Body Mass Index or Waist-Hip Ratio, Which Correlated With Arterial Stiffness Based on Brachial-Ankle Pulse Wave Velocity in Chinese Rural Adults With Hypertension?

Feng Hu

Department of Cardiovascular Medicine, the Second Affiliated Hospital of Nanchang University

Rihua Yu

Department of General Practice Medicine, Zhentou Town Health Center

Fengyu Han

Department of Cardiovascular Medicine, the Second Affiliated Hospital of Nanchang University

Juan Li

The Department of pharmacy, the second affiliated hospital of Nanchang University

Wei Zhou

Center for Prevention and Treatment of Cardiovascular Diseases, the Second Affiliated Hospital of Nanchang University

Tao Wang

Center for Prevention and Treatment of Cardiovascular Diseases, the Second Affiliated Hospital of Nanchang University

Lingjuan Zhu

Center for Prevention and Treatment of Cardiovascular Diseases, the Second Affiliated Hospital of Nanchang University

Xiao Huang

Department of Cardiovascular Medicine, the Second Affiliated Hospital of Nanchang University

Huihui Bao

Department of Cardiovascular Medicine, the Second Affiliated Hospital of Nanchang University

Xiaoshu Cheng (✉ xiaoshumenfan126@163.com)

Department of Cardiovascular Medicine, the Second Affiliated Hospital of Nanchang University

Research Article

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Abstract

Background: The aim of this study was to investigate the association between body mass index (BMI), waist-hip ratio (WHR) and arterial stiffness (AS) based on brachial-ankle pulse wave velocity (baPWV) in Chinese rural adults with hypertension.

Methods: In this analysis, selected 5,049 Chinese rural adults with hypertension were divided into three groups according to BMI ($<24 \text{ kg/m}^2$, control; $24\text{--}28 \text{ kg/m}^2$, overweight; and $\geq 28 \text{ kg/m}^2$, obesity), WHR ≥ 0.9 for male and ≥ 0.85 for female was defined as central obesity, while $\text{baPWV} \geq 18.0 \text{ m/s}$ was considered as increased AS. Multivariate analysis was used to examine the association between BMI, WHR (central obesity) and AS based on baPWV in different models. Furthermore, the generalized additive model and smooth curve fitting was used to visually show the relationship between BMI or WHR with baPWV. Finally, to ensure the robustness between BMI group or central obesity with increased AS, we also did the subgroup analyses that were performed using stratified multivariate regression and interaction analyses and presented in tabulated form or forest plot.

Results: The prevalence of overweight, general obesity, central obesity and increased AS were 32.62%, 8.58%, 63.85% and 44.01%, respectively. In comparison with control group, there are a statistically significant lower prevalence of increased AS in population with overweight or general obesity (adjusted-OR: 0.78, 95% CI 0.65 to 0.92, $P < 0.001$; adjusted-OR: 0.54, 95% CI 0.40 to 0.72, $P < 0.001$, respectively; P for trend < 0.001). Whereby in comparison with non-central obesity group, there are an statistically significant higher prevalence of increased AS in population with central obesity (adjusted-OR: 1.54, 95% CI 1.30 to 1.83, $P < 0.001$). The multivariate analyses indicated that BMI was negatively associated with baPWV (adjusted- β per SD increase: -0.49 , 95% CI -0.60 to -0.38 , $P < 0.001$). In comparison with control group, there are an statistically significant inversely relationship between BMI and baPWV in population with overweight or general obesity (adjusted- β : -0.55 , 95% CI -0.75 to -0.35 , $P < 0.001$; adjusted- β : -1.00 , 95% CI -1.32 to -0.67 , $P < 0.001$, respectively; P for trend < 0.001). On the contrary, WHR was positively associated with baPWV (adjusted- β per SD increase: 0.27 , 95% CI 0.17 to 0.38 , $P < 0.001$). In comparison with non-central obesity group, there are a statistically significant positively relationship between WHR and baPWV in population with central obesity (adjusted- β : 0.55 , 95% CI 0.34 to 0.75 , $P < 0.001$).

Conclusion: We found that there was an inversely relationship between BMI and baPWV or increased AS, whereas WHR or central obesity is positively associated with baPWV and increased AS in Chinese rural adults with hypertension.

Background

Cardiovascular disease (CVD) is the leading cause of death in the United States [1]. There is evidence that obesity is associated with increased risk of CVD [1] and other cardiovascular risk factors such as hypertension, diabetes, and dyslipidemia [2, 3]. The prevalence of global obesity is steadily increasing in the past few decades [4] and it has been responsible for 10–40% of all CVD deaths [5].

Atherosclerosis would be the main mechanism linking obesity to CVD [6, 7]. As part of the atherosclerotic process, there is an increase in arterial stiffness (AS), which has been associated with an increase in pulse wave velocity (PWV) [8–10]. In recent decades, PWV as a measure of arterial stiffness [11] has been increasingly used for CVD risk assessment [12, 13]. Brachial-ankle pulse wave velocity (baPWV), considered as an unique indicator in assessing the degree of arteriosclerosis, has been gradually adopted as an epidemiological investigation method because of its simplicity and reproducibility [11].

Body mass index (BMI) is one of the main measures for general obesity, and is commonly used in clinical assessment and screenings. However, researches on the association between BMI and PWV in various population including the hypertensive patients [14–18] which often accompanied with overweight or obesity [19], remain controversial: studies have reported an insignificant relationship [14, 18, 20–24], positive relationship [15, 17, 25–28], or negative relationship [16, 29, 30–33]. Many causes could interpret these apparent discrepancies including diverse sample size, different origins of the study population, ethnic and regional disparity. On the other hand, BMI is not able to differentiate fat-free muscle from fat mass, or account for sex and racial differences in fat content and distribution of visceral and subcutaneous fat [34]. Thus, the impact that BMI and obesity have on hypertensive patients needs further study.

Waist-hip ratio (WHR), a measure of central obesity and abdominal fat, may be better indicate risk for atherosclerosis or CVD associated with obesity than other anthropometric measures, including BMI [35–36], as WHR is potentially less influenced by muscle and bone mass. The higher WHR is often accompanied with increased arterial stiffening measured by PWV [25, 27]. Moreover, China bears the biggest hypertension burden in the world and hypertension is often accompanied with overweight or obesity [19]. Furthermore, because rural adults engage in stronger physical labor in the long run, this crowd have more muscular tissue content and better physical fitness than civil servants, thus we consider that BMI might not be suitable for the evaluation of obesity and to indicate risk for atherosclerosis in Chinese rural adults with hypertension. We therefore analysed the association between BMI, WHR and arterial stiffness based on baPWV in Chinese rural adults with hypertension.

