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ORIGINAL ARTICLE

Surgical outcomes of acute type A aortic dissection in dialysis patients

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Abstract

Background Acute type A aortic dissection (ATAAD) is relatively uncommon in dialysis patients, and characteristics and repair outcomes are not fully understood.

Patients and methods Patients with ATAAD ($n=960$) were divided into a dialysis group ($n=19$) and non-dialysis group ($n=941$), depending on whether they required dialysis for preoperative end-stage renal disease (ESRD). Hospital charts and imaging data were reviewed, and characteristics and outcomes were compared between the groups. Segmental aortic wall or intima/media flap calcification in the thoracic and abdominal aorta was assessed in the dialysis patients.

Results The leading primary causes of ESRD were polycystic kidney disease ($n=5$) and chronic glomerulonephritis ($n=5$). There were no significant differences (dialysis group vs. non-dialysis group) in age (60.5 vs. 64.5 years), preoperative hemodynamics, or organ ischemia. Dialysis patients were more likely to have an entry tear in the aortic arch (42% vs. 15%, $p=0.003$). These patients showed moderate-to-severe calcification (multiple focal or single focal calcification > 10 mm) in the ascending aorta (17%), aortic arch (61%), descending aorta (67%), and abdominal aorta (83%). Arch replacement was common in this group (37% vs. 18%, $p=0.030$). Although in-hospital mortality was increased in this group (21% vs. 7%, $p=0.059$), morbidities did not differ significantly. Six-year survival was 60.3 ± 13.4% and 78.8 ± 1.6%, respectively ($p=0.01$).

Conclusions Dialysis patients tend to have aortic calcification and a primary tear in the aortic arch. Outcomes are acceptable.

Keywords Acute aortic dissection · End-stage renal disease · Dialysis · Polycystic kidney disease

Introduction

With dialysis being an established treatment for patients with chronic kidney disease (CKD), including those with end-stage renal disease (ESRD), the number of patients on dialysis is increasing in Japan. As of 2014, according to the Japanese Society for Dialysis Therapy registry, the number of incident dialysis patients was 38,327, and the number of prevalent dialysis patients was 320,448 [1]. We in the field of cardiovascular surgery are interested in the CKD population because we see an increasing number of patients on dialysis, and we understand the major causes of death among these particular patients to be heart failure, infection, malignancy, and cerebrovascular disorders [2]. Aortic disease, a cardiovascular disorder, also occurs in dialysis patients, though not commonly, and although investigators have shown an etiologic relation between thoracic aortic disease and cystic kidney disease [3–6], the factors contributing to acute aortic dissection particularly in patients with ESRD are not well understood.

Cardiac surgery carries a high operative risk in dialysis patients [7, 8]. Recently obtained data from the US Renal Data System showed outcomes of both open and endovascular repair of thoracic aortic disease in patients with ESRD to be poor [9]. Although outcomes of treatment for acute type A aortic dissection (ATAAD) have been improving in Japan [10, 11], emergent aortic repair in ESRD patients with ATAAD is considered a surgical challenge requiring

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careful perioperative management. We therefore conducted a retrospective study in which we investigated the clinical characteristics and surgical outcomes of ATAAD in patients with ESRD on dialysis.

Methods

Patients

This was a two-center retrospective study that included 960 patients who underwent emergent aortic repair for ATAAD at either Jichi Medical University Hospital (Shimotsuke, Japan) or Saitama Medical Center, Jichi Medical University (Saitama, Japan). Both hospitals have an aortic database, with that of Jichi Medical University Hospital comprising patients treated between 2000 and 2015 and that of Saitama Medical Center comprising patients treated between 2000 and 2017. The ATAAD in these patients was diagnosed on the basis of enhanced computed tomography (CT) or echocardiography findings, and the dissection was judged to be acute if symptom onset was within 14 days of the patient's hospital admission. Of the 960 patients, 19 were dialysis patients, i.e., they had been undergoing hemodialysis or peritoneal dialysis therapy for ERSD before the onset of aortic dissection, and none had undergone renal transplantation before onset of the ATAAD.

To answer our study question, we divided the 960 patients into two groups according to whether they had been undergoing dialysis therapy (dialysis group, $n=19$; non-dialysis group, $n=941$) preoperatively. We reviewed these patients' medical records, imaging records, and the aortic databases so that we could compare the following between the two groups: patient and dissection characteristics, operative procedures, and early outcomes (in-hospital mortality and morbidity). Late survival was also compared between the groups. The institutional review board approved this study (Ref. No. 17–040) and waived the requirement for informed consent because of the retrospective nature of the study.

