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Case Report

Suspected Salt Poisoning in a 2-Month-Old Infant in a UK Paediatric Unit: A Case Report

Rehnuma Tarannum* and Iqra Saher

Clinical Fellow, Paediatric Department, East Lancashire Hospitals NHS Trust, United Kingdom

***Corresponding Author:** Rehnuma Tarannum, Clinical Fellow, Paediatric Department, East Lancashire Hospitals NHS Trust, United Kingdom.

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Abstract

This case report presents a rare and life-threatening case of severe hypernatremia in a previously well 2-month-old infant. The child was admitted with lethargy and poor feeding and was found to have a serum sodium of 168 mmol/L, which rose rapidly to 198 mmol/L within 48 hours despite controlled fluid management verified by strict monitoring. Urinary sodium levels exceeded 200 mmol/L, and osmolality results indicated preserved concentrating ability, making dehydration and diabetes insipidus unlikely. The rapid and disproportionate biochemical abnormalities, along with clinical presentation and minimal weight loss, raised strong suspicion for intentional salt poisoning. Prompt multidisciplinary action, including renal, metabolic, and safeguarding teams, led to escalation of care, transfer to PICU, and social services involvement. Neuroimaging revealed subdural and subarachnoid haemorrhages. This case highlights the importance of considering non-accidental causes in infants with unexplained hypernatremia and illustrates the critical role of early safeguarding and coordinated multidisciplinary management. The infant was discharged stable under foster care with ongoing neurodevelopmental follow-up.

Keywords: *Hypernatremia; 2-Month-Old Infant; UK Paediatric Unit*

Background

Hypernatremia in infants is uncommon and often results from dehydration or inappropriate feeding practices. However, when serum sodium levels rise acutely and to dangerously high levels, alternative causes-including accidental or intentional salt poisoning-must be considered. This case illustrates the clinical presentation, management, and safeguarding concerns arising from a suspected case of salt poisoning [1,2].

Pathophysiology

Salt poisoning leads to hypernatremia by creating an osmotic gradient that draws water out of cells. This cellular dehydration particularly affects neurons, potentially causing vascular injury, cerebral shrinkage, and intracranial hemorrhage. Rapid correction of hypernatremia can result in cerebral edema due to fluid shifts back into the intracellular space. Hence, sodium levels must be corrected cautiously to avoid iatrogenic complications.

Case Presentation

XXX, a male infant born on 12 April 2024, was admitted to the paediatric unit on 25 June 2024 at the age of 10 weeks with lethargy and poor feeding. The day prior, on 24 June, he had presented to COAU with abnormal movements suspicious for seizure and was discharged home after observation. Upon reattendance, initial investigations revealed severe hypernatremia with serum sodium of 168 mmol/L on 26 June, which remained high on subsequent tests and reached 198 mmol/L within 2 days. Urine sodium levels were also significantly elevated at over 200 mmol/L.

On admission (25 June), PEWS was zero. Examination findings:

- Vital signs: HR 138 bpm, RR 44, Temp 36.8°C, SpO_2 98% on room air.
- Warm and well-perfused.
- Heart sounds normal.
- Chest clear, good air entry bilaterally.
- Abdomen soft, non-tender, bowel sounds present.
- Femoral pulses palpable.
- Anterior fontanelle soft.
- No rash.
- Slightly pale appearance.
- Neurological reflexes: normal Moro, grasp, stepping.
- Responsive and smiling when held; slightly quiet when lying down (noted as decreased activity but awake and alert on review).
- Pupils equal and reactive to light (PEARL).

Weight was stable with no significant loss noted, supporting the absence of dehydration.

Initial management

The infant was commenced on intravenous fluids-initially 0.9% NaCl with 5% dextrose as maintenance. Following early morning review, fluid management was changed to 0.45% NaCl with 5% dextrose maintenance and 5% deficit correction considering dehydration. Strict fluid balance monitoring was initiated and verified.

Discussion with RMCH metabolic and renal team (26/06/24)

Initial consultation suggested hypernatremia might be due to dehydration. Consultant Nephrologist recommended:

- Weight monitoring twice daily.
- Strict input/output documentation.
- 4-hourly U&Es and blood gases.
- Paired plasma and urinary osmolality.
- Urinary sodium and creatinine.

Renal team monitored results closely and advised maintaining fluid strategy unless sodium trajectory or renal function changed.

Sodium trend in Blackburn hospital

Date/Time	Serum Sodium (mmol/L)
25/06/24 20:11	162
26/06/24 01:02	176
26/06/24 09:30	175
26/06/24 15:53	171
26/06/24 19:46	170
26/06/24 22:33	193
27/06/24 02:29	>200
27/06/24 06:33	198

Table

Despite appropriate fluid management, sodium levels rose above 200 mmol/L within six hours of admission, coinciding with clinical deterioration including pallor, hypotonia, and desaturation.

Safeguarding concerns

Given the rapid sodium increase despite controlled IV fluids and worsening clinical status, the renal consultant raised concerns for intentional salt poisoning. NWTS (North West and North Wales Transport Service) was contacted, and a safeguarding referral was made to Blackburn with Darwen Children's Social Care. NWTS supported informing social services.

