



Predictors and in-hospital outcomes of preoperative acute kidney injury in patients with type A acute aortic dissection

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Abstract

Background Acute kidney injury (AKI) is common after surgery for acute aortic dissection (AAD) and increases in-hospital and long-term mortality. However, few data exist on the clinical and prognostic relevance of early preoperative AKI in patients with type A AAD. We aimed to determine the incidence and predictors of preoperative AKI and the impact of AKI on in-hospital outcomes in patients with type A AAD. **Methods** From May 2009 to June 2014, we retrospectively enrolled 178 patients admitted to our hospital within 48 h from symptom onset and receiving open surgery for type A AAD. The patients were divided into no AKI and AKI groups and staged with AKI severity according to the KDIGO criteria before surgery. **Results** AKI occurred in 41 patients (23.0%). The incidence of in-hospital complications was significantly higher in patients with preoperative AKI compared to no AKI (41.5% vs. 9.5%, $P < 0.001$), including renal infarction (7.3% vs. 0, $P = 0.012$), and it increased with AKI severity ($P_{\text{trend}} < 0.001$). Patients with AKI had higher in-hospital mortality compared with patients without AKI, although no significant difference was found (14.6% vs. 5.1%, $P = 0.079$). Multivariate analysis indicated that male gender, diastolic blood pressure on admission and bilateral renal artery involvement were independent predictors of preoperative AKI in patients with type A AAD. **Conclusions** Early AKI before surgery was common in patients with type A AAD, and was associated with increased in-hospital complications. Male gender, diastolic blood pressure on admission and bilateral renal artery involvement were major predictors for preoperative AKI.

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1 Introduction

Acute aortic dissection (AAD) is a rare but potentially disastrous disease. The reported mortality rate of AAD is 1% to 2% per hour after the first 24 to 48 h of symptom onset.^[1] Type A AAD develops rapidly and is often accompanied by life-threatening complications, such as aortic rupture, cardiac tamponade, coronary obstruction, or visceral ischemia, which requires urgent surgical management.^[1–3] Despite improved surgical techniques in recent years, in-hospital mortality in patients with type A AAD remains high, ranging from 10% to 35%.^[4–10]

Acute kidney injury (AKI) is common after aortic surgery for AAD and increases in-hospital and long-term mortality.^[11–14] Recent study showed preoperative renal function could effectively predict the need for renal replacement therapy after surgery for type A AAD.^[15] However, there is a paucity of data regarding early preoperative AKI and its clinical and prognostic relevance in patients with type A AAD. In the present study, we aimed to investigate the incidence and predictors of early preoperative AKI and the impact of AKI on in-hospital outcomes in patients with type A AAD.

2 Methods

2.1 Study population

From May 2009 to June 2014, 261 consecutive patients who underwent open surgery for type A AAD were reviewed at Beijing Anzhen Hospital, Capital Medical University. Since this study focused on early AKI, patients ad-

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mitted to our hospital more than 48 h from symptom onset ($n = 73$) were excluded. Patients with recurrent AAD ($n = 1$), chronic renal failure on renal replacement therapy ($n = 1$), and those with data incompleteness were also excluded from our study ($n = 8$). Finally, a total of 178 patients were included in this study. Clinical variables were collected, including patient demographics and history, physical findings, laboratory tests, imaging findings, and patient outcomes including in-hospital complications and mortality. This study complied with the Declaration of Helsinki, and was approved by the institutional ethical committee of Beijing Anzhen Hospital, Capital Medical University. All patients provided written informed consent.

2.2 Study protocol

Aortic dissection was diagnosed by contrast-enhanced CT and defined as type A or type B according to Stanford classification.^[16] Both the true and false lumens of the dissected aorta were defined according to CT imaging. The false lumen was further classified into the thrombosed false lumen (partial thrombosis or complete thrombosis) and the patent false lumen. The extent of renal artery involvement was also acquired. The imaging findings were analyzed by two independent experienced radiologists.

Blood samples for measurements of the serum creatinine (SCr) level were obtained on admission and every day before surgery to achieve at least two time points for blood sampling. In addition, the serum C-reactive protein (CRP), hemoglobin, and glucose levels were obtained on admission.

