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Metabolic Associated Fatty Liver Disease in Type 2 Diabetes Mellitus and Correlation with Risk Factors

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Abstract

***Background:* "Metabolic Associated Fatty Liver Disease (MAFLD) previously Non-Alcoholic Fatty Liver Disease (NAFLD)" is an emerging and prevalent co-morbidity among Indian patients with type 2 diabetes mellitus (T2DM). However, data regarding the diagnostic utility of transient elastography (Liver Stiffness Measurement) in this population remain limited.**

***Objective:* This cross-sectional observational study aimed to determine the prevalence and severity of MAFLD in T2DM patients using Liver Stiffness Measurement by transient elastography and evaluate its association with metabolic parameters.**

***Methods:* A total of 135 adult T2DM patients underwent comprehensive clinical, anthropometric, and biochemical assessments. Hepatic evaluation was done using transient elastography (FibroScan) to quantify liver stiffness and steatosis. Metabolic dysfunction-associated steatohepatitis (MASH) was defined by the presence of elevated alanine aminotransferase (ALT) levels or evidence of hepatic fibrosis.**

***Results:* The prevalence of MAFLD was 67.4%, with a female predominance (62.2%), although no significant correlation with age or gender was observed. MAFLD was significantly associated with higher body mass index (BMI) ($p = 0.0365$) and presence of metabolic syndrome (74.5% vs. 25.5%, $p = 0.0001$). Patients with MAFLD exhibited elevated random plasma glucose, HbA1c, triglyceride levels, and lymphocyte counts compared to non-MAFLD counterparts. The Fatty Liver Index was significantly elevated in MAFLD patients, whereas liver function tests and fibrosis scores did not differ significantly between groups.**

***Conclusion:* This study demonstrates a high prevalence of MAFLD among T2DM patients, closely linked to metabolic syndrome, elevated triglycerides, and disrupted glucose metabolism. Non-invasive tools such as the Fatty Liver Index and liver stiffness measurement effectively identify individuals at risk.**

***Key words:* Non-Alcoholic Fatty Liver Disease (NAFLD), Metabolic Associated Fatty Liver Disease (MAFLD), Type 2 Diabetes Mellitus (T2DM), Fatty Liver Index (FLI), Liver Stiffness Measurement (LSM). Controlled Attenuation Parameter (CAP).**

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterised by persistent hyperglycaemia due to insulin resistance and/or relative insulin deficiency. With approximately 463 million adults affected globally and a projected increase to 578 million by 2030, T2DM is a growing public health concern[1]. Among its numerous complications, metabolic asociated fatty liver disease (MAFLD) has emerged as a highly prevalent hepatic manifestation, affecting up to 55.5% of individuals with T2DM[2]. The condition previously referred to as non-alcoholic fatty liver disease (NAFLD) has also been described as metabolic dysfunction – associated fatty liver disease (MAFLD), reflecting the same spectrum of disease with greater emphasis on underlying metabolic dysfunction[3].

MAFLD encompasses a spectrum ranging from simple steatosis to metabolic dysfunction associated steatohepatitis (MASH), which can progress to advanced liver fibrosis, cirrhosis, and hepatocellular carcinoma (HCC). MAFLD in patients with T2DM is often asymptomatic, yet it is significantly associated with increased morbidity and mortality. Liver fibrosis, in particular, is recognised as a key determinant of long-term outcomes in MAFLD patients. Liver fibrosis correlates with complications such as diabetic nephropathy, cardiovascular disease, neuropathy, and even certain malignancies[4,5].

Early identification and risk stratification of liver fibrosis in diabetic individuals is therefore critical for timely intervention and prevention of disease progression. Transient elastography, commonly known by the commercial name FibroScan®, is a non-invasive imaging technique that assesses liver stiffness as a surrogate for fibrosis. It has become a preferred tool for evaluating liver fibrosis in patients with MAFLD due to its ease of use, rapid results, and good diagnostic accuracy particularly for moderate to advanced fibrosis stages[6,7]. However, factors such as obesity, inflammation, and hepatic steatosis can

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influence measurement accuracy, and its effectiveness in detecting early fibrosis remains limited.

