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Extrapontine Osmotic Demyelination with Pituitary Necrosis Following Severe Hyponatremia: A Rare Neuroendocrine Intersection

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1. Abstract

1.1. Background

Osmotic demyelination syndrome (ODS) is a rare but significant complication associated with the rapid correction of hyponatremia. Pituitary necrosis, commonly observed in cases of apoplexy, may manifest in susceptible metabolic conditions but is infrequently documented in association with ODS. Case: We present the case of a 55-year-old woman who exhibited symptoms of dehydration, fever, and altered sensorium. Laboratory assessments revealed severe hyponatremia and adrenal insufficiency. Magnetic resonance imaging (MRI) revealed extrapontine demyelination and pituitary necrosis. Conclusion: This case underscores the importance of cautious sodium correction and prompt endocrine evaluation in patients with altered sensorium and metabolic stress. Neuroendocrine overlap necessitates a comprehensive approach involving integrated imaging and hormonal assessment.

2. Introduction

Osmotic demyelination syndrome (ODS), which encompasses central pontine myelinolysis (CPM) and extrapontine myelinolysis (EPM), is a severe neurological complication resulting from the rapid correction of chronic hyponatremia [1]. Initially identified in patients with chronic alcoholism and malnutrition, this condition has been observed in various clinical contexts involving electrolyte imbalance [2]. While central pontine involvement is the classic presentation, extrapontine manifestations affecting the basal ganglia, thalamus, and cerebral cortex are increasingly recognized and may occur independently or alongside pontine lesions [3-4]. The pathophysiology of ODS involves differential cellular responses to osmotic stress. In chronic hyponatremia, brain cells adapt by losing organic osmolytes to prevent cellular swelling. However, rapid sodium correction can induce cellular dehydration and subsequent demyelination, particularly affecting oligodendrocytes, which are highly susceptible to osmotic stress [5]. The vulnerability of specific brain regions appears to correlate with oligodendrocyte density and metabolic activity. Pituitary necrosis, which is most commonly encountered in pituitary apoplexy, represents a distinct yet potentially related pathophysiological process. Apoplexy typically results from hemorrhage or infarction within a pituitary adenoma; however, non-adenomatous pituitary necrosis can occur under severe physiological stress, including shock, pregnancy, and significant electrolyte disturbances [6]. The co-occurrence of ODS and pituitary necrosis represents an extremely rare clinical scenario, suggesting shared pathophysiological mechanisms involving osmotic stress and metabolic vulnerability. We present a unique case demonstrating the concurrent development of extrapontine osmotic demyelination and pituitary necrosis in a patient with severe hyponatremia and secondary adrenal insufficiency, highlighting the complex neuroendocrine interactions that can occur in critically ill patients with severe electrolyte imbalance.

3. Case Presentation

A 55-year-old woman presented with high-grade fever, vomiting, and progressive dehydration over a three-day period. She exhibited drowsiness and disorientation and reported diffuse headache. The patient had no history of endocrine or neurological disorders. On examination she had fever (38.8°C), hypotension (90/60), tachycardia (110 bpm). There were signs of dehydration (dry mucous membranes, decreased skin turgor). Detailed neurological examination was done and revealed a GCS of 11/15 (E3V3M5), Cranial nerve examination was normal. Motor, sensory, and coordination testing was also normal with normal reflexes. There was hyperpigmentation of buccal mucosa, postural hypotension, generalised weakness. Initial laboratory investigations indicated a serum sodium level of 114 mmol/L, serum os-

