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Case Report

Post dialysis osmotic demyelination syndrome: a case report

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ABSTRACT

Osmotic demyelination syndrome (ODS) is a serious, irreversible, highly lethal clinical condition that is rarely described in the literature. It is caused by rapid correction of serum sodium, which generates abrupt changes in cerebrospinal fluid osmolarity, triggering neurological signs typical of this condition. It can occur due to rapid sodium correction through intravenous fluids and after emergent dialysis treatment in patients with renal pathologies, as described in this case; therefore, we consider it important to emphasize that dialysis treatment should be individualized based on a strict assessment of the internal environment for its correct correction, thus avoiding long-term sequelae and even death. We present a 38-year-old male patient with stage 5 chronic kidney disease, which did not allow dialysis treatment, admitted to a second-level hospital due to uremic encephalopathy and severe hyponatremia. During his stay in intensive care, he developed tonic-clonic seizures and neurological impairment, which began after emergency dialysis treatment, where rapid correction of trans dialytic serum sodium values was performed, generating classic neurological signs and imaging studies compatible with ODS.

Keywords: Osmotic demyelination, Hyponatremia, Post dialysis

INTRODUCTION

Hyponatremia, defined as a serum sodium concentration <135 mmol/l, is the most common fluid and electrolyte disorder in clinical practice. Hyponatremia can cause a wide spectrum of clinical symptoms, from subtle to severe or even life-threatening, and is associated with increased morbidity and mortality and prolonged hospital stay.[1] Osmotic demyelination syndrome (ODS), also known as pontine/extrapontine myelinolysis, is caused by the rapid correction of chronic hyponatremia, which leaves neurons temporarily hypotonic relative to the extracellular environment. This results in a sudden loss of water and, consequently, an abrupt reduction in cell volume. This leads to the loss or destruction of the myelin sheath and oligodendrocytes covering nerve cells in the pontine and some extrapontine regions of the brain.[2] When renal replacement therapy is administered to patients with dysnatremia, controlled correction of sodium imbalances is necessary to reduce the risk of osmotic demyelination syndrome (hyponatremia) or dialysis imbalance and cerebral edema syndrome (hypernatremia).[3] Symptoms of this syndrome begin between the second and sixth day after rapid correction of hyponatremia. It can include neurological symptoms such as dysarthria, mutism, dysphagia, behavioral and movement disorders, lethargy, confusion, disorientation, seizures, obtundation, and coma. The most common cause of this condition is thought to be related to the severity and duration of hyponatremia, the speed of correction, and the patient's risk factors and other associated comorbidities. Of all these causes, the speed of correction is the most important, as it is the only modifiable factor; when monitored by a physician, it can be avoided or controlled.[2]

CASE REPORT

A 38-year-old male patient with a personal medical history of poorly controlled hypertension due to the discontinuation of medical treatment, stage 5 chronic kidney disease requiring renal replacement therapy, which

he had previously refused, was admitted to the emergency room with a clinical picture characterized by asthenia, malaise, and melena on two occasions within two days of each other. He was admitted requiring emergency haemodialysis with laboratory values of haemoglobin (Hb) 4.8 g/dl, sodium (Na) 94 mmol/l, urea 268 mg/dl, creatinine 20 mg/dl, urinalysis (EMO) with glucose 100 mg/dL, haemoglobin 10 ery/ul, and protein 500 mg/dl. Arterial blood gas pH 7.43, sodium 100 mEq, potassium (K) 4.0 mEq, chloride (Cl) 62 mEq, glucose 87 mg/dl, lactate (Lac) 0.8, BE EFC -13.7, respectively, with no signs of deterioration in his neurological status.

He was assessed by the nephrology service which prescribed the start of haemodialysis through a double-lumen right anterior jugular temporal catheter. Haemodialysis was started with conductivity (Na 138 mEq/l, sodium bicarbonate (HCO3) 32 mEq/l), with a dialyzed time of 2 hours, E 13H ultrafiltration filter of 1500 ml, T° 36.5, pump flow of 200 ml/min, a dialysate flow of 300 ml/min, without heparin, with transfusion of 2 red blood cell packs and 1 intradialytic fresh frozen plasma.

