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Association between cardiometabolic index and abdominal aortic calcification in US adults from the NHANES

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Abdominal aortic calcification (AAC) and atherosclerosis are prevalent conditions among older adults, and recent research suggests that their association may extend beyond the effects of aging alone. An essential instrument for determining the possibility of cardiovascular disease (CVD) is the cardiometabolic index (CMI), a new lipid-based index sensitive to visceral obesity. However, little has been established about the relationship between CMI and AAC. We examined CMI and AAC data from the National Health and Nutrition Examination Survey (NHANES) conducted in 2013–2014 for this study. The relationship between AAC, severe abdominal aortic calcification (SAAC), and CMI was assessed using multiple linear and logistic regression models. The overall trend was visualized using smoothed curve modeling. Subgroup analyses were conducted to find possible moderating factors. Among the 2704 participants included, those with higher CMI levels exhibited much greater AAC scores and a higher prevalence of SAAC. In model 3, elevated CMI positively correlated with AAC (0.25 (0.09, 0.41)) and with the odds of SAAC (OR = 1.35 (1.09, 1.67)). Participants in the highest CMI quartile had an AAC score that was 0.65 units higher (β = 0.65 (0.26, 1.04)) and an 114% higher risk of SAAC (OR = 2.14 (1.29, 3.54)). Subgroup analyses indicated sex and smoking status significantly modified the relationship between CMI, AAC, and SAAC, while previously diagnosed with congestive heart failure (CHF) and heart attack significantly moderated the association between CMI and AAC. These results imply that greater AAC scores and a higher risk of SAAC are linked to heightened CMI, which represents visceral fat storage and disturbed lipid metabolism. Our findings indicate that CMI is correlated with AAC in certain demographic and cardiovascular subgroups, suggesting its potential as an exploratory indicator of elevated AAC risk in these populations.

Keywords Cardiometabolic index, Abdominal aortic calcification, Cross-sectional study, NHANES

Abbreviations

AAC	Abdominal aortic calcification
SAAC	Severe abdominal aortic calcification
CMI	Cardiometabolic index
NHANES	National Health and Nutrition Examination Survey
CVD	Cardiovascular disease
CAC	Coronary artery calcification
CHD	Coronary heart disease
WHtR	Waist-to-height ratio
TG	Triglyceride
HDL	High-density lipoprotein
TC	Total cholesterol
PIR	Poverty-to-income ratio
BMI	Body mass index
CHF	Congestive heart failure

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An abnormal buildup of crystallization of calcium within the intima of the artery is a hallmark of vascular calcification[1]. This accumulation in the arterial wall might constrict the aorta's lumen and make it more stiff. Furthermore, lipid-rich deposits build up and solidify along the vessel walls to create atherosclerotic plaques, which are facilitated by calcium deposits. Plaque calcification over time may impede blood flow, increasing the risk of cardiovascular disease (CVD)[2,3]. AAC, a common manifestation of vascular calcification[4], develops earlier than coronary artery calcification (CAC) and may serve as a sign of vascular morbidity and mortality. Research indicates that AAC can independently predict subclinical CVD and anticipated cardiovascular risks, unrelated to traditional risk factors[5–7]. The cardiovascular consequences of AAC and CAC were examined by the Multi-Ethnic Study of Atherosclerosis (MESA). Only AAC was found to be significantly linked to CVD mortality, despite the fact that both CAC and AAC were independent predictors of CVD and coronary heart disease (CHD). Additionally, AAC outperformed CAC as a predictor of all-cause death[8]. Consequently, reducing the incidence and prevalence of CVD requires effective handling and the avoidance of AAC[9].

Globally, Obesity is now a significant public health challenge[10]. As reported by studies, 11.1% of mortality caused by non-infectious diseases is attributable to overweight or obesity[11,12]. Despite being widely used to evaluate the CVD risk linked to obesity, the amount and location of body fat are not properly described by body mass index (BMI), and BMI does not take into consideration changes in hydration status and fails to discern whether weight changes are due to preserved or increased lean mass or elevated fat mass[13–15]. In CHF, visceral fat deposits are acknowledged as a more robust indicator of death risk than BMI-defined obesity[14,16]. The CMI has been used to screen for both obesity and diabetes. It incorporates obesity indicators, such as the triglyceride-to-HDL cholesterol ratio and the waist-to-height ratio (WHtR), to evaluate visceral obesity[17]. Non-alcoholic fatty liver disease (NAFLD), hyperuricemia, chronic kidney disease (CKD), CVD, and stroke are all linked to CMI[18–22].

