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ORIGINAL ARTICLE

Acute renal injury after thoracic endovascular aortic repair of Stanford type B aortic dissection: Incidence, risk factors, and prognosis

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KEYWORDS
acute renal injury;
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Background/Purpose: Acute kidney injury (AKI) significantly increases the risk of mortality in patients following cardiovascular intervention procedures. This study was carried out to investigate the incidence, predictors, and prognostic implications of AKI after thoracic endovascular aortic repair (TEVAR) of Stanford type B aortic dissection.

Methods: A total of 156 patients with Stanford type B aortic dissection who underwent TEVAR were retrospectively analyzed between February 1, 2004 and October 31, 2011. Multivariable regression was used to predict risk factors for AKI. Association between baseline characteristics, postoperative AKI, and mortality during follow up was evaluated.

Results: AKI was identified in 48 (30.8%) of 156 patients, with seven (14.5%) patients requiring continuous renal replacement therapy. The in-hospital mortality rate was 0% in patients without AKI and 12.5% in those with AKI ($p = 0.001$). Univariate analysis identified preoperative chronic kidney disease, acute dissection, complicated dissection, malperfusion complications with comprehensive complications, and postoperative minimum estimated glomerular filtration rate within 48 hours as associated with AKI. Malperfusion complications [odds ratio (OR) = 4.828; 95% confidence interval (CI) = 1.163–20.03] were the only independent predictor of AKI. Patients suffering from AKI had a 14-fold increased risk for 30-day mortality (OR = 14.3; 95% CI = 1.7–118.4; $p = 0.014$) and a 10-fold increased risk for 1-year mortality (OR = 9.5; 95% CI = 2.02–44.9; $p = 0.004$).

Conflicts of interest: The authors have no conflicts of interest relevant to this article.

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Conclusion: A significant rate of AKI was observed following TEVAR and was associated with an increase in 30-day and 1-year mortality. Malperfusion complications were identified as an independent predictor of AKI.

Introduction

Acute kidney injury (AKI) is a common complication after cardiovascular intervention procedures.[1,2] Furthermore, the development of postoperative AKI following elective abdominal aortic aneurysm (AAA) and endovascular aortic repair (EVAR) has been associated with prolonged hospital stays and a significantly increased risk of mortality.[3,4] However, ranges of reported renal dysfunction of patients undergoing thoracic EVAR (TEVAR) were highly variable according to different criteria, varying from 11.9% to 34.0%.[5,6] TEVAR involves the insertion and deployment of a stent graft through the femoral artery. The less invasive nature of endovascular treatment provides better in-hospital survival and a less invasive alternative to open surgery for type B aortic dissection.[7] TEVAR has been increasingly used to treat type B aortic dissection. However, type B dissection differs from AAA and other aortic diseases in many aspects. There is little information focusing on AKI after TEVAR for type B aortic dissection. The clinical risks and implications of acute renal dysfunction following TEVAR for type B aortic dissection have not been well studied.

The objective of this study was to investigate the incidence, predictors, and prognostic implications of AKI following TEVAR in patients with Stanford type B aortic dissection.

Materials and methods

Patient population and procedural details

We retrospectively analyzed a total of 186 patients who underwent thoracic endovascular repair from February 1, 2004 to October 31, 2011 in Nanjing First Hospital affiliated with Nanjing Medical University. Of these, 156 patients with Stanford type B aortic dissection were included in the present analysis. We excluded 30 patients due to the following conditions: (1) stent-graft placement was technically unsuccessful, (2) patients died 24 hours after the procedure, (3) no data on renal function were available, (4) patients were already on dialysis before the procedure, or (5) patients had aneurysms of the descending thoracic aorta, penetrating aortic ulcers, or traumatic aortic transection.

Details of the arterial anatomy evaluation and implantation procedure for these patients have been described previously.[8] The type of anesthesia (local anesthesia with conscious sedation or general anesthesia) was determined by clinical status and patient preference. After graft-stent deployment, contrast aortography was performed to assess outcome.

