BMC Gastroenterology

RESEARCH

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Estimated sdLDL-C as a biomarker of hepatic steatosis severity in MASLD: a retrospective study

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Abstract

Background Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease worldwide. However, there is a lack of cost-effective and accurate biomarkers to assess the degree of hepatic steatosis. Estimated small dense low-density lipoprotein cholesterol (EsdLDL-C), a calculated value derived from triglyceride (TG) and low-density lipoprotein cholesterol (LDL-C) levels, has emerged as a potential indicator. This study aimed to explore the relationship between EsdLDL-C and the severity of hepatic steatosis.

Methods This single-center retrospective study estimated and directly measured small dense low-density lipoprotein cholesterol (sdLDL-C) in 1,969 patients who underwent serum lipid testing at Changzhou Third People's Hospital between January and July 2024. Among these, 461 patients diagnosed with MASLD were included in the study. These patients were further classified into mild (Mil) and moderate-to-severe (Mod-Sev) groups based on controlled attenuation parameter (CAP) values to explore the relationship between EsdLDL-C and the severity of hepatic steatosis.

Results The correlation coefficient (R) between EsdLDL-C and DsdLDL-C was 0.837, with a bias of 0.223. Both EsdLDL-C (OR 1.095, 95% CI 1.029–1.180) and visceral fat area (VFA) (OR 1.019, 95% CI 1.010–1.028) were identified as independent risk factors for Mod-Sev steatosis compared to the Mil group. After adjusting for all confounders, patients with MASLD had a 1.155-fold increased risk of developing Mod-Sev hepatic steatosis for each unit increase in EsdLDL-C. Furthermore, EsdLDL-C demonstrated good predictive value for Mod-Sev steatosis in MASLD patients, with an area under the curve (AUC) of 0.825 (95% CI 0.784–0.867).

Conclusions EsdLDL-C may serve as a practical and cost-effective biomarker for identifying high-risk MASLD patients.

Trial registration The retrospective study was approved by the Ethics Committee of Changzhou Third People's Hospital (02 A-A20230015), and a waiver of informed consent was agreed to, as the data were obtained from medical records, and a waiver of informed consent would not have affected the participants.

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Keywords MASLD, EsdLDL-C, DsdLDL-C, Retrospective study

Introduction

In 1980, Ludwig and colleagues introduced the term non-alcoholic fatty liver disease (NAFLD) to describe fatty liver disease occurring in the absence of significant alcohol consumption. NAFLD represents the most prevalent chronic liver disease worldwide, with a global prevalence of approximately 30.1% [1, 2]. However, despite its widespread use, the term "non-alcoholic" has been criticized for being inaccurate and for overlooking the disease's association with other metabolic disorders. Consequently, the American Association for the Study of Liver Diseases (AASLD) and the European Association for the Study of the Liver (EASL) have jointly proposed a new terminology: metabolic dysfunction-associated steatotic liver disease (MASLD), which replaces "NAFLD" as the umbrella term [3].With the continuous improvement in living standards, the prevalence of MASLD among Chinese adults has reached 29.88%, and this figure is steadily increasing, making it a significant public health concern in China [4, 5]. MASLD initially presents as simple hepatic steatosis. If left untreated, the accumulation of excess triglycerides, free cholesterol, and ceramides can trigger hepatocellular stress responses and inflammation, potentially progressing to cirrhosis and hepatocellular carcinoma (HCC) [6–8]). Once cirrhosis develops, the process becomes difficult to reverse. Therefore, monitoring the progression of hepatic steatosis and implementing timely interventions are crucial for preventing the advancement of MASLD.

