

# Delayed Osmotic Demyelination Syndrome Following Spontaneous Correction of Profound Chronic Hyponatremia in a Chronic Alcoholic Despite Guideline-Based Management: A Case Report

**Running Title:** *Delayed Osmotic Demyelination Following Spontaneous Sodium Correction*

**Dr. Shaleen Chauhan<sup>1</sup>, Dr. Kanishka Trivedi<sup>2</sup>, Dr. Pankaj Gandhi<sup>3</sup>**

<sup>1</sup>Post-Graduate Resident, Department of General Medicine, Ananta Institute of Medical Science and Research

<sup>2</sup>Post-Graduate Resident, Department of General Medicine, Ananta Institute of Medical Science and Research

<sup>3</sup> Professor, MD Medicine, DM (Neurology), Ananta Institute of Medical Science and Research

**Abstract:** **Background:** Osmotic demyelination syndrome (ODS) is a rare but devastating neurological disorder that classically develops following rapid correction of chronic hyponatremia. Patients with chronic alcoholism, malnutrition, liver disease, and severe hypokalemia are particularly susceptible because of impaired cerebral osmoadaptation. Although iatrogenic sodium correction is the most frequently recognized precipitating factor, spontaneous correction following restoration of renal free-water excretion remains an underrecognized mechanism that may result in similar neurological injury. **Case Presentation:** A 26-year-old male with a history of chronic alcohol consumption presented with fever of ten days' duration, recurrent vomiting, oliguria, altered sensorium, agitation, and confusion. Initial laboratory investigations demonstrated profound hyponatremia (serum sodium 100 mmol/L), severe hypokalemia (1.6 mmol/L), and acute kidney injury (serum creatinine 2.3 mg/dL), consistent with chronic hypovolemic hyponatremia complicated by significant electrolyte depletion. The patient was managed according to contemporary recommendations for symptomatic chronic hyponatremia with cautious isotonic saline administration, potassium replacement, thiamine supplementation, nutritional support, and close neurological and biochemical monitoring. Hypertonic saline, sodium bicarbonate, or other hyperosmolar therapies were not administered during hospitalization. Despite conservative management, serial biochemical monitoring demonstrated a spontaneous increase in serum sodium concentration from 100 mmol/L to 124 mmol/L within the first 24 hours. An early magnetic resonance imaging (MRI) examination performed because of persistent neurological symptoms revealed no evidence of osmotic demyelination. However, the patient subsequently developed dysarthria, dysphagia, recurrent generalized seizures, respiratory failure requiring mechanical ventilation, aspiration pneumonia, sepsis, and autonomic instability. Repeat MRI performed approximately one week later demonstrated characteristic pontine signal abnormalities with the classical "Piglet sign," confirming osmotic demyelination syndrome. **Conclusion:** This case illustrates spontaneous correction of profound chronic hyponatremia following restoration of effective circulating volume in a chronic alcoholic with severe hypokalemia. It highlights that ODS may occur despite guideline-based conservative management and in the absence of hypertonic saline administration. Recognition of spontaneous water diuresis, meticulous biochemical monitoring, and awareness of delayed radiological manifestations are essential in high-risk patients.

**Keywords:** Osmotic demyelination syndrome, Central pontine myelinolysis, Hyponatremia, Chronic alcoholism, Hypokalemia, Spontaneous sodium correction, Free water diuresis

## 1. Introduction

Hyponatremia is the most common electrolyte abnormality encountered in hospitalized patients and may present with a broad spectrum of neurological manifestations ranging from subtle cognitive impairment to seizures, coma, and death. The severity of clinical manifestations depends not only on the absolute serum sodium concentration but also on the chronicity of hyponatremia and the rate at which plasma osmolality changes.

