
Diagnostic Performance of Breast Arterial Calcification for Detecting Coronary Artery Calcification in Women with Chronic Kidney Disease

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Abstract

Background: Arterial calcification is common in patients with chronic kidney disease (CKD) and contributes substantially to cardiovascular morbidity and mortality. Breast arterial calcification (BAC), which occurs exclusively in arterial media, may serve as a surrogate marker of coronary artery calcification (CAC) in CKD.

Methods: This cross-sectional study included 103 women aged >40 years who underwent digital mammography, lateral lumbar spine x-ray for abdominal aortic calcification (AAC), and non-contrast CT for coronary artery calcification (CAC). The BAC score was calculated based on the number of calcified breast arteries, the maximum length of calcification, and calcification density.

Results: BAC severity increases with advancing CKD stage. In multivariable analysis, adjusting for traditional cardiovascular risk factors, BAC was independently associated with reduced kidney function and older age. In patients with advanced CKD, BAC demonstrated high sensitivity (85.7%) and moderate accuracy (67.6%) for detecting CAC. Both BAC and AAC scores were significantly associated with CAC, with area under the curve (AUC) values of 0.67 and 0.84, respectively. The combined BAC and AAC model showed improved predictive performance for CAC (AUC 0.88).

Conclusions: Combined BAC and AAC provide good predictive performance for CAC, supporting the potential role of BAC as a noninvasive surrogate marker of vascular calcification burden in female patients with CKD.

Keywords: CVD; CKD-MBD; renal failure; media calcification; mammogram

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ประสิทธิภาพเชิงวินิจฉัยของภาวะแคลเซียมสะสมในหลอดเลือดแดงของเต้านมในการทำนายการเกิดแคลเซียมสะสมในหลอดเลือดหัวใจในสตรีโรคไตเรื้อรัง

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บทคัดย่อ

บทนำ: การเกิดแคลเซียมสะสมในหลอดเลือดแดงพบได้บ่อยในผู้ป่วยโรคไตเรื้อรัง และเป็นปัจจัยสำคัญที่ทำให้เกิดการเสียชีวิตจากโรคหัวใจและหลอดเลือดเพิ่มขึ้น การพบแคลเซียมสะสมในหลอดเลือดแดงของเต้านม ซึ่งเป็นการสะสมแคลเซียมที่ชั้นกลางของหลอดเลือด อาจใช้เป็นตัวบ่งชี้ทดแทนของของแคลเซียมสะสมในหลอดเลือดหัวใจในผู้ป่วยโรคไตเรื้อรัง

ระเบียบวิธีวิจัย: การศึกษานี้เป็นการศึกษาแบบตัดขวางในผู้ป่วยหญิงจำนวน 103 ราย อายุ >40 ปี ได้รับการตรวจด้วยแมมโมแกรมเอกซเรย์กระดูกสันหลังส่วนเอวเพื่อตรวจแคลเซียมสะสมในเออร์ตาส่วนท้อง และเอกซเรย์คอมพิวเตอร์เพื่อตรวจแคลเซียมสะสมในหลอดเลือดแดงโคโรนารี คำนวณแคลเซียมในหลอดเลือดแดงของเต้านมคำนวณจากจำนวนหลอดเลือดแดงของเต้านมที่มีแคลเซียม ความยาวสูงสุดของบริเวณที่มีแคลเซียม และความหนาแน่นของแคลเซียม

ผลการวิจัย: พบแคลเซียมในหลอดเลือดแดงของเต้านมเพิ่มขึ้นสัมพันธ์กับความรุนแรงของโรคไตเรื้อรัง การวิเคราะห์พหุตัวแปรโดยปรับปัจจัยเสี่ยงโรคหัวใจและหลอดเลือด พบแคลเซียมในหลอดเลือดแดงของเต้านมมีความสัมพันธ์อย่างอิสระกับค่าอัตราการกรองของไตที่ลดลงและอายุที่มากขึ้น ในผู้ป่วยโรคไตวายเรื้อรังระยะท้าย แคลเซียมในหลอดเลือดแดงเต้านมมีความไวสูงร้อยละ 85.7 และมีความแม่นยำปานกลางร้อยละ 67.6 ในการตรวจหาแคลเซียมในหลอดเลือดแดงโคโรนารี แคลเซียมในหลอดเลือดแดงเต้านมและเออร์ตาส่วนท้อง มีความสัมพันธ์อย่างมีนัยสำคัญกับแคลเซียมในโคโรนารีโดยมีค่าพื้นที่ใต้เส้นโค้ง (area under the curve; AUC) เท่ากับ 0.67 และ 0.84 ตามลำดับ แบบจำลองรวมแคลเซียมในหลอดเลือดแดงเต้านมและเออร์ตามีประสิทธิภาพการทำนายแคลเซียมในหลอดเลือดแดงโคโรนารีดีขึ้นด้วยค่า AUC 0.88

