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Original Article

The Correlation of Renal Artery Calcification With Coronary Artery Calcification in Iraqi Patients

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ABSTRACT

Background: Coronary artery disease remains the most common cause of death. Because coronary artery calcium (CAC) scores may indicate an increased risk of developing symptomatic coronary artery disease, we sought to determine whether CAC scores correlate with renal artery calcification (RAC) identified on computed tomography (CT).

Methods: CT was employed to determine the presence and extent of RAC and CAC in 476 patients without clinical cardiovascular disease, utilizing an Agatston scoring system. Cardiovascular risk factors, including diabetes, hypertension, dyslipidemia, family history of ischemic heart disease, and smoking, were documented for all patients.

Results: RAC was present in 24% of patients, with a higher frequency in men than in women (66.7% vs 51.9%; $P = 0.006$). The RAC score was correlated with the CAC score ($r = 0.67$). In a multivariable model that included standard cardiovascular disease risk variables, the presence of RAC was associated with age, current smoking, and the CAC score (OR for CAC, 1.24; $P < 0.001$). Specifically, a CAC score greater than 100 Agatston units (AU) was observed in 73% (84/114) of patients with RAC and 3% (12/362) of patients without RAC. A RAC score of 4 AU had a sensitivity of 87% and a specificity of 99% for predicting a CAC score greater than 100 AU.

Conclusions: RAC was correlated with CAC independent of conventional risk factors. RAC scoring may be used to identify patients with a CAC score greater than 100 AU, for whom statin therapy may be beneficial. ***(Iranian Heart Journal 2026; 27(1): 17-25)***

KEYWORDS: atherosclerosis; calcium; renal arteries; coronary artery calcium score; Agatston score

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Coronary artery disease (CAD) remains the leading cause of mortality.[1] A substantial proportion of the population presents with myocardial infarction or sudden cardiac death despite significant advancements in preventive cardiology.[2] Risk stratification is crucial for the prevention and management of CAD. The Atherosclerotic Cardiovascular Disease (ASCVD) Risk Estimator Plus (2018) estimates 10-year risk for ASCVD, categorizing it as high risk (≥20%), intermediate risk (7.5%–19.9%), borderline risk (5%–7.4%), and low risk (<5%), based on variables including age, sex,

diabetes, smoking, hypertension, and lipid profile.[3] Nonetheless, many cardiovascular events occur in individuals without these traditional risk factors, and more than 50% of events occur in patients classified as low to intermediate risk, particularly among middle-aged women, as these risk scores may underestimate risk.[4–6] This has prompted the development of novel risk assessment methods, including the coronary artery calcium (CAC) score, C-reactive protein level, and the ankle-brachial index.[7–9] Based on numerous cohort studies, the CAC score has been endorsed as a useful risk indicator for screening asymptomatic patients at low to intermediate risk by both the American College of Cardiology and the American Heart Association.[7–9] Despite its clinical value, the requirement for increased radiation exposure, expense, and trained personnel and equipment restricts the use of CAC scoring as a large-scale intervention.

Atherosclerosis is a systemic inflammatory process that is often initiated at arterial branch sites.[10–12] Atherosclerotic plaques actively deposit calcium into their lipid cores via a controlled process analogous to cortical bone development.[13–15] Vascular calcification is readily detected by computed tomography (CT). Previous research has shown that atherosclerotic calcification in the coronary arteries, iliac arteries, aorta, and mitral and aortic annuli is associated, even after adjustment for cardiovascular risk factors.[16–18]

A novel risk classification method that involves no additional radiation or cost is the assessment of renal artery calcification (RAC). Abdominal CT is performed for thousands of patients annually for various indications and may help reduce the use of invasive coronary angiography, a procedure that carries a risk of serious complications.[19, 20].

The present study aimed to determine the significance of RAC as a potential risk factor for CAD by analyzing the correlation between RAC and CAC scores in Iraqi patients undergoing noncontrast abdominal and cardiac CT, a population for whom no prior data exist. Furthermore, the association between RAC and CAD risk factors, including hypertension, diabetes, smoking, dyslipidemia, and family history of ischemic heart disease (IHD), was investigated.

