Severe Hyponatremia in a Schizophrenic Patient

Gifford Lum1*

CASE DESCRIPTION

A 63-year-old white man with a history of schizophrenia plus polysubstance and alcohol abuse was seen by his primary care physician during a routine visit, at which time he was found to have a serum sodium concentration of 113 mmol/L. The patient was contacted by phone and advised to return to Urgent Care for assessment, but he did not respond until 7 days later, at which time his serum sodium concentration was 106 mmol/L. He reported that he felt fine the day before but that he started feeling very lethargic that morning and had trouble “keeping his eyes open.” The patient was noted to be somnolent and confused. Table 1 summarizes the patient’s laboratory data on admission.

PATIENT FOLLOW-UP

The patient admitted to having drunk one-half to 1 bottle of beer on the day of admission—only beer and no more than 2 beers. He later admitted, however, to a higher beer consumption of up to 12 bottles a day. He did not recall whether he had breakfast on the morning of admission, and his nutritional history was inconsistent. His severe hyponatremia and history of excessive consumption of beer, a poor source of sodium and protein, coupled with his poor intake of food was consistent with the diagnosis of beer potomania. The patient was treated with 500 mL of normal saline (300 Osm/L). The patient’s serum sodium value increased to 116 mmol/L but then increased rapidly to 126 mmol/L over a 12-h period. The rapid overcorrection of sodium (10 mmol/L in the first 12 h) increased the risk of osmotic demyelination because complications have been noted if the serum sodium change is >0.55 mmol·L⁻¹·h⁻¹ (>10 mmol/L in 24 h and >18 mmol/L in 48 h). The goal of the management of hyponatremia should be a sodium increase of <10 mmol/L in the first 24 h or <18 mmol/L in the first 48 h. Dextrose (50 g/L) and desmopressin were administered, and the serum sodium concentration was measured every 2 h to adjust the rate of the 50-g/L dextrose infusion and to correct the rate of change in the sodium concentration. The patient’s serum sodium values stabilized at 126 mmol/L. He was counseled about heavy beer drinking and adequate food intake to prevent recurrent episodes of hyponatremia.

DISCUSSION

In the vast majority of cases, hyponatremia reflects an excess of water relative to sodium stores, which leads to dilution of the total body sodium (1). Decreases in serum sodium concentrations create an osmotic gradient between the extra- and intercellular fluid in cerebral cells that causes water to move into cells, which increases the intracellular volume, promotes tissue edema, and causes neurologic symptoms (1).

Chronic hyponatremia occurs when the serum sodium decreases over a period of ≥48 h. In this situation, the brain can compensate for the decrease in sodium concentration by adjusting the organic solutes in the brain to promote a loss of water and reduce brain edema (1, 2). Aggressive treatment of chronic hyponatremia may be associated with an increased risk of osmotic demyelination, which appears to be caused by the shrinkage of the brain, leading to demyelination of pontine and extrapontine neurons. That causes neurologic dysfunction, paralysis, coma, seizures, and, in severe cases, death (2).

Severe hyponatremia, defined as a serum sodium value <120 mmol/L, is a rare finding, and a review of 25 671 sodium requests at this medical center over a 3-month period found 73 sodium values <120 mmol/L (0.28%) in 12 patients. Because of the high morbidity and mortality associated with severe hyponatremia, it is important to determine its cause.
Causes of hyponatremia include extrarenal sodium loss in diarrhea, vomiting, pancreatitis, renal loss induced by diuretics, Addison disease, salt-wasting nephropathy, an increased volume of extracellular fluid in congestive heart failure, cirrhosis, nephrotic syndrome, acute or chronic renal failure, syndrome of inappropriate secretion of antidiuretic hormone (SIADH), hypothyroidism, and excess water intake, as well as vomiting, diarrhea, or diuretic therapy. A physical examination will establish the patient’s volume status. Serum sodium is measured to establish hyponatremia. Pseudohyponatremia, a laboratory artifact, can be caused by hyperlipidemia and hyperproteinemia, which displaces water to produce falsely low sodium values when it is measured with indirect diluted ion-selective electrode methods. Pseudohyponatremia may be circumvented by measuring sodium with a direct ion-selective electrode or by measuring serum osmolality (low in hyponatremia and normal in pseudohyponatremia). Urinary osmolality should be measured to determine the ability of the kidney to dilute the urine. The urine sodium concentration should be measured to assess renal sodium handling, because most patients with intravascular volume depletion (except those with renal sodium wasting) exhibit low sodium excretion.

