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Association of obesity- and insulin resistance-related indices with subclinical carotid atherosclerosis in type 1 diabetes: a cross-sectional study

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Abstract

Background Obesity and insulin resistance are well-established risk factors for atherosclerosis and cardiovascular disease (CVD). Although some obesity- and insulin resistance-related indices (OIRIs) have been linked to CVD, their associations with subclinical carotid atherosclerosis (SCA) in individuals with type 1 diabetes (T1D) remain unclear. This study aims to systematically explore and compare the associations of various common OIRIs with SCA in T1D population.

Methods A total of 418 adult inpatients with classic T1D admitted from October 2008 to June 2021 to the First Affiliated Hospital of Air Force Medical University in Xi'an, China were included in this study. Demographic, anthropometric, and laboratory data were collected. Studied OIRIs comprised body mass index, waist-to-height ratio, waist-to-hip ratio (WHR), a body shape index, abdominal volume index, body adiposity index, body roundness index, conicity index, triglyceride-glucose index, visceral adiposity index, Chinese visceral adiposity index (CVAI), lipid accumulation product, estimated glucose disposal rate (eGDR), triglyceride-to-HDL ratio, and cardiometabolic index. Binary logistic regression, restricted cubic spline (RCS), and receiver operating characteristic curves were used to examine the associations of these indices with SCA.

Results In multivariable logistic regression analyses, after adjusting for potential confounders, per 1.0-standard deviation (SD) increase in CVAI (OR, 95% CI: 1.68, 1.16–2.47), $eGDR_{WHR}$ (eGDR calculated with WHR; OR, 95% CI: 0.44, 0.22–0.82), and $eGDR_{WC}$ (eGDR calculated with waist circumference; OR, 95% CI: 0.49, 0.24–0.93) were significantly associated with SCA. CVAI exhibited the highest area under the curve (AUC) in diagnosing SCA, with a value of 0.73 (95% CI: 0.69–0.77). RCS analyses indicated a linear and positive association between CVAI and SCA in the overall

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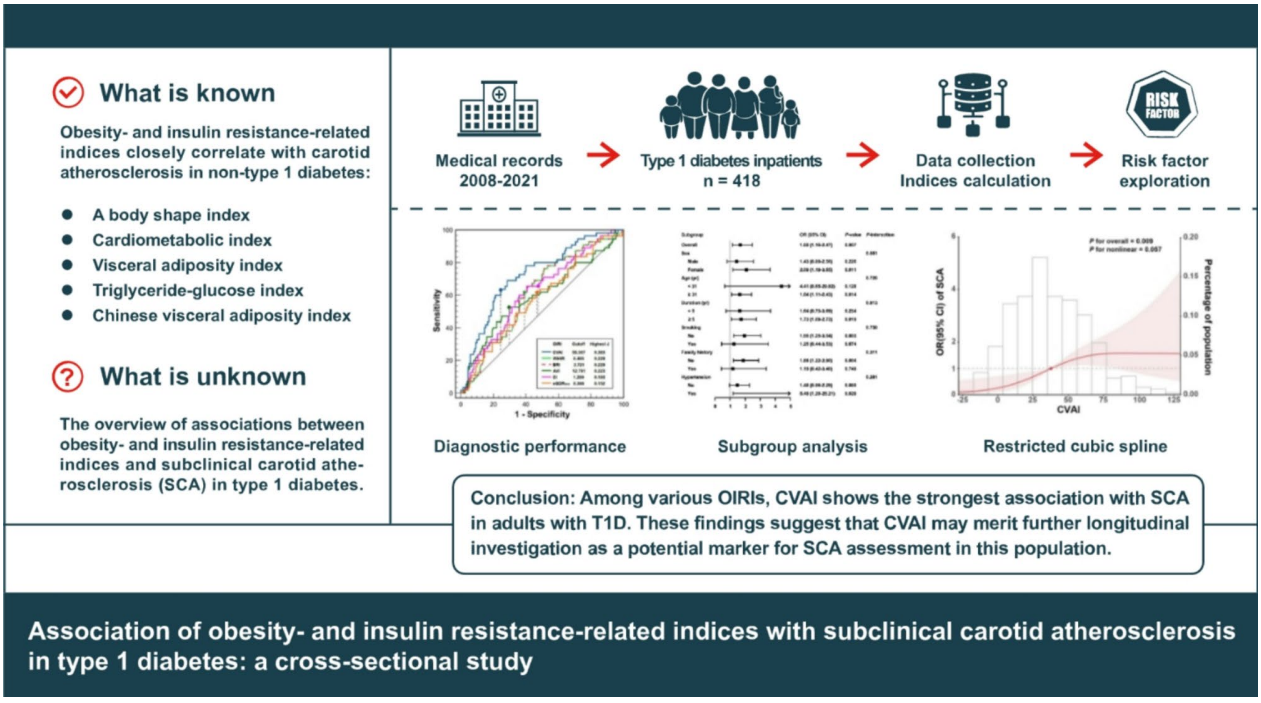
population and the females. Subgroup analyses and sensitivity analyses further supported the association between CVAI and SCA. Additionally, adding CVAI to the Steno Type 1 Risk Engine (ST1RE) improved the reclassification, but did not enhance the overall discriminative ability of ST1RE to identify SCA.

Conclusion Among various OIRIs, CVAI shows the strongest association with SCA in adults with T1D. These findings suggest that CVAI may merit further longitudinal investigation as a potential marker for SCA assessment in this population.

Keywords Chinese visceral adiposity index, Subclinical atherosclerosis, Obesity, Insulin resistance, Type 1 diabetes

Graphical abstract



Introduction

Type 1 diabetes (T1D) affects millions of individuals worldwide and poses a significant health burden, particularly due to its early onset and rapid progression of complications, including cardiovascular disease (CVD) [1, 2]. Despite advances in prediction and treatment, the mortality risk for T1D patients remains two to eight times higher than that of the general population [3, 4]. This elevated risk is primarily attributed to CVD [5, 6], and it increases with an earlier diagnosis of T1D [7, 8]. Growing evidence has also confirmed that T1D carries a higher risk of atherosclerosis [9–12]. A recent cohort study reported an independent association between subclinical atherosclerosis and overall mortality, as well as major cardiovascular events in T1D patients [13]. Therefore, identifying subclinical carotid atherosclerosis (SCA), which represents the early stage of atherosclerosis, holds significant clinical implications for the T1D population.

In recent years, obesity and insulin resistance in T1D have increasingly garnered attention among researchers [14–17]. As the cornerstone treatment for T1D, intensive insulin therapy can lead to obesity and insulin resistance [18, 19], both of which play crucial roles in the initiation, progression, and adverse outcomes of atherosclerosis [20, 21]. Given the inherent limitations of traditional methods for assessing obesity and insulin resistance, novel obesity- and insulin resistance-related indices (OIRIs) have been developed as reliable surrogates and examined for their relationship with atherosclerosis and CVD [22–25]. Existing studies have demonstrated that several OIRIs, including a body shape index (ABSI) [26], cardiometabolic index (CMI) [27], triglyceride-glucose index (TyG) [28, 29], visceral adiposity index (VAI) [30], and Chinese visceral adiposity index (CVAI) [31–33], are correlated with carotid atherosclerosis in the general population and patients with arterial hypertension or peripheral arterial disease. However, in T1D population, the associations between OIRIs and SCA have not been systematically investigated. To date, only eGDR has been specifically examined for the association with SCA, and

no studies have comprehensively evaluated the strength of associations between multiple OIRIs and SCA in T1D population.

This study aims to systematically evaluate various common OIRIs for their associations with SCA in adults with T1D and explore which OIRI shows the strongest correlation with SCA in this population. This cross-sectional analysis contributes to the understanding of relationship between OIRIs and SCA in T1D, potentially informing future longitudinal studies on SCA risk assessment in this population.

