

Research Article

# A Cross-Sectional Study to Assess the Prevalence of Non-Alcoholic Fatty Liver Disease among Type 2 Diabetes Patients

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## ABSTRACT

**Background:** Non-alcoholic Liver Disease (NAFLD) is a condition in which fat builds up in the liver of people who drink little or no alcohol. The present study was conducted to assess non alcoholic liver disease in type II diabetes mellitus patients.

**Materials & Methods:** 150 type II diabetes mellitus patients of both genders were divided into 2 groups. Group I had NAFLD, while group II did not. The following measurements were taken as part of a general physical examination: height, weight, waist circumference (WC), hip circumference, and body mass index (BMI). A common mercury manometer was used to measure the systolic and diastolic blood pressure. Venous blood was drawn following an overnight fast of at least eight hours. FBG, serum bilirubin, serum glutamic-oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), alkaline phosphate (ALP), serum total proteins (STP), and lipid profile were all tested using standard laboratory procedures.

**Results:** Group I had 35 males and 40 females and group II had 30 males and 45 females. The mean BMI (kg/m<sup>2</sup>) was 23.2±3.1 and 20.3±8.5, waist to hip ratio was 0.90±0.2 and 0.94±0.6, FBG (mg%) was 152.4±25.4 and 144.6±19.7, TC (mg%) was 224.4±15.6 and 194.2±21.3, TGL (mg%) was 215.4±34.5 and 178.4±11.2, HDL (mg%) was 45.5±7.3 and 49.2±8.3, SGOT (IU/L) was 22.1±3.7 and 18.5±1.2, SGPT (IU/L) was 23.3±2.5 and 17.3±3.6, ALP (IU/L) was 125.4±15.5 and 112.7±17.2 and STP (G%) was 6.7±1.0 and 6.1±1.1 and serum bilirubin (mg%) was 0.82±0.1 and 0.80±0.4 in group I and group II respectively. The difference was significant (P< 0.05).

**Conclusion:** NAFLD is common in patients with type 2 diabetes, and risk factors include obesity, dyslipidemia, and poor glycaemic control.

**Keywords:** Cirrhosis, Diabetes Mellitus, Non-Alcoholic Fatty Liver Disease.

## INTRODUCTION

Non-alcoholic Liver Disease (NAFLD) is a condition in which fat builds up in the liver of people who drink little or no alcohol. It's one of the most common liver disorders worldwide and is closely associated with obesity, type 2 diabetes, and metabolic syndrome.<sup>1</sup> There are two main forms- simple fatty liver (Steatosis) in which fat is present in the liver, but there is little or no inflammation or liver cell damage. It usually does not progress to serious liver damage. Non-alcoholic steatohepatitis (NASH) is more severe form with inflammation and liver cell damage. It can progress to fibrosis, cirrhosis, or even liver cancer.<sup>2</sup> Nonalcoholic steatohepatitis (NASH) is intimately associated with metabolic diseases, including central obesity, insulin resistance (IR), dyslipidemia, diabetes mellitus (DM), and hypertension. NAFLD is considered the hepatic manifestation of the metabolic syndrome.<sup>3</sup> IR is believed to be a characteristic of NAFLD and a contributing

factor, even in the absence of obesity and diabetes mellitus. Increased adiposity, commonly seen in NAFLD and T2DM, is associated with adipocyte IR and dysfunction. The danger of lipotoxicity, which happens when adipose tissue overflowing FFA and causes the liver, pancreas, or muscle to absorb too much lipid, is increased when there is an excess of FFA released into the bloodstream.<sup>4</sup> Non-alcoholic fatty liver disease (NAFLD) in diabetes mellitus (DM)—especially type 2 diabetes—is extremely common and clinically important. These two conditions are deeply interconnected through insulin resistance and metabolic syndrome. Over 70% of people with type 2 diabetes are estimated to have NAFLD.<sup>5,6</sup> Diabetes increases the risk of progression from NAFLD to NASH, fibrosis, and cirrhosis. NAFLD in diabetics is associated with a higher risk of cardiovascular disease, kidney disease, and liver-related mortality.<sup>7,8</sup>

## AIM & OBJECTIVES

**Aim** To assess the prevalence of non-alcoholic fatty liver disease among type 2 diabetes patients.

### Objectives

1. To determine the distribution of NAFLD among T2DM patients using clinical, biochemical, and anthropometric parameters.
2. To compare metabolic parameters such as BMI, waist-to-hip ratio, fasting blood glucose, lipid profile, and HbA1c between T2DM patients with and without NAFLD.
3. To evaluate liver function abnormalities (SGOT, SGPT, ALP, serum proteins, and bilirubin) in patients with NAFLD compared to those without NAFLD.
4. To analyze the association between poor glycemic control, dyslipidemia, and liver enzyme derangements in T2DM patients with NAFLD.

