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Acute Kidney Injury among hospitalised children in a young tertiary centre in south-south, Nigeria: An observational prospective study

Briggs DC[1], Aitafo JE[1], Okorie E-M[2]

[1]*Department of Paediatrics & Child Health, Rivers State University Teaching Hospital/Rivers State University.*
[2]*Department of Paediatrics, Pamo University of Medical Sciences, Port Harcourt.*

Abstract

Background:
Context: Paediatric acute kidney injury (AKI) often goes unrecognised in resource-limited emergency room settings and is a major contributor to morbidity and mortality. It is necessary to define the epidemiology of AKI in referral hospital settings where none exists.
Objective: To determine the pattern, risk factors and outcome of acute kidney injury among children admitted into the Children's Emergency Room of Rivers State University Teaching Hospital (RSUTH).
Methods: This prospective observational study involved all paediatric cases with KDIGO-defined AKI between October 2020 and October 2023 followed up for 3 months. Patients' data prospectively documented included age, sex, presenting symptoms, diagnosis, blood pressure, urine output, blood chemistry, treatment, and outcome. Data were analysed.
Results: Forty-three cases of AKI were seen, giving an incidence of 15.0 per 1000, 38 (88.4% were community-acquired). The mean age was 5.8 ±5.2 years (range: 2 months to 16 years) and 27(62.8%) were males. AKI was present in 38(88.4%) at presentation and 20(46.5%) had KDIGO stage III. The top four causes were: sepsis 16(37.2%), Primary renal diseases 10(23.3%), malaria 8(18.6%), gastroenteritis 5(11.6%). Seven (16.3%) required dialysis and mortality occurred in 7(16.3%). The risk of mortality was increased in those with sepsis [RR:13.6 (1.46 – 127.1)], late presentation [1.28(1.07 – 1.5)] and requiring dialysis [3.86(1.09 – 13.59)]
Conclusion: AKI is common in the children's emergency room of RSUTH. Early presentation, screening for AKI and prompt treatment of underlying causes are imperative to improve outcomes. Funding and support for paediatric dialysis programmes for severe AKI are needed.

Keywords: acute kidney injury, acute kidney disease, paediatric, mortality, risk factors, chronic kidney disease.

Introduction

Acute kidney injury (AKI) denotes a rapid deterioration in kidney function, often posing serious complications to both critically and non-critically ill hospitalised children, globally. Studies suggest an increasing burden of AKI among hospitalised children with resultant high morbidity, increased length of hospital stay, need for intensive care/ ventilatory support and high mortality.[1–3] However, paediatric AKI often goes unrecognised in resource-limited emergency room settings and is a major contributor to poor short- and long-term health outcomes.[4]

AKI significantly influences outcomes in hospitalised children with incidence rates varying globally from 5.0% to 7.5% and reaching up to 50% to 60% in paediatric intensive care units.[5] In Nigeria, AKI

Corresponding Author:

Dr. Briggs, Datonye Christopher
Department of Paediatrics & Child Health, Rivers State University Teaching Hospital/Rivers State University

datonye.briggs1@ust.edu.ng | ORCID: 0000-0002-2462-4964

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accounts for about 1% – 3% of all hospital admissions, with a reported incidence of 10 – 13.5 per million children[6] and prevalence rates of 17.7 – 26 per 1000 children. Over 80% of paediatric AKI cases are community-acquired, and about 70% of them present in severe AKI with mortality rates as high as 28.4% - 48.0%.[7–9] Despite these perturbing figures, paediatric AKI remains an under-recognized public health challenge in Nigeria.[10]

The commonest causes of paediatric AKI in resource-limited settings (RLS), besides primary kidney disease, include sepsis, malaria, and diarrhoeal diseases/dehydration which are mainly preventable.[7–9,11,12] In Nigeria, many children with AKI do not often receive timely and appropriate care, leading to poor outcomes.[13] The main predictors of AKI-related death as documented by earlier studies were the need for dialysis, leucocytosis and thrombocytopenia, male gender and the presence of pulmonary oedema.[7,11] However, the availability of dialysis services is limited, and many families cannot afford the exorbitant costs of treatment, resulting in a significant treatment gap.[14]

Despite global interest in reducing deaths from AKI in RLS, the high burden of AKI remains a major threat to the realization of the International Society of Nephrology (ISN) 0 by 25 initiative which aims at eliminating preventable deaths due to AKI in RLS by 2025.[15] The high prevalence of AKI in our setting stems from several factors: insufficient public health education, lax enforcement of sanitation laws, and limited access to healthcare services.[16] Other contributors include delayed identification of risk factors, inadequate medical training, and limited availability of kidney replacement therapy (KRT). Late diagnosis and intervention result in complications like hypertensive and uraemic encephalopathy, congestive heart failure, and chronic kidney disease (CKD), leading to increased AKI-related mortality.[11] Conversely, early recognition and intervention have been shown to reverse AKI with minimal long-term effects, and this is especially crucial in RLS with limited facilities for KRT in children.

