CORONARY ARTERY CALCIFICATION SCORE AS A PREDICTOR OF ALL-CAUSE MORTALITY AND CARDIOVASCULAR OUTCOME IN PERITONEAL DIALYSIS PATIENTS

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◆ **Background:** This study aimed to examine whether the coronary artery calcification score (CaCS) was associated with the prognosis of peritoneal dialysis (PD) patients.

◆ **Methods:** Adult PD patients who were clinically stable for at least 2 months were recruited for this prospective, observational cohort study. Coronary artery calcification was assessed using multislice spiral computed tomography and was recorded according to the Agatston score. The endpoints including all-cause mortality, cardiovascular events, and cardiovascular mortality were assessed. Multivariate Cox regression was used to identify independent predictors of all-cause mortality, cardiovascular events (CVEs), and cardiovascular mortality.

◆ **Results:** A total of 179 PD patients (86 men) with a mean age of 63.5 ± 14.8 years were recruited for this study. Coronary artery calcification scores ranging from 0 to 5,257 were stratified as follows: no (CaCS = 0, $n = 54$), low ($0 < \text{CaCS} < 400$, $n = 72$), and high ($\text{CaCS} \geq 400$, $n = 53$) calcification. The follow-up duration was 30.6 ± 16.2 (24–63) months. Compared with the no calcification group, patients with a higher CaCS were older and had lower diastolic blood pressure, residual renal function, and serum albumin, and higher HbA_{1c} and serum insulin. Multivariate Cox regression revealed that the CaCS was an independent predictor for all the 3 endpoints after adjustment in PD patients.

◆ **Conclusions:** CaCS was an independent predictor of all-cause mortality, cardiovascular events, and cardiovascular mortality in patients receiving peritoneal dialysis.

Cardiovascular events (CVEs) are among the leading causes of mortality in patients who are receiving peritoneal dialysis (PD), and identifying the CVE risk factors is critical to the care of PD patients (1–3). Traditional CVE risk factors in the general population include hypertension, diabetes mellitus, and hyperlipidemia. Except for these traditional factors, other factors may also play important roles on the prognosis of PD patients (4–6). Recently, vascular calcification has been reported to be an independent predictor of all-cause mortality and CVEs in the general population and those with end-stage renal disease (ESRD) (7–11). However, whether vascular calcification is associated with the prognosis of PD patients remains uncertain.

Among the evaluation methods of vascular calcification, the coronary artery calcification score (CaCS), which is quantified by multislice spiral computed tomography (MSCT), has emerged as a prognostic indicator of CVEs (12–14). In the general population without a calcium-phosphorus metabolism disorder, the CaCS can be a marker of atherosclerotic plaque burden, but in ESRD patients, the score might be more relevant for calcium phosphorus metabolism disorders than for atherosclerotic plaque (15). Hence, the prognostic value of the CaCS for PD patients might be different from its value in the general population. Thus, the present study aimed to examine whether the CaCS was associated with all-cause mortality, cardiovascular events, and cardiovascular mortality of PD patients.

MATERIALS AND METHODS

SUBJECTS

Patients from the PD center at Huashan Hospital Fudan University in China were recruited for a prospective, observational cohort study from May 2006 to May 2010. All of the patients had received regular PD for more than

Perit Dial Int 2016; 36(2):163–170 epub ahead of print: 29 Jul 2015
<http://dx.doi.org/10.3747/pdi.2014.00124>

KEY WORDS: Coronary artery calcification; all-cause mortality; cardiovascular events; cardiovascular mortality; risk factor.

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Received 28 July 2014; accepted 31 July 2014.

Supplemental material available at www.pdconnect.com

2 months and were followed up at our center. Patients were excluded if they had an acute infection or unstable cardiovascular disease or were clinically unstable with a life expectancy of less than 3 months. They could be recruited for the study if the active disease was stable for more than 2 months. The study was approved by the ethics committee of Huashan Hospital Fudan University. All of the patients provided written informed consent.

