

[Primary Care]

Exercise-Associated Hyponatremia in an Ultra-Endurance Mountain Biker: A Case Report

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Symptomatic exercise-associated hyponatremia (EAH), which is relatively common among marathon runners, is an uncommon event among ultra-endurance athletes. A 44-year-old man presented to the emergency department with increased thirst after successfully completing a 100-mile mountain bike race in Leadville, Colorado. Initial laboratory tests revealed a blood sodium level of 116 mEq/L. The primary etiologic factor in EAH is fluid consumption in excess of fluid losses in prolonged exertion. Early diagnosis and management is crucial to prevent cerebral and pulmonary edema.

Keywords: exercise-associated hyponatremia; ultra-endurance; mountain bike

Exercise-associated hyponatremia (EAH) can occur during or up to 24 hours after prolonged exertion.^{3,8} It is one of the most important medical problems in endurance events such as marathons, triathlons, and ultra-endurance events.^{5,7,8}

CASE REPORT

A 44-year-old man presented to the emergency department (ED) with increased thirst, nausea, and shakiness 6 hours after completing a 100-mile mountain bike race in Leadville, Colorado, in August. The course altitude ranges from 2,804 to 3,840 m. He finished the race just under the 12-hour cut-off time. This was his first attempt in completing this race. He does participate in long-distance mountain bike races, but never had previous symptoms such as leg cramping and dizziness as he had during the last 2 hours of this race. He had been dealing with cramping 2 months prior, which typically manifested after 5 to 6 hours of riding. He did not visit any of the aid stations during the race. He had 3 episodes of mild diarrhea the day before the race and denied any vomiting, black stools, or difficulty urinating during the race. He reported normal color urination at least 2 to 3 times during the race. The average temperature and humidity were 13.9°C (3°C-25°C) and 42% (13%-71%), respectively, on race day. He drank

approximately 15,550 mL of water and 7100 mL of electrolyte fluid (approximately 2.8 g of sodium) plus 10 salt tablets (approximately 1.9 g of sodium) during the race. He felt very thirsty after the race and had half of a burger with a few French fries and a few sips of beer for dinner. Then, he drove with his wife back to Denver (a 2-hour drive). He had another 6750 mL of water and 0.55 g of salt after completing the race before going to the ED. Thus, he ingested roughly 29 L of water and 5.25 g of sodium during and after the race. His wife took him to the ED because of his continuous nausea and shakiness.

Past medical history was positive for exertional cramping with training. He was not taking any medications on a regular basis. He denied taking any nonsteroidal anti-inflammatory drugs (NSAIDs) during the race but took 400 mg of ibuprofen afterward for back soreness. His vital signs were unremarkable with weight of 83.9 kg and height of 175.3 cm (body mass index, 27 kg/m²). He appeared alert, anxious, and in mild distress. Other than a slight confusion on his arrival to the ED, there was no alteration in mental status with no motor or sensory deficit. Otherwise, his neurological and remaining physical examinations, including respiratory, were unremarkable.

Initial laboratory tests in the ED included: sodium 116 mEq/L, glucose 102 mg/dL, phosphorus 2.4 mg/dL, magnesium 1.6 mg/dL, TSH 1.8 microIU/mL, urine osmolality 115 mOsm/kg, serum osmolality 249 mOsm/kg, urine sodium 31 mEq/L,

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Table 1. Blood test results and IV fluids given in the hospital

Tests (Reference Range)	5 Hours After the Race	IV Fluids Given	10 Hours After the Race	IV Fluids Given	21 Hours After the Race	2 Months After the Race
Sodium (136-145 mEq/L)	116	2000 mL of 0.9% NaCl	127	200 mL of 3% NaCl; 1000 mL of 0.9% NaCl	137	133
Potassium (3.6-5.1 mEq/L)	2.6		3.2		4.6	4.9
Calcium (8.9-10.3 mg/dL)	8.7		7.6		7.5	9.6
BUN (6-20 mg/dL)	19		17		17	10
Creatinine (0.64-1.27 mg/dL)	1.36		1.06		1.29	1.15
Creatinine kinase (24-195 U/L)	1955		1860			

hemoglobin 13.6 g/dL, and hematocrit 37.9% (Table 1). He was initially treated with 0.9% normal saline, but this was correctly changed to 3% hypertonic saline, of which 200 mL was given. He was given another liter of 0.9% normal saline over 6 hours after this bolus. He was given supplemental potassium chloride (40 mEq), magnesium sulfate (2 g), and calcium gluconate (1 g) in his IV fluid. He recovered fully, and his blood sodium level increased to 127 and 137 mEq/L at 5 and 16 hours after initial check, respectively (Table 1). His mild confusion and anxiety at the time of initial presentation resolved rapidly with normalization of his hyponatremia. He did not develop seizure or neurological deterioration. He was discharged home in stable condition approximately 20 hours after initial presentation to the ED.