Therefore, it is crucial to understand the epidemiology of paediatric AKI and changing trends in referral hospital settings, to optimize healthcare resource allocation. The only previous study on this research topic in Rivers State, Nigeria was retrospective and conducted over fifteen years ago in another tertiary centre.[17] Hence, this study aimed to determine the incidence, aetiologies, risk factors, and outcomes of AKI among children admitted to the Children's Emergency Room of the Rivers State University Teaching Hospital (RSUTH).

Materials and methods

This was a prospective observational study of paediatric AKI cases in the Children's Emergency Room (CHER) of the Department of Paediatrics RSUTH between October 2020 and October 2023. All paediatric cases, excluding neonates, presenting to the CHER with a clinical diagnosis of AKI or developed AKI while on admission were included and followed up for 3 months, post-discharge.

The Rivers University Teaching Hospital is located in Port Harcourt City, the capital of Rivers State, south-south Nigeria. RSUTH is a 340-bed facility and one of two publicly funded tertiary hospitals providing renal care to children in Rivers State. It has 8 and 31-bed CHER and paediatric wards respectively for children above one month of age. The critically ill children are stabilized in CHER and then transferred to the paediatric wards. The intensive care unit in the hospital lacked paediatric ventilatory support and was inaccessible to critically ill children.

Acute kidney injury (AKI) was defined according to Kidney Disease Improving Global Outcomes (KDIGO)[18] as an abrupt decrease in kidney function occurring within 7 days and CKD is defined as the persistence of kidney disease beyond 90 days.[19] Acute kidney disease (AKD) is defined according to the Acute Disease Quality Initiative (ADQI) as kidney damage lasting between 7 and 90 days after an acute kidney injury.[20] Kidney function was assessed on admission or day 0 (at diagnosis of AKI), at 48 h, day 7 or at discharge and repeated at 1 and 3 months. Urine output was monitored every 24 hours via indwelling catheters and as voluntarily voided samples. Oliguria was urine output <0.5 mL/kg/hr.

The expected normal serum creatinine level was determined by applying the equation for estimated glomerular filtration rate (eGFR) derived from the chronic kidney disease in children (CKiD) model (baseline serum creatinine = 0.41 × height in centimetres/eGFR).[21,22] This estimation yielded a baseline serum creatinine level corresponding to an eGFR of 120ml/min/1.73m^2 for children aged over 2 years. Conversely, for children aged 2 years or younger, the estimated baseline serum creatinine level was determined based on age-specific normative parameters.[22,23] Serum creatinine assays were

conducted using the modified Jaffe's reaction method.[24]

Children with AKI also got serology testing for HIV, hepatitis B surface antigen and hepatitis C antibody. Port Harcourt being an endemic area for malaria, peripheral blood thick film for Plasmodium falciparum was also done for children with suspected AKI. Other investigations done include urine analysis; and microscopy (for red cell cast for suspected acute glomerulonephritis), Urine culture and sensitivity for suspected urinary tract infection (UTI). Abdomino-pelvic ultrasonography was done to evaluate the kidney ureters and bladder in all children with AKI, particularly in those with suspected obstructive uropathy. Fluid retention/overload was defined as the presence of pulmonary oedema/heart failure on clinical examination and hypertension was defined as $\geq 95^{th}$ percentile for age/sex/height. The fluid restriction was 300-400ml/m^2 plus the previous day urine output.

