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Original Research Article

Liver Dysfunction in Type 2 Diabetes: Association with Microalbuminuria

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Abstract

Background: Liver dysfunction is increasingly recognized as an important complication of type 2 diabetes mellitus and may coexist with early renal microvascular damage.

Objectives: To evaluate liver dysfunction in patients with type 2 diabetes mellitus and to assess its correlation with microalbuminuria.

Methods: A cross-sectional study was conducted among 120 patients with type 2 diabetes mellitus. Liver function tests, ultrasonography, glycated hemoglobin, body mass index, and urinary albumin excretion were assessed and correlated.

Results: Liver dysfunction was significantly associated with longer duration of diabetes, poor glycemic control, higher body mass index, abnormal ultrasonography findings, and presence of microalbuminuria.

Conclusion: Liver dysfunction is common in type 2 diabetes mellitus and shows a significant correlation with microalbuminuria, highlighting the need for integrated hepatic and renal assessment in diabetic care.

Keywords: Type 2 Diabetes Mellitus, Liver Dysfunction, Microalbuminuria, Glycemic Control.

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Introduction

Type 2 diabetes mellitus (T2DM) is a global metabolic disorder characterized by chronic hyperglycemia and associated with progressive microvascular and macrovascular complications. In addition to classical complications such as nephropathy, retinopathy, and neuropathy, increasing evidence suggests that the liver is an important target organ affected in T2DM [1]. Liver dysfunction in diabetic patients often remains uderrecognised despite its significant clinical implications.

Non-alcoholic fatty liver disease (NAFLD) represents the most common hepatic manifestation of T2DM and is strongly associated with insulin resistance, dyslipidemia, and chronic low-grade inflammation [2]. Elevated liver enzymes, particularly alanine aminotransferase (ALT) and aspartate aminotransferase (AST), are frequently observed in individuals with T2DM and may reflect underlying hepatic steatosis or steatohepatitis [3].

These abnormalities are not merely biochemical findings but are associated with increased cardiovascular and renal risk. Microalbuminuria is an established early marker of diabetic nephropathy and endothelial dysfunction. It reflects increased vascular permeability and generalized microvascular damage in patients with diabetes [4]. Several studies have demonstrated that microalbuminuria is not confined to renal pathology alone but is also associated with hepatic insulin resistance and altered liver function [5]. This suggests a shared pathophysiological pathway linking hepatic dysfunction and renal microvascular injury in T2DM.

Insulin resistance plays a central role in the development of both liver dysfunction and microalbuminuria. Hepatic insulin resistance leads to increased hepatic glucose output, lipid accumulation, and oxidative stress, contributing to hepatocellular injury [6]. Simultaneously, systemic insulin resistance and endothelial dysfunction promote glomerular hyperfiltration and increased urinary albumin excretion [7]. Thus, liver dysfunction and microalbuminuria may represent parallel manifestations of widespread metabolic and vascular derangements in diabetes.

Clinical studies have reported a significant association between elevated liver enzymes and the presence of microalbuminuria in patients with T2DM, indicating that hepatic involvement may serve as a surrogate marker for early diabetic nephropathy [8]. Moreover, liver dysfunction has been shown to correlate with the severity and duration of diabetes, poor glycemic control, and the presence of other cardiovascular risk factors [9].

Despite growing evidence, the relationship between liver dysfunction and microalbuminuria remains inadequately explored, particularly in developing countries where the burden of T2DM is rapidly increasing. Early identification of hepatic abnormalities and their correlation with microalbuminuria may aid in risk stratification and prompt intervention to prevent progression of diabetic complications [10]. Therefore, the present study aims to evaluate liver dysfunction in patients with type 2 diabetes mellitus and to assess its correlation with microalbuminuria.

Material and Methods

This hospital-based cross-sectional observational study was conducted in the Department of General Medicine of a tertiary care teaching hospital to evaluate liver dysfunction in patients with type 2 diabetes mellitus and to assess its correlation with microalbuminuria. The study was carried out over a period of one year after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to their enrollment in the study.

