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Influence of hypertension and global or abdominal obesity on left ventricular hypertrophy: A cross-sectional study

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Abstract

Although hypertension and obesity are both risk factors for left ventricular hypertrophy (LVH), the extent of their impact on LVH in the general population is still unclear, and the predictive value of obesity indicators for LVH remains to be elucidated. In this study, obesity-related indicators, including waist circumference (WC), waist-height ratio (WHTR), and waist-hip ratio (WHR), were used to define abdominal obesity (AO), whereas body mass index (BMI) was used to measure general obesity (GO). The effects of hypertension and obesity on LVH were estimated using logistic regression analysis, as was the relative risk of LVH based on the presence of obesity, hypertension, or both. Subgroup analyses were performed based on sex and age. Of the 9134 participants (≥35 years old), 915 (10.0%) developed LVH. After adjusting for covariates, the odds ratios (95% confidence intervals) for LVH were 3.94 (3.27–4.75) in patients with hypertension, 1.90 (1.60–2.26) in those with GO, and 1.45 (1.25–1.69), 1.69 (1.43–2.00), and 1.54 (1.33–4.75) in individuals with AO defined based on WC, WHTR, and WHR, respectively. Analysis by sex showed similar values in women, but AO based on WC and WHR were not significantly associated with LVH in men. Further, after adjusting for potential confounding factors, concomitant hypertension and obesity had an increased risk of developing LVH in all age ranges, particularly in patients aged 35–45 years (risk increased 14.14-fold, 10.84-fold, 7.97-fold, and 9.95-fold for BMI-based GO and WC-, WHTR-, and WHR-based AO, respectively), and in both men and women but particularly in men (risk increased 7.71-fold, 4.67-fold, 5.83-fold, and 5.58-fold, respectively). In summary, all obesity indicators (BMI, WC, WHTR, and WHR) had predictive value for LVH in women; however, only BMI and WHTR should be considered for men. Furthermore, monitoring for the occurrence and progression of LVH is imperative for rural Chinese patients with concomitant hypertension and obesity, especially men and those aged 35–45 years.

KEYWORDS
hypertension, left ventricular hypertrophy, obesity

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1 | INTRODUCTION

Left ventricular hypertrophy (LVH) is an adverse structural change occurring in the myocardium; defined by a pathological increase in the left ventricular mass (LVM), LVH represents a maladaptive response to various cardiovascular risk factors, such as aging, hypertension, diabetes, and kidney disease.[1] A growing body of evidence supports LVH as an independent predictor of cardiovascular disease (CVD).[2] LVH is now regarded as a subclinical alternative cardiovascular endpoint, and its reversal is associated with an improved cardiovascular prognosis.[3] LVH incidence has been reported to be higher in women than in men; thus, sex discrepancies in prevention, diagnosis, and treatment should be considered.[4]

Hypertension can induce myocardial hypertrophy and interstitial fibrosis, leading to left ventricular remodeling. Hypertension is frequently associated with concentric or eccentric LVH or concentric left ventricular remodeling.[5] Many clinical and epidemiological studies have found that increased blood pressure plays a significant role in LVH occurrence and development.[6] Further, prehypertension[7] and adolescent blood pressure trajectories have been linked to LVH onset, and preliminary studies suggest that LVH occurs in approximately half of the untreated patients with hypertension.[8] Like hypertension, obesity is regarded as a major modifiable risk factor for LVH and is closely linked to the failure of antihypertensive therapies to reduce LVM.[4,9] Overweight and obesity accounted for 11.1% of noncommunicable disease deaths in China, resulting in a significant health and economic burden.[10] Obesity-related hemodynamic and metabolic alterations enhanced LVH, impairing left ventricular diastolic function.[11,12] As a result, LVH prevalence in obese individuals is approximately four times higher than in non-obese individuals.[13]

In summary, although hypertension and obesity have separate effects on LVH, the magnitude of their impact on LVH in the population of rural China is still unknown. Moreover, as components of the metabolic syndrome that may have synergistic effects, their combined effects on LVH have not been elucidated. Notably, the visceral fat content in the Chinese population is proportionally higher than in Caucasians with similar fat content,[14] and the high prevalence of visceral obesity may be the cause of the high risk of CVD observed in the Chinese population with low body mass index (BMI). Multiple indicators are currently used to measure obesity. However, the focus of various indicators is not entirely the same, and their predictive value for diseases may vary. The indicators suitable for predicting LVH in the Chinese population are still unknown.[15] Waist circumference (WC), waist-height ratio (WHTR), and waist-hip ratio (WHR) have been widely recommended as alternative indicators of abdominal and visceral obesity in places lacking medical imaging capabilities, particularly in rural areas.[16] In this study examining a population in rural China, a variety of obesity-related indicators were used to determine the risk of LVH under different obesity standards and to determine the impact of hypertension on LVH. In addition, we investigated the relative risk of LVH based on obesity, hypertension, or both. Lastly, given that sex and age can affect LVH to varying degrees, we conducted a subgroup analysis based on these two parameters.

2 | METHODS

2.1 | Study population

Data were obtained from the Northeast China Rural Cardiovascular Health Study (NCRCHS), a representative rural community-based sample.[17] A multistage stratified cluster sampling strategy was used to recruit participants in this study. First, three counties (Dawa County, Zhangwu County, and Liaoyang County) were randomly selected from different regions of the Liaoning Province. Second, we randomly selected one town from each county (three towns in total). Lastly, 8—10 rural villages were randomly selected from each town, and 11,956 individuals (≥35 years old) from 26 villages were recruited. Participants with pregnancy, malignant tumors, or serious mental disorders causing an inability to cooperate with the investigation were excluded. The response rate was 89.4% (10700/11956). Further exclusion criteria included: incomplete biochemical evaluations (n = 113), incomplete physical examinations (n = 345), moderate or severe valvular heart disease (n = 79), or incomplete cardiac ultrasound data (n = 1029). Ultimately, 1566 participants were excluded. Thus, 9134 participants were included in this study. The study was approved by the ethics committee of China Medical University (Shenyang, China AF-SDP-7-1, 0-01), and all participants provided written informed consent after they were adequately informed of the research.

