ORIGINAL ARTICLE

Acute Kidney Injury Following Paediatric Cardiac Surgery: Experience of A Paediatric Cardiac Center of Bangladesh

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Abstract

Background: *Acute kidney injury following cardiac surgery in children is a relatively common phenomenon. The risk ranges from 15% to 64% with a mortality rate of 10% to 89% for those who need dialysis.*

Objective: *To identify the occurrence of acute kidney injury following cardiac surgery in children, as well as its consequences and treatment options.*

Methods: *This study was conducted in the pediatric cardiac recovery center at Bangladesh Shishu Hospital & Institute from January 2019 to December 2020. Using the KDIGO (kidney disease improving global outcome) criteria, 50 children having cardiac surgery were assessed for the occurrence of AKI. Children were divided into two groups. The AKI group consists of 20 patients, whereas the non-AKI group consists of 30 patients. Age, sex, cardiac illness, past operations, RACHS-1 (Risk adjustment for congenital Heart surgery) and pre and post-operative creatinine levels were noted. Fisher's exact test was used to determine whether or not AKI was associated with categorical factors, while the Mann-Whitney U-test was used to see whether or not AKI was associated with continuous variables.*

Results: *Mean age of study population was 4.50±4.03 years for those who did not develop AKI and 7.55±5.79 years for those who did. Male constituted a majority in both groups. Despite the fact that patients with AKI were more acidotic at baseline, lower bicarbonate levels were not linked to an increased risk of AKI (p=0.89). Neither group had higher or lower pressure than the other. Time spent in bypass and cross-clamping was comparable across AKI and non-AKI patients. Patients with AKI did not have a higher risk of developing extracellular fluid excess than non-AKI patients. There was no significant difference between the groups in terms of the average time spent on mechanical ventilation or total hospital stay in days (p=0.17 and p=0.62, respectively). Duration of stay in the cardiac recovery increased for patients who had AKI (p=0.02). The mortality rate among the AKI patients was 20% (4/14). In the control group without AKI, there was one death (3.33%) (p 0.0001).*

Conclusion: *AKI is common complication of paediatric cardiac surgery. Prolong times spent on mechanical ventilation in the ICU and in the hospital have all been linked to AKI.*

Keywords: *Acute kidney injury (AKI), cardiac injury, cardiac surgery.*

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Introduction

After cardiac surgery, children had a 15-64% chance of developing AKI, with a 10-89% mortality rate among dialysis-dependent patients.[1-2] Acute kidney injury is associated with a worse hospital course an increased risk of infections and a higher death rate (AKI).[3] Several studies have shown variable rates of AKI prevalence, which is likely due to the syndrome being defined in different ways. The two most used classification schemes are the Risk Injury Failure Loss End-stage renal disease (RIFLE) system developed by the Acute Kidney Injury Network and the pediatric modified version of this system, pRIFLE.[4] The most up-to-date and commonly recognized definition of kidney disease is the one endorsed by the Kidney Disease: Improving Global Outcomes group. One of the main challenges of treating AKI is that it is not detected until 24-48 hours after the first insult, when the creatinine level has already increased by 50%. This means that patients undergoing heart surgery sometimes go unnoticed for days after they have suffered from AKI.[5]

Various studies have used alternative serum and urine indicators for the early detection of AKI. Serum cystatin-C and urine NGAL (Neutrophil gelatinase associated lipocalin) are two that have gained attention as potential functional indicators.[6] Hence, cystatin-C is an effective functional biomarker of damage, since it may identify AKI earlier than creatinine and with greater specificity and sensitivity. Renal ischemia and reperfusion damage, maladaptive inflammatory response, oxidative stress, microemboli, and alterations in tubular cell metabolism are all potential causes of AKI after heart surgery.[7] Despite the fact that several risk factors for AKI have been discovered, only a small number are really under the control of the individual patient. Some studies have distinguished between pre-operative and post-operative complications. In addition to preoperative risk factors including age at repair and surgical complexity, intraoperative risk factors include bypass duration, hypotensive episodes and prolong operation time.[3] Medications that may damage the kidneys, as well as the existence of systemic and wound infections are concerns to think about after surgery.[1] This study was performed to learn more about the causes, effects, and challenges of treating acute renal impairment in children after cardiac surgery. So this study was done to discuss the frequency of acute kidney injury following heart surgery in children, as well as its consequences and treatment options.

