

Simple adiposity indices for identifying visceral adiposity and associated fatty liver and atherosclerotic plaque in a health examination cohort

Peng Lu

School of Life Sciences, Ludong University

Chao Yan

Chinese Institutes for Medical Research

Haibo Yu

Beijing Chao-Yang Hospital, Capital Medical University

Yuwei Liu

The Second Affiliated Hospital of JiaXing University, JiaXing

Jie Wang

The Second Affiliated Hospital of JiaXing University, JiaXing

Bo Yu

The First Hospital of China Medical University, Shenyang, Liaoning

Shenggang Zhao

zhaoshenggang@zjxu.edu.cn

The Second Affiliated Hospital of JiaXing University, JiaXing

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Abstract

Visceral adipose tissue (VAT) is strongly linked to cardiometabolic risk, yet direct measurement is not routinely feasible in health examination settings. The comparative performance of routine anthropometric indices, triglyceride-glucose (TyG)-derived metrics, and formula-based adiposity indices for identifying VAT, as well as their clinical relevance to fatty liver and atherosclerotic plaque, remains incompletely defined.

We analysed 821 adults who underwent routine health examinations. Spearman correlation analysis was used to compare anthropometric, metabolic and TyG-derived variables with measured VAT. Receiver operating characteristic analysis was used to compare lipid accumulation product (LAP), visceral adiposity index (VAI), a body shape index (ABSI), body roundness index (BRI), metabolic score for insulin resistance (METS-IR) and metabolic score for visceral fat (METS-VF) for identifying high VAT. Logistic regression was used to assess fatty liver and atherosclerotic plaque.

VAT was strongly associated with fatty liver than plaque, with stepwise prevalence increases across VAT quartiles. For fatty liver, LAP, METS-IR, METS-VF, BRI, and VAI showed good model discrimination; for plaque, BRI, ABSI, and METS-VF remained positive after age/sex adjustment, but discrimination was modest. TyG-WC, TyG-WWI, and WC best reflected measured VAT, while METS-VF, LAP, BRI, and METS-IR effectively identified high VAT, supporting preliminary risk stratification and targeted liver/vascular assessment.

Introduction

Central obesity is a major public health challenge and an important driver of cardiometabolic disease ^[1, 2]. Among adiposity phenotypes, visceral adipose tissue (VAT) is especially relevant because it reflects pathogenic fat distribution and is closely linked to insulin resistance, dyslipidaemia, glucose dysregulation, fatty liver and atherosclerotic disease ^[3–5]. Compared with subcutaneous adipose tissue, VAT has greater lipolytic and inflammatory activity and may promote free fatty acid flux, chronic low-grade inflammation, impaired insulin signalling and lipotoxicity ^[6–8]. Identifying individuals with excess VAT is therefore important for early risk stratification in health examination populations ^[9, 10].

Direct VAT assessment requires imaging or body-composition techniques, which are informative but limited by cost, accessibility and feasibility in routine health examinations ^[10–12]. By contrast, body mass index (BMI), waist circumference (WC) and waist-to-height ratio (WHtR) are simple and readily available ^[12–15]. However, BMI cannot distinguish visceral from subcutaneous fat and is an imperfect surrogate for VAT. The triglyceride-glucose (TyG) index and TyG-derived metrics may offer additional information because they combine anthropometric and metabolic signals ^[16–18]. Their ability to reflect measured VAT, however, varies across cohorts ^[19–21].

Formula-based adiposity indices have also been developed, including lipid accumulation product (LAP), visceral adiposity index (VAI), a body shape index (ABSI), body roundness index (BRI), metabolic score for insulin resistance (METS-IR) and metabolic score for visceral fat (METS-VF) ^[22–27]. These indices are appealing because they integrate body shape, lipid metabolism and insulin-resistance-related information ^[22–25]. Yet their comparative performance for identifying excess VAT in real-world health examination cohorts remains uncertain ^[26–28].

Excess visceral adiposity is not only a body-composition phenotype but also a potential upstream contributor to organ injury ^[3, 10]. Fatty liver is a common hepatic manifestation of visceral adiposity and metabolic dysfunction, whereas atherosclerotic plaque reflects structural vascular injury and increased cardiovascular risk ^[29–37]. Previous studies have examined obesity-related indices in relation to liver or vascular disease, but fewer have evaluated routine

anthropometric and metabolic markers, formula-based indices, measured VAT and downstream outcomes within a single analytical framework [38–41].

Here, we compared routine anthropometric variables and TyG-derived metrics with measured VAT to identify practical surrogates for health examination settings. We then evaluated six formula-based adiposity indices for identifying high VAT. Finally, we examined associations of VAT with fatty liver and atherosclerotic plaque and assessed the performance of the six formula-based indices for these outcomes. This stepwise approach was designed to inform risk stratification in routine health examination practice.

Methods

Study population and data collection

We included 821 individuals who underwent routine health examinations at the Second Affiliated Hospital of Jiaxing University. Demographic data (age and sex), anthropometric measures (height, weight, BMI, WC and WHtR), laboratory indices (including TG, HDL-C and fasting blood glucose), measured VAT and derived indices were collected. Because some variables had missing values, the effective sample size differed across analyses.

Definitions and grouping

VAT area was measured by abdominal computed tomography. High VAT was defined using previously reported sex-specific thresholds for VAT measured at the L2/3 level in a Chinese population: >142 cm² in men and >115 cm² in women [28]. Fatty liver was diagnosed by abdominal ultrasonography based on standard sonographic features. Carotid atherosclerotic plaque was assessed by carotid ultrasonography and defined as plaque presence in the carotid arteries. Fatty liver and carotid atherosclerotic plaque were treated as binary outcomes and recoded as 1 for presence and 0 for absence before logistic regression analysis. For trend analyses, participants were categorised into quartiles according to the distribution of measured VAT in the overall study population.

Calculation of anthropometric and adiposity indices

Body mass index (BMI) was calculated as weight in kilograms divided by height in metres squared. Waist-to-height ratio (WHtR) was calculated as waist circumference (WC) divided by height, with both measurements expressed in the same unit. The weight-adjusted waist index (WWI) was calculated as WC in centimetres divided by the square root of body weight in kilograms.

The triglyceride-glucose (TyG) index was calculated as:

$$\text{TyG} = \ln [\text{fasting TG (mg/dL)} \times \text{fasting glucose (mg/dL)} / 2].$$

Fasting triglycerides and fasting glucose were converted to mg/dL for the calculation of TyG and TyG-derived indices. TyG-derived indices were calculated by multiplying TyG by BMI, WHtR, WC, or WWI, yielding TyG-BMI, TyG-WHtR, TyG-WC, and TyG-WWI, respectively.

