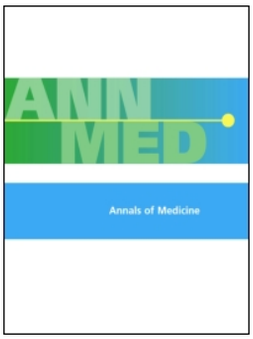

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ORIGINAL ARTICLE

Increased risk of subclinical atherosclerosis associated with high visceral adiposity index in apparently healthy Korean adults: the Kangbuk Samsung Health Study

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ABSTRACT

Background: The visceral adiposity index (VAI) is a mathematical tool that reflects a patient's visceral adiposity and insulin resistance. Recent studies have noted an association between VAI and cardiovascular event. We analyzed the association between VAI and coronary artery calcium score (CACS) in Korean adults.

Methods: For 33,468 participants (mean age 42 yrs) in a health screening program, VAI was calculated using the following formulae: [waist circumference (WC)/{39.68 + (1.88 * body mass index (BMI))}] * (triglyceride/1.03) * {1.31/high-density lipoprotein cholesterol (HDL-C)} for men and [WC/{36.58 + (1.89 * BMI)}] * (triglyceride/0.81) * (1.52/HDL-C) for women. Coronary artery calcium scores were measured with multi-detector computed tomography.

Results: CACS was positively correlated with VAI ($r = 0.027$, $p < 0.001$). Subjects with $0 < CACS < 100$ and $CACS \geq 100$ had significantly higher VAI compared to those with $CACS = 0$ (2.04 ± 1.97, 2.08 ± 1.67 vs. 1.68 ± 1.50, $p < 0.001$). In logistic regression analyses with $CACS > 0$ as the dependent variable, subjects in the highest tertile of VAI (>1.777) had significantly increased odds ratio for $CACS > 0$ compared to subjects in the lowest tertile (<0.967), even after adjusting for confounding variables, including BMI (OR 1.26, 95% CI 1.147–1.381).

Conclusions: Subjects with high VAI had increased risk for subclinical atherosclerosis, as assessed by CACS.

KEY MESSAGES

- Recent studies have noted an association between visceral adiposity index (VAI) and cardiovascular event.
- Subjects with coronary artery calcification (CAC) showed significantly higher VAI compared to those without CAC.
- The subjects with high VAI showed increased odds ratio for CAC as compared to subjects with low VAI, suggesting high VAI reflects increased risk for subclinical atherosclerosis

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Atherosclerosis; coronary artery calcification; visceral adiposity index

Introduction

The absolute risk of cardiovascular disease (CVD) is directly connected to well-established risk factors, such as hypertension, diabetes, age, smoking, etc. Better global risk assessment systems, such as so-called cardiometabolic risk, have emerged to quantify CVD risk because abdominal obesity and insulin resistance-related metabolic markers are also thought to be causal risk factors of CVD (1–4).

Visceral obesity, the most prevalent manifestation of metabolic syndrome, is a marker of dysfunctional adipose tissue and ectopic fat infiltration (5–8). Visceral obesity is related to increase in adipocytokines, proinflammatory activity, exacerbation of insulin sensitivity, diabetes risk, dyslipidemia with high triglyceride (TG)/low high-density lipoprotein cholesterol (HDL-C), hypertension, atherosclerosis, and mortality (9–12). Therefore, measuring visceral adiposity is important in individuals who are at risk for CVD. Computed tomography (CT) scan and magnetic resonance imaging (MRI) at the umbilical level are the standard techniques used to measure visceral fat accumulation (13–18). Several surrogate markers have been routinely used as indicators of visceral adipose function. Increased waist

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Supplemental data for this article can be accessed here.

circumference (WC) and increased waist-to-hip ratio, which are the indicators of central obesity, have been used as the predictors of obesity-related metabolic abnormalities, and are more accurate than body mass index (BMI) (19).

