Case in Point

High-Speed Hyponatremia: Exercise-Associated Hyponatremia in a Young Male Soldier

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The importance of recognizing and treating exercise-induced hyponatremia as soon as possible cannot be underestimated. This case report examines the effects of serum salt dilution after extreme physical activity.

A 26-year-old male soldier in advanced individual training lost consciousness and experienced an acute mental status change during a long march on a warm day. He was found to be acutely hyponatremic with an elevated core body temperature. He was treated and his condition improved. He was admitted to the hospital and his condition was diagnosed as exercise-associated hyponatremia (EAH), heat-related injury, and mild exertional rhabdomyolysis. The pathophysiology of EAH is described along with current guidelines of treatment and prevention.

CASE REPORT

A 26-year-old, 160 lb, white male soldier lost consciousness after 3 hours of marching in heavy gear. Ambient temperature was approximately 80ºF. Rectal temperature at that time was 105ºF, and he was brought to the emergency department (ED). On presentation, he had regained consciousness but was not oriented to person, place, or time. He was diaphoretic and had multiple red papules on his lower extremities, suggesting possible tick bites. Pupils were equal and reactive. Auscultation of the lungs revealed crackles over the lung bases. A grade 2 systolic murmur was also noted. Vital signs and laboratory workup from initial assessment are presented in Table 1. The patient's urine test for blood was positive, without red blood cells, and did not show signs of drug use or infection. A portable anteroposterior (AP) chest radiograph performed in the ED showed scattered multifocal parenchymal opacities and blunted costophrenic angles, suggestive of early pulmonary edema or pneumonia (Figure 1). The patient was given oxygen and a bolus of 2 L normal saline then begun on a continuous intravenous (IV) infusion of normal saline. Doxycycline and levofloxacin were administered to treat suspected pneumonia and tickborne illnesses.

The patient slowly became more oriented, and after 4 hours he was coherent enough to give a history. He stated he had no past medical or surgical history. He did not smoke or use prescription or illicit drugs. He had not experienced headache, dyspnea, chest pain, abdominal pain, or nausea. He said he did not feel ill before the march. He stated that he felt fine except for a mild, nonproductive

Table 1. Vital signs and laboratory values at presentation

Vital Sign	Lab value
Blood pressure	123/66 mm Hg
Heart rate	92 bpm
Respiratory rate	28 bpm[a]
Temperature	99.5ºF (37.5ºC)
O_2 saturation	85% on room air[a]
Sodium	122 mmol/L[a]
Chloride	91 mmol/L[a]
Potassium	4.2 mmol/L
Carbon dioxide	24 mmol/L
BUN	18.0 mg/dL
Creatinine	0.9 mg/dL
Glucose	109 mg/dL
White blood count (WBC)	7.6 x 10^3/µL
Hemoglobin	12.3 g/dL[a]
Platelets	209 x 10^3/µL

[a]Value is outside normal range.

Normal values: sodium,135-145 mmol/L; chloride, 98-107 mmol/L; potassium, 3.5-5.1 mmol/L; carbon dioxide, 22-30 mmol/L; BUN, 7-18 mg/dL; creatinine, 0.6-1.2 mg/dL; glucose, 70-115 mg/dL; WBC, 4.5-11.0 x 10^3/µL; hemoglobin 13.5-17.5 g/dL; platelets, 150-450 x 10^3/µL.

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cough that was not present before his loss of consciousness. He had not eaten anything before the march that morning. He stated that during the march he drank as much as possible, consuming about 5 quarts of water. A physical examination at this time revealed no focal neurologic deficits. Repeat serum chemistry showed a serum creatine phosphokinase (CPK) of 1,345 U/L (normal 55-170) and a serum sodium of 136 mmol/L. He was admitted to the hospital, and an echocardiogram, ordered to evaluate his murmur, revealed normal ventricular function with mildly dilated left atrium and right ventricle and a trivial pericardial effusion.

The next day the patient appeared well, alert, oriented, and his cough had resolved. He stated he felt fine. His neurologic examination was again normal. His serum sodium was 137 mmol/L, and his serum CPK was 1,369 U/L. Blood urea nitrogen and creatinine were normal, as were his liver function tests. Repeat posterior-anterior (PA) and lateral chest radiographs showed improvement in the pulmonary edema (Figure 2). The patient was discharged the next day. Follow-up visits to his primary care provider over the next several weeks showed full recovery. The patient's electrolyte levels normalized, and there was no evidence of permanent organ damage or neurologic sequelae.