Management of AKI included treating the underlying cause(s), and bed rest. Blood transfusion was done if a child was decompensating and PCV < 18% [Hb of 6g/dl] with administration of intravenous furosemide (1mg/kg) and in aliquots of 5-10ml/kg over two hours, depending on the severity of fluid overload and pulmonary oedema. Hyperkalaemia (serum potassium > 6.5mmol/L) was managed using IV calcium gluconate 0.5ml/kg (max 20 ml) or shifted with nebulized salbutamol (body weight < 25kg: 2.5mg and > 25kg 5mg). Hyponatraemia (< 130mmol/L) was managed by fluid restriction and diuretics, if due to fluid overload. Acute severe hypertension was treated with IV labetalol 0.2mg/kg over 2 minutes if no response in 5-10 minutes, the dose was increased to 0.4-1.0mg/kg and continued as intravenous infusion 0.4 – 1.0 mg/kg/hr or IV hydralazine 0.1 – 0.3mg/kg (max. 10mg) and administered slowly over 5 minutes, and repeated 4 - 6 hrly as indicated, and when stable and able to take by mouth, oral amlodipine and oral or IV furosemide 0.5 – 1mg/kg/dose, 6 – 12hrly. Metabolic acidosis was managed using IV 8.4% sodium bicarbonate (3mmol/kg) and co-existing infections were treated empirically with cefuroxime (25-50mg/kg 8hrly) or ceftriaxone (50mg/kg 12-24hrly) and then changed according to the antibiogram. Medications were renal dose adjusted.

Peritoneal dialysis (PD) was manually performed using an improvised PD fluid [composition of improvised PD fluid was adapted from the ISPD guideline[25] using 500mls of Ringers Lactate to 15ml and 23mls 50% dextrose to make a 1.5% and 2.3% solutions respectively] to which Heparin (500 iu/l) was added. Improvised PD catheters using straight uncuffed acute Haemodialysis (HD) catheters [Medcomp 9FR] or nasogastric tubes [size 10 FR] were inserted by the paediatric nephrologist using the Seldinger technique at the bedside. The haemodialysis machines and dialyzers used for those requiring intermittent HD were Fresenius 5008/4008 (Germany), Dialog+ (B. Braun + Evo Medical Inc, Germany), Dialyzer sizes [F 8, FX 50, 60, 80], Dialyzer Surface Areas: [1.0 – 1.8m^2], Vascular access was via the femoral veins using short term acute HD uncuffed dual-lumen catheters [Medcomp 8FR or 9FR]. Adult extracorporeal bloodlines were used. HD prescriptions were made using the following: extracorporeal blood volumes were kept at < 10% and were primed with either normal saline or whole blood. The blood flow rate was 6 – 8ml/kg/min and the ultrafiltration (UF) rate was 0.2 ml/kg/min [Total UF was kept < 5% of body weight]. Standard heparin was used for anticoagulation as per protocol.[26]

Random blood glucose was obtained before, during and post dialysis and blood pressure measurements were obtained as per protocol. Intradialytic hypotension was defined as a decrease in systolic blood pressure (SBP) ≥ 20mmHg or a reduction in mean arterial pressure (MAP) by 10mmHg which is associated with clinical signs needing intervention.[26] Intradialytic hypertension was defined as an increase in mean arterial pressure of ≥ 15 mmHg during or after HD or an increase in systolic BP of > 10 mmHg from pre-dialysis to post-dialysis.[26] Intra-dialysis, hypoglycaemia was corrected using intravenous (IV) 50% dextrose (1ml/kg) and severe hypertension received nifedipine 0.25 – 0.5mg/kg sublingual, seizures were managed with IV 20% Mannitol 0.2-0.5g/kg over 1 hour, and those with intra-dialytic hypotension received intravenous IV 0.9% saline 10ml/kg or IV dopamine 2 – 5 ug/kg/min respectively. The minimum haemoglobin allowed for haemodialysis was 8g/dl (24%) and crossmatched sedimented red cells were administered intra-dialysis.

Early AKI presentation was taken as when the presenting complaints and clinical features associated with AKI diagnosis was ≤72 hours duration whereas, a late AKI presentation was presumed when the presenting complaints/or signs associated with AKI diagnosis was > 72 hours duration.[27]

A study proforma containing the following variables

was used to obtain all the relevant patient data: age, sex, presenting symptoms, diagnosis, urine output, serum electrolytes, calcium and phosphate, treatment received [conservative or kidney replacement therapy (KRT): Peritoneal (PD) or Haemodialysis (HD)], duration of hospital stay, and primary outcomes were [morbidity (progression to CKD), discharged, discharged against medical advice (DAMA) and mortality]. Data analysis was done using SPSS version 25. The risk factors associated with AKI mortality were also determined. Ethical Approval was obtained from the Hospital Ethical Review Board (RSUTH/REC/2021289).