Methods

Study design and participants

Data analyzed in this study was the baseline of the ongoing China H-type Hypertension Registry Study (Registration number: ChiCTR1800017274). The method of data collection and the exclusion criteria have been described previously [37]. Briefly, the study is a real-world, multicenter, observational study, conducted from Wuyuan, Jiangxi province of China, which conducted in March 2018. Eligible participants were adults aged 18 years and older who had hypertension, defined as seated, resting systolic blood pressure (SBP) ≥ 140 mmHg or diastolic blood pressure (DBP) ≥ 90 mmHg at the screening, or who were on antihypertensive medications. The exclusion criteria included neurological abnormalities, unable to be followed-up according to the study protocol, or plans to relocate shortly, and the patients, who are not

suitable for inclusion or for long-term follow-up as assessed by study physicians. The study was conducted in accordance with the Declaration of Helsinki, and the protocol was approved by the Ethics Committee of Institute of Biomedicine, Anhui Medical University. All participants provided written informed consent.

In this study, a total of 5,233 participants completed the measurement of baPWV. In this analysis, we excluded 78 individuals with ABI < 0.9 (11,24) (in cases of peripheral arterial disease, the reliability of baPWV measurement is attenuated) and 104 patients with atrial fibrillation as well as 2 participants without BMI measurements, finally 5,049 participants were included in our analysis (Supplementary Fig. 1).

Data collection and indexes determination

Participants' demographics characteristics (age, gender), lifestyle (smoking and drinking status), past medical history [such as hypertension, diabetes mellitus (DM), dyslipidemia and atrial fibrillation (AF)] and medication usage (such as antihypertensive agents, hypoglycemic agents, lipid-lowering agents and antiplatelet agents) through questionnaires were collected by trained research staff. AF is diagnosed by medical history and resting supine standard 12-lead surface electrocardiograms (25 mm/s, 10 mm/mV).

They also collected participants' anthropometric measurement indicators included weight, height, waistline, hipline, systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR). Due to the health screening protocols, single point measurements of anthropometric indicators were obtained. Waistline and hipline were measured using an inelastic measuring tape, with 0.1 cm resolution and length of 1.5 m. Individuals were at upright position, with abdomen relaxed at the end of gentle expiration. Four consecutive office BP measurements (each time interval was 1–2 min) were taken for each individual with a validated non-invasive electronic oscillometric device using the appropriate cuff size for the upper right arm. The mean values of the last three readings were used for analysis in order to reduce the impact of reactivity on the blood pressure (higher first reading). BMI was calculated as the body weight in kilograms divided by the square of the height in meters (kg/m^2). WHR was calculated as the waistline in centimeters divided by the hipline in centimeters. In our study, Subjects were divided into three groups according to BMI (< 24 kg/m^2 , control; 24–28 kg/m^2 , overweight; and $\geq 28 \text{ kg}/\text{m}^2$, obesity) based on published values for the Chinese population [29, 38]. Furthermore, a WHR of < 0.9 for male and < 0.85 for female was defined as normal, while ≥ 0.9 for male and ≥ 0.85 for female was defined as central obesity [38].

Blood samples were collected utilizing venipuncture after an overnight fast of at least 12 hours. Blood biochemical tests for plasma total homocysteine, fasting blood glucose (FBG), total cholesterol (TC), total triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), serum uric acid and creatinine, blood urea nitrogen (BUN), total and direct bilirubin, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) measured using automatic clinical analyzers (Beckman Coulter) at the core laboratory of the National Clinical Research Center for Kidney Disease, Guangzhou, China. All laboratory measurements met a standardization and certification

program. In our study, DM was defined as the FBG levels above 7.0 mmol/L or on treatment for diagnosed DM formerly. Dyslipidemia was defined as having one of the following features: elevated TG (≥ 2.3 mmol/L), TC (≥ 6.2 mmol/L), LDL-C (≥ 4.1 mmol/L), reduced HDL-C (< 1.0 mmol/L) or on appropriate lipid-lowering agents treatment formerly. The formula for estimated glomerular filtration rate (eGFR) used the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation which more accurate than Modification of Diet in Renal Disease (MDRD) equation for subjects at higher eGFR levels.

Measurement of PWV and definition of arterial stiffness

For baPWV values we used a BP-203 RPE III networked arteriosclerosis detection device (Omron Health Care, Kyoto, Japan). Participants underwent baPWV measurement after at least 5 minutes of rest in the supine position in a quiet room. Coffee, tea, cigarette use, or alcohol use were not allowed for 30 minutes before the test. Blood pressure cuffs were wrapped on both arms and ankles and connected to a plethysmographic sensor to determine the volume pulse waveform. The lower edge of the arm cuff was positioned 2–3 cm above the cubital fossa transverse striation, while the lower edge of the ankle cuff was positioned 1–2 cm above the medial malleolus. The heart beat monitor was placed on the left edge of the sternum, and electrocardiogram electrodes value directly. The methodology for baPWV measurement remained the same for all participants. The average of baPWV measured on bilateral limbs of each patient was used for analysis. While baPWV value has been shown to have prognostic significance, with a threshold of increased risk that exists around 18 m/s [11]. Therefore, baPWV ≥ 18 m/s was considered as increased AS in our study.

Statistical Analysis

Clinical characteristics of participants were grouped by BMI group or central obesity. Continuous variables were expressed as means $\pm$ standard deviation (SD) or median (Q1- Q3). For the variables in both groups that exhibit normal distribution and homoscedasticity, independent t-test or one-way analysis of variance were used. For the variables that do not show normal distribution, nonparametric test was used. Categorical variables were expressed as count (percentage), differences between groups were measured by chi-square test or Fisher's exact probability test.

