BMC Endocrine Disorders

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Association between visceral adiposity index, lipid accumulation product and type 2 diabetes mellitus in US adults with hypertension: a cross-sectional analysis of NHANES from 2005 to 2018

Chen Lv[1] and Rui Huo[1*]

Abstract

Background The presence of hypertension significantly increases the risk of diabetes, particularly type 2 diabetes. Recently, Visceral Adiposity Index (VAI) has been introduced as a straightforward and robust alternative indicator for early detection of metabolic syndrome, cardiovascular disease, and T2DM. Visceral adiposity, more dangerous than subcutaneous fat, is associated with metabolic syndrome and cardiovascular diseases. The VAI and Lipid Accumulation Product (LAP) are indices that quantify visceral fat and lipid overaccumulation, respectively. This study aims to explore the association between VAI, LAP, and type 2 diabetes mellitus (T2DM) in US adults with hypertension using NHANES data from 2005 to 2018.

Methods We analyzed data from 5,620 participants with hypertension in The National Health and Nutrition Examination Survey (NHANES). VAI and LAP were calculated using established formulas. The VAI is calculated based on a combination of waist circumference, body mass index (BMI), triglycerides, and high-density lipoprotein (HDL) cholesterol levels. Logistic regression models were applied to evaluate the association between these indices and T2DM, adjusting for potential confounders. Subgroup analyses by age and gender were also conducted to assess variations in risk.

Results In all, 5,620 participants were enrolled in our analysis, with 2,754 (49%) being female, and a mean (standard deviation, SD) age of 57 (15) years. The mean (SD) cumulative average VAI and LAP among all participants was 241 (2.71) and 75 (67), respectively. Totally, higher VAI and LAP indices were significantly associated with an increased risk of T2DM in individuals with hypertension. For VAI, the odds ratios (OR) for T2DM were higher in older adults (≥ 60 years) [95% confidence interval (CI): 1.37, 1.22–1.53, per 1 SD increase] and females [95% confidence interval (CI): 1.39, 1.27–1.52, per 1 SD increase], indicating age and gender differences in risk. Non-linear relationships were observed, suggesting thresholds beyond which the risk of T2DM escalates dramatically.

*Correspondence:
Rui Huo
RuiDoctor123@163.com

Full list of author information is available at the end of the article

Conclusions Both VAI and LAP are reliable markers for assessing T2DM risk in individuals with hypertension. Incorporating these indices into clinical practice could enhance the identification of high-risk individuals and facilitate early intervention strategies. Future longitudinal studies are needed to confirm these associations and explore targeted interventions.

Keywords Type 2 diabetes, Hypertension, Visceral adiposity index, Lipid accumulation product, NHANES

Introduction

Hypertension is a significant risk factor for developing diabetes, particularly type 2 diabetes [1]. The relationship between these two conditions is complex and multifaceted, with each exacerbating the negative health impacts of the other. When an individual with hypertension develops diabetes, the health implications can be severe and multifarious. According to the American Heart Association, individuals with both hypertension and diabetes are four times more likely to develop cardiovascular diseases compared to those with neither condition [2–4]. Additionally, the prevalence of kidney damage increases by 60% in patients suffering from both conditions [5]. Nearly 75% of adults with hypertension also have diabetes, underscoring the frequent coexistence of these diseases [6]. For instance, individuals with hypertension or diabetes have a two to four times higher risk of heart disease compared to those without these conditions [7]. The Framingham Heart Study also concluded that the presence of both hypertension and diabetes increased the risk of congestive heart failure by up to 50% [8]. Therefore, understanding the factors that contribute to the development and progression of diabetes in individuals with hypertension is of paramount importance for public health.

Visceral adiposity, characterized by the accumulation of fat around internal organs, is considered more dangerous than subcutaneous fat due to its association with metabolic syndrome and cardiovascular diseases [9, 10]. This type of fat is metabolically active and has been linked to insulin resistance, inflammation, and dyslipidemia, which can be quantified by the visceral adiposity index (VAI) [11–13]. The VAI is calculated based on a combination of waist circumference, body mass index (BMI), triglycerides, and high-density lipoprotein (HDL) cholesterol levels [14]. Several studies have demonstrated that a higher VAI is associated with an increased risk of metabolic syndrome, cardiovascular disease, and T2DM [15]. Lipid Accumulation Product (LAP) is another index used to assess lipid overaccumulation and is calculated from waist circumference and fasting triglyceride levels [16, 17]. It serves as a surrogate marker for visceral adiposity and has been associated with several metabolic risk factors [18]. Both VAI and LAP are valuable tools in understanding how obesity contributes to health risks, particularly in individuals with hypertension. However, the impact of VAI and LAP on T2DM within the population with hypertension has yet to be fully understood.

