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Pretransplant dialysis treatment and vascular calcification of the iliac artery and abdominal aorta in kidney transplant patients

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Abstract

Introduction: Patients with chronic kidney disease often suffer from cardiovascular disease, and vascular calcification has been identified as one of the risk factors for cardiovascular disease. We aimed to examine the effect of dialysis treatment before kidney transplantation on graft survival, vascular calcification, and its progression after kidney transplantation.

Methods: Among the 102 patients who underwent kidney transplant between 2008 and 2017, two patients were excluded for moved and lost to follow-up and primary nonfunction. The clinical characteristics and laboratory data were assessed according to pretransplant treatment modality. Rapid progression of vascular calcification was defined when patients showed an increase in the highest tertile of progression of each iliac artery calcification thickness (IACT) and aortic calcification index (ACI).

Results: Cox proportional hazard models did not show any significant association between pretransplant treatment modality and graft survival to the doubling of creatinine from nadir creatinine during the first 3 months after kidney transplantation. At baseline, the IACT was significantly higher in hemodialysis patients than in preemptive kidney transplant patients, whereas the ACI was comparable among the pretransplant treatment modality groups. IACT was independently associated with dialysis vintage. There was no significant association between rapid progression of vascular calcification (IACT and ACI) and dialysis modality.

Conclusions: Dialysis modality was an independent factor related to IACT, whereas there was no legacy effect for the progression of vascular calcification after kidney transplantation.

Keywords: Aortic calcification index, Hemodialysis patients, Iliac artery calcification thickness, Peritoneal dialysis, Preemptive kidney transplantation, Vascular calcification

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Introduction

It is well known that cardiovascular complications predict the prognosis of patients and that vascular calcification is a risk factor for cardiovascular disease and mortality. Coronary artery calcification in particular correlates with cardiovascular events [1–3] and mortality [4, 5]. A past study also showed that abdominal aortic calcification is a strong predictor of cardiovascular and cerebrovascular events and death [6]. However, these reports involve the general population, and there are few reports specifically on chronic kidney disease patients. It has not been clarified whether vascular calcification of the aorta and common iliac artery may impact clinical outcomes after kidney transplantation. Moreover, there are few studies comparing the effect of dialysis treatment on vascular calcification, and there is no report about the legacy effect of dialysis treatment performed before kidney transplantation.

The measurement of the calcification of the iliac artery may be convenient and useful in comparison with aortic calcification index (ACI) which is time-consuming, complicated, and not available in clinical practice. Although peripheral arterial disease has been associated with poor graft survival and high mortality after kidney transplantation [7], it remains controversial as to whether iliac artery calcification is associated with graft survival and mortality [8, 9].

We conducted this study to clarify the relationship between vascular calcification in this area and pretransplant dialysis treatment or preemptive kidney transplantation, and the development of vascular calcification after kidney transplantation.

Materials and methods

Study design

Among the patients who underwent kidney transplant in the decade between 2008 and 2017, two patients were excluded for moved and lost to follow-up (n = 1) and primary nonfunction (n = 1). Consequently, the study population comprised 100 patients, including 61 who had been on hemodialysis, 22 on peritoneal dialysis, and 17 who underwent preemptive kidney transplantation (cross-sectional cohort). We analyzed a longitudinal sample of 62 patients who were scanned twice with an interval of at least half a year (longitudinal cohort). Data were collected through history taking, physical examination, and laboratory findings. Study variables were age, sex, co-morbid medical conditions, medications, body weight, height, systolic and diastolic blood pressure, laboratory findings, including creatinine, calcium, phosphate, HbA1c, parathyroid hormone, and low-density lipoprotein (LDL) cholesterol. Demographic information, underlying medical conditions, and medications were documented at the time of hospitalization. Following the previous report, cardiovascular disease (CVD) events were defined as stroke, myocardial infarction, hospitalization for unstable angina, coronary intervention (coronary artery bypass surgery or angioplasty), hospitalization for heart failure, and peripheral artery disease [10, 11]. The study complies with the ethical guidelines of the 1975 Declaration of Helsinki, was approved by the appropriate institutional review committee (authorization No. 448). The board waived the need for patient consent, since retrospective data originated from standard care (in which one can opt-out for the use of their data for scientific research).

