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ORIGINAL

Arterial stiffness, high fasting glucose, and fatty liver as risk factors for visceral obesity in middle-aged Chinese individuals: a cross-sectional study

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Abstract. The prevalence of obesity is increasing rapidly worldwide, particularly in Asia. Visceral obesity, characterized by intra-abdominal fat accumulation, is a precursor to metabolic syndrome, encompassing hyperglycemia, dyslipidemia, and hypertension, which elevate the risk of atherosclerosis and cardiovascular disease. A visceral fat area (VFA) of ≥100 cm^2 is a recognized threshold for diagnosing obesity-related metabolic syndrome. This study aimed to identify independent risk factors for VFA ≥100 cm^2 in middle-aged Chinese individuals from the general population. We analyzed data from 148 participants (mean age: 49.3 ± 10.8 years; 54% male) who underwent health check-ups. VFA and subcutaneous fat area were assessed using computed tomography, while arterial stiffness and fatty liver were evaluated *via* brachial-ankle pulse wave velocity (baPWV) and abdominal ultrasonography, respectively. Between-group comparisons (VFA ≥100 cm^2 *vs.* VFA <100 cm^2) were conducted using unpaired *t*-tests and Mann-Whitney U tests, and logistic regression analysis identified risk factors. Multivariable regression analysis revealed that baPWV ≥1,400 cm/s (odds ratio [OR] = 5.71, $p = 0.011$), waist circumference ≥85 cm (OR = 5.46, $p = 0.026$), fasting blood glucose (FBG) ≥100 mg/dL (OR = 5.69, $p = 0.030$), male sex (OR = 12.79, $p = 0.029$), and fatty liver (OR = 3.99, $p = 0.042$) were significant independent risk factors for VFA ≥100 cm^2. Among these, baPWV ≥1,400 cm/s was the most significant, showing a positive correlation with VFA ($r = 0.365$, $p < 0.001$). Visceral obesity (VFA ≥100 cm^2) is a critical target for interventions addressing metabolic syndrome, metabolic dysfunction-associated fatty liver disease (MAFLD), and cardiovascular disease, particularly in males.

Key words: Visceral obesity, Arterial stiffness, Metabolic syndrome, Metabolic dysfunction-associated fatty liver disease (MAFLD), Cardiovascular disease

Introduction

The prevalence of obesity is rising rapidly worldwide, becoming a major public health challenge, particularly in Asian countries where Westernized lifestyles, high-sugar and high-fat diets, and physical inactivity are prevalent. In China, overweight and obesity rates have climbed to 34.3% and 16.4%, respectively, based on Chinese body mass index (BMI) criteria of 24.0 to <28.0 kg/m^2 for overweight and ≥28.0 kg/m^2 for obesity.

These conditions are linked to increased risks of type 2 diabetes, hypertension, cardiovascular disease, and

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Abbreviations: BMI, body mass index; WC, waist circumference; VFA, visceral fat area; SFA, subcutaneous fat area; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein-cholesterol; FBG, fasting blood glucose; AST, aspartate aminotransferase; ALT, alanine aminotransferase; γ-GTP, γ-glutamyl transferase; DHEAS, dehydroepiandrosterone sulfate; hsCRP, high-sensitivity C-reactive protein; baPWV, brachial-ankle pulse wave velocity; IMT, intima-media thickness; MAFLD, metabolic dysfunction-associated fatty liver disease; NAFLD, non-alcoholic fatty liver disease

mortality [1]. Despite the importance of interventions, the risk factors for overweight and obesity remain poorly characterized, limiting effective prevention strategies and exacerbating the public health burden, including economic costs and healthcare system strain.

Intra-abdominal obesity, compared to other fat regions, is strongly associated with cardiovascular risk factors [2]. The Framingham Study demonstrated a significant link between visceral adiposity and cardiovascular disease ($p = 0.01$) [3].

