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Original Article

Prevalence of Elevated Liver Enzymes and Its Association with Diabetes

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ABSTRACT

Background: Diabetes mellitus is a chronic metabolic disorder associated with multisystem complications, including hepatic biochemical dysfunction. Elevated liver enzymes may reflect subclinical hepatocellular injury, metabolic fatty liver disease, insulin resistance, and broader metabolic dysregulation in diabetic individuals. **Objective:** To determine the prevalence of elevated liver enzymes and assess their association with diabetes mellitus, glycemic control, obesity, and dyslipidemia. **Methods:** This comparative cross-sectional study was conducted at the Biochemistry and Pathology Laboratory of Social Security Hospital, Lahore, over four months. A total of 200 participants were enrolled, including 100 diabetic and 100 non-diabetic individuals. After overnight fasting, venous blood samples were collected and analyzed for fasting blood glucose, HbA1c, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, and gamma-glutamyl transferase. Biochemical parameters were compared between groups, and associations between elevated liver enzymes and metabolic risk factors were assessed. **Results:** Diabetic participants had significantly higher mean fasting blood glucose, HbA1c, ALT, AST, ALP, and GGT levels than non-diabetic participants. At least one elevated liver enzyme was observed in 84.0% of diabetic participants compared with 11.0% of non-diabetic participants. Elevated liver enzymes were also more frequent among participants with poor glycemic control, high fasting blood glucose, obesity, and dyslipidemia. **Conclusion:** Elevated liver enzymes were markedly more prevalent among diabetic individuals and were strongly associated with poor glycemic control and metabolic risk factors, supporting routine liver function assessment in diabetic care. **Keywords:** Diabetes mellitus; liver enzymes; alanine aminotransferase; aspartate aminotransferase; gamma-glutamyl transferase; glycemic control; dyslipidemia.

INTRODUCTION

Diabetes mellitus, particularly type 2 diabetes mellitus, is a major global metabolic disorder associated with progressive systemic complications and substantial clinical burden. Although diabetic complications are traditionally recognized in relation to cardiovascular, renal, neurological, and ocular systems, hepatic involvement is increasingly being acknowledged as an important but often underdiagnosed component of the disease spectrum. The liver has a central role in glucose and lipid metabolism, including glycogen storage, gluconeogenesis, insulin clearance, fatty acid oxidation, and lipoprotein regulation. In individuals with diabetes, chronic hyperglycemia and insulin resistance disrupt these metabolic pathways, creating a hepatic environment characterized by lipid accumulation, oxidative stress, low-grade inflammation, and hepatocellular injury (1).

Elevated serum liver enzymes are among the earliest and most accessible biochemical indicators of hepatic dysfunction in diabetic individuals. Alanine aminotransferase, aspartate aminotransferase,

gamma-glutamyl transferase, and alkaline phosphatase are routinely measured laboratory markers that may reflect hepatocellular injury, cholestatic stress, steatosis, or subclinical inflammatory liver disease. These abnormalities may remain clinically silent and are often detected incidentally during routine biochemical evaluation, making liver enzyme testing a practical tool for identifying early hepatic involvement in patients at metabolic risk (2). In the context of type 2 diabetes mellitus, such abnormalities are clinically meaningful because they may indicate underlying non-alcoholic fatty liver disease, metabolic dysfunction-associated steatotic liver disease, or early hepatic injury before the development of overt liver-related symptoms.

Non-alcoholic fatty liver disease is widely considered the hepatic manifestation of metabolic syndrome and is strongly associated with insulin resistance, obesity, dyslipidemia, and impaired glycemic control. Its prevalence is substantially higher among individuals with type 2 diabetes than in the general population, and its progression to non-alcoholic steatohepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma has major implications for long-term morbidity. The biological association between diabetes and hepatic dysfunction is primarily mediated through insulin resistance, increased adipose tissue lipolysis, excessive free fatty acid delivery to the liver, enhanced hepatic de novo lipogenesis, and impaired lipid export. These mechanisms promote triglyceride accumulation within hepatocytes, mitochondrial dysfunction, oxidative injury, and inflammatory activation, which may be reflected biochemically through elevated liver enzyme levels (3,4).