Division of patients into subgroups and assessment of aortic calcification

For the purpose of analysis, we divided the total patients into subgroups according to the CKD stage. CKD stage was based on the patient's estimated glomerular filtration rate (eGFR), which was calculated from the preoperative creatinine level. Stages and the respective number of patients were as follows: normal-mildly impaired renal function [eGFR > 60 mL/min/1.73 m^2 ($n=507$)], moderately impaired renal function [30–59 mL/min/1.73 m^2 ($n=377$)], severely impaired renal function [< 30 mL/min/1.73 m^2 ($n=57$)], and dialysis-dependent ESRD ($n=19$) [12]. We next eliminated patients aged 39 years or less and those aged 80 years or more and patients with preoperative renal ischemia, leaving us with the following CKD stage-based groups: normal-mildly impaired renal function ($n=472$), moderately impaired renal function ($n=333$), severely impaired renal function ($n=48$), and dialysis-dependent ESRD ($n=19$). We then performed stratified sampling by sex and age, with proportional allocation, which yielded, for our study, a total 143 patients in the following CKD stage-based groups: normal-mildly impaired renal function ($n=50$), moderately impaired renal function ($n=50$), severely impaired renal function ($n=24$), and dialysis-dependent ESRD ($n=19$). Baseline characteristics (i.e., preoperative characteristics) of these 143 patients are shown per CKD stage-based group in Supplementary Table 1. Although there was a significant difference in age, sex ratio, and the prevalence of hypertension between these 4 CKD stage-based groups, the prevalence of Marfan syndrome was similar.

Aortic wall or intima/media flap calcification in the ascending aorta, aortic arch, descending thoracic aorta, and abdominal aorta was examined in relation to the CKD stages. Calcification was classified by degree on the basis of the preoperative CT imaging data as follows: stage 1, no calcification; stage 2, mild calcification (a focal calcification ≤ 10 mm in diameter), or stage 3, moderate-to-severe calcification (multiple focal calcifications or a focal calcification > 10 mm in diameter).

Operative procedures and postoperative care

Operative procedures were as reported previously [13–15], with the basic surgical strategy for dialysis patients being similar to that for non-dialysis patients. All surgeries were performed via median sternotomy. The femoral artery, axillary artery, left ventricular apex, or ascending aorta was used for arterial cannulation, with cannulation performed at multiple sites in some patients. When the axillary artery was used for cannulation in dialysis patients, the side contralateral to the blood access site for hemodialysis was used preferentially. Cardiopulmonary bypass was then established, and systemic cooling to a target rectal or bladder temperature of 20–25 °C was performed. Proximal repair involved aortic valve resuspension, modified Bentall-type aortic root replacement, valve-sparing aortic root replacement, or isolated aortic valve replacement. Aortic root surgery was performed for patients with aortic root dilatation or an intimal tear at the root. Basically, the distal extent of aortic resection was determined by the location of the primary entry tear, aortic diameter, and status of the false lumen in the downstream aorta. Distal repair involved ascending aorta replacement, hemiarch replacement, total or partial (1 or 2 branches) arch replacement, or total arch replacement with open aortic stent insertion. Selective antegrade cerebral perfusion was performed in cases of arch replacement. Reinforcement with Teflon felt was performed at the aortic stumps, and no gelatin–resorcin–formalin adhesive was used.

After the operation, all patients were cared for in the intensive care unit (ICU). Continuous hemodiafiltration was performed for the dialysis patients and for the non-dialysis patients who suffered severe acute kidney injury during their ICU stay. Upon the resumption of normal hemodialysis, patients who required renal replacement therapy were transferred to the surgical ward.

Statistical analysis

Data are shown as the number (percentage) of patients, as median values (interquartile range), or as mean ± standard deviation, as appropriate. Differences between the dialysis group and non-dialysis group in continuous perioperative variables were analyzed by unpaired *t* test or Mann–Whitney *U* test (for variables with skewed distribution), and differences in categorical variables were analyzed by Chi square test or Fisher's exact test (when the expected number of observations in any cell of the contingency table was less than 5). Differences between the 4 CKD stage-based groups in continuous or categorical perioperative variables were analyzed by Kruskal–Wallis test or by Chi square or Fisher's exact test when appropriate. Freedom from death was estimated by the Kaplan–Meier method, and the difference between groups was analyzed by log-rank test. The stratified sampling was performed with SAS 9.4 (SAS Institute Inc., Cary NC, USA), and all other statistical analyses were performed with SPSS 23.0 for Windows (IBM Corp., Armonk, NY, USA), with $p < 0.05$ considered statistically significant.

Results

Patient characteristics

Patients' preoperative status and anatomical characteristics of the ATAAD are shown for both the dialysis group and the non-dialysis group in Table 1. Mean age in the dialysis group was 60.5 years, close to that of 64.5 years in the non-dialysis group. The dialysis patients were more likely than non-dialysis patients to have hypertension. There were no current smokers in the dialysis group. Three patients (16%) in the dialysis group required preoperative cardiopulmonary resuscitation for profound shock caused by cardiac tamponade ($n = 2$) or severe aortic insufficiency ($n = 1$). There was no significant between-group difference in preoperative hemodynamic measurements or organ malperfusion status. DeBakey type II dissection was most prevalent in the dialysis group; however, the difference was not significant. Patients in the dialysis group were more likely than those in the non-dialysis group to have the primary entry tear in the aortic arch.

Characteristics of the renal disease, including treatment, in the dialysis group patients are shown in Table 2. The leading primary diseases causing ESRD were polycystic kidney disease and chronic glomerulonephritis, followed by diabetes and nephrosclerosis. The disease underlying ESRD was uncertain in five patients. The mean duration of dialysis therapy in the total 19 patients before the onset of ATAAD was 7.2 years, with 95% of these patients undergoing hemodialysis (rather than peritoneal dialysis).

