Sequential Evolution of Extrapontine and Pontine Myelinolysis: Role of Hypokalemia—A Case Report

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Abstract

Osmotic demyelination syndrome due to rapid correction of hyponatremia may present with myelinolysis at pontine and/or extrapontine locations. Of late, the role of hypokalemia in increasing the risk for demyelination is being recognised. A 57-year-old man underwent surgery for cervical myelopathy. Postoperatively, he developed hyponatremia and hypokalemia and went on to develop seizures followed by "locked in state". Magnetic resonance imaging (MRI) after 8 days revealed extensive extrapontine myelinolysis. The patient recovered well over 8 weeks. Sequential development of MRI lesions and their resolution has been discussed.

Keywords
- osmotic demyelination
- hyponatremia
- hypokalemia

Introduction

Central pontine myelinolysis occurs due to rapid correction of hyponatremia. Interestingly, osmotic demyelination has been described with sodium levels within normal limits and hypokalemia as well.[1,2] Osmotic demyelination at extrapontine sites with or without pontine involvement has also been described.[3] There are few studies with sequential magnetic resonance imaging (MRI) to understand the evolution of the syndrome.

A patient with hyponatremia and hypokalemia is presented, who as a result of osmotic demyelination had sequential appearance of extrapontine lesions, followed by resolution of the same.

Case Report

A 57-year-old man, a teetotaller, presented with spastic quadriparesis of 8 months, duration. He underwent discoidectomy and bone grafting for cervical disc prolapse. The patient was discharged with power of 4+/5 in all four limbs. Ten days post discharge, the patient sustained a fall and was readmitted with quadriplegia. Repeat MRI showed graft displacement with residual/recurrent compressive myelopathy. A redo bone grafting and plating was done. Postoperatively, his quadriparesis improved to 3/5; however, he required ventilation for spells of bradypnoea.

Postoperatively, electrolyte panel revealed hyponatremia (sodium 102 mmol/L) and hypokalemia (potassium 1.9 mmol/L). Hyponatremia was corrected with 300 mL of 3% sodium chloride infusion over 6 hours and 40 meq of potassium was supplemented intravenously. After 15 hours, the sodium and potassium levels were 115 and 2.1 mmol/L, respectively. By the third postoperative day, electrolyte levels had normalized. He was weaned off ventilator on the fifth postoperative day. However, he became quadriplegic with intact extraocular movements suggestive of locked in syndrome, developed persistant lip smacking movement followed by a generalized tonic–clonic seizure.

MRI brain done on the eighth postoperative day for a clinical diagnosis of locked-in syndrome revealed no lesions in the pons but extensive lesions on T2 weighted and flair images in bilateral caudate nuclei, putamina, thalami, coronae radiate, periventricular white matter, and hippocampi (►**Fig. 1**). A diagnosis of extrapontine myelinolysis was made. The patient was treated symptomatically with mechanical ventilation and anti epileptic drugs. The patient recovered completely over

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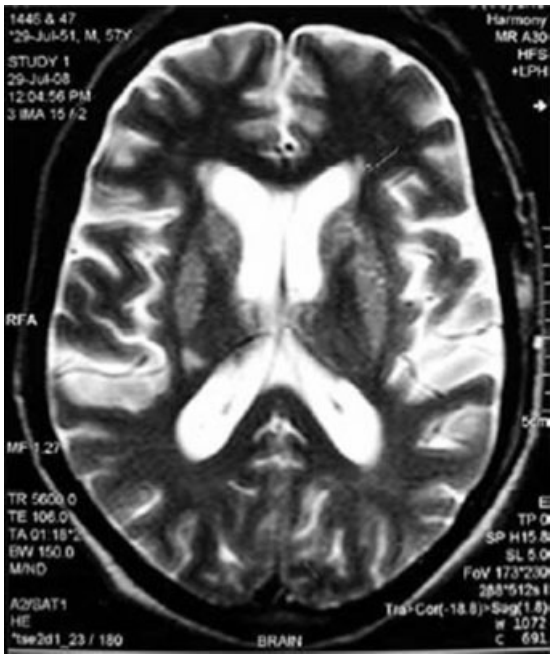

Fig. 1 Magnetic resonance imaging (flair images) on day 8 showed extensive extrapontine myelinolysis.

8 weeks. MRI after 2 months, interestingly, revealed new lesions in the pons, with partial resolution of the previous lesions in the extrapontine locations. During follow-up after 6 months, the patient showed improvement in quadriparesis to 4+/5 and repeat MRI showed complete resolution of myelinosis (►**Figs. 2** and **3**).

