CASE REPORT

Exercise-Associated Hyponatremic Encephalopathy in an Endurance Open Water Swimmer

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Exercise-associated hyponatremia and its more serious form, known as exercise-associated hyponatremic encephalopathy, are recognized as some of the most important medical problems seen in a variety of different forms of endurance exercise. We describe a case of exercise-associated hyponatremic encephalopathy presenting as altered conscious state and seizures in a woman who had completed a 20-km open ocean swim. Her serum sodium measured approximately 1 hour after her seizure was 119 mmol/L on point-of-care testing. With ongoing critical care support and the use of hypertonic saline, she was able to be extubated the next day, neurologically intact, and ultimately was discharged from hospital without neurological sequelae. This case emphasizes both the importance of considering exercise-associated hyponatremic encephalopathy as a cause of neurological impairment in all athletes and the pivotal role of hypertonic saline in the treatment of this condition.

Key words: hyponatremia, exercise, open water swimming

Introduction

Exercise-associated hyponatremia (EAH) and its more serious form, exercise-associated hyponatremic encephalopathy (EAHE), have come to be recognized as two of the most significant medical risks of endurance exercise.¹ They have been reported for a broad range of wilderness activities, from ultramarathons to mountaineering.^{2–6} Open water endurance swimming has its own medical problems and is recognized as having a significant risk of causing hypothermia.⁷ Too date, however, it has been associated with only one report in the literature of EAH⁸ and none of EAHE. We report a case of life-threatening EAHE occurring in a long-distance open ocean swimmer.

Our report arises from the 2013 Rottneest Channel Swim. This event is an iconic, mass participation swim held in late summer, crossing the 20 km of open ocean between Perth, Western Australia, and Rottneest Island, which lies due west. All swimmers are accompanied by at least 1 paddlecraft and a larger support boat able to

provide food, water, and medication to the swimmer but no other support. To our knowledge, EAHE has not previously been reported for this event over its 20-year history.

Case Presentation

A 46-year-old woman completed the swim in 8 hours and 17 minutes in conditions that were typical for the event, with an air temperature of approximately 25°C and a water temperature of 22°C to 24°C. She had a history of asthma, no history of EAH, and reported starting the race in good health. Her only regular medication was norethisterone. In the 2 hours before the start of the race, she recalled drinking as much as 1000 mL of a sports drink. During the race, her intended fluid regimen as provided to her by her support team was 200 mL of fluid every 20 minutes, alternating between a sports drink (containing 18 mmol/L Na⁺) and water. It is not possible to be certain of the amount that she actually consumed. She vomited only once, at the 10-km mark, and her memories of the swim became hazy from about the 15-km mark. She reported no sensation of thirst throughout the race.

At event finish, she was triaged to the beachside medical tent because of concerns about her mildly

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altered mental state and severe nausea. She was noted to have mild hypothermia on an oral reading, and passive rewarming was begun with drying, blankets, oral glucose, and shivering. While in the medical tent, she was reported to have drunk approximately 2000 mL of a combination of sweetened sports drink, tea, and water, and although she did not feel thirsty, she was encouraged to drink by support staff. Her altered mental state and nausea did not improve with rewarming, and after 2 hours, she was transferred to the island nursing post by ambulance. The nursing post is the sole medical facility on the island and, on race day, is capable of advanced critical care as it is staffed by emergency physicians and critical care trained nurses. Her transfer to the nursing post was her last memory until waking up in hospital the next day.

On arrival at the nursing post, approximately 3 hours after finishing the race, she was pale, nauseated, and looked unwell but oriented and appropriate. Her baseline observations were heart rate 68 beats/min, blood pressure 116/62 mm Hg, respiratory rate 24 breaths/min, temperature 37°C, and oxygen saturation 97% on room air. Her chest was clear to auscultation. An electrocardiogram was normal, and a fingerprick blood glucose was 8.9 mmol/L, but no other biochemical testing or urinalysis was available. A normal saline infusion at 500 mL · h⁻¹ was begun, and metoclopramide, 10 mg intravenous (IV), was administered for nausea. Thirty minutes later, her verbal responses were noted to be slowed and answers to questions delayed; however, she was still oriented to time, place, and person. Fifteen minutes later, she was visibly disoriented and soon progressed to a generalized seizure. The seizure terminated spontaneously within 3 minutes; however, two 1-mg boluses of midazolam were required for persistent post-ictal agitation. The possibility of EAHE was recognized at this time by the emergency physicians, and the normal saline infusion was stopped, but there was still no capacity to measure blood sodium. Arrangements were made for aeromedical retrieval to the mainland; before that, she was intubated and ventilated using a rapid sequence induction technique. After intubation, sedation and paralysis were maintained for the duration of the 90-minute transfer with midazolam/morphine infusion and bolus vecuronium, respectively.