CORONARY ARTERY CALCIFICATION SCORE

Coronary artery calcification was assessed using MSCT and was recorded according to the Agatston score (CaCS). In this method, the heart was scanned over a period of 20 to 30 seconds, with a distance of 3 mm between each slice. The software built into this equipment could detect calcified lesions with a density of at least 130 Hounsfield units and a minimal area of 0.5 mm². The acquisition time for this method was 125 milliseconds. For imaging reconstruction, this method used data obtained during the diastolic phase of the cardiac cycle. The total radiation dose during the entire procedure was 1.25 milli-Sieverts (mSV). The total CaCS calculations were based on formulas using measurements of total volume, area and mean, and maximum densities of the calcified lesions. Individual CaCSs were calculated for the left main coronary artery, descending branch of the left coronary artery, circumflex branch of the left coronary artery, and right coronary artery. Then, the scores were summed to calculate the total CaCS. The final score was expressed in modified Agatston units.

OUTCOME MEASUREMENTS

The primary endpoint was all-cause mortality. The secondary endpoints included CVEs and cardiovascular death. Cardiovascular events were defined as follows (16):

1. Coronary artery events were diagnosed by typical symptoms attributable to angina pectoris, confirmed by the presence of ST-T dynamic changes on electrocardiography or by a 3-fold increase over baseline in myocardium markers, regardless of the use of percutaneous coronary intervention or coronary artery bypass grafting;
2. Cerebrovascular disease was diagnosed based on clinical signs and symptoms that were confirmed by positive findings of infarction or bleeding on computed tomography or magnetic resonance imaging;
3. Peripheral artery disease was diagnosed in the presence of intermittent claudication and/or leg pain at rest and was confirmed by significant arterial stenosis on angiography.

Cardiovascular death was defined as mortality caused by the CVEs specified in the above section. Patients who left the study or who died were censored for survival analysis.

BASELINE DATA

Information, including age, sex, history of diabetes, and use of calcium carbonate, calcitriol, and lipid-lowering drugs, were obtained at baseline. Height and weight were measured according to a standard protocol, and body mass index (BMI) was calculated. Blood pressure was measured 3 times during the interview at the PD center. The causes of ESRD were mostly diagnosed based on clinical features.

A blood sample was collected after an overnight fast of ≥ 8 hours. Blood measurements, including hemoglobin, iron, transferrin, ferritin, transferrin saturation (TSAT), alkaline phosphatase (AKP), calcium, phosphorus, intact parathyroid hormone (iPTH), albumin, prealbumin, C reactive protein, glycated hemoglobin, insulin, and lipid levels (cholesterol, triglyceride, high-density lipoprotein, low-density lipoprotein), were performed using standard clinical laboratory methods. Insulin resistance was assessed using the homeostasis model assessment: (HOMA-IR = fasting serum insulin [mU/L] $\times$ fasting serum glucose [mmol/L/22.5]).

STATISTICAL ANALYSIS

Statistical analysis of the data was performed using SPSS software, version 17.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as the mean $\pm$ standard deviation (SD) or as median with quartile ranges, and categorical variables were expressed as percentages. Comparisons among the groups were undertaken using 1-way ANOVA or the Kruskal-Wallis H test for continuous variables and Pearson's chi-square test or Fisher's exact test for categorical variables. Univariate Cox regression was used to identify the potential risk factors for certain endpoints (including all-cause mortality, cardiovascular events, and cardiovascular mortality) in PD patients, and multivariate Cox regression was used to identify independent predictors of these endpoints. Covariates that were considered to be risk factors for the endpoints in univariate analysis were used for stepwise adjustments. The hazard ratios (HRs) for the endpoints were expressed per-100 unit increase in CaCS, 0.1-mg/dL increase in CRP, 0.1-g/dL increase in albumin, 10-mg/dL increase in prealbumin, 0.1 increase in urea clearance (Kt/V), 10-L/w increase in creatinine clearance (Ccr), and 0.1 increase in dialysate to plasma solute concentration

(D/P). A cumulative hazard plot (multivariate-adjusted) was further created to evaluate for differences among the 3 groups, stratified according to CaCS level. All of the statistical tests were 2-sided, and differences were considered to be statistically significant for a p value < 0.05 .