METHODS

From April 2023 through February 2025, 476 consecutive asymptomatic patients referred for primary prevention CAC scoring at Ibn al-Bitar Specialized Center for Cardiac Surgery in Baghdad, Iraq, underwent multidetector computed tomography (MDCT) to evaluate CAC and RAC. Cardiac risk factors were documented, including hypertension, diabetes, dyslipidemia, smoking, and family history of IHD. Exclusion criteria included coronary or carotid revascularization, myocardial infarction, angina pectoris, transient ischemic attack, stroke, and clinical or subclinical hypothyroidism.[21, 22]

Patient records were evaluated, and participants completed a comprehensive health history questionnaire regarding hypertension, diabetes, dyslipidemia, smoking habits, family history of IHD, medication history, and surgical history. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Hypertension was defined as systolic blood pressure greater than 130 mm Hg, diastolic blood pressure greater than 80 mm Hg, or current use of antihypertensive therapy.[23] Dyslipidemia was defined as total cholesterol level greater than 200 mg/dL, low HDL cholesterol level (<35 mg/dL), high LDL cholesterol level (>130 mg/dL), or use of lipid-lowering medications. Diabetes was defined as current use of antiglycemic medication, random glucose level greater than 200 mg/dL, fasting glucose level greater than 126 mg/dL, or HbA1c level greater than 6.5%.

Participants were categorized as current, former, or never smokers. Informed consent

was obtained, and the local ethics committee approved the study protocol.

Patients underwent MDCT (Brilliance 64 [64-slice]; Philips Medical Systems, the Netherlands) scanning to detect CAC while in the supine position. Imaging used a slice thickness of 3 mm and 120 kVp. Cardiac images were obtained during a breath hold using prospective electrocardiographic triggering at 50% to 80% of the RR interval. The area and peak density were calculated for identified calcifications. The CAC score was measured using the Agatston method, in which the calcification area (defined as >1 mm^2 with density >130 Hounsfield units [HU]) is multiplied by a density weighting factor (1 = 130–199 HU; 2 = 200-299 HU; 3 = 300–399 HU; 4 ≥ 400 HU).[24] The total CAC score for the entire coronary tree was calculated. Scores were categorized by coronary plaque burden: 0, no disease; 1 to 99, mild disease; 100 to 300, moderate disease; and greater than 300, severe disease.

RAC was evaluated using the same method but with a slice thickness of 6 mm through the abdomen. Agatston scores for renal arteries were adjusted for slice thickness using the following formula: adjusted score = initial score × slice thickness / 3. Homogeneous slice segments were scored independently to minimize volume averaging artifacts. Abdominal CT images were examined for calcium in both renal arteries, and Agatston scores were computed using the same software. Scores included calcium originating from the ostia or the arterial segment; calcification of the abdominal aorta wall was excluded. The RAC reader was blinded to CAC scores and viewed only images containing the renal arteries.

Statistical Analysis

Continuous variables were compared using analysis of variance or the Kruskal-Wallis test, as appropriate; categorical variables were compared using the chi-square test to assess differences between groups with and without RAC. Correlations were assessed using the Spearman rank correlation. Multivariable logistic regression was utilized to investigate the association among CAC score, conventional CVD risk factors, and the presence of RAC. Receiver operating characteristic (ROC) curves were constructed to evaluate the ability of the RAC score to distinguish the presence of moderate CAC (score >100), including calculation of sensitivity and specificity at a selected cutoff value. The area under the curve (AUC) was calculated. Statistical significance was defined as a *P* value less than 0.05. Analyses were performed using SPSS software, version 27 (IBM Corp).

RESULTS

Table 1 shows descriptive statistics stratified by RAC presence. RAC was identified in one or both renal arteries in 24% (114/476) of patients; prevalence was higher in men than women (66.7% vs 51.9%; $P = 0.006$). Mean age was 65 years in the RAC-present group and 60.59 years in the RAC-absent group; the difference was statistically significant. RAC score distributions were substantially skewed because of the high proportion of participants without RAC. RAC scores ranged from 0 to 74 (median, 0; interquartile range, 0 to 0).

The Spearman rank correlation between RAC and CAC scores was statistically significant (coefficient, 0.677; $P < 0.01$). Age, male sex, current smoking, hypertension, and CAC were associated with RAC presence in logistic regression models ($P < 0.05$ for all); former smoking, dyslipidemia, diabetes, BMI, and family history were not (Table 2). In the multivariable model, only age, current smoking, and CAC score were associated with RAC. Odds of RAC increased by 5% for every year of age and by 24% for every 10-Agatston unit (AU) increase in CAC score.