No clinical research has been performed to evaluate the correlation between CMI and AAC. We tested the hypothesis that an elevated CMI is positively connected with the likelihood of having AAC by using 2013–2014 NHANES data in order to close this gap.

Methods

Survey description and study population

The Centers for Disease Control and Prevention (CDC) administers the National Health and Nutrition Examination Survey (NHANES), A comprehensive survey program designed to assess the health status of the American population. The research began with an enrollment of 10,175 individuals. Following the removal of individuals lacking AAC scores (n = 7,035) and CMI components (total: n = 179; waist circumference: n = 39; standing height: n = 22; HDL: n = 104; TG: n = 14), as well as participants with incomplete covariate information (n = 257)—covering variables such as poverty-to-income ratio (PIR) (n = 235), BMI (n = 2), education level (n = 2), marital condition (n = 1), smoking (n = 1), CHF (n = 5), angina pectoris (n = 5), heart attack (n = 3), CHD (n = 1), and stroke (n = 2)—the final analytical sample comprised 2,704 participants. A flowchart was provided in Fig. 1.

Definition of CMI and AAC

The CMI is calculated using the WHtR and participants' lipid profiles. The formula is as follows:

$$\left(\frac{\mathrm{TG(mg/dL)}}{\mathrm{HDL\ -\ C(mg/dL)}}\right) \times \mathrm{WHtR}$$

$$\mathrm{WHtR} = \frac{\text{waist circumference (cm)}}{\text{height (cm)}}$$

The AAC score, which was measured using the Kauppila scoring method, was used to evaluate the severity of AAC. Using the Densitometer Discovery A (Hologic, Marlborough, MA, USA), lateral lumbar spine images acquired by dual-energy X-ray absorptiometry (DXA) were analyzed. More severe calcification is indicated by higher total AAC scores, which range from 0 to 24. According to earlier research, an AAC score of more than 6 was considered SAAC, which is a widely accepted cutoff point for detecting substantial aortic calcification.

The research designated CMI as the primary exposure variable, whereas the AAC score and SAAC were analyzed as outcome variables.

Covariates

Demographics, economic position, metabolic markers, and comorbidities were deemed confounding factors based on their clinical importance. Among the covariates evaluated in this study are age, sex, race, BMI, PIR, educational background, smoking status, marital condition, standing height, waist circumference, estimated glomerular filtration rate (eGFR), serum phosphorus, serum calcium, Serum Creatinine (Scr), total cholesterol (TC), high-density lipoprotein (HDL), and triglycerides (TG) were significant factors. Additionally, we accounted for the presence of comorbid conditions, including hypertension, diabetes, Chronic kidney disease (CKD), CHF, angina pectoris, heart attack, CHD, and stroke. Supplementary Table 1 contains information on the covariates, while the measurement protocols are publicly accessible through www.cdc.gov/nchs/nhanes/.

