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ORIGINAL RESEARCH

Association Between the Chinese Visceral Adiposity Index and Obstructive Sleep Apnoea in Hypertensive Patients with Normal-Weight: A Cross-Sectional Study

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Objective: Previous studies have mainly focused on the relationship between the Chinese Visceral Adiposity Index (CVAI) and obstructive sleep apnea (OSA) in general or overweight/obese populations. However, normal-weight hypertensive patients represent a clinically relevant yet understudied group, in whom OSA risk may be underestimated due to the absence of overt obesity. This study aimed to investigate the association between CVAI and OSA in normal-weight patients with hypertension, given the important role of visceral adiposity in the pathogenesis of OSA.

Materials and Methods: In this cross-sectional study, hypertensive patients admitted between January 1, 2021, and December 31, 2023, were enrolled. Multivariate logistic and linear regression analyses were conducted. Additionally, restricted cubic spline (RCS) analyses were used to examine potential dose–response relationships.

Results: A total of 6659 hypertensive patients were included. After adjusting for potential confounders, elevated CVAI levels were significantly associated with an increased risk and severity of OSA. RCS analyses demonstrated a clear linear dose–response relationship between CVAI and both the presence of OSA and apnea–hypopnea index (AHI) levels. These results were robust across both subgroup and sensitivity analyses.

Conclusion: Our study results indicate that CVAI is associated with the occurrence and severity of OSA in hypertensive patients with normal body weight. Further research is needed to confirm these findings.

Keywords: Chinese visceral adiposity index, obstructive sleep apnoea, apnoea-hypopnea index, cross-sectional study

Introduction

Obstructive sleep apnea (OSA) is a common sleep disorder characterized by recurrent upper airway obstruction during sleep.[1] While obesity is a major risk factor, OSA also has a substantial prevalence in normal-weight individuals.[2–5] Accumulating evidence indicates that visceral adipose tissue, which is deposited deep in the abdominal cavity and is prone to fostering chronic inflammation and metabolic disturbances, may play a more critical role in OSA pathogenesis than overall obesity.[6,7] Accordingly, assessing visceral adiposity is crucial for stratifying OSA risk, especially among normal-weight individuals.

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The Chinese Visceral Adiposity Index (CVAI), which integrates anthropometric and metabolic parameters, provides a more accurate estimation of visceral adiposity in Chinese adults than traditional measures.[8–10] CVAI has been shown to be a powerful predictor of metabolic diseases, and cardiovascular cerebrovascular disease.[11] Although prior studies have examined the relationship between CVAI and OSA in patients with diabetes mellitus (DM), data focusing on hypertensive patients—especially those with normal weight—remain scarce.[12] This research gap is important since OSA and hypertension frequently coexist and can mutually aggravate the risk of adverse cardiovascular outcomes.[9,13–15] Moreover, individuals with hypertension may display specific metabolic profiles and fat distribution patterns, potentially influencing OSA pathogenesis and clinical presentation.[16] Therefore, this study focuses on the population of patients with hypertension. Therefore, we conducted a cross-sectional study to investigate the relationship between CVAI levels and OSA in hypertensive patients.

Materials and Methods

Participants

This was a retrospective cross-sectional study using all eligible cases available; no a priori sample size calculation was performed. We enrolled patients diagnosed with hypertension who underwent polysomnography screening at the Hypertension Center of the People's Hospital of Xinjiang Uygur Autonomous Region from January 2021 to December 2023. A total of 9,951 patients with hypertension were initially included in the study. The exclusion criteria were as follows: age under 18 years; abnormal-weight; missing data related to CAVI calculation; pregnancy; use of sleeping pills; presence of serious debilitating diseases such as malignant tumors or significantly impaired hepatic or renal function; and a history of other respiratory disorders or upper airway surgery. After applying these exclusion criteria, 6,659 participants were deemed eligible for the study (Figure 1). We compared the baseline characteristics between participants who were excluded and those who were included in the cross-sectional analysis. No significant differences were found in age, sex, blood pressure, or several laboratory indices, indicating that the excluded subjects were representative of the broader target population and that selection bias was unlikely (Table S1). The study protocol was reviewed and approved by the Clinical Ethics Committee of the People's Hospital of Xinjiang Uyghur Autonomous Region (KY2024052407). All methods were carried out in accordance with current guidelines, and all procedures complied with the requirements of the Declaration of Helsinki. All participants provided written informed consent.

