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A Rare Case of Low-Solute Hyponatremia in a Nonalcoholic Person

Weeraporn Srisung Charoen Mankongpaisarnrung Cyriacus Anaele
Nat Dumrongmongcolgul Vaqar Ahmed

Department of Internal Medicine, Texas Tech University Health Sciences Center, Lubbock, Tex., USA

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Hyponatremia · Low-solute hyponatremia · Beer potomania · Osmotic demyelination syndrome

Abstract
Low-solute hyponatremia is a relatively uncommon entity of euvolemic hyponatremia. Classic cases were described in alcoholics as beer potomania, which is characterized by hyponatremia in the setting of low-solute intake due to heavy beer drinking. We report a case of low-solute hyponatremia in a nonalcoholic person who was given a solute load, and, subsequently, had excessive diuresis with the resultant rapid increase in serum sodium concentration.

Introduction

Hyponatremia is a very common electrolyte abnormality with its usual initial diagnostic approach involving the determination of the patient's tonicity and volume status. Euvolemic hyponatremia often poses a diagnostic challenge to physicians and nephrologists. The differential diagnosis includes the syndrome of inappropriate antidiuretic hormone (SIADH), glucocorticoid insufficiency, hypothyroidism and primary polydipsia. In this group, low-solute hyponatremia is relatively uncommon. Classic cases were described in alcoholics as beer potomania, which is characterized by hyponatremia in the setting of low-solute intake due to heavy beer drinking [1, 2]. Impaired free water clearance secondary to low solute excretion with excessive free water ingestion is the fundamental pathophysiologic process in this condition [3]. The treatment can be challenging since there is a high risk of overcorrec-

Weeraporn Srisung, MD
Department of Internal Medicine, Texas Tech University Health Sciences Center
3601 4th Street
Lubbock, TX 79430 (USA)
E-Mail weeraporn.srisung@ttuhsc.edu

tion of serum sodium concentration potentially resulting in osmotic demyelination syndrome (ODS) [4].

Case Presentation

A 69-year-old African American male with a past medical history of hypertension, seizure disorder, but no underlying psychiatric diagnoses, presented to the emergency department with confusion and urinary incontinence. He had had recurrent episodes of hyponatremia of unclear etiology associated with weakness and a fluctuating mental status. Previous records did not exist in our hospital system, and the patient was not able to provide any information regarding the severity, diagnostic workup, treatment and the number of prior episodes. On arrival, his blood pressure was 162/62 mm Hg, the heart rate 70 beats per minute, the respiratory rate 16 breaths per minute and the temperature 98.7 F. Cardiovascular, respiratory and abdominal examinations were unremarkable. He had normal skin turgor with no peripheral edema and no focal motor deficits. Computed tomography of the head was unremarkable for any intracranial findings. In the emergency department, his serum sodium concentration was 117 mEq/l, BUN was 6 mg/dl, creatinine was 0.7 mg/dl and glucose was 96 mg/dl. The urine osmolality was 132 mosm/kg, and plasma osmolality was 250 mOsm/kg. Urine sodium was not checked initially. He received 1 liter bolus of normal saline. A second serum sodium concentration was obtained at 12 h after the bolus, and the level was 127 mEq/l. The patient produced 8 liters of urine in his first 12 h after arrival. Five percent dextrose water (D5W) was then started at 100 ml per hour to relower the serum sodium concentration and to prevent rebound increase. Fifteen hours after the initial normal saline bolus, his serum sodium concentration decreased to 123 mEq/l. His urine osmolality and urine sodium were now 315 mosm/kg and 128 mEq/l, respectively. Additional history obtained after he regained consciousness revealed an inability to maintain oral intake due to poor appetite for 3 months. However, he continued to drink fluids. He was adamant that he only drank occasionally, 2–3 drinks per month, and he denied a history of liver disease. His home medications included amlodipine, lisinopril and phenytoin. His serum sodium concentration was monitored, and the rate of D5W was adjusted to control the rate of increase in serum sodium concentration to no more than 10–12 mEq/l in 24 h and 18 mEq/l in 48 h. The patient was finally discharged at a serum sodium concentration of 130 mEq/l without any neurological sequelae.

