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Relationship between acute kidney injury before thoracic endovascular aneurysm repair and in-hospital outcomes in patients with type B acute aortic dissection

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Abstract

Objective Acute kidney injury (AKI) frequently occurs after catheter-based interventional procedures and increases mortality. However, the implications of AKI before thoracic endovascular aneurysm repair (TEVAR) of type B acute aortic dissection (AAD) remain unclear. This study evaluated the incidence, predictors, and in-hospital outcomes of AKI before TEVAR in patients with type B AAD. **Methods** Between 2009 and 2013, 76 patients were retrospectively evaluated who received TEVAR for type B AAD within 36 h from symptom onset. The patients were classified into no-AKI *vs*. AKI groups, and the severity of AKI was further staged according to kidney disease: improving global outcomes criteria before TEVAR. **Results** The incidence of preoperative AKI was 36.8%. In-hospital complications was significantly higher in patients with preoperative AKI compared with no-AKI (50.0% *vs*. 4.2%, respectively; $P < 0.001$), including acute renal failure (21.4% *vs*. 0, respectively; $P < 0.001$), and they increased with severity of AKI ($P < 0.001$). The maximum levels of body temperature and white blood cell count were significantly related to maximum serum creatinine level before TEVAR. Multivariate analysis showed that systolic blood pressure on admission (OR: 1.023; 95% CI: 1.003–1.044; $P = 0.0238$) and bilateral renal artery involvement (OR: 19.076; 95% CI: 1.914–190.164; $P = 0.0120$) were strong predictors of preoperative AKI. **Conclusions** Preoperative AKI frequently occurred in patients with type B AAD, and correlated with higher in-hospital complications and enhanced inflammatory reaction. Systolic blood pressure on admission and bilateral renal artery involvement were major risk factors for AKI before TEVAR.

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Keywords: Acute aortic dissection; Kidney injury; Renal failure; Thoracic endovascular aneurysm repair

1 Introduction

Acute aortic dissection (AAD) is a rare but lethal disease with an in-hospital mortality rate of approximately 13% in patients with type B AAD.[1] Patients with uncomplicated type B disease have traditionally been treated medically, however, patients with complicated type B disease who develop a mal-perfusion syndrome or a contained rupture often require surgical intervention.[2–5]

Thoracic endovascular aortic repair (TEVAR) is emerging as an important treatment strategy with better prognosis in patients with type B AAD.[6–9] Acute kidney injury (AKI) frequently occurs after catheter-based interventional procedures and has been shown to increase the in-hospital morbidity and mortality.[10–14] Moreover, AKI is also related to poor outcomes as well as enhanced inflammatory response in conservatively-treated patients with Debakey type III AAD.[15] However, little is known about the association between AKI before TEVAR and its clinical and prognostic implications in patients with type B AAD.

The goal of this study was to evaluate the incidence, predictors, and in-hospital outcomes of AKI before TEVAR in patients with type B AAD.

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2 Methods

2.1 Study population and data collection

All patients provided their written informed consent and this study was approved by the institutional ethical committee of our hospital.

Between May 2009 and December 2013, 130 patients underwent TEVAR for type B AAD at our hospital. Patients were excluded (n = 42) if they presented more than 36 hours from symptom onset. Patients with recurrent AAD (n = 1), a history of chronic renal failure (n = 1), and those with incomplete data were also excluded from our study (n = 10). Finally, data from 76 patients were evaluated. This data included patient demographics, history and physical findings, laboratory variables, imaging studies, preoperative prescribed medications, in-hospital complications, and mortality.

2.2 Study protocol

Aortic dissection was diagnosed by enhanced computed tomography (CT) and classified into either type A or type B according to the Stanford classification.[16] The renal artery and aortic information were acquired using CT scans. The dissected aorta was divided into the true and false lumens. The false lumen was further classified into the thrombosed false lumen (partial or complete thrombosis) and the patent false lumen. The imaging results were analyzed by two independent experienced radiologists.

