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Significance of Elevated Liver Enzymes with Normal Viral Serology for Hepatitis B and C in Type 2 Diabetes Mellitus

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ABSTRACT

OBJECTIVE: To ascertain the frequency of increased liver enzymes in individuals with type 2 diabetes (T2DM) who have normal hepatitis B and C serology.

METHODOLOGY: This cross-sectional study was conducted at the Department of General Medicine, Dr. Ruth K.M. Pfau Civil Hospital, Karachi, from June to November 2025. A total of 189 adults aged 18–65 years with at least 5 years of T2DM and normal hepatitis B and C serology were enrolled. Elevated liver enzymes were defined as ALT >40 U/L, AST >40 U/L, ALP >120 U/L, GGT >40 U/L, or total bilirubin >1.3 mg/dL. Poor glycemic control was defined as HbA1c ≥7%. Data analyzed with SPSS version 26.

RESULTS: The average age was 46.9±13.1 years; 125 (66.1%) were female. Elevated liver enzymes were observed in 109 patients (57.7%). Poor glycemic control (HbA1c ≥7%) was significantly associated with a higher frequency of elevated liver enzymes (69.2% vs 43.5%, p<0.001), higher mean ALT (47.2 vs 38.1 U/L, p < 0.001), and higher mean GGT (49.8 vs 38.4 U/L, p < 0.001). U / L, p<0.001), and higher mean GGT (49.8 vs 38.4 U/L, p<0.001) compared with good glycemic control.

CONCLUSION: Elevated liver enzymes are frequently observed (57.7%) among patients with T2DM with normal hepatitis B and C serology. Poor glycemic control is significantly associated with liver enzyme abnormalities. Clinicians should consider screening all patients with T2DM for liver dysfunction, particularly those with suboptimal glycemic control. However, the cross-sectional design precludes causal inferences, and the absence of confirmatory imaging means these biochemical abnormalities should be interpreted as markers of potential liver involvement rather than confirmed MASLD/NAFLD.

KEYWORDS: type diabetes mellitus, liver enzymes, hepatitis B, hepatitis C, glycemic control, HbA1c, NAFLD, MASLD

INTRODUCTION

Chronic hyperglycemia, a metabolic illness known as diabetes (DM), results from an imbalance in insulin production or action, or both. This condition is a major contributor to the complications of diabetes, which can lead to long-term damage and failure in several organ systems[1]. The International Diabetes Federation (IDF) reported that 589 million individuals worldwide lived with diabetes (DM) in 2024[2].

This marks a significant increase from previous estimates and represents approximately one in nine adults globally[3]. In addition, the IDF estimates that this figure will grow to 853M by 2050 if left unchanged[4].

Due to its long-term health effects and financial cost on the National Health Service, the occurrence of liver dysfunction in Type II DM patients has garnered considerable attention[5]. Numerous liver disorders, such as cirrhosis, hepatocellular carcinoma, and nonalcoholic fatty liver disease (NAFLD), have been linked to Type 2 diabetes mellitus in epidemiologic studies[6,7]. A large number of deaths in type 2 diabetes are attributed to these liver disorders[8]. Liver complications represent an important clinical concern in the diabetic population and may contribute to premature mortality. Because liver enzyme derangements often reflect hepatocellular injury or cholestasis, they are commonly used as practical markers of liver involvement in NAFLD[9]. For this reason, liver enzymes have been studied as potential indicators in diabetes-related risk assessment. Several investigations have demonstrated higher liver enzyme levels in patients with diabetes; however, results vary between studies and settings[10]. While some authors have reported elevations in AST, ALT, and GGT, others observed increases in GGT, ALT, and ALP, with AST remaining unchanged[11]. Alam et al revealed that 62.53% of T2DM patients with normal serology for hepatitis B and C had abnormal liver enzyme activity[12]. A study carried out in a tertiary care hospital in Abbottabad reported a high frequency of elevated liver enzymes in patients with T2DM. 60% [13]. A study by Noroozi KM[10] showed that the elevated AST, ALT, GGT and ALP levels among T2DM patients were 4.38%, 11.40%, 16.71%, and 15.12%, respectively.

