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Single Case – Brain Injury

Hypernatremia as the Initial Presentation of Osmotic Demyelination Syndrome: A Rare Case with Favorable Neurological Outcome

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Keywords

Hypernatremia · Osmotic demyelination syndrome · Wernicke's encephalopathy · Central pontine myelinolysis · Case report

Abstract

Introduction: Osmotic demyelination syndrome (ODS) occurs due to rapid shifts in serum sodium levels and osmolality, resulting in neuronal damage. While it is most often caused by the rapid correction of chronic hyponatremia, other electrolyte imbalances have also been identified as potential triggers. Risk factors include alcohol abuse, malnutrition, and liver disease, among others, with common symptoms including tetraparesis, dysphagia, and altered consciousness. ***Case Presentation:*** We report the case of a 45-year-old male with alcohol dependence who presented to the emergency department with hypernatremia (154 mmol/L), altered mental status, and slurred speech. Despite gradual sodium correction, his condition deteriorated, and head MRI revealed findings consistent with central pontine myelinolysis and Wernicke's encephalopathy. After 5 months of hospitalization for severe ODS, including the need for a tracheostomy and gastrostomy, extensive rehabilitation enabled the patient to regain full independence in daily activities. ***Conclusion:*** This is a rare case of ODS presenting with hypernatremia in the setting of dehydration, malnutrition, and alcohol dependence. A preceding chronic osmotic adaptation, possibly related to unrecognized hyponatremia, may have contributed, although this could not be confirmed. Early detection, aided by MRI, was critical. Despite the severity of ODS, gradual correction of hypernatremia and extensive rehabilitation led to substantial recovery, highlighting the importance of long-term management and rehabilitation in improving patient outcomes.

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Introduction

Osmotic demyelination syndrome (ODS) is a rare but serious neurological complication that includes central pontine myelinolysis (CPM) and extrapontine myelinolysis (EPM). It results from significant changes in serum sodium levels and plasma osmolality, leading to cerebral apoptosis and myelin loss. The most common cause is iatrogenic rapid correction of chronic hyponatremia [1, 2]. Risk factors for ODS include excessive alcohol use, malnutrition, chronic diuretic use, psychogenic polydipsia, dehydration and eating disorders among others [2–4].

CPM presents with a range of neurological symptoms, including altered consciousness, spastic tetraparesis, oculomotor dysfunction, and pseudobulbar paresis manifesting as dysarthria and dysphagia. Prompt recognition and appropriate management are essential to mitigate the complications and reduce mortality [5]. We report a patient who presented at the emergency department (ED) with hypernatremia and is presumed to have developed CPM prior to admission.

Case Report

A 45-year-old man with severe alcohol dependence presented to the ED with abdominal pain, vomiting, generalized weakness and altered consciousness. The patient was not taking any regular medications and had no ongoing medical follow-up. Collateral history revealed significant weight loss and progressive deterioration over the past 4 months, with immobility for approximately 1 week. His speech had become increasingly slurred and difficult to understand. On examination in the ED, he was disoriented, confused, and unable to provide his medical history. He demonstrated generalized weakness with upper motor neuron signs, including brisk tendon reflexes in all extremities and bilateral extensor plantar responses, as well as dysarthria and cerebellar impairment, although he retained the ability to move in bed. Except for sinus tachycardia of 117 bpm his vital signs were normal.

Laboratory results showed hypernatremia (154 mmol/L), potassium at the lower limit of normal values (3.5 mmol/L), elevated blood urea nitrogen (8.3 mmol/L) and a creatinine of 101 μmol/L. Liver function tests revealed elevated serum alkaline phosphatase (608 U/L) and gamma-glutamyl transferase levels (1973 U/L), serum ammonia level was normal. Calculated serum osmolality was elevated at 324 mosm/kg and the blood ethanol levels were negative. Fluid replacement, along with vitamin B1 and B6 supplementation, was initiated with a goal of achieving gradual sodium correction as detailed in Figure 1. The patient received 500 mg of vitamin B1 on the first day, followed by 100 mg every 12 h thereafter. Given the elevated inflammatory markers treatment with amoxicillin and clavulanic acid was started. Due to the patient's altered consciousness, a head CT was performed, which did not reveal any acute abnormalities.

