

The severity and predictors of abdominal aortic calcification among hemodialysis patients: a cross-sectional study from Palestine

Maisam Gharayba · Ahmad Katbeh · Basem Bali · Ali Shakhshir  · Qutaiba Ja'far A. Mahmoud · Mohanad Hassan

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Abstract

Abdominal aortic calcification (AAC) is a clinically important marker of systemic vascular disease and cardiovascular risk in patients receiving maintenance hemodialysis. Evidence from Palestine remains limited. This study assessed the prevalence and severity of AAC and identified factors associated with increasing calcification burden among Palestinian hemodialysis patients. This single-center cross-sectional study was conducted at An-Najah National University Hospital, Palestine, between August 2025 and January 2026. Adults receiving maintenance hemodialysis for at least 6 months were recruited using convenience sampling. AAC was assessed on lateral lumbar radiographs using the validated 24-point Kauppila score and categorized as no calcification (0), mild-to-moderate calcification (1–6), or severe calcification (≥ 7). Clinical, laboratory, medication, and radiographic data were analyzed using appropriate parametric and non-parametric tests. A multivariable ordinal logistic regression model was used to identify independent predictors of increasing AAC severity, and a sensitivity model additionally adjusted for serum calcium and phosphate. A total of 163 patients were included, representing 95.9% of the prespecified target sample. Overall, 35.6% had no calcification, 23.9% had mild-to-moderate calcification, and 40.5% had severe AAC. Age increased progressively across AAC categories ($p < 0.001$) and showed a moderate positive correlation with total AAC score (Spearman's $\rho = 0.531$, $p < 0.001$). Serum albumin, creatinine, fasting glucose, potassium, and platelet count were associated with AAC in unadjusted group comparisons or correlation analyses. In the primary multivariable model, age was the only independent predictor of increasing AAC severity; each additional year of age was associated with a 7.9% increase in the odds of belonging to a higher AAC category (adjusted OR 1.079, 95% CI 1.049–1.108; $p < 0.001$). The proportional-odds assumption was satisfied ($p = 0.620$), and model fit was acceptable. In the CKD-MBD sensitivity analysis, age remained significant (adjusted OR 1.082, 95% CI 1.051–1.114; $p < 0.001$), whereas phosphate was not significant and calcium showed only a borderline association. Associations involving diabetes and albumin were sensitive to model specification and were therefore interpreted cautiously. AAC was highly prevalent in this Palestinian hemodialysis cohort, with severe calcification affecting approximately two in five patients. Age was the most robust and consistently significant predictor of increasing AAC severity. Lateral

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Maisam Gharayba, Ahmad Katbeh and Basem Bali contributed equally to this work.



lumbar radiography may provide an accessible adjunct to cardiovascular risk assessment, although prospective multicenter studies are needed to confirm clinical utility and clarify modifiable risk pathways.

Keywords Abdominal aortic calcification · Hemodialysis · Vascular calcification · Kauppila score · Ageing · CKD-MBD · Palestine

Abbreviations

AAC	Abdominal aortic calcification
CKD	Chronic kidney disease
CKD-MBD	Chronic kidney disease–mineral and bone disorder
HD	Hemodialysis
ICC	Intraclass correlation coefficient
IQR	Interquartile range
NNUH	An-Najah National University Hospital
OR	Odds ratio
PTH	Parathyroid hormone
SD	Standard deviation

Introduction

Cardiovascular disease remains the leading cause of morbidity and mortality among patients with chronic kidney disease (CKD), particularly those receiving maintenance hemodialysis (HD)¹. One of the most important contributors to cardiovascular complications in this population is vascular calcification, which involves pathological deposition of calcium-phosphate complexes within the arterial wall. Vascular calcification can occur in both the intimal and medial layers of arteries, with medial calcification being particularly common in patients with advanced CKD and end-stage renal disease (ESRD)¹.

Abdominal aortic calcification (AAC) represents a common manifestation of systemic vascular calcification and has been increasingly recognized as an important marker of cardiovascular risk among dialysis patients². The development of vascular calcification in CKD is driven by a combination of traditional cardiovascular risk factors and CKD-specific abnormalities such as disturbances in calcium–phosphate metabolism, chronic inflammation, and prolonged dialysis exposure¹.

Radiological imaging methods, including lateral lumbar radiography and computed tomography, are commonly used to assess the presence and severity of AAC in patients with renal disease³. Early identification of AAC is clinically important because it has been associated with increased cardiovascular events and mortality in dialysis populations⁴.

Several studies conducted in Western countries and Europe have reported a high prevalence of vascular calcification among patients undergoing maintenance hemodialysis. For instance, an international cross-sectional study of hemodialysis patients demonstrated that abdominal aortic calcification was present in approximately three-quarters of the examined patients, highlighting the widespread burden of calcification in this population².

Similarly, studies in Asian populations have shown that vascular calcification is frequently observed among dialysis patients and may worsen with increasing dialysis duration and metabolic abnormalities⁵. Research conducted in East Asian hemodialysis cohorts has also demonstrated that older age, elevated blood pressure, and abnormal mineral metabolism are significant predictors of abdominal aortic calcification⁶.

Evidence from African dialysis populations likewise indicates that vascular calcification is prevalent and is associated with cardiovascular disease history and demographic risk factors⁷. Observational studies have further

shown that abdominal aortic calcification can affect more than two-thirds of hemodialysis patients, demonstrating the significant burden of calcific vascular disease in ESRD⁸.

Several biological markers have also been investigated in relation to AAC, including circulating proteins and metabolic factors that may influence vascular mineralization processes⁹. In addition, mineral metabolism abnormalities such as magnesium imbalance have been linked to the progression of abdominal aortic calcification among patients with advanced CKD¹⁰.

These findings collectively highlight that vascular calcification in dialysis patients is a multifactorial process influenced by demographic, clinical, and biochemical factors¹. Despite the increasing global evidence regarding the burden of vascular calcification in dialysis patients, data from Middle Eastern countries remain relatively limited². In particular, there is a notable lack of published studies evaluating abdominal aortic calcification among patients undergoing hemodialysis in Palestine.

Local epidemiological data are important because demographic characteristics, dialysis practices, and comorbid conditions may vary across regions and influence the prevalence and determinants of vascular calcification. Understanding the burden of AAC and its associated factors in Palestinian dialysis patients could therefore contribute to improved cardiovascular risk stratification and targeted preventive strategies. Therefore, this study aimed to assess the prevalence and severity of abdominal aortic calcification among Palestinian patients undergoing hemodialysis and to determine the factors associated with its occurrence.

Methodology

Study design and setting

This was a cross-sectional observational study conducted at the Hemodialysis Unit (HD) of An-Najah National University Hospital (NNUH) in Nablus, Palestine. NNUH hosts the largest HD center in the North of the West Bank, which follows approximately 300 patients on chronic HD. Data collection took place between August 2025 and January 2026. The study aimed to determine the prevalence and severity of abdominal aortic calcification (AAC) among HD patients using the AAC score, and to examine associations between AAC severity and sociodemographic, clinical, and laboratory factors as potential determinants.

