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CASE REPORT



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Severe Hypertriglyceridaemia with Acute Kidney Injury in Paediatric Diabetic Ketoacidosis: A Case Report

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Abstract
Severe hypertriglyceridaemia is an uncommon but clinically significant complication of Diabetic Ketoacidosis (DKA). We report a 14-year-old female with poorly controlled Type 1 Diabetes mellitus who presented with features of DKA complicated by severe hypertriglyceridaemia, hyperviscosity-related neurological impairment and acute kidney injury requiring haemodialysis. Markedly lipaemic plasma observed during exchange blood transfusion prompted lipid evaluation, which revealed significantly elevated plasma triglyceride levels. The clinical management included insulin infusion, haemodialysis, exchange blood transfusion, and supportive care, resulting in a favourable clinical outcome. This case highlights the importance of early recognition and management of hypertriglyceridaemia in DKA, particularly in resource-limited settings.

Keywords: Diabetic ketoacidosis, Haemodialysis, Hyperlipidaemia, Hypertriglyceridaemia, Hyperviscosity.

Introduction

Diabetic ketoacidosis (DKA) is a major acute metabolic emergency in children with Type 1 Diabetes mellitus and remains a significant cause of morbidity and mortality. [1] Mild to moderate dyslipidaemia is frequently observed during DKA; however, severe hypertriglyceridaemia is uncommon in children. [2-4] Severe hypertriglyceridaemia may result in complications including hyperviscosity, neurological impairment and acute kidney injury. [5 – 7]

Reports of severe hypertriglyceridaemia complicating paediatric DKA in sub-Saharan Africa remain limited, suggesting possible under-recognition in resource-constrained settings.[1,2,8] This report describes an African adolescent with diabetic ketoacidosis complicated by severe hypertriglyceridaemia, hyperviscosity-related neurological impairment and dialysis-requiring acute kidney injury. Given the scarcity of similar reports from sub-Saharan Africa, this case highlights the diagnostic challenges and successful management of this rare complication in a resource-limited setting. The objective of this report is to highlight the clinical presentation, diagnostic challenges and multidisciplinary management of severe hypertriglyceridaemia complicating childhood DKA.

Case Presentation

A 14-year-old female adolescent who was diagnosed with Type 1 Diabetes mellitus one year prior presented to the Emergency Paediatric Unit

of the Federal Medical Centre, Jabi, Abuja, Nigeria, with a three-day history of abdominal pain, vomiting, progressive drowsiness and respiratory distress. She had two prior episodes of diabetic ketoacidosis (DKA), including one at diagnosis and another four months before presentation. She resided with her older sister, who was her legal guardian, as her parents had been separated since early childhood, and her father was largely absent. She had a history of poor medication adherence and had reportedly run out of insulin approximately one week before presentation without informing her caregiver. She attained menarche two years prior, with her last menstrual period one month before presentation.

On physical examination, she was moderately dehydrated and afebrile, with a Glasgow Coma Score of 10/15. Her sexual maturity rating was Tanner Stage IV. Respiratory examination revealed Kussmaul's breathing, and she was commenced on improvised continuous positive airway pressure (CPAP). The random blood glucose was 28.6 mmol/L, while urinalysis revealed glycosuria (3+), ketonuria (3+) and proteinuria (2+). In the absence of arterial blood gas analysis, the diagnosis of DKA was based on hyperglycaemia, ketonuria, clinical acidosis and Kussmaul-type respiratory distress. The initial laboratory evaluation was limited by grossly lipaemic samples, with the first serum electrolytes, urea, and creatinine (EUCr) specimen being non-analysable. The clinical management included continuous soluble insulin infusion at 0.1 IU/kg/hour, intravenous fluid resuscitation with isotonic saline, followed by maintenance fluids with potassium supplementation guided by serial electrolyte monitoring. Within the first 24 hours, her level of consciousness deteriorated to a Glasgow Coma Score of 8/15.

Admitting full blood count (FBC) showed leucocytosis (38,000/mm^3) that was attributed to possible sepsis and dehydration in the context of abdominal pain and vomiting with suspicion of surgical acute abdomen. She was reviewed by the surgical team, and a surgical abdomen was clinically excluded. However, the observation of markedly lipaemic plasma following an initial non-analysable blood sample shifted suspicion towards severe hypertriglyceridaemia and possible hyperviscosity.