Despite the increasing burden of MAFLD among T2DM patients in India, data on the application and diagnostic utility of Liver Stiffness Measurement (LSM) in this population remain scanty. Given the high prevalence of metabolic risk factors such as obesity, dyslipidaemia, and hypertension, there is a pressing need for regional studies that assess the burden of MAFLD and explore its correlation with these risk factors in diabetic patients. This study aims to evaluate the prevalence and severity of MAFLD among individuals with type 2 diabetes mellitus using Liver Stiffness Measurement and to investigate the correlation between liver stiffness and metabolic risk factors such as glycaemic control, lipid profile, BMI, and duration of diabetes. This will help in identifying high-risk individuals who may benefit from early intervention, thus improving long-term outcomes in this vulnerable population.

Recent studies have highlighted the burden of MAFLD among diabetics. Wong *et al* (2023) reported a 70% prevalence of MAFLD in T2DM using MRI-based techniques[8], while Yilmaz *et al* (2022) demonstrated that MAFLD significantly increases cardiovascular risk independent of glycaemic status[9]. However, previous studies often lacked uniform diagnostic criteria or adequate stratification based on fibrosis severity. Most prior Indian studies have used ultrasound, whereas this study uses Liver Stiffness Measurement and Fatty Liver Index, giving a more accurate stratification.

This lacuna underscores the need for regional studies assessing MAFLD burden in diabetic cohorts, which formed the rationale for our study.

Material and Methods

This cross-sectional observational study was conducted over a period of one year following approval from the Institutional Ethics Committee (IEC). A convenient sample of 135 participants was enrolled after obtaining informed consent. Adults aged 18 years and above with T2DM were included. Exclusion criteria comprised individuals with significant alcohol intake (≥30 g/day in men and ≥20 g/day in women), pregnant women, those using steatogenic drugs such as corticosteroids, and patients with known chronic liver diseases including hepatitis B, hepatitis C, autoimmune hepatitis, haemochromatosis, Wilson's disease, primary biliary cirrhosis, or drug-induced hepatitis. All enrolled participants underwent detailed clinical evaluation, including medical history, physical examination, and anthropometric measurements such as body mass index (BMI) and waist circumference. Fasting venous blood samples were drawn, and measurements of aspartate transaminase (AST), alanine transaminase (ALT), serum bilirubin, lipid profile (consisting of low-density lipoprotein (LDL), high-density lipoprotein (HDL), serum triglycerides, and total cholesterol), and fasting blood sugar were conducted. Screening for hepatitis B surface antigen and anti-HCV antibodies was done to exclude viral hepatitis.

Obesity was defined as a BMI ≥25 kg/m^2 as per Asian Criteria and central obesity as waist circumference ≥90 cm in males and ≥80 cm in females, as per WHO standards. Hypertension was considered present if the patient was on antihypertensive treatment or had systolic blood pressure ≥140 mmHg or diastolic pressure ≥90 mmHg. Dyslipidaemia was defined either by prior diagnosis, use of lipid-lowering therapy, or the presence of abnormal lipid parameters total cholesterol >200 mg/dL, triglycerides >150 mg/dL, HDL-C <40 mg/dL, or LDL-C >130 mg/dL. The diagnosis of metabolic syndrome was based on IDF (International Diabetes Federation) criteria, requiring central obesity along with two or more of the following: elevated triglycerides, low HDL-C, raised blood pressure, or elevated fasting plasma glucose. ALT and AST were considered elevated at values >33 IU/L in males and >25 IU/L in females. as per AASLD practice guidelines[10]."

All patients underwent transient elastography (TE) using the FibroScan 530 Compact (Echosens, Paris, France), performed by a single experienced operator. Liver stiffness measurements (LSM) and controlled attenuation parameter (CAP) values were recorded. At least 10 valid LSMs were obtained per patient, with a required success rate >60% and interquartile range/median <30% to ensure measurement reliability. Based on established thresholds, significant fibrosis (F2), advanced fibrosis (F3), and cirrhosis (F4) were defined by LSM values of ≥7.0 kPa, ≥8.7 kPa, and ≥11.5 kPa, respectively. CAP values >233 dB/m indicated hepatic steatosis, with mild (234 - 269 dB/m), moderate (270 - 300 dB/m), and severe (>301 dB/m) steatosis (EASL clinical practice guidelines)[11] further categorised. MASH was defined as disease progression in MASLD patients who had elevated ALT levels (more than 6 months) or significant fibrosis[12]. The Fibrosis-4 (FIB-4) index was calculated using the formula: FIB-4 = (Age × AST)/(Platelet count × √ALT). The primary outcomes assessed were the proportion of diabetic patients with various grades of MAFLD and the correlation of liver stiffness with cardio-metabolic risk factors and the FIB-4 score. Statistical analysis was conducted using the SPSS version 26. Descriptive statistics were used to summarise the baseline characteristics of the study population. Associations between liver stiffness and clinical or biochemical variables including age, sex, BMI, duration of diabetes, dyslipidaemia, liver function tests, and FIB-4 index were analysed using univariate and multivariate regression models.