After the procedure, patients were transferred to the coronary care unit (CCU). Serum creatinine (Cr) and blood urea nitrogen values were measured 24 hours before the procedure, immediately after the procedure, and then once daily during hospitalization. Indications for continuous renal replacement therapy (CRRT) were refractory pulmonary edema or fluid overload, metabolic acidosis, hyperkalemia, uremic symptoms, persistent oliguria (urine output $<$ 200 mL/12 hours) or anuria (urine output $<$ 50 mL/12 hours).

All patients underwent coronary angiography. When at least one main vessel of the epicardial coronary arteries had $>$70% stenosis, percutaneous coronary intervention (PCI) was carried out within 2 weeks after the TEVAR procedure.

Our institutional review board approved the study, and informed consent was obtained from each patient.

Definitions

Aortic dissection was considered an acute event if it occurred within the first 14 days from the onset of symptoms, and as chronic if it occurred after 14 days. The term "complicated dissection" was defined as persistent or unrelenting back pain despite maximal medical therapy, uncontrollable hypertension, aortic enlargement more than 5 mm/year, malperfusion syndromes, or (imminent) rupture.[9]

The baseline glomerular filtration rate (GFR) was calculated with the abbreviated Modification of Diet in Renal Disease formula [estimated GFR (eGFR; mL/minute/1.73 m^2) $=$ 186 $\times$ serum $Cr^{-1.154}$ $\times$ $age^{-0.203}$ $\times$ 0.742, if female $\times$ (1.210 if African American)].[10] Chronic renal failure was defined as a baseline eGFR $\leq$ 60 mL/minute/1.73 m^2.[11]

Patients with AKI were identified by a 25% decrease in eGFR, a $\geq$1.5-fold increase in serum Cr compared with baseline at 48 hours following the procedure (RIFLE classification),[12] or the need for CRRT during index hospitalization.

Malperfusion complications were caused by branch arterial obstruction secondary to the aortic dissection, including ischemia and/or infarction of the celiac trunk or renal, mesenteric, and limb vasculature. They were judged on the basis of clinical or imaging data.

Follow up

Clinical follow up was conducted at 1, 6, and 12 months after discharge, and annually thereafter by telephone or through a clinical visit. Patients were followed for a period from 6 to 101 months (mean: 33.8 $\pm$ 22.5 months). A total of 15 patients (9.6%) were immediately lost to follow up after discharge.

Table 1 Baseline clinical characteristics of patient population ($n = 156$).