On the third day of admission, due to persistent neurological impairment and suspicion of hyperviscosity, a single-volume exchange blood transfusion was performed. During this procedure, markedly lipaemic plasma was suspected from the colour of the blood (Figure 1), prompting serum lipid evaluation, which revealed severe hypertriglyceridaemia with plasma triglyceride levels of 29.65 mmol/L and total cholesterol of 21.64 mmol/L.

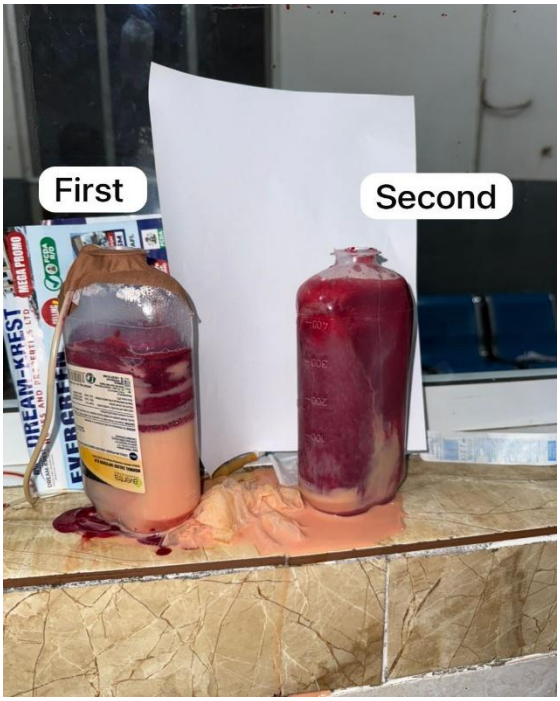


Figure 1: Markedly lipaemic plasma obtained during exchange transfusion. The first bottle (left) contains turbid, chylous plasma; the second bottle (right) contains clearer plasma after the exchange.

Subsequent evaluation of serum electrolytes, urea, and creatinine revealed hyponatremia of 124mmol/L, which rose to 160mmol/L following fluid resuscitation and later normalised after haemodialysis.

Her serum creatinine, measured between the third and fifth days of admission, increased from 420µmol/L to 770µmol/L, with eGFR declining from 13 to 7 mL/min/1.73m^2, consistent with Stage III Acute Kidney Injury (AKI) based on Kidney Disease: Improving Global Outcomes (KDIGO) criteria.

Following this management, her level of consciousness improved rapidly to a GCS of 15/15. She subsequently received three sessions of haemodialysis, following which serum potassium declined from 5.4 mmols/L to 2.7 mmols/L during treatment, necessitating careful monitoring and correction. Urinalysis showed proteinuria (2+ at presentation), which increased to 3+ by day 3 and resolved by day 7. There was no associated generalised oedema or hypoalbuminemia.

The respiratory abnormalities improved progressively, with step-down from CPAP to intranasal oxygen by day 7 and complete weaning off oxygen therapy by day 10 of admission. Insulin infusion was continued for seven days, with progressive reduction in triglyceride levels to 5.09 mmol/L by day 10. She was discharged after 13 days of admission in a stable clinical condition. The laboratory findings are tabulated in Tables I and II.

Ethical considerations:

Written informed consent could not be obtained, but verbal informed consent and assent were obtained, and anonymity was maintained.

Table I: Trend of serum electrolytes, urea, creatinine and estimated glomerular filtration rate

Days on admission	Na (mmol/L)	K (mmol/L)	Cl (mmol/L)	Urea (mmol/L)	Creatinine (µmol/L)	HCO3 (mmol/L)	eGFR (mL/min/1.73m²)
Day 3	124.8	5.3	104.9	22.9	411.5		15.8
Day 5	160.4	5	139.5	32.7	773.6		7.6
Day 6 (Post 1st Dialysis)	151.4	3.03	114	23.85		12	
Day 7 (Post 1st Dialysis)	150.6	3.72	98.8	23.6	545	31	11.4
Day 8 (Post 2nd Dialysis)	136.8	2.7	103	9.7	203.7		35.6
Day 10 (Post 3rd Dialysis)	138.4	2.6	105.5	5.5	105.1	25.4	76.3

