Original Article

Prevalence and Risk Factors of Non-Alcoholic Fatty Liver Disease in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Background:
Non-alcoholic fatty liver disease is a frequent hepatic complication of type 2 diabetes mellitus and may progress, silently, to clinically important fibrosis.

Aim
The aim of this study was to determine the prevalence of non-alcoholic fatty liver disease among patients with type 2 diabetes mellitus and to evaluate the associated metabolic and clinical risk factors.

Materials and Methods:
This cross-sectional observational study was conducted in a tertiary healthcare setting. Adults underwent abdominal ultrasonography to diagnose hepatic steatosis; vibration-controlled transient. elastography was performed in selected cases to assess steatosis and fibrosis staging. The cohort included 200 participants: 100 with type 2 diabetes mellitus and 100 controls. Multivariable logistic regression was used to identify independent predictors.

Results:
Non-alcoholic fatty liver disease was present in 73.0% of the type 2 diabetes mellitus group versus 8.0% of controls (probability value < 0.001). In the type 2 diabetes mellitus group, steatosis staging was 27.0% (S0), 16.0% (S1), 31.0% (S2), and 26.0% (S3), and fibrosis staging was 45.0% (F0), 30.0% (F1), 14.0% (F2), 6.0% (F3), and 5.0% (F4) (probability value < 0.001). Type 2 diabetes mellitus was independently associated with higher odds of non-alcoholic fatty liver disease (adjusted odds ratio 7.53; 95% confidence interval 2.40–23.62; probability value < 0.001). Body mass index was significant (adjusted odds ratio 1.14 per 1 kilogram per square meter; 95% confidence interval 1.04–1.25; probability value = 0.007), while high-density lipoprotein cholesterol showed an inverse association (adjusted odds ratio 0.93 per 1 milligram per deciliter; 95% confidence interval 0.88–0.98; probability value = 0.011).

Conclusion:
Non-alcoholic fatty liver disease was highly prevalent among patients with type 2 diabetes mellitus and was independently associated with diabetes status and adiposity, while higher high-density lipoprotein cholesterol was protective.

Keywords: Non-alcoholic fatty liver disease; Type 2 diabetes mellitus; Prevalence; Risk factors

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD), now more often termed metabolic dysfunction-associated steatotic liver disease in recent guidelines, is one of the most common chronic liver disorders worldwide. It is closely linked to obesity, insulin resistance, dyslipidemia, and type 2 diabetes mellitus. Although the newer nomenclature is increasingly used, the term NAFLD remains widely recognized in clinical research and is retained in the present study title for consistency. Recent global estimates suggest that fatty liver disease affects about 39% of the general adult population, while the burden rises markedly in patients with type 2 diabetes, among whom pooled prevalence estimates are around 65% to 69% (1,2).

The relationship between NAFLD and type 2 diabetes is not simply an overlap of two common conditions. It is a biologically close and bidirectional association driven largely by insulin resistance, excess free fatty acid flux, glucotoxicity, lipotoxicity, chronic low-grade inflammation, and adipose tissue dysfunction. These mechanisms promote hepatic fat accumulation and, over time, may contribute to hepatocellular injury and fibrogenesis. In clinical practice, this means that patients with type 2 diabetes are not only more likely to develop fatty liver but also more likely to have a more aggressive disease course. Obesity, especially central adiposity, abnormal lipid profile, hypertension, and poor glycemic control, appear to increase this risk further (3–5).

This issue matters because NAFLD in patients with diabetes is not a benign finding. A recent meta-analysis in people with type 2 diabetes found not only a very high prevalence of fatty liver, but also substantial rates of steatohepatitis and fibrosis, including advanced fibrosis in nearly 15% of cases. Such progression has major clinical consequences, since the fibrosis stage is strongly associated with long-term liver-related outcomes. Beyond the liver itself, NAFLD in patients with type 2 diabetes has been linked to higher cardiovascular risk and increased all-cause mortality, even when hepatic steatosis is mild. For that reason, recent liver and diabetes guidance has moved toward active case finding and non-invasive fibrosis assessment in high-risk groups, particularly those with type 2 diabetes (6–9).

