Vol. 5 No. 1 (2026)

Pakistan Journal of Medical & Cardiological Review

https://pakjmcr.com/index.php/1/about

Online ISSN 3007-2387

Print ISSN 3007-2379

Prevalence of Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) and Advanced Fibrosis in Type 2 Diabetes Mellitus Patients

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Received on 25 Dec, 2025
Accepted on 18 Jan, 2026
Published on 21 Jan, 2026

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Abstract

Background: Type 2 Diabetes Mellitus (T2DM) plays a key role in the development of Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) and advanced fibrosis, which is the strongest predictor of liver-related mortality. Identifying "silent" advanced disease in Pakistan, where T2DM prevalence is exceptionally high, through cost-effective non-invasive tests (NITs) is essential for early identification of risk factors and specialist referral accordingly. Objective: To estimate the prevalence of Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) and advanced liver fibrosis in patients with Type 2 Diabetes Mellitus (T2DM) using non-invasive tests (NITs) in a tertiary care setting in Pakistan, and to identify clinical predictors of advanced disease. Methods: This cross-sectional study analyzed 180 adult patients with T2DM presenting to the Diabetes Management Center, Services Institute of Medical Sciences (SIMS), Lahore. Clinical and biochemical data were extracted from the study dataset. The Hepatic Steatosis Index (HSI) was calculated to identify likely steatosis (cutoff $\geqslant$ 36), and the Fibrosis-4 (FIB-4) index was used to stratify the risk of advanced fibrosis (cutoff $\geqslant$ 2.67). Data analysis involved descriptive statistics and independent t-tests, with complete-case analysis applied to handle missing data for

the primary outcome. Results: Valid FIB-4 scores were calculable for 180 patients (82.6%) . Mean age of the cohort was 48.6 ± 11.2 years, and the mean Body Mass Index (BMI) was 28.4 ± 5.8 kg/m². The prevalence of MAFLD (HSI ⩾ 36) was found to be 68.3% (123/180). The prevalence of likely advanced fibrosis (FIB-4 ⩾ 2.67) was 5.6% (10/180), while 22.2% (40/180) were categorized as indeterminate risk (FIB-4 1.30–2.67). Patients with advanced fibrosis were significantly older (60.1 vs. 48.2 years, p<0.01) with a longer duration of diabetes (15.2 vs. 8.4 years, p<0.01) as compared to the non-advanced fibrosis group. Platelet counts were very low in the high-risk group (168 vs. 282 × 10^9/L, p<0.001). Conclusions: MAFLD is highly endemic among Pakistani T2DM patients, and affect more than two-thirds of the study population. Although the point prevalence of advanced fibrosis (5.6%) appears small but still significant as it identifies a critical high-risk subgroup that should bereferred to a hepatologist immediately. Age and duration of diabetes are strong predictors of fibrosis risk. Routine screening with FIB-4 should be included into diabetes care protocols in Pakistan.

Keywords: MAFLD, MASLD, Type 2 Diabetes, Fibrosis-4 (FIB-4), Hepatic Steatosis Index, Pakistan.

1. Introduction

Diabetes mellitus (DM) has become a global health emergency, with Pakistan reporting some of the highest prevalence rates worldwide (1). According to the International Diabetes Federation (IDF) 10th Atlas, over 33 million adults in Pakistan are diabetics, making Pakistan highly prevalent in metabolic dysfunction. (2). Type 2 Diabetes Mellitus (T2DM) affects multiple organ systems, with the liver being the primary target.

The hepatic manifestation of metabolic dysfunction, previously known as Non-Alcoholic Fatty Liver Disease (NAFLD), has recently been redefined as Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) (3) or Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) (4). This new nomenclature, implemented by the 2025 HEPNET Pakistan Position Statement, highlights the pathogenic role of metabolic dysregulation, specifically insulin resistance, independent of other liver etiologies (5). Global data suggests that MAFLD affects60–70% patients with T2DMand approximately 25–30% of the general population (6). The coexistence of T2DM and MAFLD is mutually reinforcing; diabetes accelerates the progression of simple steatosis

to metabolic dysfunction-associated steatohepatitis (MASH) and advanced fibrosis (7). Advanced liver fibrosis (stages F3–F4) is the single strongest predictor of liver-related mortality, decompensated cirrhosis, and Hepatocellular Carcinoma (HCC) (8). Despite this, liver disease often remains "silent" and undiagnosed in diabetes clinics until irreversible complications occur.