Table 1. Baseline features classified according to the presence of RAC

Characteristic	RAC > 0	RAC = 0	*P*
Age, mean (SD), y	65 (10.19)	60.59 (8.49)	<0.001
Men, No. (%)	76 (66.7%)	188 (51.9%)	0.006
Former smoker, No. (%)	8 (7.0%)	27 (7.5%)	0.875
Current smoker, No. (%)	29 (25.4%)	54 (14.9%)	0.010
Diabetes mellitus, No. (%)	21 (18.4%)	62 (17.1%)	0.751
Dyslipidemia, No. (%)	28 (24.6%)	65 (18.0%)	0.121
Hypertension, No. (%)	53 (46.5%)	126 (34.8%)	0.025
Family history of IHD, No. (%)	21 (18.4%)	65 (18.0%)	0.910
BMI, mean (SD), kg/m²	29.04 (5.43)	27.95 (5.51)	0.066
CAC score, median (IQR), AU	201 (85.75– 270.50)	0 (0 – 27)	<0.001

AU: Agatston unit; BMI: body mass index; CAC: coronary artery calcium; IHD: ischemic heart disease; IQR: interquartile range; RAC: renal artery calcium

Table 2. Univariate and multivariable risk factor analysis for RAC

	Univariable Analysis			**Multivariable** Logistic Analysis		
Risk Factor	OR	95% CI	*P*	OR	95% CI	*P*
Age	1.05	1.03-1.08	<0.001	1.05	1.01-1.09	0.011
Male sex	1.85	1.19-2.87	0.006	1.98	0.94-4.18	0.072
Former smoker	0.93	0.41-2.12	0.875	5.44	0.56-52.89	0.144
Current smoker	1.94	1.16-3.24	0.011	9.63	1.21-76.39	0.032
Hypertension	1.62	1.06-2.49	0.025	0.680	0.21-2.14	0.510
Dyslipidemia	1.48	0.89-2.46	0.122	0.707	0.19-2.63	0.605
Diabetes	1.09	0.63-1.88	0.751	0.35	0.06-1.82	0.213
BMI	1.03	0.99-1.07	0.067	1.05	0.99-1.12	0.077
Family history of IHD	1.03	0.59-1.77	0.910	0.59	0.10-3.27	0.553
CAC score	1.25	1.19-1.31	<0.001	1.24	1.19-1.30	<0.001

BMI: body mass index; CAC: coronary artery calcium; IHD: ischemic heart disease; RAC: renal artery calcium

The proportion of patients in each CAC score subgroup declined as scores increased in the RAC-negative group. Conversely, the RAC-positive group showed an inverse trend, with the proportion of patients increasing across higher CAC score subgroups (Figure 1). This pattern was most pronounced in CAC score categories greater than or less than 100 AU. A CAC score greater than 100 AU was observed in 73% (84/114) of patients with RAC and 3% (12/362) of patients without RAC ($P < 0.001$). Thus, RAC was associated with a CAC score greater than 100 AU, indicating moderate or severe coronary plaque burden. The AUC for RAC to detect moderate CAC was 0.931 (95% CI, 0.89 to 0.97), indicating high capability to identify moderate CAC (score >100). A RAC score cutoff of 4 AU predicted a CAC score greater than 100 AU with a sensitivity of 87% and specificity of 99%.

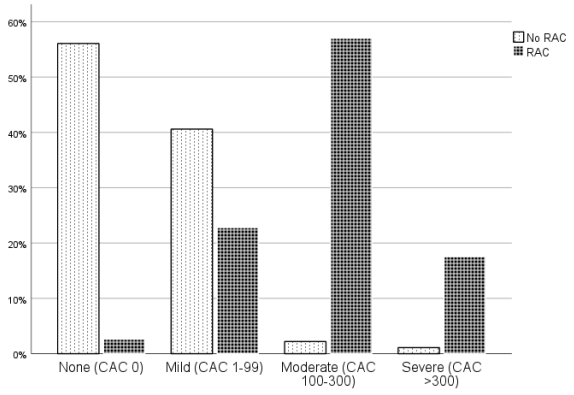

Figure 1. Coronary artery calcification (CAC) score distribution stratified by the presence of renal artery calcification (RAC)

DISCUSSION

The current study of participants without a clinical diagnosis of CVD indicates that the presence of RAC is substantially correlated with CAC independently of CVD risk

factors. After multivariable adjustment, age and current smoking were strongly correlated with any RAC among these risk factors.

