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CASE REPORT

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Severe Hyponatraemia and *Klebsiella pneumoniae* Meningitis in a Severely Malnourished Infant: A Case Report

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Abstract

Severe hyponatremia with seizures in infancy is an uncommon but life-threatening condition, especially when complicating severe acute malnutrition (SAM) and central nervous system infection. *Klebsiella pneumoniae* meningitis, rare beyond the neonatal period, is associated with high morbidity and mortality. This report highlights multifactorial causes of infantile seizures and the need for thorough evaluation and targeted management. A 4-month-old male infant with background perinatal hypoxic–ischemic encephalopathy presented with recurrent seizures, fever, and poor feeding. He had global hypertonia and non-oedematous SAM. Laboratory tests showed profound hyponatremia (serum sodium 110 mmol/L), hypocalcaemia, anaemia, and marked thrombocytosis. Cerebrospinal fluid analysis showed hypoglycorrhachia and growth of *K. pneumoniae*. He received culture-guided antibiotics for 21 days, nutritional rehabilitation, and careful sodium supplementation via feeds, thereby raising serum sodium to 131 mmol/L over 4 days, with seizure control and weight gain. Early electrolyte monitoring, correction of dyselectrolytaemia, and rational antibiotic therapy are vital therapeutic steps when a nervous system infection coexists with severe acute malnutrition in resource-limited settings.

Key words: Cerebral oedema, Childhood meningitis, Dyselectrolytaemia, Seizures, Severe Acute Malnutrition.

Introduction

Seizures in infancy are a common but clinically emergent presentation with diverse causes, including structural brain injury, central nervous system (CNS) infections, and metabolic disturbances. Among the latter, hyponatraemia is particularly important because severe reductions in serum sodium levels are strongly associated with prolonged and recurrent seizures and increased morbidity. [1] Severe hyponatraemia, defined as serum sodium <120 mmol/L, may precipitate life-threatening cerebral oedema and seizures. Its management requires prompt identification and careful correction to avoid osmotic demyelination syndrome (ODS), which emanates from rapid correction of hyponatraemia. Paediatric guidance emphasises controlled, cautious correction with 3% Normal saline at 6-8 mmol/L rise in 24 hours, with frequent monitoring and more conservative targets in high-risk patients. [1]

Bacterial meningitis remains a significant global contributor to infant morbidity and mortality

worldwide, though more pronounced in the low- and middle-income countries (LMICs). [2] While *Klebsiella pneumoniae* is a well-recognised neonatal pathogen, it is a rare aetiology of meningitis beyond the neonatal period. *Klebsiella pneumoniae* meningitis is often severe, may be complicated by multidrug resistance, and carries a high risk of poor neurologic outcome. [3] Children with severe acute malnutrition (SAM) have compromised immune functions and electrolyte imbalances, making them highly susceptible to both infections and metabolic complications, with the likelihood of unfavourable outcomes.

Severe electrolyte derangement, such as hyponatremia, frequently coexists with CNS infections and may exacerbate seizure burden.[4] Malnutrition also predisposes children to metabolic and micronutrient abnormalities that can lower the threshold for seizures and complicate clinical recovery. [5] There are varied aetiologies of hyponatremia in children, such as gastrointestinal losses, excessive free water intake or retention, inappropriate use of hypotonic fluids or diuretics, sepsis, and neurological disorders. Some of the important and preventable risk factors for infantile hyponatremia in LMICs include sociocultural beliefs, poverty, low maternal education, inadequate breastfeeding support, [6, 7] and offering plain water by caregivers, which could result in free-water intoxication. Poor feed constitution remains a significant contributor to severe hyponatremia, particularly in poor-income settings. [8] In malnourished children, rehydration with ReSoMal (Rehydration Solution for Malnutrition) is generally recommended; however, its low sodium content may worsen symptomatic hyponatremia in children with SAM. [8]

We report this case to illustrate the intersecting risks of meningitis, severe acute malnutrition, and electrolyte disturbances, and to highlight the need for early electrolyte assessment, culture-guided antimicrobial therapy, and pragmatic yet safe sodium correction strategies in such contexts.

