

lesson of the month (1)

With a pinch of salt

This report highlights a case of severe hyponatraemia secondary to excessive sweating – poor fluid consumption and low salt diet in hot conditions. The case was complicated by the presence of marked hypokalaemia caused by secondary hyperaldosteronism confirmed, for the first time, by the presence of grossly elevated serum renin and aldosterone concentrations. With the rise in global temperature affecting even temperate climates doctors, especially in acute and general medicine, may be faced with this condition more often.

Lesson

The summer of 2006 was the hottest on record in the UK.¹ A rise in global temperatures may lead to a potentially lethal hyponatraemia in the absence of severe dehydration, even in countries with temperate climates.

A 22-year-old male was admitted at 0040 hours on 27 July 2006 having been found collapsed by his mother. The patient had been feeling unwell with non-specific symptoms for several days. Blood pressure was 129/69 mmHg, pulse 80 per minute, regular rhythm, temperature 36.1°C. He was agitated with a Glasgow Coma Scale of 11/15. There were no focal neurological signs but the cerebral impairment was sufficient to justify examination of the head by computed tomography (CT) scan and of the cerebrospinal fluid, both of which were unremarkable. Initial laboratory findings are shown in Table 1 where hyponatraemia, hypokalaemia and alkalosis are apparent.

The patient was treated cautiously with 0.9% NaCl, to limit the rise of the serum sodium concentration to 1 mmol/l/hour, and with a potassium infusion. Urine output was 5,830 ml over the first 12 hours with an overall negative balance of 2,830 ml. Twenty-four hours after admission the patient was alert and orientated with normal electrolytes.

Further history was available following recovery. He had recently joined a building firm as a driver, lifting heavy materials

and driving a truck without air conditioning. He reported he had been sweating considerably, so he drank in excess of eight litres of bottled water daily, as suggested by recent advertising campaigns, to maintain his hydration. Furthermore he was taking a low salt diet as recommended by a second media campaign running concurrently.

Discussion

The combination of hyponatraemia, marked hypokalaemia and a metabolic alkalosis in a young fit normotensive person is rarely seen in the acute admissions unit. In the absence of any other identifiable cause, the collapse of this patient was probably due to hyponatraemia caused by a combination of salt loss through sweating and dilution due to a high intake of hypotonic fluid. In cases caused by poor salt intake and excessive sweating one would expect signs of dehydration such as postural hypotension and raised urea and creatinine, none of which were evident in this present case. The hypokalaemia was a consequence of secondary hyperaldosteronism, a response to the hyponatraemia. The metabolic alkalosis was caused by upregulation of the renal H⁺/K⁺ ATPase to increase K⁺ absorption at the expense of enhanced H⁺ secretion.²

Hyponatraemia secondary to excessive salt and poor fluid ingestion is now being recognised as a cause of morbidity and

Table 1. Laboratory results on admission.

Test	Result (units)	Range
Sodium	115 (mmol/l)	134–146
Potassium	1.5 (mmol/l)	3.4–5.2
Urea	5.8 (mmol/l)	3.2–7.6
Creatinine	116 (μmol/l)	60–126
Magnesium	0.80 (mmol/l)	0.70–0.95
Glucose	9.5 (mmol/l)	
Corrected calcium	2.02 (mmol/l)	2.10–2.60
Cortisol	1,030 (nmol/l)	
C-reactive protein	<3 (mg/l)	<10
Serum osmolality	241 (mosm/kg)	278–294
Urine osmolality	167 (mosm/kg)	
Urine potassium	15 (mmol/l)	
Urine sodium	<10 (mmol/l)	
Venous blood pH	7.51	7.35–7.45
Bicarbonate	33.3 (mmol/l)	
Aldosterone	3,300 pmol/l	(28–445)
Plasma renin activity	33.51 nmol/l	(0.35–2.03)

Kalyan Kumar Gangopadhyay,¹ consultant in diabetes and acute medicine; **R Gupta**,¹ specialist registrar in anaesthesia; **V Baskar**,² consultant physician; **N Gautam**,¹ consultant in medicine and intensive care; **AA Toogood**,¹ consultant physician

¹University Hospital Birmingham NHS Foundation Trust

²The Royal Wolverhampton Hospitals NHS Trust

mortality among athletes and in the military (where inadequate dietary salt intake in hot countries could be another factor) but is not well appreciated outside these communities.^{3–5} However, given the increasing temperatures, even in the temperate zones, doctors may be faced with this condition in the general population more frequently.

The potential dangers of severe dehydration during exercise have been so well publicised that some individuals choose to drink large volumes of fluid on a daily basis.^{3–7} In this case the impact of climate change and concurrent advertising campaigns lead to a potentially life-threatening condition. While a certain level of water intake may be healthy, the public need to be aware that excessive intake can be dangerous, particularly when fluid loss is due to excessive sweating.

We explained to our patient the cause of his collapse and advised him to avoid drinking excessive salt-poor fluids during periods of exertion and drink according to thirst. He has been well since.

References

- 1 www.met-office.gov.uk/climate/uk/2006/index.html
- 2 DuBose TD Jr. Acidosis and alkalosis. In: Kasper DL, Fauci AS, Longo DL *et al* (eds), *Harrison's principles of internal medicine*, 16th edn. New York: McGraw Hill, 2005:263–71.
- 3 Noakes TD, Speedy DB. Case proven: exercise associated hyponatraemia is due to overdrinking. So why did it take 20 years before the original evidence was accepted. *Br J Sports Med* 2006;40:567–72.
- 4 Noakes TD. Overconsumption of fluids by athletes. *BMJ* 2003;327:113–4.
- 5 O'Brien K, Montain SJ, Corr WP *et al*. Hyponatraemia associated with overhydration in US army trainees. *Mil Med* 2001;166,5:405–10.
- 6 Vrijens DMJ, Rehrer NJ. Sodium free fluid ingestion decreases plasma sodium during exercise in the heat. *J Appl Physiol* 1999;86:1847–51.
- 7 Irving RA, Noakes TD, Buck R *et al*. Evaluation of renal function and fluid homeostasis during recovery from exercise – induced hyponatraemia. *J Appl Physiol* 1991;70:342–8.

Address for correspondence: Dr K Gangopadhyay,
A4/4/14 Calcutta Green, 1050/2 Survey Park,
Kolkata 75, West Bengal, India 700075.
Email: jaykal69@hotmail.com