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# Abdominal volume index is associated with higher oxidized LDL, high blood pressure and lower HDL among obese adults

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## Abstract

**Objectives** Central obesity is a well-known risk factor of numerous disease. Numerous indicators are developed for central obesity measurement, among them, abdominal volume index (AVI), reflecting *total volume of the abdomen*, precisely estimates the visceral fat volume. As a relatively new health measure and potent prognostic marker of metabolic disturbances, no study is available to investigate its role in cardio-metabolic health and oxidized LDL among obese young adults. In the current study we aimed to evaluate the association between abdominal volume index (AVI) with cardio-metabolic profile including serum lipids, glycemic markers of serum glucose, hemoglobin (Hb) A<sub>1</sub>C, insulin, oxidized LDL and blood pressure among young obese adults.

**Methods** Two hundred twenty young adults aged 18 to 25 years old with overweight or obesity were enrolled in the current study. Anthropometric measurements were done and AVI were calculated. Biochemical variables including serum total cholesterol (TC), low density lipoprotein cholesterol (LDL), high density lipoprotein cholesterol (HDL), triglyceride (TG), glycemic markers, including fasting serum glucose (FBS), insulin, hemoglobin (Hb) A<sub>1</sub>C and blood pressure were also measured with an automatic analyzer.

**Results** Participants in the third tertiles of AVI had higher body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR) ( $p < 0.001$  for all). Among biochemical variables, oxidized LDL, TG and HDL demonstrated significant associations across AVI tertiles in the first and second models, with higher oxidized LDL and TG and lower HDL levels observed in higher AVI tertiles ( $p < 0.05$ ). Moreover, those at the highest AVI tertiles showed significantly higher odds ratios for elevated cardio-metabolic index and systolic and diastolic blood pressures compared to the first tertiles ( $p < 0.05$ ).

**Conclusions** In the current study, we comprehensively investigated the association between AVI with cardio-metabolic health in young obese adults and accordingly, AVI was unfavorably associated with metabolic health among obese adults. Further studies are needed to elaborate the underlying mechanisms.

**Clinical trial number** Not applicable.

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**Keywords** Abdominal volume index, AVI, Central obesity, Cardio-metabolic health, Oxidized LDL, Obesity

## Introduction

Anthropometry, the science of measuring the human body, offers a wealth of insights into human's physical composition, proportions, and structural characteristics [1]. From determining body fat percentage to assessing muscle distribution, anthropometric measurements provide invaluable data that can inform lifestyle [2–4]. Abdominal volume index (AVI) is a quantitative measure of abdominal volume, specifically focusing on visceral fat accumulation and intra-abdominal pressure [5]. AVI emerges as a pivotal metric offering nuanced insights into health and performance [6]. Derived from anthropometric measurements, AVI goes beyond traditional metrics like body mass index (BMI) to provide a more comprehensive assessment of abdominal adiposity and its implications for metabolic health [7]. AVI holds significant implications for long-term health among young adults [8].

Research has consistently shown that abdominal fat distribution and central obesity, particularly visceral adiposity, plays a significant role in the development of cardiovascular diseases (CVD), type 2 diabetes, and metabolic syndrome. Excess visceral fat accumulation is associated with increased insulin resistance, systemic inflammation, and dyslipidemia, all of which elevate the risk of type 2 diabetes and cardiovascular disease [9–11]. Visceral adipose tissue actively secretes pro-inflammatory cytokines, such as interleukin-6 and tumor necrosis factor-alpha, which promote chronic low-grade inflammation and endothelial dysfunction [11–13]. Excessive visceral fat accumulation not only compromises physical performance by impairing mobility and agility [8], but also poses a heightened risk of metabolic disorders such as insulin resistance, dyslipidemia, and cardiovascular disease [10, 14, 15].

Numerous researches indicate that AVI serves as a potent predictor of cardio-metabolic risk factors, including insulin sensitivity, lipid profiles, and blood pressure dynamics among different populations; in two studies by Perona JA et al. [16, 17], AVI was reported as the strongest predictor of metabolic syndrome among Spanish adolescents. Its strong association with high blood pressure [18, 19], diabetes and impaired glucose tolerance [5, 20] and glycemic control [21], in general population or in patients of different metabolic disorders (e.g. obesity, diabetes,...) is well studied before.

