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Association between FIB-4 index and lower extremity arterial disease in MASLD patients: a cross-sectional study

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Abstract

Background Metabolic dysfunction-associated steatotic liver disease (MASLD) is associated with an elevated risk of cardiovascular conditions, such as lower extremity arterial disease (LEAD). The Fibrosis-4 (FIB-4) index, a non-invasive marker of liver fibrosis, may have predictive value for LEAD in patients with MASLD. This study aimed to explore the association between FIB-4 and LEAD in a cohort of patients with MASLD.

Methods This cross-sectional study included 481 participants with MASLD, selected from a comprehensive health check-up database. Participants were categorized into three groups based on their FIB-4 index (< 1.3, 1.3–2.66, > 2.66) and underwent duplex ultrasonography to diagnose LEAD. Logistic regression models were employed to evaluate the association between FIB-4 and LEAD, adjusting for demographic, metabolic, and lipid-related factors. Subgroup analyses were performed by sex, age, diabetes mellitus status, hypertension, dyslipidemia, smoking status.

Results The prevalence of LEAD increased with FIB-4 levels, from 51.3% in the low FIB-4 group to 86.5% in the high FIB-4 group ($p < 0.001$). In fully adjusted models, higher FIB-4 levels were significantly associated with LEAD (adjusted odds ratio [OR]: 3.54, 95% confidence interval [CI]: 1.39–9.01) in the high FIB-4 group compared to the low group. As a continuous variable, each unit increase in FIB-4 was associated with a 66% higher likelihood of LEAD (adjusted OR: 1.66, 95% CI: 1.12–2.26, $P < 0.001$). Subgroup analyses did not reveal significant interactions (P for interaction > 0.05).

Conclusions Higher FIB-4 levels are independently associated with the prevalence of LEAD in MASLD patients, although subgroup analyses did not reveal significant interactions. This suggests that further studies with larger sample sizes are needed to explore these relationships more comprehensively.

Keywords Fibrosis-4 index, MASLD, Lower extremity arterial disease, Liver fibrosis, Type 2 diabetes mellitus, Cross-section study

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Introduction

Lower extremity arterial disease (LEAD) is a significant manifestation of systemic atherosclerosis, which remains a leading cause of cardiovascular morbidity and mortality globally. The condition is often associated with other forms of cardiovascular disease (CVD), contributing to increased rates of amputations, functional disability, and decreased quality of life [1]. LEAD is closely linked to the progression of atherosclerosis, and its association with both traditional and emerging risk factors is a key area of clinical investigation. Despite the well-documented clinical significance of LEAD, its underlying pathophysiological mechanisms in patients with metabolic dysfunction-associated steatotic liver disease (MASLD) have not been fully elucidated [2, 3].

MASLD, a newly defined entity characterized by excessive fat accumulation in the liver, is associated with a high risk of metabolic disturbances and cardiovascular complications [4]. A shift toward recognizing the broader systemic effects of MASLD has become evident, with evidence suggesting its link to various forms of atherosclerotic disease, including LEAD [5–7]. MASLD affects over a quarter of the global population, with its prevalence continually rising due to increasing rates of obesity, diabetes, and metabolic syndrome [8, 9]. In China, the prevalence of MASLD has been reported to be approximately 36.69% in the general population, with over 35% of individuals exhibiting some degree of liver fibrosis [10, 11]. Studies have also shown that the prevalence of advanced fibrosis in this population is significant, with rates reaching 7.53% for F2 fibrosis and 2.55% for F3 fibrosis [11]. Patients with MASLD are often at increased risk of developing vascular complications such as coronary artery disease, cerebrovascular events, and peripheral arterial diseases, which include LEAD [3, 12]. The disease is marked by a complex interplay of insulin resistance, adiposity, oxidative stress, and inflammation, all of which contribute to vascular damage [13].

