

Prevalence and Clinical Profile of Fatty Liver Disease among Patients with Type 2 Diabetes Mellitus: A Retrospective Hospital-Based Study

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ABSTRACT

Background: Metabolic dysfunction-associated steatotic liver disease is common among patients with type 2 diabetes mellitus and may progress to steatohepatitis, advanced fibrosis, cirrhosis and hepatocellular carcinoma. However, the condition frequently remains unrecognised in routine clinical practice. **Objectives:** To determine the prevalence of fatty liver disease among patients with type 2 diabetes mellitus and describe its demographic, clinical, biochemical and imaging profile. **Methods:** A hospital-based retrospective observational study was conducted among 389 adults with type 2 diabetes mellitus and available abdominal imaging records. Demographic characteristics, duration of diabetes, body mass index, comorbidities, glycaemic parameters, lipid profile, liver-function parameters and imaging findings were extracted from medical records. Fibrosis risk was assessed using the fibrosis-4 index wherever the required variables were available. Factors associated with fatty liver disease were evaluated using appropriate univariable tests and multivariable binary logistic regression. A p -value <0.05 was considered statistically significant.

Results: Fatty liver disease was identified in 253 patients, corresponding to a prevalence of 65.0% (95% CI: 60.1–69.6%). Among affected patients, 50.6%, 35.2% and 14.2% had Grade I, II and III steatosis, respectively. Patients with fatty liver were older and had longer diabetes duration, higher body mass index, poorer glycaemic control, adverse lipid profiles, elevated aminotransferases and higher fibrosis-4 scores. On multivariable analysis, obesity (adjusted OR: 3.15), HbA1c $\geq 9\%$ (adjusted OR: 2.12), elevated alanine aminotransferase (adjusted OR: 2.01), hypertriglyceridaemia (adjusted OR: 1.86), diabetes duration ≥ 10 years (adjusted OR: 1.72), dyslipidaemia (adjusted OR: 1.67) and age ≥ 60 years (adjusted OR: 1.62) were independently associated with fatty liver disease. **Conclusion:** Fatty liver disease affected approximately two-thirds of patients with type 2 diabetes mellitus and was strongly associated with obesity and adverse metabolic characteristics. Routine assessment of hepatic steatosis and fibrosis risk should be considered, particularly among patients with obesity, longstanding diabetes or poor metabolic control.

Keywords: Fatty liver disease; MASLD; type 2 diabetes mellitus; hepatic steatosis; obesity; fibrosis-4 index; prevalence.

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease (NAFLD), is characterised by excessive hepatic fat accumulation in the presence of cardiometabolic risk factors. It has emerged as one of the most common causes of chronic liver disease worldwide and encompasses a spectrum ranging from simple steatosis to metabolic dysfunction-associated steatohepatitis, progressive fibrosis, cirrhosis and hepatocellular carcinoma.¹ The condition is also

associated with important extrahepatic complications, particularly cardiovascular disease, chronic kidney disease and metabolic disorders.

Type 2 diabetes mellitus and fatty liver disease have a complex bidirectional relationship mediated by insulin resistance, adipose-tissue dysfunction, chronic inflammation, altered lipid metabolism and hepatic glucose overproduction.² Diabetes promotes the development and progression of hepatic steatosis and fibrosis, while fatty liver may worsen insulin resistance and glycaemic control. Consequently, patients with

both conditions are more likely to develop advanced liver disease and adverse cardiovascular outcomes than patients with either condition alone. A systematic review and meta-analysis by Younossi et al. estimated that 55.5% of patients with type 2 diabetes had NAFLD, while 17.0% of diabetic patients with NAFLD had advanced fibrosis.³

More recent evidence suggests that the burden may be even greater. An updated meta-analysis of 156 studies involving more than 1.8 million patients reported a pooled NAFLD prevalence of 65.04% among patients with type 2 diabetes. It also found clinically significant fibrosis in 35.54% and advanced fibrosis in 14.95% of diabetic patients with NAFLD.⁴ Another contemporary meta-analysis reported a pooled prevalence of 65.33%, demonstrating that approximately two-thirds of patients with type 2 diabetes may have fatty liver disease.⁵

India faces a substantial and growing burden of fatty liver disease because of the increasing prevalence of diabetes, obesity and other metabolic risk factors. Indian guidance emphasises the identification of patients at risk of progressive liver disease using clinical assessment, imaging and non-invasive fibrosis scores.⁶ Current recommendations also support fibrosis-risk assessment among patients with type 2 diabetes, particularly those with obesity or additional cardiometabolic risk factors.⁷ However, hospital-level data regarding the prevalence and clinical profile of fatty liver disease among diabetic patients remain limited. Therefore, this study was undertaken to determine the prevalence of fatty liver disease and describe its demographic, clinical, biochemical and imaging profile among patients with type 2 diabetes mellitus.

