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Original Article

Association between coronary artery calcification and cardiometabolic, hepatic, and renal markers in a Chinese population undergoing health examinations

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Abstract: Objectives: To investigate the prevalence of coronary artery calcification (CAC) in a Chinese population undergoinghealth examinations and to evaluate the associations of cardiometabolic, hepatic, and renal markers with CAC positivity and CAC burden. Methods: This single-center cross-sectional study included 2,957 Chinese adults who underwent routine health examinations and coronary artery calcium score (CACS) assessment at the Health Management Center of Shanghai Electric Power Hospital between January 1 and December 31, 2025. CAC positivity was defined as CACS > 0. Univariable and multivariable logistic regression analyses were used to identify factors associated with CAC positivity. CAC burden was further evaluated using ordinal logistic regression, linear regression for ln(CACS+1), and logistic regression for CACS ≥ 100. Results: Among the 2,957 participants, 737 (24.9%) were CAC-positive. In multivariable analyses, older age (adjusted OR=1.133, 95% CI: 1.113-1.153, P < 0.001), male sex (adjusted OR=7.990, 95% CI: 4.381-14.573, P < 0.001), higher body mass index (BMI) (adjusted OR=1.077, 95% CI: 1.036-1.119, P < 0.001), and higher HbA1c levels (adjusted OR=1.338, 95% CI: 1.202-1.491, P < 0.001) were independently associated with CAC positivity. These associations were consistent in sensitivity analyses and across CAC burden models. Conclusion: CAC is not unusual in the Chinese population undergoing health examinations. Old age, male sex, higher BMI, and higher HbA1c levels were consistently associated with both CAC positivity and higher CAC burden. Age, adiposity, and continuing glycemic exposure are among the routine clinical indicators that may help identify asymptomatic individuals at a high risk of CAC and support additional risk stratification.

Keywords: Coronary artery calcification, health check-up population, metabolic markers, hepatic markers, renal markers

Introduction

Coronary artery disease (CAD) is one of the major clinical manifestations of cardiovascular disease, which continues to be the leading cause of mortality and disease burden worldwide [1, 2]. Cardiovascular disease remains the primary cause of death in China. The prevalence of CAD among adults reached 758 per 100,000 in 2021, and the crude mortality of cardiovascular disease in urban China was 305.39 per 100,000, underscoring its substantial burden [3].

CAD is primarily driven by atherosclerosis, a chronic process that involves endothelial dysfunction, lipid retention, inflammation, plaque formation, and subsequent calcification [4, 5]. Traditional and metabolic risk factors, including age, sex, obesity, dysglycemia, dyslipidemia, and renal-metabolic dysfunction, contribute to CAD development [4, 6]. For primary prevention and cardiovascular risk stratification, early identification of high-risk individuals in asymptomatic or health examination groups is crucial.

Coronary artery calcification (CAC) is a direct imaging marker of coronary atherosclerotic burden and a key indicator of subclinical coronary atherosclerosis [7]. CAC reflects the calcified component of atherosclerotic plaque and pro-

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vides a quantitative estimate of the cumulative coronary atherosclerotic burden [7]. The coronary artery calcium score (CACS), which is calculated using the Agatston method, is the most popular and validated CAC measure in clinical practice. It has been widely used for cardiovascular risk stratification in asymptomatic adults [7-10].

Metabolic syndrome and dysglycemia are associated with both CAC prevalence and calcification burden [11]. In Chinese populations, high hemoglobin A1c (HbA1c) levels have been associated with a greater likelihood of CAC [12]. Furthermore, hepatic and renal dysfunction have been related to CAC, and the severity of liver fibrosis and chronic kidney diseases may be associated with CAC progression or a substantial coronary atherosclerotic burden [13, 14]. Nevertheless, there remains a lack of information that integrates cardiometabolic, hepatic, and renal indicators to evaluate both CAC positivity and burden in Chinese health examination populations.

Therefore, the purpose of this study was to investigate CAC prevalence and its burden distribution, as determined by CACS, in a Chinese population undergoing health examinations. Moreover, the study aimed to analyze the associations between cardiometabolic, hepatic, and renal markers and both CAC positivity and its burden.