Methods

Study design and participants

This was a cross-sectional study. A total of 1 269 medical hospitalization records of classic T1D patients from October 2008 to June 2021 were initially reviewed. The hospitalization was for routine chronic complication screening and better control of blood glucose and chronic complications. The inclusion criteria were as follows: (1) classic T1D; (2) aged ≥ 18 years at enrollment; (3) the record of patients with one visit or the latest record of those with multiple visits. Criteria for classic T1D included the occurrence of diabetic ketosis or ketoacidosis following the onset of classic hyperglycemic symptoms (e.g., polyuria, thirst, polydipsia, and weight loss), the dependence on continuous insulin treatment, and positivity for glutamic acid decarboxylase autoantibodies (GADA), protein tyrosine phosphatase-like insulinoma-associated 2 autoantibodies (IA-2A), insulin autoantibodies (IAA; only in insulin-naïve patients), or zinc transporter 8 autoantibodies (ZnT8A). The exclusion criteria were: (1) latent autoimmune diabetes in adults; (2) type 2 diabetes; (3) specific types of diabetes; (4) gestational diabetes or preexisting diabetes in pregnancy; (5) diagnosed with overt CVD (coronary artery disease, myocardial infarction, heart failure, arrhythmias, stroke, or peripheral artery disease); (6) incomplete records with missing data. Ultimately, the records of 418 adult participants with classic T1D were included in this study (Fig. 1). This study adhered to the Declaration of Helsinki and was approved by the Ethics Review Committee of the First Affiliated Hospital of Air Force Medical University (Approval No. KY20240001301). Informed consent was obtained from all study participants.

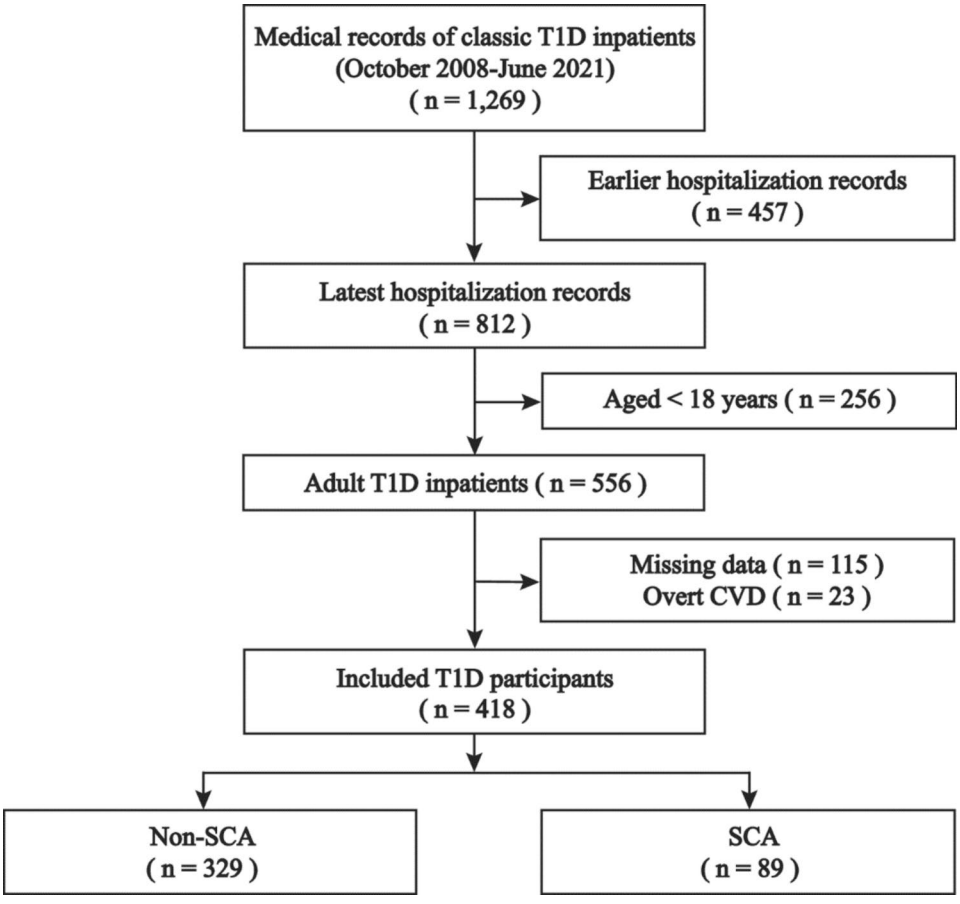


Fig. 1 The flowchart of study participants

Data collection and definitions

Data collection was performed by well-trained clinical endocrinologists. The data incorporated demographic information (age, sex, diabetes duration, smoking, alcohol drinking, family history of any diabetes), anthropometric measurements [systolic blood pressure (SBP), diastolic blood pressure (DBP), height, weight, waist circumference (WC), hip circumference (HC)], biochemical indicators [high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), triglyceride (TG), total cholesterol (TC), fasting plasma glucose (FPG), glycated hemoglobin (HbA1c), serum uric acid (SUA), alanine aminotransferase (ALT), creatinine], and medication history [metformin, angiotensin-converting enzyme inhibitor (ACEI), angiotensin II receptor blocker (ARB), calcium channel blocker (CCB), beta-adrenergic receptor blocker (BRB), statins, antiplatelet agents].

Height and weight were measured using a calibrated digital scale, with participants wearing light clothing and no shoes. Blood pressure was measured while seated after resting for at least 5 min. Three readings were taken, and the average was used for analysis. WC was measured at the level of the iliac crest, with the participant standing and relaxed, using a non-stretchable measuring tape. HC was measured at the widest point of the hips, using the same tape. Smoking and alcohol drinking states were described as current smoking and current alcohol consumption, respectively. Peripheral venous blood samples were obtained after an overnight fasting of at least 8 h to test biochemical indicators with standardized assays. Hypertension was defined as SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg, or taking antihypertensive medication. Overweight and obesity were defined as BMI ≥ 24.0 kg/m^2 and ≥ 28.0 kg/m^2, respectively, according to the Expert Consensus on Obesity Prevention and Treatment in China [34].

Calculation of OIRIs

The OIRIs included in this study were calculated using the following formulas:

$$\text{BMI (body mass index)} = \text{weight (kg)} / \text{height}^2 \text{ (m)}$$

$$\text{WHtR (waist-to-height ratio)} = \text{WC (cm)} / \text{height (cm)}$$

$$\text{WHR (waist-to-hip ratio)} = \text{WC (cm)} / \text{HC (cm)}$$

$$\text{ABSI (a body shape index)} = \text{WC (m)} / \left[\text{BMI}^{2/3}\left(\text{kg/m}^2\right) \times \text{height}^{1/2}\text{ (m)}\right]$$

$$\text{AVI (abdominal volume index)} = \left[2 \times \text{WC}^2\text{ (cm)} + 0.7 \times (\text{WC} - \text{HC})^2\text{ (cm)}\right] / 1000$$

$$\text{BAI (body adiposity index)} = \text{HC (cm)} / \text{height}^{3/2}\text{ (m)} - 18$$

$$\text{BRI (body roundness index)} = 364.2 - 365.5\left[1 - \pi^{-2}\text{WC}^2\text{ (m)}\,\text{height}^{-2}\text{ (m)}\right]^{1/2}$$

$$\text{CI (conicity index)} = 0.109^{-1}\text{WC (m)}\left[\text{weight (kg)} / \text{height (m)}\right]^{-1/2}$$

$$\text{TyG (triglyceride-glucose index)} = \text{Ln}[\text{TG (mg/dL)} \times \text{FPG (mg/dL)} / 2]$$

$$\text{THR (triglyceride-to-HDL ratio)} = \text{TG (mmol/L)} / \text{HDL (mmol/L)}$$

$$\text{CMI (cardiometabolic index)} = \text{TG (mmol/L)} / \text{HDL (mmol/L)} \times \text{WHtR}$$

$$\text{eGDR}_{\text{WHR}}\text{ (eGDR calculated with WHR)} = 24.31 - (12.22 \times \text{WHR}) - (3.29 \times \text{HTN}) - [0.57 \times \text{HbA1c (\%)}]$$

$$\text{eGDR}_{\text{WC}}\text{ (eGDR calculated with WC)} = 21.158 - 0.09 \times \text{WC (cm)} - (3.407 \times \text{HTN}) - [0.551 \times \text{HbA1c (\%)}]$$

$$\text{eGDR}_{\text{BMI}}\text{ (eGDR calculated with BMI)} = 19.02 - 0.22 \times \text{BMI}\left(\text{kg/m}^2\right) - (3.26 \times \text{HTN}) - [0.61 \times \text{HbA1c (\%)}]$$

eGDR (estimated glucose disposal rate) is expressed in mg/kg/min, and HTN refers to a hypertension history (0 = no, 1 = yes).