Secondly, we used three different linear regression models to examine the associations of BMI or WHR with increased AS or baPWV. The crude model was not adjusted for any confounder. The model Ⅱ was adjusted for age, gender, SBP, DBP, HR, WHR or BMI except the independent variable itself. The model Ⅲ was confounder model. The confounder model screened covariates including age, gender, SBP, DBP, heart rate, BMI, WHR, DM, smoking and drinking status, homocysteine, TC, TG, HDL-C, LDL-C, AST, ALT, serum uric acid, eGFR, total and direct bilirubin, lipid-lowering agents and antiplatelet agents except the independent variable itself. We selected these confounders on the basis that, when added to this model, its changed the matched odds ratio by at least 10 percent. Supplementary tables 1 show the associations of each confounder with the outcomes of interest. We considered the confounder model to be the main model. In addition, we performed tests for linear trend by entering the median value of each category of BMI levels or categorical variables as a continuous variable in the models.

Furthermore, the generalized additive model and smooth curve fitting (penalized spline method) was used to visually show the relationship between BMI or WHR with baPWV (the selection principle of confounders was equal to confounder model). Finally, to ensure the robustness between BMI group or central obesity with increased AS, we also did the subgroup analyses that were performed using stratified multivariate regression and interaction analyses and presented in tabulated form or forest plot.

All statistical analyses were performed using the statistical package R (<http://www.R-project.org>, The R Foundation) and the Empower (R; www.empowerstats.com; X&Y Solutions, Inc, Boston, MA, USA). All P-values are two-tailed, and $P < 0.05$ was considered statistically significant.

Results

Clinical characteristics of the study population

The present study included 5,049 Chinese adult hypertensive individuals (age: 64.46 ± 9.45 years, range 29–93 years; male, 49.89%), and the prevalence of overweight, general obesity, central obesity and increased AS were 32.62%, 8.58%, 63.85% and 44.01%, respectively. The clinical characteristics of the study participants were presented in Table 1 grouped by BMI group or central obesity, Supplement Table 2 grouped by baPWV quartiles.

Table 1
Clinical characteristics of participants grouped by BMI group or central obesity

Characteristics	BMI group (kg/m ²)			P-value	Central obesity		P-value
	Control (n = 2969)	Overweight (n = 1647)	Obesity (n = 433)		No (n = 1825)	Yes (n = 3224)	
Age (years)	66.40 ± 9.01	62.22 ± 9.22	59.70 ± 9.68	< 0.001	64.98 ± 9.61	64.16 ± 9.35	0.004
Male, n(%)	1521 (51.23%)	818 (49.67%)	180 (41.57%)	< 0.001	1183 (64.82%)	1336 (41.44%)	< 0.001
SBP (mmHg)	147.48 ± 18.22	146.09 ± 16.61	146.95 ± 16.29	0.029	147.05 ± 18.26	146.94 ± 17.15	0.845
DBP (mmHg)	87.32 ± 10.98	90.23 ± 10.42	92.36 ± 10.90	< 0.001	88.24 ± 11.14	88.97 ± 10.80	0.054
HR (times/min)	75.10 ± 14.65	76.30 ± 14.10	78.16 ± 13.04	< 0.001	74.16 ± 14.44	76.66 ± 14.26	< 0.001
Height (cm)	155.45 ± 8.12	156.66 ± 8.19	155.84 ± 8.24	< 0.001	157.35 ± 7.72	155.05 ± 8.30	< 0.001
Weigh (Kg)	50.97 ± 7.38	63.19 ± 7.21	73.32 ± 8.84	< 0.001	52.29 ± 8.85	59.46 ± 10.64	< 0.001
BMI (kg/m ²)	21.02 ± 2.03	25.67 ± 1.10	30.14 ± 2.32	< 0.001	21.04 ± 2.70	24.61 ± 3.21	< 0.001
BMI group (kg/m ²)							< 0.001
Control (< 24)					1574 (86.25%)	1395 (43.27%)	
Overweight (≥ 24, < 28)					234 (12.82%)	1413 (43.83%)	
Obesity (≥ 28)					17 (0.93%)	416 (12.90%)	
Waistline (cm)	76.84 ± 7.16	88.46 ± 5.49	96.51 ± 6.95	< 0.001	74.34 ± 7.10	86.84 ± 7.62	< 0.001
Hipline (cm)	88.08 ± 4.89	95.13 ± 5.07	101.01 ± 6.84	< 0.001	89.28 ± 6.46	92.74 ± 6.57	< 0.001
WHR	0.87 ± 0.06	0.93 ± 0.06	0.96 ± 0.07	< 0.001	0.83 ± 0.04	0.94 ± 0.05	< 0.001
Central obesity, n(%)	1395 (46.99%)	1413 (85.79%)	416 (96.07%)	< 0.001			

Characteristics	BMI group (kg/m ²)			<i>P</i> -value	Central obesity		<i>P</i> -value
	Control (n = 2969)	Overweight (n = 1647)	Obesity (n = 433)		No (n = 1825)	Yes (n = 3224)	
baPWV (m/s)	18.53 ± 4.06	17.54 ± 3.47	17.03 ± 3.50	< 0.001	17.94 ± 3.85	18.16 ± 3.89	0.010
Increased AS, n(%)	1424 (47.96%)	652 (39.59%)	146 (33.72%)	< 0.001	743 (40.71%)	1479 (45.87%)	< 0.001
Smoking status, n(%)				< 0.001			< 0.001
Never	1470 (49.51%)	953 (57.86%)	268 (61.89%)		766 (41.97%)	1925 (59.71%)	
Former smoker	533 (17.95%)	336 (20.40%)	63 (14.55%)		373 (20.44%)	559 (17.34%)	
Current smoker	966 (32.54%)	358 (21.74%)	102 (23.56%)		686 (37.59%)	740 (22.95%)	
Drinking status, n(%)				0.188			< 0.001
Never	1840 (61.97%)	1069 (64.91%)	285 (65.82%)		1043 (57.15%)	2151 (66.72%)	
Former drinker	380 (12.80%)	183 (11.11%)	46 (10.62%)		259 (14.19%)	350 (10.86%)	
Current drinker	749 (25.23%)	395 (23.98%)	102 (23.56%)		523 (28.66%)	723 (22.43%)	
Homocysteine (μmol/L)	15.36 (12.66–20.20)	15.07 (12.50–19.16)	14.24 (12.27–17.84)	< 0.001	15.62 (12.79–21.14)	14.91 (12.50–19.02)	< 0.001
FBG (mmol/L)	5.95 ± 1.38	6.37 ± 1.95	6.55 ± 1.69	< 0.001	5.80 ± 1.10	6.33 ± 1.84	< 0.001
TC (mmol/L)	5.04 ± 1.09	5.21 ± 1.13	5.36 ± 1.16	< 0.001	4.96 ± 1.07	5.22 ± 1.13	< 0.001
TG (mmol/L)	1.22 (0.90–1.74)	1.70 (1.22–2.44)	1.91 (1.35–2.86)	< 0.001	1.12 (0.83–1.56)	1.63 (1.16–2.35)	< 0.001
HDL-C (mmol/L)	1.56 ± 0.42	1.40 ± 0.35	1.37 ± 0.33	< 0.001	1.60 ± 0.43	1.43 ± 0.36	< 0.001
LDL-C (mmol/L)	2.80 ± 0.77	3.08 ± 0.79	3.22 ± 0.83	< 0.001	2.71 ± 0.74	3.04 ± 0.80	< 0.001