Liver biopsy is the gold standard for staging fibrosis but is invasive, costly, and impractical for routine screening (9). Due to this, the American Diabetes Association (ADA) 2025 Standards of Care emphasizes on using non-invasive tests (NITs) such as the Fibrosis-4 (FIB-4) index for risk stratification (10). In Pakistan, where healthcare resources are limited with highly prevalent hepatitis C (11), validated NITs are needed for triage. This study aims to estimate the prevalence of MAFLD and advanced fibrosis in a Pakistani T2DM cohort using the HSI and FIB-4 indices and to determine clinical predictors of severe disease.

2. Methods

2.1 Study Design and Setting

This cross-sectional study analyzed data from patients presenting to the Diabetes Management Center (DMC) at the Services Institute of Medical Sciences (SIMS), Lahore. This tertiary care facility serves a wide ranging urban and peri-urban population in Punjab, Pakistan.

2.2 Study Population

The dataset comprised 218 adult patients with a confirmed diagnosis of Type 2 Diabetes Mellitus.

- Inclusion Criteria: Adults (Age > 18 years) with diagnosed T2DM.
- Exclusion Criteria: Patients with data for AST, ALT, or platelets (which renders FIB-4 incalculable). Patients with known etiologies of liver disease other than metabolic syndrome (e.g., viral hepatitis, alcoholic liver disease) were also excluded as per the study protocol (12).

2.3 Data Collection and Definitions

Clinical parameters including age, sex, weight, height, Body Mass Index (BMI), duration of diabetes, Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), and platelet count were extracted from the dataset.

Operational Definitions

- Hepatic Steatosis Index (HSI): Used to screen for hepatic steatosis (fatty liver). HSI = 8 × (ALT/AST) + BMI + 2 (ifdiabetic) + 2 (if female)

where ALT and AST are in U/L, and BMI is in kg/m²; add +2 if the patient has diabetes (binary) and +2 if the patient is female (binary). HSI is a unitless score; HSI ⩾ 36 indicates likely hepatic steatosis (13).

- Fibrosis-4 Index (FIB-4): Used to stratify the risk of advanced liver fibrosis (F3-F4). FIB-4 = (Age × AST) / (Platelets × $\sqrt{}$ALT)

where Age is in years, AST and ALT in U/L, and platelet count in $\times 10^9$/L.
Low Risk: FIB-4 < 1.30 (High Negative Predictive Value), Indeterminate
Risk: FIB-4 = 1.30 – 2.66,
High Risk: FIB-4 ⩾ 2.67 (High Specificity for advanced fibrosis) (14).

2.4 Statistical Analysis

Data were analyzed using Python scripting. Continuous variables were expressed as mean ± standard deviation (SD). Categorical variables were presented as frequencies (n) and percentages (%).

- A complete-case analysis (N=180) was performed for the primary outcomes to ensure accuracy of the composite scores.
- Hypothesis Testing: Independent t-tests were used to compare continuous variables (Age, BMI, Duration) between the "Advanced Fibrosis" (High Risk) and "Non-Advanced Fibrosis" groups. A p-value < 0.05 was considered statistically significant.

3. Results

3.1 Baseline Characteristics

A total of 180 patients had complete laboratory and clinical data sufficient for fibrosis scoring and are included in the analytic sample. Baseline demographic and biochemical characteristics are summarized in Table 1. Continuous variables are presented as mean ± standard deviation (SD) and ranges are shown where appropriate.

	Mean ± SD	Range
Age (years)	48.6 ± 11.2	20 – 80
BMI (kg/m²)	28.4 ± 5.8	10.4 – 51.1

	Mean ± SD	Range
Duration of Diabetes (years)	8.9 ± 7.2	0.5 – 33
AST (U/L)	32.4 ± 18.6	11 – 129
ALT (U/L)	35.8 ± 21.3	11 – 163
Platelets (× 10^9/L)	276 ± 98	70 – 655

The cohort was predominantly middle-aged (mean age 48.6 ± 11.2 years) with a mean BMI of 28.4 ± 5.8 kg/m², placing the average patient in the "obese" category according to South Asian specific cutoffs (BMI ⩾ 25) (15).The median duration of diabetes was modest (mean 8.9 ± 7.2 years). Mean transaminases were only mildly elevated on average (AST 32.4 ± 18.6 U/L, ALT 35.8 ± 21.3 U/L), and mean platelet count was 276 ± 98 ×10^9/L. These baseline data demonstrate high metabolic risk in clinic population, suitable for application of routine non-invasive steatosis and fibrosis screening.

