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ORIGINAL ARTICLE

# Frequency and sonographic grading of non-alcoholic fatty liver disease (NAFLD) among Type 2 Diabetics presenting to a DHQ Hospital: A cross-sectional analytical study.

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**ABSTRACT… Objective:** To determine the frequency and sonographic grading of non-alcoholic fatty liver disease among adults with type 2 diabetes mellitus and evaluate associations with demographic, anthropometric and biochemical parameters. **Study Design:** Analytical Cross sectional. **Period:** June 2024 to December 2025. **Setting:** Department of Medicine, Bahawal Victoria Hospital Bahawalpur & Department of Radiology, District Headquarters Hospital Lodhran. **Methods:** Three hundred seventy-four eligible diabetic adults were enrolled via consecutive non random sampling. Exclusion criteria encompassed chronic alcohol consumption, viral hepatitis, and hepatotoxic medication use and body mass index exceeding 40 kg /m$^2$. Standardized anthropometric assessments and fasting laboratory evaluations were conducted alongside blinded abdominal ultrasonography. Hepatic steatosis severity was classified using the Saverymuttu echogenicity protocol. Multivariable logistic regression identified independent predictors. **Results:** The overall sonographically confirmed NAFLD frequency was 62.3% (95% CI: 57.2–67.2). Sonographic grading distribution included mild (24.1%), moderate (25.9%), and severe (12.3%) steatosis. Multivariable analysis confirmed elevated body mass index (adjusted OR: 1.28), hypertriglyceridemia (adjusted OR: 1.01), uncontrolled glycated hemoglobin (adjusted OR: 1.31), and comorbid hypertension (adjusted OR: 1.89) as significant predictors. **Conclusion:** Non-alcoholic fatty liver disease affects nearly two-thirds of type 2 diabetic patients in this district hospital setting, predominantly manifesting as moderate steatosis. Adiposity, dyslipidemia, glycemic instability and vascular comorbidities independently predict hepatic fat accumulation. Implementing systematic sonographic screening within routine diabetes management facilitates timely risk stratification and integrated metabolic care.

**Key words:** Diabetes Mellitus, Fatty Liver, Hypertriglyceridemia, Obesity, Risk Factors, Type 2, Ultrasonography.

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## INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) represents a clinicopathological spectrum that begins with isolated hepatic steatosis and may progress to non-alcoholic steatohepatitis (NASH), bridging fibrosis, cirrhosis and ultimately hepatocellular carcinoma.[1] The disease is fundamentally rooted in metabolic dysfunction, characterized by ectopic lipid deposition, chronic low-grade inflammation, and oxidative stress within hepatocytes. Recent consensus statements emphasize that NAFLD is not merely a hepatic marker of metabolic syndrome but an active contributor to systemic insulin resistance and cardiovascular risk.[2]

The burden of NAFLD is disproportionately higher in individuals with type 2 diabetes mellitus (T2DM). Systematic analyses estimate that 55% to 70% of diabetic patients harbor radiological or histological evidence of hepatic steatosis, a prevalence nearly double that of the general adult population.[3] This synergistic relationship is mediated by multiple pathophysiological pathways, including chronic hyperinsulinemia, increased peripheral lipolysis, impaired hepatic fatty acid oxidation and activation of pro-inflammatory cytokines (e.g., TNF-α, IL-6).[4] Over time, lipotoxicity and mitochondrial dysfunction drive hepatocellular injury, triggering fibrogenic pathways that accelerate disease progression.

Despite the high clinical stakes, NAFLD remains underdiagnosed in routine diabetic care, particularly in resource constrained healthcare environments.

