

ORIGINAL ARTICLE

The burden, predictors and outcomes of acute kidney injury in children admitted to the paediatric intensive care unit at the University Teaching Hospital in Lusaka, Zambia

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ABSTRACT

Background: Acute kidney injury (AKI) is common in critically ill patients and generates poor outcomes as a consequence. There is scant evidence on AKI encountered in paediatric intensive care units (PICU) in low-income countries such as Zambia. We have conducted a prospective study to investigate the burden, predictors, and consequences of AKI in children admitted to a tertiary hospital PICU in Lusaka.

Methods: A total of 134 participants, aged 29 days to 18 years and meeting the eligibility criteria, were investigated. Diagnosis of AKI was based on the Kidney Disease Improving Global Outcome (KDIGO) 2012 criteria. A pre-designed form was used to collect baseline demographic and clinical data as well as patient outcomes. Data were stored and analysed using SPSS version 25.

Results: The median age of the study population was 3.75 years (IQR 5,12), in whom AKI was present in 34%. Children under the age of one year had the highest prevalence of AKI (42%; $P = 0.001$) and sepsis was the cause in 31% of all AKI patients. Hypernatremia (AOR = 1.115; CI = 1.050–1.185; $P = <0.001$) and fluid overload (AOR = 6.207; CI = 1.020–37.591; $P = <0.047$) were predictors of AKI, which was associated with the need for vasopressor support (AOR = 10.015; CI = 1.038–51.763; $P = <0.006$) and 49% mortality (OR = 2.47; $P = <0.008$). Children with hospital-acquired AKI (HA-AKI) stayed longer in the hospital (43%) than those with community-acquired AKI (CA-AKI; 2.65% $P = 0.001$), and required prolonged ventilator support.

Conclusion: In this setting, AKI affects a third of PICU patients with infants, sepsis, fluid overload and hypernatremia being associated with a higher risk of developing AKI. This condition was linked to increased need for vasopressors and a high mortality.

Keywords: acute kidney injury; children; Africa.

BACKGROUND

Acute kidney injury (AKI) is a syndrome that refers to an abrupt decline in kidney function with a potential for reversibility [1,2]. Consensus over the definition of AKI has greatly improved the quality of information on the epidemiology and outcomes of the condition.

The incidence of AKI among children admitted to a paediatric intensive care unit (PICU) is estimated to range

between 8% and 89% [1,3-5]. The condition has short- and long-term implications, including longer hospital stay, greater morbidity including development of chronic kidney disease (CKD) and a higher chance of mortality [6-8]. Even minor increases in serum creatinine levels have been linked to poor hospital outcomes in patients undergoing heart surgery [9].

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AKI can be classified as either community-acquired (CA-AKI) or hospital-acquired (HA-AKI), depending on whether it developed prior to or after hospital admission [10,11]. The aetiologies differ according to geographical location. CA-AKI is more common in low- and middle-income countries and the causes include gastroenteritis, infections, and herbal medicine toxicity, whereas HA-AKI is often seen in high-income countries with the main causes being major surgery, haemato-oncological complications, nephrotoxic medicines, and pulmonary failure [11,12]. Compared to CA-AKI, HA-AKI patients tend to have a longer median length of hospital stay, need for dialysis and a higher mortality rate [13].

Reliable data on the epidemiology of childhood AKI from developing countries are scanty [14,15]. The exact burden of AKI in Zambian PICUs is unknown, but the associated mortality is presumably high. We conducted a study aimed at determining the prevalence, risk factors, and management outcomes of AKI in children admitted to the PICU at the University Teaching Hospitals (UTH) in Lusaka, Zambia.

METHODOLOGY

We undertook a prospective study to assess the risk factors for AKI and the outcomes of both community- and hospital-acquired AKI in children admitted to the PICU at UTH in Lusaka. UTH is the biggest and highest-referral hospital in Zambia.

