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The relationship between the ratio of triglyceride to high-density lipoprotein cholesterol and left ventricular hypertrophy in Chinese hypertension adults

Jiabin Hu^{1,2,3}, Shuang Cai^{2,4}, Zili Wan⁵, Bokai Cheng^{1,2}, Jin Sun^{1,2}, Bangguo Qin^{1,2}, Qiligeer Bao^{1,2}, Ting Huang⁴, Rongxia Chen^{4,6}, Shuxia Wang^{1,2}✉ & Ping Zhu^{1,2}✉

Left ventricular hypertrophy (LVH) is a major cardiac complication of hypertension. The ratio of triglycerides (TG) to high-density lipoprotein cholesterol (HDL-C) (TG/HDL-C) has been identified as a biomarker of insulin resistance and a predictor of atherosclerosis. We aimed to investigate the relationship between TG/HDL-C and LVH in hypertensive patients among the Han Chinese. Our community-based cross-sectional study recruited 4552 patients with hypertension. LVH was diagnosed by transthoracic echocardiography in these patients based on a criterion of left ventricular mass index (LVMI) over 49.2 g/m^{2.7} in men and 46.7 g/m^{2.7} in women. The independent association between the TG/HDL-C ratio quartiles and LVH prevalence was analyzed by logistic regression models. The ratio of TG/HDL-C was higher (1.27 ± 1.26 , 1.15 ± 1.07 , $P = 0.001$) in LVH patients. The prevalence of LVH with the first quartile of TG/HDL-C (Q1: < 0.61), second (Q2: 0.61 ~ 0.91), the third (Q3: 0.92 ~ 1.41), and the highest quartile (Q4: > 1.41) was 36.1%, 42%, 42.6%, 44.9%, respectively. Logistic regression analysis suggested that the ratio of TG/HDL-C was independently correlated with the risk of LVH with adjustment of confounding factors. The correlation was more significant in female patients rather than males. Compared to the first quartile of TG/HDL-C (Q1), the odds ratios (ORs) and their 95% confidence intervals (CIs) for LVH in the increasing quartiles (Q2-Q4) were 1.21(1.01–1.45), 1.28(1.07–1.54), and 1.48(1.23–1.78), respectively. Similar results were also observed in the subjects younger than 60 years with coronary heart disease (CAD). The ratio of TG/HDL-C may be an independent risk factor of LVH in Han Chinese with hypertension.

Keywords The ratio of triglyceride to high-density lipoprotein cholesterol, Left ventricular hypertrophy, Hypertensive patients

Abbreviations

LVH	Left ventricular hypertrophy
TG/HDL-C	triglycerides to-high-density lipoprotein cholesterol
LVM	Left ventricular mass
LVMI	left ventricular mass indexed
TG	triglyceride
HDL-C	high-density lipoprotein cholesterol
CVD	cardiovascular disease
CAD	Coronary heart disease
IVS	Interventricular septum

¹Department of Geriatrics, the second Medical Center & National Clinical Research Center for Geriatric Diseases, Chinese PLA General Hospital, Beijing 100853, China. ²Medical School of Chinese PLA, Beijing 100853, China.

³Health Service Department of the Guard Bureau of the Joint Staff Department, Beijing 100853, China. ⁴Outpatient Department of the 51st Retired Cadre Rest Home, Haidian Garrison District, Beijing 100036, China. ⁵Capital Medical University, Beijing 100069, China. ⁶Department of Geriatrics, the 2nd Medical Center, Chinese PLA General Hospital, 28 Fuxing Road, Beijing 100853, China. ✉email: wangsx301@163.com; zhuping301@139.com

PW	Posterior wall
LVEDD	Left ventricular end-diastolic diameter
IVST	Interventricular septal thickness
PWT	Posterior wall thickness
RWT	Relative wall thickness
UCG	Ultrasonic cardiogram
FPG	Fasting plasma glucose
TC	Total cholesterol
LDL-C	Low-density lipoprotein cholesterol
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
SUA	Serum uric acid
Scr	Serum creatinine
BUN	Blood urea nitrogen
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
WHR	Waist-to-Hip Ratio

LVH is a severe cardiac disease characterized with ventricular wall thickness and remodeling^{1,2} that further lead to heart failure³. LVH often occurs in patients with hypertension associated with hyperlipidemia and coronary atherosclerosis. Thus, adequate clinical cares in hyperlipidemia would reduce the risk of cardiac morbidity and mortality with hypertension⁴.