Methods

Study design and population

This single-center cross-sectional study was conducted at the Health Management Center of Shanghai Electric Power Hospital, Shanghai, China. The study population consisted of Chinese adults who underwent routine health examinations and CACS assessments between January 01, 2025, and December 31, 2025.

Eligibility criteria

The inclusion criteria were as follows: (1) Participation in routine health examinations at the Health Management Center during the study; (2) Completing a coronary artery calcium assessment with an available CACS; (3) Availability of anthropometric measurements, such as height, weight, and body mass index (BMI), as well as core laboratory measurements, such as cardiometabolic, hepatic, and renal markers; and (4) Age ≥ 18 years.

The exclusion criteria were as follows: (1) Missing or uninterpretable CACS; (2) Missing key research variables, implausible measures, or unverifiable measurements; and (3) Duplicate examination records, of which only the first or most complete record was retained.

Data collection and variable definitions

Demographic, anthropometric, cardiometabolic, hepatic, and renal markers: Demographic and anthropometric data, including age, sex, height, weight, and BMI, were extracted from the health examination database. Laboratory variables were classified as cardiometabolic, hepatic, and renal markers. Cardiometabolic markers included fasting plasma glucose (FPG), HbA1c, triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and uric acid. Hepatic markers consisted of alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP). Renal markers comprised urea, creatinine, and estimated glomerular filtration rate (eGFR).

CAC-related measures and definitions: Non-contrast electrocardiogram-gated cardiac computed tomography (CT) or standard non-contrast chest CT was used to measure CAC. Automated or semi-automatic image processing using the Agatston method was used to determine CACS. A weighting factor of 1, 2, 3, or 4 was allocated to each calcified lesion based on the peak attenuation value of 130 to 199, 200 to 299, 300 to 399, or ≥ 400 Hounsfield units, respectively. The score of each lesion was calculated by multiplying the calcified area by the corresponding weighting factor, and the total CACS was obtained by summing the scores of all calcified lesions.

CACS is classified as 0, 1 to 99, 100 to 399, and ≥ 400. Here, CAC positivity was defined as CACS > 0. CACS was categorized as 0, 1 to 99, and ≥ 100 for burden analysis. A CACS ≥ 100 suggested a high CAC burden. As a continuous measure of CAC burden, ln(CACS+1) was used because CACS displayed a right-

skewed distribution with a high percentage of zero values.

Statistical analysis

Continuous variables are shown as median (interquartile range), and categorical variables as n (%). The Mann-Whitney U test for continuous variables and the chi-square test for categorical variables were used to compare participants with CACS=0 and CACS > 0.

Crude associations between candidate variables and CAC positivity (CACS > 0) were assessed using univariate logistic regression. Next, multivariable logistic regression was conducted to detect factors that were independently associated with CAC positivity. Clinical relevance, previous research, and univariable outcomes were used to choose candidate variables for multivariable analyses. Variables representing related biological domains were not added to the same model concurrently to minimize potential collinearity and overadjustment.

Sensitivity analyses were conducted using alternative-variable multivariable models. Multiple linear regression for ln(CACS+1), multivariable logistic regression for CACS < 100 versus CACS ≥ 100, and ordinal logistic regression for CACS categories of 0, 1 to 99, and ≥ 100 were used to further evaluate factors related to CAC burden.

R software (version 4.2.1) was used for all data analyses. All tests were two-sided, and a *P*-value < 0.05 was considered significant.

Ethics statement

The Ethics Committee of Shanghai Electric Power Hospital approved this research protocol (Approval No. 2026-J001). Before analysis, all data were de-identified, and informed consent was not required. The study was conducted in accordance with the tenets of the Declaration of Helsinki.

Results

Baseline characteristics based on CAC status

Demographic and anthropometric differences: The final analysis included 2,957 participants. With a median age of 50 years (IQR, 44-55 years) and a median BMI of 24.5 kg/m^2 (IQR, 22.4-26.8 kg/m^2), the study population predominantly comprised men (2,427/2,957, 82.1%). Overall, 2,220 participants (75.1%) had a CACS of 0, whereas 737 (24.9%) had a CACS > 0. Participants with CAC positivity were older, more likely to be men, and had a higher BMI than participants with a CACS of 0 (all P < 0.001; **Table 1**).

