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ORIGINAL ARTICLE

Severe aortic arch calcification predicts mortality in patients undergoing peritoneal dialysis

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Background/Purpose: Vascular calcification can predict cardiovascular (CV) morbidity and mortality in patients with end-stage renal disease. We evaluated the prevalence, association factors, and outcomes of chest X-ray-detected aortic arch calcification (AoAC) in patients undergoing peritoneal dialysis (PD).
Methods: We included 190 patients undergoing PD (mean age, 52.6 ± 14.3 years) for whom chest radiographs were available. AoAC revealed by chest X-ray was graded from 0 to 3 according to an AoAC score (AoACS). Multiple regression analyses were used to determine the factors associated with AoACS. After adjusting for age, sex, PD duration, diabetes mellitus, mean

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blood pressure, and history of CV disease, the association between AoAC grading and mortality were assessed using the Kaplan–Meier curve and Cox proportional hazard model.
Results: Age ($p < 0.001$), PD duration ($p = 0.004$), history of CV disease ($p < 0.001$), and renal Kt/V ($p = 0.031$) were associated with AoACS. After a mean follow-up of 55.1 ± 32.1 months, patients with Grade 2 ($p = 0.011$) or Grade 3 ($p < 0.001$) AoAC had higher all-cause mortality than patients with Grade 0 AoAC. In addition, patients with Grades 2 and 3 AoAC had higher CV-related mortality than those with Grades 0 and 1 AoAC ($p = 0.013$). Grade 2 [hazard ratio (HR) = 2.736; 95% confidence interval (CI), 1.038–7.211; $p = 0.042$] and Grade 3 AoAC (HR = 3.289; 95% CI, 1.156–9.359; $p = 0.026$) remained associated with all-cause mortality after adjustment. Similarly, Grades 2 and 3 AoAC (HR = 36.05; 95% CI, 3.494–372; $p = 0.026$) significantly correlated with CV mortality after adjustment.
Conclusion: In patients undergoing PD, CXR-detected severe AoAC was an independent risk factor for all-cause and CV mortalities.

Introduction

Cardiovascular (CV) events have been a major cause of death in patients with end-stage renal disease (ESRD)[1,2]; the incidence is 10 to 30 times higher than that of the general population.[3] In addition to the traditional risk factors of CV events, uremia-specific phenomena including vascular calcification are also responsible for the higher incidence of CV events in patients with ESRD.[4] Vascular calcification, which may involve arterial media, atherosclerotic plaques, or cardiac valves,[4] is highly prevalent in patients with ESRD including younger age groups.[5,6] The existence and extent of vascular calcification are strong predictors of CV morbidity and mortality in both patients undergoing hemodialysis (HD) and those undergoing peritoneal dialysis (PD).[7–13]

In order to evaluate or quantify vascular calcification, several imaging techniques such as electron beam computed tomography (EBCT), multislice spiral CT (MSCT), echocardiography, vascular ultrasound, and plain radiography have been used. Among them, EBCT and MSCT are still considered the gold standards for quantifying coronary or aortic calcification.[14] However, these methods are expensive and expose the patients to high doses of radiation and risks of cancer.[15,16] Chest radiography (CXR), comparatively, is an inexpensive test with lower radiation exposure. Currently, it is a simple and useful tool to evaluate aortic arch calcification (AoAC); the presence or progression of AoAC has been shown to correlate with all-cause and CV mortality in patients with ESRD,[17] patients undergoing HD,[18–20] and even in patients undergoing PD.[13]

The aim of this study was to evaluate the prevalence and associated risk factors of CXR-detected AoAC in patients undergoing PD because its clinical significance has not yet been fully investigated. The prognostic value of CXR-detected AoAC in long-term CV morbidity as well as all-cause and CV mortalities was also analyzed.

Methods

Patients

In October 2006, 192 patients underwent PD for more than 3 months at the National Taiwan University Hospital. Among them, two patients were excluded from the study because chest radiographs were unavailable or death occurred in November 2006. Finally, 190 patients were enrolled and were followed-up for 8 years. More than 75% patients received continuous ambulatory PD, and all patients used lactate-buffered dialysate. The study was approved by the Institution Review Board of the hospital (number 200912054R) and was in adherence with the Declaration of Helsinki.