The liver plays a pivotal role in maintaining lipid homeostasis by synthesizing and transporting triglycerides [9, 10]. MASLD is associated with excessive daily nutrient intake, which leads to increased triglyceride production. These triglycerides accumulate abnormally in hepatocytes, resulting in systemic metabolic disorders. In addition to regulating lipid homeostasis through fatty acid oxidation, hepatocytes utilize an essential pathway to package fatty acids with cholesterol, phospholipids, and apolipoproteins, forming water-soluble very low-density lipoprotein (VLDL) particles. These VLDL particles are exported into the bloodstream, where they undergo heterogeneous transformation into low-density lipoprotein (LDL) particles [11]. Consequently, patients with MASLD frequently exhibit dyslipidemia. LDL particles are classified into two groups based on size and composition. The larger, lipid-rich LDL particles are known as large buoyant LDL (lbLDL), while the smaller, cholesterol-rich LDL particles are termed small dense low-density lipoprotein (sdLDL) [12, 13]. The production of sdLDL is closely linked to triglyceride (TG) levels. Elevated plasma TG promotes cholesteryl ester transfer protein (CETP)-mediated lipid exchange, enriching the hydrophobic core of LDL with TG. During the lipolysis of TG in LDL, the production of sdLDL is stimulated. sdLDL is considered highly atherogenic due to its increased permeability to the arterial wall. While most studies on sdLDL have focused on its association with cardiovascular disease risk [14, 15], no studies have demonstrated that sdLDL can directly damage the liver. However, recent studies have reported elevated levels of sdLDL particles in the blood of patients with MASLD [16, 17], although the relationship between sdLDL and MASLD progression remains unclear. Several methods are available to measure serum sdLDL-C levels. However, their high-cost limits widespread use. To address this limitation, Sampson et al. developed a novel formula for Estimated sdLDL-C (EsdLDL-C) based on LDL-C and TG levels [18]. This study aims to validate this formula using data from a Chinese population.

Ultrasonography is the most widely used imaging technique for diagnosing fatty liver based on image analysis. It is a standard diagnostic tool employed in medical institutions globally [19, 20]. Controlled attenuation parameter (CAP) based on transient elastography (TE) is a more sensitive modality than ultrasonography for diagnosing hepatic steatosis [21, 22]. Furthermore, CAP provides a continuous variable that quantifies the severity of hepatic steatosis. Given the high prevalence of MASLD and the need for non-invasive, cost-effective biomarkers, this study explores whether EsdLDL-C can serve as a reliable predictor of MASLD severity.

Methods

Study design and populations

This single-center retrospective study was conducted at Changzhou Third People's Hospital, China. It was approved by the Ethics Committee of Changzhou Third People's Hospital (02 A-A20230015), which agreed to waive informed consent because the data were obtained from medical records, and waiving informed consent would not have affected the participants.

The study population comprised 1969 patients who underwent TG, LDL-C, apolipoprotein B (ApoB), and DsdLDL-C tests from January to July 2024. EsdLDL-C was calculated for each patient. Of these, 482 patients were diagnosed with MASLD, and 461 were included in the study based on the following criteria:

Diagnostic criteria: MASLD was diagnosed according to the criteria recommended by the Chinese Liver Disease Association [23]. (1) CAP value > 238; (2) Ruling out excessive alcohol consumption and other possible causes

of fatty liver disease; (3) The patient has at least one metabolic syndrome (MetS) component.

Inclusion criteria: (1) Diagnosis of MASLD by established criteria; (2) Provision of comprehensive data.

Exclusion criteria: (1) Combined viral, autoimmune, and other liver diseases (positive test results for viral hepatitis antibody and antinuclear antibody); (2) A paucity of data exist about fundamental body information, laboratory indicators, and other pertinent details (height, weight, blood lipid tests, regular liver function tests, and body composition tests); (3) The consumption of excessive quantities of alcohol, defined as ≥ 210 g/week for men and ≥ 140 g/week for women.

The data were grouped according to the CAP values detected by FibroScan. The setting of CAP cut-offs was based on comprehensive data, depending on the degree of steatosis and the risk of developing other metabolic diseases: (1) Mild (Mil) group: CAP values in the range of 238–259, hepatic steatosis < 33%; (2) Moderate (Mod) group: CAP values in the range of 259–292, hepatic steatosis 33–66%; (3) Severe (Sev) group: CAP values in the range of > 292, hepatic steatosis > 66%.

