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ORIGINAL ARTICLE

Incidence, risk factors and clinical outcomes for acute kidney injury after aortic arch repair in paediatric patients

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Abstract

OBJECTIVES: Acute kidney injury (AKI) is common after paediatric cardiac surgery and associated with increased morbidity and mortality. Aortic arch surgery may be an independent risk factor for AKI because of circulatory arrest below the descending thoracic artery during anastomosis. We investigated the surgical outcomes associated with AKI after aortic arch repair in paediatric patients.

METHODS: We retrospectively analysed 120 paediatric patients who underwent aortic arch repair between 2003 and 2012. AKI was defined according to the paediatric-modified risk, injury, failure, loss and end-stage kidney disease criteria. The incidence, clinical outcomes and risk factors for AKI were analysed.

RESULTS: Aortic arch repair was performed for coarctation of aorta in 97 patients and interrupted aortic arch in 23 patients. The median age and body weight at the time of surgery were 16.5 days and 3.3 kg, respectively. The mean duration of the follow-up was 3.9 years. AKI developed in 42 patients (36.8%) and peritoneal dialysis (PD) was applied in 20 patients (16.7%). The recovery of renal function began a mean of 3.8 days after conservative management, and full recovery occurred a mean of 6.7 days after conservative management. A lower body weight (<3.0 kg) (odds ratio [OR]: 7.569, $P = 0.009$) and the absence of prerenal impairment (OR: 9.876, $P = 0.041$) were shown to be independent risk factors. Patients who required PD had prolonged intensive care unit and hospital stays ($P = 0.002$ and $P = 0.003$).

CONCLUSIONS: AKI is common in low-birth-weight patients after aortic arch repair surgery. However, patients recover from AKI after conservative management. Requiring PD increases the morbidity associated with AKI.

Keywords: Acute kidney injury • Aortic arch surgery • Congenital heart disease • Prognosis

INTRODUCTION

Acute kidney injury (AKI) is common after cardiac surgery and is associated with increased mortality [1–5], prolonged hospital stays [6, 7] and progression to chronic kidney disease [8]. While the detection of AKI can be obscure and difficult in children, the modified risk, injury, failure, loss and end-stage kidney disease (RIFLE) criteria, which are based on changes in estimated creatinine clearance (eCCr) and/or urine output, were, therefore, created and have been validated to identify AKI development in paediatric patients [9]. The incidence of AKI after paediatric cardiac surgery has been reported as 11–52% in patients with heterogeneous diagnoses [2, 4, 6, 7, 10, 11]. Elevated preoperative serum creatinine levels, an age of less than 1 year, the presence of cyanotic lesions, a prolonged cardiopulmonary bypass time and postoperative low-cardiac-output syndrome have been reported as risk factors for AKI after paediatric cardiac surgery [3, 12].

Although the reported incidence (based on the RIFLE criteria) of AKI after thoracic aortic surgery in adult patients varies from 18 to 55% [13, 14], there are limited data regarding AKI after aortic arch surgery in paediatric patients. Since the circulatory arrest below the descending thoracic artery cannot be avoided during anastomosis, aortic arch repair may be an independent risk factor for AKI in paediatric patients. However, AKI has not been carefully monitored after aortic arch repair in paediatric patients. Thus, we investigated the incidence, clinical outcomes and risk factors for AKI after aortic arch repair in paediatric patients.

MATERIALS AND METHODS

Study population

The study protocol was approved by the Institutional Review Board of the University (No. H-1305-602-492). The procedures

were performed in accordance with the institutional guidelines for the protection of patient confidentiality. Patient consent was waived due to the retrospective nature of the study.

We retrospectively reviewed paediatric patients who underwent aortic arch repair from September 2003 to December 2012. After exclusion of 2 patients who had single-ventricle physiology due to haemodynamic difference, 120 patients (55 males and 65 females) were studied.

