Exercise-associated hyponatraemia on the Kokoda Track

David A Pattison, Tomos E Walters and Eric Seal

Clinical record

A previously well 43-year-old Australian lawyer was hiking the Kokoda Track in Papua New Guinea in August 2008. She awoke with a headache on the second day and, suspecting dehydration, consumed about 7 L of fluid while hiking.

By late afternoon she complained of increased headache and nausea, which was exacerbated by her lying supine. She developed seizures several hours after profuse vomiting. Temazepam and metoclopramide were administered rectally due to limited medical resources. Three doctors present provisionally diagnosed dilutional hyponatraemia but had no facilities for intravenous therapy. Arrangements were made for urgent repatriation by helicopter to Port Moresby but this was later abandoned due to bad weather.

She deteriorated overnight, becoming unresponsive to painful stimuli and lapsing into coma. Her vomiting and convulsions continued. With no rescue imminent, salt solution approximating normal saline was administered rectally. There was some improvement in eye-opening and verbal responses on the Glasgow Coma Scale.

Fortuitously, an American naval hospital ship anchored outside Port Moresby retrieved her via helicopter the following afternoon. Her plasma sodium on arrival to intensive care was 114 mmol/L. After she was intubated and treated with intravenous hypertonic saline, the patient made a good recovery. ◆

Exercise-associated hyponatraemia (EAH) is a modern, life-threatening condition first described in 1985[1] after introduction of guidelines promoting excessive fluid intake during exercise.[2] EAH is defined as hyponatraemia occurring during or up to 24 hours after prolonged exercise (generally > 4 hours duration).[3] This "conditioned overhydration" — drinking beyond thirst, variously influenced by misunderstanding of exercise physiology, media including sports-drink advertising,[4] and forced rehydration protocols[5] — has been reported among hikers,[6] military personnel[5] and long-distance sports participants.[7] Despite being well documented in scientific literature, those most at risk are unaware of this preventable condition.

EAH is common, with reported incidences of hyponatraemia (serum sodium concentration, < 135 mmol/L) and critical hyponatraemia (serum sodium concentration, < 120 mmol/L) during the 2002 Boston Marathon of 13% and 0.6%, respectively.[7] At least eight fatalities have been documented[8] — likely an underestimation given difficulties with postmortem diagnosis.[9] The unexplained deaths in 2009 of four previously well hikers on the Kokoda Track in similar conditions provide urgency to the need to raise awareness of the association between overhydration and EAH.

Extensive research confirms EAH is primarily dilutional secondary to overhydration,[10] manifest as weight gain during exercise. That only a small proportion of individuals exposed to overhydration develop hyponatraemia suggests a role for associated underlying defects in free water excretion. These include exercise-induced non-osmotic antidiuretic hormone secretion,[11] while the recent description of an activating mutation of the arginine vasopressin receptor 2[12] may explain the undetectable antidiuretic hormone levels found in other cases.[13] Excessive-sweat sodium losses associated with subclinical cystic fibrosis have also been described.[14]

Identified risk factors for EAH[8] include excessive drinking behaviour, weight gain during exercise, female sex, slow performance pace, high availability of drinking fluids, > 4 hours' exercise duration and hot environmental conditions consistent with our scenario. Female sex hormones inhibit cellular sodium–potassium–ATPase function, which may explain the observed higher risk of EAH and cerebral oedema among women.[8] Slow performance pace may reflect insufficient physical training and provides the opportunity for overhydration.

Symptoms include lethargy, dizziness, headache, nausea and vomiting, with progression to confusion, ataxia, seizures and coma.[3] Importantly, EAH cannot be easily distinguished clinically from heat exhaustion, with subsequent mistaken "rehydration" exacerbating the condition. EAH requires a high index of suspicion to facilitate timely evacuation for biochemical diagnosis and treatment. Specific clinical features include euvolaemia and polyuria. A recent review of 145 United States military cases identified that the training cadre often mistook EAH for dehydration, and treatment by aggressive water rehydration had fatal consequences in three cases.[15] Overhydration was encouraged by well-meaning guides and colleagues in another near fatal case on the Kokoda Track reported in 2008.[5]

In this context, a prominent tour operator's media assertion that "dehydration" in "the death zone"[16] caused the recent deaths among young healthy Kokoda Track hikers may perpetuate a dangerous culture of conditioned overhydration. It is of grave concern that, in 2009, a second fatality occurred shortly after media speculation that dehydration was the cause of the first. Available evidence suggests that, in an environment of excess water (most trekkers carry > 4 L water per day), hikers on the Kokoda Track should be more concerned with severe EAH secondary to overhydration, rather than with dehydration.

Initial treatment of EAH is fluid restriction to avoid exacerbation of hyponatraemia. Those with critical hyponatraemia or symptomatic, biochemically confirmed EAH require treatment with intravenous hypertonic saline (100 mL of 3% saline solution over 10 minutes) in a supervised environment. This is based on the assumption that the hyponatraemia is acute (< 48 hours) and that no cases of osmotic demyelination syndrome have been reported in treating EAH.[3]

No single preventive fluid intake regimen can be recommended to cover all activities. The Second International EAH Consensus Development Conference statement[3] recommends drinking to thirst instead of a predetermined protocol. The aim should be never to gain weight during endurance exercise and to expect a small percentage weight loss (1%–2%) due to substrate use.[3] Fluid intake requirements could be estimated for guided treks by comparison to baseline weight. Point-of-care electrolyte testing could be used. There is insufficient evidence to recommend salt tablet use.[3] Importantly, there is no evidence that commercial sports drinks prevent hyponatraemia[3] — in fact, given their sodium hypotonicity relative to normal saline (10–20 mmol/L v 145 mmol/L), excessive consumption could worsen hyponatraemia.

Lessons from practice

- Military personnel, hikers and endurance sports participants are at risk due to overhydration during prolonged exercise.
- Non-specific symptoms are commonly mistaken for dehydration.
- Diagnosis requires a high degree of suspicion, and biochemical testing.
- Water should be consumed according to thirst and guided by weight comparison before and after exercise.
- Weight gain should be avoided, aiming for a 1%–2% weight loss during prolonged exercise.
- Education of at-risk groups is essential for prevention. ◆

At a public health level, education of those leading and participating in high-risk activities is critical. The number of EAH casualties at a New Zealand ultradistance event was reduced by spacing the distance between, and volume of fluid available at, drinking stations.[17]

EAH is a modern, life-threatening condition which is preventable through adherence to sensible fluid intake during prolonged exercise. Although American sports and military bodies have revised their guidelines, researchers have been critical of the sports-drink industry's role in perpetuating a culture of overhydration.[18] As medical practitioners, it is our responsibility to ensure the wider community is aware of the risks of conditioned overhydration during exercise in the face of lay misinformation and commercial interests.

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Competing interests

None identified.

Author details

David A Pattison, MB BS, Endocrinology Registrar[1]
Tomos E Walters, MB BS, BMedSci, Senior Cardiology Registrar[1]
Eric Seal, MB BS, FRACP, PhD, General Physician[2]
1 Royal Melbourne Hospital, Melbourne, VIC.
2 St Vincent's Hospital, Melbourne, VIC.
Correspondence: **david.pattison@mh.org.au**

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