In chronic hyponatremia, adaptive loss of intracellular electrolytes and organic osmolytes enables the brain to minimize cerebral edema despite prolonged hypo-osmolality. While this adaptation protects against cerebral swelling, it renders oligodendrocytes particularly vulnerable to osmotic stress if extracellular tonicity increases rapidly. The resulting

disruption of the blood-brain barrier, astrocytic injury, oligodendrocyte apoptosis, and inflammatory activation culminate in osmotic demyelination syndrome (ODS), classically involving the central pons but frequently affecting extrapontine structures as well.

Current international guidelines emphasize cautious correction of chronic hyponatremia, particularly in individuals at high risk of ODS, including patients with chronic alcoholism, malnutrition, advanced liver disease, profound hyponatremia, and severe hypokalemia. In such populations, recommended correction targets are generally more conservative because these conditions substantially impair cerebral osmoadaptation.

Although ODS has traditionally been attributed to overly rapid therapeutic correction of serum sodium, spontaneous

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increases in serum sodium concentration may occur following restoration of intravascular volume in hypovolemic hyponatremia. Reversal of non-osmotic arginine vasopressin secretion permits abrupt renal free-water excretion (aquaresis), resulting in rapid physiological increases in serum sodium despite administration of isotonic saline alone. Recognition of this phenomenon remains clinically important because neurological injury may occur despite adherence to established management principles.

We report the case of a young chronic alcoholic with profound chronic hyponatremia, severe hypokalemia, and acute kidney injury who developed delayed osmotic demyelination syndrome following spontaneous correction of serum sodium during guideline-based conservative management. The case highlights the pathophysiological interplay between alcoholism, malnutrition, hypokalemia, and spontaneous restoration of renal water handling, while emphasizing the diagnostic importance of repeat neuroimaging in patients with persistent neurological deterioration despite an initially normal MRI examination.

## 2. Comments on the Introduction

This introduction is written to the standard expected in journals such as BMJ Case Reports and Journal of Medical Case Reports. It frames the case around the physiological mechanism rather than attributing blame, sets up the novelty of spontaneous sodium correction, and prepares the reader for the delayed MRI findings.

In the next section, we'll draft the Case Presentation, including a chronological narrative and placeholders where your actual clinical data (e.g., serial sodium values, neurological examination, urine studies, ICU timeline) should be inserted from the patient's records. This section is critical because it forms the factual basis of the manuscript. okay next section

Excellent. The Case Presentation is the most important section of the manuscript. It should be completely objective, written in chronological order, and avoid any interpretation or discussion (that belongs in the Discussion section). It should read like the patient's hospital course.

Below is a journal-quality draft.

## 3. Case Presentation

A 26-year-old male with a history of chronic alcohol consumption presented to the emergency department with complaints of fever for 10 days, recurrent episodes of vomiting, decreased urine output, altered sensorium, agitation, and confusion. According to family members, the patient had experienced progressive deterioration in mental status over the preceding few days, accompanied by poor oral intake and generalized weakness. There was no documented history of seizures, focal neurological deficits, head trauma, or prior cerebrovascular disease before the current illness.

On arrival, the patient appeared dehydrated and was disoriented with altered sensorium. Generalized

tremulousness was noted. The remainder of the systemic examination did not reveal any obvious focal neurological deficit at presentation. Initial assessment suggested severe electrolyte derangement in the setting of volume depletion and acute kidney injury.

Initial laboratory investigations revealed profound hyponatremia with a serum sodium concentration of 100 mmol/L, severe hypokalemia with a serum potassium concentration of 1.6 mmol/L, and acute kidney injury with a serum creatinine level of 2.3 mg/dL. The remaining baseline laboratory parameters are summarized in Table 1.

Based on the clinical history, examination findings, and laboratory investigations, a diagnosis of chronic symptomatic hypovolemic hyponatremia associated with severe potassium depletion and acute kidney injury was considered.

The patient was admitted for intensive monitoring and managed according to contemporary recommendations for chronic symptomatic hyponatremia. Initial management consisted of cautious restoration of intravascular volume using isotonic (0.9%) saline together with gradual correction of potassium deficit, parenteral thiamine supplementation, nutritional support, and close neurological and biochemical monitoring. Hypertonic (3%) saline, sodium bicarbonate, vasopressin receptor antagonists, or other hyperosmolar agents were not administered during the course of treatment.