The Royal Flying Doctor Service retrieval team, consisting of a physician and flight nurse, was able to measure the patient's blood sodium as 119 mmol/L using an i-Stat point-of-care device and administered a single 100 mL 3% hypertonic saline (HTS) bolus during the transfer. The 3% saline solution is not normally carried by the Royal Flying Doctor Service but was carried on this flight because of the clinical suspicions of the

emergency physicians caring for the patient on the island. Her first sodium measurement on arrival in the receiving intensive care unit, taken from an arterial gas sample 5 hours after her initial seizure and more than 2 hours after the 3% HTS was given, was still only 118 mmol/L. In the intensive care unit, she was administered 136 mmol NaCl (given as 40 mL of a 20% solution) as specific treatment to correct her hyponatremia, as well as limited maintenance fluids. By the morning, and by then 24 hours after starting the swim, her sodium was 131 mmol/L. She was able to be extubated, neurologically intact, later the same day. After a short hospital stay, she was discharged with no residual deficits and has now returned to open water swimming but over shorter distances.

Discussion

This is the first case of EAHE in an open water swimmer reported in the literature. Its importance is to emphasize that EAH and EAHE should be considered as a diagnostic possibility in all endurance athletes presenting with nonspecific symptoms. These nonspecific symptoms may represent many possible diagnoses and are to some extent environment specific. In this case, they were initially thought to be due to hypothermia, and only with time was it considered that they may have been manifestations of developing cerebral edema from EAH, and later, EAHE.

The pathophysiology of EAH, and particularly of EAHE, is now well understood. It is described in detail elsewhere,⁹⁻¹¹ but in simple terms, the key factors in the most serious cases are fluid consumption in excess of requirements and a failure to excrete that excess fluid related to inappropriately elevated arginine vasopressin (AVP) levels. Features in our case point to these factors being at play, although the difficulties in knowing exact fluid intake and the lack of biochemical analyses at presentation leave us unable to confirm that. Our subject's reported fluid intake, when totaled, averaged more than 600 mL · h⁻¹ from 2 hours before the race until she was transported to more definitive medical care at the island nursing post. Her insensible losses might be expected to be less than that, given limited evaporation while mostly immersed in water and with prolonged exertion at only moderate levels. Her fluid intake falls at the lower end of the range reported with EAH,⁹⁻¹¹ but it is noteworthy that she is a smaller (65 kg) female athlete, which needs to be considered in the context of her fluid intake and losses. In addition, our anecdotal experience is that nausea and vomiting are common in this event, as occurred in our subject. Nausea and vomiting are themselves stimuli to increased AVP secretion,^{1,11} which

in our subject might have led to inadequate excretion of ingested fluid. If increased AVP secretion persisted in our subject, then the final decompensation to active seizures after normal saline administration may have been due to further water, but not salt, retention and to reaching a critical level of hyponatremia-induced cerebral cellular swelling and edema. Thus, both her oral fluid intake after the race and later IV fluids may have precipitated her clinical decline into EAHE. Such a decompensation in athletes with EAH after normal saline administration has been previously reported^{12,13} and serves to emphasize why IV normal saline should be used very cautiously for athletes in whom EAH is a possibility, unless signs of frank hypovolemia are present.¹¹ That our subject's sodium concentration was essentially unchanged on intensive care unit arrival may have been due to further fluid reabsorption from her gut,¹¹ despite the first dose of 3% HTS having been given.

The patient's ultimate recovery serves to emphasize the importance of recognition based on point-of-care testing and symptomatology together with the use of HTS as the specific therapy. In milder cases of EAH, oral HTS may be as effective as IV HTS,¹⁴ and it could have been used empirically early in her medical care. Once she decompensated, a firm diagnosis could not be made until the retrieval service arrived with the capacity for point-of-care testing. Their use of IV HTS and subsequent use in the intensive care unit doubtless contributed to her favorable outcome. Empiric use of IV HTS for suspected EAHE is a consideration in the wilderness setting¹¹ and would have been justified in this case had it been available. Diagnosis and treatment based on measured sodium is still preferred, and for this event, the island nursing post now has the capacity both to measure sodium by point-of-care testing and to administer IV HTS.

In reporting this case, we hope that increased vigilance and capacity for treatment of EAH and EAHE will occur for similar mass participation open water swimming and for other endurance events. The key issues to address are these: encourage athletes to use a "drink to thirst" strategy, be vigilant for the possibility of an EAH/EAHE diagnosis for unwell endurance athletes, and use empiric or sodium measurement guided HTS.^{1,11}

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