"APRI score was calculated as: "{AST (IU/L)/upper normal limit of AST (IU/L)}/platelet count (10^9/L)" "APRI was calculated as a validated non-invasive marker of fibrosis. An APRI >0.7 indicated significant fibrosis, ≥1.0 suggested progression to F3, and ≥2.0 indicated cirrhosis, based on a 2024 meta-analysis[13]". Fatty Liver Index (FLI) was calculated as a validated non-invasive marker of fibrosis and steatosis, which varies between 0 and 100. A score of FLI ≥60 was defined as having fatty liver, whereas FLI score <60 was inconclusive or non-fatty liver[14]. FIB-4 score <1.3 - rules-out advanced fibrosis, 1.3 - 2.67 ⇒ indeterminate zone, ≥2.67 ⇒ suggests advanced fibrosis[11].

Sample size calculation

Sample size estimation was performed using the OpenEpi, Version 3.0, open-source calculator (SSPropor). For a finite population size of 1,000,000 (N), with a hypothesized outcome frequency of 50% ± 8% (p), absolute precision of 8% (d), and a design effect (DEFF) of 1, the sample size was calculated using the formula: n = [DEE*Np(1-p)]/[(d2/Z21-≥ /2*(N-1)+p*(1-p)]. Considering the loss of data of 10%; adjusted sample size was 151 - 10% of (151) = 136. rounded off to 135.

Results

Among the 135 patients with T2DM, the prevalence of Metabolic Associated Fatty Liver Disease (MAFLD) was 67.4% (n = 91). The study cohort had female (62.2%) predominance as compared to males (37.8%), with a female-to-male ratio of 1.64:1. Participants were stratified into MAFLD (n = 91) and non-MAFLD (n = 44) groups. No statistically significant associations were identified between MAFLD and gender (p = 0.8144) as well as with age group (p = 0.3306). But the association of MAFLD was significant with BMI; indicating higher incidence of MAFLD with overweight and obese category (p = 0.0365) (Table I). Patients with MAFLD showed significantly higher liver stiffness values compared to non-MAFLD individuals (p <0.001) (Table II), Post-hoc analysis showed significant differences in LSM values between normal versus advanced fibrosis, normal versus cirrhosis, and significant fibrosis versus cirrhosis groups (p <0.05). The prevalence of metabolic syndrome was significantly higher in the MAFLD group (74.5%; n = 82) compared to the non-MAFLD group (25.5%; n = 28, p = 0.0001) (Table III).

Anthropometric and haemodynamic parameters reported no significant association between MAFLD and non-MAFLD groups (p >0.05). Among glycaemic parameters, MAFLD group had high random blood sugar (224.35 ± 69.47 mg/dL versus 181.64 ± 94.03 mg/dL, p = 0.0035) and HbA1c levels (9.58 ± 2.25 mmol/mol versus 7.78 ± 1.86 mmol/mol, p <0.0001). Regarding lipid profiles, triglyceride levels were significantly elevated in the MAFLD group (201.13 ± 92.28 mg/dL versus 169.48 ± 74.11 mg/dL, p = 0.0492), whereas no significant association was reported among total lipid, HDL, and LDL levels. Haematological parameters showed no significant association between MAFLD and non-MAFLD, except lymphocyte counts which were significantly higher in the MAFLD group (31.05 ± 6.95 versus 28.20 ± 8.45, p = 0.0396). Liver function test parameters did not reveal significant differences between the groups, including total bilirubin, direct bilirubin, SGOT, SGPT, ALP, total protein, serum albumin, and serum globulin levels. ALP levels were slightly higher in the MAFLD group (165.21 ± 56.85 IU/L versus 144.68 ± 59.99 IU/L, p = 0.055), though this did not reach statistical significance (Table IV).