AoAC revealed by CXR

The chest radiographs of patients between June and August 2006 were evaluated. AoAC was assessed by a specific scale developed in a previous study.[21] Briefly, a scale, divided into 16 circumferences, was applied over AoAC revealed by CXR as shown in Figure 1. The number of sectors occupied by AoAC, ranging from 0 to 16, were recorded as AoAC score (AoACS). Two radiologists, both specializing in CXR, independently reviewed these images. The images of 80 patients were reassessed by these two radiologists because of interreader variability. Among these 80 images, 19 images were initially scored 0 by only one radiologist. Among the remaining 61 images, 46 images had initial scores varying ≤2.

AoAC extent was divided into four grades: Grade 0, AoACS = 0; Grade 1, AoACS = 1–4; Grade 2, AoACS = 5–8; and Grade 3, AoACS = 9–16.

Demographic and clinical data collection

All the data were collected in October 2006. Patients' age, sex, body height (BH), body weight (BW), systolic blood pressure, diastolic blood pressure (DBP), diabetes mellitus (DM), peritoneal equilibrium test, and dialysis adequacy indices, including peritoneal urea clearance (Kt/V), renal Kt/V, total Kt/V (the sum of peritoneal and renal Kt/Vs), and standardized total weekly creatinine clearance, were recorded. Duration of PD was defined as the interval between the initiation of PD and the time when CXR was performed. CV disease (CVD) was defined as a history of coronary artery disease, cerebrovascular accident, or peripheral arterial occlusive disease. Coronary artery disease

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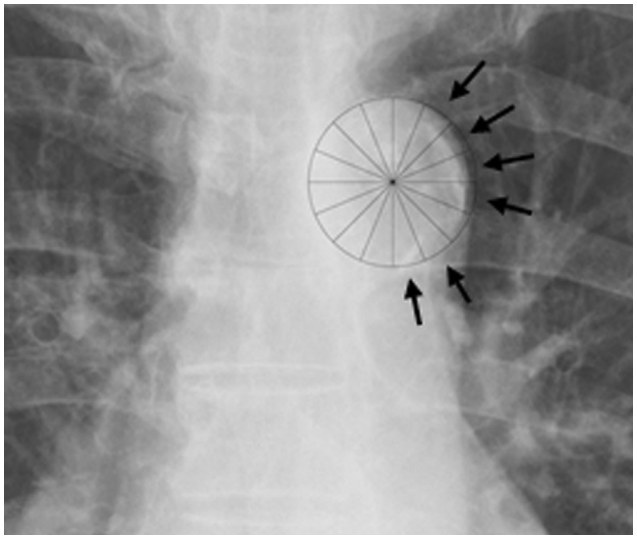

Figure 1 Measurement of the aortic arch calcification score (AoACS) using plain chest radiography. A scale, divided into 16 circumferences, was attached to AoAC on the chest radiograph. In this example, the sectors occupied with AoAC are marked by arrows. AoACS was recorded as 6.

was defined as a history of acute coronary syndrome, coronary arterial bypass grafts, or confirmation by coronary angiography. Cerebrovascular accident was defined as a history of transient ischemic attack or stroke, and peripheral arterial occlusive disease was defined as a history of claudication, ischemic limb loss, ulceration, or peripheral revascularization. Laboratory data included levels of blood hemoglobin, serum albumin, alkaline phosphatase, calcium, phosphorus, total cholesterol, triglyceride, high-density lipoprotein-cholesterol (HDL), low-density lipoprotein-cholesterol, intact parathyroid hormone, and ferritin. Body mass index (BW/BH^2), mean blood pressure (MBP), and calcium–phosphorus product (CPP) were calculated for subsequent analyses.

Follow-up for endpoints

All enrolled patients were regularly followed up at the PD clinic until September 2014. Time and causes of mortality of patients were determined carefully by both chart review and interviews with primary care nurses. Mortality due to CV events was considered as death from myocardial infarction, congestive heart failure, sudden cardiac death, cardiac arrhythmia, or stroke. Nonfatal CV events requiring hospitalization included acute coronary syndrome with or without evidence of myocardial infarction, arrhythmia, stroke or transient ischemic attack, and critical limb ischemia.

