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A Case of Osmotic Demyelination Syndrome: Diagnostic and Therapeutic Challenges

Hajar Joulal[1*], Lamia Essaadouni[1]

[1]Internal Medicine Department, Mohamed VI University Hospital BP2360 Principal, Avenue Ibn Sina, Marrakech, Morocco

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***Corresponding author:** Hajar Joulal
Internal Medicine Department, Mohamed VI University Hospital BP2360 Principal, Avenue Ibn Sina, Marrakech, Morocco

Abstract | **Case Report**

Osmotic demyelination syndrome (ODS) is a rare anatomoclinical entity characterized by the destruction of myelin sheaths, most often secondary to very rapid correction of hyponatremia. Its clinical manifestations are highly variable, and its treatment is essentially based on prevention. We report here the case of ODS in a 54-year-old patient after correction of hyponatremia secondary to uncontrollable vomiting. The diagnosis of ODS was made by magnetic resonance imaging. The patient had more than one vulnerability factor that could explain the development of this syndrome. Even after restoration of fluid and electrolyte balance, she retained neuropsychiatric sequelae such as intermittent agitated confusion and ataxic gait.

Keywords : Osmotic Demyelination Syndrome, Centropontine Myelinolysis, Extrapontine Myelinolysis, Hyponatremia, Diabetes.

INTRODUCTION

Osmotic demyelination syndrome (ODS) is a rare anatomoclinical entity characterized by the destruction of myelin sheaths, predominantly in the white matter tracts at the pons, hence its former name, centropontine myelinolysis [1]. Its precise pathophysiology is still poorly understood, although rapid correction of hyponatremia remains the classic risk factor for this condition. Its clinical manifestations are highly variable, ranging from encephalopathy to coma or even death, with a poor prognosis [2]. We report here the case of ODS associating centropontine and extrapontine myelinolysis in a 54-year-old patient following rapid correction of hyponatremia secondary to uncontrollable vomiting.

CASE REPORT

Mrs. K. A is a 45-year-old patient, known to have type 2 diabetes for 20 years and been on insulin therapy, high blood pressure for 4 years and been on perindopril 5 mg and amlodipine 5 mg/day, and recently discovered Hashimoto's disease and thiamazole 20 mg/day.

Food not relieved by antiemetics with persistent diffuse abdominal pain without other associated signs, notably no ocular, neurological or cardio-respiratory signs motivating an emergency consultation, and allowing the identification and surgical management of acute cholecystitis with multi-macro -lithiasic gallbladder.

A postoperative workup revealed hyponatremia at 113 meq/L - attributed to vomiting - following which parenteral correction was performed. The remainder of the biological assessment is summarized in Table 1.

Table 1: Biological data of our patient

Balance sheet	Results
Blood count	
Hemoglobin	9.9 g/ dL
MCV	89.1fL
MCH	28.2 pg
Leukocytes	10340/mm3

Balance sheet	Results
Platelets	403,000/mm3
C-Reactive Protein	5
Kidney function	
Serum urea	0.7g/L
Serum creatinine	13.4 mg/L
GFR	51ml/min/1.73m²
Ionogram	
Serum sodium	113 meq /L
Urinary sodium	107 meq /24h
Serum potassium	5.6 meq /L
Urinary potassium	26 meq /24h
Chloremia	51 meq /L
Serum Calcium	84 mg/L
Lipasemia	54 IU/L
Liver workup	
AST	22 IU/L
ALT	18 IU/L
ALP	45 IU/L
Total bilirubin	8.7 µmol /L
Prothrombin rate	100%
Thyroid assessment	
TSH	0.37 mU /L
T4 / T3	7.8 / 2.1 µg /dL

ALT:

Alanine Aminotransferase; AST: Aspartate Aminotransferase; ALP: Alkaline Phosphatase; GFR: Glomerular Filtration Rate; MCV: Mean Corpuscular Volume; MCH: mean corpuscular hemoglobin; TSH: thyroid stimulating hormone

On the 2nd day of correction, the patient presented with confusion, drowsiness and spatio-temporal disorientation alternating with states of agitation, dysarthria and tetraparesis. Her natremia showed a rapid correction of this with a rate of 138 mmol/l associated with a worsening of her renal function (GFR at 32ml/min/1.73m²). An emergency cerebral MRI angiography showed centropontine signal anomalies in the left cerebellum compatible with centropontine and extrapontine myelinolysis, with normal arterial or venous MRI angiography sequence, which could explain the neurological disorders of our patient.

The patient was admitted to the intensive care unit, where subsequent management included adjusting the rate of intravenous sodium supplementation, along with Proton Pump Inhibitors and thromboembolic prophylaxis. She was then transferred to the internal medicine ward once stabilized.

A renal ultrasound, performed as part of the etiological workup for her renal failure, revealed bilaterally small kidneys (7.4 cm on the right and 8.6 cm on the left in maximum length), irregular and differentiated, consistent with diabetic nephropathy.

