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CASE REPORT

Hiker Fatality From Severe Hyponatremia in Grand Canyon National Park

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We present the case of a hiker who died of severe hyponatremia at Grand Canyon National Park. The woman collapsed on the rim shortly after finishing a 5-hour hike into the Canyon during which she was reported to have consumed large quantities of water. First responders transported her to the nearest hospital. En route, she became unresponsive, and subsequent treatment included intravenous normal saline. Imaging and laboratory data at the hospital confirmed hypervolemic hyponatremia with encephalopathy. She never regained consciousness and died of severe cerebral edema less than 24 hours later. We believe this is the first report of a fatality due to acute hyponatremia associated with hiking in a wilderness setting. This case demonstrates the typical pathophysiology, which includes overconsumption of fluids, and demonstrates the challenges of diagnosis and the importance of appropriate acute management. Current treatment guidelines indicate that symptomatic exercise-associated hyponatremia should be acutely managed with hypertonic saline and can be done so without concern over central pontine myelinolysis, whereas treatment with high volumes of isotonic fluids may delay recovery and has even resulted in deaths.

Key words: arginine vasopressin, cerebral edema, exercise-associated hyponatremia, hypertonic saline, syndrome of inappropriate ADH secretion, water-electrolyte imbalance

Introduction

Exercise-associated hyponatremia (EAH) has been long recognized as a possible complication of endurance exercise that can have a fatal outcome.[1–5] The first documented EAH death was of a marathoner in 1993,[4] and EAH has also been identified as a potential problem among hikers for more than 2 decades.[6–14] As far as we are aware, the present report is the first documented fatal case of EAH in a hiker. We share this case because proper initial management of EAH can be critical to the outcome, and there is evidence that current treatment guidelines for EAH have yet to be universally adopted.[9,13,15–24]

Case presentation

On September 3, 2008, a previously healthy 47-year-old woman from London, England, hiked for approximately 10 km on the South Kaibab Trail in Grand Canyon National Park, descending and then ascending roughly 900 m in elevation over a 5-hour period. The day was clear and sunny. Temperatures were near 10°C at the start of the hike at approximately 0800 hours, and 26.6°C at the end of the hike near 1300 hours. During the hike, her husband reported she "drank a large amount of water and ate very little," but actual amounts are unknown. On arrival at the rim, she took a shuttle bus ride of approximately 15 minutes from the trailhead to Grand Canyon Village. Shortly thereafter, at 1400 hours, she had a syncopal episode, falling face forward onto a concrete sidewalk. According to her husband, she quickly regained consciousness, with no apparent head trauma. Grand Canyon National Park Service emergency medical service (EMS) responders were notified and arrived on the scene within 8 minutes of the call.

The patient was found sitting upright, awake but slow to respond to questions and complaining of a headache. She had no obvious signs of trauma to her head or elsewhere, and other than appearing lethargic, she had an unremarkable examination. An initial Glasgow Coma

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Score (GCS) was 15. Initial vital signs were stable, with blood pressure of 110/74 mm Hg, pulse of 70 beats/min, and respiratory rate of 16 breaths/min. Pulse oximetry was normal at 96%, and finger stick glucose was 82 mg/dL. An intravenous (IV) line was started, and the patient was placed in cervical spine precautions. At 1430 hours, 30 minutes after her syncopal episode, as ground ambulance transport began to the nearest hospital emergency department (128 km and 1.5 hours away), she abruptly sat upright, pulling out her IV line in the process. She then vomited a large amount of clear fluid (estimated 500 to 1000 mL) and immediately became unresponsive with a GCS of 8. She was noted to have sinus bradycardia immediately after the emesis, with a heart rate of 42 beats/min and blood pressure of 124/75 mm Hg. An electrocardiogram was unremarkable other than the bradycardia. An IV line was reestablished, and she was given 0.5 mg of IV atropine. The decision was made to expedite her transport by changing from ground to air ambulance, with the ground ambulance meeting an EMS helicopter at a highway location 48 km south of Grand Canyon Village where the transfer would be made. Over the next 30 minutes while in route to the transfer location, she received 600 mL IV normal saline. A nasopharyngeal airway was placed, and pulse oximetry was 99% on supplemental oxygen. At approximately 1540 hours, the patient and her care were transferred to the air ambulance EMS team. Her blood pressure and pulse remained stable, but her neurologic status had deteriorated to a GCS of 7. Her nasopharyngeal airway was replaced with an endotracheal tube. At approximately 1600 hours, she was on a life flight to the hospital 80 km away. An additional 400 mL IV normal saline was given before arrival at the hospital. Prehospital serum sodium testing was not done at any point because neither EMS crew was equipped with point-of-care blood analyzers at the time.