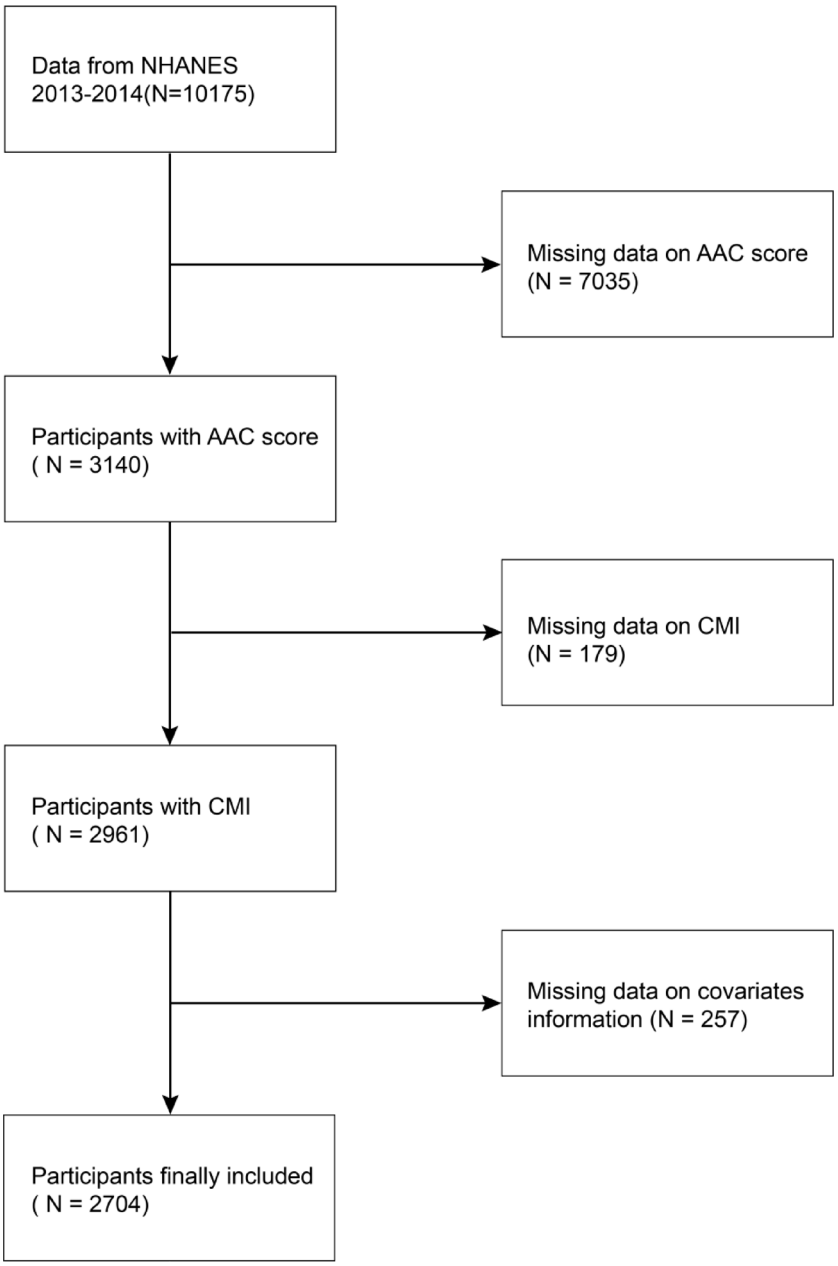

Fig. 1. Flowchart of participants included in this study.

Statistical analysis

This analysis utilized R software alongside EmpowerStats (http://www.empowerstats.com), with a two-sided *P*-value < 0.05 for statistical significance.

Mean ± standard deviation (SD) was employed to describe continuous variables, while proportions were used for categorical variables. The CMI data were normalized using a log2 transformation to guarantee an equitable distribution. Differences across quartiles of the CMI for continuous variables were assessed using Student's t-tests, and chi-square tests were employed for categorical variables. To investigate the relationship between CMI and AAC, multivariable linear regression models were used and logistic regression models were employed for the analysis of CMI and SAAC. The model 1 was unadjusted, the model 2 included adjustments for sex, age, and race, and the model 3 accounted for sex, age, race, educational background, marital condition, PIR, BMI, smoking status, total cholesterol, serum phosphorus, serum calcium, CKD, hypertension, diabetes, CHF, angina pectoris, CHD, heart attack, and stroke. Trend tests were conducted to evaluate associations across CMI quartiles. Subgroup analyses assessed the relationships between CMI, AAC, and SAAC by stratifying participants according to sex, diabetes status, smoking status, CKD, hypertension, angina, heart attack, CHD, stroke, or CHF. Interaction tests examined associations within these subgroups, with covariates from model 3 included in the analyses. Additionally, to visualize the trends, smoothed curve fitting was conducted.

Results

Baseline characteristics of participants

This analysis incorporated data from 2,704 participants, with a median age of 58.52 ± 11.99 years. The sample comprised 48.56% male and 51.44% female participants. The racial and ethnic distribution included 45.71% non-Hispanic white. The mean ± SD values for the CMI and AAC across all participants were 0.97 ± 1.35 and 1.63 ± 3.51, respectively. Clinical characteristics by CMI quartiles are detailed in Table 1. Mexican American men were overrepresented in the highest CMI quartile. Additionally, individuals with elevated CMI tended to have lower education and income, higher smoking prevalence, increased BMI and waist circumference, and elevated levels of TC, and TG. This group also had a higher risk of diabetes, hypertension, CHD, and CHF, alongside lower HDL-C levels.