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Abdominal ultrasonography continues to serve as the primary screening modality in secondary and district level hospitals due to its wide availability, non-invasive profile and cost-effectiveness.[5] Standardized sonographic grading protocols classify steatosis into three tiers based on parenchymal echogenicity relative to the renal cortex, clarity of the diaphragmatic outline and degree of posterior acoustic attenuation. While ultrasound lacks sensitivity for early fibrosis, its grading system provides a rapid, clinically actionable framework for risk stratification and follow-up planning in settings where transient elastography or magnetic resonance elastography are inaccessible.[6]

A significant evidence gap persists regarding NAFLD frequency and sonographic severity among T2DM patients managed at district headquarters (DHQ) hospitals. Most published epidemiological data originate from tertiary referral centers or high income healthcare systems, limiting their applicability to primary and secondary care networks where the majority of diabetic populations receive routine follow-up. DHQ facilities often operate under diagnostic constraints, yet they represent critical touchpoints for early metabolic complication detection. Without localized prevalence data and validated sonographic grading distributions, healthcare planners cannot design targeted screening pathways, allocate imaging resources efficiently, or integrate hepatic risk assessment into standard diabetes management protocols. Generating context specific evidence from DHQ hospitals is therefore essential to bridge the diagnostic divide and enable timely, preventive interventions.

## OBJECTIVE

To determine the frequency and sonographic grading of non-alcoholic fatty liver disease (NAFLD) among adults with type 2 diabetes mellitus presenting to a DHQ hospital, and to evaluate its association with demographic factors (age, sex), anthropometric indices (BMI, waist circumference), and diabetes duration.

## METHODS

This cross-sectional analytical study was conducted at Medicine department of Bahawal Victoria Hospital Bahawalpur & the Department of Radiology, District Headquarters (DHQ) Hospital Lodhran, from June 2024 to December 2025 after taking ethical approval (Reference No. 351/DME/QAMC, BAHAWALPUR Dated. 25-05-2024) and informed consent from each participant. The minimum sample size was calculated at anticipated NAFLD prevalence of 58.47%[7], confidence level 95%, and absolute margin of error 5% was 374 participants. Eligible patients were enrolled using non-probability consecutive sampling. Adult patients aged 18 years or older with a confirmed diagnosis of type 2 diabetes mellitus were eligible for enrollment. Participants were required to be clinically stable and physically capable of tolerating a standardized abdominal ultrasound examination. Individuals were excluded if they reported chronic alcohol consumption, tested positive for hepatitis B surface antigen or hepatitis C core antibodies, or carried a documented history of chronic liver disease, cirrhosis, or prior hepatic resection. Additional exclusion criteria encompassed concurrent use of established hepatotoxic agents (including amiodarone, methotrexate, tamoxifen, and valproate), pregnancy at the time of assessment and a body mass index greater than 40 kg/m$^2$, which was anticipated to severely limit ultrasound beam penetration and compromise image interpretation quality.

A standardized proforma was utilized to capture demographic and clinical variables, including age, gender, duration of diabetes, current antidiabetic pharmacotherapy, comorbid diagnoses (hypertension, dyslipidemia, ischemic heart disease) and tobacco use history. Anthropometric assessments were performed using calibrated digital scales and non-elastic measuring tapes; weight and standing height were recorded to compute body mass index (BMI). Fasting venous blood specimens were obtained following an 8–10 hour overnight fast and analyzed for fasting plasma glucose, glycated hemoglobin (HbA1c), complete lipid profile (total cholesterol, triglycerides, HDL-C, LDL-C), hepatic transaminases (ALT, AST, GGT), and renal function markers (serum creatinine, estimated GFR). All biochemical assays were processed in the hospital's laboratory. Abdominal ultrasonography was performed using a high-frequency convex transducer (3.5–5.0 MHz) integrated into a contemporary diagnostic ultrasound system. Examinations

were conducted with patients in a supine position following fasting to optimize hepatic visualization. Scanning adhered to standardized longitudinal and transverse planes across right and left hepatic lobes. Hepatic steatosis was classified using the validated Saverymuttu echogenicity grading system: Grade 0 represented normal parenchymal echotexture, Grade 1 (mild) demonstrated mildly increased echogenicity with preserved diaphragmatic and portal vein wall visualization, Grade 2 (moderate) exhibited moderately increased echogenicity with slight blurring of vascular margins and diaphragmatic contour and Grade 3 (severe) was defined by marked parenchymal brightness, significant posterior acoustic attenuation, and poor or absent visualization of intrahepatic vessels and the diaphragm. To mitigate assessment bias, the operating radiologist remained blinded to all anthropometric, biochemical, and clinical data at the time of scanning.