Indeed, coronary heart disease is often associated with increased plasma TG levels and decreased plasma HDL-C levels^{5,6}. The ratio of TG/HDL-C can be used as an independent predictor of CVD risk⁷⁻⁹ and is considered as a simple marker of atherosclerotic dyslipidemia and insulin resistance¹⁰⁻¹⁵. Insulin resistance is an important pathway leading to LVH¹⁶⁻¹⁸. However, the relationship between the ratio of TG/HDL-C and LVH in patients with hypertension has yet to be elucidated.

We speculated that the ratio of TG/HDL-C could be used as an effective indicator for the early identification of LVH risk in patients with hypertension. We therefore conducted a cross-sectional study to define the relationship between TG/HDL-C and LVH and evaluate whether other related factors impact this relationship.

Methods

Subjects

The present protocol has been reviewed and approved by the ethical committees of the Fuwai Hospital and local hospitals. The research procedures obeyed the ethically normative criteria. All the subjects have provided written informed consent and all investigators have been trained at the Cardiovascular Institute, Chinese Academy of Medical Sciences (Beijing, China) before the experiments. Throughout the research process, the researchers adhered strictly to relevant guidelines and regulations.

A cross-sectional study was conducted in rural Han Chinese residents aged 40–75 at Xinyang County, central China, from 2004 to 2005 with the utilization of a multi-stage cluster sampling method¹⁹⁻²¹. Total 13,444 subjects including 5270 males and 8174 females completed the survey, with a response rate of 84.9%.

5421 of the subjects have hypertension defined as diastolic blood pressure ≥ 90 mmHg (DBP) and systolic blood pressure ≥ 140 mmHg (SBP). Blood pressure was measured by trained professionals using a standard mercury sphygmomanometer. All participants were advised not to smoke, drink coffee/tea or alcohol before the measurements of blood pressure. Participants had three readings of sitting posture with 30 s intervals in between. LVM was measured by echocardiography in 4805 patients. Due to lack of blood tests, 253 subjects were excluded. Thus, eventually, total 4552 patients with complete clinical, biochemical, and echocardiographic data were recruited for the current study.

Echocardiography measurements

Transthoracic echocardiography was performed by two echocardiographers who have been trained with the echocardiographic protocol at the Cardiovascular Institute, Chinese Academy of Medical Sciences¹. M-mode, 2-dimensional (2D) and color Doppler recordings from the parasternal long-axis and short-axis windows as well as 2D and color Doppler evaluations from the apical window to yield 2-, 3- and 4-chamber images with an HP 5500 (Phillips Medical System, Boston, MA, USA) or an HDI 3000 (A TL, Bothell, W A, USA). We also measured end-systolic diameter, end-diastolic diameter, septal wall thickness, and posterior wall thickness at the end of the diastole. According to the American Society of Echocardiography recommendations, LV internal dimensions and septal and posterior wall thicknesses were measured in up to three cardiac cycles at end-diastole and end-systole. A cardiologist read the images without knowing the subjects' clinical data.

LVM was calculated by using the following equation:

$$0.8 \times 10.04[(IVS + LVEDD + PW)^3 - LVEDD^3] \times 0.6$$

IVS is the interventricular septum, PW is the posterior wall, and LVEDD is left ventricular end-diastolic diameter. LVM was divided by height^{2,7} to obtain LVMI (LVMI_{h2.7}). LVH was diagnosed with the criteria of LVM ≥ 49.2 gm^{-2,7} in males and 46.7 gm^{-2,7} in females.

Covariate measurements and definitions

Covariates including gender, age, and lifestyle were collected through a face-to-face interview by trained nurses. In the morning, blood samples were collected from the anterior cubital vein in all the patients following ~ 12 h

fasting. Fasting plasma glucose (FPG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), HDL-C, TG, electrolytes, and renal function indicators were quantified and analyzed with an autoanalyzer.