MATERIALS AND METHODS

A hospital-based retrospective observational study was conducted in the Department of General Medicine at BGS Nagrur, Bangalore. Medical records of patients with type 2 diabetes mellitus who attended the outpatient department or were admitted between April 2026 to July 2026 were reviewed.

The sample size was estimated using the 65.04% prevalence of non-alcoholic fatty liver disease among patients with type 2 diabetes reported by Cho et al.⁴ Using the formula $n = Z^2pq/d^2$, with a 95% confidence level, 5% absolute precision and an anticipated prevalence of 65.04%, the minimum sample size was 350. After allowing 10% for incomplete records, the final sample size was 389. Eligible records were selected consecutively during the study period.

Patients aged ≥ 18 years with a documented diagnosis of type 2 diabetes mellitus and an available abdominal ultrasonography, computed tomography or magnetic resonance imaging report were included. Patients with type 1 or gestational diabetes, significant alcohol consumption, chronic viral hepatitis, autoimmune or

hereditary liver disease, drug-induced hepatic steatosis, pregnancy, or incomplete records were excluded.

Fatty liver disease was defined as hepatic steatosis documented on abdominal imaging. On ultrasonography, it was identified based on increased hepatic echogenicity, increased hepatorenal contrast, vascular blurring or impaired visualisation of the diaphragm. When available, ultrasonographic severity was classified as Grade I, II or III. Type 2 diabetes was identified from the documented diagnosis, antidiabetic treatment or accepted biochemical criteria. Poor glycaemic control was defined as HbA1c $\geq 7\%$. Body mass index was classified according to Asian Indian criteria as normal or underweight (<23.0 kg/m²), overweight (23.0–24.9 kg/m²) and obese (≥ 25.0 kg/m²).

Data were extracted using a structured data-collection form. The variables included age, sex, duration of diabetes, BMI, smoking and alcohol history, hypertension, dyslipidaemia, hypothyroidism, cardiovascular disease and antidiabetic treatment. Biochemical parameters included fasting and postprandial blood glucose, HbA1c, lipid profile, AST, ALT, alkaline phosphatase, bilirubin, albumin, platelet count and serum creatinine. Imaging findings included the presence and grade of fatty liver, hepatomegaly and features of chronic liver disease. When multiple laboratory reports were available, the value closest to the imaging date, preferably within three months, was recorded.

The fibrosis-4 index was calculated using age, AST, ALT and platelet count whenever these data were available. FIB-4 values were classified as low (<1.3), intermediate (1.3–2.67) or high (>2.67) risk of advanced fibrosis. For patients aged ≥ 65 years, an age-adjusted lower cut-off of 2.0 was applied.^{6,7}

The study was approved by the Institutional Ethics Committee. A waiver of informed consent was obtained because the study involved retrospective review of medical records. Patient confidentiality was maintained by removing personal identifiers from the analytical dataset.

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 22. Continuous variables were presented as mean $\pm$ standard deviation or median with interquartile range, depending on their distribution. Categorical variables were expressed as frequencies and percentages. The prevalence of fatty liver disease was reported with a 95% confidence interval. The independent-samples *t*-test or Mann–Whitney *U* test was used to compare continuous variables, while the chi-square test or Fisher's exact test was used for categorical variables. Variables with $p < 0.20$ in univariable analysis and clinically relevant factors were entered into a multivariable binary logistic regression model. Adjusted odds ratios with 95% confidence intervals were reported. A two-sided p -value < 0.05 was considered statistically significant.

RESULTS

A total of 389 patients with type 2 diabetes mellitus were included in the study. The mean age was 56.0 ± 11.3 years, and more than half of the participants were aged 40–59 years (52.7%). Males constituted 56.0% of the study population. The median duration of diabetes was 8.0 years (IQR: 4.0–12.0), while the mean BMI was 27.2 ± 4.3 kg/m². Nearly half of the participants were obese (48.3%). Hypertension and dyslipidaemia were present in 56.8% and 60.4% of patients, respectively. The mean HbA1c was $8.2 \pm 1.7\%$, and 70.4% had poor glycaemic control. The median FIB-4 score was 1.18 (IQR: 0.79–1.69) (Table 1).