AVI, by quantifying the volume of abdominal fat, has been linked to adverse cardio-metabolic outcomes independent of traditional metrics like body mass index (BMI) [7]. Studies have demonstrated that individuals with higher AVI tend to have greater visceral fat

accumulation, which is associated with increased insulin resistance, dyslipidemia, and inflammation, all of which are key contributors to cardio-metabolic disorders [5, 10, 18]. A cross-sectional study by Ramírez-Manent JA [22], showed that AVI was a strong predictor of high values of insulin resistance among subjects with metabolic syndrome and insulin resistance, given its more direct correlation with visceral fat volume. Additionally, longitudinal studies have shown that elevated AVI is associated with higher risks of type 2 diabetes, atherosclerosis, and some types of cancers [20, 23]. Notably, the relationship between AVI and cardiometabolic health has been observed across diverse populations, including different age groups and ethnic backgrounds, suggesting its utility as a global biomarker for metabolic risk. Furthermore, interventions aimed at reducing abdominal fat, such as exercise and dietary modifications, have been shown to improve AVI scores, subsequently lowering the risk of developing cardiometabolic diseases [12, 24].

Cardio-metabolic risk index (CMI), a new metric derived from the triglyceride-glucose index and waist to height ratio, has been emerged as a potentially prognostic marker for cardiovascular risk assessment in different studies [21, 25, 26]. Most of the studies have explored its association with metabolic parameters among patients with diabetes or cardiovascular disease [21, 25, 26], while the correlation of the CMI with specific pathological and metabolic parameters among young adults that are apparently healthy is not explored yet.

Early identification and prevention of abdominal obesity in young adults aged 18 to 25 are crucial due to its long-term health effects. This age group is particularly susceptible to lifestyle factors, such as poor diet and physical inactivity, which contribute to fat accumulation and increase the risk of obesity-related comorbidities [27, 28]. Abdominal obesity is strongly linked to insulin resistance, a key precursor to metabolic syndrome, highlighting the need for early intervention. Promoting regular physical activity and healthier dietary habits can significantly reduce abdominal fat and lower the risk of chronic diseases in later life [29, 30].

In the current study, our hypothesis was to evaluate AVI and its association with metabolic parameters including lipid profile, oxidized low density lipoprotein cholesterol (LDL), glycemic markers and CMI among young adults.

## Methods

### Participants

The study recruited a group of young adults aged between 18 and 25 years old that were enrolled from

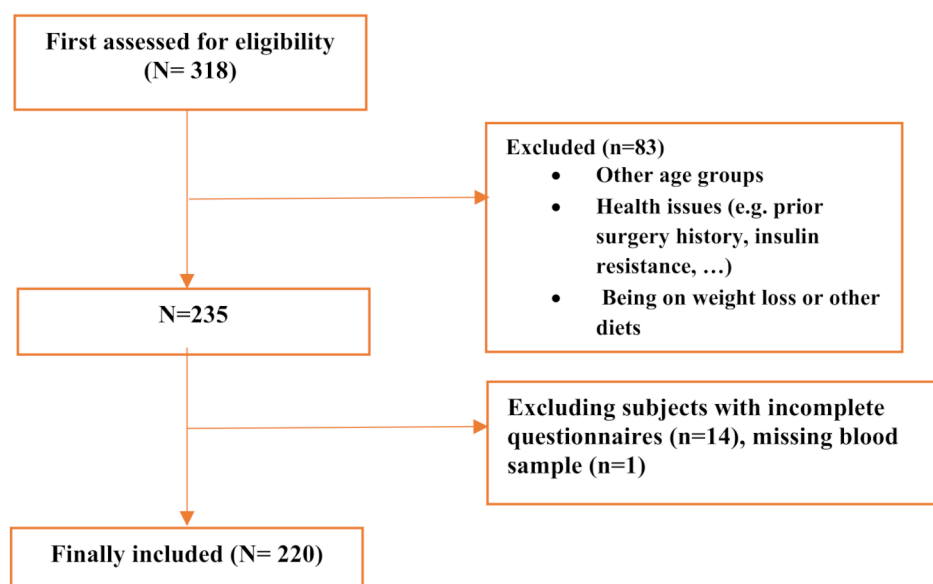
health clubs, academic centers through targeted advertisements. Participants were chosen to reflect a range of demographic backgrounds, including different socioeconomic statuses, physical activity levels, and dietary habits. Advertisements for the study were distributed both online and offline, targeting university campuses, fitness clubs or health centers to ensure broad outreach. Inclusion criteria were aged between 18 and 25 years old, having  $\text{BMI} \geq 25 \text{ kg/m}^2$ , no chronic disease or conditions including cardiovascular disorders, kidney or liver disease, diabetes, hypertension or any kinds of disability. A convenience sampling method was used. The study flowchart is represented in Fig. 1.