RESULTS

PATIENT CHARACTERISTICS

We recruited a total of 179 PD patients (86 men, 48%) with a mean age of 63.5 ± 14.8 years for this study. One hundred and twenty patients had received PD treatment for less than 1 year when enrolled. The patients were divided into the following 3 groups according to CaCS level (ranging from 0 to 5,257): no (CaCS = 0, $n = 54$); low ($0 < \text{CaCS} < 400$, $n = 72$); and high (CaCS ≥ 400 , $n = 53$) (Tables 1 and 2). Compared to the no CaCS group,

patients with a higher CaCS were older and more likely to be male and to have a history of diabetes. They had higher HbA_{1c} and serum insulin and triglyceride levels and lower residual renal Kt/V and Ccr, serum albumin, and high-density lipoprotein (HDL). Body mass index and systolic blood pressure were highest in the low CaCS group, while diastolic blood pressure was lowest in the high CaCS group. More than 90% of patients in our study received continuous ambulatory peritoneal dialysis (CAPD), while others received daytime ambulatory peritoneal dialysis (DAPD). All of the peritoneal dialysate were Dianeal PD4 (calcium 1.25 mmol/L, 2 L) and Dianeal PD2 (calcium 1.75 mmol/L, 2 L) from Baxter Healthcare Corporation. No icodextrin was used in our center. There were no significant differences in the use of CAPD and Dianeal PD4/PD2 among the 3 groups. There were also no significant differences in the cause of ESRD, PD duration, total Kt/V and Ccr, hemoglobin, biomarkers of iron metabolism,

TABLE 1
Baseline Clinical Characteristics of the PD Patients

	Total ($n=179$)	Coronary artery calcification score			p value
		0 ($n=54$)	1–400 ($n=72$)	≥ 400 ($n=53$)	
Age	63.5 $\pm$ 14.8	52.2 $\pm$ 15.8	65.8 $\pm$ 11.7	71.9 $\pm$ 9.6	<0.001
Sex (male)	86 (48.0%)	24 (44.4%)	29 (40.3%)	33 (62.3%)	0.043
Cause of ESRD					0.123
Glomerulonephritis	95 (53.1%)	37 (68.5%)	36 (50.0%)	22 (41.5%)	
Diabetes mellitus	60 (30.5%)	12 (22.2%)	25 (34.7%)	23 (43.4%)	
Hypertension	4 (2.2%)	0 (0%)	3 (4.2%)	1 (1.9%)	
Other	18 (10.1%)	5 (9.3%)	7 (9.7%)	6 (11.3%)	
Duration of PD (months)	5.5 (2.0, 19.6)	2.0 (2.0, 18.94)	2.0 (2.0, 11.74)	7.2 (2.0, 35.43)	0.070
History of diabetes mellitus	68 (38.0%)	12 (22.2%)	30 (41.7%)	26 (49.1%)	0.012
SBP (mmHg)	132 $\pm$ 19	129 $\pm$ 15	137 $\pm$ 20	128 $\pm$ 21	0.009
DBP (mmHg)	78 $\pm$ 12	81 $\pm$ 13	78 $\pm$ 11	75 $\pm$ 12	0.036
MAP (mmHg)	96 $\pm$ 13	97 $\pm$ 12	98 $\pm$ 12	93 $\pm$ 14	0.087
BMI (kg/m ²)	22.69 $\pm$ 3.50	21.55 $\pm$ 3.28	23.45 $\pm$ 3.88	22.85 $\pm$ 2.88	0.010
Total Kt/V	2.20 $\pm$ 0.62	2.30 $\pm$ 0.64	2.19 $\pm$ 0.62	2.12 $\pm$ 0.59	0.281
Total Ccr (L/week)	74.59 $\pm$ 31.38	79.91 $\pm$ 32.47	74.84 $\pm$ 31.62	68.84 $\pm$ 29.50	0.189
Residual renal Kt/V	0.41 (0.06, 1.00)	0.63 (0.09, 1.34)	0.50 (0.12, 1.09)	0.22 (0.00, 0.71)	0.034
Residual renal Ccr (L/week)	24.25 (3.34, 51.5)	30.5 (6.07, 62.9)	24.5 (5.87, 55.0)	15.1 (0, 35.8)	0.037
D/P	0.67 $\pm$ 0.10	0.64 $\pm$ 0.09	0.67 $\pm$ 0.12	0.68 $\pm$ 0.09	0.193
CAPD	90.5%	96.3%	91.7%	83.0%	0.059
PD4	83.1%	83.4%	82.2%	84.0%	0.378
CaCO ₃ use	37.2%	48.2%	33.3%	32.7%	0.161
Calcitriol use	30.7%	38.9%	30.6%	22.6%	0.190
Lipid-lowering drug use	37.3%	26.4%	43.1%	40.4%	0.141

