British Journal of Healthcare and Medical Research - Vol. 12, No. 01
Publication Date: February 25, 2025
DOI:10.14738/bjhr.1201.18299.
Ajite, A. B., Ogundare, E. O., Subulade, A. A., & Adebisi, A. O. (2025). Aetiology, Clinical Profile and Outcome of Acute Kidney Disease in Children Presenting in a Tertiary Health Institution in Southwest, Nigeria. British Journal of Healthcare and Medical Research, Vol - 12(01). 287-295.

Aetiology, Clinical Profile and Outcome of Acute Kidney Disease in Children Presenting in a Tertiary Health Institution in Southwest, Nigeria

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ABSTRACT

Acute kidney injury (AKI) in children is a disease entity resulting from acute insult to both kidneys leading to reduced glomerular filtration rate, impairment of fluid and electrolyte homeostasis in the body, and accumulation of waste product of metabolism. It is a recognized cause of morbidity and mortality in children. Aim: This study aims to detect the risk factors and prevailing causes of AKI in children presenting at a tertiary hospital in South West Nigeria, identify the various intervention/treatment modalities and determine the predictors of disease outcome. Methods: A retrospective cross-sectional study of children presenting in both the children's emergency unit and children's outpatient clinic of Ekiti State University Teaching Hospital Ado Ekiti, Nigeria with AKI over five years September 2019-September 2024. AKI was defined by reduction in urine output (<0.5mls/kg/hour for older children and urine output of< 1ml/kg /hour in infants) of more than 6hours duration and/or increased serum creatinine of either ≥ 0.3mg/dL or a percentage increase of ≥ 1.5 times baseline. Results: A total of 55 children with a diagnosis of acute kidney injury were seen over a period of 5 years. Twenty-nine (52.7%) of them were male with male to female ratio of 1.1:1. The age range was 1 month to 18 years. AKI was seen more in preschool-aged children of less than 3years (32.7%), severe dehydration and sepsis were majorly implicated while AKI secondary to haemoglobinuria was more prevalent among school-aged

children. Forty-six (83.6%) children presented with oliguric AKI. Thirty-five (63.6%) children with AKI were conservatively managed, haemodialysis was done in 12(21.8%) patients while peritoneal dialysis was done in 3(5.5%) patients. The mortality was 16.4% (9patients). Statistically significant factors that contributed to the outcome included the age of the patients (p=0.046), the aetiology of AKI (p=0.038), and modality of treatment intervention (p=0.010). Conclusion: Sepsis, severe dehydration and haemoglobinuria were major risk factors for AKI in this study especially in younger children. Primordial prevention of risk factors for AKI and being proactive may stop the progression to acute tubular necrosis and the need for renal replacement therapy.

Keywords: Acute kidney injury, primordial prevention, haemoglobinuria.

INTRODUCTION

Acute kidney injury (AKI) in children is a disease entity resulting from acute insult to both kidneys leading to reduced glomerular filtration rate, impairment of fluid and electrolyte homeostasis in the body, and accumulation of waste product of metabolism [1,2]. The Kidney Disease Improving Global Outcome (KDIGO) defined AKI using change in serum creatinine as a rise in serum creatinine ≥ 0.3 mg/ d L within 48hours, rise in serum creatinine ≥1.5 times baseline, which is known or presumed to have occurred within the prior seven days and or urine output<0.5ml /kg/hour for 6 hours [2]. AKI in children is often one of the preventable causes of morbidity and mortality.

The aetiologies of AKI in developed countries tilt towards hospital-acquired causes, usually post-surgical operation, malignancy and nephrotoxins, however in developing countries, hypovolaemia from severe dehydration and sepsis have been implicated [3,4,5]. There are new early biomarkers of acute kidney injury such as Cystatin C, NGAL, KIM-1, IL-18, TIMP-2 and IGFBP7, urinary calprotectin, URBP4, L-FABP, and clusterin [6], however, creatinine which is laden with various limitations as a marker of AKI is still routinely used in most health institutions. These shortfalls of serum creatinine include the fact that the serum level of creatinine is affected by renal tubular secretion, fluid balance, muscle mass, and medications [7]. The delay in appreciable rise in serum creatinine in instances of AKI makes accurately timing of the outset of injury and accessing the severity of kidney injury difficult [8]. It has been estimated that the rise in serum creatinine in acute kidney injury is noticed following a 50% reduction in glomerular filtration rate [9]. It is therefore imperative to identify children with risks of developing AKI early; this will aid prompt intervention, stop the progression to acute tubular necrosis and need for renal replacement therapy which is not readily available and affordable in developing countries [10]. The approach of the primordial level of prevention which entails early recognition of the risk factors and proactively nipping them in the bud will reduce the risk of acute tubular necrosis in which the renal parenchyma become compromised and the need for renal replacement therapy is inevitable.