Ethnic-specific differences in fat distribution are well-documented, particularly in Asia [4, 5]. While Asians generally exhibit lower BMIs compared to Caucasians, they display higher levels of visceral obesity when living in the United States or Canada [4, 5]. Moreover, morbidities linked to visceral obesity occur at lower BMIs in China [6], underscoring the limitations of BMI as a universal metric. These variations suggest that obesity-related risk assessments must consider ethnic and regional factors to improve diagnostic accuracy and intervention efficacy.

Visceral fat accumulation is a key contributor to type 2 diabetes, cardiovascular disease, and fatty liver [7]. Visceral obesity promotes ectopic fat deposition in the liver *via* free fatty acids delivered through the portal vein. This process contributes to insulin resistance, increased hepatic glucose production, and elevated proinflammatory cytokines (tumor necrosis factor-α, interleukin-6, plasminogen activator inhibitor-1) while reducing anti-atherogenic adiponectin [7, 8]. These metabolic disruptions highlight the critical role of visceral fat in the pathogenesis of chronic diseases, particularly in populations with high levels of intra-abdominal adiposity.

In Japan, computed tomography (CT) has been used to separately measure visceral fat area (VFA) and subcutaneous fat area (SFA) [9]. A VFA of ≥100 cm^2 was identified as a critical threshold for metabolic syndrome, associated with hyperglycemia, dyslipidemia, and hypertension, all risk factors for atherosclerotic cardiovascular disease [10]. This threshold correlates with elevated fasting glucose and insulin responses predictive of type 2 diabetes and cardiovascular disease [11] and contributed to defining metabolic syndrome globally [12]. The VACATION-J Study confirmed the utility of the VFA threshold, showing reductions in visceral fat through diet and exercise lower metabolic risk factors and cardiovascular events [13, 14]. These findings underscore the potential for lifestyle interventions in reducing disease burden and improving population health outcomes.

Recently, metabolic dysfunction-associated fatty liver disease (MAFLD) has been defined by the coexistence of fatty liver with overweight/obesity, type 2 diabetes, or metabolic dysregulation [15]. MAFLD underscores the link between fatty liver and metabolic syndrome, especially in Asia, where many individuals are non-obese but carry significant visceral fat. Addressing MAFLD requires targeted interventions accounting for these unique metabolic profiles and offers an opportunity to refine diagnostic and treatment approaches for metabolic disorders.

This study focuses on identifying risk factors for VFA ≥100 cm^2 in middle-aged Chinese individuals, aiming to inform public health interventions and clinical strategies tailored to populations with distinct fat distribution patterns. By elucidating these risk factors, the findings may contribute to more precise and effective solutions for managing obesity and its related complications.

Methods

Participants

This retrospective study reviewed data from 148 consecutive individuals aged 19–76 years (mean age: 49.3 ± 10.8 years; 80 males and 68 females) from the general population who underwent health check-ups at the Zhichengheai Health Management Center between April 2021 and July 2022. Among these participants, 14% were using antihypertensive drugs (VFA ≥100 cm^2: n = 14; VFA <100 cm^2: n = 7), 1.3% were using anti-dyslipidemia drugs, and 2.7% were using anti-diabetes drugs. The study was approved by the Institutional Ethics Committee review board and adhered to the principles of the Helsinki Declaration, as revised in 2013. Participants provided informed consent through an opt-out option available on the Zhichengheai Health Management Center homepage.

Hematology and Hormone Measurements

Morning blood samples were collected to measure concentrations of red blood cells (RBC), hemoglobin, albumin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), γ-glutamyl transferase (γ-GTP), creatinine, uric acid, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), non-HDL-C, triglycerides, fasting blood glucose, glycated hemoglobin (HbA1c), calcium, and high-sensitivity C-reactive protein (hsCRP). The estimated glomerular filtration rate (eGFR) was calculated from serum creatinine. Anthropometric data, including height, weight, waist circumference (WC), and BMI, were collected. WC was measured at the midpoint between the lower border of the costal arch and the anterior superior iliac spine. Systolic and diastolic blood pressure (SBP and DBP) were measured using a mercury sphygmomanometer with participants at rest in a seated position. Serum cortisol, dehydroepiandrosterone sulfate,

thyroid-stimulating hormone, free thyroxine, and 25-hydroxyvitamin D were measured *via* chemiluminescence enzyme immunoassays.