PD = peritoneal dialysis; ESRD = end-stage renal disease; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; BMI = body mass index; Kt/V = urea clearance; Ccr = creatinine clearance; D/P = dialysate to plasma solute concentration; CAPD = continuous ambulatory peritoneal dialysis; PD4 = Dianeal with calcium 1.25 mmol/L; CaCO₃ = calcium carbonate; CaCS = coronary artery calcification score. The data were divided into 3 groups according to CaCS. History of diabetes mellitus indicates diabetes mellitus diagnosed before the study. Duration of PD means the period from peritoneal dialysis initiation to CaCS measurement.

TABLE 2
Baseline Laboratory Data for the PD Patients

	Total (n=179)	Coronary artery calcification score			p value
		0 (n=54)	1–400 (n=72)	≥400 (n=53)	
Hemoglobin (g/dL)	10.39±1.74	10.55±1.67	10.37±1.59	10.26±2.04	0.671
Serum iron (μg/dL)	68.4±26.9	71.6±34.1	65.4±24.0	69.6±22.8	0.427
Transferrin (g/L)	1.75 (1.51, 1.98)	1.75 (1.57, 1.97)	1.74 (1.52, 1.99)	1.76 (1.48, 2.07)	0.984
Serum ferritin (μg/L)	211 (132, 356)	184 (131, 329)	198 (123, 329)	238 (131, 405)	0.701
TSAT (%)	29.0 (22.0, 37.3)	31.5 (20.8, 40.0)	27.5 (21.0, 36.3)	31.5 (24.3, 37.0)	0.142
AKP (IU/L)	93.1±50.3	96.7±52.9	83.8±35.6	102.2±62.2	0.106
Adjusted total Ca ²⁺ (mg/dL)	9.12±0.80	9.01±0.65	8.97±0.68	9.44±0.99	0.002
Phosphorus (mg/dL)	4.51±1.15	4.60±1.61	4.49±1.30	4.45±1.12	0.825
Adjusted Ca ²⁺ × P (mg/dL) ²	41.23±13.30	41.57±15.46	40.32±12.42	42.15±12.18	0.734
iPTH (pg/mL)	242 (117, 355)	283 (140, 416)	220 (108, 327)	215 (104, 392)	0.142
CRP (mg/dL)	0.330 (0.158–0.874)	0.324 (0.167–0.594)	0.400 (0.141–1.008)	0.317 (0.141–0.944)	0.637
Albumin (g/dL)	3.29±0.47	3.45±0.49	3.24±0.47	3.20±0.41	0.011
Prealbumin (mg/dL)	28.38±8.50	28.73±9.93	28.13±8.24	28.36±7.23	0.927
HbA _{1c}	5.93±1.00	5.72±1.02	5.83±0.92	6.29±1.02	0.007
Serum insulin (pmol/L)	10.3 (7.2, 18.2)	8.8 (6.7, 12.5)	10.1 (5.9, 19.5)	15.8 (8.6, 25.1)	0.001
HOMA-IR	2.36 (1.42, 4.35)	2.23 (1.44, 3.11)	2.49 (1.24, 5.28)	3.02 (1.43, 7.09)	0.157
Cholesterol (mg/dL)	177 (150, 207)	165 (144, 198)	182 (155, 204)	179 (155, 221)	0.266
Triglycerides (mg/dL)	149 (104, 226)	126 (92, 181)	152 (112, 232)	167 (107, 258)	0.042
HDL (mg/dL)	3.82±1.11	4.11±1.39	3.82±1.03	3.52±0.79	0.023
LDL (mg/dL)	9.64±3.28	9.34±3.11	9.55±3.45	10.07±3.24	0.494