Results

Description of participants

Children within the 12 – 60-month age category constituted 16 (37.2%) of the cohort. The mean age was 5.8 ± 5.2 years and ranged between 2 months and 16 years. The majority 27 (62.8%) were males. The median duration from presentation to the CHER and a nephrology consult was 2 days (1 – 5 days).

Incidence of Paediatric AKI in RSUTH

Of 2,865 admissions, forty-three cases of AKI were seen, giving an incidence of 15.0 per 1000 [14.3 cases per annum]. AKI made up 43 (39.1%) of the 110 kidney-related cases seen during the study period.

Presenting symptoms, Laboratory investigations and AKI staging

Body swelling (94.7%), fast breathing (89.5%), paleness of the body and reduced urine output (84.2%) were the main clinical symptoms that were noted in children who had AKI (Figure 1)

The mean Packed Cell Volume (PCV) was 19.8% ±6.2%, White Cell Count (WCC) was 21.6 ± 20.3 x 109/l, serum urea and creatinine at admission were, 11.95±6.78mmol, 337.3±341.1µmol/L respectively. The mean duration of hospitalization was 13.1±7.8 days. The distribution other of laboratory parameters

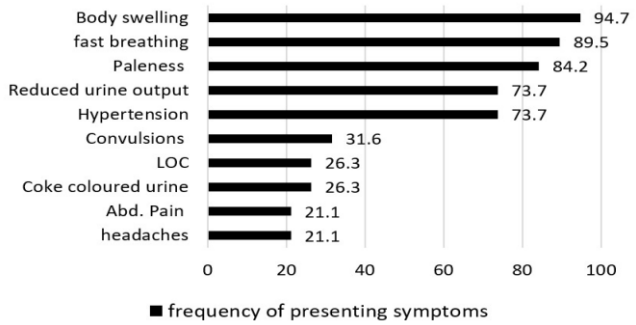

Figure 1: AKI presenting symptoms

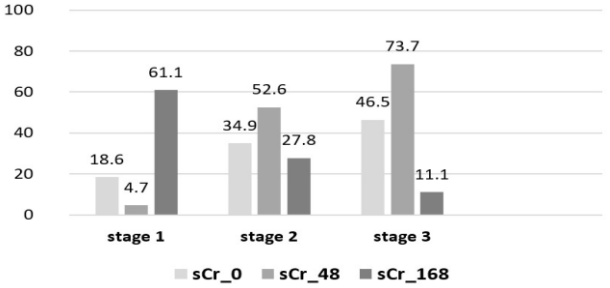

Figure 2: Proportion of AKI stage using serum Creatinine (0, 48 and 168 hrs)

Table I: Demographic characteristics, laboratory investigations and trends in mean serum urea and creatinine among children with Acute Kidney Injury

A. Demographics	Frequency (%)
Age (Months)	
< 12	12 (27.9)
12 – 60	16 (37.2)
> 60	15 (34.9)
Gender	
Male	27 (62.8)
Female	16 (37.2)
B. Laboratory investigations	
Hyponatraemia (< 130 mmol/l)	14 (32.6)
Hypernatraemia (> 150 mmol/L)	9 (20.9)
Anaemia (<21% / Hb: < 7g/dl)	26 (60.5)
Metabolic Acidosis (HCO_3 < 15)	22 (51.1)
Hyperkalaemia (> 6.0)	18 (41.9)
Hypocalcaemia (< 2.2 mmol/L)	15 (34.9)
Proteinuria (> 1+ on dipstick)	21 (48.8)
White Cell Count (> 15.0)	23 (53.5)
Platelets (< 100)	18 (41.9)
Type of AKI	
Community-acquired	38 (88.4)
Hospital-acquired	5 (11.6)
Management	
Conservative	36 (83.7)
Dialysis	7 (16.3)
Type of dialysis	
Peritoneal (PD)	2 (28.6)
Haemodialysis (HD)	5 (71.4)
C. Trends in the mean serum urea and creatinine over 3 months	
Mean serum urea [mmol/L]	
Day 0 [n 43]	11.95+6.78
Day 2 [n=42]	11.68±7.08
Day 7 [n=38]	10.55±4.68
1-month [n=35]	8.95±8.68
3-month [n=28]	4.49±1.19
Mean serum creatinine [umol/L]	
Day 0 [n 43]	377.3+ 341.10
Day 2 [n 42]	314.87+195.58
Day 7 [n= 38]	226.48±223.44
1-month [n=35]	175.5±238.08
3-month [n=28]	74.96±38.75