The patient was transferred to the Gastroenterology Intensive Care Unit, where he continued to exhibit weakness, dysarthria, and dysphagia. An EEG revealed diffuse slowing of the baseline brain activity consistent with pronounced encephalopathy. On the fifth hospital day, his mental status further deteriorated, necessitating urgent intubation due to acute hypoxemic respiratory failure. He was subsequently transferred to the Clinical Department for Intensive Internal Medicine. Upon transfer, a repeat head CT revealed a 2.5 × 2 × 2.1 cm hypodense area in the central pons, suggesting CPM. An urgent MRI of the head confirmed findings consistent with ODS, along with changes indicative of Wernicke's encephalopathy (Fig. 2).

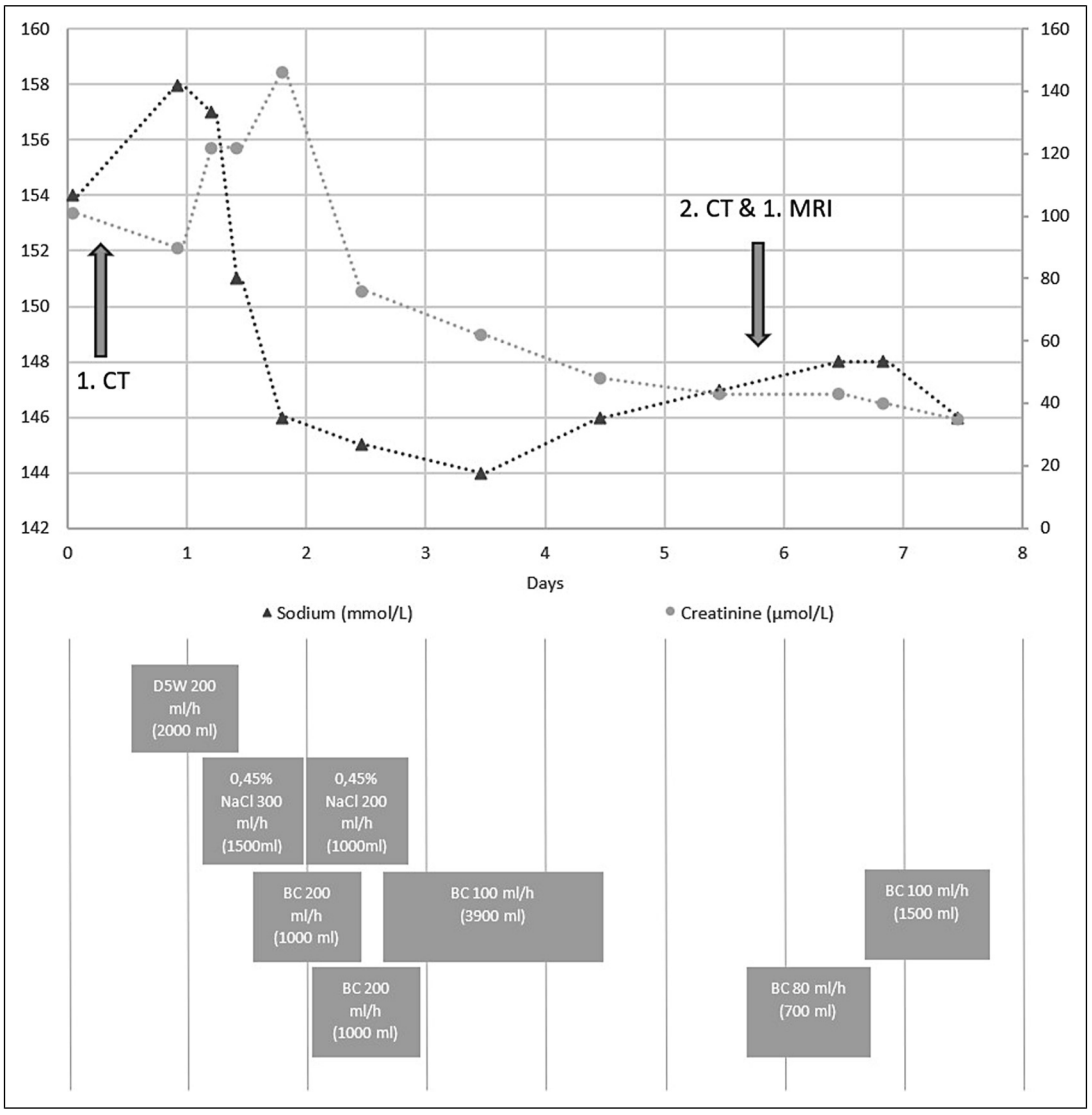

Fig. 1. Serum sodium concentration and serum creatinine over time since emergency department admission. The lower panel illustrates administered parenteral fluid therapy (type of fluid, infusion rate, and cumulative volume over time). CT, computed tomography; MRI, magnetic resonance imaging; BC, balanced crystalloids; NaCl, sodium chloride; D5W, 5% dextrose in water.

