Case Report

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Central Pontine Myelinolysis (CPM) in an Adolescent: A Case Report

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ABSTRACT

Central pontine myelinolysis (CPM) is an osmotic demyelination disorder typically precipitated by the overly rapid correction of chronic hyponatremia; early neuroimprovement may be followed by new brainstem deficits days later. A 14-year-old girl presented with two weeks of intermittent lower-abdominal pain, one week of constipation, and four months of amenorrhea. She developed three generalized seizures with decreased postictal consciousness. Initial labs showed Na 122 mmol/L, worsening to 104 mmol/L with hypokalemia. A head CT on August 1, 2025, was normal, and a repeat CT on August 22 revealed a well-defined pontine hypodensity. Risk factors included prior malnutrition and electrolyte disturbances. Working diagnosis: decreased consciousness with suspected CPM. Severe hyponatremia was corrected by administering 50 mL of 15% NaCl diluted in D5NS (1,500 mL) over 24 hours. A bowel disimpaction with Niflec® was given for concurrent constipation/ileus. Serum electrolytes were monitored during hospitalization. This case illustrates CPM risk after sodium correction in a malnourished adolescent with profound hyponatremia. The initially normal head CT, followed by a delayed pontine lesion, aligns with the temporal evolution of CPM imaging. Vigilant recognition of vulnerability, careful correction strategies, and timely repeat neuroimaging are crucial for confirming the diagnosis and guiding supportive management in suspected CPM.

Introduction

Central pontine myelinolysis (CPM) is a serious demyelinating disorder centered in the basis pontis and most often precipitated by overly rapid correction of chronic hyponatremia. Studies suggest that individuals with prolonged or severe sodium deficits (particularly sodium <120 mEq/L or hyponatremia lasting beyond 48 hours) are at a greater risk, especially those with alcoholism, malnutrition, liver disease, and hyperemesis gravidarum [1-2]. Clinically, CPM is frequently followed by a biphasic trajectory. Early encephalopathy or seizures may improve as sodium normalizes. Then, new deficits emerge several days later, such as dysarthria, dysphagia, spastic quadriparesis, pseudobulbar palsy, and, in severe cases, locked-in syndrome or coma [2-3].

Typically, diagnosis relies on the history of recent sodium correction, compatible neurological signs, and exclusion of alternative causes. A careful review of the correction rate is essential because increases exceeding roughly 0.5–1.0 mEq/L per hour have been implicated in pathogenesis [2]. Magnetic resonance imaging (MRI) further supports the diagnosis: diffusion-weighted imaging can show early diffusion restriction in the central pons within 24 hours of symptom onset. Meanwhile, T2/FLAIR sequences often demonstrate the characteristic “bat-wing” hyperintensity at a later stage.

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Importantly, a normal initial MRI does not exclude CPM, and repeat imaging is needed if suspicion remains high [1-2]. Besides imaging, histopathology shows concentrated demyelination with astrocytosis and inflammatory infiltrates, and lesions may extend to extrapontine regions in some patients [2]. These features indicate that the diagnosis only becomes clear when an injury has already occurred. For this reason, prevention still becomes the primary therapeutic approach. Studies indicate that slow, carefully monitored correction of chronic hyponatremia is preferred. In many cases, the target is to limit sodium correction to no more than 4–6 mmol/L per 24 hours, although several guidelines allow slightly higher limits, such as 6–8 or up to 8–12 mEq/L per 24 hours, depending on the severity and risk factors. However, CPM has still been reported within these conservative ranges, especially in patients with alcoholism or malnutrition [2,4-6]. In severe symptomatic cases, controlled 3% hypertonic saline may be necessary. Pairing this with scheduled desmopressin (administered every 6–8 hours in some protocols) can reduce free-water diuresis and help prevent overcorrection. Frequent monitoring of sodium levels, typically every 4–6 hours or hourly if correction accelerates, is essential. Once CPM occurs, management is largely supportive. There is insufficient evidence for the routine use of corticosteroids, plasmapheresis, immunoglobulins, or thyrotropin-releasing hormone, despite small studies investigating their application; outcomes vary from complete recovery to permanent disability [5]. In addition to rapid sodium shifts, several conditions have been identified as increasing vulnerability to CPM, such as chronic alcoholism, malnutrition, liver disease (including transplantation), major burns, critical illness, and hyperosmolar states, including uncontrolled hyperglycemia. Pregnancy and the postpartum period have also appeared in the case literature as potential contexts for onset. Although the classical osmotic demyelination model remains important in diagnosing CPM, additional hypotheses, such as impaired cellular energy and pro-apoptotic signaling, may explain inter-individual susceptibility and why some patients develop CPM despite guideline-concordant correction rates [7-9]. For this reason, recent practice recommendations continue to emphasize conservative correction ceilings—often ≤10–12 mmol/L in the first 24 hours and <18 mmol/L over 48 hours, with even slower targets (e.g., <8 mmol/L/24 h) for patients with high-risk chronic hyponatremia—plus vigilant monitoring and careful use of treatments that could trigger rapid sodium increases [10-14].

