International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2019): 7.583

Treatment and Outcome Severe Symptomatic Hyponatremia: Case Report

I Made Adi Narendranatha Komara[1], Ketut Suryana[2]

[1, 2]Department of Internal Medicine, Wangaya Hospital, Denpasar, Bali, Indonesia
[1]adinarendra40[at]gmail.com
[2]ketutsuryana[at]gmail.com

Abstract: ***Hyponatremia is one of the most common electrolyte abnormalities encountered in clinical practice. It is not a disease but rather a pathophysiologic process indicating disturbed water homeostasis. Severe hyponatremia is often defined as PNa level under 120 mmol/L and may lead to seizures, obtundation, coma, and respiratory arrest, it is of paramount importance for the clinician to be aware of the various scenarios in which hyponatremic patients can present and tailor the management strategies accordingly. We present here a case of severe hyponatremia of unknown duration with the presenting plasma sodium level of 94 mmol/L and use it to illustrate the various treatment strategies.***

Keywords: hyponatremia, sodium level, vomiting

1. Introduction

Hyponatremia, defined as a plasma sodium concentration (PNa) <135 mmol/L, is one of the most common electrolyte abnormalities encountered in clinical practice, occurring in 15%-20% of hospitalized patients, in up to 30% of all patients treated in intensive care units, and has been associated with considerable morbidity and mortality. [1,2] Hyponatremia is not a disease but rather a pathophysiologic process indicating disturbed water homeostasis. The most common causes of hyponatremia are the syndrome of inappropriate antidiuresis (SIAD), diuretic use, polydipsia, adrenal insufficiency, hypovolemia, heart failure, and liver cirrhosis (the latter two are often collectively referred to as "hypervolemic hyponatremia").[3]

Severe hyponatremia is often defined as PNa level under 120 mmol/L and may lead to seizures, obtundation, coma, and respiratory arrest. It can be acute or chronic and manifest with mild or more severe symptoms such as confusion, nausea, vomiting until coma.[2] When this occurs within 48 h, it is called acute hyponatremia and delay in management may lead to cerebral edema and brain herniation. On the other hand, overly rapid correction of chronic severe hyponatremia, defined as hyponatremia occurring over more than 48 h can lead to catastrophic complications such as osmotic demyelination syndrome (ODS) and even death. When the onset of hyponatremia is uncertain, it should be managed as chronic hyponatremia as it would be reasonable to err on the side of caution during the treatment of such cases. [4] We present here a case of severe symptomatic hyponatremia with a PNa of 94 mmol/L at the time of presentation to hospital, which need an appropriate management can prevent this complication.

2. Case report

A 74-year-old Caucasian female with past medical history of hyponatremia 1 year ago (the same as the symptoms experienced now) and pneumonia. She had nausea and more than three episodes of vomiting a day for four days, accompanied by abdominal pain and decreased appetite. This patient was referred from another hospital. When she arrived at the hospital, attached 3 lpm oxygen canules to the patient, 2 lines of intravenous fluid drops, line I (normal saline 3% 15 gtts / min) and line II (NaCl 0.9% with 50 mcq KCl 8 gtts / min). She appeared weak and restless with GCS E3V4M5.

Her vitals on presentation were as follows: blood pressure of 180/90, heart rate of 80/min, temperature of 36°C, and respiratory rate of 20/min. On examination, she was moved to localized pain dan said inappropriate words when checked for consciousness. Her pupils were equal, round, and reactive to light. Her cardiorespiratory was normal, tenderness in the field of the stomach on abdominal exam and she did not have any edema.

Laboratory tests performed upon admission showed leukocytosis (15.16 × 10^3/uL), hematocryte (35.5%), high SGOT (96 U/L), increased levels creatinine (1.4 mg/dL), and analysis of arterial blood showed hyponatremia with a sodium level (94 mmol/L)below 100 mmol/L, calium (2.8 mmol/L), cloride (75 mmol/L). SARS-CoV-2 test is non reactive.

The patient diagnosed with severe hyponatremia with hypertension, are treated together with a cardiologist. The therapy was started, including IVFD normal saline 3% 15 gtts/mins, ondansentron 8 mg every 12 h, pantoprazole 40 mg in 24 h, ceftriaxone 1 gr every 12 h, sucralfat 1 tbls every 8 h, candesartan 8 mg every 12 h, and bisoprolol 2.5 mg in 24 h. Monitoring and follow-up is performed on the patient for several days until symptoms improve and sodium levels stabilize.
Day 1: no abdominal pain, sodium 109 mmol/L; calium 4.0 mmol/L; cloride 72 mmol/L
Day 2: appetite began to exist, sodium 111 mmol/L; calium 3.0 mmol/L; cloride 74 mmol/L, IVFD normal saline 3% changed into IVFD NaCl 0.9% 20 gtts/mins
Day 3: consciousness compos mentis, sodium 122 mmol/L; calium 2.9 mmol/L; cloride 92 mmol/L

Day 4: sodium 128 mmol/L; calium 3.1 mmol/L; cloride 93 mmol/L

3. Discussion

Hyponatremia is one of the most common electrolyte disorders in clinical practice. It is defined as a serum sodium concentration <135 mmol/L and is considered severe when the serum level <125 mmol/L. Symptoms of hyponatremia are primarily neurologic, ranging from nausea, vomiting, headache and malaise, to altered mental status, seizures, confusion even coma, brain-stem herniation, and cardiorespiratory distress, which can lead to permanent brain damage or death.[2,5] In our patient, the sodium level was 94 mmol/L at admission, but surprisingly, the patient did not show any other neurological symptoms apart from decreased consciousness. Low serum sodium levels without CNS symptoms have not been previously reported.