VFA and SFA Measurements

VFA and SFA were measured using automated CT imaging (Canon Aquilion ONE320, Canon Medical Systems Corporation, Tochigi, Japan) at the umbilical level in the supine position [9]. VFA was significantly correlated with total visceral fat volume [9].

baPWV Measurement

Arterial stiffness was assessed *via* brachial-ankle pulse wave velocity (baPWV), a non-invasive and reproducible method [16]. Measurements were performed using a blood pressure monitor (Omron BP-203RPE, Omron Corporation, Kyoto, Japan) after 15 minutes of supine rest. Bilateral brachial and ankle blood pressure, pulse pressure waveforms, and heart rate were recorded simultaneously. baPWV was calculated by dividing the distance between two points by the time difference [16]. Carotid intima-media thickness (IMT) and plaques, markers of atherosclerosis progression, were measured *via* ultrasound using a Philips A30/HITACHI A60 (Philips Ultrasound, Inc., WA, USA). Bone mineral density was assessed using an AOS-100E EggQUs (Hitachi, Ltd., Tokyo, Japan).

Liver Ultrasonography

Liver ultrasound examinations were conducted by a single experienced radiologist using a Philips A30/HITACHI A60 with a 06-2 probe (Philips (China) Investment Co., Shanghai, China). Fatty liver was diagnosed based on previously established criteria [17], involving increased echogenicity of the liver compared to the renal cortex.

Statistical Analysis

Data were expressed as mean ± standard deviation or as numbers with percentages. Relationships between variables were analyzed using Pearson's correlation coefficient (r). Continuous variables between groups were compared using unpaired t-tests or Mann-Whitney tests, depending on data distribution (normal or non-normal). Categorical variables were compared using Fisher's exact test. Logistic regression analysis was performed to identify factors associated with VFA ≥100 cm^2 and to calculate odds ratios (ORs) and 95% confidence intervals (CIs). Univariable and multivariable logistic regression analyses were conducted with and without adjustment for other variables. Variables with $p < 0.10$ were selected for inclusion in the logistic regression model. In cases of collinearity, one of the two collinear variables was excluded from the multivariable model. Receiver operating characteristic (ROC) curve analysis was used to determine cut-off values for continuous parameters, with an area under the curve (AUC) and 95% CI reported.

All statistical analyses were performed using SPSS version 18.0 (IBM, Advanced-AI, Tokyo, Japan), with a significance threshold of $p < 0.05$.

Results

Clinical Characteristics of the Participants

The clinical characteristics of the participants are presented in Table 1. Among the 148 participants, 83 had VFA ≥100 cm^2 (62 males and 21 females), while 65 had VFA <100 cm^2 (18 males and 47 females). The incidence of visceral obesity (VFA ≥100 cm^2) was 56.0%. Most participants were middle-aged (40–60 years) and exhibited a predisposition toward overweight and visceral obesity.

Compared to the VFA <100 cm^2 group, the VFA ≥100 cm^2 group had a significantly higher proportion of males and higher values for body weight, BMI, WC, VFA, SFA, SBP, DBP, RBC, hemoglobin, AST, ALT, γ-GTP, uric acid, LDL-C, non-HDL-C, triglycerides, FBG, HbA1c, hsCRP, baPWV, carotid IMT, the incidence of fatty liver, as well as higher rates of drinking and smoking. In contrast, HDL-C and the cortisol/DHEAS ratio were significantly lower in the VFA ≥100 cm^2 group.

Participants with VFA ≥100 cm^2 exhibited significantly elevated risk factors for metabolic syndrome and cardiovascular disease, including higher SBP, DBP, FBG, HbA1c, triglycerides, baPWV, and carotid IMT. Additionally, they showed elevated risk factors for MAFLD, including fatty liver, high AST, ALT, γ-GTP, hsCRP, and drinking.