The purpose of this study is to determine the frequency of elevated liver enzymes in patients with type 2 diabetes mellitus who have normal serology for hepatitis B and C.

This investigation addresses a critical clinical gap. Type 2 diabetes affects over 33 million people in Pakistan, yet liver dysfunction—a common and potentially fatal complication—remains grossly underrecognized. Unlike retinopathy or nephropathy, liver disease in people with diabetes is often asymptomatic and detected only incidentally. When a diabetic patient with normal hepatitis B and C serology shows elevated liver enzymes, the finding is frequently dismissed as medication-related or idiopathic, allowing underlying metabolic dysfunction-associated steatotic liver disease (MASLD) or NAFLD to progress silently to cirrhosis or hepatocellular carcinoma. However, elevated liver enzymes can also result from other causes, including medication effects (statins, certain antidiabetic agents), other metabolic conditions, and drug-induced liver injury, which were not exhaustively evaluated in this study.

Alarmingly, local evidence is scarce. While international studies report associations between diabetes and liver enzyme elevations, data from Pakistan—especially Sindh—are limited to a few small studies with variable findings. Without population-specific evidence, clinicians lack guidelines for screening diabetic patients for liver dysfunction. This study will provide the first robust estimate of elevated liver enzyme frequency in type 2 diabetic patients with normal viral serology at a major Karachi tertiary care hospital. The findings may inform screening protocols, enable early intervention, and potentially reduce the silent burden of diabetes-related liver disease in Pakistan.

METHODOLOGY

We conducted this cross-sectional research at the Dr Ruth K.M. Pfau Civil Hospital in Karachi from June to November 2025. Using non-probability consecutive sampling, of selected adult patients aged 18-65 years with a history of type 2 diabetes mellitus (T2DM) for at least 5 years and normal serology for hepatitis B (negative HBsAg) and hepatitis C (negative anti-HCV) from the outpatient department. Patients with a history of malignancy, pregnancy, autoimmune disease, immunocompromised states, chronic liver disease, renal failure, or known alcohol use disorder (defined as regular consumption exceeding 14 units/week for men or 7 units/week for women) were excluded. The formula for the calculation of a single proportion, $n = (Z^2 \times p \times (1-p)) / d^2$, was used to obtain the sample size. The necessary sample size was determined as $n = (1.96^2 \times 0.60 \times 0.40) / (0.07^2) = (3.8416 \times 0.24) / 0.0049 = 0.921984 / 0.0049 \approx 188.16$, based on a study by Sadia A 2022[13] in Abbottabad, which found that 60% had elevated liver enzymes.
Thus, 189 patients were enrolled. This sample size provides 80% power to detect a large effect size for subgroup comparisons but is insufficient to detect small-to-moderate differences; therefore, non-significant secondary associations should be interpreted with caution.
Alcohol consumption and medication lists (including metformin, sulfonylureas, insulin, statins, NSAIDs, and other potential hepatotoxic agents) were recorded using a standardized questionnaire. Glycemic control was assessed using the most recent HbA1c value obtained within 3 months of enrollment, with poor glycemic control defined as HbA1c ≥7%. Elevated liver enzymes were defined as the presence of any of the following: total bilirubin >1.3 mg/dL, alanine aminotransferase (ALT) >40 U/L, aspartate aminotransferase (AST) >40 U/L, alkaline phosphatase (ALP) >120 U/L, or gamma-glutamyl transferase (GGT) >40 U/L. Hypertension was defined as a documented diagnosis with use of antihypertensive medications for at least six months. Based on past smoking habits, smoking status was classified as either current, ex-smoker, or nonsmoker. Weight (kg) and height (m) were measured using standard instruments, and BMI was calculated. Venous blood was drawn into 5 mL sterile blood bags and taken to the institutional laboratory for analysis of liver function tests and hepatitis B and C serology. Data were collected at one point in time only (no follow-up visits were needed) using a predesigned proforma.
The Research Evaluation Unit (CPSP) approved the study protocol (Ref No: CPSP/REU/MED-2022-183-19155) dated April 23, 2025, before the start of data collection. We obtained written informed consent from all patients, and we thoroughly discussed the objectives, procedures, risks, and benefits. Patients were told there would be no repercussions for their medical care if they left the research at any point. All collected data were kept confidential, and no personal identifiers were included in the final analysis or manuscript.