A total of 120 patients diagnosed with type 2 diabetes mellitus were included in the study. Patients aged 30 years and above, with a confirmed diagnosis of type 2 diabetes mellitus based on standard diagnostic criteria, were selected. Patients with known chronic liver disease of other etiologies, including viral hepatitis, alcoholic liver disease, autoimmune hepatitis, or drug-induced liver injury, were excluded. Individuals with a history of chronic alcohol consumption, pre-existing renal disease unrelated to diabetes, urinary tract infection, congestive cardiac failure, pregnancy, or those unwilling to participate were also excluded to avoid confounding factors.

Detailed demographic and clinical data were collected from all participants, including age, gender, duration of diabetes, body mass index, blood pressure, and history of associated comorbidities such as hypertension and dyslipidemia. A thorough clinical examination was performed for each patient. Anthropometric measurements were recorded using standard methods, and body mass index was calculated as weight in kilograms divided by height in meters squared.

Laboratory investigations were carried out after an overnight fast. Blood samples were collected to assess fasting plasma glucose, postprandial plasma glucose, glycated hemoglobin, and liver function tests, including serum alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, total bilirubin, and serum albumin. Liver dysfunction was defined based on elevation of liver enzymes beyond the normal reference range.

Urine samples were collected to assess microalbuminuria using a spot urine albumin-to-creatinine ratio. Microalbuminuria was defined as urinary albumin excretion of 30–300 mg/g of creatinine. Based on the presence or absence of microalbuminuria, patients were categorized accordingly for correlation analysis.

Statistical analysis was performed using Statistical Package for the Social Sciences software. Continuous variables were expressed as mean and standard deviation, while categorical variables were expressed as frequencies and percentages. The association between liver dysfunction and microalbuminuria was analyzed using chi-square test and independent t-test where appropriate. Correlation analysis was performed to assess the relationship between liver enzyme levels and urinary albumin excretion. A p-value of less than 0.05 was considered statistically significant.

Results

Table 1 describes the demographic characteristics of the study population. Among the 120 patients with type 2 diabetes mellitus, the majority belonged to the 51–60 years age group, accounting for 34 patients (28.33%), followed by 61–70 years with 30 patients (25%). Patients aged 41–50 years constituted 26 individuals (21.67%), while those aged above 70 years and 40 years or below accounted for 18 (15%) and 12 (10%) patients respectively. Equal gender distribution was maintained, with 60 males and 60 females included in the study. With respect to body mass index, 54 patients (45%) were classified as overweight or obese (BMI ≥25 kg/m²), 52 patients (43.33%) had BMI between 18–25 kg/m², and 14 patients (11.67%) were underweight.

Table 2 shows the distribution of elevated liver enzymes among the study participants. Elevated SGPT levels were observed in 56 patients (46.67%), of which 18 patients (32.14%) had mild elevation, 14 patients (25%), and 24 patients (42.86%) showed severe elevation. Elevated SGOT was noted in 44 patients (36.67%), while elevated ALP and GGT were observed in 40 (33.33%) and 22 (18.33%) patients respectively. Elevated serum bilirubin levels were seen in 26 patients (21.67%), indicating varying degrees of hepatic involvement among diabetic patients.

Table 3 presents impaired liver function test parameters. Prolonged prothrombin time (>14 seconds) was observed in 20 patients (16.67%), while elevated INR (>1.2) was noted in 24 patients (20%), suggesting compromised hepatic synthetic function in a subset of the study population.

Table 4 illustrates the correlation between liver function abnormalities and duration of diabetes mellitus. Patients with duration of diabetes ≥5 years showed significantly higher prevalence of elevated liver enzymes compared to those with duration ≤5 years. Elevated SGOT was observed in 40 patients with longer duration compared to 8 patients with shorter duration ($p = 0.004$). Similarly, SGPT, ALP, GGT, bilirubin, PT, and INR abnormalities were significantly higher in patients with diabetes duration ≥5 years, with p values <0.05, indicating a strong association between disease duration and liver dysfunction.