The conicity index was calculated as:

$$\text{Conicity index} = \text{WC (m)} / [0.109 \times \sqrt{\{\text{weight (kg)} / \text{height (m)}\}}].$$

Because the original values of the conicity index are close to 1, values were multiplied by 1000 for tabular presentation. Accordingly, conicity index values are reported as ×1000 in the tables.

The six formula-based adiposity indices were calculated according to their original definitions. Lipid accumulation product (LAP) was calculated using sex-specific formulas: $[WC \text{ (cm)} - 65] \times TG \text{ (mmol/L)}$ for men and $[WC \text{ (cm)} - 58] \times TG \text{ (mmol/L)}$ for women. Visceral adiposity index (VAI) was calculated as $[WC/(39.68 + 1.88 \times BMI)] \times [TG/1.03] \times [1.31/HDL-C]$ in men and $[WC/(36.58 + 1.89 \times BMI)] \times [TG/0.81] \times [1.52/HDL-C]$ in women, where WC was expressed in centimetres, BMI in kg/m^2 , and TG and HDL-C in mmol/L.

A body shape index (ABSI) was calculated as:

$$ABSI = WC / [BMI^{2/3} \times height^{1/2}],$$

where WC and height were expressed in metres.

Body roundness index (BRI) was calculated as:

$$BRI = 364.2 - 365.5 \times \sqrt{1 - [(WC / 2\pi)^2 / (0.5 \times height)^2]},$$

where WC and height were expressed in the same unit.

The metabolic score for insulin resistance (METS-IR) was calculated as:

$$METS-IR = \ln [2 \times \text{fasting glucose (mg/dL)} + TG \text{ (mg/dL)}] \times BMI / \ln [HDL-C \text{ (mg/dL)}].$$

The metabolic score for visceral fat (METS-VF) was calculated as:

$$METS-VF = 4.466 + 0.011 \times [\ln(METS-IR)]^3 + 3.239 \times [\ln(WHtR)]^3 + 0.594 \times \ln(\text{age}) + 0.319 \times \text{sex},$$

where sex was coded as 1 for men and 0 for women. Unit conversions were performed when required to ensure consistency with the original formula definitions.

Statistical analysis

Continuous variables were summarised as median and interquartile range because most anthropometric and metabolic variables were not normally distributed. Categorical variables were presented as counts and percentages. High-VAT and non-high-VAT groups were compared using the Mann-Whitney U test for continuous variables and the chi-square test or Fisher's exact test for categorical variables, as appropriate.

Spearman correlation analysis was used to assess associations between VAT and anthropometric or metabolic indices. ROC analysis was performed with high VAT as the outcome. The area under the curve (AUC), optimal cutoff, sensitivity, specificity and Youden index were calculated. The optimal cutoff was determined by the maximum Youden index.

Logistic regression was used to examine associations of VAT with fatty liver and atherosclerotic plaque, with VAT entered per 10 cm^2 increase. Logistic regression was also used to compare the six formula-based adiposity indices for fatty liver and atherosclerotic plaque, with each index standardised per 1-standard-deviation increase. Model 1 was unadjusted, and Model 2 was adjusted for age and sex. For logistic regression models, AUCs were calculated from model-predicted probabilities. Because effective sample sizes varied across indices owing to missing data, AUC comparisons were interpreted descriptively. Two-sided P values <0.05 were considered statistically significant.

Results

Baseline characteristics according to high VAT status

Using sex-specific VAT thresholds, 821 participants were classified into the high-VAT group (n = 463) and the non-high-VAT group (n = 358). The high-VAT group included a higher proportion of men than the non-high-VAT group (61.3% vs 39.1%, $P < 0.001$) and was older [53.00 (43.00–61.00) vs 48.00 (37.00–58.00) years, $P < 0.001$]. Anthropometric measures were consistently higher in the high-VAT group, including weight, BMI, WC and WHtR (all $P < 0.001$). As expected, VAT was markedly higher in the high-VAT group [190.30 (156.75–236.90) vs 86.30 (66.95–107.85) cm², $P < 0.001$]. Participants with high VAT also had a more adverse metabolic profile, including higher triglycerides (TG), fasting blood glucose, TyG index, TyG-BMI, TyG-WHtR, TyG-WC, weight-adjusted waist index (WWI), TyG-WWI and conicity index, together with lower high-density lipoprotein cholesterol (HDL-C) levels (all $P < 0.001$) (Table 1).

Table 1
Baseline characteristics according to high VAT status

Variable	Total (n = 821)	Non-high VAT (n = 358)	High VAT (n = 463)	P value
Male sex, n (%)	424 (51.6)	140 (39.1)	284 (61.3)	< 0.001
Age, years	51.00 (41.00–60.00)	48.00 (37.00–58.00)	53.00 (43.00–61.00)	< 0.001
Height, cm	166.00 (160.00–172.00)	165.00 (159.00–170.00)	167.00 (161.00–172.00)	0.001
Weight, kg	66.00 (57.00–74.00)	59.00 (53.00–67.22)	70.00 (63.00–78.00)	< 0.001
BMI, kg/m ²	23.78 (21.91–26.00)	22.10 (20.58–23.57)	24.88 (23.44–27.35)	< 0.001
Waist circumference, cm	84.00 (77.00–90.00)	77.00 (72.00–82.00)	89.00 (83.00–94.00)	< 0.001
WHtR	0.51 (0.47–0.54)	0.47 (0.44–0.50)	0.53 (0.51–0.56)	< 0.001
VAT, cm ²	140.10 (93.00–197.10)	86.30 (66.95–107.85)	190.30 (156.75–236.90)	< 0.001
TG, mmol/L	1.24 (0.92–1.83)	1.02 (0.76–1.37)	1.50 (1.08–2.16)	< 0.001
HDL-C, mmol/L	1.27 (1.07–1.52)	1.42 (1.19–1.62)	1.18 (1.02–1.37)	< 0.001
Fasting blood glucose, mmol/L	4.93 (4.62–5.33)	4.82 (4.50–5.10)	5.05 (4.71–5.55)	< 0.001
TyG index	8.52 (8.20–8.93)	8.29 (7.97–8.59)	8.74 (8.39–9.13)	< 0.001
TyG-BMI	202.61 (181.93–227.16)	182.90 (167.42–199.38)	219.18 (201.03–244.99)	< 0.001
TyG-WHtR	4.35 (3.88–4.80)	3.84 (3.59–4.22)	4.67 (4.34–5.05)	< 0.001
TyG-WC	716.80 (628.44–804.33)	634.35 (594.80–687.06)	778.72 (711.58–847.06)	< 0.001
WWI	10.32 (9.89–10.78)	9.99 (9.63–10.42)	10.52 (10.14–10.91)	< 0.001
TyG-WWI	88.67 (82.06–95.95)	83.00 (77.86–88.95)	92.43 (86.65–98.99)	< 0.001
Conicity index, ×1000	1219.62 (1164.61–1273.01)	1181.09 (1125.56–1226.77)	1247.01 (1201.92–1292.97)	< 0.001
Data are presented as median (interquartile range) or number (percentage), as appropriate. High VAT was defined using sex-specific thresholds: VAT > 142 cm ² in men and > 115 cm ² in women. P values were calculated using the Mann-Whitney U test for continuous variables and the chi-square test for categorical variables.				