Statistical Analysis

We analyzed the data using the Statistical Package for the Social Sciences version 26. We computed demographic and clinical variables as descriptive statistics. All continuous variables (age, duration of diabetes, BMI, and liver enzyme levels) were presented as mean ± standard deviation (SD) with values within 95% confidence intervals (CIs), and all categorical variables (gender, residence, hypertension, smoking, family history of liver disease, and elevated liver enzyme status) were presented as numbers and percentages. We used an independent t-test to compare continuous variables between patients with raised liver enzymes and those with normal liver enzymes, and between patients with poor glycemic control and those with good glycemic

control. The X^2 test was applied for qualitative data. We performed multivariable logistic regression to identify independent predictors of elevated liver enzymes, adjusting for age, gender, diabetes duration, BMI, hypertension, smoking status, family history of liver disease, and medication use (statins, metformin, insulin, sulfonylureas). *We considered p <0.05 significant*.

RESULTS

Of enrolled 189 patients with type 2 diabetes and normal hepatitis B and C serology. The average age of the participants was 46.90±13.15 years (95% CI, 45.01–48.79), and the average duration of diabetes was 10.10±5.34 years (95% CI, 9.33–10.87). The average BMI was 26.39±5.72 kg/m2, indicating that the body mass index was overweight on average. Among liver function parameters, mean ALT was 43.07±14.02 U / L and mean GGT was 44.75±17.94 U/L, both exceeding normal reference limits, while mean AST was 39.24±16.26 U / L and mean ALP was 110.05±36.41 U/L. Categorically, 125 patients (66.1%) were female, 136 (72.0%) resided in urban areas, 102 (54.0%) had hypertension, 61 (32.3%) were smokers, and 29 (15.3%) reported a family history of liver disease. Regarding medication use, 142 patients (75.1%) were on metformin, 87 (46.0%) on sulfonylureas, 53 (28.0%) on insulin, and 68 (36.0%) on statins. These baseline data are consistent with an under- or middle-aged cohort of predominantly female, overweight type 2 diabetic patients with significant metabolic comorbidities **(Table I).**

The mean age was similar between the elevated (46.65±13.62 years) and normal (47.24±12.57 years) groups, with no statistically significant difference ($p = 0.763$). Duration of diabetes was longer in the elevated group (10.68±5.45 vs 9.31±5.12 years), but this difference did not reach statistical significance ($p = 0.082$). BMI was nearly identical (26.30 vs 26.52 kg/m², $p = 0.794$). The proportion of females was slightly lower in the elevated group (64.2% vs 68.8%, $p = 0.516$). Hypertension was less frequent in the elevated group (50.5% vs 58.8%, $p = 0.259$), whereas smoking was more common (34.9% vs 28.7%, $p = 0.375$). Family history of liver disease was slightly higher in the elevated group (16.5% vs 13.8%, $p = 0.602$). Medication use did not differ significantly between groups: metformin (73.4% vs 77.5%, $p = 0.521$), sulfonylureas (44.0% vs 48.8%, $p = 0.517$), insulin (26.6% vs 30.0%, $p = 0.611$), and statins (34.9% vs 37.5%, $p = 0.709$)**.** We found no significant differences for these parameters (all $p > 0.05$), suggesting that elevated liver enzymes occur independently of these demographic and clinical variables in this T2DM population **(Table II).**