Statistical analysis

SPSS (Statistical Package for the Social Sciences) software version 26 (SPSS Inc., Chicago, IL, USA) was used for data management and statistical analysis. The total experimental population was divided into four groups according to the baseline TG/HDL-C ratio (Q1: < 0.61, Q2: 0.61~0.91, Q3: 0.92~1.41, Q4: >1.41), and the parameters were compared among these four groups. Data are reported as mean \pm standard deviation for continuous variables and as percentages for categorical variables. All participants were stratified by quartiles of TG/HDL-C, baseline differences in clinical variables and echocardiographic data among the groups using analysis of variance (ANOVA) for continuous variables, and Chi-squared test for categorical variables. The logistic regression analysis was used to calculate ORs and their 95% CIs. Using binary logistic regression model and multivariate analysis to evaluate the relationship between TG/HDL-C categories and LVH. Sequential models were developed. Model 1 was no adjusted variable. In Model 2, the analysis was adjusted for age, sex. In addition to the confounders analyzed in Model 2, Model 3 included the admission of smoking, drinking, SBP, DBP, waist-to-hip ratio (WHR), stroke. Model 4 added TC, LDL-C, serum uric acid (SUA), FPG, serum creatinine (SCr), blood urea nitrogen (BUN), alanine aminotransferase (ALT), aspartate aminotransferase (AST) based on Model 3. Further stratified analysis was performed by gender and age groups to obtain OR and 95% CI for LVH and TG/HDL-C in each subgroup. The differences were considered significant if a 2-tailed *P* value is less than 0.05.

Results

Clinical, biochemical characteristics and echocardiography data of patients by the TG/HDL-C ratio

Clinical and biochemical characteristics, and echocardiographic parameters in all participants were summarized in Table 1 based on quartiles of the TG/HDL-C ratio. All the parameters including DBP, TG, TC, LDL-C, FPG, SUA, SCr, ALT, WHR, stroke and CAD were significantly increased in parallel with the increase of TG/HDL-C from the first to the fourth quartile (Q1-Q4) (all *P* for trend < 0.05). On the other hand, there was an inverse association between HDL-C and TG/HDL-C ratio. In echocardiographic parameters, IVST, LVM, and LVM/H2.7 parameters were significantly increased with TG/HDL-C from Q1-Q4 (all *P* for trend < 0.001). Compared with patients with the lowest quartile of TG/HDL-C, the prevalence of LVH was higher for patients with higher quartile of the TG/HDL-C ratio. The prevalence of LVH in patients among those groups was 36.1%、42%、42.6%、44.9%, respectively.

Association between TG/HDL-C and LVH

Logistic regression analysis was performed to determine the independent association between TG/HDL-C and the risk of LVH. As shown in Table 2, quartiles of TG/HDL-C were associated with increased risk of LVH in univariate analysis (model 1). After adjustment for age and sex (model 2) and further for smoking, drinking, SBP, DBP, WHR, and stroke (model 3), the ORs for LVH remained progressively increased across quartiles of TG/HDL-C. Finally, TG/HDL-C was still independently associated with the risk of LVH after further controlling for TC, LDL-C, SUA, FPG, SCr, BUN, ALT, AST (model 4). Compared with the first quartile (Q1), the ORs (95%CI) for LVH in the increasing quartiles of TG/HDL-C (Q2-Q4) were 1.21(1.01–1.45), 1.28(1.07–1.54), and 1.48(1.23–1.78), respectively, and we found that LVH risk increased with higher quartiles of TG/HDL-C in all models (*P* for trend < 0.001).

We further performed a stratified analysis of the correlation between LVH, and TG/HDL-C categories in Fig. 1. The relationship between TG/HDL-C and LVH risk was significantly different by sex, and this association was significantly higher in women compared to men. After adjustment of several covariates, women still had a strong association. In the group younger than 60 years, the highest quartile was significantly associated with LVH. This association with LVH was not significant in those patients older than 60 years. As the ratio of TG/HDL-C is related to the development of atherosclerosis, the relationship between TG/HDL-C and LVH was respectively detected in CAD and non-CAD groups. In the non-CAD group, there was no significant correlation between TG/HDL-C and LVH. However, the third and highest quartile of TG/HDL-C was significantly associated with LVH in the CAD group. In addition, we performed an interaction analysis between the TG/HDL-C categories and each stratification factor, and found that there was no interaction between them.

