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Beer potomania:

Drink in this atypical cause of hyponatremia

By Mark McGraw, BSN, RN, CEN, CCRN, CTRN

IT WAS A TYPICAL MONDAY afternoon in the busy ED. I introduced myself to my next patient, Mr. H, a thin man in his mid-30s. After reviewing his vital signs, I asked how we could help him. As he sat there silently, his sister reported he'd been forgetful and shaky with periods of confusion for the past few days, but his condition had worsened today.

"He's always drunk...every day. This is nothing new!" She told me he'd been out of work because of alcohol abuse and dependence and had moved in with her a little over 6 months ago. The patient admitted to consuming at least thirty 12-oz cans of beer (10.8 L) a day.

Caring for Mr. H taught me about an unusual but potentially fatal consequence of excessive beer consumption known as beer potomania. This article describes this disorder and its dangers, how to recognize it, and the critical, very specific

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treatment needed to avoid serious complications, including osmotic demyelination syndrome (ODS). See *Understanding the dangers of ODS* for more information.

More than meets the eye

On physical assessment, I found Mr. H to be underweight and visibly malnourished, with a BMI of 16. He had thin, frail arms and legs, sunken eyes, coarse brittle hair, and a kwashiorkor-like distended abdomen. Although he was awake and alert, he was disoriented to time. His speech was clear most of the time, but he occasionally slurred some words, and he had bilateral upper extremity rest tremor. His lungs were clear to auscultation, his heart sounds were normal, and his abdomen was soft but distended. Mr. H stated he was worried because he hadn't urinated in 2 days.

Understanding the dangers of ODS

ODS occurs as a result of a rapid increase in osmolality, which can develop as a result of an overly aggressive increase in sodium levels during treatment for hyponatremia. The rapid change leads to dysfunction of the myelin-producing cells in the central nervous system. As these cells lose their functioning capabilities secondary to cellular injury, they lose the ability to produce and maintain the myelin sheaths. This results in a loss of myelinated neurons within the nervous system.[4,19,23]

ODS may not manifest for 2 to 8 days following the initiation of treatment for hyponatremia.[4] The signs and symptoms, which can be irreversible or only partially reversible, include paraparesis or quadriparesis, dysphagia, dysarthria, behavioral disturbances, lethargy, confusion, disorientation, obtundation, and coma. Less common signs include seizures. Some patients who are severely affected become "locked in"—despite being awake, they can't move or speak.[19]

The ED physician made a preliminary diagnosis of dehydration and electrolyte imbalance due to excessive alcohol consumption and prescribed 2 L of 0.9% normal saline to be administered I.V. Peripheral venous access was obtained, and blood specimens were sent to the lab for analysis.

Within 20 minutes, the lab called with a critical test result. Mr. H's serum sodium level was 105 mEq/L (normal 135 to 145 mEq/L). This critical sodium result triggered the lab to determine the serum osmolality to help isolate the cause of the hyponatremia, but the results weren't available yet.

The patient reported eating nothing and drinking only beer for the past few days. His history and physical assessment findings, coupled with the critical lab result, indicated that his medical problem was more than just dehydration. When the lab called back with a serum osmolality of 225 mOsm/kg (normal, 278 to 300 mOsm/kg), additional lab work was ordered.

Because the patient was unable to void, he was catheterized to obtain a very dilute urine specimen with a specific gravity of less than 1.005 (normal, 1.010 to 1.025), random urine osmolality of 75 mOsm/kg (normal, 50 to 1,200 mOsm/kg, with an average range of 500 to 800 mOsm/kg), and random urine sodium of 16 mEq/L (no established reference values).[1] The patient was admitted to the ICU with the diagnosis of beer potomania.

What's beer potomania?

Beer potomania, an unusual cause of hyponatremia, meets the following diagnostic criteria:[2]

- severe hyponatremia (serum sodium usually less than 110 mEq/L) without any other obvious cause
- low serum osmolality
- history of long-standing protein malnutrition
- consumption of a large amount of beer (usually more than fifteen 12-oz beers or 5.4 L of beer) in a relatively short time.[2-4]

Other general signs and symptoms, which are consistent with hyponatremia as well as acute alcohol intoxication, include the following:

- peripheral edema and third spacing
- cerebral edema
- profuse diaphoresis
- muscle cramping
- generalized weakness
- nausea and vomiting
- changes in mentation or memory
- restlessness and irritability
- gait disturbances
- uncontrolled tremors (fine and gross)
- seizures
- coma.[5,6]