Study population, sample size, and sampling technique

During the study period, 304 patients were receiving hemodialysis at An-Najah National University Hospital and constituted the initial source population. Because published estimates of abdominal aortic calcification prevalence vary considerably across hemodialysis populations, a conservative anticipated prevalence of 50% was used for the sample-size calculation, as this yields the largest required sample when the true prevalence is uncertain. Using a 95% confidence level, a 5% margin of error, and finite-population correction for a source population of 304 patients, the calculated minimum sample size was 170 participants. Of the 304 initially identified patients, 81 did not meet the eligibility criteria and were excluded: hemodialysis duration of less than six months ($n=19$), hybrid hemodialysis and peritoneal dialysis treatment ($n=12$), severe cognitive impairment affecting the capacity to provide informed consent ($n=12$), age younger than 18 years ($n=11$), and incomplete radiological assessment ($n=27$). Therefore, 223 patients met the eligibility criteria. Eligible patients who were available during the data-collection period were approached using convenience sampling. Of these, 60 declined participation, while 163 provided written informed consent, underwent lateral lumbar radiography, and were included in the final analysis. The participation rate among eligible patients was 73.1% (163/223). The final sample represented 95.9% of the calculated minimum sample size (163/170).

Inclusion and exclusion criteria

Patients were eligible for inclusion if they were receiving maintenance hemodialysis at NNUH for at least six months and were able to provide informed consent¹¹. All participants included in the study provided written informed consent for both data collection and radiographic assessment. Patients were excluded if they were younger than 18 years, were receiving acute dialysis, or were receiving hybrid dialysis combining hemodialysis and peritoneal dialysis. Patients with severe cognitive impairment affecting their ability to provide valid informed consent or those with incomplete radiological assessment were also excluded.

Study variables, tools, and data collection

Data were collected at a single time point using a structured data collection form developed for the study and completed from patient interviews, hospital electronic medical records, and laboratory and radiographic records. The collected variables included demographic, clinical, laboratory, medication-related, and radiographic data. Demographic variables comprised age, sex, and area of residency. Clinical variables included hypertension, diabetes mellitus, smoking status, history of myocardial infarction, number of previous cardiac catheterization procedures, duration of hemodialysis, body mass index, height, and post-hemodialysis weight. Hypertension was considered present when a physician-documented diagnosis was recorded in the medical record or when office systolic blood pressure was ≥ 140 mmHg and/or diastolic blood pressure was ≥ 90 mmHg¹². Diabetes mellitus was considered present when a physician-documented diagnosis was recorded or when available laboratory records showed fasting plasma glucose ≥ 126 mg/dL or HbA1c $\geq 6.5\%$ ¹³. Both conditions were analyzed as binary nominal variables. Laboratory variables included serum albumin, blood urea nitrogen, creatinine, ferritin, fasting glucose, calcium, phosphate, potassium, sodium, carbon dioxide, chloride, magnesium, total iron-binding capacity, serum iron, transferrin, transferrin saturation, white blood cell count, platelet count, and hemoglobin. Available CKD-mineral and bone disorder-related variables included serum calcium, phosphate, and magnesium, together with recorded use of calcium carbonate, calcium acetate, sevelamer, and alfacalcidol. Parathyroid hormone measurements were not consistently available within a standardized period around radiographic assessment. Detailed data regarding calcimimetic therapy, vitamin D status, and cumulative vitamin D exposure were also unavailable and were therefore not analyzed. Hemodialysis duration was available and included in the analyses. However, standardized contemporaneous measures of dialysis adequacy, including Kt/V or urea reduction ratio, residual kidney function, vascular access type, and detailed dialysate composition were not consistently available for all participants and were therefore not analyzed. Medication data were extracted from the electronic medical records and included drugs prescribed for at least three months before enrollment; these were grouped into major therapeutic classes and coded as binary variables (yes/no). The primary outcome variable was abdominal aortic calcification (AAC), assessed using lateral lumbar spine radiography^{14,15}. Abdominal aortic calcification was assessed using lateral lumbar radiographs obtained in the standing lateral position. Calcific deposits along the anterior and posterior walls of the abdominal aorta adjacent to vertebral levels L1–L4 were evaluated using the validated Kauppila semi-quantitative scoring system. At each vertebral level, the anterior and posterior aortic walls were scored separately from 0 to 3 according to the longitudinal extent of calcification, producing a total AAC score ranging from 0 to 24. The radiographs were reviewed by two radiologists with different levels of experience: a specialist radiologist and a senior radiologist. Both were blinded to participants' demographic, clinical, and laboratory information. The specialist radiologist performed the primary scoring, and the specialist's score was used in the statistical analysis. The senior radiologist reviewed the radiographs as an additional expert assessment, particularly when clarification of the calcification extent was required¹¹. Because separate independent scores from both radiologists were not systematically retained, formal inter-rater reliability statistics could not be calculated. For analysis, AAC was categorized as no calcification (score 0), mild-to-moderate calcification (score 1–6), and severe calcification (score ≥ 7).

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows, version 22.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality before analysis. Normally distributed continuous variables were summarized as mean $\pm$ standard deviation (SD), whereas non-normally distributed continuous variables were presented as median and interquartile range (IQR). Categorical variables were expressed as frequencies and percentages. Comparisons across abdominal aortic calcification (AAC) categories (no AAC, mild-to-moderate AAC, and severe AAC) were performed according to variable type and distribution. One-way analysis of variance (ANOVA) was used for normally distributed continuous variables, while the Kruskal–Wallis test was used for non-normally distributed continuous variables. Categorical variables were compared using the chi-square test or Fisher’s exact test, as appropriate. Correlations between the total AAC score (range 0–24) and continuous variables were assessed using Pearson’s correlation coefficient for normally distributed variables and Spearman’s rank correlation coefficient for non-normally distributed variables. To identify factors independently associated with increasing AAC severity, an ordinal logistic regression model with a logit link was fitted, with AAC ordered as no calcification, mild-to-moderate calcification, and severe calcification. Candidate predictors were selected a priori based on established clinical relevance and the findings of the bivariate and correlation analyses. Age and sex were retained as predefined demographic confounders. Diabetes mellitus, smoking status, and hemodialysis duration were included because of their established relationships with cardiovascular and vascular calcification risk. Serum albumin, creatinine, potassium, and platelet count were included because of their clinical relevance and observed associations in the preliminary analyses. Accordingly, the primary model included age, sex, diabetes mellitus, smoking status, hemodialysis duration, serum albumin, serum creatinine, serum potassium, and platelet count. A sensitivity analysis was additionally performed by adding serum calcium and serum phosphate to the primary ordinal logistic regression model because of their established relevance to chronic kidney disease–mineral and bone disorder. The proportional-odds assumption was formally evaluated using the test of parallel lines. Overall model significance was assessed using the likelihood-ratio test, while model fit was evaluated using Pearson and deviance goodness-of-fit statistics. Adjusted odds ratios with 95% confidence intervals were reported. Missing data were handled using complete-case analysis. All tests were two-sided, and $p < 0.05$ was considered statistically significant. For the sensitivity model, the test of parallel lines was interpreted cautiously because SPSS reported incomplete convergence of the general model.

Ethical considerations

The study protocol was approved by the Institutional Review Board of An-Najah National University, Nablus, Palestine (IRB Med. Oct. 2024/30), and was also reviewed by the relevant hospital and departmental authorities. All participants were informed about the purpose of the study and the content of the interview prior to enrollment, and written informed consent was obtained from each participant before data collection. Participation was entirely voluntary. All collected data were kept confidential and used solely for research purposes. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Results

During the study period, 304 patients receiving hemodialysis were initially identified. Of these, 81 did not meet the eligibility criteria and were excluded, leaving 223 eligible patients who were approached using convenience sampling. Sixty eligible patients declined participation, while 163 provided informed consent, underwent lateral lumbar radiography, and were included in the final analysis, corresponding to a participation rate of 73.1% among eligible patients. The achieved sample represented 95.9% of the prespecified minimum sample size of 170 participants (Fig. 1). The cohort was predominantly male (58.9%), and 65.0% of participants resided in cities.