AIM

This study aims to detect the prevailing aetiologies of AKI in children presenting at a tertiary hospital in South West Nigeria (Ekiti State University Teaching Hospital Ado Ekiti), this will allow primordial prevention and proactive management of such patients in a developing country and to identify the various intervention/treatment modalities and predictors of outcome of the disease.

METHODOLOGY

This was a retrospective cross-sectional study that entailed the review of the data obtained from the case notes of children presenting with features of AKI in both the Children's Emergency Unit (CEU) and Children's Outpatient clinic (COU) of Ekiti State University Teaching Hospital, a tertiary health care facility located in Ado Ekiti, an urban city in Nigeria. The hospital serves as a referral centre for the secondary as well as the primary health care facilities majorly within the state. The CEU and COU serve as the portal of entry for ill children excluding neonates presenting in the hospital facility. The diagnosis of acute kidney injury was based on reduction in urine output (<0.5mls/kg/hour for older children and urine output of< 1ml/kg /hour in infants) of more than 6hours duration and/or increased serum creatinine of either ≥ 0.3mg/dL or an increase of ≥ 1.5times baseline. Eligible children were recruited over five years September 2019-September 2024. Serum creatinine was determined by using auto analyzer that operates based on enzymatic method [11]. Ethical approval was obtained from the hospital ethical committee to access the case notes. The data obtained was analyzed using SPSS version 16. Descriptive statistics showing frequencies and percentages were used. The Independent Samples t-test was used to compare the mean age of both male and female patients. The Chi-Square Test was used to explore the relationship between categorical variables. P-value < 0.05 was considered statistically significant.

RESULTS

A total of 55 children with a diagnosis of acute kidney injury were seen over a period of 5 years. The total number of children admitted during this period was 5203 and AKI therefore constituted 1.1% of the total admission. Twenty-nine (52.7%) of them were male with male to female ratio of 1.1:1. The age range was 1 month to 18 years. The frequency of occurrence of AKI across the age groups is shown in Figure 1, AKI was seen more in preschool 2 aged children of less than 3 years (32.7%).

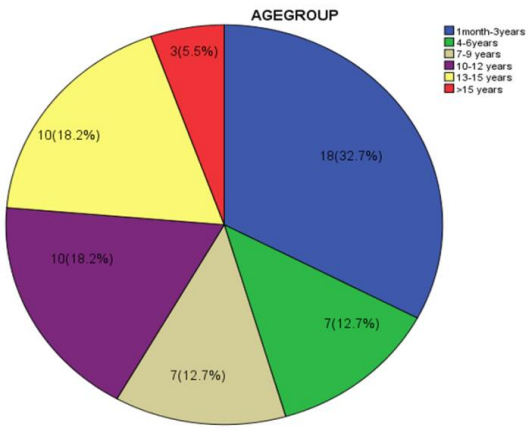

Figure 1

The aetiologies of AKI across the various age groups are displayed in Table 1. It showed that AKI secondary to sepsis and severe dehydration were most prevalent among children of agegroup1 month to 3 years while AKI with haemoglobinuria was more prevalent among school aged children. Five patients with background sickle cell anaemia had AKI secondary to haemoglobinuria.

URL: http://dx.doi.org/10.14738/bjhr.1201.18299.

Forty-six (83.6%) patients presented with oliguria, 5(9.1%) patients had non-oliguric AKI, and the diagnosis of AKI in them was based on the rise in serum creatinine alone. Only 1 patient presented with anuria (urine output less than 0.039ml/kg). Three patients presented with polyuria at admission and the diagnosis of AKI was based on rise in serum creatinine.

In the index study, 35 patients (63.6%) were managed conservatively in terms of fluid restriction to insensible fluid loss of 300-400mls/ m^2(body surface area) + the previous day's urine output volume, low protein diet, withholding sources of potassium, bed rest, treating the underlying cause, antibiotics for sepsis, strict input and output monitoring, 10% dextrose +intravenous aminophylline. Those that had haemodialysis done were 12(21.8%) while peritoneal dialysis was the least intervention practiced.