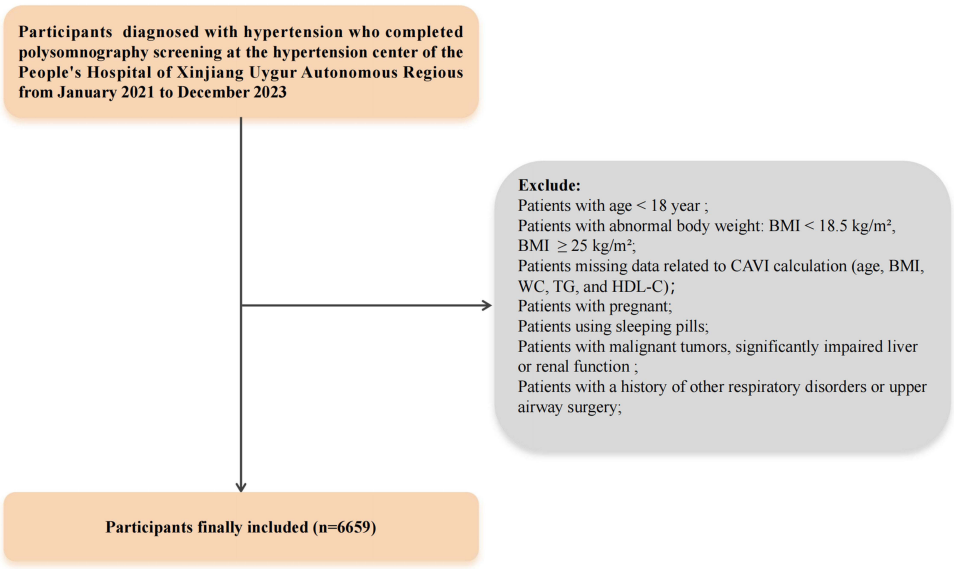

Figure 1 Flowchart of the study participant selection process.

Covariates

Patient demographic information was collected from electronic medical records, including detailed information such as age, sex, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), and current smoking status. In addition, this study collected patients' medical histories, medication histories, and relevant biochemical test results. All medications used in this study are provided in Table S2, and the definitions and diagnostic criteria for diseases are described in the Supplementary Materials. Biochemical parameters include fasting plasma glucose (FPG), glycated hemoglobin (HbA1c), triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), aspartate aminotransferase (AST), alanine aminotransferase (ALT), uric acid (UA), creatinine (Cr), and blood urea nitrogen (BUN). Detailed measurement methods for each index are described in the supplementary materials.

Chinese Visceral Adiposity Index

The CVAI was calculated using a sex-specific equation, determined by the following formula: Female = - 187.32 + 1.71 * age + 4.23 * BMI (kg/m2) + 1.12 * WC (cm) + 39.76 * lgTG (mmol/L) - 11.66 * HDL-C (mmol/L); Male = - 267.93 + 0.68 * age + 0.03 * BMI (kg/m^2) + 4.00 * WC (cm) + 22.00 * lgTG (mmol/L) - 16.32 * HDL-C (mmol/L).[8,11]

Diagnosis and Definition of the OSA

All patients received overnight monitoring using PSG (Compumedics, Australia). Specific measurement methods are detailed in the supplementary materials. OSA was defined as apnoea hypopnea index (AHI) ≥ 5. Additionally, the severity of OSA was classified as mild OSA (5 ≤ AHI < 15), moderate OSA (15 ≤ AHI < 30), and severe OSA (AHI ≥ 30).[17]