The association between CMI and AAC

Table 2 shows that higher CMI scores are linked to greater AAC scores and an increased likelihood of SAAC. CMI and AAC scores showed a positive correlation in model 3 (β = 0.25 (0.09, 0.41)), indicating that each one-unit increase in CMI corresponds to a 0.25 unit increase in AAC scores. The study authors stratified CMI into quartiles, revealing a significant relationship between the highest quartile (Q4) and AAC (β = 0.65 (0.26, 1.04), *P* for trend = 0.001). For SAAC, after full adjustment, a one-unit increase in CMI resulted in a 35% elevated risk (OR = 1.35 (1.09, 1.67)). The significant association was also evident in quartile-based analyses, where participants in the highest CMI quartile showed an 114% heightened risk of SAAC than those in the reference quartile (OR = 2.14 (1.29, 3.54), *P* for trend = 0.005).

In Fig. 2, further analysis utilizing smoothed curve fitting identified a positive correlation of CMI with both AAC and SAAC.

Subgroup analysis

To determine whether the connections between CMI, AAC, and SAAC differ across various population subcategories, Subgroup analyses were carried out with stratification by sex, smoking status, diabetes, hypertension, CHF, angina pectoris, heart attack, CHD, and stroke. The results indicated significant variations in these associations across subgroups. The findings highlight significant interaction effects between AAC and sex, smoking status, and CHD (*P* for interaction < 0.05). For SAAC, significant interactions were identified with sex and smoking status (*P* for interaction < 0.05). Specifically, in the female subgroup, CMI had a stronger positive association with AAC and SAAC. In never smokers, this association was also more pronounced relative to current smokers. Additionally, among patients without CHF and those without a history of heart attack, the positive relationship between CMI and AAC was more significant. The results indicate that the influence of CMI on AAC and varies across demographic factors, including sex, smoking status, and cardiovascular conditions, as illustrated in Fig. 3.

Discussion

The purpose of this research was to examine, within a population-based sample, the association between CMI and AAC. Within the sample of 2704 participants, both a significant positive correlation between CMI and AAC and a significant trend across higher CMI strata were observed. Subgroup analyses indicated sex, smoking status, CHF, and history of heart attack modified this association. His study identifies a robust positive correlation between CMI and AAC, which warrants further investigation to determine if CMI management might have implications for AAC risk.

In 2015, Wakabayashi et al. initially introduced the CMI, as a predictive marker of diabetes and CVD risk[17]. According to Qiu et al., people with higher levels of CMI demonstrated a markedly higher propensity for early development of diabetes (HR = 1.78), while participants transitioning from lower to higher CMI ranges during the observation period exhibited a 75% increase in diabetes risk[23]. Luo et al. discovered that both CMI and CMI-age were independently linked to elevated CVD risk in a study involving 9,008 participants, highlighting the necessity of keeping an eye on people with high CMI[16]. Xu et al. performed a cohort study demonstrating a positive link between CMI and total mortality among older individuals, with inflammation playing a significant mediating role[24]. Among those suffering from concurrent obstructive sleep apnea and hypertension, Cai et al. conducted a positive correlation between CVD risk and CMI[21]. Similarly, Higashiyama et al. revealed that those without metabolic syndrome (MetS) but with greater CMI levels had an increased risk of ischemic CVD[25]. These findings underscore the strong association between CMI and various systemic diseases and its connection to poor prognosis. At present, no studies have evaluated the potential link between CMI and AAC, particularly in elderly persons who are more likely to have comorbidities.

Additional anthropometric and metabolic measures, including the triglyceride glucose (TyG) index, BRI, WWI, and VAI, have also been validated as associated with AAC or cardiovascular mortality. Chen et al. found that in a representative sample of American adults, the TyG index was meaningfully linked to extensive AAC (OR = 1.41), regardless of additional variables[26]. Chen et al. further elucidated a positive relationship between BRI and AAC (OR = 1.22)[27]. In their 20-year longitudinal study on BRI trends among American adults, Zhang et al. reported a U-shaped association with mortality risk, with increased risks observed at both ends of the BRI spectrum relative to median values[28]. According to Qin et al., WWI had a nonlinearly positive relationship with SAAC, and greater AAC scores were linked to higher WWI levels[29]. Greater VAI levels were linked to greater AAC (β = 0.04) and a higher chance of SAAC (OR = 1.04), according to Chen et al.[30]. Moreover, the research by Chen et al. found that elevated VAI levels were linked to a greater risk of composite cardiovascular events and overall death rates in a prospective research involving 464 common hemodialysis patients[31]. In line with earlier research on the cardiovascular implications of other obesity indicators, this study observed a positive