TSAT = transferrin saturation; AKP = alkaline phosphatase; iPTH = intact parathyroid hormone; CRP = C reactive protein; HbA_{1c} = glycated hemoglobin; HOMA-IR = homeostasis model of assessment-insulin resistance; HDL = high-density lipoprotein; LDL = low-density lipoprotein.

phosphorus, intact parathyroid hormone, cholesterol, low-density lipoprotein (LDL) or use of CaCO₃, calcitriol, and lipid-lowering drugs.

The follow-up duration was 30.6 ± 16.2 (24 – 63) months. During the follow-up period, 70 patients died and 33 patients experienced CVEs. Among the patients who died, 25 patients died of CVEs and 9 patients died of heart attack or coronary disease. According to the CaCS level, 9 of 52 patients died in the no CaCS group, while 25 of 73 patients died in the low CaCS group, and 31 of 53 patients died in the high CaCS group.

CACS WAS AN INDEPENDENT PREDICTOR OF MORTALITY AND CARDIOVASCULAR EVENTS IN PD PATIENTS

We used univariate Cox regression to examine the potential risk factors for the endpoints, including all-cause mortality, CVEs, and cardiovascular mortality. We found that advanced age, a high CaCS, and low albumin were risk factors for all 3 endpoints of PD patients (Supplemental Table 1). To examine whether CaCS was an independent predictor, we then used Cox regression to adjust stepwise for the other risk factors for the

3 endpoints, and the results showed that when adjusted for the other risk factors, the CaCS was still associated with all 3 endpoints (Supplemental Table 2). As listed in Table 3, we found that advanced age, low albumin, a high CaCS, HOMA-IR, and adjusted Ca²⁺ were independent risk factors for all-cause mortality, while low albumin and a high CaCS were independent risk factors for CVEs. We also used the different CaCS levels (0, 0 – 400, ≥ 400) as cut-off points to divide the patients into 3 groups and create hazard plots (Figure 1), which showed that the cumulative hazard for the endpoints of the patients with a higher CaCS was significantly higher than those of the patients with a lower CaCS ($p = 0.028$ for all-cause mortality, 0.031 for cardiovascular events, and 0.017 for cardiovascular mortality).

DISCUSSION

This study aimed to assess whether coronary calcification could predict prognosis in a cohort of patients receiving PD. Primary and secondary endpoints, including all-cause mortality, CVEs, and cardiovascular mortality, were analyzed. The results showed that compared with

TABLE 3
Multivariate Analysis for Risk Factors of Mortality and Cardiovascular Events in PD Patients

	Mortality		Cardiovascular events		Cardiovascular deaths	
	HR (95% CI)	<i>p</i> value	HR (95% CI)	<i>p</i> value	HR (95% CI)	<i>p</i> value
Age	1.035 (1.019–1.052)	0.023	1.012 (0.977–1.048)	0.514	1.011 (0.970–1.054)	0.599
Sex (male)	1.325 (0.775–2.265)	0.304	1.297 (0.609–2.760)	0.500	1.064 (0.434–2.609)	0.892
CaCS (per 100)	1.041 (1.010–1.073)	0.010	1.052 (1.014–1.092)	0.007	1.062 (1.017–1.110)	0.007
BMI (kg/m ²)	1.012 (0.932–1.099)	0.776	1.093 (0.988–1.209)	0.085	1.059 (0.935–1.200)	0.366
Albumin (per 0.1 g/dL)	0.906 (0.852–0.964)	0.002	0.895 (0.819–0.977)	0.014	0.898 (0.818–0.978)	0.049
HbA _{1c}	1.228 (0.945–1.595)	0.124	1.160 (0.809–1.664)	0.420	1.493 (1.005–2.218)	0.047
HOMA-IR	1.036 (1.008–1.065)	0.011	1.019 (0.968–1.072)	0.481	1.020 (0.961–1.082)	0.514
DBP (mm Hg)	0.981 (0.953–1.010)	0.194	1.018 (0.980–1.058)	0.361	1.000 (0.952–1.051)	0.992
Adjusted Ca ²⁺ (mg/dL)	1.529 (1.023–2.286)	0.038	0.918 (0.502–1.678)	0.780	1.312 (0.660–2.608)	0.439
Duration of PD	0.989 (0.978–1.001)	0.079	0.999 (0.987–1.012)	0.924	0.991 (0.974–1.010)	0.355

