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Case Report

The Dilemma of Fluid Management in Acute Kidney Injury During Sickle Cell Crises: A Case Report

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Abstract

Fluid therapy remains a cornerstone in the management of sickle cell crises, as adequate hydration reduces blood viscosity and limits further sickling. However, in the presence of oliguric Acute Kidney Injury (AKI), fluid administration becomes a delicate balance to prevent volume overload and worsen morbidity. The case is that of an 8-year-old known sickle cell anemic boy who presented with fever and bone pain for 3 days, jaundice, and passage of dark brown urine. He was pale, icteric, and had non-tender hepatomegaly. He was managed for Vaso-occlusive crisis (VOC) and hyperhaemolytic crisis due to presumed sepsis. He had a blood transfusion and was subsequently placed on intravenous fluids 5% Dextrose in 0.9% Saline at 120% of maintenance. He developed Acute Kidney Injury (AKI) with oliguria (urine output 0.3ml/kg/hr), elevated serum urea (17.70mmol/L), creatinine (241.4µmol/L), and hyperkalemia (K 5.58mmol/L) by the following day on admission. His systolic blood pressure ranged from 140 to 100mmHg, while the diastolic ranged from 100mmHg to 60mmHg. In view of the oliguric AKI, the intravenous fluid was reduced to 2/3rd of maintenance fluid using 5% Dextrose Saline, while the remaining 1/3rd was given orally. He was given furosemide with alkalization of the urine. His urine output and vital signs were closely monitored, and hyperkalemia and hypertension were managed appropriately. By the end of the first week of admission, there was significant improvement in renal parameters, and urine output normalized. The blood pressure at discharge was 90/60mmHg. In conclusion, balancing adequate hydration to prevent further sickling must be weighed against the risk of fluid overload and worsening renal dysfunction in co-existing AKI.

Introduction

Sickle cell disease (SCD) is an autosomal recessive disorder that is seen more in sub-Saharan Africa [1]. Annually, about 300,000 children are born with sickle cell anaemia globally, and 80% of these children are born in Africa [2,3]. SCD occurs as a result of substitution of glutamate for valine at the sixth amino acid of the beta-globin chain, resulting in haemoglobin S (HbS) tetramers. Sickle cell anaemia (SCA) occurs when there is homozygous inheritance of HbS [1]. This haemoglobin S forms crescent-shaped crystals under instances of low oxygen tension, such as tissue hypoxia, oxidative stress, or dehydration. Accumulation of the HbS can lead to red blood cell sickling, early destruction of erythrocytes (haemolysis), and widespread vaso-occlusive episodes (VOC), subsequently resulting in multiorgan damage [4]. Intravascular haemolysis is a life-threatening condition in SCD, and consequent haemoglobinuria can lead to kidney damage as a result of the toxic effects of free haemoglobin on the kidney tubules, leading to acute kidney injury (AKI) [4].

Acute kidney injury is a potentially reversible sudden deterioration in renal function and is assessed by parameters such as urine output and serum creatinine [5]. The Kidney Disease: Improving Global Outcomes (KDIGO) classification of AKI is based on these two parameters [6]. The aetiologies of AKI in SCA are multifactorial and include haemoglobinuria, sepsis, consequent peripheral vasodilation leading to renal hypoperfusion, and recurrent exposure to non-steroidal anti-inflammatory drugs [7,8]. Haemoglobinuria is a potential intrinsic renal cause of AKI. Severe intravascular haemolysis results in a large burden of haemoglobin, which overwhelms

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the binding capacity of haptoglobin as well as the degenerative capacity of heme oxygenase [7,8]. The free haemoglobin is nephrotoxic; therefore, adequate hydration is required to maintain renal perfusion and prevent acute kidney injury, while closely monitoring the red blood cell volume for anaemia. However, in AKI with evidence of acute tubular necrosis (ATN) often manifesting as persistent oliguria, failure to respond to fluid administration and diuretics [7], it is paramount to restrict fluid intake in order to prevent circulatory overload.