The study was conducted from April 2020 to January 2021 and included 134 patients aged between 29 days and 18 years admitted in the PICU. All children meeting the inclusion criteria were recorded. Written informed consent with or without assent where applicable was obtained. Children admitted for surgery were excluded. Our PICU is largely a medical facility; most children admitted for surgery requiring intensive care are admitted to the hospital's main intensive care unit.

Age, sex and clinical data including weight, height, blood pressure, fluid status, temperature, primary diagnosis, use of common nephrotoxic drugs or use of herbal medication were obtained from the patients' medical charts using a data collection tool. The common nephrotoxic drugs recorded in the charts were gentamicin, angiotensin-converting enzymes, angiotensin receptor blockers, tenofovir disoproxil fumarate, acyclovir, amphotericin B and vancomycin.

For each participant peripheral venous blood was obtained within 24 hours of admission and sent to the lab for FBC, urea, electrolytes and serum creatinine. Blood for serum creatinine was collected on days 2 and 7. Serum creatinine was estimated using the Jaffe method. Urine output was recorded for catheterised patients, although it was not

used to define AKI as not all patients were catheterised. AKI diagnosis and staging was based on KDIGO criteria in terms of serum creatinine.

The cause of AKI was identified using the clinical features and lab investigation results obtained from each patient's file. Short-term outcomes were assessed over a period of 4 weeks. The main outcomes of interest were either death or discharge. Other parameters recorded were need for dialysis, vasopressor support or ventilation over the period of PICU admission.

The data collected were analysed using IBM's SPSS version 25 (IBM Corp., Armonk, NY, USA). AKI incidence was described using percentages. To determine the association between the primary outcome variable (AKI) and categorical independent or outcome variables with expected count less than 5, Fisher's exact test was used, otherwise Pearson's chi-squared test was employed. Means or medians of AKI and non-AKI patients were compared using Student's t-test and the Mann-Whitney test. Simple and multivariable logistic regression of AKI as the outcome variable and risk factors as the independent variables was run to determine predictors of AKI. Potential confounding factors were controlled during analysis using multivariable logistic regression. A significance level of 0.05 was used to include or exclude factors in the final model.

Ethical review and permission were sought and received from the University of Zambia Biomedical Research Ethics Committee (UNZABREC ref. no. 477-2019). Data were anonymised to protect patient confidentiality. Further clearance was obtained from the National Health Research Authority.

RESULTS

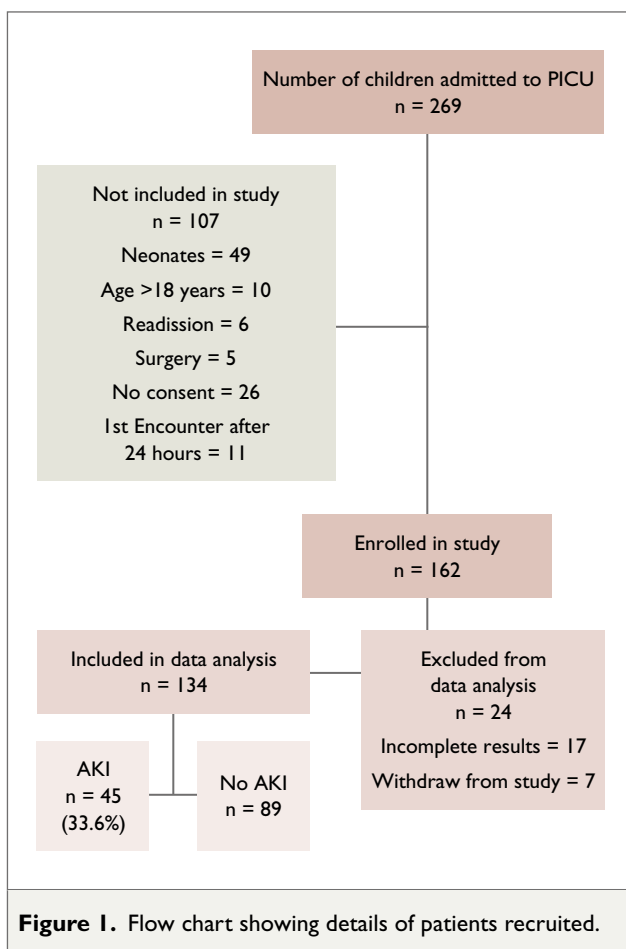
A total of 269 children were admitted to the PICU during the study. Figure 1 illustrates how study population was recruited.

