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Original Article

Prevalence and Associated Factors of Hepatic Steatosis in Patients with Type-2 Diabetes Mellitus: A Real-World Experience From a Private Healthcare Facility of South Punjab, Pakistan

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Abstract

Objective: To determine the prevalence and associated demographic, clinical, and biochemical factors of hepatic steatosis (HS) in patients of type-2 diabetes mellitus (T2DM).

Methods: This cross-sectional study was conducted at the Outpatient Department of Medicine, Aleena Hospital, Bahawalpur, Pakistan from 1st January 2024 to 30th September 2024. Patients of either gender, aged 18 to 85 years, having T2DM, and willing to participate, were included. All patients underwent liver ultrasound, conducted by an experienced radiologist with experience of more than 20 years in hepatic imaging. HS grades were defined as S0 (normal liver), S1 (slight increase in echogenicity), S2 (clear increase with mild steatosis), and S3 (marked steatosis with poor visualization).

Results: In a total of 303 patients, 184 (60.7%) were female. The mean age was 49.7±10.9 years, ranging between 18-85 years. The mean BMI was 27.9±5.6 kg/m2, while obesity was identified in 88 (29.0%) patients. The mean duration of T2DM was 7.20±5.44 years. Hypertension was present in 123 (40.6%) patients. Family history of DM was reported in 139 (45.9%) patients. HS staging S0, S1, S2, and S3 were diagnosed in 140 (46.2%), 114 (37.6%), 42 (13.9%), and 7 (2.3%) patients, respectively. The severity of HS was significantly associated with relatively shorter duration of T2DM (p=0.045).

Conclusion: The prevalence of HS is very high in patients with T2DM. All T2DM patients should be assessed for hepatic complications for the timely identification and management.

Keywords: Body mass index, hepatic steatosis, hypertension, obesity, type-2 diabetes mellitus.

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Introduction

Hepatic steatosis (HS), commonly known as fatty liver disease, is a significant global health concern, particularly among patients with "type 2 diabetes mellitus (T2DM)". It is estimated to affect approximately 25% of the global population, with prevalence rates reaching as high as 70% in individuals with T2DM.[1,2] HS forms part of the broader spectrum of "non-alcoholic fatty liver diseases (NAFLD)", which can progress to more severe conditions such as non-alcoholic steatohepatitis (NASH), cirrhosis, and even hepatocellular carcinoma.[3]

The link between T2DM and HS is underpinned by shared metabolic dysfunctions, including insulin resistance, dyslipidemia, and obesity.[4] In T2DM patients, chronic hyperglycemia and impaired insulin signaling lead to increased free fatty acid uptake and lipid accumulation in hepatocytes.[5] Data indicate that T2DM nearly doubles the risk of developing NAFLD, underscoring the importance of hepatic health in diabetic management.[6] With rising T2DM

prevalence in Pakistan, understanding HS in this group is critical for public health.

South Punjab region of Pakistan faces unique healthcare challenges, with limited access to specialized facilities and high rates of lifestyle-related diseases.[7] The local dietary patterns, often rich in refined carbohydrates and low in physical activity, contribute to obesity and metabolic syndrome, further elevating the risk for both T2DM and HS.[8,9] Data from this region are sparse, making it challenging to design targeted interventions. The high prevalence of HS in T2DM is concerning, as fatty liver disease contributes to worsening insulin resistance, creating a vicious cycle that can accelerate diabetic complications, including cardiovascular disease. This study aims to fill the gap in local data on HS among T2DM patients in South Punjab, providing insights into prevalence and associated factors within a real-world clinical setting. This study was aimed to determine the prevalence and associated demographic, clinical, and biochemical factors of HS in patients of T2DM attending a private healthcare facility in South Punjab, Pakistan. By exploring these associations, we hope to facilitate early identification of high-risk patients and inform preventive strategies tailored to this population.

Methods

This analytical, observational, cross-sectional study was conducted at the Outpatient Department of Medicine, Aleena Hospital/AVA Serene, Bahawalpur, Pakistan from 1st January 2024 to 30th September 2024. Using a prevalence estimate based on the findings of Gupta et al where they found Grade-3 HS in 85.2% T2DM patients,[10] with 95% confidence level, and 5% margin of error, the sample size was calculated to be 303. Approval from "Hospital Research Committee" was obtained prior to the commencement of this research (HRC/04/2023, dated: 16-11-2023). Inclusion criteria were patients of either gender, aged 18 to 85 years, having T2DM, and willing to participate. Exclusion criteria were patients with a history of alcohol consumption, chronic kidney disease, autoimmune disorders, sepsis, or congestive heart failure. Informed and written consents were sought from all patients. All patients were ensured about the secrecy of their data.

