Relationship between triglyceride to high-density lipoprotein cholesterol ratio and left ventricular hypertrophy in hypertensive patients among the Han Chinese

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Research

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Abstract

Background

Left ventricular hypertrophy (LVH) is the most common target organs damage in the hypertension patients. The triglycerides to-high-density lipoprotein cholesterol (TG/HDL-C) ratio has been identified as a biomarker of insulin resistance and a predictor for atherosclerosis. Our study aimed to investigate the relationship between TG/HDL-C and LVH in hypertensive patients among the Han Chinese.

Methods

The study is a community-based cross-sectional study that included 4552 patients with hypertension and integrated clinical and echocardiographic data. Left ventricular mass (LVM) was measured by transthoracic echocardiography. LVH was diagnosed by using the criteria of left ventricular mass indexed (LVMI) over 49.2g/$m^{2.7}$ for men and 46.7g/$m^{2.7}$ for women. The independent associations between TG/HDL-C ratio quartiles and LVH prevalence was analyzed by using logistic regression models.

Results

The TG/HDL-C was higher in patients with LVH than in those without (1.27 ± 1.26, 1.15 ± 1.07, P = 0.001). The prevalence of LVH in patients 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) of TG/HDL-C was 36.1%, 42%, 42.6%, 44.9%, respectively. Logistic regression analysis suggested that after adjustment for confounding factors, TG/HDL-C was independently associated with the risk of LVH, and this association was more significant in women. Compared with the first quartile of TG/HDL-C (Q1), the odds ratios (95% confidence intervals) 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.

Conclusions

The TG/HDL-C ratio is an independent risk factor for LVH in hypertensive patients among the Han Chinese. However, longitudinal studies are required to demonstrate the predictive value of TG/HDL-C on the onset and progression of LVH over time.

Background

LVH is a phenomenon of myocardial changes characterized by ventricular wall thickening, myocardial weight increase, and myocardial remodeling[1]. LVH is an independent risk factor for cardiovascular and cerebrovascular diseases and significantly increases the risk of coronary atherosclerotic heart disease, heart failure, stroke, and death in patients with hypertension[2].On the contrary, effective management of blood pressure and reversal of LVH can significantly reduce the risk of cardiovascular events and death[3]. Cardiac remodeling is a continuous process of gradual occurrence and development, and when LVH can be diagnosed, it is actually at a late stage. In the early stage of LVH, many patients have developed left ventricular diastolic dysfunction and left atrial enlargement, and these early manifestations of cardiac remodeling may have the same clinical significance as LVH[4, 5].Therefore, early recognition and diagnosis of LVH are particularly important.

Blood fat as a regular check-up index forecasting role in cardiovascular risk have to get consensus, with the deepening of the medical research, triglycerides (TG), high-density lipoprotein cholesterol (HDL-C) has become increasingly important in lipid indicators. Foreign studies have reported that the TG/HDL-C can be used as an independent predictor of the risk of cardiovascular disease (CVD)[6, 7]. First, increased plasma TG and decreased HDL-C concentration have been identified as risk factors for coronary heart disease. Compared with a single blood lipid index, it has more advantages in assessing the risk of CVD[8, 9]. Second, TG/HDL-C, which is usually elevated in patients with insulin resistance, has been shown to independently predict cardiovascular events[10, 11]. Previous studies have shown that the TG/HDL-C ratio is considered a potential tool to identify patients at increased risk for CVD, type 2 diabetes, fatty liver, and chronic kidney disease[12-15].On this basis, we speculated that TG/HDL-C could be used as an effective indicator for the early identification of LVH risk in patients with hypertension. Therefore, we conducted a cross-sectional study to explore the relationship between TG/HDL-C and LVH and to evaluate whether this relationship is influenced by other related factors.

