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Research Article

Hypernatremic, Hyperkalemic Dehydration in an 8-Month-Old with Acute Gastroenteritis Managed With Controlled Hypotonic Rehydration

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ABSTRACT
Background: Hypernatremic dehydration is a dangerous manifestation during infancy since hyperosmolality can trigger neurologic impairment and since excessive rapidity of correction may trigger cerebral edema. Even though acute gastroenteritis is typically associated with the emergence of iso- or hyponatremic dehydration, hypernatremia can also occur when the volume of free-water loss and reduced intake surpasses that of sodium loss, especially in young infants. Prerenal physiology (1) and gastrointestinal bicarbonate depletion (2), respectively, may be represented by concomitant hyperkalemia and hyperchloremia.
Case presentation: A male patient aged 8 months complained about several periods of vomiting and loose stools and a report of being drowsy with clinical signs including moderate dehydration. No previous administration of oral rehydration solution or intravenous fluids history. The first complete blood count revealed hemoconcentration (hematocrit 38.3%) and reactive thrombocytosis (platelets 470 ×10 3 /mm 3). Serum electrolytes demonstrated hypernatremia (Na 153.15 mmol/L), hyperchloremia (Cl 134.11 mmol/L), slight hyperkalemia (K 5.07mmol/L), and an increase in the ionized calcium (iCa 1.51 mmol/L). With close clinical and biochemical observation, controlled intravenous rehydration with 0.45% saline was initiated. After repeating the test about 6.7 hours later, the electrolytes focused on enhancement (Na 151.24 mmol/L; Cl 123.63 mmol/L; K 4.41 mmol/L; iCa 1.45 mmol/L), which matches approximately 0.29 mmol/L/h sodium correction rate, which is well within the safety extremes.
Conclusion: Physiologically consistent patterns of hypernatremia (net water loss and insufficient intake), hyperchloremia (diarrheal loss of bicarbonate and chloride retention under hypovolemia), hyperkalemia (temporarydownward changesin renal potassium excretion due to prerenal hypoperfusion +acidosis and mild hypercalcemia (hemoconcentration and impaired renal calcium clearance) are demonstrated in this case. Slow correction using hypotonic saline was linked to biochemical improvement without any known complications.

INTRODUCTION

Acute gastroenteritis is a major leading cause of pediatric morbidity in most countries and can often lead to dehydration that necessitates oral or intravenous rehydration therapy [1]. The majority of children in whom diarrhea occurs experience isotonic fluid losses but babies are comparatively susceptible to dysnatremias due to greater insensible losses, decreased concentrating capability, in addition to the requirement to have fluid substituted by the caregivers [2,3]. Hypernatremic dehydration is clinically significant: hyperosmolality stimulates the intracellular water excretion and may cause such signs as irritability, lethargy, or depressed sensorium, whereas iatrogenic overcorrective nonselectively may lead to cerebral edema and seizures [2,4]. Even modern series emphasize clinically significant hypernatremia among hospital-acquired gastroenteritis in infants, indicating implications on intensive monitoring and conciliatory correction strategies [5,6].

The pathogenesis of hypernatremia in diarrheal disease usually indicates an imbalance of loss of free-water compared to sodium loss, which is doubled by not taking a sufficient amount via vomiting, lack of feeding, or improper replacement fluids [2,6]. Hyperchloremia is normally associated with gastrointestinal reduction of bicarbonates (hyperchloremic metabolic acidosis) and further complicated by renal chloride conservation during hypovolemia [8]. Transient decreases in the delivery of distal sodium and glomerular filtration (prerenal physiology), the occurrence of hyperkalemia in dehydrated infants may happen, or the extracellular potassium shift is mediated by acidosis, or sampling artifact like hemolysis may cause true hyperkalemia versus pseudohyperkalemia; hence necessitating the distinction between available hyperkalemia and pseudohyperkalemia [9,10].
An 8-month-old infant with vomiting and diarrhea contacted with drowsiness and

moderate dehydration after which the baby showed simultaneous hypernatremia, hyperchloremia, mild hyperkalemia, and increased ionized calcium, which improved on controlled hypertonic intravenous rehydration are described in this report. The case points to a single physiologic account of the seen electrolyte pattern, and restates the significance of slow sodium correction with sequential clinical and biochemical reexamination [2,6].