This patient had clinically important hyponatremia and was found on admission to have a serum sodium value of 106 mmol/L. His low serum osmolality (245 mOsm/kg H2O) was consistent with hyponatremia but not with pseudohyponatremia. This patient had low urinary osmolality and a low sodium concentration, which is inconsistent with SIADH and hypovolemic hyponatremia. Given this patient’s clinical history of excessive beer intake, poor food intake, laboratory findings of severe hyponatremia and low serum and urine osmolalities, and low urinary sodium concentrations, the diagnosis of beer potomania was made.

In 1971 and 1972, beer drinker’s hyponatremia (beer potomania) was described in 7 patients with long histories of consuming at least 4 L of beer per day, sodium-deficient diets, low serum sodium values (98–107 mmol/L), unconsciousness, and seizures. One patient who ingested 6 to 12 16-oz cans of beer per day had a sodium value of 122 mmol/L. The electrolyte disturbances were attributed to possible inappropriate secretion of ADH. A later study described a specific “hypo-osmolality syndrome” in beer drinkers, and the authors attributed the hyponatremia to the consumption of beer, which is low in sodium, and poor intake of ordinary food, which led to reduced excretion of ordinary food, which led to reduced excretion of urine that is concentrated relative to serum or by ridding the body of excess water by producing urine that is dilute relative to serum. Hyponatremia most often involves relatively high concentrations of ADH compared with the prevailing serum osmolality. Causes include a decreased effective circulating volume or SIADH release. This particular case instead reflects excessive water intake relative to the body’s ability to eliminate it.

Evaluation includes an accurate clinical history to determine an obvious cause for the hyponatremia, such as vomiting, diarrhea, or diuretic therapy. A physical examination will establish the patient’s volume status. Serum sodium is measured to establish hyponatremia. Pseudohyponatremia, a laboratory artifact, can be caused by hyperlipidemia and hyperproteinemia, which displaces water to produce falsely low sodium values when it is measured with indirect diluted ion-selective electrode methods. Pseudohyponatremia may be circumvented by measuring sodium with a direct ion-selective electrode or by measuring serum osmolality (low in hyponatremia and normal in pseudohyponatremia). Urinary osmolality should be measured to determine the ability of the kidney to dilute the urine. The urine sodium concentration should be measured to assess renal sodium handling, because most patients with intravascular volume depletion (except those with renal sodium wasting) exhibit low sodium excretion.

This patient had clinically important hyponatremia and was found on admission to have a serum sodium value of 106 mmol/L. His low serum osmolality (245 mOsm/kg H2O) was consistent with hyponatremia but not with pseudohyponatremia. This patient had low urinary osmolality and a low sodium concentration, which is inconsistent with SIADH and hypovolemic hyponatremia. Given this patient’s clinical history of excessive beer intake, poor food intake, laboratory findings of severe hyponatremia and low serum and urine osmolalities, and low urinary sodium concentrations, the diagnosis of beer potomania was made.