Blood samples were measured on admission and every day before TEVAR, including white blood cell (WBC) count, percentage of neutrophils, lymphocytes, and monocytes, neutrophils/lymphocyte ratio, platelet count, and serum creatinine (SCr) levels. The serum C-reactive protein (CRP), hemoglobin, glucose, alanine aminotransferase (ALT), and aspartate aminotransferase (AST) levels were collected on admission. Body temperatures were measured at least three times per day during hospitalization.

According to the recently proposed Kidney Disease: Improving Global Outcomes (KDIGO) guidelines,[17] AKI was defined as an increase in SCr ≥ 0.3 mg/dL (≥ 26.5 μmol/L) within 48 h or an increase in SCr ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior seven days. Due to a lack of urine output data, AKI was defined by changes in SCr.

AKI was also staged for severity using the following criteria: stage 1, an increase in SCr ≥ 0.3 mg/dL (≥ 26.5 μmol/L) or 1.5 to 1.9 times baseline; stage 2, 2.0 to 2.9 times baseline; and stage 3, an increase in SCr ≥ 4.0 mg/dL, or ≥ 3.0 times baseline, or initiation of renal replacement therapy.

According to the changes in SCr, patients were divided into no-AKI (n = 48) *vs*. AKI (n = 28) groups. The AKI group was further classified as stage 1 (n = 14), stage 2 (n = 6), and stage 3 (n = 8). Stage 2 and 3 AKIs were considered more serious. We also investigated the associations between the incidence and severity of preoperative AKI and clinical outcomes, including in-hospital complications. In-hospital complications included acute renal failure, renal infarction, acute visceral ischemia, acute heart failure, stroke, aortic rupture, infectious shock, mortality, the length of intensive care unit (ICU) stay and hospital stay, and inflammatory factors. The relationship between maximum SCr levels and inflammatory factors before TEVAR was characterized. Renal infarction was defined by the presence of a low-density area without enhanced contrast on CT caused by thrombotic occlusion of either the main renal artery or the renal arterial branches. The diagnosis of acute lung injury was confirmed by a ratio of the arterial partial pressure of oxygen to the fraction of inspired oxygen ≤ 200 mmHg. Stroke included cerebral hemorrhage and cerebral infarction.

2.3 Statistical analysis

Continuous variables were expressed as mean ± SD or median (interquartile range), and categorical variables were expressed as frequencies and percentages. Categorical variables were evaluated using the chi-square test, Fisher's exact test, or Cochran-Armitage trend test, and as unpaired Student t-test or Wilcoxon rank sum test. Stepwise multivariate linear regression analyses were performed using the logarithm of maximum SCr as the dependent variable. Seven inflammatory factors [WBC; percentages of neutrophils, lymphocytes, and monocytes; neutrophils/lymphocyte ratio; PLT (platelet); and body temperature] and five covariants (age, hemoglobin, ALT, AST, and glucose) were included in the initial model to evaluate their association with maximum SCr. To identify the independent predictors of AKI, stepwise multivariate logistic regression analyses were performed including variables with $P < 0.3$ by univariate analyses. A P value < 0.05 was considered statistically significant. Data analysis was performed using SAS 9.1 software (SAS Institute, Inc, Cary, NC).

3 Results

3.1 Demographic and clinical features

Of the 76 patients with type B AAD, 28 patients (36.8%) developed AKI. Stage 1 AKI occurred in 14 patients (18.4%), stage 2 in six patients (7.9%), and stage 3 in eight patients (10.5%). Patient characteristics are shown in Table 1. Among these AKI patients, male gender predominated. On

admission, systolic blood pressure (BP) and blood glucose level were significantly higher in the AKI group than in the no-AKI group. The ratio of cardiovascular risk factors did not differ between the two groups. Patients with AKI had a higher admission SCr and maximum SCr before TEVAR compared with the no-AKI group.

Table 1. Demographic and clinical characteristics of patients with or without acute kidney injury.