According to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines,^[17] AKI was defined as an increase in SCr ≥ 0.3 mg/dL (≥ 26.5 $\mu\text{mol/L}$) 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 merely defined by changes in SCr. Preoperative AKI was defined as occurrence of AKI before aortic surgery. Preoperative AKI was also staged for severity according to the following criteria: stage 1, an increase in SCr ≥ 0.3 mg/dL (≥ 26.5 $\mu\text{mol/L}$) or 1.5 to 1.9 times baseline; stage 2, 2.0 to 2.9 times baseline; stage 3, an increase in SCr ≥ 4.0 mg/dL, or ≥ 3.0 times baseline, or initiation of renal replacement therapy.

Patients were divided into no AKI ($n = 137$) and AKI ($n = 41$) groups according to the changes in SCr. The AKI group was further classified as stage 1 ($n = 19$), stage 2 ($n = 17$), and stage 3 ($n = 5$). We evaluated the relations between the incidence and severity of early preoperative AKI and clinical outcomes including in-hospital complications such as renal infarction, acute lung injury, acute visceral ischemia,

acute heart failure, stroke, aortic rupture, re-exploration for bleeding, and multiple systemic organ failure. The in-hospital mortality, duration on mechanical ventilation, and the lengths of intensive care unit (ICU) stay and hospital stay were also compared between the AKI and no AKI groups. Renal infarction was defined by the presence of a low-density area without enhanced contrast on CT scan, caused by thrombotic occlusion of either the main renal artery or the arterial branches. Acute lung injury was confirmed by the ratio of the arterial partial pressure of oxygen to the fraction of inspired oxygen ≤ 200 mmHg. Stroke included cerebral hemorrhage and ischemic cerebral infarction.

2.3 Statistical analysis

Categorical variables were presented as frequencies and percentages, while the continuous variables were presented as means \pm SD or median (interquartile range). Chi-square test, Fisher's exact test, or Cochran-Armitage trend test were used to compare categorical variables, and unpaired Student *t* test or Wilcoxon rank sum test to compare continuous variables. To determine 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, USA).

3 Results

3.1 Demographic and clinical features

Among the 178 patients with type A AAD, 41 patients (23.0%) exhibited AKI, including 19 patients (10.7%) in stage 1, 17 patients (9.6%) in stage 2, and 5 patients (2.8%) in stage 3. Patient characteristics are presented in Table 1 and 2. Compared with patients without AKI, patients with AKI were predominantly male ($P = 0.004$). There were no significant differences in terms of cardiovascular risk factors between the two groups (Table 1). The time from symptom onset to admission was similar between AKI and no AKI groups. Patients with AKI had lower diastolic blood pressure (BP) on admission ($P = 0.033$). Patients with AKI presented significantly higher SCr on admission compared to no AKI group ($P < 0.001$). According to the CT findings, the status of the false lumen showed no significant differences between the AKI and no AKI groups. Renal artery involvement (mainly bilateral involvement) occurred more frequently in patients with AKI ($P = 0.004$). These mainly consisted of renal arterial stenosis or thrombosis. The aortic diameter and aortic regurgitation indicated no obvious differences between the two groups (Table 2).

Table 1. Demographics and patient history of patients with or without acute kidney injury.

Variables	No AKI (<i>n</i> = 137)	AKI (<i>n</i> = 41)	<i>P</i> value*	AKI		
				Stage 1 (<i>n</i> = 19)	Stage 2 (<i>n</i> = 17)	Stage 3 (<i>n</i> = 5)
Age, yrs	47.8 ± 9.4	48.1 ± 12.0	0.679	49.7 ± 14.8	47.5 ± 9.6	44.4 ± 7.8
Male	102 (74.5%)	39 (95.1%)	0.004	18 (94.7%)	17 (100%)	4 (80%)
Height, cm	171.1 ± 6.7	173.1 ± 5.5	0.097	174.5 ± 5.6	171.9 ± 2.0	170.3 ± 9.5
Weight, kg	76.9 ± 12.3	80.1 ± 9.2	0.156	80.9 ± 8.7	81.2 ± 10.3	73.8 ± 7.5
Body mass index, kg/m ²	26.2 ± 3.5	26.7 ± 2.5	0.466	26.5 ± 2.2	27.5 ± 3.3	25.4 ± 0.8
Hypertension	90 (65.7%)	31 (75.6%)	0.233	15 (79.0%)	13 (76.5%)	3 (60%)
Diabetes mellitus	4 (2.9%)	0	0.269	0	0	0
Cerebrovascular disease	2 (1.5%)	2 (4.9%)	0.195	0	2 (11.8%)	0
Smoking	65 (47.5%)	24 (58.5%)	0.213	11 (57.9%)	11 (64.7%)	2 (40%)

Data are presented as mean ± SD, or *n* (%). *Comparing between no AKI and AKI groups. AKI: acute kidney injury.