Here, we explore the association between the VAI, LAP and diabetes in US adults with hypertension based on the National Health and Nutrition Examination Survey (NHANES) data from 2005 to 2018. Incorporating VAI and LAP into clinical practice could enhance the identification of high-risk individuals and facilitate early intervention strategies aimed at reducing visceral fat and improving lipid profiles.

Methods

Study population

The National Health and Nutrition Examination Survey (NHANES) is a comprehensive, cross-sectional study assessing the health and nutritional status of adults and children across the United States. This extensive survey employs an integrated methodological approach, combining interviews, physical examinations, dietary analyses, and laboratory tests. The survey utilizes a stratified multistage probability sampling technique to ensure rigorous data collection. Conducted from 2005 to 2018, NHANES meticulously selected 5,620 participants (Fig. 1). Adhering to ethical guidelines, informed consent was secured from all participants, with signed written consent forms. Furthermore, the study's protocol rigorously complied with established ethical standards, safeguarding the integrity and ethical soundness of the research.

Outcomes and definitions

Adult Treatment Panel III (ATP 3) of the National Cholesterol Education Program (NCEP) classified Hypertension was defined as systolic blood pressure (SBP) ≥ 140 mmHg, diastolic blood pressure (DBP) ≥ 90 mmHg, or taking blood pressure control medication [19]. Hyperlipemia was defined as total cholesterol ≥ 260 mg/dl, triglycerides ≥ 160 mg/dl in blood of adults under 12–14 h fasting [20]. We used multiple methods to define type 2 diabetes mellitus (T2DM), including self-reported diabetes history, use of glucose-lowering medications (insulin or oral), and diabetic complications like retinopathy. Additionally, T2DM diagnosis is based on HbA1C levels ≥ 6.5%, fasting plasma glucose (FPG) levels ≥ 126 mg/dL after at least 8 h of fasting, and a 2-hour plasma glucose level ≥ 200 mg/dL during an oral glucose tolerance test (OGTT) following the consumption of a 75 g glucose