Table III compares liver enzyme abnormalities between patients with poor glycemic control (HbA1c ≥ 7%, n = 104) and good glycemic control (HbA1c < 7%, n = 85). Any elevated liver enzyme was significantly more frequent in the poor-control group (69.2% vs 43.5%, $p < 0.001$, Cramér's V = 0.26). Isolated ALT elevation was also significantly higher in the poor-control group (23.1% vs 11.8%, $p = 0.042$, V = 0.15), whereas isolated GGT elevation showed a similar trend but was not significant (17.3% vs 8.2%, $p = 0.065$, V = 0.13). Mixed elevation of two or more enzymes was comparable between groups (28.8% vs 23.5%, $p = 0.409$, V = 0.06). More severe elevations, defined as ALT greater than twice the upper limit of normal, occurred significantly more often in the poor control group (13.5% vs 3.5%, $p = 0.018$, V = 0.17), as did GGT greater than twice the upper limit of normal (10.6% vs 2.4%, $p = 0.029$, V = 0.16). Furthermore, mean ALT levels were significantly higher in the poor control group (47.2±15.8 vs 38.1±10.9 U / L, $p < 0.001$, Cohen's d = 0.67), as were mean GGT levels (49.8±19.4 vs 38.4±13.2 U / L, $p < 0.001$, d = 0.68). These findings show that poor glycemic control is

significantly associated with a higher frequency and greater severity of elevated liver enzymes, particularly ALT and GGT, in patients with T2DM with normal hepatitis B and C serology **(Table III).**

Multivariable logistic regression analysis identified poor glycemic control (HbA1c ≥7%) as the only independent predictor of elevated liver enzymes (adjusted OR 2.89, 95% CI: 1.58–5.28, p < 0.001). Age (adjusted OR 1.01, 95% CI: 0.98–1.04, p = 0.523), gender (adjusted OR 0.79, 95% CI: 0.42–1.48, p = 0.461), diabetes duration (adjusted OR 1.04, 95% CI: 0.98–1.10, p = 0.210), BMI (adjusted OR 0.99, 95% CI: 0.94–1.04, p = 0.651), hypertension (adjusted OR 0.72, 95% CI: 0.39–1.34, p = 0.302), smoking (adjusted OR 1.35, 95% CI: 0.69–2.64, p = 0.384), family history (adjusted OR 1.24, 95% CI: 0.51–3.02, p = 0.635), metformin use (adjusted OR 1.04, 95% CI: 0.52–2.08, p = 0.912), sulfonylurea use (adjusted OR 0.87, 95% CI: 0.47–1.61, p = 0.650), insulin use (adjusted OR 0.89, 95% CI: 0.44–1.79, p = 0.744), and statin use (adjusted OR 0.91, 95% CI: 0.48–1.72, p = 0.775) were not independent predictors.

Table I: Demographic and clinical data of patients (n = 189)

Variable	Mean ± SD	95% CI
Age (years)	46.90±13.15	45.01–48.79
Duration of diabetes (years)	10.10±5.34	9.33–10.87
Body mass index (kg/m²)	26.39±5.72	25.57–27.21
Total bilirubin (mg/dL)	0.72±0.48	0.65–0.79
AST (U/L)	39.24±16.26	36.90–41.57
ALT (U/L)	43.07±14.02	41.05–45.08
ALP (U/L)	110.05±36.41	104.83–115.28
GGT (U/L)	44.75±17.94	42.18–47.33
Categorical variable	**Frequency (n)**	**Percentage (%)**
Gender (Female)	125	66.1
Urban residence	136	72.0
Hypertension	102	54.0
Smoking	61	32.3
Family history of liver disease	29	15.3
Metformin use	142	75.1
Sulfonylurea use	87	46.0
Insulin use	53	28.0
Statin use	68	36.0

Table II: Comparison of baseline characteristics by elevated liver enzyme status (n = 189)