Nine (16.4%) of the 55 patients died, making the mortality to be 16.4% and 6 (66.7%) of them presented with oligo-anuria. Twenty-seven patients (49.1%) were discharged home, 14(25.5%) were referred to other centres to access dialysis, when the dialysis machines in the study centre were temporarily out of service and 5(9.1%) left the hospital against medical advice. Twenty patients out of those discharged home were managed conservatively without renal replacement therapy while 5 had haemodialysis and 2 had peritoneal dialysis.

Table 2 showed the association between the age group of patients, the diagnosis, the intervention and the outcome of care, either they were discharged, referred, died or left the hospital against medical advice. Most (44.4%) of the patients that were successfully discharged home belonged to the agegroup 1 month to 6years, followed by 22.2% of 4 years to 6years, while 60% of patients that left against medical advice were of ages 10 to 12 years. This is statistically significant (p=0.046). All patients (100%) with drug-induced AKI and post-surgery (Exploratory laparotomy (EX-LAP)) AKI were discharged home after care, while AKI in sickle cell anaemic patients formed the highest proportion (40%) of those who left against medical advice followed by 15.4% of AKI on CKD, and this was also statistically significant (p=0.038). Patients that had peritoneal dialysis as their intervention had the highest proportion (66.7%) of hospital discharge after care followed by 57.1% of patients that were managed conservatively. Twenty five percent of those that had haemodialysis left the hospital against medical advice, and this was statistically significant (p= 0.010).

Table 1: Aetiology of AKI Across the Various Age Groups

S/N	Aetiology of AKI	Frequency of Occurrence of AKI Across the Age Groups						
		1month-3years	4-6 years	7-9 years	10-12 years	13-15 years	>15 years	TOTAL N (%)
1	Sepsis	12	0	0	1	1	0	14(25.5)
2	SLE	1	0	0	1	0	0	2(3.6)
3	Severe dehydration	4	0	0	0	0	0	4(7.3)
4	Sickle cell anaemia	0	0	1	3	0	1	5(9.1)
5	G6PD deficiency & haemoglobinuria	1	5	1	2	0	0	9(16.4)
6	Acute glomerulonephritis	0	1	2	0	2	0	5(9.1)
7	Nephrotic syndrome	0	0	0	0	1	0	1(1.8)

8	Drug induced AIN (Alcohol based herbal concortion	0	0	0	0	1	0	1(1.8)
9	AKI ON CKD	0	0	3	3	5	2	13(23.6)
10	Post-surgery (EX - LAP) AKI	0	1	0	0	0	0	1(1.8)
	TOTAL	18	7	7	10	10	3	55(100)

Table 2: Factors Associated with The Outcome of Care

AGEGROUP	**OUTCOME**				**STATISTICAL INDEX**
	DISCHARGED HOME (%)	**REFERRED (%)**	**DIED (%)**	**LEFT AGAINST MEDICAL ADVICE (%)**	
1month-3years	12 (44.4)	3(21.4)	2(22.2)	1 (20.0)	LR = 27.400
4-6years	6(22.2)	1(7.1)	0(0.0)	0(0.0)	p=0.046
7-9 years	2(7.4)	1(7.1)	4(44.4)	0(0.0)	
10-12 years	4(14.8)	2(14.3)	1(11.2)	3(60.0)	
13-15 years	2(7.4)	5(35.8)	2(22.2)	1(20.0)	
>15 years	1(3.8)	2(14.3)	0(0.0)	0(0.0)	
Total	27(100.0)	14(100.0)	9(100.0)	5(100.0)	
DIAGNOSIS					
AKI SECONDARY TO SEPSIS	9(33.3)	1(7.1)	3(33.3)	1(20.0)	LR = 41.37
AKI WITH SLE	0(0.0)	2(14.3)	0(0.0)	0(0.0)	p = 0.038
AKI SECONDARY TO SEVERE DEHYDRATION	3(11.1)	1(7.1)	0(0.0)	0(0.0)	
AKI IN SICKLE CELL ANAEMIA	2(7.4)	0(0.0	1(11.1)	2(40.0)	
AKI SECONDARY TO HAEMOGLOBINURIA	7(26.0)	1(7.1)	1(11.1)	0(0.0)	
AKI SECONDARY TO ACUTE GLOMERULONEPHRITIS	3(11.1)	2(14.3)	0(0.0)	0(0.0)	
AKI SECONDARY TO NEPHROTIC SYNDROME	0(0.0)	0(0.0)	1(11.1)	0(0.0)	
DRUG INDUCED AKI(AIN)	1(3.7)	0(0.0)	0(0.0)	0(0.0)	
AKI ON CKD	1(3.7)	7(50.0)	3(33.3)	2(40.0)	
POST-SURGERY (EX-LAP) AKI	1(3.7)	0(0.0)	0(0.0)	0(0.0)	
Total	27(100.0)	14(100.0)	9(100.0)	5(100.0)	
INTERVENTION					
CONSERVATIVE MANAGEMENT	20(74.1)	6(42.8)	7(77.8)	2(40.0)	LR = 26.444
HAEMODIALYSIS	5(18.5)	4(28.6)	0(0.0)	3(60.0)	p = 0.010
PERITONEAL DIALYSIS	2(7.4)	0(0.0)	1(11.1)	0(0.0)	
DECLINED DIALYSIS	0(0.0)	0(0.0)	1(11.1)	0(0.0)	
REFERRED	0(0.0)	4(28.6)	0(0.0)	0(0.0)	