For males:

$$\text{VAI (visceral adiposity index)} = \text{WC (cm)} / \left[39.68 + 1.88 \times \text{BMI}\left(\text{kg/m}^2\right)\right] \times [\text{TG (mmol/L)} / 1.03] \times [1.31/\text{HDL (mmol/L)}]$$

$$\text{CVAI (Chinese visceral adiposity index)} = -267.93 + 0.68 \times \text{age (years)} + 0.03 \times \text{BMI}\left(\text{kg/m}^2\right) + 4.00 \times \text{WC (cm)} + 22.00 \times \text{Log}_{10}\text{TG (mmol/L)} - 16.32 \times \text{HDL (mmol/L)}$$

$$\text{LAP (lipid accumulation product)} = [\text{WC (cm)} - 65] \times \text{TG (mmol/L)}$$

For females:

$$\text{VAI} = \text{WC (cm)} / \left[36.58 + 1.89 \times \text{BMI}\left(\text{kg/m}^2\right)\right] \times [\text{TG (mmol/L)} / 0.81] \times [1.52/\text{HDL (mmol/L)}]$$

$$\begin{aligned}\mathrm{CVAI} = &-187.32 + 1.71 \times \mathrm{age\,(years)} + 4.23 \\ &\times \mathrm{BMI}\left(\mathrm{kg/m^2}\right) + 1.12 \times \mathrm{WC\,(cm)} \\ &+ 39.76 \times \mathrm{Log_{10}TG\,(mmol/L)} \\ &- 11.66 \times \mathrm{HDL\,(mmol/L)}\end{aligned}$$

$$\mathrm{LAP} = [\mathrm{WC\,(cm)} - 58] \times \mathrm{TG\,(mmol/L)}$$

Carotid ultrasound imaging

All enrolled participants underwent standardized carotid ultrasound examinations. A comprehensive carotid ultrasound protocol was used to assess carotid intima-media thickness (cIMT) and plaque according to Mannheim's consensus [35]. Carotid ultrasonography was conducted by experienced clinicians using a Siemens Acuson Sequoia 512 equipped with a 15 MHz linear array probe. The bilateral common carotid artery, internal carotid artery, external carotid artery, and the right subclavian artery were systematically scanned. An increased cIMT was defined as a measurement of > 0.9 mm in any carotid artery [36]. Plaques were identified using B-mode and color Doppler examinations conducted in longitudinal and transverse planes to assess circumferential asymmetry. Plaques were defined as focal structures encroaching into the arterial lumen by at least 0.5 mm or 50% of the adjacent cIMT, or demonstrating a thickness of > 1.5 mm from the intima-lumen interface to the media-adventitia interface [35]. SCA was defined as the presence of an increased cIMT and/or plaques without associated clinical manifestations.

Statistical analysis

Data analyses were performed using R version 4.3.3 and SPSS 26.0. A two-sided *P* value < 0.05 was considered to be statistically significant. The normal distribution and equality of variance for continuous variables were evaluated by the Kolmogorov-Smirnov test and Levene test, respectively. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range: 25th-75th percentile). Comparisons between two groups were performed using Student's *t*-test or Mann-Whitney *U* test, while comparisons among three groups were conducted using one-way analysis of variance (ANOVA) or Kruskal-Wallis *H* test, as appropriate. Categorical variables were presented as counts and percentages. Differences between groups were assessed using the chi-square test or Fisher's exact test, as applicable.

The OIRIs were standardized (Z-score) and then included in logistic regression models. Univariable logistic regression analysis was carried out prior to multivariable analysis to identify potential risk factors and covariates (Additional file 1, Table S1). Based on a significance level of $P < 0.05$, variables that showed significant association with SCA in univariable analysis or had clinical implications were included in multivariable analysis. Three binary logistic regression models were developed. Model 1 was unadjusted. Model 2 adjusted for age, sex, diabetes duration, smoking, alcohol drinking, and family history of any diabetes. Model 3 further adjusted for hypertension, HDL, LDL, FPG, and ACEI based on model 2. All included variables were assessed for collinearity, and no multicollinearity was found (all variance inflation factors < 5; Additional file 1, Table S2).

Receiver operating characteristic (ROC) curves and the areas under the curves (AUCs) were compared to determine the index most strongly associated with SCA. Next, subgroup analyses, categorized by sex, age, diabetes duration, smoking status, family history of any diabetes, and hypertension, were conducted to further validate the association between CVAI and SCA. Restricted cubic spline (RCS) models were applied to explore the linear or nonlinear association between continuous CVAI and SCA. The number of knots was chosen based on the minimum values of the Akaike Information Criterion and Bayesian Information Criterion. In addition, the net reclassification improvement (NRI) and integrated discrimination improvement (IDI) indices were calculated to assess the incremental diagnostic performance of CVAI for SCA beyond a basic model with the 10-year CVD risk evaluated by Steno Type 1 Risk Engine (ST1RE) [37], which was specifically developed for T1D with 10 risk factors.

Finally, sensitivity analyses were performed to further verify the association between CVAI and SCA: (1) Multinomial and ordinal logistic regression analyses were applied to explore the associations of CVAI with different SCA types, with the participants categorized into no plaque, single plaque, and multiple plaque groups. Both multinomial and ordinal regression models included plaque status as dependent variable and standardized CVAI as independent variable, adjusting for age, sex, diabetes duration, smoking, alcohol drinking, family history of any diabetes, hypertension, HDL, LDL, FPG, and ACEI; (2) Since metformin has been reported to correlate with body weight loss, analyses were conducted for participants without metformin treatment; (3) As CVAI is a surrogate indicator for obesity and insulin resistance, analyses were also conducted for participants without obesity or overweight.

Results

Baseline characteristics of participants according to SCA

This study included 418 adult participants with classic T1D, of whom 218 (52.15%) were male. The median age was 31.0 (24.0–45.0) years, and the median diabetes duration was 6.0 (1.0–12.0) years. The prevalence of SCA was 21.29% (Table 1). Compared to those without SCA, participants with SCA were older, had longer diabetes

Table 1 Baseline characteristics of type 1 diabetes participants with or without subclinical carotid atherosclerosis

Variable	Overall (*n* = 418)	Non-SCA (*n* = 329)	SCA (*n* = 89)	*P* value
Demographic				
Male (n, %)	218 (52.15)	166 (50.46)	52 (58.43)	0.224
Age (years)	31.0 (24.0–45.0)	29.0 (23.0–38.0)	50.0 (43.0–57.0)	**< 0.001**
Duration (years)	6.0 (1.0–12.0)	5.0 (1.0–11.0)	8.0 (3.0–15.0)	**0.002**
Smoking (n, %)	95 (22.73)	71 (21.58)	24 (26.97)	0.351
Drinking (n, %)	32 (7.66)	22 (6.69)	10 (11.24)	0.227
Family history (n, %)	104 (24.88)	78 (23.71)	26 (29.21)	0.354
Hypertension (n, %)	65 (15.55%)	51 (15.50%)	14 (15.73%)	0.958
Anthropometric				
SBP (mmHg)	120 (110–130)	118 (110–126)	121 (115–130)	**0.005**
DBP (mmHg)	75 (70–80)	75 (69–80)	76 (70–80)	0.438
Height (cm)	165 ± 8.60	165 ± 8.65	165 ± 8.45	0.761
Weight (kg)	55 (49–62)	55 (49–61)	55 (50–65)	0.187
WC (cm)	75.7 ± 8.3	75.1 ± 8.2	78.0 ± 8.1	**0.003**
HC (cm)	90 (85–94)	90 (85–94)	90 (85–95)	0.074
Biochemical				
HDL (mmol/L)	1.23 (1.01–1.48)	1.19 (0.97–1.44)	1.36 (1.11–1.65)	**< 0.001**
LDL (mmol/L)	2.24 (1.74–2.76)	2.17 (1.67–2.69)	2.45 (1.92–2.88)	**0.009**
TC (mmol/L)	3.95 (3.39–4.61)	3.87 (3.33–4.56)	4.18 (3.62–4.90)	**0.003**
TG (mmol/L)	0.98 (0.74–1.87)	0.98 (0.74–1.71)	1.00 (0.77–2.47)	0.313
FPG (mmol/L)	8.20 (5.41–11.97)	8.20 (5.61–12.14)	8.21 (5.29–11.50)	0.455
HbA1c (%)	9.1 (7.8–10.7)	9.0 (7.6–10.6)	9.2 (8.1–10.7)	0.354
SUA (μmol/L)	192 (125–264)	188 (121–258)	207 (142–269)	0.221
ALT (IU/L)	14 (11–21)	14 (10–21)	15 (12–21)	0.240
Creatinine (μmol/L)	83 (71–96)	83 (71–95)	83 (66–98)	0.870
OIRI				
BMI (kg/m^2)	20.22 ± 2.78	20.06 ± 2.74	20.81 ± 2.88	**0.031**
WHtR	0.46 (0.42–0.49)	0.45 (0.42–0.49)	0.47 (0.44–0.50)	**0.001**
WHR	0.85 ± 0.06	0.84 ± 0.06	0.86 ± 0.06	0.050
ABSI	0.08 (0.08–0.08)	0.08 (0.08–0.08)	0.08 (0.08–0.08)	0.050
AVI	11.57 (9.92–13.08)	11.41 (9.90–12.90)	12.80 (10.74–13.96)	**0.003**
BAI	24.20 ± 4.08	23.99 ± 4.12	25.00 ± 3.86	**0.032**
BRI	2.59 (2.02–3.15)	2.50 (1.93–3.13)	2.85 (2.38–3.36)	**0.001**
CI	1.20 (1.14–1.25)	1.19 (1.14–1.25)	1.22 (1.17–1.27)	**0.005**
TyG	8.93 (8.29–9.77)	8.93 (8.26–9.79)	8.93 (8.44–9.71)	0.766
VAI	1.31 (0.82–2.71)	1.32 (0.89–2.71)	1.18 (0.71–3.03)	0.638
CVAI	35.4 (12.9–66.3)	29.3 (5.0-56.2)	67.7 (31.2–88.6)	**< 0.001**
LAP	14.13 (6.57–30.20)	13.72 (5.85–27.90)	15.30 (9.20-37.05)	**0.021**
$eGDR_{WHR}$	8.33 (7.12–9.47)	8.44 (7.32–9.57)	7.87 (6.81–9.23)	0.077
$eGDR_{WC}$	8.86 (7.74–9.92)	8.97 (7.85–10.10)	8.56 (7.43–9.36)	0.089
$eGDR_{BMI}$	8.48 (7.29–9.54)	8.58 (7.46–9.70)	8.38 (6.90–9.28)	0.205
THR	0.88 (0.55–1.85)	0.89 (0.56–1.70)	0.83 (0.50–2.29)	0.704
CMI	0.39 (0.25–0.86)	0.39 (0.25–0.81)	0.41 (0.25–1.04)	0.981
Medication				
Metformin (n, %)	34 (8.13)	24 (7.29)	10 (11.24)	0.323
ACEI (n, %)	9 (2.15)	4 (1.22)	5 (5.62)	**0.024**
ARB (n, %)	19 (4.55)	12 (3.65)	7 (7.87)	0.145
CCB (n, %)	14 (3.35)	11 (3.34)	3 (3.37)	1.000
BRB (n, %)	11 (2.63)	7 (2.13)	4 (4.49)	0.258