Potentially serious hyponatremia

Morbidity and mortality associated with beer potomania depend on the severity of hyponatremia at admission. From 10% to 35% of hospitalized patients are found to have some degree of hyponatremia, the most common electrolyte abnormality.[7] In a 2009 study of over 98,000 hospitalized patients, even mild hyponatremia increased in-hospital, 1-year, and 5-year mortality.[8]

Mortality is greater than 50% for patients with alcoholism who present to the hospital with serum sodium levels less than 125 mEq/L.[9] In a literature review of patients diagnosed with beer potomania, 36% experienced complications with treatment, 18% developed some degree of ODS, and another 18% died.[4]

Most published case reviews of patients with beer potomania conclude with a discussion of patients left with a permanent neurologic impairment, dying from too-rapid correction of hyponatremia, or experiencing consequences related to misdiagnosis.

Understanding the disorder

The consumption of a large volume of beer, usually in conjunction with history of poor dietary intake and significant beer drinking, contributes to the pathophysiology of beer potomania. Beer is hypoosmolar: It's mostly water with a very low sodium content (about 2 mEq/L), minimal solutes, and no protein. As beer is absorbed, the osmoreceptors sense the increase in hypotonic fluids, interpret this as a drop in serum osmolality, and inhibit antidiuretic hormone (ADH) release. *Osmolality* is a calculation of the ratio of body water to sodium and other solutes.

Normally as ADH levels drop, less water is reabsorbed from the nephrons and urine is produced. However, ADH doesn't work alone to produce urine; it's the presence of electrolytes and solutes that helps to pull fluid into the nephron to be processed for elimination. To produce 1 L of maximally dilute urine, the kidneys require 50 to 60 mOsmol of solutes.[3] The patient who's chronically malnourished or protein deficient doesn't have available solutes and will have a net retention of fluid despite little or no ADH.[3,4] See *The role of ADH*.

These solutes, normally obtained from the diet, include electrolytes, glucose, proteins, and other substances dissolved in the plasma. Severe protein malnutrition, poor dietary intake, and lack of other sources of solutes cause the volume of urine output to fall until the patient becomes anuric (producing less than 100 mL of urine per day). The patient can remain in this state until sufficient solutes are reintroduced to produce a maximally dilute urine, when the urine output can become severe and overwhelming.[4,7]

As a result, intake in this patient of more than 5 L of water or the equivalent of about 14 beers will cause an overall net fluid retention and hyponatremia.

Serum sodium levels rising too quickly, whether by rapid sodium replacement or excessive diuresis with the loss of free water, places added stress on the already overstressed oligodendrocytes in the central nervous system. Functioning as a neuron support network, oligodendrocytes produce and maintain protective and insulating myelin sheaths for neurons. Because they're highly sensitive to rapid fluctuations in osmolality, the result can be injury, dysfunction, and eventual necrosis of the oligodendrocytes.[10] As myelin is lost, cellular signals and electrical impulses are increasingly disrupted.

The neurons and supportive cells will initially adapt as extracellular concentrations of sodium and electrolytes are diluted by the retention of free water, but the serum osmolality will continue to fall less than the intracellular environment. Osmosis will continue to move fluid into the cerebral cells in an ineffective effort to reach homeostasis.

As the fluid enters cells, they swell, become dysfunctional, and eventually lyse when the volume is too great for the cell walls to contain, resulting in cellular death.

Diagnostic testing

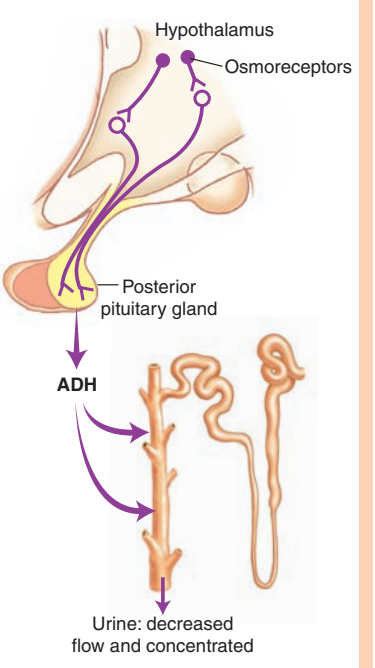
There are no specific diagnostic studies for beer potomania.[2] In beer potomania, electrolyte studies demonstrate hyponatremia, often with a critically low serum sodium level. Complete blood cell counts may show a dilutional drop in hemoglobin and hematocrit secondary to the excessive fluid intake. The urine osmolality test will likely be less than 100 mOsm/kg consistent with water intoxication as seen with beer potomania.[11]

Unless the patient has an underlying metabolic or organic disorder, thyroid studies, liver panel, renal function studies, and cortisol, glucose, and catecholamine levels will more than likely be within normal limits.

