Journal of Clinical Case Reports

Case Report Open Access

An Interesting Case of Beer Potomania

Fenando A*

Department of Internal Medicine, Michigan State University/Sparrow Hospital, Lansing, Michigan, USA

Abstract

Managing the sodium level in the beer potomania patient is so challenging. Every 5 to 6 people with beer potomania develop ODS during treatment. Close monitoring on the sodium level with severe hyponatremia occurs commonly in patient with alcoholism. Many pathological mechanisms contribute to this condition, one of the most unique mechanism presents in beer potomania. The beer does not contain enough sodium but contains enough calories to prevent protein break down. This will result lower urine osmolality due to low urea and sodium in the urine. Daily heavy beer drinker will have maximally diluted urine, hence the total volume of urine will be dependent only on the solute intake. Even little increment in solute intakes could reverse this mechanism and produce rapid elevation in serum sodium level. With that being said, correction modest correction of serum sodium are the main principle of the treatment. D5W or desmopressin or both is frequently needed to slow down the sodium progression.

Keywords: Desmopressin; Diagnosis; Treatment; Hyponatremia

Introduction

We present a 46-year-old Caucasian male with recently increased daily beer consumption with poor oral intake. He presented with altered mental status and possible seizure with a really low sodium level. Our team diagnosed him with beer potomania and treatment of hypertonic saline was started. He developed a rapid increase in sodium level subsequently. D5W and desmopressin were finally given to slow down the increment. Even the sodium level was elevated for more than 10 mmol/L in the first 24 hours, our patients did not develop any new neurologic deficit [1-3].

Case Description

A 46-year-old Caucasian male with a past medical history of alcohol abuse, hypertension, anxiety, and depression initially brought to the hospital by Emergency Medical Services (EMS) after he was found down unconscious on the floor by his family. He recently lost his job after he was diagnosed by cervical radiculopathy with left-hand weakness due to trauma. He had been drinking more beer and had poor oral intake over the past few weeks. He could drink up to 20 cans of beer per day which is 10 to 15 cans more than his regular habit. During the presentation in the emergency department, he was stupor with Glasgow Coma Scale (GCS) of 8 and then subsequently intubated for airway protection. He had stable vital signs with blood pressure 143/79 mmHg, pulse rate 72, respiratory rate 17, temperature 98.4 and oxygen saturation 99%. Physical exams were significant for tongue laceration and weakness in the left upper limb upon pain stimulation. He did not appear intravascular volume depleted.

Diagnostics

The patient's laboratory data showed haemoglobin of 12.6 g/dl, white blood cells 7.1 × 1000/mcL, platelets 177 × 1000/mcL, calcium 7.6 mg/dl, albumin 4.4 g/dl, blood urea nitrogen <5 mg/dl, creatinine 0.36 mg/dl, glucose 112 mg/dl, aspartate transaminase 247 U/L, alanine transaminase 27 U/L, magnesium 1.5 mg/dl, sodium 93 mEq/L, potassium 3.5 mEq/L, chloride 60 mEq/L, CO_2 20 meq/L, serum ethanol 210 mg/dl, thyroid stimulating hormone 0.38 mcIU/ml, serum osmolality 250 mOsm/kg, urine sodium <10 mEq/L, urine osmolality 55 mOsm/kg. A computed tomography scan of his head without intravenous contrast revealed right subdural hemorrhage (SDH) with 4 mm of mild subfalcine herniation from right-to-left.

Differential diagnosis

In our patient's case, an initial broad differential diagnosis was suspected. Syndrome of Inappropriate Antidiuretic Hormone (SIADH), Cirrhosis, Primary Polydipsia, Beer potomania could potentially contribute to low sodium level. Due to a history of drinking 20 cans of beer/day lately, poor oral intake, very low serum osmolality and neutral volume status during the clinical assessment, we came to a conclusion that beer potomania was the culprit of the very low sodium level in this patient.