Discussion

Hyponatremia is a very common electrolyte abnormality [5], and in essence, it is a disorder of water balance. Hyponatremia is categorized into 3 groups: hypovolemic, euvolemic, and hypervolemic hyponatremia. Of those, euvolemic hyponatremia becomes more diagnostically and therapeutically challenging. Low-solute hyponatremia is relatively uncommon in this group. Urine sodium and urine osmolality are clinically important; however, these parameters may be inconsistent among patients with this condition. Therefore, a comprehensive evaluation is necessary to establish the definite diagnosis in a timely fashion to start the appropriate treatment.

Free water excretion by the kidney largely depends on the degree of ADH activity. Normal human physiology with ADH suppression along with normal renal function allows humans to handle excessive water intake without developing hyponatremia until the

maximum urinary dilution capacity is reached [4]. This is the core pathophysiology of primary polydipsia. However, an important factor often overlooked is the amount of solute load and the solute excretion. In addition to sodium and potassium intake, protein intake contributes to solute excretion by urea formation; 10 g of protein produce approximately 50 mosm of urea. An analysis on free water clearance clearly demonstrates that solute excretion is an important factor determining the amount of free water clearance, and thus serum sodium concentration [6].

$V = CH_2O + C_{osm}$, since $C_{osm} = U_{osm}V/P_{osm}$; therefore, $CH_2O = V - (U_{osm}V/P_{osm})$; as V = solute excretion/U_{osm}, we can derive that CH_2O = solute excretion $(1 - [U_{osm}/P_{osm}])/U_{osm}$ (CH_2O = free water clearance, C_{osm} = solute clearance, U_{osm} = urine osmolality, P_{osm} = plasma osmolality, V = urine flow)

Figure 1 shows that at a certain urinary osmolality and the degree of solute load has a significant impact on free water clearance, especially when urinary osmolality is low [7]. It also shows that urinary osmolality does not accurately represent the ability of the kidney to excrete free water. For example, at urinary osmolality of 50 mosm/kg, a person would excrete 15 liters of free water per day if the daily solute excretion were 900 mosm/day, but he/she would excrete only 5 liters of free water per day if the daily solute excretion were 300 mosm/day. Therefore, water intake of more than 5 liters per day in the latter subject would cause hyponatremia.

In low-solute hyponatremia, low urine osmolality (less than 100 mosm/kg) is not always present. Sanghvi et al. [4] reviewed 22 published cases of beer potomania, a prototype of low-solute hyponatremia, and demonstrated that low urinary osmolality is not a consistent finding. Therefore, the possibility of low-solute hyponatremia cannot be ruled out in patients with high urine osmolality.

Once solute intake is restored, hyponatremia corrects by free water excretion, as there is no intrinsic defect in urinary dilution in these patients. A rapid increase in serum sodium concentration can occur and may lead to a significant risk of ODS. Our patient developed excessive diuresis that led to a rapid correction in serum sodium concentration from 117 to 127 mEq/l within 12 h, necessitating serum sodium concentration relowering with D5W. The recommendation in managing chronic hyponatremia is to increase the serum sodium concentration no more than 10–12 mEq/l in the first 24 h, and no more than 18 mEq/l in the first 48 h due to the risk of ODS with overcorrection exceeding these limits [8, 9]. Symptoms of ODS include ataxia, dysarthria, dysphagia, parkinsonism, paraparesis or quadriparesis, and coma [10]. Typically, these neurological symptoms occur several days after the metabolic insult [10, 11]. Therefore, the mentioned treatment goals should strictly be adhered to. Relowering of serum sodium concentration is also crucial when overcorrection occurs, and it can be achieved by the administration of D5W with the rate matching urine output [4]. It has neurological benefits even after symptoms of ODS develop [12, 13]. Sanghvi et al. [4] have developed a treatment algorithm for beer potomania, which can be applied in our case due to similar pathophysiology. The mainstay of treatment includes 0.5 liters of normal saline in those patients with major symptoms (such as seizure and coma) with the goal of an increase in serum sodium concentration by 2–3 mEq/l/h for 2–3 h, followed by fluid restriction. For those with mild or no symptoms, fluid restriction would be an appropriate treatment option. Close monitoring of serum sodium concentration is required, and D5W infusion should be started when necessary. Desmopressin administration can be considered in some settings: first, if the D5W rate cannot be matched with the rate of urine output; second, if the rate of an increase in serum sodium concentration is too rapid even with D5W infusion and the limit of increase in serum sodium concentration is expected to be

reached within a short period of time; third, the increase in serum sodium concentration has already exceeded the goal, and fourth, the presence of ODS symptoms. Sanghvi et al. [4] also recommended keeping patients nothing by mouth for 24 h before initiating the oral intake because of the risk of overcorrection after the introduction of the solute. A half-normal saline solution (0.45%) can be administered in case of failure to increase serum sodium concentration after fluid restriction.

