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BENEATH THE SURFACE OF RENAL FAILURE: CENTRAL PONTINE MYELINOLYSIS REVEALED BY UNEXPLAINED SEIZURES IN A HEMODIALYSIS PATIENT

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ABSTRACT

Osmotic demyelination syndrome, encompassing central pontine and extrapontine myelinolysis, is a rare but potentially devastating neurological complication increasingly recognised in patients with end-stage renal disease undergoing haemodialysis, in whom abrupt osmotic shifts during dialysis sessions can trigger the disease even without overt correction of hyponatraemia. We report the case of a 32-year-old woman with end-stage renal disease on maintenance haemodialysis, with no other notable medical history, who was admitted for refractory convulsive status epilepticus. Initial brain CT was unremarkable. Brain MRI performed as part of the aetiological work-up demonstrated a centropontine and mesencephalic lesion, hypointense on T1-weighted images, hyperintense on T2 and FLAIR sequences, with restricted diffusion and no contrast enhancement, consistent with central pontine myelinolysis. In addition, bifrontal and left occipital cortico-subcortical T2/FLAIR hyperintensities without diffusion restriction or enhancement were identified, without mass effect, raising the possibility of a concomitant posterior reversible encephalopathy-type process. Vascular imaging was normal. This case illustrates the importance of considering osmotic demyelination syndrome in haemodialysis patients presenting with unexplained refractory seizures, even without a classical history of hyponatraemia correction, and highlights the complementary diagnostic value of multimodal MRI in this setting.

KEYWORDS

Central pontine myelinolysis, Osmotic demyelination syndrome, Haemodialysis, End-stage renal disease, Status epilepticus

MAIN ARTICLE

Introduction

Osmotic demyelination syndrome (ODS) is a rare, non-inflammatory demyelinating disorder that classically affects the central basis pontis (central pontine myelinolysis, CPM) but can also involve extrapontine structures such as the thalami, basal ganglia, cerebellum, and subcortical white matter (extrapontine myelinolysis, EPM) [1]. It was first described by Adams et al. in 1959 in the context of chronic alcoholism and malnutrition, and its most widely recognised cause remains the overly rapid correction of chronic hyponatraemia [1]. Beyond hyponatraemia correction, patients undergoing haemodialysis, particularly those with end-stage renal disease, constitute a recognised at-risk population, in whom rapid shifts of osmotically active particles during dialysis sessions can trigger the syndrome, even without a documented episode of severe hyponatraemia [2,3].

Clinically, ODS typically follows a two-phase course: neurological symptoms are delayed relative to the inciting osmotic insult, often emerging only after an interval of apparent stability lasting from about one day up to two weeks, and then progress to reflect corticospinal and corticobulbar involvement in the pons — dysarthria, dysphagia, or spastic tetraparesis [1]. Seizures are also a recognised, if less specific, feature of ODS, reported in up to roughly a quarter of cases and sometimes outlasting any seizures attributable to the acute metabolic disturbance itself [1].

We report the case of a haemodialysis patient with end-stage renal disease who presented with refractory status epilepticus and was found on brain MRI to have imaging features of central pontine myelinolysis, together with an associated cortico-subcortical signal abnormality, illustrating the diagnostic work-up and differential considerations in this setting.

Case Report

A 32-year-old woman with end-stage renal disease on maintenance haemodialysis, with no other notable past medical history, was admitted for refractory convulsive status epilepticus. Initial brain CT performed at admission showed no abnormality, prompting further aetiological work-up with brain MRI.

MRI was performed using axial T2-weighted, diffusion-weighted, 3D FLAIR, and sagittal T1-weighted sequences, together with arterial and venous time-of-flight (TOF) angiography and 3D gadolinium-enhanced sequences.

Imaging revealed a centropontine and mesencephalic signal abnormality, hypointense on T1-weighted images and hyperintense on T2 and FLAIR sequences, with restricted diffusion and no enhancement following gadolinium injection (Figure 1). In addition, cortical signal abnormalities were identified in the bilateral frontal lobes and the left occipital lobe, hyperintense on T2 and FLAIR sequences, without corresponding abnormality on the other sequences.

The midline structures were in place, and the cisterno-ventricular system was of normal size and morphology, without mass effect. Arterial and venous TOF angiography showed good opacification of the circle of Willis and the dural venous sinuses. Bilateral chronic-appearing maxillary sinusitis was noted as an incidental finding.

The overall MRI appearance was considered consistent with central pontine myelinolysis, together with an associated cortico-subcortical bifrontal and left occipital signal abnormality of separate radiological character. The patient was managed jointly by the nephrology and neurology teams, with careful correction of any underlying electrolyte and osmotic disturbances, optimisation of the dialysis prescription, and continued antiepileptic treatment.

Discussion

Central pontine myelinolysis has long been recognised as a hallmark of ODS, classically triggered by overly rapid correction of severe or chronic hyponatraemia; however, ODS is increasingly recognised as an important complication in patients with end-stage renal disease undergoing haemodialysis, in whom the syndrome may develop even without a clearly documented episode of hyponatraemia correction [1,2]. In this population, the proposed mechanism involves rapid shifts of osmotically active particles — principally urea, but also sodium and other solutes — out of the plasma during dialysis, producing an abrupt fall in extracellular osmolality relative to the brain and triggering osmotic injury to oligodendrocytes [2]. Both central and extrapontine involvement have been described in this context, sometimes developing after only a single haemodialysis session [3].

