Severe hyponatremia develops in a runner following a half-marathon

It's essential to know how to distinguish dehydration from fluid overload in endurance athletes, because the treatment for one can greatly worsen the other.

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CASE

A 34-year old woman sought nutritional guidance at our clinic after what she described as a recent episode of dehydration. Medical records were obtained, and the following clinical picture emerged. A month earlier, the patient had participated in a half-marathon running event (21 km, 13.1 miles) conducted in extreme heat (33°C, 100°F). Her training had been limited to exercising on a treadmill 1 to 2 days per week, not exceeding 15 km (9.3 miles) weekly, in air conditioning. Beginning 3 days before the race, she experienced symptoms of viral illness, including nausea and diarrhea, but ran the race regardless.

She remembered drinking copious amounts of water before the race and approximately another 4 liters of water during the race. Upon returning home, she felt nauseous and confused and, assuming her symptoms were due to the heat, continued drinking. A neighbor brought her to the emergency department (ED) after finding her disoriented.

Initial ED notes, made approximately 2 hours after the race, note wrist swelling, foggy mentation, BP of 123/80 mm Hg, temperature of 36°C (96.8°F), and normal skin turgor and color. The patient complained of nausea and chest pressure. Initial laboratory test results are shown in Table 1 (page 28) and were remarkable for the serum sodium level of 119 mEq/L, as well as for the below-normal values for hemoglobin, the hematocrit, albumin, and calcium. The diagnosis was dehydration/hyponatremia, and treatment consisted of 800 mL normal (0.9%) saline and observation. Within 5 hours, the patient suffered a grand mal seizure and then displayed combative behavior; she was infused with approximately 2 more liters of normal saline. During her 8 hours in the ED, the patient produced only 200 mL of highly concentrated urine. She was transferred to the ICU, and hypertonic (3%) saline was started at 25 mL/hour. In the ICU she suffered a second grand mal seizure, which was managed without intubation (see Figure 1, page 29).

The day after being admitted to the ICU, the patient complained of generalized body aches; 2.8 liters of normal saline was infused, and 5.4 liters of urine were diuresed. Creatine phosphokinase (CPK) and potassium were elevated, and a diagnosis of rhabdomyolysis was made. She was treated with half-normal (0.45%) saline at 200 mL/h. On day 4, the CPK peaked at 22,830 U/L and then declined to normal limits (less than 225 U/L) by day 7. The patient was discharged on day 8 with instructions to seek nutritional counseling before returning to running.

The client sought nutritional advice in our sports medicine institute a month after being discharged. She was urged to limit fluid intake to no more than 1% of her body weight per hour during exercise, unless thirsty, and to

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TABLE 1. Initial laboratory values

Albumin	2.9 g/dL*
BUN	8 mg/dL*
Calcium (total)	7.8 mg/dL*
Creatine phosphokinase	215 U/L
Creatinine	0.4 mg/dL*
Hematocrit	31.5%*
Hemoglobin	10.7 g/dL*
Sodium	119 mEq/L*
Urinary osmality	918 mOsm/L
Urinary sodium	131 mEq/L

* Denotes abnormal value.

ingest sodium-containing beverages and foods before and during prolonged exercise. Also, the importance of heat acclimatization and adequate training were stressed.

DISCUSSION

Studies of long-distance runners, cyclists, and triathletes have found that approximately 5% to 30% of participants may develop hyponatremia during their events, although most will be asymptomatic.[1-3] Eight fatalities have been reported from encephalopathy caused by exercise-associated hyponatremia (EAH) in recent years, all in the United States.[2]

Among the risk factors for EAH are excessive fluid consumption during and after athletic events, female gender, low body weight, slower race times (as slower athletes will have more opportunity to drink), use of NSAIDs, and lack of heat acclimatization.[4,5] In the past, exercisers have been instructed to drink before feeling thirsty to avoid dehydration, but this advice appears to have led some to overload with fluid.[6]

Three abnormalities are thought to be necessary for EAH to develop: excess fluid intake, syndrome of inappropriate antidiuretic hormone (ADH) secretion, and a failure to mobilize bodily stores of sodium.[2] In our patient's case, high levels of ADH, stimulated during her recent viral illness, may have contributed to the failure to excrete the excessive amount of fluid she had ingested. Normal skin turgor, absence of postural hypotension, and swelling of the wrists suggested excess fluid rather than dehydration, as did the decreased values of most blood parameters.