Baseline demographic and clinical characteristics of study population

Table 1 illustrates demographic and clinical characteristics of participants. Infants had the highest AKI prevalence ($n = 19$, 42%). Hypotension was associated with the condition ($n = 16$, 36%) versus in the non-AKI group [$n = 11$ (36%); $P = 0.001$].

Association between laboratory parameters and AKI in children admitted to PICU

The relation between laboratory parameters and AKI is outlined in Table 2. There was a significant difference in serum sodium ($P < 0.001$), with median serum sodium being 135.8mmol/L in the non-AKI group as compared to 144mmol/L in the AKI group.



Prevalence, classification and cause of AKI

Of the 134 participants, 45 met the KIDGO 2012 criteria for AKI (based on serum creatinine), thus giving an AKI prevalence of 34%. Using the KIDGO classification, 22 (49%) had had stage 1, 11 (24%) had stage 2 and 12 (27%) had stage 3 AKI.

Of the 45 children that had AKI, 38 (84%) had CA-AKI and 7 (16%) HA-AKI. Their corresponding causes are listed in Table 3; the most common was sepsis (n = 14; 31%).

Outcome variables associated with AKI

Table 4 shows outcome variables associated with AKI. The mortality rate in the AKI group was 49% compared to 26% in the non-AKI group (P = 0.008). The need for vasopressors and ventilator support was also associated with AKI (P = <0.001 and 0.018, respectively). Days on ventilator support, median days spent in PICU and median length of hospital stay were not associated with AKI (P = 0.98, 0.85 and 0.712, respectively).

Comparison of the outcomes of CA-AKI and HA-AKI

In Table 5, outcomes of CA-AKI and HA-AKI were compared. There was no significant difference between HA-AKI and CA-AKI in terms of mortality rate (P = 0.188),

Table 1. Study participants' demographic and clinical characteristics and their relation to AKI.

	Characteristic	No AKI n (%)	AKI n (%)	P value
Sex	Male	48 (54)	26 (58)	0.672 [†]
	Female	41 (46)	19 (42)	
Age	29 days–1 year	22 (25)	19 (42)	0.016 [†]
	1 ⁺ –5 years	30 (34)	5 (11)	
	5 ⁺ –10 years	16 (18)	6 (13)	
	10 ⁺ –16 years	21 (24)	15 (33)	
Herbal medication	Yes	4 (4.5)	1 (2)	0.663 [*]
	No	85 (96)	44 (98)	
Nephrotoxic drugs	Yes	9 (10)	7 (16)	0.359 [†]
	No	80 (90)	38 (84)	
Blood pressure	Low	11 (12)	16 (36)	0.001 [†]
	Normal	72 (81)	23 (51)	
	High	6 (7)	6 (13)	
Temperature	Low	3 (3)	0	0.542 [*]
	Normal	50 (56)	29 (64)	
	High	36 (41)	16 (36)	
Fluids	Dehydrated	22 (25)	14 (31)	0.173 [†]
	Well-hydrated	59 (66)	23 (51)	
	Fluid overload	8 (9)	8 (18)	

^{*}Fisher's exact test; [†]Pearson's chi-squared test.

Table 2. Relation of laboratory parameters to AKI.

Characteristic	No AKI	AKI	P value
Serum urea level ($\mu\text{mol/L}$)	4.4	14	<0.001 [†]
Serum potassium (mmol/L)	4.2	4.5	0.143*
Serum sodium (mmol/L)	135.8	144	<0.001*
Haemoglobin (g/dl)	10.4	10.7	0.617*
Platelets ($\times 10^{11}/\text{unit}$)	308	330	0.625 [†]

[†]t-test; *Mann-Whitney test.