## MATERIALS & METHODS

### Study Design

This was a hospital-based cross-sectional observational study conducted to assess the prevalence and clinical characteristics of Non-Alcoholic Fatty Liver Disease (NAFLD) among patients with Type 2 Diabetes Mellitus (T2DM).

### Study Population

A total of 150 patients with T2DM of both genders were enrolled in the study. Patients were stratified into two groups:

- **Group I (n = 75):** Patients with NAFLD.
- **Group II (n = 75):** Patients without NAFLD.

### Study Place

The study was carried out in the outpatient department (OPD) patients, Department of General Medicine at JIET Medical College & Hospital, Jodhpur.

### Study Period

The study was carried out over a period of 1 year, from October 2023 to September 2024.

### Ethical Considerations

Ethical clearance was obtained from the Institutional Ethics Committee prior to the commencement of the study. Written informed consent was obtained from all participants. Patients were assured of confidentiality and informed about the right to withdraw at any stage without prejudice to treatment.

### Inclusion Criteria

- Patients with T2DM diagnosed for at least 6 months.
- Both males and females aged  $\geq 18$  years.

- No history of alcohol intake, as confirmed by the patient or next of kin.

### Exclusion Criteria

- History of jaundice, ascites, or liver cell failure.
- Patients on hepatotoxic drugs (methotrexate, amiodarone, glucocorticoids, synthetic estrogens, nucleoside analogs).
- Chronic renal failure, chronic cardiac diseases, history of diabetic ketoacidosis, or major abdominal surgery.
- Patients positive for Hepatitis B surface antigen (HBsAg).
- Patients with chronic liver diseases of other etiologies.

### Study Procedure

1. A detailed history was recorded, including:
  - Name, age, sex, duration of diabetes, symptoms suggestive of liver disease, alcohol consumption, past surgical history, and drug history.
2. A general physical examination was performed, including:
  - Height, weight, waist circumference (WC), hip circumference, and blood pressure (systolic/diastolic) using a mercury sphygmomanometer.
  - **Body mass index (BMI):** calculated as weight in kilograms divided by height in meters squared ( $\text{kg}/\text{m}^2$ ).
  - **Waist-Hip Ratio (WHR):** calculated from WC and hip circumference.
  - Abdominal examination was done to assess hepatomegaly ( $>15$  cm in longitudinal plane), splenomegaly, and presence of free fluid.

### Investigations

Venous blood was collected after an overnight fast of at least 8 hours. The following biochemical parameters were measured using standard laboratory procedures:

- Fasting Blood Glucose (FBG)
- **Post-Prandial Blood Glucose (PPBG)** – measured 2 hours after breakfast
- HbA1c
- Serum Bilirubin
- **Liver function tests:** Serum Glutamic-Oxaloacetic Transaminase (SGOT), Serum Glutamic Pyruvic Transaminase (SGPT), Alkaline Phosphatase (ALP), Serum Total Proteins (STP)
- **Lipid profile:** Total Cholesterol (TC), Triglycerides (TGL), High-Density Lipoprotein (HDL)

Hepatic ultrasonography was performed on all patients by a trained radiologist using a Logitech EP5 ultrasound machine with a 4 MHz probe. Hepatic steatosis was diagnosed based on ultrasonographic findings such as:

- Bright liver pattern
- Blurring of hepatic vessels
- Narrowing of hepatic vein lumen
- Hepatorenal contrast
- Presence of focal sparing.

**Outcome Measures**

The primary outcomes assessed were:

1. **Prevalence of NAFLD** among patients with T2DM.
2. **Comparison of clinical and biochemical characteristics** (anthropometric indices, lipid profile, glycemic control, and liver

function tests) between patients with and without NAFLD.

