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Case Report

Navigating the Challenges of Managing Acute Kidney Injury in a Sickle Cell Anaemic Adolescent Boy in a Resource-poor Setting - A Case Report

Adebukola Ajite[1,2]*, Ayotunde Ajibola[2] and Samuel Dada[1,2]

[1]Department of Paediatrics and Child Health, College of Medicine, Ekiti State University, Ado Ekiti, Nigeria

[2]Department of Paediatrics, Ekiti State University Teaching Hospital, Ado Ekiti, Nigeria

Abstract

Managing Acute Kidney Injury (AKI) in a patient with Sickle Cell Anaemia (SCA) can be challenging, particularly when the patient is haemodynamically unstable and peritoneal dialysis is not feasible. We report the case of a 17-year-old male with Homozygous SCA (HbSS), diagnosed at age 2years, who presented with AKI and hypertensive emergency following Graham's patch repair for a perforated duodenal ulcer, likely due to chronic NSAID use.

By postoperative day 5, he developed facial and pedal oedema, oliguria, elevated blood pressure (up to 190/140 mmHg), and a rise in serum creatinine (644.4 µmol/L), urea (26.7 mmol/L), and potassium (6.25 mmol/L). A diagnosis of AKI on a background of sickle cell nephropathy was made.

Given the haemodynamic concerns, haemodialysis was initiated cautiously. Measures taken included: (1) priming of dialysis machine and bloodlines with freshly donated genotype-AA blood, (2) intra-dialysis transfusion, (3) limiting the first dialysis session to 2 hours, and (4) ultrafiltration volume based on pre-AKI and current weight.

The patient tolerated three sessions of haemodialysis on alternate days. He experienced remarkable clinical improvement, with a stable blood pressure of 125/70 mmHg, a reduced serum creatinine level of 163.7 µmol/L, and an improved urine output of 3.1 mL/kg/hr. He was discharged on antihypertensives and scheduled for outpatient follow-up.

This case highlights the feasibility and safety of carefully monitored haemodialysis in haemodynamically unstable SCA patients with AKI, even in resource-limited settings.

More Information

***Address for correspondence:**
Adebukola Ajite, Department of Paediatrics and Child Health, College of Medicine, Ekiti State University, Ado Ekiti, Nigeria,
Email: adebukolaajite@yahoo.com

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Introduction

Sickle Cell Disease (SCD) remains a major contributor to childhood mortality, particularly in sub-Saharan Africa, where up to 80% of affected infants may die before the age of five due to limited access to early diagnosis and care [1,2]. Nigeria bears the highest burden globally, accounting for over 30% of annual births of individuals with sickle cell disease [2]. While advances in high-income countries have improved survival through newborn screening and supportive care, morbidity remains high, especially from organ complications [3].

Renal involvement is a serious manifestation of SCD [4]. Sickle cell nephropathy (SCN) encompasses a spectrum of renal complications. It manifests in different forms, including glomerulopathy, proteinuria, haematuria, tubular defects and frequently results in End-stage Renal Disease (ESRD) [5]. It also contributes significantly to SCD-related mortality [5,6]. The pathophysiology involves repeated vaso-occlusion, oxidative stress, medullary ischemia, and nephrotoxic exposures; the potential for nephropathy could be exacerbated by prolonged use of Non-steroidal Anti-inflammatory Drugs (NSAIDs) for pain control [4,7].

NSAID-induced nephrotoxicity and gastrointestinal ulceration are well-documented risks in SCD patients with frequent pain crises [7]. The inhibition of prostaglandin synthesis reduces renal medullary perfusion and may lead

to papillary necrosis and AKI, especially in the context of additional stressors such as surgery or sepsis [8,9].

Management of AKI in SCD poses unique challenges, particularly in haemodynamically unstable patients where fluid restriction may worsen vaso-occlusive crises, and peritoneal dialysis may be contraindicated. This case report describes the successful use of carefully titrated haemodialysis in a haemodynamically unstable adolescent with SCD and AKI, highlighting the clinical considerations and strategies employed in a resource-limited setting.

Case report

A 17-year-old male with homozygous sickle cell anaemia (HbSS), diagnosed at age 2years, presented to the emergency unit with generalised abdominal pain and multiple episodes of vomiting lasting nine hours. The abdominal pain was initially localised to the periumbilical region. There was no associated constipation; however, he passed a small quantity of loose, dark-coloured stool within 24 hours before presentation. No fever was reported, and he had self-medicated with NSAIDs before admission.

He had a history of regular clinic follow-up and was compliant with routine medications, including folic acid, multivitamins, and proguanil. He had eight previous admissions over the past 15 years, primarily for bone pain crises, and had received three exchange transfusions. Analgesic use included NSAIDs, pentazocine, and morphine.