Table 2. Clinical, laboratory, and imaging findings of patients with or without acute kidney injury.

Variables	No AKI (<i>n</i> = 137)	AKI (<i>n</i> = 41)	<i>P</i> value	AKI		
				Stage 1 (<i>n</i> = 19)	Stage 2 (<i>n</i> = 17)	Stage 3 (<i>n</i> = 5)
Time from onset to admission, h	28.0 ± 15.4	27.3 ± 19.8	0.325	27.6 ± 22.7	28.0 ± 19.4	23.6 ± 9.2
Systolic BP on admission, mmHg	132.1 ± 19.7	125.2 ± 23.9	0.097	120.3 ± 18.6	129.41 ± 27.47	129.4 ± 30.71
Diastolic BP on admission, mmHg	71.7 ± 12.8	66.3 ± 14.9	0.033	67.0 ± 15.9	64.5 ± 13.4	73.6 ± 17.5
LVEF, %	64.1 ± 7.4	62.6 ± 7.5	0.208	61.5 ± 5.5	61.9 ± 7.5	67.8 ± 12.0
Laboratory data on admission						
Hemoglobin, g/dL	13.1 ± 2.1	13.5 ± 1.6	0.371	13.5 ± 1.6	13.6 ± 1.4	12.9 ± 2.2
Glucose, mg/dL	142.3 ± 50.1	147.2 ± 37.1	0.101	148.8 ± 36.9	139.3 ± 39.1	168.8 ± 26.1
SCr, mg/dL	0.75 (0.69–0.83)	1.18 (1.11–1.38)	< 0.001	1.14 (1.07–1.24)	1.37 (1.15–1.41)	1.38 (1.27–1.47)
CRP, mg/dL	25.5 ± 13.4	28.6 ± 14.6	0.268	23.2 ± 13.9	30.9 ± 16.0	34.1 ± 13.6
CT findings						
Status of the false lumen			0.324			
Patent	93 (67.9%)	26 (63.4%)		8 (42.1%)	14 (82.4%)	4 (80%)
Partial thrombosis	41 (29.9%)	12 (29.3%)		9 (47.4%)	2 (11.8%)	1 (20%)
Complete thrombosis	3 (2.2%)	3 (7.3%)		2 (10.5%)	1 (5.9%)	0
Renal artery involvement			0.004			
No involvement	62 (45.3%)	13 (31.7%)		8 (42.1%)	4 (23.5%)	1 (20%)
Unilateral involvement	68 (49.6%)	18 (43.9%)		7 (36.8%)	9 (52.9%)	2 (40%)
Bilateral involvement	7 (5.1%)	10 (24.4%)		4 (21.1%)	4 (23.5%)	2 (40%)
Aortic diameter, mm	48.0 ± 8.7	49.3 ± 10.2	0.443	50.1 ± 11.5	48.45 ± 9.5	49.0 ± 8.8
Aortic regurgitation	67 (48.9%)	19 (46.3%)	0.773	11 (57.9%)	6 (35.3%)	2 (40%)

Data are presented as mean ± SD, median (interquartile range), or *n* (%). *Comparing between no AKI and AKI groups. AKI: acute kidney injury; BP: blood pressure; CRP: C-reactive protein; CT: computed tomography; LVEF: left ventricular ejection fraction; SCr: serum creatinine.