**Statistical Analysis**

- All data were entered into Microsoft Excel and analyzed using SPSS version 20.0 (Chicago, IL, USA).
- Quantitative variables were expressed as mean ± standard deviation (SD).
- Categorical variables were expressed as proportions and percentages.
- Comparisons:
  - Student’s *t*-test or Mann-Whitney U-test was used for quantitative variables.
  - Chi-square test or Fisher’s exact test was applied for categorical variables.
- P-value <0.05 was considered statistically significant

**RESULTS**

Table 1: Distribution of Patients

Groups	Group I (n=75)	Group II (n=75)
Status	NAFLD	Without NAFLD
M:F	35:40:00	30:45:00

Table 1 show that group I had 35 males and 40 females and group II had 30 males and 45 females.

Table 2: Assessment of Parameters

Parameters	Group I (n=75)	Group II (n=75)	P value
<b>Anthropometric measurements</b>			
BMI (kg/m <sup>2</sup> )	23.2±3.1	20.3±8.5	0.64
Waist to hip ratio	0.90±0.2	0.94±0.6	0.13
<b>Serum Biochemical Level</b>			
FBG (mg%)	152.4±25.4	144.6±19.7	0.05
TC (mg%)	224.4±15.6	194.2±21.3	0.01
TGL (mg%)	215.4±34.5	178.4±11.2	0.02
HDL (mg%)	45.5±7.3	49.2±8.3	0.75
HbA1c (%)	7.52±0.40	7.02±0.44	0.001
<b>Liver function tests</b>			
SGOT (IU/L)	22.1±3.7	18.5±1.2	0.02
SGPT (IU/L)	23.3±2.5	17.3±3.6	0.01
ALP (IU/L)	125.4±15.5	112.7±17.2	0.05
STP (g%)	6.7±1.0	6.1±1.1	0.91
Serum bilirubin (mg%)	0.82±0.1	0.80±0.4	0.42

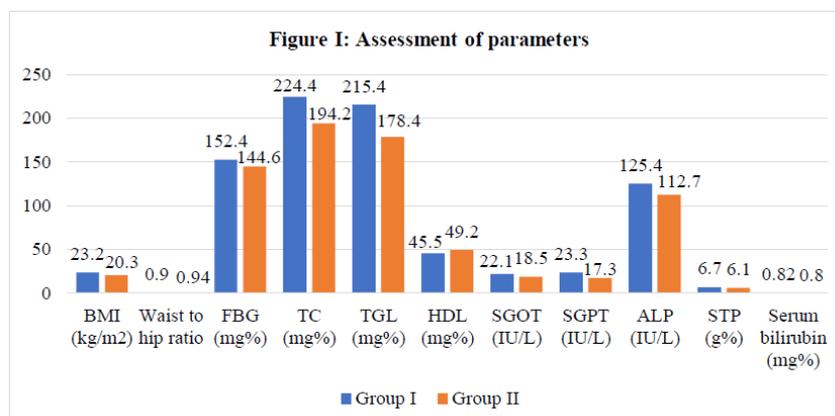


Table 2 and figure I, presents the comparative assessment of various clinical and biochemical parameters between Group I and Group II patients. The mean BMI was slightly higher in Group I ( $23.2 \pm 3.1 \text{ kg/m}^2$ ) compared to Group II ( $20.3 \pm 8.5 \text{ kg/m}^2$ ), though this difference was not statistically significant ( $p = 0.64$ ). Similarly, waist-to-hip ratio was comparable between the groups ( $0.90 \pm 0.2$  vs.  $0.94 \pm 0.6$ ,  $p = 0.13$ ).

Fasting blood glucose (FBG) levels were elevated in both groups, with Group I showing marginally higher values ( $152.4 \pm 25.4 \text{ mg\%}$ ) than Group II ( $144.6 \pm 19.7 \text{ mg\%}$ ); however, the difference approached but did not reach statistical significance ( $p = 0.05$ ). In contrast, lipid profile parameters demonstrated significant differences: total cholesterol (TC) and triglycerides (TGL) were markedly higher in Group I ( $224.4 \pm 15.6 \text{ mg\%}$  and  $215.4 \pm 34.5 \text{ mg\%}$ , respectively) compared to Group II ( $194.2 \pm 21.3 \text{ mg\%}$  and  $178.4 \pm 11.2 \text{ mg\%}$ ), with p-values of 0.01 and 0.02, respectively. HDL levels were slightly lower in Group I ( $45.5 \pm 7.3 \text{ mg\%}$ ) than in Group II ( $49.2 \pm 8.3 \text{ mg\%}$ ), though the difference was not statistically significant ( $p = 0.75$ ).