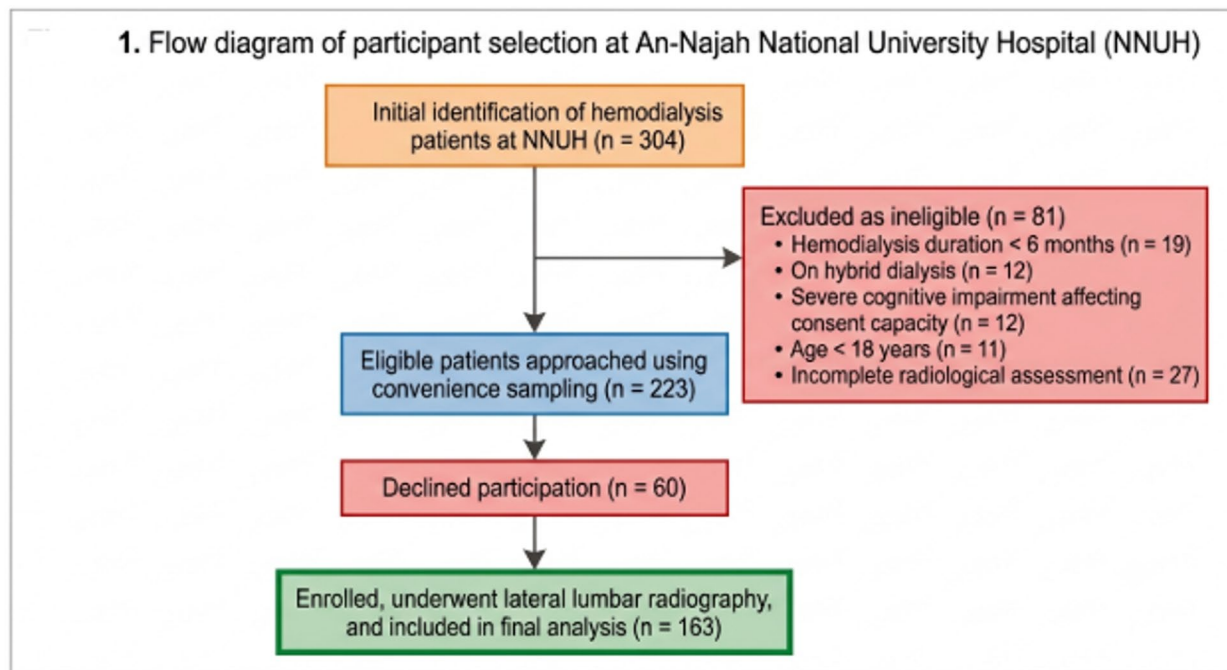


Fig. 1 Flow diagram of participant selection at An-Najah National University Hospital. Of 304 patients initially identified, 81 were excluded because they did not meet the eligibility criteria. Among 223 eligible patients approached using convenience sampling, 60 declined participation and 163 provided consent, underwent lateral lumbar radiography, and were included in the final analysis.

Diabetes mellitus was present in 50.9%, hypertension in 83.4%, and 39.9% were current smokers. Most participants had no previous myocardial infarction (68.7%). Regarding AAC severity, 58 participants (35.6%) had no calcification, 39 (23.9%) had mild-to-moderate calcification, and 66 (40.5%) had severe calcification. Thus, almost two-thirds of the cohort had evidence of AAC, with severe calcification affecting approximately two in five participants (Table 1).

The median total AAC score was 4.0 (Q1–Q3: 0.0–12.0), while the median age was 62.0 (49.0–69.0) years and the median duration of hemodialysis was 4.0 (3.0–7.0) years. Mean dry weight was 76.50 ± 18.45 kg. Mean serum phosphate, potassium, and sodium levels were 4.53 ± 1.35 , 4.81 ± 0.68 , and 137.41 ± 2.94 , respectively. The median serum albumin level was 3.93 (3.73–4.17), median creatinine was 8.24 (6.63–10.30), and median fasting glucose was 125.50 (98.20–178.00) (Table 2).

Crude associations between demographic and clinical characteristics, medication use, and AAC severity are presented in Table 3. Diabetes mellitus differed significantly across AAC severity categories ($p=0.014$). Significant differences were also observed in the distributions of atorvastatin ($p=0.011$), tamsulosin ($p=0.005$), aspirin ($p=0.039$), apixaban ($p=0.039$), clopidogrel ($p=0.025$), and calcium acetate use ($p=0.019$). These medication-related findings were based on unadjusted comparisons and should be interpreted descriptively, particularly because some exposure groups contained relatively few participants and may reflect underlying cardiovascular disease or treatment indication rather than direct medication effects. Sex, residence, hypertension, smoking status, previous myocardial infarction, cardiac catheterization history, and most other medication variables did not differ significantly across AAC categories (Table 3).

With respect to CKD–mineral and bone disorder-related treatment, calcium acetate use differed significantly across AAC categories, whereas calcium carbonate, alfacalcidol, and sevelamer use did not. These crude treatment associations were not interpreted as causal medication effects (Table 3).

Among normally distributed variables assessed using one-way ANOVA, serum potassium differed significantly across AAC categories ($p=0.020$), with lower mean potassium levels observed in the severe AAC group.

Table 1 Participant clinical characteristics, comorbidities, and medication use ($N = 163$).

Variable	Category	Frequency, n (%)
Gender	Male	96 (58.9%)
	Female	67 (41.1%)
Place of residence	City	106 (65.0%)
	Village	39 (23.9%)
	Camp	18 (11.0%)
	No diabetes mellitus	80 (49.1%)
Diabetes mellitus (DM)	Diabetes mellitus	83 (50.9%)
Hypertension (HTN)	No	27 (16.6%)
	Yes	136 (83.4%)
History of myocardial infarction	None	112 (68.7%)
	1 episode	48 (29.4%)
	2 episodes	2 (1.2%)
	3 episodes	1 (0.6%)
Smoking status	Non-smoker	98 (60.1%)
	Smoker	65 (39.9%)
Number of previous cardiac catheterizations	Once	31 (19.0%)
	Twice	19 (11.7%)
	More than two	113 (69.3%)
Carvedilol	No	144 (88.3%)
	Yes	19 (11.7%)
Bisoprolol	No	77 (47.2%)
	Yes	86 (52.8%)
Nifedipine	No	150 (92.0%)
	Yes	13 (8.0%)
Amlodipine	No	75 (46.0%)
	Yes	88 (54.0%)
Losartan	No	160 (98.2%)
	Yes	3 (1.8%)
Furosemide	No	100 (61.3%)
	Yes	63 (38.7%)
Hydralazine	No	102 (62.6%)
	Yes	61 (37.4%)
Atorvastatin	No	75 (46.0%)
	Yes	88 (54.0%)
Doxazosin	No	131 (80.4%)
	Yes	32 (19.6%)
Tamsulosin	No	144 (88.3%)
	Yes	19 (11.7%)
Calcium carbonate	No	118 (72.4%)
	Yes	45 (27.6%)
Alfacalcidol	No	25 (15.3%)
	Yes	138 (84.7%)
Insulin	No	136 (83.4%)
	Yes	27 (16.6%)
Vildagliptin	No	152 (93.3%)
	Yes	11 (6.7%)
Esomeprazole	No	117 (71.8%)
	Yes	46 (28.2%)
Aspirin	No	85 (52.1%)
	Yes	78 (47.9%)
Apixaban	No	153 (93.9%)
	Yes	10 (6.1%)
Clopidogrel	No	133 (81.6%)
	Yes	30 (18.4%)

