Central and Extrapontine Myelinolysis Following Rapid Correction of Hyponatremia: A Case Report

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ABSTRACT

Osmotic demyelination syndrome (ODS) is a serious neurological complication that occurs most frequently following rapid correction of chronic hyponatremia. It comprises central pontine myelinolysis (CPM) and extrapontine myelinolysis (EPM), which may occur together or in isolation. We report a case of a 50-year-old male with a history of rapid correction of hyponatremia who presented with loss of consciousness. Magnetic resonance imaging (MRI) of the brain was performed. Diffusion-weighted images revealed a trident pattern of restricted diffusion involving the pontine region crossing the midline, characteristic of central pontine myelinolysis. Additionally, foci of restricted diffusion were seen involving the anterior limb of the internal capsule and bilateral thalamic regions, with symmetrical hyperintensities on FLAIR images involving the bilateral caudocapsular, gangliocapsular, and thalamocapsular regions, consistent with extrapontine myelinolysis. Cortical surface hyperintensities were noted in bilateral temporoparietal lobes, insular cortex, and perisylvian region, likely related to EPM and associated hypoxia. Incidental findings included periventricular white matter hyperintensities suggestive of small vessel disease, right maxillary polyp, and bilateral ethmoidal sinusitis. This case illustrates the classic MRI findings of ODS with both pontine and extrapontine involvement, emphasizing the critical importance of slow correction of hyponatremia to prevent this devastating complication.

Keywords: Osmotic demyelination syndrome; Central pontine myelinolysis; Extrapontine myelinolysis; Hyponatremia; Rapid correction; MRI

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Introduction

Osmotic demyelination syndrome (ODS) is a neurological disorder characterized by non-inflammatory demyelination that typically affects the central pons (central pontine myelinolysis, CPM) and may also involve extrapontine sites such as the basal ganglia, thalamus, and cerebral white matter (extrapontine myelinolysis, EPM).[1] The most common precipitating factor is the rapid correction of chronic hyponatremia, although ODS has also been reported in patients with alcoholism, liver disease, malnutrition, and burns.[2] The pathophysiology involves osmotic stress to oligodendrocytes, leading to myelin sheath disruption while sparing axons and neurons.[3]

Hyponatremia is the most frequent electrolyte abnormality encountered in clinical practice. When hyponatremia develops slowly (over more than 48 hours), the brain adapts by extruding organic osmolytes such as myoinositol and taurine to reduce intracellular osmolality and prevent cerebral edema.[4] Rapid correction of serum sodium (exceeding 8–10 mmol/L in 24 hours or 18 mmol/L in 48 hours) creates a sudden osmotic gradient that draws water out of brain cells, causing osmotic injury to oligodendrocytes, particularly in regions with dense white matter tracts.[5] The pons is especially vulnerable due to its high concentration of crossing fibers and relative lack of collateral circulation.[6]

Clinically, ODS presents with a biphasic course: the initial phase of hyponatremic encephalopathy is followed by an apparent improvement, then after 2–6 days, neurological deterioration occurs with symptoms including dysarthria, dysphagia, quadriparesis, locked-in syndrome, and altered mental status.[7] EPM may present with movement disorders, parkinsonism, dystonia, and behavioral changes.[8] Magnetic resonance imaging (MRI) is the imaging modality of choice, showing characteristic T2 and FLAIR hyperintensities with restricted diffusion in the acute phase. The "trident" or "bat wing" sign in the pons is a classic finding in CPM.[9] We present a case of a 50-year-old male who developed loss of consciousness after rapid correction of hyponatremia, with MRI findings demonstrating both central and extrapontine myelinolysis.

Case Presentation

A 50-year-old male presented with a history of rapid correction of hyponatremia, details of which were not fully specified (including the initial serum sodium level, rate of correction, and the method of correction). Following this correction, the patient developed loss of consciousness. There was no prior history of alcoholism, liver disease, or malnutrition. Previous imaging reports were available from February 1, 2024, but the findings from those studies were not provided in the current document. On neurological examination at the time of MRI, the patient was noted to have altered sensorium. No further clinical details regarding motor deficits, cranial nerve function, or reflexes were available.