Liver fibrosis, a hallmark feature of advanced MASLD, plays a crucial role in linking hepatic abnormalities to extrahepatic vascular outcomes. The degree of fibrosis has been shown to correlate with the severity of CVD, including atherosclerosis and LEAD [14–16]. Studies have demonstrated that liver fibrosis may directly contribute to vascular endothelial dysfunction, arterial stiffness, and the promotion of systemic inflammation [17, 18]. Given this, the need for reliable biomarkers to assess liver fibrosis in the context of vascular risk remains paramount. The Fibrosis-4 (FIB-4) index, a widely utilized non-invasive tool for assessing liver fibrosis, has been shown to provide valuable prognostic information not only in liver diseases but also in extrahepatic complications such as CVD and atherosclerosis [19]. FIB-4 has been associated with an increased risk of coronary artery disease, carotid atherosclerosis, and peripheral vascular disease in diverse populations, further supporting its potential as a predictor of vascular complications in MASLD [14]. However, the relationship between FIB-4 and LEAD in the MASLD population remains underexplored, highlighting a gap in the current understanding of the role of liver fibrosis in peripheral arterial disease.

This study aims to address this gap by investigating the association between FIB-4 and LEAD in a cohort of patients with MASLD, using data derived from a single center health check-up database. By examining the relationship between FIB-4 and LEAD both as a continuous and categorical variable, we aim to provide deeper insights into the utility of FIB-4 for identifying patients at risk for LEAD. Subgroup analyses based on demographic factors such as sex, age, diabetes status, and other cardiovascular risk factors will help elucidate the broader applicability of FIB-4 in assessing vascular risks in MASLD patients.

Methods

Study design

This cross-sectional study was conducted at Ningbo Medical Center LiHuiLi Hospital from January 2022 to December 2024. Participants were selected from individuals who underwent routine health check-ups during this period. The aim of this study was to explore the association between the FIB-4 index and LEAD in patients with MASLD.

Patient selection

Out of 2,970 individuals screened, 627 were diagnosed with steatotic liver disease (SLD) by abdominal ultrasound using standard criteria: the presence of hepatic steatosis was confirmed if the liver parenchyma demonstrated increased echogenicity compared to the renal cortex, and a clear distinction between the liver and diaphragm was not observed [20]. Additionally, participants had to meet at least one cardiometabolic risk factor(CMRF) criterion [4]: BMI ≥ 23 kg/m^2; Fasting plasma glucose (FPG) ≥ 5.6 mmol/L or HbA1c ≥ 5.7%, diagnosed with T2DM; Blood pressure ≥ 130/85 mmHg; Triglycerides (TG) ≥ 1.70 mmol/L; HDL-C < 1.0 mmol/L for males, < 1.3 mmol/L for females. After applying exclusion criteria including absence of duplex ultrasonography, exceeded alcohol consumption(> 140 g/week for females, > 210 g/week for males) [4], hepatitis B infection, ages under 18 or over 80 years, or missing FIB-4 data), 481 participants were diagosed with MASLD and included in the final analysis.

Definition of LEAD

LEAD was diagnosed using duplex ultrasonography to assess the femoral, popliteal, anterior tibial, posterior

tibial, and dorsalis pedis arteries. LEAD was defined by the presence of arterial stenosis ≥ 50% or occlusion, as indicated by abnormal Doppler waveforms, peak systolic velocity ratios, or visible atheromatous plaques [21, 22].

Data collection

Data collected during routine medical check-ups included demographic information (age, sex, body mass index [BMI]), clinical history (hypertension, T2DM, preT2DM, smoking), and laboratory test results. Hypertension was determined by an SBP ≥ 140 mmHg or DBP ≥ 90 mmHg, or the present use of antihypertensive medication [23].T2DM was diagnosed based on fasting plasma glucose ≥ 126 mg/dL, 2-hour plasma glucose ≥ 200 mg/dL during an oral glucose tolerance test, or the use of diabetes medication [24]. **PreT2DM** was defined as fasting plasma glucose of 100–125 mg/dL or 2-hour plasma glucose of 140–199 mg/dL during an oral glucose tolerance test [24]. Dyslipidemia was defined as TC ≥ 5.18 mmol/L, LDL-C ≥ 3.37 mmol/L, or TG ≥ 1.7 mmol/L, or current use of lipid-lowering medications [25]. Smoking status was classified as current, former, or never smoker.