Variables	No-AKI (*n* = 48)	AKI (*n* = 28)	*P* Value*	AKI	
				Stage 1 (*n* = 14)	Stage 2 & 3 (*n* = 14)
Male	39 (81.3%)	26 (92.9%)	0.165	13 (92.9%)	13 (92.9%)
Age, yr	51.6 ± 11.2	49.6 ± 12.7	0.428	46.9 ± 13.4	52.4 ± 11.9
Body mass index, kg/m^2	26.3 ± 3.5	26.7 ± 3.9	0.785	27.8 ± 4.3	25.3 ± 2.9
Systolic BP on admission, mmHg	136.6 ± 23.8	152.4 ± 32.7	0.036	149 ± 35.2	155.8 ± 30.9
Diastolic BP on admission, mmHg	77.4 ± 14.5	83.9 ± 16.5	0.094	76.8 ± 16.7	91.1 ± 13.23
Pulse pressure, mmHg	59.2 ± 15.7	68.5 ± 22.3	0.142	72.2 ± 24.0	64.7 ± 20.6
LVEF,%	62.3 ± 5.9	63.5 ± 7.5	0.279	64.6 ± 7.2	62.5 ± 7.9
Diabetes mellitus	1 (2.1%)	0 (0%)	0.442	0 (0%)	0 (0%)
Hypertension	34 (70.8%)	22 (78.6%)	0.460	12 (85.7%)	10 (71.4%)
Cerebrovascular disease	3 (6.3%)	1 (3.6%)	0.614	0 (0%)	1 (7.1%)
Smoking	26 (54.2%)	19 (67.9%)	0.241	11 (78.6%)	8 (57.1%)
Drinking	13 (27.1%)	4 (14.3%)	0.196	2 (14.3%)	2 (14.3%)
Pleural effusion	9 (18.8%)	8 (28.6%)	0.322	2 (14.3%)	6 (42.9%)
Pericardial effusion	1 (2.1%)	1 (3.6%)	0.696	0 (0%)	1 (7.1%)
Time from onset to admission, h	30 ± 13.7	29.3 ± 16.8	0.360	32.8 ± 13.7	12.9 ± 23.6
Preoperative laboratory data					
Hemoglobin on admission, g/dL	142.2 ± 15.4	139.8 ± 17.8	0.503	149.6 ± 14.7	130 ± 15.3
SCr on admission, mg/dL	0.83 ± 0.11	1.49 ± 0.13	<.0001	0.97 ± 0.12	2.01 ± 0.18
Maximum SCr, mg/dL	0.82 ± 0.11	2.81 ± 1.67	<.0001	1.72 ± 0.15	3.91 ± 1.79
ALT on admission, IU/L	17.5 (13.5–2 4)	28.5 (13–5 3)	0.098	32 (20–74)	15 (11–3 8)
AST on admission, IU/L	19.5 (15–3 0.5)	26 (17.5–5 6.5)	0.119	30 (18–55)	20.5 (16–5 8)
Glucose on admission, mg/dL	6.82 ± 1.37	9.18 ± 2.9	0.005	9.1 ± 2.3	9.3 ± 3.7
Status of the false lumen			0.134		
Patent	29 (60.4%)	22 (78.6%)		12 (85.7%)	10 (71.4%)
Partial thrombosis	16 (33.3%)	5 (17.9%)		2 (14.3%)	3 (21.4%)
Complete thrombosis	3 (6.3%)	1 (3.6%)		0 (0%)	1 (7.1%)
Renal artery involvement			0.019		
No involvement	28 (58.3%)	10 (35.7%)		2 (14.3%)	8 (57.1%)
Unilateral involvement	19 (39.6%)	12 (42.9%)		9 (64.3%)	3 (21.4%)
Bilateral involvement	1 (2.1%)	6 (21.4%)		3 (21.4%)	3 (21.4%)
Aortic diameter, mm	37.8 ± 5.5	38.2 ± 4.5	0.621	37.6 ± 5.0	38.6 ± 4.0
Aortic regurgitation	11 (22.9%)	4 (14.3%)	0.362	1 (7.1%)	3 (21.4%)
Medications after admission					
Beta-blockers	40 (83.3%)	25 (89.3%)	0.477	12 (85.7%)	13 (92.9%)
Sodium nitroprussiate	34 (70.8%)	25 (89.3%)	0.063	13 (92.9%)	12 (85.7%)
Nitroglycerin	15 (31.3%)	9 (32.1%)	0.936	2 (14.3%)	7 (50%)
ARB/ACE-I	44 (91.7%)	27 (96.4%)	0.419	14 (100%)	13 (92.9%)
Calcium channel blockers	44 (91.7%)	27 (96.4%)	0.419	13 (92.9%)	14 (100%)
Morphine hydrochloride	30 (62.5%)	18 (64.3%)	0.876	11 (78.6%)	7 (50%)