Nursing staff discovered white crystalline particles on surfaces within the child's cubicle, which were confirmed to taste salty. Relevant feeding equipment including the NG tube and formula tins were secured and preserved for forensic investigation. These findings were noted on 26 June evening.

Multidisciplinary team (MDT) involvement

Safeguarding procedures were initiated promptly, involving social services, the named safeguarding consultant, and paediatric liaison. A strategy meeting and MASH (Multi-Agency Safeguarding Hub) discussion were held, leading to escalation of legal processes. The police were informed to support forensic evidence handling and conduct risk assessment.

Course in hospital

XXX was transferred to PICU at RMCH on 27 June due to respiratory decompensation and concern for neurological sequelae. He required high-flow nasal cannula oxygen and developed seizures lasting several minutes, treated with buccal midazolam and commenced on Levetiracetam (Keppra). EEG was performed showing epileptiform activity.

MRI and CT head imaging revealed subdural and subarachnoid haemorrhages with demyelination changes. Ophthalmologic exam was unremarkable. Blood tests including clotting studies, factor levels, and metabolic panels were performed. He received two blood transfusions during admission.

A skeletal survey was conducted on 08 July with a repeat planned for 22 July.

Return to local hospital and social care involvement

XXX was transferred back to the local hospital on 06 July for discharge planning. Clinically stable, feeding orally on Neocate with no further seizures. On 12 July, the court granted an Interim Care Order and foster placement was arranged. Supervised parental visits were permitted with restrictions on direct care.

Follow-up was arranged including outpatient skeletal survey, neurology review in 2 - 3 months, and community paediatrics.

Background/previous admissions

On 01 June 2024, at 7 weeks, XXX was admitted with breathing difficulty, vomiting blood, cyanosis, and unresponsiveness. CPR was administered by his grandfather; ambulance arrival required 10L oxygen. Multiple COAU attendances for vomiting with blood were recorded. Chest X-ray showed bilateral pulmonary shadowing.

Swallow assessment by SALT found unsafe swallowing with risk of aspiration. He was kept NBM with NG feeds. MRI head and ophthalmology exams were normal. ENT scope (DLTB) was normal. Dietetics optimised feeding; SALT permitted limited oral feeding followed by NG top-ups. Family was trained in NG feeding. Discharged with apnoea monitor.

No safeguarding concerns were formally raised during previous admissions.

Investigations

- Initial serum sodium (26 June 2024): 168 mmol/L.
- Sodium escalated to 198 mmol/L within 48 hours despite fluid therapy.
- Serum osmolality: 398 mOsm/kg; urine osmolality: 700 mOsm/kg (preserved concentrating ability).
- Urine sodium: >200 mmol/L.
- Blood gases and U&Es monitored 4-hourly.
- MRI and CT head: Subdural and subarachnoid haemorrhages, demyelination changes.
- Ophthalmology review: Unremarkable.
- Skeletal survey performed.
- EEG during PICU stay: Epileptiform activity detected.
- Metabolic screen and CSF studies: no abnormalities detected.

Differential diagnosis

- Hypernatremic dehydration: Ruled out due to absence of significant weight loss, normal hydration status, and absence of haemoconcentration.
- Diabetes insipidus: Unlikely given high urine osmolality and sodium [3].
- Salt poisoning (intentional): Considered likely due to disproportionate sodium levels, clinical findings, and safeguarding concerns [2].
- Other endocrine/metabolic causes: No clinical or laboratory evidence supporting alternative diagnoses.

Discussion and Conclusion

This case illustrates the need for a high index of suspicion when laboratory findings are inconsistent with clinical presentation or response to treatment. Salt poisoning, although rare, can be life-threatening and must be considered when sodium levels rise rapidly despite controlled fluid administration [2].

Multidisciplinary involvement including paediatrics, renal, safeguarding, nursing, and PICU teams enabled early identification and escalation. Collection and preservation of forensic evidence (e.g. white granules, feeding equipment) were vital. Long-term follow-up will focus on neurodevelopmental outcomes and safeguarding.

Salt poisoning is a rare but critical cause of non-accidental injury (NAI) that demands prompt recognition and intervention.

Prognosis and follow-up

Neuroimaging findings suggest risk for long-term neurological sequelae, including developmental delay, epilepsy, or motor impairment. Regular neurodevelopmental surveillance is required. The infant remains under social services care, with multidisciplinary outpatient follow-up including neurology, dietetics, and community paediatrics.

Learning points

- Always consider non-accidental injury in cases of severe, unexplained hypernatremia-especially when clinical findings do not align with dehydration [5].
- Rapidly rising sodium despite appropriate fluid management should prompt suspicion for exogenous sodium administration.
- Urine osmolality and sodium are key investigations in distinguishing causes of hypernatremia [3].
- Early safeguarding involvement is critical in protecting vulnerable infants.
- Preservation and documentation of environmental and forensic evidence (e.g. white particles, feeding equipment) are essential in suspected salt poisoning cases.
- Multidisciplinary coordination (paediatrics, renal, safeguarding, PICU) is essential in managing complex, high-risk cases.

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