



## ORIGINAL ARTICLE

# Haemodialysis for paediatric acute kidney injury in a low resource setting: experience from a tertiary hospital in South West Nigeria

Adanze O. Asinobi<sup>1,2</sup>, Adebowale D. Ademola<sup>1,2</sup>, and Michael A. Alao<sup>3</sup>

<sup>1</sup>Department of Paediatrics, College of Medicine, University of Ibadan, Oyo State, Nigeria, <sup>2</sup>Department of Paediatrics, University College Hospital, Ibadan, Oyo State, Nigeria, and <sup>3</sup>Department of Paediatrics, Bowen University Teaching Hospital, Ogbomosho, Oyo State, Nigeria

Correspondence to: Adebowale D. Ademola; E-mail: dr\_deboademola@yahoo.co.uk

## Abstract

**Background:** Acute kidney injury (AKI) is an important cause of preventable mortality among children. Management of AKI may require renal replacement therapy (RRT) but access to RRT for children in low resource settings is limited. Our study explored the role of haemodialysis in the management of children with AKI in a low resource setting in terms of aetiology and outcomes.

**Methods:** A review of patients managed in the Paediatric Nephrology Unit, University College Hospital Ibadan, South-West Nigeria, who underwent haemodialysis for AKI from January 2006 to December 2014.

**Results:** Sixty-eight patients (55.9% males), aged 3–16 (mean  $\pm$  standard deviation,  $9.0 \pm 3.4$ ) years were studied. The causes of AKI were sepsis (22.1%), malaria (17.6%) and glomerulonephritis (17.6%), intravascular haemolysis—cause unknown (16.2%), G6PDH deficiency (7.4%), malignancy (8.8%) and haemoglobinopathy (5.9%). The number of sessions of haemodialysis ranged from 1 to 10 (mode = 2 sessions) over a period of 1–55 days. Mortality was 27.9% ( $n = 19$ ) and was related to the aetiology of AKI ( $P = 0.000$ ): no deaths among patients with intravascular haemolysis or malaria, six deaths among patients with sepsis (40%), six (50%) among the patients with glomerulonephritis, while all the patients with malignancies died.

**Conclusions:** The outcome of haemodialysis for AKI in Nigeria is relatively good and is related to the underlying aetiology of AKI. In addition to peritoneal dialysis, intermittent haemodialysis may have a role in the management of paediatric AKI in low resource settings and should be supported.

**Key words:** acute kidney injury, haemodialysis delivery systems, international dialysis issues, Nigeria, paediatrics

## Introduction

Acute kidney injury (AKI) is an important cause of morbidity and mortality globally, including in sub-Saharan Africa [1–4]. One of

the ways of limiting the mortality and morbidity associated with AKI is access to renal replacement therapy (RRT). In the developed countries access to renal replacement therapy for patients in AKI is universal, and RRT has evolved over the years

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to include peritoneal dialysis, haemodialysis and continuous RRT [5, 6].

In low resource settings including many parts of sub-Saharan Africa, management of children with AKI who require RRT is a challenge [7–11]. Continuous renal replacement is unavailable in most settings because of cost, non-availability of consumables, need for technical expertise and challenges with power supply. Although haemodialysis can be carried out in all age groups, it is technically more difficult in the very young. Peritoneal dialysis may therefore be the most viable option in young children, because of its technical simplicity and non-requirement of vascular access [7, 10, 12]. Intermittent haemodialysis, however, may also be a feasible option especially in older children [6, 7, 10, 13, 14]. In addition, intermittent haemodialysis may be the preferred option of RRT in the management of conditions such as acute intoxications or poisoning [15].

Data are sparse on outcomes of intermittent haemodialysis performed in children with AKI in sub-Saharan Africa. Outcomes may be influenced by the aetiology of AKI, late presentation, financial implications and limited availability of weight-appropriate consumables. A review of intermittent haemodialysis in low socioeconomic conditions will provide valuable information regarding its role as a mode of RRT for AKI and in designing programmes for reducing mortality in AKI in the sub-region. We therefore reviewed haemodialysis carried out in children and adolescents with AKI in our centre in terms of aetiology of AKI, indications for dialysis and outcomes.

## Materials and methods

The University College Hospital is a tertiary health facility located in Ibadan, the capital city of Oyo State in Southwest Nigeria. Patients come from Oyo State and other parts of the country and include referrals from other hospitals as well as patients who are presenting to a health facility for the first time. The hospital has a paediatric nephrology unit that offers both acute peritoneal dialysis and intermittent haemodialysis [10].