Table 1 (continued)

Variable	Overall (*n* = 418)	Non-SCA (*n* = 329)	SCA (*n* = 89)	*P* value
Statins (n, %)	99 (23.68)	28 (8.51)	71 (79.78)	**< 0.001**
Antiplatelets (n, %)	64 (15.31)	12 (3.65)	52 (58.43)	**< 0.001**

P value in bold indicates statistical significance (P < 0.05)

Data are expressed as mean ± SD, median (IQR) or number (percentage) as appropriate

ABSI, a body shape index; ACEI, angiotensin-converting enzyme inhibitor; ALT, alanine aminotransferase; ARB, angiotensin II receptor blocker; AVI, abdominal volume index; BAI, body adiposity index; BMI, body mass index; BRB, beta-adrenergic receptor blocker; BRI, body roundness index; CCB, calcium channel blocker; CI, conicity index; CMI, cardiometabolic index; CVAI, Chinese visceral adiposity index; DBP, diastolic blood pressure; $eGDR_{BMI}$, estimated glucose disposal rate calculated with BMI; $eGDR_{WC}$, estimated glucose disposal rate calculated with WC; $eGDR_{WHR}$, estimated glucose disposal rate calculated with WHR; FPG, fasting plasma glucose, HbA1c, hemoglobin A1c; HC, hip circumference; HDL, high density lipoprotein cholesterol; IQR, interquartile range; LAP, lipid accumulation product; LDL, low density lipoprotein cholesterol; OIRI, obesity- and insulin resistance-related index; SBP, systolic blood pressure; SCA, subclinical carotid atherosclerosis; SUA, serum uric acid; TC, total cholesterol; TG, triglyceride; THR, triglyceride-to-HDL ratio; TyG, triglyceride-glucose index; VAI, visceral adiposity index; WC, waist circumference; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio

Table 2 Association between OIRIs and SCA in T1D participants: univariable and multivariable logistic regression analyses

Variable	Model 1		Model 2		Model 3	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value
BMI	1.32 (1.03–1.68)	**0.027**	0.97 (0.73–1.28)	0.819	0.98 (0.70–1.36)	0.892
WHtR	1.44 (1.13–1.84)	**0.003**	1.11 (0.84–1.49)	0.463	1.19 (0.84–1.69)	0.323
WHR	1.27 (1.00-1.62)	0.055	1.04 (0.78–1.37)	0.805	1.18 (0.84–1.66)	0.328
ABSI	1.22 (0.96–1.53)	0.097	1.16 (0.88–1.52)	0.287	1.23 (0.90–1.69)	0.187
AVI	1.42 (1.12–1.80)	**0.004**	1.04 (0.78–1.37)	0.802	1.11 (0.78–1.56)	0.568
BAI	1.29 (1.01–1.63)	**0.039**	1.20 (0.87–1.67)	0.263	1.16 (0.80–1.69)	0.448
BRI	1.41 (1.11–1.78)	**0.004**	1.09 (0.82–1.46)	0.534	1.16 (0.82–1.63)	0.408
CI	1.33 (1.05–1.69)	**0.018**	1.15 (0.87–1.53)	0.310	1.25 (0.90–1.74)	0.180
TyG	1.01 (0.78–1.30)	0.939	1.13 (0.85–1.52)	0.403	1.03 (0.73–1.44)	0.854
VAI	1.10 (0.88–1.36)	0.359	1.11 (0.87–1.44)	0.382	0.98 (0.69–1.35)	0.918
CVAI	2.31 (1.79–3.03)	**< 0.001**	1.48 (1.10–2.01)	**0.010**	1.68 (1.16–2.47)	**0.007**
LAP	1.18 (0.96–1.46)	0.107	1.09 (0.87–1.39)	0.435	0.96 (0.69–1.26)	0.785
$eGDR_{WHR}$	0.78 (0.57–1.06)	0.116	0.83 (0.57–1.20)	0.317	0.44 (0.22–0.82)	**0.013**
$eGDR_{WC}$	0.77 (0.56–1.05)	0.094	0.88 (0.61–1.27)	0.500	0.49 (0.24–0.93)	**0.035**
$eGDR_{BMI}$	0.84 (0.61–1.14)	0.256	0.95 (0.66–1.37)	0.771	0.64 (0.34–1.17)	0.154
THR	1.12 (0.90–1.37)	0.286	1.11 (0.88–1.42)	0.401	0.95 (0.67–1.31)	0.782
CMI	1.14 (0.92–1.41)	0.209	1.11 (0.88–1.44)	0.395	0.96 (0.66–1.34)	0.828

P value in bold indicates statistical significance (P < 0.05)

Odds ratios for SCA are presented as per 1.0-SD increase in OIRIs

Model 1: not adjusted

Model 2: adjusted for age, sex, diabetes duration, smoking, alcohol drinking, and family history of any diabetes

Model 3: model 2 + further adjusted for hypertension, HDL, LDL, FPG, and ACEI

ABSI, a body shape index; ACEI, angiotensin-converting enzyme inhibitor; AVI, abdominal volume index; BAI, body adiposity index; BMI, body mass index; BRI, body roundness index; CI, conicity index; CMI, cardiometabolic index; CVAI, Chinese visceral adiposity index; $eGDR_{BMI}$, estimated glucose disposal rate calculated with BMI; $eGDR_{WC}$, estimated glucose disposal rate calculated with WC; $eGDR_{WHR}$, estimated glucose disposal rate calculated with WHR; FPG, fasting plasma glucose; HDL, high-density lipoprotein cholesterol; LAP, lipid accumulation product; LDL, low-density lipoprotein cholesterol; OIRIs, obesity- and insulin resistance-related indices; SCA, subclinical carotid atherosclerosis; T1D, type 1 diabetes; THR, triglyceride-to-HDL ratio; TyG, triglyceride-glucose index; VAI, visceral adiposity index; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio

duration, and showed higher levels of clinical parameters (SBP, WC, BMI, WHtR, AVI, BAI, BRI, CI, CVAI, LAP), lipids (HDL, LDL, TC), and a higher use of medications (ACEI, statins, antiplatelet agents) (all $P < 0.05$).

