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Case Report

Osmotic Demyelination Syndrome in Hyponatremic Patient Despite Appropriate Sodium Correction in 48 Hours

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Abstract

Osmotic demyelination syndrome is one of the serious complications of rapid correction of severe hyponatremia that possibly can lead to irreversible damage. Here we are reporting a case of a 51-year-old male, with history of recurrent vomiting, recent thiazide diuretic use and alcohol consumption, who presented with severe symptomatic hyponatremia in form of seizure and diminished level of consciousness with an initial sodium level of 99 mEq/L. He was managed with hypertonic saline with significant improvement in his symptoms and he had overcorrected sodium, which was managed with hypotonic saline and desmopressin. Successfully his sodium level maintained at the level of 115 mEq/L in the first 48 hours. However, after 7 days of hospital stay he had drop in his level of consciousness that required intubation and his brain MRI showed features of osmotic demyelination syndrome. After 3 months of hospital stay, the patient's mentation improved and he became fully oriented and alert, able to talk and moving with assistance. The important learning points of this case are to be very cautious with managing such cases especially those who have high risk factors with limiting the rate of sodium correction to the lowest possible level as 4-6 mEq/L/day and to maintain that even after the first 48 hours until the sodium reached a safe level.

Keywords: Hyponatremia, Osmotic Demyelination Syndrome, Sodium Over-correction

INTRODUCTION

Hyponatremia is defined as decrease in serum sodium concentration to a level below 135 mEq/L, and severe hyponatremia is defined as sodium level below 120 mEq/L (Gankam and Decaux, 2018). Despite hyponatremia is the most common electrolyte disturbance, the incidence of osmotic demyelination syndrome (ODS) which is one of the major complications of rapid correction is still rare down to 0.5% or 0.611% (Gankam and Decaux, 2018; George et al., 2018; Aegisdottir et al., 2019). ODS is characterized by non-inflammatory loss of myelin while the neuronal cell body and the axons are maintained due to disturbed blood

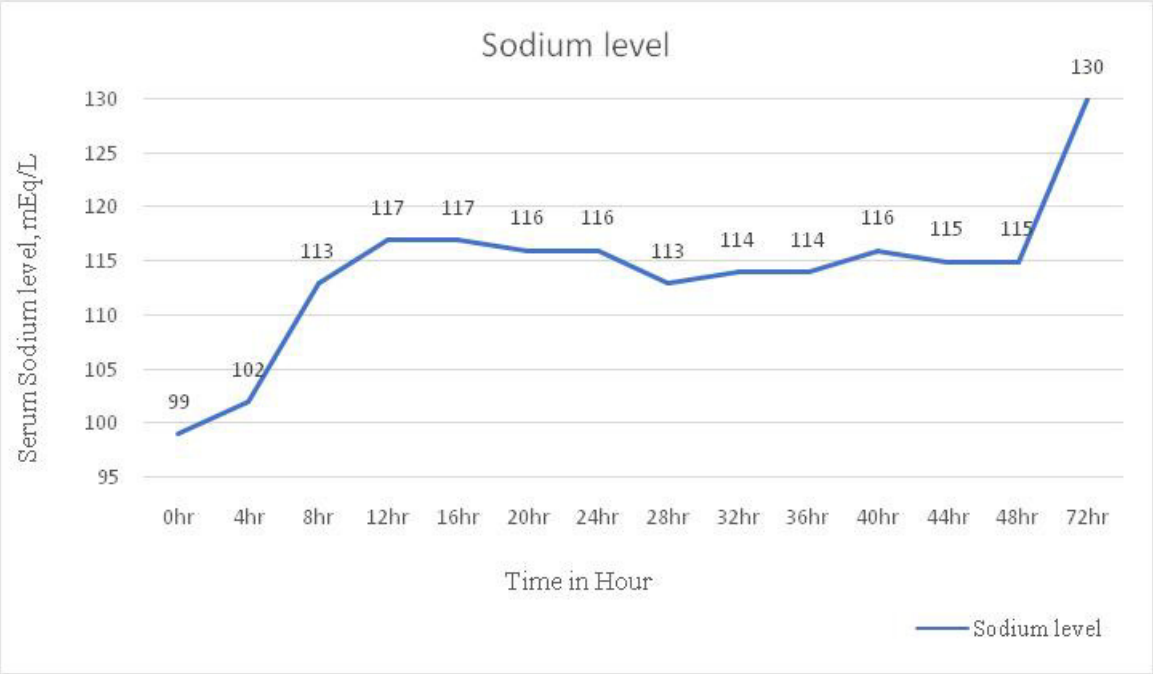

Figure 1. Timeline of Sodium Correction

brain barrier causing vasogenic edema (Alleman, 2014).