The clinical course was marked by regained consciousness and recovery of motor deficit, with persistent, though less frequent, episodes of agitation, and an ataxic gait.

Following stabilization of glycemic and blood pressure levels, the patient was discharged with an adjusted treatment regimen for her diabetes and hypertension. She was referred to a motor rehabilitation center and scheduled for a regular follow-up in outpatient consultations for ongoing clinical assessment.

DISCUSSION

ODS is a severe, albeit rare, condition with significant comorbidity. The pathophysiology of ODS remains poorly understood. One proposed theory is based on the disruption of the brain's adaptive mechanisms to chronic hyponatremia, triggered by its rapid correction — specifically through the elimination of intracellular solutes and water via ion channels, and the efflux of organic osmolytes such as glutamate and glycine [3].

This would result in dehydration of brain tissue, with impaired reuptake and loss of osmolytes, leading to astrocytic injury accompanied by inflammation and microglial activation. Consequently, demyelination occurs, predominantly affecting the white matter of the pons, but also involving the midbrain, thalamus, basal ganglia, and cerebellum [1-4].

Several factors have been implicated in the pathogenesis of ODS (Table 2). While rapid correction

of hyponatremia is a well-recognized classic cause, spontaneous (or "autocorrection") of serum sodium levels is often overlooked in such cases. Autocorrection is defined as a spontaneous rise in serum sodium concentration without therapeutic intervention, most commonly due to suppression of antidiuretic hormone secretion [5]. Administration of glucocorticoids in patients with adrenal insufficiency, discontinuation of thiazide diuretics or medications that induce syndrome of inappropriate antidiuretic hormone secretion (SIADH), such as carbamazepine or desmopressin (used in diabetes insipidus, hemophilia, and von Willebrand disease), spontaneous resolution of a transient trigger of ADH secretion, such as relief of surgical stress or improvement of nausea and vomiting, which was likely an aggravating factor in our case, among others. Thus, even a so-called "appropriate" correction of hyponatremia may not, on its own, be sufficient to prevent ODS.

Table 2: Risk Factors for the Development of Osmotic Demyelination Syndrome

Risk Factors	Ref
Chronic hyponatremia lasting more than 48 hours	[1]
Severe hyponatremia (serum sodium < 120 mmol/L)	[1]
Aggressive correction of hyponatremia	[6]
Chronic alcoholism and vitamin deficiencies	[7, 8]
Liver transplantation	[9, 10]
Extensive burns	[11]

Clinical manifestations of ODS are highly variable, depending on the patient's background, the severity of the precipitating factor, and the presence or absence of extrapontine myelinolysis. Typically, the course is biphasic, with an initial phase occurring 1 to 4 days after correction, characterized by an encephalopathic picture including seizures that resolve with normalization of serum sodium. This is followed by neurological signs consistent with central pontine myelinolysis, such as dysarthria, dysphagia, paraparesis or quadriparesis (due to involvement of spinal fibers), or extrapontine myelinolysis presenting with catatonia, postural tremor of the limbs, myoclonic jerks, and parkinsonian syndrome with dystonia. In severe cases, patients may present with lethargy, confusion, disorientation, coma, or a "locked-in" syndrome, with fatal outcomes occurring in approximately one-third of cases [1-12].

Except for autopsy, magnetic resonance imaging remains the only method to confirm the diagnosis of ODS. MRI typically shows lesions with low signal intensity on T1-weighted images and high signal intensity on T2-weighted and FLAIR sequences, affecting the central portion of the pons while sparing the peripheral areas. At later stages, the lesions may exhibit a characteristic "bat-wing" appearance [7]. It is important to note that a negative initial MRI does not exclude the diagnosis, and the imaging should be repeated within two weeks if the suspicion remains high [13].

The most effective treatment for ODS is preventive, consisting of an "appropriate" correction of hyponatremia. Although the recommended correction threshold for hyponatremia is 8–12 mEq/L per hour, the correction rate should not exceed 6–8 mEq/L per 24 hours in high-risk patients (see Table 2) [1]. In certain situations, re-lowering of serum sodium is recommended in response to rapid correction of hyponatremia, either through administration of desmopressin or infusion of dextrose-containing solutions [14, 15]. There is no curative treatment for ODS. Management of these patients involves lowering serum sodium using the methods previously described, intensive physiotherapy and rehabilitation, occasional respiratory support, and anti-parkinsonian medications depending on the clinical presentation.

Although the fatality rate has decreased from 90%–100% to 24.8%, ODS remains a condition with significant morbidity and mortality, with only 25%–40% of patients achieving complete recovery [16, 17]. Our patient, although clinically and biologically improved, continued to exhibit cognitive impairments and cerebellar ataxia—findings that have been reported in several case series [2].

Conclusion

ODS remains the most severe complication of rapid correction of hyponatremia. Although historically fatal, its prognosis has improved thanks to better understanding of the condition and its major risk factors, as well as the availability of improved diagnostic and therapeutic methods.

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