Here comes the dilemma in fluid management when a sickle cell anaemic patient presents in crisis with obvious need for increased perfusion to (i) ameliorate the incidence of VOC by improving vascular flow, (ii) improve excretion of free haemoglobin, and preserve renal perfusion. If the same patient has a complication of acute kidney injury with the need for fluid restriction to avoid volume overload, the appropriate fluid prescription might pose a challenge. AKI is usually associated with reduced glomerular filtration rate (GFR), which is a risk factor for increased mortality in SCD [9-11]. Kidney disease has been found to account for approximately 16–18% of overall mortality in patients with SCD [12,13]. This case report suggests a need for meticulous review of the fluid prescription in these patients.

Case report

An 8-year-old boy, being managed for sickle cell anemia, presented with fever and bone pain for 3 days. At admission, he was pale, mildly icteric, passing dark urine, and had hepatomegaly. He developed oliguria while on admission with urine output of 0.3ml/kg/hr and facial oedema. He was first diagnosed at 4-years of age. He had a history of recurrent vaso-occlusive crises with an average of 5 – 8 episodes/ year and suboptimal clinical follow-up. Renal evaluation showed elevated urea and creatinine with hyperkalemia, as shown in Table 1, all consistent with AKI.

Investigation results (Tables 1,2)

Treatment

He was managed for vaso-occlusive crisis (VOC) and hyperhaemolytic crisis presumed to be precipitated by sepsis. He had a blood transfusion. In an attempt to reduce the risk of haemoglobin-induced pigment nephropathy in the index patient, hydration was started at 120% of maintenance using 5% Dextrose saline. Following features of AKI, fluid management proved challenging due to the competing risks of worsening volume overload due to the renal impairment and the need to give adequate fluid to combat VOC. Subsequently, the intravenous fluid was reviewed to daily 100% maintenance fluid, 2/3rd of which was given via intravenous route and the remaining 1/3rd was given as oral. There was a strict input and output chart for close monitoring. Alkalinization of the urine was done by giving the patient intravenous sodium bicarbonate and placing on frusemide 1mg/kg while monitoring the urine output and keeping a urine rack that showed improvement in the urine colour over time. The patient responded to cautious fluid resuscitation and diuretic therapy, with gradual improvement in urine output and renal function. The clinical course was complicated by high blood pressure ranging from systolic 140 to 120mmHg and diastolic 100 to 90mmHg. This was managed with tab aldomet and amlodipine, which were subsequently changed to tab lisinopril on follow-up following resolution of the AKI. The blood pressure at follow up was 90/40mmHg. There was persistence of proteinuria on follow-up, suggesting evolving Sickle Cell Nephropathy.

Table 1: Electrolyte, Urea, and Creatinine (E/U/Cr) progression

E/U/Cr	1ST day of admission	2nd day of admission	4th day of admission	7th day of admission	At discharge
Potassium(mmol/L)	5.58	5.08	4.73	5.78	3.64
Sodium (mmol/L)	131.7	125.5	121.7	125.7	140.1
Chloride (mmol/L)	102	98.5	91.6	99.7	99.0
Urea (mmol/L)	17.7	15.9	23.8	19.34	6.98
Creatinine (µmol/L)	241.4	132.4	152.8	107.0	80.8

Table 2: Results of other investigations.

Day	Urinalysis	Full blood count	RBS	RDTMP	Total serum protein	Serum albumin
At admission	pH 5	WBC 8.56 x 10^9/L	93mg/dL	negative	65.22g/L	36.53g/L
	Haemoglobinuria	Neutrophil 74.1%,				
	Proteinuria +	Lymphocyte 16.6%,				
		Haematocrit 21.9%,				
		Platelets 320 x 10^9 /L				

WBC = White blood cell count, RDTMP = Rapid diagnostic test for malaria, RBS = Random blood sugar

Renal ultrasound showed normal-sized kidneys with preserved cortico-medullary differentiation.

Blood culture yielded no growth.