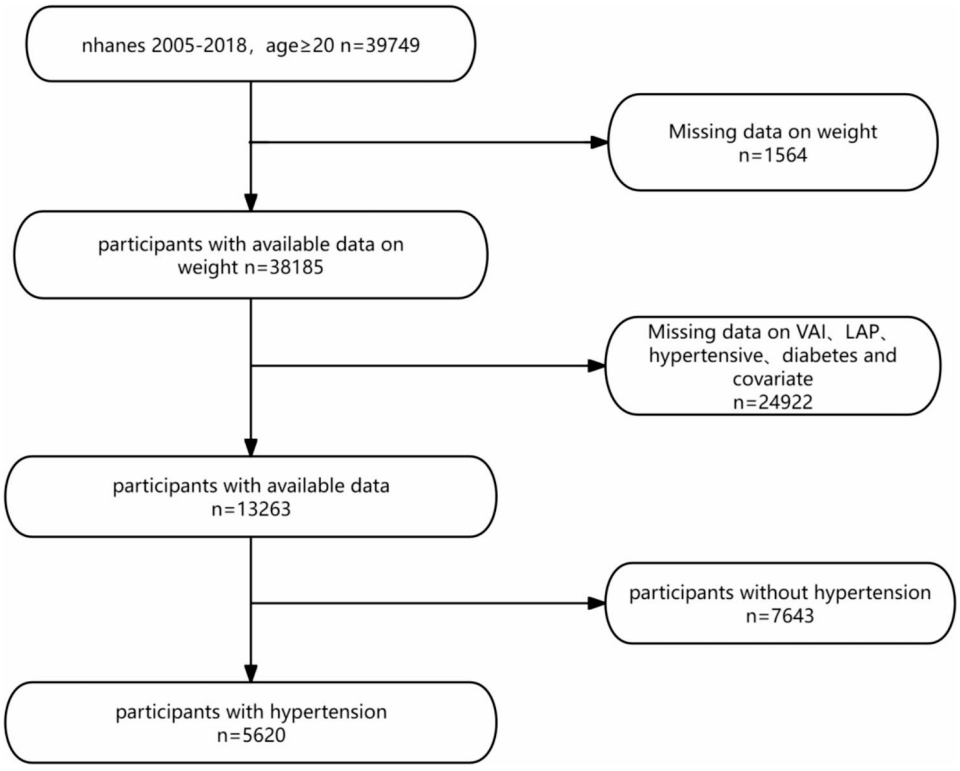

Fig. 1 The flowchart of Study population

solution. These comprehensive criteria ensure accurate T2DM identification [21]. Cardiovascular disease (CVD) was established by self-reported physician diagnoses obtained during an individual interview using an International Statistical Classification of Diseases and Related Health Problems questionnaire, Tenth Revision (ICD-10) codes. According to the questionnaire, cardiovascular diseases are classified under codes I00-I99. This broad category encompasses several specific conditions, including: Congestive heart failure (I50.0, I50.1, I50.9); Coronary artery disease (I20-I25.9); Myocardial infarction (I21-I23); Angina pectoris (I20.0-I20.9). Furthermore, cardiovascular disease mortality is typically reported using a specific set of ICD-10 codes: I00–I09, I11, I13, I20–I51, or I60–I69.

VAI index was calculated using the formula: WC / (39.68 + 1.88 * BMI) * (TG / 1.03) * (1.31 / HDL_C) [22]. LAP index was calculated using the formula: (WC −65) * TG for males; (WC −58) * TG for females. WC = Waist Circumference (cm) [23]; BMI = Body Mass Index (kg/m^2); TG = Triglycerides (mmol/L); HDL-C = High-Density Lipoprotein Cholesterol (mmol/L). eGFR = 141 × min (sCr/ k, 1) $^{\alpha}$ × max (sCr/ k, 1) $^{-1.209}$ × 0.993^{Age} (× 1.018, if female). k is 0.9 (for males) and 0.7 (for females). a is −0.411 (for males) and −0.329 (for females). min and max indicate the minimum and the maximum of sCr/ k or 1, respectively [24].

Covariates

Age, sex, race-ethnicity, education, poverty-to-income ratio (PIR), smoking status and drinking status were self-reported by participants. For the classification of race/ethnicity, the following categories were employed: Non-Hispanic White, Non-Hispanic Black, Mexican-American, and other races/ethnicities. We divide smoking into 3 levels (never: smoked less than 100 cigarettes in life, former: smoked more than 100 cigarettes in life and smoked not at all now, now: smoked moth than 100 cigarettes in life and smoke some days or every day). Current heavy alcohol use (≥3 drinks per day for females, ≥4 drinks per day for males, or binge drinking [≥4 drinks on same occasion for females, ≥5 drinks on same occasion for males] on 5 or more days per month), current moderate alcohol use (≥2 drinks per day for females, ≥3 drinks per day for males, or binge drinking≥2 days per month), or a history of daily binge drinking.

Statistical analysis

Data for continuous variables are expressed as mean ± SD (standard deviation), while categorical variables are presented as percentages. Differences between participants grouped by VAI and LAP tertiles, were assessed using a weighted t-test (for continuous variables) and a weighted chi-square test (for categorical variables). A logistic regression model was then applied to evaluate the association between the VAI or LAP and T2DM. Multiple

models were used to measure odds ratios (OR) and 95% confidence intervals (CI), adjusting for potential confounders. The VAI and LAP were categorized into tertiles, with the first tertile serving as a reference in trend analysis. Exploring the nonlinear relationship between VAI or LAP and T2DM using restricted cubic spline (RCS). All statistical analyses were conducted using the R software package version 4.3.2.

Results

Baseline characteristics

The baseline characteristics of individuals stratified by the presence of Type 2 Diabetes Mellitus (T2DM) are detailed in Table 1. Among the 5,620 participants with hypertension, 47% (N=2,394) were without diabetes (non-T2DM) and 53% (N=3,226) had diabetes (T2DM). The T2DM group exhibited a significantly higher mean age of 60 years compared to the non-T2DM group's 53 years ($p<0.001$). Gender distribution was also significantly different, with a slightly higher proportion of males in the T2DM group (53%) than in the non-T2DM group (49%) ($p=0.028$).