Associations of OIRIs with SCA

Logistic regression analyses were performed to investigate the associations between OIRIs and SCA (Table 2). First, potential confounding factors showing statistical significance in univariable analyses were identified and included in multivariable analyses, including age, diabetes duration, SBP, WC, HC, HDL, LDL, TC, and the use of ACEI, statins, and antiplatelet agents (Additional file 1, Table S1). Second, given that sex [38], smoking [39], alcohol drinking [40], family history of diabetes [41], elevated FPG [42], and hypertension [43] are traditional risk factors for atherosclerosis, they were also included in multivariable analyses. Third, based on multicollinearity tests, variables with high correlations were excluded, including WC, HC, TC, statins, and antiplatelet agents (Additional file 1, Table S2). Finally, after adjusting for age, sex, diabetes duration, smoking, alcohol drinking,

Table 3 Comparative diagnostic performance of various OIRIs for SCA in type 1 diabetes participants

Variable	Cutoff	Sensitivity (%)	Specificity (%)	Youden	AUC	95% CI	*P* value
CVAI	56.307	62.92	75.38	0.383	0.732	0.686–0.774	Ref.
WHtR	0.465	61.80	61.09	0.229	0.611	0.563–0.658	**< 0.001**
BRI	2.721	61.80	61.09	0.229	0.611	0.563–0.658	**< 0.001**
AVI	12.791	50.56	71.73	0.223	0.603	0.555–0.651	**< 0.001**
CI	1.200	65.17	54.10	0.193	0.596	0.547–0.644	**< 0.001**
$eGDR_{WHR}$	8.346	61.82	53.33	0.152	0.581	0.510–0.649	**0.007**
LAP	6.450	87.64	27.96	0.156	0.580	0.531–0.627	**< 0.001**
$eGDR_{WC}$	9.118	70.91	46.67	0.176	0.578	0.507–0.646	**0.002**
BAI	23.826	65.17	51.67	0.168	0.575	0.526–0.623	**< 0.001**
ABSI	0.076	82.02	31.61	0.136	0.568	0.519–0.616	**< 0.001**
BMI	18.796	77.53	37.08	0.146	0.563	0.514–0.612	**< 0.001**
WHR	0.848	58.43	52.28	0.107	0.562	0.513–0.610	**< 0.001**
$eGDR_{BMI}$	8.818	67.27	45.33	0.126	0.558	0.487–0.627	**0.002**
VAI	0.876	33.71	75.08	0.088	0.516	0.467–0.565	**0.001**
THR	2.888	76.40	15.20	0.084	0.513	0.464–0.562	**0.001**
TyG	8.485	74.36	36.07	0.104	0.511	0.455–0.567	**< 0.001**
CMI	1.374	22.47	84.19	0.067	0.501	0.452–0.550	**< 0.001**

P value in bold indicates statistical significance (P < 0.05)

ABSI, a body shape index; AVI, abdominal volume index; BAI, body adiposity index; BMI, body mass index; BRI, body roundness index; CI, conicity index; CMI, cardiometabolic index; CVAI, Chinese visceral adiposity index; $eGDR_{BMI}$, estimated glucose disposal rate calculated with BMI; $eGDR_{WC}$, estimated glucose disposal rate calculated with WC; $eGDR_{WHR}$, estimated glucose disposal rate calculated with WHR; LAP, lipid accumulation product; OIRIs, obesity- and insulin resistance-related indices; SCA, subclinical carotid atherosclerosis; THR, triglyceride-to-HDL ratio; TyG, triglyceride-glucose index; VAI, visceral adiposity index; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio

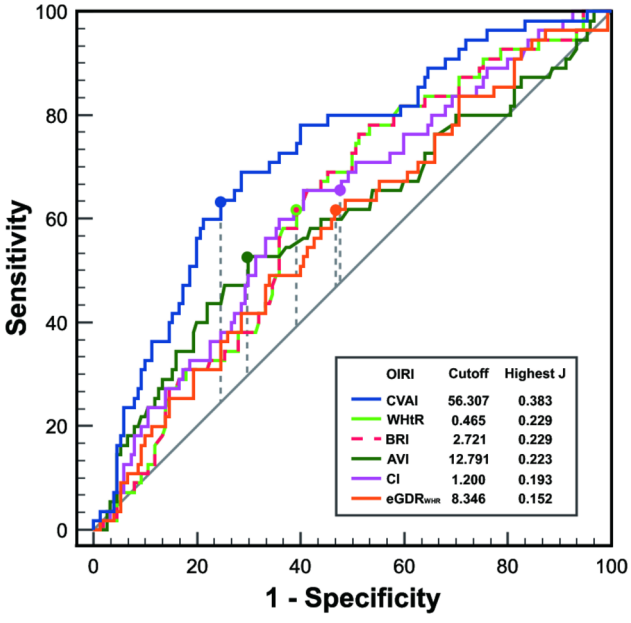


Fig. 2 ROC curves of the top six OIRIs (CVAI, WHtR, BRI, AVI, CI, $eGDR_{WHR}$) for diagnosing SCA in T1D participants. The ROC curves of WHtR and BRI completely overlap. The solid dot represents the optimal cutoff, while the gray dashed line represents the highest J value. Abbreviations: AVI, abdominal volume index; BRI, body roundness index; CI, conicity index; CVAI, Chinese visceral adiposity index; $eGDR_{WHR}$, estimated glucose disposal rate calculated with WHR; OIRIs, obesity- and insulin resistance-related indices; ROC, receiver operating characteristic; SCA, subclinical carotid atherosclerosis; T1D, type 1 diabetes; WHtR, waist-to-height ratio

family history of any diabetes, hypertension, HDL, LDL, FPG, and ACEI, CVAI (OR, 95% CI: 1.68, 1.16–2.47), $eGDR_{WHR}$ (OR, 95% CI: 0.44, 0.22–0.82), and $eGDR_{WC}$ (OR, 95% CI: 0.49, 0.24–0.93) were independently associated with the presence of SCA.

Diagnostic performance of each OIRI for SCA

ROC curves and AUCs were used to compare the diagnostic performance of each OIRI in predicting SCA. The CVAI showed superior diagnostic performance relative to other indices, achieving an AUC of 0.732 (95% CI: 0.686–0.774). The optimal cutoff value for CVAI was 56.307, with a sensitivity of 62.92% and a specificity of 75.38% (Table 3; Fig. 2).

Subgroup analysis of CVAI and SCA association

The association between CVAI and SCA was assessed in subgroup analyses categorized by sex, age, diabetes duration, smoking, family history of any diabetes, and hypertension (Fig. 3). No significant interaction was found between CVAI and these subgroup variables for SCA. However, a significant association was observed among females, participants aged ≥ 31 years, with a diabetes duration of ≥ 5 years, non-smokers, participants without a family history of any diabetes, and with hypertension.

Analysis of the linearity of CVAI and SCA association

To explore whether a linear or nonlinear association existed between the continuous CVAI and the presence of SCA, RCS models based on multivariable logistic

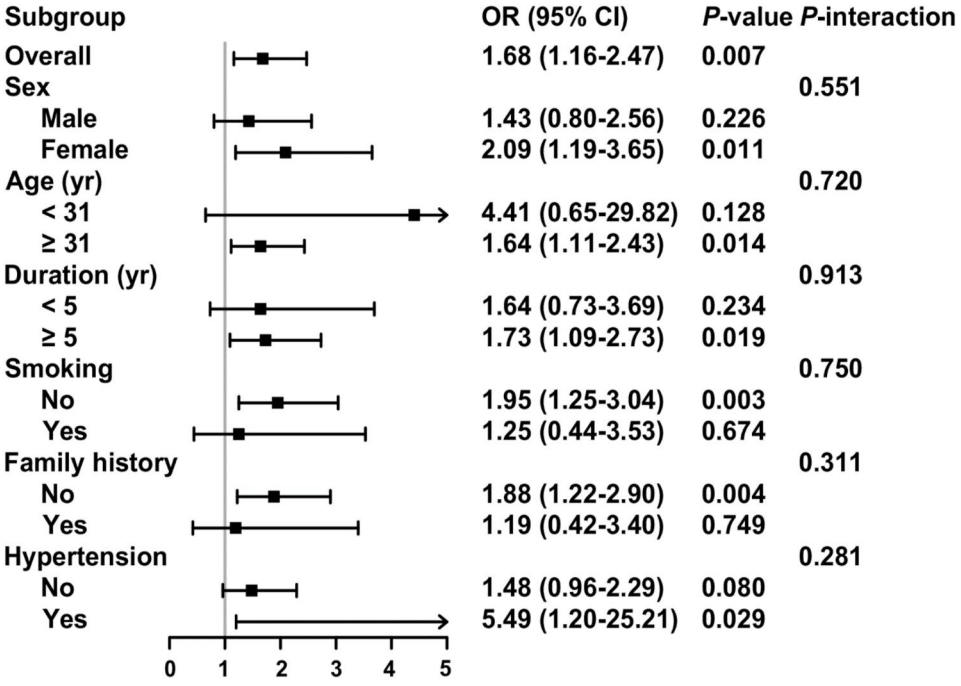


Fig. 3 Association between CVAI and SCA in different T1D participant subgroups. Each subgroup was adjusted for age, sex, diabetes duration, smoking, alcohol drinking, family history of any diabetes, hypertension, HDL, LDL, FPG, and ACEI. Odds ratios for SCA are presented as per 1.0-SD increase in CVAI. Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; CVAI, Chinese visceral adiposity index; FPG, fasting plasma glucose; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; SCA, subclinical carotid atherosclerosis; T1D, type 1 diabetes