Parameter	All	Non-AKI	AKI	p	Number of observations
Total cases	156	108 (69.2%)	48 (30.8%)		156
Clinical background					
Age (y; 30–85)	54.8 ± 11.0	54.8 ± 11.5 ($n = 108$)	53.7 ± 9.9 ($n = 48$)	0.544	156
Male	138 (88.5%)	95 (88.0%)	43 (89.6%)	0.770	156
Hypertension	102 (67.1%)	73 (69.5%)	29 (61.7%)	0.343	152
Hypertension level				0.056	145
0	50 (34.5%)	32 (32.3%)	18 (39.1%)		
1	6 (4.1%)	5 (5.1%)	1 (2.2%)		
2	30 (20.7%)	26 (26.3%)	4 (8.7%)		
3	59 (40.7%)	36 (36.4%)	23 (50.0%)		
Diabetes	11 (7.1%)	8 (7.4%)	3 (6.3%)	1.000	156
Hyperlipidemia	6 (3.8%)	3 (2.8%)	3 (6.3%)	0.555	156
Current smoking	87 (55.8%)	61 (56.5%)	26 (54.2%)	0.788	156
CAD	39 (35.1%)	27 (36.0%)	12 (33.3%)	0.783	111
Previous PCI	3 (1.9%)	2 (1.9%)	1 (2.1%)	1.000	156
Previous MI	0	0	0	—	156
Previous CABG	0	0	0	—	156
Previous stroke	11 (7.1%)	8 (7.4%)	3 (6.3%)	1.000	156
COPD	8 (5.1%)	5 (4.6%)	3 (6.3%)	0.976	156
Clinical presentation					
Preoperative creatinine (μmol/L), median (interquartile range)	82.7 (70.6–110.3)	82.4 (71.0–101.2; $n = 108$)	80.4 (69.1–130.9; $n = 48$)	0.292	156
Preoperative CKD (eGFR $\leq$ 60 mL/minute/1.73 m^2)	35 (22.4%)	19 (17.6%)	16 (33.3%)	0.030	156
Preoperative eGFR (mL/min/1.73 m^2)				0.046	156
>90	72 (46.2%)	50 (46.3%)	22 (45.8%)		
60–89	49 (31.4%)	39 (36.1%)	10 (20.8%)		
30–59	31 (19.9%)	18 (16.7%)	13 (27.1%)		
<29	4 (2.6%)	1 (0.9%)	3 (6.3%)		
Acute dissection	126 (80.8%)	82 (75.9%)	44 (91.7%)	0.021	156
Complicated dissection	43 (27.6%)	24 (22.2%)	19 (39.6%)	0.025	156
Heart rate (beats/min)	77.5 ± 13.2	76.5 ± 12.9 ($n = 106$)	79.7 ± 13.7 ($n = 48$)	0.169	154
Hemoglobin (g/dL)	130.8 ± 17.3	131.8 ± 16.9 ($n = 107$)	128.7 ± 18.2 ($n = 47$)	0.323	154
Ejection fraction (%), median (interquartile range)	64 (60–67)	64 (60.0–67.0) ($n = 92$)	65 (61.0–68.0) ($n = 42$)	0.335	134
AOD (mm)	37.2 ± 4.7	37.0 ± 4.8 ($n = 92$)	37.6 ± 5.6 ($n = 42$)	0.565	134
LVDd (mm), median (interquartile range)	52.0 (49.0–56.0)	51.0 (49.0–56.3) ($n = 92$)	52.0 (49.5–56.0) ($n = 42$)	0.937	134
Medical treatment					
β-Blocker	123 (80.4%)	87 (80.6%)	36 (80.0%)	0.937	152
ACEI/ARB	105 (69.1%)	75 (70.1%)	30 (66.7%)	0.676	152
Calcium antagonist	117 (76.5%)	81 (75.0%)	36 (80%)	0.506	152

Values are median with interquartile range, mean ± standard deviation, or number (%). For hypertension levels, see Mancia G et al.[31]
ACEI = angiotensin-converting enzyme inhibitor; AKI = acute kidney injury; AOD = ascending aorta root diameter; ARB = angiotensin receptor blocker; CABG = coronary artery bypass graft; CAD = coronary artery disease; CKD = chronic kidney disease; COPD = chronic obstructive pulmonary disease; eGFR = estimated glomerular filtration rate; LVDd = left ventricular end diastolic dimension; MI = myocardial infarction; PCI = percutaneous coronary intervention; TEVAR = thoracic endovascular aortic repair.

Statistical analysis

Continuous variables are presented as mean ± standard deviation if normally distributed and as median (interquartile range) if not normally distributed. Categorical variables are given as frequencies and percentages. Continuous variables were tested for differences with the Student *t* test or Mann–Whitney *U* test. Categorical variables were tested for differences with the Chi square test or Fisher exact *t* test. A forward stepwise multivariable logistic regression model was applied to identify predictors of AKI after TEVAR. A Cox proportional hazard model was applied to identify predictors of death after TEVAR. In addition, the Kaplan–Meier method was used to estimate a cumulative survival plot relating AKI to incidence. Survival was compared with the log-rank test. Results are reported as adjusted odds ratio (OR) with 95% confidence interval (CI). A two-sided $p < 0.05$ was considered statistically significant. All analyses were conducted with SPSS Statistics version 16.0.0 (SPSS Inc., Chicago, IL, USA).

Results

Baseline characteristics of study population patients

We retrospectively analyzed a total of 186 patients who underwent thoracic endovascular repair from February 1, 2004 to October 31, 2011. A total of 30 patients were excluded according to the exclusion criteria. A total of 156 patients (average age: 54.8 ± 11.0 years) with Stanford type B aortic dissection were included in the present analysis. Of the 156 patients, 138 (88.5%) were male and 18 (11.5%) were female patients.

