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Case Report

Acute Hyponatremia After a Religious Fast

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ABSTRACT

Objective: Our objective is to describe how polydipsia and intake of nonsteroidal anti-inflammatory drugs (NSAIDs) after fasting while breastfeeding may result in acute symptomatic hyponatremia.
Case Report: We present the case of a 24-year-old woman at 4 weeks postpartum who engaged in a 20-hour fast from both eating and drinking, during which she continued to breastfeed her newborn child. After ending her fast, she noted decreased milk supply. Attributing her decreased milk supply to dehydration, she then consumed 4 L of water with little salt and also took NSAIDs for a headache, which continued to worsen. Upon presentation to the emergency department, she was found to have a sodium level of 124 mEq/L (normal, 135-145 mEq/L) and a urine specific gravity of 1.015 (normal, 1.005 − 1.030). Thyroid function and cortisol level test results were normal. She was diagnosed with acute symptomatic hypovolemic hyponatremia. After 1 L of normal saline her sodium rapidly corrected to normal and her symptoms resolved. At 2 months of follow-up she was asymptomatic and had no further episodes of hyponatremia.
Discussion: Due to the patient's gender and small body size, 4 L of water was sufficient to lower her serum sodium rapidly from normal to 124 mEq/L. She was unable to excrete this water due to a combination of hypovolemia-mediated arginine vasopressin and NSAID use.
Conclusion: Clinicians should be cognizant that reproductive-age women are uniquely susceptible to hyponatremia and dangerous sequelae therein. They should counsel fasting individuals, particularly lactating women, to consume solute as well as fluid after fasting.

Introduction

Life threatening hyponatremia has been reported to occur in otherwise healthy people when exposed to severe exertion or heat that is followed by excessive fluid intake.[1] Additionally, nonsteroidal anti-inflammatory drugs (NSAIDs) have been suggested as a risk factor in exacerbating the development of acute severe hyponatremia.[2] We present a case of acute, symptomatic hyponatremia precipitated by fasting and breastfeeding, followed by copious fluid intake and NSAID ingestion.

Case Report

A 24-year-old woman presented to the emergency department (ED) in August with headache and nausea. She had a normal spontaneous vaginal delivery 1 month prior to admission. For religious reasons, she had fasted (abstaining from both food and water) for roughly 20 hours on the day prior to admission. She continued to breastfeed her infant while fasting and denied vomiting or diarrhea. She developed a mild headache throughout the day and noted decreased milk supply. After ending her fast, she consumed 2 L of water, 350 mL of Gatorade, and took 400 mg of ibuprofen. Over the next 4 hours, her headache abruptly worsened and she developed severe nausea. She then drank another 1.6 L of water, took another 400 mg of ibuprofen, and consumed a small amount of vegetable soup and pretzels. She reported that her consumption of water was not driven by thirst but by a desire to increase milk supply and a belief that she was still dehydrated.

Due to her severe headache, nausea, and weakness she presented to the ED. Her urine specific gravity upon admission was 1.015. She was given 1 L of normal saline for presumed migraine. At this point, her sodium was found to be 124 mEq/L. Initial laboratory values are detailed in the Table. Cortisol and thyroid function test results were normal. She then developed copious urine output though the exact amount was not quantified. Her urine osmolality was found to be 81 mOsm/kg, with urine sodium, potassium, and chloride below the level of detection. Twelve hours after admission,

Abbreviations: AVP, arginine vasopressin; ED, emergency department; NSAID, nonsteroidal anti-inflammatory drugs.

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Table
Laboratory Values

Test	Reference	On admission	8 h after admission	12 h after admission	16 h after admission
Sodium	137-145 mmol/L	124	130	139	142
Potassium	3.5-5.1 mmol/L	3.7	4.2		
Chloride	98-107 mmol/L	87	96		
Bicarbonate	19-27 mmol/L	22	23		
Blood urea nitrogen	7-26 mg/dL	7	5		
Creatinine	0.50-0.95 mg/dL	0.63	0.59		
Glucose	75-100 mg/dL	101	99		
TSH	0.41-4.81 mIU/L	2.48			
Free T4	0.83-1.90 ng/dL	0.86			
Cortisol	2.5-19.5 ug/dL	9.4			
Hemoglobin	11.2-14.7 g/dL	11	12.3		
Hematocrit	33.8-43.3 %	32.1	35.8		
Urine specific gravity	1.015				
Serum osmolality	275-295 mOsm/kg		269		
Urine osmolality	50-1200 mOsm/kg		85		
Urine Na	mmol/L		<20		
Urine K	mmol/L		<3		
Urine Cl	mmol/L		<20		
Urine Cr	mg/dL		7.1		

Abbreviations: Cl = chloride; Cr = creatinine; K = potassium; Na = sodium; TSH = thyroid stimulating hormone; T4 = thyroxine.

her sodium had increased to 139 mEq/L. She was given 5% dextrose in water to slow the rise of her serum sodium but was not given desmopressin. Her sodium rose to 142, where it remained stable for the duration of her hospitalization. During follow-up phone calls at 2 days and at 2 months after presentation she reported no neurological deficits and no recurrence of similarly severe headaches.