Patient Information

A 8 months old male patient was brought with the history of having several vomiting episodes and loose stool. No history of any previous administration of oral rehydration solution and intravenous fluids. The provided draft was not able to provide past medical history, birth history, immunization status, feeding practices (exclusive breastfeeding vs formula), and comorbidities. The draft did not mention any history of chronic medications or known toxin/salt ingestion. The identity of the child and the facility identifiers are not given due to confidentiality.

Figure 1. Clinical Appearance of the 8-Month-Old Infant at Presentation with Drowsiness in the Setting of Acute Gastroenteritis and Moderate Dehydration.

Clinical Findings

Upon examination, the infant was found to be sleepy and the clinical presentation was that of moderate dehydration. The draft did not record specific vital signs (heart rate, respiratory rate, blood pressure, temperature, oxygen saturation), weight, urine output, capillary refill time, mucous membrane assessment, and fontanelle status and thus it cannot be reported. No seizures were described. The initial clinical impression was acute gastroenteritis and dehydration and altered sensorium presumably due to hyperosmolality.

Timeline (chronological narrative)

Day 0–1 (pre-presentation)

- Multiple episodes of vomiting and loose stools; progressive reduced intake presumed (not quantified).

Day 1 (Presentation; 24 Jan 2026):

- Drowsiness and moderate dehydration noted clinically.

- CBC obtained showing hemoconcentration and reactive thrombocytosis.
- Serum electrolytes demonstrated hypernatremia, hyperchloremia, mild hyperkalemia, and elevated ionized calcium.
- Intravenous rehydration initiated with 0.45% saline (route: IV).

~6–7 Hours after Initiation of IV Fluids (Same Day):

- Repeat electrolytes showed improvement in sodium, chloride, potassium, and ionized calcium.

Diagnostic Assessment

Differential Diagnosis

In an infant with vomiting, diarrhea, dehydration, and hypernatremia, the differential includes:

1. Acute gastroenteritis with free-water–predominant losses and reduced intake (most likely).
2. Improperly prepared high-solute feeds (overconcentrated formula) or sodium-rich home fluids (not supported by history, but not fully assessable without feeding details).
3. Salt poisoning (no supporting history; typically involves higher sodium levels and concerning social/ingestion history) .
4. Diabetes insipidus (would be suggested by polyuria and persistent hypernatremia despite rehydration; not suggested in the available draft) .
5. Renal impairment (prerenal vs intrinsic) contributing to hyperkalemia and hypernatremia.
6. Adrenal insufficiency/congenital adrenal hyperplasia (typically hyponatremia with hyperkalemia; discordant with this case's hypernatremia).
7. Pseudohyperkalemia due to hemolysis or prolonged tourniquet time (must be considered whenever potassium is mildly elevated).

Tests and Results

Complete Blood Count (CBC):

- WBC 13.92 $\times 10^3/mm^3$ (mild leukocytosis)
- Hemoglobin 12.7 g/dL; Hematocrit 38.3% (hemoconcentration consistent with dehydration)
- Platelets 470 $\times 10^3/mm^3$ (reactive thrombocytosis)
- Differential: neutrophilia (60.8%; ANC 8.47 $\times 10^3/mm^3$)

Serum Electrolytes (Serial):

- **07:38:** Na 153.15 mmol/L; K 5.07 mmol/L; Cl 134.11 mmol/L; iCa 1.51 mmol/L
- **14:17:** Na 151.24 mmol/L; K 4.41 mmol/L; Cl 123.63 mmol/L; iCa 1.45 mmol/L

Renal function (urea/creatinine), serum bicarbonate/venous blood gas, glucose, and urinalysis/urine electrolytes were not provided but would ordinarily be central to evaluating prerenal injury, acid–base status, and renal water handling

