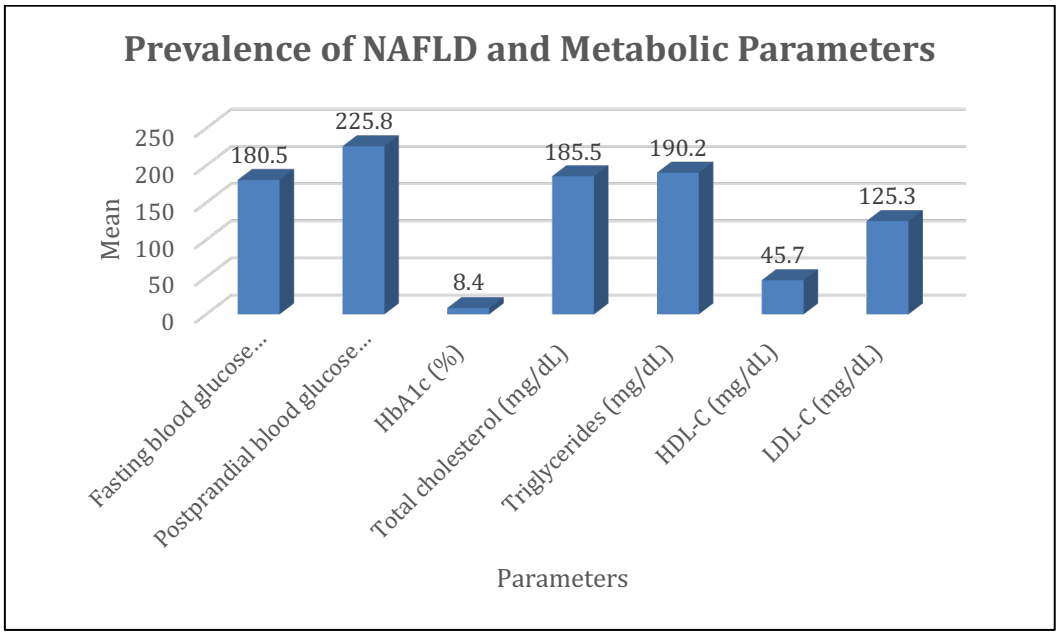


Figure 1: Prevalence of NAFLD and Metabolic Parameters among Study Participants

Table 3: Liver Function Profile and FibroScan Parameters of Study Participants

Parameter	Mean ± SD	Median (IQR)
AST (U/L)	55.3 ± 10.2	55 (50–60)
ALT (U/L)	62.7 ± 12.8	60 (55–70)
Alkaline phosphatase (U/L)	120.2 ± 20.5	118 (110–130)
CAP (dB/m)	259.8 ± 25.4	260 (245–275)
LSM (kPa)	8.4 ± 3.2	8.0 (7.0–10.0)

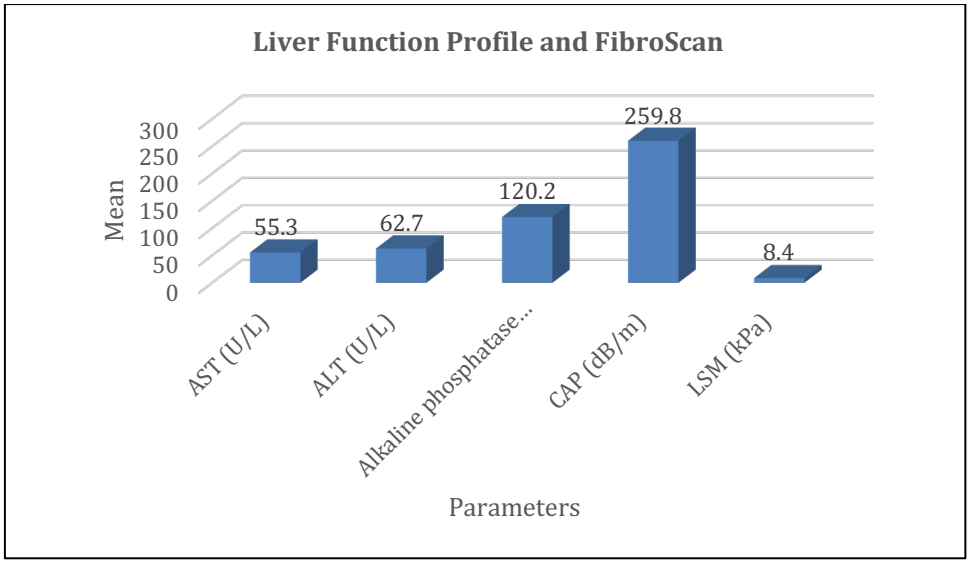


Figure 2: Liver Function Profile and FibroScan Parameters of Study Participants

Table 4: Comparison of Clinical and Biochemical Parameters between NAFLD and Non-NAFLD Groups