Laboratory tests were recorded after the haemodialysis session with a basal Na of 110 mEq/l. Authors observed that the serum Na value changed after the first conventional dialysis treatment by 16 mEq, no neurological symptoms were reported, subsequently continued with 2 haemodialysis sessions, reporting in the last session an episode of tonic-clonic seizure lasting approximately 30 seconds, requiring the administration of intravenous benzodiazepine, without observing improvement in the state of consciousness deciding advanced airway management and start of mechanical ventilation, laboratory tests report Na 125 mmo/l, K 2.7 mmo/l, Cl 87.4 mmol/l, urea 103 mg/dl, creatinine 10.4 mg/dl, hb 6.7 mg/dl, hematocrit 18%.

Ventilatory weaning was performed on the 6th day achieving successful extubation, with tetraplegia predominantly in the right side of the body, mutism, dysphagia, emotional lability, suggesting pseudobulbar syndrome, 9 days after admission he presented with hematemesis greater than 200 ml with the need again for airway protection, blood transfusion, with evaluation and resolution by the gastroenterology service. As an analysis of his evidenced state, we suspect that it may be an event of osmotic imbalance vs changes due to aggressive sodium correction, being evaluated by the neurology service, with a simple magnetic resonance imaging study of the brain (Figure 1) in which structural lesions such as infarction or hemorrhage in the brainstem were ruled out, however, a diffuse hyperintense image was already appreciated at the central level of the pons that justified his symptoms.

After 18 days after admission, he again had tonic-clonic seizures, antiepileptic drugs were started, a lumbar puncture was performed which ruled out neuroinfection, due to his prolonged stay a percutaneous tracheostomy was performed, a new magnetic resonance study was requested confirming pontine demyelination, due to evidence of the radiological sign of the trident (Figure 2). Subsequently, he evolved torpidly, presenting multiple cardiological, hematological, hemodynamic and renal complications, due to which he suffered cardiorespiratory arrest without being able to restore his spontaneous circulation after basic and advanced support and resuscitation measures.

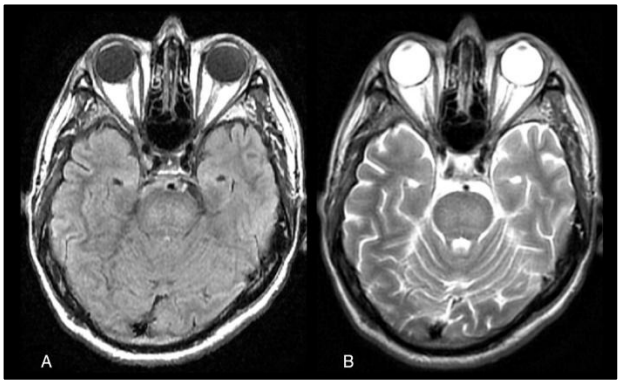

Figure 1: Brain magnetic resonance imaging in axial sections at the pontine level (A) FLAIR and (B) T2 with diffuse hyperintensity of central bridge distribution.

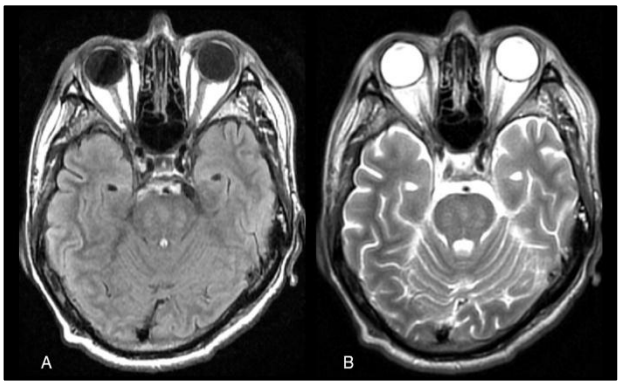

Figure 2: Brain MRI (A. FLAIR, B. T2) in axial sections at the pontine level; the predominant involvement of the transverse pontine fibres and the relative sparing of the descending corticospinal tracts are responsible for this hyperintense image with the characteristic "trident sign."