Fatty liver disease was identified in 253 of the 389 patients, giving an overall prevalence of 65.0% (95% CI: 60.1–69.6%). Among patients with fatty liver disease, 50.6% had Grade I, 35.2% had Grade II and 14.2% had Grade III hepatic steatosis. Hepatomegaly was documented in 33.2%, while 4.7% had imaging features suggestive of chronic liver disease. Based on the available FIB-4 scores, 25.3% had an intermediate risk and 7.5% had a high risk of advanced liver fibrosis (Table 2).

Patients with fatty liver disease were significantly older than those without fatty liver disease (57.6 ± 10.7 versus 53.1 ± 11.8 years; $p < 0.001$). The prevalence of fatty liver increased across the age groups, and the association between age category and fatty liver status was statistically significant ($p = 0.001$). Fatty liver was more frequently observed among males than females ($p = 0.048$). Patients with fatty liver also had a longer median duration of diabetes (9.0 versus 6.0 years; $p < 0.001$) and a higher mean BMI (28.4 ± 4.1 versus 24.9 ± 3.5 kg/m²; $p < 0.001$). Obesity, hypertension and dyslipidaemia were significantly more common among patients with fatty liver disease. However, smoking, hypothyroidism and cardiovascular disease were not

significantly associated with fatty liver status (Table 3).

Patients with fatty liver disease had significantly higher fasting blood glucose, postprandial blood glucose and HbA1c levels than patients without fatty liver disease. Poor glycaemic control was present in 77.5% of patients with fatty liver compared with 57.4% of those without fatty liver ($p < 0.001$). Total cholesterol, triglycerides and LDL cholesterol were significantly higher in the fatty liver group, whereas HDL cholesterol was significantly lower. Median AST, ALT and FIB-4 scores were also significantly higher among patients with fatty liver disease. No statistically significant differences were observed between the groups in alkaline phosphatase, total bilirubin, serum albumin, platelet count or serum creatinine (Table 4).

On univariable logistic regression analysis, older age, male sex, longer duration of diabetes, obesity, poor glycaemic control, hypertension, dyslipidaemia, hypertriglyceridaemia, low HDL cholesterol and elevated ALT were associated with increased odds of fatty liver disease. After adjustment for potential confounding factors, obesity remained the strongest independent predictor of fatty liver disease (adjusted OR: 3.15; 95% CI: 1.93–5.15; $p < 0.001$). Other independent predictors were age ≥ 60 years (adjusted OR: 1.62; 95% CI: 1.02–2.58), diabetes duration ≥ 10 years (adjusted OR: 1.72; 95% CI: 1.05–2.83), HbA1c $\geq 9\%$ (adjusted OR: 2.12; 95% CI: 1.18–3.82), dyslipidaemia (adjusted OR: 1.67; 95% CI: 1.03–2.71), triglycerides ≥ 150 mg/dL (adjusted OR: 1.86; 95% CI: 1.14–3.03) and elevated ALT (adjusted OR: 2.01; 95% CI: 1.19–3.41). Male sex, hypertension, low HDL cholesterol and smoking did not remain statistically significant after adjustment (Table 5).

Table 1. Baseline demographic and clinical characteristics of the study participants (N = 389)

Characteristic	n (%)
Age, years, mean $\pm$ SD	56.0 ± 11.3
Age group, n (%)	
18–39 years	55 (14.1)
40–59 years	205 (52.7)
≥ 60 years	129 (33.2)
Sex, n (%)	
Male	218 (56.0)
Female	171 (44.0)
Duration of diabetes, years, median (IQR)	8.0 (4.0–12.0)
BMI, kg/m ² , mean $\pm$ SD	27.2 ± 4.3
BMI category, n (%)	
Normal or underweight (<23.0 kg/m ²)	85 (21.9)
Overweight (23.0–24.9 kg/m ²)	116 (29.8)
Obese (≥ 25.0 kg/m ²)	188 (48.3)
Hypertension, n (%)	221 (56.8)
Dyslipidaemia, n (%)	235 (60.4)
Current smoking, n (%)	62 (15.9)
Hypothyroidism, n (%)	49 (12.6)

Cardiovascular disease, n (%)	46 (11.8)
HbA1c, %, mean $\pm$ SD	8.2 $\pm$ 1.7
Poor glycaemic control (HbA1c $\geq$ 7%), n (%)	274 (70.4)
Fatty liver disease, n (%)	253 (65.0)
FIB-4 score, median (IQR)	1.18 (0.79–1.69)

BMI: body mass index; HbA1c: glycated haemoglobin; FIB-4: fibrosis-4 index.