Fasting blood samples underwent analysis for liver and lipid profiles, which encompassed total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and lipoprotein(a) [Lp(a)]. The formula was used to calculate the FIB-4 index [26]:

$$FIB-4=\frac{Age\ (years)\times AST\ (U/L)}{Platelet\ count\ (10^9/L)\times\sqrt{ALT\ (U/L)}}.$$

Based on established thresholds, participants were stratified into three risk categories according to FIB-4 values: low liver fibrosis risk (< 1.3), intermediate liver fibrosis risk (1.3–2.66), and high liver fibrosis risk (> 2.66) [27]. These categories are widely accepted for estimating the likelihood of significant liver fibrosis, with higher FIB-4 levels indicating an increased probability of advanced fibrosis or cirrhosis.

Statistical analysis

Continuous variables were described using mean ± standard deviation (SD) or median (interquartile range [IQR]), and categorical variables were represented by counts and percentages. Between group comparisons were performed using chi-square tests for categorical variables and ANOVA or Kruskal-Wallis tests for continuous variables. Multivariate logistic regression was used to assess the association between FIB-4 and LEAD, adjusting for sex, age, BMI, hypertension, T2DM, smoking status, TC, TG, HDL-C, LDL-C, and Lp(a). Subgroup analyses were conducted to evaluate potential effect modifications by sex, age, T2DM, and other factors. Interaction terms were assessed using likelihood ratio tests. Additionally, a sensitivity analysis was performed to assess the association between FIB-4 and LEAD in the metabolic dysfunctionassociated fatty liver disease (MAFLD) population [5], using the same statistical model and covariates. Statistical significance was defined as $P < 0.05$, and all analyses were performed using Free Statistics software version 1.9, which incorporates the R statistical software version 4.3.2 (https://www.R-project.org, R Foundation).

Results

Participant selection and baseline characteristics

Based on the participant selection flow shown in Fig. 1, a total of 481 MASLD patients were included for subsequent analysis. These participants were stratified into three groups based on FIB-4 index: <1.3 (low risk), 1.3–2.66 (intermediate risk), and > 2.66 (high risk). Significant differences in demographic and clinical characteristics were observed among the groups (Table 1).

Participants with higher FIB-4 levels were older, with mean ages of 50.8 ± 11.2, 64.9 ± 9.2, and 66.1 ± 11.7 years for the low, intermediate, and high FIB-4 groups, respectively ($P < 0.001$). The prevalence of LEAD increased across the groups, from 61.1% in the low FIB-4 group to 86.5% in the high FIB-4 group ($P < 0.001$). Male participants were more frequent in the low (57.5%) and high (53.8%) FIB-4 groups compared to the intermediate group (42.9%) ($P = 0.009$). Lipid profiles also differed among groups: LDL-C levels were lower in the intermediate group compared to the low and high groups ($P = 0.009$), and triglyceride levels showed significant variability ($P = 0.004$).

Associations between FIB-4 and LEAD

Logistic regression analyses showed that higher FIB-4 levels, whether evaluated as continuous or categorical variables, were associated with the prevalence of LEAD (Table 2). As a continuous variable, each unit increase in FIB-4 was associated with 66% higher odds of LEAD in the fully adjusted model for MASLD (adjusted odds ratio (OR): 1.66, 95% confidence interval (CI): 1.21–2.26, $P = 0.001$), and 38% higher odds for MAFLD (adjusted OR: 1.38, 95% CI: 1.12–1.69, $P = 0.002$) (Table 2 and Table S1).

When analyzed as a categorical variable, participants in the high FIB-4 group (> 2.66) had significantly higher odds of LEAD compared to the low FIB-4 group (< 1.3). In the unadjusted model for MASLD, the OR was 6.1 (95% CI: 2.64–14.09, $P < 0.001$), and 4.97 (95% CI: 2.43–10.18, $P < 0.001$) for MAFLD (Table 2 and Table S1). After adjusting for sex, age, BMI, and metabolic factors (Model 3), the association remained significant for MASLD (adjusted OR: 3.54, 95% CI: 1.39–9.01, $P < 0.001$) and