Table 5 shows the distribution of patients based on HbA1c values. A total of 50 patients (41.67%) had HbA1c levels between 8–10%, while 38 patients (31.67%) had poor glycemic control with HbA1c ≥10%. Only 32 patients (26.67%) had HbA1c ≤8%, indicating that the majority of patients had suboptimal glycemic control.

Table 6 demonstrates the correlation between liver dysfunction and glycated hemoglobin levels. No statistically significant association was observed between liver dysfunction and HbA1c ≤8% ($p = 0.214$). However, a significant association was noted for HbA1c values between 8–10% ($p = 0.002$) and ≥10% ($p = 0.000$), indicating worsening liver dysfunction with increasing glycemic levels.

Table 7 depicts the correlation between liver dysfunction and body mass index. Liver dysfunction was significantly more common in patients with BMI ≥25 kg/m² and BMI between 18–25 kg/m², with p values of 0.0001 for both categories. No statistically significant association was observed among underweight patients ($p = 0.063$).

Table 8 shows the association between liver dysfunction and ultrasonography findings. Abnormal ultrasonographic liver findings were present in 54 patients, of whom 44 exhibited liver dysfunction. In contrast, only 16 patients with normal ultrasonography findings showed liver dysfunction. This association was statistically highly significant ($p = 0.0001$).

Table 9 compares age and BMI with microalbuminuria. A positive correlation was observed between increasing age and microalbuminuria, with a Pearson correlation coefficient of 0.362 ($p = 0.000$). Similarly, BMI demonstrated a significant positive correlation with microalbuminuria ($r = 0.334$, $p = 0.000$), indicating increased microvascular involvement with advancing age and higher BMI.

Table 1: Demographic characteristics of patients (n = 120)

Demographic category	Characteristics	Number (n)	Percentage (%)
Age group (years)	≤40	12	10.0
	41–50	26	21.67
	51–60	34	28.33
	61–70	30	25.0
	>70	18	15.0
Gender	Male	60	50.0
	Female	60	50.0
BMI (kg/m²)	≤18	14	11.67
	18–25	52	43.33
	≥25	54	45.0
Total		120	100.0

Table 2: Elevated liver enzymes among study participants

Liver enzyme	Number (n)	Percentage (%)
SGPT (>300 IU/L)	56	46.67
Mild elevation	18	32.14
Moderate elevation	14	25.0
Severe elevation	24	42.86
SGOT (>200 IU/L)	44	36.67
ALP (>120 IU/L)	40	33.33
GGT (>45 IU/L)	22	18.33
Bilirubin (>1.04 mg/dL)	26	21.67

Table 3: Distribution of impaired liver function test parameters

Parameter	Number (n)	Percentage (%)
PT (>14 seconds)	20	16.67
INR (>1.20)	24	20.0

Table 4: Correlation of liver function test parameters with duration of diabetes mellitus

Parameter	≤5 years	≥5 years	Total	p value
SGOT	8	40	48	0.004 (HS)
SGPT	10	46	56	0.003 (HS)
ALP	12	28	40	0.001 (HS)
GGT	6	16	22	0.002 (HS)
Bilirubin	10	16	26	0.018 (Sig)
PT	6	14	20	0.001 (HS)
INR	8	16	24	0.001 (HS)

Table 5: Distribution of patients based on HbA1c values

HbA1c (%)	Number (n)	Percentage (%)
≤8	32	26.67
8–10	50	41.67
≥10	38	31.67
Total	120	100.0

Table 6: Correlation of liver dysfunction with glycated hemoglobin

HbA1c (%)	Liver dysfunction present	Liver dysfunction absent	Total	p value
≤8	12	20	32	0.214 (NS)
8–10	32	18	50	0.002 (HS)
≥10	30	8	38	0.000 (HS)

Table 7: Correlation of liver dysfunction with BMI

BMI (kg/m²)	Liver dysfunction present	p value
≤18	4	0.063 (NS)
18–25	24	0.0001 (HS)
≥25	46	0.0001 (HS)