molality of 260 mosm/kg, serum cortisol of 3.2 µg/dL, elevated C-reactive protein, and a Glasgow Coma Scale score of 11/15. No focal neurological deficits were observed during the examination. The patient received intravenous 5% dextrose, and a subsequent blood examination revealed a serum sodium level of 143 mmol/L after 36 h. Her neurological drowsiness persisted, prompting plain and contrast-enhanced MRI, followed by MR spectroscopy. Imaging revealed non-enhancing T2/FLAIR hyperintensities in the midbrain bilaterally and both thalami, consistent with extrapontine myelinolysis (Figure 1 A-C). The sella was of normal size, with an enlarged pituitary exhibiting heterogeneous signal intensity and hypodense, non-enhancing necrotic areas (Figure 1 D, E). The sella and stalk were normal. The MR venogram was normal (Figure 1F). No evidence of hemorrhage or restricted diffusion was observed on the diffusion images. MR spectroscopy showed presence of inverted lactate peak with mild increased choline with mildly reduced n-acetyl aspartate levels (Figure 2). The spectroscopic findings demonstrated several key features characteristic of osmotic demyelination syndrome. The presence of an inverted lactate peak at 1.3 ppm indicated anaerobic metabolism and cellular dysfunction within the affected brain regions, reflecting the metabolic stress imposed by rapid osmotic changes on neural tissue. The mild elevation in choline levels suggested membrane breakdown and increased cell turnover, consistent with the demyelinating process affecting oligodendrocytes. Her blood tests showed serum Cortisol level of: 3.2 µg/dL (low) - indicating adrenal insufficiency with low serum ACTH: 8.5 pg/mL again confirming central origin. The thyroid functions were normal with normal however Free T4 and T3 were at lower normal limits with similar levels of Growth hormone/IGF-1 suggesting early GH deficiency. Serum Prolactin was mildly elevated at 45 ng/ml.

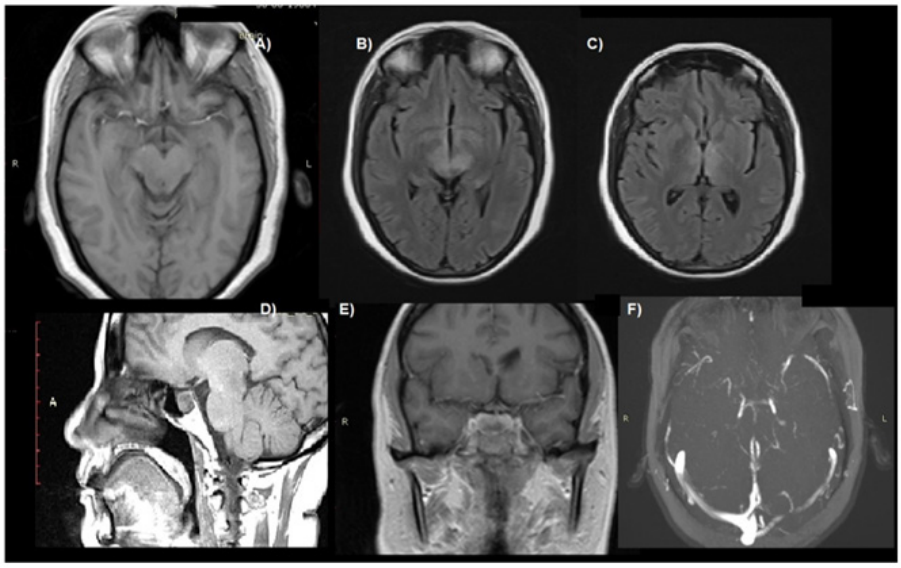

Figure 1: A-F: A: Plain T1WSE axial image showing hypointense signal of midbrain on right side. B-C) Flair axial images showing bilateral midbrain and thalamic edema D) T1WSE saggital image showing necrosis in a prominent size pituitary gland E) Post contrast image with central non enhancing pituitary necrosis F) Plain MR venogram of brain showing normal deep veins and the sinuses.