Liver function parameters showed notable variation: SGOT ( $22.1 \pm 3.7$  vs.  $18.5 \pm 1.2 \text{ IU/L}$ ,  $p = 0.02$ ) and SGPT ( $23.3 \pm 2.5$  vs.  $17.3 \pm 3.6 \text{ IU/L}$ ,  $p = 0.01$ ) were significantly higher in Group I, suggesting greater hepatic involvement. Alkaline phosphatase (ALP) was also higher in Group I ( $125.4 \pm 15.5 \text{ IU/L}$ ) compared to Group II ( $112.7 \pm 17.2 \text{ IU/L}$ ), with borderline significance ( $p = 0.05$ ). Serum total protein (STP) levels ( $6.7 \pm 1.0 \text{ g\%}$  vs.  $6.1 \pm 1.1 \text{ g\%}$ ) and serum bilirubin ( $0.82 \pm 0.1 \text{ mg\%}$  vs.  $0.80 \pm 0.4 \text{ mg\%}$ ) were comparable between groups, with no significant differences ( $p = 0.91$  and  $p = 0.42$ , respectively).

## DISCUSSION

Non-alcoholic Fatty Liver Disease (NAFLD) is the most common chronic liver disorder globally and is highly prevalent in individuals with Type 2 Diabetes Mellitus (T2DM).<sup>9,10</sup> NAFLD encompasses a spectrum ranging from simple hepatic steatosis (fat accumulation) to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and even hepatocellular carcinoma (HCC).<sup>11,12</sup> The present study was conducted to assess non-alcoholic liver disease in type II diabetes mellitus patients. We found that group I had 35 males and 40 females and group II had 30 males and 45 females. Gaharwar et al.<sup>13</sup> studied the clinical profile of patients of NAFLD

with varying degrees of severity as diagnosed by ultrasonography and to study the correlation between the non-alcoholic fatty liver disease and metabolic syndrome along with its individual components. All patients diagnosed as NAFLD were investigated for metabolic syndrome according to the NCEP ATP 3 Criteria and a relationship between NAFLD and metabolic syndrome was studied. 51.4% of patients of NAFLD had metabolic syndrome and statistical significance was found in AST, diabetes mellitus and lipid profile. In this cross-sectional analysis comparing 75 patients each in Group I and Group II, several noteworthy differences emerge. While anthropometric parameters—BMI and waist-to-hip ratio—did not significantly differ ( $p = 0.64$  and  $0.13$ , respectively), serum biochemical markers showed important distinctions. Patients in Group I exhibited higher fasting blood glucose ( $152.4 \pm 25.4 \text{ mg\%}$  vs.  $144.6 \pm 19.7 \text{ mg\%}$ ,  $p = 0.05$ ), elevated total cholesterol ( $224.4 \pm 15.6 \text{ mg\%}$  vs.  $194.2 \pm 21.3 \text{ mg\%}$ ,  $p = 0.01$ ), and higher triglycerides ( $215.4 \pm 34.5 \text{ mg\%}$  vs.  $178.4 \pm 11.2 \text{ mg\%}$ ,  $p = 0.02$ ). HDL levels were comparable ( $45.5 \pm 7.3 \text{ mg\%}$  vs.  $49.2 \pm 8.3 \text{ mg\%}$ ,  $p = 0.75$ ). Additionally, HbA<sub>1c</sub> was significantly higher in Group I ( $7.52 \pm 0.40\%$  vs.  $7.02 \pm 0.44\%$ ,  $p = 0.001$ ). Liver enzymes—SGOT, SGPT, and ALP—were all elevated in Group I, particularly SGOT and SGPT ( $p = 0.02$  and  $p = 0.01$ ), indicative of hepatic involvement. Conversely, serum total protein and bilirubin showed no significant differences. These observations are consistent with earlier research. Butt *et al.* (2019) reported a high prevalence of NAFLD (71.9%) in recently diagnosed T2DM patients in Pakistan, where dyslipidemia, elevated LDL, higher HbA<sub>1c</sub>, and lower HDL were significant predictors of NAFLD (adjusted ORs: dyslipidemia 2.38; LDL, HbA<sub>1c</sub> also significant).<sup>14</sup> Similarly, a systematic analysis by Younossi *et al.* (2019) found a global NAFLD prevalence of 55.5% among T2DM patients, with 37.3% exhibiting NASH and 17% having advanced fibrosis.<sup>15</sup> These prevalence data align with the metabolic derangements observed in your Group I cohort. Elevated triglycerides and low HDL emerged repeatedly as key metabolic markers in NAFLD. In an Indian cohort, increased BMI, AST, ALT, GGT, and triglycerides, along with reduced HDL, were significantly associated with NAFLD in T2DM patients.<sup>16</sup> Moreover, the meta-analysis by Fan *et al.* (2017) confirmed a pooled NAFLD prevalence of ~60% among T2DM patients, highlighting shared metabolic risk factors and