Table II: Causes of Paediatric Acute Kidney Injury in RSUTH

Aetiology	Frequency n (%)
Sepsis&	16 (37.2)
Primary renal disease*	10 (23.2)
Plasmodium falciparum Malaria	8 (18.6)
Acute Gastroenteritis	5 (11.6)
Others [SCD, post-renal (PUV)] **	4 (9.3)

& Sepsis with comorbidities [Anaemic Heart failure, Bronchopneumonia, Cellulitis, Meningitis]
* Post Infectious GN (70.0%) Nephrotic Syndrome (20.0%), Haemolytic uraemic syndrome (10.0%)
** SCD: sickle cell disease, SLE: systemic lupus erythematosus; post-obstructive polyuria [PUV]

Table III: Indications for Kidney Replacement Therapy

Indication	Frequency (%) *
Fluid overload/Pulmonary oedema + Uncontrolled hypertension	4 (66.7)
Oliguria/anuria unresponsive to diuretics	4 (66.7)
Uraemic encephalopathy (LOC/Seizures)	3 (42.8)
Intractable hyperkalaemia > 7.0 mmol/l	2 (33.3)
Intractable metabolic acidosis < 10mmol/l	1 (16.7)
*Multiple symptoms apply	

Table IV: Factors associated with AKI-related mortality

Characteristics	Alive	Died	[adjusted Relative Risk]		
			RR	CI	P-value
Age (months)					
<60	28	3	0.65	0.27 – 1.59	0.3531
>60	15	4			
Gender					
Male	23	4	0.89	0.45 – 1.77	0.7502
Female	13	3			
Aetiology					
Sepsis	11	6	13.6	1.46-127.1	**0.0143****
Non-sepsis	25	1			
Presentation					
Late	28	7	1.28	1.07 – 1.5	**0.0048****
Early	8	0			
Seizure					
Yes	10	3	1.85	0.70 – 4.83	0.2089
No	27	3			
Oliguria					
Yes	24	6	2.6	0.34 – 19.49	0.3526
No	12	1			
Pulmonary oedema					
Yes	8	4	3.4	0.9 – 13.16	0.0706
No	28	3			
Anaemia					
Yes	16	5	2.62	0.57 – 12.05	0.2165
No	20	2			
Hypertension					
Yes	21	5	1.63	0.36 – 7.49	0.5268
No	15	2			
AKI stage					
Stage III	14	6	6.90	0.91 – 52.56	0.0623
Stage I – II	22	1			
White Cell Count					
>15.0	18	5	2.1	0.47 – 10.00	0.4205
<15.0	18	2			
Dialysis					
Yes	4	3	3.86	1.09 13.59	**0.0356****
No	32	4			
Type of AKI					
Community	33	5	0.45	0.12 – 1.69	0.2366
Hospital	3	2			
Duration of hospitalization					
< 7 days	9	5	5.18	1.14 23.46	**0.0329****
>7 days	27	2			

** statistically significant

is displayed in Table 1.

AKI was present in 38 (88.4%) presentations with 38 (88.4%) being community-acquired. The majority (46.5%) of the cases were AKI stage III at day 0 or at presentation and a notable improvement in kidney function KDIGO stage I (61.1%) at 168 hours post diagnosis (Figure 2)

Aetiology of AKI

Sepsis (37.2%), Primary kidney disease (23.2%) and malaria (18.6%) were the leading causes of AKI in this series as displayed in Table II. Children with Sepsis AKI had other coexisting comorbidities [Anaemic heart failure (10, 62.5%), Bronchopneumonia (4, 25%), Cellulitis (2, 12.5%), Meningitis (1, 6.25%)]. As regards the primary kidney disease, post-infectious glomerulonephritis (7, 63.6%) Nephrotic Syndrome (2, 18.1%), Haemolytic uraemic syndrome (1, 9.1%) and lupus nephritis (1, 9.1%) were the main contributors.

AKI management and the need for Kidney Replacement Therapy

Whereas most children (36, 83.7%) were managed for AKI conservatively, 7 (16.3%) required dialysis. Two of the children received manual peritoneal dialysis and five received intermittent haemodialysis. The commonest indications for KRT were fluid overload/ pulmonary oedema and hypertension 4 (66.7%) and oliguria/anuria 4 (66.7%) (Table III). The mean number of HD sessions was 3 (2 – 15), and PD sessions were 48 -161 cycles (12 – 14 days).