Characteristics	log2-transformed CMI				
	Q1	Q2	Q3	Q4	
	N = 676	N = 676	N = 676	N = 676	*P*-value
Gender, n (%)					< 0.001
Male	269 (39.79%)	313 (46.30%)	335 (49.56%)	396 (58.58%)	
Female	407 (60.21%)	363 (53.70%)	341 (50.44%)	280 (41.42%)	
Age, years	57.95 ± 12.38	58.91 ± 12.02	59.47 ± 11.95	57.77 ± 11.55	0.028
Race, n (%)					< 0.001
Mexican American	41 (6.07%)	81 (11.98%)	95 (14.05%)	115 (17.01%)	
Other Hispanic	47 (6.95%)	56 (8.28%)	77 (11.39%)	69 (10.21%)	
Non-Hispanic white	300 (44.38%)	294 (43.49%)	306 (45.27%)	336 (49.70%)	
Non-Hispanic black	198 (29.29%)	149 (22.04%)	116 (17.16%)	55 (8.14%)	
Other race	90 (13.31%)	96 (14.20%)	82 (12.13%)	101 (14.94%)	
Education level, n (%)					< 0.001
Below high school	127 (18.79%)	136 (20.12%)	150 (22.19%)	170 (25.15%)	
High school	134 (19.82%)	148 (21.89%)	154 (22.78%)	177 (26.18%)	
Above high school	415 (61.39%)	392 (57.99%)	372 (55.03%)	329 (48.67%)	
Marital status, n (%)					0.543
Married/living with partner	416 (61.54%)	428 (63.31%)	443 (65.53%)	438 (64.79%)	
Widowed/divorced/separated	204 (30.18%)	187 (27.66%)	189 (27.96%)	186 (27.51%)	
Never married	56 (8.28%)	61 (9.02%)	44 (6.51%)	52 (7.69%)	
Family PIR					< 0.001
< 1.3	168 (24.85%)	181 (26.78%)	213 (31.51%)	238 (35.21%)	
> = 1.3, < 3.5	227 (33.58%)	242 (35.80%)	230 (34.02%)	241 (35.65%)	
> = 3.5	281 (41.57%)	253 (37.43%)	233 (34.47%)	197 (29.14%)	
BMI, kg/m^2	25.34 ± 4.84	27.89 ± 5.32	29.79 ± 5.39	30.92 ± 5.17	< 0.001
BMI (kg/m^2), n (%)					< 0.001
< 25	361 (53.40%)	208 (30.77%)	127 (18.79%)	69 (10.21%)	
> = 25, < 30	209 (30.92%)	267 (39.50%)	249 (36.83%)	256 (37.87%)	
> = 30	106 (15.68%)	201 (29.73%)	300 (44.38%)	351 (51.92%)	
Smoking status, n (%)					0.003
Never	396 (58.58%)	371 (54.88%)	341 (50.44%)	329 (48.67%)	
Former	159 (23.52%)	192 (28.40%)	211 (31.21%)	203 (30.03%)	
Now	121 (17.90%)	113 (16.72%)	124 (18.34%)	144 (21.30%)	
Waist circumference, cm	90.38 ± 12.06	97.48 ± 12.41	102.94 ± 12.33	106.86 ± 12.42	< 0.001
Standing Height, cm	166.46 ± 9.37	166.28 ± 10.14	166.37 ± 10.31	167.31 ± 10.35	0.204
HDL, mg/dL	1.85 ± 0.45	1.46 ± 0.28	1.26 ± 0.24	1.02 ± 0.21	< 0.01
TC, mg/dL	4.90 ± 0.97	4.92 ± 1.03	5.05 ± 1.16	5.26 ± 1.20	< 0.001
TG, mg/dL	0.76 ± 0.22	1.20 ± 0.29	1.78 ± 0.45	3.49 ± 2.91	< 0.001
Serum phosphorus, mmol/L	1.23 ± 0.17	1.22 ± 0.17	1.23 ± 0.19	1.23 ± 0.20	0.813
Serum calcium, mmol/L	2.36 ± 0.09	2.36 ± 0.09	2.36 ± 0.09	2.37 ± 0.10	0.572
Scr, umol/L	80.82 ± 42.27	83.11 ± 62.07	85.05 ± 42.16	86.28 ± 39.43	0.162
eGFR, mL/min/1.73 m^2	84.89 ± 21.70	84.44 ± 21.81	82.21 ± 22.21	84.46 ± 24.53	0.121
CKD, n (%)					0.067
Yes	593 (87.72%)	600 (88.76%)	569 (84.17%)	581 (85.95%)	
No	83 (12.28%)	76 (11.24%)	107 (15.83%)	95 (14.05%)	
Diabetes, n (%)					< 0.001
Yes	79 (11.69%)	134 (19.82%)	167 (24.70%)	249 (36.83%)	
No	597 (88.31%)	542 (80.18%)	509 (75.30%)	427 (63.17%)	
Hypertension, n (%)					< 0.001
Yes	391 (57.84%)	407 (60.21%)	464 (68.64%)	469 (69.38%)	
No	285 (42.16%)	269 (39.79%)	212 (31.36%)	207 (30.62%)	
CHF, n (%)					0.048
Yes	12 (1.78%)	28 (4.14%)	28 (4.14%)	26 (3.85%)	
No	664 (98.22%)	648 (95.86%)	648 (95.86%)	650 (96.15%)	
CHD, n (%)					0.046
Yes	24 (3.55%)	35 (5.18%)	34 (5.03%)	47 (6.95%)	