The relationship between diabetes and liver dysfunction is bidirectional. Diabetes accelerates hepatic steatosis and fibrosis progression, while fatty liver disease further worsens insulin resistance and increases the risk of incident diabetes. This reciprocal relationship suggests that liver enzyme abnormalities in diabetic patients should not be interpreted as isolated laboratory findings but as part of a broader metabolic and hepatic disease process (5). Longitudinal evidence supports this concept. Fraser et al. reported that alanine aminotransferase and gamma-glutamyl transferase were associated with incident diabetes in the British Women's Heart and Health Study, while findings from the Atherosclerosis Risk in Communities study showed that higher liver enzyme levels, particularly gamma-glutamyl transferase, were associated with increased future diabetes risk, even within conventional reference ranges (6,7). Similarly, systematic and longitudinal data have shown that elevated alanine aminotransferase is associated with progression to impaired glucose tolerance and future type 2 diabetes, although the strength of association varies by enzyme type and population characteristics (8,9).

Cross-sectional studies in patients with established type 2 diabetes have consistently demonstrated a higher prevalence of liver enzyme abnormalities compared with non-diabetic individuals. Forlani et al. reported that elevated liver enzymes were common among patients with type 2 diabetes and were associated with features of metabolic syndrome, including central obesity, hypertension, dyslipidemia, and insulin resistance (10). Chen et al. observed a high prevalence of abnormal serum liver enzymes among Chinese patients with type 2 diabetes, with significant associations involving obesity, male sex, hypertension, and alcohol exposure (11). Similar findings were reported by Teshome et al., who found that abnormal liver function tests were frequent among diabetic patients, with alanine aminotransferase being one of the most commonly affected markers (12). These studies indicate that hepatic biochemical abnormalities are not confined to one region or population but occur across diverse ethnic and clinical settings.

Evidence from South Asian populations is particularly relevant because diabetes occurs at younger ages and often at lower body mass index thresholds compared with Western populations. Islam et al. reported that elevated liver enzymes were significantly more common among Bangladeshi adults with type 2 diabetes than among non-diabetic controls, and gamma-glutamyl transferase was identified as an independent predictor of diabetes (13). Alam et al. similarly demonstrated a significant relationship between elevated liver enzymes and type 2 diabetes among North Indian adults, supporting the clinical importance of liver function testing in regional populations with high metabolic risk (14). More recent

meta-analytic evidence has further shown that non-alcoholic fatty liver disease is highly prevalent in individuals with type 2 diabetes, with a considerable proportion having clinically significant fibrosis, indicating that abnormal liver enzymes may represent early biochemical signals of more advanced underlying liver disease in selected patients (15).

Despite growing international evidence, local data regarding the prevalence and pattern of elevated liver enzymes among diabetic individuals remain limited, particularly in relation to glycemic control, obesity, and dyslipidemia. In resource-limited settings, where ultrasound, elastography, and advanced fibrosis assessment may not be routinely available, liver function tests offer a low-cost and widely accessible method for early identification of possible hepatic involvement. The population of interest in the present study comprised diabetic and non-diabetic adults, the exposure was diabetes mellitus and related metabolic dysregulation, the comparison was between diabetic and non-diabetic participants, and the primary outcome was the presence of elevated liver enzymes, including alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, and gamma-glutamyl transferase. Therefore, this study aimed to determine the prevalence of elevated liver enzymes among diabetic individuals and to assess their association with diabetes mellitus, glycemic control, obesity, and dyslipidemia in a local clinical laboratory setting.

MATERIALS AND METHODS

This comparative cross-sectional observational study was conducted in the Biochemistry and Pathology Laboratory of Social Security Hospital, Lahore, over a four-month period to determine the prevalence of elevated liver enzymes and assess their association with diabetes mellitus and related metabolic factors. The cross-sectional design was selected because it allowed simultaneous assessment of glycemic status, liver enzyme abnormalities, obesity, and dyslipidemia in diabetic and non-diabetic participants within a defined clinical laboratory setting.