In our centre haemodialysis is carried out in children needing dialysis who weigh 17 kg or above when blood lines and dialysers are available; occasionally we have successfully carried out haemodialysis in children weighing as little as 12 kg, particularly when peritoneal dialysis consumables were not available. In most instances, patients' care-givers paid for haemodialysis whilst in others the haemodialysis was carried out with hospital waivers.

We performed a review of the database of patients who underwent haemodialysis for AKI from January 2006 to December 2014 in the Paediatric Nephrology Unit, University College Hospital Ibadan. The database included patient's age, gender, and clinical features such as anthropometry, blood pressure at presentation, weight and presence of oliguria. It also included laboratory investigation results such as serum urea and creatinine values, indications for dialysis and outcome.

AKI was defined as increase in serum creatinine  $\geq 0.3$  mg/dL within 48 h, or increase in serum creatinine to  $\geq 1.5$  times baseline, which is known or presumed to have occurred within the prior 7 days or urine volume less than  $<0.5$  mL/kg for 6 h. Baseline serum creatinine corresponded to glomerular filtration rate of 120/min as determined by the Schwartz formula [16–18].

Fluid retention was defined based on the presence of oedema and/or heart failure on clinical examination. Hypertension was defined as blood pressure  $\geq 95$ th centile for age, sex and height [19], while oliguria was urine output  $<0.5$  mL/kg/h. Aetiology of AKI was grouped into intravascular haemolysis with

haemoglobinuria, sepsis [20], glomerulonephritis, malignancy, malaria and miscellaneous causes.

Haemodialysis were performed with Fresenius (Fresenius SE & Co. Bad Homburg, Germany), Dialog+ (B. Braun Medical Inc., Melsungen, Germany) or Nipro-surdial (Nipro Medical Industries Co., Gunma, Japan) machines. Dialysis was carried out with F4, F5 or F6 dialyser (Fresenius SE & Co. Bad Homburg) or Gambro Paediatric Dialysers (Baxter, Deerfield, IL, USA). We aimed for the dialyser that had a surface area that was 0.75–1 times the patient's body surface area [21]. Haemodialysis was performed using the femoral venous access. Femoral venous cannulation was carried out with Medcomp 7F single lumen catheters (Medcomp<sup>®</sup>, Harleysville, PA, USA). Extracorporeal blood was usually returned through a 16G intravenous cannula placed in a peripheral vein; in a few children in whom we were not able to get peripheral venous access, blood was returned through a separate femoral catheter. Adult blood lines (Medcomp<sup>®</sup>) were used. When the extracorporeal volume was  $>10\%$  of the patient's blood volume, the dialysis circuit was primed with whole blood [21]. The blood flow rate was usually at 6–8 mL/kg/min [21]. Target ultrafiltration usually did not exceed a maximum of 0.2 mL/kg/min [21, 22]. In patients with marked clinical features of fluid retention, heart failure and pulmonary oedema maximum ultrafiltration was 5–10% of the body weight while monitoring the patient for hypovolaemia and hypotension.

Other management for patients with AKI included treatment of underlying causes or concomitant diseases such as use of antibiotics and antimalarials. Management included fluid restriction to 300–400 mL/m<sup>2</sup>/day and previous days fluid output in patients with oliguric AKI. Patients with packed cell volume  $<17\%$  were usually transfused with packed cells before haemodialysis to raise packed cell volume to  $>17\%$ , otherwise anaemic patients were transfused intradialysis with whole blood. Hyperkalaemia were medically managed with 10% calcium gluconate infusion at a dose of 0.3–0.5 mL/kg given slowly over 30min while monitoring the patient for bradycardia. Additional measures used to treat hyperkalaemia were nebulized salbutamol, intravenous 8.4% sodium bicarbonate or the use of insulin and glucose infusion. Oral antihypertensives and/or intravenous frusemide were indicated in the management of hypertension. Anti-hypertensives are usually omitted on the day of dialysis until after the dialysis. Severe hypertension was managed with intravenous hydralazine 0.1 mg/kg. Hypotension was managed with boluses of normal saline 20–30 mL/kg over 30 min to 1 h. Two patients demonstrated hypotension before commencement of dialysis and both needed vasopressors and received intravenous dopamine. None of the patients required mechanical ventilation prior to haemodialysis. Management of oliguric AKI with fluid retention included use of intravenous frusemide [23].