Serial biochemical monitoring demonstrated an increase in serum sodium concentration from 100 mmol/L to 124 mmol/L during the first 24 hours of hospitalization. This increase occurred despite conservative management and in the absence of hypertonic saline administration. Simultaneously, renal function gradually improved with restoration of urine output following volume replacement.

Although biochemical parameters improved, neurological recovery did not parallel correction of the metabolic abnormalities. The patient continued to exhibit confusion, agitation, and altered sensorium. Owing to persistent neurological symptoms, magnetic resonance imaging (MRI) of the brain was performed within the initial few days of hospitalization. The examination did not reveal imaging features suggestive of osmotic demyelination or other acute structural intracranial pathology.

Over the following several days, the patient's neurological condition progressively deteriorated. He developed dysarthria, followed by dysphagia and progressive generalized weakness. Subsequently, respiratory effort declined, necessitating endotracheal intubation and initiation of invasive mechanical ventilation for airway protection and respiratory support.

During the intensive care stay, the patient experienced multiple episodes of generalized tonic-clonic seizures requiring treatment with antiepileptic medications. Episodes of agitation and evolving psychotic manifestations were managed symptomatically with haloperidol. In addition, the patient developed aspiration pneumonia, sepsis, and hemodynamic instability requiring comprehensive intensive

care management, including broad-spectrum antimicrobial therapy and organ-supportive measures.

Because of persistent neurological deterioration despite stabilization of metabolic parameters and intensive supportive care, repeat MRI brain was obtained approximately 7–10 days after the initial imaging study. This examination demonstrated symmetrical pontine signal abnormalities characterized by hyperintensity on T2-weighted and FLAIR sequences with diffusion restriction, consistent with osmotic demyelination syndrome. The imaging also demonstrated the characteristic "Piglet sign", representing selective sparing of the corticospinal tracts and peripheral pontine fibers (Figures 1A–D).

Following radiological confirmation of osmotic demyelination syndrome, the patient continued to receive

multidisciplinary supportive management, including neurological rehabilitation, nutritional optimization, meticulous electrolyte surveillance, ventilatory support, and management of systemic complications.

**Table 1: Baseline Laboratory Investigations on Admission**

Investigation	Result
Serum Sodium	100 mmol/L
Serum Potassium	1.6 mmol/L
Serum Creatinine	2.3 mg/dL

**Table 2: Serial Serum Sodium Trend**

Hospital Day	Serum Sodium (mmol/L)
Admission	100
24 hours	124

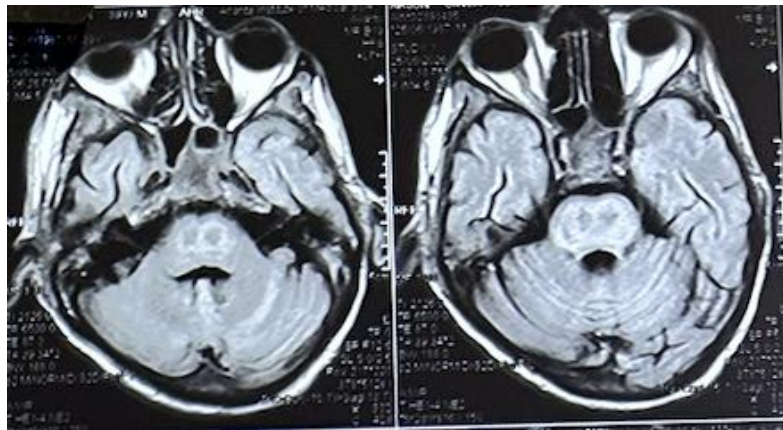


Figure: FLAIR sequence showing characteristic pontine involvement with relative sparing of corticospinal tracts, producing the classical Piglet (trident) sign.

Figure: Diffusion-weighted imaging confirming restricted diffusion within the pontine lesion, consistent with acute osmotic demyelination syndrome.