Table 1 (continued)

Variable	Category	Frequency, <i>n</i> (%)
Warfarin	No	161 (98.8%)
	Yes	2 (1.2%)
Isosorbide mononitrate	No	152 (93.3%)
	Yes	11 (6.7%)
Levothyroxine	No	152 (93.3%)
	Yes	11 (6.7%)
Prednisone	No	157 (96.3%)
	Yes	6 (3.7%)
Calcium acetate	No	131 (80.4%)
	Yes	32 (19.6%)
Sevelamer	No	122 (74.8%)
	Yes	41 (25.2%)
Allopurinol	No	142 (87.1%)
	Yes	21 (12.9%)
Premixed human insulin	No	140 (85.9%)
	Yes	23 (14.1%)
Darbepoetin alfa	No	112 (68.7%)
	Yes	51 (31.3%)
Iron sucrose	No	130 (79.8%)
	Yes	33 (20.2%)
AAC category	No calcification	58 (35.6%)
AAC category	Mild to moderate calcification	39 (23.9%)
AAC category	Severe calcification	66 (40.5%)

Table 2 Baseline anthropometric and laboratory measurements (mean $\pm$ SD or median [Q1–Q3])

Variable	Mean $\pm$ SD
Dry weight	76.50 $\pm$ 18.45
Carbon dioxide	21.14 $\pm$ 3.13
Phosphate	4.53 $\pm$ 1.35
Potassium	4.81 $\pm$ 0.68
Sodium	137.41 $\pm$ 2.94
Total iron binding capacity	203.98 $\pm$ 41.32
Transferrin	163.43 $\pm$ 31.14
	Median (Q1–Q3)
AAC scores	4.000 (0.000–12.000)
Duration of hemodialysis (years)	4.000 (3.000–7.000)
Age	62.000 (49.000–69.000)
Serum calcium	9.000 (8.700–9.500)
Serum chloride s	96.900 (94.800–99.300)
Serum iron	52.850 (40.900–68.000)
Fasting glucose	125.500 (98.200–178.000)
White blood cell count	6.655 (5.510–8.090)
Hemoglobin (HGB)	10.800 (10.000–11.600)
Transferrin saturation	26.000 (20.000–34.000)
Ferritin	721.500 (432.000–1042.000)
Creatinine	8.240 (6.630–10.300)
Albumin	3.930 (3.730–4.170)
Blood urea nitrogen	53.700 (46.100–67.000)
Magnesium	2.5 (2.36–2.74)

Table 3 Association between clinical characteristics, medication use, and AAC severity.

Variable	Category	No calcification	Mild to moderate calcification	Severe calcification	<i>P</i> value
Gender	Male	35 (60.3%)	24 (61.5%)	37 (56.1%)	0.826
	Female	23 (39.7%)	15 (38.5%)	29 (43.9%)	
Residency code	City	37 (63.8%)	20 (51.3%)	49 (74.2%)	0.162
	Village	13 (22.4%)	14 (35.9%)	12 (18.2%)	
	Camp	8 (13.8%)	5 (12.8%)	5 (7.6%)	
Diabetes mellitus (DM)	No diabetes mellitus	37 (63.8%)	18 (46.2%)	25 (37.9%)	0.014
	Diabetes mellitus	21 (36.2%)	21 (53.8%)	41 (62.1%)	
Hypertension (HTN)	No	12 (20.7%)	5 (12.8%)	10 (15.2%)	0.591
	Yes	46 (79.3%)	34 (87.2%)	56 (84.8%)	
History of myocardial infarction	None	45 (77.6%)	27 (69.2%)	40 (60.6%)	0.143
	1 episode	12 (20.7%)	11 (28.2%)	25 (37.9%)	
	2 episodes	1 (1.7%)	0 (0.0%)	1 (1.5%)	
	3 episodes	0 (0.0%)	1 (2.6%)	0 (0.0%)	
Smoking status	Non -smoker	32 (55.2%)	27 (69.2%)	39 (59.1%)	0.373
	smoker	26 (44.8%)	12 (30.8%)	27 (40.9%)	
Carvedilol	No	52 (89.7%)	32 (82.1%)	60 (90.9%)	0.365
	Yes	6 (10.3%)	7 (17.9%)	6 (9.1%)	
Bisoprolol	No	28 (48.3%)	19 (48.7%)	30 (45.5%)	0.931
	Yes	30 (51.7%)	20 (51.3%)	36 (54.5%)	
Nifedipine	No	55 (94.8%)	37 (94.9%)	58 (87.9%)	0.329
	Yes	3 (5.2%)	2 (5.1%)	8 (12.1%)	
Amlodipine	No	28 (48.3%)	12 (30.8%)	35 (53.0%)	0.079
	Yes	30 (51.7%)	27 (69.2%)	31 (47.0%)	
Losartan	No	58 (100.0%)	39 (100.0%)	63 (95.5%)	0.182
	Yes	0 (0.0%)	0 (0.0%)	3 (4.5%)	
Furosemide	No	40 (69.0%)	22 (56.4%)	38 (57.6%)	0.330
	Yes	18 (31.0%)	17 (43.6%)	28 (42.4%)	
Hydralazine	No	39 (67.2%)	25 (64.1%)	38 (57.6%)	0.527
	Yes	19 (32.8%)	14 (35.9%)	28 (42.4%)	
Atorvastatin	No	35 (60.3%)	18 (46.2%)	22 (33.3%)	0.011
	Yes	23 (39.7%)	21 (53.8%)	44 (66.7%)	
Doxazosin	No	43 (74.1%)	31 (79.5%)	57 (86.4%)	0.229
	Yes	15 (25.9%)	8 (20.5%)	9 (13.6%)	
Tamsulosin	No	57 (98.3%)	32 (82.1%)	55 (83.3%)	0.005
	Yes	1 (1.7%)	7 (17.9%)	11 (16.7%)	
Calcium carbonate	No	37 (63.8%)	32 (82.1%)	49 (74.2%)	0.130
	Yes	21 (36.2%)	7 (17.9%)	17 (25.8%)	
Alfacalcidol	No	7 (12.1%)	7 (17.9%)	11 (16.7%)	0.680
	Yes	51 (87.9%)	32 (82.1%)	55 (83.3%)	
Insulin	No	49 (84.5%)	33 (84.6%)	54 (81.8%)	0.900
	Yes	9 (15.5%)	6 (15.4%)	12 (18.2%)	
Vildagliptin	No	54 (93.1%)	37 (94.9%)	61 (92.4%)	1.000
	Yes	4 (6.9%)	2 (5.1%)	5 (7.6%)	
Esomeprazole	No	40 (69.0%)	28 (71.8%)	49 (74.2%)	0.809
	Yes	18 (31.0%)	11 (28.2%)	17 (25.8%)	
Aspirin	No	38 (65.5%)	18 (46.2%)	29 (43.9%)	0.039
	Yes	20 (34.5%)	21 (53.8%)	37 (56.1%)	
Apixaban	No	57 (98.3%)	38 (97.4%)	58 (87.9%)	0.039
	Yes	1 (1.7%)	1 (2.6%)	8 (12.1%)	
Clopidogrel	No	53 (91.4%)	32 (82.1%)	48 (72.7%)	0.025
	Yes	5 (8.6%)	7 (17.9%)	18 (27.3%)	
Warfarin	No	58 (100.0%)	39 (100.0%)	64 (97.0%)	0.344
	Yes	0 (0.0%)	0 (0.0%)	2 (3.0%)	