Educational attainment varied significantly between the groups, with a greater proportion of individuals in the T2DM group having less than a high school education (19% vs. 15% in the non-T2DM group) ($p<0.001$).

Table 1 Baseline characteristics of individuals with hypertension grouped by T2DM

Characteristic	Overall, $N=5,620$	non-T2DM, $N=2,394$(47%)	T2DM, $N=3,226$ (53%)	*p*-value
Age(years)	57 (15)	53(15)	60(14)	< 0.001
Gender				0.028
male	2,866 (51%)	1,172(49%)	1,694(53%)	
female	2,754 (49%)	1,222(51%)	1,532(47%)	
Education				< 0.001
te < High school	1,489(17%)	525(15%)	964(19%)	
High School Grad/GED	1,412(26%)	590(25%)	622(27%)	
> High school	2,719(57%)	1,279 (60%)	1,440(54%)	
Race				0.072
Mexican	664 (5.4%)	225 (4.6%)	439(6.0%)	
White	2,638 (72%)	1,146(72%)	1,492(72%)	
Black	1,377 (12%)	640(13%)	737(11%)	
Other	941 (10.6%)	385(10.4%)	558(11.0%)	
PIR	3.01 (1.62)	3.05(1.62)	2.98(1.62)	0.2
smoke				< 0.001
former	1,755 (32%)	639 (27%)	1,116 (36%)	
never	2,789(49%)	1,202 (50%)	1,587 (48%)	
now	1,076(19%)	553(23%)	523 (15%)	
alcohol user				< 0.001
former	1,200 (18%)	412(14%)	788(21%)	
heavy	879(17%)	422 (19%)	457(15%)	
mild	2,011 (39%)	899(41%)	1,112(38%)	
moderate	709(15%)	361(17%)	348 (13%)	
never	821(11%)	300 (9.3%)	521 (13%)	
VAI	241(2.71)	1.94(1.89)	2.83(3.21)	< 0.001
LAP	75(67)	59(53)	89(74)	< 0.001
Triglyceride(mg/dL)	145 (112)	127 (88)	161 (127)	< 0.001
FPG(mg/dL)	115 (38)	98 (7)	131 (46)	< 0.001
HbA1c%	5.90 (1.10)	5.41 (0.35)	6.33 (1.33)	< 0.001
SBP	133 (19)	131 (18)	135 (20)	< 0.001
DBP	73 (15)	74 (14)	71 (16)	< 0.001
Albuminuria	1,143 (16%)	265 (7.4%)	878 (23%)	< 0.001
Hypolipidemic treatment	2,040 (35%)	560 (23%)	1,480 (46%)	< 0.001
Hypertensive treatment	3,774 (66%)	1,325 (54%)	2,449 (76%)	< 0.001
eGFR	86 (22)	89(21)	83(22)	< 0.001
Hyperlipidemia	4,633 (83%)	1,834(77%)	2,799 (88%)	< 0.001
CVD	1,126 (17%)	348 (12%)	778(22%)	< 0.001

PIR: Poverty-to-Income Ratio; VAI: Visceral Adiposity Index; LAP: Lipid Accumulation Product; eGFR: estimated Glomerular Filtration Rate; CVD: Cardiovascular disease; FPG: fasting plasma glucose

Table 2 The association between VAI and LAP index with T2DM in individuals with hypertension

	continuation	T1	T2 (OR 95%CI)	T3 (OR 95%CI)	*p* for trend
Model 1					
VAI	1.24(1.17,1.31)	Ref	1.67(1.36,2.04)	3.23(2.60,4.00)	<0.0001
LAP	1.01(1.00,1.02)	Ref	1.67(1.38,2.03)	4.10(3.30,5.10)	<0.0001
Model 2					
VAI	1.14(1.06,1.24)	Ref	1.82(1.27,2.62)	2.90(1.84,4.57)	<0.0001
LAP	1.01(1.00,1.01)	Ref	1.40(0.89,2.20)	2.82(1.80,4.41)	<0.0001
Model 3					
VAI	1.26(1.18,1.34)	Ref	1.69(1.34,2.13)	3.43(2.69,4.38)	<0.0001
LAP	1.01(1.00,1.01)	Ref	1.67(1.33,2.10)	4.17(3.29,5.29)	<0.0001

