Osmotic demyelinisation syndrome in a patient with severe hyponatremia; the role of hypokalaemia

Case 11986

A 70-year-old woman, with a clinical history of high blood pressure and dyslipidaemia, admitted for severe hyponatraemia secondary to chronic vomiting associated to hypokalaemia. Initially the patient showed an improvement of her state of stupor, but during the correction of the hyponatraemia, the patient presented a neurological deterioration with obnubilation.

Imaging Findings:

A cerebral CT was performed and was normal.
A cerebral MRI followed and showed numerous lesions in hypersignal on T2WI and FLAIR, involving the pons, the two thalami, the external capsules, the corona radiata and the semioval centres (Fig. 1)
Diffusion sequence showed restriction of the diffusion in the pons and the corona radiata (Fig. 2).
These lesions do not enhance with contrast.

Discussion:

Osmotic demyelination syndrome (ODS) is a clinicopathologic entity characterized by oedema and demyelination in the pons and extrapontine areas [1].
Metabolic factors would play a capital role [1].
It is generally associated with rapid correction of hyponatraemia (>12mmol/day), but its exact pathogenesis is not fully established. Recently, it has been demonstrated that hyponatraemia causes downregulation of a neutral amino acid transporter that impairs cellular reuptake of amino acids, making them more susceptible to injury as hyponatraemia is corrected [2].
In a report, 89% of cases of ODS had associated hypokalaemia at presentation, which, as in the case of our patient, had not normalized before the rapid correction of hyponatraemia [3].
Diminished endothelial cell membrane concentration of NaK-ATPase in hypokalaemia may induce cell injury by osmotic stress associated with the rapid elevation of the serum sodium concentration [3, 4].
Classically, ODS presents with mental status changes and rapidly progressive quadriplegia associated with dysphagia, dysarthria and other pseudobulbar symptoms. The clinical presentation can also include ataxia and
Parkinsonian symptoms [5].
MRI of the brain is the method of choice for diagnosis [5].
Typically, the lesions are hypointense on T1-WI and hyperintense on T2-WI and FLAIR [5, 6]. These lesions are located in the central pons, with a ruffle of intact white matter surrounding the lesion; extrapontic myelolysis lesions affect the thalamus, the lentiform nucleus, the head of caudate nucleus, the internal and external capsules, the hemispherical, cerebellar and medullary white matter and the lateral geniculate bodies. These lesions do not enhance with contrast [5, 6].
In most cases of ODS, increased rather than decreased ADC is observed, although decreased ADC (evocative of cytotoxic oedema) has also been described in few cases [7].
Typically, radiological findings do not improve over time, even if the patient has made a complete or nearly complete clinical recovery [8].
To prevent the ODS, the optimal rate of sodium correction is <10 to 12 mEq/L per day and <18 mEq/L in 48 hours [5].
The patient who presents with both risk factors for development of ODS (RFODS) and symptoms of hyponatraemia necessitating an immediate rise in serum sodium (seizures or obtundation), is difficult to treat. Generally a patient with a serum sodium of 105 mEq/L and seizing will usually respond with an elevation of the serum sodium on the order of 5-7 mEq/L) [9].
In patients with concomitant RFODS (alcoholism, malnutrition or hypokalaemia), the correction in serum sodium must be <8 mEq/L per day [5].
Repleting potassium has effects on serum sodium. The potassium ingested will increase serum sodium concentration through the action of the Na-K-ATPase. So, correction of hypokalaemia should be initiated with prudence in patients at risk for ODS, especially those with malnutrition and alcoholism [5].

**Differential Diagnosis List:** Osmotic demyelination syndrome, Hypoxic encephalopathy, Multiple sclerosis

**Final Diagnosis:** Osmotic demyelination syndrome

**References:**
Figure 1

Description: Cerebral MRI: Axial Flair:
lesions in hypersignal involving the two thalami and the external capsules. Origin: Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco
Description: Cerebral MRI: Axial Flair: lesions in hypersignal involving the corona radiata. Origin: Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco
Description: Cerebral MRI: Axial Flair: lesions in hypersignal involving the semi-oval centres. Origin: Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco
Description: Cerebral MRI: Axial Flair: lesions in hypersignal involving the pons. Origin: Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco
Description: Cerebral MRI: Axial T2: lesions in hypersignal T2 involving the pons. Origin: Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco
Description: Cerebral MRI: Axial T2: lesions in hypersignal T2 involving the cerebral peduncles.
Origin: Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco
Description: Cerebral MRI: Axial T2: lesions in hypersignal T2 involving the corona radiate. Origin: Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco
**Description:** Cerebral MRI: Axial T2: lesions in hypersignal T2 involving the semi-oval centres.

**Origin:**
Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco
Figure 2

Description: DWI: restriction of diffusion in the corona radiata: hypersignal on DWI, with corresponding areas of decreased ADC (in blue) on the ADC map. Origin: Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco

Description: DWI: restriction of diffusion in the pons: hypersignal on DWI, with corresponding areas of decreased ADC (in blue) on the ADC map. Origin: Tizniti S, Department of Radiology, CHU hassan II, Fes, Morocco