Discussion

Although mild serum lipid derangements are frequently observed in childhood DKA, severe hypertriglyceridaemia (HTG) is uncommon. When present, marked elevations (often exceeding 21.6 mmol/L [2000 mg/dL]) are typically seen in the setting of uncontrolled diabetes mellitus and may be associated with underlying familial lipid disorders. [2-4] The pathophysiology of HTG in DKA is primarily driven by insulin deficiency, [7] which leads to increased lipolysis via activation of hormone-sensitive lipase, resulting in elevated free fatty acids. These are subsequently re-esterified in the liver to form triglycerides. Concurrently, reduced activity of lipoprotein lipase impairs the clearance of triglyceride-rich lipoproteins, leading to marked accumulation of chylomicrons and very low-density lipoproteins. [4]

Severe hypertriglyceridaemia may increase plasma viscosity, thereby causing impaired microcirculatory flow and contributing to neurological dysfunction.[2,7,8] Neurological impairments in this case were likely multifactorial. Although cerebral oedema is a recognised complication of DKA, the temporal relationship between clinical deterioration and subsequent improvement following exchange blood transfusion and haemodialysis suggests that hyperviscosity and metabolic derangements contributed significantly to neurologic impairment.

Table II: Trend of lipid profile parameters during admission

Date – Days on admission	Parameter	Value (mmol/L)	Value (mg/dL)
03-07-25 – Day 3	Total Cholesterol	21.64	836.8
03-07-25 – Day 3	HDL	10.78	416.9
03-07-25 – Day 3	LDL	0	0
03-07-25 – Day 3	Triglycerides	29.65	2626.1
08-07-25 – Day 8	Total Cholesterol	7.1	274.6
08-07-25 – Day 8	HDL	0.39	15.1
08-07-25 – Day 8	LDL	1.66	64.2
08-07-25 – Day 8	Triglycerides	6.37	564.2
10-07-25 – Day 10	Total Cholesterol	6.92	267.6
10-07-25 – Day 10	HDL	0.41	15.9
10-07-25 – Day 10	LDL	1.79	69.2
10-07-25 – Day 10	Triglycerides	5.09	450.8

Uraemic encephalopathy was considered less likely, as urea levels did not reach thresholds typically associated with severe neurological dysfunction. The rapid improvement of consciousness after haemodialysis may be explained by correction of metabolic acidosis, removal of circulating toxins, and improved plasma osmolality.

AKI in DKA is multifactorial, resulting from dehydration, renal hypoperfusion and metabolic disturbances. [6] In this case, the severity of renal impairment requiring haemodialysis highlights that significant kidney injury can occur even in the presence of preserved urine output. Pseudohyponatraemia observed in this patient was likely due to interference with sodium measurement caused by severe lipaemia, [7] with correction following dialysis.

The clinical management of severe HTG in DKA is centred on insulin therapy, which enhances lipoprotein lipase activity and promotes triglyceride clearance. [3,4] Exchange blood transfusion may reduce circulating lipid burden and improve plasma viscosity, while haemodialysis contributes to the correction of the metabolic derangements.[11] Prolonged intravenous insulin infusion beyond resolution of DKA has been reported in children with severe hypertriglyceridaemia, with treatment continued for several days to facilitate triglyceride clearance. [12, 13]

Similar cases have been reported predominantly in Asia and the Middle East, with limited reports from Africa, highlighting possible under-recognition in this region. [2-4,7,8,10] This case is notable for the combination of severe hypertriglyceridaemia, hyperviscosity-related

neurological impairment and acute kidney injury requiring haemodialysis in an African adolescent, highlighting the need for heightened clinical suspicion and adaptable management strategies in resource-limited settings.

Limitations

Both poor resources and logistics, including a lack of access to arterial blood gas analysis, pancreatic enzyme assays and lipid genetic studies, limited laboratory investigations. It is also important that baseline investigative data were missed due to grossly lipaemic blood samples. The non-availability of plasmapheresis, a recognised modality for rapid triglyceride reduction in severe triglyceridaemia, further limited the clinical management of the case. [10]

Conclusion

Severe hypertriglyceridaemia should be considered in childhood DKA with atypical features such as lipaemic plasma or unexplained neurological impairment. Early recognition and multidisciplinary management can result in favourable outcomes even in resource-limited settings.

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Conflicts of Interest: None declared.

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