3.2 In-hospital outcomes

Patients with AKI before surgery had a significantly higher incidence of in-hospital complications compared with those without AKI (41.5% vs. 9.5%, $P < 0.001$; Table 3). There was a significant trend toward increased frequency of overall complications along with the severity of AKI (no AKI, 9.5%; stage 1, 31.6%; stage 2, 47.1%; stage 3, 60.0%; $P_{\text{trend}} < 0.001$; Table 3 and Figure 1). In particular, renal infarction occurred more frequently in the AKI group, and these complications were more pronounced in patients with

relatively severe AKI. Other complications, such as acute lung injury, acute visceral ischemia, acute heart failure, stroke, aortic rupture, re-exploration for bleeding, and multiple systemic organ failure, showed no significant differences between the two groups, although they were more common in the AKI group. The in-hospital mortality was 14.6% and 5.1% in patients with AKI and without AKI, respectively, although no significant difference was observed ($P = 0.079$). Duration of ICU stay was longer in patients with AKI than in those without AKI (Table 3).

Table 3. Impact of preoperative acute kidney injury on in-hospital outcomes.

Variables	No AKI (n = 137)	AKI (n = 41)	*P-value	AKI		
				Stage 1 (n = 19)	Stage 2 (n = 17)	Stage 3 (n = 5)
In-hospital complications	13 (9.5%)	17 (41.5%)	< 0.001	6 (31.6%)	8 (47.1%)	3 (60%)
Renal infarction	0	3 (7.3%)	0.012	1 (5.3%)	1 (5.9%)	1 (20%)
Acute lung injury	2 (1.5%)	2 (4.9%)	0.228	0	1 (5.9%)	1 (20%)
Acute visceral ischemia	0	2 (4.9%)	0.052	1 (5.3%)	1 (5.9%)	0
Acute heart failure	0	1 (2.4%)	0.230	0	1 (5.9%)	0
Stroke	3 (2.2%)	3 (7.3%)	0.136	1 (5.3%)	2 (11.8%)	0
Aortic rupture	3 (2.2%)	2 (4.9%)	0.325	1 (5.3%)	1 (5.9%)	0
Re-exploration for bleeding	1 (0.7%)	1 (2.4%)	0.409	0	0	1 (20%)
Multiple systemic organ failure	1 (0.7%)	2 (4.9%)	0.133	1 (5.3%)	0	1 (20%)
Mortality	7 (5.1%)	6 (14.6%)	0.079	3 (15.8%)	3 (17.7%)	0
Mechanical ventilation duration, h	19.5 (15–63.5)	36.5 (20–65)	0.266	26.5 (20–36)	71.5 (37–279)	114.5 (40–189)
Duration of ICU stay, h	39 (22–86)	69 (34–120)	0.008	65 (32–96)	62 (29–120)	134.5 (109–164.5)
Duration of hospital stay, days	15 (10–20)	12 (4–20)	0.164	11 (4–22)	11 (2–17)	20 (16–25)

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

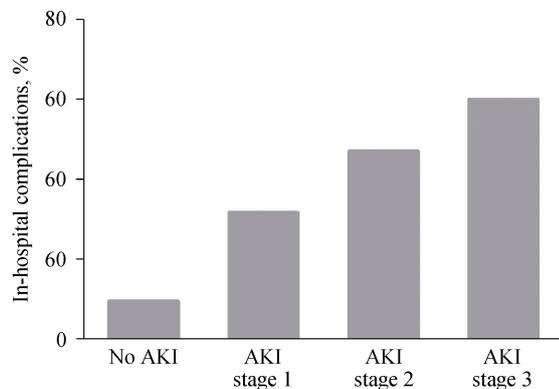


Figure 1. The incidence of in-hospital complications in patients without AKI, developing AKI stage 1, AKI stage 2, and AKI stage 3. AKI: acute kidney injury.

3.3 Predictors of AKI

On the final logistic regression, after adjusting for baseline variables, we identified male gender (OR = 5.398; 95% CI: 1.497–19.468; $P = 0.01$), diastolic BP on admission (OR = 0.961; 95% CI: 0.943–0.980; $P < 0.001$), and bilateral renal artery involvement (OR = 5.392; 95% CI: 1.390–20.914; $P = 0.015$) were independent predictors of preoperative AKI in patients with type A AAD (Table 4).

4 Discussion

In the present study, we demonstrated that early preoperative AKI was associated with increased in-hospital complications in patients with type A AAD. The incidence of in-hospital complications increased with increasing AKI stage. Moreover, we found that male gender, diastolic BP

Table 4. Multivariate logistic regression analyses for predictors of acute kidney injury.