Cardiometabolic differences: FPG, HbA1c, TG, and uric acid levels were higher in participants with CACS > 0, whereas HDL-C levels were lower than in participants with a CACS of 0 (all P < 0.001). No statistically significant differences were observed in TC or LDL-C between the two groups (P=0.947 and P=0.424, respectively; **Table 1**).

Hepatic and renal differences: ALT, AST, GGT, ALP, total bile acid, prealbumin, and cholinesterase levels were higher in participants with a CACS > 0 than in participants without CAC. In contrast, no statistically significant differences were observed in total bilirubin, direct bilirubin, total protein, albumin, globulin, albumin/globulin ratio, or α-HBDH between the two groups. Notably, participants with a CACS > 0 had higher urea and creatinine levels but lower eGFR values (all P < 0.001; **Table 1**).

Overall, several cardiometabolic, hepatic, and renal markers showed differences between participants with and without CAC.

Factors associated with CAC positivity in univariable analyses

Demographic and anthropometric factors: According to univariable logistic regression analyses, old age, male sex, and higher BMI were associated with CAC positivity (**Table 2**). Specifically, the odds of CAC positivity rose with age (OR 1.147, 95% CI 1.129-1.165, P < 0.001) and BMI (OR 1.136, 95% CI 1.108-1.165, P < 0.001). Furthermore, men were more likely to test positive for CAC than women (OR 16.907, 95% CI 9.685-29.516, P < 0.001).

Cardiometabolic factors: Among cardiometabolic markers, high levels of FPG, HbA1c, TG, and uric acid were associated with greater odds of CAC positivity, whereas high HDL-C levels were associated with lower odds of CAC positivity (all P < 0.001; **Table 2**). In contrast,

Table 1. Baseline characteristics of the study population according to coronary artery calcification status

Characteristics	Overall (n=2957)	CACS=0 (n=2220)	CACS > 0 (n=737)	*P* value
Age, years	50 (44, 55)	48 (43, 54)	55 (50, 58)	< 0.001
Sex, n (%)				< 0.001
Female	530 (17.9)	517 (23.3)	13 (1.8)	
Male	2427 (82.1)	1703 (76.7)	724 (98.2)	
BMI, kg/m^2	24.5 (22.4, 26.8)	24.2 (22.0, 26.4)	25.6 (23.7, 27.8)	< 0.001
Cardiometabolic markers				
FPG, mmol/L	5.1 (4.8, 5.7)	5.1 (4.7, 5.5)	5.5 (5, 6.4)	< 0.001
HbA1c, %	5.6 (5.4, 5.8)	5.5 (5.4, 5.7)	5.7 (5.5, 6.3)	< 0.001
TG, mmol/L	1.6 (1.1, 2.3)	1.5 (1.0, 2.2)	1.8 (1.3, 2.7)	< 0.001
TC, mmol/L	5.3 (4.7, 5.9)	5.3 (4.7, 5.9)	5.3 (4.6, 5.9)	0.947
HDL-C, mmol/L	1.2 (1.0, 1.4)	1.2 (1.1, 1.4)	1.1 (1.0, 1.3)	< 0.001
LDL-C, mmol/L	2.9 (2.4, 3.5)	2.9 (2.4, 3.4)	2.9 (2.4, 3.6)	0.424
Uric acid, umol/L	355.0 (298.0, 412.0)	349.0 (291.0, 408.3)	371.0 (324.0, 425.0)	< 0.001
Hepatic markers				
ALT, u/L	23.0 (17.0, 34.0)	22.0 (16.0, 33.0)	25.0 (18.0, 36.0)	< 0.001
AST, u/L	20.0 (17.0, 25.0)	20 (17.0, 24.0)	21.0 (18.0, 26.0)	< 0.001
GGT, u/L	29.0 (20.0, 46.0)	27.0 (19.0, 43.0)	35.0 (25.0, 55.0)	< 0.001
ALP, u/L	71.0 (60.0, 85.0)	71.0 (59.0, 84.0)	73.0 (62.0, 86.0)	0.006
Total bilirubin, umol/L	11.8 (9.1, 15.3)	11.9 (9.0, 15.4)	11.8 (9.4, 15)	0.832
Direct bilirubin, umol/L	3.6 (2.8, 4.6)	3.6 (2.8, 4.6)	3.6 (2.8, 4.6)	0.900
Total bile acid, umol/L	3.6 (2.9, 4.5)	3.6 (2.8, 4.3)	3.6 (2.9, 4.9)	0.011
Total protein, g/L	76.5 (73.2, 78.7)	76.5 (73.2, 78.7)	76.5 (73.2, 78.6)	0.742
Albumin, g/L	46.6 (45.0, 47.8)	46.6 (45.0, 47.8)	46.6 (45.0, 47.4)	0.574
Globulin, g/L	30.2 (27.6, 32.3)	30.2 (27.6, 32.3)	30.2 (27.6, 32.3)	0.925
A/G	1.6 (1.4, 1.7)	1.6 (1.4, 1.7)	1.6 (1.4, 1.7)	0.853
Prealbumin, mg/L	291.0 (260.0, 334.0)	291.0 (260.0, 334.0)	300 (262.0, 333.0)	0.022
Cholinesterase, u/L	9246.0 (8115.0, 10250.0)	9171.0 (7974.5, 10200.0)	9476.0 (8507.0, 10380.0)	< 0.001
α-HBDH, u/L	126.0 (114.0, 140.0)	125 (113.0, 139.0)	127.0 (115.0, 141.0)	0.081
Renal markers				
Urea, mmol/L	5.0 (4.3, 5.8)	4.9 (4.2, 5.7)	5.3 (4.5, 6.1)	< 0.001
Creatinine, umol/L	71.0 (62.0, 80.0)	71.0 (61.0, 79.0)	73.0 (65.0, 81.0)	< 0.001
eGFR, mL/min	98.9 (85.8, 115.4)	100.2 (87.2, 116.7)	95.4 (82.2, 110.5)	< 0.001