Discussion

We performed a cross-sectional study which analyzed the association between TG/HDL-C and LVH risk in hypertensive patients. We found that the prevalence of LVH was significantly increased accompanied with the increase of TG/HDL-C and that there was a significant independent positive association between LVH and TG/HDL-C, regardless of adjustments of potential confounders. Thus, the ratio of TG/HDL-C can be used to further identify patients who have high risk of LVH in hypertensive populations.

LVH refers to cardiac enlargement characterized with hypertrophy of cardiomyocytes and proliferation of fibrous connective tissue. LVH is one of the most common cardiac damages associated with hypertension and it increases the risk of heart failure^{22–24}. However, so far, the pathogenesis of LVH is not entirely understood and it is probably related to age, sex, weight, race, genetic predisposition, metabolic status (such as insulin resistance) and hypertension etc. Leptin also increases the activity of extracellular matrix metalloproteinases and other enzymes which may induce interstitial fibrosis and participate in inflammatory response during the development of LVH²⁵.

Characteristics	Q1 (< 0.61)	Q2(0.61–0.91)	Q3(0.92–1.41)	Q4(>1.41)	P for trend
n	1117	1173	1133	1129	
Age (years)	58.1±8.5	58.4±8.3	58.4±8.4	57.6±8.0	0.049
Males (%)	446(39.4)	342(30.2)	373(32.8)	355(31.3)	<0.001
Smokers (%)	76(6.7)	57(5.0)	59(5.2)	60(5.3)	0.017
Drinkers (%)	53(4.7)	52(4.6)	52(4.6)	54(4.8)	0.005
Pulse(times/min)	71.9±11.9	71.8±12.0	73.6±12.6 ^{a,b}	73.6±12.3 ^{a,b}	<0.001
SBP (mmHg)	162.2±24.4	163.7±24.4	164.2±24.5	164.0±24.6	0.216
DBP (mmHg)	96.2±12.6	96.8±12.1	97.2±12.8	97.9±12.9 ^a	0.015
ALT (mmol/L)	18.5±13.2	18.9±12.4	19.9±13.7	21.6±13.8 ^{a,b,c}	<0.001
AST (mmol/L)	28.9±18.1	27.2±13.7 ^a	27.6±13.1 ^{a,b}	27.8±14.4 ^{a,b}	0.036
FPG (mmol/L)	5.3±1.2	5.4±1.5	5.6±1.7 ^{a,b}	6.0±2.2 ^{a,b}	<0.001
HDL-C(mmol/L)	1.9±0.3 ^{a,b,c}	1.6±0.3 ^{a,b,c}	1.5±0.2 ^{a,b,c}	1.3±0.2 ^{a,b,c}	<0.001
LDL-C(mmol/L)	2.9±0.8 ^{a,b,c}	3.1±0.8 ^{a,b,c}	3.3±0.9 ^{a,b,c}	3.2±0.9 ^{a,b}	<0.001
TC (mmol/L)	5.3±1.0 ^{a,c}	5.4±1.0 ^{a,b,c}	5.6±1.1 ^{a,b,c}	5.8±1.2 ^{a,b,c}	<0.001
TG (mmol/L)	0.8±0.2	1.2±0.2	1.6±0.3	3.1±1.8	<0.001
SUA(μmol/L)	269.8±78.2 ^{a,b,c}	281.5±81.6 ^{a,b,c}	298.8±95.2 ^{a,b,c}	321.2±92.8 ^{a,b,c}	<0.001
BUN (mmol/L)	5.76±1.85 ^{b,c}	5.50±1.64 ^a	5.33±1.69 ^a	5.29±2.00 ^{a,b}	<0.001
SCr (μmol/L)	65.8±24.8 ^{a,b,c}	64.5±20.4 ^{a,b,c}	66.8±24.7 ^{a,b,c}	67.9±32.6 ^{a,c}	0.014
TG/HDL-C	0.46±0.10 ^{a,b,c}	0.75±0.09 ^{a,b,c}	1.13±0.14 ^{a,b,c}	2.46±1.72 ^{a,b,c}	<0.001
WHR	0.85±0.07 ^{a,b,c}	0.87±0.22 ^{a,b,c}	0.87±0.06 ^{a,b,c}	0.89±0.06 ^{a,b,c}	<0.001
Stroke (%)	90(8.0)	108(9.7)	139(12.4)	130(11.6)	0.003
CAD (%)	72(6.3)	95(8.4)	97(8.6)	151(13.3)	<0.001
UCG indices					
IVST (cm)	0.98±0.16 ^c	0.99±0.16	1.00±0.16 ^a	1.01±0.16 ^{a,b}	<0.001
LVEDD (cm)	4.54±0.51	4.54±0.50	4.55±0.52	4.59±0.51	0.064
PWT (cm)	0.96±0.14	0.97±0.13	0.98±0.14	0.98±0.13 ^a	0.006
RWT	0.43±0.08	0.43±0.07	0.43±0.07	0.43±0.07	0.387
LVM (g)	154.8±45.0 ^c	156.5±42.2	160.0±45.2 ^a	163.3±43.5 ^{a,b}	<0.001
LVM/H2.7(g/h2.7)	45.2±12.7 ^c	46.4±12.1	46.7±12.8 ^a	47.4±12.4 ^a	<0.001
LVH	411(36.1%)	477(42%)	485(42.6%)	511(44.9%)	<0.001