Discussion

Here, we report a case of a 24-year-old woman presenting with hyponatremia. Despite the moderate severity, she had significant symptoms, probably related to the acuity of onset.[3] The presentation was most consistent with appropriate elevations of arginine vasopressin (AVP) due to hypovolemia and enhanced sensitivity to the AVP due to NSAIDS. A number of factors likely contributed to this patient's hypovolemia, including fasting, increased fluid and solute losses due to nursing, sweating during the hot summer, and small body size.

Insensible losses of water are significantly increased in the summer compared to winter months.[4] This patient's insensible water losses in a 20-hour summer fast were approximately 1000 mL, which were not replenished due to her ongoing fast from both food and liquids.[5] In addition, she lost an additional 700 to 750 mL due to milk production,[6] though, notably, elevated prolactin does not itself cause hyponatremia.[7] Cumulatively, these fluid losses likely led to hypovolemia, triggering nonosmotic AVP stimulation.

After ending her fast she then consumed roughly 4 L of water with a relatively small amount of solute. In this relatively small woman of 58 kg, her total body water is roughly 29 L (0.5 x 58 kg).[8] Assuming a high-normal sodium of 142 mEq (after 20 hours of fasting with hypotonic fluid loss) prior to water ingestion, her total body sodium would be 4118 mEq. After consumption of roughly 4 L of water, this would be diluted in a total of 33 L, which results in a serum sodium concentration of 124 mEq/L. This acute development of hyponatremia led to symptoms of mild cerebral edema, severe headache, and nausea.

Another contributing factor to her hyponatremia was her consumption of NSAIDs in addition to fluid. NSAIDs inhibit prostaglandin synthetase activity, thereby potentiating the renal effects of AVP.[9] The ongoing renal effects of AVP upon presentation to the ED were evidenced by her concentrated urine specific gravity, though urine electrolytes and osmolality were not checked initially. Due to ongoing AVP stimulus, she was unable to excrete the water she consumed, resulting in dilution of her serum sodium.

One liter of normal saline replaced her intravascular volume depletion, which removed AVP stimulus, and no further NSAIDs were given. This led to the excretion of highly dilute urine, as evidenced by subsequent low urinary osmolality and copious urine output as well as the rapid rise of serum sodium. Her low urine sodium and chloride (checked after administration of normal saline) was not due to the upregulated renin-angiotensin-aldosterone system, but rather the large amounts of free water in the urine, diluting the concentration of these electrolytes.

In this patient's case, her serum sodium was not relowered despite the rapid rise from 124 to 139 mEq/L in 12 hours. First, it was noted that as the patient's copious fluid intake occurred in the 4 hours prior to admission, the development of hyponatremia was likely acute (developing in under 48 hours). Thus, according to the US expert panel recommendations, there was no need to restrict the rate of sodium rise.[3] An additional rationale was that her sodium nadir was 124 mEq/L, and osmotic demyelination is exceptionally rare with a sodium nadir above 120 mEq/L.[10]

It should be noted that a serum osmolality was not checked, and thus pseudohyponatremia cannot be definitively excluded. However, she did not have any of the typical causes of hyperosmolar hyponatremia, such as severe hyperglycemia or mannitol administration, nor was she known to have any of the typical causes of pseudohyponatremia, such as hypertriglyceridemia or paraproteinemia. Additionally, her improvement in serum sodium with saline administration further suggests that her hyponatremia was hypo-osmolar hyponatremia.

Conclusion

Patients undertake fasts for a variety of reasons and may consult physicians prior to undertaking a particularly rigorous fast. Physicians need to be aware of patient physiology and the particular risks that fasting poses to certain demographics in order to provide individualized counseling. Clinicians should be cognizant that, after a period of fasting, patients are prone to hyponatremia unless sodium is replaced along with fluids. Furthermore, small women, particularly those losing volume from lactation or diarrhea, are at high risk of developing both acute hyponatremia and severe

sequelae if water is ingested in vast excess to solute. Additionally, patients should be counseled to avoid NSAIDs in the immediate postfast period, as this will prolong the effects of AVP.

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