Case Report

A 14-year-old girl presented on 25 July 2025 with intermittent lower-abdominal pain lasting two weeks. She had not defecated for one week and had experienced amenorrhea for the past four months. There was no fever, cough, or urinary complaint. She was admitted to the ED observation area (also known as a buffer room) on the same day. The following day, her consciousness declined after three morning seizures. Each seizure lasted about two minutes, characterized by upward eye deviation, tonic stiffening of the hands and feet, and frothing at the mouth. Additionally, she was difficult to communicate with postictally. Her medical history included a hospitalization in July 2024 in the Bona Ward for acute post-streptococcal glomerulonephritis, salt-wasting nephropathy, hypertension, urinary tract infection, and bilateral grade-1 hydronephrosis, all of which improved. At that time, she was also reported to have hyponatremia, hypokalemia, anemia, and moderate malnutrition. The malnutrition and hypokalemia identified in the patient are known to increase vulnerability to CPM.

Besides the clinical signs mentioned above, neuroimaging findings help confirm the diagnosis. A contrast head CT was conducted on August 1, 2025, and the result was normal. Another contrast head CT was performed on 22 August 2025 and showed a well-defined pontine hypodensity, initially read as 'chronic ischemic infarction,' but overall, more compatible with CPM evolution. Given the clinical context of profound hyponatremia with subsequent correction and a biphasic neurological course, we interpret this lesion as most compatible with osmotic demyelination (CPM). MRI with DWI/FLAIR would best differentiate CPM from ischemic stroke; however, it was unavailable at the time. The evolution from a normal early CT to a later pontine lesion is consistent with the dynamics of CPM imaging.

During hospitalization, the working diagnosis was decreased consciousness with suspected central pontine myelinolysis. Some comorbidities were also recorded, such as chronic constipation or functional ileus, neurogenic bladder with recurrent urinary tract infections, a history of bilateral hydronephrosis, secondary amenorrhea, nosocomial pneumonia, and moderate malnutrition. The differential diagnoses considered at that time were brainstem stroke, encephalitis, brain tumor, and drug intoxication from agents such as lithium or cytotoxic medications.

The clinical timeline indicates that risk factors—hyponatremia, hypokalemia, and malnutrition—were already present during the 2024 admission. In July 2025, the dominant complaints were abdominal pain, constipation, and amenorrhea, followed by progressive decline in consciousness with recurrent seizures. Serum sodium reached 104 mmol/L on July 29, 2025, prompting rapid correction. The initial head CT on August 1, 2025, appeared normal, which aligns with the possibility of a normal early-phase study in central pontine myelinolysis. Meanwhile, as shown in (Figure 1), the follow-up scan on August 22, 2025, showed a delayed pontine lesion compatible with CPM evolution.