URL: http://dx.doi.org/10.14738/bjhr.1201.18299.

TOTAL	27(100.0)	14(100.0)	9(100.0)	5(100.0)	

LR- Likelihood ratio

DISCUSSION

In the index study, community-acquired AKI was more prevalent than hospital-acquired AKI, this agrees with earlier observations that aetiologies of AKI are highly dependent on geographical regions with hospital-acquired AKI being more pronounced in the developed world while sepsis and dehydration are majorly implicated in developing countries [12,13]. This may be explained by the challenges of resource poor countries that make the vicious circle of poverty, malnourishment, ignorance, poor hygiene and disease prevalent.

There appear to be differences in diseases contributing to AKI across various ages. AKI was commonly seen in children of age group 1-3 years in this study and the most implicated causes of AKI in this age group were sepsis and severe dehydration. However, AKI with haemoglobinuria from severe malaria and glucose 6 phosphate dehydrogenase deficiency was more prevalent among children aged 4-6 years while five patients (9.1%) with background sickle cell anaemia had AKI secondary haemoglobinuria all at various ages above or equal to 7years. Previous studies identified malaria and sepsis as common aetiologies of AKI in children without any specification to a particular age group in developing countries [5,12,13]. The younger children are more vulnerable to severe dehydration, which is a risk factor for AKI, they need more fluid for homeostasis as adult level of total body fluid is achieved only after the age of 1 year [14]. This may explain the prevalence of severe dehydration as a cause of AKI in this age. Likewise, the immunity of younger children that is not yet well developed predisposes them to sepsis, an identifiable cause of AKI. Haemoglobinuria is a recognized cause of AKI and has been associated with sepsis, severe malaria, and G6PD deficiency [15]. In the index study, 9 patients had haemoglobinuria from severe malaria and G6PD deficiency while 5 sickle cell anaemic patients with hyperhaemolytic crises had AKI secondary to haemoglobinuria, all of them were of aged 7years and above. Older children with haemoglobinopathy (HBSS) were more at risk of AKI secondary to haemoglobinopathy in this study. It could be that recurrent exposure of the nephron tubules to nephrotoxic haemoglobin over time makes acute tubular necrosis inevitable.

Prevention is necessary in resource poor countries where a panel of biomarkers that may help to establish injury timely and plan appropriate timely interventions are not readily available. It is therefore necessary to be vigilant with high index of suspicion while attending to children with these risk factors. There is a need to advocate for preventive measures, drugs that have the tendency of inducing haemolysis in G6PD deficiency should be prescribed with caution and may be taken off from being part of over-the-counter drugs. Treatment of underlying causes of haemolysis and liberal fluid administration with or without alkalinisation of the urine may prevent acute tubular necrosis [15]; monitoring urine output in such patient is also needful. Good hygiene, environmental sanitation, provision of portable water and oral rehydration therapy are likely to reduce the occurrence of diarrhea as well as severe dehydration while adequate nutrition, food security and appropriate immunization coverage will play key roles in reducing the risk of sepsis. All these preventive measures are part of the Child Survival Strategies [13] which if well implemented will reduce the disease burden.