Statistical analysis

The data were analyzed using the SAS software, Version 9.1 (SAS Institute Inc., Cary, NC, USA). A p value of <0.05 was considered statistically significant. Continuous variables were expressed as mean $\pm$ standard deviation, and categorical variables were expressed as number, percentage. Trend test was used to compare data between groups. Multivariate regression analysis with stepwise variable selection was used to determine the associated factors of AoAC. Variables with $p < 0.05$ in the trend test were selected. Then the stepwise procedure was applied to select variables with both significance level for entry and significance level for stay set to 0.15.

The Kaplan–Meier survival curves were drawn, and between-group survival was compared using the log-rank test. Factors that predicted all-cause and CV mortalities were determined with the Cox proportional hazard model. Age, sex, duration of PD, DM status, history of CVD, and MBP were included as variables first, and then stepwise procedure was applied to select remaining variables with both significance level for entry and significance level for stay set to 0.15.

Results

Comparison of baseline clinical data between patients with different grades of AoAC

The baseline characteristics of these 190 patients are shown in Table 1. The mean age was 52.6 $\pm$ 14.3 years, and 85 patients (44.7%) were men. They had received PD for 39 $\pm$ 38 months on average. Only 29 patients (15.3%) had DM, and 21 patients (11.1%) developed ESRD due to DM nephropathy. Moreover, 40 patients (21.1%) had a history of CVD.

According to the severity of AoAC, these patients were divided into four groups: Grade 0 (80, 42.1%), Grade 1 (47, 24.7%), Grade 2 (36, 18.9%), and Grade 3 (27, 14.2%). As shown in Table 1, patients with higher grades of AoAC were older ($p < 0.001$), received longer duration of PD ($p = 0.01$), had lower MBP ($p < 0.001$), DBP ($p < 0.001$), serum P ($p = 0.029$), HDL ($p = 0.029$), renal Kt/V ($p = 0.037$), and CPP ($p = 0.039$), as well as higher serum ferritin ($p = 0.01$). In addition, higher prevalence rates of DM ($p = 0.008$) and CVD ($p < 0.001$) were found in patients with higher grades of AoAC.

Factors associated with AoAC severity

In order to determine the associated factors of AoAC severity in these patients, multiple regression analysis was performed. As shown in Table 2, age ($p < 0.001$), duration of PD ($p = 0.004$) and history of CVD ($p < 0.001$) were independent factors positively associated with AoACS, whereas renal Kt/V ($p = 0.031$) was another independent factor negatively associated with AoACS.

Because abdominal aortic calcification has been shown to be positively correlated with left ventricular diastolic dysfunction,[11] we evaluated if there was any correlation between AoAC and cardiac dysfunction. Only 81 enrolled patients had echocardiographic reports available between 2005 and 2007. There was no association between grading of AoAC and abnormal echocardiographic findings such as left atrial dilation, left ventricular dilation, left ventricular hypertrophy, poor/impaired left ventricular contractility, and probable left ventricular diastolic dysfunction (Table S1).

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Table 1 Comparisons of baseline characteristics and clinical data among patients who underwent peritoneal dialysis with different grades of aortic arch calcification using the trend test.