Data collection and definitions

Sex and age were obtained from basic registration information. Height, weight, and visceral fat area (VFA) were measured using a body composition analyzer. Medical professionals collected all blood samples between 8:00 am and 10:00 am after an 8-hour fasting period, and serum was collected within one hour. All biochemical tests were completed by 2:00 pm on the same day. The levels of glucose (GLU), alanine aminotransferase (ALT), gamma-glutamyl transferase (γ-GGT), ApoB, TG, total cholesterol (CHOL), high-density lipoprotein cholesterol (HDL-C), and LDL-C were determined using a fully automated Hitachi biochemistry analyzer. The patient's liver CAP value was determined using the FibroScan M probe. The EsdLDL-C was calculated using the following equation: EsdLDL-C = 8.99 + 0.14 × ln (TG) × LDL-C– 0.43 × LDL-C.

Statistical analysis

The data were analyzed statistically using SPSS (version 23) and GraphPad Prism (version 9.0) software. The correlation between EsdLDL-C and LDL-C, DsdLDL-C, and ApoB was evaluated using Spearman correlation analysis. For continuous variables in the baseline data, normality was assessed using the Kolmogorov-Smirnov test. Variables that did not meet the normality assumption were described using the median and interquartile range (IQR). Group differences were assessed using the Kruskal-Wallis H test. The chi-square test was used to compare categorical variable (gender) between groups. This test evaluates whether the observed frequencies differ significantly from the expected frequencies under the null hypothesis. Odds ratios (OR) and 95% confidence intervals (95% CI) were calculated using logistic regression analysis to examine the association between EsdLDL-C and the severity of hepatic steatosis. Multicollinearity was assessed for variables with $P > 0.05$ in univariate analyses, and all variables had a variance inflation factor (VIF) < 5 after excluding LDL-C. These variables were included in multifactorial logistic regression analyses, and the risk of MASLD progression was compared after adjusting for confounders. A receiver operating characteristic (ROC) curve was used to evaluate the predictive ability of EsdLDL-C for Mod-Sev steatosis.

Results

Correlation between EsdLDL-C and LDL-C, ApoB, DsdLDL-C

Patients were divided into four categories according to TG content. Correlation analysis showed that EsdLDL-C and DsdLDL-C had a strong correlation with LDL-C and ApoB (Fig. 1A and D). EsdLDL-C and DsdLDL-C levels were significantly higher in patients with high TG levels. The correlation coefficient (R) between EsdLDL-C and DsdLDL-C was 0.837 (Fig. 1E). The Bland-Altman plot showed a bias of 0.223 between EsdLDL-C and DsdLDL-C (Fig. 1F).

Baseline characteristics of patients

A total of 461 patients were included, with 90 in the Mil group and 371 in the Mod-Sev group (194 in the moderate group and 177 in the severe group). The table presents the data for the four groups, and the *P* - value indicates the difference between the Mil and Mod-Sev groups. There was no significant difference between patients in the Mil and Mod-Sev groups regarding age and gender. However, compared to the Mil group, the Mod -Sev group had significantly higher BMI, VFA, LDL-C, EsdLDL-C, and CAP values, indicating a strong association between lipid metabolism and hepatic steatosis (Table 1).

Relationship between EsdLDL-C and the severity of hepatic steatosis in patients with MASLD

Univariate logistic regression analysis was used to assess the correlation between variables and the extent of hepatic steatosis (Table 2). Variables with $P < 0.05$ were selected for multicollinearity analysis, which revealed multicollinearity between LDL-C and EsdLDL-C (VIF > 5), while no multicollinearity was observed between the respective variables after removing LDL-C. These variables were included in a multivariate logistic regression analysis, which showed that VFA (OR = 1.019, 95% CI: 1.010–1.028, $P < 0.001$) and EsdLDL-C (OR = 1.095, 95% CI: 1.029–1.180, $P = 0.002$) were independent risk factors for Mod-Sev steatosis compared

