

Symptomatic Hypernatraemia Secondary to Ingestion of Poorly Constituted Oral Rehydration Solution – A Case Report

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Authors' contributions

The work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

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Case Report

ABSTRACT

Aim: Hypernatremia is a recognized complication of poorly constituted Oral Rehydration Solution (ORS) use that can cause neurological manifestations which require careful but often challenging management to prevent morbidity and mortality. Herein we report a case of symptomatic hypernatremia in a 2-year-old boy following ingestion of poorly constituted Oral Rehydration Solution (ORS) and highlight challenges in management.

Presentation of Case: This is a case report of a 2-year-old male who presented with a four-day history of vomiting and fever. He received four litres of hyper-concentrated ORS (1 packet of ORS to 0.7 l of water) prior to presentation. At presentation, he was lethargic and subsequently lapsed into unconsciousness. He also developed focal and generalized seizures. Serial serum electrolytes testing showed hypernatremia which ranged from 157-161 mmol/L in the first 48 hours of admission for which appropriate intravenous fluids were given. Serum sodium levels gradually returned to normal (140-142 mmol/L) with treatment. He subsequently regained full consciousness after four days in a coma and was discharged home in satisfactory clinical condition.

Conclusion: This case is being reported to highlight the important role of proper history taking, careful fluid and electrolyte monitoring in the diagnosis and care of this condition and emphasise on adequate education of caregivers by clinicians and the mass media on appropriate preparation of ORS.

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1. INTRODUCTION

Hypernatremia is a recognized rare complication of Oral Rehydration Solution (ORS) use that can manifest with severe symptoms requiring careful but often challenging management to prevent morbidity and mortality. The common causes of hypernatraemia in children include dehydration from not drinking adequate amounts of water or continuous total body fluid losses from diarrhoea, persistent vomiting, renal losses or severe sepsis [1]. Central nervous system manifestations like lethargy, seizures and coma from brain haemorrhage usually occur at higher levels of serum sodium (> 160mmol/L) and can also occur without dehydration [2,3]. The neurological sequelae arising from neurological complications include seizure disorders, spastic plegias and intellectual deficits [3]. Deaths from hypernatraemia in children can be as high as 20% with neurological complications occurring in 15% [1,3]. ORS is a readily, available and affordable salt admixture used very commonly in developing countries like Nigeria for home and hospital management of mild to moderate dehydration during diarrhoeal illnesses. Improper re-constitution of ORS, such as preparing with incorrect proportions and excessive intake frequency could result in hypernatraemia [3,4]. This condition and resultant consequence is underreported in our settings possibly due to lack of awareness among caregivers and other allied healthcare workers alike [5,6]. Herein we report a case of symptomatic hypernatremia in a 2-year-old boy following ingestion of poorly constituted Oral Rehydration Solution (ORS) and highlight challenges in management.

2. CASE PRESENTATION

A 2-year-old male presented to the children emergency room with a 4-day history of vomiting and fever and a 1-day history of body swelling. He vomited up to 10 times a day and by the 3rd day started vomiting coffee-brown coloured substances. He drunk poorly constituted concentrated ORS (1 packet of ORS to 0.7L of water) and though not tolerated due to repeated bouts of vomiting, was said to have drunk about four litres since onset, as well as received some oral medications purchased from a chemist shop before presenting to the hospital. No history of diarrhoea or reduction in urine volume. On presentation, he was febrile (39°C), mildly pale, had moist buccal mucosa, capillary refill time 3

seconds and no loss of skin turgor, no significant lymph node swellings, SPO₂ was 97% in room air. He was also found to be lethargic, GCS 12/15, tachypnoeic (RR 48 cpm), tachycardia (136 bpm), had non-tender hepatomegaly 4cm below the right costal margin. He lapsed into unconsciousness with GCS ranging from 6-10/15 on the second day of admission and later that day developed initially focal and subsequently generalized seizures.

He was initially admitted for persistent vomiting with severe dehydration and electrolyte derangement? Malaria. Serum electrolytes done showed hypernatraemia Na – 161mmol, potassium 3.8 mmol, bicarbonate 16 mmol/l, urea 6 mmol/l, creatinine 90mmol/l. Malaria Parasite thick film microscopy, random blood glucose, full blood count and urinalysis were normal. He initially received IV fluids 0.9% normal saline 30 mls/kg over 30 minutes and then continued on 4.3% dextrose in 1/5th saline continued as maintenance. With the retrieval of serum electrolyte results, the clinical working diagnosis was subsequently coma secondary to hypernatraemia after a history of ingestion of poorly constituted ORS was further clarified. Brain MRI/CT scan and urine fractional excretion of sodium (FeNa%) requested at the onset of seizures and coma were not done. The goal of fluid administration was then to decrease the serum sodium by <12 mEq/L every 24 hr, a rate of 0.5 mEq/L/hr. Total fluid administered was calculated as follows: For a 12kg child (total body water = 0.6 times body weight) and estimated to have a 10% hypovolaemic loss, with a serum sodium concentration of 161 mEq/L. The following calculations were made: Total body water deficit (in L)= [(current sodium level in mEq/L ÷ 145mEq/L) – 1] x 0.6 x weight (in Kg) = [(161÷145 mEq/L) – 1] x 0.6 x 12 = 0.79L and volume of replacement fluid needed to correct the water deficit by using the concentration of the sodium in the replacement fluid was determined as follows: replacement volume (in L) = TBW deficit x 1÷ [1 – (sodium concentration in replacement fluid in mEq/L ÷ 154 mEq/L)] = 0.79L x 1 ÷ [1-(77÷154)] = 1.58L. This volume was replaced intravenously by using 5% dextrose in half normal saline, manually reconstituted and then afterwards changed to 4.3% dextrose in 1/5th saline at a rate estimated to correct serum sodium over 72hrs. Monitoring of intravenous fluid rates was manually done using a soluset. Serum electrolytes were

monitored twice daily until serum sodium and other electrolyte values normalized. Seizures were not controlled with intramuscular paraldehyde and intravenous phenobarbitone but resolved when serum sodium became normal. While on admission he also received antibiotics and antipyretics. He gradually regained consciousness after three days of coma but was observed to have residual paresis in both lower limbs for which he was commenced on physiotherapy. He was discharged in good clinical condition after eight days and is being followed up in the consultant paediatric clinic.