Patients' clinical status was monitored and dialysis was discontinued based on improvement of urine output, serum creatinine and fluid status. Availability of funds and dialysis consumables were also factors that limited dialysis in some cases, for instance we do not have chronic RRT programmes, and are not yet able to offer chronic RRT to children who develop end-stage renal disease.

The primary outcome measure was mortality while secondary outcome measures were duration of hospital admission and number of sessions of haemodialysis. Patient follow-up was for the period of hospital admission.

## Statistics

Continuous data were summarized as mean  $\pm$  standard deviation, or median and interquartile range. Discrete variables

were summarized as proportions or presented as maximum, minimum and mode. Categorical variables were compared using chi-square tests, Fisher's exact test or Kruskal-Wallis test as appropriate. Means of variables were compared using the Student's t-test. P-values of <0.05 were considered significant. Analysis was carried out using the SPSS IBM version 21 software (IBM Corporation).

### Ethical approval

Ethical approval for the study was obtained from the University of Ibadan/University College Hospital Ibadan Ethical Review Committee. The ethical review committee waived the need to obtain informed consent for the use of the database for the study and the data used in this study are not traceable to individual subjects.

### Results

A total of 68 patients were included in this study. They were aged 3–16 (9.0 ± 3.4) years and there were 38 males (55.9%). They weighed 13–52 (26.0 ± 9.4) kg, and patient distribution according to age classes 0–4, 5–9, 10–14 and 15–20 years, was 8 (11.8%), 31 (45.6%), 24 (35.3%) and 5 (7.4%) patients, respectively. The serum urea before dialysis was 20.1–86.1(41.1 ± 14.3) mmol/L [121–517 (247 ± 86) mg/dL] while serum creatinine was 203–1670 (610 ± 345) µmol/L [2.3–18.9 (6.9 ± 3.9) mg/dL]. The pattern of blood pressure on admission indicated that 29 patients (42.6%) were hypertensive.

### Aetiology of AKI

The main identified causes of AKI were sepsis (22.2%), malaria (17.6%) and glomerulonephritis (17.6%). AKI was associated with massive intravascular haemolysis in 24 patients (35.7%), but in 11 (16.6%) the cause of intravascular haemolysis was unknown. Table 1 shows the aetiology of AKI. Sepsis was associated with ruptured appendix in two patients, and infective endocarditis, meningitis, wound sepsis and pyomyositis in one patient each. Table 2 shows the underlying cause/focus among patients with sepsis. Glomerulonephritis was secondary to acute glomerulonephritis in 10 patients and nephrotic syndrome in two. Malignancy was the cause of AKI in six patients, and was secondary to non-Hodgkin's lymphoma in five patients, including one patient with Burkitt lymphoma. One patient had AKI complicating acquired ventricular septal defect and heart failure, which followed blunt injury to the chest [24].

### Indications for dialysis

The indications for dialysis among the patients are indicated in Table 3. Apart from azotaemia, which occurred in all patients, oliguria (72.1%), electrolyte abnormalities (57.4%), fluid retention (48.5%), neurologic abnormalities (22.1%) and abnormal bleeding tendencies (11.8%) were the main indications for dialysis.

### Management

The number of sessions of dialysis ranged from 1 to 10 sessions per patient with a modal value of two sessions. The modal number of sessions of dialysis for patients with sepsis, malaria and glomerulonephritis (GN) were two, two and five respectively. The number of days on haemodialysis ranged from 1 to 55 days.

**Table 1.** Aetiology of AKI and outcome

Diagnosis	N (%)	Mortality
Sepsis	15 (22.1)	6
Glomerulonephritis	12 (17.6)	6
Malaria <sup>a</sup>	12 (17.6)	–
Intravascular haemolysis — cause unknown <sup>a</sup>	11 (16.2)	–
G6PDH deficiency <sup>a</sup>	5 (7.4)	–
Malignancy <sup>b</sup>	6 (8.8)	6
Haemoglobinopathy <sup>a,c</sup>	4 (5.9)	–
AIHA <sup>a</sup>	1 (1.4)	–
HUS	1 (1.4)	–
Acquired VSD following blunt chest trauma	1 (1.4)	1

AIHA, autoimmune haemolytic anaemia; HUS, haemolytic uraemic syndrome; VSD, ventricular septal defect.

<sup>a</sup>Massive intravascular haemolysis with haemoglobinuria was also a feature in five patients with malaria and one of the patients with haemoglobinopathy as well as in all patients with G6PDH deficiency and autoimmune haemolytic anaemia.