Acute kidney injury assessment

AKI was defined as a decrease in eCCr for more than 2 continuous days after surgery based on the pRIFLE criteria (Table 1). To calculate the eCCr, the following updated Schwartz formula was used [15]:

$$\text{eCCr} = 0.413 \times \text{height (cm)/serum creatinine.}$$

We chose to only estimate the eCCr using the pRIFLE criteria. The urine output criteria were not used to determine the postoperative AKI because variations in postoperative haemodynamics and diuretic doses might influence the patients' urine output. In addition, we defined postoperative AKI as a decrease in creatinine clearance for more than 2 continuous days to exclude patients with prerenal AKI, which could be corrected by hydration. Creatinine levels were obtained for 3 days during preoperative to postoperative intensive care unit (ICU) stays. We also recorded the peak creatinine and nadir eCCr levels during the ICU stay. If a patient showed evidence of AKI, we continued measuring the creatinine levels until they normalized.

Risk factor assessment

Demographic variables including age, sex and surgical status (emergency or elective surgery), as well as laboratory variables including preoperative urine analysis, preoperative and postoperative creatinine levels (up to 3 days) and the use of preoperative antibiotics were recorded. Surgery-related variables, including cardiopulmonary bypass (CPB) time, aorta cross clamp time, body temperature during regional perfusion and regional perfusion time; perioperative haemodynamic variables, including vasoactive-inotropic score (VIS); and postoperative variables, including the duration of mechanical ventilation, hospital stay, ICU stay, the initiation of peritoneal dialysis, early and late mortality and postoperative complications, were recorded.

Clinical variable definitions

The hourly doses of vasoactive medications were recorded for the first 3 days after admission to the postoperative ICU. We calculated the VIS using a formula modified by Gaies *et al.* [16]. The maximum VIS on each day was noted for 3 days.

The VIS was calculated as follows:

$$\begin{aligned}\text{VIS} = {} & \text{dopamine dose } (\mu\text{g/kg/min}) \\ & + \text{dobutamine dose } (\mu\text{g/kg/min}) \\ & + 100 \times \text{epinephrine dose } (\mu\text{g/kg/min}) \\ & + 10 \times \text{milrinone dose } (\mu\text{g/kg/min}) \\ & + 10\,000 \times \text{vasopressin dose (U/kg/min)} \\ & + 100 \times \text{norepinephrine dose } (\mu\text{g/kg/min}) \\ & + 10 \times \text{ phenylephrine dose } (\mu\text{g/kg/min}).\end{aligned}$$

We investigated the postoperative VIS based on the patient's risk factors for AKI.

Preoperative renal impairment was defined as decreased eCCr compared with the normal range (which varied according to age) (Table 2). Preoperative urine was analysed by dipstick (Siemens Multistix, Tarrytown, NY, USA) to identify any unpredicted renal problems. Preoperative aminoglycoside antibiotic use was defined as use for more than 3 continuous days before surgery. Preoperative systemic ventricle dysfunction was defined as an ejection fraction <45% measured by preoperative echocardiography. Early mortality was defined as death within 30 days of surgery. We calculated the fluid balance index during 3 days of the ICU stay using the following formula:

$$\begin{aligned}\text{fluid balance index} = {} & \text{fluid input} \\ & - \text{output (ml)/body surface area } (\text{m}^2)\end{aligned}$$

Early mortality was defined as death related to the operation within 30 days.

Surgical procedures

After body temperature probe insertion into the rectum, full median sternotomy was performed, and then, an arterial cannula was inserted directly through the innominate artery and standard bicaval cannulation was applied. In cases of ductal-dependent

Table 1: Paediatric-modified (pRIFLE) criteria for acute kidney injury

	Estimated CCr	Urine output
Risk	eCCr decrease by 25%	<0.5 ml/kg/h for 8 h
Injury	eCCr decrease by 50%	<0.5 ml/kg/h for 16 h
Failure	eCCr decrease by 75% or eCCr <35 ml/min/1.73 m^2	<0.3 ml/kg/h for 24 h or anuria for 12 h
Loss	Persistent failure >4 weeks	
End stage	End-stage renal disease (persistent failure >3 months)	

eCCr: estimated creatinine clearance; pRIFLE: paediatric risk, injury, failure, loss and end-stage renal disease.

