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Case Report

From Hyponatremia to Osmotic Demyelination: A Clinical Odyssey and Lessons Learned

Jafrin Sadiq Abdul Razack *[1], Sahil Asgar Ali [1], Syeda Juveria Hussaini [1], Saud Khaled Aldoseri [1], Harshavardhan Rajagopal [2], Antony Calvin Ossanam [1], Ibtisam Akram [1], Viorica Khalili [3]

[1]MBBS Students, College of Medicine, Gulf Medical University, Ajman, UAE.
[2]Intern Doctor, American Hospital Dubai, Dubai, UAE.
[3]Nephrology Consultant, American Hospital Dubai, Dubai, UAE.

*Corresponding author: Jafrin Sadiq Abdul Razack; *sadiq.jafrin@gmail.com*

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Abstract

A 76-year-old female presented with acute confusion and delirium of 3 days duration. The patient was admitted to the ICU in a state of acute confusion. History taken from the patient's daughter revealed that the patient had nausea, vomiting, diarrhea, and fever two days before admission. Blood tests and imaging were ordered, and after reviewing the results, a diagnosis of osmotic demyelination syndrome (ODS) was made.

Keywords: ***Hyponatremia, ODS, ICU, Case report, sodium, potassium***

Introduction

Osmotic demyelination syndrome (ODS) is a rare clinical condition that causes both pontine and extra-pontine myelinolysis (EPM). While many different etiological factors can cause it, the main pathophysiology that has been identified is either a diminished neuroglia's ability to adapt to significant changes in serum osmolarity, or cellular edema brought on by variations in electrolyte forces, which causes compression and subsequently demyelination of fiber tracts. The clinical entity has a highly dramatic prognosis, ranging from complete neurological recovery to vegetative condition [1].

A blood sodium correction of more than 12 mmol/l in 24 hours is frequently linked to ODS. ODS accounts for around 0.4-0.56% of all neurological hospitalizations at tertiary referral institutions, with MRI-based studies reporting a frequency of 0.3 to 1.1%. ODS is characterized by low incidence rates and high rates of death and disability [2].

Case presentation

A 76-year-old female was shifted from the tertiary hospital to the intensive care unit with acute confusion and delirium for the last 3 days. She was admitted in an acutely confused state. Labs revealed a low sodium level of 132 mmol/L (reference range of sodium is 135-145 mmol/L), despite being on 3% saline hypertonic. Her sodium dropped to 128 mmol/L after a few days. The daughter reported pre-admission symptoms of nausea, vomiting, 4 episodes of diarrhea, and a low-grade fever while being alert and oriented with normal mental status. Other physical examinations revealed no convulsion, cough, chest pain, or urinary symptoms. The patient's current sodium level as of January 2024 was 140 mmol/L. Currently, the patient is on dextrose 5% at 50 ml/hr, and mentally she is alert and oriented, with a Glasgow Coma Scale: 15/15.

The patient has hypertension, dyslipidemia, right eye glaucoma, COVID-19 pneumonia in 2021, and bouts of depression for more than 30 years. There is no significant family, social, or travel history. On arrival, the pulse was 104 beats per minute, respiratory rate of 15 breaths per minute, temperature of 36.6 °C, blood pressure was 139/69 mmHg, and the weight was 68 kg. Day 2: On the general examination appearance on the day, the patient was oriented to place and time. On the physical examination, there were no significant findings. On the nervous system examination, GCS 13/15, a basic metabolic panel, coagulation panel, liver function test, urine sample, TSH, and random cortisol were also tested. A urine culture was sent. A CT was taken for suspected SIADH due to lung disease. After looking into the lab values, a diagnosis of ODS was made.

Discussion

ODS mainly occurs in chronically ill patients and is exclusively seen in patients admitted to the hospital. The primary cause is rapid intravenous correction of hyponatremia; however, in a chronically ill patient, ODS can arise from mild hyponatremia as well as slow correction. There are multiple factors supported by the literature that predispose a patient to develop ODS, and they include factors like burns, chronic alcoholism, the syndrome of inappropriate antidiuretic hormone, malnutrition, etc. Our study found that hypokalemia was the most common associated factor that led to ODS. In a study conducted by Lampl and Yazdi, it was found that alcoholism and liver transplantation were the most common associated factors leading to the development of ODS [3].

A general deficit of organic osmolytes or a disturbed state of solute metabolism may be the general background that facilitates the

development of ODS in these patients. This alone predisposes cells to experience a change in volume and size, which increases the likelihood of cell shrinkage. The extent of the clinical picture varies according to the part of the central nervous system affected by demyelination. When the pons, corticobulbar, and corticospinal pathways are affected, the classic presentation is a flaccid paralysis that eventually becomes spastic, combined with dysphagia and dysarthria [4,5].

A significant factor in the diagnosis of ODS is radiology. It helps rule out alternative alternatives in addition to supporting the clinical suspicion. Demyelination manifests as a region of reduced attenuation on CT scans, but unfortunately, this may mislead one about the severity of the illness. Because it can detect lesions in both the pontine and extra-pontine regions, magnetic resonance imaging (MRI) is the preferred technique due to its increased sensitivity. Acute demyelinating lesions are visible as symmetric and hypointense lesions on T1-weighted and hyperintense T2-weighted images in the subacute phase. The usual picture appears as a trident-shaped area of hyperintense or hypointense lesion in the central pons with sparing of the ventrolateral pons and does not enhance with contrast. Lesions on MRI may appear days to weeks after the onset of symptoms, and these may or may not resolve even though there is partial or complete clinical recovery [6,7].

Once a diagnosis is established, treatment is supportive. Reports on random case reports and small case series have found the benefits of a diversity of treatment modalities such as steroids, intravenous immunoglobulin, thyrotrophin-releasing hormone, re-induction hyponatremia, administration of organic osmolytes (urea, myoinositol), and dopaminergic compounds, especially in EPM cases, etc. As there is no randomized human trial to date, all these possible therapies still need to be recommended for ODS patients.

The findings of this study indicate that to get better results, a well-organized supportive therapy and multidisciplinary approach are needed, rather than one of the many available but unproven forms of therapy. Additionally, as ODS is a complication rather than an illness in and of itself, the focus must be prevention rather than treatment. The blood sodium level of patients should be checked at regular intervals, and rapid overcorrection of sodium should be avoided to prevent the development of ODS.

Conclusion

In conclusion, the presented case underscores the critical importance of monitoring and managing serum sodium levels, particularly in patients with a history of hyponatremia. The rapid correction of hyponatremia can lead to Osmotic Demyelination Syndrome, as evidenced by the demyelination observed in the central pontine myelin. Early recognition of clinical symptoms and prompt intervention are crucial for optimizing patient outcomes.

This case report highlights the challenges in balancing the correction of electrolyte imbalances while preventing the onset of ODS. Further research and awareness are needed to refine treatment guidelines and prevent the occurrence of this debilitating neurological condition. Clinicians should remain vigilant, especially when managing patients with a history of hyponatremia, to mitigate the risk of Osmotic Demyelination Syndrome.

Conflict of Interest

The authors declared they do not have anything to disclose regarding conflicts of interest with respect to this manuscript

Ethics approval and consent to participate

Not Applicable

Data Availability

A data availability Regarding this case report can be accessed upon request to Corresponding Author (Jafrin Sadiq Abdul Razack)

Authors' contributions

JSAR, SAA, SJH, SKA, HR, ACO, IA, and VK analyzed and interpreted the patient data regarding case presentation . JSAR was a major contributor in writing the manuscript. SAA analyzed and writing under literature part of the case report. All authors read and approved the final manuscript."

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