Common comorbidities were hypertension (67.1%), diabetes (7.1%), smoking history (55.8%), coronary artery disease (35.1%), previous stroke (7.1%), history of previous percutaneous artery intervention (1.9%), and chronic obstructive pulmonary disease (COPD; 5.1%). These comorbidities are summarized in Table 1. TEVAR was performed in 126 (80.8%) patients during the acute phase and in 30 (19.2%) patients during the chronic phase in the interventional catheterization lab. The underlying disease and baseline characteristics of patients undergoing TEVAR are shown in Table 1. PCI was performed in eight patients with coronary heart disease during the perioperative period.

Incidence of postprocedural AKI

AKI according to the RIFLE classification[12] occurred in 48 patients (30.8%) after TEVAR, seven patients (4.5%) needed CRRT, of which four (2.6%) patients died in the hospital; another three patients survived (one patient was transferred to continue hemodialysis, one patient maintained chronic renal insufficiency, and one patient had complete recovery of renal function).

The AKI incidence was related to preoperative chronic kidney disease (CKD; 33.3% vs. 17.6%, $p = 0.030$), acute aortic dissection (91.7% vs. 75.9%, $p = 0.021$), complicated aortic dissection (22.2% vs. 39.6%, $p = 0.025$), malperfusion complications (18.8% vs. 7.4%, $p = 0.036$), and preoperative eGFR ($p = 0.046$), but was independent of age, hypertension, current smoking status, COPD, left ventricular ejection fraction, left ventricular end-diastolic diameter, heart rate, preoperative serum Cr, and preoperative hemoglobin (Table 1).

Compared with the non-AKI group, AKI patients showed significantly increased postoperative serum Cr levels ($p < 0.001$) and decreased eGFR (47.4 ± 22.9 vs. 80.7 ± 21.8 mL/minute/1.73 m^2; $p < 0.001$) within 48 hours after TEVAR and comprehensive complications (29.2% vs.12.0%; $p = 0.009$). The length of stay in the CCU was longer in patients with AKI [4 (2–6) days vs. 3 (2–4) days; $p = 0.021$]. However, endograft leak (type I); LSCA (left subclavian artery) total coverage, stroke, red blood cell transfusion, peak leukocyte count, peak lactic acid, peak glutamic oxaloacetic transaminase, and peak glutamic pyruvic transaminase levels after TEVAR were not significantly different between the AKI and non-AKI groups (Table 2).

Malperfusion complications were celiac trunk ischemia ($n = 1$), acute renal ischemia, and/or infarction ($n = 5$), mesenteric ischemia and/or infarction ($n = 2$), and limb ischemia ($n = 9$). Interestingly, no significant difference was seen in the contrast medium doses between the two groups ($p = 0.081$). Changes in eGFR of postoperative TEVAR are shown in Fig. 1.

Predictors for AKI after TEVAR

Multivariable logistic regression analysis identified malperfusion complications (OR = 4.828; 95% CI = 1.163–20.03) as an independent predictor for AKI.

Mortality

In-hospital mortality rate was 3.8% (6 of 156 patients). Of all the patients within the first 30 days after TEVAR, 4.5% (7 of 156) died. The 1-year mortality rate was 5.8% (9 of 156 patients).

A total of six patients died in the hospital. The causes of death for these patients were multiorgan failure ($n = 1$), muscle dissolution following lower limb ischemia ($n = 1$), distal aneurysm rupture ($n = 1$), cerebral infarction ($n = 1$), and ischemic bowel disease ($n = 2$). Another seven patients died during the follow-up period. The causes of death in these patients were intracerebral bleeding ($n = 1$), retrograde tear Stanford type A aortic dissection ($n = 1$), myocardial infarction ($n = 1$), and unknown cause ($n = 4$).