Assessment of vessel calcification

A previous study reported that maximal thickness was only associated with kidney transplant outcomes for three parameters of iliac artery calcification: (1) maximal thickness, (2) greatest percentage circumference, and (3) percentage length [8]. Regions of interest were placed around all lesions found within the bilateral common iliac arteries. The threshold for a calcific lesion was set at a computed tomographic density of 130 Hounsfield units within an area of ≥ 1 mm^2 (Fig. 1). ACI was obtained as previous reported [12, 13]. Briefly, the images were obtained with a 5-mm slice thickness from the site where the renal artery arises to the bifurcation into the common iliac arteries of the aorta. Calcification was present if an area displayed a density of 130 Hounsfield units. The cross-section of the abdominal aorta on each slice was radially divided into 12 segments. Aortic calcification index was calculated as follows: ACI = ([total calcification score on 10 slices]/[12 × 10]) × 100%.

Statistical analysis

Patient clinical characteristics and laboratory data are shown in each pretransplant treatment modality (hemodialysis [HD], peritoneal dialysis [PD], and preemptive kidney transplantation [PEKT]). Data for categorical variables are given as number of patients (%) and data for continuous variables are given as mean ± standard deviation or median (interquartile range: IQR) values. Rapid progression was defined when patients showed increasing highest tertile of each ACI and increasing iliac artery calcification thickness (IACT).

Differences between groups were evaluated, when appropriate, using a student's t test and Turkey post-hoc test for multiple comparisons for continuous variables and the chi-squared test for categorical variables. Spearman correlation coefficients were calculated for univariate analysis of the associations among clinical and biochemical variables. Cox proportional hazards models evaluated the associations of pretransplantation treatment modality with graft survival, initially without adjustment, and subsequently adjusting for confounding factors, as follows: (1) the demographic adjusted model

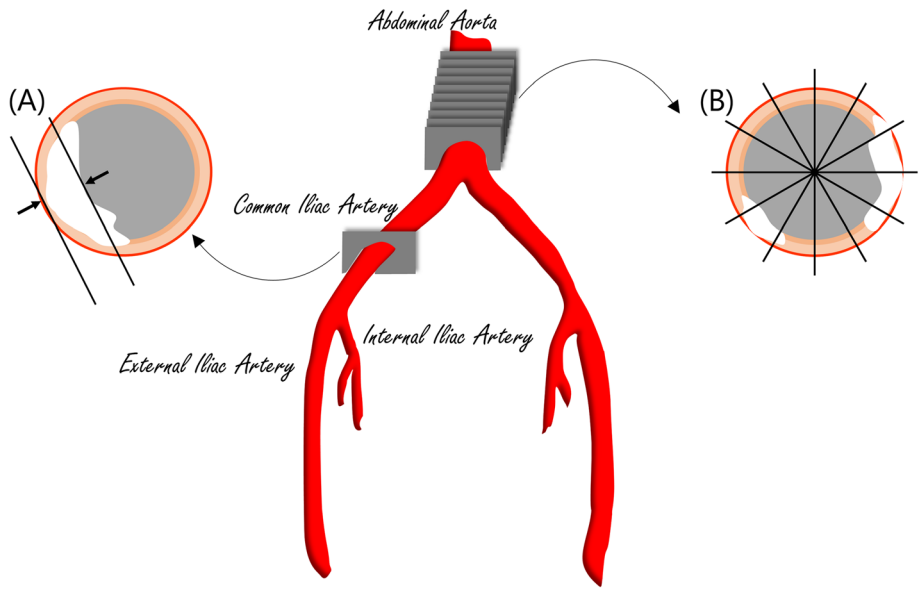

Fig. 1 Assessment of iliac artery calcification and abdominal aorta. Regions of interest are placed around all lesions found within bilateral common iliac arteries (**A**). The maximum thickness of common iliac artery calcification is measured perpendicular to the vessel wall. The threshold for a calcific lesion is set at a computed tomographic density of 130 Hounsfield units within an area of ≥ 1 mm^2. The aortic calcification index (ACI) is obtained, as previous reported. Briefly, the cross-section of each of 10 slices of the abdominal aorta is divided into 12 sectors (**B**). The number of sectors showing calcification in each slice is counted. ACI is calculated using the following formula: ACI = ([total calcification score on 10 slices]/[12 × 10]) × 100%