สรุป: การใช้แคลเซียมสะสมในหลอดเลือดแดงของเต้านมร่วมกับในหลอดเลือดแดงเออร์ตาส่วนท้อง ให้ประสิทธิภาพที่ดีในการทำนายการเกิดแคลเซียมในหลอดเลือดแดงโคโรนารี ซึ่งสนับสนุนบทบาทของแคลเซียมสะสมในหลอดเลือดแดงของเต้านมเป็นตัวบ่งชี้การเกิดแคลเซียมสะสมในผู้ป่วยโรคไตเรื้อรังโดยเฉพาะในผู้หญิง

คำสำคัญ: แคลเซียมเกาะในหลอดเลือด; ตะกรันในหลอดเลือดแดง; แคลเซียมในเส้นเลือดแดง; ตะกรันในเส้นเลือดแดง; ไตวาย

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Introduction

Chronic kidney disease (CKD) remains a major global health problem, with a rapidly increasing prevalence worldwide, making it the 12th leading cause of death globally.¹ Cardiovascular disease is the leading contributor to morbidity and mortality among patients with CKD. One of the principal mechanisms underlying cardiovascular disease in this population is vascular calcification. Both the prevalence and severity of vascular calcification are strong predictors of cardiovascular events and all-cause mortality in patients with CKD.²

Based on pathogenesis and clinical characteristics, vascular calcification is broadly classified into two distinct types: intimal and medial calcification. Intimal calcification is primarily associated with atherosclerosis and is commonly observed in patients with traditional cardiovascular risk factors, such as hypertension, hyperlipidemia, and diabetes mellitus. In contrast, medial calcification is a non-occlusive process that develops along elastic fibers within the arterial wall, leading to increased arterial stiffness and reduced vascular compliance. In patients with CKD, both forms of calcification are frequently observed; however, medial calcification is more prevalent and more specific in advanced CKD and end-stage kidney disease (ESKD).^{2,3}

Currently available imaging modalities for the assessment of vascular calcification include lateral abdominal radiographs for detecting abdominal aortic calcification (AAC), echocardiography for valvular and vascular calcification, and computed tomography (CT) for quantifying coronary artery calcification (CAC).⁴ Among these modalities, non-contrast CT coronary artery imaging is considered the gold standard for the detection and quantification of vascular calcification in clinical practice. However, CAC assessed by CT primarily reflects intimal calcification and is not specific for medial calcification, which predominates in CKD.⁵

Early studies demonstrated that calcification of breast arteries is confined to the medial layer of the vessel wall and is significantly more prevalent on mammography in patients with ESKD.⁶ Breast arterial calcification (BAC), therefore, represents a potentially

attractive and noninvasive imaging marker for medial vascular calcification. Subsequent studies have reported that the prevalence of BAC increases in parallel with CKD severity and dialysis duration.⁶⁻⁹ Despite these observations, the diagnostic performance of BAC as a screening tool for vascular calcification in patients with CKD has not been well quantified. In this study, we aimed to evaluate the feasibility of BAC for detecting vascular calcification and to investigate its associations with clinical and biochemical factors in patients with CKD.

Methods

Study Design and Population

This cross-sectional study was designed as a diagnostic study. Eligible participants were women aged >40 years with documented CKD, categorized according to CKD stages 2–5. All participants underwent evaluation for vascular calcification using digital mammography, lateral lumbar spine radiography, and non-contrast coronary CT scanning. Exclusion criteria included a history of breast cancer, prior coronary artery bypass grafting or coronary stent implantation, and any contraindication to mammography or coronary CT imaging. The study flow is illustrated in **Figure 1**.

Ethics approval

The study was conducted in accordance with the Declaration of Helsinki and approved by the Institute Review Board of the Thai Army Medical Department (Approval ID: R050h/64). Informed consent was obtained from all subjects.