reinforcing the need for early assessment.<sup>17</sup> Furthermore, in patients with T2DM but normal aminotransferases, NAFLD prevalence remained surprisingly high (~50%), and elevated HbA<sub>1c</sub> correlated with greater prevalence and severity of liver fat accumulation, echoing your findings of elevated HbA<sub>1c</sub> and liver enzymes in Group I.<sup>18</sup> Finally, hypertriglyceridemia has been identified as the most potent predictor of NAFLD in regression models, surpassing blood glucose and HbA<sub>1c</sub> in predictive strength, aligning with your observation of significantly elevated triglycerides in Group I.<sup>19</sup> Yi et al.<sup>20</sup> investigated the prevalence of and risk factors for non-alcoholic fatty liver disease in overweight and obese patients with Type 2 diabetes mellitus. The study included 3861 patients (1860 men) with a mean age of 58.91 ± 13.06 (18-90) years. Non-alcoholic fatty liver disease was found in 1751 patients (45.4%), with a significantly higher prevalence among men than women (48.0 vs 42.9%). The peak of non-alcoholic fatty liver disease prevalence was in patients with a BMI of 34-35 kg/m<sup>2</sup>, those with a triglyceride/HDL cholesterol ratio of 5.5-6.0, men aged < 30 years and women aged 40-50 years. Assessment using the BARD (BMI, as part at aminotransferase/alanine aminotransferase ratio, diabetes) score system showed that the prevalence of advanced fibrosis was 80.52% in all patients and that women had a higher prevalence than men (86.52 vs. 74.16%). Multiple logistic regression analyses showed that dyslipidaemia, BMI and 2-h postprandial plasma glucose were independent risk factors for non-alcoholic fatty liver disease, while heart rate and female gender were protective factors.

#### LIMITATIONS OF THE STUDY

- Sample size was relatively small (150 patients), which may limit the generalizability of the findings.
- The study was cross-sectional, so causal relationships between NAFLD and metabolic parameters cannot be established.
- NAFLD diagnosis was based on biochemical and clinical parameters; imaging (ultrasound) or histological confirmation was not included, which could have provided greater diagnostic accuracy.
- The study did not account for lifestyle factors (diet, physical activity, smoking, alcohol intake) that might influence NAFLD prevalence.

- The absence of longitudinal follow-up restricts the ability to assess disease progression or the impact of interventions.

#### CONCLUSION

The present study highlights a high prevalence of NAFLD among patients with Type 2 Diabetes Mellitus. Patients with NAFLD demonstrated significantly higher fasting blood glucose, HbA<sub>1c</sub>, total cholesterol, triglycerides, and deranged liver enzymes (SGOT, SGPT, ALP) compared to those without NAFLD. These findings suggest that poor glycemic control and dyslipidemia are strongly associated with the presence of NAFLD in T2DM. Routine screening of diabetic patients for NAFLD, particularly through metabolic profiling and liver function tests, is crucial for early identification and management. This would help prevent progression to advanced liver disease and reduce associated cardiovascular risks.

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