3.2 Prevalence of MAFLD (Steatosis) and Advanced Fibrosis (FIB-4)

Using the Hepatic Steatosis Index (HSI; HSI ⩾ 36 = likely steatosis) and the FIB-4 index (FIB-4 cutoffs as defined in Methods), we estimated the prevalence of probable MAFLD and of FIB-4–based fibrosis risk categories in the complete-case sample. Prevalence counts and proportions are presented in Table 2.

Table 2: PreavlenceofMAFLDandAdvancediFbriso

Clinical Condition	Definition	Frequency (n)	Prevalence
MAFLD (Likely Steatosis)	HSI ⩾ 36	123	68.3%
No/Low Steatosis	HSI < 36	57	31.7%
Advanced Fibrosis Risk	FIB-4 ⩾ 2.67	10	5.6%
Indeterminate Fibrosis Risk	FIB-4 1.30 – 2.66	40	22.2%
Low Fibrosis Risk	FIB-4 < 1.30	130	72.2%

By HSI, 123/180 (68.3%) of patients had scores consistent with likely hepatic steatosis (HSI ⩾ 36), while 57/180 (31.7%) had HSI < 36. FIB-4 classification showed that a minority of patients were at high risk for advanced fibrosis: 10/180 (5.6%) had FIB-4 ⩾

2.67. The indeterminate or "grey-zone" group (FIB-4 1.30–2.66) comprised 40/180 (22.2%),wherefibrosis cannot be ruled out without secondary testing.The majority (130/180; 72.2%) were in the low-risk category (FIB-4 < 1.30). Together these results indicate a high burden of probable steatosis in this T2DM clinic population, with a modest but clinically important subgroup at high risk for advanced fibrosis, who should be prioritized for confirmatory elastography or hepatology referral.

3.3 Predictors of Advanced Fibrosis

To explore clinical variables associated with high FIB-4 (⩾2.67), we compared demographic and laboratory parameters between patients with advanced-risk FIB-4 and those with non-advanced FIB-4 (FIB-4 < 2.67). Univariable comparisons are summarized in Table 3. Due to the small number of high-risk events (n = 10), multivariable modelling is limited by low event count and should be interpreted cautiously unless supported by pooled or larger samples.

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	Non-Advanced Fibrosis (FIB-4 < 2.67)	Advanced Fibrosis (FIB-4 ⩾ 2.67)	P-value
Age (years)	48.2 ± 10.9	60.1 ± 8.5	< 0.01
BMI (kg/m²)	28.5 ± 5.9	27.2 ± 4.3	0.45
Duration of Diabetes (years)	8.4 ± 6.9	15.2 ± 8.1	< 0.01
AST (U/L)	31.2 ± 17.8	53.6 ± 21.4	< 0.001
Platelets (× 10^9/L)	282 ± 95	168 ± 70	< 0.001

Patients with FIB-4 ⩾ 2.67 were significantly older (mean 60.1 ± 8.5 years) than those with FIB-4 < 2.67 (mean 48.2 ± 10.9 years; p < 0.01), and had a substantially longer duration of diabetes (15.2 ± 8.1 vs 8.4 ± 6.9 years; p < 0.01). Markers consistent with more advanced liver disease were also observed in the high-risk group: mean AST was higher (53.6 ± 21.4 U/L vs 31.2 ± 17.8 U/L; p < 0.001), and platelet counts were markedly lower (168 ± 70 ×10^9/L vs 282 ± 95 ×10^9/L; p < 0.001), consistent with hypersplenism secondary to portal hypertension. BMI did not differ significantly between groups (27.2 vs 28.5 kg/m²; p = 0.45), indicating that advanced fibrosis in this cohort was driven more

by age and disease duration than by higher body mass alone. These univariable findings identify older age, longer diabetes duration, elevated transaminases and lower platelet counts as the clinical features most strongly associated with a FIB-4 result suggesting advanced fibrosis in this sample.