Variable	All patients ($N = 190$)	Grading of Aortic Arch Calcification				p Value of trend test
		0 ($N = 80$)	1 ($N = 47$)	2 ($N = 36$)	3 ($N = 27$)	
Age (y)	52.6 ± 14.3	45.4 ± 13.3	51.9 ± 10.8	57.7 ± 10.1	68.7 ± 11.6	<0.001
Male (no. %)	85, 44.7	35, 43.8	19, 40.4	21, 58.3	10, 37.0	0.293
DM (no. %)	29, 15.3	10, 12.5	6, 12.8	3, 8.3	10, 37.0	0.008
Duration of PD (mo)	39.0 ± 38	30.1 ± 26	39.1 ± 42	54.9 ± 50.4	43.8 ± 35.6	0.010
BMI (kg/m^2)	22.7 ± 3.5	22.9 ± 3.9	22.5 ± 3.3	22.8 ± 3	22.5 ± 3.2	0.914
MBP (mmHg)	98.0 ± 10.5	100.8 ± 10.7	98.4 ± 9.6	96.3 ± 8.9	91.3 ± 10.8	<0.001
SBP (mmHg)	138.4 ± 16.3	139.4 ± 16.1	139.5 ± 15.6	135.5 ± 16.1	136.9 ± 18.9	0.609
DBP (mmHg)	84.5 ± 9.8	87.8 ± 9.8	84.8 ± 8.7	82.7 ± 7.3	76.2 ± 9.3	<0.001
Hb (g/dL)	9.8 ± 1.5	9.7 ± 1.5	9.9 ± 1.4	10.1 ± 1.8	9.6 ± 1.3	0.446
Albumin (g/dL)	4.0 ± 0.4	4.1 ± 0.5	4.1 ± 0.4	4.0 ± 0.3	3.9 ± 0.3	0.189
iPTH (μg/dL)	377.7 ± 414.8	372.5 ± 449	391.6 ± 350.7	427.4 ± 463.6	302.5 ± 348.8	0.693
ALP (U/L)	200.9 ± 115.1	212.8 ± 122	174.2 ± 94	194.8 ± 102.1	220.6 ± 138.8	0.234
Calcium (mg/dL)	9.4 ± 0.9	9.2 ± 1	9.4 ± 0.9	9.5 ± 0.8	9.5 ± 0.6	0.274
P (mg/dL)	5.1 ± 1.3	5.0 ± 1.2	5.5 ± 1.4	5.1 ± 1.0	4.7 ± 1.3	0.029
CPP (mg^2/dL^2)	48.0 ± 13.3	46.1 ± 12.7	52.4 ± 14.7	48.7 ± 11.2	44.9 ± 13.3	0.039
Ferritin (ng/mL)	534.7 ± 595.8	494.9 ± 446.8	489.5 ± 515.5	422 ± 404.5	881.9 ± 1063.8	0.010
TCHO (mg/dL)	201.1 ± 47.1	195.5 ± 39.8	212.4 ± 48.1	205.1 ± 51.1	192.4 ± 57.3	0.169
LDL (mg/dL)	96.2 ± 34.2	97.3 ± 30.2	92.1 ± 32.4	98.1 ± 39.4	97.7 ± 42.0	0.823
HDL (mg/dL)	42.4 ± 10.5	43.4 ± 11.5	44.9 ± 10.3	39.9 ± 9.2	38.6 ± 7.2	0.029
TG (mg/dL)	192.2 ± 148.3	162.6 ± 113.8	217.7 ± 202.3	222.3 ± 143.8	195.6 ± 123.4	0.108
H/HA PET (no. %)	64, 33.7	25, 31.3	15, 31.9	14, 38.9	10, 37.0	0.837
PKTV	2.0 ± 0.4	2.0 ± 0.4	1.9 ± 0.4	2.0 ± 0.4	2.1 ± 0.3	0.155
RKTV	0.3 ± 0.4	0.3 ± 0.4	0.3 ± 0.4	0.2 ± 0.3	0.1 ± 0.2	0.037
KTV	2.2 ± 0.3	2.3 ± 0.3	2.2 ± 0.3	2.2 ± 0.3	2.2 ± 0.3	0.164
WCC	60.8 ± 15.1	61.9 ± 16.4	61.9 ± 16.6	61.8 ± 11.8	54.5 ± 11.1	0.135
CAD (no. %)	29, 15.3	7, 8.8	4, 8.5	7, 19.4	11, 40.7	<0.001
PAOD (no. %)	8, 4.2	0, 0	0, 0	4, 11.1	4, 14.8	<0.001
CVA (no. %)	12, 6.3	1, 1.3	1, 2.1	2, 5.6	8, 29.6	<0.001
CVD (number, %)	40, 21.1	8, 10.0	5, 10.6	10, 27.8	17, 63.0	<0.001

ALP = alkaline phosphatase; BMI = body mass index; CAD = coronary artery disease; CPP = calcium–phosphorus product; CVA = cerebrovascular accident; CVD = cardiovascular diseases including CAD, PAOD, and CVA; DBP = diastolic blood pressure; DM = diabetes mellitus; HA/H PET = high average/high peritoneal equilibrium test; Hb = hemoglobin; HDL = high-density lipoprotein cholesterol; iPTH = intact parathyroid hormone; KTV = Kt/V; LDL = low-density lipoprotein cholesterol; MBP = mean blood pressure; P = phosphorus; PAOD = peripheral arterial occlusive disease; PD = peritoneal dialysis; PKTV = peritoneal Kt/V; RKTV = renal Kt/V; SBP = systolic blood pressure; TCHO = total cholesterol; TG = triglyceride; WCC = weekly creatinine clearance.

