Independent risk factors of left ventricular hypertrophy in non-diabetic individuals in Sierra Leone - a cross-sectional study

Yuanxin Xu[1,2,3†], Yingxin Celia Jiang[3,4,5†], Lihua Xu[6], Weiyu Zhou[1], Zhiying Zhang[1], Yanfei Qi[3,4], Hongyu Kuang[2,7*] and Shuang Yan[1,7*]

Abstract

Background Left ventricular hypertrophy (LVH) is a critical factor in heart failure and cardiovascular event-related mortality. While the prevalence of LVH in diabetic patients is well-documented, its occurrence and risk factors in non-diabetic populations remain largely unexplored. This study addresses this issue by investigating the independent risk factors of LVH in non-diabetic individuals.

Methods This cross-sectional study, conducted meticulously, utilized data from a robust and comprehensive source, DATADRYAD, in the Sierra Leone database, collected between October 2019 and October 2021, including LVH and various variables. All variables were described and screened using univariate analysis, Spearman correlation, and principal component analysis (PCA). The lipid profile, including total cholesterols (TC), triglycerides (TG), high-density lipoprotein (HDL-C), non-high-density lipoprotein (Non-HDL-C), and low-density lipoprotein cholesterol (LDL-C), TC/HDL-C ratio, TG/HDL-C ratio, Non-HDL-C /HDL-C ratio and LDL-C/HDL-C ratio, which quartiles were treated as categorical variables, with the lowest quartile serving as the reference category. Three adjusted models were constructed to mitigate the influence of other variables. To ensure the robustness of the model, receiver operating characteristic (ROC) curves were used to calculate the cutoff values by analyzing the ROC curves. A sensitivity analysis was performed to validate the findings further.

Results The dataset encompasses information from 2092 individuals. After adjusting for potential factors that could influence the results, we found that TC (OR = 2.773, 95%CI: 1.805–4.26), Non-HDL-C (OR = 2.74, 95%CI: 1.7723–4.236), TC/HDL-C ratio (OR = 2.237, 95%CI: 1.445–3.463), Non-HDL-C/HDL-C ratio (OR = 2.357, 95%CI: 1.548–3.588), TG/HDL-C ratio (OR = 1.513, 95%CI: 1.02–2.245) acts as independent risk factors of LVH. ROC curve analysis revealed the predictive ability of blood lipids for LVH, with Non-HDL-C exhibiting area under the curve (AUC = 0.6109), followed by TC (AUC = 0.6084).

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†Yuanxin Xu and Yingxin Celia Jiang contributed equally to this work.

*Correspondence:
Hongyu Kuang
ydyneifenmi@163.com
Shuang Yan
qingmei0724@163.com

Full list of author information is available at the end of the article

Conclusions TC, non-HDL-C, TC/HDL-C ratio, Non-HDL-C/HDL-C ratio, and TG/HDL-C ratio were independent risk factors of LVH in non-diabetic people. Non-HDL-C and TC were found to be essential indicators for predicting the prevalence of LVH.

Keywords Left ventricular hypertrophy, Left ventricular mass index, Lipid profile, Non-diabetic

Introduction

Heart failure (HF) syndrome is a major clinical and public health problem associated with notable morbidity and mortality [1], which affects more than 64 million people worldwide [2]. Adverse changes in cardiac structure and function, known as cardiac remodeling, play a crucial role in the development of heart failure [3]. A particularly significant aspect of this process is LVH, defined as LVMI greater than 95 g/m²in women and greater than 115 g/m²in men [4, 5]. In LVH, the thickening of the heart wall leads to increased muscle stiffness, known as diastolic dysfunction, which impairs the heart's ability to relax and appropriately fill with blood during the resting phase of the cardiac cycle [6, 7]. Consequently, the heart's efficiency in pumping blood is diminished, resulting in decreased cardiac output and an increased workload on the heart [8]. Over time, this can lead to the heart being unable to pump with the necessary force [9, 10]. Approximately 8.7–13% of patients with LVH develop dysfunction of the left ventricular ejection fraction, which is a significant risk factor for cardiovascular mortality [11]. Early identification of individuals at higher risk for developing LVH is crucial for reducing related disability and mortality [12, 13]. According to some cross-sectional studies, diabetic individuals with high levels of glycated hemoglobin (HbA1c) may have increased left ventricular mass, which may be related to blood glucose uncontroled [14]; the prevalence of LVH in diabetes was 17.1-20.7% in Italy and Iran [15, 16]. Most studies have concentrated on examining risk factors for heart failure (HF) among individuals with diabetes, leaving a significant void in understanding the same in populations without diabetes [17]. Triglycerides and high-density lipoprotein have been observed to be associated with LVH in the general population in China [18]. In comparison, this study aimed to investigate whether lipid profile, including TC, TG, HDL-C, LDL-C, and Non-HDL-C, are independent risk factors that will predict LVH in the non-diabetic population and to assess their predictive value.
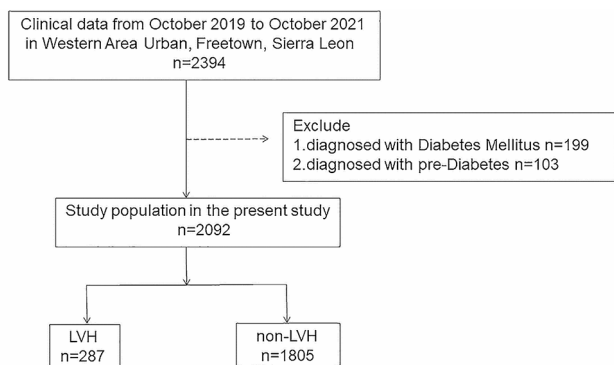
Methods

Study population

This cross-sectional clinical data was collected from pedestrians living in Western Area Urban, Freetown, Sierra Leone, between October 2019 and October 2021 to evaluate the prevalence of cardiometabolic risk factors among adults in Sierra Leone. This study examined the prevalence of cardiometabolic risk factors, target organ damage, and their associated factors among adults [19]. This project's data were segregated from the "DATADRYAD" database (https://datadryad.org/stash). As the study was a secondary study examining public data from a database, no ethical approval was required. This study obtained data from a database comprising information on 2,394 participants. After exclusions, which involved removing 199 participants with a history of diabetes mellitus and 103 participants with pre-diabetes, the final cohort included 2,092 individuals. Among them, 287 were identified as LVH, while the remaining 1,805 were classified as non-LVH (Fig. 1).