regression were performed (Fig. 4). In the overall population and the females, RCS showed a linear and positive association in a dose-response pattern (both *P* for overall < 0.05; both *P* for nonlinear > 0.05). No significant association was observed in males (*P* for overall > 0.05).

Assessment of CVAI's incremental diagnostic performance for SCA

ROC curves were constructed to compare the diagnostic performance of ST1RE for SCA with and without CVAI (Table 4; Fig. 5). The NRI analysis indicated an improvement in reclassification for SCA [continuous NRI (95% CI): 0.348, 0.041–0.655, $P = 0.027$]. However, it did not enhance the overall discriminative ability of ST1RE, as the difference in AUC was not statistically significant ($P = 0.249$).

Sensitivity analysis of CVAI and SCA association

Firstly, as shown in Table 5, multinomial logistic regression analyses revealed that per 1.0-SD increase in CVAI (OR, 95% CI: 1.71, 1.07–2.72) was independently associated with multiple plaques compared with the no plaque group. This association was further supported by ordinal logistic regression analysis (OR, 95% CI: 1.57, 1.09–2.28). Secondly, a positive association, where per 1.0-SD increase in CVAI was associated with higher odds of SCA (OR, 95% CI: 1.57, 1.08–2.32), remained even after removing participants treated with metformin (Additional file 1, Table S3). Thirdly, the result (OR, 95% CI: 1.70, 1.15–2.57) aligned with the primary analysis after excluding T1D participants who were either obese or overweight (Additional file 1, Table S4).

Discussion

This study explored and compared a series of common OIRIs for their associations with SCA in T1D participants. The main findings were as follows: (1) Among all indices included, the CVAI, $eGDR_{WHR}$, and $eGDR_{WC}$ were independently associated with the presence of SCA after adjusting for potential confounders; (2) CVAI was positively associated with the presence of SCA and showed the highest AUC in diagnosing SCA. The independent and positive association of CVAI with SCA was consistently observed across subgroup analyses, sensitivity analyses, and RCS analyses.

CVAI is a comprehensive index that incorporates multiple anthropometric and metabolic parameters to assess visceral adiposity and was developed specifically for the Chinese population based on body fat characteristics [44]. It has been proven to be an independent predictor for CVD [45]. Cross-sectional studies reported that CVAI was independently associated with an increased risk of carotid atherosclerosis in individuals aged ≥ 55 years [32], steelworkers [31], and individuals with normal body weight [33]. Additionally, in a large cohort study, CVAI was an independent predictor for carotid plaques

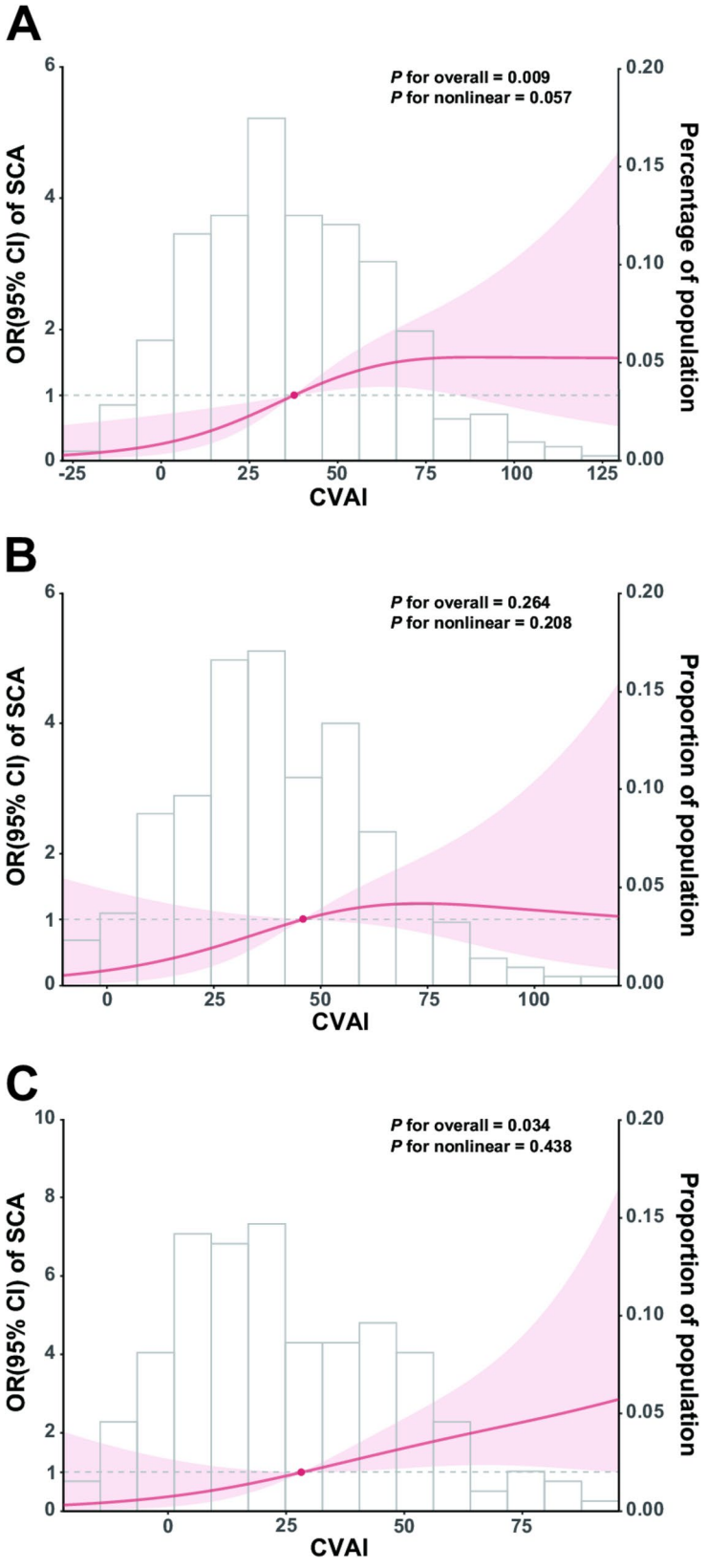


Fig. 4 (See legend on next page.)

(See figure on previous page.)

Fig. 4 Restricted cubic spline curves for association of CVAI with SCA in T1D participants. **A** Association in the overall population; **B** Association in males; **C** Association in females. Odds ratios are indicated by solid lines and 95% CIs by shaded areas. In A, RCS model adjusted for age, sex, diabetes duration, smoking, alcohol drinking, family history of any diabetes, hypertension, HDL, LDL, FPG, and ACEI. In B and C, RCS model adjusted for age, diabetes duration, smoking, alcohol drinking, family history of any diabetes, hypertension, HDL, LDL, FPG, and ACEI. Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; CVAI, Chinese visceral adiposity index; FPG, fasting plasma glucose; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; RCS, restricted cubic spline; SCA, subclinical carotid atherosclerosis; T1D, type 1 diabetes

(HR, 95% CI: 1.53, 1.48–1.59), and reached an AUC (95% CI) value of 0.71 (0.56–0.76), which was higher than other adiposity indices [46]. It's noteworthy that all the studies were conducted in the general population and focused on atherosclerosis. Our results align with these studies and extend the limited evidence on the association between CVAI and atherosclerosis to T1D population, but we emphasized SCA, which is the early stage of atherosclerosis. Longitudinal studies with large cohorts are needed to validate the association between CVAI and SCA and then explore CVAI's predictive value for SCA and its progression in T1D.