Still, prevalence estimates vary across studies because of variation in ethnicity, lifestyle, obesity patterns, glycemic control, diagnostic methods, and study settings. The relative importance of individual risk factors may also vary from one population to another. This makes local data clinically useful. Identifying how common NAFLD is among patients with type 2 diabetes, and which factors are associated with it, may help clinicians recognize high-risk patients earlier and guide preventive strategies before progression to advanced liver disease or cardiovascular complications (2,4,9). Accordingly, the aim of this study is to determine the prevalence of NAFLD among patients with type 2 diabetes mellitus and to identify the main risk factors associated with its presence.

MATERIALS AND METHODS

This cross-sectional observational study was conducted in the ultrasound unit and outpatient clinics of a tertiary healthcare center to determine the prevalence of NAFLD among patients with type 2 diabetes mellitus and to evaluate associated metabolic and clinical factors. The study included 200 adult participants, comprising 100 patients with established type 2 diabetes mellitus and 100 controls. All participants were aged 18 years or older and were evaluated during the study period after providing written informed consent.

Eligible participants in the diabetes group were adults with a confirmed diagnosis of type 2 diabetes mellitus who underwent abdominal ultrasonographic examination and fibroscan assessment. The control group consisted of adults without type 2 diabetes mellitus who were included for comparison. Participants were excluded if they had a history of significant alcohol consumption, known chronic liver disease other than NAFLD, including viral hepatitis, autoimmune hepatitis, or cirrhosis of known etiology, current use of hepatotoxic medications, pregnancy, or incomplete clinical or laboratory data.

Data were collected at the time of evaluation using a structured data collection form. Demographic variables included age and sex. Anthropometric assessment included body weight, height, body mass index, and waist circumference. Clinical variables included blood pressure, smoking status, physical activity level, and relevant comorbidities, including hypertension, dyslipidemia, metabolic syndrome, cardiovascular disease, and chronic kidney disease. In patients with type 2 diabetes mellitus, diabetes-related variables such as duration of diabetes, type of antidiabetic treatment, and glycated hemoglobin were also recorded. Laboratory data included fasting plasma glucose, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transferase, and platelet count.

Abdominal ultrasonography was performed by an experienced radiologist using standard ultrasound equipment. Hepatic steatosis was diagnosed according to established sonographic criteria, including increased hepatic echogenicity relative to the renal cortex, blurring of intrahepatic vascular margins, and attenuation of the ultrasound beam. NAFLD was defined as the presence

of hepatic steatosis on ultrasonography in the absence of significant alcohol intake or other known causes of chronic liver disease. The severity of steatosis was categorized according to sonographic appearance.

All participants also underwent vibration-controlled transient elastography using FibroScan to assess hepatic steatosis and fibrosis. The controlled attenuation parameter was used to quantify hepatic fat content and classify steatosis grade, while liver stiffness measurement was used to estimate the degree of hepatic fibrosis and assign fibrosis stage. FibroScan findings were used to provide a standardized non-invasive assessment of liver steatosis and fibrosis in both study groups.

The primary outcome of the study was the prevalence of NAFLD among patients with type 2 diabetes mellitus, compared with that in controls. Secondary outcomes included steatosis severity, fibrosis stage, and the association of NAFLD with anthropometric, metabolic, biochemical, and clinical variables.

Data were entered and analyzed using statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, as appropriate, while categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using appropriate statistical tests according to variable type and distribution. Multivariable logistic regression analysis was used to identify independent factors associated with non-alcoholic fatty liver disease. Results were expressed as adjusted odds ratios with 95% confidence intervals, and a p-value of less than 0.05 was considered statistically significant.

The study was approved by the institutional review board before data collection. Written informed consent was obtained from all participants, and confidentiality of patient information was maintained throughout the study. All collected data were used exclusively for research purposes.