2.2 | Data source and covariates

A self-administered questionnaire on anthropometric parameters and lifestyle was used to collect survey data. The Central Steering Committee managed data sources and quality assurance processes. All investigators completed training and examination tests. The detailed methods have been described previously.[17] Anthropometric indicators were measured following standard procedures, and the participants were barefoot and wore light clothing. Measurements were recorded to the nearest 0.1 cm and 0.1 kg. WC was measured at belly level with an inelastic anthropometric belt at the end of a typical exhale, and hip circumference was measured at the maximal gluteal muscle protrusion. BMI was calculated as follows: BMI = body weight (kg)/height^2 (m^2). WHTR and WHR were defined as follows: WHTR = WC (cm)/height (cm) and WHR = WC (cm)/hip circumference (cm). Smoking, drinking stimulants (e.g., tea, coffee, and alcohol), and excessive exercise were discouraged before blood pressure measurement. A fully automatic sphygmomanometer (HEM-907; Omron, Tokyo, Japan) was used to determine the blood pressure of the right upper arm of the participants while sitting, and the measurement was repeated three times to obtain the average value. Blood samples were collected after a fasting period of approximately 12 h. The parameters analyzed included serum creatinine, fasting plasma glucose (FPG), and estimated

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glomerular filtration rate (eGFR) calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula.[18] Uric acid (UA) level above 420 mg/dL was defined as elevated. Dyslipidemia was diagnosed according to the National Cholesterol Education Program Adult Treatment Panel III as total cholesterol (TC) ≥6.21 mmol/L, triglyceride (TG) ≥2.26 mmol/L, low-density lipoprotein cholesterol (LDL-C) ≥4.16 mmol/L (high), and high-density lipoprotein cholesterol (HDL-C) ≤1.03 mmol/L (low). Transthoracic echocardiography was performed using a Doppler echocardiography device (Vivid, GE Healthcare, Chicago, IL, USA) with a 3.0 MHz transducer on patients placed in the left lateral decubitus position.[17] At least two experienced cardiac ultrasound specialists analyzed the echocardiograms, and echocardiographic measurements were determined by three doctors specializing in echocardiography. The parasternal long-axis view was used to record the interventricular septal thickness dimension (IVSTd), left ventricular (LV) end-diastolic internal dimension (LVIDd), and posterior wall thickness dimension (PWTd). LVM was calculated according to the American Society of Echocardiography (ASE) formula: $LVM = 0.8 \times [1.04 \times ([IVSTd + LVIDd + PWTd]^3 - [LVIDd]^3)] + 0.6$ g.

2.3 | Parameter definitions

Considering the small body size of the participants in rural China, based on the criteria of the China Obesity Working Group standards, a BMI of 18.5–23.9 kg/m^2 was considered as normal, 24–27.9 kg/m^2 as overweight, and ≥28 kg/m^2 as obesity. A BMI ≥28 kg/m^2 was defined as general obesity (GO).[10] The criteria for abdominal obesity (AO) were WC ≥90 cm, WHTR ≥0.5, or WHR ≥0.9 in men and WC ≥80 cm, WHTR ≥0.5, or WHR ≥0.85 in women.[19] Hypertension diagnostic criteria were mean systolic blood pressure (SBP) ≥140 mmHg, mean diastolic blood pressure (DBP) ≥90 mmHg, self-reported hypertension, or currently receiving antihypertensive treatment.[20] The primary indicator for diagnosing LVH by echocardiography is LVM index, corrected for body surface area (BSA), and the criteria are >115 g/m^2 in men and >95 g/m^2 in women.[21]

2.4 | Statistical analysis

Continuous variables are presented as mean ± standard deviation or median (interquartile range), whereas categorical variables are presented as numbers and percentages. Pearson's partial correlation coefficient was used to determine the association between blood pressure and the four obesity indices. The independent effects of GO, AO (as defined by the three criteria stated above), and hypertension on LVH were estimated using univariate (Model I: crude model) and multivariate (Model II: adjusted model) logistic regression analyses and presented as odds ratios (ORs) and 95% confidence intervals (CIs). A subgroup analysis was performed after classifying the participants based on age, sex, and hypertension with or without obesity (based on the four definitions described above). SPSS version 23.0 (IBM Corp., Armonk, NY, USA) was used for data analysis, and statistical significance was defined as $P < 0.05$.

3 | RESULTS

3.1 | Demographic, anthropometric, and plasma biochemical characteristics

Of the 9134 participants in our study, 915 (10%) had LVH. Table 1 shows the demographic, anthropometric, and plasma biochemical characteristics of the participants.

Among patients with LVH, 36.1% (330/915) were men with an average age of 59.8 ± 10.0 years. Compared to the non-LVH group, the LVH group was older and had higher levels of UA, FPG, TG, TC, and LDL-C; higher SBP, DBP, BMI, WC, WHTR, and WHR; and lower HDL-C levels. In addition, the LVH group had a lower proportion of participants with spouses or high school education and a higher proportion of patients with diabetes and regular exercise than the non-LVH group. Patients with LVH had higher levels of GO; AO based on WC, WHTR, and WHR; and hypertension when compared with patients without LVH (all $P < 0.05$). However, when we analyzed men separately, there were no differences in the prevalence of diabetes ($P = 0.201$), AO defined based on WC ($P = 0.113$), and levels of TC ($P = 0.267$), TG ($P = 0.697$), and HDL-C ($P = 0.223$) between participants with and without LVH.