The association between BMI and SCA in T1D has been noted in prior studies, but has not been the primary objective. In a cohort study aiming to explore the effect of intensive insulin treatment on cIMT, BMI was one of the traditional predictors for increased cIMT [47]. In our study, CVAI showed superior diagnostic performance to other indices, including BMI. CVAI considers ethnic differences and incorporates not only BMI but also sex, age, and lipid profiles, which can explain its advantages in diagnosing SCA. On the other hand, CVAI reflects the accumulation of visceral adipose tissue, which is more prone to releasing free fatty acids and proinflammatory factors compared to subcutaneous adipose tissue, thereby accelerating atherosclerosis [48].

Previous studies have reported the relationship of novel insulin resistance-related indices with CVD [22–24]. For instance, a review systematically evaluated the application value of TyG for different types of CVD and its potential limitations in predicting CVD events [24]. In the FinnDiane cohort study, baseline eGDR was a strong predictor for subsequent coronary artery disease in T1D subjects [23]. With regard to SCA in T1D, only the association of eGDR has now been specifically examined in two studies [49, 50]. In a cross-sectional study with 191 T1D participants, eGDR was independently related to multiple plaques and maximum plaque height [49]. Kuper et al. explored risk factors for SCA in 45 females with T1D and reported a negative correlation between eGDR and both cIMT and carotid plaques [50]. Consistently, our results showed a significant association between eGDR and SCA in the fully adjusted model. The mechanisms underlying the associations of eGDR and CVAI with SCA are different. Hyperinsulinemia resulting from insulin resistance (decreased eGDR) directly promotes inflammatory factors and endothelial dysfunction, and also contributes to adipose tissue accumulation and metabolic abnormalities [51–53]. In contrast, CVAI primarily exerts its effects through direct visceral adiposity-mediated pathways. In other words, insulin resistance might influence SCA through multiple pathways and both direct and indirect mechanisms, while CVAI might affect SCA through a relatively singular pathway and mainly direct mechanisms. Taken together, our results further support the association between OIRIs and atherosclerosis. The results also indicate that mechanistic studies are needed to elucidate the pathophysiological pathways connecting visceral adiposity to SCA in T1D.

The sex differences in the risk of atherosclerosis associated with obesity and/or insulin resistance have been reported before. Rodrigues et al. found that eGDR was independently associated with coronary artery calcification only in female T1D patients, but not in males [54]. In a cross-sectional study with 1457 community inhabitants, an independent association was observed between TyG and subclinical atherosclerosis in non-diabetic females, while no significant association was found in non-diabetic males and diabetic patients [55]. Moreover, substantial epidemiological evidence suggests that female patients with T1D suffer from a higher risk of CVD morbidity and mortality compared to male patients. This may be due to diabetes-associated reproductive hormone disturbance and greater lifetime hyperglycemia exposure caused by a greater impairment of insulin sensitivity [56]. These findings could partially explain the sex difference in the association of CVAI with SCA, as reported here. In participants without a family history of any diabetes and who were non-smokers, a positive association between CVAI and SCA was found in this study. This suggests that CVAI may correlate with SCA independently of a family history of any diabetes and smoking status. Similarly, a cross-sectional study also reported an independent association between CVAI and carotid atherosclerosis in steelworkers who did not smoke [31].

ST1RE, which incorporates multiple risk factors, has been developed and validated for CVD risk assessment in T1D [37, 57]. A recent study showed that ST1RE outperformed ESC/EASD-2019 in identifying preclinical atherosclerosis in T1D adults, with an AUC of 0.691 [58]. In our study, ST1RE showed high diagnostic performance with an AUC of 0.861 (95% CI: 0.804–0.917). The difference in diagnostic performance may be caused by the younger participants and shorter diabetes duration in our

Table 4 Assessment of CVAI's incremental diagnostic performance for SCA in type 1 diabetes participants

Model	C-Statistic			NRI-continuous			IDI		
	AUC	95% CI	*P* value	Estimation	95% CI	*P* value	Estimation	95% CI	*P* value
ST1RE	0.861	0.804–0.917	Ref.	–	–	–	–	–	–
ST1RE + CVAI	0.851	0.790–0.912	0.249	0.348	0.041–0.655	**0.027**	0.006	− 0.011–0.022	0.519

P value in bold indicates statistical significance (P < 0.05)

Due to the lack of information on regular exercise, it was not included in our analysis

CVAI, Chinese visceral adiposity index; IDI, integrated discrimination improvement; NRI, net reclassification index; SCA, subclinical carotid atherosclerosis; ST1RE, Steno Type 1 Risk Engine

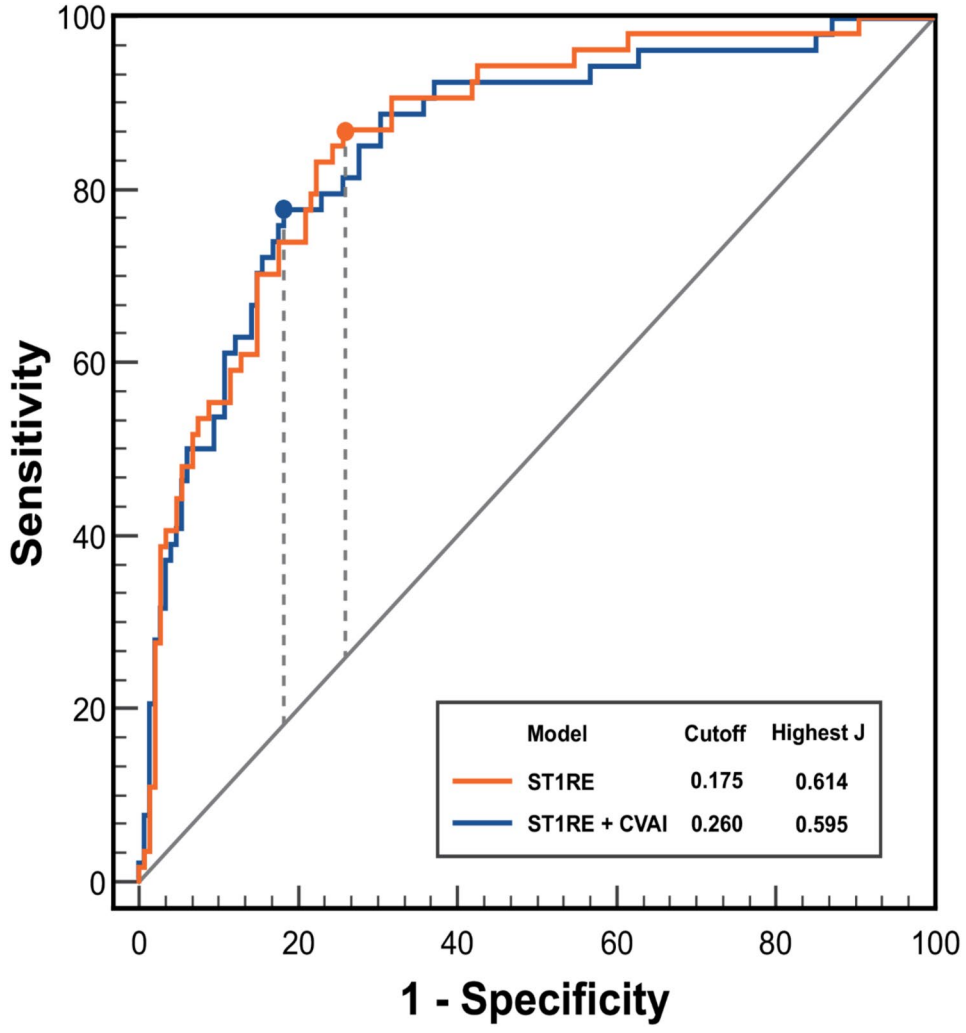


Fig. 5 Receiver operating characteristic curve of CVAI to predict SCA in T1D participants. The ST1RE incorporated age, female, smoking, diabetes duration, SBP, LDL, HbA1c, eGFR, and albuminuria. The solid dot represents the optimal cutoff, while the gray dashed line represents the highest J value. Abbreviations: CVAI, Chinese visceral adiposity index; eGFR, estimated glomerular filtration rate; HbA1c, hemoglobin A1c; LDL, low density lipoprotein cholesterol; SBP, systolic blood pressure; SCA, subclinical carotid atherosclerosis; ST1RE, Steno Type 1 Risk Engine; T1D, type 1 diabetes

study. Notably, the incorporation of CVAI did not further enhance ST1RE's diagnostic capability, which may be attributed to the already excellent discriminative power of ST1RE alone.