Table 1. Clinical, biochemical characteristics and echocardiogram parameters of the study subjects according to quartiles of TG/HDL-C. Abbreviations: SBP, Systolic Blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; TC, total cholesterol; HDL-C, high-density lipoprotein; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; TG/HDL-C, TG to HDL ratio; SUA, serum uric acid; SCr, serum creatinine; BUN, serum urea nitrogen; ALT, alanine aminotransferase; AST, aspartate aminotransferase; WHR, Waist-to-Hip Ratio; CAD, coronary heart disease; IVST, interventricular septal thickness; LVEDD, left ventricular end diastolic diameter; LVH, left ventricle hypertrophy; LVM, left ventricular mass; PWT, posterior wall thickness; RWT, relative wall thickness; UCG, ultrasonic cardiogram. Data are presented as mean ± standard deviation or median (interquartile range) for continuous variables, and number (percentage) for categorical Variables. ^aP<0.05 compared with Quartile 1. ^bP<0.05 compared with Quartile 2. ^cP<0.05 compared with Quartile 3.

Baseline TG/HDL-C	Model 1	P-value	Model 2	P-value	Model 3	P-value	Model 4	P-value
Q1 (< 0.61)	1 (References)		1 (References)		1 (References)		1 (References)	
Q2(0.61–0.91)	1.28(1.08–1.52)	0.004	1.23(1.04–1.46)	0.019	1.21(1.01–1.44)	0.034	1.21(1.01–1.45)	0.033
Q3(0.92–1.41)	1.31(1.11–1.56)	0.002	1.27(1.07–1.51)	0.006	1.24(1.04–1.48)	0.015	1.28(1.07–1.54)	0.007
Q4(>1.41)	1.44(1.22–1.71)	<0.001	1.42(1.20–1.69)	<0.001	1.38(1.16–1.64)	<0.001	1.48(1.23–1.78)	<0.001
P for trend	<0.001		<0.001		<0.001		<0.001	

Table 2. ORs and 95% CIs of TG/HDL-C ratio and LVH in all subjects. Abbreviations: 95% CIs, 95% confidence intervals; ORs, odds ratios. Model 1: no adjusted variable. Model 2: Adjusted for sex, age. Model 3: Model 2 and further adjusted for smoking, drinking, SBP, DBP, WHR, stroke. Model 4: Model 3 and further adjusted for TC, LDL-C, SUA, FPG, SCr, UREA, ALT, AST.