Case Presentation

A four-month-old male infant was admitted at the Federal Medical Centre, Abeokuta, Ogun State in July 2025, with a 1-week history of high-grade, intermittent fever and poor suck. Four days into the illness, he developed recurrent generalised tonic-clonic seizures that occurred multiple times per day, each episode lasting about 20-30minutes but self-aborting. There was no history of cessation of breath, bluish discolouration of lips, vomiting, diarrhoea, cough, rash, or abdominal distension. He was born at term, via spontaneous vaginal delivery at a primary health care centre. The neonatal period was complicated by perinatal asphyxia, stage II hypoxic-ischaemic encephalopathy and germinal matrix haemorrhage requiring special care at our facility. Developmental milestones were delayed with persistent head lag and fisting, and he had yet to achieve a social smile. Feeding practices were suboptimal, as the mother reported frequent use of diluted formula, in addition to breastfeeding. There was no family history of seizures, epilepsy, or features suggestive of metabolic disorders.

On examination, the infant appeared wasted with sparse hair, febrile (temperature of 38.4 °C), and moderate pallor. He was not cyanosed, and there was no pedal oedema. He had poor nutritional status, with anthropometric measurements consistent with non-oedematous SAM (weight 3.0 kg, length 50cm, weight-for-length z score < -3, OFC 38cm). The anterior fontanelle was tense, and neurological examination revealed impaired consciousness with a Blantyre coma score of 3/5 and global hypertonia. No cranial nerve deficits were elicited, and other systemic examinations were unremarkable.

Laboratory investigations showed severe hyponatremia (serum sodium 110 mmol/L), normal potassium (4.3mmol/L), and hypocalcaemia (8.7 mg/dL). The full blood count picture revealed moderate anaemia (packed cell volume 23%), extreme thrombocytosis (platelet count 1,043 × 10^9/L), leucocytosis (WBC 19.6 × 10^9/L), neutrophil predominance (55%), and elevated C-reactive protein (100 mg/L). Cerebrospinal fluid (CSF) analysis showed elevated protein (63 mg/dL), hypoglycorrhachia (CSF-to-blood glucose ratio of 0.25; CSF glucose 20 mg/dL, concomitant Random Blood Glucose 80mg/dL). CSF microscopy revealed 10–12 pus cells/hpf, 2–4 red blood cells/hpf, and pleocytosis with a predominance of neutrophils; the CSF culture yielded *Klebsiella pneumoniae* sensitive to ceftriaxone, cefotaxime, gentamicin, imipenem, and levofloxacin.

The infant received empirical treatment of intravenous ceftriaxone and gentamicin, and later levofloxacin was added based on culture sensitivity and persistent fever despite the initial antimicrobials. Seizure control was achieved after escalation from phenobarbitone with eventual stabilisation on levetiracetam and correction of hyponatraemia. Measured-dose sodium supplementation was carefully incorporated into feeds, raising serum sodium to 131 mmol/L over 4 days. Calcium supplementation was also provided. He had nutritional rehabilitation with F-75, which was later transitioned to F-100 feeds. Steady weight gain and improved feeding tolerance were observed during admission. The infant completed the 21-day course of intravenous antibiotics and remained seizure-free on oral levetiracetam. He showed clinical improvement in nutritional status, with a weight of 3.8kg at discharge. He was discharged on continued nutritional rehabilitation and follow-up at the neurodevelopmental clinic.

Parental consent was obtained for the use of the child's data in this research.

Discussion

Hyponatremia is one of the most important electrolyte abnormalities encountered in paediatric practice, and its prevalence varies depending on population, comorbidities, and healthcare setting. Severe hyponatraemia (<120 mmol/L) is less common but associated with high morbidity due to its neurological manifestations, such as the risk of cerebral oedema and seizures. A retrospective cohort study in hospitalised children found hyponatremia in up to 30% of admissions, with a strong association between severe forms and CNS infections. [9]