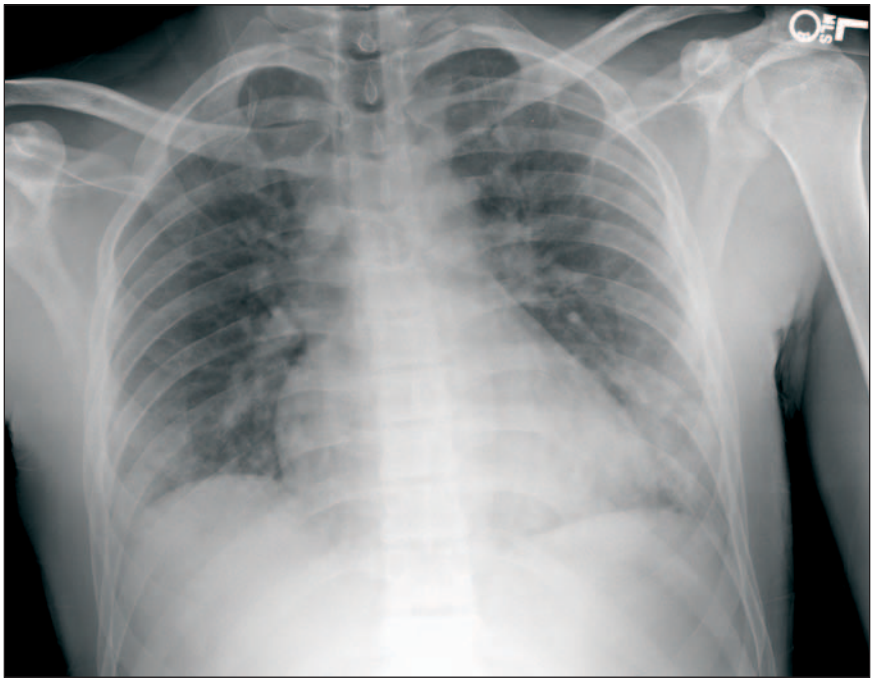

Figure 1. Portable AP chest radiograph taken in the ED. Radiology report indicated scattered multifocal parenchymal opacities and blunted costophrenic angles, suggestive of early pulmonary edema or pneumonia.

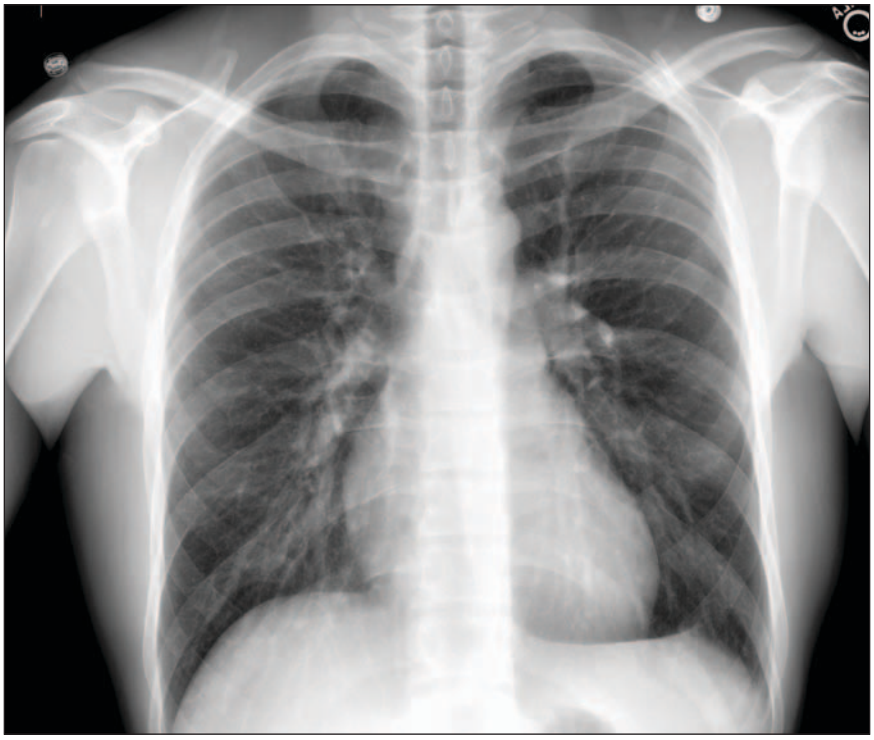

Figure 2. PA chest radiograph the day after admission. Radiology report indicated findings of resolved pulmonary volume overload and clear lungs.

DISCUSSION

The patient presented with EAH. He also presented with comorbid conditions of heat-related injury and mild exertional rhabdomyolysis. Of these conditions, hyponatremia was his most immediate life-threatening condition and the focus of our discussion.

EAH is hyponatremia occurring ≤ 24 h after an endurance event. EAH is relatively common during multihour endurance events. In one study

of runners in the Boston marathon, about 13% of the runners developed EAH, and about 0.6% developed critical hyponatremia (serum sodium ≤ 120 mmol/L).[1] Many health care providers have little knowledge of EAH, its presentation, or how to treat it. It is important that physicians who treat endurance athletes acutely recognize the clinical presentation of EAH and understand the relevant pathophysiology underlying its development. This practice allows rapid and rational treatment, as well as effective recommendations to athletes so they can prevent EAH from recurring.

In 2005,[2] and again in 2007,[3] an international expert panel generated a consensus statement on EAH. Their recommendations follow.