Figure 1 depicts the prevalence of LVH according to the presence of hypertension and GO or AO as defined based on WC, WHTR, and WHR. The incidence of LVH was higher in patients with both obesity and hypertension. Among patients with both hypertension and AO (based on WC, WHTR, and WHR), the prevalence of LVH was 12.74%, 13.70%, and 13.66% for men and 20.74%, 20.92%, and 19.04% for women, respectively.

3.2 | Partial correlation analysis of blood pressure and obesity indices

Table 2 shows the partial correlation coefficients between SBP, DBP, GO, and AO levels. Adjustments were made for age, sex, FPG, TC, TG, LDL-C, HDL-C, marital status (no spouse), education, eGFR, current smoking, current drinking, UA, family history of hypertension, exercise, and antihypertensive drug treatment. Among blood pressure and obesity indices, SBP had the strongest correlation with BMI ($r = 0.22$). Among the four obesity indices, the correlation coefficient between WHTR and WC was the highest ($r = 0.932$).

3.3 | Univariate and multivariate logistic regression analyses of risk factors for LVH

Table 3 shows the logistic regression analysis results used to explore independent risk factors for LVH. In the general population, hypertension and obesity, as defined based on BMI, WC, WHTR, and WHR, were

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TABLE 1 Baseline demographic, anthropometric, and plasma biochemical characteristics of the study population based on LVH and sex.

	Total			Male			Female		
	LVH			LVH			LVH		
Variables	No ($n = 8219$)	Yes ($n = 915$)	p value	No ($n = 3808$)	Yes ($n = 330$)	p value	No ($n = 4411$)	Yes ($n = 585$)	p value
Age (years)	52.81 ± 10.17	59.82 ± 10.04	<0.001	53.47 ± 10.45	59.77 ± 10.55	<0.001	52.23 ± 9.88	59.85 ± 9.75	<0.001
BMI (kg/m2)	24.73 ± 3.60	25.59 ± 4.13	<0.001	24.69 ± 3.43	25.46 ± 4.32	0.001	24.76 ± 3.74	25.67 ± 4.02	<0.001
FPG (mmol/L)	5.85 ± 1.57	6.11 ± 1.74	<0.001	5.91 ± 1.63	6.01 ± 1.61	0.008	5.80 ± 1.52	6.17 ± 1.81	<0.001
TC (mmol/L)	5.22 ± 1.08	5.44 ± 1.18	<0.001	5.17 ± 1.04	5.24 ± 1.12	0.267	5.26 ± 1.11	5.54 ± 1.20	<0.001
TG (mmol/L)	1.58 ± 1.44	1.76 ± 1.43	<0.001	1.64 ± 1.65	1.62 ± 1.67	0.697	1.53 ± 1.22	1.84 ± 1.26	<0.001
LDL-C (mmol/L)	2.93 ± 0.83	3.09 ± 0.88	<0.001	2.88 ± 0.80	2.95 ± 0.87	0.223	2.97 ± 0.85	3.17 ± 0.87	<0.001
HDL-C (mmol/L)	1.43 ± 0.39	1.38 ± 0.36	0.001	1.43 ± 0.44	1.37 ± 0.39	0.026	1.42 ± 0.35	1.38 ± 0.33	0.007
UA (mg/dL)	4.81 ± 1.38	4.99 ± 1.43	<0.001	5.53 ± 1.35	5.66 ± 1.39	0.11	4.19 ± 1.08	4.62 ± 1.32	<0.001
Mean SBP (mmHg)	139.71 ± 21.87	161.46 ± 26.91	<0.001	142.05 ± 21.34	163.27 ± 25.12	<0.001	137.68 ± 22.13	160.44 ± 27.83	<0.001
Mean DBP (mmHg)	81.39 ± 11.17	88.12 ± 14.17	<0.001	83.21 ± 11.34	91.47 ± 13.91	<0.001	79.82 ± 10.78	86.22 ± 13.98	<0.001
Hypertension (%)	3909 (47.6)	756 (82.6)	<0.001	1952 (51.3)	285 (86.4)	<0.001	1957 (44.4)	471 (80.5)	<0.001
Antihypertensive drug treatment (%)	4318 (52.5)	497 (54.3)	0.306	2015 (52.9)	170 (51.5)	0.625	2303 (52.2)	327 (55.9)	0.093
Mean WC (cm)	82.00 ± 9.76	84.40 ± 9.92	<0.001	83.57 ± 9.60	85.31 ± 10.09	0.002	80.65 ± 9.69	83.89 ± 9.79	<0.001
WHTR	0.51 ± 0.06	0.54 ± 0.07	<0.001	0.50 ± 0.06	0.52 ± 0.06	<0.001	0.52 ± 0.06	0.55 ± 0.07	<0.001
WHR	0.85 ± 0.08	0.88 ± 0.09	<0.001	0.87 ± 0.07	0.88 ± 0.09	<0.001	0.84 ± 0.08	0.88 ± 0.08	<0.001
eGFR (mL/min/1.73 m2)	93.72 ± 15.32	93.91 ± 15.73	0.707	93.76 ± 14.56	93.00 ± 15.10	0.412	93.68 ± 15.96	94.42 ± 16.07	0.269
Male (%)	3808 (46.3)	330 (36.1)	<0.001						
Han nationality (%)	7748 (94.3)	873 (95.4)	0.155	3587 (94.2)	318 (96.4)	0.101	4161 (94.3)	555 (94.9)	0.594
Current smoking (%)	2937 (35.7)	313 (34.2)	0.36	2241 (58.8)	180 (54.5)	0.128	696 (15.8)	133 (22.7)	<0.001
Current drinking (%)	1904 (23.2)	155 (16.9)	<0.001	1773 (46.6)	134 (40.6)	0.037	131 (3.0)	21 (3.6)	0.412
Diabetes (%)	754 (9.2)	150 (16.4)	<0.001	346 (9.1)	37 (11.2)	0.201	408 (9.2)	113 (19.3)	<0.001
No spouse (%)	681 (8.3)	135 (14.8)	<0.001	318 (8.4)	39 (11.8)	0.031	363 (8.2)	96 (16.4)	<0.001
Education (%)			<0.001			<0.001			<0.001
Illiteracy	679 (8.3)	143 (15.6)		164 (4.3)	34 (10.3)		515 (11.7)	109 (18.6)	
Middle school and below	6764 (82.3)	731 (79.9)		3231 (84.8)	274 (83.0)		3533 (80.1)	457 (78.1)	
High school and above	776 (9.4)	41 (4.5)		413 (10.8)	22 (6.7)		363 (8.2)	19 (3.2)	
Family history of hypertension (%)	788 (20.7)	80 (24.2)	0.129	1058 (24.0)	180 (30.8)	<0.001	788 (20.7)	80 (24.2)	0.129
Exercise (%)	738 (19.4)	90 (27.3)	<0.001	906 (20.5)	147 (25.1)	0.011	738 (19.4)	90 (27.3)	<0.001
GO by BMI (%)	1384 (16.8)	246 (26.9)	<0.001	613 (16.1)	78 (23.6)	<0.001	771 (17.5)	168 (28.7)	<0.001
AO by WC (%)	3377 (41.1)	491 (53.7)	<0.001	1045 (27.4)	104 (31.5)	0.113	2332 (52.9)	387 (66.2)	<0.001
AO by WHTR (%)	4827 (58.7)	681 (74.4)	<0.001	2029 (53.3)	217 (65.8)	<0.001	2798 (63.4)	464 (79.3)	<0.001
AO by WHR (%)	3401 (41.4)	523 (57.2)	<0.001	1270 (33.4)	136 (41.2)	0.004	2131 (48.3)	387 (66.2)	<0.001