#### Anthropometric and dietary assessments

Anthropometric measurements were conducted following standardized procedures by trained personnel to ensure accuracy and consistency across all assessments. Body weight was measured using a calibrated digital scale while participants were barefoot and wearing minimal clothing to the nearest 0.1 kg. Standing height was measured using a stadiometer with participants in an upright posture. Waist circumference (WC) was measured at the narrowest point between the lower costal margin and iliac crest using a non-stretchable measuring tape. Hip circumference (HC) was measured at the widest point of the buttocks with participants standing upright. Waist-to-height ratio (WHtR) was calculated. A measuring tape was placed horizontally around the hips. Measurements for height, WC and HC were recorded to the nearest 0.1 cm. Abdominal volume index (AVI) was calculated as follows:  $\{2 \times \text{WC (cm)}^2 + 0.7 \times (\text{WC (cm)} - \text{hip (cm)})^2\}$

/1000 as previously described by Guerrero-Romero F et al. [5]. Waist to hip ratio (WHR) and waist to height ratio were calculated.

Mid-arm circumference (MAC) was measured at the midpoint between the acromion process of the scapula and the olecranon process of the ulna with the arm relaxed and hanging freely. Thigh circumference (THC) was measured at the midpoint between the inguinal crease and the proximal border of the patella with participants standing upright and legs slightly apart. Calf circumference (CC) was measured at the widest point of the calf muscle with the participant in a seated position and knees bent at a 90-degree angle. Measurements for height, WC, HC, MAC, THC and CC were recorded to the nearest 0.1 cm. Diet was assessed by a validated food frequency questionnaire (FFQ) with 199 food items and an acceptable validity and reliability [31]. The FFQ consisted of a list of commonly consumed food items, with participants asked to report the frequency and portion size of each item in the daily, weekly, monthly or yearly manner. The final quantitative data were entered to Nutritionist IV software to analysis nutritional intake. Body composition assessments were conducted using BIA (Inbody 770 Co., Seoul, Korea) in accordance with the manufacturer's guidelines. Measurements were taken with participants in a fasting state and after urination. Prior to the measurements, participants were instructed to remove any metal objects or jewelry and abstain from consuming caffeinated beverages and spices for at least 12 h [32]. According to the World Health Organization (WHO), the definitions for overweight and obesity are based on Body Mass Index (BMI): overweight BMI



**Fig. 1** Study flowchart

25–29.9 kg/m<sup>2</sup> and obesity a BMI of 30 kg/m<sup>2</sup> or higher [33]. Physical activity was measured with a validated short form of international physical activity questionnaire (IPAQ) [34].

#### Biochemical assays and CMI calculation

Blood samples were collected from participants following an overnight fast to ensure standardized conditions for biochemical analyses. Venous blood samples were drawn by trained phlebotomists using sterile techniques and collected into appropriate vacutainer tubes for subsequent laboratory analysis. Biochemical assays including serum total cholesterol (TC), high-and low density lipoprotein cholesterol (HDL-C, LDL-C), triglyceride (TG) and oxidized LDL (ox-LDL) were measured by standard laboratory assays with an auto-analyzer (Alpha Classic E analyzer). Serum insulin was measured by commercial kits (*AccuBind*, USA, Monobind Inc.). Hemoglobin (Hb) A1C was measured with an automatic analyzer (SySMEX HLC-723G8). Blood pressure was measured with a standard mercury sphygmomanometer. CMI was calculated as  $CMI = TG / HDL-C \times WHtR$  [35].

The criteria for categorization of serum lipids (e.g. LDL, HDL, TG, TC), and blood pressure were performed according to the national cholesterol education program adult treatment panel (NCEP-ATP) III criteria [36]. High serum insulin and ox-LDL levels were defined as  $\geq 15 \mu U/mL$  [37] and  $\geq 1.48 \text{ mg/dL}$  [38] respectively. For CMI, the highest tertile was used as the higher category.