Data are presented as mean ± SD, median (interquartile range), or *n* (%). *Comparing between no-AKI and AKI groups. ACE-I: angiotensin converting enzyme inhibitors; AKI: acute kidney injury; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ARB: angiotensin receptor blocker; BMI: body mass index; BP: blood pressure; CT: computed tomography; LVEF: left ventricular ejection fraction; SCr: serum creatinine.

Table 2. Impact of preoperative acute kidney injury on in-hospital outcomes.

Variables	No-AKI (n = 48)	AKI (n = 28)	P Value*	AKI	
				Stage 1 (n = 14)	Stage 2 & 3 (n = 14)
In-hospital complications	2 (4.2%)	14 (50%)	< 0.001	4 (28.6%)	10 (71.4%)
Length of hospital stay, day	10 (8–1 3)	10.5 (8.5–1 7)	0.2517	10 (8–2 1)	12 (10-15)
Length of ICU stay, h	24 (21–3 3)	276 (24–6 24)	0.2948	528 (24–720)	24 (24–720)
Acute renal failure	0 (0%)	6 (21.43%)	0.0008	2 (14.29%)	4 (28.57%)
Infectious shock	0 (0%)	1 (3.57%)	0.1875	0 (0%)	1 (7.14%)
Acute visceral ischemia	0 (0%)	1 (3.57%)	0.1875	0 (0%)	1 (7.14%)
Stroke	0 (0%)	1 (3.57%)	0.1875	0 (0%)	1 (7.14%)
Acute lung injury	0 (0%)	1 (3.57%)	0.1875	1 (7.14%)	0 (0%)
Aortic rupture	1 (2.08%)	0 (0%)	0.4420	0 (0%)	0 (0%)
Renal infarction	1 (2.08%)	1 (3.57%)	0.6958	0 (0%)	1 (7.14%)
Mortality	0 (0%)	3 (10.7%)	0.1426	1 (7.14%)	2 (14.2%)

*Comparing between no-AKI and AKI groups. Data are presented as median (interquartile range), or n (%). AKI: acute kidney injury; ICU: intensive care unit.

According to the CT findings, the status of the false lumen was comparable between the AKI and no-AKI groups. Renal artery involvement (primarily bilateral involvement) consisted primarily of arterial stenosis or thrombosis and occurred more frequently in patients with AKI. No obvious differences in aortic diameter or frequency of aortic regurgitation occurred between groups.

With regards to medications, sodium nitroprusside alone was used more frequently in patients with AKI, however, other antihypertensive use was not significantly different between groups.

3.2 The relationship between AKI and in-hospital outcomes

Patients with AKI experienced a more complicated clinical course. In-hospital complications occurred more frequently in patients developing AKI compared with no-AKI patients (50.0% *vs*. 4.2%, respectively, $P < 0.001$; Table 2).