A total of 200 adult participants were enrolled, comprising 100 individuals with diabetes mellitus and 100 non-diabetic individuals. Participants were selected through non-probability convenience sampling from individuals presenting to the laboratory during the study period. Eligible participants included adults aged 18–65 years who provided informed consent and were willing to undergo fasting blood sampling and clinical data collection. The diabetic group included participants with established diabetes mellitus or biochemical evidence of diabetes based on fasting blood glucose and HbA1c assessment, while the non-diabetic group included individuals without known diabetes and with glycemic indices within the non-diabetic range. Participants with known chronic liver disease, viral hepatitis, alcohol-related liver disease, pregnancy, malignancy, acute systemic infection, recent major surgery, or current use of hepatotoxic drugs were excluded to reduce non-diabetic causes of liver enzyme elevation.

After obtaining informed consent, demographic and clinical information was collected using a structured data collection form. Recorded variables included age, sex, diabetic status, fasting blood glucose, HbA1c, liver enzyme profile, obesity status, and dyslipidemia status. Diabetes mellitus was treated as the primary exposure variable, while elevated liver enzymes were considered the principal outcome variable. Glycemic control was assessed using HbA1c and fasting blood glucose values. Poor glycemic control was defined on the basis of raised HbA1c, while high fasting blood glucose was defined according to fasting biochemical results. Obesity was assessed using body mass index classification, and dyslipidemia was defined according to abnormal lipid profile status or documented lipid abnormality.

All participants were instructed to observe overnight fasting before sample collection. Under aseptic conditions, approximately 3–5 mL of venous blood was collected from each participant by trained laboratory personnel. Blood samples were processed according to standard laboratory procedures. Serum was separated and analyzed for alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, gamma-glutamyl transferase, and fasting blood glucose using an automated biochemical analyzer. HbA1c was measured according to routine laboratory protocol. Liver enzyme values were

interpreted against laboratory reference ranges, and participants were categorized as having elevated liver enzymes if at least one of the assessed enzymes was above the reference limit. The individual prevalence of elevated ALT, AST, ALP, and GGT was also recorded separately.

To minimize measurement bias, blood collection, sample processing, and biochemical analysis were performed using standardized laboratory procedures. Samples were analyzed under routine internal quality-control conditions, and results were recorded directly from laboratory outputs to reduce transcription error. Selection bias was addressed by enrolling diabetic and non-diabetic participants from the same laboratory setting during the same study period. Potential confounding by obesity and dyslipidemia was assessed by comparing the frequency of elevated liver enzymes across these metabolic risk categories.

The sample size consisted of 200 participants, equally distributed between diabetic and non-diabetic groups, allowing balanced comparison of biochemical parameters and liver enzyme abnormalities between the two groups. Data were entered, cleaned, and analyzed using statistical software. Continuous variables, including fasting blood glucose, HbA1c, ALT, AST, ALP, and GGT, were summarized as mean and standard deviation. Categorical variables, including diabetic status, sex, age group, elevated liver enzyme status, glycemic-control category, obesity, and dyslipidemia, were summarized as frequency and percentage. Mean biochemical parameters were compared between diabetic and non-diabetic groups using an independent-samples t-test. Associations between elevated liver enzymes and categorical variables, including diabetic status, glycemic control, fasting blood glucose category, obesity, and dyslipidemia, were assessed using the chi-square test. A p-value of less than 0.05 was considered statistically significant.

Data integrity was maintained through structured data collection, consistent coding of categorical variables, and cross-checking of entered values before analysis. Participant confidentiality was maintained by anonymizing data and using study codes instead of personal identifiers. The study was conducted after ethical approval, and informed consent was obtained from all participants before enrollment.

RESULTS

A total of 200 participants were included, with equal representation of diabetic and non-diabetic individuals. The diabetic group comprised 100 participants, and the non-diabetic group also comprised 100 participants, allowing balanced comparison between both groups. The overall study population was predominantly middle-aged to older, with 94 participants aged 46–65 years and 88 aged 31–45 years. Males represented 55.0% of the sample, while females represented 45.0%.