Univariable and Multiple Regression Analysis

Univariable and multiple regression analyses were performed to identify risk factors for VFA ≥100 cm^2, as shown in Table 2. Univariable regression analysis identified the following significant risk factors: male sex, BMI ≥25 kg/m^2, WC ≥85 cm, SFA ≥165 cm^2, RBC ≥470 × 10^4/μL, hemoglobin ≥14.2 g/dL, ALT ≥29 U/L, uric acid ≥339.0 μmol/L, triglycerides ≥1.69 mmol/L, FBG ≥5.55 mmol/L, baPWV ≥1,400 cm/s, fatty liver, drinking, and smoking.

Multiple regression analysis identified the following independent risk factors for VFA ≥100 cm^2: baPWV ≥1,400 cm/s, WC ≥85 cm, FBG ≥5.55 mmol/L, male sex, and fatty liver.

Table 1 Clinical characteristics of participants stratified by VFA value

	All Participants $n = 148$	VFA ≥100 cm^2 $n = 83$	VFA <100 cm^2 $n = 65$	p values
Age (years)	49.3 ± 10.8	50.3 ± 10.4	48.1 ± 11.2	0.227[1)]
Male (%)	80 (54.1)	62 (74.7)	18 (27.7)	<0.001[2)]
Weight (kg)	67.2 ± 13.3	73.9 ± 12.1	58.7 ± 9.3	<0.001[1)]
BMI, (kg/m^2)	24.3 ± 3.6	26.0 ± 3.2	22.1 ± 2.9	<0.001[1)]
WC (cm)	85.4 ± 11.1	91.6 ± 8.3	77.5 ± 9.0	<0.001[1)]
VFA (cm^2)	111.0 ± 52.7	149.6 ± 32.5	61.7 ± 25.2	<0.001[1)]
SFA (cm^2)	165.5 ± 69.1	175.6 ± 71.7	152.5 ± 63.9	0.043[1)]
SBP (mmHg)	118.2 ± 15.2	123.7 ± 13.2	111.1 ± 14.7	<0.001[1)]
DBP (mmHg)	72.4 ± 10.3	76.0 ± 9.3	67.7 ± 9.7	<0.001[1)]
RBC ($\times 10^4/\mu L$)	472.3 ± 49.2	484.6 ± 40.9	456.6 ± 54.4	<0.001[1)]
Hemoglobin (g/dL)	14.2 ± 1.5	14.7 ± 1.2	13.4 ± 1.5	<0.001[1)]
Albumin (g/dL)	4.40 ± 0.21	4.42 ± 0.21	4.37 ± 0.21	0.188[1)]
AST (U/L)	25.4 ± 18.8	28.4 ± 22.7	21.4 ± 11.3	<0.001[3)]
ALT (U/L)	29.1 ± 28.6	37.4 ± 35.2	18.5 ± 9.2	<0.001[3)]
γ-GTP (U/L)	38.4 ± 32.8	49.7 ± 35.5	24.0 ± 22.1	<0.001[3)]
Creatinine (μmol/L)	63.65 ± 33.59	65.42 ± 15.03	61.88 ± 47.74	0.514[1)]
Uric acid (μmol/L)	338.44 ± 91.6	378.89 ± 88.03	286.69 ± 67.81	<0.001[1)]
eGFR ($mL/min/1.73\ m^2$)	118.1 ± 16.8	116.5 ± 15.0	120.1 ± 18.9	0.198[1)]
LDL-C (mmol/L)	2.85 ± 0.72	2.96 ± 0.74	2.7 ± 0.67	0.026[1)]
Non-HDL-C (mmol/L)	3.45 ± 0.99	3.67 ± 1.06	3.15 ± 0.88	0.002[1)]
HDL-C (mmol/L)	1.65 ± 0.43	1.48 ± 0.33	1.87 ± 0.46	<0.001[1)]
Triglyceride (mmol/L)	1.85 ± 1.66	2.34 ± 1.99	1.22 ± 0.71	<0.001[3)]
FBG (mmol/L)	5.42 ± 1.21	5.79 ± 1.34	4.95 ± 0.79	<0.001[1)]
HbA_{1c} (%)	5.72 ± 0.80	5.89 ± 0.88	5.51 ± 0.63	0.004[1)]
Calcium (mmol/L)	2.41 ± 0.08	2.42 ± 0.08	2.39 ± 0.08	0.056[1)]
hsCRP (mg/L)	2.52 ± 4.51	3.17 ± 5.61	1.68 ± 2.25	0.021[3)]
TSH (μIU/mL)	2.47 ± 1.80	2.55 ± 1.81	2.37 ± 1.81	0.205[3)]
FT4 (pmol/L)	16.22 ± 2.7	16.34 ± 2.96	16.09 ± 2.32	0.595[1)]
25 hydroxy vitamin D (pmol/L)	47.25 ± 16.5	48.5 ± 17.25	45.75 ± 15.50	0.323[1)]
DHEAS (nmol/L)	787.01 ± 468.05	825.15 ± 454.18	735 ± 457.64	0.241[1)]
Cortisol (nmol/L)	584.91 ± 1,442.96	504.9 ± 1,092.56	689.75 ± 1,801.63	0.780[3)]
Cortisol/DHEAS ratio	0.09 ± 0.12	0.08 ± 0.08	0.10 ± 0.15	0.045[3)]
baPWV (right side) (cm/s)	1,374 ± 252	1,453 ± 245	1,269 ± 313	<0.001[1)]
ABI (right side)	1.14 ± 0.10	1.14 ± 0.10	1.14 ± 0.10	0.860[1)]
Carotid IMT (right side) (mm)	0.77 ± 0.21	0.82 ± 0.21	0.72 ± 0.20	0.005[1)]
Carotid plaque (%)	38 (25.7)	–1.15 ± 0.86	15 (23.1)	0.573[2)]
BMD (T score)	–1.15 ± 0.86	–1.26 ± 0.77	–1.01 ± 0.94	0.069[1)]
Fatty liver (%)	61 (41.2)	52 (62.7)	9 (13.8)	<0.001[2)]
Drinking (%)	89 (60.1)	60 (72.3)	29 (44.6)	<0.001[2)]
Smoking (%)	36 (24.3)	29 (34.9)	7 (10.8)	<0.001[2)]