Table I: Comparison of baseline characteristics of T2DM patients with respect to MAFLD.

Variables	MAFLD (N = 91)		Non-MAFLD (N = 44)		p-value
	N	%	N	%	
Gender					
Male	35	68.6	16	31.4	0.8144
Female	56	66.7	28	33.3	
Age Group					
35 - 49 years	44	65.7	23	34.3	0.3306
50 - 59 years	29	76.3	9	23.7	
≥60 years	18	60.0	12	40.0	
BMI					
18.5 - 24.9 (Kg/m^2)	32	56.1	25	43.9	**0.0365**
25.0 - 29.9 (Kg/m^2)	36	80.0	9	20.0	
≥30.0 (Kg/m^2)	23	69.7	10	30.3	

Table II: Liver stiffness measurement in T2DM patients with respect to MAFLD.

Liver stiffness measurement	MAFLD (N = 91)		Non-MAFLD (N = 44)		p-value
	N	%	N	%	
Normal (kPa)	23	42.6	31	57.4	**p <0.001**
Significant liver fibrosis (kPa)	18	58.1	13	41.9	
Advanced liver fibrosis (kPa)	29	100.0	0	0.0	
Cirrhosis (kPa)	21	100.0	0	0.0	
Comparison					**p-value**
Normal *versus* Significant fibrosis					(p >0.05)
Normal *versus* Advanced fibrosis					**<0.05**
Normal *versus* Cirrhosis					**<0.05**
Significant fibrosis *versus* Advanced fibrosis					**<0.05**
Significant fibrosis *versus* Cirrhosis					**<0.05**
Advanced fibrosis *versus* Cirrhosis					ns (p >0.05)

Table III: Association of metabolic syndrome in T2DM patients with respect to MAFLD.

Metabolic Syndrome	MAFLD (N = 91)		Non-MAFLD (N = 44)		p-value
	N	%	N	%	
Present	82	74.5	28	25.5	**0.0001**
Absent	9	36.0	16	64.0	

Table IV: Comparison of laboratory parameters in T2DM patients with respect to MAFLD.

	MAFLD (N = 91)	Non-MAFLD (N = 44)	p-value
Anthropometric parameters			
Waist Circumference (cm)	95.51 ± 12.55	92.65 ± 12.59	0.2172
Hip Circumference (cm)	100.11 ± 13.68	97.59 ± 15.83	0.3426
Blood pressure measurement			
Systolic blood pressure (mmHg)	133.03 ± 16.37	128.86 ± 14.58	0.1533
Diastolic blood pressure (mmHg)	81.11 ± 11.02	78.55 ± 9.70	0.1911
Glycaemic parameters			
Random Blood Sugar (mg/dL)	224.35 ± 69.47	181.64 ± 94.03	**0.0035**
HbA1c (mmol/mol)	9.58 ± 2.25	7.78 ± 1.86	**<0.0001**
Haematological parameters			
Haemoglobin (g/dL)	12.86 ± 2.11	13.18 ± 1.58	0.3742
Platelets (10^9/μL)	2.33 ± 0.88	2.24 ± 0.96	0.5897
RBC (Cells/μL)	4.98 ± 3.82	4.63 ± 0.55	0.5471
PCV (%)	39.74 ± 10.36	39.28 ± 4.16	0.7774
MCV (fl)	80.50 ± 11.18	82.25 ± 10.39	0.3848
TLC (cells/μL)	7306.02 ± 2121.80	7527.05 ± 2026.36	0.5659
Neutrophils (cells/μL)	62.10 ± 7.92	62.06 ± 12.78	0.9822
Lymphocytes (cells/μL)	31.05 ± 6.95	28.20 ± 8.45	**0.0396**
Lipid Profile			
Total lipid (mg/dL)	226.48 ± 101.03	194.68 ± 100.44	0.0882
HDL (mg/dL)	41.31 ± 11.26	40.75 ± 10.38	0.7817
LDL (mg/dL)	131.24 ± 43.68	124.86 ± 71.38	0.5226
Triglycerides (mg/dL)	201.13 ± 92.28	169.48 ± 74.11	**0.0492**
Liver function test			
Total bilirubin (mg/dL)	0.66 ± 0.12	0.8 ± 0.77	0.0917
Direct bilirubin (mg/dL)	0.24 ± 0.07	0.25 ± 0.16	0.6138
SGOT (IU/L)	31.42 ± 17.18	29.36 ± 20.21	0.5390
SGPT (IU/L)	34.63 ± 20.37	29.43 ± 17.18	0.1466
ALP (IU/L)	165.21 ± 56.85	144.68 ± 59.99	0.055
Total Protein (g/dL)	7.64 ± 0.63	7.68 ± 0.67	0.7354
Serum albumin (g/dL)	4.5 ± 0.56	4.44 ±0.54	0.5560
Serum Globulin (g/dL)	3.14 ±0.4	3.22 ± 0.42	0.2858