Characteristics	BMI group (kg/m ²)			<i>P</i> -value	Central obesity		<i>P</i> -value
	Control (n = 2969)	Overweight (n = 1647)	Obesity (n = 433)		No (n = 1825)	Yes (n = 3224)	
Serum uric acid (μmol/L)	417.69 ± 117.52	448.82 ± 126.40	460.18 ± 114.07	< 0.001	422.65 ± 116.69	436.49 ± 123.63	< 0.001
Serum creatinine (mmol/L)	68.00 (56.00–83.00)	69.00 (57.00–84.50)	66.00 (55.00–80.00)	0.036	71.00 (59.00–86.00)	66.00 (55.00–81.00)	< 0.001
BUN (mmol/L)	5.52 ± 1.89	5.38 ± 1.80	5.17 ± 1.50	< 0.001	5.58 ± 2.05	5.36 ± 1.70	0.002
eGFR (ml/min/1.73 m ²)	84.91 ± 19.42	87.06 ± 20.01	90.10 ± 18.71	< 0.001	85.33 ± 20.32	86.47 ± 19.19	0.120
Total bilirubin (mmol/L)	14.19 ± 6.27	14.72 ± 6.53	14.04 ± 6.02	0.022	14.42 ± 6.24	14.31 ± 6.40	0.306
Direct bilirubin (mmol/L)	5.37 ± 2.09	5.38 ± 2.03	5.18 ± 1.83	0.361	5.54 ± 2.13	5.26 ± 2.00	< 0.001
AST (U/L)	24.00 (20.00–29.00)	24.00 (20.00–30.00)	25.00 (21.00–34.00)	< 0.001	24.00 (20.00–30.00)	24.00 (20.00–30.00)	0.212
ALT (U/L)	15.00 (12.00–21.00)	19.00 (14.00–27.00)	23.00 (17.00–34.00)	< 0.001	15.00 (12.00–21.00)	18.00 (13.00–26.00)	< 0.001
DM, n(%)	431 (14.52%)	376 (22.83%)	128 (29.56%)	< 0.001	199 (10.90%)	736 (22.83%)	< 0.001
Dyslipidemia, n(%)	857 (28.86%)	759 (46.08%)	235 (54.27%)	< 0.001	440 (24.11%)	1411 (43.77%)	< 0.001
Antihypertensive agents, n(%)	1721 (57.97%)	1089 (66.12%)	268 (61.89%)	< 0.001	1037 (56.82%)	2041 (63.31%)	< 0.001
Hypoglycemic agents, n(%)	96 (3.23%)	103 (6.25%)	25 (5.77%)	< 0.001	30 (1.64%)	194 (6.02%)	< 0.001
Lipid-lowering agents, n(%)	77 (2.59%)	78 (4.74%)	17 (3.93%)	< 0.001	47 (2.58%)	125 (3.88%)	0.014
Antiplatelet agents, n(%)	81 (2.73%)	86 (5.22%)	17 (3.93%)	< 0.001	58 (3.18%)	126 (3.91%)	0.184
Abbreviations: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; WHR, waist hip rate; baPWV, brachial-ankle pulse wave velocity; AS, arterial stiffness; FBG, fasting blood glucose; TC, total cholesterol; TG, total triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; AST, aspartate aminotransferase; ALT, alanine aminotransferase; DM, diabetes mellitus.							

Table 2
Relationship between BMI and increased AS in different models

Variables	Event, n(%)	Crude Model		Model Ⅰ		Model Ⅱ	
		OR (95%CI)	P-value	OR (95%CI)	P-value	OR (95%CI)	P-value
BMI (kg/m^2)							
Per 1 increase	2222 (44.01%)	0.93 (0.91, 0.94)	< 0.001	0.94 (0.92, 0.96)	< 0.001	0.92 (0.89, 0.94)	< 0.001 ^a
Per SD increase		0.77 (0.73, 0.82)	< 0.001	0.80 (0.74, 0.88)	< 0.001	0.75 (0.68, 0.82)	< 0.001 ^a
BMI group (kg/m^2)							
Control (< 24)	1424 (47.96%)	Ref		Ref		Ref	
Overweight (≥ 24, < 28)	652 (39.59%)	0.71 (0.63, 0.80)	< 0.001	0.84 (0.71, 0.99)	0.034	0.78 (0.65, 0.92)	0.003 ^b
Obesity (≥ 28)	146 (33.72%)	0.55 (0.45, 0.68)	< 0.001	0.61 (0.46, 0.80)	< 0.001	0.54 (0.40, 0.72)	< 0.001 ^b
P for trend		< 0.001		< 0.001		< 0.001	
BMI tertile (kg/m^2)							
T1 [13.83, 21.68]	877 (52.11%)	Ref		Ref		Ref	
T2 [21.68, 24.69]	715 (42.48%)	0.68 (0.59, 0.78)	< 0.001	0.81 (0.67, 0.96)	0.018	0.73 (0.60, 0.87)	< 0.001 ^a
T3 [24.69, 46.43]	630 (37.43%)	0.55 (0.48, 0.63)	< 0.001	0.67 (0.54, 0.82)	< 0.001	0.57 (0.46, 0.71)	< 0.001 ^a
P for trend		< 0.001		< 0.001		< 0.001	
BMI quartile (kg/m^2)							
Q1 [13.83, 20.90]	666 (52.82%)	Ref		Ref		Ref	
Q2 [20.90, 23.22]	576 (45.61%)	0.75 (0.64, 0.88)	< 0.001	0.89 (0.73, 1.09)	0.254	0.83 (0.68, 1.01)	0.065 ^a
Q2 [23.22, 25.53]	506 (40.10%)	0.60 (0.51, 0.70)	< 0.001	0.79 (0.64, 0.98)	0.033	0.69 (0.55, 0.86)	< 0.001 ^a