In infants, dilutional hyponatremia is particularly linked to inappropriate feeding practices such as feeding infants with large volumes of solute-poor liquids or giving excess free-water intake as a result of the misconception that breast milk alone is insufficient. [6, 10] These may overwhelm the immature kidneys, with the inability to excrete free water, further diluting the blood sodium level.[11] Although the index case was still breastfed, artificial milk feeds were being overtly diluted (1 scoop to 90mls of water) due to the misconception that infant formula causes constipation. Indeed, the coexistence of SAM and hyponatraemia further complicated the clinical picture in the patient. Severe acute malnutrition predisposes to electrolyte disturbances due to renal tubular dysfunction, altered sodium–potassium balance, and low body stores of essential micronutrients. [10] Malnutrition increases the risk of infection, which can also drive inappropriate ADH secretion, hyponatremia, and electrolyte disturbances. Seizures may be provoked independently by electrolyte disturbances that may be refractory to antiseizure medications alone. [12]

In the instance of coexistence of malnutrition, severe hyponatremia and bacterial meningitis,

each condition may amplify the others. Bhutta *et al.,* [12] described overlapping features of electrolyte derangements and infectious diseases in malnourished children, making timely recognition and correction critical for survival. These overlapping pathophysiological pathways could create diagnostic and therapeutic challenges. These challenges are particularly magnified in resource-limited settings where rapid and frequent biochemical monitoring, advanced neuroimaging, and a broad range of antimicrobials may be unavailable, inaccessible and unaffordable. In the index case, SAM not only increased susceptibility to infection but could also have contributed to impaired sodium handling and vulnerability to seizures.

Klebsiella pneumoniae is a rare but important cause of bacterial meningitis beyond the neonatal period. Its occurrence has been increasingly reported in LMICs, often associated with high case fatality and neurologic sequelae. [13,14] A report by Zhang *et al.,* [15] documented meningitis due to hypermucoviscous *K. pneumoniae* in an infant, emphasising the pathogenic potential of this organism even outside the neonatal period. Microbiological confirmation and sensitivity-guided therapy are highly imperative, particularly in the era of rising multidrug resistance. In the index case, *K. pneumoniae* was isolated from the cerebrospinal fluid.

Fluid and electrolyte management in malnourished children requires caution; ReSoMal is generally recommended by the World Health Organisation based on the theory that malnourished children have high risks of sodium overload and hypokalaemia. [8] Rehydration Solution for the Malnourished is the fluid of choice in SAM, but it is contraindicated in symptomatic hyponatremia as it may worsen the existing hyponatremia after rehydration. [16] The management of severe hyponatraemia requires active correction with sodium chloride (3% saline) infusion. [1] Careful sodium supplementation within feeds, as used in this patient, provided a practical alternative in a resource-limited setting where hypertonic saline infusion and close laboratory monitoring may not be feasible.

The multiplicity of seizure triggers and aetiology in this infant vis-à-vis background perinatal hypoxic–ischemic encephalopathy, severe hyponatraemia, hypocalcaemia, and meningitis illustrate the diagnostic complexity often encountered in paediatric neurology. Importantly, seizures persisted despite escalating anticonvulsant therapy until metabolic derangements were corrected, underscoring that antiseizure therapy alone is insufficient without addressing underlying cause(s).

Conclusion

This case demonstrates the rare convergence of SAM, severe hyponatraemia, and *K. pneumoniae* meningitis as multifactorial drivers of infantile seizures in a child with background hypoxic encephalopathy. The diagnosis may be easily overlooked, especially when seizures are attributed to underlying neurological conditions, yet timely recognition and correction are lifesaving. Early electrolyte assessment, culture-guided antimicrobial therapy, and individualised sodium correction strategies are critical to survival and recovery. Timely recognition and pragmatic supportive care can significantly improve outcomes in resource-limited countries.

Learning Points

Infants with seizures should undergo early electrolyte evaluation, as derangements such as hyponatremia and hypocalcemia may coexist with central nervous system infection.

Severe acute malnutrition predisposes to both serious bacterial infections and metabolic derangements, necessitating careful nutritional and fluid management. Individualised sodium supplementation is safer in the context of

symptomatic hyponatraemia in children with SAM. Solitary use of an anticonvulsant may be ineffective for seizure control in the presence of metabolic derangements.

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