Methods

Subjects: The details of our research program have been described above[16]. In short, this community-based cross-sectional study was conducted in Xinyang County, central China, from 2004 to 2005. We adopted a multi-stage cluster sampling method and selected rural community residents aged 40 to 75 as a representative sample. A total of 13,444 subjects (5270 males and 8174 females) completed the survey, and the response rate was 84.9%. Among them, 5421 hypertensive patients were identified and thoroughly examined. Hypertension is defined as diastolic blood pressure ≥90 mmHg (DBP), systolic blood pressure ≥140 mmHg (SBP), physician diagnosis or current hypertension medication (defined by WHO in 1999). Of 5421 hypertensive patients, 4805 patients had measured LVM through echocardiography. 253 subjects with no blood indicators were excluded. Ultimately, a total of 4552 patients with integrated clinical and echocardiographic data were enrolled in the present study. The blood pressure was measured by a well-trained professional with a standard mercury sphygmomanometer, and one of three cuff sizes (normal adult, large or small) was selected according to the circumference of the participant's right arm. All participants were advised not to drink coffee/tea, drink alcohol, smoke, or exercise for at least 30 minutes before measuring blood pressure. Participants rested for at least 5 minutes, at least 30 seconds apart, and the three average readings of the sitting posture were taken for analysis.

The study protocol was reviewed and approved by the ethical committees of the Fuwai Hospital and local hospitals. All participants gave their informed consent before they were recruited and reported themselves to be Han people. All investigators were trained at the Cardiovascular Institute, Chinese Academy of Medical Sciences (Beijing, China) and to be eligible by test.

Echocardiography Measurements: Transthoracic echocardiography was performed according to a standard protocol[17]including 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 a HP 5500 (Phillips Medical System, Boston, MA,USA) or a HDI 3000 (A TL, Bothell, W A, USA). The echocardiographic examination was supervised by 2 physician-echocardiographers with at least 2 years of experience. Two technicians from each center performed all the echocardiographic studies. Before the study, they were trained in the echocardiographic protocol at the Cardiovascular Institute, Chinese Academy of Medical Sciences. Correct orientation of planes for 2D and Doppler imaging was confirmed using standard procedures[18]. LV internal dimensions and septal and posterior wall thicknesses were measured in up to three cardiac cycles at end-diastole and end-systole according to the American Society of Echocardiography recommendations[17]. When optimal orientation of the L V views could not be obtained, as is common in subjects who are overweight or over age 60, correctly oriented

2-dimensional linear dimension measurements were made following the leading edge convention of the American Society of Echocardiography[19].

LVM was calculated by using the equation: $0.8x1.04[(IVS+LVEDD+PW)^3-LVEDD^3]x0.6$, which yields values closely related (R=0.90) to necropsy LV weight[20]. IVS is interventricular septum, PW is 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 using the criteria of $LVM \geq 49.2gm^{-2.7}$ for men and 46.7 $gm^{-2.7}$for women[21].

Covariate Measurements and Definitions: Information about covariates, such as age, gender, and lifestyle, was collected in a single outpatient visit by cardiologists and trained nurses using standard face-to-face interviews. All subjects took fasting blood samples in the morning after fasting for at least 12 hours. Blood samples were collected from the anterior cubital vein in a vacuum tube containing EDTA. Fasting plasma glucose (FPG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), HDL-C, TG, electrolytes, and renal function indicators were analyzed enzymatically on the autoanalyzer. All laboratory equipment was calibrated, and blinded duplicate samples were used.

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 entire study population was divided into four groups according to their quartiles of 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 between these four groups. Data are reported as the mean ± standard deviation for continuous variables and as percentages for categorical variables. Continuous variable independent sample t-test and classified variable chi-square test were used for the differences between LVH group and non-LVH group. All participants were stratified by quartiles of TG/HDL-C, baseline differences in clinical variables, and echocardiography data between groups using analysis of variance (ANOVA) for continuous variables, and Chi-squared test for categorical variables. The logistic regression analysis was used to calculate odds ratios (ORs) and their 95% confidence intervals (CIs). We used the 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 TC, LDL-C, eGFR, ALB, and ALT. Model 3 included admission of smoking, drinking, SBP, DBP, WHR, stroke. Model 4 added TC, LDL-C, SUA, FPG, SCr, BUN, ALT, 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 < 0.05.