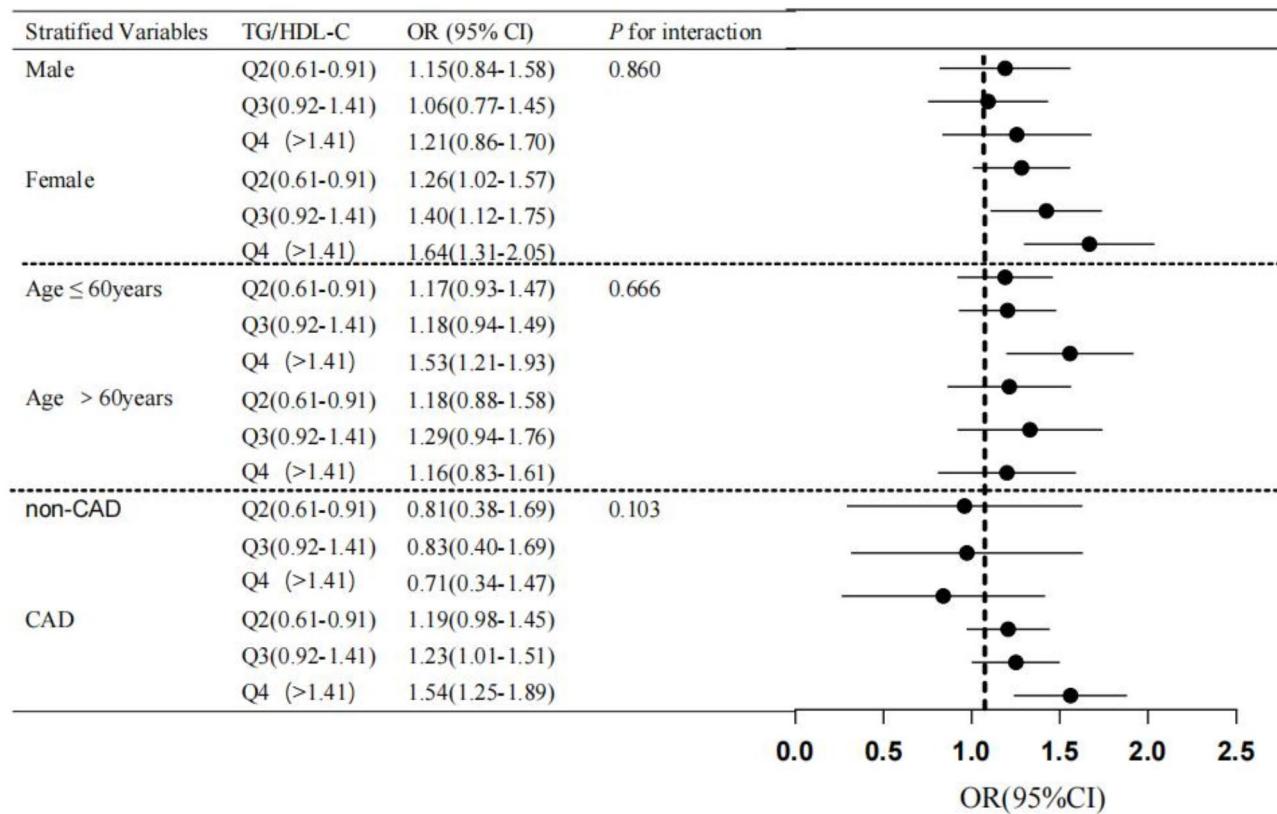


Fig. 1. Odds ratio for LVH by TG/HDL in stratification analysis. Abbreviations: 95% CI, 95% confidence interval; OR, odds ratio. Adjusted: sex, age, smoking, drinking, SBP, DBP, WHR, stroke, TC, LDL-C, SUA, FPG, SCr, UREA, ALT, AST, except for the stratified variable.