Table 2: Preoperative renal impairment according to age

Age	Preoperative estimated creatinine clearance	Number with prerenal impairment (%)	Total number	Renal impairment cut-off value according to age
<2 weeks	38.6 ± 19.5	20 (33.9)	59	≥30
2–8 weeks	47.6 ± 16.3	17 (44.7)	38	≥45
8 weeks to 24 months	56.8 ± 35.2	10 (66.7)	15	≥65
>24 months	100.9 ± 30.5	1 (14.3)	7	≥90
Total	47.4 ± 26.4	48 (40.3)	119	

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descending thoracic aortic circulation, another arterial cannula was applied through the patent ductus arteriosus (PDA). During cooling, an aortic root cannula was inserted and a T-connection was formed with the side hole of the innominate artery cannula following aortic arch dissection. Regional perfusion, including cerebral and myocardial perfusion, was initiated after the body temperature reached ~28 °C. A distal anastomosis using synthetic monofilament suture material was performed after the distal aorta was clamped [17].

Statistical analysis

Continuous variables are expressed as the means ± standard deviation or as medians with 25th and 75th percentile values. Categorical variables are expressed as frequencies and percentages. A one-way analysis of variance (ANOVA) was used for comparisons among more than three groups. The Bonferrini test with equal variances and the Tamhene test with unequal variances were used as *post hoc* methods. Changes in parameters over time between two groups were compared using a repeated-measures analysis of variance. The Bonferrini test was used as *post hoc* methods in repeated-measures ANOVA. All variables of possible risk factors for AKI were entered into multivariate logistic regression models with deterioration as the dependent variable and a significance level of 0.2 by the enter method. The Hosmer–Lemeshow test was used for goodness of fit for logistic regression. A *P*-value of <0.05 was considered statistically significant. The analyses were performed using the SPSS statistical package (IBM SPSS version 19.0, SPSS, Inc., Chicago, IL, USA).

RESULTS

Clinical outcomes

The median age and body weight at surgery were 16.5 days (9–44.5 days) and 3.3 kg (2.9–4.0 kg). The aortic arch anomalies were coarctation of aorta (CoA) in 97 patients and interrupted aortic arch (IAA) in 23. Ventricular septal defect ($n = 63$) was the most common combined anomaly. We performed an extended end-to-side anastomosis in 64 patients, an extended end-to-end anastomosis in 33 and IAA repair in 22 (Supplementary material, Appendix 1). We performed preoperative computed tomography or angiography using contrast media to evaluate the preoperative status of 54 patients (45.0%). Twenty patients (16.7%) underwent emergency surgery due to haemodynamic instability. The mean CPB time and aorta cross-clamp time were 156.3 ± 51.4 min and 51.8 ± 34.4 min, respectively. The mean thoracic aorta clamping time was 23.9 ± 6.8 min. The mean body temperatures during CPB and regional perfusion were 24.1 ± 3.0°C and 26.0 ± 2.5°C, respectively. The median ventilation duration was 107.1 h (range: 72.5–141.1 h). The median ICU stay and the median hospital stay were 7.6 days (range: 4.7–9.7 days) and 14.0 days (range: 10.0–21.0 days), respectively. The mean follow-up duration was 3.9 ± 2.7 years (range: 0.0 years–9.0 years).

There was 1 early death and 2 late deaths. The early mortality case was a female patient who developed AKI-I and had severe coarctation of aorta and double-outlet right ventricle (Fallot type) with diffuse aortic wall thickening and arch vessel hypoplasia. The aortic wall of this patient exhibited diffuse thickening; therefore, we speculated that elastin arteriopathy was present. The patient died due to the dysfunction of both ventricles 7 days after surgery. One of the 2 late mortality cases was a female patient who developed AKI-R and had a type B IAA with ventricular septal defect (VSD). This patient exhibited clinical features of the Zellweger syndrome and died from hepatic failure caused by the Zellweger syndrome 2 months after surgery. The other late mortality case was a female patient who had type A IAA with ventricular septal defect but did not have AKI. This patient also had the Alagille syndrome (arteriohepatic dysplasia), which caused the hepatic failure. She died due to gastrointestinal bleeding 4 months after surgery. There were also 11 surgery-related morbidities in 10 patients, as follows: bronchial stenosis requiring aortopexy in 4 patients, vocal cord palsy in 2, chylothorax in 2, postoperative temporary seizure in 1, diaphragmatic palsy in 1 and mediastinitis in 1.