Continuous data are presented as mean ± standard deviation; categorical data are presented as n (%).

Abbreviations: AO, abdominal obesity; BMI, body mass index; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; GO, general obesity; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LVH, left ventricular hypertrophy; SBP, systolic blood pressure; TC, total cholesterol; TG, triglyceride; UA, uric acid; WC, waist circumference; WHR, waist-hip ratio; WHTR, waist-height ratio.

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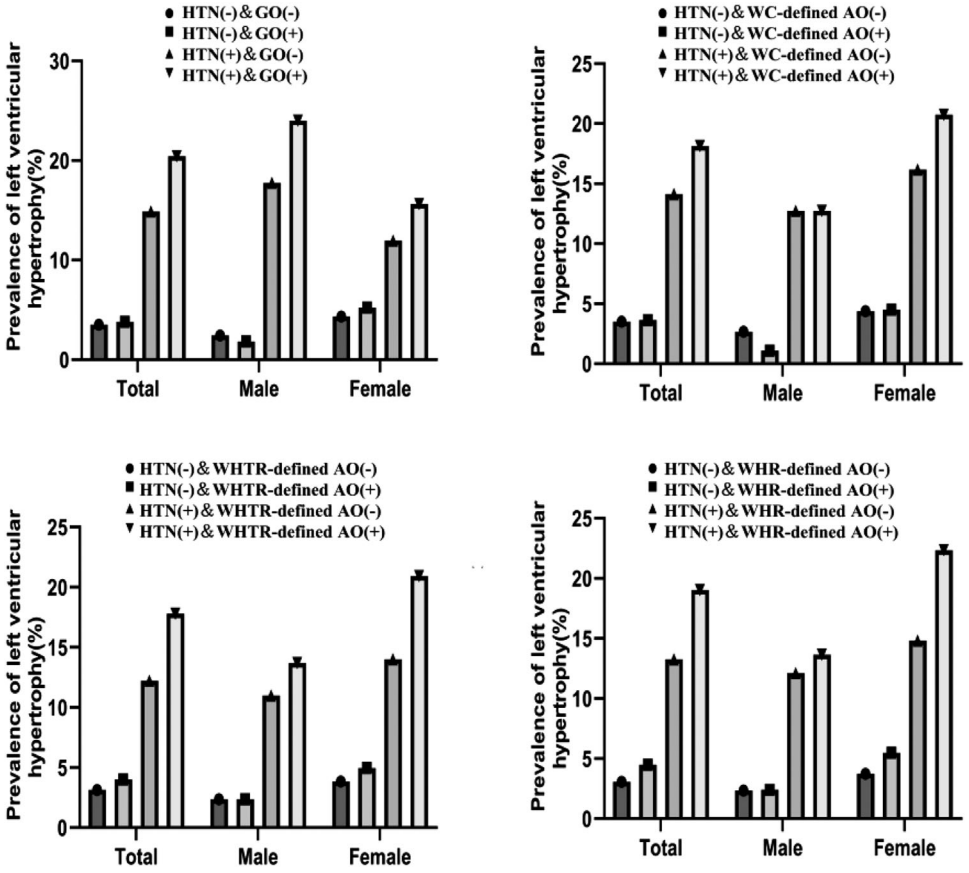


FIGURE 1 Prevalence of left ventricular hypertrophy (LVH) based on the combined presence of hypertension and obesity. AO, abdominal obesity; BMI, body mass index; GO, general obesity; HTN, hypertension; WC, waist circumference; WHR, waist-hip ratio; WHTR, waist-height ratio. (+) represents with; (-) represents without.

TABLE 2 Pearson's partial correlation between blood pressure and obesity indices.

	SBP	DBP	BMI	WC	WHTR	WHR
SBP	1.000					
DBP	0.719	1.000				
BMI	0.220	0.178	1.000			
WC	0.209	0.173	0.744	1.000		
WHTR	0.213	0.163	0.781	0.932	1.000	
WHR	0.111	0.098	0.349	0.660	0.650	1.000

Numbers indicate Pearson's partial correlation coefficients (adjusted for age, sex, FPG, TC, TG, LDL-C, HDL-C, marital status, education, eGFR, current smoking, current drinking, UA, family history of hypertension, exercise, and antihypertensive drug treatment).