Recently, the visceral adipose index (VAI) was developed as a sex-specific mathematical scoring system that uses simple, classical anthropometric (BMI and WC) and metabolic parameters (TG and HDL-C) to identify insulin resistance and cardiometabolic risk (20). In several studies, VAI has been found to be independently associated with visceral adiposity, making it a useful substitute for visceral CT scan (20–22). One study reported that VAI is a reliable indicator of visceral fat function and insulin sensitivity (22). Some studies have reported that VAI is a valuable indicator of visceral adipose function, insulin sensitivity, and cardiometabolic risk (20,23,24). Another study found that higher VAI was positively associated with prehypertension and hypertension in both men and women (25).

Coronary artery calcium score (CACS), calculated from computed tomography scans, correlates closely with plaque burden and is a reliable surrogate marker of atherosclerosis (25,26). CACS measurement improves risk prediction for cardiovascular events and mortality beyond traditional risk factors and outperforms other risk markers. However, there are no studies that have examined the association of VAI with subclinical atherosclerosis as determined by CACS. Herein, we evaluate the association between VAI and CACS in apparently healthy Korean subjects in order to determine whether VAI is a reliable indicator of subclinical atherosclerosis.

Materials and methods

Study participants

This is a cross-sectional study of subjects from the Kangbuk Samsung Health Study, a large database of a medical health check-up program at the Health Promotion Center of Kangbuk Samsung Hospital, Sungkyunkwan University, Seoul, Korea. The purpose of the program is to promote the health of employees of the industrial companies through a regular health check-up and to improve early detection of existing diseases.

Of the 34,462 subjects who participated in the medical check-up program between January 2010 and December 2012, we excluded subjects with a self-reported history of ischemic heart disease (n = 419) or ischemic stroke (n = 270) and subjects who were using aspirin (n = 38) or a statin (n = 1,249). Final analyses were performed in 33,468 subjects (26,836 men and 6,632 women) who had a mean age of 41.5 years.

Participants provided written informed consent to use their medical check-up data in the research. The design, protocol, and the consent procedure were reviewed and approved by the Institutional Review Board of Kangbuk Samsung Hospital (KBS12089) and are in accordance with the Helsinki Declaration of 1975.

Anthropometric and laboratory measurements

Height and weight were measured by experienced nurses while the subjects were wearing lightweight gowns. Each participant's WC was measured at the midpoint between the top of the iliac crest and the lower border of the last palpable rib. BMI was calculated by dividing the participant's weight (kg) by the square of height (m). Blood pressure was measured using a standardized sphygmomanometer after the patient had been at rest for 5 min. Systolic blood pressure and diastolic blood pressure were measured three times with the participant in a seated position, with 1 min of rest between each measurement. The average of the second and third measurements was used in our analyses.

Blood samples were collected from the antecubital vein after an overnight fast. The hexokinase method was used to test fasting glucose concentrations (Modular D2400; Hitachi, Tokyo, Japan). Fasting insulin concentrations were determined using an electrochemiluminescence immunoassay (Modular E170; Hitachi). An enzymatic calorimetric test was used to measure total cholesterol and TG concentrations. The selective inhibition method was used to measure HDL-C levels, and a homogeneous enzymatic calorimetric test was used to measure low-density lipoprotein cholesterol (LDL-C) levels.

Lifestyle habits were assessed by self-report. A patient was classified as a smoker if he or she had smoked at least five packs of cigarettes in his or her lifetime. If a patient drank more than 20 g of alcohol daily, he or she was classified as an alcohol drinker. Having a regular exercise habit was defined as exercising with at least moderate intensity at least three times every week.

Diabetes mellitus status was determined by self-report, fasting blood glucose level, and glycated hemoglobin (HbA1c) level, according to the American Diabetes Association guidelines (27).

Definition of VAI score

The VAI was calculated using the following sex-specific formula (24):

$$\text{Males: VAI} = [\text{WC}/\{39.68 + (1.88 * \text{BMI})\}] * (\text{TG}/1.03) * (1.31/\text{HDL} - \text{C})$$

$$\text{Females: VAI} = [\text{WC}/\{36.58 + (1.89 * \text{BMI})\}] * (\text{TG}/0.81) * (1.52/\text{HDL} - \text{C})$$

Subjects were divided into tertiles according to VAI: <0.967, 0.967–1.777, >1.777.