Mortality at 30 days (12.5% ± 4.8% vs. 0.9% ± 0.9%; $p = 0.001$), 6 months (14.9% ± 5.2% vs. 2.0% ± 1.4%; $p = 0.001$), and 1 year (17.4% ± 5.6% vs. 2.0% ± 1.4%; $p = 0.001$) was significantly higher in patients with postprocedural AKI than in those without AKI. Survival at 1 year was significantly lower in patients with postprocedural AKI (82.6% ± 5.6% vs. 98.0% ± 1.4%; $p < 0.001$), but did not differ between the two groups at 6 years after TEVAR (77.4% ± 0.8% vs. 78.2% ± 12.8%; $p > 0.05$; Fig. 2).

All patients who died in the hospital belonged to the AKI group. Patients suffering from AKI had a 14-fold increased risk for 30-day mortality (OR = 14.3; 95% CI = 1.7–118.4; $p = 0.014$) and a 10-fold increased risk for 1-year mortality (OR = 9.5; 95% CI = 2.02–44.9; $p = 0.004$; Table 3).

Table 2 Perioperative and postoperative characteristics of patient population ($n = 156$).

Parameter	All	Non-AKI	AKI	p	Number of observations
Total cases	156	108 (69.2%)	48 (30.8%)		156
Perioperative data					
Stent length (mm), median (interquartile range)	150.0 (127.0—157.0)	150.0 (127.0—160.0; $n = 108$)	150.0 (125.0—157.0; $n = 47$)	0.705	155
Contrast medium volume (mL)	238.22 ± 65.57	231.45 ± 61.17 ($n = 93$)	252.20 ± 65.57 ($n = 45$)	0.081	138
General anesthesia	146 (93.6%)	99 (91.7%)	47 (97.9%)	0.264	156
Endoleak (type I)	12 (9.0%)	5 (12.2%)	7 (7.5%)	0.383	134
LSCA total coverage	30 (22.4%)	18 (19.4%)	12 (29.3%)	0.205	134
Retrograde aortic dissection	0	0	0	—	134
PCI	8 (5.1%)	6 (5.6%)	2 (4.2%)	1.000	156
Laboratory data					
Creatinine postoperative day 1 (μmol/L), median (interquartile range)	90.0 (74.0—113.7)	84.8 (72.4—98.4; $n = 105$)	111.7 (83.8—141.9; $n = 48$)	<0.001	153
Creatinine postoperative day 2 (μmol/L), median (interquartile range)	92.1 (74.6—116.4)	84.5 (73.0—104.2; $n = 103$)	126.6 (97.8—171.4; $n = 46$)	<0.001	149
Postoperative minimum eGFR within 48 h	70.7 ± 26.9	80.7 ± 21.8 ($n = 108$)	47.4 ± 22.9 ($n = 48$)	<0.001	156
Peak GOT (U/L), median (interquartile range)	28.0 (17.0—50.0)	27.0 (17.0—50.0; $n = 105$)	30 (18.0—45.0; $n = 47$)	0.742	152
Peak GPT (U/L), median (interquartile range)	28.0 (18.0—69.0)	30.0 (15.0—70.0; $n = 105$)	25.0 (19.0—67.0; $n = 47$)	0.720	152
Postoperative peak lactic acid (μmol/L), median (interquartile range)	1.7 (1.3—2.5)	1.7 (1.3—2.5; $n = 99$)	1.7 (1.4—2.7; $n = 46$)	0.870	145
Postoperative minimum hemoglobin (g/dL)	103.8 ± 16.3	105.3 ± 14.6 ($n = 107$)	100.5 ± 19.5 ($n = 47$)	0.129	154
Peak leukocyte count (10^9/L)	13.6 ± 3.9	13.4 ± 3.8 ($n = 108$)	14.1 ± 4.3 ($n = 47$)	0.290	155
In-hospital complications					
Postoperative stroke	9 (5.8%)	5 (4.6%)	4 (8.3%)	0.587	156
Malperfusion complications	17 (10.9%)	8 (7.4%)	9 (18.8%)	0.036	156
Postoperative MI	0	0	0	—	156
Postoperative paraplegia	0	0	0	—	156
Multiple organ failure	2 (1.3%)	0 (0%)	2 (4.2%)	0.093	156
Comprehensive complications (any of the aforementioned complications)	27 (17.3%)	13 (12.0%)	14 (29.2%)	0.009	156
Death in hospital	6 (3.8%)	0(0%)	6 (12.5%)	0.001	156
Reversible disturbance of consciousness	18 (11.5%)	9 (8.3%)	9 (18.8%)	0.060	156
Blood transfusion	12 (7.7%)	6 (5.6%)	6 (12.5%)	0.239	156
CRRT	7 (4.5%)	0 (0%)	7 (14.6%)	<0.001	156
Postoperative days in CCU, median (interquartile range)	3.0 (2.0—5.0)	3.0 (2.0—4.0; $n = 108$)	4.0 (2.0—6.0; $n = 48$)	0.021	156
Postoperative days in hospital, median (interquartile range)	13.0 (9.0—16.0)	14.0 (10.0—16.3; $n = 108$)	13.0 (9.0—17.0; $n = 48$)	0.816	156