adjusted for age and gender; (2) the fully adjusted model adjusted for the demographic variables as well as complications of diabetes mellitus, nadir creatinine (lowest creatinine during the first 3 months after transplantation), dialysis vintage, body mass index (BMI), donor-specific antigen, ABO incompatibility, rejection after transplantation, and baseline IACT or ACI. Multiple stepwise linear regression analysis was performed to assess potential independent predictors of vascular calcification and its rapid progression. These were also evaluated with repeated-measures ANOVA. The adjusted odds ratios were provided with 95% confidence intervals and p values. All statistical tests were two-sided, and a $p < 0.05$ was considered statistically significant. The statistical analyses were performed with the JMP version 13 (SAS institute Inc., Cary, NC, USA).

Results

Study population

The final study population included 100 patients with a mean age of 47 ± 13 years, 69% were male, 18% had diabetes, and 80% received living kidney transplantation. At baseline, calcification of the common iliac artery was present in 62% of patients, and the IACT was 2.4 (IQR, 0–4.6) mm. Similarly, abdominal aortic calcification was present in 66% of patients, and median ACI was 10 (IQR, 0–30). The mean interval of CT from kidney transplantation was 40.3 ± 28.6 months, and the mean duration of follow-up was 68.2 ± 29.1 months. During the follow-up period, four (4%) patients died, eight (8%) had graft loss, and 18 (18%) showed a doubling of creatinine from baseline (lowest creatinine during the first 3 months after transplantation). Baseline characteristics of study subjects according to pretransplant treatment modality are shown in Table 1. Prevalence of male sex, diabetes, history of CVD, and BMI were comparable among the three groups. Dialysis vintage was longer in the HD group than the PD group. There were no significant differences among the three groups in terms of immunological factors, such as ABO incompatibility, donor-specific antigen, and HLA mismatch counts. In the cross-sectional cohort, diastolic blood pressure was higher in the PEKT group than the HD group. The prevalence of living donor transplantation was higher in the PD and PEKT groups than the HD group. In the longitudinal cohort, patients in the PEKT group were significantly younger than the HD group (not significant in cross-sectional cohort).

Association of dialysis modality with vascular calcification, graft survival, and mortality

At baseline, the prevalence of calcification of the common iliac artery was comparable among the three groups (70% in HD group, 55% in PD group and 41% in PEKT group, $p = 0.07$). Similarly, the prevalence of abdominal aortic calcification was comparable among the three groups (74% in HD group, 55% in PD group and 53% in PEKT group, $p = 0.12$). The baseline IACT was

Table 1 Characteristics of the 100 patients included in the cross-sectional analysis and of 62 patients included in the progression analyses, stratified by pre-transplant treatment modality