Table 3 (continued)

Variable	Category	No calcification	Mild to moderate calcification	Severe calcification	<i>P</i> value
Isosorbide mononitrate	No	56 (96.6%)	37 (94.9%)	59 (89.4%)	0.316
	Yes	2 (3.4%)	2 (5.1%)	7 (10.6%)	
Levothyroxine	No	55 (94.8%)	38 (97.4%)	59 (89.4%)	0.291
	Yes	3 (5.2%)	1 (2.6%)	7 (10.6%)	
Prednisone	No	55 (94.8%)	38 (97.4%)	64 (97.0%)	0.765
	Yes	3 (5.2%)	1 (2.6%)	2 (3.0%)	
Calcium acetate	No	43 (74.1%)	28 (71.8%)	60 (90.9%)	0.019
	Yes	15 (25.9%)	11 (28.2%)	6 (9.1%)	
Sevelamer	No	41 (70.7%)	33 (84.6%)	48 (72.7%)	0.264
	Yes	17 (29.3%)	6 (15.4%)	18 (27.3%)	
Allopurinol	No	51 (87.9%)	35 (89.7%)	56 (84.8%)	0.834
	Yes	7 (12.1%)	4 (10.3%)	10 (15.2%)	
Premixed human insulin	No	54 (93.1%)	31 (79.5%)	55 (83.3%)	0.101
	Yes	4 (6.9%)	8 (20.5%)	11 (16.7%)	
Darbepoetin alfa	No	38 (65.5%)	24 (61.5%)	50 (75.8%)	0.255
	Yes	20 (34.5%)	15 (38.5%)	16 (24.2%)	
Iron sucrose	No	47 (81.0%)	27 (69.2%)	56 (84.8%)	0.150
	Yes	11 (19.0%)	12 (30.8%)	10 (15.2%)	
Cardiac catheterization number	Once	11 (19.0%)	7 (17.9%)	13 (19.7%)	0.684
	Twice	4 (6.9%)	5 (12.8%)	10 (15.2%)	
	More than two	43 (74.1%)	27 (69.2%)	43 (65.2%)	

No statistically significant differences were identified for dry weight, carbon dioxide, phosphate, sodium, total iron-binding capacity, or transferrin (all $p > 0.05$) (Table 4).

For non-normally distributed variables assessed using the Kruskal–Wallis test, age increased progressively across AAC severity categories, from a median of 50.5 years in participants without calcification to 61.0 years in those with mild-to-moderate calcification and 66.0 years in those with severe calcification ($p < 0.001$). Serum albumin also decreased across the three groups ($p = 0.014$), while creatinine was lower in participants with greater AAC severity ($p = 0.001$). Fasting glucose increased across AAC categories ($p = 0.002$). No significant between-group differences were observed for height, body mass index, hemodialysis duration, magnesium, blood urea nitrogen, calcium, ferritin, transferrin saturation, hemoglobin, white blood cell count, serum iron, or chloride (Table 4).

Notably, serum calcium, phosphate, and magnesium were not significantly associated with AAC severity in the unadjusted group comparisons (all $p > 0.05$) (Table 4).

Spearman correlation analysis demonstrated a moderate positive correlation between total AAC score and age (Spearman's $\rho = 0.531$, $p < 0.001$). AAC score was also weakly positively correlated with fasting glucose ($\rho = 0.235$, $p = 0.003$) and weakly negatively correlated with serum albumin ($\rho = -0.215$, $p = 0.006$) and creatinine ($\rho = -0.260$, $p = 0.001$). No statistically significant Spearman correlations were observed with patient height, body mass index, hemodialysis duration, magnesium, hemoglobin, chloride, iron, white blood cell count, transferrin saturation, ferritin, calcium, or blood urea nitrogen (Table 5).

In the Pearson correlation analysis, total AAC score was weakly negatively correlated with potassium ($r = -0.199$, $p = 0.011$) and weakly positively correlated with platelet count ($r = 0.163$, $p = 0.038$). No significant correlations were identified with dry weight, carbon dioxide, phosphate, sodium, total iron-binding capacity, or transferrin (Table 6).

The multivariable ordinal logistic regression analysis included 162 participants with complete data. The dependent variable was ordered as no calcification, mild-to-moderate calcification, and severe calcification. The model included age, sex, diabetes mellitus, smoking status, hemodialysis duration, serum albumin, serum creatinine, serum potassium, and platelet count. The final model significantly improved fit compared with the intercept-only model (likelihood-ratio $\chi^2(9) = 60.044$, $p < 0.001$). Pearson ($p = 0.460$) and deviance ($p = 0.832$)

Table 4 Comparison of anthropometric and laboratory variables across AAC severity categories.

Variable	No AAC, score 0	Mild-to-moderate AAC, score 1–6	Severe AAC, score ≥ 7	ANOVA or Kruskal–Wallis test <i>p</i> value
Dry weight	73.90 $\pm$ 20.96 (<i>n</i> = 58)	78.62 $\pm$ 17.45 (<i>n</i> = 39)	77.53 $\pm$ 16.58 (<i>n</i> = 66)	0.394
Carbon dioxide CO ₂	21.24 $\pm$ 3.30 (<i>n</i> = 58)	21.09 $\pm$ 2.87 (<i>n</i> = 39)	21.09 $\pm$ 3.15 (<i>n</i> = 65)	0.956
Phosphate	4.72 $\pm$ 1.33 (<i>n</i> = 58)	4.35 $\pm$ 1.31 (<i>n</i> = 39)	4.48 $\pm$ 1.40 (<i>n</i> = 65)	0.400
Potassium	4.94 $\pm$ 0.59 (<i>n</i> = 58)	4.93 $\pm$ 0.71 (<i>n</i> = 39)	4.63 $\pm$ 0.70 (<i>n</i> = 65)	0.020
Sodium	137.45 $\pm$ 2.68 (<i>n</i> = 58)	137.81 $\pm$ 2.80 (<i>n</i> = 39)	137.13 $\pm$ 3.25 (<i>n</i> = 65)	0.525
Total iron binding capacity (TIBC)	202.11 $\pm$ 36.97 (<i>n</i> = 37)	212.41 $\pm$ 36.15 (<i>n</i> = 24)	200.73 $\pm$ 47.68 (<i>n</i> = 41)	0.519
Transferrin	168.03 $\pm$ 29.06 (<i>n</i> = 58)	162.00 $\pm$ 26.56 (<i>n</i> = 39)	160.18 $\pm$ 35.17 (<i>n</i> = 65)	0.360
Age	50.50 (36.00–62.00)	61.00 (52.00–68.00)	66.00 (61.00–73.00)	<0.001
Height	166.00 (158.00–173.00)	170.00 (160.50–175.00)	165.00 (159.00–172.00)	0.551
Body mass index (BMI)	25.27 (22.40–29.94)	26.44 (24.92–30.23)	27.37 (24.51–30.93)	0.149
Duration of hemodialysis (years)	5.00 (3.00–7.00)	4.00 (2.75–6.50)	4.00 (3.00–8.00)	0.846
Magnesium	2.53 (2.41–2.82)	2.57 (2.37–2.71)	2.53 (2.30–2.74)	0.415
Albumin	4.00 (3.82–4.17)	3.96 (3.74–4.20)	3.85 (3.61–4.13)	0.014
Blood urea nitrogen	56.95 (47.70–69.40)	54.40 (45.20–65.80)	52.20 (44.40–66.40)	0.334
Serum calcium	8.98 (8.60–9.50)	9.00 (8.70–9.40)	9.20 (8.70–9.70)	0.461
Creatinine	9.84 (7.39–10.90)	7.92 (6.47–9.59)	7.62 (5.80–9.22)	0.001
Ferritin	800.50 (391.00–1012.00)	721.00 (453.50–1082.50)	688.00 (432.00–1080.00)	0.883
Transferrin saturation	25.00 (19.00–36.00)	27.00 (22.50–29.00)	25.00 (20.00–34.00)	0.949
Hemoglobin (HGB)	10.80 (10.00–11.70)	10.80 (10.00–11.45)	10.80 (9.80–11.60)	0.926
White blood cell count	6.60 (5.10–8.18)	6.32 (5.21–8.02)	6.90 (6.00–8.09)	0.385
Fasting glucose	105.00 (89.70–136.00)	134.00 (112.50–206.50)	141.00 (104.00–193.00)	0.002
Serum iron	54.75 (41.00–69.00)	52.50 (43.10–68.50)	51.00 (39.00–62.00)	0.635
Serum chloride	96.65 (94.00–98.50)	97.50 (95.55–99.60)	97.40 (95.10–99.40)	0.227