Conclusion

Our case highlights the importance of the detection of low-solute hyponatremia since the solute load such as normal saline, which is commonly given in the emergency department to hyponatremic patients, potentially leads to a rapid increase in serum sodium concentration in these patients. The clinical picture of low-solute hyponatremia can mimic SIADH as urine osmolality may exceed 100 mosm/l. However, low-solute hyponatremia will typically cause significant water diuresis resulting in a rapid increase in serum sodium concentration after a solute load as opposed to worsening serum sodium concentration in SIADH. Rapid correction of serum sodium concentration in hyponatremic patients can predispose to ODS. If overcorrection occurs, lowering serum sodium concentration with D5W is crucial. Management includes fluid restriction in mild or asymptomatic patients, finite amounts of intravenous fluids in symptomatic cases, and maintenance of the solute load with oral intake after 24 h of nothing by mouth.

Disclosure Statement

Figure 1 is originally from a previously published article [7]. Permission for reuse has been obtained from both the original author, Dr. Tomas Berl, and the original publisher, Elsevier.

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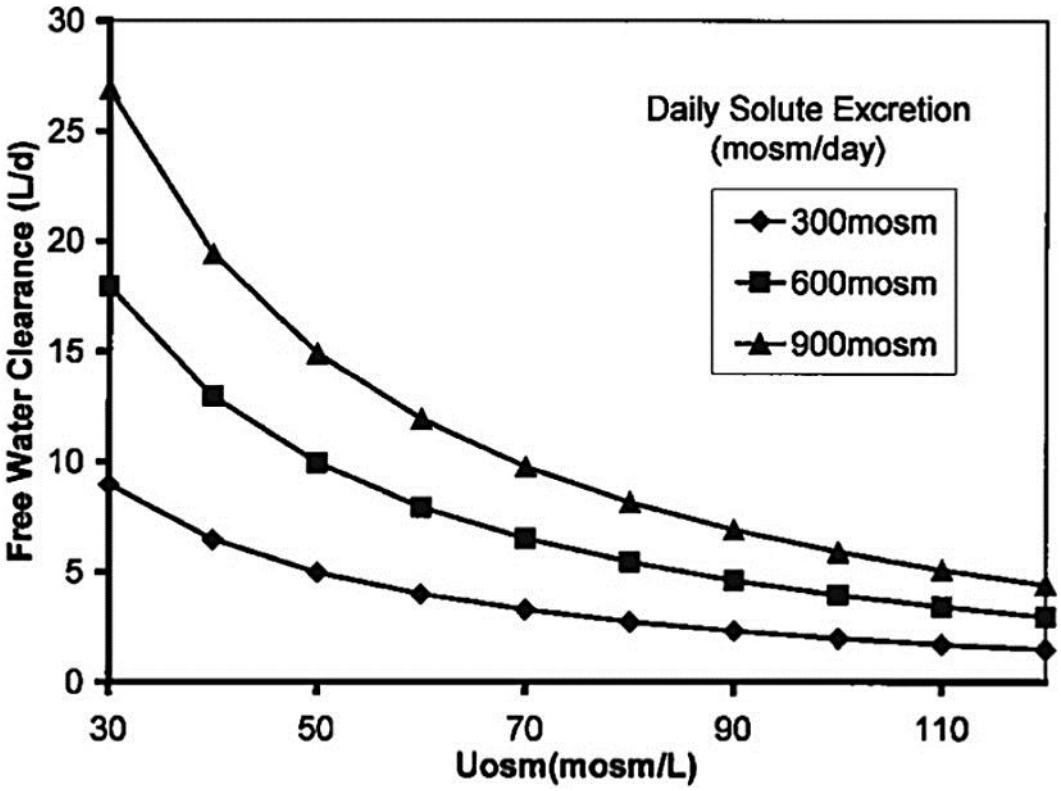

Fig. 1. Free water clearance in relation to urine osmolality at a certain level of daily solute excretion.