Table 1. Demographic Characteristics of Study Participants

Variable	Category	Frequency, n	Percentage, %
Study group	Diabetic	100	50.0
Study group	Non-diabetic	100	50.0
Age group	18–30 years	18	9.0
Age group	31–45 years	88	44.0
Age group	46–65 years	94	47.0
Gender	Male	110	55.0
Gender	Female	90	45.0
Total	—	200	100.0

Diabetic participants had markedly higher fasting blood glucose, HbA1c, and liver enzyme levels than non-diabetic participants. Mean fasting blood glucose was 171.3 ± 32.7 mg/dL in the diabetic group compared with 95.1 ± 9.2 mg/dL in the non-diabetic group, giving a mean difference of 76.2 mg/dL. Mean HbA1c was also higher among diabetic participants, with a difference of 2.9 percentage points. Among liver enzymes, GGT showed a mean difference of 20.0 U/L, ALT showed a mean difference of 15.9 U/L, ALP showed a mean difference of 18.7 U/L, and AST showed a mean difference of 8.7 U/L. All between-group differences were statistically significant at $p < 0.001$.

Table 2. Comparison of Biochemical Parameters Between Diabetic and Non-Diabetic Participants

Parameter	Diabetic Group Mean ± SD	Non-Diabetic Group Mean ± SD	Mean Difference	95% CI for Mean Difference	Effect Size, Cohen's d	p-value
FBG, mg/dL	171.3 ± 32.7	95.1 ± 9.2	76.2	69.5 to 82.9	3.17	<0.001
HbA1c, %	8.2 ± 1.4	5.3 ± 0.3	2.9	2.6 to 3.2	2.86	<0.001
ALT, U/L	44.4 ± 10.4	28.5 ± 7.2	15.9	13.4 to 18.4	1.78	<0.001
AST, U/L	36.7 ± 8.8	28.0 ± 6.0	8.7	6.6 to 10.8	1.16	<0.001
ALP, U/L	126.9 ± 25.3	108.2 ± 17.8	18.7	12.6 to 24.8	0.85	<0.001
GGT, U/L	51.1 ± 14.2	31.1 ± 9.2	20.0	16.7 to 23.3	1.67	<0.001

The prevalence of elevated liver enzymes was substantially higher among diabetic participants. At least one elevated liver enzyme was present in 84 diabetic participants compared with 11 non-diabetic participants, corresponding to 84.0% versus 11.0%. The odds of having any elevated liver enzyme were approximately 42 times higher in diabetic participants than in non-diabetic participants. ALT was the most frequent abnormal enzyme among diabetic participants, affecting 51.0%, followed by AST in 39.0%, ALP in 36.0%, and GGT in 36.0%.

Table 3. Prevalence of Elevated Liver Enzymes by Diabetic Status

Liver Enzyme Status	Diabetic, n/N (%)	Non-Diabetic, n/N (%)	Odds Ratio	95% CI	p-value
Elevated ALT	51/100 (51.0)	2/100 (2.0)	51.00	11.92 to 218.26	<0.001
Elevated AST	39/100 (39.0)	2/100 (2.0)	31.33	7.30 to 134.43	<0.001
Elevated ALP	36/100 (36.0)	7/100 (7.0)	7.47	3.13 to 17.84	<0.001
Elevated GGT	36/100 (36.0)	0/100 (0.0)	113.74*	6.86 to 1885.97	<0.001
Any elevated liver enzyme	84/100 (84.0)	11/100 (11.0)	42.48	18.64 to 96.79	<0.001

Elevated liver enzymes were strongly associated with poor glycemic control. Among participants with poor glycemic control, 76 of 85 had elevated liver enzymes, representing 89.4%, whereas only 19 of 115 participants with good glycemic control had elevated liver enzymes, representing 16.5%. The odds of elevated liver enzymes were approximately 43 times higher among participants with poor glycemic control. A similar pattern was observed for fasting blood glucose status: elevated liver enzymes were present in 76 of 90 participants with high fasting blood glucose compared with 19 of 110 participants with normal fasting blood glucose.