Variables	Event, n(%)	Crude Model		Model Ⅹ		Model Ⅹ	
		OR (95%CI)	P-value	OR (95%CI)	P-value	OR (95%CI)	P-value
Q4 [25.53, 46.43]	474 (37.53%)	0.54 (0.46, 0.63)	< 0.001	0.70 (0.55, 0.89)	0.004	0.58 (0.46, 0.75)	< 0.001 ^a
P for trend		< 0.001		0.003		< 0.001	
Abbreviations: BMI, body mass index; AS, arterial stiffness; <i>Ref</i> , reference; OR, odds ratio; CI, confidence interval; SD, standard deviation.							
Model Ⅹ adjusted for age, gender, SBP, DBP, HR and WHR.							
Model Ⅹ: ^a adjusted for age, SBP, DBP, HR, WHR, DM, smoking status, TG, LDL-C and eGFR. ^b adjusted for age, SBP, DBP, HR, WHR, DM, smoking status, LDL-C, ALT and eGFR.							

On the one hand, the participants with overweight or general obesity were more likely to be central obesity, had lower extents of baPWV and lower prevalence of increased AS. On the other hand, the subjects with central obesity were more likely to be overweight or general obesity, had elevated extents of baPWV and higher prevalence of increased AS (Table 1). There were statistically significant differences in the above clinical characteristics among different groups ($P < 0.05$). Furthermore, Compared to $\text{baPWV} \leq 15.36$ m/s, there were a statistically significant elevated extents of SBP and DBP, lower prevalence of overweight or general obesity as well as higher prevalence of central obesity in the third and highest baPWV quartiles (Supplement Table 2).

Associations between BMI and increased AS

The multivariate analyses indicated that BMI was negatively associated with the risk of increased AS (adjusted-OR per SD increase: 0.75, 95% CI 0.68 to 0.82, $P < 0.001$; Table 2). In comparison with control group, there are a statistically significant lower prevalence of increased AS in population with overweight or general obesity (adjusted-OR: 0.78, 95% CI 0.65 to 0.92, $P < 0.001$; adjusted-OR: 0.54, 95% CI 0.40 to 0.72, $P < 0.001$, respectively; P for trend < 0.001). Compared to $\text{BMI} \leq 20.90$ kg/m², there were a statistically significant lower prevalence of increased AS for the participants in the third and highest BMI quartiles (adjusted-OR: 0.69, 95% CI 0.55 to 0.86, $P < 0.001$; adjusted-OR: 0.58, 95% CI 0.46 to 0.75, $P < 0.001$, respectively; P for trend < 0.001).

Associations between WHR and increased AS

The multivariate analyses indicated that WHR was positively associated with the risk of increased AS (adjusted-OR per SD increase: 1.24, 95% CI 1.14 to 1.35, $P < 0.001$; Table 3). In comparison with non-central obesity group, there are a statistically significant higher prevalence of increased AS in population with central obesity (adjusted-OR: 1.54, 95% CI 1.30 to 1.83, $P < 0.001$). Compared to $\text{WHR} \leq 0.85$, there were a statistically significant higher prevalence of increased AS for the participants in the highest BMI quartiles (adjusted-OR: 1.71, 95% CI 1.06 to 2.77, $P = 0.029$; P for trend < 0.001).

Table 3
Relationship between WHR and increased AS in different models

Variables	Event, n(%)	Crude Model		Model Ⅰ		Model Ⅱ	
		OR (95%CI)	P-value	OR (95%CI)	P-value	OR (95%CI)	P-value
WHR							
Per 0.1 increase	2222 (44.01%)	0.44 (0.20, 0.98)	< 0.001	3.3 (0.99, 11.01)	< 0.001	2.07 (0.62, 6.91)	< 0.001 ^a
Per SD increase		1.11 (1.05, 1.18)	< 0.001	1.28 (1.18, 1.40)	< 0.001	1.24 (1.14, 1.35)	< 0.001 ^a
Central obesity							
No	743 (40.71%)	Ref		Ref		Ref	
Yes	1479 (45.87%)	1.23 (1.10, 1.39)	< 0.001	1.62 (1.36, 1.92)	< 0.001	1.54 (1.30, 1.83)	< 0.001 ^b
WHR tertile							
T1 [0.53, 0.87]	697 (41.41%)	Ref		Ref		Ref	
T2 [0.87, 0.93]	716 (42.70%)	1.05 (0.92, 1.21)	0.452	1.41 (1.18, 1.69)	< 0.001	1.34 (1.11, 1.61)	0.002 ^c
T3 [0.93, 1.68]	809 (47.90%)	1.30 (1.14, 1.49)	< 0.001	1.93 (1.57, 2.36)	< 0.001	1.76 (1.43, 2.17)	< 0.001 ^c
P for trend		< 0.001		< 0.001		< 0.001	
WHR quartile							
Q1 [0.53, 0.85]	534 (42.31%)	Ref		Ref		Ref	
Q2 [0.85, 0.90]	514 (40.76%)	0.94 (0.80, 1.10)	0.429	1.16 (0.95, 1.42)	0.151	1.21 (0.95, 1.55)	0.120 ^d
Q3 [0.90, 0.94]	553 (43.78%)	1.06 (0.91, 1.24)	0.456	1.67 (1.34, 2.07)	< 0.001	1.34 (0.96, 1.86)	0.085 ^d
Q4 [0.94, 1.68]	621 (49.17%)	1.32 (1.13, 1.54)	< 0.001	2.03 (1.61, 2.56)	< 0.001	1.71 (1.06, 2.77)	0.029 ^d
P for trend		< 0.001		< 0.001		< 0.001	

Variables	Event, n(%)	Crude Model		Model Ⅰ		Model Ⅱ	
		OR (95%CI)	P-value	OR (95%CI)	P-value	OR (95%CI)	P-value
Abbreviations: WHR, waist hip rate; AS, arterial stiffness; <i>Ref</i> , reference; OR, odds ratio; CI, confidence interval; SD, standard deviation.							
Model Ⅰ adjusted for age, gender, SBP, DBP, HR and BMI.							
Model Ⅱ: ^a adjusted for age, SBP, HR, BMI, DM, ALT and HDL-C. ^b adjusted for ^a + gender, smoking status and eGFR. ^c adjusted for ^b + DBP, LDL-C and serum uric acid. ^d adjusted for ^c + drinking status.							