Variables of interest

Physical examinations were conducted on all participants as part of the study, encompassing various aspects such as demographic data (age, gender, and income), lifestyle factors (fruit and vegetable consumption, smoking, and alcohol consumption), medical history (family history of hypertension and diabetes mellitus), anthropometric measurements (Body weight, height, and waist circumference), echocardiography, and blood tests. Information about the participants was collected using standard survey questionnaires. Blood pressure was measured using an Omron electronic sphygmomanometer with an appropriately sized cuff. Participants were seated during the recording, preceded by 3–5 min of rest. The average of two recordings was taken as the blood pressure reading. Weight, height, and waist circumference measurements were taken while participants were lightly clothed and barefoot. These measurements provide insight into body composition and health status. Blood samples were collected from the median cubital vein after an overnight fast (8–10 h) on the morning of the second day. Hypertension was defined as an average systolic blood pressure of 140 mm Hg or higher, diastolic blood pressure of 90 mm Hg or greater, or a participant-reported current use of antihypertensive medication. A participant who smoked more than 100 sticks of cigarettes in their lifetime and was still smoking at the interview was referred to as a smoker, while an ex-smoker was someone who had stopped smoking at least 28 days before the interview. Data on alcohol consumption were based on the WHO step survey tool. The body mass index (BMI) was calculated as a ratio of the weight in kilograms and the square of the height in meters. BMI-based body habitus was classified as obese (BMI≥30 kg/m²), and abdominal obesity was defined as WC>88 cm for women and

Clinical data from October 2019 to October 2021
in Western Area Urban, Freetown, Sierra Leon
n=2394

Exclude
1.diagnosed with Diabetes Mellitus n=199
2.diagnosed with pre-Diabetes n=103

Study population in the present study
n=2092

LVH
n=287

non-LVH
n=1805

Fig. 1 Study protocol. Baseline with 2398 participants, 302 were excluded. Total of 2092 subjects were finally included in the analysis

102 cm for men. Diabetes mellitus was defined as a fasting blood glucose (FPG) level of 7.0 mmol/L or greater, HbA1c≥6.5%, or using insulin or an oral hypoglycaemic agent. Pre-diabetes was defined as FPG between 6.1 mmol/L and 6.9 mmol/L. The blood tests included analysis for total TC, TG, HDL-C, and LDL-C levels. TC calculated Non-HDL-C minus HDL-C. All lipid profile were grouped according to the interquartile method. LVMI was calculated by dividing Left ventricular mass by body surface area.

Statistical analyses

SPSS (Version 25.0), GraphPad Prism (Version 9.5.0), and R (Version 4.3.0) were used for data analysis. Univariate analysis was performed for all variables using SPSS. Categorical variables were presented as percentages and compared using a chi-square test. Continuous variables perform a Kolmogorov-Smirnov test, The T-test was used for normal distribution, and the Mann-Whitney U was used for non-normal distribution. The correlation between LVMI and other variables was analyzed using Spearman correlation analysis. Depending on whether LVH or not, PCA was performed on a dataset containing continuous variables using R to reduce variable number. Lipid quartiles were treated as a categorical variable, with the lowest quartile as the reference category. The logistic regression test was performed to investigate the correlation between lipids and LVH using Graphpad. Three adjusted models were constructed to reduced confounders. Model I adjusted for age and income, and model II further adjusted for hypertension, alcohol consumption history, and smoking history. Model III was further adjusted for waist circumference and estimated glomerular filtration rate (eGFR). ROC curves were used to analyze the sensitivity and heterogeneity of the model and calculate the cut-off values. $P < 0.05$ were considered statistically significant.

Table 1 Comparison of characteristics of the LVH and Non-LVH group

Characteristics	LVH (*n* = 287)	Non-LVH (*n* = 1805)	*P* value
Male, n (%)	158(55.05)	941(52.13)	0.358
Age > 50, n (%)	112(39.02)	369(20.44)	<0.001
HTN, n (%)	199(69.33)	455(25.21)	<0.001
Fruits/ Vegetable > 3 serving, n (%)	25(8.71)	162(8.98)	0.884
Alcohol, n (%)	132(45.99)	650(36.01)	0.004
Smoking, n (%)	15(5.23)	263(14.57)	<0.001
Obese, n (%)	36(12.54)	185(10.25)	0.24
Income, n (%)	108(37.63)	597(33.07)	0.215
EGFR Stages > II, n (%)	46(16.03)	44(2.43)	<0.001
Dyslipidemia, n (%)	78(27.18)	367(20.33)	0.008
Male WC, n (%)	60(20.91)	316(17.51)	0.538
Female WC, n (%)	50(17.42)	315(17.45)	0.99
HbA1c (%)	5.1(4.8,5.7)	4.7(5.1,5.6)	0.16
Creatinine (mmol/L)	73(65,85)	73(66,87)	0.86
WC (cm)	89.5(79.4,93.4)	89(78.9,92.7)	0.03
TC (mmol/L)	4.89(4.7,6)	4.77(4.66,4.95)	<0.001
LDL-C (mmol/L)	2.92(2.6,3.2)	2.99(2.63,3.22)	0.18
Non-HDL-C (mmol/L)	3.58(3.34,4.7)	3.4(3.28,3.64)	<0.001
HDL-C (mmol/L)	1.34(1.21,1.42)	1.34(1.3,1.41)	0.14
TG (mmol/L)	1.56(1.47,2)	1.53(1.46,1.63)	<0.001
TG/HDL-C ratio	1.18(1.06,1.49)	1.14(1.06,1.21)	<0.001
TC/HDL-C ratio	3.63(3.38,4.435)	3.51(3.31,3.88)	<0.001
Non-HDL-C/HDL-C ratio	2.6(2.4,3.45)	2.5(2.3,2.9)	<0.001
LDL-C/HDL-C ratio	2.2(1.99,2.655)	2.19(1.96,2.48)	0.1