Associations of anthropometric and metabolic indices with VAT

VAT correlated most strongly with TyG-WC ($R = 0.6726$, $P < 0.001$), followed by TyG-WWI ($R = 0.6593$, $P < 0.001$), WC ($R = 0.6553$, $P < 0.001$), WWI ($R = 0.6443$, $P < 0.001$), TyG-WHtR ($R = 0.6310$, $P < 0.001$), TyG-BMI ($R = 0.6085$, $P < 0.001$), WHtR ($R = 0.6057$, $P < 0.001$), BMI ($R = 0.5774$, $P < 0.001$) and body weight ($R = 0.5710$, $P < 0.001$). VAT was also positively correlated with conicity index ($R = 0.4606$, $P < 0.001$), TyG index ($R = 0.3978$, $P < 0.001$), TG ($R = 0.3344$, $P < 0.001$), height ($R = 0.2443$, $P < 0.001$) and fasting blood glucose ($R = 0.1668$, $P < 0.001$). HDL-C was inversely correlated with VAT ($R = -0.3591$, $P < 0.001$) (Table 2). Fitted trends showed the strongest positive associations for TyG-WC, TyG-WWI, WC, WWI, TyG-WHtR and TyG-BMI, together with a clear inverse association with HDL-C (Fig. 1).

Table 2
Spearman correlations of VAT with anthropometric and metabolic indices

Variable	n	Spearman R	P value
TyG-WC	516	0.6726	< 0.001
WC	523	0.6553	< 0.001
TyG-WWI	497	0.6593	< 0.001
WWI	504	0.6443	< 0.001
TyG-BMI	739	0.6085	< 0.001
BMI	748	0.5774	< 0.001
TyG-WHtR	497	0.6310	< 0.001
WHtR	504	0.6057	< 0.001
Weight	748	0.5710	< 0.001
Conicity index, ×1000	504	0.4606	< 0.001
TyG	804	0.3978	< 0.001
TG	807	0.3344	< 0.001
Height	749	0.2443	< 0.001
Fasting blood glucose	805	0.1668	< 0.001
HDL-C	807	-0.3591	< 0.001
Spearman correlation coefficients were calculated using available cases for each variable. Missing values were not imputed.			

ROC analysis of six formula-based adiposity indices for identifying high VAT

Among the six formula-based adiposity indices, METS-VF showed the highest discrimination for high VAT, with an AUC of 0.837 (95% CI 0.800-0.874), an optimal cutoff of 6.352, sensitivity of 0.799 and specificity of 0.765. LAP and BRI showed comparable performance, each with an AUC of 0.827, followed closely by METS-IR (AUC 0.825). VAI showed moderate discrimination (AUC 0.733), whereas ABSI had the lowest AUC (0.642) (Table 3). Overall, METS-VF, LAP, BRI and METS-IR identified high VAT well, while VAI was moderate and ABSI weaker (Fig. 2).

Table 3
ROC analysis of six formula-based adiposity indices for identifying high VAT

Indicator	n	AUC	AUC (95% CI)	Optimal cutoff	Sensitivity	Specificity	Youden index
METS-VF	497	0.837	0.837 (0.800 to 0.874)	6.352	0.799	0.765	0.565
LAP	518	0.827	0.827 (0.791 to 0.864)	25.48	0.776	0.749	0.525
BRI	504	0.827	0.827 (0.789 to 0.864)	3.127	0.882	0.660	0.543
METS-IR	739	0.825	0.825 (0.794 to 0.856)	33.262	0.839	0.702	0.541
VAI	499	0.733	0.733 (0.689 to 0.778)	1.537	0.656	0.738	0.394
ABSI	504	0.642	0.642 (0.593 to 0.691)	0.078	0.647	0.633	0.280
High VAT was defined using sex-specific thresholds: VAT > 142 cm ² in men and > 115 cm ² in women. Optimal cutoffs were determined using the maximum Youden index.							

Associations of VAT with fatty liver and atherosclerotic plaque

Logistic regression showed that VAT was associated with both fatty liver and atherosclerotic plaque. For every 10 cm² increase in VAT, the odds ratio (OR) for fatty liver was 1.119 (95% CI 1.095–1.144, $P < 0.001$) in the unadjusted model and 1.114 (95% CI 1.087–1.141, $P < 0.001$) after adjustment for age and sex. VAT was also associated with atherosclerotic plaque, although the effect size was smaller, with an OR of 1.039 (95% CI 1.020–1.059, $P < 0.001$) in the unadjusted model and 1.032 (95% CI 1.011–1.053, $P = 0.003$) in the adjusted model (Table 4).

Table 4
Logistic regression analyses of VAT with fatty liver and atherosclerotic plaque

Outcome	n	Model 1 OR	Model 1 95% CI	Model 1 P value	Model 2 OR	Model 2 95% CI	Model 2 P value
Fatty liver	821	1.119	1.095 to 1.144	< 0.001	1.114	1.087 to 1.141	< 0.001
Atherosclerotic plaque	821	1.039	1.020 to 1.059	< 0.001	1.032	1.011 to 1.053	0.003
VAT was entered per 10 cm ² increase. Model 1 was unadjusted, and Model 2 was adjusted for age and sex.							