AKI = acute kidney injury; CCU = coronary care unit; CRRT = continuous renal replacement therapy; eGFR = estimated glomerular filtration rate; GOT = glutamic oxaloacetic transaminase; GPT = glutamic pyruvic transaminase; LSCA = left subclavian artery; MI = myocardial infarction; PCI = percutaneous coronary intervention.

Discussion

In contrast to previous research, the present study focused on postoperative acute renal function change and prognosis of patients with aortic type B dissection following TEVAR. The major findings based on our results were: (1) acute renal injury following TEVAR in patients with aortic type B dissection was associated with malperfusion complications;

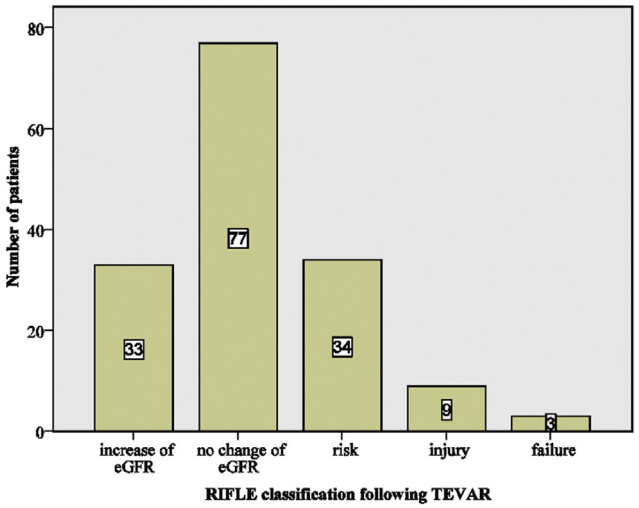

Figure 1 Incidence of postprocedural AKI after TEVAR according to changes in GFR. AKI was determined based on risk, injury, and failure status according to the RIFLE classification. AKI = acute kidney injury; GFR = glomerular filtration rate; TEVAR = thoracic endovascular aortic repair.

(2) acute renal injury after TEVAR of patients with aortic type B dissection was associated with poor short-term and middle-term survival.

Based on an inconsistent definition of AKI and differences in research populations, a wide range of incidence of renal dysfunction has been reported in patients undergoing TEVAR. In a series of 175 patients (including those with thoracic aortic aneurysms, Stanford type B aortic dissections, penetrating thoracic ulcers, and traumatic aortic transection) undergoing TEVAR, Pisimisis and co-workers[7] reported 11.9% of patients with postoperative AKI (per the RIFLE classification). Chang et al[13] reported 32% of patients with postoperative acute renal insufficiency (increase > 50% in preprocedural serum Cr) in patients undergoing complex endovascular thoracoabdominal aneurysm repair. The RIFLE classification is a more sensitive indicator of renal function and can be used as a risk factor to predict long-term survival, which was suggested by the Acute Dialysis Quality Initiative Group.[2] In our study, we focused on renal functional analysis of patients with type B aortic dissection undergoing TEVAR according to the RIFLE classification. The incidence of AKI was observed to be 30.8% in patients undergoing TEVAR, which was similar to the value of 34% reported by Eggebrecht et al,[5] confirming that acute renal injury after TEVAR is a common problem in patients with type B aortic dissection.

