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Case Report

Extrapontine myelinolysis in severe hypernatremia despite controlled sodium correction: a case report

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ABSTRACT

Osmotic demyelination syndrome (ODS) is a condition characterized by sudden loss of myelin, typically triggered by rapid shifts in the body's osmotic balance. We report a 53-year-old male with recurrent carcinoma of the buccal mucosa who developed severe hypernatremia following sodium bicarbonate infusion. Despite careful and gradual correction of his elevated sodium levels, he subsequently developed quadriparesis, and MRI confirmed extrapontine myelinolysis. Supportive neurological, oncological, and hematological care was provided, but the patient ultimately succumbed to his illness. This case highlights that even cautious correction of sodium disturbances can precipitate ODS, particularly in patients with malignancy and metabolic stress, emphasizing the need for vigilant monitoring and individualized management.

Keywords: Electrolyte disturbances, Extrapontine myelinosis, Hypernatremia, Neurology, Critical care, Osmotic demyelination syndrome

INTRODUCTION

Osmotic demyelination syndrome (ODS) is a rare but serious neurological disorder characterized by demyelination predominantly affecting the central pons (central pontine myelinolysis) and extrapontine regions of the brain. Traditionally, ODS has been associated with overly rapid correction of chronic hyponatremia, leading to osmotic stress and subsequent brain cell injury. However, emerging evidence indicates that rapid fluctuations in serum sodium levels-whether from hyponatremia to normonatremia or normonatremia to hypernatremia-can precipitate ODS. This expands the clinical spectrum of ODS beyond its classical presentation, emphasizing the danger of abrupt electrolyte shifts in any direction, especially in vulnerable patients with complex medical conditions and electrolyte imbalances.[1,2] Hypernatremia, defined as a serum sodium concentration exceeding 145 mmol/l, is similarly associated with significant morbidity, including encephalopathy, seizures, rhabdomyolysis, and osmotic demyelination. Cases of extrapontine myelinolysis directly linked to hypernatremia, although infrequent, are increasingly recognized as discussed in table 2, underscoring the need for cautious management of sodium levels regardless of the initial electrolyte disturbance.[3] Patients with malignancies, malnutrition, chronic illness, or those receiving chemotherapy and multiple interventions are at heightened risk for developing ODS due to their compromised physiological state and frequent fluctuations in fluid and electrolyte status. This represents a case of extrapontine myelinolysis following rises and careful correction of hypernatremia in a patient with recurrent carcinoma of the right buccal mucosa, highlighting the clinical challenges and considerations arising from this complex interplay.

CASE REPORT

A 53-year-old male with recurrent squamous cell carcinoma of the right buccal mucosa, previously treated with surgery, radiotherapy, and chemotherapy including

Paclitaxel and Carboplatin, was admitted to the tertiary care centre with generalized weakness, poor oral intake (PEG tube in situ), throat pain, and shortness of breath. He required ICU management and underwent an emergency tracheostomy for airway compromise. He was diagnosed with starvation ketosis and metabolic acidosis therefore started on sodium bicarbonate infusion. On day 9, as mentioned in Table 1, the patient developed quadriparesis at a serum sodium concentration of 141 mmol/l.

Table 1: Clinical progression and serum sodium levels.