Sites of aortic calcification in the dialysis group are shown per calcification stage in Table 3. Preoperative CT imaging data were missing for 1 patient; thus, aortic calcification was assessed in the remaining 18 patients. Calcification of the ascending aorta was relatively uncommon in these patients, with no calcification seen in two-thirds of the patients. The prevalence of moderate-to-severe aortic calcification was, to the contrary, quite high in the downstream aorta: aortic arch, 61% (11/18); descending thoracic aorta, 67% (12/18); and abdominal aorta, 83% (15/18).

CKD stages are shown in relation to calcification stages per aortic segment in Fig. 1. CKD stages varied in relation to calcification stages, depending on the site of calcification, with significant differences seen when calcification was found in the aortic arch, descending aorta, and abdominal aorta. Stages of calcification of the aortic arch, descending aorta, and abdominal aorta were similar between patients with moderately impaired renal function and those with normal-mildly impaired renal function. To the contrary, calcification of the aortic arch, descending aorta, and abdominal (but not ascending) aorta was advanced in patients with severely impaired renal function and in those requiring dialysis. Representative CT images showing calcification in a dialysis patient are presented in Fig. 2. The patient was a 75-year-old man with ESRD attributed to nephrosclerosis and who had been undergoing hemodialysis for 18 months. A small focal calcification was seen in this patient's ascending aorta (Fig. 2b), and multiple calcifications were depicted in the aortic arch and abdominal aorta (Fig. 2a, c).

Operative variables and outcomes of the surgery

Operative variables are shown in Table 4. Arterial cannulation sites did not differ significantly between the two study groups. The axillary artery on the side opposite the blood access site was cannulated in all patients in the dialysis group. Patients in the dialysis group were more likely than patients in the non-dialysis group to have undergone aortic arch replacement. Of the nine dialysis patients whose entry tear was located in the aortic arch, eight underwent partial

Table 1 Patient characteristics, preoperative status, and dissection details among the total dialysis and non-dialysis patients

	Dialysis $n = 19$	Non-dialysis $n = 941$	p Value
Age (years)	60.5 ± 9.6	64.5 ± 12.8	0.18
Male sex	12 (63%)	542 (52%)	0.34
Marfan syndrome	0 (0%)	30 (3%)	0.96
Obesity (BMI > 30 kg/m^2)	0 (0%)	70 (7%)	0.48
Current smoking	0 (0%)	339 (33%)	< 0.01
Comorbidity			
Hypertension	18 (95%)	743 (71%)	0.048
Diabetes mellitus	2 (11%)	68 (7%)	0.82
History of cerebrovascular disease	2 (11%)	86 (8%)	1.0
Chronic obstructive pulmonary disease	1 (5%)	26 (3%)	0.98
Preoperative status			
Shock (systolic BP < 80 mmHg)	6 (31%)	244 (24%)	0.41
Preoperative CPR	3 (16%)	46 (4%)	0.074
Severe aortic insufficiency	3 (16%)	145 (14%)	1.0
Organ ischemia			
Brain	2 (11%)	101 (10%)	1.0
Coronary	2 (11%)	79 (8%)	1.0
Visceral	0 (0%)	39 (4%)	0.49
Kidney	0 (0%)	92 (9%)	0.34
Lower limb	3 (16%)	128 (12%)	0.92
DeBakey classification			
Type I	14 (74%)	926 (89%)	0.083
Type II	5 (26%)	114 (11%)	0.083
Location of the entry tear[a]			
Ascending aorta	9 (47%)	599 (64%)	0.14
Aortic arch	8 (42%)	139 (15%)	0.003
Descending aorta or unidentified	2 (11%)	210 (22%)	0.34

Mean ± standard deviation values or number (percentage) of patients are shown

BMI body mass index, *BP* blood pressure, *CPR* cardiopulmonary resuscitation

[a]Primary entry tear includes multiple entry tears in some patients

Table 2 Characteristics of the renal disease in the dialysis patients ($n = 19$)

Primary cause of the end-stage renal disease	
Polycystic kidney disease	5 (26%)
Chronic glomerulonephritis	5 (26%)
Diabetes	2 (11%)
Nephrosclerosis	2 (11%)
Non-diabetic unknown causes	5 (26%)
Type of dialysis	
Hemodialysis	18 (95%)
Peritoneal	1 (5%)
Type of blood access	
Upper limb hemodialysis shunt	17 (89%)
Superficialization of the brachial artery	1 (5%)
Duration of dialysis (years)	7.2 ± 7.6

Mean ± SD values or number (percentage) of patients are shown

Table 3 Sites of aortic calcification per calcification stage in the dialysis patients

	Stage 1 No calcification	Stage 2 Mild calcification	Stage 3 Moderate-severe calcification
Ascending aorta	67% (12/18)	17% (3/18)	17% (3/18)
Aortic arch	22% (4/18)	17% (3/18)	61% (11/18)
Descending aorta	22% (4/18)	11% (2/18)	67% (12/18)
Abdominal aorta	6% (1/18)	11% (2/18)	83% (15/18)