Along with supportive care, percutaneous tracheostomy was performed in anticipation of prolonged rehabilitation. The patient regained consciousness but remained confused, with significant attention deficits, dysphagia, and anarthria. He exhibited severe tetraparesis, with only minimal distal movement noted in the fingers bilaterally. After 5 days of treatment in the medical intensive care unit, the patient was transferred to the Neurology Department. At admission, he exhibited severe tetraparesis with brisk reflexes and bilateral extensor plantar responses, with minimal distal movement remaining in the left lower limb. He was aphagic and required a nasogastric tube, anarthric, and communicated only by eye blinking. At this stage, ataxia could not be reliably assessed due to the extent of weakness, but pronounced nystagmus was present. Due to persistent dysphagia, a percutaneous gastrostomy was performed.

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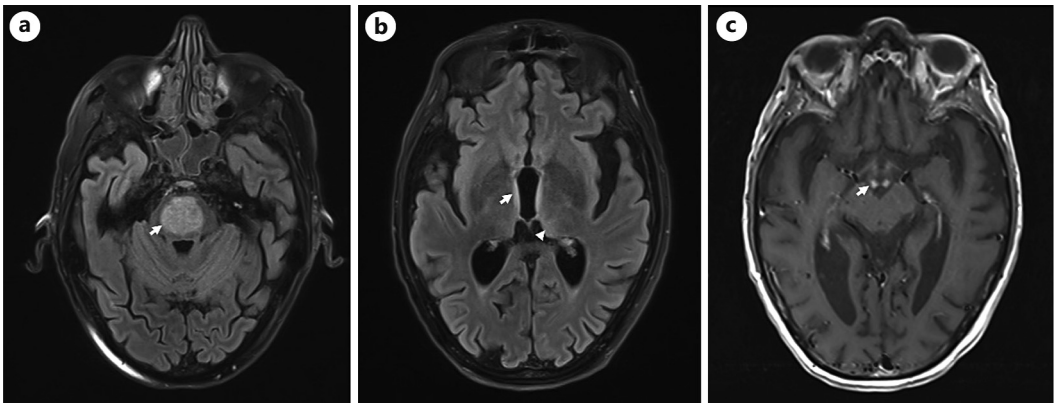

Fig. 2. MRI demonstrating: a FLAIR/T2 hyperintense lesion in the central pons (arrow), consistent with central pontine myelinolysis (**a**); FLAIR/T2 hyperintensity in the wall of the third ventricle (arrow) and dorsomedial thalami (arrowhead) (**b**); and post-contrast enhancement of the mammillary bodies on the T1-weighted sequence (arrow) (**c**). The findings in (**b**) and (**c**) together are consistent with Wernicke encephalopathy.

Over the next months, the patient demonstrated steady progress, eventually being able to sit independently in a wheelchair and walk with the assistance of one person. The tracheostomy tube was removed, and oral feeding was successfully initiated. After approximately 5 months of hospitalization, he was discharged to an intensive neurorehabilitation program.

During inpatient neurorehabilitation, the patient received an intensive multidisciplinary programme consisting of daily physiotherapy (targeting passive and active range of motion, strengthening, balance training and gait re-education), occupational therapy (training of dressing, hygiene, transfers and functional routines), and speech-swallowing assessment, which confirmed safe oral intake with modified food consistency. Nutritional optimization was provided in collaboration with the clinical nutrition team. Neuropsychological assessment revealed a marked cognitive deficit, and supportive cognitive-behavioral strategies were implemented. Over the course of rehabilitation, the patient progressed from requiring two-person assistance for transfers and ambulation to walking with a four-wheel walker under supervision, and achieved supervision-level independence in dressing, feeding, toileting and basic hygiene.

One year later the patient continued to make significant progress. At follow-up he walked several kilometers daily using Nordic walking poles. He actively participated in daily activities at home. Neuropsychological testing confirmed cognitive improvement, although deficits in attention and orientation persisted. Neurological examination showed mild spastic tetraparesis and a broad-based ataxic gait.

Discussion

ODS presents with a range of neurological deficits typically emerging 2–7 days following changes in serum electrolyte levels and osmolality, most commonly after rapid correction of chronic hyponatremia [6]. Less frequently, other electrolyte abnormalities have been reported as a cause, such as hyperglycemia, hypoglycemia, hypokalemia, and hypernatremia have been implicated [7, 8]. A literature review by Ismail et al. reported five cases of isolated CPM in patients presenting with hypernatremia: two were attributed to iatrogenic causes,

two to diabetes insipidus, and one to acute respiratory distress syndrome (ARDS) with sepsis [2]. A similar case to ours was reported, describing hypernatremia in a patient with liver cirrhosis [9].