Vascular Calcification Imaging

Screening mammograms for BAC and lateral abdominal radiographs for AAC were independently evaluated by a radiologist blinded to patients' clinical characteristics and CAC results. BAC was identified as thin, parallel, linear calcifications along the margins of breast arteries and was distinguished from other breast calcifications, which typically appear as punctate or irregular, thicker linear densities. The BAC score was recorded on a scale of 0 to 12 and calculated based on the number of calcified vessels, the maximum length of calcification, and calcification density.¹⁰

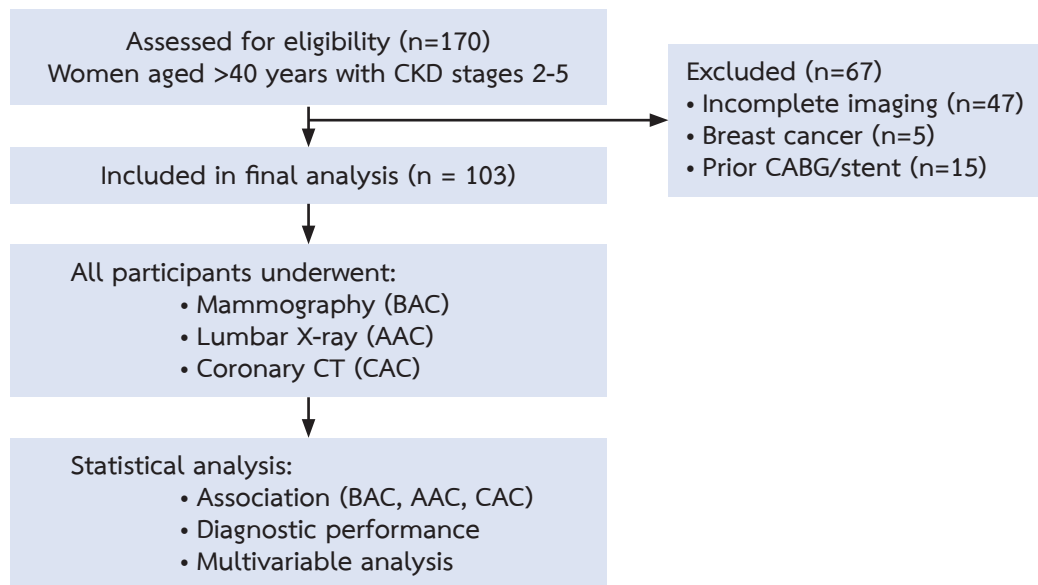


Figure 1 Study Flow Diagram

CKD, chronic kidney disease

AAC was assessed using the Kauppila scoring system, which grades the severity of anterior and posterior aortic calcification at lumbar vertebral segments L1–L4.¹¹ Coronary artery calcification was quantified by a cardiologist specializing in cardiac imaging using the Agatston scoring method on non-contrast CT images.¹²

All vascular imaging studies were performed within a closely defined timeframe, not exceeding 1 month. Patients were included if complete vascular imaging data were available within this period, rather than being prospectively referred for study-specific imaging.

Clinical and Laboratory Data

Clinical data collected included age, body mass index (BMI), smoking status, comorbidities, and medication use. Cardiovascular risk factors, including diabetes mellitus, hypertension, and dyslipidemia, were recorded. Laboratory parameters assessed for each participant included blood urea nitrogen (BUN), serum creatinine, estimated glomerular filtration rate (eGFR), calcium, phosphorus, albumin, intact parathyroid hormone (PTH), 25-hydroxyvitamin D [25(OH)D], lipid profiles, hemoglobin A1c, and alkaline phosphatase levels. Cardiovascular disease was defined as a documented history of ischemic heart disease, ischemic stroke, or peripheral arterial disease.

Data Analysis

Continuous variables are presented as mean $\pm$ standard deviation or median with interquartile range (IQR), as appropriate. Comparisons between groups were performed using the independent t-test for normally distributed data and the Mann–Whitney U test for non-normally distributed data. Categorical variables were compared using the chi-square test. Logistic regression analysis was conducted to evaluate the association between BAC and clinical or biochemical factors. Multivariable models were adjusted for potential confounders, including age, diabetes mellitus, hypertensive nephropathy, BMI, systolic blood pressure, baseline eGFR, and statin use.