Predictive performance of six formula-based adiposity indices for fatty liver and plaque

For fatty liver, all six formula-based indices remained significantly associated after adjustment for age and sex. Based on Model 2 AUCs, LAP (AUC 0.840), METS-IR (AUC 0.837), METS-VF (AUC 0.820), BRI (AUC 0.797) and VAI (AUC 0.787) showed useful discrimination, whereas ABSI performed less well (AUC 0.673). Adjusted ORs per 1-standard-deviation increase were 6.416 for LAP (95% CI 4.313–9.545, $P < 0.001$), 7.766 for VAI (95% CI 4.565–13.212, $P < 0.001$), 1.539 for ABSI (95% CI 1.222–1.937, $P < 0.001$), 3.810 for BRI (95% CI 2.827–5.135, $P < 0.001$), 5.508 for METS-IR (95% CI 4.186–7.246, $P < 0.001$) and 6.927 for METS-VF (95% CI 4.635–10.354, $P < 0.001$). For

atherosclerotic plaque, BRI and ABSI were associated in unadjusted models, whereas LAP, VAI, METS-IR and METS-VF were not. After adjustment for age and sex, BRI (OR 1.512, 95% CI 1.256–1.821, $P < 0.001$; AUC 0.638), METS-VF (OR 1.310, 95% CI 1.073–1.599, $P = 0.008$; AUC 0.630) and ABSI (OR 1.264, 95% CI 1.062–1.506, $P = 0.009$; AUC 0.604) were positively associated with plaque. METS-IR yielded a modest adjusted AUC (0.618) but was not independently associated after adjustment ($P = 0.073$), and LAP and VAI were also not independently associated. Thus, the indices were more informative for fatty liver than for atherosclerotic plaque, although BRI, ABSI and METS-VF provided modest plaque-related risk signals (Table 5).

Table 5

Logistic regression comparison of six adiposity indices for predicting fatty liver and atherosclerotic plaque

Outcome	Indicator	n	Model 1 OR	Model 1 95% CI	Model 1 P	Model 1 AUC	Model 2 OR	Model 2 95% CI	Model 2 P	Model 2 AUC
Fatty liver	LAP	518	6.203	4.285 to 8.981	< 0.001	0.830	6.416	4.313 to 9.545	< 0.001	0.840
Fatty liver	VAI	499	7.604	4.510 to 12.819	< 0.001	0.773	7.766	4.565 to 13.212	< 0.001	0.787
Fatty liver	ABSI	504	1.514	1.253 to 1.830	< 0.001	0.627	1.539	1.222 to 1.937	< 0.001	0.673
Fatty liver	BRI	504	3.369	2.578 to 4.402	< 0.001	0.773	3.810	2.827 to 5.135	< 0.001	0.797
Fatty liver	METS-IR	740	5.439	4.193 to 7.055	< 0.001	0.836	5.508	4.186 to 7.246	< 0.001	0.837
Fatty liver	METS-VF	497	3.756	2.817 to 5.009	< 0.001	0.770	6.927	4.635 to 10.354	< 0.001	0.820
Atherosclerotic plaque	LAP	518	1.068	0.895 to 1.276	0.465	0.504	1.026	0.857 to 1.229	0.781	0.614
Atherosclerotic plaque	VAI	499	0.957	0.799 to 1.145	0.628	0.504	0.955	0.785 to 1.161	0.641	0.610
Atherosclerotic plaque	ABSI	504	1.308	1.102 to 1.552	0.002	0.592	1.264	1.062 to 1.506	0.009	0.604
Atherosclerotic plaque	BRI	504	1.569	1.310 to 1.879	< 0.001	0.635	1.512	1.256 to 1.821	< 0.001	0.638
Atherosclerotic plaque	METS-IR	740	0.917	0.793 to 1.060	0.242	0.525	0.872	0.750 to 1.013	0.073	0.618
Atherosclerotic plaque	METS-VF	497	1.105	0.923 to 1.321	0.276	0.525	1.310	1.073 to 1.599	0.008	0.630
Model 1 was unadjusted, and Model 2 was adjusted for age and sex. Each adiposity index was entered per 1-standard-deviation increase. AUCs were calculated from model-predicted probabilities.										

Trends in fatty liver and plaque prevalence across VAT quartiles

When participants were stratified by VAT quartiles, fatty liver prevalence increased progressively from 13.6% in Q1 to 31.7% in Q2, 47.3% in Q3 and 63.9% in Q4. Atherosclerotic plaque prevalence also increased across VAT quartiles, from 44.7% in Q1 to 48.8% in Q2, 57.6% in Q3 and 72.7% in Q4 (Fig. 3). Thus, higher VAT burden was accompanied by a greater prevalence of both fatty liver and atherosclerotic plaque, with a stronger gradient for fatty liver.

Discussion

Principal findings

This study evaluated practical approaches for identifying visceral adiposity and related organ risk in a routine health examination setting. Four findings emerged. First, TyG-WC, TyG-WWI, WC, WWI, TyG-WHtR and TyG-BMI were the routine and TyG-derived variables most closely associated with measured VAT. Second, METS-VF, LAP, BRI and METS-IR showed good discrimination for high VAT, whereas VAI was moderate and ABSI weaker. Third, VAT was associated with fatty liver and atherosclerotic plaque after adjustment for age and sex, and both outcomes increased across VAT quartiles. The association was stronger for fatty liver than for plaque. Fourth, formula-based indices performed more consistently for fatty liver than for plaque. For plaque, BRI, ABSI and METS-VF were positively associated after adjustment, but their AUCs were modest, supporting a cautious exploratory interpretation.

Why some indices better reflect VAT

VAT is a more informative marker of cardiometabolic risk than general obesity, but direct measurement remains difficult in routine practice. BMI is widely used to define obesity, yet it cannot distinguish fat from lean mass or visceral from subcutaneous fat. Consistent with this limitation, BMI was associated with VAT but was not the strongest surrogate ^[1–5]. WC directly captures abdominal fat accumulation and remained one of the strongest anthropometric correlates of measured VAT. Correlations strengthened when WC or related body-shape measures were combined with the TyG index, as shown by the leading performance of TyG-WC and TyG-WWI. TyG-derived metrics integrate anthropometric and metabolic information and may better reflect insulin resistance and metabolic dysfunction ^[12–16]. In this cohort, TyG-WC, TyG-WWI, TyG-WHtR and TyG-BMI were all closely related to VAT, suggesting that combining body-size or body-shape information with metabolic information may improve VAT identification. Among formula-based indices, METS-VF, LAP, BRI and METS-IR showed similarly good high-VAT discrimination. This pattern is biologically plausible because these indices capture complementary dimensions of visceral adiposity, including central body geometry, lipid accumulation, insulin-resistance-related metabolism and, for METS-VF, age and sex. The comparable AUCs suggest that no single formula-based index clearly dominated in this cohort; the most practical choice may depend on which variables are available in a given health examination workflow ^[25–28,39–40].