Upon enrollment, a trained clinician collected the demographic data, including age, gender, and BMI. Diabetes duration and HbA1c levels were documented. Lipid profiles, including triglycerides and HDL levels, were documented to assess dyslipidemia, while comorbidities such as hypertension was verified through medical records or physical examination. All participants underwent liver ultrasound, conducted by an experienced radiologist with experience of more than 20 years in hepatic imaging. All ultrasound exams were conducted by a single ultra sonographer, blinded to participants' clinical and lab results, using a "Sonoline Elegra Ultrasound Imaging System (Siemens), version 6", with a 3.5-MHz transducer. The liver's echogenicity was assessed using standard criteria, such as increased brightness compared to the right kidney, echo beam attenuation, and focal fatty sparing. Liver grades were defined as follows: grade 0 (normal liver), grade 1 (slight increase in echogenicity), grade 2 (clear increase with mild steatosis), and grade 3 (marked steatosis with poor visualization). Signs of hepatic cirrhosis were evaluated. Ultrasound findings were recorded at the time of the patient's initial assessment, providing standardized imaging data across the cohort. Hypertension was labeled when systolic blood pressure ≥130 mmHg and/or diastolic ≥80 mmHg, or a documented history of hypertension. Data were collected on a specially formatted proforma at the clinic during the initial visit, with all measurements and evaluations standardized to ensure reliable and comparable data across the study population.

Data analysis was performed employing IBM-SPSS Statistics, version 26.0. Qualitative variables were shown and frequency and percentages. Quantitative variables were represented as mean and standard deviation. The prevalence of HS was calculated, and associated factors were analyzed using chi-square or Fisher's exact test for categorical variables, while analysis of variance was applied for continuous variables. For all inferential statistics, $p<0.05$ was considered significant.

Results

In a total of 303 patients, 184 (60.7%) were female. The mean age was 49.7±10.9 years, ranging between 18-85 years. The mean BMI was 27.9±5.6 kg/m2, while obesity was identified in 88 (29.0%) patients. The mean duration of T2DM was 7.20±5.44 years. Hypertension was present in 123 (40.6%) patients. Family history of DM was reported in 139 (45.9%) patients. HS staging S0, S1, S2, and S3 were diagnosed in 140 (46.2%), 114 (37.6%), 42 (13.9%), and 7 (2.3%) patients, respectively. The severity of HS was significantly associated with relatively shorter duration of T2DM (p=0.045). Table-1 is showing association of HS severity with various demographic and clinical variables.

Table 1: *Association of hepatic steatosis with study variables (N=303)*

Study variables		Total (%)	Hepatic steatosis staging				P-value
			S0 (n=140)	S1 (n=114)	S2 (n=42)	S3 (n=7)	
Gender	Male	119 (39.3%)	56 (40.0%)	44 (38.6%)	18 (42.9%)	1 (14.3%)	0.55
	Female	184 (60.7%)	84 (60.0%)	70 (61.4%)	24 (57.1%)	6 (85.7%)	
Age (years)		49.7±10.9	51.3±11.1	48.9±10.4	47.1±11.8	48.3±11.2	0.113
Body mass index (kg/m^2)		27.9±5.6	27.9±5.8	28.1±5.7	27.7±4.9	27.2±5.2	0.965
Obesity		88 (29.0%)	40 (28.6%)	32 (28.1%)	13 (31.0%)	3 (42.9%)	0.852
Duration of diabetes (years)		7.20±5.44	8.38±5.85	6.38±5.22	5.82±3.81	4.50±3.79	0.045
Smoking		22 (7.3%)	9 (6.4%)	11 (9.6%)	1 (2.4%)	1 (14.3%)	0.375
Hypertension		123 (40.6%)	57 (40.7%)	48 (42.1%)	14 (33.3%)	4 (57.1%)	0.61
Family history of diabetes		139 (45.9%)	58 (41.4%)	62 (54.4%)	15 (35.7%)	4 (57.1%)	0.088
Impotence		9 (3.0%)	4 (2.9%)	4 (3.5%)	1 (2.4%)	-	0.943
Anti-HCV		30 (9.9%)	19 (13.6%)	7 (6.1%)	3 (7.1%)	1 (14.3%)	0.219
HBsAg		3 (1.0%)	2 (1.4%)	1 (0.0%)	-	-	0.854

Table 2: *Association of hepatic steatosis with laboratory parameters (N=303)*

Study variables	Total (%)	Hepatic steatosis staging				P-value
		S0 (n=140)	S1 (n=114)	S2 (n=42)	S3 (n=7)	
HbA1c (%)	9.17±2.11	9.26±2.29	9.33±2.05	8.68±1.34	7.74±1.51	0.307
Random blood sugar (mg/dl)	227.17±87.99	227.92±93.39	218.76±82.03	251.37±93.07	260.17±51.24	0.335
Serum albumin (mg/dl)	4.01±0.27	3.99±0.31	4.04±0.20	4.03±0.28	4.02±0.8	0.736
Low density lipoprotein (mg/dl)	121.84±46.75	108.95±52.26	131.63±45.72	126.80±15.22	156.00±8.82	0.424
High density lipoprotein (mg/dl)	46.14±19.96	47.21±15.55	48.44±26.00	36.60±5.37	32.00±12.82	0.601
Triglyceride (mg/dl)	228.71±233.74	183.89±89.80	206.53±95.24	464.60±658.75	275.50±171.82	0.106
Cholesterol (mg/dl)	194.3±54.9	188.0±62.5	191.5±47.7	209.8±55.5	245.0±25.5	0.503
Serum creatinine (mg/dl)	0.85±0.49	1.03±0.64	0.91±0.36	0.84±0.19	0.75±0.14	0.2
Hemoglobin	12.57±1.91	12.43±1.98	12.80±1.87	12.24±1.72	12.85±2.29	0.499
Platelet (10^9/L)	234.2±85.3	235.4±95.3	229.5±79.4	245.7±64.5	214.5±56.7	0.754
Total leukocyte count (ul)	9494±3533	10425±4494	8018±1972	10657±2006	10838±3048	0.16