Percentage (ratio) of patients is shown. Stage 2 (mild calcification) is defined as a focal calcification with a diameter of ≤ 10 mm, and stage 3 (moderate-to-severe calcification) is defined as multiple focal calcifications or a focal calcification with a diameter > 10 mm

or total arch replacement, and the remaining patients underwent hemiarch replacement. The primary entry tear was

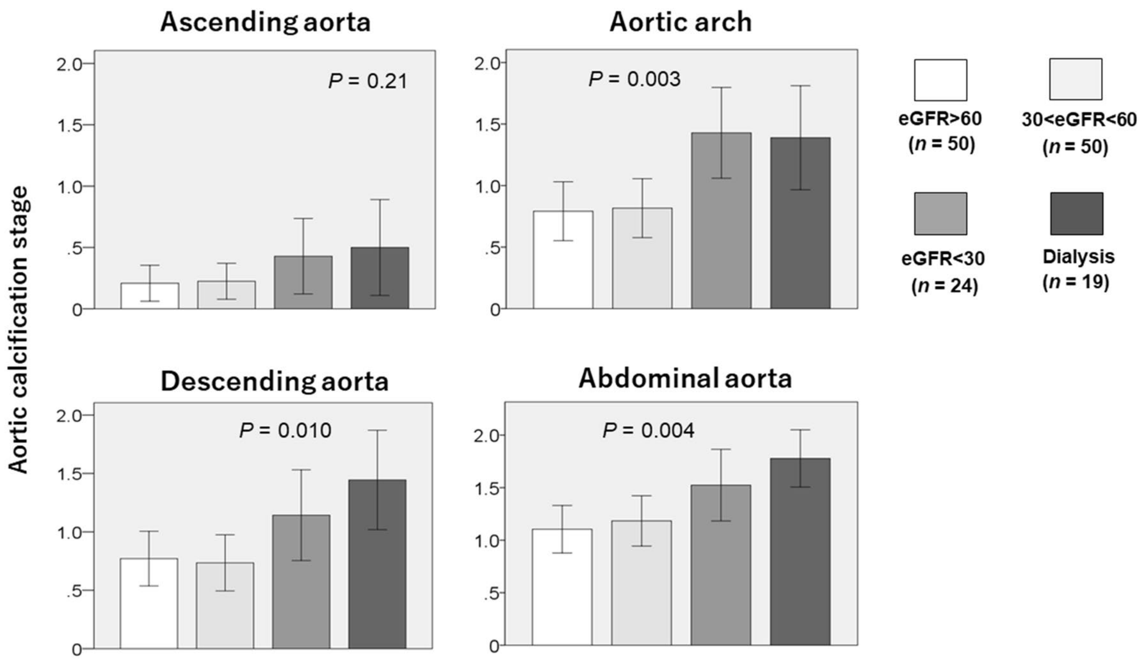

Fig. 1 Bar graphs showing CKD stages in relation to stages of aortic calcification, per the various aortic segments examined. CKD stages were determined on the basis of patients' preoperative eGFR and were as follows: normal-mildly impaired renal function (eGFR > 60 mL/min/1.73 m^2), moderately impaired renal function: (30–59 mL/min/1.73 m^2), severely impaired renal function: (< 30 mL/min/1.73 m^2), and dialysis-dependent ESRD. Stages of segmental aortic calcification were as follows: stage 1, no calcification; stage 2, mild calcification (focal calcification ≤ 10 mm in diameter); and stage 3, moderate-to-severe calcification (multiple focal calcifications or a focal calcification > 10 mm in diameter). Probability (*p*) values were obtained by Kruskal–Wallis test. *CKD* chronic kidney disease, *eGFR* estimated glomerular filtration rate