Continued

Characteristics	log2-transformed CMI				
	Q1	Q2	Q3	Q4	
	N = 676	N = 676	N = 676	N = 676	*P*-value
No	652 (96.45%)	641 (94.82%)	642 (94.97%)	629 (93.05%)	
Angina, n (%)					0.219
Yes	14 (2.07%)	27 (3.99%)	20 (2.96%)	23 (3.40%)	
No	662 (97.93%)	649 (96.01%)	656 (97.04%)	653 (96.60%)	
Heart attack, n (%)					0.217
Yes	26 (3.85%)	37 (5.47%)	37 (5.47%)	43 (6.36%)	
No	650 (96.15%)	639 (94.53%)	639 (94.53%)	633 (93.64%)	
Stroke, n (%)					0.991
Yes	31 (4.59%)	29 (4.29%)	29 (4.29%)	29 (4.29%)	
No	645 (95.41%)	647 (95.71%)	647 (95.71%)	647 (95.71%)	

Table 1. Baseline characteristics of the study participants. Data are presented as mean ± SD or n (%). CMI, cardiometabolic index; PIR, poverty-to-income ratio; BMI, body mass index; TG, triglyceride; HDL, high-density lipoprotein; TC, total cholesterol; CHD, coronary heart disease; CHF, congestive heart failure.

	AAC		SAAC	
	B (95% CI)	*P* value	OR (95% CI)	*P* value
Model 1				
log2-transformed CMI	0.18 (0.03, 0.33)	0.022	1.15 (0.99, 1.34)	0.060
log2-transformed CMI quartile				
Q1	0 (Reference)		1 (Reference)	
Q2	0.27 (−0.11, 0.64)	0.163	1.22 (0.82, 1.82)	0.316
Q3	0.40 (0.03, 0.77)	0.036	1.32 (0.89, 1.94)	0.168
Q4	0.51 (0.13, 0.88)	0.008	1.55 (1.06, 2.26)	0.024
P for trend		0.006		0.023
Model 2				
log2-transformed CMI	0.17 (0.03, 0.32)	0.018	1.25 (1.05, 1.49)	0.014
log2-transformed CMI quartile				
Q1	0(Reference)		1(Reference)	
Q2	0.16 (−0.18, 0.51)	0.348	1.17 (0.76, 1.80)	0.465
Q3	0.23 (−0.12, 0.58)	0.192	1.21 (0.79, 1.86)	0.371
Q4	0.49 (0.14, 0.85)	0.006	1.81 (1.18, 2.76)	0.006
P for trend		0.007		0.007
Model 3				
log2-transformed CMI	0.25 (0.09, 0.41)	0.003	1.35 (1.09, 1.67)	0.006
log2-transformed CMI quartile				
Q1	0(Reference)		1(Reference)	
Q2	0.33 (−0.01, 0.67)	0.057	1.47 (0.92, 2.34)	0.109
Q3	0.42 (0.07, 0.78)	0.021	1.55 (0.96, 2.50)	0.073
Q4	0.65 (0.26, 1.04)	0.001	2.14 (1.29, 3.54)	0.003
P for trend		0.001		0.005

Table 2. Association between log2-transformed cardiometabolic index and abdominal aortic calcification. AAC, Abdominal aortic calcification; SAAC, severe abdominal aortic calcification; CI, confidence interval; CMI, cardiometabolic index. Model 1: no covariates were adjusted. Model 2: adjusted for gender, age, race. Model 3: adjusted for gender, age, race, education level, marital status, PIR, BMI, smoking status, total cholesterol, serum phosphorus, serum calcium, CKD, hypertension, diabetes, heart failure, angina pectoris, CHD, heart attack, and stroke.

relationship between CMI and AAC and found that CMI might be useful in cross-sectional assessments of AAC risk.