Abbreviations: CAC, coronary artery calcification; CACS, coronary artery calcification score; BMI, body mass index; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; ALP, alkaline phosphatase; A/G, albumin/globulin ratio; α-HBDH, alpha-hydroxybutyrate dehydrogenase; eGFR, estimated glomerular filtration rate. Note: Data are presented as median (interquartile range) for continuous variables and n (%) for categorical variables. Comparisons between the two groups were performed using the Mann-Whitney U test for continuous variables and the chi-square test for categorical variables.

TC and LDL-C were not significantly associated with CAC positivity.

Hepatic and renal factors: High levels of hepatic markers, such as ALT, AST, GGT, ALP, prealbumin, and cholinesterase, were significantly associated with CAC positivity in univariable analyses (**Table 2**). However, there was no significant correlation between total bilirubin, direct bilirubin, total bile acid, total protein, albumin, albumin/globulin ratio, and α-HBDH and CAC positivity. Renal markers, such as higher urea and creatinine levels, were associated with greater odds of CAC positivity, whereas higher eGFR was associated with lower odds of CAC positivity (all P < 0.001; **Table 2**).

According to the univariable logistic regression analyses, CAC positivity was associated with negative cardiometabolic, hepatic, and renal markers.

Table 2. Univariable logistic regression analyses of factors associated with CAC positivity