RESULTS

Participants in the type 2 diabetes group were significantly older than those in the control group (55.32 ± 10.89 vs 48.03 ± 12.75 years, P < 0.001) and had higher body weight (87.48 ± 16.20 vs 75.96 ± 14.39 kg, P < 0.001), BMI (30.95 ± 4.76 vs 26.63 ± 3.90 kg/m², P < 0.001), and waist circumference (104.25 ± 10.73 vs 94.85 ± 9.19 cm, P < 0.001) compared with controls. Systolic and diastolic blood pressures were also higher in the type 2 diabetes group (139.78 ± 14.57 vs 128.28 ± 15.61 mmHg, P < 0.001; 87.87 ± 10.33 vs 82.10 ± 9.29 mmHg, P < 0.001), and hypertensive status was more frequent (66.0% vs 38.0%, P < 0.001). Metabolic syndrome (69.0% vs 16.0%, P < 0.001), dyslipidemia (73.0% vs 31.0%, P < 0.001), cardiovascular disease history (23.0% vs 7.0%, P = 0.002), chronic kidney disease (22.0% vs 7.0%, P = 0.002), smoking status distribution (P = 0.001), physical activity level distribution (P < 0.001), and statin use (63.0% vs 0.0%, P < 0.001) differed significantly between groups, whereas sex distribution (P = 0.474) and height (P = 0.627) did not.(Table 1).

Table 1. Baseline characteristics by study group (n=200).

Characteristic	Type 2 DM (n=100)	Control (n=100)	P value
Age, years	55.32 ± 10.89	48.03 ± 12.75	**<0.001**
Weight, kg	87.48 ± 16.20	75.96 ± 14.39	**<0.001**
Height, cm	167.88 ± 8.08	168.47 ± 9.06	0.627
BMI, kg/m²	30.95 ± 4.76	26.63 ± 3.90	**<0.001**
Waist circumference, cm	104.25 ± 10.73	94.85 ± 9.19	**<0.001**
Systolic BP, mmHg	139.78 ± 14.57	128.28 ± 15.61	**<0.001**
Diastolic BP, mmHg	87.87 ± 10.33	82.10 ± 9.29	**<0.001**
Sex			0.474
Female	55 (55.0%)	60 (60.0%)	
Male	45 (45.0%)	40 (40.0%)	
Presence of Metabolic syndrome	69 (69.0%)	16 (16.0%)	**<0.001**
History of cardiovascular disease	23 (23.0%)	7 (7.0%)	**0.002**
Chronic kidney disease	22 (22.0%)	7 (7.0%)	**0.002**
Presence of hypertensive status	66 (66.0%)	38 (38.0%)	**<0.001**
Smoking status			**0.001**
Current	18 (18.0%)	8 (8.0%)	
Former	35 (35.0%)	46 (46.0%)	
Never	47 (47.0%)	46 (46.0%)	
Physical activity level			**<0.001**
High	12 (12.0%)	32 (32.0%)	
Low	45 (45.0%)	21 (21.0%)	
Moderate	43 (43.0%)	47 (47.0%)	
Presence of dyslipidemia	73 (73.0%)	31 (31.0%)	**<0.001**
Statin use	63 (63.0%)	0 (0.0%)	**<0.001**

Glycemic markers were higher in the type 2 diabetes group, including HbA1c (8.31 ± 1.28% vs 5.17 ± 0.30%, P < 0.001) and fasting plasma glucose (190.41 ± 42.12 vs 100.75 ± 9.77 mg/dL, P < 0.001). Lipid parameters differed

significantly for total cholesterol (187.48 ± 32.49 vs 174.01 ± 30.48 mg/dL, P = 0.003), HDL-C (39.10 ± 7.81 vs 51.72 ± 9.06 mg/dL, P < 0.001), and triglycerides (226.20 ± 78.89 vs 114.10 ± 44.30 mg/dL, P < 0.001), while LDL-C did not differ significantly (103.20 ± 26.27 vs 97.75 ± 24.56 mg/dL, P = 0.131). Liver enzymes were higher in the type 2 diabetes group (ALT 45.12 ± 14.69 vs 22.18 ± 8.07 U/L; AST 38.26 ± 11.24 vs 20.96 ± 7.46 U/L; GGT 50.58 ± 16.30 vs 23.56 ± 11.34 U/L; all P < 0.001). FibroScan-related measures were also higher in the type 2 diabetes group, including LSM (7.23 ± 3.41 vs 4.96 ± 1.52 kPa, P < 0.001) and CAP (268.54 ± 42.87 vs 217.57 ± 24.45 dB/m, P < 0.001), whereas platelet count did not differ significantly (P = 0.319)(Table 2).