PD = peritoneal dialysis; HR = hazard ratio; CI = confidence interval; CaCS = coronary artery calcification score; BMI = body mass index; HbA_{1c} = glycated hemoglobin; HOMA-IR = homeostasis model of assessment-insulin resistance; DBP = diastolic blood pressure. Cox regression was used to calculate HRs for the endpoints. The HRs were expressed as per 100 increase for CaCS and per 0.1 g/dL increase for albumin.

Figure 1A — Cumulative hazard ratios for all-cause mortality according to different CaCS levels. CaCS was divided into 3 groups: CaCS = 0; 0 < CaCS < 400; and CaCS ≥ 400. Patients with a higher CaCS had a significantly increased all-cause mortality ($p = 0.027$) after adjusting for age, sex, BMI, albumin, HbA_{1c}, HOMA-IR, DBP, adjusted Ca²⁺ and duration of PD. CaCS = coronary artery calcification score; BMI = body mass index; HOMA-IR = homeostasis model of assessment-insulin resistance; DBP = diastolic blood pressure; PD = peritoneal dialysis.

the patients with coronary calcification, those with no coronary calcification survived significantly longer and had fewer CVEs and lower cardiovascular mortality, while those with a CaCS > 400 had the poorest prognosis. This analysis indicated that the CaCS contributed significantly to the prediction of mortality and CVEs, in addition to other risk factors, in PD patients.

Figure 1B — Cumulative hazard ratios for the cardiovascular events according to different CaCS levels. CaCS was divided into 3 groups: CaCS = 0; 0 < CaCS < 400; and CaCS ≥ 400. Patients with a higher CaCS had a significantly increased rate of cardiovascular events ($p = 0.020$) after adjusting for age, sex, BMI, albumin, HbA_{1c}, HOMA-IR, DBP, adjusted Ca²⁺ and duration of PD. CaCS = coronary artery calcification score; BMI = body mass index; HOMA-IR = homeostasis model of assessment-insulin resistance; DBP = diastolic blood pressure; PD = peritoneal dialysis.

Consistent with previous studies, coronary calcification was common in ESRD patients (17). In Goodman *et al.*'s study in 2000, 14 of 16 dialysis patients who developed ESRD in childhood or adolescence and were aged 20 to 30 years had evidence of coronary calcification, and its severity was strongly associated with age and dialysis

Figure 1C — Cumulative hazard ratios for cardiovascular mortality according to different CaCS levels. CaCS was divided into 3 groups: CaCS = 0; $0 < \text{CaCS} < 400$; and $\text{CaCS} \geq 400$. Patients with a higher CaCS had a significantly increased cardiovascular mortality ($p = 0.035$), after adjusting for age, sex, BMI, albumin, HbA_{1c} , HOMA-IR, DBP, adjusted Ca^{2+} and duration of PD. CaCS = coronary artery calcification score; BMI = body mass index; HOMA-IR = homeostasis model of assessment-insulin resistance; DBP = diastolic blood pressure; PD = peritoneal dialysis.