#### Statistical analysis

Descriptive statistics, including means and standard deviations, were calculated for continuous variables, while frequencies and percentages were computed for categorical variables. Normality of continuous variables

was assessed using Shapiro-Wilk test. For comparisons between groups, one-way analysis of variance (ANOVA) was employed for continuous variables with post-hoc Tukey's test for pairwise comparisons. Chi-squared test was utilized for categorical variables. Multivariate regression analysis was performed to explore the association between anthropometric measurements, biochemical variables, and cardio-metabolic risk indices. Models were adjusted for potential confounding factors, including age, sex, BMI, physical activity, and energy intake. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated to quantify associations. All statistical analyses were conducted using SPSS version 23, with statistical significance set at  $p < 0.05$ . The required sample size for this cross-sectional study was determined based on an estimated prevalence of the central obesity, considering 95% confidence level and margin of error 5%. Using the following formula:  $\frac{Z^2 P (1-P)}{d^2}$ ; where: n, is the estimated sample size; z is the z-score for the 95% confidence level; P is the prevalence of obesity in target population and d is the margin error. Assuming an obesity prevalence of 32% according to previous study [39] and a margin error of 5%, the minimum required sample size was calculated to be 198 participants. Considering a potential non-response rate of 10%, the final target sample size was adjusted to 220 participants. According to the study power (1- $\beta$ ) of 0.2 and  $\alpha$  of 5%, the AVI was categorized into tertiles to avoid power loss in each subgroup [40]; therefore, AVI in each tertile was categorized as tertile 1, 18.30–19.45; tertile 2, 22.70–23.28 and tertile 3, 27.10–24.48.

#### Results

The demographic characteristics of study participants, stratified by tertiles of AVI, are presented in Table 1. Among the 220 participants included in the analysis,

**Table 1** General demographic characteristics of study participants by tertiles of abdominal volume index

Variable	All participants (n = 220)		Tertiles of AVI						P* Value
	Mean	SD	T <sub>1</sub> (n=73)		T <sub>2</sub> (n=74)		T <sub>3</sub> (n=73)		
			Mean	SD	Mean	SD	Mean	SD	
Age (y)	23.03	6.58	20.91	6.09	25.50	6.61	23.39	6.38	<b>0.003</b>
Sex (% Male)	105	52	7	14.3	42	56.8	56	70.9	<b>&lt;0.001</b>
BMI (kg/m <sup>2</sup> )	34.45	4.03	32.00	2.81	33.45	3.20	36.91	4.08	<b>&lt;0.001</b>
WC (cm)	98.64	9.82	96.36	5.69	97.10	2.59	100.69	6.53	<b>&lt;0.001</b>
HC (cm)	116.45	8.55	113.14	6.53	114.24	7.83	120.56	8.72	<b>&lt;0.001</b>
WHR	0.930	0.07	0.85	0.06	0.94	0.06	0.97	0.05	<b>&lt;0.001</b>
WHtR	0.65	0.06	0.59	0.039	0.64	0.038	0.69	0.053	<b>&lt;0.001</b>
MAC (cm)	28.77	3.71	29.03	4.34	28.60	3.61	28.20	3.27	0.460
THC (cm)	55.58	6.21	56.85	5.9	54.84	6.63	55.15	5.97	0.182
CC (cm)	36.96	3.49	37.68	3.75	36.65	3.69	36.71	3.27	0.235
FM (%)	23.81	8.13	22.31	5.85	21.64	7.11	26.54	9.43	<b>0.002</b>
FFM (%)	62.25	12.35	50.91	7.30	62.62	11.70	68.56	10.52	<b>&lt;0.001</b>

T, tertile; AVI, Abdominal Volume Index; BMI, Body mass index; WC, Waist Circumference; HC, hip circumference; WHR, waist-to-hip ratio; WHtR, waist to height ratio; MAC, mid-arm circumference; THC, thigh circumference; CC, calf circumference; FM, Fat Mass; FFM, Fat Free Mass; all data are mean ( $\pm$  SD) except sex that is presented as the number and percent of males. P\* values derived from One-Way ANOVA with Tukey's post-hoc comparisons. \*\* P values derived from chi-squared test