Discussion

This case highlights the therapeutic challenge of fluid management when there is co-existing AKI and the complex interplay between haemolysis, vaso-occlusion, and renal impairment in a child with sickle cell anaemia. In this patient, the low packed cell volume, jaundice, presence of dark brown urine, oliguria, and biochemical derangements suggest a hyperhaemolytic crisis with intravascular haemolysis and AKI. Free haemoglobin released during haemolysis is nephrotoxic and can precipitate acute tubular necrosis, which may contribute to the development of acute kidney injury [14]. Haemoglobinuria is likely to trigger hydroxyl radical-mediated peroxidation of lipid and acute tubular necrosis [15,16]. The structure of the kidney microvasculature makes the renal medulla susceptible to hypoxia and renal medullary hypoxia from sickling; this may also further promote renal dysfunction [17].

The diagnosis of AKI was made in this patient following the presence of oliguria and rising creatinine, and this necessitated careful monitoring of fluid balance and the introduction of diuretics to promote urine output. Most sickle cell crisis

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guidelines encourage hydration to reduce sickling [5-7,18], but this patient simultaneously developed oliguric AKI where excessive hydration could worsen fluid overload. This created a therapeutic dilemma.

Fluid therapy remains a cornerstone in the management of sickle cell crises, as adequate hydration reduces blood viscosity, limits further sickling, and reduces pain [18]. The routine administration of extra fluid regardless of the individual's state of hydration was advocated to be a useful adjunct mode of intervention [18]. However, in the presence of AKI, fluid administration becomes a delicate balance. Excess fluid may lead to volume overload, worsening hypertension, and risk of pulmonary complications such as acute chest syndrome, while inadequate hydration may perpetuate renal hypoperfusion and ongoing sickling [18]. Distinguishing between the need for fluid resuscitation in SCA and restriction of fluid in associated acute tubular necrosis may be difficult [8], however, close attention to features suggestive of acute tubular necrosis, such as persistent oliguria and elevated fractional excretion of sodium (more than 2%), is helpful [8,9]. In this patient, the decision to reduce intravenous fluid to 2/3rd of maintenance was based on the persistent oliguria following initial hydration and risk of impending volume overload. The remaining 1/3rd of the maintenance fluid was administered orally, as the risk of volume overload is low in this route of fluid administration. The gradual improvement in urine output and decline in serum creatinine suggested recovery from acute tubular injury. The successful outcome following reduction of intravenous fluids while maintaining careful monitoring suggests that individualized fluid prescription may be preferable to routine aggressive hydration in selected patients with sickle cell crisis complicated by AKI.

This case emphasizes that fluid management in sickle cell patients with acute kidney injury may sometimes not be protocol-driven but highly individualized. It requires continuous reassessment of clinical status, urine output, and renal parameters. This is in line with the previous documented guideline, which stated that measures to mitigate renal damage should include daily weighing and ensuring fluid balance [20].

Alkalinization of the urine by giving sodium carbonate combined with a loop diuretic was done in the index patient; this might have contributed to the good outcome in this patient, as previously documented that alkalization is effective in pigment-induced AKI secondary to haemoglobinuria [21]. The reason for this could be attributed to the solubility of pigments like haemoglobin in alkaline urine, thereby limiting the risk of cast and crystal formation as well as kidney damage. The urine alkaline medium also impairs the degradation of haemoglobin to heme and free toxin, which have a nephrotoxic effect [17]. The hypertension in this patient might have been related to volume overload, although other contributors, including acute kidney injury itself, cannot be excluded. Lisinopril, an angiotensin converting enzyme inhibitor, was considered following resolution of the acute kidney injury; this is because of its reno-protective impact by reducing proteinuria in sickle cell anaemic patients [22].

To our knowledge, pusblished reports discussing individualized fluid restriction strategies in children with sickle cell crisis complicated by oliguric AKI remain limited, making this case a useful illustration of the challenges clinicians may face. Multidisciplinary care involving hematologists and nephrologists is essential to navigate these challenges.

In conclusion, the co-existence of sickle cell crisis and AKI presents a therapeutic dilemma and underscores the complexity of fluid management where the clinician must balance the need for adequate hydration to prevent vaso-occlusive crisis against the risks of fluid overload, electrolyte disturbances, and worsening of renal dysfunction. The case report is intended to bridge a gap between clinical knowledge and lifesaving action. Early recognition, judicious fluid selection, and close monitoring are critical in improving outcomes and preventing long-term renal sequelae in sickle cell anaemia.

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