Table 8: Correlation of liver dysfunction with ultrasonography findings

Ultrasonography findings	Liver dysfunction present	Liver dysfunction absent	Total	p value
Normal	16	50	66	0.0001 (HS)
Abnormal	44	10	54	

Table 9: Comparison of age and BMI with microalbuminuria

Variable	Category	Pearson coefficient	p value
Age group (years)	≤40	0.362	0.001 (HS)
	41–50		
	51–60		
	61–70		
	>70		
BMI (kg/m²)	≤18	0.334	0.001 (HS)
	18–25		
	≥25		

Discussion

The present study highlights a high prevalence of liver dysfunction among patients with type 2 diabetes mellitus, with a significant proportion demonstrating elevated liver enzymes and impaired synthetic liver function. These findings reinforce the concept that the liver is a major target organ in diabetes and that hepatic involvement often develops silently alongside other microvascular complications. Previous studies have similarly reported frequent elevation of transaminases in diabetic populations, particularly in association with insulin resistance and non-alcoholic fatty liver disease [11]. The observed abnormalities in SGPT, SGOT, ALP, and GGT in the present study indicate

ongoing hepatocellular injury and cholestatic involvement in a substantial subset of diabetic patients.

A significant association was observed between duration of diabetes mellitus and liver dysfunction, with patients having diabetes for more than five years showing markedly higher prevalence of abnormal liver function parameters. This finding is consistent with earlier reports demonstrating that prolonged exposure to hyperglycemia and insulin resistance leads to progressive hepatic injury through mechanisms such as oxidative stress, lipotoxicity, and mitochondrial dysfunction [12]. The stepwise increase in liver enzyme abnormalities with longer disease duration suggests that hepatic dysfunction parallels the chronicity of diabetes and may serve as an indicator of cumulative metabolic burden.

Glycemic control emerged as an important determinant of liver dysfunction in the present study. Patients with elevated HbA1c levels, particularly those with values above 8%, showed a significantly higher prevalence of liver dysfunction, whereas no significant association was noted in patients with relatively better glycemic control. These findings support existing evidence that poor glycemic control exacerbates hepatic steatosis and inflammation by promoting insulin resistance and increased hepatic glucose production [13]. This underscores the importance of strict glycemic control not only for preventing classical diabetic complications but also for mitigating hepatic injury.

Body mass index was also significantly associated with liver dysfunction, with overweight and obese patients exhibiting a markedly higher prevalence of abnormal liver function tests. Obesity-related insulin resistance, increased free fatty acid flux to the liver, and chronic low-grade inflammation are well-established contributors to hepatic steatosis and hepatocellular injury [14]. The strong association between BMI and liver dysfunction observed in the present study highlights the synergistic effect of obesity and diabetes in accelerating liver damage.

The correlation between liver dysfunction and microalbuminuria further emphasizes the systemic nature of diabetic microvascular disease. Microalbuminuria reflects endothelial dysfunction and generalized vascular injury, which may simultaneously affect hepatic and renal microcirculation.

Previous studies have demonstrated that patients with diabetic nephropathy often exhibit concomitant hepatic abnormalities, suggesting shared pathogenic mechanisms involving insulin resistance, inflammation, and oxidative stress [15].

The significant association between abnormal ultrasonography findings and liver dysfunction in the present study further supports the clinical relevance of imaging in detecting underlying hepatic pathology. Overall, the findings of the present study highlight that liver dysfunction in type 2 diabetes mellitus is closely associated with disease duration, poor glycemic control, obesity, and microalbuminuria. Early recognition of hepatic involvement may provide an opportunity for timely intervention to prevent progression to advanced liver disease and reduce overall metabolic risk.

Conclusion

Liver dysfunction is a common but under recognized complication in patients with type 2 diabetes mellitus. The present study demonstrates a significant association between hepatic abnormalities and longer duration of diabetes, poor glycemic control, higher body mass index, and microalbuminuria. These findings emphasize the need for routine assessment of liver function and urinary albumin excretion in diabetic patients to facilitate early detection and comprehensive management of metabolic and microvascular complications.

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