Measurement of CACS

Multi-detector computed tomography (MDCT) for coronary calcium scoring was calculated from images generated from a 64-slice, spiral computed tomograph (GE Healthcare, Little Chalfont, Buckinghamshire, UK) and using HeartBeat-CS software (Philips, Amsterdam, The Netherlands). The 64-slice MDCT followed this protocol: 0.625 mm slice thickness, 120kVP, 800 effective mAs, and 400ms rotational speed. Severity of coronary artery calcification was assessed using the Agatston score (28). Total CACS was determined based on the sum of the individual scores of four major epicardial coronary arteries: left main, left anterior descending, left circumflex, and right coronary artery. The technicians who performed MDCT were blinded to all patient information, and CACS was automatically detected with the HeartBeat-CS software.

The presence of coronary artery calcification was defined as CACS >0 and was further subdivided by severity: 0 < CACS <100 or CACS ≥100. CACS was analyzed in natural logarithm form plus 1: ln(CACS +1).

Statistical analysis

All data were analyzed using SPSS for Windows, version 18.0 (IBM, Armonk, NY). Bivariate correlation analyses were performed to identify associations between VAI and candidate independent variables; as all the variables including CACS did not show normal distribution and were skewed, Spearman's correlation analysis was used to analyze the correlations. As for CACS, since 85.9% of the participants had CACS of zero, natural logarithmic transformation of CACS +1 was used for the bivariate correlation analyses instead of raw CACS values (29–31). Student's *t*-test was used to compare the mean values of the parameters for subjects with and without CACS >0. Comparisons of the proportion of subjects in the groups with and without CACS >0 and of the proportion of subjects with CACS >0 among the VAI tertiles were performed using the Chi-square test. Logistic regression analyses were performed with CACS >0 as the dependent variable after adjusting for age and sex in the model 1. In the model 2, the analysis was performed with adjustment for age, sex, total cholesterol, systolic blood pressure, smoking, fasting glucose and fasting insulin. In the model 3, the analysis was performed with adjustment for the variables in the model 2 plus TG and HDL-C, which are the lipid parameters for dyslipidemia. In the final model, the analysis was performed with the adjustment for the variables in the model 3 plus BMI, which is the most influential variable.

Results

Table 1 shows the general and metabolic characteristics of all study participants (n = 33,468). The mean participant age was 41.5 years, 80.2% of total participants were men, and the mean BMI was 24.3 kg/m^2. The mean VAI was 1.73 ± 1.58 and the mean CACS was 11.2 ± 72.0.

When the mean values of the metabolic parameters were compared among the groups of VAI tertiles, the subjects in the 3rd tertile of VAI was the eldest and the mostly obese (Table 1). Mean values of fasting blood glucose and HbA1c increased from 1st to 3rd tertile groups of VAI. All other metabolic parameters worsened from 1st to 3rd tertiles of VAI (Table 1). Mean CACS increased from 1st to 3rd tertile groups of VAI (Table 1).

Table 2 shows results from bivariate correlations between VAI and the metabolic values, including CACS. Subjects with CACS >0 had worse metabolic parameters compared to subjects with CACS = 0. CACS and ln(CACS +1) were weakly positively correlated with VAI ($r = 0.027$, $p < 0.001$ and $r = 0.070$, $p < 0.001$), respectively.

Table 3 shows the comparisons of the metabolic parameter values among the groups divided degree of CACS. Of all the subjects, only 14.9% had CACS >0. The mean metabolic parameters showed significant differences among the three groups, with the values in group with CACS ≥100 the highest. Mean VAI was the highest in subjects with CACS ≥100 among the groups (Figure 1). The subjects with CACS >0 were least represented in the lowest VAI tertile group (<0.967) and most represented in the highest VAI tertile group (>1.777, Figure 2).