Treatment

Saline 3% was initiated due to a concern of a seizure episode in the setting of hyponatremia. The patient received 2 times bolus of 100 ml of Saline 3% intravenously then was transferred to the Intensive Care Unit (ICU) with monitoring of sodium level every two hours. A brisk diuresis and rapidly elevated sodium level in this patient triggered initiation of D5W to slow down the risen of sodium level to prevent ODS. In the first 6 hours, the sodium level increased by 15 mmol/L and patient had polyuria with urine output of around 600 ml per hour. D5W was initiated to slow down the increment of the sodium level. In the next 2 hours, the sodium level kept on increasing to 6 mmol/L despite

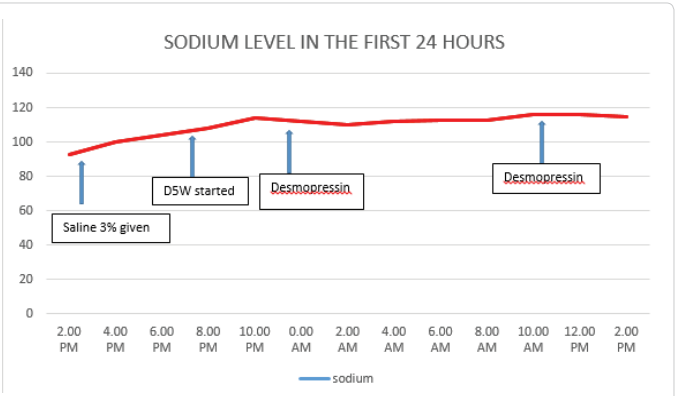

Figure 1: Sodium levels in the first 24 hrs intravenous doses of desmopressin.

***Corresponding author:** Fenando A, Department of Internal Medicine, Michigan State University/Sparrow Hospital, Lansing, Michigan, USA, Tel: + 5174322404; E-mail: Fenandoa@msu.edu

Received July 02, 2019; **Accepted** July 12, 2019; **Published** July 19, 2019

Citation: Fenando A (2019) An Interesting Case of Beer Potomania. J Clin Case Rep 9: 1261.

being on D5W 167 ml/hour thus desmopressin 1 mcq bolus was given. In the first 24 hours, sodium level was maintained around 112 mmol/l with D5W and 2 intravenous doses of desmopressin 1 mcq (Figure 1).

Outcome and follow-up

Patient's sodium level improved gradually over the next few days and finally returned to normal level on the 12th day of hospitalization. Patient had to stay in ICU for 7 days before being transferred to the regular unit. His ICU stay was complicated by alcohol withdrawal and aspiration pneumonia. Regarding his SDH, no intervention was done because his neurologic status had been improving gradually and repeat CT scan showed a stable result from the previous one. He was back to his normal mental status and later discharged in a stable condition.

Discussion

Hyponatremia is commonly seen in chronic alcoholic patients. Multiple pathologic mechanisms contribute to this condition. In one prospective study of 241 chronic alcoholic patients, 22 patients have hyponatremia with 54.5% due to hypovolemia, 27.3% due to pseudohyponatremia (high triglyceride level), 9.1% due to cerebral salt wasting and reset osmostat syndrome [1].

The first study of beer potomania was conducted in 1972 after several report studies on beer drinker patients admitted with hyponatremia. The subjects were patients with a regular diet (no fluid/salt restriction). They were given ten 16-ounce cans of beer containing alcohol 4.6 percent by volume and sodium 16 mg/L over 16 hours of every day for seven days. This study showed that there was a progressive decrease in serum sodium and chloride paralleled with the falling of serum osmolality, increased in body weight and strongly positive in fluid balance [4].

To understand the unique mechanism of beer potomania, we have to know the mechanisms below. In a normal healthy person, kidneys can produce urine with osmolality ranging from 50 to 1200 mOsm/kg H_2O with corresponding urine volume as little as 0.5 L to 18 L per day without becoming hyponatremic [5]. We all know the fact that as urinary osmolality decreases free water clearance proportionally increases, but with the same level of urine osmolality the free water clearance is dependent on the solute intake. The higher the solute intake is, the more free water that will be excreted. For example in a person with urine osmolality of 60 mOsm/kg, 12 L of free water will be excreted if the solute intake is 900 mOsm/day. On the other hand, only 4 L of free water can be excreted with only 300 mOsm/day of solute intake [3,6].