VAT and downstream clinical outcomes

VAT was strongly associated with fatty liver, and fatty liver prevalence increased markedly across VAT quartiles ^[3, 10]. This association is biologically plausible because VAT is an important source of free fatty acids and pro-inflammatory mediators. Excess VAT may increase portal free fatty acid delivery, aggravate insulin resistance, promote hepatic lipid deposition and amplify metabolic inflammation, thereby contributing to fatty liver development and progression ^[29–34]. VAT also remained associated with atherosclerotic plaque after adjustment for age and sex, and plaque prevalence increased from the lowest to the highest VAT quartile. This finding suggests that visceral fat accumulation may be linked to structural vascular injury, but the magnitude of the VAT-plaque association was smaller than that observed for fatty liver. The plaque association should therefore be interpreted cautiously. Atherosclerotic plaque formation is long term and multifactorial, with contributions from age, blood pressure, lipid profile, inflammation, smoking, glycaemic status and genetic susceptibility ^[39–41]. In addition, plaque was recorded as a binary variable rather than as plaque number, plaque burden or another quantitative vascular phenotype. The present study therefore could not assess the dose-response relationship between VAT and plaque severity ^[37, 38, 42].

Implications for health examination-based screening

These findings may support risk stratification in routine health examination settings. In clinical practice, decisions about additional liver imaging or vascular assessment are often empirical [4, 10, 17–18]. Simple examination-based indices may help identify individuals who are more likely to have excess VAT and fatty liver [25, 28, 29, 43, 44]. For VAT screening, TyG-WC, TyG-WWI and WC were closely aligned with measured VAT, whereas METS-VF, LAP, BRI and METS-IR showed good high-VAT discrimination. For fatty liver, LAP, METS-IR, METS-VF, BRI and VAI showed useful model-based performance. The association between VAT and plaque, together with the increase in plaque prevalence across VAT quartiles, suggests that individuals with high visceral fat burden may warrant closer vascular risk assessment. However, formula-based plaque models had only modest AUCs. Although BRI, ABSI and METS-VF remained positively associated with plaque after age and sex adjustment, their discrimination was insufficient for stand-alone vascular screening. These indices should therefore be viewed as preliminary screening or risk-alert tools, not as substitutes for standardised vascular imaging [42, 43].

Strengths and limitations

This study has several strengths. Measured VAT, rather than an estimated surrogate, was used as the reference standard, providing a stronger basis for comparing alternative indices. The study also evaluated routine anthropometric and TyG-derived variables before comparing formula-based indices, creating a stepwise framework from basic examination measures to VAT and clinically relevant outcomes. This structure improves the practical relevance of the findings for health examination workflows. Several limitations should be acknowledged. The cross-sectional design precludes causal inference. Missing data led to small differences in sample size across analyses. Plaque was recorded as a binary rather than quantitative variable, which may have underestimated its relationship with VAT. The study population came from a specific examination cohort, so external validity should be tested in broader and more diverse cohorts. Finally, the performance of formula-based indices may vary by ethnicity, population structure, measurement units and disease prevalence.

Conclusions

Among routine and TyG-derived examination variables, TyG-WC, TyG-WWI and WC most closely reflected measured VAT. Among the six formula-based adiposity indices, METS-VF, LAP, BRI and METS-IR identified high VAT with good discrimination. VAT was strongly associated with fatty liver and remained associated with atherosclerotic plaque after adjustment for age and sex, and both outcomes increased across VAT quartiles. Formula-based indices performed more consistently for fatty liver than for plaque, with LAP, METS-IR, METS-VF, BRI and VAI showing useful fatty liver discrimination. For plaque, BRI, ABSI and METS-VF showed statistically significant but modest adjusted signals, whereas LAP, VAI and METS-IR were not independently associated. These findings support the use of simple health examination-based adiposity indices for preliminary identification of excess visceral fat and for guiding further evaluation of fatty liver and vascular risk. Their utility for vascular screening should be validated using quantitative vascular phenotypes.

Abbreviations

VAT, visceral adipose tissue; WC, waist circumference; BMI, body mass index; WHtR, waist-to-height ratio; WHR, waist-to-hip ratio; BAI, body adiposity index; WWI, weight-adjusted-waist index; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; TyG, triglyceride-glucose index; TyG-BMI, triglyceride-glucose index combined with body mass index; TyG-WHtR, triglyceride-glucose index combined with waist-to-height ratio; TyG-WC, triglyceride-glucose index combined with waist circumference; TyG-WWI, triglyceride-glucose index combined with weight-adjusted-waist index; LAP, lipid accumulation product; VAI, visceral adiposity index; ABSI, a body shape index; BRI, body roundness index;

METS-IR, metabolic score for insulin resistance; METS-VF, metabolic score for visceral fat; ROC, receiver operating characteristic; AUC, area under the curve; OR, odds ratio; CI, confidence interval; SD, standard deviation; IQR, interquartile range.

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Second Affiliated Hospital of Jiaxing University (approval number: 2026-114). The study was conducted in accordance with the principles of the Declaration of Helsinki. All participants provided written informed consent to participate before inclusion in the study.

Consent for publication

Not Applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are not publicly available because they contain health examination data and participant privacy could be compromised, but de-identified data may be available from the corresponding author on reasonable request and subject to institutional approval and applicable privacy restrictions. Derived data supporting the conclusions of this article are included within the article and its tables.

Competing interests

The authors declare no competing interests.

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Authors' contributions

Shenggang Zhao, Jie Wang and Bo Yu contributed to Conceptualization and Supervision. Peng Lu and Chao Yan contributed to Writing – original draft and Data curation. Peng Lu, Chao Yan, Haibo Yu, and Yuwei Liu contributed to Formal analysis, Visualization, and preparation of tables and figures. All authors contributed to Writing – review & editing and approved the final version of the manuscript.