Materials and Methods

Fifty children who had heart surgery in 2019 and 2020 were analyzed for the occurrence of AKI according to the KDIGO criteria. The pediatric cardiac recovery center at Bangladesh Shishu Hospital & Institute was where all children were sent after surgery. Children were divided into two groups. The AKI group consists of 20 patients, whereas the non-AKI group consists of 30 patients. Age, sex, cardiac illness, past operations, RACHS-1 (Risk adjustment for congenital Heart surgery) and pre and post-operative creatinine levels were noted. No newborns were included in this analysis. To do descriptive statistics, we used the mean and standard deviation for continuous variables, while counts and percentages were used for categorical ones. Fisher's exact test was used to determine whether or not AKI was associated with categorical factors, while the Mann-Whitney U-test was used to see whether or not AKI was associated with continuous variables.

Results

Demographic distribution of the study subjects shows that in non-AKI group mean age was 4.50±4.03 years whereas in AKI group it was 7.55±5.79. However, in both group majority were male (Table I).

Table I
Demographic distribution

Demographic distribution	Non-AKI N (%)	AKI N (%)	p value
Mean age	4.50 ± 4.03	7.55 ± 5.79	0.03
Gender			
Male	20 (66.67)	15 (75)	0.005
Female	10(33.33)	5(25)	

Thirty percent of patients had repair for tetralogy of Fallot, 20% had VSD closure, 14% had a Fontan operation, 12% had pulmonary valve repair, and 10% had ASD closure with AV canal defect repair 4% (Table II).

Table II
Types of surgery (N=50)

Types of surgery	Percentage
ASD closure	10
VSD closure	20
TOF repair	30
Fontan procedure	14
Pulmonary valve repair	12
Mitral valve repair	10
AV canal defect repair	4

Surgical characteristics of patients shows that bypass time in non-AKI group was 2.52±0.80 mins whereas in AKI group it was 2.75±0.59 mins (Table III).

Table III
Surgical characteristics of patients

Patients surgical status	Non-AKI	AKI	p value
RACHS	2.52 ± 0.80	2.75 ± 0.59	0.12
Bypass time (mins)	131.88 ± 85.64	133.67 ± 34.04	0.24
Cross clamp time (mins)	80.04 ± 45.15	75.30 ± 28.60	0.92

Most patients (52%) had a RACHS-1 score of 2, as seen in Fig.-1.

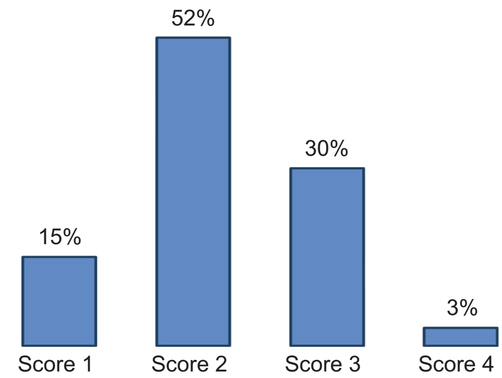

Fig.-1 *RACHS-1 score status*

Although patients with AKI tended to be more acidotic initially, we found no correlation between low bicarbonate levels and an increased risk of AKI. Blood pressure measurements showed no significant differences in terms of mean systolic and diastolic readings across the groups. Our patients with AKI did not have a higher risk of experiencing extracellular fluid overflow than our non-AKI patients, therefore this was not a major concern for them (Table IV).

Table IV
Clinical and biochemical status of patients

Clinical and biochemical status of patients	Non-AKI	AKI	p value
Hemoglobin	15.19 ± 1.46	10.81 ± 1.93	<0.0001
EF			
<60	20 (66.67%)	5(25%)	1.00
>60	10(33.33%)	15 (75%)	
Use of contrast	9 (30%)	3 (15%)	0.20
Creatinine pre-op	0.39 ± 0.15	0.81 ± 1.38	0.31
Bicarbonate	31.45 ± 2.92	20.66 ± 3.15	0.78
SBP	104.40 ± 11.97	106.55 ± 12.35	0.43
DBP	61.69 ± 10.25	58.86 ± 8.41	0.71
Creatinine post-op	0.37 ± 0.14	2.27 ± 1.86	<0.0001
Lactic acid	2.88 ± 1.55	7.00 ± 6.91	0.04

Table V
Management and treatment outcome of the patients

Treatment & outcome	Non-AKI	AKI	p value
Hospital days	9.84 ± 9.79	10.79 ± 7.85	0.62
Ventilation days	1.60 ± 1.92	2.57 ± 2.93	0.17
Length of PICU days	2.56 ± 1.44	4.00 ± 2.66	0.02
Peritoneal dialysis	-	1(5%)	-
Death Rates	1 (3.33%)	4 (20%)	<0.0001

No significant differences were seen in either on mechanical ventilation or days spent in the hospital between the two groups (p=0.17 and 0.62, respectively). A longer PICU stay was seen for individuals with AKI (p = 0.02). Twenty percent (4/14; 4) of the AKI patients that were treated ultimately passed away. When compared to the group without AKI, there was only one mortality (3.33%) (p=0.0001). Of the AKI patients, one individual needed 48 hours of peritoneal dialysis (Table V).