Data management and statistical computations were executed using SPSS Statistics version 25.0. Continuous variables were evaluated for distributional normality via the Shapiro-Wilk test; parametric data were summarized as mean ± standard deviation, while non-parametric variables were reported as median with interquartile range. Categorical outcomes were expressed as frequencies and percentages. Unadjusted associations between NAFLD status/grading and independent predictors were examined using Chi-square or Fisher's exact test for categorical variables, and independent samples t-test or Mann-Whitney U test for continuous measures. Covariates yielding p-value <0.20 in bivariate analysis, alongside clinically established metabolic risk factors, were entered into multivariable binary logistic regression to identify independent determinants of NAFLD presence. Disease severity grading was evaluated using logistic regression to assess monotonic relationships with anthropometric and laboratory indices. Statistical significance was established at p-value <0.05, with all effect estimates reported alongside 95% confidence intervals.

## RESULTS

The mean age of the study participants was 52.3 ± 9.7 years with slight female predominance (54.3%). The median duration of diabetes was 7.0 years (IQR: 4.0–11.0) and the majority of participants were on oral antidiabetic agents (86.1%), with metformin being the most frequently prescribed therapy. Hypertension and dyslipidemia were the most prevalent comorbidities, affecting 68.2% and 61.5% of participants, respectively (Table-I).

The overall frequency of NAFLD, as determined by abdominal ultrasonography using the Saverymuttu grading system, was 62.3% among adults with type 2 diabetes. Among those diagnosed with NAFLD, the distribution of sonographic severity was as follows: Grade 1 (mild steatosis) accounted for 38.7%, Grade 2 (moderate steatosis) for 41.4%, and Grade 3 (severe steatosis) for 19.9% of cases. No participant was classified as Grade 0 within the NAFLD-positive subgroup (Table-II).

Participants with NAFLD had significantly higher mean BMI (29.8 ± 4.1 vs. 26.2 ± 3.9 kg/m$^2$, p value <0.001), waist circumference (102.4 ± 9.3 vs. 94.1 ± 8.7 cm, p value <0.001), fasting plasma glucose (168.3 ± 42.5 vs. 151.2 ± 38.9 mg/dL, p-value=0.002), HbA1c (8.4 ± 1.6 vs. 7.8 ± 1.4%; p value <0.001), triglycerides (186.4 ± 78.2 vs. 142.3 ± 61.5 mg/dL, p value <0.001) and ALT levels (48.7 ± 22.3 vs. 36.2 ± 18.1 U/L, p value <0.001) compared to those without hepatic steatosis. No significant differences were observed for age, sex, diabetes duration, smoking status, HDL-C, LDL-C, or renal function parameters (Table-III).

Multivariable binary logistic regression analysis, adjusting for covariates with p<0.20 in bivariate screening and established clinical relevance, identified four independent predictors of NAFLD presence among adults with type 2 diabetes. After controlling for potential confounders, elevated BMI (adjusted OR: 1.28 per 1 kg/m$^2$ increment; 95% CI: 1.19–1.38, p<0.001), higher triglyceride levels (adjusted OR: 1.01 per 1 mg/dL increment; 95% CI: 1.00–1.02; p value=0.012), elevated HbA1c (adjusted OR: 1.31 per 1% increment; 95% CI: 1.14–1.51; p value <0.001) and presence of hypertension (adjusted OR: 1.89; 95% CI: 1.18–3.02; p=0.008) remained significantly associated with increased odds of NAFLD (Table-IV).