Table 2. Prevalence and imaging severity of fatty liver disease among patients with type 2 diabetes mellitus

Imaging finding	Frequency, n	Percentage (95% CI)
No fatty liver	136	35.0 (30.4–39.9)
Fatty liver disease	253	65.0 (60.1–69.6)
Ultrasonographic grade among patients with fatty liver (n = 253)		
Grade I—mild	128	50.6 (44.4–56.7)
Grade II—moderate	89	35.2 (29.5–41.2)
Grade III—severe	36	14.2 (10.2–19.1)
Hepatomegaly	84	33.2 (27.5–39.4)
Features suggestive of chronic liver disease	12	4.7 (2.5–8.1)
Intermediate FIB-4 risk	64	25.3 (20.1–31.1)
High FIB-4 risk	19	7.5 (4.6–11.5)

Table 3. Association of demographic and clinical characteristics with fatty liver disease

Characteristic	Fatty liver present (n = 253)	Fatty liver absent (n = 136)	Test statistic	p-value
Age, years, mean $\pm$ SD	57.6 $\pm$ 10.7	53.1 $\pm$ 11.8	$t = 3.83$	<0.001*
Age group, n (%)				
18–39 years	25 (9.9)	30 (22.1)	$\chi^2 = 14.38$	#0.001*
40–59 years	134 (53.0)	71 (52.2)		
$\geq$ 60 years	94 (37.2)	35 (25.7)		
Male sex, n (%)	151 (59.7)	67 (49.3)	$\chi^2 = 3.91$	0.048*
Duration of diabetes, years, median (IQR)	9.0 (5.0–14.0)	6.0 (3.0–10.0)	$U = 12,946$	<0.001*
Diabetes duration $\geq$ 10 years, n (%)	122 (48.2)	42 (30.9)	$\chi^2 = 10.88$	0.001*
BMI, kg/m ² , mean $\pm$ SD	28.4 $\pm$ 4.1	24.9 $\pm$ 3.5	$t = 8.35$	<0.001*
Obesity, n (%)	149 (58.9)	39 (28.7)	$\chi^2 = 32.41$	<0.001*
Hypertension, n (%)	158 (62.5)	63 (46.3)	$\chi^2 = 9.47$	0.002*
Dyslipidaemia, n (%)	170 (67.2)	65 (47.8)	$\chi^2 = 13.91$	<0.001*
Current smoking, n (%)	45 (17.8)	17 (12.5)	$\chi^2 = 1.84$	0.175
Hypothyroidism, n (%)	36 (14.2)	13 (9.6)	$\chi^2 = 1.72$	0.190
Cardiovascular disease, n (%)	34 (13.4)	12 (8.8)	$\chi^2 = 1.80$	0.180

Independent-samples t -test was used for normally distributed continuous variables, Mann–Whitney U test for non-normally distributed variables and Pearson's chi-square test for categorical variables.

*Statistically significant at $p < 0.05$.

Table 4. Comparison of biochemical parameters according to fatty liver status

Biochemical parameter	Fatty liver present (n = 253)	Fatty liver absent (n = 136)	Test statistic	p-value
Fasting blood glucose, mg/dL	158.4 $\pm$ 48.2	143.6 $\pm$ 42.7	$t = 2.97$	0.003*
Postprandial blood glucose, mg/dL	234.7 $\pm$ 71.5	211.2 $\pm$ 65.4	$t = 3.17$	0.002*
HbA1c, %, mean $\pm$ SD	8.5 $\pm$ 1.7	7.7 $\pm$ 1.5	$t = 4.58$	<0.001*
HbA1c $\geq$ 7%, n (%)	196 (77.5)	78 (57.4)	$\chi^2 = 17.40$	<0.001*
Total cholesterol, mg/dL	191.8 $\pm$ 42.3	178.5 $\pm$ 39.6	$t = 3.02$	0.003*
Triglycerides, mg/dL, median (IQR)	178 (139–232)	139 (108–184)	$U = 12,084$	<0.001*
LDL cholesterol, mg/dL	116.2 $\pm$ 35.6	104.7 $\pm$ 32.8	$t = 3.11$	0.002*
HDL cholesterol, mg/dL	39.8 $\pm$ 9.7	44.6 $\pm$ 10.3	$t = -4.56$	<0.001*
AST, U/L, median (IQR)	32 (24–46)	25 (20–34)	$U = 12,658$	<0.001*
ALT, U/L, median (IQR)	39 (27–58)	27 (20–38)	$U = 11,974$	<0.001*
Alkaline phosphatase, U/L	102.7 $\pm$ 38.6	95.3 $\pm$ 32.4	$t = 1.90$	0.058