Abbreviations HTN, Hypertension; WC, Circumference; TC, total cholesterol; TG, triglyceride, HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol. Continuous variables that follow a normal distribution are compared using a t-test. Non-normally distributed continuous variables are compared using the Mann-Whitney U test. *P* < 0.05 is considered as statistically significant

Sensitivity analysis

A series of stratified analyses were conducted to evaluate the potential modification of classical risk factors, namely gender and HbA1c. Additionally, BMI adjustments will be made to mitigate the confounding effects of metabolic syndrome. Considering hypertension is an independent risk factor for LVH, its confounding effect will be further examined in the subgroup analysis stratified by hypertension (HTN).

Results

Comparison of characteristics of the LVH and Non-LVH group

Univariate analysis was performed for all variables based on the presence or absence of LVH, and the results are presented in Table 1. A significant increase in TC, TG, Non-HDL-C, and TC/HDL-C levels was observed in the LVH group when compared to the non-LVH group ($P < 0.001$) (Fig. 2). However, compared to the non-LVH group, patients with LVH group exhibited a significant decrease in HDL-C levels ($P < 0.01$). The two groups had
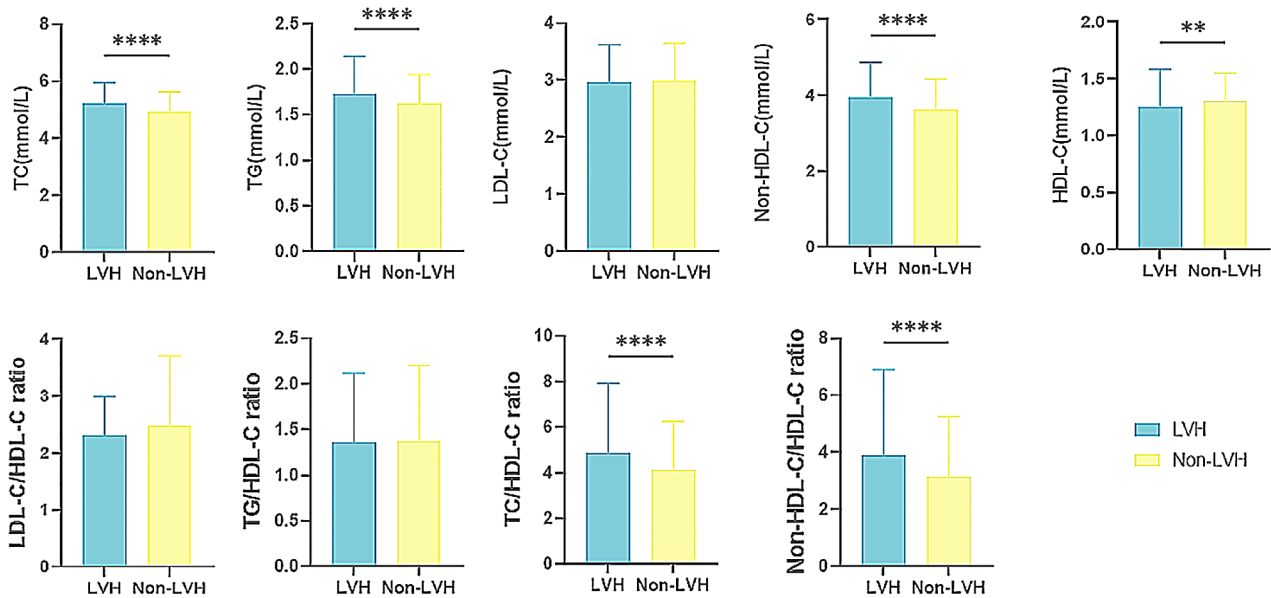
Fig. 2 Lipid levels in LVH (*n*=287) and Non-LVH (*n*=1805) groups. Data are mean ± SD. ******p<0.0001

Table 2 Correlation analysis of LVMI and other clinical variables in non-diabetes individuals

| Variables	LVMI	
	r	P value
Male	-0.2	0.358
Age	0.18	0.001
HTN	0.52	0.001
Fruits/Vegetables> 3 servings	0.01	0.93
Alcohol	0.07	0.001
Smoking	-0.09	0.001
Obese	0.03	0.24
Income	-0.60	0.001
EGFR Stages > II	0.35	0.001
Dyslipidemia	-0.60	0.001
Male WC	0.05	0.04
Female WC	0.01	0.54
HbA1c	-1.41	0.16
Creatinine	-0.18	0.86
WC	-2.20	0.03
TC	0.57	0.001
LDL-C	-0.35	0.001
Non-HDL-C	0.59	0.001
HDL-C	-0.44	0.001
TG	0.72	0.001
TG/HDL-C	-0.50	0.001
TC/HDL-C	0.44	0.001
Non-HDL-C/HDL-C	0.31	0.001
LDL-C/HDL-C	-0.60	0.001

Abbreviations HTN, Hypertension; WC, Circumference; TC, total cholesterol; TG, triglyceride, HDL-C, high-density lipoprotein cholesterol, LDL-C, low-density lipoprotein cholesterol. *P*<0.05 is considered as statistically significant

no significant differences regarding LDL-C, TG/HDL-C, and LDL-C/HDL-C.