significant differences were observed across AVI tertiles for various demographic variables. Participants in the highest tertile of AVI (T3) were found to be older, with a mean age of 25.50 years, compared to those in the lower tertiles (tertile 1 and 2) (20.91 years and 23.39 years, respectively;  $p=0.003$ ). Additionally, there was a significant difference in the distribution of sex across AVI tertiles ( $p<0.001$ ), with tertile 3 exhibiting a higher proportion of male participants (70.9%) compared to tertiles 1 and 2. Body mass index (BMI) showed a significant gradient across AVI tertiles ( $p<0.001$ ), with participants in tertile 3 demonstrating the highest mean BMI (36.91 kg/m<sup>2</sup>) compared to tertile 1 and 2. Similarly, waist circumference (WC), hip circumference (HC), waist-to-hip ratio (WHR), and waist-to-height ratio (WtHR) exhibited significant differences across AVI tertiles ( $p<0.001$  for all), with participants in T3 showing the highest values. However, MAC, THC, and CC did not significantly differ across AVI tertiles ( $p>0.05$  for all). Furthermore, fat mass (FM) and fat-free mass (FFM) showed significant differences across AVI tertiles ( $p=0.002$  and  $p<0.001$ , respectively), with participants in T3 having higher FM and lower FFM compared to tertile 1 and 2. Table 2 displays the dietary intake of study participants stratified by tertiles of AVI. Analysis revealed no significant differences in energy, macronutrients and food groups' intake across AVI tertiles ( $p>0.05$  for all). Table 3 illustrates the association between biochemical variables and AVI among study participants. Glucose levels, HbA1c, insulin, HOMA-IR, Ox-LDL, LDL, TC, and TG did not show significant associations across AVI tertiles in most models. However, HDL demonstrated significant associations across AVI tertiles in the first and second models, with lower HDL levels observed in higher AVI tertiles ( $p<0.05$ ). Moreover, CMI, SBP, and DBP exhibited significant associations across AVI tertiles in all models, with higher AVI tertiles showing increased odds ratios

for elevated CMI, SBP, and DBP compared to the reference tertile ( $p<0.05$ ). Figure 2 illustrates the heat map of the distribution of CMI by sex according to different AVI tertiles.

## Discussion

In the current research, AVI was a positive predictor of cardiometabolic parameters among young adults. Higher AVI was associated with higher CMI, oxidized LDL, TG, SBP and DBP and lower HDL in our study. Abdominal adiposity, characterized by excess fat accumulation in the abdominal region, is strongly associated with an increased risk of cardiovascular diseases (CVD) and metabolic disorders such as insulin resistance, type 2 diabetes, and dyslipidemia [5, 8, 10]. In young adults even, who typically engage in physical activity, the distribution of adipose tissue, particularly visceral adipose tissue (VAT) around the abdominal organs, can significantly impact cardio-metabolic risk [41, 42]. In young adults aged 18 to 25, early identification and prevention of abdominal obesity are particularly important due to its long-term impact on health. Studies have shown that the accumulation of abdominal fat during these formative years increases the likelihood of continued obesity and related comorbidities in later life [27]. This age group is particularly vulnerable to lifestyle factors, such as poor dietary habits and lack of physical activity, which contribute to the onset of abdominal obesity [28]. Furthermore, abdominal obesity in young adults is strongly associated with the development of insulin resistance, a key precursor to metabolic syndrome [43]. Early interventions, including promoting regular physical activity and improving dietary patterns, have been shown to significantly reduce abdominal fat and mitigate the associated health risks [29, 30]. Given that abdominal obesity often remains underdiagnosed in this population, increased awareness and prevention efforts targeting young adults

**Table 2** Dietary intake of participants according to abdominal volume index

Variable	Tertiles of AVI						P* Value
	T <sub>1</sub> (n=73)		T <sub>2</sub> (n=74)		T <sub>3</sub> (n=73)		
	Mean	SD	Mean	SD	Mean	SD	
Energy (kcal/d)	2962.27	1002.77	3121.80	1114.99	3067.06	1075.77	0.721
Carbohydrate (%)	0.56	0.08	0.58	0.05	0.58	0.07	0.157
Protein (%)	0.12	0.02	0.13	0.02	0.13	0.02	0.578
Fat (%)	0.33	0.08	0.31	0.05	0.30	0.06	0.077
Fruit (g/d)	565.41	394.66	542.37	327.01	602.52	541.58	0.693
Vegetables (g/d)	381.13	414.55	337.83	227.87	329.33	231.16	0.586
Grains (g/d)	517.47	239.08	611.69	261.58	613.90	259.75	0.078
Low fat dairy (g/d)	258.61	193.22	259.01	222.07	245.18	189.12	0.896
High fat dairy (g/d)	102.30	126.95	128.32	146.17	93.11	111.70	0.227
MFP (g/d)	68.11	12.08	53.31	28.80	73.36	36.53	0.548

T, tertile; AVI, Abdominal Volume Index; MFP, meat, fish, poultry. P\* values derived from energy-adjusted ANCOVA, except for energy intake that is obtained from ANOVA