There was a significant trend toward overall increased frequency of complications with increasing severity of AKI (no-AKI, 4.2%; stage 1 AKI, 28.6%; stages 2 and 3 AKI, 71.4%; $P < 0.001$) (Table 2 and Figure 1). In particular, acute renal failure occurred more frequently in the AKI group. Other complications, such as acute renal infarction, acute visceral ischemia, acute heart failure, stroke, aortic rupture, and infectious shock did not differ significantly between the two groups, although they were more common in the AKI group. The in-hospital mortality was 3% in patients with AKI and 0 in no-AKI patients, although the difference was not significant ($P = 0.1426$).

3.3 Effect of AKI on inflammatory factors

The effects of AKI on inflammatory factors are shown in Table 3. The maximum SCr level and the SCr level at discharge differed significantly between the AKI and no-AKI groups. The maximum CRP level and body temperature were comparable between AKI and no-AKI groups. The maximum WBC, percentage of neutrophils, percentage of lymphocytes, and neutrophils/lymphocyte ratio before TEVAR were significantly higher in patients with AKI compared with those without AKI.

Based on the stepwise multivariate linear regression model, the correlation between inflammatory factors and maximum SCr before TEVAR are shown in Table 4. Eight inflammation factors (CRP; maximum WBC; percentage of neutrophils, lymphocytes, monocytes; neutrophils/lymphocyte ratio; PLT; and body temperature) and five covariants (age, hemoglobin, ALT, AST, and glucose) were included in the model. Body temperature, WBC count, and Hemoglobin level were independent correlates for maximum SCr.

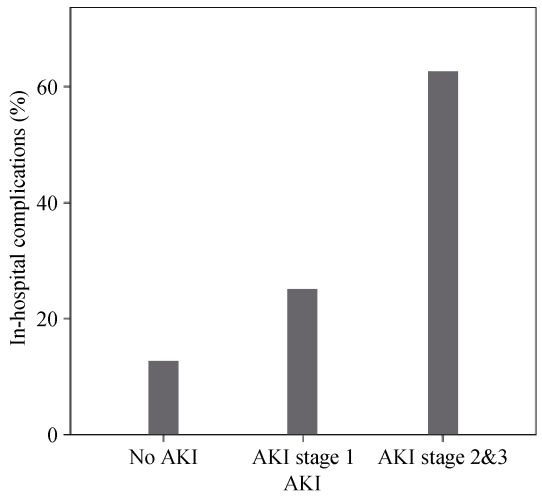

Figure 1. The incidence of in-hospital complications in patients without AKI, developing AKI stage 1, AKI stage 2&3.

Table 3. Effects of AKI on inflammatory factors.

Variables	No-AKI (n = 48)	AKI (n = 28)	P Value*	AKI	
				Stage 1 (n = 14)	Stage 2 & 3 (n = 14)
CRP on admission, mg/dL	26.0 ± 12.7	30.2 ± 11.9	0.244	30.5 ± 11.4	30.0 ± 13.0
Maximum WBC count, g/L	11.6 ± 2.7	14.3 ± 4.6	0.021	14.6 ± 4.2	13.9 ± 5.1
Maximum, N%	81.7 ± 7.4	85.7 ± 4.2	0.017	86.1 ± 4.3	85.3 ± 4.2
Maximum, L%	11.0 ± 5.1	8.5 ± 3.4	0.043	8.6 ± 3.4	8.3 ± 3.4
Maximum, N/L%	9.8 ± 6.3	12.2 ± 6.1	0.037	12.0 ± 5.7	12.5 ± 6.6
Maximum, M%	6.6 ± 2.7	5.6 ± 2.2	0.078	5.1 ± 1.8	6.1 ± 2.5
Maximum platelet count, g/L	164.2 ± 56.3	152.2 ± 44.1	0.398	152.1 ± 47.0	152.3 ± 42.8
Maximum body temperature, °C	37.5 ± 0.5	37.7 ± 0.6	0.288	37.7 ± 0.6	37.7 ± 0.7

*Comparing between no-AKI and AKI groups. Data are presented as mean ± SD. AKI: acute kidney injury; CRP: C-reactive protein; L%: percent of lymphocyte; M%: percent of monocyte; N%: percent of neutrophils; N/L: neutrophils/lymphocyte; WBC: white blood cell.