As already noted, ODS typically evolves over a period of days up to two weeks after the inciting osmotic insult before neurological signs become manifest, most classically dysarthria, dysphagia, and progressive tetraparesis reflecting corticospinal and corticobulbar tract involvement in the pons [1]. Seizures occur in a meaningful minority of patients and, when present, may outlast the seizures attributable to the acute metabolic derangement itself, particularly when extrapontine or cortical involvement coexists, as in the present case [1].

The additional bifrontal and left occipital cortico-subcortical signal abnormality identified in our patient, characterised by T2/FLAIR hyperintensity without diffusion restriction or contrast enhancement, raises the possibility of a concomitant posterior reversible encephalopathy syndrome (PRES)-type process rather than extrapontine myelinolysis alone. PRES is a well-recognised complication in patients with end-stage renal disease, in whom hypertension, uraemia, fluid shifts during dialysis, and endothelial dysfunction are thought to impair cerebral autoregulation and disrupt the blood-brain barrier, producing vasogenic oedema that characteristically, though not exclusively, involves the posterior and parieto-occipital regions [4]. Importantly, PRES is itself a well-recognised cause of seizures in the dialysis population [4], which may further account for the clinical presentation in our patient.

Diagnostically, MRI is central to distinguishing ODS from its mimics, including ischaemic stroke, demyelinating disease, and PRES. Diffusion-weighted imaging is particularly informative in this respect: the pontine lesion of ODS has been shown to demonstrate restricted diffusion within the first 24 hours of neurological onset, ahead of conspicuous changes on conventional T1, T2, and FLAIR sequences, reflecting acute cytotoxic injury to the myelin sheath [5]. This restricted-diffusion pattern contrasts with the facilitated diffusion typically seen in the vasogenic oedema of PRES, a distinction that can help separate the two processes when both are considered in the differential, as in our patient's cortical lesion. On conventional sequences, the pontine lesion of CPM classically takes on a trident- or piglet-shaped configuration on axial imaging, reflecting relative sparing of the corticospinal and corticobulbar tracts, a morphological clue that can support the diagnosis alongside the signal and diffusion characteristics described above [6]. The absence of contrast enhancement in both lesions in our case is consistent with the non-inflammatory nature of ODS and the reversible vasogenic oedema of PRES, respectively, and helped exclude alternative diagnoses such as infection or neoplasm.

Management of ODS in the haemodialysis setting is largely supportive, centred on careful correction of any underlying electrolyte disturbance, avoidance of further rapid osmotic shifts during subsequent dialysis sessions, and management of associated complications such as seizures [3]. The prognosis of ODS is variable and historically regarded as poor, though modern case series suggest that meaningful neurological recovery is possible with supportive care and time, particularly when the diagnosis is made early and further osmotic insults are avoided [1].

Conclusion

This case illustrates central pontine myelinolysis as an under-recognised cause of refractory seizures in patients with end-stage renal disease undergoing haemodialysis, occurring even without a classical history of rapid hyponatraemia correction. It underscores the importance of considering osmotic demyelination syndrome in the differential diagnosis of unexplained neurological deterioration in this population, and highlights the value of multimodal MRI, including diffusion-weighted imaging, in distinguishing central pontine myelinolysis from its principal mimics, including posterior reversible encephalopathy syndrome.

FIGURES

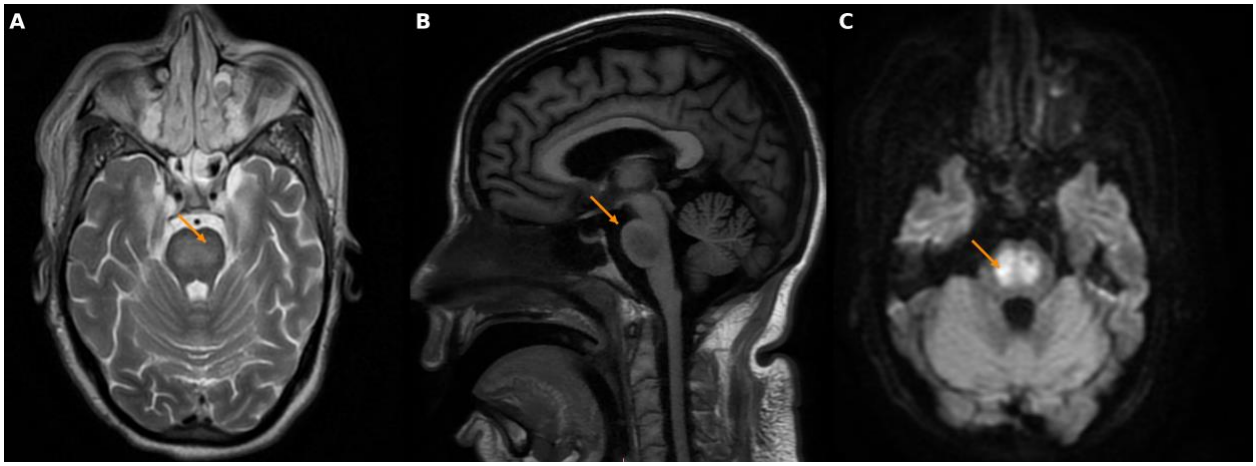

Figure 1. Brain MRI showing the centropontine lesion (orange arrows). (A) Axial T2-weighted image at the level of the pons. (B) Midsagittal image showing the centropontine and mesencephalic extent of the lesion. (C) Axial diffusion-weighted image showing marked restricted diffusion within the central pons, consistent with acute central pontine myelinolysis.

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Consent: Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

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