Variables	OR (95% CI)	P Value
Gender		
Female	Reference	
Male	5.398 (1.497–19.468)	0.01
Diastolic blood pressure	0.961 (0.943–0.980)	< 0.001
Renal artery involvement		
Non	Reference	
Unilateral	0.902 (0.346–2.352)	0.833
Bilateral	5.392 (1.390–20.914)	0.015

on admission, and bilateral renal artery involvement were independent predictors of preoperative AKI.

Emergency open surgery is the mainstay of treatment for type A AAD. However, the operative mortality remains substantial (between 10% and 35%) in the past two decades.^[4–10] In the contemporary International Registry of acute Aortic Dissection (IRAD) study, the in-hospital mortality for type A AAD receiving timely and successful surgery was 26.9%, although it was less than the 56.2% mortality with medical therapy.^[5] AKI is common after thoracic aortic surgery and is associated with increased in-hospital complications and short- and long-term mortality.^[11–14] It can be speculated that type A AAD evolves rapidly and AKI might have occurred early before surgery. However, there are few data concerning the incidence and prognostic value of preoperative AKI in patients with type A AAD. In the present study, according to the new KDIGO criteria, the incidence of AKI before surgery was 23.0%, which was relatively low compared with that of AKI (RIFLE criteria) after surgery (48%–55%) in other studies.^[11,12,18,19] Even so,

patients with preoperative AKI had a 4.5-fold higher rate of in-hospital complications than those without AKI (41.5% vs. 9.5%), and the risk of complications increased with each AKI stage, which indicates that preoperative AKI may serve as a good outcome predictor as postoperative AKI. In consistent with previous studies,^[11,12,20] patients with AKI experienced more renal infarction, which was an indicator of poor outcomes. More recently, Imasaka, *et al.*,^[15] confirmed that preoperative estimated glomerular filtration rate was useful in predicting the need for postoperative renal replacement therapy for type A AAD, whereas it was not an effective predictor for in-hospital mortality. In our study, the in-hospital mortality tended to be higher in patients with AKI compared to no AKI, but with no significant difference. These results may be partly explained by the fact that various complications or surgery-related factors affect in-hospital outcomes.

After adjusting for baseline variables, multivariate analyses showed that male gender, diastolic BP and bilateral renal artery involvement were independent predictors of AKI. Of the AKI patients in our study, males far outnumbered females, which was similar to those of previous studies.^[11,12,14,20] Reduction of renal blood flow, whether generalized or localized, plays a pivotal role in the pathophysiology of AKI.^[21] Lower diastolic BP on admission was associated with reduced visceral perfusion, causing generalized ischemia to the kidney leading to AKI. Extension of the dissection may involve the renal artery causing thrombosis or stenosis, which may directly impair renal perfusion, thus resulting in AKI.^[22] Obviously, bilateral renal artery involvement was more harmful. However, in the setting of acute dissection, strategies to improve renal perfusion (e.g., increasing the baseline BP or renal replacement therapy) are not feasible and may delay the timing of surgery. In this case, prompt surgery is needed to inhibit extension of the dissection and improve renal perfusion. Previous report showed that a long cardiopulmonary bypass duration (> 180 min) was a strong risk factor for postoperative AKI.^[12] Therefore, shorter cardiopulmonary bypass time should be considered to prevent further renal impairment and improve in-hospital outcomes.

There are several potential limitations. First, our study was a single-center retrospective study, which might include a non-homogeneous population. Second, although each group's sample size is relatively small, statistical significances were observed between AKI and no-AKI groups regarding in-hospital complications. However, the statistical power may not be sufficient for comparison among different AKI stages due to the relatively small number of patients. Third, patients with AKI were classified only by SCr levels, which

may not be the most sensitive marker. Urine output changes were not available for this retrospective study. Several markers of AKI (i.e., Cystatin C, N-acetyl-beta-D-glucosaminidase, neutrophil gelatinase-associated lipocalin, etc) should be considered and allow a more precise diagnosis and earlier management of AKI in the future study. Fourth, patients with deaths before admission were not included in this study, and some patients abandoned treatment and were automatically discharged from the hospital due to various reasons. Thus, the in-hospital mortality may be underestimated.