Table 4. Multivariate linear regression analyses for maximum serum creatinine before surgery in relation to inflammatory indicators.

Variables	β	SE	P Value
Body temperature	0.149	0.016	< 0.001
WBC	0.059	0.018	0.023
HBG	–0.012	0.006	0.040

The model R-Square is 0.9929. HBG: hemoglobin; WBC: white blood cell.

3.4 Independent predictors of AKI

On final logistic regression analysis, after adjusting for baseline variables, the following variables were independent predictors of preoperative AKI, systolic BP on admission (OR: 1.023; 95% CI: 1.003–1.044; P = 0.0238), and bilateral renal artery involvement (OR: 19.076; 95% CI: 1.914–190.164; P = 0.0120), (Table 5).

Table 5. Multivariate logistic regression analyses for predictors of acute kidney injury.

Variables	OR (95% CI)	P Value
Systolic blood pressure on admission	1.023 (1.003–1.044)	0.0238
Renal artery involvement		
Unilateral	1.751 (0.604–5 .077)	0.3025
Bilateral	19.076 (1.914–190.164)	0.0120

4 Discussion

In patients with type B AAD and enhanced inflammatory reaction, our results demonstrated that AKI occurred more frequently before TEVAR and was associated with increased in-hospital complications. The risk of in-hospital complications increased with severity of AKI. Moreover, we found that systolic BP on admission and bilateral renal artery involvement were significant independent predictors of preoperative AKI.

AAD is a catastrophic cardiovascular disease with high mortality. Medical treatment is commonly recommended for uncomplicated type B AAD. However, life-threatening, complicated type B dissection (such as aortic rupture, visceral ischemia, aortic progressive expansion, and repeated pain) requires surgical intervention.[2–5] TEVAR has greatly improved in recent years and is now widely used. Its efficacy and safety have been confirmed by multiple multicenter clinical studies.[6–9]

AKI frequently occurs after catheter-based interventional procedures, especially in patients with preexisting renal impairment and AKI is associated with increased in-hospital complications and short-and long-term mortality.[10-14] Wang, *et al*.[18] reported that emergency surgery and baseline SCr levels > 2.0 mg/dL were powerful predictors of poor prognosis in patients with renal insufficiency before TEVAR. However, no study has evaluated the incidence and prognostic value of AKI based on KDIGO criteria before TEVAR in patients with type B AAD. According to the new KDIGO criteria, the incidence of AKI before TEVAR was 36.8% in the current study. In addition, patients with preoperative AKI had a 12-fold higher rate of in-hospital complications compared with those without AKI (50% *vs*. 4.2%, respectively), and the risk of complications was significantly increased with AKI severity. These findings indicate that preoperative AKI may serve as a powerful predictor of poor outcome after TEVAR. Consistent with previous studies,[11,15,19] patients with AKI developed more acute renal failure, which was an indicator of poor prognosis. Accordingly, the severity of AKI before TEVAR could guide the optimal timing of TEVAR in high-risk patients. In addition, methods of preventing AKI exacerbation (including adequate hydration, use of minimal effective volumes of con-

trast medium, and discontinuance of nephrotoxic drugs during the perioperative period) should be considered.

Inflammation plays a major role in the development of AAD and the elevation of many inflammatory markers has correlated with worse prognosis in patients with AAD.[20–23] Our results showed that maximum levels of body temperature and WBC count were significantly associated with maximum SCr before TEVAR.

Inflammatory reaction may be involved in pathophysiology of AKI. We found that inflammatory mediators were generated to regulate inflammatory reaction in impaired kidneys and inflammatory response to aortic injury was further enhanced. This enhanced inflammatory reaction may have further impaired renal function.[24] Therefore, the combination of AKI and inflammation may be a strong predictor of adverse in-hospital outcomes.