Receiver operating characteristic (ROC) curve analysis was performed, and the area under the curve (AUC) was calculated to assess the predictive performance of BAC, AAC, and their combination for CAC. A two-sided P value <0.05 was considered statistically significant. All statistical analyses were performed using IBM SPSS Statistics for Windows, version 17. Sample size estimation was based on an expected vascular calcification incidence of 36%, yielding a minimum required sample size of 96 participants.⁶

Results

A total of 103 women were included in the final analysis. Baseline characteristics of patients with and without BAC are summarized in **Table 1**. The mean age of the study population was 70.06 ± 8.93 years. Patients with BAC were significantly older (72.95 ± 8.63 vs. 65.86 ± 7.66 years; $P < 0.001$), had lower eGFR (36.63 ± 18.88 vs. 49.56 ± 23.55 mL/min/1.73 m²; $P = 0.003$), higher hemoglobin A1c levels (6.75 ± 1.24 vs. $6.09 \pm 0.92\%$; $P = 0.003$), and higher AAC scores (median 9 vs. 4; $P = 0.010$) compared with patients without BAC.

There were no significant differences between groups with respect to underlying comorbidities, including hypertension, dyslipidemia, type 2 diabetes mellitus, coronary artery disease, and cerebrovascular disease. Similarly, no significant differences were observed in the use of calcium carbonate, vitamin D₂, calcitriol, warfarin, sevelamer or lanthanum, or statins. Laboratory parameters, including serum calcium, phosphate, intact parathyroid hormone (iPTH), 25-hydroxyvitamin D [25(OH) D], alkaline phosphatase, and lipid profiles, were also comparable between groups.

Table 1 Baseline characteristics of patients with and without breast arterial calcification (BAC)

Characteristic	Total	BAC positive	BAC negative	p-value
Demographics and clinical parameters				
Age (years)	70.06±8.93	72.95±8.63	65.86±7.66	<0.001
Body mass index (kg/m ²)	25.88±4.55	25.28±4.41	26.76±4.66	0.104
Systolic blood pressure (mmHg)	138.08±15.48	140.33±15.64	134.81±14.82	0.075
Diastolic blood pressure (mmHg)	73.0±11.38	73.0±11.29	73.0±11.64	0.933
Comorbidities, n (%)				
Type 2 diabetes mellitus	58 (56.3)	38 (62.3)	20 (47.6)	0.140
Hypertension	91 (88.3)	55 (90.2)	36 (85.7)	0.489
Dyslipidemia	86 (83.5)	49 (80.3)	37 (88.1)	0.297
Coronary heart disease	1 (1.0)	1 (1.6)	0 (0)	0.404
Cerebrovascular disease	1 (1.0)	1 (1.6)	0 (0)	0.404
Medications, n (%)				
Calcium carbonate	53 (51.5)	31 (50.8)	22 (54.2)	0.876
Vitamin D ₂	82 (79.6)	51 (83.6)	31 (73.8)	0.225
Calcitriol	3 (2.9)	2 (3.3)	1 (2.4)	0.790
Warfarin	7 (6.8)	6 (9.8)	1 (2.4)	0.140
Sevelamer or lanthanum	6 (5.8)	2 (3.3)	4 (9.5)	0.184
Statin therapy	88 (85.4)	55 (92.0)	33 (78.8)	0.101
Vascular calcification scores				
Coronary artery calcification (CAC) score	151 (19–681)	235 (26–751)	86 (4–346)	0.081
Abdominal aortic calcification (AAC) score	8 (1–13)	9 (3–14)	4 (0–11)	0.010

Table 1 Baseline characteristics of patients with and without breast arterial calcification (BAC) (continued)

Characteristic	Total	BAC positive	BAC negative	p-value
Laboratory parameters				
Estimated GFR (mL/min/1.73 m ²)	41.9±21.76	36.63±18.88	49.56±23.55	0.003
Serum calcium (mg/dL)	9.52±0.47	9.47±0.53	9.58±0.38	0.251
Serum phosphate (mg/dL)	3.68±0.66	3.68±0.64	3.68±0.69	0.997
Intact parathyroid hormone (pg/mL)	53 (42–103)	61 (42–115)	51 (41–89)	0.374
25-hydroxyvitamin D (ng/mL)	37.57±13.61	38.60±14.62	35.99±11.89	0.360
Alkaline phosphatase (U/L)	85.34±32.25	89.16±35.67	79.79±25.93	0.148
Hemoglobin A1c (%)	6.48±1.16	6.75±1.24	6.09±0.92	0.003
LDL cholesterol (mg/dL)	96.21±33.61	91.15±34.10	103.57±31.85	0.065
Triglycerides (mg/dL)	135.38±59.20	129.23±55.72	144.54±63.62	0.202