Model 1 adjusted for the following variables: age, education level, gender, race, family income, smoking status, alcohol use, eGRF, hyperlipidemia, and cardiovascular disease. Model 2 excluded individuals taking antihypertension, antidiabetes, or lipid-lowering medications. Model 3 excluded individuals with a history of cardiovascular disease (CVD)

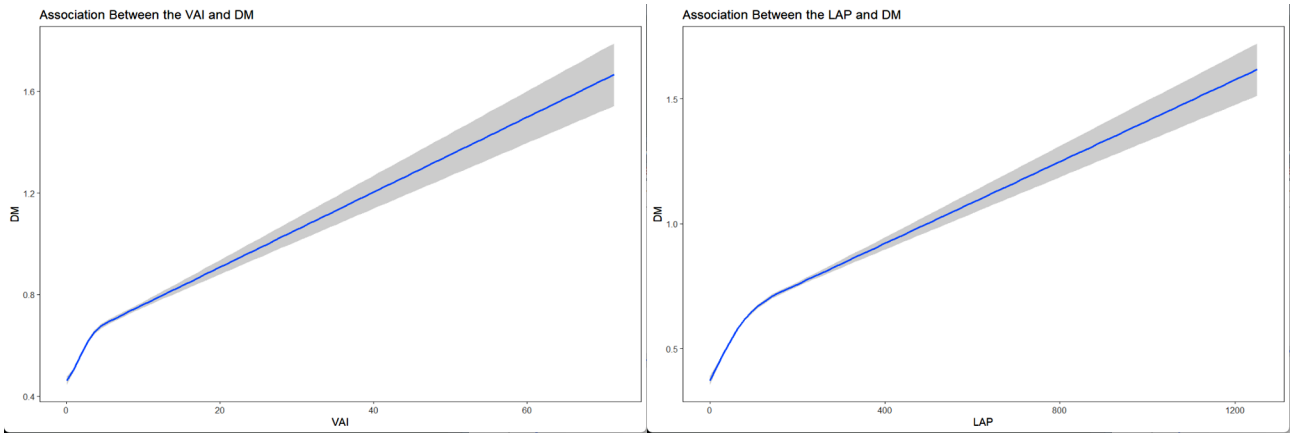

Fig. 2 The nonlinear relationship between VAI or LAP and T2DM. Associations between VAI and LAP with T2DM were evaluated by RCS after adjustment for the covariables. The solid blue lines correspond to the central estimates, and the gray-shaded regions indicate the 95% confidence intervals

Smoking habits were distinct, with a higher percentage of former smokers in the T2DM group (36%) compared to the non-T2DM group (27%), and a lower percentage of current smokers in the T2DM group (15% vs. 23% in the non-T2DM group) ($p<0.001$). Alcohol consumption patterns were significantly different, with a higher percentage of former alcohol users in the T2DM group (21%) compared to the non-T2DM group (14%), and a lower percentage of heavy drinkers in the T2DM group (15% vs. 19% in the non-T2DM group) ($p<0.001$).

The T2DM group had a significantly higher Visceral Adiposity Index (VAI) and Lipid Accumulation Product (LAP), indicating greater adiposity and lipid accumulation, respectively. Furthermore, the T2DM group had a lower average estimated Glomerular Filtration Rate (eGFR), suggesting reduced kidney function (83 vs. 89 in the non-T2DM group) ($p<0.001$). The prevalence of hyperlipidemia was also significantly higher in the T2DM group (88% vs. 77% in the non-T2DM group) ($p<0.001$), as was the prevalence of cardiovascular disease (CVD) (22% vs. 12% in the non-T2DM group) ($p<0.001$).

The association between VAI and LAP index with T2DM in individuals with hypertension

Table 2 illustrates the association between the VAI or LAP index and T2DM. It was observed that the higher the VAI group, the higher the risk of T2DM, consistent with the results for LAP. The higher VAI or LAP index levels were associated with an increased risk of T2DM compared with lower one. Since drug treatment and cardiovascular disease (CVD) can affect the effectiveness of VAI and LAP in determining T2DM, we adjusted for these two factors. Sensitive analysis showed that the VAI and LAP indices remained positively correlated with the occurrence of T2DM. Furthermore, there were nonlinear relationship between VAI or LAP and T2DM (Fig. 2 ,p for nonlinear<0.001).