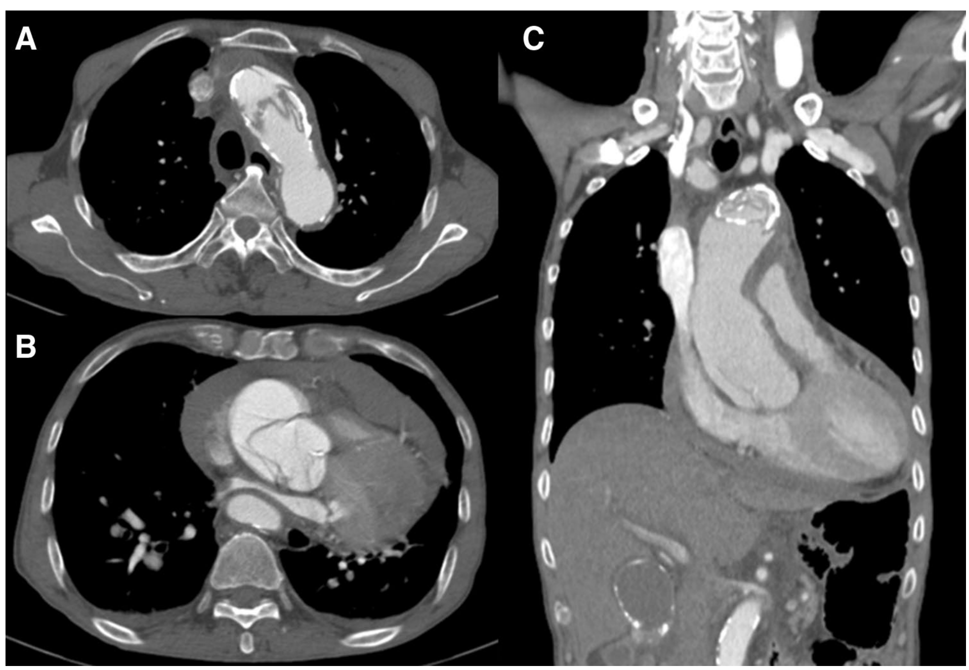

Fig. 2 Computed tomography images of a 75-year-old male dialysis patient with acute type A aortic dissection. The axial slices show calcifications throughout the aortic arch (**a**) and a small focal calcification in the ascending aorta (**b**). The coronal reconstruction shows widespread calcification in the aortic arch and abdominal aorta (**c**)

resected in all nine patients. Proximal reconstruction techniques, cardiopulmonary bypass time, and operation time did not differ significantly between the two study groups.

Early outcomes are shown per group in Table 5. Four patients in the dialysis group died during the hospitalization period. Although the in-hospital mortality rate was increased

Table 4 Operative variables in the dialysis and non-dialysis patients

	Dialysis $n=19$	Non-dialysis $n=941$	*p* Value
Arterial cannulation site			
Axillary artery	7 (37%)	456 (49%)	0.32
Ipsilateral side of blood access site	0 (0%)	NA	NA
Contralateral side of blood access site	7 (37%)	NA	NA
Femoral artery	7 (37%)	457 (49%)	0.31
Left ventricular apex	5 (26%)	197 (21%)	0.57
Ascending aorta	1 (5%)	9 (1%)	0.49
Proximal reconstruction			
Commissure resuspension	19 (100%)	859 (91%)	0.35
Modified Bentall procedure	0 (0%)	48 (5%)	0.63
Isolated aortic valve replacement	0 (0%)	31 (3%)	0.88
Valve-sparing aortic root surgery	0 (0%)	3 (0.3%)	1.0
Distal extent of aortic resection			
Ascending aorta or hemiarch replacement	12 (63%)	776 (83%)	0.030
Total or partial arch replacement	7 (37%)	165 (18%)	0.030
Resection of the entry tear	17 (89%)	727 (77%)	0.38
Coronary artery bypass grafting	1 (5%)	59 (6%)	1.0
Cardiopulmonary bypass time (min)	186 ± 77	175 ± 76	0.54
Operation time (min)	371 ± 101	375 ± 126	0.88

Mean ± standard deviation values or number (percentage) of patients are shown

NA not applicable

Table 5 Early outcomes among the dialysis and non-dialysis patients

	Dialysis $n=19$	Non-dialysis $n=941$	*p* value
30-day mortality	16% (3/19)	6% (54/941)	0.18
In-hospital mortality	21% (4/19)	7% (66/941)	0.059
Length of ICU stay (days)	7 (4–11)	6 (4–9)	0.59
Length of hospital stay (days)	25 (15–34)	22 (16–30)	0.54
Resumption of hemodialysis (postoperative days)	7 (4–11)	NA	NA
Complications			
Heart failure requiring PCPS	0 (0%)	27 (3%)	0.96
Re-exploration for bleeding	0 (0%)	33 (4%)	0.85
New-onset neurological deficit	2 (11%)	66 (7%)	0.89
Tracheostomy	1 (5%)	44 (5%)	1.0
Deep sternal wound infection	0 (0%)	13 (1%)	1.0
Obstruction of blood access site	0 (0%)	NA	NA

Median (interquartile range) or number (percentage) of patients are shown

ICU intensive care unit, *NA* not applicable, *PCPS* percutaneous cardiopulmonary support

in this group, the difference was not statistically significant. The in-hospital deaths were due to cerebral infarction ($n=2$), sepsis ($n=1$), and multi-organ failure ($n=1$). One of the two patients who died of cerebral infarction had suffered cardiac tamponade and undergone CPR preoperatively. The median ICU and hospital stays did not differ significantly between in-hospital survivors in the dialysis group and those in the non-dialysis group. Similarly, complication rates, including those for re-exploration for bleeding and new-onset neurological deficit, did not differ significantly between the two groups. The median duration of postoperative continuous hemodiafiltration was 6 days, and there was no obstruction of the hemodialysis access site.

Survival is shown in Fig. 3. The 6-year survival rate for the dialysis group was 60.3 ± 13.4%, and that for the non-dialysis group was 8.8 ± 1.6% ($p=0.01$). Two patients died during follow-up, one due to pneumonia 6 months after surgery and the other to cerebral bleeding 21 months after surgery.