Table 4 shows risk for CAC according to VAI tertile. In logistic regression analyses with presence of

Table 1. General characteristics of all participants and comparisons among groups with tertiles of VAI.

N = 33,468	All	1st tertile of VAI	2nd tertile of VAI	3rd tertile of VAI	p value with one-way ANOVA
Age (years)	41.5 ± 7.6	40.8 ± 7.8	41.8 ± 7.7	41.9 ± 7.3	<0.001
Sex: men (%)	26,836 (80.2)	8,200 (73.5)	8,989 (80.6)	9,647 (86.5)	<0.001
BMI (kg/m^2)	24.3 ± 3.2	22.7 ± 2.7	24.4 ± 2.9	25.8 ± 3.0	<0.001
SBP (mmHg)	113.5 ± 12.9	110.2 ± 12.6	113.5 ± 12.7	116.8 ± 12.6	<0.001
DBP (mmHg)	73.6 ± 10.1	70.8 ± 9.6	73.7 ± 10.0	76.3 ± 9.9	<0.001
Waist circumference (cm)	85.1 ± 8.7	80.3 ± 7.8	85.5 ± 7.8	89.5 ± 7.8	<0.001
FBS (mg/dL)	98.8 ± 16.8	94.9 ± 12.0	98.7 ± 15.2	102.8 ± 21.1	<0.001
HbA1c (%)	5.7 ± 0.5	5.6 ± 0.4	5.7 ± 0.5	5.8 ± 0.7	<0.001
Fasting insulin (μIU/mL)	6.3 ± 6.8	4.4 ± 9.6	6.1 ± 3.6	8.3 ± 4.9	<0.001
Total cholesterol (mg/dL)	202.5 ± 35.6	192.8 ± 32.6	202.7 ± 34.3	211.9 ± 37.3	<0.001
Triglyceride (mg/dL)	136.8 ± 91.7	71.3 ± 19.9	116.6 ± 26.1	222.6 ± 110.1	<0.001
LDL-C (mg/dL)	129.1 ± 32.4	117.2 ± 29.6	132.8 ± 31.1	137.2 ± 33.1	<0.001
HDL-C (mg/dL)	53.8 ± 13.6	65.3 ± 12.7	53.0 ± 9.2	43.2 ± 7.8	<0.001
CACS	11.2 ± 72.0	8.5 ± 64.2	10.7 ± 70.6	14.4 ± 80.2	<0.001
ln(CACS +1)	0.47 ± 1.26	0.34 ± 1.10	0.46 ± 1.25	0.60 ± 1.40	<0.001
VAI	1.73 ± 1.58	0.67 ± 0.18	1.33 ± 0.23	3.19 ± 1.99	<0.001
Smoking (%)	9,194 (27.5)	2,112 (18.9)	2,941 (26.4)	4,141 (37.1)	<0.001
Diabetes (%)	2,011 (6.0)	347 (3.1)	601 (5.4)	1,063 (9.5)	<0.001
Alcohol drinking (%)	5,792 (17.3)	1,747 (15.7)	1,856 (16.6)	2,189 (19.6)	<0.001
Physical activity (%)	6,366 (19.0)	2,475 (22.2)	2,116 (19.0)	1,775 (15.9)	<0.001

Values are presented as mean ± standard deviation or n (%).
VAI: visceral adiposity index; ANOVA: analysis of variance; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; FBS: fasting blood sugar; HbA1c: glycated hemoglobin; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; CACS: coronary artery calcium score; ln(CACS +1): natural logarithmic transformation of coronary artery calcium score plus 1.

Table 2. Bivariate correlations between metabolic parameters and visceral adiposity index.

	r	p value
Age	0.031	<0.001
BMI	0.315	<0.001
Systolic blood pressure	0.180	<0.001
Diastolic blood pressure	0.186	<0.001
Waist circumference	0.331	<0.001
Fasting blood glucose	0.196	<0.001
HbA1c	0.168	<0.001
Fasting insulin	0.219	<0.001
Total cholesterol	0.221	<0.001
Triglyceride	0.942	<0.001
LDL-C	0.08	<0.001
HDL-C	−0.550	<0.001
Coronary artery calcium score	0.027	<0.001
ln(CACS +1)	0.070	<0.001

BMI: body mass index; HbA1c: glycated hemoglobin; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; ln(CACS +1): natural logarithmic transformation of coronary artery calcium score plus 1.