Correlation between LVH and potential risk factors

The Spearman-correlation analysis revealed positive correlations between LVMI and age (*r*=0.18, *P*=0.001), alcohol consumption history(*r*=0.07, *P*=0.001), hypertension history(*r*=0.52, *P*=0.001), eGFR(*r*=0.35, *P*=0.001), TC(*r*=0.57, *P*=0.001), Non-HDL-C (*r*=0.59, *P*=0.001), TG(*r*=0.72, *P*=0.001), TC/HDL-C ratio(*r*=0.44, *P*=0.001), and Non-HDL-C/HDL-C ratio(*r*=0.31, *P*=0.001). Conversely, negative correlations were observed with income(*r*=-0.6, *P*=0.001), smoking history(*r*=-0.09, *P*=0.001), waist circumstance(*r*=-2.2, *P*=0.03), LDL-C(*r*=-0.35, *P*=0.001), HDL-C(*r*=-0.44, *P*=0.001), TG/HDL-C (*r*=-0.5, *P*=0.001), and LDL-C/HDL-C(*r*=-0.6, *P*=0.001) (Table 2).

PCA was conducted with all continuous variables to reduce the dimensionality potentially influencing LVH; the results indicated that LVH and non-LVH populations show significant separation in the principal component space. The first principal component explained 36.8% of the variance in whether LVH occurs, suggesting that this component was the most important in distinguishing between the two groups (Fig. 3A). In the first principal component, the variables making significant contributions were the TC/HDL-C ratio, Non-HDL-C/HDL-C ratio, TG/HDL-C ratio, LDL-C/HDL-C ratio, and Non-HDL-C, each contributing around 15%. The contribution of HDL-C was slightly lower but still close to 15%, indicating it also significantly contributed to the first principal component. The contributions of LDL-C were relatively small. These results suggested lipid profile were

A

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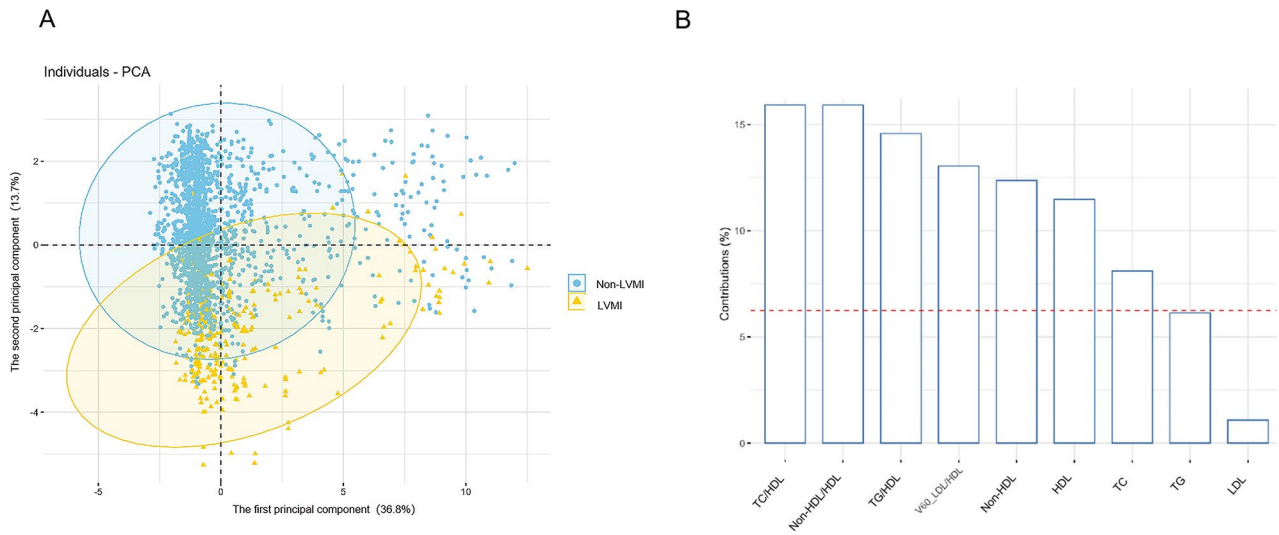
Fig. 3 PCA in LVH and Non LVH groups
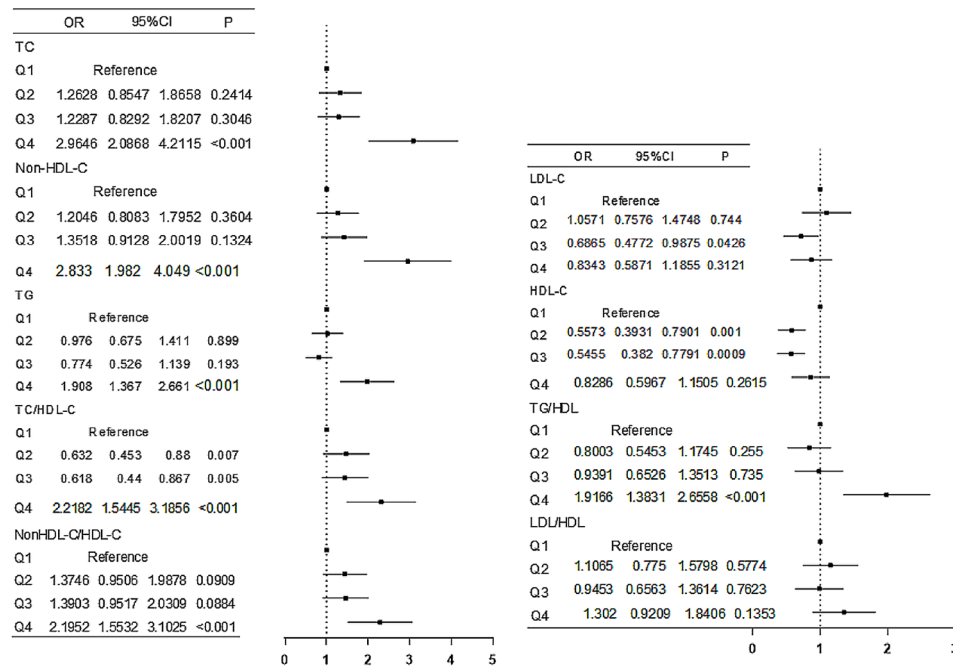
Fig. 4 Relationship between the levels of lipids and the prevalence of LVH (Unadjusted model)