duration (18). Another study reported that coronary calcification was present in 29 of 49 PD patients (59.2%), which was lower than in our study (19). In our patients, coronary calcification was present in 83 of 120 patients who had received dialysis for < 1 year (69.2%) and in 42 of the 59 patients who had received dialysis for > 1 year (71.2%). Even in the 88 incidental PD patients, coronary calcification was found in 61 patients (69.3%). Coronary calcification at the initiation of PD was as prevalent as in those patients already undergoing dialysis, suggesting that coronary calcification could develop rapidly during chronic kidney disease (CKD) progression before dialysis. These results were consistent with the study by Kramer *et al.*, which found that diabetic patients with stage 3 – 5 CKD experienced a 9-fold increase in $\text{CaCS} > 10 / \text{CaCS} < 10$, compared with those without CKD (20).

Although coronary calcification is common in ESRD patients, few studies have focused on its correlation with prognosis. Most of the prognostic studies were performed in the general population. Clearly, the presence of coronary calcification is significantly associated with CVDs and all-cause mortality, and the CaCS has provided improved predictive ability beyond that provided by standard risk factors in asymptomatic individuals. For example, a study by Detrano *et al.* that included 6,722 individuals who underwent coronary calcium examinations reported that

compared to the participants with no coronary calcium, the adjusted risk of a coronary event was increased by a factor of 7.73 among the participants with $100 < \text{CaCS} < 300$ and by a factor of 9.67 among the participants with a $\text{CaCS} > 300$ (7). Another study that included a multi-ethnic cohort of 5,878 subjects indicated that adding the CaCS to a prediction model based on traditional risk factors significantly improved risk classification (21). A systematic review of 9 studies showed that the CaCS improved risk stratification in cardiovascular risk categories when added to traditional risk factors, especially among individuals at intermediate risk for cardiovascular disease (4,8,9,21–26). However, the patients with CKD or ESRD represent a special population because of their notable calcium-phosphorus disorders, and artery calcification is much more common in this group than in the general population, albeit via a different mechanism. The results for the general population cannot be extended to the CKD population. Therefore, we conducted this study, and the results also indicated that the CaCS was an independent risk factor for all-cause mortality and CVDs, consistent with other studies in hemodialysis patients (27,28).

In our prospective cohort, all of the patients at our center were referred to have their CaCS calculated, and the endpoints were all validated by our clinical doctors, not only the patients' raw reports, which made the bias as low as possible. However, there were still some limitations. First, this report was an observational, single-center, and relatively small-sized study, and our results described only the association between coronary calcification and prognosis, but not the causes of coronary calcification. Using the baseline data, the correlations between risk factors and the CaCS were analyzed, and the results showed that age, HbA_{1c} , serum insulin, and triglyceride levels were positively associated with the CaCS, while diastolic blood pressure, residual renal function, albumin, and high-density lipoprotein levels were negatively associated with the CaCS. However, this study used cross-sectional analysis, and the association did not prove causality; therefore, additional studies are needed to determine the risk factors for coronary calcification or calcification progression. Second, the study population consisted of a heterogeneous population of ESRD patients, who suffered from glomerulonephritis, hypertension, or diabetes for different periods of time before dialysis, and these factors also contributed to coronary calcification and prognosis. However, the exact period of time could not be determined in the present study. In addition, even if coronary calcification predicted a worse prognosis, and these ESRD patients were classified into a high-risk group, there were few beneficial

interventions, unlike in the general population. Hence, cost-effectiveness analysis is needed before the widespread use of CaCSs in ESRD patients.

In conclusion, the present study assessed the correlation between the CaCS and prognosis in patients receiving PD. A higher CaCS in these patients was independently associated with higher rates of all-cause mortality, cardiovascular events, and cardiovascular mortality, after adjusting for other risk factors. The coronary artery calcification score could be a useful risk stratification tool for PD patients.

DISCLOSURES

The authors have no financial conflicts of interest to declare. This study was supported by the Science Development Foundation of Baoshan District (09-E-24) and the National Science and Technology Ministry (ID: 2011BAI10B05). Qionghong Xie was supported by the Natural Science Foundation of China (NSFC: 81300569).

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