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; TG, triglyceride; UA, uric acid; WC, waist circumference; WHR, waist-hip ratio; WHTR, waist-height ratio.

significant risk factors for LVH. In the crude model (Model I, univariate logistic regression analysis), the OR (95% CI) for LVH was 5.24 (4.40–6.25) in patients with hypertension, 1.82 (1.55–2.13) in those with GO, and 1.66 (1.45–1.90), 2.05 (1.75–2.39), and 1.89 (1.65–2.17) in those with AO as defined based on WC, WHTR, and WHR, respectively. After further adjustments for confounding factors (Model II), these correlations remained in the overall population. Similarly, GO defined based on BMI and AO defined based on WHTR were linked to an increased LVH risk among all participants (All $P < .05$). Moreover, in Model II, AO defined based on WC and WHR were independently associated with LVH in women [OR 1.34 (95% CI:1.10–1.64) and OR 1.47 (95% CI:1.21–1.80), respectively] but not in men.

3.4 | Subgroup analysis

Table 4 shows the ORs of LVH based on the combined presence of hypertension and obesity indicators stratified by age and sex. Participants without hypertension and obesity served as the reference group. Among the total population, the risk of LVH in patients with hypertension and BMI-defined GO was approximately six times higher than that in the reference group. LVH risk in patients with hypertension and AO, as defined based on WC, WHTR, or WHR, was also higher than that in the reference group. In the youngest group (35–44 years old), patients with hypertension and various definitions of obesity had a 7.97–14.14-fold increase in the risk of LVH. However,

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TABLE 3 Logistic regression analyses of the association of left ventricular hypertrophy with obesity and hypertension.

	Total		Male		Female	
	OR (95%CI)		OR (95%CI)		OR (95%CI)	
	Model I	Model II	Model I	Model II	Model I	Model II
GO by BMI	1.82 (1.55–2.13)*	1.90 (1.60–2.26)*	1.61 (1.23–2.11)*	1.84 (1.36–2.49)*	1.90 (1.56–2.31)*	1.85 (1.49–2.29)*
AO by WC	1.66 (1.45–1.90)*	1.45 (1.25–1.69)*	1.22 (0.95–1.55)	1.14 (0.87–1.50)	1.74 (1.45–2.09)*	1.34 (1.10–1.64)*
AO by WHTR	2.05 (1.75–2.39)*	1.69 (1.43–2.00)*	1.68 (1.33–2.13)*	1.54 (1.18–2.01)*	2.21 (1.79–2.73)*	1.63 (1.30–2.04)*
AO by WHR	1.89 (1.65–2.17)*	1.54 (1.33–1.79)*	1.40 (1.11–1.76)*	1.26 (0.98–1.61)	2.09 (1.75–2.51)*	1.47 (1.21–1.80)*
Hypertension	5.24 (4.40–6.25)*	3.94 (3.27–4.75)*	6.02 (4.37–8.30)*	5.13 (3.67–7.18)*	5.18 (4.19–6.41)*	3.49 (2.78–4.40)*

Model I: crude model (univariate logistic regression analysis).
Model II: multivariate logistic regression analysis adjusted for age, UA, FPG, TC, TG, LDL-C, HDL-C, marital status (no spouse), education, eGFR, current smoking and drinking, family history of hypertension, hypertension, exercise, and antihypertensive drug treatment.
Abbreviations: AO, abdominal obesity; BMI, body mass index; CI, confidence interval; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; GO, general obesity; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio; TC, total cholesterol; TG, triglyceride; UA, uric acid; WC, waist circumference; WHR, waist-hip ratio; WHTR, waist-height ratio.
*Denotes statistical significance, set at $p < 0.05$.

this risk gradually decreased to 2.51–3.12-fold with increasing age (≥65 years old).

Among all the groups, the combination of BMI-defined obesity and hypertension conferred the greatest risk of LVH in both men and women. However, the combination of hypertension and obesity increased the risk of LVH by 4.67–7.71-fold in men, which was higher than the 3.55–5.19-fold increase in women.

4 | DISCUSSION

Our study revealed that hypertension (OR:3.94) and obesity (OR: 1.45–1.90) were independently associated with the risk of LVH and hypertension had a more significant effect on LVH than did obesity. Obesity, as defined based on BMI, and AO had a similar impact on LVH risk in women, whereas focusing on BMI and WHTR in men may be more beneficial. Moreover, in non-obese patients, hypertension increased the risk of LVH. These findings suggest that regardless of obesity, appropriate blood pressure control may have health advantages; however, the combination of obesity and hypertension had a significantly greater effect, which was more pronounced in men. Compared with the reference group without hypertension and obesity, men with hypertension and obesity had a 4.67–7.71-fold increase in risk and women had a 3.55–5.19-fold increase. Additionally, we observed that the combined effect of obesity and high blood pressure on LVH was more significant in younger individuals, which decreased with age. This finding highlights the importance of effective weight and blood pressure control in middle-aged populations. In our study, the overall prevalence of LVH in women was higher than in men (11.7% vs. 7.9%), which is consistent with the findings of earlier studies.[22,23]

