ORIGINAL RESEARCH

Liver Stiffness Measurement is Useful in Predicting Type 2 Diabetes Mellitus Among Nonalcohol Fatty Liver Disease Patients

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Background: Type 2 diabetes mellitus (T2DM) and non-alcoholic fatty liver disease (NAFLD) are closely related conditions.

Aim: This study investigated a group of individuals with NAFLD to evaluate if liver fibrosis, identified by FibroScan, correlated with T2DM.

Methods: 154 NAFLD patients obtained FibroScan, liver ultrasonography (US), and a thorough assessment of clinical implications and chemical biomarkers.

Results: In comparison to the NAFLD without T2DM group, the hemoglobin A1c(HBA1c)(mmol/mol%), homeostasis model of assessment for insulin resistance index (HOMA-IR), gamma-glutamyl transferase (GGT), fibrosis indices, and liver stiffness measurement (LSM) values were all considerably higher in the NAFLD with T2DM group. Patients with NAFLD and T2DM had considerably lower serum uric acid(SUA) levels than those with NAFLD alone.Those with severe fibrosis (79.3%, 23/29) in the NAFLD group showed a greater frequency of T2DM than those with mild fibrosis (45.6%, 21/46) or no fibrosis (27.85%, 22/79) (P=0.000). LSM value and elements of the metabolic syndrome (MetS) were independent risk factors for incident T2DM among NAFLD patients (OR=1.466, 95% CI [1.139-1.888], P=0.003; and OR=0.273, 95% CI [0.081-0.916], P=0.036).

Conclusion: FibroScan can identify significant fibrosis, which is independently linked to a higher prevalence of T2DM. As a result, it is crucial to make use of this technology to predict T2DM in NAFLD patients.

Keywords: liver stiffness measurement, NAFLD, T2DM, risk factors, early warning

Introduction

The main characteristic of non-alcoholic fatty liver disease (NAFLD) is excessive fat deposition in the liver. It has a close connection to metabolic syndrome (MetS), whose prevalence has increased as obesity has become more widespread. Type 2 diabetes mellitus (T2DM) and obesity are two metabolic risk factors that are associated with NAFLD, which is defined by 5% hepatocyte steatosis. The exclusion of other chronic liver disorders and excessive alcohol use (over 30 g/day for men and 20 g/day for women) are additional qualifiers.[1] In 2020, a global panel of experts categorized fatty liver as metabolic dysfunction-associated fatty liver disease (MAFLD), changing it from a negative exclusion diagnosis.

NAFLD is the most common chronic liver disease worldwide, whereas non-alcoholic steatohepatitis (NASH) is the more severe form of NAFLD that can lead to liver fibrosis and cirrhosis. T2DM is a recognized risk factor for the development of NASH in NAFLD. Thus, the development of T2DM may potentially be influenced by NAFLD and fibrosis.[2,3] It had been determined that patients with NAFLD and T2DM had an approximately 37% global prevalence of NASH and an approximately 17% global prevalence of advanced fibrosis, respectively.[4] NAFLD increases the probability of becoming T2DM.[5,6] The risk of acquiring T2DM at its early starting doubles in patients with NAFLD, despite the fact that NAFLD is present in more than 70% of patients

with T2DM.[2,7] The emergence of hyperglycemia and T2DM is the most clear consequence of NAFLD's association with extrahepatic insulin since insulin resistance (IR) and NAFLD are connected.[8] The risk of developing T2DM is higher in patients with NAFLD and extensive fibrosis.[4] Several pharmacological strategies have been evaluated in the treatment of NASH and NAFLD utilizing already available medications such as antidiabetic and anti-obesity medicines.[9] At different stages of clinical trials, a number of pharmacological treatments are presently being investigated, including FXR agonist (MET409), GLP-1 analogue (XW003), and GLP-1/glucagon agonist (DD01).[10] Physicians' interest in employing natural products as an alternative to conventional treatments for NAFLD has grown recently.[11] Given its potential benefits, further investigation and study should be done on this topic.