The Fib-4 score, the APRI score showed no significant difference between the MAFLD and non-MAFLD groups (p >0.05). However, the Fatty Liver Index (FLI) was significantly higher in the MAFLD group (62.8 ± 23.7) compared to the non-MAFLD group (48.8 ± 25.5, p = 0.0021) (Table V).

Table V: Fib 4 score, APRI score and fatty liver index in T2DM patients with respect to MAFLD.

Variables	MAFLD (N = 91)	Non-MAFLD (N = 44)	p-value
Fib 4 score	1.52 ± 1.37	1.40 ± 0.93	0.6005
APRI Score	0.65 ± 0.63	0.59 ± 0.48	0.5779
Fatty liver index (FLI)	62.8 ± 23.7	48.8 ± 25.5	**0.0021**

Discussion

Metabolic Associated Fatty Liver Disease (MAFLD) previously Non-alcoholic fatty liver disease is increasingly recognised as a major health concern, particularly among individuals with T2DM. This study highlights the intricate relationship between MAFLD and cardiometabolic risk factors, emphasizing the importance of early identification and management in diabetic patients. The incidence of MAFLD in the present study was found to be 67.4%. While Kosmalski *et al* reported a higher MAFLD incidence of 71%[15]. Studies by Choudhary *et al*, Agarwal *et al*, and Kalra *et al*, reported a lower incidence of MAFLD, at 55.7%, 57.2%, and 56.5%, respectively[16-18]. These variations in prevalence may be attributed to differences in study populations (e.g., sample size, demographics), diagnostic criteria (e.g., biopsy vs. imaging), and regional metabolic profiles (e.g., dietary habits, genetic predispositions). Nonetheless, the consistently high prevalence across these studies underscores the need for proactive screening for MAFLD in patients with T2DM.

Metabolic syndrome is a well-recognised risk factor for MAFLD, driven by shared pathophysiological mechanisms such as insulin resistance, obesity, and dyslipidaemia. In the present study, metabolic syndrome was significantly more prevalent in the MAFLD group (74.5%) compared to the non-MAFLD group (25.5%; p = 0.0002), reinforcing the strong association between these conditions. This finding is consistent with existing literature, which suggests that metabolic syndrome not only increases the risk of MAFLD but also contributes to disease progression, leading to fibrosis and potential liver-related complications. Insulin resistance, a hallmark of metabolic syndrome, promotes hepatic fat accumulation by increasing de novo lipogenesis and impairing lipid clearance, thereby exacerbating MAFLD severity. Additionally, obesity and dyslipidaemia further contribute to liver steatosis and inflammation, creating a pro-fibrotic environment. Our results are consistent with Agarwal *et*

al, who also reported the higher prevalence of metabolic syndrome in the MAFLD subgroup compared to those without MAFLD (61.9% vs. 13.2%, p <0.001). Despite no statistically significant differences in other anthropometric parameters (p >0.05), the MAFLD group exhibited a significantly higher mean BMI (27.3 ± 5.1 kg/m^2) compared to the non-MAFLD group (25.96 ± 5.24 kg/m^2; p = 0.0365), reinforcing obesity as a key contributing factor in MAFLD development. Our findings align with those of Choudhary *et al*, and Kosmalski *et al*, who also reported significant differences in anthropometric parameters between individuals with and without MAFLD[15,16].