Acute kidney injury analysis

Intraoperative and postoperative acute kidney injury analyses. We excluded 6 patients from the analysis of postoperative AKI due to insufficient creatinine data. Among 114 patients, 42 (42/114, 36.8%) exhibited AKI during the ICU stay, including 29 patients (29/114, 25.4%) who were classified as AKI-R and 13 patients (13/114, 11.4%) who were classified as AKI-I. The eCCr values during 3 days of the ICU stay exhibited a significant difference among the three groups (no-AKI group vs AKI-R, $P = 0.026$; no-AKI group vs AKI-I, $P = 0.04$ and AKI-R vs AKI-I, $P = 0.265$) (Fig. 1). All of the patients younger than 24 months developed postoperative AKI-R. Moreover, all of the patients younger than 8 weeks developed postoperative AKI-I. The eCCr values during 3 days of the ICU were decreased over time in all groups (all $P < 0.001$). The decrease in eCCr began 0.9 ± 0.7 days after surgery in the AKI-R group and 0.9 ± 0.8 days after surgery in the AKI-I group. The recovery of renal function in the AKI-I group began a mean of 3.8 ± 0.9 days after surgery, and the eCCr values fully recovered to the preoperative level 6.7 ± 1.7 days after conservative management.

The AKI-I and AKI-R groups had a tendency towards a prolonged hospital stay compared with the no-AKI group ($P = 0.099$, $P = 0.057$, respectively). However, the ICU stay ($P = 0.921$, $P = 0.798$) and the mechanical ventilation duration ($P = 0.230$, $P = 0.381$) did not show a significant difference compared with the no-AKI group

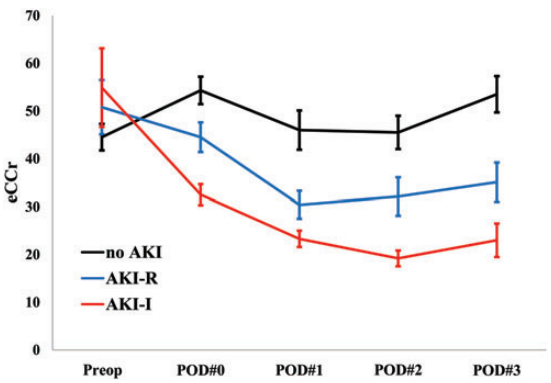

Figure 1: eCCr levels during postoperative 3 days among the three groups. The AKI-I group had a significant decrease in eCCr during the postoperative period. Error bar means ± standard error of mean. AKI: acute kidney injury; AKI-I: acute kidney injury-injury; AKI-R: acute kidney injury-risk; eCCr: estimated creatinine clearance; Preop: preoperative; POD: postoperative day.

in the AKI-I and AKI-R groups. The rates of in-hospital death and late death did not differ significantly among the three groups ($P = 0.110$ and $P = 0.627$, respectively). In addition, the CPB time ($P = 0.597$), aorta cross clamp time ($P = 0.338$) and regional perfusion time ($P = 0.507$) were not significantly different among the three groups (Table 3). The actuarial 5-year survival rates were 97.9 ± 2.1% in the no-AKI group, 96.0 ± 3.9% in the AKI-R group and 92.3 ± 7.4% in the AKI-I group, with no significant difference among the three groups ($P = 0.399$).

Preoperative renal impairment analysis

Proteinuria was observed in 2 patients (2/56, 3.6%) and hematuria in 2 patients (2/59, 3.4%) in the preoperative urine analysis by dipstick. Preoperative renal impairment was detected in 48 patients (40.3%). The patients younger than 24 months showed a high incidence of preoperative renal impairment. The incidence of preoperative renal impairment according to age is given in Table 2. The mean preoperative creatinine and eCCr values were 0.6 ± 0.3 ($n = 120$) and 47.4 ± 26.4 ($n = 120$), respectively. Preoperative aminoglycoside antibiotic use significantly increased the incidence of AKI ($P = 0.017$). The preoperative creatinine levels were significantly decreased in the group of patients with no AKI compared with the group with AKI, and there was an inverse correlation between preoperative creatinine and preoperative clinical characteristics ($P = 0.012$). The other preoperative clinical characteristics did not exhibit any significant differences between the groups (Table 4).