Magnetic resonance imaging (MRI) of the brain was performed using diffusion-weighted imaging (DWI), apparent diffusion coefficient (ADC) mapping, T2-weighted, and fluid-attenuated inversion recovery (FLAIR) sequences. The findings were as follows. On diffusion-weighted images, there was a trident type of restricted diffusion involving the pontine region, crossing the midline, which is characteristic of central pontine myelinolysis (Figure 1). Additionally, foci of restricted diffusion were seen faintly involving the anterior limb of the internal capsule and bilateral thalamic regions. On FLAIR images, symmetrical hyperintensities were observed involving the bilateral caudocapsular region (head of caudate nucleus and anterior limb of internal capsule), gangliocapsular region (basal ganglia and internal capsule), and thalamocapsular region (thalamus and posterior limb of internal capsule), consistent with extrapontine myelinolysis (Figure 2). Cortical surface hyperintensities were noted on FLAIR images in the bilateral temporoparietal lobes, also involving the gyri, insular cortex, and perisylvian region. These findings were attributed to extrapontine myelinolysis with associated hypoxic injury. No evidence of intracranial hemorrhage was seen.

Incidental findings included periventricular white matter hyperintensities on T2 and FLAIR images, suggestive of chronic small vessel disease. The hippocampus showed no abnormal signal intensity or volume loss. The pituitary gland, infundibulum, and hypothalamus were normal for the patient's age. The cerebellum was normal. Both cerebellopontine angles were clear, and the basal cisterns were normal. Normal flow voids were seen in the major dural venous sinuses and arteries. Extracranial incidental findings included a right maxillary polyp and bilateral ethmoidal sinusitis.

The final diagnosis was central pontine myelinolysis (trident sign) with extrapontine myelinolysis involving the caudocapsular, gangliocapsular, and thalamocapsular regions, as well as cortical involvement, in the setting of rapidly corrected hyponatremia.

Discussion

This case demonstrates the classic MRI findings of osmotic demyelination syndrome (ODS) with both central pontine and extrapontine involvement following rapid correction of hyponatremia. The trident or "bat wing" pattern of restricted diffusion in the pons is pathognomonic for CPM.[9] This pattern occurs because the descending corticospinal tracts and the ascending sensory tracts are relatively spared, while the central pontine white matter tracts (transverse pontocerebellar fibers) are selectively affected. The resulting trident-shaped area of demyelination is best visualized on axial DWI and ADC maps in the acute phase (first 2–3 weeks), as seen in this patient.[7]

Extrapontine myelinolysis (EPM) occurs in approximately 10–50% of patients with CPM and may precede, accompany, or follow the pontine findings.[4] The most common extrapontine sites are the basal ganglia (particularly the putamen and caudate nucleus), thalamus, internal capsule, and cerebral white matter.[8] In this case, symmetrical involvement of the caudocapsular, gangliocapsular, and thalamocapsular regions was observed, which is typical for EPM. The cortical surface hyperintensities in the temporoparietal lobes, insula, and perisylvian region are less common

but have been described in severe cases, especially when associated with hypoxia.[10] The patient's loss of consciousness could be attributed to the combined effect of brainstem involvement (reticular activating system) and diffuse cortical/subcortical demyelination.

The temporal evolution of MRI findings in ODS is important for interpretation. In the first week after rapid correction, DWI shows restricted diffusion with low ADC values due to cytotoxic edema.[1] After 1–3 weeks, T2 and FLAIR hyperintensities become more prominent, and contrast enhancement may be seen. In the chronic phase, gliosis and atrophy develop. The presence of both DWI restriction and FLAIR hyperintensities in this patient suggests subacute disease, likely within the first few weeks of symptom onset.[1]

The differential diagnosis for symmetrical pontine and basal ganglia lesions includes hypoxic-ischemic encephalopathy, Wilson's disease, Leigh syndrome, and methanol poisoning.[6] Hypoxic-ischemic injury typically affects the basal ganglia and thalami but usually spares the pons or causes a different pattern (e.g., "lentiform fork" sign). Wilson's disease shows T1 hyperintensity in the basal ganglia and brainstem. The clinical history of rapid correction of hyponatremia is essential for making the correct diagnosis of ODS.[5]

The prognosis of ODS varies widely. Some patients recover partially or completely with supportive care, while others suffer permanent neurological deficits such as spastic quadriparesis, pseudobulbar palsy, or cognitive impairment.[10] Mortality rates in severe cases can reach 20–30%. No specific treatment exists, although some case reports suggest a potential benefit from re-lowering serum sodium or from intravenous immunoglobulins.[7] Prevention is the key: guidelines recommend that chronic hyponatremia (duration >48 hours or unknown duration) should be corrected slowly, with a goal of raising serum sodium by no more than 4–6 mmol/L in 24 hours and 8–10 mmol/L in 48 hours.[5]

Limitations of this case report include the lack of specific biochemical data (initial and corrected sodium levels, rate of correction), absence of detailed clinical follow-up, and unavailability of prior imaging for comparison. However, the imaging findings are highly characteristic, and the clinical history of rapid correction of hyponatremia with subsequent loss of consciousness strongly supports the diagnosis of ODS.