Variables		Total (N)	Odds Ratio (95% CI)	*P* value
Age, years		2,957	1.147 (1.129-1.165)	< 0.001
Sex		2,957		
Female		530	Reference	
Male		2,427	16.907 (9.685-29.516)	< 0.001
BMI, kg/m^2		2,957	1.136 (1.108-1.165)	< 0.001
Cardiometabolic markers	FPG, mmol/L	2,957	1.425 (1.336-1.519)	< 0.001
	HbA1c, %	2,957	1.815 (1.631-2.019)	< 0.001
	TG, mmol/L	2,957	1.201 (1.136-1.270)	< 0.001
	TC, mmol/L	2,957	0.961 (0.884-1.045)	0.354
	HDL-C, mmol/L	2,957	0.261 (0.185-0.368)	< 0.001
	LDL-C, mmol/L	2,957	1.003 (0.904-1.112)	0.961
	Uric acid, umol/L	2,957	1.003 (1.002-1.004)	< 0.001
Hepatic markers	ALT, u/L	2,957	1.006 (1.003-1.010)	< 0.001
	AST, u/L	2,957	1.012 (1.005-1.019)	< 0.001
	GGT, u/L	2,957	1.005 (1.003-1.007)	< 0.001
	ALP, u/L	2,957	1.005 (1.001-1.009)	0.007
	Total bilirubin, umol/L	2,957	0.995 (0.980-1.010)	0.507
	Direct bilirubin, umol/L	2,957	0.994 (0.943-1.048)	0.825
	Total bile acid, umol/L	2,957	1.002 (0.988-1.017)	0.736
	Total protein, g/L	2,957	0.999 (0.979-1.020)	0.952
	Albumin, g/L	2,957	0.993 (0.954-1.033)	0.729
	A/G	2,957	0.982 (0.657-1.468)	0.930
	Prealbumin, mg/L	2,957	1.002 (1.001-1.004)	0.008
	Cholinesterase, u/L	2,957	1.000 (1.000-1.000)	< 0.001
	α-HBDH, u/L	2,957	1.002 (0.999-1.006)	0.157
Renal markers	Urea, mmol/L	2,957	1.187 (1.117-1.262)	< 0.001
	Creatinine, umol/L	2,957	1.016 (1.011-1.022)	< 0.001
	eGFR, mL/min	2,957	0.990 (0.986-0.994)	< 0.001

Abbreviations: CAC, coronary artery calcification; CI, confidence interval; BMI, body mass index; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; ALP, alkaline phosphatase; α-HBDH, alpha-hydroxybutyrate dehydrogenase; eGFR, estimated glomerular filtration rate. Note: Univariable logistic regression analyses were performed with CAC positivity (CAC > 0) as the dependent variable. Continuous variables were entered as continuous terms, and sex was treated as a binary categorical variable with female as the reference category.

Independent factors associated with CAC positivity

In the clinically driven multivariable logistic regression model, old age, male sex, high BMI, and high HbA1c levels were independently associated with CAC positivity (**Table 3**). Specifically, the odds of CAC positivity increased with age (adjusted OR 1.133, 95% CI 1.113-1.153, P < 0.001), BMI (adjusted OR 1.077, 95% CI 1.036-1.119, P < 0.001), and HbA1c levels (adjusted OR 1.338, 95% CI 1.202-1.491, P < 0.001). Men demonstrated a higher likelihood of CAC positivity than women (adjusted OR 7.990, 95% CI 4.381-14.573, P < 0.001).

By contrast, after multivariable adjustment, TG, HDL-C, uric acid, GGT, and eGFR were not independently associated with CAC positivity.

Overall, age, male sex, BMI, and HbA1c were independent variables associated with CAC positivity.

Sensitivity analyses

Results from sensitivity analyses using alternative-variable multivariable logistic regression

Table 3. Multivariable logistic regression analyses of factors associated with CAC positivity

Variables	Total (N)	Odds Ratio (95% CI)	*P* value
Age, years	2,957	1.133 (1.113-1.153)	< 0.001
Sex	2,957		
Female	530	Reference	
Male	2,427	7.990 (4.381-14.573)	< 0.001
BMI, kg/m^2	2,957	1.077 (1.036-1.119)	< 0.001
HbA1c, %	2,957	1.338 (1.202-1.491)	< 0.001
TG, mmol/L	2,957	1.050 (0.990-1.114)	0.105
HDL-C, mmol/L	2,957	1.074 (0.701-1.646)	0.743
Uric acid, umol/L	2,957	1.000 (0.999-1.002)	0.722
GGT, u/L	2,957	1.001 (0.999-1.003)	0.362
eGFR, mL/min	2,957	1.003 (0.998-1.009)	0.226

Abbreviations: CAC, coronary artery calcification; OR, odds ratio; CI, confidence interval; BMI, body mass index; HbA1c, glycated hemoglobin; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; GGT, gamma-glutamyl transferase; eGFR, estimated glomerular filtration rate. Note: Multivariable logistic regression was performed with CAC positivity (CACS > 0) as the dependent variable. The model included age, sex, BMI, HbA1c, TG, HDL-C, uric acid, GGT, and eGFR. Female sex was used as the reference category.

models were in line with the primary model's findings (**Table 4**). Age, male sex, BMI, and HbA1c continued to be independently associated with CAC positivity in both alternative models, supporting the robustness of the key findings.