DISCUSSION

Osmotic demyelination syndrome is a serious neurological complication resulting from rapid correction of chronic hyponatremia. Slow sodium correction is essential to prevent this complication. The current recommendation is not to increase serum sodium by more than 8–10 mmol/l in the first 24 hours and no more than 18 mmol/l in 48 hours.[4] Huang et al, documented a case of ODS that developed after rapid correction of hyponatremia by haemodialysis in a uremic patient.[4] The study highlights the importance of gradual and controlled correction of

serum sodium to prevent serious neurological complications. They suggest two strategies to achieve this goal. First, the dialysate sodium level should not be 15–20 mEq/l higher than the plasma level, we can see that in our patient the dialysate sodium level was 44 mEq/l different from the plasma sodium. Second, low blood flow should be used; this results in slow movement of Na from the dialysate to the serum and corrects hyponatremia slowly. However, according to Wendland et al whether hemofiltration (CVVH) or haemodialysis (CVVHD) is used, the factor that we could control to manipulate the rate of change of serum Na would be the flow rate of the replacement fluid (CVVH) or the dialysate (CVVHD).[5]

The most likely pathogenetic mechanism behind osmotic demyelination syndrome in patients with end-stage renal disease may be the rapid change in plasma solute levels, resulting in the shift in serum osmolality that occurs during or after hemodialysis.[6] A study by Pattanashetti et al described the successful management of severe hyponatremia in patients with chronic kidney disease in a resource-limited setting, using conventional haemodialysis with a dialysate sodium concentration of 128 mEq/l and a blood flow reduced to 50 ml/min.[7] This approach allowed for gradual correction of sodium without inducing ODS. Among the valuable data in our case, it can be analyzed that the dialysis was carried out with an elevated sodium concentration and a blood pump flow of 200 ml/min, well above the regimens suggested in the reviewed literature.

In this case, the initial sodium correction from 94 mmol/l to 110 mml/l in the first haemodialysis session was significant and, although apparently safe, may have contributed to the subsequent development of ODS. Haemodialysis with a dialysate sodium concentration of 138 mEq/l is standard. However, in cases of severe hyponatremia, more controlled methods for gradual sodium correction could be considered, as suggested in the reviewed studies.[10] As noted by Ağildere et al the neurological evaluation of patients with chronic kidney disease on dialysis is difficult due to the wide spectrum and complexity of complications in this setting.[8] Therefore, multidisciplinary management by intensivists, nephrologists, and neurologists is essential to reduce their incidence, influenced by close monitoring through laboratory tests and clinical behavior. The use of brain MRI is clinically relevant at the end of all previous links in health care and solely as a diagnostic method, as it has greater sensitivity and specificity for determining SDO.

However, once brain injury is established, morbidity and mortality are higher and generally irreversible. It is essential to adhere to the recommendations for gradual sodium correction to minimize the risk of ODS. The exact mechanism remains hypothetical, and further human studies are needed to answer this question.[9] However, the literature suggests that in patients with extremely low serum sodium levels before haemodialysis, dialysate sodium reduction and multiple, brief dialysis sessions with low blood flow are recommended to decrease the risk of developing ODS.[4,8,9]

CONCLUSION

Osmotic demyelination, although rare, is potentially life-threatening and is characterized by a progressive neurological condition that requires the combination of laboratory and imaging findings. Most cases are associated with accelerated sodium repletion in patients with severe hyponatremia. This case highlights the importance of careful sodium correction in patients with severe hyponatremia and CKD. A multidisciplinary approach and rigorous monitoring can help prevent serious complications such as osmotic demyelination syndrome. The strategies described in the literature provide a useful framework for the safe and effective management of these patients.

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