The severity of hyponatremia depends on the rate and extent of changes in sodium concentration. In chronic hyponatremia, neurologic symptoms are much less severe due to cerebral adaption which begins within a day of sustained hyponatremia and generally takes several days for full measures to be in place. The adaptive mechanism involves (a) a compensatory displacement of fluid from the interstitial space into the cerebrospinal fluid and from there into the systemic circulation and (b) the extrusion of intracellular solutes together with osmotically obligated water to reduce cellular swelling and normalize brain volume. The process of brain volume regulation is complex and essential to understand the variability of the clinical presentation of hyponatremia.[5]

The first cause of hyponatremia in this case was vomiting and the patient's health was threatened by the cause of vomiting for 4 days with a frequency of more than 3 times a day as a potential hyponatremia. The first line to treat hyponatremia is intravenous fluids. Besides that, therapy was given for symptomatic symptoms such as in this case, namely the patient experienced nausea and vomiting, then given ondansentron, pantoprazole, and sucralfat. Administration of ceftriaxone to patients as prophylactic therapy in inpatients at the hospital.[3,4] The hypertension experienced by the patient in this case was stage 2 hypertension, therapy was given the Angiostenin II receptor blockers (ARB) such as candesartan and beta blockers such as bisoprolol in accordance with hypertension guidelines.[6]

Correction of hyponatremia with close monitoring of PNa levels to avoid over-correction is an important management of hyponatremia with objective focus and limitation of plasma sodium correction.[4,7]The improvement in plasma sodium concentration in this patient in this case from PNa 94 mmol/L to 109 mmol/L in the first 24 hours (day 1) and 111 mmol/L in 48 hours (day 2) are in accordance with the theoretical basis and existing studies where the limits are <10-12 mmol/L in 24 hours and <18 mmol/L within 48 hours have been recommended by US and European guidelines published in 2013 and 2014 respectively.[1,8] These results are also consistent with the case conducted by Ayus et al. (2015), correction to 12 to 14 mmol / L in the first 24 to 48 hours was found to be safe without evidence of osmotic demyelination syndrome (ODS).[9] Some researchers have proposed a more conservative limit for plasma sodium concentration of 6 to 8 mmol/L per day.[10] Adequate hyponatremia treatment in our patient led to recovery without consequences. Gradually increasing the sodium concentration, as was done in this case, results in a gradual but complete recovery without any complications due to an appropriate increase in serum sodium.[11]

4. Conclusion

Accurate diagnosis of the cause, pathogenesis and chronicity, and monitoring during treatment are the critical parts of the management of severe hyponatremias. The treatment of severe hyponatremia requires a methodical approach with close monitoring of PNa levels for appropriate rates of correction. Although there are disagreements on the optimal goals and limits of correction of plasma sodium, recommended limits of correction is an increase of < 10–12 mmol/L in the first 24 h and < 18 mmol/L in the first 48 h per US and European guidelines on hyponatremia management.

References

[1] Spasovski G, Vanholder R, Allolio B, et al. Clinical guideline on diagnosis and treatment of hyponatraemia. Nephrol Dial Transplant. 2014;29:2.
[2] Icin T, Medic-Stojanoska M, Kuzmanovic V, et al. Multiple causes of hyponatremia: A case report. Med Princ Pract. 2017;26.
[3] Hoorn EJ, Zietse R. Diagnosis and treatment of hyponatremia: Complilation of the guidelines. J Am Soc Nephrol. 2017;28.
[4] Tandukar S, Rondon-Berrios H. Treatment of severe symptomatic hyponatremia. Physiol Rep. 2019;7(21).
[5] Zhou N, Yang C. A case of extreme hyponatremia without neurologic symptoms. Clin Case Rep. 2019;7(10).
[6] Urger T, Borghi C, Charchar F, et al. 2020 International Society of Hypertension Global Hypertension Practice Guidelines. Hypertension. 2020;75.
[7] Sahay M, Sahay R. Hyponatremia: A practical approach. Indian Journal of Endrocrinology and Metabolism. 2014;18(6).
[8] Verbalis JG, Goldsmith SR, Greenberg A, et al. Am J Med. 2007;120:11.
[9] Ayus JC, Caputo D, Bazerque F, et al. Treatment of hyponatremic encephalopathy with a 3% sodium chloride protocol: a case series. Am J Kidney Dis. 2015;65.
[10] Adrogue HJ, Madias NE. Diagnosis and treatment of hyponatremia. Am J Kidney Dis. 2014;64.
[11] Tzamaloukas AH, Malhotra D, Rosen BH, et al. Principles of management of severe hyponatremia. JAHA. 2013;2(1).