CACS >0 as the dependent variable, the highest VAI tertile group (>1.777) had significantly higher odds ratio (OR) for having CAC compared to the lowest tertile group (<0.967), even after adjusting for age and sex (Model 1: odds ratio (OR) = 1.81, 95% CI: 1.669–1.972). After further adjustment for total cholesterol, systolic blood pressure, smoking, fasting blood glucose, and fasting insulin (Model 2), the OR for CAC was 1.39 (95% CI: 1.267–1.517) in the highest tertile compared to the lowest tertile. Although further adjustment, BMI showed attenuated, but still statistically significant OR for CACS >0 (Model 3) (OR = 1.26, 95% CI: 1.147–1.381). When similar analyses were performed with CACS >100 as the dependent variable, similar results were observed, although the ORs were attenuated (Table 4). When similar analyses were performed with VAI included in the models as the continuous variable, VAI showed positive correlation with the presence of CAC even after adjustment for confounding variables (Supplemental Table).

Discussion

In this study of apparently healthy Korean subjects, VAI showed weak but statistically significant correlation with CACS, and the subjects who had higher CACS had significantly higher VAI values. In logistic regression analyses, the highest VAI tertile had significantly increased odds of having CACS >0 after adjusting for confounding variables, including BMI. Although the risk was attenuated after adjustment for confounding variables, similar results were observed with CACS >100 as the dependent variable.

Visceral adiposity rather than mere obesity, which is frequently observed in patients with hypertriglyceridemia and low HDL-C (32), is known to affect the development of CVD through increased adipokine production, proinflammatory milieu, and deterioration of insulin sensitivity (9–12). This is why the assessment of visceral adiposity is important in the identification of patients at high risk for CVD. Several surrogate markers, such as BMI, WC, and dyslipidemia, have been used as the markers of visceral adiposity. The VAI, which was first developed by Amato et al., is a sex-specific scoring tool based on WC, BMI, TG, and HDL-C

Table 3. Comparison of metabolic parameters according to presence/absence of coronary artery calcification.

N = 33,468	CACS =0 (n = 28,470, 85.1%)	0 < CACS <100 (n = 4,116, 12.3%)	CACS ≥100 (n = 882, 2.6%)	p value
Age (years)	40.4 ± 6.96	46.8 ± 8.0	52.1 ± 9.6	<0.001
Sex: men (%)	22,304 (78.3)	3,755 (91.2)	798 (90.5)	<0.001
BMI (kg/m^2)	24.2 ± 3.2	25.1 ± 3.0[a]	25.3 ± 3.1[a]	<0.001
Systolic blood pressure (mmHg)	112.8 ± 12.7	117.3 ± 13.0	119.9 ± 13.5	<0.001
Diastolic blood pressure (mmHg)	73.0 ± 10.0	77.1 ± 10.1	77.9 ± 10.5	<0.001
Waist circumference (cm)	84.7 ± 8.7	87.5 ± 8.0	88.3 ± 8.1	<0.001
Fasting blood glucose (mg/dL)	97.7 ± 15.0	104.2 ± 22.8	109.1 ± 28.3	<0.001
HbA1c (%)	5.68 ± 0.5	5.90 ± 0.7	6.07 ± 0.9	<0.001
Fasting insulin (μIU/mL)	6.2 ± 7.1	6.7 ± 4.3[a]	7.0 ± 4.8[a]	<0.001
Total cholesterol (mg/dL)	201.0 ± 34.8	211.3 ± 39.2	207.5 ± 39.7	<0.001
Triglyceride (mg/dL)	132.9 ± 87.4	158.4 ± 112.9[a]	161.1 ± 100.7[a]	<0.001
LDL-C (mg/dL)	127.8 ± 31.8	137.2 ± 34.6	133.0 ± 36.1	<0.001
HDL-C (mg/dL)	54.3 ± 13.6	51.3 ± 12.7[a]	51.6 ± 13.4[a]	<0.001
Visceral adiposity index	1.68 ± 1.50	2.04 ± 1.97[a]	2.08 ± 1.67[a]	<0.001
Smoking (%)	7,596 (26.7)	1,338 (32.5)	270 (30.6)	<0.001
Diabetes (%)	1231 (4.3)	585 (14.2)	200 (22.7)	<0.001
Alcohol drinking (%)	4621 (16.2)	965 (23.4)	211 (23.9)	<0.001
Physical activity (%)	5,344 (18.8)	831 (20.2)	195 (22.1)	0.013