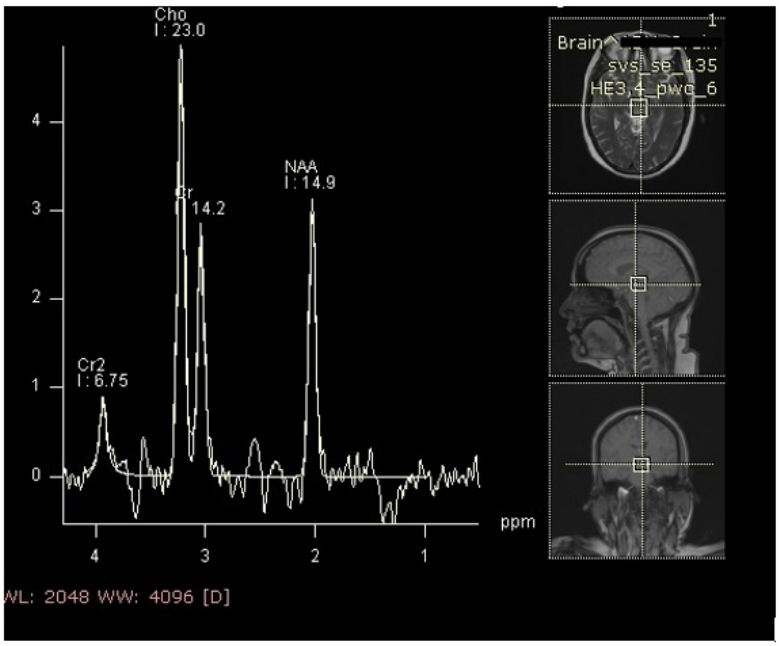

Figure 2: MR spectroscopy with long spin echo time of 135 showing inverted lactate peak at 1.3 pmm with mild increased choline with mildly reduced N-acetyl aspartate peak.

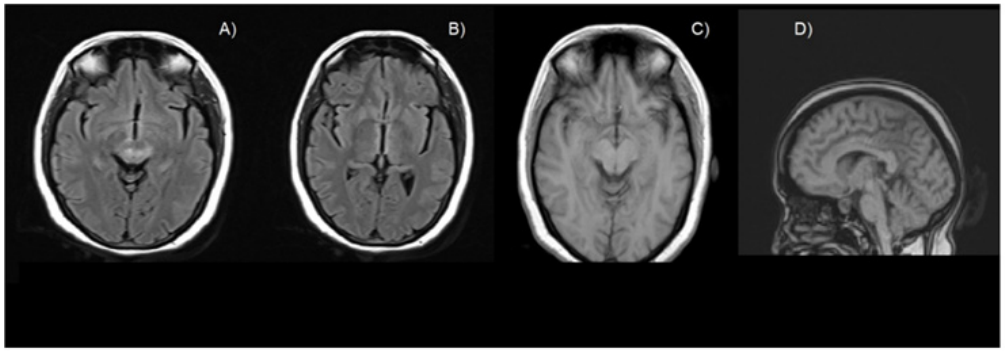

Figure 3: A-B: Plain MRI FLAIR axial images show minimal residual edema in mid brain with normal signal of the thalami, C) Normal plain T1W axial image. D) T1WSE saggital image showing necrosis in prominent size pituitary gland.

4. Differential Diagnosis

The clinical presentation and imaging findings in this case necessitated consideration of several differential diagnoses, given the constellation of altered mental status, electrolyte abnormalities, and characteristic brain lesions. Viral Encephalitis was considered given the patient's fever, altered sensorium, and bilateral thalamic involvement. However, the symmetric nature of the lesions, absence of hemorrhagic components, and the temporal relationship with rapid sodium correction made viral encephalitis less likely. Additionally, the concurrent pituitary involvement and the specific MR spectroscopy findings were more consistent with osmotic demyelination.

Acute Disseminated Encephalomyelitis (ADEM) could present with similar bilateral brain lesions and altered consciousness. However, ADEM typically follows viral infections or vaccinations and demonstrates more widespread, asymmetric white matter involvement with contrast enhancement, which was absent in our case.

