Rare disease

Exercise associated hyponatraemia leading to tonic-clonic seizure

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Summary

A 34-year-old Filipino lady presented to the emergency department with breathlessness and muscle cramping following a Bikram yoga workout. The patient reported sweating excessively while performing 90 min of strenuous exertion in a humidified room heated to an ambient temperature of 40.6°C. After the workout she drank 3.5 litres of water before experiencing breathlessness, severe muscle cramps, nausea and general malaise. Initial investigations revealed severe hyponatraemia (120 mmol/l). Despite early sodium replacement the patient dropped her Glasgow coma scale to 9/15 and developed tonic clonic seizures, requiring intubation and admission to the intensive care unit. The hyponatraemia was slowly corrected on the intensive care unit and the patient made a full recovery over the course of 5 days. This case highlights the dangers of overzealous fluid replacement following severe exertion in a hot environment.

BACKGROUND

Bikram yoga is an increasingly popular pastime. While it is perceived as a healthy activity, this case demonstrates it is not without risk and that care must be taken following exercise in the rehydration phase. Bikram yoga induced hyponatraemic tonic clonic seizure has not been described before. It is an important and significant adverse outcome that is likely to be encountered by other healthcare workers in an acute medical setting.

CASE PRESENTATION

A previously well 34-year-old Filipino lady presented to the emergency department with breathlessness, muscle cramps, nausea and general malaise following her first session of Bikram yoga. The breathlessness had come on gradually and there was no associated chest pain, calf pain, palpitation, sweating or vomiting. Before the onset of symptoms, the patient reported sweating excessively while performing 90 min of strenuous exertion in a humidified room heated to an ambient temperature of 40.6°C and then drinking 3.5 litres of water. Physical examination was remarkable only for the patient appearing euvoalaemic and having a low body mass index of 19.

An intravenous infusion of normal saline with 20 mmol KCL added was set up. The patient then had a self-terminating tonic clonic seizure lasting approximately 30 s and was transferred to the resuscitation area. Her postictal Glasgow coma scale (GCS) was 11/15 (M5, V2, E4). A nasopharyngeal airway was inserted and urgent CT head arranged. The GCS fell further to 9/15 and the patient was transferred to the intensive care unit (ICU) and intubated.

INVESTIGATIONS

Initial bedside tests revealed 3+ urinary ketones, β human chorionic gonadotropin negative and a blood glucose of

8 mmol/l. An arterial blood gas performed on air showed a respiratory alkalosis with a severe hyponatraemia:

Initial blood gas on air

- ▶ pH 7.67
- ▶ pCO₂ 1.8 kPa
- ▶ pO₂ 18 kPa
- ▶ Na 120 mmol/l
- ▶ K 3.6 mmol/l
- ▶ Cl 90 mmol/l
- ▶ Lactate 2.7 mmol/l
- ▶ Base excess (BE) 1.9 mmol/l
- ▶ HCO₃ std 23.4 mmol/l

Post seizure blood gas on 15 l via a non-rebreath mask

- ▶ pH 7.10
- ▶ pCO₂ 3.7 kPa
- ▶ pO₂ 55.6 kPa
- ▶ Na 122 mmol/l
- ▶ K 3.0 mmol/l
- ▶ Cl 86 mmol/l
- ▶ Lactate 12.6 mmol/l
- ▶ BE 19.6 mmol/l
- ▶ HCO₃ std 9.7 mmol/l

Lab urea and electrolytes at presentation

- ▶ Na 120 mmol/l
- ▶ K 3.7 mmol/l
- ▶ Cl 89 mmol/l
- ▶ Urea 2.7 mmol/l
- ▶ Creatinine 69 μ mol/l

Serum osmolality (S_{osm})=(2×serum (Na))+(serum (glucose)/18)+(blood urea nitrogen/2.8).

S_{osm}=241.4 (285–295 mOsm/kg).

Chest x-ray: no abnormality noted.

CT head: no evidence of intracerebral mass, haemorrhage or acute ischaemia.