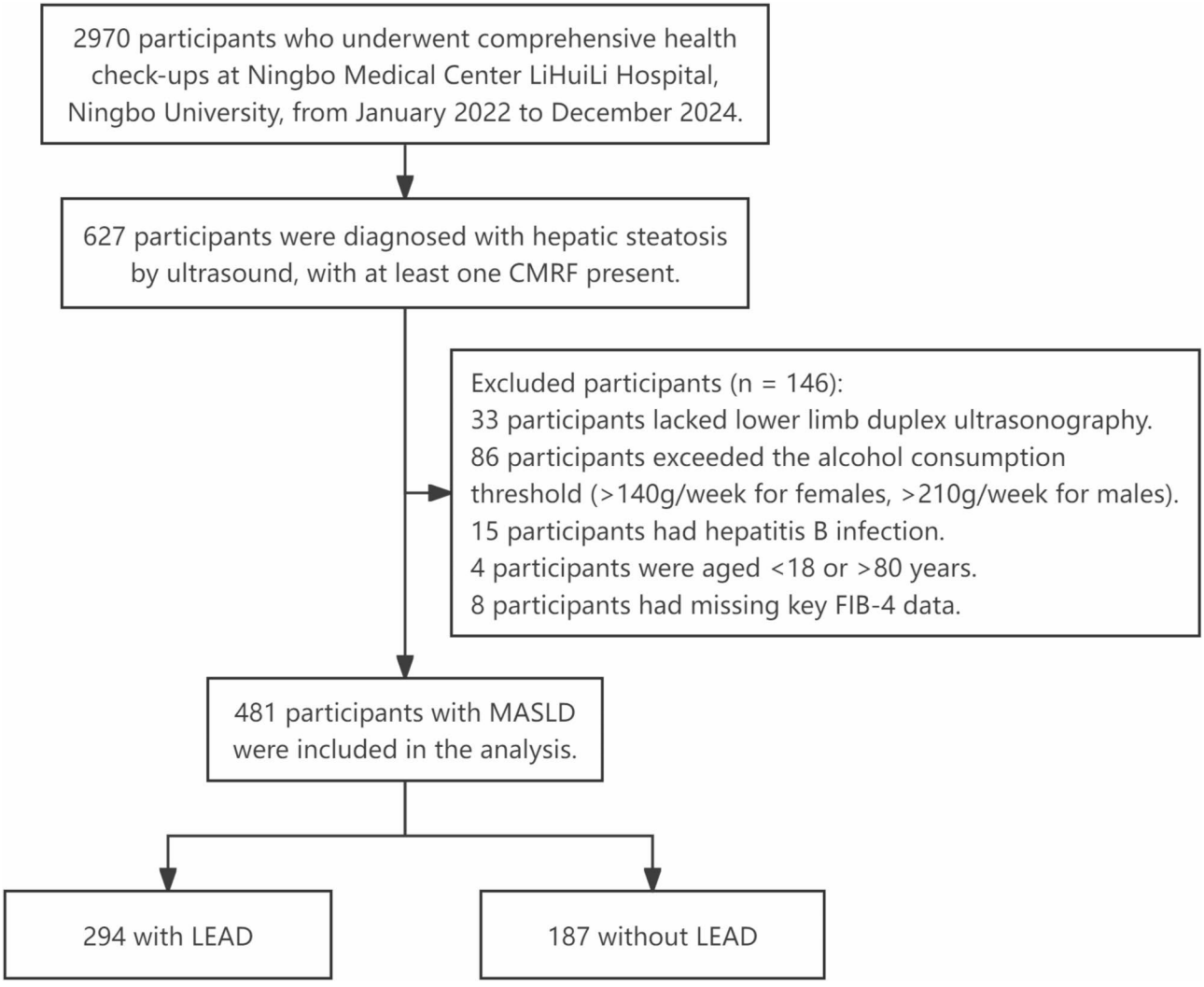

Fig. 1 Study flowchart. The study initially included 2,970 participants who underwent comprehensive health check-ups at Ningbo Medical Center LiHuiLi Hospital, Ningbo University, from January 2022 to December 2024. A total of 627 participants were diagnosed with hepatic steatosis by ultrasound, with at least one cardiometabolic risk factor(CMRF). Of these, 146 participants were excluded for the following reasons: 33 lacked lower limb duplex ultrasonography, 86 exceeded the alcohol consumption threshold (> 140 g/week for females, > 210 g/week for males), 15 had hepatitis B infection, 4 were aged < 18 or > 80 years, and 8 had missing key Fibrosis-4 (FIB-4) index data. The final cohort consisted of 481 participants, among which 294 were diagnosed with lower extremity arterial disease (LEAD) based on duplex ultrasonography, and 187 were without LEAD

MAFLD (adjusted OR: 3.07, 95% CI: 1.38–6.84, $P < 0.001$) (Table 2 and Table S1). In contrast, the intermediate FIB-4 group (1.3–2.66) did not exhibit a statistically significant association with LEAD in any adjusted models for either MASLD or MAFLD.