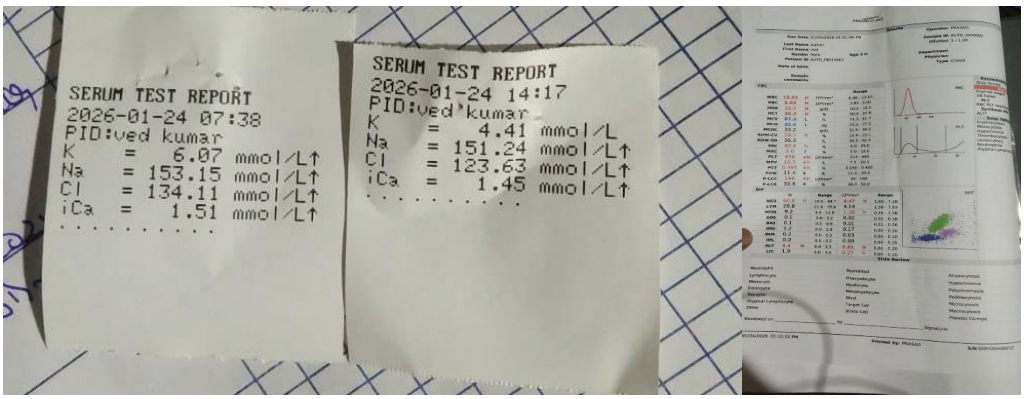

Figure 2. Laboratory Printouts Demonstrating Hypernatremia, Hyperchloremia, Mild Hyperkalemia, And Elevated Ionized Calcium With Partial Biochemical Improvement Following Controlled Intravenous Rehydration.

Diagnostic Reasoning

A clinical syndrome (gastrointestinal losses + decreased intake + moderate dehydration variable with sustained nightmares) with hemoconcentration has been strongly suggestive of hypernatremic dehydration by net free-water loss [2,4]. The concomitant hyperchloremiais physiologically compatible with diarrheal loss of bicarbonates (hyperchloremic metabolic acidosis) and kidney retention of chloride in the face of volume loss [8]. Mild hyperkalemia resolved astronomically with rehydration, favouring a momentary decrease of renal excretion of potassium by prerenal hypoperfusion and/or acidosis as

opposed to an underlying disorder of mineralocorticoids. Minor rises in ionized calcium might be due to dehydration through hemoconcentration and decreased renal calcium clearance, and it decreased as intravascular volume normalised.

Therapeutic Intervention

The newborn was administered to 0.45% saline (half-normal saline) through intravenous administration followed by monitoring and clinical assessment. Route: IV. In the draft, period and total volume were not indicated. Nothing had been administered prior to presentation of ORS or IV fluids.

As the initial sodium was 153.15 mmol/L, the measured decrease of 153.15 mmol/L to 151.24 mmol/L over a period of approximately 6.7 h is equivalent to an approximation correction rate of 0.29 mmol/L/h, which is consistent with the conservative advice not to use rapid changes in the brain osmolality during hypertonic dehydration [2,6]. None of the potassium-lowering drugs were reported; the normalization of potassium by volume replacement.

Inpatient IV therapy does not have adherence, but the reported serial improvement shows that the intervention was implemented as planned.

Follow-up and Outcomes

Objective outcomes

Table 1. Serial laboratory trends during rehydration

Parameter (units)	Baseline	Follow-up	Δ
Sodium (mmol/L)	153.15	151.24	−1.91
Potassium (mmol/L)	5.07	4.41	−0.66
Chloride (mmol/L)	134.11	123.63	−10.48
Ionized calcium (mmol/L)	1.51	1.45	−0.06
Hematocrit (%)	38.3	Not available	—

Patient-Centered Outcomes

Clinically, the child's drowsiness and dehydration were managed with IV rehydration; implies improvement in biochemical abnormalities and clinical status without reporting complications.

DISCUSSION

The case shows a consistent frequency of electrolyte phenotype; an increase and diminution in the concentration of sodium, chloride, and BHC along with a mild infiltration of ionized calcium and BHC result in an infant with vomiting, diarrhea and moderate dehydration. Despite the fact that diarrheal dehydration is commonly isotonic, hypernatremia exerts clinical significance and poses risk of neurologic morbidity in infancy, especially when the dehydration is corrected overly fast [2, 4]. The epidemiologic and hospital-based information supports the ongoing problem of hypernatremic dehydration in infants with gastroenteritis and its relevance to clinically meaningful outcomes, which results in the necessity to employ intentional monitoring and correction strategies [3, 5, 6].

Hypernatremia is an expression of a relative decrease in water relative to sodium. Water loss comes through stool and emesis in gastroenteritis, but hypernatremia means that the losses of water as free water and/or inability to take water in were more than sodium losses [2]. The vulnerable population is infants as they are unable to independently take water and might also have less water intake with sickness. Hypernatremia may occur in the absence of prior administration of ORS or IV fluids (i.e., when the vomiting restricts oral intake and diarrhea persists)-especially when providing small volumes of higher-solute feeds or increased insensible losses (vomiting, tachypnea) though these factors were not provided in this case [2,6]. Clinically, the hyperosmolality may be expressed by lethargy or depressed consciousness, which is in line with the drowsiness of the infant upon presentation [2, 4].