3. DISCUSSION

Hypernatraemia is a common electrolyte abnormality defined as a rise in serum sodium to a value greater than 145 mmol/l [6]. An increase in serum sodium concentration creates an osmotic gradient between the extracellular and intracellular fluid in brain cells causing movement of water into the extracellular space to maintain the osmotic equilibrium at the expense of a decrease in the cell volume [7,8]. Thus, the predominantly neurological symptoms of hypernatraemia are attributed to brain cell shrinkage and related to both the severity and rapidity of the rise in the serum sodium level [7,8].

Typical symptoms of severe hypernatremia are seizures, somnolence and coma, as was seen in the index patient. In acute hypernatraemia, the combination of hyperosmolality and shrinkage in brain volume results in encephalopathy due to altered synaptic structure and function of the CNS cells [9,10]. There are three basic mechanisms by which hypernatremia occurs-excessive sodium intake, water deficit and sodium and water deficit. The first two mechanisms were likely causes in the index patient. Some common examples of these include ingestion of improperly mixed artificial infant milk, inadequate fluid intake and gastroenteritis respectively [11]. Most children with gastroenteritis do not have hypernatremia because they drink enough hypotonic fluid to compensate for stool water losses. However, replacing fluid deficit with concentrated ORS would lead to hypernatraemia [12].

The use of ORS as a treatment for mild to moderate dehydration both at home and within the hospital dates back to the 1970s [13]. The nationwide introduction of ORS to Nigeria has led to the improvement in diarrhoea case

management and a fall in infant and child mortality [13]. With the wider use of ORS prepared from packets, the incidence of hypernatraemia has increased despite intense media campaigns and community health education programmes [9]. A packet of ORS should be mixed with one liter of clean water and used within 24 hours. This knowledge is lacking even amongst educated mothers like the mother in the index case [14].

Management of a case of symptomatic hypernatremia requires careful fluid management and serum electrolyte monitoring to achieve good results. Serum sodium should be checked at least three to four times a day and appropriate intravenous fluids judiciously administered [11]. Intravenous fluid correction of hypernatraemia should be done cautiously because of the resultant neurologic effects. Intravenous fluids that can be used should contain sodium. E.g. Half normal saline, 4.3% dextrose in 1/5th saline, normal saline. The goal is to decrease the serum sodium by <12 mEq/L every 24 hr, a rate of 0.5 mEq/L/hr [11]. The too rapid correction could lead to cerebral oedema. The most critical aspect in the management of moderate or severe hypernatraemia is frequent monitoring of serum sodium levels, to enable appropriate titration of fluids to minimize too rapid or too slow correction. Identifying and treating the cause of the hypernatraemia is also necessary where a cause is found.

The challenges encountered in the management of this patient are two-pronged: to the paediatrician and the patient/ caregiver. To the former, the need to always reconstitute half normal saline and unavailability of infusion pumps which would enable precise administration of intravenous fluids is a challenge in our setting and would, therefore, require undue use of manpower to monitor the administration of fluids hour by hour where this could have been automated. The fact that seizures responded poorly to anticonvulsants was also a challenge and could indicate too rapid fluid administrations but similar reports have been documented in other studies [15]. Our findings however contrasted with other studies that suggested seizures responded to anticonvulsants [16]. Inability to perform a brain MRI/CT Scan or urinary Na level also posed diagnostic difficulties because while the former was due to financial constraints, the later was not a routine laboratory test done in tertiary facilities in our setting. To the patient/ caregiver, the need for an urgent brain

MRI/CT scan to assess for a likely intracerebral bleed, admission into an intensive care unit and frequent monitoring of serum sodium which ideally should be done 4-6 hourly were not possible due to financial constraints as the child's parents paid out-of-pocket. Furthermore, with the onset of paresis after regaining consciousness, review by physiotherapist and physiotherapy sessions prolonged hospital stay and eventually increased the financial burden.

This case report, therefore, highlights the need for increased awareness among mothers on the proper reconstitution of ORS by both healthcare providers and the mass media; and the need for a high index of suspicion and good clinical history taking in the assessment, diagnosis and management of children with gastrointestinal diseases among healthcare providers. Although CNS complications of hypernatremia in children are associated with high morbidity and mortality, careful management with serial serum sodium monitoring is the key.

4. CONCLUSION

Hypernatremia is a rare complication when poorly constituted Oral Rehydration Solution (ORS) is used. It can be life-threatening and can result in neurological complications. This is a case of symptomatic hypernatremia in a 2-year-old boy following ingestion of large volumes of poorly constituted Oral Rehydration Solution.

CONSENT

Authors declare that consent was obtained from the child's parents for publication of this case report about their ward's clinical presentation and response to treatment.

ETHICAL APPROVAL

As per international standard, written and informed ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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