People in rural China have increasingly shifted to a high-fat diet and sedentary lifestyle, mirroring the current economic and social growth. According to Chinese population data, the percentage of energy supplied by fat increased from 18.4% to 31.5% between 1982 and 2012. This trend corresponded to increased edible oil consumption and rising obesity rates.[10] The WHO standard defines adult obesity as a BMI ≥30.0 kg/m^2. However, mounting evidence suggests that Chinese people may have a higher body fat ratio than do Caucasians with comparable BMI,[24] with a corresponding increase in the risk of CVD and higher rates of all-cause mortality.[25] Therefore, the China Obesity Working Group recommended that a BMI ≥28.0 kg/m^2 be defined as GO.[10] However, some studies have questioned the predictive value of BMI because it cannot distinguish between fat mass and muscle mass and does not reflect the individual fat distribution. Therefore, we examined several other obesity indicators. We found that GO is associated with LVH risk in both sexes, whereas the effect of AO defined based on WC and WHR on LVH was not significant in men, which is consistent with the findings of previous studies.[5,23] This indicates that in men the abdominal obesity index (WHTR) or BMI should be prioritized in the prevention and control of LVH. In contrast, all of the obesity indices examined in this study have practical guiding significance in the clinical prevention of LVH in women. For the diagnosis of LVH, although the LVM/height correction method accounts for the effects of height and is believed to be more sensitive in identifying obesity-related LVH,[26] a recent study in the Chinese population found that LVM/height[27] was not significantly associated with all-cause mortality; only LVM/height$^{1.7}$ (using the Chinese threshold) was significantly associated with mortality, whereas LVM/BSA was associated with both all-cause and cardiovascular mortality.[27] In our experience, the LVH detection rate of LVM/height[17] is indeed higher, at 18.2% (1658/9134). Therefore, in order to avoid the risk of false positive diagnoses, we recommend the commonly used diagnostic LVH/BSA method for domestic practice.

The mechanisms underlying the increased effects of hypertension combined with GO (defined by BMI) or AO (defined by WC, WHTR, and WHR) in men compared with that in women remain unclear, although several have been proposed. First, sex-related variances in body shape characteristics may be an underlying factor, as adipose tissue generally preferentially accumulates in the hip area of women. Nonetheless, the male body is more prone to visceral obesity, characterized by fat

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TABLE 4 Odds ratios for left ventricular hypertrophy according to the coexistence of hypertension and obesity.

	Statistics n(%)	Total OR (95%CI)	Age (35–44) OR (95%CI)	Age (45–54) OR (95%CI)	Age (55–64) OR (95%CI)	Age (≥65) OR (95%CI)	Female OR (95%CI)	Male OR (95%CI)
HTN & GO (defined by BMI)								
HTN(-) & GO(-)	3945 (43.19)	1†	1†	1†	1†	1†	1†	1†
HTN(-) & GO(+)	524 (5.74)	1.21 (0.74–1.96)	2.36 (0.91–6.16)	1.55 (0.70–3.44)	0.90 (0.35–2.34)	0.40 (0.05–3.12)	1.26 (0.72–2.19)	0.89 (0.31–2.54)
HTN(+) & GO(-)	3559 (38.96)	3.54 (2.89–4.34)*	5.06 (2.73–9.39)*	5.10 (3.40–7.63)*	2.62 (1.89–3.63)*	2.54 (1.68–3.83)*	3.15 (2.45–4.05)*	4.57 (3.20–6.53)*
HTN(+) & GO(+)	1106 (12.11)	6.08 (4.76–7.75)*	14.14 (7.38–27.07)*	6.98 (4.31–11.29)*	5.23 (3.54–7.74)*	2.93 (1.69–5.06)*	5.19 (3.86–6.99)*	7.71 (5.01–11.88)*
HTN & AO (defined by WC)								
HTN(-) & AO(-)	3016 (33.02)	1†	1†	1†	1†	1†	1†	1†
HTN(-) & AO(+)	1453 (15.91)	1.01 (0.72–1.42)	1.61 (0.70–3.71)	1.25 (0.67–2.34)	0.77 (0.42–1.41)	0.74 (0.33–1.68)	0.89 (0.60–1.32)	0.42 (0.15–1.20)
HTN(+) & AO(-)	2250 (24.63)	3.41 (2.69–4.33)*	4.21 (2.00–8.87)*	5.34 (3.28–8.68)*	2.34 (1.61–3.41)*	2.44 (1.50–3.95)*	2.88 (2.08–3.99)*	4.50 (3.14–6.46)*
HTN(+) & AO(+)	2415 (26.44)	4.53 (3.57–5.75)*	10.84 (5.63–20.89)*	5.85 (3.62–9.45)*	3.46 (2.35–5.08)*	2.51 (1.52–4.15)*	3.55 (2.62–4.80)*	4.67 (3.11–7.02)*
HTN & AO (defined by WHTR)								
HTN(-) & AO(-)	2299 (25.17)	1†	1†	1†	1†	1†	1†	1†
HTN(-) & AO(+)	2170 (23.76)	1.22 (0.88–1.68)	0.97 (0.43–2.22)	1.45 (0.77–2.73)	1.18 (0.69–2.04)	1.08 (0.52–2.25)	1.10 (0.74–1.62)	1.05 (0.57–1.92)
HTN(+) & AO(-)	1327 (14.53)	3.30 (2.46–4.44)*	2.99 (1.24–7.23)*	5.83 (3.20–10.59)*	2.25 (1.39–3.63)*	2.46 (1.32–4.62)*	2.69 (1.81–4.01)*	4.45 (2.81–7.05)*
HTN(+) & AO(+)	3338 (36.54)	4.92 (3.76–6.43)*	7.97 (4.07–15.62)*	6.58 (3.82–11.34)*	3.96 (2.56–6.14)*	3.00 (1.66–5.43)*	4.02 (2.86–5.64)*	5.83 (3.72– 9.12)*
HTN & AO (defined by WHR)								
HTN(-) & AO(-)	2930 (32.08)	1†	1†	1†	1†	1†	1†	1†
HTN(-) & AO(+)	1539 (16.85)	1.30 (0.94–1.80)	1.30 (0.56–3.04)	1.71 (0.92–3.17)	1.00 (0.57–1.74)	1.05 (0.51–2.16)	1.14 (0.78–1.68)	0.98 (0.50–1.94)
HTN(+) & AO(-)	2280 (24.96)	3.66 (2.85–4.71)*	4.47 (2.23–8.96)*	6.00 (3.61–9.99)*	2.49 (1.68–3.71)*	2.43 (1.40–4.23)*	3.05 (2.18–4.26)*	4.85 (3.26–7.20)*
HTN(+) & AO(+)	2385 (26.11)	5.20 (4.05–6.68)*	9.95 (5.19–19.08)*	7.09 (4.27–11.77)*	3.70 (2.49–5.49)*	3.12 (1.79–5.43)*	4.21 (3.07–5.78)*	5.58 (3.66–8.51)*