CLINICAL PICTURE OF EAH

Although hyponatremia is usually defined as a serum sodium concentration of < 135 mmol/L, symptoms of EAH are not usually severe at concentrations above 130 mmol/L. In the early stages of hyponatremia, patients may present with nonspecific signs and symptoms such as bloating, weakness, dizziness, nausea, vomiting, or headache. Later signs and symptoms include cerebral edema, altered mental status (eg, confusion, delirium, obtundation), seizures, noncardiogenic pulmonary edema (which may be noticed as respiratory distress), stupor, coma, and eventually death (Table 2).[4] Serum sodium should be measured in all patients presenting with any of these signs and symptoms in the context of an endurance event.[5]

The 2nd International EAH Consensus Panel[3] identified several risk factors associated with EAH (Table 3).

PATHOPHYSIOLOGY OF EAH

EAH occurs when volume of fluid intake exceeds volume lost, diluting the blood and causing serum osmolality to fall. The normal physiologic response to hypervolemia and hyposmolality is to suppress antidiuretic hormone (ADH). This causes the kidneys to excrete free water, which relieves the circulation of excess volume and raises serum osmolality.

During prolonged exercise, ADH may not be fully suppressed.[6,7] This can result in free water retention and dilutional hyponatremia when excessive fluid is consumed during lengthy athletic activity. When the serum sodium concentration falls acutely, an osmotic gradient is created between serum and the brain, favoring a fluid shift into the brain. The resultant cerebral edema accounts for the mental status and neurologic changes seen in EAH.

Sodium depletion has not been proven to cause EAH; sodium loss is similar in athletes who develop EAH compared with those who do not.[8] Electrolyte-containing sports drinks have not been proven to prevent EAH from occurring with excessive drinking, likely because conventional sports drinks are still hypotonic when compared with blood.[1,3]

TREATMENT OF EAH

Patients with EAH may be symptomatic or asymptomatic. Generally, asymptomatic hyponatremia is not detected unless serum electrolytes are assessed for some other reason. Individuals found to have asymptomatic hyponatremia following an endurance event should be advised to monitor the possible effects of EAH for 24 h after as well as in future endurance events. Fluid intake should be restricted until the onset of urination. They should not be given isotonic or hypotonic fluids, because this can worsen the degree of hyponatremia or cause fluid overload. If symptoms appear, treatment should be initiated as follows.

Table 2. Signs and symptoms of EAH[4]

Early
Bloating
Weakness
Dizziness
Nausea and vomiting
Headache
Late
Cerebral edema
Altered mental status
Pulmonary edema
Seizures
Coma
Death

Table 3. Risk factors associated with EAH[3]

Athlete related
Excessive drinking behavior
Weight gain during exercise
Low body weight
Female sex
Slow running or performance pace
Event inexperience
Nonsteroidal anti-inflammatory agents
Event related
High availability of drinking fluids
> 4 h exercise
Unusually hot environmental conditions
Extreme cold temperature

Patients with symptomatic EAH can be recognized by their clinical picture. Treatment should begin as soon as the condition is recognized and therapy is available, no matter the location. IV access should be obtained and high-flow oxygen begun. The serum sodium concentration should be quickly measured and treatment begun as soon as the condition is diagnosed. EAH emergency treatment

Continued on page 22

Continued from page 18

consists of rapid administration of hypertonic saline, given as a 100-mL IV bolus of 3% NaCl. Up to 2 more boluses can be given at 10-minute intervals if the symptoms do not improve. Isotonic and hypotonic fluids are contraindicated in these patients because they have been associated with poor outcomes, including worsening hyponatremia, cerebral herniation, and death.[5] It is important to note that although our patient recovered after he received a normal saline bolus, this treatment is not recommended. ADH receptor antagonists are not recommended for treatment of life-threatening EAH. Their role in milder forms of EAH is unclear.[3]

We are not aware of any cases of osmotic demyelination (ie, central pontine myelinolysis) resulting from rapid correction of acute hyponatremia, so treatment should not be delayed because of this concern.[3,9] The more immediate danger to athletes is cerebral herniation unless the cerebral edema is rapidly corrected with 3% saline. Any saline solution with a concentration < 3% is not sufficiently hypertonic to treat hyponatremic encephalopathy.[5]

PREVENTION OF EAH

For many years athletes have been encouraged to hydrate as much as possible during endurance events, especially in the heat. The rationale behind this is clear since dehydration can be a significant threat to their performance and health. In recent years, the dangers of overhydration have also become apparent. It is important that we educate athletes and their leaders that while they need to stay hydrated, more is not always better.

Since EAH is a dilutional hyponatremia, athletes should avoid overconsumption of water or sports drinks before, during, and after exercise.[8] Fluid retention is manifest as weight gain or even a lack of weight loss during exercise. During endurance exercise athletes should expect to lose about 2% of body weight. A simple and inexpensive way to provide information about the athletes' fluid status is the common practice of weighing athletes before, after, and even during an endurance event.

Electrolyte-containing sports drinks do not prevent EAH in the presence of overconsumption of fluids.[1] Sodium supplementation may be beneficial, but its use is controversial. The mainstay of prevention of EAH is to limit fluid intake. Athletes should be counseled on the dangers of overconsumption of fluid and encouraged to drink to their level of thirst. ●

Author disclosure

The author reports no actual or potential conflicts of interest with regard to this article.

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