He was advised against drinking excessive water during long exercises and advised to take sodium supplementation during exercises lasting more than a few hours. He gradually eased back into normal daily function and mountain biking and has not had an exacerbation. His follow-up laboratory tests 2 months later, including comprehensive metabolic, hematologic, thyroid, and lipid panel, were completely normal except sodium, which was 133 mEq/L (Table 1).

DISCUSSION

Previous EAH symptomatic cases in cyclists have been reported in a road race² and laboratory setting,¹ and an asymptomatic case has also been described.⁶ The primary

pathophysiological mechanism seen in this case appeared to be dilutional.³ The cyclist ingested ~228 mEq of sodium during his 12-hour ride, with 29 L of fluid; he likely exceeded his sweat rate while cycling ~8 mph in temperate ambient conditions (~14°C). A fluid ingestion rate of a 1.9 L/hour with an average sodium concentration of 8 mEq/L would have lowered this cyclist's blood sodium level to a critically low level of 109 mEq/L (Figure 1). Although this estimation does not include fluid output data (which were not available) and assumes a baseline blood sodium level similar to that reported during a routine check-up 2 months after hospitalization (133 mEq/L), these rough calculations highlight the higher contribution of excessive fluid intake over the presumed protective effects of sodium ingestion during cycling exercise.¹⁰

Other important and novel features of this case include: (1) a nonosmotically stimulated thirst sensation; (2) lack of severe central nervous system symptomatology, other than confusion, despite critically low blood sodium concentrations (116 mEq/L); and (3) verification that acute EAH can be treated aggressively with hypertonic saline without adverse neurological consequences (central pontine myelinolysis from a blood sodium increase of 21 mEq/L during a 16-hour recovery period). Furthermore, it remains unclear why the incidence of EAH in endurance cycle races⁹ is remarkably low compared with much higher incidences (30%) during endurance running events.⁴ These contradictions, combined with the novel findings presented here, highlight the complexity of EAH during different modes of activity.

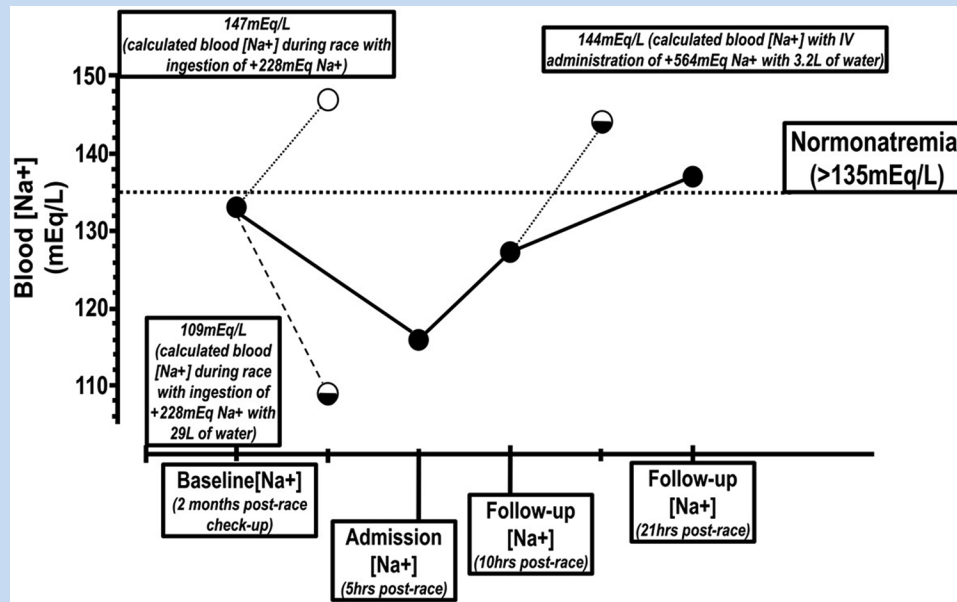


Figure 1. Actual (filled circles) and estimated blood sodium concentrations ($[Na^+]$) from input data of sodium (open circle) and water ingestion (half-filled circles) during the race (oral) and in the hospital (intravenous; IV). Sodium and water output data (urine, sweat, and respiratory losses) were not available. All sodium ingestion was assumed to enter the extracellular fluid compartment (ECF). All water ingested and administered was assumed to equilibrate equally between all total body water (TBW) compartments. Estimated TBW was calculated as 60% of reported body weight (83.9 kg), with estimations of ECF fluid volume distribution and input calculated as 20% of body weight (one third of TBW is located within the ECF).

CONCLUSION

EAH is one of the most significant possible medical conditions among endurance athletes. The primary etiologic factor in EAH is fluid consumption in excess of fluid losses in prolonged exertion. Although less common in cycling, sports medicine providers should be aware that EAH can develop in these athletes. The treatment of choice for dilutional EAH is prompt administration of hypertonic—not isotonic—saline without risk of osmotic demyelination because EAH is an acute hyponatremia.

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