AKI outcome

Although 38 (88.4%) had AKD, with 61.1%, 27.8% and 11.1% being in AKI stages I, II and III respectively on the 7th day post AKI diagnosis, 34 (79.1%) had resolution of AKD. This occurred in 28 (82.4%) and 6 (17.6%) within one and two-to-three months of follow-up, respectively. However, two progressed to chronic kidney disease and one absconded. Overall, mortality occurred in 7 (16.3%) of cases. Three of them who died had dialysis – one peritoneal dialysis and two haemodialysis.

Associated Risk factors of AKI-related deaths

Subgroup analyses (Table IV) revealed that the mortality seen in this study was not associated with the age or gender of the children or other indicators of severe AKI (seizure, oliguria, hypertension, pulmonary oedema). Also, deaths were unrelated to the type of AKI (hospital-acquired or community-acquired). However, children with sepsis, late presentation (> 3 days), short hospital stay and those who received dialysis were 13.6 (1.46 – 127.1)], 1.28 (1.07 – 1.5), 5.18 (1.14 – 23.46) and 3.86 (1.09 – 13.59) times more likely to die. There was also some weak evidence to suggest that those with AKI stage III and pulmonary oedema were at increased risk of AKI-related deaths (p = 0.06 and 0.07) respectively.

Discussion

This study set out to describe the epidemiology, risk factors and treatment outcome of AKI among a non-selected cohort of children admitted into the Children's Emergency Room of Rivers State University Teaching Hospital (RSUTH) which previously had not been documented. The incidence of AKI was 15 per 1000 (14 cases per year), the identified critical risk factors for mortality in this cohort were late presentation, sepsis-induced AKI, the need for dialysis and one in six children succumbed to the illness.

The study demonstrated that 43 cases of AKI were seen over three years, which corresponds to an incidence rate of 15 cases per 1000 children. This was similar to the 17.4 per 1000 reported by Esezobor et al[13] but lower than 29 per 1000 by Solarin et al[28] both conducted in Lagos, South-West, Nigeria. Similarly, our finding of about 14 cases per annum, was comparable to the 123 cases over nine years reported

by Olowu et al[29] in Ile-Ife, Nigeria; higher than the 22 cases over four years reported by Adedoyin et al[30] in Ilorin, Nigeria, but lower than the 43 cases per year reported by Anigilaje et al[27] in Abuja. In comparison with other parts of West Africa, the 14 cases of AKI in our study was higher than the 87 cases over nine years in Cameroon,[31] but was lower than the 105 cases over six years in Congo-Brazzaville[32] and the 68 cases per annum reported by Antwi et al[33] in Ghana. The reported variations could be attributed to the varying definitions of paediatric AKI.

Notwithstanding, our study suggests an increase in the occurrence of AKI within our setting when compared to 211 cases seen over 18 years reported by Anochie and Eke in Port Harcourt[17] Rivers State Nigeria, which is in alignment with recent studies that report an increase in AKI burden among hospitalized children.[2,6,34,35] It is plausible that the current unprecedented rate of inflation, out-of-pocket payment for healthcare and lack of universal healthcare coverage for common childhood illnesses leading to late presentation are the underlying contributors. Furthermore, because the causes of AKI have remained largely unchanged – being mostly due to preventable causes, it could be argued that a higher index of suspicion and case finding by paediatricians at our centre may account for our reported incidence.

Although the findings demonstrate a relatively high incidence of AKI in this cohort, it is conceivable that this figure may have been higher if all paediatric patients presenting to the emergency room had undergone comprehensive assessments of their kidney function and 24-hour urine monitoring upon admission. This study underscores the importance of heightened awareness and more rigorous surveillance to actively identify cases of both community and hospital-acquired AKI among children admitted for other medical conditions. This assertion finds validation in the observation that conspicuous clinical manifestations, including generalized oedema, tachypnoea, pallor, and oliguria, served as the primary indicators prompting the attention of the CHER team toward the potential occurrence of renal impairment. Such symptoms often manifest in advanced stages of AKI, underscoring the imperative for a heightened index of clinical suspicion.