Variable	NAFLD Present (n=45)	NAFLD Absent (n=35)	p-value
Total cholesterol (mg/dL)	190.0 ± 24.5	180.0 ± 18.0	0.045
Triglycerides (mg/dL)	195.0 ± 35.0	180.0 ± 28.0	0.042
HDL-C (mg/dL)	43.5 ± 9.0	48.0 ± 10.2	0.022
LDL-C (mg/dL)	130.0 ± 25.0	120.0 ± 18.0	0.015

Table 5: Association of NAFLD with FibroScan-Based Steatosis Parameters and Metabolic Factors

Variable	NAFLD with Steatosis (n=40)	NAFLD without Steatosis (n=5)	p-value
CAP (dB/m)	280.0 ± 30.0	240.0 ± 20.0	0.001
LSM (kPa)	9.5 ± 3.5	7.0 ± 1.5	0.023
BMI (kg/m²)	32.0 ± 5.0	28.0 ± 4.0	0.035
HbA1c (%)	8.9 ± 1.3	8.0 ± 1.5	0.047

Table 6: Comparison of Liver Function Tests between NAFLD and Non-NAFLD Groups

Liver Function Parameter	NAFLD Present (n=45)	NAFLD Absent (n=35)	p-value
AST (U/L)	58.0 ± 12.0	50.0 ± 8.5	0.031
ALT (U/L)	65.0 ± 15.0	55.0 ± 10.0	0.039
Alkaline phosphatase (U/L)	125.0 ± 23.0	110.0 ± 20.0	0.022

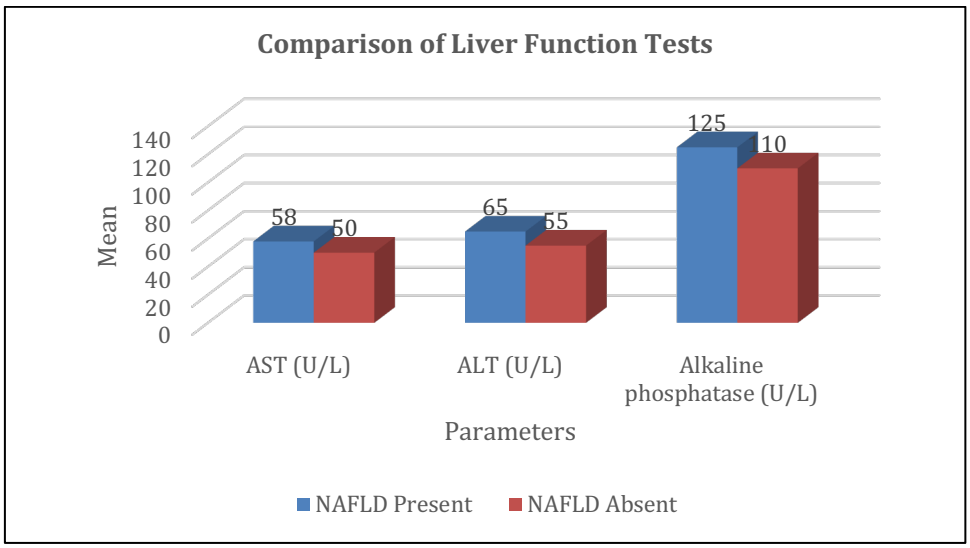


Figure 3: Comparison of Liver Function Tests between NAFLD and Non-NAFLD Groups

Discussion

The present hospital-based cross-sectional study evaluated the prevalence of non-alcoholic fatty liver disease (NAFLD) among 80 patients with Type 2 Diabetes Mellitus (T2DM) and assessed its association with demographic, metabolic, biochemical, and FibroScan parameters. NAFLD was detected in 57% (45/80) of participants, indicating a high burden of hepatic steatosis among diabetic patients. This finding is comparable with previous studies. Dharmalingam et al. reported NAFLD prevalence of approximately 70% among T2DM patients [5], while Zhou et al. and Sinha et al. reported prevalence rates of 58.67% and 57%, respectively [11,12]. Meta-analyses by Dai et al. and Younossi et al. also demonstrated a high prevalence of NAFLD among diabetic populations (59.67% and 55.5%, respectively) [13,14]. Cho et al. further highlighted the clinical importance of NAFLD by reporting significant proportions of NASH and fibrosis among T2DM patients [15].