Fluid balance and peritoneal dialysis treatment analysis

We recorded and calculated fluid balance index of 101 patients (84.2%). There was no significant difference in fluid balance index among the three groups ($P = 0.717$) (Table 3). We performed PD on 20 patients (16.7%) in the postoperative ICU. The median PD initiation time was 9.4 h (0.5–40.1 h) after ICU admission, and the mean PD duration was 1.1 ± 0.8 days. The patients who required PD had a significantly higher incidence of AKI [14 patients, (AKI-R: $n = 9$, 31.0%; AKI-I: $n = 5$, 38.5%)] than those who did not require PD ($n = 6$, 8.3%) ($P = 0.003$). Fourteen of the patients who required PD (among the 42 AKI patients) exhibited significant decreases in eCCr during the 3-day ICU stay compared with those who did not require PD ($P = 0.029$). Furthermore, PD treatment did not shorten the eCCr recovery period in the ICU (postoperative day 0 [POD 0]: 10.2 ± 4.9 (mean difference ± standard of error); POD 1: 9.6 ± 4.3; POD 2: 11.4 ± 6.1 and POD 3: 13.5 ± 6.4) (Fig. 2). The patients who required PD therapy had longer stays in the ICU [12.0 ± 7.7 ($n = 21$) vs 7.4 ± 6.5 ($n = 99$), $P = 0.002$] and in the hospital (24.0 ± 12.5 [$n = 21$] vs 15.7 ± 11.2 [$n = 99$], $P = 0.003$) compared with those who did not require PD therapy. There was no correlation between the need for PD and the presence of preoperative renal impairment ($P = 0.84$).

Vasoactive-inotropic score

The hourly vasoactive inotrope doses of 114 patients (95.0%) were recorded for 3 days in the ICU, and the maximal VIS scores were calculated (Table 4). The AKI-I group had the highest VIS score compared with the AKI-R and no AKI groups to maintain stable haemodynamic vital signs during the 3-day postoperative ICU stay ($P = 0.001$) (Fig. 3).

Risk factor analysis

According to the univariate analysis, the factors that were independently associated with the development of AKI-I were a body weight at surgery of <3 kg, the absence of prerenal impairment

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Table 3: Preoperative patient characteristics according to pRIFLE criteria in patients with AKI

Characteristics	No-AKI	AKI-R	AKI-I	*P*-value
Number	72	29	13	
Age at surgery (months)	11.5 ± 35.9	1.2 ± 2.6	0.4 ± 0.3	0.174
<2 weeks, *n* (%)	35 (48.6)	13 (44.8)	10 (76.9)	0.442
2 weeks to 8 weeks, *n* (%)	19 (26.4)	13 (44.8)	3 (23.1)	0.440
8 weeks–24 months, *n* (%)	12 (16.7)	3 (10.3)	0 (0)	0.768
>24 months, *n* (%)	6 (8.3)	0 (0)	0 (0)	
Sex (M/F)	40/32	15/14	7/6	0.940
Body weight (kg), mean ± SD	6.2 ± 9.0	3.6 ± 2.1	2.6 ± 0.7	0.12
Preoperative contrast use, *n* (%)	37 (51.4)	12 (41.4)	5 (38.5)	0.523
Preoperative aminoglycoside ≥3 days	5 (8.2)	4 (17.4)	5 (38.5)	0.017[a]
Emergency surgery, *n* (%)	13 (18.1)	3 (10.3)	3 (23.1)	0.517
Preoperative left ventricle dysfunction, *n* (%)	9 (12.5)	3 (10.3)	3 (23.1)	0.534
VSD, *n* (%)	46 (63.9)	19 (65.5)	10 (76.9)	0.660
Preoperative kidney assessment				
Preoperative renal impairment, *n* (%)	36 (50)	10 (34.5)	1 (7.7)	0.012[a]
Preoperative creatinine	0.6 ± 0.3	0.5 ± 0.2	0.4 ± 0.2	0.012[a]
Preoperative eCCr (ml/min/1.73 m^2)	44.5 ± 23.5	50.8 ± 30.2	54.9 ± 29.7	0.298
Hematuria, *n* (%)	2 (6.9)	0	0	0.346
Proteinuria, *n* (%)	2 (6.7)	0	0	0.329

AKI: acute kidney injury; AKI-R: acute kidney injury-Risk; AKI-I: acute kidney injury-Injury; eCCr: estimated creatinine clearance; SD: standard deviation; VSD: ventricular septal defect.
[a]Significant difference among the groups.