Subgroup analysis

The relationship between FIB-4 and LEAD was further analyzed in subgroups stratified by sex, age, T2DM status, hypertension, dyslipidemia, smoking status (Fig. 2). The association remained statistically significant in the overall analysis after adjusting for sex, age, BMI, hypertension, T2DM, smoking, and lipid profiles (adjusted OR: 1.66, 95% CI: 1.21–2.26, $P < 0.05$). Subgroup analyses did not reveal any significant interactions (P for interaction > 0.05). Specifically, while a significant association was observed in individuals aged ≥ 60 years (adjusted OR: 2.03, 95% CI: 1.18–3.49), no significant correlation was found in those under 60 years, or in subgroups stratified by T2DM status, non-hypertension, dyslipidemia, or former/current smoking status (P for interaction > 0.05 in all cases).

Discussion

In this study, we observed a significant association between the FIB-4 index, a non-invasive marker of liver fibrosis, and the prevalence of LEAD in patients with MASLD. Higher FIB-4 levels were independently

Table 1 Baseline characteristics of the study participants

Variables	Total ($n=481$)	Fibrosis-4 index			*p* value
		< 1.3 ($n=226$)	1.3–2.66 ($n=203$)	> 2.66 ($n=52$)	
Sex, n (%)					0.009
Male	245 (50.9)	130 (57.5)	87 (42.9)	28 (53.8)	
Female	236 (49.1)	96 (42.5)	116 (57.1)	24 (46.2)	
Age, years	58.4 ± 12.6	50.8 ± 11.2	64.9 ± 9.2	66.1 ± 11.7	< 0.001
BMI, kg/m^2	27.3 ± 3.7	27.6 ± 4.1	26.9 ± 3.2	27.2 ± 4.0	0.137
LEAD, n (%)	294 (61.1)	116 (51.3)	133 (65.5)	45 (86.5)	< 0.001
T2DM, n (%)					0.272
Non-T2DM	241 (50.1)	121 (53.5)	99 (48.8)	21 (40.4)	
Prediabetes	105 (21.8)	44 (19.5)	50 (24.6)	11 (21.2)	
T2DM	135 (28.1)	61 (27)	54 (26.6)	20 (38.5)	
Hypertension, n (%)	266 (55.3)	115 (50.9)	121 (59.6)	30 (57.7)	0.18
Dyslipidemia, n (%)	97 (20.2)	52 (23)	34 (16.7)	11 (21.2)	0.267
Former/Current smoker, n (%)	116 (24.1)	66 (29.2)	38 (18.7)	12 (23.1)	0.04
TC, mmol/L	4.5 ± 1.4	4.6 ± 1.2	4.4 ± 1.4	4.6 ± 2.3	0.18
HDL-C, mmol/L	1.1 ± 0.3	1.1 ± 0.3	1.1 ± 0.2	1.1 ± 0.5	0.425
LDL-C, mmol/L	2.6 ± 0.9	2.7 ± 0.8	2.5 ± 0.9	2.7 ± 1.1	0.009
FIB4[a]	1.3 (1.0, 1.9)	0.9 (0.7, 1.1)	1.6 (1.4, 2.1)	3.5 (3.1, 4.5)	< 0.001
TG[a], mmol/L	1.6 (1.2, 2.3)	1.8 (1.3, 2.4)	1.5 (1.1, 2.2)	1.6 (1.0, 2.3)	0.004
Lp(a)[a], g/L	0.1 (0.1, 0.2)	0.1 (0.1, 0.2)	0.1 (0.1, 0.2)	0.1 (0.1, 0.2)	0.887

Note: BMI, body mass index; FIB-4, Fibrosis-4 index; LEAD, lower extremity artery disease; T2DM, type 2 diabetes mellitus; TC, Total cholesterol; TG, Triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; Lp(a), lipoprotein(a); [a]FIB-4, triglycerides and Lp(a) expressed as median (IQR) and rest are expressed as mean ± SD