Currently, liver biopsy is the gold standard for diagnosing and staging liver fibrosis. This invasive technical operation, however, limits its clinical utility. Recently, a trustworthy non-invasive approach for identifying hepatic steatosis and fibrosis in patients with various stages of NAFLD has emerged: transient elastography with FibroScan, which gives controlled attenuation parameter (CAP) and liver stiffness measurement (LSM) parameters.[12,13] It is rare to examine the association between liver stiffness value and diabetes among individuals with NAFLD, despite the fact that hemoglobin A1c (HbA1c), the fasting blood glucose level(FBG), is straightforward and immediately useful in the diagnosis of diabetes.We sought to examine if FibroScan can evaluate the relationship between fibrosis and the incidence of T2DM in a group of individuals with NAFLD.

Materials and Methods
Patients
This observational cross-sectional study included adult inpatients having a diagnosis of NAFLD by Fibro-Scan screened from Department of Gastroenterology and Hepatology, Characteristic Medical Center of the Chinese People's Armed Police Force between January 2016 and January 2021. Patients with chronic liver diseases, such as steatosis-inducing drugs, viral B and C hepatitis, autoimmune hepatitis, Wilson's disease, genetic hemochromatosis, or alpha 1-antitrypsin deficiency, as well as excessive alcohol consumption (defined as 30 g/day for men and 20 g/day for women) were also excluded from the study. There were 154 inpatients participants in total in this research (Figure 1).

Clinical and Laboratory Assessments
Clinical, anthropometric, and laboratory data were acquired throughout the FibroScan evaluation. Based on the diagnostic criteria (from clinical history, or according to ADA criteria) for T2DM, patients were classified as either having NAFLD without T2DM or having NAFLD with T2DM. Low-density lipoprotein(LDL) - cholesterol>3.37mmol/L, triglycerides(TG)>1.7 mmol/L, total cholesterol(TC)>5.2mmol/L, high-density lipoprotein (HDL)-cholesterol<1.55 mmol/L, or use of lipid-lowering drugs were the local laboratory cut-offs used to identify dyslipidemia. The blood pressure threshold for hypertension was 140/90 mmHg.[14] Transaminase levels above 40 U/L for aspartate aminotransferase (AST) and 50 U/L for alanine aminotransferase(ALT), respectively, and gamma glutamyl transferase (GGT) readings above 60 U/L were considered abnormal. Smoking history and age at diagnosis were reported.

Liver Assessment
One professional physician evaluated the existence and severity of hepatic fibrosis and steatosis using the FibroScan 502. When a measurement failed, the expert physician used the M probe or the XL probe while being blinded to the subjects' clinical data.[15] The final values were obtained by the use of standardized procedures.[16] The existence of hepatic steatosis was detected using CAP value of 248 obtained by the M probe and described in the literature (severe steatosis is defined as 280 dB/m).[17] While the LSM value was used to assess the level of fibrosis (F0, ≤5.9 kPa), mild fibrosis (F1,6–6.9kPa; F2,7–9kPa); and severe fibrosis (F3,9.1–10.3kPa; F4,≥10.4kPa), respectively.[18,19]

Vascular Complications and T2DM Clinical Status
For all patients, information was collected on the development of macrovascular disorders like coronary artery disease, hypertension, the MetS, and T2DM whether or not they had microangiopathy.
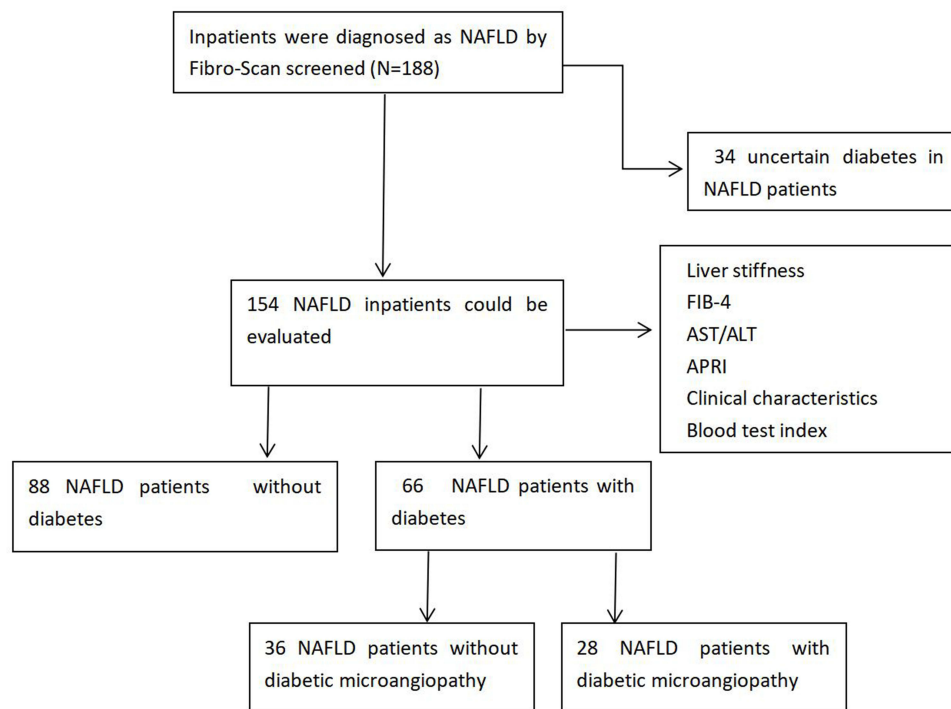
Figure 1 Flowchart of study population enrollment.