Table 4. Association of Elevated Liver Enzymes With Glycemic Status

Glycemic Variable	Elevated Liver Enzymes Present, n/N (%)	Elevated Liver Enzymes Absent, n/N (%)	Odds Ratio	95% CI	p-value
Poor glycemic control	76/85 (89.4)	9/85 (10.6)	42.67	18.27 to 99.65	<0.001
Good glycemic control	19/115 (16.5)	96/115 (83.5)	Reference	—	—
High FBG	76/90 (84.4)	14/90 (15.6)	26.00	12.23 to 55.29	<0.001
Normal FBG	19/110 (17.3)	91/110 (82.7)	Reference	—	—

Elevated liver enzymes were also more common among participants with obesity and dyslipidemia. Among obese participants, 48 of 64 had elevated liver enzymes, compared with 47 of 136 non-obese participants. This corresponded to 75.0% versus 34.6%, with an odds ratio of 5.68. Dyslipidemia showed a similar association: 57 of 84 participants with dyslipidemia had elevated liver enzymes compared with 38 of 116 participants without dyslipidemia, corresponding to 67.9% versus 32.8%.

Table 5. Association of Elevated Liver Enzymes With Obesity and Dyslipidemia

Metabolic Factor	Elevated Liver Enzymes Present, n/N (%)	Elevated Liver Enzymes Absent, n/N (%)	Odds Ratio	95% CI	p-value
Obese	48/64 (75.0)	16/64 (25.0)	5.68	2.92 to 11.07	<0.001
Non-obese	47/136 (34.6)	89/136 (65.4)	Reference	—	—
Dyslipidemia present	57/84 (67.9)	27/84 (32.1)	4.33	2.38 to 7.90	<0.001
Dyslipidemia absent	38/116 (32.8)	78/116 (67.2)	Reference	—	—

Overall, the results demonstrate that diabetic participants had significantly higher glycemic indices, higher mean liver enzyme levels, and a substantially greater prevalence of liver enzyme abnormalities than non-diabetic participants. The strongest associations with elevated liver enzymes were observed for

poor glycemic control, diabetic status, high fasting blood glucose, obesity, and dyslipidemia, indicating that hepatic biochemical abnormalities clustered with broader metabolic dysregulation.

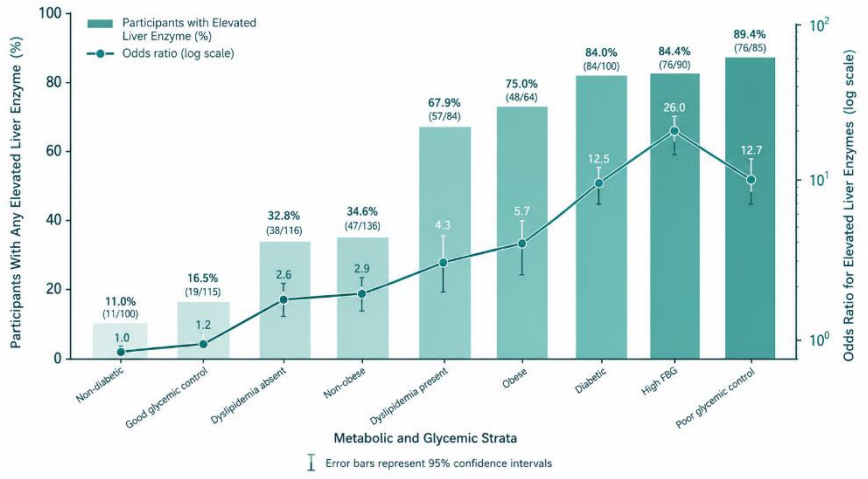

Figure 1. Metabolic Risk Gradient for Elevated Liver Enzyme Abnormalities

The prevalence of any elevated liver enzyme increased progressively across worsening metabolic and glycemic strata, rising from 11.0% among non-diabetic participants and 16.5% among participants with good glycemic control to 67.9% in those with dyslipidemia, 75.0% in obese participants, 84.0% in diabetic participants, 84.4% in those with high fasting blood glucose, and 89.4% among participants with poor glycemic control. The odds-ratio layer demonstrated a parallel escalation in risk, with dyslipidemia showing a 4.3-fold higher odds of elevated liver enzymes, obesity a 5.7-fold higher odds, high fasting blood glucose a 26.0-fold higher odds, diabetes a 42.5-fold higher odds, and poor glycemic control a 42.7-fold higher odds, indicating that hepatic enzyme abnormalities clustered most strongly with hyperglycemia and poor glycemic regulation while remaining clinically elevated across obesity and lipid abnormality strata.