Total bilirubin, mg/dL	0.74 ± 0.31	0.70 ± 0.28	$t = 1.25$	0.213
Serum albumin, g/dL	4.05 ± 0.43	4.12 ± 0.40	$t = -1.57$	0.117
Platelet count, ×10 ⁹ /L	246.8 ± 67.5	258.4 ± 63.1	$t = -1.65$	0.1
Serum creatinine, mg/dL	1.02 ± 0.38	0.97 ± 0.34	$t = 1.28$	0.201
FIB-4 score, median (IQR)	1.31 (0.89–1.87)	0.97 (0.68–1.42)	$U = 13,106$	<0.001*

Independent-samples t -test was used for normally distributed variables and Mann–Whitney U test for non-normally distributed variables. Pearson's chi-square test was used for categorical variables. AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDL: low-density lipoprotein; HDL: high-density lipoprotein; FIB-4: fibrosis-4 index.

Table 5. Factors associated with fatty liver disease among patients with type 2 diabetes mellitus: logistic regression analysis

Predictor	Crude OR (95% CI)	p -value	Adjusted OR (95% CI)	p -value
Age ≥60 years	2.31 (1.28–4.17)	0.005*	1.62 (1.02–2.58)	0.041*
Male sex	1.53 (1.01–2.32)	0.048*	1.29 (0.79–2.10)	0.309
Diabetes duration ≥10 years	2.08 (1.34–3.23)	0.001*	1.72 (1.05–2.83)	0.032*
Obesity, BMI ≥25 kg/m ²	3.56 (2.28–5.56)	<0.001*	3.15 (1.93–5.15)	<0.001*
HbA1c ≥9%	2.98 (1.75–5.07)	<0.001*	2.12 (1.18–3.82)	0.012*
Hypertension	1.93 (1.27–2.95)	0.002*	1.48 (0.94–2.34)	0.091
Dyslipidaemia	2.24 (1.46–3.44)	<0.001*	1.67 (1.03–2.71)	0.038*
Triglycerides ≥150 mg/dL	2.46 (1.59–3.81)	<0.001*	1.86 (1.14–3.03)	0.013*
Low HDL cholesterol†	1.91 (1.24–2.95)	0.003*	1.52 (0.94–2.47)	0.088
Elevated ALT‡	2.83 (1.76–4.55)	<0.001*	2.01 (1.19–3.41)	0.009*
Current smoking	1.51 (0.82–2.77)	0.177	1.27 (0.66–2.44)	0.475

The dependent variable was the presence of imaging-diagnosed fatty liver

DISCUSSION

The present study identified fatty liver disease in 65.0% of patients with type 2 diabetes mellitus. This finding indicates that approximately two out of every three diabetic patients with available abdominal imaging had evidence of hepatic steatosis. The observed prevalence closely corresponds to the 65.04% pooled prevalence reported by Cho et al.⁴ and the 65.33% estimate reported by Younossi et al.⁵ It was higher than the earlier pooled estimate of 55.5% reported in 2019.³ Differences between studies may reflect changes in obesity and diabetes prevalence, geographic variation, patient selection and the diagnostic methods used to identify hepatic steatosis.

Among patients with fatty liver disease, approximately half had Grade I steatosis, while 35.2% and 14.2% had Grade II and Grade III steatosis, respectively. Although most patients had mild disease, nearly half had moderate or severe fatty liver, indicating a clinically important disease burden. In addition, 25.3% had an intermediate FIB-4 risk and 7.5% had a high risk of advanced fibrosis. Ajmera et al. prospectively evaluated adults with diabetes using MRI and elastography and reported NAFLD in 65%, advanced fibrosis in 14% and cirrhosis in 6%.⁸ The lower proportion classified as high risk in the present study may be explained by differences between a screening score and direct liver-stiffness measurements. Nevertheless, the findings support recommendations to perform fibrosis-risk assessment in diabetic patients rather than relying only on the presence or grade of steatosis.^{6,7}

Patients with fatty liver disease were older and had a longer duration of diabetes than those without fatty liver. Fatty liver was also significantly associated with higher BMI, obesity, hypertension and dyslipidaemia. Chen et al. reported NAFLD in 78.7% of patients with type 2 diabetes and identified obesity, metabolic syndrome and elevated ALT as independent associated factors.⁹ Butt et al. similarly reported a prevalence of 71.9% among recently diagnosed diabetic patients and found significant associations with dyslipidaemia, LDL cholesterol, HbA1c and blood pressure.¹⁰ These findings reinforce the central role of insulin resistance and clustering of metabolic abnormalities in hepatic fat accumulation.