BAC, breast arterial calcification; GFR, glomerular filtration rate; CAC, coronary artery calcification; AAC, abdominal aortic calcification; LDL, low-density lipoprotein

Prevalence of BAC in CKD and Associated Factors

When stratified by CKD stage, the prevalence of BAC increased significantly with worsening kidney function. BAC was detected in 9 patients (39.1%) with CKD stage 2, 26 patients (56.5%) with CKD stage 3, and 26 patients (76.5%) with CKD stages 4–5 ($P=0.017$) (**Figure 2**). In contrast, no significant increase in the prevalence of CAC or AAC was observed across advancing CKD stages.

Consistently, in multivariable logistic regression analysis adjusted for traditional cardiovascular risk factors—including age, hypertensive nephropathy, diabetes mellitus, body mass index, systolic blood pressure, and statin use—the presence of BAC remained independently associated with lower eGFR (adjusted HR 5.67; 95% CI 1.78–17.83; $P=0.003$) and older age (adjusted HR 1.09; 95% CI 1.02–1.17; $P < 0.001$) (**Table 2**).

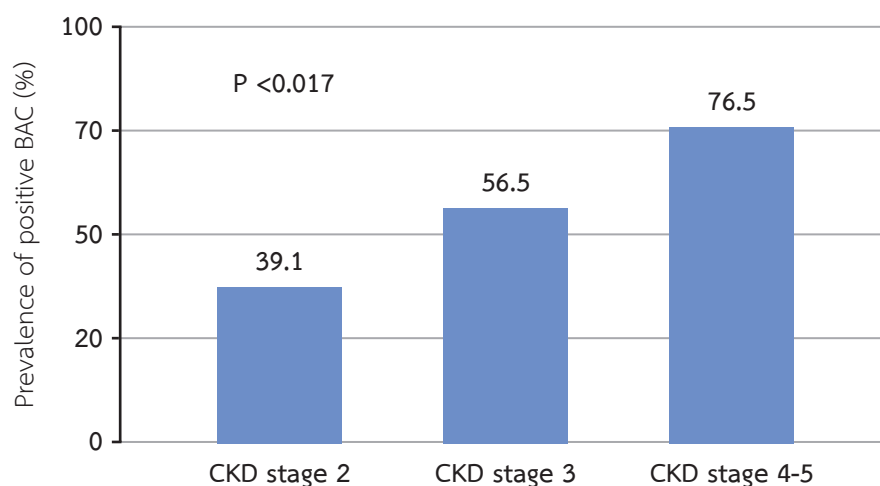


Figure 2 Prevalence of breast arterial calcification according to stages of chronic kidney disease
BAC, breast arterial calcification; CKD, chronic kidney disease

Table 2 Univariate and multivariate logistic regression analyses of factors associated with breast arterial calcification

Variable	Univariate			Multivariate		
	Unadjusted HR	95% CI	p-value	Adjusted HR	95% CI	p-value
Age (per year)	1.10	1.02–1.18	0.005	1.09	1.02–1.17	0.019
Body mass index (kg/m ²)	1.01	0.90–1.13	0.845	0.99	0.87–1.14	0.898
Systolic blood pressure (mmHg)	1.04	1.00–1.09	0.036	1.03	0.99–1.08	0.144
Diastolic blood pressure (mmHg)	0.98	0.94–1.03	0.452	-	-	-
Type 2 diabetes mellitus	1.80	0.65–5.03	0.262	1.56	0.46–5.27	0.475
Hypertension	1.64	0.40–6.80	0.492	1.06	0.19–5.73	0.951
Dyslipidemia	0.99	0.25–3.87	0.984	-	-	-
Coronary heart disease	1.00	0.00–1.00	1.000	-	-	-
Cerebrovascular disease	1.00	0.00–1.00	1.000	-	-	-
Statin therapy	0.67	0.14–3.28	0.624	0.51	0.10–2.72	0.434
Estimated GFR <30 mL/min/1.73 m ²	3.16	1.26–7.94	0.015	5.67	1.78–17.83	0.003

Multivariate analysis was adjusted for age, type 2 diabetes mellitus, hypertensive nephropathy, body mass index, systolic blood pressure, baseline estimated glomerular filtration rate (eGFR), and statin therapy. HR, hazard ratio; CI, confidence interval; GFR, glomerular filtration rate.