Discussion

Aortic dissection is somewhat rare in dialysis patients. In two separately reported series of patients who underwent surgery for ATAAD, dialysis patients accounted for only 1.8% and 2.6% [16, 17] of the total patients, percentages that were similar to ours (2.0%) (19/960). Although preoperative renal dysfunction has been reported to increase the operative risk of aortic repair for patients with ATAAD [18, 19], the effect of ESRD on outcomes of ATAAD has not been

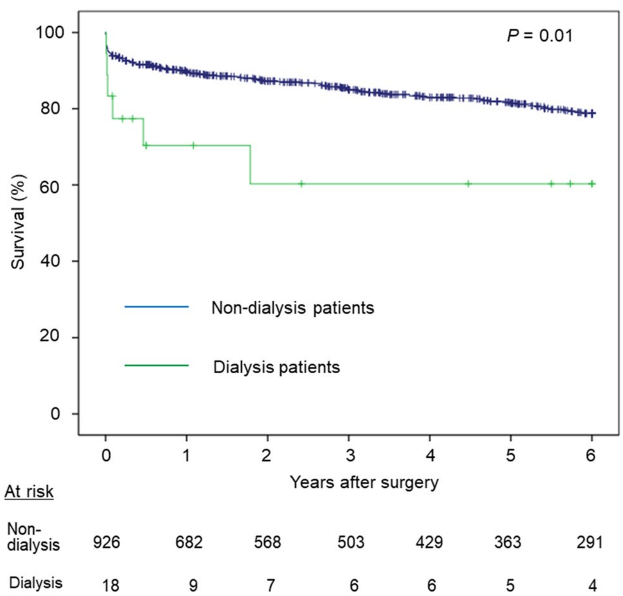

Fig. 3 Kaplan–Meier curves for late survival of dialysis and non-dialysis patients with acute type A aortic dissection. Probability (*p*) values were obtained by log-rank test

elucidated. To the best of our knowledge, the study we report herein is the first to investigate patient and disease characteristics and outcomes of emergent surgery for ATAAD in dialysis patients. We showed no difference in patients' preoperative status, including organ malperfusion and hemodynamics, between dialysis patients and non-dialysis patients. Key findings in the dialysis patients included the presence of moderate-to-severe aortic wall calcification in the aortic arch and the downstream aorta and the increased likelihood that the primary entry tear would be located in the aortic arch.

CKD is known to increase the operative risk of cardiovascular surgery. The operative risks for ESRD patients who are on dialysis are increased in comparison to those for CKD patients not on dialysis. Yamauchi et al. reported from the Japan Adult Cardiovascular Database that the operative mortality and mortality including major morbidities after coronary artery bypass grafting were 4.8% and 23.1%, respectively, for dialysis patients ($n = 1300$) and significantly higher than the respective 1.4% and 13.7% for non-dialysis patients ($n = 18{,}387$) [20]. On the basis of data obtained from the United States Renal Data System, Liang et al. reported unfavorable outcomes for open and endovascular descending thoracic aortic repair in patients with ESRD, with 30-day mortality rates of 30.1% (41/136) for open repair and 15.7% (34/216) for endovascular repair [9]. Surgical outcomes of ascending aorta replacement or aortic arch replacement in patients with ESRD have not been well investigated. Okada et al. identified severe renal dysfunction, defined as a preoperative eGFR < 30 mL/min/1.73 m^2, as an independent risk factor for in-hospital death in non-dialysis patients who underwent elective total arch replacement [21]. In-hospital mortality was considerably higher in our dialysis group than in our non-dialysis group. However, considering that 3 of the 19 patients required CPR preoperatively, we consider the in-hospital mortality rate of 21% acceptable.

Multiple focal or diffuse aortic wall or intima/media flap calcifications were seen in our dialysis patients: in the ascending aorta in 17% (3/18) of the patients, in the aortic arch in 61% (11/18), in the descending thoracic aorta in 67% (12/18), and in the abdominal aorta in 83% (15/18). de Jong et al. investigated aortic wall calcification in 69 patients aged 60 years or older with acute or chronic aortic dissection. They included 37 patients with type A dissection and 15 with renal dysfunction defined as eGFR < 60 mL/min/1.73 m^2. The overall incidence of aortic wall or intima/media flap calcification was 32% (22/69) [22], which was lower than the incidence documented for the dialysis patients in our study. Our study also showed that dialysis patients tend to have a calcified aortic arch. Meticulous operative techniques are important, especially for aortic arch replacement in dialysis patients. We try to create the distal anastomosis at the site of less severe calcification. Careful preoperative evaluation is essential. Application of arch replacement with frozen elephant technique [23] may decrease the risk of bleeding in patients with aortic calcification.

A high incidence of peripheral artery disease is another characteristic of patients with ESRD. In the present patient series, we preferably used the axillary artery on the contralateral side of the blood access site when axillary artery cannulation was required. More important, however, is the presence or absence of organ malperfusion and the degree of arterial calcification. A patient-specific approach is needed when establishing cardiopulmonary bypass in dialysis patients with ATAAD.