Dehydration versus EAH Given the catastrophic consequences of inappropriate fluid administration, emergency caregivers should be familiar with the symptoms of dehydration versus those of EAH. Exercise-associated hyponatremia often becomes symptomatic after the event, as water continues to be absorbed from the gut, when hyperthermia is unlikely to be a problem. The classic presentation of athletes with mild EAH (serum sodium, 125-130 mEq/L) includes nausea, vomiting, lightheadedness or feeling "out of it," lethargy, and edema (particularly of the ring fingers and wrists, which suggests fluid-overload).[7] As plasma sodium falls further, mental status worsens and encephalopathy, seizures, pulmonary edema, or death may occur. In contrast, the symptoms displayed by athletes suffering from dehydration primarily involve postural hypotension (which can easily be alleviated by elevation of the legs while supine); this hypotension generally occurs immediately following cessation of exercise.[6] Dehydrated patients typically also present with sunken eyeballs, thirst, difficulty spitting, and poor skin turgor.[8]

The body aches described by the patient the day after admission are typical during rhabdomyolysis. Persons with rhabdomyolysis may present with cramping muscle pain, muscle swelling and tenderness, progressive weakness, and discolored urine.[9] Rhabdomyolysis has been linked to hyponatremia.[10-11] It has been suggested that acute hyponatremia leads to intracellular swelling.[10] Rapid changes in the intracellular osmolyte concentration during the correction of hyponatremia may lead to membrane disruption and to the leakage of enzymes seen in rhabdomyolysis.[11]

Treatment of EAH Patients presenting with a scenario suggestive of either dehydration or fluid overload after exercise should be questioned closely regarding their fluid intake and output, their level of heat acclimatization, and their level of competitiveness, because faster athletes are less apt to overhydrate. Prompt assessment should be made of body tempera-

TEACHING POINTS

- Approximately 5% to 30% of long-distance runners, cyclists, and triathletes may develop hyponatremia during their events, although most will be asymptomatic. Eight fatalities have been reported from encephalopathy caused by exercise-associated hyponatremia (EAH) in recent years, all in the United States.
- Given the catastrophic consequences of inappropriate fluid administration, emergency caregivers should be familiar with the symptoms of dehydration versus those of EAH.
- Most cases of mild hyponatremia do well with monitoring until diuresis occurs, which can take hours. The bladder should be catheterized to assure that urine is dilute and is being produced at increasing rates during recovery.
- More severe cases of hyponatremia may be treated with diuretic agents and hypertonic saline. Administration of normal saline will increase fluid overload and should be avoided.

COMPETENCIES

- ●●●●● Medical knowledge
- ● Interpersonal & communication skills
- ●● Patient care
- ● Professionalism
- ●● Practice-based learning and improvement
- ● Systems-based practice