Statistical Analysis

We used the R package "missForest" to perform multiple imputation for missing values in the study data. Participants were categorized into four groups based on CVAI quartiles: Q1 (<79.57), Q2 (79.57–99.00), Q3 (99.00–126.66), and Q4 (>126.66). Continuous variables with a normal distribution are presented as mean ± SD; skewed variables as median [IQR]; and categorical variables as count (percentage). To compare characteristics among the four groups, the chi-square test was used for categorical variables, one-way ANOVA for continuous variables with a normal distribution, and the Kruskal–Wallis test for continuous variables with a skewed distribution. Before building the logistic regression model, multicollinearity was assessed by calculating the variance inflation factor (VIF), and variables with VIF > 5 were excluded from the analysis (Table S3). Associations between OSA and CVAI were assessed using multiple logistic regression, while associations between AHI and CVAI were examined using multiple linear regression. In these models, logistic regression results are reported as odds ratios (ORs) with 95% confidence intervals (CIs), and linear regression results as beta coefficients (β) with 95% CIs. Restricted cubic spline (RCS) analysis was performed to assess the potential linearity of the dose-response relationship between CVAI and OSA. Subgroup and sensitivity analyses were conducted to further test the robustness of the findings. Statistical analyses were conducted using R software (version 4.2.2). Further methodological details are provided in the Supplementary Materials.

Result

Participant Baseline Characteristics

A total of 6,659 patients were included in this study. Their baseline characteristics are summarized in Table 1. The prevalence of OSA across CVAI quartiles is shown in Figure 2. Compared to those in the low CVAI group, participants in the high CVAI group were older on average, had higher BMI, and had higher rates of current smoking and alcohol consumption. They also exhibited higher SBP, as well as elevated levels of ALT, FPG, HbA1c, Cr, BUN, UA, TC, TG, and LDL-C, and lower levels of HDL-C. Additionally, participants in the higher CVAI group had a higher prevalence of DM, coronary heart disease (CHD), and dyslipidemia. The use of antidiabetic drugs, Lipid-lowering drugs, and antihypertensive medications was also significantly higher in this group.

Table 1 Baseline Characteristics of Participants Based on CVAI Quartiles

Variables	Q1 (<79.57)	Q2(79.57–99.00)	Q3 (99.00–126.66)	Q4 (>126.66)	P
N	1692	1691	1691	1691	
Age (year)	44.65 ± 10.38	52.39 ± 8.83	56.59 ± 12.15	58.59 ± 16.39	<0.001
Male, n (%)	249 (14.95)	474 (28.49)	676 (40.60)	1071 (64.32)	<0.001
WC (cm)	87.74 (82.00,94.00)	86.77 (84.00,93.87)	92.00 (88.00,98.00)	102.00 (96.00,109.00)	<0.001
BMI (kg/m^2)	22.22 (20.90,23.14)	22.66 (21.77,23.31)	22.68 (21.76,23.44)	22.86 (21.97,23.44)	<0.001
SBP (mmHg)	141.00 (130.00,154.00)	141.00 (130.00,154.00)	143.00 (130.00,156.00)	144.00 (132.00,156.00)	0.006
DBP (mmHg)	88.00 (80.00,97.00)	85.00 (77.00,93.25)	83.00 (74.00,93.00)	82.00 (73.00,91.00)	<0.001
Current smoking, n (%)	123 (7.39)	279 (16.77)	382 (22.94)	581 (34.89)	<0.001
Current drinking, n (%)	151 (9.07)	270 (16.23)	338 (20.30)	495 (29.73)	<0.001
ALT (U/L)	15.00 (11.00,21.00)	17.00 (13.00,23.83)	18.00 (13.00,25.00)	18.00 (13.00,25.80)	<0.001
AST (U/L)	17.00 (14.80,20.40)	18.00 (15.00,21.40)	18.30 (15.34,22.40)	19.00 (16.00,22.00)	<0.001
FPG (mmol/L)	4.64 ± 1.12	4.87 ± 1.35	4.99 ± 1.42	5.14 ± 1.65	<0.001
HbA1c%	5.57 (5.38,5.80)	5.69 (5.49,5.94)	5.70 (5.50,6.09)	5.73 (5.50,6.10)	<0.001
Cr (U/L)	54.50 (48.20,62.64)	56.70 (50.10,66.23)	60.40 (52.30,70.41)	66.80 (57.21,76.60)	<0.001
BUN (U/L)	4.65 ± 2.00	4.90 ± 1.42	5.15 ± 1.69	5.54 ± 2.15	<0.001
UA (U/L)	274.69 ± 75.40	296.78 ± 75.36	315.12 ± 82.90	329.83 ± 85.29	<0.001
TC (mmol/L)	4.36 (3.84,4.97)	4.54 (3.94,5.24)	4.52 (3.86,5.19)	4.42 (3.77,5.08)	<0.001
TG (mmol/L)	0.98 (0.75,1.28)	1.28 (0.97,1.72)	1.36 (0.99,1.94)	1.36 (1.00,1.92)	<0.001
HDL-c (mmol/L)	1.30 ± 0.31	1.21 ± 0.29	1.16 ± 0.29	1.10 ± 0.27	<0.001
LDL-c (mmol/L)	2.64 ± 0.75	2.82 ± 0.87	2.75 ± 0.85	2.67 ± 0.84	<0.001
AHI, events/hour	1.20 (1.00,5.40)	2.30 (1.90,6.90)	3.50 (3.00,9.40)	4.90 (4.20,27.20)	<0.001
Mean SaO^2, %	94.00 (93.00,95.00)	93.00 (92.00,95.00)	93.00 (92.00,95.00)	93.00 (92.00,94.00)	<0.001
Lowest SaO^2, %	87.00 (85.00,89.00)	86.00 (83.00,88.00)	86.00 (81.00,88.00)	85.00 (80.00,87.00)	<0.001
CVAI	66.87 (56.63,74.07)	89.46 (84.77,94.16)	111.16 (104.96,118.53)	150.61 (136.17,171.63)	
Disease history, n (%)					
DM	83 (4.98)	160 (9.62)	219 (13.15)	310 (18.62)	<0.001
Dyslipidemia	153 (9.19)	264 (15.87)	286 (17.18)	257 (15.44)	<0.001
CHD	16 (0.96)	23 (1.38)	35 (2.10)	65 (3.90)	<0.001
OSA, n (%)					
Mild	319 (19.16)	325 (19.53)	329 (19.76)	375 (22.52)	<0.001
Moderate	134 (8.05)	181 (10.88)	201 (12.07)	179 (10.75)	<0.001
Severe	64 (3.84)	75 (4.51)	177 (10.63)	270 (16.22)	<0.001
Medication use, n (%)					
Lipid-lowering drugs	80 (4.80)	167 (10.04)	227 (13.63)	234 (14.05)	<0.001
Antihypertensive drugs	1002 (60.18)	1120 (67.31)	1194 (71.71)	1168 (70.15)	<0.001
Antidiabetic drugs	45 (2.70)	103 (6.19)	131 (7.87)	190 (11.41)	<0.001