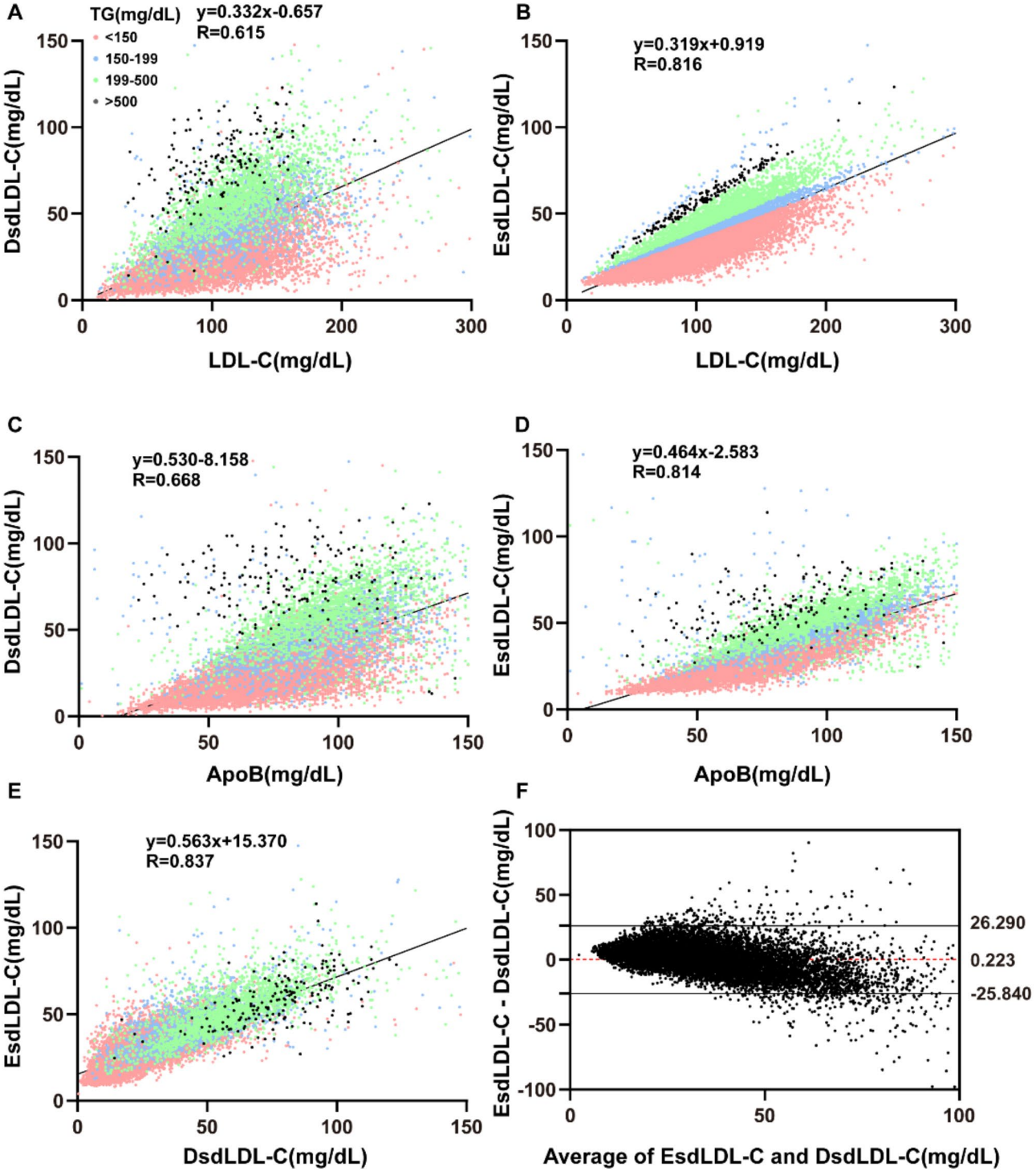

Fig. 1 Correlation analysis between EsdLDL-C, DsdLDL-C and LDL-C, ApoB. (**A**) Correlations between DsdLDL-C and LDL-C; (**B**) Correlations between EsdLDL-C and LDL-C; (**C**) Correlations between DsdLDL-C and ApoB; (**D**) Correlations between EsdLDL-C and ApoB; (**E**) Correlations between EsdLDL-C and DsdLDL-C; (**F**) The Bland–Altman plot of the relation between EsdLDL-C and DsdLDL-C. The red horizontal line showed the bias. The black horizontal lines showed 95% limits of agreement