Table 2. Laboratory and FibroScan-related parameters by study group (n=200)

Variables	Type 2 DM (n=100)	Control (n=100)	P value
HbA1c, %	8.31 ± 1.28	5.17 ± 0.30	<0.001
Fasting plasma glucose, mg/dL	190.41 ± 42.12	100.75 ± 9.77	<0.001
Total cholesterol, mg/dL	187.48 ± 32.49	174.01 ± 30.48	0.003
LDL-C, mg/dL	103.20 ± 26.27	97.75 ± 24.56	0.131
HDL-C, mg/dL	39.10 ± 7.81	51.72 ± 9.06	<0.001
Triglycerides, mg/dL	226.20 ± 78.89	114.10 ± 44.30	<0.001
ALT, U/L	45.12 ± 14.69	22.18 ± 8.07	<0.001
AST, U/L	38.26 ± 11.24	20.96 ± 7.46	<0.001
GGT, U/L	50.58 ± 16.30	23.56 ± 11.34	<0.001
Platelet count, ×10⁹/L	250.40 ± 54.41	258.41 ± 58.79	0.319
Liver stiffness (LSM), kPa	7.23 ± 3.41	4.96 ± 1.52	<0.001
CAP, dB/m	268.54 ± 42.87	217.57 ± 24.45	<0.001

Liver stiffness measurement (LSM) in kPa assesses fibrosis, while Controlled Attenuation Parameter (CAP) in dB/m quantifies hepatic steatosis (fat) using FibroScan

NAFLD prevalence differed significantly between groups, with 73.0% in the type 2 diabetes group compared with 8.0% in controls (P < 0.001). Steatosis grade distribution was significantly different (P < 0.001), with the type 2 diabetes group showing S0 27.0%, S1 16.0%, S2 31.0%, and S3 26.0%, compared with controls (S0 92.0%, S1 5.0%, S2 2.0%, S3 1.0%). Fibrosis stage distribution also differed significantly (P < 0.001), with F0–F4 frequencies of 45.0%, 30.0%, 14.0%, 6.0%, and 5.0% in the type 2 diabetes group versus 94.0%, 3.0%, 1.0%, 2.0%, and 0.0% in controls, respectively (Table 3).

Table 3. NAFLD prevalence and staging (FibroScan-derived) by study group (n=200)

Outcome / Stage	Type 2 DM (n=100)	Control (n=100)	P value
Presence of NAFLD			**<0.001**
Yes	73 (73.0%)	8 (8.0%)	
No	27 (27.0%)	92 (92.0%)	
Steatosis grade (CAP-derived)			**<0.001**
S0	27 (27.0%)	92 (92.0%)	
S1	16 (16.0%)	5 (5.0%)	
S2	31 (31.0%)	2 (2.0%)	
S3	26 (26.0%)	1 (1.0%)	
Fibrosis stage (LSM-derived)			**<0.001**
F0	45 (45.0%)	94 (94.0%)	
F1	30 (30.0%)	3 (3.0%)	
F2	14 (14.0%)	1 (1.0%)	
F3	6 (6.0%)	2 (2.0%)	
F4	5 (5.0%)	0 (0.0%)	

In multivariable analysis, type 2 diabetes was associated with higher odds of NAFLD (adjusted OR 7.53, 95% CI 2.40–23.62; P < 0.001). BMI was also statistically significant (adjusted OR 1.14 per 1 kg/m², 95% CI 1.04–1.25; P = 0.007), while HDL-C showed a statistically significant inverse association (adjusted OR 0.93 per 1 mg/dL, 95% CI 0.88–0.98; P = 0.011). Age (P = 0.776), male sex (P = 0.555), hypertensive status (P = 0.472), triglycerides (P = 0.102), and low physical activity (P = 0.476) were not statistically significant (Table 4).

Table 4. Multivariable logistic regression for NAFLD (entire cohort, n=200)

Predictor	Adjusted OR	95% CI	P value
Type 2 DM (vs Control)	7.53	2.40–23.62	<0.001
Age (per 1 year)	0.99	0.96–1.03	0.776
Male sex (vs Female)	1.29	0.55–3.03	0.555
BMI (per 1 kg/m²)	1.14	1.04–1.25	0.007
Hypertensive (vs Normotensive)	0.71	0.29–1.78	0.472
Triglycerides (per 1 mg/dL)	1.01	1.00–1.01	0.102
HDL-C (per 1 mg/dL)	0.93	0.88–0.98	0.011
Low physical activity (vs Moderate/High)	1.36	0.58–3.17	0.476

DISCUSSION

This study found a high prevalence of NAFLD among patients with type 2 diabetes mellitus, with clear increases in steatosis burden, fibrosis burden, and adverse metabolic features compared with controls. These findings are consistent with the current understanding that fatty liver disease is a major metabolic complication of diabetes rather than a minor associated condition (4,7,9,10).