Table 2 Associations between FIB-4 and LEAD in the multiple regression model

Variable	FIB-4($n=481$)		FIB-4		
			< 1.3 ($n=226$)	1.3–2.66 ($n=203$)	> 2.66 ($n=52$)
	OR_95CI	P_value	OR_95CI	OR_95CI	OR_95CI
Unadjusted	2.11 (1.6 ~ 2.78)	< 0.001	1.00(ref)	1.8 (1.22 ~ 2.66)	6.1 (2.64 ~ 14.09)
Model1	1.54 (1.16 ~ 2.05)	0.003	1.00(ref)	1.06 (0.67 ~ 1.69)	2.96 (1.21 ~ 7.25)
Model2	1.58 (1.18 ~ 2.13)	0.002	1.00(ref)	1.1 (0.69 ~ 1.77)	3.32 (1.32 ~ 8.36)
Model3	1.66 (1.21 ~ 2.26)	0.001	1.00(ref)	1.26 (0.76 ~ 2.07)	3.54 (1.39 ~ 9.01)

Note: Model 1: adjusted for sex, age, BMI. Model 2: adjusted for model 1 + hypertension, T2DM and smoking status. Model 3: adjusted for model 2 + TC, TG, HDL, LDL, LPa(a). OR, odds ratio; 95% CI, 95% confidence interval; FIB-4, Fibrosis-4 index; LEAD, lower extremity artery disease; BMI, body mass index; T2DM, type 2 diabetes mellitus; TC, Total cholesterol; TG, Triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Lp(a), lipoprotein(a)

associated with increased odds of LEAD, even after adjusting for potential confounders such as age, sex, BMI, hypertension, T2DM, and lipid profiles. Subgroup analyses did not reveal any significant interactions (P for interaction > 0.05). The association between FIB-4 and LEAD remained consistent across most groups.

Comparison with existing research

Our findings are consistent with prior studies that have established links between liver fibrosis markers and systemic vascular complications. For example, **Su et al.** demonstrated an independent association between FIB-4 and carotid atherosclerosis in nonalcoholic fatty liver disease (NAFLD) patients, emphasizing the systemic implications of liver fibrosis [14]. Similarly, **Barbosa et al.** reported that FIB-4 predicts major adverse cardiovascular events in NAFLD, underscoring its prognostic value [15]. The close connection between MASLD and CVD has been well documented in recent literature. Targher et al. [28] underscore that MASLD is not only associated with liver dysfunction but also significantly increases the risk of cardiovascular morbidity and mortality, with metabolic disturbances such as insulin resistance and chronic inflammation contributing to vascular complications. These findings reinforce the importance of considering MASLD as a critical cardiovascular risk factor, further supporting the need for early detection and management of vascular health in these patients. Expanding on these studies, our research uniquely focuses on MASLD patients and highlights the specific relationship between FIB-4 and LEAD. Unlike **Orfanos et al.**, which Lp(a) as a predictor of cardiovascular outcomes, our study concentrated on liver fibrosis as measured by FIB-4, demonstrating its independent association with