Table 1 Characteristics of the patients

	Mil (*n* = 90)	Mod (*n* = 194)	Sev (*n* = 177)	Mod-Sev (*n* = 371)	Test of significance	*P* value
Age (years)	45.5(36.0–56.0)	48(36.8–58.0)	40(31.0–53.0)	44(33.0–56.0)	H = 5.231	0.426
Male (%)	51(56.7)	112(57.7)	115(65.0)	227(61.2)	χ^2=0.472	0.472
BMI (kg/m^2)	24.6(22.8–26.5)	26.3(24.4–28.0)	28.0(25.4–31.0)	26.9(24.7–29.0)	H = 25.891	< 0.001
VFA (cm^2)	88.1(73.4-107.5)	106.8(82.6-135.1)	120.1(94.2-161.1)	112.6(88.8–150.0)	H = 30.452	< 0.001
FBG (mg/dL)	95.4(88.2-108.1)	95.4(90.0-109.8)	99.0(90.0-113.4)	97.2(90.0-111.6)	H = 2.015	0.359
ALT (U/L)	27.2(18.8–45.8)	27.3(18.0-50.1)	41.7(25.8–83.7)	34.0(21.2–74.0)	H = 15.231	< 0.001
γ-GGT (U/L)	26.0(18.0-47.3)	34.0(20.9–60.1)	46.0(30.3–74.5)	38.9(23.3–66.0)	H = 3.012	0.239
TG (mg/dL)	85.0(63.8-108.5)	119.6(87.7-171.8)	142.6(108.1-199.3)	132.0(94.8-187.3)	H = 45.231	< 0.001
CHOL (mg/dL)	150.8(135.0-168.2)	174.4(151.1-195.7)	180.2(159.3-207.9)	177.5(155.1-202.6)	H = 30.891	< 0.001
HDL-C (mg/dL)	46.4(38.2–52.6)	43.5(37.5–51.8)	42.2(35.9–50.7)	42.9(36.7–51.0)	H = 2.512	0.474
LDL-C (mg/dL)	88.6(73.1–105.0)	111.9(89.2-132.2)	114.1(99.2-135.5)	113.3(94.4-133.4)	H = 35.231	< 0.001
CAP (db/m)	252(245–255)	279(269–286)	337(315–360)	291(279–335)	H = 50.231	< 0.001
EsdLDL-C (mg/dL)	25.8(21.6–30.2)	35.5(28.1–44.9)	40.1(32.5–48.9)	37.9(29.4–46.3)	H = 40.231	< 0.001

The *P* values between Mod-Sev group with Mil group

BMI, body mass index; VFA, visceral fat area; FBG, fasting blood glucose; ALT, alanine aminotransferase; γ-GGT, γ-glutamyl transpeptidase; TG, triglyceride; CHOL, cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein; CAP, controlled attenuation parameter; EsdLDL-C, estimated small dense low-density lipoprotein cholesterol

Table 2 Univariate analysis of factors associated with Mod-Sev steatosis

Variable	Univariate logistic regression analysis		
	OR (95%CI)	β	*P* value
Age (years)	0.994(0.978–1.009)	-0.006	0.426
Male (%)	1.205(0.753–1.918)	0.186	0.432
BMI (kg/m^2)	1.176(1.077–1.292)	0.162	0.153
VFA (cm^2)	1.021(1.013–1.029)	0.021	< 0.001
FBG (mmol/L)	1.003(0.997–1.010)	0.003	0.360
ALT (U/L)	1.000(0.998–1.003)	0	0.865
γ-GGT (U/L)	1.000(0.999–1.003)	0	0.753
TG (mg/dL)	1.020(1.014–1.027)	0.020	< 0.001
CHOL (mg/dL)	1.033(1.023–1.043)	0.032	< 0.001
HDL-C (mg/dL)	0.993(0.975–1.012)	-0.007	0.473
LDL-C (mg/dL)	1.035(1.025–1.046)	0.034	< 0.001
EsdLDL-C (mg/dL)	1.153(1.116–1.197)	0.142	< 0.001

OR, odds ratio; 95%CI, 95%confidence interval; β, regression coefficient; BMI, body mass index; VFA, visceral fat area; FBG, fasting blood glucose; ALT, alanine aminotransferase; γ-GGT, γ-glutamyl transpeptidase; TG, triglyceride; CHOL, cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein; CAP, controlled attenuation parameter; EsdLDL-C, estimated small dense low-density lipoprotein cholesterol