The process of brain adaptation to hyponatremia by moving osmotically active particles to the extracellular compartment to restore brain volume needs around 24-48 hours to be fully completed. Therefore, there is no limit for correction of acutely developed hyponatremia giving the duration of hyponatremia a key role in the response to rapid correction (Verbalis et al., 2013). Apart from the duration, the presence of other risk factors like sodium level <105 mEq/L, alcohol consumption, hypokalemia, malnutrition and chronic liver disease are exposing the patient for further risk to develop ODS (George et al., 2018; Alleman, 2014).

Current guidelines, based on expert opinion, have recommended to limit the sodium correction maximally to 8 mEq/L/day (Verbalis et al., 2013; Sterns, 2015; Arampatzis et al., 2012; Sood et al., 2013). Also, it is recommended to correct sodium by not more than 18 mEq/L in 48 hours (Verbalis et al., 2013; Braun et al., 2015; Pfennig and Slovis, 2012; Spasovsk et al., 2014).

Here we are reporting a case of ODS in a patient with severe hyponatremia, with risk factors of alcohol consumption, who had severe neurological symptoms at presentation. It ends in favorable neurological outcome.

CASE REPORT

A 51-year-old Saudi male who presented with 1 week history of imbalance, unsteady gait, progressive recurrent vomiting that associated with nausea and poor oral intake; two days before presentation he devolved intermittent visual hallucination and then he had an episode of generalized tonic colonic seizure (GTCS) that witnessed by his wife for which he was shifted to the emergency department (ED). While in ED, he experienced another episode of GTCS that lasted for a few seconds then it aborted spontaneously.

His past medical history is significant for recently diagnosed hypertension and 10 days before presentation he was started on Irbesartan-Hydrochlorothiazide and Nifedipine, he is not using other medications. His social history is significant for heavy cigarette smoking and chronic alcohol consumption for the last 20 years.

Physical examination revealed a body temperature of 36.4°C, heart rate of 73 beats/minute, blood pressure of 137/83 mmHg, respiratory rate of 16 breath/minute and oxygen saturation of 97% on room air. He was lethargic, disoriented to place and time and had no focal neurological deficit. He had very dry mucous membrane. Other systems examination were unremarkable.

His initial complete metabolic panel revealed severe hyponatremia with serum sodium of 99 mEq/L, plasma osmolality of 207 mOsm/Kg, potassium of 3.7 mEq/L, bicarbonate of 30 mEq/L, blood urea nitrogen of 3 mmol/L and creatinine of 30 µmol/L. PH was 7.50 and $PaCO_2$ of 37.5 mmHg. Urine sodium was 10 mEq/L. CT brain was negative for acute intracranial insults.

In ED, he was managed right away with 3% NaCl total of 200 ml, repeated sodium at 4 and 6 hours was 102 mEq/L. As the patient was still lethargic with impaired consciences, he was given another 100 ml of 3% NaCl,