Days	Clinical progression & sodium levels
Day 1	Serum sodium was 143 mmol/l at baseline
DAY 2	Sodium rose sharply to 165 mmol/l. Sodium bicarbonate infusion was discontinued and normal saline was withheld
DAY 2	At 10:00 am, sodium increased further to 180 mmol/l. The patient was switched to 5% dextrose at 100 ml/hr, along with potassium phosphate infusion for correction of hypophosphatemia. At 6:30 pm (~8.5 hours later), sodium decreased to 168 mmol/L. Dextrose was stopped, and normal saline restarted at 100 ml/hr with instructions to avoid additional sodium-containing fluids after 10 pm. By 10:00 pm, sodium remained 168 mmol/l. Arterial blood gas showed resolution of metabolic acidosis (pH 7.454, HCO_3^- 26.3 mmol/l, pCO_2 38 mmHg). Intake and output were balanced (1470/1370 ml). Urine osmolarity was elevated (897 mOsm/kg), serum osmolarity was 380 mOsm/kg, indicating preserved renal concentrating ability. Neurological examination remained normal.
DAY 3	Sodium rose to 181 mmol/l. Free water intake was increased to 75 ml every 2 hours. Nephrology consultation was obtained, and oral desmopressin (0.1 mg twice daily) was initiated to limit renal free water losses and enable gradual sodium correction.
DAY 4	Serum sodium levels gradually normalized: 171 mmol/l
DAY 5	Na: 159 mmol/l
DAY 6	Na: 152 mmol/l
DAY 7	Na: 145 mmol/l
DAY 8	Na: 141 mmol/l

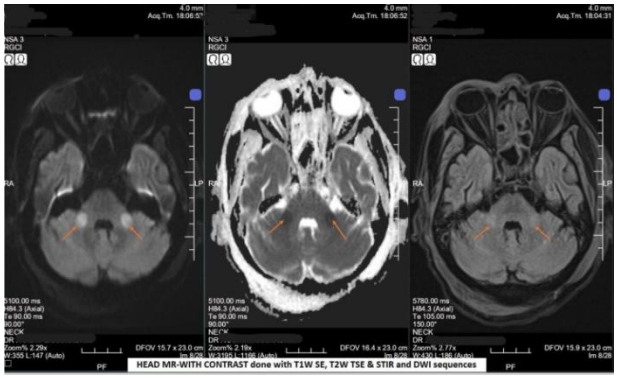

Figure 1: MRI brain showing bilateral middle cerebellar peduncle changes consistent with extrapontine myelinolysis.

Figure 1 displays the MRI images of the brain demonstrating symmetrical ovoid area of diffusion restriction in the bilateral middle cerebellar peduncles with FLAIR hyperintensity, consistent with extrapontine myelinolysis. Additional small ischemic foci were present; no metastatic disease or meningeal enhancement was identified.

After the onset of quadriparesis, neurologist opinion was obtained and appropriate management instituted. The patient also received three doses of cetuximab-based targeted therapy during the hospital stay as part of ongoing cancer-directed treatment. He was subsequently stabilized and shifted to the ward, but persistent thrombocytopenia required hematology consultation. He was managed with romiplostim, G-CSF, transfusions, and supportive care. During this period, he also developed a urinary tract infection due to Escherichia coli, which was treated with antibiotics. Later, the patient developed pancytopenia, sepsis, hypotension, and respiratory failure, necessitating ICU readmission with mechanical ventilation, inotropic support, and broad-spectrum antibiotics. Despite aggressive management, his condition deteriorated, and he eventually succumbed to his illness.

DISCUSSION

Osmotic demyelination syndrome (ODS) is traditionally associated with rapid correction of hyponatremia; however, previously reported cases have described osmotic demyelination in the setting of hypernatremia as well. The present case describes extrapontine myelinolysis in a patient with severe hypernatremia who developed neurological deterioration despite subsequent controlled correction of serum sodium. The occurrence of quadriparesis when the serum sodium had decreased to 141 mmol/l together with the characteristic MRI findings, supports the clinical diagnosis of extrapontine myelinolysis in this setting. Previously reported cases have described osmotic demyelination associated with hypernatremia under different clinical circumstances.

As summarized in Table 2, cases have included hypovolemic hypernatremia, rapid correction of severe hypernatremia, postpartum hypernatremia, and hypernatremia associated with nutritional supplementation. These reports demonstrate that hypernatremia-associated osmotic demyelination has been described in clinical settings other than the traditionally recognized correction of hyponatremia.

Table 2: Clinical progression and serum sodium levels.