In conclusion, early AKI before open surgery was common in patients with type A AAD. Preoperative AKI was associated with increased in-hospital complications. Furthermore, male gender, diastolic BP on admission and bilateral renal artery involvement were independent predictors for preoperative AKI.

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References

- 1 Sheikh AS, Ali K, Mazhar S. Acute aortic syndrome. *Circulation* 2013; 128: 1122–1127.
- 2 Clough RE, Nienaber CA. Management of acute aortic syndrome. *Nat Rev Cardiol* 2015; 12: 103–114.
- 3 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.
- 4 Trimarchi S, Nienaber CA, Rampoldi V, *et al.* Contemporary results of surgery in acute type A aortic dissection: The international registry of acute aortic dissection experience. *J Thorac Cardiovasc Surg* 2005; 129: 112–122.
- 5 Mehta RH, Suzuki T, Hagan PG, *et al.* Predicting death in patients with acute type A aortic dissection. *Circulation* 2002; 105: 200–206.
- 6 Apaydin AZ, Buket S, Posacioglu H, *et al.* Perioperative risk factors for mortality in patients with acute type A aortic dissection. *Ann Thorac Surg* 2002; 74: 2034–2039.
- 7 Tan ME, Kelder JC, Morshuis WJ, *et al.* Risk stratification in acute type a dissection: proposition for a new scoring system. *Ann Thorac Surg* 2001; 72: 2065–2069.
- 8 Hagan PG, Nienaber CA, Isselbacher EM, *et al.* The international registry of acute aortic dissection (IRAD): New insights into an old disease. *JAMA* 2000; 283: 897–903.
- 9 Safi HJ, Miller CC 3rd, Reardon MJ, *et al.* Operation for acute and chronic aortic dissection: recent outcome with regard to

- neurologic deficit and early death. *Ann Thorac Surg* 1998; 66: 402–411.
- 10 Fann JI, Smith JA, Miller DC, *et al.* Surgical management of aortic dissection during a 30-year period. *Circulation* 1995; 92: III13–III21.
 - 11 Tsai HS, Tsai FC, Chen YC, *et al.* Impact of acute kidney injury on one-year survival after surgery for aortic dissection. *Ann Thorac Surg* 2012; 94: 1407–1412.
 - 12 Roh GU, Lee JW, Nam SB, *et al.* Incidence and risk factors of acute kidney injury after thoracic aortic surgery for acute dissection. *Ann Thorac Surg* 2012; 94: 766–771.
 - 13 Piffaretti G, Mariscalco G, Bonardelli S, *et al.* Predictors and outcomes of acute kidney injury after thoracic aortic endograft repair. *J Vasc Surg* 2012; 56: 1527–1534.
 - 14 Ko T, Higashitani M, Sato A, *et al.* Impact of acute kidney injury on early to long-term outcomes in patients who underwent surgery for type a acute aortic dissection. *Am J Cardiol* 2015; 116: 463–468.
 - 15 Imasaka K, Tayama E, Tomita Y. Preoperative renal function and surgical outcomes in patients with acute type A aortic dissection. *Interact Cardiovasc Thorac Surg* 2015; 20: 470–476.
 - 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* 2012; 2: 1–138.
 - 18 Hobson CE, Yavas S, Segal MS, *et al.* Acute kidney injury is associated with increased long-term mortality after cardiothoracic surgery. *Circulation* 2009; 119: 2444–2453.
 - 19 Arnaoutakis GJ, Bihorac A, Martin TD, *et al.* Rife criteria for acute kidney injury in aortic arch surgery. *J Thorac Cardiovasc Surg* 2007; 134: 1554–1560.
 - 20 Takahashi T, Hasegawa T, Hirata N, *et al.* Impact of acute kidney injury on in-hospital outcomes in patients with debakey type iii acute aortic dissection. *Am J Cardiol* 2014; 113: 1904–1910.
 - 21 Bonventre JV, Yang L. Cellular pathophysiology of ischemic acute kidney injury. *J Clin Invest* 2011; 121: 4210–4221.
 - 22 Ren HM, Wang X, Hu CY, *et al.* Relationship between acute kidney injury before thoracic endovascular aneurysm repair and in-hospital outcomes in patients with type B acute aortic dissection. *J Geriatr Cardiol* 2015; 12: 232–238.