Table 5 Spearman correlations between total AAC score and clinical and laboratory variables.

Variable	Spearman's rho	<i>p</i> value	<i>N</i>
Age	0.531	<0.001	163
Patient height	−0.042	0.598	163
Body mass index (BMI)	0.121	0.124	163
Duration of hemodialysis (years)	0.076	0.334	163
Serum magnesium	−0.123	0.118	163
Serum albumin	−0.215	0.006	162
Hemoglobin (HGB)	−0.012	0.875	162
Serum chloride	0.041	0.606	162
Serum iron	−0.051	0.520	162
White blood cell count	0.147	0.062	162
Fasting glucose	0.235	0.003	162
Transferrin saturation	−0.006	0.941	162
Ferritin	−0.003	0.968	162
Creatinine	−0.260	0.001	162
Serum calcium	0.062	0.436	162
Blood urea nitrogen (BUN)	−0.115	0.145	162

Table 6 Pearson correlations between total AAC score and selected laboratory variables.

Variable	Pearson's <i>r</i>	<i>p</i> value	<i>N</i>
Dry weight	0.033	0.671	163
Carbon dioxide	−0.051	0.517	162
Phosphate	0.022	0.782	162
Potassium	−0.199	0.011	162
Sodium	−0.100	0.204	162
Platelets (PLT)	0.163	0.038	162
Total iron binding capacity (TIBC)	0.000	0.996	102
Transferrin	−0.043	0.587	162

Table 7 Multivariable ordinal logistic regression analysis of factors associated with increasing abdominal aortic calcification severity.

Predictor	β (SE)	Adjusted OR (95% CI)	<i>p</i> value
Age, per year	0.076 (0.014)	1.079 (1.049–1.108)	<0.001
Hemodialysis duration, per year	0.057 (0.038)	1.059 (0.983–1.139)	0.134
Serum albumin, per 1 g/dL	−0.745 (0.460)	0.475 (0.193–1.170)	0.105
Serum creatinine, per 1 mg/dL	−0.001 (0.003)	0.999 (0.993–1.005)	0.729
Serum potassium, per 1 mmol/L	−0.376 (0.254)	0.687 (0.417–1.131)	0.139
Platelet count, per $10^3/\mu\text{L}$	0.003 (0.003)	1.003 (0.997–1.009)	0.295
Non-diabetic vs. diabetic	−0.592 (0.334)	0.553 (0.287–1.064)	0.076
Male vs. female	0.141 (0.376)	1.151 (0.552–2.406)	0.707
Non-smoker vs. smoker	−0.302 (0.378)	0.739 (0.352–1.551)	0.425

AAC, abdominal aortic calcification; OR, odds ratio; CI, confidence interval; SE, standard error. The dependent variable was ordered as no calcification, mild-to-moderate calcification, and severe calcification. Adjusted ORs greater than 1 indicate increased odds of being in a higher AAC severity category, whereas adjusted ORs below 1 indicate decreased odds. The model included age, sex, diabetes mellitus, smoking status, hemodialysis duration, serum albumin, serum creatinine, serum potassium, and platelet count. Reference categories were female, diabetic, and smoker. The proportional-odds assumption was satisfied (test of parallel lines: $\chi^2(9)=7.164$, $p=0.620$). The overall model was statistically significant (likelihood-ratio $\chi^2(9)=60.044$, $p<0.001$) and demonstrated acceptable goodness-of-fit according to the Pearson ($p=0.460$) and deviance ($p=0.832$) statistics. The Nagelkerke pseudo- R^2 was 0.350. The analysis included 162 complete cases.

goodness-of-fit statistics indicated acceptable model fit, and the Nagelkerke pseudo- R^2 was 0.350. The proportional-odds assumption was formally assessed and satisfied, as demonstrated by the non-significant test of parallel lines ($\chi^2(9)=7.164$, $p=0.620$) (Table 7).

After adjustment, age was the only statistically significant independent predictor of increasing AAC severity. Each additional year of age was associated with a 7.9% increase in the odds of belonging to a higher AAC severity category (adjusted OR 1.079, 95% CI 1.049–1.108; $p<0.001$). Non-diabetic participants had lower adjusted odds of greater AAC severity compared with diabetic participants, although the association did not reach statistical significance (adjusted OR 0.553, 95% CI 0.287–1.064; $p=0.076$). The inverse association between serum albumin and AAC severity observed in the unadjusted analyses was attenuated after adjustment and was no longer statistically significant (adjusted OR 0.475, 95% CI 0.193–1.170; $p=0.105$). Hemodialysis duration, serum creatinine, potassium, platelet count, sex, and smoking status were not independently associated with increasing AAC severity (all $p>0.05$) (Table 7).

In a sensitivity analysis additionally adjusting for serum calcium and phosphate, the expanded model remained statistically significant (likelihood-ratio $\chi^2(11)=65.784$, $p<0.001$) and demonstrated acceptable Pearson ($p=0.568$) and deviance ($p=0.870$) goodness-of-fit statistics, with a Nagelkerke pseudo- R^2 of 0.378. Age remained independently associated with increasing AAC severity (adjusted OR 1.082, 95% CI 1.051–1.114; $p<0.001$). Serum phosphate was not independently associated with AAC severity (adjusted OR 1.196, 95% CI 0.909–1.575; $p=0.200$), while serum calcium showed a borderline positive association (adjusted OR 1.636, 95% CI 0.996–2.688; $p=0.052$). Higher serum albumin (adjusted OR 0.345, 95% CI 0.133–0.893; $p=0.028$) and non-diabetic status (adjusted OR 0.506, 95% CI 0.259–0.987; $p=0.046$) were associated with lower AAC severity in the expanded model. However, these associations were considered model-sensitive because they

were not statistically significant in the primary model. Although the test of parallel lines was non-significant ($\chi^2(11)=5.558$; $p=0.901$), SPSS reported incomplete convergence of the general model; therefore, the sensitivity analysis was interpreted cautiously (Supplementary Table S1).