Factors associated with CAC burden

Ordinal logistic regression for CACS categories (0, 1-99, and ≥ 100), linear regression for ln(CACS+1), and logistic regression for CACS ≥ 100 were used for multivariable analyses of CAC burden (**Table 5**). All three models produced results that were consistent. Old age, male sex, high BMI, and high HbA1c levels remained independently associated with greater CAC burden. In contrast, TG, HDL-C, uric acid, GGT, and eGFR levels did not show significant independent associations after adjustment. Taken together, age, male sex, BMI, and HbA1c were consistently identified as independent predictors of a high CAC burden.

Discussion

The prevalence of CAC among asymptomatic adults in this Chinese population undergoing health examinations suggests a quantifiable burden of subclinical coronary atherosclerosis. Several cardiometabolic, hepatic, and renal markers were associated with CAC in univariable analyses. Nonetheless, only old age, male sex, high BMI, and high HbA1c levels were independently associated with CAC positivity in the clinically driven multivariable model. Sensitivity analyses and CAC burden models further supported these associations. Overall, these findings are in line with previous research that supports CAC as a direct imaging indicator of subclinical coronary atherosclerosis and its strong relationship with metabolic abnormalities, particularly glycemic dysfunction [7, 8, 11, 12].

In this study, age, male sex, BMI, and HbA1c were most consistently associated with CAC. The associations of old age and male sex with a high risk of CAC and greater CAC burden are consistent with previous reports. Therefore, CAC reflects the prolonged cumulative process of atherosclerosis and may be influenced by sex-related differences in cardiovascular risk exposure [15]. In keeping with earlier findings that overweight (OR=1.13, 95% CI: 1.10-1.20) and obesity (OR=1.50, 95% CI: 1.40-1.60) are linked to a higher risk of CAC, BMI remained independently associated with both CAC positivity and a high CAC burden [6]. Notably, HbA1c remained significant in the primary, sensitivity, and burden analyses, making it the most stable cardiometabolic marker. Furthermore, this finding is consistent with the following previous findings: (I) The pooled prevalence of CAC in patients with metabolic syndrome was 39.8%, (II) Men had a two-fold higher risk of CAC than women, and (III) Metabolic syndrome was associated with a high risk of CAC (OR range, 1.34-1.50) [11]. Furthermore, on examining 8,955 Chinese adults, Huang et al. reported that HbA1c levels between 5.7% and 6.4% and ≥ 6.5% were associated with 28% (OR=1.28, 95% CI: 1.07-1.52) and 116% (OR=2.16, 95% CI: 1.48-3.16) higher risks of CAC, respectively, compared with HbA1c < 5.7% [12]. Together, continuing glycemic exposure and clustering of metabolic abnormalities may contribute to

Table 4. Sensitivity analyses using alternative-variable multivariable logistic regression models for factors associated with CAC positivity

