

Hypernatraemia and rhabdomyolysis

Jason P Denman

A 44-year-old man with a history of childhood brain injury presented with dysarthria, confusion, reduced oral intake and reduced mobility after a week of heatwave conditions. He had severe hypernatraemia and raised serum creatine kinase levels, consistent with rhabdomyolysis. In most previous case reports linking hypernatraemia and rhabdomyolysis, other factors have potentially contributed. From the available evidence, severe hypernatraemia alone appears sufficient to induce muscle injury. (MJA 2007; 187: 527-528)

Clinical record

A 44-year-old man presented to a rural hospital with a 48-hour history of reduced mobility, dysarthria, confusion and reduced oral intake. He had sustained a traumatic brain injury at the age of 6 years and had intellectual impairment, mild right hemiplegia and post-traumatic epilepsy, but had not experienced a seizure for many years. Ambient temperatures were anecdotally reported to be up to 48°C during the week before presentation.

The patient was cared for by his brother, who confirmed there had been no recent seizures, exertion, significant falls or prolonged recumbency. His only regular medication was rabeprazole. At presentation, he was dehydrated, febrile (temperature, 38°C), anuric and hypotensive, with a blood pressure of 90/50 mmHg.

Initial blood biochemical tests showed the following levels: serum sodium, >180 mmol/L (reference range [RR], 135–145 mmol/L); serum potassium, 4.3 mmol/L (RR, 3.2–4.5 mmol/L); bicarbonate, 13 mmol/L (RR, 22–33 mmol/L); haemoglobin, 204 g/L (RR, 135–180 g/L); and serum creatinine, 455 µmol/L (RR, 70–120 µmol/L). Creatine kinase (CK) concentration was 13 000 U/L (RR, <200 U/L), while troponin I was minimally elevated, with a peak value of 0.8 µg/L (RR, <0.2 µg/L), consistent with rhabdomyolysis. Laboratory technical difficulties precluded a urinary myoglobin assay. A snake venom assay was negative, and the patient's coagulation profile was normal. On clinical examination, there was no evidence of trauma or compartment syndrome.

Initial treatment comprised fluid resuscitation (with a total of 5 L intravenous fluid) and empirical intravenous antibiotics. The patient was transferred to a tertiary hospital intensive care unit, where he received further fluid replacement, electrolyte correction and renal replacement therapy. A single brief generalised tonic-clonic seizure several hours after his arrival was treated with phenytoin. The CK concentration peaked at 31 200 U/L. Renal function recovered to baseline over 6 weeks, and the man was ultimately discharged home from the hospital's rehabilitation unit, having returned to his premorbid functional state.

Discussion

The patient developed severe dehydration and hypernatraemia as a result of increased insensible losses and inadequate fluid intake during a week of extreme weather conditions, despite having unrestricted access to water. Whether he had a degree of hypothalamic hypodipsia as a result of acquired brain injury is uncertain. A careful history and physical examination did not suggest a single obvious cause for his rhabdomyolysis. Core temperature was not sufficiently elevated to favour heat stroke as a diagnosis. The CK concentration was already raised before the seizure, which

was not prolonged. A review of the literature identified hypernatraemia as a potential contributor.

Rhabdomyolysis is a clinical and laboratory syndrome characterised by muscle necrosis and release of intracellular muscle constituents into the circulation. It ranges in severity from asymptomatic elevation of CK concentration to severe life-threatening cases associated with extreme rise in CK concentration, myoglobinuria and acute renal failure. The most common causes include muscle compression or trauma, hyperexertional states (such as prolonged seizure or extreme exercise), metabolic and inflammatory myopathies, heat stroke, and drug- or toxin-related muscle injury.¹ Severe electrolyte derangements, including hyponatraemia, hypokalaemia and hypophosphataemia, are also described, with the proposed mechanism being cell membrane disruption as a result of deranged sodium–potassium–ATPase pump function.²

Multiple case reports and series have linked hypernatraemia and rhabdomyolysis.^{3–5} Most cases have been in the setting of hyperosmolar states, such as those associated with diabetes mellitus. In many cases, other factors have potentially contributed to the development of rhabdomyolysis, including recumbency resulting from altered levels of consciousness or seizures.

Evidence for a direct causal link between hypernatraemia and rhabdomyolysis is provided by three reported cases of central diabetes insipidus resulting in severe hypernatraemia and rhabdomyolysis in the absence of other potential causes.^{6–8} In all cases, the serum sodium concentration was greater than 180 mmol/L.

In 1992, Abramovici and colleagues, reporting a case series of 18 patients with hypernatraemia, showed a significant linear correlation between serum sodium concentration and serum CK level. They were then able to induce rhabdomyolysis in laboratory rats by rendering them severely hypernatraemic after fasting and intraperitoneal injection of hypertonic saline. Pre- and post-injection biochemical analyses confirmed that only serum sodium concentration and CK concentration were altered significantly.⁹

In summary, rhabdomyolysis can be induced by a range of insults, and is often multifactorial. From the available evidence, severe hypernatraemia alone appears sufficient to induce muscle injury, and should be considered as a potential cause.

Competing interests

None identified.

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