Discussion

Congenital Cardiac Heart surgery following AKI is a frequent complication and it's been linked to higher death rates in both adults and children. Our study's overarching objective was to use the KDIGO criteria to determine the prevalence of AKI after heart surgery in pediatric patients hospitalized to Bangladesh Shishu Hospital & Institute at PICU. According to KDIGO criteria, the incidence of AKI in cardiac surgery patients was 9.3 percent, which is lower than the 15 to 64 percent seen in other published research. The prevalence of AKI in children after cardiac surgery varies with study population age and terminology utilized. Although we employed the KDIGO criteria in this investigation, additional well defined instruments such as the pRIFLE (pediatric Risk, Injury, Failure, Loss of function, and End-stage renal disease) criteria and the AKIN (acute kidney injury network) criteria have been used to diagnose AKI in children. The AKIN criteria were the most specific, whereas the pRIFLE criteria were the most sensitive, according to a comparison conducted by Lex et al[8] in pediatric patients. KDIGO criteria, they said, were similarly nuanced. We decided for the KDIGO since it has been shown to be effective in the pediatric critical care population.[9] Nephropathy may be caused by congenital heart defects (CHDs), which have been known about for quite some time. If patients with cyanotic heart disease do not undergo prompt correction, they are more likely to develop AKI and perhaps chronic renal disease. Alterations in renal blood flow and intraglomerular hemodynamics may impact renal function in the context of hypoxia, secondary polycythemia and aberrant arterial venous shunts.[9] Age is a significant risk factor for acute kidney injury (AKI) after heart surgery. A greater post-operative lactate level, indicative of hypoperfusion, was shown to be an independent risk factor for AKI, as was the case in the research by Cardoso et al[10] lending credence to the idea of hypoperfusion as a suggested cause of AKI. Patients who went on to develop AKI had a higher preoperative serum creatinine level, although this difference was not statistically significant (p=0.32). We could not find statistical significance in the mean RACHS-1 category. A higher RACHS-1 score has been linked to a lower risk of postoperative AKI.[11] Although not all investigations have shown this to be the case.[12] Possible explanations include a lack of instances that fall into the "high risk" (RACHS-1-category) group, as shown in.[3,4] Hemoglobin levels before surgery are one of the modifiable parameters documented in the literature as independent predictors of AKI.[13] According to the previous research[12], our results showed that individuals with preoperative anemia had a higher risk of developing AKI. Those who had low hemoglobin while not having cyanotic cardiac disease were at a higher risk for developing AKI. Therefore, treating anemia before to heart surgery in this population may mitigate postoperative AKI. Consistent with previous research, we discovered that AKI is linked to a higher risk of death and illness.[14]

Several studies have shown that CPB times longer than 120 minutes are associated with an increased risk of developing postoperative acute kidney injury.[1,12] Acute kidney injury (AKI) risk rises in tandem with CPB duration, which has been hypothesized to be associated with worsening ischemia and escalating inflammation. Patients with a CPB time >130 minutes in our population did not differ in their risk of developing AKI. Nevertheless, the length of cross-clamping or the percentage of ejection fraction at baseline were both shown to be unreliable predictors of AKI. In contrast to previous research that has indicated a correlation between nephrotoxic drugs such aminoglycosides, NSAIDs, and ACE inhibitors and an elevated risk of AKI, our study revealed no such link.[15] Frequent checks on plasma levels were done to avoid potentially hazardous accumulations of nephrotoxic medications. It is vital to appropriately estimate fluid volume status before providing any therapy since hyper or hypovolemia may result from an inaccurate evaluation. Weight gain of more than 7-10%, in addition to other signs of hypervolemia, showed that the patient had been overloaded with fluids. In our research population, we found no indication that fluid excess had a significant impact in the onset of AKI or its related mortality rates. This may be a result of using loop diuretics when absolutely required. We acknowledge that the results may have been affected by the lack of information regarding the use of inotropes, the volume of intraoperative blood loss, the amount of blood loss through the chest tube and the correction of cyanosis with hemoglobin. Nevertheless, there are difficulties in consistently quantifying all of these aspects due to the retrospective nature of the study.

Conclusion

AKI is common complication of pediatric cardiac surgery. Prolong mechanical ventilation in the ICU and in the hospital have all been linked to AKI. To improve the cardiopulmonary bypass techniques, improve renal perfusion and eradicate the causative risk factors are necessary for prevention of AKI in pediatric patients.

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