Multivariate logistic regression analysis adjusted for UA, FPG, TC, TG, LDL-C, HDL-C, marital status (no spouse), education, eGFR, current smoking and drinking, family history of hypertension, exercise, and antihypertensive drug treatment.

Abbreviations: AO, abdominal obesity; BMI, body mass index; CI, confidence interval; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; GO, general obesity; HDL-C, high-density lipoprotein cholesterol; HTN, hypertension; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio; TC, total cholesterol; TG, triglyceride; UA, uric acid; WC, waist circumference; WHR, waist-hip ratio; WHTR, waist-height ratio.

*Denotes statistical significance, set at $P < 0.05$.

† Represents the reference. (-) indicates without; (+) indicates with.

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deposition in the abdomen, especially around the abdominal and internal organs.[28] Visceral fat is associated with higher metabolic activity than subcutaneous fat and is correlated with a higher risk of hypertension, diabetes, and metabolic syndrome.[28] Second, we found that women in our study cohort had higher drug compliance than men; Table 1 shows that while there was no significant difference in the treatment rate of antihypertensive drugs between the LVH and non-LVH groups, the drug treatment rate in the hypertensive group was 51.2% for men and 53.4% for women (This data is not presented in the results or tables). Therefore, differences in compliance with antihypertensive treatment may play a role in the observed sex discrepancies.[29] Third, animal studies on the effect of obesity on LVH have demonstrated that the increase in LVM in mice fed a high-fat diet was more pronounced in males than in females. Compared with female mice, males had significantly increased adiponectin and leptin mRNA expression in epicardial adipose tissue.[30] This suggests that the regulation of diet-induced LVH in mammals is sex-dependent. Fourth, obesity and hypertension are well known to activate the sympathetic nervous system and renin-angiotensin-aldosterone system (RAAS), with men showing more robust activation of myocardial RAAS-related genes than women.[31] In this regard, estrogen-mediated reductions in sympathetic activity, leading to decreased RAAS activation, may play a protective role against CVD.[32] Lastly, clinicodemographic data show that the prevalence of AO in China peaks between the ages of 40 and 49 years for men; in contrast, it peaks between 60 and 69 years for women.[33] This sex difference may have contributed to the different joint influences of obesity and hypertension on LVH observed in men and women in our study. Further, despite adjusting for age and many confounding factors in our analysis, the observed sex disparity remained, potentially due to unmeasured or unknown confounding factors. Thus, further research on the combined effect of hypertension and obesity on LVH is warranted; however, the evidence presented herein suggests that the sex discrepancy in the combined effect of blood pressure and obesity on LVH is plausible and real.

This study had some limitations. First, we could not determine causal relationships between risk factors and LVH because data from a cross-sectional study were used in our study. Second, our study population included rural residents from Northeast China, a region characterized by a cold and arid geographical environment and distinct dietary preferences (e.g., high-salt pickled foods). Thus, the extrapolation of our findings to other populations may be limited. Nevertheless, this study provides data that can be used to identify high-risk groups for disease prevention in primary or community medical departments.

5 | CONCLUSIONS

Obesity and hypertension were independently and positively associated with an increased risk of LVH in middle-aged and elderly rural Chinese populations. In non-obese participants, active blood pressure control may prevent LVH. Further, obesity indicators had predictive value for LVH in women, whereas only WHTR and BMI should be considered for men. Lastly, the combined effect of hypertension and obesity had a notable synergistic correlation with the risk of LVH, and its impact was more significant in men and relatively younger adults.

AUTHOR CONTRIBUTIONS

Xueyao Zhang provided study conceptualization, performed literature retrieval and data analysis and interpretation, and drafted the article. Guangxiao Li and Dongyuan Zhang interpreted the data and revised the work critically for important intellectual content. Yingxian Sun contributed to the study design and revised the work critically for important intellectual content. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The de-identified participant data are available for validation analysis and will be shared by the corresponding author upon reasonable request. Only the data underlying this article are available, and no additional related documents will be shared.

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REFERENCES

1. Zhang T, Li S, Bazzano L, He J, Whelton P, Chen W. Trajectories of childhood blood pressure and adult left ventricular hypertrophy: the Bogalusa Heart Study. *Hypertension*. 2018;72(1):93-101.
2. Miller R, Mikami Y, Heydari B, et al. Sex-specific relationships between patterns of ventricular remodelling and clinical outcomes. *Eur Heart J Cardiovasc Imaging*. 2020;21(9):983-990.
3. van Kleef M, Visseren F, Vernooij J, et al. Four ECG left ventricular hypertrophy criteria and the risk of cardiovascular events and mortality in patients with vascular disease. *J Hypertens*. 2018;36(9):1865-1873.
4. de Simone G, Devereux RB, Izzo R, et al. Lack of reduction of left ventricular mass in treated hypertension: the strong heart study. *J Am Heart Assoc*. 2013;2(3):e000144.
5. Devereux RB, de Simone G, Ganau A, Roman MJ. Left ventricular hypertrophy and geometric remodeling in hypertension: stimuli, functional consequences and prognostic implications. *J Hypertens Suppl*. 1994;12(10):S117-S127.
6. Kasiakogias A, Tsioufis C, Dimitriadis K, et al. Cardiovascular morbidity of severe resistant hypertension among treated uncontrolled hypertensives: a 4-year follow-up study. *J Hum Hypertens*. 2018;32(7):487-493.