Table 4: Intraoperative and postoperative patient assessments according to pRIFLE criteria

Characteristics	No AKI	AKI-R	AKI-I	*P*-value
Intraoperative assessment				
CPB time, min (*n*)	155.3 ± 52.2 (67)	166.2 ± 54.0 (27)	152.2 ± 42.3 (13)	0.597
ACC time, min (*n*)	57.7 ± 32.7 (60)	57.2 ± 36.3 (26)	43.1 ± 24.7 (13)	0.338
RP time, min (*n*)	23.6 ± 5.1 (64)	22.5 ± 6.7 (27)	24.6 ± 6.8 (13)	0.507
Lowest BT, °C (*n*)	24.0 ± 3.2 (64)	23.9 ± 3.1 (27)	24.7 ± 1.9 (13)	0.701
BT at RP, °C (*n*)	25.8 ± 3.3 (65)	26.0 ± 2.5 (27)	26.9 ± 2.1 (13)	0.510
CPB input/output (*n*)	−113.9 ± 158.7 (64)	−158.1 ± 134.9 (28)	−62.7 ± 60.8 (13)	0.133
Postoperative assessment				
AG antibiotic use, *n* (%)	18 (25.0)	9 (31.0)	5 (38.5)	0.561
Patient required PD, *n* (%)	6 (8.3)	9 (31.0)	5 (38.5)	0.003
MV time, h (*n*)	109.7 ± 88.6 (72)	127.0 ± 82.7 (27)	140.2 ± 45.3 (13)	0.382
ICU stay, days (*n*)	11.3 ± 34.8 (72)	9.6 ± 6.7 (27)	12.3 ± 10.5 (13)	0.950
Hospital stay, days (*n*)	15.3 ± 9.1 (72)	20.1 ± 15.2 (27)	22.7 ± 14.6 (13)	0.040
In-hospital death, *n* (%)	0 (0)	0 (0)	1 (7.7)	0.110
Late death, *n* (%)	1 (1.4)	1 (3.4)	0 (0)	0.627
eCCr				
Preoperative	44.5 ± 23.5 (72)	50.8 ± 30.2 (28)	54.9 ± 29.7 (13)	0.298
POD 0 mean ± SD (*n*)	54.3 ± 24.3 (72)	44.5 ± 16.7 (29)	32.5 ± 7.9 (13)	0.002
POD 1	46.0 ± 34.8 (72)	30.4 ± 15.7 (29)	23.3 ± 6.2 (13)	0.007
POD 2	45.5 ± 28.5 (67)	32.1 ± 20.8 (27)	19.2 ± 5.7 (12)	0.001
POD 3	53.5 ± .29.9 (62)	35.1 ± 21.6 (28)	23.0 ± 12.7 (13)	<0.001
VIS score, *n*	72	29	13	
POD 0	13.3 ± 6.8	15.5 ± 5.8	22.5 ± 15.7	0.001
POD 1	11.3 ± 6.5	16.4 ± 10.6	20.3 ± 10.1	<0.001
POD 2	9.3 ± 5.6	11.0 ± 4.1	14.6 ± 9.4	0.09
POD 3	7.6 ± 5.0	8.4 ± 3.3	11.0 ± 10.2	0.125
Fluid balance index				
POD 0, mean ± SD (*n*)	−146.0 ± 656.1 (69)	−1690 ± 652.1 (29)	715.1 ± 663.9 (13)	<0.001
POD 1	128.9 ± 486.9 (70)	229.0 ± 675.5 (29)	145.5 ± 778.1 (13)	0.734
POD 2	−258.9 ± 497.2 (65)	−335.8 ± 557.9 (29)	−740.5 ± 420.4 (13)	0.009
POD 3	−299.9 ± 509.2 (62)	−315.0 ± 453.5 (27)	−404.2 ± 392.7 (13)	0.778

ACC: aortic cross clamp; AG: aminoglycoside; AKI: acute kidney injury; AKI-R: acute kidney injury-risk; AKI-I: acute kidney injury-injury; BT: body temperature; CPB: cardiopulmonary bypass; eCCr: estimated creatinine clearance; ICU: intensive care unit; MV: mechanical ventilation; PD: peritoneal dialysis; POD: postoperative day; RP: regional perfusion; SD: standard deviation; VIS: vasoactive-inotropic score.