A multi-center registry study from Germany reported that the incidence of primary entry tear located in the aortic arch in patients with ATAAD was 14.6% (314/2137) [24], which was similar to the 15% (139/941) rate in the present non-dialysis patients. In contrast, 42% (8/19) of the dialysis patients had a primary entry tear in the aortic arch. Pathologically, ATAAD in patients aged ≥ 65 years is associated with atherosclerosis, whereas ATAAD in patients younger than 65 years is more likely to be associated with medial degeneration [25]. Penetrating aortic ulcer, a condition in which ulceration of an aortic atherosclerotic plaque penetrates through the internal elastic lamina into the media [26], may be related to the development of aortic dissection in dialysis patients. In an autopsy study of six patients with atherosclerosis-related aortic dissection, Tamura et al. found that the intimal tear was located in the ascending aorta in one patient and the descending aorta in five [27]. Similarly, in an autopsy study of 111 patients with aortic dissection, Nakashima et al. found more severe atherosclerosis in patients with DeBakey type III dissection than in those with DeBakey type I or type II dissection [28]. We found that dialysis patients and non-dialysis patients with severely impaired renal function tend to have calcification in the aortic arch and the descending aorta. Although the intimal tear was in the descending aorta in only two dialysis patients in this series, we hypothesized that dialysis patients are likely to have an intimal tear in the atherosclerotic aortic arch and descending aorta. A recent fluid dynamics simulation study reported that areas of high aortic wall stress are susceptible to the formation of an intimal tear [29]. Additional fluid dynamic simulation studies will be needed to elucidate the underlying mechanisms in detail.

The 2014 Japanese Society for Dialysis Therapy registry data showed that the three major primary diseases for prevalent dialysis in Japan were diabetes (38.1%), chronic glomerulonephritis (31.3%), and nephrosclerosis (9.1%), followed by polycystic kidney disease (3.5%), rapidly progressive glomerulonephritis (1.0%), chronic pyelonephritis (0.8%), lupus (0.7%), and undetermined (8.9%) [1]. Although the proportion of polycystic kidney disease in the dialysis population is small, the present data showed that it was the most common primary disease causing ESRD, along with chronic glomerulonephritis, in the patients with ATAAD. Autosomal-dominant polycystic kidney disease (ADPKD) is the most common hereditary kidney disease, affecting about one in 400–1000 individuals [4]. A recent nationwide study from Taiwan showed that patients with ADPKD ($n = 2076$) had a significantly increased incidence of aortic aneurysm or dissection compared to an age- and sex-matched non-ADPKD population ($n = 20{,}760$) (0.92% vs. 0.11%, $p < 0.0001$). Alterations in TGF-β (transforming growth factor-β) signaling caused by mutation in *PKD1* or *PKD2* are reported to contribute to aneurysm formation [4]. In patients with ADPKD, dissections and aneurysm formations can occur in systemic large arteries. Of note, the frequency of intracranial aneurysm in patients with ADPKD ranges between 9 and 12%, which is higher than the 2–3% frequency in the general population [4]. Postoperative imaging studies to check for intracranial aneurysm would be preferable in patients with polycystic kidney disease.

There are several limitations in this study. First, the study was retrospective, and the number of dialysis patients was relatively small. In the future, a larger-scale, multi-center study is needed to validate our findings. Second, the primary cause of ESRD was uncertain in 26% (5/19) of the dialysis patients. Third, we did not conduct genetic testing in patients with polycystic kidney disease to confirm the gene mutation specific for ADPKD.

Conclusion

ESRD requiring dialysis is uncommon in patients with ATAAD. In the dialysis patients with ATAAD, the most common primary causes of ESRD were polycystic kidney disease and chronic glomerulonephritis and, less commonly, diabetes and nephrosclerosis. Dialysis patients showed moderate-to-severe aortic wall calcification, and the primary entry tear was more likely to be located in the aortic arch. With a similar surgical approach to that used in non-dialysis patients, acceptable outcomes can be expected even in dialysis patients. Careful and meticulous operative techniques are important, especially when performing aortic arch replacement.

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Compliance with ethical standards

Conflict of interest Kei Akiyoshi and the other authors have no conflict of interest.

References

1. Masakane I, Nakai S, Ogata S, Kimata N, Hanafusa N, Hamano T, Wakai K, Wada A, Nitta K. Annual dialysis data report 2014, JSDT renal data registry (JRDR). Ren Replace Ther. 2017;3:18.
2. Hanafusa N, Nakai S, Iseki K, Tsubakihara Y. Japanese society for dialysis therapy renal data registry—a window through which we can view the details of Japanese dialysis population. Kidney Int Suppl (2011). 2015;5:15–22.
3. Ziganshi BA, Theodoropoulos P, Salloum MN, Zaza KJ, Tranquilli M, Mojibian HR et al. Simple renal cysts as markers of thoracic aortic disease. J Am Heart Assoc. 2016;5:e002248.
4. Perrone RD, Malek AM, Watnick T. Vascular complications in autosomal dominant polycystic kidney disease. Nat Rev Nephrol. 2015;11:589–98.
5. Fukunaga N, Yuzaki M, Nasu M, Okada Y. Dissecting aneurysm in a patient with autosomal dominant polycystic kidney disease. Ann Thorac Cardiovasc Surg. 2012;18:375–8.
6. Sung PH, Yang YH, Chiang HJ, Chiang JY, Chen CJ, Liu CT, et al. Risk of aortic aneurysm and dissection in patients with autosomal-dominant polycystic kidney disease: a nationwide population-based cohort study. Oncotarget. 2017;8:57594–604.
7. Rahmanian PB, Adams DH, Castillo JG, Vassalotti J, Filsoufi F. Early and late outcome of cardiac surgery in dialysis-dependent patients: single-center experience with 245 consecutive patients. J Thorac Cardiovasc Surg. 2008;135:915–22.
8. Hori D, Yamaguchi A, Adachi H. Coronary artery bypass surgery in end-stage renal disease patients. Ann Vasc Dis. 2017;10:79–87.
9. Liang NL, Yuo TH, Al-Khoury GE, Hager ES, Makaroun MS, Singh MJ. High mortality rates after both open surgical and