While the mechanisms underlying hypernatremic ODS are not fully understood, various hypotheses have been proposed [2]. In our case, chronic hyponatremia was considered a possible contributing factor based on the patient's history of alcohol abuse, although this remains speculative in the absence of prior laboratory data. Due to general malaise, inadequate water intake, and dehydration, he subsequently developed hypernatremia, whereas potassium levels were at the lower limit of normal and, therefore, hypokalemia correction was not clinically relevant in this case. Although the patient's creatinine and BUN levels were relatively low upon presentation, a significant decline in creatinine was observed over the following days, returning to his low baseline values (Fig. 1). This was likely a consequence of cachexia. Hypernatremia correction was gradual and unlikely to have caused neurological damage.

Coexisting Wernicke's encephalopathy likely contributed significantly to the initial clinical presentation in this patient. The history of chronic alcohol abuse, malnutrition, confusion preceding hospital admission and ataxia observed before the development of tetraparesis are all consistent with thiamine deficiency. MRI findings demonstrating mammillary body enhancement and periventricular signal changes further supported this diagnosis. The coexistence of Wernicke's encephalopathy and ODS has been previously reported and may exacerbate the severity of neurological deficits, complicating both diagnosis and early clinical assessment [10, 11]. More broadly, this case illustrates the complex clinical syndrome that may arise from the combined effects of dehydration, malnutrition, and chronic alcohol dependence. These factors frequently coexist in clinical practice and contribute to overlapping metabolic and nutritional encephalopathies, complicating early neurological assessment [12]. This case, therefore, underscores the importance of maintaining a broad diagnostic perspective and reassessing the diagnosis when neurological deterioration occurs, particularly in vulnerable patients with alcohol dependence, dehydration, and malnutrition [13].

Despite the severity of neurological impairment and the need for prolonged intensive care, the patient demonstrated substantial neurological recovery. Gradual correction of hypernatremia, early administration of thiamine, and prolonged multidisciplinary rehabilitation were likely key factors contributing to the favorable outcome. This favorable outcome is particularly noteworthy, as only about one-third of ODS patients demonstrate satisfactory recovery without persistent neurological deficits and highlights the potential for significant improvement even after severe presentations when comprehensive supportive care and rehabilitation are provided [14, 15].

Published observational data indicate that the strongest predictors of poor outcome are severe hyponatremia, hypokalemia, low GCS at presentation, poor baseline functional status, malnutrition, liver disease and alcohol dependence [15, 16]. Our patient lacked several of these predictors, particularly severe hyponatremia and hypokalemia, and may have had some preserved premorbid functional reserve, which may partly explain the favorable trajectory despite malnutrition and alcohol dependence.

Several case reports also describe substantial recovery, including from locked-in syndrome, following prolonged and structured multidisciplinary rehabilitation, supporting its role as an important adjunct in functional improvement [17, 18]. In our case, the patient's rehabilitation programme included coordinated physiotherapy, occupational therapy, swallowing assessment, nutritional optimization and neuropsychological support, all of which enabled progressive gains in mobility, self-care and participation. While these interventions likely contributed meaningfully to the patient's recovery,

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rehabilitation should be regarded as one of several interacting factors such as the absence of severe hyponatremia and some preserved functional reserve, rather than the sole determinant of outcome.

Conclusion

This case emphasizes that ODS may occur in the setting of hypernatremia, particularly in vulnerable patients with chronic alcoholism and malnutrition. Clinicians should consider ODS even in the absence of rapid hyponatremia correction. Importantly, substantial neurological recovery is possible with early recognition and prolonged rehabilitation.

Statement of Ethics

According to national regulations and institutional policies, ethical approval was not required for this case report. Written informed consent was obtained from the patient for publication of the details of this medical case and any accompanying images. The authors have completed the CARE Checklist for this case report. The completed checklist has been submitted as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000552584).

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Gasper Govekar drafted the manuscript and collected the clinical data. Tomaz Rus contributed to data interpretation and critically revised the manuscript. Peter Kordis supervised the project and provided final approval of the version to be published. All authors read and approved the final manuscript.

Data Availability Statement

The data that support the findings of this case report are not publicly available because they were derived from the patient's medical records and contain information that could compromise patient privacy (GDPR) if disclosed. De-identified data extracts relevant to this report may be made available from the corresponding author upon reasonable request, subject to institutional approval and applicable data protection requirements.

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