Beer Potomania—An Unusual Cause of Hyponatremia

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ABSTRACT

The first case of severe hyponatremia, since referred to as beer potomania, in a heavy beer drinker patient was reported in 1972. Electrolyte abnormalities are common findings in patients with a history of heavy alcohol use. Excessive consumption of beer in particular, which has a low solute content (sodium concentration, 1.8 mEq/L and potassium concentration, 7.2 mEq/L), to the exclusion of other solute intake may result in severe hyponatremia. We report a case of severe hyponatremia that occurred in a patient who, owing to his underlying colon cancer, was drinking beer and ingesting little other food. His hyponatremia improved with increased solute intake and, upon correction of his serum sodium, he had no subsequent neurologic sequelae.

INTRODUCTION

The first case of severe hyponatremia in a heavy beer drinker patient was reported in 1972 by Gwinup et al.[1] This condition has since been referred to as beer potomania. Electrolyte abnormalities are common findings in patients with a history of heavy alcohol use. In the study by Liamis et al,[2] among hospitalized patients with history of chronic alcohol consumption, 17.3% had hyponatremia. The excessive consumption of beer in particular, which has a low solute content (sodium concentration, 1.8 mEq/L; potassium concentration, 7.2 mEq/L), to the exclusion of other solute intake may result in severe hyponatremia. We report a case of severe hyponatremia that occurred in a patient who, owing to his underlying colon cancer, was drinking beer and ingesting little other food. His hyponatremia improved with increased solute intake and, upon correction of his serum sodium, he had no subsequent neurologic sequelae.

CASE PRESENTATION

A Hispanic man, age 84 years, with history of chronic alcohol abuse and recently diagnosed stage IV sigmoid adenocarcinoma presented to the Emergency Department with nausea, weakness, decreased appetite, and abdominal pain for the past 3 to 4 days. The patient had been ingesting approximately 12 cans of beer daily in addition to his usual diet for the preceding 50 years, but during the week before his presentation, because of worsening abdominal pain, he was drinking his habitual quantities of beer but otherwise eating minimally. On physical examination, his temperature was 37.1°C, blood pressure 142/81 mmHg, pulse rate 78 beats/min, without orthostatic changes, respiratory rate 18 breaths/min, and oxygen saturation 97% on room air. He was in no acute distress and did not appear intravascularly volume depleted. His lungs were clear bilaterally, and his heart sounds were normal without murmurs. He had tenderness in the epigastric area without rebound tenderness, with normal bowel sounds and no organomegaly. He was oriented and coherent in conversation. His neurologic exam and deep tendon reflexes were normal. He had no tremors.

The patient's laboratory data showed white blood cells 15.4 × 1000/mcL, hemoglobin 12.7 g/dL, platelets 347 × 1000/mcL, international normalized ratio 1.1, glucose 160 mg/dL, sodium 116 mEq/L, potassium 4.1 mEq/L, chloride 85 mEq/L, CO_2 19 mEq/L, blood urea nitrogen 6 mg/dL, creatinine 0.58 mg/dL, serum osmolality 250 mOsm/kg, uric acid 3 mg/dL, phosphorus 2.8 mg/dL, calcium 8.3 mg/dL, magnesium 1.1 mg/dL, alanine transaminase 20 U/L, aspartate transaminase 27 U/L, total bilirubin 0.8 mg/dL, thyroid stimulating hormone 2.19 mcIU/ml, albumin 2.8 g/dL, and cortisol 23.8 mcg/dL. His urinalysis showed specific gravity 1.005, pH 6, negative leukocyte esterase, negative nitrite, white blood cells 0-2, red blood cells 0-3, urine sodium 35 mEq/L, urine chloride 37 mEq/L, urine potassium 11 mEq/L, urine creatinine 26 mg/dL, and urine osmolality 182 mOsm/kg. A computed tomography scan of his abdomen and pelvis with intravenous contrast revealed a new abscess adjacent to the previously seen sigmoid carcinoma with local lymphadenopathy and hepatic metastatic disease.