Table 3 Multivariate analysis of factors associated with Mod-Sev steatosis

Variable	Multivariate logistic regression analysis		
	OR (95%CI)	β	*P* value
VFA (cm^2)	1.019(1.010–1.028)	0.019	< 0.001
TG (mg/dL)	1.005(0.999–1.013)	0.005	0.137
CHOL (mg/dL)	1.012(0.999–1.026)	0.012	0.090
EsdLDL-C (mg/dL)	1.095(1.029–1.180)	0.091	0.002

OR, odds ratio; 95%CI, 95%confidence interval; β, regression coefficient; VFA, visceral fat area; TG, triglyceride; CHOL, cholesterol; EsdLDL-C, estimated small dense low-density lipoprotein cholesterol

with patients in the Mil group (Table 3). A logistic regression model was constructed according to Table 4, and after adjusting for all confounders, patients with MASLD had a 1.155-fold increased risk of developing Mod-Sev hepatic steatosis for each unit increase in EsdLDL-C (OR = 1.155, 95% CI: 1.096–1.218, $P < 0.001$).

The EsdLDL-C levels were significantly higher in the Mod-Sev group than in the Mil group. As shown in Fig. 2, an EsdLDL-C cut-off of 28.5 mg/dL is an effective predictor of Mod-Sev steatosis (AUC = 0.825, 95% CI: 0.784–0.867, $P < 0.001$).

Discussion

Liver biopsy has historically been the gold standard for monitoring and diagnosing MASLD. However, it has several inherent limitations, including invasiveness, sampling error, high cost, and patient acceptability issues, making it unsuitable for wide-scale application. Some studies have shown that the CAP value obtained with the M probe shows a strong correlation with the histological assessment of steatosis and can more accurately and non-invasively evaluate the extent of hepatic steatosis [24–26]. However, due to the high cost, this procedure is only conducted in major hospitals in China, and its adoption rate remains low. In contrast, serum markers are less accurate than CAP values but have wide applicability and high reproducibility.

It has been demonstrated that elevated serum triglycerides resulting from prolonged overnutrition contribute to MASLD and are a determining factor in the excessive synthesis of VLDL by the liver [27–29]. Lipid disorders in hepatocytes increase the secretion of VLDL into the bloodstream, which is then isomerized to LDL in the bloodstream [30]. It has been observed that for a given

Table 4 Association of EsdLDL-C and Mod-Sev steatosis

Variable	Model1		Model2		Model3	
	OR (95%CI)	*P* value	OR (95%CI)	*P* value	OR (95%CI)	*P* value
VFA	1.021 (1.013–1.028)	< 0.001	1.020 (1.011–1.030)	< 0.001	1.013 (0.997–1.029)	0.125
EsdLDL-C	1.153 (1.114–1.194)	< 0.001	1.142 (1.102–1.183)	< 0.001	1.155 (1.096–1.218)	< 0.001

OR, odds ratio; 95%CI, 95%confidence interval; VFA, visceral fat area; EsdLDL-C, estimated small dense low-density lipoprotein cholesterol