Results

Clinical characteristics of the study population: Table 1 shows the clinical and demographic characteristics of the study population by the presence or absence of LVH. Participants of LVH were older than those with non-LVH group (P < 0.001). Male patients comprised 29.5% of the LVH group and 36.6% of the non-LVH group. The indicators including age, SBP, DBP, TG, SCr, BUN, SUA, WHR, and TG/HDL-C were all higher in the LVH group compared with the non-LVH group. In contrast, HDL-C was significantly reduced in the LVH group. Moreover, patients with LVH were more frequently women, had a longer history of hypertension and a higher morbidity of stroke. In terms of echocardiographic parameters, the mean levels of IVST, LVEDD, PWT, RWT, LVM, and $LVM/H^{2.7}$were significantly higher among participants with LVH group.

Table1 Baseline characteristics of study population

Variables	Overall study population (n=4552)	LVH (n=1884)	Non-LVH (n=2668)	p-value
Age (years)	58.1±8.3	59.0±8.0	57.5±8.5	< 0.001
Males (%)	1532(33.3)	567(29.5)	990(36.6)	< 0.001
Smokers (%)	252(5.5)	99(5.2)	157(5.8)	0.001
Drinkers (%)	211(4.6)	104(5.4)	115(4.3)	< 0.001
Pulse(times/min)	72.8±12.3	72.3±12.0	72.9±12.4	0.115
SBP (mmHg)	163.5±24.5	168.7±25.5	160.0±22.9	< 0.001
DBP (mmHg)	97.0±12.6	98.2±13.6	96.3±11.6	< 0.001
ALT (mmol/L)	19.70±13.34	19.31±12.17	20.04±14.18	0.07
AST (mmol/L)	27.87±14.40	27.55±13.35	28.17±15.11	0.154
FPG (mmol/L)	5.57±1.69	5.52±1.65	5.60±1.71	0.114
HDL-C(mmol/L)	1.55±0.34	1.52±0.33	1.57±0.35	< 0.001
LDL-C(mmol/L)	3.15±0.85	3.16±0.87	3.15±0.84	0.562
TC (mmol/L)	5.54±1.09	5.53±1.09	5.55±1.10	0.597
TG (mmol/L)	1.69±1.24	1.75±1.30	1.64±1.19	0.001
SUA(μmol/L)	292.85±86.81	295.96±86.12	290.65±87.22	0.044
SCr (μmol/L)	66.26±26.12	66.92±28.86	65.79±24.02	0.155
BUN (mmol/L)	5.47±1.81	5.54±1.85	5.41±1.78	0.016
TG/HDL-C	1.20±1.15	1.27±1.26	1.15±1.07	0.001
WHR	0.87±0.12	0.872±0.06	0.866±0.07	0.002
Stroke (%)	467(10.3)	234(12.6)	233(8.9)	< 0.001
Duration of hypertension	6.92±7.58	7.85±7.98	6.18±7.12	< 0.001
UCG indices				
IVST (cm)				
LVEDD (cm)	1.00±0.16	1.09±0.15	0.93±0.13	< 0.001
PWT (cm)	4.55±0.51	4.82±0.47	4.35±0.45	< 0.001
RWT	0.97±0.14	1.05±0.13	0.91±0.11	< 0.001
LVM (g)	0.43±0.08	0.44±0.08	0.42±0.07	< 0.001
LVM/$H^{2.7}$($g/h^{2.7}$)	158.77±44.12	193.36±38.72	134.24±28.53	< 0.001
	46.38±12.48	58.04±9.42	38.11±6.21	< 0.001

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; 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.

Clinical, biochemical characteristics and echocardiogram parameters of all participants by quartiles of the TG/HDL-C ratio were summarized in Table 2. With the increase of TG/HDL-C from the first to the fourth quartile (Q1-Q4), parameters including DBP, TG, TC, LDL-C, FPG, SUA, SCr, ALT and WHR were all significantly elevated (all P for trend < 0.05). On the other hand, there was an inverse association between HDL-C and TG/HDL-C ratio. In terms of echocardiographic parameters, IVST, LVM, and LVM/$H^{2.7}$ parameters were significantly increased with TG/HDL-C from Q1-Q4 (all P for trend < 0.001).