DISCUSSION

The present study demonstrated a substantially higher burden of liver enzyme abnormalities among diabetic participants compared with non-diabetic individuals, indicating a clear biochemical association between diabetes mellitus and hepatic dysfunction. Diabetic participants showed significantly higher mean values of ALT, AST, ALP, and GGT, and the prevalence of at least one elevated liver enzyme was markedly greater in the diabetic group. This pattern is consistent with previous evidence from South Asian populations, where liver enzyme elevation has been reported more frequently among adults with type 2 diabetes than among non-diabetic controls (6,7). The findings support the concept that diabetes is not limited to impaired glycemic regulation but represents a multisystem metabolic disorder in which the liver is an important target organ.

The higher mean liver enzyme levels observed among diabetic participants are biologically plausible because the liver has a central role in maintaining glucose and lipid homeostasis. In diabetes, insulin resistance increases adipose tissue lipolysis and free fatty acid flux to the liver, while hyperinsulinemia and hyperglycemia stimulate hepatic de novo lipogenesis. These mechanisms promote intrahepatic triglyceride accumulation, oxidative stress, mitochondrial dysfunction, inflammatory activation, and hepatocellular injury, which may be reflected by elevated circulating aminotransferases and other liver enzymes (8,9). Therefore, the observed increases in ALT, AST, ALP, and GGT are likely to represent metabolic hepatic stress rather than isolated biochemical variation.

ALT was the most frequently elevated enzyme among diabetic participants, suggesting that the dominant pattern of injury was hepatocellular rather than cholestatic. ALT is more liver-specific than AST and is commonly associated with metabolic fatty liver disease and early hepatocellular injury. The

predominance of ALT elevation in the diabetic group aligns with previous regional and international studies that identified ALT as one of the most commonly abnormal liver function parameters in patients with type 2 diabetes (10–13). This finding is clinically important because ALT elevation, even when mild, may reflect underlying hepatic steatosis or early inflammatory liver injury in metabolically vulnerable individuals.

A major finding of the study was the strong association between poor glycemic control and elevated liver enzymes. Participants with poor glycemic control had a much higher frequency of enzyme abnormalities than those with good glycemic control, suggesting that worsening chronic hyperglycemia may intensify hepatic injury. Persistent hyperglycemia contributes to oxidative stress, advanced glycation end-product formation, lipid peroxidation, and inflammatory signaling, all of which may worsen hepatocyte damage and enzyme leakage into the circulation (11). This relationship supports the interpretation that liver enzyme elevation in diabetes is closely linked with the severity of metabolic dysregulation rather than diabetic status alone. Similar real-world evidence has shown associations between liver enzyme abnormalities and type 2 diabetes, further emphasizing the metabolic connection between glycemic disturbance and hepatic biochemical injury (12).

Obesity was also significantly associated with elevated liver enzymes, reinforcing its role as a major driver of hepatic dysfunction in diabetic and metabolically at-risk populations. Obesity, particularly central adiposity, increases insulin resistance and promotes excessive free fatty acid delivery to the liver. This creates a metabolic environment favorable to hepatic steatosis, inflammation, and progressive liver injury. The higher prevalence of elevated liver enzymes among obese participants in this study is consistent with previous observations that obesity increases the risk of NAFLD and advanced fibrosis among people with type 2 diabetes (13,14). These findings suggest that obesity may amplify the hepatic effects of diabetes and should be considered an important coexisting risk factor when interpreting abnormal liver function tests.

Dyslipidemia showed a similarly important association with elevated liver enzymes. Participants with dyslipidemia had a higher prevalence of hepatic enzyme abnormalities than those without lipid disturbances. This association is expected because dyslipidemia reflects impaired lipid handling, increased triglyceride availability, altered lipoprotein metabolism, and hepatic lipid accumulation. These abnormalities may worsen hepatic steatosis and contribute to hepatocellular stress. Previous studies have also reported relationships between abnormal lipid profiles and elevated liver enzymes among patients with type 2 diabetes, supporting the view that liver enzyme elevation is part of a broader metabolic syndrome phenotype rather than an isolated hepatic event (15,16).