The finding that nearly half of the paediatric cases presented in stage III AKI is a cause for concern and may be potentially indicative of community-wide unfamiliarity with the symptoms of kidney dysfunction and delays in seeking medical intervention. Regrettably, there was also an increased risk of mortality in such cases. A recent study conducted in Rivers State, Nigeria, which employed point-of-care creatinine (POC-Cr) technology among adult patients exhibiting high-risk clinical features, demonstrated the feasibility of using this approach to identify cases of community-acquired AKI. Considering this, a similar evaluation using POC-Cr could be conducted in children, serving as a screening tool for those presenting to the Emergency Room, to promptly identify cases of AKI. However, the findings in this study were more favourable in comparison to a report from a Paediatric Intensive Care Unit (PICU) in Ethiopia, where 88.9% of children were documented to have stage III AKI.[36]

While most of the AKI cases at presentation in this study, were community-acquired, the main causes were sepsis, primary kidney diseases particularly AGN and severe falciparum malaria which are preventable. Our findings were consistent with other Nigerian studies.[6,13,17,27,28,35] However, there were variations regarding the commonest causes in other centres within and outside Nigeria. For instance, Anochie and Eke[17] reported gastroenteritis and malaria as the major causes of AKI in Port-Harcourt, Nigeria while Anigilaje et al[27], in Abuja, reported sepsis, acute glomerulonephritis, diarrheal diseases, and severe falciparum malaria as the commonest causes of AKI. In Congo, Assounga et al,[32] documented that acute gastroenteritis, nephrotic syndrome, sepsis, malaria and acute glomerulonephritis were the commonest causes. In contrast, Antwi et al,[33] in Ghana reported that haemoglobinuria, obstructive uropathy and tumour infiltration of the kidneys were the leading causes of AKI in their cohort. Therefore, it is crucial for paediatricians and healthcare workers attending to children in resource-limited settings to actively identify these largely preventable causes of AKI among critically ill children in emergency departments. This proactive approach enables the timely recognition and treatment of those at risk for AKI.

Sepsis was the most common cause of AKI in our cohort, and some of these children developed AKI while on admission at our hospital. Our findings suggest a shift from diarrhoeal dehydration to sepsis as the leading cause of AKI in Rivers State, Nigeria, when compared to an earlier study conducted over a decade ago.[17] While the obvious gains in the control of acute diarrhoeal diseases and the prevention of dehydration or hypovolemia with the use of oral

rehydration therapy for diarrhoeal illnesses over the last decades continue to need sustenance,[37] efforts at recognising children with severe sepsis at increased risk for AKI cannot be downplayed. In addition, because most children with sepsis receive initial treatment with broad-spectrum antibiotics like aminoglycosides (which are known to have nephrotoxic side effects), it is important to recognize the potential harm of prolonged use of these antibiotics, especially when they are not adjusted for the kidney function of children with AKI. This practice should be discouraged. Given that previous studies[38] proved that earlier and appropriate antimicrobial use has been linked to a lower risk of AKI and an increased likelihood of kidney recovery from sepsis-associated AKI, our finding calls for a more dedicated monitoring of AKI among children being managed for sepsis.

Recently, besides creatinine, biomarkers which have potential in detecting subclinical AKI like plasma neutrophil gelatinase-associated lipocalin (NGAL), urine kidney injury molecule–1 (KIM-1), and soluble triggering receptor expressed on myeloid cells 1 (sTREM-1), procalcitonin (PCT), alpha-1-microglobulin, and AKI offer promise for early detection of sepsis-induced AKI and may also be used for monitoring recovery.[39,40] These can be explored in resource-constrained settings to potentially reduce the impact of sepsis-induced AKI. With the use of these biomarkers, though expensive, detection of subclinical AKI will undoubtedly facilitate early intervention and edge us closer to realising the dream of the ISN 0by25 initiative which aims to eliminate all AKI-preventable deaths by 2025.

In this study, kidney replacement therapy was required in about one-sixth of the children with AKI. This proportion was low when compared to the 66% reported in a review on paediatric AKI in sub-Saharan Africa.[1] This was in tandem with the study by Ademola et al[41] in Ibadan, Nigeria but varied from reports by others possibly due to differences in the definition of AKI.[13,17,31,33,34] Therefore, this study supports the proposition by Ademola and colleagues[41] that approximately 20% of patients with AKI in the emergency room may need kidney replacement therapy.