Discussion

In this cross-sectional study of 163 maintenance hemodialysis patients, abdominal aortic calcification was common and often advanced, with nearly two-thirds of participants showing some degree of calcification and approximately two in five meeting the threshold for severe AAC. Across all analytic approaches, age emerged as the central finding: it increased progressively across AAC categories, correlated moderately with total AAC score, and was the only variable that remained independently associated with greater AAC severity in both the primary multivariable ordinal model and the CKD-MBD sensitivity model. By contrast, the associations of diabetes and serum albumin were not stable across model specifications, and calcium-phosphate variables did not show definitive independent relationships in this dataset. These findings support the view that AAC in hemodialysis reflects the cumulative vascular consequences of aging superimposed on kidney failure-related risk, while also underscoring that several biologically plausible correlates become attenuated after adjustment for confounding and shared pathways^{16–18}.

The observed AAC burden is broadly consistent with international literature, although direct comparison requires attention to differences in age structure, dialysis vintage, imaging thresholds, and score categorization. In a Moroccan hemodialysis cohort assessed with the Kauppila method, AAC prevalence was 65%¹⁹. In the Thai multicenter CKD study, AAC was present in 70.8% of dialysis patients²⁰. In a relatively young Australian dialysis cohort awaiting transplantation, 49% had AAC score ≥ 1 , and among those with calcification a substantial subset had severe disease²¹. By contrast, a Pakistani single-center study reported a lower AAC prevalence of 26%, which the authors themselves interpreted in the context of a markedly younger population than many published hemodialysis cohorts²². A recent imaging study of abdominal vascular calcification in hemodialysis reported even higher prevalence estimates when broader vascular territories and volumetric methods were used, illustrating how modality and anatomical scope materially influence observed burden²³. Taken together, the present estimates place this Palestinian cohort within the higher-risk range reported internationally and suggest that substantial AAC burden is not confined to highly selected research settings.

The most important finding in this study is that age was the only predictor consistently significant across model specifications. This robustness matters more than crude contrasts alone. Age was associated with AAC in unadjusted analyses, correlated strongly with total calcification burden, and remained independently associated with ordinal increases in severity after adjustment for demographic, clinical, and laboratory covariates, with nearly identical effect estimates in the primary and sensitivity models. This pattern accords closely with the wider literature. Advanced age has repeatedly emerged as a key determinant of AAC or vascular calcification in CKD and dialysis populations, including hemodialysis cohorts from Morocco, Thailand, and India, as well as broader CKD cohorts²⁴. Mechanistically, this consistency is also biologically coherent. Vascular calcification is increasingly regarded as a manifestation of vascular aging, and CKD appears to accelerate these aging-associated processes through uremic toxicity, smooth muscle cell phenotypic transition, mineral dysregulation, inflammation, and loss of endogenous calcification inhibitors^{17,25,26}. From an epidemiologic perspective, age likely captures cumulative exposure to both traditional cardiovascular risk factors and CKD-specific insults over time, which may explain why it remained informative even when contemporaneous biochemical markers did not. This interpretation is also consistent with the notion that imaging markers such as AAC reflect the integrated result of prolonged exposure, whereas single serum measurements represent only a limited time point²⁴.

Diabetes and fasting glucose require a more nuanced interpretation. In the present study, fasting glucose differed across AAC categories and correlated positively with total AAC score, supporting a crude association between dysglycemia and greater calcification burden. Non-diabetic status was also associated with lower AAC

severity in the CKD-MBD sensitivity model, but diabetes was not independently significant in the primary multivariable model. Accordingly, diabetes should not be interpreted here as a robust independent predictor, but rather as a biologically plausible, model-sensitive association. This cautious interpretation is supported by the literature. In Thai dialysis patients, diabetes was identified as a predictive factor for higher AAC burden²⁰. In peritoneal dialysis, diabetes was more common in patients with high AAC in crude comparisons, although age remained the more stable multivariable factor in that cohort as well²⁷. More broadly, diabetes is repeatedly discussed as part of the adverse cardiovascular and calcific milieu in dialysis populations^{23,28}. However, because diabetes co-travels with age, inflammation, prior vascular disease, and medication exposure, its apparent effect on AAC severity may be partly mediated or confounded by those pathways. The current data therefore support plausibility, not independence.

Serum albumin and creatinine also appear to reflect underlying systemic status rather than direct causal determinants of calcification. Albumin differed across AAC categories and correlated inversely with total AAC score, and lower albumin became associated with greater AAC severity in the sensitivity model. Yet albumin was not significant in the primary model, so this association was clearly model-sensitive and should not be described as protective. In hemodialysis populations, albumin is an integrative marker shaped by nutritional intake, systemic inflammation, protein-energy wasting, infection, fluid distribution, hepatic synthesis, and dialysis-related losses, which limits causal interpretation^{28–30}. Indeed, prior dialysis studies show that the mortality association of albumin is partly explained by inflammation and not specifically by malnutrition alone³⁰. Large international HD data similarly link low albumin within a malnutrition-inflammation complex to adverse outcomes³¹. The inverse correlation between creatinine and AAC in the present study may likewise indicate lower muscle mass, frailty, or illness burden among patients with heavier calcification rather than a direct biochemical effect. A similar pattern of lower creatinine in patients with high AAC has been described in peritoneal dialysis cohorts²⁷. In this context, both albumin and creatinine are better viewed as markers of case-mix severity and protein-energy status than as direct mechanistic drivers.

Potassium and platelet count showed only limited and non-robust signals. Potassium differed across AAC categories and correlated inversely with total AAC score, whereas platelet count showed a weak positive correlation, but neither variable remained independently associated with AAC severity in adjusted models. These findings suggest that any relationship is likely indirect and vulnerable to confounding. Potassium in hemodialysis patients is influenced by diet, residual kidney function, intracellular shifts, acidosis, and dialytic removal, while platelet count may reflect inflammation, comorbidity, or hematologic variation rather than calcification biology itself. Because the current literature provided here does not identify either potassium or platelet count as established independent determinants of AAC, these findings should be interpreted as exploratory and hypothesis-generating rather than confirmatory.

Hemodialysis duration was not independently associated with AAC severity in the present primary model, despite its frequent discussion as a risk factor for vascular calcification. Some hemodialysis cohorts have reported longer dialysis vintage as an independent predictor of AAC or vascular calcification^{19,20,32}. Other datasets, however, have shown attenuation after multivariable adjustment or lack of clear association, particularly when age and other competing risk factors are considered^{21,27}. This inconsistency is understandable because dialysis duration is an imperfect proxy for cumulative uremic and treatment exposure. Important related factors were unavailable or incomplete in the present study, including dialysis adequacy metrics such as Kt/V, residual kidney function, vascular access type, dialysate calcium concentration, longitudinal exposure to calcium and phosphate abnormalities, inflammatory markers, and time-updated CKD-MBD therapy. Without these data, a null association for dialysis duration alone should not be taken to exclude dialysis-related contributions to calcification burden.