In our study, the CRP level on admission did not differ significantly between AKI and no-AKI groups and it did not correlate with maximum SCr levels. Thus, we presumed that maximum SCr level was not associated with CRP levels on admission. Komukai, *et al*.[25] reported that maximum CRP was significantly related to impaired oxygenation in patients with AAD. Sakakura, *et al*.[26] reported that peak CRP level was associated with poor long-term outcomes in patients with type B AAD. Moreover, Schillinger, *et al*.[27] reported that an elevated CRP level on admission was a strong predictor of adverse outcome in patients with AAD. Accordingly, our findings require confirmation using a larger cohort of patients with type B AAD.

We identified systolic BP on admission and bilateral renal artery involvement as strong predictors of AKI. Regarding the AKI patients in our study, reduction of renal blood flow, whether generalized or localized, played a critical role in the pathophysiology of AKI.[24] Bilateral renal artery involvement was more harmful. Extension of the dissection may involve the renal artery causing thrombosis or stenosis, which may directly impair renal perfusion, thus resulting in AKI. In addition, the renin-angiotensin aldosterone system, which was activated because of renal artery involvement, induced a dramatic increase in blood pressure. Therefore, higher systolic BP on admission may be associated with renal artery involvement. Besides, higher systolic BP may lead to sustainable expansion of the false lumen, causing generalized ischemia to the kidney leading to AKI. Therefore, strategies to improve renal perfusion before TEVAR should be considered in order to prevent AKI exacerbation and to lower the rate of in-hospital complications.

Our study had several limitations including its single-center, retrospective nature. In addition, patients were grouped into AKI *vs*. no-Aki based on only their SCr levels. Urine output changes were not considered due to the retrospective nature of the study. The short and long-term mortality was also unclear in AKI patients with type B AAD, thus, in-hospital mortality may have been underestimated. Both short and long-term follow-up studies are needed to assess the prognostic impact of TEVAR in AKI patients with type B AAD. Some patients abandoned treatment and were discharged early from hospital for a variety of reasons. Finally, we did not evaluate the volume of contrast medium administered which may have affected our results.

In conclusion, preoperative AKI was common before TEVAR of type B AAD, and was a strong predictor of in-hospital complications and enhanced inflammatory reaction. Furthermore, systolic BP on admission and bilateral renal artery involvement were independent risk factors for preoperative AKI. Knowledge of AKI severity before TEVAR would be advantageous in guiding treatment and reducing the risk of adverse events in high-risk patients. Strategies to prevent deterioration of renal function should be further investigated during the perioperative period.

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References

1 Suzuki T, Mehta RH, Ince H, *et al*. Clinical profiles and outcomes of acute type B aortic dissection in the current era: lessons from the International Registry of Aortic Dissection (IRAD). *Circulation* 2003; 108: 312–317.

2 Sheikh AS, Ali K, Mazhar S. Acute aortic syndrome. *Circulation* 2013; 128: 1122–1127.

3 Clough RE, Nienaber CA. Management of acute aortic syndrome. *Nat Rev Cardiol* 2015; 12: 103–114.

4 Tsai TT, Trimarchi S, Nienaber CA. Acute aortic dissection: perspectives from the International Registry of Acute Aortic Dissection (IRAD). *Eur J Vasc Endovasc Surg* 2009; 37: 149–159.

5 Marui A, Mochizuki T, Mitsui N, *et al*. Toward the best treatment for uncomplicated patients with type B acute aortic dissection: a consideration for sound surgical indication. *Circulation* 1999; 100: 275–280.

6 Kim JB, Sundt TM. Best surgical option for arch extension of type B aortic dissection: the open approach. *Ann Cardiothorac Surg* 2014; 3: 406–412.

7 Nienaber CA, Kische S, Ince H, *et al*. Thoracic endovascular aneurysm repair for complicated type B aortic dissection. *J Vasc Surg* 2011; 54: 1529–1533.