As stated earlier, the lack of solutes needed to produce even maximally dilute urine will cause a low urine output. The urine specific gravity will be low because of the effect of maximal dilution. The absence of solutes causes the urine to be hypoosmolar (less than 100 mOsmol/kg).[12] The low urinary osmolality is rare and seen only with beer potomania, primary polydipsia, reset osmostat syndrome, or a very low dietary sodium intake.[7,11,13]

The role of ADH

ADH, which regulates the ability of the kidneys to concentrate urine, is synthesized by neurons in the hypothalamus and transported down their axons to the posterior pituitary gland and then released into the circulation. One of the main stimuli for synthesis and release of ADH is an increase in serum osmolality. ADH release is also controlled by cardiovascular reflexes that respond to changes in BP or blood volume.



Source: Porth CM. *Essentials of Pathophysiology: Concepts of Altered Health States*. 3rd ed. Philadelphia, PA: Wolters Kluwer Health/ Lippincott Williams & Wilkins; 2011.

Although the excretion of urinary sodium is partially dependent on intake, it also reflects the kidney's concentrating capacity. It may be low in conditions of volume overload and conditions affecting the heart, thyroid, liver, and kidney.[5]

Look out for this clinical picture

Patients with beer potomania will likely arrive for treatment several hours to days following the onset of symptoms. Many patients have signs and symptoms consistent with acute hyponatremia, hypervolemia, and long-standing protein malnutrition.

The earliest clinical indicators of hyponatremia are vague neurologic complaints. Their severity depends on how quickly sodium is decreasing and cerebral edema is developing. Signs and symptoms of acute hyponatremia include difficulty concentrating, confusion, lethargy, agitation, headache, seizures, anorexia, nausea, and vomiting. The more severe the signs and symptoms, the more severe the degree of hyponatremia.[6,14] Many of the signs and symptoms of beer potomania may be noted in those with acute alcohol intoxication.

To evaluate a significant change in mental status, the healthcare provider may need to rely heavily on a comprehensive assessment and accounts of patients' activities and behavior. Patients with severe signs and symptoms of acute hyponatremia, such as coma and respiratory arrest, require very aggressive treatment to prevent permanent neurologic disability or death.

The lack of dietary protein, excessive beer intake, and oliguria or anuria can lead to fluid overload, including hypertension with bounding pulses, dyspnea on exertion, pulmonary crackles, and peripheral edema. Third spacing occurs as the loss of circulating plasma proteins lowers vascular oncotic pressure.

These patients, despite being hypervolemic, will remain oliguric or anuric until sufficient amounts of solutes have been restored. The sudden introduction of solutes or other metabolites from snacking, drinking soda, or even taking certain prescription medications can cause a dangerous and overwhelming diuresis that will cause sodium levels to dramatically increase. Even patients without neurologic manifestations should be considered high risk and be kept N.P.O. for at least first 24 hours following admission. Meticulously monitor serum sodium levels at least every 2 hours in the early acute phase of treatment.

Beer is hypoosmolar: It's mostly water with a very low sodium content, minimal solutes, and no protein.

Protein malnutrition causes many physical assessment findings including brittle, coarse, and thick hair and nails. Patients may look older than their chronologic age because their hair can be prematurely gray and dermal proteins that maintain skin tone may have degenerated. Their skin may be so fragile that it separates under pressure. The tongue may be atrophied and there may be muscle wasting.[15]

Protein malnutrition delays wound healing, so any ecchymoses, abrasions, and lacerations will be slow to heal. Patients will also report a diminished urine output.

Many treatment options

Goals for patients with beer potomania are to

- slowly correct hyponatremia
- prevent neurologic disabilities secondary to ODS
- prevent alcohol-related complications such as alcohol withdrawal syndrome (AWS) or Wernicke encephalopathy.

The many treatment options for patients with beer potomania include both fluid restriction and infusion of various concentrations of saline, diuretics, and hypotonic fluids with vasopressin.[11,16,17]

Treatment parameters must be developed and implemented on a case-by-case basis to carefully balance the benefits with possible risks. This is challenging because underlying conditions may be masked by the alcohol ingestion, which could impact treatment. Judiciously monitor urine output and serum sodium levels and perform frequent neurologic assessments as priority nursing interventions.