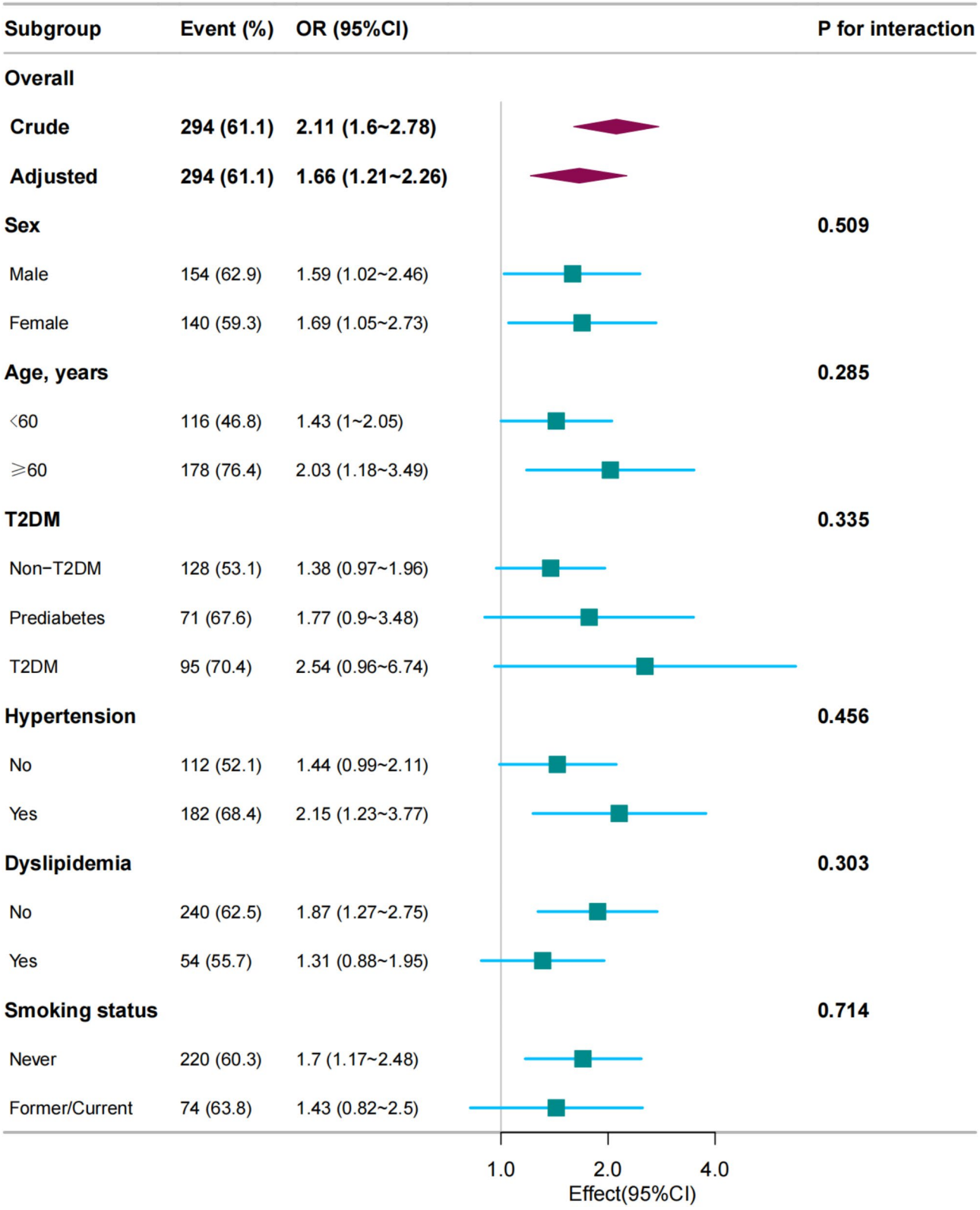

Fig. 2 Associations between FIB-4 and LEAD in different subgroups. The forest plot illustrates the associations between the Fibrosis-4 (FIB-4) index and lower extremity arterial disease (LEAD) across various subgroups. Each subgroup analysis was adjusted for all factors except the stratification variable itself, including sex, age, BMI, hypertension, diabetes mellitus (DM), smoking status, total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and lipoprotein(a) [Lp(a)]. Odds ratios (ORs) with 95% confidence intervals (CIs) are presented. No significant interactions were observed across subgroups (*P* for interaction > 0.05)

vascular complications, even after adjusting for lipid-related parameters. These distinctions underscore the importance of considering liver fibrosis in the context of systemic vascular health [29].

Unique observations in this study

One notable observation in this study is the strength of the association between FIB-4 and LEAD when FIB-4 is analyzed as both a continuous and categorical variable. Each unit increase in FIB-4 corresponded to a 38% higher likelihood of LEAD, reflecting the incremental value of FIB-4 as a risk stratification tool. Additionally, the prevalence of LEAD was significantly higher in the high FIB-4 group (> 2.66) compared to the low (< 1.3) and intermediate groups (1.3–2.66). This result aligns with the growing recognition of advanced liver fibrosis as a systemic risk factor for atherosclerotic diseases [6, 14, 30]. Our subgroup analyses further revealed that traditional risk factors, such as hypertension and smoking, did not significantly modify the association between FIB-4 and LEAD, suggesting that this relationship is independent of these variables.