Treatment of hyponatremia from beer potomania is challenging as these patients have a low Antidiuretic Hormone (ADH) state which could produce dramatic response from the correction. ODS is mostly caused by the overcorrection of hyponatremia, which is defined as the increment of sodium level of 10 mmol/L in 24 hours, 18 mmol/L in 48 hours, and 20 mmol/L in 72 hours. In a literature done by Sanghvi et al, they recommend using normal saline even in symptomatic patients and just fluid restriction in asymptomatic patients due to the risk of rapidly increasing sodium level. D5W and desmopressin should be used to prevent sodium level from keep rising [3].

In our patient, daily consumption of 20 cans of 12-oz beers (per can contains 14 mg of sodium and 96 mg of potassium) will produce 61 mOsm [(20 × 14/23) + (20 × 96/39)]. These solutes will be added to urea and other solutes produced by proteins breakdown from his body. The beer has enough calories that will prevent proteins break down. So our patient will roughly excrete 300 mOsm/day of urine. Without good oral intake, the amount of fluid produced in maximally diluted urine per day only 4 L (6). Water intake greater than 5 L (or 14 cans of beer) results in hyponatremia [3].

ODS is not a rare event amongst beer potomania patients. From a review of the literature of 22 cases of beer potomania patients, they found that 18% of patients presenting with beer potomania developed ODS [3]. Unresponsive to D5W and a brisk diuresis in a patient with SDH warranted us to start desmopressin. Although it is a normal response from beer potomania patients to have a diuresis after initiation of solute, we also considered the possibility of having central diabetes insipidus in this setting. Based on our clinical judgment we started desmopressin and finally were able to slow down the sodium progression. Despite the sodium level increased rapidly, this patient did not develop ODS and no new neurologic symptoms during follow up in the outpatient clinic few months later.

Conclusions

Following conclusions can be drawn from the above case discussion:

1. Beer potomania has unique pathophysiology that resulting hyponatremia. Low sodium content with enough calories would produce low sodium and urea in the urine. These will make our urine maximally diluted and the solute intake as the only dependent factor for urine volume.
2. Most of beer potomania patients have baseline low ADH state which could produce a dramatic response to sodium correction. Hypertonic saline is often not needed for correction of sodium even in symptomatic patients. Aggressive monitoring and effort to re-lower sodium during correction could be pivotal in preventing ODS.
3. ODS is not an uncommon in beer potomania patients. 18% of patients of 22 reviewed cases of beer potomania patients develop this devastating complication. It usually appears 2-3 days after the change of sodium level. Rate of correction of less than 10 mmol/L in 24 hours, 18 mmol/L in 48 hours, and 20 mmol/L in 72 hours are preferred to prevent this complication.

References

1. Berl T (2008) Impact of solute intake on urine flow and water excretion. J Am Soc Nephrol 19: 1076-1078.
2. Kujubu DA, Khosraviani A (2015) Beer potomania: An unusual cause of hyponatremia. Permanente J 19: 74.
3. Sanghvi SR, Kellerman PS, Nanovic L (2007) Beer potomania: An unusual cause of hyponatremia at high risk of complications from rapid correction. Am J Kidney Dis 50: 673-680.
4. Gwinup G, Chelvam R, Jabola R (1972) Beer drinkers hyponatremia: An inappropriate concentration of the urine during ingestion of beer. Calif Med 116: 78-81.
5. Koeppen BM, Stanton BA (2013) Regulation of body fluid osmolality: Regulation of water balance. Renal Physiol 79.
6. Lodhi MU, Saleem TS, Kuzel AR, Khan D, Syed IA, et al. (2017) Beer Potomania: A syndrome of severe hyponatremia with unique pathophysiology: Case studies and literature review. Cureus 9: 1.