Central pontine myelinolysis

Reuben Grech, Leo Galvin, Paul Brennan, John Thornton

Department of Neuroradiology, Beaumont Hospital, Dublin, Ireland

Correspondence to
Dr Reuben Grech,
reubengrech@yahoo.com

DESCRIPTION

Case report

A 56-year-old gentleman was referred to the A&E department following an episode of loss of consciousness. His medical and surgical history was unremarkable except for a background history of alcohol dependency. Serological tests revealed a serum sodium concentration of 118 mEq/l. The patient developed acute pseudobulbar palsy less than 24 h after admission and an urgent MRI of the brain was requested.

DISCUSSION

Central pontine myelinolysis is an acute non-inflammatory demyelinating disorder first described by Adams *et al* in 1958. It is a rare condition that predominantly affects the pons but may involve extrapontine structures including the midbrain, thalamus and cerebellum. The condition was originally described in patients with severe hyponatraemia, particularly in the setting of alcoholism and malnutrition. It has since been linked to diabetes mellitus, AIDS and hepatic failure.[1] Central pontine myelinolysis is precipitated by the rapid correction of chronic severe hyponatraemia (defined as serum sodium <120 mEq/l). The pathophysiology is still not well understood.

Presentation can be catastrophic with pseudobulbar palsy, spastic tetraparesis and the 'locked-in' syndrome.[2] MRI is the imaging modality of choice (figures 1 and 2). Diffusion-weighted imaging is particularly useful and becomes positive within few hours of symptom onset.[3] The pontine abnormality characteristically spares the ventrolateral pons and corticospinal tracts.

The overall prognosis is poor. Prevention is crucial and requires cautious correction of chronic hyponatraemia. Most experts would advocate correction of serum sodium concentration at no more than 10 mmol/l/day. Treatment is supportive and aims to prevent complications such as deep vein thrombosis and aspiration pneumonia.

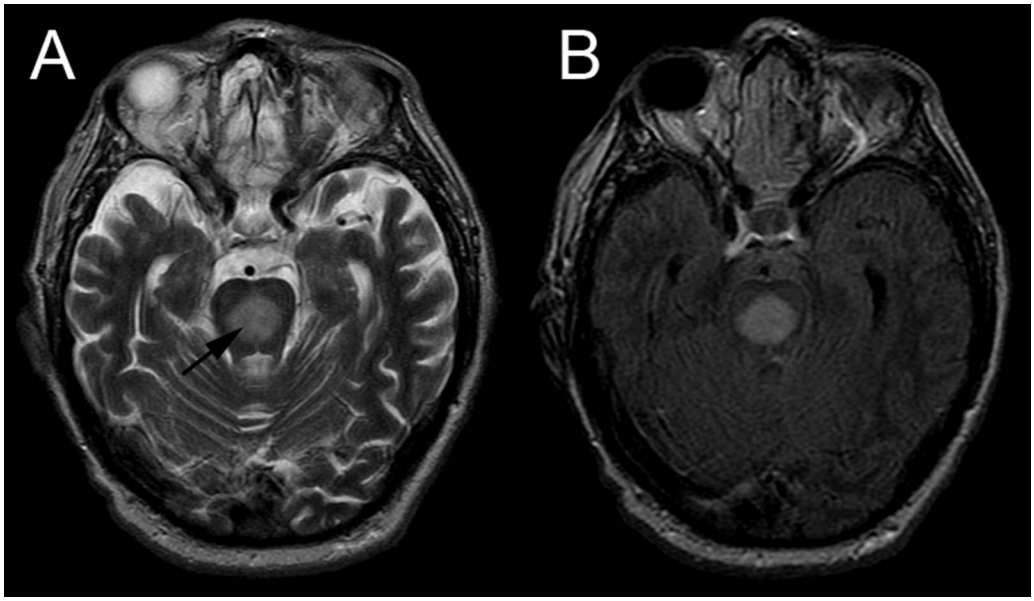

Figure 1 T2 (A) and fluid-attenuated inversion recovery (B) images demonstrate a central hyperintense lesion within the pons.

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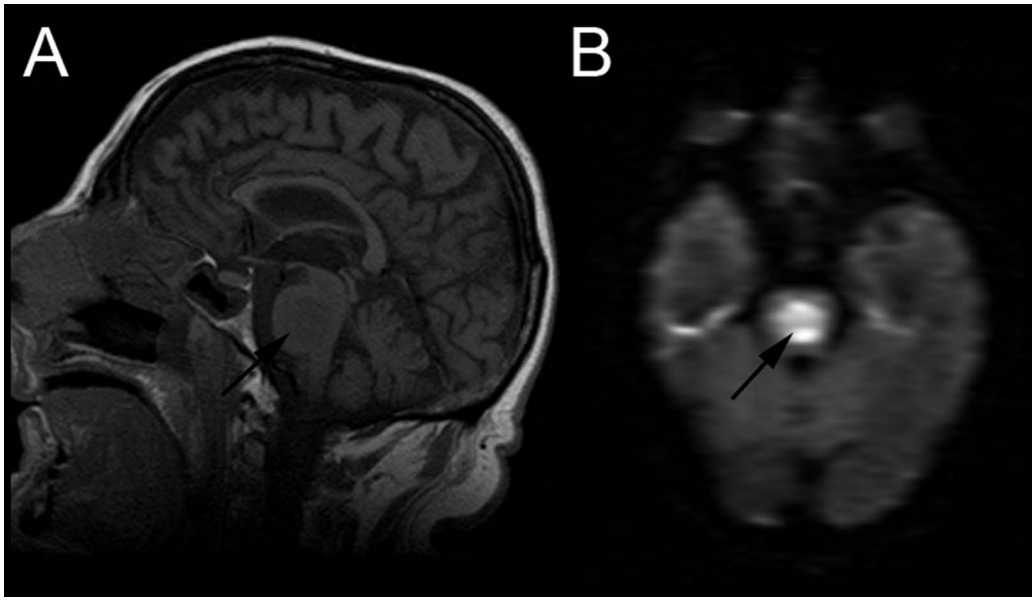

Figure 2 The lesion is hypointense on T1 (A) and hyperintense on diffusion-weighted imaging (B) sequences. Diffusion restriction is seen within 24 h of symptom onset. Sparing of the ventrolateral longitudinal fibres and corticospinal tracts is characteristic.

Learning points

- Central pontine myelinolysis is an acute non-inflammatory demyelinating disorder.
- It is precipitated by the rapid correction of severe chronic hyponatraemia.
- MRI is the imaging modality of choice with diffusion-weighted imaging becoming positive within a few hours of symptom onset.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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