Statistical Analysis

The chi-squared test or Fisher's exact test was used to express categorical variables as absolute and relative frequencies (n,%) and continuous variables as means standard deviation (SD) or medians (range values). To compare the incidence of T2DM among the various NAFLD severity levels, chi-square segmentation was employed. For continuously distributed variables with normal and non-normal distributions, respectively, the unpaired Student's t-test and Mann–Whitney U-test were applied. In order to investigate the risk factors connected to T2DM, multivariate logistic correlation analysis was employed. SPSS for Windows (version 18.0; Chicago, Illinois, USA) was used for data analysis and quality assurance.

Results

Demographic and Clinical Complications Characteristics

The participants' mean age was 51.27 ± 12.16 years, as indicated in Table 1. Among them, 59.7% patients (92/154) were male. A total of 71.0% patients (88/124) had MetS, 65.3% patients (96/147) had dyslipidemia and 38.5% patients (45/117) had hypertension. Coronary atherosclerotic heart disease (CHD) was present in 22.2% (26/117) of patients. Of these, 29.9% (35/117) were current smokers. In the NAFLD cohort, 66 patients (42.86%) had NAFLD and T2DM. The prevalence of MetS, Hypertension, CHD was higher in the T2DM group than in the group without T2DM (80.0% (48/60) vs 62.5% (40/64), P=0.032; 51.9% (28/54)vs 27%(17/63), P=0.006;33.3% (18/54) vs 12.7% (8/63), P=0.007). The age of NAFLD with T2DM patients was 55.71 ± 10.62 years old which was older than that of NAFLD without T2DM patients (P=0.000). The Systolic blood pressures (SBP) of NAFLD with T2DM patients were 131.22 ± 14.71 mmHg, higher than that in the group of NAFLD without T2DM patients (124.37 ± 14.49 mmHg) (P=0.013)(Table 1).

Chemical Characteristics and LSM Values or Fibrosis Indices

The homeostasis model of assessment for insulin resistance index (HOMA-IR) and HbA1c values between NAFLD patients with and without T2DM were significantly different (HOMA-IR: median 6.76 (2,26) vs 4.37 (2,12), P=0.019; HbA1c: $8.25\pm1.66\%$ vs $5.65\pm1.97\%$, P=0.000). Similar results were seen at all GGT levels. P=0.017 (55 μmol/L (6,1385

Table I Demographic Data of NAFLD Patients Without T2DM and NAFLD with T2DM Patients

Characteristics	All NAFLD Patients (n=154)	NAFLD Without T2DM (n=88)	NAFLD with T2DM (n=66)	P value
Age(y)	51.27±12.16	47.93±12.23	55.71±10.62	P=0.000
Male, N(%)	92(59.7)	58(65.9)	34(51.5)	P=0.071
BMI	27.22(20.98,36.75) (41)	27.43(23.89,36.05) (22)	26.85(20.98,36.75) (19)	P=0.433
MetS n(%)	71.0%(88/124)	62.5%(40/64)	80.0%(48/60)	P=0.032
Dyslipidemia(%)	65.3%(96/147)	67.9%(57/84)	61.9%(39/63)	P=0.453
Current smoking(%)	29.9%(35/117)	25.4%(16/63)	35.2%(19/54)	P=0.249
Drinking(%)	27.6%(32/116)	25.8%(16/62)	29.6%(16/54)	P=0.646
Hypertension(%)	38.5%(45/117)	27%(17/63)	51.9%(28/54)	P=0.006
SBP	127.53±14.95 (117)	124.37±14.49 (63)	131.22±14.71 (54)	P=0.013
DBP	81.03±9.147 (117)	81.59±9.52 (63)	80.39±8.74 (54)	P=0.482
CHD(%)	22.2%(26/117)	12.7%(8/63)	33.3%(18/54)	P=0.007
Cerebrovascular disease (%)	11.1%(13/117)	6.3%(4/63)	16.7%(9/54)	P=0.070

