Osmotic demyelination syndrome – all is not lost!

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Introduction

Osmotic demyelination syndrome (ODS) is characterised by partial destruction of myelin sheath at the pons.

The advised rate of correcting chronic hyponatremia in high risk patients is 4-6 mmol/l in any 24 hour period [1]. Risk factors that increase the susceptibility of ODS even at slower rates of correction are alcoholism, malnutrition, liver disease and hypokalaemia [2].

Case reports and case series have often shown a high mortality [3], however the prognosis is variable. Some patients do make a full neurological recovery.

Here, we present a case of osmotic demyelination syndrome where multidisciplinary approach, practitioner vigilance and timely intervention were key to the patient making full neurological recovery.

Description

A 43-year-old female with known alcoholic liver disease was admitted after a history of fall from stairs and coffee ground vomit. On presentation she was alert with a GCS of 15, maintaining her airway with oxygen saturation of 99% on room air with blood pressure of 124/64 mm Hg and HR of 102/min. She underwent Upper GI endoscopy for banding and was admitted with a diagnosis of alcoholic liver disease with acute on chronic upper GI bleed.

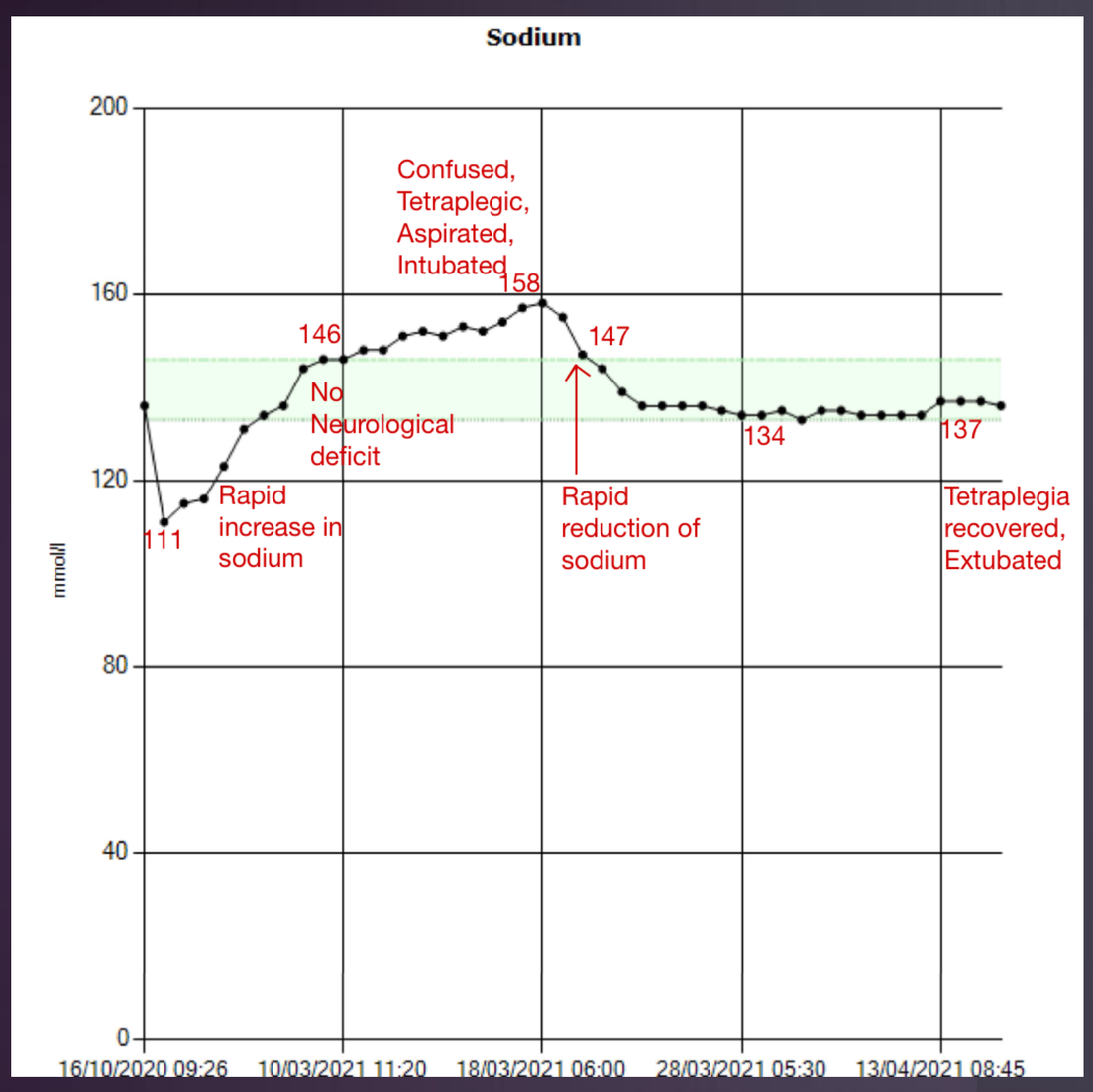
During her admission, the patient was found to be intermittently confused, could move her eyes but had weakness in her arms and legs with a muscle power score of 0/5 and was found to be in type 1 respiratory failure.