Table 2 Clinical, biochemical characteristics and echocardiogram parameters of the study subjects according to quartiles of TG/HDL-C

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±13.7a	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±85.2 [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
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
$LVM/H^{2.7}(g/h^{2.7})$	45.2±12.7 [c]	46.4±12.1	46.7±12.8 [a]	47.4±12.4 [a]	<0.001

Data are presented as mean ± standard deviation or median (interquartile range) for continuous variables, and number (percentage) for categorical Variables

[a]p< 0.05 compared with Quartile 1

[b]p< 0.05 compared with Quartile 2

[c]p< 0.05 compared with Quartile 3

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 3, quartiles of TG/HDL-C were associated with increased risk of LVH in univariate analysis (model1). 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, UREA, ALT, AST (mode4). 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 (Table 3).

Table3 Odds ratios and 95% confidence intervals of TG/HDL-C ratio and LVH in all subjects

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

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

Given differences in TG and HDL cholesterol metabolism between men and women[22, 23], we further performed a sex-stratified analysis of the correlation between LVH and TG/HDL-C categories and the results are shown in Table 4. Our study showed that the relationship between TG/HDL-C and LVH risk was significantly different by sex, and this association was significantly higher in women than in men. After adjusting for several covariates, women still showed a strong association.

Table 4 Odds ratio for left ventricular hypertrophy by TG/HDL in stratification analysis

	Q1 (<0.61)	Q2(0.61-0.91)		Q3(0.92-1.41)		Q4（>1.41）	
		Crude OR (95% CI); p-value	Adjusted OR (95% CI); P-value	Crude OR (95% CI); p-value	Adjusted OR (95% CI); P-value	Crude OR (95% CI); p-value	Adjusted OR (95% CI); P-value
Sex							
Male	Reference	1.26(0.94-1.70); 0.120	1.15(0.84-1.58); 0.376	1.19(0.90-1.59); 0.227	1.06(0.77-1.45); 0.727	1.22(0.91-1.63); 0.184	1.21(0.86-1.70); 0.265
Female	Reference	1.24(1.01-1.53); 0.043	1.26(1.02-1.57); 0.036	1.33(1.08-1.64); 0.007	1.40(1.12-1.75); 0.003	1.51(1.22-1.86); <0.001	1.64(1.31-2.05); <0.001

Abbreviations: 95% CI, 95% confidence interval; OR, odds ratio

Adjusted: age, smoking, drinking, SBP, DBP, WHR, stroke, TC, LDL-C, SUA, FPG, SCr, UREA, ALT, AST

Discussion

LVH is one of the most common hypertension-mediated organ damages and is an independent predictor of cardiovascular morbidity and mortality[24, 25]. LVH is closely associated with decreased cardiac systolic function, decreased coronary artery reserve, and susceptibility to various arrhythmias. LVH significantly increases the risk of coronary atherosclerotic heart disease, heart failure, stroke, and death in hypertensive patients. In contrast, effective management of blood pressure and reversal of LVH can significantly reduce the risk of cardiovascular events and death. Previous studies have shown that reversal of LVH or sustained normal LVM was associated with a 46% reduction in the risk of overall cardiovascular events (P<0.05) compared with persistence or progression of LVH[26]. Hypertensive cardiac remodeling refers to the changes in the structure and function of the heart associated with hypertension, involving various aspects such as ventricle and atrium, cardiac contraction and diastolic function. Cardiac remodeling is a continuous process of progressive occurrence and development. When LVH can be diagnosed, it has actually developed to a late stage, and many patients have left ventricular diastolic dysfunction and left atrial enlargement in the early stage of LVH. Therefore, early effective identification, active prevention and reversal of LVH have important clinical value.