Initial clinical findings showed that he was conscious, in painful distress, mildly pale and icteric, with signs of mild dehydration. There was no facial or pedal oedema. He weighed 60 kg, his height was 1.62 m, pulse rate 104 bpm, blood pressure 110/70 mmHg, respiratory rate 26 cycles per minute and axillary temperature of 37.2 °C. The abdomen was full, tender with guarding, not moving with respiration, and bowel sounds were reduced. A diagnosis of acute abdomen in a known HbSS patient was made. Abdominal ultrasound revealed findings consistent with peritonitis and suspected visceral perforation, along with hepatomegaly. He underwent exploratory laparotomy, where a perforated duodenal ulcer was repaired using a modified Graham's patch technique. The postoperative course was initially stable.

Relevant laboratory findings are as follows; Packed Cell Volume (PCV): 25%, WBC: 15.25 × 10^9/L (Neutrophils 56.6%, Lymphocytes 28.8%, Platelets 861 × 10^9/L, Blood group: O positive, HIV screening: Non-reactive, Malaria parasite: Trophozoites of Plasmodium falciparum seen, Urinalysis: Haemoglobin positive, bilirubin positive, protein + and pH of 5.0.

Postoperative deterioration and renal decline

On postoperative day 4, he developed oliguria (urine output 0.48 mL/kg/hr), facial and pedal oedema, and elevated blood pressure (140/90 mmHg). By day 6, his serum creatinine had risen to 281.3 µmol/L, with progressive increases to 644.4 µmol/L by day 9. Serum urea was also 26.7 mmol/L, and potassium peaked at 6.25 mmol/L (Table 1). He remained afebrile (36.0 °C), but his pulse rate rose to 120 bpm and blood pressure increased to 190/140 mmHg.

A diagnosis of Acute Kidney Injury (AKI) on a background of Sickle Cell Nephropathy (SCN) with hypertensive emergency was made. Hyperkalaemia was initially managed with 10% calcium gluconate. Antihypertensive therapy included intravenous labetalol, oral amlodipine, methyldopa, and torsemide. Potassium-rich medications and IV fluids were discontinued.

Due to persistent oliguria, progressive azotaemia, hyperkalaemia, and poorly controlled hypertension (up to 230/140 mmHg), haemodialysis was commenced.

Dialysis and outcome

Dialysis strategy: Given his haemodynamic instability, careful steps were taken to minimise risks as follows: Dialysis machine and blood lines were primed with freshly donated genotype AA whole blood, and a unit of donor blood was transfused intra-dialysis. The first dialysis session was limited to 2 hours to reduce the risk of dialysis disequilibrium syndrome, while the subsequent sessions lasted 3 hours, with ultrafiltration volumes determined by comparing pre-AKI and current weight. The ultrafiltration rate was kept below 10 mL/kg/hour. Volume of intradialytic transfusion was included in the ultrafiltration calculation. Fluid input during the oligo-anuric phase was limited to insensible loss (400 mL/m^2) plus urine output.

Course and response: He had three dialysis sessions on alternate days, starting postoperative day 6. The procedure was well tolerated. Maintenance fluid was given post-dialysis with 5% dextrose saline to reduce the risk of VOC. Antihypertensives were continued throughout. By day 18, serum creatinine had declined to 163.7 µmol/L, urea to 17.4 mmol/L, and urine output improved to 3.1 mL/kg/hr. Blood pressure stabilised at 125/70 mmHg. He was subsequently discharged on oral antihypertensives and routine SCD medications, with scheduled follow-up in the nephrology and haematology clinics.

Discussion

Advances in the management of Sickle Cell Disease (SCD) have significantly improved life expectancy, resulting in a growing population of adolescents and adults at risk for chronic complications, including Sickle Cell Nephropathy (SCN) [3,10]. Renal involvement in SCD manifests in a spectrum of disorders such as hyposthenuria, hematuria, proteinuria, papillary necrosis, and progression to Acute Kidney Injury (AKI) and Chronic Kidney Disease (CKD) [5]. The index patient was a 17-year-old boy who developed AKI

Table 1: Electrolyte urea and creatinine (blood).

Days Post OP	Potassium	Sodium	Chloride	Bicarbonate	Urea	Creatinine
Pre-operation	4.85 mmol/l	133.9 mmol/l	100.9 mmol/l	20.7 mmol/l	4.1 mmol	77.1 µmol/l
DAY 6	5.83	141.1	106.7	-	26.2	281.3
DAY 9	6.25	139.7	109.7	-	26.7	644.4
DAY 11	4.98	135.2	101.9	-	29.7	391.8
DAY 12	3.87	139.3	102.0	-	24.9	451.1
DAY 16	3.07	134.8	96.6	-	18.0	311.0
DAY 18	2.95	142.6	103.2	-	17.4	163.7
DAY 23	3.38	141.1	108.3	22.4	5.6	91

*- investigation result for serum bicarbonate was not available

following surgical intervention for a perforated duodenal ulcer, likely precipitated by prolonged non-steroidal anti-inflammatory drug (NSAID) use. Considering the age of the patient, long-term survival of SCD patients puts them at risk of chronic complications that involve target organ damage [10]. The various organs, including the kidneys, must have been subjected to the negative impact of recurrent vaso-occlusion and renal function compromise. Chronic NSAID exposure might have further worsened renal function, as non-steroidal anti-inflammatory drugs have been documented to cause renal impairment [8,9].