Data were expressed as means ± SD or numbers (%).
[1)] p value determined by an unpaired t test.
[2)] p value determined by a Fisher exact test or Mann-Whitney test.
[3)] p value determined by a Fisher exact test.

Table 2 Risk factors for VFA ≥100 cm^2 by the logistic regression analysis

Variables	Before adjustment		After adjustment	
	OR (95%CI)	*p* values	OR (95%CI)	*p* values
Male	7.71 (3.70–16.07)	<0.001	12.79 (1.30–126.27)	0.029
BMI ≥25 kg/m^2	7.07 (3.32–15.03)	<0.001	086 (0.20–3.74)	0.844
WC ≥85 cm*	15.51 (7.32–37.26)	<0.001	5.46 (12.3–24.23)	0.026
SFA ≥165 cm^2*	2.15 (1.09–4.21)	0.027	4.58 (0.89–23.44)	0.068
SBP ≥140 mmHg	2.83 (0.75–10.75)	0.126	0.58 (0.05–6.85)	0.663
RBC ≥470 × $10^4/\mu L$*	3.28 (1.66–6.47)	<0.001	0.29 (0.04–2.34)	0.244
Hemoglobin ≥14.2 g/dL*	7.73 (3.68–16.27)	<0.001	1.80 (0.17–19.08)	0.627
Calcium ≥2.4 mmol/L*	1.49 (0.78–2.87)	0.231	0.57 (0.17–1.86)	0.352
ALT ≥29 U/L**	5.72 (2.80–11.69)	<0.001	–1.15	0.302
Uric acid ≥339.0 μmol/L*	4.61 (2.28–9.339)	<0.001	0.44 (0.09–2.13)	0.306
Triglyceride ≥1.69 mmol/L	4.31 (2.07–8.97)	<0.001	1.44 (0.40–5.09)	0.576
FBG ≥5.55 mmol/L	7.91 (3.07–20.33)	<0.001	5.69 (1.18–27.42)	0.030
hsCRP ≥1.3 mg/L**	1.62 (0.84–3.12)	0.148	0.63 (0.18–2.18)	0.467
baPWV ≥1,400 cm/s	6.33 (2.77–14.48)	<0.001	5.71 (1.50–21.77)	0.011
BMD (T-score ≤–1.15)	1.58 (0.82–3.04)	0.170	1.69 (0.51–5.59)	0.391
Fatty liver (%)	10.44 (4.54–24.00)	<0.001	3.99 (1.05–15.12)	0.042
Drinking (%)	3.24 (1.63–6.43)	<0.001	0.53 (0.11–2.42)	0.411
Smoking (%)	4.45 (1.80–11.00)	0.001	2.16 (0.48–9.71)	0.315