The high prevalence of elevated liver enzymes among diabetic participants may also indicate a substantial underlying burden of metabolic fatty liver disease, although structural liver assessment was not performed. Previous systematic reviews and meta-analyses have shown that NAFLD is highly prevalent among individuals with type 2 diabetes, and a meaningful proportion of these patients may have non-alcoholic steatohepatitis, advanced fibrosis, or other clinically significant liver outcomes (17,18). In this context, liver enzyme elevation should be interpreted as a useful biochemical signal of possible hepatic involvement, although normal liver enzymes do not exclude fatty liver disease or fibrosis. The findings therefore highlight the need to consider hepatic health as part of routine metabolic assessment in patients with diabetes.

From a clinical perspective, the findings are particularly relevant for settings where advanced liver imaging, elastography, and fibrosis assessment tools are not routinely accessible. Liver function tests are inexpensive, widely available, and commonly performed in clinical laboratories, making them practical for identifying diabetic patients who may require closer hepatic evaluation or intensified metabolic management (19). Current evidence also supports structured risk stratification pathways for fatty liver disease in patients with type 2 diabetes, especially when biochemical abnormalities coexist with obesity, poor glycemic control, or dyslipidemia (20). Therefore, liver enzyme abnormalities in diabetic patients

should prompt careful clinical interpretation in conjunction with glycemic indices, anthropometric measures, and lipid profile.

The magnitude of association observed in this study may be influenced by several population and methodological factors. Hospital-based recruitment may include participants with greater metabolic burden than community-based samples, and prevalence estimates may vary depending on enzyme reference ranges, age distribution, obesity frequency, diabetes duration, medication exposure, alcohol intake, and exclusion of pre-existing liver disease. Similar variability has been reported across studies evaluating liver enzyme abnormalities in patients with type 2 diabetes (21). Nevertheless, the consistent direction of association across ALT, AST, ALP, and GGT strengthens the evidence that diabetes and metabolic dysregulation are closely linked with hepatic biochemical abnormalities.

The study has several strengths, including the inclusion of both diabetic and non-diabetic comparison groups, assessment of multiple liver enzymes, and evaluation of clinically relevant metabolic factors such as glycemic control, obesity, and dyslipidemia. At the same time, the cross-sectional design limits causal interpretation because exposure and outcome were assessed at the same time. The absence of ultrasound, transient elastography, fibrosis scoring, or liver biopsy prevents direct confirmation of NAFLD, fibrosis, or other structural liver disease. In addition, the single-center setting and convenience sampling approach may limit generalizability to broader populations. These limitations are important when interpreting the results, particularly because abnormal liver enzymes may arise from multiple hepatic and extrahepatic causes (22).

Overall, the study supports a strong association between diabetes mellitus and elevated liver enzymes, with the highest burden observed among participants with poor glycemic control, high fasting blood glucose, obesity, and dyslipidemia. These findings reinforce the concept that hepatic biochemical abnormalities are closely integrated with metabolic dysfunction and may serve as accessible indicators of possible early liver involvement in diabetic patients. Given the bidirectional relationship between metabolic fatty liver disease and diabetes, early recognition of enzyme abnormalities may help identify individuals who require closer monitoring and comprehensive metabolic risk reduction.

CONCLUSION

This study demonstrated a significantly higher prevalence of elevated liver enzymes among diabetic individuals compared with non-diabetic participants, with 84.0% of diabetic subjects showing at least one abnormal liver enzyme compared with 11.0% of non-diabetic subjects. Diabetic participants also had higher mean levels of ALT, AST, ALP, and GGT, indicating a clear pattern of hepatic biochemical disturbance associated with diabetes mellitus. The predominance of ALT elevation suggests a mainly hepatocellular pattern of injury, which may reflect underlying metabolic liver involvement. Elevated liver enzymes were strongly associated with poor glycemic control, high fasting blood glucose, obesity, and dyslipidemia, showing that hepatic dysfunction in diabetes is closely linked with broader metabolic derangement. These findings support the clinical value of routine liver function assessment in diabetic patients, particularly among those with uncontrolled glycemia or coexisting metabolic risk factors, as early identification of liver enzyme abnormalities may help guide timely monitoring, lifestyle modification, metabolic optimization, and further hepatic evaluation when clinically indicated.

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