Because all patients needed to undergo coronary angiography, and some patients underwent PCI, larger volumes of contrast medium were used in our study compared with a previous study,[7] which may contribute to the higher incidence of AKI. Contrast-induced nephropathy (CIN) is a widely recognized cause for renal dysfunction in interventional procedures, especially in patients with pre-existing renal insufficiency.[14] Interestingly, the amount of contrast media used in the AKI group compared with that used in the non-AKI group did not reach statistical significance in the present study, inconsistent with a previous report.[5] CIN is generally defined as an increase in serum Cr over 25% or 44 mmol from the baseline value 48—72 hours after contrast medium administration in the absence of any other etiology.[15] However, aortic dissection often results in tearing of blood vessels and renal ischemia. Contrast agents are not likely to be the only factor that causes AKI after TEVAR. We speculate that the mechanisms of CIN should not be the major cause of the occurrence of postoperative AKI in patients with type B aortic dissection undergoing TEVAR.

Impaired renal function at baseline has been identified as a risk factor for AKI after multivariate adjustment for comorbidities in the settings of endovascular AAA repair (EVAR), sepsis, PCI, and cardiac surgery.[16] In our study, preoperative eGFR level and CKD were associated with AKI. Although previous studies showed that preoperative renal dysfunction was a significant and independent risk factor for death after endovascular aneurysm repair,[17] our data showed there was no statistically significant correlation between preoperative CKD and mortality. A possible explanation for this discrepancy is that the pathological changes of aortic dissection are different from aortic aneurysm. Preoperative CKD alone does not play an important role in the mortality that follows TEVAR.

Our study showed acute aortic dissection and complicated dissection were associated with an increased risk of AKI following TEVAR. Data from Eggebrecht and colleagues[18] indicated that marked inflammation occurs after TEVAR, especially in patients with acute aortic pathology (e.g., acute dissection or aortic transection). The development of inflammation may, therefore, contribute to the occurrence of AKI. Furthermore, emergency interventions were often prompted by complications of the dissection with an inherently increased individual risk as compared with chronic dissection. Nevertheless, the acute phase repair of acute complicated type B aortic dissection should not be altered or discontinued on the basis of AKI, inasmuch

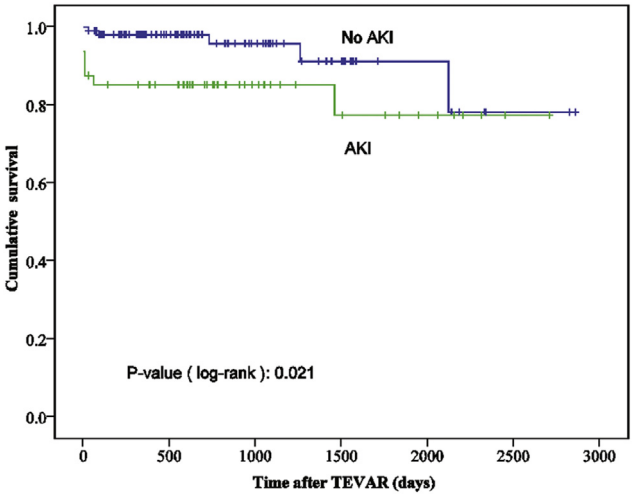

Figure 2 Cumulative survival according to the occurrence of periprocedural acute kidney injury (AKI). Survival of patients with and without AKI after endovascular intervention for type B dissection (TEVAR). Kaplan—Meier estimates of overall cumulative survival rate in both groups; $p = 0.02$ by log-rank test. TEVAR = thoracic endovascular aortic repair.

Table 3 Cox regression analysis of association between clinical characteristics and outcome.