Variable	Elevated (n=109)	Normal (n=80)	p value
Age (years)	46.65±13.62	47.24±12.57	0.763
Duration of diabetes (years)	10.68±5.45	9.31±5.12	0.082
BMI (kg/m²)	26.30±5.41	26.52±6.15	0.794
Gender (%)	64.2%	68.8%	0.516
Hypertension (%)	50.5%	58.8%	0.259
Smoking (%)	34.9%	28.7%	0.375
Family history of liver disease (%)	16.5%	13.8%	0.602
Metformin use (%)	73.4%	77.5%	0.521
Sulfonylurea use (%)	44.0%	48.8%	0.517
Insulin use (%)	26.6%	30.0%	0.611
Statin use (%)	34.9%	37.5%	0.709

Table III: Comparison of Liver Enzyme Elevations between T2DM Patients with Poor vs Good Glycemic Control (n = 189)

Parameter	Poor glycemic control (HbA1c ≥ 7%) (n = 104)	Good glycemic control (HbA1c < 7%) (n = 85)	p value
Any elevated liver enzyme	72 (69.2%)	37 (43.5%)	<0.001*
Isolated ALT elevation	24 (23.1%)	10 (11.8%)	0.042*
Isolated GGT elevation	18 (17.3%)	7 (8.2%)	0.065
Mixed elevation (≥2 enzymes)	30 (28.8%)	20 (23.5%)	0.409
ALT > 2× upper limit of normal	14 (13.5%)	3 (3.5%)	0.018*
GGT > 2× upper limit of normal	11 (10.6%)	2 (2.4%)	0.029*
Mean ALT ± SD (U/L)	47.2 ± 15.8	38.1 ± 10.9	<0.001*
Mean GGT ± SD (U/L)	49.8 ± 19.4	38.4 ± 13.2	<0.001*

**P-values are statistically significant*

DISCUSSION

Hepatic dysfunction is a major yet frequently overlooked clinical problem in type 2 diabetes (T2DM), which is frequently a multisystem involvement. The diagnostic dilemma is especially great in those with normal viral hepatitis B and C serology, with liver enzyme abnormalities being often incidental findings in routine laboratory testing. Clinically, it is important to recognize the burden of elevated liver enzymes in this population, as these abnormalities could be markers of underlying metabolic liver disease, like metabolic dysfunction-associated steatotic liver disease (MASLD) or nonalcoholic fatty liver disease (NAFLD), and may lead to long-term morbidity if not diagnosed. However, elevated liver enzymes can also result from other causes, including medication effects (statins, certain antidiabetic agents), other metabolic conditions, and drug-induced liver injury, which were not exhaustively evaluated in this study.

In our research, we sought to evaluate the frequency of elevated liver enzymes among individuals with T2DM with normal levels of hepatitis B and C serology and explore relationships with demographic and clinical parameters. This discussion will outline and provide interpretation of key findings from each of these tables and compare and contrast them with regional and international literature published between 2021 and 2026, to include clinical implications within the context of the study's rationale and objectives.

Normal viral serology for HBsAg, anti-HBsAg, and anti-hepatitis C antibodies was observed in 189 patients with T2DM; 109 of them (57.7%) presented with elevated liver enzymes. The high prevalence shows the significant burden of biochemical liver involvement in diabetic patients even in the absence of viral hepatitis. This finding is similar to data from the region after 2020. A longitudinal study by Alia A et al.14 in a tertiary care hospital in Lahore revealed abnormal liver function tests in 53.6% of T2DM patients with negative viral serology, similar to our finding of abnormal LFTs in 57.7% of T2DM patients with negative viral serology[14]. Similarly, Alam S et al.[15] observed abnormal liver enzyme activity in 62.5% of patients with T2DM with normal hepatitis serology from a longitudinal study conducted in North India.[15]

A national study by Ahamed F et al.16 found elevated ALT in 55.8% of T2DM patients, similar to our results[16]. The consistency across South Asian studies suggests a regionally high burden of diabetes-related liver involvement, likely attributable to genetic susceptibility, dietary patterns, and the metabolic syndrome epidemic.

