

CASE REPORTS

“Beer Potomania” in Non–Beer Drinkers: Effect of Low Dietary Solute Intake

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● A ovo-lacto-vegetarian patient presented with hyponatremia. She had maximally dilute urine and undetectable vasopressin levels. Dietary history revealed very low protein intake but no beer intake. We postulated that the very low intake of solute limited her water excretion and caused the hyponatremia despite only a modest increase in fluid intake. When protein intake was increased in a clinical research center setting, free water excretion increased and serum sodium normalized despite maintaining the water intake at 4 to 5 L daily. We discuss the role of dietary solute in water excretion. Previously described in beer drinkers, the phenomenon can occur in the absence of beer drinking. In this era of weight consciousness, hyponatremia because of low solute intake may be seen with increased frequency.

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INDEX WORDS: Hyponatremia; solute intake.

HYPONATREMIA is the most common electrolyte disorder encountered in hospitalized patients.¹ In the face of hypotonicity, the urine should be maximally dilute (< 80 mOsm/kg). Yet, in hyponatremic patients, the urinary osmolality is almost always inappropriately elevated, indicating that the pathogenesis underlying the disorder is almost uniformly the nonosmotic release of vasopressin.¹ However, when hyponatremia occurs in the presence of a maximally dilute urine, vasopressin secretion is not operant, and alternative mechanisms must be implicated. The excessive administration of fluids leads to urinary dilution, but under normal circumstances, hyponatremia supervenes only with extreme levels of intake exceeding 20 to 25 L per 24 hours.² Hyponatremia has been described in beer drinkers, implicating the very low content of sodium in the beverage as limiting water excretion.³⁻⁵ It is not generally appreciated that hyponatremia may also occur independently of high levels of beer consumption. We recently saw three patients who denied alcohol consumption and in whom limited solute intake culminated in hyponatremia despite normal urinary dilution. We studied one of these patients in

detail and ascertained the peculiar dietary habits that, when accompanied by generous water consumption, can culminate in hyponatremia.

CASE REPORT

A 34-year-old woman sought medical attention when she noted that she was becoming somewhat fatigued while running her usual 30 to 40 miles per week. On initial evaluation by her primary care physician, she had a serum sodium level of 124 mEq/L without obvious cause. When measured on six subsequent occasions over the ensuing 3 months, her serum sodium level was normal only once; the other values ranged from 124 to 131 mEq/L. The thyroid stimulating hormone level, serum cortisol level before and after Cortrosyn (Organon, West Orange, NJ), and the chest radiograph were all normal. The patient was referred for further evaluation. The medical history was otherwise noteworthy for secondary amenorrhea since age 22, attributed to her high degree of physical activity. The patient was a nulliparous nonsmoker who consumed alcohol only occasionally and did not use illicit drugs or herbs. She was a strict ovo-lacto-vegetarian who attempted to minimize her salt intake and who drank four to six 16-oz glasses of water daily. Her medications included vitamin C, calcium carbonate, prenatal vitamins, and occasional use of selenium, chromium, and tolmetin. She denied use of laxatives or diuretics.

Physical examination showed her to be a somewhat thin but well-developed white woman weighing 51.8 kg. Lying supine, she had a pulse of 52 beats/min with a blood pressure of 100/60 mm Hg. After assumption of the erect posture for 2 minutes, her pulse was 64 beats/min with a blood pressure of 104/80 mm Hg. Skin turgor was normal. Examination of the heart and lungs were entirely normal. There was no peripheral edema and the neurologic examination was normal. Laboratory evaluation showed a complete blood count noteworthy only for a hemoglobin level of 12.6 g/dL and a normal total lymphocyte count. The serum sodium level was 134 mEq/L, potassium level 4.0 mEq/L, CO₂ level 30 mEq/L, chloride level 99 mEq/L, glucose level 66 mg/dL, creatinine level 0.9 mg/dL, phosphorus level 4.6 mg/dL,

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total protein level 6.9 g/dL, and cholesterol level 213 mg/dL. Simultaneously obtained urine had a sodium level of 16 mEq/L, chloride level less than 15 mEq/L, and osmolality of 93 mOsm/kg. On multiple subsequent determinations, the serum sodium level was consistently 133 mEq/L or less, potassium level was consistently 3.8 mEq/L or greater, glucose level was 91 mg/dL or less, the serum creatinine level was 0.8 to 0.9 mg/dL, and the BUN level was 6 to 7 mg/dL. Repeated urine osmolality was 64 mOsm/kg when the serum sodium level was 124 mEq/L. Two randomly obtained urine samples were analyzed for the presence of diuretics; none were detected in either sample. The plasma vasopressin level measured when the serum sodium level was 133 mEq/L was below the limits of detection. Twenty-four-hour urinary sodium and urea nitrogen excretion were 39 mEq and 3 g (normal, 12 to 20), respectively, in a total volume of 2.6 L. We hypothesized, therefore, that despite normal glomerular filtration rate and urinary diluting capacity, this patient was hyponatremic as a result of her exceedingly low-solute intake coupled with modestly increased water intake.