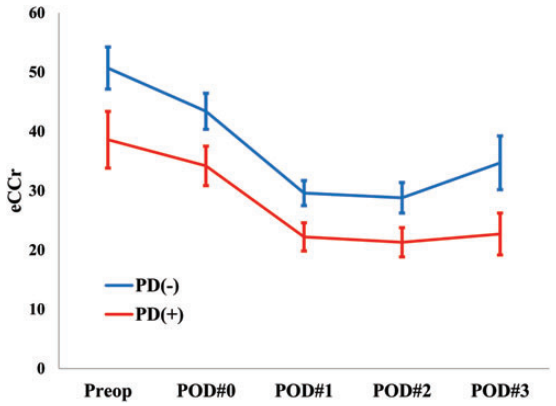

Figure 2: eCCr levels during the postoperative period. The patients who required PD had a significant decrease in eCCr compared with the patients who did not require PD (among the patients with AKI). Error bar means ± Standard error of mean. AKI: acute kidney injury; eCCr: estimated creatinine clearance; PD: peritoneal dialysis; Preop: preoperative; POD: postoperative day.

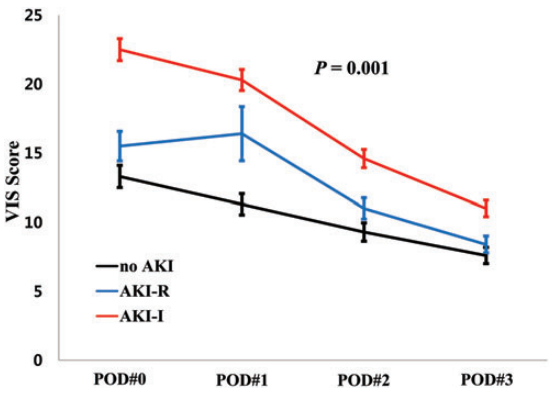

Figure 3: VIS scores according to group. The VIS was increased in the AKI-I group compared with the other groups to maintain the appropriate haemodynamics. Error bar means ± standard error of mean. AKI: acute kidney injury; AKI-I: acute kidney injury-injury; AKI-R: acute kidney injury-risk; eCCr: estimated creatinine clearance; Preop: preoperative; POD: postoperative day; VIS: vasoactive-inotropic score.

and preoperative aminoglycoside antibiotic use for more than 3 days. A body weight at surgery of <3 kg and the absence of prerenal impairment were also associated with the development of AKI-I in the multivariate analysis (Table 5). However, we did not find any risk factors that were significantly associated with preoperative renal impairment.

Table 5: Univariate and multivariate analyses of risk factors for acute kidney injury according to the pRIFLE-I criteria compared with no acute kidney injury

Variables	Univariate analysis			Multivariate analysis		
	OR	95% CI	*P*-value	OR	95% CI	*P*-value
Sex						
Male	1					
Female	1.071	0.327–3.506	0.909			
Age						
<2 weeks	1					
2–8 weeks	0.553	0.135–2.254	0.408			
8 weeks to 24 months	0.000		0.999			
>24 months	0.000		0.999			
Body weight						
<3 kg	7.889	2.027–30.705	0.003	7.569	1.64–34.93	0.009
>3 kg	1			1		
Preoperative aminoglycoside use	5.208	1.4–19.38	0.014	3.464	0.680–17.65	0.135
Preoperative contrast	0.591	0.176–1.981	0.394			
Preoperative left ventricle dysfunction	2.067	0.476–8.966	0.332			
Emergency surgery	1.362	0.328–5.650	0.671			
Cardiopulmonary bypass time	0.999	0.987–1.011	0.838			
Aortic cross-clamp time	0.981	0.957–1.006	0.136	0.988		0.394
No prerenal impairment	10.036	1.257–80.114	0.030	9.876	0.96–1.02	0.041
Aminoglycoside	1.875	0.544–6.467	0.320		1.10–89.05	

CI: confidence interval; OR: odds ratio.