In 1971 and 1972, beer drinker’s hyponatremia (beer potomania) was described in 7 patients with long histories of consuming at least 4 L of beer per day, sodium-deficient diets, low serum sodium values (98–107 mmol/L), unconsciousness, and seizures. One patient who ingested 6 to 12 16-oz cans of beer per day had a sodium value of 122 mmol/L. The electrolyte disturbances were attributed to possible inappropriate secretion of ADH. A later study described a specific “hypo-osmolality syndrome” in beer drinkers, and the authors attributed the hyponatremia to the consumption of beer, which is low in sodium, and poor intake of ordinary food, which led to reduced excretion...
of urinary solutes and inhibition of water diuresis with the development of hyponatremia (6). ADH concentrations are usually suppressed in patients with beer potomania, because they have an excess of water without the solute needed for diuresis (7, 8).

In 2007, a review of 22 published cases of beer potomania revealed a shared history of excessive beer intake, often long-term, with frequent recent episodes of binge drinking and poor dietary intake (8). Clinically, patients showed mild neurologic symptoms, rapid diuresis in response to solute intake, and laboratory values, if available, that showed severe hyponatremia (mean serum sodium, 108 mmol/L), hypokalemia (mean potassium, 3 mmol/L), and low urine sodium concentrations. The findings were inconsistent with regard to low urine osmolality (8).

Typically, individuals on a regular diet generate 1000 mOsm/day of solute from dietary sources and protein catabolism that must be excreted in the urine (8). The maximum diluting capacity of the kidney in healthy individuals is 50 mOsm/L; therefore, up to 20 L of water (1000 mOsm or 50 mOsm/L) can be ingested per day before hyponatremia develops (7, 8). However, individuals who drink large quantities of beer, which has a low sodium content (1–2 mmol/L), without an adequate intake of food have a much lower daily solute intake, which reduces the total daily amount of solute excreted in the urine to approximately 250 mOsm/day. That leads to hyponatremia after an intake of about 5 L (250 mOsm or 50 mOsm/L) of water per day (8). This volume is roughly equivalent to 14 cans of beer (12-oz cans), a level of beer consumption common among patients with beer potomania. Retention of 5 L of free water will produce a serum sodium concentration of 125 mmol/L in a 70-kg man over a 24-h period and a serum sodium concentration of 113 mmol/L on the second day (7).

Finally, we mention another example of hyponatremia, in which water intake exceeds the osmolar load in non-beer drinkers who have low intake of dietary solute (“tea and toast” syndrome, crash dieters) but excessive water intake, a phenomenon that in this age of dieting and weight consciousness can be seen with increasing frequency (9, 10). The case studies involved an ovolactovegetarian with hyponatremia because of a low intake of solute, which limited the patient’s ability to excrete water (9), and a recent case of crash-diet potomania in a patient who had a low-calorie diet and consumed 6 L of water per day. In the latter case, more water was consumed than the patient could excrete, owing to the smaller amounts of solute in the patient’s diet (10).

**POINTS TO REMEMBER**

- Severe hyponatremia is caused by an excess of water relative to sodium stores and may be caused by diuretics, vomiting, diarrhea, congestive heart failure, cirrhosis, inappropriate secretion of ADH, and, rarely, beer potomania and excess intake of water alone.
- Healthy patients on a normal diet may consume up to 20 L of water per day before they develop hyponatremia.
- Beer potomania with biochemical findings of low serum sodium, low serum and urine osmolalities, and low urine sodium values can cause severe hyponatremia. This case of hyponatremia is characterized by an excessive intake of beer and a poor intake of food. This reduces solutes in the urine, thereby limiting free-water excretion.
- If hyponatremia is corrected too rapidly in patients with beer potomania, sodium overcorrection and osmotic demyelination may occur.
- Severe hyponatremia may occur in patients who have a low solute intake (tea-and-toast and crash diets) and ingest excess water in the absence of beer drinking.

**Author Contributions:** All authors confirmed they have contributed to the intellectual content of this paper and have met the following 3 requirements: (a) significant contributions to the conception and design, acquisition of data, or analysis and interpretation of data; (b) drafting or revising the article for intellectual content; and (c) final approval of the published article.

**Authors’ Disclosures of Potential Conflicts of Interest:** No authors declared any potential conflicts of interest.

**References**