	Cross-sectional cohort			Longitudinal cohort		
	HD (*n* = 61)	PD (*n* = 22)	PEKT (*n* = 17)	HD (*n* = 39)	PD (*n* = 16)	PEKT (*n* = 7)
Age, years	47.9 ± 12.5	47.3 ± 14.9	44.2 ± 13.2	48.5 ± 10.7	46.3 ± 14.6	36.3 ± 11.5 [a]
Male, *n* (%)	39 (64)	16 (73)	14 (82)	24 (62)	13 (81)	5 (71)
Diabetes mellitus, *n* (%)	12 (20)	3 (14)	3 (18)	8 (21)	1 (6)	1 (14)
Dialysis vintage, months	91 [11.5, 180.5]	21.5 [11.5, 35.3] [a]	–	91 [12, 179]	19.5 [7, 32.5] [a]	–
History of CVD, *n* (%)	8 (13)	1 (5)	0 (0)	6 (15)	0 (0)	0 (0)
Body mass index	21.3 ± 3.3	22.3 ± 3.3	21.6 ± 4.4	21.7 ± 3.6	21.8 ± 2.5	20.0 ± 4.9
Systolic BP, mmHg	130 ± 16	125 ± 13	129 ± 10	130 ± 16	123 ± 12	129 ± 14
Diastolic BP, mmHg	76 ± 10	76 ± 10	83 ± 10 [a]	76 ± 10	76 ± 11	79 ± 13
ABO incompatible, *n* (%)	19 (31)	5 (23)	6 (35)	15 (38)	5 (31)	1 (14)
Donor-specific antigen, *n* (%)	13 (21)	1 (5)	5 (29)	7 (18)	1 (6)	1 (14)
HLA mismatch count (A, B, DR)	3.1 ± 1.4	3.6 ± 1.4	3.1 ± 1.9	3.3 ± 1.5	3.8 ± 1.5	3.0 ± 2.2
Donor characteristics						
Living donor, *n* (%)	43 (70)	20 (91) [a]	17 (100) [a]	30 (77)	15 (94)	7 (100)
Male, *n* (%)	32 (52)	7 (32)	7 (41)	25 (64)	6 (38)	4 (57)
Age, years	52.3 ± 13.2	56.9 ± 10.1	56.5 ± 8.0	54.0 ± 12.5	56.3 ± 11.1	55.9 ± 5.6
Body weight, kg	58.2 ± 9.2	55.2 ± 10.1	55.3 ± 9.3	59.8 ± 9.4	55.6 ± 11.4	57.9 ± 10.2
Medication before transplantation, *n* (%)						
Calcium-based phosphate binders	39 (64)	13 (59)	2 (12)	26 (67)	9 (56)	1 (14)
Calcium-free phosphate binders	29 (48)	12 (55)	1 (6)	16 (41)	7 (44)	1 (14)
Vitamin D	48 (79)	8 (36)	2 (12)	30 (77)	6 (38)	1 (14)
Calcimimetics	18 (30)	2 (9)	0 (0)	11 (28)	2 (13)	0 (0)
HbA1c, %	5.5 ± 0.8	5.3 ± 0.3	5.3 ± 0.5	5.6 ± 0.8	5.3 ± 0.3	5.5 ± 0.4
Whole PTH, pg/mL	89.2 [34.8, 162.0]	117.0 [78.4, 145.3]	204.0 [176.5, 262.0] [a]	74.9 [29.3, 189.0]	123.6 [80.4, 150.5]	215.0 [202.0, 238.0]
Calcium, mg/dL	9.5 ± 0.9	9.1 ± 0.7	8.5 ± 1.3 [a]	9.4 ± 1.0	9.1 ± 0.7	8.2 ± 1.7 [a]
Phosphate, mg/dL	4.9 ± 1.2	5.6 ± 1.2	5.3 ± 1.6 [a, b]	4.9 ± 1.3	5.5 ± 1.3	5.4 ± 2.1
LDL cholesterol, mg/dL	91.2 ± 31.9	91.9 ± 26.5	84.2 ± 33.9	88.3 ± 30.4	89.3 ± 25.9	78.5 ± 31.9
Nadir creatinine, mg/dL	1.16 ± 0.45	1.05 ± 0.19	1.05 ± 0.23	1.13 ± 0.41	1.05 ± 0.17	0.98 ± 0.22
Laboratory data at 3 months after transplantation						
Calcium, mg/dL	10.1 ± 1.1	10.1 ± 0.6	9.3 ± 0.3 [a, b]	10.1 ± 1.1	10.1 ± 0.7	9.3 ± 0.4
Phosphate, mg/dL	2.8 ± 0.8	2.7 ± 0.6	2.8 ± 0.5	2.9 ± 0.9	2.6 ± 0.6	2.7 ± 0.5
LDL cholesterol, mg/dL	116.3 ± 30.3	111.8 ± 27.0	106.2 ± 28.1	116.0 ± 28.6	115.5 ± 26.1	87.1 ± 17.5 [a]