The prevalence of NAFLD in the diabetes group was 73.0%, compared with 8.0% in the control group. This estimate is slightly higher than the pooled prevalence reported in recent meta-analyses, including 65.0% by Cho and colleagues (9) and 55.5% by Younossi and others (10), but it is very close to several imaging-based studies in diabetic populations, which reported prevalence figures ranging from about 72% to 84% as was found by Lai et al., Butt et al., Chen et al., Almahmoud et al., Mikolasevic et al., and Barana et al. (11–16). This suggests that the present result lies within the upper range of previously published diabetic cohorts, particularly those using imaging-based assessment. The very low prevalence in controls likely reflects the selected nature of the comparison group rather than the true community prevalence, but this does not alter the main finding that diabetes was strongly associated with hepatic steatosis.

Disease severity was also considerable in the diabetic group. More than half of participants with diabetes had moderate to severe steatosis, while 25.0% had F2-F4 fibrosis and 11.0% had advanced fibrosis or cirrhosis. Among patients with diabetes with NAFLD, clinically significant fibrosis was present in 34.2% and advanced fibrosis in 15.1%. These figures closely match pooled estimates reported by Cho and others and are also comparable to earlier data from Younossi and colleagues (9,10). Similar fibrosis burdens have been reported in FibroScan-based cohorts from different settings, although the exact proportions vary according to referral pathway, diagnostic threshold, and study population (11,15–18). Compared with biopsy-enriched studies, such as Castera and co-workers, the lower advanced fibrosis rate in the present study is expected because the current sample was broader and cross-sectional and relied on non-invasive staging (19).

The diabetes group also showed a more adverse metabolic profile, with older age, greater body weight, higher BMI and waist circumference, and more frequent metabolic syndrome, hypertension, and dyslipidemia. These findings are in agreement with the established metabolic phenotype of diabetic NAFLD. In the multivariable model, BMI remained independently associated with NAFLD, with an adjusted odds ratio of 1.14, which is very similar to estimates reported in previous studies by Kuchay et al. and Dai et al. This supports the central role of adiposity, especially visceral adiposity, in the development of hepatic steatosis among patients with diabetes (17,20).

The lipid profile findings were also consistent with previous literature. Patients with diabetes had higher total cholesterol and triglycerides and lower HDL-C, while only HDL-C remained independently associated with NAFLD after adjustment. The adjusted odds ratio of 0.93 suggests that higher HDL-C was associated with lower odds of fatty liver. Similar inverse associations between HDL-C and NAFLD or fibrosis have been described in earlier studies (11,17,21). The loss of significance of triglycerides in the final model may reflect overlap with obesity and other metabolic variables.

Although age, hypertension, triglycerides, male sex, and low physical activity showed group differences, they were not independently associated with NAFLD after adjustment. This pattern is not unusual in metabolic liver disease, where several variables reflect the same underlying metabolic burden. Poor glycemic control was also more pronounced in the diabetes group, with substantially higher HbA1c and fasting plasma glucose levels. While glycemic measures were not included in the multivariable model, the observed differences are consistent with reports linking worse glycemic control to greater NAFLD burden and fibrosis severity (12,22).

Liver enzymes and FibroScan-related measures showed a coherent pattern. ALT(Alanine Aminotransferase), AST (Aspartate transaminase), and GGT (Gamma-glutamyltransferase) were all higher in the diabetes group, and both CAP(Controlled Attenuation Parameter) and liver stiffness were significantly increased. These findings agree with previous studies showing that abnormal liver enzymes often accompany diabetic NAFLD, although they do not reliably exclude significant fibrosis when normal (11,16,18,20). The present results therefore support the clinical value of structured non-invasive assessment, particularly in patients with type 2 diabetes, in line with recent guideline recommendations (4).