<sup>b</sup>Five patients with non-Hodgkin's lymphoma and one patient with recurrent and metastatic Wilms tumour and obstructive uropathy.

<sup>c</sup>Three patients with haemoglobin S, and a patient with haemoglobin SC.

**Table 2.** Underlying cause/focus of sepsis and outcomes

Underlying cause/focus of sepsis	N	Mortality
Unidentified cause	6	3
HBV infection	1	–
HIV	1	–
Lassa fever	1	–
Infective endocarditis	1	1
Meningitis	1	–
Ruptured appendix with peritonitis	1	1
Enterocutaneous fistula—post exploratory laparotomy, pelvic abscess, ruptured appendix	1	1
Wound sepsis	1	–
Pyomyositis	1	–

HBV, hepatitis B virus; HIV, human immunodeficiency virus infection.

**Table 3.** Gender, hypertension and indication for dialysis among patients with AKI who underwent haemodialysis at the paediatric nephrology unit University College Hospital Ibadan compared with outcome

Parameter	n (% of N = 68)	Mortality (% of n)	P
Gender			
Male	38 (55.9)	10 (26.3)	0.737
Female	27 (44.3)	8 (29.6)	
Comorbidity			
Hypertension	29 (42.6%)	10 (34.5)	0.3
Indication for dialysis			
Azotaemia	68 (100)	17 (27.9)	
Oliguria	49 (72.1)	11 (22.4)	0.105
Fluid retention	33 (48.5)	14 (42.5)	0.010 <sup>a</sup>
Electrolyte abnormalities <sup>b</sup>	37 (54.4)	12 (32.4)	0.367
Abnormal bleeding	8 (11.8)	3 (37.5)	0.521
Neurologic abnormalities	15 (22.1)	5 (33.3)	0.598

<sup>a</sup>P < 0.05.

<sup>b</sup>Serum sodium <120 mmol/L (120 mEq/L), serum K >6.5 mmol/L (6.5 mEq/L) or serum HCO<sub>3</sub><sup>2-</sup> <15 mmol/L (15 mEq/L).

## Outcome

Forty-nine patients (72.1%) were discharged, while 19 (27.9%) patients died. Two of the discharges were against medical advice. The distribution of mortality by aetiology of AKI was significant ( $P = 0.000$ ). Mortality was recorded only among patients with sepsis (46.7%), GN (50%), malignancy (100%) and the patient with traumatic ventricular septal defect also died, while the other patients survived. Mortality was less among patients who were oliguric compared with those who were not, 22.4 versus 42.1% ( $P = 0.105$ ). Mortality was significantly higher among patients who had fluid retention compared with those who did not, 42.5 versus 14.3% ( $P = 0.01$ ). Mortality did not vary significantly across the various age groups ( $P = 0.424$ ). Table 3 compares gender, hypertension and indications for dialysis with mortality.

## Duration of admission

The duration of hospital admission ranged from 2 to 125 days [median 20.5 (interquartile range 15.0–33.0) days]. There was no significant difference in the duration of hospital admission and the aetiology of AKI ( $P = 0.809$ ).

## Discussion

Our study evaluated Nigerian children and adolescents who received haemodialysis for AKI and found that sepsis (22%), malaria (18%) and glomerulonephritis (18%) were the commonest identified causes of AKI. Haemoglobinuria from massive intravascular haemolysis was an important contributor to AKI (35% of cases). Mortality was relatively low and was related to the underlying cause of AKI. Intermittent haemodialysis is feasible in the management of AKI in Nigerian children and should be used as a modality to reduce mortality from AKI in the sub-Saharan Africa in addition to peritoneal dialysis. To the best of our knowledge our study appears to be the largest series on haemodialysis in paediatric AKI from sub-Saharan Africa.

The pattern of aetiology of AKI among our patients is consistent with reports from other parts of sub-Saharan Africa and Nigeria which have identified sepsis, acute GN and malaria as leading causes of paediatric AKI. In various studies from Nigeria and studies from elsewhere in sub-Saharan Africa, sepsis, malaria and GN accounted for 7.1–41.8, 13.7–42.8 and 13–17.8% of cases of paediatric AKI in various studies from Sub-Saharan Africa [2–4, 11, 25]. On the other hand, massive intravascular haemolysis with haemoglobinuria has been reported in 6–42.8% of AKI patients in reports from India and sub-Saharan Africa [25–27]. The intravascular haemolysis is frequently due to malaria, G6PDH deficiency or haemoglobinopathy in these studies. Early presentation, treatment of the underlying cause where possible, urine alkalization and appropriate fluid and diuretic management may prevent AKI secondary to haemoglobinuria [28].