Table 3. Causes of AKI in the study population.

Causes	Frequency [n (%)]		Total
	CA-AKI	HA-AKI	
Severe sepsis	10 (26%)	4 (57%)	14 (31%)
Acute gastroenteritis	6 (16%)	—	6 (13%)
DKA	10 (36%)	—	10 (22%)
Primary renal disease	5 (13%)	—	5 (11%)
AGN	2	—	2
PUV	2	—	2
CKD with hypertensive crisis	1	—	1
Cardiogenic shock	5 (13%)	—	5 (11%)
Severe pneumonia	2 (5%)	2 (29%)	4 (9%)
Meningitis with high ICP	—	1 (14%)	1 (2%)
Total	38	7	45

Abbreviations: CA-AKI, community-acquired AKI; HA-AKI, hospital-acquired AKI; DKA, diabetic ketoacidosis; AGN, acute glomerulonephritis; PUV, posterior urethral valves; CKD, chronic kidney disease.

inotropic support ($P = 0.225$), peritoneal dialysis ($P = 1.000$) and haemodialysis ($P = 0.710$). However, there was an increased length of stay in PICU for 3 children with HA-AKI, for more than 14 days, compared to one child in the CA-AKI group ($P = 0.005$). The need for ventilator support was noted to be more in the HA-AKI group than for the CA-AKI patients ($P = <0.001$).

Logistic regression analysis to determine predictors of AKI in children admitted to PICU

The predictive value of independent variables was assessed using an operator-led regression analysis, in Table 6, where variables with the highest P values were eliminated. On univariate analysis, high sodium ($OR = 1.09$), mechanical ventilation ($OR = 2.881$), vasopressor support ($OR = 0.195$) and hypotension ($OR = 4.533$) were predictive of AKI. Age, fluid status and potassium were not predictive.

Increase in sodium ($AOR = 1.115$), and fluid overload ($AOR = 6.207$) were associated with AKI as was vasopressor support ($AOR = 10.015$). Low BP was not predictive of AKI.

DISCUSSION

In our study AKI was highly prevalent in children admitted to PICU and was associated with a high mortality rate. In

Table 4. Relation of outcome variables to AKI.

Characteristic		No AKI	AKI	P value
Outcome	Discharge	66 (74%)	23 (51%)	0.008 [†]
	Mortality	23 (26%)	22 (49%)	
Length of hospital stay	<48 hr	18 (20%)	13 (30%)	0.98 [†]
	48 hr–7 days	26 (29%)	11 (25%)	
	8–14 days	21 (24%)	7 (16%)	
	>14 days	24 (27%)	13 (30%)	
Median length of hospital stay (IQ range)		11*	15.5	0.712 [†]
		38 (43%)	16 (36%)	
Number of days in PICU		42 (47%)	21 (47%)	0.339*
		7 (8%)	4 (9%)	
		2 (2%)	4 (9%)	
Median length of stay in PICU		4*	6.1	0.85 [†]
Need for ventilation	Yes	11 (12%)	13 (29%)	0.018 [†]
	No	78 (88%)	32 (71%)	
Ventilation days	No ventilation	78 (88%)	32 (71%)	0.98*
	<48 hr	5 (6%)	4 (9%)	
	2–7 days	5 (6%)	6 (13%)	
	7–14 days	0 (0%)	2 (4%)	
	>14 days	1 (1%)	1 (2%)	
Vasopressor use	Yes	13 (15%)	21 (47%)	< 0.001 [†]
	No	76 (85%)	24 (54%)	

*Fisher's exact test; [†]Pearson's chi-squared test.

Table 5. Comparison of community-acquired AKI with hospital-acquired AKI.

