CLINICAL VIGNETTE

Beer Potomania: A Cause of Severe Hyponatremia

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Case Presentation

A 59-year-old male with alcohol use disorder presented to the emergency department with facial trauma. He reported that he was carrying a case of beer when he fell onto the ground, hitting his face. He denied loss of consciousness or preceding lightheadedness, chest pain, or palpitations. He reported chronic nausea for over a month and denied headache, mental status changes, or seizures. He had a significant history of alcohol consumption, drinking up to twenty beers per day for over thirty years. He reported little oral intake including water intake, occasionally not eating any food for several days. He quit smoking one month ago and denied any illicit drug use. He had never seen a physician before and denied any other prior medical history.

On presentation, he was afebrile, normotensive, had a heart rate in the 90s, and was breathing comfortably on room air with SpO2 98%. He appeared intoxicated. He was only oriented to person but otherwise had a normal neurological exam. He was euvolemic with no jugular venous distention, pulmonary crackles, or lower extremity edema. Labs were remarkable for severe hyponatremia with a sodium of 100 mmol/L, confirmed on a repeat measurement. His basic metabolic panel was also notable for chloride of 64 mmol/L and creatinine of 0.84 mg/dL. Thyroid stimulating hormone (TSH) was 1.3 µIU/mL. Serum osmolality was 259 mOsm/kg, Urine osmolality was 146 mOsm/kg and urine sodium was <20 mmol/L. Computed tomography (CT) of the brain was negative for any acute intracranial pathology.

One liter of normal saline was initially ordered but only two hundred milliliters was given before it was discontinued when the initial sodium level resulted. Because he was asymptomatic, his hyponatremia was thought to be chronic. He was diagnosed with beer potomania, given his history of heavy beer intake, poor dietary intake, and hyponatremia in the absence of other known causes. He was admitted and made NPO for the first day followed by fluid restriction with strict urine output measurements and sodium checks every two hours. He intermittently received dextrose 5% in water (D5W) infusions according to the change in serum sodium level to prevent rapid autocorrection of the serum sodium level with risk for osmotic demyelination syndrome (ODS). His serum sodium increased to 109 mmol/L after 24 hours, 117 mmol/L after 48 hours, 121 mmol/L after 72 hours, and 127 mmol/L after 96 hours. It then remained stable between 127-131 mmol/L for three more days without the development of any neurological signs or symptoms concerning for ODS.

Discussion

Beer potomania is an infrequent cause of hyponatremia. This syndrome is characterized by hyponatremia with excessive beer ingestion, protein malnutrition, and no evidence of other causes of hyponatremia.[1] Our patient consumed up to twenty beers in a day with very little other oral intake and presented with a sodium level of 100 mmol/L. He denied excessive water intake, appeared euvolemic, had a normal TSH, and had urine studies not consistent with SIADH, making beer potomania the likely diagnosis. Additionally, the low serum osmolality, urine osmolality, and urine sodium levels are consistent with beer potomania. Beer potomania typically causes a severe hyponatremia, defined as a serum sodium level below 120 mmol/L. One review reported twenty-two published cases of beer potomania, had mean initial sodium level of 108 mmol/L.[2]

Understanding the pathophysiology of beer potomania is necessary to appropriately manage the hyponatremia. The ability of the kidney to excrete free water depends on solute excretion and urinary diluting capacity.[3] In patients with normal renal function, excessive water intake does not result in hyponatremia because of the kidney's ability to excrete large amounts of free water. Beer has very little protein and sodium content, so only a small amount of osmoles is generated that can be excreted by the kidneys. This low solute excretion in turn impairs the maximal free water excretion of the kidneys, so a high volume of the free water in beer is retained. This results in hyponatremia and a low antidiuretic hormone (ADH) state, which is unlike that seen in most other causes of hyponatremia. When these patients are presented with solute in this low ADH state, they undergo a rapid diuresis that can rapidly increases the serum sodium concentration.

Serum sodium levels in beer potomania should be judiciously corrected to prevent ODS. ODS is caused by the rapid rise in serum tonicity following treatment of hyponatremia, which leads to osmotic stress on oligodendrocytes and presents as paralysis, dysphagia, dysarthria, and other neurological symptoms. Patients who have hyponatremia due to beer potomania are at a higher risk of developing ODS because of the chronicity of the hyponatremia, the severity of the hyponatremia, and the likelihood of rapid correction because of the underlying

pathophysiological state.[4] One literature review, reports ODS in four of twenty-two patients with beer potomania (18%).[5] The treatment goal in these patients should be a serum sodium level increase less than 10 mEq/L in the first 24 hours and less than 18 mEq/L in the first 48 hours. Patients should be monitored in the intensive care unit with frequent monitoring of sodium levels with cautious initiation of oral intake with restriction of fluid intake.[5] If the serum sodium level increase occurs at a higher than desired rate, a D5W infusion should be started to match the urine output.

Conclusion

Beer potomania is an infrequent cause of hyponatremia but typically causes severely low sodium levels. These patients are at high risk of ODS due to rapid sodium correction and require very careful correction during treatment.

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