DISCUSSION

In this study, we found that AKI after aortic arch surgery using a strategy of cerebral regional perfusion and distal descending thoracic aortic clamping during anastomosis was relatively common in paediatric patients, with an incidence of 37.8% according to the pRIFLE criteria. However, these paediatric patients recovered from AKI within 7 days of conservative management.

Albabas *et al.* [18] reported that 75% of patients developed postoperative AKI within 48 h of ICU admission. Among these patients, 43% received renal replacement therapy for a median duration of 4 days. In addition, Aydin *et al.* [6] reported that 85% of patients exhibited evidence of AKI within 24 h of admission to the ICU, and the duration was <48 h in 75% of these patients. In this study, the decline in renal function began a mean 0.9 days after surgery, renal function began to recover ∼4 days after surgery in the patients who developed AKI, and renal function recovered fully ∼7 days after surgery with conservative management.

There have been several reports that a long CPB duration was related to AKI incidence [1, 6, 9]. The patients in these studies had heterogeneous diagnoses, and the group with a high incidence of AKI had a higher risk-adjusted classification for congenital heart surgery (RACHS-1) score; thus, unstable haemodynamics and a longer CPB time may be associated with AKI incidence. We also identified that the AKI-I group required high doses of vasoactive inotropic medications to maintain the appropriate blood pressure levels in this study. However, the disease categories in the present work were relatively homogeneous compared with the above studies, although there were diverse combined operations. Therefore, the CPB time, body temperature during CPB and postoperative ICU haemodynamics were relatively similar among our patients, and we did not find significant differences in those parameters in this study. There have been several reports that moderate hypothermia for circulatory arrest is safe, effective for organ protection and not associated with AKI [19, 20]. However, we usually selected moderate degrees of hypothermia during descending thoracic aortic clamping, and we demonstrated that moderate hypothermia itself was not sufficient to prevent postoperative AKI. Our strategy in paediatric aortic arch surgery includes myocardial and cerebral perfusion during anastomosis with descending thoracic aortic clamping during moderate hypothermia [17]. Imoto *et al.* [21] reported inserting an additional cannula into the descending thoracic aorta for lower body perfusion; the postoperative renal function was tolerable. However, although the development of AKI during aortic arch surgery, due to total circulatory arrest during anastomosis, was inevitable in this study, we found that kidney function could be recovered with conservative management. Thus, additional descending thoracic aortic perfusion may not be necessary. Neither age nor the degree of AKI was associated with early or late mortality in this study, in contrast to other reports that AKI after cardiac surgery increased both early and late mortality [1, 2]. The incidences of early and late mortality after paediatric aortic arch surgery were low in this study therefore, we suspect that AKI did not influence mortality.

Coarctation of aorta and IAA were representative diagnoses in this study. These diagnoses can be affected by kidney function during the preoperative period due to the decreased blood flow to the kidney caused by severe coarctation of aorta and the decreased PDA flow and left ventricle dysfunction caused by pressure loading at the aortic arch level (although this study identified no risk factors for prerenal impairment). Interestingly, we found that patients with prerenal impairment had a low incidence of postoperative AKI during the ICU stay, in contrast to other reports [22, 23]. Although there is a wide spectrum of prerenal impairment that is associated with many compensatory mechanisms in the potential reversibility of the condition, we suspect that decreased blood flow to the kidney may have caused the preconditioning to the deconditioning that the incidence of AKI after

aortic arch repair may have decreased inversely [24, 25]. We also suspect that a preoperative decrease in the renal creatinine clearance level may make it difficult to achieve a >50% decrease in creatinine clearance postoperatively.

CONCLUSIONS

Using the pRIFLE criteria, AKI incidence after aortic arch surgery reached 35% in paediatric patients in this study. However, the vast majority of patients recovered within 7 days of conservative management. The patients who developed AKI and required PD treatment experienced prolonged stays in the ICU and in the hospital. A body weight of <3 kg at surgery was a risk factor for AKI development after aortic arch repair in paediatric patients.

Study limitations

This was a retrospective and single-centre study. We also did not examine the patients to evaluate their most recent level of kidney function. Although the AKI group recovered kidney function, further follow-up may be required to assess long-term outcomes.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *EJCTS* online.

Conflict of interest: none declared.

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