HCV: Hepatitis C virus; HBsAg: Hepatitis B surface antigen

Severity of HS was not found to have any significant association with any of the biochemical parameters (table-2).

Discussion

The present study noted the prevalence of HS among T2DM as 53.8% which is lower than the 69.4% prevalence reported by Leite et al., in a Brazilian cohort of T2DM patients.[11] The difference could have stemmed from variations in study populations, diagnostic criteria, and risk factor profiles. Leite et al., reported higher levels of obesity and hypertriglyceridemia, which were strongly associated with steatosis in their cohort. Makker et al.,[6] from United States found 81% prevalence of HS diagnosed using elastography, which included patients with metabolic syndrome and advanced

T2DM. The higher prevalence in their study may reflect the inclusion of patients with more severe metabolic derangements. The present study population had lower obesity prevalence (29.0%) and a shorter T2DM duration, factors that likely contributed to the relatively lower prevalence of advanced steatosis. Sinha and Bankura from eastern India reported a prevalence of 57% for HS in a comparable population.[12] They also noted that approximately 26% of NAFLD cases progressed to NASH. While the present study did not assess the progression to NASH, the similarity in steatosis prevalence reinforces the importance of regional differences in metabolic risk profiles. The study by Poustchi et al.,[13] in Iran found similar associations between central obesity, female gender, and liver fibrosis in T2DM patients. The prevalence of HS as 53.8% in this study is very close to another study conducted by Salas-Flores et al from Mexico where they 57.1% healthcare workers with T2DM to have HS.[14]

Unlike the findings of Morieri et al.[15] who identified a significant association between dyslipidemia (elevated triglycerides and low HDL) and hepatic steatosis using the HS index (HSI), this study did not observe a similar relationship. This discrepancy could arise from methodological differences, including the use of ultrasonography in our study versus HSI and transient elastography in theirs, which may have greater sensitivity in detecting subtle changes in hepatic fat.

The prevalence of HS in over half of the T2DM population highlights the urgent need for routine screening in this high-risk group.[16] Early identification of HS could prompt lifestyle interventions, such as weight reduction and glycemic control, which have been shown to mitigate disease progression. The absence of significant associations between biochemical parameters and HS severity suggests that reliance on standard laboratory markers alone may not suffice for risk stratification.[17,18] Comprehensive evaluations, including imaging modalities like ultrasonography, should be integrated into routine care.[19] The significant association between shorter T2DM duration and severe HS ($p=0.045$) raises questions about the temporal dynamics of hepatic fat accumulation. These observations suggest that HS may develop early in the disease course, possibly driven by insulin resistance, even before significant metabolic derangements become apparent.[20,21] Early screening for HS in newly diagnosed T2DM patients may therefore be warranted.[16]

The observations found in this study underscore the importance of addressing obesity, particularly central obesity, as a modifiable risk factor. Although obesity was observed in 29% of our cohort, its role in driving HS and fibrosis is well-documented. Gupta et al.,[10] demonstrated that each unit increase in BMI above 23 kg/m² significantly increases the risk of HS in T2DM patients aged over 50 years. Lifestyle modifications targeting weight loss and improved metabolic control should remain central to managing T2DM-associated NAFLD.

Some limitations of this research must be acknowledged. The cross-sectional design precludes causal inferences about the relationships between T2DM and HS. Longitudinal studies are needed to clarify the temporal dynamics and causality of these associations. The use of ultrasonography, while practical and cost-effective, may underestimate the prevalence of mild HS compared to more advanced imaging modalities. Non-invasive fibrosis markers (e.g., FIB-4 index) were not studied.

Conclusion

The prevalence of hepatic steatosis is very high in patients with T2DM. The patients with significant steatosis had relatively shorter duration of disease. All T2DM patients should be assessed for hepatic complications for the timely identification and management. Future research should focus on longitudinal studies to elucidate causal pathways, incorporate more sensitive diagnostic tools, and explore the interplay between genetic, dietary, and environmental factors in the pathogenesis of NAFLD in T2DM. Addressing modifiable risk factors, particularly obesity and insulin resistance, remains pivotal in mitigating the burden of hepatic steatosis and its complications in T2DM populations.

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Authors' Contribution

QMA,AM: Conception

SA,RK: Design of the work

AI,SS: Data acquisition, analysis, or interpretation

SA,RK,SS,AM: Draft the work

QMA,AI: Review critically for important intellectual content

All authors approve the version to be published

All authors agree to be accountable for all aspects of the work

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