Notes: Data are presented as mean ± standard deviation (SD), median [interquartile range (IQR)], or number (percentage), as appropriate.
Abbreviations: CVAI, Chinese visceral adiposity index; WC, Waist circumference; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; ALT, alanine transaminase; AST, aspartate transaminase; FPG, fasting plasma glucose; Cr, creatininel; eGFR, estimated glomerular filtration rate; BUN, blood urea nitrogen; UA, uric acid; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; HbA1c, glycosylated hemoglobin; AHI, apnea-hypopnea index; OSA, obstructive sleep apnoea, CHD, coronary heart disease; DM, diabetes mellitus.

Relationship Between CVAI and OSA

The associations between CVAI and OSA risk in logistic regression models are presented in Table 2. In the unadjusted model, each standard deviation (SD) increase in CVAI was positively associated with OSA risk (OR = 1.38, 95% CI: 1.31–1.45). This association remained significant in the fully adjusted model (OR = 1.39, 95% CI: 1.26–1.54). Upon categorizing CVAI into quartiles, the odds ratios (ORs) for OSA risk relative to Q1 were 1.32 (95% CI: 1.09–1.60) for Q2, 1.76 (95% CI: 1.47–2.09) for Q3, and 2.31 (95% CI: 1.95–2.72) for Q4. After adjustment for all confounders, the risk of OSA increased progressively across CVAI quartiles compared to Q1, with ORs of 1.21 (95% CI: 0.98–1.49) for Q2, 1.49 (95% CI: 1.20–1.84) for Q3, and 1.67 (95% CI: 1.29–2.17) for Q4, with a significant trend (p for trend < 0.001).