Abbreviations: T2DM, type 2 diabetes mellitus; MetS, Metabolic syndrome; SBP, Systolic blood pressures; DBP, Diastolic blood pressure; CHD, Coronary atherosclerotic heart disease; Drinking (defined < 30 g/day for men and< 20 g/day for women).

μmol/L) vs 40.5 μmol/L (11,711 μmol/L). In comparison to NAFLD patients without T2DM, individuals with T2DM had reduced serum uric acid (SUA) levels (324.32±90.94 mol/L vs 382.69±98.84 mol/L, P=0.004). The LSM by FibroScan (10.37±10.26 vs 5.73±2.58, P=0.000), AST to platelet ratio index (APRI) (P=0.044), and Fibrosis-4 score (FIB-4) (P=0.001) were higher in the NAFLD with T2DM group than the NAFLD without T2DM individuals. There were no discernible differences in ALT and AST levels, TG, TC, LDL, and HDL levels between NAFLD patients with and without T2DM. Hepatic steatosis (CAP value) between the two groups did not differ significantly (Table 2).

The Correlation Between the Degree of Fibrosis and the Frequency of T2DM in NAFLD Patients

Non fibrosis was seen in 79 (51.3%) of the NAFLD patients at enrollment (F0,≤LSM 5.9 kPa), mild fibrosis was present in 46 (29.9%) of the patients (F1,6–6.9kPa; F2,7–9kPa), and severe fibrosis was present in 29 instances (18.83%) of the

Table 2 Laboratory Variables of NAFLD Without and with T2DM in NAFLD Patients

Characteristics	All NAFLD Patients (n=154)	NAFLD without T2DM Patients (n=88)	NAFLD with T2DM Patients (n=66)	P value
Peripheral mononuclear cells	0.466±0.169	0.467±0.166	0.466±0.175	P=0.957
PLT count (I×10⁹/L)	230.67±82.64	239.28±76.33	219.18±89.68	P=0.136
HBA1c(mmol/mol;%)	7.891±1.789 (44)	5.65±1.97 (6)	8.250±1.66 (38)	P=0.000
HOMA-IR	5.44(2,26) (35)	4.37(2,12) (18)	6.76 (2,26) (17)	P=0.019
AST(U/L)	32.62±21.88	30.09±18.68	36.00±25.30	P=0.097
ALT(U/L)	50.58±42.52	46.69±33.98	55.77±51.61	P=0.191

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Table 2 (Continued).

Characteristics	All NAFLD Patients (n=154)	NAFLD without T2DM Patients (n=88)	NAFLD with T2DM Patients (n=66)	P value
GGT(U/L)	43.0 (6,1385)	40.5 (11,711)	55 (6,1385)	P=0.017
TBil (mg/dL)	16.43±8.22	17.37±8.15	15.17±8.21	P=0.100
DBil (mg/dL)	5.11±3.05	5.01±2.45	5.24±3.72	P=0.636
SUA(mol/L)	357.44±99.49 (141)	382.69±98.84 (80)	324.32±90.94 (61)	P=0.004
Flns (U/L)	21.57±12.51 (42)	20.55±10.19 (22)	22.71±14.84 (20)	P=0.583
TC(mmol/L)	5.11±0.99 (140)	5.17±0.88 (80)	5.03±1.11 (60)	P=0.401
TG(mmol/L)	2.54±1.92 (140)	2.57±1.86 (80)	2.49±2.02 (60)	P=0.794
HDL(mmol/L)	1.10±0.56 (138)	1.14±0.70 (78)	1.04±0.30 (60)	P=0.301
LDL(mmol/L)	3.02±0.95 (138)	3.06±0.92 (78)	2.97±0.99 (60)	P=0.558
CAP	306.13±34.30	306.34±36.42	305.85±31.52	P=0.930
AST/ALT ratio	0.732(0.093, 2.73)	0.718 (0.096, 2.73)	0.748 (0.26, 2.32)	P=0.664
APRI	0.42(0.083, 6.932)	0.370 (0.089,6.93)	0.530 (0.083,2.658)	P=0.044
FIB-4	1.02(0.130,12.07)	0.905 (0.130,10.18)	1.330 (0.378,12.07)	P=0.001
LSM	7.71±7.34	5.73±2.58	10.37±10.26	P=0.000