The mean age of study participants was 54.3 ± 10.5 years, with the majority belonging to the 50–59 years age group. Similar findings were reported by Younossi et al., Zhou et al., and Sinha et al., who observed higher NAFLD prevalence among middle-aged and older diabetic individuals [11,12,14]. The present study showed equal gender distribution (50% males and 50% females). Although mild male predominance has been reported by Sinha et al. and Younossi et al., Dai et al. found no significant gender difference, suggesting that metabolic factors are more important determinants than sex alone [11,13,14].The mean BMI in the present study was 29.2 ± 4.6 kg/m², with 37.5% participants being overweight and 37.5% obese. These findings support the strong association between obesity, insulin resistance, and NAFLD. Dharmalingam et al. and Dai et al. reported obesity and metabolic abnormalities as important risk factors for NAFLD in diabetic patients [5, 13]. Cho et al. demonstrated that increased metabolic risk contributes to progression from steatosis to NASH and fibrosis, while Raval et al. also reported higher NAFLD prevalence among overweight and obese diabetic individuals [15,16].The study population showed poor glycemic control, with mean fasting blood glucose of 180.5 ± 45.0 mg/dL, postprandial glucose of 225.8 ± 50.5 mg/dL, and HbA1c of 8.4 ± 1.5%. Poor glycemic control contributes to hepatic fat accumulation through insulin resistance and increased hepatic lipogenesis. Raval et al. reported a significant association between NAFLD and poor glycemic control, while Puneem et al. demonstrated associations between NAFLD, uncontrolled glucose levels, triglycerides, and liver enzyme abnormalities [28,35]. Dai et al. also reported that HbA1c and other metabolic factors influence NAFLD prevalence among diabetic patients [22].The present study demonstrated elevated liver enzymes, with mean AST 55.3 ± 10.2 U/L, ALT 62.7 ± 12.8 U/L, and alkaline phosphatase 120.2 ± 20.5 U/L. NAFLD patients showed significantly higher liver enzyme levels compared with non-NAFLD patients. Similar findings were reported by Dai et al. and Dharmalingam et al., supporting the role of biochemical abnormalities in NAFLD detection [5,13]. Acierno et al. explained that insulin resistance-induced hepatic fat accumulation and oxidative stress contribute to hepatocellular injury and transaminase elevation [17].

The present study also demonstrated an adverse lipid profile among NAFLD patients, with higher total cholesterol, triglycerides, and LDL-C and lower HDL-C levels compared with non-NAFLD patients. These findings are consistent with Dai et al. and Cho et al.who identified dyslipidemia as an important contributor to NAFLD development and progression [13,15].

FibroScan evaluation showed mean CAP of 259.8 ± 25.4 dB/m and LSM of 8.4 ± 3.2 kPa, indicating hepatic steatosis with possible fibrosis changes. Patients with steatosis had significantly higher CAP, LSM, BMI, and HbA1c values, suggesting an association between metabolic dysfunction and hepatic disease severity. Similar findings were reported by Sinha et al., Cho et al., Ciardullo et al., and Dharmalingam et al., who emphasized the importance of transient elastography for early detection and staging of NAFLD and fibrosis in diabetic patients [5,12,15,18].

Conclusion

NAFLD was highly prevalent among patients with Type 2 Diabetes Mellitus, affecting 57% of the study population. NAFLD was significantly associated with obesity, poor glycemic control, dyslipidemia, elevated liver enzymes, and abnormal FibroScan parameters. Routine screening for NAFLD using non-invasive tools such as FibroScan may help in early identification and prevention of progressive liver disease in diabetic patients.

Limitations

The study was conducted at a single center with a relatively small sample size, limiting the generalizability of the findings. The cross-sectional design prevents assessment of temporal relationships and long-term progression of NAFLD.

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