The ratio of TG/HDL-C has been increasingly recognized as an important predictor of coronary artery disease, cardiovascular disease death[27], and cardiac metabolism[28], which may be attributed to the fact that high TG/HDL-C ratio is associated with insulin resistance and metabolic syndrome, which increase the traditional risk factors of CVD. Accelerates the progression of coronary atherosclerosis and leads to plaque instability, which leads to adverse events of coronary heart disease[27]. TG is directly involved in the synthesis of cholesterol and cholesterol esters and is the main lipid component leading to atherosclerosis.TG varies greatly among different individuals, and age, diet, and living habits can all affect its level. HDL-C is an anti-atherosclerosis lipoprotein, and its plasma content is negatively correlated with the risk of cardiovascular and cerebrovascular diseases. Studies have found that the serum HDL-C level is often low in patients with hyper-triglyceridemia, which may be related to the decrease of serum HDL-C level due to the unstable structure of HDL-C particles rich in TG and their easy degradation. Compared with the

components of blood lipid, TG/HDL-C ratio can better reflect the comprehensive level of lipid metabolism in patients[29]. Abnormal lipid metabolism accelerates the atherosclerosis process, resulting in decreased compliance and dilation of the great arteries. The compliance of the great arteries is the main determinant of the left ventricular afterload. The decreased compliance leads to the increase of the stress on the left ventricular wall during the systolic period, prolongation of the left ventricular ejection time, and leads to LVH.

In our study, we observed a strong cross-sectional association between increased risk of TG/HDL-C and LVH in Chinese hypertensive patients who are at high risk for developing cardiovascular disease. The main findings support our hypothesis that people with a high TG/HDL-C ratio are at a higher risk of LVH in Chinese hypertensive patients. After adjusting for all potential confounders, Subjects in the quartile with the highest TG/HDL-C levels were nearly 1.5 times more likely to develop LVH than those in the quartile with the lowest TG/HDLC levels. Our results suggest that an increased TG/HDL-C ratio can further identify patients at high risk of LVH in hypertensive populations.

A recent editorial in the Lancet reminded researchers of the importance of "analyzing data by gender, not only when scientifically appropriate, but also as a matter of routine[30]." Therefore, we conducted a stratified analysis to explore whether the association between TG/HDL-C and LVH was influenced by gender. Our study showed that there were significant gender differences in the relationship between TG/HDL-C ratio and LVH risk and that this association was significantly higher in women than in men. Notably, similar results have been observed for associations between TG/HDL-C and nonalcoholic fatty liver disease, chronic kidney disease, and the risk of diabetes [13-15]. While the underlying mechanism for this sex difference remains unknown, one possible explanation could be due to sex hormones. Gender differences between LVH and TG/HDL-C provide new insights into the pathophysiology of LVH in men and women and provide new impetus for further research. In addition, our study examined a large population of patients with hypertension in the Chinses rural area, which minimizes selection bias. We estimated the LVM by echocardiographic, which is more sensitive and specific than ECG.

There are some limitations to this study. This cross-sectional study did not carry out prospective follow-up and intervention studies, and the causal relationship between TG/HDL-C and LVH could not be concluded. However, this study suggested that high TG/HDL-C was independently correlated with LVH, suggesting a possible mechanism for the occurrence and development of related diseases caused by such dyslipidemia. In addition, the proportion of male subjects in this study was relatively low (33.3%), and there were fewer male LVH patients (29.5%). Therefore, the gender difference between TG/HDL-C and LVH needs to be further verified by expanding the proportion of male subjects. In addition, the lack of studies in non-hypertensive groups should also be taken into account, as demonstrating a strong association between TG/HDL-C and LVH in non-hypertensive high-risk populations would reinforce the results of this study and would expand its clinical significance in other populations.

Abbreviations

LVH: Left ventricular hypertrophy

TG/HDL-C: triglycerides to-high-density lipoprotein cholesterol

LVM: Left ventricular mass

LVMI: left ventricular mass indexed

TG: triglycerides

HDL-C: high-density lipoprotein cholesterol

CVD: cardiovascular disease

IVS: interventricular septum

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: serum urea nitrogen

ALT: alanine aminotransferase

AST: aspartate aminotransferase

WHR: Waist-to-Hip Ratio

Declarations

Conflict of interest

The authors declare no conflict of interest.

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Authors' contributions

Sx.W., P.Z. and S.C. designed the research. S.C., J.D. and Bk.C, Ah.Z. collected the data. S.C. and J.D. wrote the paper. J.S., Yk.S. and Q.B. help optimize the research and proofread the paper. The authors read and approved the final manuscript.

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