8 Andersen ND, Keenan JE, Ganapathi AM, *et al*. Current management and outcome of chronic type B aortic dissection: results with open and endovascular repair since the advent of thoracic endografting. *Ann Cardiothorac Surg* 2014; 3: 264–274.
9 Fattori R, Tsai TT, Myrmel T, *et al*. Complicated acute type B dissection: is surgery still the best option? a report from the International Registry of Acute Aortic Dissection. *JACC Cardiovasc Interv* 2008; 1: 395–402.
10 Eggebrecht H, Breuckmann F, Martini S, *et al*. Frequency and outcomes of acute renal failure following thoracic aortic stent-graft placement. *Am J Cardiol* 2006; 98: 458–463.
11 Zhu JC, Chen SL, Jin GZ, *et al*. Acute renal injury after thoracic endovascular aortic repair of Stanford type B aortic dissection: incidence, risk factors, and prognosis. *J Formos Med Assoc* 2014; 113: 612–619.
12 Marenzi G, Cabiati A, Bertoli SV, *et al*. Incidence and relevance of acute kidney injury in patients hospitalized with acute coronary syndromes. *Am J Cardiol* 2013; 111: 816–822.
13 Choi JS, Kim YA, Kim MJ, *et al*. Relation between transient or persistent acute kidney injury and long-term mortality in patients with myocardial infarction. *Am J Cardiol* 2013; 112: 41–45.
14 Dangas G, Iakovou I, Nikolsky E, *et al*. Contrast-induced nephropathy after percutaneous coronary interventions in relation to chronic kidney disease and hemodynamic variables. *Am J Cardiol* 2005; 95: 13–19.
15 Takahashi T, Hasegawa T, Hirata N, *et al*. Impact of acute kidney injury on in-hospital outcomes in patients with De-Bakey type III acute aortic dissection. *Am J Cardiol* 2014; 113: 1904–1910.
16 Daily PO, Trueblood HW, Stinson EB, *et al*. Management of acute aortic dissections. *Ann Thorac Surg* 1970; 10: 237–247.
17 Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int (Suppl* 1) 2012; 2: S1–S138.
18 Wang GJ, Fairman RM, Jackson BM, *et al*. The outcome of thoracic endovascular aortic repair (TEVAR) in patients with renal insufficiency. *J Vasc Surg* 2009; 49: 42–46.
19 Ellenberger C, Schweizer A, Diaper J, *et al*. Incidence, risk factors and prognosis of changes in serum creatinine early after aortic abdominal surgery. *Intensive Care Med* 2006; 32: 1808–1816.
20 Luo F, Zhou XL, Li JJ, *et al*. Inflammatory response is associated with aortic dissection. *Ageing Res Rev* 2009; 8: 31–35.
21 Kuehl H, Eggebrecht H, Boes T, *et al*. Detection of inflammation in patients with acute aortic syndrome: comparison of FDG-PET/CT imaging and serological markers of inflammation. *Heart* 2008; 94: 1472–1477.
22 Shimada S, Nakamura H, Kurooka A, *et al*. Fever associated with acute aortic dissection. *Circ J* 2007; 71: 766–771.
23 He R, Guo DC, Estrera AL, *et al*. Characterization of the inflammatory and apoptotic cells in the aortas of patients with ascending thoracic aortic aneurysms and dissections. *J Thorac Cardiovasc Surg* 2006; 131: 671–678.
24 Bonventre JV, Yang L. Cellular pathophysiology of ischemic acute kidney injury. *J Clin Invest* 2011; 121: 4210–4221.
25 Komukai K, Shibata T, Mochizuki S. C-reactive protein is related to impaired oxygenation in patients with acute aortic dissection. *Int Heart J* 2005; 46: 795–799.
26 Sakakura K, Kubo N, Ako J, *et al*. Peak C-reactive protein level predicts long-term outcomes in type B acute aortic dissection. *Hypertension* 2010; 55: 422–429.
27 Schillinger M, Domanovits H, Bayegan K, *et al*. C-reactive protein and mortality in patients with acute aortic disease. *Intensive Care Med* 2002; 28: 740–745.