Abbreviations: PLT, Platelet; HBA1c, hemoglobin A1c; HOMA-IR, homeostasis model of assessment for insulin resistance index; ALT, Alanine aminotransferase; AST, Aspartate Transaminase; TBL, total bilirubin; GGT, Gamma glutamyl transpeptidase; HDL, High-density lipoprotein cholesterol; TC, Total cholesterol; LDL, Low-density lipoprotein cholesterol; TG, Triglyceride; APRI, AST to platelet ratio index; FIB-4, Fibrosis-4 score; SUA, serum uric acid; CAP, Controlled attenuation parameter; LSM, Liver stiffness measurement; Flns, fasting insulin.

patients (F3,9.1–10.3kPa; F4,≥10.4kPa) (Table 3). Greater LSM values were also associated with a significantly greater incidence of T2DM. Patients with severe fibrosis (23/29) exhibited a higher prevalence of T2DM compared to patients without fibrosis (22/79) (79.3% vs 27.8%, χ^2=23.11, P<0.0001) and patients with mild fibrosis (21/46) (79.3% vs 45.7%, χ^2=8.31, P<0.0001). In addition, we found that the frequency of T2DM events varied significantly between the groups with mild and non fibrosis NAFLD (45.7% vs 27.8%, χ2=4.084, P=0.043). (Figure 2).

Table 3 Distribution of Patients According to the Stage of Liver Fibrosis

Stage of Fibrosis	No(%)
F0(0–5.9)	79(51.3)
F1(6–6.9)	22(14.3)
F2(7–9)	24(15.6)
F3(9.1–10.3)	6(3.9)
F4(≥10.4)	23(14.9)
Total	154(100)
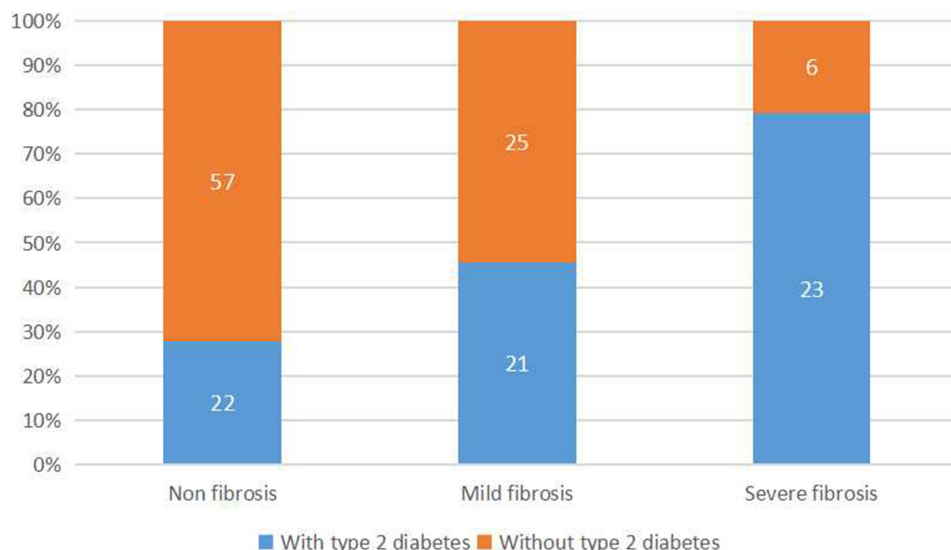
Figure 2 Fibrosis level and the rate of type 2 diabetes in NAFLD patients.

Regression Analysis for Presence of T2DM in NAFLD

We evaluated the association between T2DM in NAFLD patients and each clinical consequence after controlling for recognized risk variables and new possible confounders. We discovered that the existence of T2DM in NAFLD patients was still substantially correlated with age, CHD, hypertension, MetS, SBP, LSM, GGT, and SUA levels (P<0.01). Also shown in Table 4 was an independent relationship between the prevalence of T2DM in NAFLD patients and LSM value (OR 1.466, 95% CI 1.4–1.888, P=0.003) and MetS (OR 0.273, 95% CI 0.081–0.916, P=0.036).