Predictive Value of BAC for Coronary Artery Calcification in CKD

In the overall CKD population, the sensitivity and accuracy of BAC >0 for identifying CAC >100 were 67.2% and 60.8%, respectively. Among patients with eGFR <60 mL/min/1.73 m², the sensitivity of BAC increased to 70.0%. Notably, in patients with advanced CKD (eGFR <30 mL/min/1.73 m²), BAC demonstrated markedly higher sensitivity (85.7%) and accuracy (67.6%). When a BAC score >2 was applied in this subgroup, diagnostic accuracy further improved to 73.5%. These findings indicate that BAC's predictive performance for CAC

increases as kidney function declines.

Receiver operating characteristic (ROC) curve analysis was performed to assess the ability of BAC and AAC to predict CAC >100 in patients with CKD. Both BAC and AAC scores were significantly associated with CAC, with area under the curve (AUC) values of 0.67 (95% CI 0.57–0.78; P=0.004) and 0.84 (95% CI 0.76–0.92; P <0.001), respectively (**Figure 3**). Importantly, the combined model incorporating both BAC and AAC demonstrated superior predictive performance for CAC (AUC 0.88; 95% CI 0.81–0.96; P=0.003).

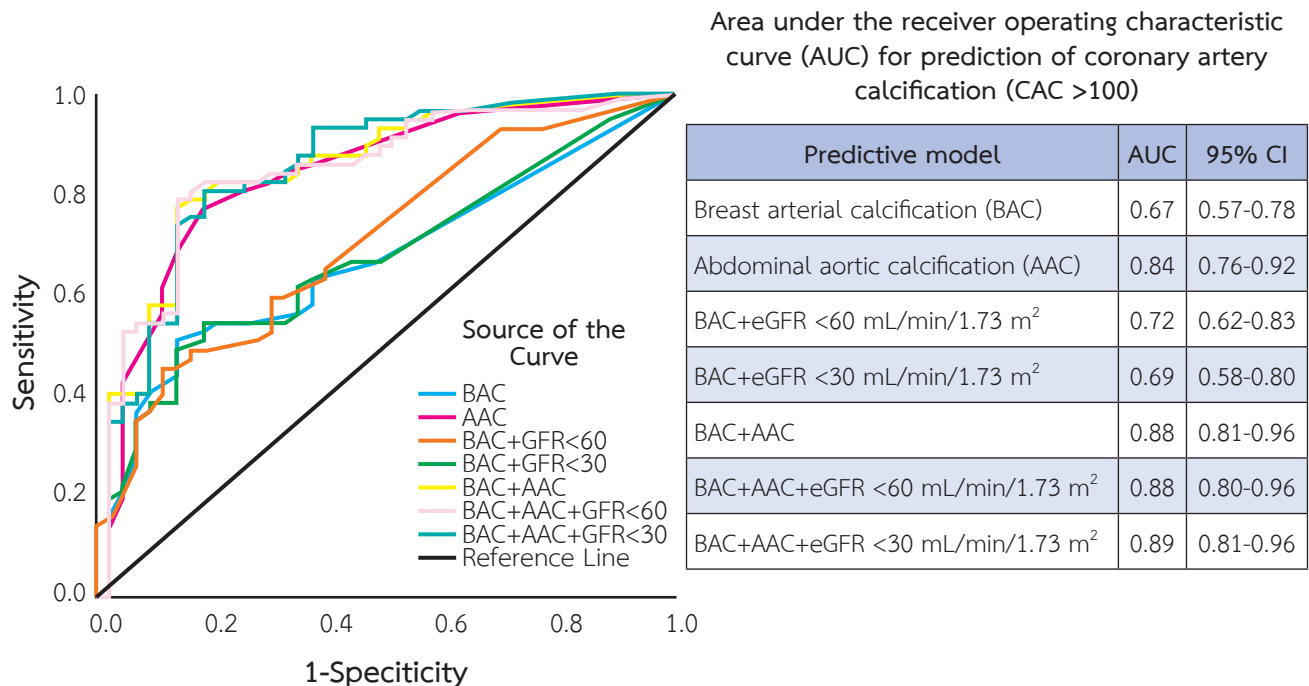


Figure 3 Receiver operating characteristic curve showing the area under the curve of breast arterial calcification for predicting the coronary artery calcification score >100.

