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Case Report

Extreme hypernatraemia in a nonverbal adult with cerebral palsy: interpreting sodium during hyperglycemia

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ABSTRACT

Extreme hypernatremia may develop with few warning signs when a patient cannot communicate thirst or obtain water independently. We describe a 66-year-old nonverbal woman with ataxic cerebral palsy, chronic dysphagia, diabetes, and dependence on others for fluid provision who presented with lethargy, hypotension, a sodium concentration of 182 mmol/l, glucose of 581 mg/dl, and prerenal acute kidney injury. Hyperglycemia both worsened water loss through osmotic diuresis and masked the severity of hypernatremia; Katz-corrected sodium was 189.7 mmol/l and effective osmolality was 396 mOsm/kg. She received isotonic saline for initial intravascular volume restoration, followed by hypotonic fluids and insulin with frequent biochemical monitoring. Sodium normalized, kidney function recovered, and her usual interaction returned. This case connects interpretation of sodium during hyperglycemia with the need for structured, swallow-safe hydration surveillance in care-dependent adults.

Keywords: Hypernatremia, Cerebral palsy, Dysphagia, Hyperglycemia, Dehydration

INTRODUCTION

Hypernatraemia usually reflects a deficit of water relative to total body sodium and becomes more severe when thirst and access to water is impaired.[1] Patients with impaired communication, dysphagia, immobility or dependence on others for hydration are particularly vulnerable because water loss may progress before dehydration is recognised.[1,2] Extreme hypernatraemia is clinically important not only because of its association with neurologic dysfunction and mortality but also because its treatment requires simultaneous attention to circulatory volume, free-water deficit and probable duration of the disorder.[3] Concurrent hyperglycemia aggravates water depletion by renal free-water loss through osmotic diuresis while simultaneously lowering the measured sodium concentration through transcellular water movement, thereby obscuring the full severity of the underlying water deficit.[4,5] Previously published cases demonstrate that sodium concentrations at or above 180 mmol/l can be survivable with hemodynamic stabilisation and closely monitored fluid replacement.[6,7] We report extreme hypovolemic hypernatraemia with severe hyperglycaemia in a non-verbal, wheelchair dependent woman with cerebral palsy and chronic dysphagia. The case demonstrates how barriers to fluid intake and hyperglycemic osmotic diuresis can converge with few recognizable warning signs, while highlighting the need to interpret sodium in relation to glucose and tonicity, sequence fluid therapy according to changing physiologic goals and establish structured hydration surveillance after recovery.

CASE REPORT

A 66-year-old woman with ataxic cerebral palsy was transferred from a group home for lethargy, global weakness, and inability to transfer to the commode even with assistance. She was non-verbal, wheelchair dependent, required a pureed diet for chronic dysphagia, and depended on others to provide fluids. She also has diabetes mellitus, hypothyroidism, sinus-node dysfunction with permanent pacemaker placement, and frequent urinary tract infections. According to caregivers, she was normally awake and interactive, recognized caregivers, babbled and clapped when pleased. On arrival, she was lethargic and clinically dehydrated, with extremely dry mucous membranes, blood pressure 90/60 mm Hg, and a heart rate of 115 beats/min. Pulmonary or abdominal examinations were unremarkable.

Initial laboratory evaluation showed sodium 182 mmol/l, glucose 581 mg/dl, blood urea nitrogen 87 mg/dl, creatinine 1.84 mg/dl, hemoglobin 16.8 g/dl, hematocrit 57.2%, consistent with marked hemoconcentration and prerenal azotemia. Bicarbonate was 30 mmol/l, with an anion gap of 6 mmol/l, and beta-hydroxybutyrate was 0.95 mmol/l, arguing against diabetic ketoacidosis. Using correction factors of 1.6 and 2.4 mmol/l per 100 mg/dl of glucose above 100 mg/dl, the corrected sodium was 189.7 and 193.5 mmol/l, and the calculated effective osmolality was 396 mOsm/kg. A head computed tomography was done, which showed no acute lesion.

The findings supported hypovolemic hypernatremia from inadequate free-water intake, likely compounded by osmotic diuresis from hyperglycemia. She was admitted to the intensive care unit and received isotonic saline to restore perfusion, with close neurologic and electrolyte monitoring. When the measured sodium transiently increased, fluids were changed to 0.45% saline, then to 5% dextrose as free-water replacement became the priority. Insulin was given for glycemic control, and empiric antibiotics were started for a presumed urinary tract infection; but the blood cultures remained negative, and no infectious source was confirmed.

The serum sodium level briefly increased to 187 mmol/l during the early treatment, followed by a decrease to 169 mmol/l over approximately 39 hours, and reached 142 mmol/l at discharge. Serial laboratory values of sodium over the course of hospitalization are shown in figure 1. Creatinine improved from 1.84 mg/dl to 0.93 mg/dl within 72 hours and later to 0.69 mg/dl. Her mental status improved within 24 hours. Repeat head computed tomography showed no acute intracranial abnormality.