4. Discussion

This study highlights the high burden of liver disease within a diabetes clinic in Lahore. We found a 68.3% prevalence of MAFLD (steatosis) using the HSI score. This result aligns closely with recent data from Pakistan; for instance, a 2025 study by Memon et al. reported a high prevalence of fatty liver in the diabetic population (16), and Latif et al. (2024) reported a MASLD prevalence of 61.3% in patients with obesity and T2DM (17). Other local data reinforces this trend, with Khan et al. (2024) finding similar rates in a veterinary and medical collaborative study (18). These findings confirm that hepatic steatosis is the norm rather than the exception for Pakistani diabetic patients.

The prevalence of advanced fibrosis (5.6%) observed in our cohort is clinically significant. Although lower than Western estimates of ~15-20%, it is comparable with regional studies. A recent meta-analysis of South Asian populations reported variable fibrosis rates depending on the diagnostic method used (19). In Pakistan, Kakar et al. (2024) validated the use of FIB-4, demonstrating its utility in resource-limited settings (20). However, that standard cutoffs might underestimate disease in younger, non-obese Asian populations, as suggested by Deb et al. in a recent Indian cohort study (21). Recent work by Batool et al. (2025) in Rawalpindi utilized FIB-4 and found a slightly higher cirrhosis risk (14.4%) in a hospital-based cohort, suggesting that inpatient populations may carry a higher disease burden than our outpatient sample (22).

Our analysis identified Age and Duration of Diabetes as the strongest predictors of fibrosis. This supports the concept of "time-toxicity," where prolonged exposure to hyperglycemia and insulin resistance drives cumulative hepatic injury (23). Interestingly, BMI was not elevated in the advanced fibrosis group. This phenomenon is consistent with "burnt-out NASH," where advanced fibrosis leads to hepatic shrinkage and a paradoxical reduction in steatosis (24). Clinically, this highlights that normal BMI does not rule out advanced liver disease in older diabetics with long-standing diabetes. The detection of silent fibrosis is critical because MAFLD is associated with cardiovascular

complications, which remain the leading cause of mortality in this group. Early identification allows for the targeted use of newer therapeutic agents such as SGLT2 inhibitors and GLP-1 receptor agonists, which have demonstrated benefits in both diabetic and hepatic parameters. The lack of routine screening remains a gap; Ananchaisarp et al. noted similar screening deficits in other Asian primary care settings (25). In resource-poor settings where FibroScan is not available, FIB-4 correlates well enough to serve as a "gatekeeper" for referral (26).

Limitations: The primary limitation was the cross-sectional design and the lack of histological validation (liver biopsy) or elastography (FibroScan) for all patients. Additionally, 17.4% of the original dataset was excluded due to missing labs, which may introduce selection bias. However, the use of validated NITs (FIB-4, HSI) provides a pragmatic, real-world assessment of disease load, a necessity emphasized by Bhatti and Kamani in their editorial on the Pakistani context (27).

5. Conclusion

MAFLD is endemic in the Pakistani Type 2 Diabetes population, with nearly 70% affected by steatosis and 5.6% at high risk for advanced fibrosis. The strong association of fibrosis with age and diabetes duration, independent of BMI, highlights the need for a change in screening practices. We recommend that the FIB-4 index be calculated annually for all T2DM patients in Pakistan to identify those requiring urgent hepatology referral (28). Early detection is important to prevent disease progression to decompensated cirrhosis and hepatocellular carcinoma, and to manage cardiovascular risk factors aggressively (29). As the global burden of metabolic disease rises, local data such as this is essential for health policy planning (30). Routine screening guidelines, as recomended by Wongtrakul et al. (31), and Schwärzler J et al. (32), must be implemented immediately.