Subgroup analysis of VAI and LAP index with T2DM

In individuals with hypertension/diabetes, the association between higher Visceral Adiposity Index (VAI) and Lipid Accumulation Product (LAP) with the risk of T2DM shows significant differences across age and gender groups. For VAI, the odds ratio (OR) for T2DM in the ≥60 age group is 1.37 (95% CI: 1.22, 1.53) compared to

1.16 (95% CI: 1.10, 1.23) in the <60 age group, indicating that older individuals have approximately 18% higher risk than younger individuals. For LAP, the OR in the ≥60 age group is 1.01 (95% CI: 1.01, 1.02) compared to 1.01 (95% CI: 1.00, 1.01) in the <60 age group, showing a slight increase in risk for older individuals. In terms of gender differences, the OR for T2DM associated with VAI is 1.39 (95% CI: 1.27, 1.52) in females and 1.13 (95% CI: 1.06, 1.21) in males, indicating that females have approximately 23% higher risk than males. For LAP, the OR is 1.02 (95% CI: 1.01, 1.02) in females and 1.01 (95% CI: 1.00, 1.01) in males, indicating a slightly higher risk in females.With increasing values of VAI and LAP, non-White individuals with hypertension exhibit a higher risk of developing T2DM compared to their White counterparts (Table 3).

Discussion

This study aimed to investigate the association between the VAI, LAP, and T2DM in US adults with hypertension using data from the NHANES from 2005 to 2018. Our findings indicate that both VAI and LAP are significantly associated with an increased risk of T2DM in individuals with hypertension. In our study, higher VAI and LAP indices were strongly associated with an increased risk of T2DM, even after adjusting for potential confounders such as age, sex, race, education, poverty-to-income ratio, smoking status, and drinking status. Our study also examined the association of VAI and LAP with T2DM after excluding individuals undergoing drug treatment and those with CVD. The results remained consistent, demonstrating that VAI and LAP are reliable markers for assessing T2DM risk in individuals with hypertension.

Table 3 Subgroup analysis of VAI and LAP index with T2DM

Characteristic	continuation	*p* for interaction
VAI		
Age		< 0.001
< 60	1.16(1.10,1.23)	
≥ 60	1.37(1.22,1.53)	
Gender		< 0.001
female	1.39(1.27,1.52)	
male	1.13(1.06,1.21)	
Race		0.008
Non-white	1.37(1.26,1.48)	
white	1.21(1.14,1.29)	
LAP		
Age		< 0.001
< 60	1.01(1.00,1.01)	
≥ 60	1.01(1.01,1.02)	
Gender		< 0.001
female	1.02(1.01,1.02)	
male	1.01(1.00,1.01)	
Race		0.002
Non-white	1.02(1.01,1.02)	
white	1.01(1.00,1.01)	

The results align with previous research exploring the role of visceral adiposity in metabolic disorders. For instance, a study by Amato et al. also found that VAI is a reliable marker of adipose dysfunction and is strongly associated with cardiometabolic risk, including T2DM [15]. Similarly, another study by Kahn et al. demonstrated that LAP is a practical index for identifying individuals at risk for metabolic syndrome and T2DM, independent of traditional anthropometric measures like BMI [25]. Comparable findings were reported in a study conducted by Chen et al. in a Chinese population, which showed that both VAI and LAP were better predictors of T2DM than BMI [26]. This reinforces the notion that visceral fat, rather than overall obesity, plays a more critical role in the development of diabetes, especially in individuals with hypertension who already have a predisposed risk due to elevated blood pressure and associated metabolic disturbances [27]. The recent study by Zhou et al. provides valuable insights that complement our findings. Their research, conducted on a general US population, found that VAI was significantly associated with an increased risk of diabetes. This finding aligns with our results in the US hypertensive population, thereby strengthening the evidence for VAI as a valuable tool in diabetes risk assessment. However, the variations in results also underscore the need for population-specific research and tailored approaches in clinical practice [28].

The mechanisms underlying the relationship between VAI, LAP, and T2DM involve complex interactions between adipose tissue, lipid metabolism, and insulin signaling pathways [29]. Adipose tissue, particularly visceral fat, secretes various bioactive substances that influence metabolic processes. These substances include adipokines (e.g., adiponectin and leptin) and pro-inflammatory cytokines (e.g., TNF-α and IL-6) [30, 31]. These cytokines can impair insulin signaling pathways by interfering with insulin receptor substrate (IRS) proteins and reducing glucose uptake in peripheral tissues [32, 33].