essential in distinguishing individuals with and without LVH (Fig. 3B).

Relationship between the levels of lipids and LVH

Univariate analysis, correlation analysis, and PCA results showed a moderate correlation between lipid levels and LVMI. Participants were categorized into quartiles based on their lipid levels. In the unadjusted model, participants in the highest quartile, as compared to those in the lowest quartile, exhibited a higher risk of LVH in the TC, Non-HDL-C, TG, TC/HDL-C ratio, Non-HDL-C/HDL-C ratio, and TG/HDL-C ratio groups (TC, OR=2.9646,

95%CI: 2.0868–4.2115; Non-HDL-C, OR=2.833, 95%CI: 1.982–4.049; TG, OR: 1.908, 95%CI: 1.367–2.661; TC/HDL-C, OR=2.2182, 95%CI: 1.5445–3.1856; Non-HDL-C/HDL-C, OR=2.1952, 95%CI: 1.5532–3.1025; TG/HDL-C, OR=1.9166, 95%CI: 1.3831–2.6558) (Fig. 4). Adjusting for confounding factors: Model I was adjusted for age and income (Fig. 5); Model II further adjusted for hypertension, alcohol history, and smoking history (Fig. 6); Model III additionally adjusted for waist circumference and eGFR (Fig. 7). The results of Model III indicated that TC (OR=2.773, 95%CI: 1.805–4.26), Non-HDL-C (OR=2.74, 95%CI: 1.7723–4.236), TC/HDL-C
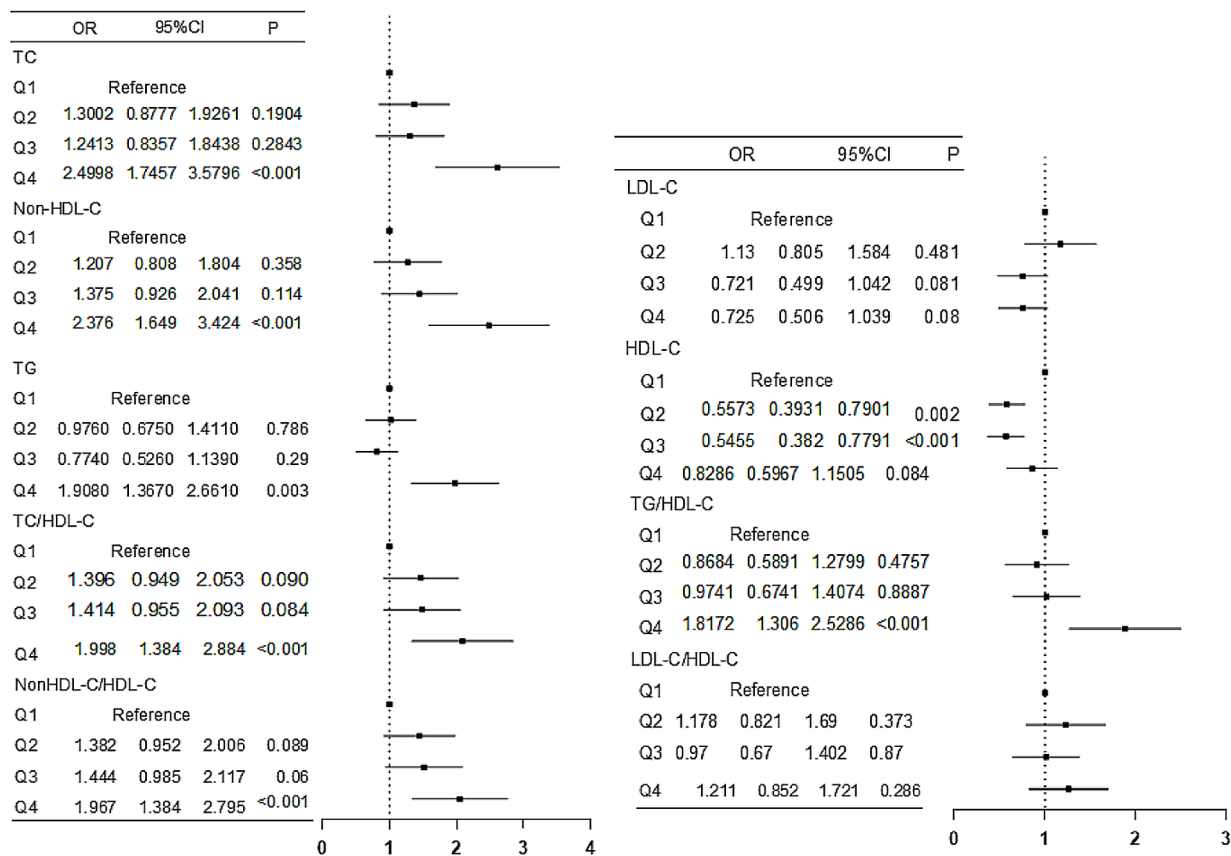
Fig. 5 Relationship between the levels of lipids and the prevalence of LVH (Model 1)