The patient received 1 L of 0.9% sodium chloride intravenously in the Emergency Department and was started on antibiotics for the sigmoid abscess. The surgical consultant did not recommend surgical intervention. He received intravenous thiamine and magnesium supplementation for his history of heavy alcohol drinking and hypomagnesemia. He was encouraged to increase his oral intake with a normal diet as tolerated. He subsequently underwent a brisk diuresis of approximately 1.8 L during the first 8 hours. His intravenous fluid was promptly discontinued and the patient's serum sodium was checked every 2 to 3 hours to monitor for overly rapid correction. His serum sodium increased by 8 mEq/L in the first 24 hours and 14 mEq/L in the first 48 hours. His serum sodium remained between 130 mEq/L and 133 mEq/L

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Common Causes of Hyponatremia

Hyponatremia with high osmolality
- Hyperglycemia, mannitol infusion, advanced renal failure

Hyponatremia with normal osmolality
- Hyperlipidemia, paraproteinemia

Hyponatremia with low osmolality
- Hypovolemic hyponatremia
  - Volume depletion with sodium loss in excess of water
- Hypervolemic hyponatremia
  - Heart failure, cirrhosis, nephrotic syndrome
- Euvolemic hyponatremia
  - Syndrome of inappropriate antidiuretic hormone, reset osmostat
  - Hypothryoidism, glucocorticoid deficiency, renal failure
  - Primary polydipsia, beer potomania, low solute intake
  - Medications, including thiazide diuretics

throughout the remainder of his hospitalization. He was closely observed for any change in his neurologic status or signs and symptoms of acute alcohol withdrawal. None appeared.

DISCUSSION

The most common electrolyte abnormality seen in clinical practice is hyponatremia, which is also found in up to 30% of hospitalized patients.[3] In the study by Liamis et al,[2] hyponatremia was the third most common electrolyte abnormality detected in 127 hospitalized chronic alcoholic patients, with a prevalence of 17.3%. Traditionally, the evaluation of hyponatremia begins with the determination of serum osmolality followed by a clinical assessment of volume status (see Sidebar: Common Causes of Hyponatremia). Most clinical hyponatremia is associated with a low serum osmolality.

In patients with normal renal function, excessive water ingestion does not result in severe hyponatremia because of the kidneys' ability to excrete large amounts of free water, provided the daily dietary intake of solute is normal. For example, if the normal daily intake of solute is between 600 mOsm/day and 900 mOsm/day for an individual on a typical Western diet, and if the maximal urinary dilution capacity is 50 mOsm/kg, a person with normal kidney function would be able to excrete between 12 L to 18 L of free water daily without becoming hyponatremic. In contrast, if such a person were to have a daily intake of only 200 mOsm to 300 mOsm solute, his ability to excrete free water becomes limited to 4 L/day to 6 L/day provided he is able to dilute his urine to the same degree.[4] The intake of fluid in excess of this amount will result in a dilutional hyponatremia. Moreover, the ingestion of large volumes of fluid in these patients results in the washout of the medullary urea concentration gradient, reducing the kidneys' ability to produce maximally dilute urine.[5] When additional solute is provided, a brisk diuresis ensues, resulting in rapidly increasing serum sodium concentrations, necessitating careful, frequent monitoring as well as the administration of electrolyte-free fluids or even antidiuretic hormone analogues should the serum sodium rise by more than 8 mEq/24 hours to 10 mEq/24 hours.

Management Recommendations for Hyponatremia Caused by Beer Potomania

- Nothing by mouth except medications for 24 hours
- No intravenous fluids unless symptomatic
- Prescribe intravenous fluids in finite amounts if needed
- Intensive care status
- Check serum sodium every 2 hours
- Goals
  - Serum sodium increase < 10 mEq/L in first 24 hours
  - Serum sodium increase < 18 mEq/L in first 48 hours
- Reduce serum sodium levels if necessary
- Give any intravenous medications in sugar solution (5% dextrose in water)
- If caloric intake is needed, use intravenous sugar solution (5% dextrose in water)

Precipitating the osmotic demyelination syndrome (ODS) in chronically hyponatremic patients by infusing saline load is a real danger.[5] Case reports of ODS with rapid sodium correction in patients with beer potomania have been published. In a literature review done by Sanghvi et al,[6] of 22 patients with beer potomania, 4 (18%) had ODS. Although infusion of hypertonic saline classically has been associated with development of ODS, Karp and Laureno[7] suggest that the rapid correction of hyponatremia may occur even with infusion of normal saline or with fluid restriction alone, resulting in ODS. On the basis of the underlying pathophysiology of beer potomania, Sanghvi et al[6] provided clinical recommendations for the management of these patients (see Sidebar: Management Recommendations for Hyponatremia Caused by Beer Potomania).

Because of the potential for overly rapid correction of serum sodium resulting in irreversible neurologic consequences in these patients with beer potomania, intensive care and frequent, serial measurements of serum electrolytes are recommended.