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7. Cuspidi C, Facchetti R, Bombelli M, et al. High normal blood pressure and left ventricular hypertrophy echocardiographic findings from the PAMELA population. *Hypertension*. 2019;73(3):612-619.
8. Ganau A, Devereux RB, Roman MJ, et al. Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension. *J Am Coll Cardiol*. 1992;19(7):1550-1558.
9. Ng M, Fleming T, Robinson M, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the global burden of disease study 2013. *Lancet*. 2014;384(9945):766-781.
10. Pan XF, Wang L, Pan A. Epidemiology and determinants of obesity in China. *Lancet Diabetes Endocrinol*. 2021;9(6):373-392.
11. Banerjee R, Rial B, Holloway CJ, et al. Evidence of a direct effect of myocardial steatosis on LV hypertrophy and diastolic dysfunction in adult and adolescent obesity. *JACC Cardiovasc Imaging*. 2015;8(12):1468-1470.
12. Savji N, Meijers WC, Bartz TM, et al. The association of obesity and cardiometabolic traits with incident HFpEF and HFrEF. *JACC Heart Fail*. 2018;6(8):701-709.
13. Bartkowiak J, Spitzer E, Kurmann R, et al. The impact of obesity on left ventricular hypertrophy and diastolic dysfunction in children and adolescents. *Sci Rep*. 2021;11(1):13022.
14. Lear SA, Lesser IA. A review of obesity and body fat distribution and its relationship to cardio-metabolic risk in men and women of Chinese origin. *Cardiovasc Hematol Disord Drug Targets*. 2012;12(2):113-118.
15. Ge Q, Qi Z, Xu Z, et al. Comparison of different obesity indices related with hypertension among different sex and age groups in China. *Nutr Metab Cardiovasc Dis*. 2021;31(3):793-801.
16. Neeland IJ, Ross R, Després JP, et al. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol*. 2019;7(9):715-725.
17. Du Z, Xing L, Ye N, Lin M, Sun Y. Complementary value of ECG and echocardiographic left ventricular hypertrophy for prediction of adverse outcomes in the general population. *J Hypertens*. 2021;39(3):548-555.
18. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. *Ann Intern Med*. 2009;150(9):604-612.
19. Nishida C, Ko GT, Kumanyika S. Body fat distribution and noncommunicable diseases in populations: overview of the 2008 WHO expert consultation on waist circumference and waist-hip ratio. *Eur J Clin Nutr*. 2010;64(1):2-5.
20. Fuchs FD, Scala L, Vilela-Martin JF, et al. Effectiveness of chlorthalidone/amiloride versus losartan in patients with stage I hypertension and diabetes mellitus: results from the PREVER-treatment randomized controlled trial. *Acta Diabetol*. 2021;58(2):215-220.
21. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH guidelines for the management of arterial hypertension: the task force for the management of arterial hypertension of the European Society of Cardiology and the European Society of Hypertension. *J Hypertens*. 2018;36(10):1953-2041.
22. Izzo R, Losi MA, Stabile E, et al. Development of left ventricular hypertrophy in treated hypertensive outpatients: the Campania Salute Network. *Hypertension*. 2017;69(1):136-142.
23. De Simone G, Devereux RB, Chinali M, et al. Sex differences in obesity-related changes in left ventricular morphology: the Strong Heart Study. *J Hypertens*. 2011;29(7):1431-1438.
24. Wang D, Li Y, Lee SG, et al. Ethnic differences in body composition and obesity related risk factors: study in Chinese and white males living in China. *PLoS One*. 2011;6(5):e19835.
25. He W, Li Q, Yang M, et al. Lower BMI cutoffs to define overweight and obesity in China. *Obesity (Silver Spring)*. 2015;23(3):684-691.
26. Chirinos JA, Segers P, De Buyzere ML, et al. Left ventricular mass: allometric scaling, normative values, effect of obesity, and prognostic performance. *Hypertension*. 2010;56(1):91-98.
27. Zhou D, Yan M, Cai A, et al. Prognostic implications of left ventricular hypertrophy defined by the thresholds from the international and Chinese guidelines. *J Clin Hypertens (Greenwich)*. 2023;25(7):628-637.
28. Liu J, Fox CS, Hickson DA, et al. Impact of abdominal visceral and subcutaneous adipose tissue on cardiometabolic risk factors: the Jackson Heart Study. *J Clin Endocrinol Metab*. 2010;95(12):5419-5426.
29. Xing L, Liu S, Jing L, et al. Trends in prevalence, awareness, treatment, and control of hypertension in rural northeast China: 2008 to 2018. *Biomed Res Int*. 2020;2020:1456720.
30. Böhm C, Benz V, Clemenz M, et al. Sexual dimorphism in obesity-mediated left ventricular hypertrophy. *Am J Physiol Heart Circ Physiol*. 2013;305(2):H211-H218.
31. Kararigas G, Bito V, Tinel H, et al. Transcriptome characterization of estrogen-treated human myocardium identifies myosin regulatory light chain interacting protein as a sex-specific element influencing contractile function. *J Am Coll Cardiol*. 2012;59(4):410-417.
32. Tiyerili V, Müller CF, Fung S, Panek D, Nickenig G, Becher UM. Estrogen improves vascular function via peroxisome-proliferator-activated-receptor-γ. *J Mol Cell Cardiol*. 2012;53(2):268-276.
33. Zhai Y, Fang HY, Yu WT, et al. Epidemiological characteristics of waist circumference and abdominal obesity among Chinese adults in 2010–2012 [in Chinese]. *Zhonghua Yu Fang Yi Xue Za Zhi*. 2017;51(6):506-512.

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