The aetiology of AKI in developed countries has expanded to include AKI due to advancements in cardiac surgery, solid organ and bone marrow transplantation and malignancies [29–32]. In Kuwait 50% of the children with AKI had haematological malignancies [33]. In our setting cardiac surgery and solid organ and bone marrow transplantation are not routinely available, so we do not have causes secondary to such advancements in medical care, and as such most of our patients were not critically ill. In addition, haemolytic uraemic syndrome, which is a common cause of AKI in many other parts of the world including South Africa and East Africa, is not a common cause among our patients [25, 29, 34–37]. Previous studies from Nigeria have not identified

haemolytic uraemic syndrome as a common cause of AKI among children [2–4, 10]. This may be partly due to the cultural practice of cooking meat for a long period that is prevalent in Nigeria.

Haemodialysis in paediatric AKI was associated with 72% survival. Studies from developed countries showed overall survival following RRT in AKI to range from 52 to 58% while outcome of haemodialysis (HD) in AKI ranged from 73 to 90% [31, 32, 38, 39]. Survival following intermittent haemodialysis among our patients is relatively good when compared with reports on RRT for AKI from other developing countries, which ranged from 19 to 67% [37, 40, 41]. The outcome of HD for AKI in our study is comparable to the range of 59–78% for RRT in Nigerian children with AKI [7, 10, 12]. Haemodialysis outcome in AKI patients in the present study is also comparable to a previous report on peritoneal dialysis for AKI from our centre in which 70% of patients survived until discharge.

Factors associated with mortality among our patients included the underlying cause of AKI, and patients with an irreversible cause of AKI were more likely to die than patients who had reversible causes of AKI. For instance all the patients who had malignancies died whereas no mortality was recorded among patients with intravascular haemolysis or malaria. Mortality was also high among patients with glomerulonephritis; this was probably because the patients with glomerulonephritis may have had rapidly progressive glomerulonephritis, and are more likely to become dialysis dependent. The absence of chronic RRT might have contributed to the high mortality among patients with glomerulonephritis [10, 30]. Fluid retention was significantly associated with mortality among our patients and this is may be consistent with studies that have associated fluid overload with mortality in patients who receive RRT for AKI [42–44]. Mortality was also recorded among patients in whom AKI was secondary to heart disease, and haemodynamic instability may have contributed to mortality in these patients [32]. However, haemodynamic instability was not a prominent feature in many of our patients. The very high mortality among patients with AKI secondary to malignancies is also related to the underlying illness, with late presentation and limited access to definitive and optimal supportive treatment for children with malignancies.

Many Nigerian studies have documented the low dialysis access rate in children with AKI [2–4, 11]. Our study indicates that intermittent haemodialysis is a feasible option for reducing preventable deaths from AKI in low resource settings, especially in settings with established haemodialysis units and paediatric nephrologists. There are about 100 centres with haemodialysis units in Nigeria, although most units are small and many have fewer than 5–10 haemodialysis machines. There are also about 26 paediatric nephrologists in the country and virtually all of them are located in 15 of the centres with haemodialysis units. However, while intermittent haemodialysis has long been established as a mode of therapy for adults with AKI in our setting, the same services need to be made more available to children [45].

Challenges that will need to be overcome to provide paediatric haemodialysis include cost and access to weight-appropriate consumables such as dialysers, blood lines and catheters. Consumables for paediatric haemodialysis are not readily available in many parts of sub-Saharan Africa. We did not have regular supply of paediatric size dialysers, in addition we did not have paediatric and neonatal blood lines. Haemodialysis was therefore carried out using adult blood lines. Modifications had to be made in very young children, such as sometimes priming the dialysis circuit with blood if the extracorporeal volume was >10%. Ready

access to weight-appropriate blood lines and dialysers will allow us to carry out routine haemodialysis in young children. This will particularly be relevant in, for instance, situations of AKI secondary to poisoning such as diethylene glycol poisoning where haemodialysis is the RRT of choice. There was an epidemic of diethylene glycol poisoning among toddlers in Nigeria in 2008 in which about 88 out of 89 affected children died and non-availability of paediatric haemodialysis appeared to have contributed to their mortality [15]. However, especially in many young children with AKI peritoneal dialysis will be a feasible way of treating AKI, and both haemodialysis and peritoneal dialysis should be promoted in the management of paediatric AKI in developing countries.