Data are expressed as the mean ± standard deviation, median [interquartile range], or number of patients (%). *HD* hemodialysis, *PD* peritoneal dialysis, *PEKT* pre-emptive kidney transplantation, *CVD* cardiovascular diseases, *BP* blood pressure, *PTH* parathyroid hormone, *LDL* low-density lipoprotein

[a] $p < 0.05$ compared to HD patients

[b] $p < 0.05$ compared to PD patients

significantly higher in the HD group than the PEKT group ($p = 0.049$, Fig. 2A). The baseline ACI was comparable among the three groups (Fig. 2B). Death and RRT were comparable among the three groups (Log-rank test, $p = 0.34$), and the time for doubling of serum creatinine from nadir creatinine during the first 3 months after kidney transplantation was comparable among the three groups (Log-rank test, $p = 0.70$, Fig. 3). After adjustment for multiple variables, including ACI and IACT, Cox proportional hazard regression models showed that pretransplant treatment modality was not associated with doubling of creatinine (Table 2). The results of stepwise multiple regression analyses in terms of the determinants of vascular calcification at kidney transplantation are shown in Table 3. In multivariate regression analyses, age, sex, diabetes, and dialysis vintage

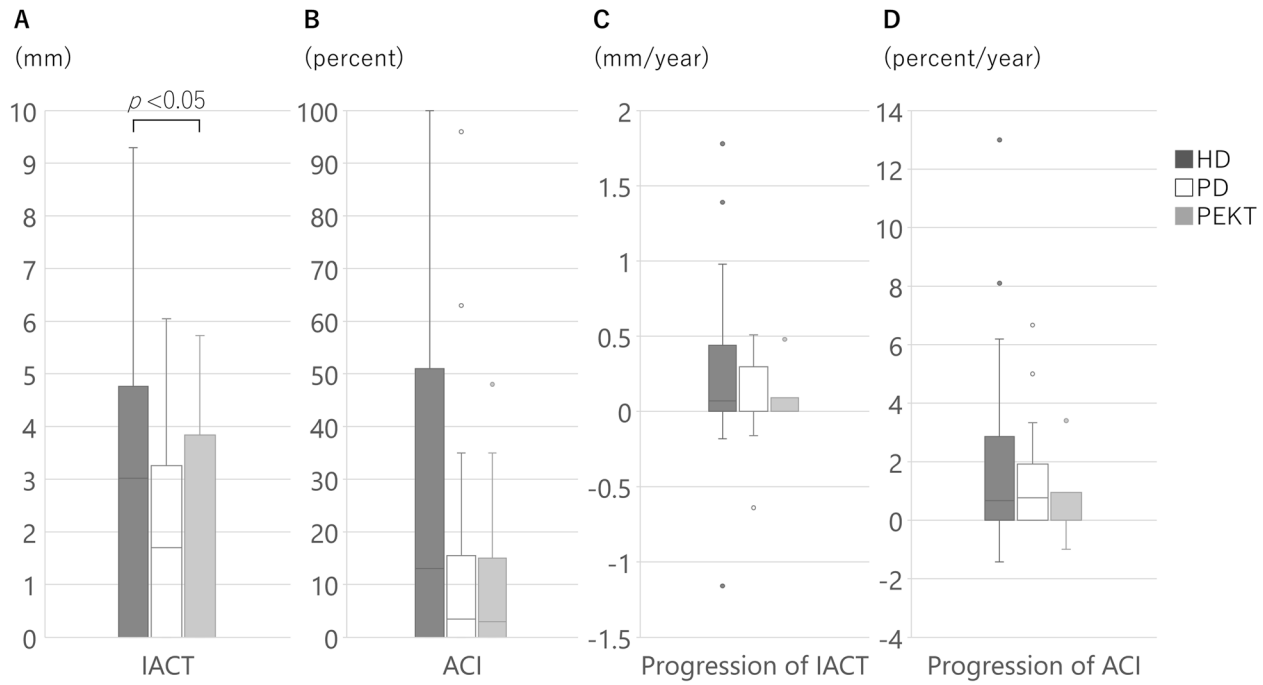

Fig. 2 Baseline and progression of vessel calcification. Boxplots (minimum, bottom quartile, median, top quartile, maximum) are shown for iliac artery calcification thickness (IACT, **A**) and aortic calcification index (ACI, **B**). IACT is significantly higher in the HD group than in the PEKT group, whereas ACI is comparable among the three groups. There are no significant differences in terms of progression of IACT (**C**) and ACI (**D**) among the three groups