OR, odds ratio; CI, confidence interval. * mean value, ** median value.

Table 3 Cut-off values and AUCs for VFA ≥100 cm^2 determined by ROC curve analysis

Variables	Cut-off value	AUC	95%CI	*p* values
WC	86 cm	0.872	0.816–0.928	<0.001
FBG	5.23 mmol/L	0.761	0.685–0.838	<0.001
baPWV	1,301 cm/s	0.753	0.672–0.834	<0.001

AUC, area under the curve; ROC, receiver operating characteristics; CI, confidence interval.

ROC Curve Analysis

ROC curve analysis was conducted to determine the cut-off values for continuous variables associated with VFA ≥100 cm^2, as presented in Table 3. For WC, the cut-off value was 86 cm, with a predictive value of 87% and an area under the curve (AUC) of 0.872 (95% CI: 0.816–0.928, $p < 0.001$). For FBG, the cut-off value was 5.23 mmol/L, with a predictive value of 76% and an AUC of 0.761 (95% CI: 0.685–0.838, $p < 0.001$). For baPWV, the cut-off value was 1,301 cm/s, with a predictive value of 75% and an AUC of 0.753 (95% CI: 0.672–0.834, $p < 0.001$).

Correlation Between VFA and baPWV

The relationship between VFA and baPWV was further analyzed (Fig. 1). A significant positive correlation was observed between VFA and baPWV ($r = 0.365$, $p < 0.001$).

Discussion

This study identified baPWV ≥1,400 cm/s, WC ≥85 cm, FBG ≥5.55 mmol/L, male sex, and fatty liver as significant independent risk factors for VFA ≥100 cm^2 among 148 individuals from the general Chinese population using multiple regression analysis. Among these, high baPWV was the most significant risk factor, with VFA significantly positively correlated with baPWV ($r = 0.365$, $p < 0.001$). These findings underscore the interplay between visceral obesity and arterial stiffness, with broader implications for cardiovascular health and metabolic disorders in middle-aged populations.

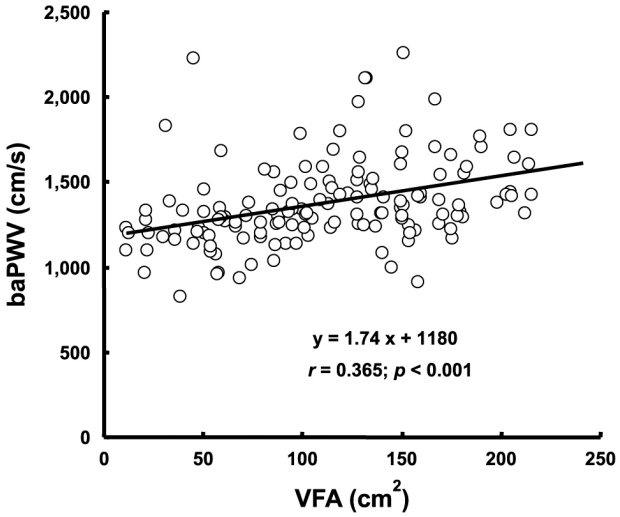


Fig. 1 Relationship between VFA and baPWV.
There was a significant correlation between VFA and baPWV in all participants.
VFA, visceral fat area; baPWV, brachial-ankle pulse wave velocity.