Characteristic		CA-AKI	HA-AKI	Odds ratio	P value
Outcome	Discharge	21 (55%)	2 (29%)		0.188 [†]
	Mortality	17 (45%)	5 (71%)		
Day of outcome	<48 hr	15 (40%)	0 (0%)		0.005 [†]
	48 hr–7 days	29 (50%)	3 (43%)		
	8–14 days	3 (8%)	1 (14%)		
	>14 days	1 (3%)	3 (43%)		
	<48 hr	15 (40%)	0 (0%)		
Number of days in PICU	2–7 days	19 (50%)	3 (43%)		0.005 [†]
	7–14 days	3 (8%)	1 (14%)		
	>14 days	1 (3%)	3 (43%)		
Need for ventilator support	No	31 (82%)	1 (14%)	0.038	<0.001 [†]
	Yes	7 (18%)	6 (86%)		
Vasopressor use	Yes	16 (42%)	5 (71%)	0.050	0.225
	No	22 (58%)	2 (29%)		
Haemodialysis	Yes	2 (5%)	0 (0%)		0.710 [*]
	No	36 (95%)	7 (100%)		
Peritoneal dialysis	Yes	4 (11%)	0 (0%)		1.000 [*]
	No	34 (90%)	7 (100%)		

*Fisher's exact test; [†]Pearson's chi-squared test.**Table 6.** Logistic regression analysis showing relationship of selected independent variables to AKI.

Variable	Odds ratio (95% CI)	P value	Adjusted odds ratio (95% CI)	P value
Urea	1.225 (1.128–1.328)	<0.001	1.198 (1.088–1.318)	<0.001
Serum sodium	1.096 (1.047–1.145)	<0.001	1.115 (1.050–1.185)	<0.001
Serum potassium	1.341 (0.963–1.867)	0.082	1.432 (0.864–2.272)	0.163
Haemoglobin	1.030 (0.920–1.153)	0.606	1.253 (1.003–1.565)	0.057
Age	1.014 (0.952–1.081)	0.565	1.046 (0.931–1.175)	1.176
Vasopressor support				
No	1			
Yes	0.195 (0.085–0.448)	<0.001	10.015 (1.938–51.763)	0.006
Ventilation				
No	1			
Yes	2.881 (1.169–7.101)	0.022	2.710 (0.505–14.527)	0.244
Blood pressure				
Normal BP	1			
Low BP	4.533 (1.851–11.197)	0.001	3.376 (0.602–18.923)	0.166
High BP	3.130 (0.919–10.657)	0.068	2.906 (0.384–22.025)	0.302
Fluid status				
Well-hydrated	1			
Dehydration	1.632 (0.715–3.726)	0.245	1.149 (0.321–4.116)	0.830
Fluid overload	2.565 (0.861–7.645)	0.091	6.207 (1.02–37.591)	0.047

addition, other adverse outcomes, such as need for mechanical ventilation and for vasopressor support, and length of PICU stay and length of hospital stay, were associated with patients admitted to PICU with AKI. CA-AKI had a better prognosis than HA-AKI.

We have shown that AKI was prevalent in 34% of children admitted to the PICU, compared to 27% in multicentre studies involving 32 institutions [1]. A survey conducted in Lagos, Nigeria, reported a prevalence of AKI of 19% among critically ill children at a tertiary hospital [4], which is lower

than in our cohort. One reason for the disparity could be that the Lagos study included critically ill children admitted to general wards as well as PICU patients. In addition, the pRiffle criteria used for the diagnosis of AKI in the Lagos observational study are less sensitive than the KDIGO 2012 criteria used here.

The majority of children (48%) had KDIGO class 1 AKI whereas only 24% and 2.7% represented KDIGO classes 2 and 3, respectively. This is similar to what has been described in other studies [1,2,15]. Most of our patients (84.4%) had CA-AKI as described in studies from other low-resource settings [1,17,18]. In contrast, reports from high-income countries found that HA-AKI accounts for most AKI cases [19,20]. One reason for this finding is related to recent advances in medical care, which have improved survival rates and a consequent increase in patients with complex conditions, who present a higher risk of PICU hospitalisation than the general population [20].