Association between AoAC and nonfatal CV events

As vascular calcification has been shown to correlate with CV morbidities,[11,12] we wanted to know if there was any association between CXR-detected AoAC and CVD. During 8 years of follow-up, 10 nonfatal CV events requiring hospitalization were noted. As shown in Figure 2A, patients with Grade 3 AoAC had lower CV event-free survival rate than those with Grade 0 AoAC (log-rank test, $p = 0.002$). However, AoAC grading could not predict subsequent CV events that required hospitalization in further multivariate Cox regression analyses (data not shown).

AoAC grading as an independent risk factor for all-cause and CV mortalities

During a mean follow-up period of 55.1 ± 32.1 months, 47 patients (24.7%) died. Among them, 10 patients (21.3%)

Table 2 Multiple regression analysis of association factors for AoACS by stepwise variable selection method ($N = 190$, $R^2 = 0.449$).

	Parameter estimate	Standard error	p
Intercept	0.727	3.220	0.822
Age	0.119	0.022	<0.001
CVD	2.873	0.634	<0.001
Duration of PD	0.020	0.007	0.004
DBP	−0.053	0.029	0.071
RKTV	−1.694	0.780	0.031

AoACS = aortic arch calcification score; CVD = cardiovascular diseases including coronary artery disease, peripheral arterial occlusive disease, and cerebrovascular accident; DBP = diastolic blood pressure; PD = peritoneal dialysis; RKTV = renal Kt/V.

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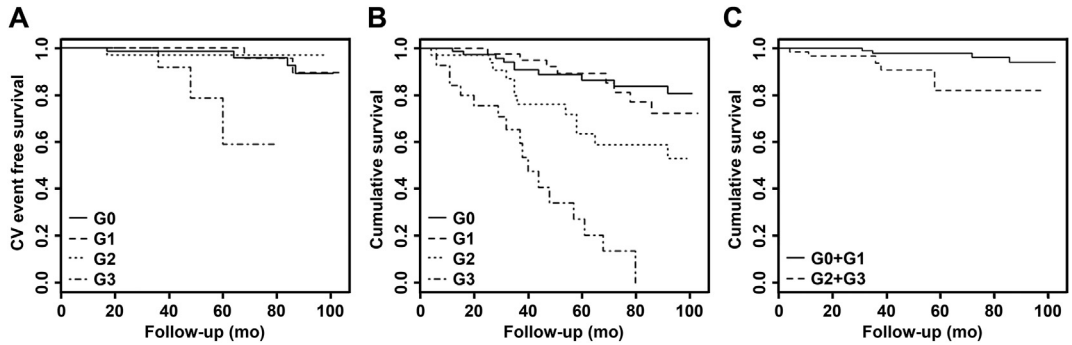

Figure 2 Kaplan–Meier analyses of (A) nonfatal cardiovascular (CV) events requiring hospitalization, (B) all-cause mortality, and (C) CV mortality in 190 patients. (A) Patients with Grade 3 aortic arch calcification (AoAC) had lower CV event-free survival rate than those with Grade 0 AoAC (log-rank test, $p = 0.002$). (B) All-cause mortality was higher in patients with Grade 2 (log-rank test, $p = 0.011$) or Grade 3 (log-rank test, $p < 0.001$) AoAC than those with Grade 0 AoAC. (C) CV mortality was higher in patients with severe AoAC (Grades 2 and 3 AoAC) than those with mild AoAC (Grade 0 and 1 AoAC) (log-rank test, $p = 0.013$). G: grade.

died of CV events. All-cause and CV mortalities-free survival rates were 62.9% and 90.5%, respectively. Compared with patients with Grade 0 AoAC, patients with Grade 2 (log-rank test, $p = 0.011$) or Grade 3 (log-rank test, $p < 0.001$) AoAC had higher all-cause mortality rate. In addition, patients with Grades 2 and 3 AoAC had higher CV mortality than those with Grades 0 and 1 AoAC (log-rank test, $p = 0.013$) (Figures 2B and 2C).