The Management of AKI in SCD poses unique challenges. First, early recognition is often delayed because baseline serum creatinine levels in SCD patients tend to be deceptively low due to hyperfiltration, a common adaptive response to chronic anaemia [11-13]. Consequently, significant renal impairment may not be immediately apparent, leading to delays in initiating definitive therapy. In the present case, hemodialysis was initiated on the sixth postoperative day, only after a persistent rise in serum creatinine.

Differentiating AKI from an acute-on-chronic decline in kidney function can also be difficult, particularly in patients with undiagnosed or early-stage SCN. Additionally, chronic hemolysis in SCD releases free haemoglobin and iron, contributing to oxidative stress, endothelial injury, and tubular damage, which further exacerbate renal impairment [11]. This was evidenced in the index patient, who presented with hemoglobinuria and features of hyperhemolytic crisis.

Another complexity was fluid management. Balancing fluid restriction—critical in oligo-anuric AKI—with the need for hydration to prevent or manage vaso-occlusive crises proved challenging. The patient required close monitoring of intake and output in addition to individualised and strict fluid replacement calculations based on insensible losses and residual urine output. The decision to initiate haemodialysis was driven by progressive azotaemia, refractory hypertension, hyperkalaemia, and worsening oliguria. Haemodialysis in SCD patients requires additional precautions, as it may precipitate or worsen vaso-occlusive crises due to rapid fluid shifts, hypotension, or hypoxia [11]. These risks were mitigated in this case by: Priming the dialysis circuit with freshly donated genotype-AA blood, performing intra-dialysis transfusion to correct anaemia (PCV 23%) and limiting the first dialysis session to two hours to avoid dialysis disequilibrium. Likewise, the ultrafiltration volume was adjusted based on the patient's pre- and post-AKI weight, as well as the addition of the volume of blood given intra-dialysis to the ultrafiltrate volume. Maintenance of hydration with 5% dextrose saline was also ensured post-dialysis. Boyle, et al. [14] in their review of management of dialysis in sickle cell disease patients recognised anaemia as a unique challenge and suggested the need for considerations and modifications in dialysis of such patients. The index case report showed that intra-dialysis transfusion and adjustment of the ultrafiltrate volume to incorporate the additional volume given will make haemodialysis feasible in the face of anaemia, especially when peritoneal dialysis is contraindicated, as seen in the index patient with previous abdominal surgery. The blood transfused into the patient was freshly donated whole blood and of genotype AA. The aim was to ensure a relative decrease in the circulating haemoglobin S level and reduce morbidity. Previously, a similar approach was suggested as a means of reducing haemoglobin S levels preoperatively in SCD patients for renal transplant to reduce post-transplant complications [15,16].

Vascular access for haemodialysis is another challenge in SCD due to the prothrombotic state characteristic of the disease [11]. All components of Virchow's triad—hypercoagulability, endothelial dysfunction, and impaired blood flow—are present. In this case, the patient was anticoagulated with enoxaparin postoperatively, and dialysis was performed without intradialysis heparin. A temporary double-lumen femoral venous catheter was used, as peripheral venous access was inadequate.

Peritoneal Dialysis (PD), while often considered in younger children with AKI, was not feasible in the index patient due to the previous abdominal surgery and likely compromise of the peritoneal space. Haemodialysis proved to be more practical and effective in this resource-limited setting.

Beyond the clinical considerations, psychosocial and financial implications are significant in the management of SCD, with renal complications imposing a heavy emotional burden on parents and families, often leading to anxiety, depression, and social isolation [17,18]. In many Low - and Middle-income Countries (LMICs), where dialysis costs are borne out-of-pocket, the need for renal replacement therapy can represent catastrophic health expenditure, as most patients cannot afford it [19].

Conclusion

Haemodialysis in adolescents with SCD and AKI is feasible and effective when carefully individualized, even in resource-limited settings. Attention to fluid balance, anaemia correction, infection prevention, and good vascular access is critical to improving outcomes. This case underscores the importance of early recognition of renal complications in SCD and highlights the need for standardized guidelines tailored to this vulnerable population. Further research is warranted to define optimal management strategies and improve long-term renal outcomes in children and adolescents living with SCD.

Ethical approval

Ethical approval was obtained from the Ethics and Research Committee of the institution for the data on the demographics and management of children with kidney disease (PROTOCOL NUMBER – EKSUTH A67/2019/11/008). Informed consent of the parent was obtained before procedures, and confidentiality was also ensured.

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