Model1: Crude

Model2: Adjust: Gender, Age

Model3: Adjust: Gender, Age, BMI, ALT, GGT, TC, HDL, FBG

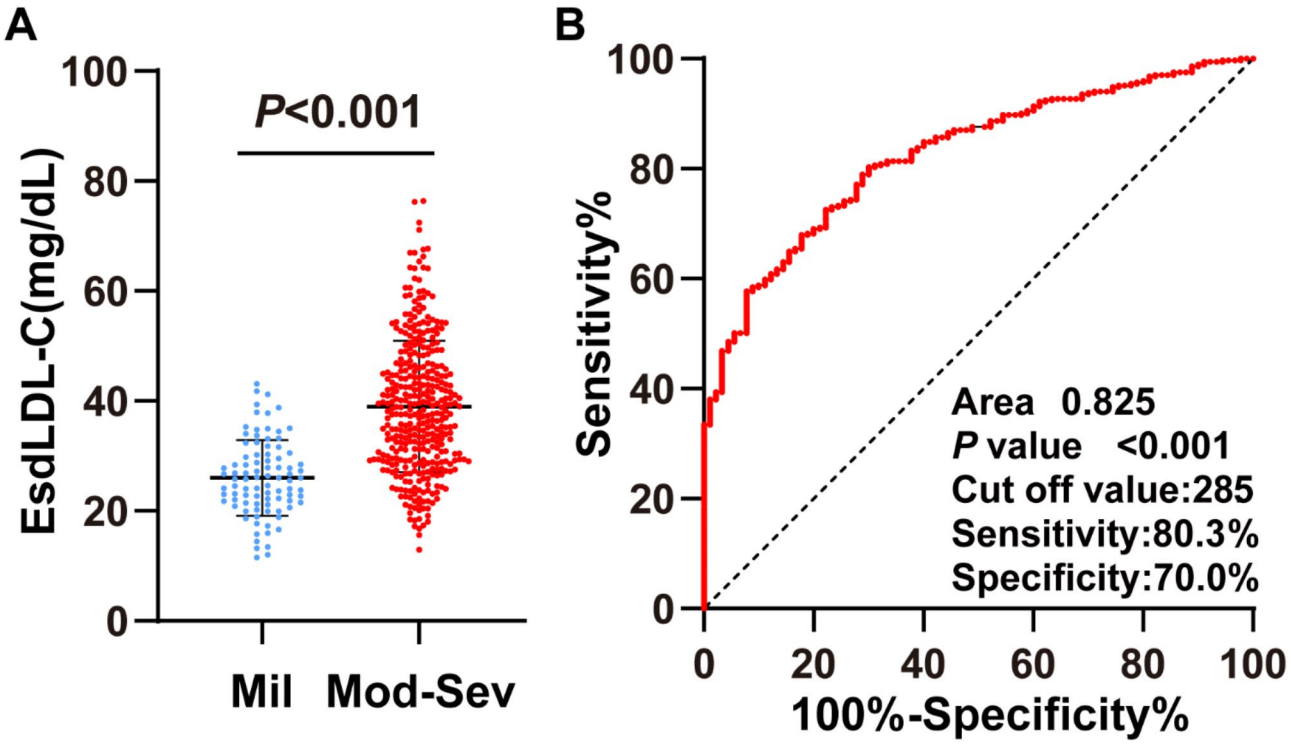

Fig. 2 EsdLDL-C predicts Mod-Sev steatosis. (**A**) EsdLDL-C levels of Mil group and Mod-Sev group; (**B**) ROC curve for the use of EsdLDL-C in the detection of Mod-Sev steatosis

LDL mass, the number of LDL molecules increases with increasing levels of sdLDL. This may be one of the reasons for the stronger correlation between sdLDL-C and MASLD [31, 32].

The enzymatic measurement of sdLDL-C is a commonly used clinical practice. However, it is a more costly test, and its results can be influenced by high TG levels, limiting its accuracy [33]. Additionally, this test is not universally available, as fewer hospitals have the capacity to perform it. To address these limitations, Sampson et al. developed an equation for estimating sdLDL-C based on TG and LDL-C levels. This equation has been shown to be an independent risk factor for predicting atherosclerotic cardiovascular disease (ASCVD) (OR = 1.28, 95% CI: 1.18–1.38, $P < 0.001$) [18]. Furthermore, the correlation between EsdLDL-C and DsdLDL-C was validated in a multiracial population in the United States, demonstrating a coefficient of determination of 0.745 [34]. In our study, the coefficient of determination of EsdLDL-C for DsdLDL-C was 0.726 in the Chinese population. The findings also indicated that elevated TG levels were associated with increased DsdLDL-C levels at specific LDL-C phase levels, with this association being more pronounced for EsdLDL-C [18]. This suggests that TG levels may influence both measurement methods.

A previous study investigated the relationship between EsdLDL-C and the risk of MASLD in a healthy population. The results revealed a 2.93-fold increased risk of MASLD in the fourth quartile of EsdLDL-C compared to the first quartile (OR = 2.93, 95% CI: 2.10–4.11, $P < 0.001$) [35]. However, no study has yet explored the relationship between EsdLDL-C and the severity of hepatic steatosis