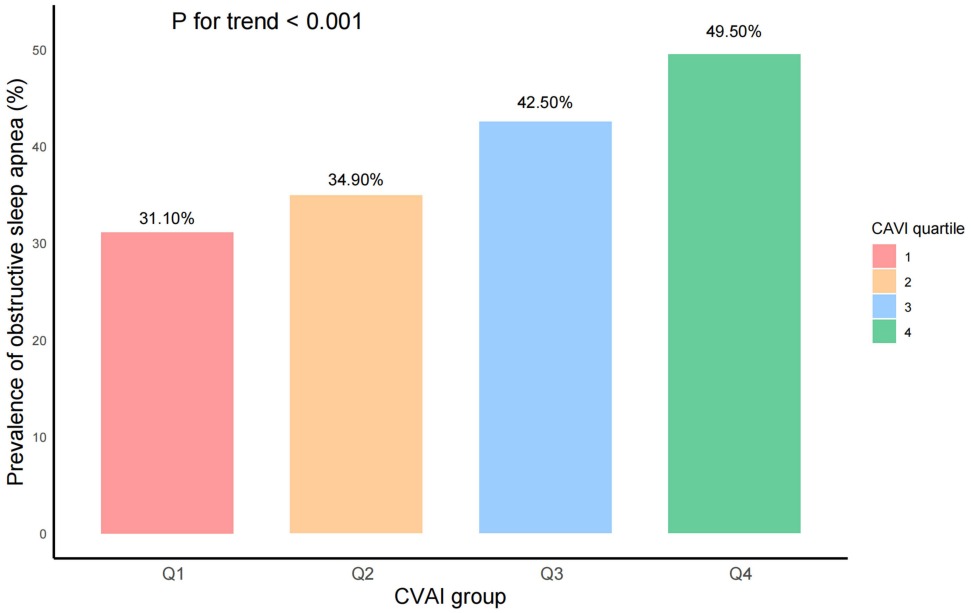

Figure 2 Prevalence of obstructive sleep apnoea (OSA) stratified by Chinese visceral adiposity index (CVAI) quartiles.

The RCS model demonstrated a linear dose-response relationship between CVAI and OSA risk (p for overall association < 0.001; Figure 3A).

Relationship Between CVAI and AHI

Furthermore, we conducted additional analyses to clarify the relationship between CVAI and AHI. As shown in Table 3, CVAI levels were significantly and positively associated with AHI (β = 4.72, 95% CI: 4.37–5.08). This association remained significant after full adjustment (β = 4.84, 95% CI: 4.39–5.29). When CVAI was categorized into quartiles for further analysis, the fully adjusted model showed that, compared to Q1, the β coefficients (95% CI) were 2.49 (1.12-–3.86) for Q2, 6.72 (5.37–8.07) for Q3, and 11.75 (10.30–13.19) for Q4. Trend analysis was statistically significant (p for trend < 0.001 for all). Moreover, the RCS model showed a linear dose–response association between CVAI and AHI (p for overall association < 0.001; Figure 3B).

Table 2 Relationship Between CVAI with OSA

Exposure	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)	Model 4 OR (95% CI)
OSA				
CVAI (per SD increase)	1.38 (1.31 ~ 1.45)	1.42 (1.33 ~ 1.52)	1.40 (1.27 ~ 1.54)	1.39 (1.26 ~ 1.54)
Quartile of CVAI				
Q1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2	1.32 (1.09 ~ 1.60)	1.32 (1.08 ~ 1.61)	1.22 (0.99 ~ 1.49)	1.21 (0.98 ~ 1.49)
Q3	1.76 (1.47 ~ 2.09)	1.75 (1.44 ~ 2.13)	1.50 (1.21 ~ 1.85)	1.49 (1.20 ~ 1.84)
Q4	2.31 (1.95 ~ 2.72)	2.29 (1.86 ~ 2.83)	1.69 (1.30 ~ 2.20)	1.67 (1.29 ~ 2.17)
P for trend	<0.001	<0.001	<0.001	<0.001

Notes: Model 1: unadjust; Model 2: sex, age, current smoking, current drinking, BMI, SBP, DBP; Model 3: sex, age, current smoking, current drinking, BMI, SBP, DBP, HbA1c, BUN, Crea, HDL-C, UA, AST, ALT, FPG, TG; Model 4: sex, age, current smoking, current drinking, BMI, SBP, DBP, HbA1c, BUN, Crea, HDL-C, UA, AST, ALT, FPG, TG, CHD, DM, dyslipidemia, lipid-lowering drugs, antihypertensive drugs, antidiabetic drugs. P for trend: refers to the statistical significance of the trend across ordered groups.