Oliguric AKI was more prominent in this study. A previous study associated it with a more severe disease process than non-oliguric AKI [16]. However, this cannot be said about index study as majority of the patients had oliguric AKI. It is likely that the underlying cause, duration of oliguria and co-morbidities also contributed to the mortality. Community acquired AKI [17] referring to patients who were previously healthy prior to developing features of AKI that necessitated presentation in the hospital appears to be more associated with oliguric AKI in index study. Oliguria or anuria has been frequently linked with prerenal AKI, acute renal cortical necrosis [18] which are common aetiologies in community acquired AKI. It is therefore necessary to ask about reduction in urinary output while evaluating a patient with risk factors for AKI. Some of the patients with AKI presented with polyuria, especially the ones referred from peripheral hospitals. In these patients, the diagnosis of AKI was based on a rise in serum creatinine which maintained a downward trend to normal value while on admission. Polyuria can be explained by the different phases of AKI [18], i.e. oligoanuric phase, polyuric phase and full recovery. A patient might present in the polyuric phase of AKI at admission, therefore, in the setting of known risk factors for AKI, rise in serum creatinine will be of help in identifying such patient. In the index study, some patients with chronic kidney disease had acute insult to the kidneys from hypovolaema. For these patients, diagnosis of AKI was made based on acute reduction in urine output and sudden rise in serum creatinine. The patients in this category included patients managed for chronic glomerulonephritis, based on haematuria, oedema, high blood pressure and loss of cortico-medullary differentiation on renal ultrasonogram as well as children with delayed diagnosis of congenital anomaly of the kidneys and urinary tracts such as posterior urethral valve (PUV). Zhou et al [19] described a new onset kidney injury (AKI) or acute deterioration of pre-existed chronic kidney disease (CKD) (acute-on-chronic kidney injury, ACKI) as a major contribution to patient's morbidity and mortality. Children with delayed diagnosis of congenital anomaly of the kidney and urinary tract (CAKUT) and consequent CKD have increased morbidity, this can be traced to failure of auto-regulatory as well as compensatory mechanism such as the renin- angiotensin pathway to improve glomerular filtration rate [19]. The reversibility of AKI depends on these mechanisms.

Most of the patients were conservatively managed on the following principles; treatment of identified underlying cause, fluid restriction to what the remnant functioning tubule could cope with i.e., 300-400ml/min /1.73m^2, salt restriction, aminophylline administration which has been associated with improvement in glomerular filtration rate and reduction of serum creatinine [20,21] and other supportive management. Majority of patients managed conservatively responded well to treatment, were discharged home and followed up in the clinic. It could be that these patients were identified early as it is routine in my facility to ask about reduction in urine output and monitor serum creatinine closely once a patient presents with any of the common risk factors of AKI. Early intervention before acute tubular necrosis sets in has been known to improve the outcome of AKI [22]. Three patients had peritoneal dialysis and the low figure is because PD is usually done for children less than 5years of age with indications for dialysis, who may not have adequate vascular access for Haemodialysis. Majority of these younger children had prerenal AKI and they responded to conservative management. Twenty-five of those who had haemodialysis left against medical advice majorly because of financial constraint. The observation is that after 3 sessions of dialysis, most caregivers develop burn out syndrome and they are unwilling to continue haemodialysis until renal recovery. Peritoneal dialysis is more affordable and should be promoted for managing

URL: http://dx.doi.org/10.14738/bjhr.1201.18299.

AKI in children. The limitation commonly encountered from previous study is non-availability of the consumables such as peritoneal dialysis fluids.

In this study, AKI in sickle cell anaemia had the highest proportion (40%) of those who left against medical advice followed by 15.4% of AKI on CKD, these could result from the double burden of the background diseases entity (sickle cell anaemia, and other causes of CKD e.g congenital anomalies of the kidneys and the urinary tract (CAKUT). In most instances the modality of payment of health bill in developing country is via out of pocket. The caregivers seek alternatives to prompt hospital visit, such as; herbal homes, procuring over the counter drug e.t.c. causing delay in diagnosis and prompt care.

CONCLUSION

The younger children are more vulnerable to AKI and the aetiologies vary across the age groups. Severe dehydration, sepsis and malaria complicated by haemoglobinuria are major causes of AKI in them. Primordial prevention of risk factors for AKI across the various age groups and being proactive may stop the progression to acute tubular necrosis and need for renal replacement therapy.

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