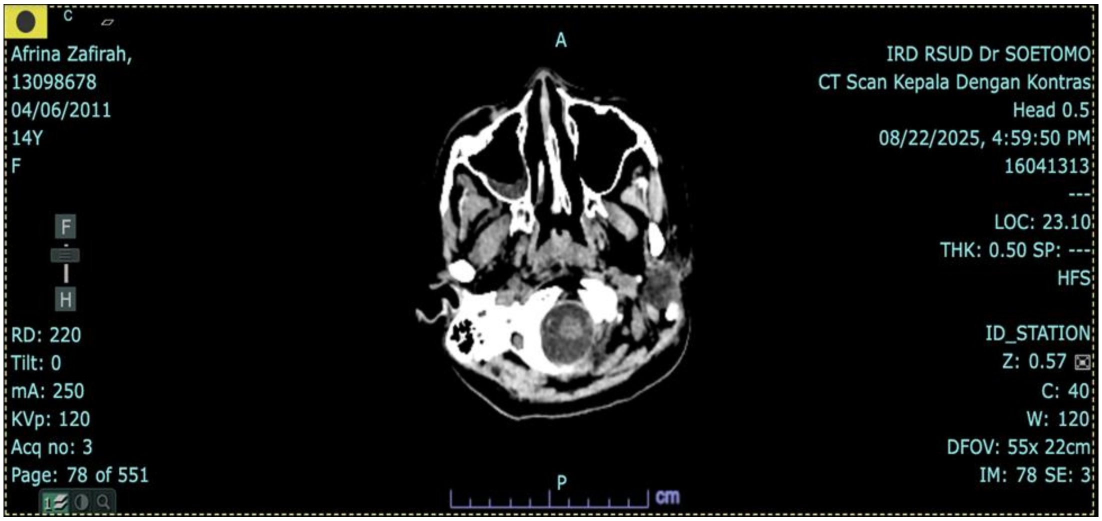

Figure 1- Result of a contrast head CT on 22 August 2025.

Results

Laboratory tests conducted on July 25, 2025, yielded the following results: hemoglobin 14.3 g/dL; platelets 353,000/µL; leukocytes 13,020/µL; sodium 122 mmol/L; potassium 4.5 mmol/L; and chloride 88 mmol/L. Repeat testing on July 29, 2025, showed progression to severe hyponatremia. Serum sodium decreased to 104 mmol/L, with potassium 3.1 mmol/L and chloride 65 mmol/L; calcium was 8.4 mg/dL, BUN 14 mg/dL, and creatinine 0.78 mg/dL. Due to progression to severe hyponatremia, correction was initiated using 50 mL of 15% NaCl diluted in D5NS (1,500 mL) over 24 hours. An oral lavement of polyethylene glycol (PEG) electrolyte solution (Niflec®) was administered at an impaction dose of 1 × ½. Management included potassium repletion together with controlled correction of hyponatremia, because normalizing potassium levels can inadvertently accelerate the rise in serum sodium.

Discussion

This case highlights three main points: (1) a high-risk milieu—malnutrition and hypokalemia—in severe hyponatremia, (2) a biphasic course with an initially normal CT followed by a delayed pontine lesion, and (3) the importance of slow, measured sodium correction with close monitoring to prevent overcorrection.

Diagnostic Criteria for Central Pontine Myelinolysis (CPM)

Central pontine myelinolysis (CPM) is a rare yet serious neurologic disorder characterized by selective injury to the central pons. It commonly develops due to rapid correction of chronic hyponatremia, particularly when sodium levels rise beyond the recommended safe limits. Patients with chronic hyponatremia (>48 hours) or severe sodium deficits (<120 mEq/L) are particularly vulnerable to CPM, especially when serum sodium increases by more than 8–10 mEq/L within 24 hours. Additional risk factors include alcoholism, malnutrition, liver disease, and hyperemesis gravidarum [1-2].

In this patient, the clinical course followed a biphasic pattern consistent with CPM. Initial neurological symptoms such as encephalopathy or seizures may transiently improve as serum sodium normalizes. Neurological deterioration may then occur after a latency of 3–5 days. Reported manifestations include dysarthria, dysphagia, spastic quadriparesis, and pseudobulbar palsy, with locked-in syndrome or coma in severe cases. Although not universal, this delayed deterioration is commonly observed in clinical practice and may help differentiate CPM from other neurological conditions [2-3].

On hospital day 1, the patient developed three generalized seizures followed by decreased consciousness. Communication was difficult during the postictal period. The initial CT performed on 1 August showed no acute abnormalities. During the subsequent weeks, the patient remained clinically stable without documented new neurological deficits. However, approximately three weeks later, repeat CT on 22 August demonstrated a new pontine hypodensity.

In this patient, the diagnosis of CPM was based on clinical context, severe hyponatremia, compatible neurological findings, and exclusion of alternative causes. In this patient, severe hyponatremia (nadir serum sodium 104 mmol/L) was treated with sodium correction over 24 hours. Subsequent neurological deterioration in this clinical context was consistent with osmotic demyelination.