Abbreviations: OR, Odds Ratio; CI, Confidence Interval. Other abbreviations, see Table 1.

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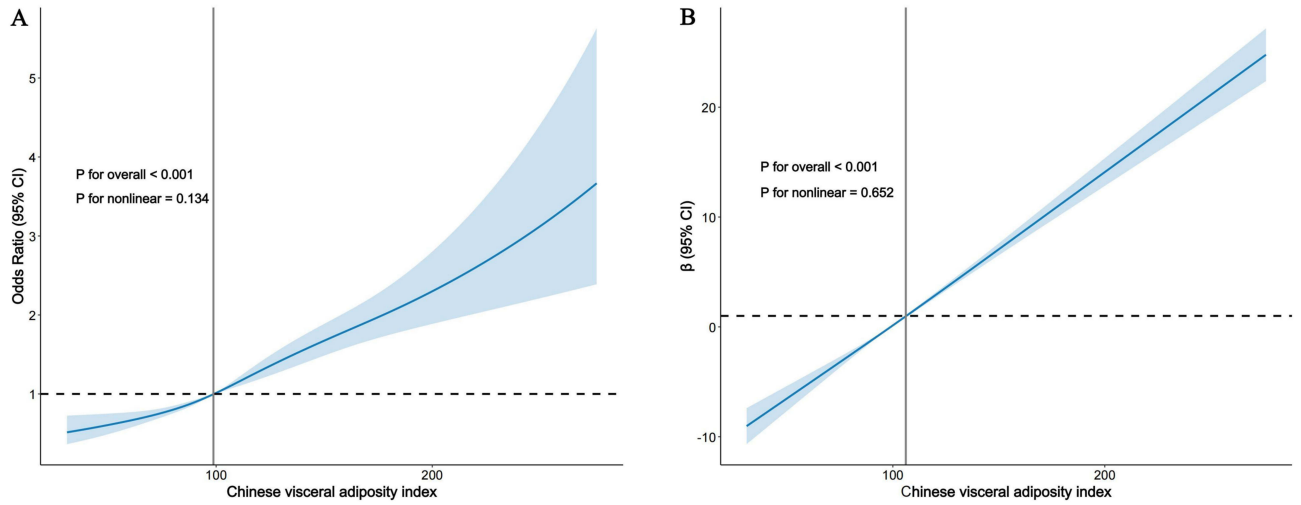

Figure 3 Dose-response relationship between CVAI and OSA, AHI. (**A**) Association with OSA. (**B**) Association with AHI.

Subgroup and Sensitivity Analyses

We evaluated the association between CVAI and OSA within subgroups defined by age, gender, current smoking status, and current drinking status, further investigating the potential influence of these factors. None of the baseline variables, including gender (P for interaction = 0.629 and 0.487 for OSA and AHI, respectively), age (P for interaction = 0.906 and 0.609), current smoking (P for interaction = 0.304 and 0.712), or current drinking (P for interaction = 0.969 and 0.067), significantly modified the relationship between CVAI and the risk of OSA or AHI (Figure 4). Consistent associations were observed across all subgroups, with no significant interaction effects detected. To ensure the robustness of our findings, we performed sensitivity analyses by systematically excluding specific populations. First, we excluded participants with missing data; the results remained stable (Table S4). We then excluded participants with dyslipidemia (Table S5), DM (Table S6), and CHD (Table S7), and found that the results remained consistent in all sensitivity analyses.