DIFFERENTIAL DIAGNOSIS

Causes of euvolaemic hypotonic hyponatraemia

- ▶ Effective arterial blood volume depletion¹
 - ▶ True volume depletion
 - ▶ Heart failure and cirrhosis
- ▶ Syndrome of inappropriate antidiuretic hormone (ADH) secretion
- ▶ Hormonal changes (adrenal insufficiency, hypothyroidism, pregnancy)
- ▶ Exercise-associated hyponatraemia
- ▶ Hyponatraemia despite appropriate suppression of ADH
 - ▶ Advanced renal failure
 - ▶ Primary polydipsia
- ▶ Ecstasy (MDMA) intoxication
 - ▶ Low dietary solute intake.

TREATMENT

In ICU, the patient was treated with a slow infusion of 3% hypertonic saline (50 ml/h representing 1 mg/kg/h. A right femoral line was inserted using aseptic non-touch technique to facilitate hourly venous blood gas monitoring of serum sodium concentration. When the serum sodium concentration reached 126 mmol the saline infusion was switched to 0.9% normal saline (80 ml/h).

OUTCOME AND FOLLOW-UP

Sodium levels normalised by day 3 and the patient was stepped down from the ICU. Full recovery occurred by day 5 and the patient was discharged.

DISCUSSION

Exercise induced hyponatraemia (EAH) is well described in the literature and defined as hyponatraemia occurring within 24 h of prolonged physical activity.² The major risk factor for EAH appears to be overzealous fluid replacement during and after exercise leading to haemodilution.³ Other independent risk factors include having a lower body mass index and a longer duration of exercise. Pathogenesis is thought to involve a combination of haemodilution, persistent secretion of ADH and excessive sodium losses from sweating.^{2 4 5} EAH may

be asymptomatic or present with non-specific manifestations such as malaise, weakness, headache, nausea and/or vomiting. Severe manifestations include seizure, confusion, coma and death.² Mild to moderate symptoms may be managed conservatively with fluid restriction. Those with more severe symptoms and proven hyponatraemia benefit from hypertonic (typically 3%) saline. The rate of correction is controversial; case series have found in EAH hyponatraemia may be corrected more rapidly than the generally recommended rate of 10 meq/l per 24 h without the development of the osmotic demyelination syndrome.² The major pitfall to avoid is the use of hypotonic and isotonic fluids such as 0.9% saline in this population since this may worsen hyponatraemia in cases where the patient is euvolaemic and ADH is elevated. Prevention is achieved by advising athletes to drink according to their thirst.²

Learning points

- ▶ Exercise-associated hyponatraemia should be considered in patients with a history of prolonged exercise and excessive water intake.
- ▶ Symptoms of hyponatraemia include weakness, dizziness, bloating, headache, nausea and/or vomiting
- ▶ Athletes should be advised to drink according to their thirst.
- ▶ Isotonic and hypotonic intravenous fluid infusions should be avoided in patients with established exercise-associated hyponatraemia.

Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Sterns RH.** Causes of hyponatremia. In: UpToDate, Basow DS, ed. Waltham, MA: UpToDate 2011.
2. **Hew-Butler T,** Ayus JC, Kipps C, *et al.* Statement of the Second International Exercise-Associated Hyponatremia Consensus Development Conference, New Zealand, 2007. *Clin J Sport Med* 2008;**18**:111–21.
3. **Almond CS,** Shin AY, Fortescue EB, *et al.* Hyponatremia among runners in the Boston Marathon. *N Engl J Med* 2005;**352**:1550–6.
4. **Buono MJ,** Ball KD, Kolkhorst FW. Sodium ion concentration vs. sweat rate relationship in humans. *J Appl Physiol* 2007;**103**:990–4.
5. **Shibasaki M,** Wilson TE, Crandall CG. Neural control and mechanisms of eccrine sweating during heat stress and exercise. *J Appl Physiol* 2006;**100**:1692–701.

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