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CASE REPORT

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Hemodialysis Induced Osmotic Demyelination Syndrome in a Eunatremic Patient

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Abstract

Osmotic demyelination syndrome (ODS) has been described in end-stage renal disease patients recently started on hemodialysis and is usually attributed to a rapid correction of hyponatremia. We describe a case of ODS developing after recent hemodialysis and unrelated to serum sodium changes. Our aim is to help provide more data about the pathophysiology and precipitating factors of this syndrome. ODS is seen in the setting of acute osmotic changes, which has been historically linked to sodium levels. We hope that our clinical case highlights the other possible contributing factors leading to this syndrome and will aid in proper avoidance and suitable management of this condition. **(International Journal of Biomedicine. 2018;8(3):250-252.)**

Key Words: osmotic demyelination syndrome • hemodialysis • chronic renal failure • hypertension

Introduction

ODS (formerly called central pontine myelinolysis or CPM) is a well-known clinicopathologic entity characterized by edema and demyelination in the pons and extra-pontine areas.[1] It is caused by an acute shift in serum osmolality, which is classically related to rapid correction of hyponatremia.[2] We present a case of ODS occurring after recent dialysis in a patient with end-stage renal disease and normal serum sodium level.

Case presentation

A 34-year-old male with a medical history of uncontrolled hypertension presented to the emergency department as a referral from his primary care doctor for hypertensive urgency. Upon presentation, he was noted to have an elevated blood pressure with a systolic of 185 mmHg and diastolic of 120 mmHg. He was asymptomatic and denied any headache, blurry vision, chest pain or shortness of breath. However, he confirmed decreased exercise tolerance for 3 weeks and dark urine for 2 days; he denied any dysuria or flank pain. His examination was unremarkable and there was no jugular venous distention, pulmonary crackles or lower extremity edema. His neurological exam was also intact with no focal deficits.

The initial basic metabolic panel showed an acute kidney injury with blood urea nitrogen (BUN) of 142 mg/dl and creatinine (Cr) of 16.6 mg/dl; sodium (Na) level was 133 mEq/L and potassium (K) was 4.3 mEq/L. Urine analysis was notable for +3 hematuria and +3 proteinuria. Patient was started on a nicardipine drip and admitted to the medical intensive care unit (MICU) for close monitoring. A few hours later, he was noted to be in respiratory distress with tachypnea, diaphoresis and accessory muscle use, and the decision was made to intubate the patient. A post-intubation chest X-ray showed acute pulmonary edema. Renal service was consulted and recommended starting hemodialysis. Patient was also started on oral antihypertensive medications and titrated off the nicardipine drip.

Apart from poor mental status off sedation and failure to tolerate the ventilation weaning trials, the remainder of his MICU course was unremarkable. Off sedation, patient was noted to open his eyes, but was unable to track movements or to follow simple commands; he had a right gaze preference, was able to withdraw to pain with a weak movement and had intact reflexes. In view of the unexplained deterioration in mental status, a brain CT (Fig. 1a, 1b) was obtained, which showed periventricular white matter changes necessitating a brain MRI for better visualization.

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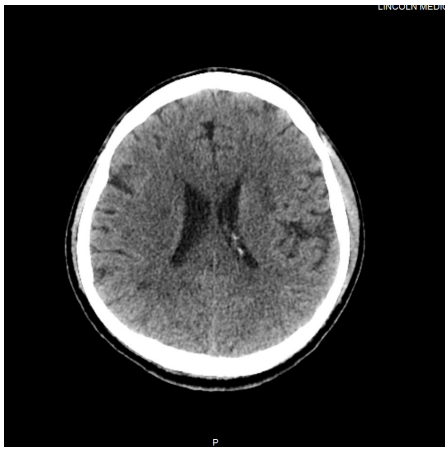

Fig. 1a

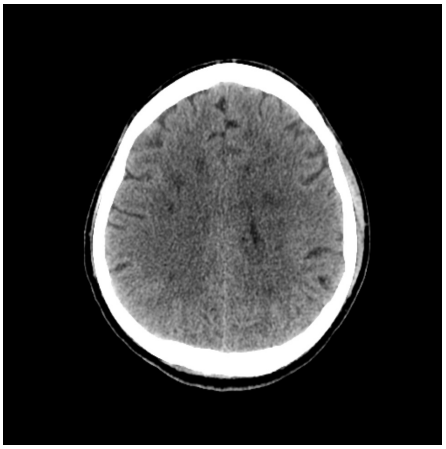

Fig. 1b

The brain MRI (Fig. 2a-d) was remarkable for numerous T2 hyper-intense lesions in the white matter, particularly seen within the corpus callosum and periventricular white matter. The majority of these lesions demonstrated diffusion restriction, suggesting active demyelination.

The trend of the patient's sodium and BUN levels before and after starting the dialysis is provided in Table 1. Lumbar puncture was done to exclude other etiologies, specifically multiple sclerosis and acute disseminating meningoencephalitis.