BMI: body mass index; HbA1c: glycated hemoglobin; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol.
[a]No differences between the two groups by post-hoc analysis.

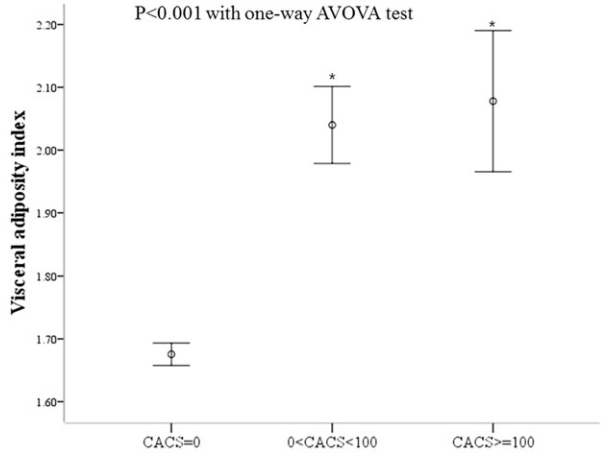

Figure 1. Comparison of mean visceral adiposity index between subjects divided by coronary artery calcium score severity.

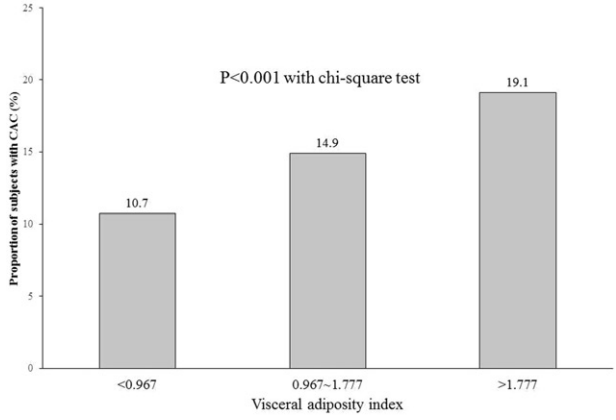

Figure 2. Comparison of the proportion of subjects with coronary artery calcium score >0 according to tertiles of visceral adiposity index.

and offers a quantitative estimate of visceral adiposity and insulin sensitivity (20). Our results partly support the reports by Amato et al. in that VAI is a novel indicator of visceral obesity, and that it has high correlation with the classic markers of cardiometabolic risk (20–22).

Herein, we found a significant association between VAI and CAC, which, to our knowledge, is the first report of this association in the literature. Furthermore, subjects with higher CACS had a significantly higher VAI compared to those without CAC. The risk of CACS >0 was higher in the upper VAI tertiles compared to the lowest tertile, even after adjusting for several factors, including BMI. These results are consistent with previous results that showed a strong association between VAI and CVD (20,24). VAI increased significantly in metabolically healthy obese individuals compared to metabolically healthy normal-weight individuals and was a novel risk factor of CVD (33). In another study, VAI increased the cardiometabolic risk of type 2 diabetes and decreased significantly after 12 months of intervention (34). VAI is also known to be a predictive marker of CKD or CV events in hemodialysis patients and patients with polycystic ovary syndrome (22,35), as well as in patients with pre-hypertension or hypertension (21). The direct mechanism for the association between VAI, which reflects visceral fat and insulin resistance, and CACS, which is a marker for subclinical atherosclerosis and reflects cardiovascular burden, could be the explained through the condition called, 'adiposopathy', which is featured by increased circulating free fatty acid, systemic inflammation and vasculopathy in subjects with abdominal obesity and insulin resistance (36).