Variables	Total (N)	Odds Ratio (95% CI)	*P* value
Model 1: ALT and creatinine as alternative hepatic and renal markers			
Age, years	2,957	1.127 (1.108-1.145)	< 0.001
Sex	2,957		
Female	530	Reference	
Male	2,427	8.185 (4.464-15.005)	< 0.001
BMI, kg/m^2	2,957	1.096 (1.061-1.132)	< 0.001
HbA1c, %	2,957	1.352 (1.214-1.505)	< 0.001
TG, mmol/L	2,957	1.057 (0.996-1.121)	0.068
HDL-C, mmol/L	2,957	1.085 (0.709-1.661)	0.706
Uric acid, umol/L	2,957	1.000 (0.999-1.002)	0.595
ALT, u/L	2,957	0.998 (0.994-1.003)	0.442
Creatinine, umol/L	2,957	0.996 (0.989-1.003)	0.264
Model 2: AST and urea as alternative hepatic and renal markers			
Age, years	2,957	1.127 (1.109-1.145)	< 0.001
Sex	2,957		
Female	530	Reference	
Male	2,427	7.549 (4.170-13.665)	< 0.001
BMI, kg/m^2	2,957	1.093 (1.059-1.129)	< 0.001
HbA1c, %	2,957	1.349 (1.211-1.503)	< 0.001
TG, mmol/L	2,957	1.053 (0.993-1.117)	0.085
HDL-C, mmol/L	2,957	1.084 (0.708-1.660)	0.709
Uric acid, umol/L	2,957	1.000 (0.999-1.001)	0.865
AST, u/L	2,957	0.999 (0.991-1.007)	0.877
Urea, mmol/L	2,957	1.013 (0.946-1.084)	0.712

Abbreviations: CAC, coronary artery calcification; OR, odds ratio; CI, confidence interval; BMI, body mass index; HbA1c, glycated hemoglobin; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase. Note: Sensitivity analyses were performed using alternative-variable multivariable logistic regression models with CAC positivity (CACS > 0) as the dependent variable. Model 1 replaced GGT and eGFR in the main model with ALT and creatinine, respectively. Model 2 replaced GGT and eGFR in the main model with AST and urea, respectively. Female sex was used as the reference category.

the development and progression of coronary calcification.

In univariable analysis, TG, HDL-C, uric acid, GGT, and eGFR levels were significantly associated with CAC. Nonetheless, after multivariable adjustment, these associations lost significance, suggesting a possible shared metabolic background rather than independent effects. Similarly, some metabolic and organ function markers may be associated with CAC or coronary atherosclerosis in crude analyses. However, their independent effects are not always stable after additional adjustment. For example, in 14,439 asymptomatic Korean adults, Lee et al. reported that the association between GGT and CAC was significant in men (OR=1.49, 95% CI: 1.21-1.85) but not in women (OR=1.33, 95% CI: 0.62-2.87). This relationship may be influenced by sex and other metabolic factors [16]. Likewise, Grossman et al. demonstrated that participants in the highest uric acid group had a greater risk of CAC than participants in the lowest uric acid group (OR=1.84, 95% CI: 1.10-3.07). In contrast, a meta-analysis showed substantial heterogeneity across studies, suggesting that the effect of uric acid may vary according to population characteristics and accompanying metabolic abnormalities [17, 18]. Furthermore, hepatic and renal dysfunction have been related to CAC. In a meta-analysis involving 10,060 participants, nonalcoholic fatty liver disease (NAFLD) was associated with a high risk of CAC progression (OR=1.50, 95% CI: 1.34-1.68) [19]. In a longitudinal cohort of 10,153 asymptomatic adults, NAFLD was independently associated with CAC incidence (adjusted HR= 1.348, 95% CI: 1.051-1.730) [20], suggesting that hepatic steatosis and its metabolic milieu may contribute to coronary calcification. Furthermore, CAC is prevalent in patients with chronic kidney disease (CKD), with a baseline prevalence of approximately 66% in adults with early CKD [21]. Its progression appears to be more pronounced in patients with advanced CKD [14]. This may explain why renal dysfunction-related markers showed crude associations with CAC in the cohort but did not remain independently significant after multivariable adjustment. Therefore, TG, HDL-C, uric acid, GGT, and eGFR levels may still indicate metabolic or organ dysfunction in the current Chinese population undergoing health examinations. However, their associations with CAC

Table 5. Summary of multivariable analyses for factors associated with CAC burden