On arrival in the hospital emergency department at 1645 hours, the patient's blood pressure was 161/108 mm Hg, heart rate 76 beats/min, respiratory rate 16 breaths/min, temperature 33.3°C (method not recorded), and oxygen saturation 100% on ventilator. On physical examination, her head was found to be normocephalic and atraumatic. Her eyes showed fixed and dilated pupils at 5 mm. Lungs were clear, heart tones were normal, and no extremity edema was noted. A comprehensive metabolic profile showed the following: low serum sodium at 127 mmol/L (normal range 135–145 mmol/L), low potassium at 2.5 mmol/L, chloride 91 mmol/L, bicarbonate 22 mmol/L, glucose 204 mmol/L, anion gap 17, calcium 8.4 mg/dL, albumin 4.7 g/dL, total bilirubin 0.5 mg/dL, alanine aminotransferase 21 U/L, aspartate aminotransferase 33 U/L, alkaline phosphatase 60 U/L, blood urea nitrogen 13 mg/dL, creatinine 0.6 mg/dL, and slightly low calculated serum osmolality of 275 mOsm/kg. Creatinine kinase was 361 IU/L; complete blood count and cardiac enzymes and coagulation studies were normal. A Foley catheter was placed, and 2000 mL clear urine was obtained with specific gravity of 1.007. Urinalysis and urine toxicology were negative. Urine osmolality was not done. Severe closed head injury was considered part of the differential diagnosis, and a head computed tomography scan was obtained. It showed severe cerebral edema and impending herniation, but no evidence of trauma or intracranial hemorrhage. Cervical spine radiographs were normal.

During the 2 hours of treatment in the emergency department before transfer to the intensive care unit, an intensivist and neurosurgeon were consulted. The patient received 60 g IV mannitol and 40 mg IV furosemide, and was started on a 3% hypertonic saline drip at 40 mL/h. She was also provided IV normal saline at 125 mL/h. She was hyperventilated to a P_{CO_2} of 34 mm Hg, and the head of her bed was elevated to 25 degrees. Her neurologic status never improved. Although her serum sodium eventually corrected to 143 mmol/L a little more than 4 hours after admission, a cerebral flow study obtained early the next morning showed the absence of blood flow to the brain, confirming brain death. She was pronounced dead at 0838 hours from "severe cerebral edema from water intoxication resulting in uncal herniation and brain death," less than 19 hours after her syncopal episode.

Discussion

Exercise-associated hyponatremia is a potentially serious condition that is now generally known to be due to overhydration in combination with fluid retention from nonosmotic stimulation of arginine vasopressin (AVP) secretion.[25,26] Nonosmotic stimuli for secretion of AVP include intense exercise, nausea or vomiting, hypoglycemia, and nonspecific stresses such as pain and emotion.[27–29] Excessive losses of urine sodium due to secretion of brain natriuretic peptide may also contribute to the pathophysiology of EAH.[14,30,31]

Early symptoms of EAH include nausea, vomiting and headache, which can rapidly progress to confusion, altered mental status, seizure, and death if untreated.[26] Often, there is a delay in the presentation of symptomatic EAH after exercise cessation.[1,17,32] This delay is thought to be due to absorption of fluid remaining in the gastrointestinal tract once exercise has stopped and blood flow is redistributed away from skeletal muscles. The signs and symptoms of EAH encephalopathy are due to cerebral edema resulting from water following the