Our patient's history, clinical euvolemic state, and prompt diuresis in response to 1 L of intravenous normal saline administered in the Emergency Department led us to suspect that he indeed had beer potomania. Our patient was consuming 12 cans (12 ounces each) of beer daily, which is approximately 4.3 L. According to the published content of beer, our patient's solute intake was approximately 30 mEq potassium, 7 mEq sodium, 150 g carbohydrate, and 20 g protein per day. If the metabolism of every 10 g of protein results in the generation of 50 mmoles of urea, we estimate that our patient's daily solute intake was in the range of 200 mOsm/day to 250 mOsm/day. Our patient was older so it was unlikely that he would be able to maximally dilute his urine to 50 mOsm/kg, because with aging the kidneys' ability to produce dilute urine declines.[8] The capacity is further limited by the washout of his medullary concentration gradient owing to excessive fluid intake. If the maximally dilute urine

our patient could produce was 75 mOsm/kg rather than 50 mOsm/L, with daily solute intake of 200 mOsm/day to 250 mOsm/day he would need to drink only in excess of 2.7 L to 3.3 L of fluid per day before he would become hyponatremic, which was well below the range of his intake.

After the initial correction of his hyponatremia during the first 48 hours, his serum sodium remained in the range of 130 mEq/L to 133 mEq/L for the rest of his hospitalization. His persistent hyponatremia was most probably because of an underlying syndrome of inappropriate antidiuretic hormone secretion from metastatic cancer or a reset osmostat owing to his poor nutritional state, malignant tumor, or alcoholism.[9,10] His underlying tendency toward hyponatremia prevented his serum sodium from overcorrecting while his solute intake increased and his water intoxication resolved. ❖

Disclosure Statement

The author(s) have no conflicts of interest to disclose.

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References

1. Gwinup G, Chelvam R, Jabola R, Meister L. Beer drinker's hyponatremia. Inappropriate concentration of the urine during ingestion of beer. Calif Med 1972 Mar;116(3):78-81.
2. Liamis GL, Milionis HJ, Rizos EC, Siamopoulos KC, Elisaf MS. Mechanisms of hyponatraemia in alcohol patients. Alcohol Alcohol 2000 Nov-Dec;35(6):612-6. DOI: http://dx.doi.org/10.1093/alcalc/35.6.612.
3. Upadhyay A, Jaber BL, Madias NE. Incidence and prevalence of hyponatremia. Am J Med 2006 Jul;119(7 Suppl 1):S30-5. DOI: http://dx.doi.org/10.1016/j.amjmed.2006.05.005.
4. Berl T. Impact of solute intake on urine flow and water excretion. J Am Soc Nephrol 2008 Jun;19(6):1076-8. DOI: http://dx.doi.org/10.1681/ASN.2007091042.
5. Kelly J, Wassif W, Mitchard J, Gardner WN. Severe hyponatraemia secondary to beer potomania complicated by central pontine myelinolysis. Int J Clin Pract 1998 Nov-Dec;52(8):585-7.
6. Sanghvi SR, Kellerman PS, Nanovic L. Beer potomania: an unusual cause of hyponatremia at high risk of complications from rapid correction. Am J Kidney Dis 2007 Oct;50(4):673-80. DOI: http://dx.doi.org/10.1053/j.ajkd.2007.07.015.
7. Karp BI, Laureno R. Pontine and extrapontine myelinolysis: a neurologic disorder following rapid correction of hyponatremia. Medicine (Baltimore) 1993 Nov;72(6):359-73. DOI: http://dx.doi.org/10.1097/00005792-199311000-00001.
8. Sands JM. Urinary concentration and dilution in the aging kidney. Semin Nephrol 2009 Nov;29(6):579-86. DOI: http://dx.doi.org/10.1016/j.semnephrol.2009.07.004.
9. Kahn T. Reset osmostat and salt and water retention in the course of severe hyponatremia. Medicine 2003 May;82(3):170-6. DOI: http://dx.doi.org/10.1097/01.md.0000076007.64510.15.
10. DeFronzo RA, Goldberg M, Agus ZS. Normal diluting capacity in hyponatremic patients. Reset osmostat or a variant of the syndrome of inappropriate antidiuretic hormone secretion. Ann Intern Med 1976 May;84(5):538-42. DOI: http://dx.doi.org/10.7326/0003-4819-84-5-538.

Hard Drinking

The effects of hard drinking are flatulence, loss of appetite, morning sickness, wasting of the flesh and strength, tremblings, pains of the stomach, cough, jaundice, dropsy, forgetfulness and inattention, giddiness, diarrhea, broken sleep.

— William Heberden, MD, 1710-1801, English physician