**Table 3** Association between biochemical variables and abdominal volume index among participants

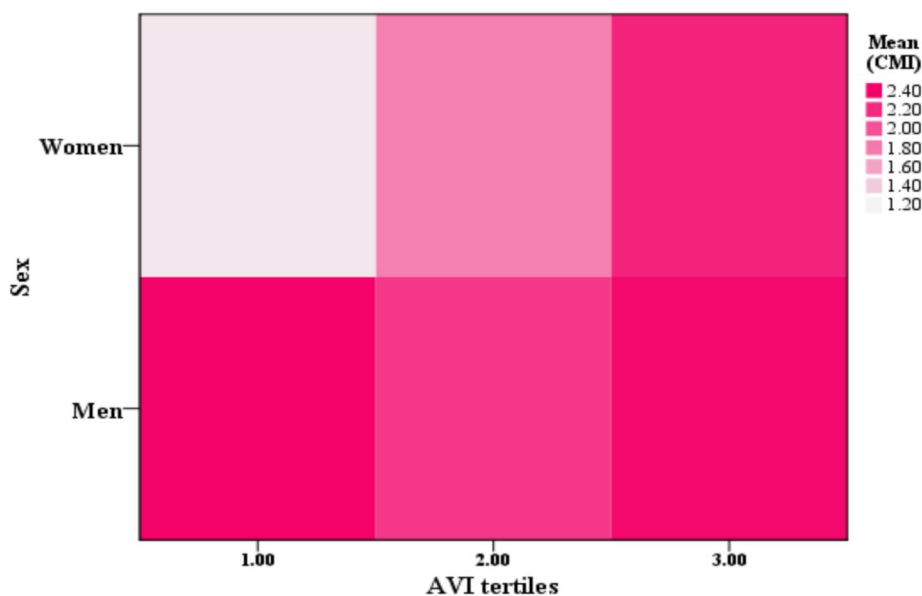
Variable		Tertiles of AVI				
		T <sub>1</sub> (n=73)	T <sub>2</sub> (n=74)		T <sub>3</sub> (n=73)	
			OR(CI)	P-value	OR(CI)	P-value
Glucose (mg/dl)	1st Model	Ref.	1.012 (0.986–1.038)	0.381	1.018 (0.993–1.044)	0.160
	2nd Model		0.999 (0.974–1.025)	0.935	1.000 (0.976–1.026)	0.973
	3rd Model		0.990 (0.958–1.024)	0.562	0.985 (0.948–1.024)	0.438
Hb A <sub>1</sub> C (%)	1st Model	Ref.	1.003 (0.970–1.053)	0.780	1.003 (0.980–1.089)	0.670
	2nd Model		1.016 (0.998–1.078)	0.560	1.034 (0.99–1.095)	0.324
	3rd Model		1.005 (0.973–1.089)	0.540	1.078 (0.998–1.109)	0.342
Insulin (mIU/l)	1st Model	Ref.	1.006 (0.964–1.050)	0.772	1.011 (0.971–1.054)	0.594
	2nd Model		1.016 (0.972–1.063)	0.470	1.024 (0.977–1.073)	0.324
	3rd Model		1.017 (0.970–1.067)	0.483	1.032 (0.967–1.103)	0.342
Ox-LDL (mg/dl)	1st Model	Ref.	0.999 (0.988–1.001)	0.567	1.003 (0.998–1.001)	0.560
	2nd Model		0.989 (0.978–1.007)	0.430	0.998 (0.967–1.013)	0.678
	3rd Model		0.994 (0.978–1.011)	0.493	1.001 (1.012–1.022)	<b>0.035</b>
LDL (mg/dl)	1st Model	Ref.	0.998 (0.987–1.010)	0.777	1.003 (0.992–1.015)	0.589
	2nd Model		0.995 (0.983–1.008)	0.440	0.999 (0.985–1.012)	0.833
	3rd Model		0.994 (0.978–1.011)	0.493	1.001 (0.981–1.022)	0.912
HDL (mg/dl)	1st Model	Ref.	0.959 (0.920–0.999)	<b>0.044</b>	0.927 (0.888–0.968)	<b>0.001</b>
	2nd Model		0.978 (0.936–1.021)	0.313	0.953 (0.908–0.999)	<b>0.046</b>
	3rd Model		0.981 (0.929–1.036)	0.487	0.933 (0.868–1.003)	0.059
TG (mg/dl)	1st Model	Ref.	1.006 (0.999–1.013)	0.086	1.008 (1.001–1.015)	<b>0.024</b>
	2nd Model		1.002 (0.994–1.010)	0.669	1.002 (0.994–1.010)	0.600
	3rd Model		1.002 (0.991–1.013)	0.771	1.002 (0.990–1.014)	0.774
TC (mg/dl)	1st Model	Ref.	0.998 (0.987–1.009)	0.689	1.002 (0.992–1.013)	0.710
	2nd Model		0.994 (0.982–1.006)	0.307	0.997 (0.985–1.009)	0.611
	3rd Model		0.995 (0.980–1.009)	0.462	0.997 (0.979–1.016)	0.749
CMI	1st Model	Ref.	1.850 (1.183–2.918)	<b>0.007</b>	2.294 (1.464–3.594)	<b>&lt;0.001</b>
	2nd Model		1.507 (0.965–2.353)	0.071	1.764 (1.120–2.779)	<b>0.014</b>
	3rd Model		1.576 (0.968–2.564)	0.067	1.876 (1.059–3.325)	<b>0.031</b>
SBP (mmHg)	1st Model	Ref.	1.019 (0.994–1.045)	0.139	1.067 (1.038–1.098)	<b>&lt;0.001</b>
	2nd Model		1.017 (0.992–1.043)	0.185	1.059 (1.027–1.092)	<b>&lt;0.001</b>
	3rd Model		1.014 (0.981–1.047)	0.416	1.054 (1.007–1.103)	<b>0.025</b>
DBP (mmHg)	1st Model	Ref.	1.010 (0.980–1.041)	0.535	1.043 (1.011–1.077)	<b>0.009</b>
	2nd Model		1.014 (0.979–1.050)	0.451	1.041 (1.003–1.080)	<b>0.034</b>
	3rd Model		1.014 (0.968–1.062)	0.552	1.038 (0.981–1.098)	0.194