Lipid dysfunction accelerates atherosclerosis, resulting in decreased compliance and dilation of greater arteries. The pathological procedure leads to a stress on the left ventricular wall during the systolic period and prolongs the left ventricular ejection time, further contributing to the development of LVH. TG is involved in the synthesis of cholesterol and cholesterol esters and thus it is the major lipid component leading to atherosclerosis. Triglycerides appear to be causative of myocardial remodeling by increasing LV mass, suggesting that it influences the development of cardiovascular disease not only by atherosclerosis but also by causing adverse alterations in cardiac structure and function²⁶. Recent studies have shown that high TG levels can lead to increased oxidative stress and inflammation, which in turn can contribute to LVH^{26,27}. HDL-C is an anti-atherosclerotic lipoprotein that is negatively correlated with the risk of cardiovascular and cerebrovascular diseases. Previous studies indicated that the levels of serum HDL-C are usually lower in patients with hypertriglyceridemia. The low levels of serum HDL-C are probably due to the unstable structure of HDL-C particles rich in TG. Thus, compared to other lipid components, the ratio of TG/HDL-C could better reflect the comprehensive level of lipid metabolism in patients²⁸. Recently, a study in Mexico also confirmed that the TG/HDL-C ratio is a better predictor of the prevalence of obesity and lipid triad in the Mexican population²⁹.

The ratio of TG/HDL-C is associated with concentrations of high-risk particles that predict risk of coronary heart disease^{30,31}. A high ratio of TG/HDL-C is also used to recognize concentric LVH and the elevated levels of TG/HDL-C are tightly associated with increased risk of concentric LVH^{32,33}. In our present study, we observed a significant independent positive association between TG/HDL-C and LVH in Chinese Han patients with hypertension and LVH, suggesting that the high ratio of TG/HDL-C contributes to development of LVH in Chinese hypertensive patients. Following adjustments in all potential confounders, the group in the quartile with the highest TG/HDL-C levels is nearly 1.5 times higher chance to develop LVH than those with the lowest TG/HDL-C levels. The prevalence rates of LVH were also progressively increased across the quartiles of TG/HDL-C (36.1, 42, 42.6 and 44.9%, respectively). Previous studies have shown a prevalence of 36–41% of echocardiographic LVH in all hypertensive participants, which is basically consistent with our finding³⁴.

We next conducted a stratified analysis to explore whether the association between TG/HDL-C and LVH could be influenced by gender. In fact, the association between TG/HDL-C ratio and LVH was more profound in women rather than men. A similar finding has been also reported in the associations between TG/HDL-C and nonalcoholic fatty liver disease, chronic kidney disease, and the risk of diabetes^{35–37}. The underlying mechanisms are currently unknown but one possible explanation could be the effects of sex hormones. Our current findings provide new insights into the pathophysiology of LVH in male and female patients and provide new impetus for

further research. In addition, we stratified our data by age and found that the correlation between TG/HDL-C ratio and LVH in younger patients (< 60 years) seems more significant compared to older patients (> 60 years).

Although our present study recruited a large sample size of hypertensive patients, which effectively minimizes selection bias, there are still some limitations. The cross-sectional study did not carry out prospective follow-up and intervention studies. High TG/HDL-C was independently correlated with LVH, suggesting a possible mechanism of dyslipidemia contributing to the occurrence and development of LVH. However, the causal relationship between TG/HDL-C and LVH could not be concluded eventually. The proportion of male subjects in our present study was relatively low (33.3%). Thus, the impacts of gender on the correlation between TG/HDL-C and LVH need to be further clarified by expanding the proportion of male subjects.

Conclusions

Our study suggests that the ratio of TG/HDL-C is independently correlated with LVH, especially in females, suggesting that the ratio of TG/HDL-C could be used to identify patients at a higher risk of LVH in hypertensive populations.

Data availability

The research data used to support the finding of this study are available from the corresponding authors upon request.

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Author contributions

P.Z., S.W., R.C. and J.H. designed the research. J.H., S.C., W.Z., B.C., J.S., B.Q., Q.B. and T.H. collected the data. J.H. and S.C. wrote original draft. W.Z., B.C., J.S., B.Q., Q.B. and T.H. help optimize the research and proofread the paper. R.C. and W.Z. supplements relevant information and provide guidance. All authors critically reviewed this draft. All authors approved the final draft for submission.

Declarations

Competing interests

The authors declare no competing interests.

Additional information

Correspondence and requests for materials should be addressed to S.W. or P.Z.

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