The chloride of 134.11 mmol/L is significantly high. During diarrheal illness, loss of bicarbonate in stools results in a non-anion gap (hyperchloremic) metacidosis as bicarbonate is lost, chloride increases to maintain electroneutrality [8]. Simultaneously, hypovolemia triggers neurohormonal mechanisms which encourage sodium and chloride retention in the kidney and decreased glomerular filtration may restrict the excretion of chloride [8,10]. In spite of the fact that no serum bicarbonate was prescribed, hyperchloremic acidosis is still a sparse explanation of the chloride abnormality because of its clinical context. The significant

rehydration-associated loss of chloride indicates a reversible, volume-determined factor over a given fixed disorder of the renal tubules.

The potassium was slightly in elevation, which is 5.07 mmol/L and it came to its normalcy almost immediately following rehydration. The low renal perfusion in hypovolemia reduces the distal sodium delivery and glomerular filtration, worsening potassium excretion characteristics of the prerenalazeotemia physiology [10]. Hydrogen to potassium shifts could also have been a factor in an elevated measured potassium in the case of hyperchloremic metabolic acidosis [8]. Notably, mild hyperkalemia in infants may be artifactual; in phlebotomy, hemolysis releases intracellular potassium, which creates pseudohyperkalemia [9]. The fast decline of potassium in this situation without targeted potassium-lowering treatment is, respectively, compatible with (a) disappearance of transient prerenal impairment and acidosis after volume replacement, and (b) minor sampling artifact in the first place. In any case, the answer highlights the clinical idea that verifying hyperkalemia and measuring acid-base and renal functions are important before proceeding with more aggressive potassium-reduction measures in case the rise is mild, and the child is not in hemodynamically unstable condition [9,10].

Calcium ionization was slightly increased (1.51 mmol/L) and reduced on rehydration (1.45 mmol/L). Hemoconcentration can increase calcium during dehydration and reduce renal calcium excretion by causing a decrease in glomerular filtration rate and an increase in proximal tubular reabsorption rate [11]. A massive review underlines that dehydration is a clinical expression of clinically significant hypercalcemia as well as one of its major elements of management through hydration in the symptomatic cases [11]. In newborns, relentless hypercalcemia would necessitate diagnosis of an endocrine, genetic, granulomatrous or even vitamin-D-associated pathophysiology; nonetheless, the propensity towards normalization in response to volume supplement workers in this manifestation.

Another myth is that hypernatremic dehydration is predominantly iatrogenic (i.e. too much sodium due to wrongly-made ORS, or wrong fluid administration). Although there are observational studies indicate that hypernatremia also occurs in typical gastroenteritis cases, especially among infants, due to a combination of continued manifestations of similar losses through the pores, inadequate intake, and the failure of the renal conservation of water that is unable to make adequate compensation [5,6]. Hypernatremia, which is greater than 150 mmol/L, was not uncommon among infants admitted with acute gastroenteritis in the Durban cohort, which supports the idea that the condition is also common when sodium loading is not recorded [5]. Thus, net water loss, which has been augmented by decreased intake during vomiting is the most justifiable to explain this situation as opposed to exogenous administration of sodium.

Restoration of intravascular volume and a progressive reversal of hyperosmolality are foundations of management to decrease neurologic risk [2]. Saline (0.45 big percentage) made the child get better biochemically. Even though the given volumes have not been provided, the measured sodium correction rate (approximately, 0.29 mmol/L/h) during the interval under observation is low and is aligned with the common safety of correction in infant hypernatremia [2,6]. Serial monitoring is necessary since the optimal fluid tonicity is variable, and rhythmic gastrointestinal losses, urinary output, and renal functioning [2, 10].

Strengths and limitations

One of its strengths is that serial electrolyte tests indicate an improvement following controlled hypotonic rehydration, which is evidence of biologic plausibility and cause-effect relationship between dehydration and electrolyte abnormalities. The limitations are the lack of recorded weight, vital signs, urine output, bicarbonate/pH, and renal functions tests as well as history of feeding limit definitive ruling out of other etiologies like diabetes insipidus, ingestion of salt, or intrinsic renal pathology [2,10]. Urine studies, acid-base values, and calculated explicit corrections including total fluid volumes would enhance future similar reports.

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