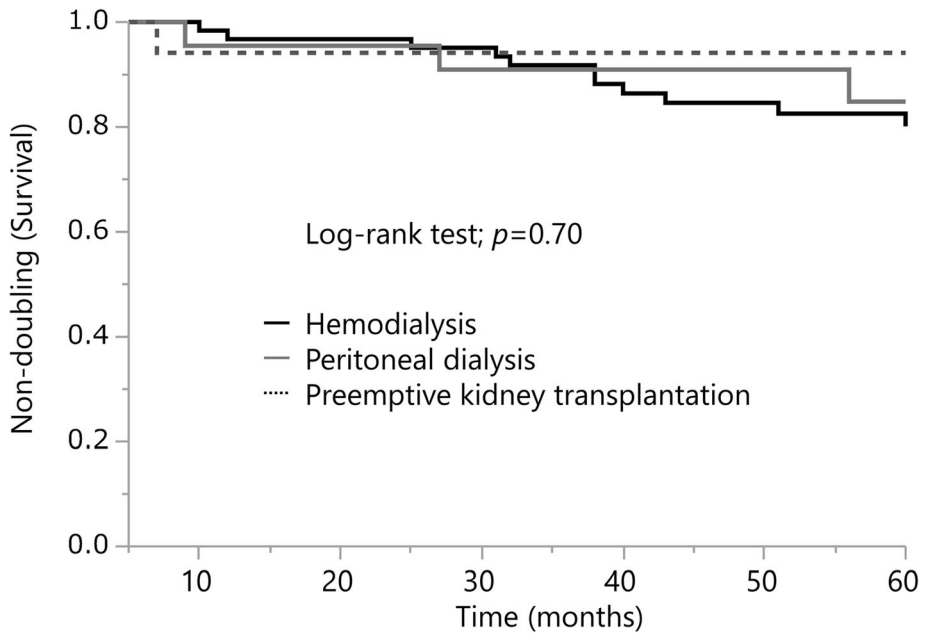

Fig. 3 Kaplan-Meier curves for the doubling of serum creatinine. Kaplan-Meier curves for the doubling of serum creatinine in incident transplant patients by pretransplant treatment modality

Table 2 Association of pretransplant treatment modality and graft survival to the doubling of creatinine (N=100).

	PEKT	HD	PD
Unadjusted: HR (95% CI)	1.00 (reference)	2.27 (0.44–41.6)	1.77 (0.23–35.9)
Demographic adjusted: HR (95% CI)[a]	1.00 (reference)	1.94 (0.36–35.9)	1.71 (0.22–34.7)
Multivariable adjusted: HR (95% CI)[b]	1.00 (reference)	3.50 (0.49–74.1)	3.31 (0.25–88.6)

HR hazard ratio, *CI* confidence interval, *HD* hemodialysis, *PD* peritoneal dialysis, *PEKT* pre-emptive kidney transplantation
[a]Adjusted for age and gender
[b]Adjusted for demographic variables and body mass index, donor-specific antigen, ABO incompatible, rejection after transplantation, aortic calcification index, and iliac arteries calcification thickness

were associated with IACT at baseline. Similarly, age, diabetes, blood pressure, and prescription of calcimimetics before transplantation were associated with ACI at baseline. Dialysis modality was not a significant predictor of vascular calcification (IACT nor ACI), when the degree of calcification was evaluated as continuous variables. Moreover, dialysis modality did not correlate with the presence or absence of calcification of common iliac artery (odds ratio = 2.92, confidence interval = 0.46–18.7, p = 0.26 , HD vs. PD) and abdominal aorta (odds ratio = 3.71, confidence interval = 0.69–19.9, p = 0.13 , HD vs. PD).