Table 4 Multivariate Analysis of Factors Associated with Diabetes in NAFLD Patients

Variables	Univariate Analysis			Multivariate Analysis		
	OR	95% CI	P value	OR	95% CI	P value
Age(ys)	1.061	(1.029 1.094)	0.000	1.018	(0.959 1.082)	0.554
Sex	0.550	(0.286 1.057)	0.073			
ALT(U/L)	1.005	(0.997 1.013)	0.196			
AST(U/L)	1.013	(0.997 1.029)	0.108			
GGT(U/L)	1.005	(1.000 1.009)	0.045	1.002	(0.997 1.007)	0.510
TBiL(mg/dL)	0.965	(0.925 1.007)	0.105			
SUA(mol/L)	0.993	(0.989 0.997)	0.001	0.995	(0.989 1.001)	0.095
TC(mmol/L)	0.862	(0.612 1.216)	0.398			
TG(mmol/L)	0.977	(0.818 1.165)	0.792			
HDL(mmol/L)	0.619	(0.227 1.689)	0.349			

(*Continued*)

Table 4 (Continued).

Variables	Univariate Analysis			Multivariate Analysis		
	OR	95% CI	P value	OR	95% CI	P value
LDL(mmol/L)	0.898	(0.627 1.286)	0.556			
APRI	1.188	(0.758 1.863)	0.452			
FIB4	1.226	(0.982 1.531)	0.072			
BMI	0.929	(0.786 1.099)	0.393			
AST/ALT	1.302	(0.518 3.273)	0.574			
CAP	1.000	(0.990 1.009)	0.930			
LSM	1.271	(1.125 1.435)	0.000	1.466	(1.139 1.888)	0.003
Hypertension	2.914	(1.348 6.300)	0.007	0.567	(0.176 1.830)	0.343
CHD	3.437	(1.352 8.737)	0.009	0.459	(0.129 2.341)	0.418
MetS	2.400	(1.068 5.395)	0.034	0.273	(0.081 0.916)	0.036

Abbreviations: PLT, Platelet; HBA1c, hemoglobin A1c; HOMA-IR, homeostasis model of assessment for insulin resistance index; ALT Alanine aminotransferase; AST Aspartate Transaminase; TBL total bilirubin; GGT Gamma glutamyl transpeptidase; HDL High-density lipoprotein cholesterol; TC Total cholesterol; LDL Low-density lipoprotein cholesterol; TG, Triglyceride; APRI, AST to platelet ratio index; FIB-4, Fibrosis-4 score; SUA, serum uric acid; CAP, Controlled attenuation parameter; LSM, Liver stiffness measurement; CHD, coronary atherosclerotic heart disease; MetS, metabolic syndrome.

Discussion

According to previous epidemiological reports,[20] NAFLD is currently regarded as the most common cause of chronic liver disease in Western countries, with prevalence rates in the general population ranging from 13.5% in Africa to 31.8% in the Middle East, 29.62% in Asia, and 29.81% on the Chinese mainland.[21,22] According to recent studies, NAFLD affects several organ systems, including the hepatic, renal, cardiovascular, and cerebrovascular systems.[23] NAFLD has also been connected to a number of extra-hepatic tumors.[24] Most recently, bladder cancer-one of the most deadly diseases for the elderly-has been found associated to NAFLD and insulin resistance(IR).[25] In the most recent guidelines for patients with NAFLD, T2DM screening is frequently been recommended.[1,26] The risk of developing T2DM in NAFLD patients was thus examined in our study utilizing clinical laboratory tests, FibroScan, and other techniques.

In terms of fundamental clinical characteristics, we discovered that individuals with NAFLD and T2DM had considerably higher incidence rates of the MetS, hypertension, and CHD. Additionally, they were older and had greater SBP than the participants. When metabolic markers are tested, NAFLD and T2DM patients had lower SUA levels and higher HOMA-IR. These results imply that metabolic variables may be important in explaining the association between liver fat content and T2DM prevalence. A key role for IR and visceral fat is played in the pathophysiology of CHD, T2DM, and NAFLD.[27] Patients with NAFLD and T2DM had considerably higher GGT levels than those without T2DM. One of the important enzymes involved in the metabolism of glutathione and cysteine, GGT is strongly linked to central obesity,[28] diabetes,[29] and cardiovascular[30] risk. These findings imply that in individuals with NAFLD, GGT may act as a critical diagnostic marker for T2DM. According to the findings, those with T2DM had greater levels of the fibrosis index, APRI, FIB-4, and LSM when it evolved to liver fibrosis. The incidence of T2DM in individuals with NAFLD also increased in a stepwise fashion with increasing NAFLD fibrosis severity, which is interesting. It was seemingly shown that liver fibrosis and MetS were independent risk factors for the development of NAFLD to T2DM.