Table 3 Relationship Between CVAI with AHI

Exposure	Model 1 β (95% CI)	Model 2 β (95% CI)	Model 3 β (95% CI)	Model 4 β(95% CI)
AHI				
CVAI (per SD increase)	4.72 (4.37 ~ 5.08)	4.90 (4.46 ~ 5.34)	4.84 (4.39 ~ 5.29)	4.84 (4.39 ~ 5.29)
Quartile of CVAI				
Q1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2	2.52 (1.21 ~ 3.83)	2.51 (1.15 ~ 3.87)	2.51 (1.14 ~ 3.88)	2.49 (1.12 ~ 3.86)
Q3	6.85 (5.66 ~ 8.04)	6.85 (5.52 ~ 8.17)	6.74 (5.39 ~ 8.09)	6.72 (5.37 ~ 8.07)
Q4	12.32 (11.19 ~ 13.45)	12.00 (10.60 ~ 13.41)	11.78 (10.34 ~ 13.22)	11.75 (10.30 ~ 13.19)
P for trend	<0.001	<0.001	<0.001	<0.001

Notes: Model 1: unadjust; Model 2: sex, age, current smoking, current drinking, BMI, SBP, DBP; Model 3: sex, age, current smoking, current drinking, BMI, SBP, DBP, HbA1c, BUN, Crea, HDL-C, UA, AST, ALT, FPG, TG; Model 4: sex, age, current smoking, current drinking, BMI, SBP, DBP, HbA1c, BUN, Crea, HDL-C, UA, AST, ALT, FPG, TG, CHD, DM, dyslipidemia, lipid-lowering drugs, antihypertensive drugs, antidiabetic drugs. P for trend: refers to the statistical significance of the trend across ordered groups.
Abbreviations: OR, Odds Ratio; CI, Confidence Interval. Other abbreviations, see Table 1.

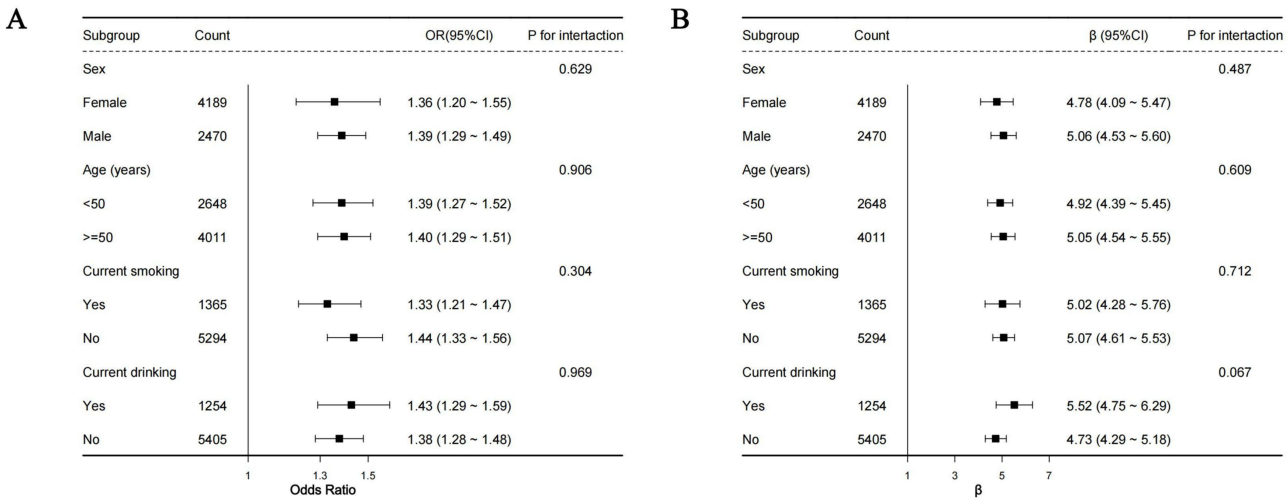

Figure 4 Subgroup analyses for per SD increment in Chinese visceral adiposity index (CVAI) on obstructive sleep apnoea (OSA). (**A**) Impact on OSA. (**B**) Impact on AHI.

Discussion

In this cross-sectional analysis, we investigated the association between CVAI and OSA among normal-weight individuals with hypertension and identified several notable findings. First, a significant positive association was observed between CVAI and OSA risk, which persisted even after adjustment for potential confounders. Second, when OSA was defined by the AHI, higher CVAI levels were significantly associated with higher AHI. Restricted cubic spline analysis further revealed a linear dose-response relationship of CVAI with both OSA risk and AHI scores.