In our centre haemodialysis may be relatively cheaper than peritoneal dialysis unlike in developed countries. The cost of peritoneal dialysis increases with the weight and therefore the age of the patient, mainly because bigger children will require more peritoneal dialysis fluid. The cost of acute peritoneal dialysis, in our centre, will range from 75 to 220 USD (15 000–43 750 Nigerian Naira) in a child who weighs about 10 kg to about 175–300 USD (35 000–60 000 Nigerian Naira) in a 17 kg patient depending on the prevailing local cost of continuous ambulatory peritoneal dialysis (CAPD) fluid. Cost of CAPD fluid tends to vary between 8 and 18 USD (1500–3500 Nigerian Naira) for a 2 L bag, and we have estimated cost of provision of acute peritoneal dialysis over a period of 5 days. On the other hand, haemodialysis for paediatric patients seen in our hospital is half the cost in adult patients and is currently at about 70 USD (13 750 Nigerian Naira) per session, so the cost of about two sessions of haemodialysis will be comparable to the cost of peritoneal dialysis in young children. Similarly, Obiagwu in Kano Nigeria found that the cost of haemodialysis, carried out mostly in children >5 years, was not significantly higher than peritoneal dialysis [46]. In their study peritoneal dialysis was usually carried out in younger children. Many times however, parents are unable to afford the cost of haemodialysis or peritoneal dialysis. Efforts to make both modalities more affordable are needed to improve access to dialysis in AKI in many parts of sub-Saharan Africa.

Government should support the cost of intermittent haemodialysis for children with AKI for example through waivers, subsidies or medical insurance. Many of our patients recovered with one to five sessions of haemodialysis. The cost of one to five sessions of haemodialysis for a potentially reversible illness is not too prohibitive and should therefore be made affordable. Continued advocacy at both governmental and non-governmental levels for paediatric haemodialysis consumables to be available locally is necessary. Training of nursing staff in paediatric haemodialysis is required, while training of more paediatric nephrologists will also be needed. The International Society of Nephrology and International Paediatric Nephrology Association currently have fellowship programmes for training paediatric nephrologists who practice in low resource settings; such programmes should be sustained. In addition, guidelines on haemodialysis including anticoagulation in the paediatric patients are available, which can also guide paediatric haemodialysis treatment in low resource settings [21, 22, 47].

Paediatric haemodialysis will also be relevant in other countries in sub-Saharan Africa as the pattern of aetiology of AKI is similar across the countries with sepsis, malaria and glomerulonephritis as common causes of AKI [2–4, 11, 25, 26, 48]. Although many countries in sub-Saharan Africa may not have the same number of paediatric nephrologists or haemodialysis units as in Nigeria, in many of these countries there is some form of haemodialysis available for adult patients, particularly in tertiary

centres. Paediatric intermittent haemodialysis may also be relevant in these regions, particularly where paediatric nephrologists work in tertiary institutions with facilities for adult haemodialysis. Our study also underscores the fact that haemodialysis will have to be combined with other modalities for the prevention and management of AKI. Additional efforts will include education of medical practitioners and health workers about risk factors, treatment, referral and prevention of AKI. Attention may need to be paid to meticulous management of fluid balance in children and adolescents with AKI. There will be need for peritoneal dialysis particularly in young patients, and haemodynamically unstable patients. Our study however, indicates that haemodialysis is also a feasible option for reducing preventable deaths from AKI in low resource settings, especially in regions where there are paediatric nephrologists working in centres that have haemodialysis units. Availability of both haemodialysis and peritoneal dialysis for paediatric AKI will go a long way in actualizing the global goal of zero preventable deaths from AKI by the year 2025 [49].

In conclusion, we reviewed outcome of haemodialysis in children who were in AKI in our centre. Sepsis, malaria and glomerulonephritis were the commonest causes of AKI. Survival was relatively good, and outcome was related to the aetiology of AKI. Intermittent haemodialysis is a feasible option, in addition to peritoneal dialysis, for improvement of outcomes of paediatric AKI in sub-Saharan Africa. Paediatric haemodialysis should be supported in low resource settings through access to appropriately sized consumables and training of nurses in centres that have paediatric nephrologists and haemodialysis units.

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## Conflict of interest statement

The authors have no conflict of interest to declare and the results presented in this paper have not been published previously in whole or in part, except in an abstract format.