In order to determine if AoAC grading independently predicted all-cause and CV mortalities, the multivariate Cox proportional hazard model was used. Grade 2 AoAC [hazard ratio (HR) = 2.736; 95% confidence interval (CI), 1.038–7.211; $p = 0.042$], Grade 3 AoAC (HR = 3.289; 95% CI, 1.156–9.359; $p = 0.026$), and serum albumin levels (HR = 0.276; 95% CI, 0.114–0.673; $p = 0.005$) correlated independently with all-cause mortality after adjustment by age, sex, DM status, MBP, and history of CVD (Table 3). Similarly, severe AoAC including Grades 2 and 3 AoAC (HR = 36.05; 95% CI, 3.494–372.0; $p = 0.003$), body mass index (HR = 13.56; 95% CI, 1.438–127.8; $p = 0.0023$), and serum albumin levels (HR = 0.071; 95% CI, 0.009–0.533; $p = 0.01$) were independently associated with CV mortality after adjustment by age, sex, DM status, MBP, and history of CVD. Furthermore, CV mortality was negatively predicted by age (HR = 0.931; 95% CI, 0.875–0.990; $p = 0.024$), but was not predicted by history of CVD (Table 3).

Discussion

This prospective cohort study was designed to identify the clinical significance of CXR-detected AoAC in patients undergoing PD. We demonstrated that age, PD duration, previous CVD, and renal Kt/V were independently associated with AoAC. Other significant factors associated univariately with AoAC were DM, DBP, serum P, ferritin, and HDL. Moreover, severe AoAC predicted all-cause and CV mortalities in patients who underwent PD after adjustment by age, sex, PD duration, DM, and CVD history.

Our study has shown that residual renal function (RRF) decline correlates with vascular calcification in patients who underwent PD, which is in agreement with previous studies.[22,23] This correlation is probably because of the increased serum indoxyl sulfate levels as patients undergoing PD lose their RRF gradually.[24] Given that indoxyl sulfate can cause aortic calcification,[25] patients undergoing PD with decreased RRF have higher levels of indoxyl sulfate, resulting in progression of aortic calcification. Therefore, interventions to lower indoxyl sulfate in patients undergoing PD might delay the progression of aortic calcification.

Arterial stiffness is common in patients with chronic kidney disease. Arterial stiffness involving the aorta leads to an increase in systolic blood pressure and a decrease in DBP; hence, it increases pulse pressure. Its severity can also be evaluated by pulse wave velocity.[26] In patients undergoing HD, both pulse pressure and pulse wave velocity have been positively associated with AoAC.[21,27] Our study also demonstrates that in patients undergoing PD, DBP was negatively associated with AoAC; this indicates that AoAC in the dialysis population was strongly correlated with aortic stiffness. This is probably because AoAC in the setting of renal dysfunction is mainly composed of calcified medial vasculopathy, which is known to induce arterial stiffness.[28] Therefore, AoAC detected by CXR can be considered a marker of aortic stiffness in patients undergoing dialysis.

Inflammation, commonly evaluated by the serum C-reactive protein levels, has been considered an important risk factor of CV calcification in patients undergoing PD.[13,29–31] In our study, as serum C-reactive protein levels were not available, serum ferritin levels were evaluated instead to represent inflammation status. We showed that the serum ferritin levels had a significant association with CXR-detected AoAC. Furthermore, lower serum P and CPP levels were also correlated with higher grades of AoAC. Because our study population was characterized by well-controlled CPP levels (<55 mg^2/dL^2), lower serum P levels probably indicated a status of malnutrition, which is linked to inflammation.