Although the follow-up CT on August 22, 2025, was initially read as a chronic ischemic infarction, the pontine hypodensity appears more compatible with CPM evolution. Several clinicoradiologic factors support CPM over other conditions (brainstem stroke, encephalitis, or drug/toxic leukoencephalopathy). The patient had profound hyponatremia with subsequent correction and clinical worsening after a brief improvement, a biphasic course characteristic of CPM. Vascular pontine stroke was less likely in this adolescent with no documented major atherothrombotic risk factors, and the lesion did not clearly follow a single arterial territory. Encephalitis was unsupported by the available clinical context, as there was no reported febrile prodrome, meningismus, or CSF inflammatory profile. The timing of deterioration also aligned more with osmotic injury than viral or autoimmune inflammation. There was no history of exposure to agents classically associated with toxic leukoencephalopathy, and the topography (symmetric central pontine involvement with a normal early CT) was compatible with CPM imaging dynamics. Together, the trigger (hyponatremia correction), the biphasic evolution, and the lesion distribution provide a more coherent explanation for CPM; MRI with DWI/FLAIR, when obtainable, would further consolidate this distinction [2].

Magnetic resonance imaging (MRI) is the preferred radiologic tool to confirm the diagnosis. Diffusion-weighted imaging (DWI) can detect diffusion restriction in these pontine regions within 24 hours after symptom onset. Meanwhile, T2 and FLAIR sequences often show a "bat-wing" shaped hyperintense signal within the central pons. These findings, however, may be delayed up to two weeks. A negative initial MRI does not exclude the diagnosis; repeat imaging is recommended when clinical suspicion remains high [1-2].

Histopathologically, CPM shows concentrated demyelination with astrocytosis and infiltration by lymphocytes and macrophages. Extrapontine structures may occur in up to 10% of cases, involving the midbrain, thalamus, basal ganglia, or cerebellum, which may complicate the clinical picture. Because an MRI picture was not obtained in this patient, the diagnosis relied on the clinical-biochemical context and CT evolution. The overall pattern of risk factors, timing, and symmetric central pontine involvement supports CPM [2].

Aggressive Hyponatremia Correction Therapy Leads to Central Pontine Myelinolysis (CPM)

CPM is a form of osmotic demyelination that can occur when chronic hyponatremia is corrected too rapidly. This section outlines key principles in prevention, safe correction limits, and management considerations.

The fundamental principle in preventing CPM is the gradual correction of serum sodium. Current evidence suggests that sodium correction should generally not exceed 4 to 6 mmol/L in any 24 hours during treatment, though many guidelines allow for up to 6–8 or 8–12 mEq/L per 24 hours, depending on the duration and severity of hyponatremia and patient risk factors. If correction is faster than these recommendations, it risks precipitating demyelination. However, cases demonstrate that CPM can occasionally occur even within these limits, possibly influenced by comorbidities such as chronic alcoholism or malnutrition [2,4-6].

In the present case, management involved intravenous hypertonic saline (3% NaCl) for severe symptomatic hyponatremia. The infusion rate is carefully controlled to prevent overly rapid correction of serum sodium. Serum sodium was closely monitored during the correction period. This permits early detection of sodium overcorrection and allows timely interventions [2].

In this patient, management after the diagnosis of CPM was primarily supportive, as no disease-specific therapy has been proven effective in randomized controlled trials. Interventions such as corticosteroids, plasmapheresis, immunoglobulins, and thyrotropin-releasing hormone have been reported. However, evidence is insufficient to make them standard care. Neurological recovery varies widely, from full recovery to permanent disability, though prognosis appears more favorable with early recognition and supportive care [5].

Predisposing Factors for Central Pontine Myelinolysis (CPM)

Rapid correction of hyponatremia is still the strongest and most frequent factor associated with CPM. When the serum sodium level is less than 120 mEq/L for more than 48 hours and rises too quickly, it can lead to osmotic stress, causing brain cell injury and demyelination. Although most cases occur in hyponatremia, significant shifts in serum osmolality from other causes may also precipitate CPM [2,7].