Arterial stiffness, measured by baPWV, is a recognized predictor of cardiovascular events and all-cause mortality, as demonstrated by studies using carotid-femoral PWV and baPWV [18-20]. Many previous studies used anthropometric parameters like BMI, WC, and waist-height ratio to explore the relationship between arterial stiffness and obesity but often failed to establish definitive associations [21-26]. In contrast, the current study supports recent evidence linking arterial stiffness directly to visceral fat mass, as indicated by correlations with baPWV and carotid-femoral PWV [27, 28]. This reinforces the significance of visceral obesity as a key driver of arterial stiffness.

Ethnic-specific WC cut-off values are central to the global definition of metabolic syndrome [12]. In this study, WC ≥85 cm emerged as a significant predictor of VFA ≥100 cm^2, aligning with findings from the Kadoorie Biobank and a large international CT imaging study [1, 29]. These results highlight WC as a practical and reliable metric for identifying visceral obesity, particularly in populations like the Chinese, where visceral adiposity is prevalent despite relatively lower BMI thresholds.

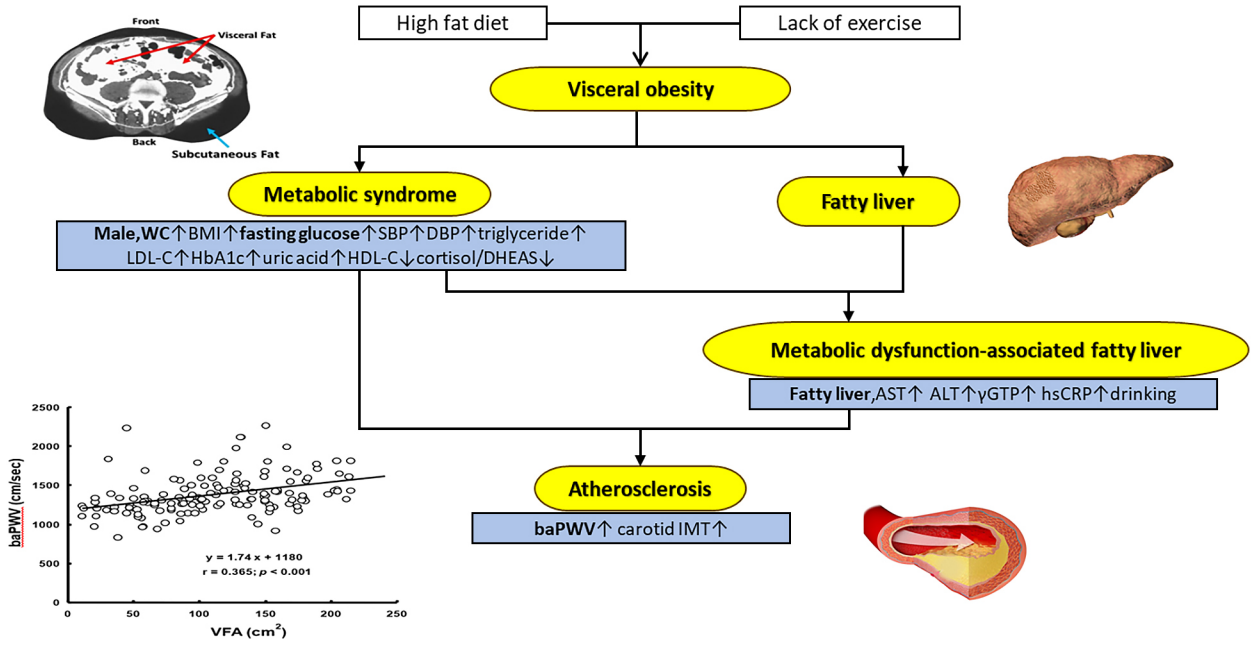
Male sex was another strong predictor for VFA ≥100 cm^2. The male's predisposition to android-type obesity, characterized by central fat accumulation, likely explains this finding. The higher incidence of visceral obesity in males (77.5%) compared to females (32.3%) in this study corresponds with higher dietary fat intake and alcohol consumption observed in males (88.1%) *versus* females (26.4%). These behavioral factors exacerbate the physiological tendency for visceral fat accumulation in males.