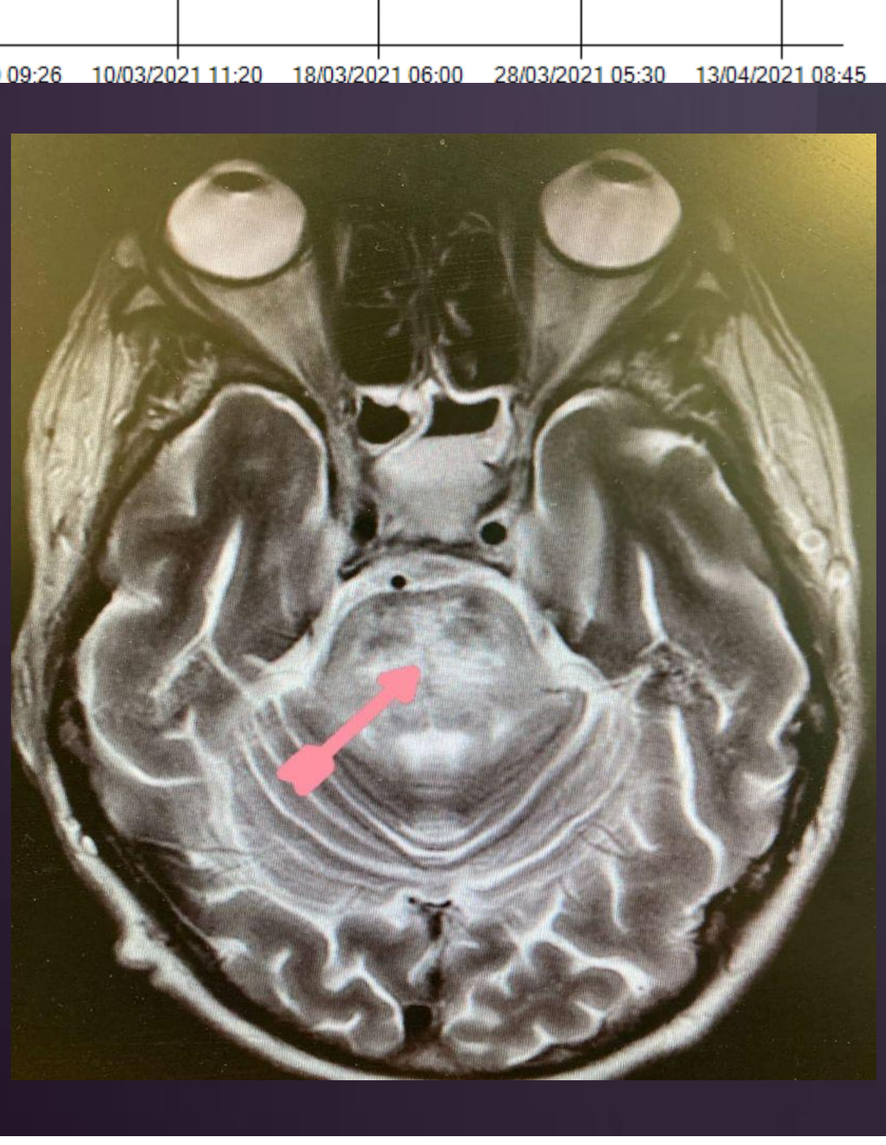
She was admitted to critical care, intubated and ventilated for a possible aspiration pneumonia. Intravenous antibiotics were escalated, and hepatic encephalopathy was ruled out.

Lumbar puncture revealed no pathogens. EEG was suggestive of an alpha coma pattern; CT was not suggestive of any acute pathology. MRI showed altered signal intensity of the pons suggestive of osmotic demyelination (central pontine myelinolysis).

Her serum sodium was lowered and supportive therapy was continued. Over the next few days the patient had a gradual improvement in mentation and oral buccal – lingual movements and could manage her own secretions. Her tetraplegia recovered with a muscle power score of 2/5 in all 4 limbs.

Patient was extubated a discharged to neuro rehabilitation ward after a safe swallow assessment. She was followed up for the next 3 months and is now able to walk with a frame.





Discussion

Neurological symptoms of ODS typically occur 6-7 days after correction of hyponatremia. The optimal approach to treat is unclear, we tried relowering serum sodium with ongoing supportive therapy. Relowering therapy should be initiated as quickly as possible after the onset of neurologic symptoms. It can reverse the breakdown of the blood-brain barrier that occurs with overly rapid correction and can prevent the infiltration of microglia that is a feature of osmotic demyelination [4].

Data in humans is limited to case reports that suggest if relowering is initiated within a few hours after the onset of neurologic symptoms, it is associated with a better outcome. [5,6].

References

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