T, tertile; AVI, Abdominal Volume Index; HbA<sub>1c</sub>, hemoglobin A<sub>1c</sub>; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; Ox-LDL-C, Oxidized Low Density Lipoprotein Cholesterol; HDL-C, High Density Lipoprotein Cholesterol; TG, Triglyceride; TC, Total Cholesterol; CMI, cardiometabolic index; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; The multivariate multinomial logistic regression was used for estimation of ORs and confidence interval (CI). Model I: crude, Model II: adjusted for age and sex, Model III: adjusted for age, BMI, sex, physical activity and energy intake

are essential to reduce the burden of chronic diseases later in life.

Excessive abdominal adiposity leads to an imbalance in adipokine secretion, with increased production of pro-inflammatory adipokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-alpha), and decreased secretion of anti-inflammatory adipokines like adiponectin [13, 44]. This imbalance contributes to chronic low-grade inflammation, insulin resistance, and endothelial dysfunction, all of which are key components of the cardiometabolic syndrome [45]. Additionally, visceral fat accumulation is associated with increased release of free fatty acids (FFAs) into the bloodstream, leading to lipid accumulation in non-adipose tissues such as the liver,

pancreas, and skeletal muscle. This ectopic lipid deposition further exacerbates insulin resistance and dyslipidemia, contributing to the development of CVD [9, 11, 46]. Additionally, adipose tissue-derived factors such as leptin, adiponectin, interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha) can directly influence vascular tone, endothelial function, and renal sodium handling, thereby affecting blood pressure regulation [47, 48]. Also, visceral fat accumulation is associated with increased production of angiotensinogen, which is a precursor of angiotensin II, a potent vasoconstrictor involved in the renin-angiotensin-aldosterone system (RAAS) pathway. Dysregulation of the RAAS can lead to hypertension [49]. While adults in young ages typically



**Fig. 2** Heat Map graph of the distribution of CMI by sex according to different AVI tertiles

engage in some types of physical activity, which may help mitigate abdominal adiposity and reduce dyslipidemia and hypertension risk, other factors such as genetic predisposition, dietary habits due to poor nutritional knowledge, and lifestyle behaviors may also influence metabolic profile and blood pressure in these population [50, 51].

In the current study, we did not find any significant association between AVI and glycemic markers, including glucose, HbA1C, and insulin levels, across all models. These findings suggest that AVI may not be an important predictor of glycemic control in this specific population. The participants in this study were likely relatively young, with an average age that may not yet be associated with significant changes in glycemic markers. Previous research has shown that the relationship between abdominal fat and insulin resistance, as well as glucose metabolism, may become more apparent after a certain age. For example, a study by Liu et al. demonstrated that visceral fat began to significantly correlate with insulin resistance and impaired glucose tolerance in individuals over the age of 40, with these effects being more pronounced as participants aged [52]. Thus, the lack of association in this younger cohort may be due to the relatively early stage of metabolic changes, which might not yet manifest in significant alterations in glycemic markers.