References

1. Azeem S, Khan U, Liaquat A. The increasing rate of diabetes in Pakistan: A silent killer. Ann Med Surg (Lond). 2022;79:103901.https://doi.org/10.1016/j.amsu.2022.103901
2. Magliano DJ, Boyko EJ; IDF DIABETES ATLAS. 10th edition. Brussels: International Diabetes Federation; 2021. Available from: https://www.ncbi.nlm.nih.gov/books/NBK581934/

3. Eslam M, Newsome PN, Sarin SK, et al. A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. J Hepatol. 2020;73(1):202-209.https://doi.org/10.1016/j.jhep.2020.03.039
4. Rinella ME, Lazarus JV, Ratziu V, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. J Hepatol. 2023;79(6):1542-1556.https://doi.org/10.1016/j.jhep.2023.06.003
5. Ali B, Kamani L, Salim A, et al. HEPNET Position Statement-I, Case Definition, Classification, Screening & Diagnosis of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) in Pakistan. Pak J Med Sci. 2025;41(3):929-938. https://pmc.ncbi.nlm.nih.gov/articles/PMC11911726/
6. Younossi ZM, Golabi P, de Avila L, 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(4):793-801.https://doi.org/10.1016/j.jhep.2019.06.021
7. Bhatt HB, Smith RJ. Fatty liver disease in diabetes mellitus. Hepatobiliary Surg Nutr. 2015;4(2):101-108.https://doi.org/10.3978/j.issn.2304-3881.2015.01.03
8. Taylor RS, Taylor RJ, Bayliss S, et al. Association BetweenFibrosis Stage and Outcomes of Patients With Nonalcoholic Fatty Liver Disease: A Systematic Review and Meta-Analysis. Ann Hepatol. 2022;27(1):100561.https://doi.org/10.1053/j.gastro.2020.01.043
9. Castera L, Friedrich-Rust M, Loomba R. Noninvasive Assessment of Liver Disease in Patients With Nonalcoholic Fatty Liver Disease. Gastroenterology. 2019;156(5):1264-1281.https://doi.org/10.1053/j.gastro.2018.12.036
10. American Diabetes Association Professional Practice Committee. 4. Comprehensive Medical Evaluation and Assessment of Comorbidities: Standards of Care in Diabetes—2025. Diabetes Care. 2025;48(Suppl 1):S59-S92. https://diabetesjournals.org/care/issue/48/Supplement_1
11. Qureshi H, Alam E, Dharejo Z, Mahmood H. Prevalence of hepatitis and HIV in Pakistan. East Mediterr Health J. 2024;30(10):689-697. https://pubmed.ncbi.nlm.nih.gov/39574368/

12. Basit A, Fawwad A, Qureshi H, Shera AS. Prevalence of diabetes, pre-diabetes and associated risk factors: second National Diabetes Survey of Pakistan (NDSP), 2016–2017. BMJ Open. 2018;8(8):e020961.https://doi.org/10.1136/bmjopen-2017-020961
13. Lee JH, Kim D, Kim HJ, et al. Hepatic steatosis index: a simple screening tool reflecting nonalcoholic fatty liver disease. Dig Liver Dis. 2010;42(7):503-508.https://doi.org/10.1016/j.dld.2009.08.002
14. Sterling RK, Lissen E, Clumeck N, et al. Development of a simple noninvasive index to predict significant fibrosis in patients with HIV/HCV coinfection. Hepatology. 2006;43(6):1317-1325.https://doi.org/10.1002/hep.21178
15. Misra A, Khurana L. The metabolic syndrome in South Asians: epidemiology, determinants, and prevention. Metab Syndr Relat Disord. 2009;7(6):497-514.https://doi.org/10.1089/met.2009.0024
16. Memon HL, Farooq S, Aslam M, et al. Fatty Liver Disease in the Diabetic Population: A Cross-Sectional Study From Pakistan. Cureus. 2025;17(6):e85510.https://doi.org/10.7759/cureus.85510
17. 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(2):129-133.https://doi.org/10.5005/jp-journals-10018-1437. Also available from: https://pubmed.ncbi.nlm.nih.gov/39802849/
18. Khan F, Khan UM, Khan AM, Khan UM. Trend of Non-Alcoholic Fatty Liver Disease in Type II Diabetes Mellitus. Pak J Med Dent. 2024;13(1):11-16.https://doi.org/10.36283/PJMD13-1/004
19. James M, Smuk M, Rahman N, et al. Epidemiology of Metabolic Dysfunction-Associated Steatotic Liver Disease in South Asian Ethnicities: A Systematic Review andMeta-Analysis. J Gastroenterol Hepatol. 2025. doi:10.1111/jgh.70080.https://doi.org/10.1111/jgh.70080
20. Kakar F, Dar MA, Rind SRA, et al. Comparison of Fibrosis-4 with FibroScan for Liver Fibrosis Assessment in Non-Alcoholic Fatty Liver Disease Patients: A Cross-sectional Study. J Dow Univ Health Sci. 2024;18(2):79-83.https://doi.org/10.36570/jduhs.2024.2.2167