To test this hypothesis, the patient was admitted to the Clinical Research Center at University Hospital and initially permitted to continue food and water intake "ad lib." Her daily water intake was ~5 L. Analysis of her diet showed a protein intake of ~26 g daily, a fact reflected in the very low rate of daily urea nitrogen excretion (Table 1). The dietary sodium intake was estimated at 48 mmol/d and the potassium intake at 53 mmol/d, rather closely reflected in the urinary sodium excretion of ~40 mmol/d and potassium excretion of 68 mmol/d. After 2 days, her diet was changed to contain 80 g of protein (1.56 g/kg daily) and her daily water intake was fixed at 5,088 mL, which was the average of her 2 prior days' intake. The sodium and potassium intake was also maintained at the level of her "ad lib" diet. On this diet, her electrolyte-free water clearance increased from 3.3 to 4.0 L daily, and serum sodium promptly increased (Table 1) despite a very generous intake of water. The patient was

then discharged and asked to return with a 24-hour urine collection 3 days later. She was encouraged to maintain a high protein intake. However, she was again hyponatremic as her protein intake decreased, reflected by a decrement in urea nitrogen excretion.

DISCUSSION

The development of hyponatremia is viewed as reflecting an impairment in urinary dilution most often brought about by the persistent secretion of vasopressin. Under such conditions, the urinary osmolality is less than maximally dilute, ie, greater than 100 mOsm/kg H₂O. However, a vasopressin-independent mechanism must be operant in the patient discussed here, as well as in two other non-beer drinkers we recently observed, whose urinary osmolalities were 80 and 54 mmol/L at a time when their serum sodium levels were 117 and 116 mmol/L. Under normal circumstances (normal glomerular filtration rate and absence of vasopressin), an inordinate intake of water is required to overwhelm the large capacity for urinary dilution (~20 to 25 L daily); the patient reported here shows the critical role of solute intake in establishing the limits of water intake that safely maintain normal levels of serum sodium. The ability to generate and therefore excrete free water becomes limited if solute excretion is markedly decreased. This becomes evident from the following analysis:

(1) Urine flow (V)

$$= cH_2O \text{ (free water clearance)} \\ + \text{Cosm (solute clearance);}$$

substituting $\frac{U_{osm}V}{P_{osm}}$ for Cosm yields

$$(2) cH_2O = V - \frac{U_{osm}V}{P_{osm}} .$$

Because $V = \frac{\text{Solute excretion}}{U_{osm}}$, we derive that

$$(3) cH_2O = \frac{\text{Solute excretion}}{U_{osm}} \left(1 - \frac{U_{osm}}{P_{osm}} \right)$$

Equation (3) shows that the excretion of free water requires that U_{osm} is less than P_{osm}, with more free water generated at lower urinary osmolality. However, the total volume so generated is limited by low solute excretion, even if urinary

Table 1. Blood and Urinary Parameters

	Ad Lib Diet	After 2 Days on Protein-Supplemented Diet	3 Days Postdischarge on Ad Lib Diet
P _{Na} (mEq)	131	138	134
P _K (mEq/L)	4.2	4.2	4.1
Posm (mOsm/kg)	268	288	274
V (mL/d)	4,010	4,710	4,960
U _{osm} (mOsm/kg)	81	122	114
U _{Na} (mmol/d)	10	13	12
U _K (mmol/d)	16.6	15.6	20.9
Urea (g/d)	2.45	8.1	5.95
H ₂ O intake (mL/d)	4,820	5,082	—
cH ₂ O _e (L/d)	3.3	4.0	3.7

Abbreviations: P_{Na}, plasma sodium; P_K, plasma potassium; Posm, plasma osmolality; V, urinary volume; U_{osm}, urinary osmolality; U_{Na}, urinary sodium; U_K, urinary potassium; Urea, urea nitrogen excretion; H₂O, water intake; cH₂O_e, electrolyte free water excretion.

osmolality is very low. This relationship is shown in Fig 1, which shows the effect of urine osmolality on maximal free clearance at various levels of solute excretion. Thus, at urinary osmolality of 80 mOsm/kg, free water clearance is 2.7 L daily when solute excretion is 300 mosm daily, 5.4 L daily at 600 mosm daily, and as high as 8.1 L daily at 900 mosm daily. Therefore, a patient such as the one reported here may exceed maximal free water excretory capacity at levels of fluid intake as low as 3 L daily.