In this patient, multiple pre-existing conditions associated with increased CPM risk were present prior to the 2025 admission. As previously documented in the Case Report (July 2024), the patient had malnutrition, hypokalemia, and kidney-related comorbidities that amplify CPM risk. During the current hospitalization, for example, additional conditions were recorded, including chronic constipation or functional ileus, neurogenic bladder with recurrent urinary tract infections, bilateral hydronephrosis, secondary amenorrhea, nosocomial pneumonia, and moderate malnutrition. Among these, malnutrition and liver diseases, including patients undergoing liver transplantation, have been frequently associated with CPM development. Malnutrition has been reported as one of the most common conditions associated with CPM in several recent case series, emphasizing the role of compromised metabolic and energy states in susceptibility to brain injury from osmotic shifts [8].

Overall, these findings suggest that CPM tends to occur in a vulnerable metabolic environment. Current understanding suggests that osmotic imbalance and possibly impaired energy provision in brain cells converge. The classical osmotic demyelination model is now supplemented by hypotheses that a pro-apoptotic environment and insufficient cellular energy contribute to vulnerability in certain individuals, explaining why not all patients with rapid sodium correction develop CPM and why other risk factors modulate susceptibility [9].

Caution in Management of Hyponatremia

As documented in the patient's admission in July 2024, the patient already had malnutrition, hypokalemia, and kidney-related comorbidities. In this patient, correction of sodium levels required cautious and controlled management to prevent neurological complications such as central pontine myelinolysis (CPM), also known as osmotic demyelination syndrome (ODS).

In this patient, the clinical course was consistent with the known pathophysiology of CPM, which involves rapid shifts in brain cell volume caused by overly rapid correction of sodium in hyponatremia. Brain cells adapt to chronic low sodium by decreasing their intracellular osmolytes to avoid swelling. If serum sodium is corrected too rapidly, these cells cannot reaccumulate osmolytes quickly enough, which may result in cellular dehydration and demyelination, particularly in the central pons region, leading to potentially severe neurological impairment. This mechanism likely explains the neurological deterioration observed in the present case. [10-14].

In this patient, sodium correction was guided by recommended safety limits, which advise that increases should not exceed 10 to 12 mmol/L within the first 24 hours and should be less than 18 mmol/L after 48 hours. More recent expert consensus suggests even slower correction rates—such as less than 8 mmol/L per 24 hours—for patients with chronic hyponatremia and cerebral edema to further reduce the risk of CPM. Rapid correction exceeding these rates has been associated with an increased risk of CPM, manifesting with symptoms like dysarthria, dysphagia, paraparesis, or quadriparesis [10-14]. These recommendations provide important context for interpreting the clinical course observed in the present case.

In this patient with severe symptomatic hyponatremia, initial management involved hypertonic saline to reduce cerebral edema and mitigate the risk of acute neurological deterioration. However, this should be carefully limited to the initial few hours until symptoms improve, then slowed to avoid surpassing recommended correction thresholds. The use of vasopressin receptor antagonists also requires caution and specialist experience, as they may cause unpredictable increases in sodium levels [10-14].

Frequent monitoring of serum sodium—often every 4 to 6 hours—is crucial during correction to tailor therapy carefully. Desmopressin may be administered to prevent overly rapid rises in sodium if overcorrection occurs. Individual risk factors, such as concomitant malnutrition, alcoholism, or liver disease, further increase vulnerability to CPM and necessitate even greater vigilance. These considerations are relevant to understanding the clinical course in the present case [10-14].

Conclusion

In this case, CPM developed following rapid correction of chronic hyponatremia, presenting with delayed neurological deterioration after initial improvement. Diagnosis involves a comprehensive assessment of the history of correction, predisposing factors, evolving brainstem signs, and radiological imaging findings. An early CT scan of the head is normal, with pontine hypodensity appearing only after several weeks. MRI is considered the imaging modality of choice, offering earlier findings, such as early diffusion restriction and T2/FLAIR hyperintensities. The clinical course and delayed radiologic findings in the present patient illustrate these diagnostic challenges. This case underscores the importance of cautious sodium correction to reduce the risk of CPM.

In this case, careful sodium control and close laboratory monitoring were essential to minimize the risk of excessive correction. Although factors such as alcoholism, malnutrition, liver disease, critical illness, hyperosmolar states, and even pregnancy can increase vulnerability, no definitive disease-specific therapy exists once CPM develops, making early recognition and supportive management essential to improving patient outcomes.

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