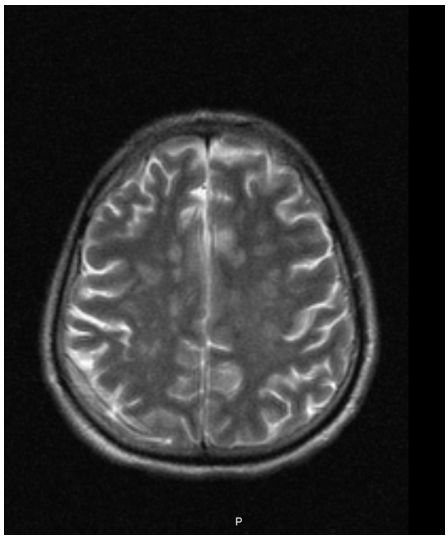

Fig. 2a

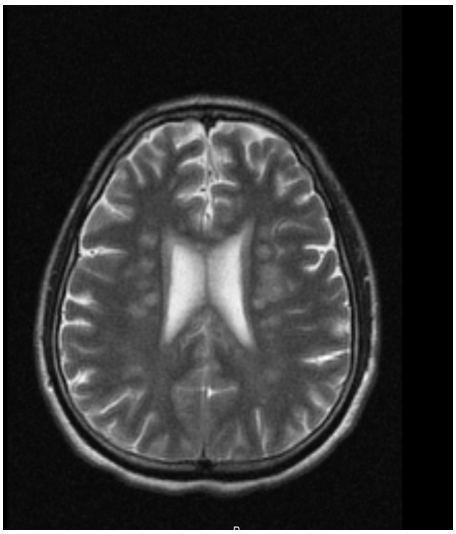

Fig. 2b

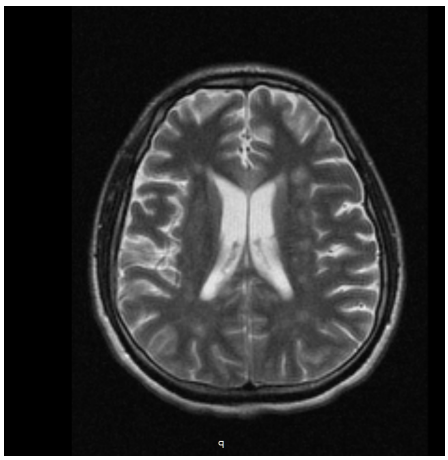

Fig. 2c

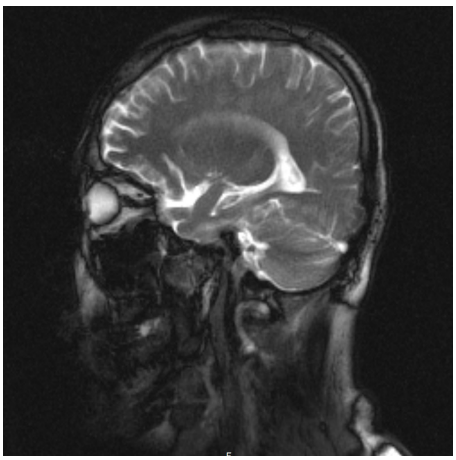

Fig. 2d

Table 1.

	Na (mEq/L)	BUN (mg/dl)
Before dialysis	133	142
Day 1 after dialysis	138	79
Day 2 after dialysis	138	51

Results were remarkable for normal CSF white cell count and protein level, as well as negative oligoclonal bands. The patient was started on high dose steroid therapy with methylprednisolone sodium succinate 1000 mg daily for 5 days; unfortunately, there was no improvement in the patient's mental status; hence, tracheostomy and percutaneous endoscopic gastrostomy were pursued.

Discussion

Central pontine myelinolysis was initially described by R.Adams in 1959 as a disease affecting alcoholics and malnourished patients.[1,3,4] The concept was extended in 1962 with the recognition that the disease can also affect extra-pontine sites (extra pontine myelinolysis or EPM).[4] Since then the name "osmotic demyelination syndrome" has become more popular.(1) Various mechanisms have been proposed for the development of ODS; the primary pathophysiology is reduced adaptive capacity of the glial cells to large shifts in serum osmolarity.[5] The condition is classically related to rapid correction of hyponatremia. However, it has also been reported in normonatremic patients, especially in patients with chronic renal failure, hypokalemia, and liver disease and in patients following liver transplantation.[6]

In patients with end-stage renal disease, ODS may develop as a result of the disease itself or because of osmotic changes during hemodialysis.[1] Y.Endo et al. reported a 14% incidence of ODS in patients with end-stage renal disease receiving hemodialysis.[7]

The proposed hypothesis of osmotic demyelination is osmotic injury to the endothelium from the rapid changes in serum osmolality, resulting in the release of myelinotoxic factors leading to disruption of the oligodendrocytes.[8] As mentioned before this has been historically related to changes in serum sodium, which is the most important solute contributing to serum osmolality. However, a few case reports described the syndrome in eunatremic patients, suggesting that other solutes also play an important role in the disease pathophysiology. Our case possibly developed ODS due to rapid changes in the serum BUN level as serum sodium has been stable through his hospital course and there were no exogenous solutes contributing to the effective serum osmolality.

Competing interests

The authors declare that they have no competing interests.

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