The effect that alterations in water excretion have on serum sodium levels is also dependent on the nature of the excreted solute.^{2,6} Urinary osmolality is a function of both electrolytes (primarily sodium, potassium, and their accompanying anions) and urea excretion. However, because the latter readily crosses cell membranes and is not truly an "effective" solute, it has no ultimate impact on serum sodium.^{2,6} In such a formulation, much more relevant is the electrolyte-free water. In calculating this entity, the urinary osmolality is replaced by the concentration of sodium plus potassium, and the plasma osmolality by plasma sodium.

$$cH_2O_e = \frac{\text{solute excretion}}{U_{\text{osm}}} \left(1 - \frac{U_{\text{na}} + U_{\text{k}}}{P_{\text{na}}} \right)$$

Therefore, for any given urinary concentration of sodium and potassium and urinary osmolality, the electrolyte-free water excretion is also dependent on the total rate of solute excretion. In this patient, the serum sodium increased when solute intake increased because electrolyte-free water

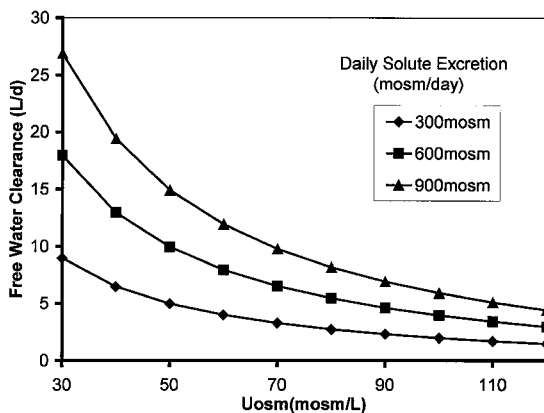


Fig 1. Dependence of water clearance on daily solute excretion at low urinary osmolalities.

increased (Table 1). This occurred despite a small increase in urinary osmolality and constant sodium and potassium concentrations in the urine. The products of catabolism obligate the excretion of ~100 to 150 mmol of urea per day. Most of the urea that appears in urine is a consequence of protein intake. Every 10 g of protein intake leads to ~50 mOsm of urea excreted. Thus, under normal conditions (~70 gm protein intake), ~450 mOsm of urea are excreted ([50 x 7] + 100). The intake of 150 mmol/L of sodium and 50 mmol/L of potassium (with accompanying anions) and the contribution of other solutes (NH₄, creatinine) allow for the excretion of ~900 mOsm of solutes. Such an individual can imbibe large volumes of fluid because at a urinary osmolality of 60 mOsm/kg (which is not even the lowest that can be achieved), 13.5 L of electrolyte-free water per day are excreted. However, if sodium and overall food intake is limited, decreasing excretion of solutes to only 300 mOsm daily, fluid intake in excess of 5 L will result in water retention and hyponatremia. The well-described development of hyponatremia seen in beer drinkers was ascribed to such a process.^{3,7} However, this description of 12 Belgian patients lacks data on urinary osmolalities, and many of the patients had clinical entities known to be associated with vasopressin secretion, such as congestive heart failure and liver disease. Hilden and Svendsen⁴ described patients in whom the evidence was more compelling because urinary osmolalities were less than 100 mOsm/kg. Since these descriptions, the syndrome has been exclusively associated with the intake of beer (beer potomania),^{5,8-11} a beverage poor in sodium content. It is of interest that these patients usually present as profoundly hyponatremic (mean sodium level, 105 mEq/L) and often hypokalemic as well (mean potassium level, 3.1 mEq/L), as seen in the patients reviewed by Fenves et al.⁷ We report here that a syndrome similar to that of beer potomania can supervene in individuals who limit their intake of electrolytes and protein yet do not curtail their water intake. In this regard, patients with anorexia would appear to be prime candidates for such a disorder. However, such patients display various abnormalities in osmoregulation,¹² including erratic vasopressin secretion and rarely diabetes insipidus. The serum sodium levels in the patients in the above study

were normal. However, in a survey of approximately 30 patients with anorexia nervosa, the mean serum sodium level was only 132 mmol/L, significantly lower than normal (P. Mehler, personal communication).

Because much of water intake is habitual, in this era of weight consciousness and low-protein diets, the development of hyponatremia is clearly not restricted to individuals who consume large volumes of beer and may be seen with increased frequency.

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