A mechanism analogous to endochondral skeletal bone development appears to be responsible for atherosclerotic plaque calcification.[25] These calcium deposits are indicative of more advanced plaques (stage 4) according to histology.[26] Clinical studies of calcified atherosclerosis have mostly involved the coronary arteries and found that plaque burden and, to a lesser degree, coronary stenosis are strongly associated with calcium levels in the coronary arteries.[27]

So far, only a small number of studies have shown that RAC on CT is associated with either clinical or subclinical CAD. There is a statistically significant correlation between RAC and CAD, risk factors for CAD, renal outcomes, and death, according to small cohort studies conducted in both the United States and European Union countries.[18, 28–39] Coronary artery and other vascular bed atherosclerotic calcifications were found to be strongly correlated with RAC on CT in a study of 1461 participants.[18] Even after adjustment for more common cardiovascular risk factors, RAC was found to increase the risk of all-cause mortality in otherwise healthy outpatients in an 8.2-year follow-up analysis.[32] A recent study of 268 patients with severe aortic stenosis undergoing transcatheter aortic valve implantation (TAVI) showed that RAC is a strong and independent predictor of cardiovascular death.[39] Results from studies examining the association between RAC and renal artery luminal stenosis have been contradictory. Significant associations were found between the severity of calcifications and the degree of renal artery stenosis (r = 0.7) in a study that included 350 patients. Additionally, high-grade narrowing was associated with the presence of bilateral calcifications.[30] Nonetheless, Siegel et al[40] found no statistically strong correlation between RAC and luminal stenosis, suggesting that RAC might not be a good indicator of severe hemodynamic stenosis, a result comparable to that observed for CAC.[27]

Thomas et al[34] found that 33% of participants had RAC greater than zero, and a total of 38% of White Americans had RAC, with 31.1% of Hispanic individuals, 30% of Chinese American individuals, and 28.7% of African American individuals following closely behind. The median Agatston score was 63.9 (IQR, 17.1–179.9) among individuals with any RAC (RAC >0). The current literature on RAC is limited, especially in populations of Middle Eastern descent, and RAC may behave in a similar manner to CAC, which shows differences across races and ethnicities.[6, 41] The current study findings are comparable with those of a previous study that examined 96 diabetic White adults and found a strong association between RAC and atherosclerotic calcium in the carotid, coronary, iliac, and infrarenal arteries.[28] Moreover, prediabetes was associated with higher CAC and left ventricular dysfunction.[42, 43] In this study, the overall prevalence of RAC was 24%, whereas the prevalence was 65% in a group of diabetic individuals studied by Freedman et al.[28]

Similar to calcium in other vascular beds, RAC is associated with risk factors previously identified as relevant, including age and sex.[16, 44, 45] Consistent with several investigations conducted in various vascular beds, including the mitral and aortic annuli, the current cohort also showed a strong association between RAC and hypertension and smoking.[16,17,45] This study confirms previous findings showing no major association between vascular calcium and cholesterol levels (HDL and LDL).[16, 46]

Ischemic renal disease is usually caused by atherosclerotic renal artery stenosis, and individuals with the most severe disease also tend to have higher mortality rates and greater hypertension severity.[47, 48] An elevated serum

creatinine level has also been associated with preclinical atherosclerosis.[49] The current study's findings indicate that treating hypertension should be a priority because it is the only modifiable CVD risk factor that was found to be significantly correlated with RAC.

Limitations

Participants were selected by cardiologists for this study. Consequently, the findings may not be generalizable to the broader population. Cardiovascular CT cannot differentiate between calcium in the intima and media of the arterial wall. Medial calcification is most commonly observed in the lower limbs of patients with diabetes mellitus or chronic kidney disease.[50, 51] The number of patients included in the study was relatively small, and RAC quantification was based on the Agatston scoring methodology, which is primarily used for CAC. Because younger patients are not routinely screened for CVD risk, it would be useful to determine whether RAC predicts higher CVD risk. Nevertheless, the present cohort included few patients younger than 50 years; therefore, statistical analysis was not possible.

CONCLUSIONS

The results of the present study indicate that RAC is correlated with CAC independent of conventional CVD risk factors. The discovery of RAC on an abdominal CT scan performed for any indication, at no additional cost or radiation exposure, may prompt further evaluation of CAD risk factors and could help identify individuals who may benefit from statin therapy or other preventive strategies.

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Conflict of Interest

None reported.

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