Metabolic Encephalopathy from various causes including hepatic, uremic, or diabetic etiologies was considered. However, the focal nature of the brain lesions and the normal liver and kidney function tests made systemic metabolic causes less likely. Central Nervous System Lymphoma can present with bilateral thalamic lesions and altered consciousness. However, the lack of contrast enhancement, the absence of mass effect, and the clinical context of rapid sodium correction strongly favored osmotic demyelination over neoplastic processes. Wernicke Encephalopathy shares some similarities with osmotic demyelination, particularly bilateral thalamic involvement. However, the absence of mammillary body involvement, oculomotor abnormalities, and ataxia, combined with the temporal relationship to sodium correction, made this diagnosis unlikely. Pituitary Apoplexy was considered for the sellar abnormalities but typically presents with acute headache, visual field defects, and ophthalmoplegia. The gradual onset and absence of visual symptoms, combined with the concurrent brain lesions, suggested a different pathophysiological process. The key discriminating features that established the diagnosis of osmotic demyelination syndrome with concurrent pituitary necrosis included: the temporal relationship between rapid sodium correction and symptom onset, the characteristic distribution of brain lesions in osmotically vulnerable regions, the specific MR spectroscopy pattern showing lactate elevation and membrane breakdown markers, and the concurrent development of hypopituitarism in the setting of severe electrolyte disturbance. The patient was administered controlled dextrose saline infusion with careful monitoring, intravenous hydrocortisone for suspected adrenal crisis, comprehensive supportive care, and continuous neurological monitoring. The patient's sensorium gradually improved within 72 h. Serum sodium was cautiously corrected to 130 mmol/L over four days, adhering to the recommended correction rates. A follow-up MRI obtained three weeks later showed partial resolution of the demyelinating changes (Figure 3). Comprehensive hormonal evaluation confirmed secondary adrenal insufficiency and hypopituitarism, necessitating long-term hormone-replacement therapy.

5. Discussion

This case exemplifies a rare yet clinically significant confluence of osmotic demyelination syndrome and pituitary necrosis, manifesting in the context of severe hyponatremia and metabolic stress. The simultaneous occurrence of these pathologies offers valuable insights into the intricate pathophysiological mechanisms underlying severe electrolyte disturbances and their neurological effects. The emergence of extrapontine osmotic demyelination in our patient likely resulted from the rapid correction of chronic severe hyponatremia in the presence of adrenal insufficiency [1]. During chronic hyponatremia, brain cells engage in adaptive mechanisms, including the loss of intracellular organic osmolytes such as taurine, glycine, and myo-inositol, to avert cellular swelling and maintain a normal intracranial pressure [2]. However, when serum sodium levels are corrected too swiftly, depleted osmolytes cannot be rapidly replenished, leading to cellular dehydration, subsequent oligodendrocyte dysfunction, and demyelination. The preferential involvement of extrapontine structures, particularly the basal ganglia and thalamus, in our case is consistent with previous reports suggesting that these regions may be more susceptible to osmotic stress owing to their high metabolic activity and dense oligodendrocyte populations [3]. The absence of pontine involvement, although atypical, has been documented in approximately 10-15% of ODS cases and may reflect individual variations in regional brain vulnerability or the specific metabolic milieu present during correction [4]. The simultaneous occurrence of pituitary necrosis in our patient