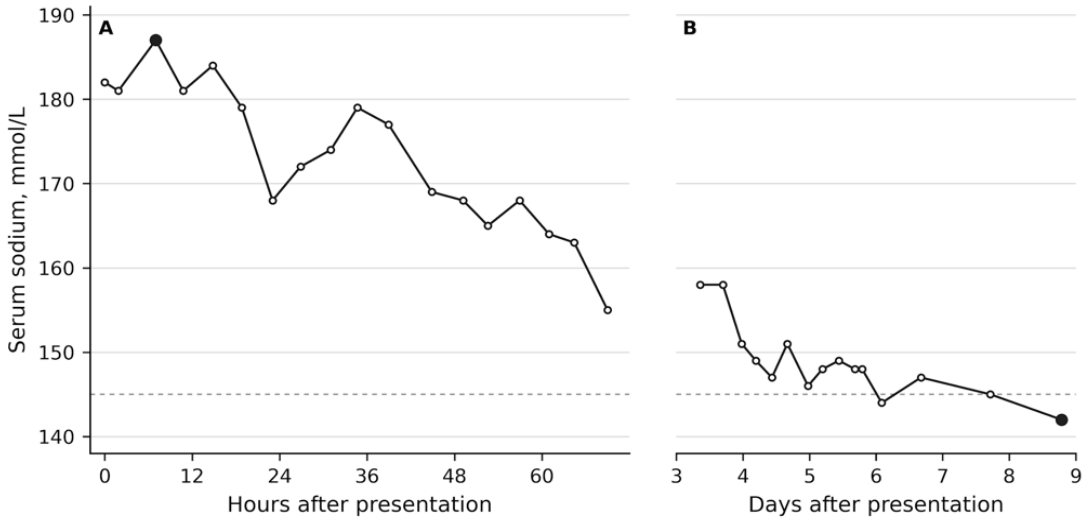


Figure 1: Serial serum sodium concentrations during hospitalization; (A) shows measurements during the first 72 hours after presentation; (B) shows the subsequent course in days through discharge.

Measurements are positioned according to their actual elapsed times. The dashed horizontal line represents the upper limit of the serum sodium reference interval (145 mmol/l). Serum sodium increased from 182 mmol/l at presentation to a peak of 187 mmol/l at approximately 7 hours and subsequently declined nonlinearly to 142 mmol/l by discharge.

DISCUSSION

Hypernatraemia most commonly reflects a deficit of water relative to total-body sodium caused by net water loss or inadequate water intake and is less commonly from hypertonic sodium administration.[1] In our patient, nonverbal status, chronic dysphagia and dependence on caregivers for fluid provision created plausible barriers at each step of adequate hydration. This vulnerability is supported by a retrospective study of 21,610 adults over 65 years, in which hypernatremia was present on admission in 12% of care-home residents compared with

1.3% of patients admitted from their own homes and the association persisted after adjustment for age, sex, dementia and mode of admission.[3] Dysphagia may have further increased this risk. In one long-term care study, four or more laboratory markers of dehydration were present in approximately 75% of orally fed residents with grade 2 swallowing difficulty, compared with 18% of nasogastric tube-fed residents.[2] These observations do not establish inadequate care in an individual patient; rather, they identify dysphagia and dependence on others for hydration as indications for active surveillance instead of reliance on expressed thirst.

The patient's concurrent hyperglycemia intensified both the water deficit and the hypertonic burden. It increased urinary water loss through osmotic diuresis while simultaneously lowering measured sodium level through transcellular water movement.[4,5] At presentation, the paired sodium of 182 mmol/l and glucose of 581 mg/dl corresponded to a Katz-corrected sodium of approximately 189.7, and an effective osmolality of 396 mOsm/kg. These values demonstrate that hyperglycemia masked rather than explained the severity of hypernatremia and confirm profound hypertonicity.[4] The early increase in sodium concentration to 187 mmol/l may partly have reflected redistribution of water as glucose declined with insulin therapy, although osmotic diuresis during initial volume resuscitation may also have contributed. During treatment of hyperglycemia, sodium levels should be interpreted alongside glucose, effective osmolality, urine output and hemodynamic response rather than in isolation.[4,5]

Management should be understood as a sequence of physiologic goals rather than as a fixed choice of fluid. In hypovolemic hypernatremia, restoration of effective circulating volume with isotonic crystalloid takes priority to address the immediate threats of impaired perfusion and prerenal kidney injury.[1,5] Accordingly, the patient initially received 0.9% saline at 100 mL/h despite the high sodium level, followed by 0.45% saline and then 5% dextrose in water when sodium reached 187 mmol/l, shifting the therapeutic goal from intravascular volume restoration to free-water replacement. The sodium trajectory was nonlinear; frequent measurements permitted repeated adjustment of fluid therapy as sodium, glucose, renal function, and ongoing water losses evolved. Sodium ultimately reached 142 mmol/l by discharge.

Mental status improved within 24 hours, while creatinine levels decreased from 1.84 to 0.93 mg/dl over the first 64 hours, without documented seizures or cerebral edema. Galili et al reported a comparable case in a 78-year-old with hyperosmolar hyperglycemia state and a corrected sodium of 184 mmol/l at presentation, who achieved complete clinical and electroencephalographic recovery with closely monitored fluid therapy.[6] More recently, Balkan et al reported complete recovery in a nonverbal, bedridden 21-year-old man with a sodium concentration of 186 mmol/l after vomiting and diarrhea through hemodynamic stabilization and controlled sodium correction.[7]

The most important lesson begins before the hospital admission. In a patient unable to report thirst or independently access fluids the earliest evidence of clinically important dehydration may be reduced interaction, increasing lethargy or loss of familiar functional ability such as inability to assist with transfers. Prevention plans should include a swallow-safe hydration plan, scheduled assisted fluid provision, closer documentation of fluid intake during reduced intake or acute illness.[8] In patients with diabetes, glucose surveillance and predefined criteria for a medical evaluation should also be incorporated.[5]

CONCLUSION

This case is valuable not simply because sodium levels exceed 180 mmol/l, but because it highlights how impaired communication, dysphagia, limited mobility, dependence for fluid access, and hyperglycemic osmotic diuresis can converge to produce extreme hypertonicity with few recognizable warning signs.

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