Relationship between BMI or WHR and baPWV

The multivariate analyses indicated that BMI was negatively associated with baPWV (adjusted-β per SD increase: -0.49, 95% CI -0.60 to -0.38, $P < 0.001$; Table 4). In comparison with control group, there are a statistically significant inversely relationship between BMI and baPWV in population with overweight or general obesity (adjusted-β: -0.55, 95% CI -0.75 to -0.35, $P < 0.001$; adjusted-β: -1.00, 95% CI -1.32 to -0.67, $P < 0.001$, respectively; P for trend < 0.001). The generalized additive model and penalized spline method were used to find the negative relationship between BMI and baPWV (Fig. 1). On the contrary, the multivariate analyses indicated that WHR was positively associated with baPWV (adjusted-β per SD increase: 0.27, 95% CI 0.17 to 0.38, $P < 0.001$; Table 4). In comparison with non-central obesity group, there are a statistically significant positively relationship between WHR and baPWV in population with central obesity (adjusted-β: 0.55, 95% CI 0.34 to 0.75, $P < 0.001$). The generalized additive model and penalized spline method were used to find the positive relationship between WHR and baPWV (Fig. 2).

Table 4
Relationship between WHR or BMI and baPWV in different models

Variables	Crude Model		Model Ⅱ		Model Ⅲ	
	β (95%CI)	P-value	β (95%CI)	P-value	β (95%CI)	P-value
BMI (kg/m²)	<i>Ref</i>		<i>Ref</i>		<i>Ref</i>	
Per 1 increase	-0.21 (-0.24, -0.18)	< 0.001	-0.15 (-0.18, -0.12)	< 0.001	-0.14 (-0.17, -0.11)	< 0.001 ^a
Per SD increase	-0.72 (-0.82, -0.61)	< 0.001	-0.52 (-0.63, -0.42)	< 0.001	-0.49 (-0.60, -0.38)	< 0.001 ^a
BMI group (kg/m²)						
Control (< 24)	<i>Ref</i>		<i>Ref</i>		<i>Ref</i>	
Overweight (≥ 24, < 28)	-0.99 (-1.22, -0.76)	< 0.001	-0.52 (-0.72, -0.32)	< 0.001	-0.55 (-0.75, -0.35)	< 0.001 ^b
Obesity (≥ 28)	-1.50 (-1.89, -1.12)	< 0.001	-0.96 (-1.28, -0.63)	< 0.001	-1.00 (-1.32, -0.67)	< 0.001 ^b
P for trend	< 0.001		< 0.001		< 0.001	
WHR						
Per 0.1 increase	0.17 (0.02, 0.32)	0.030	0.50 (0.36, 0.65)	< 0.001	0.39 (0.24, 0.54)	< 0.001 ^c
Per SD increase	0.12 (0.01, 0.23)	0.030	0.36 (0.26, 0.46)	< 0.001	0.27 (0.17, 0.38)	< 0.001 ^c
Central obesity						
No	<i>Ref</i>				<i>Ref</i>	
Yes	0.22 (0.00, 0.44)	0.050	0.62 (0.42, 0.82)	< 0.001	0.55 (0.34, 0.75)	< 0.001 ^d

Abbreviations: WHR, waist hip rate; BMI, body mass index; baPWV, brachial-ankle pulse wave velocity; AS, arterial stiffness; *Ref*, reference; β , effect size; CI, confidence interval; SD, standard deviation.

Model Ⅱ adjusted for age, gender, SBP, DBP, HR, WHR or BMI except the independent variable itself.

Model Ⅲ: ^aadjusted for age, SBP, DBP, WHR and eGFR. ^badjusted for ^a + HR and DM. ^cadjusted for age, DBP, HR, BMI, DM, ALT, eGFR and HDL-C. ^dadjusted for ^c + gender, SBP, smoking and drinking status, homocysteine.

Subgroup analyses by potential effect modifiers

To explore whether the inverse association between BMI group and increased AS were still stable in different subgroups, we conducted the stratified and interaction analyses. The subgroup analyses showed that there were not statistically significant interactions in any of the subgroups, including age (< 60 vs. ≥ 60 years), sex (male vs. female), SBP dichotomy (≤ 145.3 vs. ≥ 145.3 mmHg), DBP dichotomy (≤ 88.7 vs. ≥ 89.0 mmHg), Central obesity (no vs. yes), DM (no vs. yes), smoking habit (no vs. yes), LDL (< 3.4 vs. ≥ 3.4 mmol/L), eGFR (≥ 60 vs. <60 ml/min) and ALT dichotomy (≤ 16.0 vs. ≥ 17.0 U/L) (all P for interactions > 0.05; Table 5).

Table 5
Effect size of BMI group on increased AS in prespecified and exploratory subgroups

Subgroup	Events, n(%)	Control (24 kg/m^2)	Overweight (≥ 24, < 28 kg/m^2)		Obesity (≥ 28 kg/m^2)		Interactive <i>P-value</i>
			OR (95% CI)	P- value	OR (95% CI)	P- value	
Age (years), tertile							0.784
T1 [29.0, 60.0]	335 (21.49%)	Ref	0.60 (0.43, 0.84)	0.003	0.40 (0.24, 0.65)	< 0.001	
T2 [61.0, 68.0]	773 (43.23%)	Ref	0.79 (0.61, 1.03)	0.081	0.55 (0.35, 0.86)	0.010	
T3 [69.0, 93.0]	1114 (65.45%)	Ref	0.75 (0.56, 1.00)	0.047	0.48 (0.28, 0.84)	0.011	
Gender							0.537
Male	1035 (41.09%)	Ref	0.85 (0.67, 1.09)	0.206	0.59 (0.37, 0.92)	0.020	
Female	1187 (46.92%)	Ref	0.70 (0.56, 0.89)	0.003	0.50 (0.35, 0.73)	< 0.001	
SBP (mmHg), dichotomy							0.467
Low [83.3, 145.3]	708 (28.30%)	Ref	0.81 (0.63, 1.03)	0.088	0.48 (0.30, 0.75)	0.001	
High [145.7, 255.0]	1514 (59.44%)	Ref	0.70 (0.56, 0.88)	0.002	0.56 (0.39, 0.80)	0.002	
DBP (mmHg), dichotomy							0.403
Low [41.7, 88.7]	1045 (41.93%)	Ref	0.81 (0.63, 1.03)	0.083	0.68 (0.44, 1.05)	0.080	
High [89.0, 129.7]	1177 (46.03%)	Ref	0.75 (0.60, 0.96)	0.020	0.46 (0.31, 0.67)	< 0.001	
Central obesity							0.910