Elevated CMI, which reflects visceral obesity and metabolic abnormalities, is linked to Elevated triglyceride levels accompanied by reduced HDL cholesterol concentrations. These metabolic disturbances are key risk factors for atherosclerosis and vascular calcification[32,33]. Lipid-loaded vascular wall lesions are a hallmark of

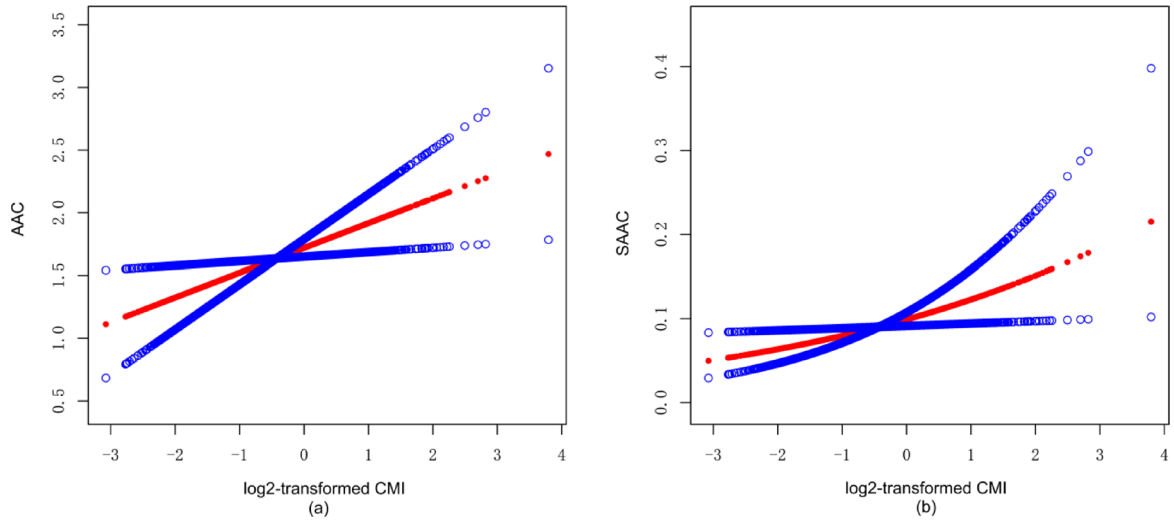

Fig. 2. Smooth Curve Fitting Detected the Linear relationship between log2-transformed CMI and AAC score by the Model 3 (**a**); the Linear relationship between log2-transformed CMI and SAAC by the Model 3 (**b**).