A key finding of this study was that type 2 diabetes remained a strong independent predictor of NAFLD after adjustment, with an adjusted odds ratio of 7.53. This suggests that diabetes is not simply a coexisting condition but a major pathophysiological driver of fatty liver. This interpretation is consistent with current evidence describing a close bidirectional link between diabetes, insulin resistance, hepatic fat accumulation, and fibrosis progression (4,23). Overall, the findings indicate that NAFLD in patients with type 2 diabetes should be viewed as part of a wider network of metabolic and vascular disease rather than an incidental liver abnormality. Early recognition and non-

invasive assessment of fibrosis in patients with diabetes may therefore have important clinical value.

CONCLUSION:

In conclusion, NAFLD was highly prevalent among patients with type 2 diabetes mellitus in this study and was far more common than in controls. Patients with type 2 diabetes also showed more severe steatosis and a markedly greater fibrosis burden, including clinically important proportions with F2-F4 and F3-F4 disease. After adjustment for major metabolic variables, type 2 diabetes remained a strong independent correlate of NAFLD. Higher BMI independently increased the odds of NAFLD, whereas higher HDL-C showed a protective inverse association.

Based on these findings, patients with type 2 diabetes should be evaluated more actively for fatty liver disease, particularly when obesity, central adiposity, dyslipidemia, or abnormal liver enzymes are present. Assessment should not stop at detecting steatosis alone, but should also aim to identify fibrosis at an early stage. Weight reduction and broader metabolic optimization should be emphasized, given the clear independent association between adiposity and NAFLD, while closer attention should also be paid to HDL-C and overall lipid control. Future research should include larger multicenter studies with longitudinal follow-up to better define temporal relationships and disease progression, and should further explore the roles of diabetes duration, medication exposure, diet, and insulin resistance markers. Combining routine clinical data with non-invasive liver assessment may improve early identification of high-risk patients with diabetes.

CONFLICTS OF INTEREST

The authors declare no conflict of interest related to this work.

REFERENCES:

1. Younossi ZM, Golabi P, Price JK, Owrangi S, Gundu-Rao N, Satchi R, et al. The Global Epidemiology of Nonalcoholic Fatty Liver Disease and Nonalcoholic Steatohepatitis Among Patients With Type 2 Diabetes. *Clin Gastroenterol Hepatol*. 2024;22:1999-2010.e8.
2. Younossi ZM, Golabi P, Paik J, Owrangi S, Yilmaz Y, El-Kassas M, et al. Prevalence of metabolic dysfunction-associated steatotic liver disease in the Middle East and North Africa. *Liver Int Off J Int Assoc Study Liver*. 2024;44:1061–70.
3. Colosimo S, Tan GD, Petroni ML, Marchesini G, Tomlinson JW. Improved glycaemic control in patients with type 2 diabetes has a beneficial impact on NAFLD, independent of change in BMI or glucose lowering agent. *Nutr Metab Cardiovasc Dis*. 2023;33:640–8.
4. Nogueira JP, Cusi K. Role of Insulin Resistance in the Development of Nonalcoholic Fatty Liver Disease in People with Type 2 Diabetes: From Bench to Patient Care. *Diabetes Spectr*. 2024;37(1):20-28.
5. Latif S, Ahsan T. Prevalence of Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD) in Persons with Obesity and Type 2 Diabetes Mellitus: A Cross-sectional Study. *Euroasian J Hepato-Gastroenterol*. 2024;14:129–33.
6. Zoncapè M, Tsochatzis EA. Liver Fibrosis Testing in Patients With Type 2 Diabetes: The Time Is Now. *Diabetes Care*. 2025;48:871–3.
7. Tacke F, Horn P, Wong VW-S, Ratziu V, Bugianesi E, Francque S, et al. EASL–EASD–EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). *J Hepatol*. 2024;81:492–542.
8. Kim KS, Hong S, Han K, Park CY. Association of non-alcoholic fatty liver disease with cardiovascular disease and all cause death in patients with type 2 diabetes mellitus: nationwide population based study. *The BMJ* 2024;384:e076388.
9. En Li Cho E, Ang CZ, Quek J, Fu CE, Lim LKE, Heng ZEQ, et al. Global prevalence of non-alcoholic fatty liver disease in type 2 diabetes mellitus: an updated systematic review and meta-analysis. *Gut*. 2023;72:2138–48.
10. Younossi ZM, Golabi P, de Avila L, Minhui Paik J , Srishord M , Fukui N, et al. The global epidemiology of NAFLD and NASH in patients with type 2 diabetes: A systematic review and meta-analysis. *J Hepatol*. 2019;71:793–801.
11. Lai LL, Wan Yusoff WNI, Vethakkan S, Nik Mustapha NR, Mahadeva S, Chan WK. Screening for non-alcoholic fatty liver disease in patients with type 2 diabetes mellitus using transient elastography. *J Gastroenterol Hepatol*. 2019;34:1396–403.
12. Butt AS, Hamid S, Ahmad W, Sharif F , Salih M , Awan S, et al. Nonalcoholic Fatty Liver Diseases among Recently Diagnosed Patients with Diabetes Mellitus and Risk Factors. *Euroasian J Hepato-Gastroenterol*. 2019;9:9–13.
13. Chen K, Sng WK, Quah JHM, Liu J, Chong BY, Khim Lee H, et al. Clinical spectrum of non-alcoholic fatty liver disease in patients with diabetes mellitus. *PLOS One*. 2020;15:e0236977.
14. Almahmoud MH, Al Khawaja NM , Alkinani A , Khader Y , Ajlouni KM . Prevalence of fatty liver disease and its associated factors among Jordanian patients with type 2 diabetes mellitus: A cross-sectional study. *Ann Med Surg*. 2021;68:102677.
15. Mikolasevic I, others. Screening for nonalcoholic fatty liver disease in patients with type 2 diabetes mellitus using transient elastography - a prospective, cross sectional study. *Eur J Intern Med*. 2020;82:68–75.
16. Barana L, Nelva A, Scivetti P, Costanzo M, Fanelli CG. Diagnosis of hepatic steatosis and steatohepatitis in people with new-onset type 2 diabetes: a multidisciplinary approach. *Endocrine* 2024;84:412–9.