constitutes a notably rare manifestation that may share underlying pathophysiological mechanisms with those of osmotic demyelination. The pituitary gland, particularly its anterior lobe, is characterized by high vascularity and metabolic activity, rendering it vulnerable to ischemic injury during periods of significant physiological stress [5]. Several factors may have contributed to the development of pituitary necrosis in this patient. First, severe dehydration and potential hemodynamic instability may compromise pituitary perfusion. Second, osmotic stress associated with severe hyponatremia may directly impact pituitary cell function. Third, the presence of underlying adrenal insufficiency suggests a pre-existing dysfunction of the hypothalamic-pituitary-adrenal axis, which may have predisposed the patient to pituitary injury. The concurrent occurrence of extrapontine demyelination and pituitary necrosis in our patient highlights several critical considerations. First, patients presenting with severe hyponatremia and altered mental status necessitate comprehensive neuroimaging that extends beyond the assessment of pontine involvement to include the evaluation of extrapontine structures and the sella turcica [6]. Neurological symptoms in such patients may arise from multiple concurrent pathologies rather than a single disease process. Second, adrenal insufficiency in patients with severe hyponatremia may represent both a cause and consequence of the underlying pathophysiology. Adrenal insufficiency can contribute to hyponatremia through impaired free water clearance and aldosterone deficiency, whereas severe electrolyte disturbances and osmotic stress may precipitate pituitary dysfunction, leading to secondary adrenal insufficiency. This creates a potentially self-perpetuating cycle that requires early recognition and intervention to break. The management of patients with severe hyponatremia in the setting of suspected or confirmed adrenal insufficiency requires careful attention to multiple physiological parameters [2]. Current guidelines recommend limiting sodium correction to 6-8 mEq/L in the first 24 hours and 12-14 mEq/L in the first 48 h to minimize the risk of osmotic demyelination. However, patients with concurrent endocrine dysfunction may require modified approaches, including the earlier initiation of corticosteroid replacement and more intensive monitoring of both electrolyte status and hormonal parameters. The use of desmopressin (DDAVP) to slow or reverse overly rapid sodium correction has been proposed as a potential rescue strategy, although its efficacy in preventing ODS is controversial. In our case, the controlled correction approach using hypertonic saline with frequent monitoring appeared to be effective in preventing further neurological deterioration while allowing for gradual recovery. The prognosis for patients with osmotic demyelination syndrome varies considerably depending on the extent and location of the demyelinating lesions, the rapidity of correction, and the underlying comorbidities [1]. Extrapontine involvement generally carries a somewhat better prognosis than central pontine myelinolysis, although significant neurological sequelae can still occur. The concurrent pituitary necrosis in our patient necessitated long-term hormone replacement therapy, highlighting the importance of comprehensive endocrine follow-up. This case highlights the need for further research into the mechanisms underlying the vulnerability of different brain regions and endocrine structures to increased osmotic stress. Advanced neuroimaging techniques like magnetic resonance spectroscopy are helpful in making the diagnosis and also in differentiating ODS from other causes which have similar findings on routine MRI. Spectroscopic findings provided valuable metabolic confirmation of the structural abnormalities observed on conventional MRI sequences and the combination of lactate elevation, choline increase, and NAA reduction created a characteristic metabolic signature that support the diagnosis of osmotic demyelination syndrome. Furthermore, MR spectroscopy may serve as a useful tool for monitoring treatment response and predicting prognosis, as normalization of these metabolic markers often correlates with clinical improvement and provide additional insights into the pathophysiology of osmotic demyelination. The findings of ODS on MRS show increase in choline-containing compounds due to myelin breakdown and cellular membrane disruption. The mildly reduced N-acetyl aspartate (NAA) levels, a marker of neuronal and axonal integrity, indicates neuronal dysfunction secondary to the osmotic injury, and are potential biomarkers for the early detection of osmotic demyelination or pituitary dysfunction which could improve clinical outcomes through earlier intervention.

6. Conclusion

This case demonstrates the rare but significant co-occurrence of extrapontine osmotic demyelination and pituitary necrosis in a patient with severe hyponatremia and adrenal insufficiency. Clinicians should maintain a high clinical suspicion for neuroendocrine complications in patients presenting with severe electrolyte disturbances and altered mental status. Comprehensive neuroimaging extending beyond pontine assessment and early hormonal evaluation are essential components of the management. The implementation of careful sodium correction protocols, early corticosteroid replacement when indicated, and multidisciplinary care coordination may prevent irreversible neurological and endocrine complications. This case emphasizes the complex pathophysiological interactions between severe metabolic disturbances and their effects on both neural and endocrine tissues, warranting an integrated approach to diagnosis and management of these patients.

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