Serial No	Author details	Case details
Case 1	Beraldo et al, 2020[4]	A 70-year-old woman with non-Hodgkin's lymphoma developed central pontine myelinolysis (CPM) after acute hypernatremia precipitated by severe diarrhea and chronic diuretic use. Despite cautious sodium correction, neurological deficits persisted for years, highlighting hypernatremia alone as a causative factor for osmotic injury.
Case 2	Rajasekharan et al, 2014[5]	A 53-year-old male developed acute hypernatremia (170 mmol/l) following treatment for hypoglycemia and cerebral edema, with subsequent rapid sodium correction causing extrapontine myelinolysis and reversible quadriparesis. This report emphasized the link between rapid hypernatremia correction and osmotic injury.
Case 3	Bhatia et al, 2014[3]	A 26-year-old postpartum female with sodium levels as high as 204 mEq/l developed severe extrapontine lesions with multisystem complications. Despite gradual sodium correction, neurological deterioration occurred.
Case 4	Levin et al, 2012[6]	A 74-year-old woman with lymphoma developed osmotic myelinolysis following rapid sodium increase suspected from nutritional supplementation, without prior hyponatremia correction, underscoring hypernatremia as an independent risk for ODS.

Hypernatremia leads to brain cellular dehydration and hyperosmolarity, causing significant neurological risks including seizures, encephalopathy, and ODS. Traditionally, cautious correction has been emphasized limiting serum sodium reduction to no more than 10-12 mmol/l per day to prevent cerebral edema and demyelination. Management of hypernatremia involves identifying and treating the underlying cause and carefully correcting the hyperosmolar state. Patients with acute symptomatic hypernatremia may benefit from somewhat more aggressive correction (1-2 mmol/l/hr for the first few hours), while chronic or uncertain duration hypernatremia warrants slower correction. Close monitoring of serum sodium every 2-4 hours during correction is essential to avoid complications such as cerebral edema or seizures.[7,8]

In the present case, serum sodium increased from 143 mmol/l to a peak of 181 mmol/l following sodium bicarbonate administration. Sodium bicarbonate was discontinued, sodium-containing fluids were withheld, and free-water replacement was initiated. Nephrology consultation was obtained and desmopressin was subsequently administered to limit renal free-water losses and facilitate gradual sodium correction. Serum sodium subsequently decreased from 181 mmol/l to 141 mmol/l over the following days. Despite this controlled correction, the patient developed quadriparesis when the serum sodium had reached 141 mmol/l.

The optimal rate of correction of severe hypernatremia remains clinically important. While traditional recommendations have emphasized cautious correction, recent observational data have not demonstrated increased mortality with faster rates of correction in adults with severe hypernatremia.[9] These findings highlight the complexity of determining an appropriate correction strategy and support consideration of the individual clinical context when managing severe sodium disturbances.

The neurological findings were associated with MRI abnormalities demonstrating symmetrical areas of diffusion restriction and FLAIR hyperintensity involving the bilateral middle cerebellar peduncles, consistent with extrapontine myelinolysis. Additional small ischemic foci were identified, while no metastatic disease or meningeal enhancement was seen.

This case adds to the limited reports of hypernatremia-associated extrapontine myelinolysis and highlights the occurrence of neurological deterioration despite controlled subsequent sodium correction. Careful monitoring of serum sodium and neurological status remains important during the management of severe hypernatremia, particularly in patients with malignancy and other significant systemic complications.

CONCLUSION

This case describes extrapontine myelinolysis occurring in the setting of severe hypernatremia despite controlled subsequent correction of serum sodium. Vigilant sodium monitoring, prevention of iatrogenic sodium loads, and careful, patient-tailored correction strategies are critical for minimizing harm. This case highlights the limitations

of current therapies once osmotic injury occurs and underscores the need for further research focused on biomarkers for early detection and novel therapies for the acute and long term management of osmotic demyelination.

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