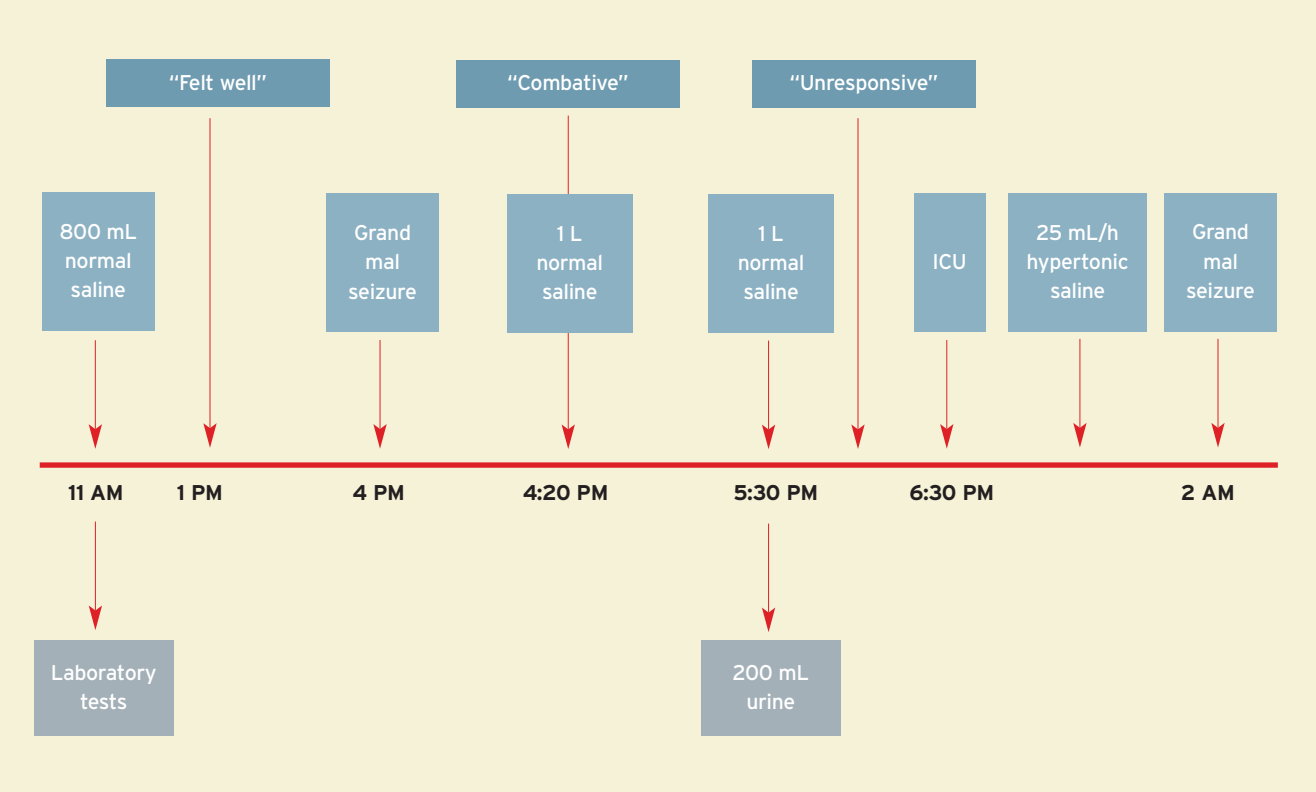

FIGURE 1. Sequence of events

ture and plasma electrolytes. In the absence of clinical signs of dehydration, oral or IV fluids are not recommended.[7] Most cases of mild hyponatremia do well with monitoring until diuresis occurs, which can take hours.[6,7] The bladder should be catheterized to assure that urine is dilute and is being produced at increasing rates during the recovery period. Salty foods can be given.[7] The clinician should be aware that fluid sequestered in the gut during exercise and absorbed after exercise cessation may prolong recovery time,[4] especially because renal mechanisms for clearing fluid may be initially impaired.

More severe cases of hyponatremia may be treated with judicious use of diuretic agents, such as loop diuretics or mannitol,[6] and with hypertonic saline at a rate of 25 mL/h.[12] Administration of normal saline will increase fluid overload[13] and should be avoided. Rapid correction of hyponatremia may lead to pontine myelinolysis, and correction rates should not exceed 1 to 1.5 mEq/L/h.[6]

Given the growth of recreational activities such as running and triathlons in recent years, emergency personnel are increasingly likely to be confronted with cases of EAH. They may be tempted to assume that a patient who reports prolonged exercise in the heat is dehydrated and to initiate treatment with IV normal saline, as was done here. A basic understanding of the typical etiology and presentation of fluid overload versus dehydration in the athletic setting is crucial to the prompt initiation of appropriate treatment. JAAPA

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