The CKD-MBD findings also warrant careful interpretation. Vascular calcification is a core component of CKD-MBD, and experimental and clinical literature consistently implicates positive calcium-phosphate balance, hyperphosphatemia, intermittent or persistent hypercalcemia, disordered PTH-vitamin D-FGF23-Klotho signaling, and loss of calcification inhibitors in its pathogenesis^{16,18}. In observational CKD and dialysis cohorts, phosphate and corrected calcium have sometimes been associated with AAC severity^{24,32}. In the present study,

however, serum phosphate was not independently associated with AAC severity in the sensitivity model, and calcium showed only a borderline positive association. These results should be interpreted cautiously for several reasons. First, the sensitivity model was supportive rather than definitive because SPSS issued a convergence warning regarding the general model, making the test of parallel lines uncertain. Second, single serum calcium and phosphate measurements may not capture cumulative mineral exposure, temporal variability, treatment response, or calcium load from binders and dialysate. Third, magnesium, PTH, alkaline phosphatase, vitamin D status, FGF23, Klotho, and calcification inhibitors were not incorporated into the model, although these factors are mechanistically relevant to CKD-MBD-related vascular calcification^{26,33}. The current data therefore do not support an independent phosphate signal in this cohort and suggest, at most, a borderline calcium association that requires confirmation in larger studies with longitudinal mineral profiling.

Crude medication associations should also be interpreted conservatively. Apparent relationships involving atorvastatin, aspirin, clopidogrel, apixaban, tamsulosin, and calcium acetate are more likely to reflect confounding by indication and underlying comorbidity than drug effects on AAC. Patients prescribed antiplatelet agents, anticoagulants, or statins typically have greater baseline cardiovascular disease burden, and those receiving calcium-containing binders may differ in mineral metabolism severity or treatment history. The broader CKD-MBD literature also cautions that calcium-containing phosphate binders can contribute to positive calcium balance and possibly calcification progression, whereas non-calcium binders are associated with less progression in some studies^{16,33}. Nevertheless, the present cross-sectional data were not designed to infer medication effects, and no causal interpretation should be drawn from these exploratory crude comparisons.

From a clinical perspective, the findings reinforce the potential utility of lateral lumbar radiography as an accessible tool for opportunistic cardiovascular risk stratification in dialysis care. Plain radiography has been proposed as an accessible method for AAC assessment in selected patients with CKD. Lateral lumbar radiography is widely available, less costly than computed tomography, and may provide clinically informative estimates of vascular calcification burden^{20,24}. In dialysis cohorts, Kauppila-based AAC measures have been associated with mortality and may improve risk prediction beyond traditional cardiovascular factors. Related studies in CKD and peritoneal dialysis populations likewise support the broader prognostic relevance of AAC^{24,27}. For resource-limited settings, this is especially relevant. However, the present study was cross-sectional and cannot determine whether screening changes management or outcomes; therefore, these findings support risk awareness and further study, not universal screening recommendations.

This study has several strengths. It used the validated 24-point Kauppila score, an accepted semiquantitative radiographic method for AAC assessment in CKD and dialysis research²¹. The ordinal categorization of no, mild-to-moderate, and severe AAC allowed severity-sensitive analysis rather than simple presence/absence classification. The primary multivariable model showed acceptable fit and satisfied the proportional-odds assumption, and the added CKD-MBD sensitivity analysis strengthened the interpretation by testing whether the central age association persisted after inclusion of calcium and phosphate. Importantly, it did.

The limitations are also substantial. The cross-sectional design precludes causal inference and cannot distinguish determinants of AAC initiation from progression. The study was conducted at a single tertiary center using convenience sampling, which introduces potential selection bias and limits external validity. The achieved sample size was slightly below target, with 163 of the planned 170 radiographs obtained, representing 95.9% of the calculated sample; this modest shortfall may have reduced power to detect weak associations or effects of uncommon exposures. Several potentially important covariates were unavailable or incomplete, including inflammatory markers, PTH and broader CKD-MBD biomarkers, magnesium in multivariable modeling, residual kidney function, Kt/V, vascular access type, dialysate composition, cumulative medication exposure, and prior cardiovascular disease burden. The specialist radiologist's score was used for analysis, and although a senior radiologist provided additional expert review, separate systematically retained independent scores were unavailable; therefore, inter-rater ICC and weighted kappa could not be calculated. Finally, the sensitivity model should be interpreted with caution because the parallel-lines test was accompanied by a convergence warning, making that assumption less certain than in the primary model.

In summary, AAC was highly prevalent in this Palestinian hemodialysis cohort, and severe calcification affected a substantial proportion of patients. Among all examined variables, age was the most robust determinant, remaining the only predictor consistently associated with greater AAC severity across both the primary and sensitivity models. Diabetes and albumin showed biologically plausible but model-sensitive associations, while phosphate was not independently associated and calcium showed only a borderline signal that should not be overinterpreted. These findings support the value of AAC as a clinically relevant vascular phenotype in dialysis populations and suggest that simple lateral lumbar radiography may be useful for opportunistic risk stratification in appropriate settings. Prospective multicenter studies with larger samples, standardized imaging reproducibility, time-updated mineral and inflammatory markers, and longitudinal outcome assessment are needed to validate these findings and clarify whether modifiable pathways beyond age meaningfully influence AAC burden and progression.

Conclusion

Abdominal aortic calcification was common among Palestinian patients receiving maintenance hemodialysis, with severe calcification affecting approximately two in five participants. Age was the only predictor consistently associated with increasing AAC severity across both the primary and CKD-MBD-adjusted models. Diabetes and serum albumin demonstrated biologically plausible but model-sensitive associations, while serum phosphate was not independently associated and serum calcium showed only a borderline signal in the sensitivity analysis. Lateral lumbar radiography may provide an accessible adjunct to cardiovascular-risk assessment in hemodialysis care; however, prospective multicenter studies with standardized imaging reliability, longitudinal CKD-MBD measurements, and clinical outcome assessment are needed before routine screening or intervention pathways can be recommended.

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Author contributions M.G., A.K., and B.B. contributed to study conception, questionnaire development, data collection, data curation, statistical analysis, and manuscript drafting. A.S. contributed to study design, supervision, data interpretation, manuscript drafting, and critical revision of the manuscript. Q.J.A.M. and M.H. contributed to critical revision of the manuscript. All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

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Data availability The datasets generated and/or analyzed during the current study are not publicly available but are available from the corresponding author on reasonable request, subject to institutional and ethical approval requirements.

Declarations

Competing interests The authors declare no competing interests.

Ethics approval and consent to participate The study protocol was approved by the Institutional Review Board of An-Najah National University, Nablus, Palestine (IRB Med. Oct. 2024/30), and was also reviewed by the relevant hospital and departmental authorities. All participants were informed about the purpose of the study and the content of the interview prior to enrollment, and written informed consent was obtained from each participant before data collection. Participation was entirely voluntary. All collected data were kept confidential and used solely for research purposes. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

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Authors and Affiliations

Maisam Gharayba¹ · Ahmad Katbeh¹ · Basem Bali¹ · Ali Shakhshir^{2,3}  · Qutaiba Ja'far A. Mahmoud⁴ · Mohanad Hassan⁵

✉ Basem Bali
basembali12@gmail.com

✉ Ali Shakhshir
shakhshirali@gmail.com; ali.shakhshir@aaup.edu

¹ Department of Medicine, Faculty of Medicine and Health Sciences, An-Najah National University, Nablus, Palestine

² Scientific Research Committee, Department of Medicine, Faculty of Medicine, Arab American University, Jenin, Palestine

³ Scientific Research Committee, Faculty of Medicine, Arab American University, Jenin, Palestine

⁴ Department of Radiology, An-Najah National University Hospital, Nablus, Palestine

⁵ Department of Nephrology, An-Najah National University Hospital, Nablus, Palestine