This study has several strengths: First, to the best of our knowledge, this is the first study to systematically investigate and compare the majority of common OIRIs for their associations with SCA in T1D. This comprehensive evaluation within a single study population has the potential to provide an overview of the associations between OIRIs and SCA in T1D without inter-study variability; the comparison contributes to identifying the index that is most strongly associated with SCA. Second, this is the first study to investigate CVAI in T1D and

Table 5 Association between OIRIs and SCA in T1D participants: multinomial and ordinal logistic regression analyses

Variable	Single plaque ($n=31$)*		Multiple plaques ($n=47$)*		Overall ($n=418$)#	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value
BMI	0.87 (0.54–1.37)	0.538	0.92 (0.61–1.37)	0.669	0.91 (0.66–1.25)	0.551
WHtR	1.11 (0.68–1.81)	0.676	1.00 (0.65–1.53)	0.997	1.02 (0.73–1.43)	0.909
WHR	1.18 (0.73–1.90)	0.504	1.00 (0.66–1.50)	0.985	1.03 (0.74–1.44)	0.856
ABSI	1.34 (0.85–2.10)	0.209	1.15 (0.77–1.70)	0.498	1.16 (0.85–1.59)	0.353
AVI	1.05 (0.64–1.71)	0.844	1.01 (0.66–1.55)	0.968	1.01 (0.71–1.42)	0.975
BAI	1.03 (0.61–1.75)	0.908	1.05 (0.66–1.67)	0.840	1.04 (0.72–1.51)	0.817
BRI	1.08 (0.67–1.75)	0.753	0.97 (0.63–1.48)	0.874	0.99 (0.71–1.39)	0.958
CI	1.30 (0.82–2.08)	0.267	1.12 (0.75–1.68)	0.583	1.14 (0.82–1.57)	0.439
TyG	1.19 (0.73–1.93)	0.489	1.04 (0.67–1.62)	0.853	1.05 (0.74–1.48)	0.790
VAI	1.07 (0.67–1.70)	0.782	0.93 (0.59–1.46)	0.747	0.94 (0.66–1.34)	0.741
CVAI	1.48 (0.87–2.52)	0.150	1.71 (1.07–2.72)	**0.025**	1.57 (1.09–2.28)	**0.017**
LAP	1.06 (0.72–1.56)	0.785	0.90 (0.59–1.38)	0.628	0.93 (0.68–1.27)	0.642
$eGDR_{WHR}$	0.64 (0.27–1.53)	0.313	0.46 (0.21–0.97)	**0.042**	0.56 (0.31–1.01)	0.055
$eGDR_{WC}$	0.87 (0.35–2.19)	0.767	0.40 (0.18–0.89)	**0.024**	0.54 (0.29-1.00)	**0.049**
$eGDR_{BMI}$	1.26 (0.51–3.11)	0.624	0.45 (0.21–0.99)	**0.047**	0.63 (0.35–1.14)	0.126
THR	1.05 (0.65–1.69)	0.847	0.92 (0.59–1.44)	0.716	0.93 (0.66–1.32)	0.699
CMI	1.05 (0.64–1.72)	0.845	0.93 (0.59–1.46)	0.738	0.94 (0.65–1.34)	0.717

P value in bold indicates statistical significance (P < 0.05)

Odds ratios for SCA are presented as per 1.0-SD increase in OIRIs

*Multinomial logistic regression model: results compared to participants with no plaque

#Ordinal logistic regression model: all satisfied the proportional odds assumption

*, #All models adjusted for age, sex, diabetes duration, smoking, alcohol drinking, family history of any diabetes, hypertension, HDL, LDL, FPG, and ACEI

ABSI, a body shape index; ACEI, angiotensin-converting enzyme inhibitor; AVI, abdominal volume index; BAI, body adiposity index; BMI, body mass index; BRI, body roundness index; CI, conicity index; cIMT, carotid intima-media thickness; CMI, cardiometabolic index; CVAI, Chinese visceral adiposity index; $eGDR_{BMI}$, estimated glucose disposal rate calculated with BMI; $eGDR_{WC}$, estimated glucose disposal rate calculated with WC; $eGDR_{WHR}$, estimated glucose disposal rate calculated with WHR; FPG, fasting plasma glucose; HDL, high-density lipoprotein cholesterol; LAP, lipid accumulation product; LDL, low-density lipoprotein cholesterol; OIRIs, obesity- and insulin resistance-related indices; SCA, subclinical carotid atherosclerosis; T1D, type 1 diabetes; THR, triglyceride-to-HDL ratio; TyG, triglyceride-glucose index; VAI, visceral adiposity index; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio

report its association with SCA among these individuals. The findings could inform further research into the potential clinical utility of CVAI in SCA assessment and the underlying mechanisms connecting visceral adiposity to SCA, which have seldom been studied in T1D, a population typically characterized by a lean or normal weight phenotype. Third, the data were collected from inpatients with consistent standards, while the association between CVAI and SCA was determined using a combination of multiple statistical tests, which can strengthen the reliability and robustness of the findings.

On the other hand, several limitations should be considered: First, this is a single-center study in a Chinese T1D population, limiting the generalizability of its conclusions. Second, due to the cross-sectional design, we cannot establish causal associations. Third, our study population of hospitalized adult T1D patients may introduce selection bias and potentially overestimate the association between CVAI and SCA. The magnitude of this bias could be substantial, given that hospitalized patients often have more advanced disease stages. Fourth, despite sample size calculation for the main analysis, the wide confidence intervals in subgroup analyses suggest limited statistical power. Fifth, the binary classification of smoking and alcohol drinking status, rather than more detailed categories, may have led to misclassification bias. This simplification might have underestimated or masked the true associations between these lifestyle factors and SCA, as former smokers/drinkers may have different cardiovascular risk profiles compared to both current and never users. Finally, our database lacked information on dietary habits and physical activity, which are important lifestyle factors that may influence both body composition and cardiovascular risk. However, our sensitivity analysis excluding overweight or obese participants partially mitigated this limitation.

Conclusion

This cross-sectional study revealed a significant association between CVAI and SCA in adults with T1D. The findings suggest that CVAI, an easily accessible index derived from routine clinical measurements, might be a potential marker for SCA risk assessment in this population. Future prospective studies are needed to validate these preliminary findings and further investigate whether CVAI might have potential clinical utility in the assessment of SCA in T1D population.

Abbreviations

ABSI	A body shape index
ACEI	Angiotensin-converting enzyme inhibitor
ALT	Alanine aminotransferase
ANOVA	Analysis of variance
ARB	Angiotensin II receptor blocker
AUC	Area under the curve
AVI	Abdominal volume index
BAI	Body adiposity index
BMI	Body mass index
BRB	Beta-adrenergic receptor blocker
BRI	Body roundness index
CCB	Calcium channel blocker
CI	Conicity index
cIMT	Carotid intima-media thickness
CMI	Cardiometabolic index
CVAI	Chinese visceral adiposity index
CVD	Cardiovascular disease
DBP	Diastolic blood pressure
eGDR	Estimated glucose disposal rate
HbA1c	Glycated hemoglobin
HC	Hip circumference
HDL	High-density lipoprotein cholesterol
HR	Hazard ratio
HTN	Hypertension
IDI	Integrated discrimination improvement
LAP	Lipid accumulation product
LDL	Low-density lipoprotein cholesterol
NRI	Net reclassification improvement
OIRIs	Obesity- and insulin resistance-related indices
OR	Odds ratio
RCS	Restricted cubic spline
ROC	Receiver operating characteristic
SBP	Systolic blood pressure
SCA	Subclinical carotid atherosclerosis
SD	Standard deviation
SUA	Serum uric acid
VAI	Visceral adiposity index
T1D	Type 1 diabetes
TG	Triglyceride
THR	Triglyceride-to-HDL ratio
TyG	Triglyceride-glucose index
WC	Waist circumference
WHR	Waist-to-hip ratio
WHtR	Waist-to-height ratio

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12933-025-02736-2.

Supplementary Material 1.

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Author contributions
QJ and TL designed the study; DY, QX and QL collected the data; TL and DY performed the statistical analyses; TL and JZ wrote the first draft of the manuscript; MAG, LW and LR revised the manuscript. All authors read and approved the final manuscript.

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Data availability
The data supporting the findings of this study are available on reasonable request from the corresponding authors.

Declarations

Ethics approval and consent to participate
This study was approved by the Ethics Review Committee of the First Affiliated Hospital of Air Force Medical University (Approval No. KY20240001301). Informed consent was obtained from all participants included in the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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Declarations.

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