Dialysis modality as determinant of progression of vascular calcification

After kidney transplantation, the progression rate of IACT and ACI was 0.03 (IQR, 0–0.33) mm per year and 0.61 (IQR, 0–2.36) per year, respectively. Figure 2C, D shows that the progression rate of IACT and ACI was comparable among the three groups by pretransplant treatment modality. The results of stepwise multiple regression analyses of progression of vascular calcification after kidney transplantation are shown in Table 4. In multivariate regression analyses, diabetes and LDL cholesterol were associated with progression of IACT after kidney transplantation (diabetes: F (1, 33) = 5.621, p = 0.024; LDL cholesterol: F (1, 33) = 4.313, p = 0.046). Similarly, age and presence of donor-specific antigen tended to be associated with progression of ACI after kidney transplantation (age: F (1, 58) = 2.983, p = 0.090; BMI: F (1, 58) = 0.062, p = 0.804, donor-specific antigen: F (1, 58) = 3.053, p = 0.086). On the other hand, no significant association was found between progression of vascular calcification (IACT nor ACI) and dialysis modality, when the progression of calcification was evaluated as continuous variables. Moreover, dialysis modality did not correlate with the rapid progression of IACT (odds ratio = 1.78, confidence interval = 0.24–13.3, HD vs. PD) and ACI (odds ratio = 1.79, confidence interval = 0.31–10.3, HD vs. PD). Significant predictors of rapid progression of IACT were age (odds ratio = 1.13, confidence interval = 1.05–1.25), male sex (odds ratio = 12.4, confidence interval = 1.79–85.8), and presence of diabetes (odds ratio = 9.25, confidence interval = 1.11–76.9), after adjustment with other covariates (age, sex, diabetes, pretransplant treatment modality, dialysis vintage, calcimimetics prescription, systolic and diastolic blood pressure). Similarly, age was significantly associated with rapid progression of ACI after adjustment with other covariates (odds ratio = 1.08, confidence interval = 1.02–1.17).

Discussion/conclusion

The present study showed that long dialysis period was associated with vascular calcification of the common iliac artery, as well as age and the presence of diabetes at kidney transplantation. Whereas, dialysis modality did not contribute to the progression of vascular calcification, unlike sex and diabetes after kidney transplantation. Moreover,

Table 3 Stepwise multiple regression analyses for iliac artery calcification thickness (IACT) or aortic calcification index (ACI) (n = 100)

	Analysis of IACT			Analysis of ACI		
	β	95% CI	*P* value	β	95% CI	*P* value
Age, years	0.09	0.06 to 0.11	< 0.01	0.76	0.45 to 1.06	< 0.01
Male	0.70	0.33 to 1.07	< 0.01	–	–	–
Diabetes mellitus	0.75	0.28 to 1.22	< 0.01	7.01	1.80 to 12.22	< 0.01
Dialysis vintage, months	0.009	0.005 to 0.013	< 0.01	–	–	–
Systolic blood pressure, mmHg	–	–	–	0.81	0.51 to 1.11	< 0.01
Diastolic blood pressure, mmHg	–	–	–	− 0.60	− 1.02 to − 0.17	< 0.01
Calcimimetics	–	–	–	5.10	0.42 to 9.77	0.03

IACT iliac arteries calcification thickness, *ACI* aortic calcification index. Plausible predictors (age, gender, diabetes mellitus, pretransplant treatment modality, dialysis vintage, body mass index, past history of cardiovascular disease, systolic and diastolic blood pressure, calcium-based and calcium-free phosphate binder, vitamin D therapy, calcimimetics, HbA1c, parathyroid hormone, calcium, phosphate, and low-density lipoprotein cholesterol) were included in the original model

Table 4 Stepwise multiple regression analyses for progression of iliac arteries calcification thickness (IACT) or aortic calcification index (ACI) ($n = 62$)