17. Kuchay MS, Choudhary NS, Mishra SK, Bano T, Gagneja S, Mathew A, et al. Prevalence of clinically relevant liver fibrosis due to nonalcoholic fatty liver disease in Indian individuals with type 2 diabetes. *JGH Open* 2021;5:915–22. https://doi.org/10.1002/jgh3.12606.
18. Lee XH, Nor LM, Ang CS, Yeow TP, Lim SL. The Prevalence of Advanced Liver Fibrosis Among Patients With Type 2 Diabetes Mellitus: A Single-Centre Experience in Penang, Malaysia. *J ASEAN Fed Endocr Soc* 2023;38:52–61.
19. Castera L, Laouenan C, Vallet-Pichard A, Vidal-Trécan G, Manchon P, Paradis V, et al. High Prevalence of NASH and Advanced Fibrosis in Type 2 Diabetes: A Prospective Study of 330 Outpatients Undergoing Liver Biopsies for Elevated ALT, Using a Low Threshold. *Diabetes Care*. 2023;46:1354-62.
20. Dai CY, Fang TJ, Hung WW, Tsai HJ, Tsai YC. The Determinants of Liver Fibrosis in Patients with Nonalcoholic Fatty Liver Disease and Type 2 Diabetes Mellitus. *Biomedicines* 2022;10:1487.
21. Trojak A, Waluś-Miarka M, Woźniakiewicz E, Trojak A, Waluś-Miarka M, Woźniakiewicz E.Nonalcoholic fatty liver disease is associated with low HDL cholesterol and coronary angioplasty in patients with type 2 diabetes. *Med Sci Monit* 2013;19:1167–72.
22. Asero C, Giandalia A, Cacciola I, Morace C, Lorello G, Caspanello AR, et al. High Prevalence of Severe Hepatic Fibrosis in Type 2 Diabetic Outpatients Screened for Non-Alcoholic Fatty Liver Disease. *J Clin Med*. 2023;12:2858.
23. Ciardullo S, Vergani M, Perseghin G. Nonalcoholic Fatty Liver Disease in Patients with Type 2 Diabetes: Screening, Diagnosis, and Treatment. *J Clin Med*. 2023;12:5597.