endovascular thoracic aortic interventions in patients with end-stage renal disease. J Vasc Surg. 2017;66:991–6.
10. Committee for Scientific Affairs The Japanese Association for Thoracic Surgery, Masuda M, Okumura M, Doki Y, Endo S, Hirata Y, et al. Thoracic and cardiovascular surgery in Japan during 2014: annual report by the japanese association for thoracic surgery. Gen Thorac Cardiovasc Surg. 2016;64:665–697.
11. Okita Y. Current surgical results of acute type A aortic dissection in Japan. Ann Cardiothorac Surg. 2016;5:368–76.
12. Dhanani J, Mullany DV, Fraser JF. Effect of preoperative renal function on long-term survival after cardiac surgery. J Thorac Cardiovasc Surg. 2013;146:90–5.
13. Itagaki R, Kimura N, Mieno M, Hori D, Itoh S, Akiyoshi K, et al. Characteristics and treatment outcomes of acute type A aortic dissection with elevated d-dimer concentration. J Am Heart Assoc. 2018;7:e009144.
14. Sasabuchi Y, Kimura N, Shiotsuka J, Komuro T, Mouri H, Ohnuma T, et al. Long-term survival in patients with acute kidney injury after acute type A aortic dissection repair. Ann Thorac Surg. 2016;102:2003–9.
15. Aizawa K, Kawahito K, Misawa Y. Long-term outcomes of tear-oriented ascending/hemiarch replacements for acute type A aortic dissection. Gen Thorac Cardiovasc Surg. 2016;64:403–8.
16. Lawton JS, Liu J, Kulshrestha K, Moon MR, Damiano RJ Jr, Maniar H, et al. The impact of surgical strategy on survival after repair of type A aortic dissection. J Thorac Cardiovasc Surg. 2015;150:294 e291–301 e291.
17. Etz CD, von Aspern K, da Rocha ESJ, Girrbach FF, Leontyev S, Luehr M, et al. Impact of perfusion strategy on outcome after repair for acute type a aortic dissection. Ann Thorac Surg. 2014;97:78–85.
18. Mehta RH, Suzuki T, Hagan PG, Bossone E, Gilon D, Llovet A, et al. Predicting death in patients with acute type A aortic dissection. Circulation. 2002;105:200–6.
19. Goda M, Imoto K, Suzuki S, Uchida K, Yanagi H, Yasuda S, et al. Risk analysis for hospital mortality in patients with acute type a aortic dissection. Ann Thorac Surg. 2010;90:1246–50.
20. Yamauchi T, Miyata H, Sakaguchi T, Miyagawa S, Yoshikawa Y, Takeda K, et al. Coronary artery bypass grafting in hemodialysis-dependent patients. Circ J. 2012;76:1115–20.
21. Okada K, Omura A, Kano H, Nakai H, Miyahara S, Minami H, et al. Outcome of elective total aortic arch replacement in patients with non-dialysis-dependent renal insufficiency stratified by estimated glomerular filtration rate. J Thorac Cardiovasc Surg. 2014;147:966 e962–72 e962.
22. de Jong PA, Hellings WE, Takx RA, Isgum I, van Herwaarden JA, Mali WP. Computed tomography of aortic wall calcifications in aortic dissection patients. PLoS One. 2014;9:e102036.
23. Uchida N, Katayama A, Tamura K, Sutoh M, Kuraoka M, Ishihara H. Frozen elephant trunk technique and partial remodeling for acute type A aortic dissection. Eur J Cardiothorac Surg. 2011;40:1066–71.
24. Conzelmann LO, Hoffmann I, Blettner M, Kallenbach K, Karck M, Dapunt O, et al. GERAADA Investigators. Analysis of risk factors for neurological dysfunction in patients with acute. aortic dissection type A: data from the German Registry for Acute Aortic Dissection type A (GERAADA). Eur J Cardiothorac Surg. 2012;42:557–65.
25. Juraszek A, Bayer G, Dziodzio T, Kral A, Laufer G, Ehrlich M. Evaluation of the intraoperative specimens of the thoracic and abdominal aorta. J Cardiothorac Surg. 2013;8:110.
26. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). Eur Heart J. 2014;35:2873–926.
27. Tamura K, Sugisaki Y, Kumazaki T, Tanaka S. Atherosclerosis-related aortic dissection. Kyobu Geka. 2000;53:194–201.
28. Nakashima Y, Kurozumi T, Sueishi K, Tanaka K. Dissecting aneurysm: a clinicopathologic and histopathologic study of 111 autopsied cases. Hum Pathol. 1990;21:291–6.
29. Plonek T, Zak M, Rylski B, Berezowski M, Czerny M, Beyersdorf F, et al. Wall stress correlates with intimal entry tear localization in type A aortic dissection. Interact Cardiovasc Thorac Surg. 2018;27:797–801.