The leading causes of AKI in our cohort were sepsis ($n = 14$, 31%) followed by DKA ($n = 10$, 22%) and acute gastroenteritis ($n = 6$, 13%). This is similar to findings from other studies conducted in Africa [5,6], where sepsis, malaria, toxins and HUS predominate.

Surprisingly, no one suffered malaria-related AKI even though malaria is endemic throughout most of Zambia [22]. This is in contrast to reports from other sub-Saharan African countries [14,21]. One rationale for this finding in this cohort could be that most patients with malaria-related AKI have single-organ dysfunction and do not meet the criteria for PICU admission. Furthermore, most patients with malaria-associated AKI are referred from lower-level hospitals, where they receive initial treatment and stabilisation, and are thus less likely to require critical care in the PICU but instead are admitted to the renal unit.

Age was linked to the development of AKI, with a higher prevalence in younger children, comparable to what has been described in the literature, where a predominance in children younger than one year has been found [1]. However, regression analysis revealed no link between age and AKI.

Hyponatremia and fluid overload were found to be predictive of AKI using logistic analysis. Several studies have demonstrated the relationship between hyponatremia and AKI [28,29]. Patients admitted to 30 intensive care units in China demonstrated that fluid accumulation was independently associated with an increased risk of AKI [27]. It is, however, difficult to determine whether AKI has caused fluid overload or vice versa [30].

Our cohort showed no link between herbal intoxication and AKI. Other research in sub-Saharan Africa found the opposite [14]. One reason for this could be the small

number of children in our study who used herbal medication ($n = 5$, 4%), thus, a clear causal association cannot be concluded. Lusaka is a city with free health care, so that most parents take their children to the hospital rather than to herbalists. Another problem was that we depended on caregiver history; as a result, some caregivers may have kept this information from us.

The median length of PICU stay was higher in AKI patients (6.1 days) than in non-AKI patients (4 days). Similarly, length of hospital stay was also greater in the patients with AKI (15.5 days) than those without AKI (11 days), although not statistically significant. Studies from other centres have also reported longer PICU and hospital stay in AKI patients [3-5]. More resources at family, hospital and national levels are spent on those who stay longer in PICU and hospital. Thus, AKI-associated costs have huge implications at all levels of the country's economy, hence the importance of AKI in PICU cannot be overemphasised [23].

The need for ventilation and use of vasoactive drugs was higher in the AKI group than in non-AKI patients ($P = 0.018$ and <0.001 , respectively) in our study. This is similar to a survey in India that described the need of ventilation and vasoactive drugs requirement to have had increased risk of kidney injury [24]. However, patients with AKI were associated with an increased need for vasopressor support. This means more resources, including human resources, medical equipment and drugs are needed to manage patients with AKI in PICU than those without the condition [23,25].

There was increased length of stay in PICU in the HA-AKI group, with 43% of such patients staying in PICU for more than 14 days as compared to only 2.6% in the CA-AKI group. The need for ventilator support was significantly higher in the HA-AKI group (86%) compared to 18% of the CA-AKI group. This reflects other studies [18,26], and implies more resources are to be used in patients who develop AKI in hospital than those who are admitted with AKI.

The mortality rate of patients with AKI was 49%. Several surveys have clearly shown that any degree of AKI is an indicator of poor prognosis for critically ill patients [5,9,17-19]. Other studies reported mortality in AKI of 37%, 46.3% and 50.4%, respectively [6,10,23], similar to that described in our study.

Our study was a single-centre investigation; hence, the AKI prevalence and risk factors may not be generalisable to other settings. AKI, especially hospital-acquired, might have been underestimated as urine output was not used in the diagnosis of the condition as not all patients were catheterised. In addition, long-term mortality, CKD development, and renal function progression were not evaluated in our study but should be considered in future research.

A third of children admitted to PICU at UTH had AKI. Young age, sepsis, fluid overload and hyponatremia were associated with a higher risk of developing AKI. As reported from earlier studies, mortality was high (49%) among our PICU patients with AKI. Furthermore, such patients had a relatively high need for vasopressor support.

Conflict of interest

The authors have no conflicts of interest to declare.

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