Variables	Ordinal logistic regression CACS: 0, 1-99, ≥ 100		Linear regression ln(CACS+1)		Logistic regression CACS ≥ 100	
	Adjusted OR (95% CI)	*P* value	Adjusted β (95% CI)	*P* value	Adjusted OR (95% CI)	*P* value
Age, years	1.130 (1.111-1.149)	< 0.001	0.059 (0.050 to 0.068)	< 0.001	1.134 (1.101-1.167)	< 0.001
Sex						
Female	Reference		Reference		Reference	
Male	7.887 (4.330-14.368)	< 0.001	0.427 (0.220 to 0.633)	< 0.001	6.733 (2.048-22.138)	0.002
BMI, kg/m^2	1.079 (1.040-1.119)	< 0.001	0.040 (0.015 to 0.065)	0.002	1.068 (1.008-1.131)	0.025
HbA1c, %	1.382 (1.253-1.523)	< 0.001	0.355 (0.278 to 0.432)	< 0.001	1.440 (1.273-1.629)	< 0.001
TG, mmol/L	1.045 (0.989-1.104)	0.115	0.025 (-0.017 to 0.068)	0.239	1.045 (0.973-1.123)	0.230
HDL-C, mmol/L	1.074 (0.707-1.632)	0.738	0.046 (-0.208 to 0.301)	0.722	1.129 (0.572-2.228)	0.727
Uric acid, μmol/L	1.000 (0.999-1.001)	0.850	-0.000 (-0.001 to 0.001)	0.536	1.000 (0.998-1.002)	0.863
GGT, U/L	1.001 (0.999-1.003)	0.338	0.000 (-0.001 to 0.002)	0.631	1.001 (0.999-1.003)	0.405
eGFR, mL/min	1.002 (0.997-1.008)	0.409	0.000 (-0.003 to 0.004)	0.933	0.999 (0.990-1.007)	0.789

Abbreviations: CAC, coronary artery calcification; CACS, coronary artery calcification score; OR, odds ratio; CI, confidence interval; BMI, body mass index; HbA1c, glycated hemoglobin; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; GGT, gamma-glutamyl transferase; eGFR, estimated glomerular filtration rate. Note: All three models included age, sex, BMI, HbA1c, TG, HDL-C, uric acid, GGT, and eGFR as independent variables. The ordinal logistic regression used three CAC burden categories (0, 1-99, and ≥ 100). The linear regression used ln(CACS+1) as the dependent variable. The logistic regression used CACS < 100 versus CACS ≥ 100 as the dependent variable. Female sex was used as the reference category.

were at least partially explained by more dominant clinical factors, such as age, sex, BMI, and HbA1c.

Ordinal logistic regression, logistic regression for CACS ≥ 100, and linear regression for ln(CACS+1) were used to further evaluate CAC burden. Across all models, age, male sex, BMI, and HbA1c were consistently correlated with both CAC and a high calcification burden. This consistency implies that these factors may be involved in both the onset and progression of coronary calcification, strengthening the findings' robustness. CAC and its burden categories are central to cardiovascular risk categorization in asymptomatic adults. Higher CACS categories suggest a greater risk of atherosclerotic cardiovascular events [7, 8]. Therefore, standard clinical markers, such as age, sex, BMI, and HbA1c, may be used in Chinese populations undergoing health assessments to identify individuals who are at a high risk of CAC, direct further CACS assessments, and improve primary prevention strategies.

This study has several strengths. It was based on a sizeable Chinese population undergoing health examinations, which facilitated the assessment of CAC prevalence and related factors in asymptomatic adults. In addition to CAC positivity, three complementary models were used to determine the CAC burden, which strengthened the robustness of the findings. Simultaneously, cardiometabolic, hepatic, and renal markers were assessed, providing a comprehensive evaluation of their associations with CAC. Nonetheless, this study has several limitations. First, this study had a single-center and cross-sectional design, which precluded causal inference. Second, generalizability may be limited because the population was drawn from a health examination cohort that predominantly comprised men. Third, confounding cannot be ruled out because several confounders, including smoking, alcohol consumption, blood pressure, medication use, and history of cardiovascular diseases, were inaccessible.

Conclusion

CAC was not uncommon in this Chinese population undergoing health examinations. Old age, male sex, high BMI, and high HbA1c levels were associated with both CAC positivity and a high CAC burden. In contrast, several cardiometabolic, hepatic, and renal markers were not independently related to CAC after multivariable adjustment. Therefore, routine clinical indicators, particularly age, adiposity, and continuing glycemic exposure, may facilitate the identification of asymptomatic individuals at a high risk of CAC and allow for additional risk stratification.

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Disclosure of conflict of interest

None.

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