ratio (OR=2.237, 95%CI: 1.445–3.463), Non-HDL-C/HDL-C ratio (OR=2.357, 95%CI: 1.548–3.588), TG/HDL-C ratio (OR=1.513, 95%CI: 1.02–2.245) adjusted for confounding factors, the risk of LVH in participants in the highest quartile is increased compared to those in the lowest quartile.

Discrimination of lipids on LVH

The ROC curve analysis revealed the predictive ability of blood lipid profile for LVH (Fig. 8), with Non-HDL-C (AUC=0.6109) exhibiting the highest area under the curve, followed by TC (AUC=0.6084), and the weakest predictor being TG/HDL-C ratio (AUC=0.5787). Youden's index was utilized to identify the best threshold value of 4.9 for TC and 3.575 for Non-HDL-C in predicting LVH, with a specificity of 47.74% and a sensitivity of 74.4%, a specificity of 50.52% and a sensitivity of 70.86%, respectively (Table 3).

Sensibility analysis

After further adjustment for sex and HbA1c, the results remained consistent with those before adjustment (Supplement 1). Similarly, after adjusting for BMI, the results remained consistent (Supplement 2). Upon stratification

analysis for hypertension, the results were consistent among hypertensive patients (Supplement 3).

Discussion

This study is the first to delve into the relationship between lipid components such as TC, Non-HDL-C, TG, TC/HDL-C ratio, Non-HDL-C/HDL-C ratio, TG/HDL-C ratio, and LVH in non-diabetic populations. Our result showed that Non-HDL-C, TC, TC/HDL-C ratio, Non-HDL-C/HDL-C ratio, and TG/HDL-C ratio can serve as independent risk factors for predicting the preservation of LVH in non-diabetic individuals. Additionally, Non-HDL-C and TC can be utilized as predictive markers due to their higher area beneath the curve. Furthermore, this correlation exhibited notable strength and consistency across correction groups for gender and HbA1c, BMI correction group, and stratified analysis of hypertension.