When patients' sodium is being actively replaced, treatment goals should be to achieve a serum sodium level increase of less than 6 mmol/L in the first 24 hours, less than 12 mmol/L in the first 48 hours, and less than 14 mmol/L in 72 hours to prevent cerebral injury.[18] These recommendations are lower than the previously held ideal of less than 10 mmol/L in the first 24 hours and 18 mmol/L in 48 hours because they've been associated with an even lower incidence of neurologic complications.[18]

Patients with severe signs and symptoms such as respiratory distress, seizure, or coma require an acute increase in serum sodium level by 1 to 2 mmol/L in the first 2 to 3 hours, without exceeding the 24- and 48-hour goals.[18]

Achieving this small but life-saving spike in the serum sodium is the most important nursing

intervention to prevent long-term disability in these patients.[18] The quickest and most reliable method for acutely raising the serum sodium level is to administer 50 to 100 mL of 4% hypertonic saline over 30 minutes and repeat once if severe symptoms haven't improved.[18]

An asymptomatic patient may be more likely to develop ODS with faster rates of correction as previously recommended (10 mmol/L in 24 hours) because his or her brain has adapted to a hyponatremic environment and isn't displaying outward signs of distress.[6]

Patients should remain N.P.O. for at least 24 hours following admission. Remember that any additional fluids will only potentiate the patient's condition by further diluting plasma. The reintroduction of solutes, including sodium from I.V. fluids, can precipitate an overwhelming diuresis in extreme cases, approximately 10 L within a few hours. Keeping the patient N.P.O. and monitoring urine output are key nursing interventions to help prevent an overwhelming diuresis that may inadvertently cause sodium levels to spike and increase patients' risk of ODS.[5,19]

To prevent the serum sodium level from rising too quickly, anticipate an infusion of D_5W to replace free water in patients with a sustained urine output greater than 500 mL/hour.[2,20] D_5W is rapidly metabolized and has no osmotically active particles after it's infused into the vascular compartment; once the dextrose is metabolized, only free water remains.[21]

The infusion rate is based on the hourly urine output; the replacement ratio is 0.25 to 1 mL of D_5W per mL of urine due to be infused within the following hour.[2] Administer thiamine as prescribed to prevent Wernicke encephalopathy.

If the patient's serum sodium level rises too quickly with the treatment described above, anticipate the addition of desmopressin to the clinical treatment plan.[2] Desmopressin is synthetic ADH that acts at the level of the collecting tubules to increase water absorption.[22] When desmopressin is given intranasally, its antidiuretic effect will begin within 15 to 30 minutes and peak within the hour. The duration of action ranges from 6 to 14 hours.[23] This pharmacologic intervention will limit urine volume, increase urine concentration, and increase free water retention.

Lack of dietary protein, excessive beer intake, and oliguria or anuria can lead to fluid overload.

The healthcare provider will consider the results of a comprehensive assessment, including mental status, in combination with lab values when prescribing treatment. Follow institutional policies and procedures; some facilities require ICU monitoring.

Be on alert for alcohol withdrawal

The healthcare team should recognize the potential for AWS in these patients. Hospitalization will interfere with their alcohol consumption; this should be taken into consideration during care planning. The more time that's elapsed since their last drink, the closer these patients will be to developing signs and symptoms of AWS. Other factors to consider include comorbidities, coagulopathies, and hepatic and pancreatic function. For more information, see "Managing Alcohol Withdrawal in Hospitalized Patients" (April, *Nursing2012*).

Prompt recognition and treatment

Mr. H was managed with conservative measures in the ICU. A central venous access device was inserted. He was kept N.P.O. for 24 hours and voided less than 3 L during the first 24 hours. The patient was later advanced to a clear diet.

About 14 hours after admission, his sodium level increased to 112 mEq/L. He received 100 mg thiamine, followed shortly by an infusion of D_5W. His sodium level remained at 112 mEq/L for the remainder of the first 24 hours.

He spent the next 6 days in the hospital with blood draws every 2 hours for the first 2 days, then slowly decreasing to every 4, then every 8 hours. Nausea was controlled with around-the-clock doses of ondansetron, which was discontinued on hospital day 2. At the end of day 2, the patient received multivitamins and folic acid in his I.V. fluid.

Lorazepam was administered for withdrawal signs and symptoms, including increasing tremors, anxiety, and tactile hallucinations. On hospital day 3, Mr. H had his first meal, which was well tolerated. On day 4, he was discharged from the ICU to a progressive care bed. Multiple consults were arranged to help Mr. H deal with his chronic alcohol use and dependence. The patient was subsequently discharged without any signs or symptoms of ODS on hospital day 6.

Ready for a case?

Your recognition of a patient with beer potomania and understanding

of the careful management needed could prevent serious disabilities or death. Educate your colleagues and vulnerable patients about this little-known condition. ■

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