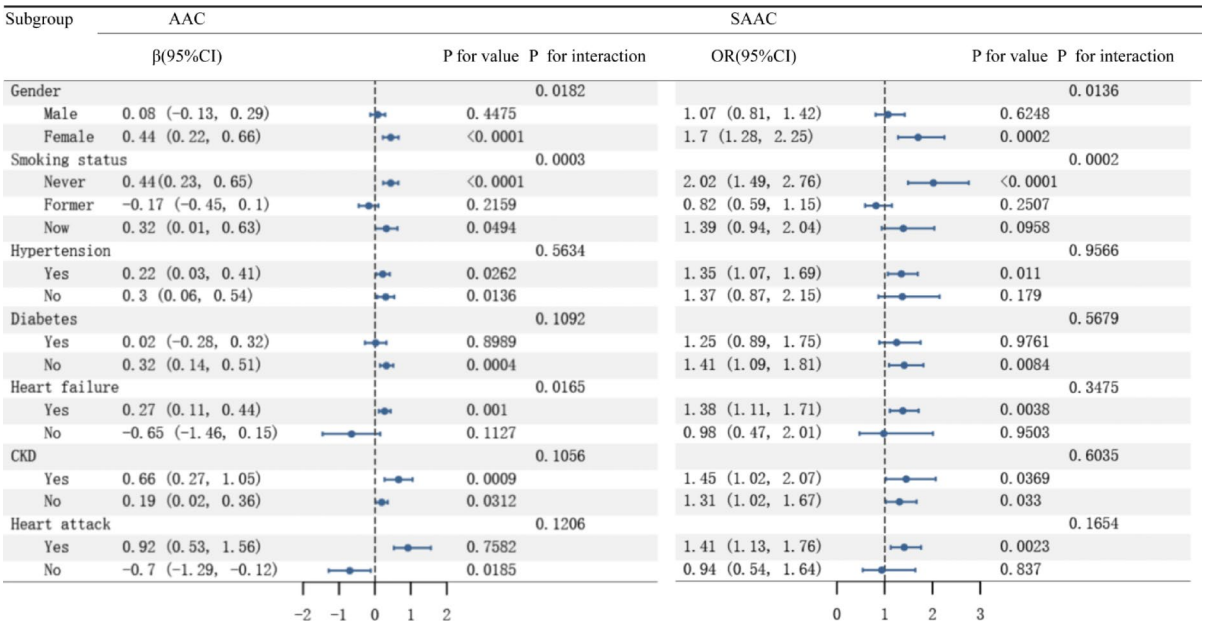

Fig. 3. Results of subgroup analysis and interaction analysis for log2-transformed CMI and AAC.

atherosclerosis. Chronic inflammation brought on by lipid buildup in the arterial wall activates the vascular endothelium and adhesion molecules are released in significant quantities, attracting circulating monocytes to the endothelium and promoting their maturation into macrophages. Macrophages that are laden with lipids then migrate into foam cells[34]. Foam cell apoptosis speeds up the establishment of a necrotic core and encourages the growth of lesions[35]. One of the main causes of atherosclerosis is the dysregulation of macrophage phenotypes. High-plasticity macrophages polarize to the pro-inflammatory M1 type and release a lot of inflammatory factors, including IL-6 and TNF-α, further activating immune cells within arterial walls, making plaques more prone to rupture and increasing the risk of cardiovascular events[36–39]. As atherosclerosis advances, the prolonged inflammatory environment and cell death within plaques promote vascular calcification[40–43]. Vascular calcification is fundamentally characterized by the transdifferentiation of vascular smooth muscle cells (VSMCs) from a mesenchymal lineage into osteoblast-like cells, constituting a central pathological mechanism[44]. The presence of arterial calcific plaques induces biomechanical stress through compromised vascular compliance, resulting in sustained mechanical strain that promotes both proliferative and osteogenic differentiation of VSMCs[45]. Transcriptomic analyses reveal partial activation of osteogenic transcriptional programs in phenotypically modulated VSMCs, though the magnitude of osteoblast-specific mRNA expression remains

markedly reduced compared to terminally differentiated osteoblasts[46]. Pathological elevations in extracellular calcium-phosphate concentrations synergistically potentiate vascular mineralization through dual mechanisms: direct physicochemical precipitation and induction of VSMC phenotypic reprogramming via upregulation of osteogenic differentiation-associated proteins[47,48]. This calcification cascade is primarily mediated by the canonical Wnt/β-catenin signaling cascade, with RUNX2 emerging as the principal transcriptional regulator orchestrating VSMC osteochondrogenic transformation, osteoblastic differentiation, and subsequent initiation of ectopic mineralization processes[49,50]. These processes work together to drive calcification, inflammation, and stress from oxidation in the arterial intima.[51,52].

This research is affected by several limitations. The primary limitation is that the cross-sectional design employed in this analysis precludes causal inferences between CMI and AAC. Therefore, future prospective research is necessary to validate these results. Second, because of restrictions within the NHANES database, not all relevant covariates that may affect BRI and AAC were included. These excluded variables may have predictive value and merit further investigation. Third, AAC-related data are only available in the 2013–2014 NHANES cycle, which restricts the sample size and may limit the generalizability of our results. Lastly, NHANES data are representative of the U.S. population and include only individuals aged 40 and older (due to AAC measurement constraints). To confirm the broader applicability of these findings, additional research should focus on diverse countries and younger populations.

Conclusion

In this cross-sectional NHANES study, Our results indicate a significant positive relationship between CMI and AAC, highlighting the potential of CMI as a screening indicator to detect individuals with a higher risk for vascular calcification, particularly in populations with metabolic risk factors. Future research should focus on investigating the impact of CMI management on AAC and related cardiovascular risks, providing valuable insights for clinical and public health strategies.

Data availability

Publicly available datasets were analyzed in this study. This data can be found here: The National Health and Nutrition Examination Survey dataset at www.cdc.gov/nchs/nhanes/.

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