## References

- Li PK, Burdmann EA, Mehta RL. Acute kidney injury: global health alert. *Kidney Int* 2013; 83: 372–376
- Olowu WA, Adelusola KA. Pediatric acute renal failure in southwestern Nigeria. *Kidney Int* 2004; 66: 1541–1548
- Anochie IC, Eke FU. Acute renal failure in Nigerian children: Port Harcourt experience. *Pediatr Nephrol* 2005; 20: 1610–1614
- Esezobor CI, Ladapo TA, Osinaike B et al. Paediatric acute kidney injury in a tertiary hospital in Nigeria: prevalence, causes and mortality rate. *PLoS One* 2012; 7: e51229
- Fleming GM. Renal replacement therapy review: past, present and future. *Organogenesis* 2011; 7: 2–12
- Warady BA, Bunchman T. Dialysis therapy for children with acute renal failure: survey results. *Pediatr Nephrol* 2000; 15: 11–13
- Anochie IC, Eke FU. Paediatric acute peritoneal dialysis in southern Nigeria. *Postgrad Med J* 2006; 82: 228–230
- Olowu WA. Renal failure in Nigerian children: factors limiting access to dialysis. *Pediatr Nephrol* 2003; 18: 1249–1254
- Esezobor CI, Oniyangi O, Eke F. Paediatric dialysis services in Nigeria: availability, distribution and challenges. *West Afr J Med* 2012; 31: 181–185