Subgroup	Events, n(%)	Control (24 kg/m ²)	Overweight (≥ 24, < 28 kg/m ²)		Obesity (≥ 28 kg/m ²)		Interactive <i>P</i> -value
			OR (95% CI)	P- value	OR (95% CI)	P- value	
No	743 (40.71%)	<i>Ref</i>	0.78 (0.54, 1.13)	0.186	0.74 (0.19, 2.80)	0.654	
Yes	1479 (45.87%)	<i>Ref</i>	0.76 (0.63, 0.93)	0.006	0.54 (0.41, 0.73)	< 0.001	
DM							0.512
No	1723 (41.88%)	<i>Ref</i>	0.75 (0.62, 0.91)	0.003	0.57 (0.41, 0.80)	0.001	
Yes	499 (53.37%)	<i>Ref</i>	0.87 (0.60, 1.27)	0.480	0.48 (0.28, 0.83)	0.008	
Smoking habit							
No	1237 (45.97%)	<i>Ref</i>	0.68 (0.54, 0.85)	< 0.001	0.49 (0.34, 0.71)	< 0.001	0.224
Yes	985 (41.77%)	<i>Ref</i>	0.92 (0.71, 1.19)	0.531	0.61 (0.38, 0.96)	0.032	
LDL (mmol/L)							0.533
<3.4	1625 (42.96%)	<i>Ref</i>	0.73 (0.60, 0.88)	0.002	0.50 (0.35, 0.71)	< 0.001	
≥3.4	597 (47.16%)	<i>Ref</i>	0.91 (0.65, 1.27)	0.562	0.61 (0.37, 1.01)	0.052	
eGFR (ml/min/1.73 m²)							0.574
≥60.0	1896 (42.18%)	<i>Ref</i>	0.76 (0.64, 0.91)	0.003	0.56 (0.41, 0.75)	< 0.001	
<60.0	326 (58.84%)	<i>Ref</i>	0.86 (0.52, 1.42)	0.551	0.37 (0.14, 0.99)	0.048	

Subgroup	Events, n(%)	Control (24 kg/m ²)	Overweight (≥ 24, < 28 kg/m ²)		Obesity (≥ 28 kg/m ²)		Interactive <i>P</i> -value
			OR (95% CI)	P- value	OR (95% CI)	P- value	
ALT (U/L), dichotomy							0.199
Low [3.0, 16.0]	1150 (46.98%)	<i>Ref</i>	0.66 (0.51, 0.84)	0.001	0.51 (0.30, 0.85)	0.010	
High [17.0, 423.0]	1072 (41.21%)	<i>Ref</i>	0.90 (0.71, 1.13)	0.363	0.59 (0.41, 0.84)	0.004	
Abbreviations: AS, arterial stiffness; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; DM, diabetes mellitus; LDL-C, low-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate; ALT, alanine aminotransferase; <i>Ref</i> , reference; OR, odds ratio; CI, confidence interval.							
Each stratification adjusted for age, gender, SBP, DBP, HR, WHR, smoking status, DM, LDL-C, eGFR and ALT except the subgroup variable.							

Similarly, to explore whether the positive association between central obesity and increased AS were still stable in different subgroups, we conducted the stratified and interaction analyses. The subgroup analyses showed that there were not statistically significant interactions in any of the subgroups, including age (< 60 vs. ≥60 years), sex (male vs. female), SBP dichotomy (≤ 145.3 vs. ≥145.3 mmHg), BMI group (control vs. overweight vs. obesity), DM (no vs. yes), smoking habit (no vs. yes), HDL (≥ 1.0 vs. <1.0 mmol/L), eGFR (≥ 60 vs. <60 ml/min) and ALT dichotomy (≤ 16.0 vs. ≥17.0 U/L) (all *P* for interactions > 0.05; Fig. 3).

Discussion

In the current study, we presented an interesting finding that there was an inversely relationship between BMI and baPWV or increased AS in Chinese rural adults with hypertension, whereas WHR or central obesity is positively associated with baPWV and increased AS. To our knowledge, these findings have not been previously described.

Hypertension is often accompanied with overweight or obesity [19]. In our study, We found that the prevalence of overweight, general obesity, central obesity and were 32.62%, 8.58% and 63.85%, respectively. Moreover, PWV was increased in hypertensive patients, and the degree of PWV increase was associated with baseline blood pressure [39]. In our study, baPWV was also associated with most clinical characteristics including SBP and DBP, nearly half (44.01%) of the hypertensive participants have increased AS (baPWV ≥ 18 m/s) (Supplement Table 1 and 2), which has been considered to be an independent factor in indicating the relapse of CVD as well as an irreversible progress of atherosclerosis [11].

Obesity is an accredited dangerous factor for CVD [23]. However, some studies have shown that obesity as defined by BMI has a survival benefits on some specific populations [40]. The phenomenon of the obesity paradox may be related to genetics, cardiorespiratory fitness, beneficial adipose tissue and weaker sympathetic activation [34, 40].

In our study, we found that there was an inversely relationship between BMI and baPWV or increased AS in our crowd. Similarly, Huang et al. [29] and Liu et al. [18] found that there was a negative relationship between BMI and baPWV among male hypertension participants. On the contrary, previous studies showed that a higher BMI was associated with increased PWV in Grade I essential hypertension [15] or obese and non-obese hypertensive patients [17]. Different origins of the study participants might contribute to the study differences. The Liu et al. [18] study was based on 699 male hypertensive patients who were hospitalized or had other complications. The Samir et al. [15] study was based on 114 patients with Grade I essential hypertension who were civil servants. The Huang et al. [29] study enrolled 10 1510 participants, a coal occupation group in labor-intensive enterprise, from 11 hospitals in the Kailuan community, which is most similar to our rural hypertensive crowd. In consideration of that BMI fails to account for body composition [34] and physical habits, whereby rural adults are associated with increased muscle mass (muscle mass is greater than fat mass) and better physical fitness, both of which may account for the inversely relationship between BMI and baPWV or increased AS in our crowd.