Dyslipidaemia is a well-established metabolic abnormality in MAFLD, characterised by elevated triglycerides, increased LDL cholesterol, and reduced HDL cholesterol. These lipid disturbances contribute to hepatic fat accumulation, insulin resistance, and inflammation, all of which play a critical role in MAFLD pathogenesis. In our study, total lipid levels were elevated in the MAFLD group (226.48 ± 101.03 mg/dL) compared to the non-MAFLD group (194.68 ± 100.44 mg/dL; (p = 0.0882). While this suggests a trend toward lipid dysregulation in MAFLD patients, the lack of statistical significance may be attributed to inter-individual metabolic variability, dietary factors, or sample size limitations. Notably, triglyceride levels were significantly higher in the MAFLD group (201.13 ± 92.28 mg/dL versus 169.48 ± 74.11 mg/dL, p = 0.0492). This finding reinforces the strong association between hypertriglyceridaemia and MAFLD, as elevated triglycerides contribute to hepatic lipid accumulation, oxidative stress, and the progression of liver disease. Increased triglyceride levels are often a marker of insulin resistance, a key driver of MAFLD development. Additionally, LDL cholesterol levels were slightly elevated in the MAFLD group, but the difference was not statistically significant. While LDL is known to contribute to cardiovascular risk and liver fat deposition, its impact on MAFLD progression were less pronounced compared to triglycerides. Other metabolic factors, such as insulin resistance and inflammation, may play a more dominant role in the lipid dysregulation observed in MAFLD. Liver function tests (SGOT, SGPT, ALP) showed elevated levels in MAFLD patients; however, these differences did not reach statistical significance, suggesting that many cases in our cohort may represent early-stage MAFLD without substantial liver damage.

The FIB-4 and APRI scores are widely used non-invasive clinical tools for assessing liver fibrosis, particularly in MAFLD patients, as they help estimate fibrosis severity without the need for liver biopsy. These indices are calculated using routine biochemical and clinical parameters, making them practical for use in clinical settings. In our study, the mean FIB-4 and APRI scores were slightly higher in the MAFLD group compared to the non-MAFLD group; however, the differences did not reach statistical significance. While this suggests a possible trend toward increased fibrosis risk in MAFLD patients, the lack of statistical significance may be attributed to sample size limitations, inter-individual variability, or the early-stage nature of fibrosis in some patients. Liver Stiffness Measurement (LSM) and Fatty Liver Index (FLI) are valuable non-invasive tools for assessing liver fibrosis and hepatic steatosis, respectively. LSM, typically measured using elastography techniques, evaluates fibrosis severity by detecting liver stiffness, which increases as fibrosis progresses, making it particularly useful for identifying high-risk MAFLD patients. FLI, a metabolic score derived from clinical and biochemical parameters such as BMI, triglycerides, gamma-glutamyl transferase (GGT), and waist circumference, serves as an effective screening tool for hepatic steatosis, especially in high-risk populations like individuals with T2DM. Both markers offer a cost-effective, non-invasive approach to MAFLD assessment, reducing the need for liver biopsy and facilitating early diagnosis and management. Our study found a significant association between LSM and fibrosis severity in MAFLD patients, reinforcing its role as a reliable fibrosis assessment tool. This finding aligns with Esteban *et al,* who reported that T2DM patients with MAFLD had higher median liver stiffness (5.6 kPa [4.5 - 7.3]) compared to non-MAFLD individuals (4.8 kPa [4.2 - 5.8], p = 0.004), suggesting an increased risk of fibrosis progression in MAFLD patients, emphasizing the need for early detection and monitoring[19]. Similarly, FLI was significantly higher in the MAFLD group, with 77.8% of MAFLD patients having an FLI ≥60 compared to 60.5% in the non-MAFLD group (p = 0.0365), and the mean FLI was also significantly elevated in MAFLD individuals (62.8 ± 23.7 versus 48.8 ± 25.5, p = 0.0021).

Conclusion

This study confirms the high prevalence of MAFLD in T2DM patients and its strong association with metabolic syndrome, hypertriglyceridaemia, and impaired glucose metabolism. Non-invasive markers like the Fatty Liver Index and liver stiffness measurement proved useful in identifying at-risk individuals. Further research with larger, more diverse cohorts and longitudinal designs is needed to confirm these findings and explore the long-term implications of FLI in MAFLD progression and management.

Ethics statement and Consent

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee. Informed consent was obtained from the patients regarding the use of their clinical data. All the patient specific data was kept in strict confidence.

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