10. Ademola AD, Asinobi AO, Ogunkunle OO et al. Peritoneal dialysis in childhood acute kidney injury: experience in southwest Nigeria. *Perit Dial Int* 2012; 32: 267–272
11. Esezobor CI, Ladapo TA, Lesi FE. Clinical profile and hospital outcome of children with severe acute kidney injury in a developing country. *J Trop Pediatr* 2015; 61: 54–60
12. Esezobor CI, Ladapo TA, Lesi FE. Peritoneal dialysis for children with acute kidney injury in Lagos, Nigeria: experience with adaptations. *Perit Dial Int* 2014; 34: 534–538
13. Ladapo TA, Esezobor CI, Lesi FE. Pediatric kidney diseases in an African country: prevalence, spectrum and outcome. *Saudi J Kidney Dis Transpl* 2014; 25: 1110–1116
14. Just PM, Riella MC, Tschosik EA et al. Economic evaluations of dialysis treatment modalities. *Health Policy* 2008; 86: 163–180
15. Akuse RM, Eke FU, Ademola AD et al. Diagnosing renal failure due to diethylene glycol in children in a resource-constrained setting. *Pediatr Nephrol* 2012; 27: 1021–1028
16. Schwartz GJ, Gauthier B. A simple estimate of glomerular filtration rate in adolescent boys. *J Pediatr* 1985; 106: 522–526
17. Schwartz GJ, Haycock GB, Edelmann CM Jr et al. A simple estimate of glomerular filtration rate in children derived from body length and plasma creatinine. *Pediatrics* 1976; 58: 259–263
18. Zappitelli M, Parikh CR, Akcan-Arikan A et al. Ascertainment and epidemiology of acute kidney injury varies with definition interpretation. *Clin J Am Soc Nephrol* 2008; 3: 948–954
19. National High Blood Pressure Education Program Working Group on High Blood Pressure in C, Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics* 2004; 114 (2 Suppl 4th Report): 555–576
20. Goldstein B, Giroir B, Randolph A et al. International pediatric sepsis consensus conference: definitions for sepsis and organ dysfunction in pediatrics. *Pediatr Crit Care Med* 2005; 6: 2–8
21. Strazdins V, Watson AR, Harvey B et al. Renal replacement therapy for acute renal failure in children: European guidelines. *Pediatr Nephrol* 2004; 19: 199–207
22. Donckerwolcke RA, Bunchman TE. Hemodialysis in infants and small children. *Pediatr Nephrol* 1994; 8: 103–106
23. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney Int Suppl* 2012; 2: 1–138
24. Ogunkunle OO, Duru CO, Omokhodion SI et al. Acquired ventricular septal defect: a rare sequel of blunt chest trauma in a 7-year-old boy. *Niger J Clin Pract* 2015; 18: 297–299
25. Aloni MN, Nsibu CN, Meeko-Mimaniye M et al. Acute renal failure in Congolese children: a tertiary institution experience. *Acta Paediatr* 2012; 101: e514–e518
26. Adu D, Anim-Addo Y, Yeboah ED et al. Acute renal failure in Ghanaian children. *J Trop Pediatr* 1984; 30: 36–39
27. Srivastava RN, Bagga A, Moudgil A. Acute renal failure in north Indian children. *Indian J Med Res* 1990; 92: 404–408
28. Andreoli SP. Acute renal failure: clinical evaluation and management. In: Avner ED, Harmon WE, Niaudet P (eds). *Pediatric Nephrology*, 5th edn. Philadelphia: Lippincott Williams & Wilkins, 2004, pp. 1231–1251
29. Bailey D, Phan V, Litalien C et al. Risk factors of acute renal failure in critically ill children: a prospective descriptive epidemiological study. *Pediatr Crit Care Med* 2007; 8: 29–35
30. Moghal NE, Brocklebank JT, Meadow SR. A review of acute renal failure in children: incidence, etiology and outcome. *Clin Nephrol* 1998; 49: 91–95
31. Williams DM, Sreedhar SS, Mickell JJ et al. Acute kidney failure: a pediatric experience over 20 years. *Arch Pediatr Adolesc Med* 2002; 156: 893–900
32. Bunchman TE, McBryde KD, Mottes TE et al. Pediatric acute renal failure: outcome by modality and disease. *Pediatr Nephrol* 2001; 16: 1067–1071
33. Ghani AA, Al Helal B, Hussain N. Acute renal failure in pediatric patients: etiology and predictors of outcome. *Saudi J Kidney Dis Transpl* 2009; 20: 69–76
34. Hui-Stickle S, Brewer ED, Goldstein SL. Pediatric ARF epidemiology at a tertiary care center from 1999 to 2001. *Am J Kidney Dis* 2005; 45: 96–101
35. Moghal NE, Embleton ND. Management of acute renal failure in the newborn. *Semin Fetal Neonatal Med* 2006; 11: 207–213
36. Shimelis D, Tadesse Y. Clinical profile of acute renal failure in children admitted to the department of pediatrics, Tikur Anbessa Hospital. *Ethiop Med J* 2004; 42: 17–22
37. Otukesh H, Hoseini R, Hooman N et al. Prognosis of acute renal failure in children. *Pediatr Nephrol* 2006; 21: 1873–1878
38. Goldstein SL. Overview of pediatric renal replacement therapy in acute kidney injury. *Semin Dial* 2009; 22: 180–184
39. Maxvold NJ, Smoyer WE, Gardner JJ et al. Management of acute renal failure in the pediatric patient: hemofiltration versus hemodialysis. *Am J Kidney Dis* 1997; 30 (5 Suppl 4): S84–S88
40. Krishnamurthy S, Mondal N, Narayanan P et al. Incidence and etiology of acute kidney injury in southern India. *Indian J Pediatr* 2013; 80: 183–189
41. Vachvanichsanong P, Dissaneewate P, Lim A et al. Childhood acute renal failure: 22-year experience in a university hospital in southern Thailand. *Pediatrics* 2006; 118: e786–e791
42. Foland JA, Fortenberry JD, Warshaw BL et al. Fluid overload before continuous hemofiltration and survival in critically ill children: a retrospective analysis. *Crit Care Med* 2004; 32: 1771–1776
43. Goldstein SL, Currier H, Graf C et al. Outcome in children receiving continuous venovenous hemofiltration. *Pediatrics* 2001; 107: 1309–1312
44. Lane PH, Mauer SM, Blazar BR et al. Outcome of dialysis for acute renal failure in pediatric bone marrow transplant patients. *Bone Marrow Transplant* 1994; 13: 613–617
45. Bamgboye EL, Mabayoje MO, Odutola TA et al. Acute renal failure at the Lagos University Teaching Hospital: a 10-year review. *Ren Fail* 1993; 15: 77–80
46. Obiagwu PN, Abdu A. Peritoneal dialysis vs. haemodialysis in the management of paediatric acute kidney injury in Kano, Nigeria: a cost analysis. *Trop Med Int Health* 2015; 20: 2–7
47. Davenport A. Anticoagulation options for pediatric hemodialysis. *Hemodial Int* 2003; 7: 168–176
48. Balaka B, Agbere D, Bonkoungou P et al. Post-hemolytic renal failure in children with glucose-6-phosphate dehydrogenase deficiency at the University Hospital Center in Lome. *Med Trop (Mars)* 2003; 63: 151–154
49. Mehta RL, Cerda J, Burdmann EA et al. International Society of Nephrology's Oby25 initiative for acute kidney injury (zero preventable deaths by 2025): a human rights case for nephrology. *Lancet* 2015; 385: 2616–2643