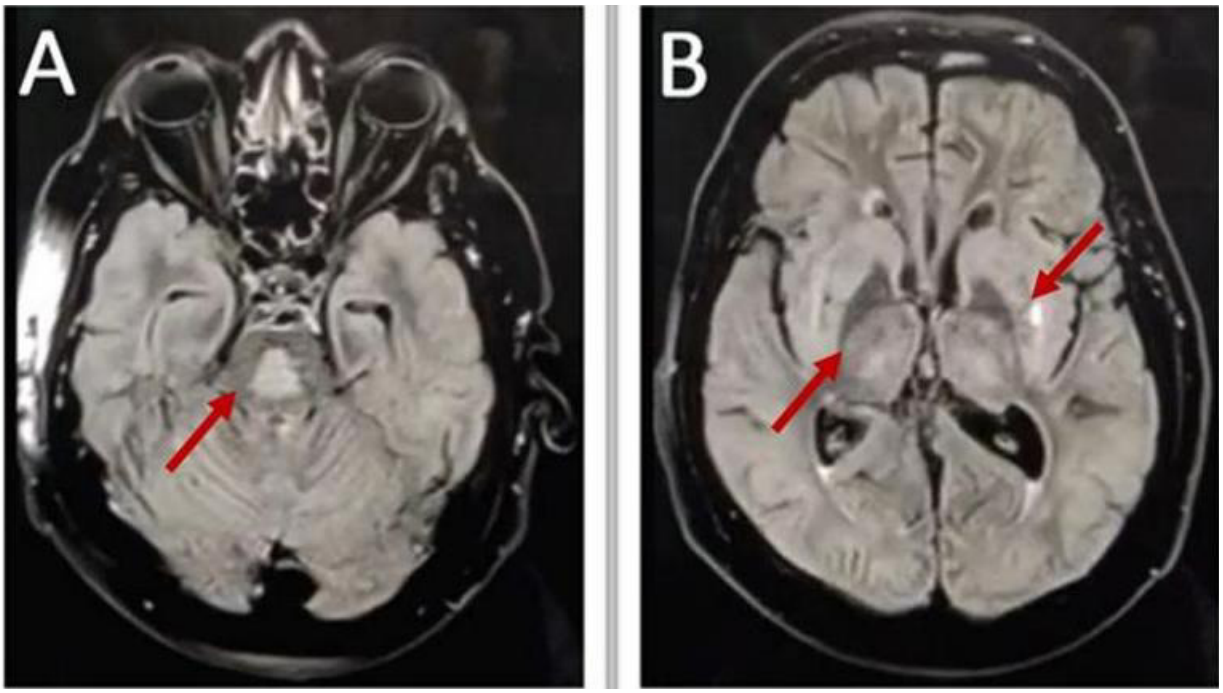

Figure 2: A: abnormal T2/flair hyperintensity crossing the midline; B: abnormal signal in the putamen and external capsule, high signal in bilateral ventrolateral thalami.

his level of consciousness started to improve and he was admitted to the intensive care unit (ICU). At 9 hours, his repeated sodium was 113 mEq/L and he started to be polyuric with urine output of 400 ml/hr, for which dextrose 5% water and single dose of desmopressin of 20 mcg IV were administered, his sodium level was 116 mEq/L at 24 hours and urine output was 70 ml/hr. The following day, he kept on 0.9% NaCl and sodium level at 48 hours was 115 mEq/L (Figure 1). His mentation was back to baseline, thiazide diuretic stopped and he was downgraded to the medical floor.

In the floor, he was fully oriented till day 7 of admission as he had drop in level of consciousness with Glasgow Coma Scale (GCS) of 10/15, along with new upper and lower limbs spasticity. Metabolic panel was normal, an urgent CT brain was negative and lumper puncture study was negative. He was shifted to ICU and there his GCS dropped further to 7/15 that required intubation. An urgent MRI brain was done and it showed abnormal signal intensity and diffusion restriction involving the central pons, ventrolateral thalami, basal ganglia, external capsules and gray-white matter junction of precentral gyri bilaterally and symmetrically, both central pontine and extrapontine myelinolysis, characteristic of ODS (Figure 2).

The patient remained in ICU for 3 weeks intubated with low GCS, then he underwent tracheostomy. He was shifted back to the medical floor with GCS of 9/15 and kept on supportive care that included nasogastric tube for feeding and aggressive physical therapy.

After 3 months of admission, he started gradually to improve, forming words, recognizing his family and following simple commands. By month 4, he became fully oriented, able to talk and moving with assistance. Tracheostomy closed and the patient was discharged with a follow up with physical and speech therapy.

DISCUSSION

Most of the time patients with severe hyponatremia will present with seizure and coma that are likely reflecting cerebral edema (Sterns et al., 2009), thus rapid correction with hypertonic saline is mandated to prevent brain swelling and subsequent fatal herniation (Gankam and Decaux, 2018; Sterns et al., 2009). It was suggested that a fast correction of symptomatic hyponatremia by 4-6 mEq/L in the first few hours usually will lead to cessation of symptoms, mainly seizure (Sterns et al., 2009). The brain cell adaptation will take place within 24-48 hours however the actual and real duration is unknown specifically (Sterns, 2018).

Overcorrection of chronic hyponatremia can lead to ODS, and the risk factors are serum sodium level of <105 mEq/L, concomitant hypokalemia, alcoholism, malnutrition and chronic liver disease. The rate of sodium correction is varied among the studies, as to be limited to 4-6 mEq/L/day (Sterns, 2018; Adrogué and Madias, 2012), maximumly to 8 mEq/L/day (Sterns, 2015; Arampatzis et al., 2012; Sood et al., 2013) and not

exceeding 18 mEq/L in 48 hours (Braun et al., 2015; Pfennig and Slovis, 2012; Spasovsk et al., 2014).