21. Deb R, et al. Redefining Liver Fibrosis Risk Assessment in Indians with Type 2 Diabetes: New FIB-4 Score Cutoff for Optimizing Sequential Assessment with Transient Elastography. Indian J Endocrinol Metab. 2025;29(2):237.https://doi.org/10.4103/ijem.ijem_457_24
22. Batool R, Fatima K, Pervez H, et al. Frequency of NAFLD related cirrhosis in Diabetes Mellitus (DM) using APRI and fibrosis-4 (FIB-4) score. Pak Postgrad Med J. 2025;36(1):15-19.https://doi.org/10.51642/ppmj.v36i03.803
23. Ciardullo S, Perseghin G. Prevalence of elevated liver stiffness in patients with type 1 and type 2 diabetes: A systematic review and meta-analysis. Diabetes research and clinical practice, 190, 109981.https://doi.org/10.1016/j.diabres.2022.109981
24. Van der Poorten D, Samer CF, Ramezani-Moghadam M, et al. Hepatic fat loss in advanced nonalcoholic steatohepatitis: Are alterations in serum adiponectin the cause?. Hepatology (Baltimore, Md.), 57(6), 2180–2188.https://doi.org/10.1002/hep.26072
25. Ananchaisarp T, Phisalprapa P. Prevalence of advanced fibrosis risk using FIB-4 in patients with type 2 diabetesand its associated factors: a cross-sectional study in a primary care unit of a university hospital in Southern Thailand. BMC Prim Care. 2025;26(1):15.https://doi.org/10.1186/s12875-025-03039-x
26. Das AK, Sharma A, Labrez MZ. Correlation of FIB-4 and APRI Score with FibroScan Score to Predict Fibrosis in Obese Type-2 Diabetic Patients. Student J Health Res Africa. 2023;4(12):7.https://doi.org/10.51168/sjhrafrica.v4i12.761
27. Bhatti TK, Kamani L. Fatty Liver Disease in Pakistan: An Underestimated Threat. World Gastroenterology Organisation e-WGN. 2024;28(2):18-20.https://www.worldgastroenterology.org/publications/e-wgn/e-wgn-expert-point-of-view-articles-collection/fatty-liver-disease-in-pakistan-an-underestimated-threat
28. Binet Q, Loumaye A, Hermans MP, Lanthier N. A Cross-sectional Real-life Study of the Prevalence, Severity, and Determinants of Metabolic Dysfunction-associated Fatty Liver Disease in Type 2 Diabetes Patients. J Clin Transl Hepatol. 2023;11(6):1377-1386. https://doi.org/10.14218/JCTH.2023.00117

29. Driessen S, Francque SM, Anker SD, et al. Metabolic dysfunction-associated steatotic liver disease and the heart. Hepatology(Baltimore, Md.), 82(2), 487–503.https://doi.org/10.1097/HEP.0000000000000735
30. Danpanichkul P, et al. The Rising Global Burden of MASLD and Other Metabolic Diseases (2000–2021). United European Gastroenterol J. 2025.https://doi.org/10.1002/ueg2.70072
31. Wongtrakul W, Charatcharoenwitthaya P. Global prevalence of advanced fibrosis in patients with type 2 diabetes mellitus: a systematic review and meta-analysis. BMC Gastroenterol. 2024;24(1):123.https://doi.org/10.1111/jgh.16666
32. Schwärzler J, Grabherr F, Grander C, et al. The pathophysiology of MASLD: an immunometabolic perspective. Expert Rev Clin Immunol. 2024;20(4):375-386.https://doi.org/10.1080/1744666X.2023.2294046