Table 4. Odds ratios (95% CI) for coronary artery calcification according to tertiles of visceral adiposity index.

	Model 1	Model 2	Model 3
Odds ratio (95% confidence interval) for CACS >0			
VAI <0.967	1 (Ref)	1 (Ref)	1 (Ref)
0.967 ≤VAI ≤1.777	1.33 (1.216∼1.447)	1.17 (1.065∼1.273)	1.10 (1.006∼1.205)
VAI >1.777	1.81 (1.669∼1.972)	1.39 (1.267∼1.517)	1.26 (1.147∼1.381)
Odds ratio (95% confidence interval) for CACS >100			
VAI <0.967	1 (Ref)	1 (Ref)	1 (Ref)
0.967 ≤VAI ≤1.777	1.14 (0.938∼1.377)	1.01 (0.830∼1.225)	0.93 (0.765∼1.136)
VAI >1.777	1.80 (1.502∼2.149)	1.37 (1.135∼1.655)	1.20 (0.983∼1.549)

Model 1: adjusted for age and sex.
Model 2: adjusted for age, sex, total cholesterol, systolic blood pressure, smoking, fasting glucose, and fasting insulin.
Model 3: adjusted for age, sex, total cholesterol, systolic blood pressure, smoking, fasting glucose, fasting insulin, and BMI.
CI: confidence interval; VAI: visceral adiposity index; HDL-C: high-density lipoprotein cholesterol; BMI: body mass index.

Excess visceral adiposity, featured as adiposity dysfunction, increased adipocytokine production and proinflammatory activity that might cause the insulin resistance, and atherogenic dyslipidemia with high TG and low HDL-C, could be the main reason for the link between pathologic adipose tissue and cardiometabolic risk in human body (37). There are many surrogate markers or methods to measure visceral adiposity in vivo (38). In several studies, visceral obesity, as assessed by CT scan, correlated well with diabetogenic, atherogenic, prothrombotic, and proinflammatory metabolic abnormalities—also known as metabolic syndrome; metabolic syndrome is also known to be associated with increased risk of CVD (9–12,39). However, CT and MRI imaging are not frequently used to measure visceral fat because they are not cost-effective and the measurement protocols are complex. As the calculation for VAI contains TG and WC, the main components of visceral obesity and insulin resistance, VAI could be considered as an accurate marker for cardiometabolic risk. However, further studies are needed to evaluate the effectiveness of newly developed surrogate markers.

This study has some limitations. First, it is a cross-sectional study; thus, we cannot imply causality between VAI and CACS. Second, subjects of this study were all Korean, so we cannot generalize the results to the general population. Third, there is no appropriate VAI cut-off value to estimate cardiovascular risk; we used tertile values and compared CACS or the proportion of subjects with CACS >0. Fourth, there was gender imbalance in our study population. Fifth, the correlation coefficient between absolute CACS value and VAI was 0.027, somewhat weak to interpret as a significant correlation. In a large sample, almost any correlation coefficient could be significantly different from zero. Lastly, failure to consider the menopausal status of the subjects, other disease status such as hypertension, the lack of adjustment for diet pattern and socioeconomic status could weaken the plausibility of the results. However, this is first large study to explore the relationship between VAI and subclinical atherosclerosis using CACS. Therefore, despite these limitations, our exploratory study is valuable.

In conclusion, VAI had a significantly positive association with subclinical atherosclerosis, as assessed by CACS. Furthermore, VAI was an independent risk factor of CAC. Our results suggest that VAI could be a useful clinical marker of cardiometabolic risk, at least in the Korean population.

Disclosure statement

The authors report no conflicts of interest.

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