The prevalence observed in our study is substantially higher than that reported in some population-based studies from Western countries. A large European cohort study found that only 28.4% of patients with T2DM had elevated ALT, increasing to 42.1% when GGT was included.[17] The discrepancy between South Asian and Western populations may be explained by the higher metabolic risk at lower BMI levels observed in South Asians, a phenomenon well-documented in recent literature[18].

Notably, a recent Pakistani study by Zahoor F et al.[19] conducted in Gujranwala, Pakistan, reported that 57.9% of T2DM patients had ultrasound-confirmed MASLD, with elevated liver enzymes present in 58.1% of those patients[19]. The close agreement between our biochemical findings and their imaging-confirmed prevalence supports the validity of using liver enzymes as screening markers in resource-limited settings where ultrasound may not be readily available. However, without imaging confirmation in our study, we cannot definitively attribute the enzyme elevations to MASLD/NAFLD, as other etiologies, including drug-induced liver injury and other metabolic conditions, could contribute.

The average age was 46.90 years (95% CI, 45.01–48.79), and the average duration of diabetes was 10.10±5.34 years (95% CI, 9.33–10.87). These are all middle-aged and have long-term diabetes. The average BMI was 26.39 kg/m², which is classified as overweight. This aligns with the established link between T2DM and higher levels of adiposity but is significantly lower than those seen in Western countries, where the mean BMI is frequently over 30 kg/m² [20].

Mean ALT level was 43.07±14.02 U/L while mean GGT was 44.75±17.94 U/L, both above the upper limits of normal (40 U / L for both). Mean AST (39.24±16.26 U / L) and ALP (110.05 ± 36.41 U / L) were near or slightly above the reference ranges. The disproportionate elevation of ALT and GGT relative to AST is characteristic of hepatocellular injury rather than cholestasis and is highly suggestive of MASLD as the underlying pathology[21].

Categorically, 125 patients (66.1%) were female, 136 (72.0%) resided in urban areas, 102 (54.0%) had hypertension, 61 (32.3%) were smokers, and 29 (15.3%) reported a family history of liver disease. The female predominance mirrors that reported by Latif S 2024[22] in a cohort from Rawalpindi, where 64.8% of T2DM patients with elevated liver enzymes were female.

The mean age was similar between the elevated (46.65±13.62 years) and normal (47.24±12.57 years) groups ($p = 0.763$). This contrasts with Liao NH et al.[23], who reported that younger age at diabetes diagnosis was associated with a higher risk of MASLD progression. However, our finding is consistent with that of Zahoor F et al.[19] who found no consistent association between age and liver enzyme elevation in South Asian T2DM populations, suggesting ethnic-specific pathophysiology.

Duration of diabetes was longer in the elevated group (10.68±5.45 vs 9.31±5.12 years). Han E et al.[24] confirmed that every 5-year increase in diabetes duration was related to a 15% higher risk of incident MASLD (HR 1.15, 95% CI: 1.07-1.23). Still, this association was attenuated after adjustment for glycemic control.

No significant associations were found with sex ($p = 0.516$). This contrasts with a recent Pakistani study by Khan F 2024[25] that reported male gender as a significant predictor of advanced fibrosis in T2DM patients (OR 2.34, 95% CI: 1.12-4.89). The discrepancy may relate to our predominantly female sample (66.1%) and the cross-sectional design.

Table III presents the most clinically actionable findings of this study, demonstrating a strong and statistically significant association between poor glycemic control (HbA1c ≥ 7%) and both the frequency and severity of liver enzyme elevations.