In this study, the high rate of T2DM accompany with the high prevalence of increased liver stiffness in NAFLD patients, prompts physicians to consider IR and inflammation disorder as an important comorbidity that could impact on NAFLD and T2DM.[27] Therefore, it is absolutely not possible to define on this basis a cause-effect relationship between measurement of liver stiffness and risk of T2DM.All we can conclude is that, while evaluating NAFLD, individuals with greater liver stiffness values require special attention to the identification of T2DM diagnostic markers and ongoing

awareness about the potential onset of T2DM.Can NAFLD fibrosis serve as a trigger for type 2 diabetes?It is necessary to do more prospective studies that support our findings.

While, recent research has revealed that fatty liver fibrosis and T2DM are related.[31] Mantovani et al found that the magnitude of the risk of incident T2DM mirrored the severity of NAFLD, especially the severity of liver fibrosis, which was consistent with our results[32] T2DM and NASH both develop and progress as a result of insulin resistance. NASH was present in 37.3% of those with T2DM worldwide.[4] Some worldwide professionals renamed NAFLD to MAFLD in 2020[33] due to the significant correlation between NAFLD and T2DM.

This recommends that patients with NAFLD, especially those who have clinical indications of MetS, should have liver fibrosis screenings. A clinical experiment with a significant sample size found that the severity of NAFLD may influence the probability of becoming T2DM in the future, and conversely, its resolution may lower that risk of development diabetes.[34] Transient elastography (TE) is a simple, reproducible, and noninvasive tool used to measure liver steatosis by CAP value and measure liver fibrosis by LSM. Liver fibrosis may be the primary risk factor for T2DM. TE screening has important clinical significance as a NAFLD monitoring method, and LSM can be used to dynamically monitor the grade of fibrosis in relation to IR and the emergence of T2DM during follow-up.

It is important to recognize the limitations of this study. First, the research sample may be biased toward older participants due to its near 60% male composition and average age of 51 years. Additionally, the sample may not be sufficiently balanced or have a high clinical comorbidity status when participants who are eligible for admission to tertiary hospitals are included. Consequently, the individuals in the sample frequently have higher rates of T2DM than the general population.[35] Second, The association between LSM values and T2DM-related biomarkers was examined in this study using a cross-sectional design. Additionally, the degree of liver fibrosis as assessed by FibroScan assessment was used to predict the occurrence of T2DM, and follow-up data on the dynamic changes of LSM value and T2DM are absent.Thirdly, the individuals were enrolled in a single study center, and the sample size was small. To verify our findings, multicenter controlled and randomized investigations are required. In addition, a large sample cohort with a well-balanced range of ages and clinical comorbidity status can be used to design this study in a longitudinal prospective way.

Abbreviations

NAFLD, Non-alcoholic fatty liver disease; NASH, Non-alcoholic steatohepatitis; T2DM, Type 2 diabetes mellitus; BMI Body mass index; DBP, Diastolic blood pressure; SBP, Systolic blood pressure; ALT, Alanine aminotransferase; AST, Aspartate Transaminase; TBL, total bilirubin; GGT, Gamma glutamyl transpeptidase; HDL, High-density lipoprotein cholesterol; TC, Total cholesterol; LDL, Low-density lipoprotein cholesterol; TG, Triglyceride; HbA1c, hemoglobin A1c; HOMA-IR, homeostasis model of assessment for insulin resistance index; APRI, AST to platelet ratio index; FIB-4, Fibrosis-4 score; CAP, Controlled attenuation parameter; LSM, Liver stiffness measurement.

Ethics Declarations

All patients provided written informed permission to participate in the trial, which followed to the 1975 Helsinki Declaration and was approved by the Characteristic Medical Center of the Chinese People's Armed Police Force ethics committee.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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