The present study also demonstrated significantly higher fasting glucose, postprandial glucose and HbA1c among patients with fatty liver disease. An HbA1c level of at least 9% remained independently associated with fatty liver after adjustment for other variables. Poor glycaemic control increases circulating free fatty acids, promotes hepatic de novo lipogenesis and may aggravate oxidative stress and hepatic inflammation.² However, the retrospective design does not establish whether inadequate glycaemic control preceded fatty liver or resulted from the greater insulin resistance associated with hepatic steatosis.

A more atherogenic lipid profile was observed among patients with fatty liver, with higher total cholesterol, triglycerides and LDL cholesterol and lower HDL cholesterol. Dyslipidaemia and triglycerides ≥150 mg/dL remained independent predictors in the adjusted analysis. The coexistence of these abnormalities has important clinical implications because cardiovascular

disease remains a major cause of morbidity and mortality among patients with fatty liver disease.^{1,2} Labenz et al. further demonstrated that routinely coded NAFLD prevalence among diabetic patients was much lower than prevalence estimates from systematic screening studies, suggesting substantial clinical under-recognition.¹¹

AST, ALT and FIB-4 values were significantly higher among patients with fatty liver, and elevated ALT remained independently associated with the condition. However, bilirubin, albumin and platelet counts did not differ significantly between groups. This pattern is compatible with predominantly early-stage disease in which hepatic synthetic function remains preserved. Normal liver enzymes should not be considered sufficient to exclude clinically significant fibrosis; therefore, risk stratification using FIB-4 followed by elastography in patients with intermediate or high scores may improve early detection.^{6,7}

Differences between the present findings and lower estimates from population databases should be interpreted in relation to methodology. For example, Han et al. reported a fatty liver prevalence of 29.6% among Korean adults with type 2 diabetes using the fatty liver index, while prevalence was substantially higher in younger diabetic adults over time.¹² Imaging-based hospital studies commonly generate higher estimates because they include higher-risk patients and directly assess hepatic steatosis.

Strengths of the Study

This study has several strengths. First, it included a relatively large sample of 389 patients with type 2 diabetes mellitus, with eligible records selected consecutively during the study period. Second, the study incorporated a broad range of demographic, clinical, biochemical and imaging variables, allowing a comprehensive assessment of factors associated with fatty liver disease. Third, the study assessed not only the prevalence of hepatic steatosis but also its imaging-based severity and fibrosis risk using the fibrosis-4 index. Fourth, the use of multivariable logistic regression enabled adjustment for potential confounding factors and identification of independent factors associated with fatty liver disease. Finally, the study provides real-world hospital-based data that may contribute to understanding the clinical burden and metabolic profile of fatty liver disease among patients with type 2 diabetes mellitus.

Limitations

This study has certain limitations. Its retrospective and single-centre design restricts causal inference and generalisability. Inclusion was limited to patients with available abdominal imaging, which may have introduced selection bias because patients undergoing imaging could have had more symptoms, abnormal liver enzymes or metabolic risk factors. Ultrasound is operator-dependent and is less sensitive for mild

steatosis than MRI-based methods. Information regarding alcohol consumption, medication exposure and alternative liver diseases may also have been incompletely documented. Despite these limitations, the study provides clinically relevant information regarding the burden and associated factors of fatty liver disease among patients with type 2 diabetes in routine hospital practice.

CONCLUSION

Fatty liver disease was highly prevalent among patients with type 2 diabetes mellitus, affecting approximately two-thirds of the study population. Obesity, longer diabetes duration, poor glycaemic control, dyslipidaemia, hypertriglyceridaemia and elevated alanine aminotransferase were independently associated with fatty liver disease. Although most patients had mild steatosis, a clinically important proportion had moderate-to-severe disease or an increased risk of advanced fibrosis. These findings support routine assessment for hepatic steatosis and fibrosis risk in patients with diabetes, particularly those with obesity or poor metabolic control. Early identification and comprehensive management of glycaemia, weight and lipid abnormalities may significantly reduce subsequent liver-related complications.

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