Also, we find that the association between AVI and CMI is more pronounced in men rather than women (Fig. 2). It is important to consider that sex-specific differences may play a significant role in the association between abdominal fat and glycemic markers. Several studies suggest that men and women exhibit distinct

patterns of fat distribution, which may influence how abdominal fat affects metabolic health. In men, abdominal fat tends to accumulate primarily in the visceral region, which is more metabolically active and associated with increased insulin resistance and higher risk for type 2 diabetes [53]. On the other hand, women tend to accumulate fat in both subcutaneous and visceral areas, but the hormonal changes during menopause—such as the decrease in estrogen levels—are associated with a shift toward more abdominal visceral fat accumulation [54]. This change may enhance the risk of metabolic disorders, including poor glycemic control, particularly in postmenopausal women [55].

The findings from this study have important clinical implications, particularly in the early identification and management of cardio-metabolic risk in young obese adults. Given that participants in the higher tertiles of the abdominal volume index (AVI) exhibited significantly poorer metabolic health markers, AVI appears to be a useful indicator of adverse metabolic changes. Clinically, AVI could be used as a more precise and reliable metric than traditional anthropometric measures, such as BMI alone, for assessing abdominal obesity and its associated risks. Also, our findings suggest that early intervention targeting abdominal fat reduction—through lifestyle modifications like diet and physical activity—could mitigate these risks and guide personalized treatment strategies aimed at improving lipid metabolism and reducing oxidative stress.

Assessing AVI in young ages, may provide valuable insights into their dyslipidemia and hypertension risk profile in the future. Young adults with higher AVI

values, indicative of greater abdominal adiposity, may be at increased risk of cardio-metabolic risks and hypertension and could benefit from targeted interventions to optimize their metabolic profile; lifestyle modifications, including dietary changes, weight management, regular physical activity, and stress reduction strategies, can help mitigate abdominal adiposity among young adults with elevated AVI values.

Current study has some strengths and limitations; one of the key strengths of this study is its comprehensive assessment of the abdominal volume index (AVI) and its relationship with cardiometabolic health markers in young obese adults. The use of AVI, a more precise measure of visceral fat accumulation, allows for a more detailed understanding of abdominal adiposity compared to traditional anthropometric measures such as body mass index (BMI). Furthermore, the study's focus on young adults (aged 18–25) is particularly valuable, as this age group is often overlooked in metabolic health research. Despite its strengths, this study has several limitations; first, the cross-sectional design of the study limits the ability to draw conclusions about causality. Also, focusing on a specific population of young, obese adults limits the generalizability of the findings to other age groups or populations. Finally, the potential for measurement bias exists, as the self-reported nature of certain lifestyle factors, such as diet and physical activity, may have led to inaccuracies in the data. Larger longitudinal studies may be needed to support the relationship between central obesity and metabolic health.

In conclusion, the association between AVI and increased CMI and blood pressure in young adults underscores the importance of assessing abdominal adiposity as a potential risk factor of cardiovascular disease and implementing preventive measures to optimize cardiovascular health in this population. Longitudinal studies are needed to elucidate the causal relationships between AVI, abdominal adiposity, and cardio-metabolic risk in young adults. Additionally, research exploring the effectiveness of targeted lifestyle interventions and pharmacological therapies in reducing the risk of cardiovascular disease in these population with abdominal adiposity is warranted.

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#### Author contributions

MAA was the supervisor and generated the main hypothesis of the study, MMA was also involved in data collection, and the main data collection was performed by KA, FHA, TA and YIA, these authors were also involved in registration of the study proposal, writing the first draft of the article, doing the assessments, HFL performed the statistics and NKAA was involved in lab works, all of the participants were involved in writing the first draft while MAA also revised the manuscript and finalized its submission. All of the authors agreed with its submission to the journal. MAMA was involved in revision

and data analysis, he also was involved in data analysis and supervised all of statistics in the current work.

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#### Data availability

The datasets of the current study are available from the corresponding author on reasonable request.

#### Declarations

##### Ethics approval and consent to participate

Written informed consent was obtained from all of the participants of the study. All methods in the current research were performed in accordance with the declaration of Helsinki's guidelines and regulations. The protocol of the current study has been approved by the Ethics Committee of University of Tabuk.

##### Consent for publication

Not applicable.

##### Competing interests

The authors declare no competing interests.

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