Mechanistic insights

The association between FIB-4 and LEAD may reflect shared pathological processes in MASLD, including systemic inflammation, oxidative stress, and endothelial dysfunction. Advanced liver fibrosis promotes the release of pro-inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which accelerate vascular damage and atherogenesis [17, 31]. Furthermore, endothelial dysfunction plays a critical role in the co-existence of MASLD and arterial disease. Alterations in endothelial function are mediated by hepatic dysfunction and the release of inflammatory molecules from the liver, contributing to vascular endothelial injury, a key step in the progression of atherosclerosis in MASLD patients [32]. In addition, dysregulated lipid metabolism, including elevated LDL-C and TG, contributes to arterial stiffness and vascular remodeling [33, 34]. The inclusion of age as a component of the FIB-4 index may also explain the observed association, as age-related arterial stiffening and accumulation of metabolic risk factors exacerbate vascular complications in MASLD patients [1, 35].

Limitations and future directions

This study has several limitations. First, the cross-sectional design prevents establishing causality between FIB-4 and LEAD, and longitudinal studies are necessary to determine temporal relationships. Second, the study population was drawn from a single health check-up center, which may limit the generalizability of the findings to other populations or clinical settings. Third, unmeasured factors such as Hepatitis C infection, genetic predispositions, physical activity levels, and dietary patterns were not accounted for and could influence the observed associations. While duplex ultrasonography is an effective tool for diagnosing LEAD in most cases, it has certain limitations, including operator dependency and challenges in detecting early-stage disease. The inclusion of additional confirmatory imaging techniques, such as angiography, in future studies could help address these limitations and strengthen the reliability of our findings.

Future research should explore the longitudinal relationship between liver fibrosis markers and vascular outcomes to better understand causality. Mechanistic studies investigating the role of systemic inflammation and lipid dysregulation in MASLD-associated vascular disease may provide insights into potential therapeutic targets. Additionally, multicenter cohort studies with more diverse populations are needed to enhance the generalizability of these findings. Lastly, clinical trials evaluating interventions aimed at reducing liver fibrosis or systemic inflammation in MASLD patients may clarify whether these strategies can mitigate vascular complications.

Conclusions

This study suggests a potential association between liver fibrosis, assessed by the FIB-4 index, and the prevalence of LEAD in patients with MASLD. The findings indicate that FIB-4 may be a useful tool for assessing vascular risk in this population. However, further longitudinal studies are needed to confirm the causality of this relationship and explore the underlying mechanisms. Such research could help refine risk stratification and guide clinical management strategies for MASLD patients.

Abbreviations

CMRF	Cardiometabolic risk factor
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
BMI	Body mass index
CI	Confidence interval
CVD	Cardiovascular disease
DM	Diabetes mellitus
FIB-4	Fibrosis-4 index
SLD	Steatotic liver disease
HDL-C	High-density lipoprotein cholesterol
HOMA-IR	Homeostatic model assessment of insulin resistance
IQR	Interquartile range
LDL-C	Low-density lipoprotein cholesterol
LEAD	Lower extremity arterial disease
Lp(a)	Lipoprotein(a)
MASLD	Metabolic dysfunction-associated steatotic liver disease
NAFLD	Nonalcoholic fatty liver disease
OR	Odds ratio
SD	Standard deviation
T2DM	Type 2 diabetes mellitus
TC	Total cholesterol
TG	Triglycerides
TNF-α	Tumor necrosis factor-alpha

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12944-025-02516-7.

Supplementary Material 1

Acknowledgements
Not applicable.

Author contributions
Chunxia Zhang (C.Z.) designed the study, conducted the statistical analysis, and drafted the manuscript. Yuchen Ying (Y.Y.) and Yuanhui Ru (Y.R.) contributed to data collection and analysis. Ziliang Wu (Z.W.) and Yumeng Tian (Y.T.) assisted in interpreting the results and critically reviewed the manuscript. Pingping Shen (P.S.) and Shiyuan Cao (S.C.) supported the literature review and provided administrative support. Jing Zhang (J.Z.) participated in manuscript editing and submission preparation. Ri Liu (R.L.) supervised the study, provided expert guidance throughout the research process, and is the corresponding author. All authors have read and approved the final version of the manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This study was conducted in accordance with the principles of the Declaration of Helsinki and approved by the Institutional Review Board of Ningbo Medical Center LiHuiLi Hospital (Ethics Approval Number: KY2024SL448-01). The requirement for individual informed consent was waived due to the retrospective design and the use of anonymized health check-up data. All data were handled confidentially and used solely for research purposes.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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