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References

1. Shuster, A., Patlas, M., Pinthus, J. H. & Mourtzakis, M. The clinical importance of visceral adiposity: a critical review of methods for visceral adipose tissue analysis. *Br. J. Radiol.* **85** (1009), 1–10. 10.1259/bjr/38447238

- (2012).
2. Després, J. P. & Lemieux, I. Abdominal obesity and metabolic syndrome. *Nature* **444** (7121), 881–887. 10.1038/nature05488 (2006).
 3. Després, J. P. Abdominal obesity and cardiovascular disease: is inflammation the missing link? *Can. J. Cardiol.* **28** (6), 642–652. 10.1016/j.cjca.2012.06.004 (2012).
 4. Ross, R. et al. Waist circumference as a vital sign in clinical practice: a consensus statement from the IAS and ICCR Working Group on Visceral Obesity. *Nat. Rev. Endocrinol.* **16** (3), 177–189. 10.1038/s41574-019-0310-7 (2020).
 5. Fox, C. S. et al. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. *Circulation* **116** (1), 39–48. 10.1161/CIRCULATIONAHA.106.675355 (2007).
 6. Pou, K. M. et al. Patterns of abdominal fat distribution: the Framingham Heart Study. *Diabetes Care.* **32** (3), 481–485. 10.2337/dc08-1359 (2009).
 7. Liu, J. et al. Impact of abdominal visceral and subcutaneous adipose tissue on cardiometabolic risk factors: the Jackson Heart Study. *J. Clin. Endocrinol. Metab.* **95** (12), 5419–5426. 10.1210/jc.2010-1378 (2010).
 8. Foster, M. C. et al. Association of subcutaneous and visceral adiposity with albuminuria: the Framingham Heart Study. *Obes. (Silver Spring).* **19** (6), 1284–1289. 10.1038/oby.2010.308 (2011).
 9. Rosenquist, K. J. et al. Visceral and subcutaneous fat quality and cardiometabolic risk. *JACC Cardiovasc. Imaging.* **6** (7), 762–771. 10.1016/j.jcmg.2012.11.021 (2013).
 10. Neeland, I. J. et al. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol.* **7** (9), 715–725. 10.1016/S2213-8587(19)30084-1 (2019).
 11. Powell-Wiley, T. M. et al. Obesity and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation* **143** (21), e984–e1010. 10.1161/CIR.0000000000000973 (2021).
 12. Kershaw, E. E. & Flier, J. S. Adipose tissue as an endocrine organ. *J. Clin. Endocrinol. Metab.* **89** (6), 2548–2556. 10.1210/jc.2004-0395 (2004).
 13. Ouchi, N., Parker, J. L., Lugus, J. J. & Walsh, K. Adipokines in inflammation and metabolic disease. *Nat. Rev. Immunol.* **11** (2), 85–97. 10.1038/nri2921 (2011).
 14. Gregor, M. F. & Hotamisligil, G. S. Inflammatory mechanisms in obesity. *Annu. Rev. Immunol.* **29**, 415–445. 10.1146/annurev-immunol-031210-101322 (2011).
 15. Ibrahim, M. M. Subcutaneous and visceral adipose tissue: structural and functional differences. *Obes. Rev.* **11** (1), 11–18. 10.1111/j.1467-789X.2009.00623.x (2010).
 16. Simental-Mendía, L. E., Rodríguez-Morán, M. & Guerrero-Romero, F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. *Metab. Syndr. Relat. Disord.* **6** (4), 299–304. 10.1089/met.2008.0034 (2008).
 17. Er, L. K. et al. Triglyceride Glucose-Body Mass Index Is a Simple and Clinically Useful Surrogate Marker for Insulin Resistance in Nondiabetic Individuals. *PLoS One.* **11** (3), e0149731. 10.1371/journal.pone.0149731 (2016).
 18. Yang, Q. et al. Serum triglyceride glucose index is a valuable predictor for visceral obesity in patients with type 2 diabetes: a cross-sectional study. *Cardiovasc. Diabetol.* **22** (1), 98. 10.1186/s12933-023-01834-3 (2023).
 19. Conus, F., Rabasa-Lhoret, R. & Peronnet, F. Characteristics of metabolically obese normal-weight (MONW) subjects. *Appl. Physiol. Nutr. Metab.* **32** (1), 4–12. 10.1139/H06-092 (2007).
 20. Ding, C., Chan, Z. & Magkos, F. Lean, but not healthy: the 'metabolically obese, normal-weight' phenotype. *Curr. Opin. Clin. Nutr. Metab. Care.* **19** (6), 408–417. 10.1097/MCO.0000000000000317 (2016).