	30-day mortality		1-year mortality	
	OR (95% CI)	*p*	OR (95% CI)	*p*
Hypertension	0.49 (0.09–2.42)	0.380	0.38 (0.10–1.43)	0.152
Preoperative creatinine	1.01 (0.99–1.02)	0.501	1.0 (0.99–1.02)	0.507
Preoperative CKD (eGFR $\leq$ 60 mL/min/1.73 m^2)	0.57 (0.07–4.71)	0.600	0.87 (0.18–4.08)	0.855
Peak leukocyte count (10^9/L)	1.16 (0.98–1.39)	0.090	1.16 (0.96–1.29)	0.150
Postoperative peak lactic acid	1.52 (1.22–1.89)	<0.001	1.39 (1.13–1.71)	0.002
Malperfusion complications	5.19 (1.01–26.8)	0.049	3.95 (1.02–15.29)	0.047
CRRT	39.5 (8.68–179.7)	<0.001	21.21 (5.90–76.20)	<0.001
Comprehensive complications	31.7 (3.82–263.89)	0.001	8.396 (2.364–29.82)	0.001
Acute dissection	28.1 (0.01–87,508)	0.416	28.20 (0.03–23,214)	0.330
AKI	14.3 (1.72–118.4)	0.014	9.52 (2.02–44.87)	0.004

AKI = acute kidney injury; CI = confidence interval; CKD = chronic kidney disease; CRRT = continuous renal replacement therapy; eGFR = estimated glomerular filtration rate; OR = odds ratio.

as survival rates without TEVAR are poor.[19] In our study, although various factors were associated with AKI in the univariate analysis, only malperfusion complications remained predictive in the multivariate analysis. There were some possible explanations for this strong association. Aortic dissection is one of the most frequent causes of vascular acute serious complications, such as visceral ischemic necrosis (bowel, liver, kidney, and limb) with possible organ failure, shock, usually due to obliteration of splanchnic arteries by the intimal flap.[20] Acute renal ischemia induces severe alterations of the endothelium in small peritubular arterioles and capillaries, postischemic interstitial inflammation and microvasculopathy, both of which contribute to ongoing renal dysfunction.[21] Furthermore, acute artery endothelial injury, vital organ malperfusion, ischemia-reperfusion injury, rhabdomyolysis, and hemodynamic instability were also important causes of acute renal failure.[22–25]

Development of AKI is an important factor in the mortality of patients after other interventional procedures.[2,26] Despite some studies reporting that CRRT is associated with a better prognosis in patients with AKI,[27] the impact of CRRT on outcomes of patients with severe AKI after TEVAR has not been explored. Our study showed that AKI necessitating CRRT following TEVAR was associated with increased 30-day and 1-year mortality. In this study, 30-day and 1-year survival rates were lower in patients with AKI compared with those without AKI. However, there were no differences in survival at 6 years after TEVAR. Although the study populations were not identical, these findings are similar to previous reports.[5] Renal function is usually improved over time rather than being deteriorated after TEVAR in complicated dissection.[28] These indicate that postoperative AKI after TEVAR may only have impact on short- and medium-term survival of patients, with no significant effects on long-term survival, whereas TEVAR itself plays a crucial role in long-term prognosis of patients. Although previous studies showed that oral administration of *N*-acetylcysteine, adequate hydration, use of gadolinium contrast agents, and withdrawal of nephrotoxic drugs are recommended in patients at risk for AKI,[29,30] further studies are warranted to evaluate AKI prevention following TEVAR.

There were several limitations associated with this study. First, it was a retrospective, uncontrolled study. Second, some patients were lost to follow up and some data were unobtainable, making results less accurate. Third, only univariate Cox regression analysis for mortality was presented because the event count was not sufficient to support multivariate analysis with inclusion of all mortality predictors in our study. Thus, a multivariate model would not have been appropriate.

Our data suggest that postprocedural AKI was a common complication of thoracic aortic stent-graft repair in type B aortic dissection. Malperfusion complications were the only independent predictor of postprocedural AKI identified, and the occurrence of postprocedural AKI was a strong predictor of 30-day and 1-year mortality after TEVAR. Because TEVAR has become a more widely practiced procedure, strategies to further reduce postprocedural AKI should be investigated.

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