LVH, characterized by thickening of the myocardial wall of the left ventricle or dilation of the left ventricular chamber, poses a significant risk, with individuals displaying consistently high LVH showing the highest mortality risk [16]. Early detection and intervention and reducing the independent risk factors for LVH can serve as early preventive and intervention measures for the disease [18, 20]. Hypertension, diabetes, and renal
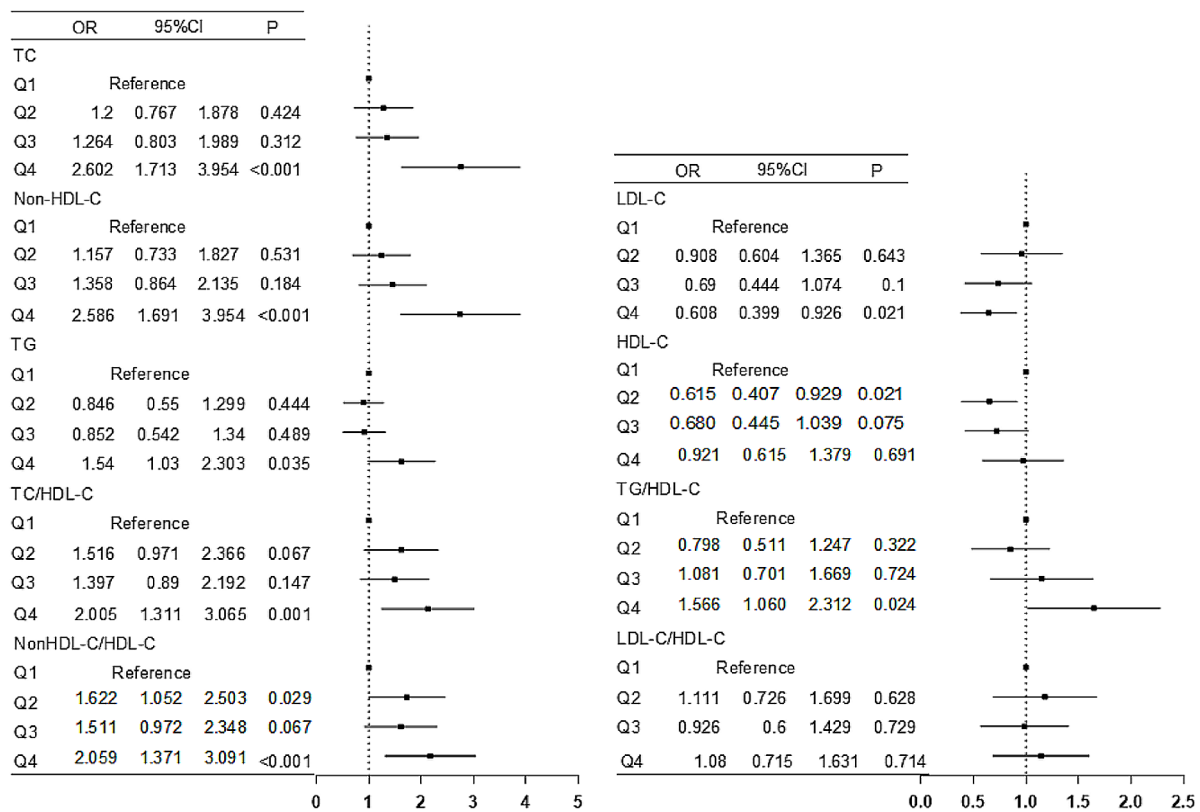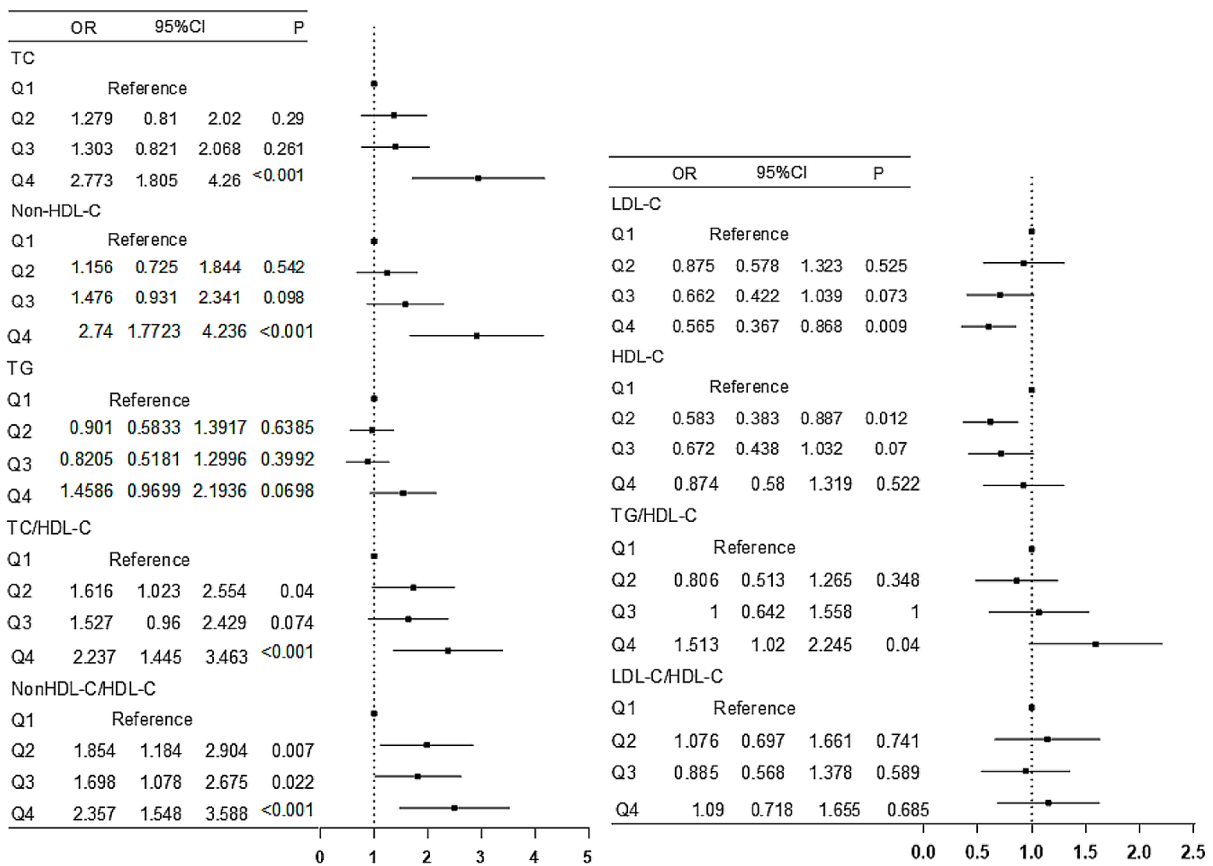
Fig. 6 Relationship between the levels of lipids and the prevalence of LVH (Model 2)

insufficiency are major known risk factors for LVH [21]. A study in Mauritius found a higher prevalence of LVH among individuals with hypertension [22]. In pre-dialysis patients, the ratio of HDL-C to c-reactive protein and LVMI have been linked to baseline renal function and have shown an independent correlation with the progression of chronic kidney disease [23]. Additionally, African Americans with non-glomerular chronic kidney disease showed a higher prevalence of ambulatory hypertension and increased LVMI [24].

LVH is characterized by progressive impairment in β-oxidation, resulting in dysregulated fatty acid oxidation and significant metabolic remodeling of the heart [25]. Several studies suggest that alterations in lipid profile play a role in the pathophysiology of LVH. However, a consensus regarding the relationship between dyslipidemia and LVH is yet to be reached. Elevated HDL-C levels in mice models have been shown to impede LVH progression, evidenced by reduced left ventricular weight in mice fed a high-fat diet [26]. Change in leptin-signal transducer and activator of transcription three pathway signaling is related to the occurrence of LVH in Rats [27]. While drug interventions targeting lipid profile have shown promise in alleviating LVH by reducing TC, TG, and LDL-C while increasing HDL-C, the exact relationship between lipid regulation and LVH improvement remains

uncertain [28]. Evidence suggests that individuals with dyslipidemia, defined by non-traditional lipid indices such as high atherosclerosis index and high TC/HDL-C, along with hypertension, are at higher risk of developing LVH [29]. A 7-year longitudinal and mediation study on increased lipids with risk of worsening cardiac damage in 1595 adolescents recognized that increased TC, Non-HDL-C, and TG were associated with worsening diastolic function and higher LV filling pressure [30]. In non-diabetic patients, the elevated TG in males with hypertension is independently associated with LVH [31]. This may be related to high TG causing myocardial steatosis, leading to left ventricular hypertrophy [32]. Increased TG/HDL-C ratio in obese children has been associated with eccentric LVH development [33]. The uncertainty of the effect of blood lipids on LVH may be related to insulin resistance and abnormal blood pressure caused by metabolic syndrome caused by dyslipidemia [27].