21. Zeng, Q. et al. CT-derived abdominal adiposity: Distributions and better predictive ability than BMI in a nationwide study of 59,429 adults in China. *Metabolism* **115**, 154456. 10.1016/j.metabol.2020.154456 (2021).
22. Kahn, H. S. The lipid accumulation product performs better than the body mass index for recognizing cardiovascular risk: a population-based comparison. *BMC Cardiovasc. Disord.* **5**, 26. 10.1186/1471-2261-5-26 (2005).
23. Amato, M. C. et al. Visceral Adiposity Index: a reliable indicator of visceral fat function associated with cardiometabolic risk. *Diabetes Care.* **33** (4), 920–922. 10.2337/dc09-1825 (2010).
24. Krakauer, N. Y. & Krakauer, J. C. A new body shape index predicts mortality hazard independently of body mass index. *PLoS One.* **7** (7), e39504. 10.1371/journal.pone.0039504 (2012).
25. Thomas, D. M. et al. Relationships between body roundness with body fat and visceral adipose tissue emerging from a new geometrical model. *Obes. (Silver Spring).* **21** (11), 2264–2271. 10.1002/oby.20408 (2013).
26. Bello-Chavolla, O. Y. et al. METS-IR, a novel score to evaluate insulin sensitivity, is predictive of visceral adiposity and incident type 2 diabetes. *Eur. J. Endocrinol.* **178** (5), 533–544. 10.1530/EJE-17-0883 (2018).
27. Bello-Chavolla, O. Y. et al. Metabolic Score for Visceral Fat (METS-VF), a novel estimator of intra-abdominal fat content and cardio-metabolic health. *Clin. Nutr.* **39** (5), 1613–1621. 10.1016/j.clnu.2019.07.012 (2020).
28. Huo, L. et al. Optimal cut-points of visceral adipose tissue areas for cardiometabolic risk factors in a Chinese population: a cross-sectional study. *Diabet. Med.* **36** (10), 1268–1275. 10.1111/dme.14060 (2019).
29. Hanlon, C. L. & Yuan, L. Nonalcoholic Fatty Liver Disease: The Role of Visceral Adipose Tissue. *Clin. Liver Dis. (Hoboken).* **19** (3), 106–110. 10.1002/cld.1183 (2022).
30. Younossi, Z. M. et al. Global epidemiology of nonalcoholic fatty liver disease—Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology* **64** (1), 73–84. 10.1002/hep.28431 (2016).
31. Rinella, M. E. et al. AASLD Practice Guidance on the clinical assessment and management of nonalcoholic fatty liver disease. *Hepatology* **77** (5), 1797–1835. 10.1097/HEP.0000000000000323 (2023).
32. Speliotes, E. K. et al. Fatty liver is associated with dyslipidemia and dysglycemia independent of visceral fat: the Framingham Heart Study. *Hepatology* **51** (6), 1979–1987. 10.1002/hep.23593 (2010).
33. Liu, J. et al. Fatty liver, abdominal visceral fat, and cardiometabolic risk factors: the Jackson Heart Study. *Arterioscler. Thromb. Vasc Biol.* **31** (11), 2715–2722. 10.1161/ATVBAHA.111.234062 (2011).
34. Liu, J. et al. Fatty liver, abdominal adipose tissue and atherosclerotic calcification in African Americans: the Jackson Heart Study. *Atherosclerosis* **224** (2), 521–525. 10.1016/j.atherosclerosis.2012.07.042 (2012).
35. Ohman, M. K., Wright, A. P., Wickenheiser, K. J., Luo, W. & Eitzman, D. T. Visceral adipose tissue and atherosclerosis. *Curr. Vasc Pharmacol.* **7** (2), 169–179. 10.2174/157016109787455680 (2009).
36. Öhman, M. K. et al. Perivascular visceral adipose tissue induces atherosclerosis in apolipoprotein E deficient mice. *Atherosclerosis* **219** (1), 33–39. 10.1016/j.atherosclerosis.2011.07.012 (2011).
37. Tanaka, T. et al. Impact of abdominal fat distribution, visceral fat, and subcutaneous fat on coronary plaque scores assessed by 320-row computed tomography coronary angiography. *Atherosclerosis* **287**, 155–161. 10.1016/j.atherosclerosis.2019.06.910 (2019).
38. Yu, Y. et al. Visceral adiposity index and cervical arterial atherosclerosis in northeast China: a population based cross-sectional survey. *Eur. J. Neurol.* **28** (1), 161–171. 10.1111/ene.14513 (2021).
39. Strasser, B. et al. Abdominal obesity is associated with arterial stiffness in middle-aged adults. *Nutr. Metab. Cardiovasc. Dis.* **25** (5), 495–502. 10.1016/j.numecd.2015.01.002 (2015).
40. Liu, K. H. et al. Sonographic measurement of mesenteric fat thickness is a good correlate with cardiovascular risk factors: comparison with subcutaneous and preperitoneal fat thickness, magnetic resonance imaging and

- anthropometric indexes. *Int. J. Obes. Relat. Metab. Disord.* **27** (10), 1267–1273. 10.1038/sj.ijo.0802393 (2003).
41. Xu, C. et al. Association between organ damage and visceral adiposity index in community-dwelling elderly Chinese population: the Northern Shanghai Study. *Aging Clin. Exp. Res.* **33** (8), 2291–2297. 10.1007/s40520-020-01752-4. =38744 (2021).
 42. Sun, J. et al. Higher visceral adiposity index and lipid accumulation product in relation to increased risk of atherosclerotic burden in community-dwelling older adults. *Exp. Gerontol.* **174**, 112115. 10.1016/j.exger.2023.112115 (2023).
 43. Motamed, N. et al. Body Roundness Index and Waist-to-Height Ratio Are Strongly Associated With Non-Alcoholic Fatty Liver Disease: A Population-Based Study. *Hepat. Mon.* **16** (9), e39575. 10.5812/hepatmon.39575 (2016).
 44. Jiang, N., Zhang, S., Chu, J., Yang, N. & Lu, M. Association between body roundness index and non-alcoholic fatty liver disease detected by Fibroscan in America. *J. Clin. Lab. Anal.* **37** (10), e24973. 10.1002/jcla.24973 (2023).

Figures

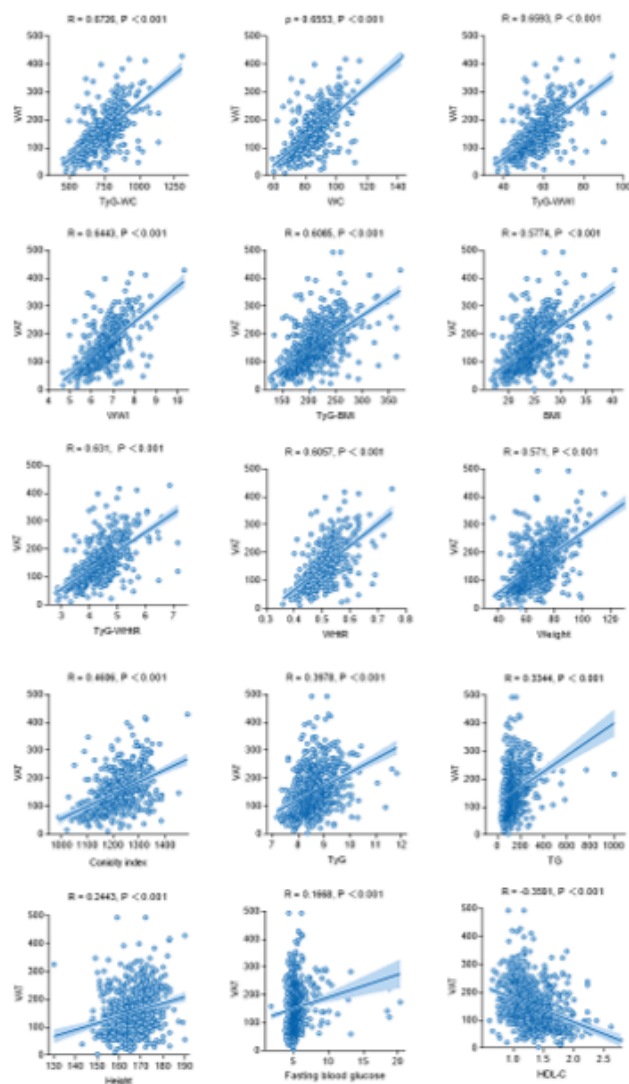


Figure 1

Associations between VAT and anthropometric or metabolic indices. Scatter plots and fitted trends are shown for height, weight, WHtR, BMI, TG, TyG, TyG-BMI, TyG-WHtR, WC, TyG-WC, conicity index, fasting blood glucose, HDL-C, WWI and TyG-WWI. Each point represents one participant, and the solid line indicates the fitted linear trend. Panel titles show Spearman correlation coefficients (R). TyG-WC, TyG-WWI, WC, WWI, TyG-WHtR, TyG-BMI and WHtR showed relatively strong positive correlations with VAT, whereas HDL-C showed an inverse correlation.