In our patient who presented with serious neurological symptoms, including seizure and impaired consciousness, the aim was to rapidly raise the sodium level to stop the symptoms and for that hypertonic saline was used, and then to maintain the sodium level within the acceptable target of correction in 48 hours. Initially, sodium level raised by 3 mEq/L in the first 4-6 hours however he was still confused at which another bolus of hypertonic saline was given, then sodium level raised up and overcorrected by 14 mEq/L in the first 9 hours with devolving of polyuria that was managed with hypotonic saline and desmopressin. After these measures, the sodium level maintained at 116 mEq/L in the first 24 hours and 115 mEq/L in the first 48 hours, meaning that it corrected by 15 mEq/L within 48 hours.

Our idea was to keep the Na level almost within the desire target in the first 48 hours in respect to the time of brain adaptation to avoid ODS particularly in this patient who has multiple risk factors. Although we achieved our goal in term of correction, the patient unfortunately developed ODS that evident by MRI brain.

The possible reasons of having ODS in this case despite appropriate sodium correction, first that he was a high-risk patient given the initial sodium level of 99 mEq/L, being alcoholic and malnourished; secondly, the overcorrection happened in the first 24 hours by 14 mEq/L as it has been reported that ODS can occur with sodium correction of 9 mEq/L/day (Sterns, 2015; Arampatzis et al., 2012; Sood et al., 2013; Sterns, 2018). Furthermore, that we allowed for sodium autocorrection after 48 hours and also it has been reported that overcorrection in any day, not only in the first 2 days, might lead to ODS (Sterns, 2015; Arampatzis et al., 2012; Sood et al., 2013; Sterns, 2018).

CONCLUSION

Managing patients with symptomatic severe hyponatremia is really challenging as it needs to balance controlling the serious symptoms and to avoid overcorrection, moreover, it seems that limiting the correction to 4-6 mEq/L/day and to maintain that rate of correction till the sodium reached an acceptable level is a safe practice to avoid serious complications like ODS.

DISCLOSURE

The authors declared no conflicts of interest. No financial support. Informed consent was obtained from the patient's brother.

REFERENCES

Adrogué HJ, Madias NE (2012). The challenge of hyponatremia. J. Ame. Soc. Nephrol. 23(7):1140-8.

Aegisdottir H, Cooray C, Wirdefeldt K, Piehl F, Sveinsson O (2019). Incidence of osmotic demyelination syndrome in Sweden: A nationwide study. Acta Neurologica Scandinavica;140(5):342-9.

Alleman AM (2014). Osmotic demyelination syndrome: Central pontine myelinolysis and extrapontine myelinolysis. Seminars in Ultrasound, CT and MRI;35(2):153-9.

Arampatzis S, Frauchiger B, Fiedler GM, Leichtle AB, Buhl D, Schwarz C, *et al*. (2012). Characteristics, symptoms, and outcome of severe dysnatremias present on hospital admission. Am J Med;125:1125.e1-7.

Braun MM, Barstow CH, Pyzocha NJ (2015). Diagnosis and management of sodium disorders: hyponatremia and hypernatremia. Am Fam Physician;91(5):299-307.

Gankam KF, Decaux G (2018). Hyponatremia and the Brain. Kidney International Reports;3(1):24-35.

George JC, Zafar W, Dan Bucaloiu I, Chang AR (2018). Risk factors and outcomes of rapid correction of severe hyponatremia. Clin. J. Ame. Soc. Nephrol. 13(7):984-92.

Pfennig CL, Slovis CM (2012). Sodium disorders in the emergency department: a review of hyponatremia and hypernatremia. Emerg Med Pract;14(10):1-26.

Sood L, Sterns RH, Hix JK, Silver SM, Chen L (2013). Hypertonic saline and desmopressin: A simple strategy for safe correction of severe hyponatremia. Am J Kidney Dis; 61:571–8.

Spasovski G, Vanholder R, Allolio B, Annane D, Ball S, Bichet D, *et al*. (2014). Hyponatraemia Guideline Development Group. Clinical practice guideline on diagnosis and treatment of hyponatraemia. Eur. J. Endocrinol; 170(3):G1-47.

Sterns RH (2015). Disorders of plasma sodium–causes, consequences, and correction. N Engl J Med; 372:55–65.

Sterns RH (2018). Treatment of severe hyponatremia. Clin. J. Ame. Soc. Nephrol. 13(4):641-9.

Sterns RH, Nigwekar SU, Hix JK (2009). The treatment of hyponatremia. Semin Nephrol; 29: 282–99.

Verbalis JG, Goldsmith SR, Greenberg A, Korzelius C, Schrier RW, Sterns RH, *et al*. (2013). Diagnosis, evaluation, and treatment of hyponatremia: expert panel recommendations. Am J Med;126(10 Suppl 1):S1-42.