OSA is a prevalent sleep disorder. Mounting evidence indicates a close link between visceral adiposity and OSA, especially among obese and diabetic individuals.[4,12,18–22] CVAI is a composite index for assessing visceral fat content.[8] Recent studies have shown that CVAI is positively associated with OSA risk in individuals with DM.[12] However, limited research has explored its relevance to normal-weight patients with hypertension. This study therefore investigates whether CVAI can help identify OSA risk in this specific group, which may enhance early detection and support targeted screening strategies.

Both our study and that of Zheng et al demonstrated an association between increased visceral adiposity and a higher risk of OSA.[12] Notably, Zheng et al focused on patients with DM, who typically have pronounced metabolic disturbances and higher BMI, whereas our study investigated normal-weight individuals with hypertension. In diabetic patients, metabolic dysfunction may amplify the effect of visceral adiposity on OSA risk. However, our findings indicate that even in the absence of obesity or diabetes, elevated visceral adiposity remains an independent risk factor for OSA among hypertensive individuals. These results underscore the importance of considering population heterogeneity when interpreting the relationship between visceral adiposity and OSA.

Although only patients with normal-weight were included, a considerable proportion nonetheless had elevated CVAI, suggesting that increased visceral adiposity is prevalent even among individuals who are not overweight by conventional measures. Several potential mechanisms may underlie the relationship between CVAI and OSA in normal-weight patients with hypertension. First, visceral adiposity may contribute to the development of OSA through mechanical pathways. Studies have shown that 14% of individuals with normal-weight exhibit abdominal obesity.[2] Consistent with these findings, our results demonstrated that normal-weight hypertensive patients with elevated CVAI had significantly greater waist circumference, and an increased prevalence and severity of OSA. These results suggest that increased visceral adiposity, is closely associated with OSA risk in this population. Specifically, abdominal obesity can increase intra-abdominal pressure, reduce diaphragm mobility, and increase the risk of upper airway collapse.[20] It may also decrease thoracic compliance, thereby impairing normal respiratory function and further increasing the risk of OSA. Moreover, increased abdominal visceral adiposity may reduce lung volumes, and diminish pharyngeal traction, thus promoting pharyngeal collapse and further contributing to OSA development.[23,24] In addition to these mechanical effects, visceral adiposity serves as a source of various inflammatory mediators and adipokines, resulting in systemic inflammation, oxidative stress, and insulin resistance. These factors can

collectively reduce upper airway muscle tone, thereby increasing the risk of upper airway collapse.[1,18,24] Consistent with this mechanism, our study found that patients with higher CVAI also exhibited elevated fasting glucose and HbA1c levels, reflecting poorer metabolic control and a potential state of chronic low-grade inflammation. Although we did not directly measure systemic inflammatory markers, these metabolic changes may indicate underlying inflammatory processes and insulin resistance. This further supports the role of visceral adiposity as an active endocrine organ that promotes OSA through inflammation-mediated weakening of the upper airway musculature. Furthermore, OSA itself can induce insulin resistance and stimulate an inflammatory response, which further contributes to lipid metabolism disturbances. This cycle forms a complex negative feedback loop that accelerates the development and progression of OSA.[25]

To our knowledge, this is the first study to explore the relationship between CVAI and OSA in normal-weight hypertensive patients. However, this study still has several limitations. First of all, this study was designed as a cross-sectional study, therefore, it is unable to investigate the causal relationship between CVAI and OSA. Furthermore, despite adjustment for a wide range of potential confounders in the statistical analysis, there is still the possibility of residual confounding from unmeasured variables. Since the data were collected from a single hypertension center, there may be limitations in generalizing our findings to other regions.

Conclusion

In conclusion, this study suggests that CVAI may be independently associated with OSA risk in normal-weight hypertensive patients. Clinicians might consider incorporating CVAI into routine assessments to help identify individuals who could benefit from earlier OSA screening and intervention, even in the absence of obesity. However, as these findings are limited to a specific population, further studies are warranted to validate the predictive value and clinical utility of CVAI among different ethnic groups, as well as to examine its applicability in broader clinical settings.

Data Sharing Statement

The dataset used and analysed during the current study is available from the corresponding author on reasonable request.

Informed Consent

All the participants gave their informed consent agreement.

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Disclosure

The authors have no conflicts of interest to declare for this work.

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