	Analyses of progression of IACT			Analyses of progression of ACI		
	β	95% CI	*P* value	β	95% CI	*P* value
Age	–	–	–	0.059	0.007 to 0.110	0.03
Diabetes mellitus	0.24	0.06 to 0.43	0.01	–	–	–
Body mass index, kg/m^2	–	–	–	0.0045	− 0.1769 to − 0.1859	0.96
Donor specific antigen	–	–	–	− 0.70	− 1.57 to 0.16	0.11
LDL cholesterol, mg/dL	0.006	0.001 to 0.012	0.02	–	–	–

IACT iliac arteries calcification thickness, *ACI* aortic calcification index, *LDL* low-density lipoprotein. Variable selection is performed by a stepwise backward selection method with a threshold of 0.1 for removal from the model. Plausible predictors (age, gender, diabetes mellitus, pretransplant treatment modality, dialysis vintage, body mass index, past history of cardiovascular disease, donor-specific antigen, ABO incompatible, rejection, nadir creatinine, calcium, phosphate, and LDL cholesterol) were included in the original model

graft and patient survival was good regardless of the type of pretransplant dialysis modality.

A recent meta-analysis suggested that there were no differences in survival rates based on the method of dialysis in incident dialysis patients (overall pooled hazard ratio of death for PD versus HD was 1.06 [95% CI 0.99–1.14]) [14]. In terms of coronary artery calcification, PD was not significantly associated with higher progression of vascular calcification during the 3-year observation period, after adjustment for age, sex, and dialysis vintage [15]. The presence of coronary artery calcification in kidney transplant patients was associated with age and dialysis vintage; however, its progression was significantly associated with age, but not with dialysis vintage [16]. Eight of 67 patients in that study were receiving PD before kidney transplantation, but the association with dialysis modality was not described. As far as we know, this is the first report to show that the pretransplant dialysis modality did not influence the vascular calcification and its progression after kidney transplantation. Another report showed that before the initiation of dialysis therapy, vascular calcification was present and associated with mineral bone disorder and uremic toxins in chronic kidney disease patients [12]. Our results showed that PD patients had a shorter history of dialysis and tended to have less vascular calcification, as did patients who underwent transplantation without dialysis. Multivariate analysis that included dialysis vintage, showed no relationship between the dialysis modality and the progression of vascular calcification.

Recent studies have demonstrated that zero coronary artery calcium predicted a low cardiovascular event rate, which was close to 1% in a 10-year risk assessment in a general population free from CVD at baseline [3]. In non-dialyzed chronic kidney disease patients, severe abdominal aorta calcification was associated with mortality [17], and coronary artery calcification was associated with kidney survival [18]. Whereas, although calcification of the abdominal aorta and iliac artery is associated with surgical complexity and of delayed graft function in kidney transplant patients [19], the association between kidney survival and vascular calcification has not been well studied. Our study showed no apparent significant association between vascular calcification and kidney survival.

A major limitation of this study was the relatively short observational period (the mean duration of follow-up was 68.2 ± 29.1 months). In one multi-center cohort study involving 1614 kidney transplant patients, the rate of CVD events increased markedly after 7.5 years of follow-up, especially in patients with diabetic nephropathy and CVD history [20]. In order to evaluate patient and kidney survival, longer observation periods seem to be necessary, and this is why surrogate markers of progression of vascular calcification and doubling of creatinine were evaluated in this study.

In conclusion, this study suggests that dialysis vintage was an independent variable related to calcification of the common iliac artery, whereas dialysis modality was not a significant predictor of vascular calcification and its progression in this area. Pretransplant dialysis treatment was associated with vascular calcification at transplantation but did not adversely affect subsequent prognosis.

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Authors' contributions

KN contributed to the conception and design of study, acquisition of data, or analysis and/or interpretation of data. SY contributed to data acquisition and analysis. KM and KM contributed to drafting the manuscript or revising the manuscript critically for important intellectual content. All authors approved the version of the manuscript to be published.

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Availability of data and materials
The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The study complies with the ethical guidelines of the 1975 Declaration of Helsinki, was approved by the appropriate institutional review committee (authorization No. 448). The board waived the need for patient consent, since retrospective data originated from standard care (in which one can opt-out for the use of their data for scientific research).

Consent for publication
The authors consent to publication of the entire text of this dissertation.

Competing interests
The authors declare that they have no competing interest.

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