in patients with MASLD. Our study identified EsdLDL-C and VFA as independent risk factors for MASLD progression. After adjusting for various confounders, EsdLDL-C remained statistically significant. Therefore, we used EsdLDL-C as the independent variable and the development of Mod-Sev steatosis in patients with MASLD as the dependent variable to generate ROC curves, with a cut-off value of 28.5 mg/dL. Clinicians often recommend that people with mild MASLD manage their disease through a healthy diet and exercise. In contrast, timely pharmacological intervention is an effective means of controlling the progression of Mod-Sev MASLD. Liver injury biomarkers such as ALT are often used as indicators of MASLD progression, but when ALT is elevated, the liver is already damaged. A comparison of the Mild and moderate (Mod) groups showed that ALT levels remained almost unchanged, while EsdLDL-C was significantly elevated. Therefore, early intervention based on the EsdLDL-C cut-off value can effectively prevent liver injury. In summary, EsdLDL-C could be a useful, non-invasive biomarker for identifying MASLD patients at risk of disease progression (AUC = 0.825, 95% CI: 0.784–0.867, $P < 0.001$).

EsdLDL-C levels increase with the progression of MASLD. Does sdLDL similarly promote the progression of MASLD? The small size of sdLDL enables it to have strong endothelial penetration. Due to its lower affinity for LDL receptors, sdLDL circulates in the blood for longer periods and is more susceptible to glycation and oxidation. Consequently, it promotes atherosclerosis, a process that also occurs in the liver vessels and may exacerbate the hepatic inflammatory response or fibrosis in MASLD patients. Therefore, exploring the effects of sdLDL on the liver in the future would be valuable.

Study Trengths and limitations

This study has several advantages. Firstly, it demonstrated that EsdLDL-C strongly correlates with DsdLDL-C in the Chinese population. Secondly, this study demonstrated that EsdLDL-C can be used as an effective biomarker to monitor MASLD progression. Compared with other tests related to MASLD, EsdLDL-C requires lower-cost testing of TG and LDL-C, making it suitable for economically underdeveloped or remote areas. Lastly, the data were collected directly by the authors to ensure the accuracy and integrity of the dataset.

The present study is not without limitations. Firstly, the study period is relatively short, and there is scope to increase the sample size. We will continue to collect samples to further verify the reliability of the findings. Secondly, the patients were recruited from a single region, which may limit the generalizability of the data. For instance, Changzhou is located in southern China, where dietary habits differ from those in northern China, where fat intake is generally higher. To address this, we are seeking collaborations across both northern and southern China and plan to conduct a multicenter study to validate our findings. Thirdly, data on comorbidities in patients with MASLD were not collected. Given that MASLD is often associated with type 2 diabetes and cardiovascular disease (CVD), we are currently collecting additional data to explore the relationship between EsdLDL-C and MASLD comorbidities.

Conclusion

EsdLDL-C correlated strongly with DsdLDL-C, although both were influenced by TG levels. EsdLDL-C and VFA are independent risk factors for the development of Mod-Sev steatosis in patients with MASLD. Furthermore, EsdLDL-C serves as a potent biomarker for identifying disease progression in MASLD patients.

Abbreviations

MASLD	Metabolic dysfunction associated steatotic liver disease
BMI	Body mass index
VFA	Visceral fat area
FBG	Fasting blood glucose
ALT	Alanine aminotransferase
γ-GGT	γ-Glutamyl transpeptidase
TG	Triglyceride
CHOL	Cholesterol
HDL-C High	density lipoprotein cholesterol
LDL-C Low	density lipoprotein
CAP	Controlled attenuation parameter
EsdLDL-C	Estimated small dense low-density lipoprotein cholesterol
DsdLDL-C	Direct measurement small dense low-density lipoprotein cholesterol
CI	Confidence interval
VIF	Variance inflation factor

Acknowledgements

Not applicable.

Author contributions

JS, ZGJ and FY designed the idea for the study; JS, ZF, YH, HX, and MJR contributed to the analysis of the data; JS and FY wrote the manuscript.

Funding

This study was supported by the Changzhou Natural Science Foundation. (CJ20245033; CJ20230075; CJ20220226)

Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The study followed guidelines outlined by the Helsinki Declaration and was approved by the Ethics Committee of Changzhou Third People's Hospital (02 A-A20230015). In addition, the Ethics Committee of Changzhou Third People's Hospital has waived informed consent for the current retrospective study.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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Received: 17 December 2024 / Accepted: 4 March 2025
Published online: 13 March 2025

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