Although AoAC detected by CXR has been shown to predict all-cause and CV mortalities in patients undergoing HD,[18–20] the significance of CXR-detected AoAC in patients undergoing PD has just been elucidated. To our knowledge, only one study showed that CXR-detected AoAC progression could predict all-cause and CV mortalities in patients undergoing PD. However, its mean follow-up time was less

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Table 3 Factors predicting all-cause and cardiovascular mortalities in patients undergoing peritoneal dialysis using the Cox model.

Variable	Parameter estimate	Standard error	Hazard ratio	95% CI	*p*
All-cause mortality					
Age	0.019	0.018	1.019	0.984–1.055	0.296
Male	−0.369	0.324	0.692	0.366–1.305	0.255
Duration of PD	0.001	0.003	1.001	0.995–1.007	0.721
DM	1.074	0.406	2.926	1.321–6.480	0.008
CVD	0.662	0.381	1.939	0.919–4.091	0.082
MBP	−0.025	0.018	0.975	0.942–1.010	0.157
Grade 1 versus 0	0.059	0.494	1.061	0.403–2.796	0.904
Grade 2 versus 0	1.027	0.503	2.792	1.041–7.484	0.041
Grade 3 versus 0	1.254	0.547	3.506	1.200–10.24	0.022
Albumin	−1.387	0.478	0.250	0.098–0.638	0.004
Ferritin	0.000360	0.000220	1.000	1.000–1.001	0.102
Cardiovascular mortality					
Age	−0.148	0.059	0.862	0.768–0.968	0.012
Male	−0.910	1.028	0.402	0.054–3.018	0.376
Duration of PD	−0.030	0.017	0.971	0.940–1.003	0.074
DM	3.695	1.159	40.26	4.156–390.1	0.001
CVD	0.997	0.919	2.711	0.447–16.43	0.278
MBP	−0.071	0.050	0.931	0.844–1.027	0.155
Grade 2 + 3 versus 0 + 1	4.621	1.520	101.6	5.160–2000	0.002
BMI	2.894	1.228	18.06	1.627–200.5	0.019
Albumin	−3.493	1.148	0.030	0.003–0.288	0.002
Phosphorus	0.629	0.345	1.876	0.954–3.691	0.068
HDL	−0.070	0.045	0.932	0.854–1.018	0.120

BMI = body mass index; CI = confidence interval; CVD = cardiovascular diseases including coronary artery disease, peripheral arterial occlusive disease, and cerebrovascular accident; DM = diabetes mellitus; HDL = high-density lipoprotein cholesterol; MBP = mean blood pressure; PD = peritoneal dialysis.

than 3 years (34.2 ± 20.4 months).[13] Our study confirms that severe CXR-detected AoAC predicted all-cause and CV mortalities in patients undergoing PD after adjustment by age, sex, PD duration, DM, and CVD history. Moreover, the mean follow-up period of our study was much longer (55.1 ± 32.1 months). Therefore, CXR-detected AoAC has a significant prognostic value in predicting all-cause and CV mortalities in patients undergoing PD.

History of CVD has been regarded as a risk factor for CV mortality in patients undergoing PD.[11,32] However, history of CVD could not predict CV mortality in our population. Because our population was characterized by lower prevalence of DM (15.3% vs. 34–47%),[11,32] previous CVD (21.1% vs. 22.4–35%),[11,32] and CV mortality (21.3% of total death vs. 43.3–50% of total death),[11,32] most of the population still have medial vasculopathy, which might not be clinically diagnosed as CVD but could be clearly represented by AoAC. Therefore, CXR-detected AoAC, not history of CVD, dominated in predicting CV mortality in our population. It indicates that AoAC could be a good marker in representing medial vasculopathy especially in non-DM patients undergoing PD.

To conclude, our study shows that in addition to age and PD duration, RRF and CV history were strongly associated with CXR-detected AoAC in patients undergoing maintenance PD. We also show with evidence that arterial stiffness (represented by DBP) and inflammation (represented by serum ferritin levels) had correlations with CXR-detected AoAC. Furthermore, we demonstrated that severe AoAC predicted all-cause and CV mortalities in patients undergoing PD independent of age, sex, PD duration, DM, and CVD history. Finally, we show that CXR could be considered a valuable tool to evaluate AoAC in patients undergoing PD.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.jfma.2016.06.006.

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