**TABLE-I**

**Baseline demographic, clinical, anthropometric and laboratory characteristics of adults with type 2 diabetes mellitus (n=374)**

| Characteristic | Total (n=374) | NAFLD Absent (n=141) | NAFLD Present (n=233) | P-Value |
|---|---|---|---|---|
| **Demographics** | | | | |
| Age, years, mean ± SD | 52.3 ± 9.7 | 51.8 ± 10.2 | 52.6 ± 9.4 | 0.44 |
| Female sex, n (%) | 203 (54.3) | 78 (55.3) | 125 (53.6) | 0.75 |
| Diabetes duration, years, median (IQR) | 7.0 (4.0–11.0) | 6.5 (3.5–10.0) | 7.0 (4.0–11.5) | 0.28 |
| **Anthropometry** | | | | |
| BMI, kg/m², mean ± SD | 28.5 ± 4.3 | 26.2 ± 3.9 | 29.8 ± 4.1 | <0.001 |
| Waist circumference, cm, mean ± SD | 99.3 ± 10.1 | 94.1 ± 8.7 | 102.4 ± 9.3 | <0.001 |
| **Glycemic parameters** | | | | |
| Fasting glucose, mg/dL, mean ± SD | 162.1 ± 41.8 | 151.2 ± 38.9 | 168.3 ± 42.5 | 0.002 |
| HbA1c, %, mean ± SD | 8.2 ± 1.6 | 7.8 ± 1.4 | 8.4 ± 1.6 | <0.001 |
| **Lipid profile** | | | | |
| Total cholesterol, mg/dL, mean ± SD | 198.4 ± 42.3 | 195.2 ± 40.1 | 200.3 ± 43.6 | 0.26 |
| Triglycerides, mg/dL, mean ± SD | 170.2 ± 74.5 | 142.3 ± 61.5 | 186.4 ± 78.2 | <0.001 |
| HDL-C, mg/dL, mean ± SD | 42.1 ± 9.8 | 43.2 ± 10.1 | 41.4 ± 9.6 | 0.09 |
| LDL-C, mg/dL, mean ± SD | 122.6 ± 35.4 | 120.8 ± 33.9 | 123.7 ± 36.2 | 0.45 |
| **Liver enzymes** | | | | |
| ALT, U/L, mean ± SD | 44.3 ± 21.5 | 36.2 ± 18.1 | 48.7 ± 22.3 | <0.001 |
| AST, U/L, mean ± SD | 38.9 ± 16.2 | 37.1 ± 15.3 | 40.1 ± 16.7 | 0.08 |
| GGT, U/L, median (IQR) | 42.0 (28.0–65.0) | 38.0 (25.0–58.0) | 45.0 (30.0–68.0) | 0.03 |
| **Comorbidities, n (%)** | | | | |
| Hypertension | 255 (68.2) | 87 (61.7) | 168 (72.1) | 0.04 |
| Dyslipidemia | 230 (61.5) | 82 (58.2) | 148 (63.5) | 0.31 |
| Ischemic heart disease | 67 (17.9) | 24 (17.0) | 43 (18.5) | 0.73 |
| Current smoker | 89 (23.8) | 35 (24.8) | 54 (23.2) | 0.72 |

**TABLE-II**

**Frequency and sonographic grading of non-alcoholic fatty liver disease among adults with type 2 diabetes mellitus (n=374)**

| Sonographic Grade (Saverymuttu Classification) | Description | Frequency | Percentage |
|---|---|---|---|
| Grade 0 | Normal hepatic echogenicity | 141 | 37.7% |
| Grade 1 | Mild steatosis: slightly increased echogenicity, preserved diaphragm/vascular margins | 90 | 24.1% |
| Grade 2 | Moderate steatosis: moderate echogenicity, slightly impaired diaphragm/portal vein visualization | 97 | 25.9% |
| Grade 3 | Severe steatosis: marked echogenicity, posterior attenuation, poor vessel/diaphragm visualization | 46 | 12.3% |
| Total NAFLD (Grades 1–3) | | 233 | 62.3% |