Visceral obesity's pathophysiological effects are well-documented. It facilitates ectopic fat deposition in the liver, promoting insulin resistance and increased hepatic glucose production *via* free fatty acids and triglycerides delivered through the portal vein [7]. The resulting metabolic disturbances, including elevated proinflammatory cytokines and reduced anti-atherogenic adiponectin, heighten risks for type 2 diabetes, atherosclerosis, and cardiovascular disease [7, 8, 13, 14].

In addition to the identified independent risk factors, univariable regression analysis revealed further associations, highlighting the multifaceted nature of visceral obesity and its metabolic risks. Univariable regression analysis revealed additional risk factors for VFA ≥100 cm^2, including triglycerides ≥1.69 mmol/L, ALT ≥29 U/L, uric acid ≥339.0 μmol/L, and RBC count ≥470 × 10^4/μL. This study also identified RBC ≥470 × 10^4/μL and hemoglobin ≥14.2 mg/dL as risk factors for VFA ≥100 cm^2. High hemoglobin levels have been significantly associated with the presence of non-alcoholic fatty liver disease (NAFLD) in patients with type 2 diabetes, particularly in men [30]. Hemoglobin was also significantly associated with insulin resistance [31], and free fatty acid deposition in the liver exacerbates insulin resistance in NAFLD [7]. Notably, both FBG ≥5.55 mmol/L and triglycerides ≥1.69 mmol/L align with established metabolic syndrome criteria, further validating the robustness of VFA ≥100 cm^2 as a robust marker for metabolic risk [12]. Additionally, the higher prevalence of fatty liver observed in this study (41.2%) compared to the national average of 30% [32] highlights the close association between visceral obesity, fatty liver, and metabolic dysfunction. These findings underscore the importance of addressing visceral obesity to mitigate its associated metabolic risks.

The evolving definition of MAFLD provides a more inclusive framework for diagnosing fatty liver disease, considering metabolic dysfunction alongside fatty liver, regardless of alcohol intake [15]. This framework aligns with findings in this study, where fatty liver was independently associated with VFA ≥100 cm^2. Unlike NAFLD, MAFLD emphasizes the significant role of visceral fat in metabolic dysfunction, insulin resistance, inflammation, and hepatic fibrosis, particularly in non-obese individuals with high visceral fat accumulation [33-40].

Interventions targeting visceral obesity are vital for mitigating risks associated with metabolic syndrome and MAFLD. Health promotion programs emphasizing exercise and dietary modifications have proven effective in reducing visceral fat, as evidenced in studies involving middle-aged populations [41, 42]. Exercise, in particular, has been identified as more effective than calorie



Graphical Abstract

reduction in reducing visceral fat [43]. Pharmacological options, including thiazolidinedione and sodium-glucose co-transporter 2 inhibitors, show promise for improving MAFLD outcomes in patients with type 2 diabetes [44-46].

Limitations

This study has several limitations. Its cross-sectional design precludes establishing causal relationships between visceral obesity, arterial stiffness, and MAFLD. Additionally, the small sample size limits the statistical power to identify additional risk factors and may reduce the generalizability of findings to other populations. Future longitudinal studies with larger, more diverse cohorts are necessary to validate these findings and explore potential causal pathways.

Conclusion

High baPWV, elevated FBG, large WC, fatty liver, and male sex are independent risk factors for VFA ≥100 cm^2 in middle-aged Chinese individuals. Among these, baPWV, a measure of arterial stiffness, is the most significant predictor. These findings highlight the importance of targeting visceral obesity through lifestyle modifications and medical interventions to prevent metabolic syndrome, MAFLD, and cardiovascular disease (Graphical Abstract).

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Disclosure

All authors declare no conflict of interest associated with this study.

Preprint paper disclosure

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