The poor-control group was significantly more likely to have no elevated liver enzymes than the well-controlled group (69.2% vs 43.5%, $p < 0.001$). Isolated ALT elevation was also significantly higher in poor control (23.1% vs 11.8%, $p = 0.042$). More severe elevations—defined as ALT or GGT exceeding twice the upper limit of normal—occurred significantly more often in the poorly controlled group (ALT >2× ULN: 13.5% vs 3.5%, $p = 0.018$; GGT >2× ULN: 10.6% vs 2.4%, $p = 0.029$). These findings are strongly supported by recent literature. A study by Gursoy O 2025[20] demonstrated that intensive glycemic control (target HbA1c < 6.5%) resulted in a 34% relative reduction in ALT levels after 12 months compared with standard control. Similarly, a large Japanese registry study of T2DM patients by Fukai K et al.[26] found that each 1% increase in HbA1c was associated with a 12% higher odds of having ALT >40 U/L (OR 1.12, 95% CI: 1.08-1.16). From Pakistan, a prospective study by Naguib R 2025[27] followed 450 patients with T2DM over 18 months. It demonstrated that improvement in HbA1c from 8.5% to 7.2% was accompanied by a significant reduction in ALT (from 48.3 to 36.7 U/L, $p < 0.001$) and GGT (from 52.1 to 39.4 U / L, $p < 0.001$).

Multivariable logistic regression identified poor glycemic control as the only independent predictor of elevated liver enzymes (adjusted OR 2.89, 95% CI: 1.58–5.28, $p < 0.001$), while other factors including age, gender, diabetes duration, BMI, hypertension, smoking, family history, and medication use were not significant. This finding is clinically important as it suggests that glycemic optimization may be a key modifiable target to reduce liver enzyme abnormalities in this population, although causal inference requires prospective studies.

Study Limitations

This study has several limitations that should be acknowledged. First, the cross-sectional design precludes establishing causality; we can only demonstrate associations. Second, the absence of confirmatory imaging (ultrasound or FibroScan) means we cannot definitively attribute enzyme elevations to MASLD/NAFLD, as other etiologies, including drug-induced liver injury, other metabolic conditions, or unidentified causes could contribute. Third, while medication use was recorded, the study was not powered to evaluate the hepatotoxic potential of individual drugs comprehensively. Fourth, the single-centre design at a tertiary care hospital limits generalizability to primary care or community settings. Fifth, the sample size, while adequate for the primary objective, was insufficient to detect small-to-moderate effect sizes for subgroup analyses. Sixth, unmeasured confounders such as dietary patterns, physical activity, and genetic factors may influence the observed associations. Finally, alcohol consumption was assessed by self-report, which may be subject to recall and social desirability biases.

CONCLUSION

Elevated liver enzymes were frequently observed (57.7%) among patients with type 2 diabetes despite normal hepatitis B and C serology. Demographic characteristics, diabetes duration, BMI, hypertension, smoking status, and family history of liver disease showed no statistically significant associations with enzyme elevation in bivariate analyses or multivariable regression. Poor glycemic control (HbA1c ≥ 7%) was closely linked to more frequent and severe increases in liver enzymes, especially ALT and GGT, and emerged as the only independent predictor in logistic regression. These findings, consistent with recent South Asian and international literature from 2021-2026, suggest that liver enzyme abnormalities occur commonly in T2DM regardless of traditional risk factors. Clinicians should consider screening all patients with T2DM for liver dysfunction, with particular vigilance in those with suboptimal glycemic control. However, given the cross-sectional design and absence of confirmatory imaging, these biochemical abnormalities should be interpreted as markers of potential liver involvement requiring further evaluation, rather than a confirmed MASLD/NAFLD diagnosis. Future prospective studies incorporating imaging modalities and comprehensive medication assessment are needed to confirm these findings and establish causal pathways.

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AUTHOR CONTRIBUTION

Hassan S: Conceived and designed the study
Seetlani NK: Conceived and designed the study
Nizamani H: Data collection and performed laboratory analysis
Naz T: Data collection and performed laboratory analysis
Chanderkanta: Statistical analysis
Siddiqui SJ: Contributed to study conceptualization, statistical plan, critical revision of the manuscript, and public health interpretation.
All authors reviewed and approved the final manuscript.

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