**TABLE-III**

**Bivariate associations between selected variables and presence of non-alcoholic fatty liver disease among adults with type 2 diabetes mellitus (n=374)**

| Variable | Category/Unit | NAFLD Absent (n=141) | NAFLD Present (n=233) | Unadjusted OR (95% CI) | P-Value |
|---|---|---|---|---|---|
| Age | Per 1-year increment | 51.8 ± 10.2 | 52.6 ± 9.4 | 1.01 (0.99–1.03) | 0.44 |
| Sex | Female vs. Male | 78/63 | 125/108 | 0.94 (0.62–1.42) | 0.75 |
| BMI | Per 1 kg/m² increment | 26.2 ± 3.9 | 29.8 ± 4.1 | 1.27 (1.19–1.35) | <0.001 |
| Waist circumference | Per 1 cm increment | 94.1 ± 8.7 | 102.4 ± 9.3 | 1.11 (1.08–1.14) | <0.001 |
| Diabetes duration | Per 1-year increment | 6.5 (3.5–10.0) | 7.0 (4.0–11.5) | 1.02 (0.98–1.06) | 0.28 |
| Fasting glucose | Per 10 mg/dL increment | 151.2 ± 38.9 | 168.3 ± 42.5 | 1.05 (1.02–1.08) | 0.002 |
| HbA1c | Per 1% increment | 7.8 ± 1.4 | 8.4 ± 1.6 | 1.35 (1.19–1.53) | <0.001 |
| Triglycerides | Per 10 mg/dL increment | 142.3 ± 61.5 | 186.4 ± 78.2 | 1.07 (1.04–1.10) | <0.001 |
| HDL-C | Per 1 mg/dL increment | 43.2 ± 10.1 | 41.4 ± 9.6 | 0.98 (0.96–1.00) | 0.09 |
| ALT | Per 10 U/L increment | 36.2 ± 18.1 | 48.7 ± 22.3 | 1.32 (1.18–1.48) | <0.001 |
| Hypertension | Yes vs. No | 87/54 | 168/65 | 1.63 (1.04–2.55) | 0.04 |
| Current smoker | Yes vs. No | 35/106 | 54/179 | 0.92 (0.58–1.46) | 0.72 |

**TABLE-IV**

**Multivariable binary logistic regression analysis identifying independent predictors of non-alcoholic fatty liver disease among adults with type 2 diabetes mellitus (n=374)**

| Predictor Variable | Adjusted OR | 95% CI | P-Value |
|---|---|---|---|
| BMI (per 1 kg/m² increment) | 1.28 | 1.19–1.38 | <0.001 |
| Triglycerides (per 1 mg/dL increment) | 1.01 | 1.00–1.02 | 0.012 |
| HbA1c (per 1% increment) | 1.31 | 1.14–1.51 | <0.001 |
| Hypertension (Yes vs. No) | 1.89 | 1.18–3.02 | 0.008 |
| Age (per 1-year increment) | 1.00 | 0.97–1.03 | 0.92 |
| Gender (Female vs. Male) | 0.88 | 0.55–1.41 | 0.60 |
| Diabetes duration (per 1-year increment) | 1.01 | 0.97–1.05 | 0.68 |
| ALT (per 10 U/L increment) | 1.08 | 0.94–1.24 | 0.28 |
| Current smoker (Yes vs. No) | 0.95 | 0.57–1.58 | 0.84 |

## DISCUSSION

The present investigation demonstrates a substantial burden of hepatic steatosis among adults with type 2 diabetes mellitus attending a secondary-care facility, with an overall NAFLD frequency of 62.3%. This burden closely mirrors recent South Asian epidemiological reports, which consistently document NAFLD rates exceeding 55% in diabetic patients, substantially higher than figures reported in Western populations where metabolic phenotypes and genetic predispositions differ.[8,9] The higher burden observed at DHQ Hospital Lodhran likely reflects the synergistic impact of uncontrolled glycemia, central adiposity and high rates of concomitant dyslipidemia and hypertension, all of which are increasingly recognized as core components of metabolic dysfunction in resource constrained healthcare environments.[10] The predominance of moderate steatosis (Grade 2, 41.4%) over mild or severe categories aligns with the progressive nature of lipid accumulation in longstanding diabetes, suggesting that a significant proportion of patients may be transitioning from simple steatosis toward steatohepatitis or early fibrotic remodeling.[11]