Similar to the report by Anigilaje et al[27] in Abuja, the dialysis access rate in our cohort was 100%. This is probably because when adequate information about the need for dialysis for AKI is provided, the caregivers often strive to make finances available for dialysis. Albeit, in our setting, access was often delayed and almost always not sustainable since self-funding is the norm. While Anigilaje and colleagues attributed their findings to better socioeconomic conditions of residents in Abuja, dialysis accessibility is sub-optimal in many other parts of Nigeria ranging from 24.1%[42] (in an earlier study in Rivers State), 39.1%[43] (Enugu, Southeast Nigeria) to 88.0% (Ogbomosho, Southwest, Nigeria).[44] Most of the children in our series had intermittent HD simply because they were older and caregivers paid out-of-pocket, hence the frequency of HD sessions was often sub-optimal. In our experience, PD was manually improvised and initially funded by generous donations from the managing team/ department of paediatrics. This was necessary because parents were unable to afford all the needed consumables. The challenges of PD we encountered such as peritonitis and PD fluid leaks were similarly reported in other centres in Nigeria.[41,45,46] Our findings underscore the importance of ensuring the availability of essential consumables for acute peritoneal dialysis, including PD fluids, and appropriate-sized PD catheters, by incorporating them into the essential medicines list of publicly funded hospitals. This measure would enhance dialysis access and alleviate the strain on healthcare professionals and caregivers alike.

As regards outcomes observed, one of the two children who had acute PD progressed to AKD but was lost to follow-up. Two patients (both females) progressed to chronic kidney disease; one had lupus nephritis and the other had glomerulonephritis. Seven children (16.3%) with AKI succumbed [one child with overwhelming septicaemia who had PD, two children with complications from AGN and Nephrotic syndrome had HD, and four were managed conservatively for severe acute malnutrition, malaria nephropathy, haemolytic uraemic syndrome and nephrotic syndrome with cellulitis/hypovolaemia]. These deaths were mostly driven by poverty and caregivers' inability to procure recommended medications. Also, children in our setting did not have access to intensive care services due to the non-availability of this service during the time frame of this study, albeit such service would have been very expensive to access even if available. The observed mortality rate aligns with a study in Ibadan, West Nigeria.[6] However, our results are more favourable compared to mortality rates of 20.9%[33] (Ghana), 37.0%[32] (Congo), and 50.7%[31] (Cameroon), reported in other parts of sub-Saharan Africa. Additionally, our

mortality rate compares favourably with rates of 20.83%[34] (Jos) 28.4%[13] (Lagos), 30.2%[27] (Abuja), and 40.5%[17] (Port Harcourt) reported in other studies across Nigeria.

This study identified several critical risk factors for mortality in children presenting to the emergency room with AKI. Sepsis and late presentation, emphasizing the need for prompt sepsis identification and early medical intervention. Likewise, the need for dialysis underscores the importance of close monitoring of children who present with severe AKI complications. Short hospital stay, although not a known risk factor for mortality was significantly associated with mortality in this study possibly reflecting the severity of AKI at the time of presentation and hence the increased risk of dying within a short period of hospitalisation. Overall, this study emphasizes the necessity of recognizing these risk factors to improve outcomes in children with AKI in the emergency room setting.

Limitations of the study

The study is limited because of the inability to screen all children admitted into the CHER of RSUTH for AKI and urine outputs were not completely monitored for all the children. These could have missed some cases which were probably in the early stages of AKI, and so impact on the reported incidence. Also, this was a single-centre study which could affect the generalizability of the findings. Most cases of sepsis in the study were not culture-proven, however, features including fever, marked leucocytosis and features suggestive of multi-system involvement were considered as severe sepsis. Another limitation of this study is the relatively small sample size which may have reduced the power in detecting other risk factors of mortality at multivariate analysis. However, all children with a clinical AKI diagnosis were enrolled and the prospective design of the study remains a strong strength and the findings warrant attention.

Conclusion

Acute kidney injury represents a significant challenge in the paediatric emergency room at Rivers State University Teaching Hospital. Given its high incidence, early recognition and management are crucial to enhancing patient outcomes. Timely identification through screening protocols tailored to the paediatric population is essential, as early intervention can prevent the progression of AKI to more severe stages. This underscores the importance of adequate funding and support for paediatric dialysis services. Establishing and maintaining such service would not only facilitate the prompt initiation of kidney replacement therapy when indicated but also ensure that children with severe AKI receive comprehensive and specialized care, ultimately improving their chances of recovery.

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