WHR, a more sensitive marker for central obesity, may be better indicate risk for atherosclerosis or CVD associated with obesity than BMI [35, 36], as high WHR can reflect both increased visceral fat as well as low gluteal muscle mass (and/or low peripheral fat mass), both of which have been found to be independently associated with cardiovascular disease risk [41, 42]. We demonstrated that WHR or central obesity is positively associated with baPWV and increased AS in our crowd. The result was consistent with similar findings in the general population [25, 27]. For example, the Whitehall II study, a prospective study of 10 308 civil servants, showed that standardized effects of central adiposity on aortic PWV increase was obvious and previous adiposity was associated with aortic stiffening independent of change in adiposity, glycaemia, and lipid levels across PWV assessments [25].

Seidell et al. [43] demonstrated that WHR is highly correlated with visceral fat but not with subcutaneous fat (the latter which in fact may be protective), while waist circumference (WC) was highly correlated with both that. The incorporation of a reference body size may explain why WHR is a better predictor of outcomes than WC, a primitive measurement for abdominal obesity [35].

The lack of consistency between WHR and BMI may reflect that these measures identify different characteristics of obesity (central obesity in case of WHR vs. subcutaneous/total fat in case of BMI). Bouchi et al. [44], in a cross-sectional study with patients with diabetes and non-obese (normal BMI), noticed that increased visceral fat seems to be associated with increased AS based on baPWV. Notably, BMI fails to account for body composition, however, skeletal muscle mass [45] and physical fitness [46] counteract risk with obesity. As Chinses rural adults are associated with increased muscle mass and better physical fitness, thus BMI might not be suitable for the evaluation of obesity and the degree of

atherosclerosis may be underestimated by relying on BMI in this crowd, while WHR (central obesity) may be better indicate risk for atherosclerosis.

There are some strengths in our study, which should be considered in the interpretation of results. Firstly, the study was conducted in Wuyuan, a typical county in China, which offered us an exceptional opportunity to examine the association between obesity indicators and PWV in this crowd under a natural state. In addition, although the real-world, multicenter, observational study was susceptible to potential confounding, we used rigorous statistical adjustments to minimize residual confounding.

Limitations

Our study also has several limitations that should be considered. Firstly, the study was performed by cross-sectional design and so provides the association of relationships instead of predictive values of WHR on the progression of arterial stiffness. Secondly, results are limited by the absence of gold standard for measurement of visceral fat, such as computerized tomography (CT) and magnetic resonance imaging (MRI) for quantification of body fat composition. Furthermore, WHR measurements were performed by different individuals, although they were provided with clear instructions. Finally, this study was conducted in Chinese hypertension populations, the generalizability of the findings to other populations remains to be determined.

Conclusions

In conclusion, hypertension was often accompanied with central obesity and increased AS in Chinese rural adults with hypertension. We presented an interesting finding that there was a inversely relationship between BMI and baPWV or increased AS in this crowd, whereas WHR or central obesity is positively associated with baPWV and increased AS. Given that rural adults are associated with increased muscle mass and better physical fitness, BMI might not be suitable for the evaluation of obesity and the degree of atherosclerosis may be underestimated by relying on BMI in this crowd, whereas WHR (central obesity) may be a helpful tool for risk stratification in Chinese rural adults with hypertension. Additional studies should also evaluate whether intentional reduction in WHR, whether through exercise, medication or surgical intervention, is associated with a decrease in CVD events.

Abbreviations

BMI

body mass index; AS:arterial stiffness; CVD:Cardiovascular disease; SBP:systolic blood pressure; DBP:diastolic blood pressure; HR:heart rate; WHR:waist hip rate; WC:circumference; baPWV:brachial-ankle pulse wave velocity; AS:arterial stiffness; AF:atrial fibrillation; FBG:fasting blood glucose; TC:total cholesterol; TG:total triglyceride; HDL-C:high-density lipoprotein cholesterol; LDL-C:low-density lipoprotein cholesterol; BUN:blood urea nitrogen; eGFR:estimated glomerular filtration rate; AST:aspartate aminotransferase; ALT:alanine aminotransferase; DM:diabetes mellitus; CKD-EPI:Chronic Kidney Disease

Epidemiology Collaboration; MDRD:Modification of Diet in Renal Disease; CT:computerized tomography; MRI:magnetic resonance imaging; SD:standard deviation; OR:odds ratio; CI:confidence interval.

Declarations

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Not applicable.

Authors' contributions

FH, RHYJ, FYH and JL, contributions to conceptualization and design, or acquisition of data, or analysis and interpretation of data, involved in drafting, reviewing and editing the manuscript; WZ and TW, contributions to acquisition of data, involved in reviewing and editing the manuscript; LJZ and XH analysis and interpretation of data, involved in reviewing and editing the manuscript; HHB and XSC, revising it critically for important intellectual content. All authors read and approved the final manuscript.

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Availability of data and materials

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

Ethics approval and consent to participate

This study was approved by the ethics committee of the Institute of Biomedicine, Anhui Medical University, China (No. CH1059). Written and informed consent was obtained from the patients included in this study.

Consent for publication

Not applicable.

Competing interests

Not applicable.

Declarations

This study was approved by the ethics committee of the Institute of Biomedicine, Anhui Medical University, China (No. CH1059). Written and informed consent was obtained from the patients included in this study.

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Figures

Figure 1

Smooth curve of correlation between BMI and baPWV. Abbreviations: BMI, body mass index; baPWV, brachial-ankle pulse wave velocity. Smooth curve adjusted for age, SBP, DBP, eGFR and WHR.

Figure 2

Smooth curve of correlation between WHR and baPWV. Abbreviations: WHR, waist hip rate; baPWV, brachial-ankle pulse wave velocity. Smooth curve adjusted for age, DBP, HR, BMI, DM, ALT, eGFR and HDL-C.

Figure 3

Effect size of central obesity on increased AS in prespecified and exploratory subgroups. Abbreviations: AS, arterial stiffness; SBP, systolic blood pressure; BMI, body mass index; DM, diabetes mellitus; HDL-C, high-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate; ALT, alanine aminotransferase; OR, odds ratio; CI, confidence interval. Each stratification adjusted for age, gender, SBP, HR, BMI, smoking status, DM, HDL-C, eGFR and ALT except the subgroup variable.

Supplementary Files

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