Discussion

The theories explaining the etiopathogenesis of osmotic demyelination have undergone frequent revision. Initially described in alcoholic cirrhotics, it was realized later that too rapid correction of hyponatremia possibly leads to osmotic shrinkage of axons, inducing demyelination. Coexisting hypokalemia is believed to increase the risk of myelinolysis in patients with rapidly corrected hyponatremia.[2] The exact

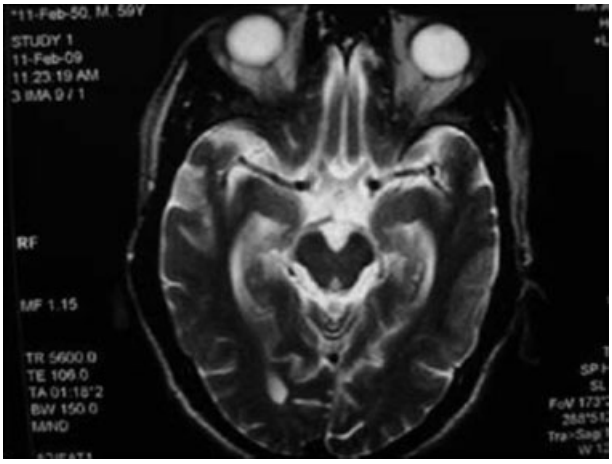

Fig. 2 Magnetic resonance imaging after 6 months showed

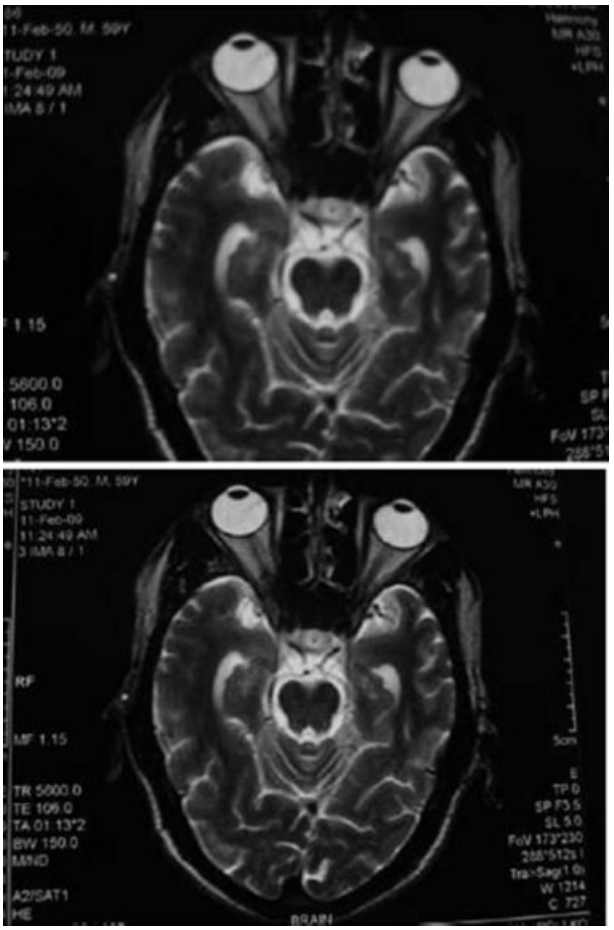

Fig. 3 Magnetic resonance imaging after 6 months showed resolution of all lesions.

mechanism of this interaction is not understood. Our patient highlights the compounding role played by persistent hypokalemia in precipitating myelinolysis. MRI on the eighth postoperative day had revealed extensive osmotic demyelination in the brain, sparing the pons. After 2 months, when the patient had completely recovered, MRI paradoxically revealed evidence of central pontine myelinolysis too. There are few reports in literature of such sequential development of lesions.[4] Delayed clinical manifestations, especially with extrapyramidal syndromes, from 10 days 5 months after osmotic demyelination at extrapontine locations have been reported.[5] Lesions on MRI are known to present up to 14 days after osmotic dysregualtion. However, sequential new lesions on MRI, in clinically improving patients have not been described.

The lack of correlation between the clinical course and MRI findings is well known. Our patient with a locked in syndrome had no demonstrable lesion in the pons. The prognosis, considered previously to be grave, has improved with better understanding and management of the disorder.[6] Despite the extensive lesions, and a clinical presentation initially confused by the cervical myelopathy resulting in a delay in diagnosis, our patient made complete recovery.

This case underlines the fact that there are factors other than the rapid correction of hyponatremia, such as persistent hypokalemia that possibly contributes to the

course of this syndrome. There is a need for more sequential MRI studies in patients with osmotic demyelination in order to shed light on this queer syndrome. With appropriate treatment, the prognosis is favorable.

Conflict of Interest
The authors have nothing to declare.

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