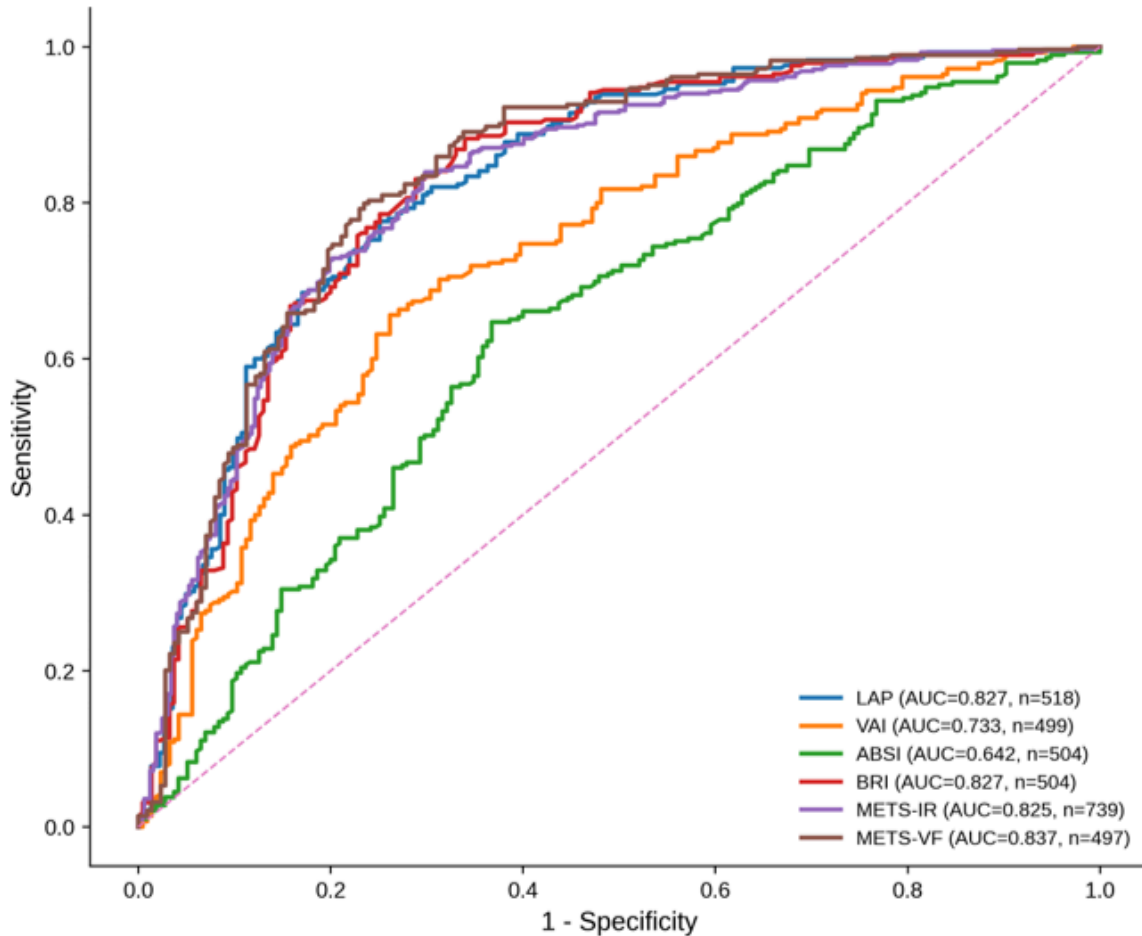


Figure 2

ROC curves of six formula-based adiposity indices for identifying high VAT. Curves are shown for LAP, VAI, ABSI, BRI, METS-IR and METS-VF. High VAT was defined using sex-specific cutoffs (VAT >142 cm² in men and >115 cm² in women). Discrimination was evaluated by the area under the curve (AUC). METS-VF showed the highest AUC, whereas LAP, BRI and METS-IR showed similarly good performance. VAI showed moderate discrimination, and ABSI showed weaker discrimination.

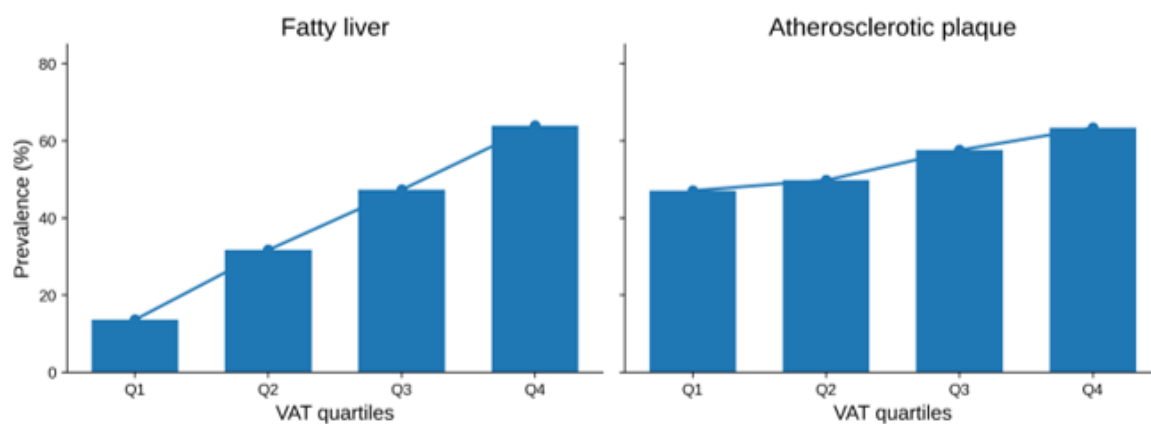


Figure 3

Prevalence of fatty liver and atherosclerotic plaque across VAT quartiles. Participants were stratified into four groups (Q1-Q4) according to VAT quartile. Fatty liver prevalence increased progressively with higher VAT quartiles. Atherosclerotic plaque prevalence also increased across quartiles, indicating that greater VAT burden was associated with a higher prevalence of both hepatic and vascular outcomes.

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