Furthermore, the most significant discovery in this study is that a comprehensive lipid profile has varying degrees of impact on the increase in LVH in the non-diabetic population. While lipids are affected by factors such as glucose metabolism, insulin resistance, and obesity, their influence on LVH remains notable [34]. Meanwhile, due to the variety of lipid types, they become an undeniable independent influencing factor in LVH. This study

Fig. 7 Relationship between the levels of lipids and the prevalence of LVH (Model 3)
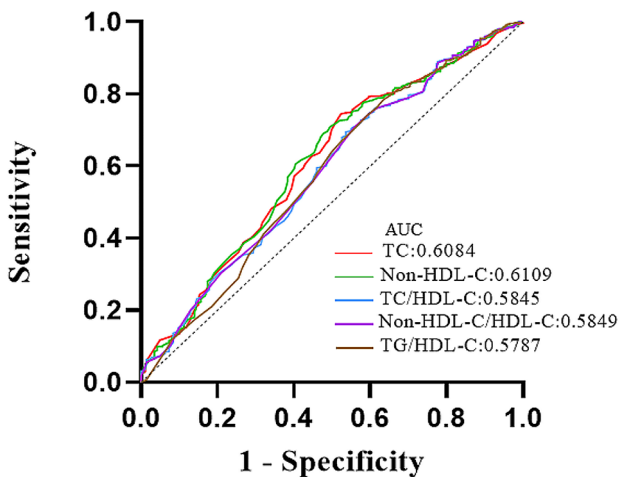
Fig. 8 Receiver operating characters (ROC) Curve and area under eclipse

provides new evidence for the direct correlation between blood lipids and LVH and a new direction for managing and treating this disease.

Study strengths and limitations
This study is the first to investigate the risk determinant linked to LVH in a non-diabetic Sierra Leone population.

PCA was used to determine the relative importance of blood lipids in the study, an innovation. Multiple corrections were performed to remove confounding factors and demonstrate the correlation between lipids and LVH. But it has some limitations. First, data focuses on individuals in Africa, which is constricted. It is better to enlarge samples and include other regions. Second, if participants are tracked and followed up, monitoring blood lipids will provide a more comprehensive understanding of the impact of blood lipids on LVH. Finally, conducting lipidomics analysis on the population to focus on the effect of the lipid profile on LVH would be even more convincing.

Conclusion
Lipid profile, represented by TC, Non-HDL-C, TC/HDL-C, Non-HDL-C/HDL-C ratio, and TG/HDL-C ratio, are independent risk factors of LVH in non-diabetic people. In addition, Non-HDL-C, with a cutoff value of 3.575, and TC, with a cutoff value of 4.9, were found to be an essential indicator for predicting the prevalence LVH disease with specific predictive values.

Table 3 Areas under the receiver operating characteristic curves (ROC) for lipid profiles in LVH

Variables	AUC (95% CI)	Best threshold	Specificity	Sensitivity	Youden index
TC (mmol/L)	0.6084 (0.5726–0.6442)	4.9	0.4774	0.744	0.2214
Non-HDL-C (mmol/L)	0.6109 (0.5750–0.6469)	3.575	0.5052	0.7086	0.2138
TC/HDL-C ratio	0.5845 (0.5483–0.6207)	3.715	0.4634	0.6947	0.1581
Non-HDL-C/HDL-C ratio	0.5849 (0.5488–0.6211)	2.75	0.4425	0.7075	0.15
TG/HDL-C ratio	0.5787 (0.5413–0.6162)	1.225	0.3659	0.7834	0.1493

Abbreviations AUC, the area under the curve; ROC, receiver operating characteristic; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol. $P < 0.05$ is considered as statistically significant

Abbreviations
BMI   Body mass index
eGFR   Estimated glomerular filtration rate
FPG   Fasting blood glucose
HbA1c   Glycated hemoglobin
HDL   C-High-density lipoprotein
HF   Heart failure
LDL   C-Low-density lipoprotein cholesterol
LVH   Left ventricular hypertrophy
LVMI   Left Ventricular Mass Index
Non   HDL-C-Non-high-density lipoprotein
PCA   Principal component analysis
TC   Total cholesterols
TG   Triglycerides

Supplementary Information
The online version contains supplementary material available at https://doi.org/10.1186/s12944-024-02232-8.

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Author contributions
YX contributed to conceptualization, data curation, formal analysis, visualization, and writing original draft. YCJ contributed to validation, visualization, and software. LX contributed to investigation, WZ and ZZ contributed to methodology. YQ contributed to project administration, resources, supervision and writing review and editing. HK and SY contributed to funding acquisition and final writing review and editing.

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

Declarations

Ethics approval and consent to participate
In the previously published article, all participants signed informed consent forms.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Endocrine and Metabolic Diseases, Fourth Affiliated Hospital of Harbin Medical University, Harbin 150001, China
[2]Department of Endocrinology, First Affiliated Hospital of Harbin Medical University, Harbin 150001, China
[3]Centenary Institute of Cancer Medicine and Cell biology, The University of Sydney, Sydney, NSW 2050, Australia
[4]School of Medical Sciences, Faculty of Medicine and Health, The University of Sydney, Sydney, NSW 2006, Australia
[5]Charles Perkins Centre, The University of Sydney, Sydney, NSW 2006, Australia
[6]Faculty of health and medicine, Sanya University, Sanya 572000, China
[7]Harbin Medical University, Harbin 150081, China

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