Multivariable analysis identified body mass index, triglyceride concentration, glycated hemoglobin, and hypertension as independent determinants of NAFLD presence, reinforcing the concept that

hepatic fat deposition in diabetes is driven by systemic metabolic derangement rather than isolated glycemic excursions. The association between BMI and NAFLD underscores the pathogenic role of adipose tissue expansion, increased free fatty acid flux to the liver and subsequent lipotoxic hepatocellular injury.[12] Similarly, the independent contribution of elevated triglycerides and HbA1c reflects impaired insulin-mediated suppression of hepatic VLDL synthesis and chronic hyperglycemia-driven de novo lipogenesis.[13] Hypertension emerged as a significant predictor, likely serving as a surrogate marker for endothelial dysfunction, systemic inflammation and activated renin-angiotensin-aldosterone signaling, all of which exacerbate hepatic microvascular congestion and oxidative stress.[14] Notably, alanine aminotransferase, while significantly elevated in unadjusted comparisons, lost statistical significance after multivariable adjustment, highlighting the well-documented limitation of transaminases as standalone screening tools, given their poor sensitivity for detecting isolated steatosis in metabolically complex diabetic populations.[15]

Several methodological strengths enhance the validity and clinical applicability of these findings. The study was conducted in district hospital setting, capturing representative cross-section of diabetic patients who typically navigate primary and secondary care pathways. Consecutive sampling minimized selection bias, while the use of a validated sonographic grading system with radiologist blinding reduced assessment subjectivity. Furthermore, the inclusion of readily available anthropometric and routine laboratory parameters ensures that the identified predictors can be immediately integrated into existing diabetic follow-up protocols without requiring specialized infrastructure.[16]

Despite these advantages, certain limitations warrant consideration. The cross-sectional design precludes causal inference and longitudinal tracking of steatosis progression or regression. Ultrasound, while highly practical for resource limited environments, lacks the capacity to reliably differentiate simple steatosis from non-alcoholic steatohepatitis or quantify fibrosis, potentially underestimating the proportion of patients at imminent risk for advanced liver disease.[17] The exclusion of individuals with BMI >40 kg/m$^2$, while necessary for technical imaging feasibility, may have slightly attenuated the true burden. The single-center nature of the study also limits broad generalizability across diverse geographic and socioeconomic strata within Pakistan.[18]

To address these challenges, district hospitals should institutionalize routine abdominal ultrasonography as part of annual metabolic risk assessments for all adults with type 2 diabetes, particularly those with elevated BMI, poor glycemic control, hypertriglyceridemia, or concomitant hypertension. Standardized reporting templates incorporating sonographic grading should be adopted to facilitate longitudinal monitoring and timely referral to hepatology services when moderate-to-severe steatosis is identified. Future research should prioritize prospective cohort designs integrating vibration-controlled transient elastography or serum fibrosis biomarkers to validate ultrasound findings and establish progression trajectories. Health systems evaluations examining the cost-effectiveness of targeted screening algorithms within Pakistani secondary care networks would further inform policy level resource allocation.[19,20]

## CONCLUSION

Non-alcoholic fatty liver disease affects nearly two-thirds of adults with type 2 diabetes in this district hospital setting, with moderate steatosis predominating and independently predicted by elevated BMI, hypertriglyceridemia, poor glycemic control, and hypertension. Routine sonographic screening and integrated metabolic management are essential to mitigate hepatic disease progression in resource-constrained diabetic care pathways.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

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| AUTHORSHIP AND CONTRIBUTION DECLARATION | |
|---|---|
| 1 | **Zarmina Irum:** Conception of idea, study design, drafting. |
| 2 | **Muhammad Naveed Iqbal:** Data collection, data analysis. |
| 3 | **Fahad Qaisar:** Interpretation of data. |