The study finds that older adults (≥60 years) have a higher odds ratio (OR) for T2DM associated with VAI and LAP compared to younger individuals (<60 years). This could be attributed to age-related metabolic changes, increased insulin resistance, and a longer duration of hypertension. These factors may amplify the detrimental effects of visceral adiposity, making older individuals more vulnerable to T2DM [34, 35]. Females exhibit a higher OR for T2DM in relation to both VAI and LAP compared to males. Hormonal differences, particularly the role of estrogen, and variations in fat distribution and metabolic responses are likely contributors. Estrogen can influence fat distribution and insulin sensitivity, potentially increasing females' susceptibility to

T2DM when visceral adiposity is high [36]. One of the intriguing findings of the study is the non-linear relationship between VAI, LAP, and T2DM risk. Unlike a straightforward linear relationship where risk increases consistently with higher levels of VAI and LAP, the study suggests the presence of thresholds or tipping points. Beyond certain levels of VAI and LAP, the risk of T2DM may escalate dramatically. This non-linearity could be due to complex metabolic interactions, genetic factors, and the cumulative impact of multiple risk factors over time. Understanding these non-linear dynamics is crucial for developing targeted interventions.

The findings of this study have several clinical implications. First, incorporating VAI and LAP into routine assessments of individuals with hypertension could enhance the identification of individuals at high risk for developing T2DM. Second, these indices provide valuable information about visceral adiposity and lipid overaccumulation, which are not captured by traditional measures such as BMI. Therefore, using VAI and LAP can offer a more comprehensive understanding of the metabolic health of individuals with hypertension. Despite its valuable insights, the study has several limitations. The study's cross-sectional nature limits the ability to establish causality between VAI, LAP, and T2DM. Longitudinal studies are needed to confirm these associations over time. Moreover, there may be other unmeasured factors, such as genetic predispositions or lifestyle variables not captured in the survey, that could influence the relationship between VAI, LAP, and T2DM. The study population was derived from NHANES data, which may not fully represent all individuals with hypertension, particularly those from different ethnic or socioeconomic backgrounds. Future research should aim for more diverse and representative samples. Therefore, the future research should expand on these findings through several parts as followed. While this study provides valuable cross-sectional data, longitudinal cohort studies are necessary to establish causal relationships between VAI, LAP, and T2DM. Tracking changes in these indices over time and their correlation with T2DM onset will offer deeper insights into the dynamic nature of these risk factors. Implementing RCTs to evaluate the effectiveness of targeted interventions aimed at reducing VAI and LAP levels could provide robust evidence for clinical practice. Future studies should consider incorporating genetic predispositions and more detailed lifestyle variables, such as diet and physical activity, to provide a comprehensive risk assessment. Understanding how these factors interact with VAI and LAP could lead to personalized prevention and treatment strategies.

Conclusion

Our study demonstrates that both VAI and LAP are significantly associated with an increased risk of T2DM in US adults with hypertension. These findings highlight the importance of considering visceral adiposity and lipid overaccumulation when assessing the risk of T2DM in individuals. Future research should focus on prospective studies to further validate these associations and explore the potential benefits of targeted interventions aimed at reducing VAI and LAP levels to prevent T2DM in this high-risk population.

Acknowledgements
We thank the investigators, staff, and participants of the US National Health and Nutrition Examination Survey (NHANES) for their valuable contributions.

Author contributions
CL contributed to the methodology, formal analysis, manuscript drafting, software development, investigation, data curation, and validation. RH handled visualization, conceptualization, review, editing, and supervision. Both authors have read and approved the final manuscript.

Funding
This work was supported by the Exploratory Study on the Impact of Semaglutide on Weight in Obese and Overweight individuals with Type 2 diabetes: A Randomized Controlled Trial and Potential Mechanisms of Weight Loss. [Grant Number: 2022ZD038].

Data availability
All data in the current analysis are publicly available on the NHANES website. [https://wwwn.cdc.gov/nchs/nhanes/Default.aspx].

Declarations

Ethical approval
This article does not contain any experiments on human participants or animals performed by any of the authors. Informed consent was obtained from all individual participants included in the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Endocrinology, The First Affiliated Hospital of Shihezi University, No. 107 Beier Road, Shihezi City, Xinjiang Uygur Autonomous Region, China

Received: 13 August 2024 / Accepted: 9 October 2024
Published online: 15 October 2024

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