Conclusion

Central and extrapontine myelinolysis are serious complications of rapid correction of chronic hyponatremia. MRI, particularly diffusion-weighted imaging, provides a definitive diagnosis by demonstrating characteristic patterns such as the trident sign in the pons and symmetrical involvement of the basal ganglia, thalami, and internal capsule. This case underscores the importance of slow, controlled correction of hyponatremia to prevent osmotic demyelination and the need for prompt MRI evaluation in patients who develop neurological symptoms after sodium correction.

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Figure Legends

Figure 1: Diffusion-weighted axial images show trident-type restricted diffusion involving the pontine region crossing the midline (white arrow), characteristic of central pontine myelinolysis.

Figure 2: Axial FLAIR images demonstrate symmetrical hyperintensities involving bilateral head of caudate nucleus, bilateral basal ganglia, and bilateral thalamic region (arrowheads), consistent with extrapontine myelinolysis.

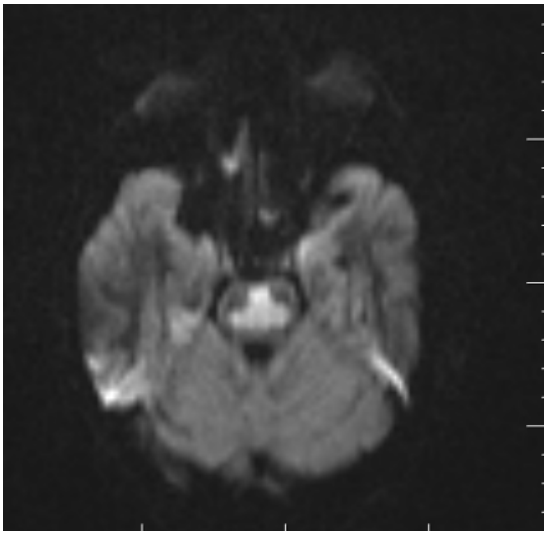

Figure 1

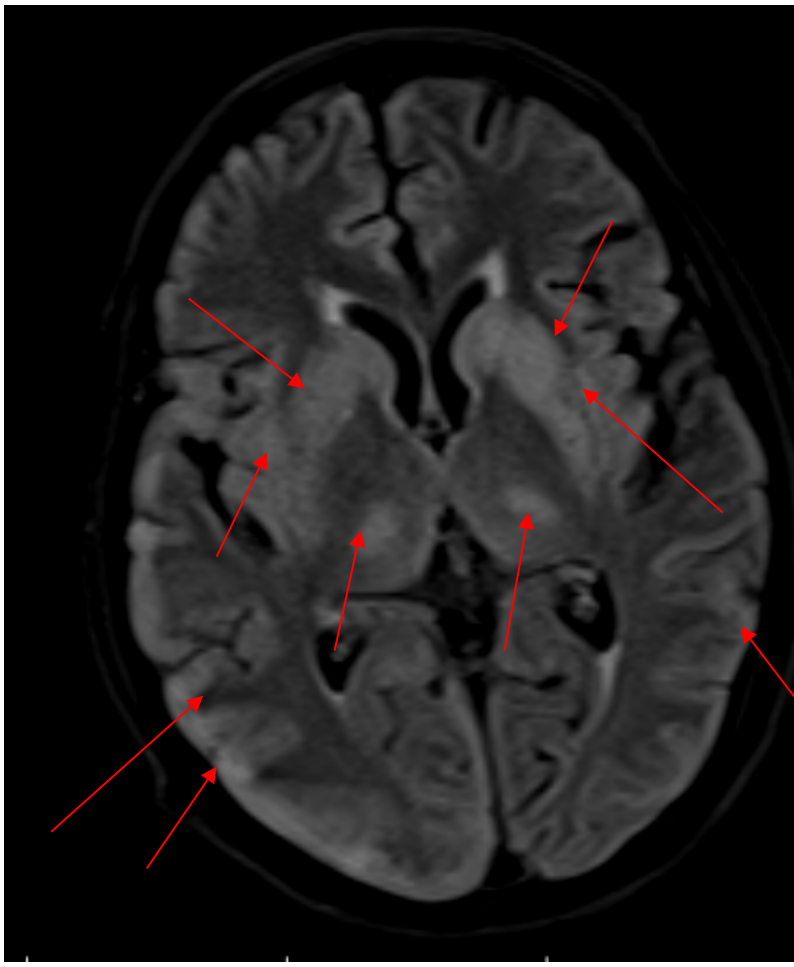

Figure 2