AUC, area under the curve; CI, confidence interval; BAC, breast arterial calcification; AAC, abdominal aortic calcification; eGFR, estimated glomerular filtration rate; CAC, coronary artery calcification.

Discussion

This study demonstrates that the prevalence and severity of BAC increased significantly with declining kidney function. BAC is a feasible and clinically meaningful imaging marker for CAC in patients with CKD. Combined AAC and BAC increased the predictive value for CAC. These findings support the role of BAC as a surrogate marker of CAC.

The findings of the present study are consistent with prior reports showing a higher burden of BAC, exclusively medial calcification, in patients with CKD and ESKD.^{6,13,14} Importantly, BAC was independently associated with older age and reduced eGFR after adjustment for traditional cardiovascular risk factors. This observation highlights the strong link between CKD-related disturbances in mineral metabolism and medial arterial calcification. In CKD, abnormalities in phosphate handling, calcium balance, and inhibitors of vascular calcification promote osteogenic transformation of vascular smooth muscle cells, leading to progressive

medial calcification and arterial stiffness.^{15,16} The strong association between BAC and kidney function underscores its potential utility as a CKD-specific vascular risk marker, rather than a marker of atherosclerotic disease alone.^{17,18}

From a clinical perspective, our findings have several implications. First, BAC assessment may provide a readily available, noninvasive screening tool for vascular calcification in women with CKD, particularly those with advanced disease. Mammography is already routinely performed for breast cancer screening in women aged >40 years, a population that overlaps substantially with individuals at risk for CKD and cardiovascular disease. The incidental detection of BAC, therefore, offers an opportunity for opportunistic cardiovascular risk stratification without additional cost, radiation exposure, or imaging procedures.

BAC demonstrated increasing sensitivity and accuracy for predicting CAC as kidney function declined. This suggests that BAC may be particularly useful in

identifying high-risk CKD patients who may benefit from intensified cardiovascular risk modification, closer monitoring, or further cardiovascular evaluation. In resource-limited settings where CT-based CAC assessment is not routinely available, BAC could serve as a pragmatic alternative marker to guide clinical decision-making.

Although AAC showed superior predictive performance for CAC, BAC remains more specific for medial calcification, which is the dominant form of vascular calcification in CKD.⁷ The improved predictive performance observed with the combined BAC and AAC model supports a multimodal imaging approach that integrates information from different vascular beds to better characterize calcification burden and cardiovascular risk in CKD.^{19, 20}

Several limitations of this study should be acknowledged. First, the study population consisted exclusively of women, limiting the generalizability of the findings to male patients. However, this reflects the inherent limitation of BAC assessment, which is restricted to individuals undergoing mammography. Future studies should explore alternative markers of medial calcification applicable to both sexes.

BAC assessment is semi-quantitative and subject to interobserver variability. Although readings were performed by experienced radiologists blinded to clinical data, the lack of automated or standardized scoring systems may introduce measurement bias. The development and validation of automated or artificial intelligence-based BAC quantification tools could enhance reproducibility and clinical applicability.

This study was cross-sectional, precluding causal inference and longitudinal outcome assessment. While BAC was associated with CAC, cardiovascular events, and kidney function, we were unable to evaluate its prognostic value for cardiovascular morbidity, mortality, or CKD progression.

CAC assessed by non-contrast CT reflects a composite of intimal and medial calcification and may not fully capture the medial calcification burden characteristic of CKD. Advanced imaging techniques capable of differentiating calcification compartments may further

clarify the relationship between BAC and true medial vascular calcification.

Finally, the sample size was relatively modest and derived from a single-center cohort, which may limit external validity. Larger, multicenter studies are warranted to confirm these findings across diverse CKD populations.

Conclusion

The presence and severity of breast arterial calcification increase markedly with advancing CKD and are independently associated with older age and reduced kidney function. The combination of BAC and AAC demonstrates good predictive performance for CAC, particularly in patients with advanced CKD. BAC is a noninvasive, readily available imaging marker that may complement existing modalities for assessing vascular calcification burden in female patients with CKD.

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The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Conflict of Interest

The authors declare that they have no financial or non-financial conflicts of interest.

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